Choke: causes, signs, diagnosis, complications and prevention strategies

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ABSTRACT

Oesophageal obstruction, or choke, is a common clinical presentation in the horse with many causes, which can be categorised into intraluminal, intramural, extramural and functional disorders (**Table 1**). The clinical signs are primarily related to the regurgitation of food, water and saliva caused by oesophageal dysphagia and include an anxious expression, extended neck, frequent gagging or retching, bilateral nasal discharge containing saliva, food and water, ptyalism, coughing and possibly a distension in the jugular furrow, depending on the location of the obstruction.

A diagnosis is based on the clinical signs and on the results of further diagnostic tests. Endoscopy is often the most informative of these as it provides details relating to the presence, location and nature of the obstruction as well as allowing the condition of the oesophagus to be assessed. Potential complications include dehydration, electrolyte and/or acid-base disturbances, aspiration pneumonia, weight loss, oesophageal rupture, and oesophageal stricture or diverticulum formation.

Prevention strategies are based on management changes and include withholding food following sedation, regular dental examination, adequate soaking of certain feedstuffs, adequate access to water, tactics to reduce bolting of food and cutting up, or avoidance, of certain feedstuffs.

Oesophageal obstruction or choke is a common clinical presentation in the horse that has many causes.

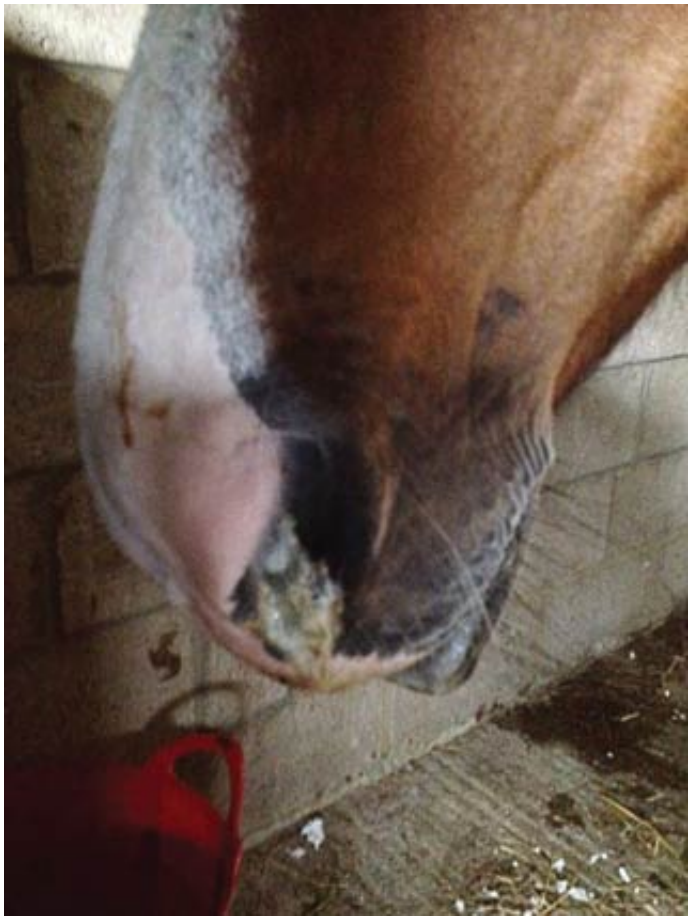

Figure 1. The clinical signs suggestive of oesophageal obstruction include a nasal discharge containing saliva, food and water.

It is most commonly manifested clinically by impaction of food material and resulting oesophageal dysphagia. A simple impaction of roughage, particularly hay, bedding or even grass, can occur. Prior oesophageal trauma or poor mastication caused by dental abnormalities may predispose horses to oesophageal impactions. Wolfing or gulping food may also precipitate an impaction, particularly if the horse is exhausted or mildly dehydrated after a long ride or is weakened from chronic debilitation.

In addition, impactions may result from intramural or extramural disorders that physically impede the passage of food by narrowing the oesophageal lumen, reduce the oesophageal wall compliance or alter the oesophageal wall conformation to the point that food accumulates in a diverticulum. Finally, impactions may result from functional disorders where altered oesophageal motility is the underlying problem. Functional disorders vary in aetiology from pharmacological to neurological disease.

Clinical signs

The clinical signs associated with oesophageal obstruction are primarily related to the regurgitation of food, water and saliva caused by oesophageal dysphagia.

