



Diagnostic Exercise

From The Davis-Thompson Foundation*

Case #: 83 Month: Aug Year: 2017

Answer Sheet

Contributor: Yianelly Rodriguez Ruiz¹; Tatiane Terumi Negrão Watanabe², DVM, MS, PhD; Ingeborg M. Langohr², BS, DVM, MS, PhD, DACVP. ¹Tuskegee University College of Veterinary Medicine, Tuskegee, AL 36088. ²Louisiana State University College of Veterinary Medicine, Department of Pathobiological Sciences and Louisiana Animal Disease Diagnostic Laboratory, Baton Rouge, LA 70803.

Clinical history, laboratory and necropsy findings: A 10-year-old American Miniature gelding presented for evaluation of being down and unable to rise. The horse had muscle atrophy on both fore and hind limbs, inability to rise, tachycardia, tachypnea, and dehydration. Mild gas distention of the small intestine was observed on abdominal radiographs. An intussusception of the small intestine or the ileocecal junction was suspected based on abdominal ultrasound. Due to poor prognosis, euthanasia was elected and the horse was submitted for post-mortem examination.

Necropsy Findings: In the distal duodenum, approximately 45.0 cm distal to the pylorus, is a 30.0 cm long segment of intestine that is five times thicker than the remaining small intestine (Figure 1A). The wall of the affected intestinal segment is 1.8 cm thick while the wall of the remaining duodenum and the jejunum is 0.1-0.2 cm thick. The mural thickness of the affected segment of duodenum is due primarily to a very prominent tunica muscularis externa. The affected region of the intestine also has a distinct serosa and a corrugated mucosa and submucosa, with overall narrowing of the lumen (Figure 1B). The increase in thickness of the wall of this intestinal segment is gradual at the proximal aspect of the lesion, whereas it returns rather abruptly to normal at its distal aspect. The wall of the ileum measures 0.5 cm in thickness. The wall of the esophagus, stomach and large intestine are unremarkable. In addition, the horse has muscle atrophy, reduced fat stores and, on the right body side, several cutaneous abrasions overlying bony prominences.

Microscopic Findings: In the severely thickened segment of the proximal small intestine noted at necropsy, particularly the inner circular layer but to a lesser extent also the outer longitudinal layer of the muscularis externa are thickened due to moderate to marked hypertrophy and presumably hyperplasia of the smooth muscle fibers (Figure 2).

Numerous Brünner's glands are present throughout this segment, confirming it to be duodenum. Similar but much milder muscular hypertrophy is also present in the ileum. The muscularis mucosae and the myenteric plexi are unremarkable.

Morphologic Diagnosis: Distal duodenum: Muscular hypertrophy and hyperplasia, segmental, marked; Ileum: Muscular hypertrophy, segmental, mild.

Condition: Idiopathic segmental muscular hypertrophy of the small intestine.

Possible Associated Lesions: Hypertrophy of the tunica muscularis in the esophagus and ileum.

