

Neurological Diseases of Sheep and Goats

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Abstract

This paper discusses the most common neurological diseases in sheep and goats, including listeriosis and poliоencephalomalacia. Because diagnostic techniques may be limited, a thorough history and complete physical examination are imperative. Tips for taking a history, performing a physical exam and some diagnostic techniques are presented. Treatment options are also presented where applicable.

Résumé

Cette communication traite des maladies neurologiques les plus répandues chez les moutons et les chèvres, incluant la listériose et la poliо-encéphalomalacie. Puisque les techniques de diagnostic sont parfois limitées, il est important d'obtenir une bonne anamnèse et un examen physique adéquat. On présente des conseils pour la prise d'anamnèse et l'exécution d'un examen physique, ainsi que quelques techniques de diagnostic. On y présente aussi différents traitements possibles, le cas échéant.

Introduction

Diagnosis of neurological diseases can be one of the most frustrating aspects of small ruminant practice. Many diseases can present with similar signs, and diagnostics are limited. But because some neurological diseases have herd implications, or are zoonotic, the practitioner must try to find a list of most likely diagnoses, so that treatment and/or preventative measures can be taken.

History

Because of limitations of the physical examination and diagnostics, a thorough history becomes very important. Many times a management or feeding change has taken place that leads to neurological disease. Remember that many neurological signs are not caused by diseases of the nervous system, but are secondary to other systemic diseases, so all aspects of the history are important.

Physical Examination

A thorough physical examination should be done when possible. Injury to the animal and personnel must always be considered when trying to restrain goats with neurological disease for examination. Since many neurological signs are caused by other systemic diseases (grain overload, severe endotoxemia, severe azotemia, hepatoencephalopathy), a complete general physical examination should be performed before concentrating on the neurological examination. Trying to observe the animal in its surroundings before restraint is important. Mentation, head posture, gait abnormalities, tremors, head tilt, etc. should be noted. Blindness can be very difficult to prove, since animals can be very adept at avoiding obstacles using other senses. The main objective is to classify the disease as a brain disease or a spinal cord disease. Although multifocal diseases can occur, they are less common. Gait abnormalities, especially ataxia, can be present with both brain and spinal cord disease. Also, generalized weakness from other diseases can be very difficult to differentiate from true neurological ataxia. If changes in mentation, headpressing, blindness, bizarre behavior, seizures, etc. are seen, cerebral disease should be suspected. Cerebellar diseases usually cause hypermetria and intention tremors. Circling, ataxia, proprioceptive deficits and/or cranial nerve signs are seen with brainstem disease. Blindfolding may worsen cerebellar or brainstem signs.

Once the animal is observed at a distance, the practitioner usually already has an idea whether the disease involves the brain or spinal cord. Cranial nerve examination needs to be performed to help further differentiate brain disease, or if no brain signs were seen from distant observations, to rule out brain disease so spinal cord disease can be focused on. Localization of spinal cord disease is the same as in other species.

Diagnostics

The size of goats makes them candidates for diagnostic procedures commonly used in small animals, such as plain and contrast radiography, computed tomography and magnetic resonance imagery. However, the cost of some of these procedures may be prohibitive.

Cerebrospinal fluid (CSF) is easily collected from the lumbosacral space and can provide valuable information. A 20-gauge, 1-1/2 inch needle is used for neonates, an 18-gauge, 1-1/2 inch needle for adult goats. Ambulatory patients can be tapped standing. Non-ambulatory patients should be placed in lateral recumbency or in sternal recumbency in a "dog-sitting" position with the rear legs forward on either side of the animal. The pelvis needs to be straight and level. Light sedation and a local anesthetic will help with restraint. The lumbosacral area should be clipped and surgically prepared. Wearing sterile gloves, the indentation of the lumbosacral junction should be palpated. A needle is inserted into the deepest part of the indentation, directly on midline. Keep the needle perpendicular to the spine from the side view, and straight up and down from the rear view. If bone is encountered, redirect the needle slightly cranial or caudal until the needle drops into the lumbosacral space. Advance the needle slowly until a slight "pop" is felt. The animal usually jumps slightly when the needle punctures the dura mater. CSF should flow from the needle or can be gently aspirated with a syringe. If the needle is in the lumbosacral space and advanced until bone is encountered again, back the needle out 1-2mm and try to aspirate. Place the CSF in an EDTA tube for fluid analysis and a plain tube if cultures are desired. Normal, non-traumatically obtained fluid should be perfectly clear with no discoloration, sediment or turbidity. It is best to have the sample analyzed locally as soon as possible (within one hour). If this is not possible, place half of the sample in an equal volume of 40% ethanol to preserve the cells (inform the laboratory that this has been done), or centrifuge half the sample to concentrate the cells and prepare slides to be sent with the rest of the fluid.

Differential Diagnosis

The most common diseases involving the brain that I see in adult goats are polioencephalomalacia (PEM) and listeriosis. PEM can be seen in any age animal, and is associated with disruption of normal diet or eating habits. High-concentrate diets, sudden changes in diet, and high molasses or sulfur content in the diet have all been associated with PEM. Any stressful situation (weaning, change in housing, introduction of new animals, bad weather) that causes a decrease in appetite can cause PEM. Signs associated with PEM are central blindness, depression, incoordination, head pressing, recumbency, opisthotonus, seizures, vocalization, and/or dorsomedial strabismus. PEM and lead toxicity can both cause central blindness, and lead toxicity cases can transiently respond to thiamine treatment, so lead toxicity should be a differential diagnosis for PEM. Definitive diagnosis of PEM requires measurement of red blood

cell enzymes not readily available in practice, so a diagnosis of PEM is usually made by response to treatment with thiamine. An attempt should be made to determine and correct the underlying cause. PEM can be treated with thiamine (5 mg/lb SQ, TID to QID). If caught early the prognosis is good. If no response to treatment is seen in one-to-two weeks, chances are the animal will have permanent deficits. Supportive care is important if animals cannot eat or drink. Transfaunation of rumen contents from a normal ruminant may help if gastrointestinal disease is severe. Control of seizures can usually be accomplished with valium, but occasionally phenobarbital is needed.

