

Malignant Catarrhal Fever in a Shira's Moose (*Alces alces shirasi* Nelson)

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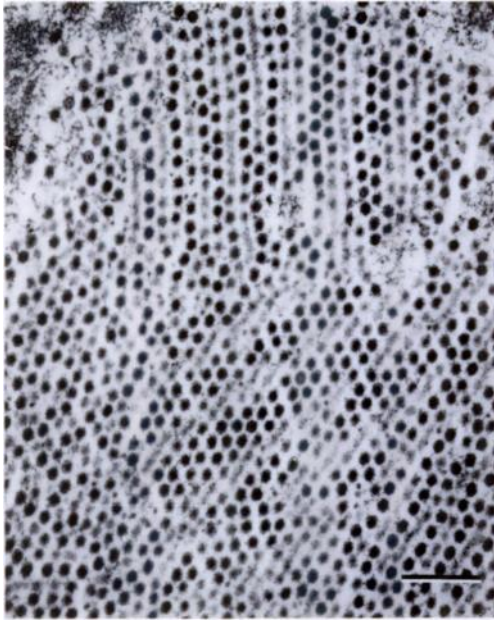


FIGURE 6. Electron micrograph of virions in an intranuclear body of a cell from Figure 3. Cubic symmetry and paracrystalline formations are evident. Bar = 500 nm.

birds. Immunodeficiency has been reported in infectious bursal disease, a reovirus infection of the bursa in domestic chickens (Hitchner, 1978, *In Diseases of Poultry*, 7th Ed., Hofstad et al. (eds.), Iowa State Univ. Press, Ames, Iowa, pp. 647–653).

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Malignant catarrhal fever (MCF), a sporadic, generally fatal, viral disease has been reported in a variety of domestic and wild ruminants, including many cervid species (Plowright, 1981, *In Infectious Diseases of Wild Mammals*, 2nd Ed., Davis et al. (eds.), Iowa State Univ. Press, Ames, Iowa, pp. 126–138; Heuschele, 1982, *Proc. U.S. Anim. Health Assoc.* 86:

552–570). Two European reports (Andersson, 1953, *Nord. Veterinaarmed.* 5: 847–854; Altmann et al., 1973, *Proc. 15th Int. Symp. Dis. Zoo Anim.*, Berlin, pp. 41–49) describe MCF in captive “elk” but do not give the species of the affected animals; presumably these case reports refer to *Alces alces*. We believe this is the first report of MCF in a moose in North America.

Malignant catarrhal fever is being rec-

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ognized with increasing frequency as an important disease of farmed deer and cervids in zoological gardens (Heuschele, 1982, op. cit.; McAllum, 1982, N.Z. Vet. J. 30: 99–101; Wilson et al., 1983, N.Z. Vet. J. 31: 7–9). In most North American cases, cervine MCF in zoological gardens is probably associated with alcelaphine herpesvirus from wildebeest (*Connochaetes* spp.), however, in farmed or captive deer not in zoological gardens, herpesviruses seldom have been isolated and there is often a history of association with sheep (Heuschele, 1982, op. cit.). Malignant catarrhal fever in domestic cattle of the United States, Europe, and Australia often has been associated with contact between cattle and domestic sheep, frequently at the time of lambing. The etiologic agent of sheep-associated MCF in domestic cattle in North America and Europe has not been positively identified (Liggett et al., 1978, Am. J. Vet. Res. 39: 1249–1257; Plowright, 1981, op. cit.).

In October 1980, a 2½-yr-old captive female Shira's moose at the Wyoming Game and Fish Department Sybille Wildlife Research Unit was noted to be anorectic, weak, and to have bilateral limbal corneal opacity. The condition of the animal deteriorated over the next 2 days to lateral recumbency with struggling and paddling, nystagmus, and shallow rapid respiration and was euthanized by intravenous barbiturate (Sleepaway, Fort Dodge Laboratories, Inc., Fort Dodge, Iowa 50501, USA).

At necropsy there was little subcutaneous fat and the hair coat was dry and brittle. Hemorrhages were present in the subcutis over bony prominences. The pericardial sac contained a small amount of clear fluid and there were a few 1–5 mm subendocardial and myocardial hemorrhages. Interlobular emphysema and scattered dark-red, firm, depressed lobules were present in the lungs, and thick mucus containing plant material was seen in small bronchi. Most lymph nodes were

enlarged and edematous. A few scattered, 1–3 mm diameter, fluid-filled cysts were on the mucosa of the soft palate. Petechial hemorrhages occurred in the serosa of the small intestine and the mucosa was roughened and hyperemic. Whipworms (*Trichostrongylus* sp.) were numerous in the cecum. The wall of the urinary bladder was thickened and the mucosa appeared reddened and mottled. Gluteal and quadriceps muscles were pale and thin yellow fluid was found along fascial planes. The cerebral vessels were congested and pale soft masses filled the slightly dilated lateral ventricles of the brain.

