SIX MULE DEER (ODOCOILEUS HEMIONUS) DEAD IN SMITH VALLEY, NEVADA, USA

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WILDLIFE HEALTH PUZZLE

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Six Mule Deer (*Odocoileus hemionus*) Dead in Smith Valley, Nevada, USA

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THE PUZZLE

In February 2021, concerned citizens reported illness and approximately six deaths of unknown cause in free-ranging mule deer (*Odocoileus hemionus*) in Smith Valley, Nevada, US, to the Nevada Department of Wildlife (NDOW).

INITIAL INVESTIGATION

The NDOW found one mule deer dead and chemically immobilized two apparently sick deer. One deer died during chemical immobilization, and the other was euthanized due to poor prognosis. Both were immobilized using a combination of butorphanol, azaperone, and medetomidine delivered by remote injection. Field necropsies were performed on all three deer.

INITIAL FINDINGS

Collectively, findings in the three mule deer included firm, dark red lungs (3/3), multifocal ecchymoses and petechiae in the liver (2/3), heart (2/3), kidneys (1/3), brain (1/3), and bladder (1/3), hemorrhagic peritoneal (2/3), thoracic (1/3), and pericardial effusion (1/3), and enlarged mesenteric lymph nodes (1/3). After fixation and sectioning, the hemorrhages in one kidney were identified as multifocal renal infarcts (Fig. 1).

CHALLENGE FOR THE READER

What is your list of top probable causes?

PROBABLE CAUSES

Death in multiple mule deer and gross findings of multi-organ hemorrhage prompted concern for toxicity or infectious diseases. Toxins causing hemorrhage include anticoagulant rodenticides, amanitin-containing mushrooms, vernal grass, or other plant toxins; however, viral infections of deer were considered most likely, including adenovirus hemorrhagic disease virus (AHDV) infection and malignant catarrhal fever (MCF), as well as bluetongue virus (BTV) and epizootic hemorrhagic disease virus (EHDV) infections. Bacterial infection causing septicemia was also considered, including but not limited to infection due to *Salmonella* spp., *Histophilus somni*, and *Yersinia pseudotuberculosis.*

FURTHER INVESTIGATIONS

The most significant microscopic finding was multisystemic lymphohistiocytic vasculitis in the lungs (3/3), heart (3/3), kidneys (2/3), urinary bladder (2/3), tongue (2/3), gastrointestinal tract (2/3), brain (1/3), liver (1/3), skeletal muscle (1/3), and a large artery (1/3). In affected organs, perivascular aggregates of many lymphocytes, often with large nuclei, and small numbers of macrophages frequently sur-

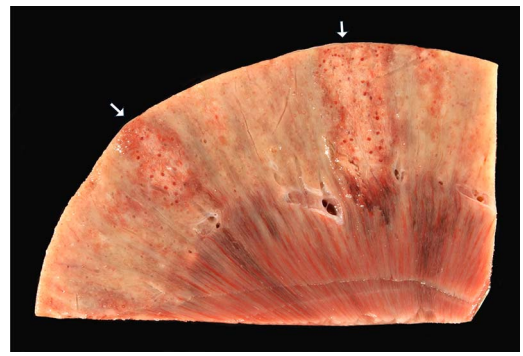

FIGURE 1. Multifocal renal infarcts (arrows) in the kidney of a mule deer (*Odocoileus hemionus*). Fixed tissue treated with ethanol to retrieve color.

