



PATHOLOGICAL STUDIES ON CAPRINE CLOSTRIDIUM PERFRINGENS COMPLICATED WITH SOME PARASITIC AND VIRAL INFECTION

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ABSTRACT

An outbreak of *C. perfringens* in complication with other pathogens was investigated in mixed herd of sheep and goat at Al Gharbiyah provinces, Egypt during late March 2011 to July 2012. Bacteriological and parasitological examination reflects the presence of *C. perfringens* in all examined flocks. *Eimeria* was detected in suckling kids less than 3 months of age in one flock. Meanwhile the clinical findings, post-mortem and histopathological examination suggestive *PPR* in suckling kids less than 3 months of age in other two flocks. The morbidity and mortality rate in suckling kids infected with *cl. Perfringens* in complication with *PPR* in the flocks were (92%,94%), (32%,94%) respectively. on clinical examination big head, pyrexia, sneezing, coughing, lacrimation, serous-mucopurulent nasal discharge, frothy mouth, diarrhea and conjunctivitis were reported. Terminal convulsion, opisthotonus and death were observed. The gross and histopathological findings are reported as erosive, ulcerative stomatitis and necrotic enteritis. Presence of inclusion bodies and syncytial cell formation was pronounced. The pulmonary lesions were necrotic bronchitis, bronchiolitis and broncho-interstitial pneumonia, with edema and pleuritis. Lymphoid tissue showed extensive hemorrhage, edema and acute necrosis, depletion of the lymphoid cells and macrophage cell infiltration in the germinal centers of sinusoid. Interstitial myocarditis, hyalinization of the myocytes and hemorrhages were pronounced. While in suckling Damascene kids, less than 3 months of age, infected with *C. perfringens* in complication with *coccidiosis*, out of 14 Damascene goat 12 developed clinical signs (84%), dead 6 (43%). Clinical findings are big head, pyrexia and diarrhea. Gross findings revealed, congestion, edema and wide spread petechial hemorrhages and ecchymosis of mucosa of intestine. Histopathological findings include necrotic enteritis with large number of necrosed epithelial cells and hyperplasia of remaining epithelium in which *Eimeria* in different stages of schizogony.

KEY WORDS: *C. perfringens*, Goat, *Eimeria*, Histopathological, Diarrhea.

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1. INTRODUCTION

Caprine enterotoxaemia is caused primarily by *C. perfringens* type D, which produces both α , β , and ϵ toxins, with ϵ toxin being the main virulence factor. *C. perfringens* type C producing α , and β toxins, has occasionally been associated with

enterotoxaemia, particularly in young kids [32]. *C. perfringens* type B producing α , β and ϵ toxins, rarely B contributes to disease in goats. *C. perfringens* type A producing α toxin, is a common isolate from cases of caprine enterotoxaemia [10]. Caprine

Enterotoxaemia characterized by abdominal pain, bloat, mild to severe (blood-tinged to bloody) diarrhea and recumbancy were seen [16, 36]. In some animals nervous symptoms were observed, while in other cases death was the only clinical sign observed [13, 14]. The leading lesion in goats with Clostridial enterotoxaemia is bloody and fibrinous inflammation of the entire digestive tract, especially of the intestinal mucosa in the ileum and in the colon. A pulmonary edema, and often a Hydropericardium [14, 16]. [5, 37, 38] reported that histopathological changes in caprine enterotoxemia are not specific but can be highly suggestive. The changes consist of severe, regionally extensive, necro-haemorrhagic, fibrinous enteritis with intralesional bacteria detected. The main symptoms of caprine coccidiosis are poor coat, anemia, diarrhea, weight loss, dehydration, and mortality of untreated kids. [15]. [27] mentioned that the faeces are watery with clumps of mucus and colour changes from brown to yellowish mucoid. The pathological picture in small ruminants is that of a catarrhal enteritis which is congestive and more or less haemorrhagic with mucus and fibrin. The serosa of the intestines was pale and the gut was distended with gas. The lumen of the intestines contained reddish bloody content. [1, 27]. The histopathological lesions showed loss of surface epithelial cells and villous atrophy associated with first generation schizonts, and crypt destruction or hyperplasia associated with gamonts [12, 27]. The clinical signs of Peste des petits ruminants (PPR) included fever, depression, and anorexia, serous to mucopurulent nasal discharge, erosions and ulcerations in the buccal cavity, diarrhoea and respiratory distress. Frothy salivation and conjunctivitis were also found. [29, 30]. Sixty three percent (63%) morbidity and thirty two percent (32%) mortality were noted being higher in kids as compared to young and adult one while Goats under one year of age had highest morbidity and mortality with typical

signs and lesions of PPR. (51 %) developed clinical disease, of which (13.5 %) died. Sixty three percent (63) % [29]. The pathological lesions caused by PPR infection includes: stomatitis with erosions, ulcerations and necrotic lesions in the buccal cavity, severe congestion throughout the intestinal tract, zebra stripes in caeco-colic junction, were the main gross lesions in the digestive system [29]. The heart was congested and contained white necrotic patches. The spleen was enlarged and showed petechial to ecchymotic haemorrhages. The kidneys were highly congested. Lungs were consolidated [9, 30]. The histopathological findings were non-purulent enteritis with congestion and infiltration of large mononuclear cells in the lamina propria and loss of lining epithelium. In the lung, bronchopneumonia, syncytial cells, giant cells and intracytoplasmic inclusion bodies in bronchiolar epithelium. Lymph nodes are necrotic and haemorrhagic. Depletion of lymphocytes and infiltration of macrophages were in lymph nodes [3, 6, and 29].

