

Large Animal VETERINARY Rounds

AS PRESENTED IN THE ROUNDS OF THE DEPARTMENT OF LARGE ANIMAL CLINICAL SCIENCES
OF THE WESTERN COLLEGE OF VETERINARY MEDICINE, UNIVERSITY OF SASKATCHEWAN

Diseases of Farmed Ungulates

Part 1. Necrobacillosis in Deer

by Murray Woodbury, DVM

Necrobacillosis is also known as necrotic stomatitis, hepatic necrobacillosis, foot rot, calf diphtheria, or lumpy jaw. It produces a variety of diseases, with abscessation occurring in almost any body organ or joint cavity. In white-tailed deer, it is usually seen as individual cases of lumpy jaw or as herd outbreaks of a fatal, multisystemic disease in fawns.

Cause

Necrobacillosis is caused by *Fusobacterium necrophorum*, an anaerobic, Gram-negative, often highly filamentous rod-like bacteria. This organism is found in the intestines of many species as part of the normal flora. It survives well in wet soil that has a high manure content. Other bacteria are often associated with the disease, *Arcanobacterium pyogenes* being the most common.

In cattle, sheep, and goats there are variations in virulence, biotype, and antibiotic sensitivity of both *F. necrophorum* and *A. pyogenes*, making prevention through vaccination and successful treatment with antibiotics difficult to accomplish. Studies at the Western College of Veterinary Medicine show that this is true for deer as well. Initial results show that *F. necrophorum* subspecies *necrophorum* is the anaerobic subspecies most often isolated (28%), followed by subspecies *varium* (19%). It also appears that strains isolated from deer cases are notably different from those of bovine origin.

Geographic distribution

F. necrophorum occurs throughout the world. Outbreaks of necrobacillosis in wild populations have been reported in mule deer in California, elk in Wyoming, white-tailed deer and pronghorn antelope in Saskatchewan, and reindeer in Siberia.^{1,6,9} New Zealand and North American deer farms suffer significant losses from the foot rot and necrotic stomatitis forms (tongue and throat abscesses) of the disease.^{3,7,10}

Transmission

Animals develop necrobacillosis when a number of factors are present. Stress such as heat, cold, overcrowding, or poor nutrition predispose to infection. *Fusobacterium* cannot penetrate intact skin. The organism gains entry into the body through cuts or abrasions to the skin or mucous membranes. In the mouth, grass awns, coarse feed, metal objects, and unevenly worn or newly erupting teeth can penetrate or damage the oral mucosa and create points of entry. Contamination of the abrasions with soil containing *F. necrophorum* results in infection. Changes in the rumen environment such as grain overload or sudden changes in feed can damage the lining. This provides a portal of entry for *Fusobacterium*, where they may then pass to the liver, causing hepatic necrobacillosis, or to other major organs.

In some situations, the disease appears to be contagious and there is, no doubt, direct contamination of shared feed bunks and drinking water by infected animals with open lesions. The infectivity or invasiveness of *F. necrophorum* is enhanced by the presence of other bacteria such as *E. coli* and *A. pyogenes* and the presence of these bacteria permit infection by relatively few *F. necrophorum* organisms.⁸



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Species affected

Cases of necrobacillosis have been reported in many species of wild and domestic hoofed animals. In farmed white-tailed and fallow deer, necrobacillosis is recognized as a serious problem of young or newly weaned fawns. There have been large die-offs of wild mule deer and white-tailed deer, reindeer and elk.⁷ Heavy losses of kangaroos and wallabies have occurred in zoos and in the wild.⁴ Foot rot can be a significant problem in cattle, sheep, and bison, and also wild hoofed stock.⁶

Clinical signs

The location of lesions in the body determine clinical signs, but systemic infection is common, especially in fawns where it results in rapid death. Other clinical signs include:

- Affected animals are usually depressed and febrile. Often the hair coat is rough and the animal is thin and doing poorly.
- Lesions involving the mouth are often deep, invading the surrounding soft tissue (Figure 1) and bone creating the classic swollen jaw or face.
- Lesions in the oropharynx of fawns are common. These are often unseen by producers. The lack of nutritional reserve and the toxic by-products of the infection lead to rapid death.
- In the throat, necrotic laryngitis will show itself as loud wheezing. Some dead tissue and bacteria may be sucked into the lungs causing abscess formation and pneumonia.
- Lameness is seen when the feet and associated joints are involved. Swelling between the toes is the first visible sign, followed by localized tissue death. The infection spreads to the joints and bones in more advanced cases.^{5,7,10}
- Cases of necrotic rumenitis frequently have few signs except weight loss in chronic forms or death.

