Persistent hypoxaemia and pulmonary oedema in a horse anaesthetised for emergency exploratory laparotomy

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CASE REPORT

Horses and other equids

Persistent hypoxaemia and pulmonary oedema in a horse anaesthetised for emergency exploratory laparotomy

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Abstract

A 16-year-old, 550 kg Connemara gelding was anaesthetised for resection of multiple small intestine strangulating lipomas via ventral midline celiotomy. Severe hypoxaemia, detected throughout the anaesthetic period (lowest PaO_2 58 mmHg [7.6 kPa]), was unresponsive to the ventilatory strategies implemented. Flared nostrils and increased respiratory rate were present at recovery from anaesthesia. When the horse was returned to the yard, bilateral foamy nasal discharge and increased respiratory effort and rate were noticed, consistent with pulmonary oedema. The horse received oxygen supplementation and furosemide, which led to complete resolution within 24 hours.

KEYWORDS

horses, emergency exploratory laparotomy, hypoxaemia, pulmonary oedema

BACKGROUND

Development of hypoxaemia in horses anaesthetised for emergency exploratory laparotomy is a well-recognised complication, primarily resulting from compression atelectasis,[1,2] with a variable reported incidence of 13%,[3] 23%[4] and 15.3%.[5] Several risk factors have been identified, such as dorsal recumbency[6–8]; distended intestines, with the likelihood of hypoxaemia being three times greater in horses suffering from lesions of the large intestine[4,5]; peak inspiratory pressures (PIP) greater than 30 cmH_2O[4]; low pulse pressure and emergency procedures[9]; and bodyweight more than 550 kg.[4,5]

Pulmonary oedema is a relatively infrequent but potentially serious event in the perianaesthetic period in horses.[10,11] It has been observed more commonly in the post-operative period in association with upper airway obstruction (UAO),[11–13] although the cause is often multifactorial.[10,11] Other suggested predisposing factors are air embolism due to dislodgement of intravenous catheter,[14] microembolism associated with orthopaedic surgery,[15] endotoxaemia, anaphylaxis, and underlying cardiac disease,[16] alveolar volutrauma resulting from high inflation pressures during mechanical ventilation,[17] and secondary to administration of drugs such as morphine[11] and xylazine-carfentanil.[18]

The aim of this case report is to describe a case of persistent hypoxaemia in a horse anaesthetised for emergency exploratory laparotomy, and discuss its association with the pulmonary oedema observed post-operatively.

CASE PRESENTATION

A 16-year-old, 550 kg Connemara gelding was referred for investigation and treatment of recurrent signs of colic.

On presentation, heart rate (HR) was 60 beats per minute, respiratory rate (RR) was 20 breaths per minute, auscultation of the thorax was unremarkable, rectal temperature was 37.8°C, mucous membranes were pink and dry, intestinal borborygmi were absent in all quadrants. Packed cell volume (PCV) was 45%, serum total protein concentration 65 g/L and serum lactate concentration 2.4 mmol/L. Total protein and lactate concentrations in the free peritoneal fluid were 6 g/L and 0.8 mmol/L, respectively. Approximately 2 L of reflux was obtained via a nasogastric tube. Mural thickening of the small intestine, abnormal contractions of the duodenum, distended small intestinal loops and a secondary soft pelvic impaction were detected at abdominal ultrasonographic and transrectal examinations. As these findings were consistent with a strangulating small intestinal lesion, a decision was made to proceed with surgical exploration. A polyurethane 14 G catheter (Milacath-extended use, Mila International) was inserted in the left jugular vein. Antimicrobial therapy was provided with procaine penicillin (22 mg/kg intramuscularly [IM]; Depocillin 300 mg/mL, MSD Animal Health UK) and gentamicin (6.6 mg/kg intravenously [IV]; Genta-Equine 100 mg/mL, Dechra Veterinary Products, UK). Flunixin meglumine (1.1 mg/kg IV; Meflosyl 5%, Zoetis UK) was also administered.

