Case Report

Management of oesophageal rupture by tube feeding through the oesophageal wound

E. K. Leus* and P. Randleff-Rasmussen

Vetscape Referrals, Paarl, South Africa

*Corresponding author email: emileleus@hotmail.com

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Summary

This case report describes the management of a perforation of the oesophagus after an obstruction of long duration by creating an oesophagostomy with an indwelling tube through the oesophageal wound. The oesophageal perforation was diagnosed by clinical examination, oesophagoscopy, ultrasonography and contrast radiography. The perforation was managed by insertion of a feeding tube through the original oesophageal wound and careful nutritional and antimicrobial management with a good short-term outcome after closure of the fistula.

Introduction

Oesophageal perforation is an uncommonly encountered injury in horses that can be spontaneous or traumatic in origin (Stick 2006; Fubini et al. 2019). It can be caused by external neck trauma, penetration by a foreign body, nasogastric intubation or due to mucosal necrosis following chronic oesophageal obstruction (Cathcart et al. 2012). These perforations are usually associated with a poor prognosis due to various complications such as necrotising cellulitis of the ventral aspect of the neck, aspiration pneumonia (Lunn and Peel 1985; Lane 2002), laryngeal hemiplegia, rupture of the common carotid artery, septic mediastinitis, endotoxaemia, peritonitis and development of non-healing oesophageal fistulas (Whitfield-Cargile et al. 2013). Diagnostic tools for the evaluation of oesophageal perforations include passage of a nasogastric tube, oesophagoscopy and contrast radiography or fluoroscopy (Feige et al. 2000). Both a medical treatment approach and a surgical treatment approach are possible and dependant on location, extent and duration of the rupture and also on the degree of infection and any associated complications (Whitfield-Cargile et al. 2013). Few cases of oesophageal fistulas and their treatment have been described, and the purpose of this case report is to describe the management and treatment of a complicated oesophageal perforation caused by chronic obstruction through creating an artificial fistula with use of an indwelling oesophageal tube.

Case history and clinical findings

A 10-year-old pony (400 kg, body condition score 4/10) arrived at our referral clinic with clinical signs of oesophageal obstruction of at least 72 h duration (Duncanson 2006; Jennings 1984). He presented with purulent nasal discharge, regurgitation, coughing, respiratory stridor and anorexia. The clinical signs had been present for 72 h, and the referring veterinarian had been unable to resolve the obstruction with administration of intravenous Buscopan compositum1 or passage of a nasogastric tube. On admission, the pony was presented in a lethargic state with congested mucous membranes, tachycardia (68 beats/min), decreased barbarygni and pyrexia. On auscultation, increased inspiratory sounds and crackles were heard on both the right and left cranioventral hemithoraces. A slight swelling was visible in the distal third of the jugular groove on the left side of the neck.

Further diagnostic tests

A full blood count revealed a leucopenia (2.7 x 109/L) with neutropenia (1.46 x 109/L). The serum amyloid A (SAA) was elevated (1088 mg/L). Ultrasonography of the thorax was performed and revealed signs of aspiration pneumonia with multiple areas of lung consolidation and abscessation cranioventrally in the right and left hemithorax with linear pleural fluid present. Endoscopic examination of the oesophagus showed an obstruction with fibrous feed material at one metre from the nares, which corresponds to the lower third of the neck.

