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CASE REPORT

Horses and other equids

Spontaneous pulmonary haemorrhage in a standing sedated horse

Abraham Calero Rodriguez | **Janny de Grauw**

Department of Equine Sciences, Faculty of Veterinary Medicine, University of Utrecht, Utrecht, The Netherlands

Correspondence
Abraham Calero Rodriguez, University of Utrecht, Yalelaan 112, 3584 CM Utrecht, The Netherlands.
Email: a.n.calerorodriguez@uu.nl

Abstract

A 615 kg, 20-year-old, male, neutered Dutch warmblood horse was admitted for complete removal of incisor teeth under sedation. The horse was sedated with intramuscular acepromazine (0.1 mg/kg) and several intravenous detomidine boli (total dose of 0.008 mg/kg), followed by detomidine constant rate of infusion. At the beginning of the surgery (47 minutes after starting sedation), the horse retched and coughed out haemorrhagic fluid. Surgery was discontinued and profuse bleeding started from the nostrils. Mild hypoxaemia (PaO_2 = 63.4 mmHg) was observed. Oxygen therapy by nasal canula (10 L/min) was started, detomidine constant rate of infusion stopped and antagonised with atipamezole given intravenously to effect (total dose of 0.14 mg/kg). The horse also received tranexamic acid. Pulmonary haemorrhage was suspected after tracheal-bronchoscopic examination. Fresh whole-blood transfusion was initiated (estimated blood loss: 4 L). Bleeding reduced gradually during the following 2 hours and the horse was monitored in the intensive care unit, with supplemented nasal oxygen. During the following 2 days, oxygen administration was gradually reduced and finally discontinued. The nasal bleeding resumed 8 days after the initial episode, but the horse was euthanased due to the uncertainty of the prognosis.

KEYWORDS
EORTH, equine, pulmonary haemorrhage

BACKGROUND

Equine odontoclastic tooth resorption and hypercementosis (EOTRH) is a progressive and painful disease of uncertain aetiology, common in horses older than 15 years. There is currently no treatment for this condition. As the condition will progressively worsen over time, extraction of the affected teeth under sedation or general anaesthesia is a frequently chosen option to improve the quality of life of the patients in the short and medium time period.[1]

In the present case report, an uncommon complication during teeth extraction in a standing sedated horse is described, as well as its treatment and outcome. The treatment is provided in steps with a logical approach, making the case useful for other veterinarians facing the same complication.

CASE PRESENTATION

A 615 kg, 20-year-old, male, neutered Dutch warmblood horse was admitted to the clinic for removal of the incisor teeth of both the upper and lower jaw during standing sedation, due to a previous diagnosis of EOTRH. During the preanaesthetic examination, a diastolic heart murmur (grade 2 out 6) was observed. The pulse was strong and regular and had a pulse rate (PR) at rest of 36 beats per minute. The owners were informed about the murmur but declined further workup. The rest of the preanaesthetic examination was unremarkable.

In the stable, the patient received 0.1 mg/kg acepromazine (Neurotranq 10 mg/ml, Alfasan, the Netherlands) intramuscularly (IM). Thirty minutes later, 0.005 mg/kg detomidine (Domosedan 10 mg/ml, Orion, Finland) was given intravenously (IV) and the animal was moved to the preparation room. A 12G catheter (Intraflon 2, Vygon, France) was placed in the left jugular vein and 0.1 mg/kg morphine (Morphine HCl 10 mg/ml, Centrafarma, the Netherlands) and 0.6 mg/kg meloxicam (Metacam 20 mg/ml, Boehringer-Ingelhein, Germany) were administered IV. Also, 20 million IU benzyl penicillin (Benzylpenicilline 10 million IU, Dechra, UK) and 6.6 mg/kg gentamycin (Gentamycin 5%, Dechra, UK) were given IV as preoperative antimicrobial therapy.

