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

Fatty liver hemorrhagic syndrome in a hen

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Necropsy:

Diffusely pale yellow, soft, friable liver with focal rupture of the right lobe and regionally extensive intrahepatic and coelomic hemorrhage (Fig.1)

Follow-up questions:

- Morphologic diagnosis
- Microscopic features
- Name the disease

ANSWERS

Morphologic diagnosis:

Hepatic lipidosis, liver lobe rupture, and intrahepatic hemorrhage with concurrent intracoelomic hemorrhage (Fig. 1).

Gross findings (Fig. 1).

Diffusely pale yellow, soft, friable liver with focal rupture of the right lobe and regionally extensive intrahepatic and coelomic hemorrhage.



*The Diagnosti 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/

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Microscopic findings:

Diffusely, the hepatocytes have variably-sized clear vacuoles that displace and compress the nuclei (Figs. 2 and 3). The vacuoles are discrete, well-defined, round, and sometimes coalesce. There is hepatic cord disorganization with multifocal to coalescing pools of extravasated erythrocytes disrupting and dissecting the hepatic lobules (Fig. 3). The reticulin fibers of the hepatic sinusoids are fragmented in the areas of hemorrhage (Fig. 4).

Name the disease:

Fatty liver hemorrhagic syndrome; Hemorrhagic liver syndrome

Comments:

Fatty liver hemorrhagic syndrome (FLHS) affects chickens worldwide and is the most common noninfectious



Figure. 1. Diffusely pale yellow, soft, friable liver with focal rupture of the right lobe and regionally extensive intrahepatic and coelomic hemorrhage

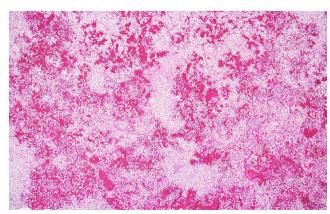


Figure 2. Liver. Multifocal to coalescing hemorrhages with severe diffuse hepatocellular lipidosis. H&E.

cause of mortality in backyard layers in Northern California (1, 4). It was first reported in 1956 and initially corresponded with the system of confining birds to cages. FLHS has been associated with high-energy diets in conjunction with limited physical activity. The syndrome was replicated when hens were force-fed and may be caused by multiple simultaneous factors (1). Caged layers as well as obese backyard laying hens are susceptible to FLHS development, in which livers become enlarged, light orange, and friable, with hemorrhages located within the parenchyma (2). Clinical signs include a sudden drop in egg production and sudden death with pale heads, particularly in obese laying hens (3,4). Dead birds are pale from hypovolemic anemia and have torn livers and blood clots in the abdomen. Hematomas may also be found in clinically healthy birds within the same flock. Significant amounts of fat are found in the coelomic cavity and around viscera, along with an enlarged, pale, friable, hemorrhagic liver (1,4).

Fatty liver hemorrhagic syndrome is considered a misnomer as a significant number of animals with this syndrome do not have fatty livers (4).

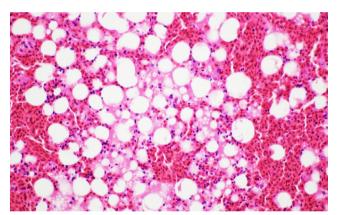


Figure 3. Liver. Diffuse hepatocellular lipidosis with disruption of the hepatic cord architecture and hemorrhage. H&E.

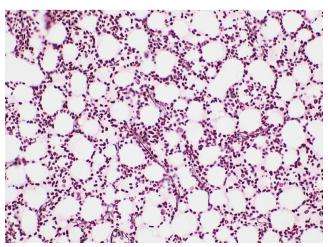


Figure 4. Liver. Fragmented reticulin fibers (arrows) in the areas of hemorrhage. Reticulin stain.

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Microscopic examination demonstrates swollen hepatocytes, sometimes with intracytoplasmic fat vacuoles, and hemorrhages of variable severity and small, irregular masses of eosinophilic material within the parenchyma, which is likely a product of plasma protein. The cause of FLHS is multifactorial and can be due to hormonal, environmental, genetic, and nutritional factors. The pathogenesis of FLHS involves an increase in fat content in the liver, associated with the onset of egg production and the influence of estrogen. High temperatures have also been shown to put birds in a positive energy balance, one of the risk factors for FLHS. Consumption of high-energy diets by birds with limited exercise leads to excessive fat deposition in the liver, which disrupts and weakens its architecture and vasculature. FLHS is associated with the lysis of the hepatic reticulin framework and consequent liver hemorrhage, which is based on the severity of reticulolysis. Excessive lipid metabolism that occurs in obese laying hens has been suggested to cause oxygen radical-mediated destruction of the reticulin framework. Hepatic hemorrhage may also be caused by focal necrosis of hepatocytes, leading to vascular injury (3,4).

Reducing hen obesity has been the only successful preventive strategy. Providing supplemental lipotropic agents such as vitamin E, vitamin B12, biotin, methionine

and choline may help reduce incidence of FLHS (1, 2, 4). Reducing heat stress as well as minimizing mold in barn environments, particularly in feed, may prove beneficial (1).

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