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GRANULOMATOUS HEPATITIS IN BALADY RABBITS ASSOCIATED WITH COCCIDIAL INFECTION

(With 6 Figures)

By

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الالتهاب الكبدى التكاثرى في الأرانب البلدية المصابة للعدوى بالكوكسيديا

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لوحظ التهاب كبدى تكاثرى نادرا في بعض الأرانب البلدية المصابة بالكوكسيديا ولقد امتدت هذه الالتهابات لتشمل مساحات كبيرة من الكبد مع وجود طفيل الكوكسديا في وسط هذه العقد التكاثرية ولقد نتج عن هذه الاصابات تليف أجزاء كبيرة من خلايا الكبد وبالتالي اثار ضارة على صحة وانتاجية الحيوان.

SUMMARY

Unusual granulomatous hepatitis apparently caused by coccidial infection was incidentally observed in balady rabbits. The granulomas were large and extend to involve a considerable areas of the liver. Coccidial oocysts were observed in the central zone of the granuloma. The later resulted in destruction and fibrosis of a large area of the hepatic lobules. These pathological alteration will leading to deleterious effect on the productivity of the rabbits.

INTRODUCTION

Coccidia are highly specific as to host and anatomic location,(JOHN and JOSEPH, 1983). Hepatic coccidiosis is a protozoal disease of rabbits and wild lagomorphs. Sporulated oocysts excyst in the duodenum and pass via blood and lymph to the liver and other organs (BARRIGA and ARONNI, 1979). Shizogony and gametogony occur in the biliary epithelium and unsporulated oocyst pass via bile ducts to the intestine (HOENIG et al., 1974). In hepatic coccidiosis lesion is primarily a papillary hyperplasia of the biliary epithelium (JONES and HUNT, 1983). The aim of this work is to describe some of the extrabiliary hepatic lesions, which may be resulted from unusual localization of the coccidial oocysts in the hepatic parenchymal cells.

MATERIALS and METHODS

Hepatic gross lesions used in this study were incidentally oseved in 20 out of 100 balady rabbits after slaughtering. Samples from these focal areas, lung, spleen and intestine were obtained fixed in 10% neutral buffer formalin and further processed for histopathological studies. Sections of 6 micron thickness were stained with H & E and examined.

RESULTS

I- Macromorphological findings :

White foci, small in size were constantly observed in the liver. They were multiple and often seen in the left lobe of the liver. Examination of the intestine revealed no detectable gross pathological changes.

II- Micromorphological studies :

According to the nature of the lesions and it's location, microscopical alteration in the liver could be classified into four categories.

The first type consisted of a distinctly large hepatic granulome. The latter had a necrotic centre which mostly occupied by coccidial oocsts, and surrounded by remnant of degenerated and necrotic hepatocytes (Fig. 1). The oocyst had a duplex wall, pear shaped, transparent structure. It contained an acidophilic round or elongated body with a small basophilic nucleus. This elongated body has two flagella at each end. Dissolution of the nucleus may occur, leading to basophilic staining of the body (Fig. 2).

Sometimes the necrotic centre was not present and in this case it was only occupied by the oocysts with few remnant of necrosed hepatocytes (Fig. 3).

Not infrequently the centre of the granuloma contained multiple hollow spaces filled with coccidial oocysts and were seprated by a thick dense fibrous connective tissue. The capsule was surrounded from outside by a zone of lymphoid, macrophages and eosinophil cells. The hepatic cells close to these lesions were suffering pressure atrophy and degeneration. It is evident that the granulomas were not only large but also extended to involve several hepatic lobules.

The second type was consisted of a small granulomas which periportally located (Fig. 4). The centre of the granuloma was made of a necrotic or degenerated hepatic cells and a relatively few number of coccidial oocysts. The central zone was encircled by a thick zone of cellular infiltration, in which esinophil, macrophages, lymphoid, fibroblast and giant cells could be clearly identified (Fig. 5).

The third type was observed in the portal tract and consisted of connective tissue proliferation which in few instance contained newly formed bile ducts.

The last type of lesion was observed in the bile ducts and characterized by alterative changes. The epithelium revealed degenerative and necrotic changes. Few number of loucocytic cells infiltration were observed in and around the bile ducts. Extensive proliferation of the connective tissue around the bile duct was a constant findings. Focal areas of proteinous and hydropic dystrophy were frequently observed involving the hepatic parenchymal cells.

