قسم: الباثولوجيا
كلية: الطب البيطرى - جامعة أسيوط
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مرض الباستيرلا في الأرانب والموال الصيغة له

عبد اللطيف بومي، الرفاعي محمد*، عوض عبد الحافظ*، صلاح ديب

تتم هذه الدراسة على خمسة وعشرون من ذكور وأرانب البسكات تَسَمَّى
احذارها من وحدة الحيوانات الطبية بجامعة أسيوط. بعد التأكيد
من خمسة من الأمراض البكتيرية والطفيلية قُسمت إلى خمسة مجموعات
منفصلة كل منها خمسة أرانب.

تتم عدد الحيوانات صانعًا لما بيفروب الباستيرلا وحده أو بالإضافة
إلى ميكروب البوكرت، بيتلا أو ميكروب الدبلوكوس أو الميكروب العصوى
القفلوني وتشكل المجموعة الخامسة بدون عدد كضابط للتجربة.

لوحظ الأعراض، أجريت الصفة التشريحية للحيوانات النافقة وأخذت
عينات من القصية الهوائية والرئة، الكلية، الامعاء، الكبد، المخ
الفصوص الميكروسكوبية.

فحصت العينات الميكروسكوبية ونوقشت النتائج وقد تبين أن المواد
البكتيرية التي استخدمت في البحث تلعب دورًا في شدة الأصابات
بميكروب الباستيرلا.

** قسم الأمراض الباطنة وأمراض الدواجن
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RABBIT PASTEURELLOSIS AND ITS INITIATING FACTORS
(With 2 Tables and 15 Figures)

By
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SUMMARY

The present work was carried out on twenty five male Boskot rabbits, obtained from the laboratory Animal Unit, Assiut University. The animals were divided into five groups and experimentally infected with Pasteurella multocida alone or associated with Diplococcus pneumoniae, E.Coli or Bordetella bronchiseptica. The clinical picture was recorded and post-mortem examination was carried out as well as reisolation of the inoculated strains was conducted. Trachea, Lung, Kidney, intestine, liver and brain were examined microscopically.

The present work indicated that such bacterial agents not only play an important role in initiating rabbit pasteurellosis but also shared in the severity of infection.

INTRODUCTION

Rabbits, in addition to their nutritive value, are also used as experimental animals in medical and biological institutes for both diagnostic and research purposes. Pasteurellosis still represents the most common bacterial infection in rabbits and cause high losses almost in all ages especially in those under adverse conditions.

In Egypt, isolation, identification and capsular typing of Pasteurella multocida in rabbits was studied by SAAD (1970), ZAHER, et al. (1976), EL-GHAWAS (1980) and in upper Egypt by YOUNES (1982).

A number of secondary microorganisms were isolated in association with Pasteurella multocida infection in rabbits (YOUNES, 1982) and it was presumed that lowering the resistance of animals caused and extension of the previously existing inflammatory process.

The present work was designed to elucidate the role of Diplococcus pneumoniae, Bordetella bronchiseptica and E.coli in the pathogenesis of Pasteurellosis induced in rabbits.

MATERIALS and METHODS

Pasteurella multocida type A, E.coli 0 25K11 (L), Diplococcus pneumoniae and Bordetella bronchiseptica strains isolated from rabbits by YOUNES (1982) were used in our study.

The present work was carried out on thirty male Boskot rabbits, 8-weeks-old, obtained from the laboratory Animal Unit, Assiut University. The animals were housed in separate cages, fed standard ration supplemented with vitamins and minerals and were put under observation for a week before the experimental infection.

* : Dept. of pathology.
**: Dept of poultry Dis.

Random samples of five animals were slaughtered, and subjected to post-mortem examination, also bacteriological and parasitological examinations were conducted. Twenty five bacteriologically and parasitologically free animals were removed and isolated for the experiment. The animals were divided into five groups, each consisted of five animals. The rabbits were experimentally infected as shown in Table (1). During the experimental period, the clinical picture was recorded and post-mortem examination was carried out shortly after death of the animals. Resolution of the inoculated strains was conducted. From all, dead and sacrificed animals, specimens for histopathological examination were taken from the lung, trachea, kidney, intestine, liver and brain. The samples were fixed in 10% neutral buffered formalin, embedded in paraffin, sectioned, stained with haematoxylin and eosin and examined. The control animals were sacrificed at the end of the experiment which lasted for and specimens for microscopical examination were taken.

