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SHORT COMMUNICATIONS

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Leukoencephalomalacia in a White-tailed Deer from North Carolina

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ABSTRACT: Leukoencephalomalacia similar to equine leukoencephalomalacia occurred in a white-tailed deer (*Odocoileus virginianus*) from North Carolina. The deer was recumbent and stuporous when presented for examination. It was believed that the disease resulted from ingestion of moldy field corn, infected with *Fusarium moniliforme*.

Key words: White-tailed deer, *Odocoileus virginianus*, leukoencephalomalacia, *Fusarium moniliforme*, mycotoxin, nervous system disease, case history.

In early March 1985, a wildlife biologist was called to investigate an adult male white-tailed deer (*Odocoileus virginianus*) which was acting abnormally in Stony Fork Community, Wilkes County, North Carolina (USA; 36°12'N, 81°29'W). The deer was unafraid of humans and exhibited incoordination and constant twitching of the head and neck. The animal was captured and submitted to the Southeastern Cooperative Wildlife Disease Study (College of Veterinary Medicine, The University of Georgia, Athens, Georgia 30602, USA) for examination. On arrival, the deer was in sternal recumbency and stuporous, and there was trembling of the head. Blood was collected and the deer was euthanitized (T-61® Euthanasia Solution, Hoechst-Roussel Agri-Vet Company, Somerville, New Jersey 08876, USA).

At necropsy, the deer was estimated to be 2.5-yr-old based on tooth replacement and wear (Severinghaus, 1949). The animal weighed 54.5 kg and was in poor condition, with minimal body fat. The rumen was filled with green plant material and a small amount of corn. Gross lesions were confined to the brain and consisted of severe edema characterized by swelling and flattening of the gyri, narrowing of sulci and herniation of the cerebellum through the foramen magnum. Multiple petechial and ecchymotic hemorrhages were present in the white matter of the brain. These hemorrhages were bilateral but were most severe in the subcortical white matter of the two most dorsal cerebral gyri. A few hemorrhages were present in the midbrain. Hemorrhages were most numerous on the left side, especially at the level of the internal capsule. Malacia and cavitation accompanied the hemorrhages in the subcortical white matter and were most severe on the left side at the level of the internal capsule (Fig. 1).

Hematologic values (red blood cell count $9.6 \times 10^6/\mu l$; white blood cell count 3,000/μl with 78% neutrophils, 6% bands, 13% lymphocytes and 3% monocytes; packed cell volume 40%; total plasma protein 7.1 g/dl; and hemoglobin 16.2 g/dl) were within the normal ranges (Seal et al., 1981). A reagent strip (Multistix®, Ames Company, Box 70, Elkhart, Indiana 46515, USA) revealed no significant alterations in the urine. Aerobic culture of the brain yielded *Escherichia coli* and *Streptococcus salivarius*; these organisms were considered contaminants.

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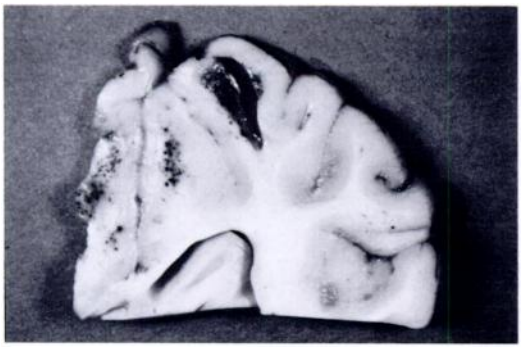


FIGURE 1. Cerebrum of white-tailed deer at level of internal capsule. Notice multiple hemorrhages, malacia and cavitation in subcortical white matter.

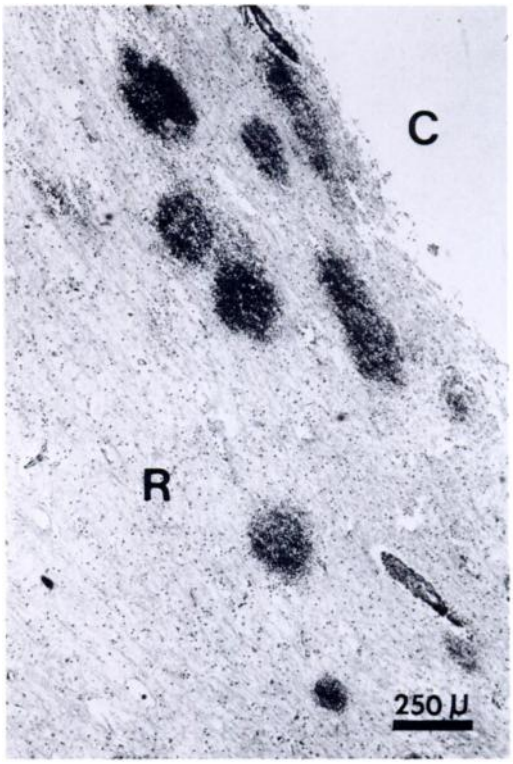


FIGURE 2. Cerebrum of white-tailed deer with cavitation (C), parenchymal and perivascular hemorrhages, and rarefaction (R) in the subcortical white matter. H&E.

Histopathological examination of the cerebrum revealed rarefaction of the subcortical white matter, with multiple parenchymal and perivascular hemorrhages. In some rarefied areas marked disintegration and disruption of the neuropil resulted in large cavitations (Fig. 2). Rarefied white matter gradually changed to more normal white matter with mild gliosis. Diffuse areas with perivascular edema, satellitosis, neuronal necrosis and microcavitation were present in the overlying cortical gray matter. Endothelial hypertrophy and hyperplasia were present in these areas. A few blood vessels contained minimal numbers of eosinophils and mononuclear cells in their walls and in the perivascular space. Additional findings were centrilobular hepatic fatty change characterized by large lipid vacuoles in hepatocytes, and sinusoidal histiocytosis in lymph nodes.

