Recognizing Common Infectious Diseases of Small Ruminants

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Abstract

Caseous lymphadenitis, paratuberculosis, ovine progressive pneumonia, caprine arthritis-encephalitis, soremouth, infectious keratoconjunctivitis and foot rot are important infectious diseases of small ruminants. Although diagnosis is often based on clinical signs alone, laboratory testing to detect subclinically infected animals and culling or segregation are required for control or eradication of the more chronic diseases.

Introduction

Currently many new sheep and goat operations are being established, often by producers who lack previous experience with small ruminants. These people assemble a herd before they learn enough about common diseases to avoid introduction of serious pathogens to the farm. They naively assume that an interstate health certificate equates to freedom from disease, or else they buy and transport animals without any evaluation of the animals or the flock of origin. Practitioners will need to discuss the concept of quarantine with these clients or potential clients. Three weeks would be a bare minimum quarantine period for a purchased or borrowed animal or one returning from a show or breeding farm. The new animals should be housed in readily cleaned stalls and handled last, using dedicated boots and coveralls. Serologic testing for selected chronic diseases should be done (or repeated) during quarantine. After at least three weeks the animals should be re-examined for any signs of illness, including poor body condition, enlarged lymph nodes, swollen joints, crusty skin lesions, keratoconjunctivitis, footrot or foot scald. Animals that still appear healthy can then be released into a small group of animals already on the farm. The mixed group should be observed for disease activity in the sheep or goats from the home farm (if the purchased animals are carrying new pathogens) and the new arrivals (if they are naïve to endemic diseases on the farm).

New owners who lack medical training will also need an introduction to the germ theory, to sanitation of boots and equipment (including single use of sterile needles), to the concepts of carrier animals and prolonged incubation times, and to the risks of disease transmission associated with colostrum, milk and animals of other species. Often information reaches the new owner filtered through the seller who either is unaware of the existence or importance of certain infectious diseases or holds to the ethics of "buyer beware".

This paper reviews the diagnosis and treatment of several of the most important infectious diseases of small ruminants. Abortion diseases and resistant parasites are not discussed here, but should be addressed by herd health programs.

Caseous Lymphadenitis, CLA

Corynebacterium pseudotuberculosis enters the body through small breaks in skin or mucous membranes or through shearing wounds and localizes in a regional lymph node. The incubation period until abscesses are noted in superficial lymph nodes is typically two to six months or longer. These abscesses may rupture and drain spontaneously. It is common for one or more additional lymph nodes, following the lymphatic drainage pattern, to develop abscesses several months later. Internal (visceral) abscesses, especially in the lungs, may develop if the organism reaches the thoracic lymph duct or if it is inhaled.

The ewe or goat with only external abscesses typically shows no clinical signs other than enlargement and abscessation of one or more peripheral lymph nodes. Most sheep infected during the shearing process have involvement of the superficial cervical (prescapular) or subiliac (prefemoral) or popliteal nodes. Goats often develop abscesses of the head (parotid, mandibular nodes) and neck, presumably from infection of wounds experienced during fighting or browsing. The pus in an abscessed node may be white, yellowish, or greenish, creamy, pasty, or cheesy; it is typically odorless. Internal abscesses are often associated with pneumonia or wasting.

Diagnosis is based on the presence of a firm to slightly fluctuant subcutaneous swelling in the anatomic location of a lymph node. When a flock history is lacking, insert a needle through shaved, disinfected skin into the mass. If aspiration yields nothing, the

needle is withdrawn and saline is flushed through it to obtain material for a stained smear and culture. Pus can leak back out the needle hole so the animal should be handled as if caseous lymphadenitis were present until culture results become available. Cultures of pus from draining caseous lymphadenitis lesions are frequently overgrown. Serologic tests[a] are of value in identifying sheep and goats with early (no abscess yet developed) or internal forms of caseous lymphadenitis. However, to date, tests have not been specific enough for current infection to justify their use for culling decisions. Testing is of value in avoiding the purchase of infected animals.

