Chapter

Bacterial Diseases of Goat and Its Preventive Measures

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Abstract

Bacterial diseases of goats can cause huge economical loss to the farmers. Due to intensification of goat farming and poor hygienic practices there is increase in the number of bacterial diseases that affect the goats. Diseases like tuberculosis, Johne's disease and Brucellosis are chronic diseases that may be identified in the initial stages of infection during which they spread to other animals. Similarly, brucellosis, tuberculosis and also anthrax are zoonotic diseases hence due consideration has to be provided while handling animals suspected for these diseases. Use of vaccine before onset of the disease in endemic areas can prevent the disease outbreak and spread to other naïve population. Good hygienic practices and biosecurity measures at farm are essential to prevent disease spread. The present chapter deals with various bacterial diseases affecting goats and its preventive measures. This chapter can be a guide to field veterinarians, students and farmers as it highlights the important bacterial diseases of goats.

Keywords: Goat, Bacterial disease, Brucella, Anthrax, Preventive measures

1. Introduction

Due to intensification of small ruminant farming, there is increase in the number of disease outbreaks in the recent years. Among the various infectious diseases, diseases caused by bacterial pathogens contribute to severe economic loss to the goat farmers. Various factors like increase in herd size, reduced ventilation in farm and poor husbandry practices can predispose to diseases. Bacterial diseases like anthrax, enterotoxaemia, tetanus, gas gangrene, caseous lymphadenitis, listeriosis, tuberculosis, Johne's disease, dermatophilosis, pasteurellosis/mannheimiosis, brucellosis, foot rot, contagious caprine pleuropneumonia, colibacillosis, salmonellosis, etc., affect goats and can cause various ailments and some diseases can cause heavy mortality leading to huge economic loss to the farmer [1]. Different bacterial pathogens affect different organs of goat thereby eliciting various clinical signs based on which a tentative diagnosis can be made (**Figure 1**).

Antibacterial agents can be used to treat various bacterial diseases but these drugs should be used judiciously due to the risk of development of antimicrobial resistance. Vaccination is the best way to prevent infectious diseases and based on the pattern of the disease annual vaccination should be practiced to prevent disease outbreaks. Diseases like anthrax, brucellosis and tuberculosis pose threat to human since these diseases can be transmitted to human through direct or indirect route of transmission [2]. Due care should be taken while handling infected goats or dead goats in farm as the zoonotic diseases can cause severe aliments in human.

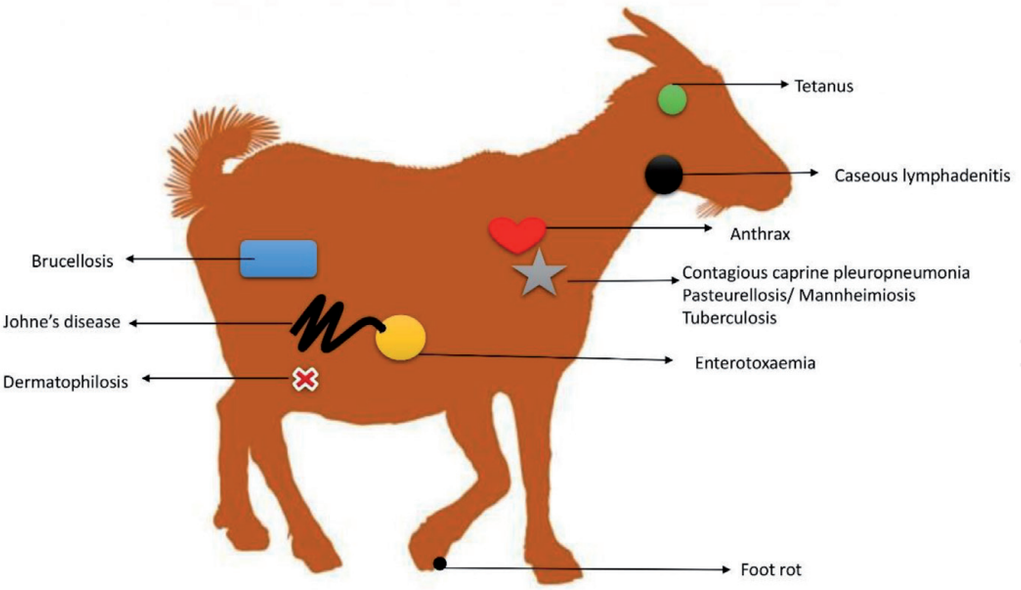

Figure 1.
Different bacterial diseases of goat and the organ/ tissues affected. Brucellosis affects reproductive tract, dermatophilosis affect the skin, johne's disease causes corrugation of intestine, pasteurellosis/ mannheimiosis, tuberculosis, contagious caprine pleuropneumonia affects the respiratory system, caseous lymphadenitis affects the lymph nodes and tetanus affects the nervous system. This figure is propriety of the authors.

2. Methods

This chapter is a comprehensive summary of important bacterial diseases of goats and this can be a guide to veterinary students, field veterinarians and goat farmers regarding the impact of these bacterial diseases. This chapter also highlights the preventive measures and zoonotic potential associated with the bacterial diseases of goats. Important bacterial diseases that are zoonotic and economically important like anthrax, brucellosis, tetanus, enterotoxaemia, Johne's disease, Pasteurellosis/ Mannheimiosis, caseous lymphadenitis, contagious caprine pleuropneumonia, dermatophilosis and foot rot are discussed. Each disease is delt with various subsections like definition of the diseases, etiology, epizootiology, transmission clinical signs, diagnosis, treatment, preventive measures and public health significance, if any.

3. Anthrax

Anthrax is a peracute, acute or subacute, often fatal disease of animals including goats. In goats the disease is mainly characterized by septicaemia, splenomegaly and gelatinous infiltration of subcutaneous or subserosal tissues. The disease is commonly known as woolsorter's disease, splenic fever, charbon, and milzbrand.

3.1 Etiology

The disease is caused by *Bacillus anthracis*, a gram positive, capsulated, non-motile, aerobic, spore-forming, rod shaped bacterium [3].

3.2 Epizootiology

The disease is worldwide in distribution and is endemic in some countries, while occurs in defined regions of other countries. It was reported to be associated

with heavy mortalities in goats and sheep of sub-saharan region in 1960–70s and in other countries. In recent days, through strict vaccination procedures the incidence reduced in most countries, however, sporadic cases are still being reported. *B. anthracis* is widely distributed in the environment, as they produce highly resistant endospores. They can tolerate extremely adverse conditions such as desiccation, high temperatures and chemical disinfectants. When the vegetative bacteria are exposed to atmospheric oxygen under favorable temperature (20–40°C) and relative humidity (>60%), the spores are formed. Further, the calcium plays a role in spore formation and in combination with dipicolinic acid, enhances the spore survival. Hence, its survival is more in alkaline soil that is rich in calcium and nitrogen and with high moisture content, the endospores can survive for more than 50 years. Further, recurrent cycles of flooding and evaporation may concentrate spores in particular low-lying regions [3].

3.3 Transmission

Goats are infected by ingestion of food, water or soil contaminated with spores. The infection can also occur through inhalation or abraded skin and oral mucosa. Mechanical transmission by biting insects is also reported. Wild animals acting as carriers makes the control programme challenging as it is least possible to vaccinate all wild animals.

3.4 Clinical signs

The incubation period ranges from hours to days. The disease is usually fatal, especially in sheep and goats, after 1–3 days. The peracute case is characterized by sudden death without any premonitory signs. However, there may be fever, dysponea, congestion of mucous membranes, muscular tremors and terminal convulsions in few animals. In acute cases, fever, anorexia, labored breathing, increased heart rate, ruminal stasis and reduce milk production may be observed. There may be bloody discharges from orifices like mouth, nostrils, anus and/or vulva. Diarrhea or dysentery and oedema and swelling of the tongue, throat, flank and perineum (anus, vulva) may be seen. Pregnant animal abort and blood-tinged milk is produced. Animals then collapse with terminal convulsions and die [4].

