



جهاز أبوظبي للرقابة الغذائية  
ABU DHABI FOOD CONTROL AUTHORITY

## **CODE OF PRACTICE**

**No. (7) of 2011**

# **FARM ANIMAL DISEASES EPIDEMIOLOGY AND CONTROL GUIDELINES**

**Endorsed by BOD**

28 June 2011



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			ETEC	Enterotoxigenic Escherichia coli
			EVA	Equine Viral Arteritis
			EVAV	Equine Viral Arteritis Virus
			FMD	Foot and Mouth Disease
			FMIA	Fluorescent microsphere immunoassay
			FT	Fowl Typhoid (FT)
			HA	Hemagglutination
			H & E	Hematoxylin and Eosin stain
			HPAI	Highly Pathogenic Avian Influenza
			IB	Infectious Bronchitis
			IBD	Infectious Bursal Disease
			IBDV	Infectious Bursal Disease Virus

IBP	Infectious balanoposthitis
IBR	Infectious Bovine Rhinotracheitis
IgG	Immunoglobulin G
IgM	Immunoglobulin M
ILT	Infectious Laryngotracheitis
IP	Infected Premises
IPV	Infectious Pustular Vulvovaginitis
IU	International Unit
LSD	Lumpy Skin Disease
LSDV	Lumpy Skin Disease Virus
MCF	Malignant Catarrhal Fever
MCFV	Malignant Catarrhal Fever Virus
ND	Newcastle disease
NDV	Newcastle disease virus
NTEC	Necrotoxogenic Escherichia coli
NVND	Neurotropic Velogenic Newcastle Disease
OIC	Officer In Charge
OIE	Office International des Epizooties World Organization for Animal Health
PCR	Polymerase Chain Reaction
PD	Pullorum disease (PD)
PDS	Personal Decontamination Site
PPR	Peste des Petits Ruminants
PPRV	Peste des Petits Ruminants Virus
PT	Paratyphoid (PT)
rMSP5	recombinant 19 kDa major surface protein 5
RT-PCR	Real Time - Polymerase Chain Reaction
RVF	Rift Valley Fever
RVFV	Rift Valley Fever Virus
SRM	Specified Risk Material
TAD	Trans-boundary animal disease
TSE	Transmissible Spongiform Encephalopathy
VN	Virus Neutralization
VTEC	Verocytotoxic Escherichia coli
VVND	Viscerotrophic Velogenic Newcastle Disease
WNF	West Nile Fever
WNFV	West Nile Fever Virus

## 1. Introduction

Today, the veterinary profession is confronted with a different set of problems compared with the middle of last century. Veterinarians often have to deal with herds or regions that remain infected with diseases even after lengthy disease prevention or control campaigns have taken place. Exotic livestock diseases have serious consequences that cause severe production losses, illness and/or death in animals and possibly humans. These diseases also have serious economic consequences and could be devastating. Veterinary epidemiology deals with the investigation of animal diseases and the frequency of their occurrence while considering their economic and public health impact at national levels. Rapid detection and response is needed to stop the spread of any infectious disease within a population. This requires coordination and cooperation between local and federal agencies. Constant preparedness at the local level will greatly aid in the response and recovery efforts needed to protect animal and human health.

The ultimate goals through the use of preventive veterinary medicine techniques are to prevent or control a disease problem, reduce productivity losses and improve animal welfare. This manual contributes to these goals by assisting with the diagnosis of diseases in the Abu Dhabi Emirate and supporting the preventive actions. An extensive review of available literature was performed to provide the most recent and up-to-date knowledge to fulfill the demands of the field practice. Federal and Local laws and by-laws in UAE regarding quarantine and other veterinary medicine activities were considered. It is a challenge to prepare Veterinary manual that fits all the needs of veterinary practitioners in Abu Dhabi. However, this manual is not an alternative to veterinary textbooks; it aims at providing veterinarians with brief information about some important viral, bacterial, parasitic as well as poultry diseases that they may encounter during their practice. It also provides guidelines on animal diseases notification and description of normal healthy animal appearance usually observed in the field. Additionally, this manual describes methods of destruction, disposal & decontamination that constitute the essential components of any disease control efforts in outbreak situations and epidemics.

## Chapter (1) Preface

- 1.1 Appearance of the healthy animal
- 1.2 Notification Guidelines
- 1.3 Animal Destruction, Disposal and Premises Decontamination

### 1.1 Appearance of the healthy animal

Veterinary practitioner is able to distinguish between the sick and the healthy animal. Identifying the signs of ill health in livestock will mean that he can:

- a) Give first aid and treat ill animals quickly
- b) Prevent the spread of disease to other animals
- c) Recognize any problems in animals offered for sale
- d) Recognize any signs of health problems in animals to be used for rearing or breeding

The healthy animal is alert and aware of its surroundings. It is active and holds its head up watching what is happening around it. It should stand on all of its feet. The separation of an animal from the others in its group is often a sign of a health problem. An animal which is not interested in its surroundings and does not want to move has health problems.

1.1.1 Movement (gait): The healthy animal will walk easily and steadily with all of its feet taking its weight. Steps should be regular. Irregular movement results from pain in the feet or limbs or other diseases. If you go near an animal that is lying down it should stand up quickly otherwise it has health problems.

1.1.2 Eyes: The eyes should be bright and alert with no discharge at the corners.

1.1.3 Ears: Most animals have erect ears which move in the direction of any sound. Ear movements will also be quick to get rid of flies.

1.1.4 Nose and Muzzle: The nose should be clean with no discharge. In cattle and buffalo the muzzle should be moist not dry. In sheep and goats the nose should be cool and dry. Healthy animals frequently lick their noses with their tongues.

1.1.5 Mouth: There should be no saliva dripping from the mouth. If chewing is slow or incomplete there must be a problem with the teeth.

1.1.6 **The coat:** In short-haired animals, e.g. goat and cattle, the hair or coat of the healthy animal will be smooth and shiny. Healthy cattle, buffalo and their calves lick their coat and the lick marks will show. Horses should not sweat when resting. In poultry the feathers should be smooth and glossy and not ruffled.

1.1.7 Behavior: If a horse, cow or buffalo keeps looking at its flanks or kicks at its belly it has a pain in the stomach. Behavior changes are significant and diagnostic in diseases like rabies and BSE.

1.1.8 Breathing: Breathing should be smooth and regular at rest. Remember that movement and hot weather will increase the rate of breathing. If the animal is resting in the shade it should be difficult to notice the chest moving as it breathes.

1.1.9 Pulse: Taking the pulse is important when examining an animal. In man the pulse can be easily taken but in animals it is more difficult and requires practice.

a) In sheep and goats you can feel the pulse on the inside of the top of the back leg (femoral artery). The rate of the pulse is 70 - 130 per minute in the adult.

b) The pulse of cattle is taken at a point on the underside of the base of the tail (middle coccygeal artery), the normal rate is 40 - 80 per minute in the adult. In buffalo the pulse rate is 40 - 60 per minute.

c) The pulse of the horse is taken on the inside of the cheek (external maxillary artery). The normal rate is 35 - 40 per minute.

d) The pulse of the camel is taken at a point on the underside of the root of the tail like cattle. The normal rate is 35 - 45 beats per minute.

Remember that the pulse will be higher in the young animal. To take the pulse you should feel for it with the first two fingers of the hand.

1.1.10 Droppings or dung: The droppings of the healthy animal will be firm. Very soft droppings are signs of diarrhea which is a sign of ill health. If the animal has difficulty in defecating (constipation) this is also a bad health sign.

1.1.11 Urine: The urine should be clear and the animal shows no signs of pain or difficulty in urinating. Horses, mules and donkeys can have thick yellow urine which is normal.

1.1.12 Appetite and rumination: The animal should eat and drink normally. Failure to eat is an obvious sign of ill health. If feed is available the healthy animal will have a full belly.. Sheep, goats, cattle, buffalo and camels chew the cud (ruminate) for 6 to 8 hours each day. It is a sign of ill health when these animals stop ruminating.

1.1.13 Milk: In milk producing animal a drop in the amount of milk produced can mean a health problem. Any sign of blood or other matter in the milk points to infection in the udder. There should be no swelling of the udder and no sign of pain when it is touched. There should be no injury to the teat.

1.1.14 Body temperature: If you suspect that an animal is sick you should check its body temperature. A higher than normal body temperature is a sign of an infection.

### 1.1.15 Spread of disease

Disease occurs when something goes wrong with the body or part of the body. They can be caused by germs (infectious diseases), bad feed, chemicals or injuries. An infectious disease can spread from one animal to another.

- a) How animals become ill.
  - b) What is meant by the spread of disease?
  - c) What is an infectious disease?
  - d) What is a non-infectious disease?
  - e) How to prevent the spread of disease.
- 1.1.16 The main causes of disease: Disease can be classified as acute or chronic. An acute disease starts quickly and lasts for a short period when the animal either recovers or dies. A chronic disease lasts for a long time and weakens the animal. Diseases are said to be infectious (will spread from one animal to another) or noninfectious (will not spread from one animal to another). Non- infectious diseases can be caused by poor feed and the lack of minerals, salts and vitamins that the body needs. Non-infectious disease can also be caused by poisoning with chemicals or plants, by cuts, burns and broken bones. Some diseases pass from the parent to the young (hereditary). Many non- infectious diseases are chronic but they can be acute. They can cause large losses of meat, milk and wool. Working (draught) animals do not work well and the rate of reproduction can be low with the young being born dead or dying before they are weaned. Chronic diseases are often thought to be "normal" but when the cause is known and eliminated production can be greatly increased. Infectious diseases are caused when the body is attacked by pathogenic microorganisms.
- 1.1.17 The spread of disease: Infectious diseases can be spread by:
- a) Direct contact between animals.
  - b) Germs in feed and water.
  - c) By feces and urine from sick animals.
  - d) By flies, mosquitoes, ticks, lice and fleas.
  - e) By dirty housing or shelters.
  - f) Young and old animals become infected more easily.
- 1.1.18 Preventing infectious diseases: Animals, like humans, must be clean in order to be healthy.
- a) The animal must be provided with clean feed, water, bedding and shelter.
  - b) Sick animals should be kept separate from the others.
  - c) Some diseases can be cured by drugs.
  - d) Vaccination can protect animals against some diseases.
  - e) The spread of disease can be avoided by good livestock management.
  - f) Keeping animals together increases the chance of disease spreading by contact
  - g) New livestock should be kept separate from the others for two weeks so they can be checked for signs of disease.
  - h) Avoid mixing herds. Try to keep herds separate at watering and feeding points.
  - i) Separate and isolate any animal which shows signs of disease.
  - j) Dead animals and waste should be disposed of properly.

#### 1.1.19 Preventing non-infectious diseases

The chronic non-infectious disease may not be recognized as a disease. The affected animals may not die but will not produce as much milk, meat or wool, or work as well as could be expected. If we continually look for ways of improving feed, water, mineral and vitamin supplies we will find the way to control the non-infectious diseases. This will lead to greater production of wool, meat and milk, draught animals will be stronger and more young will be produced. Poultry will produce more eggs and get fatter

## 1.2 Animal Diseases Notification Guidelines

1.2.1 Notification in UAE: It is the procedure by which a veterinary practitioner informs the veterinary authority of the occurrence of an outbreak of disease or infection that is required to be reported by law (Ministerial Decree 132 / 2004) , which will inform the Federal Headquarters of Diseases Notification (MOEW) or vice versa according to the federal provisions.

#### 1.2.2 Purpose of Notification and Surveillance

- a) To assist in successful disease control through the diagnosis and treatment of cases.
- b) To identify potentially exposed veterinary care and laboratory personnel (particularly zoonotic diseases) and to provide counseling.
- c) To identify sources of transmission (e.g., an infected animal or a contaminated animal product) and to prevent further transmission from such sources
- d) To raise the index of suspicion of any possible emerging disease event when no natural exposure source is identified.
- e) To provide early warning of possible outbreaks.

#### 1.2.3 Notification information should include the following details:

- a) Suspected Disease
- b) Nature of onset from owners or attendants
- c) No. of cases, species affected, approximate age and animal identification No.
- d) Main clinical findings including degree of severity morbidity and mortality rate
- e) Total number of herd
- f) Vaccination history
- g) Animal Movement Information
- h) History of newly introduced or replaced animals
- i) Whether the disease has been reported in the neighboring herds
- j) Submitting the filled Notification Form supplied by vet. authorities

### 1.3 Animal Destruction, Disposal and Premises Decontamination

1.3.1 Animal destruction: In certain situations of outbreak of a trans-boundary animal disease (TAD), or other serious disease it may be necessary to destroy a large number of animals if stamping-out policy is adopted for its control and eradication. This usually takes place after humane slaughter of the diseased or susceptible animals in the area, to assure death before disposal of carcasses commences. Firearms, humane killers (captive-bolt pistols) or other means are also used for the purpose. An experienced veterinarian should be supervising destruction operations. Where possible, the livestock owner must be informed and convinced as they may experience considerable distress. The policy regarding compensation for destroyed animals should be communicated clearly to owners before destruction is attempted. What animals should be slaughtered will depend on the disease in question and the epidemiological circumstances. In some non-emergency disease, e.g. bovine tuberculosis, slaughter of individual infected animal only may be necessary.

For emergency diseases, one of two options is usually selected:

a) If animals in the infected zone are not well controlled and there is a serious risk of further rapid spread of the disease or spillover to feral or wild animals, or if inadequate resources are available for surveillance and imposition of quarantine and movement controls, it may be expedient to slaughter all animals in the infected zone or in specific areas of the zone.

b) If animals are well contained on farms and holdings, and resources are available for surveillance and imposition of quarantine and movement controls, the best decision would probably be to slaughter only animals on known infected farms and dangerous-contact premises. This decision will depend on the mode of disease transmission; it will be different for diseases capable of airborne dissemination over distances and those requiring direct contact.

1.3.2 Operational Plan: Planning is essential to ensure that the task of destruction is carried out efficiently. An action plan should be drawn up in consultation with owners and appropriate officials. The procedures below should be followed. The veterinary officer should undertake the tasks listed below.

- a) Discuss the situation with affected farmers and village leaders to establish:
  - i. Farm layout, facilities and equipment.
  - ii. The number, species and location of animals to be destroyed.
  - iii. The destruction technique to be used.
  - iv. The time-frame for commencement and completion of animal destruction.
- b) Decide on the methods and facilities needed for safe, humane and efficient destruction of the animals.
- c) Advice the team leader of immediate resources needed to move and secure animals in preparation for destruction.
- d) Consult with the officer in charge (OIC) of the disposal team if different from the

destruction team, determine the disposal method and site; if necessary, identify centrally located carcass disposal sites as close as practicable to the site of destruction.

- e) Draw up a concise written plan for approval, including:
  - i. Destruction methods and site(s).
  - ii. Order of destruction.
  - iii. Personnel required.
  - iv. Facilities and equipment needed.
  - f) Make a diagram of the infected property (IP) or dangerous contact premises (DCP), including details of the destruction operation.
  - g) Make sure that there is a complete inventory of animals to be destroyed on the property, not delaying destruction because there has been no agreement on valuation; where possible, all animals should be valued before destruction.
  - h) Seek authority to destroy in terms of the law(s) pertaining to control of animal diseases when there is a delay in reaching agreement on valuation with the owner his/her agent; delay may endanger the success of the operation.
  - i) Request livestock owners to assemble, confine and restrain their animals the day before the commencement of operations, and ensure that animals not to be destroyed, including domestic pets, are confined well away from the destruction site.
  - j) Send a team into the surrounding countryside to assess the presence of free-roaming or unrestrained susceptible animals. Arrange for any necessary support services, such as police and army personnel, to be made available.

1.3.3 Duties of the destruction team leader: Before commencing destruction, the team leader (a trained veterinarian) should carry out the tasks below:

- a) Move animals to the centre of the infected premises or areas remote from susceptible and wild animals.
- b) Brief the destruction teams then supervise and coordinate their activities, and ensure the following:
  - i. Destruction takes place away from the public view if possible.
  - ii. Destruction facilities, methods and working conditions are consistent with personal safety.
  - iii. Destruction is humane and that no animal is removed for disposal until it is dead.
  - iv. Destruction teams receive adequate rest and breaks.
- c) Assure that other tasks, such as disinfection, can be started without delay; carcasses and the destruction area should be disinfected as soon as destruction is complete.

In selection of destruction site the following must be considered carefully:

- a) Facilities and equipment are available on site.
- b) Animal security.
- c) Proximity of the disposal site and ease of access.

- d) Safety of personnel.
- e) Acceptability to the owner/manager.
- f) Safe and secure means of transporting carcasses to the destruction site.
- g) Likelihood of damage to property and services.
- h) Protection from public view.

The order of destruction must be determined by the veterinary officer in charge of the operation and details concerning the affected animals, their direct contacts, and other susceptible animals in order of epidemiological importance must be clearly explained. In certain disease like foot-and-mouth disease if destruction is decided, highly susceptible groups and fractious and potentially dangerous animals, such as bulls, should be destroyed first.

**1.3.4 Destruction methods:** An appropriate method of destruction of various domestic species should be decided according to existing circumstances and nature of the disease in question. Usually, firearms (rifles and guns), captive-bolt pistols, pithing, gaseous agents (Carbon dioxide) or gaseous anaesthetic agent (halothane) are used for the purpose. Injectable agents e.g. barbiturates can be used in overdose for euthanasia by the intravenous route in large animals. Physical methods also employed like neck dislocation especially in poultry using burdizzo, bone cutters or manually. Rabid or suspect rabid animals and animals with Bovine Spongiform Encephalopathy (BSE) or Scrapie should not be shot through the head, as brain tissue is the best diagnostic specimen required for testing.

Example of destruction of some species:

- a) (Large animals): Cattle and camels: Under most circumstances large ruminants will be gathered into yards and shot. In extensive areas where 100 percent musters cannot be achieved, unmustered animals will be paddock shot after first mustering as many as possible. Captive-bolt pistols are most suitable when animals can be adequately restrained. Injectable agents may be most suitable for small number of calves.
- b) Goats: Using either a captive-bolt pistol or firearm, aim the weapon to the skull behind the horns. Aim in line with the animal's mouth. Kids may also be shot from the front, as for cattle. This method is not suitable for mature goats, as the brain is located well back in the skull compared to other livestock. Sodium pentobarbitone is also appropriate in case of newborn kids.

**1.3.5 Disposal of destructed animals:** This process is a basic part of an emergency animal disease control or eradication programs, particularly when a stamping-out policy is followed. Disposal should be completed soon after destruction, to minimize opportunities for carcasses decomposition and infectious material to disperse.

The disposal methods appropriate for the emergency animal disease most readily transmitted by fomites like foot-and-mouth disease, Newcastle disease, and avian influenza must be carefully and professionally followed. Less rigorous disposal methods may be appropriate for less readily transmitted disease and non-zoonotic disease. Carcasses and other items awaiting disposal should be guarded to prevent unauthorized access and to

prevent domestic pets, wild animals and birds and control of insects should be considered as well. If disposal is delayed, carcasses should be thoroughly sprayed with an approved disinfectant. Before disposal work starts, personnel should be fully briefed. The nature of the disease and any hygiene requirements associated with zoonotic disease should be explained on site. Respirators should be supplied to personnel when there is any risk to human from the organism involved or if large amounts of dust are generated.

**1.3.6 Selection of disposal method and site:** It is crucial to select a site which is well-protected from people and scavenging animals. On some occasions it may be necessary to mount a guard at the site. Disposal on the infected premises (or dangerous contact premises). Depending on local circumstances, burial may be the preferred method of disposal because it is quicker, cheaper, environmentally cleaner and easier to organize. Important factors to be considered are the amount of material for disposal, nearness to destruction site, accessibility to the site by heavy transport vehicles, Weather conditions, including prevailing winds. Disposal of animal carcasses and other infectious material may involve some adverse environmental consequences. It is important here to seek consultancy or advice from environmental agencies so as to minimize any potential hazardous impact. Disposal off the infected premises, where burial, cremation or rendering are not considered practical or difficult to carry out to the infected or dangerous contact premises, transference of the infectious material to alternative site must be considered. This may be necessary where site limitations, such as available space prevents on-site disposal. Transport should be in a leak-proof container, such as a large skip, covered with tough polyethylene covers and sealed at the top. It should not be overloaded half a meter or more (depending on distances to be travelled and temperature) should be left clear for expansion of carcasses. Carcasses should not be slashed before loading. Staff should carry supplies of an approved disinfectant and basic equipment to deal with minor spills during a journey. All vehicles must be cleaned and disinfected before leaving the premises and after unloading.

**1.3.7 Methods of disposal:** Mainly include burial, cremation, composting, incinerators, pit burning, and rendering.

a) Burial: Important considerations for selecting burial site are the accessibility for equipment to dig the burial pit and for the delivery of livestock, carcasses or other material to be buried. Environmental aspects, such as the distance to watercourses, bores and wells, heights of the waterable, proximity to houses or roads, permeability of soil and direction of prevailing wind (odour) are also important. In construction rocky areas should be avoided and use of diversion banks to prevent surface run-off from entering the pit is of utmost importance. Fencing may be necessary to exclude animals until the site safe for use.

b) Burial pit construction & dimensions of the burial pit will depend on the equipment used, site considerations and the volume of material to be buried. If bulldozer is used, for example, the pits should be no more than one blade width, about 3 meters to avoid having to move carcasses once they are in the pit. The length will be determined by the volume of

material buried. Excavators are useful in filling pits if soil stability does not permit trucks or other heavy equipment to operate close to the pit edge.

The base of the pit must be at least 1m above the waterable. Allow a fill capacity of about 1.5m<sup>3</sup> for each adult beast or 5 adult sheep. At least 2m depth of soil is required to cover carcasses to ground level. For example, a pit 3m wide and 5m deep filled with carcasses to within 2.5m of ground level can accommodate 5 adult cattle per linear meter or 25 sheep.

When closing the pit, surplus soil should be heaped over it as overfill. Poultry to be destroyed will normally be in a container the dimensions of which should be used as a guide to the volume of the pit required. Large animal carcasses must be opened by slashing the rumen (cattle) or the caeca (horses) to permit escape of gas. Cover the carcasses with soil, 400mm is suggested, and unbroken layer of slaked lime Ca(OH)<sub>2</sub> before filling is completed. Lime should not be placed directly on carcasses. Inspection of the burial site several months after pit closure is recommended. Utmost safety and hygiene of personnel working on the site should be maintained.

c) Cremation this should be considered only when burial is not possible. Examples of available convenient methods include incinerators and pit burning. Incinerators: Biological incinerators facilitate achieving safe and complete disposal with minimal pollution. Incinerators are usually only suited to disposal of small amounts of material. Special procedures must be followed for transportation of infected material from infected premises to the incinerator and disinfection of containers and vehicles.

d) Pit burning known as air curtain incineration is a technique for burning material in a pit, using fan-forced air. Pit burners are used by some local authorities to burn vegetable matter with high moisture content. Pit burners would be suitable for continuous operation on a relatively small scale and have the advantage of being transportable. They are especially suited to goats and fat sheep in situations where mechanical equipment is not available. A combination of burning and burial has been used successfully to dispose of small ruminants and possibly small numbers of cattle as well. After the trench had been dug, it was lined with old motor tires, on which the carcasses were placed. The carcasses were soaked with diesel and ignited with a small amount of petrol.

e) Rendering plants using a high-temperature batch-rendering process should be used. It involves grinding of the raw product, solvent extraction of lipids at about 100C for one hour and high temperature (160C) treatment of both carcass meal and tallow for at least a further 40 minutes. The end product of rendering must pass relevant microbiological tests before release.

f) Composting where there is a minor risk of fomite spread, composting of stable manure, feed, hay, litter and bedding is a possible alternative to burial or burning. Composting should be done in a secure area not accessible to susceptible animals.

1.3.8 Decontamination procedures: Decontamination is an important part of the control and eradication of major exotic or emergency livestock disease. It is applied by combination of physical and chemical processes. Identification of the disease agent is fundamental for designing an appropriate decontamination strategy which also requires adequate awareness and close cooperation between farmers and the involved personnel. The basic microbiological principles of isolation of the source of infection and decontamination of personnel, equipment, vehicles and sites are of major importance. Personal decontamination procedures, when properly carried out, permit the safe movement of personnel from property to property in the extensive surveillance activities form a vital part of any eradication campaign.

Preliminary cleaning is invariably needed before any chemical disinfectants are used; and is fundamental for achieving effective chemical decontamination. Generally, disinfectants and related chemicals (eg. pesticides) are classified into six general categories:

- a) Soaps and detergents.
- b) Oxidizing agents.
- c) Alkalies.
- d) Acids.
- e) Aldehydes.
- f) Insecticides.

All of these disinfectants are effective against a broad range of pathogens. Basic assessments of the epidemiological characteristics of the concerned pathogens spread is the most important initial information, then a plan can be devised to establish priorities for decontamination. Such a plan usually includes: Buildings with wooden, metallic components; Machinery of mostly metallic components; Pipe work of various types; Water tanks; Animal food storage areas; Sewage waste.

Depending on the disease agent involved, different decontamination procedures and disinfectant are likely to be used for different sites on a property. In case where the disease agent does not spread directly from animal to animal, e.g. blue tongue, a comprehensive decontamination of a property is not warranted. In contrast, some viruses, such as FMD virus, are relatively stable on inanimate objects and can be spread to remote animals on contaminated people, and equipment which necessitate comprehensive decontamination programmes. Preliminary cleaning work is invariable needed before any chemical disinfectants are used. The natural processes of time, dehydration warm temperature and sunlight will greatly assist the decontamination operation. Hot, dry, sunny, day will cause rapid natural inactivation of an agent like Newcastle disease virus, whereas cold, damp, overcast condition will assist its persistence. Every effort should be made to remove fat, grease ad organic dirt from all surfaces to be decontaminated. Hot water and steam are effective in cleaning many crack and crevices where pathogens are likely to linger. Choice of disinfectant depends on the method of application and how an adequate wet contact time is to be maintained. Choosing the most appropriate disinfectant is dependent on

the nature of the virus particles. Useful clue for predicting susceptibility are the presence or absence of lipid in the virus particles and the virus size. Effective disinfectants are often toxic for animal and human tissues as well and have to be used with care to avoid occupational injuries or health problems.

- 1.3.9 Selection of disinfectants: It should be remembered that in any large-scale decontamination of a farm or other infected premises, the cost of disinfectants will be relatively minor. Flame guns may be useful supplements for drying decontaminated surfaces but they are dangerous and the risk of fire and injury must always be considered. They are not recommended as a primary means of decontamination. In general, chemical names of disinfecting substances are used, because they are easily understood by all personnel with basic technical knowledge. Phenolics and quaternary ammonium compounds are very effective antibacterial but have limited effectiveness against some viruses.

Products effective for decontamination of viruses on the hand and skin are limited. Virkon is reported to have low toxicity and to be effective against members of all 17 viruses' families but has not been approved for use on skin. Alternatively citric acid or sodium carbonate may be added to washing water to induce antiviral conditions by lowering or raising the PH as required.

- 1.3.10 Personal decontamination: The aim of person decontamination is to remove safely any contamination of the body or clothing. A site designated for personal decontamination (PDS) should be arranged near the premises exit. The PDS should be placed in an area that can be easily and safely disinfected and sprayed with disinfectant applicable to the disease. Ideally, it should be on an impervious surface and include a building with a water supply and drainage. If there is no hard standing available, a 10 x 10m plastic ground cover can be used. Each person should have a change of clean clothes kept in plastic bags or in a caravan at the outer point of the area; there should be a store of clean coveralls in case of mishaps. Other more effective equipments for personal decontamination are emergency service shower vans.

- 1.3.11 Personal decontamination procedure: On arrival at the decontamination site, a disinfected solution safe for skin contact should be ready in buckets. Warm, soapy water is recommended for washing face, hair, skin, etc. Alternatively, the washing solution PH can be raised by adding sodium carbonate or lowered by adding citric acid to enhance antiviral action. The latter is recommended for decontamination from foot-and-mouth disease virus. Heavy-gauge plastic garbage bags are used for disposable items that can be buried or burnt on the site or for items to be removed from the site for further disinfections and cleaning. Industrial hard hat must be scrubbed and set aside. If a neck cloth is worn, it must be removed and soaked in disinfectant for example 1% Virkon for 10 minutes then placed in a plastic bag. Disposable gloves must be decontaminated before discarding; reusable gloves must be decontaminated before reuse. Hands must be washed in disinfectant and scrubbed. Using a sponge or low-pressure pump, wash the plastic overalls to remove gross material, Jackets, trousers, underclothes and boots must be removed and placed in disinfectant. Personnel returning to the site on a subsequent day can remove hats, gloves

and plastic overalls from the disinfectant and can remain on site. If personnel are not returning, the equipment should be placed in plastic bags and the outsides of the bags disinfected. Personnel can then walk across the area, treat the soles of footwear again, change into street shoes and leave directly, without re-exposure to contaminated areas. Plastic bags containing used overalls and other articles should be sealed, given a second wash in disinfectant and placed at the outer limit of the area for collection by courier. Personnel leaving IP or DCP for other duties must not have contact with susceptible stock.

- 1.3.12 Personnel decontamination in difficult circumstances: When a disease is suspected on a property, there will be visitors or private veterinarians present until the government veterinary officer arrives. If this is not practical, they have had contact with livestock or contaminated areas. Use of the following substances as personal disinfectant can be recommended where no other approved disinfected is available:
- a) Domestic washing soda (10 parts in 100 parts hot water).
  - b) Soap (or household detergent) and hot (60C) water for scrubbing.
  - c) Household concentrated chlorine bleaches (1 part in 3 parts water to give 2-3 percent available chlorine). There must be no visits to properties with livestock until the situation has been resolved. If the suspect property proves positive, people will be directed to present the vehicle for appropriate decontamination.

- 1.3.13 Property decontamination:  
In property decontamination the following regime is recommended:
- a) Inspection of the IP or DCP and prepare a map of the property.
  - b) Start a log book to record all events.
  - c) Indicate areas or sites requiring specific decontamination action; consult with the officers-in-charge of slaughter, disposal and epidemiology.
  - d) List procedure in chronological order to be undertaken in each area.
  - e) Estimate the duration of the decontamination program.
  - f) Seek approval from the veterinary authority operations manager for the proposed program.
  - g) Implement the agreed decontamination plan, maintaining liaison with the IP operation manager and submitting a daily progress report. The composition of a typical property decontamination program is as follows:
    - i. Presumptive identification of the disease agent.
    - ii. Property assessment
    - iii. Preliminary disinfection
    - iv. First cleaning
    - v. First disinfection
    - vi. First inspection
    - vii. Second disinfection
    - viii. Final inspection

Continuous close liaison with the owner/manager is essential to achieve an effective program.

## Chapter (2) Viral & Prion Diseases

- 2.1 Foot and Mouth Disease
- 2.2 Peste des Petits Ruminants (PPR)
- 2.3 Lumpy Skin disease(LSD)
- 2.4 Rabies
- 2.5 Rift valley fever(RVF)
- 2.6 West Nile fever(WNF)
- 2.7 African horse sickness(AHS)
- 2.8 Blue tongue (BT)
- 2.9 Bovine Ephemeral Fever (BEF)
- 2.10 Crimean – Congo Hemorrhagic Fever(CCHF)
- 2.11 Equine infectious anemia(EIA)
- 2.12 Equine Viral Arteritis (EVA)
- 2.13 ORF
- 2.14 Sheep and goat pox
- 2.15 Infectious Bovine Rhinotracheitis (IBR)
- 2.16 Camel pox
- 2.17 Mad Cow Disease

### 2.1 Foot and Mouth Disease

2.1.1 Definition: (synonyms - Aphthous Fever) Foot and mouth disease (FMD) is a highly contagious, notifiable disease of cattle and other cloven hooved animals. It is characterized by the formation of vesicles and erosions in the buccal cavity and on the udder and feet.

2.1.2 Epidemiology: FMD caused by any of various viruses of the *Genus Aphthovirus* family Picornaviridae and, characterized by fever and the formation of vesicles around the mouth, udder and hooves around coronet & inter digital spaces. There are seven principal antigenic types, known as serotypes, including A, O, C, Asia1, SAT1, SAT2, and SAT3 . Each serotype might include several subtypes. There are number of strains identified under each subtype. There is no cross immunity or protection between the known serotypes or subtypes.

Clinical signs of infection begin 24 to 48 hours after exposure and may last several days fever of 104-107 F i.e.: 40 - 41.66 C, anorexia , lameness ,feet, vesicles, lingual epithelial erosion, lesions, Mortality may reach up to 5% in adult and it is higher in young animals, due to myocardial necrosis. Hosts could be described as (Reservoir, Maintenance, Amplifier, Indicator and Final).

Cattle, pig, sheep, goats and deer and all cloven hooved animals such as wild ruminants like antelope, deer and elk are susceptible. (Maintenance Host: Sheep, Amplifier Host: swine, Indicator Host: cattle). Once an outbreak begins, most transmission is by aerosol from one infected animal to another. Sheep often do not appear very ill when infected with foot-and-mouth disease and have been accused of much of the transmission as apparently unaffected sheep are transported from one area to another, carrying the virus with them

(and so have been called the maintenance host). When these sheep mix with cattle, the cattle develop severe clinical signs of slobbering and lameness, often raising the red flag of infection (and so the term indicator host). Aerosol from infected animal, direct and indirect contact, consumption of contaminated feed, fomites, artificial insemination, contaminated biological and possibly migratory birds have all been incriminated in the spread of FMD. Recovered cattle and sheep can remain carriers for prolonged period of time.

2.1.3 Diagnosis: The signs and gross lesions are suggestive. Gross lesions are readily observable clinically as vesicles, erosions, ulcerations in tongue, snout, and feet. Differential diagnosis: FMD should be differentiated from vesicular stomatitis, foot rot, chemical and thermal burns, photosensitization. In cattle the disease resembles rinderpest, IBR, BVD and MCF. In sheep and goat FMD can be confused with ORF and BT.



Fig.1 FMD erosions on gum of cattle

The preferred samples for laboratory diagnosis include vesicular fluid, epithelium from unruptured or freshly ruptured vesicles, esophageal pharyngeal samples, blood (serum), and myocardial tissue from young dead animals. Laboratory diagnosis of FMD is by virus isolation, detection of viral antigens or nucleic acids and serological tests like ELISA.

2.1.4 Prevention and Control: All biosecurity steps are needed for control of FMD. Cull all infected cases and consider them as source of infection & No Treatment. Any trial to treat cases is not recommended. It might amplify & spread the virus. Immediate halting of all movement of animals is important. This includes animal products in the affected area. Isolate all infected from susceptible in contact animals. Disinfection of all involved premises, vehicles and personal equipment is part biosecurity steps. Effective disinfectants for FMDV include sodium hydroxide 2%, sodium carbonate 4% citric acid 0.2% and Virkon-S. Whether or not a "ring" vaccination would be undertaken will depend on the nature of the outbreak. Mass vaccination of susceptible livestock is recommended using inactivated vaccines composed of the relevant serotypes and subtypes of the virus circulating in the country or region. Vaccination should be done at least twice yearly.

## 2.2 Peste des Petits Ruminants (PPR)

- 2.2.1 Definition: Peste des petits ruminants (PPR). It is a notifiable severe fast-spreading highly contagious viral disease. It affects small ruminants mainly sheep & goats and wild ruminants such as deer and gazelles. It is characterized by the sudden onset of depression, fever, discharges from the eyes and nose, sores in the mouth (stomatitis), disturbed breathing and cough, foul-smelling diarrhea & death.
- 2.2.2 Epidemiology: The causative agent is a virus (PPRV), which is similar to the closely related Rinderpest virus. PPRV is a species of Genus: *Morbillivirus*, Family *Paramyxoviridae*. Sources of virus are all secretions and excretions of incubating and sick animal's tears, nasal discharge and coughed secretions. Incubation period is 3-10 days.

Clinical signs are variable from peracute, acute, subacute to chronic forms, highly influenced by the species, as well as the animal's immunity to PPRV and its breed. Peracute cases are frequent in goats with high mortality showing high fever and depression to death. The clinical picture of the acute form is characterized by sudden rise in body temperature (40 - 41°C) with effects on the general state: restlessness, dull coat, dry muzzle, depression of appetite. Serous nasal discharge becoming mucopurulent and resulting, at times, in a profuse catarrhal exudate which crusts over and occludes the nostrils leading to respiratory distress. There are small areas of necrosis on the visible nasal mucous membrane. There is congestion of conjunctiva, crusting on the medial canthus and sometimes profuse catarrhal conjunctivitis. Serous ocular and nasal discharges may complicate cases to pneumonia and diarrhea by direct contact between animals. There is no carrier state in PPR.

Seasonal variations: more frequent outbreaks during the rainy season or the dry cold season. Necrotic stomatitis with halitosis is common. Severe non hemorrhagic diarrhea is not uncommon. Bronchopneumonia evidenced by coughing is a common feature abortion, dehydration, emaciation, dyspnea, hypothermia and death within 5-10 days Subacute and chronic forms are frequent in some areas because of breed susceptibility 10-15 days development with inconsistent symptoms & pneumopathy. Mortality rate is 50-80% (susceptible population). Emaciation, conjunctivitis, erosive stomatitis involving the inside of the lower lips and adjacent gum near the commissures & the tongue free portion are common clinical findings.



Fig. 2 PPR Distribution in Asia and Africa

- 2.2.3 Diagnosis: PPR is characterized by serous ocular and nasal discharges, pneumonia and diarrhea. Morbidity rate is up to 90% (susceptible population). Lesions are seen mainly in the hard palate, pharynx and upper third of the esophagus in severe cases. Rumen, reticulum and omasum rarely show lesions. Small streaks of hemorrhages and sometimes erosions are seen in the first portion of the duodenum and the terminal ileum. Extensive necrosis and sometimes severe ulceration of Peyer's patches Congestion around the ileo-caecal valve, at the caeco- colic junction and in the rectum. 'Zebra stripes' of congestion are seen in the posterior part of the colon.

Small erosions and petechiae are encountered on the nasal mucosa, turbinates, larynx and trachea. Bronchopneumonia is a constant lesion. Differential diagnosis includes Rinderpest, Contagious caprine pleuropneumonia, FMD, BT, Pasteurellosis, ORF, Heartwater, Coccidiosis and Mineral poisoning.

Laboratory tests like PCR or serological tests like ELISA are used to confirm diagnosis.

- 2.2.4 Prevention and Control: Attenuated PPR vaccine is capable of providing protection for at least 1.5 years. Disease outbreaks are controlled by a combination of quarantine and movement control, culling of infected animals, cleaning and disinfection of infected premises.

## 2.3 Lumpy Skin Disease

- 2.3.1 Definition: (synonyms - Neethling) Lumpy skin disease (LSD) is a notifiable viral disease with significant morbidity in cattle. Asian water buffalo & Oryx are susceptible.
- 2.3.2 Epidemiology: LSD is caused by LSDV from Genus: *Capripoxvirus*, Family: *Poxviridae*. It is closely related antigenically to sheep and goat poxviruses. Transmission of LSDV is primarily by biting mosquitoes and flies. Epidemics occur in the rainy seasons. Direct contact is also a minor source of infection. LSDV can be present in cutaneous lesions, saliva, nasal discharge, milk and semen. The virus can survive in dessicated crusts for up to 35 days. There is no carrier state. The spread of the virus is often related to movement of cattle.

