

Pathological study of Hepatic coccidiosis in naturally infected rabbits

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Abstract

Thirty young domesticated rabbits (age 2-2.5 mo) of sexes showed clinical signs of anorexia, debilitation, diarrhea, icterus, rough hair coat and pendulous abdomen with hepatomegaly. Fecal samples were collected for demonstrated the presence of oocysts. Postmortem examination revealed presence of discrete yellowish-white nodules of 1mm to 2 cm size on the surface and throughout the parenchyma containing a thick creamy white fluid. The histopathological changes showed biliary hyperplasia with different developmental stages of *Eimeria stiedae* in the epithelial cells, cholangitis and peribiliary fibrosis with newly formed bile ductules, severe congestion and dilation of central veins and sinusoids with disruption hemorrhagic areas. The hepatocytes showed degenerative changes to necrosis with areas of fibrosis and mononuclear cell aggregation, obstructive jaundice and a tendency to form oocyst granuloma. In conclusion hepatic coccidiosis lead to severe pathological changes both in bile ducts and liver parenchyma especially in young animals.

Key words: Coccidiosis. *Eimeria*. Rabbits.

Introduction

Coccidiosis is a common and worldwide protozoal disease of domestic rabbits but only rarely reported in other mammals (1). Kids are most susceptible; however, infected adults become carriers. There are 2 anatomical forms: hepatic caused by *Eimeria stiedae* (2), and intestinal caused by *E. magna*, *E. irresidue*, *E. media*, *E. perforans*, *E. flavescens*, *E. intestinalis* or other *Eimeria* species, transmission of both the hepatic and intestinal forms is by ingestion of sporulated oocysts usually in contaminated feed or water (3, 4). The oocysts of *Eimeria stiedae* were first seen by Antoni Van-Leewenhoek in the bile of a rabbit in 1674 (5). The disease is a ubiquitous protozoan infection of animals seriously impairing their growth and food utilization (6). It causes significant

mortality in domestic rabbits (7, 8). Most of the researchs indicates that there is a strong relationship between the infection and the host age; they believe that the infection decrease with increasing age of the host (9). The coccidia of rabbits have not been studied to the same degree as the species which occur in other hosts. In Iraq there is a lack in information about this parasite; a few studies only were reported regarding the parasitological aspect of the disease (10, 11). No to our knowledge has attempted to show the effect of *E. stiedae* in liver tissue. Therefore, the aim of the study is to accomplish this task and study the pathological changes in affected livers. The results will increase our basic information on the current situation of this disease and for future researchs.

Materials and Methods

Animals

Thirty young domestic rabbits both sex (males and females) weighting 2-3 kg were kept in the Laser Institute for Higher Education for Experimental Studies. Rabbits were fed by concentrate and vegetable *ad libitum*, all the rabbits were found infected with *E. stiedae* as detected by fecal examination and post-mortem examination.

Parasitological procedure:

The symptoms of the naturally infected animals were recorded daily. The samples were then processed in the laboratory and examined by direct smear as well as saturated sugar flotation technique for the presence and identification of eimerian oocysts (12).

Histopathological procedure:

Livers of the dead rabbits were thoroughly examined for the gross lesions and liver tissues were collected in 10 % buffered formalin solution. Tissues were processed and 5-6 micrometer thick paraffin

embedded sections were cut for histopathological examinations after staining with hematoxylin and eosin (H&E) according to (13).

Results and Discussion

Clinical signs:

The main clinical signs showed by the infected rabbits were depression, anorexia, brown watery diarrhea, emaciation, rough hair coat, pendulous and distended abdomen and hepatomegaly noted on abdominal palpation, progression weakness and death. Jaundice was also reported in 5 cases. The size and weight of livers of dead animals increased due to excessive proliferation of bile duct epithelium resulting in hepatomegaly which is characteristic of this disease leading to pendulous and distended abdomen. These results were agreed with (14, 10) stated that diarrhea, emaciation, and rough hair was due to infection of the intestine before the penetration of the sporozoites to the mucosa of small intestine and passing via the hepatic portal system to the liver to start the hepatic form (15).