Necropsy of suspected carcass is not recommended, as the vegetative bacteria may get transformed into spore and hence contaminate the environment. The pathological features such as absence of rigor mortis and rapid putrefaction and bloating of the carcass are common clinical features. Oozing of unclotted dark, tarry colored blood from orifices, soft and enlarged spleen, blood-stained fluid in body cavities and widespread ecchymotic hemorrhages are frequently observed post mortem findings.

3.5 Diagnosis

Though clinical signs are highly suggestive, the diagnosis based on clinical signs alone is difficult. Thin smears of blood from ear tip can be stained with polychrome methylene blue stain to reveal short chains of truncated blue color rods, surrounded by pink capsules (McFadyean reaction). The organism can be cultured on Sheep or Ox blood agar which shows flat, dry grayish colonies with 'ground glass' appearance after 24–48 hours of incubation (**Figure 2**). The selective media for the organism is PLET (Polymyxin-lysozyme-EDTA thallous acetate) medium. The Ascoli's thermo-precipitation test is also commonly used test to detect antigens of *B. anthracis*. Agar gel immunodiffusion, complement fixation test, ELISA and immunofluorescence

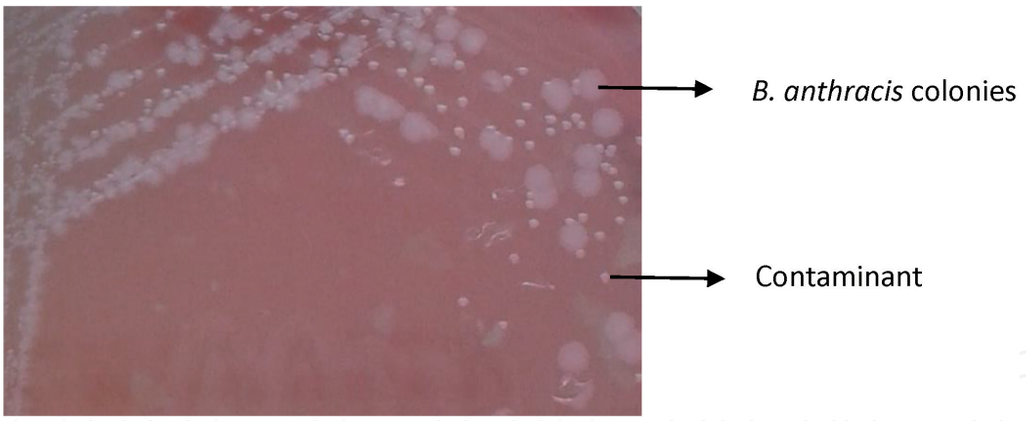

Figure 2.
Ground glass appearance Bacillus anthracis colonies on sheep blood agar. This figure is propriety of the authors.

tests though available are insensitive and not routinely used [4]. The PCR test can be used for direct detection of the organism from decomposed samples and can also be employed for targeting the pXO1 and pXO2 plasmids to confirm the virulence of isolates from the culture.

3.6 Treatment

Ailing animals in early stages of infection can be treated with penicillin or oxytetracycline or other long-acting antibiotics. An anthrax antiserum may result in recovery if used in early stages. Vaccination should follow 7–10 days after the conclusion of antibiotic therapy [4].

3.7 Preventive measures

In endemic areas, annual vaccination is advisable. The goat should be vaccinated with 'Sterne strain' live spore vaccine one month before the anticipated outbreaks. In non-endemic areas, movement of animals and their products should be restricted; feed and bedding materials etc., should not be transferred from affected herds. Disinfection of the premises with 5% formalin, 5% sodium hydroxide or 3% peracetic acid and placing foot-baths containing these sporicidal disinfectants at the entrances of the affected farms will help to control the spread of infection. Contaminated building should be fumigated with formaldehyde before removing the bedding materials [5]. Proper disposal of carcasses and the infected materials should be done either by deep burial or incineration.

3.8 Public health significance

B. anthracis is considered a bioterrorism agent. Three forms of disease occur in human beings. When endospores enter through abraded skin, the cutaneous form of anthrax (malignant pustule) develops. While, the pulmonary form (woolsorters' disease) follows inhalation route and intestinal form results from ingestion of infective material. The disease usually fatal if not treated early [6].

4. Brucellosis

Caprine brucellosis is an infectious zoonotic disease having substantial economic impact on both livestock and human. Caprine brucellosis is reported since

ancient days; Hippocrates II first described the human brucellosis in 400 B.C. which was most likely to be associated with consumption of raw milk or derivatives of infected sheep or goats.

4.1 Etiology

The causative agent is *Brucella* species mainly *B. melitensis* that are small, non-motile, non-spore forming, gram-negative coccobacilli. Goats are also susceptible to infection by *B. abortus,* particularly when housed in close proximity with infected cattle; however, they do not sustain the infection in the herd [7].

4.2 Epizootiology

The disease is prevalent worldwide and it remains a major burden in parts of Mediterranean region, the Middle East, Central and Southeast Asia (including India and China), sub-Saharan Africa, and parts of Latin America [8]. Goat herds from USA, Canada, Colombia, Chile, and Uruguay are reported to be free from *B. melitensis* infection.

4.3 Transmission

Infection occurs primarily through ingestion of the organisms. Goats acquire infection by licking the aborted fetuses, placentas, newborn kids, vaginal discharges, or by consumption of feed contaminated with these infectious materials [9]. Milkers can also spread the infection through unsanitary milking practices.

4.4 Clinical signs

The disease is more severe in goats and is protracted than in sheep. Clinical manifestations include high abortion rates particularly during the fourth month of pregnancy and retained placentas, orchitis in bucks, arthritis and hygromas. In goats, mastitis and lameness may also be seen. The abortion rate can be high when this bacterium first enters a naive flock or herd [10]. The abortion rates are usually much lower once *B. melitensis* has become established in a herd and only a few animals abort repeatedly but affected animals shed bacteria during parturition. Healthy asymptomatic carriers become a potential source of infection. Other clinical signs include death of weak offspring, low weaning weight, decreased milk production, and epididymitis, and reduced fertility which is more common in sheep. In case of abortions, fetus might reveal excess of blood-stained fluids in the body cavities, with enlarged spleen and liver. Moreover, infected foetal membranes can show thickened and dull-gray color necrotic cotyledons [11].

4.5 Diagnosis

Diagnosis is made based on clinical signs, direct examination of MZN-stained smears of fluids or tissues, isolation and identification of *B. melitensis* from milk or an aborted fetus or by serum agglutination tests. The Rose-Bengal agglutination test and the complement fixation test are the most widely used methods for detecting *B. melitensis* infection and are approved for international trade. Indirect enzyme-linked immunosorbent assays (ELISA) have been developed and are also approved tests for the purposes of international trade [12]. Isolation and identification of *B. melitensis* from aborted foetal stomach contents, placenta and uterine fluids can be

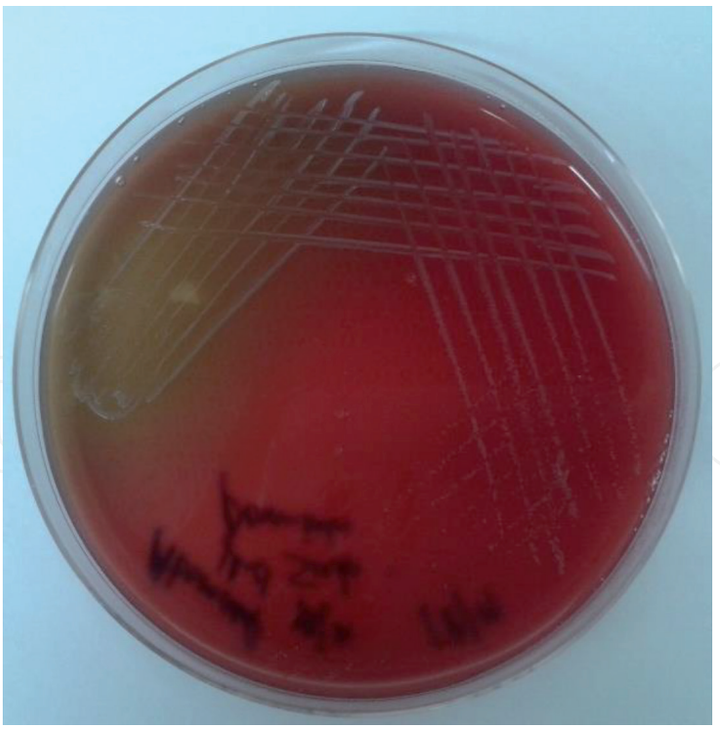

Figure 3.
Brucella melitensis colonies on sheep blood agar. This figure is propriety of the authors.

attempted and isolation is the gold standard technique for confirmation of brucellosis (**Figure 3**). Isolation should be carried out in biosafety cabinet class III as the organism is zoonotic.

