



EXERTIONAL RHABDOMYOLYSIS IN A FALLOW DEER (*CERVUS DAMA*)

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Summary

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The aim of this report was to present a case of exertional rhabdomyolysis in a fallow deer. The diagnosis was made on the basis of clinical and blood laboratory findings. Despite the treatment, the outcome was fatal. Gross anatomy and histopathological examinations were performed, detecting degenerative changes in the heart, skeletal muscles, the kidneys and the liver. On the basis of *ante* and *post mortem* findings providing explanations of the pathophysiological mechanism of severe tissue damage – hypoxaemia, severe lactic acidosis, muscle rupture, electrolyte imbalance and renal block, it was concluded that all they have led to the death of the animal.

Key words: doe, myopathy, pathology, rhabdomyolysis

Myopathy induced by extreme exertion during capture is a fatal condition, whose mechanisms remain still not understood regardless of the numerous investigations.

The disease may occur in any animal in extreme conditions, yet some species are more susceptible due to their natural behavioural, physical and evolutionary characteristics. Myopathy is most commonly observed in wild ungulates (Paterson, 2008) – white-tailed deer, mountain goats, sheep, antelopes, bison, moose and long-legged wild birds e.g. ratites, snow geese, Canada geese, wild turkeys, seagulls, bald eagles and golden eagles (Menon *et al.*, 2014). The condition was

also detected in coyotes, badgers, primates, fish and amphibians.

Myopathy is caused by various forms of physical and emotional stress. In hoofed mammals, factors predisposing to the development of exertional rhabdomyolysis are the higher brain mass, higher maximum running speed, living in large social groups and longer life span (Blumstein *et al.*, 2015). Higher ambient temperatures and immobilisation increase the risk from exertional rhabdomyolysis (Woodbury, 2005).

Occurrence of death in deer during chasing and capture ranges between 2.1 and 48% depending on the used technique

for immobilisation (Cromwell *et al.*, 1999).

Four different forms (syndromes) of exertional rhabdomyolysis are acknowledged depending on the time elapsed from the stress event, clinical and within minutes to 6 hours, and death results from sudden circulatory or cardiac block. The second form/stage is the ataxic myoglobinuric syndrome that develops from the 6th to the 48th hour. It is characterised with azotaemia, acidosis and renal insufficiency. If the animals survive after these acute stages, muscle damage progresses to necrosis and rupture of big muscle groups. The stage is termed muscle breakdown syndrome. Some animals do not exhibit signs of enumerated stages, but could perish within several days to weeks after capture. This form is termed delayed peracute syndrome, and death occurs suddenly consequently to heart failure.

The disease is unpredictable and of multifactorial nature, and its control poses a great challenge to the veterinarian. The development of efficient methods for treatment requires better understanding of pathophysiological events leading to death, therefore specific research information on the topic is essential.

This case report describes the clinical, blood laboratory and pathological findings in a fallow deer doe, affected with exertional rhabdomyolysis.

Case presentation

A female fallow deer (*Cervus dama*), one year old, weighing 30 kg owned by the State hunting farm in Gramatikovo, Burgas region, was referred to the Small Animal Clinic of the Faculty of Veterinary Medicine, Trakia University – Stara Zagora on 18 September 2017 with the following disease history: 6 days ago, after prolonged chase, the animal has been

immobilised with xylazine and ketamine by darting for resettlement purposes. The duration of anaesthesia was 2 hours, and after the animal woke up, was not able to stand on its feet. Since then, it did not feed, did not drink; no treatment was applied. The hunting farm was located in the middle Strandzha mountain, altitude 46–487 m with settlement coordinates 42.062180, 27.654790, and maximum day ambient temperature of 21–25 °C.