Fever is the initial sign. It is usually followed within two days by the development of nodules on the skin and mucous membranes. These nodules vary from 1 cm to 7 cm and penetrate the full thickness of the skin. They are particularly common on the head, neck, udder, genitalia, perineum and legs. Although the nodules may exude serum initially, they develop a characteristic inverted conical zone of necrosis, which penetrates the epidermis and dermis, subcutaneous tissue, and sometimes the underlying muscle. Nodules on the mucous membranes and udder ulcerate rapidly. The superficial lymph nodes become enlarged and edematous. Nodules can also occur in the gastrointestinal tract, trachea and lungs; the latter may result in primary or secondary pneumonia. Feed intake decreases in affected cattle this leads to emaciation, milky yield drop. Mortality rate is generally low. Losses occur from drop in milk production, abortion, infertility, loss of condition and damaged hides Rhinitis, conjunctivitis and keratitis can also be seen; ocular and nasal discharges are

initially serous but become mucopurulent. Inflammation and necrosis of the tendons, or severe edema of the brisket and legs, can result in lameness. Secondary bacterial infections can cause permanent damage to the tendons, joints, teats and mammary gland. Abortions and temporary or permanent sterility may occur in both bulls and cows. A few animals die, but the majorities slowly recover. Recovery can take several months, and some skin lesions may take a year or two to resolve. Deep holes or scars are often left in the skin. Morbidity rate varies between 5 and 45% Mortality rate up to 10%.

- 2.3.3 Diagnosis: LSD clinical diagnosis depends upon clinical signs with characteristic skin nodules. Differential diagnosis could be confused with many diseases like Bovine herpes mammillitis, Bovine papular stomatitis, Pseudocowpox, Vaccinia virus and cowpox virus, Insect or tick bites, *Hypoderma bovis* infection, cutaneous tuberculosis and urticaria. Laboratory diagnosis is by virus isolation, electron microscopy and serological tests like ELISA.
- 2.3.4 Prevention and Control: No specific treatment. Strong antibiotic therapy may avoid secondary infection Vector control in ships and aircraft is highly recommended Free countries: import restrictions on livestock, carcasses, hides, skins and semen Infected countries:
- strict quarantine to avoid introduction of infected animals into safe herds
  - in cases of outbreaks, isolation and prohibition of animal movements
  - slaughtering of all sick and infected animals (as far as possible)
  - proper disposal of dead animals (e.g. incineration)
  - cleaning and disinfection of premises and implements
  - vector control in premises and on animals

Homologous live attenuated virus vaccine: Neethling strain: immunity conferred lasts up to 3 years.

## 2.4 Rabies

- 2.4.1 Definition: Rabies is a notifiable zoonotic viral disease that affects the central nervous system of warm-blooded animals, including humans. The disease has a long incubation period (six months) and symptoms may take several weeks to appear after infection. However, once symptoms appear, rabies is always fatal in animals.
- 2.4.2 Epidemiology: The rabies virus is from Genus: *lyssavirus*, Family: *Rhabdoviridae*. The rabies virus is present almost in all continents; the occurrence of rabies in domestic dogs posing a threat to humans that is of major concern in several developing and in-transition countries. Rabies is transmitted through the saliva of an infected animal. Infection occurs primarily via bite wounds, or infected saliva entering an open cut or wound or mucous membrane, such as those in the mouth, nasal cavity or eyes. Infection through inhalation of the virus has been documented, for example, in the environment of a bat cave. The virus will generally remain at the entry site for a period of time before travelling along the nerves to the brain. In the brain, the virus multiplies quickly, resulting in clinical signs. The virus then moves

from the brain along nerves to the salivary glands. The period of time before clinical signs appear in an infected animal can vary depending on the strain of virus and entry point. It is thus important to realise that the disease can be transmitted via the saliva of an infected animal to other animals and humans before the onset of clinical signs of the disease in the infected animal. Infection does not occur by consumption of meat from a rabid animal.

### Clinical signs:

There are two forms of rabies:

- Furious form of Rabies in which animals may be anxious, highly excitable and/or aggressive with intermittent periods of depression. With the loss of natural caution and fear of other animals and humans, animals with this form of rabies may demonstrate sudden behavior changes, and attack without provocation. As the disease progresses, muscular weakness, incoordination and seizures are common. Death ends progressive paralysis.



Fig.3 Furious Form of Rabies

- Dumb Form of Rabies in which animal is depressed or unusually docile. The animal will often have paralysis, generally of the face, throat and neck, causing abnormal facial expressions, drooling and inability to swallow. Paralysis may affect the body, first affecting the hind legs. The paralysis progresses rapidly to the whole body with subsequent coma and death.
- 2.4.3 Diagnosis: The disease may be suspected based on clinical signs, however, laboratory tests are required to confirm the diagnosis. Samples taken from suspect live or dead animals must be collected in highly biosafety careful precautions and sent with careful biosecurity measures to competent laboratories for diagnosis (saliva from live animal particularly biting dogs for detection of Rabies virus antigen or brain from dead animal for histopathology).
- 2.4.4 Prevention and Control: Veterinarians, animal control and wildlife officers should obtain protection through pre exposure vaccination. Abattoir personnel, particularly in endemic

areas, must take preventive actions to prevent infection from saliva, salivary gland and nervous tissue of infected animals. In countries where the disease is endemic, measures are implemented to address and reduce the risk of infection in susceptible populations (wildlife, stray and domestic animals) and create a buffer between the animal source of the disease and humans. Surveillance and reporting of suspected cases of rabies in animals. Points that should be considered are: Research in disease dynamics, vaccines and effective delivery mechanisms for target populations. Control of stray dogs and cats and vaccination programs is part of joint work between local and federal authorities in UAE.

## 2.5 Rift Valley Fever

2.5.1 Definition: Rift Valley fever (RVF) is a notifiable zoonotic, (arthropode borne) or Arbo viral disease in ruminants. This disease is characterized by high mortality rates in young animals and abortion in pregnant ruminants.

2.5.2 Epidemiology: Rift Valley fever is endemic in Africa. It appeared first outside Africa in 2000, when outbreaks were reported in Saudi Arabia and Yemen.

RVFV is from *Genus Phlebovirus.Family Flaviviridae*. RVF is transmitted by mosquitoes and is usually amplified in ruminant hosts. The virus appears to survive in the dried eggs of Aedes mosquitoes so epidemics occur after heavy rainfalls because infected mosquito eggs hatch, and large numbers of susceptible animals are present. The incubation period is 3 days in sheep, cattle, goats and camels. In newborn lambs, it is 12 to 36 hours clinical signs start by sudden onset of abortion storms and up to 100 percent mortality in lambs under five to six days old. High fever, lymphadenitis, nasal and ocular discharges in mature animals .Profuse fetid diarrhoea (often haemorrhagic).Vomiting, abdominal colic and severe prostration, dysgalactia, jaundice .Epizootic period of 8-16 weeks.

2.5.3 Diagnosis: PM findings like liver enlargement, necrosis , congestion, then later a bronze to yellow colour Petechial and ecchymotic haemorrhages throughout the carcass severe haemorrhagic gastroenteritis generalized lymphadenopathy pulmonary oedema and emphysema similar pathology in foetus, with autolysis. Differential diagnosis could be confused with diseases like BT mouth and foot lesions (coronitis) Heartwater - serous fluids in body cavities; neurological signs ,Ephemeral fever, recumbency and rapid recovery Toxoplasmosis, leptospirosis, brucellosis, Q fever, salmonellosis - basic diagnostic methods for differentiation, PPR - high mortality in lambs and FMD - neonatal mortality and abortions in small ruminants. Laboratory confirmation is done by: 1. RVF antigen detection using AGD test or virus isolation in mice/hamsters/tissue culture or RT-PCR identification of RVF virus or capture ELISA test or immunochemical staining of fixed tissues 2. RVF antibody detection using ELISA tests for IgM/IgG or Indirect immune fluorescent / peroxidase tests or Microtitre virus-serum neutralization tests or Plaque reduction assays or indirect haemagglutination tests.

2.5.4 Prevention and Control: The three essential prerequisites for an epidemic to occur are a susceptible livestock population, a massive buildup in the populations of vector

mosquitoes and the presence of the RVFV. Assuming the continuing presence or at least the close proximity of the virus in regions where the disease has occurred previously, the first two factors become the key to early forecasting of likely RVF activity. To control RVF use 3 tracks: RVF Vector control, Movement controls & Preventive vaccination. Attenuated live vaccine (Smithburn vaccine) is widely used in endemic regions, but it induces abortion in animals and it is pathogenic for human. Safe inactivated vaccines are available but require two inoculations and annual revaccination. Public Health Importance: Human can be infected by aerosol or direct contact with infected tissues.

## 2.6 West Nile Fever

2.6.1 Definition: West Nile Fever (WNF) is a notifiable zoonotic mosquito borne viral disease in species of mammals, birds and reptiles. Most clinical cases occur in humans and horses.

2.6.2 Epidemiology: WNFV is a virus from *Genus: Flavivirus: Family Flaviviridae*. Birds are the primary vertebrate reservoir hosts of WNFV. Birds known to shed WNFV in oral secretions and/or feces include domesticated geese. Some bird species are more susceptible especially the crow family. Finding of dead crows signals the presence of WNF. WNFV is an arbovirus transmitted by mosquitoes. Most horses are infected asymptotically. In clinical cases, the illness is characterized by anorexia, depression and neurological signs, which may include ataxia, weakness or paralysis of one or more limbs, teeth grinding, aimless wandering, convulsions and/or circling. Tremors of the face and neck muscles are very common. Some animals have cranial nerve deficits, particularly weakness or paralysis of the face and tongue, which may lead to difficulty in swallowing. Attitudinal changes including somnolence, apprehension, hyperesthesia or periods of hyperexcitability are also common. Some horses with severe depression and facial paralysis may hang their heads; this can result in severe facial edema. Coma, impaired vision and head pressing can be seen, but tend to be less common than in cases of encephalitis. A few clinical cases have been reported in ruminants. Sheep & deer have had neurological signs that resembled the syndrome in horses. In many cases, these were the first signs observed in the animal. Horizontal transmission occurs in some avian species.

2.6.3 Diagnosis: Postmortem findings in infected bird hemorrhages of the brain, splenomegaly, pancreatitis meningoencephalitis and myocarditis, lesions are uncommon in horses. Isolation of WNFV is definitive in all species but this is time-consuming and requires level-3 biosafety containment. The identity of WNFV can be confirmed by immunofluorescence or RT-PCR. WNFV is difficult to recover from live horses because the viremia is usually low-level and short-lived.

2.6.4 Prevention and Control: To control WNF use 3 tracks: RVF Vector control, Movement controls of hosts & Preventive vaccination. No specific treatment is available, but animals may recover on their own if they are given supportive care. Supportive treatment has the goal of reducing inflammation in the CNS, preventing self-inflicted injuries and adverse effects from recumbency, and providing supportive nutrition and fluids. Therapy is empiric, and similar to the treatment of other causes of viral encephalomyelitis. Mild cases

have sometimes recovered without treatment. West Nile encephalitis occasionally occurs in vaccinated horses, and mosquito control measures should not be neglected. Topical repellents should be used on horses and other susceptible animals during the mosquito season. Repellents should be approved for the species; products that are safe in one species (including humans) can sometimes be toxic in others.

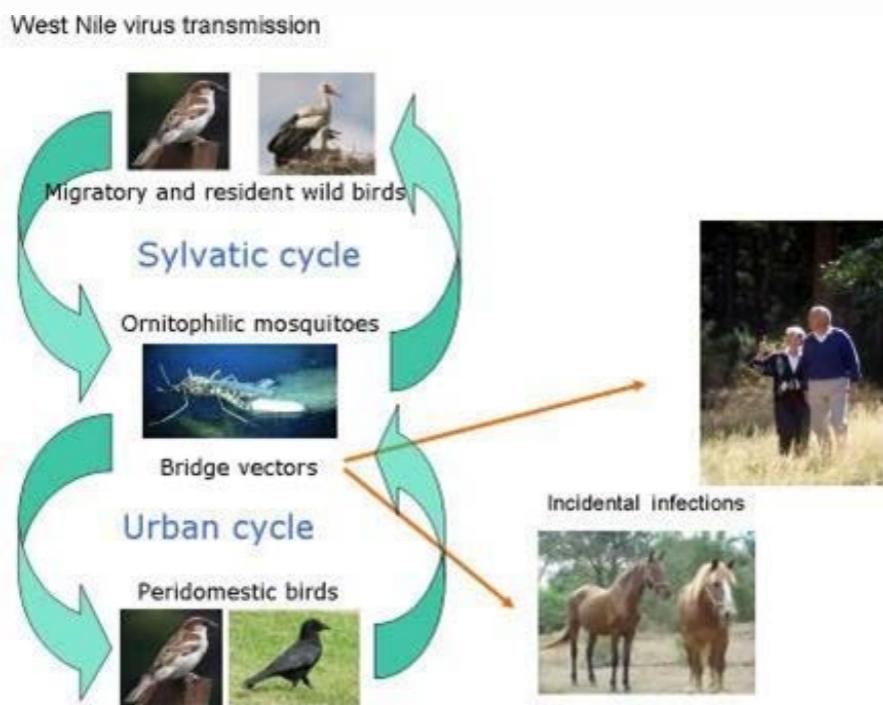


Fig. 4 West Nile Fever Virus Cycle

## 2.7 African Horse Sickness

- 2.7.1 Definition: African horse sickness (AHS) is a notifiable, serious, often fatal, arthropod-borne viral disease of horses and mules. The mortality rate can be as high as 95% in some forms of this disease. Asymptomatic or mild infections can occur in horses, as well as zebras and donkeys, previously infected with a different serotype of the virus. Infected animals or vectors may carry the virus into AHS-free regions.
- 2.7.2 Epidemiology: AHSV belongs to Genus: *Orbivirus* Family: *Reoviridae*. The AHSV can infect horses, donkeys, mules, zebras, camels and dogs. A midge *Culicoides imicola*, is the principal vector for AHS. Biting flies and mosquitoes may be able to transmit the virus mechanically. There are nine serotypes of AHSV. Serotype 9 has been responsible for the majority of AHS outbreaks outside Africa. In natural infections, the incubation period appears to be approximately 3 to 5 days for the pulmonary form, 7 - 14 days for the cardiac form, 5 - 7 days for the mixed form and 5 - 14 days for horse-sickness fever.

### Clinical signs:

The peracute or pulmonary form of AHS usually begins with an acute fever, followed by the sudden onset of severe respiratory distress. Infected animals often stand with forelegs spread, head extended, and nostrils fully dilated. Other clinical signs may include tachypnea,

forced expiration, profuse sweating, spasmodic coughing, and a frothy serofibrinous nasal exudate. Dyspnea usually progresses rapidly, and the animal often dies within a few hours after the respiratory signs appear.

The subacute: edematous or cardiac form of AHS usually begins with a fever that lasts for 3 - 6 days. Shortly before the fever starts to subside; edematous swellings appear in the supraorbital fossae and eyelids. These swellings later spread to involve the cheeks, lips, tongue, intermandibular space, laryngeal region, and sometimes the neck, shoulders and chest. It is important to note that no edema of the lower legs is observed. Other clinical signs, usually seen in the terminal stages of the disease, can include severe depression, colic, ecchymoses on the ventral surface of the tongue, and petechiae in the conjunctivae. Death often occurs from cardiac failure. The mortality rate is usually 50% to 95% in the previous forms. If the animal recovers, the swellings gradually subside over the next 3 to 8 days. The mixed form: of AHS, symptoms of both the pulmonary and cardiac forms are seen. Horse sickness fever form is mild and the animal spontaneously recovers.

- 2.7.3 Diagnosis: AHS should be suspected in animals with typical symptoms of the cardiac, pulmonary or mixed forms of the disease. The differential diagnosis includes equine viral arteritis, equine infectious anemia, Hendra virus infection, purpura hemorrhagica and equine piroplasmosis. Toxins and anthrax, as well as and other causes of sudden death and severe respiratory distress, should also be considered. In laboratory can be diagnosed by viral isolation. AHSV antigens can be detected with (ELISA). PCR technique is used to detect viral RNA.
- 2.7.4 Prevention and Control: Vector control by insect repellents and targeted applications of insecticides or larvicides may also be useful. All biosecurity measures are needed. Attenuated vaccine is used in endemic countries.

## 2.8 Bluetongue

- 2.8.1 Definition: Bluetongue (BT) is an insect-borne viral notifiable disease of ruminants. Among domestic animals, clinical disease occurs most often in sheep,, and can result in significant morbidity.
- 2.8.2 Epidemiology: BT results from infection by the BTV which is a member of the Genus: *Orbivirus*, family: *Reoviridae*. Twenty four serotypes have been identified worldwide. BTV is closely related to epizootic hemorrhagic disease EHD Most of domesticated and wild ruminants are susceptible. Affected sheep may have erosions and ulcerations on the mucous membranes, dyspnea, or lameness from muscle necrosis and inflammation of the coronary band. Some sheep may slough their hooves, and surviving animals can lose part or all of their wool. Some strains of the virus can result in mortality rates as high as 70% in highly susceptible sheep. The BTV has recently expanded its geographic range. BTV is transmitted by biting midges in the Genus: *Culicoides*. Cattle are the major amplifying host due to their prolonged viremia and the feeding preferences of many *Culicoides* species.

### Clinical signs:

In sheep, the clinical signs may include fever, excessive salivation, depression, dyspnea and panting. Initially, animals have a clear nasal discharge; later, the discharge becomes mucopurulent and dries to a crust around the nostrils. The muzzle, lips and ears are hyperemic, and the lips and tongue may be very swollen. The tongue is occasionally cyanotic and protrudes from the mouth. The head and ears may also be edematous.

Erosions and ulcerations are often found in the mouth; these lesions may become extensive and the mucous membranes may become necrotic and slough. The coronary bands on the hooves are often hyperemic and the hooves painful; lameness is common and animals may slough their hooves if they are driven. Pregnant ewes may abort their fetuses, or give birth to "dummy" lambs. Additional clinical signs can include torticollis, vomiting, pneumonia or conjunctivitis. The death rate varies with the strain of virus. Three or four weeks after recovery, some surviving sheep can lose some or all of their wool. The severity of BT disease varies with the breed of sheep, virus strain and environmental stresses. BT is seen often in sheep, occasionally in goats, and rarely in cattle. BT is not a contagious disease; however, the virus can be spread mechanically on surgical equipment and needles. BTV can be found in semen and venereal transmission from bulls is possible, but does not appear to be a major route of infection. In sheep, the incubation period is usually 5 - 10 days. Cattle can become viremic starting at four days post-infection, but rarely develop symptoms. Animals are usually infectious to the insect vector for several weeks. The vast majority of infections with are clinically inapparent. In a percentage of infected sheep and occasionally other ruminants, more severe disease can occur. Cattle that have clinically apparent disease may develop severe breaks in the hooves several weeks after infection; such breaks are usually followed by foot rot. In cattle, up to 5% of the animals may become ill, but deaths are rare. In some animals, lameness and poor condition can persist for some time. Infections in goats are usually subclinical, and similar to disease in cattle. Although many infections in wild ruminants are inapparent, severe disease can occur in some species. In pronghorn antelope and whitetail deer, the most common symptoms are hemorrhages and sudden death. Bluetongue is usually severe in whitetail deer and pronghorn antelope, with a morbidity rate as high as 100% and a mortality rate of 80-90%.

**Postmortem findings:** In sheep, the face and ears are often edematous. A dry, crusty exudate may be seen on the nostrils. The coronary bands of the hooves are often hyperemic; petechial or ecchymotic hemorrhages may be present and extend down the horn. Petechiae, ulcers and erosions are common in the oral cavity, particularly on the tongue and dental pad, and the oral mucous membranes may be necrotic or cyanotic. The nasal mucosa and pharynx may be edematous or cyanotic, and the trachea hyperemic and congested. Froth is sometimes seen in the trachea, and fluid may be found in the thoracic cavity. Hyperemia and occasional erosions may be seen in the reticulum and omasum. Petechiae, ecchymoses and necrotic foci may be found in the heart. In some cases, hyperemia, hemorrhages and edema are found throughout the internal organs. Hemorrhage at the base of the pulmonary artery is particularly characteristic of this disease. In addition, the skeletal muscles may have focal hemorrhages or necrosis, and the intermuscular fascial planes may be expanded by edema fluid. In deer, the most prominent lesions are widespread petechial to ecchymotic hemorrhages. More chronically infected deer may have ulcers and necrotic debris in the oral cavity. They may also have lesions on the hooves, including severe fissures or sloughing.



Fig. 5 Blue Tongue Lesion

- 2.8.3 **Diagnosis:** Bluetongue should be suspected when typical clinical signs are seen during seasons when insects are active. A recent history of wasting and foot rot in the herd supports the diagnosis. The differential diagnosis includes foot-and-mouth disease, vesicular stomatitis, PPR, plant photosensitization, MCF, IBR, parainfluenza- 3 infection, ORF, sheep pox, foot rot and *Oestrus ovis* infestation. In cattle and deer, EHD can also result in similar symptoms. BT can be diagnosed by isolating the virus in embryonated chicken eggs or cell cultures. Bluetongue viruses can be identified to the serogroup level by immunofluorescence, antigen-capture ELISA or the Immunospot test, as well as other techniques. These viruses can be serotyped with virus neutralization tests. PCR techniques are widely used to identify BTV in clinical samples. These techniques allow for rapid diagnosis and can identify the serogroup and serotype. Serology is sometimes used for diagnosis.
- 2.8.4 **Prevention and Control:** BTV is transmitted by insect vectors and is not contagious by casual contact. Disinfectants cannot prevent the virus from being transmitted between animals; however, where disinfection is warranted, sodium hypochlorite or 3% sodium hydroxide are effective. Insect control is important in limiting the spread of the disease; synthetic pyrethroids or organophosphates are effective against *Culicoides*. Moving animals into barns in the evening can also reduce the risk of infection. Although the BTV does not infect equids, horses and stables should be considered in any control scheme, as *Culicoides* can feed on horses, and manure piles are ideal breeding sites for these vectors. In countries where BT is endemic, vaccines are also used for control. Attenuated vaccines, which are available in countries including the U.S., are generally serotype specific. Multivalent live vaccines are also sold in South Africa. During the vector season, the viruses in attenuated vaccines can be transmitted to unvaccinated animals, and could reassort with field strains, resulting in new viral strains. In addition, vaccines can cause fetal malformations in pregnant ewes.

## 2.9 Bovine Ephemeral Fever

- 2.9.1 Definition: (Synonyms - Three Day Scikness) Bovine ephemeral fever (BEF) is an economically important viral disease of cattle and water buffalo. Its impact includes lost production, decreased milk yield, loss of condition, abortion, temporary infertility in bulls, and prolonged recovery in some animals as well as trade restrictions
- 2.9.2 Epidemiology: It occurs in Africa, Australia, Asia and the Middle East, often in sweeping epizootics. Although mortality is usually low, cattle in good condition are affected more severely; mortality rates as high as 30% have been reported in very fat cattle. BEF is caused by the BEFV, a member of the Genus: *Ephemerovirus* in the family: *Rhabdoviridae*. There is only one serotype. BEF appears to be transmitted by arthropods. The vector or vectors are not known, mosquitoes & biting midges are involved. The natural incubation period is 1-10 days. BEF clinical signs can be either mild or severe in cattle, with the most severe cases occurring in bulls and high- producing cows. Subclinical infections are also seen. The symptoms vary in individual animals, but the classic course begins with a fever, which is often biphasic, triphasic or polyphasic. The temperature peaks typically occur 12 to 18 hours apart. During the first fever spike, milk production in lactating cows often drops dramatically, but other clinical signs tend to be mild. Some animals may be depressed, stiff or reluctant to move. On the second day of illness, which may coincide with a second elevation in temperature, the symptoms are more severe. Animals usually become inappetent and depressed, with an increased heart rate, tachypnea, and serous or mucoid discharges from the nose. Profuse salivation, muscle twitching, waves of shivering or a watery ocular discharge may also be seen. Some animals develop submandibular or periorbital edema, or patchy edema on the head. Shifting lameness, stiffness and joint pain are common; the joints may or may not be swollen. The lameness can be severe enough to mimic a fracture or dislocation. Pulmonary emphysema and rales may be found in severe cases. Many animals, particularly cows in good condition and bulls, become recumbent for eight hours to days. Most animals lie in sternal recumbency, but in severe cases, animals may become laterally recumbent. Some animals temporarily lose their reflexes and are unable to rise. Recumbent animals may be bloated, have ruminal stasis, or lose their swallowing reflex. These clinical signs can be exacerbated by severe environmental stress or forced exercise. Most animals begin to improve a day or two after the first symptoms appear, and recover completely within another one to two days. Lactating cows and animals in good condition are usually affected more severely and may take up to a week to recover. Generally, animals lose condition rapidly during the illness, and regain their weight only slowly. Complications are uncommon but can include temporary or (rarely) permanent paralysis, as well as gait impairment, aspiration pneumonia, emphysema, mastitis, and the subcutaneous accumulation of air along the back. Many of these complications may be the result of trauma or complications of recumbency. Temporary infertility (up to 6 months) can develop in bulls, and abortions can occur in cows. Permanent infertility is rare. In recovered animals, milk production is decreased by 10-15% for the rest of the lactation, but usually returns to normal after subsequent pregnancies. Cows that become ill late in lactation may not return to production.

Death is uncommon, but may occur during either the febrile or the convalescent stage. Deaths are usually the result of secondary complications such as pneumonia or trauma. Water buffalo have similar symptoms, but the disease is usually milder. Experimentally infected sheep remain asymptomatic. The morbidity rate is highly variable, and can be as high as 80% or as low as 1-10%. Morbidity varies with the age and condition of the animal, as well as any immunity it may have. The clinical signs are usually more severe in adults than calves; symptomatic infections are rare in cattle less than 6 months of age, even when they have no maternal antibodies. Bulls, animals in good condition and high-producing cows are more severely affected. The mortality rate is 1-2% in most outbreaks, but it can be as high as 30% in very fat cattle.

- 2.9.3 Diagnosis: BEF is usually diagnosed clinically during outbreaks in endemic areas. This disease should be suspected in cattle herds that develop severe but transient symptoms including fever, lameness, temporary paralysis or recumbency. The mortality rate tends to be surprisingly low for the severity of the signs. This disease may be difficult to diagnose when a single animal is affected.

Differential Diagnosis BEF in a single animal can be confused with early RVF, heartwater, BT, botulism, babesiosis or blackleg. The salivation may also resemble foot-and-mouth disease, but no vesicles are found. Laboratory Tests: Most cases of BEF are confirmed by serological tests.

- 2.9.4 Prevention & Control: Because both illness and viremia are transient, and the incubation period is short, import restrictions are usually effective unless the country shares a border with an endemic region. The vectors for BEFV are unknown, and successful eradication has not been reported once this disease becomes endemic. If an outbreak occurs among imported animals in a limited area, placing them in an insect-proof area and treating the area with insecticides has a chance of success. Sodium hypochlorite and other disinfectants effectively destroy BEFV; however, disinfection is relatively unimportant in preventing the spread of this virus. BEFV is not spread by casual contact or in secretions, and it is rapidly inactivated in carcasses after death.

In endemic areas, vaccination is generally used to prevent disease, particularly in lactating cattle and bulls. Vaccines are not always necessary in endemic areas where outbreaks occur regularly and most animals are immune before they become adults. Vaccination can also be used in the face of an outbreak. Although insect control would theoretically be helpful, its efficacy is unknown. Moving valuable animals into insect-proof facilities may be considered during outbreaks or in high-risk seasons.

Treatment is often unnecessary in non-lactating cows, but bulls or high-producing, lactating animals are often treated, particularly when they have become recumbent. Anti-inflammatory drugs and calcium borogluconate injections are effective. Good nursing can also aid recovery. Recumbent animals should be provided with water, food and shelter

if necessary, but animals should not be forced to stand or move. Force-feeding is not advisable due to the risk of aspiration pneumonia. Laterally recumbent animals may be rolled periodically to prevent loss of circulation and muscle damage.

## 2.10 Crimean-Congo Hemorrhagic Fever

2.10.1 Definition: Crimean-Congo haemorrhagic fever (CCHF) is a notifiable zoonotic viral hemorrhagic fever of the *Genus: Nairovirus, Family: Bunyaviridae*. Although primarily a zoonosis, sporadic cases and outbreaks of CCHF affecting humans do occur. The disease is endemic in many countries in Africa, Europe and Asia.

2.10.2 Epidemiology: CCHF is a severe disease in humans, with a high mortality rate. Fortunately, human illness occurs infrequently, although animal infection may be more common. The geographical distribution of the virus, like that of its tick vector, is widespread. All of the 32 members of the *Nairovirus* genus are transmitted by argasid or ixodid ticks, but only three have been implicated as causes of human disease: the Dugbe and Nairobi sheep viruses, and CCHF, which is the most important human pathogen amongst them. The CCHF virus may infect a wide range of domestic and wild animals. Many birds are resistant to infection, but ostriches are susceptible and may show a high prevalence of infection in endemic areas. Animals become infected with CCHF from the bite of infected ticks. A number of tick genera are capable of becoming infected with CCHF virus, but the most efficient and common vectors for CCHF appear to be members of the *Hyalomma* genus. Transovarial and sexual transmission have been demonstrated amongst some vector species, indicating one mechanism which may contribute to maintaining the circulation of the virus in nature. However, the most important source for acquisition of the virus by ticks is believed to be infected small vertebrates on which immature *Hyalomma* ticks feed. Once infected, the tick remains infected through its developmental stages, and the mature tick may transmit the infection to large vertebrates, such as livestock. Domestic ruminant animals, such as cattle, sheep and goats, are viremic for around one week after becoming infected. Humans who become infected with CCHF acquire the virus from direct contact with blood or other infected tissues from livestock during this time, or they may become infected from a tick bite. The majority of cases have occurred in those involved with the livestock industry, such as agricultural workers, slaughterhouse workers and veterinarians. The length of the incubation period for the illness appears to depend on the mode of acquisition of the virus. Following infection via tick bite, the incubation period is usually one to three days, with a maximum of nine days. The incubation period following contact with infected blood or tissues is usually five to six days, with a documented maximum of 13 days.

Clinical signs in human:

Onset of symptoms is sudden, with fever, myalgia, dizziness, neck pain and stiffness, backache, headache, sore eyes and photophobia. There may be nausea, vomiting and sore throat early on, which may be accompanied by diarrhoea and generalised abdominal pain. Over the next few days, the patient may experience sharp mood swings, and may become confused and aggressive. After two to four days, the agitation may be replaced

by sleepiness, depression and lassitude, and the abdominal pain may localize to the right upper quadrant, with detectable hepatomegaly. Other clinical signs which emerge include tachycardia, lymphadenopathy, and a petechial rash, both on internal mucosal surfaces, such as in the mouth and throat, and on the skin. Bleeding in the upper bowel changes the color of feces. There is hematuria and epistaxis with bleeding in the gum.

The mortality rate from CCHF is approximately 30%, with death occurring in the second week of illness. In those patients who recover, improvement generally begins on the ninth or tenth day after the onset of illness.

2.10.3 Diagnosis: Diagnosis of suspected CCHF is performed in specially-equipped, high biosafety level laboratories. IgG and IgM antibodies may be detected in serum by ELISA from about day six of illness. IgM remains detectable for up to four months, and IgG levels decline but remain detectable for up to five years. Patients with fatal disease do not usually develop a measurable antibody response and in these individuals, as well as in patients in the first few days of illness, diagnosis is achieved by virus detection in blood or tissue samples. The virus may be isolated from blood or tissue specimens in the first five days of illness, and grown in cell culture. Viral antigens may sometimes be shown in tissue samples using immunofluorescence. Recently, PCR became the most significant diagnostic test.

2.10.4 Prevention and control: Although an inactivated, mouse brain-derived vaccine against CCHF has been developed and used on a small scale in Eastern Europe, there is no safe and effective vaccine widely available for human. The tick vectors are numerous and widespread and tick control with acaricides is only a realistic option for well-managed livestock production facilities. Persons living in endemic areas should use personal protective measures. Persons who work with livestock or other animals in the endemic areas can take practical measures to protect themselves. These include the use of repellents on the skin and clothing personal protective equipment. Healthcare workers in endemic areas should be aware of the illness and the correct infection control procedures to protect themselves and their patients from the risk of nosocomial (hospital-acquired) infection.

## 2.11 Equine Infectious Anemia

2.11.1 Definition: (Synonyms - Swamp Fever, Mountain Fever, Slow Fever, Equine Malarial Fever, Coggins Disease) Equine infectious anemia (EIA) is a notifiable retroviral disease of Equids that is characterized by acute and/or chronic recurring clinical signs including fever, anemia, edema and cachexia in some animals. Many horses have very mild or inapparent signs on first exposure, and carry this virus in a subclinical form. The owners of these animals are unlikely to realize that they are infected unless serological testing is done. All infected horses, including those that are asymptomatic, become carriers and are infectious for life. Infected animals must either be destroyed or remain permanently isolated from other equids to prevent transmission.

2.11.2 Epidemiology: EIA is caused by EIAV; it is a member of *Genus lentivirus*, *Family: Retroviridae*. EIAV is reported to infect all members of the Equidae. Clinical cases occur in horses and ponies (*Equus caballu*), and have also been reported in mules. Some horse-adapted viral isolates replicate to low levels without clinical signs in donkeys (*E. asinus*); however, unpublished evidence suggests that serially-passaged, donkey-adapted isolates may be pathogenic for this species.

EIA disease has been found nearly worldwide. This disease appears to be absent from a few countries including Iceland and Japan.

EIAV is transmitted mechanically on the mouthparts of biting insects. In horses, this virus persists in blood leukocytes for life, and also occurs in plasma during febrile episodes. Symptomatic horses are more likely to transmit the disease than animals with inapparent infections. High levels of viremia have also been reported during the early stages of the infection in mules. Significantly lower titers have been reported in donkeys inoculated with certain horse-adapted strains. Although other insects including stable flies (*Stomoxys calcitrans*) can transmit EIAV, the most effective vectors are biting flies in the family *Tabanidae*, especially horse flies (*Tabanus spp.* & *Hybomitra spp.*) and deer flies (*Chrysops spp.*). The bites of these flies are painful, and the animal's reaction interrupts feeding. The fly attempts to resume feeding immediately, either on the same animal or on another nearby host, resulting in the transfer of infectious blood. EIAV survives for a limited time on the mouthparts of insects, and it is less likely to be spread to more distant hosts. This virus can also be transmitted in blood transfusions or on contaminated needles, surgical instruments and teeth floats. It is reported to persist for up to 96 hours on hypodermic needles. EIAV may also be passed from a mare to her foal in uterus. Other, minor routes of transmission might be possible. EIAV does not appear to be shed in saliva or urine. However, it can be found in milk and semen, and horses can be infected by inoculating these secretions subcutaneously. Possible transmission through milk has been reported in some nursing foals. Although venereal transmission does not seem to be a major route of spread, one stallion appears to have transmitted the virus to a mare with a vaginal tear during breeding. The possibility of aerosol transmission by infectious material during close contact was raised during the 2006 outbreak in Ireland.

The incubation period is 7 - 45 days or longer. Some horses remain asymptomatic until they are stressed. The clinical signs of acute EIA are often nonspecific. In some cases in horses, the only sign is a fever, which is sometimes accompanied by transient inappetence. In mild cases, the fever can last less than 24 hours. More severely affected horses can become weak, depressed and inappetent, with additional signs that may include jaundice, tachypnea, tachycardia, and ventral pitting edema, and thrombocytopenia, petechiae on the mucus membranes, epistaxis or blood-stained feces. Anemia can occur, although it is more likely to be severe in chronically infected animals. Occasionally, horses become gravely ill and may die during the acute stage. After the initial bout, most horses become asymptomatic carriers; however, some animals develop recurring clinical signs that vary from mild illness and failure to thrive to fever, depression, and petechial hemorrhages on the mucus membranes, weight loss, anemia and dependent edema. Inapparent

infections may become symptomatic during concurrent illnesses, severe stress or hard work. Death is possible during these febrile episodes. Ophthalmic lesions, characterized by depigmentation with prominent choroidal vessels, have been reported in chronically infected horses.

The spleen, liver and abdominal lymph nodes may be enlarged, and the mucous membranes can be pale. In chronic cases, emaciation may also be noted. Edema is often found in the limbs and along the ventral abdominal wall. Petechiae may be observed on internal organs, including the spleen and kidney. Mucosal and visceral hemorrhages and blood vessel thrombosis have also been reported. Chronically infected horses that die between clinical episodes usually have no gross lesions, but some animals may have proliferative glomerulonephritis or ocular lesions. The infection rate varies with the geographic region. Virus transmission is influenced by the number and species of flies, their habits, the density of the horse population, the level of viremia in the host and the quantity of blood transferred. Infections are particularly common in humid, swampy regions. Seroprevalence rates as high as 70% have been seen on farms where the disease has been endemic for many years. The morbidity rate and severity of the clinical signs are influenced by the strain and dose of the virus, and the health of the animal. Horses are more likely to develop clinical signs than donkeys or mules, but many horses are infected subclinically. The presence of EIAV in a herd often goes unnoticed until some horses develop the chronic form of the disease or routine testing is done. Epizootics with high morbidity and mortality rates have been reported, but deaths are otherwise uncommon in naturally infected horses. Experimental inoculation with a high viral dose can result in mortality rates as high as 80%. EIA disease should be among the differentials in individual horses with weight loss, edema and intermittent fever. It should also be considered when several horses experience fever, anemia, edema, progressive weakness or weight loss, particularly when new animals have been introduced into the herd or a member of the herd has died.

2.11.3 Diagnosis: The differential diagnosis includes other febrile illnesses including EVA, purpura hemorrhagica, leptospirosis, babesiosis, severe strongyliasis or fascioliasis, phenothiazine toxicity, autoimmune hemolytic anemia and other diseases that cause fever, edema and/or anemia.

EIA disease is often confirmed by serology. Once an animal is infected, it becomes a carrier for life. The two most commonly used serological tests are the AGID or Coggins test and ELISA tests.

Reverse-transcriptase polymerase chain reaction (RT-PCR) assays can also be used to detect infected horses. These tests are valuable in determining the infection status of foals born to infected mares, because young animals may have maternal antibodies up to the age of 6-8 months. PCR tests can also be used to supplement or confirm serological tests, particularly when there are conflicting results or when an infection is suspected but serology is negative or equivocal (e.g., in early cases where antibodies have not developed). In addition, this technique can ensure that blood donors and horses used for vaccine

or antiserum production are uninfected. RT-PCR appears to be an effective method of diagnosis in mules as well as horses.

- 2.11.4 Prevention and Control: Many countries have control programs requiring equids to be tested for equine infectious anemia. Regular voluntary testing of the equids on a farm, as well as testing of new animals before introduction, is helpful in maintaining an EIA-free herd. No vaccine is available.

Infected equids become lifelong carriers, and must be permanently isolated from other susceptible animals or euthanized. Reactor must be marked with a brand, freezemarking or a lip tattoo before it is moved locally or between countries. The risk of transmission from carriers varies, but because it is impossible to quantify this risk, all infected horses are treated alike. Asymptomatic carriers often give birth to uninfected foals. The risk of congenital infection is higher if the mare has clinical signs before she gives birth. Foals born to infected mares should be isolated from other equids until the foal is determined to be free of infection. During an outbreak, spraying to control insect vectors, as well as the use of insect repellents and insect- proof stabling, may aid in interrupting transmission. Placing animals in small groups separated by at least 200 yards might be beneficial when the virus is being transmitted within a farm. Care should be taken to prevent iatrogenic transmission. In countries where equine infectious anemia is not present, outbreaks are contained with quarantines and movement controls, tracing of cases and surveillance.

## 2.12 Equine Viral Arteritis

- 2.12.1 Definition: (Synonyms - Equine Typhoid, Epizootic Cellulitis-Pinkeye, Epizootic Lymphangitis Pinkeye, Rotlaufseuche) Equine viral arteritis (EVA) is an economically important viral disease of equids. Stallions can become long term carriers of the virus, and transmit it during breeding. Although carrier stallions can be bred if precautions are taken, the need to mate them with seropositive or vaccinated mares decreases their desirability as breeders. Acute illness also occurs in some horses. Although deaths are very rare in healthy adults, pregnant mares that become infected may abort, and very young foals may die of fulminating pneumonia and enteritis. Equine viral arteritis has recently increased in prevalence, possibly due to increased transportation of horses and semen.

