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

Strangles and infarctive purpura hemorrhagica in a horse

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History:

A euthanized 23-year-old Quarter Horse gelding with an ~1-week history of fever (102.5°F), clear nasal discharge and lymphadenopathy was euthanized.

Necropsy findings:

Externally, the ventral aspect of the neck at the level of the larynx was swollen. In this area, there was a ~20 cm diameter abscess that effaced the left retropharyngeal and submandibular lymph nodes and extended towards the adjacent parotid gland through multifocal draining tracts. The abscess contained thick light green pus and was lined by a thick fibrous capsule (Fig. 1). Multifocally, within the subcutis and most skeletal muscles, there were coalescing and dissecting hemorrhages. These changes were the most severe in the pectoral, longissimus dorsi and thigh muscles, where there was also subcutaneous and intramuscular edema (Fig. 2).

Histopathology:

Skeletal muscle: The vessels had one or more of the following changes (Fig 3A): hypereosinophilic, thickened vascular walls (fibrinoid necrosis); intramural infiltrates of degenerate neutrophils with karyorrhectic debris (leukocytoclastic

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vasculitis); and intraluminal polymerized fibrin aggregates (thrombosis). Adjacent muscle fibers had one or more of the following changes (Fig 3B): swelling and loss of cross striations (degeneration); hypereosinophilia, hypercontraction bands, and fragmentation (necrosis); subtle sarcolemmal deposits of granular basophilic material (mineralization); infiltrates of macrophages and neutrophils over necrotic cells; and rare proliferation of satellite cells and internalization and rowing of nuclei (regeneration). There were well-demarcated, whole muscle fascicles that displayed the mentioned degenerative and necrotic changes with peripheral hemorrhage (infarction; Fig. 3C). The adjacent interstitium (endo-, peri- and epimysium) was expanded by neutrophils, fibrin, hemorrhage, debris, edema and occasional fibroplasia.

Left submandibular and retropharyngeal lymph nodes and parotid gland: The lymph node tissue was effaced by viable and degenerate neutrophils surrounded by a fibrous capsule. Infiltrates of neutrophils were present in the interstitium of the parotid gland. There were rare, intralesional, ~1 um gram-positive cocci that often occurred in pairs or short chains intermixed with the neutrophils in the lymph node (Fig. 3D).



Figure 1. Proximal neck: Abscess within the left retropharyngeal and submandibular lymph nodes, with thick, light green pus.

Follow-up questions:

- Morphologic diagnoses:

Etiology:

- Name of the conditions:
- Pathogenesis:

ANSWERS:

Ancillary test results:

- *Streptococcus equi* ssp. *equi* was isolated from the submandibular lymph node.
- The isolate was typed as a "non-vaccine strain" via PCR for *SEM* and *SZP* genes.
- Fluorescent antibody testing on impression smears from affected skeletal muscles were negative for *Clostridium chauvoei, C. septicum, C. novyi* and *Paeniclostridium sordellii,* and there was no growth on aerobic and anaerobic cultures.

- Morphologic diagnoses:

1. Skeletal muscles: Leukocytoclastic vasculitis and fibrinoid necrosis with thrombosis and infarction, multifocal to coalescing, acute, severe

