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Aspergillosis and Renal Oxalosis in a White-tailed Deer

Case History

An adult wild male white-tailed deer (*Odocoileus virginianus*) was submitted to the Department of Animal Diseases on January 2, 1970 because of severe skin lacerations of the left rear leg and rump. Other than exhaustion there were no other clinical problems and any previous history was unknown.

The skin wounds were sutured and one intramuscular injection of penicillin was given. The deer was transferred to a

holding facility and kept with two other deer. A diet of grain concentrate, timothy hay, maple and ash branches, and free choice 1:10 phenothiazine-salt mixture was fed.

Clinical improvement was rapid and within a week the deer appeared normal. Two days before death signs of depression, anorexia, and diarrhea appeared. Death occurred ten days after arrival at the laboratory.

Materials and Methods

A complete necropsy was performed and representative tissues were fixed in 10% buffered formalin, sectioned at 6 μ , and stained with the hematoxylin and eosin, periodic-acid-Schiff, and Grocott methods.

Kidney sections were subjected to three methods (Turner, J. Lab. Clin. Med., 24: 1096-1108, 1939; Johnson, Arch. Pathol.,

74: 347-351, 1962; Pizzolato, J. Histochem. Cytochem., 12: 333-335, 1964) for the histochemical identification of the renal tubular crystals.

Focal lung lesions were streaked on blood agar plates and incubated at 37 C. A wet mount of the aerial mycelium which developed was prepared using lactophenol cotton blue stain.

Results

Necropsy Findings. Gross examination of the lungs revealed miliary 3 to 5 mm pale firm nodules, many of which had surrounding zones of hemorrhage or congestion.

The significant microscopic lesions were limited to the lungs, brain, and kidneys. In the lungs there were disseminated focal necrotizing granulomas which contained dichotomous branching septate hyphae, neutrophils, and pyknotic nuclear debris (Fig. 1). Within the lesions complete necrosis of alveolar walls occurred. There was alveolar hemorrhage, congestion, and edema in the adjacent lung tissue (Fig. 2). In some areas hyphae were penetrating the pleura from contiguous necrotic foci resulting in small raised colonies of branching hyphae.

Involvement of the central nervous system was principally hyphae within blood vessels (Fig. 3) and the adjacent

brain. Many were seen in the meninges underlying the basal ganglia. These hyphae were located free in the meningeal spaces and particularly in perivascular areas accompanied by infiltrates of neutrophils, eosinophils, and macrophages. Many vessels in the gray matter contained hyphae with smaller blood vessels showing only acute inflammatory cellular infiltrates and necrosis of the vascular walls. Hyphae with no inflammatory reaction were common in the adjacent brain parenchyma. The cerebral cortical gray matter had similar morphologic findings except that vascular necrosis and cellular infiltrates were more severe. The spinal cord was not examined.

The septate hyphae with dichotomous branching found in the lung and brain were morphologically consistent with *Aspergillus* species.

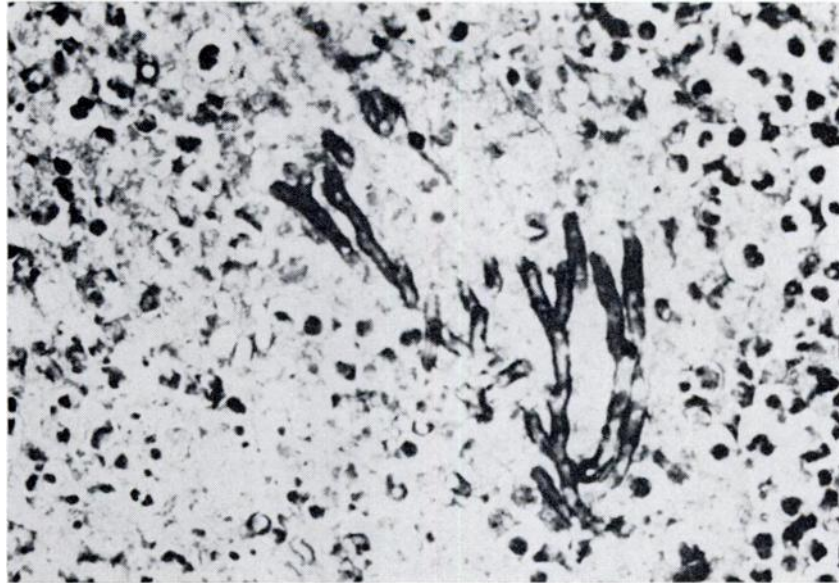


FIGURE 1. *Septate branching hyphae surrounded by necrotic inflammatory cells in the center of a pulmonary granuloma. H & E X 480.*