Treatment

Initially, the obstruction was resolved with the aid of a nasogastric tube and gentle lavage with tap water after the intravenous administration of detomidine2 (20 μg/kg bwt) and butorphanol3 (0.1 mg/kg bwt). After resolution of the obstruction, oesophagoscopy revealed large ulcerative lesions and erosions in the oesophagus from 85 to 125 cm from the nares (Fig 1). A 14 gauge intravenous catheter was placed in the right jugular vein, and the pony received maintenance intravenous fluid therapy with Lactated Ringers4 solution for 72 h. The pony was treated with 5 days of procaine penicillin5 (22 mg/kg bwt b.i.d. i.m.) and gentamycin6 (6.6 mg/kg bwt s.i.d. i.v.), 8 days of fluimixin meglumine7 (1.1 mg/kg bwt b.i.d. i.v./per os) and sucralfate8 (40 mg/kg bwt q.i.d. per os). He was denied food for 48 h and then started on a liquid diet four times a day for 4 days (bran mash and soaked short fibre alfalfa). After 5 days of the initial antibiotic therapy, the horse was changed onto oral doxycycline9 for the remaining pneumonia (10 mg/kg bwt b.i.d. per os) and was fed small amounts of wet lucerne chaff and concentrates. Three days later, the swelling on the left side of the neck, which was initially a suspected perivenous Buscopan injection reaction, enlarged, and radiographs of the region revealed a large gas-filled mass. Metronidazole10 (20 mg/kg bwt q.i.d. per os) was...
added to his treatments due to the suspicion of a subcutaneous abscess and the risk of spreading to the mediastinum. The abscess was opened (purulent material) and flushed with sterile saline. A Foley catheter was placed to maintain drainage, and the opening was covered with a bandage. Two days later, ingesta were discovered in the draining material. Further investigation revealed a large pocket of accumulated ingesta dorsal to the trachea. A fistula between the oesophagus and the subcutaneous tissue was confirmed by contrast radiography (Fig 2).

A large bore nasogastric tube (20 mm outer diameter, 12 mm inner diameter) was placed through the original oesophageal defect under endoscopic guidance as for an oesophagostomy (Fig 3). The surrounding tissues were debrided and flushed thoroughly under standing sedation with aid of local anaesthesia (Lidocain®). The tube was sutured to the left side of the neck and maintained with a clean bandage (Fig 4). A liquid diet (6 L of water with 1 kg of Coolstance®, bran mash, a handful of lucerne, 15 g of sodium chloride, 10 g of potassium chloride, 15 g of sodium bicarbonate, a scoop of Myoplast®, 50 mL of corn oil and casein protein four times a day) was initiated. This diet contains more than 13,320 kcal (33.3 x bodyweight of 400 kg) and meets the fluid requirements of 24 L a day (60 mL/kg bwt/day). The pony was also put on trimethoprim–sulfonamide® (20 mg/kg bwt b.i.d. per os) for 10 days and was received 15 mL of Kyrovital 15® (vitamin supplement) every other day while on the tube feeding regimen. The tube was left in place for 7 days with ongoing liquid ration feeding, and the weight of the pony stabilised at 370 kg over the course of the enteral feeding period. Once the infection was controlled and healthy granulation tissue present along the entire wound, the oesophagostomy tube was removed and the tube was replaced via the nostril for a further 4 days (Fig 5).

Following the removal of the nasogastric tube, the pony was placed on a liquid diet combined with small amounts of wet teff and lucerne chaff. A patent fistula remained, with feed, saliva and water draining from the ventral aspect of the neck during peristaltic movements of the oesophagus (Fig 6). Daily cleaning and application of paraffin cream were required to avoid damage to the surrounding skin. Two weeks before closure of the fistula, oesophagoscopy was performed and a butterfly probe was inserted through the wound. Although the external opening appeared to be
The fistula gradually reduced in size and closed after a total of 48 days of treatment at which time the pony was turned out in a grass field during the day and received normal ad libitum teff, hay and concentrates in the stable. The bodyweight at this stage had increased to 420 kg, which would be considered a healthy bodyweight for the size of the pony.