Pathological Study:

Gross Lesions:

Hepatomegaly with irregular yellowish white nodules on the surface. (Figure1). Thick creamy white exudates from their cut surface, hepatic parenchyma was firm and gall bladder was distended. The peritoneal cavity showed increased dirty dull straw colored peritoneal fluid. The hepatomegaly was due to marked proliferation and distention of bile ducts forming nodules raised above the surface causing increase in secretion of mucous appeared as a creamy, white fluid on cut surface. The proliferated bile ducts cause damage to the liver parenchyma leading to post-necrotic scarring this explain, the firm consistency of hepatic parenchyma. These results agreed with (14, 16) that these lesions lead to disturbance of liver functions leading to decrease in α -lipoprotein, glucose and proteins; in addition to increase in bilirubin in blood serum (17).

Histopathology:

The lesions of the liver tissue sections of infected rabbits mainly confined to the liver and bile ducts consisted primarily of extensive hyperplasia of bile ducts. The bile ducts were markedly enlarged and lined by hyperplastic columnar epithelial cells thrown into multiple arborizing papillary fronds extending into the ductal lumina, resembling adenomatous hyperplasia with presence of developmental stages of the parasite (figure2). Numerous protozoal stages including microgametocytes, macrogameteocytes and oocysts were also seen (figure3). Other sections showed that the bile ducts were highly dilated with flattened epithelium having no or minimum projection to the lumen which are filled with numerous thin walled, ovoid oocysts (figure4, 5). The hyperplastic bile ducts were surrounded by large amount of fibrous connective tissue infiltrated with mononuclear cells (figure6). Furthermore, the ducts were filled with sloughed biliary epithelial cells. The hepatic parenchyma showed dilation and congestion of central veins and sinusoids with rupture of lining endothelial layer; in addition to vacuolar degeneration and necrosis of hepatocytes (figure7). The portal areas and liver parenchyma were infiltrated with mononuclear cells (figure8), severe hemorrhage in the necrotic area were also seen (figure9). Other areas showed the replacement of parenchyma with fibrous connective tissue infiltrated with mononuclear cells with a tendency to formation of newly formed bile ductules. Oocyst granuloma in which coccidial oocyst were observed in the central region were also noticed (figure10). In animals with jaundice their liver sections showed deposition of bile pigment in hepatic parenchyma (figure11). The present result

showed that all animals were affected with the *E. stiedae* and that may be due to their younger age since it is almost known that hepatic coccidiosis which caused by *E. stiedae* is a primary disease of young rabbits (9). Coccidial infection is affected by the host age; The highest incidence was in 2-months rabbits and then the infection rate decreased as the age increased. The high level of susceptibility of infection in young rabbits may be due to their immune, feeding and reproductive status. This observation is consistent with the results previously reported (18, 19). This hepatic coccidiosis caused severe damage to the liver and it is more pathogenic in young rabbits and led to death among these animals (20). The proliferation of the bile duct epithelium might be due to the predilection and proliferation of the *E. stiedae* within the epithelium, whereas extensive dilation with little or no proliferation of bile duct epithelium might indicate the cell turn over which was also proved by the fact that these ducts contained more numbers of oocysts as compared to ones that had conspicuous proliferation. The wide spread sinus dilation, associated with

fibrosis in and around the cords might be attributed to the obstructed hepatic blood flow especially in the portal veins by immensely proliferating and dilating bile ducts. The stagnation of the blood flow would also result in hepatocyte cellular degeneration and atrophy of the cords. These histopathological observations are in agreement with those described by others (21, 14, and 22). If the hepatic continuity of epithelium of bile ductules is broken the coccidian organism or oocyst would act as foreign bodies it might involve typical foreign body granuloma. (23) reported granulomatous hepatitis in coccidial infection in which coccidial oocysts were observed in central region of granuloma which later results in destruction and fibrosis of large area of hepatic lobules. The hepatic parenchyma showed areas of fibrosis with obstructive jaundice and that occurred due to massive necrosis of parenchyma leading to post-necrotic scarring. The bile pigment deposition was due to obstruction of the main ductal system from local swelling and jaundice is almost always present and that in agreed with previous studies (24, 14).

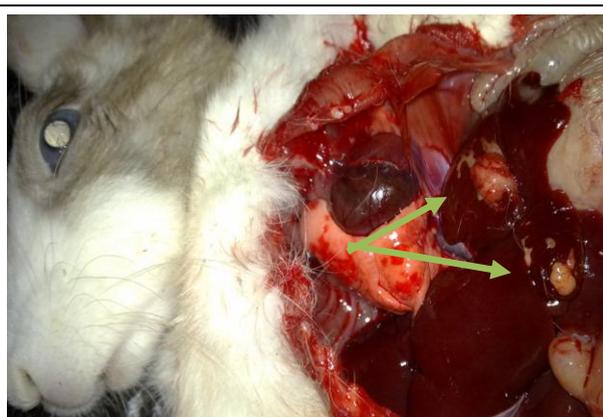


Figure 1: Gross lesion in the liver of rabbit infected with *E. stiedae* showing irregular yellowish white nodules on the surface (→).