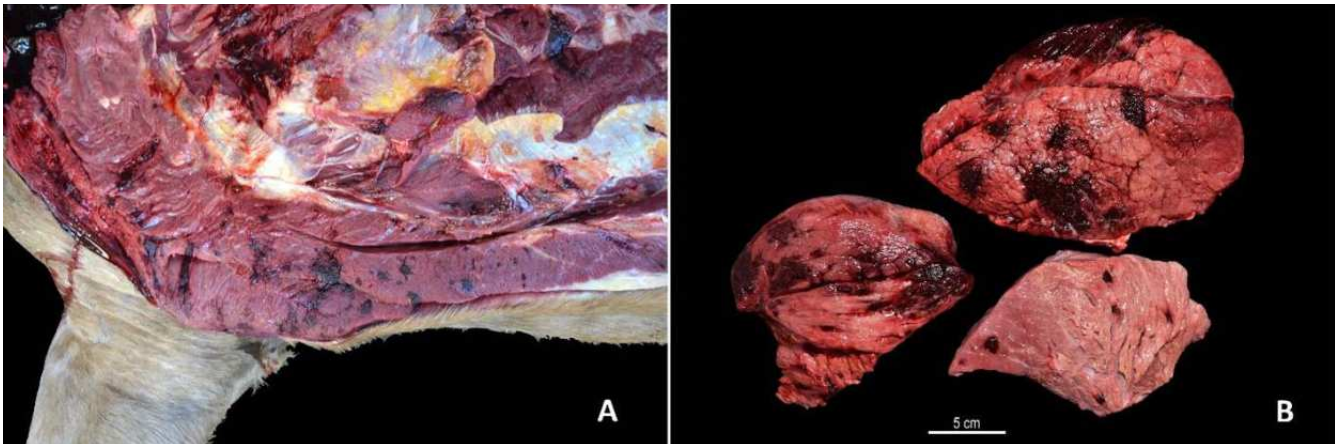


Figure 2. Multifocal to coalescing, well-demarcated hemorrhages within skeletal muscles of the pectoral region (A) and thigh (B).