Discussion

Oesophageal perforation is an uncommon condition, usually associated with poor prognosis, especially if the rupture occurs after necrosis of the wall due to long-term oesophageal obstruction (Bezdekova 2012; Whitfield-Cargile et al. 2013). Prompt surgical intervention is required to prevent complications such as pneumonia, damage to the vagosympathetic trunk or recurrent laryngeal nerve, rupture of the carotid artery, endotoxaemia or non-healing fistula formation (Whitfield-Cargile et al. 2013). Diagnosis was delayed and challenging in this case because the oesophageal rupture remained closed until the enlarging swelling in the left distal jugular groove was drained (Risnes and Mair 2003). Unlike ruptures following recent trauma, primary closure of the oesophageal defect was not indicated in this case because of the infected surrounding tissues and the fragile wound edges of the oesophageal tear. Surgical management of the infected area is usually indicated in order to excise all necrotic and infected tissues and to produce ventral drainage (Craig et al. 1989; Whitfield-Cargile et al. 2013). Debridement under general anaesthesia can be indicated in severe cases (Freeman 1989). In order to obtain healing of the tear, oesophageal rest is required. Many case reports suggest placing an oesophagostomy tube through the initial defect (Craig et al. 1989; Freeman and Naylor 1978; Risnes and Mair 2003; Bezdekova 2012), although there is also the possibility of placing the tube through a surgical incision distally from the original one (Read et al. 2002; Bezdekova 2012; Whitfield-Cargile et al. 2013). Freeman and Naylor (1978) originally described a technique for oesophagostomy tube placement. A nasogastric tube (18 mm OD) is placed and local anaesthetic infiltrated at the junction between the middle and lower third of the ventral midline of the neck, at the level of the 5th cervical vertebra, where a 6-cm incision is made. Sharp dissection is continued through the cutaneous colli and sternocleidomastoid muscles.
until the sternothyroides muscle and trachea are exposed. Blunt division of the deep fascia then frees the left sternocleidomastoideus muscle from the tract and sternothyroides muscle allowing access to the oesophagus. With gentle traction, the oesophagus is exteriorised through the skin incision and a 1-cm longitudinal incision is made in the muscle wall of the ventral aspect of the oesophagus. The incision is spread, and the submucosa is identified, grasped and everted with the mucosa. A transverse incision is made through half the width of the everted mucosa creating an opening into the lumen for a 9.5-mm oesophagostomy tube. The edges of the mucosal layer can then be everted to expose the lumen, while the nasogastric tube is removed allowing insertion of the oesophagostomy tube into the stomach. The tube is then sutured to the right side of the neck with ‘butterfly’ bandages. The caudal half of the muscle and fascial edges is closed with simple interrupted sutures as well as the skin incision (Freyman and Naylor 1978).

If the tube is placed through the original defect for 7–10 days, tissues will be able to granulate and contract around it before removal and a fistula will be able to form (Craig 1989: Freeman 1989; Risnes and Mair 2003; Whitfield-Cargile et al. 2013). In this case, after removing the tube from the initial defect, a nasogastric tube was placed through the left nostril for a further 4 days to continue the oesophageal rest. Both techniques seem to result in a granulated fistula which has been reported to close between 50 and 80 days after the initial rupture (Craig et al. 1989: Bezdekova 2012). In this case, the loss of saliva, feed and water through the healing fistula was not sufficient to cause the pony trouble in meeting the caloric and electrolyte requirements. However, in one other case it was necessary to create a ‘patch’ out of dental plastic that was sutured over the healing defect to prevent ongoing nutritional losses (Read et al. 2002). The loss of saliva was described to be compensated by collection from the fistula in a bucket and refeeding by stomach tube in a feed mixture through the oesophageal defect in an early case report. In this report, the tube was removed and replaced for every feed until the defect was too closed for the tube to pass (Raker and Sayers 1958).

In cases of oesophageal rupture, it is required to cover the horse with broad-spectrum antibiotics. For the initial treatment, procaine penicillin (25 mg/kg bwt b.i.d. i.m.) or benzylpenicillin [20,000–25,000 IU/kg bwt q.i.d. i.v.], gentamicin [6.6 mg/kg bwt s.i.d. i.v.] and metronidazole (25 mg/kg bwt b.i.d. per os) are generally the antibiotics of choice in order to prevent bacterial infectious complications. Tetanus vaccination records should be checked, or anti-tetanus serum should be administered (Freeman 1989). Non-steroidal anti-inflammatory drugs such as flunixin meglumine (1.1 mg/kg bwt b.i.d. i.v.) are required to keep the horse comfortable and to prevent further swelling of the surgical site (Whitfield-Cargile et al. 2013).