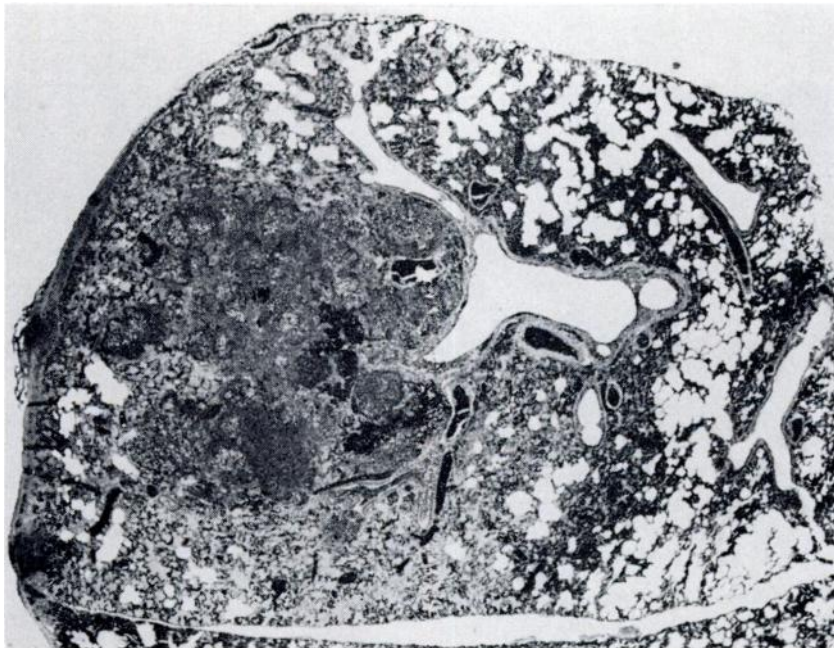


FIGURE 2. *Focal necrotizing granuloma and adjacent pulmonary edema and congestion. H & E X 12.*

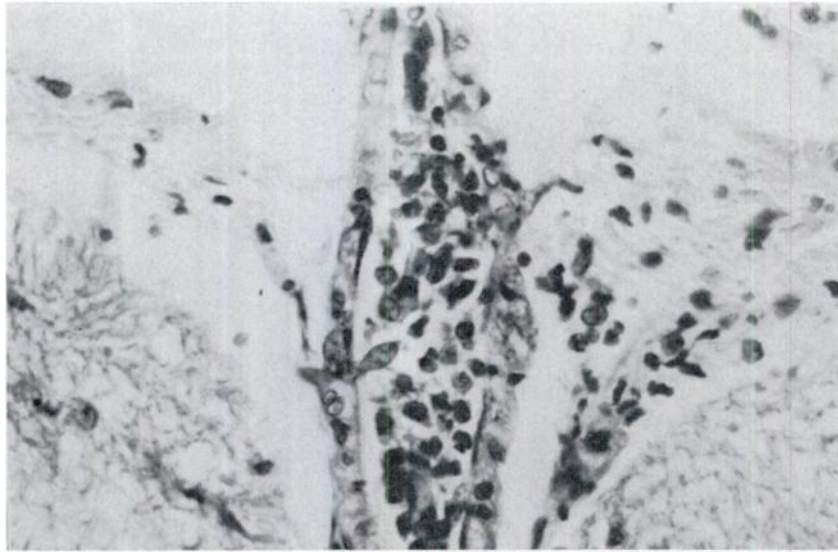


FIGURE 3. Note fungal hyphae penetrating the wall of the left side of a meningeal vein. Fragments of hyphae are visible in the lumen. *H & E X 480.*

