Myocardial injury following acute abdominal hemorrhage and septicemia in a seventeen-month-old draft horse

Myocardschade na een acute abdominale bloeding en septicemie bij een zeventien maanden oud trekpaard

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ABSTRACT

A seventeen-month-old draft horse gelding was presented with acute abdominal bleeding following castration. After two blood transfusions and medical treatment, clinical parameters stabilized, and the horse seemed to recover. Four days after admission, the horse suddenly developed tachycardia, tachypnea and pyrexia. Electrocardiography revealed paroxysmal monomorphic ventricular tachycardia related to myocardial injury. The horse recovered progressively after medical treatment and returned to normal sinus rhythm. In this case report, the importance to monitor for myocardial injury and cardiac arrhythmias after acute hemorrhage is highlighted.

SAMENVATTING

Een zeventien maanden oud trekpaard werd aangeboden met een acute, abdominale bloeding na castratie. Na twee bloedtransfusies en medicamenteuze behandeling stabiliseerden de klinische parameters en leek het paard te herstellen. Vier dagen na hospitalisatie ontwikkelde het paard plots tachycardie, tachypnee en koorts. Tijdens het elektrocardiografisch onderzoek werd paroxysmale monomorfe ventriculaire tachycardie vastgesteld als gevolg van myocardbeschadiging. Het sinusritme herstelde geleidelijk na medicamenteuze behandeling. In deze casereport wordt het belang benadrukt om bij een acute bloeding na te gaan of er myocardschade is opgetreden en of dit gepaard gaat met aritmie.

INTRODUCTION

Myocardial disease is poorly defined and rarely diagnosed in horses. Myocardial injury can be reflected by clinical signs that vary from absence of detectable clinical signs to dangerous tachyarrhythmias, heart failure and even sudden death (Nath et al., 2012b; Schwarzwald et al., 2013). The broad variety of clinical signs may result from different underlying etiologies and their effect on the myocardium (Decloedt, 2019; Dufourni, 2017). Multiple etiologies are known to cause myocardial disease, including infectious causes (viral or bacterial disease, including septicemia and bacteremia), endo- or exotoxemia, nutritional deficiencies (vitamin E or selenium), chronic systemic hypertension and an excessive exposure to catecholamines (pheochromocytoma), hypoxia and acute hemorrhage, immune-mediated processes, drugs, trauma, neoplasia, or can be of idiopathic or genetic origin (glycogen branching enzyme deficiency, dilated or hypertrophic cardiomyopathy) (Decloedt, 2019; Dufourni et al., 2017; Nath et al., 2012a; Navas de Solis et al., 2015; Schwarzwald et al., 2003; Schwarzwald, 2018; van Loon, 2019). The diagnosis is based on electrocardiography (ECG), echocardiography and blood analysis (Decloedt, 2019). Cardiac biomarkers, such as cardiac troponin I and T, significantly increase in the presence of myocardial disease (Van Der Vekens

et al., 2015). Increases in cardiac troponin concentrations can be a result of inflammatory myocardial disease or systemic disease, such as hemorrhage or endotoxemia (Navas de Solis, 2020). Definitive diagnosis of myocardial disease can be made by transvenous myocardial biopsies, although this technique is yet to be used in a clinical setting (Decloedt, 2019). Ventricular arrythmias detected on ECG might indicate myocardial injury. Depending on the localization of the myocardial injury, a different morphology and prolonged duration of the QRS complex can be observed (van Loon, 2019). Paroxysmal ventricular tachycardia, especially as result of myocardial injury, demands immediate therapy to avoid myocardial failure or ventricular fibrillation, which might result in sudden cardiac death (Schwarzwald et al., 2003; van Loon, 2019).