At referral, the patient was recumbent, awake and reacted to environmental stimuli. The clinical parameters comprised a preserved consciousness, alertness; body temperature – 39.8 °C (reference range 37.5–40.5 °C) (Galka *et al.*, 1999), heart rate 56 min⁻¹ (reference range 24–52 min⁻¹); respiratory rate 24 (reference range 6–28 min⁻¹); rumen movements 6/5 minutes, pale rose visible coats, lymphatic and blood vessels – normal; skin and haircoat – 10% dehydration.

Specific orthopaedic and neurological tests showed neither fractures and dislocations of the axial and appendicular skeleton, no neurologic deficit. The only abnormal finding was the pronounced muscle weakness of all limbs with inability for getting up.

Laboratory analyses. Venous blood samples of 5 mL was collected from v. jugularis in vacutainers with K₂EDTA and heparin for haematological and biochemical analysis. Complete blood counts were assayed on an automated haemalyser Mindray BC 5000 Vet (China). Blood biochemical parameters and electrolytes were assayed on an automated biochemical analyzer (BS-120 Mindray Biochemistry Analyzer, China) with commercial kits (Biolabo SAS, France).

Arterial blood was obtained aseptically by puncture of the femoral artery for blood gas analysis. The employed single-

use Respiratory/blood gases VetStat® cassettes for VetStat® electrolyte and blood gas analyzer, IDEXX Laboratories, Inc., USA) assayed blood pH, haemoglobin (Hb) – $\mu\text{g/L}$, HCO_3^- (mmol/L), PaCO_2 (mmHg), tCO_2 (mmol/L), PaO_2 (mmHg), BE (mmol/L), tHb (g/L), Sat (%), K^+ (mmol/L), Na^+ (mmol/L), Cl^- (mmol/L), and anion gap (mmol/L).

A urine sample was obtained by manual pressure on the urinary bladder through the abdominal wall. It was assessed organoleptically and with urine test strips Urocolor 10 (China) on a reader (Uro meter 120, China). Urine sediment was evaluated by microscopy.

The blood tests showed no deviations in CBC parameters (Table 1). Blood bio-

chemistry demonstrated increased activities of creatine kinase, alanine aminotransferase and aspartate aminotransferase. Electrolytes and blood gases were also within reference ranges. Urine sample did not show any macroscopic, microscopic and chemical deviations.

On the basis of performed examinations, exertional rhabdomyolysis was diagnosed and the following treatment was performed: daily intravenous infusions with Ringer or sodium chloride 0.9% – 500 mL; glucose 5% – 500 mL; sodium hydrogen carbonate 8.4% – 20 mL; Cato-sal – 5 mL; vitamin C – 5 mL; dexamethasone 0.2% (Alfasan, Holland) – 3 mL. Subcutaneously, vitamin B complex (Introvit) – 5 mL and antibiotic (procillin)

Table 1. Morphological, biochemical parameters and electrolytes in venous blood and arterial blood gases in a fallow deer doe

Parameter	Measured value	Reference value*
Erythrocytes, T/L	10.0	9.3±1.5
Haemoglobin, g/L	142	131±19
Haematocrit, L/L	0.43	0.386±0.53
Total leukocytes, G/L	12.0	9.1±1.2
Platelets, G/L	336	457±155
Total protein, g/L	62	65.33±4.16
Albumin, g/L	35.21	33.33±3.05
Urea, mmol/L	6.3	6.96±3.53
Creatinine, $\mu\text{mol/L}$	111	134.33±28.36
Glucose, mmol/L	7.8	6.47±1.44
Creatine kinase, $\mu\text{kat/L}$	11.96	3.51±1.19
ALAT, $\mu\text{kat/L}$	3.64	0.62±0.25
ASAT, $\mu\text{kat/L}$	7.18	1.11±0.25
LDH, $\mu\text{kat/L}$	18	19.86±9.70
Potassium, mmol/L	5.34	4.43–6.24
Calcium, mmol/L	2.84	2.54–3.14
Phosphate, mmol/L	2.4	2.71–3.70
Magnesium, mmol/L	0.85	0.56–1.13
pH	7.38	7.35–7.50
PaO_2 , mmHg	76.6	80–100
PaCO_2 , mmHg	48.7	35–44
Lactate, mmol/L	1.68	1.5–2.0

* Vengust *et al.* (2002; 2006); Poljicak *et al.* (2004); Kucer *et al.* (2013).