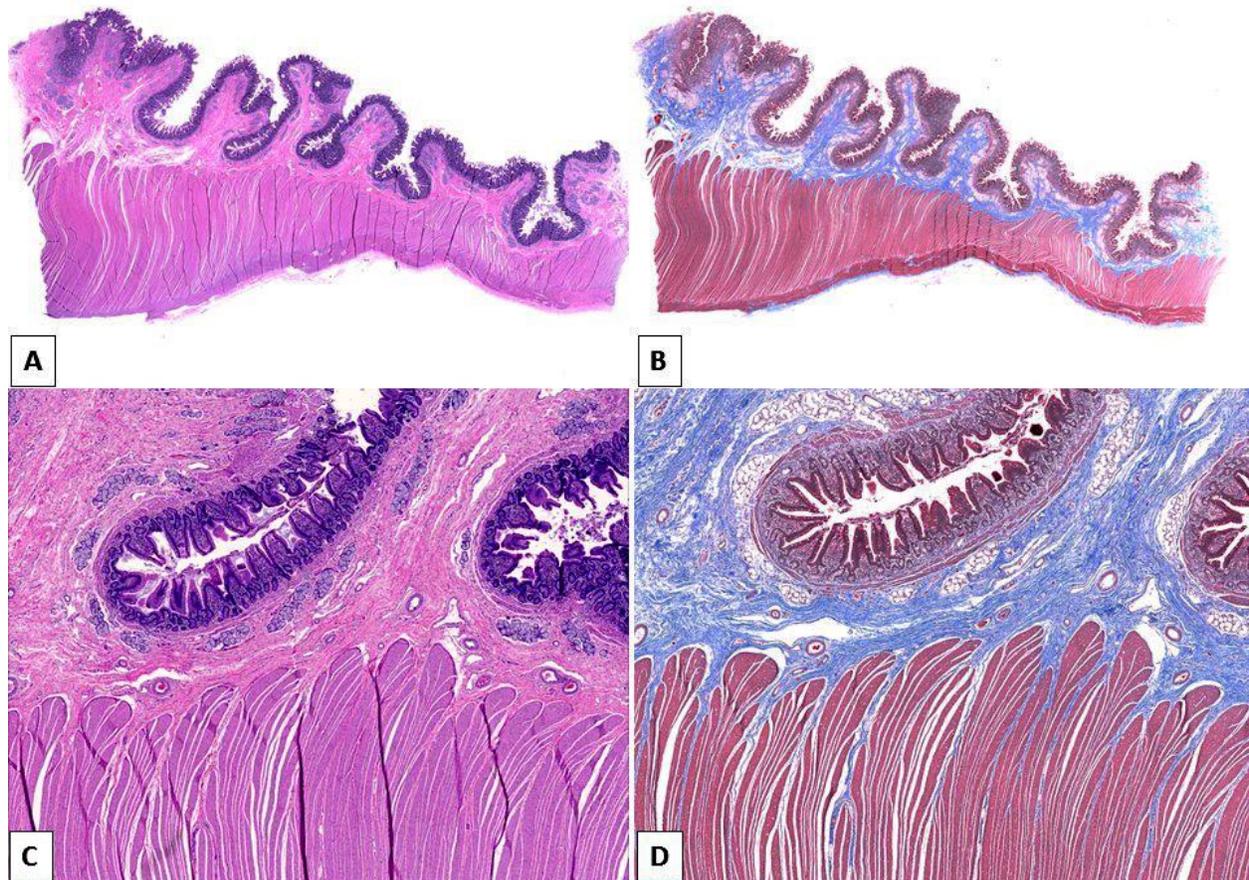


Figure 2. Horse, Idiopathic segmental muscular hypertrophy of the small intestine, Duodenum. (A) and (B) There is segmental hypertrophy of the tunica muscularis. H&E and Masson's Trichrome stains, respectively. Subgross photographs. (C) and (D) Detail of the hypertrophic smooth muscle of the tunica muscularis externa, affecting

particularly the inner circular layer, without fibrosis or inflammation. The overlying submucosa and mucosa are folded but otherwise unremarkable. H&E and Masson's Trichrome stains, respectively. 2.5x magnification.

Discussion: Idiopathic muscular hypertrophy of the intestine, most often observed in horses, has the potential to affect any segment of the small intestine.⁹ The median length of the affected small intestine is reportedly 1.0 m (ranging from 0.20 to 21.3 m).² Ileum is the most common affected site, but the jejunum and duodenum might have the alteration.^{3,8} A similar condition has been described in swine and a goat but, in contrast to horses, in these other animals it affected only the ileum and jejunum, respectively.⁹ No breed or sex predilection has been recognized in horses, but adult animals appear to be the ones most often affected (the median age of the affected horses is 10 years, ranging from 5–18 years).^{1,2}

There are two forms of the muscular hypertrophy of the small intestine: idiopathic (primary) and compensatory (secondary).^{2,4,8} Cases of idiopathic muscular hypertrophy do not have any overt cause, i.e., no detectable stenosis of the caudal intestine that would predispose the tunica muscularis of the cranial intestinal segment to undergo hypertrophy.^{2,3,4} In contrast, the compensatory form represents the response to a focus of chronic caudal intestinal stenosis.^{2,4} Clinical signs in horses include chronic or intermittent colic,^{2,3,5,7,8,9} weight loss,^{2,3,4,5,7,8,9} partial anorexia,^{2,4,9} and intermittent diarrhea.⁹ Histologically, there is hypertrophy of both the circular and longitudinal layers of the tunica muscularis externa,^{2,3,8} potentially predisposing to ileal impaction or leading to narrowing of the intestinal lumen with partial or complete obstruction ultimately causing pain.^{4,8} Pseudodiverticula have also been seen with idiopathic muscular hypertrophy in horses and pigs,^{7,9} as has full-thickness rupture, which can lead to septic peritonitis.^{2,8}