The present investigation hence was undertaken to study the major causes of complication to clostridium perfringens infection in goats, with description of gross and histopathological changes accompanied the outbreaks of complicated clostridium perfringens infections in goats.

2. MATERIALS AND METHODS

2.1. Animals and sampling

During late March 2011 July 2012 a disease outbreaks were reported, in Damascene goat flocks showed high mortality rate. Most of the affected kids showed big head, pyrexia, oculonasal discharge, diarrhea, and respiratory manifestation. Samples taken from morbund, dead kids. Specimens from skin, intestine, lung, kidneys, spleen, lymph node, tongue salivary gland. Samples were divided into two parts, one part was collected in plastic bags and transferred in an icebox to

the laboratory for anaerobic bacteriological examination, and the second one was immersed in 10% neutral formalin for histopathological examination.

2.2. Postmortem examination

Postmortem examination of naturally infected goats was carried out and the gross picture of the internal organs of these goats was recorded .

2.3. Bacteriological examination

Tissue samples (a total 45 samples) for anaerobic examinations were taken from liver, intestine, kidney and spleen. Samples were cultured anaerobically on cooked meat media [40]; sheep blood agar and neomycin sulphate blood agar media [8]. The same organs were inoculated directly onto DAS medium, MConkey bile salt agar, and blood agar and incubated aerobically at 37°C for 24-48 hrs. The suspected growing colonies were studied for morphological and biochemical characters according to [21]. Determination of toxigenic isolates of *C. perfringens* was carried out according to [31]. 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 [35].

2.4. Parasitological examination

Individual fecal samples were collected from all kids and kept in sterile plastic bottles for parasitological examination using centrifugal floatation technique for the detection of oocysts according to [33].

Histopathological examination: specimens were preserved in 10 %buffered neutral formalin. After proper fixation, the specimens were dehydrated in ascending grades of ethyl alcohol, then cleared in xylol and embedded in paraffin .Thin tissue sections about 5 microns in thickness were prepared and stained with hematoxylin and

eosin stain for general microscopic examination according to [7].

3. RESULTS

Natural *Clostridial perfringens* infection in complication with viral infection:

1. Bacteriological examination: *C. perfringens* type D had been isolated.
2. Clinical signs, gross lesion, histopathological examination suggestive of PPR

A-Clinical signs, morbidity and mortality rate:

Among affected Flock No1, No2; The morbidity 92 %, 94% respectively and the mortality of affected kids 32 %, 94% respectively. The prominent clinical signs were big head, fever, stomatitis, diarrhoea and dyspnea. The big head was characterized by swelling of the face and around the eyes and submandibular together with swelling of the neck. frothy of the mouth and grinding of the teeth had been noticed. Pyrexia (40.5c to 42c) persist till the animals died. Diarrhoea was initially yellow –green and pasty but rapidly became watery and mucoid, with shreds of bowl mucosa and blood which soiled hind quarters. Intermittent paddling of the limbs had been seen. Terminal convulsions often with opisthotonus represented by limbs rigidity were seen. Apparent blindness, coma, cold extremities and death were also observed. Discrete areas of erosion and ulceration on gingival, and /or anterior dorsal part of the tongue with froth salivation had been observed. A froth discharge from nostrils was seen. A serous nasal discharge was progressively turned into mucopurulent discharge. Dried exudates partially occluded the external nares. The eyes showed copious ocular discharge and matting of the facial hairs when sneezing and coughing. Hard crust on surrounding areas of nasal and ocular region had been observed. Conjunctivitis was recorded with lacrimal discharge initially serous, lead to clumping



Figure 1.



Figure 2.



Figure 3.



Figure 4.



Figure 5.

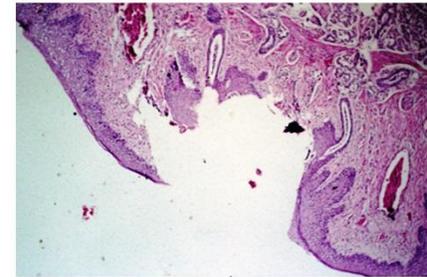


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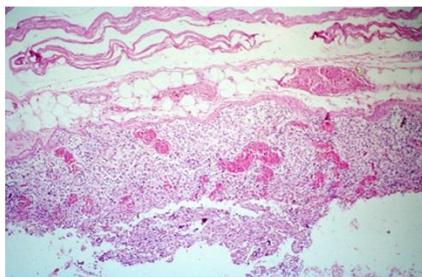


Figure 7.

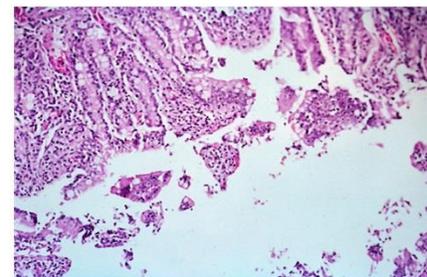


Figure 8.

