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ENDEMIC MALIGNANT CATARRHAL FEVER AT THE SAN DIEGO WILD ANIMAL PARK

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Abstract: Malignant Catarrhal Fever was diagnosed in an Indian Gaur *(Bos gaurus gaurus),* a Barasingha Deer, *(Cervus duvauceli duvauceli),* and four Javan Banteng *(Bos javanicus javanicus)* at the San Diego Wild Animal Park between July, 1976 and January, 1979. Three of the four Banteng lived adjacent to an exhibit in which wildebeest were born at 29,)8 and 82 days prior to the Banteng's deaths. The disease was characterized by pyrexia, conjunctivitis, diarrhea, dyspnea and rhinitis. Mortality was 100%. Post mortem lesions in the respiratory, digestive, lymphoid and nervous systems were erosions, ulcers, necrosis and hemorrhage. Microscopic lesions included lymphoid necrosis, reticuloendothelial hyperplasia and diffuse vasculitis. All virus isolation attempts were negative.

INTRODUCTION

Malignant Catarrhal Fever (MCF), a sporadic, highly fatal disease of cattle,[2,4] bison,[4,8] deer[4] and other ruminants[2,8] was first recognized in the United States in 1915.[5] Wildebeest are the natural reservoir in Africa;[2] there is some evidence that domestic sheep are carriers in the United States[2,3] African MCF is caused by a cell associated virus, Bovid herpesvirus 3, but the etiologic agent of North American and European MCF has yet to be identified. The purpose of this report is to describe the clinicopathologic features of six cases of MCF in wild animals at the San Diego Wild Animal Park (SDWAP).

MATERIALS AND METHODS

Individual case records for three animals (the Gaur, the Barasingha Deer and one Banteng) were retrieved from the files of the Zoological Society of San Diego. Slides, when available, also were retrieved and examined. The three remaining Banteng *(Bos javanicus javanicus)* were examined at necropsy within 12 h after death and representative tissue sections were fixed in 10% neutral buffered formalin, sectioned at 6 μm and stained with hematoxylin and eosin.

Virus isolations were attempted from two Banteng *(Bos javanicus javanicus).* Samples of liver, spleen, lymph node, thymus, oral ulcers, brain, cerebrospinal fluid, synovial fluid and Peyer's patches were minced and overlaid on Vero and PK-15 cells. Subsequently, these same tissues were passed in Madin-Darby Bovine Kidney (MDBK) cells. The cells were grown in Minimum Essential Media [2] with added sodium pyruvate and sodium bicarbonate. Immediately prior to adding the cells, fetal calf serum (FCS) and penicillin-streptomycin-fungizone (PSF) solution were added to yield a final concentration of 10% FCS and 1% PSF in the medium. Cells were incubated at 37 C for five days and then observed for cytopathic effects. The supernatant was then removed and passed on the same cell line for at least nine passages.

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RESULTS

All animals were housed at the SDWAP and had been there at least three months prior to their illness. The clinical signs were similar to previously reported outbreaks of MCF: conjunctivitis, diarrhea, dyspnea, rhinitis, pyrexia (39.4-40 C) and mucopurulent nasal exudate.[2,3,4,5,8] One Banteng was found dead. The Barasingha had a temperature of 39.4-40 C and was anorectic for 10 days but lacked other clinical signs usually associated with MCF. Of the remaining animals, four had conjunctivitis, three had diarrhea and rhinitis, two had corneal cloudiness, dyspnea and anorexia. Blindness, fever and hypoperistalisis were each noted in a single animal. The disease persisted up to 10 days in a Banteng and the Barasingha but five days was the mean duration of the illness. A summary of the clinical signs of MCF in animals at the SDWAP is presented in Table 1. Banteng 1, 2, and 3 (Table 1) were in an enclosure adjacent to a pen in which two wildebeest were born two days apart. Calves were born 24, 67 and 72 days, respectively, prior to the Banteng becoming ill.

Gross lesions were most common in the digestive, lymphoid, respiratory and central nervous systems. The lips, mucocutaneous junctions, mucus membranes, hard palate, gingiva, tongue, esophagus, rumen and abomasum were eroded and ulcerated. The erosions ranged from punctate or focal (approx. 0.5 cm diameter) to continuous linear erosions up to 5 cm long, some of which were hemorrhagic. The lymph nodes were diffusely reddened, swollen and hemorrhagic. On cut surface, they bulged at the edge and had a wet, glistening appearance. Spleens were normal. Rhinitis was characterized by a clear seromucoid to mucopurulent discharge with congestion, small fissures and cracks ($\leq$ 1 cm in length) or frank ulceration of the hairless skin of the nose. Sometimes, the nares were caked with dried exudate. Tracheal lumina were foamy and the mucosa were congested, congestion sometimes extending into the mainstem bronchi. Lungs were pneumonic. Aspiration of ruminal contents had occurred in two animals with resultant secondary aspiration pneumonia. One Banteng had a fibrinous pleuritis. Leptomeningitis was manifested by cloudy fluid between the meninges and brain parenchyma. Urinary bladders were normal in all animals. Slight reddening of synovial

TABLE 1. Summary of clinical signs in animals at San Diego Wild Animal Park with Malignant Catarrhal Fever.

