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Malignant catarrhal fever in bison

Murray R. Woodbury DVM, MSc

Malignant catarrhal fever (MCF) is an infectious lymphoproliferative disease of cattle, bison, deer, and other ungulates. It has a global distribution and outbreaks occur sporadically in many geographical areas.^{1,2} As early as the late 1700s, the occurrence of MCF in European domestic cattle in contact with domestic sheep was reported. In Africa, MCF was known as "wildebeest disease" because it occurred when cattle were grazed on the calving areas of wildebeest.³ Domestic sheep and wildebeest are now known to be inapparent carriers of viruses that produce clinical MCF in other species. Relatively little is known about MCF in bison. However, most of the world's bison, and therefore bison-related cases of MCF, are found in North America. It is suspected that MCF in bison is linked to contact with domestic sheep.

MCF poses an economic threat to the bison producer. A variety of diseases, including MCF, commonly affect bison (Table 1). Although the occurrence of MCF is not widespread in the industry, individual producers suffer high losses and MCF is a major cause of mortality (Table 2). Outbreaks of clinical MCF are uncommon and sporadic. The morbidity rate in infected cattle herds is generally 3% to 53%, but can go as high as 100%.² The mortality rate, however, is almost 100%.

Causes

MCF is caused by viruses in the herpesvirus family. In Africa, a gammaherpesvirus called alcelaphine herpesvirus-1 (AHV-1) is responsible for most cases of MCF. In 1960, the virus was isolated and identified from the principal host and carrier, the wildebeest.⁴ MCF caused by AHV-1 is known as wildebeest-associated MCF (WA-MCF).

In North American livestock, including bison, the disease is more commonly associated with a gammaherpesvirus named ovine herpes virus-2 (OvHV-2).^{1,2} This virus can be found in most sheep and possibly goats, but does not cause clinical disease in these hosts.⁵ Therefore, MCF caused by OvHV-2 is known as sheep-associated MCF (SA-MCF). OvHV-2 has never actually been isolated, but evidence of its existence is found in the consistent detection of antibodies to AHV-1 in sheep. Also, the culture of lymphoblastic cell lines from cattle and deer with SA-MCF has produced cells containing DNA that hybridize with clones of a unique region of the AHV-1 genome.⁶ OvHV-2



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Table 1: The most common diseases of bison as reported by producers (from 477 survey respondents)²⁵

Pink eye	151
Pneumonia	53
Lice	45
Calf scours	41
Capture myopathy	31
Foot rot	25
Lungworm	22
White muscle	21
Grain overload	17
BVD	16
Blackleg	11
Liver fluke	12
MCF	8
Bloat	8
Redwater (<i>Clostridium hemolyticum</i>)	4
IBR	2

Table 2: The most common postmortem diagnoses in adult bison submitted to selected pathology laboratories in 1999-2000.²⁵

No diagnosis	36
MCF	22
Pneumonia	21
Enteritis	17
Trauma	15
Liver disease	15
Fatty liver	11
BVD	7
Copper deficiency	7
Hardware	7
Lungworm	7
Emaciation/starvation	6
Abscesses	5
Abomasitis	4
Rumenitis	4
Kidney disease	4

DNA clones have been derived from such cell lines and used to identify a DNA sequence unique to OvHV-2. Using polymerase chain reaction (PCR) techniques based on this sequence, OvHV-2 has been detected in the tissues and body fluids of sheep and other animals, including bison.^{6,7}

It was recently recognized that goats are naturally infected with their own MCF-group virus known as caprine herpesvirus-2 or CpHV-2.⁸ In addition, a newly identified MCF herpesvirus has been found using DNA virus typing technology in clinical cases of MCF in white-tailed deer.⁹ This virus has not yet been named because the reservoir or carrier species has not been recognized. Whether or not these viruses can be transmitted to and cause disease in cattle or bison is not known, but researchers have observed that there are no official reports of MCF in cattle or bison without the detection of either OvHV-2 or AHV-1.¹⁰ On the other hand, CpHV-2 reportedly causes a non-responsive dermatitis and alopecia in sika deer.¹¹