FIGURE 1: Severe tongue lesions are common.



Courtesy of Dr G. Wobeser

Postmortem findings

The most common lesions are necrotic ulcers or abscesses of varying depth in oral, pharyngeal, or laryngeal mucous membranes. In severe cases, lesions extend into the nasal cavity, upper and lower jaw bones (Figure 2), larynx, trachea and lungs. Bacteremia may cause abscesses in internal organs. The abscesses contain foul smelling, thick purulent material having a greenish tinge. Rumen lesions are characterized by well-defined, yellow, raised foci of necrosis. These extend deep into the rumen wall and can perforate, leading to peritonitis.¹⁰

Diagnosis

Clinical signs and history are usually sufficient to establish a diagnosis. The organism is difficult to grow, making culture and isolation difficult without special anaerobic culture swabs and growth medium. Identification of the organism in lesions can be made with a fluorescent antibody test.⁷ There is also a blood test (ELISA) for the detection of serum antibodies to *F. necrophorum* in sheep and cattle.² However, these tests may not be available in all pathology laboratories.

Treatment

Early detection of necrobacillosis is important if the disease is to be treated successfully. Delayed or half-hearted treatment frequently gives only temporary results. Chronic osteomyelitis of the jaw and recurrent draining abscesses are common. Affected animals should be isolated from healthy animals because they shed infective organisms. Feeding and drinking areas and working facilities should be cleaned and disinfected to prevent spread of the organism. Although antibiotics are not currently licenced for use in deer, they are indicated for treatment of necrobacillosis.

Selection of an antibiotic should be based ideally on culture and sensitivity, but resistance appears to be less of a problem than inadequate drug penetration and low antibiotic concentrations in infected tissues. Variation in reported success rates for treating deer probably results more from the difficulties of drug delivery and direct wound treatment than from bacterial antibiotic resistance.

Procaine penicillin is effective, but it has a short half-life, requires frequent administration, and does not penetrate abscesses well, making it an impractical choice. Sulfatrimethoprim combinations or long-acting tetracyclines are better choices and have been used with good success. Florphenicol works well in abscesses, is effective at low tissue concentrations, and can be given at less frequent intervals. There is anecdotal evidence that florphenicol causes diarrhea in fawns.

Oral administration of tetracycline for therapeutic or prophylactic use is frequently attempted because deer can be difficult to handle and restrain. Gastrointestinal absorption can be erratic and food may decrease absorption, but sometimes there are no alternatives. The type of tetracycline used orally is important. Bioavailability is lowest for

FIGURE 2: Lower jaw with abscess formation involving bone and teeth.



Courtesy of Dr G. Wobeser

chlortetracycline, intermediate for tetracycline or oxytetracycline, and highest for doxycycline.

All antibiotic use in deer is considered off-label and there are no published data on effective dose rates. Cattle and sheep dose rates are commonly used in deer with minimal side effects.

Where possible, abscesses should be drained and flushed to remove debris and toxins. Debris and pus from the lesion should not be allowed to contaminate the treatment area. Collect and incinerate the material in a plastic bag. Amputation of a claw may be required in severe cases of foot rot where there is bone and joint involvement.

Prevention

As with most diseases, prevention is much more rewarding than treatment. Good management practices form a large part of any control or prevention program. These include:

- Stress from overcrowding, social instability, inappropriate mixing of sexes and ages, or poor animal handling technique should be minimized.
- Good general hygiene should be practised with special attention to the control of excessive fecal contamination of paddocks, holding and handling facilities, and feeding equipment. Hay should not be offered on the ground to prevent contamination with urine and feces.
- Do not use preventative antibiotics without a diagnosis or good evidence of infection. In the long-term, more problems will be created than will be prevented.
- Food structure (coarseness) is probably not as important as the nutritional quality of the food. Poor quality food decreases resistance to disease in general. Mucosal skin integrity depends on adequate levels of vitamin A and possibly C.⁴ Deficiency of these vitamins may predispose animals to necrobacillosis infection by making invasion easier.
- The type of soil underfoot may play a role in the persistence and transmission of *F. necrophorum*. Clay and other water-retaining types of soil are thought to support

bacterial life better than well-drained sandy soils.⁴ Whatever the soil type, avoid poorly drained paddocks and moist areas.

