Pathological studies on pigeon trichomoniasis with reference to the associated bacteria

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SUMMARY

One hundred and twenty apparently healthy and diseased squabs (up to 2 month old) were collected from Sharkia Governorate our work revealed that 80 out of 120 examined squabs were positive for T. gallinae. The birds suffered depression, off food and dyspnea. Offensive odor-fluid drilled from their opened beak. Macroscopically, the oropharynx esophagus and crop revealed caseated materials, besides necrotic foci in the liver and heart. Experimental infection with trichomonas gallinae was done on twenty examined healthy squabs and treated with flagyle. The result of these experimentally infected squabs revealed typical symptoms and postmortum lesions of Trichomoniasis, and flagyle was effective against T.gallinae. Specimens were collected from the oropharynx, liver, heart-blood and lungs for bacteriological examination and from the crop, esophagus and brain for histopathological examination.

The isolated bacteria were Pasteurella multocida type D1, Streptococcus bovis and E.coli at the rates of 65%, 22.5% and 12.5% respectively. Microscopically the natural and experimental trichomoniasis showed mild to massive caseous necrosis with bacterial colonies were found on the pharyngeal mucosa. Submucosal hemorrhage and leukocytic infiltration were noticed. The esophagus showed caseous necrosis and congestion. The hepatic parenchyma showed hydropic degeneration and focal replacement with radially arranged pericytes. The portal areas were infiltrated with numerous leukocytes. The myocardium showed hemorrhage, mononuclears, and necrosis of the myocytes. The lungs showed congestion and hemorrhage with leukocytic aggregations. The brain showed necrotic Purkenje cells and necrosis of the large cerebral neurons. The most effective drugs, against the isolated bacteria were Avitryle, Norfloxacin, Gentamicin and Ampicillin. Flagyl was effective against trichomoniasis.

Our work revealed that the trichomonas infection and the secondary
INTRODUCTION

Pigeons contribute to the poultry industry, in Egypt. Their meat is palatable for the majority of the Egyptians and other nationalities.

Pigeons are affected with diseases of diverse etiological nature. Less effort has been attempted, in Egypt, to investigate the prevalent diseases in pigeons except for a few reports which dealt with certain infections (Shihata 1978, Nagwa 1995, Eman 2005 and Hebat-Allah and Abd El-motelib (2007). Little attention has been directed toward diseases of pigeons. This may be due to the fact that the majority of these birds are unconfined as they usually wander over a long distance searching for food, and the population of pigeons is few when compared with chickens, that are reared intensively (Abd El-Motelib and Galal, 1993).

Trichomoniasis is a common disease of squabs that affects the upper digestive and respiratory tracts, causing high losses (Levine, 1995). The severity of the disease and mortality, in squabs, may vary according to the susceptibility of the squabs and the virulence of the pathogenic strain of Trichomonas gallinae (Abd El-Motelib and Galal 1993, MacDaugald 2003 and Swinnerton et al. 2005).


The streptococcal infection is usually peracute or acute, and rarely chronic (Devriese et al. 1990) Meanwhile Andreasen and Sandhu (1993), found that Pasteurella is associated with respiratory diseases, in pigeons, as pneumonia and tracheitis. Gross (1991) found that E.coli infection led to great economic losses in poultry.

The objectives of this study were aimed to detect natural (single or mixed bacterial infection) associated with trichomonas with isolation and identifecation of bacterial causes besides studying the macro and microscopic lesions, and the antibiogram for isolated bacteria. Moreover, traile for treatment of experimentally infected squabs with trichomonas gallinae.

MATERIAL AND METHODS

SAMPLES:-

A total of 120 (40 apparently healthy and 80 diseased) pigeons up to 2 month old and from different breeds were collected from different markets in Sharkia Governorate. The specimens were col-
lected from oropharynx, liver, heart blood and lungs (from each birds) for bacteriological examination. And the crop, esophagus and brain were used for histopathological examination.