Transmission

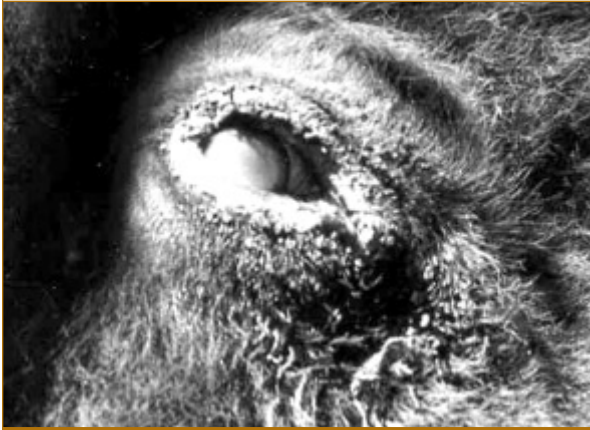
The incidence of clinical MCF in wild bison is unknown. A serosurvey for antibodies against OvHV-2 in ranches bison from Montana and Washington revealed positive titers in 2% of the animals tested.¹ Other researchers have placed this figure much higher, at 15% to 20% for the general bison population.^{12,13} The high antibody prevalence

rates and relatively low clinical disease rates suggest that many infections are inapparent and nonlethal. The alternative hypothesis is that bison have their own endemic herpes virus (BiHV) that causes a cross-reaction to ELISA tests designed to detect evidence of OvHV-2 infection.¹³ Levels of serological evidence for MCF infection in Canadian bison have never been established. Seropositive animals and clinical MCF are most common in bison from mixed sources assembled under intensive management.¹⁰

Transmission of AHV-1 occurs both vertically, through transplacental infection of the fetus, and horizontally, through inhalation of aerosol droplets or ingestion of food contaminated with tears, nasal secretions, or feces of calves shedding AHV. In adult wildebeest, AHV is highly cell-associated and not as readily transmissible.¹⁴ This is probably also true of adult carriers of the other MCF group viruses.

In sheep, infection occurs early in life with most lambs infected via horizontal transmission from the mother or members of the flock by the time they are 6-months old. There is strong evidence that sheep carrying OvHV-2 are the primary source of MCF in bison. However, the mechanism of transmission of the virus from sheep to bison is unknown. Since the virus is shed in nasal secretions of infected animals, bison are thought to be infected through contact with infected sheep and the transfer of the virus via nasal secretions. Hay feeders, common water sources, birds, and caregivers may facilitate virus transmission, although

Figure 1a: Corneal opacity and epiphora



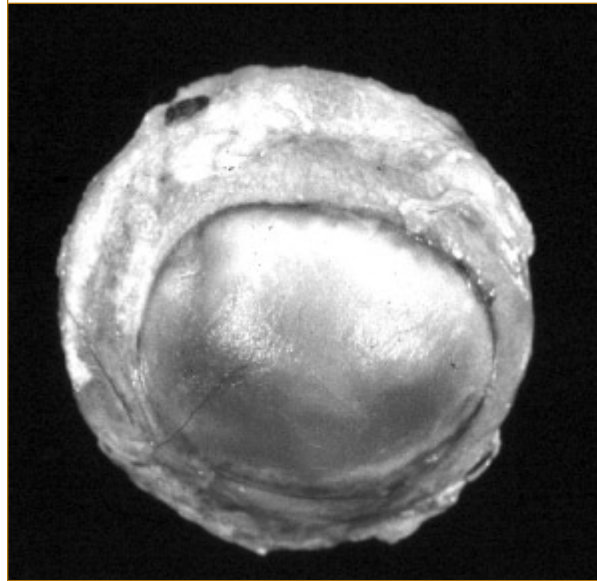
Courtesy of Donal O'Toole, University of Wyoming

OvHV-2 does not live long outside the host.¹⁵ Apparently, direct contact with sheep may not be necessary for transmission since cattle and bison have been infected by sheep held some distance away. This suggests windborne transfer of aerosolized virus, although this has never been proven.¹⁶

Bison-to-bison transmission has not been demonstrated. It is not known if bison calves can become infected from seropositive mothers or herd mates soon after birth as is the case in sheep. Because OvHV-2 is a cell-associated virus, bison and cattle have been infected experimentally by injection of either whole blood or white blood cells from a bovine calf with MCF.^{17,18} At one time, bison and other species like deer were considered “dead end” hosts, meaning that they were susceptible to infection, but were unable to pass the infection to other animals. The existence of MCF in feedlot cattle^{19,20} and more recent outbreaks of MCF in feedlot bison,¹⁶ with no apparent history of exposure to sheep, have challenged this assumption. Outbreaks occurring when there is no immediate history of previous contact with sheep also raise the possibility of latent infection of bison with OvHV-2. Epidemiologic evidence indicates that the incubation period may be as long as 200 days in cattle²¹ and from 52 to 220 days in bison.¹³

Although OvHV-2 has been associated or linked with MCF in bison, there are likely to be other factors involved in the development of MCF in bison. PCR testing for the presence of OvHV-2 in clinically normal animals has shown that not all persistently infected animals become diseased and die.¹⁰ The incidence of MCF increases in winter and under intensive management conditions. This suggests that stress or other factors that suppress the immune function may be necessary to develop clinical MCF.