- 2.12.2 Epidemiology: EVA is caused by equine viral arteritis virus (EVAV), an RNA virus in the *Genus: Arterivirus, family: Arteriviridae*. Isolates vary in their virulence and potential to induce abortions. Only one serotype has been recognized. Limited genetic analysis suggests that EVAV strains found among donkeys in South Africa may differ significantly from isolates in North America and Europe.

EVAV is found in the Equidae. Antibodies to this virus have been reported in horses, ponies, donkeys and zebras. Illness occurs mainly among horses and ponies, but clinical signs have also been reported in experimentally infected donkeys.

EVAV can be transmitted by the respiratory and the venereal routes. Acutely affected horses

excrete the virus in respiratory secretions; aerosol transmission is common when horses are gathered at racetracks, sales, shows and other events. This virus has also been found in urine and feces during the acute stage. It occurs in the reproductive tract of acutely infected mares, and both acutely and chronically infected stallions. In mares, EVAV can be found in vaginal and uterine secretions, as well as in the ovary and oviduct, for a short period after infection. Mares infected late in pregnancy may give birth to infected foals. Stallions shed EVAV in semen, and can carry the virus for years. Transmission from stallions can occur by natural service or artificial insemination. Some carriers may eventually clear the infection.

The incubation period varies from 2 days to 2 weeks. Infections transmitted venereally tend to become apparent in approximately one week. Most EVAV infections, especially those that occur in mares bred to long-term carriers, are asymptomatic.

The clinical signs are generally more severe in old or very young animals, and in horses that are in poor condition. Fulminant infections with severe interstitial pneumonia and/or enteritis can be seen in foals up to a few months of age. Systemic illness also occurs in some adults. In adult horses, the clinical signs may include fever, depression, anorexia, limb edema (particularly in the hind limbs), and dependent edema of the prepuce, scrotum, mammary gland and/or ventral body wall. Conjunctivitis, photophobia, periorbital or supraorbital edema and rhinitis can also be seen. Some horses develop urticaria; the hives may be localized to the head or neck, but are sometimes generalized. Abortions or stillbirths can occur in mares that are pregnant when they are exposed. Abortions are not necessarily preceded by systemic signs. In acute cases, the lesions are characterized by edema, congestion and hemorrhages of the subcutaneous tissues, visceral organs and lymph nodes. These changes are often found in the subcutaneous tissues of the limbs and abdomen, the thoracic and abdominal lymph nodes, and the small and large intestines (especially the colon and cecum), but may occur throughout the body. Accumulations of clear, yellowish fluid may be found in the peritoneal cavity, pleura and pericardium. Foals may also have pulmonary edema, interstitial pneumonia, emphysema, splenic infarcts and enteritis. In mares that abort, the endometrium may be swollen and congested, and can contain hemorrhages. Aborted fetuses are often partially autolyzed, but may be well preserved. In some fetuses, the only gross lesions may be excess fluid in the body cavities and signs of interlobular interstitial pneumonia.

- 2.12.3 Diagnosis: EVA should be considered when the clinical signs include fever, depression, edema, conjunctivitis, nasal discharges and abortions. This disease is difficult to differentiate from other systemic and respiratory illnesses of horses. The differential diagnosis includes equine influenza, EIA and AHS, as well as infections with Geta virus, Hendra virus, equine rhinitis A and B viruses, equine adenoviruses, and equine herpesviruses 1 & 4. EVA also resembles purpura hemorrhagica and other streptococcal infections, as well as poisoning from the toxic plant *Berteroia incana* (hoary alyssum).

EVA can be diagnosed by virus isolation, the detection of viral antigens or nucleic acids, and serology. RT-PCR can also detect viral nucleic acids in clinical samples from live horses or tissues taken at necropsy. Serological tests include virus neutralization (VN), complement fixation test (CFT), AGID, indirect immunofluorescent antibody, fluorescent microsphere immunoassay (FMIA) and ELISA.

- 2.12.4 Prevention and Control: Acutely infected horses should be isolated to prevent transmission in secretions and excretions. Precautions should also be taken to avoid spreading the virus on fomites. EVAV is readily inactivated by detergents, common disinfectants and lipid solvents. No specific treatment is available; however, most horses other than young foals recover on their own.

Good nursing and symptomatic treatment should be used in severe cases. Vaccination can also help contain outbreaks. Venereal transmission can be controlled by good management and vaccination. To protect pregnant mares from abortion, they should be separated from other horses and maintained in small groups according to their predicted foaling dates. Prepubertal colts are given the vaccine when they are 6-12 months old. Carrier stallions are identified and bred only to well vaccinated or naturally seropositive mares. Similarly, semen that contains EVAV should be used only in these mares. Because first time vaccination may shed field viruses for a short time after exposure, these mares should be isolated from seronegative horses, particularly pregnant mares, for three weeks after breeding. Naturally infected mares and those that were vaccinated are going to be isolated for 24 - 48 hours, to protect other horses from the viruses present in semen.

Carrier stallions should be housed where they can be physically separated from uninfected horses.

## 2.13 ORF

- 2.13.1 Definition: (Synonyms - Contagious Ecthyma, Ecthyma Contagiosum, Contagious Pustular Dermatitis, Contagious Pustular Stomatitis, Infectious Labial Dermatitis, Soremouth, Scabby Mouth) ORF is a highly contagious, zoonotic, notifiable, viral skin disease that affects sheep, goats and some other domesticated and wild ruminants. Severe generalized infections have been described in Boer and Boer cross goats. Although ORF usually resolves spontaneously and the mortality rate is generally low, fatality rates up to 10% have been reported. Most infections in humans are localized and heal spontaneously; however, large, poorly healing lesions can occur in people who are immunosuppressed.

- 2.13.2 Epidemiology ORF results from infection by the orf virus, a member of the *Genus: Parapoxvirus* in the family: *Poxviridae*. ORF has been found worldwide in all countries that raise sheep. ORF occurs in sheep, goats, alpacas, camels, reindeer, musk oxen, bighorn sheep, deer, prong-horn antelope and wapiti. Rare cases have been reported in dogs that ate infected carcasses. The incubation period in sheep and goats is 2 to 3 days. The initial signs are papules, pustules and vesicles, found on the lips, nose, ears and/or eyelids, and

sometimes on the feet or perineal region.

Lesions can also occur inside the mouth, particularly in young lambs. Massive oral lesions have been described in some reindeer. Rarely, the lesions may extend into the esophagus, stomach, intestines or respiratory tract. Nursing lambs can transmit the virus to their dam, resulting in lesions on the teats and udder. The skin lesions eventually develop into thick, brown, rapidly growing scabs over areas of granulation, inflammation and ulceration. The scabs are often friable and bleed easily. Papillomatous growths sometimes occur. ORF lesions are painful and may result in anorexia or even starvation. Young animals may refuse to nurse, and lesions on the udder of the dam can cause it to abandon its offspring. Foot lesions can cause lameness. Uncomplicated infections usually resolve in 1 to 4 weeks. Secondary bacterial infections and maggot infestations can occur. ORF may predispose animals to bacterial mastitis.

In these animals, the disease consisted of multifocal, severe proliferative dermatitis accompanied by chronic pneumonia, arthritis and moderate to severe lymphadenopathy. The disease persisted for three months until the animals were euthanized. ORF is highly communicable. The ORF virus is present in the skin lesions and crusts. It can also remain viable on the wool and hides for approximately one month after the lesions have healed.

The ORF virus, which is found in skin lesions and scabs, is thought to enter the skin through cuts and abrasions. This virus can be carried by clinically normal sheep as well as sick animals. It can be transmitted by direct contact or on fomites. It is very resistant to inactivation in the environment and has been recovered from dried crusts after 12 years.

The incubation period in humans is 3 to 7 days. In humans, ORF usually occurs as a single skin lesion or a few lesions. The initial lesion is a small, firm, red to blue papule at the site of virus penetration, most often a finger, hand or other exposed part of the body. The papule develops into a hemorrhagic pustule or bulla, which may contain a central crust and bleeds easily. In the later stages, the lesion develops into a nodule, which may weep fluid and is sometimes covered by a thin crust.

It eventually becomes covered by a thick crust. The skin lesion(s) may be accompanied by a low grade fever that usually lasts only a few days, or by mild lymphadenopathy. In uncomplicated disease, the lesion heals spontaneously in 3 to 6 weeks without scarring. Secondary infections can occur. Large lesions refractory to treatment can occur in people who are immunosuppressed. Unusually large lesions have also been reported in people with atopic dermatitis. Rare cases involving the eye, as well as a generalized vesiculopapular rash on the skin and mucosa, have also been reported. Possible complications include toxic erythema, erythema multiforme and bullous pemphigoid. Human-to-human transmission is nonexistent or very rare.



Fig. 6 ORF Lesions on Mouth and Nostrils

**2.13.3 Diagnosis:** ORF can be confirmed by electron microscopy of the crust, a small biopsy or fluid from the lesion; however, this technique cannot distinguish the ORF virus from other parapoxviruses. PCR assays can give a definitive diagnosis. Histopathology can also be helpful. Virus isolation can be attempted but the ORF virus grows slowly and cannot always be isolated. Animal inoculation into lambs has been reported. Serology and the detection of viral antigens can be used in research, but are not ordinarily used for diagnosis.

In immunocompetent humans, ORF is usually self-limiting. Treatment in humans is supportive and typically consists of moist dressings, local antiseptics, finger immobilization and/or antibiotics to treat secondary bacterial infections. Large lesions can be removed by surgery, and curettage and electrodesiccation may be used for persistent lesions. Cryotherapy has been reported to hasten recovery. Abraded or cut skin should not be allowed to contact infected animals, scabs and crusts, wool or hides. Non-porous gloves (rubber or latex) should also be considered when asymptomatic sheep, goats or other susceptible ruminants including deer are handled. This precaution may be particularly advisable when handling an animal's mouth. The ORF vaccines are pathogenic for humans, and gloves should also be used when vaccinating animals. Any skin that has been exposed should be washed with soap and water. Some sources suggest additional disinfection with 70% isopropyl alcohol after washing. People who are immunosuppressed should avoid contact with infected animals. Serological tests include VN, AGID, CFT and agglutination test. ELISA tests have been developed but are rarely used for diagnosis.

**2.13.4 Control and Prevention:** There is no specific treatment for ORF. Repellents can be used to keep flies away from the wounds, and antibiotics are given for secondary infections. Supportive care, including tube feeding, may be necessary. To prevent ORF from entering an uninfected herd, new animals should be quarantined; some carriers may not have clinical signs. Precautions should be taken to prevent virus introduction on equipment and other fomites. Harsh vegetation should be removed from pastures or feed, to reduce the risk of cuts in the mouth or on the muzzle. At shows and fairs, some exhibitors prefer to open their own animal's mouth, to prevent inadvertent spread between animals on the hands. Vaccination is practiced in some areas. ORF vaccines contain live virus prepared from dried scabs or propagated in tissue culture. Vaccines should be used only on premises where infections have occurred in the past, and recently vaccinated animals should be isolated

from unvaccinated animals. The duration of immunity after vaccination is controversial; outbreaks have occurred in vaccinated animals, but vaccine breaks may be due to the virulence of the strain. ORF vaccines contain live virus and can infect humans. Recently vaccinated animals can also transmit infections to humans. Isolation of infected animals can prevent the spread of the disease. ORF virus is difficult to eradicate once it has entered a flock or herd. The best disinfectants for the poxviruses are detergents, hypochlorite, alkalis, Virkon® and glutaraldehyde. The morbidity rate is usually high, often 80% in an unvaccinated flock. In endemic areas, annual outbreaks may occur on ranches. Any age can be infected in a naïve flock; however, in endemic regions, most cases occur in sheep and goats less than a year old, as older animals usually have some degree of immunity. The duration of protective immunity is controversial.

## 2.14 Sheep & Goat Pox

**2.14.1 Definition:** Sheep and goat pox are contagious viral diseases of small ruminants. These diseases may be mild in indigenous breeds living in endemic areas, but are often fatal in newly introduced animals. Economic losses result from decreased milk production, damage to the quality of hides and wool, and other production losses. Sheep and goat pox can limit trade and prevent the development of intensive livestock production. They may also prevent new breeds of sheep or goats from being imported into endemic regions.

**2.14.2 Epidemiology:** Sheep pox and goat pox result from infection by sheep pox virus or goat pox virus, closely related members of the Genus: *Capripox* in the family: *Poxviridae*. Most isolates are host-specific, with sheep pox virus mainly causing disease in sheep and goat pox virus predominantly affecting goats. However, some isolates can cause serious disease in both species. Sheep pox virus and goat pox virus cannot be distinguished from each other with serological techniques (including VN), and were once thought to be strains of a single virus. Genetic sequencing has now demonstrated that these viruses are distinct, but recombination can occur between them. Recombinant strains usually have intermediate host specificity. Both species are closely related to LSDV that causes LSD in cattle. The relationships between these three capripoxviruses are still being established, but one recent analysis suggests that goat pox virus and LSDV are more closely related to each other than sheep pox virus is to LSDV. Sheep and goat capripoxviruses cause disease only in these two species. Many sheep pox virus isolates are specific for sheep, and many goat pox virus strains are specific for goats, but some strains of these viruses readily affect both species. Infections have not been reported in wild ungulates.

Sheep pox and goat pox are found in parts of Africa and Asia, the Middle East, and most of the Indian subcontinent. Sheep pox and goat pox viruses are often transmitted by the respiratory route during close contact, but they may also enter the body through other mucous membranes or abraded skin. These viruses can be found in saliva, nasal and conjunctival secretions, milk, urine and feces, as well as in skin lesions and their scabs. Ulcers on the mucous membranes are important sources of virus. Whether Sheep pox and goat

pox viruses can be transmitted in semen or embryos has not been established. Animals are most contagious before neutralizing antibodies develop, which occurs approximately a week after the onset of clinical signs. Experimentally infected sheep and goats can shed poxviruses in nasal, conjunctival and oral secretions for 1 to 2 months, but shedding peaks during the second week after inoculation, then declines rapidly. Chronically infected carriers are not seen. Sheep pox and goat pox viruses can also be spread on fomites or transmitted mechanically by insects such as stable flies (*Stomoxys calcitrans*), although the latter route may be uncommon. These viruses can remain infectious for up to six months in shaded sheep pens. They may also be found on the wool or hair for as long as three months after infection, and possibly longer in scabs. Whether the viruses in scabs are infectious is unknown; these viruses are complexed with antibodies and can be difficult to recover on tissue culture media. The incubation period varies from 4 to 21 days, but it is usually 1 to 2 weeks.

Clinical signs generally appear sooner when the virus is inoculated by insects than when it is transmitted in aerosols.

After experimental inoculation into the dermis, primary lesions can develop at the site within 2 to 4 days. The clinical signs vary from mild to severe, depending on the animal's age, breed, immunity and other factors. Inapparent infections also occur. In affected animals, an initial fever is usually followed in one to five days by the characteristic skin lesions, which begin as erythematous macules, and develop into 0.5 - 1.5 cm hard papules. In the common, papulovesicular form of the disease, the centers of the papules become depressed, whitish gray and necrotic, and are surrounded by an area of hyperemia. Dark, hard, sharply demarcated scabs eventually form over the necrotic areas. Vesicles might be seen during the intermediate stage, but are uncommon. In the uncommon, nodular form of the disease (stonepox), the papules develop into nodules. These nodules can be found in the epidermis, dermis and subcutaneous tissues. They become necrotic and slough, leaving a hairless scar. Some European breeds of goats may develop a flat hemorrhagic form of goat pox. In this form, the papules seem to coalesce over the body, and the animal invariably dies. Capripox lesions have a predilection for areas of sparsely wooled/ haired skin such as the axillae, muzzle, eyelids, ears, mammary gland and inguinal area, but in more severe cases, they may cover the body. In animals with heavy wool, the lesions can be easier to find by palpation than visual inspection. Mild infections can easily be missed; only a few lesions may be present, often around the ears or the tail. All superficial lymph nodes usually become enlarged within a day of the appearance of generalized papules; the prescapular lymph nodes are particularly noticeable. Lesions can also develop on the mucous membranes and internal organs, causing systemic signs. In some cases, these symptoms may precede the onset of skin lesions by a day or two. Lesions in the mouth, nares, eyes or eyelids can cause salivation or inappetence, as well as rhinitis, conjunctivitis or blepharitis with mucopurulent discharges. Affected mucous membranes may become necrotic and ulcerate or slough. Animals with lung lesions may have respiratory signs including coughing, nasal discharge and dyspnea. Nodules in the intestines can cause diarrhea. Depression and emaciation may be seen in some animals. Abortions can occur but are not common. Some breeds of sheep can die of acute disease before the

characteristic skin lesions appear. Capripox lesions can take several weeks to heal, and may leave permanent scars on the skin. During healing, they are susceptible to fly strike. Secondary bacterial infections, including pneumonia, are common, and death can occur at any stage of the disease. Recovery can be slow if the animal was severely affected. The skin usually contains macules, papules and/or necrotic lesions and scabs, surrounded by areas of edema, hemorrhage and congestion.

The papules penetrate through both the dermis and epidermis; in severe cases, they may extend into the musculature. Skin lesions may not be as apparent at necropsy as they are in living animals. The mucous membranes of the eyes, nose, mouth, vulva and prepuce may be necrotic or ulcerated. The lungs often contain congested, edematous or consolidated areas, and firm gray or white nodules. Nodules in the lungs can be up to 5 cm in diameter, and are particularly common in the diaphragmatic lobes. In early stages of the disease, they may appear as red spots.

Papules or ulcerated papules are common on the abomasal mucosa. They may also be found on the rumen, large intestine, pharynx, trachea and esophagus.

Pale, discrete subcapsular foci are sometimes present on the surface of the kidney, liver and testes. Lymph nodes throughout the body are usually enlarged and edematous, and may be congested and hemorrhagic. Morbidity and mortality vary with the breed of the animal, its immunity to capripoxviruses, and the strain of the virus. Mild infections are common among indigenous breeds in endemic areas, but more severe disease can be seen in young or stressed animals, animals with concurrent infections, or animals from areas where pox has not occurred for some time. Morbidity rates in indigenous breeds are 1% - 75% or higher. Although the mortality rate is often less than 10%, case fatality rates of nearly 100% have been reported in some young animals. Imported breeds of sheep and goats usually develop severe disease when they are moved into an endemic area. The morbidity and mortality rates can approach 100% in newly imported, highly susceptible flocks.

**2.14.3 Diagnosis:** Lesions are so clear and suggestive. A definitive diagnosis can be made by recovering the causative viruses. SPV and GPV can be isolated in lamb testis, sheep or goat kidney cell cultures, as well as in other (less sensitive) sheep, goat or bovine cell lines. Inhibition of the cytopathic effect (CPE) by specific antibodies in the medium provides presumptive identification. Capripoxviruses can be identified to at least the genus level by immunofluorescence or immunoperoxidase staining, nucleic acid recognition methods and other techniques. In some circumstances, these viruses have also been recovered by inoculation into sheep or goats. PCR assays can detect capripoxvirus genomes in tissue samples or cultures, but cannot identify whether the virus is sheep pox virus or goat pox virus. How-ever, these two viruses can be distinguished if PCR is combined with a restriction fragment length polymorphism (RFLP) assay. Recombination between Sheep pox and goat pox viruses can complicate identification of the virus.



Fig. 7 Sheep pox skin lesions

Viral antigens can be detected in tissues by AGID or various ELISAs. Counter-immunoelectrophoresis, latex agglutination and indirect agglutination tests (reverse- phase passive hemagglutination, coagglutination, passive hemagglutination and spot agglutination) have also been used. In the AGID test, cross reactions occur between capripoxviruses and parapoxviruses; however, these two groups of viruses can be distinguished with electron microscopy.

Serology can identify Sheep pox and goat pox viruses as capripoxviruses, but cannot distinguish these two viruses from each other. Antibodies to capripoxviruses can be found approximately one week after the skin lesions appear. Serological tests include virus neutralization, AGID, indirect immunofluorescent test, ELISAs and immunoblotting (Western blotting). VN is the most specific serological test, but it is not sensitive enough to detect infections in all animals. Crossreactions occur with other viruses in the AGID and indirect immunofluorescent tests.

**Samples to collect:** In live animals, biopsies of skin lesions should be taken for virus isolation and antigen detection. Viruses can also be found in vesicular fluid, scabs and scrapings of skin lesions, as well as lymph node aspirates and blood (collected into heparin or EDTA). At necropsy, samples should be collected from skin lesions, lymph nodes and lung lesions. An additional set of samples should be taken for histology; these samples should include a wide range of lesions from the skin, as well as spleen, rumen, trachea, lungs and other affected tissues. PCR can detect capripoxviruses in blood, nasal or oral swabs, scabs, skin lesions and tissue samples. Neutralizing antibodies can interfere with virus isolation and some antigen-detection tests; samples for these tests must be collected during the first week of illness. Samples for PCR can be taken after neutralizing antibodies have developed. Paired serum samples should be collected for serology. Samples for virus isolation must be sent to the laboratory as soon as possible. They should be kept cold and shipped on wet ice or gel packs. If these samples must be shipped long distances without refrigeration, glycerol (10%) can be added; the tissue samples must be large enough that the medium does not penetrate into the center of the tissue and destroy the viruses there.

**2.14.4 Prevention and Control:** Capripoxviruses are most likely to be introduced in infected animals, but fomites and animal products such as wool can also spread disease. Outbreaks can be controlled by quarantines, movement controls, and segregation of infected and exposed animals, followed by stringent cleaning and disinfection of farms and equipment. Proper disposal of infected carcasses is important; burning or burial is often used. Capripoxviruses may persist for up to 6 months in shaded, uncleaned pens and for at least a few months in dry scabs on skin, fleece and hair. Poxviruses are resistant to drying, and can also survive freeze/ thaw cycles, although the infectivity may be reduced. When the disease has spread more widely, vaccination may also be considered. Capripoxviruses are reported to be destroyed by heating to 56°C (133°F) for 2 hours, or to 65°C (149°F) for 30 minutes. Heat sensitivity may vary between strains of capripoxviruses; 56°C for one hour can inactivate some isolates, but does not significantly reduce the titer of others. Capripoxviruses are generally sensitive to ether (20%), formalin and chloroform, although some strains were resistant to ether in studies done in the 1940s.

Capripoxviruses are also reported to be susceptible to sodium hypochlorite and detergents that contain lipid solvents. In these regions, new animals should be quarantined before adding them to the flock or herd. Infected herds and sick animals should be isolated for at least 45 days after they have recovered from clinical signs. In some outbreaks, the herd may be culled. Sheep pox virus and goat pox virus do not appear to infect humans. Two published cases suggested that capripoxviruses might be transmitted to humans, but these reports are questionable. Annual vaccination of susceptible sheep and goat flocks with attenuated vaccines is essential in endemic regions.

## 2.15 Infectious Bovine Rhinotracheitis (IBR)

**2.15.1 Definition:** (Synonyms - Red Nose, infectious pustular vulvovaginitis (IPV) and infectious balanoposthitis) Infectious bovine rhinotracheitis (IBR) is a cattle highly contagious notifiable viral disease caused by *Bovine Herpesvirus-1(BHV-1)* Family: *Herpesviridae*. The disease has two main presentations. The respiratory form of IBR which is the more common and venereal forms infectious pustular vulvovaginitis (IPV) and infectious balanoposthitis (IBP), which occur in cows and bulls respectively.

**2.15.2 Epidemiology:** BHV1 is the IBR virus is of the Herpes group. As is characteristic of many Herpes viruses, the IBR is capable of producing a variety of clinical disease forms according to which tissues of the animal body it infects. In general, this virus produces five clinical forms of disease in cattle—respiratory, ocular, abortion, infectious pustular vulvovaginitis, and encephalitis. All ages and breeds of cattle are susceptible. IBR is endemic in many parts of the world, but some countries are free. IBR is transmitted via direct contact or infected semen. The incubation period is usually 4 to 6 days with the entire herd involved and the infection lasting for 10 to 14 days.

**Main Clinical signs:** The respiratory form of IBR presents as an upper respiratory tract infection with fever and additionally sometimes a marked conjunctivitis. Outbreaks are most often seen in cattle between six and 18 months of age though all ages of cattle are susceptible.

IBR may cause abortion if susceptible cows or heifers are infected during pregnancy. Not all cattle infected with the virus show obvious clinical signs. In the absence of secondary bacterial complications most animals recover from the disease. Latent infection can occur and infected cattle can shed the virus intermittently. Lesions are related to the forms of IBR: Respiratory Form, IBR Abortion, Ocular Form of IBR, IPV (Infectious Pustular Vaginitis)

- 2.15.3 Diagnosis: With experience, a diagnosis of uncomplicated IBR can usually be made based on the clinical signs, pattern of onset and lesions. However, this diagnosis should be confirmed by laboratory tests. BHV-1 can be easily isolated from swabs from all lesions, since the virus grows well in bovine cells in culture. Swabs for virus isolation should be taken early in the course of the disease since maximal virus replication and shedding occur between three and six days, after infection. The swabs should be kept cool and shipped as quickly as possible to the diagnostic laboratory. A number of diagnostic tests are available. Among them is the specific and sensitive PCR, which can detect very small quantities of virus in nasal secretions, tissue and semen. One of the most relevant diagnostic tools for the detection of BHV-1 infected herds is still serology using VN tests or ELISAs. Preferably, two blood samples should be taken, one at the onset of disease and the other three weeks later. A four-fold increase in BHV-1 specific serum antibody titre should be considered diagnostic of an active BHV-1 infection.
- 2.15.4 Prevention and Control: Infection can be controlled by vaccination and marker vaccines are available that allow the differentiation of vaccinated from infected cattle in antibody tests. Prevention and control of IBR is mainly dependent on development of immunity to BHV-1, which occurs following natural exposure or vaccination. Since BHV-1 is a ubiquitous, highly contagious virus, vaccination is recommended as soon as passive immunity in calves has disappeared, usually around four to six months of age. Currently available vaccines for IBR include modified-live-virus vaccines and inactivated or killed-virus vaccines.

## 2.16 Camel Pox

- 2.16.1 Definition: Camel pox is a notifiable viral disease characterized by fever, local or generalized pox lesions on the skin of camel and in the mucous membranes of the mouth, respiratory and digestive tracts.
- 2.16.2 Epidemiology: Camel pox is a wide-spread infectious viral disease of old world camelids. New world camelids are also susceptible. It occurs throughout the camel- breeding areas causing economic impact through loss of production and sometimes death. Camelpox virus belongs to the *Genus: Orthopoxvirus family: Poxviridae*, Camelpox virus is very host specific and does not infect other animals. Only one suspected case of human camelpox involving mild skin lesions has been described, underlining that camelpox is of no public health importance. There are relations between smallpox of human & camelpox. It occurs throughout the camel-breeding areas of Africa, north of the equator, the Middle East and Asia.

Camelpox occurs in almost every country in which camel husbandry is practiced apart

from the introduced dromedary camel in Australia and tylopods (llama and related species) in South America. Outbreaks have been reported in the Middle East (Bahrain, Iran, Iraq, Oman, Saudi Arabia, United Arab Emirates and Yemen), in Asia (Afghanistan and Pakistan), in Africa (Algeria, Egypt, Ethiopia, Kenya, Mauritania, Morocco, Niger, Somalia and Sudan) and in the southern parts of Russia and India. Transmission is by either direct contact between infected and susceptible animals or indirect infection via a contaminated environment. The role of insects in transmission has been suspected because the disease is often observed after rainfall. The incubation period is usually 9 –13 days (varying between 3 and 15 days).

Main clinical signs of camelpox are characterized by fever, local or generalized pox lesions on the skin and in the mucous membranes of the mouth, respiratory and digestive tracts. The clinical manifestations range from inapparent infection to mild, moderate and, less commonly, severe systemic infection and death. The disease occurs more frequently and more severely in young animals. The animals may show salivation, lacrimation and a mucopurulent nasal discharge. Diarrhea and anorexia may occur in the systemic form of the disease. Pregnant females may abort. Death is usually due to secondary infections and septicaemia. The morbidity rate of camelpox is variable and depends on whether the virus is circulating in the herd.

Serological surveys taken in several countries reveal a high prevalence of antibodies to camelpox. The incidence of disease is higher in males than females, and the mortality rate is greater in young animals than in adults. The mortality rate in adult animals is between 5% and 28% and in young animals between 25% and 100%.

Skin lesions appear 1–3 days after the onset of fever, starting as erythematous macules, developing into papules and vesicles, and later turning into pustules. Crusts develop on the ruptured pustules. These lesions first appear on the head, eyelids, nostrils and the margins of the ears. In severe cases the whole head may be swollen. Later, skin lesions may extend to the neck, limbs, genitalia, mammary glands and perineum. In the generalised form, pox lesions may cover the entire body. Skin lesions may take up to 4–6 weeks to heal.

- 2.16.3 Diagnosis: The presumptive diagnosis of camelpox infection is based on clinical signs. However, infections of camels with ORF, papilloma virus and reaction to insect bites are considered differential diagnosis in the early clinical stages and in mild cases of camelpox. Serological tests: A wide range of serological tests are available to identify camelpox. The tests used for the detection of the antibodies against camelpox virus include neutralisation, agar gel precipitation, haemagglutination, haemagglutination inhibition, CFT, fluorescent antibody and antibody capturing ELISA. Identification of the causative agent is done by:
- Transmission electron microscopy.
  - Virus isolation in cell cultures.



Fig. 8 Camel Pox skin lesions on hindquarters

2.16.4 Prevention and Control: A live attenuated vaccine, Ducapox, is manufactured by Highveld Biologicals, Onderstepoort, South Africa and an inactivated vaccine by Biopharma, Rabat, Morocco are two commercial vaccines available for control of camel pox. A live attenuated vaccine gives long-term protection against camelpox.

However, a booster vaccination is recommended for young animals vaccinated before the age of 6–9 months. When inactivated vaccine is used, the animals must be vaccinated annually

## 2.17 Mad Cow Disease

2.17.1 Definition: (Synonyms –Bovine Spongiform Encephalopathy (BSE) the scientific name so BSE is more common in literature text).

BSE is a chronic degenerative disease affecting the central nervous system of cattle. BSE is a progressive and fatal neurological disease of cattle caused by an unconventional transmissible agent (Prion). BSE belongs to the family of diseases known as transmissible spongiform encephalopathies (TSEs)

2.17.2 Epidemiology: The disease was first diagnosed in 1986 in the United Kingdom. Since that time, it has been found in many European countries, and also in countries outside of Europe, including Japan, Canada, and the United States. To date, however, more than 95 percent of the total cases worldwide have occurred in the United Kingdom. In addition to BSE, TSEs include scrapie, which affects sheep and goats; transmissible mink encephalopathy; chronic wasting disease of deer and elk; and in humans, kuru, both classic and variant Creutzfeldt–Jakob disease (CJD), Gerstmann– Sträussler–Scheinker syndrome (GSS), and fatal familial insomnia. The agents that cause BSE and other TSEs have yet to be fully characterized. The theory most widely accepted in the scientific community is that the agent is a Prion an abnormal protein. The BSE agent is extremely resistant to heat and to normal sterilization processes. It also does not evoke any detectable immune response or inflammatory

reaction in host animals. Cattle affected by BSE experience progressive degeneration of the nervous system. Affected animals may display nervousness or aggression, abnormal posture, difficulty in coordination and rising, decreased milk production, or loss of body weight despite continued appetite. All infected cattle die. There is neither any treatment nor a vaccine to prevent the disease. The incubation period is the time from when an animal becomes infected until it first shows clinical signs averages 4 to 6 years, although the period can be longer or shorter. Following the onset of clinical signs, the animal's condition deteriorates until it either dies or is destroyed. The process of deterioration usually takes from 2 weeks to 6 months.

2.17.3 Diagnosis: Currently, there is no test to detect the disease in live cattle; veterinary pathologists confirm BSE by postmortem microscopic examination of brain tissue or by the detection of abnormal prions in brain tissue. BSE is so named because of the spongy appearance of the brain tissue of infected cattle when examined under a microscope BSE is not a contagious disease and therefore is not spread through casual contact between cattle. The primary source of BSE infection in cattle is commercial feed contaminated with the infectious agent. Scientific evidence shows that feed contamination results from incorporating ingredients (for example, meat and bone meal) that contain protein derived from rendered infected cattle. Standard rendering processes do not completely inactivate or kill the BSE agent.

Therefore, rendered protein such as meat and bone meal derived from infected animals may contain the infectious agent. Regulations prohibiting the inclusion of mammalian or ruminant protein in ruminant feed, including cattle feed, are used to prevent BSE transmission.

Consumption of feed contaminated with the BSE agent is the only documented route of field transmission of BSE. However, limited research cannot rule out the possibility of maternal or vertical transmission.

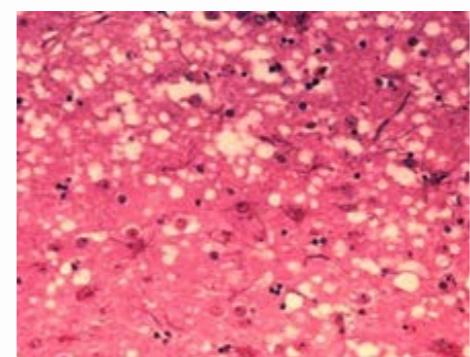


Fig. 9 Sponge like Brain Tissue of BSE



Fig.10 Clinical BSE in cow

2.17.4 Prevention and Control: The measures in the strategy for dealing with BSE are early detection and warning systems and prevention and rapid response measures and mechanisms in place.

- a) targeted surveillance of occurrences of clinical neurological disease;
  - b) awareness programs to enhance surveillance;
  - c) screening tests at routine slaughter;
  - d) transparency in reporting findings of BSE;
  - e) safeguards on importation of live ruminant species and their products, in accordance with the OIE Terrestrial Code ;
  - f) removal of specified risk material (SRM) (brain, spinal column) during slaughter and processing of carcasses;
  - g) prohibit the inclusion of SRM in animal feeds, thus removing potentially contaminated material from the food chain;
  - h) humane destruction of all suspected and susceptible animals exposed to contaminated feed (cohorts);
  - i) appropriate disposal of infected carcasses and all animal by-products;
- Animal identification enables effective surveillance and tracing of suspected livestock.

## Chapter (3) Bacterial Diseases

- 3.1 Enterotoxaemias
- 3.2 Lamb dysentery
- 3.3 Pulpy kidney
- 3.4 Malignant edema
- 3.5 Big head
- 3.6 Black disease
- 3.7 Tetanus
- 3.8 Septicaemic pasteurllosis
- 3.9 Pneumonic pasteurllosis
- 3.10 Brucellosis
- 3.11 Colibacillosis
- 3.12 Salmonellosis
- 3.13 Anthrax
- 3.14 Listeriosis
- 3.15 Paratuberculosis
- 3.16 Bovine tuberculosis
- 3.17 Caseous lymphadenitis
- 3.18 Footrot
- 3.19 Leptospirosis
- 3.20 Contagious Caprine Pleuropneumonia
- 3.21 Q Fever

### 3.1 Enterotoxemias

- 3.1.1 Definition: Enterotoxaemias are group of diseases that usually affect the intestine, caused by different types of *Clostridium perfringens* (A, B, C, and D). These bacteria usually present in the soil and animal alimentary tract and are characterized by having noticeable ability to produce various amounts of potent exotoxins.



Fig. 11 Sudden Death is Common in Enterotoxaemia

### 3.2 Lamb Dysentery

- 3.2.1 Definition: (*Synonyms - Clostridium perfringens* type B enterotoxemia) Lamb dysentery affects young lambs less than three weeks of age. The disease has a short incubation period. Generally, there are two forms of the disease: A) Peracute form: which is characterized by sudden death. B) The acute form: manifested by loss of suckling drive and, recumbency, severe abdominal pain, and fetid yellowish to brown fluid feces or blood - tinged diarrhea as a result of toxinal irritation and increased peristaltic movement. Death usually occurs within 24 hours of the onset of illness.
- 3.2.2 Epidemiology: The disease initially affects 1-4 day old lambs. In endemic areas, lamb dysentery may affect lambs up to 3 weeks of age. Morbidity may reach 20-30 % while case fatality approaches 100 %. The disease occurs in Europe, Asia and Africa, rare or absent in Australia, New Zealand, Infection occurs mostly through ingestion.
- 3.2.3 Diagnosis:
- a) Direct smears and bacteriological culture from the intestinal contents from cases showing typical history and clinical signs.
  - b) Polymerase chain reaction. Detection and identification of beta toxin.
  - c) Gross Lesions: Enteritis with hemorrhagic zones and discrete to confluent ulceration of the mucosa mostly evidenced in the ileum. The intestinal mucosa is dark red and the ulcers are large (up to 2.5 cm in diameter).

- d) Differential Diagnosis: Colibacillosis, Salmonellosis, Cryptosporidiosis, Rotavirus and Coronavirus infections, hemorrhagic enteritis by type C *Clostridium perfringens*.

### 3.2.4 Prevention and Control: Control measures during the outbreaks are:

- a) In newborn animals from unvaccinated dams, antiserum should be administered immediately after birth. Lamb dysentery antiserum will protect against type C infections as well. Dead lambs should be incinerated or buried in quick lime.
- b) All pregnant ewes should be vaccinated at least 2 weeks before giving birth to produce sufficient protective antibodies in the colostrum.
- c) The lambing area should be vigorously cleaned and disinfected.
- d) Long-term control rests with vaccination of the dams. A pregnant dam should be vaccinated during the last third of pregnancy: initially, two vaccinations one month apart, the second injection should be given 2-3 weeks before parturition and annually thereafter.

## 3.3 Pulpy Kidney Disease

3.3.1 Definition: (Synonyms - *Clostridium perfringens* type D enterotoxemia, Overeating disease, milk colic) Pulpy kidney disease is an acute infectious but non-contagious fatal enterotoxemia mostly of suckling lambs caused by the proliferation of *Clostridium perfringens* type D. in the intestine and liberation of epsilon toxin that causes damages in the vascular & nervous systems. Predisposing conditions include consumption of large amounts of milk and excessive concentrate ingestion. In the feedlot, lambs switched rapidly to high-grains diets are mostly affected. There is a rapid clinical course usually ends in sudden death, depression, frothing at the mouth and convulsions and other signs of CNS involvement. Cases which survive for a few hours show green, pasty diarrhea, staggering, recumbency, severe clonic convulsions and coma before death. Adult sheep, usually survive for longer period up to 24 hours. Weakness, incoordination, and convulsions are observed. They lag behind the flock, show staggering and knuckling, champing of the jaws, saliva drips, and rapid, shallow, irregular oral respiration.

3.3.2 Epidemiology: The highest incidence primarily occurs in suckling as well as weaned lambs. It is seen less frequently in goats and rarely in cattle. The disease is worldwide in distribution. Ingestion is main mode of transmission. The prevalence in flocks seldom exceeds 10 %. The case fatality rate approximates 100 %.

### 3.3.3 Diagnosis: There are steps to reach diagnosis:

- a) Direct smears from the intestinal contents
- b) Detection and identification of the toxins
- c) Gross Lesions: Rapid decomposition of the carcasses, hyperemic areas on the intestine. Fluid-filled pericardial sac, hemorrhagic areas on the myocardium may be found as well as petechiae and ecchymoses of the abdominal muscles and serosa of the intestine. The rumen and abomasums contains an abundance of feed, and undigested feed often is found in the ileum. Rapid post-mortem autolysis of the kidney (soft and pulpy) has led to the popular name, pulpy kidney disease.