Parasitological examination and preparation.

a- Microscopical examination for detection of the parasite:

Samples were collected from the different lesions and caseated materials of 80 diseased birds and examined microscopically for detection of trichomonas gallinae.

b- Preparation of *Trichomonas gallinae* culture:

Wet swabs were prepared, by gentle rotation, through the lesions. The swabs were dipped into sterile saline or culture-media (Glucose-serum broth medium) according to Nagwa (1995). The inoculated tubes were incubated at 37°C for 7 days. One drop, from the bottom of each tube, was microscopically examined 4 and 7 day post-inoculation (PI) for the presence of trichomonads according to Abd El-Motelib and Galal (1993). The positive tubes were subcultured into new tubes of media and incubated for 3 days.

Estimation of the population density of the parasite:

A measuring dropper (giving 3 drops/0.1 ml) was used to count number of the parasites in one drop according to Nagwa (1995).

Inocula were used to seed the culture-tubes. Those cultures, yielding between 800-1000 parasite /ml, were used according to Nagwa (1995).

c- Experimental infection:

Thirty apparently healthy squabs up to one month age were collected from different markets in Sharkia governorate and were subjected to parasitolgical Examination to assure that the examined squabs were free from infection. The examined squabs were reared in two separated rooms under complete hyagenic measures, and devided into 2 group the first group (20) squabs were infected orally by dropper with 1ml containing (800 – 1000) parasite. The second group 10 squabs were left as control.

Bacteriological examination:

1- Isolation and identification:

The collected Specimens (oropharynx, liver, heart-blood and lung) were inoculated into tubes of nutrient broth and incubated aerobically at 37°C over night followed by subculturing into nutrient agar, MacConkey’s agar and blood agar plates and incubated for 24-48
hours at 37°C. The suspected colonies were identified morphologically according to Cruickshank et al. (1975) and confirmed biochemically according to Krieg and Holt (1984).

2- Serological identification of Pasteurella Sp.:

Pasteurella organism identified serologically according to Carter and Roppy (1963) for capsular typing with indirect hemagglutination test and somatic typing using gell diffusion precipitation test.

3- Pathogenicity of Pasteurella for mice:

All strains of Pasteurella multocida were tested for pathogenicity to mice according to Wilson and Milies (1975).

4- Antibiotic sensitivity test:

In vitro the disc diffusion technique was performed on the isolated bacteria using seven chemotherapeutic discs namely (Avitryl, Erythromycin, Gentamicine, Septrin, Norfloxacin and Ampicillin and Amoxycillin) susceptibility of the predominant pathogenic isolates to different chemotherapeutic agents according to Finegold and Martin (1982).

Histopathological preparation:

Tissue specimens, of 0.5 cm thickness were collected from the pharynx, esophagus, crop, liver, lungs, heart and brain, from freshly dead and slaughtered infected squabs, to detect the different tissue reactions against the parasitic and bacterial infections. The specimens were fixed in 10% neutral buffered formalin. Five micron-thick paraffin sections were prepared and stained with hematoxylin and eosin and examined microscopically (Bancroft and Gamble 2002).

The experimentally infected squabs were treated with Flagyl at a dose of one gm/liter drinking water for five successive days (Sampurnanand et al. 2002).

RESULTS AND DISCUSSION

Our work revealed that, Pasteurella multocida and Streptococcus bovis are considered facultative pathogens. They may be a part of the normal pharyngeal and intestinal flora in pigeons. Predisposing factors may cause some streptococci to break through the intestinal mucosa and enter the blood to stream to induce septicaemia (Vanrobaey et al. 2000 & Kimpe, et al. 2003 and Hebat-Allah & Abd El-Motelib 2007).

Table (1) shows the percentage of single and mixed bacterial infection in the appar-
ently healthy and diseased pigeons. Table (2) reveals that the isolated *Streptococcus bovis* was 22.5% which is nearly in agreement with Hebat - Allah and Abd - El - Motelib (2007). Meanwhile more isolates were recorded by De Herdt *et al.* (1993) who isolated bacteria was *Pasteurella multocida* type D1 at percentage of 65%, *Streptococcus bovis* at the rate of 40% from diseased pigeons of all ages. The rate of *E. coli* isolation in our study was 12.5% which are in accordance with Pedersen *et al.* (2006) who isolated *E. coli* at rate of 10%. In the vitro sensitivity test table (3) showed that Ampicillin, Norfloxacin, Erythromycin, Gentamicin, and Avitryle were highly effective against the isolated bacteria. Similar results were reported by De-Herdt *et al.* (1993) who found that Ampicillin, Doxycycline and Erythromycin were that most effective against the bacterial isolates. Hebat-Allah and Abd El-Motelib (2007) showed that ampicillin and Enrofloxacin was the most effective drugs. Shereen (2007) found that E.coli isolated from ostrich was sensitivs to enrofloxacin Gehan (2006) found that the isolated E.coli from quails was sensitive to Enrofloxacin and less sensitive to Erythromycin. Table (4) showed that bacterial isolates from oropharynx of apparently healthy and diseased pigeons but in liver, lung and heart blood the bacterial isolates were from diseased pigeons only by percentage as shown in the table.

