Malignant catarrhal fever in wild Swedish moose (*Alces alces* L).

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Abstract: Malignant catarrhal fever (MCF) was diagnosed in two moose (*Alces alces* L) from the county of Uppland, Sweden. The animals showed an abnormal behaviour e.g. nervous signs as circling movements, nodding of the head and lack of awareness. One of these two animals was shot. The other was found dead on the day after being observed. Gross and histologic findings revealed characteristic lesions of MCF. Serological investigation and viral isolation for BVD and IBR/IPV viruses were negative.

Key words: *Alces alces* L, moose, malignant catarrhal fever.

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Introduction

Malignant catarrhal fever (MCF) is a disease of wild and domestic ruminants with a world wide distribution. Two or may be three forms with similar clinical and pathological lesions are recognised. The African form or «wildebeest derived» is caused by a herpesvirus (bovine herpesvirus 3, Alcelaphine herpesvirus 1, MCF virus) (Plowright et al. 1960,1965). The transmission of the natural infection is not fully known. Nasal shedding of MCF virus may be the method for virus transmission among the wildebeests and from wildebeest to cattle (Plowright 1968; Rweyemamu et al. 1974). The agent of the so-called «sheep-related» form has not been determined. MCF in cattle has often been associated with lambing ewes (Piercy 1954; Pierson et al. 1973; Selman et al. 1974). All reported cases, however are not associated with sheep.

A new form of MCF has been recently reported, affecting feedlot cattle without sheep or wild ruminant contact (Fenner et al. 1987). The causal agent of this form is not known.

The African and the sheep-associated forms show differences in incubation period, disease course and the results of experimental transmission (Pierson et al. 1979). The incubation period is usually longer in the sheep-associated form than in the African form. The disease course is short and most of the affected animals die because of diarrhea.

The experimental transmission is difficult and more infected material (e.g. blood) is usually required to transmit experimentally the sheep-associated form (European form).

The incidence of the disease is generally sporadic and low but the mortality is high (Plowright 1968). The disease has been reported in cervidae in Europe, Australia, New Zealand and North America (Clark et al. 1970; Wobeser et al. 1973; Sanford et al. 1977; Denholm and Westbury 1982; McAllum et al. 1982; Reid et al. 1987). Borg (1978) has reported the occurrence of blindness and opacity of the eyes in Swedish moose. He suggested it to be a result of MCF. The African and the European forms of MCF have experimentally been trasmitted to cattle (Piercy 1952; Plowright 1953, 1960; Reid et al. 1986), red deer (*Cervus elaphus* L) (Huck et al. 1961; Reid et

al. 1986), roe deer (*Capreolus capreolus* L) (Reid et al. 1986) and laboratory animals (e.g. rabbits, guinea-pigs, hamsters and rats) (Reid et al. 1986; Buxton et al. 1988; Jacoby et al. 1988).

This report describes MCF in two wild Swedish moose (*Alces alces* L).

Material and methods

In the autumn of 1988, two female moose, age ranging from three to four years, were seen displaying abnormal behaviour. They moved their head from side to side, and in addition showed extraordinary aberrations with staggering, circling and high movements of the legs. The animals seemed not to be aware of the surroundings. One of them was shot (Case I), and the other was found dead the day after having been observed (Case II).

Both animals came from the same area of the county of Uppland. Earlier an other dead moose had been found in the same surroundings. All three were reported to have had close contact with sheep. The two cases showing neurological signs were submitted to the National Veterinary Institute. Routine necropsy was performed. Samples for histology were taken from the CNS, liver, kidney, eye, lymphnode, spleen, nasal-, buccal- and esophageal mucosa, mucous membranes of the lower alimentary tract, lungs, urinary bladder and the uterus. They were fixed in 10% formaline, embedded in paraffin, sectioned at 5–6 μ and stained with hematoxyline – eosin.

Materials were also collected for routine bacteriology and isolation of BVD and IBR/IPV viruses. In addition, a serum sample was collected for antibody detection against both viruses in Case II.

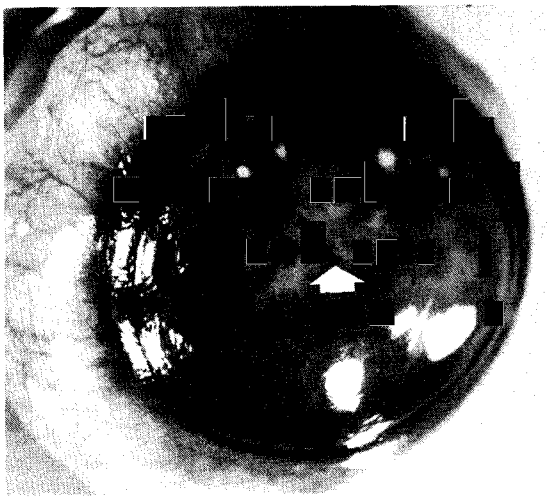

Figure 1. Case II. Eye exhibiting irregular opaque areas of the corneas (arrow).

