PATHOLOGY OF EXPERIMENTAL ASPERGILLOSIS IN PIGEONS

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Introduction

Aspergillosis is a disease of mammals and birds produced by the growth of various species of the fungus Aspergillus within the animal tissues.It is one of the earliest animal mycoses to be reported (Ainsworth & Austwick, 1973).

Avian Aspergillosis is primarily a respiratory infection but generalised infection was also seen. Many species of domesticated and captive birds were known to harbour the infection (Ainsworth & Austwick, 1973).

Hare (1937) described a chronic nodular consuming pneumonia and pleurisy in two pigeons where **A.niger** was isolated in pure culture from the lungs. An outbreak of mycotic pneumonia in pigeons caused by **A.fumigatus** was described by Taylor (1944) and the postmortem examination revealed no significant gross pathological lesions except for some congestion and consolidation of the lungs.

The authors encountered caseous and granulomatous lesions containing large number of septate fungal hyphae in an adult pigeon. These were diagnosed as Aspergillus hyphae, but isolation of the organism was not done as tissues were not available for culture. The finding of such a case prompted us to explore the role that Aspergillus can play in adult pigeons.

Materials and Methods

17 pigeons of the local breed of the Sudan were selected for this study; four of them were about 2 months old and the remaining were adults. The organism used as an infective agent in the study was isolated from an apparently healthy chicken's lung of the local breed "Baladi". Primary isolation was done by streaking parts of the lungs onto malt extract agar (Oxoid) slants with chloramphenicol (o.05 mg ml).

These were incubated at 26^0 C for two weeks. The isolated organism was identified as **A.fumigatus** according to the methods of Raper and Fennell, (1973), and was used in this study for experimental infection of the pigeons. To prepare the infective inoculum, spores were suspended nto sterile Pijou bottles containing normal saline. The spores were counted with a hemacytometer and the numbers in the saline were adjusted to $2.5x10^6$ spores/ml. The pigeons were divided into four groups. Four young birds (Group 1) and five of the adults (Groups2) were inoculated with 0.2 mi ($5x10^5$ spores) interavenously. Anthor five of the adults were inoculated with 0.8 ml ($2x10^6$ spores)(Group 3) and ne remaining 3 were left as uninoculated controls (Group 4).

Each of the 4 groups was kept in a separate clean and disinfected cage in a well—ventilated room. The brids were observed for clinical signs and temperature was recorded daily. One month post—inoculation 2 of theyoung brids (Group 1) were sacrificed. Two months post—inoculation the remaining 2 young pigeons (Group 1) and 2 of adult brids (group2) were sacrificed. Four months post—inoculation the last 2 of the adults (Group 2), all 5 of the adults (Group 3) and the controls (Group 4) were sacrificed.

Post—mortem examination was performed following the standard procedures of necropsy. Parts of the lungs, livers and brains were removed aseptically to re-

isolate the organism. Tissues taken for histopathological examination were fixed in 10% formaline, embeded in paraffin and sections were stained with Hematoxylin & eosin. These included lung, liver spleen, pancreas, heart, intestine, brain and bursa.

Results

No clinical abnormalities and no gross lesions were observed on necropsy of 2 of the young birds in group 1, all the birds of group 2 and the controls.

Two months post—inoculation the remaining 2 of the young brids in group 1 showed depression, respiratory distress and were emaciated. When they were killed, post—mortem examination revealed congestion of the liver and a haematoma—like lesion of a maize—grain size. The bursa showed interfolicular edema, depletion of lymphocyte and reticulo—endothelial cell proliferation.spleen there was lymphocyte depletion, reticuloendothelial cell proliferation, cystic degeneration and infilteration of macrophages and fibroblast. Numerous septate hyphae could be seen. The lungs showed focal mononuclear infilteration with few hyphae.

Abnormalities were also detected in 3 out of 5 adult birds that were inoculated with the higher doses (Group 3). The first bird was very emaciated. Post—mortem examination showed the liver to be brownish in colour. Histologically there was granulomatous hepatitis, focal lesions with necrotic centre surrounded by giant cells, macrophages, heterophils and lyphocytes.

Post—mortem examination of the second bird showed maizesize nodules in the liver which is congested. There were adhesions and nodules of various sizes in peritoneal cavity and visceral organs. The air—sacs were cloudy. Microscopically the blood vessels of the liver were dilated and congested. There was accumulation of macrophages and coagulative focal necrosis. In the kidney there was focal lesion surrounded by inflammatory cells: giant cells, macrophages, lymphocytes and heterophills with necrotic centre.

The third bird showed proliferation of lymphocytes and necrotic foci in the lung. In the liver blood vessels were congested and there was lymphocytic infilteration. No lesions were detected in the brain, heart, intestines and pancreas of all birds during this study. Temperature of all birds remained within the normal range throughout the experiment.

A. fumigatus was reisolated from 2 out of 4 young pigeons (Group 1) and 6 out of 10 adults (I from Group 2 & 5 from Group 3). It was isolated from the lungs and liver. No isolations were made from the brain.

Discussion

This experiment showed that adult pigeons were refractory to infection with **A. fumigatus** when inoculated with small doses. When the dose was increased 4 times as much for a longer period, hepatic and pulmonary aspergillosis was detected. Small does of the inoculum for a short period did not affect the young birds. When the duration of the experiment was doubled the birds showed clinical signs of respiratory insufficiency; the lungs, liver, spleen and bursa were the organs most affected.

These findings showed that the pigeons are susceptible to infection with **A. fumigatus.** This confirms what was demonstrated by Austwick & Ainsworth (1973): that the pigeon could be easily infected experimentally.

Pigeons in the Sudan are kept indoors only during the night and set free during the day—time. The birds may inhale the infective spores occasionally when kept

outdoors but their free time in the air increases chances for exposure and may lead to development of some immunity. This explains why some of the pigeons in this experiment remained refractory to infection.

Summary

Experimental inoculation of fumigatus showed the pigeons of the local breed in the Sudan to be susceptible to infection. The intravenous route lead to emaciation, hepatic and pulmonary aspergillosis. Gross and histopathological lesions were described. Reisolation of fumigatus was confirmed from the livers and lungs.

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