Vaccination

Vaccination will never replace good management practices. However, when faced with persistent disease, vaccination may be an option. There is no commercial vaccine licensed for prevention of necrobacillosis in deer, but variable results have been observed with sheep and cattle *Fusobacterium necrophorum* vaccines. These are pure *F. necrophorum* bacterins designed to prevent foot rot in cattle and sheep. Some deer producers claim to have achieved good results and others have been disappointed. Their usefulness in deer species has never been scientifically established.

Because necrobacillosis is most often a mixed bacterial infection (*F. necrophorum* plus others), every farm outbreak may involve a similar, but unique combination of bacteria. The use of “autogenous” vaccines may therefore hold more promise. Autogenous bacterins are made up separately for each infected farm, with bacteria derived from infections on that particular property.

Animals are inoculated with a product made from bacteria obtained from lesions on that farm, creating a more specific and effective immunity to the necrobacillosis organisms. Autogenous vaccines have been used to treat necrobacillosis in sick animals, as well as to prevent disease in healthy ones. Both uses have not been adequately tested in white-tailed deer and further investigation is needed to determine their usefulness.

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Part 2. Chronic Wasting Disease Update

by Murray Woodbury DVM, MSc. and Shelagh Copeland, DVM, MVSc.

Chronic wasting disease (CWD) of elk (wapiti) and deer continues its high public profile in North American animal agriculture. The emergence of “mad cow disease” or BSE (bovine spongiform encephalopathy) has increased public awareness of CWD and similar neurologic diseases in other animals. Both are in the transmissible spongiform encephalopathy group of neurologic diseases.^{1,3} The cause of CWD remains unknown, but the generally accepted theory is infection by abnormal prions (proteinaceous infectious particles or agents consisting largely or solely of protein) which accumulate in the brain until they cause neurologic symptoms.² While there is no demonstrated risk to human health, there is concern that CWD may cross species barriers to infect humans who have eaten infected animals.

Geographic distribution

CWD is endemic in free-ranging Rocky Mountain elk, mule-deer, white-tailed deer⁴ and black-tailed deer⁶ in an area involving northern Colorado, southern Wyoming, and southwestern Nebraska.⁵ These states plus South Dakota, Oklahoma, Montana, and the province of Saskatchewan have seen cases of CWD in captive cervids. So far, all cases of CWD in Canadian farmed elk can be traced back to animals imported from the United States.⁷ Except for a single case in a Saskatchewan mule deer, CWD has not been reported in Canadian wild deer or elk.

Canadian CWD

As a result of the Canadian Food Inspection Agency's (CFIA) investigation and trace-out efforts, 38 positive elk have been found in Saskatchewan.^{7,8} To date, approximately 3470 animals have been destroyed. So far, all positive CWD animals have originated from one Canadian “source” herd that originally contained imported infected animals from the United States. Despite the large number of animals involved in trace-out slaughter activities, the number of farms with positive animals remains very low. Eradication activities will continue on Saskatchewan elk farms until no further cases of CWD exist. Increased wildlife surveillance activities in Saskatchewan and neighbouring Alberta are directed at defining the problem in wild deer. Prevalence in Canadian wildlife is presently unknown.

Transmission

The mode of CWD transmission is not known with certainty. In an outbreak in captive Rocky Mountain elk, transmission appeared to be from animal

to animal within a herd, with no preference for spread by mother to offspring.⁴ This is different from the pattern of scrapie transmission in sheep where outbreaks appear to be related to lambing and move from mother to offspring; spread to others may also occur from environmental contamination by the placenta and birthing fluids.³

It is thought that the CWD agent is passed in saliva, feces and/or urine and is transmitted via an oral route. Once ingested, the disease has a natural incubation period of 1.5 to 3 years before the onset of clinical signs.⁴

A genetic resistance to CWD infection may exist in some elk.⁹ The PrP gene is responsible for creating (encoding) the prion protein that gets changed when an animal is infected with a transmissible spongiform encephalopathy. This gene exists in more than one form (polymorphism); each form having differences in their amino acid sequences. These polymorphisms are associated with relative susceptibility to the transmissible spongiform encephalopathy of humans, mice, and sheep. Some forms of the gene make animals more susceptible to transmissible spongiform encephalopathy. Conversely, some forms of this gene may actually prevent an elk from creating harmful prion proteins that cause CWD. Researchers have found that wild elk possessing a PrP form known as “LL” (Lysine/Lysine) have never been known to have CWD.⁹ This finding, if proven to be accurate, presents the possibility of breeding animals that are more resistant to CWD.

