Case Report —

Pulmonary oxalosis in association with
Aspergillus niger infection
in a Great Horned Owl (Bubo virginianus)

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INTRODUCTION

Aspergillosis is a common disease of avian species, with Aspergillus fumigatus the pathogen isolated by far the most commonly (1). Infection with other species of Aspergillus or with other mycotic agents except Candida sp. has been reported infrequently in birds. Pulmonary oxalosis caused by A. niger infection has been reported in one series of human cases (4) but is not a widely recognized condition.

HISTORY

A mature female Great Horned Owl (Bubo virginianus) was submitted for necropsy from a local animal collection. The bird had been observed to be unable to fly in the wild approximately 2 weeks earlier, and consequently had been donated to the collection. It had appeared healthy in other respects and had fed well in captivity, but was found dead unexpectedly.

NECROPSY AND LABORATORY FINDINGS

On necropsy the bird was in excellent body condition, with abundant subcutaneous and intra-abdominal fat. Several sharply demarcated pale foci, 1-2 cm in diameter, were evident in the super-

Fig. 1. Lung of owl showing abundant oxalate crystals surrounding bronchus filled with septate fungal hyphae. H&E, polarized light, ×85.

Figs. 2 & 3. Oxalate crystals lying in layer of necrotic debris beneath hyphal growth. Fig. 2, H&E, polarized light, ×145; Fig. 3, PAS, polarized light, ×225.
Aspergillus niger in a Great Horned owl

A puncture wound 2 mm in diameter was found in the skin at the left proximal humeral articulation, and a necrotizing tract extended caudally from this point between the sternum and the deep pectoral muscle on the left side. This lesion resembled that caused by a shotgun pellet, and was likely responsible for the bird's inability to fly when captured in the wild.

The thoracic and abdominal air sacs were thickened by caseous material covered with a black mat of sporulating heads of fungi. The lungs were swollen and consolidated, with hemorrhage and necrotic foci evident. The major bronchi were filled with black fungal spores. Other organs were unremarkable.

Portions of pectoral muscle, lung, liver, spleen, kidney, heart, and brain were fixed in 10% buffered formalin, and processed routinely, and sections cut at 6 μ were stained with hematoxylin and eosin (H&E). Also used were Grocott's stain and the periodic acid-Schiff (PAS) reaction with light-green counterstain for fungi (3), and the Pizzolato (3) and Roscher techniques (6) for demonstration of calcium oxalate.

Portions of lung were cultured on Sabouraud's agar at room temperature.

Histopathologically, the lung lesions consisted of a hemorrhagic necrotizing pneumonia with massive aerial mycelial growth within the airways. Many heterophils were present in some areas of the lung, as were occasional giant cells. The areas of mycelial growth were surrounded by a rim of necrotic debris within which were numerous large crystals. These crystals were distinctly birefringent in polarized light (Figs. 1, 2, 3) and were found only in association with areas of growth of septate, acutely branching fungal hyphae of the type produced by aspergilli, or in the walls of vessels immediately adjacent to these areas (Fig. 4). The crystals were identified as calcium oxalate on the basis of their appearance in polarized light and by histochemical reactions in the Pizzolato and Roscher stains.

Found in areas of the lung distant from the growth of aspergill
gilli were larger (up to 7 μ in diameter) nonseptate irregular hyphae with abundant perpendicular branches. These hyphae, which resembled those of the class Phycomycetes (1), were invading vessels and causing thrombosis (Fig. 5). The pale areas seen grossly in the pectoral muscles were foci of mycotic myositis with marked necrosis, edema, and heterophil infiltration. Fungal hyphae similar to those present in pulmonary vessels were evident in these areas (Fig. 6). Oxalate crystals were not found in association with the large nonseptate hyphae in either the lung or muscle tissue.

Culture of lung yielded a very heavy growth of *A. niger*, plus a few colonies of a fungus identified as *Scopulariopsis* sp. No Phycomycetes were isolated.

**DISCUSSION**

This appears to be the second report of oxalosis in association with *A. niger* infection. As mentioned previously, infection with this species has been reported rarely, although species of the *A. niger* group are “one of the commonest mold groups found in the environment of man and animals” (5). Hare (2) described caseating pneumonia in pigeons due to *A. niger*, but did not report any crystals in the lesions. Raper and Fennel (5) stated that under certain *in vitro* conditions some strains of *A. niger* can produce appreciable quantities of oxalic acid. Nime and Hutchins (4) suggest that oxalic acid production in tissue may be characteristic of infection with *A. niger*, and that the tissue necrosis adjacent to mycelial growth may be due to the toxic action of this acid. Those suppositions are supported by the close association of oxalate crystals with both mycelial growth and necrosis in the present case.

**REFERENCES**


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