Pathological and bacteriological studies on *Clostridium perfringens* infection in kidney of cattle, camel and sheep

By

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**SUMMARY**

In the present study a total of 36, 10 and 39 affected kidneys samples were collected from cattle, camel and sheep respectively from El basatin abattoir for Anaerobic bacteriological and histopathological examination. Also 30 albino guinea pigs were divided into 3 groups for experimental infection. 1st group kept as control, 2nd and 3rd group were injected with 0.3 ml of culture supernatant toxin and I/M with 0.5 ml whole culture of *C. Perfringens* type A and D; respectively which isolated from naturally affected kidneys of cattle. Blood samples were collected from guinea pigs for haematological and biochemical examinations.

Tissue specimens from (lung, heart, spleen, kidney, intestine and brain) were taken for bacteriological and histopathological examination. The obtained results revealed high percentage of Clostridium perfringens which were recovered from naturally clinically infected kidneys of examined cattle (69.4%) followed by sheep (28.2%) then camels (10%).

The toxogenic typing of *C. Perfringens* indicated that most *C. perfringens* isolate were type D. eight isolates (32%) from cattle, one isolate (100%) from camel and six isolates (54.5%) from sheep, while type A isolated from cattle two isolates (8%), 1 isolate (9%) from sheep, while not isolated from camels kidney. Non toxogenic type of *C. perfringens* isolated from affected kidney of cattle and sheep in percentage of (60%), (36.36%) respectively. The histopathological examination of kidney revealed shrinkage in renal glomeruli, hemorrhage, edema in wall of blood vessels, tubular necrosis. The previous lesions were mild in camel, moderate in cattle, more severe in sheep. Concerning to experimental infection reisolation of *C. perfringens* were positive from different organs of guinea pigs. The haematological examination revealed haemolytic anemia in both group of guinea pigs. Total leucocytic count revealed non significant changes while differential count revealed neutrophilia and lymphopenia. Serum biochemical analysis in both groups showed significant increase in liver enzyme, globulin, urea and creatinine. Albumin and A/G ratio revealed significant decrease. Con-
cerning to histopathological examination of both groups showed lung edema, congestion and edema in wall of blood vessel in different organs, necrosis in hepatocyte, haemosidrosis in spleen, thrombus in some blood vessels, shrinkage in some glomeruli while other showed hypercellularity with degeneration and necrosis of renal tubules, there were degeneration in epithelial cells lining the intestinal villi with severe inflammatory reaction were observed in the brain associated with necrosis in the purkenji cells in cerebellum.

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INTRODUCTION

Clostridium perfringens is the causative organism of several diseases of animals and human. It is Gram positive anaerobic, spore-forming bacilli. C. perfringens is naturally present in soil or the intestinal tracts of animal and human. Strains of this species are divided into five types (A, B, C, D and E), all types producing a different set of toxins (McDonel, 1986).

Many ruminants harbor C. perfringens type D in various parts of the alimentary tract. The infection almost follows changes in diet from relatively poor diets to rich diets or overeating in fattening small ruminants or calves and sudden death is the most common symptoms of this infection especially in small ruminants (Radostits et al., 2000 and Gyles and thoen 2004).

C. perfringens type A produce α toxin. This toxin is a phospholipase in nature which is lethal and necrotizing. It causes lysis and disrupting cell membranes leads to cell death. Also causes increase vascular permeability through endothelial damage and necrosis at the tips of villi of intestine (Feldman, 2000).

Epsilon toxin binds to receptors in the luminal surface of the vascular endothelium particularly in the brain, cells lining the loops of Henel, distal convoluted tubules of kidney and hepatic sinusoids, producing degeneration of vascular endothelial cells, allows ion leakage and alteration of fluids dynamics (McDonel, 1986 and Jones et al., 1997).

MATERIAL AND METHODS
(A) Field samples:
A total of 36, 10 and 39 af-
affected kidneys of cattle, camel and sheep; respectively were collected from condemnation room of Bassigni abattoir. Kidney samples were divided into two parts, one part was collected in a plastic bags and transferred in an ice box to the laboratory for anaerobic bacteriological examination, and the second one was immersed in 10% neutral formalin for histopathological examination.