Pathological examination of the lung revealed proliferation of the peribronchial lymphoid cells aggregation which sometimes leading to displacement of the bronchial epithelium.

Hyperplasia of lymphoid cells of the white pulp, increased macrophages and eosinephil cells popultion of the red pulp were observed in the spleen.

Microscopically in the intestine (Fig. 6), the sexual stages of the parasite (Zygot) were found in the intestinal glands. It was consisted of a relatively large pear or oval structure, which contain coarse eosinophilic granules. With exception of the local distruction of the cells in which, the Zygot was observed, no evidence of inflammation or hyperplasia of the mucosal epithelium were seen.

DISCUSSION

JONES and HUNT (1983) stated that hepatic coccidiosis in rabbits affects the intrahepatic biliary epithelium in somewhat the same manner as other species of coccidia affect the intestinal epithelium. Destruction or proliferation of the biliary epithelium becomes the predominant feature. In this study, the lesions observed in the liver due to hepatic coccidiosis were completely differ. Wide spread grnaulomatous hepatitis with extensive necrotic changes and proliferation of connective tissue together with esinophil and lymphoid cells reaction dominate the picture. JUBB and KENNEDY (1985) added that in hepatic coccidiosis of rabbits the sporozites reach the intrahepatic bile ducts through the portal veins or lymphatics, not by the way of the common bile ducts. Such mechanism was likely to occur in the present study. However, the sporozite instead of being transformed to the intrahepatic bile duct become localised in the hepatic parenchyma and produce this granulomatous reaction. It was also possible that the intrahepatic bile ducts may also ruptured and the parasite become in contact with hepatic parenchyma leading to the inflammatory process.

Minimal lesions observed in the intestine were probabley due to local immunity (KLESIUS et al., 1976). Reactive lymphoid hyperplasia observed in the spleen and lung may be due to defence mechanism of the body against the infection (TAYLOR and BURRAUNHS, 1973). From this study, we can concluded that coccidia was responsible for extensive granulomatous hepatitis with prominent necrosis and fibrosis of the liver, which may have a serious effect on the productivity of the rabbits.

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LEGENDS OF FIGURES

Fig. (1): Showing a large granuloma with necrotic centre and outer zone of dense-fibrous C.T. (H&E., 10 x 10 X).

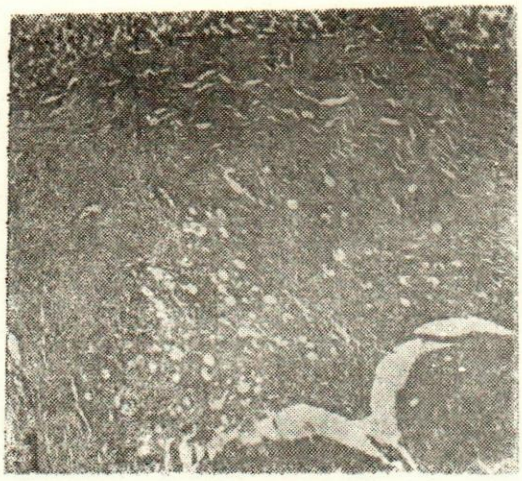
Fig. (2): The centre of the granuloma consists of Eimeria Oocysts and necrotic hepatic cells (H&E., 10 x 25 X).

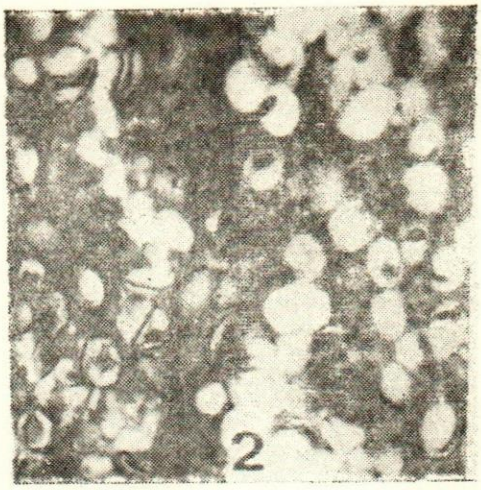
Fig. (3): Showing single granuloma (H&E., 10 x 10 X).

