Diagnosis: Necrotizing myositis, multifocal to diffuse, with fibrinohemorrhagic to fibrinocellular neutrophilic exudation.

Clostridial myonecrosis (also known as clostridial myositis and malignantedema/gas gangrene).

Etiology: presumptive *Clostridium sordelli* (by IHC).

Typical Gross findings:

- Extensive myonecrosis (mottled dark red/tan musculature of the neck extending to the regions of the mid thorax, chest and the upper forelimbs).
- Exudative cellulitis/fasciitis (severe subcutaneous and intermuscular yellow/tan edema)
- Petechial hemorrhages in the laryngotracheal mucosa and serosal surfaces of viscera organs; subendocardial hemorrhage of left ventricular papillary muscle (toxemia).

Typical microscopic findings:

Multifocal to diffuse myonecrosis/myofiber degeneration with perimysial/endomysial fibrinohemorrhagic to fibrinocellular exudation (including scattered to numerous neutrophils), endomysial edema and vascular thrombosis.

Discussion: The rapid progression of severe tissue swelling after intramuscular injection and the histological findings of severe, acute myofiber necrosis and fragmentation with identification of rare intralesional IHC-
positive *C. sordelli*, led to the presumptive diagnosis of clostridial myositis. The failure to isolate clostridial bacteria from the affected muscle tissues on anaerobic culture could be explained by the aggressive antibiotic therapy.

The finding of rare numbers of intralesional *C. sordelli* by immunohistochemistry is the presumptive cause of the myonecrosis in this horse. The most common causative agents of clostridial myonecrosis/cellulitis in horses are *Clostridium perfringens* (type A); *C. septicum*, *C. chauvoei*, *C. novyi*; however *C. fallax* has also been reported (2,4,6,8). *Clostridium* spp. are large gram-positive, obligatory anaerobic rods that are ubiquitous in the soil and environment. They are also commensals of the skin, oral cavity, and intestinal tract. There is evidence for the presence of dormant clostridial spores in equine skeletal muscle (8).

Clostridial myositis is a rapidly progressive condition causing severe coagulative to liquefactive necrosis of
muscle, gas formation, and associated clinical signs of toxemia. It is a relatively rare disease and a series of specific events must occur to trigger specific cascade of pathological events (3, 8). Intramuscular injection with pharmacological compounds are known to cause soft tissue trauma and can create a favorable environment for clostridial growth (1). Both the toxin and the organism are required to be present in order to produce the disease; and attempts to produce clostridial disease by introduction of bacteria alone or toxin alone have been unsuccessful (5). Complications of intramuscular injections include type I hypersensitivity, mild local pain and inflammation, iatrogenic contamination with abscess formation, or, rarely, diffuse cellulitis or myositis and fulminant systemic toxemia (7). The survival rates of clostridial myositis range between 31-73% and a rapid diagnosis and early therapeutic interventions are crucial for success (3).

References and Recommended literature:


Please send your comments/questions to the whole LCPG list by hitting “reply to all”. A final document containing this material with answers and a brief discussion will be posted on the C. L. Davis website by the end of the current month (http://www.cldavis.org/lcpg_english.html).