Treatment of individual animals involves either lancing or surgically removing the abscessed nodes. Wear gloves, as the infection is potentially zoonotic. Isolate the animal until the lesion is completely covered by healthy skin, typically 20 to 30 days later. Surgical removal of the encapsulated abscess offers the advantage that the treated animal does not need to be quarantined afterwards. Extirpation of abscessed parotid nodes is, however, dangerous. Abscesses on pet animals can be treated by injecting them with formalin. When the abscess is ripe and attached to the overlying skin, a 14 gauge needle is inserted into the dorsal aspect of the abscess. A 35 ml syringe containing 20 ml of 10% formalin is used to repeatedly inject formalin into the abscess and aspirate and reinject the resulting admixture of formalin and pus. The encapsulated abscess will eventually be sloughed from the body. Avoid formalin treatment if the abscess is not fixed to the overlying skin or if the animal is being used for food production.

A commercial vaccine[b] developed in Australia and available in Canada, but not the US, reduces the number of infected sheep or goats and the number and size of abscesses. Another vaccine[c] is marketed for sheep in the United States and is best given to replacement animals. Vaccination should probably be performed before four months of age, but colostrally derived antibody may interfere when vaccination is begun before three months. Lambs and kids are given two doses and then annual boosters. Severe reactions may occur in adult dairy goats if vaccinated with the Colorado Serum Co. product, especially if these animals are already infected with CLA.

Eradication of caseous lymphadenitis from a herd is difficult. The owner must be willing to cull animals with multiple abscesses and forego purchase of animals from infected herds. A herd that vaccinates should be presumed to be doing so because the disease is endemic. Valuable infected animals may be kept isolated; their offspring should be removed at birth. Needles, tattooers and surgical instruments should be sterilized between animals. As shearing is a major mode of transmission in sheep, avoid introduction of the organism on shearing equipment. Shear the youngest sheep first, as these animals are least apt to be infected. Disinfect clipper blades before shearing is begun and any time an abscess is entered with the clippers. Animals with chronic respiratory disease or wasting should be culled, or at least isolated from the flock.

Paratuberculosis

Paratuberculosis, or Johne's disease, is caused by *Mycobacterium avium* subsp *paratuberculosis*, previously and in most circles still referred to as *M. paratuberculosis*. Cattle strains will grow in culture after 6-12 weeks of incubation. Sheep strains rarely grow in standard culture media. The cattle type is more common in goats but does occur in sheep.

The primary mode of transmission is fecal-oral. Young animals are most susceptible. Transmission *in utero* and potentially via milk has been documented. The incubation period is usually at least one year and appearance of clinical disease is hastened by relocation, pregnancy/parturition, or inadequate nutrition. Fecal shedding usually precedes clinical signs. The typical range for clinical disease is two to seven years of age; however, in some heavily infected goat herds clinical infection and fecal shedding occur as early as one year of age. Excellent nutrition may delay the appearance of clinical signs.

The primary sign of Johne's disease in sheep and goats is chronic weight loss. Only 10-20% of clinical cases exhibit diarrhea or clumping of feces in the end stages of the disease. A drop in milk production is a common early sign in dairy goats. Animals with paratuberculosis often show mild anemia (packed cell volume below 25%) and mild to marked hypoproteinemia. [Spin sheep and goat blood samples 10 minutes to get an accurate packed cell volume.] Hypoproteinemia can result in bottle-jaw which mimics the effects of endoparasitism. As a heavier than average parasite load often accompanies the terminal stages of paratuberculosis, the two conditions compound each other. Do necropsies on any emaciated animals that die under eight years of age. Look for dilated lymphatics on the intestines and mesentery. Only rarely is there marked thickening of the intestinal mucosa.

Fecal culture will detect 40-60% of infected animals in goat herds with clinical infection. In sheep flocks, the classic fecal culture is very insensitive and rarely yields a positive; as a result it is an impractical diagnostic test. Acid-fast smears of feces will identify approximately 55% of histopathologically positive sheep with clinical signs of paratuberculosis. The best tissues for postmortem diagnosis are the ileocecal junction and ileocecal and mesenteric lymph nodes. These should be submitted for culture (except in sheep) and for histol-

ogy. Acid-fast stains of impression smears of these tissues are often diagnostic.