Fig (1) kids (< 3month of age) naturally infected with *C. perfringens* in complication with PPR showing encrustation in the medial canthus of the eye and sticky eyelids. Fig (2) caecum of kid (< 3month of age) naturally infected with *C. perfringens* in complication with PPR showing dark red areas with streaks on the mucosa with fibrino-necrotic membrane on surface . Fig (3) lung of kid (< 3month of age) naturally infected with *C. perfringens* in complication with PPR showing large dark red patches. Fig (4) submandibular region of kid (< 3month of age) naturally infected with *C. perfringens* in complication with PPR showing A-enlarged and oedematous lymph node B- enlarged and oedematous and dark red cut section of tonsils. Fig (5) tongue of kid (< 3month of age) naturally infected with *C. perfringens* in complication with PPR showing vesiculation, which infiltrated with few number of neutrophils. H&E stain X 200. Fig (6) tongue of kid (< 3month of age) naturally infected with *C. perfringens* in complication with PPR showing erosion. H&E stain X 100. Fig (7) intestine of kid (< 3month of age) naturally infected with *C. perfringens* in complication with PPR showing complete necrosis of intestinal epithelium with Congested blood vessels and capillaries and leukocytic infiltration. H&E stain X 100. Fig (8) intestine of kid (< 3month of age) naturally infected with *C. perfringens* in complication with PPR showing formation of syncytial cells. H&E stain X 200.

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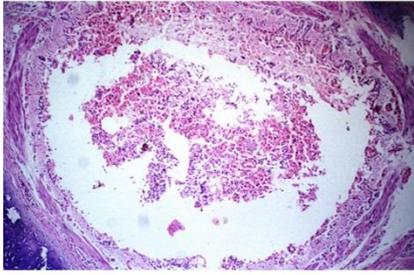


Figure 9.

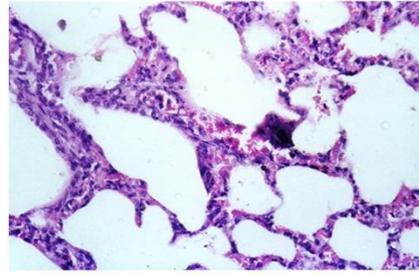


Figure 10.

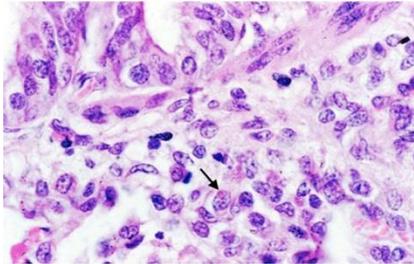


Figure 11.

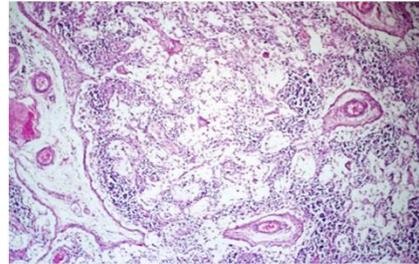


Figure 12.

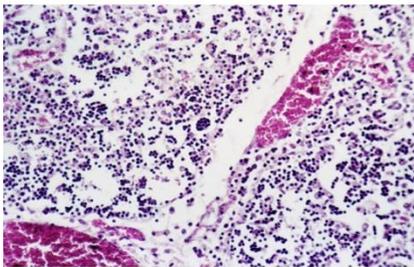


Figure 13.

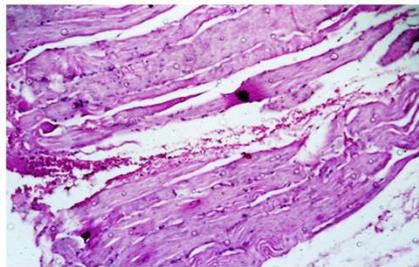


Figure 14.



Figure 15.

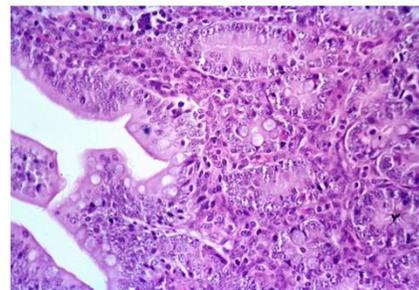


Figure 16.

Fig (9) lung of kid (<3month of age) naturally infected with *C. perfriengens* in complication with PPR showing A-epithelium of bronchi destructed, erosion of bronchi with ulceration. B- Lumen filled with desquamated epithelium and erythrocytes. H&E stain X 100. Fig (10) lung of kid (<3month of age) naturally infected with *C. perfriengens* in complication with PPR showing proliferation of type 11 pneumocytes. H&E stain X 400. Fig (11) lung of kid (<3month of age) naturally infected with *C. perfriengens* in complication with PPR showing eosinophilic intranuclear inclusion bodies. H&E stain X 400. Fig (12) lymph node of kid (<3month of age) naturally infected with *C. perfriengens* in complication with PPR showing lymphoid depletion. H&E stain X 100. Fig (13) lymph node of kid (<3month of age) naturally infected with *C. perfriengens* in complication with PPR showing lymphoid depletion with syncytial cells. H&E stain X 400. Fig (14) heart of kid (<3month of age) naturally infected with *C. perfriengens* in complication with PPR showing area of hemorrhage and edema. H&E stain X 200. Fig (15) intestine of kid (<3 month of age) naturally infected with *C. perfriengens* in complication with Coccidiosis showing serosa of intestine hyperemic and edematous with wide spread petechial hemorrhages and ecchymosis in the mucosa. Fig (16) intestine of kid (<3 month of age) naturally infected with *C. perfriengens* in complication with suspicion of Coccidiosis showing some stage of schizogony in the intestinal villi. H&E stain X 400.

and sticky eye lashes which progressively turned into mucopurulent; resulting in sticky eyelids. Crusty exudate over the conjunctiva had been noticed. Conjunctival encrustation in the medial canthus of the eye was seen (Fig.1).

