

Diagnostic Exercise

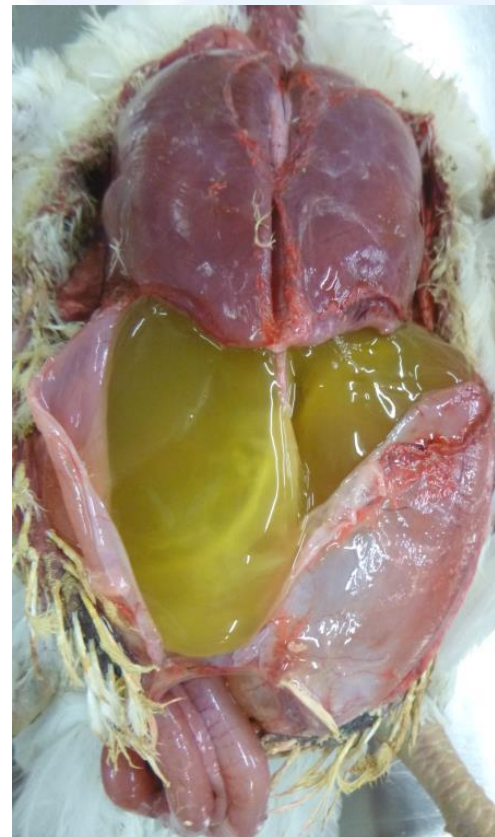
From The Davis-Thompson Foundation*

Case #: 145 Month: May Year: 2020

Contributors: Cleide Sprohnlé-Barrera, DVClinSc candidate, Anatomic Pathology Resident, & Associate Professor Rachel Allavena BVSc (hons), BVBIOL, GradDip (Anatomic Pathology), GCHEd, MANZCVS, PhD, DACVP. School of Veterinary Science, University of Queensland, Gatton, Australia.

Clinical History: This Ross broiler cockerel was one of six birds found deceased on day 28 of a research trial, and was part of an experimental heat-stress group. The research study was investigating the growth, immune and antioxidant responses of heat stress-exposed broilers fed variously supplemented diets. Clinical observations included lethargy, respiratory distress, and dark skin, with an overall 2% mortality rate.

Gross Images:



Figures 1 and 2

Necropsy Findings: Diffusely the skin and muscles were dark red, with congestion of peripheral vessels. The celomic cavity was 80% distended and filled with 225 ml of clear yellow gelatinous fluid (Figure 1 and 2). The pericardium contained 4 ml of clear yellow fluid and the right ventricular wall and atrium were dilated. The right ventricular wall was 2 mm and the left ventricular wall was 2.5 mm thick (Figure 3). The lungs were diffusely dark red with pink steady froth oozing out (Figure 4). The liver had rounded edges and there was a 10 x 15 x 10 mm subcapsular cyst filled with clear yellow fluid within the visceral surface of the right liver lobe (Figure 5).



Figures 3 and 4: Heart (left) and Lungs (right)



Figure: 5 Liver

Disease: Ascites syndrome

Microscopic findings:

Histological morphological diagnosis:

Heart (Figure 5): Myocardial fibers disarray and fibrosis, chronic, severe (consistent with dilated right ventricular cardiomyopathy).

Liver (Figure 7): Hepatic necrosis (coagulative), acute, mild.

Lungs (Figure 8): Pulmonary congestion and oedema, peracute, moderate

Discussion: The modern broiler is very prone to develop Ascites Syndrome, probably due to genetic selection for rapid growth rate and efficient feed conversion. The subsequent high rate of metabolism and relatively reduced cardiovascular capacity lead to progressive pulmonary arterial hypertension and *cor pulmonale* (Gupta, 2011; Wideman et al., 2013). Resulting hypoxia could and pulmonary hypertension would cause a pressure and volume overload in the right heart leading to right ventricular hypertrophy and dilation, and subsequent right heart failure (Bagheri Varzaneh et al., 2017; Janwari et al., 2018). Right heart failure results in venous hypertension, which is commonly associated with the development of ascites and congestive hepatopathy. The elevated central venous pressure extends to the hepatic veins and sinusoids, causing congestion, dilation of sinusoidal fenestrae, and exudation of protein and fluid into the space of Dissé, impairing diffusion of oxygen and nutrients to hepatocytes leading to necrosis (Wilson et al., 1988; Wideman et al., 2013). Other commonly reported histological features are likely the direct result of increased venous hydrostatic pressure caused by right heart failure, including myocardial degeneration; pulmonary congestion and edema; and hepatic degeneration and necrosis associated with decreased oxygen exchange (Wideman et al., 2013; Janwari et al., 2018).