The agar gel immunodiffusion (AGID) test and fecal culture can be used together to detect infected animals. Believe the positive! Screen the herd with the AGID and then do fecal cultures on AGID negative animals over one year of age to identify light shedders. The frequency of testing depends on the herd owner's goals (control vs eradication) and economics (labor of sample collection and test costs). Commercial ELISA tests for paratuberculosis in cattle are not adequately validated for small ruminants and should not be used.

There is no treatment for Johne's disease. Improved nutrition and supportive care of a clinically affected pet or breeding animal can certainly prolong its life but will also greatly increase environmental contamination. As the organism persists in the soil, keeping one animal alive may eventually lead to the premature death of many others. Also, the risk of *in utero* transmission in clinically affected animals and our inability to detect these infections in a timely fashion should discourage the rebreeding of any known infected animals except for the production of slaughter animals.

Once the disease has been diagnosed in a herd or flock, test and cull if feasible. Body condition score regularly and cull to slaughter any animal without a good excuse for being thin. Cull all offspring of suspect animals. Avoid fecal build up in birthing areas to limit fecal-oral transmission of *M. paratuberculosis* to offspring. Shear or crutch ewes prelambing. Do not overcrowd. Use elevated feeders to prevent fecal-oral transmission. As *M. paratuberculosis* can survive for a year or more in the environment, overgrazing and spreading of infected manure on pasture must also be avoided to prevent transmission. Do not house thin or sick animals in the lambing/kidding area just for the convenience of easy access or supplemental feeding. Do not introduce the disease with purchased or rescued animals or colostrum or milk from infected dairy herds.

OPP and CAE

The ovine progressive pneumonia (OPP) virus is a retrovirus closely related to but distinct from the caprine arthritis-encephalitis virus (CAE). The viruses gain access to the body orally or through mucosal surfaces (especially respiratory) and are spread throughout the body via infected lymphocytes, monocytes, or macrophages. Virus integrates itself into the genome of the host cells via reverse transcriptase. Infected animals never rid themselves of the virus, despite antibody response. The CAE virus is readily spread in colostrum or milk to nursing kids, but this route of transmission is less important in sheep. On the other hand, horizontal transmission of OPP via respiratory secretions is well documented. Infection usually remains latent for several years or the lifetime of the animal.

Management systems influence the transmission of the virus. Close housing and crowding at the feed trough facilitate transmission of the virus, either by aerosol or by contamination of feed and water with nasal discharges. Transmission via the milking machine and the practice of feeding whey back to dairy animals lead to rapid spread of infection in dairy herds. The reuse of blood-contaminated surgical equipment or needles is an additional risk. *In utero* infection has been documented to occur occasionally. Breeding to an infected ram or buck is a minor means of transmission. The virus is not normally present in semen, but venereal spread has been demonstrated in rams simultaneously infected with *Brucella ovis*, via white cells in semen.

The predominant clinical presentation of OPP is gradual weight loss with reduced exercise tolerance in mature sheep, often three or four years of age or older. Eventually dyspnea becomes obvious, but fever, cough and a purulent nasal discharge are not recognized in an uncomplicated OPP viral infection. An interstitial mastitis ("hard bag") occurs clinically in some infected ewes and subclinically in many more. When recognized, the udder is firm at lambing time with scant but normal appearing secretion. Lambs are hungry because adequate milk is not available. Occasionally OPP causes a clinical arthritis (similar to CAE, including stiffness and swollen carpal joints) or meningoencephalitis.

In goats, CAE frequently causes leukoencephalomyelitis (especially in kids two to six months old) with spinal cord or brain stem signs. Older animals may develop acute or slowly progressive arthritis, often with marked enlargement of the carpal joints. Mineralization of periarticular tissues and bursae occurs in advanced cases. Some goats develop a severe respiratory disease that mimics the pulmonary form of caseous lymphadenitis. An interstitial mastitis with a "hard udder" is another common clinical presentation of CAE. General illthrift without localizing signs is also seen.