4.6 Preventive measures

Test and slaughter policy of the infected herd is generally implemented in countries where the disease is considered exotic. This can also reduce the prevalence of disease in endemic areas. In most countries where *B. melitensis* is endemic, vaccination with the Rev. 1 strain is commonly employed [13]. It is a live attenuated strain of *B. melitensis*; administered by the subcutaneous or conjunctival routes and is used for vaccination of kids and lambs up to 6 months of age.

4.7 Public health significance

B. melitensis is highly pathogenic than other species of Brucella for human beings. The infection in human is characterized by fever, chills, headache, malaise, back pain, myalgia and lymphadenopathy, which may be accompanied by splenomegaly and/ or hepatomegaly. The patients may experience drenching sweats at night and nonspecific gastrointestinal signs such as vomition, diarrhea and/or constipation [14]. Localized manifestations such as arthritis, spondylitis, sacroiliitis, osteomyelitis, bursitis and tenosynovitis may be observed. Epididymo-orchitis, prostatitis and seminal vesiculitis can be seen in males, whereas abortion or premature births are seen in pregnant women. Deaths are usually uncommon except in infants caused by endocarditis or infections of the brain.

5. Tetanus

Tetanus (Lockjaw) is an acute, highly fatal intoxication of all domestic animals and humans caused by neurotoxin produced by the bacteria *Clostridium tetani* [15].

Though all species of livestock are susceptible, sheep and goats are more susceptible than cattle and horses being the most susceptible. It is characterized by hyperasethesia, tetany and convulsions.

5.1 Etiology

The etiological agent, *Clostridium tetani* is a strictly anaerobic, motile, slender, straight, spore forming ('drumstick appearance'), Gram-positive rod. Based on flagellar antigens, so far 10 serotypes of *C. tetani* have been described and all produce antigenically a similar neurotoxin called tetanospasmin. Though endospores are resistant to chemicals and boiling, they are destroyed by autoclaving.

5.2 Epizootiology

Tetanus is worldwide in distribution and occurs sporadically. The organism is normal inhabitant of intestinal tract of animals and persists as resistant spores in soil, manure [16].

5.3 Transmission

The toxemia in tetanus is caused by a specific neurotoxin produced by *C. tetani* in necrotic tissue. Spores are introduced into the tissue through wounds, specifically deep puncture wounds that provide the favorable anaerobic environment. Most outbreaks occur following mass contamination of animals during castration, vaccination, ear tagging, docking and other surgical procedures [17]. Grazing on rough and spiky pastures may injure the oral mucosa and hence may facilitate the invasion of the bacteria. The spores remain dormant in tissues and proliferate to liberate toxins under favorable conditions.

5.4 Clinical signs

The incubation period is usually of 4 days to 3 weeks. The initial signs include muscle stiffness, tremors and prolapse of the third eyelid. This is followed by rigidity and extension of the limbs leading to a stiff gait and abnormal flexion of the joints. Tetany of masseter muscles causes drooling of saliva (lock jaw) and regurgitation through nostrils [17]. The animals may exhibit bloat, an inability to chew, and hyperthermia. Retracted lips, hypersensitivity to external stimuli, and a 'saw-horse' stance are frequent signs. The spasms of alimentary and urinary tract muscle may cause constipation and retention of urine [17]. The abnormal muscular contracture may result in opisthotonus, curvature of the spine and bending of the tail. The disease is highly fatal and death occurs within 3–10 days with mortality nearing 100%, primarily as a result of respiratory failure. Necropsy features usually are nonspecific except for the inflammatory reaction associated with the wound.

5.5 Diagnosis

Diagnosis can be made based on clinical features such as muscular spasms, prolapse of third eyelid and based on history of trauma or surgery. The Gram-positive rods with terminal spores can be demonstrated in the smears prepared from necrotic tissue or wound [18]. Anaerobic culture of the bacteria from necrotic tissue may be attempted but is often unsuccessful. PCR and real-time PCR techniques can be employed for the detection of neurotoxin genes of the organism. Mouse inoculation test can be performed to demonstrate circulating neurotoxin from the serum of affected animals.

5.6 Treatment

Treatment mainly aimed at wound management, antibiotic therapy, antitoxin administration and vaccination. Wound management consists of surgical debridement of infected wounds and removal of debris, flushing with hydrogen peroxide to produce aerobic condition that helps to inhibit replication of the bacteria at the site of infection. The antibiotics (large doses of Penicillin) can be given both parenterally and flushed into the cleaned wound to prevent further replication of the bacteria and production of toxin [19]. Affected animals must be kept in a quiet and dark environment. Fluid replacement therapy, sedatives and muscle relaxants can minimize clinical discomfort and maintain vital functions. To neutralize unbound toxin, the tetanus antitoxin must be administered on time, either intravenously or into the subarachnoid space for three consecutive days. Vaccination with tetanus toxoid may be given subcutaneously to promote an active immune response even in those animals that are treated with antitoxin.

5.7 Preventive measures

Tetanus can be controlled by following good sanitation measures, aseptic surgical and management procedures and vaccination. Goats in a herd must be vaccinated routinely with tetanus toxoid which is very effective for stimulating long-term immunity. They can be vaccinated 2–3 times during the first year of life followed by booster vaccination before parturition to ensure colostral antibodies [20]. Further, a booster dose may be advisable if a vaccinated animal sustains a deep wound.

6. Enterotoxemia

6.1 Etiology

Enterotoxaemia in goats is caused by *Clostridium perfringens,* a gram-positive, non-motile, spore-forming bacilli that grows well in anaerobic or micro-aerophilic conditions. This disease condition tends to be associated mainly with sheep and is of less importance in goats and cattle. *C. perfringens* Type D primarily produce enterotoxamia and Type C sometimes causes sudden death in goats. Grain-fed kids (3–12 weeks old) on a high-concentrate diet are most susceptible, but adult goats may also be affected. Goats are commonly affected with a hemorrhagic form of enterotoxemia.

6.2 Epizootiology

C. perfringens is worldwide in distribution and is found in soil, feces, and in the intestinal tracts of animals and humans. *C. perfringens* types B, C and D may survive in soil as spores for several months. *C. perfringens* type A constitutes a part of the normal intestinal flora and is widely dispersed in soil. Overcrowding and prolonged confinement may increase the spread and severity of the condition.

6.3 Transmission

The *C. perfringens* type D is found as obligate parasite in the intestinal tract of animals [21]. The animals on a high grain diet or on succulent pasture are predisposed to this condition (hence described as 'over-eating disease'). Thus, the disease is more common in well-fed animals in intensive feedlots.

6.4 Clinical signs

The peracute condition is characterized by sudden death of younger and healthy kids. This is occasionally preceded by other signs such as loss of appetite, lack of rumen activity and rumination, bloat, depression and a drunken appearance; the animals may show neurological signs such as incoordination, inability to stand, and convulsions. There may be watery diarrhea and glucosuria. In goat's acute disease is mainly characterized by dysentery, abdominal discomfort and convulsions.

In acute cases of goats, the necropsy findings include pulmonary edema, necrosis of intestinal walls and scattered hyperaemic areas of intestine. Intestinal contents may be green, blood-stained or mucoid, and fibrinous casts may be present in the lumen of the large intestine [22]. Mesentric lymph nodes may be edematous. Fluid accumulation in the pericardial sac, extremely necrotic, soft kidneys ('pulpy kidneys'), focal encephalomalacia, and petechiae of serosa of the brain, diaphragm, gastrointestinal tract and heart are common findings.

