Postanaesthetic bilateral laryngeal paralysis in a pony – a case report

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Abstract

A twenty-one-year-old 420 kg pony underwent general anaesthesia for colic surgery. After recovery, the pony developed severe inspiratory stridor and collapsed. Emergency tracheotomy was performed immediately. Endoscopic examination of upper airways confirmed total bilateral laryngeal paralysis. For three months, the pony was kept alive with a non-permanent tracheostomy. Unilateral (left) laryngeal nerve recovery occurred after three months. The tracheostomy tube was removed and secondary intention healing allowed. Tracheostomy site healed uneventfully.

Horse, anaesthesia, recovery, tracheotomy

Bilateral laryngeal paralysis (BLP) is a rare, life-threatening event, leading to upper airway obstruction. It has been associated with hepatic encephalopathy, organophosphate intoxication and lead toxicosis (Rose et al. 1981; Duncan and Brook 1985; Dixon et al. 2001; Allen 2010). Other possible causes include trauma in the neck region, neoplasia, perivascular administration of irritant substances in the jugular vein, or guttural pouch mycosis. Temporary post-anaesthetic bilateral laryngeal paralysis after transoral intubation has been previously described in horses, dogs, cats, and humans (Abrahamsen et al. 1990; Dixon et al. 1993; Schachter and Norris 2000; Cinar et al. 2005). Recurrent laryngeal nerve (RLN) damage causing bilateral arytenoid cartilage hypomobility is usually observed immediately after extubating, but it can manifest anytime within the first 24 h after general anaesthesia, especially after prolonged anaesthesia in dorsal recumbency (Flaherty et al. 1996; Canada et al. 2017).

The aetiology of this condition is not fully known, and several theories have been discussed. Hyperextension of the head during anaesthesia or hoist transport can cause tension on laryngeal nerves, causing traumatic insult to RLN and its temporary or permanent damage. Hypoxaemia or hypotension leading to compromised blood flow, decreased capillary perfusion and tissue hypoxia may contribute to RLN insult (Abrahamsen et al. 1990; Flaherty et al. 1996).

Ponies seem to be more susceptible to bilateral laryngeal paralysis compared to other breeds. In a study of 375 cases of laryngeal paralysis, 12 cases of bilateral laryngeal nerve involvement were detected. Interestingly, 11 out of these 12 cases were ponies, compared to unilateral laryngeal hemiplegia, where ponies were minimally represented (Dixon et al. 2001).

Case history

A twenty-one-year-old 420 kg pony gelding used for equine-assisted therapy, without any history of exercise intolerance or abnormal respiratory noise during exercise, was admitted to the Equine Clinic at VETUNI Brno with an episode of acute onset of abdominal pain. Prior to the admission, the pony had been treated with flunixin meglumine, metamizole, intravenous and oral fluids.

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Clinical examination and treatment prior to surgery

Clinical examination revealed tachycardia (48 beats per min [bpm]), sticky hyperaemic mucous membranes with capillary refill time 3 s, decreased gut sounds and rectal temperature of 37.3 °C. Respiratory rate was increased (20 per min) but no dyspnoea or abnormal respiratory noise was apparent during clinical examination. Rectal examination and ultrasound of the abdominal cavity were inconclusive. A 14-gauge catheter was placed into the right jugular vein and maintenance fluid therapy with isotonic sodium chloride solution (2 ml/kg/h) was started. Pony passed faeces and did not show any signs of abdominal pain until the next morning when he was fed. After feeding, he showed severe signs of abdominal pain (tachycardia 80 bpm, sweating, rolling). Segmental intestinal wall oedema was palpated during rectal examination and confirmed by ultrasonography, and the pony underwent exploratory laparotomy under general anaesthesia. A stomach tube was not placed prior to surgery. Potassium penicillin 22,000 IU/kg (Penicilin G 5 MIU, BB Pharma a.s., Czech Republic), gentamicin 6.6 mg/kg (Aagent 50 mg/ml, Fatro S.p.A., Italy) and flunixin meglumine 1.1 mg/kg (Flunbix 50 mg/ml, Fatro) were administered intravenously 45 min prior to induction of anaesthesia.

