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Diagnostic Exercise
From the Latin Comparative Pathology Group and the Davis-Thompson Foundation

Hepatic eimeriosis in rabbits

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History

One hundred and fifty 5-month-old crossbred rabbits raised for meat production were examined at a slaughterhouse. No abnormalities were observed during ante-mortem inspection.

Necropsy Findings

Ten of 150 animals had similar hepatic changes identified during routine post-mortem meat inspection. The most severe case is described here. The liver was mildly swollen and form, with coalescing, slightly elevated, round to linear, white to yellow, well-demarcated nodules (Fig. 1) with thickened gallbladder luminal contents (Fig. 2). On cut sections, affected areas

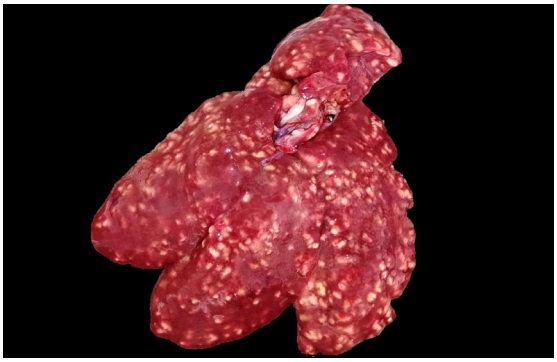

Figure 1. The livers mildly swollen with coalescing, slightly elevated, round to linear, white to yellow, well-demarcated nodules.

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were filled with variable amounts of thick, amorphous, white to yellow material (Fig. 3). The liver was collected, fixed in formalin, processed routinely for histology, and stained with hematoxylin and eosin.

Follow-up questions:

- Histologic description
- Morphologic diagnosis:
- Etiology:
- Name the disease:

ANSWERS

Histologic Description

Liver: Biliary ducts were moderately to severely dilated, occasionally tortuous, and lined by markedly hyperplastic epithelium that formed irregular papillary projections into the lumen. These were supported by a markedly thickened basement membrane (Figs. 4A and 4B). The periductal stroma was often expanded by lymphoplasmacytic infiltration and scarce epithelioid macrophages and multinucleated giant cells, some containing intracytoplasmic, round to oval oocysts measuring

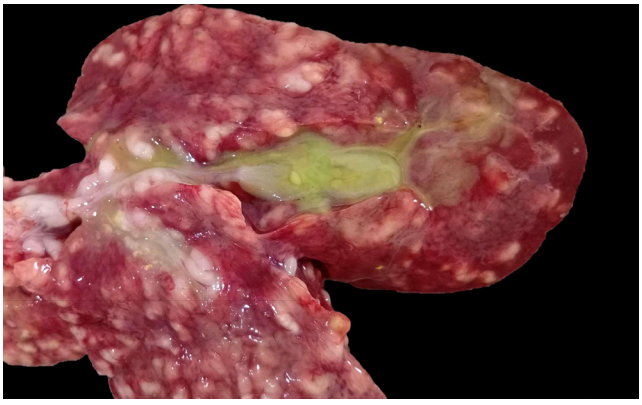

Figure 2. Rabbit, liver and gallbladder. gallbladder luminal content is viscous.

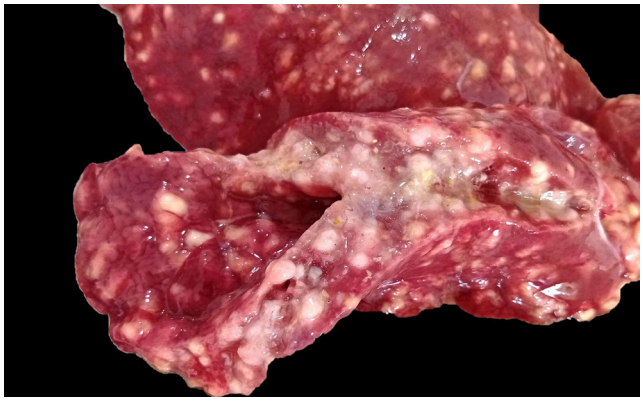

Figure 3. Rabbit, liver, cut section. Affected areas were filled with variable amounts of viscous amorphous, exudate