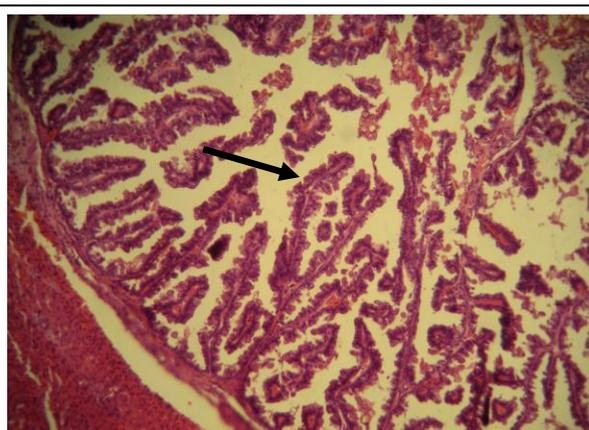


Figure 2: liver of naturally infected rabbit with coccidiosis showing multiple papillary fronds in the bile ducts due to extensive hyperplasia of biliary columnar epithelial cells (→) (H&Ex100).

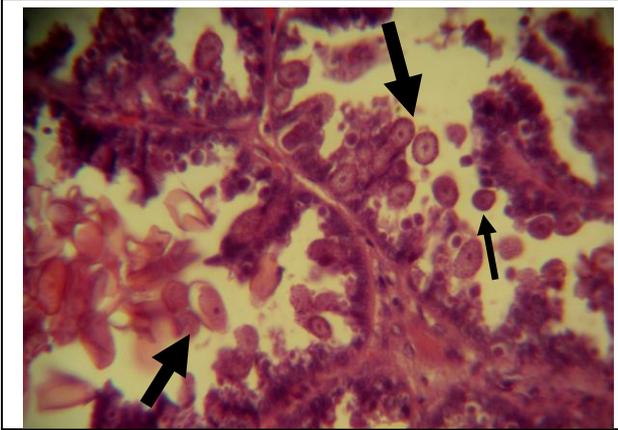


Figure 3: liver of naturally infected rabbit with coccidiosis showing different stages of the protozoal stages including microgametocytes (→), macrogametocytes (→) and oocysts (→) (H&Ex400).

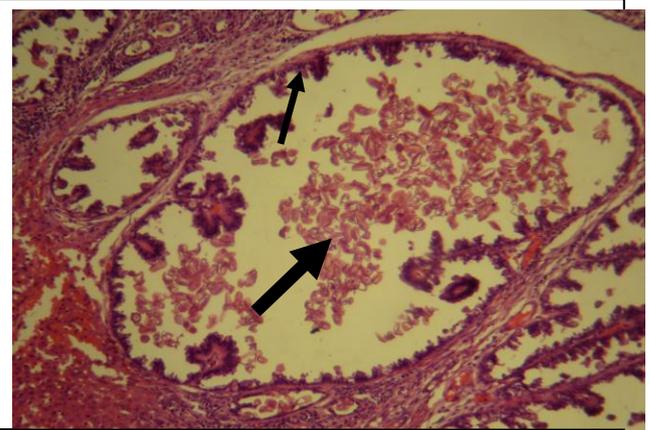


Figure 4: liver of naturally infected rabbit with coccidiosis showing bile ducts highly dilated with flattened epithelium having no or minimum projections to the lumen (→) which are filled with numerous oocysts (→) (H&Ex100).

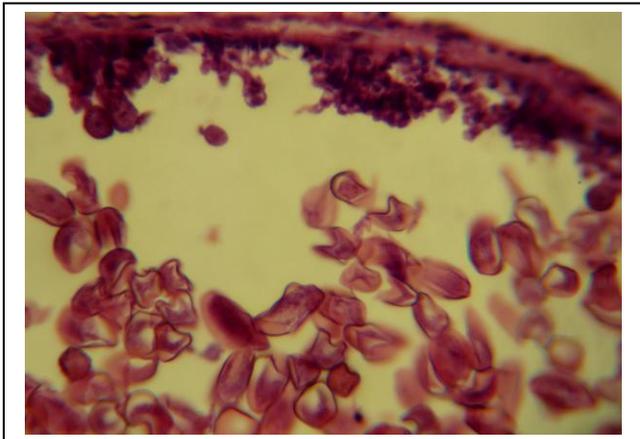


Figure 5: liver of naturally infected rabbit with coccidiosis at high magnification of the previous section (H&Ex400).