All mice were tested for pathogenicity to *P. multocida* dead within 24-48 h. These results agreement with Okerman *et al.* (1979).

The natural and experimental affected squabs with trichomoniasis showed depression, off-food and dyspnea, in addition to offensive-odor-fluid, drilling from the opened peak, due to lesions in the oral cavity. Moreover, a large caseated mass was present in the buccal cavity. Sometimes greenish watery diarrhea was noticed. Similar signs were reported by Abd-El-Motelib and Galal (1993), Sohair and Effat (2004), Eman (2005) and Hebat-Allah and Abd-El-Motelib (2007). The treated squabs against trichomoniasis with Flagyl led to improvement in the clinical signs with decreased mortality rate within few days after the beginning of the treatment. The complicated trichomoniasis, with bacterial infection, frequently showed early death or chronic lameness. Similar findings were observed by Devriese *et al.* (1990) in trichomoniasis complicated with streptococcal infection in pigeons.

Macroscopically large whitish-yellow caseated masses of
variable sizes (3 – 30 mm diameter) were seen in the oropharynx (Fig.1). They reached the esophagus and crop. Sometimes the lumen of the crop was occluded with such caseated material. Similar observations were previously recorded by Abd-El-Motelib and Galal (1993), Nagwa (1995) and Sohair and Effat (2004). Moreover, trichomonas caused yellowish necrotic foci in the liver and lungs. Similar lesions were observed by Nagwa (1995), El-Metenawy (2000), MacDaugald (2003), Sohair and Effat (2004), Mohamed and Nahla (2005) and Eman (2005). Pneumonia and hepatic congestion were noticed. Similar lesions were recorded by Hebat-Allah and Abd-El-Motelib (2007). The ability of the parasites to reach these organs may be attributed to a disturbance between the resistance of the host and virulence of the parasite (Charlton et al. 1991; Narcisi et al., 1991 and MacDaugald 2003). This study revealed that the lesions varied from mild to moderate. The mild lesions could indicate single infection or a high resistance of the pigeons.

