Infectious diseases causing abortion

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 (2^{nd}sample to be collected 2-3 weeks after 1^{st} 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 abortu*s require 10% Co_2 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 5^{th} 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 4th-5thmonth 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 4th month of life and the 5th 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 $\times 10^9$in 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 × 10^{10}per calf and one billion cells1–3 × 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

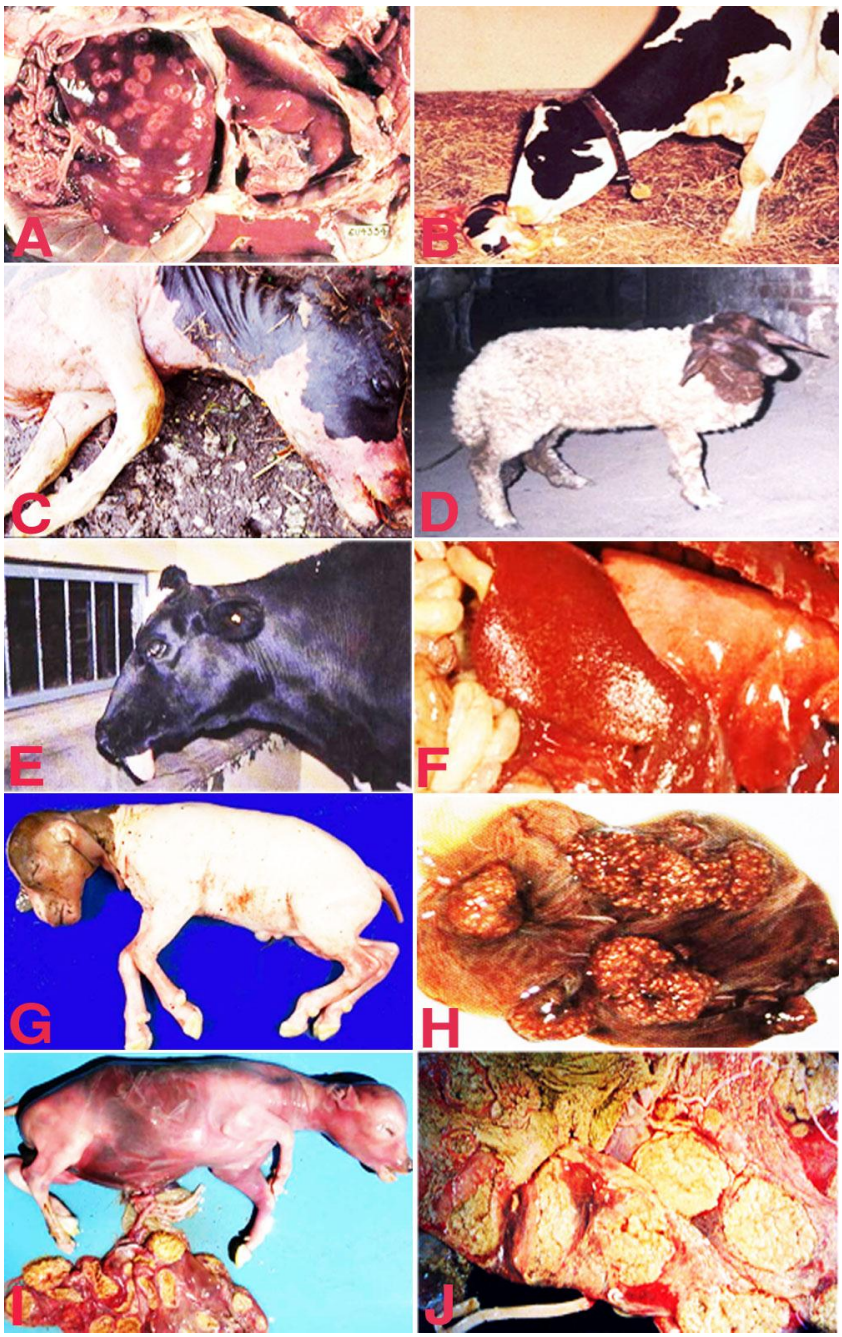
Samples: Semen, blood serum, epididymis and seminal vesicle should be collected.