It is not known whether contact with a prion-contaminated environment plays a role in perpetuating outbreaks of CWD, but current research suggests it may. Initial re-stocking of a research facility in Colorado, approximately 1 year after depopulation to control CWD, was followed by CWD infection in the re-stocked animals.⁴ As the status of the re-stocked animals was unknown, it was not possible to know if the new animals became infected from living in a contaminated environment, or were infected before arrival. In a more recent experiment, animals from a certified-free CWD herd were put into a contaminated paddock. Deaths from CWD occurred approximately 18 months later.¹⁰ Investigation into the role of environmental exposure is ongoing.

Other species affected

Bovine spongiform encephalopathy may have arisen from modification of the scrapie agent. It has been implicated as a cause of dementia in humans known as Creutzfeldt-Jakob Disease (CJD) The possibility that CWD could behave similarly is being investigated. No evidence for natural transmission of CWD to other species has been found so far. Studies include the following:

- 10 cattle given a single oral dose of CWD-infected brain tissue, at a level that would easily cause CWD in cervids, have not had any signs of CWD 4 years later;¹⁰ study is ongoing.
- 20 cattle put in with CWD infected deer and elk have no signs of CWD 4 years later;¹¹ study is ongoing.

- A 10-year investigation of 22 cattle herds that graze in areas with a 13% incidence of CWD in wild cervids, has found no CWD in over 260 cattle tested (animals in herds for an average of 7 years).¹¹

- Inoculation of CWD-infected material directly into the brains of 13 cattle caused infection in 3 animals, but method is not a natural route of exposure;¹⁰ study is ongoing.

- In laboratory “test tube” experiments, CWD converted prion protein from humans and cattle only at a very slow rate suggesting an interspecies barrier.¹¹

- The case rate of CJD among humans in Wyoming and Colorado, where CWD has been endemic in wild cervids for over 20 years, is less than the national average of 1/1,000,000.¹¹

- The three cases of CJD found in young hunters occurred outside the CWD endemic areas and they did not hunt in endemic areas. Genetic and diagnostic testing of the CJD strains in these people also found no evidence for transmission from CWD.¹¹

Experiments on the transmissibility of CWD to other species (ferrets, mink, goats, squirrel monkeys, cattle, sheep) are ongoing, and epidemiological investigations continue as well. Until the lack of transfer can be confirmed, it is recommended that offal, brain, and spinal cord, as well as, all meat from clinically affected deer should be avoided for use as food, or as a protein source in animal food.¹²

Clinical signs

Clinical signs of CWD include: emaciation, excessive salivation, behavioural changes (eg, loss of fear of humans), ataxia, drooping of head and ears, weakness, protruding eyes, increased thirst and urination (especially in deer), pneumonia and trauma-induced lesions.⁵ Aspiration pneumonia resulting from a reduced ability to swallow is considered an important clue to the presence of CWD, and elk or deer dying with this problem should be suspect. Infected animals may be more prone to trauma (eg, broken necks, broken legs) due to behavioural changes. Clinical signs may last for weeks to months before the animal dies, with most elk succumbing to the disease in less than 12 months.⁴ One study concluded that the duration of clinical disease in deer ranged from 4-32 weeks, while clinical signs in elk ranged from 4-24 weeks.⁴ In contrast to animals with scrapie, postural, proprioceptive, and motor abnormalities are rarely observed in cervids, despite the presence of cerebellar damage.¹³

Diagnosis

Currently there is no definitive way to diagnose CWD before death. The tentative diagnosis is based on clinical signs. A confirmed diagnosis is made by microscopic examination of brain tissue, immunohistochemistry (IHC)¹⁴ to look for protease-resistant prion protein (PrPres), and/or Western immunoblot. Presently there is no antemortem test capable of detecting prion in blood or other body fluids.

Differential diagnosis

Whenever nervous signs and excessive salivation are seen, rabies must be suspected; however, clinical signs of CWD are less rapid in onset than those of rabies. Other viral diseases, such as pseudo-rabies are possible, but have yet to be reported in wapiti. Bacterial diseases that affect the central nervous system, such as listeriosis, should also be considered. Meningeal worm infection may also cause loss of fear of handlers and loss of condition. Johne's disease causes weight loss, debilitation, and eventually death in farmed cervids, but is also frequently accompanied by progressive diarrhea that is not a clinical sign of CWD.

