

# A Review of Malignant Catarrhal Fever and a Case Report in Bison

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## INTRODUCTION

Malignant Catarrhal Fever (MCF) is a clinicopathological syndrome which occurs in many ruminant species including domestic cattle,<sup>1,6,14</sup> white tailed deer,<sup>16,17</sup> Greater Kudu,<sup>2</sup> and bison.<sup>9,13</sup> In Africa MCF is caused by a cell associated herpes virus<sup>11</sup> for which the wildebeest is thought to be the carrier.<sup>14</sup> The etiologic agent is less clearly defined for the European-North American form of MCF. Viruses isolated from animals with MCF in the United States include bovine syncytial virus,<sup>15</sup> herpesvirus,<sup>15</sup> togavirus,<sup>3</sup> and a morbillivirus.<sup>4</sup> The North American form occurs most often when sheep and cattle are in close contact and is most frequent following the lambing season.<sup>6,10</sup> Once the disease has been introduced into a herd it may spread to other animals even after the removal of the sheep.<sup>14</sup> Transmission of MCF from a calf with clinical signs of MCF to an adult bison by intravenous inoculation of whole blood has been successful.<sup>9</sup>

Consistent clinical signs of MCF in cattle are high fever (106–107°F), enlargement of all lymph nodes, hyperemia of mucous membranes, erosions of mucous membranes, nasal and ocular discharge, depression, and peripheral corneal opacity. Variable clinical signs include edema of the eyelids, necrosis of epithelial tissues (muzzle, vulva, teats, and at the skin-horn junction of the feet), blepharospasm, hypopyon, congestion of scleral vessels, hematuria, diarrhea, constipation, melena, and a wide range of nervous signs.<sup>1,5,6</sup>

Gross changes may not be visible in the animal which dies acutely from MCF.<sup>6</sup> Typically however MCF is characterized by erosions of mucosal surfaces in the alimentary tract, respiratory system, and urinary bladder.<sup>1,6,14</sup> Enlargement of all lymph nodes, which are also frequently hemorrhagic, is another characteristic of MCF. The histopathologic changes of MCF seen in lymphoid tissues and small blood vessels are considered pathognomonic. The vascular lesion of MCF is a generalized fibrinoid necrotizing vasculitis without thrombosis and with accumulations of lymphoid cells within the intima.<sup>6</sup> A recent study suggests that lymphoid cell infiltration with myocyte necrosis may be more prominent features of MCF than fibrinoid necrosis.<sup>7</sup> These vascular lesions are thought to be responsible for the clinical manifestations of MCF.<sup>6,14</sup> There is, however, evidence that many of the epithelial lesions of MCF may be lymphocyte mediated.<sup>8</sup> Lymphoid lesions are characterized by destruction of small mature lymphocytes and proliferation of immature lymphoblasts.<sup>6</sup>

The purpose of this report is to describe the clinical and pathologic findings in an epizootic of MCF in a herd of bison (*Bison bison*). We shall also compare those findings to the clinicopathologic syndrome seen in cattle and establish a basis for making a differential diagnosis.

## CLINICAL FINDINGS

Five head of bison, two cows, one heifer, and two bulls were pastured in north-central Iowa during the summer of 1981. The bison had no history of vaccination. Sheep were present on the same farm but had no contact with the bison. Other species on the farm included llamas and a herd of cattle. The cattle

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shared a common pasture with the sheep at various times including the lambing season.

On July 19 one cow was found dead, no signs of illness had been previously observed. A necropsy was performed by the attending veterinarian but a definitive diagnosis was not made. On July 28 the owner noticed depression, coughing, and diarrhea in the heifer and the older of the two bulls. The bull proceeded to die the morning of July 29; a necropsy was performed by the attending veterinarian and tissue samples were sent to the ISU Veterinary Diagnostic Laboratory. The heifer was treated with oxytetracycline,<sup>a</sup> erythromycin,<sup>b</sup> and B-vitamins. Following treatment the heifer was transported to the ISU College of Veterinary Medicine Teaching Hospital.

Physical examination upon arrival revealed a depressed, incoordinated animal. The bison had a mucopurulent nasal discharge, eroded mucous membranes, diarrhea, rapid respiration, elevated temperature (106°F), injected sclera, and peripheral corneal opacification. The animal died at 6:30 pm on July 29 and was sent to the ISU Department of Veterinary Pathology for necropsy.

### **PATHOLOGIC FINDINGS**

The female submitted for necropsy had numerous erosions and ulcerations of oral mucosa and dark tarry feces which were evident on external examination. The larynx and surrounding tissues were edematous and covered with reddish brown to black necrotic tissue. A yellow tenacious exudate extended into the trachea. The dorsal third of the lung was dark red and firm in consistency. Lymph nodes throughout the body were enlarged and appeared hemorrhagic on the cut surface. The caudal third of the esophagus had linear ulcerations and was covered with green necrotic tissue. The mucosa overlying Peyer's patches was necrotic and hemorrhagic. There was a diffuse congestion of the subarachnoid vessels of the brain. The mucosal surface of the bladder was streaked in appearance due to areas of hemorrhage.