B-Macroscope findings:

Big head was characterized by massive subcutaneous edema of the face, around the eye, submandibular and neck areas. The presence of yellowish fluid in the thoracic cavity and hydroperitonium were commonly found. The buccal cavity mucosa was congested with presence of discrete minor erosions and ulcers on the hard palate, gums and the dorsum of the tongue. The most consistent gross changes were seen in small and large intestine which were filled with watery contents, blood and fibrin clots. The serosa and subserosa of the distal part of the small intestine, colon and caecum revealed dark red area, edema, and streaks of haemorrhages. Similar lesions were also seen in the mucosa of the caecum and localized areas of the colon. Overlying these areas, there was a variably severe fibrino-necrotic membrane and fibrino-mucinous casts in the colonic and caecum lumen (Fig.2). Trachea, bronchi and bronchioles were filled with mucopurulent froth. The lung revealed either large dark red patchy areas consolidated or small focal areas of dark red colour and firm in consistency. The interlobular septa was prominent and cut sections of lungs showed large quantities of frothy exudate indicative of pulmonary oedema. Lymph nodes especially mediastinal lymph nodes and retropharyngeal were congested, edematous with cut surface revealed dark red areas. Tonsils were enlarged, congested, oedematous and revealed area of haemorrhages on cut surface (Fig.4). Spleen was enlarged, congested and oedematous together with petechial and ecchymotic haemorrhages. Kidney revealed subcapsular petechiae with focal areas of hemorrhages in

the cortex. Linear haemorrhages had been noticed in the renal medulla. Heart with Pericardial effusion had been noticed. Epicardial petechiae and congestion with ecchymotic haemorrhages were found on myocardium.

C-Microscopical findings:

Skin:

The epidermal lesions of the skin were acanthosis, ballooning degeneration, hyperkeratosis and parakeratosis in some areas was noticed. Subepithelial and subcutaneous tissue were edematous, congested and infiltrated with leucocytes mainly lymphocytes, macrophages and few neutrophils together with focal areas of haemorrhages. The dermal and subcutaneous blood vessels showed severe congestion and thrombosis. The blood vessels were congested with its wall infiltrated with massive aggregation of erythrocytes. Some congested blood vessels showed perivascular oedema and focal aggregation of leucocytes in its lumen. Occasionally, blood vessel wall infiltrated with massive number of leucocytes mainly mononuclear cells. Subcutaneous muscles showed congestion, oedema, focal areas of haemorrhages with few numbers of mononuclear leucocytes infiltration, hyaline degeneration and necrosis. Focal aggregation of leukocytes mainly mononuclear was pronounced in some cases.

Tongue:

The epithelial cells of tongue mucosa revealed acanthosis, ballooning degeneration, hyperkeratosis, multiple vesicles and parakeratosis; some vesicles contained few numbers of neutrophils (Fig.5). In area of severe degeneration epithelial cells contained amorphous eosinophilic intranuclear inclusion bodies. There was marked erosions and ulcers of the tongue mucosa with neutrophil cell, lymphocyte macrophage infiltration (Fig.6). Subepithelial tissue was

congested, edematous and infiltration with few numbers of mononuclear cells together with perivascular oedema.

Sublingual salivary gland:

Sublingual salivary glands showed focal areas of degeneration and necrosis, leucocytic cell infiltration mainly mononuclear cell and desquamation of the lining cells.

Intestines:

Histopathological finding revealed erosive and ulcerative lesions in gastrointestinal tract. Mucosal hemorrhages were observed along with congested blood vessels. There was coagulative necrosis of the superficial mucosa with patchy coagulative necrosis of intestinal epithelium, congested blood vessels and capillaries and leukocytic cell infiltration (Fig.7). Syncytial cell in villi and in lumen of intestinal crypts together with coagulative necrosis of crypts were pronounced (Fig.8). Eosinophilic intracytoplasmic hyalinized bodies in the intestinal epithelium and crypts of Lieberkuhn. Massive leucocytic cells infiltration mainly lymphocytes, macrophage and plasma cells were pronounced in the lamina propria and submucosa. Submucosal blood vessels revealed hyperplasia in endothelial cells (endotheliosis). Lymphoid depletion with marked areas of necrosis was evident in the Peyer's patches. Tunica muscularis revealed intermuscular edema, congestion with leucocytic cell infiltration. The predominant histopathological finding in the colon and caecum consisted of coagulative necrosis of the most intestinal epithelium. Severe congestion and hemorrhages in the superficial one third of lamina propria, necrosis with leucocytic cell infiltration and linear hemorrhages in the submucosa were observed. Marked submucosal edema and hemorrhages had been seen.