6.5 Diagnosis

Diagnosis of enterotoxaemia depends on epidemiological features, type of diet, clinical and pathological features. Gram positive rods can be demonstrated in the smears of intestinal contents or in the lesions of intestine. The culture of bacteria from fecal samples in cooked meat media may be suggestive of the disease (**Figure 4**). Organism on blood agar plates show double zone of hemolysis which is suggestive of *C. perfringens*. Demonstration of the epsilon toxin in the intestinal content is highly reliable method. Protection of mice injected with infiltrates of toxin from intestinal contents against specific antisera is diagnostic. Genotyping by PCR can be used to type isolates of *C. perfringens* as an alternative to in vivo toxin neutralization tests.

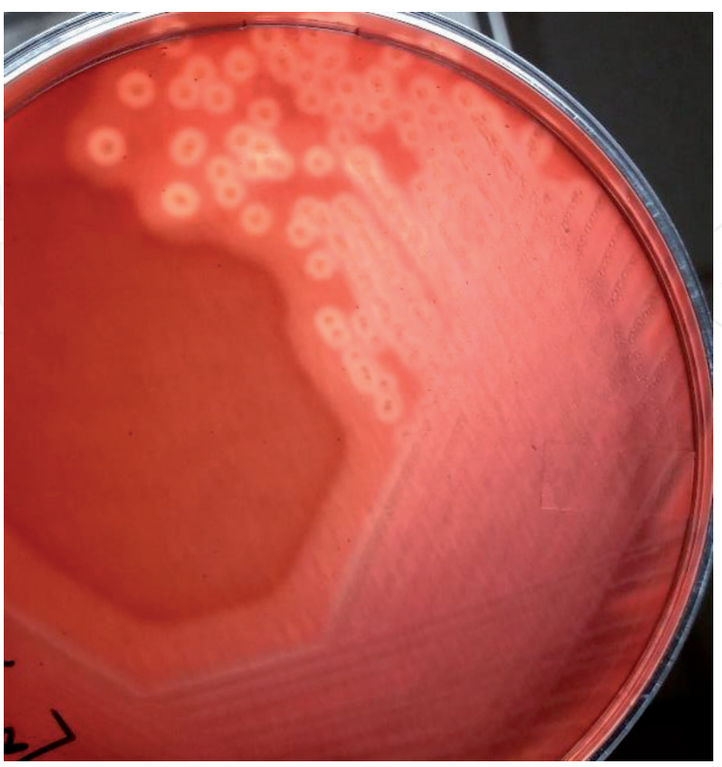

Figure 4.
Double zone hemolytic colonies of Clostridium perfringens on sheep blood agar. This figure is propriety of the authors.

Though ELISA tests can be performed, misdiagnosis may occxur as this test detects low levels of toxin in the intestinal contents of normal animals [23].

6.6 Treatment

Treatment generally is ineffective as most cases are acute in nature. A hyperimmune serum, if available, can be used and a combination of hyperimmune serum along with sulphadimidine has been found useful in goats. Chelating agents can be used to neutralize toxins [21].

6.7 Preventive measures

Vaccination before the anticipated outbreaks is the primary method of control. Alum precipitated formalin killed whole culture toxoid vaccines are commercially available. In ruminants, maternal antibodies last about 5–6 weeks postpartum and hence, the young animals must be vaccinated at this time. Kids are usually vaccinated twice at 4 weeks interval and then re-vaccinated at once in 6 months. However, several anaphylactic reactions have been reported in Sannen kids re-vaccinated with toxoids [24]. Sudden dietary changes and other predisposing factors to enterotoxaemias must be managed. Feeding regimens and feeding of concentrates even to adult goats should be monitored carefully.

7. Johne's disease (JD)

A chronic, contagious, granulomatous disease affecting small intestine of adult ruminants and the affected animals show weight loss and intermittent diarrhea [25].

7.1 Etiology

JD is caused by *Mycobacterium avium* subspecies *paratuberculosis*, a fastidious, acid-fast, gram-positive rod [25].

7.2 Transmission

The organism is present in the environment and animals at young are affected either through ingestion of contaminated milk or direct contact. Infected goats may excrete the bacteria in the feces thereby contaminating the environment [26].

7.3 Clinical signs

The incubation period is usually months to years. Chronic wasting is a characteristic sign in goat and at times pasty feces or diarrhea (in advanced cases) can be witnessed. In advanced cases the animals may lose weight rapidly and will have a hide and bone condition. During PM examination intestine of the affected animals have a corrugated appearance [27].

7.4 Diagnosis

Affected animals can be identified in the herd by intradermal skin testing using Johnin purified protein derivative (PPD). Alternatively, Interferon gamma assay (IGRA) can also be used to assess the cellular immunity. Lymph nodes (Ileal and ileocecal) aspirates, intestinal scrapping can show acid fast bacilli in staining

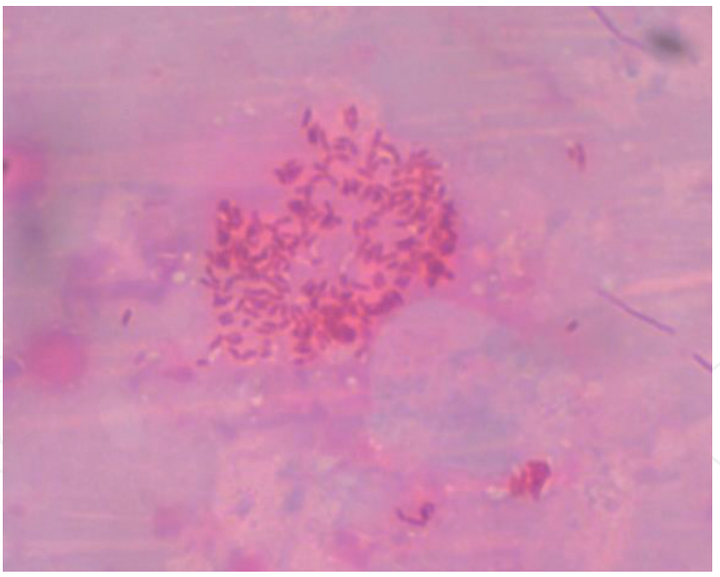

Figure 5.
Acid fast bacilli in intestinal scrapping. This figure is propriety of the authors.

(**Figure 5**). Organism my shed intermittently in feces and hence, bacilli can be found by acid fast staining [27]. Organism can be detected intestinal tissues, lymph node and feces by culture and PCR. Detection of antibody in the later or final stages of the disease can also be attempted for diagnosis.

7.5 Treatment

Treating animals with antimycobacterial agents are not fruitful.

7.6 Preventive measures

Due to its chronic nature, it is difficult to identify the disease early hence, it is advised to test a newly purchased animal before letting into the farm. Test and cull policy is better to break the chain of infection. Suspected animals should be separated from the herd and affected animals milk should not be fed to neonates [25]. The organism may survive longer in the pasture hence, once an animal is found positive it is best to change the pasture land.

7.7 Public health significance

A similar condition in human named as Crohn's disease has been suspected to be caused by *Mycobacterium avium* subspecies *paratuberculosis* still there is no clear evidence for zoonotic transmission of the pathogen [28].

8. Pasteurellosis and Mannheimiosis

Pasteurellosis and Mannheimiosis is an acute fatal disease characterized by pneumonia and septicemia.

8.1 Etiology

Pasteurella multocida and *Mannheimia haemolytica* are aerobic, bipolar, nonmotile, non-spore forming gram-negative rods [29].

8.2 Epizootiology and transmission

P. multocida and *M. haemolytica* are ubiquitous and even present in respiratory tract of healthy animals. Young animals are prone to infection than adults. Stress including weaning, transportation (hence termed as shipping fever), change in diet/weather and overcrowding are the predisposing factors for the condition. Viral diseases can also predispose Pasteurellosis and Mannheimiosis and the organism can be transmitted directly or indirectly through inhalation or ingestion [30].