CASE REPORT

A seventeen-month-old draft horse gelding was presented at the equine internal medicine department, Ghent University, one day following castration in the field. Castration was performed under general anesthesia, but complications arose during the surgical procedure such as anesthetic difficulties and persistent bilateral bleeding from the spermatic cords, especially at the left side. Despite placement of additional ligatures on the spermatic cords, the bleeding could not be stopped. The referring veterinarian applied counter-pressure by means of tamponade and closed the scrotum with forceps. The morning after castration, the horse showed anorexia, lethargy, lateral decubitus, tachycardia and tachypnea. The referring veterinarian performed a rectal palpation which revealed an impaction of the pelvic flexure. The horse received six liters of tap water and two liters of paraffin oil by nasogastric intubation. Treatment at home prior to referral included the intravenous administration of 25 mg/kg metamizole (Calmagine®, Vétoquinol, France), 1.1 mg/kg flunixine meglumine (Finadyne®, MSD Animal Health, France), 0.16 mg/kg butyl scopolamine (Spasmizole®, Axience sas, France) and five liters of isotonic poly-ionic intravenous fluid. The horse was subsequently referred to the clinic.

At presentation, the horse was apathic, tachycardic (120 beats per minute (bpm)) and tachypneic (80 breaths/min) with pale mucous membranes. There was only mild swelling at the scrotal area, with mild loss of serosanguineous fluid from the castration wound. Hematology and blood biochemistry showed severe anemia (packed cell volume (PCV) (12%; ref. 30-47%) with leukocytosis ($16.5x10^9$/L; ref. 4.9-$11.1x10^9$/L), neutrophilia ($10.1 x10^9$/L; ref. 2.5-$6.9x10^9$/L), a decreased serum total protein concentration (40 g/L; ref. 55-75 g/L) and albumin concentration (17 g/L; ref. 19-32 g/L) (Table 1). Blood-gas and electrolyte analysis revealed hyponatremia (120 mmol/L; ref. 135-150 mmol/L), hypochloremia (81 mmol/L; ref. 97-107 mmol/L), hyperlactatemia (15.6 mmol/L; ref. < 2.0 mmol/L) and hyperglycemia (386 mg/dL; ref. 80-120 mg/dL). Abdominal ultrasonography revealed a moderate amount of cloudy, free abdominal fluid suggestive of intra-abdominal hemorrhage. Examination and ultrasound of the prepuce and scrotal area did not show signs of severe, active hemorrhage. An intravenous catheter was placed aseptically in the left jugular vein and two liters of hypertonic saline solution (Hypertonic NaCl-solution®, B. Braun, Germany) followed by five liters isotonic poly-ionic fluid (Ringer lactate, Baxter Healthcare Corporation, Ireland) were administered to restore the circulating volume and correct the electrolyte deficiencies, whilst a blood transfusion

Table 1. Overview of the parameters during hospitalization.

Parameter (reference interval)	Day of arrival	Day 4 after arrival		Day 8 after arrival
		10 AM	12 AM	
Packed cell volume (30-47%)	12	27	34	18
Leukocytes (4.9-11.1x109/L)	16.5	6.6	12.3	16.2
Neutrophil count (2.5-6.9x109/L)	10.1	4.58	9.41	10.0
Total protein (55-75 g/L)	40	N/A	62	62
Albumin (19-32 g/L)	17	N/A	24	22
Thrombocytes (100-250 K/µL)	38	52	56	40
L-Lactate (< 2.0 mmol/L)	15.6	0.8	1.0	N/A
Serum creatinine (35-157 µmol/L)	N/A	N/A	148	79
Blood urea nitrogen (4.1-8 mmol/L)	N/A	N/A	2.8	1.9
Potassium (3.0-4.9 mmol/L)	4.5	3.6	4.4	4.0
Chloride (97-107 mmol/L)	81	93	93	97
Calcium (1.4-1.7 mmol/L)	1.3	1.5	1.6	1.5
Sodium (135-150 mmol/L)	120	134	131	127