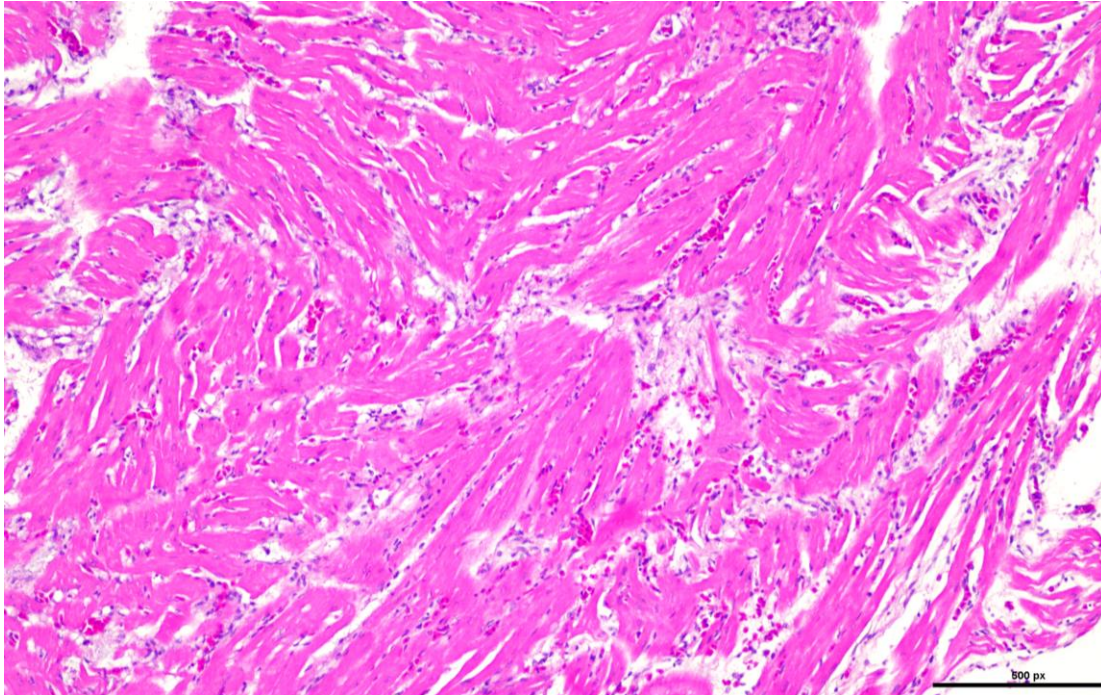


Figure 6: Heart, Hematoxylin & Eosin. 10x. Cardiac myofiber bundles are haphazardly arranged in between multifocal fascicles of fibrous connective tissue (myocardial fiber disarray and fibrosis).