The horse was premedicated with xylazine (0.7 mg/kg IV; Chanazine 10%, Chanelle Pharmaceuticals Manufacturing), followed by morphine (0.3 mg/kg IV; Morphine Sulfate 30 mg/mL, Martindale Pharma, UK), given over 2 minutes. Sedation was deemed not adequate for induction, and additional 0.3 mg/kg IV of xylazine (Chanazine 10%, Chanelle Pharmaceuticals Manufacturing) was administered. Five minutes later, general anaesthesia was induced with ketamine (2.5 mg/kg IV; Ketamidor 100 mg/mL, Chanelle Pharmaceuticals Manufacturing) combined with midazolam (0.06 mg/kg IV; Dormazolam 5 mg/mL, Dechra Veterinary Products, UK), which provided a smooth transition to sternal then lateral recumbency, and the trachea was intubated with a 26-mm ID endotracheal tube (ETT) (Kruuse UK). After inflation of the ETT cuff, the horse was hoisted onto the surgical table in dorsal recumbency and connected to a large animal anaesthetic machine (Tafonius, Vetronic Services) via a circle breathing system, and mechanical ventilation started immediately. Initial settings were tidal volume (T_v) of 12 mL/kg, RR of 6, inspiratory to expiratory ratio (*I*:*E*) of 1:3 with an inspiratory time of 2.2 seconds; PIP was 32 cmH_2O. Anaesthesia was maintained with sevoflurane (Sevotek 1000 mg/g, Animalcare, UK) (end-tidal concentration 1.8%–2.4%) in oxygen (fraction inspired of oxygen [FiO_2] 0.79–0.95). A polytetrafluoroethylene 20G catheter (Biovalve safe, Vygon UK) was placed in the left transverse facial artery for arterial blood gas analyses (ABGs) and continuous monitoring of invasive arterial pressure after zeroing the transducer at the level of the manubrium sterni. Other intraoperative monitoring (Datex-Ohmeda S5 or Tafonius, Vetronic Services) consisted of pulse oximetry (SpO_2), end-tidal anaesthetic agent, end-tidal carbon dioxide concentration, electrocardiography, RR, PIP, positive-end expiratory pressure (PEEP), *I*:*E*, T_v and FiO_2. All variables were manually recorded every 5 minutes. Intravenous fluid therapy was provided with Hartmann's solution (9 mL/kg/h; Aqupharm 11, Animalcare, UK), with this rate achieved through insertion of a polyurethane 13G catheter (Intranule, Vygon UK) in the left jugular vein distal to the first catheter. A lidocaine (Lidocaine Hydrochloride injection 2%, Hameln Pharma) loading dose of 1.5 mg/kg was administered over 20 minutes, followed by an infusion at 50 μg/kg/min for the duration of the procedure, and discontinued 40 minutes before the end of anaesthesia. Dobutamine (Dobutamine 12.5 mg/mL, Hameln Pharma) was infused at 0.25 μg/kg/min and discontinued after 3 hours of anaesthesia as mean arterial pressure (MAP) was consistently above 90 mmHg (11.8 kPa).

The first ABG revealed severe hypoxaemia with mild hypercapnia (Table 1, sample 39 minutes). T_v was increased to 13 mL/kg, inspiratory time to 2.5 seconds maintaining an *I*:*E* of 1:3, and 10 cmH_2O PEEP was added. MAP gradually decreased from 85 to 70 mmHg (11.2 to 9.2 kPa), and a bolus of 4 mL/kg of 7.2% hypertonic saline (Hypertonic 72 mg/mL, Dechra Veterinary Products, UK) was administered over 20 minutes, which improved MAP to 80 mmHg (10.5 kPa). The second ABG showed no improvement in PaO_2 (Table 1, sample 74 minutes). An alveolar recruitment manoeuvre (ARM) was initiated, with an increase in T_v to 15 mL/kg and stepwise incremental (to 15, then 20 cmH_2O) and decremental (to 15, then 12 cmH_2O) PEEP, with each step implemented for 2 minutes. Maximal PIP recorded during the ARM was 40 cmH_2O.

