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

Acute hepatic fasciolosis in sheep

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Clinical history:

Three adult sheep presented a history of being apathetic, lethargic, and with weight loss for one week duration and then succumbed to death. The flock comprised 60 sheep; in the last month, ten have died. The sheep were raised on a field with swampy areas and native pasture and supplemented with soy, corn, and proteinate salt. Treatment included antitoxic (hepatoprotective), moxidectin, and levamisole, with meager results.

Gross Findings:

On postmortem examination, all sheep were in poor body condition and with pale tan discoloration of the ocular conjunctiva. The abdominal cavity of all sheep had mild fibrinous peritonitis that extended throughout all organ serosae. On the hepatic capsule, there was a marked deposition of fibrin and multiple multifocal hemorrhagic linear areas that extended on the cut surface. Also, there was a moderately enlarged gallbladder (Figure 1a, b). On the cut surface, hepatic ducts were slightly thick. During the inspection of the lungs of all necropsied sheep, marked pulmonary edema was noted, in addition to multifocal pinpoint to linear hemorrhagic areas (Figure 2a, b).

Follow-up Questions:

- *Histologic Description*
- *Morphologic diagnoses*
- *Etiology*

**The Diagnostic Exercises are an initiative of the Latin Comparative Pathology Group (LCPG), the Latin American subdivision of The Davis-Thompson Foundation (DTF). These exercises are contributed by members and non-members from any country of residence. Consider submitting an exercise! A final document containing this material with answers and a brief discussion will be posted on the DTF website (https://davist- hompsonfoundation.org/diagnostic-exercise/).*

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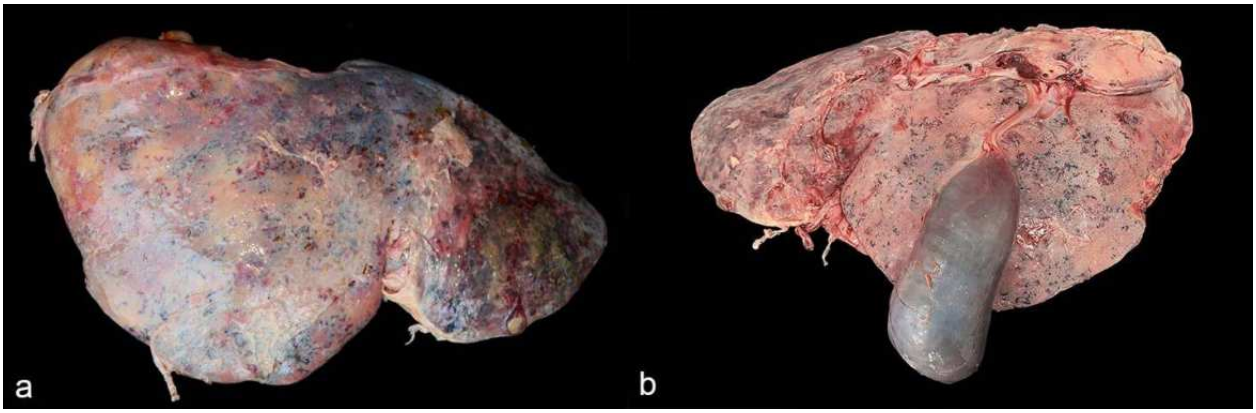


Figure 1. Liver. Gross findings (a, b). On the capsule, there is a moderate deposition of fibrin, in addition to multifocal to coalescent linear hemorrhagic areas.