- d) Differential Diagnosis: In lamb: Acute *pasturellosis*, *Clostridium Sordelli*, Polioencephalomalacia in sheep: Acute poisonings, Black disease, Blackleg, Bloat, rumen overload, hypocalcaemia and pregnancy toxæmia.

3.3.4 Prevention and Control: Good management and properly timed and balanced rations, gradual transition by reduction of concentrate in the diet and proper ration mixing. Immunization passive protection stimulation by administration of epsilon antitoxin 200 units/kg body weight to all sheep as soon as outbreak commence will provide for protective circulating antitoxin levels for 21-29 days.

Breeding ewes should be given two injections of type D toxoid in their first year and one injection, 4-6 weeks before lambing, each year thereafter. This lamb protection is transferred through the ewe's colostrum and can provide immunity for up to 8-10 weeks. A multivalent bacterin-toxoids containing antigens to all of the clostridial diseases is commonly used in sheep. Active immunization of young lambs could be achieved by:

- a) Vaccination of the lambs using type C and D toxoid.
- b) Vaccination must be carried out at the age of 4 - 10 weeks old and again at month later.
- c) Two injections, at the age of 2 - 4 weeks apart, will protect lambs through the feedlot period. The first injection should be administrated prior to the feeding period, and the booster three weeks later.

Treatment is as follows:

- a) Treatment of severe cases is not very rewarding. In goats antitoxins can be given either orally or by injection. Antibiotics such as penicillin may be recommended, but is frequently of little value after clinical signs appear.
- b) Other supportive therapy, such as fluids, vitamins, or cortisones may be prescribed

## 3.4 Malignant Edema

3.4.1 Definition: (Synonyms - Gas gangrene, false blackleg) Malignant edema is an acute, generally fatal toxemia of cattle, horse, sheep, and goats. The disease is usually caused by *Clostridium septicum*; other clostridia implicated in wound infections include *Clostridium chauvoei*, *Clostridium perfringens*, *Clostridium novyi*, and *Clostridium sordelli*. It is characterized by acute onset with fever and toxemia, as well as inflammation and swelling at site of the wound with heat, edema, pain on palpation, and usually subcutaneous emphysema.

Dirty environment and contamination of traumatic deep puncture wounds, castration, docking, unsanitary vaccination, and parturition are the most important predisposing factors. The disease run a stormy course and signs appear within 12-48 hours following predisposing injury and wound infection. There is high fever, stiffness or lameness. The local lesions are soft, doughy, swellings that pit on pressure with erythema accompanied

by severe pain on palpation. The swelling extends rapidly because of the formation of large quantities of exudates that infiltrates the subcutaneous and intramuscular connective tissue of the affected areas. At the later stage, the swelling becomes tense and the skin dark and taut. Emphysema may or may not be present, depending on the type of infection. The mucosae are dry and congested and have very poor capillary refill.

**3.4.2 Epidemiology:** All ages and species of animals are affected, the disease occurs worldwide. *Cl. septicum* is found in soil and intestinal contents. Usually wounds are portals of entry. Infection follows contamination of the contained devitalized tissue, soil, or some other tissue debilitant. The disease occurs sporadically.

**3.4.3 Diagnosis** Direct smears and bacteriological culture of the samples from the lesions. Gross Lesions: Tissue changes occur rapidly after death. Gangrene of the skin with edema of the subcutaneous and intramuscular around the site of infection. The muscle in such areas is dark brown to black. The edema fluid varies from serum like to a gelatinous deposit. It is usually blood-stained and contains bubbles of gas. Except in *Cl. novyi* infections, where there are no gases. Differential Diagnosis: Blackleg disease, Braxy and Anthrax.

#### 3.4.4 Prevention and Control

- a) Hygiene at lambing, shearing, castration and docking is essential.
- b) Disinfection of wounds using effective disinfectants.
- c) Penicillin can be given prophylactically to animals at risk of the disease.
- d) Annual vaccination is indicated in high risk areas and revaccination if severe trauma occurs. In endemic areas, animals should be vaccinated before they are castrated, dehorned, or docking. Calves should be vaccinated at about 2 months of age. Two doses 2-3 weeks apart generally give protection.

### 3.5 Big Head

**3.5.1 Definition:** (*Synonyms - Clostridium novyi* type B infection, swelled head) Big head is an acute, infectious disease, caused by *Cl. novyi*, is characterized by a nongaseous, non – hemorrhagic, edematous swelling of the head, face, and neck of young rams. Mixed infection with other clostridial organisms is common. Incubation period ranges between 1-3 days.

**3.5.2 Epidemiology:** Big head occurs in young rams 6 months to 2 years old when they run in bands and fight among themselves. It is a worldwide disease. Swelled head of rams occurs after infection of wounds inflicted by fighting, shearing or docking.

**3.5.3 Diagnosis:** Direct smears from the lesions, and bacteriological culturing. Gross lesions reveal swelling of the head, face, and the neck as a characteristic sign in post mortem findings. Differential Diagnosis: Malignant edema

**3.5.4 Prevention and Control:** Separation of young rams at fighting age. Disinfection of any wounds or abrasions at the head regions is necessary. Vaccination using multivalent clostridial vaccine is helpful. Treatment with broad-spectrum antibiotics or penicillin may be effective; unless treatment is instituted in early stages the death rate is extremely high.

### 3.6 Blackleg Disease

**3.6.1 Definition:** (*Synonyms: Black quarter, quarter-ill, clostridial myositis of skeletal muscle*) Blackleg disease is an acute febrile infectious disease of cattle and sheep caused by *Clostridium chauvoei* (*feseri*), characterized by sudden death, emphysematous swelling of the upper part of the affected leg and severe lameness. Infection is initiated with trauma which occurs in musculature and bruising. Incubation period is 24-48 hours and onset is sudden. A main clinical sign in cattle is sudden death, and when the animal is observed before death, there is severe lameness with pronounced emphysematous swelling of the upper part of the affected leg. The animal stands with back arched and head lowered. Muscular swelling with characteristic edematous and crepitant swelling develop in the hip, shoulder, chest, back, neck. At the first, the swelling is small, hot, and painful but soon becomes cold and painless, and edema and emphysema can be felt. As the disease rapidly progresses, the swelling enlarges, with crepitation on palpation.

**3.6.2 Epidemiology:** Blackleg is a disease of cattle .It has also been reported to occur in sheep and other animals. Cattle 6 months to 2 years of age are rapidly growing and on high plane of nutrition are highly susceptible. The disease is found worldwide. Contaminated pasture appears to be a source of organisms. Outbreaks have occurred in cattle on farms in which recent excavations have occurred, which suggests that disturbance of soil may activate latent spores. The portal of entry is through the alimentary tract mucosa after ingestion of contaminated feed is associated with erupting teeth. It is more prevalent during the fall, winter, and spring, and during the times when pasture is very good following good rains.

**3.6.3 Diagnosis:** It needs steps

- a) Bacteriological culture of the samples from the typical liver lesions
- b) Detection of *Cl. novyi* by fluorescent antibody techniques or ELISA or PCR

Gross Lesions: Cattle found dead of blackleg are often in a characteristic position; lying on the side with the affected hind limb stuck out stiffly. Bloating and putrefaction occurs quickly. Blood stained froth exudes from the nostrils and anus. Clotting of the blood occurs rapidly. Incision of the affected muscle mass reveals dark red to black, swollen tissue with a rancid odor, this, sanguineous fluid containing bubbles of gas. In sheep the muscle lesions are more localized and deeper and the subcutaneous edema is not so marked except around the head. Differential Diagnosis: Anthrax, Malignant edema and Lighting streak

**3.6.4 Prevention and Control:** It may be successful if it is attempted in the early stage of the disease, namely, during the fever stage. It consists of the injection of large doses of antiblackleg serum into and around the swelling, or, better, intravenously.

- a) Administration of penicillin and surgical debridement of the lesion, including fasciotomy, is indicated if the animal is not moribund.
- b) Large dose (400 000 units /kg BW) should be administered, commencing with crystalline penicillin intravenously and followed by longer-acting preparations.
- c) When outbreaks are encountered, all susceptible cattle should be vaccinated and treated prophylactically with penicillin intramuscularly.
- d) Bacterin containing *Clostridium chauvoei* and *Clostridium septicum* is indicated for both cattle and sheep. Calves should be vaccinated twice, 2 weeks apart, between 3 and 6 months of age.
- e) In sheep in areas where the disease is enzootic, the ewes should be vaccinated twice, the last vaccination given about 1 month before lambing and a subsequent yearly booster dose given at the same time before lambing.

### 3.7 Tetanus

- 3.7.1 Definition: (Synonyms: Locked jaw) Tetanus is an acute, often fatal, infectious non-contagious toxemic disease affecting all mammals caused by an exotoxin (tetanospasmin) produced by *Clostridium tetani*. It is characterized by generalized muscular rigidity and spasm, hyperesthesia, prolapse of the third eyelids, trismus, respiratory arrest, and death. Deep puncture dirty wounds and unsanitary conditions at parturition, docking, shearing and castration are the main precipitating factors, where the growth of causative bacteria is favored under the anaerobic conditions in necrotic tissues.
- 3.7.2 Epidemiology: *C. tetani* is a slender, gram-positive, anaerobic rod that may develop a spherical terminal spore, giving it a drumstick appearance. In contrast, the spores are very resistant to heat, but can be destroyed by heating at 115°C for 20 minutes. The spores are relatively resistant to phenol and other chemical agents. The incubation period varies between 3 days and 4 weeks, with occasional cases occurring as long as several months after the infection is introduced. Clinical findings are similar in all animal species.

- a) Initially, there is an increase in muscle stiffness accompanied by tremor.
- b) The stiffness, often involving the masseter muscles and muscles of the neck, the hind limbs, and the region of the infected wounds is seen first.
- c) There is hyperesthesia with exaggerated responses to normal stimuli and the animal is easily excited by sudden movement or noise.
- d) Generalized stiffness becomes pronounced about one day later, and tonic spasms and hyperesthesia become evident.
- e) Stiffness of the hind limbs causes an unsteady, straddling, gait, and the tail is held out stiffly especially when turning.
- f) There is trismus with restriction of jaw movements and prolapse of the third eyelid. Spasms of head muscles cause difficulty in prehension and mastication of food, hence the common name, locked jaw.
- g) In horses, the ears are erected; the tail stiff and extended, the anterior nares dilated, and the third eyelid prolapsed.
- h) As the disease progresses there is an increase in muscular tetany of the neck and

- back muscles which causes extension of the head and neck. Stiffness of the leg muscles causes the animal to assume a "sawhorse" stance.
- i) General spasms disturb circulation and respiration, which results in increased heart rates, rapid breathing, and congestion of mucous membranes.
- j) Usually, the temperature remains slightly above normal, but it may rise to 41-42°C, towards the end of a fatal attacks.

Horses are the most sensitive of all species and cattle, sheep and goats being less susceptible. Tetanus also affects humans. Occurrence is worldwide, but is most frequently in hot, damp climates with soil rich in organic matter. *C. tetani* is commonly present in the feces of animals especially horses, and in the soil. Tetanus may follow elective surgery, burns, deep puncture wounds, crush wounds, otitis media, dental infection, animal bites, injuries due to rectal palpation, post-castration, post-tail docking, severe skin lacerations and postparturient uterine infections, vaccination or injections of pharmaceuticals especially anthelmintics. Genital tract is the usual portal of entry in cattle at the time of parturition. Neonatal tetanus occurs when there is infection in the umbilical cord associated with unsanitary conditions at parturition. Outbreaks of 'idiopathic tetanus' occur occasionally in young cattle without wound being apparent, usually in association with the grazing of rough fibrous feed.

- 3.7.3 Diagnosis: Direct smears from the wound site, anaerobic culture and detection of tetanus toxin. There are no gross findings by which a diagnosis can be confirmed Differential Diagnosis: In all species: Strychnine poisoning, Meningitis. In horses: Hypocalcaemia, Acute laminitis, Hyperkalaemic periodic paralysis, and Myositis, particularly after injection in the cervical region. In ruminants: Hypomagnesaemia, White muscle disease, Polioencephalomalacia, Enterotoxemia.

Treatment: The main principles in the treatment of tetanus are:

- a) Wounds cleaning and disinfection to eliminate the causative bacteria.
- b) Neutralization of the residual toxin.
- c) Control muscle spasms until the toxin is eliminated or destroyed.
- d) Maintain hydration and nutrition.
- e) Provide supportive treatment.
- f) Intravenous administration of penicillin is recommended.
- g) Tetanus antitoxin
- h) Relaxation of the muscle tetany by
  - i. Chlorpromazine (0.4-0.8 mg/kg BW IV, 1.0 mg/kg BW IM, t.i.d. to q.i.d.) and acetyl promazine (0.05 mg/kg BW b.i.d.)
  - ii. A combination of diazepam (0.1-0.4 mg/kg) and Xylazine (0.5-1.0 mg/kg IV or IM) may be effective in horse refractory to phenothiazine tranquilizers.
  - iii. Supportive treatment best attained by good nursing during the acute period of spasms. Affected animal should be kept as quiet as possible and provided with dark, and plenty of room with non-slip flooring to avoid injury if convulsions occur. Administration of enemas and catheterization may relieve the animal's discomfort.

j) Adequate rehydration and balanced nutrition.

3.7.4 Prevention and Control: Many cases of tetanus could be avoided by proper skin disinfection at surgical operations, castration, docking, and shearing, these should be carried out in clean surroundings. Only the oxidizing disinfectants such as iodine or chlorine are reliable in killing the spores.

a) Passive immunity:

- i. If the animal or horse with a penetrating wound has not been immunized previously, it should be treated with 1,500-3000 IU or more of tetanus antitoxin, which usually provides passive protection for up to 2 weeks.
- ii. Tetanus antitoxin must routinely be given to mares following foaling and to newborn foals. In high-risk areas tetanus antitoxin is given immediately after birth and every 2-3 weeks until they are 3 months old, at which time they can be given toxoid. In farms where incidence of tetanus in lambs is high, antitoxin is usually given at the time of docking or castration; 200 IU has been shown to be effective.

b) Active immunity:

- i. By tetanus toxoid and yearly booster injections of toxoid for horses are advisable.
- ii. Mares should be vaccinated during the last 6 weeks of pregnancy to provide the mare with active immunity and foals with passive colostral immunity, which protect them during the first 10-12 weeks of life. They should receive primary vaccination at 3-4 months of age.

### 3.8 Septicaemic Pasteurellosis

3.8.1 Definition: (Synonyms - Hemorrhagic Septicemia Barbone) Acute contagious highly fatal septicaemic disease of cattle, camels and buffaloes caused by certain serotypes of *Pasteurella multocida* e.g 1(B), 4(D) and 1,2(E). The disease is characterized by sudden deaths, high fever, salivation, hot painful subcutaneous swellings, mucosal petechiae and death in about 24 hours. The precipitating factors of the disease are radical changes in weather, debility and low nutrition, the pressure of work (draft animals), rainy season and poor physical condition.

3.8.2 Epidemiology: *Pasteurella multocida* is a small Gram negative rod or coccobacillus. The incubation period is usually 1-3 days. The majority of cases in cattle and buffalo are acute or peracute with death occurring after 6-24 hours after recognition of signs. The clinical signs are:

Sudden onset of fever (41-42 C), dullness, reluctance to move, profuse salivation, nasal discharge, submucosal petechiation, severe depression and death within 24 hours. In a few outbreaks, animal may survive as long as 72 hours. Localization may occur in subcutaneous tissue, resulting in the development of warm, painful swellings around the

throat, dewlap, brisket, or perineum. Edematous swellings are seen in the pharyngeal region and then spread to the ventral cervical region. Severe dyspnea may occur if the respiration is obstructed, congestion of visible mucous membranes which is soon followed by collapse and death. In the later stages of an outbreak, some animals develop signs of pulmonary or alimentary involvement.

The disease principally affects cattle and buffaloes. The most susceptible age group is animals between 6 months to 2 years. Some serotypes of *Pasteurella multocida* can cause a variety of human infections. The disease occurs in southern Europe, Russia, Africa and Asia, where it causes heavy death losses. It spreads by direct and indirect contact through the ingestion of contaminated foodstuffs and water.

3.8.3 Diagnosis:

- a) Differential Diagnosis: Anthrax, Blackleg and Acute leptospirosis
- b) Bacteriological examination.
- c) Lab animal inoculation.
- d) Identification or serotyping.
- e) Gross lesions reveal generalized petechial hemorrhages particularly under the serosae. Lymph nodes in the thorax region are enlarged and hemorrhagic. Edematous swelling of the head, neck, and brisket region. Incision of the edematous swellings shows a coagulated serofibrinous mass with straw-colored or blood-stained fluid. This edema, which distends tissue spaces, is also found in the musculature. Petechiae may be found scattered throughout the pharyngeal and cervical nodes, which are also swollen and often hemorrhagic. There are lesions of hemorrhagic gastroenteritis and early pneumonia.

3.8.4 Prevention and Control: Several of the sulfonamides and antibiotics such as penicillin and the Oxytetracycline can be used successfully in the early stages. Notification as well as isolation and treatment of affected animals and avoidance of stress factors are important. Sanitary precautions and hygienic disposal of the carcasses.

In endemic areas the only practical ways to protect animals are by an organized program of vaccination using a stable vaccine composed of formalin-killed organisms prepared from appropriate serotype in an oil adjuvant base containing paraffin and lanolin.

### 3.9 Pneumonic Pasteurellosis

3.9.1 Definition: (Synonyms - Shipping fever, transient fever) Pneumonic pasteurellosis is an acute highly contagious respiratory bacterial disease of cattle caused by *Pasteurella hemolytica* biotype A serotype 1 and, less commonly *Pasteurella multocida*. It is characterized by sudden death, acute bronchopneumonia, fever, toxemia, anorexia, abnormal lung sounds and response to treatment with antimicrobials. *Pasteurella hemolytica* and *Pasteurella multocida* also cause bronchopneumonia in young sheep and goat characterized by cranioventral lung distribution. In adult sheep *Pasteurella hemolytica* biotype A causes

pneumonia while biotype T causes septicemia. Predisposing factors are transportation over long distances, exhaustion, starvation and dehydration, chilling or overheating, improper animal housing, nutritional stress associated with a change to high-energy rations in the feedlot and primary viral infection. Biotype T strains have been isolated from cases of pneumonic pasteurellosis and occasionally *Pasteurella multocida*. The incubation period of the disease is usually 3-7 days.

- 3.9.2 Epidemiology: Bovine pneumonic pasteurellosis is seen principally in young growing, newly housed, recently weaned beef calves from 6-8 months of age, shortly (1-3 weeks) after being placed into feedlots. Beef cattle are at greater risk than young dairy cattle. Nursing beef calves, yearlings and mature dairy and beef cows may also be affected but less frequently. Pneumonic pasteurellosis is world-wide distributed disease. It is a common disease in Europe, United States and Canada. Transmission of the disease principally occurs by the inhalation.

Animals found dead without any previous signs may be the first indication of an outbreak. When viewed from a distance, affected cattle are depressed with rapid and shallow respirations. There is pyrexia (40 - 41 C) and difficult breathing. There may be a weak protective cough which becomes more pronounced and frequent if animals urged to walk. A muco-purulent nasal discharge, a crusty nose and ocular discharge are common. In the early stages, there are loud breath sounds over the anterior and ventral parts of the lungs. As the disease progresses, increased breath sounds, crackles, and wheezes are observable on auscultation of the cranioventral lung field. In severe cases, pleurisy develops and duration of the dyspnea is marked which is characterized by an irregular breathing pattern and grunting on expiration.

- 3.9.3 Diagnosis: Differential Diagnosis includes Bovine herpes-1 virus disease, Bovine respiratory syncytial virus infection, Bovine viral diarrhea, Parainfluenza-3 virus, Corona virus, adenovirus, and *Pasteurella hemolytica* infection.

- a) Bacteriological and Hematological examination.
- b) Gross Lesions: Extensive reddish black to grayish brown cranioventral regions of consolidation with gelatinous thickening of interlobular septa and fibrinous pleurisy. Extensive thromboses, foci of lung necrosis, and limited evidence of bronchitis and bronchiolitis. Lungs are firm and the cut surface usually reveals an irregular pattern of red, white, and grey tissue due to hemorrhage, necrosis, and consolidation. Fibrinous pleuritis is accompanied by a fibrinous pericarditis.

- 3.9.4 Prevention and Control: The use of anti-inflammatory drug, Flunixin meglumine in combination with Broad-spectrum antibiotics such as Oxytetracycline, Trimethoprim -sulfadoxine has definite therapeutic benefits. Mixing of cattle from different sources at livestock market should be avoided. Transport time should be minimized, and rest periods with access to food and water, should be provided during prolonged transport. Calves should be weaned 2-3 weeks before shipping. Adaptation to high-energy rations should be gradual. Preventive medication with antibiotics is a consideration for suspected cattle

judged to be at arrival to the feedlot. Early identification, isolation and treatment of infected animal minimize the spreading of the infection in the herd. Vaccinations for the viral and bacterial components of shipping fever pneumonia should be given 2-3 weeks before transport and can be repeated on entry to the feedlot.

### 3.10 Brucellosis

- 3.10.1 **Definition:** (Synonyms - Contagious abortion, Bang's disease, Malta fever, Undulant fever, Mediterranean fever) Brucellosis is highly contagious, bacterial, zoonotic disease, widespread in most countries of the world, affecting cattle, buffalo, bison, camel, sheep, goats, deer, dogs and horses in addition to human.

The disease in animals is characterized by abortion in late pregnancy, retained placenta, acute metritis and infertility in female and inflammation of the accessory sex organs in male.

- 3.10.2 Epidemiology: The species of Brucella and their principal farm animal host are listed below:

- |    |  |                 |
|----|--|-----------------|
| a) | <i>B. abortus</i> , <i>B. melitensis</i> , <i>B.</i> | Cattle& buffalo |
| b) | <i>B. melitensis</i> , <i>B. ovis</i>                | Sheep           |
| c) | <i>B. melitensis</i>                                 | Goats           |
| d) | <i>B. abortus</i> , <i>B. suis</i>                   | Horse           |
| e) | <i>B. abortus</i> , <i>B. melitensis</i>             | Camels          |

Brucella is a Gram negative bacteria, aerobic coccobacilli or short rods (0.6-1.5 µm long by 0.5-0.7 µm in width, non motile, non encapsulated and non spore forming, The incubation period in cattle is quite variable, ranging from 2 weeks to 7 or 8 months or even longer. Infected animal usually aborts once and rarely twice or three times.

Later on the animal becomes carrier to the disease, secretes the organisms in the milk, urine, and vaginal secretion. Orchitis and epididymitis in male may involve both scrotal sacs, with acute painful swelling to twice the normal size and the testes are eventually destroyed. Brucellosis affects all farm animals' particularly pregnant females, due to fact that the gravid uterus sustains growth of Brucella by secretion of Erythritol. The disease is now diagnosed in more than 175 countries. Brucellosis is an important zoonosis worldwide and readily transmissible to humans. It causes an acute febrile illness (*B.abortus*: undulant fever and *B.melitensis*: Malta fever), which may progress to more chronic form. Infection is often due to occupational exposure and may be via the conjunctiva or abraded skin, but ingestion of dairy products constitutes the main risk to the public. The prevalence

of infection varies considerably between herds, areas, and countries. Transmission occurs via oral route, penetration of the intact skin and conjunctiva, contamination of the udder during milking, and from infected bull semen. Calves are likely exposed to *Brucella* organisms in utero or when calves born to healthy dams are fed on colostrum or milk from infected dams. *Brucella* can survive for several months at 4-8 C, at zero C for 2- 5 years and in frozen tissues for several years. *Brucella* is sensitive to direct sun light, pasteurization and common disinfectants.

### 3.10.3 Diagnosis

- a) Direct smear from placenta, cotyledon, vaginal discharge and fetal lung, liver and abomasal contents
- b) Bacteriological examination- PCR and ELISA.
- c) Serological tests include milk ring test, serum agglutination test, Rose Bengal plate test, complement fixation test, and ELISA are used for diagnosis.

**Gross Lesions:** The placenta is usually edematous. There may be leathery plaques on the external surface of the chorionic membrane. Affected cotyledons may be normal to necrotic, reddish or yellow. Thickening of the intercotyledony spaces, which exhibit wet and leathery appearance are distinct lesions.

The fetus may be normal or autolytic with bronchopneumonia. There is sero- hemorrhagic fluid in the body cavity, and pneumonia is the constant finding. Often there is granulomatous lesions and focal necrosis in several internal organs.

**Differential Diagnosis:** In cattle: Campylobacteriosis, Leptospirosis, Listeriosis, Chlamydiosis, Trichomoniasis, Infectious bovine rhinotracheitis. In sheep and goats Campylobacteriosis, Listeriosis, Chlamydiosis, Rift valley fever, Blue tongue, pestis des petits ruminants, Border disease, Tick borne fever, Toxoplasmosis.



Fig. 12 Abortion caused by Brucellosis

**3.10.4 Prevention and Control:** Treatment is unsuccessful because of the intracellular sequestration of the organisms in the lymph nodes, the mammary glands, and reproductive organs. Biosecurity and good hygienic conditions in terms of cleaning and disinfection are essentials.

- a) Serological examination of all animals inside the farm should be carried out and positive animals should be culled. The test should be repeated after one month, and after two successful negative test results, the farm is free from the disease. Retest every three months, then every six months.
- b) Avoidance of introduction of new pregnant animals directly to the farm, unless quarantined for three weeks after delivery, and then tested for the disease.
- c) Avoidance of the introduction of new non pregnant animals directly to the farm, unless quarantine them for one month and test against the disease, and if get two successful negative results, the animal is free from the disease.
- d) Immediate isolation of the abortion cases and subjected them to laboratory examination in order to make sure that the aborted animal is free from the disease. Immediate removal and burning all the placenta and aborted fetus.
- e) It is advisable to check the male used for natural insemination. In countries with endemic brucellosis, all calves (4-8 months) should be vaccinated by strain 19, and adult cattle by strain 45/20A or RB51. All ewes in the farm should be vaccinated by Rev 1 vaccine

## 3.11 Colibacillosis

**3.11.1 Definition:** (Synonyms- *Escherichia coli* infection, White Scour Disease) Colibacillosis is a very common and serious contagious bacterial disease of neonatal calves within the first 3 days of life. Other farm animals are also susceptible. The disease is caused by pathogenic serotype of *Escherichia coli* (E.coli). Most calves are affected, several forms of Colibacillosis occur with some variation in the symptoms. While septicaemia and rapid death are the distinguishing features of septicaemic Colibacillosis, enterotoxic Colibacillosis is characterized by diarrhea, dehydration, and toxemia of varying severity. Inadequate colostrum intake, lack of specific antibodies in dams, stress factors such as poor environmental conditions and insufficient inappropriate diet are the main predisposing factors.

**3.11.2 Epidemiology:** The most common enteropathogens which cause diarrhea in newborn farm animals are the Enterotoxigenic E.coli (ETEC) which are not invasive and cause diarrhea by adhering and colonizing the small intestine, and producing enterotoxins. Enterohemorrhagic E.coli (EHEC) is uncommon cause of disease in newborn farm animals and attaches to the colon and distal small intestine "attaching and effacing E.coli result in hemorrhagic colitis. They are also known as Verocytotoxic E.coli (VTEC) because they produce Verocytotoxins. Necrotoxigenic E.coli (NTEC) produces Cytotoxic Necrotizing Factors (CNF) eg. (CNF1 and CNF2), NTEC, 2 isolates are restricted to calves and lambs with diarrhea and septicemia. E.coli strains serogroup - 078 are invasive and cause septicaemia in calves and lambs. Their powerful endotoxins cause endotoxic shock with a high case

fatality rate. Generally, there are two types of colibacillosis, coliform septicaemia and enterotoxigenic colibacillosis.

**Coliform septicaemia:** This form occurs most commonly in calves during the first 4 days of life.

- a) The illness is peracute, the course varying from 24 to 96 hours with sudden death and high case fatality. Affected animals are depressed, commonly recumbent, and dehydrated. Tachycardia is evident.
- b) Although the body temperature may be high initially, it falls rapidly to subnormal levels when the calf becomes weak and moribund. The oral mucous membranes are dry and cool. Scleral injection is common. Diarrhea and dysentery may occur but are uncommon.
- c) If a calf survives the septicaemic state, clinical evidence of post septicaemic panophthalmitis and pneumonia are very common.
- d) The most frequent clinical signs in neonatal meningitis are lethargy, anorexia, and recency, loss of the suck reflex, stupor, and coma. Opisthotonus, convulsions, tremors, and hyperesthesia are less frequent. Case fatality rate is usually 100 %.

a) **Enterotoxigenic Form of Colibacillosis:**

- i. This is the most common form of Colibacillosis in newborn calves.
  - ii. It occurs primarily from 3-5 days of age. It may occur in calves as early as 1 day of age and calves up to 3 weeks.
  - iii. There are two clinical forms of this type :
- b) **Peracute Form of Colibacillosis (enteric toxemia):** The presence of a single enterotoxigenic strain of E.coli may cause a state of collapse usually designated as enteric toxemia. In this form, the clinical signs include:
- i. Severe weakness, subnormal body temperature, and coma.
  - ii. Pale mucosa and wetness around the mouth.
  - iii. Diarrhea is usually not evident although the abdomen may be slightly distended and auscultation may reveal splashing sounds suggesting a fluid filled intestine.
  - iv. Prognosis for these calves is poor and they commonly die 2-6 hours after onset of signs

c) **Acute Form of Colibacillosis:** It is a common form of the disease in calves. There is diarrhea in which the faeces are profuse and watery, usually pale yellow to white in color and occasionally streaked with blood and it has a very foul-smelling. Defecation is frequent and effortless. The tail and perineum are soiled with feces. The temperature is usually normal in the initial stages but later become subnormal. Affected calves may or may not suck or drink depending on the degree of acidosis, dehydration and weakness. In the early stage

of the disease, the abdomen may slightly distend due to fluid -filled intestine. Affected calves can lose 10-16 % of their original body weight during the first 24 -48 hours of the diarrhea. In calves with 8 % dehydration, 5-10 seconds will be required for the skin fold to return to normal; in 10-12 % dehydration, it takes up to 30 seconds. The clinical signs of dehydration occur first when the fluid loss reaches 5-6 % of the body weight.

10 % loss of fluid results in depression, sunken eyes, dry skin, and the calf will probably be unable to stand. A 15 % loss of fluid usually results in death. Veal calf hemorrhagic enteritis is a fatal syndrome characterized by anorexia, fever, diarrhea with mucous containing faeces which become bloody in the later stages, and hemorrhagic diathesis on the conjunctivae and mucous membranes of the mouth and nose. Typically, calves out of first calf heifers are more susceptible. Other associated factors include seasonal variation, overcrowding and poor sanitation.

**Colostrum-deprived calves** are highly susceptible to infection with enteropathogens. The disease is seen in all animals and occurs worldwide. Enteropathogens associated with diarrhea are commonly found in the faeces of healthy calves; whether intestinal infection leads to diarrhea depends on a number of determinants, including differences in virulence of different strains of a pathogen and the presence of more than one pathogen. Healthy adult cattle may be carriers and periodically shed the organism in faeces. Excretion may increase with the stress of parturition. Other sources of infection include the faeces of healthy calves and the faeces of diarrheic calves, which contain large numbers of organisms early in the course of infection.

### 3.11.3 Diagnosis:

Blood culture specially in septicaemic colibacillosis. Fecal microbiological culture is needed in case of enteric colibacillosis. Hematology and serum biochemistry are required.

**Gross Lesions:** In Coliform septicemia: Subserosal and submucosal hemorrhages accompanied by enteritis and gastritis. Fibrinous exudates are found in the joints and serous cavities. Pneumonia and meningitis might be present.

**In Enteric Colibacillosis:** The carcass is dehydrated; the intestine is flaccid and fluid- filled. The abomasum is usually distended with fluid and may contain milk clots and numerous small hemorrhages. The intestinal mucosa may be hyperemic and there may be edema of the mesenteric lymph nodes.

**Differential Diagnosis:** Rotavirus and Coronavirus infections, Cryptosporidiosis and dietary diarrhea.

### 3.11.4 Prevention and Control:

Treatment is based on isolation and good nursing. Rehydration: Oral and parenteral fluid electrolyte therapy. Antimicrobial therapy with broadspectrum antimicrobials such as Neomycin sulphate, oxytetracycline, sulfonamides, Trimethoprim-sulfonamide mixture, and Ampicillin are used based on previous successful experience. Immunity enhancement, by colostrum administration is started within

the first 6-8 hours after birth. Vaccination program must be developed based on a good diagnostic knowledge of the diseases present in the herd. More recently, with the development of a vaccine containing monoclonal antibody to ETEC, it can offer immediate protection in the face of colibacillosis outbreaks if administered as soon as possible following birth (within the first 8-12 hours).

### 3.12 Salmonellosis

3.12.1 Definition: (Synonyms - Paratyphoid) Salmonellosis is an infectious common problem in farm animals, particularly newborn dairy calves. Salmonellosis is caused by many species of *salmonellae* and characterized clinically by one or more of three major syndromes: septicemia, acute enteritis, and chronic enteritis.

3.12.2 Epidemiology: Young animals (Calves & lambs or kids) usually develop the septicaemic form. Factors that facilitate infection include : low colostrum intake , sudden deprivation of feed and water , overcrowding , chilling , contaminated feed or water supply , inadequate cleaning and disinfecting, long transportation , parturition , drought and prolonged surgical procedures . *Salmonellae* are classified into three main groups, based on their association with human and animal hosts. The first group is characterized by specificity for the human host includes *S.typhi* and *S.paratyphi* A and C. The second group consists of serotypes which adapted to specific animal hosts, such as *S.dublin* in cattle, *S.abortovis* in sheep and *S.abortsequi* in horses. The third group consists of unadapted serotypes that cause disease in humans and a variety of animals as *S.typhimurium*. In ruminants, two clinical forms exist: a septicaemic and an enteric form.

- a) Septicemia Form: Septicemia is the usual syndrome in newborns characterized by marked depression and high fever. Pneumonia, polyarthritis, and meningitis are potential consequences of septicaemia. Death within 24- 48 hours and mortality may reach 100 %.
- b) Enteric Form: This form includes:
  - i. Acute enteritis: Acute enteritis is the common form in calves, usually  $\geq$  1- 2 week old. It also occurs in adults. Initially, there is fever (40.5 - 41.5 C), followed by severe watery diarrhea, sometimes dysentery, and often tenesmus. The faeces, which vary considerably, may have a putrid odor and contain mucus, fibrinous casts, shreds of mucous membrane, and in some cases, large blood clots. Adult cattle generally develop enteric Salmonellosis, and very rarely develop septicaemia .It is characterized by depression, fever, decreased milk production and severe diarrhea.

Abortion is a common manifestation of Salmonellosis in cattle between 124 - 270 days of gestation. Horses are usually plagued by severe abdominal pain, and become severely dehydrated and may die within 24 hours of the onset of diarrhea; mortality may reach 100 %.

ii. Subacute enteritis: Subacute enteritis may occur in adult horses and sheep on farms where the disease is endemic. Signs are mild fever (39 - 40 C), soft faeces, inappetance, and some dehydration. There may be a high incidence of abortion in cows and ewes and some deaths in ewes after abortion. There is high mortality rate due to enteritis in lambs under a few weeks of age. In cattle, the first signs may be fever and abortion, followed several days later by diarrhea.

iii. Chronic enteritis: Chronic enteritis is common in adult cattle. There is persistent diarrhea, severe emaciation, intermittent fever, and poor response to treatment. The faeces is scant and may be normal or contain mucus, casts or blood. In older animals, the disease is manifest by dysentery and toxemia.

Salmonellosis can affect all ages of domestic animal species, where diarrhea is a constant feature. It usually affects calves between 10 days and 3 months of age, but most severe in calves under a month of age. It is important to realize that *Salmonella* is a zoonotic disease and is one of the most important animal-borne bacterial infections in people. Transmission to man is mainly through ingestion. Contaminated poultry meats and eggs are particularly important sources of infection. Salmonellosis has been recognized in all countries, especially in areas of intensive animal husbandry. The source of salmonella infection in a herd can be from other cattle, wild birds, cats, rodents, the water supply, and chronic human or animal carriers. Faeces of infected animals can contaminate feed and water, milk, fresh and processed meats from abattoirs, plant and animal products used as fertilizers or feedstuffs pasture and rangeland, and many inert materials.

3.12.3 Diagnosis: Differential diagnosis considers many diseases. Septicemic salmonellosis should be differentiated from Coliform septicemia, acute enteric salmonellosis in young calves: Diarrhea due to ETEC, diarrhea due to Rotavirus, coronavirus and other viruses, cryptosporidiosis, Hemorrhagic enteritis due to *Clostridium perfringens* type B and C, Coccidiosis, Dietetic diarrhea. Acute enteric Salmonellosis in adult cattle should be differentiated from mucosal disease, winter dysentery, and ostertagiasis. Chronic enteric Salmonellosis: should be differentiated from Paratuberculosis, arsenic poisoning, secondary copper deficiency. When abortion occurs it should be differentiated from Brucellosis, trichomoniasis, vibriosis, leptospirosis, listeriosis, chlamydiosis and mycotic abortion.

Lab. findings include:

- a) Bacterial culture and identification.
- b) Detection of the organism by Antigen capture ELISA or PCR. Serological tests like tube agglutination test.

In postmortem findings the septicemic form gross lesions include reddened or necrotic intestinal mucosa with a fibrin coating. Regional lymph nodes are swollen and hemorrhagic. There are loose, bloody feces in the bowel lumen. There are usually extensive submucosal and subserosal petechial hemorrhages. Lesions are most severe in the lower ileum and the large intestine.

3.12.4 Prevention and Control: Treatment of Septicemic Salmonellosis is best achieved by broad-spectrum antibiotics. Trimethoprim sulfonamide combinations are often effective. Alternatives are Ampicillin, fluoroquinolones, or third generation cephalosporins. Treatment should be continued daily for up to 6 days. Effective treatment of enteric salmonellosis on the other hand can be successfully attained by antibiotics therapy, fluid rehydration therapy to correct acid-base imbalance, in addition to non-steroidal anti-inflammatory drugs. It is important to perform bacteriologic sensitivity test to determine the antibiotics of choice. In practice, ceftiofur is an excellent initial choice because it is effective, approved and affordable.

Generally, three broad principles should apply in all herds:

- a. Control of exposure of neonates by practicing good general hygiene.
- b. Non-specific resistance should be maximized by providing good and adequate nutrition and assuring that newborn calves consume high-quality colostrum within 6 hours of birth and throughout the following days.
- c. Vaccination of the dam or the newborn to enhance specific resistance.

### 3.13 Anthrax

3.13.1 Definition: (Synonyms - malignant carbuncle, splenic fever, Wool sorter's disease). Anthrax is an acute, highly fatal, febrile contagious bacterial disease of all warm-blooded animals including man, caused by a large spore-forming bacterium *Bacillus anthracis*. Most commonly, it is manifested as a septicaemia characterized principally by a rapidly fatal course, sudden death, and fever. The oozing of black, tarry blood from natural orifices just after death is characteristic.

3.13.2 Epidemiology: The causative organism, *Bacillus anthracis*, is a large, gram-positive, facultative anaerobic, non-motile, spore-forming, encapsulated, rod-shaped bacterium, which is stored in long-living spores maintaining the disease on a farm for many years. Pathogenic strains have three plasmid encoded virulence factors. *B. anthracis* spores are resistant to extremes of temperature, chemical disinfectants, and desiccation. Their spores resist destruction and remain viable in soil and animal products for decades. The incubation period is generally 3-7 days in all animals. In cattle and sheep, only two forms of the disease occur, peracute and acute.