Fig. (4): Showing periportal small granuloma (H&E., 4 x 25 X).

Fig. (5): Showing the main reacting cells in the small granuloma (H&E., 10 x 25 X).

Fig. (6): Showing coccidial organisms in the small intestine (H&E., 10 x 16 X).

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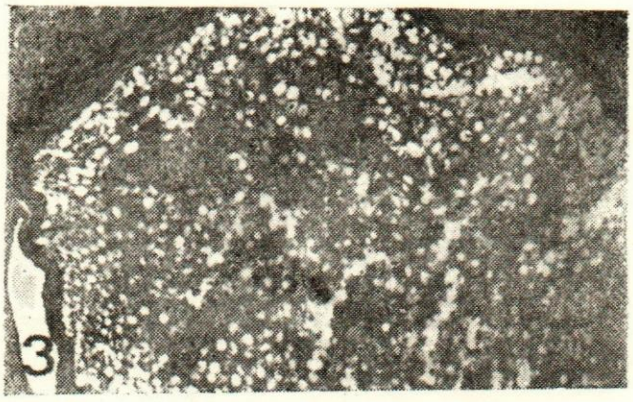
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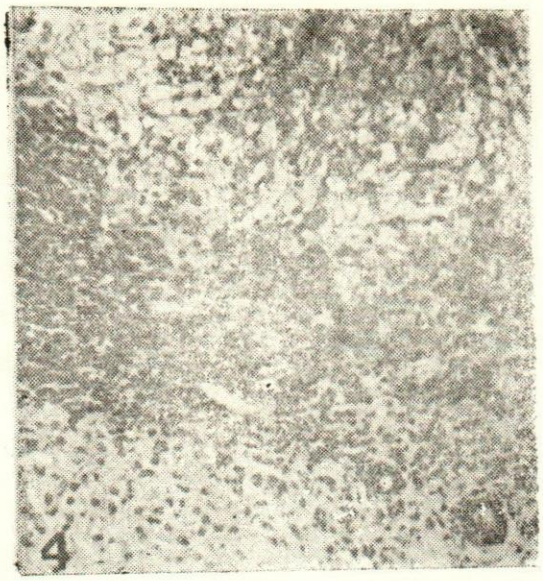
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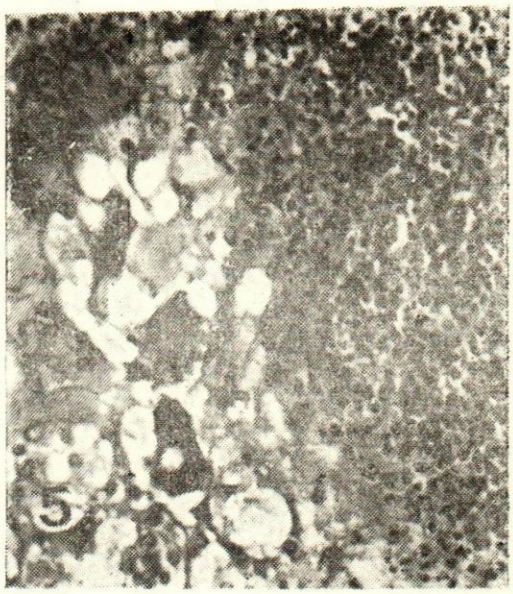
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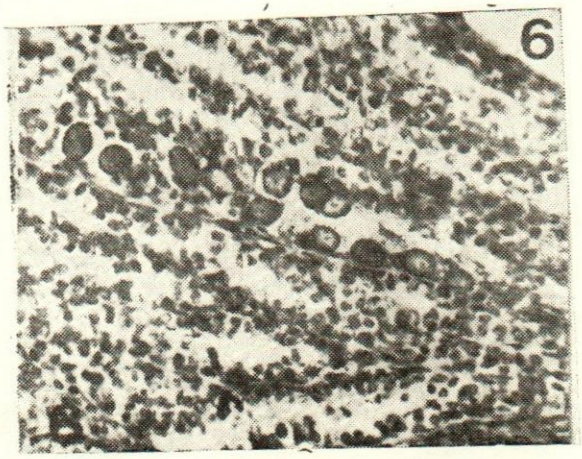
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