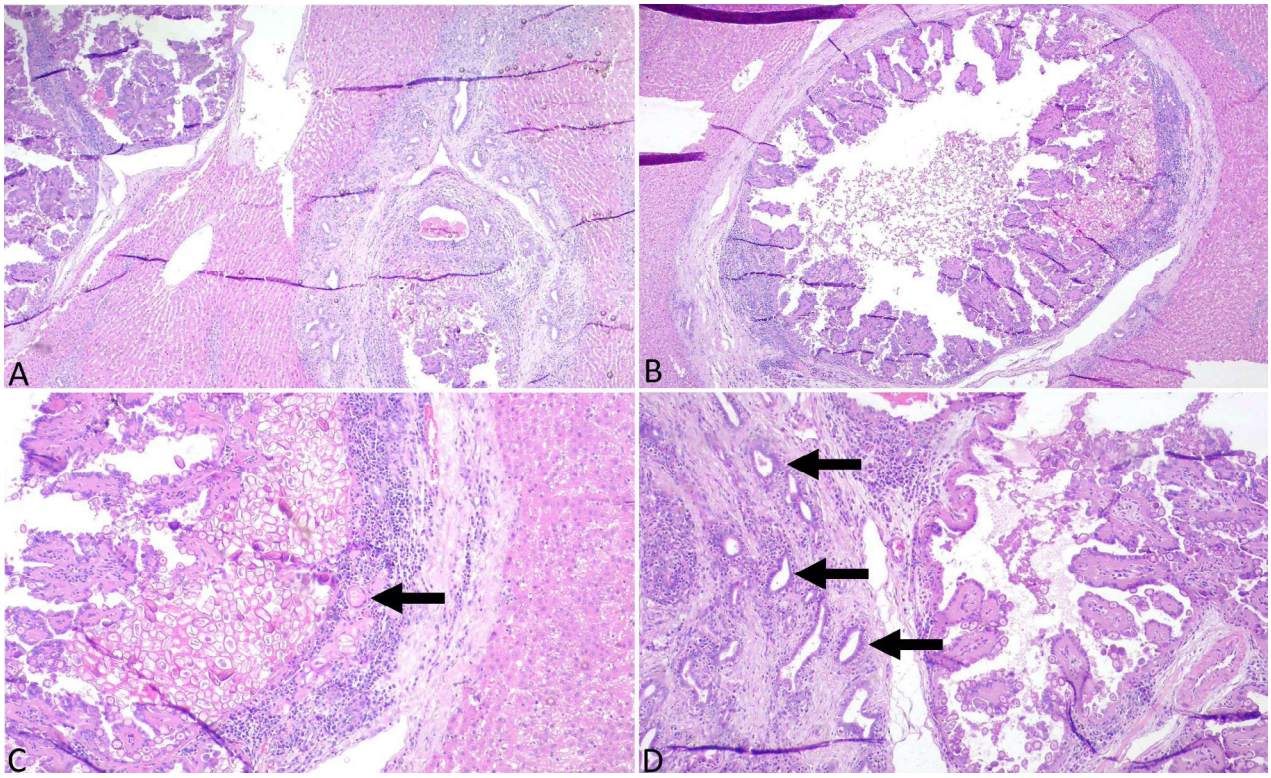

Figure 4A-D. Rabbit, liver. Hyperplasia of biliary ducts (arrows)

approximately 30–40 µm in diameter with a dense, thick, amphophilic wall (Fig. 4C, black arrow). There was moderate fibroblast proliferation within variably loose to organized and vascularized collagen bundles (granulation tissue) accompanied by moderate biliary hyperplasia (Fig. 4D, black arrows). Within biliary epithelial cells, protozoal organisms in multiple developmental stages were observed (Fig. 5), including macrogametocytes (pink arrow), which were round to oval and approximately 30–40 µm in diameter, with a fibrillar eosinophilic central nucleus with a 3–4 µm amphophilic nucleolus; microgametocytes (blue arrow), which were basophilic, round to oval, and approximately 20–30 µm in diameter; and abundant oocysts (black arrow). Lumina frequently contain moderate amounts of cellular debris and free oocysts. In a dilated biliary duct (Fig. 6A), there was complete loss of epithelium (black arrows) with abundant cellular debris (asterisk) and numerous oocysts (Fig. 6B, black arrows). Hepatocytes had mild cytoplasmic microvacuolization (lipid degeneration).

