Esophageal Obstruction in Horses

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ABSTRACT:
Esophageal obstruction is a commonly encountered problem in equine practice. This article discusses the multifactorial causes, clinical presentation, differential diagnosis, confirmed diagnosis, and potential complications of esophageal obstruction in horses as well as available treatment options.

Obstruction is the most commonly encountered problem affecting the equine esophagus. Obstruction may be primary or secondary and is regarded as an emergency because affected horses may become compromised by dehydration, acid–base and electrolyte imbalances, and aspiration pneumonia.

ANATOMY AND PHYSIOLOGY OF THE ESOPHAGUS
The esophagus is a musculomembranous tube that transports ingesta, water, and saliva from the pharynx to the stomach.1 The mucosa of the esophagus is composed of stratified squamous epithelium. Elastic fibers exist in the submucosa, which contributes to the longitudinal folding of the mucosa. The muscle tissue of the esophagus comprises two layers.1 The muscularis mucosa of the lamina propria is thin in the cranial esophagus but becomes more substantial as it approaches the cardia. It is composed of smooth muscle fibers with longitudinal orientation. The second layer, the muscularis externa, is present along the entire length of the esophagus and is composed of striated muscle to the midthoracic region (i.e., the upper two-thirds). The caudal one-third of this muscle layer is composed of smooth muscle fibers. The fibers of this muscle tissue have a longitudinal orientation at the outer (adventitial) surface but become transversely oriented as they extend toward the luminal surface. Only the abdominal esophagus has a serosal covering.1

Food boluses are passed through the upper esophageal sphincter into the cranial esophagus during the involuntary act of swallowing. Peristaltic waves propel the food along the esophagus. The lower esophageal sphincter reflexively relaxes to allow passage of ingesta into the stomach.2 Motor innervation of the striated muscle of the esophagus is via the pharyngeal and esophageal branches of the vagus nerve, which also provides parasympathetic innervation to the smooth muscle of the distal esophagus. Sympathetic innervation of the esophagus is minimal.3

CAUSE
Esophageal obstruction in horses may be primary or secondary. Although foreign bodies may be involved, primary obstruction is most commonly caused by intraluminal impaction with feedstuffs (e.g., grain, pelleted feed, hay, straw, fruit, vegetables). Dental problems, such as those associated with young horses with erupting teeth and/or deciduous caps and those of older horses with missing teeth, large hooks, and/or wave mouth, can contribute to development of esophageal obstruction by impairing adequate mastication of feed before swallowing. Dehydration and exhaustion may predispose a
Primary esophageal obstruction in horses is most commonly caused by intraluminal impaction with ingesta.

Clinical Signs of Esophageal Obstruction in Horses

- Distress
- Dysphagia (outstretched neck, food material at nares)
- Coughing and retching
- Ptyalism
- Dehydration
- Palpable enlargement in the cranial cervical region

Table 1. Differential Diagnosis for Esophageal Obstruction in Horses

<table>
<thead>
<tr>
<th>Condition</th>
<th>Differentials</th>
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<tbody>
<tr>
<td>Dysphagia</td>
<td>Botulism, Congenital defects, Hyperkalemic periodic paresis, Rabies, End-stage liver disease, Lead toxicosis</td>
</tr>
<tr>
<td>Fetal nasal discharge</td>
<td>Pneumonia, Pharyngeal abscessation, Gullet pouch empyema, Upper respiratory tract neoplasia</td>
</tr>
<tr>
<td>Ptyalism</td>
<td>Gastrointestinal ulceration in foals, Botulism, Sepsis, Pharyngeal trauma, Slaframine</td>
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Horses with esophageal obstruction typically present with an outstretched neck and food material at the nares (Figure 1). They may retch or cough intermittently. Coughing is precipitated by agitation of accumulated saliva and ingesta in the esophagus or by feedstuffs within the trachea. Ptyalism may also be observed. If the obstruction is in the cervical esophagus, an enlargement may be externally palpable in that region of the neck. Affected horses can quickly become dehydrated with electrolyte derangements and metabolic acidosis because neither bicarbonate-rich saliva nor water can be swallowed.

Differential Diagnosis

The differential diagnosis of equine esophageal obstruction includes any disease or condition that causes dysphagia and feed material to appear at the nares and mouth (Table 1). Hyperkalemic periodic paresis is a genetic disease primarily of quarter horses, but it is also recognized in paints and appaloosas. It can cause laryngopharyngeal dysfunction (i.e., laryngospasm) and may lead to evidence of dysphagia. Rabies is another important diagnostic differential for dysphagia in horses, particularly if other neurologic deficits are present. Laryngeal dysfunction has also been observed in horses with end-stage liver disease leading to hepatic failure.
encephalopathy and with lead toxicosis.\textsuperscript{13–15} Feed material at the nares, especially when mixed with salivary secretions, may resemble fetid discharge seen in horses with severe pneumonia, pharyngeal abscessation, guttural pouch empyema, or upper respiratory tract neoplasia (Figure 1).

