Diagnostic Exercise

From The Davis-Thompson Foundation*

Case # **250** Month: **December** Year:**2024**

Answer Sheet

Title: Acute hepatic fasciolosis in sheep with erratic migration through the lungs.

Contributors: Anderson H. Gris[1], Paola Sônego[1], Emanoelly M. S. Silva[1], Claudio S. L. Barros[2], David Driemeier[1].
[1]Setor de Patologia Veterinária, Faculdade de Medicina Veterinária, Universidade Federal do Rio Grande do Sul (SPV-UFRGS), Porto Alegre, RS, Brazil.
[2]Laboratório de Anatomia Patológica, Universidade Federal de Mato Grosso do Sul, Campo Grande, MS, Brazil.
Corresponding author: anderson_gris@hotmail.com.br

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 animals were raised on a field with swampy areas and native pasture and supplemented with soy bran, corn, and proteinate salt. Affected sheep were treated with antitoxic (hepatoprotective), moxidectin, and levamisole, unsuccessfully.

Gross Findings: on postmortem examination, all sheep were in poor body condition and with pale tan discoloration of 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 a yellow fibrinous material and multiple multifocal hemorrhagic linear areas that extended on the cut surface. Also, there was a moderately enlarged gallbladder (Fig. 1**a**, **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 (Fig. 2**a**, **b**).

Gross and histological images:

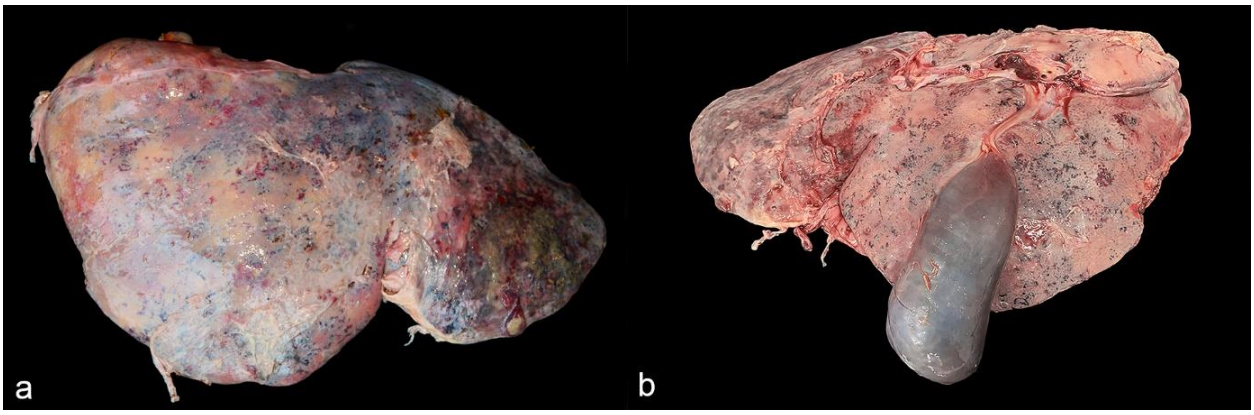

Figure 1. Liver. Gross findings (**a**, **b**). On the capsule, there is a moderate deposition of fibrinous material, in addition to multifocal to coalescent linear hemorrhagic areas.

