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Acute life-threatening laryngeal dysfunction in a draft horse recovering from general anesthesia: a case report

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Abstract

A 13-year-old Shire horse was anesthetized for an elective orthopaedic procedure. During recovery from anesthesia, the occurrence of severe acute dyspnea required a second anesthetic to allow endoscopy-guided nasotracheal intubation. Endoscopic findings were decreased mobility and swelling of the arytenoids with narrowing of the laryngeal *aditus*. Owing to a dislodgement of the nasotracheal tube during recovery, a third anesthetic was carried out to perform emergency tracheostomy. Recovery from the third anesthetic was long and the horse developed a post-anesthetic myopathy. The clinical conditions improved during the following 48 hours, and an endoscopic examination performed prior to discharge revealed unremarkable laryngeal function. It was hypothesized that mechanical stimulation of the trachea during the phases of intubation and extubation caused traumatic laryngeal dysfunction, and that draft horses might require additional care during the perioperative period.

Keywords: Anesthesia, draft horse, larynx, perioperative complication

1. Introduction

Equine anesthesia is associated with an incidence of fatalities of 1.1% [1]. Compared to other domestic mammals, horses have a higher risk for peri-anesthetic complications, of which bone fractures, myopathies and respiratory impairment have been frequently reported [1-3]. Among respiratory complications, upper airway obstruction and pulmonary edema have been described in equine patients [4-6]. The former mainly occurs after extubation, as a result of either nasal obstruction by secretions or edema, or displacement of the soft palate owing to a combination of mechanical and pharmacological effects [7]. Regarding pulmonary edema, this complication is most commonly the ultimate result of upper airway obstruction. In an attempt to overcome the non-patent airways, large negative intra-thoracic pressures are generated, resulting in movement of fluid from the vascular compartment to the interstitium and, ultimately, into the alveolar space [3, 8-11]. Other causes of peri-anesthetic pulmonary edema, including microembolism following orthopaedic surgery and side effects of opioids and alpha 2- adrenoreceptor agonists [12,13], have also been reported in horses.

This report describes the management of a life-threatening upper airways complication and post-anesthetic myopathy in a draft horse recovering from general anesthesia for an elective procedure. The occurrence of transient post-anesthetic laryngeal dysfunction in the absence of pre-existing respiratory or neurological conditions has been previously documented in horses [4,5]. Nevertheless, to the best of the authors' knowledge, cases that resolved within 24 hours have not been reported yet.

2. Case description

A 13-year-old female Shire was referred to the Equine Referral Hospital of the Royal Veterinary College for evaluation of an acute onset lameness of the right hind limb associated with digital sheath effusion of three weeks duration. On examination, lameness was mild and was resolved with intra-thecal diagnostic analgesia of the digital flexor tendon sheath (DFTS). A concurrently performed contrast tenogram showed no obvious abnormality of the *manica flexoria* but did suggest the presence of a constricted plantar annular ligament because contrast did not pass freely into the proximal pouch of the DTFS after injection via the distal pouch. Ultrasonographic examination of the DFTS revealed marked synovial thickening and effusion of the tendon sheath but no other significant abnormalities. Tenoscopy under general anesthesia was scheduled for the next day upon request of the orthopaedic surgeon.

Pre-anesthetic physical examination revealed heart rate (HR) of 40 beats minute^{-1}, respiratory rate (*f*R) of 12 breaths minute^{-1}, rectal temperature of 37°C, and pink mucous membranes with capillary refill time less than 2 seconds. Body weight and body condition score were 694 kg and 3/5 [14], respectively. Chest auscultation and blood results were unremarkable. An American Society of Anaesthesiologists category of 1 was assigned to the horse; pre-operative withholding time for food was 12 hours whereas water was made available until premedication.

Intramuscular (IM) acepromazine (AceSedate, Jurox, UK; 0.02 mg kg^{-1}) was administered as a tranquilizer, 60 minutes before placement of a 14 G catheter in the left jugular vein. The horse was then transferred from the stable to the induction box, where it received intravenous (IV) romifidine (Sedivet, Boehringer Ingelheim, UK; 0.04 mg kg^{-1}) and morphine (Morphine Sulphate, Mercury Pharmaceuticals, Ireland; 0.1 mg kg^{-1}) as premedication. General anesthesia was induced with IV ketamine (Ketamidor, Chanelle, UK; 2.2 mg kg^{-1}) and midazolam (Dormicum, Roche, UK; 0.05 mg kg^{-1}); induction was smooth and uneventful. Additional ketamine (1 mg kg^{-1}) was administered prior to attempting endotracheal

