FURTHER OBSERVATIONS ON MALIGNANT CATARRHAL FEVER IN TEXAS DEER

Authors: CLARK, KEITH A., ROBINSON, R. M., WEISHUHN, L. L., and McCONNELL, STEWART

Source: Journal of Wildlife Diseases, 8(1) : 72-74

Published By: Wildlife Disease Association

URL: https://doi.org/10.7589/0090-3558-8.1.72

FURTHER OBSERVATIONS ON MALIGNANT CATARRHAL FEVER IN TEXAS DEER

KEITH A. CLARK, R. M. ROBINSON, L. L. WEISHUHN, Department of Veterinary Pathology

STEWART McCONNELL, Department of Veterinary Microbiology

Texas A&M University, College Station, Texas

Abstract: The original description of MCF in Texas deer was based on the occurrence of clinical signs and characteristic pathologic lesions. Since that time, continued observations have demonstrated the sporadic occurrence, irregular transmission, and the causative organism's apparent susceptibility to freezing which is typical of MCF. These findings lend strong supportive evidence to the diagnosis of MCF as a continuing infection in this herd.

INTRODUCTION

The description of the clinical signs and pathological lesions of malignant catarrhal fever in a herd of axis deer *(Axis axis)* and one whitetail *(Odocoileus virginianus)* was previously reported.[1] Since then, another spontaneous case with typical lesions occurred in that herd, and the disease was experimentally transmitted to a white-tailed deer.

A 2 year old axis buck was observed with weakness, emaciation, lethargy, and corneal opacity following an 18 month disease free interval in the herd. This animal was killed and necropsied. Tissues were fixed in formalin for microscopic examination and heparinized whole blood, spleen, lymph node, and brain were placed on ice for transmission and virus isolation attempts.

MATERIALS AND METHODS

A crude suspension was prepared by mincing spleen and lymph nodes in 0.85% saline solution. This suspension and whole blood were used as an inoculum. Within 24 hours of the death of the affected deer, three animals were injected intravenously according to the following schedule:

Yearling axis deer:

20 ml blood and 20 ml tissue suspension

Holstein calf:

20 ml blood and 60 ml tissue suspension

Yearling whitetail buck:

20 ml blood and 40 ml tissue suspension

In attempted virus isolation, 20% suspensions of kidney, lung, heart, liver, brain, spleen, and lymph nodes were prepared in Hank's balanced salt solution. The suspensions were clarified by differential centrifugation, checked for bacterial contamination, and stored at —70 C until needed.

Monolayers of primary bovine spleen cells were prepared using a modification of the technique described by Malmquist *et al.*[2] Cells were dispersed by enzymatic digestion and seeded into 500 ml prescription bottles. Subcultures were made from confluent monolayers of cells and used for virus isolation attempts.

RESULTS

Pathologic findings:

Necropsy of the spontaneous case revealed serous atrophy of mesenteric fat and kidneys which were greatly swollen

This paper was presented at the Annual Conference of the Wildlife Disease Association, Colorado State University, Fort Collins, Colorado, U.S.A., August 25-27, 1971.

with reddening of the renal pelvis. Microscopic examination revealed severe arteritis, phlebitis and lymphangitis involving vessels in all lymph nodes, both kidneys, hepatic portal areas, heart, epididymus, and mesentery. The arterial lesions were characterized by marked intimal proliferation with obstruction of the lumen, infiltration of lymphocytes and macrophages throughout all layers of the vessel, and fibrinoid degeneration in the tunica media. Pyknosis of smooth muscle nuclei was evident (Figure 1). Widespread severe perivasucular cuffing was present in the brain and spinal cord (Figure 2). The eyes had degeneration of the matrix of the cornea with infiltration by lymphocytes and a few neutrophils (Figure 3). Corneal erosions were present, and perivascular cuffs were found at the limbus and in the retina.

Experimental findings:

Eighteen days after inoculation, the white-tailed deer died suddenly of spontaneous hemothorax, apparently the result of massive diapedisis; no break in vascular walls could be found. There was

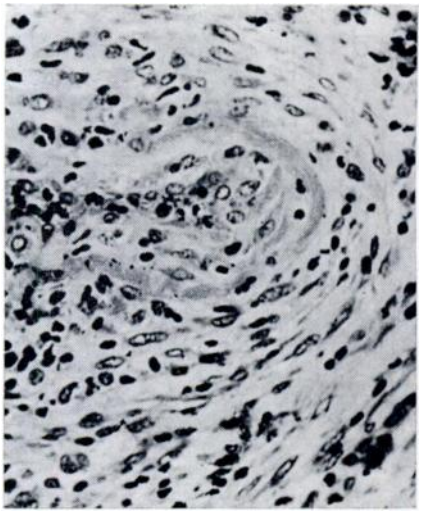

FIGURE 1. Necrotizing arteritis. Pyknotic and karyorrhectic nuclei, with fibrinoid present in the vessel wall which is greatly thickened by macrophages, fibrous tissue, and lymphocytes. Lumen is almost obliterated.