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 clinical syndrome ranges from unapparent to acute or chronic infection.
- The disease is sudden in onset.
- In enzootic areas, the disease occurs sporadically.

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.

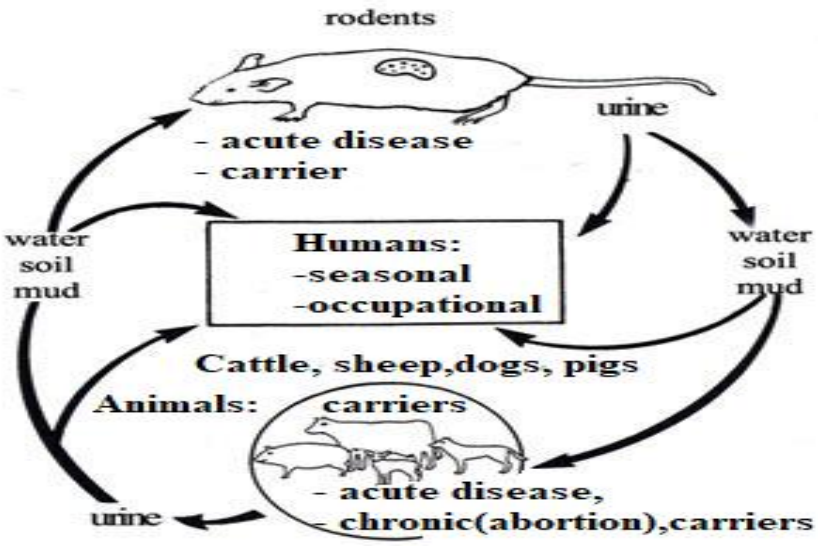


Figure (23): Transmission cycle of Leptospirosis

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.

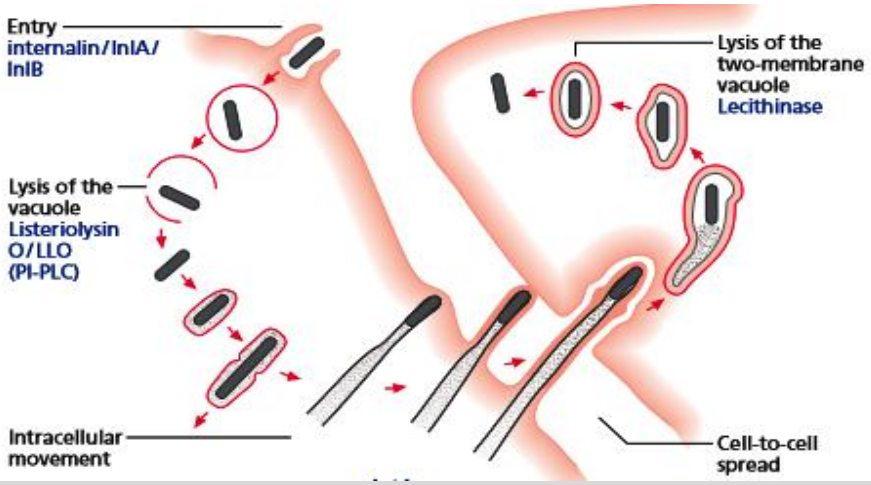


Figure (24): Cell to cell spread of Listeria Spp.

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 moves 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 develops, 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.

Cache Valley Virus infection

It is an Infectious disease of sheep, characterized by infertility, abortions, stillbirths, and multiple congenital abnormalities (hydranencephaly, hydrocephalus, cerebral and cerebellar hypoplasia and arthrogryposis).
The disease is caused by Cache Valley virus; a member of the Bunyaviridae family, Orthobunyavirus genus which was first isolated in 1956 from *Culisetainornata* mosquitos collected in Utah's Cache Valley, USA.