To maintain an adequate level of sedation, a top-up of 0.003 mg/kg detomidine IV and a constant rate of infusion (CRI) of detomidine was started at a rate of 0.01 mg/kg/h to maintain the level of sedation, and IV fluid therapy (Ringer Fresenius, Fresenius Kabi, Germany) was started at a rate of 10 ml/kg/h. Electrocardiogram (ECG) was evaluated with a multiparameter monitor (Datex-Ohmeda S/5, GE Healthcare, USA). After the start of the detomidine CRI, local anaesthesia was performed. This consisted of a bilateral infraorbital nerve block with 3 ml of levobupivacaine (Chirocaine 0.25%, AbbVie, USA) per side, and left mental nerve block with

3 ml levobupivacaine, performed in the first 30 minutes after starting the detomidine CRI. A right mental nerve block was also attempted, but the attempt failed due to the animal resenting the placement of the needle. Before attempting an alternative local anaesthetic approach, we decided to see if the animal would tolerate the surgical manipulation on the upper quadrants first, and surgery started. During placement of the local blocks, the horse had an adequate level of sedation (separated ear tips, no response to environment, semi-closed eyes and leaning on head support) with a respiratory rate (f_R) of 16 breaths per minute and PR in a range of 28–32 beats per minute. A second-degree atrioventricular block every six or seven normal QRS complexes was noticed in the ECG.

Five minutes after starting the surgical procedure, the level of sedation was deemed inadequate for the performance of the surgery; therefore, an extra bolus of detomidine of 0.003 mg/kg was administered IV. Twelve minutes after starting the surgery, the horse retched and immediately after that the patient coughed out haemorrhagic fluid. The surgery was stopped, and profuse bleeding started from the nostrils. Due to the origin of the bleeding (nostrils as opposed to the oral cavity) and as the patient was not swallowing, the lower airways were identified as the most likely source of the bleeding. The detomidine CRI and fluids were stopped and the head was put in a lower position. The horse coughed frequently (approximately around twice per minute) expectorating blood but also some whitish, putrid smelling tissue. In between the coughs, the profuse bleeding continued. The f_R of the patient increased up to 25 breaths per minute, and it was breathing with wide nostrils. The PR stayed around 32–34 beats per minute. During thoracic auscultation, bubbling lung sounds were noticed on the right side.

A presumptive diagnosis of pulmonary haemorrhage was made, and oxygen therapy was started through a nasal line at a rate of 10 L/min. Subsequently, a tracheo-bronchoscopy was performed. This revealed a large flow of blood covering the ventral aspect of the trachea, originating mainly from the right lower airways.

Approximately 20 minutes after the start of the bleeding, tranexamic acid (20 mg/kg IV) (Cyklokapron 100 mg/ml, Pfizer, USA) was administered, and the detomidine was antagonised with two boluses of atipamezole (Atipam 5 mg/ml, Dechra, UK) (0.06 and 0.08 mg/kg IV, respectively). Thirty minutes after the beginning of the bleeding and coughing, the first arterial blood sample was analysed (Table 1). Mild hypoxaemia was noticed, with an arterial partial pressure of oxygen (PaO_2) of 63.4 mmHg and a haemoglobin saturation (SaO_2) of 93.5%.

LEARNING POINTS/TAKE HOME MESSAGES

- Sedation with detomidine boli can cause pulmonary bleeding, probably as a consequence of pulmonary hypertension, despite administration of acepromazine.
- Blood transfusion should be considered in the event of severe blood loss when oxygen-carrying capacity or circulating volume is compromised.
- Nasal oxygen supplementation is an option when hypoxaemia is presented in horses.
- Tranexamic acid is a therapeutical option in cases of bleeding in horses.

Due to the abundant blood loss experienced by the horse in a short period (estimated in around 4 L), blood transfusion was started 40 minutes after the episode started. The animal was still coughing blood, with a frequency decreased to once every 15 minutes. The amount of blood coming from the nostrils was also reduced, from profuse bleeding to a rate of approximately three drops of blood per second. Heart and respiratory rates remained unchanged (f_R 25 breaths per minute, PR 34 beats per minute), and the ECG was regular. Rectal temperature was 36.4 degrees Celsius (°C).