	Gaur	Barasingha	Banteng 1	Banteng 2	Banteng 3	Banteng 4
Anorexia	x	x				
Blindness				x		
Conjunctivitis	x		x	x	x	
Corneal cloudiness			x		x	
Diarrhea			x	x	x	
Fever	x	x				
Hypoperistalisis	x					
Dyspnea	x		x			
Photophobia			x			
Rhinitis	x		x		x	
Sudden death						x
Duration of illness	5 days	10 days	5 days	1 day	10 days	

x = presence of lesions

surfaces in the coxofemoral and femorotibial joints were noted in a single Banteng.

Histologic examination revealed a diffuse vasculitis characterized by perivascular edema and cuffs of lymphocytes, macrophages, and large undifferentiated reticuloendothelial cells. Fibrinoid necrosis was irregularly present in the tunica media of the small muscular arteries. Endothelial hypertrophy was an occasional finding but frank thrombosis was present in only a single Banteng. Severe fibrinonecrotic vasculitis was in the vascular plexus of the sella turcica in two Banteng. A bright eosinophilic coagulum containing nuclear fragments was all that remained of the tunica media. Frequently, these lesions encompassed a majority of the affected vessel's circumference when viewed in cross section. A diffuse lymphocytic and reticuloendothelial infiltrate was between the vessels. Similar cells were in the renal interstitium too. Though a proliferative membranous glomerulonephritis was in the Gaur, mild hypercellularity of the glomerular tufts was more common. In two Banteng, the urinary bladders were edematous and a histiocytic infiltrate was between the muscle fibers and in the vascular adventita. Vascular lesions similar to, but not as severe as those previously described, were in the Gaur's urinary bladder.

Epithelial surfaces in the digestive and respiratory tracts, including the lips, oral and nasal mucosa, forestomachs, abomasum, small and large intestine were all similarly affected. In four of the six animals (a Gaur and three Banteng) there was either focal vaculation or ballooning of epithelial cells. Epithelial surfaces in all animals were diffusely eroded, ulcerated and necrotic. The lamina propria of both systems had a diffuse mononuclear cell population: lymphocytes, plasma cells, and large reticuloendothelial cells. Secondary neutrophilic accumulations were associated with the ulcerated foci. Hepatic lesions were characterized by focal necrosis and a mononuclear infiltrate of the portal region. Microscopic changes in the lymph nodes were consistently present in all animals. The adventita of capsular blood vessels and perinodal fat frequently had an infiltrate of lymphocytes and plasma cells. Lymphocytes were in the subcapsular sinuses. Lymphoid follicles were depleted and contained necrotic cells. Reticuloendothelial cells and endothelial hyperplasia were constant features. Spleen had similar lesions in the lymphoid follicles, reticuloendothelial and endothelial components. A Banteng had necrosis of the tonsillar lymphoid tissue with sloughing of the overlying epithelium. A diffuse vasculitis occurred in the central nervous system, including the leptomeninges, which was essentially identical to the vasculitis in the vascular plexus of the sella turcica, though not as dramatic. The remaining organs had lesions similar to those described above, or as reported by others.[1,3,4,8] The microscopic lesions are summarized in Table 2.

RESULTS OF VIRUS ISOLATION

During the early passages (1-3) some of the Vero and MDBK cells formed large rounded syncytial clumps that tended to break free of the monolayers. This manifestation was suggestive of MCF virus replication or infection but was lost on subsequent passages. Typing serum for MCF virus was unavailable; thus the presence of MCF antigen in the cell culture systems was not confirmed.

DISCUSSION

Three of the six animals in this report had clinical signs suggestive of the peracute form of MCF, two Banteng had the typical head and eye form and a Barasingha deer had an atypical form with no clinical signs other than pyrexia and anorexia. The clinical disease in these animals varied from sudden death up to 10 days duration which is consist-

TABLE 2. Prevalence of tissue involvement with lymphoid, vascular or epithelial changes.

