

All Choked Up: Esophageal Obstructions

Amelia Munsterman, DVM, MS, DACVS, DACVECC
Auburn University
Auburn, AL

Esophageal obstruction is the most common abnormality of the equine esophagus.¹ Causes can include ingestion of bedding, such as straw or wood shavings, poorly masticated feeds including apples, carrots or hay, as well as processed dry feeds such as beet pulp, pellets or hay cubes. Predisposing factors may include poor dentition, improperly moistened feeds (ie beet pulp), dehydration, or sedation, among others. Choke can also occur in horses that are inclined to bolting their feed, as well as in horses that have had esophageal impaction previously which caused an esophageal stricture or diverticulum. While the diagnosis of an intraluminal obstruction is relatively straightforward, resolution can be frustrating, and complications are often devastating for the future function of the horse.

Clinical findings in esophageal obstruction

While the classic signalment of a horse with an esophageal obstruction is the older horse with dental disease, the average age is actually quite lower in the literature, around 10.5 years on average.¹ There is no one breed that is predisposed to choke, however, Friesians have been noted to have esophageal pathology caused by a distal hypertrophy that may lead to similar clinical findings.² On examination, horses with an obstruction will often have saliva or feed-tinged mucous exiting both nostrils, and sometimes the mouth. Tachypnea and tachycardia may be present if the obstruction is chronic, due to dehydration, electrolyte abnormalities, and pulmonary inflammation from aspiration of feedstuffs. Horses may appear anxious, and may repeatedly stretch their neck, gulp, cough or retch.

Identification of esophageal obstructions

The first step in treatment of esophageal obstructions is to sedate the horse to lower the head for both diagnosis and treatment of the obstruction. Diagnosis is often confirmed by passing a nasotracheal tube down the esophagus to the obstruction, which will prevent the tube from passing into the stomach. If the obstruction is in the cervical esophagus, it may be palpable externally. If an endoscope is available, it would be preferable to pass it before passing a nasogastric tube, to assess the degree of trachea contamination prior to lavage, and to determine where the food is lodged and what type of feed stuff it appears to be. An endoscope will also allow the clinician to identify mucosal damage or lacerations that may complicate therapy. Obstructions are most commonly found in the proximal esophagus or at the thoracic inlet.¹

Advanced diagnostics, such as radiographs, are not necessary to diagnose a simple esophageal obstruction, and barium should not be administered to horses with esophageal obstruction due to the risk of aspiration and the development of barium pneumonitis. However, horses with recurrent episodes of choke should be evaluated thoroughly for morphologic or functional disturbances, including megaesophagus, esophageal stricture or diverticulum. Endoscopy can be cheaper and more informative than other diagnostics for the identification of esophageal strictures, and it can also be helpful in evaluating the severity of the damage after the obstruction has resolved.

Treatment of esophageal obstructions

Simple esophageal obstructions may pass into the stomach due to normal peristaltic waves on their own after a single dose of sedation, or may easily pass into the stomach with gentle pressure from the nasogastric tube. However, if the choke has not resolved on its own in 15-20 minutes, or with manual pressure from the nasogastric tube, esophageal lavage should be used to break down the impaction. The typical large bore nasogastric tube may be used for retrograde lavage. Alternatively, a cuffed endotracheal tube may be passed first into the esophagus, followed by the nasogastric tube inside its lumen, to reduce the aspiration of water and feed. Lavage should be performed only in a well-sedated animal to encourage fluid to drain from the nose, rather than down the trachea. Alternatively, the horse may be anesthetized to allow for better control of the airway. If the obstruction is a solid foreign body (ie. apples, carrots), use of the endoscope and a small biopsy instrument has allowed for piece-meal dissection of the obstruction where lavage alone was not successful. Finally, if progress is slow or difficult, the horse may be allowed time to rehydrate with intravenous fluids prior to a second attempt, to allow for softening of the impaction.

Sedation is used not only to make the horse more tractable for treatment, but also to promote relaxation of the esophageal musculature. Depending on the site of obstruction, sedation can be tailored to help dilate the musculature at the site of the impaction. Acepromazine (0.07 mg/kg, IV) is a phenothiazine tranquilizer that causes sedation by acting as a dopamine antagonist, as well as smooth muscle relaxation through antagonism of the alpha-1 receptor. It has been shown to work on the musculature of the distal esophagus, causing esophageal dilation and decreasing spontaneous swallowing reflex.³ While it could be useful to treat a distal esophageal obstruction, acepromazine should be used with caution in hypovolemic animals, due to the side effect of peripheral vasodilation. Alpha-2 adrenergic receptor agonists, alternatively, work on the skeletal muscle of the proximal esophagus by reducing