Peracute form is characterized by: a) Sudden onset and rapidly fatal course being probably only 1-2 hours, b) Sudden death: death may occur in cattle and sheep without any previous evidence of illness.

If the animal is observed shortly before death, fever up to 42 C, muscle tremors, staggering, dyspnea, trembling, and mucosal convulsions, collapse are common signs.

Following death, unclotted blood may exude from the anus, vulva, nostrils, and / or mouth.

Incomplete rigor mortis is also common.

Acute form:

- a) Acute anthrax destroys animals in a few hours 24 - 48 hours.
- b) There is an abrupt rise in body temperature (42 C) and a period of excitement followed by depression, stupor, respiratory or cardiac distress, staggering, convulsions and death.
- c) The respiration is rapid and deep, the mucosa congested and hemorrhagic, and the heart rate is much increased.
- d) No food is taken and ruminal stasis is evident.
- e) Pregnant animals may abort.
- f) Local edema of the tongue and edematous lesions in the region of the throat, sternum, perineum, and flanks may occur.

The disease in horse is always acute but varies in its manifestations with the mode of infection. When infection is by ingestion there is septicemia with enteric and colic symptoms. In case it occurs by insect transmission, hot, painful, edematous, subcutaneous swellings appear about the throat, lower neck, floor of the thorax and abdomen, prepuce and mammary glands. There is fever, chills, anorexia, depression and weakness. Dyspnea due to swelling of the throat or colic due to intestinal irritation may be witnessed. The course is 48 - 72 hours and death usually occurs within 2 - 3 days of onset. The disease occurs in all vertebrates but is most common in cattle and sheep and less frequent in goats and horses. Swine, dog and cats are less susceptible. Anthrax also occurs in exotic wildlife such as hippos, elephants, and Cape buffalo. Human infection is usually through the skin. Veterinarians may develop localized cutaneous lesions (malignant carbuncle) from contact of broken skin with infected blood or tissues. Inhalation of the spores may result in pulmonary anthrax (wool sorters disease), which is often fatal. Anthrax occurs worldwide, irregularly distributed in districts especially in tropical and sub-tropical countries with high rainfalls where environmental conditions favor spores survival resulting in repeated outbreaks. In some countries these occur every summer. In cool climates only sporadic outbreaks derive from the soil borne infection. *B. anthracis*, when exposed to air, forms extremely resistant spores that can survive for decades under a variety of conditions. Infection occurs by ingestion, inhalation or through the skin. Anthrax spores are very resistant to inactivation and can persist in soil for many years, particularly in warm climates and in calcareous soils with a neutral to alkaline pH and which contain organic matter. The spores may also persist for long periods in contaminated animal products such as hides, hair, or wool, which may also be sources of infection. Epidemics tend to occur in association with marked climatic or ecological change, such as heavy rainfall, flooding, or drought.

3.13.3 Diagnosis: Direct blood smears, bacteriological culture of the blood samples and Identification of the agent by fluorescent antibody test, PCR and Ascoli's test.

Note: Postmortem examinations should not be undertaken on suspected anthrax cases (including any cow that has died suddenly for no apparent reason) until a blood smear has proved negative. If a carcass is opened accidentally, the spleen is usually swollen and there is bloodstained fluid in all body cavities

Differential Diagnosis: There are some diseases with similar clinical findings like, Lighting strikes, peracute blackleg, malignant edema, Bacillary Hemoglobinuria, acute leptospirosis, peracute lead poisoning and hypomagnesaemic tetany.

3.13.4 Control and Prevention: Specific control and prevention procedures are necessary to contain the disease and prevent its spread. These include the following:

- Notification of the appropriate regulatory officials.
- Rigid enforcement of quarantine on the affected farm.
- Prohibition of movement of milk and meat from the affected farm during the quarantine period.
- Prompt disposal of dead animals, manure, bedding, or other contaminated material by cremation or deep burial. Burial should be at least 2 meters deep with an ample supply of quicklime added.
- Proper vaccination of the rest of the flock or herd, using effective like non-encapsulated Sterne-Strain vaccine.

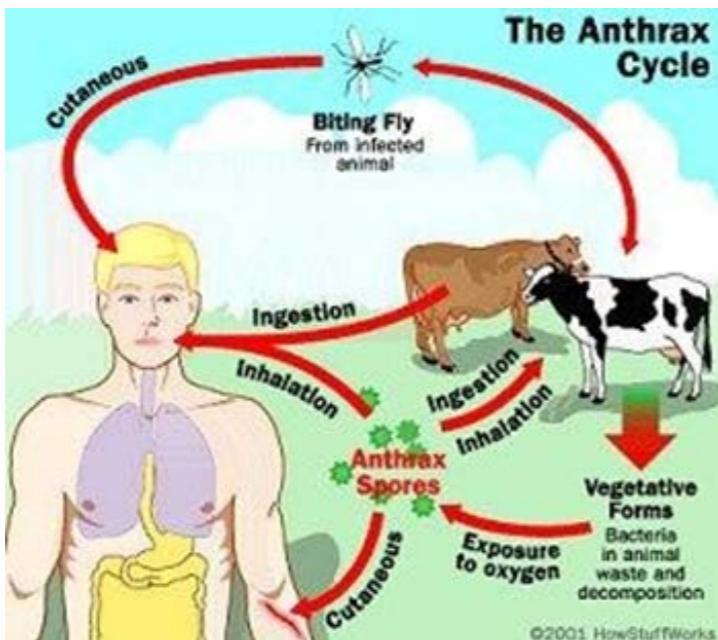


Fig. 14 Anthrax zoonotic Cycle

### 3.14 Listeriosis

3.14.1 Definition: (Synonyms: Circling disease, Silage sickness) Listeriosis is an infectious sporadic zoonotic, often fatal bacterial disease of ruminants, particularly sheep, caused by *Listeria monocytogenes*. The most characteristic clinical syndromes are: encephalitis or meningoencephalitis in adult ruminants, abortion and prenatal mortality in all species, septicaemia in neonatal ruminants and monogastric animals.

*Listeria monocytogenes* is a small, motile, gram positive nonspore forming cocco- bacillus.

3.14.2 Epidemiology: This ubiquitous saprophyte lives in a plant-soil environment and can be found in soil, vegetables, sewage, genital secretions and nasal mucous of apparently healthy animals. The organism is very resistant to drying and can survive up to two years in dry soil and feces. It grows well under a wide range of temperatures, 4 - 44° C. The incubation period is 3 - 7 days. Sheep and goats are most susceptible with death occurring 4 - 48 hours after the onset of clinical signs. Usually the disease occurs in an outbreak of either encephalitis or abortion. Encephalitis is the most common prevalent manifestation in sheep. In cattle, the disease is sporadic, less acute and most survive for 4-14 days. There are four common manifestations of listeriosis: encephalitis in adult ruminants, septicaemia in monogastric and neonatal ruminants, abortion and prenatal deaths in all species, and mastitis in ruminants.

**Listerial encephalitis:** Lesions are localized in the brain stem, and signs indicate dysfunction of the third to seven cranial nerves. Fever (40 - 41 C) is usual in the early stages of the disease, but the temperature is usually normal when overt clinical signs are present. Signs vary between individual sheep but incoordination, head deviation sometimes with head tilt, walking in circles, unilateral facial paralysis is usually present. Facial paralysis with a drooping ear, deviated muzzle, flaccid lip, and lowered eyelid often develops on the affected side, as well as lack of a menace response and profuse continuous salivation; food material often becomes impacted in the cheek. There may be keratitis and corneal ulceration. The animal may stand for long periods drooling saliva and with food hanging from its mouth. Affected sheep usually walk in a circle in the direction of the deviation and the circle of small diameter. There is ataxia, often with consistent falling to one side, and affected sheep may lean against the examiner or a fence. Terminally affected animals fall and, are unable to rise, lie on the same side; involuntary running movements are common. Death is due to respiratory failure. Listerial abortion usually occurs in the last trimester without premonitory signs. Fetuses usually die in utero, but stillbirths and neonatal deaths occur. The abortion rate varies and has been up to 20 % in sheep flocks. Septicaemic listeriosis usually occurs in monogastric animals, including newborn lambs and calves. It has a rapid, fatal course of 3-4 days. All age and breeds, particularly of sheep and cattle are susceptible, although occasional septicemia occurs in horse. In human, listeriosis may occur as a sporadic disease or as a food-borne outbreak (e.g. improperly pasteurized milk) that results in septicaemia, meningoencephalitis, abortion, and infections in other organs. Listeriosis is a disease with world-wide prevalence, more frequently in temperate and cold climates. In general, the disease is more frequent in winter and early spring and has been associated with silage feeding. The natural reservoirs of *L. monocytogenes* appear to be soil and mammalian gastro-intestinal tracts, both of which contaminate vegetation and soil. Animal to animal transmission occurs via the fecal-oral route.

3.14.3 Diagnosis: Bacteriological examination of specimens from brain, aborted placenta and fetus. Fluorescent antibody or immunoperoxidase tests with histopathological examinations. Gross Lesions: In septicaemic listeriosis, the liver consistently contains characteristic multiple, pinpoint, grey-white necrotic foci. While in listerial encephalitis, there is some congestion of meninges. In aborted fetuses, there is slight to marked autolysis, clear to blood-tinged fluid in the serous cavities, and numerous small necrotic foci (0.5 - 2 µm) in the liver.

Differential Diagnosis: Pregnancy toxemia in ewes or ketosis in cattle, bovine spongiform encephalopathy, rabies, lead poisoning, brain abscesses and coenurosis.

**3.14.4 Control and Prevention:** Treatment of clinical cases using antibiotics, because of susceptibility of the organism to penicillin, ceftiofur, erythromycin and Trimethoprim/sulfonamide .The intravenous injection of chlortetracycline (10 mg/kg body weight per day for 5 days) is reasonably effective in meningoencephalitis of cattle but less so in sheep. Penicillin G should be given at 44,000 IU /kg body weight, IM, daily for 1-2 weeks; the first injection should be accompanied by the same dose given IV. Supportive therapy, including fluids and electrolytes, is required for animals having difficulty eating and drinking.

In an outbreak, affected animals should be segregated and treated as early as possible. Carcasses should be hygienically disposed off. Change of diet to include heavy feeding of silage should be made slowly. Silage removed from the clamp should be fed as soon as possible and spoiled silage should be avoided .Tetracyclines can be fed in the ration of animals at risk in a feedlot. A live attenuated vaccine has been shown to induce protection against listeriosis; also commercial killed vaccines are available for the control of the disease in some countries.

### 3.15 Paratuberculosis

**3.15.1 Definition:** (Synonyms: Johne's disease) Paratuberculosis is incurable chronic contagious granulomatous bacterial enteritis of ruminants caused by *Mycobacterium paratuberculosis*. It is characterized by a long incubation period, chronic diarrhea, progressive emaciation, debilitation, and eventually death.

**3.15.2 Epidemiology:** The causative organism is *Mycobacterium avium* subspecies *paratuberculosis*, formerly known as *M.paratuberculosis* or *M.johnei* (Johne's bacilli). Causative agent is an acid-fast short bacillus. The organism is quite resistant and can survive on pasture for more than one year, but sunlight, alkaline soils, and drying reduce its survival rate. Long incubation period of 2 years or more is an important feature of the natural disease. Four stages of paratuberculosis in cattle have been described:

**Stage 1 (silent infection):** It occurs in calves, heifers and young cattle up to two years of age. These animals may shed the organism without apparent clinical signs without effects on body weight gain or body condition.

**Stage 2 (subclinical disease):** Carrier adult animals are without observable clinical signs, but may be affected by other abnormalities such as mastitis or infertility.

**Stage 3 (clinical disease):**

- a) Clinical signs do not appear before 2 years of age and are most common in the 2-6 year age group

- b) Cases occur sporadically because of the slow rate of spread of the disease.
- c) Gradual loss of body weight despite a normal appetite. Within several weeks, concurrent weight loss and continuous or intermittent diarrhea develops.
- d) Milk production declines. Temperature, heart rate and respirations are within normal limits. The animal eats well throughout but thirst is excessive.
- e) The feces are soft and thin, like pea soap, homogenous and without offensive odor. There is marked absence of blood, epithelial debris and mucus.

#### Stage 4 (Advanced clinical disease):

- a) As the disease worsens, emaciation is the most obvious abnormality and is usually accompanied by intermandibular edema which has tendency to disappear as diarrhea develops.
- b) The diarrhea is characterized by a fluid "waterhose" passage of feces.
- c) The course of the disease varies from weeks to months but always terminates in severe dehydration, emaciation and weakness necessitating destruction.

In sheep and goats, the disease is manifested principally by emaciation, shedding of wool in sheep. Diarrhea is not severe but the feces may be soft enough to lose its usual pelleted form. Affected sheep may lose weight for up to 4 months, be partially anorectic and feces may appear normal until the terminal stages when it changes to soft and pasty consistency. Depression and dyspnea are evident in goats but less obvious in sheep.

Cattle are the main species affected, with goats, sheep, and deer also susceptible. Susceptibility is greatest in young animals less than 3 months old. Clinical disease is usually seen in older animals. The bacteria are shed by some asymptomatic cattle into their milk. The disease is spread to human through consumption of unpasteurized milk. *M. paratuberculosis* has been isolated from some human patients with Crohn's disease. Therefore, animals with paratuberculosis should be considered as potential zoonotic risks. It occurs in many countries, especially those with cool climates, and is most prevalent in intensive dairy areas. The organism is shed in large numbers in faeces of infected animals. Infection is acquired by ingestion of contaminated feed and water. Subclinically infected cattle pose an important source of infection. Cattle can shed bacterial organisms as long as 18 months after developing clinical signs.

#### 3.15.3 Diagnosis

- a) Microscopical examination of Ziehl-Neelsen stained faecal samples.
- b) Isolation and identification of *M. paratuberculosis*.
- c) Polymerase chain reaction PCR.
- d) Serological testing such as complement fixation test, agar gel immunodiffusion test & ELISA.

#### Gross Lesions:

- a) At necropsy, carcasses may be emaciated and edematous. Lesions can be mild, but typically the distal small intestinal wall is diffusely thickened with a non-ulcerated mucosa thrown into prominent transverse folds (corrugated).

- b) Lesions may extend proximally and distally to the jejunum. Serosal lymphangitis and enlargement of mesenteric and other regional lymph nodes is usually apparent.

**Differential Diagnosis:** In cattle: Salmonellosis, Coccidiosis, Parasitism, Chronic molybdenum poisoning, cobalt deficiency, malnutrition. In small ruminants: Caseous lymphadenitis, internal abscess, gastrointestinal parasitism, caprine arthritis - encephalitis, ovine progressive pneumonia and Dietary deficiencies.

**Treatment:** Currently, no antimicrobials are approved for the treatment of Johne's disease. Because of the lack of efficacy and the failure of any of the antimicrobials to provide a cure, treatment is not recommended.

#### 3.15.4 Prevention and Control

- a) Herds with confirmed cases should be tested to determine the extent of infection and positive animals sent to slaughter. Retesting, at 6 months to one year intervals, should be continued until three or more negative tests are obtained.
- b) Infected animals should be culled and calves from dams that have or develop signs of the disease should also be culled due to possibility of intrauterine infection.
- c) Calves should be removed from cows to clean quarters immediately after birth, bottle-fed colostrum that has been pasteurized or obtained from negative cows, and then reared completely segregated from adults until more than one year old. Methods to minimize faecal contamination across the farm can also help.
- d) Isolation of cattle purchased from non-tested herds until they are proven negative by faecal cultures. Negative blood tests on purchased animals are unreliable.
- e) Vaccination reduces, but does not eliminate, bacterial shedding by the infected animals.

### 3.16 Bovine Tuberculosis

**3.16.1 Definition:** Bovine tuberculosis (Bovine TB) is a contagious zoonotic chronic debilitating bacterial disease of cattle caused by *Mycobacterium bovis* and associated with progressive emaciation and tubercle (granuloma) formation involving most usually the respiratory system but also other organs.

**3.16.2 Epidemiology:** *Mycobacterium bovis* is an acid-fast slow growing rod-shaped (bovine tubercle bacillus). It is moderately resistant to heat, desiccation and many disinfectants. The clinical signs usually take months to develop. Infections can also remain dormant for years and reactivate during periods of stress or in older age. Generalized signs include progressive emaciation, lethargy and weakness. A capricious appetite and a low-grade, fluctuating fever are also commonly associated with the disease.

#### Respiratory form:

- a) There is bronchopneumonia that causes a chronic intermittent cough with later

signs of dyspnea and tachypnea. The cough is never loud or paroxysmal, occurring only once or twice at a time and is low, suppressed, and moist.

- b) Chronic, painless swelling of the submaxillary, prescapular and precrural lymph nodes is rare.

#### Intestinal form:

- a) Rarely tuberculous ulcers of the small intestine cause intermittent diarrhea.
- b) Enlargement of retropharyngeal lymph nodes causes dysphagia and noisy breathing due pharyngeal obstruction. Enlargement of mediastinal lymph nodes is associated with recurrent and then persistent ruminal tympany.

#### Genital form:

- a) Lesions sometimes found on female genitalia but rarely on the male genitalia.
- b) In tuberculous meteritis, there may be infertility, recurrent abortion late in pregnancy, or neonatal mortality due to generalized tuberculosis.
- c) In cows that fail to conceive, there may be a chronic uterine purulent discharge. Rare cases of tuberculous orchitis are characterized by the development of large, induced, painless testicles.

#### Mastitis:

Involvement of the udder is rare and usually results in chronic inflammation and induration of the udder (tuberculous mastitis), associated with supramammary lymph nodes enlargement. Tuberculous mastitis is of major importance because of the danger to public health, and of spread of the disease to calves.

In sheep and goats bronchopneumonia is the commonest sign of the disease, manifested by cough and terminal dyspnea. In some goats intestinal ulceration, with diarrhea and enlargement of the lymph nodes of the alimentary tract are witnessed. All species, including man are susceptible to *M.bovis*. Cattle and pigs are most susceptible while sheep, goats and horses are showing a high natural resistance.

Bovine TB is a significant zoonosis and constitutes a public health problem. Humans usually become infected by ingestion of infected raw (unpasteurized) milk or dairy products, handling or consumption of meat from tuberculous animals. Bovine TB is widespread throughout the world. In many countries bovine tuberculosis is still a major infectious disease among cattle and other domesticated animals, and among certain wildlife populations. A few countries including Australia, Denmark, Sweden, Norway, and Finland are considered to be free of bovine tuberculosis. Cattle and buffalo are considered to be the maintenance hosts for *M.bovis*. Organisms are excreted in the exhaled air, in sputum, faeces (from both intestinal lesions and swallowed sputum from pulmonary lesions), milk, urine, vaginal and uterine discharges and discharges from open peripheral lymph nodes. Transmission occurs either by inhalation or ingestion. Bovine tuberculosis is most likely to be introduced with imports of live cattle, embryo and semen.

#### 3.16.3 Diagnosis

- a) Microscopic examination of direct smears from clinical samples.

- b) Culture and isolation.
- c) PCR or Nucleic acid recognition methods.
- d) Serological testing such as complement fixation test, fluorescent antibody test, ELISA.

Gross Lesions:

- a) After death, a preliminary diagnosis of TB can be made by the presence of typical lesions (granulomas or tubercles) in various organs and their associated lymph nodes. Tuberculosis granulomas may be found in any of lymph nodes, but particularly in bronchial, retropharyngeal and mediastinal nodes.
- b) In the lung, miliary abscesses may extend to cause a suppurative bronchopneumonia. The pus has a characteristic cream to orange color and varies in consistency from thick cream to crumbly cheese. In advanced cases, the tubercles are also common in the lung, spleen, liver, female genitalia and the surfaces of body cavities.
- c) In disseminated cases, multiple small granulomas may be found throughout the organs. Tuberculous nodules may appear on the pleura and peritoneum.
- d) Granulomatous TB starts as microscopic lesions, but can develop into large nodules containing thick, yellow, and cheese like pus (caseation), sometimes with a gritty texture (calcification).

Differential Diagnosis: chronic contagious bovine pleuropneumonia, Bacterial pneumonia caused by *pasteurella* or *Corynebacterium pyogenes*, Lung abscesses due to aspiration pneumonia, Pleurisy and pericarditis following traumatic reticulitis, Actinobacillosis, Bovine leucosis and lymphadenopathy.

Treatment: This is not usually undertaken in livestock because of the chronic, contagious nature of the disease, its potential public hazards and the danger of encouraging drug resistance.

#### 3.16.4 Control and Prevention

- a) Removal of the infected animals
  - i. Early diagnosis with the tuberculin test,
  - ii. Segregation or slaughter of infected animals,
  - iii. Tracing and containment of animals that have been in contact with reactors.
  - iv. The test and slaughter policy is the only one assured method of eradicating Bovine TB and relies on the slaughter of reactors to the tuberculin test. It involves repeated tuberculin testing and removal reactors on a herd basis until the whole herd has passed 'clean' at two successive tests. It has been used successfully in the UK, USA, Canada, New Zealand, and Australia. Tests in infected herds should be conducted every 3 months. Herds should be considered free of TB after two negative consecutive tests 6 months apart.
- b) Prevention of spread of infection
  - i. Control of movement of infected cattle is very important.
  - ii. Routine hygiene measures aimed at cleaning and disinfecting contaminated food and water troughs, etc are also useful.
  - iii. It is important that calves being reared as herd replacements be fed on `

- iv. tuberculosis free milk, either from known free cow or pasteurized.
- iv. From the public health perspective, pasteurization of milk is essential to inactivate tubercle bacilli. Meat inspection is essential to remove tuberculous animals from the food chain. Infected carcasses detected at abattoirs should be traced back to identify infected herds. Farm attendants should be checked as they may provide a source of *M.tuberculosis* infection.
- c) Avoidance of further introduction of the disease: Imported animals should come from free countries or certified tuberculosis-free herds, supplemented by tuberculin testing during quarantine.

### 3.17 Caseous Lymphadenitis

3.17.1 Definition: (Synonyms: Pseudotuberculosis, Cheesy gland, Thin-ewe syndrome) Caseous Lymphadenitis is a worldwide chronic contagious, recurring bacterial disease affecting particularly sheep and goats. Clinically, there is abscess formation in the external and less frequently in the internal organs and internal lymph nodes. The disease is of great economic importance.

3.17.2 Epidemiology: The causative agent is *Corynebacterium pseudotuberculosis*, which is found on fomites and in soil (8 months) and manure contaminated with purulent exudates (more than 2 months). Two biotypes have been identified: a nitrate-negative group that infects sheep and goats, and a nitrate-positive group that infects horses. Isolates from cattle are a heterogeneous group bovine biotype. Both biotypes produce an exotoxin, phospholipase D, can damage endothelial cells and promote spread from the initial site of infection to regional lymph nodes and visceral organs. The incubation period ranges between 1-4 months. There are two basic forms

- a) External form:
  - i. Obvious enlargement in the size of one or more of the peripheral lymph nodes, as it appears painful, hard and warm in texture and sometimes accompanied by lameness, especially in camels.
  - ii. In sheep and goats, often appear in the lymph nodes of the head and neck especially retropharyngeal, mandibular, parotid, prescapular, and some times in the supramammary, prefemoral, and popliteal lymph nodes, but in camels often occur in the cervical lymph nodes.
  - iii. In advance cases of sheep and goats, the affected lymph nodes appear soft, painless and of cold texture, due to the presence of pus. It may open spontaneously pouring out thick (caseous) yellow or greenish – white odorless pus. In camel these are often transformed in to a solid mass abscess.
  - iv. With the progression of the infection, the pus become dry and the lymph nodes appear harder than normal and are surrounded by layers of dense fibrous tissue, giving the form of onion rings.
- b) Internal form:
  - i. Partial loss of appetite with general weakness and dullness.
  - ii. Marked decrease in the weight of affected animal.

- iii. Marked decrease in the milk production.
- iv. The internal organs most affected are the lungs, kidney, liver and udder.
- v. Cough and dyspnea, especially when the lungs are affected.
- vi. Sometimes seen as purulent discharge from the eye and nostrils.
- vii. The disease affects mainly sheep and goats, and occurs as sporadic cases in cattle, horses and camels frequently in adult animals (2-4 years).

*C.pesudotuberculosis* rarely causes disease in man. *Pseudotuberculosis* is an important endemic infection in regions with large sheep and goat populations. It occurs worldwide. Infection usually occurs by different ways ; through skin abrasions or wounds caused by shearing, barbwire fencing or exposed nails, through ingestion of contaminated feed and water , through mucus membrane of the mouth specially when giving coarse or roughage feed , infection may result from aerosol infection of the lungs also. Infected animal is considered as the most important source of infection to other animals in the flock.

#### 3.17.3 Diagnosis

- a) Hematological examination and culture from pus.
- b) Serological testing such as indirect hemagglutination, and ELISA
- c) Gross Lesions:
  - i. Caseous abscess filled with greenish-yellow pus occur chiefly in lymph nodes and to lesser extent in internal organs. In the early stages the pus is soft and pasty but in later stages it is firm and dry. In sheep, the abscess often has the classically described laminated "onion-ring" appearance in cross section, with concentric fibrinous layers separated by caseous exudate.
  - ii. Abscesses and caseous lesions may be seen in visceral organs (lung, kidney, liver and spleen). Extensive bronchopneumonia, with pus of a similar color, may also be present.

Differential Diagnosis: Actinobacillosis, tuberculosis and Suppurative lymphadenitis and abscesses can also be caused by various other pyogenic organisms, such as *Actinomyces pyogenes* and, *Staphylococcus aureus*.

#### Treatment:

- a) Antibiotics and surgical intervention have no any practical value due to the ineffectiveness of antibiotics to reach directly to the pathogen which often exist within the immune cells surrounded by a fibrous wall in the affected lymph node. It's preferable to exclude the infected animal from the herd.
- b) In case of external form , surgical intervention includes the following :
  - i. Apply ecthymol ointment on the affected glands for the purpose of maturity.
  - ii. After ripening, open the abscess surgically.
  - iii. Wash the glands from inside with solution of iodine (7 %).
  - iv. Apply a piece of gauze soaked in iodine and change it every 3 days.
  - v. Sometimes, complete surgical removal of the infected gland.

#### 3.17.4 Control and Prevention:

- a) Reducing exposure to the organism:

- i. Control of spread is based on reducing transmission of the organism from infected to susceptible animals. Reducing exposure to possibly contaminated fomites. Young animals should be isolated from older, infected animals.
- ii. Reducing risk of injury and contamination from the environment using dipping fluids, feeders and feed.

- iii. Skin wounds should be treated topically with iodine and sutured if necessary.
- iv. Affected sheep should be remaining in isolation until complete healing of skin.
- v. Older animals and infected ones should be shorn last, and equipment such as shearing blades should be disinfected periodically.
- vi. It is necessary for the shearer to have a high index of awareness of abscesses while shearing. Shearing floor should be thoroughly cleansed disinfected, and ventilated after shearing of the flock is completed.
- vii. Reduction of prevalence is based on the detection and removal of infected sheep (ELISA is positive as early as 30-60 days after infection).
- viii. Prevention of entry of disease into a clean flock is based on serologic screening and isolation of incoming animals.
- b) Culling:
  - i. Eradication is difficult and requires rigorous culling of infected animals with enlarged lymph nodes and emaciated animals with recurrent abscesses.
  - ii. Culling on the basis of the newer ELISA tests is a more sensitive method for detection of early infection and animals with internal abscesses.
  - iii. Eradication is reported in endemically infected flocks by initial culling of all sheep with clinical signs and subsequent serological testing.
- c) Vaccination:
  - i. Commercially available vaccines contain killed bacteria and a variable residue of phospholipase D toxoid is effective. Initially given to lambs and kids at 3 and 4 months of age, with annual booster dose to all breeding stock one month before expected parturition.
  - ii. Vaccination must be repeated annually after 2 initial vaccinations and boosters should be given at least 2 weeks before shearing.

## 3.18 Foot Rot

- 3.18.1 Definition:(Synonyms: Necrotic pododermatitis, Interdigital necrobacillosis, Foul foot, Hoof-rot, Interdigital phlegmon and, Infectious pododermatitis) Foot rot is a highly contagious disease affecting the interdigital tissue of ruminants, foot rot can be very difficult to control and results in serious economic loss, but it is a manageable and preventable disease. The disease is neither zoonotic nor notifiable.

- 3.18.2 Epidemiology:Foot rot is caused by a combination of two anaerobic bacteria, *Fusobacterium*

*necrophorum* and *Dichelobacter nodosus* (more common in sheep), and *Bacteroides melaninogenicus* (more common in cattle). Mechanical injury or softening and thinning of the interdigital skin by puncture wounds or continuous exposure to wet conditions can provide entrance points for these infectious agents.

The disease affects all ruminants, it is a common cause of lameness in cattle and sheep, and found worldwide, especially during wet, humid conditions. The disease is transmitted, when feet infected with *F. necrophorum*, organism travels from the infected animal to the soil and then to non infected animal, human can spread the disease on their boots. Once an opening is created in the tissues of feet, bacteria gain entrance into subcutaneous tissues and begin rapid multiplication and production of toxins and leads to the penetration of infection into the deeper structures of the foot.

- 3.18.3 Diagnosis: following exposure incubation period takes five to seven days and the clinical signs are lameness, interdigital swelling and separation of the interdigital skin may occur, with distinctive lesions and odor of the feet.

Bacterial culture is of diagnostic importance. Differential diagnosis includes: FMD, Pox, foreign bodies in the interdigital space, interdigital dermatitis (hairy heel warts), interdigital hyperplasia sole bruises and abscesses or fractures of the bones of the foot

- 3.18.4 Control and Prevention: Treat infected animals by topical antibiotic and systemic antimicrobial therapy; feet should be trimmed regularly and soaked in foot baths of zinc sulfate or copper sulfate. Management practices that help to reduce hoof damage will help to reduce the incidence of foot rot. Prevention also requires application of biosecurity measures.

### 3.19 Leptospirosis

- 3.19.1 Definition: (Synonyms: Redwater of calves). Leptospirosis is a contagious zoonotic bacterial disease of many animal species, most frequently reported in cattle, caused by various immunological distinct serovars, for *Leptospira interrogans*. The disease is primarily caused by serovars *L. Pomona*, *L. hardjo*, *L. grippotyphosa*, *L. canicola* and *icterohemorrhagica*. The organism can survive for up to six months in moist, warm conditions. Stagnant water is the ideal source of infection. On the other hand, high temperature (greater than 30C), freezing, drying, or an acid or alkaline environment rapidly kills the bacteria. The incubation period is generally 3 to 7 days in all animals. Infections may be asymptomatic or cause various signs, including fever (40.5 - 41C), anorexia, dyspnea, icterus, acute hemolytic anemia, hemoglobinuria, renal failure, infertility, stillbirth, mastitis and death. Man is susceptible to all the pathogenic serovars in domestic animals, and transmission from wildlife generally occurs when contact is made with tissues of infected animals or surface waters contaminated by urine from infected animals. The most common signs are fever, headaches, rash, myalgia, and malaise.

- 3.19.2 Epidemiology: Leptospirosis is recognized worldwide as a cause of abortion, systemic

illness, and mastitis. Leptospirosis is seen most frequently in areas of the world that have a temperate climate and a high rainfall. It is mostly reported in cattle. Rodents, sheep, horses, goats, dogs, and man are also susceptible. All age are susceptible, but calves and lambs are highly susceptible to infection than adult.

Leptospirosis usually infects the kidney; thus urine from diseased and carrier animals can contaminate pens, corrals, pasture, drinking water, feed, etc. Alternatively, aborted fetuses or uterine discharges can contaminate the environment. Infections can be readily established via the conjunctiva, vaginal mucosa, or skin abrasions. Because the organisms survive in surface waters for extended periods, the disease is often waterborne. Ingestion of contaminated food or water is also common mode of transmission of the disease. Infected bulls can also transmit the infection through semen to females in the herd. Leptospirosis may be transmitted to cattle by many infected species-rats and other rodents, raccoons, foxes and dogs. The disease characterized by high morbidities. Mortalities in adult cattle may reach 5%, while in young calves is 5 - 15%.

- 3.19.3 Diagnosis: Demonstration of leptospires in urine, blood or tissues is helpful in diagnosis. Older techniques such as dark field microscopy of urine and Warthin- Starry silver staining of tissues are not sensitive or specific. Newer tests include fluorescent antibody techniques, which are specific, and polymerase chain reactions to amplify DNA from small numbers of leptospires. Definitive confirmation of leptospirosis is made by isolation of the organism from urine or tissue of infected animals. Detection and identification of the agent are done by Immunofluorescence, immunochemistry and PCR. Serological tests such as the microscopic agglutination test and ELISA. Differential diagnosis include: Diseases that cause abortion such as brucellosis, trichomoniasis, Campylobacteriosis, listeriosis, Salmonellosis, IBR, BVD, Q-Fever, Chlamydial abortions and mycotic abortions. Regarding hemoglobinuria and/or anemia such as bacillary hemoglobinuria, babesiosis, anaplasmosis, post parturient hemoglobinuria, chronic copper poisoning and red kale poisoning.

- 3.19.4 Prevention and Control: Treatment by Dihydrostreptomycin, chlortetracycline and oxytetracycline was reported to be successful if given early; Dihydrostreptomycin (12mg/kg BW IM twice daily for 3 days) is effective in the treatment of the systemic infection. For elimination of leptospiruria in cattle, a single dose of Dihydrostreptomycin (25 mg/kg BW IM) is recommended.

Good management and vaccination are effective in the control of leptospirosis, and involves:

- Isolation and treatment of affected animals as early as possible.
- Detection and elimination of carrier animals.
- Eliminating access of cattle to surface water or streams used by other livestock.
- Removing trash that harbours wildlife to livestock feed.
- Limiting access of rodents and wildlife to livestock feed.
- Elimination urine drainage into water sources.
- Cleaning, disinfecting, and drying barns, pens, and other confinement areas after use by infected cattle.
- Inactivated vaccines for veterinary use are suspensions of one or more

- i) Bacterins generally confer protection against abortions and death and significantly reduce renal infections.
- j) Effective inactivated vaccines containing the five most important strains of leptospira that are available and should be administered to calves at 4 to 6 months of age, with annual revaccination.
- k) The best time to vaccinate cows is 30 days before breeding.

### 3.20 Contagious Caprine Pleuro-Pneumonia

3.20.1 Definition: Contagious caprine pleuropneumonia (CCPP) is a highly contagious, fatal, respiratory disease, affecting primarily goats of all ages. This disease causes heavy economic losses. The classical CCPP is caused by *Mycoplasma capricolum* subspecies *capripneumoniae*, which originally known as Mycoplasma F- 38 biotype. The other mycoplasmas associated with CCPP are *Mycoplasma mycoides Capri* and *Mycoplasma mycoides mycoides* (large colony type). The mycoplasmas are polymorphic, 0.2- 0.5 mm in size, Gram negative, organism. They are resistant to penicillin due to lack of cell wall, sensitive to heat and dryness and die within few minutes at 60 °C but remain alive in the frozen tissues.

3.20.2 Epidemiology: The disease is characterized by severe respiratory distress, coughing, nasal discharge, and severe fibrinous pleuropneumonia. The incubation period of the disease varies from 6-10 days but in some herds it may extend up to 4 weeks. The disease occurs in goats in three forms, peracute, acute and chronic form. The typical CCPP having high mortality rate is usually associated with *Mycoplasma capricolum* subspecies *capripneumoniae*, while other mycoplasma species are comparatively less virulent.

a) Peracute and Acute Forms:

- i. Fever (40 - 41 C), dullness, loss of appetite, and isolation of the infected animal from the rest of the flock.
  - ii. Difficult respiration with frequent painful cough. Loud frictional rub sounds and respiration rales heard on auscultation. These respiratory rales may disappear gradually due to fluid filling the chest cavity.
  - iii. Copious nasal discharge, which initially is seromucous and later changes to thick purulent type.
  - iv. Occasional abortion in pregnant animals.
  - v. More deaths in per acute form and usually associated with septicemia and respiratory failure.
- b) Chronic Form: This form is characterized by progressive loss of weight, poor bodily condition, chronic painful cough and purulent nasal discharge.

CCPP is highly contagious disease of goats; sheep can be infected experimentally, while cattle are resistant to these mycoplasmas. Goats of all ages and of both sexes are susceptible to this disease but a high morbidity and mortality is observed in young animals at 4 - 6 months of age. The morbidity rate with virulent type of CCPP can reach up to 100% and the mortality rate more than 60%. The disease is widespread in most countries of Africa, Middle East, South Asia, and Eastern Europe.

The stress associated with malnutrition, transportation, high humidity and exposure to some viral diseases like PPR and pox are considered the most important predisposing factors. The highest incidence of the disease often occurs during the extreme cold and warm months. The disease spreads through inhalation of infected nasal secretions. Sick animals affected by the chronic form are important source of infection within the herd.

3.20.3 Diagnosis: There are steps to reach the right diagnosis.

- a) Laboratory examination, includes, Isolation of the mycoplasma, serological tests such as latex agglutination test, immunofluorescence, CFT and ELISA, Direct examination of lung and pleural samples using PCR. Gross lesions: The consolidation and granular appearance of lungs present in all stages of pneumonia. Pulmonary congestion, fibrinous adhesions covering entire lungs, and thick pleura. Accumulation of transparent straw fluids in the thoracic cavity. Enlargement of the surrounding lymph nodes. Occasionally pericarditis with accumulation of fluid in the pericardium, meningitis, and arthritis lesions.
- b) Differential Diagnosis: PPR, Pasteurellosis and Contagious agalactia syndrome.

3.20.4 Prevention and Control: The success of treatment depends on the early diagnosis of the disease. The infected goats respond well to treatment with antibiotics and anti-inflammatory drugs.

- a) Intramuscular injection of Tylosin at a dose rate of 10-12 mg /kg BW every 12 hourly for four subsequent days., or I/V injection of oxytetracycline (10%) in a dose of 5-10 mg /kg BW daily for five days., or A combination of injection of Tylosin and oxytetracycline gives a good response, or Injection of lincomycin, erythromycin, or spiramycine in a dose of 5-10 mg /kg BW daily for 5 days.
- b) Antipyretic, anti - inflammatory drugs; I.V. injection of flunixin meglumine (1.1- 2.2 mg / kg BW) for at least two days injection is also recommended in per acute of acute cased of CCPP.



Fig. 15 CCPP Purulent Nasal Discharge



Fig. 16 CCPP Lung & Pleural adhesion

The steps of the control of CCPP vary according to the countries where the disease is present. In endemic countries, the following protocol for control of the disease is recommended:

- a) Good hygienic measures such as immediate isolation of the infected animals, disinfection of animal houses, and all equipments of the farm house by strong disinfectant.
- b) Rapid treatment of infected animals.
- c) Restricted movement of the animals.
- d) Prevent introduction of new goats to the farm, unless they are clinically free from the disease.
- e) Provision of adequate water and balanced feed with vitamins and mineral supplements to enhance the immunity of the animals and to prevent malnutrition.
- f) The ideal method for the prevention of the disease is by vaccination.