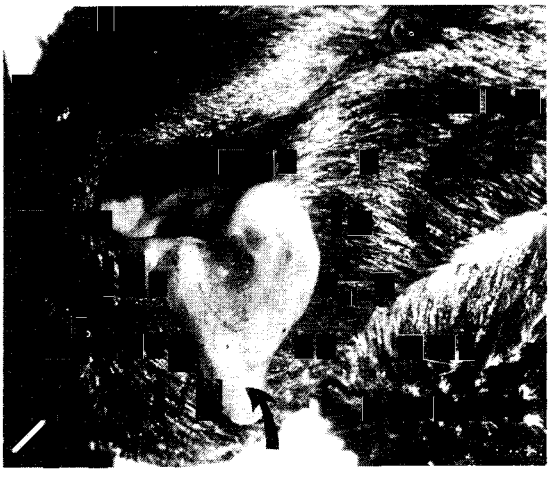

Figure 2. Case II. Muco-purulent discharge coming out from the nostril (arrow).

Results

Gross pathology

Case I. A three years old female. The body condition was below normal and the carcass was relatively fresh. Lesions were distributed in different organs. The brain was congested and edematous. Bilateral corneal cloudiness with white small spots was observed. The conjunctiva was hyperemic and edematous. Erosions and ulcers were not found in the digestive and respiratory mucosa, but the abomasal mucosa was hyperemic. The intestinal content was watery – tinged with blood. The mucosa was severely hyperemic in all segments of the intestine indicating hemorrhagic enteritis. The liver was enlarged, congested and friable in consistency. A moderate amount of froth was found in the trachea and the bronchi. In the lungs edema, hyperemia, hemorrhages and emphysema were observed.

The visceral pleura had fibrotic adherence to the diaphragm. The pericardial sac was partially attached to the heart. Approximately one litre of blood-tinged fluid was found in the sac. There were small points of necrosis on the kidney surface looking like infarctions. The mucosa of the uterus was hype-

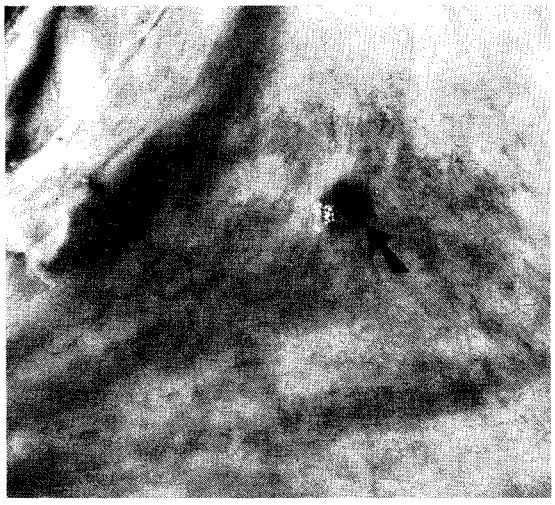

Figure 3. Case II. Ulcer (arrow) in the mucosa of the urinary bladder.

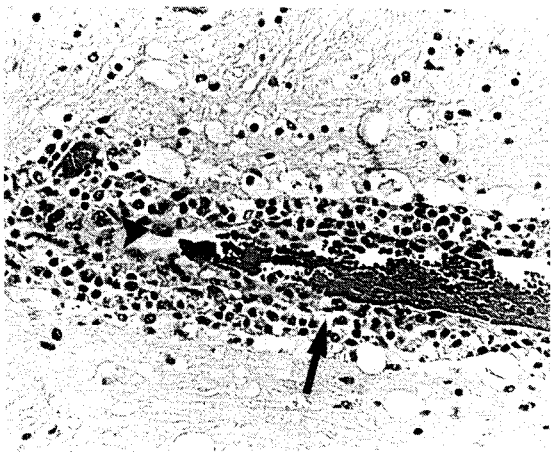

Figure 4. Brain from case I. Fibrinoid necrosis of the vessel wall (arrow) with accumulation of lymphocytes, large undifferentiated reticulo-endothelial cells and endothelial hyperplasia (arrow head). H&E x 250.