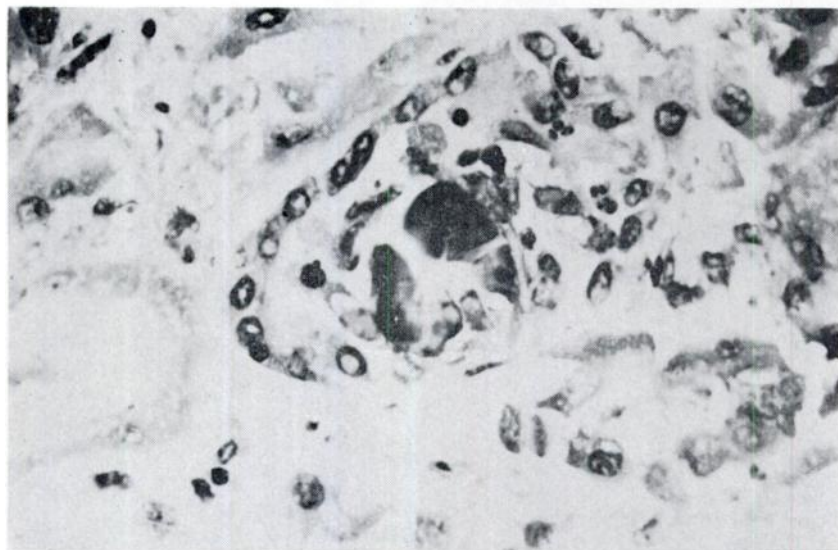


FIGURE 4. Rosette-shaped calcium oxalate crystal in a renal convoluted tubule. *H & E X 480.*

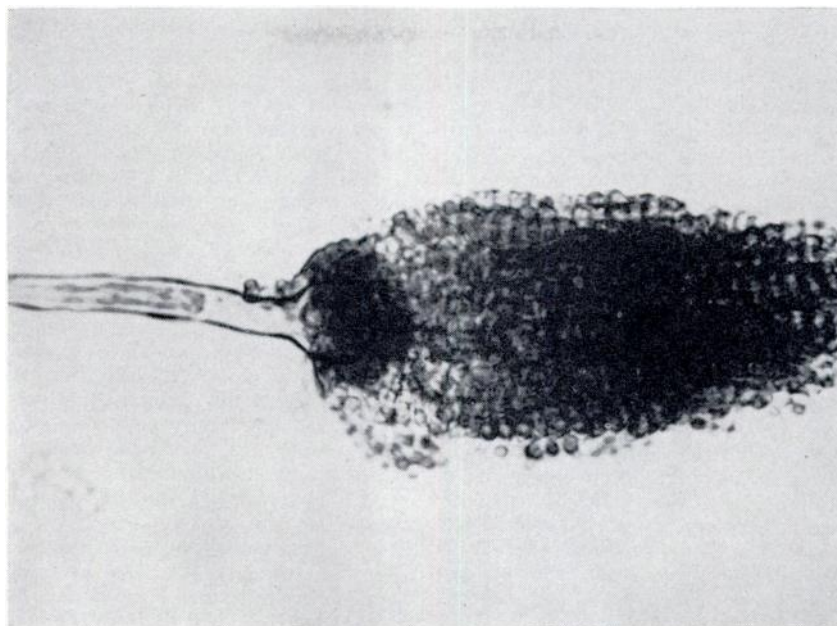


FIGURE 5. Mature conidial head of *Aspergillus fumigatus*. Note the parallel rows of conidia which arise from a single row of sterigmata which are on the terminal portion of a flask-shaped vesicle. Lactophenol cotton blue X 480.

Renal damage was extensive and was related to large numbers of predominantly rosette-shaped crystals in the convoluted tubules (Fig. 4). The crystals were strongly birefringent when viewed with polarized light and stained lightly basophilic with hematoxylin and eosin. Marked tubular changes consisting of hydropic degeneration, coagulation necrosis, and loss of epithelial cells were associated with presence of the crystals. Many necrotic tubules contained eosinophilic casts with aggregates of neutrophils. Tubular dilatation was widespread and pronounced.

The histochemical methods of Turner,

Johnson, and Pizzolato indicated the renal crystals were calcium oxalate.