### 3.21 Q Fever

- 3.21.1 Definition: (Synonyms: Goat Flu). Q fever is a highly zoonotic disease with a worldwide occurrence, Caused by the intracellular bacteria called *Coxiella burnetii*. Many domesticated and wild animals including mammals, birds, reptiles and arthropods can carry *C. burnetii*. In most cases, the infection is asymptomatic, but abortions or stillbirths can occur in ruminants. The incubation period is usually 2 to 3 weeks. The disease is characterized clinically by abortion in late pregnancy, stillbirths, retained placenta, endometritis, infertility and small or weak offspring in ruminants. Animals may appear asymptomatic. Goats will have decreased appetite and may be depressed one or two days before an abortion. Q-fever in humans results in "flu-like" symptoms which can persist. Symptoms include fever, headache, chills and sweats, loss of appetite, dry cough, chest pains and sore muscles.
- 3.21.2 Epidemiology: Primary reservoirs of *C. burnetii* infection are goats, sheep, and cattle. Infection has been noted in a wide variety of other animals, including other species of livestock and in domesticated pets. The infection results from inhalation of contaminated particles in the air, and from contact with the milk, urine, feces, vaginal mucus, or semen of infected animals. Rarely, the disease is tick borne. The organisms are resistant to the heat, drying, and common disinfectants. These features enable the bacteria to survive for long periods in the environment. Ticks may be important in transmission among wildlife, and can also spread infections to domesticated ruminants. Infection of humans usually occurs by inhalation of these organisms from air that contains airborne barnyard dust contaminated by dried placental material, birth fluids, and excreta of infected herd animals. Humans are often very susceptible to the disease, and very few organisms may be required to cause infection. Ingestion of contaminated milk, followed by regurgitation and inspiration of the contaminated food, is a less common mode of transmission. Other modes of transmission to humans include tick bites. Human to human transmission is rare.
- 3.21.3 Diagnosis: Routine diagnosis of Q fever in aborted ruminants is generally performed by the detection of bacteria in smears or impressions of placentas stained by the Stamp, Gimenez

or Machiavello methods, direct isolation of the organism from tissues such as placenta and combined with the serological analysis of at least ten sera samples by the CFT, or better by ELISA or by detection of DNA specific for *C. burnetii* using one of several PCR protocols, or by immunohistochemical staining for the pathogenic antigens. Differential diagnosis includes diseases like: Campylobacteriosis, Leptospirosis, Listeriosis, Chlamydiosis, Trichomoniasis, IBR, RVF, BT, PPR, Border disease, Tick borne fever and Toxoplasmosis.

- 3.21.4 Prevention and Control: For treatment of ruminants, oral tetracycline at therapeutic dose may be given for 2 - 4 wk. In a *C. burnetii*-free flock, introduction of new stock should be minimized, and contact with wildlife should be prevented as much as possible. Good tick control should also be practiced. In an infected flock, isolating infected pregnant animals and burning or burying the reproductive membranes and placenta can decrease transmission. Antibiotics may be given prophylactically before animals give birth. Isolate aborted animals until discharges cease; restrict access by animals and people where possible. Regularly clean and disinfect lambing sheds, calving pens and similar buildings to prevent accumulation of potentially contaminated material. Control ticks and other parasites on livestock. Animals should be routinely tested for antibodies.

## Chapter (4) Fungal and Parasitic Diseases

### Fungal Diseases – Mycoses - Dermatomycoses

- 4.1 Ringworm
- 4.2 Epizootic Lymphangitis
- 4.3 Sporotrichosis Parasitic Diseases Arthropodes
- 4.4 Myiasis
- 4.5 Mange

### Helminths

- 4.6 Echinococcosis
- 4.7 Fascioliasis

### Protozoa

- 4.8 Trypanosomiasis
- 4.9 Anaplasmosis
- 4.10 Babesiosis
- 4.11 Theileriasis
- 4.12 Toxoplasmosis

### 4.1 Ringworm

- 4.1.1 Definition: (Synonyms: Dermatophytosis or Tinea) Ringworm is a fungal skin disease that can affect many animal species and human, it is known as dermatophytosis
- 4.1.2 Epidemiology: Dermatophytosis is caused by different kinds of fungi in the genera of *Trichophyton* and *Microsporum*. Many animal species can get ringworm including cows, sheep, goats, pigs, horse, dogs, cats, rabbits, rodents, birds and humans. People can get

the disease from an infected animal especially kittens and puppies or from other people. Dermatophytes grow best in warm and humid environments and more common in tropical and subtropical regions, *T. canis* and *T. equinum* occur worldwide.

The disease spread by direct contact with asymptomatic or symptomatic animals. Infective spores in hair and dermal scales can remain viable for several months to years in the environment. Fomites have an important role in indirect contact infection. Humidity is known to be more important, a high humidity being conducive to multiplication of the fungus. Direct contact with infected animals is a common method of spread of ring worm and licking with the tongue undoubtedly aid spread of fungus. Premises and harness may remain infective for long periods because fungal spores remain viable for years provided they are kept dry and cool. Moderate heat and desiccation destroy them.



Fig. 17 Ringworm Lesion Details



Fig. 18 Ringworm Lesions on Camel Skin

- 4.1.3 Diagnosis: Incubation period is 2-4 weeks. Main clinical signs are the appearance of round areas of hair loss and inflamed skin, crusts or scales may develop on the skin in the affected areas, and the skin in these areas is red and itchy.

Sometimes the causative fungi die in the centre of the affected area leaving an appearance of circle or ring. Thick crusty lesions and pustules or thickened red and weepy skin and affected areas may be up to 2 inches in diameter. Scarring may occur. Hair regrowth may be black. Wood's lamp can be used to examine the fur/hair of the animal for fungi; diagnosis can also be made by direct examination and fungal culture. The disease should be differentiated from *Staphylococcus* infection and eczema.

- 4.1.4 Prevention and Control: Failure to control an outbreak of ringworm is usually due to the wide spread contamination of the environment before the treatment is attempted.

Isolation and treatment of the infected animals, the provision of separate blankets, feeding utensils and grooming tools, and disinfection of the items after use on affected animals are necessary if the disease is to be controlled. For treatment, many topical applications are apparently successful.

Since spontaneous recovery is common the main virtue of topical therapy is to prevent the progression of existing lesion and limit spread of infective material to other animals. Treat infected animals by 7% Iodine. Reduce the spreading of the disease by applying of biosecurity measures, clean and disinfect areas where animals live.

## 4.2 Epizootic Lymphangitis

- 4.2.1 Definition: (Synonyms: Pseudoglanders, Equine Blastomycosis, Equine histoplasmosis) Epizootic lymphangitis is a chronic granulomatous disease of the skin, lymph vessels, and lymph nodes of the limbs and neck of equids caused by the dimorphic fungus *Histoplasma fraciminosum* (*H. capsulatum* var. *fraciminosum*)

- 4.2.2 Epidemiology: Epizootic lymphangitis is OIE notifiable listed diseases, mainly affects horses, donkeys and mules. Infections have also been reported in camels and cattle, rare cases of human infection have been reported.

The disease is more common in tropical and subtropical region and is endemic in some countries in the Mediterranean region. *H.farciminosum* exists as yeast in tissues and mycelium in environment. The organism infect animals through open wounds (the skin form.), (the pulmonary form), which is rare, probably develops when an animal inhales the organism. Biting flies in the Genera *Musca* & *Stomoxys* are thought to spread the (conjunctival form). Epizootic lymphangitis is a debilitating fungal disease seen mainly in equids. The most common form of this disease is an ulcerative, suppurative, spreading dermatitis and lymphangitis; however, other forms including pneumonia or ulcerative conjunctivitis also occur. *H. capsulatum* var. *farciminosum* has also been reported in camels, cattle and dogs. The source of the organisms can be the skin lesions and nasal and ocular exudates of infected animals, or the soil. In its saprophytic mycelial phase, *H. capsulatum* var. *farciminosum* can survive for many months in warm, moist environments. This organism can also be spread on fomites such as grooming or harness equipment. Epizootic lymphangitis must be reported to state or federal authorities immediately upon diagnosis or suspicion of the disease.

- 4.2.3 Diagnosis: The incubation period is usually several weeks. The most common form of the disease often occurs on the extremities, chest wall face and neck but can be seen wherever the organism is inoculated into a wound. Clinically, this disease is characterized by freely movable cutaneous nodules, which originate from infected superficial lymph vessels and nodes and tend to ulcerate and undergo alternating periods of discharge and closure. Affected lymph nodes are enlarged and hard.

On post-mortem examination, areas of skin and subcutaneous tissues are thickened, fibrous, and firm. Purulent foci may be noted on the cut surfaces. The lymphatic vessels are usually distended and contain purulent material. The regional lymph nodes are soft, swollen, and reddened and can contain purulent foci. The lungs, spleen, liver, testes and other organs may also contain nodules and abscesses. The clinical features are highly suggestive. Diagnosis can be confirmed by microscopic examination of exudates and biopsy specimens.

The yeast forms of the organisms distend the cytoplasm of macrophages and appear in H&E stained sections as globose or oval bodies (3 – 4 um) with a central basophilic body surrounded by an unstained zone. Before collecting or sending any samples from suspected animals, the official veterinary authorities should be contacted and samples should be sent under secure conditions to the accredited official laboratory. Differential Diagnosis Epizootic lymphangitis can resemble glanders, strangles, ulcerative lymphangitis, sporotrichosis, and histoplasmosis.

If you suspect Epizootic lymphangitis, call your local and/or Federal veterinarians' office immediately.

- 4.2.4 Prevention and Control: There is no satisfactory treatment. Surgical excision combined with antifungal drugs (Amphotericin B) could be used. Quarantine and restriction of movement, strict hygienic precautions and biosecurity measures are necessary to prevent the spread of the disease. Control of the disease is usually through elimination of the infection. This is achieved by culling infected horses and application of strict hygiene practices to prevent spread of the organism. Vaccination has been utilised on a limited scale in areas where enzootic lymphangitis is endemic e.g., Iraq, but it is not authorised for widespread use.

### **4.3 Sporotrichosis**

- 4.3.1 Definition: (Synonyms: Rose Handler's Disease) Sporotrichosis is a sporadic chronic granulomatous disease of humans and various domestic and laboratory animals caused by *Sporothrix schenckii*. Sporotrichosis is caused by fungus *Sporothrix schenckii*. The fungus grows in the environment and can survive for months or years in soil, vegetation, wood and other objects. It forms single walled spores.
- 4.3.2 Epidemiology: Sporotrichosis occurs most often in horses. Cases have also been seen in cats, dogs, cattle, goats, swine, mules, camels, non-human primates, birds, and various wild animals. Zoonotic infections can occur, cat may be the species with the greatest zoonotic potential, and transmission from cat to human has been reported, people most commonly get sporotrichosis infection from the environment through wounds or other minor injuries that result in broken skin (e.g. Rose thorns) allow an entry site for the fungal spores. Sporotrichosis occurs worldwide, particularly in areas with high humidity and temperatures.

The disease is acquired from the environment when the fungal spores enter a break or abrasion in the skin (direct contact). Spores may be in dead vegetation, wood splinters, thorns or hay, bites, stings, pecks and scratches can "inject" the organism. Infected cats can carry the fungus on their nails and in their mouth and nasal cavity and may transmit the organism by bites and scratches. Sporotrichosis may be grouped into 3 forms: lymphocutaneous, cutaneous or disseminated.

- 4.3.3 Diagnosis: The disease usually develops 1 week to 3 months after exposure to fungal spores, in horses, the lower limbs, particularly the fetlocks are most often affected. One or more firm round bumps or nodules occur at the site of infection, additional bumps may follow and spread up the leg, these may ulcerate and drain, followed by crusting and scabbing. The nodules may become hard and contribute to generalize swelling of the affected leg. Occasionally the disease can spread throughout the body and cause more severe disease and death.

Disseminated disease caused by inhalation of spores is rare. On necropsy all the coetaneous lesions and changes are noticed. Granulomatous nodules are seen in the skin, and may occur in chains along lymphatic vessels, exudation, ulceration, cavitations, crusting, scabbing or scarring. Lymphatics are thickened and cordlike. Lab. diagnosis can be made by cultural (samples obtained from unopened lesions) or microscopic examination of the exudates or biopsy specimens. In tissue and exudates, the organism is presented as few to numerous, cigar-shaped, single cells within macrophages the fungal cells are polymorphic and small buds may be present and give the appearance of a ping-pong paddle. A fluorescent antibody technique has been used to identify the yeast-like cells in tissues.

- 4.3.4 Prevention and Control: Administration of ketoconazole, itraconazole, and amphotericin B has been used to treat sporotrichosis in animals. Potassium or sodium iodide can be used in the cutaneous or lymphocutaneous forms. In horses, single nodules are sometimes removed surgically. Infected animals particularly cats, should be isolated to prevent organism spreading and follow biosecurity measures.

### **4.4 Myiasis**

- 4.4.1 Definition: Myiasis is the screwworm invasion of the living tissues of any warm blooded animals by larvae of flies in the family Calliphoridae. New world screwworm myiasis is caused by the larvae of *Cochliomyia hominivorax* (coquerel). Old world screwworm myiasis is caused by the larvae of *Chrysomya bezziana* (villeneuve).
- 4.4.2 Epidemiology: All warm blooded animals can be infested by screwworms however these parasites are common in mammals and rare in birds. Humans can get screwworm infestation. It is a notifiable disease (Ministerial decision no 193 /2010 Ministry of Environment & Water) and it is a zoonotic. *C. bezziana*, the old world screwworm, can be found in Southeast Asia, the Indian subcontinent, much of topical and sub-Saharan Africa and some countries in the Middle East. The situation is changeable in the Middle East, while Kuwait reported its

last case in 1998; infestations have recently occurred in Iraq and Iran. Occasional cases or outbreaks of screwworm are reported in screwworm – free countries. Screwworms are transmitted when a female fly lays eggs on a superficial wound of an animal.

Screwworms can infest a wide variety of wounds, e.g. tick bites, dehorning. Navel of newborn mammals are a common site for screwworm infestation. Wounds infested by screwworms often attract other female screwworms and multiple infestations are common. Screwworm eggs laid in the wound hatch into larval screwworm, which feed on the living tissue of the animal.

- 4.4.3 Diagnosis: Screwworm larvae emerge from the eggs in 12 - 24hrs, but they are difficult to detect in wounds for the first day or two. Later larvae are visible in wounds and they feed causing the wound to gradually enlarge and deepen. A bloody tinged fluid with a distinct odor may seep from the wound. Infested animals usually separate from the herd, show discomfort with decreased appetite and lowered milk production are common. Untreated animals may die in 7 – 14 days from toxicity or secondary infection. Smaller animals are more affected. Humans can get screwworms in the same manner as animals, by flies depositing larvae on open wound.

Screwworm myiasis should be suspected when clinical signs are observed. Differential diagnosis includes any flies that are capable of infesting wounds. Several types of larvae may be present due to likelihood of multiple infestations; Larvae should be collected from the deepest part of the wound. Before collecting or sending any samples, the proper authorities should be contacted. Samples should only be sent under secure conditions to authorized laboratories to prevent the spread of the disease.

- 4.4.4 Prevention and Control: Removal of larvae, debridement, if necessary, and good hygiene. Recommended actions include notification of authorities, treat infested wounds with larvicides, and suspend animal movement.

## 4.5 Mange

- 4.5.1 Definition: Synonyms: (Scabies, Itch) Mange or scabies are a contagious skin diseases caused by tiny microscopic arthropod parasites called mites affecting animals and humans skin. Scabies the term most commonly referring to human cases of mange is caused by a particular family of mites. Zoonotic species include: *Family Sarcoptidae*, *Family Cheylellidae*, *Family Dermanyssidae* and *Family Trombiculidae*. Mites can be host specific or affect a variety of species.

- 4.5.2 Epidemiology: Different types of mites infest different species of animals. Scabies and mange can occur in more than 100 species of animals including farm animals, wild animals, zoo animals and birds, some species of mange are zoonotic, the disease is found worldwide in different species of animals with some species of mange seen in tropical areas. The mange, mites or scabies are highly contagious and spread by direct or indirect contact. Some mites can survive for several days off an animal's body, on bedding, harnesses and

horse blankets, so these objects can be a source of infestation. People can get infested by some mite's species from infested animals by direct contact.

- 4.5.3 Diagnosis: Incubation is 2 – 6 weeks, severe itching and hair loss. Disease usually starts on head and areas of body with delicate skin such as ears, nose and elbows, vigorous scratching with oozing fluid, skin may become thickened, secondary infection of the skin may occur. Mites from animals cause an allergic reaction to human skin. Mites are parasites of skin and the lesions seen at necropsy resemble those in live animals, secondary bacterial infections or signs of wasting may be seen. For laboratory diagnosis, skin scraping is examined by microscope 40X magnifications for demonstration of mites, eggs or feces, lice and other ectoparasites.

- 4.5.4 Prevention Control: Treatment of animals with ivermectin, preventing admission of infested animal, treatment of animal's environment and humans to use protective clothing are important steps for prevention and control. Ivermectin and moxidectin (200 µg/kg) given twice with a 7 or 10 days interval, respectively, are effective. Doramectin (300 µg/kg) given once is also effective. Dipping is most effective if done within 2 wk after shearing and must be repeated after 14 days. Approved treatments for mange in sheep are 0.3% coumaphos, 0.15-0.25% phosmet, 0.03-0.1% diazinon, and 2% hot lime-sulfur. Other sprays or dips such as propetamphos, phoxim, amitraz, or flumethrin are available.



Fig. 19 Mange in camel



Fig. 20 Mange in sheep

## 4.6 Echinococcosis

- 4.6.1 Definition: (Synonyms: Hydatosis or Hydatid Disease) Echinococcosis is a parasitic disease that can affect animals and humans. It is caused by a tiny tape worm cestode of Genus *Echinococcus*.
- 4.6.2 Epidemiology: Five species of *Echinococcus* have been identified which infest a wide range of domestic and wild animals. *Echinococcus* tape worms have a complex life cycle which involves both a definitive and an intermediate host. Carnivores, such as, dogs, and cats, are definitive hosts. Intermediate hosts include a large number of domestic and wild animals such as sheep, cattle, horses, and reindeer. Cystic Echinococcosis, the most common form of the disease in people and domesticated animals, is caused by *Echinococcus granulosus* and *Echinococcus multilocularis*. The definitive hosts which include dogs, other canids, hyenas and cats, carry the adult tape worms subclinically. Echinococcosis is a zoonotic disease with a major public health problem in some countries. The parasites can be found worldwide. Intestinal infection of definitive host occurs when an animal ingests (oral) tapeworm cysts in the tissues of an intermediate host. Intermediate hosts become infected after ingesting *Echinococcus* eggs passed in the feces of the definitive hosts. Humans can be infected by accidentally ingesting the eggs of the tapeworms. This can occur by eating food (e.g. unwashed fruits or vegetables) contaminated by the feces of infected carnivores or by handling dogs or cats infected with the tapeworms.
- 4.6.3 Diagnosis: The incubation periods are variable as symptoms are caused by the slow growing cyst. Infected carnivores as dogs and cats usually show no signs of disease. Intermediate

hosts can show a range of symptoms depending on the severity of infection. In humans, signs of disease can take months to years to appear.

Infection involves the development of cysts (tumor-like lesions) caused by the growth of the immature larval tape worms. Cysts can vary in size and location in the body; they most commonly involve the liver but can spread to other organs. Signs of disease can include abdominal pain or discomfort, weakness, and weight loss. Laboratory examination is done for demonstration of adult cestodes in feces of infected carnivores and also for detection of *Echinococcus* eggs in fecal sample, detection of copro antigen by ELISA, detect the progression of cyst by imaging techniques (X ray, Ultrasound, CT). Post mortem diagnosis (coprodiagnosis) is to differentiate between cyst and tumor growth.

- 4.6.4 Prevention and Control: Do not feed or allow dogs or cats to eat infected tissues of intermediate hosts. Regular examination and treatment of dogs that are routinely exposed to livestock can decrease infestation in domestic animals, deworming of dogs and livestock. Avoid contact with wild canines, such as foxes, coyotes. Wash all fruits or vegetables thoroughly before eating them. Always wash your hands with soap after handling the animals.



Fig. 21 Hydatid Cysts from liver

## 4.7 Fascioliasis

- 4.7.1 Definition: (Synonyms: Liver fluke disease, liver distomatosis, liver rot or common liver fluke) Fascioliasis is a disease of domestic herbivorous animals caused by a trematode parasite it is caused by *Fasciola hepatica* (sheep liver flukes) or *Fasciola gigantica* the parasites of herbivores that can affect humans accidentally.
- 4.7.2 Epidemiology: Susceptible animal reservoir hosts for fasciola species include the main domestic animals which are cattle, sheep, buffaloes, and donkeys, other domestic animals are horses, goats, camels and llamas, rabbits and rodents. *Fasciola gigantica*, the parasite of herbivores, can infect humans accidentally. Fascioliasis occurs worldwide where sheep and cattle are grazed. The mode of transmission is fecal - oral route. Parasite eggs are passed

in the feces of infected animals and contaminate water where they develop within snails. The snails release mature larvae that attach to vegetation and encysted as metacercariae. Animals and humans become infected by ingesting the encysted metacercaria with herbage.

- 4.7.3 Diagnosis: The incubation period varies depending on the fluke burden from few days to 3 months the prepatent period is usually 2-3 month. Fascioliasis ranges in severity from a devastating disease in sheep to an asymptomatic infection in cattle. The course usually is determined by the number of metacercariae ingested. Sheep with the acute disease have distended, painful abdomen, anemia and sudden death may occur within 6 weeks of infection. In chronic fascioliasis signs include anemia, unthriftiness, submandibular edema, and reduced milk secretion. On post mortem examination liver damage is diagnostic. Adult flukes are readily seen in the ducts and immature stages may be squeezed from the cut surface. Repeated fecal examinations detect the fluke eggs. ELISA helps diagnosis before the prepatent period. The acute syndrome must be differentiated from black disease. Consider during diagnosis the following parasites (*Paramphistomum spp.*), (Rumen fluke), Shistosomiasis, Ascariasis, Toxascariasis and Echinococcosis.
- 4.7.4 Prevention and Control: Prevention of livestock access to potential area of infection, use of anthelmintics , in human avoid eating unthoroughly washed raw plants, and general public health precautions.

## 4.8 Trypanosomiasis

- 4.8.1 Definition: (Synonyms Surra Disease) Trypanosomiasis is an acute or chronic disease caused by a blood parasite flagellated protozoa transmitted by fly vectors. *Trypanosoma evansi* causes the most serious protozoal disease of camels.
- 4.8.2 Epidemiology: Trypanosomiasis can affect all domesticated animals and many other species. Most Trypanosomiasis is transmitted by Tsetse flies. *Trypanosoma brucei gambiense* and *Trypanosoma brucei rhodesiense* cause human African trypanosomiasis (sleeping sickness) which affect both humans and animals. Trypanosoma can be found wherever the tsetse fly exists. Tsetse flies are endemic in Africa. *Trypanosoma vivax* is also found in areas free of tsetse flies. Surra is caused by infection with the *Trypanosoma evansi*. Camels are most often affected in the Middle East and Africa. The disease is transmitted when infected vector fly bites an animal, by fomites and surgical instruments. Carnivores may become infected after feeding on infected meat. Transmission during coitus has also been documented.



Fig.22 Blood smear shows Trypanosoma infection

- 4.8.3 Diagnosis: Incubation period in camels varies from 5 - 60 days, with morbidity rates of 20-70 % in northeastern Africa. Symptoms includes fever, progressive anemia, weight loss and icterus, progressive weakness and lethargy, oedematous swellings of the lower parts of the body, petechial haemorrhages of the serous membranes (eyelids and nostrils), abortion of female camels, and death may occur in two weeks to four months. Examination of blood smears of 10 affected camels showed that only 4 camels had parasites in their blood smears. The parasite is only seen in blood smear when the fever is present. Hematological indices including packed cell volume and hemoglobin indicated that affected camels had anemia. Increasing of lymphocytes caused leukocytosis. Lymphocytosis can be occurring following chronic infection in camel (Al-Ani, 2004). In this report presence of chronic infection was confirmed by monocytosis. Animals infected with any pathogenic trypanosome may develop concurrent and even fatal bacterial, viral and other protozoan infections as a result of immunosuppression. Specific parasit diseases, fungal infection and allergic reactions can lead to Eosinophilia Post-mortem lesions include emaciation of the carcass, anemia, enlarged lymph nodes and spleen with petechial haemorrhages on some internal organs. Trypanosomiasis should be a consideration in endemic areas when an animal is anaemic and in poor condition. Animals imported from endemic areas can be subclinical carriers and may become ill when they are stressed. Other diseases that cause anemia and weight loss including babesiosis, anaplasmosis, hemonchosis, theileriosis, should be ruled out. Laboratory examination includes: wet unstained blood films to be examined by light microscopy. Parasites may be found in stained blood smears (Precautions are recommended when handling blood tissues and infected animals). *T evansi* antigen can be detected with latex agglutination test or ELISAs, PCR and hemagglutination inhibition.
- 4.8.4 Control and Prevention: Control measures aim at the host rather than the vector, detect and treat infected animals with antiparasitic drugs. For camels melarsamine hydro chloride (Cymelarsan) is very effective against *T evansi*, prophylactic treatment of susceptible animals. Controlling anthropod vectors is important in preventing infections, use insecticides and other means

## 4.9 Anaplasmosis

4.9.1 Definition: (Synonyms gall sickness) Anaplasmosis is not contagious; it is a tick transmitted disease. Anaplasmosis traditionally refers to a disease of ruminants caused by obligate intracellular (intraerythrocytic) Rickettsial bacteria of Genus *Anaplasma*. Cattle, sheep, goats, buffalo, and some wild ruminants can be infected with *Anaplasma*. Anaplasmosis occurs worldwide in tropical and subtropical regions.

4.9.2 Epidemiology: *A. bovis* (previously *Ehrlichia bovis*), and *A. platys*, all of which invade blood cells other than erythrocytes of their respective mammalian hosts. Bovine anaplasmosis is of economic significance in the cattle industry. Clinical bovine anaplasmosis is usually caused by *A. marginale*. Cattle are also infected with

*A. centrale* which generally results in mild disease. *A. ovis* may cause mild to severe disease in sheep, deer, and goats.

A replicative cycle occurs in the infected tick. Transplacental transmission has been reported and is usually associated with acute infection of the dam in the second or third trimester of gestation. Anaplasmosis may also be spread through the use of contaminated needles or dehorning or other surgical instruments.

There is a strong correlation between age of cattle and severity of disease. Calves are much more resistant to disease than older cattle. Recovered animals are chronically infected carriers. However, these chronically infected cattle may relapse to anaplasmosis when immunosuppressed (eg, by corticosteroids), when infected with other pathogens, or after splenectomy. In animals <1 yr old anaplasmosis is usually subclinical, in yearlings and 2 yr olds it is moderately severe, and in older cattle it is severe and often fatal. Anaplasmosis is characterized by progressive anemia due to extravascular destruction of infected and uninfected erythrocytes. The prepatent period of *A. marginale* is directly related to the infective dose and typically ranges from 15-36 days (although it may be as long as 100 days). After the prepatent period, peracute (most severe but rare), acute, or chronic anaplasmosis may follow. Rickettsemia approximately doubles every 24 hr during the exponential growth phase. Generally, 10-30% of erythrocytes are infected at peak rickettsemia, although this figure may be as high as 65%. RBC count, PCV, and hemoglobin values are all severely reduced. Macrocytic anemia with circulating reticulocytes may be present late in the disease. Animals with peracute infections succumb within a few hours of the onset of clinical signs. Acutely infected animals lose condition rapidly. Milk production falls. Inappetence, loss of coordination, breathlessness when exerted, and a rapid bounding pulse are usually evident in the late stages. The urine may be brown but, in contrast to babesiosis, hemoglobinuria does not occur. Mucous membranes appear pale and then yellow. Pregnant cows may abort. The carcasses lesions of cattle are generally markedly anemic and jaundiced. Blood is thin and watery. The liver may be mottled and yellow-orange. The gallbladder is often distended and contains thick brown or green bile.

4.9.3 Diagnosis: *A. marginale*, together with the hemoprotozoa *Babesia bovis* and *Babesia*

*bigemina* are the causative agents of tick fever in cattle. Blood in anticoagulant should also be obtained for hematologic testing. In Giemsa-stained thin blood films, *Anaplasma* spp appear as dense, homogeneously staining blue- purple inclusions 0.3-1.0 µm in diameter. Chronically infected carriers may be identified with a fair degree of accuracy by serologic testing using rMSP5 - ELISA, CFT, or card agglutination tests.

DNA-based detection methods are most useful as species & strain differentiation tests. At necropsy, thin blood films of liver, kidney, spleen, lungs, and peripheral blood should be prepared for microscopic examination.

4.9.4 Prevention and Control: Tetracycline and imidocarb are used for treatment. Imidocarb is highly efficacious against *A. marginale* as a single injection (as the dihydrochloride salt at 1.5 mg/kg, SC, or as imidocarb dipropionate at 3.0 mg/kg). Cattle may be sterilized by treatment with these drugs and remain immune to severe infection. Imidocarb is a suspected carcinogen with long withholding periods and is not approved for use in the USA or Europe. *A. marginale* grown in tick cell cultures are being investigated as an alternative live vaccine source. Subunit vaccines to control bovine anaplasmosis are under investigation.

In some areas, sustained stringent control or elimination of the arthropod vectors may be a viable control strategy; however immunization is recommended in other areas.

## 4.10 Babesiosis

4.10.1 Definition: (Synonyms - Babesiosis, Red water, Tick fever) Babesiosis is a tick borne disease of cattle caused by tick transmitted intraerythrocytic protozoan parasites *Babesia bovis*, *B. bigemina*, *B. divergens* and others. *B. bovis* is more pathogenic than *B. bigemina* and *B. divergens*. Infections are characterised by high fever, ataxia, anorexia, general circulatory shock, and sometimes also nervous signs as a result of sequestration of infected erythrocytes in cerebral capillaries.

4.10.2 Epidemiology: The main vectors of *Babesia bigemina* and *B. bovis* are 1-host tick *Boophilus* spp. in which transmission is transovarially. The risk of clinical disease in endemic areas is determined by 2 features: 1) Calves have a degree of immunity that related both to colostral derived antibodies and to age persist for nearly 6 months. 2) Recovered animals are generally solidly immuned but they become carriers.

Clinical signs: in the acute form generally runs a course of ~1 wk. The first sign is fever (frequently 105.8°F [41°C] or higher), which persists throughout the day, and is accompanied later by inappetence, increased respiratory rate, muscle tremors, anemia, jaundice, and weight loss; hemoglobinemia and hemoglobinuria occur in the final stages. Involvement of CNS is due to adhesion of parasitized erythrocytes in brain capillaries, this can occur with *B. bovis* infections. Either constipation or diarrhea may be present. Late-term pregnant cows may abort, and bulls may undergo temporary infertility due to transient fever. With

virulent strains of *B. bovis*, a hypotensive shock syndrome, combined with generalized nonspecific inflammation, coagulation disturbances, and erythrocytic stasis in capillaries, contribute to the pathogenesis. With most strains of *B. bigemina*, the pathogenic effects relate more directly to erythrocyte destruction.

- 4.10.3 Diagnosis: In differential diagnosis conditions that cause anemia, jaundice and red urine should be considered. Microscopic examination of Giemsa-stained blood or organ smears is essential. From the live animal, thick and thin blood smears should be prepared, preferably from capillaries in the ear or tail tip. The most commonly used serological tests are the indirect fluorescent antibody test and ELISA.
- 4.10.4 Prevention and Control: Diminazene aceturate, imidocarb dipropionate and long-acting tetracycline are used commonly for treatment. Supportive treatment is advisable, particularly in valuable animals, and may include the use of anti- inflammatory drugs, antioxidants, and corticosteroids. Blood transfusions may be life-saving in very anemic animals. Vaccination and tick elimination are helpful in successful efforts.

## 4.11 Theileriasis

- 4.11.1 Definition: (Synonyms –Theilerioses, Theileriases, East Coast fever ) It is tick transmitted, acute disease of cattle, characterized by high fever, swelling of the lymph nodes, dyspnea, and high mortality. East coast fever is caused by *Theileria parva*; it is a serious problem in east and central Africa.
- 4.11.2 Epidemiology: Theileria parasites of cattle are a major constraint to the improvement of the livestock industry in large parts of the old world. *T parva* sporozoites are injected into cattle by infected vector ticks, *Rhipicephalus appendiculatus*, during feeding. Clinical signs vary from mild to severe and fatal. There is fever, lymph node swelling that become pronounced and generalized. Anorexia develops and the animal rapidly loses condition; lacrimation and nasal discharge may occur. Terminally, dyspnea is common. Just before death, a sharp fall in body temperature is usual, and pulmonary exudate pours from the nostrils. Death usually occurs 18-24 days after infection.
- 4.11.3 Diagnosis: Important postmortem findings include lymph node enlargement with massive pulmonary edema and hyperemia. Hemorrhages are common on the serosal and mucosal surfaces of many organs, sometimes together with obvious areas of necrosis in the lymph nodes and thymus. Conventional Giemsa stained lymph node biopsy smears, obtained by needle puncture may contain multinuclear characteristic schizonts. The macroshizonts, are diagnostic they are called Koch's blue bodies.
- 4.11.4 Prevention and Control: Theilericidal compound parvaquone (Clexon) and, its derivative buparvaquone (Butalex) with long acting Tetracyclines are used for effective therapy. Rigid tick control is helpful.

## 4.12 Toxoplasmosis

- 4.12.1 Definition: Toxoplasmosis is a disease caused by a microscopic intracellular protozoan parasite *Toxoplasma gondii*, in the Phylum Apicomplexa. It is characterized clinically by abortion and stillbirth in pregnant ewes, and in all species by encephalitis, pneumonia and neonatal mortality. Human can get infected by ingesting *Toxoplasma gondii* eggs or cysts from fecal contaminated raw vegetables or uncooked meat. It can also be spread through contact with feces from an infected cat.
- 4.12.2 Epidemiology: The major forms of the parasite are: Oocysts (containing sporozoites), which are shed in the feces, Tachyzoites, rapidly multiplying organisms found in the tissues, Bradyzoites, slowly multiplying organisms found in the tissues, tissue cysts walled structures, often found in the muscles and central nervous system, containing dormant *T. gondii* bradyzoites. *Toxoplasma gondii* oocysts (eggs) are shed in the feces of infected cats. These eggs are then incidentally ingested by other animals through grazing. The protozoa can also be transmitted during pregnancy thereby infecting the unborn fetuses. Cats are required for the life cycle of the organism. Infection is most common in cats, sheep, goats, and dogs. Cattle seem to relatively resistant. Toxoplasmosis is a zoonotic disease and found worldwide.
- 4.12.3 Diagnosis: In naturally acquired disease incubation period varies from 5-23 days. Most animals that have toxoplasmosis show no signs of illness. In adult animals, particularly sheep, the most commonly noted sign is abortion. Disease is rare in people who are healthy. The greatest risk of disease is to, children, or pregnant women. Symptoms can begin with mild, flu-like signs. Severe disease can occur if the protozoan affects the nervous system, heart, lungs or eye. The organism can cause abortion or birth defects in pregnant women. Postmortem lesions are related to parasite migration through the tissues and organs, with accompanying necrosis. *T. gondii* is morphologically similar to other protozoa and must be differentiated from sarcocystis sp (in cattle) and neospora in (dogs) Toxoplasmosis is often diagnosed by serology. Serologic tests used in animals include ELISA, an indirect immunofluorescent antibody test, CFT, the Sabin-Feldman dye test, direct and indirect hemagglutination, latex agglutination and modified agglutination tests. IgG and IgM titers may be used.
- 4.12.4 Prevention and Control: Antibiotics and supportive therapy are used to treat clinical diseases. Cats should be kept away from pastures and barns where pregnant sheep and goats are kept. A modified live vaccine is available for sheep in Europe and New Zealand.

## Chapter (5) Poultry Diseases

- 5.1 Newcastle disease
- 5.2 Infectious laryngotracheitis
- 5.3 Infectious bursal disease
- 5.4 Infectious bronchitis
- 5.5 Fowl pox
- 5.6 Avian chlamydiosis (Psittacosis, Ornithosis)
- 5.7 Avian influenza
- 5.8 Marek's disease
- 5.9 Avian colibacillosis
- 5.10 Infectious coryza
- 5.11 Avian salmonellosis
- 5.12 Pullorum disease
- 5.13 Fowl Typhoid
- 5.14 Paratyphoid infection 5.15 Campylobacteriosis
- 5.16 Clostridial Necrotic dermatitis
- 5.17 Clostridial Nercotic enteritis
  
- 5.18 Fowl cholera
- 5.19 Mycoplasmosis (Mycoplasma gallisepticum infection)
- 5.20 Mycoplasma synoviae infection

### 5.1 Newcastle disease

5.1.1 Definition: (Synonyms: Avian pneumoencephalitis, pseudo-fowl pest, pseudo- poultry plague) Newcastle disease (ND) is an acute, rapidly spreading viral disease of domestic fowl characterized by the respiratory signs (coughing, sneezing, and rales) which are often accompanied or followed by nervous manifestations. In infections with some strains diarrhea and swelling of the head are witnessed.

5.1.2 Epidemiology The casual agent is (NDV) which is in *Genus Avulavirus Family Paramyxoviridae*, with varying severity of pathogenicity and transmissibility for chickens. Trade activities play a key role in the spread eg. frozen chicken products and transport containers. ND has been divided according to the clinical manifestations in chickens into five forms:

- a) Viscerotropic velogenic (VVND): characterized by acute fatal infection of birds of all ages, with predominant hemorrhagic lesions of the gastrointestinal tract and severe depression, mortality may reach 100%.
- b) Neurotropic velogenic (NVND): manifested with respiratory and nervous signs and high mortality. Cleavability of the viral fusion protein is a basic virulence attribute.
- c) Mesogenic: associated with milder forms of respiratory and nervous signs but accompanied by low mortality. Mortality may approach 25% or higher in young birds.
- d) Lentogenic respiratory: mild or unapparent respiratory infections.

- e) Asymptomatic enteric where there is an unapparent enteric infection.

Generally, respiratory signs are gasping and coughing. Nervous signs, which may accompany, but usually follow the respiratory signs, usually dominate in chronic cases, these include drooping wings, dragging legs, twisting of head and neck (torticollis), circling, walking backward (particularly after drinking water) and complete paralysis. Colonic spasms are seen in moribund birds. Viscerotropic signs include watery and greenish diarrhea and swellings of the tissues around the eyes and lower eyelid, and in the neck. Laying flocks may have partial or complete cessation of production that lasts up to 8 weeks or might fail to recover. Eggs abnormal in color, shape, or surface and with watery albumen are produced. ND is a worldwide notifiable disease that should be reported to the local veterinary authorities. In countries with developed poultry industries lentogenic form is most common and velogenic form considered exotic. It is perpetuated and disseminated by the movement of live birds and defects in biosecurity including multi-aged housing and failure to decontaminate clothing, equipments and vehicles. The virus can be introduced also by companion and migratory species. NDV occasionally infects humans and considered as an occupational hazard. Some cases show systemic symptomatology and conjunctivitis with signs resembling those of influenza. Susceptibility includes other variety of domestic and wild birds. In ducks, pheasants and geese mainly CNS involvement is observed.

Incubation period is varying; disease appears almost simultaneously throughout the flock 5 to 12 days after exposure, the average being 5 days.



Fig.23 Enlarged and necrotic cecal tonsils- ND



Fig.24 Hemorrhage in the mucosa of the proventriculus- ND

5.1.3 Diagnosis: Recently as traditional virus isolation is considered too tedious and requires extensive resources and time to be beneficial in an out breaks situations, the need for rapid diagnosis of both VVND and differentiation from HPAI has resulted in extensive adoption of PCR diagnostic technology. In the future, it may be possible to use multiplex PCR and gene microarray technology for identification. Serology is used to monitor the response of flocks to vaccination and to conduct retrospective diagnosis following exposure. Paired serum samples showing a rise in Hemagglutination inhibition antibodies serve to identify the disease. The disease must be differentiated from HPAI, laryngotracheitis, fowl cholera, fowl pox (diphtheric form), psittacosis in parrots. Gross Lesions are highly variable reflecting the variation in tropism and pathogenicity of the virus. Suggestive diagnosis can be built on signs that include head and lower eyelids edema accompanied by conjunctivitis. Severe inflammation of the trachea and air sacs, Hemorrhages and erosion of the esophagus. Hemorrhagic, necrotic or ulcerative lesions are noted throughout the GIT. In the intestine proper there is generally marked involvement of the cecal tonsils and Peyer's patches. In layers ovaries are regressed or hemorrhagic with a tendency of presence of misshapen eggs or development of yolk peritonitis in survivors.

