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EPORNITICS OF ASPERGILLOSIS IN MALLARDS *(Anas platyrhynchos)* IN NORTH CENTRAL COLORADO

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INTRODUCTION

Aspergillosis is a well-documented fungal disease of wild and domestic avian species.[6] Outbreaks in free-ranging waterfowl have been recorded in wood ducks *(Aix sponsa)*,[1] mallards *(Anas platyrhynchos)*[2,4] and Canada geese *(Branta canadensis)*.[3] A more complete list of susceptible species is presented by O'Meara and Witter.[5] This paper describes the history, gross necropsy, histopathology and predisposing factors of two epornitics of acute aspergillosis in mallard ducks in north central Colorado.

HISTORY

The first epornitic occurred in October, 1975, the second in October, 1976. Both were restricted to single irrigation reservoirs. The 1975 outbreak occurred at Timnath Reservoir, located 113 km north of Denver, Colorado. In 1976, an outbreak occurred at Woods Lake, located 8 km east of Timnath Reservoir. Both lakes are 16-20 ha in area, have a rock and/or mud shoreline and mud bottom. Very little vegetation grows on the shoreline as the water level fluctuates to meet irrigation needs. Mallards, coots *(Fulica americana)*, and Canada geese were the predominant species on Timnath Reservoir, while mallards and Canada geese were the most common species on Woods Lake. Cornfields and open ensilage pits are common in the area around both lakes. Ducks and geese utilize the fields for feeding, and during inclement weather, ducks also may use the ensilage pits. The 1975 outbreak was preceded by a heavy, wet snow. Clear and warm weather prevailed during the 1976 outbreak.

The hunting season was not open during the 1975 outbreak at Timnath Reservoir, and although the season was open in the Central Flyway during the 1976 outbreak, Woods Lake was closed to hunting.

METHODS AND MATERIALS

Most mortalities were picked up from the shore of the lakes during the die-offs. A few that were floating, dead or moribund on the water were picked up by a boat or a Labrador retriever. A total of 270 mallards were picked up from 28 through 30 October 1975 from Timnath Reservoir, but due to predation and/or decay only 128 were suitable for necropsy. Ducks were picked up at Woods Lake from 10 through 14 October 1976; 117 birds were found and all were fresh enough to be examined at necropsy. Samples of moldy corn were picked up from the surrounding corn fields and ensilage pits.

During necropsy, heart blood was taken and cultured for pathogenic organisms. Lungs and air sacs from 10 birds were cultured on Sabouraud's agar for fungus. The gizzard of each bird was carefully examined for the presence of lead shot and the lower right lobe of the liver was saved and later dried and analyzed for lead content by atomic absorption spectroscopy. When the bird was fresh, appropriate tissues were taken and preserved in 10% buffered formalin, processed by the usual paraffin histologic procedure, sectioned at 6 μm and stained with hematoxylin and eosin. Sec-

tions of lung and air sacs also were stained with periodic-acid-Schiff (PAS) and Grocott's stains for fungus.

RESULTS

Mallards were the only species involved in the epornitics. The sex ratio in 1975 was 52% males to 48% females; in 1976 it was 60% males to 40% females. These ratios are typical for wintering mallards in this region. A total of 86 of the 1976 birds were aged; 60 (70%) were young-of-the-year.

Gross Necropsy

The great majority of the birds in both epornitics were in good-to-excellent body condition. A few were in poor condition, but none were emaciated. Corn was found in the crop, proventriculus, and/or ventriculus of 10%, but no food was found in the remainder. Aspergillus lesions were remarkably similar in all birds examined. The organs predominantly affected were the lungs and air sacs. The lungs were dark red, wet and contained many small, white miliary nodules disseminated throughout the lung parenchyma (Fig. 1). A slightly higher prevalence of white nodules were present around the mesobronchus of the abdominal air sac (Fig. 2) in the posterior ventral aspect of the lungs. Seventy percent of the birds had white-to-yellow plaques in the interclavical, anterior and posterior thoracic, and abdominal air sacs. These plaques varied in texture from cotton-like, with visible hyphae and fruiting bodies, to firm, flat, yellow plaques. The size and number of mycotic plaques on the air sacs varied from bird to bird, but all had the white nodular lung lesions. The birds with clear air sacs appeared to have more lung damage than birds with both lung and air sac involvement. Petechial or ecchymotic hemorrhages were found on the epicardium of most birds. The vessels of the ventriculus and intestinal tract were slightly engorged. A few of the birds had firm, raised, yellow mycotic plaques in the proventriculus and small intestine. The spleen was slightly to moderately enlarged. The kidneys, adrenals, thyroids, parathyroids, and gonads appeared normal on gross examination.

