# Diagnostic Exercise

## From The Davis-Thompson Foundation*

**Case #: 108 Month: November Year: 2018**

*Answer Sheet*

**Title:** Hepatic fascioliasis (*Fasciola hepatica*) in a cow

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**Microscopic Images:**

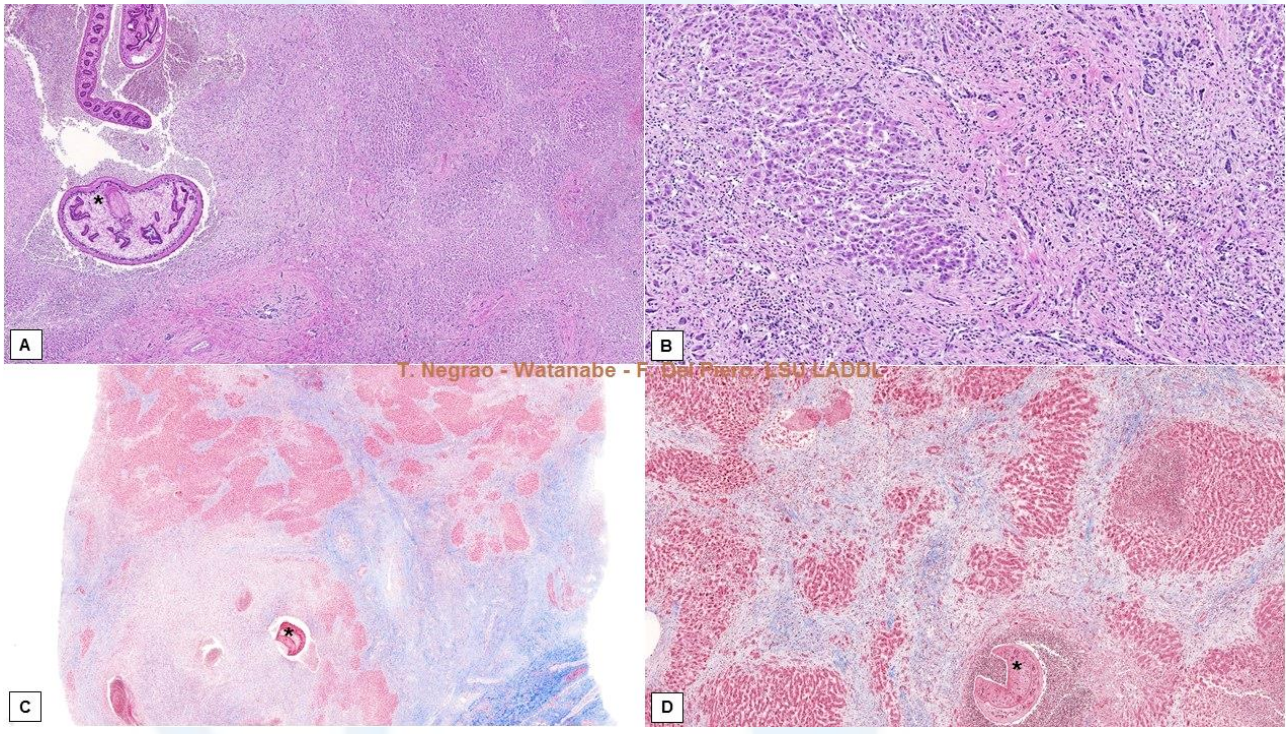


**Figure 3.** Cow. Hepatic fascioliasis (*Fasciola hepatica*). Liver (A-D). (A) More than 80% of the examined section is characterized by effacement of the normal parenchyma by fibrous connective tissue that bridges portal areas. Immature forms of *F. hepatica* (*) (Figures 3A, C, and D) are embedded within areas of necrosis mixed with fibrin and inflammatory exudate composed of numerous viable and degenerate eosinophils and neutrophils, and moderate numbers of macrophages and lymphocytes. H&E, 2.5x. (B) There is marked bile duct epithelial

hyperplasia within areas of marked fibrosis. H&E, 10x. (C). The abundant fibrous connective tissue entraps islands of the remaining hepatocytes, which are variably compressed, dissociated, and atrophic. Trichrome of Masson stain, subgross photograph. (D) Higher magnification of Figure 3C. Masson's Trichrome stain, 5x.