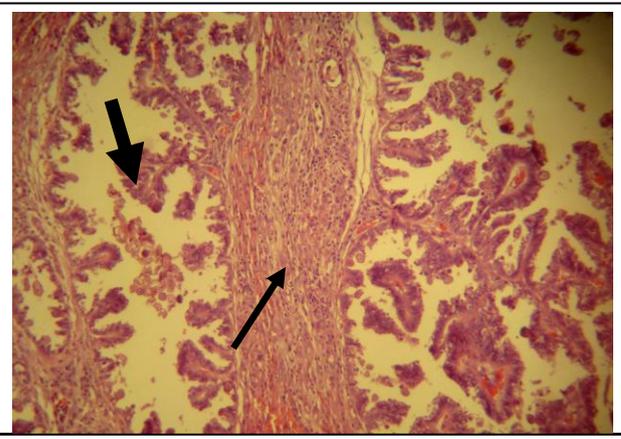


Figure 6: liver of naturally infected rabbit with coccidiosis showing peribiliary fibrosis with infiltration of mononuclear cells (→) and papillary projections of epithelium (→) with different stages of the parasite (H&Ex100).

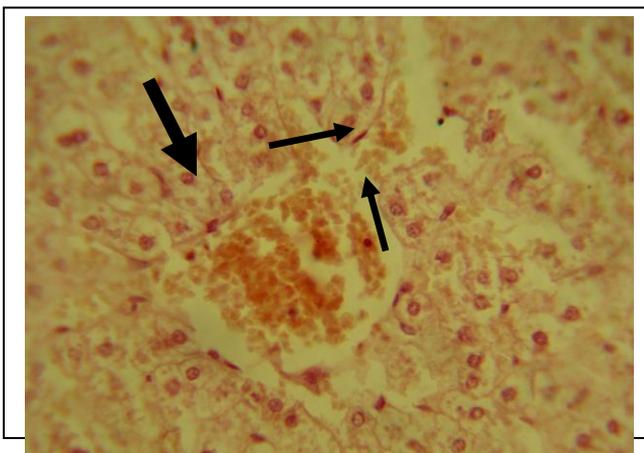


Figure 7: liver of naturally infected rabbit with coccidiosis showing dilation and congestion of central vein with rupture of lining endothelial cells (→) with vacuolar degeneration of hepatocytes (→) (H&Ex400).

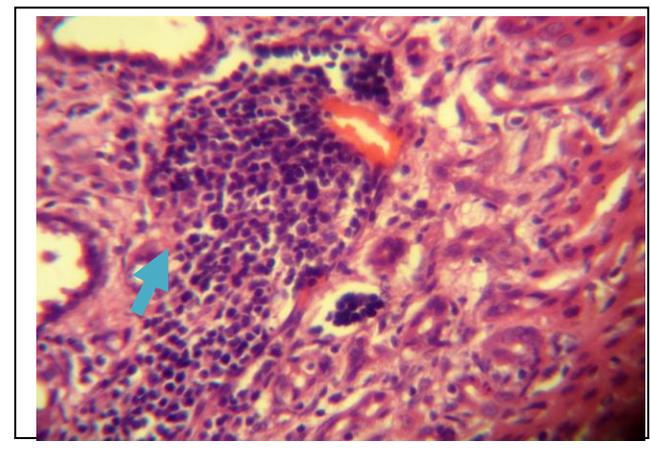


Figure 8: liver of naturally infected rabbit with coccidiosis showing focal infiltration of mononuclear cells infiltration within hepatic parenchyma (→) (H&Ex400).

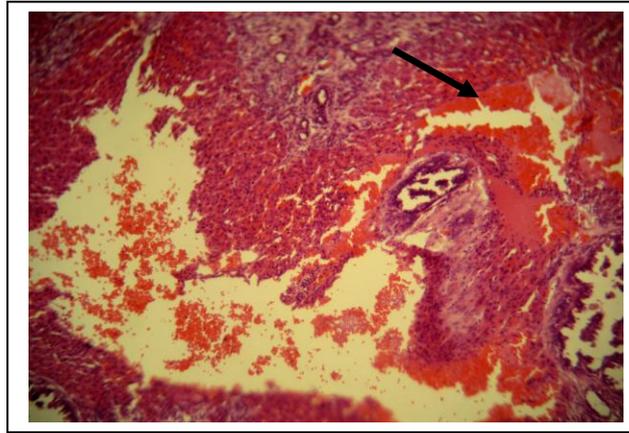


Figure 9: liver of naturally infected rabbit with coccidiosis showing severe hemorrhage (↔) within the necrotic area (H&Ex100).