Ptyalism may be seen secondary to ingesting slaframine or in animals with pharyngeal trauma (e.g., due to traumatic passage of a nasogastric tube). It may also be seen in foals with gastroduodenal ulceration, botulism, or sepsis.

**DIAGNOSIS**

Clinical signs, palpation of the neck, and passage of a nasogastric tube usually permit a tentative diagnosis to be made. However, endoscopy of the esophagus can be
used to confirm the diagnosis and visualize the obstruction (Figures 2 and 3). Radiography of the neck is impractical for field diagnosis but may be used in the referral setting, with or without contrast medium, to delineate a causative lesion. The normal esophagus blends in with tissues of the neck and thoracic cavity on plain radiographs and is usually imperceptible. Gas may be seen if the esophagus is dilated proximal to the obstruction. A barium contrast study can be used to evaluate the position of the obstruction and/or the presence and extent of a diverticulum or stricture (Figure 4).

In the case of perforation, gas is seen in the soft tissues adjacent to the esophagus. If rupture or perforation of the esophagus is suspected, iodinated compounds should be used instead of barium for contrast studies because barium irritates extraesophageal tissue. Recurrent esophageal obstruction may indicate a persistent functional or anatomic disorder. In addition to endoscopy, this may require evaluation by electromyography, manometry, and muscle or nerve biopsies.

**Table 2. Drug Therapy for Esophageal Obstruction in Horses**

<table>
<thead>
<tr>
<th>Drug</th>
<th>Dose (ml/500 kg IV)</th>
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<tr>
<td>Acepromazine</td>
<td>2–4</td>
</tr>
<tr>
<td>Xylazine hydrochloride</td>
<td>2–3</td>
</tr>
<tr>
<td>Detomidine</td>
<td>0.5–1</td>
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**INITIAL MANAGEMENT**

Esophageal obstruction should be treated as a medical emergency because extensive pressure necrosis of the mucosa can occur, resulting in perforation of the esophagus or long-term damage and complications with scarring and stricture formation. Treatment should be directed primarily at relieving the obstruction and correcting fluid deficits or electrolyte and/or acid–base imbalances.

In mild cases, tranquilizing a horse with acepromazine maleate (0.04 to 0.1 mg/kg IV) may be sufficient. This may permit the esophageal musculature to relax enough to allow an obstruction to pass. Tranquilization is usually evident within 15 to 20 minutes. At this time, gentle pressure with a nasogastric tube may help pass an obstruction to the stomach, although this should be attempted with great caution because perforation of the esophagus is possible, especially if the obstruction is longstanding and the esophageal tissue compromised. Sedation with xylazine hydrochloride, an \( \alpha_2 \)-adrenergic agonist, allows the head to be lowered to prevent aspiration of esophageal reflux into the airways (Table 2). Using xylazine can also calm a distressed animal and relax muscles to help pass the obstruction. Lavage of the esophagus via a nasogastric tube is the most common method of managing obstructions. If this procedure is attempted, the horse should be well sedated. The intravenous combination of butorphanol tartrate and an \( \alpha_2 \)-adrenoceptor agonist provides profound sedation. We recommend using 0.01 mg/kg of detomidine hydrochloride with 0.02 mg/kg of butorphanol tartrate. Once the horse is adequately sedated, a 26-mm internal diameter endotracheal tube (for a 990-lb [450-kg] horse)

**Dysphagia is the most common presenting sign of equine esophageal obstruction. Horses typically present with an outstretched neck and food material at the nares.**
with an inflated cuff can be placed in the trachea to prevent aspiration of the reflux and lavage fluid. This can be done in a standing horse, but significant coughing and patient resistance may be encountered. The antitussive effects of butorphanol may be useful here. A nasogastric tube can then be passed through the other nostril and into the esophagus, and large-volume lavage can be performed with warm water or saline while gently advancing the tube as the obstruction softens and loosens.

If manipulation of a nasogastric tube is unsuccessful, it is often necessary to directly visualize the obstruction with an endoscope (Figure 3). Furthermore, biopsy forceps can be passed through the biopsy channel of the endoscope to manipulate and fragment the esophageal obstruction (Figure 5). This can be time-consuming but is less likely to result in long-term damage to the esophageal mucosa than is blunt pressure with a nasogastric tube. In cases of refractory esophageal obstruction, spasms may be present after manipulation. In such cases, repeated sedation may assist in relaxing the esophagus. Detomidine hydrochloride can be administered intramuscularly for prolonged sedation at 0.02 to 0.04 mg/kg. All fluid deficiencies and electrolyte and acid–base imbalances should be addressed while intermittently attempting to remove the obstruction.