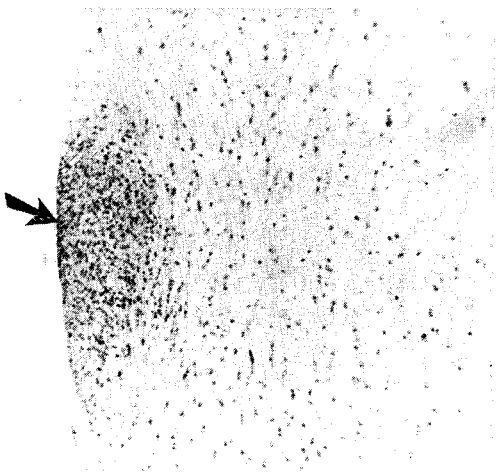

Figure 5. Cornea from case I. Corneal ulceration with remaining basement membrane of the epithelium and accumulation of neutrophils (arrow). H&E x 110.

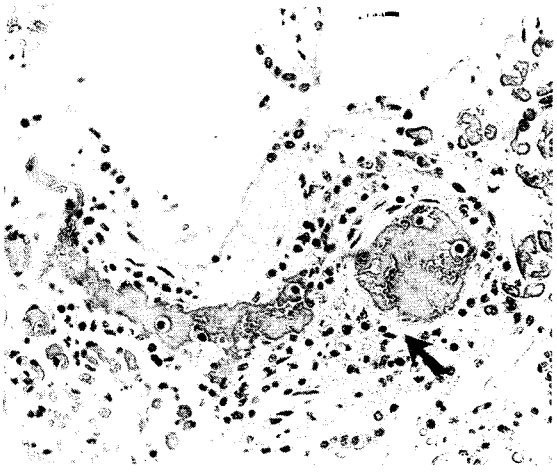

Figure 6. Lung from case I. Necrosis and mononuclear cell infiltration of the vessel wall (arrow). H&E x 250.

remic. Enlarged spleen with rounded edges and prominent follicles on the cut surface were seen. Lymph nodes were markedly enlarged, hyperemic and often edematous.

Case II. A three or four years old female. The state of nutrition was normal. The carcass was moderately decomposed. The brain was edematous and congested as in Case I. Petechial hemorrhages were also present in the brain parenchyma. Irregular bilateral corneal opacity was observed (Fig. 1). The small white spots in and on the cornea were the same in both cases. The conjunctival changes were similar to that of the previous case.

Muco-purulent discharge was oozing from the nostrils (Fig. 2).

In contrast to case I, numerous erosions and ulcers were seen in the mucosa of the mouth, nose, esophagus and the abomasum. The abomasal mucosa was hyperemic. The appearance of the intestinal content was the same as in the other moose and the mucosa of all parts of the intestine was hyperemic. The liver was

fatty with a greasy cut surface. A small amount of froth was observed in the trachea and the bronchi. The lungs were hyperemic, edematous, and there was hemorrhages as well.

The kidneys were highly decomposed. The urinary bladder mucosa was severely hyperemic. Ulcers and erosions were also found on the mucosa of the urinary bladder (Fig. 3) and the vagina.

The changes observed in the uterus, spleen and the lymph nodes appeared the same in both cases.

Histopathology

Case I. The brain showed a nonpurulent meningo-encephalitis characterized by edema, hyperemia and slight mononuclear cell infiltration in the meninges. Mono-nuclear perivascular cuffs with occasional hemmorrhages were present. In the vessel walls, fibrinoid necrosis with lymphocytes, macrophages and large undifferentiated reticuloendothelial cells were seen (Fig. 4). Endothelial hyperplasia was also found in some vessels (Fig. 4).

Bilateral panophtalmitis was encountered. The blood vessels were congested. There was an exudate with heavy infiltrates by neutrophils in the anterior and posterior chambers of the eye. Marked vasculitis and perivascular cuffing were present in the iris and choroid. There was an increased vascularization of the cornea. Corneal ulceration surrounded by neutrophils was also present (Fig. 5).

The intestines showed no changes except hyperemia. Centrolobular necrosis and slight mononuclear cell reaction in the portal spaces of the liver was observed.

The lungs were hyperemic, edematous and emphysematous. A subacute bronchopneumonia characterized by mononuclear cells in the bronchioli and the alveoli was evident. In addition, there was a very marked mononuclear perivascular cuffing. The vessel walls showed presence of mononuclear cells and necrosis indicating vasculitis (Fig. 6). In the uterine wall, hemorrhages and dilated vessels filled with blood were observed. The lymphoid follicles of the spleen and the lymph nodes were slightly hyperplastic. Marked edema and hyperemia were also seen in the lymph nodes.

Case II. The lesions in the brain were similar to those described in Case I, although there was no endothelial hyperplasia in the vessel walls. The neurons were surrounded by glial cells in the cerebrum.

The histological lesions found in the eyes were much the same as for Case I, except for the lack of corneal ulceration but there were areas of focal necrosis and neutrophilic cellular infiltration in the cornea associated part of the conjunctivae. Necrosis associated with polymorphonuclear cell infiltration was found in the outer part of the conjunctivae.