5.1.4 Prevention and control: Live virus vaccines are widely used to prevent disease losses. Lentogenic strains, chiefly Hitchner B1 and LaSota are administered in drinking water or in spray or dust. Sometimes administration is by eye or nose drops.

Healthy chicks are vaccinated as early as the fourth or even the first day of life. However, delay until the second or third week avoids partial blockage of the active immune response by maternal antibody.

When other infections are present in the flock and where required by law, killed vaccines should be used. Vaccines with oil adjuvant give the longest protection. Programmed repeated vaccination must be considered to protect chickens throughout its life. The frequency of revaccination is largely dependent upon the risk of exposure and the virulence of the field virus. Mild respiratory reactions following administration of live attenuated vaccine is an unfortunate complication of protection.

Flocks which respond adversely should be evaluated for immuno-suppression due to early exposure to infectious bursal disease, Marek's disease or chicken anemia viruses. Climatic stress coupled with deficiencies in ventilation may lead to high level of ammonia, litter moisture and chilling which will exacerbate an adverse vaccine response.

## 5.2 Infectious Laryngotracheitis

5.2.1 Definition: Infectious Laryngotracheitis (ILT) is an acute and highly contagious viral disease of chickens and pheasants and may also take a sub acute form. The disease characterized by severe dyspnea, coughing, gasping, and expulsion of bloody exudates from nostrils and mouth. Mild form is associated with general unthriftiness, lacrimation, mucoid tracheitis, conjunctivitis and mild rales. In the acute form clinical findings are gasping, coughing, rattling and extension of the neck during inspiration. There is loss of appetite and activity.

The mouth and beak may be bloodstained from the tracheal exudates. Mortality may reach 50% in adults. Signs usually subside after 2 weeks, although some birds may cough for a month. The sub acute form has a low mortality, a more protracted course and is characterized by milder respiratory signs, lacrimation and conjunctival edema. Recovery takes place in a couple of weeks whereas severe cases take up to 4 weeks.

5.2.2 Epidemiology: The causative agent is a member of *Herpesviridae* family. Primarily chickens and pheasants are susceptible. No evidence that ILT is transmissible to human beings or other mammals. Geographic distribution is worldwide. The disease spreads via inhalation of aerosol and droplets from acutely infected birds or contact with clinically recovered or vaccinated birds. It spreads more slowly compared to ND, AI or IB. Mechanical transmission is another possibility. Incubation period ranges from 6 to 12 days following natural exposure.

5.2.3 Diagnosis: Clinical signs especially blood, mucus and yellow caseous exudates in the trachea are characteristic. These may be combined with dyspnea, loud gasping and coughing. In the sub acute form, punctiform hemorrhagic areas in trachea and larynx and conjunctivitis with lacrimation permit a presumptive diagnosis. A conclusive diagnosis may be made by

(1) demonstrating intranuclear inclusion bodies in the tracheal epithelium and isolation and identification of the specific virus.

(2) Nucleic acid probes and PCR especially when very small amount of viral DNA is present in the sample. Microscopically, a desquamative necrotizing tracheitis is characteristic. Differential diagnosis: Newcastle disease, infectious bronchitis, and infectious coryza.

The gross prominent lesion is the inflammation of the larynx and trachea leading to necrosis. It may extend to involve bronchi, lungs and air sacs. Occasionally occlusion of tracheal lumen with clotted blood is usually encountered. Formation of diphtheric or Pseudo membranes or cheesy material usually encountered in the lungs.



Fig. 25 Fibrino-hemorrhagic tracheitis- ILT

- 5.2.4 Prevention and Control: Keeping the birds quiet, lowering the dust level and using mild expectorants offer some relief. Vaccination should be practiced in endemic areas or farms where a specific diagnosis is made. Immediate vaccination of adults in the face of outbreak with modified strains of lower virulence supplied by drop to the eye (conjunctiva) shorten the disease course .Broiler flocks must be vaccinated when young, but protection is not likely to be solid when done under 4 weeks of age therefore revaccination is recommended when birds reach maturity. Its also feasible to establish and maintain an ILT free flocks particularly broiler flockswhere all-in all-out policy is practiced.

### 5.3 Infectious Bursal Disease (IBD)

- 5.3.1 Definition: (Synonyms - Gumboro disease.) IBD is an acute highly contagious viral disease of young chickens. The causative virus targets the lymphoid tissue with special predilection for bursa of Fabricius. It is characterized by edema and swelling of the cloacal bursa, necrosis of lymphoid elements, vent picking, prostration and mortality. Infection may be clinical or subclinical especially before the third week. Main clinical signs are sudden onset of severe prostration, dehydration, incoordination, diarrhea and straining, soiled vent feathers, vent picking and inflammation. The predilection of the virus for bursal lymphocytes leads to immunopathological consequences in recovered birds.

The occurrence of what called (viral bursectomy) lead to diminished antibody response to challenging pathogens. In the early stages the bursa is swollen, appears gelatinous and occasionally hemorrhagic. Concomitant immune-suppression due to destruction of immature lymphocytes in early subclinical infections, reduces the birds response to vaccination effects against other important poultry diseases e.g. ND with increased susceptibility to Coccidiosis, Salmonella and E.coli. Losses range to more than 20%. Maternally conferred antibodies may persist as long as 5 to 6 weeks, altering the clinical pattern. Recovered birds develop perfect immunity.

- 5.3.2 Epidemiology: IBDV is classified as Avibirnavirus that belong to *Birnaviridae* family. Chickens &turkey are the only birds that known to develop clinical disease. The disease has been diagnosed in younger as well as older birds; however it is most severe in chicks 3-6 weeks oldwhen the target organ bursa of Fabricius reaches its maximal stage of development. Accordingly chicks 1 - 4 days of age are less sensitive due to protection conveyed through maternal antibodies. Light breeds appear to be affected more severely than are meat-type chick. The disease has worldwide occurrence. The causal virus persists in the environment and is probably transferred by fomites. Water, feed and droppings taken from infected pens were infectious even after 52 days. Transmission occurs directly or indirectly, vertical transmission probably occurs through the eggs. Incubation period is very short, clinical signs are evident after only 2-3 days following exposure. Two serotypes have been identified. Serotype 2 seems to be less pathogenic.

- 5.3.3 Diagnosis: Acute clinical signs in susceptible flocks are easily recognized. Necropsy findings potentiate the evidence of occurrence. Distinctive changes in the size and color of the bursa during the course of infection in addition to isolation and identification of

the causative virus support diagnosis. Immunofluorescence of impession smears of bursal tissues, AGID, electron microscopy of bursal specimen are all of diagnostic value. VN and ELISA are reliable serodiagnostic methods. The disease should be differentiated from coccidiosis and certain nephrotoxic strain of infectious bronchitis virus. Gross lesions are prominent renal changes like swellings and degeneration due to acute nephrosis seen in dead birds. Dark discolored Pectoral Muscle congestion and hemorrhages in the thigh usually observed. The infected bursa often shows necrotic foci and at times petechial or ecymotic hemorrhages on the mucosal surface, when these are extensive birds may void blood in their droppings. Recovered birds usually have atrophied cloacal bursa due to the destruction and lack of follicle regeneration.

- 5.3.4 Prevention and control: There is no treatment. Depopulation and rigorous disinfection of contaminated farms have achieved limited success for IBDV is persistent. IBD live vaccines, of chick-embryo or cell-culture origin, can be administered by eye drop, drinking water or subcutaneously at 1 to 7 days of age. Chicks in broiler flocks (and in some commercial layer operations) should carry high levels of parental immunity to provide protection during early brooding. In situation where chicks have low or inconsistent level of maternal antibody, vaccination shall be carried out with attenuated virus starting at 1-2 weeks of age.

### 5.4 Infectious Bronchitis

- 5.4.1 Definition: Infectious Bronchitis (IB) or (gasping disease) is an acute, highly contagious viral infection of chickens, characterized by respiratory signs, rales, coughing and sneezing, range of kidney pathological changes, and significant drop in egg production feed efficacy and weight gain.

- 5.4.2 Epidemiology: IB is caused by a *Corona virus*. Many different serotypes are recognized and at least 10 serologic variant strains are so far identified. Geographic distribution is worldwide. Airborne transmission occurs from flock to flock in the direction of prevailing wind. Bird to bird spread is rapid. Transmission through fomites, secretions and discharges exist for many months after the primary infection.

Spread to other birds in the same pen takes place in 24 to 48 hours and usually all susceptible birds on a farm become infected. Incubation period is 18 to 48 hours after exposure. Coughing and rattling, slight nasal discharges occasionally are seen in young chicks only. The virus may be implicated in mixed infections that results in air sacculitis which cause increase in condemnation of broilers. The morbidity is 100%. Signs severity varies considerably. Respiratory signs cease in 2 or 3 weeks .Mortality ranges from 25-30% and is highest in very young chicks where it may reach 60% due to nephrogenic strains, but is negligible in birds older than 5 or 6 weeks. Feed consumption and egg production in laying fowls drop sharply, with production of many misshapen thin-or soft-shelled eggs of poor internal quality.

5.4.3 Diagnosis: Virus isolation in chick embryos, with the production of embryonic lesions, is the most certain procedure. A history of an acute, rapidly spreading, respiratory disease, which is not Newcastle disease, is good presumptive evidence. The primary target of viral replication is the trachea, but lungs ovaries and lymphoid tissue are also involved. RT-PCR methods are also employed in detection of the virus in the infected material after preliminary amplification in embryonated eggs. Serum neutralizing tests (3 weeks after onset or later) will yield significant antibody titers. At necropsy young chicks show sinusitis, catarrhal tracheitis, bronchitis and pulmonary edema. Lesions include facial edema and wet eyes, presence of serous, catarrhal and caseous exudates, excess mucus in the upper respiratory tract. Cloudy and thickened inflamed air sacs may also be present along with yellow caseous exudates in the abdominal air sacs. Swollen ureters containing uric acid crystals are occasionally noted. In fatal cases, primary and secondary may contain caseous plugs. Yolks and eggs with shells are often found in the abdominal cavity. Differential diagnosis includes Newcastle disease, ILT, infectious coryza.

5.4.4 Prevention and control: No medication is available. Increasing the temperature in the room and under the hover by (3 to 4°C) may lower mortality. Food consumption should be stimulated. Immunization by the use of live-virus vaccines given in the drinking water, or as a dust or spray administered according to the manufacturer instructions. Standard vaccines may not afford complete protection against some of variants, which has led to use of a variant strain vaccine in certain localized areas where I.B outbreaks in broilers is a significant problem. Vaccination earlier than 7 days of age may be unsuccessful, due to maternal passive immunity.

Use of ELISA serology in poultry health management has been widely accepted for many poultry diseases including infectious bronchitis. It is useful tool to monitor seroconversion or rise in I.B virus antibody titer as well as the immune response following vaccination. This help both in diagnosis of initial infection and vaccination failure and enables taking the required corrective actions.

## 5.5 Fowl Pox

5.5.1 Definition: A relatively slow-spreading viral infection characterized by the formation of cutaneous lesions on the head, neck, legs and feet, or diphtheric lesions in the mouth and upper digestive and respiratory tract. There are two forms, probably associated with different mode of infection. The most common form is transmitted probably by biting arthropods.

Clinically there are cutaneous lesions on the un-feathered parts of the head and upper neck manifested as small papules on the comb, wattles and around the beak. Generalized skin infection occurs in some flocks. Lesions in chicks are usually limited to feet and legs and around the cloaca, there is initially raised, blanched, nodular area that enlarges, becomes yellowish prior progressing to a heavy dark scab. The other form is probably associated with droplet infection and involves the mucus membranes of the mouth, pharynx, larynx and occasionally the trachea, with poor prognosis. Cutaneous infections alone ordinarily cause

little mortality and these flocks generally return to normal production upon recovery.

5.5.2 Epidemiology: Susceptibility includes chickens, pigeons and turkeys. Geographic distribution is worldwide. Fowl pox virus, a large DNA virus belongs to the family *Poxviridae*. It is highly resistant and may survive for years in dried scabs. The virus usually transmitted to pen mates by direct contact, or through skin abrasions and also via number of mosquito's vectors and possibly by inhalation, or movement of birds into contaminated building. Apparently some carriers remain following clinical recovery and reactivation may be caused by stress.

5.5.3 Diagnosis: When only small skin lesions are present, it is often difficult to distinguish the disease from abrasions caused by fighting. Appropriately stained sections or scrapings of affected areas usually reveal characteristic eosinophilic intracytoplasmic inclusion bodies which are diagnostic. Electron microscopy as well can be used to confirm the clinical diagnosis. Isolation can be performed via inoculation of avian cell cultures or chorioallantoic membrane of chick embryo. Differential diagnosis includes ILT and other respiratory diseases. Only few birds develop lesion at one time and multiple lesions often coalesce. Lesions in various stages of development can often be found on the same birds.



Fig. 26 Pox lesion in the eye & nostrils in poultry

Localization around the nostrils may cause nasal discharge. Lesions on the eyelids may result in lacrimation, closing of the lids and predispose birds to secondary bacterial infections. Lesions may also occur in the buccal mucosa or in the esophagus, larynx and trachea (wet pox or fowl diphtheria). Tracheal lesions, which may diffuse and progress to a heavy, brown pseudo membrane, may cause death by suffocation.



Fig.27 Pox lesions on the comb

- 5.5.4 Prevention and control: In areas where pox is prevalent, vaccination of chickens and turkeys with live-embryo-propagated virus is practiced. The most widely used vaccines are attenuated fowl pox virus and pigeon pox virus isolates with high immunizing ability and low pathogenicity. Because the infection spreads slowly, vaccination is often useful in limiting spread of infection if administered when less than 20% of the birds are infected.

## 5.6 Avian Chlamydiosis

- 5.6.1 Definition: (Synonyms: Psittacosis, Ornithosis) It's an acute or subacute or chronic disease of wild and domestic birds that characterized by respiratory and systemic infections. The main clinical findings include ocular and nasal discharges, conjunctivitis, and green to yellow diarrhea, dullness, weakness, hyperthermia, inappetence and loss of weight. The disease is commonly called psittacosis in psittacine birds (parrots), ornithosis in other birds (turkeys, pigeons, etc.) and psittacosis or ornithosis in man depending upon the type of bird from which it is contracted. It's usually systemic and rarely fatal.
- 5.6.2 Epidemiology: The cause is avian strains of *Chlamydia psittaci*, an extremely fastidious organism that requires living cells to multiply. All Chlamydia strains contain identical group-specific antigens but may differ in the antigenic specificity of their cell-wall antigens. The disease is particularly important in colonially nesting species, in domestic poultry (turkeys, pigeons, and ducks) and in caged birds. All strains appear to be infectious to man following heavy exposure to infective aerosols or dusts. The disease is of Public Health Significance. Outbreaks should be reported to public health and regulatory officials. Aerosols and dusts from respiratory discharges or digestive dejecta are infective. Infected birds may become carriers if they survive. Such carriers, under environmental stress, may have a recurrence and transmit the infection. Chlamydial strains of unusually high virulence that cause high mortality ranges up to 30% have been found in gulls and turkeys. Strains of less virulence are usually found in psittacine birds, pigeons, ducks and chickens.
- 5.6.3 Diagnosis: A tentative diagnosis may be made by detection of intra-cytoplasmic groups of Chlamydia in impression smears of diseased organs stained by the Gimenez, Giemsa or Machiavellos methods. Confirmation should be made by a laboratory competent to

isolate and identify Chlamydia. Freshly collected liver, spleen, kidney and lungs should be shipped frozen. Attempts to isolate the organism from cloacal excretions may also be made if it is necessary to preserve the life of a valuable bird. Serologic tests, particularly complement fixation and agar gel precipitation, direct and competitive ELISA are of value. Differential diagnosis includes Influenza; mycoplasmosis, air-sac disease. Fowl cholera may produce similar lesions in turkey. Air sacculitis, pericarditis, perihepatitis and peritonitis with sero-fibrinous exudates are common necropsy findings in acutely affected birds and hepatosplenomegaly with discoloration which characterize chronic cases.

- 5.6.4 Prevention and Control: Chlamydiosis is relatively rare in food birds. Why the outbreaks are sporadic is not clear. Preventive measures, such as screening houses against wild bird entry, are justified. A simple preventive measure for the veterinarian examining dead birds is the use of a detergent disinfectant to wet the feathers; in addition to all appropriate protective clothing. No effective vaccine for use in bird is available. Tetracyclines (chlortetracycline, oxytetracycline, and doxycycline) are the antibiotic of choice. Pet birds should come from breeding establishments free of the infection. If the infection status is not known, chlortetracycline (CTC) impregnated seed should be fed to the birds to reduce the possibility of infection during transport and during stays in pet stores.

In confirmed cases treatment should be continued for a minimum of 3 weeks. Appropriate biosecurity practices are vital to control introduction and spread of infection.

## 5.7 Avian Influenza

- 5.7.1 Definition: (Synonyms - Bird flu, fowl plague, typhus exudative gallinarium, Brunswick bird plaque). Avian influenza (AI) is an infectious viral disease of most avian species, manifested by short course and high mortality occasionally approaching 100%. In poultry, the viruses cause two distinctly different forms of disease ranges from asymptomatic common mild form or (MPAI) to acute generalized highly fatal disease (HPAI). Most frequently the signs include decreased activity, loss of appetite, emaciation, increased broodiness of hens, sharp decrease in egg production and egg often without shells. Profuse watery diarrhea, ruffled feathers, swollen and cyanotic combs, edematous wattle and edema around the eyes, head and neck are frequently seen in mature chickens. Conjunctivae may be congested, swollen and occasionally hemorrhagic. Respiratory signs may vary from mild to severe with coughing, sneezing, rales. Excessive lacrimation and sinusitis are evident, occasionally there is blood stained cloacae and dark red skin. The disease is a cause of tremendous economic losses due to mortality drastic drop in egg and meat production and the cost of control measures that include stamping out and depopulation.
- 5.7.2 Epidemiology: The epidemiology of AI is poorly understood because of the role of wild birds, the great variety of different strains and the variable effects in different host species. AI is caused by type A strains of the influenza virus belong to *Orthomyxoviridae* family. There are known serologically distinct subtypes based on the hemagglutinins & neuraminidase

surface glycoproteins (16 HA and 9 NA), each with virulent and a non-virulent members. The vast majority of these viruses cause no harm. To date, all outbreaks of the highly pathogenic (HPAI) form of avian influenza have been caused by viruses of the H5 and H7 subtypes. Highly pathogenic viruses possess a distinctive set of basic amino acids in the cleavage site of the HA – that associated with their exceptional virulence. This facilitates the HA activation by proteolytic cleavage in a wide host cell spectrum both *in vitro* and *in vivo*. All birds are thought to be susceptible however, e.g. free flying aquatic birds, migrating waterfowl and shore birds can carry these viruses with no apparent signs of harm. It has been isolated from 90 species of free living birds representing 13 different orders. Suspected outbreaks should be reported to veterinary authorities. Since 1959, instances of human infection have been documented on only 10 occasions. Of the hundreds of strains of avian influenza A viruses, H5N1, H7N3, H7N7, and H9N2 are known to have caused human infections resulting in mild and very little severe illness, with one notable exception: the highly pathogenic H5N1 virus. The disease occurs worldwide. Transmission requires very close contact between birds and airborne spread does not seem to play a major role. Indirect contact through aerosol droplets or exposure to virus contaminated fomites is also important.

Since virus is excreted in large amounts with feces in which it can survive for a long periods, mechanical spread may occur via birds, man, fomites, water and food, equipments shared in production or in live bird markets (LBM). Highly pathogenic viruses can survive for long periods when temperatures are low. Incubation period is highly variable from few hours to few days. The fear caused by highly pathogenic Avian Influenza (HPAI) viruses to some times obscures the importance of the much larger and more common low pathogenic group of (LPAI) viruses. LPAI viruses include all the serotypes other than H5 & H7 groups; the best known among them is the H9N2 sero-group which is widespread and endemic virus in far and Middle Eastern countries. H9N2 caused serious disease and mortality in birds under field conditions and have been reported to infect humans causing a slight disease. H9N2 shares some genomic characteristics with H5N1. The clinical and pathological signs include swollen heads, sinusitis, and respiratory signs with accumulation of a cheese-like content in the air sacs.

**5.7.3 Diagnosis:** Definitive diagnosis is established by direct detection of AI viral proteins or nucleic acid (genes) in the specimens such as tissues, swabs, cell cultures using molecular diagnostic methods like (PCR), and by Isolation and identification of the causative virus. Presumptive diagnosis can be made by detecting the virus specific antibodies. Important infections that must be considered in the differential diagnosis are viscerotropic velogenic Newcastle disease, avian pneumovirus, fowl cholera, ILT, IB, mycoplasmosis and chlamydiosis (ornithosis).

Affected birds may show no significant gross lesions, other than dehydration and pronounced carcass congestion. In severe cases there are various congestive, hemorrhagic and necrotic changes. Necrotic foci on the skin, wattles and comb and mild to moderate conjunctivitis, sinusitis, tracheitis, and air sacculitis encountered in most disease outbreaks. Bloody cloacae, dark colored skin, tracheal edema and fibrinous exudates in the air sacs, pericardium, peritoneum, and oviduct are also described. Visceral lesions vary with necrotic

foci appearing in spleen, liver, kidney, and lung. Petechial and ecchymotic hemorrhages are found in the abdominal fat, proventriculus, and small intestine.

**5.7.4 Prevention and control:** Suspected outbreak must be reported to the regulatory authorities. Legislative measures including trade limitation, quarantine are of importance in control. Effective biosecurity measures should be in place to prevent the introduction of the infection and its spread. This could be attainable through rigorous control of movement of birds, people and equipments. Vaccination is useful only after the identification of the circulating virus strain. Because of the risk of reassortment with wild viruses only inactivated vaccines should be used and because of the diversity of the serotypes these must be polyvalent. Inactivated vaccines, vectored and DNA vaccines incorporating HA genes are used successfully to confer immunization. Antibiotic treatment to combat secondary bacterial infection and increasing rearing houses temperature help in reducing mortality.



Fig. 28 A.I. Severe necrosis of comb and wattle

## 5.8 Marek's Disease

**5.8.1 Definition:** A highly contagious, progressive lymphoproliferative disease of poultry, characterized by gross enlargements of the nerves; and tumors of the visceral organs, skin and muscle caused by mononuclear and lymphoid cellular infiltration and proliferation. Atrophy of the follicles and inter follicular infiltration of the Bursa of Fabricius may also occur. Synonyms are fowl paralysis, neuro-lymphomatosis gallinarum and acute leucosis.

Four overlapping syndromes are described: neurolymphomatosis or classical Marek's disease which associated with paralysis of one or both legs or wings, acute Marek's disease that occurs in explosive outbreaks without localizing neurologic signs, ocular lymphomatosis which leads to graying of the iris, irregular eccentric pupil and blindness, and the cutaneous Marek's disease which is characterized by nodular lesion up to 1 cm of feather follicle. Clinical findings are lameness or paralysis of the legs, wings, neck, eyelids or other parts of the body which are classical observations. Some cases show what is called the hurdle jumper

position. Occasionally, dyspnea, ataxia, crop dilation, diarrhea and emaciation occur. Young birds affected by the acute form may only show signs of anorexia and depression. When the eyes are involved the iris is discolored gray and the papillary margin becomes irregular. Terminal blindness occurs due to loss of light accommodation.

- 5.8.2 Epidemiology: The causative agent is Mareks disease virus which is a DNA virus from Family: *Herpesviridae*. It has three identified serotypes. Different forms of infection result from interaction between viruses and host cell: productive (occurs in B lymphocytes & the feather follicles), latent (in activated T lymphocytes) and neoplastic transformation in latently transformed T cells that eventually result in lymphoid neoplasms. The main route is Inhalation of aerosols of contaminated dander. The virus has the ability to spread readily over long distances through aerosols of infectious dander, and to lesser extend through eggs.

Commonly birds aged 12 - 24 week are principally affected; some cases observed as early as 3 weeks and as late as 18 months when chicken attain sexual maturity. The occurrence of disease depends on the genetic resistance of the chicken, the age at which the bird becomes infected, the dose and strain pathogenicity of the virus.

- 5.8.3 Diagnosis: Classifications of the avian leucosis complex can often be made by gross inspection of lesions. In uncertain cases, histo-pathological sections of tissues, bone marrow and blood smears are needed for definitive diagnosis. The characteristic feature is predominant neoplastic lesions of the viscera, skin and muscle. Enlargements of one or more of the peripheral or autonomic nerves, where the brachial, sciatic, celiac and vagus nerves are most frequently involved. There are losses of striations of the thickened nerves which show grayish and edematous appearance. Lymphomas are frequent in the ovary, but may also develop in the, liver, lung, skin, muscle, kidney, proventriculus and other organs. Differential diagnosis include lymphoid leucosis, and reticuloendotheliosis, however this need high skill and expertise. Other diseases, such as tuberculosis and pullorum disease, must be differentiated from the neoplastic diseases by tissue distribution, color, consistency, texture and association with other inflammatory and degenerative lesions. Detection of viral antigens could be done by immunofluorescence, gel diffusion and indirect immunofluorescence. The disease must be differentiated from other diseases of the nervous system, such as Newcastle disease, encephalomyelitis, and encephalomalacia in addition to riboflavin deficiency.

- 5.8.4 Prevention and Control: Control is based on vaccination and implementation of strict biosecurity measures. Bivalent vaccines consisting of turkey herpesvirus and either the SB-1 or 301B/1 strains of serotype 2 in addition to attenuated types like CVI988/Rispens are successfully used. Hygienic rearing techniques based on preventing the introduction of disease by careless caretakers, infected embryos or chicks, and prevention of contact infection between infected and uninfected stock. Eggs or chicks from different strains or hatcheries should not be hatched or brooded together. Premises should be thoroughly cleaned and disinfected before the introduction of new stock. All-in, all-out brooding should be practiced for any one location. During the first 3 months of life where the

susceptibility is high, birds should be maintained in isolation away from sources of Marek's disease, including aerosols of dust and dander. It is highly recommended to provide filtered, positive pressure air supply within the house.

## 5.9 Avian Colibacillosis

- 5.9.1 Definition: (Synonyms - Colisepticemia; *Escherichia coli* infection) Colibacillosis refers to any localized or systemic infection caused entirely or partly by many serotypes Avian Pathogenic *Escherichia coli* (APEC). These include coli septicemia, coligranuloma (Hjarres disease), air sac disease, coliform cellulitis, swollen head syndrome, panophthalmitis, and coliform peritonitis. However, signs are nonspecific and vary with age, organ involved and concurrent disease. The disease may occur as an acute fatal septicemia or sub acute pericarditis and air sacculitis.

- 5.9.2 Epidemiology: *E.coli* is normally found in the intestine of poultry and most other animals and generally is not pathogenic. *E.coli* Strains of 02, 078 and 01 are most pathogenic.

Systemic infection occurs when it gain access to the blood stream from the respiratory or intestine. Susceptible hosts include chicken and turkey principally. Large numbers of *E.coli* are maintained in the poultry house environment through fecal contamination but the initial exposure to pathogenic strains may occur in the hatchery. Most APEC isolated forms poultry represent a low risk for people or other animals. Exception is chicken colonization with *E.coli* 0157:H7 an important Shiga toxin-producing enterohemorrhagic pathogen of human. Infection persists as worldwide problem responsible for extensive financial loss. Outbreaks occur following exposure to primary respiratory pathogens responsible for lentogenic ND, metapneumovirus infection and IB, especially when coupled with environmental stress. Broiler flock subjected to early immunosuppression with Mareks disease, IBD or chicken anemia or flocks fed diet containing mycotoxin or rancid fats will be susceptible to secondary *E.coli* infection. Range of various clinical diseases like air sacculitis, swollen head syndrome, infectious peritonitis, coliform salpingitis and omphalitis are associated with *E.coli* secondary infections. Transmission is by direct or indirect contact. Free living birds are especially important as they might be colonized with avian adapted strains.

- 5.9.3 Diagnosis: Isolation & identification of the organism from lesions typical of colibacillosis. In conducting the differential diagnosis many other organisms should borne in mind including viruses, mycoplasmas, and other bacteria which can cause similar synovial lesions. Organism like Aerobacter spp., Klebsiella spp., Proteus spp., Salmonellae, Bacillus spp. Staphylococci, enterococci or clostridia frequently isolated from yolk sac of embryos and chicks. Pericarditis can also be caused by Chlamydia. Peritonitis can also caused by Pasteurella or streptococci. Certain bacteria, Mycoplasmas, and Chlamydia can cause air sacculitis. There are few lesions in the young birds succumb to acute septicemia that include enlarged hyperemic liver perihepatitis and spleen with increased fluid in body cavities.

- 5.9.4 Prevention and control: Effective measures include improvement of management procedures to control the predisposing factors. Introduction of effective intervention strategies, elimination of carriers, early use of antibacterial indicated by susceptibility tests, in addition to immunization is of utmost importance. In acute cases antibiotics like streptomycin, tetracycline, gentamycin or sulfaquinoxaline are effective.

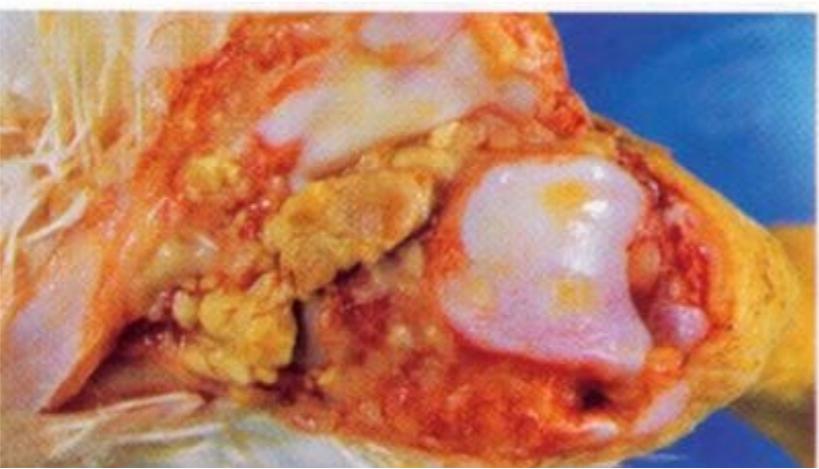


Fig.29 Arthritis involving hock joint and flexor tendon in broiler (E.coli)

## 5.10 Infectious Coryza

- 5.10.1 Definition: An acute respiratory disease of chickens, affecting primarily the nasal passages and characterized by nasal discharge sneezing, facial edema and swelling. Less frequently there is a lower respiratory tract infection causing rales and difficult breathing. In its mildest form, the only signs are a serous mucoid nasal discharge, depression and occasional facial swelling with little or no systemic effect. In the more severe form (usually in adult Leghorn or heavy breeders) there is severe swelling of one or both infra-orbital sinuses which may lead to closure of both eyes. In adult birds (especially in males) the edema might extend to the inter-mandibular space and wattles. In chronic cases where other infectious agents are involved the swelling may persist for months.

Sinuses become distended with yellow caseous exudates. Other signs may include conjunctivitis, tracheitis, and bronchitis and air sacculitis.

- 5.10.2 Epidemiology: *Haemophilus paragallinarum (gallinarum)*, the causative agent is a hemophilic, gram-negative, polymorphic, nonmotile bacterium. There are several serotypes with at least 3 distinct immuno-types. Susceptible host are mainly chicken. All ages are susceptible but it is usually less severe in juvenile birds and has no public health significance. Geographic distribution is worldwide and is quite important in tropical climates; it's a common problem in the intensive chicken industry. Infectious coryza is not an egg transmitted disease. Chronic healthy carriers are the main reservoir of infection. Air borne route is also important. The disease has a very short incubation period of 24 - 48 hours. In the absence of a concurrent infection the course extends to 2-3 weeks.

- 5.10.3 Diagnosis: The most reliable diagnostic procedure is isolation from flocks with rapidly spreading coryza. The reproduction of the typical disease by contact or intranasal inoculation and demonstration of *H.paragallinarum* may confirm the disease. A history of a nasal discharge that quickly affects a large percentage of the chickens is good presumptive evidence. However, swelling of the face and wattles must be differentiated from mycoplasmosis, ILT, ND and vitamin A deficiency.

- 5.10.4 Prevention and Control: Prevention which is the only rewarding method for reliable control is best achieved by the (all-in all-out) policy. In endemic area, all replacements should only be made with day-old chicks unless the bird source is known to be free of infectious respiratory disease. Survivors of an outbreak should be completely and permanently separated from all other chickens either by segregation in a separate house or removal from the premises. Early treatment is important. Oxytetracycline and Erythromycin are recommended. Various sulfonamides have been used successfully but must not be used in layers. In more severe outbreaks although treatment may result in improvement, the disease may recur when medication is discontinued. When proper disinfection of rearing house is difficult to assured, preventive medication may be combined with a vaccination program using bacterins for started pullets.

## 5.11 Avian Salmonellosis

- 5.11.1 Definition Infections with bacteria of Genus *Salmonella* are responsible for a variety of acute and chronic diseases in poultry. They could be divided into those caused by : (1) Two species highly host-adapted to the chicken and turkey that have significant economic importance, like *S. pullorum*, the causative agents of pullorum disease and *S. gallinarum* which causes fowl typhoid. (2) The non-host adapted species (paratyphoid species) e.g. *S. typhimurium* and *S. enteritidis*, may be transmitted among almost all animals. These groups have major public health significance because of the possible infection of man from contaminated poultry food products.

## 5.12 Pullorum Disease (PD)

- 5.12.1. Definition: (Synonyms: Bacillary white diarrhea) Pullorum disease (PD) infection by *S. pullorum* and *S. gallinarum* respectively, usually cause high mortality in young chickens and turkeys and occasionally in adult chickens. Affected birds huddle near a source of heat, do not eat, appear sleepy and show whitish fecal pasting around the vent. Survivors frequently become asymptomatic carriers with localized infection in the ovary. Some of the eggs laid by infected hens hatch and produce infected progeny.

- 5.12.2 Epidemiology: PD can infect human but only in cases of massive exposure following ingestion of heavily contaminated food, where it causes rapid onset of acute enteritis followed by prompt recovery without treatment. Both disease PD& FT are of a little public health significance. Transmission chiefly occurs through egg but may also occur by direct or indirect contact. Egg or hatchery-transmitted infection usually results in high mortality may rang up to 100% during the first few days of life and continue to 2 to 3 weeks of age.



Fig.30 Nodular lesion in the heart- *Salmonella pullorum*



Fig.31 Swollen hock containing yellow viscous fluid- *Salmonella*

5.12.3 Diagnosis: Lesions may be highly suggestive, but diagnosis should be confirmed by isolation and identification of *S. pullorum*. Infections in mature birds can be identified by serologic tests followed by necropsy and culturing for confirmation. The clinical signs and Lesions of PD and FT are not pathognomonic. Similar lesions can be produced by other pathogenic micro organisms e.g. Aspergillus and other fungi in the lungs. Similar lesions in joints and tendon sheath are caused by Mycoplasmas, *Staphylococcus aureus*, *Pasteurella multocida*. Marek's disease, on the other hand produces white nodules in the heart of young chicks. Lesions in young birds usually include unabsorbed yolk sacs, focal necrosis of the liver and spleen and grayish nodules in the lungs, heart and gizzard muscle. Firm cheesy material in the ceca and raised plaques in the mucosa of the lower intestine are sometimes seen. Occasionally synovitis is prominent. Adult carriers sometimes have no gross lesions but usually they develop pericarditis, peritonitis or distorted ovarian follicles with coagulated contents. Acute infections in mature chickens produce lesions that are indistinguishable from those of fowl typhoid.

5.12.4 Prevention and control: Improvement of management procedures, introduction of effective intervention strategies e.g. probiotics, and elimination of carriers are basic efforts in any

control attempts. Vaccination using variant or polyvalent antigens is sometime necessary. Several sulfonamides, antibiotics and other antibacterial are effective in reducing mortality but not eliminate the infection from a flock. Furazolidone at a level of 0.002% in the feed is one of the most effective treatments .Control is based on selection of commercial flocks from tested breeding stock to assure freedom from infection.

### 5.13 Fowl Typhoid (FT)

- 5.13.1 Definition: The casual agent of Fowl Typhoid (FT) is *S. gallinarum* which is very similar to *S. pullorum*, and many workers consider them as one species. It shares many common feature& similarities with PD in clinical sings, epizootiology, lesions and control and eradication procedures.
- 5.13.2 Epidemiology: Transmission occurs in several ways, horizontally through contact and vertically via eggs. Egg transmission results from contamination of ovum following ovulation, but localization of *S. Pullorum* and *gallinarum* in the ovules before ovulation is likely constitutes the chief mode of vertical transmission. The disease is frequently referred to as a disease of adult birds. It produces lesions in chicks and poult similar to those produced by *S. pullorum*; it has a much greater tendency to be spread among growing or mature flocks. Mortality at all ages is usually high.
- 5.13.3 Diagnosis: Diagnosis is accomplished by isolation and identification of the causal agent by standard bacteriologic methods. In the older bird gross lesions consist of dehydration, swollen, friable and often bile stained liver with or without necrotic foci, enlarged spleen and kidneys, anemia and enteritis.
- 5.13.4 Prevention and control: The same as for pullorum disease. Vaccine made of a rough stain of *S. gallinarum* (9R) is useful in controlling mortality; it is usually most effective if administered at 9 to 10 weeks for age before natural exposure occurs. The standard serologic tests for pullorum disease are equally effective in detecting fowl typhoid.

### 5.14 Paratyphoid infection

- 5.14.1 Definition: Paratyphoid (PT) infection may be caused by any of the many non host-adapted motile salmonella serotypes. *S. typhimurium* is the most common cause, followed by *S. enteritidis* PT4 and *S. heidelberg*.Infections are often subclinical. Mortality is chiefly confined to the first few weeks of age. Shipping, delayed feeding, chilling or overheating increase mortality which is usually higher in ducks and turkeys than in chickens. The contamination of eggs may lead to high level of embryo mortality and rapid death of newly hatched birds before clinical sings are observed. Depression, progressive somnolence with closed eyes, drooping wings, and ruffled feathers, poor growth, weakness, diarrhea and dehydration maybe observed.
- 5.14.2 Epidemiology: Infections are common in all species of domesticated birds. Frequently several *Salmonellae* infect a bird or flock concurrently. Geographic distribution is world

wide. The prevalence of species other than *S.typhimurium* and *S. enteritidis* varies widely by geographic location and strain of bird. For instance phage type 4 *S. enteritidis* is widespread in parts of Europe and cause mortality of up to 20% in the first 3 weeks of life. This serotype and some of its strains may cause a substantial incidence of infection of the reproductive tract in hens, with true vertical transmission and important public health implications. Usually the incidence in young flocks is high, but declines when maturity is attained. PT salmonellae can be introduced into poultry flock by contaminated feed, biologic vectors wild birds, insects and rodents. Mice have been identified as particularly important vectors for *S. enteritidis* in laying flocks. Humans can also be a source of salmonella transmissible to poultry. Efforts to control paratyphoid infections in domestic poultry are chiefly stimulated by public health consideration.

5.14.3 Diagnosis: Isolation and identification of the causative agent is necessary. Direct culture from liver and yolk sac is adequate for isolation. Samples must be obtained from different sources, principally tissues, eggs and poultry house environment. Rapid detection technologies e.g. ELISA methods or molecular diagnosis PCR have revolutionized the laboratory based salmonella diagnosis. Usually, there may be no characteristic observable gross Lesions. Occasionally, enlarged liver with or without areas of focal necrosis, unabsorbed yolk sac are encountered. Localization in the eye or synovial tissues is reported.