CVV has been known to cause outbreaks of spontaneous abortion and congenital malformations in ruminants such as sheep and cattle. CVV rarely infects humans, but when they are infected it has caused encephalitis and multiorgan failure.

The disease spreads by mosquitoes to pregnant ewes. Infection is similar to Akabane disease except that it only affects sheep.
Congenital defects include fused joints, curved or twisted spines, unusually thin and under developed muscles, and enlarged skulls.

Infection

*At less than 28 days of gestation, the embryos usually die and are reabsorbed.
*Between 28 and 45 days of gestation, the fetuses usually develop various congenital abnormalities affecting the central nervous system.
*After 45 days of pregnancy, there are usually no adverse effects.
Ewes that are infected usually show no signs of disease and develop a good immunity that lasts for several years.

Diagnosis

Determining whether Cache Valley was the cause of the abortions is difficult because the virus is not viable by the time its effects are noticed.
Diagnosis often requires the demonstration of viral antibodies in serum or body fluids.

Control

The most effective method of protecting ewes from the Cache Valley virus is to minimize their exposure to mosquito-infested areas during and shortly after the breeding season.
No vaccine or treatment is available.

Other diseases causing abortion:

- **Bovine Viral Diarrhea (BVD):** ***See page 307***
- **Border Disease (BD):** ***See page 318***
- **AkabaneDisease:** ***See page 321***
- **Infectious Bovine Rhinotracheitis (IBR):** ***See page 335***
- **Blue tongue (BT)** ***See page 350***
- **Rift Valley Fever (RVF):** ***See page 362***
- **Wesselsbron Disease:** ***See page 366***

Mastitis

Mastitis is a disease of all animal species but most commonly occurs in cattle and sheep causing severe economic losses. Mastitis is an inflammation of the udder caused by microorganisms that enter the gland through the teat canal. Once inside the gland, these organisms find ideal conditions to multiply and, in turn, to damage the lining of the milk ducts, cistern, and alveoli.

Clinical mastitis is an inflammation of the parenchyma of the mammary gland, characterized by:

- Pathological changes in the glandular tissues of the udder.
- Physical and chemical changes in the milk.
- Presence of pathogenic organisms in milk.

Subclinical mastitis is characterized by significant increase in

leucocytes count of milk without development of obvious signs.

Mastitis is an economically important disease due to:

- Decrease or loss of milk yield.
- Losses of cows either due to premature slaughtering or death as a result of peracute septicemic mastitis.
- Transmission of infection to suckling calves.
- Cost of treatment and control.

In addition, different types of bacteria causing mastitis have public health significance due to shedding of bacteria in milk e.g. streptococcal sore throat, food poisoning and tuberculosis.

Beside the specific mechanism of udder immunity which is mediated by both humoral (immunoglobulin) and cell mediated immunity, a non-specific mechanism also contributes in immunity of the udder through the action of different antibacterial substances that include:

1. The esterified and non-esterified fatty acids present in teat canal and sphincter keratin.
2. Lactoferrin; an iron chelating protein.
3. Lactoperoxidase enzyme which has a bacteriostatic effect on Gram negative bacteria.
4. Xanthine oxidase which liberates H_2O_2.
5. Lysozyme and cationic proteins.

Etiology

The most common organisms causing mastitis in cattle include, *Streptococcus agalactiae, Streptococcus dysgalactiae, Streptococcus uberis, Streptococcus zooepidemicus, Streptococcus pyogenes, Staphylococcus aureus, Escherichia coli,* Klebsiella spp., *Enterobacter aerogenes*, Pasteurella spp., Leptospira spp., Mycobacterium spp., Mycoplasma spp., *Actinomyces pyogenes*, Asprigillus spp., Candida spp., *Cryptococcus neoformans*, Toxoplasma ....etc.

Organisms causing mastitis can be divided into **two main categories**; contagious organisms and environmental organisms.