RESULTS

Clinical observations:

The animals in group one, which were infected with Pasteurella lepisepctica only, showed depression, rough fur, dull eyes, laboured breathing, frothy nasal discharge and only two animals showed diarrhea. Similar picture, although severe form, was also observed in group two, in which the animals were infected with Pasteurella and Diplococcus pneumoniae. In group three where Bordetella bronchiseptica was used in addition to Pasteurella Infection, Laboured breathing and the mucopurulent discharge were prominent. In addition to the respiratory symptoms aforementioned, severe diarrhea occurred in all animals of group four infected with both Pasteurella and E.coli.

In regard to the survival period after infection (Table 2), all animals of group one died within the fifth day after infection. Animals of group two and those of group three died within the third day after infection, but animals of group four died within the sixth day post-infection.

Macromorphological results:

In group one, the trachea was congested, inflamed and filled with a frothy blood-stained exudate. The lungs were congested and showed scattered, well demarcated reddish brown to reddish grey focal areas of consolidations. The pneumonic foci were usually bilaterally and mainly affecting the ventral portion of the cardiac lobes. The kidneys were slightly congested while the other organs showed no changes.

In group two, the post-mortem picture was similar to group one except, that, in the lungs, the areas of consolidations progressed to involve much of the entire cardiac lobes. On cut section the lung was firm and the bronchi were filled with thick exudate. The meningeal vessels were congested.

In group three, in almost all the animals, the pleural cavity contained varied amounts of clear sanguineous fluid. The lung was more severely affected when compared with the above two groups the foci of consolidation could be observed in the cranioventral portions of the diaphragmatic lobes. The bronchial lumens were filled with slimy, yellowish grey exudate. Areas of atelectasis and others of emphysema could be also observed.

In group four, the trachea was inflamed but to a lesser degree than in other groups. Lung consolidation and bronchial exudations were restricted to few focal areas in the cardiac
lobes. The kidneys were swollen and pale in colour. The liver was slightly enlarged, congested and had rounded borders. The intestines contained a large amount of catarhal exudate and showed a congested mucosa. These inflammatory changes could be seen only in the small intestine.

**Micromorphological results:**

In group one the trachea showed epithelial desquamation and severe hyperaemia (Fig. 1). The lungs revealed areas of fibrinous pneumonia in which the fibrin deposited in the alveolar spaces (Fig. 2). Many areas showed neutrophilic cellular infiltrations. The interalveolar capillaries were greatly congested. In some areas varied numbers of alveolar macrophages could be observed (Fig. 3). The kidneys showed marked tubulonephrosis manifested by degeneration of the epithelial lining of their tubules, the intertubular capillaries were severely congested and some of them were ruptured (Fig. 4).

In group number two, degenerative changes and desquamation of the epithelium associated with hyperaemia and haemorrhages of the mucous membrane could be observed in the trachea (Fig. 5). The lungs revealed the picture of fibrinous pneumonia similar to animals of group one. Moreover slight degree of bronchitis and bronchiolitis manifested by degeneration of their epithelial lining as well as peribronchial hyperaemia and oedema could be seen. The pulmonary ramifications showed intimal degenerative changes and perivascular oedema Fig. (6). Changes in the kidneys were identical to those in group one. The brain showed congestion of the cerebral blood vessels associated with oedema in virchow Robin spaces and slight microglosis (Fig. 7).

In the third group, the tracheal wall showed severe oedema and hyperaemia (Fig. 8). Areas of haemorrhages and mononuclear cell infiltrations could be observed. In the lung bronchitis and bronchiolitis were marked in which the lumens were partially filled with cellular exudate containing mononuclear cells and desquamated epithelial cells. The rest of the lung parenchyma showed severe hyperaemia, oedema and inflammatory cellular infiltrations (Fig. 9), in which polymorphnuclear leucocytes consisted the main type.

In the fourth group, microscopic examination of the trachea revealed moderate oedema, inflammatory cell infiltration and slight hyperaemia (Fig. 10). Animals of this group suffered from interstitial pneumonia (Fig. 11), in which the interalveolar septa were thickened due to accumulation of cellular inflammatory exudate. The predominant were mononuclears. In the kidneys, in addition to tubulonephrosis, slight interstitial reaction, degeneration of vascular endothelium and perivascular oedema could be observed (Fig. 12). Changes in the liver consisted of hepatic cells with marked pyknotic and karyorhectic nuclei could be seen (Fig. 13). In the intestine, epithelial degeneration and desquamation in addition to submucosal hyperaemia and oedema (Fig. 14 a,b) resembled the main findings.