In general the upper alimentary tract was hyperemic. Ulcers with mono- and polymorphonuclear cell infiltration in the buccal and esophageal mucosa were seen. The epithelial lining was also sloughed off in some areas of the mucosa.

The intestinal mucosa was autolytic. Mononuclear inflammatory cells were found in the submucosa, however.

The microscopic changes in the lungs were strictly confined to the bronchi and the vessels. There was thickening of the smooth muscles of the bronchial walls. In the lumen of the bronchi desquamated epithelial cells and a slight number of mononuclear cells were present.

Vasculitis and perivascular cuffing had the same appearance as in Case I.

The urinary bladder showed heavy infiltration of mononuclear and polymorphonuclear cells in the propria and the submucosa. Also some ulcers with infiltrates of neutrophilic cells were evident. Hyperemia and ulcers with neutrophilic infiltrates were seen in the vagina.

The lesions in the uterine wall, lymphoid follicles of the spleen and the lymph nodes were in accordanee with changes already described for Case I.

Microbiologic results

Bacteriological examination and attempts to isolate BVD and IBR/IPV viruses were negative. In Case II, serum neutralisation test for both viruses and the immunofluorescence test for BVD virus were also negative.

Discussion

The two moose in this study had had contact with sheep. Sheep has been suspected as carriers of MCF virus (Blood 1967; Mare 1980). Evermann (1980) proposed that BVDV or border disease agent (BD) and non-IBR herpes virus could be the causal agent. Evermann (1980) also stated that the frequency of non-IBR herpes virus carriers is increasing in cattle.

The main differences between the African form and the European form of MCF is the acuteness of the disease and the frequency of diarrhoea occurring in cattle with the latter form (Pierson et al. 1979). According to Göetze (1930) MCF is divided into four types, peracute, alimentary, head and eye and the mild type. The head and eye type is the most commonly described in cattle and deer (Huck et al. 1961; Clark et al. 1970; Sanford et al. 1977). The diagnosis of MCF is usually based on clinical, gross and histopathological findings and the transmission to susceptible animals.

The clinical and pathological changes observed in this study were comparable to the head and eye type of the MCF (Jubb et al. 1985). The characteristics almost pathognomonic vascular lesions were found in the brain, the lungs and the eyes. Earlier, MCF has been suspected in moose in Sweden but was not histologically confirmed (Borg et al. 1978). The diseases known in Sweden which could be confused with MCF are IBR, BVD/MD and Tick-borne encephalitis (TBE).

Antibodies to IBRV and BVDV in moose have been previously reported in North America (Thorsen and Henderson 1971; Zarnke and Yuill 1981; Kocan et al. 1986). IBR is mainly a respiratory disease with conjunctivitis but lacking the corneal involvement of MCF: An infection by herpes-virus antigenically related to IBRV can produce ocular and mucosal lesions in reindeer. However, this virus (CHV-1) does not induce vascular lesions typical for the MCF (Rockborn et al. 1989).

The lesions of BVD/MD are usually found in buccal- and nasal mucosa, pharynx, abomasum, intestine and the lymphatic tissue; and lacking the vascular involvement of MCF.

The erosions of the MCF and the mucosal disease are macroscopically similar though, those in the MCF appears after the lymphocytic infiltration (Plowright 1953). The erosions in BVD/MD start from the epithelial degenerations (Dow et al. 1956).

Tick-borne encephalitis (TBE) has been reported in a moose calf in Sweden. This animal showed a nonpurulent meningoencephalitis with focal demyelination, chromatolysis and destruction with neuronophagia of the Purkinje cells, reactive gliosis, and perivascular cuffings with mononuclear cells (Svedmyr et al. 1965). In the present investigation, neuronophagia was found in one case, but it did not concern Purkinje cells.

Epizootic hemorrhagic disease (EHD) in wild ruminants is a disease of the alimentary tract. The thrombosis and widely spread hemorrhages reported in EHD (Fletch et al. 1971) were not encountered in this study. Experimental infection of moose with EHD did not cause mortality (Hoff and Trainer 1978).

Serological evidence of Blue tongue in moose has been reported in North America (Hoff and Trainer 1978).

Histologically, vasculitis, lymphocytic perivascular cuffing and endothelial hyperplasia were reported in Blue tongue infected deer (Stair et al. 1968). Endothelial hyperplasia was encountered in this investigation, but the vasculitis was more severe than that described in the Blue tongue. In addition EHD and Blue tongue are not recorded in Sweden.

In the present study, the distribution of the lesions with the characteristic almost pathognomonic histological findings and the negative results for BVD and IBR/IPV virus isolations speak in favour of the MCF.

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