Schweiz. Arch. Tierheilk.
© 2011 by Verlag Hans Huber, Hogrefe AG, Bern

K. O. Veres-Nyéki, C. Graubner, F. Aloisio, C. Spadavecchia, Band 153, Heft 9, September 2011, 401–404
DOI 10.1024/0036-7281/a000232

Pulmonary edema at recovery after colic operation with in-situ nasogastric tube in a horse

K. O. Veres-Nyéki [1], C. Graubner [2], F. Aloisio [3], C. Spadavecchia [1]

[1] Department of Clinical Veterinary Medicine, Anaesthesiology Division, [2] Equine Clinic and [3] Institute for Animal Pathology, University of Berne

Summary

After an uneventful general anesthesia, in a horse negative pressure pulmonary edema developed due to acute upper airway obstruction during the anesthetic recovery phase after colic surgery. No pathologic alteration of respiration was observed until the horse stood up and began suffocating. The horse had recovered with the nasogastric tube in situ. This, together with the postmortem diagnosis of laryngeal hemiplegia resulted in impairment of airflow through the larynx and development of pulmonary edema. Our objective is to alert clinicians about the possible hazard of recovery with an in-situ nasogastric tube.

Keywords: pulmonary edema, post-anesthetic complication, equine, upper airway obstruction, laryngeal hemiplegia

Entstehung eines Lungenödems in der Aufwachphase eines Pferdes mit in-situ belassener Nasenschlundsonde nach Kolikoperation

Nach einer unproblematisch verlaufenen Anästhesie entwickelte ein Pferd in der Aufwachphase eine plötzlich auftretende obere Atemwegsobstruktion, welche durch den dadurch hervorgerufenen negativen Druck ein akutes Lungenödem verursachte. Es konnten keine pathologischen Veränderungen der Atmung beobachtet werden bis zum Zeitpunkt als das Pferd aufstand und Erstickungsanzeichen zeigte. Für die Aufwachphase wurde die Nasenschlundsonde in situ belassen. Dies, zusammen mit der Tatsache einer postmortem diagnostizierten Hemiplegie des Larynx, führte zu einer Beeinträchtigung des Luftflusses im Larynx und zur Entstehung eines Lungenödems. Mit diesem Fallbericht möchten wir Tierärzte auf mögliche Risikofaktoren einer Aufwachphase mit Nasenschlundsonde aufmerksam machen.

Schlüsselwörter: Lungenödem, post-anästhetische Komplikationen, Pferd, Obstruktion der oberen Atemwege, Hemiplegia laryngis

Case history

Clinical and laboratory findings

An 8-year-old warmblood stallion weighing 540 kg was referred to the Equine Clinic of the University of Berne with colic symptoms. On preoperative examination, the heart rate was 44 beats/minute, respiratory rate was 12 breaths/minute, no pathological lung sounds were found at auscultation, and the mucous membranes were red with 2 s of capillary refill time. The animal was anxious and showed moderate signs of abdominal discomfort. Rectal palpation of the distended small intestines indicated one of the loops entrapped in the right inguinal canal. The stomach was full of contents which were emptied by flushing with water. The nasogastric tube was left in-situ for the operation to avoid regurgitation and aspiration of reflux. The packed cell volume was 35 %, total solids were 77 g/L, and leucocytes were 7.7 10^9/L.

Anesthesia

Preoperatively, flunixin meglumine (1.1 mg/kg, Flunixin, Biokemia), gentamycin (6.5 mg/kg, Pargenta 50, Dr. Gräub) and penicillin (30, 000 IU/kg, Penicillin Natrium, Streuli) and 10 l of lactated Ringer's solution

