INTRODUCTION

Anywhere on the earth, all the creatures are surrounded by many microbes. Therefore we can say that animals and humans are swimming in a sea of microbes. Some of these microbes are harmful and capable of causing many pathological phenomena. However, the wisdom of God that he gave them the secret of life or the license to survive, which is called the immune system. At the same time, God give these microbes many strategies, which ensure their survival and maintenance to emerge and reemerge. Around these facts let us swim in the chapters of this book.

Disease is a general term for conditions resulting from changes in the physiology, immunology or anatomy of an animal that impact, interrupt or modify the performance of the vital functions. These conditions can be infectious or non-infectious.

Infectious disease

Infectious disease is the result of the invasion of a host by specific infectious agent that creates a disturbance leading to the development of signs of illness. Infectious agents include bacteria, fungi, viruses, parasites, and prions.

- Bacteria are single cell microorganisms that do not require living cells to multiply. They can reproduce and persist in the environment or an animal's body. Pathogenic bacteria often produce toxins, e.g. *Mannheima haemolytica* causes pneumonia.
- Fungi are eukaryotic organisms that reproduce primarily by forming spores. Most fungi are multicellular, although some, such as yeasts, are unicellular. Examples of diseases caused by fungi are Ringworm and Histoplasmosis.
- Viruses are infectious pathogens that are smaller than bacteria and only reproduce inside a cell. As viruses reproduce, they destroy cells causing the specific symptoms of the disease. Unlike bacteria, viruses do not produce toxins and antimicrobials do not destroy them.
- Parasites live within or upon another living organism at whose expense they obtain some advantages. Intestinal and lung worms, ticks, warbles, mange mites, lice and coccidia are parasites.
- Prions are aberrant modified infectious proteins that do not have any genes. Prions are the cause of a group of diseases known as transmissible spongiform encephalopathies **(TSEs)** or **Prion diseases** including Bovine spongiform encephalopathy of cattle (BSE) and Scrapie of sheep and goats. There is no treatment in live animals at this time for prions.

Infection and Infectious disease

The term **infection** is derived from the Latin word (inficere) that means **(to put into)**. "Infection" refers to the process in which living agents enter animal's body with colonization of the host's cells, tissues, or body cavities without clinical or subclinical disease.

When such agents create disturbances in their hosts leading to development of signs of illness (clinical manifestation), infectious disease is said to have occurred.

- Infectious disease results from the interplay between pathogens and the defenses of the hosts they infect. The appearance and severity of disease resulting from any pathogen depends upon the ability of that pathogen to damage the host as well as the ability of the host to resist the pathogen. Infectious disease can arise if the host's protective immune mechanisms are compromised and the organism inflicts damage on the host.
- Pathogens can cause tissue damage by releasing a variety of toxins or destructive enzymes. For example, *Clostridium tetani* releases a neurotoxin, which causes spasmodic tonic contractions of the voluntary muscles.
- Not all infectious agents cause disease in all hosts, for example, Rinderpest virus causes disease in cattle and buffaloes, while Foot and mouth disease virus causes disease in cattle, buffaloes, sheep, goats, and pigs.
- Some infectious agents cause mild disease, while others are highly virulent, for example, the prion causing "mad cow disease" kills almost all animals that are infected.
- Persistent infections occur because the body is unable to clear the organism after the initial infection. Persistent infections are characterized by the continual presence of the infectious agent. Some viruses can maintain a persistent infection and never leave the body, e.g. Bovine virus diarrhea virus (BVDV).
- Often some pathogens cause latent infection with occasional recurrent relapses of active infection, a typical example is the herpes virus causing Infectious Bovine Rhinotracheitis (IBR) which tends to hide in nerves and become reactivated when specific circumstances arise.
- In some infections, the resistance of the host is overwhelmed so quickly that the organism multiplies in all parts of the body with rapid death. Such infections are known as **acute** or **peracute** infection. In peracute infections possibly few or no infecting organisms escape from the host e.g. peracute salmonella infection and Anthrax. Generally, in peracute fatal diseases, the causative

organisms are eliminated in small numbers or not at all.

- On the other hand, when the resistance of the body is unable to prevent the multiplication of the infectious agent but the infection does not become extensive and the agent persists, such infection is called **chronic** infection. In chronic infection, the host usually eliminates large numbers of the infectious agents, e.g. Brucellosis, chronic Salmonellosis, and chronic Fasciolosis.
- An important example of interaction between infectious agents is the mixed infection, that is, an infection with more than one type of agents. Two categories of infectious diseases can be identified; diseases in which clinical signs can be reproduced by single agent independently and diseases in which two or more microbial agents are necessary to induce disease. For example *E. coli*, rotaviruses and Cryptosporidium spp., all of which can induce diarrhea. Another example is the mixed infections caused by Mannheimia (Pasteurella) spp., bovine respiratory syncytial virus, parainfluenza-3 virus, and bovine virus diarrhea.

Contagious and non-contagious diseases

Diseases caused by specific infectious agents may be classified into highly contagious, slightly contagious, and non-contagious diseases (infectious diseases).

A contagious disease is one that can be transmitted from one animal to another by direct or indirect contact e.g. Rinderpest, Foot and mouth disease and Brucellosis, on the other hand **non-contagious (infectious)** diseases are transmitted by indirect means e.g. Fasciolosis, Rift valley fever and Blue tongue. Therefore, all contagious diseases are infectious but infectious diseases are not necessarily contagious.

The contagiousness of a disease depends on two main factors:

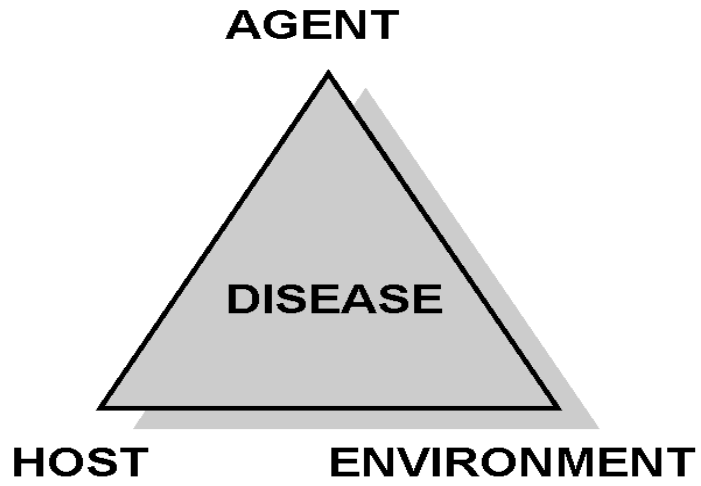
a. The method of discharging of the infectious agent.

b. The chance that the infectious agent reaches other susceptible animals.

- **Communicable Disease:** A disease that occurs due to transmission of an infectious agent from a diseased or infected animal or reservoir to a susceptible animal either directly or indirectly through a vector or the environment.
- **Virulence factors:** Virulence refers to the disease producing power of an organism. Pathogenic microorganisms vary in their virulence. Virulence factors are specific attributes of an organism

that are essential to cause disease. They include adhesions, toxins, anti-phagocytic factors, an ability to survive and multiply in phagocytes, and an ability to sequester iron, etc.

Studying of any infectious disease requires the application of four models, namely: the "Epidemiological triangle", the "Chain of infection", the "Natural history of the disease" as well as the "Infection cycle". This will enable us to deal properly and control the epizootic under study.

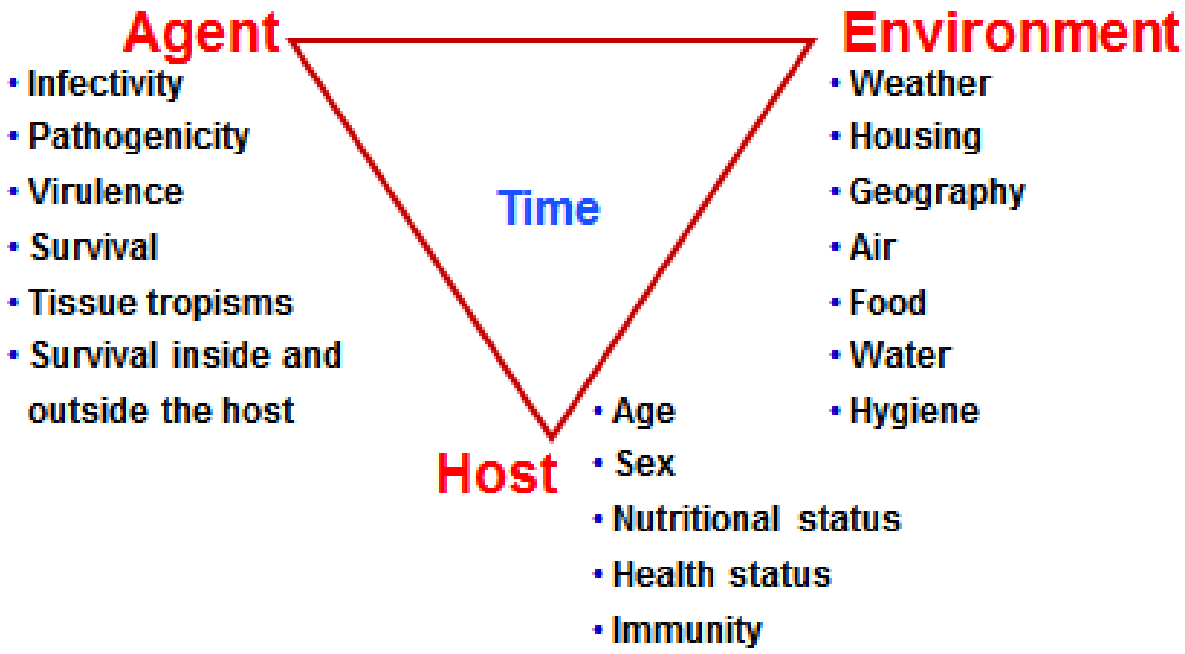


Disease is the result of interaction of three forces within a dynamic system consisting of:

1- Agent of infection 2- Host 3- Environment

Health and disease dynamics in a population are affected by host, agent and environmental factors.

For a disease to be introduced and maintained in a population, the host has to be **susceptible** to colonization and infection, the agent has to be **infective** for the host, and the environment in which host and agent are interacting has to be **conducive** to provide effective contact between host and agent for colonization and infection of the host to take place.



Epidemiologic Triad-Related Concepts

Three terms are used to describe an infectious disease according to the various outcomes that may occur after exposure of the host to its causative agent.

Infectivity: (ability to infect). It refers to the proportion of exposed animals that become infected.
(Number of infected animals / numbers of susceptible animals) x 100

Pathogenicity: (ability to cause disease). It refers to the proportion of infected animals that develop clinical disease.
(Number of diseased animals / numbers of infected animals) x100

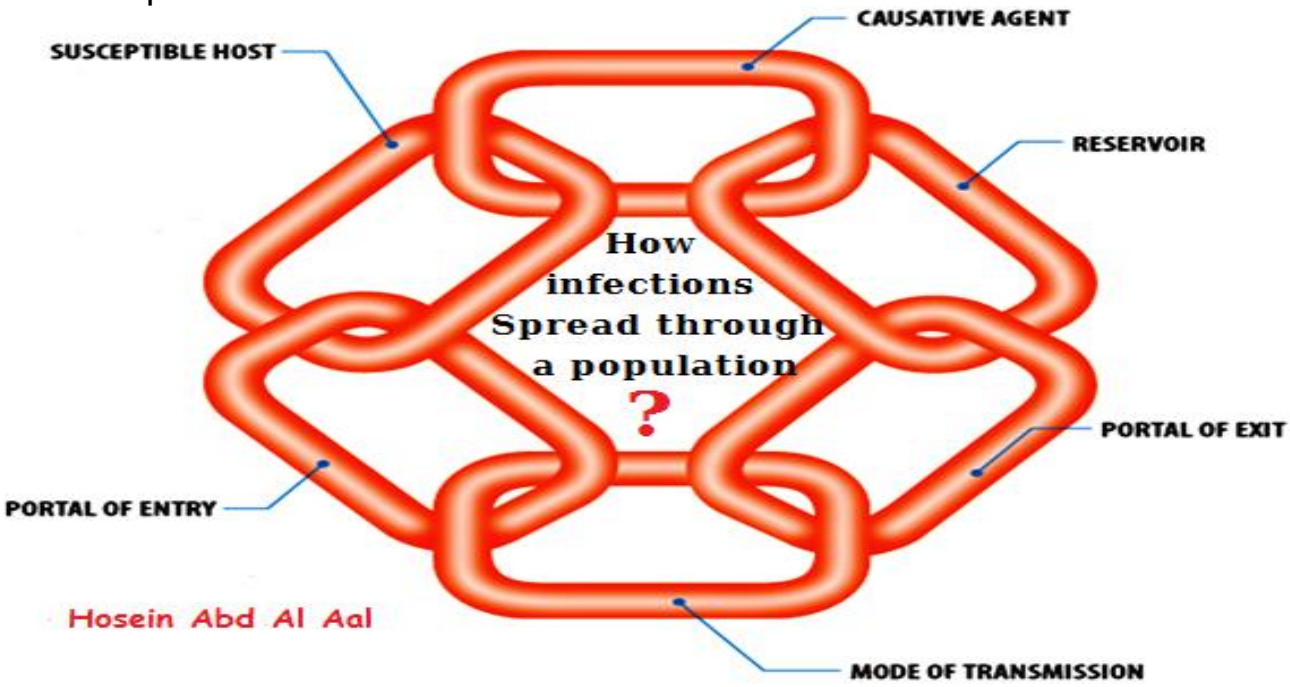
Virulence: (ability to cause death), a measure of the severity of a disease. It refers to the proportion of animals with clinical disease that become severely ill or die.
(Number of deaths and animals with serious disease / number of diseased animals) x 100

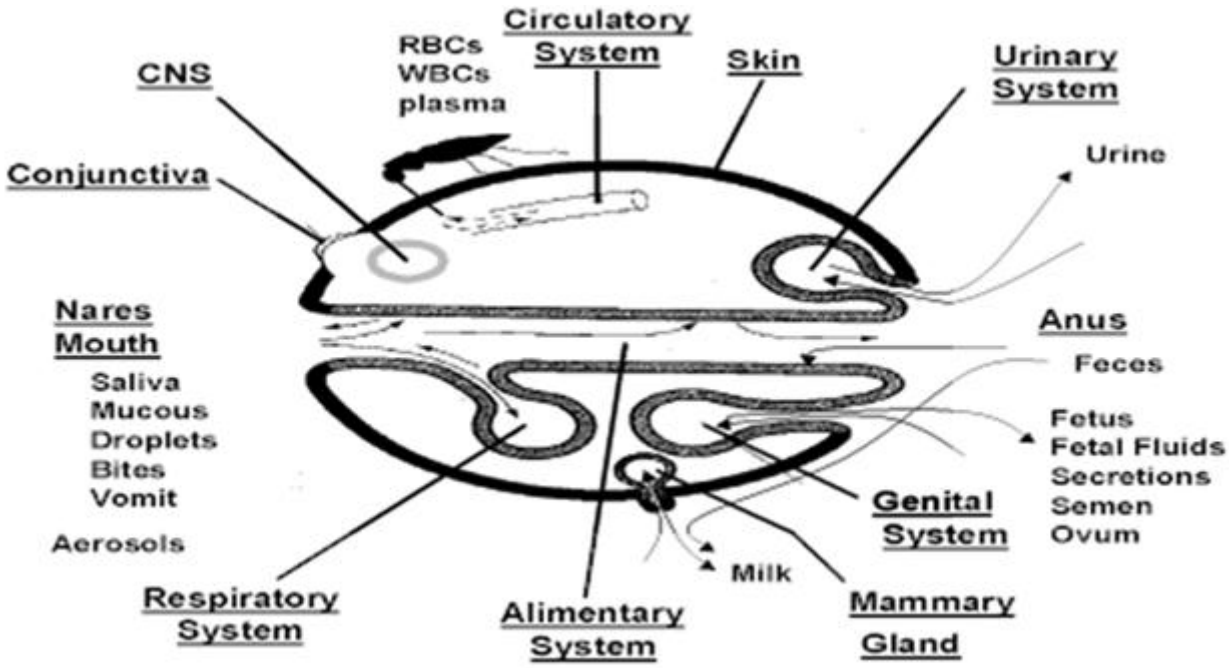
Model (2): The chain of infection

For an infection process and a disease to be occurred in an individual, a process involving six related components must occur. This process has been referred to as the “Chain of Infection.” It is a model that describes the way how infections spread through a population.

The six steps or "links" in the chain are:

- Etiologic agent (infectious agent)
- Reservoir
- Portal of Exit
- Mode of Transmission
- Portal of Entry
- Susceptible Host





Portals of exit and entry

Model (3): Natural history and spectrum of an infectious disease "Time line of infection"

The natural history of an infectious disease refers to the progress of a disease process in an individual over time. The natural history begins with the appropriate exposure to a pathogen. Usually, a period of subclinical or inapparent pathologic changes follows exposure, ending with the onset of symptoms. This period is usually called the **incubation period**. Even for a single disease, the characteristic incubation period has a range. The onset of symptoms marks the transition from subclinical to clinical disease. In some animals, however, the disease process may never progress to clinically apparent illness. In others, the disease process may result in a wide spectrum of clinical illness, ranging from mild to severe or fatal. This range is called the "spectrum of disease". Most diseases have a characteristic natural history, although the time frame and specific manifestations of disease may vary from individual to individual

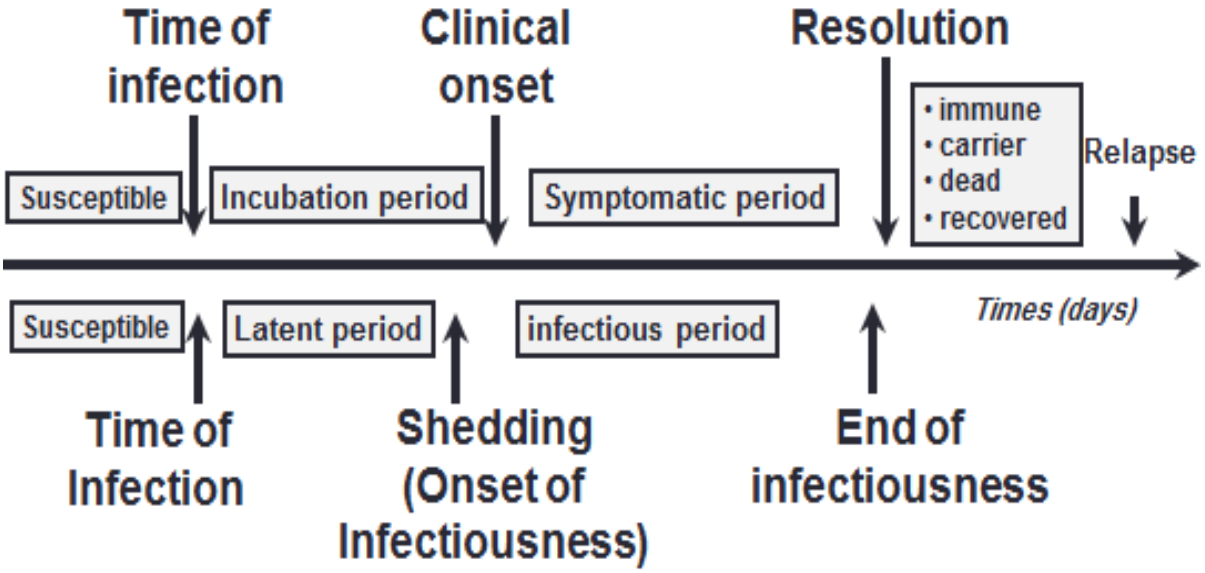


Figure (5): Timeline of infection

Model (4): Infectious Cycle

For the pathogen to cause disease, pathogen must be able to:

a. Enter the host body,
b. Adhere to specific host cells,
c. Invade and colonize host tissues (Targets) and inflict damage on

those tissues.

Understanding the infectious cycle for each infectious disease is critical in order to identify accessible targets for control strategies.