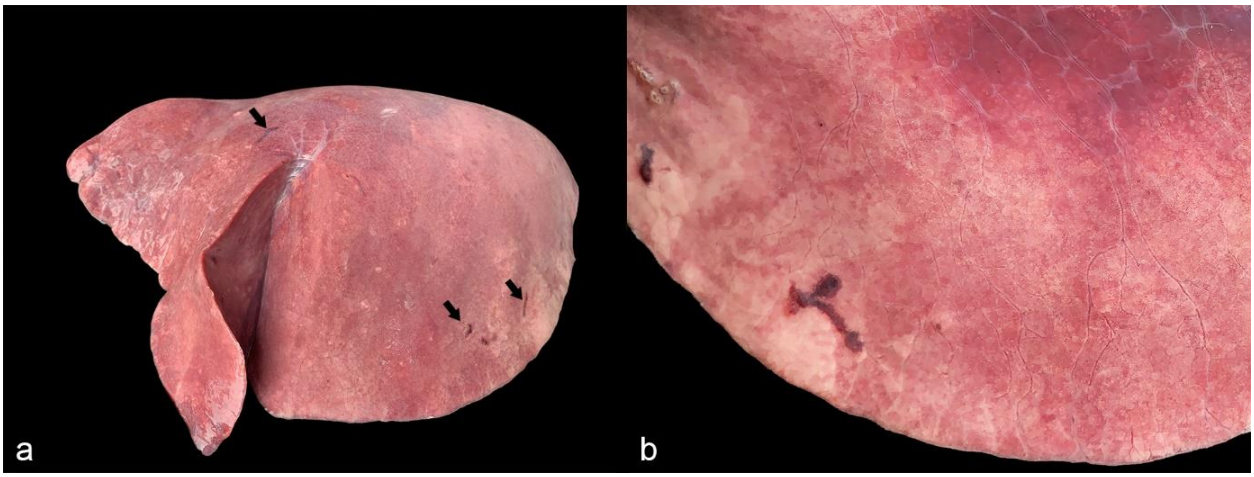

Figure 2. Lung. Gross findings. **a**, diffusely brightly and with pink discoloration, associated with multifocal pinpoint to linear hemorrhagic areas (*arrows*). **b**, close-up view of the hemorrhagic area in **a**.

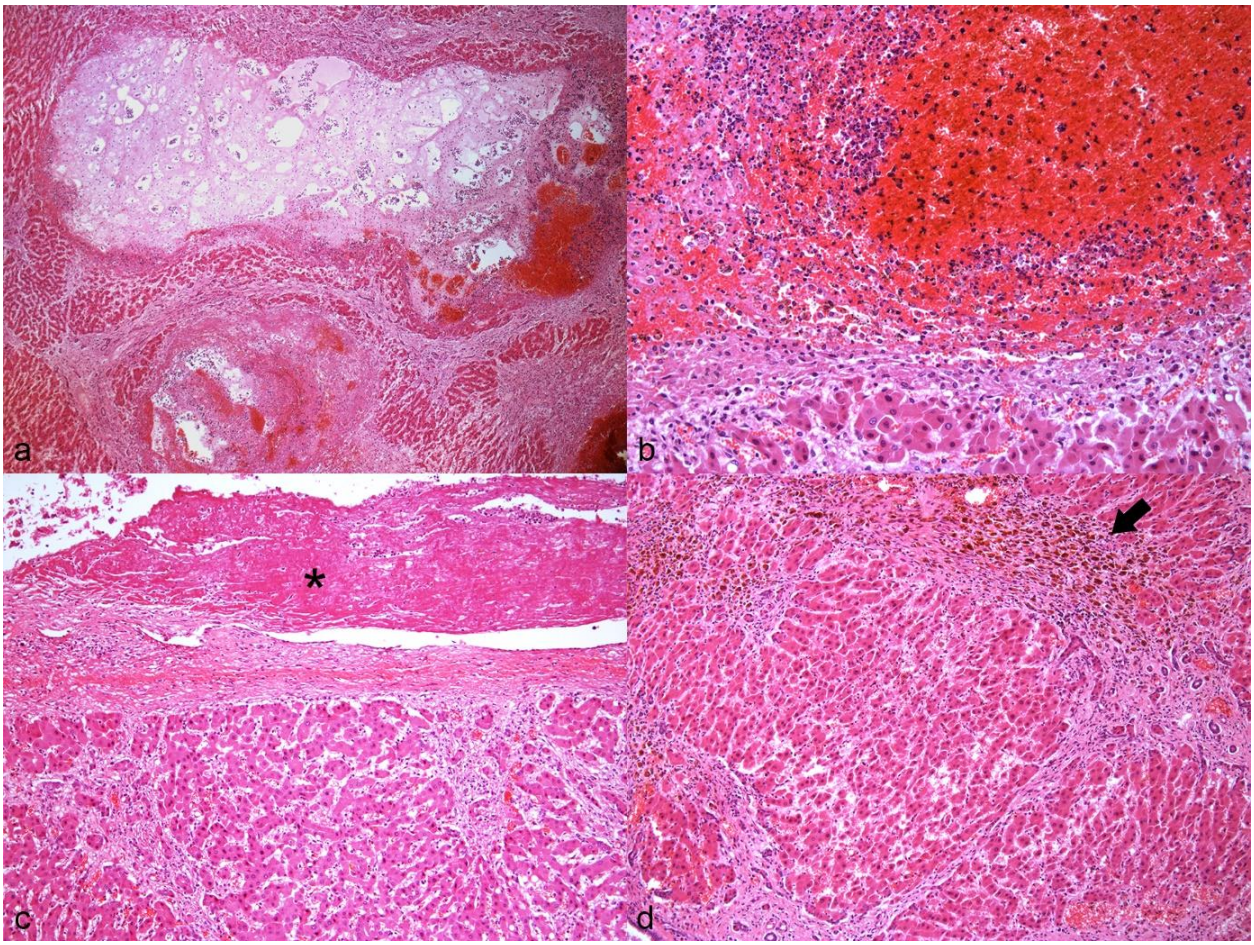

Figure 3. Liver. Microscopic findings. In the hepatic parenchyma, there were multifocal areas of necrosis of hepatocytes that were characterized by cellular debris accumulation associated with hemorrhage, fibrin deposition, and inflammatory infiltrate of neutrophils and eosinophils (**a, b**). Also, 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**).