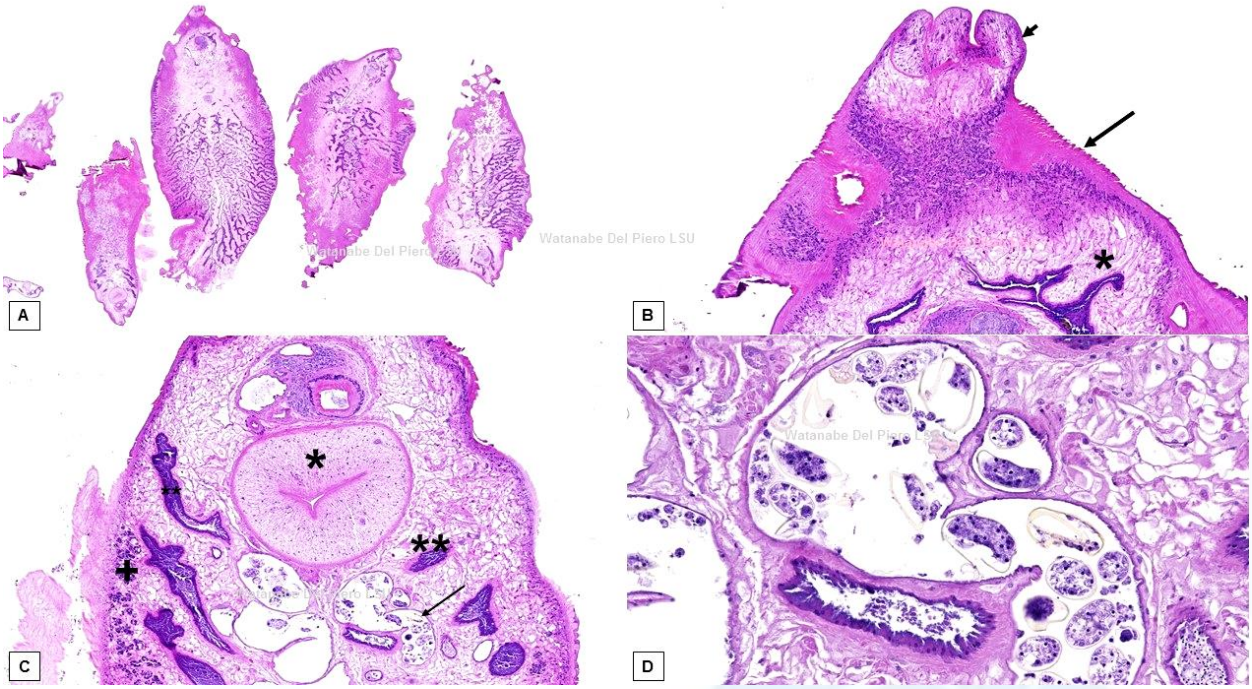


**Figure 4.** Tremadode. *Fasciola hepatica*. (A) Subgross histologic photograph of the trematodes shown in Figure 2E. H&E. (B) Note the lack of body cavity, which is filled with parenchyma (*). Note the anterior oral sucker (small arrow), and tegument with spines (long arrow). H&E, 5x. (C) The body contains a ventral sucker (*), paired convoluted intestinal ceca (**), peripherally located vitellaria (yolk forming glands) (+), and one ovary containing yellow-sheld eggs (long arrow). The paired testes are not represented in this section. H&E, 5x. Higher magnification of the ovary with embryonated eggs with yellow to light green shells. H&E, 20x.

**Morphologic Diagnoses:**

Liver: Cholangiohepatitis and capsulitis, lymphohistiocytic, neutrophilic and eosinophilic, chronic, severe, diffuse, with severe portal bridging fibrosis and intralesional adult trematodes (morphologically consistent with *Fasciola hepatica*).

Abdominal cavity: Peritonitis, serofibrinous, chronic, diffuse, severe.

**Typical Gross Findings:** Bile ducts standing out on the visceral surface as white, firm, branching cords with occasional mineralization ("pipe stem" appearance in cattle), bile ducts containing dark brown fluid of viscous or near solid consistency, migratory pathways in the liver characterized by hemorrhagic tortuous tunnels (2-3 mm in diameter), occasional acute or

chronic peritonitis due to immature fluke migration to the liver, hilar lymph nodes with black iron-porphyrin pigments, leaf-shaped, ~2.5 cm long flukes in the bile ducts and gall bladder.

**Typical Microscopic Findings:** Fresh tunnels filled with blood, degenerate hepatocytes, infiltration of eosinophils, lymphocytes, and macrophages, portal areas with irregular fibrosis, immature flukes encysted in the parenchyma, mature flukes in enlarged bile ducts causing cholangitis (long-standing or heavy infestation).

