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Case Report

Rhabdomyolysis Triggered by Septic Shock in a Dog: A Case Report

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Abstract

The purpose of this report is to describe an unusual case of severe rhabdomyolysis associated with septic shock. A 13-year-old Pekingese male dog was admitted to our department with a history of respiratory distress, fatigue, severe myalgias, vomiting, and generalized severe asthenia. The creatine kinase (CK) level was very high (13.690 UI/L) on the 1st day of hospitalization and increased to 31.587 UI/L on the 2nd day. CK values during the 4th and 9th days were 15.796 UI/L and 1.064 UI/L, respectively. Despite aggressive shock treatment and adequate treatment of secondary infections and the complication of rhabdomyolysis (azotemia and liver failure), the patient developed progressive myalgia, progressive respiratory failure, and low compliance, resulting in death on the 9th day of hospitalization.

Keywords: *Rhabdomyolysis, Creatine kinase, Septic shock, Dog*

Bir Köpekte Septik Şoka Bağlı Gelişen Rabdomiyoliz Olgusu: Olgu Sunumu

Öz

Bu vaka raporunun amacı septik şokla birlikte ender görülen şiddetli rabdomiyoliz olgusunu bildirmektir. Onüç yaşlı, erkek, Pekinez ırkı köpek hastanemize solunum güçlüğü, halsizlik, şiddetli kas ağrısı, kusma ve güçsüzlük şikayetleri ile başvurdu. Serum biyokimyası analizi sonucu 1. gün CK seviyesi 13.690 UI/L olurken, 2. gün 31.587 UI/L değerine yükseldi. Dördüncü ve 9. günlerdeki CK değerleri sırasıyla 15.796 UI/L ve 1.064 UI/L olarak ölçüldü. Agresif şok tedavisi ve rabdomiyoliz sonucu gelişen komplikasyonlara yönelik yapılan tüm tedaviye rağmen hastada ileri miyalji ve şiddetli solunum yetmezliğine bağlı 9. günde ölüm gerçekleşti.

Anahtar sözcükler: *Rabdomiyoliz, Kreatin kinaz, Septik şok, Köpek*

INTRODUCTION

Rhabdomyolysis is an acute necrosis of striated muscle [1]. It ranges in severity from an asymptomatic elevation of creatine kinase (CK) level in the blood, to severe life-threatening cases associated with very high CK levels [2]. Muscle cell contents such as myoglobin are released into the circulatory system, causing acute tubular necrosis and resulting in acute renal failure. Presence of myalgias, significant muscle weakness, red-to-brown urine (myoglobinuria), and elevated CK levels are considered as clues to rhabdomyolysis [3]. There are many traumatic and non-traumatic causes of rhabdomyolysis in humans. In the first category, causes include: crush injuries, long-lasting muscle compressions such as that caused by prolonged immobilization, electrical shock injury, and venom from a snake or insect bite. Nontraumatic causes of rhabdomyolysis include extreme muscle strain (exertional rhabdomyolysis), the use of medications such as antipsychotics or statins, especially when given in high doses, elevated body temperature (hyperthermia) or heat stroke, seizures or delirium tremens, metabolic disorders such as diabetic ketoacidosis, viral infections such as the flu, HIV, or herpes simplex virus, bacterial infections leading to the presence of toxins in tissues or bloodstream (sepsis) [4].

In veterinary medicine, exertional rhabdomyolysis has been reported as a common cause of rhabdomyolysis in dogs and racehorses [5,6]. Holahan et al.[7] reported a case in a dog with presumptive hepatotoxicity and rhabdomyolysis secondary to phenazopyridine toxicity. Lechowski et al.[8] reported acute idiopathic rhabdomyolysis in a dog in Poland.

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Septic shock-induced changes in the lung and kidneys have been studied extensively both clinically and modeled experimentally, but little is known of alterations in other organ systems. Skeletal muscle is a well-perfused and voluminous tissue and when in shock it may be assumed that its endothelium reacts similarly to the endothelium of the lung and kidneys. We present the clinical findings of severe rhabdomyolysis triggered by septic shock in dogs.