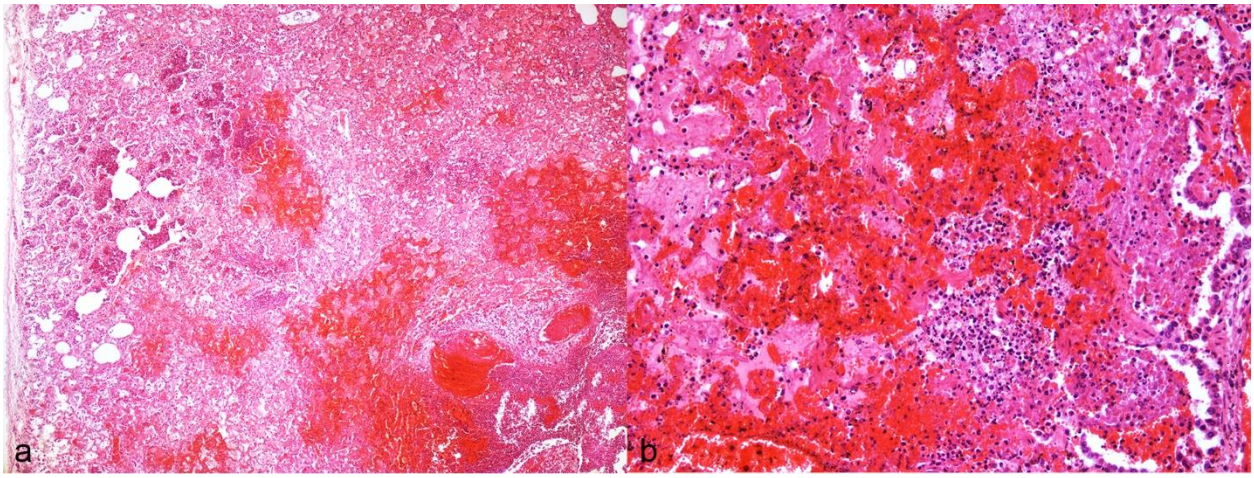

Figure 4. Lungs. Microscopic findings (**a, b**). In the parenchyma there were multifocal areas of coagulative necrosis associated with hemorrhage, fibrin deposition, cellular debris, thrombosis and inflammatory infiltration of neutrophils and eosinophils. On the remaining pulmonary parenchyma there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

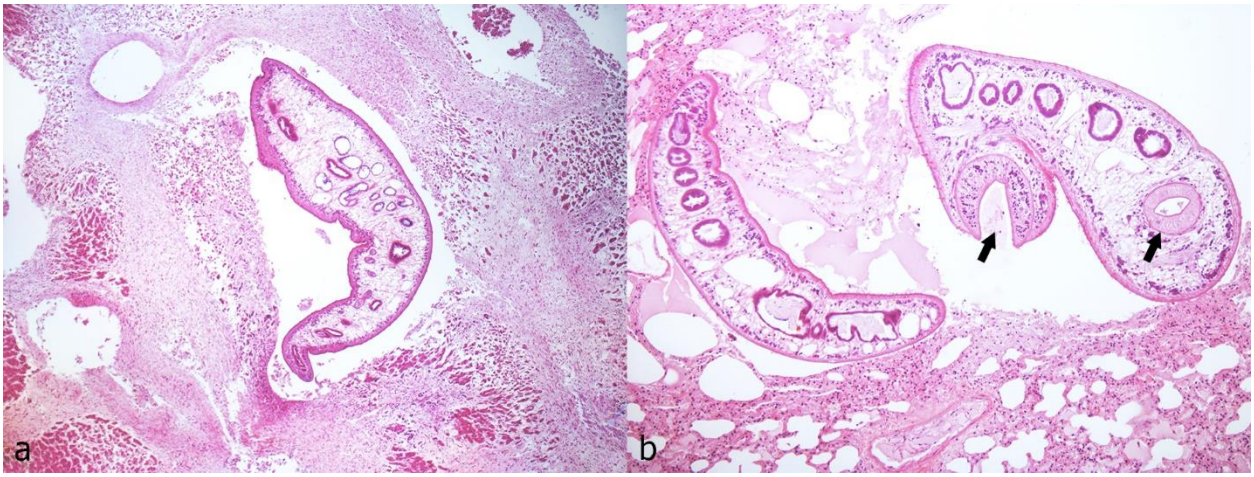

Figure 5. Liver and lung. Microscopic findings (**a, b**). In the lung and hepatic parenchyma, there were transversal and longitudinal sections of trematodes composed of a thick tegument associated with eosinophilic spicules, suckers (oral and ventral) (*arrows*), and internally there were alimentary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

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 3**a**, **b**, **c**). Additionally, in periportal regions, there was 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 increased cytoplasm and filled by a granular brown pigment (Fig 3**d**). 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. 4**a** and **b**). On the remaining pulmonary parenchyma, there was marked pulmonary edema. In some sections of the liver, there was transversal and longitudinal cuts of trematodes which were also seen in some areas of the lung (Fig 5**a** and **b**).