1. **Contagious organisms** are those colonizing the mammary gland and having no long existence outside the host. These organisms usually result in development of new cases of mastitis within the first two months of lactation. They usually spread by milking machines and hands of milkers. The major contagious pathogens are *Staphylococcus aureus, Streptococcus agalactiae* and *Streptococcus dysgalactiae* and Mycoplasma spp.

Contagious bacteria are spread from a cow with an infected udder to a healthy cow. Transfer of pathogenic bacteria between cows usually occurs

at milking time. Hands, towels, or the milking machine can all act as reservoirs for contagious bacteria.

2. Environmental organisms are those that have a long existence in the environment (bedding, soil, manure, etc.) and thus are highly influenced by management practices. They do not persist in the udder.

The most common environmental bacteria are the coliforms (*E. coli,* Klebsiella spp, and Enterobacter), whose main origin is manure and soil, and the environmental streptococci (*S. uberis* and *S. dysgalactiae*) that come from the environment but also from infected udders. Coagulase negative staphylococci are typically found on teat skin, nasal tissue, in the teat canal and on the hands of milk technicians and they also come from infected udders. Environmental bacteria thrive under wet conditions in the presence of the manure.

Epizootiology

Geographical distribution

The disease is widely distributed all over the world.

Susceptibility

1. All breeds especially the high producing animals are commonly affected.
2. Old cows are more susceptible.
3. Unhygienic conditions and unhygienic milking predispose for mastitis.
4. Faulty milking machines as excessive suction predisposes for mastitis.
5. Teat skin lesions caused by FMD, Pox and lumpy skin disease increase the susceptibility.
6. Vitamins A, E, and Selenium deficiency may predispose for clinical mastitis.

Sources of infection

1. Infected udder is the main source of infection by contagious pathogens that reside in the udder.
2. Infected milk is an important source of infection.
3. Contaminated environment especially the bedding as well as contaminated utensils and milking machines are sources of infection.
4. Infected milker hands may transmit the infection.
5. In hematogenous infection, the blood of the same animal acts as a source of infection.

Transmission

1. Infection occurs through the teat canal in most cases.
2. In rare cases, infection may occur via the blood stream

(hematogenous).
3. Mastitis may arise from previously infected udder.
4. Flies may transmit the infection.

Pathogenesis

In most cases of mastitis infection occurs via the teat canal and the causative organisms pass from exterior of the teat to the milk inside the teat canal. The organisms rapidly multiply in the milk and invade the mammary tissue. According to the degree of susceptibility, inflammation of the mammary gland tissue occurs with development of clinical signs associated with increase of leucocytic count in milk. Some pathogens secrete toxins which result in increase of vascular permeability leading to edema and local swelling of the udder. Systemic involvement and toxemia may develop.

General types of mastitis

According to the severity of infection, mastitis can be classified into:

- Peracute mastitis, manifested by marked systemic reaction, toxemia, and severe udder inflammation e.g. coliform mastitis.
- Acute mastitis, in which severe inflammation of the udder occurs, associated with changes in the nature of the milk and mild systemic reaction.
- Subacute mastitis is characterized by mild inflammation of the udder with persistent changes in milk.
- Chronic mastitis is characterized by changes in milk in the form of clots associated with fibrosis and atrophy.
- Subclinical mastitis, the milk appears grossly normal, there is no visible sign of inflammation of the udder but results in decreased milk production.
- Gangrenous mastitis, in which parts of the mammary gland become cold and secretions are watery and sanguineous. Areas of the mammary gland may slough within 10 to 14 days.

Mastitis may be classified into, catarrhal, suppurative, gangerenous and interstitial mastitis. This depends on the causal microorganism.

Clinical signs

Clinical signs of mastitis vary according to the resistance of the udder and the type and virulence of the causative microorganism.

I- General signs of mastitis:

1. Abnormalities in milk include blood staining, wateriness, presence of flakes or pus. Such changes can be detected by strip cup method.
2. Abnormalities of the udder include changes in the udder size and consistency, swelling, hotness, pain, enlargement of supramammary lymph nodes, fibrosis and atrophy. Such changes can be detected by palpation and visual inspection.