CASE HISTORY

A 13-year-old Pekingese male dog was admitted to our department with a history of respiratory distress, fatigue, severe myalgias, vomiting, and generalized severe asthenia. There was no history of trauma, seizures, surgery, snake or insect bite, contact with chemical agents. Additionally, the patient was not regularly receiving prescribed drugs.

Clinical Examination

On the initial examination, the dog was unconcise, and in a stupor state, with dehydration (8-10 percent). The patient was unable to stand, lying on his sternum, with pain in the abdominal cavity and muscles *(Fig. 1)*.

There were cyanotic mucosal membranes and prolonged capillary refill time ([CRT]: 4sec). The dog had reduced bilateral palpebral reflexes and decreased myotatic reflexes in all four limbs. The panniculus reflex was considered normal, and all other cranial nerve reflexes were intact. Pulse was very weak.

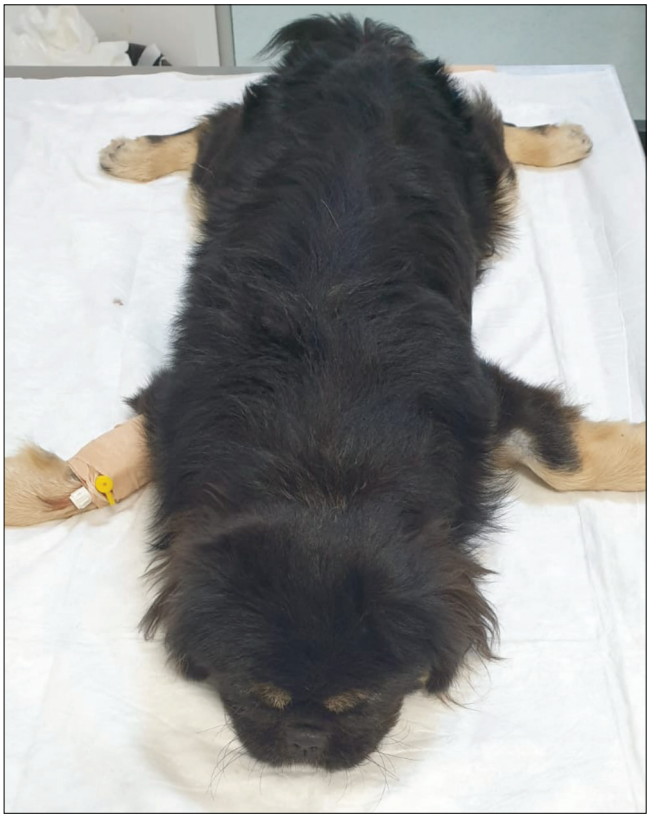

Fig 1. The patient was unable to stand, lying on his sternum, with pain in the abdominal cavity and muscles

After clinical examination, blood samples were collected from the cephalic vein. The diagnosis of severe sepsis was based on meeting the criteria of at least two variables, compatible with SIRS and the dysfunction of no less than one organ or have evidence of tissue hypoperfusion [9]. The considered SIRS variables were hypothermia (36°C) *(Table 1),* tachypnoea (35/min [reference range: <25/min]), tachycardia (180 bpm [reference range: 60-160 bpm]) and leukocytosis (30.20×10^9/L) with granulocytosis (26.71×10^9/L) and a thrombocytopenia (85.00×109/L) *(Table 2)*. Haematocrit value was decreased (35.40%) *(Table 2)*. C-reactive protein (C-RP) value was elevated (47.13 mg/L) *(Table 2)* (Mindray BS120, Shenzen, China). Venous acid-base analysis (Epocal Inc., Ottawa, ON, Canada) showed that the dog had hypobasemia (pH: 7.37; bicarbonate [HCO_3]: 18 mmol/L; partial pressure of carbon dioxide [pCO_2]: 31.80 mmHg; base excess [BE]: -7 mmol/L) *(Table 3)*.