Example 1: Brucella infection:

Brucella organisms need to cross mucosal barriers of the digestive, respiratory or genital tract to reach its target cells, where it undergoes phagocytosis by resident macrophages and DCs, resulting in dissemination of the organism to lymphoid and reproductive organs.

Example 2: Foot and mouth disease.

FMDV initially spread from the pharyngeal region (respiratory tract). Primary replication occurs in epithelial cells of the pharyngeal mucosa-associated lymphoid tissue crypts and subsequent widespread replication in pneumocytes in the lungs, which coincides with viremia resulting in dissemination of the organism to oral mucous membranes, feet, teats, mammary glands skin and cardiac muscle.

Infectious diseases of newborns

Epizootiology:

Infectious diseases of newborn calves represent a major cause of economic loss to cattle industry due to the high mortality and morbidity.

Health of the newborn animal is a matter of balance involving the pathogens, immune system of the newborn animal, environment and management system. When there is an infectious disease, in newborn calves we should not ask what organisms are involved but we should ask:

What is the wrong with the system?
What has upset the balance?
What has upset the equilibrium in favor of the pathogen?

Exposure to infectious agents is not a sufficient cause for the development of diseases in calves. The difference between health and disease is often a delicate balance that weighs calf and environmental factors against the pathogens to which the calf will be exposed.

Newborn calf at birth faces three important problems, which should be kept in consideration when we are going to control the diseases of newborn calves, these are:

1. The agammaglobulinemia.
2. The need for protection against extremes in ambient temperature.
3. The contamination of the environment and infection by microorganisms within minutes after birth.

In the last few years, due to shortage of meat and the need for animal proteins, most of developing countries have created what is called **calf-open units** for increasing meat production as a commercial operation. Such open units collect calves from different sources at an early age. Under such practice, calves are taken from their dams during the first few days of life and transported to new areas in a strange microbial environment in company with other calves from other sources. In addition, they are reared on substitute diet. Newborn calves that reared under such conditions are exposed to many pathogenic organisms to which they may have little or no specific immunity.

Management of newborn calves plays an important role in the process of buildup of infection and it has a great influence on two main factors:

1. The exposure of calves to infection

2. The ability of calves to modulate the infection.

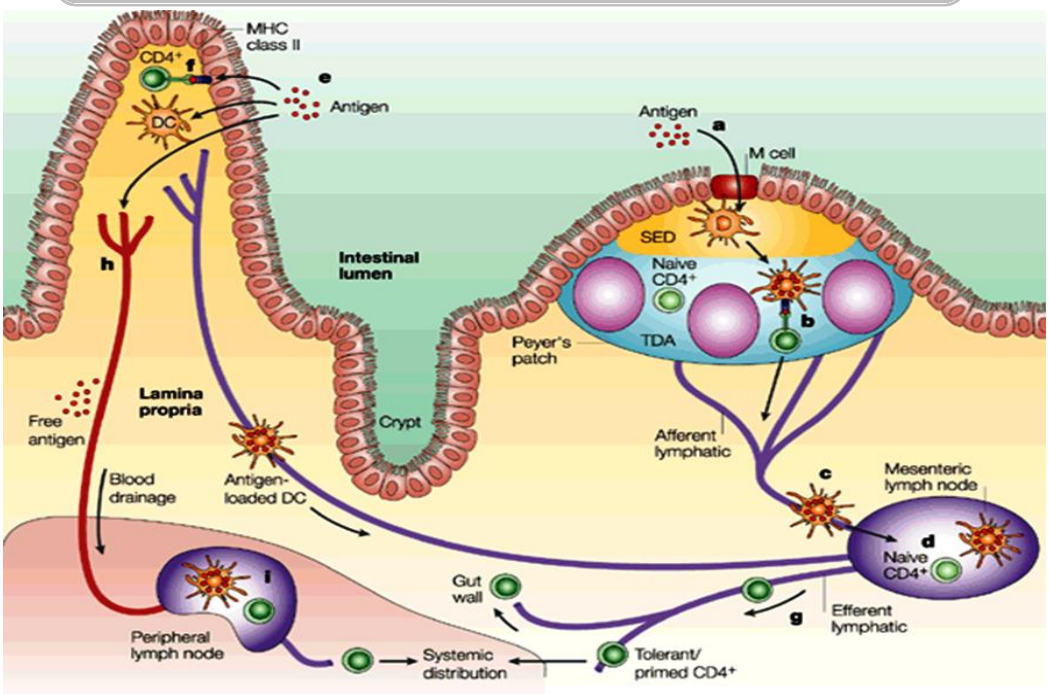
To ensure a healthy calf, the aim is to minimize the calf's exposure to disease, and maximize its defense against disease.

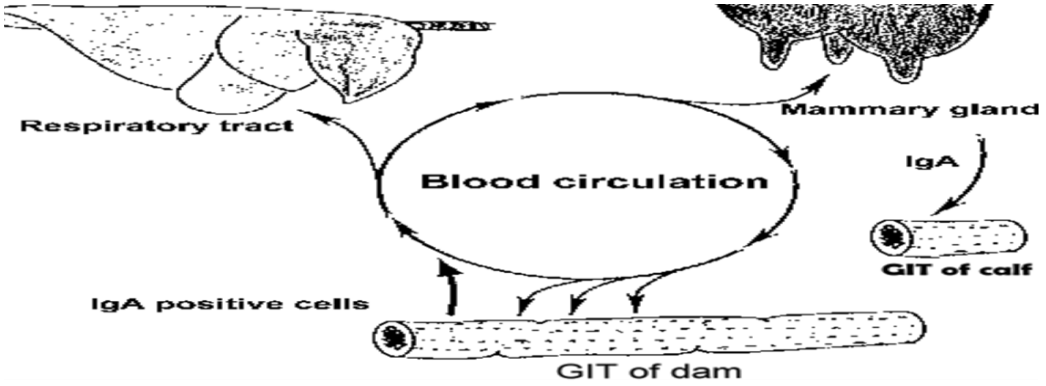
Buildup of infection in newborn calves' units is a complex process and it is unwise to follow a single line of approach.

Disease problems of newborn calves are **multifactoria**l in nature and it seems to be a result of interaction between different factors. In this chapter we shall deal with the important aspects or factors, which may contribute to the buildup of infection.
These include:

I. Immunological factor.
II. Nutritional factor.
III. Environmental factor.
IV. Management factor.
V. Microbial factor.

Immunity of the gastrointestinal tract





Immunity of the respiratory tract

1. Tonsils and other lymphoid tissues in the form of nodules in the wall of bronchi.
2. Lymphocytes distributed diffusely throughout the lungs.
3. IgA, which prevent the adherence of antigen particles.
4. IgG in acute infection.
5. IgE (mediated hypersensitivity).
6. Respiratory mucociliary clearance mechanism plays an important role in protection of the respiratory tract against infection. Mucous traps and transports inhaled particles to pharynx where they are swallowed and also it keeps the underlying mucosa hydrated. Mucous contains antibodies especially IgA which together with lactoferrin and lysozyme provide immunological defense.

II. **Nutritional factor**

a. Nutrition of the dam

The size and vigor of the newborn calf is influenced by the feeding of the dam before calving. The birth of small weak calves is a prominent predisposition to infection.

Vitamin A is an important factor for the protection mechanism of mucous membranes of the body. Vitamin A in the alcohol form as it occurs in the blood of the cow from the breakdown of carotene does not pass the placental barrier, but in the ester form, as it occurs in cod liver oil is transmissible. Therefore, the high green food intake does not increase vitamin A storage in the fetus but it increases its content in the colostrum.

In the fetal stage (the last 6-8weeks of pregnancy) there is a flow of sugar to the liver and muscles of the fetus where it is stored as glycogen, which represents the source of energy to the calf immediately after birth to be able to exert muscular strength to obtain its milk supply.

Poor feeding of dams during this stage results in low store of glycogen in fetus. High mortalities in the first few days of life are attributed to that the dams were insufficiently fed during the fetal stage.

b- Nutrition of the calf

The newborn calf should receive colostrum for several reasons:

1. It supplies the calf with immunoglobulins Igs and many of the immune cells (B cells, CD cells, macrophages and neutrophils). Igs are absorbed into the circulation only during the first 24 hours of life. The Igs found in the colostrum are IgG1, IgG2, IgM and IgA. IgG1 predominates and accounts for 75%-85% of total Igs; IgG2, IgM and IgA each account for 3-7% of the total Igs. There are two sources of colostral Igs, transportation from the systemic circulation into the udder and local synthesis by cells in the mammary gland. The total amount of Igs available to a calf is determined by the concentrations of Igs in colostrum and the volume of colostrum produced. The colostrum contains specific antibodies for the antigens to which the dam has been exposed.
2. Colostrum also contains non-specific antimicrobial factors as lactoperoxidase (prevent the attachment of *E. coli* possessing K88 and K99), lysozyme, lactoferrin, and interferon.
3. Colostrum supplies calves with nutrients and vitamins A, D, E and B.
4. The high-energy content of colostrum is necessary for the calf's energy requirement during the first few days of life. The newborn calf is wet with amniotic fluid that requires body heat for its evaporation.

Feeding a first-milking colostrum volume equivalent to 5% of birth weight (50 ml/kg bw) within one hour of birth resulted in relatively good immunity levels. The immunoglobulin level of second-milking colostrum is only half that of first-milking colostrum.

Newborn calves left with their dams ingest about 7-8 kg colostrum on the first day of life, increasing to 10-12 kg by the fourth day.

III. Environmental factor

1. Calves should be born in an environment which is clean and dry. Calving place should be prepared in advance for parturition.
2. Cold weather has an adverse effect on body insulation; in addition, the newborn calf is wet with amniotic fluid that requires body heat for evaporation.
3. Hot weather also has an adverse effect on young calves. Newborn

calve produce more sweat per Kg of body weight than does adult cattle; this may lead to dehydration and thus greatly reduces the calf's ability to cool itself by evaporation heat loss.

4. Calves should be kept in clean, disinfected, warm and dry pens and must be protected from draughts and extremes of temperature. They must be provided with clean dry bedding (straw), there must be sufficient ventilation, sufficient space and adequate light. The optimal temperature ranges from 20-24 ^{o}C and the preferred relative humidity is 70%. The floor of the calf pen should be hard and impervious.
5. Noxious gases, dusts, and molds in the air put calves at significant risk for developing pneumonia.

IV. Management factor

The practice of collecting calves from multiple sources and transporting them from farms to sales yard during the first weeks of life especially during cold weather contribute to the high mortality caused by pneumonia and enteritis.

1. Overcrowding and keeping calves of different sizes and ages together are responsible for spreading of several diseases among calves.
2. Increased level of exposure occurs when calves remain in the calving area and have continued contact with adult cattle.
3. It was observed that mortality is higher among calves of the first calf heifers due to the poor mothering behaviors. Such dams may not lick and stimulate the calf to get up and nurse; therefore, secretions should be removed from the nostrils and skin of the newborn calves. Calves should be also hanged from the hind limbs downward to stimulate the respiration.
4. In case of death of the dam, calf should be given colostrum from other dam or supplied with stored frozen colostrum using plastic bottle.
5. The newborn calf may be unable to stand due to decreased vigor as a result of lack of glycogen storage. Such calf should be injected as soon as possible with 5% dextrose.
6. Some calves may be unable to suckle their dams due to edematous tongue because of prolonged parturition, also in case of physiological edema of the udder; it is difficult for the calf to suckle its mother. Such calves should receive colostrum by a plastic bottle.
7. Some mothers may refuse the calf to suckle due to injuries or fissures on the teat skin, which should be observed earlier and treated.

8. Umbilicus of newborn calf should be touched with tincture iodine daily to prevent inflammation of the navel.
9. Procedures such as castrations, dehorning, weaning, and movement need to be considered as stresses and all have the potential to decrease immune system function temporarily.

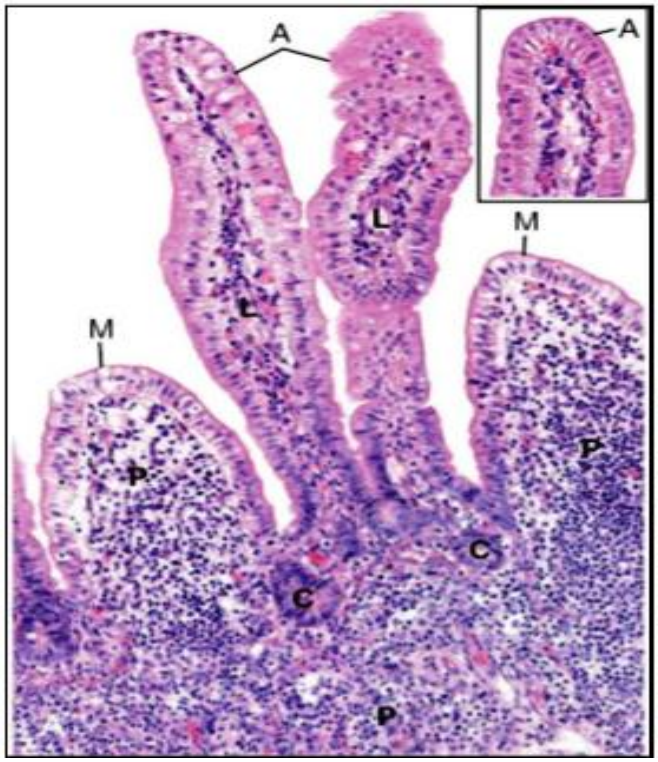
In conclusion, to ensure a healthy calf, the aim is to minimize the calf's exposure to disease, and maximize its defense against disease.

- Minimizing a calf's exposure to disease necessitates providing a clean, disease-free environment.
- Maximizing a calf's defense against disease requires adequate nutrition of the pregnant cow, vaccination of cows for control of pathogens, ensuring that each calf receives sufficient colostrum immediately after calving.

V- Microbial factor

1. Enteritis

Diarrhea in new born calves particularly under 30 days of age is considered as one of the most important disease complexes due to the significant economic loss. This problem continues to assume major importance due to the intensified livestock production.



- A: Absorptive cells,
- C: undifferentiated crypt cells,
- M: M cells,

- P: peyer's patch

Infectious diseases causing diarrhea in newborn calves and lambs

Colibacillosis

It is a contagious disease of newborn calves. The clinical syndrome varies according to the type of *E. coli* involved and the immune status of the newborn calf from collapse, sudden death, septicemic disease to enteric disease.

Etiology

German pediatrician and bacteriologist Theodor Escherich discovered *E. coli* in 1885.Now it is classified as part of the Enterobacteriaceae family of gamma-proteobacteria.

The disease is caused by Pathogenic serotypes of *E. coli.* They are Gram-negative common environmental bacteria. Some serotypes cause diarrhea and others cause septicemia.

The common enteropathogenic *E. coli* include:

- Enterotoxogenic *E. coli* (ETEC): They are not invasive and have the ability to adhere to mucosal surface, colonize and produce enterotoxins.
- Enterohemorrhagic *E. coli* (EHEC): They attach to the colon and distal small intestine.
- Attaching and effacing *E. coli* (AEEC): They produce attaching and effacing lesions producing a hemorrhagic colitis.
- Some of the AEEC are known as verocytotoxic *E. coli* (VTEC) because they produce verotoxins.
- Necrotoxogenic *E. coli* (NTEC): They produce cytotoxic necrotizing factor I or 2 (CNF1 or CNF 2).
- Some of the enterohemorrhagic *E.coli* found in animals are pathogenic for humans and produce a verotoxins such as *E. coli* O157 H 7.

Pathogenic *E. coli* strains fall into two categories: intestinal pathotypes and extraintestinal pathotypes.

- Intestinal pathotypes cause diarrhea caused by different *E. coli* pathotypes such as enterotoxinogenic "ETEC", *e*ntero*p*athogenic "EPEC" or *e*ntero*h*emorrhagic "EHEC".
- Extraintestinal pathotypes are another important group of pathogenic *E. coli* causing a diversity of infections in both humans and animals including urinary tract infections, meningitis and

septicemia. Uropathogenic *E. coli* "UPEC" are able to colonize the urinary tract and cause cystitis and pyelonephritis. Indeed, extraintestinal PEC are widespread in animal reservoirs. In cattle, they are also responsible for urinary tract infection, but more importantly, an important cause of bovine mastitis.

Epizootiology

Geographical distribution

It is a worldwide disease.

Susceptibility

The disease is most common in calves under three days of age. It may occur as early as 12-18 hours after birth and sometimes occurs in calves up to several days when there is a mixed infection with viral enteropathogens. Enterotoxogenic *E. coli* affects calves mainly during the first three days of life.

Lambs, foals, and piglets are also affected.

Sources of infection

These include:

1. The primary source of infection is the feces of diseased calves.
2. Recovered calves can continue to shed the bacteria for several months.
3. Contaminated litters and water.
4. Milk from *E. coli* infected udder.

Transmission

Infection occurs through ingestion. It may also occur through the umbilicus or the nasopharyngeal mucosa.

Colibacillosis is a complex disease in which several different risk factors interact with certain enteropathogens to induce the disease.

Pathogenesis

The pathogenesis of colibacillosis depends on the immune status of the calf and the virulence attributes of *E. coli* strain.

1- Septicemic colibacillosis

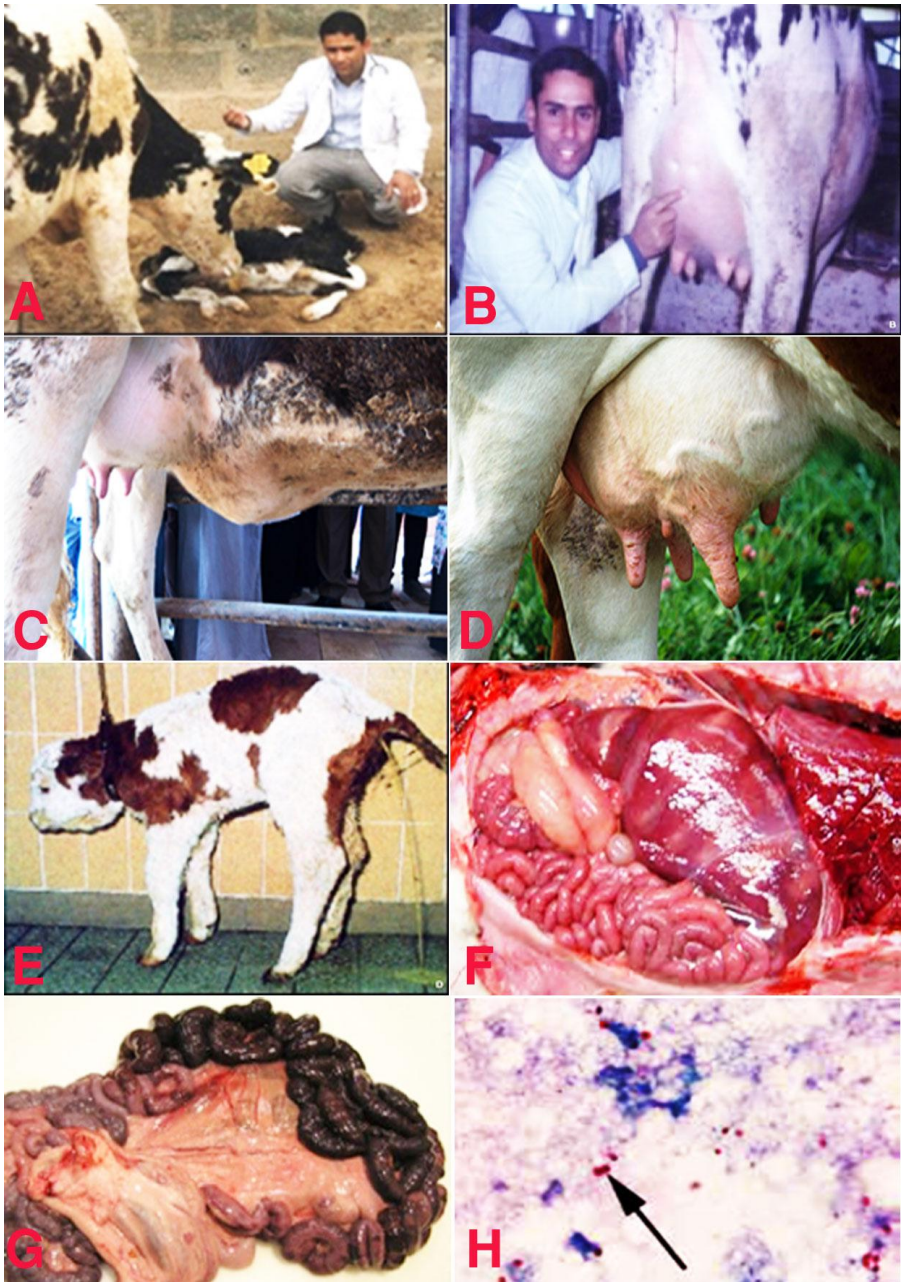
This occurs due to invasive strains of *E.coli*, which invade the circulation and tissues via the intestinal lumen, nasopharyngeal mucosa, tonsillar crypts or the umbilical vessels. The clinical findings and lesions in septicemic colibacillosis are due to the effects of endotoxins that cause shock. Calves, which are deficient in colostral immunoglobulins, are highly susceptible to septicemia. Animals, which recover from septicemia, may develop lesions due to localization in other organs causing meningitis, polyarthritis, panophthalmia or pneumonia (post septicemic localization).