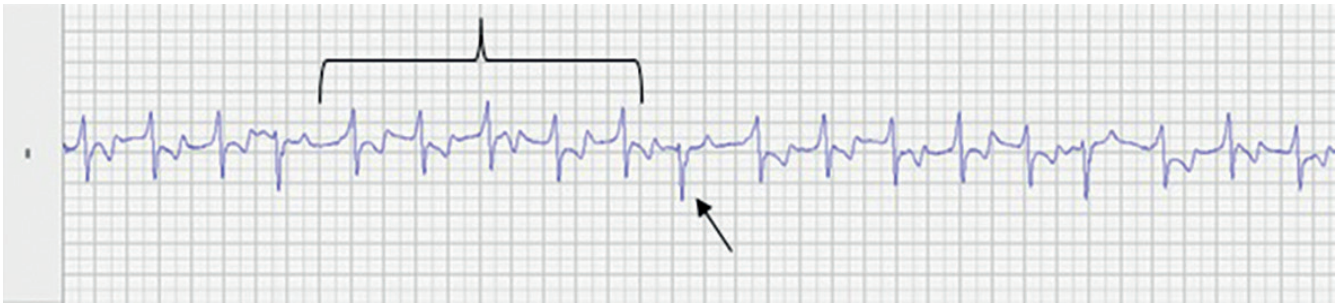

Figure 1. A modified base-apex electrocardiogram (Televet100) (50 mm/s) on day 4 shows runs of paroxysmal monomorphic ventricular tachycardia (accolade) interrupted by sinus beats (arrow) (128 bpm).

was prepared. The horse received 13 mg/kg etamsylate (Hemosilate®, Ecuphar Veterinary, Belgium) i.v. q6h, and 10 mg/kg tranexamic acid (Exacyl®, Sanofi, Belgium) i.v. q12h diluted in one liter of 0.9% sodium chloride solution, to stimulate coagulation and prevent fibrinolysis. A transfusion of six liters of blood was performed. The horse subsequently received 2.5 mg/kg trimethoprim and 12.5 mg/kg sulfadoxine i.v. (Borgal®, Virbac Animal Health, Belgium) and 1.1 mg/kg flunixine meglumine i.v. (Emdofluxine®, Emdoka b.v.b.a., Belgium). Heart rate, PCV and lactate were monitored every four hours. An improvement could be seen after the first blood transfusion: HR of 60 bpm, PCV at 16% (ref. 30-45%) and venous blood lactate at 10.7 mmol/L (ref. <2.0 mmol/L). Four hours after the initial blood transfusion, HR increased again up to 120 bpm and venous blood lactate increased to 14.2 mmol/ L (ref. <2.0 mmol/L). These changes were thought to be related to ongoing tissue hypoxia and hypoperfusion. Therefore, a second blood transfusion was performed. The horse received 0.07 mg/kg dexamethasone i.v. (Rapidexon®, Dechra, Belgium) to reduce the risk of transfusion reaction. Another blood donor was used since the maximum blood volume that could safely be taken of one and the same healthy blood donor was reached. Follow-up abdominal ultrasound the next morning revealed an increased amount of cloudy abdominal fluid. A laparoscopic approach to ligate both spermatic cords was considered, but was not performed due to the fact that the heart rate decreased to 60 bpm and venous blood lactate decreased to 7.6 mmol/L (ref. <2.0 mmol/L) in the morning and subsequently to 0.86 mmol/L in the evening. Intravenous broad spectrum antimicrobial treatment was continued with 6.6 mg/kg gentamicin i.v. (Emdogent®, Emdoka b.v.b.a., Belgium) q24h and 21000 IE/kg sodium benzylpenicillin i.v. (Penicilline®, Kela Pharma nv, Belgium) q8h. Three days (D3) after arrival, the horse remained stable, with a heart rate of 60 bpm and PCV 17% (ref.: 30-45%). Compressive gauzes and forceps that were placed by the referring veterinarian on the scrotum, were removed.

On day 4 (D4), the horse suddenly developed severe resting tachycardia (128 bpm), tachypnea (60 breaths/min) and pyrexia (39.3°C), while abdominal ultrasound revealed a decreased amount of free fluid and venous blood lactate (0.8 mmol/L; ref. <2.0 mmol/L), PCV (34%; ref. 30-47%), and electrolytes had further improved. An ECG recording revealed runs of paroxysmal monomorphic ventricular tachycardia, interrupted by sinus beats. (Figure 1). On echocardiography, the ventricular myocardium had a heterogenous aspect with hyperechogenic areas. Trivial pericardial effusion was observed. (Figure 2). M-mode ultrasonography on a short axis view of the left ventricle at chordal level revealed a decrease in the left ventricular internal diameter ($LVIDd_{M\_ch}$) and left ventricular fractional shortening (LV FS), and an increase in relative wall thickness at end-diastole ($RWTd_{M\_ch}$). Mild pulmonic regurgitation was also present (Table 2). Hematology revealed an increase in leukocyte count ($12.3x10^9$/L; ref. $4.9\text{-}11.1x10^9$/L) with a band neutrophil count, compared to the re-examination 14 hours earlier ($6.6x10^9$/L). Serum amyloid A (SAA) was measured for the first time and was also increased (2046 µg/ml; ref. <50 µg/ml). Serum biochemistry revealed increased aspartate aminotransferase (AST) (1080 IU/L; ref. 0-317 IU/L), lac-