(B) Bacteriological examination:
1. Isolation and identification of C. perfringens from pathologically affected kidneys:
   1 gm of affected kidney tissues were inoculated into cooked meat broth medium and were anaerobically incubated at 37 °C for 48 hr. A loopfull from the inoculated broth was streaked onto 7-10% sheep blood agar plates containing 75mg/lit. neomycin sulphate.

   The blood agar inoculated plates were incubated anaerobically at 37°C for 24 hr. The growing surface colonies which showed catalase negative reaction were picked up in pure form and reinoculated into cooked meat broth for further identification and typing according to Koneman et al. (1992).

2. Determination and typing of toxigenic isolates of C. perfringens:
   Determination of toxigenic isolates of C. perfringens was carried out according to Smith and Holdman (1968). Typing of toxigenic isolates were undertaken using the dermo-necrotic test of albino Guinea pig and serum neutralization test of Swiss mice by injection of culture supernatant in tail vein according to Stern and Batty (1975).

3. Detection of the minimal lethal dose (MLD) of C. perfringens toxins:
   MLD of C. perfringenes type A and D toxins were made by I/V injection in Swiss mice according to Niilo (1985).

C. Experimental Designs:
A total of 30 albino guinea pigs with an average weight of 350-450 gm were kept under observation for 2 weeks before the beginning of the experiment. Guinea pigs were divided into 3 groups each group contain 10 animals. 1st group kept as control, 2nd group and 3rd group respectively injected I/D with 0.3 ml of culture supernatant of C. perfringens type A and type D toxin. Also injected I/M with 0.5 ml whole culture of type A and type D which isolated from affected kidney plus calcium chloride (2.5%) the protocol used as Willis (1977). Animals left overnight.

Samples:
a. Samples were collected from
thigh muscles at the site of injection, heart blood and internal organs (kidney – liver and intestine), reisolation and identification of *C. perfringens* A and D was performed according to Kock's postulate.

b. Blood samples were collected from heart puncture of guinea pigs with anticoagulant dipotassium (EDTA) for haematological examination. Another blood samples were collected without anticoagulant and clear serum was separated and kept at – 20°C for biochemical analysis.

c. After postmortem examination of all died albino guinea pigs, tissue specimens (lung, heart, spleen, liver, kidney, intestine and brain) were collected from each animals and fixed in 10% neutral buffered formalin for histopathological examination.

* Haematological examination:

Blood samples were previously collected with anticoagulant for determination of erythrocytic count (RBCs), haemoglobin concentration (Hb), haematocrit value (PCV), total and differential leucocytic count according to *Feldman et al.* (2000).

* Biochemical analysis:

Serum previously separated were performed for estimation of total protein (TP) according to *Sonnenwith and Jattr* (1980), albumin according to *Drupt* (1974), Alanine aminotransferase (ALT), Aspartate aminotransferase (AST) according to *Reitman and Frankel* (1957), blood urea nitrogen according to *Patton and Crouch* (1977) and serum creatinine according to *Husdan* (1968).

* Histopathological examination:

Tissue specimens were fixed in 10% neutral buffered formalin, processed, paraffin embedded and sectioned at 4-6 µ thickness. Tissue were stained with Hematoxylin and Eosin stain according to *Bancroft et al.* (1994).

* Statistical analysis:

The obtained data were statistically analysed using student T test as outlined by *Snedecor and Cochlran* (1976).

**RESULTS**

**Bacteriological studies:**

In the present studies the numbers and percentages of anaerobic bacterial isolates from naturally clinically infected kidneys (Table 1) showed the high percentage of isolates were recovered from examined kidney of cattle (69.4%) followed by sheep (28.2%) then camels (10%). The microbial identification of anaerobic bacterial organisms isolated from naturally infected kidney proved that *C. perferingens* was the most common isolated anaerobic organisms.

The toxogenic typing of *C.
The results indicated that most *C. perfringens* isolate were type D. 8 isolates (32%) from cattle, 1 isolate (100%) from camel and 6 isolates (54.5%) from sheep, while *C. perfringens* type A isolated from 2 isolates (8%) from cattle, 1 isolate (9%) from sheep. *C. perfringens* type A not isolated from kidney of affected camels. Non toxogenic type of clostridium isolated from affected kidneys of cattle and sheep in percentage of (60%), (36.36%) respectively.

Gross Appearance:
The post mortem examination of the collected kidneys naturally infected by *C. perfringens*: revealed enlarged size, pale in colour and softer in consistency especially in sheep.