There is no treatment for OPP or CAE infection. Analgesics such as phenylbutazone (4.5 mg/lb [10 mg/kg] once a day) may moderate the discomfort in animals with arthritis, but culling to slaughter is always advised once a clinical diagnosis has been made, to avoid further weight loss and transmission of infection to herdmates. Phenylbutazone should not be used in food producing animals.

Most test programs have been based on an agar gel immunodiffusion (AGID) test or enzyme-linked immunosorbent assay (ELISA)[d]. The AGID is easier to perform but the ELISA appears to be more sensitive, and sometimes may detect seroconversion a year or more earlier than the AGID test does. The ideal test would

detect infection, latent or active, rather than just the host's antibody response to that infection. The polymerase chain reaction (PCR) is now available commercially from Colorado State University, but the sensitivity of this test in preclinical infections is unknown.

An initial serologic screening of the entire flock over one year of age, using either AGID or ELISA, should be done. Most control programs are based on culling all seropositive animals and their offspring to slaughter, followed by twice a year retests. In some herds, artificial rearing on cow colostrum or goat colostrum heat treated for one hour at 133°F (56°C) and milk replacer has been successful. Ideally these lambs and kids are removed at birth, before suckling, and reared in isolation from the original flock. A two herd system also decreases the risk of horizontal transmission by respiratory secretions. The negative dairy herd must be milked before the positive animals to avoid transmission by the milking machine.

Soremouth, Orf

Soremouth or orf is a zoonotic skin infection that occurs on sheep and goats around the world. The cause is a parapoxvirus and is distinct from the exotic capripox virus which causes sheep and goat pox. Because the two diseases might be confused clinically, states often require reporting of suspected soremouth. The virus is destroyed rapidly in wet conditions but can persist for years in dry bedding in scabs that fall to the ground during the healing process. Chronic lesions can persist for years on the poll, ears, or external genitalia of rams. These animals and probably other subclinical carriers serve as a further source of infection.

The disease develops after entry of the virus into skin abrasions. Papules evolve into pustules that are followed by scab formation, commonly at the commissures of the lips. Lesions typically last two to four weeks. Rarely cauliflower-like excrescences form or confluent scabs cover the nostrils and interfere with feeding or breathing. Most infected animals show no change in behavior. Some young animals develop severe stomatitis and gingivitis and die, but otherwise lip lesions are of little significance except for the potential to spread the virus to people or to the dam's teat ends during nursing. Teat end lesions are painful and become secondarily infected with *Staphylococcus aureus*. The lamb or kid may starve because of rejection or the dam may develop severe staphylococcal mastitis.

Diagnosis is usually based on the presence of typical papillomatous lesions around the lips or nostrils or on teats. Diagnosis can be confirmed by submitting scab material for electron microscopy or immunofluorescence testing. Serology documents exposure to the virus but is of little diagnostic value in an endemic flock.

Treatment is usually not necessary or helpful and increases the risk of human exposure. Systemic antibiotics are indicated for animals with internal mouth lesions or mastitis from teat lesions. The disease can be prevented by keeping a closed flock or by vaccination. Fomites such as halters and ear tagging pliers can easily spread the infection. It is common for show animals to become infected, presumably because of exposure to contaminated pens or equipment.

Because the presence of soremouth lesions precludes exhibition, many owners vaccinate their animals in advance of the show season. Available vaccines are all live virus products and should be given a minimum of six weeks before the first show. Vaccination is not recommended for closed, uninfected flocks, as it introduces the disease to the premises. Wear gloves when handling the vaccine or the recently vaccinated sheep and goats. The vaccine is brushed into scarified skin in the inside of the ear, underside of the tail, or inside of the thigh. Avoid the thigh in lactating animals because the lesion produced by "take" of the vaccine there can easily spread to the adjacent udder.