Microscopically, the examined pharynx of the naturally infected squabs with Trichomonas galliniae showed submucosal hemorrhage (Fig. 2). Another case, of natural trichomoniasis complicated with Pasteurlla multocida showed partial replacement of the pharyngeal wall by caseous necrosis (Fig. 3). Massive infiltration with heterophils and macrophages was detected in the pharyngeal wall of the naturally infected squab (Fig. 4). Similar lesions were previously detected by Shihata (1978), Narcisi et al. (1991), Nagwa (1995) and Eman (2005). The concurrent findings are in a partial agreement with Mohamed and Nahla (2005), who described focal homogenous and vacuolated necrosis. Another case showed acanthosis, necrosis and mucosal leukocytic infiltration, associated with submucosal hemorrhage of the pharynx (Fig. 5). These changes are in a partial agreement with Honigberg (1978) and Eman (2005). Massive caseous necrosis associated with bacterial colonies (Pasteurella multocida and Streptococcus bovis) were noticed in natural trichomoniasis (Fig. 6). Massive caseous necrosis was detected in the pharyngeal wall of the experimentally infected squab (Fig. 7). These results are in partial concurrence with those reported by Eman (2005) who found caseous necrosis in the pharynx. Large masses of caseous necrosis occluded the pharyngeal lumen of the naturally infected squab (Fig. 8). The previously reported lesions are in complete agreement with Eman (2005). Moreover, the esophagus of the experimentally infected...
squabs revealed caseous necrosis (Fig. 9). Moderately necrotic and congested mucosa was noticed (Fig. 10), due to natural trichomoniasis complicated with *Streptococcus bovis*. These changes are in agreement with those reported by Sohair and EFFAT (2004). Vacuolated crop mucosal epithelium was observed in experimental trichomoniasis (Fig. 11). Focal submucosal leukocytic aggregations were seen (Fig. 12). This lesion is in accordance with those recorded by Narcisi et al. (1991) and Nagwa (1995). Edema and submucosal hemorrhage were found in natural trichomoniasis complicated with E. coli (Fig. 13). The naturally complicated trichomoniasis with Pasteurella multocida and E. coli showed hepatic hydropic degeneration and focal replacement of the hepatic parenchyma by radially arranged pericytes around the central vein (Figs. 14, 15). These lesions are in a partial agreement with Abd-El-Rahman and Mousa (2000) who reported that *E. coli* infection in chicks induced coagulative necrosis and congestion. Sohair and Moursi (2003) found vacuolar degeneration and necrosis of hepatic cells due to pasteurellosis in ostrich. Eman (2005) found hydropic degeneration of some hepatocytes in pigeons infected with trichomoniasis. Moreover our findings are in complete agreement with Mousa and Magda (2006) who found hydropic generation in duck pasteurellosis. Medani et al. (2008) found diffuse vacuolar degeneration in the liver of infected seagulls with *E. coli*. The experimental trichomoniasis induced extensive portal infiltration with leukocytes (Fig. 18). This result is in complete agreement with that reported by Sohair and EFFAT (2004) and Eman (2005) due to trichomoniasis in pigeons.

The myocardium in the natural trichomonas complicated with Pasteurella multocida and *Streptococcus bovis* revealed focal replacement of the hepatic parenchyma by radially arranged pericytes around the central vein (Fig. 16). These lesions are partially in agreement with Mostafa (2002) who found coagulative necrosis of hepatic parenchyma due to pasteurellosis in poultry. Natural trichomoniasis, complicated with *Pasteurella multocida* and *E. coli* showed hydropic degeneration of hepatocytes (Fig. 17). Almost nearly similar lesions were reported by Sohair and Moursi (2003) who found vacuolar degeneration and necrosis of the hepatic cells due to pasteurellosis in ostrich. The myocardium in the natural trichomonas complicated with Pasteurella multocida and *Streptococcus bovis* revealed focal replacement of the hepatic parenchyma by radially arranged pericytes around the central vein (Fig. 16). These lesions are partially in agreement with Mostafa (2002) who found coagulative necrosis of hepatic parenchyma due to pasteurellosis in poultry. Natural trichomoniasis, complicated with *Pasteurella multocida* and *E. coli* showed hydropic degeneration of hepatocytes (Fig. 17). Almost nearly similar lesions were reported by Sohair and Moursi (2003) who found vacuolar degeneration and necrosis of the hepatic cells due to pasteurellosis in ostrich. Eman (2005) found hydropic degeneration of some hepatocytes in pigeons infected with trichomoniasis. Moreover our findings are in complete agreement with Mousa and Magda (2006) who found hydropic generation in duck pasteurellosis. Medani et al. (2008) found diffuse vacuolar degeneration in the liver of infected seagulls with *E. coli*. The experimental trichomoniasis induced extensive portal infiltration with leukocytes (Fig. 18). This result is in complete agreement with that reported by Sohair and EFFAT (2004) and Eman (2005) due to trichomoniasis in pigeons...
were previously reported by Sohair and Effat (2004). The myocardium of the naturally infected squabs complicated with E.coli was focally replaced with mononuclears (Fig. 20). Similar results were previously recorded by Abd El-Rahaman et al. (2003) due to E. coli infection. The experimental trichomoniassis led to proliferation of sarcolemmal nuclei with interstitial edema (Fig. 21). Similar results were previously reported by Sohair and Effat (2004) and Eman (2005). Such lesions could be attributed to the toxic metabolites, produced by the infective agents, these metabolites damaged the vascular endothelium, leading to edema and hemorrhage.