– 3 mL were administered at 72-hour intervals. The doe was provided with a separate, quiet box with a thick straw litter and water in close proximity. Hay, fresh tree branches and concentrate were offered as feed. During the entire treatment, no deviations in clinical parameters were found out, the appetite was variable, water intake unsatisfactory, no changes in the locomotor activity was noted.

Six days later, after fatal outcome, the carcass was submitted to gross anatomy and histopathological examinations. The gross anatomy exam of the doe's body was performed in line with the standard necropsy protocol. After detection of macrolesions of the femoral muscle, heart, liver and kidneys, samples for histopathological examinations were collected. Specimens (1×1 cm of size) were fixed in 10% neutral buffered formalin. After fixation, they were embedded in paraffin blocks, 4 µm sections were cut and routinely stained by haematoxylin/eosin – H/E (Dzhurov *et al.*, 1989; Dyakov *et al.*, 1989) and Masson-Goldner trichrome (Merck Millipore, Germany).

Macroscopic changes found out during the external body inspection comprised dehydration, enophthalmos, weight loss and emaciation. Visible mucous coats of conjunctivae and the oral cavity were pale. After skin removal, muscles of the thigh and thoracic wall were atrophied, oedematous and spattered with numerous petechiae (Fig. 1). Dissection of the abdominal cavity revealed diffuse hyperaemia on the surface of abdominal organs. The liver was brown, with rounded margins, and the gallbladder was enlarged. Kidneys appeared atrophied, with hyperaemic medullar zone. The heart was pale, with thinned and relaxed walls – dilation. Dissection of the endocardium demonstrated gray-whitish foci (Fig. 2). No visi-

ble pathological alterations were found out in the other organs and systems.

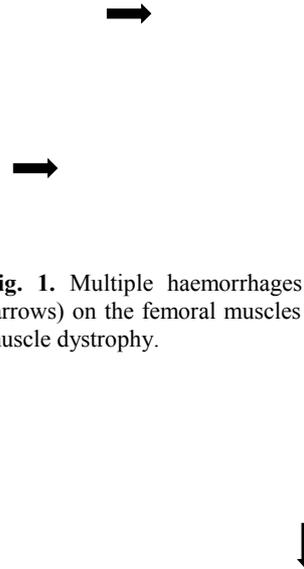


Fig. 1. Multiple haemorrhages and oedema (arrows) on the femoral muscles of a doe with muscle dystrophy.

Fig. 2. Gray-whitish foci on the endocardium (arrow) in a doe with muscle dystrophy.

Microscopic changes of femoral and heart muscles were manifested with hyaline degeneration (hyalinisation) with fragmentation and oedema of muscle fibres (Fig. 3). In single areas, degenerative necrobiotic processes with loss of striated pattern and proliferation of new connective tissue were observed (Fig. 4).

Histopathologically, liver epithelial cells showed parenchymatous degeneration and necrobiotic lesions. Sinusoidal