White colonies of aerial hyphae which changed to bluish-green after several days were isolated in pure culture on blood agar plates which had been inoculated with lung lesion material. Wet mounts of the mycelium prepared in lactophenol cotton blue demonstrated mature conidial heads (Fig. 5) with parallel chains of conidia extending from a single row of sterigmata, characteristic of *Aspergillus fumigatus* (Merchant and Packer, *Veterinary Bacteriology and Virology*, 7th ed., Iowa State University Press, Ames, Iowa, 1967).

Discussion

Reports of mycotic infections in wild animals are infrequent. Ainsworth and Austwick (*Fungal Diseases of Animals*, Review Series No. 6, Commonwealth Agricultural Bureaux, England, 1959), Pier (Proc. 1st National White-Tailed

Deer Disease Symposium, University of Georgia, Athens, pp. 151-159, 1962), and Fletch and Anderson (Bull. Wildlife Disease Assoc., 5: 12-15, 1969) have presented comprehensive literature reviews. The purpose of the present report

is to document an additional case of *Aspergillus fumigatus* infection and to report for the first time renal oxalosis in a white-tailed deer.

It is accepted that systemic aspergillosis usually begins by inhaling large numbers of spores to establish a pulmonary infection from which hematogenous spread occurs. The recent case seems to support this pathogenesis since the hyphal emboli in the cerebral venules are a much less advanced lesion than the pulmonary granulomas. Although the pulmonary nodules were diffusely distributed throughout the lungs the lesions observed did not appear extensive enough to be the cause of death.

Renal damage was severe enough to cause death from kidney failure. Although histochemical tests, morphology, and birefringence to polarized light established the tubular crystals as calcium oxalate, it could not be determined whether the oxalosis resulted from ingestion of oxalate-containing plants or ethylene glycol (antifreeze) intoxication.

In carnivores the presence of birefringent calcium oxalate crystals in convoluted tubules is pathognomonic of ethylene glycol poisoning (Kersting and Nielsen, J. Am. Vet. Med. Assoc., 146: 113-118, 1965). We could not find published papers describing the effects of ethylene glycol in the ruminant, thus we do not know if it was possible for the deer to have ingested ethylene glycol before arrival at the University and then not show clinical signs of illness until eight or more days later. There did not seem to be any possibility for ingestion of ethylene glycol while at the holding facility.

An alternative possibility that the renal oxalosis was due to ingestion of plants containing large amounts of oxalates is also difficult to justify. Since the oxalate content of plants is highest at the leafy growth stage (Stewart and McCallum, Vet. Record, 56: 77-78, 1944), the occurrence of the illness in January is more compatible with ethylene glycol consumption than eating plants high in oxalates. The timothy hay was of good quality and

was not likely a significant source of potential oxalate bearing weeds.

A third but less likely pathogenesis for the renal oxalosis could be imagined as similar to the rare primary disturbance of oxalate metabolism in man. Calcium oxalate is deposited in the kidneys and other tissues with eventual death from renal failure (Anderson, *Pathology*, 4th ed., C. V. Mosby, St. Louis, Missouri, 1961).

It has been shown that oxalates may be formed by various species of fungi in moldy hay (Wilson and Wilson, Am. J. Vet. Res., 91: 961-969, 1961). Formation of oxalates by this method could not be ruled out, however, examination of the hay revealed it was of high quality with no observable mold growth.

The effects of either ethylene glycol or plants containing toxic amounts of oxalate are unknown in deer. Cattle are nearly resistant to poisoning by calcium oxalate due to rumen degradation of the compound (Jubb and Kennedy, *Pathology of Domestic Animals*, Vol. 2, p. 258, Academic Press, New York, 1963). Sheep are not easily poisoned, being able to tolerate up to 750 gm dryweight of a high oxalate containing plant per day (Dodson, Australian Vet. J., 35: 225-233, 1959). A small percentage of sheep fail to detoxify all of the oxalate by the rumen and develop renal lesions.

Regardless of the pathogenesis of the renal oxalosis we feel that renal failure was the primary problem and that the *Aspergillus fumigatus* acted as an opportunist and established a mycotic infection in a debilitated subject.

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