Horses with intestinal idiopathic muscular hypertrophy frequently have concomitant idiopathic hypertrophy of the tunica muscularis in other segments of the digestive tract as well, particularly in the esophagus.^{1,9} Clinical signs associated with esophageal dysfunction is very rare,¹ however, except for Friesian horses, in which marked caudal muscular hypertrophy of the esophagus is generally associated with megaesophagus and choke.⁶ In the present case, while there was mild thickening of the tunica muscularis externa of the ileum, the esophagus had no significant gross and histologic abnormalities.

The etiology and the pathogenesis of the idiopathic muscular hypertrophy of the intestine are still unclear.^{2,8,9} Possible causes include mucosal inflammation, autonomic imbalance producing uncontrolled peristalsis or prolonged spastic contraction of the ileocecal orifice, and obstruction of the ileocecal orifice by *Anoplocephala* spp, or large amounts of sand.^{1,2,8,9} Clinical diagnosis is a challenge. A tentative diagnosis can be made either by palpation per rectum or in surgery of a thickened segment of intestine² or by transcutaneous or transrectal abdominal ultrasound localizing the thickened muscular layer of the small intestine.^{3,7} Differential diagnoses should include intestinal impaction, intussusception, edema of the intestinal wall, inflammation, neoplasia, stricture, adhesions, spasmodic intestinal

contraction, or abnormalities of the ileocecal orifice.^{2,3} A definitive diagnosis in vivo requires exploratory celiotomy.^{2,4} Surgical resection of the affected segment is the treatment of choice;^{2,4} however, if the duodenum is affected, surgical treatment may not be possible due to the anatomic anchoring of the duodenum in the abdominal cavity.⁸ Often the condition is only diagnosed post-mortem after the animal was euthanized, as was also the case in this horse.

References:

1. Benders NA, Veldhuis Kroeze EJB, Van Der Kolk JH. Idiopathic Muscular Hypertrophy of the Oesophagus in the Horse: A Retrospective Study of 31 Cases. *Equine Veterinary Journal* 36(1), 2004:46-50.
2. Chaffin MK, Carmen Fuenteabla I, Schumacher J, Welch RD, Edwards JF. Idiopathic Muscular Hypertrophy of the Equine Small Intestine: 11 Cases (1980-1991). *Equine Veterinary Journal* 24(5), 1992:372-378.
3. Dechant JE, Whitcomb MB, Magdesian KG. Ultrasonographic Diagnosis - Idiopathic Muscular Hypertrophy of the Small Intestine in a Miniature Horse. *Veterinary Radiology & Ultrasound* 49(3), 2008: 300-302.
4. Hanson RR, Baird AN, Pugh DG. Ileal Impaction in Horses. *Compendium on Continuing Education for the Practicing Veterinarian* 17(10), 1995:1287-1293.
5. Kemper DL, Perkins GA, Schumacher J, Edwards JF, Valentine BA, TJ Divers, Cohen ND. Equine Lymphocytic-plasmacytic Enterocolitis: A Retrospective Study of 14 Cases. *Equine Veterinary Journal* S32, 2000:108-112.
6. Komine M, Langohr IM, Kiupel M. Megaesophagus in Friesian horses associated with muscular hypertrophy of the caudal esophagus. *Veterinary Pathology* 51(5), 2014:979-985.
7. Navas de Solís C, Biscoe EW, Lund CM, Labbe K, Muñoz J, Farnsworth K. Imaging Diagnosis - Muscular Hypertrophy of the Small Intestine and Pseudodiverticula in a Horse. *Veterinary Radiology & Ultrasound* 56(2), 2015:E13-E16.
8. Tóth J, Hollerrieder J. Idiopathic non dilatative muscular hypertrophy of the equine duodenum. *Pferdeheilkunde* 21(5), 2005:423-426.
9. Uzal FA, Plattner BL, Hostetter JM. Alimentary System. In: Jubb, Kennedy, and Palmer's Pathology of Domestic Animals. Maxie MG (Ed) 6th ed., St. Louis, MO: Elsevier, 2016. V.2, p.

*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).

Associate Editor for this Diagnostic Exercise: Ingeborg Langohr

Editor-in-chief: Vinicius Carreira

GPCC
L