While feeding the horse through the oesophagostomy tube, care should be taken that the caloric requirements are met. Other challenges with tube feeding are electrolyte and acid-base imbalance (because of the loss of saliva) and shortage of fibrous feed with the risk of colic developing. Loss of saliva through the fistula can cause hyponatraemia, hypochloraemia, transient metabolic acidosis and then metabolic alkalosis (Freeman 1989; Craig et al. 1989). These changes need to be anticipated by providing electrolytes in the liquid feeding ration and by continuous monitoring of the electrolyte balance (Craig et al. 1989; Freeman 1989; Cortley and Marr 1998). Daily monitoring of packed cell volume and total protein are good indicators for hydration status and sufficiency of feed intake (Risnes and Mair 2003; Bezdekova 2012). The horse should receive 4–6 meals a day, calculated on bodyweight basis, containing a commercially available pelleted feed (like Coolstance or Equine Senior), have access to fresh water and supplementation of sodium chloride and potassium chloride should keep the hydration/electrolyte status balanced and should allow the horse to maintain its weight. In this case, the horse lost weight (dropped to 370 kg, BCS 3/10) while the tube was in place (11 days in total) but recovered quite quickly when the tube was removed and the uptake of fibrous feed could be increased. In some cases, where the lesion takes more time to heal, it may be necessary to apply a method of hyperalimentation mainly with oil and sucrose in order to meet the horse’s long-term requirements (Read et al. 2002). Close monitoring of colic signs is required because of the changes in feed, stall confinement and antibiotic therapy being risk factors (Whitfield-Cargile et al. 2013).

In this case, there did not seem to be any long-term complications. Other case reports describe recurrent obstruction, strictures, persistent respiratory disease, Horner’s syndrome or recurrent laryngeal nerve paralysis and fistula formation (Craig et al. 1989; Read et al. 2002; Whitfield-Cargile et al. 2013).

Conclusion
Oesophageal rupture is an uncommon condition in horses with a challenging diagnosis when closed but should be treated carefully to prevent further complications. When the oesophageal wound is closed at skin level, feed, saliva and mucus get entrapped in the surrounding tissues as opposed to an open oesophageal wound where drainage is clearly visible. The critical points in the treatment of this condition consist of careful and selective debridement, wound cleaning, broad-spectrum antimicrobial therapy and ensuring nutritional requirements are met. This case report described the successful management of an oesophageal rupture after obstruction by placing an oesophagostomy tube through the initial defect, allowing granulation tissue to form in order to initiate closure. Long-term treatment, including surgical debridement, the placement of an indwelling tube and intensive nursing, resulted in a good prognosis for this pony.

Authors’ declaration of interests
No conflicts of interest have been declared.

Ethical animal research
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Authorship
P. Randleff-Rasmussen contributed to study design, study execution, and data analysis and interpretation. E. Leus...
Manufacturers’ addresses

1Boehringer Ingelheim, Auckland, East Tamaki, New Zealand.
2Zoetis, Sandton, South Africa.
3MSD Animal Health, Kenilworth, New Jersey, USA.
4Fresenius Kabi, Midrand, South Africa.
5Virbac, Centurion, South Africa.
6Aspen Pharmacare, Durban, KwaZulu-Natal, South Africa.
7Biotech, Koelenhof, Stellenbosch, South Africa.
8Adcock Ingram, Midrand, South Africa.
9Bayer, Isando, Kempton Park, South Africa.
10Stance Equitec, Seventeen Mile Rocks QLD 4073, Australia (dry matter, 3585 kcal/kg DE, Crude protein 20% or 200 000 mg/kg, crude fibre 15%, fat 9%, NSC 11%, Ca 0.06%, P 0.65%).
11Kyron Laboratories, Johannesburg, Benrose, South Africa.

References