2- Enterotoxogenic colibacillosis

In this form the bacterial fimbriae (K99 (F5) or F41 fimbrial antigens) attach to specific receptor sites on villous epithelial cells, then multiply and form microcolonies that cover the surface of the villi. The production of enterotoxins causes deregulation of the ion pump mechanism where there is increased secretion of chloride by the cells and decreased absorption of sodium and loss of HCO3 in feces. This results in net secretion of fluid *and* electrolytes from the systemic circulation into the lumen of the gut resulting in varying degrees of diarrhea (a non-inflammatory secretory diarrhea), dehydration, electrolyte imbalance, acidosis and hyperkalemia. Enterotoxins stimulate mucosal adenyl cyclase activity leading to an increased cyclic AMP, which increases intestinal fluid secretion.

Attaching and effacing *E.coli* do not produce enterotoxins but adhere to the surface of the enterocytes of the large intestine causing diarrhea, dysentery and passage of bright red blood in the diarrheic feces.

Enteropathogenic *E.coli* infection in calves older than three days will in most cases be associated with a virus infection.



Clinical signs

A- Coliform septicemia

It is a peracute form with a course ranging from 24-96 hours.

1. The body temperature may be high initially but it falls rapidly to subnormal levels.
2. Calves suffer from weakness, depression and recumbency followed by death.
3. In calves that survived the septicemic state; post-septicemic localization may occur in about one week. This includes polyarthritis, meningitis, panophthalmitis or pneumonia.

B- Enterotoxogenic colibacillosis

I- Severe form:

1. Calves show pale mucosa, cold skin, and collapse of the

superficial veins.
2. Diarrhea is usually not evident but the abdomen may be slightly distended.
3. Calves commonly die 2-6 hours after the onset of signs

II- Mild common form (enteric colibacillosis):

1. Calves show profuse watery diarrhea usually of pale yellow to white color and sometimes streaked with blood and of very foul smelling.
2. The tail and perineum are soiled with feces.
3. Temperature is usually normal in the initial stages but becomes subnormal later on.
4. Weakness, dehydration, and death within 3- 5 days.

NB:
5-6% dehydration: no clinical signs.
6-8% dehydration: sunken eyes, skin tenting for 3-5 seconds, dry mouth.
8-10% dehydration: loss of body weight, more distinct sunken eyes, skin tenting for 8-10 seconds, dry mm, increased pulse.
10-14% dehydration: comatose, cool extremities, poor peripheral pulse.

Postmortem lesions

1. **Coliform septicemia**: There may be no gross lesions but sometimes subserosal and submucosal hemorrhages may be present.
2. **Enteric Colibacillosis**:
   a. The carcass appears dehydrated.
   b. The abomasal mucosa may contain numerous small hemorrhages.
   c. Edema of the mesenteric lymph nodes.
   d. Mild atrophy or even fusion of jejunal and ileal villi occurs.
3. In calves affected with *AEEC* there are pseudo-membranous ileitis and muco-hemorrhagic colitis.

Diagnosis

1. Epizootiological situation of the disease.
2. Clinical signs and lesions.
3. Laboratory diagnosis.

Samples: The following samples should be collected:

1. Enteric colibacillosis: Segments of ileum and colon, fecal swabs

and blood.

2. Septicemic colibacillosis: blood, spleen, lung and liver.
   a. Isolation and characterization of *E. coli* from blood (septicemic), intestines and feces (enteric).
   b. Isolates are examined for the presence of toxins using an enzyme immune assay and latex agglutination test.
   c. Detection of the K88 and K99 antigens using FAT and ELISA.
   d. Demonstration of the enterotoxins by infant mouse test.
   e. Detection of verotoxins in feces by ELISA using monoclonal antibodies.
   f. PCR test for detection of verotoxin genes.
   g. The use of DNA probes specific for genes encoding enterotoxins.
   h. Determination of the level of serum immunoglobulins in diarrheic calves.
   i. Estimation of the PCV of the blood of diarrheic calves.

Differential diagnosis

The disease should be differentiated from:

1. Dietetic diarrhea (feces are voluminous and pasty to gelatinous in consistency).
2. Other causes of infectious diarrhea using the combined diagnostic approach for detection of enteropathogens in the feces.

Treatment

- Treatment should be directed toward correction of the dehydration, acidosis, and electrolyte loss.
- Antibiotic treatment can be given simultaneously with the treatment for dehydration.
- Dehydration can be overcome with simple fluids given by mouth early in the course of the disease.
- If dehydration is in an advanced state, intravenous fluid treatment becomes necessary

Coliform septicemia

Parenteral antimicrobials and the use of I/V fluid therapy may be used until recovery is apparent.

Enteric colibacillosis:

1. Fluid and electrolyte therapy:

Dehydration, acidosis and electrolyte imbalances are corrected by the parenteral and oral use of electrolyte solutions. An equal mixture of isotonic saline (0.85%), isotonic sodium bicarbonate (1.3%) and isotonic dextrose (5%) is an effective solution for parenteral use. Bicarbonate is highly essential for correction of the metabolic

acidosis.

Severe dehydration (10 - 12% of BODY WEIGHT):

Hydration therapy: 100 ml/kg body weight, I/V in the first 2 hours at the rate of 50 ml/kg BODY WEIGHT

Maintenance therapy: 140 ml/ kg body weight, I/V over the next 8 hours at the rate of 20 ml/ kg body weight

Moderate dehydration (6 - 8% of BODY WEIGHT):

Hydration therapy: 50 ml/ kg body weight**,** I/V in the first 1-2 hours followed by the maintenance therapy 140 ml/ kg body weight over the next 8 hours.

***** Maintenance therapy can be provided using oral fluids and electrolytes using nipple bottle.

***** A solution for oral administration can be prepared by using one tablespoon baking soda, one teaspoon salt, and 250 cc of 50 percent dextrose.

***** Calves, which respond to the hydration therapy, begin to urinate within an hour after fluid administration had begun. Calves, which do not respond, may not begin to urinate because of irreversible renal failure.

2. Antimicrobial therapy

Tetracycline, Sulfonamides, Trimethoprim-Sulfonamide mixtures Neomycin sulfate and Ampicillin parenterally are commonly used but should be discontinued after three successive days of treatment to avoid elimination of drug sensitive intestinal flora.

- Non-steroidal anti-inflammatory should be included to reduce the risk of endotoxic shock as a result of death of large numbers of the organisms with release of endotoxins.

3. Intestinal protectants as kaolin and pectin should be used.

4. Diet

Milk intake should be reduced in diarrheic calves for up to 24 hours or until clinical improvement due to impairment of lactose digestion. Oral fluids and electrolytes should be used as milk replacement and after recovery milk can be introduced gradually.

3. **The use of probiotics** which contain intestinal microflora (lactobacillus and streptococcus species) to provide conditions, which are unfavorable for growth of the enteropathogenic bacteria.

Control

I. Reduction of exposure of newborn calves to infectious agents:

1. Calves should be kept in individual well-bedded clean box for at least the first month of life.
2. Calf pen should be cleaned and disinfected.

3. The perineum and udder of the cow should be washed shortly before calving.
4. The umbilicus should be swabbed with 2% iodine and tied at the level of the abdominal wall with cotton thread.
5. Affected calf should be removed and isolated.
6. Newly purchased calves should be reared separately.
7. Contamination of water should be avoided.
   - Milk intake should be restricted to 10% of the body weight daily for the first 7-10 days.

II. Raising of the non-specific resistance

1. Pregnant dam should be provided with the optimal nutrition.
2. Prepartum milking should be prohibited.
3. Parturition injuries must be minimized.
4. The optimum amount of colostrum should be ingested by the calf at the proper time. The calf should be encouraged and assisted to suckle within one hour.

III. Increase of specific resistance of the newborn calf:

1. Vaccination of pregnant dams with either *E.coli* K99 pili or whole cell preparation containing sufficient K99 antigen. *E.coli* bacterin is given once or twice to pregnant cows 2-4 weeks before parturition.
2. Oral administration of a K99 specific monoclonal antibody to calves during the first 12 hours after birth particularly during outbreaks.
3. The use of egg yolk powder prepared from hens vaccinated with heat extracted antigens from K99-piliated enterotoxogenic *E.coli.*

Watery mouth in lambs

It is a highly fatal disease affecting lambs in the first three days of life characterized by profuse salivation, gut stasis, retained meconium, collapse and death.

Etiology

The disease is caused by *E.coi*; a Gram-negative bacteria.

Epizootiology

Geographical distribution

It is a worldwide disease.

Susceptibility

Newborn lambs are highly susceptible due to contamination of the lambing site especially when they do not ingest colostrum sufficiently.

Sources of infection

These include contaminated bedding, udder, teats, and coat of the dam by discharges of lambing.

Pathogenesis

Due to the absence of colostral immunoglobulins, the bacteria multiply rapidly with development of bacteremia. Large quantities of endotoxins are released into the gut after death of the organisms. The endotoxemia is responsible for the clinical signs. Within 2 to 6 hours there are profuse salivation, a wet lower jaw and increasing abdominal distension although the lamb has not been sucking. The condition can quickly progress to coma and death.

Clinical signs

The disease is manifested by:

1. Marked depression.
2. Some lambs develop swollen tense abdomen due to gas formation in the abdomen.
3. Large amounts of saliva drool from the mouth.
4. Diarrhea may develop.
5. Dehydration, collapse, coma, and death occur within 24 of the onset of signs.

Diagnosis

1. Epizootiological situation of the disease.
2. Clinical signs.
3. Laboratory diagnosis.

This is based on the isolation of the causative agent from swabs or intestinal contents and mesenteric lymph nodes of dead lambs.

Treatment and prevention

1. The use of soapy water enemas and mild laxatives to promote gut activity and expulsion of meconium.
2. The use of electrolyte fluids orally or I.V.
3. Parental antibiotics and non-steroidal anti-inflammatory to reduce the risk of endotoxic shock.

- Lambs should obtain adequate colostrum as early as possible.

Calf Salmonellosis (Paratyphoid)

It is an infectious disease of calves manifested by three clinical syndromes; peracute septicemia, acute enteritis or chronic enteritis. A symptomatic carrier state occurs in adults. Reactivation of the disease in carriers occurs due to exposure to stress.

Etiology

The disease is caused by Salmonella organisms. They are Gram-negative rods.

- Some serotypes are adapted to certain host such as *S. typhi* and *S. paratyphi* A and C which affect only man, *S. dublin* in cattle, *S. abortus ovis* in sheep, *S. abortus equi* in horses and *S. cholera suis* in swine.
- Other serotypes can cause disease in man and a variety of animals (ubiquitous type) such as *S. typhimurium*, which is widely distributed and causes outbreaks in all kinds of animals.
- The organism may survive for months or even years in the environment (moist soil, dry feces, and animal feeds).
- Salmonellae produce enterotoxins but are also invasive and produce inflammatory changes within the intestine.
- In the host the organism persists in mesenteric lymph nodes tonsils and gall bladder.

Epizootiology

Geographical distribution

It is a worldwide disease.

Susceptibility

Cattle, buffaloes, sheep, goats, camels, horses, and dogs are infected.

Newborn calves up to 3-6 months are susceptible. Adults commonly act as carriers.

Sources of infection

1. Secretions and excretions of diseased or carrier animals; these include milk, colostrum, feces and vaginal discharges.
2. Rodents as rats and mice, dogs, cats, beetles, lizards, snakes and earthworms may act as carriers of the organism and have a role in transmission.
3. Animal products such as bone and meat meal, fish meal, and milk powders may also act as sources of infection.

After infection the animal may become a clinical case or an active carrier. Active carriers shed the organisms constantly or intermittently in the feces. In latent carriers, the infection persists in mesenteric lymph nodes or tonsils but no salmonellae in feces, such animals can become active carriers or even clinical cases under stress especially at calving time. Passive carriers usually acquire infection from pasture or the calf-pen floor and invasion of the tissues may not occur, these animals excrete the organism but stop excretion when they are removed from the infected area. In *S. dublin* infection, a long term of latent infection occurs in cattle (years or all life). The problem of latent carriers is that they cannot be identified by fecal culture or serologically.

Transmission

Infection spreads mainly by ingestion of contaminated food or water. It may also occur via inhalation or umbilicus. In carriers, the disease occurs after exposure to stress.

The response to infection depends on the infective dose, immunological status of the animals and exposure to stress especially in older animals as transportation, exhaustion, parturition, vaccination or concurrent infections as piroplasmosis or viral infections.

Some barriers for salmonella infection include acidity of the stomach or rumen, intestinal mucous, lysozyme, lactoferrin and normal bacterial flora.

Pathogenesis

After infection the organisms adhere and colonize the terminal ileum and cecum, penetrate it and reach to the submucosa where they are engulfed by macrophages. The organisms secrete enterotoxins which cause increase in adenyl cyclase and increase in CAMP content and prostaglandin concentration, this stimulates fluid secretion (active chloride secretion and inhibition of sodium absorption from the intestine). Release of cytotoxins causes tissue damage, distortion, degeneration and shortening of the villi. The organisms in macrophages reach lymphatics then the regional lymph nodes and then enter the blood and carried within macrophages throughout the body. In blood the organisms release endotoxins, and then localize and proliferate in intestine, mesenteric lymph nodes, gall bladder, liver, spleen, and bone marrow.

Clinical signs

A- Septicemia (peracute form)

Fever (40-42^{o}C) depression, dullness are followed by death within 24-48 hours.

*****Newborn animals that survive the septicemic state usually develop pneumonia, severe enteritis and if they survive this stage, polyarthritis and meningitis may develop.

B- Acute enteritis:

1. Fever, congestion of mucous membranes and increase thirst.
2. Severe fluid diarrhea and sometimes dysentery
3. Fever subsides precipitously with the onset of diarrhea.
4. Feces have a putrid smell and contain mucous, blood and fibrinous casts.
5. Dehydration, sunken eyes and prolonged skin tent.
6. The animal dies in 2-5 days if no treatment.

C- Chronic enteritis

It may occur in calves and adult cattle. Infected animals show intermittent fever, intermittent or persistent diarrhea with occasional passage of spots of blood and mucous in feces. This form is common in carrier cows after parturition due to immune suppression.

Post mortem lesions

Septicemic form: Submucosal and subserosal petechial hemorrhages may be observed.

Acute and chronic forms

Commonly there are:

1. Abomasitis and petechiation of the abomasal wall.
2. Intestinal contents are watery and contain mucous and blood.
3. Severe necrotic enteritis in the ileum and large intestine.
4. Submucosal petechiation and diffuse hemorrhagic enteritis.
5. Mesenteric lymph nodes are enlarged, edematous and hemorrhagic.
6. The liver is enlarged, dark and friable.

Diagnosis

1. Epizootiological situation of the disease.
2. Clinical signs and lesions.
3. Laboratory diagnosis.

Samples: These include:

Living animals: Feces, rectal swabs, blood and blood serum.

Dead animals: Liver, gall bladder, bile, mesenteric lymph nodes and spleen.

a. Microscopic examination of thick smears from the epithelial lining of the gall bladder.
b. Isolation of salmonellae from feces, gall bladder, bile, mesenteric lymph nodes and blood.

c. Detection of antigen in feces using Antigen-capture ELISA or in tissues using FAT.
d. The use of DNA probes and PCR.
e. Serological examination as serum ELISA or agglutination tests for detection of carriers.

Differential diagnosis

The disease should be differentiated from:

1. Dietetic diarrhea (feces are voluminous and pasty to gelatinous in consistency).
2. Other causes of infectious diarrhea using the combined diagnostic approach for detection of enteropathogens in the feces.

Treatment: As in colibacillosis.

Control

1. Reduction of exposure to infection.
2. Increase of non-specific resistance.
3. Rising of specific resistance: Killed bacterins and live attenuated vaccines are available. Live salmonella vaccines are more effective immunogens in calves than killed vaccines. Cows are vaccinated during late pregnancy, this will give passive protection to the calves for 6 weeks following sufficient colostrum intake. Calves can be vaccinated at that time if danger still exists.
4. Detection and elimination of carriers.

Calf Enterotoxemia

It is an infectious intoxication of young calves characterized by diarrhea, dysentery and sudden death.

Etiology

The disease is caused by toxins of *Cl. perfringens* type B or C. *Cl. perfringens* type A is involved in severe cases causing: Hemorrhagic bowel syndrome"HBS".

- *Cl. perfringens* type B produces necrotizing toxins as Alpha, Beta and Epsilon
- *Cl. perfringens* type C produces Alpha and Beta toxins.
- *Clostridium perfringens* type A has been associated with several conditions in cattle:
- Hemorrhagic abomasitis and abomasal ulceration,
- Hemorrhagic enteritis in adult cattle and calves,
- Hemorrhagic enteritis and sudden death in veal calves during the feeding period.
- Some isolates of *C. perfringens* type A produce β2-toxin which may contribute, along with α-toxin, to the development of hemorrhagic lesions in the small intestine in cases of bovine enterotoxemia.
- Isolates of *C. perfringens* type A have also been suggested as a cause of **jejunal** hemorrhage syndrome (JHS) in beef and dairy cattle.
- They are present commensally in the intestine of normal animals and humans, soil and feces.

Epizootiology

Geographical distribution

The disease is reported in different countries of the world.

Susceptibility

Young calves in good conditions of less than two weeks age are more susceptible but the disease may occur up to 10 weeks of age. Sudden change in feed or ingestion of large quantities of milk after a long period of starvation especially during inclement weather where mothers are delayed to return to their calves may predispose for infection.

Sources of infection

Soil is the main source of infection.

Transmission

It occurs by ingestion of spores.

Pathogenesis

Spores are ingested from soil. Under suitable conditions (anaerobic), spores germinate, proliferate, and attach to the surface of epithelial cells of the intestinal villus with toxin production.

Alpha-toxin; a lethal toxin, is a phospholipase, which results in cell lysis. Beta toxin causes increased capillary permeability; it is also a necrotizing toxin and produces damage to the microvilli, desquamation of epithelial cells resulting in hemorrhagic enteritis and ulceration of the intestinal mucosa.

Clinical signs

1. In per-acute cases, death occurs in a few hours, some times without diarrhea.
2. Acute abdominal pain and bellowing usually occur.
3. Diarrhea, dysentery, and bloody feces are characteristic.
4. In severe cases of Hemorrhagic bowel syndrome "HBS", there is severe sweating, teeth grinding sternal recumbence, lethargy, Slight bloating and distended gut loops per rectal palpation.
5. In the terminal stage, neurological signs including tetany may be observed.

Post mortem lesions:

1. Ulceration at the intestinal mucosa.
2. Intestinal contents are bloody.
3. In "Hemorrhagic bowel syndrome", there is segmental hemorrhage and clotting forming a plug.
4. Subendocardial and subepicardial hemorrhages.