Lung:

Focal degeneration, necrosis and ulceration of bronchial epithelium were conspicuous with lumen filled with desquamated epithelium and erythrocytes (Fig.9). Recovering epithelial cells were often hyperplastic and irregular with normal cuboidal lining cells with massive perivascular oedema and haemorrhages was seen. Alveoli adjacent to affected bronchioles were also altered with marked hyperplasia of type 11 pneumocytes (Fig.10). In more severely affected lungs, foci of broncho-interstitial pneumonia reaction coalesced to form area of consolidated pulmonary parenchyma. Broncho-interstitial pneumonia represented by proliferation of bronchiolar lining epithelium, intense diffusion and aggregation of mononuclear cells mainly lymphocytes, macrophages in its wall and peribronchial. Alveolar changes were characterized by presence of alveolar macrophages and multinucleated syncytial cells in the alveolar lumen with presence of intranuclear inclusion bodies (Fig.11). Distension and dilatation of the alveoli with oedema, congestion of the alveolar capillaries with infiltration of mononuclear cells in the alveoli along with haemorrhages and hyalinized membrane. Thickened interalveolar septa caused by the infiltration of erythrocyte, mononuclear cells were evident. The pleura was thickened with oedema fibrin and leucocytes.

Lymph nodes:

The most conspicuous histopathological finding in lymph nodes particularly retropharyngeal and mediastinal lymph nodes were lymphoid depletion (Fig.12). In some cases, multifocal areas of necrosis, in addition to lymphocytes with fragmented nuclei (apoptotic cells) were seen. Syncytial cells and intranuclear inclusion bodies were seen (Fig.13). Lymph node was severely congested with hemorrhages involved entire lymphoid follicle.

Spleen:

The most distinguishing feature was necrosis of white pulp with depletion of lymphoid cells. Massive haemorrhages had been noticed. Lymphocytes with fragmented nuclei (apoptotic cells) were seen. Splenitis was evident with splenic capsule and trabeculae revealed edema and infiltrated with leucocytes mainly mononuclear. The spleen infiltrated with basophilic clusters of bacteria. Massive haemorrhages had been noticed.

Tonsil:

The tonsil was congested and revealed focal area of hemorrhages and necrosis of lymphocytes.

Kidney:

The renal tubules revealed degenerative changes with areas of severe coagulative necrosis particularly periglomerular and in the renal medulla had been observed. Multifocal areas of haemorrhages were pronounced in the renal cortex together with linear haemorrhages in-between the renal collecting tubules. Glomerular tuft showed hypercellularity and congestion with periglomerular infiltration of a few number of leucocytes. In the renal hilus syncytial cell was noticed. The renal blood vessels were congested with air emboli had been observed. In addition to hyperplasia of its endothelial lining together with hypertrophy of blood vessels wall.

Heart:

Cardiac muscle showed edema, hyaline degeneration and focal area of haemorrhages (Fig14).

Natural *Clostridial perfriengens* infection in complication with Coccidiosis:

Clinical signs, morbidity and mortality rate:

Among affected flock the morbidity rate % and the mortality of affected kids up to %.

Fever, edema and diarrhea were the most consistently clinical signs. Body temperature rise up to 42C. Big head resulted from edema of the face, around eyes and submandible, together with edema of the neck had been noticed. Crusty exudate in the medial canthus of the eye was seen. Diarrhea was the most consistently clinical signs. The first sign is softness of unpeleted feces, but rapidly becomes watery and mucoid with blood. Hair of the tail, perineum and medial aspects of the hind limbs becomes soiled and may attract fly strike. Kids became dehydrated and weak. Depression, inactivity, incoordination and recumbancy, followed by intermittent paddling of the limbs, bleating terminate periodic muscular tremors and transient convulsions, opisthotonus coma and death.

Macroscopical findings:

Primarily the serosa of some segments of the proximal small intestine, colon and caecum was hyperemic and edematous with wide spread petechial hemorrhages and ecchymosis in the mucosa. Their contents revealed watery fluid with multiple blood clots and distended with gases. The lung showed multiple focal intra alveolar edema.

Microscopical findings:

The intestine was congested, Haemorrhagic and desquamated epithelium in its Lumina was pronounced. Multifocal coagulative necrosis of the superficial mucosa was also observed. Diffuse infiltration of the lamina propria of the intestine by lymphocytes, plasma cells and few eosinophils and neutrophils. Large number of the epithelial cells was necrosed, together with hyperplasia of the remaining epithelium which represented by large long papillary folds in which *coccidia* in different stages of schizogony are most numerous. The alveoli and interlobular septa frilled with homogenous eosinophilic fluid.

4. DISCUSSION

In this study, morbidity rate 92% and mortality 32% of *C. perfringens* infection in combination of suspicion of PPR among kids which were also reported in goats. Results in our study higher than mentioned by [4, 9, 26, 29] due to *C. perfringens* infection in combination with PPR. In present outbreak the clinical findings in flock under observation, of *C. Perfringens* infection in combination with suspicion of PPR among kids, the onset of the disease was seen to be sudden with pyrexia, big head, intense oculonasal discharge, salivation, lacrimation, cough, diarrhoea, terminal convulsion, opisthotonus and death together with conjunctivitis. It has been informed in many studies in goats [9, 20, and 41]. In this study the clinical findings severe than described by previous authors due to mixed infection in our study. The clinical signs reported in our study in a partial agreement with those previously mentioned [2, 4, 29, and 30]. Microvascular endothelial damage by the *cl. perferingens* toxins and PPRv appear to be fundamental cause of sub cutaneous oedema of the face, around eyes, submandibular and neck. In present study, lesions in the buccal cavity and intestinal lesions were characterized by erosive and ulcerative stomatitis; fibrino-necrotic enteritis and the presence of inclusion bodies and syncytial cell formation are pathognomonic. Distension of intestine with gases as well as filled with watery contents, blood and fibrin clots. The serosa and subserosa of the distal parts of small intestine, colon and caecum revealed dark red area; oedema and streaks of haemorrhages together with in the mucosa of caecum and localized area of colon were observed through post mortem examination. Similarly, it has been informed in many studies in goats [4, 18, 19, 26, 29, and 30]. In our study, the gross finding of the respiratory system consisted of trachea, bronchi and bronchioles were filled with mucopurulent froth with cut sections, the lung showed large quantities of frothy exudate. The lung revealed large