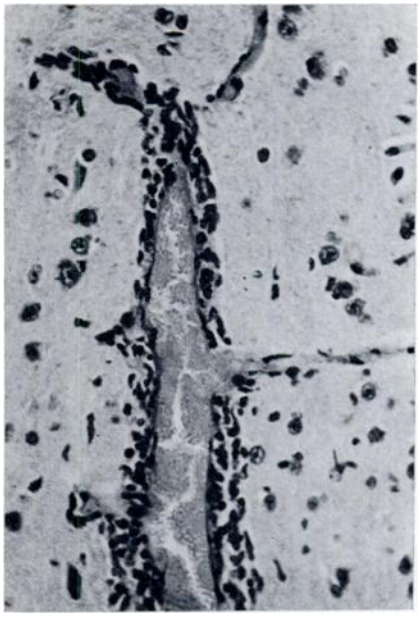

FIGURE 2. Perivascular lymphocytic cuffing, cerebrum.

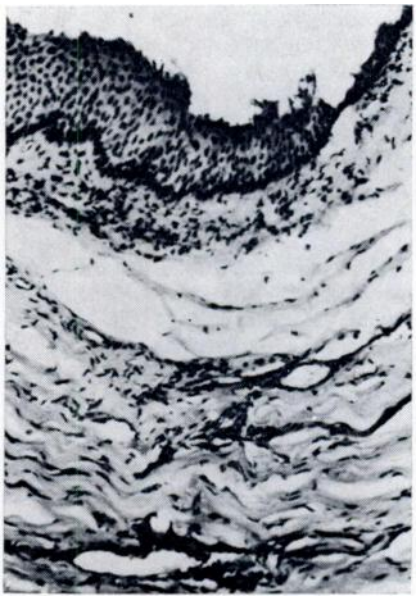

FIGURE 3. Cornea. Collagen strands are separated by edema; hypercellularity of lamina propria caused by infiltration by lymphocytes. Erosions are present in the epithelium.

catarrhal rhinitis and enlargement of lymph nodes and spleen. Microscopic examination revealed widespread necrotizing arteritis, perivascular lymphocytic cuffs throughout the brain, and keratitis identical to that seen in the axis deer. Neither of the other animals showed any signs of illness.

No evidence of a virus-induced cytopathic effect was seen in any of the three blind passages made from the suspect tissues. Some slight cytotoxicity was seen on primary isolation attempts with lung and lymph node suspensions but no organisms were isolated.

DISCUSSION

A definitive diagnosis of malignant catarrhal fever is extremely difficult to obtain. This difficulty exists because of the variable clinical syndrome, the irregular transmissibility of the disease, and a lack of knowledge concerning the character of the viral agent. Several investigators have successfully transmitted the disease from infected to susceptible animals, but usually only after several attempts, and with less than 100% morbidity.[3] The causative virus has been isolated *in vitro* in a bovine thyroid cell culture system.[4] To date we have been unable to duplicate this work primarily because our cases have been sporadic and we have not found a suitable method for storing the infected tissue to preserve the virus until we are ready to use it. Diagnosis is usually based upon clinical signs, pathological lesions, and the absence of positive tests that would incriminate other diseases.

ADDENDUM

Approximately six weeks after completion of the work described above, the first of six spontaneous cases of a similar syndrome occurred in a captive herd of white-tailed deer at this research facility. All cases were sporadic and direct contact between individuals was impossible, since they were housed in separate nonadjacent pens. The clinical signs were highly variable, including weakness, reluctance to stand, ulceration of the muzzle, hemorrhage within dermal papillae, and diarrhea. Death occurred in all cases. Neither these animals nor fawns inoculated with blood from them had increases in complement - fixation titers to bluetongue virus. Histologically, the lesions were identical to those described above. This information is included to further demonstrate the susceptibility of white-tailed deer to MCF.

LITERATURE CITED

1. CLARK, K. A., R. M. ROBINSON, R. G. MARBURGER, L. P. JONES, and J. H. ORCHARD. 1970. Malignant catarrhal fever in Texas cervids. J. Wildlife Dis. 6: 376-383.
2. MALMQUIST, W. A., M. J. VAN DER MAATEN, and A. D. BOOTHE. 1969. Isolation, immunofluorescence, and electron microscopy of a syncytial virus of lymphosarcomatous and apparently normal cattle. Cancer Res. 29: 188-200.
3. PIERCY, S. E. 1952. Studies in bovine malignant catarrh. II. Experimental infection of cattle (continued). Brit. Vet. J. 108: 35-47.
4. PLOWRIGHT, W., R. D. FERRIS, and G. R. SCOTT. 1960. Blue wildebeest and the aetiological agent of bovine malignant catarrhal fever. Nature, Lond. 188: 1167-1169.

Received for publication September 8, 1971