General anaesthesia and perioperative management

The pony was sedated with xylazine 1.1 mg/kg i.v. (Xylazine Ecuphar 20 mg/ml, Ecuphar N.V., Belgium), and general anaesthesia was induced with diazepam 0.02 mg/kg i.v. (Apaurin 10 mg/2 ml, Krka, Slovenia) and ketamine 2.2 mg/kg IV (Narkamon 100 mg/ml, Bioveta a.s., Czech Republic). He was orotracheally intubated using a 20 mm internal diameter cuffed silicone endotracheal tube (Smiths Medical Pm Inc., Waukesha, USA). Orotracheal intubation was successful on third attempt. The pony was transported to surgical table using electric hoist. Padded hobbles were attached to each limb and the head was supported during the transport.

The pony was placed in dorsal recumbency and connected to a large animal rebreathing system (Stephan GmbH; Gackenbach, Germany). Anaesthesia was maintained with isoflurane (Isoflurin, Vetpharma Aimal Health, S.L., Spain) to effect (sluggish palpebral reflex maintained) in 100% oxygen (5 l/ min) and a continuous rate infusion of medetomidine 0.0035 mg/kg/hour (Dorbene 1 mg/ml, Laboratorios SYVA S.A.U., Spain).

A 22-gauge arterial catheter was placed into the facial artery to monitor arterial blood pressures invasively and to obtain arterial blood samples for blood gas analysis. Vital signs were monitored using a multiparameter monitor (DatexOhmeda S/5, Datex-Ohmeda Inc, Madison, USA). Initial blood gas analysis and pH determination (Radiometer ABL 830, Radiometer Medical ApS, Denmark) at 25 min after beginning of anaesthesia showed the following results: pH 7.246, pCO_2 52 mmHg, pO_2 69 mmHg, ABE-5.2, estimated shunt 31%. This value was calculated by the analyser using a formula described in an official reference manual (Radiometer Medical).

Spontaneous ventilation was switched to intermittent positive pressure ventilation (IPPV), (respiratory rate of 7 and peak inspiratory pressure between 20 to 25 cm H_2O) after blood gas analysis results were obtained. Pulse oximeter with a probe on the tongue was used to monitor peripheral saturation of haemoglobin. In the first 10 min after anaesthesia induction, peripheral haemoglobin saturation dropped to 86%, but remained over 90% after IPPV was initiated. Mean arterial blood pressure was maintained above 70 mmHg by dobutamine hydrochloride infusion (0.4–1.2 μg/kg/min). Continuous rate of isotonic saline (4 ml/kg/h) was administered during anaesthesia. Arterial blood gas analysis measurements were not repeated but SpO_2 ranged from 86 to 96% (mean 94%) and $ETCO_2$ ranged from 36 mmHg to 44 mmHg (mean 40 mmHg). Mean arterial blood pressure was 91 mmHg ranging from 62 mmHg to 128 mmHg, with mean systolic pressure of 122 mmHg and mean diastolic pressure of 91 mmHg. Heart rate ranged 30 to 40 bpm (mean 35 bpm).

Respiratory rate ranged from 4 to 9 (mean 6) breaths per minute. Duration of general anaesthesia was 175 min, with the surgery time of 125 min.

Median laparotomy was performed, and an oedematous mass was found in the left dorsal colonic wall compromising the lumen of the colon. Colonic content was evacuated via pelvic flexure enterotomy. Approximately 20 cm of the left dorsal colon was resected and end to end colonic anastomosis was performed. Histopathological examination of resected mass revealed chronic pyogranulomatous inflammation.

After the surgery, the pony was transported to a padded recovery box, where it was placed into lateral recumbency. Oxygen was administered via free flow (10 l/min). Ephedrine 1% nasal drops were instilled intranasally (100 mg total). Sedation with medetomidine (0.002 mg/kg) was administered intravenously with onset of rapid nystagmus. Eight minutes after sedation, the pony was breathing regularly, the endotracheal tube was removed, and free flow oxygen was administered intranasally. No inspiratory or expiratory sounds were present after extubating, and the pony was free recovered. Twenty-five minutes after extubating, the pony moved into sternal recumbency and stood up 10 min later at the first attempt. Strong inspiratory stridor was present for 10 breaths immediately after recovery, but it resolved without any intervention. Forty-five minutes later, the pony was walked into intensive care unit approximately 20 m away. In the intensive care stall, the pony tried to vocalize, became distressed, and suffered an episode of acute onset of dyspnoea and collapsed.