Figure 1b: Dissected eyeball from bison dying of MCF showing extensive corneal edema



Courtesy of Dr. Dorothy Middleton, Veterinary Pathology, University of Saskatchewan

Clinical signs

MCF in bison is frequently manifested by sudden death of the animal following a brief course of hemorrhagic diarrhea. General clinical signs of MCF are depression, separation from the herd, anorexia, incoordination, and persistent fever (40°–41° C). In animals surviving long enough, signs appear that are similar to the so-called “head and eye” form of MCF in cattle, but are much less dramatic and not particularly diagnostic. There may be excessive salivation and nasal discharge due to erosions on the tongue and palate. Ocular discharge, conjunctivitis, and corneal opacity are often observed (Figures 1a and b). Affected animals may have diarrhea (occasionally with blood), or hematuria (bloody urine).^{1,18,22} These signs can occur in any combination and last from 1 to 4 days prior to death. Most commonly, bison infected with MCF are simply found dead. While the clinical course in bison is usually very rapid, there have been reports of survival for months.^{1,12}

Postmortem findings

The MCF virus is presumed to infect lymphoid tissues (spleen and lymph nodes) and cells lining the gut, urogenital tract, and nasal passages. The severe inflammation generally causes gastroenteritis and widespread formation of ulcers, particularly in the large bowel. This is the basis for bloody diarrhea, a common and useful diagnostic sign of

MCF in bison. The virus causes changes consistent with encephalitis, panophthalmitis, and generalized vasculitis.

Postmortem lesions vary considerably depending on the severity and course of the disease. Animals dying from peracute MCF have few lesions other than hemorrhagic enterocolitis.¹ In cases of acute and sub-acute disease, there are ulcerations throughout the alimentary tract, particularly in the esophagus and trachea, as well as corneal opacity, conjunctivitis, enlarged lymph nodes, and ulcerations or hemorrhage in the urinary bladder wall.^{1,18,22}

Microscopic findings reveal the lymphoproliferative nature of the disease. Lymphocytes infiltrate the tissue around blood vessels due to vasculitis and cause destructive changes in organs such as the kidney and liver. These lymphoid cells infiltrate the liver and kidneys of most cases. Blocked blood vessels result in local cell necrosis and inflammation leading to ulcerations in the upper respiratory, urinary, and gastrointestinal tracts.¹ Typical herpesvirus inclusion bodies are not present.

Diagnosis

A definitive diagnosis is made by microscopic postmortem examination of tissues. In live animals, the clinical signs described above and a detailed history that might include exposure to sheep or wildebeest can be used to form a presumptive diagnosis. However, presumptive diagnoses of MCF should be supported by laboratory tests.

In the live animal, a PCR test for the presence of OvHV-2 can be done on an unclotted blood sample taken in an EDTA tube. A recently developed, but less reliable, competitive-inhibition (CI)-ELISA test can also be performed on serum. Interpretation of the test results is not always straightforward. Positive CI-ELISA or PCR tests in otherwise healthy animals do not seem to be indicators of impending clinical disease and, at the moment, their significance is not well understood.^{5,23}

A postmortem diagnosis depends upon finding the typical vasculitis and perivascular lymphoproliferative lesions.¹⁹ Gross lesions include erosions and petechial hemorrhage in the nasal passages, esophagus, and

urinary bladder, as well as enlarged lymph nodes and corneal edema with opacity. There may be ulceration and hemorrhage in the large intestines. Fresh samples of kidney, liver, urinary bladder, lymph nodes in the thorax and abdomen, lung, and spleen should be taken for PCR testing and also fixed in 10% formalin for microscopic evaluation. The microscopic tissue changes in cases of MCF are unmistakable. Vasculitis and perivascular lymphoproliferation, in addition to positive diagnostic tests, form the diagnosis of MCF.

Differential diagnosis

There are many diseases that cause sudden death, including rabies,²⁴ bloat, and anthrax. If the disease is more protracted, infectious bovine rhinotracheitis (IBR), bovine virus diarrhea (BVD), vesicular stomatitis, or foot-and-mouth disease might be confused with MCF unless appropriate laboratory tests are performed.

