SNUFFLES DISEASE IN RABBITS 1- CLINICAL AND BACTERIOLOGICAL STUDY

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ABSTRACT
Snuffles disease is a major problem in rabbits caused by *Pasteurella multocida*, for the importance of this disease, this study aimed to identify clinical and bacteriological findings associated with experimental *P. multocida* infection in rabbits. Two groups of rabbits: 1st group (7) control group injected i/p with pbs. 2nd group infected group (27) injected i/p with 10^2 CFU/ml of *P. multocida* CLD50 was 10^7 CFU/ml. Different clinical signs were observed in rabbits at 1st – 20th day post infection, the clinical signs included excitation, then dullness, anorexia, fever, congestion of conjunctiva and loss of weight and mucopurulent nasal discharge later on bacterial dissemination, *P. multocida* were isolated from the different organs of the infected rabbits at 4th, 8th, 12th, 16th and 20th days post infection. The levels of the bacterial isolates were very heavy, heavy and moderate at the beginning and mild and very mild isolates at the end of infection.

KEYWORDS: *Pasteurella multocida*, CLD50, mucopurulent, bacterial dissemination.

INTRODUCTION
Snuffles disease is a major disease in the rabbits caused by various serotypes of *Pasteurella multocida*, occurring during the stress such as mating, shipping and experimental handling. The various serotypes of *P. multocida* may replicate rapidly causing disease such as pneumonia, otitis, media, conjunctivitis and septicemia and atrophic rhinitis[1,2]. Snuffles occurs in 50%–70% in conventional and non-barrier maintained rabbitaries harboring *P. multocida* in the upper respiratory tract and tympanic bullae and there is seasonal influence as the most problem occur in the spring[3] also there is predisposing factors include increased atmospheric ammonia, pregnancy, concomitant disease and environment disturbances. For the importance of this disease problem in rabbits. This study aimed to identify the clinical signs and the dissemination of *P. multocida* in rabbits following the experimental infection.

MATERIALS & METHODS
A local strain of *P. multocida*, reidentified[4], LD50 and infective dose determined by[5] LD50 was 10^7 CFU/ml and the lethal dose was 10^2 CFU/ml.

RESULTS & DISCUSSION
1-clinical signs
The main clinical signs were appeared on the rabbits after 24h include the minimal excitation for short time, later all rabbits become depressed, showed anorexia, dullness, fever 40.2 - 40.5 C, congestion of the conjunctiva, loss of body weight and sever mucopurulent nasal discharge (Fig-1) were observed along the experiment and death of rabbits.

FIGURE 1: clinical manifestation of infected rabbit showed mucopurulent nasal discharge (snuffles disease) after 8-12 days of infection
These clinical signs occurred in relation to the multiorgan dysfunction following bacterial endotoxemia[6,7]. Also anorexia and loss of weight were observed in all the infected rabbits which associated with catabolic effect of inflammatory mediator like TNF-α which induced glucose catabolism in muscle following the effect of LPS of *P. multocida* via it's spread in blood stream[8,9]. Similarly LPS endotoxins act as exogenous pyrogens will activate macrophage due to produce ILI as endogenous pyrogens that would enhanced conversion of arachidonic acid to prostaglandins by increase cyclooxygenase then subsequent elevation of thermal set up point in the thermo regular center of hypothalamus[10,11].

**2-Bacterial dissemination**

The results showed that *P. multocida* was isolated from the most of examined organs of the infected rabbits. The levels of bacterial isolates showed variation (very heavy, heavy, moderate, mild and very mild). According to period of infection and organs; Lungs, liver, spleen, lymph nodes, heart, blood and kidneys are the main organs which showed more extensive bacterial isolation (Table-1).

TABLE 1: The degree of bacterial isolation from some examined organs of infected rabbits with $10^2$ CFU/ml intraperitoneally

<table>
<thead>
<tr>
<th>Days</th>
<th>Organs</th>
<th>Lung</th>
<th>Liver</th>
<th>Spleen + Lymph node</th>
<th>Heart blood</th>
<th>Kidney</th>
</tr>
</thead>
<tbody>
<tr>
<td>4 days</td>
<td>++++</td>
<td>++++</td>
<td>++++</td>
<td>++++</td>
<td>++++</td>
<td>++++</td>
</tr>
<tr>
<td>8 days</td>
<td>+++</td>
<td>+++</td>
<td>+++</td>
<td>+++</td>
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<td>+++</td>
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<tr>
<td>12 days</td>
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<td>++</td>
<td>++</td>
<td>++</td>
<td>++</td>
<td>++</td>
</tr>
<tr>
<td>16 days</td>
<td>++</td>
<td>++</td>
<td>+++</td>
<td>+</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>20 days</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
</tr>
</tbody>
</table>

++++ Very heavy (61-100 C), +++ heavy (31-60 C), +++ moderate (16-30 C), ++ mild (6-15 C), + very mild (1-5 C)

The results also showed that all the organs carried high levels of bacterial isolates in early stage of infection but in advance stage these bacterial isolates began decline gradually. These results agreed with[11,12,13,14,15] the *P. multocida* were established in a primary sites of infection and multiply and spread to other organs the organism in this study spread through the periton into the lymphatic and blood vessels causing bacteremia and to spread into different organs and induce the disease following their multiplication in each organ[16]. *P. multocida* has several virulence factors play a major role in pathogenesis and pathogenicity and their distribution in the different organs[4]. Among the virulence factor, fimbria, hyaluronidase, neuraminidase, protease, lipase and sialidase are the important enzymes help the *P. multocida* for colonization and inhibit phagocytosis and enhance bacterial invasion of host tissue[17-18]. The level of bacterial isolation declined in the organs during the advance days of infection because of development of the immunity. The LPS of *P. multocida* will stimulate the innate immune system whereby inflammatory response plays a critical role in bacterial clearance and subside the infection[19].

**REFERENCES**