Histopathological alterations:
Kidneys of cattle:
Two cases (isolated *C. perfringenes* type A) showed congestion and hypetrophy in the wall of blood vessels with degeneration and necrosis in tubules (Fig.1). While 8 cases (isolated *C. perfringenrs* type D) showed atrophy of the glomeruli, while the renal tubules revealed haemolysed blood in tubular lumen associated with degeneration and necrosis in the lining epithelium of the tubules (Fig. 2).

Kidneys of camel:
One case (isolated *C. perfringenes* type D) showed focal haemorrhage (Fig. 3) with degeneration and necrosis in the renal tubules (Fig. 4).

Kidneys of sheep:
One case (isolated *C. perfringenes* type A) showed homogenous esinophilic transudate in Bawman space with hypercellularity of glomeruli and focal inflammatory cells infiltration in between the tubules (Fig.5). Moreover, 6 cases (isolated *C. pergringenes* type D) showed degeneration and necrosis of the epithelial cells lining the renal tubules associated with haemolysed blood in interstitial tissue as well as inflammatory cell infiltration (Fig.6).

Haematological examination of experimental animals:
Table (3, 4) revealed that the control group showed normal values of comparing with other infected group. Erythrogram (Table 3) revealed significant decrease in total erythrocytic count, haemoglobin concentration and hematocrit values in both group of injected animals with *C. perfringens* type A and D. While blood indices showed non significant changes (normocytic normochromic anemia). Concerning to leukogram (Table 4) showed non significant changes in total leukocytic count,
while the differential count revealed neutrophilia and lymphopenia but eosinophils and monocytes didn't show any changes.

**Serum biochemical analysis of experimental animals:**

Table (5) showed significant increase in liver enzyme (ALT and AST), globulin, urea, creatinine but revealed significant decrease in albumin and A/G ratio. While total protein showed non significant changes in both infected groups with *C. perfringens* type A and D.

**Gross and histopathological examinations of experimental animals:**

Reisolation of microorganism were positive in different organs

The postmortem examination of albino Guinea pigs of experimental infection with *C. perfringens* type A revealed hydrothorax and dark kidney with enlarged, friable liver and congested intestine. While animals infected with type D revealed severe pulmonary edema, soft congested kidney associated with intestinal haemorrhage

The histopathological examination of guinea pigs infected experimentally with *C. perfringens* type A, revealed hyptertrophy and edema in wall of blood vessels of lung with focal inflammatory cells infiltration and emphysema of air alveoli (Fig.7). Perivasular leucocytes inflammatory cells infiltration was observed in the lung (Fig. 8)

Heart revealed congestion in blood vessels accompanied with edema in the vascular wall. There were degenerative changes in the myocardium with lose of myocardial striation.

The central and portal veins showed congestion and haemolysed blood in the vascular lumen with necrosis, vacuolar degeneration in hepatocyte and diffuse kupffer cells proliferation in between (Fig.9).

There were depletion in lymphoid follicles with haemosiderosis in spleen (Fig.10)

Kidney capsule was thick and had haemorrhage with inflammatory cells infiltration (Fig.11). The renal tubules showed degenerative changes associated with congestion in renal blood vessels (Fig.12).

There were dilatation and congestion of the intestinal blood vessels, with haemorrhage, while the epithelial cells lining the villi showed degenerative change with proliferation of goblet cells formation (Fig.13), with severe infiltration of inflammatory cells.

The histopathological examination of guinea pigs infected with *C. perfringens* type D, revealed congestion of the pulmonary blood vessels with haemolysed blood. There was thickening and hypetro-
Inflammatory cells infiltration was observed in the interstitial spaces, with bronchiolar hyperplasia and emphysema in alveoli (Fig. 14).

The myocardium showed congestion in the myocardial blood vessels (Fig. 15).

Liver showed granular and vacuolar degenerations in the hepatocytes with inflammatory cells infiltration in the portal area.

There was depletion in the lymphoid follicles of spleen.

There was atrophy in some glomeruli associated with necrosis in the cells epithelial lining the renal tubules (Fig. 16).

Intestine showed depletion in pyer's patches and infiltration with edema inflammatory cells in lamina propria.

Brain showed congestion of the blood vessels. Necrosis was observed in pyrkenji cells of cerebellum (Fig. 17). Focal gliosis was seen.