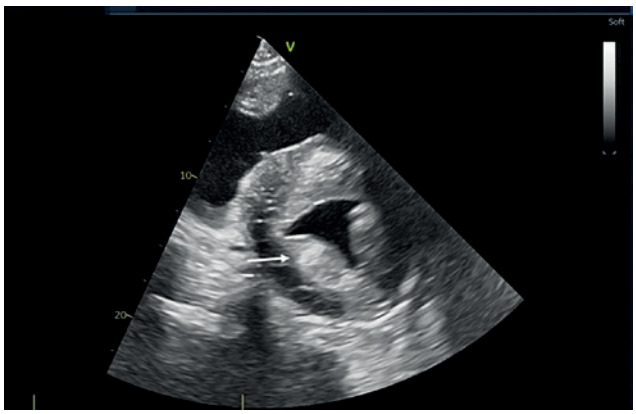

Figure 2. Echocardiography on day 4: a right parasternal short-axis view of the left ventricle at chordal level shows a heterogenous aspect of the left ventricular myocardium with hyperechogenic areas (arrow). Left ventricular internal diameter ($LVIDd_{M\_ch}$) and left ventricular fractional shortening (LV FS) are decreased, with an increased relative wall thickness at end-diastole ($RWTd_{M\_ch}$).

tate dehydrogenase (LDH) (5750 IU/L; ref. 0-1337 IU/L) and creatine phosphokinase (CK) (586 IU/L; ref. 0-354 IU/L), mild hypomagnesemia (0.5 mmol/L; ref.: 0.7-1.1 mmol/L) and an increase in total bilirubin concentration (106 µmol/L; ref. 0-60 µmol/L). Cardiac troponin I (cTnI) was severely increased to 12.40 ng/ml (ref. 0-0.06 ng/ml). Serum creatinine, blood urea nitrogen (BUN) and electrolytes were within normal limits. An antiarrhythmic treatment was immediately started. Magnesium sulphate (0.03 mg/kg magnesium sulphate in one liter of 0.9% sodium chloride solution) was administered intravenously. Subsequently, 1.3 mg/kg lidocaine (Lidor®, Ecuphar, Belgium) i.v. over 15 minutes followed by a constant rate infusion (CRI) of 0.05 mg/kg/min was given. Further supportive treatment included intravenous isotonic poly-ionic fluid, intranasal oxygen and 0.01 mg/kg i.v dexamethasone (Rapidexon®, Dechra, Belgium). In addition, 20 mg/kg of vitamin E and 0.40 mg/kg selenium (Etosol-SE®, Dechra, Belgium) were administered intramuscularly. A blood sample was aseptically collected from the right jugular vein for bacteriological examination. Metronidazole (Flagyl®, Famar Health Care, Spain) at 15 mg/kg p.o. q6h was added to the initial antimicrobial treatment because of persistent high fever. After two hours of lidocaine CRI, the horse converted to sinus tachycardia with frequent ventricular premature depolarizations (VPDs). The frequency of VPD's decreased progressively and a consistent sinus tachycardia was observed. Lidocaine treatment was terminated after 52 hours and an oral antiarrhythmic treatment with 10 mg/kg phenytoïn sodium p.o. (Diphantoine®, Kela Pharma nv., Belgium) q12h was initiated for twelve days. Echocardiography was repeated two days after the onset of ventricular tachycardia. Cardiac measurements normalized (Table 2). Blood culture tested positive for *Acinetobacter johnsonii*, which showed in vitro resistance against penicillin and intermediate susceptibility to gentamycin. Antimicrobial treatment was changed to 10 mg/kg doxycycline p.o. (Doxylin 50% WSP®, Dopharma, the Netherlands) q12h according to the antimicrobial susceptibility results. The ongoing pyrexia resolved subsequently.