A recent clinical report supports using oxytocin to relax the esophageal muscles in cases of equine esophageal obstruction. Obstruction most commonly occurs just aboral to the larynx and at the thoracic inlet, where the musculature is predominantly striated. It is unclear through what mechanism oxytocin may affect the striated muscle of the esophagus. This study demonstrated short-term reduction of striated muscle tone in the equine esophagus when using oxytocin. This resulted in reduced intraluminal pressure. The greatest magnitude of effect occurred at intravenous doses of 0.11 and 0.22 IU/kg 3 to 15 minutes after administration. The half-life of oxytocin is undocumented in horses, but it is reportedly 13 minutes in dogs.

Another recent study investigated the effects of xylazine, detomidine, butorphanol, acepromazine, oxytocin, and guaifenesin on esophageal manometric pressures in horses. The results indicated that detomidine, acepromazine, and a combination of xylazine and butorphanol had the greatest effect on esophageal motility. Decreased spontaneous swallowing and changes in normal, coordinated peristaltic activity were the most clinically relevant effects. We have used oxytocin in cases of esophageal obstruction in which sedation with an α2-agonist and nasogastric lavage were not sufficient to remove an obstruction, but we have not found oxytocin to be useful in this regard.

In cases in which resolution of an obstruction cannot be achieved by the means already discussed, general anesthesia may be warranted. General anesthesia permits complete relaxation of the esophageal musculature and thus can aid in removing an obstruction. The horse should be placed in lateral recumbency with its head below body level. A cuffed endotracheal tube should be placed in the trachea to prevent aspiration of reflux while the esophagus is lavaged continuously.

Surgical resolution of esophageal obstruction is infrequently necessary but has been described. The surgical techniques used include esophagotomy and surgical repair of the incision or esophagotomy and second-intention wound healing of the site. The esophagus, lacking a serosal layer, does not heal well, and complications of surgery include development of strictures at the site and recurrence of the obstruction. In a study of 61 horses with esophageal obstruction, surgical repair was attempted in two cases of foreign body obstruction by longitudinal nonsutured or sutured cervical esophagotomy. The nonsutured esophagotomy healed by second intention, and the horse returned to a normal diet. The sutured esophagotomy was followed by severe postoperative infection and laminitis, and the horse required euthanasia. Esophageal diverticula have been corrected by diverticulectomy or inversion of the redundant...
mucosa, followed by repair of the defect in the tunica muscularis.\textsuperscript{5,18}

**COMPLICATIONS**

Complications of esophageal obstruction include esophageal mucosal ulceration (Figure 6) and stricture formation, esophageal perforation, aspiration pneumonia, chronic recurrent obstruction, postoperative infection, pleuritis, laminitis, laryngeal paralysis, and Horner’s syndrome.\textsuperscript{5}

Early mucosal erosions can be managed conservatively by controlling inflammation and secondary infection. If there is circumferential loss of mucosa as a result of esophageal obstruction, strictures may develop. Stricture formation appears to be most severe up to 30 days following the initial insult.\textsuperscript{19} Bougienage has been attempted without success in horses.\textsuperscript{18}

Resection of strictures and successful anastomosis of the esophagus has been described, but surgical complications are common.\textsuperscript{20–26} In one study, surgical repair of strictures was attempted in nine horses.\textsuperscript{5} Surgical repair of mural strictures originating in the tunica muscularis was performed by modified esophagomyotomy using longitudinal incision and transverse closure of the tunica muscularis. Annular strictures involving the mucosa and submucosa were repaired by partial resection and anastomosis, patch grafting with the sternoclephalicus muscle, or mucosal scar fenestration. In two horses, the surgical sites were approached but not repaired because of extensive esophageal damage in one horse and inaccessibility in another.\textsuperscript{5} Two horses had successful outcomes after modified esophagomyotomy.\textsuperscript{5} Periesophageal infection, dehiscence, and subsequent failure of partial resection and anastomosis of strictures developed in three horses.\textsuperscript{5}

In two horses, patch grafting failed because of extensive periesophageal leakage, postoperative infection, and esophageal fistulation.\textsuperscript{3} In total, postoperative resolution of the stricture and return to normal diet occurred in four of the nine horses.\textsuperscript{5}

Sharp, penetrating foreign bodies and overly vigorous efforts to relieve choking by using a stomach tube may result in a penetrating wound in the esophagus, particularly if necrosis of the wall has occurred in the region of the obstruction. Surgical management of cervical esophageal perforation includes exploring the neck with wide excision of devitalized tissues, without primary closure and repair of the esophagus.\textsuperscript{5}