f

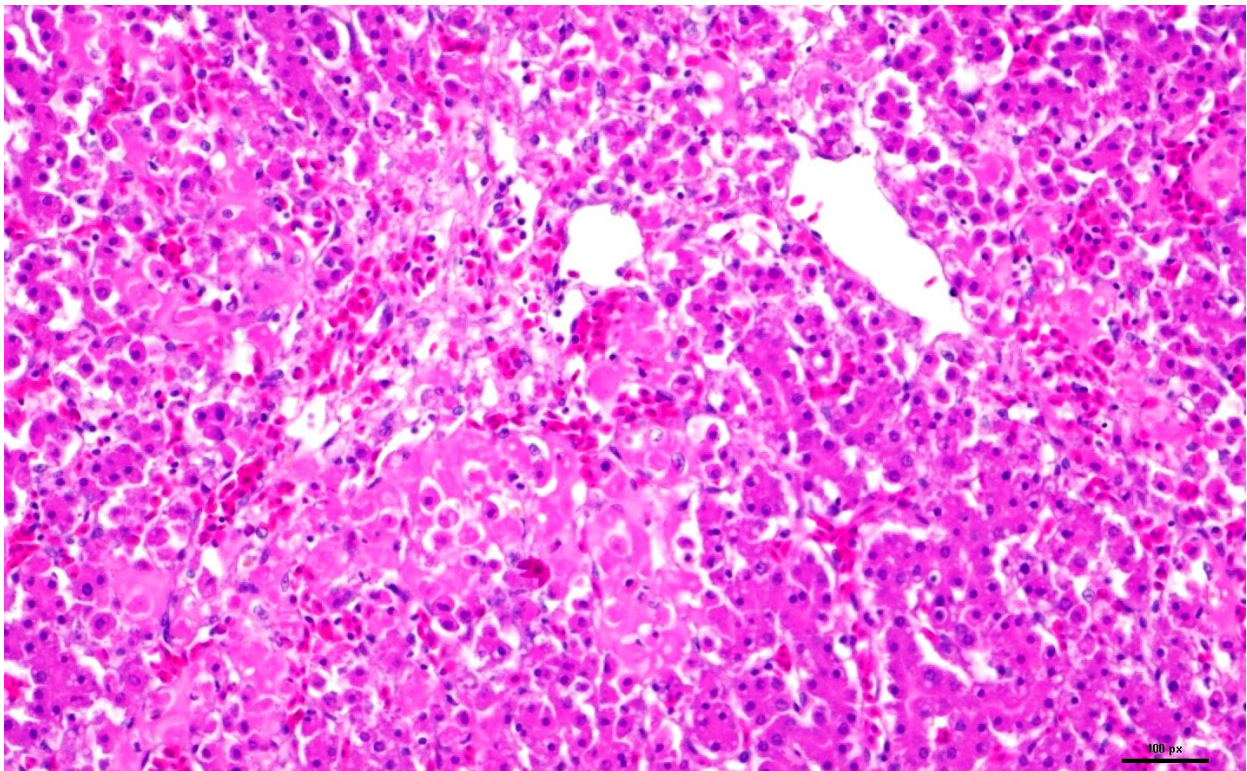


Figure 7: Liver, Haematoxylin & Eosin. 20x. There are multifocal to coalescing areas of the hepatic parenchyma with loss of cellular detail (coagulative necrosis).

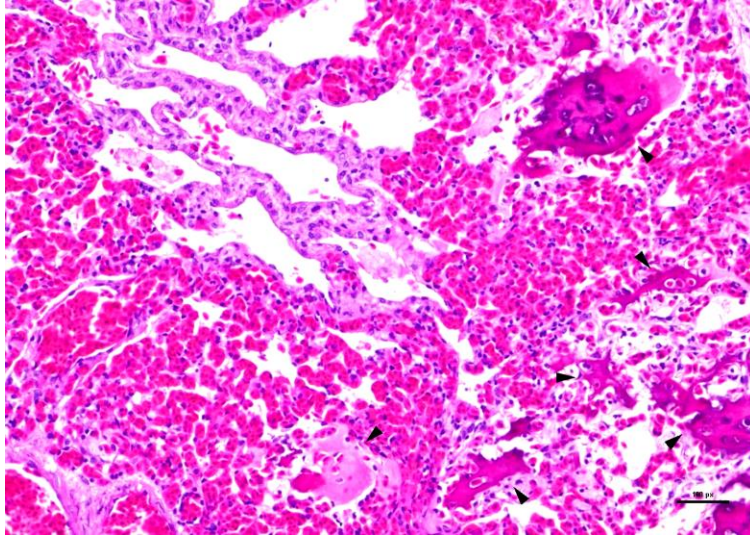


Figure 8: Lung, Hematoxylin & Eosin. 20x. Lung changes include dilated parabronchi and presence of more surfactant material than normal. There is also marked congestion, edema and thickening of the septal wall, as well as incidental osseous metaplasia (arrowheads).

References and Recommended Literature:

- Gupta, A. R. "Ascites syndrome in poultry: a review." *World's poultry science journal* 67.3 (2011): 457-468.
- Julian, R. J. "Physiological, management and environmental triggers of the ascites syndrome: A review." *Avian Pathology* 29.6 (2000): 519-527.
- Janwari, A.Q., et al. "Pathomorphological alterations due to pulmonary hypertension syndrome in broiler chicken reared under temperate climatic conditions of Northern Himalayas." *Journal of Entomology and Zoology Studies* 6 (2018): 1347-1353.
- Bagheri Varzaneh, M., et al. "The influence of oral copper-methionine on matrix metalloproteinase-2 gene expression and activation in right-sided heart failure induced by cold temperature: a broiler chicken perspective." *Journal of Trace Elements in Medicine and Biology* 39 (2017): 71-75.
- Wideman, R. F., et al. "Pulmonary arterial hypertension (ascites syndrome) in broilers: a review." *Poultry Science* 92.1 (2013): 64-83.
- Wilson, J. B., et al. "Lesions of right heart failure and ascites in broiler chickens." *Avian Diseases* (1988): 246-261.

*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).

Editor-in-chief: Claudio Barros; **Associate Editor for this Diagnostic Exercise:** Rachel Allavena.