

*Case report*

MYOCARDIAL INFARCTION IN A HORSE WITH COLIC –  
CLINICAL, LABORATORY AND PATHOMORPHOLOGICAL  
FINDINGS

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**Summary**

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A clinical case of a horse with colic due to large intestinal meteorism and secondary proximal enteritis is reported. The physical examination revealed signs of endotoxaemia (cyanotic mucosae with petechiae and a toxic line), fever, polypnea, tachycardia and pulse deficit. The haematological results indicated haemoconcentration with marked leukopaenia, neutropaenia and a left shift. Blood biochemistry demonstrated changes in blood electrolytes, muscle enzymes, glucose, creatinine, urea and very high levels of serum cardiac troponin I. Electrocardiography findings consisted in premature ventricular complexes and ventricular tachycardia. Cardiac ultrasound did not show any structural changes. Myocardial infarctions were observed *post mortem*.

**Key words:** cardiac troponin I, ECG, horse, infarction, myocardium, ventricular extra systole

*Case history*

An 8-year-old Hanoverian gelding, weighing 520 kg, was referred to the Equine Clinic of the Faculty of Veterinary Medicine, Trakia University, Stara Zagora, Bulgaria with 48-hour moderately strong bout of colic, interrupted by short painless pauses, lack of defecation and bilateral abdominal distention. According to the history, two punctures of the left iliac fossa have been performed. The horse has been repeatedly treated with analgesics (xylazine), spasmolytics (buscolysin), non-steroid anti-inflammatory

drugs (metamizole, flunixin meglumine) and electrolytes (Ringer, Duphalyte, 5% Glucose with sodium chloride). Laxatives with sites of action small and large intestine (castor oil and Istizin) were also applied.

*Clinical signs*

The clinical examination revealed a marked depression, food refusal, hyperhydrosis and tachycardia. The conjunctivae were diffusely inflamed, of a dirty red colour, with petechiae and thick sticky discharge. The mucosa of the

mouth was dry, cyanotic, and with “toxic” rings around the base of incisors. The capillary refill time was prolonged over 3 s. Skin elasticity was reduced, and the skin fold in the shoulder region returned to position after more than 4 s. The rectal body temperature was 38.5 °C. A pulse deficit was detected: with manually measured 60 *a. facialis* pulse waves vs. a heart rate of 165 min<sup>-1</sup> during auscultation. The pulse was weak, barely detectable and arrhythmic.

The auscultation of the heart showed tones of various strength, some of them rather dull and unclear. There were no pathological heart sounds.

The bilateral abdominal auscultation revealed a complete lack of small and large intestinal peristalsis. The rectal investigation showed a distended pelvic flexure, occupying the pelvic inlet and impeding the additional penetration of the hand into the abdominal cavity. No faeces were present in the ampulla. After placement of an oesophageal probe, gas masses and 4–5 L rusty-brown fluid with unpleasant putrid odour were evacuated (gastric reflux). The breathing was rapid (48 min<sup>-1</sup>) and superficial. There were no stridor, cough, pathological sounds (rales, spasms, grating noise). The palpation of digital arteries at the base of proximal

sesamoid bones of forelimbs and hindlimbs did not show acceleration of the pulse and laminitis.

The electrocardiography (Cardipia 400, Trismed, Korea, Y-lead) showed premature ventricular complexes, appearing as bigeminy and multifocal paroxysmal ventricular tachycardia with atrioventricular dissociation on the background of tachycardia (approximately 160 ventricular systoles/min – Fig. 1). Cardiac ultrasound was performed through the left and right cardiac “window”, parasternal and transverse planes (SonoScape S6V, linear microconvex head 3.5 MHz, B mode). Echocardiography did not show any structural changes in the pericardium, walls, the septum and the valves.