normal peristalsis, presumed to be caused by effects on the central nervous system. These effects have been demonstrated with both xylazine (0.5 mg/kg, IV) and detomidine (0.04 mg/kg), while detomidine may cause distention of the esophagus at the thoracic inlet as well.³ When combined with butorphanol (0.02 mg/kg), xylazine was effective in reducing the number of swallowing events, which could also reduce peristaltic waves. Guafenesin (25 mg/kg IV) can decrease spontaneous swallowing, but may cause significant ataxia in standing horses, even at this low dose. Of these medications, the alpha-2 agonists alone, and combined with butorphanol, produced the most dramatic effects on the esophagus.³ While oxytocin was once reported to possibly reduce esophageal tone, it has no effect on esophageal pressures in vivo.³ Smooth muscle relaxants, such as n-butylscopolammonium bromide (0.3 mg/kg, IV), have been purported to improve smooth muscle relaxation, and have been used anecdotally for esophageal obstruction in the lower esophagus. In experimental studies, this medication has been shown to eliminate the swallowing reflex in the distal third of the esophagus.⁴

Due to the accompanying dysphagia, all horses with esophageal obstruction aspirate feed and saliva to some degree, and the costs to a horse from aspiration pneumonia far outweigh the costs of prophylactic use of antimicrobials and their side effects. In all horses with choke, broad spectrum antibiotics (ie. potassium penicillin 22,000 U/kg, IV, QID and gentamicin 6.6 mg/kg, IV, SID, or trimethoprim sulfadiazine 22 mg/kg, PO, BID), including metronidazole for anaerobic bacteria (15 mg/kg, PO, TID) should be prescribed for a minimum of 5 days, and up to 2-8 weeks for confirmed aspiration pneumonia. While it would be tempting to associate the degree of feed contamination in the trachea noted on endoscopy with the risk of aspiration pneumonia, it has shown no diagnostic sensitivity for this complication.¹ However, the duration of the obstruction was more likely to correlate with an increase the risk of aspiration pneumonia. Non-steroidal anti-inflammatory medications should be provided, while monitoring hydration status, and clenbuterol (0.08 µg/kg, IV, BID) may be administered to improve mucociliary function and bronchodilation. Sucralfate (20 mg/kg, PO, QID) can also be used to treat esophagitis and minor mucosal irritation.

Horses should remain off feed for at least 24 hours for minor chokes, and up to 3-4 days for horse with severe injury to the esophageal mucosa. The horse may need to be muzzled during this time, and bedding should be removed from the stall to prevent ingestion. Fluids can be provided PO, or IV if severely dehydrated. Feed should be introduced gradually, and softened mashes of pelleted feed supplemented with mineral oil are recommended for 1-3 weeks based on the duration of the obstruction and the appearance of the esophagus on endoscopy. After this time, the normal diet can be gradually re-introduced. If the cause of the obstruction can be determined, dietary and management changes may help prevent re-obstruction. Prognosis is best for horses presented for the first time with an esophageal obstruction and for horses that resolve quickly with lavage.¹

Complications of esophageal obstruction

Esophageal stricture

Esophageal strictures can be a consequence of circumferential damage or extensive linear tears of the esophagus. The damage can be assessed by endoscopy after relieving the obstruction, and horses that may be predisposed should be reevaluated in 2-4 weeks to determine the duration of feed restriction, and need for further treatments including antibiotic and anti-inflammatory medications. If a stricture develops, the contracture that results from normal wound remodeling will cause the lumen to constrict to a minimal diameter at 30 days after the injury.⁵ However, remodeling of this scar tissue will continue for up to 60 days. Therefore, medical and dietary management of a stricture can be recommended, and can be successful, if extended for at least 2 months after the choke. If the horse has not resolved the stricture at this time, the modified diet may be maintained indefinitely, or surgical management, including bougienage, esophagomyotomy or esophagopexy may be considered for horses that continue to re-obstruct.⁶

Esophageal diverticula

There are two types of esophageal diverticula, traction and pulsion, and both are caused by esophageal trauma. Traction diverticula typically have a shallow body and wide neck. These characteristics, and the fact that peristalsis is not affected, mean that a traction diverticulum rarely causes clinical problems. Pulsion diverticula, however, appear as a protrusion of esophageal mucosa through a defect in the muscular wall. The narrow neck through the musculature predisposes it to impaction of feed, and can lead to esophageal rupture. Clinical signs of a diverticula will be similar to esophageal obstruction, in addition to the presence of diffuse swelling and possibly an external wound communicating with the diverticula. Most diverticula occur in the cervical neck, but pulsion diverticula have been reported in the thoracic esophagus.⁷ Diagnosis of esophageal diverticula is similar to that for esophageal obstruction, with the use of plain and contrast radiographs and endoscopy. Surgical repair should be recommended, especially for a pulsion diverticula.