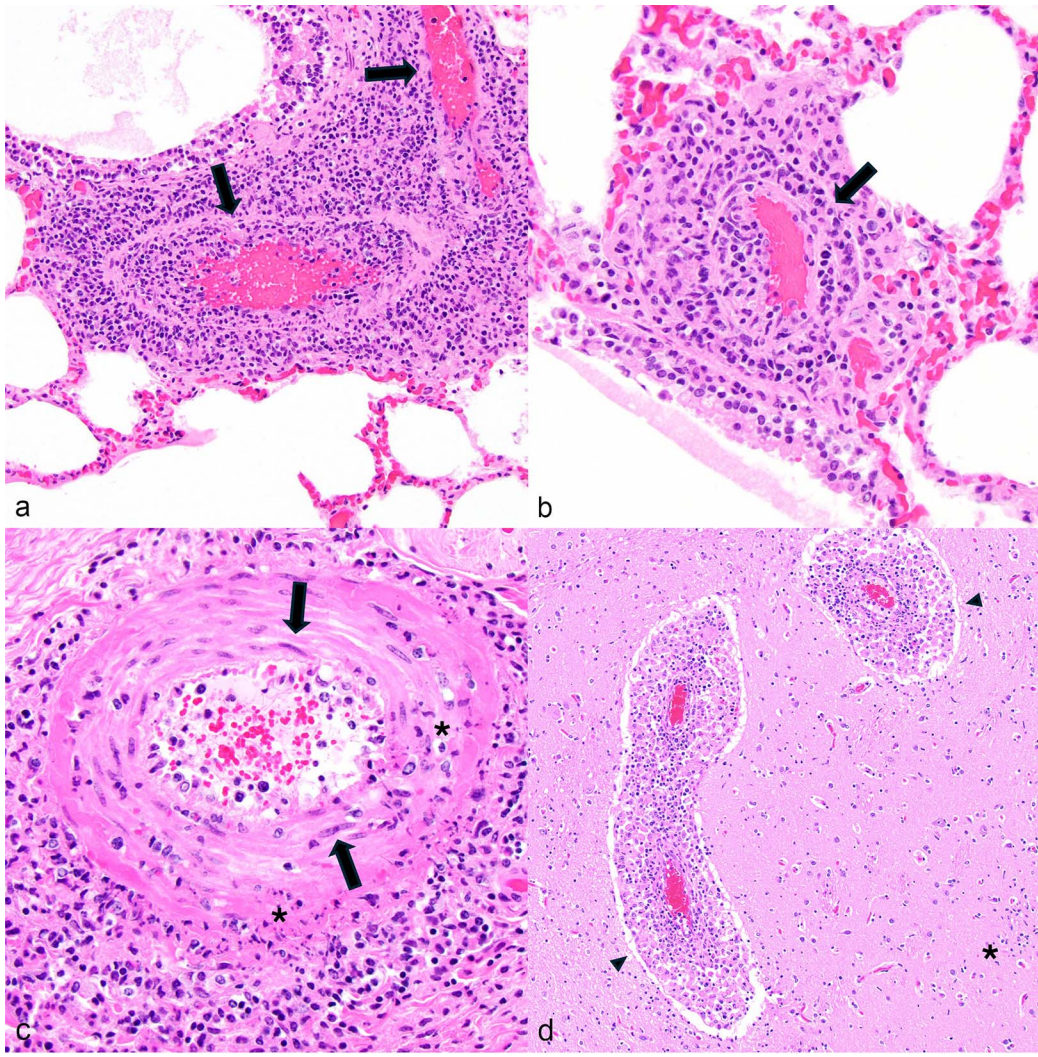

FIGURE 2. Histologic lesions of lymphohistiocytic vasculitis in mule deer (*Odocoileus hemionus*). (a, b) Lung, mule deer. Perivascular aggregates of macrophages and lymphocytes extend into and efface the adventitia, media, and intima (arrows). H&E. (c) Lung, mule deer. Hypertrophy of endothelial cells (arrows) and necrosis of smooth muscle cells (asterisks). H&E. (d) Brain, mule deer. Large lymphohistiocytic cuffs expand Virchow-Robin spaces (arrowheads), with associated gliosis (asterisk). H&E.

rounded small and medium-sized blood vessels, often extending into and expanding and effacing the adventitia, media, and intima (Fig. 2a, b) with hypertrophy of endothelial cells and necrosis of smooth muscle cells (Fig. 2c). Vasculitis in the brain was associated with meningoencephalitis with large lymphohistiocytic cuffs in Virchow-Robin space and neuronal necrosis and gliosis (Fig. 2d). Vasculitis was also associated with necrotizing myocarditis, renal infarcts and tubular proteinosis, ulcerative to necrotizing cystitis, necrotizing hepatitis, peritonitis, pulmonary edema, erosive rumenitis, and ulcerative to necrotizing glossitis, in the respective organs. Additional findings included sarcocystosis in heart and skeletal muscle (2/3) and serous atrophy of fat (1/3).