LEARNING POINTS/TAKE-HOME MESSAGES

- Hypoxaemia is frequently observed in dorsally recumbent horses anaesthetised for emergency exploratory laparotomy.
- Development of pulmonary oedema, an uncommon complication reported in the perioperative period in horses, might be both responsible for and resulting from severe hypoxaemia.
- It can be difficult to determine the exact causative factor/s of perioperative pulmonary oedema, although it is likely to be multifactorial.
- Careful patient monitoring and attention to possible alerting signs may lead to prompt recognition and treatment, thus improving patient outcome.

At the end of the ARM, salbutamol (2 μg/kg; Ventolin Evohaler 100 micrograms, GlaxoSmithKline UK) was aerosolised in the inspiratory limb of the Y-piece during inspiration over five breaths. The following ABG (Table 1, sample 97 minutes) showed a positive response, with a PaO_2 of 90 mmHg (11.8 kPa). PEEP and T_v were maintained at 12 cmH_2O and 15 mL/kg, respectively. However, at the time of the next ABG, PaO_2 had decreased to 73 mmHg (9.6 kPa) (Table 1, sample 129 minutes); administration of salbutamol (Ventolin Evohaler 100 micrograms, GlaxoSmithKline UK) was repeated, PEEP was increased to 15 cmH_2O and T_v was increased to 16 mL/kg, but no improvement was observed (Table 1, sample 157 minutes). A second ARM was implemented in a similar fashion to the previous one, followed by administration of salbutamol (Ventolin Evohaler 100 micrograms, GlaxoSmithKline UK), although T_v and starting–final PEEP were higher (16 mL/kg and 15 cmH_2O, respectively), which led to a higher maximal PIP achieved (50 cmH_2O). The horse remained nonetheless hypoxaemic with a PaO_2 of 63 mmHg (8.3 kPa) (Table 1, sample 180 minutes).

During the exploratory laparotomy, three pedunculated lipomas of the distal jejunum (one of which strangulating the associated small intestine) were removed and an end-to-end jejunojejunostomy was performed.

Hypoxaemia was still present at the last ABG analysed during closure of the abdominal wall (Table 1, sample 219 minutes). Five minutes before completion of surgery, the arterial catheter and the second jugular catheter were removed. At completion of surgery (total surgical time 205 minutes), sevoflurane was discontinued (total anaesthesia time 230 minutes) without transition to spontaneous ventilation, as a Hudson de mand valve is available at our institution. The horse was moved into a padded recovery box and attached to head and tail ropes, and 10 mL of 0.1% phenylephrine (Phenylephrine hydrochloride eye drops 2.5% w/v, Bausch & Lomb UK) was nebulised in the nasal cavities, 5 mL each side. As spontaneous ventilation was already regained, xylazine (0.25 mg/kg; Chanazine 10%, Chanelle Pharmaceuticals Manufacturing) was administered IV, the trachea was extubated and oxygen was supplemented through a nasal tube at 10 L/min. No signs of reflux were noticed.

TABLE 1 Fractional inspired oxygen and arterial blood gas values in a horse anaesthetised for emergency exploratory laparotomy.

Variable (reference range) [minutes]	39[a]	74	97	129	157	180	219
FiO_2	0.86	0.79	0.92	0.94	0.86	0.85	0.90
PCV (37%–42%)	37	36	35	32	32	32	29
pH (7.38–7.44)	7.30	7.33	7.32	7.30	7.31	7.33	7.37
$PaCO_2$ (36.3–54) mmHg [kPa]	56 [7.3]	49 [6.4]	48 [6.3]	52 [6.8]	51 [6.7]	50 [6.5]	44 [5.8]
PaO_2 (82.6–112.3) mmHg [kPa]	59 [7.7]	58 [7.6]	90 [11.8]	73 [9.6]	65 [8.5]	63 [8.3]	66 [8.7]
BE (−0.51 + 8.8 mmol/L)	−0.5	−1.1	−2.3	−1.9	−1.5	−0.8	−0.3
SpO_2 (95%–100%)	87	86	95	91	88	87	89
HCO_3^- (23.87–32.45 mmol/L)	26.8	25.3	24.1	24.8	25	25.5	25.1

Abbreviations: BE, base excess; FiO_2, fractional inspired oxygen; PCV, packed cell volume; SpO_2, pulse oximetry.
[a]Times indicate minutes from induction of anaesthesia.