A field inspection of the area in which the deer was found was conducted within 1 wk of necropsy. Local residents were unaware of anyone feeding grain to wildlife, and grain being fed to cattle and horses was fed in barns that would not normally be utilized by deer. There were no other reports of sick or dead animals in the area. Most of the fresh corn had been harvested during early winter, but three small fields of corn stubble containing some grain were located. Samples of corn from each of these sites were collected and examined for total mold spores, types of molds present and specific mycotoxin contamination.

All ears of corn had evidence of insect damage and were heavily infected with fungi (Fig. 3). Six ears of corn were shelled and examined separately. The total mold spore counts were determined by standard methodology (Jarvis, 1978) using 0.005% Triton X-100 in 0.85% saline as the diluent and Sabouraud dextrose agar containing 40 μg chloramphenicol/ml as the culture medium. The total mold spore counts on these samples were high and ranged from 3.4×10^4 to 1.4×10^6 spores/g. Approximately 60% of the total molds isolated was *Fusarium* spp., including *F. moniliforme*, *F. graminearum* and an unidentified *Fusarium* sp. (Booth, 1971). Aflatoxin, zearalenone, T-2 toxin and deoxynivalenol concentrations were determined by thin layer chromatography in all samples of

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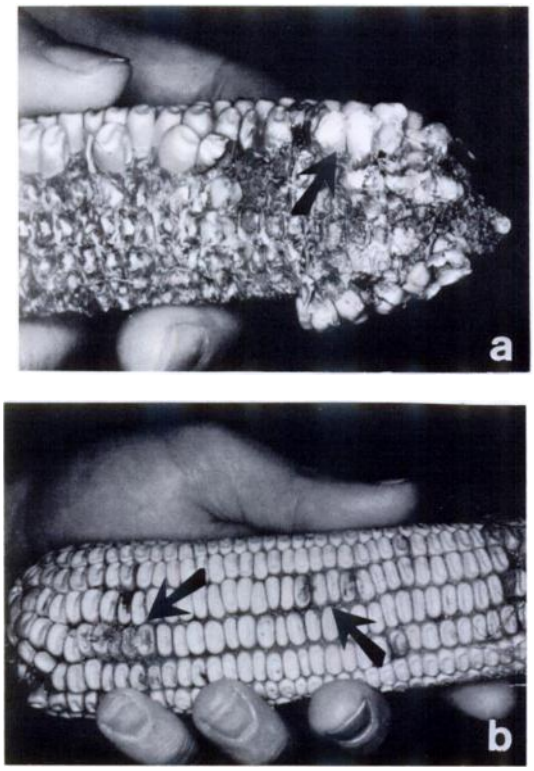


FIGURE 3. Ears of field corn; notice (a) evidence of insect damage (arrow), (b) moldy kernels (arrows).

corn (Scott et al., 1970; Pons et al., 1973; Vesonder et al., 1973; Eppley, 1975). Zearalenone levels were high in three cases (1,800 ppb, 3,600 ppb and 7,000 ppb). One ear, found to be contaminated with zearalenone, also had 60 to 80 ppb aflatoxin, but all other ears were negative for aflatoxins. Analysis for T-2 toxin and deoxynivalenol were negative.

The clinical, gross and histopathological findings in this deer were similar to those which have been reported in leukoencephalomalacia of horses and other Equidae (Buck et al., 1979; Haliburton et al., 1979). Equine encephalomalacia, also known as moldy corn disease, is seasonal in the United States, occurring from late fall through early spring (Buck et al., 1979). The natural disease is most often characterized by an acute and severe central nervous system disorder (Buck et al., 1979; Haliburton et al., 1979), but a hepatic syndrome manifested by icterus, systemic hemorrhage and edema has been reported in experimental trials (Kellerman et al., 1972; Marasas et al., 1976). The pathognomonic gross lesion of equine encephalomalacia is liquefactive necrosis in the subcortical white matter of the cerebral hemispheres (Buck et al., 1979; Haliburton et al., 1979; Marasas and Nelson, 1987). The pathogenesis of this lesion is unknown.

Equine leukoencephalomalacia is associated with the feeding of corn or commercial rations infected with the fungus, *Fusarium moniliforme* (Buck et al., 1979; Haliburton et al., 1979; Wilson et al., 1985). Insect damage, as seen in this case, is thought to enhance the growth of *F. moniliforme* on corn ears (Pienaar et al., 1981). The causative mycotoxin(s) has not been identified (Marasas and Nelson, 1987). A strictly comparable condition has not been authenticated in any species other than Equidae. Attempts to reproduce the characteristic brain lesion in other species, including sheep, goats, baboons, pigs, rats and monkeys, have been unsuccessful (Wilson et al., 1973; Kriek et al., 1981).

We postulate that leukoencephalomalacia in this deer was associated with the ingestion of moldy corn. Corn was present in the rumen of the deer, and field investigation revealed unharvested corn in a nearby field that contained *F. moniliforme*, the causative agent of equine leukoencephalomalacia. The corn was visibly moldy and *Fusarium* spp. were the predominant molds proliferating on the corn. Unfortunately, an adequate amount of corn was not collected to conduct a feeding trial to prove that the corn was toxic and could produce disease.

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