patchy area of dark red colour with focal consolidation. These results in accordance with [20]. In a partial agreement [2, 19, 30]. In present study, the pulmonary lesions were characterized by necrotic bronchiolitis and bronchitis together with broncho-interstitial pneumonia, pleuritis, viral inclusion bodies and the presence of syncytial cell formation. These results in a partial agreement [4, 9, 20, 24, 29]. Also these results disagreement with [3, 30]. In this study, the gross picture of lymphoid tissue was characterized by congestion, edematous, enlarged with cut surface revealed dark red areas. These result in accordance with those mentioned [4, 19, 20, 26, 29, 30] and in disagreement with [17]. Lymphoid tissue in our study showed extensive hemorrhage and acute necrosis, depletion of lymphoid cells and macrophages infiltration in the germinal centers of sinusoids, which implies compromise of the immune system. Similar lesions were reported [9, 19, and 29]. In a disagreement with [30]. In present study, the Histopathological finding reported in the heart was in accordance with [22], which reported interstitial myocarditis hyalinization of the myocytes and hemorrhages. These results are consideration novel pathologic feature of PPRV infection. In this study mortality rate of *C. perferingens* infection in complication with coccidiosis among outbreak in 2 flocks mortality rate 42% and 46% respectively. These results in a partial agreement with informed studies [15, 27]. The clinical finding in flocks under observation in our study were big head, pyrexia and diarrhea. These symptoms were referred to the isolation of *Eimeria*. These clinical findings in a partial accordance with result previously mentioned [11, 15, 27] whose studies carried out on single infection (*Eimeria*). In our study, the gross picture of the intestine reported as the proximal intestine, colon and caecum were congested and edematous with wide spread petechial hemorrhages and ecchymosis in its mucosa.

Their content revealed watery fluid with multiple blood clots, fibrin and distended with gases. These results in a partial similarity with that reported by previous authors in goats [1, 27, 28]. In our work, histopathological finding in the intestine are reported as congestion, necrotic enteritis with large number of the epithelial cells were necrosed together with hyperplasia of the remaining epithelium in which *Eimeria* in different stages of schizogony. Also The intestinal lesion were characterized by necrotic enteritis of the small intestine colon and caecum there is inflammatory cell infiltration mainly lymphocytes macrophages plasma cell and few neutrophils with presence of basophilic clusters of bacteria .Marked submucosa and subserosa edema congestion and leucocytic cell infiltration these lesions in agree with lesions mentioned by previous authors [12, 25]. In this study, the pulmonary lesions consisted of severe pulmonary edema and congestion.

5. REFERENCES

1. Abdel Wahab, M.B., Abakar, A.D., Nusa, T.M and Adam, M.E. (2008). Outbreaks of concurrent pneumonia and coccidiosis in sheep in South Darfur State, Sudan. *Sudan. J. Vet. Res.*, 23: 97-102.
2. Ahmad, K., Jamal, S.M., Ali, Q., Hussain, M., (2005). An outbreak of peste des petits ruminants (PPR) in a goat flock in Okara, Pakistan. *Pakistan Vet. J.* 25 (3), 146–148.
3. Aktas, M.S., Ozkanlar, Y., Simsek, N., Temur, A. and Kalkan, Y. (2011). peste des petits ruminants in suckling lambs case report. *Israel journal of veterinary medicine* 66 (1).
4. Al-Dubaib, M.A. (2009). Peste des petitis ruminants morbillivirus infection in lambs and young goats at Qassim region, Saudi Arabia. *Trop Anim Health Prod*, 41: 217-220.
5. Al-Humiany, M., Abdulrahman, A. (2012). Microbiological Studies On Enteritis Caused By *Clostridium Perfringens* Type A, In Sheep In Saudi Arabia. *Journal of Applied Sciences Research*, 8(2): 836-844.
6. Aytekin I., Mamak N., Ulucan A., Kalınbacak A., (2011). Clinical, haematological, biochemical and pathological findings in lambs with peste des petits ruminants, *Kafkas Üniversitesi Veteriner Fakültesi Dergisi*, 17(3): 349-355.
7. Bancroft, J.D. and Stevens, A. (1996). *Theory and Practice of Histological technique*.4th edition. Churchill, Livingstone, New York, London, San Francisco, Tokyo.
8. Carter, R. and Cole, J.R. (1990). *Diagnostic procedures in veterinary bacteriology and mycology*. 5th.ed. Academic Press, Harcourt, Boace Jov., Publisher, New York, Boston, London.
9. Chauhan, H.C., Lambade, P.S., Sen, A., Dadawala, A.I., Ranaware, P.B., Chandel, B., Joshi, D.V., Patel, S.S., Pankaj, K., Shah, N.M. and Kher, H.N. (2011). The use of pathological and histopathological techniques in the diagnosis of Peste des petits ruminants in India. *Veterinaria Italiana*, 47(1): 41-47.
10. Daube, G., Simon, P., Limbourg, B., Manteca, C.; Mainil, J.; Kaeckenbeeck, A. (1996). Hybridization of 2,659 *Clostridium perfringens* isolates with gene probes for seven toxins (alpha, beta, epsilon, iota, theta, mu, and enterotoxin) and for sialidase. *Am J Vet Res.* 57(4): 496–501.
11. Faris, A.E., Ahmed, E.A., Shawky, N.A., and Mahmoud, F.A. (2011). Field Trial For Treatment Of Coccidiosis In Goat Kids In Sharkia Governorate *Zag. Vet. J.* 39 (1): 136-143.
12. Fernandez, M.E., Saputo, J., Leger J.S., (2008). Necrotizing enterocolitis and death in a goat kid associated with