intubation, owing to the presence of nystagmus and poor myorelaxation that were considered by the anesthesiologist in charge to be indicative of superficial anesthetic depth. A 24 mm internal diameter endotracheal (ET) tube was then inserted into the trachea via the oro-tracheal route and cuffed with a syringe to ensure air-tight seal. Following the patient was transported to theatre by an overhead crane system where it was positioned in dorsal recumbency on the surgical table with the head and neck slightly elevated to avoid their possible over-extension. The ET tube was connected to a circle breathing system, and isoflurane (IsoFlo, Abbott, UK) was delivered in a mixture of oxygen and medical air (1:1). Intermittent positive pressure ventilation was initiated at a rate of 8 breaths/minute with initial setting of 6.5 L and 17 cm H_2O as tidal volume (TV) and peak inspiratory pressure (PIP), respectively. The horse was instrumented with a multiparametric monitor that included electrocardiography, capnography, pulse oximetry and continuous measurement of invasive arterial blood pressure (via facial artery) and oesophageal temperature. Physiological variables were manually recorded on the anesthetic record every 5 minutes. Arterial blood was sampled from an indwelling 21G catheter, placed in the facial artery for continuous blood pressure measurement, and analyzed at 30-60-minute intervals with a portable device (i-STAT blood analyzer and cartridges CG4+; Abbott, NJ, USA). During surgery, the horse received IV fluids (Hartmann's solution, Fresenius Kabi, UK; 5 mL kg hr^{-1}) as well as a continuous rate of infusion (CRI) of romifidine (0.04 mg kg hr^{-1}). A CRI of dobutamine (Dobutamine Concentrate, Hameln Pharmaceutical, Germany; 0.5-1 mcg kg $minute^{-1}$) was initiated before successful arterial catheter placement owing to poor peripheral pulse quality, as judged subjectively by the anesthesiologist in charge.

Tenoscopic examination of the DFTS revealed the presence of a small tear on the lateral border of the *manica flexoria*, as well as constriction of the plantar annular ligament of the fetlock canal; these lesions were treated with resection of the *manica flexoria* and annular ligament desmotomy, respectively.

General anesthesia was without critical events. Transient hypotension (mean arterial pressure less than 70 mmHg) occurred during surgery; however, it was promptly detected and successfully treated with dobutamine infusion. Total anesthesia and surgery time were 200 and 105 min, respectively.

Recovery from anesthesia was assisted, with head-tail-rope method. At recovery, airflow was detected from both nares when the cuff of the ET tube was deflated and judged subjectively as adequate by the anesthesiologist in charge. The trachea of the horse was extubated after spontaneous swallowing resumed. The horse showed an unremarkable breathing pattern during left lateral recumbency, which lasted for approximately 30 minutes. Thereafter, after one unsuccessful attempt to stand, the horse managed to achieve sternal recumbency; at this point, the occurrence of sudden onset severe inspiratory dyspnoea, characterized by marked inspiratory stridor and visible cyanosis of the oral mucous membranes, required emergency reintubation. For this purpose, general anesthesia was induced with IV ketamine (2.5 mg kg^{-1}). Blind orotracheal intubation of the trachea with a 24 mm ET tube was unsuccessfully attempted; it was therefore decided to use endoscopic guidance. Endoscopy revealed swelling of the larynx resulting in narrowing of the laryngeal *aditus*, as well as absence of arytenoid movement during breathing, which suggested bilateral laryngeal dysfunction, possibly as a result of mechanical trauma. The soft palate was not displaced. Nasotracheal intubation was performed under endoscopic guidance, with an 18 mm ET tube, with the intention of leaving the ET tube in place after standing. However, during recovery from anesthesia, while the horse attempted to stand the nasotracheal tube was dislodged and kinked, and severe dyspnea reoccurred. This complication required immediate interventions; a third anesthetic was carried out to allow emergency tracheostomy in the recovery box. General anesthesia was induced with IV ketamine (2.5 mg kg^{-1}),

following xylazine (Chanazine, Chanelle, UK; 0.05 mg kg^{-1}, IV) premedication. Endoscopy-guided orotracheal intubation was accomplished with a 22 mm ET tube that was removed once the surgical procedure was completed and the tracheostomy tube was in place and secured. At this point, the horse was allowed to recover. Recovery from anesthesia was protracted and the horse remained in sternal recumbency for approximately two hours. In order to treat possible hypotension, suspected based on poor peripheral pulse, 2 L of hypertonic 7.2% NaCl and ephedrine (Ephedrine hydrochloride, Hameln Pharmaceutical, Germany; 0.05 mg kg^{-1}) were administered IV. At physical exam, rectal temperature was 35.5°C, pulse rate was 40 beats $minute^{-1}$ and respiratory rate was 10 breaths $minute^{-1}$. Hypothermia was treated with the use of blankets and infrared lamp heathers. Blood results from a sample collected during recovery revealed increased serum aspartate transaminase (676 U/L; reference interval: 198-476 U/L) and creatine kinase (15088 U/L; reference interval: 133-738 U/L). Based on these findings, a myopathy was suspected as further post-anesthetic complication. After three hours in the recovery box and five unsuccessful attempts to stand, an Anderson Sling was used to assist and maintain standing.