8.3 Clinical signs

Acute rhinitis or pharyngitis is the common sign noticed in animals. Animals may have high fever, anorexia, and rapid breathing along with profuse mucopurulent nasal/ocular discharges. Kids are more susceptible than adult goats and death may occur without any clinical signs [30]. PM changes include marbling of lungs, pleural adhesion, sero-fibrinous fluid in the thorax, frothy exudate in trachea and also in bronchi.

8.4 Diagnosis

Bipolar organisms of *P. multocida* can be noticed in the impression smears from dead animals (**Figure 6**). Isolation of the organism on selective media and biochemical confirmation is the standard diagnostic procedure. *M. haemolytica* produces hemolytic colonies on blood agar and it can grow on MacConkey agar which are the differentiating features from *P. multocida*. Molecular diagnosis can be carried out by PCR [31].

8.5 Treatment

Use of antibiotics based on antimicrobial susceptibility testing can be used to control the bacterial propagation and anti-inflammatory agents can be used to control fever [30].

8.6 Preventive measures

M. haemolytica and *P. multocida* bacterins can be used as vaccines to prevent the occurrence of the disease [32]. Other measures like reducing overcrowding thereby improving the ventilation in enclosures and also reducing the stress during transportation can prevent the occurrence of the disease.

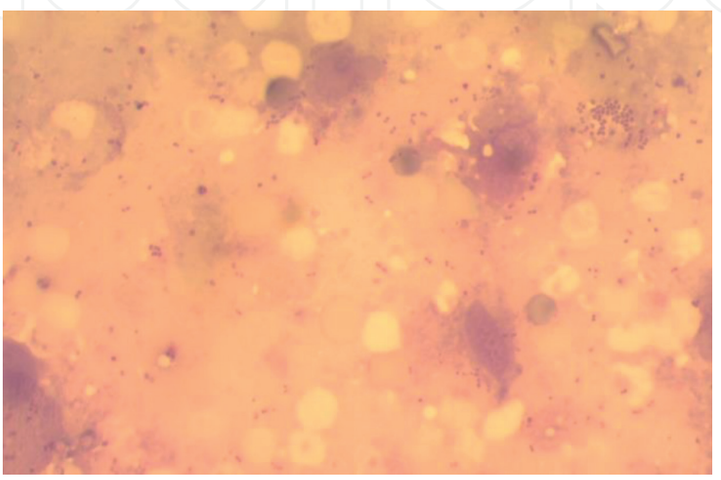

Figure 6.
Bipolar organism in lung impression smear. This figure is propriety of the authors.

9. Caseous lymphadenitis

Caseous lymphadenitis (CLA) is contagious, subclinical and chronic suppurative condition of sheep and goats, occasionally in cattle and is characterized by the formation of abscesses in lymph nodes and visceral organs [33].

9.1 Etiology

CLA is caused by *Corynebacterium pseudotuberculosis*, small, non-motile, non-spore forming, pleomorphic, Gram-positive bacteria which may occur in curved, coccoid, club and rod forms (coryneform morphology) [33]. Two biotypes of *C. pseudotuberculosis* are recognized; ovine/caprine biotypes that lack nitrate-reducing capacity mainly affect sheep and goats, causing superficial and visceral abscesses. The second equine/bovine biotype usually reduce nitrate and mainly affects horses and cattle, causing ulcerative lymphangitis.

9.2 Epizootiology

CLA is worldwide in distribution and the probable dissemination of the disease throughout the world occurred through importation of infected animal [34]. This disease is found in parts of North and South America, Australia, New Zealand, the Middle East, Asia and Africa and is being reported more often in Britain and other European countries.

9.3 Transmission

The bacteria can survive in the environment for about 6 months or more. Transmission can occur either through direct or indirect contact or through wounds contaminated with pus from the abscesses of infected animals. The organism enters through contamination of skin wounds arising from castration, ear tagging or tattooing, docking or shearing operations. Arthropod bites or contaminated dips can also be the source of infection [34]. Goats having traumatized buccal mucosa have more chances of taking the bacterium from contaminated feed. The organism has also been isolated from the milk of affected goats.

9.4 Clinical signs

The incubation period varies from weeks to months; usually is about 3 months. CLA may be manifested in two forms: in its superficial form it is characterized by infection of peripheral lymph nodes, such as the submandibular, parotid, prescapular and supramammary lymph nodes (**Figure 7**). These peripheral lymph nodes enlarge, may erode and eventually leads to formation of abscess in chronic cases. Visceral form is characterized by abscessation of internal organs, such as lungs, liver, kidneys, uterus, spleen and internal lymph nodes (mainly mediastinal and bronchial lymph nodes) that may not be detectable antemortem [35]. These two forms can co-exist; however, the visceral form is more common among sheep, while superficial form is more frequent among goats with external abscesses in the lymph nodes particularly of the head and neck regions.

Eventually, the affected animal become exercise-intolerant, anorectic, ill-thrift and debilitated (often known as thin-ewe syndrome in sheep). Fever, increased respiratory rates, and pneumonia may also be noticed. Morbidity up to 15% is common, and morbid animals will often eventually succumb to the disease. The infection can also lead to abortion in doe and orchitis and/or epididymitis in bucks.

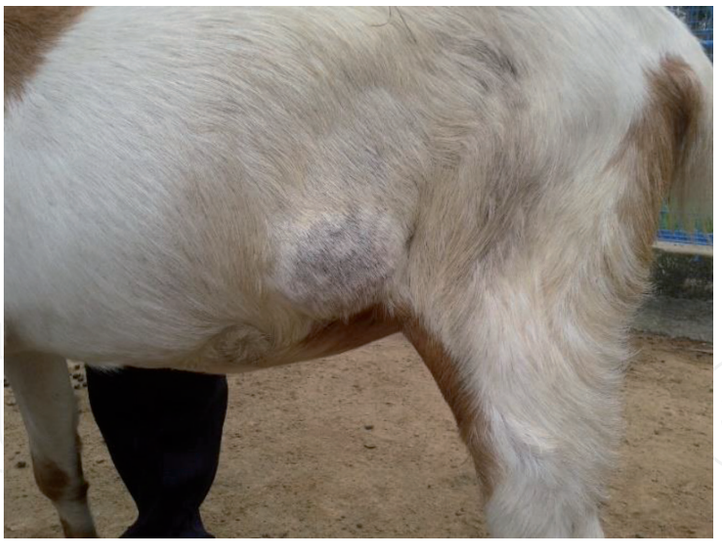

Figure 7.
Lymph node enlargement in goats noted in caseous lymphadenitis. This figure is propriety of the authors.

Though less common, orchitis can be acute in which the buck develops fever, reduced appetite, lack of walking ability and loss of libido. The infected testes appear swollen, hot and painful to touch.

9.5 Diagnosis

Diagnosis is based on clinical signs and lesions and abscessation of both superficial and visceral lymph nodes is typical. Radiographs may be useful in identifying affected central nodes which also must be confirmed by culture of tracheal washings. Gram and Giemsa staining can be used for identification of the bacteria. Isolation of organism from purulent material from abscessed lymph nodes in case of live animals and /or from abscesses of internal organs from dead animals. ELISA tests which detect antibodies directed against either cell wall antigens or the exotoxin (Phospholipase D - PLD) are available [34]. Further, the detection of INF-γ by ELISA, an indicator of cell-mediated immunity, has also been potentially used for demonstration of CLA in eradication programs. Molecular techniques such as PCRs targeting 16S rDNA, *rpo* and *pld* genes have also been used in the recent years for the diagnosis of caseous lymphadenitis.

9.6 Treatment

Though *C. pseudotuberculosis*, in vitro is susceptible to antibiotics, the antibiotic therapy is usually not much effective in animals. The chronic nature of infection, the intracellular location of the bacteria and the formation of biofilm in natural infections reduce the antibiotic efficacy, making them useless. Draining of abscesses, followed by cleansing and chemical cauterization with 10% iodine may be helpful or the localized abscesses may be removed entirely from valuable animals [34].