- enterotoxin (CPE)–producing *Clostridium perfringens* type A. Can Vet J 48:1266–1269.
13. Finnie, J.W. (2004). Neurological disorders produced by *Clostridium perfringens* type D epsilon toxin. Anaerobe 10: 145–150.
 14. Gasteiner, J. (2009). Erkrankungen durch Clostridien bei Ziegen. Fachtagung für Ziegenhaltung 15: 25 – 28.
 15. Ghanem, M.M., Mohamed, A.D.A., and Ramadan, M.Y. (2009). Clinical, biochemical and histopathological study on parasitic gastroenteritis associated with caprine coccidiosis: comparative effect of toltrazuril and propolis. Lucrări Științifice, 52.
 16. Greco, G., Madio, A., Buonavoglia, D., Totaro, M., Corrente, M., Martella, V., Buonavoglia, C. (2005). Clostridium perfringens toxin-types in lambs and kids affected with gastroenteric pathologies in Italy. Vet. J. 170: 346-350.
 17. Housawi, F.M.T. , Abu-Elzein, E.M.E., Mohamed, G.E. , Gameel, A.A., Al-Afaleq, A.I., Hegazi A., Al-Bishr, B. (2004). Emergence of Peste Des Petits Ruminants in Sheep and Goats in Eastern Saudi Arabia. Rev. d'Élev. Méd. Vét. Pays. Tropic. 57: 31- 34.
 18. Ismail, T.M., Yamanaua, M.K., Saliki, J.T., El-Knoly, A., Mebus, C. and Yilma, T. (1995). Cloning and expression of the nucleoprotein of Peste des petits ruminants virus in baculovirus for use in serological diagnosis. Virology 20: 776-778.
 19. Jagtap, S. P., Rajak, K. K., Garg, U. K., Sen, A., Bhanuprakash, V., Sudhakar, S. B., Balamurugan, V., Patel, A., Ahuja, A., Singh, R. K. and Vanamayya, P. R. (2012). Effect of immunosuppression on pathogenesis of peste des petits ruminants (PPR) virus infection in goats. Microbial Pathogenesis, 52(4): 217-226.
 20. Kataria A.K., Kataria N. & Gahlot A.K. (2007). Large-scale outbreaks of peste des petits ruminants in sheep and goats in Thar Desert of India. Slovenian vet. Res., 44(4): 123-132.
 21. Kreig, N.K. and Holt, J. (1984). Bergeys manual of systemic bacteriology. Vol 1.
 22. Kul, O., Kabakci, N., Atmaca, H.T. and Özkul, A. (2007). Natural peste des petits ruminants virus infection: novel pathologic findings resembling other Morbillivirus infections. Veterinary Pathology 44: 479–486.
 23. Kumar, V., Cotran, R. S. and Robbins, S.L. (1997). Basic Pathology. Sixth Ed., Philadelphia, London, Tokyo.
 24. Kumar, P., Tripathi, B.N., Sharma, A.K., Kumar, R., Sreenivasa, B.P., Singh, R.P., Dhar, P., Bandyopadhyay, S.K. (2004). Pathological and immunohistochemical study of experimental peste des petits ruminants virus infection in goats. J Vet Med B Infect Dis Vet Public Health. 51: 153-9.
 25. Miyashiro, S., Nassar, A.F.C., Delfava Cabral, A.D. and Silva, M. (2007). "Clostridium perfringens type A and D associated with enterotoxemia in an 18-month old goat." J. Venom. Anim. Toxins incl. Trop. Dis., 13(4): 885-893.
 26. Narayanan, R., Gopu, P., Baegan, S. (2008). Clinical Management in an outbreak of Peste Des Petits Ruminants in Barbari Goats. Vet World. 1(3): 81-82.
 27. Ozmen, O., Adanir, R., Haligur, M. (2012), Immunohistochemical detection of the cytokine and chemokine expression in the gut of lambs and kids with coccidiosis. Small Ruminant Research 105: 345– 350
 28. Rad, M.A., Movassaghi R., Sharifi K., Naseri, Z., Seifi, H.A. (2011): Two outbreaks of *Pasteurella Multocida* Septicemia , Comparative Clinical Pathology 20(1): 57-59
 29. Rahman, M.A., Shadmin, I., Noor, M., Parvin, R., Chowdhury, E.H. and Islam, M.R. (2011). Peste des petits ruminants virus infection of goats in Bangladesh:

- Pathological investigation, molecular detection and isolation of the virus. *The Bangladesh Veterinarian* 28(1): 1 – 7.
30. Rashid, A., Asim, M. and Hussain, A. (2008). An outbreak of peste des petits ruminants in goats at district Lahore. *The J. Anim. and Plant Sci.* 18(2-3): 72-75.
 31. Smith, L.D. and Holdman, L.V. (1968). *The pathogenic anaerobic bacteria.* Charles Thomas Publishers, USA. PP. 53- 188.
 32. Smith, M.C. and Sherman, D.M. (1994). *Goat Medicine, (Lea & Febiger, Philadelphia), pp. 620.*
 33. Soulsby, E. (1986). *Parasitology of Domesticated Animals. Seventh Ed, Bailliere, Tindell, London.*
 34. Stamp, J.; Watt, J. and Thomlinson, J. (1955). "*Pasteurella heamolytica septicaemia* of calves." *J. Comp. Path.,* 66: 183-185.
 35. Stern, M., and Batty, I. (1975). *Pathogenic Clostridia .Butter worth, London Boston.*
 36. Tooloei, M. and Masodei, M.H. (2008). The distribution and prevalence rate of enterotoxaemia in sheep in east Azerbaijan province, Northwestern Iran in spring 2008. *J of Animal and Veterinary Advances* 7(11): 1434- 1439.
 37. Uzal, F.A., Songer, J.G. (2008). Diagnosis of *Clostridium perfringens* intestinal infections in sheep and goats. *J Vet Diagn. Invest.* 20: 253–265.
 38. Vettorato, E, Schoeffmann, G, Beard, P, Clutton, E. (2011). Postoperative complications in a lamb after major surgery. *Veterinary Anaesthesia and Analgesia,* 38: 63–69
 39. Wessman, E. (1964). Interrelationship of smooth and non-smooth variant in the dissociation of *Pasteurella heamolytica.* *J. Bacterial.,* 88: 325-360.
 40. Willis, A.T. (1977). *Anaerobic bacteriology clinical and laboratory practice."* 3rd.ed. Butterworth, London, Boston. pp .1131-132.
 41. Zahur, A.B., Ullah, A., Irshad, H., Farooq, M.S., Hussain, M., and Jahangir, M., (2009). Epidemiological investigations of a peste des petits ruminants (PPR) outbreak in Afghan sheep in Pakistan. *Pakistan Veterinary Journal,* 29: 174–178



مجلة بنها للعلوم الطبية البيطرية

دراسات باثولوجية على عدوى الكلوسترديوم بيفرنجينز في الماعز المركبة مع عدوى بعض الطفيليات والفيروسات

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الملخص العربي

تم دراسة انتشار وبائي بالإصابة بالكولسترديوم بيفرنجينز المركبة في عدد من قطعان الماعز (الدمشقي والبلدي) والاغنام في محافظة الغربية - مصر في الفترة من أواخر مارس 2011 وحتى يوليو 2012 وأظهر الفحص البكتريولوجي إصابة ثلاث قطعان من الماعز الدمشقي بالكولسترديوم بيفرنجينز كما أظهر الفحص الطفيلي إصابة صغار الماعز الرضع اقل من ثلاث شهور في القطيع الثالث بالكوكسيديا بينما أظهرت نتائج الاعراض الظاهرة والفحص التشريحي والهستوباثولوجي اشتباه إصابة الماعز الدمشقي الرضع الصغيرة اقل من ثلاث شهور في القطيع الاول والثاني بمرض طاعون المجترات الصغيرة. القطيع الاول والثاني: أظهرت النتائج إصابتها بالكولسترديوم بيفرنجينز المركبة باشتباه الإصابة بمرض الطاعون المجترات الصغيرة وكانت نسبة الإصابة 92%، 94% ونسبة الوفيات 32% و 94% على التتابع. أظهر فحص الحيوانات المصابة تضخم في الراس والرقبة وارتفاع في درجة الحرارة والعطس والكح والدمع ورشح رغوي من الفم مع وجود رشح بلازمي سرعان ما يتحول الى مخاطي صديدي من العين والانف واسهال والتهاب بملتحمة العين وعند نهاية المرض حدوث تشنجات وتصلب الظهر مع التواء الراس والرقبة عليها. أظهر الفحص المرضي التهاب الفم التقرحي والتهاب الامعاء التكرزي مع وجود اجسام الفيروس وكذلك خلايا المسيدسنبال التهاب القصبة الهوائية والعصييات التكرزي مع التهاب رئوي بينى مصبى واوديميا والتهاب غشاء البلورا. اختفاء وتتركز الخلايا الليمفاوية مع وجود انتشار الخلايا الماكروفاج بالجهاز المناعي: التهاب بعضلات القلب البينى مع تحلل زجاجي وتتركز عضلة القلب. القطيع الثالث: صغار ماعز رضيعه اقل من ثلاث أشهر وكانت نسبة الوفيات 43%. أظهر فحص الحيوانات المريضة تضخم في الراس والرقبة وارتفاع في درجة الحرارة واسهال. وأظهر الفحص الباثولوجي وجود نقط نزفية بالأمعاء مع وجود تتركز مع انتشار طفيل الكوكسيديا بالخلايا.

(مجلة بنها للعلوم الطبية البيطرية: عدد 24 (1)، يونيو 2013: 196-208)