A transient moderate inspiratory stridor after tracheal extubation was present, although sound air flow was detected from both nostrils and thoracic expansion was normal in inspiration. The horse attempted to stand after 10 minutes, and a second dose of xylazine (0.2 mg/kg; Chanazine 10%, Chanelle Pharmaceuticals Manufacturing) was administered IV. Standing position was achieved 30 minutes later without complications, although flared nostrils and an increased RR were noted. When returned to the yard, the horse developed a bilateral foamy nasal discharge with increased RR and effort, consistent with pulmonary oedema.

TREATMENT

The horse was moved to the intensive care unit (ICU) and was started on 10 L/min oxygen via nasal tube. Administration of furosemide (1 mg/kg IV; Dimazon 50 mg/mL, MSD Animal Health UK) rapidly led to discontinuation of the foamy nasal discharge. Supportive therapy consisted of a 1.5-L bolus of 6% hydroxyethyl starch (Hydroxy Ethyl Starch 6% [130/0.4], Fresenius Kabi AG Germany), followed by Hartmann's (2 mL/kg/h; Aqupharm 11, Animalcare, UK). HR was 80 beats per minute and serum lactate 6.4 mmol/L, which decreased to 60 beats per minute and 0.8 mmol/L, respectively, the morning after surgery. Nasogastric intubation failed to yield any reflux. An ABG revealed adequate oxygenation, and intranasal oxygen was discontinued. A final dose of furosemide (1 mg/kg IV; Dimazon 50 mg/mL, MSD Animal Health UK) was administered. Ongoing antimicrobial therapy consisted of procaine penicillin (22 mg/kg IM twice a day [BID]; Depocillin 300 mg/mL, MSD Animal Health UK) and gentamicin (6.6 mg/kg IV once a day; Genta-Equine 100 mg/mL, Dechra Veterinary Products, UK); flunixin meglumine (Meflosyl 5%, Zoetis UK) was continued (at 1.1 mg/kg IV BID).

OUTCOME AND FOLLOW-UP

Clinical signs of pulmonary oedema resolved within 24 hours, and no further complications developed. Owners confirmed no previous history of lower respiratory tract disease and/or associated clinical signs. The patient was discharged from the hospital 9 days after the surgical procedure.

DISCUSSION

We present a case of persistent hypoxaemia in a horse anaesthetised for emergency exploratory laparotomy complicated by the occurrence of pulmonary oedema detected at recovery. This latter complication may either represent a primary aggravating factor for the intraoperative hypoxaemia from the early stages of anaesthesia or may be secondary to the perioperative anaesthetic management.

Hypoxaemia is defined as an arterial partial pressure of oxygen less than 80 mmHg (10.5 kPa)[3,9] and less than 60 mmHg in severe cases (7.9 kPa).[6] Formation of extensive areas of atelectasis in the dependent lung regions is responsible for a significant right-to-left vascular pulmonary shunt associated with a marked inequality between distribution of perfusion and ventilation,[1,7] which is exacerbated in horses undergoing emergency exploratory laparotomy.[9,19,20] Although correction of hypoxaemia in anaesthetised horses remains a challenge,[2] multiple interventions have been suggested to maximise oxygenation.

Numerous studies have demonstrated that IPPV is more effective in minimising formation of atelectasis when started immediately after induction of anaesthesia,[8,19] and this approach, adopted in the present case, is supported by multiple studies in the equine literature.[21–23] However, high values of PIP in patients with atelectasis have been associated with an increased risk of hypoxaemia,[5] presumably related to volutrauma[11] due to overexpansion of a reduced number of open alveoli. As the initial PIP in this case was already greater than 30 mmHg (>3.9 kPa), the first attempt to improve oxygenation and respiratory mechanics was conservative and consisted of an increase in T_v and inspiratory time[22,24–26] and by the addition of PEEP.