One hour after the beginning of the nasal bleeding, a 20G arterial catheter was placed in the right facial artery. A new arterial blood sample was analysed, showing a slight improvement in PaO_2 and SaO_2 (Table 1) and a haematocrit of 0.24 L/L. Arterial blood pressures varied in the ranges 112–126, 85–90 and 67–71 mmHg for systolic, mean and diastolic arterial blood pressures, respectively (SAP, MAP and DAP, respectively). Thoracic ultrasound was performed, showing a normal pleural line on both sides, no free fluid and no substantial significant consolidation of pulmonary tissue.

Due to the increased lactate in the arterial blood gas analysis, fluid therapy was restarted through a second 12G catheter placed in the right jugular vein at a rate of 10 ml/kg/h. At that time, the first 3 L bag of fresh whole blood was transfused, and a second bag was started. Ninety minutes after the onset of the bleeding, a third arterial blood sample was analysed showing a further improvement of PaO_2 and SaO_2. At that time, the

TABLE 1 Arterial blood gas analysis during and after the episode of nasal bleeding

Time (after beginning of the nasal bleeding)	30 Minutes	60 Minutes	90 Minutes	4 Hours	24 Hours
pH	7.48	7.43	7.44	7.44	7.40
$PaCO_2$ (mmHg)	41.6	45.7	47.3	44.3	37
PaO_2 (mmHg)	63.4	68.8	71.4	120.5	107.6
Base excess (mmol/L)	6.1	4.8	6.3	4.2	−1.7
SaO_2 (%)	93.5	94.2	94.8	98.5	98
Lactate (mmol/L)	1.37	1.54	1.72	1.3	0.8
Oxygen by nasal canula (L/min)	10	10	10	6	4

Abbreviations: $PaCO_2$, arterial partial pressure of carbon dioxide; PaO_2, arterial partial pressure of oxygen; SaO_2, arterial saturation of oxygen.

values of blood pressure were 114, 79 and 62 mmHg for SAP, MAP and DAP, respectively, and although there was no change in PR (32 beats per minute) and f_R (24 breaths per minute), the frequency of coughing up blood had decreased to two times per half hour.

Two hours after the start of the bleeding episode, the respiratory pattern had improved, and the breathing rate decreased to 20 breaths per minute. Due to the clinical improvement of the horse, discontinuation of the nasal oxygen was attempted, and the response of the horse was evaluated. Tachycardia (60 beats per minute) and tachypnoea (32 breaths per minute) were noticed after approximately 2 minutes, and values normalised once the oxygen was restarted.

Once the blood transfusion was finished (total of 6 L of fresh whole blood given in 2 hours, 9.7 ml/kg), the patient was transferred to a stable in the intensive care unit (2 hours and 10 minutes after the start of the bleeding episode), where oxygen therapy by nasal line was continued at a rate of 6 L/min. By that time, the bleeding through the nostrils had stopped. Two hours later (4 hours from the beginning of the episode), arterial blood gas analysis was repeated, showing a clear improvement in PaO_2 (120 mmHg) and SaO_2 (98.5%).

The following day (Day 1 after bleeding episode), the horse presented with a respiratory rate of 12 breaths per minute and heart rate of 48 beats per minute. Temperature was 38°C. Arterial blood gas was analysed with values similar to the last arterial blood gas of the previous day (Table 1). Haematocrit was 0.32 L/L. The horse was still considered oxygen dependant as discontinuation of oxygen supplementation resulted in an increase in both respiratory and heart rates. Oxygen was supplemented at 4 L/min. Antimicrobial therapy was initiated with 6.6 mg/kg IV gentamycin once a day (SID) (Gentamycin 5%, Dechra, UK), 20 mg/kg IM procaine penicillin SID (Procapen 300 mg/ml, ASTfarma, Germany) and 25 mg/kg metronidazole (375 mg/ml, magistral formula), twice daily, orally (PO). Meloxicam (Metacam 15 mg/ml, Boehringer-Ingelhein, Germany) treatment was continued at 0.6 mg/kg PO SID. Thoracic radiographic images were compatible with pulmonary haemorrhage.

Samples of the blood and foul-smelling tissue expectorated by the horse were sent to pathology and for bacterial culture. Pathology examination of the samples detected plant material, with fibrin.