5.14.4 Prevention and Control: Several antibacterial agents e.g. enrofloxacin are of value in preventing mortality; none is capable of eliminating flock infection. Antibiotics have also sometimes been added to poultry feeds at therapeutic and subtherapeutic levels have been shown to select drug resistant strains thereby potentially compromising their effectiveness in human and animals. Biosecurity and strict sanitation is mandatory in preventing transmission between successive lots of birds in a house. Maintenance of poultry in confinement and exclusion of all pets, wild birds and rodents help to prevent introduction. Early fumigation of hatching eggs is recommended to prevent penetration of the shell surface. The heat of pelletting is reasonably effective in destroying salmonellae in feed ingredients. Several methods of determining the salmonella status of breeding flocks have been devised. Multiple resistant strains insensitive to antimicrobial agents necessitate other intervention strategies like competitive exclusion, where early colonization of the gut with selected normal micro flora results in significant resistance to subsequent exposure. Vaccination with either killed or live preparations has been found to reduce the susceptibility to the infection. However, control methods have not been developed to the point of dependability.

## 5.15 Campylobacteriosis

5.15.1 Definition: *Campylobacter jejuni* colonizes the intestine of chickens, turkeys, and waterfowl but is generally nonpathogenic and may form normal part of the gut flora in mature poultry. It belongs to a thermophilic subgroup, which is widely distributed in most warm-blooded domestic and wild animals. The majority of infections appear to be sporadic. Some strains cause enteritis and death in newly hatched chicks and pouls. Poultry meat intended for human consumption is the major vehicle incriminated as a source of infection. Generally,

infection in mature commercial poultry flocks is not associated with any obvious clinical signs. Experimental infection on the other hand did not produce any clinical abnormalities in broiler chicks aged 2 days or 3 weeks despite of intestinal colonization.

5.15.2 Epidemiology: *Campylobacter jejuni* is commonly isolated from poultry. It is the predominant species and is clinically innocuous. Commercial poultry and free-living birds are natural reservoirs of (*C. jejuni*, *C. coli*, and *C. lari*). The disease is prevalent in food animals such as poultry, cattle, pigs, sheep, ostriches and shellfish. The livestock presence near or on broiler farms has been considered as risk factor. Campylobacteriosis is considered to be a zoonosis. The high incidence of campylobacter diarrhea in human and its possible sequelae makes it highly important from a socio-economic perspective and fatal outcome is rare except in children. In almost all developed countries, the incidence of human campylobacter infections has been steadily increasing for several years. The reasons for this are unknown. It is present in all areas where commercial poultry is raised. On some occasion the bacteria has been cultured from up to 100% of both live broilers and 100% of processed poultry. Egg borne transmission from parent flocks to broilers progeny has been evidenced. Personnel and equipments movement mechanically spread the infection, whereas fomites and insects especially beetles and houseflies may be responsible for indirect transmission.

As *C. jejuni* is intolerant to desiccation, recovery from broiler litter is limited to substrate with water activity value exceeding 0.85. Within 24 days of identifying fecal sheds flocks show 90% - 100% prevalence rate due to rapid inter-flock transmission. Incubation period is 24 hours in experimental trials.

5.15.3 Diagnosis: Isolation and identification is essential to prove the infection. However, conventional microbiological procedure for isolation and identification and enumeration of bacteria are laborious and time consuming. Thus, direct plating with secondary enrichment for qualitative detection is more reliable. Other techniques employed in epidemiological studies include: Penner & Loir serotyping, multi-locus enzyme electrophoresis, DNA restriction endonuclease analysis, phage typing, and plasmid analysis, in addition to highly sensitive PCR methods. Principal pathologic changes of diagnostic value that noted in chicks are intestinal tract distension and accumulation of watery fluid with or without hemorrhages. Focal hepatic necrosis may be noted. In some occasions red or yellow mottled liver is seen in newly hatched chicks.

5.15.4 Prevention and Control: Treatment is not generally indicated, except electrolyte replacement and rehydration. Antimicrobial treatment (erythromycin, tetracycline, and quinolones) is indicated to eliminate the carrier state. The prevention of infection requires control measures at all stages of the food chain, from agricultural production on the farm, to processing. Specific intervention methods on the farm reduced the incidence in poultry. Measures include enhanced biosecurity to avoid horizontal transmission from the environment to the birds. This control option is feasible only where birds are kept in closed housing conditions. Good hygienic slaughtering practices are essential to reduce contamination of carcasses by faeces such as improved washing of carcasses, use of counter-flow scalding, elimination of immersion chillers, and reduction of manual handling by installation of

advanced automated equipment. Chemical disinfectants (glutaraldehyde (0.125%) and succinic acid (3%), and organic ones (lactic and acetic acids) may be used to destroy *C. jejuni*. Education in hygienic handling of foods for poultry slaughterhouses workers in the production of raw chicken meat is necessary to keep microbiological contamination to a minimum.

## 5.16 Clostridial Necrotic Dermatitis

- 5.16.1 Definition: (Synonyms: Clostridial dermatomyositis, Gangrenous dermatitis, and Gangrenous cellulitis). Necrotic dermatitis is an infectious disease of chickens characterized by sudden onset, sharp increase in mortality, and gangrenous necrosis of the skin over the thighs and breast. Clinically there is sudden dramatic increase in mortality in the affected flock ranging from 10 to 60% within 8 to 24 hours. This preceded by signs of extreme depression, lameness, lethargy and prostration. Externally, there are patches of red to black gangrenous skin over the breast, abdomen, wing tips or thighs. Frequently feather loss or sloughing of the epidermis is noted. Palpation of the affected area often reveals crepitation due to gas bubbles in the subcutaneous tissues, and musculature usually exhibits a cooked appearance.
- 5.16.2 Epidemiology: *Clostridium septicum* is the most commonly isolated pathogen but other clostridia, notably *Cl. perfringens* Type A, and *Cl. novyi* has been reported. Other bacteria, mainly *E. coli* might be involved. Important predisposing factors include skin trauma from surgical procedures, mechanical devices or cannibalism, heavily contaminated moist built-up litter and devitalization of the skin as occurs in staphylococcal infection and in selenium deficiency. The disease occurs sporadically in growing chickens from 4 to 16 weeks of age, affects both broilers and layer replacement stock, and occasionally causes outbreaks in turkeys.
- 5.16.3 Diagnosis: Histopathology demonstration of gas gangrene and numerous large filamentous bacilli in the skin, musculature and liver, and isolation of the causative clostridia, coupled with the history and clinical finding will differentiate this condition from exudative diathesis (selenium deficiency), staphylococcal infection and other diseases involving the skin. At necropsy, there is an accumulation of gaseous, serosanguinous fluid in the subcutis, and the musculature has a pale appearance. There is a combined hepatomegaly and spleenomegaly with abundant areas of necrotic infarcts or pale focal areas of necrosis.



Fig.32 Necrotic Dermatitis in wing (left) & Abdomen (right)

The kidneys are usually swollen and the lungs congested and edematous or necrotic. Atrophy of the bursa of Fabricius may be a frequent finding in chickens that were exposed to IBD.

- 5.16.4 Prevention and Control: Usually, this disease can be prevented by maintaining proper litter condition, minimizing traumatic and mechanical injury and controlling cannibalism through effective debeaking or other procedures. The predisposing effects of early infection with IBDV may be avoided by assuring a substantial maternal antibody level in day old chicks. The causative organisms are sensitive to a wide variety of antibiotics in vitro, especially oxytetracycline or chlortetracycline at 0.02% in the feed which produced a rapid decline of mortality in field outbreaks.

## 5.17 Clostridial Necrotic Enteritis

- 5.17.1 Definition: An acute enterotoxaemia of young chickens. It characterized by sudden onset, explosive mortality, and confluent necrosis of the mucosa of the small intestine. Infected chickens are extremely depressed with decreased appetite and reluctant to move, and may have diarrhea. The disease progresses rapidly, and death occurs within an hour or two. This disease persists in a flock for 5 to 10 days. Flock mortality varies from 2 to 50%.
- 5.17.2 Epidemiology: Recent evidence indicates that toxins elaborated by *Cl. perfringens*, Types A and C. Some studies consider *Cl. perfringens* as the principal obligate anaerobic bacterium in the intestinal tract of chickens. The disease affects primarily broiler at younger ages throughout the world. However, outbreaks occasionally reported in broiler chickens 2 weeks to 6 months old, especially at 2-5 weeks ages. Also outbreaks occurred in 3-6 month old commercial layers. Mixed infection with coccidiosis reported in 12-16 old cage reared layer replacement broilers. The disease has been reported from most areas of the world where poultry is produced. The organism is found in large numbers in feces, soil, dust, contaminated feed and litter.
- 5.17.3 Diagnosis: It can be made based on typical gross & microscopic lesions and isolation of the causative agent. Mucosal intestinal surfaces look pale and necrotic and usually fissured and detached.



Fig. 33 Necrotic enteritis *Cl. perfringens* with concurrent coccidiosis

Lesions produced by coccidia can be similar to those in necrotic enteritis, but uncomplicated coccidiosis is seldom occurs as an acute or severe clinically. Ulcerative enteritis can resemble necrotic enteritis clinically, but the intestinal lesions are usually focal and located in the ileum, cecaum and rectum. Gross lesions reveal extreme dehydration and darkening of the breast muscle. The liver is usually swollen and congested, but necrosis is rare. Lesions usually confined to the small intestine, primarily jejunum and ileum, it appears ballooned and friable and contains foul-smelling brown fluid. The mucosa is covered with a loose brownish diphtheritic membrane.

5.17.4 : Control and Prevention: Strict sanitation efforts to prevent coccidiosis, salmonellosis and other intestinal infections minimize the risk of necrotic enteritis. Drastic changes in feed should be avoided and feed and water should be monitored for contaminants that alter intestinal motility or devitalize intestinal mucosa. Probiotics and competitive exclusion strategies can be employed with greater success in minimizing the possibility of N.E. Bacitracin is the drug of choice for preventing and treating necrotic enteritis. Preventive levels range from 0.05 to 0.1 % in the feed while 0.02 to 0.05% is therapeutic; Penicillin, erythromycin and the tetracycline at 0.02% in the feed are also effective. In outbreaks however, clinical surveys demonstrate the causative bacteria resistance to lincomycin and bacitracin but sensitive to virginiamycin and penicillin.

## 5.18 Fowl Cholera

5.18.1 Definition: (Synonyms: Avian pasteurellosis, avian hemorrhagic septicemia and avian cholera) A contagious widely distributed disease that affects domesticated and wild birds. It usually occurs as a septicemia with sudden onset and high morbidity and mortality, but chronic and asymptomatic benign infections also occur. Clinical signs vary greatly depending upon the course of the disease. In acute cases, dead birds may be the first indication of disease. Fever, depression, anorexia, mucus discharge from the mouth, ruffled feathers; diarrhea and increased respiratory rate are usually seen. Pneumonia is common in turkeys. In chronic fowl cholera, signs and lesions are generally related to localized infections. Sternal bursae, wattles, joints, tendon sheaths, and footpads are often swollen because of accumulated fibrino-suppurative exudates. There may be exudative conjunctivitis and pharyngitis. Torticollis may result when the meninges, middle ear, or cranial bones are infected.

5.18.2 Epidemiology: *Pasteurella multocida*, the casual agent, is a small, gram-negative, non-motile rod which may exhibit polymorphism after repeated subculture. Causative strains represent a number of immuno-types that complicate prevention of the disease by the use of vaccines. The organism is susceptible to ordinary disinfectants, sunlight, drying and heat. Turkeys and waterfowl are more susceptible than young chickens. The disease has been reported sporadically or enzootically in most countries. Infection rate is believed to rise in winter. Chronically infected birds are thought to be the major source of infection. Free flying birds also transmit the pathogen. In spite of presence in most species of farm animals,

strains isolated from non-avian hosts generally do not produce fowl cholera. Incubation period varies according to the bird immunity. Chickens become more susceptible as they reached maturity.

5.18.3 Diagnosis: A presumptive diagnosis may be based on the observation of characteristics signs; a more definitive diagnosis should include isolation and identification of *P. multocida*. Hyperemia is usually evident in the vessels of the abdominal viscera. Petechial and ecchymotic hemorrhages are common, particularly in sub-epicardial and sub-serosal locations. Increased amounts of peritoneal and pericardial fluids are frequently observed. Liver usually contain multiple small necrotic foci and may be swollen. Localized infections are generally related to chronic disease. Sternal bursas, wattles, joints and footpads are often swollen because of accumulated fibrino suppurative exudates. Exudative conjunctivitis and pharyngitis may occur.

5.18.4 Prevention and Control: Anti microbial treatment options like sulfonamides are commonly used; early treatment and adequate dosages are important. Sensitivity testing often aids in drug selection. Sulfaquinoxaline sodium in feed or water usually controls mortality, as do sulfamethazine and sulfadimethoxine. Sulfas should be used with caution because of potential toxicity. High levels of tetracycline antibiotics in the feed (0.04%), drinking water, or administered parenterally may be useful. Penicillin is often effective for sulf-resistant infections. Good management practice. Rodent control must be effective in poultry houses. Polyvalent bacterins are generally effective. Adjuvant bacterins are widely used; autogenous bacterins are recommended when polyvalent bacterins are found to be ineffective.

Attenuated vaccines are also administered in drinking water and by wing web inoculation to chickens and turkey. They are efficient in producing immunity against spectrum of serotypes, however they are recommended for use only in healthy flock.

## 5.19 *Mycoplasma gallisepticum* Infection

5.19.1. Definition: (Synonyms: Chronic Respiratory Disease (CRD), infectious sinusitis, PPLO infection) *Mycoplasma gallisepticum* infection is commonly known as chronic respiratory disease (CRD) of chickens and infectious sinusitis of turkeys, characterized in chickens by respiratory rales, coughing, nasal discharge, and conjunctivitis and infra-orbital sinusitis in turkey. Clinical manifestations are usually

"Air sac disease" describes a severe air sacculitis that is the result of *Mycoplasma gallisepticum* or *Mycoplasma synoviae* infection complicated by a respiratory virus infection (e.g. IB or ND) and usually Escherichia coli.

5.19.2 Epidemiology: *M.gallisepticum* is one of the most economically significant pathogen of poultry. Many strains show marked virulence variability. It was first classified and differentiated from other avian mycoplasmas by serotyping and commonly was designated serotype A. Sizable economic losses result from condemnations or downgrading of

carcasses, reduced feed and egg production efficiency, and increased medication costs. Occurrence is worldwide. The organism infects a relatively narrow range of exclusively avian host species and is of no public health significance. Horizontal transmission occurs readily by direct or indirect contact. The upper respiratory tract and/or conjunctiva are portals of entry for the organism in aerosols or droplets, fomites, contaminated airborne dust especially in suboptimal biosecurity situations. Backyard flocks, multiple age commercial layer flocks and some free-ranging bird species may act as potential reservoirs of *M. gallisepticum*. Vertical transmission (*in ovo, transovarian*) is considered a major source of infection in some parts of the world. *M.gallisepticum* incubation period varies from 6—21 days. However, development of clinical signs following a known exposure, even in turkeys that are considered highly susceptible can be highly variable depending on *M.gallisepticum* strain virulence, complicating infections, and environmental and other stressors.

- 5.19.3 Diagnosis: The gold standard for *M.gallisepticum* diagnosis is isolation and/or identification of the organism. Swabs can be taken from the trachea or choanal cleft (palatine fissure) from 10—20 live birds for culture is sufficient to recover the organism during the acute stages. Flocks should be sampled prior to initiation of antimicrobial therapy. A positive serologic test, namely tube agglutination test and serum plate agglutination with history and signs typical of the *M.gallisepticum* diseases, allows a presumptive diagnosis. More recently, detection of *M.gallisepticum* using ELISA or specific DNA by PCR is becoming widely practiced. *M.gallisepticum* infections should be differentiated from other respiratory diseases as it may concurrently present. In chickens, care must be taken to differentiate *M.gallisepticum* infection from other common respiratory diseases like Newcastle disease, infectious bronchitis, *E.coli* infection, Infectious coryza & influenza.

Gross lesions in chickens consist primarily of catarrhal exudates in nasal and para-nasal passages, trachea, bronchi, and air sacs. Air sacs frequently contain caseous exudates, with pronounced thickening and turbidity due to concurrent *E.coli* infection. Some degree of pneumonia may be observed. In severe cases, there is the triad of airsacculitis, fibrinous or fibrino-purulent perihepatitis, and adhesive pericarditis resulting in high mortality and extensive condemnations at processing. Commercial layer chickens with *M.gallisepticum* associated kerato-conjunctivitis had marked edema in the facial subcutis and eyelids, with occasional corneal opacity. Conjunctivitis is characteristic. Oviducts distended with exudate (salpingitis) have been associated with decreased egg production.

- 5.19.4 Prevention and control: Several antibiotics including macrolides, Chlortetracycline, Oxytetracycline, erythromycin, tylosin and fluoroquinolones were used to combat *M.gallisepticum*, and effective treatment of secondary invaders.

However the organism shows resistance to penicillins or antibiotics, which act by inhibiting cell wall biosynthesis. Antimicrobials given in water or feed for 5-7 days may reduce the severity of clinical signs and lesions but do not eliminate it. Egg injection or dipping with a temperature or pressure differential have been used to introduce antimicrobials into hatching eggs to control *M.gallisepticum* in ovo transmission with variable degree of success. Strict biosecurity to avoid introduction of the organism is essential. Vaccination

may also be an option in some situations, employing both inactivated and living MG vaccines. It has been proofed that bacterins could protect broilers from air sacculitis or layers from reductions in egg production but with limited effects and minimal value in case of commercial egg layers with endemic infections, or in long-term control of infection on multiple-age production sites. Complete elimination of MG from all birds in an infected flock by mass antimicrobial therapy is an unrealistic expectation.

## 5.20 *Mycoplasma synoviae* Infection

- 5.20.1 Definition: (Synonyms: infectious synovitis) *Mycoplasma synoviae* infection most frequently occurs as a subclinical upper respiratory infection. It may cause air sac lesions when combined with ND, IB, or both. At other times, *Mycoplasma synoviae* becomes systemic and results in infectious synovitis, an acute to chronic infectious disease of chickens and turkeys, involving primarily the synovial membranes of joints and tendon sheaths producing an exudative synovitis, tendonitis, or bursitis. Main clinical sings include pale comb, lameness, and retarded growth as early manifestations.

- 5.20.2 Epidemiology: As the disease progresses, feathers become ruffled and the comb shrink and may become bluish red. Swellings usually occur around joints, and breast blisters are common. Hock joints and foot pads are principally involved, but in some birds most joints are affected; however, birds occasionally are found with a generalized infection but not with apparent swelling of the joints. Birds become listless, dehydrated, and emaciated. A greenish discoloration of droppings is frequently seen. Synovitis may persist for the life of the flock. In other instances, the acute phase is not noticed, and only a few chronically infected birds are seen in a flock. Chondrodystrophy was noted in the opposite leg of chickens inoculated via the foot pad.

This may have been due to increased weight bearing stress on the leg opposite the affected leg. Air sac infection may occur at any age but is most often observed as a cause of condemnation in broilers. Under field conditions, most air sac lesions resulting from *M. synoviae* infection occur in winter. Chickens, turkeys, and guinea fowl are the natural hosts of *M. synoviae*. Natural infection in chickens has been observed as early as 1 week, but acute infection is generally seen when chickens are 4—16 weeks old. Chronic infection follows the acute phase and may persist for the life of the flock. Infection of the upper respiratory tract is permanent. Morbidity ranges between 2 - 15% while mortality is around 1 - 10%. Lateral transmission occurs readily by direct contact. In many respects, the spread appears to be similar to that of *M. gallisepticum* except that it is more rapid ,usually 100% of the birds become infected, although none or only a few develop joint lesions.

Birds are infected for life and remain carriers and may shed at any time. Vertical transmission also plays a major role in spread of MS in chickens and turkeys. Infectious synovitis has been seen in 6 day old chicks, suggesting that a short incubation period in birds infected by egg transmission. Following contact exposure it is generally 11—21 days.

- 5.20.3 Diagnosis: A presumptive diagnosis may be made on the basis of pale comb, droopiness,

emaciation, leg weakness, breast blisters, enlarged foot pads or hock joints, spleenomegaly, and enlarged liver or kidneys. Positive diagnosis may be made by isolation and identification of *M. synoviae* from the upper respiratory tract in chronically infected birds. The fluorescent antibody technique using colony imprints or intact colonies may be used for the identification. ELISA may be commonly used and can effectively replace serum plate agglutination as the primary serologic test. PCR methods are comparable in sensitivity to isolation and identification. In early stages of the infectious synovitis form of the disease, chickens frequently have a viscous creamy to gray exudates involving synovial membranes of the tendon sheaths, joints, and keel bursa and hepatosplenomegaly. Kidneys are usually swollen, mottled, and pale. As the disease progresses, caseous exudates may be found involving tendon sheaths, joints, and extending into muscle and air sacs. In the respiratory form, airsacculitis may be present. *Staphylococcus aureus*, *Escherichia coli*, *pasteurellae*, and *salmonellae* may also be present as primary causes of synovitis. *M. gallisepticum* may also be a cause of breast blisters and joint lesions. Fibrosis of metatarsal extensor or digital flexor tendons and lymphocytic infiltration of the myocardium associated with the viral arthritis agent help to differentiate it from *M. synoviae*.

- 5.20.4 Prevention and Control: *M. synoviae* is egg transmitted, and the only effective method of control is to select chickens or turkeys from MS-free flocks. Effective biosecurity measures should be used to prevent introduction of the infection. Outbreaks of MS infection in broilers can often be traced to a specific breeder flock.

*M. synoviae* is susceptible in vitro to several antibiotics, including chlortetracycline, oxytetracycline, spectinomycin, spirofloxacin, tetracycline in feed, and tylosin. In contrast to *M. gallisepticum*, *M. synoviae* isolates appear to be resistant to erythromycin. Generally, suitable medication is of value in preventing airsacculitis or synovitis, but treatment of existing lesions is less effective. Antibiotic medication will not eliminate MS infection from the flock. An inactivated, oil emulsion bacterin is commercially available, but its role in the control is limited.

## Appendix – Tables (1-11)

Table (1) Gestation Periods, Body temperature and Pulse rate in animals

Pulse Rate	Temperature Celsius	Gestation Period (days)	Animal
35 – 45	35 – 38.6	406	Camel
40 – 80	37.8 – 39.2	280	Cattle
70 – 130	38.9 – 40	148	Sheep
70 – 130	38.6 – 40.2	151	Goat
35 – 40	37.5 – 38.5	337	Horse
60 – 120	37.5 – 38.6	62	Dog
110 – 130	37.8 – 39.2	62	Cat

Table (2) Respiratory diseases commonly found in small ruminants

Respiratory infectious diseases		
Viruses	Bacteria	Parasites
Herpes viruses, e.g. IBR (BHVI)	pasteurellosis	<i>Muellerius capillaris</i>
Respiratory syncytial virus	<i>Corynebacterium pyogenes</i>	<i>Dictyocaulus filaria</i>
Parainfluenza virus 3	<i>Staphylococcal spp. Haemophilus spp</i>	<i>Protostrongylus rufescens</i>
	<i>Klebsiella pneumoniae</i>	Hydatid cysts ( <i>Echinococcus granulosus</i> ).
Pulmonary adenomatosis	Tuberculosis	

Table (3) Infectious Agents of Diarrhea and Abdominal pain in small ruminants

Respiratory infectious diseases				
Diarrhea			Abdominal pain	
Birth – 4 weeks	4 – 12 weeks	Over 12 weeks	Diarrhea /enteritis	
Enterotoxigenic <i>E.coli</i> Salmonella spp. <i>Clostridium perfringens</i> type C <i>Clostridium perfringens</i> Type B	Parasitic gastroenteritis Coccidiosis <i>Clostridium perfringens</i> Type D Giardiasis	Parasitic gastroenteritis Coccidiosis <i>Cl.perfringens</i> type D Liver disease (Johne's disease)	Diarrhea /enteritis Abomasal bloat Coccidiosis Ruminal bloat Constipation Enterotoxaemia ( <i>Clostridium perfringens</i> type D) Urolithiasis Visceral cysticercosis	Diarrhea /enteritis Abomasal bloat Coccidiosis Ruminal bloat Constipation Enterotoxaemia ( <i>Clostridium perfringens</i> type D) Urolithiasis Visceral cysticercosis

Table (4) Diseases with major clinical signs in farm animals

Recumbency	Emaciation	Anorexia	Dehydration	Respiration increased depth, hyperpnoea	Dyspnoea
Abomasal impaction.	Actinobacillosis.	Abomasitis. Acidosis.	Abomasal dilataion.	Anthrax CBPP	Abomasal dilation.
Abomasal torsion.	Actinomycosis.	Alimentary neoplasia.	Abomasal impaction.	Congestive heart failure. Fever.	Actinomycosis.
Acidosis. Anaemia.	Amyloidosis.	Anthrax.	Abomasal ulceration	Lungworm infestation.	Anaphylaxis. Anthrax.
Arthropathy (prolonged periods).	BSE	Bacillary haemoglobinuria.	with perforation.	Hemorrhages.	Aspergillosis.
Babesiosis.	Contagious pyelonephritis.	Blackleg.	BVD	Pulmonary	Atypical interstitial pneumonia
Botulism (lateral). BSE	Degenerative joint disease.	Botulism.	Caecal torsion. Cereal overload.	Pulmonary congestion.	pneumonia Bacillary
Cereal overeating.	Acute cerebral anoxia.	Bovine malignant catarrh.	Diarrhea. Displaced abomasums. Enteritis.	Pulmonary emphysema.	Bovine malignant catarrh.
Choline deficiency (calves).	Enzootic bovine leukosis.	Bovine viral diarrhoea.	Pulmonary oedema.	Septicaemia.	Bronchitis Candida
Downer cow.	Fascioliasis.	Bronchitis. Colienteritis.	Heat stroke.	Thrombosis of vena	spp.pneumonia. CBPP
Endocarditis. Enzootic haematuria.	Haemonchosis. Johne's disease. liver abscess	Colisepticaemia. CBPP	Hyperthermia.		
Fat cow syndrome. FMD	Necrobacillosis.	Encephalitis. Enteric toxæmia (calves).	Johne's disease	cava.	Enzootic bovine
Haemorrhages.	Malnutrition.	Fascioliasis. FMD	Mucosal disease.	Tuberculosis	leukosis.
Hepatitis.	Oesophagostomiasis	Gas gangrene. IBR.	Oesophageal obstruction. Parasitic	Pneumonic pasteurllosis. BVD	FMD IBR Leptospirosis. Listeria
Hypomagnesaemia (lateral).	Ostertagiasis typell.	Hepatitis. Leptospirosis ,	gastroenteritis	Tick fever	infection. Lungworm infestation.
Listeria infection.	Parasitic gastroenteritis.	L. hardjo.	Pharyngeal paralysis.		Parasitic bronchitis.
Louping ill.	Pulmonary abscess.	Liver abscess. Malignant	Post-parturient		Pasteurellosis.
Osteomalacia.	Redwater fever.	oedema. Mucosal	haemoglobin- uria		Pharyngeal
Post-parturient haemoglobinuria.	Sarcosporidiosis.	disease.	Rinderpest.		obstruction.
Peritonitis, acute.	Sporadic bovine	Oesophagostomiasis	Shock.		Pulmonary neoplasia.
Ruminal tympany.	leukosis.	Peritonitis.	Viral diarrhea (Calves).		Pulmonary oedema.
Shock.	Chronic toxæmia.	Pulmonary abscess.	Toxaemia.		Rinderpest.
Tetanus. Transit tetany.	Tuberculosis.	Rabies.	Redwater fever.		Tuberculosis.
		Rinderpest.	Salmonellosis.		Toxoplasmosis
			Sarcosporidiosis.		
			Septicaemia		

Table (5) External parasites of small ruminants

Case	Agent	Distribution of lesions	Lesions	Diagnosis	Treatment
Lice infestation	<i>Damalinia caprae(biting)</i> <i>linognathus stenopsis (sucking)</i>	Head , neck, back	Hair loss broken hairs moth eaten coat	naked eye	Organophosphorus dips γ BHC bromocyclopyrethroid pourons
Chrioptic mange	<i>Chorioptes ovis</i> , <i>Chorioptes caprae</i>	Lower posterior limb Occ. Ventral abdomen, sternum	Crusting erythema	Skin scraping	γBHC bromocyclopyromectin
Demodectic mange	<i>Demodex caprae</i> <i>D. ovis</i>	Head neck and body	Hard nodules with yellow caesous material	Microscopy on expressed material skin biopsy	Rotenone
Pustular dermatitis	<i>Staphylococcus aureus</i>	Udder, teats ventral abdomen groin, body	Pustular scabs dry scaly coat	Culture	Local/ parental antibiotics or udder washes
Sarcoptic mange	<i>Sarcoptes scabiei</i> <i>S. ovis</i>	Head ears and body	Alopecia, crusting weight loss	Skin biopsy (skin scraping)	Ivomectin γBHC
Ringworm	<i>Trichophyton verrucosum</i> , <i>Microsporum canis</i> , <i>Trichophyton mentagrophytes</i>	Head body	Raised , circular crusty	Microscopy culture	Griseofulvin in feed topical prep
Psoroptic mange	<i>Psoroptes cuniculi</i> <i>P.ovis</i>	Ear head body	Head shaking	Skin scraping	γBHC

Table (6) Clinical findings associated with infectious diseases in farm animals

Severe Pyrexia(41-41.9)	Rapid heart beat Over100/min.	jaundice	Mucous membrane hemorrhages	Enlarged lymph nodes
Anthrax	Abomasal torsion.	Bacillary haemoglobinuria.	Anthrax	Actinobacillosis
Bovine malignant catarrh	Acidosis Anaemia	Endo-carditis.	Leptospirosis	Atypical mycobacterium infection.
Diphtheria(laryngeal)	Anthrax	Hepatitis.	Mycotoxicosis	Bovine malignant catarrh.
Gas gangrene	Babesiosis	Lepto-spirosis.	Pyrexia/pruritus/haemorrhagic syndrome.	Enzootic bovine leukosis.
IBR	Bacillary haemoglobinuria.	Lupinosis		Infections(generalized or localized).
Louping ill Malignant oedema Peritonitis, acute diffuse.	Blackleg	Photosensitization		Neoplasia(generalized or localized).
Pneumonia	Louping ill Malignant oedema Peritonitis, acute diffuse.	Post-patentient haemoglobinuria.		Septicaemia Sporadic bovine leukosis.
Rinderpest Septicemic	Leptospirosis			Tuberculosis(avain)
Salmonellosis.	Lungworm infestation.			Tuberculosis(bovine)
septicaemia	Parasitic gastroenteritis.			
	Acute peritonitis			
	Rinderpest			
	Salmonellosis.			
	Septicaemia (some cases).			

Table (7) Veterinary Chemicals (Disinfectants &amp; Insecticides)

1.	Soaps and detergents
2.	Oxidizing agents: a: Sodium hypochlorite b: calcium hypochlorite c: Virkon®
3.	Alkalis: do not use with Aluminium and similar alloys. a: Sodium hydroxide(caustic soda, NAOH) b: Sodium carbonate anhydrous(Na <sub>2</sub> CO <sub>3</sub> ) washing soda(Na <sub>2</sub> CO <sub>3</sub> .10H <sub>2</sub> O)
4.	Acids A: Hydrochloric acid B: Citric acid
5.	Aldehydes: A: Glutaraldehyde: glutaraldehyde is not too corrosive on metals but must not be used on humans or animals B: Formalin C: Formaldehyde gas: gaseous formaldehyde is dangerous and subject to error ; it should only be used by experience personnel and in controlled condition
6.	Insecticides: A: Organophosphates B: Synthetic pyrethroids C: Ivermectin D: Phostoxin

Table (8) Disinfectants and Precautional Aspects

Disinfectant	Health aspects	Environment problems and contra-indications
Hypochlorites	Toxic for eyes and skin	Strong bleach. Inhibited by high concentrations of organic matter. Corrosive for many metals
Virkon®	Reasonable care necessary	
Sodium hydroxide	Caustic for eyes and skin	Avoid contact with strong acids. Cannot be used on aluminum or similar alloys
Hydrochloric acid	Toxic for eyes skin and respiratory passages	Corrosive for concrete and many metals. Avoid contact with strong alkali
Glutaradehyde	Avoid eye and skin contact	
Formalin solution	Releases toxic gas; irritating for mucous membranes	
Formaldehyde gas	Very toxic for mucous membranes in concentrations down to 2 ppm.	Can not be used in presence of water, hypochlorites or chlorides. Cannot be released to atmosphere without neutralization. Corrosive for some metals

Table (9) Clostridium perfringens toxins

Types	Toxins		Action of major toxins	Diseases
	Major	Minor		
A	α	-	Lethal, necrotizing, hemolytic, leukocidal	1. yellow lamb 2. Gas gangrene 3. Necrotic enteritis in poultry
B	β	α ε	Lethal, necrotizing	1. Lamb dysentery 2. Hemorrhagic enterotoxaemia ET in lambs, calves, foals
C	β	α	Lethal, necrotizing	1. Struck of young adult sheep. 2. Hemorrhagic ET in lambs, calves, piglets
D	ε	α	Lethal, necrotizing, enhances permeability of gut and capillaries	1. ET -(pulpy kidney) of young feedlot sheep, young lush- pasture sheep, nursing lambs
E	l	α K	Lethal, necrotizing	ET of calves

Table (10) Intestinal helminthes of ruminants

Parasite	Cattle			Sheep		
	A	B	C	A	B	C
Haemonchus contortus				+		
Haemonchus placei	+					
Trichostrongylus axei	+			+		
Ostertagia ostertagi	+					
Ostertagia circumcincta				+		
Ostertagia trifurcata				+		
Trichostrongylus colubriformis		+			+	
Cooperia curticei					+	
Cooperia onchophora		+			+	
Cooperia mcmasteri		+			+	
Cooperia punctata		+			+	
Cooperia pectinata		+			+	
Neoascaris vitulorum		+			+	
Nematodirus helvetianus			+			
Nematodirus battus					+	
Nematodirus filicollis					+	
Nematodirus spathiger					+	
Bunostomum phlebotomum		+				
Bunostomum trigonocephalum					+	
Strongyloides papillosum					+	
Moniezia expansa					+	
Moniezia bedenini				+		
Oesophagostomum radiatum.					+	
Oesophagostomum venulosum.						+
Oesophagostomum columbianum						+
Chabertia ovina				+		+
Trichuris ovis					+	+
Trichuris globulosa						+

A: abomasums, B: small intestine, C: large intestine

Table (11) Diseases and causative pathogenic bacteria found in poultry

SPECIES AFFECTED	DISEASE	BACTERIA
chicken and turkey	Colibacillosis	<i>E. coli</i>
Chicken, turkey rarely other poultry	pullorum disease	<i>Salmonella pullorum</i>
Chicken, turkey rarely other poultry	fowl typhoid	<i>Salmonella gallinarum</i>
Chicks, poulets, and other young birds	Paratyphoid	<i>Salmonella enteritidis PT4, salmonella typhymurium,</i>
Ducks, turkey and other birds	Pasteurell anatipestifer infection	<i>Pasteurella (moraxella) anatipestifer</i>
Domestic and wild birds	Fowl cholera	<i>Pasteurella multocida</i>
Domestic, wild and caged birds	Yersiniosis	<i>Yersinia pseudotuberculosis</i>
Mainly turkey	Bordetellosis (turkey coryza)	<i>Bordetella avium</i>
Free-ranging and confinement-reared	Botulism	<i>Clostridium botulinum</i>
Chicken, quail, pheasant,	Ulcerative enteritis	<i>Clostridium colinum</i>
Domestic chicken	Necrotic enteritis	<i>Cl. perfringens types A or C</i>
Primarily broiler chicken	Gangrenous dermatitis	<i>Cl.septicum, C.perfringens type A, and staphylococcus aureus</i>
Domestic birds and other poultry	Arthritis and tenosynovitis, yolk sac infection spondylitis osteomyelitis,	<i>Staphylococcus aureus</i>
Chicken and wild birds	Acute septicemic and chronic infections	<i>Streptococcus zooepidemicus and S. faecalis</i>
Mainly turkey, ducks, geese, quail, and chicken are less susceptible	Erysipelas	<i>Erysipelothrix rhusiopathiae</i>
Primarily in domestic fowl	Avian tuberculosis	<i>Mycobacterium avium</i>
Broiler chicken and turkey	Campylobacteriosis	<i>Campylobacter jejuni, C. coli, and C. lardis</i>
Many avian species including poultry	Listeriosis	<i>Listeria monocytogenes</i>
Poultry chicken and turkey	Yolk sac infection	<i>Bacillus cereus</i>
Turkey and chickens	Chronic respiratory disease (CRD)of chicken and infectious sinusitis of turkey	<i>Mycoplasma gallisepticum</i>
Chicken and turkey	Air sac disease, exudative synovitis, tendonitis.	<i>Mycoplasma synoviae</i>
Cage, wild, and migratory Birds ,pigeons, pheasants ducks,	Chlamydiosis (psittacosis/ ornithosis)	<i>Chlamydia psittaci</i>
Primarily chicken	Infectious coryza	<i>Haemophilus paragallinarum</i>
Chicks and poulets	Mortality and excess losses in chicks and poulets	<i>Klebsiella</i>

**References****Books References****Abd El- Rahim, I. H. 2005**

*Infectious Diseases of Domestic Animals Part 1: Viral and Bacterial Diseases*  
1st Edition Publisher: Faculty of Veterinary Medicine, Assiut University

**Aitken I.D. 2007**

*Diseases of Sheep*  
4th Edition Publisher: Blackwell Science

**Al-Ani, F.K., 2004**

*Camel Management and Diseases*  
1st Edition, Jordan Publisher: Dar Ammar Book, ISBN-10: 9957445006

**Al-Juboori A.A. 2003**

*Camels: Diseases and Treatment*  
Dubai, UAE 1st Edition Publisher: OMRAIT ISBN 9948-03-059-1

**Andrews A.H. ; Blowey R.W. ; Boyd H. and Eddy R.G. 2004**

*Bovine Medicine, Diseases and Husbandry of Cattle*  
2nd Edition, Publisher: Blackwell Science

**Kahn C. M. et al 2005**

*The Merck Veterinary Manual*  
USA 9th Edition (50th Anniversary Edition) Publisher: Merck & CO.INC Whitehouse Station N.J. in educational partnership with Merial Limited

**Radostits O.M. et al 2008**

*Veterinary Medicine*  
(A textbook of the diseases of cattle, horses, sheep, pigs, and goats)  
Edinburgh 10th Edition Publisher: Saunders Elsevier

**Saif Y.M. 2003**

*Diseases of Poultry*  
11th Edition Publisher: Blackwell Science

**Websites References**

FAO: Animal health manual on procedures for disease eradication by stamping out (ISSN-1020-5187)  
.Rome 2001  
<http://www.fao.org/docrep/004/Y0660E/Y0660E00.HTM>

FAO: Manual for Primary Animal Health Workers  
<http://www.fao.org/docrep/t0690e/t0690e00.htm>

International Livestock Research Institute publications  
<http://www.ilri.org/>

OIE Publications  
[http://www.oie.int/eng/publicat/en\\_numerosrt.htm](http://www.oie.int/eng/publicat/en_numerosrt.htm)

USDA Animal Diseases Publications  
[http://www.aphis.usda.gov/animal\\_health/animal\\_diseases/](http://www.aphis.usda.gov/animal_health/animal_diseases/)