Table (1): Distribution of *C. perfringens* among examined kidneys of different animals.

<table>
<thead>
<tr>
<th>Animal species</th>
<th>No. of examined affected kidney</th>
<th>Positive kidney sample for <em>C. perfringens</em></th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cattle</td>
<td>36</td>
<td>25</td>
<td>69.4%</td>
</tr>
<tr>
<td>Camel</td>
<td>10</td>
<td>1</td>
<td>10%</td>
</tr>
<tr>
<td>Sheep</td>
<td>39</td>
<td>11</td>
<td>28.2%</td>
</tr>
</tbody>
</table>
Table (2) Typing of *C. perfringens* isolates from affected kidneys of different species.

<table>
<thead>
<tr>
<th>Examined Kidney of different species</th>
<th>No. of tested isolates</th>
<th>No. of toxogenic isolates and typing</th>
<th>Non toxogenic isolates</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Type (A)</td>
<td>%</td>
</tr>
<tr>
<td>Cattle</td>
<td>25</td>
<td>2</td>
<td>8</td>
</tr>
<tr>
<td>Camel</td>
<td>1</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Sheep</td>
<td>11</td>
<td>1</td>
<td>9</td>
</tr>
</tbody>
</table>

% was calculated according to the total number of tested isolate.

Table (3): Erythrogram of G. pigs infected with *C. perfringens* type A and D (Mean ± S.E.).

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Control</th>
<th>Type A</th>
<th>Type D</th>
</tr>
</thead>
<tbody>
<tr>
<td>RBCs (X 10^6/µ)</td>
<td>6.66 ±0.36</td>
<td>5.05* ±0.37</td>
<td>5.11* ±0.24</td>
</tr>
<tr>
<td>Hb (gm/dl)</td>
<td>13.75 ±0.84</td>
<td>11.32* ±0.37</td>
<td>10.9* ±0.4</td>
</tr>
<tr>
<td>PCV (%)</td>
<td>40.34 ±1.12</td>
<td>32.2* ±1.11</td>
<td>33.00* ±1.58</td>
</tr>
<tr>
<td>MCV (fl)</td>
<td>72.1 ±1.28</td>
<td>73.00 ±2.3</td>
<td>71.11 ±2.11</td>
</tr>
<tr>
<td>MCH (pg)</td>
<td>26.3 ±1.28</td>
<td>23.85 ±1.18</td>
<td>22.16 ±1.85</td>
</tr>
<tr>
<td>MCHC (gm/dl)</td>
<td>34.00 ±1.41</td>
<td>31.47 ±0.98</td>
<td>36.83 ±2.01</td>
</tr>
</tbody>
</table>

* Significant at (P ≤ 0.05)
Table (4): Leucogram of G. piges experimentally infected with *C. perfringens* type A and type D (Mean ±S.E.).

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Control</th>
<th>Type A</th>
<th>Type D</th>
</tr>
</thead>
<tbody>
<tr>
<td>T.L.C (X 10^3/µ)</td>
<td>5.59 ±0.19</td>
<td>5.96 ±0.18</td>
<td>4.90 ±0.27</td>
</tr>
<tr>
<td>Neutrophils (X 10^3/µ)</td>
<td>2.03 ±0.18</td>
<td>4.10* ±0.29</td>
<td>3.02* ±0.15</td>
</tr>
<tr>
<td>Stab (X 10^3/µ)</td>
<td>0.50 ±0.1</td>
<td>0.26 ±0.11</td>
<td>0.30 ±0.1</td>
</tr>
<tr>
<td>Lymphocytes (X 10^3/µ)</td>
<td>3.07 ±0.26</td>
<td>1.07* ±0.17</td>
<td>1.49* ±0.16</td>
</tr>
<tr>
<td>Monocytes (X 10^3/µ)</td>
<td>0.16 ±0.01</td>
<td>0.25 ±0.15</td>
<td>0.45 ±0.13</td>
</tr>
<tr>
<td>Eosinophils (X 10^3/µ)</td>
<td>0.19 ±0.01</td>
<td>0.37 ±0.1</td>
<td>0.33 ±0.13</td>
</tr>
</tbody>
</table>

* Significant at (P ≤ 0.05)
Table (5): Serum biochemical analysis of G. pigs infected with *C. perfringens* type A and type D (Means ±S.E.).