FIGURE 1. Acute type *Aspergillus* lesions in the lungs of a mallard.

Histopathology

Sections of brain, heart, lungs, air sacs, liver, intestinal tract, spleen and kidney were examined from two birds. Both birds had lesions grossly identical to those of all other birds.

The primary histologic lesions were in the lungs and air sacs. Three slightly different types of cellular reactions were seen around mycotic nodules in the lung parenchyma: acute, subacute and chronic. The acute-type nodule (Fig. 3) was the largest and was characterized by a central core of necrotic cellular debris with many branching septate mycelia. The predominant reaction around this acute nodule was macrophages and heterophils. The subacute-type nodule (Figs. 4 and 5) was similar to the acute type except for the addition of a few multinucleated giant cells together with macrophages and heterophils. A moderate number of branching septate mycelia were found in the central core of necrotic cellular debris of the subacute

FIGURE 2. Chronic type *Aspergillus* plaques in the air sacs of a mallard.

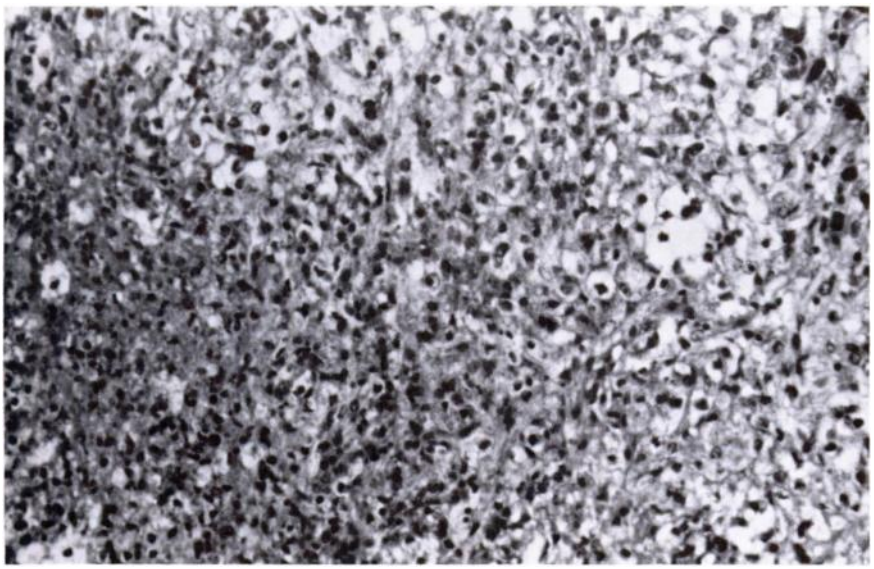

FIGURE 3. Acute mycotic foci within the lungs. Note the central core of necrosis with the reaction around the periphery composed predominantly of macrophages and heterophils. (H & E × 250).