Pinkeye, Infectious Keratoconjunctivitis

Mycoplasma conjunctivae is a common cause of pinkeye in sheep and goats in the northeastern United States. It frequently is introduced to a farm by contact with other small ruminants and petting hands at fairs or by the purchase of carrier animals. Conjunctivitis with chemosis, epiphora and photophobia make the ocular involvement obvious in some animals. Keratitis may be absent or severe enough to seriously impair vision. Practitioners in the western United States report that chlamydia cause pinkeye in their region. Diagnosis will require culture (using appropriate transport media as recommended by the receiving diagnostic laboratory) or conjunctival scrapings for staining of the organisms. Severely ulcerated eyes will have secondary bacterial infections.

Mild cases of pinkeye may be left untreated, especially in commercial herds. Subcutaneous injections of long-acting oxytetracycline[e] at 9 mg/lb (20 mg/kg) will provide temporary remission in non-dairy animals, but a meat withdrawal of longer than 28 days must be observed. Oxytetracycline ointment[f] may be used when keratitis is severe and atropine ointment is helpful when the eye seems especially painful (uveitis). Topical application of a local anesthetic followed by a few drops of 5% silver nitrate solution will arrest severe melting lesions and appears to promote rapid and complete healing of the worst ulcers. With or without treatment, immunity eventually develops and signs resolve, but the organism may remain latent and be reactivated by shipping or other stress.

Foot Rot

Foot rot is a chronic contagious infection of the feet of sheep and occasionally goats. It causes debilitating lameness and loss of production. Foot rot is bought and paid for. It is caused by a synergistic infection with *Dichelobacter* (formerly *Bacteroides*) *nodosus* and *Fusobacterium necrophorum*. *Dichelobacter* does not multiply well below 68°F (20°C) and does not survive more than one week on pasture. The disease is introduced with a purchased or borrowed ram or ewe, but does not spread much until conditions become warm and wet. The sole is undermined, beginning at the heels. Foot scald is an interdigital condition that can be a benign infection with an avirulent strain or can be an early stage of virulent foot rot. Maggots can complicate foot rot.

Never bring in an animal from a known infected flock, and treat every other introduced animal as if it were infected. This means foot trimming, dipping feet in 10% formalin or other foot bath, and possibly giving systemic antibiotics. Zinc sulfate, 10% foot bath with a wetting agent as a one hour soak, is the preferred treatment for footrot. Chronically infected sheep should be culled to slaughter.

The commercial foot rot vaccine[g] contains 10 strains but the flock problem may be yet another strain. Two doses of vaccine are given followed by boosters before each period favorable for transmission. The vaccine makes a large abscess and therefore should be given just behind the ear. Vaccination reduces but does not eliminate foot rot incidence. Available cattle vaccine for foot rot[h] is for the *Fusobacterium* component only and probably is high in endotoxin. Goats have reacted very badly to this vaccine and I do not recommend its use.

Conclusions

Infectious diseases cause suffering and serious economic losses in small ruminant herds. They are easily purchased along with new animals but eradicated or controlled on the farm only with great effort and expense. Veterinarians and their clients need to learn as much as possible about disease identification and control programs. Education, along with implementation of appropriate biosecurity measures, will prevent the introduction and spread of these infectious diseases.

Footnotes

[a] Synergistic Hemolysis Inhibition test, University of California, Davis, CA, 530-752-7577

[b] Glanvac®, Pfizer Australia, <www.pfizeranimalhealth.com.au>

[c] Casebac® or Caseous DT®, Colorado Serum Company Co., Denver, CO, 1-800-525-2065

[d] Washington Animal Disease Diagnostic Laboratory, Pullman, WA, 509-335-9696.

[e] Bio-mycin® 200, Boehringer Ingelheim, St. Joseph, MO

[f] Terramycin® ophthalmic ointment, Pfizer Animal Health, Exton, PA

[g] Footvax® 10 Strain, Schering-Plough, Union, NJ

[h] Volar®, Intervet, Millsboro, DE