Treatment

Emergency tracheotomy was performed in lateral recumbency, without local or general anaesthesia, only with an assistant holding the pony's head during the procedure. Tracheotomy was performed on unclipped and unprepared site using the technique described by Prange (2019). Large haemostatic forceps were used to insert a J-shaped metallic tracheostomy tube. The pony recovered immediately and stood up. The tracheostomy tube was kept in position using elastic bandage.

Oxygen was administered for the next 60 min through the tracheotomy site and a single dose of dexamethasone (0.1 mg/kg; Colvasone 2 mg/ml, Norbrook Laboratories Ltd, Northern Ireland) was administered intravenously. After 60 min, the respiratory rate decreased to 16 breaths per minute and no signs of pulmonary oedema or respiratory distress were present. Respiratory tract endoscopy at rest was performed the following morning, showing complete bilateral laryngeal paralysis with passive adduction of both arytenoid cartilages to the midline with marked reduction of the lumen of the rima glottis. No active movement was evident after nostril occlusion and no slap response could be induced on any side. Reddening and mucosal inflammation from the endotracheal tube was present on the ventral aspect of the trachea, more prominent at the level of the endotracheal cuff. Guttural pouches were without any abnormal finding. Haematology and biochemical parameters (including hepatic enzymes and bile acids) were unremarkable, except for leukopaenia, 3.24×10^9/l (reference interval: $5.5-12 \times 10^9$), neutropaenia 1.86×10^9/l (reference interval: 2.5- 8×10^9), lymphoocytopaenia 0.56 × 109/l (reference interval: $1.1-7 \times 10^9$) and erythrocytopaenia 5.43×10^{12} (reference interval $6–12 \times 10^{12}$).

Outcome and follow-up

Respiratory tract endoscopy was repeated on the non-sedated pony on days 3, 7, 10 and 15 post surgery, without any improvement of arytenoid cartilage function. Tracheal reddening had resolved by day 3 post surgery. Permanent tracheostomy was suggested, but refused by the owner, and the pony was discharged with the tracheotomy tube in place. Recommendations given to the owner were to clean the wound using 0.5% povidone iodine solution and replace the tracheotomy tube twice daily. No further surgical debridement of the tracheotomy wound was necessary.

Two weeks after discharge, the pony developed pruritus at the tracheostomy site and at the top of the neck, where the bands from tracheotomy tube were tightened. Elastic bandages holding the tube in place were then replaced by custom made leather straps, which were well tolerated by the pony.

Follow-up endoscopic examination three months after surgery revealed recovery of the left laryngeal arytenoid cartilage function. Right arytenoid cartilage showed no active inspiratory movement. The tracheotomy tube was removed at this point, and the tracheotomy wound was left to heal by secondary intention.

Repeated endoscopic examination was offered to but refused by the owner, as the pony was without clinical problems and showed no inspiratory/expiratory stridor. Telephone follow-up was done three years later when the pony was being used again for light hobby riding to the same degree as prior to surgery. He did not show any episodes of respiratory distress. The owner reported episodes of occasional coughing at the beginning of the ridden exercise, which were not further examined. The tracheotomy wound healed uneventfully and endoscopic examination was not repeated.

Discussion

In the present case, the sudden onset of dyspnoea and collapse after general anaesthesia suggested that BLP developed because of damage to n. laryngeus recurrens, which had happened during the general anaesthesia. The pony was used only for light ridden exercise and equine-assisted therapy and had not performed any high intensity work. No upper-airway endoscopy was performed prior to surgery and it is unknown if some degree of laryngeal dysfunction had been present prior to general anaesthesia.

Irritant perivascular injection to the jugular vein may cause iatrogenic trauma to the laryngeal nerve (Marks et al. 1970; Gilbert 1972) but no irritant substances were administered either by the referring veterinarian or at the clinic. Patency of both jugular veins was checked by ultrasound after surgery and no signs of perivascular damage or inflammation were detected.