(*Ringer-Lactat*, Dr. G. Bichsel) were administered via a 12 G polyurethane catheter placed into the left jugular vein. Romifidine (0.04 mg/kg, Sedivet, Boehringer Ingelheim) and L-methadone (0.05 mg/kg, L-Polamivet, Intervet) were given intravenously for sedation and anesthesia was induced on a tilting operational table with guaifenesin (20 mg/kg, Myolaxin, Vetoquinol) administered until ataxia, followed by the administration of diazepam (0.05 mg/kg, Valium, Roche) and ketamine (2.2 mg/kg, Narketan-10, Vetoquinol) IV. The induction quality was good and the trachea was smoothly intubated with a 26 mm-inner-diameter silicon endotracheal tube. The horse was positioned on the table into dorsal recumbency, with the head elevated and the neck bent in a physiological position. Anesthesia was maintained with isoflurane (Attane, Provet) carried in 50 % oxygen and lidocaine (Lidocain 2 %, Streuli) at continuous rate infusion (CRI) 30 mcg/kg/min after 1.5 mg/kg bolus administered over 15 minutes. Infusions of Ringer's lactate solution and dobutamine CRI (Dobutrex, Teva) 0.3 mcg/kg/min were immediately started. Heart frequency, invasive blood pressure, respiratory frequency, SpO_2, $EtCO_2$ and EtIso were monitored continuously with a multiparameter patient monitor (Datex-Ohmeda S/5) and values were recorded every 5 minutes on the anesthesia protocol together with the evaluation of movements, palpebral reflex activity and eye position. The horse breathed spontaneously and the tidal volume estimated by an H-lite spirometry sensor was adequate (10 ml/kg) during the anesthetic period.

Recovery phase

The lidocaine CRI was disconnected when the surgeons began to close the abdomen. After 210 minutes of uneventful general anesthesia, the horse was transported to the recovery box where romifidine (0.01 mg/kg) was given as the horse already showed nystagmus. The nasogastric tube was fixed with tapes to the halter and left in-situ for the recovery phase. No edema was observed around the horse's head or neck but phenylephrine nose drops (10 – 10 ml, Phenylephrini hydrochloridum 1.5 mg/ml, Streuli) were administered into both nasal cavities as a part of the standard recovery protocol. During recovery, supplementary oxygen was first insufflated at a rate of 10 L/min into the endotracheal tube. Following extubation (after 20 minutes), oxygen was insufflated via the ventral nasal meatus. The horse spent 15 more minutes in a lateral position, then 10 minutes calmly in sternal recumbency and, subsequently, during which no stridor, snoring, dyspnoe or other type of respiratory changes were observed.

The horse stood at the first attempt assisted with head-tail rope, subsequently bent the neck and immediately started panicking. Tachypnoea with an extremely high, sharp respiratory noise was observed during this period. Two minutes later the horse collapsed and rolled violently in the recovery box making it impossible to approach it. Abdominal breathing was observed without chest wall movement and acute upper airway obstruction was suspected. Approximately half liter of blood-tinged, frothy fluid discharged from the lower nostril. As soon as possible the horse was sedated with romifidine (0.01 mg/kg) and L-methadone (0.025 mg/kg) IV and then a small-diameter tube connected to the oxygen cylinder was positioned into the pharynx with maximal oxygen flow (over the scale). The horse had tachycardia (80/min), the mucosal membranes seemed to be pale pink with prolonged capillary refill time but poor lighting made its evaluation difficult. Crackles and wheezes were audible throughout the chest and the trachea with maximal stridor above the larynx. The nasogastric tube was removed and the quality of respiration improved immediately, so tracheostomy was not performed.

One hour later the horse stood up at first attempt without assistance. The administration of oxygen was stopped. A few minutes later an arterial blood sample showed a PaO_2 of 38 mmHg. Therefore, oxygen therapy was continued as well as furosemide (2.5 mg/kg, Dimazon, Intervet) was given IV in order to improve oxygenation and reduce lung edema. Four hours later, respiration was almost normal and PaO_2 was over 70 mmHg breathing room air. Blood gas measurement after 24 hours indicated normal gas exchange ($PaO_2 = 91.2$ mmHg, at 720 mmHg ambient pressures). Twelve days later, the horse again showed severe colic symptoms and the owner decided to perform euthanasia. Pathological examination revealed left laryngeal hemiplegia.