Diagnosis

1. Epizootiological situation of the disease.
2. Clinical signs and lesions.
3. Laboratory diagnosis.

Samples: A lobe of small intestine is taken on ice.

a. Microscopical examination of stained smears from intestinal content and ulcers (Gram positive rods).
b. Cultural examination of intestinal contents on blood agar anaerobically at 37^{o}C. Smooth round colonies surrounded by double zone of hemolysis are formed.
c. Detection of toxins in the intestinal contents or bacterial culture. The supernatant of the intestinal content filtrate is injected in 3 mice I/V in the tail vein. If death occurs within 12 hours, it indicates presence of toxins.
d. Identification of toxins: (toxin assay or neutralization test).

Mouse toxin assay

Mixture of clarified intestinal filtrate and known antitoxins of various types of C. *perferingens* are inoculated into 2 groups of mice I/V in the

tail vein. Survived mouse will indicate the type of *Cl. perferingens.*

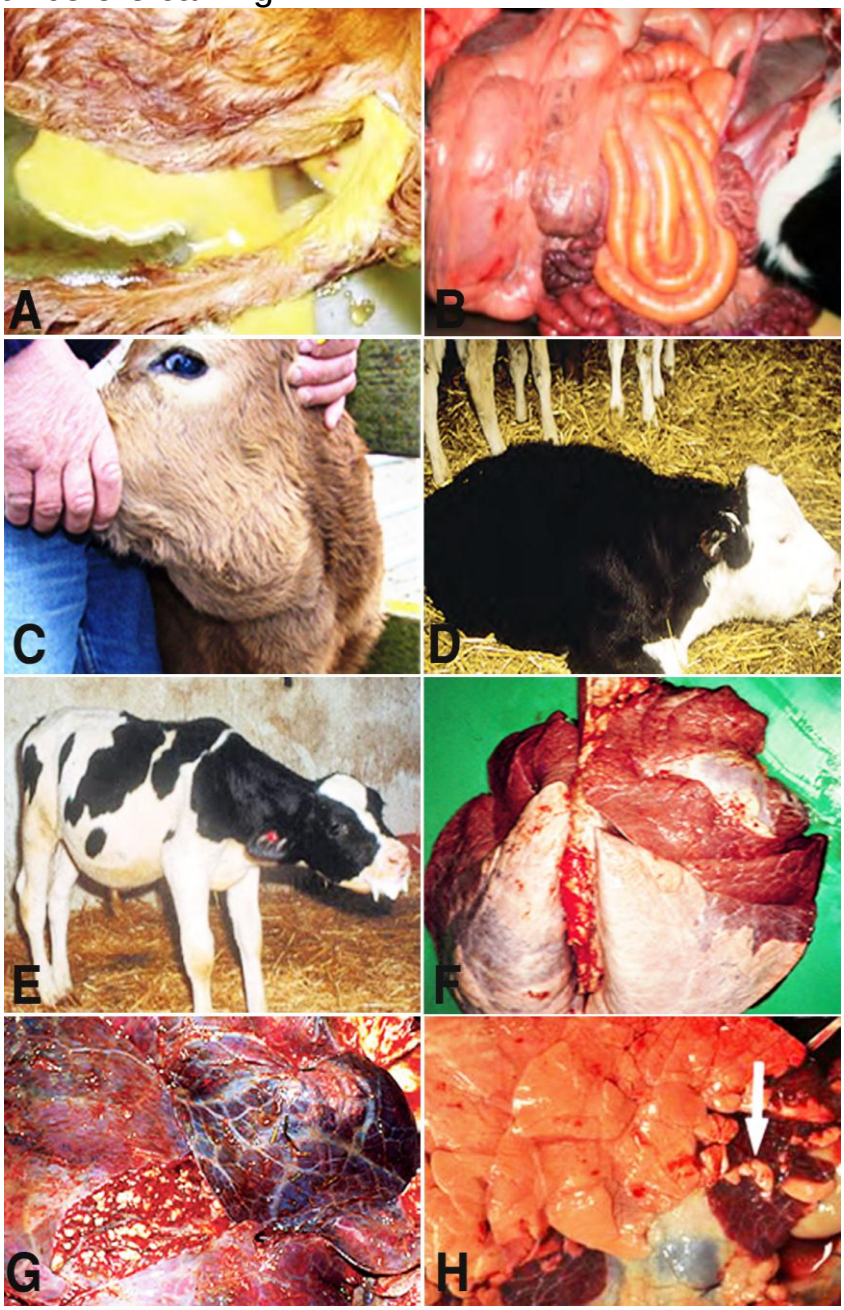
Guinea pigs toxin assay

Mixture of clarified intestinal filtrate is injected in an area of skin and another area is injected with intestinal filtrate and antitoxins. Skin necrosis occurs after 48hours in the area injected with the intestinal filtrate alone.

Specific antitoxins can be detected in sera of recovered animals

Control

1. Predisposing causes should be avoided.
2. Vaccination by using of *Cl. perferingens* toxid type B and C. Cows are vaccinated twice 2-4 weeks apart with yearly boostering, one month before calving.



Viral diarrhea in newborn calves

It is an infectious disease of newborn calves characterized by acute profuse watery diarrhea, and rapid death.

Etiology

The disease is caused by

- Rota viruses, family Reoviridae (RNA viruses): Seven sero-groups are recognized. Viruses are excreted in feces of diseased calves and inapparently infected cattle. They affect the upper two thirds of the villus. They destruct the absorptive cells of intestinal villi of small intestine (duodenum). They are inactivated by 4% iodophore, 5% lysol and 10% formalin.
- Corona viruses, (RNA viruses): They affect the tip and middle of the villus. These viruses destruct the villi of small intestine (ileum) and large intestine (colon), they are inactivated by ether and chloroform.
- Toro virus (Breda virus).
- Other viruses as Adenoviruses, Astrovirus and Calici- like virus
- Bovine Parvovirus affects the crypt and peyer's patches.
- Pestivirus (BVD).
- Morbillivirus (Rinderpest and Peste des petitis ruminants)

Mixed viral infections associated with *E.coli* or cryptosporidium are common.

The infection process depends on several factors:

1. Age of the calf.
2. Immune status of the dam and calf.
3. Weather condition (ambient temperature).
4. Weaning.
5. Presence of other pathogens.

Epizootiology

Geographical distribution

It is a worldwide disease.

Susceptibility

Newborn calves, lambs and kids are affected. Adults usually suffer from inapparent or latent infection. Rotavirus usually infects calves 1-3 weeks of age. Corona virus infects calves mostly 1-2 weeks of age. This age of occurrence is related to the rapid decline in specific colostral antibody because the protection lasts only as long as colostral antibody is present within the lumen of the intestine.

Sources of infection

Feces of newborn infected calves and inapparent infected cattle are the main source of infection as it contains high titers of viruses.

Transmission

It occurs by ingestion of the virus in contaminated feed, teats and fomites.

Pathogenesis

After ingestion, Rotavirus infects the brush border villous epithelial cells in the small intestine and replicates in the mature absorptive and enzyme-producing enterocytes on the villi of the small intestine, leading to rupture and sloughing of the enterocytes with release of virus to infect adjacent cells. This results in atrophy of epithelial cells, stunting, sloughing, thickening and fusion of villi. The activity of lactase enzyme in the brush border of the villous epithelium decreases causing decrease of utilization of lactose. Such morphological and functional changes in the intestine result in mal-absorption, diarrhea, dehydration, loss of electrolytes and acidosis. Pathogenesis of Corona virus infection is similar to rotavirus but villous atrophy occurs in both small and large intestines with mucous in feces due to enterocolitis. Secondary bacterial infection usually develops.

Incubation period is 10-30 hours

Clinical signs

The disease usually occurs in calves over four days of age.

A. Rota virus infection

1. Sudden onset of profuse liquid watery feces.
2. Feces are pale yellow and may contain blood.
3. Dehydration, abdominal distension and recumbence.

B. Corona virus infection

1. Severe watery feces which contain mucous.
2. Dehydration, weakness and depression.

C. Bovine Parvo virus infection: It causes diarrhea **in neonatal calves** and also **respiratory and reproductive disease** in adult cattle.

1. Diarrhea is the main clinical sign and is often alone.
2. Abortion and birth of weak or stillborn calves. Fetuses in the first trimester are most susceptible.
3. Cough, dyspnea and nasal discharge may develop.
4. Lymphopenia is common on hematology.

*Mortality is low in pure virus infection but high in mixed infection.

*Recurrent infections may occur due to the presence of different serotypes of bovine rotavirus or may be due to rotavirus antigenically modified in the host.

Post mortem lesions

1. Emaciation and dehydration of the carcass.

2. Distension of the abomasum.
3. Ulcerative enterocolitis (Corona virus).

Diagnosis

1. Epizootiological situation of the disease.
2. Clinical signs and lesions.
3. Laboratory diagnosis.

Samples: Fecal samples (20-30 grams) should be collected within few hours of the onset of diarrhea. Samples of intestinal mucosa (small and large intestine) and Paired blood serum samples.

Diagnosis must include consideration of all the common pathogens.

a. Virus isolation on cell cultures.
b. Electron microscope for demonstration of the virus in feces.
c. FAT of fecal smears or cell cultures.
d. ELISA for detection of the virus.
e. PCR assay for detection of the virus.
f. Serology as ELISA, CFT and latex agglutination test.

Differential diagnosis

The disease should be differentiated from other infectious diseases causing diarrhea in newborn calves.

Control

1. Avoid the predisposing factors.
2. Proper management of pregnant dams and newborn calves.
3. Good sanitation and hygiene to minimize the spread of infection.
4. Vaccination of dams or newborn calves by inactivated or living attenuated vaccines or multi-vaccines (Rota virus, Corona virus and *E.coli*).

Dams are vaccinated at the late stage of pregnancy.

* Failure of calf vaccination in enzootic areas is due to the presence of specific antibodies in the colostrum (neutralization).

Cryptosporidiosis

It is an infectious disease of newborn calves characterized by diarrhea, retardation of growth and emaciation.

Etiology

The disease is caused by *Cryptosporidium spp.,* an intracellular protozoan of the intestinal tract. Two species are important in livestock animals.

Cryptosporidium parvum affects small intestine of cattle, sheep, goats, deer and man.

Cryptosporidium andersoni affects the abomasum of cattle.

- Oocyst is spherical ovoid or rounded (5-6 nm in diameter). It contains four elongated sporozoites.
- Oocysts are resistant to most disinfectants and can survive for several months in cool and moist conditions.
- Infectivity of oocysts can be destroyed by ammonium hydroxide, hydrogen peroxide, 10% formol saline and 5% ammonia.
- Oocysts are readily sporulated and infective when excreted in the feces.

Epizootiology

Geographical distribution

It is a widely distributed disease.

Susceptibility

Neonatal calves, lambs, goat kids, foals and piglets are susceptible. Calves under one month of age are usually affected. It also affects man.

Sources of infection

Feces are the main source of infection where oocysts are readily sporulated and infective when excreted in the feces.

Adults are inapparent carriers and shed the organisms.

Transmission

Infection occurs by fecal oral route. It occurs directly from calf to calf or indirectly via fomites, contaminated feed, or water supply.

Infection from other species such as rodents or cats is also possible (non-host specific).

Pathogenesis

Infection occurs by ingestion of the oocysts, which undergo excystation, asexual multiplication, gamete formation, fertilization, and sporozoite formation.

The cryptosporidium is present in the lower part of the small intestine and occasionally in the cecum and colon. Cryptosporidia do not require fecal excretion for sporulation to infective stages, and they sporulate in

the intestine. The prepatent period ranges from 2-7 days in calves and 2-5 days in lambs. The cryptosporidium appears free in the lumen of the intestine and attached to the microvilli of the villous epithelial cells.
The parasite does not invade but adheres to the apical surface of enterocytes in the distal small intestine and the colon. This results in loss of microvilli, decreased mucosal enzyme activity with villous blunting and fusion (leading to a reduced villous surface absorptive area), and inflammatory changes in the submucosa. Diarrhea occurs due to villous atrophy blunting of the villi, crypt cell hyperplasia and impaired digestion and absorption.

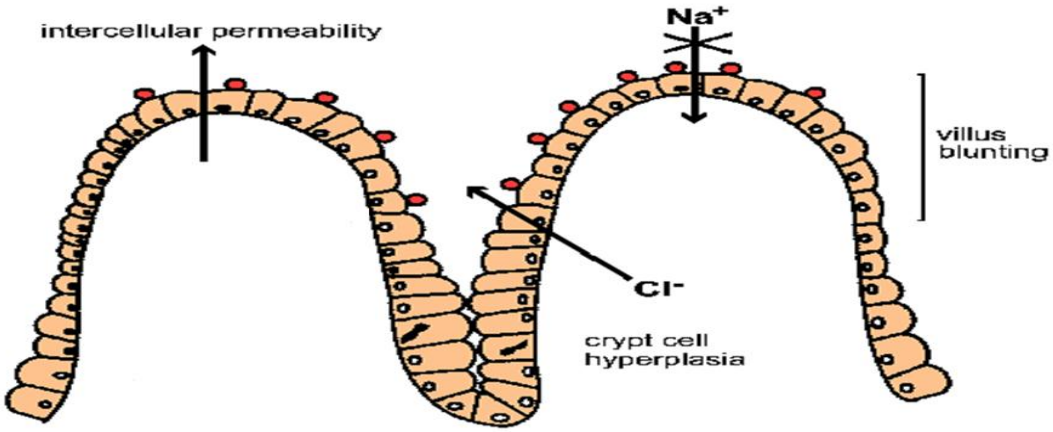


Figure (22): Pathogenesis of cryptosporidiosis

Clinical signs

1. Feces are yellow or pale, watery and contain mucous.
2. There are loss of body weight, emaciation, and dehydration.

Post mortem lesions

1. Dehydration and emaciation.
2. Congestion of small and large intestines.

Diagnosis

1. Epizootiological situation of the disease.
2. Clinical signs and lesions.
3. Laboratory diagnosis.

Samples: Feces, intestinal scrapings and blood sera.

a. Detection of the fecal oocysts in fecal smears stained by different stains as:

- Giemsa stain: Oocysts appear blue containing red granules with blue background.
- Safranin/methylene blue: Oocysts appear orange with blue background.
- Methylene blue/eosin stain: Oocysts appear blue with purple nuclei with green background.
- Modified Ziehl-Neelsen: Oocysts appear dense red with green background.

b. Oocysts are readily detected by phase contrast microscopy.
c. Immunofluorescence technique on fecal smear.
d. Demonstration of oocysts concentrated from fecal samples by centrifugal flotation in salt or sugar solution.
e. Immunological assays such as immunofluorescence (IF) or enzyme-linked immunosorbent assays (ELISA).
f. PCR.

Treatment

1. Halofuginone 60 - 125 ug / kg body weight, daily for 7 days orally.
2. Paromomycine sulfate 100 mg/kg body weight, daily for 11 consecutive days from the second day of age is successful in prevention of the disease.
3. Fluid and electrolyte therapy is indicated.

Control

1. Detection and isolation of infected animals.
2. Treatment of infected calves.
3. Application of hygienic measures and application of suitable disinfectants.
4. Newborn calves should be separated from adults (carriers).
5. Rats, mice, pets are regarded as potential reservoir of infection.

Calf pneumonia

Pneumonia in calves has a complex etiology with many pathogens implicated in this syndrome. Pneumonia may be caused by viruses, bacteria, mycoplasmas and usually a combination of them. In most cases of calf pneumonia, the disease is bronchogenic in origin but in some cases the disease originates hematogenously such as the cases of pneumoenteritis which develop due to post septicemic localization. Inflammation of the pulmonary parenchyma is usually accompanied by inflammation of bronchioles and sometimes by pleurisy. Generally, the disease is clinically manifested by an increase in the respiratory rate, change in the depth and character of respiration, coughing, and abnormal sounds on auscultation.

Causes of pneumonia in newborn calves: Acute undifferentiated bovine respiratory disease has a complex etiology.

1. Pneumonia associated with *E.coli* and salmonella infections (pneumoenteritis).
2. *Fusobacterium necrophorum:* A complication of calf diphtheria.
3. *Klebsiella pneumonae* infection.
4. *Mannheimia (Pasteurella) haemolytica*

5. *Pasteurella multocida* associated or not associated with parainfluenza-3 virus.
6. *Histophilus (Haemophilus somni*),"feed lot cattle".
7. Several viruses: Parainfluenza-3, Bovine respiratory syncytial virus, Adenovirus 1,2 and 3, Rhinovirus, Reovirus, BVD, IBR virus associated with Pasteurella spp., *Trueperella* (*Arcanobacter or Actinomyces) pyogenes,* Streptococcus spp*.,* Mycoplasma spp. and Chlamydia spp*.*

A complex of biochemical, physiological and immunological defense mechanisms protects the respiratory tract of the calf against the inhaled particles. This includes the aerodynamic filtration by the nasal cavities, sneezing, cough reflex, mucociliary transport mechanism, local and systemic antibody systems and alveolar macrophages.

Respiratory mucociliary clearance mechanism plays an important role in protection of the respiratory tract against infection. Mucous traps and transports inhaled particles to pharynx where they are swallowed; also, it keeps the underlying mucosa hydrated. Mucous contains antibodies especially IgA which together with lactoferrin and lysozyme provide immunological defense. In case of infection the mucociliary clearance is impaired due to disruption of effective ciliary activity and changes in quantity or quality of the mucous.

In viral infections, ciliary activity is disrupted due to deciliation or lesions of the respiratory mucosa.

Stress is one of the most important factors, which play a role in buildup of respiratory infection. Stress, generally reduces the resistance to infection due to the high adrenocortical activity. Exposure of calves to adverse environmental conditions, transportation, and weaning increases their susceptibility to respiratory infection.

Calf Diphtheria
Oral necrobacillosis, Necrotic stomatitis, Necrotic Laryngitis

It is an infectious disease of calves characterized by ulcerative necrosis in the check, tongue, pharynx and larynx.

Diphtheria is derived from the Greek word for leather, due to the type of exudates that forms.

Etiology

Fusobacterium necrophorum is a non-spore forming Gram negative obligate anaerobe and a normal inhabitant of the alimentary tract and the respiratory tract.

- The factors, which contribute to the pathogenicity of the organism, include a potent endotoxin, a polysaccharide capsule, an exotoxin (leukocidin) and a hemolysin.
- The organism is a common inhabitant of the gut and environment.

Epizootiology

Geographical distribution

It is a disease of worldwide distribution.

Susceptibility

Oral infection occurs in calves less than three months. A laryngeal infection is more common in older animals up to 18 months of age.

- One predisposing cause is thought to be traumatic injury.
- Low grade infections by some infectious agents of BRD may play a role.

Sources of infection

Saliva of infected animals and contaminated foods are the main sources of infection.

Transmission

Infection occurs by penetration of the organism to the injured oral mucosa, which is caused by rough feed or erupting teeth.

In crowded conditions, calves can spread infection via feed buckets.

Pathogenesis

The organism causes inflammation and necrosis of the injured oral mucosa, pharynx and larynx. Edema and inflammation of the mucosa of the larynx result in inspiratory dyspnea. Exudates can fill the lumen of the larynx and suffocate the calf. Swelling of the pharyngeal area develops. Fatal suppurative bronchopneumonia and toxemia finally lead to death.

Clinical signs

1. Fever, depression and complete anorexia.
2. Necrotic stomatitis and necrotic deep ulcers in oral mucosa.
3. Salivation.
4. Affected calves have difficulty breathing due to constriction of the larynx with audible sound as the calf breaths.
5. Moist painful cough and severe inspiratory dyspnea.
6. Breath has foul rancid smell.
7. Swelling at pharyngeal region which is painful on external palpation.
8. Spread to lungs causes fatal pneumonia, toxemia and death.

Post mortem lesions

1. Necrotic deep ulcers on the oral mucosa which are filled with necrotic material and covered with caseous material and diphtheretic membranes that bleeds when removed
2. Similar lesions are present in pharynx and larynx (necrotic laryngitis).
3. In severe cases similar lesions are present in lungs and abomasum.