Horses may develop aspiration pneumonia if successful management of esophageal obstruction is not timely. In a study of 34 horses with esophageal obstruction, the duration of the obstruction was a good indicator of whether aspiration pneumonia would develop.\textsuperscript{9} Endoscopic evidence of tracheal contamination with food was a poor predictor of subsequent pneumonia. However, all horses that developed aspiration pneumonia, diagnosed on clinical and radiographic evaluation, responded well to broad-spectrum antimicrobial therapy with penicillin G, gentamicin, and metronidazole without residual impairment of lung function. The onset of aspiration pneumonia varies with the severity and duration of the obstruction and the amount of material aspirated. Signs may be noticed within 24 hours or may not become apparent for a few days. If aspiration pneumonia is suspected, broad-spectrum antimicrobials should be used. Transtracheal aspirate can be examined to identify pathogens and their sensitivities to antimicrobials as well as to determine the extent of inflammation.

**LONG-TERM MANAGEMENT**

NSAIDs (e.g., flunixin meglumine [1.1 mg/kg IV or PO], phenylbutazone [4.4 mg/kg IV or PO]) can be used to treat pain and control inflammation at the obstruction site. However, prolonged therapy at these doses is associated with ulceration of the gastrointestinal tract, particularly in the right dorsal colon. Decreasing the dose and/or dose interval is recommended after 3 to 5 days. To promote epithelial regeneration, control pain, and reduce fibroplasia and subsequent stricture formation, feeding a low-bulk minimally abrasive diet in conjunction with administering antiinflammatories is appropriate. Any complete pelleted feed that can be soaked enough to make a soft mash is appropriate. Corticosteroids should not be
used within 48 hours of injury because they inhibit and retard synthesis of collagen.\textsuperscript{20} Corticosteroids are also contraindicated in the presence of aspiration pneumonia. However, after 48 hours without aspiration pneumonia, corticosteroids allow epithelialization to proceed at a faster rate than fibrous tissue synthesis.\textsuperscript{20} In a study of seven horses in which esophageal stricture was managed medically, a soft diet was fed and NSAIDs were given.\textsuperscript{20} Five of the horses were clinically normal by 60 days after obstruction. The esophagus should be periodically reexamined with an endoscope to assess the esophageal mucosa and development of strictures.

REFERENCES

1. The most common cause of primary esophageal obstruction is
   a. esophageal stricture.
   b. vascular ring anomaly.
   c. food bolus.
   d. neuromuscular disease.

2. Which of the following is not a clinical sign of primary esophageal obstruction?
   a. feed material at the nares
   b. coughing
   c. head extension
   d. head pressing

3. The differential diagnosis for esophageal obstruction does not include
   a. gutteral pouch empyema.
   b. rabies.
   c. lead toxicosis.
   d. gastric ulcer syndrome.

4. Which of the following is not useful in diagnosing equine esophageal obstruction?
   a. endoscopy
   b. ultrasonography
   c. passage of a nasogastric tube
   d. radiography

5. Plain radiography may identify
   a. the position of an obstruction.
   b. neuromuscular dysfunction.
   c. stricture of the esophagus.
   d. presence of a diverticulum.

6. Initial medical management of esophageal obstruction should include
   a. manometry.
   b. sedation or tranquilization and lavage via a nasogastric tube.
   c. general anesthesia.
   d. contrast radiography.

7. Which drug or drug combination is most useful in sedating horses for esophageal lavage?
   a. acepromazine
   b. xylazine
   c. an α₂-adrenoceptor agonist and butorphanol
   d. detomidine

8. Which of the following is not a potential complication of esophageal obstruction?
   a. esophageal stricture formation
   b. aspiration pneumonia
   c. Horner’s syndrome
   d. anterior enteritis

9. Which statement regarding stricture formation is incorrect?
   a. Stricture formation occurs when there is circumferential loss of mucosa.
   b. Stricture formation appears to be most severe up to 30 days following the initial insult.
   c. Bougienage is a successful means of managing esophageal strictures in horses.
   d. Corticosteroids may be used 48 hours after an injury to allow epithelialization to proceed faster than fibrous tissue synthesis.

10. Which statement regarding development of aspiration pneumonia due to prolonged obstruction is correct?
    a. Endoscopic evidence of tracheal contamination is a good predictor of subsequent aspiration pneumonia.
    b. Every horse with esophageal obstruction develops aspiration pneumonia.
    c. Penicillin is the drug of choice in treating aspiration pneumonia because the condition usually involves only gram-positive bacteria.
    d. The duration of obstruction is a good indicator of whether aspiration pneumonia will develop.