Diagnostic Exercise
From The Davis-Thompson Foundation*


Clinical history, laboratory and necropsy findings: formalin-fixed pectoral muscle samples of slaughter-age broiler chickens were submitted without available information related to the clinical history, laboratory or necropsy findings of the affected flock(s).

Figure 1: Transverse section (HE, 4X): Diffuse proliferation of fibrous tissue is present in the interstitium.
Figure 2: Transverse section (HE, 20X): Several myofibers have degenerative and necrotic changes (e.g. fragmentation). Abundant adipose tissue (*) expands the interstitial and...
perivascular areas. Perivascular and intramural, mild to moderate lymphocytic infiltration is seen in a blood vessel (black arrow).

**Figure 3:** Sagittal section (HE, 40X): Mild inflammatory cell infiltration mainly composed of heterophils and macrophages is evident among few remaining necrotic myofibers (‡).

**Figure 4:** Sagittal section (HE, 20X): Fragmentation and inflammatory cell response is apparent within some myofibers (†).

**Figure 5:** Sagittal section (HE, 60X), higher magnification of Figure 4: Detail of the inflammatory cells, mainly heterophils and macrophages, that infiltrate a fragmented myofiber.

**Figure 6:** Sagittal section (HE, 40X): Perivascular and intramural, severe lymphocytic infiltration in an interstitial blood vessel with partially obliterated lumen.

**Figure 7:** Transverse section (Gomori’s trichrome stain, 10X): Extensive proliferation of interstitial fibrous tissue is evident.

1-Microscopic description: Both longitudinal and transversal sections of the pectoral muscle have prominent degenerative and necrotic changes. Many myofibers show loss of striations, hyalinization, and fragmentation. Heterophils and macrophages infiltrate scattered affected myofibers. Similar mild inflammatory infiltrate is also noted in the interstitium, together with interstitial fibrous and adipose tissue deposition. Several blood vessels have variable degrees of
perivascular lymphocytic infiltration, with and without intramural infiltration and vascular luminal occlusion.

2-Microscopic morphologic diagnosis: Skeletal muscle: Myocyte degeneration and necrosis, multifocal to coalescing, severe, with interstitial fibrosis, lipidosis, vasculitis, and mild heterophilic and histiocytic inflammation.

3-Typical gross findings:
Wooden breast: Focally extensive or multifocal, hard, bulging, pale areas of the pectoralis major muscle.
White striping: Variably sized, white lines or bands parallel to the surface of the pectoralis major muscle fibers.

4-Associated clinical pathology finding: Increased serum levels of creatine kinase (CK).

5-Name of the condition/s: Wooden breast/white striping.

6-Differential diagnoses: Vitamin E, selenium and sulfur-containing amino acid deficiencies; deep pectoral myopathy; pale, soft and exudative meat; ionophore (e.g. monensin, lasalocid, salinomycin, narasin) intoxications; Senna occidentalis intoxication; clostridial myositis accompanying gangrenous dermatitis; mycotic myositis; and myositis due to injection site injury.

Discussion: Although deep pectoral myopathy, which affects the pectoralis minor muscle in chickens and turkeys, is widely known as one of the most studied breast degenerative conditions of commercial poultry, both wooden breast (WB) and white striping (WS) are currently identified as emerging myodegenerative presentations of the pectoralis major muscle (PMM) in chickens causing high economic losses in the poultry industry worldwide. It was recently proposed that both gross presentations, which can be seen separately or together, belong to the same condition, sharing a common pathogenesis and microscopic features.

Several authors concluded that a genetic basis could be underlying WB/WS. A recent study also demonstrated that WB is directly related to localized hypoxia, oxidative stress, increased intracellular calcium and muscle fiber-type switching in affected PMM. Several factors are considered to increase the incidence of this condition, including the selection of genotypes with higher breast-yield, increased growth rate, higher slaughtering age, higher proportion of males at slaughter, and diets with higher level of energy.

The grow-related metabolic stress of affected myofibers, particularly those of the breast, leads to accumulation of intracellular calcium and production of reactive oxygen species. Then, both can activate lipases and proteases, which can cause alterations of myofiber membranes. The sarcoplasmic and myofibrillar protein degradation and fiber necrosis are accompanied by fibrosis and lipidosis, altogether corresponding to the typical findings of WB and WS, with the consequent nutritional value deterioration of the affected breast.
References and Recommended literature:

Note from the contributor: I would be pleased to hear from colleagues who have diagnosed WB/WS in natural or experimental cases of broiler chickens (e-mails: danielgornatti@fcv.unlp.edu.ar/danielgornatti@gmail.com).

*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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