During the following 12 hours, the horse was closely monitored in the Intensive Care Unit of the hospital; support therapy consisted of IV fluids (Hartmann's solution, 2 mL kg hr^{-1}), analgesics (IV phenylbutazone, 4.4 mg kg^{-1} SID, and IM morphine, 0.1 mg kg^{-1} every four hours). Additionally, for the first two hours after standing oxygen was supplemented, via tracheostomy tube, at variable rate (2-4 L min^{-1}); thereafter, an arterial blood sample collected from the external carotid artery while the horse was breathing room air revealed normal blood gas results (arterial partial pressure of oxygen [PaO_2]: 98 mmHg, arterial oxygen saturation [SaO_2]: 97%; arterial partial pressure of carbon dioxide [$PaCO_2$]: 48 mmHg). Oxygen supplementation was discontinued; breathing remained unremarkable and the clinical condition of the horse was deemed good.

An endoscopic re-examination repeated 24 hours after the third anesthetic revealed normal abduction of the arytenoids. Thereafter, the tracheostomy tube was first blocked off to verify the laryngeal function, and then removed as dyspnea had not occurred. The surgical wound at the tracheostomy site was allowed to heal by second intention. Phenylbutazone administration was continued in the postoperative period for a total of five days.

The patient was discharged from the hospital 48 hours after recovery from the third anesthetic, and then reexamined after eight weeks. During this time, the horse owner did not notice any sign of respiratory dysfunction.

3. Discussion

This report describes the occurrence of acute, severe laryngeal dysfunction, confirmed by endoscopic findings, as a potentially life-threatening peri-anesthetic complication in a healthy horse undergoing anesthesia for an elective surgical procedure. The narrowing of the laryngeal *aditus* occurred without complete closure of the glottis; moreover, it was noticed that the arytenoids remained stationary in a neutral position throughout the respiratory cycle. These findings seem to suggest that, even if some degree of laryngospasm might have occurred, most likely the primary cause of the acute life-threatening dyspnea observed in this horse was rather laryngeal hypomotility or paralysis, a complication that has described before in horses recovering from anesthesia [4,5]. Since the laryngeal dysfunction resolved spontaneously within 24 hours , and considering that the horse had not suffered from respiratory disease either prior to anesthesia, or during the eight weeks after that, it would be reasonable to assume that the condition was the result of a mechanical trauma or stimulation to the arytenoids, possibly occurring during tracheal intubation/extubation. In support of this hypothesis, the laryngeal swelling detected on endoscopic examination would suggest a

recent traumatic injury. It is however unclear how laryngeal trauma occurred in the horse of this report. Orotracheal intubation was performed by an Anesthesia resident in-training, supervised by a recognized specialist in Veterinary Anesthesia. Furthermore, a relatively small endotracheal tube was used, which should have decreased the risk of iatrogenic mechanical injury. Although orotracheal placement of the ET tube was without major complications, mild resistance was encountered at first attempt, which required reinsertion of the ET tube after repositioning of the head and neck. It is hypothesized that repeated, although gentle, manipulation of the airways was sufficient to cause laryngeal trauma that might have affected the normal laryngeal function of this horse.

Laryngospasm seemed unlikely also based on the onset of the dyspnea. Laryngospasm is a transient dysfunction of the arytenoids, usually triggered by peri-glottic stimulation that activates the vagus nerve and characterized by prolonged contraction of the intrinsic laryngeal muscles, which results in partial or complete airways closure [15-17]. This condition has been reported in horses and cats following mechanical stimulation associated with tracheal intubation [16-18], and in humans as a postoperative complication during removal of the ET tube [19,20]. Literature suggests that, in human patients, post-anesthetic laryngospasm is more likely to occur in the presence of underlying neurological or upper respiratory diseases [19,21]. Extubation-induced laryngospasm is likely to cause signs of respiratory impairment shortly after extubation, whereas in the horse of this report the onset of the dyspnea was approximately 30 minutes after extubation.

Underlying neurological or respiratory conditions were not suspected prior to anesthesia based on history and clinical findings; nevertheless, it should be emphasized that idiopathic laryngeal hemiplegia is overrepresented in draft horses, in which a prevalence of 35% has been reported [22]. Factors predisposing draft horses to laryngeal dysfunction may be the long neck and large body size, potentially causing a greater tension on the left recurrent laryngeal nerve, which in turn might compromise the blood supply to the larynx and result in nerve damage [23]. Additionally, working with a flexed head and neck carriage may possibly cause decreased degree of arytenoid abduction and atrophy of the muscles surrounding the laryngeal saccules [24-26]. Although unlikely, the presence of an underlying, subclinical laryngeal disease that may have predisposed the horse to peri-anesthetic respiratory complications cannot be completely excluded.