3. Systemic reaction includes toxemia, pyrexia, and anorexia.

II- Specific signs: it varies according to the causative organism.

- ***Streptococcus agalactiae* mastitis**: The causative agent is a common inhabitant of the udder. The disease is characterized by fever, udder inflammation, and watery milk.
- ***Staphylococcus aureus* mastitis:** It is characterized by severe systemic reaction, toxemia, severe swelling of the udder, gangrene and sloughing of the infected quarter may occur 6-8 days later due to toxin production which causes vasoconstriction resulting in ischemia and death of tissues. Such type of mastitis is poor in its response to antibiotic therapy due to the survival of the organism in macrophages and epithelial cells.
- ***Arcanobacter pyogenes* (summer mastitis):** *Arcanobacter pyogenes* is a common inhabitant of the udder tissue causes udder abscessation in pregnant heifers, dry cows and lactating cows. The disease is characterized either by development of sporadic cases of suppurative mastitis or outbreaks in summer months referred to as summer mastitis. The presence of anaerobes such as *peptostreptococcus indolicus* increases the severity of summer mastitis. The disease is usually peracute with a severe systemic reaction. The teats and quarters become swollen. The secretion is watery with clots, then purulent with putrid odor. The abscesses develop and rupture through the floor of the udder commonly at the base of the teat.
- **Coliform mastitis** (*E. coli*, Klebsiella spp. And *Enterobacter aerogenes*): It is an environmental mastitis occurs due to contamination by coliform organisms from bovine feces. The disease occurs commonly around calving. It is characterized by sudden onset, development of peracute form, systemic reaction, diarrhea with swollen and hot quarters and yellowish watery milk that contains clots and flakes. Cow may become recumbent within hours and death occurs due to endotoxemia. Acute or chronic forms may develop.
- **Tuberculosis mastitis** (Mycobacterium spp.): It is a chronic disease of hematogenous origin characterized by marked induration and hypertrophy firstly in the upper part of the udder then involves the entire gland. The supramammary lymph nodes are enlarged. Milk contains very fine floccules, which settle after the milk stand leaving clear amber fluid.
- **Mycoplasmal mastitis**: The disease is caused by *mycoplasma bovis, Mycoplasma candensis* and *Mycoplasma bovigenitalium*. The disease is characterized by its sudden onset, involvement of all quarters and severe swelling of the udder. Granular deposits

in the form of fine sandy materials and flakes can be recognized in milk on standing leaving turbid whey like supernatant. *Mycoplasma* spp. may damage the secretory tissue and produce fibrosis in the udder, as well as abscesses with thick fibrous walls and marked enlargement of the supramammary lymph nodes. Complete cessation of milk production may occur. Some cases are associated with fever and arthritis.

- **Pasteurella spp mastitis:** Mastitis caused by pasteurella spp. is common in sheep. It is characterized by systemic reaction, lameness, acute swelling of the udder and watery milk. The udder may become blue and cold with development of gangrenous mastitis. In cattle, there are systemic reaction and marked swelling of the udder then fibrosis and atrophy.
- **Mycotic mastitis**: Different types of fungi and yeasts may cause mastitis. *Cryptococcus neoformans* causes swelling of the quarters and supramammary lymph nodes, severe fall in milk yield and viscid mucoid gray white secretion. Candida spp. causes fever, severe inflammation of the quarters, enlargement of supramammary lymph nodes and large yellow clots in a watery supernatant fluid. Asprigillus spp. causes development of abscesses in quarters surrounded by granulation tissues.

Diagnosis

1. Epizootiological situation of the disease.
2. Clinical signs:

It is of importance to determine if mastitis is associated with systemic reaction or not. The udder is examined visually and by digital palpation for detection of udder abnormalities such as swelling, induration, pain, heat, abnormal size, atrophy, abscessation and enlargement of the supramammary lymph nodes.

3. Field examination

Field tests are carried out for detection of changes in milk.