9.7 Preventive measures

As CLA is contagious in nature, the animals with draining and punctured lesions should be kept isolated until healed. Reducing the environmental contamination, proper sanitation and biosecurity of facilities and instruments and safety measures to prevent injuries are all important in control. The causative agent is sensitive to

common disinfectants such as hypochlorite, formalin and cresol; however, the surfaces should be cleaned before disinfection, as organic matter usually interferes with the action of these agents. The control measures vary with the prevalence of infection. In countries with a high incidence, rigorous sanitary procedures must be implemented, along with vaccination. Disease eradication can be achieved in endemically-infected herds by test and disposal policy [36].

Most of the commercially available vaccines contain inactivated PLD of either *C. pseudotuberculosis* or of other pathogens, such as *Clostridium* species. Glanvac vaccine (Vetrepharm, Inc. London) is licensed for use in sheep and goats in Canada, Australia and New Zealand. However, the use of PLD toxoid in goats may result in some adverse consequences such as reduction in milk, fever, ventral edema, ataxia and convulsions; therefore, its use is restricted [34]. On the other hand, live vaccines targeting the attenuation of PLD gene, confers the best and longest-lasting immune response, due to its similarity to natural infection.

9.8 Public health significance

Human beings are rarely affected, some cases of human infections have been documented as occupational infection in veterinary doctors and assistant as well as farm experts.

10. Contagious caprine pleuropneumonia

Contagious caprine pleuropneumonia (CCPP) is a highly contagious and rapidly spreading mycoplasmal disease of goat, occasionally sheep and wild ruminants. CCPP is characterized by severe sero-fibrinous pleuropneumonia, very high morbidity (100%), and mortality (80–100%) and results in heavy economic losses.

10.1 Etiology

CCPP is caused by *Mycoplasma capricolum* subspecies *capripneumoniae* (Mccp), which was earlier known as *Mycoplasma* biotype F38. It belongs to the class *Mollicutes* that lack cell wall. *M. ovipneumoniae*, *M. mycoides* subspecies *capri*, and *M. mycoides* subspecies *mycoides* (Large Colony Type) are also considered as etiological agents of caprine pneumonia in the United States [1].

10.2 Epizootiology

CCPP is becoming a novel emerging and rapidly spreading disease in most parts of the world and at present, goat populations in more than 40 countries are affected with CCPP and sporadic cases of CCPP are also being reported from many more countries [37]. It mostly occurs in countries of Africa, Middle East and Asia.

10.3 Transmission

The disease is highly contagious and main mode of transmission is through inhalation of infected aerosols. The direct contact with affected animals is the main source of transmission. Airborne transmission can result in distant spread of about 50 m distance. However, the shorter survival time (3–14 days) of the organisms in external environment limits transmission of Mccp [38]. Yet under cold, moist and overcrowded environment these bacteria can persists for longer durations and may lead to severe outbreaks mostly in winter.

10.4 Clinical signs

CCPP is strictly a respiratory illness and is characterized by severe dyspnea, nasal discharge, cough, and fever. This can occur in peracute, acute and/or chronic forms in endemic areas. In peracute form, affected goats may die within 1–3 days without premonitory clinical signs. In acute infection, the initial signs are high fever (41–43°C), lethargy and anorexia, followed within 2–3 days by coughing and laboured breathing. The cough is frequent, violent and productive. In the final stages of infection, the goat may not be able to move and stands with its front legs wide apart and its neck stiff and extended [37]. Saliva can drip continuously from the mouth, and the animal may exhibit grunt or bleat in pain. Frothy nasal discharge and stringy saliva may be seen terminally. Pregnant goats may abort. Acutely affected goats generally die within seven to 10 days. In the chronic cases, there is chronic cough, nasal discharge and debilitation. These forms with resembling clinical signs in goats were also reported from captive wild goats.

Pathological features during necropsy are also limited to respiratory system. Acute form is characterized by unilateral pneumonia and sero-fibrinous pleuritis with straw colored fluid in the thorax. The lung is granular with copious straw-colored exudates oozing out on cut section. Pea-sized, yellow-colored nodules may be noticed in lungs and these nodules are surrounded by areas of congestion. Varying degrees of lung consolidation or necrosis may also be noticed [37]. The regional lymph nodes mainly bronchial lymph nodes are enlarged. Some long-term survivors reveal chronic pleuropneumoniae or chronic pleuritis, with encapsulation of acute lesions and numerous adhesions to the chest wall. The interlobular septa are not usually thickened in domesticated goats.

10.5 Diagnosis

CCPP can be diagnosed based on cultural, biochemical, serological, and molecular methods following a tentative clinical diagnosis. Ultrasonography and X-rays may help in diagnosis and CCPP-associated changes may be evident in lungs, pleura, thorax, and associated structures. Cultural isolation and identification ('fried egg-like appearance' of the colonies under microscope), though is conventional but is still considered as standard method for detection of Mccp from lung tissue and/or pleural fluid at necropsy. Due to the difficulty in isolation, PCR is the technique of choice for the diagnosis of CCPP. The agglutination tests, ELISA, FAT, CFT (most widely used), passive or indirect haemagglutination tests (IHT) are the immunological methods employed for diagnosis of CCPP [38]. Latex agglutination test is being increasingly used in diagnostic laboratories as a pen side test. It can used to test whole blood as well as serum.

10.6 Treatment

Tylosin is considered the drug of choice against Mccp. Further, oxytetracycline is also found effective when administered in early stages of infection. However, some infections are slow to resolve.

10.7 Preventive measures

In endemic areas, proper care should be taken while introducing new goats into the flock. Flock testing, slaughter, and on-site quarantine may be helpful in controlling the spread of disease. Vaccines available in some areas may help in prevention

of the disease. The commercially available CCPP vaccine containing inactivated Mccp suspended in saponin provides protection for over 1 year [37].

11. Dermatophilosis

Dermatophilosis is a chronic, exudative and sometimes proliferative dermatitis occurs in domestic ruminants, wild animals and occasionally in human beings. Also known as Cutaneous streptothricosis, Strawberry foot rot or Lumpy wool.

11.1 Etiology

Dermatophilosis is caused by *Dermatophilus congolensis*, which is a gram positive, nonacid-fast, facultative anaerobic actinomycete that produces motile zoospores.

11.2 Epizootiology

The disease occurs worldwide and is more common in tropics and subtropics. The organism is believed to be a saprophyte of soil and persists in dry scabs and crusts, to survive for up to 42 months. It has been reported from many countries, but occurs particularly in humid climates and areas where ticks of the genus *Amblyomma* are endemic [39].

11.3 Transmission

Transmission occurs by direct contact with infected animals. The infection can be transmitted indirectly by mechanical vectors (ectoparasites) and also through intradermal inoculation by contaminated thorny bushes. The pathogenesis may be influenced by factors such as mechanical injury to the skin, rainfall, tick infestation, concurrent diseases and/or stresses that compromise the host's immune system.

11.4 Clinical signs

The disease is painful but non-pruritic, and is characterized by exudative, proliferative or hyperkeratotic dermatitis, accompanied by the production of crusts and folliculitis. In sheep, it may be seen in two forms: mycotic dermatitis (lumpy wool) and strawberry foot rot. While in goats and cattle, similar signs of crusty, suppurative dermatitis are seen and are often referred as cutaneous streptothricoses. The skin lesions appear raised, thick, yellow-brown colored discrete or confluents crusts containing matted hair. Sometimes may be seen in nodular form also with discrete encrustation of scab. The whole body may be affected but less hairy parts such as ears, axilla, scrotum, prepuce, ventral abdomen, limbs etc., show severe lesions [40]. Lesions in younger goats are mostly seen along the tips of the ears and under the tail. Most affected animals will recover within 3–4 weeks and lesions have little effect on overall health. In severe generalized infections, the animals often loose condition. If there are lesions at the feet, lips and muzzle, the movement of animals and eating become difficult.