Morphologic diagnosis:
1. Fibrinous suppurative perihepatitis, acute, diffuse.
2. Multifocal extensive necrotic, hemorrhagic, and fibrinous hepatitis, acute.
3. Bridging portal fibrosis, diffuse, marked, chronic.
4. 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 only survives in humid environments, requiring a snail of the genus *Lymnaea* as intermediate host, that lives exclusively in flooded areas. This explains why this parasitosis is only found in flooded and flat areas (4).

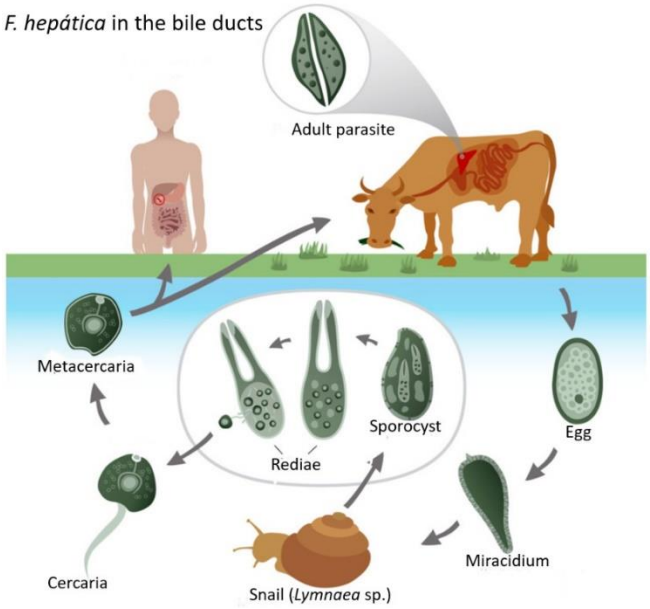

Figure 6. Life cycle of *Fasciola hepatica*. The eggs of *F. hepatica* are eliminated in the bile, and then in feces, which then goes to the environment. The egg so hatch and produce a miracidium that survives only in humid environments. The miracidium actively penetrates the intermediate host (snail 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. Pesq Vet Bras. 2013; 33:873-89*).

Twenty-four hours after reaching the duodenum, most immature trematodes already penetrate the gut wall and 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 reach the liver usually via transcoelomic (through the abdominal cavity); in some cases, hematogenous route is also described. The process of migration through the liver parenchyma lasts for 5-6 weeks. Some trematodes can accidentally penetrate the hepatic veins, reaching the systemic circulation and locate in unusual sites, particularly in the lungs, which was a finding of this report (4,1).

In general, the fluke reaches the bile ducts and remains there for 2 to 3 months where it develops the sexual maturity and releases the eggs through the bile. Also, during this phase the liver of the infected animal develop chronic lesions, which is the most common form, and clinically is seen as a chronic wasting disease. On gross examination of these cases are observed bile duct thickness and atrophy of the hepatic lobe. Microscopically, a proliferation of fibrous connective tissue on the periportal region associated with bile ducts hyperplasia and cholangiohepatitis are visualized (4,2).

However, when there is a massive larval migration through the hepatic parenchyma over a short period of time, 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 on the reported cases, but the death of the animals were linked to the acute lesions.

The gross lesions observed of fibrinous perihepatitis in addition to multiple multifocal hemorrhagic linear areas that was 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 corroborates with this classification (3).

References:

1. Cullen JM, Stalker MJ. Liver and Biliary System. In: Maxie MG, editor. Jubb, Kennedy & Palmer's Pathology of Domestic Animals: Vol. 2. 6th. Ed Philadelphia: Elsevier; 2016. p. 258-352.

2. Hashemnia M, Rezaei F, Nikousefat Z, Ghashghaii A. Acute caprine fasciolosis: a case with unusual migration to lung. J Parasit Dis. 2015; 39(3):514–517.

3. Stuen S, Ersdal C. Fasciolosis—An increasing challenge in the sheep industry. Animals. 2022 8;12(12):1491.

4. Tessele B, Brum JS, Barros CSL. Lesões parasitárias encontradas em bovinos abatidos para consumo humano. Pesq Vet Bras. 2013; 33(7):873–89.

*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://davisthompsonfoundation.org/diagnostic-exercise/)

Associate Editor for this Diagnostic Exercise: Saulo Pavarini
Editor-in-chief: Claudio Barros