The lung of natural trichomoniassis complicated with Pasteurella multocida showed congestion and hemorrhage (Fig. 22) due to endothelial damage by the bacterial and parasitic metabolites which increased the permeability of the blood vessels leading to edema and hemorrhage (Jones et al. 1997). These changes are almost in agreement with Sohair and Moursi (2003) due to pasteurellosis in ostrich and Sohair and Effat (2004) due to trichomoniassis. Focal leucocytic aggregation were seen in naturally trichomoniassis and mixed infection with Pasteurella multocida (Fig. 23).

The brain of the naturally trichomoniassis revealed necrotic Purkenje cells of the cerebellum. Moreover, nerosis of large cerebral neurons was noticed (Fig. 24). These lesions are nearly similar to those reported by Narcisi et al. (1991) and Sohair and Effat (2004) who found neuronal degeneration and mild gliosis due to trichomoniassis in pigeons.

Our work revealed that the treatment of the trichomonas experimentally infected squabs with flagyl at a dose of 1 gm/liter drinking water for five successive days improved the general condition of the birds and decreased the mortality rate. A nearly similar results were reported by Sohair and Effat (2004), within few days after the beginning of the treatment.

It could be concluded that trichomoniassis, in most infected squabs was associated with secondary bacterial infection. P. multocida E. coli and Streptococcus bovis which became pathogenic under the stress of trichomoniassis.

Acknowledgement: We appreciate the fruitful advices of Dr. Nagwa Anwar helmy for her great helping with the parasitological study.

LEGENDS

Fig.
1. Natural trichomoniassis, squab showing whitish-yellow caseated
masses (3-30 mm in diameter) in the oropharynx.

2. Natural trichomoniasis, pharynx showing submucosal hemorrhage, arrow, H & E., x 200.

3. Natural trichomoniasis, pharynx complicated with Pasteurella multocida showing partial replacement of the pharyngeal wall with caseous necrosis, H & E., x 200.

4. Natural trichomoniasis, pharynx showing massive infiltration with heterophils and macrophages, arrows, H & E., x200.

5. Natural trichomoniasis, pharynx acanthosis (short arrow), necrosis, mucosal leukocytic infiltration (long arrow) associated with submucosal (L) hemorrhage, H & E., x 200.

6. Natural trichomoniasis complicated with Pasteurella multocida, pharynx showing massive caseous necrosis and bacterial colonies, (arrows), H & E., x200.

7. Experimental trichomoniasis, pharynx showing massive caseous necrosis of the pharyngeal wall, H & E., x200.

8. Natural trichomoniasis, pharynx showing a large mass of caseous necrosis occluding the pharyngeal lumen, H & E., x 100.

9. Experimental trichomoniasis, esophagus showing caseous necrosis, H & E., x200.

10. Natural trichomoniasis, esophagus complicated with Streptococcus bovis showing moderately necrotic and congested mucosa (arrows), (H & E., x100).

11. Experimental trichomoniasis, crop showing vacuolation of mucosal epithelium, H & E., x200.

12. Natural trichomoniasis, crop showing focal submucosal leukocytic aggregations, H & E., x200.

13. Natural trichomoniasis, crop complicated with E.coli showing submucosal edema and hemorrhage, H & E., x200.

14. Naturally complicated trichomoniasis with E.coli and Pasteurella sp., liver showing hydropic degeneration associated with focal replacement of the hepatic parenchyma by radially arranged pericytes around the central vein, H & E., x100.

15. A high power for Fig. 14 to show the radially arranged histiocytes around the central vein, H & E., x200.

16. Naturally complicated trichomoniasis with Pasteurella multocida, liver showing focal replacement of the hepatic parenchyma by radially arranged pericytes around a central vein, H & E.,
17. Natural trichomoniasis complicated with Pasteurella multocida and E.coli, liver showing hydropic degeneration of hepatocytes, H & E., x200.

18. Experimental trichomoniasis, liver showing extensive portal infiltration with leukocytes, H & E., x200.

19. Naturally complicated trichomoniasis with Pasteurella multocida, and Streptococcus bovis, myocardium showing focal hemorrhage, H & E., x200.

20. Naturally complicated trichomoniasis with E.coli, myocardium showing focal replacement of myocytes by mononuclears, H & E., x200.