11.5 Diagnosis

Diagnosis of dermatophilosis is mainly based clinical signs particularly based on the appearance of the characteristic skin lesions. The same can be confirmed

by the demonstration of the organism from the lesions beneath the scabs. The softened scab materials stained by the Giemsa method, reveal the characteristic branching filaments containing zoospores. The organism can be cultured on blood agar at 37°C under 2.5–10% CO_2 for up to 5 days and Haalstra technique based on chemotaxis of the zoospores to CO_2 can be employed for efficient recovery of the organism.

11.6 Treatment

Animals can be treated with antibiotics such as high doses of penicillin or long acting tetracyclines. Topical applications alone are ineffective. Antibiotic therapy is augmented by topical treatment with lime sulfur as well as control of ectoparasites and biting flies. Povidone iodine shampoos or chlorhexidine solutions also help in clearing the disease.

11.7 Preventive measures

Control measures are based on minimizing the effects of predisposing factors and prompt treatment of affected goats. Animals with skin lesions must be isolated and treated at the earliest. Minimizing moist conditions (such as providing shelter during rainfall) is helpful in control and prevention. Grazing management especially removal of thorny bushes in pasture land that damages skin will also help. Prophylactic antibiotic therapy can also be given.

11.8 Public health significance

D. congolensis is a zoonotic organism and rare human infections have occurred from handling diseased animals.

12. Foot rot

A contagious, either acute or chronic dermatitis of the hoof and its underlying tissues leading to lameness [41].

12.1 Etiology

Foot rot is caused by *Dichelobacter nodosus* and *Fusobacterium necrophorum*, anaerobic, non-spore forming, gram negative rods.

12.2 Epizootiology

The organism *F. necrophorum* is ubiquitous in nature while *D. nodosus* is obligate pathogen, can be present in skin and hoof of animals and cannot survive much longer in environment. Moist environment, humid condition, wet grounds and overcrowding are the predisposing factors for foot rot [42].

12.3 Clinical signs

Interdigital region will be moist and will have a foul odor due to necrosis (**Figure 8**). Lameness is the common sign of foot rot. Based on the severity of the infection animals may lose weight due to anorexia and there will be decrease in production [43].

Figure 8.
Moist, necrotic interdigital region seen in foot rot condition. This figure is propriety of the authors.

12.4 Diagnosis

Diagnosis is based on clinical signs and isolation of organism from the foot lesions. Since the organisms are anaerobic isolation is tricky and hence molecular diagnosis like PCR can be used for diagnosis.

12.5 Treatment

Hooves of the animals should be trimmed so as to remove the necrotic material thereby eliminating the anaerobic environment. Local antibiotics may be applied to the affected hoof after trimming. 10% zinc or copper sulfate or 10% formalin can be used for footbath [44].

12.6 Preventive measures

D. nodosus, though present in epidermal tissues of the hoof, survives for less than 7 days in the environment and hence, affected animals should be separated from the herd to prevent spread to other animals. Regular hoof trimming and cleaning should be practiced. Bacterins can be used as vaccines to prevent the infection.

13. Conclusions

Goat is called as poor man's cow but there are various bacterial diseases that cause economic loss to the goat farmers. Serval bacterial diseases cause acute infection hence there will be sudden onset of infection leading to huge mortality. Measures like use of vaccines before onset of disease, good management practices, etc., are essential to prevent the disease outbreaks. Animals with infection or clinical signs should be separated from rest of the animals so that infectious pathogens do not transmit to naïve animals and it is also recommended to quarantine newly purchased animals before admitting them into the farm. These practices can curtail

the spread of infectious agents. It is also advisable to screen for diseases before purchasing the animals to the farm. Diseases like TB, JD and brucellosis should be screened before the purchase since these diseases are chronic in nature hence can remain undiagnosed. Animals infected with diseases that can affect human like anthrax, brucellosis, etc., should be handled carefully and better bio-security measures should be followed to prevent spread of disease within herd and also to human beings. Most of the bacterial infection can be treated with antimicrobial agents but these agents should be used judiciously because in the recent times antimicrobial resistance is a major problem.

Conflict of interest

The authors declare no conflict of interest.

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References

[1] Underwood WJ, Blauwiekel R, Delano ML, Gillesby R, Scott A, Mischler SA, Schoell, A. Biology and Diseases of Ruminants (Sheep, Goats, and Cattle). Editor(s): Fox JG, Anderson LC, Otto GM, Pritchett-Corning KR, Whary MT, In American College of Laboratory Animal Medicine, Laboratory Animal Medicine (Third Edition): Academic Press; 2015. p. 623-694, https://doi.org/10.1016/B978-0-12-409527-4.00015-8.

[2] Rahman MT, Sobur MA, Islam MS, Ievy S, Hossain MJ, El Zowalaty ME, Rahman AT, Ashour HM. Zoonotic Diseases: Etiology, Impact, and Control. Microorganisms. 2020;8(9):1405.

[3] Shafazand S, Doyle R, Ruoss S, Weinacker A, Raffin TA. Inhalational Anthrax: Epidemiology, Diagnosis and Management. Chest. 2001; 116: 1369-1376.

[4] Misgie F, Atnaf A, Surafel K. A Review on Anthrax and its Public Health and Economic Importance. Academic Journal of Animal Diseases. 2015; 4(3): 196-204, 2015

[5] Sekar N, Shah NK, Abbas SS, Kakkar M. Research options for controlling zoonotic disease in India, 2010-2015. PLoS ONE 2011: 6.

[6] Siddiqui, MA, Khan MAH, Ahmed SS, Anwar KS, Akhtaruzzaman SM, Salam MA. Recent outbreak of cutaneous anthrax in Bangladesh: Clinico-demographic profile and treatment outcome of cases attended at Rajshahi Medical College Hospital. BMC Res. Notes 2012; 5: 464.

[7] Alton GG. Brucellamelitensis. In: "Animal brucellosis", editors Nielsen K, Duncan JR. CRC Press. Boston; 1990. p. 383-409.

[8] Corbel MJ. Brucellosis: an overview. Emerg. Infect. Dis. 1997; 3: 213-221.

[9] Alton GG, Jones LM, Angus RD, Verger JM. Techniques for the brucellosis laboratory. INRA, Paris, France; 1988. p. 192

[10] Durán-Ferrer M. Comparación entre métodos inmunológicos de diagnóstico de la brucelosis ovina por Brucella-melitensis y eficacia de la inmunización de ovejas adultas con la vacuna Rev.1 por vía conjuntival. PhDThesis, University of Murcia, Spain; 1988.

[11] Neta AVC, Mol JPS, Xavier MN, Paixão TA, Lage AP, Santos RL. Pathogenesis of bovine brucellosis. The Veterinary Journal. 2010; 184(2): 146-155.

[12] Biancifiori F, Nannini D, Di Matteo A, Belfiore P. Assessment of an indirect ELISA in milk for the diagnosis of ovine brucellosis. Comp. Immunol. Microb. Infec. Dis. 1996; 19: 17-24.

[13] González D, Grilló MJ, De Miguel MJ, Ali T, Arce-Gorvel V, Delrue RM, Conde-Álvarez R, Muñoz P, López-Goñi I, Iriarte M, Marín CM, Weintraub A, Widmalm G, Zygmunt M, Letesson JJ, Gorvel JP, Blasco JM, Moriyón I. Brucellosis Vaccines: Assessment of *Brucella melitensis* Lipopolysaccharide Rough Mutants Defective in Core and O-Polysaccharide Synthesis and Export. PLoS ONE. 2008; 3(7): e2760. doi:10.1371/journal.pone.0002760.

[14] Kochar DK, Gupta BK, Gupta A, Kalla A, Nayak KC, Purohit SK. Hospital-based case series of 175 cases of serologically confirmed brucellosis in Bikaner. J Assoc Physicians India. 2007; 55:271-275.

[15] Muralidharan J, Ramesh V, Saravanan S. Tetanus in sheep of an organized livestock farm- a case report. Indian Journal of Field Veterinarians 2010; 5:43-44.

[16] Harish BR, Chandranaik BM, Bhanuprakash RA, Jayakumar SR, Renukaprasad C, Krishnappa G. *Clostridium tetani* infection in goats. Intas Polivet. 2006; 7: 72-74.