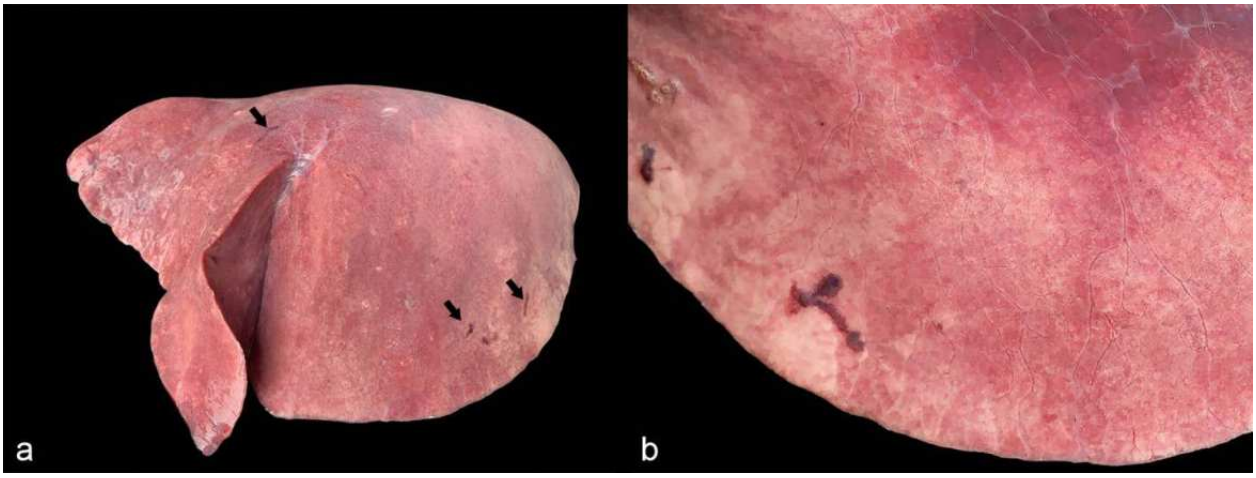


Figure 2. Lung. Gross findings. a, diffusely brightly and with pink discoloration, associated with multifocal pinpoint to linear hemorrhagic areas (arrows). b, close-up view of the hemorrhagic area in a.

ANSWERS

Histologic Description:

In the liver, there were multifocal random areas of necrohemorrhagic foci associated with cellular debris, fibrin, and inflammatory infiltrate of neutrophils and eosinophils (Fig 3a, b, c). Additionally, in periportal regions, there was a proliferation of fibrous connective tissue that often formed bridges among the periportal areas associated with biliary duct hyperplasia and inflammatory infiltrate of macrophages that had enlarged cytoplasm filled by a granular brown pigment (Fig 3d). In the lungs, there were also multifocal areas of coagulative necrosis associated with hemorrhage, fibrin deposition, cellular debris, thrombosis, and inflammatory infiltrate of neutrophils and eosinophils (erratic migration of *F. hepatica*) (Fig 4a and b). On the remaining pulmonary parenchyma, there was marked pulmonary edema. In sections of the liver and lungs, one could observe transversal and longitudinal cuts of trematodes (Fig 5a and b).

- *Morphologic diagnoses*

I. Fibrinous suppurative perihepatitis, acute, diffuse.

II. Multifocal extensive necrotic, hemorrhagic, and fibrinous hepatitis, acute.

III. Bridging portal fibrosis, diffuse, marked, chronic.

IV. Multifocal extensive necrotic, hemorrhagic, and fibrinous pneumopathy, acute with intralesional trematode larvae.

- Etiology:

Fasciola hepatica

Comments:

The parasitosis caused by *Fasciola hepatica* is called fasciolosis, which could develop among varieties of species, including ruminants, humans, and wild animals. The life cycle of the parasite is represented in Fig.6. Some special attention to the life cycle is required, as the miracidium stage of the parasite