Klebsiella pneumoniae (Schroeter, 1886) and *Escherichia coli* (Migula, 1895) were isolated from the lung by standard bacteriologic techniques; bacteria were not isolated from blood, spleen, lymph nodes, or cerebrospinal fluid. Fluorescent antibody tests and mouse inoculation trials were negative for rabies virus. Virus isolation was not attempted.

Microscopic examination of formalin fixed, hematoxylin-eosin stained sections of most organs revealed widespread vasculitis characterized by perivascular infiltrates of lymphoid cells occasionally involving the tunica media and intima. Only rarely was fibrinoid change observed. Vasculitis was particularly extensive in the brain (Fig. 1). The choroid plexus of the lateral ventricles was essentially replaced by a mass of fibrin containing numerous lymphoid cells, macrophages, and occasional neutrophils (Fig. 2). Eosinophilic inclusion bodies were observed in the cytoplasm of neurons in the hippocampus and, rarely thalamus and medulla oblongata. Similar inclusions have been found in brains of non-rabid moose (Leighton and Williams, 1983, J. Wildl. Dis. 19: 285–288). Corneal edema and lymphoid cell infiltrates in the lamina propria of the cornea, the ciliary body, and around choroidal and retinal blood vessels, were present. Focal aggregates of lymphoid cells were observed in the in-

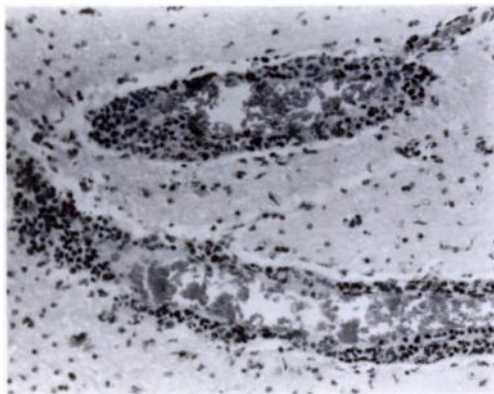


FIGURE 1. Vasculitis and perivascular lymphoid cell infiltrates in the brain of a Shiraz moose with MCF. H&E, $\times 110$.

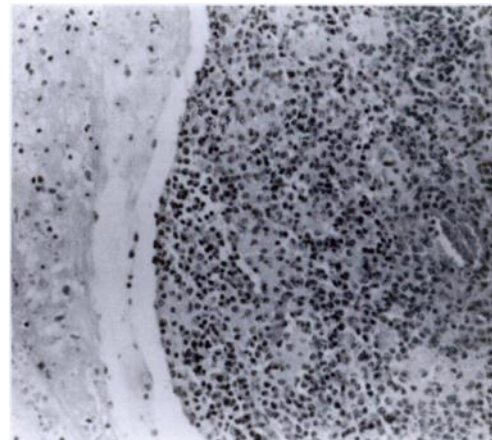


FIGURE 2. Destruction of the choroid plexus of the lateral ventricle by lymphoid cell infiltration and fibrin exudation in a Shiraz moose with MCF. H&E, $\times 125$.

terstitium and pelvis of the kidneys and in periportal regions of the liver. Diffuse or focal lymphoid cell infiltrates, often associated with focal hemorrhages and/or necrosis were observed in subepithelial or intraepithelial locations in urinary bladder, oral mucosa, small and large intestine, and uterus. Paracortical hyperplasia, focal hemorrhages, and medullary histiocytosis were observed in lymph nodes. Acute, severe, degenerative myopathy of skeletal muscle was probably due to struggling while recumbent. Various striated muscles contained protozoal cysts (*Sarcocystis* sp.). Changes in the lung were compatible with aspiration pneumonia.

The clinical and pathologic features of the disease in this moose were consistent with those described in other cervids and bovids with MCF (Liggett et al., 1978, op. cit.; Plowright, 1982, op. cit.). The source of infection for the moose was not identified. Although caught in the wild as a neonate, she was hand-raised on the premises and held in a large semi-natural enclosure with another adult female moose raised at Sybille. Rocky Mountain elk

(*Cervus elaphus nelsoni* Bailey), mule deer (*Odocoileus hemionus hemionus* (Rafinesque)), white-tailed deer (*O. virginianus* Zimmermann), bighorn sheep (*Ovis canadensis canadensis* Shaw), mouflon (*Ovis musimon* Pallas), and pronghorn (*Antilocapra americana americana* (Ord)) had fence-line or direct contact with the moose. Six wk prior to onset of disease in the moose, two healthy adult wether domestic goats were introduced into the facility. No births had occurred at Sybille for 3 mo. Disease suggestive of MCF was not observed in any animal at Sybille prior to or following the death of the moose.

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