Aerobic culture of liver, spleen, and kidney (pooled) was performed in two cases. One grew moderate numbers of *Bibersteinia trehalosi* (a Gram-negative pathogenic bacterium) plus

mixed bacterial growth, suggestive of post-mortem overgrowth or contamination. The second case similarly resulted in moderate mixed bacterial growth, but without *B. trehalosi*. Fecal flotation was negative for parasites.

Further tests included PCR on fresh lung, plus on formalin-fixed paraffin-embedded lung, liver, and spleen, for ovine herpesvirus-2 (OvHV-2), adenoviruses, bluetongue virus, and epizootic hemorrhagic disease virus. In all three deer, OvHV-2 was detected in lung. Adenoviruses, BTV, and EHDV were not detected. Virus isolation was unsuccessful.

ANSWER AND ACTIONS

Based on the reported gross lesions and histopathology, MCF was considered to be the most likely diagnosis. Due to transmission by vector-competent *Culicoides* spp., BTV and EHDV, both orbiviruses, occur most commonly during late summer and early fall when the weather is warm and dry and vectors are plentiful (Savini et al. 2011). These cases occurred during February, thus BT and EHD were considered less likely. Infections with AHDV, BTV, or EHDV involve viral infection of endothelial cells, resulting in similar gross and histologic lesions: widespread hemorrhage, necrotizing vasculitis, thrombosis, ischemic necrosis, and edema, plus ulceration of the alimentary tract resulting from endothelial cell necrosis (Howerth et al. 2001). While BT and EHD typically induce microvascular disease (Maclachlan et al. 2009), AHD tends to affect large and medium-sized vessels, and in cases of systemic AHD, adenoviral intranuclear inclusion bodies are frequently detected within endothelial cells (Woods et al. 2018).

Malignant catarrhal fever can cause gross and histologic lesions similar to those seen in AHDV, BT, and EHD, but MCF involves viral infection of lymphocytes, resulting in immune dysregulation, lymphoproliferation, and, importantly, lymphocyte-mediated vasculitic damage of epithelium and blood vessels (Russell et al. 2009), which is histologically unique. Diagnosis of MCF was based initially on robust, widespread mononuclear vasculitis with the presence of lymphocytes with large nuclei, multi-organ necrosis, and epithelial ulceration, characteristic of the disease (O'Toole and Li 2014). It was confirmed by PCR detection of OvHV-2 in formalin-fixed lung tissue.

The Smith Valley region of Nevada, where the outbreak occurred, consists of residential plots situated between mountain ranges (public land) and agricultural fields. Domestic sheep (*Ovis aries*) are kept on agricultural fields in the valley during the winter and moved to private holdings or public land allotments for the rest of the year. Mule deer acquire MCF infection from domestic sheep, the major reservoir host of OvHV-2 in North America (Li et al. 1995; Russell et al. 2009). This outbreak corresponded with grazing of domestic sheep on fields in the valley during the winter months on mule deer winter range, and mortalities subsided after sheep were moved from the valley.

These cases represented the first report of MCF in mule deer in this region. In February 2022, one additional mule deer was found dead in the Smith Valley by the NDOW; histopathologic findings in this case, although severely autolyzed, similarly included pulmonary perivasculitis and cystitis, and PCR on formalin-fixed lung detected OvHV-2, again confirming MCF.

Although infrequently reported, fatal OvHV-2-associated MCF does occur in free-ranging mule deer with overlapping range and consequent exposure to domestic sheep, particularly where sheep management during the winter months increases the possibility of transmission of OvHV-2 to mule deer (Schultheiss et al. 2007). Death and illness of unknown cause in wild cervids known to be in proximity to sheep should prompt consideration for MCF and testing for OvHV-2. Transmission typically does not occur between deer (Reid et al. 1979; Cripps et al. 2019), thus outbreaks can be controlled by separating deer from sheep.

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