Two weeks after castration, suppurative discharge from the castration wounds was observed, which was suggestive for the presence of a bilateral funiculitis. Bacterial culture of the scrotal suppuration revealed the presence of *Morganella morganii spp morganii* and *Streptococcus uberis*. Topical treatment by means of scrotal rinsing with a 0.05% diluted chlorhexidine solution was performed. The patient was discharged after nineteen days of hospitalization.

DISCUSSION

Myocardial insult after hemorrhage is well described in humans and companion animal medicine, but rarely in equine medicine (Navas de Solis et al., 2015). Myocardial hypoxia and hypoperfusion might lead to myocardial damage in horses with acute hemorrhage (Navas de Solis et al., 2015). Sympathetic nervous system stimulation is a part of the autonomic response to acute stress and hypovolemia. It provides

Table 2. M-mode variables obtained from a right parasternal short-axis view of the left ventricle at chordal level.

Ultrasonographic variables	Unit	Day 4	Day 6	Reference Draft horse mare of the same age	Reference value Warmbloods	Reference value Friesians
$RVIDd_{M\_ch}$	cm	3.7	3.5	3.5	2.3 - 5.5	1.4 - 4.5
$RVIDs_{M\_ch}$	cm	3.1	2.6	2.4	1.7 - 4.9	0.4 - 3.9
RV FS	%	16	25	32	11 - 26	13 - 71
$IVSd_{M\_ch}$	cm	4.2	3.1	2.7	2.4 - 3.7	2.2 - 3.4
$IVSs_{M\_ch}$	cm	3.9	4.2	4.1	3.4 - 5.3	3.7 - 5.4
$LVIDd_{M\_ch}$	cm	5.4	10.3	10.5	9.4 - 12.9	9.5 - 12.6
$LVIDs_{M\_ch}$	cm	4.4	5.8	4.1	4.8 - 8.6	4.8 - 7.8
$LVFWd_{M\_ch}$	cm	3.0	1.8	1.9	1.8 - 3.0	1.9 - 3.1
$LVFWs_{M\_ch}$	cm	4.0	3.7	3.9	3.2 - 5.1	3.3 - 5.2
LV FS	%	17	43	48	27 - 53	32 - 53
$RWTd_{M\_ch}$		1.1	0.48	0.44	0.37 - 0.62	0.39 - 0.59
HR	bpm	128	74	32-44	32 - 44	34 - 44

Abbrevations: $RVIDd_{M\_ch}$ (Right ventricular (RV) internal diameter at end-diastole); $RVIDs_{M\_ch}$ (Right ventricular (RV) internal diameter at peak-systole); RV FS (Fractional shortening of the RV); $IVSd_{M\_ch}$ (Interventricular septal thickness at end-diastole); $IVSs_{M\_ch}$(Interventricular septal thickness at peak-systole); $LVIDd_{M\_ch}$ (Left Ventricular (LV) internal diameter at end-diastole); $LVIDs_{M\_ch}$ (LV internal diameter at peak-systole); $LVFWd_{M\_ch}$ (LV free wall thickness at end-diastole); $LVFWs_{M\_ch}$ (LV free wall thickness at peak-systole); LV FS (Fractional shortening of the LV); $RWTd_{M\_ch}$ (Relative LV wall thickness at end-diastole); HR (Heart Rate); bpm (beats per minute)