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Control</th>
<th>Type A</th>
<th>Type D</th>
</tr>
</thead>
<tbody>
<tr>
<td>ALT (U/L)</td>
<td>31.80 ± 0.9</td>
<td>36.40* ± 1.2</td>
<td>37.20* ± 0.86</td>
</tr>
<tr>
<td>AST (U/L)</td>
<td>38.40 ± 0.68</td>
<td>41.50* ± 1.3</td>
<td>45.20* ± 0.95</td>
</tr>
<tr>
<td>Urea (gm/dL)</td>
<td>23.60 ± 0.93</td>
<td>59.00* ± 1.4</td>
<td>68.00* ± 1.07</td>
</tr>
<tr>
<td>Creatinine (gm/dL)</td>
<td>0.90 ± 0.08</td>
<td>1.50* ± 0.03</td>
<td>1.70* ± 0.08</td>
</tr>
<tr>
<td>Total protein (gm/dL)</td>
<td>5.30 ± 0.23</td>
<td>5.16 ± 0.12</td>
<td>5.18 ± 0.11</td>
</tr>
<tr>
<td>Albumin (gm/dL)</td>
<td>3.17 ± 0.14</td>
<td>2.60* ± 0.11</td>
<td>2.30* ± 0.2</td>
</tr>
<tr>
<td>Globulin (gm/dL)</td>
<td>2.13 ± 0.10</td>
<td>2.48* ± 0.11</td>
<td>2.68* ± 0.15</td>
</tr>
<tr>
<td>A/G ratio</td>
<td>1.38 ± 0.11</td>
<td>1.04* ± 0.09</td>
<td>0.85* ± 0.08</td>
</tr>
</tbody>
</table>

* Significant at (P ≤ 0.05)
Fig. (1): Kidney of cattle showing hypertrophy in wall of blood vessels with degeneration and necrosis in renal tubule. H&E (x 100)

Fig. (2): Kidney of cattle showing atrophy in glomeruli with haemolyzed blood in tubular lumen and degeneration as well as necrosis in tubule. H&E (x 100)

Fig. (3): Kidney of camel showing haemorrhage in interstitial tissue. H&E (x4)

Fig. (4): Kidney of camel showing degeneration and necrosis of renal tubule. H&E (x 200)
Fig. (5): Kidney of sheep showing hypercellularity in glomeruli with pink exudate in Bowmans space and inflammatory cells infiltration in between. H&E (x200)

Fig. (6): Kidney of sheep showing haemolysed blood in interstitial tissue with necrosis in renal tubule associated with inflammatory cells. H&E (x 200)

Fig. (7): Lung of G.pigs infected with *C.perfringens* type A showing edema and hypertrophy in the wall of blood vessels, haemorrhage with presence of alveolar emphysema and inflammatory cells infiltration. H&E (x200)

Fig. (8): Lung of G.pigs infected with *C.perfringens* type A showing perivascular inflammatory cells infiltration. H&E (x 200)
Fig. (9): Liver of G.pigs infected with *C. perfringens* type A showing congestion in central and portal vein, vacuolar degeneration in hepatocyte with diffuse kupffer cells proliferation. H&E (x200)

Fig. (10): Spleen of G.pigs infected with *C. perfringens* type A showing lymphoid depletion and haemosidrosis. H&E (x100)

Fig. (11): Kidney of G.pigs infected with *C. perfringens* type A showing thickening of renal capsule, haemorrhage and inflammatory cells infiltration. H&E (x200).

Fig. (12): Kidney of G.pigs infected with *C. perfringens* type A showing congestion in renal blood vessels and degeneration in tubule. H&E (x200)

Fig. (13): Intestine of G.pigs infected with *C. perfringens* type D showing haemorrhage, degeneration in epithelial lining and with goblet cells formation. H&E (x200)
Fig. (14): Lung of G.pigs infected with *C.perfringens* type D showing hyperplastic proliferation of epithelial lining the bronchiol, with inflammatory cells infiltration and compensatory emphysema. H&E (x200)

Fig. (15): Heart of G.pigs infected with *C.perfringens* type D showing congestion myocardial blood vessels. H&E (x200)

Fig. (16): Kidney of G.pigs infected with *C.perfringens* type D showing atrophy in some glomeruli with, degeneration and necrosis of renal tubule. H&E (x200)

Fig. (17): Brain of G.pigs infected with *C.perfringens* type D showing necrosis of pyrkingye cells. H&E (x200)
DISCUSSION

Clostridium perfringens is causative agents of enterotoxemia in farm animals. Organisms are normally inhabitant in alimentary tract and under certain conditions, the organism proliferate rapidly in the intestine and produce lethal quantities of exotoxin (Radostits et al., 2000). C. perfringens toxins cause rapid and severe tissue damage and death of the body cells (Kumar et al., 1997).