#### *Blood analysis*

CBC findings (Serono Plus System, USA) consisted in erythrocytosis – 13.62 T/L, hyperchromaemia (haemoglobin 223 g/L), haemoconcentration (haematocrit 59.6%), leukopaenia (total leukocyte counts 3.6 G/L), mean corpuscular volume 51.3 fL. Differential leukocyte count test established 5% monocytes, 2% metamyelocytes, 25% band neutrophils, 42% segmented neutrophils and 30% lymphocytes. There was a neutropaenia (2.48 G/L), left shift and lymphopaenia (1.08 G/L).



**Fig. 1.** ECG showing premature ventricular complexes – bigeminy and paroxysmal ventricular tachycardia, Y lead, rate 25 mm/s.

Blood serum analysis (Hematology Analyzer BA-88, Mindray) was characterised with reduced total protein concentration – 61 g/L, increased urea (21.2 mmol/L) and creatinine (604.3 µmol/L), hyperglycaemia (15 mmol/L), increased total bilirubin (85 µmol/L). Liver enzymes were within the normal range (ALAT 18 U/L, ASAT 286 U/L). Muscle enzyme activities were elevated: LDH – 1783 U/L, creatine kinase – 1850 U/L. Serum potassium was increased – 5.61 mmol/L, calcium was lowered (2.03 mmol/L), sodium and chlorides were also reduced (126.4 mmol/L and 82 mmol/L, respectively). Serum cardiac troponin I concentration was 3.45 ng/mL.

#### *Medication therapy*

The therapy of the horse included a massive fluid and electrolyte infusion for correction of fluid and electrolyte imbalance. Ringer lactate at 80 mL/kg/24 h with added 5 g/L calcium gluconate was intravenously administered. Lidocaine 2% was applied intravenously as a 1.3 mg/kg bolus (Vetprom, Bulgaria) followed by 0.05 mg/kg/min during the subsequent hours because of its antiarrhythmic effect and beneficial local anaesthetic properties described by Lester (2000) and Lohmann & Barton (2004). For enhancement of intestinal motility, a single subcutaneous dose of 0.022 mg/kg neostigmine (Konstigmin<sup>®</sup>, Vetoquinol AG) was administered.

Protein loss was compensated with infusion of 5 L fresh plasma. Additionally, amino acids and vitamins were injected (Duphalyte<sup>®</sup>, Ford Dodge, USA) to provide vital substances to the organism. As *Clostridium* spp. was suspected as a possible cause for proximal enteritis, antimicrobial therapy with 20,000 UI/kg procaine-benzylpenicillin

and 20 mg/kg dihydrostreptomycin (Intramycin<sup>®</sup>, Ceva) was done i.m. at 12-hour intervals. The analgesic combination of 0.01 mg/kg detomidine (Domosedan<sup>®</sup>, Orion) and 0.06 mg/kg butorphanol (Butomidor<sup>®</sup>, Richter Pharma) was selected due to its minimum effect on intestinal motility as reported by McConnico (2004).

#### *Course of disease*

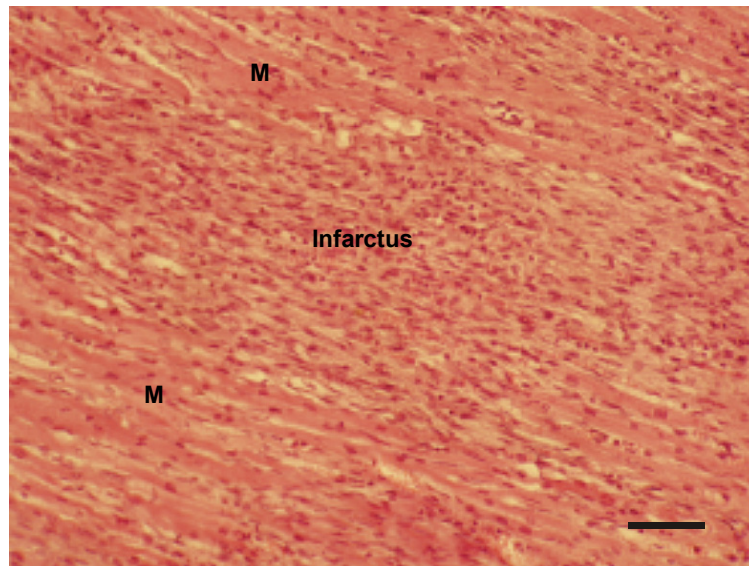
The main clinical signs of the horse, ECG and blood biochemical parameters were monitored at 2-hour intervals. Despite the efforts, the clinical status of the patient did not improve. By the 10<sup>th</sup> hour after the admission, rapid worsening of clinical parameters, collapse with ataxia, and fatal outcome after 4–5 min have occurred.