The signs include:

- anxious expression or behaviour
- extended neck
- frequent gagging or retching
- bilateral nasal discharge containing saliva, food and water (**Figure 1**)
- ptyalism (**Figure 2**)
- coughing
- distension in the jugular furrow, depending on the location of the obstruction.

Diagnosis

Figure 2. Ptyalism may result in pools of saliva on the floor.

A diagnosis of an oesophageal obstruction is based on the clinical signs and the results of further

diagnostic tests. A number of appropriate further diagnostic tests are available.

- **Passage of a nasogastric tube**. This will confirm the presence and location of an oesophageal obstruction, but provides little information about the nature of the obstruction or the condition of the oesophagus.
- **Endoscopy**. This is the most direct method for diagnosis of an oesophageal obstruction as it allows the presence and location of an obstruction to be confirmed, the nature of the obstruction to be visualised (**Figure 3**), the condition of the oesophagus to be assessed – including determination of the presence of mucosal ulceration, rupture, stricture, diverticula and so on – and the possible retrieval of foreign bodies.
- **Ultrasonography**. Useful in confirming the presence, location and extent of a cervical oesophageal obstruction and to provide information on oesophageal wall thickness and integrity.
- **Radiography**. Contrast radiography can confirm the presence of an obstruction in cases where endoscopy is not possible or in cases where an oesophageal stricture, dilation, diverticulum, rupture, functional disorder or luminal narrowing caused by extraluminal compression is suspected following relief of the obstruction. Care should be taken interpreting radiographs in sedated horses or after passage of a nasogastric tube or other oesophageal manipulation that may contribute to temporary oesophageal dilation.

Complications

Most cases of oesophageal obstruction resolve quickly or are easily treated with no long-term complications. However, the loss of food, water and saliva can result in a number of complications.

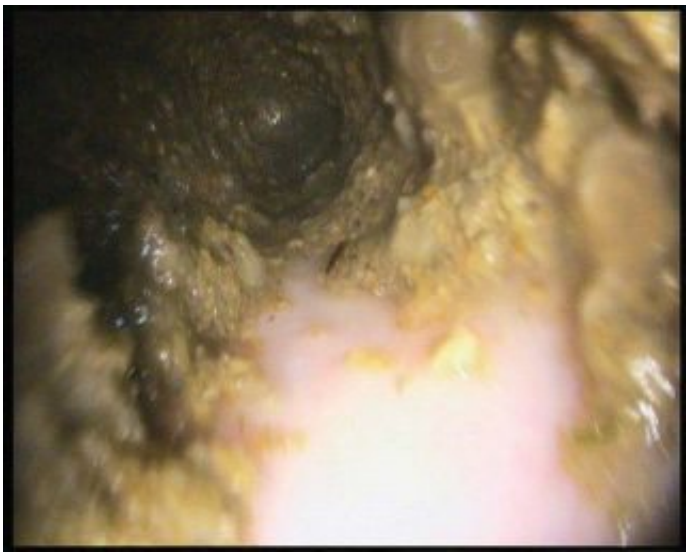

Figure 3. Oesophageal endoscopy findings in a horse with an oesophageal obstruction

caused by a simple impaction of food material.

Dehydration. This will manifest clinically as tachycardia, prolonged jugular refill time, congested mucous membranes, prolonged capillary refill time, reduced skin turgor, cold extremities and reduced or absent urine output. The clinical pathology parameters that should ideally be measured include the blood PCV, total protein, creatinine, urea and venous oxygen concentrations, and urine-specific gravity. The deficit should be estimated; a horse will start to show mild signs at 5% dehydration and severe signs at 12% dehydration. The deficit should then be replaced via the intravenous route if the obstruction has not been relieved or if there are any oesophageal abnormalities following obstruction relief, otherwise the enteral route can be considered.

- **Electrolyte and acid-base imbalances**. Salivary loss results in hypochloraemia, hyponatraemia and hypokalaemia. Hyponatraemia and dehydration resulting in lactic acidosis cause metabolic acidosis, while hypochloraemia and hypokalaemia result in metabolic alkalosis. These imbalances should be treated with fluids supplemented with appropriate electrolytes.
- **Aspiration pneumonia**. This is one of the most frequent complications after oesophageal obstruction and it occurs if food, water and/or saliva are inhaled into the lower respiratory system. This results in bacterial pneumonia caused by predominantly oropharyngeal flora, usually in the cranioventral lung lobes, which manifests clinically as depression, lethargy, anorexia, tachypnoea, dyspnoea and pyrexia. It should be treated with broad-spectrum antibiotics while culture and sensitivity results are awaited from a lower respiratory tract sample, such as a tracheal wash. If not appropriately treated, it may extend to become bacterial pleuropneumonia.
- **Weight loss**. This will occur in cases of chronic oesophageal obstruction due to a chronic inability to consume food.