Discussion

Pulmonary edema is a relatively rare but severe post-anesthetic complication in horses which mainly results from upper airway obstruction during recovery. Most often obstruction of the orotracheal tube or the nostrils (Kaartinen et al., 2010) is responsible for the symptoms. The latter is typically caused by nasal congestion (Ball und Trim, 1996; Borer, 2005) due to dependent position of the head in dorsal recumbency. Laryngeal dysfunction due to surgical manipulation (Tute et al., 1996) or over-extension of the neck and bilateral arytenoid paralysis related to hypoxic injury (Abrahamsen et al.; 1990, Dixon et al., 1993; Flaherty et al., 2005) were also described as causes of upper airway obstruction evoking pulmonary edema. Besides these negative pressure pulmonary edemas, some cases were reported in which hydro-pool recovery (Tidwell et al., 2002), alveolar hypoxia (Ball und Trim, 1996), air embolism (Holbrook et al., 2007) or pulmonary microembolism (Jones et al., 1988) were suspected as initiating factors of the disease.

Affected horses are distressed and tachypnoic with labored breathing, cyanotic mucous membranes, sweating and violent movements. The blood-tingled frothy fluid

discharging from the nostrils and mouth or endotracheal tubes is characteristic of the diagnosis of post-anesthetic pulmonary edema in horses (Doherty und Valverde, 2006; Muir und Hubbell, 2009). In the case reported here, the diagnosis of pulmonary edema was confirmed by clinical signs. The sudden onset and quick relief of the symptoms indicate that temporary airway obstruction evoked negative pressure pulmonary edema in our patient.

Edema of the head usually occurs in horses positioned in dorsal recumbency for long surgeries. Elevated positioning of the head prevents edema of the nasal turbinate and alar fold while phenylephrine nosedrops administered at the end of the surgery reduce nasal congestion (Coumbe, 2005; Flaherty et al., 2005).

Bilateral laryngeal paralysis is a result of an ischemic insult to the nerves supplying the larynx. The complete recovery of function takes weeks and it fails to improve with pharyngeal oxygen insufflations (Abrahamsen et al., 1990; Dixon et al., 1993). Although left laryngeal hemiplegia was neither reported in the anamnesis of the horse nor diagnosed during hospitalization, the atrophy of the crycoarytenoideus muscle discovered during the pathological exam indicated its presence before the anesthesia. However, left laryngeal hemiplegia has never been reported as a single cause of laryngeal obstruction resulting in pulmonary edema, but rather as a predisposing factor.

Nasogastric intubation is an essential part of the medical attendance in the case of colic. It helps to relieve gastric distension and pain; furthermore, it prevents gastric rupture. Stomach tubes are usually kept in place during anesthesia to drain gastric content and reduce the risk of aspiration (Doherty und Valverde, 2006; Muir und Hubbell, 2009). For the recovery, the silicone nasogastric tube was fixed to the halter with tape and probably kinked in the pharynx as the horse bent its neck when getting up. The authors suppose that it either pushed the paralyzed arytenoid cartilage into the laryngeal aperture or it made an obstruction on its own as the reextension of the neck after collapse and removal of the nasogastric tube resulted in immediate improvement of respiration. Some authors recommend the removal of the tube at the end of the surgery (Doherty und Valverde, 2006), but that can provoke severe nasal hemorrhage (Trim et al., 1997). Because colic patients are at risk for coagulopathy (Dallap et al., 2009) and because the gastric tube tends to stick to the mucosa if left in situ for a prolonged time, the occurrence of nasal bleeding after stomach tube removal is not rare. If this happens while the horse is still recumbent, the blood loss can be particularly severe. To avoid this potentially fatal complication, we recover colic horses with the nasogastric tube left in situ if they have one for the surgery, as to our experience fatal bleeding is less likely in the standing position and the risk of blood aspiration is lower.

In the case of post-anesthetic pulmonary edema, immediate intervention is crucial. The first line treatment is to restore the patency of the airway either with re-intubation or with tracheostomy and to administer oxygen. Medical treatment consists of diuretics, corticosteroids and sedation (Senior, 2005; Doherty und Valverde, 2006; Muir und Hubbell, 2009). In our case, oxygen administration was immediately started and removal of the nasogastric tube considerably improved the quality of respiration. Because the labored breathing ceased and no cyanosis was present, tracheostomy was not performed, but since crackles and wheezes over the lungs and low PaO_2 level were detected, furosemide was administered. Since a clear improvement of lung function was observed and then confirmed by blood gas analysis, corticosteroids were not considered essential and were avoided due to the possible negative effect on wound healing after major abdominal surgery.