Treatment

There is no effective treatment known for MCF. There is evidence that supportive care with antibiotics and corticosteroids may prolong the course of disease,¹² but the case fatality rate remains at or near 100%.

Prevention

There is no vaccine against MCF. Prevention is aimed at minimizing risk factors associated with the occurrence of MCF.

- It is good practice to avoid any contact with sheep and goats including wild varieties. These species should never be in contact with bison during calving or when the carrier species are nursing their young.
- Avoid the common use of stock trailers and other equipment that might have been recently contaminated.
- Minimize stress to bison caused by feeding inadequate diets and excessive handling or inappropriate handling techniques.

There is evidence that bison can be infected with OHV-2 for long periods, perhaps years, before developing clinical MCF.¹² Bison are considered “dead end

hosts,” but it may be wise to quarantine or segregate newly acquired animals from the main herd for at least 1 or 2 months as a general biosecurity measure.

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Abstracts of Interest

Malignant catarrhal fever in a bison (*Bison bison*) feedlot, 1993-2000.

O'TOOLE D, LI H, SOURK C, MONTGOMERY DL, CRAWFORD, TB.

A fatal enteric syndrome was identified in American bison (*Bison bison*) at a large feedlot in the American Midwest in early 1998. An estimated 150 bison died of the syndrome between January 1998 and December 1999. The syndrome was identified as malignant catarrhal fever (MCF), primarily the alimentary form. Clinical onset was acute and most affected bison died within 1-3 days; none recovered. Consistent lesions were hemorrhagic cystitis, ulcerative enterotyphlocolitis, and arteritis-phlebitis. Vasculitis was milder and more localized than in cattle with MCF, and in contrast to the situation in cattle, lymphadenomegaly was minimal. Virtually all infected bison examined were positive for ovine herpesvirus-2 (OvHV-2) by polymerase chain reaction (PCR) assay. A retrospective study of archived tissues established that MCF occurred in the yard as early as 1993. A prospective study was undertaken to establish the importance of MCF relative to other fatal diseases at the feedlot. The fate of a group of 300 healthy male bison in a consignment of 1,101 animals was followed for up to 7 months to slaughter. At entry, 23% (71/300) of bison were seropositive for MCF viruses, and 11% (8/71) of these seropositive bison were PCR positive for OvHV-2. Forty seronegative bison were selected at random from the group, and all were PCR negative for OvHV. There was no change in seroprevalence in the group during the investigation. The minimum infection rate for MCF virus was 36.3% (93/256). Twenty two (7.3%) of the 300 bison in the feedlot died. Of these, 15 had MCF, 4 had acute or chronic pneumonia and 3 were unexamined. Losses in the entire consignment were higher (98/1,101; 8.8% death loss);

76% of deaths were attributable to MCF. The study failed to reveal a relationship between subclinical infection and development of clinical disease.

J Vet Diagn Invest 2002; 14(3):183-193.

Breeding soundness examination of North American bison bulls

KEEN JE, RUPP GP, WITTENBURG PA, WALKER RE.

Objective: To evaluate the breeding soundness examination procedure in plains bison bulls.

Design: Multiyear (1993 through 1997) cross-sectional clinical procedure evaluation.

Animals: Two hundred and thirty-four 28- to 30-month-old bison bulls at Custer State Park.

Procedure: Breeding soundness examinations were performed on all bison bulls using 1992 Society for Theriogenology guidelines for beef cattle semen evaluation and reproductive tract examination. Linear and logistic regression analysis were used to detect correlations and associations among breeding soundness examination variables.

Results: Scrotal circumference (SC) was significantly correlated with body weight, percentage of normal spermatozoa, percentage of primary spermatozoal defects, and percentage of motile spermatozoa. Scrotal circumference was positively associated with increased odds of semen collection, satisfactory motility ($\geq 30\%$ motility), satisfactory morphology ($\geq 70\%$ normal spermatozoa), and simultaneous satisfactory motility and morphology. Receiver-operator characteristic curve analysis selected 29 cm as the optimal SC cutoff most predictive of simultaneous satisfactory spermatozoal motility and morphology. Only 36.2% (83/229) of the bison bulls had a SC of 29 cm or greater and satisfactory spermatozoal motility and morphology.

Clinical implications: SC is a good indicator of adequate spermatozoal motility and structure in bison. We recommend use of 30% spermatozoal motility, 70% normal spermatozoal morphology, and 29-cm SC as minimal satisfactory measurements for breeding soundness examinations of 28- to 30-month-old bison bulls that have been raised on forage-based nutrition.

J Am Vet Med Assoc 1999;214:1212-1217.

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