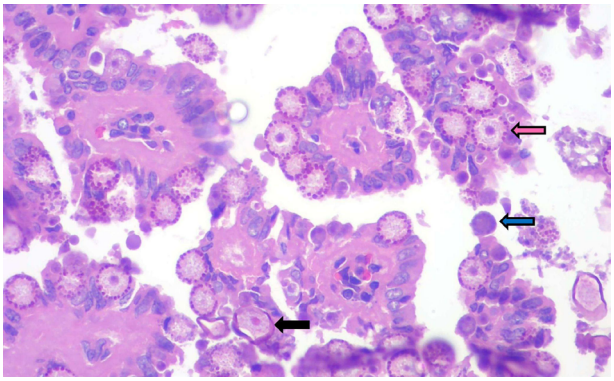

Figure 5. Rabbit, liver, biliary epithelium with varying protozoal stages (arrows)

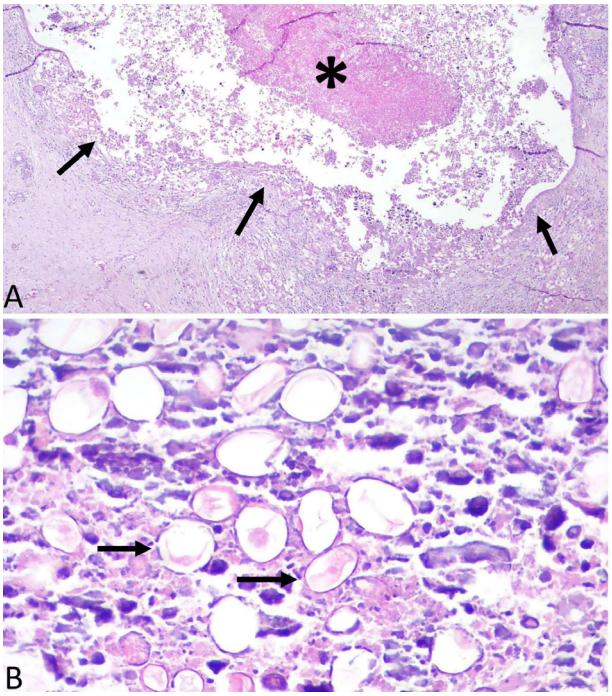

Figure 6. Rabbit, liver and biliary ducts (A) Intraductal inflammatory and necrotic debris (B). Degenerated protozoa (arrows) amidst intralaminar necrotic debris.

Morphologic Diagnosis

Liver: Multifocal, severe, chronic, proliferative, necrotizing, and granulomatous cholangitis and pericholangitis with intralesional zoites (*Eimeria stiedae*).

Etiology:

Eimeria stiedae.

Name of the disease:

Hepatic eimeriosis.

Comments:

Eimeria stiedae causes hepatic eimeriosis in rabbits (*Oryctolagus cuniculus*) and is a widespread protozoan parasite of the apicomplexan genus *Eimeria* (1,2). It is particularly important in commercial rabbit farms and is considered one of the most economically significant diseases of rabbits due to high morbidity and mortality (3). It also affects wild rabbits and is one of the causative agents of "white-spotted liver" in the United Kingdom (4).

Like all *Eimeria* species, *E. stiedae* is monoxenous (5), meaning its life cycle is completed within a single host species. It is strictly host-specific to rabbits and poses no zoonotic risk. The life cycle is clearly described and illustrated and includes three phases: merogony, gametogony, and sporogony. After the ingestion of sporulated oocysts, sporozoites invade the hepatic biliary epithelium. Merogony and gametogony occur in these cells, producing oocysts that are shed in feces. Oocyst maturation (sporogony) occurs in the environment, generating new infective forms to continue the life cycle. Lesions develop during merogony and gametogony, and severity correlates with parasite load (1,2).

Weaning animals are the most affected age group and may have poor body condition with depletion of adipose tissues, abdominal distension due to ascites, and jaundice in advanced cases (1). Biochemical changes may include hyperlipidemia, hyperbilirubinemia, hypoglycemia, and hypoproteinemia (6), as well as elevated ALT, AST, ALP, and GGT (3).

Grossly, the liver is enlarged with multiple, slightly raised, linear to bosselated yellow to gray lesions (0.5–2 cm), often with green to dark tan content on cut surface. The gallbladder may be distended and filled with dense green bile

(1,3). In survivors, lesions consistent with cirrhosis may occur, such as parenchymal nodules with diffusely increased consistency and gaseous intestinal distension with green ingesta (3).

Histologically there is a marked dilation of bile ducts lined by papillary hyperplastic epithelium with numerous schizonts, gametocytes, and oocysts. Mitotic activity may be increased. Periportal regions show moderate to severe fibrosis with inflammatory infiltrates composed mainly of lymphocytes and macrophages. In chronic stages, parasites may be sparse, with predominant fibrosis in portal areas and associated hepatocellular vacuolar degeneration (1,3). The animal in this case, along with others from the same rabbitry, had good body condition and no significant findings at ante-mortem inspection, suggesting subclinical infection. The granulomatous pericholangitis described here, although uncommon in hepatic coccidiosis, has been previously reported and likely results from oocyst leakage or entrapment in the periductal stroma (7).

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