Tissue	Species					
	Gaur	Barasingha	Banteng 1	Banteng 2	Banteng 3	Banteng 4
Adrenal	x	x	x	x		x
Cerebellum	x	x	x	x	x	x
Cerebrum	x	x	x	x	x	x
Choroid plexus						x
Esophagus	x			x		x
Eye	x		x		x	
Intestine		x	x			
Kidney	x	x	x	x	x	x
Liver		x	x	x	x	x
Lung		x	x		x	
Lymph node	x	x	x	x	x	x
Meninges	x		x	x	x	x
Myocardium		x	x	x	x	x
Oral mucosa	x		x	x	x	x
Pituitary			x		x	
Salivary gland			x		x	
Skeletal muscle					x	x
Spleen			x		x	
Stomachs	x		x			x
Trachea		x	x		x	x
Uterus						

x = lesions are present

ent with previous reports of MCF.[4,5] Some clinical features of MCF such as erythema of the skin of the udder, coronary bands and interdigital spaces, which have been observed in dairy cattle,[5] may not have been seen for a variety of reasons. First, the animals, with a single exception, were maintained in large outdoor enclosures. Though daily observation was attempted it was not always accomplished. Secondly, the animals in this report have pigmented skin. While the degree of pigmentation varies, it probably was sufficient to mask cutaneous erythema, had it been present.

The microscopic lesions observed in these exotic animals paralleled the changes described in cattle with MCF.[1,3,4] Furthermore, the vascular lesions of MCF are unique and, in themselves, sufficient for diagnosis when found in nervous tissue.[3] Lesions of the type described in the lymph nodes, kidney and adrenal are also highly diagnostic of MCF.[1] Hyaline necrosis of striated muscle fiber is a direct action on the muscle by the virus. It does not appear to be a constant feature of the disease.[3]

Berkman, *et al.*[1] indicated that endothelial hyperplasia with resultant vascular obstruction may be the basis of the epithelial necrosis. In this series endothelial hyperplasia was seen in some sections but did not produce vascular obstruction and was not widely distributed. Thrombi were found in only one Banteng. Furthermore, ischemic injury usually is accompanied by a granulocytic response,[4] but granulocytes were seldom encountered. Recently, it was suggested that the epithelial lesions associated with North American MCF are the result of cell mediated immunopathologic disease.[4]

Though viral isolation was not achieved, it is possible to speculate on the type of MCF present. In Africa, MCF occurs in cattle in contact with wildebeest; moreover, MCF is believed to be transmitted from wildebeest to cattle during the wildebeest calving season.[2] The Banteng (1, 2 & 3) becoming ill on 12 November, 25 December and 30 December were housed adjacent to an exhibit in which wildebeest were born on 18 and 20 October. Both animals gave birth near the fence separating the exhibits. The intervals between the first wildebeest calf and the Banteng deaths, 29, 68 and 82 days, respectively, are within the incubation period for MCF which can vary from 18-100 days.[3,4,6] Near the SDWAP is a dairy farm which had sporadic cases of MCF during the winters of 1975-76 and 1976-77.[5] No direct association between the dairy animals and the exotic animals was established, however, numerous free-ranging mule deer *(Odocoileus hemionus)* live in the vicinity and are known to have contact with the zoologic specimens. Their degree of contact with the dairy herd is unknown. Previous reports indicate that deer are highly susceptible natural hosts of MCF but probably are not latent carriers.[6,8]

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LITERATURE CITED

1. BERKMAN, R.N., R.D. BARNER, C.C. MORRILL and R.F. LANGHAM. 1960. Bovine malignant catarrhal fever in Michigan. II. Pathology. Am. J. Vet. Res. 21: 1015-1026.
2. KARSTAD, L.H. 1970. Malignant Catarrhal Fever. Pp. 131-133. In: *Infectious Diseases of Wild Mammals.* Davis, J.W., L.H. Karstad, D.O. Trainer, Eds. Iowa State University Press, Ames, Iowa.
3. JUBB, K.V.F., P.C. KENNEDY. 1963. *Pathology of Domestic Animals* Vol. 2, 1st ed. Academic Press, New York, N.Y. pp. 27-34.
4. LIGGETT, H.D., J.C. DeMARTINI, A.E. McCHESNEY, R.E. PIERSON and J. STORZ. 1978. Experimental transmission of malignant catarrhal fever in cattle: Gross and histopathologic changes. Am. J. Vet. Res. 39: 1249-1257.
5. ORSBORN, J.S., C.J. MARÉ, J.L. AYERS and R.E. REED. 1977. Diagnostic features of malignant catarrhal fever outbreaks in the western United States, Am. Ass. Vet. Lab. Diag. 20th Ann. Proc. pp. 215-224.
6. PIERSON, R.E., J. STORZ, A.E. McCHESNEY and D. THAKE. 1974. Experimental transmission of malignant catarrhal fever. Am. J. Vet. Res. 35: 523-525.
7. ——, D. THAKE, A.E. McCHESNEY and J. STORZ. 1974. An epizootic of malignant catarrhal fever in feedlot cattle. J. Am. vet. med. Ass. 163: 349-350.
8. WYAND, D.S., C.F. HELMBOLDT and S.W. NIELSON. 1971. Malignant catarrhal fever in white-tailed deer. J. Am. vet. med. Ass. 159: 605-610.

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