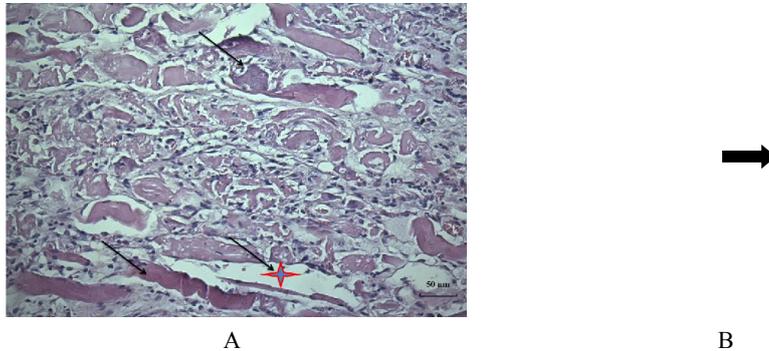


Fig. 3. Femoral muscle of a doe with muscle dystrophy. **A.** Fragmentation with oedema (star) and hyalinisation (arrows) of femoral muscle fibres in a doe with muscle dystrophy, H/E, bar=50 µm **B.** Masson-Goldner trichrome staining of connective tissue, bar=50 µm.

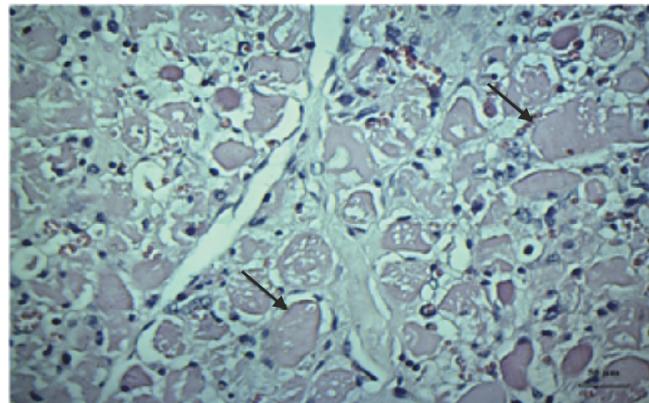


Fig. 4. Degenerative necrobiotic changes, hyperaemia and hyalinisation (arrows) of cardiac muscle fibres in a doe with muscle dystrophy, H/E, bar=50 µm.

capillaries were dilated and overfilled with red blood cells (Fig. 5). Vascular endothelial cells were activated, and many of them were transformed in siderocytes. Microscopic changes of kidneys (Fig. 6) comprised signs of cloudy swelling and granular degeneration, disintegration and desquamation of epithelial cells lining the basement membrane.

Exertional rhabdomyolysis affects different animal species, but wild ungulates and waterfowl are the most susceptible. In men, genetic mutations encoding disorders in muscle metabolic pathways were also

reported (Nance & Mammen, 2015). The predisposition of animals to myopathies may be genetically mediated as well.

Exertional rhabdomyolysis is clinically manifested with myalgia (in 84% of cases), muscle swelling (in 8.1% of patients) and muscle weakness (74%), developing within hours to several days after muscle injury (Chen *et al.*, 2013). In the present case, muscle swelling was not observed by the 6th day after the chase. The animal demonstrated no acute myalgia. The only clinical sign was muscle weakness.

Exertional rhabdomyolysis in a fallow deer (Cervus dama)

The exertional rhabdomyolysis is diagnosed on the basis of clinical findings accompanied with presence of myoglobin in urine or marked elevation of serum creatine kinase (CK) activities (Nance & Mammen, 2015). Plasma myoglobin increases rapidly after the traumatic injury but is also rapidly excreted through the kidneys and normal concentrations are

regained within 24 hours. Unlike myoglobin, blood serum CK increases during the first 2–12 hours after muscle injury, attained peak levels on day 3–5 and slowly decreased during the next 6–10 days. This specific time course of the two rhabdomyolysis parameters explained why in the described case myoglobinuria was not found out, but only increased blood CK.