Treatment and control

There is currently no treatment for CWD. CWD is now a federally reportable disease (required by law to be reported to the CFIA) and all cases are dealt with by the Canadian Food Inspection Agency. Their present program is based on the following principles established from current scientific knowledge.⁸

- An animal exposed to infection will develop the disease within 36 months.

- Clinically diseased animals could be infectious to other animals up to 18 months prior to death.

- CWD contaminates the environment with infective prions in amounts varying from heavy to minimal.

- Decontamination is assumed to be possible and procedures are carried out as follows:

- Feed and manure is scraped off until undisturbed soil is reached, or deeper if a clinical animal spent considerable time at one site. Removed material is buried.

- New material is added to form a barrier and kept topped up.

- All facilities and equipment exposed to clinical animals are cleaned of organic material and disinfected by soaking for one hour in sodium hydroxide or sodium hypochlorite.

- Wild cervids are not permitted on any infected premises; if found they are destroyed and tested for CWD

When a positive CWD animal is found these actions are taken:⁸

- Incineration and/or deep burial of the carcass of the affected animal in an approved site

- Quarantine and inventory of all animals on the farm

- Depopulation of all cervids in the herd that had direct contact with the positive animal, and proper disposal in an approved site

- Clean-up and disinfection of contaminated areas

- Evaluation and compensation to owner

- Removing and testing trace-out animals that have left the herd in the last 3 years, depending on results of testing, further depopulation may occur in the trace-out herds

- Monitoring of trace-out animals that left the herd in the last 36-60 months

For further information on the CFIA program, please contact the local CFIA district office or their web site at <http://inspection.gc.ca/english/animal/heasan/disemala/cwdmdce.shtml>

Because the farmed cervid industry has been highly regulated from its beginning, trace-outs and monitoring are relatively easy. CFIA routinely monitors for CWD by examining animals for signs of neurologic disease at the time of tuberculosis testing (once every three years). All deer and wapiti over 18 months of age received at provincial laboratories for post-mortem examination are also routinely screened for CWD. In 1990, it became policy to issue movement permits to deer and elk so as to monitor their movement. Also, from 1990 until the summer of 1999, there was a ban on the importation of deer and wapiti from the United States.

Saskatchewan Agriculture and Food (SAF) and the farmed cervid industry have recently created a voluntary surveillance program for CWD. The Saskatchewan Cervid Health Surveillance Program is based on an accurate herd inventory, individual identification of herd members, and reporting of all deaths in animals over 12 months of age to SAF. Reporting is followed by submission of the head to a laboratory and examination for CWD by immunohistochemistry.

Saskatchewan Environment and Resource Management (SERM) has also asked hunters, in certain areas of the province, to submit harvested cervid heads for CWD testing. Over 1300 heads have been submitted from last year's hunt with one positive mule deer found to date.

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Abstract of Interest

Chronic Wasting Disease in Mule Deer: Disease Dynamics and Control

GROSS, J.E., MILLER, M.W.

A mechanistic model was developed to simulate dynamics of chronic wasting disease (CWD) in mule deer (*Odocoileus hemionus*) populations. The model projected age-specific disease dynamics, changes in population size, and effects of control strategies. Parameters were estimated from observations of infected and uninfected mule deer in Colorado. Monte Carlo techniques were used to evaluate likely responses. Simulations of CWD epidemics were highly unstable. Disease was not sustained in projected populations when transmission rates were low, but CWD eliminated populations when more realistic transmission rates were used. Stable coexistence of CWD in simulated mule deer populations was not achieved. Even low CWD prevalence reduced potential harvest via combined effects of diminished per capita production and decreased population density. Changes in CWD prevalence within population were highly sensitive to transmission rate, and small decreases resulted in noticeable damping of prevalence increases. Simulated selective culling programs revealed the importance of initiating control while CWD prevalence was low (<0.01). Low selective culling rates (<20% of infected populations) effectively eliminated CWD if initiated when prevalence was low, but the likelihood of control diminished rapidly as prevalence increased. Management programs will likely require an effort sustained over many decades if eliminating CWD is the desired goal.

J Wildl Manage 2001; 65(2):205-215.

Popular Websites

www.usask.ca/wcvm/herdmed/specialstock

The Specialized Livestock Research and Development Program

Western College of Veterinary Medicine
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