#### *Pathoanatomical findings*

The walls of the duodenum and jejunum were haemorrhagically inflamed, filled with rusty-brownish fluid of a putrid odour. The gastric findings were similar: its content was of similar colour, odour and consistency. The large colon and the caecum were filled with excess gas, and their content was little and of soft consistency.

The liver, the kidneys and the spleen did not exhibit visible gross changes. The lungs were obviously hyperaemic (venous congestion), and foamy exudates were observed on its cut surface and within the bronchi.

Macroscopically, the left ventricular wall had gray-whitish foci with irregular margins, sharply delineated from the adjacent intact tissue. Some of these areas were surrounded by a hyperaemic zone. Tissue samples were collected from the parts of the myocardium showing gross signs of infarction, near to the intact tissue. They were fixed in 10% neutral formalin, routinely processed and embed-



**Fig. 2.** Relatively recent infarction (Infarctus) of the myocardium (M). The necrotic tissue is infiltrated mainly with erythrocytes and at a lesser extent with leukocytes and macrophages. H/E, bar =25  $\mu$ m.

ded in paraffin. The cut sections of approximately 5  $\mu$ m, were stained with haematoxylin and eosin (H/E).

Histologically, areas with abnormal structure of various size and shape were detected among the studied myocardial tissue. At the same time, infarctions showing features of different occurrence in time were present. In relatively more recent infarctions, the necrotic tissue was infiltrated mainly with erythrocytes and at a lesser extent – with leukocytes and macrophages (Fig. 2).

The main clinical problem identified throughout the examination, was the large intestinal meteorism and small intestinal paralytic ileus. An additional aggravating factor was the secondary dilatation of the stomach and the reflux of small intestine content. The massive myocardial injury also contributed to the critical clinical condition, which, together with the dehydration reduced tissue perfusion with

blood, as evidenced by cyanotic mucous coats and the prolonged capillary refill time.

Increased erythrocyte counts, haemoglobin content and haematocrit resulted from the insufficient fluid intake and the simultaneous water release through urine, sweat and expired air. Haemoconcentration was also provoked by the fluid collection in small intestinal lumen and the stomach. The resulting dehydration was the cause for reduced skin elasticity and prolonged capillary refill time. The fever is attributed to the inflamed gastric and intestinal wall (enterocolitis, gastritis). The leukopaenia, neutropaenia and lymphopaenia were specific indicators of occurring endotoxaemia (Barton, 1998). As neutropaenia is observed during the early stages of endotoxaemia, and is replaced by neutrophilia in the recovery stage, it could serve to indicate patients at risk, which require a close monitoring.

The observed haemorrhages on conjunctivae, the cyanosis and the “toxic” line of oral mucosa are consequent to endotoxins, absorbed from the gut.

Increased blood urea and creatinine concentrations are probably due to dehydration (prerenal azotaemia) although the development of renal insufficiency could not be definitely excluded. The observed hypoproteinaemia in combination with increased haematocrit are resulting from protein loss and sympathetically stimulated splenic contraction (Lohmann & Barton, 2004). Moderately increased blood bilirubin on the background of normal liver enzyme activities could result from the 48-hour fasting. In the view of Lohmann & Barton (2004), liver enzymes may be elevated in endotoxaemic horses, but liver failure is extremely rare. Increased muscle enzyme concentrations are probably due to impaired muscle fibre integrity following soft tissue trauma during the colic bouts.

