الالتهاب الرئيسي والنزيف القطني التجريبي في الديجالة الروسي

عبد النعيم، محمد، عبد النعيم، محمد، محمد، محمد، إبراهيم، إبراهيم، إبراهيم، إبراهيم، إبراهيم.

تم عزل مشيرة أزواج من الخفريات من مادة مأخوذة من 15 ككتوت، روسي، حديث الوفاة، وكذا خسون عينة من دجاج روسي، حي، والعينات قد أعثرت من محافظات البلاد الجديدة، وقنا، بجميد مصر.

استمر أحد هذه الأنواع وهو أسيم، وجيم، باليا، وقد كتبت، روسي، سبيبة، أعطرت، مين، محافظة، بالعا، ورشت النتائج المجهرية الميكروسكوبية، وتوفقت النتائج، وكذلك تم اختبار حساسية، هذا.

التفسير لمعدة نسبة من الأدلة والكفاءات المختارة ورشت النتائج وتوفقت.

* معمل بحوث صحية الحيوان - محافظة قنا
* قسم الميكروبيولوجيا - كلية الطب البيطري - المعبزة
* قسم طب الحيوان وأراض الدواجن - كلية الطب البيطري - أسوان
EXPERIMENTAL MYCOTIC PNEUMONIA AND ENCERHALOMALACIA IN TURKEY POULTS
(With 8 Figures & 3 Tables)

By
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SUMMARY

Ten fungal species could be isolated and identified from freshly dead and apparently healthy turkeys in upper Egypt. Three weeks old native breed trudey poults were inoculated into the left abdominal air sac, each with 0.5 ml. of A. flavus suspension. The symptoms as well as the macroscopic and microscopic appearance of the lesions suggest a pneuma-encephalomalacic disease. Reisolation of the inoculated fungus and its sensitivity to nine drugs and chemical agents were carried out.

INTRODUCTION

Mycotic affections have been reported from all domestic animals and birds in different parts of the world ROSEN, 1964, ALLER, 1967, and POLYA and BELEGH, 1971.

In Egypt, several studies concerning this problem among poultry were carried out, ABO-GABAL et al., 1974, REFAI, 1976, ABO-GABAL et al., 1977, and NARJAD, 1978.

Most of the brain lesions described in the literature regarding the mycotic affections are inflammatory in nature, RAINES et al., 1966, GEDKE, BIEBER and COOKE, 1964. Fibrinous air saculitis, pneumonia and typical mycotic granulomas were the main pulmonary lesions described in turkey poult mycosis, KUNDISON and MEINECKE, 1972, ZAHIAY et al., 1977.

The aim of the present work, was typing of fungal species present in upper Egypt, moreover to throw more light on the brain and lung lesions in experimentally infected poults, as well as the sensitivity of these strains against drugs and chemicals used in other parts of the world.

MATERIAL and METHODS

In the present work, 210 freshly dead and living turkeys of different ages and sources, (Table 1) from upper Egypt were studied. Crop and lung samples as well as tracheal swabs were subjected for mycological isolations. The isolates were cultured on modified Sabourauds agar and subcultured till the colonies appeared. Morphological and microscopical identification of the isolates was carried out.

A. flavus suspension was made by adding 10 ml. fresh distilled water containing one drop of leconal as a wetting to the culture, CHUTE and OMEARA, 1958.

Twenty, apparently healthy, three weeks old native breed turkey poults were obtained from Bani-Snaef Governmental turkey farm. Four poults were taken at random and subjected to post-mortem and mycological examinations. Eight poults were inoculated into the left abdominal air sac with 0.5 ml/ bird of the fungal suspension using 1 ml. syringe of 18 gaug needle. The other eight poults were kept as controls and each was inoculated by the same method with 0.5 ml. normal saline solution.

The infected poults were observed daily, during which symptoms and deaths were recorded. Post-mortem examinations were adopted on dead birds. Brain and lung specimens were taken for histopathological investigations. Trials for reisolation of the inoculated fungus were carried out. Moreover sensitivity of the fungus against nine drugs and chemical agents was tested.

RESULTS

In field cases, the most characteristic post-mortem lesions seen in the examined dead poults, brought for
mycological isolations were air saculitis, congestion of the lungs and ulcer like lesions in the crop. The species, number and percentage of the isolated fungal species from both the apparently healthy and dead turkey samples and awaps are illustrated in table II.