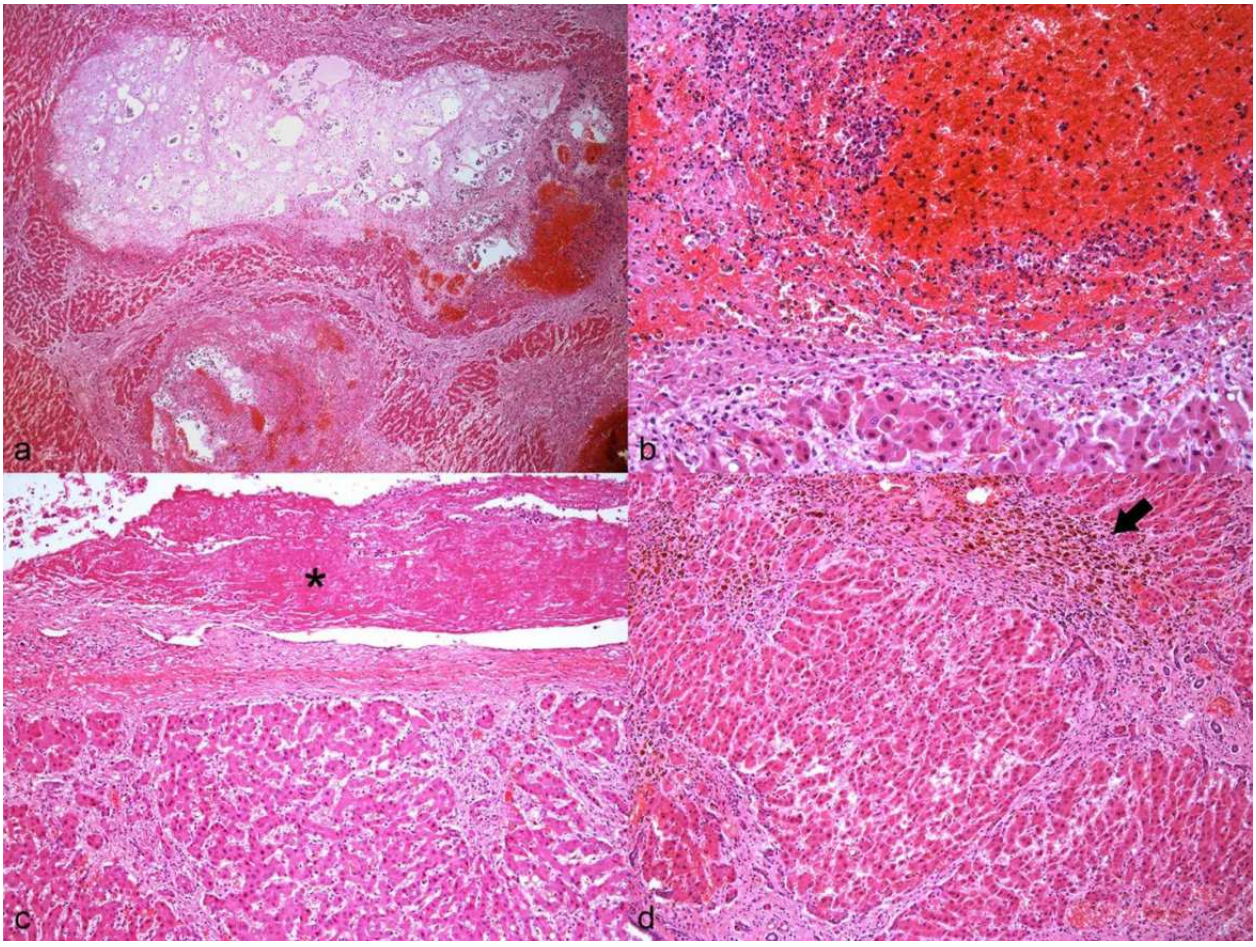


Figure 3. Liver. Microscopic findings. In the hepatic parenchyma, there were multifocal areas of necrosis of hepatocytes that were characterized by cellular debris accumulation associated with hemorrhage, fibrin deposition, and inflammatory infiltrate of neutrophils and eosinophils (a, b). Also, in the capsule, there was a moderate to severe fibrin deposition (*) associated with inflammatory infiltration of neutrophils (c). Adjacent of the necro-hemorrhagic areas, in the periportal region, there was a moderate bridging periportal areas composed of fibrous connective tissue, associated with biliary duct hyperplasia and inflammatory infiltrate of macrophages that had increased cytoplasm and filled by a granular brown pigment (arrow) (d).