The increase in serum creatine kinase, together with the observed muscular weakness and inability of the horse to stand, suggests that post-anesthetic myopathy may have occurred as further complication. An association between the post-anesthetic myopathy (PAM) and acute laryngeal dysfunction, although unlikely also based on previously published literature, cannot be completely ruled out [5].

Post-anesthetic myopathies represent a life-threatening complication more likely to occur in heavy and large horses, and after prolonged anesthetics resulting in prolonged compression of the *logissimus dorsi* and gluteal muscles, and less commonly, of the *adductor* group when the horse is positioned in dorsal recumbency [27]. The horse of this report was relatively large, and the total recumbency time, during which muscular compression could have occurred, was more than six hours. Because the muscular weakness resolved spontaneously and the clinical condition of the horse continued to improve during the days following surgery, it is believed that the PAM was not generalized but affected some isolated muscle groups. Another condition that was not possible to exclude was the Equine Polysaccharide Storage Myopathy (EPSM), an inheritable disease characterized by alterations of glycogen storage and possibly over-represented in the draft horse [28]. It has been reported that EPSM can cause 'PAM-like' symptoms similar to the ones observed in the horse of this report [29]. Ideally, EPSM screening tests and a complete ultrasonographic examination could have been useful to make a more precise diagnosis through identification

of the affected muscle [30]; nevertheless, further diagnostics was declined by the horse owner owing to restricted budget.

Reduced anesthetic time and correct positioning on the surgical table are important factors to minimize the risk of morbidities associated with equine anesthesia [31]. Previous reports suggest that there might be an association between postoperative upper airways obstructions and either dorsal recumbency [4,6], or extension of the neck [5,6], or both, during anesthesia. Abrahamsen and colleagues [4] hypothesized that extension of the neck during anesthesia might alter the blood supply to the local anatomical structures, potentially leading to nerve damage. The horse of this report, although it had the head slightly elevated to avoid over-extension of the neck during anesthesia, was positioned in dorsal recumbency for several hours. The reasons for such long duration of anesthesia and surgery were the involvement of residents-in-training, which is common in teaching Institutions, and the fact that an emergency colic surgery had to be initiated while the horse of the report was already being anaesthetized – an inconvenience that decreased the availability of technicians and other non-medical personnel in the operating theatre. Whenever possible, optimization of time and resources is strongly advisable in order to decrease the anesthetic time and therefore the risk of anesthesia-related complications in equine patients.

Prompt recognition of the complication, rapid interventions and teamwork were essential for the good outcome of this case. One critical moment was the induction of the second anesthetic to allow emergency nasotracheal intubation, which required entrance into the recovery box while the horse was very excitable and struggled to breath. The rationale for placing a nasotracheal tube at that time was because it was believed that, once conscious, the horse would have tolerated a nasotracheal tube better than an orotracheal one, which would have ensured maintenance of the airways' patency during recovery. However, the presence of the tube did not allow a proper endoscopic evaluation of the larynx prior to extubation, a diagnostic test which, with hindsight, would have been useful in this case. It is therefore sensible to state that performing an emergency tracheostomy immediately, once a patent airway had been established, would have been a safer approach to manage this case.

4. Conclusions

In conclusion, this report suggests that draft horses might require additional care during the perioperative period. The optimization of the anesthetic management and surgical time are essential in horses that for size and body weight may be prone to develop PAM or other anesthesia-related complications.

Additionally, ensuring prompt availability of a portable endoscope as well as of a surgical kit to perform emergency tracheostomy are essential for successful treatment of upper airway dysfunction during the peri-anesthetic period.

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Highlights

- Transient laryngeal dysfunction is a life-threatening peri-anesthetic complication
- Intubation and extubation may cause traumatic laryngeal dysfunction
- Draft horses might require additional care in the perioperative period

Author contributions for the manuscript entitled 'Acute life-threatening laryngeal dysfunction in a draft horse recovering from general anesthesia: a case report'

Hayley Linda Ronaldson: Data Curation, Writing-Original Draft

Paolo Monticelli: Writing-Review & Editing, Visualization

Roger Smith: Writing-Review & Editing

Chiara Adami: Data Curation, Writing-Original Draft & Editing, Supervision

Ethical statement:

The horse object of this case report received high standard care and all the procedures carried out -including monitoring and blood sampling- are part of the routine standard care of the horse undergoing anesthesia. Data were collected from the clinical case and used retrospectively for publication.

Conflict of interest statement:

The authors declare no conflicts of interest associated with the research and preparation of this manuscript.