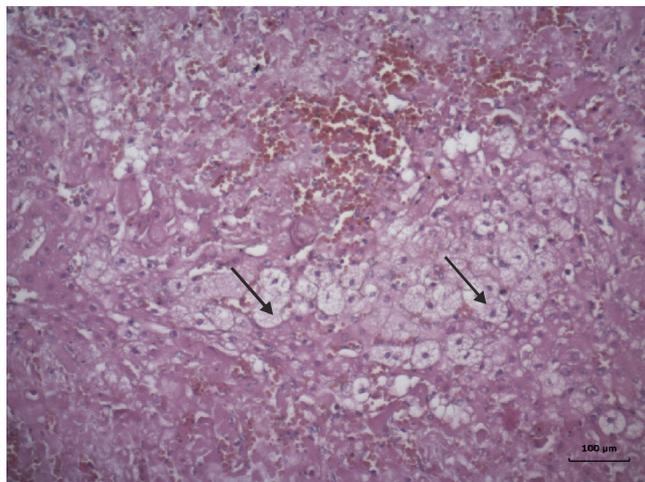


Fig. 5. Degenerative changes in liver epithelial cells (arrows), hyperaemic sinusoid capillaries in a doe with muscle dystrophy, H/E, bar=50 µm.

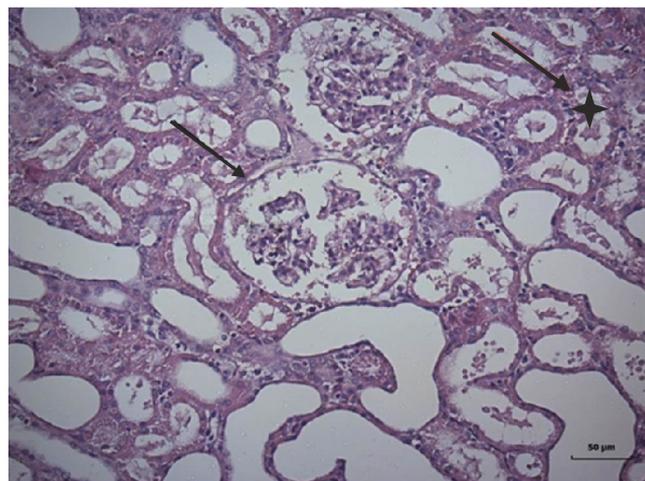


Fig. 6. Degeneration, disintegration and desquamation of epithelial cells lining the basement membrane of kidney tubules (star) and glomeruli (arrows) in a doe with muscle dystrophy, H/E, bar=50 µm.

According to most authors, blood CK activity increase more than 5 times above upper reference values is a diagnostic sign for rhabdomyolysis (Park *et al.*, 2013). Yet, this feature is not specific, as many chronic muscle diseases, either inflammatory or dystrophic, were accompanied with several fold increase in blood CK.

Pigmenturia accompanies 5.3 to 68% of cases (Zepeda-Orozco *et al.*, 2008). It is specific for acute form of rhabdomyolysis which is accompanied with azotaemia and animals die from renal failure. In the present case, neither myoglobinuria nor changes in other urinalysis parameters were found out. Plasma urea and creatinine were without deviations, evidencing a preserved renal function. The findings of Fitte (2017) also revealed no extensive changes in urea and creatinine, both in early hours and until the 30th day after the chase.

Muscle-specific serum enzymes ASAT and CK were indicative for a substantial muscle injury on the 6th day after capture. According to Fitte (2017) ASAT elevation began several hours after the muscle trauma, reached peak concentrations on the 2nd day and thereafter gradually decreased until the 16th day. Muscle enzymes reacted to hypoxia with increased activities, which were preserved over a longer period (10–14 days). Jones & Price (1992) have used LDH and its skeletal muscle isoenzyme LDH-5 as indicator of stress in intense muscle activity during capture of wild fallow deer and found out that activities of both plasma LDH-5, and total LDH increased proportionally to chasing time, without correlation with blood cortisol. These results revealed that the enzyme reacted immediately to severe exertion of 1 hour duration and declined rapidly after elimination of the stress. This was also confirmed by the results of Nu-

voli *et al.*, (2014), that 35-minute anaesthesia with xylazine/tiletamine and 3-hour transportation of freely living deer have caused drastic increase in plasma potassium, cortisol, CK and LDH following by sudden death on the 16th hour. In our patient, blood LDH activity was not elevated although histopathological findings confirmed serious muscle and heart injury, due to the subacute course of the disease.

