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# Pathology in Practice

In collaboration with the American College of Veterinary Pathologists

## History

A 1.5-year-old 190-kg Quarter horse filly was submitted for necropsy after it died on a pasture following a brief episode of colic. The filly had been dewormed approximately 1 week prior to the onset of clinical signs (unknown medication). The filly had been kept on a pasture with cattle before being relocated to a pasture with horses of various ages approximately 8 weeks prior to death. This filly was 1 of 2 fillies from the pasture to develop colic and die within 24 to 48 hours of each other. A necropsy was not performed on the other filly.

## Gross Findings

The filly was underconditioned. The peritoneal cavity contained abundant red-brown, opaque, watery fluid with suspended strands of fibrin that coated all serosal surfaces. The mesenteric arteries and arteries supplying the right ventral colon and diaphragmatic flexure were regionally extensively and transmurally thickened with multiple nodules **(Figure 1)**. On cut section of the nodules, the arterial wall was transmurally and circumferentially expanded by concentric rings of tan tissue surrounding luminal accumulations of necrotic debris and hemorrhage sometimes centered on thin, tan nematode larvae measuring approximately 1 to 3 cm in length **(Figure 2)**. The wall of the left dorsal colon, pelvic flexure, and cecum were multifocally, transmurally dark green-red-brown and friable (necrosis).

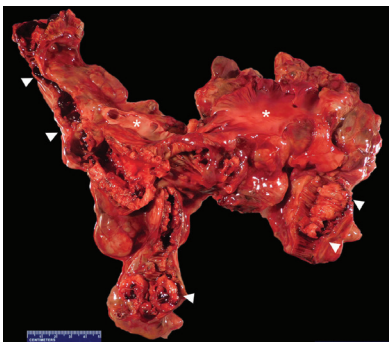

**Figure 1**—Photograph of the mesenteric arteries from a 1.5-year-old filly that died following a brief period of colic. Notice the normal vascular intimal surface (asterisks) and thickened, irregular vascular intimal surfaces (arrowheads).

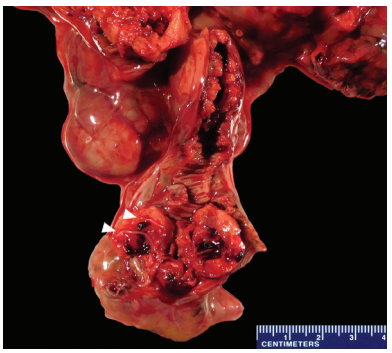

**Figure 2**—Photograph of an incised mesenteric artery nodule containing multiple 2- to 3-cm-long, thin nematode larvae (arrowheads).

## Histopathologic Findings

Microscopic evaluation of the mesenteric artery nodules revealed circumferential expansion and effacement of the tunica intima by mature fibrocollagenous stroma and granulation tissue with fibrin, neutrophils, eosinophils, macrophages, and hemosiderophages. The arterial lumina were filled with coagula of fibrin, hemorrhage, and neutrophils with numerous cross-sectional nematode larvae. Larvae measured approximately 200 to 300 µm in diameter, with a smooth eosinophilic cuticle, platymyarian musculature, and pseudocoelom with a central intestinal tract **(Figure 3)**. Sections of the left dorsal colon,

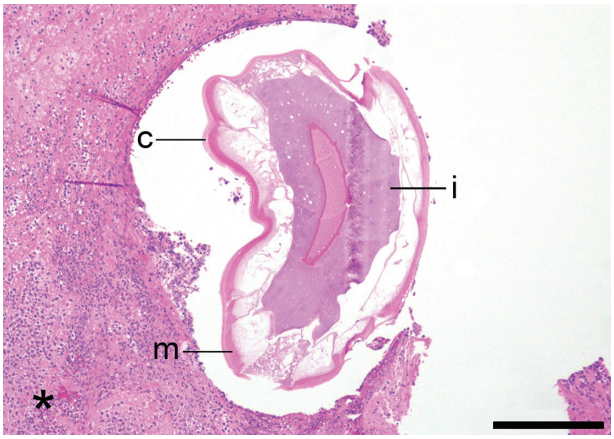


**Figure 3**—Photomicrograph of a cross section of a nematode larvae embedded within a coagulum of fibrin, neutrophils, and cellular debris (asterisk). The nematode larvae has a smooth eosinophilic cuticle (c), platymyarian musculature (m), and centrally located intestinal tract (i). H&E stain; bar = 200 µm.