a short-term adaption to stressful situations, resulting in increased inotropy and chronotropy, as well as an increased demand of myocardial oxygen, which might worsen ischemia (Bellotto et al., 2015; Liaudet et al., 2014). The increase in circulating catecholamines changes the membrane permeability, which might lead to electrolyte imbalances, additionally contributing to myocardial injury (Decloedt, 2022; Liaudet et al., 2014). Electrolyte imbalances are commonly found in horses after acute hemorrhage, as in this case (Navas de Solis et al., 2015). Navas de Solis et al. (2015) showed that low plasma protein, low PCV and high plasma creatinine concentrations are correlated with the development of arrhythmias. Beside cardiac ischemia, blood loss causes systemic and renal hypoperfusion with subsequent (pre)-renal increases in plasma creatinine concentrations and cardiac troponin concentrations. Due to the emergency at admission of the patient, tachycardia was thought to be the result of ongoing abdominal bleeding, hypovolemia, hypoxia, ischemia and stress. Therefore, unfortunately, no electrocardiography was performed at admission. Routine ECG examination and troponin measurement in patients with acute hemorrhage are useful for the early diagnosis of myocardial injury and arrhythmias. Beside the severe hemorrhage, several signs of sepsis were observed in this patient. *Acinetobacter johnsonii* was isolated from bacterial blood culture. Milton et al. (2015) described *Acinetobacter spp.* as bacteria that are part of the normal flora on skin and mucosal membranes, which may cause opportunistic infections. Although sample contamination could not be excluded, a broad-spectrum antimicrobial treatment was immediately initiated, because of the presence of a hemoabdomen. Septicemia and bacteremia have been described as predisposition factors for the development of myocardial disease. In this patient, it is thought that both the severe hemorrhage and septicemic spread of opportunistic bacteria have contributed to the myocardial injury. Scrotal suppuration was not likely to be associated with the myocardial damage because it was caused by different bacteria and appeared two weeks after the initial insult.

Because of the severity of the blood loss, a combination therapy with etamsylate and tranexamic acid was administered in addition to the blood transfusion. Etamsylate is a procoagulant drug stimulating platelet adhesion, while tranexamic acid improves clot formation and decreases fibrinolysis (Dunkel, 2018). The combination of these products has not been studied in horses yet. However, a study on pediatric cardiac surgery revealed synergistic effects by reducing post-operative blood loss and therefore the need for a blood transfusion compared to the sole administration of tranexamic acid (El Baser et al., 2021).

The blood transfusions performed in this case were not preceded by a cross match, due to the urgency of the situation. Since the patient was a young gelding that had no prior exposure to blood products according to the owner, the risk of a blood transfusion reaction was thought to be low. An incidence of 16% for blood transfusion reactions, with only a fraction of the blood transfusion reactions being related to red blood cell alloantigen incompatibility has been described (Radcliffe et al., 2022). Routine crossmatching evaluates the hemagglutination. Other reactions cannot be predicted with this technique (Mudge, 2014; Radcliffe et al., 2022). Corticosteroids were administered i.v. and a slow initial transfusion rate was used while constantly checking clinical parameters, to minimize risks associated with anaphylactic reactions.

In this specific case, a sudden increase in heart rate was observed concurrent with pyrexia. An ECG was recorded because of the high heart rate, despite a blood lactate of 1.0 mmol/L, PCV of 27% and an improvement of abdominal ultrasonographic findings. Electrocardiography revealed a monomorphic ventricular tachyarrhythmia. When ventricular arrhythmia is diagnosed, it is important to assess whether the cause of the rhythm disturbance originates from structural heart disease or is of extra-cardiogenic origin (Navas de Solis, 2020). Definitive diagnosis is made based on history, ECG, echocardiography and blood examination, including cardiac troponin I or T determination (Decloedt, 2019; Van Der Vekens et al., 2015). Cardiac troponin I was highly increased in this case. Since cardiac troponins are regulatory proteins in the contraction and relaxation of cardiac muscle cells, increases are indicative of myocardial damage. However, these increases can also be found without a primary cardiac insult, for example in case of endotoxemia, hemorrhage, hypovolemia, drug toxicity and chronic renal failure (Daubert and Jeremias, 2010; Navas de Solis; 2020). In this specific case, it was thought that severe hypovolemia could have been the cause of myocardial injury. Due to the fact that an ECG was not performed at admission, it remains uncertain whether the arrhythmia was present on arrival.

CONCLUSION

In this case report, the importance of electrocardiography, echocardiography and cardiac troponin determination is highlighted in patients with severe blood loss for early diagnosis of myocardial injury and presence of potentially dangerous arrhythmias. Further research is necessary to identify parameters that allow better assessment of the effect of hemorrhage on myocardial injury and to assess potential long-term effects.

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