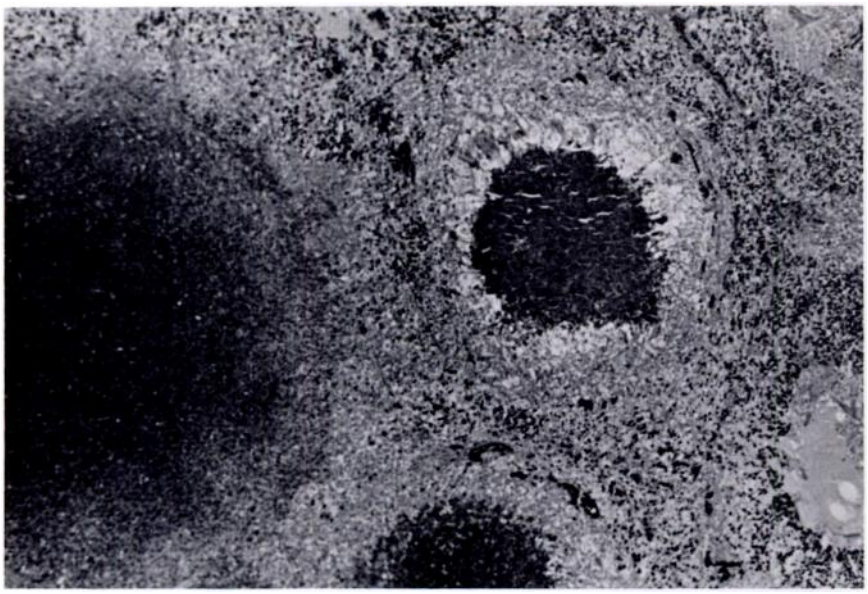

FIGURE 4. Subacute mycotic foci with a central core of necrotic debris surrounded by multinucleated giant cells with many macrophages and a few heterophils. (H & E × 160).

nodules. Chronic nodules (Fig. 6) were surrounded predominantly by multinucleated giant cells with a few macrophages and heterophils around the periphery. Few mycelia were found in these small, chronic, tubercle-like nodules. The remaining lung parenchyma was very congested and many parabronchi were filled with blood. The air sacs which contained mycotic plaques were greatly thickened. The lumenal surface of these plaques was composed of a thick layer of fibrinonecrotic debris. The surface was laced with many branching septate mycelia supporting a few fruiting bodies that contained numerous spores. The cellular response toward this mycotic fibrinonecrotic membrane (or plaque) consisted of multinucleated giant cells, macrophages, heterophils and a few lymphocytes and plasma cells. The lymphocytes and plasma cells appeared to be more numerous around vessels under the plaques.

Nodules from the esophageal-proventricular junction and the small intestine were histologically identical. They were composed of necrotic cellular debris laced with numerous branching septate hyphae. The lumenal surface of these plaques contained many branching septate mycelia, spores and a few fruiting bodies. The bases of these plaques were infiltrated predominantly with multi-nucleated giant cells, macrophages, and heterophils, with a few lymphocytes and plasma cells. The nodules involved the mucosa, lamina propria, submucosa and extended to the tunica muscularis. Inclusion bodies were not observed near the edges of the mycotic plaques found in the intestine and/or proventriculus. Liver sections had an increased number of lymphocytes and plasma cells in the connective tissue stroma of the hepatic portal triads. One bird had several small granulomas in the liver; however, sections of nematodes

were found in them. The spleen had a normal ratio of lymphocytes to reticuloendothelial cells and was not engorged with blood.

All ventriculi were free of lead shot in the Timnath (1975) die-off; one ventriculus contained a single lead pellet in the Woods Lake die-off. Liver tissue lead levels showed that only two birds (Woods Lake die-off) had high values, 42 and 71 ppm dry wt. One additional bird had 19 ppm and the remainder had less than 6 ppm dry-wt values. No pathogens were isolated from the blood. *Aspergillus* sp. was isolated from all twelve lung and/or air sac cultures. *Aspergillus* sp. was also isolated from cultures taken from ensilage pits near Timnath Reservoir (1975).

DISCUSSION

Both epornitics began suddenly and lasted less than 7 days, with most of the mortality occurring the first 3 days. Because mallards were the only species affected and since the die-off each year was restricted to a single lake, this indicates a common source of infection to a group of birds which were resting and feeding together. The snow in 1975 covered the available feed in the fields and the birds probably fed in a contaminated ensilage pit. However, there was no adverse weather before the 1976 outbreak and therefore cannot be considered a contributing factor. Only a low percentage of the affected birds were mature; this may reflect an increased resistance to *Aspergillus* or it may be due to the high percentage of young in the population, or both. Neither die-off showed any correlation to sex or lead poisoning.

Lungs and air sacs were the primary organs affected by *Aspergillus*. The slightly different cellular reactions described in the lungs were believed to be due to duration and amount of fungus deposited at one focus. The large acute and subacute nodules were the predominant type, thus indicating the duration of the disease was short.