21. Experimental trichomoniasis, myocardium showing proliferated sarcolemmal nuclei with interstitial edema, H & E., x200.

22. Naturally complicated trichomoniasis with Pasteurella multocida, lung showing congestion and hemorrhage, H & E., x100.

23. Naturally mixed infection with T. gallinae and P. multocida, lung showing focal leukocytic aggregation, H & E., x200.

24. Natural trichomoniasis, brain showing necrosis of large central neurons, H & E., x200.

Table (1): Bacteriological examination for oropharyngeal swabs collected from apparently healthy and diseased pigeons.

<table>
<thead>
<tr>
<th>State of pigeons</th>
<th>Total No. of samples</th>
<th>Positive samples</th>
<th>Single isolates</th>
<th>Mixed isolates</th>
<th>Total positive</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>No.</td>
<td>%</td>
<td>No.</td>
<td>%</td>
</tr>
<tr>
<td>Apparently</td>
<td>40</td>
<td>20</td>
<td>25</td>
<td>8</td>
<td>28.6</td>
</tr>
<tr>
<td>Diseased</td>
<td>80</td>
<td>60</td>
<td>75</td>
<td>20</td>
<td>71.4</td>
</tr>
<tr>
<td>Total</td>
<td>120</td>
<td>80</td>
<td>100</td>
<td>28</td>
<td>100</td>
</tr>
</tbody>
</table>
Table (2): Prevalence of bacterial isolates in the examined pigeons.

<table>
<thead>
<tr>
<th>Isolated bacteria</th>
<th>Total No. of samples</th>
<th>Positive samples</th>
<th>Single isolate</th>
<th>Mixed isolates</th>
<th>No.</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td><em>Pasteurella multocida</em></td>
<td>80</td>
<td>52</td>
<td>18</td>
<td>65</td>
<td>18</td>
<td>64.3</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td><em>P. multocida</em> (12)</td>
<td>16</td>
<td>30.8</td>
</tr>
<tr>
<td><em>Streptococcus bovis</em></td>
<td>80</td>
<td>18</td>
<td>6</td>
<td>22.5</td>
<td>6</td>
<td>21.4</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td><em>P. multocida</em> (14)</td>
<td>21</td>
<td>40.4</td>
</tr>
<tr>
<td><em>E. coli</em></td>
<td>80</td>
<td>10</td>
<td>4</td>
<td>12.5</td>
<td>4</td>
<td>14.3</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td><em>P. multocida</em> (8)</td>
<td>15</td>
<td>28.8</td>
</tr>
<tr>
<td>Total</td>
<td>80</td>
<td>100</td>
<td>28</td>
<td>100</td>
<td>Total</td>
<td>52</td>
</tr>
</tbody>
</table>

Table (3): Sensitivity test for bacterial isolates from the examined pigeons.

<table>
<thead>
<tr>
<th>Antibiotic disc</th>
<th><em>P. multocida</em></th>
<th><em>E. coli</em></th>
<th>Strept. sp.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Avitryl (10 Mg)</td>
<td>+++</td>
<td>+++</td>
<td>++</td>
</tr>
<tr>
<td>Erythromycin (15 Mg)</td>
<td>+++</td>
<td>++</td>
<td>+++</td>
</tr>
<tr>
<td>Gentamicin (10 Mg)</td>
<td>+++</td>
<td>++</td>
<td>++</td>
</tr>
<tr>
<td>Septrin (100 Mg)</td>
<td>+</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>Norfloxacin (10 Mg)</td>
<td>+++</td>
<td>+++</td>
<td>++</td>
</tr>
<tr>
<td>Ampicillin (10 Mg)</td>
<td>++</td>
<td>+++</td>
<td>+++</td>
</tr>
<tr>
<td>Amoxycillin (25 Mg)</td>
<td>+ve</td>
<td>+ve</td>
<td>+ve</td>
</tr>
</tbody>
</table>

+++ Highly sensitive          ++ Moderate sensitive
+ Less sensitive
Table (4): The total isolates of bacterial groups in examined pigeons.