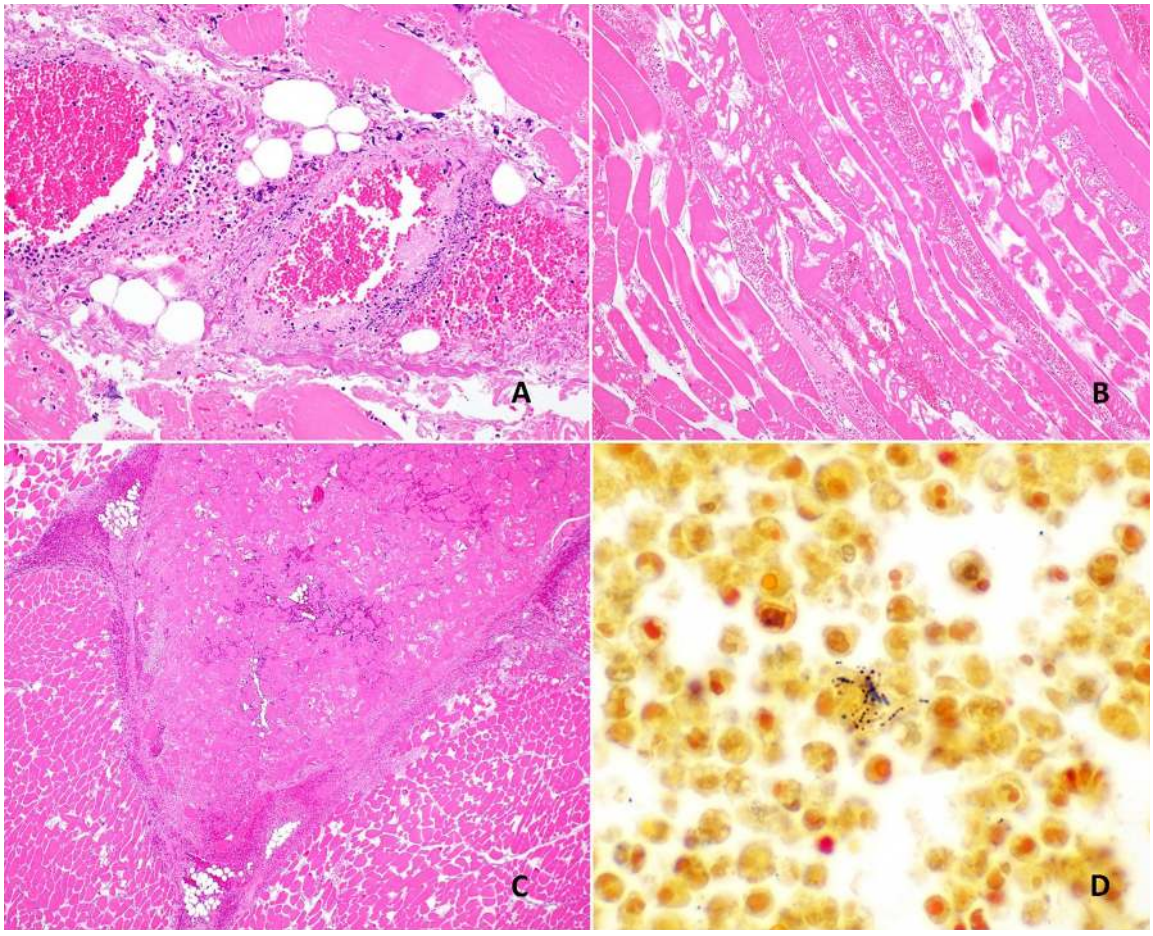


Figure 3. A, B & C: Skeletal muscles; D: Abscess. A: Fibrinoid necrosis of vessel walls and leukocytoclastic vasculitis characterized by a hypereosinophilic, thickened wall and intramural infiltrates of degenerate neutrophils with karyorrhectic debris. H&E, 200X. B: Muscle fibers with different changes consistent with degeneration and/or necrosis: swelling, loss of cross striations, hypercontraction bands, fragmentation and hypereosinophilia. H&E, 200X. C: A well-demarcated area of necrosis and hemorrhage affecting a whole muscle fascicle (infarct). H&E, 40X. D: Gram-positive cocci organized in pairs or short chains intermixed with the neutrophils. Gram stain, 400X.

2. Left submandibular and retropharyngeal lymph nodes: Lymphadenitis, suppurative, focally extensive, subacute with occasional intralesional gram-positive cocci
3. Left parotid gland: Parotiditis, suppurative, multifocal, moderate, subacute

- Etiology: *Streptococcus equi* ssp. *equi*

- Name of the conditions: Strangles with secondary infarctive purpura hemorrhagica

- Pathogenesis: Type III hypersensitivity reaction

S. equi ssp. *equi* infection (or vaccination) → Formation of IgA-*S. equi* M protein immune-complexes → Deposition of immune-complexes in blood vessel walls → Vasculitis and thrombosis → Local ischemia and infarction

Comments:

Strangles is an acute infectious and contagious disease of equids characterized by inflammation of the upper respiratory tract and abscessation of the retropharyngeal and/or submandibular lymph nodes; it typically affects young horses, although it can also occur in older animals [1, 4]. It is caused by *Streptococcus equi* ssp. *equi,* a gram-positive coccus that spreads through sub-clinically infected animals, which shed bacteria in nasal exudates, or via other fomites [1, 4].

Although most animals recover, ~20% of the cases develop complications such as purulent sinusitis, guttural pouch empyema, and facial nerve paralysis [1]. Less frequently, there are more severe complications, including pneumonia, pleuropneumonia, myocarditis, abscesses in internal organs (in particular in the mesenteric lymph nodes; also known as “bastard strangles”), and purpura

hemorrhagica [1, 4]. The latter is a type III hypersensitivity reaction associated with formation of *S. equi* M protein (SeM) and IgA immunocomplexes within vessel walls, with secondary vasculitis, hemorrhage and edema [4]. Other infectious agents such as *Streptococcus equi* ssp. *zooepidemicus*, equine influenza virus, equine herpesvirus-1, or *Corynebacterium pseudotuberculosis* can also induce purpura hemorrhagica [1].

Infarctive purpura hemorrhagica is a myopathy in which the SeM-IgA complexes deposit in the skeletal muscle vessel walls, and the subsequent vasculitis and thrombosis induce infarction of adjacent myofibers [3]. Several muscles may be affected at the same time, but the most commonly involved ones are the pectoral, hind limb adductor, and gaskin muscles [2]. Affected horses usually have pronounced elevations of creatine kinase, aspartate aminotransferase, and serum antibodies for SeM. Grossly, there are well-demarcated, multifocal hemorrhages in the affected muscles. Abscesses in retropharyngeal/submandibular lymph nodes and other organs may be observed concurrently. Histologically, there is leukocytoclastic vasculitis surrounded by well-demarcated areas of infarcted muscle fascicles with myofiber degeneration and necrosis, and hemorrhage. Inflammation is usually scarce, except in the borders of the infarcts. Vasculitis and infarction in other organs can also occur [2, 1].

Diagnosis is based on a history of previous exposure to *S. equi* ssp. *equi*, either via natural infection or vaccination, clinical signs, and gross and histologic lesions [2]. Since this is an immune-mediated disorder, no bacteria are isolated from affected muscles. Multiple muscles in different parts of the body are involved, as opposed to clostridial myositis, in which usually one muscle group is affected. Other differential diagnoses include systemic calcinosis, in which there is severe mineralization of muscle fibers, particularly in the gluteal muscles, associated with elevated calcium x phosphorus product; and other immune-mediated or infectious myopathies, such as rhabdomyolysis, atypical myopathy, or *Sarcocystis* spp. infection, which generally do not have such a remarkable vascular component histologically [2].

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