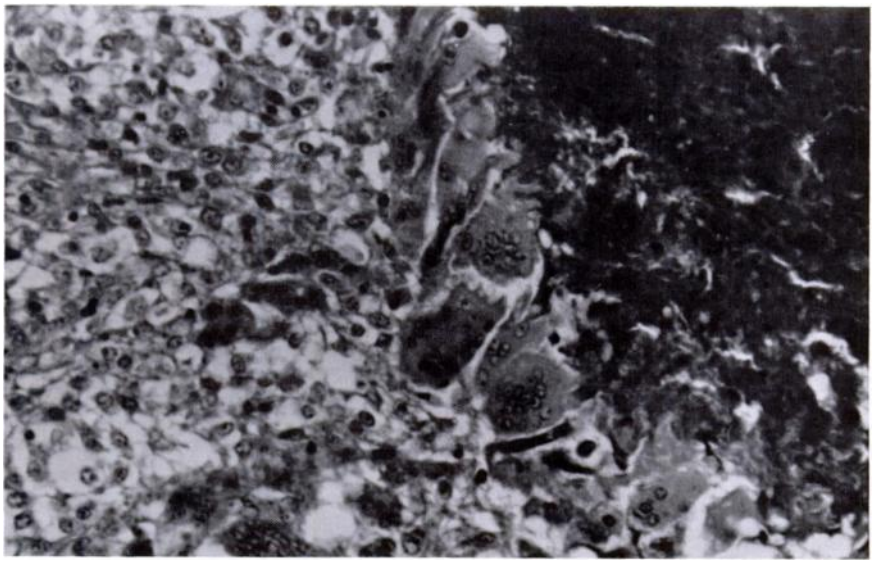

FIGURE 5. Subacute mycotic nodule. (H & E × 400).

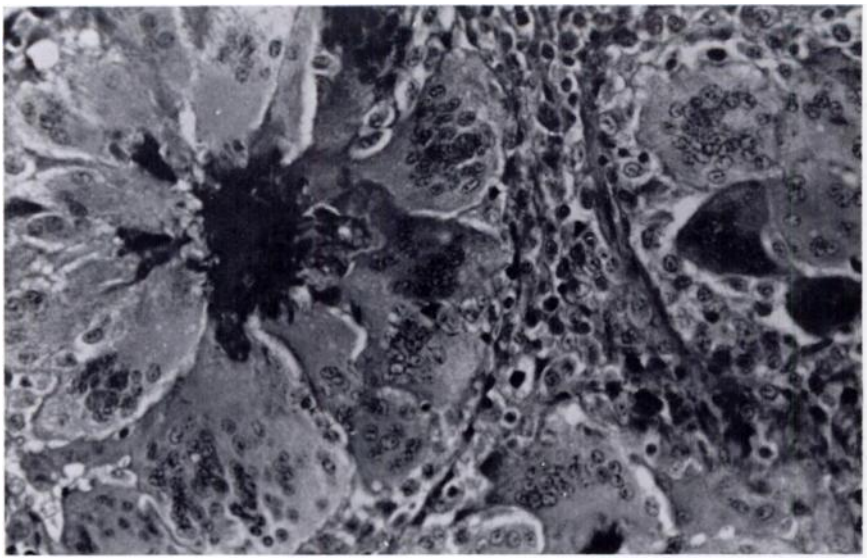

FIGURE 6. Chronic mycotic nodule with a small central core of necrotic debris with a zone of predominantly large multinucleated giant cells with a few macrophages on the periphery. (H & E × 400).

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LITERATURE CITED

1. BELLROSE, F.C., H.C. HANSON and P.P. BEAMER. 1945. Aspergillosis in wood ducks. J. Wildl. Manage. 9: 325-326.
2. HERMAN, C.M. 1943. An outbreak of mycotic pneumonia in mallards. Calif. Fish and Game 29: 204.
3. McDOUGLE, H.C. and R.W. VAUGHT. 1968. An epizootic of aspergillosis in Canada geese. J. Wildl. Manage. 32: 415-417.
4. NEFF, J.A. 1955. Outbreak of aspergillosis in mallards. J. Wildl. Manage. 19: 415-416.
5. O'MEARA, D.C. and J.F. WITTER. 1971. Aspergillosis. In: *Infectious and Parasitic Diseases of Wild Birds,* p. 153-162. Davis, J.W., R.C. Anderson, L. Karstad and D.O. Trainer, eds. Iowa State Univ. Press, Ames.
6. ROSEN, M.N. 1964. Aspergillosis in wild and domestic fowl. Avian Dis. 8: 1-6.

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