<table>
<thead>
<tr>
<th>Bacterial isolates</th>
<th>Oropharyngeal swabs</th>
<th>Liver</th>
<th>Heart</th>
<th>Lungs</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Apparent Diseases</td>
<td>Apparent Diseases</td>
<td>Apparent Diseases</td>
<td>Apparent Diseases</td>
<td>No.</td>
</tr>
<tr>
<td>P. multocida</td>
<td>14 70</td>
<td>48 80</td>
<td>00 00</td>
<td>35 58</td>
<td>0.0</td>
</tr>
<tr>
<td>St. bovis</td>
<td>5 25</td>
<td>7 17</td>
<td>0.0 0.0</td>
<td>15 25</td>
<td>0.0</td>
</tr>
<tr>
<td>E. coli</td>
<td>1 5</td>
<td>5 3</td>
<td>0.0 0.0</td>
<td>10 17</td>
<td>0.0</td>
</tr>
<tr>
<td>Total</td>
<td>20 100</td>
<td>60 100</td>
<td>0.0 0.0</td>
<td>60 100</td>
<td>0.0</td>
</tr>
</tbody>
</table>

No. = number
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bacterial isolates from os-


دراسات باثولوجية على الإصابة بطفيل الترايكوموناس في الحمام مع الإشارة للبكتريا المصاحبة

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المليلس العربي

في هذا البحث تم تجميع (120) مانعة وعشرون حمامًا (64% سليمة ظاهرية و(36% ميكونية) مصابة بالترايكوموناس عمر شهر من أسواق مختلفة بمحافظة الشرقية وبالفحص الظاهرة للفيروال المصابة لوحظ ظهور علامات الانكماش ونقص الوزن وإسهال أخطر وأعراض ذاتية من المانعة رائحة كريهة والفحص الداخلي لها وجدد نكت متجينة بالتقيف الفيروال البلعومي ووجد على بعضها احتقان بالكبد والرئتين وتم أخذ عينات من التقيف الفيروال البلعومي والكبد والقلب والرئتين وذلك للزم بعض البكتريا التي قد تصاحب مرض الترايكوموناس في الحمام وعينات من الحيوسية والبلعوم والقصة البولانية والمخ وذلل للتقصي الميكروسكوبية.

وقد أظهر هذا العزل بعد عمل الاختبارات الميكروسكوبية للبكتريا المعزولة وتأكيدها بالاختبارات البيوكيميائية عن تواجد ميكروب باستريلا ملتسيديا ذات نسبة 20% ونسبة 10% من الفحص ستريتوكس بوز فر تكيم الفحص 20% وميكون معروفة كولاتي نسبة 10% ونسبة 20% وتتم توحيد ضراعة عينات باستريلا ملتسيديا في الفيروال البلاضاي وللحظت الأعراض الظاهرة والغذاء التشريحيات بينما أظهر اختبار الحساسية للميكروب المعزولة أن الامبيلسيلي والبوتليسي والورفولكساني الاقترب في الأدوار الأكثر فاعليًا ضد الميكروب المعزول. كما ثبت أن الفلافج تأثير فعال في علاج الترايكوموناس.

ويعمل عدوى صناعية بالترايكوموناس لعشرين من الزاغيلل ظهر عليها نفس الأعراض الظاهرة السابقة والفحص الباثولوجي لكل من العدوى الطبية والصناعية لوحظ وجود نخر متجمج وأحيانا نزف تحت الميكروز في البلعوم وتعلو هذه شيديه وفيتوكول والماكروفاك وأحيانا نزف متجمج مصحوب بيكرادي وتعلو ارخصية ونخر متجمج بالقصبة الهوائية بينما لوحظ في الحيوسية نخر متجمج أو زيادة مس وارتخاء أو نزف. بينما لوحظ في الكبد استبدال بورى لخلايا الكبد الجديدة حول الأوعية الدموية وكذلك اضطرابات مائية أو تعلو خلوي شديد للسبيح الكديري.

وبالطلب شهد نزف بين الألياف العصبية مع الألياف السرطالية التي حلل محل بعض الألياف. وبالنسبة للاختناق ونزف والتهاب رئوي وتعلو خلوي ونخر نظر بخلايا بركانج وبخلايا العصبة الكبيرة في قص المخ.

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