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Popular Article

Rapidly spread disease of small ruminants: Enterotoxemia

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Sheep and goats of all ages can develop enterotoxemia due to Clostridium perfringens type D. (from one week to several years of age). There are two common times when this disease most often occurs. The first occurs when young animals nurse an abundant milk supply while grazing on improved pastures or being fed high quality hays and/or concentrates (grains). The second corresponds with the finishing or feedlot period where an animal is fed a high grain diet. Enterotoxemia is one of the most common causes of death in feeding lambs. In “explosive” outbreaks, losses may range from 10 to 40%. In unvaccinated feeder lambs, one can expect a minimum of 1% of the lambs to die from this disease, with an average death loss of between 2-2.5%. One of the most serious illnesses, and the most common disease affecting goats in particular regions, is enterotoxemia. The term "Overeating Disease" is also used to refer to it, however overeating is not the cause. The toxin (poison) generated by the bacteria Clostridium perfringens type C or type D is really the cause (aetiology) of the sickness. Sheep provide the majority of the information we have regarding the illness. There are some important differences in purpose and manner of raising (management systems) that exist between sheep and goats that should be kept in mind when one reads and applies sheep information on goats. First, the sickness generally affects a single lamb of a high milk production ewe; the lamb consumes all the milk. Contrarily, the majority of goat births are twins, triplets, or quadruplets, and in the finest caprine management systems, the young are taken away from the mother immediately after delivery. Thus, the type of birth is no factor in goats. Second, dairy children are seldom given huge quantities of high-energy meals for making meat, thus they do not often have the same potential to be exposed to grain (unless by mistake or poor management). Last but not least, goats naturally browse (feed from shrubs and trees while sticking their head out or up); although they do graze, they do not eat as much lush feed (often on a pasture) as quickly as sheep. In conclusion, goats are not specifically affected by the well-known risk factors for the sheep sickness.

Causative Agent

The disease is caused by the bacterium Clostridium perfringens type D. It is normally present in low numbers in the bowel of most animals. Under circumstances brought about by heavy volume and high-quality feeding, the organism grows rapidly and produces a powerful poison (toxin). Death results from this poison within a few hours of absorption through the gut wall. In many cases, fatalities happen so abruptly that owners are unaware their animals are ill. Any situation that causes a decrease in the movement of material out of the intestines will give the organism time to overgrow and produce lethal toxin levels in the animal.

The following list outlines typical causes that might lead to lethal enterotoxaemia in animals:

1. Abrupt feed change
2. Feeding a meal that is excessively rich in energy (excessive carbohydrates, starches, and sugars)
3. Unconventional feeding
4. Too quick an increase in concentration ration
5. Animal with a significant incidence and severity
6. When the digestive track is devitalized by a big volume of uneaten or partially digested food, this circumstance makes it easier for toxins to enter the body.
7. The absence of any immunity, natural or acquired

Clinical Signs

Deaths from enterotoxaemia usually occur suddenly. In some cases, the animal may be sick for several hours or even a day or longer before it dies. Lambs and kids who are affected may exhibit neurological symptoms as shaking, rigid limbs, and convulsive movements. The animal may occasionally fall unconscious and pass away softly. In rare circumstances, diarrhea may be present, especially in goats.

Despite the fact that the symptoms mentioned above are indicative of enterotoxaemia, they can also be present in other conditions that result in abrupt mortality, such as acute acidosis or grain founder, polio encephalomalacia, listeriosis, acute pasteurellosis, tetanus, and blackleg. It is crucial to identify the cause of these fatalities as soon as possible and accurately.

Diagnosis/Treatment

A diagnosis of enterotoxaemia is suggested when the sudden death of concentrate-fed animals that have not been vaccinated for Clostridium perfringens type D has occurred. Necropsy findings can also help lead to a diagnosis. If a necropsy or post-mortem is done immediately or shortly after death, few changes may be observed, particularly if the animal died suddenly. When changes are observed, they usually include congestion and fluid in the lungs, and an increase in fluid in the heart sac (pericardial sac) with clots of gelatinous material (fibrin). Small hemorrhages and blood splashes will be seen under the clear membrane which lines the outer and inner muscle walls of the heart. Fluid can accumulate in the renal tissues, which then quickly degrade. In reference to this, the phrase "pulpy kidney" is used. The carcass quickly decays and

develops a gas-filled enlargement. When examined, the urine of an animal that succumbed to enterotoxemia typically tests very positive for high amounts of glucose (sugar). This disease can be confirmed by laboratory tests performed on the dead animal. It is wise to seek the counsel of a veterinarian to help in establishing a correct diagnosis and to outline control measures.

Sulfas, an oral antibiotic, may be helpful for certain animals who are already ill. Preventive measures should take precedence over therapy since diagnosing ill animals can be challenging and treatment is sometimes unsatisfying.

Prevention

- To prevent this disease, efforts should be focused on management and vaccination:
- Make the adjustment from range or pasture to feedlot conditions gradual. Place lambs/kids on either alfalfa hay or prairie hay first, and then gradually accustom them to concentrates.
- Check animals for parasites and de-worm prior to vaccination. A burden of parasites interferes with the production of protective antibodies from the vaccines.
- Have plenty of feed for the lambs/kids at all times. This prevents them from overeating at one given time.
- All lambs/kids from mothers that have been vaccinated previously, should be vaccinated at 6-8 weeks of age with a Clostridium perfringenstype D toxoid vaccine; repeat this vaccine in 3-4 weeks. Lambs/kids from mothers that have not been vaccinated, should receive their first vaccine at 1-3 weeks of age; repeat this vaccine twice, every 3-4 weeks.
- All adult animals should have an annual booster with a Clostridium perfringens type D toxoid vaccine 4-6 weeks prior to the lambing/kidding season. In pregnant animals, this will help boost antibodies shed in the colostrum.
- All adult goats should be vaccinated a minimum of twicea year. They require more frequent vaccination because they do not respond as well as sheep do to the vaccine.
- Vaccinate lambs soon after their arrival in the feedlot. Allow at least 10-14 days after vaccination for immunity to develop. Under certain conditions, re-vaccination (booster dose) is required 3-4 weeks later.