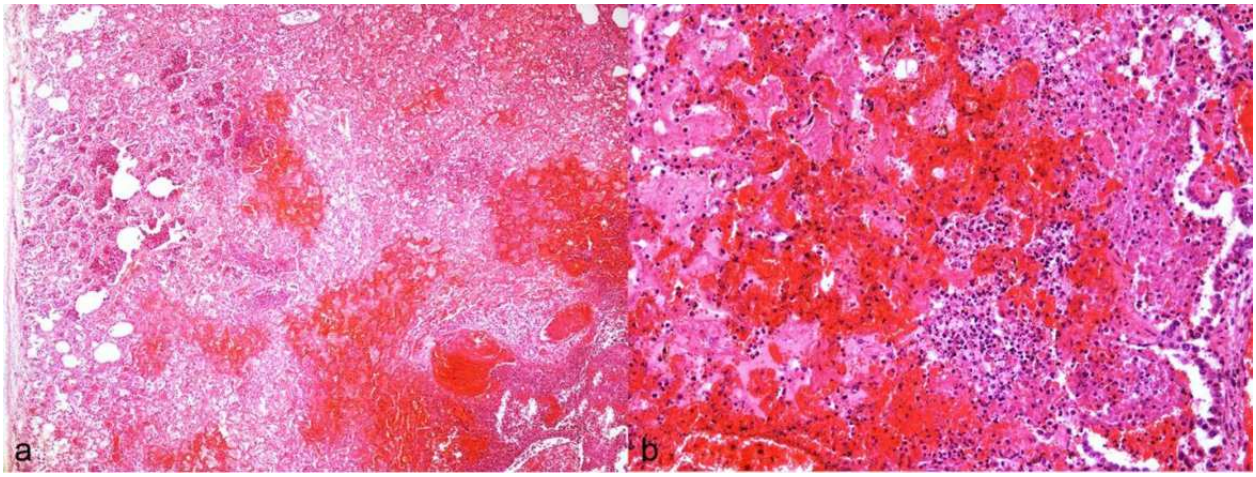


Figure 4. Lungs. Microscopic findings (a, b). In the parenchyma, there were multifocal areas of coagulative necrosis associated with hemorrhage, fibrin deposition, cellular debris, thrombosis, and inflammatory infiltration of neutrophils and eosinophils. On the remaining pulmonary parenchyma there was marked pulmonary edema.

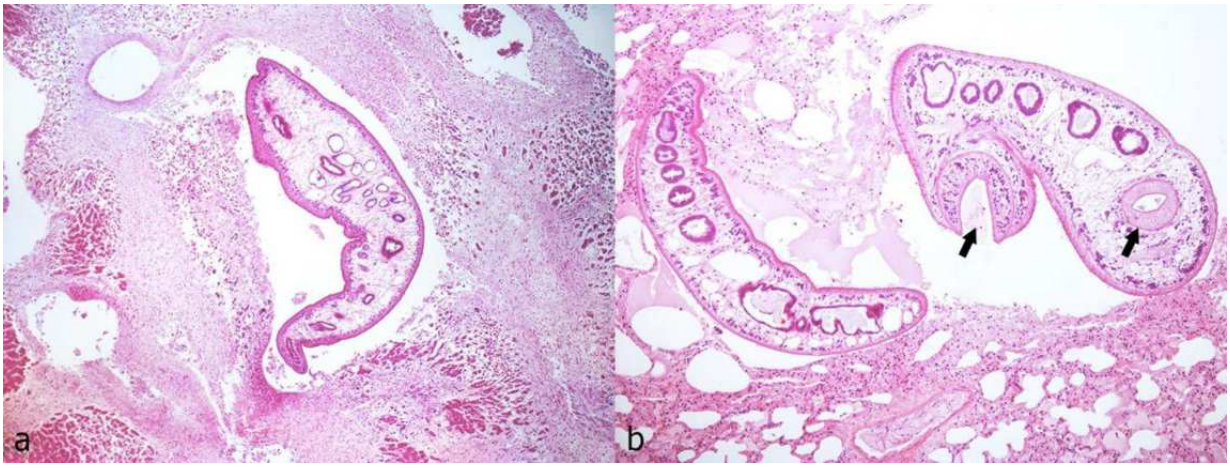


Figure 5. Liver and lung. Microscopic findings (a, b). In the lung and hepatic parenchyma, there were transversal and longitudinal sections of trematodes composed of a thick tegument associated with eosinophilic spicules, suckers (oral and ventral) (arrows), and internally there were alimentary and reproductive structures. These trematodes had morphology compatible with *Fasciola hepatica*.