In experimentally infected cases symptoms observed on all inoculated poult's except the controls were more or less similar and included depression, ruffled feathers, off food, drooping of wings, transient diarrhoea and respiratory signs as gasping and rattling sounds. Those symptoms was followed by nervous manifestations in the form of torticolis, lack of equilibrium, incoordination of movement and paresis. Deaths occurred 1, 4, 6, 7 and 8 days post-infection.

On post-mortem examination, the most obvious lesions were located in the brain and lung. Macroscopically, the brain revealed no specific alterations except hyperaemia of the meninges. The lungs showed severe pulmonary congestion and focal areas of consolidation in those poult's died one and four days post-infection. In other poult's many illar sized, turbid greyish or even greyish white well defined nodules were detected. Most of the nodules were surrounded by hyperaeamic zones and on cutting they showed centrally a fragmented, greenish, caseated material which could be easily removed.

On microscopic examination, the poult's which died one and four days post infection showed meningitis. The meninges were thickened, hyperaeamic zones and showed cellular infiltrations Fig. 1. In the other cases which later on, typical focal ischaemic and liquifactive cerebral necrosis was observed Fig. 2. The blood vessels in the affected areas showed intravascular thrombosis, moreover endovasculitis and some cases, hyalination of the wall Fig. 3. Mycelia of the fungus could be seen in the area of malacia Fig. 4. In the cerebellum, demylinating encephalitis could be observed Fig. 5.

Microscopical examination of the lungs in birds which died one and four days after infection showed diffuse serofibrinous pneumonia with heavy cellular infiltrations Fig. 6. In the other cases, two forms of pulmonary alteration were noticed, either a diffuse chronic productive inflammatory reaction in which great number of giant cells could be seen and the condition could be termed as giant cell pneumonia Fig. 7, or a focal granulomatous pulmonary form, in the latter, the nodules consisted of caseated central core surrounded by inflammatory cellular infiltrations and peripherally surrounded by a connective tissue capsule, Fig. 8. The surrounding alveoli showed pressure atelectasis with areas of compensatory emphysema.

Trials to determine the sensitivity of the isolated fungi to the fungicidal and chemical agents used, proved that all isolates were sensitive to mercuric iodide, Copper sulphate and thiobenzazol, while they were resistant to Erthromycine and Emetine Table III.

DISCUSSION

As reported by CHUTE 1978, lesions in aspirogillosis depend cons derably on the site of infection. Frequent occurrence of lesions in the lungs in spontaneously affected cases showed the importance of inhalation as a route of infection under field conditions. Also, as revealed by experimental inoculation of A. flavus in the abdominal sac in the present study, the lung was the organ mostly severely affected. Microscopically, the lesion consisted of either focal or diffuse productive inflammation which developed relatively rapidly that it can be observed as early as the first week of infection. These results are parallel to those of many investigators who recorded the occurrence of granulation reaction due to aspirogillosis in the lung, KURASEVA, 1966, ADAMSTREANU et al., 1969, KUNDTSON and MEINCECKE, 1972. Giant cells were also frequently observed, both in the lung, KUNDTSON and MEINCECKE, 1972 and brain REINES et al., 1966.

The present study revealed, moreover, that inoculation of A. flavus in the abdominal air sacs can be followed by a generalized infection, this is indicated by the presence of pathological lesions and hypaeae in the brain. It can be suggested that after reaching the lung through the air sacs the fungus gain access to the brain most probably by invasion of the alveolar walls, interalveolar capillaries then to the blood circulation.

Lesions in the brain was related more to a local circulatory disturbance, resulting from damage of the vascular bed than to a direct effect of the myotoxin.
PNEUMONIA and ENCEPHALAMALACIA

Culture growth of the fungus, A. flavus could be inhibited in vitro by mercuric iodide, copper sulphate and thiobenzazol. CISZEWSKI, 1968 reported the advantage of copper sulphate in drinking water as a treatment of aspirognillosis in ducklings. KLIINES and KRIZ 1968 mentioned that thiobenzazol could be used as a preventative therapy for chickens. SAIF and REFAI 1977 successfully used thiobenzazol for control of moulds in poultry farms.