In the present study C. perfringens was the most anaerobic bacterial isolates recovered from naturally affected kidneys of cattle, camel and sheep. They were isolated at rate of (69.4%), (10%) and (28.2%) respectively. These results were nearly similar to those obtained by El-Amrousi et al. (1986); Abdurahman and Borbstein (1991) and El-Naenaeey (2000). However, the identification and typing of organisms revealed isolation of C. perfringens type D at a rate of (100%) from affected camel's kidney, the same results recorded by Abdurahman and Borbstein (1991) and Hala (1996); (54.5%) from sheep, nearly the same results obtained by Peter (1998) and (32%) from kidneys of cattle, this recorded by Assis and Uzal (2002). While C. perfringens type A isolated at rate of (9%), (8%) from affected kidneys of sheep and cattle. Nearly the same results obtained by Mariano et al. (2005).

In the present study the experimentally infected guinea pigs with C. perfringens type A and D revealed in their haematological examination, decrease in erythrocytic count, Hb concentration and PCV values. While blood indices didn't show any changes. These results observed in hemolytic type of anemia. This results attributed to action of α toxin which causes break down of phospholipids of erythrocytes membrane and cause hemolysis by damaging circulating erythrocytes. Gardner (1973) recorded that ratio of Hb to haematocrit value remain constant. Coles (1986) recorded hemolytic anemia associated with excessive destruction of erythrocyte may caused by variety of diseases like bacterial infection the most common bacterial diseases clostridium and leptospirosis. Also, Topley and Wilsons (1998) reported that C. perfringens bacteremia is commonly associated with intravascular haemolysis. Concerning to leucogram revealed neutrophilia and lymphopenia, this results common in acute inflammatory response because inflammatory mediators stimulate movement of neutrophil during acute inflammation, also stimulate movement of lymphocytes from blood to the inflamed tissue and lymphoid tissues The severity of lymphopenia reflect the severity of
systemic inflammatory response. (Imhof and Dunon, 1995).

Serum biochemical results recorded significant increase in liver enzyme activities, urea, creatinine and globulin. Also significant decrease in albumin and A/G ratio. Nearly same results were obtained by Gardner (1973); Amany and Morsi (1995). The previous results were confirmed by microscopic examination which revealed liver and kidney damage. C. perfringens cause myonecrosis which causes increase in the level of urea. There is no changes observed in the levels of total protein.

Regarding to histopathological finding of cattle and camel kidneys revealed edema in wall of renal blood vessel and shrinkage in glomeruli. While the kidneys of sheep showed the previous results in addition to severe haemorrhage, edema in glomeruli and thrombus formation in some blood vessels with necrosis of cells lining the renal tubules. The same results obtained by Gardner (1973); Tamai et al. (2003); Tammy (2004) and Uzal et al. (2004). The previous changes due to exotoxin of C. perfringens which cause haemolysis of erythrocyte, aggregation of platelets. Also, increase vascular permeability through endothelial cell damage which leads to edema and necrosis (Feldman et al., 2000).

Concerning to postmortem examination of infected Guinea pigs which revealed hydrothorax and pleural effusion, pulmonary edema, haemorrhagic and soft kidney as well as friable and enlarged liver. Similar results recorded by Gardner (1973); EL Bardisy et al. (1995); Mariano and Uzal (1998); Uzal et al. (2002) and Adamson et al. (2005).

Microscopical examination of guinea pigs infected experimentally with C. perfringens, pulmonary edema, Myocardial necrosis, thrombus in some hepatic blood vessels. While others filled with haemolysed blood, mild to severe vacuolar degeneration, necrosis of some hepatocytes with activation of Kupffer cells. Necrosis in epithelial lining intestine and increase goblet cells, depletion of pyer's patches, haemosidrosis and depletion in lymphoid follicle of spleen, degeneration and necrosis in renal tubules, atrophy in some glomeruli, and loss of granular layer of brain were evident. The previous findings were obtained by Finnie (1984); EL Bardisy et al. (1995), Uzal et al. (2002); Uzal et al. (2004) and Mariano et al. (2005). Topley and Wilsons (1998) recorded that injection of toxic filtrates into animals revealed edema, necrosis inaddition to capillary and venous thrombosis. Niilo
reported epsilon toxin bind to receptors in the luminal surface of the vascular endothelium particularly in the brain, renal tubules and hepatic sinusoids cause degeneration of vascular endothelium and alteration of fluid dynamic and leakage of plasma protein.