Diagnosis

1. Epizootiological situation of the disease.
2. Clinical signs and lesions.
3. Laboratory diagnosis.

Samples: Swabs from the deep ulcers should be collected.

a. Microscopical examination of Gram stained smears.
b. Isolation of the organism.
c. Animal inoculation: Rabbit inoculation, I.P. causes death within 5-10 days with fibrinous peritonitis.

Treatment

1. Removal of necrotic material with application of antiseptic solution.
2. Oral administration of sulphamethazine at a dose of 150 mg/ kg body weight, daily for 3-5 days.
3. Broad-spectrum antibiotics can be used for up to 3 weeks.
4. Non-steroidal anti-inflammatory drugs to reduce swelling and fever.
5. Tracheotomy may be necessary.

Control

1. Application of hygienic measures.
2. Avoidance of rough feed.
3. Prophylactic feeding of antibiotics in enzootic areas.
4. Measures that reduce the risk of BRD may reduce calf diphtheria since it removes some of the predisposing factors that allow F. necrophorum become established.

Enzootic pneumonia of calves

It is an infectious disease of calves characterized by varying degrees of severity of pneumonia caused by different viruses associated or not associated with bacterial bronchopneumonia.

Etiology

The disease is caused by combination of respiratory viruses, bacteria and mycoplasmas associated with environmental factors such as inadequate housing and ventilation.

- Host risk factors such as the immune status of the calf also contribute in infection.
- Viruses and mycoplasmas act as primary pathogens while bacteria may cause secondary complications.

Pathogens include:

- Viruses

1. Parainfluenza-3 virus.
2. Bovine respiratory syncytial virus.
3. IBRV
4. BVDV
5. Other viruses: Adenovirus, Rhinovirus, and Reovirus.

- Mycoplasma spp., especially *Mycoplasma bovis.*
- Bacteria:

1. *Mannheimia haemolytica,*
2. *Pasteurella multocida,*
3. *Histophilus somni,*
4. *Trueperella* (*Arcanobacter or Actinomyces) pyogenes.*

Bacterial infections of the lungs, leading to pneumonia, are the ultimate cause of death in the majority of cases.

Epizootiology

Geographical distribution

It is a worldwide disease.

Susceptibility

Calves during the first week of life may be affected. The disease commonly affects calves of 2-5 months of age. The disease commonly occurs in calves raised under intensified conditions. Excessive moisture, bad ventilation, overcrowding and adverse environmental conditions are the main predisposing factors.

Sources of infection

Nasal discharges and expired air of infected animals are the main sources of infection.

Transmission

Infection occurs by aerosol infection and direct contact.

Pathogenesis

Respiratory viruses cause viral interstitial pneumonia that affects the cranial lobes of the lung leading to subclinical, mild clinical or severe fatal disease. Following infection, bronchitis, bronchiolitis and bronchiolar epithelial hyperplasia and giant cell syncytial formation occur. After the primary viral pneumonia, bacterial invasion may occur. Viruses reduce the resistance of mucous membranes allowing bacteria to invade tissues. Viruses destroy the cilia on the bronchial mucosa, this predisposes to secondary bacterial infection and inflammation. The resulting pneumonia will vary with species of bacteria.

Clinical signs

When a feedlot steer is depressed, off food, blowing, feverish and with its ears down, pneumonia should be suspected.

1. Infection by respiratory syncytial virus causes severe respiratory disease and death in individual animals. Severe signs as polypnea, dyspnea, mouth breathing and expiratory grunting and death may occur in 2 - 4 days due to lung edema and interstitial pneumonia. The virus kills epithelial cells lining airways. The virus has a fusion protein that, when expressed on the surface of the infected cells, causes fusion and formation of multinucleated ‘syncytial’ cells.
2. PI3 Infection is common in cattle. Clinical disease is usually mild or subclinical. The virus, however, predisposes to bacterial pneumonia by infecting epithelial cells lining the respiratory tract.
3. IBR is characterized by rhinitis, pharyngitis, laryngitis, tracheitis, mucopurulent nasal discharge, respiratory distress and conjunctivitis, erosions and ulceration of the oral cavity and upper alimentary tract, convulsions, circling and death.
4. BVD is characterized by salivation, mucopurulent nasal discharge, respiratory distress and discrete oral shallow erosions that include the inside of lips, gums, dental pad, and posterior part of the hard palate and on the tongue.
5. A bacterial pneumonia is at least contributory in most cases with different forms of pneumonia develop according to the secondary bacterial invaders. Following secondary bacterial infection, fever, dyspnea and toxemia develop. Variable PM findings may be observed.
6. *Mannheimia haemolytica*: This Gram-negative bacillus inhabits the nasal passages and throat of healthy cattle. It is the most important single cause of BRD. Under conditions of stress, the

bacterium moves into the lower airways and causes a virulent, fulminant pneumonia with deaths commonly occurring from 2-3 or up to 7-8 days. *M. haemolytica* produces a potent toxin that affects neutrophils and macrophages. This is the basis for the distinctively severe necrosis and inflammation that characterize *M. haemolytica* pneumonia.

7. *Pasteurella multocida*: This is a Gram-negative bacillus that also inhabits the upper respiratory tract of cattle, and is generally found living in the tonsils. The pneumonia caused by *P. multocida* is typically less fulminant than that induced by *M. haemolytica*. It is a common cause of the milder pneumonias seen in young calves, especially in dairies "enzootic pneumonia".
8. *Histophilus somni* (*Haemophilus somnus):* This is a Gram-negative bacterium. It is a normal inhabitant of the nasal passages and lower urogenital tract in cattle. It can cause either an acute fulminant pneumonia resembling *M. haemolytica* or more chronic pneumonia. *H. somnus* can cause a severe myocarditis, encephalitis as well as genital and joint infections.
9. *Trueperella* (*Arcanobacter or Actinomyces) pyogenes*: This is a Gram-positive coccobacillus or bacillus. This bacterium is commonly a lung pathogen occurring in mixed infections with other bacteria during the later stages of pneumonia. It causes extensive liquifactive necrosis and abscesses.
10. *Mycoplasma spp.*: There is evidence that *M. bovis* may act as contributory, late secondary invaders.

Postmortem findings

1. In viral uncomplicated pneumonia there are emphysema in the apical and cardiac lobes and dark red consolidation.
2. In respiratory syncytial virus infection there are lung edema, severe interstitial pneumonia and emphysema and lungs fail to collapse,
3. When bacterial or mycoplasmal invasion occurs, the lesions vary with the invading agent.
4. A severe fibrinous pleuritis with minimal or no involvement of lung may be observed with *M. haemolytica* and *P. multocida* and occasionally with *Histophilus somni*.
5. Extensive hepatization occurs with *P. multocida* infection.
6. Extensive suppuration occurs with *Trueperella* (*Arcanobacter or Actinomyces) pyogenes.*

Diagnosis

1. Epizootiological situation of the disease.
2. Clinical signs and lesions.
3. Laboratory diagnosis.

Samples: Nasal swabs, portions of the lungs and paired serum

samples are collected.

a. Isolation of the virus on cell cultures. Isolation of BRSV is difficult due to the labile nature of the virus.
b. IFA test for antigen detection of BRSV.
c. Serological tests as SNT, ELISA and FAT.

Differential diagnosis

The disease should be differentiated from:

1. Bacterial pneumonia as Pasteurella spp*, K. pneumonae* and *H. somnus*. They are characterized by severe toxemia.
2. Calf diphtheria: Sporadic cases with oral and laryngeal lesions.
3. Lung worm pneumonia usually affects young calves at pasture.

Treatment

1. Antimicrobial therapy is indicated to combat secondary bacterial pneumonia.

Tetracyclines, Trimethoprim potentiated sulphonamides,Erythromycin, Spectinomycin are effective. Danofloxacillin is effective against Mycoplasma spp.

2. Non-steroidal anti-inflammatories like Ibuprofen, Phenyl butazone and Acetyl salicylic acid.
3. Corticosteroids have been recommended for treatment of BRSV infections in calves.
4. Bronchodilators and expectorants.
5. Atropine 0.048 mg/kg IM.
6. Antihistaminics such as Tripelennamine hydrochloride 1 mg/kg IM twice daily.
7. 6. Furosemide 250 mg (if severe pulmonary edema is present) IV or IM once or twice daily.
8. Supportive treatment including Vit. A and B.

Control

1. Application of effective animal and environmental management.
2. Avoidance of all predisposing factors such as overcrowding and bad ventilation.
3. Isolation of recently purchased calves for several weeks.
4. Vaccination:

- Intranasal vaccine of PI3 stimulates serum antibody and nasal antibody production.
- Inactivated PI3 vaccine induces high levels of serum antibody.
- Combined vaccines containing PI3, BRS, IBR, BVD viruses are available.

Generally, the control of acute or chronic enzootic respiratory disease within groups of calves or adult cattle consists of four components:

- Definitive diagnosis of the causative agent(s).

- Specific medical therapy.
- Correction of management, environmental, or ventilation deficiencies that contribute to or perpetuate the respiratory disease.
- Preventive medicine, including management techniques and vaccination.

Navel ill, Omphalophlebitis, Joint ill

It is an infectious disease of calves and lambs characterized by swelling, inflammation and abscessation of the navel, arthritis and lameness.

Etiology

Different types of bacteria are involved including staphylococcus spp., *Trueperella* (*Arcanobacter or Actinomyces) pyogenes, F. necrophorum,* Salmonella spp*, E. coli* and Pasteurella spp.

Epizootiology

Geographical distribution

It is a worldwide disease.

Susceptibility

Calves of 1-2 weeks age are highly susceptible.

Sources of infection

Contaminated soil and bedding, uterine discharges from infected dams and discharges from opened lesions of other animals are the main sources of infection.

Transmission

It occurs by contamination of the umbilicus.

Pathogenesis

Contamination of the navel leads to local inflammation (navel ill). Extension may occur to the liver or via the urachus to the bladder. Systemic extension may cause septicemia. Localization in the joints causes suppurative arthritis. Localization in the eye may cause panophthalmitis and in meninges causes meningitis.

Clinical signs

1. In septicemic form there is fever.
2. The umbilicus is enlarged, painful on palpation discharging purulent material.
3. Large abscess may develop along the course of the umbilical vein.
4. Post septicemic localization in joints, eyes or brain may occur.

Diagnosis

1. Epizootiological situation of the disease.

2. Clinical signs and lesions.
3. Laboratory diagnosis.
4. Isolation of the causative agents from smears or pus from the navel or the joints.

Treatment

1. Surgical treatment of the abscess at the umbilicus.
2. Washing with antiseptic solutions and antibiotics.
3. Systemic antibiotics as penicillin streptomycin combination or sulpha drugs.

Control

1. Proper care of the navel immediately after birth, umbilical cord should be legated and swabbed with tincture iodine.
2. Avoid contamination of the bedding with the uterine discharges or discharges from opened lesions.

Abortion in dairy cattle is commonly defined as fetal death and expulsion between the age of 42 days and 260 days of pregnancy.

- Early embryonic death refers to the losses which occur in the period between fertilization and the day 42.
- Stillbirth refers to dead fetus expelled between 260 days and full term.
- Gestation length in ewes is usually 147 days but breed differences mean that it can vary from 140 to 150 days. During any abortion episode, some fetuses may be infected yet still carried to term and some may be carried to term and fully viable.
- An annual abortion rate up to 3% is considered to be normal. This figure excludes most abortions occurring during the second and third month of gestation as these often go undetected.
- An abortion rate in excess of 10% is considered an abortion storm.

Causes of abortion

Abortions may result from a broad range of causes.

1. **Non-infectious causes**
    - Genetic.
    - Hormonal disturbances.
    - Heat stress.
    - Nutritional deficiencies.
    - Toxic agents including phytotoxins and mycotoxins.
    - Administration of abortifacient drugs.
    - Trauma.

2. **Infectious causes**
    - Severe general infections of the pregnant dam that cause high fever such as Babesiosis, Theileriosis, LSD and acute mastitis may also lead to abortion.
    - Specific infections such as Brucellosis, Leptospirosis, BVD etc.

Some specific infectious diseases causing abortion

1. Brucellosis.
2. Leptospirosis.
3. Campylobacteriosis.
4. Listeriosis.
5. Salmonella abortion.
6. Mycotic Abortion.
7. Chlamydophilosis (Chlamydiosis).
8. Trichomoniasis.
9. Toxoplasmosis.
10. Neosporosis "*Neospora caninum* infection".
11. Bovine Viral Diarrhea (BVD).
12. Border Disease (BD).
13. Infectious Bovine Rhinotracheitis (IBR).
14. Bluetongue (BT).
15. Rift Valley Fever (RVF).
16. Akabane disease.
17. Cache Valley Virus disease.
18. Wesselbron Disease.
19. Bovine Parvovirus infection

Management of specific infectious abortions

1. Proper hygienic and biosecurity measures in the animal's environment and feed storage.
2. Isolation of aborting cows and immediate removal and proper disposal of aborted material by burning or deep burying (deep enough to protect from dogs and other scavengers).
3. Remove the bedding and apply lime to the contaminated area.
4. If the stable has a compacted earth floor remove the top layer of the floor and dispose of safely.
5. Immediately separate the abortion animal from the rest of the herd.
6. Do not allow calves, kids or lambs to feed on milk from aborted animals.
7. It is important to disinfect the place where the abortion happened as early as possible.

8. Immediate diagnosis of the infectious cause of abortion on epidemiological, clinical and laboratory basis.
9. Adequate immunization against enzootic infectious diseases causing abortion.
10. Ewe lambs fostered on to aborted ewes should not be retained for future breeding.
11. Ponds and surface water should be fenced.
12. Do not allow calves, kids or lambs to feed on milk from aborted animals.
13. Control of rodents, dogs and cats.

Prevention of abortion:

Freedom from most infectious causes of abortion is best achieved by maintaining a closed clean flock or herd. This can be achieved by application of Biosecurity and Biocontainment measures to maintain a closed clean flock or herd. A closed herd or flock:

- Never buy animals.
- Animals that leave do not return.
- No shared pasture or fence with neighbor livestock.
- Use artificial insemination for breeding.
- Do not exhibit at shows.
- Use home-grown replacements.

Investigation of a problem of abortion:

The classical presenting complaint is usually numerous abortions clustered in time.

Diagnosis of the cause of abortion is a difficult task. The history will help create an appropriate list of diagnostic hypotheses. The following information will help to approach diagnosis.

- Number, proportion, type, and age of animals aborting.
- Clustering of cases of (dates of abortions and gestational age).
- Recent introductions to the herd or flock.
- Previously diagnosed abortions or illness on the farm (History of abortions and epizootics).
- History of application of abortion vaccines and timing and frequency of administration.
- Evidence of recent septicemic diseases in the farm or flock.
- Evidence of diarrhea in aborting and contact animals.
- Evidence that the animals have access to water.
- Evidence of presence of rodent, dogs and cats in vicinity.

Criteria for submission of aborted fetuses for diagnostic evaluation.

a. When the abortion rate exceeds 3%.
b. When a number of animals abort over a short period of time.

Requirement for laboratory diagnosis of abortion

The minimum requirement for laboratory submissions for abortion diagnosis includes the aborted fetus or fetal stomach contents, a piece of placenta, and a maternal serum sample.

To confirm the presence of a particular abortion agent in a herd two blood samples should be taken from 10% of the animals that have aborted (2nd sample to be collected 2-3 weeks after 1st sample).

Brucellosis
Contagious abortion, Bang's disease

It is a chronic highly contagious disease of sexually mature animals characterized by abortion, retention of placenta and high rate of infertility. Brucellosis causes serious economic losses to livestock breeders due to abortion, decrease in milk yield and infertility. The disease is also important zoonoses.

- The microorganism responsible for Malta fever was recovered by a British army physician, Sir David Bruce, on July 9, 1887, in Malta from the spleen of a British soldier who had died of the disease. He called it *Micrococcus melitensis*.
- Latent infection is one of the important features of brucellosis. This occurs in calves following in utero infection or by ingestion of infected milk. These animals show no signs of disease and there is no seroconversion until they reach sexual maturity.
- Infected cows usually remain as chronic carriers for life and discharge brucellae in milk.

Etiology

The disease is caused by different species and serovars of brucella organisms including *Brucella abortus, Brucella melitensis, Brucella suis, Brucella ovis* and *Brucella cains*.

- They are Gram negative coccobacilli or short rods (0.6-1.5um x 0.5-0.7um) and filterable forms are recorded.
- Brucella species are not truly acid-fast, but they are resistant to decolorization by weak acids, and stain red against a blue background.

- They grow intracellularly and intercellularly.
- Some biovars of *Brucella abortus* require 10% Co2 tension for their growth.
- The organisms can persist in organic substances in the environment for up to 4-6 months. They are susceptible to heat, sun light, dryness and disinfectants as phenols and quaternary ammonium. Disinfectants reported to destroy brucella on contaminated surfaces include 2.5% sodium hypochlorite, 2-3% caustic soda, and 2% formaldehyde solution. Ethanol, isopropanol, iodophores and substituted phenols can be used on contaminated skin.
- In the host, brucellae survive for life.
- Brucellae can't survive in acidic medium such as sour milk.

Epizootiology

Geographical distribution

Brucellosis is a worldwide disease. It has been reported in Egypt since 1939 and still constitutes a serious problem.

Susceptibility

Cattle, buffaloes, sheep, goats, camel, swine, horse, and wild animals as deer, bison and elk are susceptible.

Organism	Animal Reservoir
Brucella melitensis	*Goats, *sheep, camels.
Brucella abortus	*Cattle, buffalo, camels, yaks.
Brucella suis	*Pigs (biotypes 1-3).
Brucella canis	*Canines.
Brucella ovis	*Sheep.
Brucella neotomae	Rodents.
Brucella pinnipediae *Brucella cetaceae*	Marine animals, mink, whales, Dolphins, seals.
Brucella microti	Common vole *"Microtus arvalis"*
Brucella inopinata	Humans
B. papioni	Baboons
B. vulpis	Red fox

***** = Preference host

- Most species of Brucella can infect animals other than their preferred hosts, when they come in close contact (Inter-species transmission).

Transmission of Brucella organisms from their preference host to a nonspecific host is of an epidemiological importance such as

transmission of *Brucella melitensis* from sheep to cattle in Mediterranean area including Egypt. In such condition, *Brucella melitensis* infection causes only sporadic abortion with localization of the organisms in the udder and shedding in the milk. Another example is the transmission of *Brucella suis* to cattle. This represents an epidemiological importance because *Brucella suis* is highly pathogenic causing severe serious disease in human beings and has a pyogenic activity.

Among the Brucella species known to cause disease in humans (*Brucella abortus, Brucella melitensis, Brucella canis, Brucella suis* and marine mammal brucella species), *Brucella melitensis* is thought to be the most virulent and causes the most severe and acute cases of brucellosis.

Sources of infection

1. Uterine discharges, fetal membranes, and aborted fetuses of aborted animals.
2. Even in the absence of abortion, profuse excretion of the organism occurs in the placenta, fetal fluids and vaginal discharges1×10^{13} organism /ml
3. Milk of infected animals. Colostrum after calving or parturition contains 2×10^{5} organism /ml.
4. Contaminated litters, feed stuffs, water, pastures and utensils.
5. Contaminated clothes, shoes and gloves of workers and attendants.
6. Semen of infected animals.
7. Latent infected heifers because of in-utero infection or ingestion of infected milk.
8. Other susceptible animals as dogs, cats, rats, insects as ticks, birds and wild life may act as reservoirs and play role in transmission.

Transmission

Brucellosis spreads in a herd by contamination of the environment by aborted fetuses, afterbirths and uterine secretions (lochia).

Infection occurs through:

1. Ingestion of contaminated food or water.
2. Mucous membranes.
3. Skin either intact or abraded.
4. Inhalation.
5. The genital tract by infected bulls but very rare due to the acidic pH of the mid cervix and bactericidal substances.
6. *Brucella ovis*, *Brucella suis* and *Brucella canis* are spread by venereal transmission.
7. Artificial insemination with infected semen. Infection occurs when

the semen is deposited in the uterus but not in the mid cervix.