**Cause:** *Fasciola hepatica*

**Discussion:**
This is a case of severe fluke infestation with severe chronic cholangiohepatitis, eosinophilic "abscesses", associated fibrinous peritonitis, multicavitary effusion, and pulmonary atelectasis. The trematode morphology is consistent with *Fasciola hepatica*. The polyserositis was an unusual finding and perhaps caused by the inoculation of bacteria by the parasitic tegument spines. Hypoalbuminemia associated with the severe liver disease may also have contributed to the multicavitary effusion.

Hepatic fascioliasis is observed in numerous vertebrates, most often in cattle, sheep, pigs, and horses. Large animals generally tolerate the infestation, whereas severe disease may develop in sheep due to continuous hepatic parenchymal parasitic migration. The gall bladder with numerous parasites is a feature observed in cattle and humans. The poor body condition of the cow in this case was likely the result of the severe fibrosis and loss of hepatocytes leading to reduced synthesis of plasma protein, abnormal production and excretion of bile, inadequate bilirubin, carbohydrate, and lipid metabolisms, and decreased immune function.[2] The *F. hepatica* antigen Teg (tegument) can suppress the immune response of the mammalian host. Bovine lungs lesions secondary to aberrant fluke migration are quite common. They are grossly characterized by subpleural, ~1 to many cm diameter nodules of thinly encapsulated abscess. [2] Evidence of aberrant fluke migration was microscopically noted in the lung of this cow as well.

The life cycle of *F. hepatica* consists of the following stages: egg (within the feces) > embryonated egg > myracidium (aquatic stage, penetrates the snail) > free swimming cercariae (encyst on water plants) > ingestion of water plants > intraduodenal excystation > liver migration.[1] In brief, *Fasciola* has the following organs: oral sucker, larger sucker (acetabulum), tegument with syncytial epithelium and spines, mouth, pharynx, esophagus, blind large intestine with ceca, excretory canal, excretory pore, flame cell (protonephridia), nerve ganglia, esophageal nerve ring, genital atrium, genital pore, testes with testicular tubules and sperm, vas deferens, seminal vesicle, ejaculatory duct, genital atrium, prostate glands, tubular ovary, oviduct, vitelline duct, uterus, yolk reservoir atrium, and Mehlis' glands.[3]

*F. hepatica* reproduces both sexually, via the hermaphrodite adult flukes, and asexually.[3] The miracidia can reproduce asexually within the intermediate host. *F. hepatica* relies on extracellular

digestion, which occurs within the intestine of the host. The waste material is egested through the mouth. The non-waste matter is adsorbed through the tegument and the general surface of the fluke. The tegument facilitates this adsorption by containing many small folds that increase the surface area.[1-3] Adult flukes respire anaerobically. Glycogen taken from within the host is broken down via glycolysis to produce carbon dioxide and fatty acids. This process provides the fluke with energy. In contrast, the free-living miracidia stages of the parasite generally develop within oxygen-rich environments. It is therefore believed that the free-living stages of the parasite respire aerobically to gain the most energy from their environment. The area where this cattle grazed is probably densely populated by trematodes and their intermediate hosts. Numerous limneid snails, generally one genus per continent, can be the intermediate host. This outbreak is a demonstration of the economic significance of this parasite and the necessity to perform regular anthelminthic programs.

**References and Recommended literature:**

1. Centers for Disease Control and Prevention. CDC 24/7: Saving Lives, Protectin PeopleTM. 2013. Parasites - Fascioliasis (Fasciola Infection). [ONLINE] Available at: https://www.cdc.gov/parasites/fasciola/biology.html. [Accessed 23 March 2018].
2. Cullen JM and Stalker MJ. 2016. Liver and biliary System, p.258-352. In: Maxie MG (Ed.), Jubb, Kennedy, and Palmer's Pathology of Domestic Animals. 6th ed. Elsevier, St. Louis.
3. Gardiner CH and Poynton SL. 2006. Morphological characteristics of trematodes in tissue section. p. 46-49. In: Gardiner CH and Poynton SL (Eds.), An atlas of metazoan parasites in animal tissues. 1st ed. Armed Forces Institute of Pathology, Washington, DC.

*The Diagnostic Exercises are an initiative of the **Latin Comparative Pathology Group (LCPG)**, the Latin American subdivision of The Davis-Thompson Foundation. 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 CL Davis website (http://www.cldavis.org/diagnostic_exercises.html).

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