In conclusion, when a horse is in recovery with an in-situ nasogastric tube, the potential for laryngeal obstruction due to kinking the tube must be seriously taken into account. The post-anesthetic period carries the most potential hazards in equine anesthesia; awareness of possible complications is mandatory to reduce morbidity and mortality.

References

Abrahamsen E. J., Bohanon T. C., Bednarski R. M., Hubbell J. A., Muir W. W., 3rd: Bilateral arytenoid cartilage paralysis after inhalation anesthesia in a horse. J. Am. Vet. Med. Assoc. 1990, 197: 1363–1365.

Ball M. A., Trim C. M.: Post anaesthetic pulmonary oedema in two horses. Equine Vet. Educ. 1996, 8: 13–16.

Boere K. E.: Pulmonary oedema associated with anaesthesia for colic surgery in a horse. Vet. Anaesth. Analg. 2005, 32: 228–232.

Coumbe K.: Anaesthetic complications and emergencies: Part 2. Equine Vet. Educ. Manual 7. 2005, 81–88.

Dallap Schaer B. L., Epstein K.: Coagulopathy of the critically ill equine patient. J. Vet. Emerg. Crit. Care (San Antonio). 2009, 19: 53–65.

Dixon P. M., Railton D. I., Mcgorum B. C.: Temporary bilateral laryngeal paralysis in a horse associated with general anaesthesia and post anaesthetic myositis. Vet. Rec. 1993, 132: 29–32.

Doherty T., Valverde A.: The respiratory system. In: Manual of Equine Anesthesia and Analgesia. Eds. T. Doherty, A. Valverde, Blackwell Publishing, Oxford, 2006, 37–66.

Flaherty D., Nolan A., Reid J.: Complications during recovery from anesthesia in the equine patient. Equine Vet. Educ. Manual 7. 2005, 53–59.

Holbrook T. C., Dechant J. E., Crowson C. L.: Suspected air embolism associated with post-anesthetic pulmonary edema and neurologic sequelae in a horse. Vet. Anaesth. Analg. 2007, 34: 217–222.

Jones R. S., Payne-Johnson C. E., Seymour C. J.: Pulmonary micro-embolism following orthopaedic surgery in a Thoroughbred gelding. Equine Vet. J. 1988, 20: 382–384.

Kaartinen M. J., Pang D. S., Cuvelliez S. G.: Post-anesthetic pulmonary edema in two horses. Vet. Anaesth. Analg. 2010, 37: 136–143.

Muir W. W. 3rd, Hubbell J. A.: Anesthetic-Associated Complications. In: Equine Anesthesia: Monitoring and Emergency Therapy. Eds: W. W. 3rd Muir, J. A. Hubbell, Saunders Elsevier, St Luis, 2009, 397–417.

Senior M.: Post-anaesthetic pulmonary oedema in horses: a review. Vet. Anaesth. Analg. 2005, 32: 193–200.

Tidwell S. A., Schneider R. K., Ragle C. A., Weil A. B., Richter M. C.: Use of a hydro-pool system to recover horses after general anesthesia: 60 cases. Vet. Surg. 2002, 31: 455–461.

Trim C. M., Eaton S. A., Parks A. H.: Severe nasal hemorrhage in an anesthetized horse. J. Am. Vet. Med. Assoc. 1997, 210: 1324–1327.

Tute A. S., Wilkins P. A., Gleed R. D., Credille K. M., Murphy D. J., Ducharme N. G.: Negative pressure pulmonary edema as a post-anesthetic complication associated with upper airway obstruction in a horse. Vet. Surg. 1996, 25: 519–523.

Corresponding author

K. O. Veres-Nyéki
Tel.: + 41 (0)31 631 2111
Fax: + 41 (0)31 631 2644
E-mail: kata.veres@vetsuisse.unibe.ch

Received: 14 January 2011
Accepted: 27 April 2011