If the obstruction persists, it can result in:

- Oesophageal rupture. Pressure necrosis from the oesophageal impaction may result in oesophageal perforation or iatrogenic perforation may occur in response to excessive force with a nasogastric tube. Extensive cellulitis and necrosis of tissues surrounding the perforation occurs because of the drainage of food and saliva within fascial planes. Systemic inflammation associated with endotoxaemia may occur. Additionally, air may migrate along fascial planes into the mediastinum and pleural space causing a pneumomediastinum or pneumothorax. Alternatively, air leakage into the tissues may result in extensive subcutaneous emphysema. Treatment includes extensive debridement and lavage of affected tissues, broad-spectrum antibiotic therapy, tetanus prophylaxis, antiendotoxic therapy, fluid therapy and oesophageal rest achieved using a feeding tube placed into the oesophagus via the wound or a small diameter nasogastric tube. Healing is prolonged and potential complications include oesophagocutaneous fistula, oesophageal diverticula, oesophageal stricture, Horner’s syndrome and laryngeal hemiplegia. The

prognosis is extremely guarded.

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TABLE 1. Categories of oesophageal obstruction and differentials

Category	Differential
Intraluminal	• Foreign body • Food material (simple impaction)
Intramural	• Oesophageal abscess • Granuloma • Neoplasia – for example, squamous cell carcinoma • Cysts – for example, intramural cysts, duplication cysts • Diverticulum • Stenosis
Extramural	• Neoplasia – for example, lymphoma • Vascular ring anomaly – for example, persistent right aortic arch • Granuloma
Functional disorders	• Dehydration • Exhaustion • Pharmacological – for example, acepromazine, detomidine • Primary megaoesophagus • Oesophagitis • Equine dysautonomia (equine grass sickness) • Vagal neuropathies

Table 1. Categories of oesophageal obstruction and differentials.

Oesophageal diverticulum. Two types of diverticula exist: traction diverticula, as a result of wounding and subsequent contraction of perioesophageal tissues with resultant tenting of the wall of the oesophagus; and pulsion diverticula, arising from protrusion of the oesophageal mucosa through defects in the muscular wall of the oesophagus and usually resulting from acute changes in intraluminal pressure. Diverticula may be amenable to conservative management through dietary alterations to prevent impaction of feed material in the diverticulum, for example, slurry feeding, or may require surgical correction.

- **Oesophageal stricture**. This occurs due to pressure necrosis from oesophageal impaction that induces circumferential erosion or ulceration of the oesophageal mucosa. The stricture will then result in partial obstruction of the oesophageal lumen, resulting in recurrence of oesophageal obstruction. The maximal reduction in oesophageal lumen tends to occur within 30 days of the initial obstruction and the oesophagus continues to remodel for up to 60 days following ulceration. Although surgery has been used to relieve these strictures, initial medical management, including feeding of a slurry diet and administering anti-inflammatory and antibiotic medications, is warranted because strictures may resolve with conservative therapy. If this is unsuccessful, dilation using bougienage or surgery should be considered.

Prevention strategies

The most important management considerations to try to prevent oesophageal obstruction from occurring are:

- Withholding feed following sedation.
- Soaking dried foodstuffs thoroughly to allow them to swell before they are eaten and swallowed.
- Regular routine dental care to allow the horse to chew food thoroughly and effectively before it is swallowed.
- Providing continuous access to clean water to encourage the horse to drink normally.
- Some horses choke on a particular feed and, once this is recognised, access should be avoided.
- Strategies to slow food consumption in animals that bolt their food include feeding the horse away from others so it does not rush while eating for fear another horse will snatch the food, feeding a smaller amount at a higher frequency and putting an object that is too big to eat, such as a large stone or salt block, in the feed bowl, so the horse has to search for its feed slowly.
- Change feeds gradually.
- Cut apples, carrots and so on into small pieces.