8. Congenital transmission (in utero infection).

- Movement of the tail of recently aborted cow or following parturition usually spreads the infection among animals.
- Dogs and rats may spread the infection in farms through dealing with or feeding on aborted material.

Some of the means of perpetuating brucellosis in infected populations.

- Transmission of infection from dam to offspring before birth.
- Transmission of infection from dam to offspring via milk.
- Transmission of infection from male to female by sexual contact or A.I.
- Transmission of infection by direct physical contact particularly licking.
- Transmission of infection from contaminated environments to susceptible animals.
- Transmission of infection by eating of infected placenta and fetus by dogs and rats.

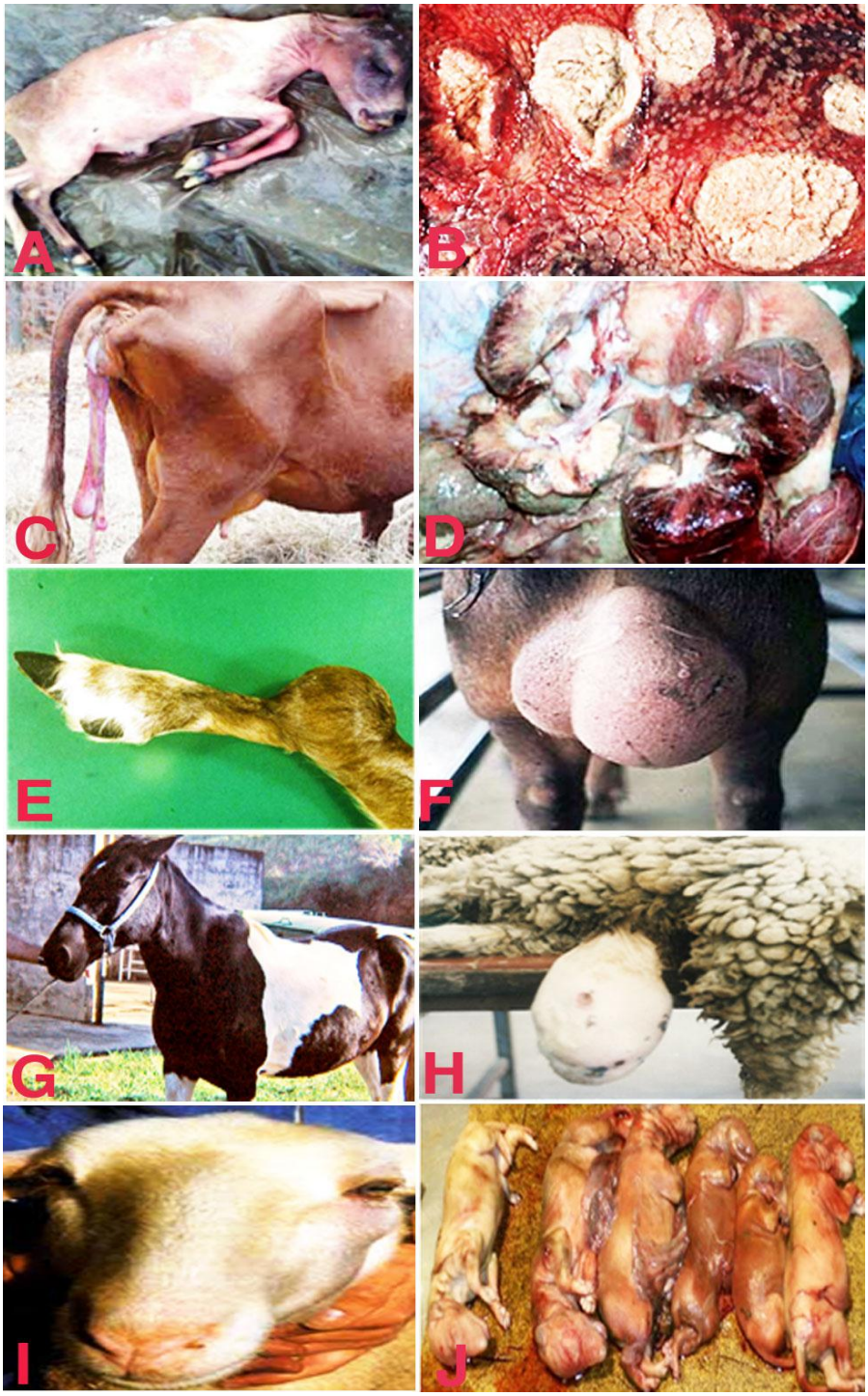
Pathogenesis

After infection, the organisms are phagocytosed by phagocytes in which they survive, multiply and reach to the regional lymph nodes then reach to the blood and spread via blood macrophages with localization in other lymphoid tissues such as spleen, iliac and mammary lymph nodes as well as joints. In non-pregnant cows localization occurs in the udder. When the uterus becomes gravid, it becomes infected from periodic bacteremic phases originating in the udder. Gravid uterus, placenta, and fetal fluids are predilection sites for brucellae due to secretion of erythritol; a sugar alcohol by the placenta and fetus which stimulates its growth. Invasion of the gravid uterus results in a severe ulcerative endometritis and the villi are destroyed. Abortion occurs at the last three months of pregnancy. After abortion and in non-pregnant cows the organisms persist in the udder and supra-mammary lymph nodes with occurrence of periodic bacteremia and interstitial mastitis with shedding of the organisms in milk. In bulls localization occurs in testicles with development of orchitis and epididymitis.

In the course of infection Brucella can enter and overcome the defenses of phagocytes and replicates within them. The infected phagocytes are a reservoir of infection which are protected from the general immune system response and probably inhibit antibiotic effectiveness. Such infected phagocytes can carry the disease to every organ in the body.

Incubation period depends upon the stage of fetal development and the time of infection. Brucella organisms are taken up by macrophages

but they prevent the fusion of the phagosome with the lysosome, protecting themself from the bactericidal actions of the lysosomal contents.



Clinical signs

Cattle

1. In highly susceptible non-vaccinated pregnant cattle, a storm of abortion occurs after the 5th month of pregnancy. In unprotected

herds 40% to 80% of pregnant females may abort or give birth to very weak newborns.

2. In subsequent pregnancies the fetus is usually carried to full term.
3. Retention of placenta and metritis which often causes infertility.
4. Some cows may die due to septic metritis.
5. Orchitis and epididymitis in bulls, one or both testes may be affected with acute painful swelling to twice normal size. Testis may undergo liquefaction and necrosis.
6. Lack of sexual activity and possibly infertility may occur.
7. Hygroma of the joints especially of the knees.

Sheep and goats

1. Abortion occurs in ewes at 4^{th}-5^{th}month of pregnancy.
2. Mastitis may occur in infected animals.
3. Infection in males may be followed by orchitis.

Horses

Clinically, horses suffer from inflammation in the supraspinous bursa (Fistulous wither) or supra-atlantal bursa (Poll evil). The bursal sac becomes distended by a clear, viscous, straw- colored exudate and develops a thickened wall. It can rupture, leading to secondary inflammation. Brucella-associated abortions are rare in horses.

Swine

Abortion can occur up to 80%, when abortions occur early in gestation, infected animals often go undetected. Temporary or permanent sterility is common and is sometimes the only sign. Boars can have unilateral or bilateral orchitis affecting their fertility. Other signs include lameness, posterior paralysis, metritis, and abscess formation in various locations of the body.

Dogs

Dogs abort in the last trimester of pregnancy (seventh to ninth week of gestation) and have prolonged vaginal discharge. Other clinical signs include stillbirths, early embryonic death, lymphadenitis, epididymitis, periorchitis, and prostatitis.

Post mortem lesions

1. Necrotizing placentitis.
2. Placenta is usually edematous.
3. Leathery plaques on the external surface of the chorion. The intercotyledonary region is typically leathery, with a wet appearance and focal thickening.
4. Necrosis of cotyledons.
5. Fetus is swollen and its cavities contain reddish fluid and sometimes covered with purulent material**.**

Diagnosis

1. Epizootiological situation of the disease.
2. Clinical signs and lesions.
3. Laboratory diagnosis.

Samples:

- Abomasal contents of the fetus, fetal lungs, spleen and lymph nodes.
- Uterine and vaginal discharges and placenta.
- Milk, colostrum and semen.
- Blood serum samples three weeks after abortion.
- Spleen, udder and supramammary lymph nodes from slaughtered cows.

a. Examination of direct smears from the suspected material (placenta and fetal stomach contents) stained by Gram's stain, modified ziehl Neelsen stain, Kazlovski stain or coster's stain.
b. Isolation of Brucellae from fetal stomach contents, fetal organs, udder, supramammary lymph node, spleen, milk and semen. It is performed on specific media as albimi-agar, tryptose soya agar or serum dextrose agar in the presence of 10% Co2.
c. Laboratory animal inoculation: It is a reliable method especially from contaminated samples. Guinea pigs or mice are inoculated S/C, I/M or I/P after two weeks blood serum is examined for antibodies. After four weeks and six weeks cultures are done from their spleens to isolate brucella organisms.
d. Serological examination including Plate agglutination test, tube agglutination test, Rose Bengal test, Buffered acidified plate antigen test, mercaptoethanol test, rivanol test, coombs test, CFT, FAT, ELISA, milk ring test, whey agglutination, semen agglutination test and Western blot technique.

- False positive reactions have been reported. Such serological cross reactions have been demonstrated between Brucella species and *E. coli* (O: 116, O 157), some salmonella serotypes, *Vibrio cholera* and *Yersinia enterocolitica* serotype (O: 9).

The use of multiple tests for diagnosis of brucellosis increases the confidence in the diagnosis and sequential tests over time provide a better insight than single test.

e. Detection of brucella antigens in tissues using immunostaining techniques e.g. immunoperoxadase staining technique.
f. DNA probe and PCR.

4. Allergic test (brucellin test) using rough strain of *Brucella abortus.* The test is used in sheep and goats where the antigen is injected intradermopalpebrally in the lower eye lid and read after 48 hours. It causes local swelling in sensitized animals.

Control

Control of brucellosis depends on the use of efficient diagnostic procedures for detection of infected animals.

Control of brucellosis based on:

1. Removal of the source of infection:

All cattle should be tested and those, which are positive, are sent to slaughter. This reduces exposure and transmission within the herd. Policy of test and slaughter is not practical in some countries due to the economic loss and the high incidence of the disease. It is also impossible to detect latent infections therefore the disease continues to spread without end.

2. Application of hygienic measures and management of outbreaks:
   a. Reactors should be isolated.
   b. Pastured animals should be stabled with proper application of hygienic measures.
   c. Destruction of infected material by burning.
   d. Proper disinfection of premises.
   e. Avoid contamination of food and water.
   f. Newborn calves should be separated, fed on brucella free milk, and checked four times between the 4^{th} month of life and the 5^{th} month of the first pregnancy.
   g. Control of rodents, insects, birds, and dogs in farms.
   h. Milk in infected farms should be pasteurized or used in manufacture of ghee.
   i. Control of movement of animals, workers, and attendants in infected farms.
3. Vaccination: Vaccines protect uninfected animals living in a contaminated environment.

It is certain that vaccination will remain a major aspect of the control of bovine brucellosis.

Different vaccines are used for immunization of animals:

a- ***Brucella abortus strain 19*** **(calf hood vaccine):** It is a living attenuated (by its nature), smooth *Brucella abortus* strain of low virulence. It is used for female calves in its reduced dose (5-8 x 10^{10})

SlC between 3-6 months of age. A reduced dose of from 3×10^8 - 3×10^9 organisms can be administered SlC. Serum agglutination test returns negative by the time the animals are of breeding age except in 6% of cases. The proportion of animals showing persistent post vaccinal serum agglutinins increases with increasing age of vaccinates.

- Vaccination of adult cattle has been carried out in some countries but it is not suitable for eradication programs. However, it may be of value in reducing the rate of abortion.
- Vaccination of bulls causes orchitis and excretion of the organism in semen.
- Vaccination of cows at late pregnancy causes abortion.
- It can also cause undulant fever in man.
- *Brucella abortus* S.19 is secreted in milk of vaccinated cows.
- The vaccine confers adequate immunity against abortion for five or more subsequent lactations under conditions of field exposure.
- S.19 vaccine was also used by conjunctival route5×10^9in adult cattle with the same protection as subcutaneous route.
- The complement fixation test becomes negative sooner than the standard tube agglutination test following vaccination. This can be used to distinguish post vaccine titers from naturally infected ones.
- A western blot technique is able to distinguish the serological reaction of calf hood vaccination from infected cattle.
- Systemic reactions to vaccination with S19 have been reported rarely in both calves and adults in the form of fever, anorexia, drop in milk production and a local swelling.

b. ***Brucella abortus* strain RB51 vaccine:** It is a live stable rough mutant of *Brucella abortus* strain 2308 which lacks much of lipopolysaccharide O-side chain. It is prepared by serial passage on media containing rifampicin and penicillin. It does not induce positive results in the standard tube agglutination test. Heifer calves are vaccinated at 3-7 months of age subcutaneously. They are protected when challenged against infection during the first pregnancy. A dose of injected organisms are10 billion cells $1–3.4 \times 10^{10}$per calf and one billion cells$1–3 \times 10^9$ per adult animal.

- Field experience also indicates that it can induce abortion in some cases if applied to pregnant cattle.
- RB51 is considered infectious for humans (vaccine strain is highly resistant to rifampicin; one of the antibiotics of choice for treating human brucellosis).

c. ***Brucella abortus strain 45/20*, Duphavac, McCewen vaccine:** It is a rough non agglutinogenic killed *Brucella abortus* vaccine used in cattle, sheep and goats. It is given in two doses as I/M or S/C. The

first dose is taken at six months of age and the second dose one month later. This vaccine has no agglutinogenic activity but produces CFT antibodies. It does not cause abortion. It gives about 70% protection with a short duration.

d. ***Brucella melitensis* Rev.I vaccine:** It is a living attenuated *Brucella meletensis* strain used for vaccination of sheep and goats. Vaccination of lambs 3-6 months of age with 0.5 - 2.0 x 10^9 viable organisms SIC confers a high degree of immunity which lasts for four years in goats and 2.5 years in sheep.

- It can be administered conjunctivally.
- It induces strong interferences in serological tests
- Vaccination of sheep and goats at late pregnancy causes abortion.

e.***Brucella melitensis* H38 Vaccine:** It is a formalin killed adjuvant vaccine prepared from *Brucella meletensis* strain. It confers less immunity than Rev.I vaccine and produces local reaction and prolonged allergic and serological responses.

Brucella ovis infection
Ram epididymitis

It is an infectious disease of sheep characterized by epididymitis and infertility in rams, abortion in ewes and neonatal mortality in lambs.

Epididymitis due to *Brucella ovis* infection is considered the most important cause of infectious reproductive disease in sheep that is associated with subfertility and infertility.

Etiology

The disease is caused by *Brucella ovis*, which is a rough brucella organism that shares many antigenic and other characteristics of genus brucella. *Brucella ovis* preferentially infects sheep.

Epizootiology

Geographical distribution

Brucella ovis infection is a worldwide disease.

Susceptibility

Only sheep are affected. Rams are more susceptible to infection than ewes.

Sources of infection

Infected rams excrete the organism in semen.

The organism is also present in the placenta, vaginal discharges and milk of infected ewes.

Transmission

- Passive venereal transmission occurs from ewes that have been bred by an infected ram in the same heat cycle.
- Infection also occurs between rams when they lick each other's prepuce.

Pathogenesis

Infection with *Brucella ovis* results in localization and inflammation in the epididymis that cause sperm stasis and infertility. In ewes, placentitis occurs causing fetal death and more commonly producing lambs of low birth weight and poor viability.

Clinical signs

1. Acute edema and inflammation of the scrotum may occur and may be associated with systemic reaction.
2. After a long period there are palpable lesions in the epididymis and tunicae of one or both testicles. The epididymis is enlarged and hard, more commonly at the tail.
3. The scrotal tunicae are thickened and hardened and the testicles are usually atrophic.
4. Palpable lesions may be present in less than 50% of serologically positive rams.
5. In ewes abortion may occur or the birth of weak or stillborn lambs.

Diagnosis

1. Epizootiological situation of the disease
2. Clinical signs and lesions
3. Laboratory diagnosis

Samples: Semen, blood serum, epididymis and seminal vesicle should be collected.

a. Isolation of *Brucella ovis* from semen but it is fastidious in its growth. It needs 20% CO_2 tension.
b. The serological tests used routinely to diagnose brucella infection do not detect antibodies to B. ovis. These tests use "smooth phase" antigens. CFT, ELISA, immunodiffusion in agar gel (AGID)and immunoblotting are used.
c. Allergic test.

Control

1. Detection and culling of infected rams
2. Vaccination using killed *Brucella ovis* vaccine or a combined vaccine containing killed *Brucella ovis* and *Brucella abortus* S19 that is more effective but vaccinated animals become seropositive and *S19* may cause epididymitis with excretion of S19 in semen.

3. *Brucella melitensis* Rev.1 vaccine was found to be more effective.

Campylobacteriosis

It is an infectious disease of cattle and sheep characterized by abortion and infertility. In cattle the disease is typically venereal disease.

Etiology

The disease is caused by campylobacter spp. compylobacters are Gram-negative, non sporing curved or spiral rods. They are

microaerophilic and grow in atmosphere of 6% oxygen, 10% carbon dioxide and 84 % hydrogen or nitrogen.

- Cattle are infected with *Campylobacter fetus subspecies venerialis* which is present in the bovine reproductive tract. *Campylobacter foetus intermedius* also affects cattle.
- Sheep are infected with *Campylobacter fetus subspecies fetus* (intestinalis) which present in the intestinal tract of sheep and cattle, this type causes enzootic abortion in sheep, sporadic abortion in cattle and occasionally septicemic disease in humans.
- The organism is not resistant to adverse conditions; it is rapidly destroyed by drying, light and heat. It can survive in manure, soil and hay for about 10 days at room temperature.

Epizootiology

Geographical distribution

The disease was reported in many countries such as USA, Canada, Britain, Denmark and South Africa.

Susceptibility

Cattle and sheep are affected. Young animals are less susceptible than older one.

Sources of Infection

1. The organism is present in the mucosa of the glans penis, distal portion of the urethra and prepuce of bulls, which is readily discharged at service.
2. In heifers and cows the organism is present in the lumen of vagina, cervix, uterus and oviducts. It persists for long periods of months in the anterior end of the vagina.
3. Contaminated semen-collecting apparatus.
4. Semen of infected bulls.
5. In sheep the organism is present in the intestine and excreted in feces.

Transmission

Cattle

1. Transmission occurs at sexual intercourse.
2. Bull is infected through coitus of an infected cow or through an infected artificial vagina at the artificial insemination centers.
3. Cows are infected by an infected bull or through infected semen during artificial insemination.

- Introduction of an infected bull or cow causes rapid spread of the disease among the herd.

Sheep

Infection occurs orally.

Pathogenesis

Following exposure of cow to infection the organism traverses the cervix and establishes itself in the uterus few days later. This results in endometritis and salpingitis and penetration of the organism into the epilhelium of the reproductive tract. The organism is expelled from the oviducts, uterus and cervix but it persists for months in the anterior end of the vagina. Infection during pregnancy causes placentitis and the cotyledons separate from the caruncles.

In bulls the organism lives and multiplies in the prepuce, macosa of glans penis and the distal portion of the urethra. Sometimes no signs appear in infected bulls but the organism persists for years.

In sheep, infection occurs orally and then the organism passes to the uterus.

Clinical signs

Cattle

1. Vaginitis, cervicitis and endometritis are the early signs which develop immediately after infection.
2. Increase in the number of cows returning to the bull and abnormal long and irregular heat periods are the most characteristic features.
3. Abortion may occur at any period of gestation but most commonly occurs during 4 – 7 months of pregnancy.
4. Bulls usually show no clinical signs.

Sheep

1. Abortion usually occurs during the late pregnancy.
2. Lambs may be carried to full term but are born dead or in a weak condition.
3. Placentitis is mild with hemorrhagic cotyledons and an edematous intercotyledonary area.