Compression of RLN or its branches by the endotracheal tube or a noncompliant pony's neck position and crushing injury of RLN by the endotracheal tube can lead to bilateral paralysis, especially when combined with local myopathy/myositis (Dixon et al. 1993). Mild reddening and mucosal irritation from the endotracheal tube on the ventral aspect of the tracheal wall was noticed on endoscopic examination. However, no correlation has been described between the severity of post-intubation laryngeal/tracheal trauma and decrease of the laryngeal function (Bradbury et al. 2008). Muscle enzyme activity was within normal limits following the surgery. The pony was transported to and from the surgical theatre using an electric hoist, with hobbles on his legs but with his head supported during the transport. No obvious hyperextension of the head and neck region occurred during the transport.

Endoscopic examination precluded guttural pouch mycosis as a possible cause of BLP. During anaesthesia, mild permanent hypoxaemia was present. Hypoventilation with subsequent hypoxaemia during general anaesthesia for colic surgery is a well-known complication in equine patients. In a study on 600 horses, hypoxaemia was present in 17% (Trim et al. 2015). Hypoxaemic injury to the laryngeal nerve could cause temporary or permanent laryngeal paralysis. Only one single case of temporary bilateral laryngeal paralysis due to hypoxaemia has been described so far (Abrahamsen et al. 1990). In this case, laryngeal function gradually recovered in the following 7 days. Two other published case reports describing BLP in equids describe horses that underwent surgery in the mandibular region with their head and neck extended (Abrahamsen et al. 1990; Dixon et al. 1993). In our case, the head and neck were more extended than usual

because of the pony's size, as the pony was placed on a standard size large animal surgical table (HaicoTelgte II, Haico, Finland). However, no special surgery table or head support has ever been used at our clinic for ponies of this size and no similar complications were recorded during the 1,922 inhalation anaesthesia cases performed in the years 2007–2018.

Other cases of bilateral laryngeal paralysis in ponies were all diagnosed subsequently to hepatic disease (Pearson 1991; McGorum et al. 1999; Dixon et al. 2001; Hughes et al. 2009). Affected animals showed clinical signs of hepatic encephalopathy and hyperammonaemia. The pathogenesis of BLP as a complication of hepatic diseases is unclear, however, due to lack of histopathological abnormalities in the laryngeal nerves or muscles, it is more likely to be a neuromuscular dysfunction rather than morphological damage to RLN. Gamma-glutamyl transferase and bile acids, which are the most sensitive and reliable biochemical markers for hepatic disease in horses, were not elevated in our case, which precluded hepatic encephalopathy to be the cause of BLP.

Ammonia concentrations were not measured in this case, as it is known that hyperammonaemia without concurrent hepatic disease is not associated with laryngeal paralysis (McConnico et al. 1997).

Bilateral laryngeal paralysis subsequent to lead toxicosis and organophosphate intoxication has been reported previously. Poisoning by lead and organophosphates leads to irreversible bilateral laryngeal paralysis because of degenerative lesions in long peripheral nerves (Rose et al. 1981; Duncan and Baker 1987; Cahill and Goulden 1987). Neither organophosphate nor lead concentrations were measured in this pony, but it is unlikely that they were responsible for the paralysis in a pony showing no other signs of toxicosis.

Pituitary adenomas were present in all three euthanized ponies with bilateral laryngeal paralysis on postmortem examination in the study by McGorum et al. (1999), although ponies were clinically not showing signs of hyperadrenocorticism. Pituitary adenomas are quite common in older ponies and their role in pathogenesis of the actual disease is unknown. No other central nervous clinical signs were present in our pony and adrenocorticotropic hormone levels were within normal limits; however, the presence of an adenoma cannot be excluded.

In general, it appears that ponies are more susceptible to bilateral laryngeal paralysis than the larger breeds of horses (Dixon et al. 2001).

Conclusion

To the authors' knowledge, this is the first case of long-term post-anaesthetic BLP that recovered over a prolonged period of time. With motivated owners and a compliant animal, temporary tracheostomy can be maintained and long-term recovery of arytenoid cartilage function is possible.

Conflict of interest

The authors declare that there is no conflict of interests regarding the publication of this paper.

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