PEEP increases functional residual capacity (FRC) over the critical closing volume, hence helping prevent alveolar collapse at the end of expiration.[23,27] While application of PEEP without an ARM did not prove effective in improving gas exchange in spontaneously breathing anaesthetised horses,[28] it was demonstrated to change distribution of ventilation and increase PaO_2 and FRC when mechanical ventilation was used.[29–31] Nonetheless, high levels of PEEP are associated with high intrathoracic pressures, which may decrease the ventilation/perfusion ratio by forcing blood into already atelectatic lung areas, leading to a higher degree of right-to-left shunt and decreased oxygenation.[27] Therefore, we set our initial

PEEP at 10 cmH_2O. However, this strategy did not lead to any improvement.

An alternative ventilatory strategy, known as 'open-lung concept', involves the application of IPPV with stepwise increases in inspiratory pressures to recruit collapsed alveoli (ARM), used in combination with PEEP titration to prevent re-collapse at the end of expiration.[32] This technique has been successfully applied by multiple authors in anaesthetised ponies and horses.[33–36] Implementation of an ARM with stepwise incremental and decremental PEEP, followed by aerosolisation of salbutamol, initially raised PaO_2 to 90 mmHg (11.8 kPa) in our case. Other authors[35] implemented each incremental and decremental step for 15 minutes, while this was done for 2 minutes in this case. It is therefore possible that a longer duration of each step could have recruited a higher number of alveolar units leading to better oxygenation. Moreover, the effects of the ARM were short-lasting, as also observed in a previous study conducted by Hopster et al.[20] In this study, ARMs repeated at intervals as short as 20 minutes were applied to maintain PaO_2 greater than 400 mmHg (53.3 kPa). In the present case, a second ARM was repeated after approximately 1 hour and 15 minutes, with no improvement observed. It is reasonable to speculate that a better outcome could have been achieved if the second ARM had been repeated earlier, possibly preventing de-recruitment of the previously opened alveoli.

Salbutamol, administered after each ARM to maximise the effects of alveolar recruitment, yielded only a mild positive response after the first administration and no response after two additional doses. Salbutamol proved effective in increasing PaO_2 in previous studies,[37,38] although the exact mechanism is yet to be elucidated. The increase in PaO_2 after salbutamol administration might be the result of reductions in pulmonary vascular resistance, with consequent reduction in ventilation/perfusion inequality,[37] and increase in cardiac output and oxygen delivery, hence potentially exerting its effects by altering pulmonary perfusion rather than ventilation.[38] Adverse systemic effects after aerosolisation of salbutamol in anaesthetised horses have been reported, such as sinus and ventricular tachycardia,[39] and reduction in serum potassium concentrations,[40,41] none of which was observed in this case. Lack of improvement in PaO_2 has also been described,[39] as it seems to be the case in our horse.

The horse was noted to have flared nostrils and increased RR after standing, which progressed to increased respiratory effort and bilateral foamy nasal discharge shortly after. Based on these clinical signs, consistent with pulmonary oedema,[11,42,43] appropriate treatment was initiated.

It is difficult to determine when the pulmonary oedema developed in this case and what the most likely causative factors were, although a few considerations can be made.

Despite its development likely being multifactorial,[10,11,13,43] the majority of case reports ascribed pulmonary oedema to UAO at recovery.[12,42,44–47] The negative intrathoracic pressures generated during inspiration against an obstructed airway promote venous return, increasing the amount of blood in the pulmonary vasculature and afterload, consequently increasing the hydrostatic pressure.[48] This in turn favours the passage of fluid from the intravascular space to the interstitium first and finally to the alveoli. In the present case, a transient inspiratory stridor was noted soon after tracheal extubation, possibly associated with displacement of the soft palate, which is unlikely to cause complete obstruction.[10] Furthermore, thoracic expansion in inspiration was normal, intranasal phenylephrine was administered immediately upon arrival into the recovery box, and sound passage of air at the nostrils was detected bilaterally. Therefore, we consider unlikely for UAO, in the absence of evident clinical signs, to be responsible for the pulmonary oedema in this case.

Another predisposing factor is fluid overload, which can promote fluid shift into the interstitial and/or alveolar compartment due to increased hydrostatic pressure.[11] Recommended rates of isotonic fluids in horses under general anaesthesia sit between 3 and 10 mL/kg/h, depending on clinical judgement.[49] Pre-operative PCV was 45%, thus indicating some degree of haemoconcentration (see Table 1 for reference ranges), and mucous membranes were dry at clinical examination. Hence, it is unlikely that the 9 mL/kg/h rate of crystalloids used intraoperatively led to fluid overload. Furthermore, as part of the supportive therapy in ICU, the horse received a bolus of 6% hydroxyethyl starch, which would have likely worsened clinical signs of pulmonary oedema if fluid overload had already been present.