Several field tests are used:

a. Strip cup test: The test is done for visual detection of flakes, clots, pus, blood and watery milk by drawing the first few streams of milk from each quarter into a cup.
b. Electrical conductivity test: This test is based on detection of the increase of sodium and chloride ions of mastitic milk, which consequently increase the electrical conductivity.
c. pH indicator paper or pH meter: Normal milk has a pH of 6.4-6.8 while mastitic milk has a pH of 6.7 up to 7.4.
d. California mastitis test (CMT), white side test, and Wisconsin test and the NAGase test. These tests determine approximately the number of leukocytes and used as screening individual and herd

tests. These tests can't be used in cows 7 – 10 days after calving due to the increased leukocytic count.

California mastitis test is carried out by mixing equal quantities of milk samples and California reagent in a white plastic paddle and the result is read as the degree of gel formation as follow:

Test result	Leukocytes / ml of milk
Negative	0 – 200,000
Trace	150,000 – 500,000
+	500,000 – 1500,000
++	1500,000 – 5000,000
+++	> 5000,000

- **NAGase** test measures the cell associated enzyme N. acetyl B-D glucosaminidase in milk. The high level of this enzyme indicates high cell count.

4. Laboratory diagnosis

Samples: Milk samples are collected after washing the udder with warm water and soap and swabbing of the teats with 70% alcohol. The first two streams should be discarded and 25 ml of milk from each quarter must be collected. Milk of the last streams should be collected when tuberculosis is suspected. Laboratory diagnosis includes:

a. Direct microscopic examination of milk smears prepared from fresh milk or milk sediment or milk incubated at 37 ^{o}C for 1- 2 days. Milk smears are stained with methylene blue, newman's stain or Ziehl-Neelsen stain. A count of more than 4 x 10^5 cells/ml in bulk milk sample indicates mastitis.

b. Isolation and identification of the causative agents.

c. Chlorine test: Mastitic milk contains more than 0.14% chlorine.

Differential diagnosis

Mastitis should be differentiated from the following conditions:

1. Physiological edema and congestion of the udder that occurs precalving. The udder becomes swollen and edema may extend along the abdomen in front of the udder. Doughy swelling that pits under pressure can be detected. This may occur due to circulation interference by the gravid uterus, passage of beta and gamma globulins from blood to the mammary glands and mostly due to hereditary factors. Such condition usually disappears within 2-3 days. It responds rapidly to injection of diuretics.
2. Bloody milk: It occurs in heavily milk producing cows after calving. Leptospiral infection is characterized by bloody milk and flaccid

udder without udder inflammation.

3. Actinomycosis may cause hard painless masses in the udder.

Treatment

General considerations:

- The udder should be washed and dried under hygienic conditions.
- Complete emptying of the udder is necessary either by hand stripping or injection of oxytocin (20 IU) can facilitate the removal of contaminated milk.
- Application of sensitivity test is advisable.
- The udder should be massaged after drug infusion.
- Milk of treated cows should not be used for calves feeding or human consumption for at least three days from the last dose.
- Some types of mastitis require specific treatment.

Line of treatment:

1. Parentral treatment: It is indicated in cases of mastitis associated with systemic reaction to control or prevent septicemia or bacteremia as well as to help in local treatment of the udder. Antibiotics used for parentral treatment should be diffusible from blood to the udder, these include: Ampicillin 10 mg/kg body weight, erythromycin and tylosin 12.5 kg body weight, penicillin 16.000 I.U/kg body weight, oxytetracycline 10 mg/kg body weight, and sulphadimidine 200 mg/kg body weight, Treatment should continue for at least 5 days.

2. Isotonic solution and non-steroid anti-inflammatory should be used in case of peracute mastitis.

3. Udder infusion: It is the principal method for treatment of mastitis using disposable tubes containing antibiotics with or without anti-inflammatory agents and used for 3-5 successive days. The commonly used antibiotics are ampicillin, cephalosporins, penicillin G sodium, cloxacillin, tetracycline, erythromycin and tylosin. Cases of mycotic mastitis respond to Nystatin and Clotrimazole udder infusion therapy.