Esophageal rupture

Esophageal rupture may occur secondary to pressure necrosis caused by the obstruction itself, hypovolemia, and the peristaltic waves of the esophagus attempting to move the obstruction aborally. It may also occur as a result of attempts to relieve the obstruction with the nasogastric tube and lavage. Clinical signs would be consistent with cellulitis, noted by a painful swelling and crepitus in the ventral neck, along with signs of endotoxemia. If the feed and air dissect caudally or if the rupture is intrathoracic, a pneumomediastinum or pneumothorax may result.⁸ Treatment involves establishing ventral drainage and bypassing the rent with an indwelling feeding tube placed through the defect or below it.

Idiopathic megaesophagus

Megaesophagus is a recently recognized disorder noted mainly in Friesian horses, with a possible X linked mechanism of transmission.^{2,9} Although it has been documented in Dutch Warmbloods and Welsh ponies,¹⁰ the incidence in Friesians in one report was 70 times that of other breeds, making it an important disorder to consider for a Friesian presenting with esophageal obstruction. Affected horses may show clinical signs of esophageal dysfunction and chronic dysphagia, including coughing, gagging, ptialism, nasal discharge, weight loss, anorexia, fever, pleural effusion, and recurrent bouts of esophageal obstruction. However, muscular hypertrophy of the esophagus may be silent and only identified on necropsy as an incidental finding.¹⁰

Diagnosis can be made with endoscopy, where a dilation of the esophagus will be visible, along with retrograde reflux of feed, and an abnormal appearance to the mucosal lining.⁹ Both plain and contrast radiography may also be helpful. On histopathology, megaesophagus can be confirmed by identification of gross hypertrophy of the tunica muscularis of the caudal esophagus.¹⁰ Many horses were also diagnosed with mucosa ulceration and fibrinous pleuritis consistent with chronic aspiration pneumonia. Esophageal tears can be concurrent with megaesophagus. While the cause of equine megaesophagus has not been determined, it was morphologically similar to esophageal leiomyomatosis (Alport Syndrome) seen in humans.² This condition is associated with a mutation in the gene encoding type IV collagen, resulting in hypertrophy of smooth muscle cells of the distal esophagus. Unlike neoplastic lesions, there is low cellularity, and no evidence of mitosis or cellular atypia. There is no treatment in humans, and in horses supportive care is recommended. To reduce the risk of obstruction, soft feeds, such as a mash or grazing, are recommended, as well as offering feed at chest height.¹¹

References

1. Feige K, Schwartzwald C, Furst A, Kaser-Hotz B. Esophageal obstruction in horses: a retrospective study of 34 cases. *Can Vet J.* 2000;41:207-210.
2. Komine M, Langohr, IMJ, Kiupel M. Megaesophagus in Friesian horses associated with muscular hypertrophy of the caudal esophagus. *Vet Pathol.* 2014;51:979-985.
3. Wooldridge AA, Eades S, Hosgood GL, Moore RM. Effects of treatment with oxytocin, xyazine, butorphanol, guaifenesin, acepromazine, and detomidine on esophageal manometric pressure in conscious horses. *Am J Vet Res.* 2002;63:1738-1744.
4. Bertone JJ, Hoover AM, Greene HM, Wickler SJ. N-butylscopolammonium bromide relaxes the distal equine esophagus: a mechanism for is purported usefulness in esophageal obstruction. In *Proceedings of the 57th Annual Convention of the American Association of Equine Practitioner.* Nov. 18-22, 2011, San Antonio, TX. p. 193-194.
5. Todhunter RJ, Stick JA, Trotter GW, Boles C. Medical management of esophageal stricture in seven horses. *J Am Vet Med Assoc.* 1984;185(7):784-787.
6. Lillich JD, Frees KE, Warrington K, et al. Esophagomyotomy and esophagopexy to create a diverticulum for treatment of chronic esophageal stricture in 2 horses. *Vet Surg.* 2001;30(56):449-453.
7. Yamout SZ, Magdesian KG, Tokarz DA, le Jeune SS. Intrathoracic pulsion diverticulum in a horse. *Can Vet J.* 2012;53:408-411.
8. Hassel DM. Thoracic trauma in horses. *Vet Clin Equine.* 2007;23:67-80.
9. Boerma S, Sloet Vnan Oldruitenborgh-Oosterbaan M. Megaesophagus in the Friesian horse: a hereditary problem? In *Proceedings of the 10th International Congress of World Equine Veterinary Association.* Jan. 28-Feb. 1, 2008, Moscow, Russia. p. 483-484.
10. Benders NA, Veldhuis Kroeze EJ, van der Kolk JH. Idiopathic muscular hypertrophy of the oesophagus in the horse: a retrospective study of 31 cases. *Equine Vet J.* 2004;36(1):46-50.
11. Broekman LEM, Kuiper DS. Megaesophagus in the horse. A short review of the literature and 19 own cases. *Vet Quart.* 24;4:199-202.