The results from blood gas analysis showed no data for hypoxaemia and lactate acidosis on the 6th day after chasing and capture. These data agree with findings of Fitte (2017), who affirmed that life-threatening deviations in pH and blood gases occurred during the first 25 hours and then returned to normal. The cited study demonstrated 8–9 fold increase in lactate concentration but only during the 1st day after the chase. In the present case, lactate levels on the 6th day were unchanged.

Plasma electrolyte concentrations vary within broad ranges in healthy animals, but are comparable in different deer species, depending largely on the method of capture with the purpose of blood sample collection (Vengust *et al.*, 2006). In cases of muscle injury associated to acute stress, plasma CK, LDH and potassium concentrations are rapidly increased (Stringer *et al.*, 2011; Nuvoli *et al.*, 2014). In this study, no deviations from normal ranges were found out on the 6th day after the stressful event.

Zeiler & Meyer (2017) and Chinnadurai *et al.* (2016) affirmed that chemical immobilisation with anaesthetic drugs is the technique of choice compared to physical capture, as it reduces the stress from manipulations and muscle injury. It is associated with lower mortality (4%) and morbidity (23%) than physical capture (11%) (Nuvoli *et al.*, 2014; Nyambe *et al.*, 2017).

The choice of anaesthetics is focused on drugs with rapid and marked anxiolytic and antistress effect, leading to good muscle relaxation, minimum physiological and metabolic changes, with possibility for antagonisation (Miller *et al.*, 2009). The application of opioids (etorphine and thiafentanyl) causes respiratory distress, which may lead to death (Chinnadurai *et al.*, 2016; Zeiler & Meyer, 2017). Both medication and physical restraint have been connected to accidents at the time of fixation of animals, e.g. fractures, fatal injuries when an inappropriate technique of anaesthesia with dart that may puncture thoracic or abdominal cavity and cause death of the animal is used (Paterson, 2008).

The established pathological macro- and microlesions of striated musculature (skeletal and cardiac), as well as in parenchymal organs (liver and kidneys) of necropsied fallow deer doe were characteristic of rhabdomyolysis in wild even-hoofed mammals and correspond to those described by other researchers (Williams *et al.*, 1996; Valentine, 2007).

The detected microlesions of hyaline degeneration (hyalinisation) with fragmentation and swelling of muscle fibres, loss of the striated pattern and degenerative necrobiotic changes in muscles were similar to those previously reported by Zahid *et al.* (2018). Unlike this case of late peracute rhabdomyolysis with presence of renal cortical necrosis and tubular degeneration, we have not found out similar fatal renal damage. The presented case is also referred to as late peracute syndrome, but the death has occurred as a result of extensive irreversible changes in the heart muscle.

In conclusion, the exertional rhabdomyolysis in the fallow deer doe on the 6th day after chasing did not demonstrate the

specific biochemical finding explaining the pathophysiological mechanism of serious tissue damage – hypoxaemia, high-degree lactate acidosis, muscle rupture with electrolyte imbalance and renal block. Only the sequels of hypoxia on striated and cardiac muscles, reflected by increased blood serum activities of muscle enzymes were detected. The blood biochemistry could neither reflect adequately the severity of injuries, nor could predict the fatal outcome.

The mortality rate is high, despite the treatment and occurs in all stages of the disease. Therefore, prevention is the only approach for its reduction. Necessary measures include short chase at a lower speed in colder weather, minimum stress and capture of animals, short duration of transportation, sedation and familiarisation of animals with manipulations, rapid induction and recovery from anaesthesia, short anaesthesia with alpha-2 agonists reducing stress response and phenothiazines abolishing anxiety, short stay in closed facilities; capture in groups instead of individual capture, no unnecessary noise.

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