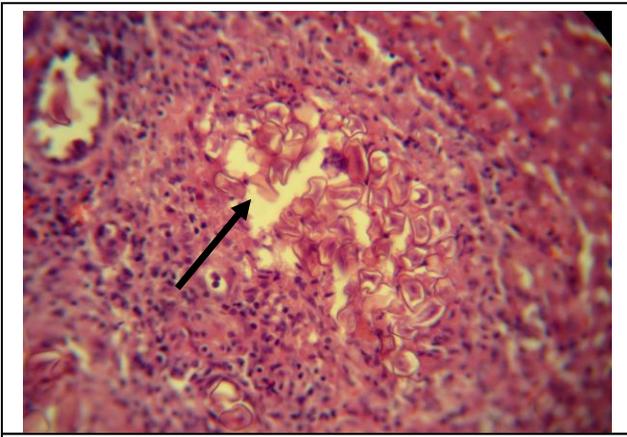


Figure 10: liver of naturally infected rabbit with coccidiosis showing oosyst granuloma within hepatic parenchyma (↔) (H&Ex400).

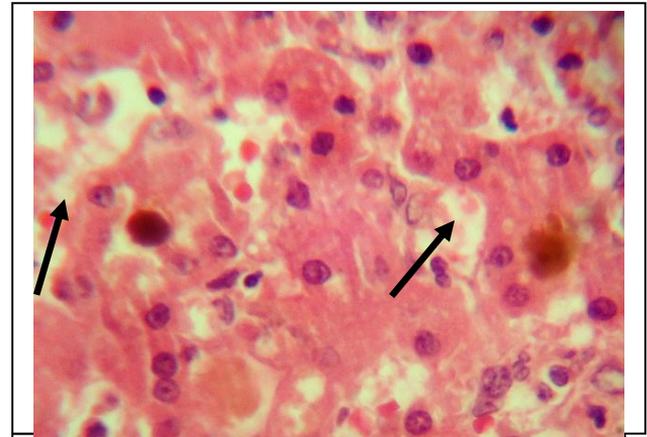


Figure11: liver of naturally infected rabbit with coccidiosis showing deposition of bile pigment in hepatic parenchyma (↔) (H&Ex400).

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دراسة التغيرات المرضية لمرض الكوكسيديا في أكباد الأرناب المصابة طبيعياً

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الخلاصة

أستعمل في هذه الدراسة 30 أرناب من النسل المحلي ذات اعمار صغيرة تراوحت بين (2-2.5 شهر) ومن كلا الجنسين. أظهرت هذه الحيوانات علامات سريرية تميزت بالقهم، الضعف، الاسهال، اليرقان، خشونة الفرو مع تدلي البطن نتيجة ضخامة الكبد. تم تشخيص المرض من خلال وجود البيوض في عينات البراز وأفات العيانية والمرضية النسجية للأكباد المصابة. لقد أظهر الفحص العياني للكبد وجود عقيدات صفراء- بيضاء وباحجام من 1 ملم- 2سم منتشرة على سطح الكبد وفي داخل المتن تحتوي على سوائل قشدية بيضاء ثخينة. أما الفحص المرضي النسجي فقد أمتازت آفات بفرط التنسج الصفراوي مع وجود مراحل مختلفة من تطور حياة الطفيلي داخل الخلايا الظهارية المبطنة لقنوات الصفراء مع حصول التهاب قنوات الصفراء وتليف حول القنوات. فضلاعن أحتقان شديد للاوردة المركزية والجيبانيات مع حصول تمزق في جدران الاوردة المركزية مؤدية الى النزف الشديد وحصول تغيرات تنكسية ونخرية للخلايا الكبدية وتليفات أرتشحت بالخلايا وحيدة النواة ويرقان انسدادى مع اورام حبيبية تحتوي بداخلها على أكياس البيوض. نستنتج مما سبق ان مرض الكوكسيديا في الأرناب يؤدي الى حدوث آفات مرضية شديدة في قنوات الصفراء ومتن الكبد خصوصاً في الاعمار الصغيرة.

مفاتيح الكلمات: مرض الكوكسيديا. الأيميريا. الأرناب.