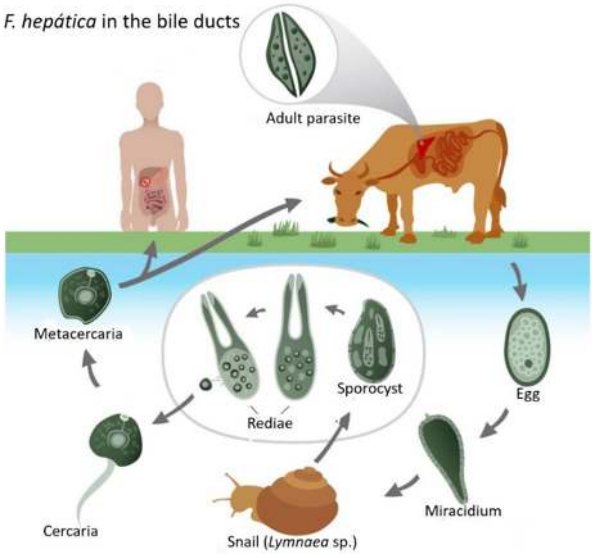


Figure 6. Life cycle of Fasciola hepatica. The eggs of *F. hepatica* are eliminated in the bile, and then in feces, which then goes to the environment. The egg hatches and produces a miracidium that survives only in humid environments. The miracidium actively penetrates the intermediate host (snail of the genus *Lymnaea*). Inside the snail, each miracidium develops into a sporocyst. Each sporocyst gives rise to 5-8 rediae, which, in turn, give rise to daughter rediae and cercariae. The cercariae leave the snail and encyst in the plant leaves, just below the water level, becoming metacercariae. The final host (in this case, ruminant) ingests the metacercariae along with the plants. People and other domestic and wild mammals can also become infected. The ingested metacercariae break down the cyst in the duodenum of the definitive host and penetrate the intestinal wall, migrate through the coelom, penetrate the liver capsule, and migrate through the liver parenchyma until reaching the hepatic ducts, where reproduce and deliver the eggs. Reproduced with permission from Tessele et al. (4)

only survives in humid environments, requiring a snail of the genus *Lymnaea* as an intermediate host that lives exclusively in flooded areas. The ned of a snail in the cycle explains why this parasitosis is only found in flooded flat areas (4).

Twenty-four hours after reaching the duodenum, most immature trematodes have already penetrated the gut wall. They are in the abdominal cavity (coelom), and after 4-6 days, most have penetrated the liver capsule and migrated through the parenchyma until they reach the bile duct. For this part of the migration, the immature forms of the trematode usually reach the liver via transcoelomic (through the abdominal cavity); in some cases, the hematogenous route is also described. The migration process through the liver parenchyma lasts 5-6 weeks. Some trematodes can accidentally penetrate the hepatic veins, reaching the systemic circulation and located in unusual sites, particularly in the lungs (1,4), which was a finding of this report

During the 2 to 3 months that the fluke resides in the bile ducts, it reaches sexual maturity and begins to release eggs through the bile. This phase is marked by chronic lesions in the host's liver, a common manifestation of the disease. Clinically, this is observed as a chronic wasting disease. On gross examination, thickening of the bile ducts and atrophy of the hepatic lobe are evident. Microscopically, there is a proliferation of fibrous connective tissue in the periportal region, along with bile duct hyperplasia and cholangiohepatitis (4, 2).

However, when there is a massive larval migration through the hepatic parenchyma over a short period, often results in sudden death, characterizing the acute form of fasciolosis. Additionally, acute parasitism would concurrently develop within chronic lesions (3). Both lesions were observed in the reported cases, but the death of the animals was linked to the acute lesions.

The gross lesions observed of fibrinous perihepatitis, in addition to multiple multifocal hemorrhagic linear areas

widespread through the hepatic parenchyma, are compatible with the findings of the acute form already described in the literature. Also, the microscopic findings of multifocal random necrohemorrhagic foci corroborate with this classification (3).

References:

1. Cullen JM, Stalker MJ. Chapter 2 - Liver and biliary system: tremadodes. In: Maxie MG, editor. Jubb, Kennedy & Palmer's Pathology of Domestic Animals: Volume 2. Sixth Edition. W.B. Saunders; 2016. p. 258-352.
2. Hashemnia M, Rezaei F, Nikousefat Z, Ghashghaii A. Acute caprine fasciolosis: a case with unusual migration to lung. Journal of Parasitic Diseases. 2015 Sep 20;39(3):514–7.
3. Stuen S, Ersdal C. Fasciolosis—An Increasing Challenge in the Sheep Industry. Animals. 2022 Jun 8;12(12):1491.
4. Tessele B, Brum JS, Barros CSL. Lesões parasitárias encontradas em bovinos abatidos para consumo humanob[Parasitic lesions observed in cattle slaughtered for human consumption.]. Pesquisa Veterinária Brasileira. 2013 Jul;33(7):873–89. Portuguese