In the fifth group, which was used as controls, neither clinical nor morphological alterations could be observed except in one animal, its liver showed minute, focal greyish white spots which on microscopic examination revealed hyperplastic cholangitis (Fig. 15).

**DISCUSSION**

As it is, reported by (WATSON et al., 1975, SPANOOGHE and OKERMAN 1978 and HIME 1979), the infectious and non infectious agents which act as stressful conditions were necessary for overt the rabbit snuffles to appear. The present work was designed to elucidate the role of Bordetella bronchiseptica, Diplococcus pneumoniae and Ecoli which were isolated by YOUNES (1982) in association with Pasteurella multocida as infectious agents which may diminish...
the animal resistance.

For the present study, twenty five, male Bosket rabbits, about 800 - 1,000 gm in weight were obtained from the laboratory animal unit, Assiut University. These were selected to exclude if possible, previous attacks of rabbit pasteurellosis. The rabbits were exposed to cultures of either Pasteurella multocida alone or associated with, Diplococcus pneumoniae, Bordetella bronchiseptica or E.coli.

Symptoms of respiratory affection were a prominent features in all groups. More or less, these clinical signs were also described by (OSTLER, 1961, ZAHER et al., 1976 and YOUNES 1982). Deaths occurred between 1st. and 6th. day-post-infection, the mortality rate was 100% and this indicates that the locally isolated strains were highly pathogenic for susceptible rabbits. Nearly in all groups, the microscopic lesions of dead rabbits were restricted to the respiratory tract. In group three, the lung was more severely affected when compared with the other groups. Not only in character but also in distribution where the pneumonic foci extended also to involve the cranioventral portions of the diaphragmatic lobes. This could be probably due to the synergistic effect of the two microorganisms especially that both have been associated with pneumonia (WISSNER 1960, SATO and NAMIOKA 1967, FLATT and DUNGWORTH 1971 and SPANOGLHE and OKERMANN 1978). The involvement of the tracheal, bronchial and peribronchial as well as involvement of the lung alveolar tissue indicates that the infection having entered by the bronchial route. Microscopically it was not easy to differentiate pneumonia produced by either organism, since the characteristics of the lesion were more or less similar and this also was observed by WATSON et al., (1975). Similar lesions were also noted in the naturally occurring disease (LÖLGER, ALBERTI and MATTHES 1972, HIPPE 1979). In the vicinity of the pneumonic foci, they were almost areas of emphysema and others of atelectasis. The formers were due to the decreased space occupied by neighbouring alveoli which were either atelectatic or consolidated. The atelectatic areas could be attributed not only due to the plugging of the air ramifications by masses of exudate but also due to the pressure of the consolidated areas. The presence of the large cells with eccentrically situated nuclei and a rather extensive cytoplasm in varied numbers, some of these cells must be monocytes from the blood, others doubtfuls came from the reticuloendothelial system and there is a considerable evidence that many of them are proliferating alveolar epithelium. In the fourth group, interstitial pneumonia was observed, this could probably be due to the relative prolonged survival time which gave the chance for the interstitial inflammatory reactions.

The brain lesions of group two in the present study could be assumed either to the toxins produced by pneumocci (DICK and GIMMEL 1971, BURNNET and SCHUSTER 1073) or due to Pasteurella multocida, Khera, Pandurangrrao and Mall (1971).

The vascular degenerative changes observed in the second and fourth groups could be attributed to the toxins of Diplococcus pneumoniae and E.coli respectively. In the fourth group, the inflammatory process with its alterative and exudative phenomenas were observed in the intestines and could be assumed to the E.coli infection, PRESCOTT (1978).

The hyperplastic cholangitis seen in one animal of the control group was due to infect with coccidia.

The present work indicated that bacterial agents as Bordetella bronchiseptica, Diplococcus pneumoniae and E.coli not only play an important role in initiating rabbit pasteurellosis but also shered in the severity of infection.
RABBIT PASTEURELLOSIS

REFERENCES


DESCRIPTION OF FIGURES

Fig. (1): Showing epithelial desquamation and hyperaemia of the trachea. H & E. (X400).