The following day (Day 2 after bleeding episode), nasal oxygen insufflation was discontinued, and the animal did not present abnormalities on physical examination. The horse was eating normally and treatment was continued during the following 5 days. During this period, the horse did not show any clinical abnormalities.

OUTCOME AND FOLLOW-UP

Eight days after the bleeding episode, the patient showed blood loss again from the nostrils; therefore, a relapse of the pulmonary haemorrhage was suspected. After discussion with the owners and due to the uncertainty of the prognosis, euthanasia was chosen. A postmortem examination was performed. The report of the postmortem examination concluded that the horse had signs of polyphasic haemorrhage within the lungs. The main haemorrhagic lesion was encapsulated, suggesting a chronic process; however, histologically there were also lesions of a more acute nature suggesting ongoing pathology. There was no evidence of infection in the lung samples taken during postmortem examination. Also, there was no evidence of neoplastic or vascular disease in the lungs

DISCUSSION

The present case report describes an episode of spontaneous pulmonary haemorrhage of unknown origin in a horse during a dental procedure under sedation, with final euthanasia after initial stabilisation.

Once the haemorrhage was detected, one of the first steps in the treatment was to discontinue the detomidine CRI and to antagonise its effects with atipamezole. The atipamezole could have two main benefits in the case described here. First, atipamezole would be beneficial by antagonising the respiratory side effects of detomidine. Alpha-2 agonists produce a reduction in respiratory rate and minute volume in horses and an increase in ventilation–perfusion mismatch,[2] which could possibly add to the dyspnoea that the animal developed. Second, atipamezole antagonises the cardiovascular side effects of detomidine. Alpha-2 agonists produce vasoconstriction that can produce systemic and pulmonary hypertension. Pulmonary hypertension has been described as one of the possible factors involved in the development of exercise-induced pulmonary haemorrhage. Although not under sedation, fatal postanaesthetic pulmonary haemorrhage has been described in a horse with underlined chronic active exercise-induced pulmonary haemorrhage.[3] In the cited case, the use of alpha-2 agonists is mentioned (among others) as possible cause of the haemorrhage.

Acepromazine, used as premedication in the present case, has been proven to improve PaO_2 and ventilation–perfusion mismatch when given before alpha-2 agonist sedation[4] and has vasodilatory effects possibly also in the pulmonary vascular system. Nevertheless, we decided to use atipamezole in this case, because we did not know at that time what the aetiology of the haemorrhage was, and excessive pulmonary hypertension could have still been the cause. The use of atipamezole in this case could have had some negative consequences, such as arousal from sedation and increase in oxygen consumption. Doses from 0.1 to 0.16 mg/kg of atipamezole have been recommended for the antagonism of the effects of detomidine in horses.[5] To avoid a fast arousal from anaesthesia and due to the uncertainty of the origin of the bleeding, a lower dose of atipamezole was chosen in each of the two boli administered to the horse. The total dose was in the recommended dose range.

Nasal oxygen insufflation was also started soon in the treatment in the present clinical case. Pulmonary bleeding reduces the pulmonary area available for gas exchange once the alveoli fill with blood. In this case, hypoxaemia (defined as PaO_2 <80 mmHg[6]) was soon observed in blood gas analysis. Even with nasal oxygen supplementation, initially the PaO_2 and SaO_2 only marginally improved, maybe because of the reduced area available for gas exchange due to the presence of blood in the alveoli. The patient benefitted from oxygen supplementation, as tachypnoea and tachycardia were observed as soon as it was discontinued. The effect of nasal oxygen insufflation has been evaluated in standing healthy horses and

horses with severe and moderate recurrent airway obstruction, showing good tolerability of the technique and improved PaO_2 with flow rates of 10–20 L/min.[7]

In this case, the horse was given the antifibrinolytic drug tranexamic acid. Antifibrinolytic drugs stop haemorrhage due to inhibition of the breakdown of blood clots after their formation. Tranexamic acid avoids the interaction between fibrin and plasminogen and therefore, inhibiting the activation of plasminogen to plasmin and the dissolution of fibrin clots.[8] Tranexamic acid, due to its mechanism of actions, has no primary effect on blood coagulation, such as platelet counts, activated partial thromboplastin time, prothrombin times or levels of clotting factors.