OIE (2004) recorded α toxin is a necrotizing toxin produced by all five strain of C. perfringens which cause vascular leakage, hemolysis and liver damage. Mlyashiro et al. (2007) recorded C. perfringens type D produced epsilon toxin which resistant to digestive enzyme, these enzyme convert freshly secreted less active protoxin into fully toxic large amounts of epsilon toxin produced in gut, absorbed into systemic circulation increases the capillary permeability in many organs and tissues causes gastroenteritis, extensive renal damage, multi and systemic haemorrhage in different organs.

It is concluded that naturally kidney of sheep more affected and highly susceptible to Clostridium perfringens type D. Experimentally, Clostridium perfringens type D is more affected kidney while type A is mainly affected intestine. These results were confirmed by alterations estimated in haematological, biochemical and histopathological studies.

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دراسات باثولوجية وبكتيرية عن الإصابة بميكروب الكلوستريديم بفرنجزنّ على الكلى المشاع والأنفام

هبة الشريفي حمزّة*، هالة أحمد محمود عامر**
قسم الباثولوجيا، قسم البكتيرولوجيا - معهد بحوث صحة الحيوان

* قسم الباثولوجيا

الملخص العربي

اجريت هذه الدراسة على عدد 360 ذكر و39 كلي مصابين من الأبقار والجمال والأغنام على التوالي من مجزر الأبقار في محافظة القاهرة، وذلك لدراسة البكتيرية والباثولوجية. أيضاً تم إجراء تجربة على عدد 30 ذكر و39 غنم من الأبقار لتقييم صحة الكلى في هذه المجموعة. تم جمع نتائج الدراسة وتصنيفها في طبقتين: A و D. ينتمي البقولية A إلى نوع M.1000 وأيضًا تم التقييم المعدل على 050 في الميكروب والذي تم عزله من الكلى المصابية.

وقد أوضح 결과 الدراسة البكتيرية أن نسبة الإصابة في الكلى بميكروب الكلوستريديم بفرنجزنّ كانت في ذكر الأبقار بنسبة 28.2% وأيضًا نسبة 20% ومثل هذه النتائج يمكن استنباطها عن نسبة 23% والجمال بنسبة 10% وأيضًا النتائج المذكورة تشير إلى أن نوع D تم نجد 7% تم تصنيفه في الأبقار بنسبة 8% والأغنام بنسبة 6% هذا النوع لم يتم عزله من الكلى المصابية في الحمام. أوضح الدراسة أيضاً أن ميكروب الكلوستريديم بفرنجزنّ غير البكتيرية فعلى الرغم من أن الكلى المصابية في الأبقار والأغنام بنسبة 40% ونسبة 26% على التوالي تم إعادة عزل وتصنيف الميكروب من الأعضاء المختلفة للخلايا الغنية عينة المصابية تجريبياً بالميكروب المزعول من الكلى المصابية.

أوضح الدراسة فحص الدم إلى وجود إتيا ناتجة عن تكبير كرات الدم الحمراء مع عدم تغير في المعد الكلى للكرات الدم الحمراء زيادة في خلايا الليفوسيرت. أوضح الدراسة الكيميائية لمستخلص الدم إلى زيادة في النزولات الكبد والجلويولين والبيرويا الكرياتينين وقلة في الألبومين. أظهرت الدراسة الاستنتاجية إلى وجود احتقان وأيضًا في الأوعية الدموية للأنسجة المختلفة ووجود أمبوبية الكبد. ونجد صحة الميكروبيدريين بالرئة واستحالة بالخلايا المنفعة للاصابة وارتشاج بالخلايا الالتهابية ونجد بلطاط في بعض الأوعية الدموية الكلى مع ارتشاج بالخلايا الالتهابية فيهما ونجد تكزّر في الطبقة الحبيبية.

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