[17] Pugh DG, Baird AN. Sheep and Goat Medicine,2nd edn. Saunders, an imprint of Elsevier Inc:Philadel-phia, PA; 2012. p. 621

[18] Popoff MR. Tetanus in animals. J Vet Diagn Invest. 2020;32(2):184-191. doi: 10.1177/1040638720906814.

[19] Lotfollahzadeh S, Heydari M, Mohebbi MR, Hashemian M. Tetanus outbreak in a sheep flock due to ear tagging. Vet Med Sci. 2019;5(2):146-150. doi: 10.1002/vms3.139. Epub 2018 Dec 13. PMID: 30549234; PMCID: PMC6498517.

[20] Centers for Disease Control and Prevention. Epidemiology and Prevention of Vaccine Preventable Diseases. Hamborsky J, Kroger A, Wolfe S, eds. 13th ed. Washington DC: 348 Public Health Foundation, 2015.

[21] Blackwell TE, Butler DG. Clinical signs, treatment, and postmortem lesions in dairy goats with enterotoxemia: 13 cases (1979-1982). J Am Vet Med Assoc. 1992;200(2):214-217. PMID: 1559880.

[22] Karthik K, Manimaran K, Bharathi R, Shoba K. Report of enterotoxaemia in goat kids. Adv. Anim. Vet. Sci. 2017; 5(7): 289-292.

[23] Uzal FA, Songer JG. Diagnosis of Clostridium perfringens intestinal infections in sheep and goats. Journal of Veterinary Diagnostic Investigation. 2008; 20: 253-265.

[24] Kusiluka L, Kambarage D. Diseases of small ruminants in Sub-Saharan Africa - A handbook. 1996. p. 25-62

[25] Sweeney RW, Collins MT, Koets AP, McGuirk SM, Roussel AJ. Paratuberculosis (Johne's disease) in cattle and other susceptible species. J Vet Intern Med. 2012;26(6):1239-1250. doi: 10.1111/j.1939-1676.2012.01019.x. Epub 2012 Oct 28. PMID: 23106497.

[26] McGregor H, Dhand NK, Dhungyel OP, Whittington RJ. Transmission of Mycobacterium avium subsp. paratuberculosis: dose–response and age-based susceptibility in a sheep model. Prev Vet Med. 2012;107(1-2): 76-84.

[27] Whittington R, Donat K, Weber MF, Kelton D, Nielsen SS, Eisenberg S, Arrigoni N, Juste R, Sáez JL, Dhand N, Santi A, Michel A, Barkema H, Kralik P, Kostoulas P, Citer L, Griffin F, Barwell R, Moreira MAS, Slana I, Koehler H, Singh SV, Yoo HS, Chávez-Gris G, Goodridge A, Ocepek M, Garrido J, Stevenson K, Collins M, Alonso B, Cirone K, Paolicchi F, Gavey L, Rahman MT, de Marchin E, Van Praet W, Bauman C, Fecteau G, McKenna S, Salgado M, Fernández-Silva J, Dziedzinska R, Echeverría G, Seppänen J, Thibault V, Fridriksdottir V, Derakhshandeh A, Haghkhah M, Ruocco L, Kawaji S, Momotani E, Heuer C, Norton S, Cadmus S, Agdestein A, Kampen A, Szteyn J, Frössling J, Schwan E, Caldow G, Strain S, Carter M, Wells S, Munyeme M, Wolf R, Gurung R, Verdugo C, Fourichon C, Yamamoto T, Thapaliya S, Di Labio E, Ekgatat M, Gil A, Alesandre AN, Piaggio J, Suanes A, de Waard JH. Control of paratuberculosis: who, why and how. A review of 48 countries. BMC Vet Res. 2019;15(1):198. doi: 10.1186/s12917-019-1943-4. PMID: 31196162; PMCID: PMC6567393.

[28] McNees AL, Markesich D, Zayyani NR, Graham DY. Myco-bacterium paratuberculosis as a cause

of Crohn's disease. Expert Rev Gastroenterol Hepatol. 2015;9(12):1523-1534. doi:10.1586/17474124.2015.1093931.

[29] Mohamed RA, Abdelsalam EB. A review on pneumonic pasteurellosis (respiratory mannheimiosis) with emphasis on pathogenesis, virulence mechanism and predisposing factors. Bulg. J. Vet. Med. 2008;11(3):139-160.

[30] Laishevtsev AI. IOP Conf. Ser. Earth Environ. Sci. 2020: 548 072038.

[31] Hussain R, Mahmood F, Ali HM, Siddique AB. Bacterial, PCR and clinico-pathological diagnosis of naturally occurring pneumonic pasturellosis (mannheimiosis) during subtropical climate in sheep. Microb Pathog. 2017;112:176-181. doi: 10.1016/j.micpath.2017.09.061. Epub 2017 Sep 29. PMID: 28970175.

[32] Srinand S, Hsuan SL, Yoo HS, Maheswaran SK, Ames TR, Werdin RE. Comparative evaluation of antibodies induced by commercial *Pasteurella haemolytica* vaccines using solid phase immunoassays Veterinary Microbiology. 1996; 49:181-195.

[33] Kuria JK, Mbuthia PG, Kang'ethe EK, Wahome RG. Caseous lymphadenitis in goats: the pathogenesis, incubation period and serological response after experimental infection. Vet Res Commun. 2001; 25(2):89-97. doi: 10.1023/a:1006400617235. PMID: 11243659.

[34] Osman AY, Nordin ML, Kadir AA, Saharee AA. The Epidemiology and Pathophysiology of Caseous Lymphadenitis: A Review. J. Vet. Med. Res. 2018; 5: 1129.

[35] Fontaine MC, Baird GJ. Caseous lymphadenitis. Small Rumin. Res. 2008; 76: 42-48.

[36] Guimarães AS, Carmo FB, Heinemann MB, Portela RWD, Meyer R, Lage AP, Seyffert N, Miyoshi A. Azevedo V, Gouveia AM. High sero-prevalence of caseous lymphadenitis identified in slaughterhouse samples as a consequence of deficiencies in sheep farm management in the state of Minas Gerais, Brazil. BMC Vet Res. 2011; 7:68. doi: 10.1186/1746-6148-7-68.

[37] Yatoo MI, Parraya OR, Bashir ST, Muheet RA, Bhat, Gopalakrishnan A, Karthik K, Dhama, K, Singh SV. Contagious caprine pleuropneumonia – a comprehensive review. Veterinary Quarterly, 2019; 39 (1): 1-25

[38] OIE. World Organisation for Animal Health – contagious caprine pleuropneumonia. In Manual of Diagnostic Tests and Vaccines for Terrestrial Animals, Chapter 2.7.5. Paris; 2014.

[39] Loria GR, La Barbera E, Monteverde V, Sparagano OAE, Caracappa S. Dermatophilosis in goats in Sicily. Veterinary Record. 2005; 156: 120-121

[40] Msami HM, Khaschabi D, Schöpf K, Kapaga AM, Shibahara T. Dermatophilus congolensis infection in goats in Tanzania. Trop Anim Health Prod. 2001 Oct;33(5):367-377. doi: 10.1023/a:1010587621843. PMID: 11556616.

[41] Kaler J, Green LE. Naming and recognition of six foot lesions of sheep using written and pictorial information: A study of 809 English sheep farmers. Prev. Vet. Med. 2008; 83(1):52-64.

[42] Green LE, George TRN. Assessment of current knowledge of foot rot in sheep with particular reference to *Dichelobacter nodosus* and implications for elimination or control estrategies for sheep in Great Britain. Vet. Journal 2008; 175:173-180.

[43] Bennett G, Hickford J, Sedcole R, Zhou H. *Dichelobacter nodosus, Fusobacterium necrophorum* and the epidemiology of footrot. Anaerobe. 2009; 15: 173-176

[44] Abbott KA, Lewis CJ. Current approaches to the management of ovine footrot. The Veterinary Journal. 2005; 169: 28-41