4. Fluid therapy, anti histaminics and cold fomentation of the udder to reduce toxins absorption should be used.

5. Treatment of dry cows: This method is used for chronic cases and to prevent new infections due to the same pathogens. It is also applied in herds with high prevalence of contagious mastitis. It is applied after the last milking of lactation by udder infusion of long acting antibiotics. Long acting cloxacillin 10.000 I.U and cephalosporins are commonly used. Teats are then dipped in 0.5% chlorhixidine or 0.5% iodine for 20 seconds then the teat orifice is closed by wax.

6. Treatment of coliform mastitis:

- Toxins can cause a state of shock and dilatation of blood vessels and fall of blood pressure leading to poor circulation and consequently poor tissue perfusion with blood and dehydration that frequently

reaches 7–10% of body weight. A fast way of administering large volumes of fluid (e.g. 10–20 liters) is by using an oral pump or the use of intravenous administration of 2.0 liters hypertonic salt solution (70 g per liter NaCl). This stimulates extreme thirst in the cow and encourages drinking.

- Shock can also be counteracted by the use of anti-inflammatory drugs, or cortisone. Cortisone given locally or parenterally reduces swelling and the inflammatory response, 10 mg prednisolone reduces the hardness and swelling in the affected quarter. This permits better antibiotic penetration.
- Some sick cows are naturally hypocalcaemia and hypoglycemic. For this reason, 400 ml of calcium borogluconate, possibly mixed with glucose may be used.
- Broad-spectrum antibiotics.

7. Treatment of mycotic mastitis:

- Iodine preparations can be used.
- Nystatin and Clotrimazole udder infusion therapy.
- Infusion of infected quarters with 20 ml of natural live yogurt at 12–hourly intervals for 2 to 3 days. The objectives are to decrease the raised pH of mastitic milk and to eliminate residual mastitis organisms by the probiotic effect of natural lactobacilli in yogurt.

8. Drying off chronically infected quarters: Incurable quarter is dried off by producing chemical mastitis using one of the following:

- 30-60 ml of 3% silver nitrate solution.
- 20 ml of 5% copper sulphate solution.
- 100-300 ml of 1/500 acriflavine solution. Two infusions may be necessary.

9. Treatment of gangrenous mastitis: Cases of gangrenous mastitis are treated by immediate amputation of the udder and massive doses of antibiotics and corticosteroids.

Control

1. Detection of infected quarters: This is carried out by periodical application of screening tests as CMT for detection of subclinical infection. Milk should be examined bacteriologically two times at weekly intervals. Clinical cases are detected by physical palpation of the milked-out gland and application of the field screening tests.
2. Treatment of infected quarters: Infected quarters should be treated and followed by three examinations at two weeks interval for detection of relapsing quarters and to evaluate the efficacy of treatment.
3. Culling of incurable cases and chronically affected cows.
4. Dry cow treatment: All quarters should be treated after the last

milking of lactation using long acting antibiotics.

5. Prevention of spread of infection through:
   a. Udder sanitation: The udder and teats should be washed and dried before each milking and teats dipped in iodophore solution containing 1% iodine or chlorhixidine 0.2%.
   b. Personal hygienic measures for milkers as disinfection of hands between milking of each cow.
   c. Regular disinfection of animal house, floor, cleaning and disinfection of milking equipments and maintenance of the milking machine periodically.
   d. Infected cows should be milked separately by special attendant and in separate containers.
   e. Muzzling of calves in infected farms to avoid suckling of immature udders as some pathogens may become established in immature udders.
6. Nutritional management of dry cows: Adequate of levels vitamin E and selenium in dry cow rations have been proved essential for udder health at calving and in the early lactation.
7. Immunization: Some vaccines have been developed to control mastitis e.g. R. mutant *E. coli.*
8. The use of recombinant bovine cytokines to enhance the specific immunity in the mammary gland of cows.