REFERENCES


DESCRIPTION OF FIGURES

Fig. 1: Showing meningitis. H. & E. (X 250).
Fig. 2: Brain showing focal ischaemic liquifactive necrosis. H. & E. (X 160).
Fig. 3: Cerebral blood vessel showing intravascular thrombosis and vasculitis. H. & E. (X 400).
Fig. 4: Showing mycelia of the fungus in the brain tissue. H. & E. (X 400).
Fig. 5: Cerebellum showing demyelination. H. & E. (X 160).
Fig. 6: Lung showing serofibrinous pneumonia. H. & E. (X 160).
Fig. 7: Lung showing diffuse productive inflammation. H. & E. (X 400).
Fig. 8: Lung showing focal productive inflammation. H. & E. (X 400).

Table (I)
Showing the frequency, age, breed, state, and sources of the examined samples.

<table>
<thead>
<tr>
<th>Frequency of samples</th>
<th>Age</th>
<th>Breed</th>
<th>State of samples</th>
<th>Source</th>
</tr>
</thead>
<tbody>
<tr>
<td>160</td>
<td>1-4 weeks</td>
<td>White indeco</td>
<td>Freshly dead</td>
<td>El-Wadi El-Gadid Governmental Turkey Farm.</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>50</td>
<td>8-12 months</td>
<td>Native</td>
<td>Living, apparently healthy</td>
<td>Popular flocks from Kena province.</td>
</tr>
</tbody>
</table>

Table (II)
Showing species, number and percentage of isolated fungi.

<table>
<thead>
<tr>
<th>Species</th>
<th>Number of isolated fungi</th>
<th>Percentage to total isolates</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Crop.</td>
<td>Lung.</td>
</tr>
<tr>
<td>A. funigatus</td>
<td>28</td>
<td>40</td>
</tr>
<tr>
<td>A. flavus</td>
<td>18</td>
<td>10</td>
</tr>
<tr>
<td>A. niger</td>
<td>13</td>
<td>19</td>
</tr>
<tr>
<td>A. flavipsis</td>
<td>20</td>
<td>3</td>
</tr>
<tr>
<td>Penicillium spp.</td>
<td>-</td>
<td>41</td>
</tr>
<tr>
<td>Mucor spp.</td>
<td>16</td>
<td>5</td>
</tr>
<tr>
<td>Rhizopus spp.</td>
<td>-</td>
<td>4</td>
</tr>
<tr>
<td>Paecilomyces</td>
<td>8</td>
<td>3</td>
</tr>
<tr>
<td>Scopulariopsis</td>
<td>6</td>
<td>3</td>
</tr>
<tr>
<td>Yeast and yeast-like fungi</td>
<td>21</td>
<td>12</td>
</tr>
</tbody>
</table>
## Table (III)

**Table showing the effect of Fungicides & Chemical agents on isolated fungi**

<table>
<thead>
<tr>
<th>Species of fungi</th>
<th>Erythro-mycin 50 mg/ml</th>
<th>Pot. iodide 20 mg/ml</th>
<th>Mor. iodide 10 mg/ml</th>
<th>Emetine 6 mg/ml</th>
<th>Cop. Sulphate 20 mg/ml</th>
<th>Chloramphenical 20 mg/ml</th>
<th>Nystatin 80 i. u/ml</th>
<th>Griseofulvin 16 mg/ml</th>
<th>Thiobendazole 2.5 mg/ml</th>
</tr>
</thead>
<tbody>
<tr>
<td>A. fumigatus</td>
<td>--</td>
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<td>++</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>++</td>
</tr>
<tr>
<td>A. flavus</td>
<td>--</td>
<td>--</td>
<td>++</td>
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<td>++</td>
<td>+</td>
<td>+</td>
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<tr>
<td>A. niger</td>
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<td>++</td>
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<tr>
<td>A. flavipra</td>
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<tr>
<td>Paecilomyces</td>
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<td>++</td>
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<td>+</td>
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<tr>
<td>Scopulariopsis</td>
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<td>+</td>
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<td>++</td>
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<tr>
<td>Rhizopus</td>
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<td>++</td>
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<td>+</td>
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<tr>
<td>Mucor</td>
<td>--</td>
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<td>++</td>
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<td>+</td>
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<tr>
<td>Penicillium</td>
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<td>++</td>
<td>++</td>
<td>++</td>
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</tr>
</tbody>
</table>

++ : Complete inhibition of culture growth.
+ : Moderate growth of the culture (Moderate inhibition).
- : Complete or heavy growth of the culture (No inhibition).

*Abd el Galil, *et al.*, (1972).*