Fig. (2): Lung showing fibrinous pneumonia. H. & E. (X400).

Fig. (3): Lung showing alveolar macrophages. H. & E. (X400).

Fig. (4): Kidney showing tubulonephrosis and focal haemorrhage. H. & E. (X250).
Fig. (5): Trachea showing epithelial desquamation, hyperaemia and haemorrhage. H. & E. (X250).
Fig. (6): Pulmonary vessel showing intimal degeneration and perivascular oedema. H. & E. (X400).
Fig. (7): Brain showing congestion and oedema. H. & E. (X250).
Fig. (8): Trachea showing oedema and hyperaemia. H. & E. (X250).
Fig. (9): Lung showing bronchiolitis. H. & E. (X400).
Fig. (10): Trachea showing oedema. H. & E. (X250).
Fig. (11): Lung showing interstitial pneumonia. H. & E. (X160).
Fig. (12): Kidney showing tubulonephrosis, vascular endothelial degeneration and perivascular oedema. H. & E. (X400).
Fig. (13): Liver showing karyorrhetic and pyknotic nuclei. H. & E. (X250).
Fig. (14): Showing enteritis. H. & E. (X250).
Fig. (15): Liver showing hyperplastic cholangitis. H. & E. (X400).
Table (1): Showing the animal grouping type of infection, its route and dosage

<table>
<thead>
<tr>
<th>Animal group</th>
<th>Number of animals</th>
<th>Type of Infection</th>
<th>Route and dose</th>
</tr>
</thead>
<tbody>
<tr>
<td>G.One</td>
<td>5</td>
<td>Pasteurella multocida A</td>
<td>Intranasal 6X10&lt;sup&gt;9+&lt;/sup&gt;</td>
</tr>
<tr>
<td>G.Two</td>
<td>5</td>
<td>Pasteurella multocida A +</td>
<td>Intranasal 5X10&lt;sup&gt;9+&lt;/sup&gt;</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Diplococcus pneumoniae</td>
<td>Intraperit. 1 ml broth cult. ++</td>
</tr>
<tr>
<td>G.Three</td>
<td>5</td>
<td>Pasteurella multocida A +</td>
<td>Intranasal 5X10&lt;sup&gt;9+&lt;/sup&gt;</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Bordetella bronchiseptica</td>
<td>Intranasal 3.5X10&lt;sup&gt;10+&lt;/sup&gt;</td>
</tr>
<tr>
<td>G.Four</td>
<td>5</td>
<td>Pasteurella multocida A +</td>
<td>Intranasal 5X10&lt;sup&gt;9+&lt;/sup&gt;</td>
</tr>
<tr>
<td></td>
<td></td>
<td>E.coli 025 K 11 (L)</td>
<td>Oro-gastrie 4X10&lt;sup&gt;10&lt;/sup&gt;+++</td>
</tr>
<tr>
<td>G.Five</td>
<td>5</td>
<td>Non infected controls</td>
<td></td>
</tr>
</tbody>
</table>


Table (2): Showing the survival period of animals after infection

<table>
<thead>
<tr>
<th>Animal group</th>
<th>Type of Infection</th>
<th>day-post infection</th>
<th>Number of Dead animals</th>
</tr>
</thead>
<tbody>
<tr>
<td>First</td>
<td>Pasteurella multocida A</td>
<td>3 rd.</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td></td>
<td>4 th.</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td></td>
<td>5 th.</td>
<td>3</td>
</tr>
<tr>
<td>Second</td>
<td>Pasteurella multocida A +</td>
<td>1 st.</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>Diplococcus pneumoniae</td>
<td>2 nd.</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td></td>
<td>3 rd.</td>
<td>2</td>
</tr>
<tr>
<td>Third</td>
<td>Pasteurella multocida A +</td>
<td>1 st.</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>Bordetella bronchiseptica</td>
<td>2 nd.</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td></td>
<td>3 rd.</td>
<td>1</td>
</tr>
<tr>
<td>Fourth</td>
<td>Pasteurella multocida A +</td>
<td>3 rd.</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>E.coli</td>
<td>4 th.</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td></td>
<td>5 th.</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td></td>
<td>6 th.</td>
<td>1</td>
</tr>
</tbody>
</table>