Postmortem lesions

1. Placentitis with hemorrhagic necrotic cotyledons and edematous or leathery intercotyledonary areas.
2. The fetus is usually autolysed, with orange-yellow necrotic foci (1–2 cm diameter) in the liver.
3. Fetuses may have accumulated serosanguineous fluid in the thoracic and peritoneal cavities.

Diagnosis

1. Epizootiological situation of the disease.
2. Clinical signs.
3. Laboratory diagnosis.

Samples

- Vaginal mucus at estrus for bacteriological examination.
- Vaginal mucous taken between heats for mucus agglutination test.
- Vaginal discharge from recently aborted animals for bacteriological examination.
- Aborted fetus (stomach contents) and fetal membranes.
- Preputial scrapings or washing from bulls especially those aged four years or more.

a. Microscopical examination of smears from semen, preputial washings cotyledons, abomasal contents of aborted fetuses stained by Gram's Method.
b. Culturing of cotyledons, fetal stomach contents, liver, vaginal discharge, semen and preputial washing.
c. Vaginal mucus agglutination test: Agglutinins appear as early as 14 days of exposure and last for months.

4. Test – mating of heifers: Maiden heifers (12- 18 months of age) are inseminated with semen to which preputial washing has been added.

Control

1. Application of artificial insemination.
2. Detection of the source of infection and regular application of mucus agglutination test as a herd test.
3. Cows are treated by intrauterine infusion of one gram streptomycin and 300,000 I.U. penicillin in 60 ml. of water for 3 times at 24 hours intervals.
4. The infected bull may be treated with Streptomycin injection combined with oil-based Streptomycin applied locally to the penis. This treatment must be carried out on 3 consecutive days.
5. Cows are given sexual rest for 3 months.
6. Application of streptomycin solution into the anterior chamber of the vagina during estrus followed by mating with the bull.
7. The use of vaccines. Formalin killed vaccines have been used effectively in many countries
8. In sheep hygienic measures should be taken to prevent spread of infection from fetal membranes and aborted fetuses and contamination of food, water, and pasture.

Salmonella abortion in ewes

It is an infectious disease of sheep characterized by abortion. It is usually associated with stress conditions, contaminated water supply, and introduction of infected sheep.

Etiology

The disease is caused by *Salmonella abortus ovis* which is a host

adapted organism. *Salmonella montevideo, Salmonella typhimurum* and *Salmonella dublin also cause abortion in ewes.*

Epizootiology

Geographical distribution

The disease is of worldwide distribution especially when they are under stress.

Susceptibility

Sheep are commonly affected.

Sources of infection

These include:

Salmonella abortus ovis: Feces, vaginal discharges and aborted material, carriers and reservoirs.

Other Salmonellae: Contaminated feedstuffs and water courses, sewage effluent overflow, carrier cattle, carrion and wild birds and rodents.

Transmission

Infection occurs either by ingestion or by venereal transmission through infected rams.

Clinical signs

***Salmonella abortus ovis* infection**

1. Systemic and enteric signs (fever, anorexia, depression and diarrhea).
2. Abortion storm with 10% of ewes aborting at the last six weeks of gestation.
3. Post parturient septic metritis.
4. Lambs in contact with aborted ewes usually scouring.
5. Lambs may also be stillborn or die within a few hours of birth from septicemia. Occasionally, lambs appear to be healthy but die within 3 weeks.
6. Sheep may simply be found dead with rotten lambs still present in the womb.

***Salmonella montevideo* infection**

When infection with *S. montevideo* occurs at 12-14 weeks of gestation, abortion occurs two to three weeks later but not associated with any other signs.

***Salmonella typhimurium* infection**

It causes enteric and systemic signs and abortion. Infected ewe may die before abortion.

***Salmonella dublin* infection**

It is very similar to *S. abortus ovis* and characterized by fever, diarrhea and abortion.

Post mortem lesions

1.No typical characteristic macroscopic lesions in aborted fetus and

placenta.
2.Gall bladder is distended.
3.Liver is swollen and friable.
4.Liver of fetus my show necrotic foci (*S. abortus ovis*).
5.Inflammation of the intestine and mesenteric lymph nodes.

Diagnosis

1.Epizootiological situation of the disease.
2.Clinical signs and lesions.
3.Laboratory diagnosis:

Samples: Aborted material, uterine discharges, gall bladder, mesenteric lymph nodes and blood serum should be collected.

a. Isolation of the organism. *Salmonella abortus ovis* will grow on MacConkey, desoxycholate citrate (DCA), or Salmonella–Shigella agar.
b. Detection of Gram - ve bacilli from aborted material and the gall bladder.
c. PCR.
d. A strong positive agglutination test.

Control

1. Avoid contamination of food and water.
2. Proper disposal of aborted material and proper disinfection.
3. Control of rodents.
4. Detection and elimination of carriers.
5. The use of broad-spectrum antibiotics for one week during outbreaks. Whole group long-acting oxytetracycline injections (20 mg/kg) may reduce the number of abortions during an outbreak of salmonellosis in sheep.

- Non steroid anti-inflammatory should be used combined with antibiotics to reduce the risk of endotoxic shock.

6. The use of autogenous dead vaccine.
7. Avoid introducing infected animals.

Leptospirosis

Weil's syndrome, Hemorrhagic Jaundice, Mud Fever, Canicola Fever, Cane cutter's disease, Rice field fever.

It is a contagious, waterborne disease of animals and man characterized by fever, icterus, hemoglobinuria, bloody milk, hepatic and renal disorders, abortion and death in calves.

The The disease was first described in humans by Adolf Weil in 1886 when he reported an acute infectious disease with enlargement of spleen, jaundice, and nephritis. *Leptospira* was first observed in 1907 from a post mortem renal tissue slice.

Etiology

The disease is caused by different types of Leptospires. They belong to the order of Spirochaetales, family Leptospiraceae, genus Leptospira. Leptospires are bacteria which can be either pathogenic, i.e. having the potential to cause disease in animals and humans or saprophytic, i.e. free living and generally considered not to cause disease.

- Pathogenic Leptospires belong to a single species *Leptospira interrogans,* which contains more than 250 serovars.
- Leptospires are saprophytic aquatic spirochetes.
- They are spiral organisms that have a hook at one or both ends 0.2-0.3 um to 20-30 um.
- They require special media (10% rabbit serum and bovine serum albumin) and special stains (silver impregnation technique or Fontana method). They can be demonstrated by dark ground microscope.
- The most common serovars are:

L. pomona: Cattle, horses and sheep.
L. hardjo: Cattle and buffaloes
L. gripotyphosa: Cattle, buffaloes and swine.
L. canicola: Cattle, buffaloes, swine and dogs.
L. ictero haemorrhagica: Dogs, horses, cattle, buffaloes and swine.

Cattle are considered the maintenance host for *L. hardjo.*

Epizootiology

Geographical distribution

It is a worldwide disease occurs in tropical, subtropical and temperate zones.

Susceptibility

Cattle, buffaloes, sheep, horses, swine, dogs and camels are susceptible.

The incidence of clinical disease is much higher in young animals (septicemic form) than in adults.

- There are two main categories for serovars and species susceptibility; host adapted and non-host adapted leptospirosis. Animals which are infected with a host adapted serovar are the maintenance or reservoir host.

Leptospira serovar	**Maintenance host**

hardjo-bovis	Cattle
Pomona	Pigs andskunks
Canicola	Dogs
icterohaemorrhagica	Rats, other rodents
grippotyphosa	Raccoons and skunks

Sources of infection

1. Urine, aborted material, semen, and mud.
2. Pasture, drinking water, and feed contaminated by infected animals.
3. Recovered cattle may discharge leptospires for 180 days and in case of *L. hardjo* for 280 days, dogs for 700 days and pigs for all their life. In recent studies carrier status allowing shedding of leptospires to continue for up to two years in cattle.
4. Rodents and dogs play an important role in transmission of the disease.
   - Leptospires colonize the kidneys and reproductive tract of infected animals and cause the bacteria to be shed in urine and reproductive discharges.
   - The types of habitats most likely to carry infective bacteria are muddy riverbanks, ditches, gullies, and muddy livestock rearing areas.
   - There is a direct correlation between the amount of rainfall and the incidence of leptospirosis, making it seasonal in temperate climates and year-round in tropical climates. Water contaminated by urine from animal reservoirs is the main source of human infection.

Transmission

Infection occurs through:

1. Mucosal abrasions, mucous membranes and cutaneous abrasions.
2. Ingestion of contaminated food, or water.
3. Inhalation of urine droplets
4. Coitus and artificial insemination.
5. Transplacental transmission may occur.

Generally warm humid climate with abundance of surface water and alkaline soil act as predisposing factors for infection.

Risk factors:

• Introduction of infected carriers particularly bulls.

• Access to watercourses, especially where there is a risk of contamination by urine.

• Co-grazing with sheep, as sheep are significant carriers of leptospires.

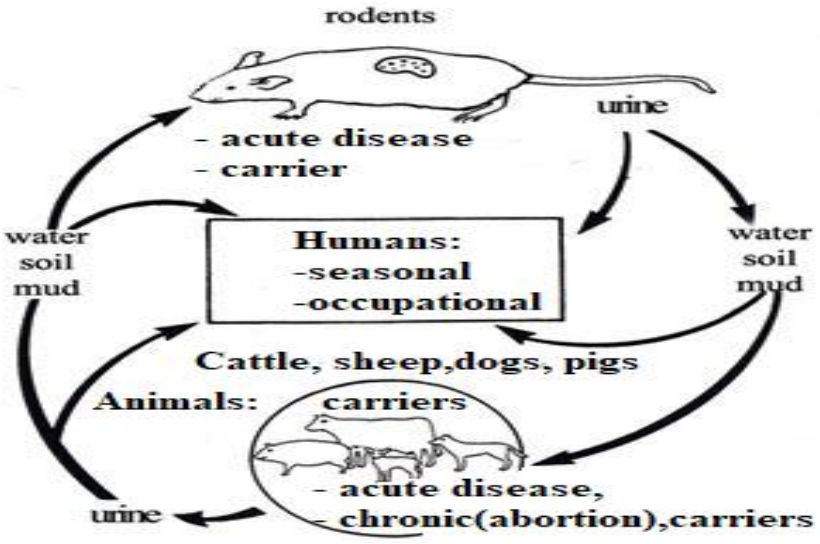


Figure (23): Transmission cycle of Leptospirosis

Pathogenesis

Leptospires invade the host mostly across mucosal surfaces or softened skin followed by hematogenous dissemination (leptospiremia). Leptospires are then found extracellularly between cells of the liver and kidney. Endotoxic activity of leptospires results in release of lymphokines such as tumor necrosis factor (TNF-alpha) from monocytes, this causes damage to endothelial cells with resultant hemorrhage. Leptospires cause local damage to blood vessels, and so the early results are hemorrhages, kidney damage (nephritis) leading to blood in the urine. Release of hemolysin causes intravascular hemolysis. Hemoglobinuria and icterus develop. Degenerative changes occur in the kidney and liver resulting in interstitial nephritis and hepatitis. Following to septicemia, localization in other organs as uterus, meninges and eye may occur. Invasion of the uterus results in fetal death and abortion. In the acute phase of the disease the animal may die from septicemia and hemolytic anemia. The milk from all four teats would be red in color, thick in consistency and it contains blood clots but with flaccidity of the udder and with no signs of inflammation.

The primary lesion is damaged to the endothelium of small blood vessels leading to localized ischemia in organs, resulting in renal tubular necrosis, hepatocellular and pulmonary damage, meningitis and placentitis.

- *L. pomona* causes interstitial nephritis and intravascular hemolysis causing hemolytic anemia.
- *L. hardjo* does not produce hemolysin and does not cause interstitial nephritis but it causes septicemia, abortion and mastitis.

In **horses** periodic ophthalmia 'moon blindness' occurs. The strain of leptospira that is most commonly incriminated is *Leptospira interrogans* serovar pomona. Agglutinins are present in the aqueous humor in greater concentration than in the serum. Opacity develops in the cornea and lens due to the antigenic relationship between leptospires and equine ocular tissues. This impedes the nutrition of ocular structures producing iris atrophy and corneal opacity and recurrent uveitis.

Clinical signs

Acute septicemic form

Calves under two months are most susceptible but adult dairy cattle may be affected. This form is characterized by:

1. Fever, anorexia and petechial hemorrhage on mucosae.
2. Hemolytic anemia with hemoglobinuria and jaundice.
3. Lameness due to synovitis.
4. High fatality rate (10-15%) among calves.
5. Milk becomes thick in consistency and tinged with blood for few days.
6. The udder appears flaccid without cardinal signs of inflammation but increase of leukocytes and change of milk color suggest mastitis.
7. Some animals may show arched back due to pain in lumbar region.
8. Meningitis may occur manifested by incoordination and excessive salivation.

Chronic phase

1. Storm of abortion usually occurs at the last third of pregnancy, three months or longer after the acute phase.
2. Neonatal mortality or birth of weak calf (weak calf syndrome) also occurs.

***L. hardjo* infection is characterized by:**

1. Sudden onset of fever.
2. Abortion 3-12 weeks following infection.
3. Milk is thick yellow to orange and contains blood clots.
4. Agalactia“milk drop syndrome”.
5. Udder is flaccid and flabby “flabby bag udder”.

Postmortem lesions

1. Icterus and submucosal hemorrhage.
2. Ulcers in the abomasal mucosa.
3. Liver is swollen and kidney is dark and swollen.
4. In chronic cases there are small white raised areas in renal cortex.
5. Aborted fetus is autolysed and fetal membranes are thick and edematous.

6. The leptospires cause a diffuse placentitis with avascular, light tan cotyledons and edematous yellowish intercotyledonary areas.

Diagnosis

1.Epizootiological situation of the disease.
2.Clinical signs and lesions.
3.Laboratory diagnosis.

Samples:

Living animal:

- Two urine samples to the first one, one drop of formalin / 20 ml of urine is added, the second is kept on ice and used for isolation of the organism.
- Blood and milk samples should be collected.
- Aborted material should be collected in cases of abortion.

Dead animals: Urine, heart blood, kidney, liver, and spleen are collected.

- Samples should be preserved because in unpreserved samples leptospires die quickly as they are highly sensitive to acidic media produced by multiplication of other bacteria after death.
- Direct inoculation of Guinea pigs at the same time of sample collection in the field is the best reliable and practical method.

a. Dark field microscopy of urine samples.
b. Isolation of the organism is laborious, expensive and slow.
c. Guinea pig inoculation I/P using blood, urine, or milk samples with periodical culture of blood or urine and examination of the kidneys for leptospires.
d. Serological examination:

- Microscopic agglutination test (considered the gold standard in diagnosing leptospirosis).
- CFT.
- FAT for urine sample is an accurate test.
- ELISA.

e. The use of PCR on body fluids and tissues.
f. Detection of leptospirae in fixed tissue using silver impregnation technique.
g. Histopathololgy reveals interstitial nephritis and centrilobular hepatic necrosis.

Differential diagnosis

The disease should be differentiated from:

1.Hemolytic diseases, such as babesiosis, bacillary hemglobinuria, post parturient hemoglobinuria, Rape and Kale poisoning, acute

hemolytic anemia of calves and chronic copper poisoning.
2.Hemorrhagic mastitis, but in leptospirosis there are no cardinal signs of udder inflammation.
3.Diseases causing abortion.

Treatment

1. Dihydrostreptomycin 12 mg/kg bw, I.M. twice daily for 3-5 days or 25 mg/kg bw daily is effective.
2. Blood transfusion
3. Hematinics and tonics.
4. In horses the two main drugs used in the treatment of uveitis are atropine and corticosteroids. Atropine works by paralyzing some of the muscles of the iris, thus stopping the painful spasm and allowing the pupil to dilate.

Control

1. Detection and elimination of carriers.
2. During outbreaks of leptospirosis, animals having a rise in body temperature should be injected I.M with dihydrostreptomycin, 25 mg/kg bw.
3. Proper disposal of carcasses and aborted material.
4. Proper disinfection of the farm using acidic disinfectants.
5. Restriction of the use of liquid and solid manure.
6. Drainage or fencing of the areas of stagnant water.
7. Control of rodents and pets.
8. Vaccination using formalized killed bacterins containing one or more than one serotype. Calves are vaccinated at 4-6 months of age with two doses one month apart then after 6 months and revaccinated annually.

Listeriosis
Circling disease, Silage sickness

It is an infectious disease of domestic animals characterized by septicemia, abortion and meningo-encephalitis.

Etiology

The disease is caused by *Listeria monocytogenes* (different serotypes) and *Listeria ivanovi*.

- They are intracellular, Gram-positive coccobacilli or short bacilli, grow aerobically but facultatively anaerobic.
- It can grow well under reduced oxygen and increased CO_2 tension at 4ºC to 45ºC and the optimum temperature is 30-37ºC.
- The pH range is 5-9(can tolerate a pH from 3.6 to 9.5).
- A pH of greater than 5 (e.g. spoiled silage) favors the growth of this organism.
- They are resistant to high salt concentration (can tolerate sodium chloride content of 20%).
- They can survive in the environment for long periods, five years or more and persist in feces for months.
- *Listeria monocytogenes* is susceptible to 1% sodium hypochlorite and 70% ethanol.
- *Listeria monocytogenes* is associated with septicemia, abortion and encephalitis while *Listeria ivanovi* is associated with abortion only.

Epizootiology

Geographical distribution

The disease is more prevalent in cold areas as North America, New Zealand, UK and Japan.

Susceptibility:

Sheep, goats, cattle, horses, pigs, dogs, cats, rabbits, some wild animals and humans are susceptible to infection.

Intercurrent diseases, pregnancy, climatic stress and viral damage of mucosal surfaces are important predisposing factors.

Sources of infection

1. Diseased animals through aborted material, feces, and milk.
2. Soil and bad silage.
3. Wild rodents and other carriers.

- The reservoirs of infection are the soil and the intestinal tracts of asymptomatic animals, including wild and feral mammals, birds, fish and crustaceans.

Transmission

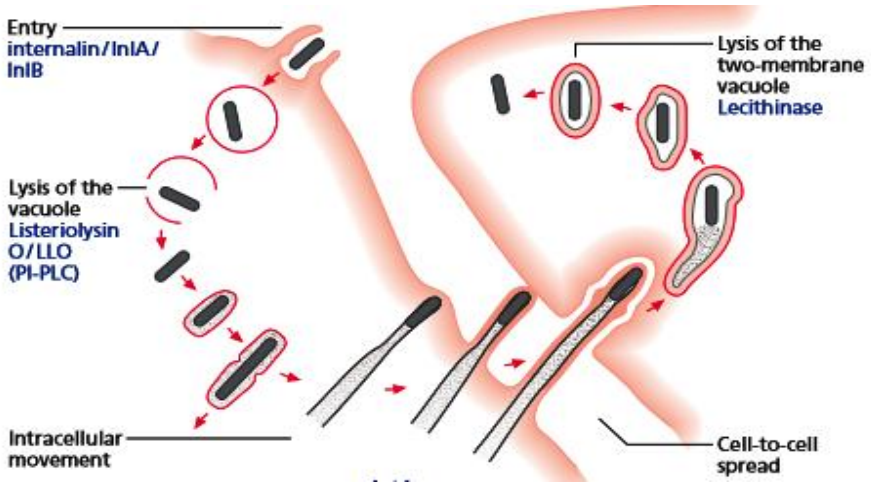
1. Ingestion of contaminated food is associated with septicemic and abortion forms.
2. Newborn animals can be infected through ingestion of infected milk.
3. Breaks in buccal mucosa, losing teeth or contamination of the conjunctiva may be associated with the encephalitic form. Infection occurs through the peripheral branches of trigeminal nerve.
4. Congenital transmission is a possible way.
5. Venereal transmission may occur.

- Silage (poor quality silage) of a pH above 5 provides suitable media for growth of Listeria and molds. (Good Silage is characterized by anaerobic storage, high density, a high concentration of organic acids and a pH below 4.5).
- Experimental dosing of pregnant ewes, results in abortion six to ten days post dosing.
- Natural infection of pregnant ewes, results in abortion seven days post silage intake.
- Listeriosis is most prevalent during spring and winter seasons. In the northern hemispheres, listeriosis has a distinct seasonal occurrence, probably associated with seasonal feeding of silage, with the highest prevalence in winter.