Hyperkalaemia was due, on one hand, to the decreased potassium excretion with urine, induced by hypovolaemia and impaired renal function, and to muscle cells trauma and necrosis resulting in intracellular potassium transfer into the extracellular space on the other. Hypochloraemia and hyponatraemia resulted from losses with sweat and by internal losses into the gastric and intestinal lumen, a.k.a. “fluid sequestration” (Lohmann & Barton, 2004). Hypocalcaemia has been observed in Gram-negative sepsis in humans (Zaloga & Chernow, 1987), as well as in horses with strangulation ileus and intestinal infarctions (Dart & Snyder, 1992). A possible reason for the low blood calcium could be endotoxin-induced secondary parathyroid gland insufficiency, as well as renal damage leading to inhibited 1,25 hydroxylation of vitamin D (Lohmann & Barton, 2004).

Hypoglycaemia was reported in neonatal sepsis and foals with endotoxaemia (Lavoie *et al.*, 1990; Morris, 1991). In our case, hyperglycaemia was present, confirming the findings of Burrows (1971) in adult toxaemic horses.

The high serum concentration of cardiac troponin I that is highly specific for myocardial tissue (3.45 ng/mL), compared to reported normal values up to 0.3 ng/mL (Cornelisse *et al.*, 2000; Schwarzwald *et al.*, 2003) indicated a massive damage of the muscle wall. Numerous studies in patients with myocardial damage have confirmed the role of cardiac troponins (I, T) as extremely sensitive and specific diagnostic tools, no influenced by physical exercise, renal or skeletal muscle damage (Guyton & Hall, 2000; Collinson *et al.*, 2001; Fromm & Roberts, 2001). That is why cardiac troponins became the “gold standard” in myocardial damage diagnostics in human medicine. Recently, they are increasingly used for diagnostics of cardiac diseases in animals. Normal troponin I values have been reported in dogs (<0.03–0.07 ng/mL) and cats (<0.03–0.16 ng/mL) (Sleeper *et al.*, 2001).

Myocardial lesions were responsible for the heart-related clinical signs. The presence of pulse deficit is due to the functioning of additional “ectopic” foci of electric activity in ventricular walls, which impair the systolic synchrony and sinus node impulses. Clinical manifestations of myocardial disorders could vary from a mild discomfort and decreased working capacity to severe congestive heart failure, leading to cardiogenic shock. Reimer *et al.* (1992) and Marr *et al.* (1998) outline cardiac arrhythmias combined with multiform ventricular tachycardia as a bad prognostic sign in horses.

According to Patteson (1999) and Schwarzwald *et al.* (2003) the lesions of the myocardium could not be observed by ultrasonography, due to the small size of necroses on one hand, and the technical limitations of the animal size, on the other.

Myocardial fibrosis is a very frequent finding in necropsy studies of horses, which were asymptomatic for cardiovascular disorders *ante mortem* (Brown, 1985; Dudan *et al.*, 1985). There are single case reports for alterations of the heart muscle accompanied by specific clinical signs. This impedes extremely the detection of the underlying cause for myocardial damage (Marr, 1999). Agents causing myocardial injuries could be of toxic, viral, bacterial, traumatic, ischaemic and parasitic origin (Reef, 1992).

The diagnostics of myocardial disorders in human medicine is based on assay of serum concentrations of cardiac enzymes: CK-MB and ASAT, as well as specific proteins (troponins I and T). In this case, the total CK blood level was very elevated. As ASAT activity was within the normal range, we suggest that the high CK is rather due to muscle traumas.

The present case report is interesting from a clinical point of view, as it follows up the course of an episode of horse colic caused by pathological changes in small (proximal enteritis) and large intestines (meteorism). Apart the typical clinical signs, the CBC changes resulting from the endotoxaemia and dehydration are described. An additional adverse prognostic factor for the outcome was the myocardial damage (infarction), followed by cardiac arrhythmia and death. In such complicated cases, the time diagnostics and adequate treatment are essential to improve the patients' chance for survival.

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