Listeriosis affects goats more often than cattle and sheep, and is most commonly seen in winter and early spring. Any damage to the oral mucosa (erupting teeth, introduction of hard feeds or browse) can predispose to listeriosis. It occurs in animals grazing close to the ground and eating wet, moldy hay. It is not commonly associated with silage feeding. Listeriosis in goats is characterized by depression and cranial nerve deficits. Circling is frequently seen, but listeriosis should be considered with any cranial nerve deficit, especially if multiple, asymmetric deficits exist, even if circling is not present. Disease progression can be quick, with many animals found recumbent. Migration of *Paralaphostrogylus tenuis* is occasionally seen in goats and can present like listeriosis, but confirmation of this disease can be difficult. The CSF tap shows elevations in protein and mononuclear cells, which can also be seen with other diseases. CSF may also be normal in a small percentage of cases, especially if taken from the lumbosacral space. *P. tenuis* migration sometimes causes increased numbers of eosinophils in CSF. Listeria can be difficult to culture, and a negative CSF culture does not rule it out. If the clinical signs are consistent with listeriosis, antimicrobial therapy is indicated. Tetracyclines and penicillins are effective if instituted early. Some cases seem to respond to one but not the other of these antibiotics, and predicting which will work in a certain case is difficult. If no response is seen to either tetracycline or penicillin in 48 hours, therapy should be changed to the other drug. Since listeria can occur in multiple animals on a farm, historical response to therapy is important to note. Anti-inflammatory therapy (non-steroidal or steroidal) and supportive care are also very important. These animals may be unable to eat or drink, and can become acidotic due to excessive saliva loss.

Goats are very susceptible to tetanus. This disease should be suspected in nonvaccinated animals showing signs of lameness or stiffness. Signs progress to a stilted gait, raised tailhead, sawhorse stance and lockjaw. Retraction of the lips and hyperesthesia to sound and light are also seen. The disease eventually

progresses to recumbency and death. Treatment, if successful, is usually prolonged for months and requires extensive supportive care. However, the small size of goats makes this easier than in other large livestock. Tetanus antitoxin (150-300 IU) and high doses of penicillin are needed to stop progression of the disease. Fluid and nutritional support are imperative. If lockjaw is present, oral fluids and feeding may be impossible. Fluids can be administered intravenously, and fluids and food can be administered through a rumenostomy. Acid-base status should be monitored, and baking soda can be added to fluids and food if necessary. A quiet, dark environment is also important; sedation and muscle relaxants can be administered. Many animals will be unable to rise, but will be able to stand and walk if lifted. Slings can also be made to help support weight and prevent decubital ulcers. Tetanus vaccines are highly efficacious and cheap. I recommend a toxoid in the first week of life, at one month of age, at weaning, and yearly after that. Older animals that have not been vaccinated should have an initial dose and a booster according to the product label.

No discussion of neurologic diseases in small ruminants would be complete without mention of scrapie and rabies. Scrapie is a transmissible spongiform encephalopathy, the cause of which is debatable. It's more common in sheep, but does occur in goats and is transmitted both horizontally and vertically. Host genetics and stain of the infectious agent determine whether an animal will develop the disease. Genetics of susceptibility are well defined in sheep, but not in goats. Clinical signs are intense pruritis, ataxia and wasting, and the only antemortem test is immunohistochemistry of lymphoid tissue from the nictitating membrane. There is no treatment for scrapie.

Rabies in goats can present with a variety of clinical signs. Sometimes the dumb form occurs, where ani-

mals are depressed and progress to recumbency. Other times they are more aggressive, attacking people and other animals and sometimes becoming sexually excited. Any type of paralysis should be suspected to be rabies. Collection of CSF should be avoided if rabies is suspected, or handled with precaution to prevent human exposure. Increased protein, mononuclear cells and neutrophils are seen. Rabies can be prevented through vaccination with products approved for sheep. Vaccination can start at three to four months of age with appropriate booster, then yearly thereafter.

Other diseases such as bacterial meningitis, brain abscesses, otitis, toxicosis and injuries that occur in other livestock species can occur in goats. The clinical signs, diagnosis and treatment are also similar to other livestock species. Organisms most often causing meningitis are *Escherichia coli*, *Pasteurella* spp, and *Mycoplasma* spp, and are also implicated in otitis media and interna cases. Brain abscesses are most often caused by *Actinomyces* spp. Common toxicities include rhododendron (azalea), organophosphate and lead toxicity. Salt toxicity/water deprivation can also occur.

Although much less common than bacterial meningitis/otitis, goat kids may also have neurologic disease due to enzootic ataxia and spinal abscesses, and the neurologic form of caprine arthritis encephalitis virus (CAE). Enzootic ataxia, also called swayback, is caused by primary or secondary copper-deficient diet in does. Signs of weakness and ataxia are seen within a few weeks of birth. Treatment is usually unsuccessful, as damage to neurologic tissues is typically irreversible. Spinal abscesses usually present as acute spinal paresis/paralysis when the vertebral body fractures through the infection site. CAE most commonly presents as arthritis in adult goats, but can cause ataxia/paresis/paralysis in goat kids from one to four months of age.