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https://doi.org/10.2460/javma.22.05.0197

pelvic flexure, and cecum revealed transmural acute coagulative to lytic necrosis with abundant fibrin, edema, necrotic cellular debris, neutrophils, fewer eosinophils, and mats of coccobacilli. Multifocal medium-caliber vessels within the tunica muscularis were occluded by fibrin thrombi. The serosa was coated by mats of fibrin and neutrophils.

## Ancillary Diagnostic Results

A fecal flotation identified strongyle-type eggs (125 eggs/g). Larvae present within the mesenteric artery nodules were identified as large strongyle species based on the morphology of the buccal cavity, leaf crown, and teeth.

## Morphologic Diagnosis and Case Summary

Morphologic diagnosis: Proliferative mesenteric endarteritis with intralesional nematode larvae with acute necrosis of the large intestine, and fibrinosuppurative peritonitis.

Case summary: *Strongylus vulgaris* verminous arteritis in a filly.

## Comments

Equine strongylosis is caused by a group of large intestinal blood-sucking nematode parasites belonging to the family *Strongylidae*. The subfamily *Strongylinae*, or large strongyles, include the genera *Strongylus*, *Triodontophorus*, *Oesophogodontus*, and *Craterostomum*. Of these, species in the *Strongylus* genus exhibit extensive aberrant larval migration. *S vulgaris* is considered one of the most pathogenic intestinal parasites of equids due to the migration of the late-stage larvae through the mesenteric and systemic vasculature, resulting in vascular damage, tissue infarctions, and death. *S vulgaris* late-stage larvae exhibit a propensity for migration to the cranial mesenteric artery with extension into the ileocecocolic arteries. Other less commonly affected vessels include the aorta, renal, splenic, and celiac arteries. The larvae of other species within the *Strongylus* genera, including *Strongylus edenatus* and *Strongylus equinus,* typically exhibit aberrant migration to the liver, pancreas, and peritoneal cavity and do not cause the severe vascular lesions seen with *S vulgaris* infection.[1,2] To date, *Strongylus* spp infection is increasingly uncommon due to the usage of anthelminthics.[3,4]

*Strongylus* spp have a direct life cycle. Infection is initiated when third-stage larvae are ingested from a contaminated environment. Third-stage larvae penetrate the intestinal mucosa, where they molt to fourth-stage larvae that subsequently enter the vasculature system through submucosal small arterioles. Fourth-stage larvae migrate to the cranial mesenteric artery, where they continue to migrate for a 2- to 4-month period, causing vascular intimal damage. Fourth-stage larvae then mature to fifth-stage larvae (immature adults), which return to the wall of the large intestine and eventually reenter the lumen of the large intestine, where they mature to adults approximately 6 to 7 months after initial infection.[1,2,5] Some larvae become entrapped and die within the mesenteric arteries.[2]

Clinical signs associated with *S vulgaris* aberrant larval migration are most common in young horses and include nonspecific abdominal pain (colic), fever, and sudden death.[1,3] Necropsy findings associated with *S vulgaris* aberrant larval migration include, but are not limited to, nodular thickening of the mesenteric arteries, nonstrangulating large intestinal infarctions, fibrinous peritonitis, and possible intestinal rupture. The presence of intestinal antimesenteric subserosal hemorrhagic plaques (hemomelasma ilei) is considered an incidental finding associated *Strongylus* spp larval migration, with *S edentatus* being the most commonly implicated species.[2]

In conclusion, *S vulgaris* aberrant larval migration should be considered as a differential diagnosis in equids of all ages exhibiting signs of colic, with or without peritonitis, and sudden death.

## References


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