Drug involvement has been suspected as a possible contributing factor in horses in association with morphine, potentially due to histamine release,[11] and with carfentanil co-administered with xylazine.[18] In this case, no signs of an allergic or non-allergic anaphylactic reaction were evident after administration of morphine, while xylazine might be implicated in the genesis of pulmonary oedema, which may have contributed to the unresponsiveness of hypoxaemia to treatment. Xylazine is known to cause pulmonary oedema in sheep,[50] albeit the pathophysiologic mechanism is not fully elucidated.[10] Intraoperatively, clinical signs might have been masked by IPPV and maximal FiO_2,[51] while discontinuation of supportive therapy and administration of two additional doses at recovery may have precipitated clinical signs. Despite no nasal discharge and only a mild amount of clear fluid in the ETT were observed during anaesthesia, thoracic auscultation was not performed, which represents a limitation.

Alveolar hypoxia and sudden re-expansion of atelectatic lung areas were suspected to be determinant aetiological factors in previous cases described in horses.[12,52] Hypoxaemia resulting from atelectasis may lead to periods of ischaemia and hypoxia, followed by possible atelectrauma[43] and reperfusion injury[13] on lung re-inflation. Additionally, hypoxia per se determines pulmonary vasoconstriction, which increases pulmonary capillary hydrostatic pressure,[13] although the role of hypoxia in exacerbating pulmonary oedema remains controversial.[10] Hypoxia may have occurred in this case, as hypoxaemia was a major complication encountered. Secondary volutrauma and/or barotrauma and reperfusion injury may have also occurred during the ARMs applied. However, hypoxaemia and atelectasis are frequently observed in anaesthetised horses, and ARMs are commonly applied, while pulmonary oedema is a rare event.

Primary ventilator-associated lung injury (VALI) is another possibility in the case reported here. Levels of PIP above 40 cmH_2O have been associated with potential harm to a healthy horse lung,[43] and PIP recorded in this case varied between 32 and 50 cmH_2O, thus possibly leading to volutraumatic damage of the alveolar cell membrane. Nonetheless, some authors utilised PIP up to 55 mmHg (7.2 kPa)[34] and 80 mmHg (10.5 kPa)[20] during ARMs without adverse effects.

Finally, it is worth considering that our initial T_v was 12 mL/kg, and this generated a PIP of 32 cmH_2O, which may be suggestive of reduced chest/pulmonary compliance. Many factors might be associated with reduced compliance in anaesthetised horses, such as dorsal recumbency[43] and distended intestines.[20] However, it is also possible that the suspected decreased compliance was a consequence of pulmonary oedema,[43] which would imply this complication was present from the onset of anaesthesia.

In conclusion, although correction of hypoxaemia might have required a more aggressive treatment, we suspect it was aggravated by the development of pulmonary oedema, which was potentially multifactorial encompassing drug involvement, hypoxia and secondary atelectrauma, and primary VALI. Careful patient monitoring and attention to possible alerting signs in horses exposed to one or more of these predisposing factors may lead to prompt recognition and treatment, thus improving outcome.

AUTHOR CONTRIBUTIONS

Barbara Testa and Robert Ward: identification of the project and data acquisition. Barbara Testa: project design, data interpretation, writing of the first draft, and reviewing and editing of the manuscript. Robert Ward and Gudrun Schoeffmann: reviewing and editing of the manuscript. All authors reviewed and agreed to the submitted version of the manuscript.

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CONFLICT OF INTEREST STATEMENT

The authors declare they have no conflicts of interest.

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ETHICS STATEMENT

This manuscript describes a clinical case treated following the standards in place at our institution and according to the primary anaesthetist's (Barbara Testa) clinical judgement. Informed owner consent, including consent for the veterinary treatment of this horse and for the retention of data and anonymous use in retrospective publications, was obtained.

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