Pathogenesis

Septicemic form occurs in young ruminants and mono-gastric animals. Meningo-encephalitic form is common in adult ruminants. Abortion occurs in all mammals.

After ingestion, Listeria organisms probably penetrate the M-cells in Payer's patches in the intestine. Spread, occurs via lymph and blood to various tissues. *L. monocytogenes* has the ability to invade both phagocytic and non-phagocytic cells, to survive and replicate intracellulary and transfer from cell to cell without exposure to humoral defense mechanism. After multiplication in ileal cells and Peyer's, they reach to the blood resulting in septicemia with localization in different organs especially the liver, uterus and udder. In pregnant animals, organisms localize in placentomes and reach to the amniotic fluid, the fetus aspirates the organism causing fetal death and abortion. Localization in the udder also causes mastitis. Listeria organisms may pass through breaks in buccal mucosa or conjunctival contamination and ascend the trigeminal nerve to reach the brain resulting in micro abscesses and development of the encephalitic form. Inapparent infection with prolonged fecal excretion of the organism also occurs.



Clinical signs

It is unusual for all the forms of the disease to occur at one time in a flock of sheep due to difference in incubation period for each form. Abortion will be followed at later date by the encephalitic form.

A. Abortion

This form is characterized by:

1. The rate of abortion is low but may reach up to 15% and may be repeated each year in the flock or the farm. It occurs at any time after three months of pregnancy in sheep and four to seven months in cattle.
2. Fever and abortion may be the only signs of the disease.
3. Metritis and retention of fetal membranes for two to three days then shed without help.
4. Mastitis.

B. Septicemic Listeriosis

This form is common in lambs and calves. The onset is fast, and is characterized by:

1. Fever, dullness, and depression.
2. Sudden death may occur.
3. Profuse diarrhea followed by death may occur.
4. Calves and lambs may show corneal opacity and blindness and death occurs within 12 hours.

C. Encephalitic form

It is the most common form.

1. Affected animals are febrile at the beginning but the temperature falls to normal before the clinical cases are examined.
2. The animal move in single direction with the head turned or tilted.

3. Unilateral facial paralysis of the same side.
4. Drooling of saliva with food hanging from the mouth or

accumulates in the buccal cavity.

5.Paralysis of the lower jaw.
6.Protrusion of the tongue and dropping of ears.
7.The animal moves in a circle in one direction. The circle becomes narrower and narrower until the animal is practically turning around its own axis.
8.Finally the animal becomes recumbent and unable to rise followed by death.

- Death may occur 24 to 48 hours after onset of clinical signs.

Postmortem lesions

1.Gray white foci of necrosis in the liver and sometimes the lung, spleen or other organs (0.5–1 mm in diameter).
2.The aborted fetus is edematous and may be slightly or significantly autolyzed.
3.Necrosis of cotyledons and the intercotyledonary areas, and the fetus is usually autolyzed.
4.Placenta is thickened and leathery.
5.In encephalitic form the CSF is cloudy due to increased globulin and leukocytes.

Diagnosis

1.Epizootiological situation of the disease.
2.Clinical signs and lesions.
3.Laboratory diagnosis.

Samples include: abomasal contents, liver and spleen of the aborted fetus, placenta, uterine discharge, blood, milk, brain stem, cerebrospinal fluid, feces, and silage.

a. Direct examination of smears from infected tissues or fluids stained by Gram's Stain
b. Isolation of the organism on sheep blood agar. Cold enrichment, 5-10 % CO2 and 15% NaCl in media are necessary.
c. Animal inoculation; such as mice, G. pigs and rabbits. Inoculation of adult rabbits results in high temperature and CNS disturbance while young rabbits show monocytosis.
d. DNA hybridization and PCR.
e. Histopathological examination of the brain for detection of micro abscesses particularly in the mid brain.

- Serological examination is not reliable due to cross-reactions with *Staph. aureus*, *Enterococcus feacalis* and *Actinomyces*

pyogenes as well as due to the presence of positive titers in apparently normal animals.

Differential diagnosis

Sheep: the disease is confused with:

1. Diseases causing abortion.
2. Diseases causing nervous signs as Rabies, Enterotoxemia, Toxoplasmosis, Coenurosis*****, brain abscess and pregnancy toxemia.

Cattle: the disease is confused with:

1. Diseases causing abortion.
2. Diseases causing nervous signs as Rabies, Enterotoxemia, BSE, MHC, Ketosis and acute lead poisoning.

***** ***Coenurosis***

Coenurus cerebralis *is the cystic larval stage of T. multiceps, a tapeworm of dogs and other wild carnivores. The coenurus develops in the brain of sheep. Egg-filled segments of T. multiceps are passed in the feces of dogs. When sheep ingest the eggs, the embryos migrate via the circulatory system to the brain and spinal cord where the coenurus develops. The pathogenic effect is the result of pressure applied to the brain by the cyst during its development. The clinical signs include uncoordinated movements of the legs and abnormal positioning of the head. Affected animals may become blind in one or both eyes and indifferent to food and water. This can result in emaciation and eventual death. Clinical diagnosis of coenurosis is difficult. No specific treatment is available for this infection and slaughter of the animal is usually recommended. Prevention of the disease includes the treatment of dogs with taenicidal drugs and the education of farmers and butchers so that offal and condemned material should not fed to dogs after slaughtering a parasitized animal.*

Control

1. Isolation of affected and suspected cases.
2. Proper disposal of aborted material.
3. Proper disposal of infected carcasses.
4. Proper disinfection.
5. Production of good silage and avoidance of feeding of animals on poor quality silage.
6. Eradication of rodents.
7. Prophylactic use of antibiotics.
8. Treatment of infected cases: Early treatment is recommended. In severe cases especially when encephalitis develop, the response for treatment is very poor.

Septicemic cases and cases of abortions can be treated using sodium penicillin I/V as 40,000 i.u/kg bw., every six hours for one to two weeks followed by procaine penicillin I/M 44,000 IU/kg bw., twice daily for seven days.

- *Vaccination is impractical due to the sporadic occurrence of the disease in enzootic areas.*

Chlamydophilosis
Chlamydial abortion
Enzootic chlamydial abortion

It is an infectious disease of sheep characterized by abortion and stillborn lambs.

Etiology

The disease is caused by *Chlamydiophilia abortus* (*Chlamydia psittaci* immunotype 1).

It is characterized by development of two phases: The extracellular infectious phase known as the elementary body and the intracellular replicative phase which is non-infectious.

Epizootiology

Geographical distribution

The disease was reported in many European countries, North America and Middle East.

Susceptibility

The organism affects many free living and domestic birds, mammals and also man.

- Chlamydiosis can cause abortion in pregnant women who handle sick sheep or lambs.

Source of infection

Ewes which abort or deliver stillborn or weak lambs are the main source of infection. The organism is shed in fetal membranes and uterine discharges.

Transmission

Infection occurs by the oral route. Ewes are infected when they graze in areas contaminated by infected aborted material of lambing ewes.

- Infection of 30 -120 days pregnant ewes may cause abortion while infection of non-pregnant or pregnant ewes greater than 120 days may cause abortion the next year.
- It is usually a problem in 2ndlambers and kidders.
- Lambs and kids born to infected ewes become infected at birth and may abort the next year.
- It takes about 5-6 weeks after infection for abortion to occur.

Pathogenesis

After oral infection the tonsils are involved as a primary focus. The infection remains inapparent and usually does not affect the current pregnancy. In the subsequent pregnancy, placentitis and abortion occur.

Clinical signs

1. Lambs are sometimes born dead 2 – 3 weeks before the date of lambing.
2. Abortion, premature, full term deliveries of stillborn or weak lambs are features of the disease.
3. Aborted ewes pass discolored uterine discharge for several days.
4. Ewes and does do not exhibit signs prior to aborting.

Diagnosis

1. Epizootiological situation of the disease.
2. Clinical signs.
3. Laboratory diagnosis.

Samples: Aborted fetuses, fetal membranes and blood serum samples should be collected.

a. Examination of a modified Ziehl neelsen stained smears from cotyledons or chorion for demonstration of large numbers of elementary bodies as red bodies against a blue background.
b. Examination of Giemsa stained tissue sections for demonstration of the intracellular chlamydial inclusions.
c. The use of immunoperoxidase method for examination of tissue sections.
d. Serological examination using CFT.
e. Isolation of the organism in chick embryo yolk sacs or on cell culture monolayers.
f. PCR.

Control

1. Isolation of aborted ewes.
2. Proper disposal of aborted fetuses, fetal membranes, dead lambs, and contaminated bedding.
3. Cleaning and disinfection of lambing pens.
4. The use of long acting oxytetracycline, 20 mg/kg body weight, two doses at two weeks interval. This will reduce the multiplication of the organism.
5. The use of vaccines before breeding.

Mycotic Abortion

It is an infectious disease causing abortion following to systemic mycosis. Transient systemic infection is usually followed by localization in the pregnant uterus causing placentitis and abortion.

Etiology

Mucor and Asperigillus spp are the most common causes.

Pathogenesis

Infection occurs either by inhalation of spores with localization in the lungs or through the alimentary tract with localization in the abomasum and intestine. Hematogenous spread from these foci occurs to other organs including the placenta in pregnant cows and subsequently placentitis and abortion.

Clinical signs

Abortion usually occurs in cows at 6-8 months of pregnancy.

Postmortem lesions

These include:

1. Necrosis of the maternal cotyledons.
2. Ringworm like lesions occurs on the fetal skin.

Diagnosis

1. Epizootiological situation of the disease.
2. Clinical signs and lesions.
3. Laboratory diagnosis.

Samples: Placental cotyledons and fetal stomach should be collected.

a. Examination of direct smears of cotyledons and fetal stomach for demonstration of the hyphae.
b. Culturing for isolation of the causative agent.

Treatment

Treatment of systemic mycoses is carried out by the use of antimycotic drugs such as enilconazole, ketoconazole, amphotericin and nystatin.

Toxoplasmosis

It is an infectious disease of animals and man characterized by abortion and stillbirth in pregnant ewes, and encephalitis, pneumonia and neonatal mortality in all animal species.

Etiology

The disease is caused by *Toxoplasma gondii,* an intracellular

protozoan.

Toxoplasma gondii is an obligate intracellular protozoan that was first described in 1908. The species designation originated from the name of the North African rodent "*Ctenodactylus gondii*" from which this parasite was isolated. The genus name in Greek, mean "bow" referring to the crescent shape of the organism. Toxoplasma belongs to the phylum *Apicomplexa.*

- It has a two-host life cycle with both sexual and asexual stages.
- The major forms of the parasite are:
    1. Oocysts (containing sporozoites), which are shed only in the feces of cats.
    2. Tachyzoites; rapidly multiplying organisms found in the tissues during the acute stage of infection in the intermediate host.
    3. Bradyzoites, slowly multiplying organisms found in the tissue cysts.
    4. Tissue cysts: walled structures, often found in the muscles and central nervous system (CNS), containing dormant *T. gondii* bradyzoites.
- *Toxoplasma gondii* oocysts are resistant to most disinfectants but can be inactivated by iodine, formalin and ammonia.
- Tachyzoites and tissue cysts are susceptible to l% sodium hypochlorite and 70% ethanol.

Cats are the only definitive host that becomes infected by eating raw infected meat from the intermediate host (any vertebrate but frequently a rodent).

Life cycle

When a cat ingests an infected rat, the cysts are digested with release of large number of bradyzoites. The bradyzoites penetrate the intestinal epithelial cells and undergo series of chizogony and gametogony to produce oocysts that discharged in feces for two weeks, ten days after ingestion of cysts. Oocysts remain viable for 17 months on pasture and contaminate the animal feed. Intermediate hosts such as sheep, cattle and rats become infected by ingestion of oocysts. In the intermediate host the development is asexual, the oocyst wall is disrupted releasing eight sporozoites which penetrate the intestinal cells and reach lymph and blood vessels forming tachyzoites (banana shape). Tachyzoites spread to muscles, liver, heart, lung, uterus and CNS. Slow growing cysts containing bradyzoites develop in these tissues.

- Cats acquire the infection by ingestion of any of the three infective stages (Cysts, oocysts and tachyzoites).

- In cats some bradyzoites may penetrate more deeply into the wall of intestine where they multiply as tachyzoites and spread to infect other body sites via lymphatics and blood vascular system. This is called extra intestinal infection cycle.

Epizootiology

Geographical distribution

It is a worldwide disease.

Susceptibility

1. Domestic and wild animals, birds, rodents and man are susceptible.
2. Cats are the main reservoir.
3. Sheep are considered the most important domestic animal acting as intermediate host.

Sources of infection

1. Infected cats (definitive host)
2. Rodents (intermediate host)
3. Tissues of infected animals (intermediate host).
4. Pastures, feed and water contaminated with oocysts.

Transmission

1. Farm animals: Infection occurs by ingestion of food and water contaminated with cat feces, which contain the oocysts.
2. Cats: Infection occurs by ingestion of infected tissues of intermediate host as rodents.
3. Congenital transmission also occurs.

Pathogenesis

The clinical signs of toxoplasmosis vary according to the organ or tissues involved.

- In postnatal infection, fever, pneumonitis and enterocolitis are common. In congenital infection the principal manifestation is encephalitis.
- In pregnant ewes the pathological events depend upon the age of the fetus at the time of infection:

a. In the first two months there is embryonic death with resorption.

b. Two months to 100 days, there is death of the fetus with either mummification or abortion and if the fetus survives this period, there may be still birth or a weak lamb that may die within hours of birth.

c. Infection from 110 days to full term results in congenitally infected lambs.

Clinical signs

Sheep

1. Sheep with encephalitis show circle walking, incoordination and prostration.

2. Infected ewe aborts during the last month of pregnancy with metritis and placentitis.
3. There may be still birth or mummification.
4. Congenitally infected lambs are mentally dull, physically weak and unable to nurse and die due to starvation.

Cattle

The disease is similar to that occurs in sheep but toxoplasmosis appears to play no important role in bovine abortion.

Cats

Generally, there may be no clinical signs; sometimes there are pneumonia, hepatitis, diarrhea, jaundice, and eye and CNS affections.

Postmortem lesions

1. Fetal membranes show swollen, bright to dark cotyledons with white spots as gray foci 1-2 mm in diameter.
2. Hydrothorax and ascites may be observed.

Diagnosis

1. Epizootiological situation of the disease.
2. Clinical signs and lesions.
3. Laboratory diagnosis.

Samples include aborted fetus and placenta as well as blood serum samples.

a. Microscopical examination of tissue sections or impression smears directly or using fluorescent antibody stains to demonstrate the parasite (tachyzoites).
b. Laboratory animal inoculation: Intrapritoneal inoculation of mice with suspected material (cotyledons and fetal brain). After 7-14 days, ascitis occurs and tachyzoites can be demonstrated.
c. Serological examination using CFT, ELISA, IFA and Sabin-Feldman dye test. Sabin-Feldman dye test measures the total amount of antibody in serum which is capable of complement mediated killing of toxoplasma tachyzoites.
d. PCR.
e. **Treatment:** The use of sulfonamides as sulphadiazine, sulphadimidine and sulphamerazine combined with pyrimethamine during the early stage of the disease is recommended. Cats are treated using Clindamycine 10-20 mg/ kg bw. every 12 hours for two weeks.

Control

1.Eradication of rodents.
2.Prevention of cat fecal contamination for food and pasture.
3.Proper disposal of aborted material and carcasses of lambs.
4.Chemoprophylaxis:
   a. In cats: 200 mg/kg bw. monensin, suppress the shedding of

oocysts.

b. In sheep: Monensin is used to prevent the development of the disease, which significantly reduces the abortion rate.

Neospora caninum infection

Neospora caninum is harbored by dogs worldwide and is a common cause of abortion in dairy cattle.

Neospora caninum is a coccidian parasite that was misclassified as Toxoplasma gondii due to structural similarities. *Neospora caninum* is an important cause of abortion in infected livestock. Oocysts passed in the feces of the definitive host, such as canines are ingested by an intermediate host, such as cattle.

After ingestion of oocyst, it ultimately becomes a tachyzoite, which accumulates in the muscles and tissues of intermediate hosts forming tissue cysts. If the intermediate host acquires the disease during pregnancy, it activates these cysts, and active infection often causes spontaneous abortion.

- Abortion most commonly occurs between 4^{th} and 6^{th} month of gestation.
- Abortion storms have been observed and repeat abortions in affected cows have also been reported.
- Stillbirth can also occur. Occasionally infected calves are born alive; they are underweight, weak and often show signs of paralysis.
- Sometimes paralysis develops as late as 4 weeks after birth.
- Usually the aborted fetus is decomposed.
- Cows are not clinically ill and the placenta is not retained.

The definitive hosts become infected after ingestion of tissues of intermediate hosts containing tissue cysts.

Pups born from infected dogs show signs of paralysis.

Prevention:

- There is no safe treatment and no vaccine.
- Strict hygiene to prevent contamination of feed by dogs' feces is the only prevention.
- Dogs should be strictly kept out of feeding areas and pastures.

Trichomonosis, Trichomoniasis

It is an infectious venereal disease of cattle characterized by early abortion, pyometra and infertility.

Etiology

The disease is caused by a protozoan; *Trichomonas foetus*. It is an

ovoid protozoan of 5 – 15 microns in length, has three anterior flagellae.

Epizootiology

Geographical distribution

The disease has been reported in many countries of the world.

Susceptibility

Both cows and bulls are susceptible, older animals are more susceptible.

Sources of infection

1. The organism is present on the glans penis and in the prepuce of bulls.
2. The organism may contaminate semen collected by an artificial vagina.
3. The organism is present in the uterus, cervix and vagina of infected cows.

Transmission

1. Infection is transmitted by sexual intercourse of infected bull or cow.
2. Infection may be transmitted during artificial insemination.

Pathogenesis

Following exposure, the protozoan multiplies in the vagina and uterus, and then the protozoan establishes itself in the uterus leading to its inflammation which prevents conception or causes an early abortion. Abortion usually occurs at 2 – 4 months of pregnancy. The fetus is not always expelled but may be liquefied and expelled as discharges or retained as pus – filled uterus (pyometra).

Clinical signs and lesions

1. Infected cows return to the bull for four to five months after they have been served.
2. There is early abortion at 2 – 4 months of pregnancy.
3. Vaginitis and cervicitis usually develop.
4. Some cows suffer from endometritis with pus in the uterus. On pressing the uterus per rectum, a mucopurulent odorless material comes from the neck of the uterus into the vagina.
5. Affected bulls usually show no signs and sometimes they are lazy at service due to painful condition of the penis. Some bulls discharge mucopurulent discharge from the prepuce.
6. The placenta is often retained, and there may be pyometra.
7. The fetus has no specific lesions.
8. Cotyledons are hemorrhagic and thickened
9. Intercotyledonary areas are covered with flocculent exudate.

Diagnosis

1.Epizootiological situation of the disease.
2.Clinical signs.
3.Laboratory examination.
Samples:
-Aborted fetus or fetal stomach contents.
- Vaginal or uterine exudates.
- Pus from uterus using artificial insemination pipette fitted with a rubber bulb.
- Preputial wash.

a. Microscopical examination of fetal stomach contents, pus from uterus, vaginal discharges and preputial wash after its centrifugation to demonstrate the moving trichomonads.
b. Cultural examination using special media and special technique.
4. Test mating is used for bulls to be used for artificial insemination.

Control

1.Application of artificial insemination.
2.Cows should be given sexual rest for three months. (Usually a 90days period of sexual rest eliminates the organisms from the uterus).
3.Infected bulls should be culled.
4.Introduction of infected bulls or cows should be avoided.
5.Treatment of infected cows has been practiced by squeezing of corpus luteum in the ovaries per rectum, injection of 30mg stilboestrol IM and intrauterine infusion with lotagen 4 % and the use of antiprotozoal drugs such as metronidozole.
6. In bulls Ipronidazole is effective but can cause sterile abscesses at the injection site.