The dosages of tranexamic acid in human medicine are not very well defined and seem to be dependent on the indication of use. In horses, its use is anecdotal, and dosages are generally extrapolated from human medicine (5–25 mg/kg IV every 12 hours). In the present case, the dose of tranexamic acid was within the range mentioned. There is evidence that horses have a lower fibrinolytic activity than humans and therefore this dosage could be excessive in horses. Fletcher et al.[9] measured the therapeutic plasma concentrations of tranexamic acid in healthy horses and they found inhibition of fibrinolysis occurred at 1/20 of the plasma concentration needed for the inhibition in humans. Usage of high dosages can potentially lead to thrombi formation, but there is no clear evidence of it either in equine literature or in our clinical case.

Antifibrinolytic therapy in horses has been used frequently to control bleeding in haemoperitoneum,[10,11] and improvement in outcome has been suggested. For the treatment of pulmonary bleeding, the evidence is scarce: a case report of successful conservative treatment of haemothorax in two horses[12] included the treatment with epsilon-aminocaproic acid, although its use could not be linked to the outcome. Also, there is no evidence supporting the efficacy in control or reduction of the haemorrhage by antifibrinolytic therapy for the treatment of exercise-induced pulmonary haemorrhage in horses.[13,14] Some more evidence exists supporting the use of tranexamic acid for the treatment of pulmonary haemorrhage of different aetiologies, when administered by inhalation or by bronchoscopy.[15–17] Although it is not possible to assess if the use of tranexamic acid had any positive effect in the present case, the relative safety of the drug in humans and dogs and the potentially life-threatening situation justified its inclusion as part of the treatment.

The necessity of a blood transfusion in this case is debatable. According to the recommendations,[18] blood transfusion is indicated in acute haemorrhage with an estimated blood loss of 30%. In this case, being the total blood volume of approximately 50 L, the blood loss (4 L) would be around 8% of total blood volume. It can be argued, analysing the case retrospectively, that a blood transfusion was not needed at that point, but the amount of blood loss in a short period of time and the uncertainty of knowing if the haemorrhage could have been controlled or not, made us make the decision.

Fresh whole blood was selected as product for transfusion due to two main criteria. First, in cases of acute haemorrhage, fresh whole blood is the product indicated because of the need of replacement of all the components of the blood, including clotting factors and platelets. Packed red blood cells will be more indicated in cases of normovolemic anaemia, and plasma transfusion or aggressive fluid therapy will not improve oxygen-carrying capacity.[18] The second criterion was availability, because in our facility there are no other options for blood transfusion. Reported blood transfusion reactions are around 16% in horses.[19] The reactions described can be acute or delayed haemolytic reactions, as well as, type I hypersensitivity reactions, ranging from moderate to severe intensity. Pretransfusion testing is recommended to reduce side effects. In case of emergency, a healthy gelding can be used as a donor.[18] Successful blood transfusion without previous crossmatching for treatment of an acute haemorrhage has been previously reported in equine anaesthesia.[20] Possible side effects of blood transfusion reactions were weighted against the benefits of blood administration and the emergency of the situation, and the transfusion was considered necessary.

In conclusion, the present case report describes the treatment of acute spontaneous pulmonary haemorrhage in a horse during standing sedation. Although the primary pathology could not be identified and the horse was eventually euthanased, the treatment can be considered as successful. An improvement in the condition was observed early after initiation of the treatment, and the horse's condition normalised for 5 days. The logical approach in treatment could be followed in similar cases. Also, it is important to highlight the fast and coordinated work and communication by the anaesthesia and internal medicine service of the veterinary hospital in the troubleshooting and treatment of the patient.

CONFLICT OF INTEREST

The authors declare they have no conflicts of interest.

ETHICS STATEMENT

In this case report, a complication during a clinical case has been described. All the treatment was approved by the owner at the time of administration.

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ORCID

Abraham Calero Rodriguez https://orcid.org/0000-0002-7491-9584

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