Histologic examination revealed a nonsuppurative meningo-encephalomyelitis with a generalized necrotizing vasculitis. Fibrinoid

degeneration and perivascular hemorrhage was found in the cerebral cortex, cervical spinal cord, brain stem, rostral colliculus, cerebellum, liver, kidney, lung, esophagus, and lymph nodes. Within the meninges and neuroparenchyma there was perivascular cuffing with mononuclear cells and varying stages of tigrolysis in large neurons. Glial nodules, axonal balls, and myelin balls were also seen in some sections. There were also accumulations of mononuclear cells in the liver and lung. Ulceration was noted on tissues from the tongue, stomach, and esophagus. Both the spleen and lymph nodes were congested and hemorrhagic and lymphoid depletion was apparent in the spleen. The interstitium of the kidney was infiltrated with lymphocytes and plasma cells and there was focal degeneration of the renal tubules.

Tissues submitted from the male bison to the Iowa State University Veterinary Diagnostic Laboratory revealed similar findings. The portal areas of the liver were infiltrated with mononuclear cells and had an accompanying degeneration of blood vessel walls. There were areas of necrosis in small vessels and glomeruli of the kidney with foci of mononuclear cells.

### **SEROLOGIC FINDINGS**

Fluorescent antibody tests on tissue sections of both the heifer and bull proved to be negative for BVD virus. Tissues from the heifer were also negative for BVD virus on inoculation of cell culture.

### **DISCUSSION**

The most prominent clinical signs of MCF in this case (depression, diarrhea, nasal discharge, increased temperature, and corneal opacity) are identical to the clinical signs most often described in both cattle and bison.<sup>1,5,9,13</sup> Palpable enlargement of superficial lymph nodes is frequently noted in cattle but no enlargement of lymph nodes was detected in this case until necropsy. Other authors have noted that while lymphoid hyperplasia may be marked in the bison with MCF, it is less than that seen in cattle.<sup>9</sup> The nature of bison may, in general, make the detection of the more subtle clinical signs of MCF relatively difficult.

The gross lesions in this case are similar to those that have been described for cattle.<sup>6,14</sup> There are, however, some differences in the

<sup>a</sup> Liqueamycin, Pfizer Inc, NY, NY.

<sup>b</sup> Erythro-200, CEVA/Abbott Laboratories Inc, Overland Park, KS.

severity and location of lesions in this case of bison MCF and those reported elsewhere in the literature. We found no significant lesions in the rumen, reticulum, omasum, and abomasum while others have noted reddening and ulceration of the mucosa in these areas.<sup>9,13</sup> The most striking lesion in this case was the severe necrosis and edema of the larynx which has not been documented in other cases of bison MCF.<sup>9,13</sup>

The microscopic lesions in tissues submitted from the bull and heifer are classical and form the basis for a diagnosis of MCF.<sup>6,7,8,14</sup>

Therapy in this instance was not successful, nor has an effective treatment regimen been described in the literature. Mortality in this epizootic was 100% as it has been in previous outbreaks of bison MCF.<sup>9,13</sup> The possibility of subclinical and nonfatal forms of MCF does exist but since the only definitive method of diagnosis is by histopathology, such forms of MCF have not been confirmed.<sup>13</sup>

The variety of animals present on the farm in this case raises some interesting epidemiologic questions. Sheep are thought to be carriers of MCF<sup>6,10,14</sup> and were present in this instance. The sheep had no contact with the bison but grazed on the same pasture as the cattle. The fact that there were no reports of MCF in the cattle herd may suggest that there is a greater susceptibility of bison to MCF when compared to cattle. It may also be possible that the bison are carriers of the causative agent with clinical signs becoming apparent due to secondary factors. The llamas could also play a role similar to sheep as inapparent carriers.

BVD, IBR, Rinderpest, Bluetongue, and Shipping Fever must be included on a list of differentials with MCF.<sup>1,5,13</sup> BVD does not present with eye lesions and does not have the histopathological lesions associated with MCF. In addition serologic and isolation techniques are available for the BVD virus. In IBR, oral lesions are usually not present although eye lesions may occur. IBR is also transmitted more rapidly than MCF and is very rarely fatal. Rinderpest is characterized by a rapid spread and karyorrhexis of lymphocytes. Bluetongue is rarely clinically

evident in cattle and may be differentiated from MCF by histopathology. Shipping Fever responds well to treatment and does not have oral lesions.<sup>1</sup>

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