The variables associated with organ dysfunction included arterial hypotension (systolic blood pressure [SBP]: 83 mmHg, mean arterial pressure [MAP]: 63 mmHg), decreased oxygen saturation [SpO_2]: 74%) *(Table 1)* (Mindray BS120, Shenzen, China), decreased ionized calcium (0.69 mmol/L), decreased glucose (55 mg/dL) (Epocal Inc., Ottawa, ON, Canada), increased alanine aminotransferase (ALT) (141 UI/L), increased ALP (201 UI/L), decreased albumin (2.47 g/dL) (Mindray BS120, Shenzen, China), increased blood urea nitrogen (BUN) (53.85 mg/dL), increased creatinine (1.60 mg/dL) (Epocal Inc., Ottawa, ON, Canada), and increased phosphor (8.13 mg/dL), slightly increased LDH (297 UI/L) and extremely high creatine kinase (CK) level (13690 UI/L) *(Table 3)* (Mindray BS120, Shenzen, China). Plasma lactate

Table 1. *Body temperature, blood pressure and tissue oxygenation parameters in dog on day 1, 2, 4, 9*

Parameters	Reference Range	Day1	Day2	Day4	Day9
Body temp. (°C)	37.5-39.3	36	38.5	38.2	35.5
SBP (mmHg)	>90	83	110	115	78
MAP (mmHg)	>65	63	85	85	60
SpO_2 (%)	>92	74	91	94	71

Table 2. *Hematological parameters and C-reactive protein (C-RP) values in dog on day 1, 2, 4, 9*

Parameters	Reference Range	Day1	Day2	Day4	Day9
WBC ($\times10^9$/L)	6-17	30.20	21.00	30.20	16.20
Granulocyte ($\times10^9$/L)	4-12	26.71	18.11	26.71	18.11
Haematocrit (%)	39-56	35.40	30.50	35.40	19
Thrombocyte ($\times10^9$/L)	180-460	85	27	41	25
C-RP (mg/L)	0-10	47.13	45	37.03	30.33

Table 3. *Acid-base balance and biochemical analysis parameters in dog on day 1, 2, 4, 9*

Parameters	Reference Range	Day1	Day2	Day4	Day9
pH	7.35-7.45	7.37	7.40	7.45	7.45
PCO_2 (mmHg)	35-38	31.80	35.30	39.20	49.00
HCO_3 (mmol/L)	20-30	18	22	27.50	18.80
BE (mmol/L)	-5-0	-7	–2.7	3.50	-4.7
Lactate (mmol/L)	<2.3	1.59	1.70	1.63	7.37
Ionized calcium (mmol/L)	1.12–1.40	0.69	1.34	0.89	0.37
Glucose (mg/dL)	60-110	55	121	123	91
BUN (mg/dL)	10-28	53.85	7.45	21.49	15.14
Creatinine (mg/dL)	0.5-1.5	1.60	0.90	0.87	1.27
Phosphor (mg/dL)	2.60-6.20	8.13	3.25	4.24	3.24
ALT (UI/L)	21-102	141	142	140	340
ALP (UI/L)	20-156	201	195	178	274
Total bilirubin (mg/dL)		0.64	0.41	0.59	4.74
LDH (UI/L)	45-233	297	219	189	299
CK (UI/L)	48-261	13.690	31.587	15.796	1.064
Albumin (g/dL)	2.60-3.30	2.47	2.42	2.46	1.89

(1.59 mmol/L) *(Table 3),* sodium (143 mmol/L [reference range: 139-150 mmol/L]) chlorine (116 mmol/L [reference range: 106-127 mmol/L]) and potassium (3.20 mmol/L [reference range: 3.40-4.90 mmol/L]) concentrations were in normal reference ranges (Epocal Inc., Ottawa, ON, Canada). Thyroid function tests were normal. Tests for toxoplasmosis and neosporosis were negative (Mindray BS120, Shenzen, China). Urine was normal in color with no proteinuria and myoglobinuria. Blood culture was performed, and *S. pseudintermedius* was the Gram-positive isolate.

Volume resuscitation was initiated using normal saline infusion (90 mL/kg, bolus) by peripheral venous access, to improve the clinical parameters and blood pressure (BP), followed by administration of diuretics (Furosemide (Lasix®, Sanofi Ltd.,Turkey) 4 mg/kg. IV, every 6 h). Maintenance fluid therapy was administrated with NaCl 0.9% (Polifarma, Turkey) at a dose of 40 mL/kg/day to maintain urine output.

After one bolus volume resuscitation, BP did not increase. Vasopressor (Norepinephrine) (1-2 mg norepinephrine (Cardenor®, Vem, Turkey) in 250 mL 0.9% NaCl (Polifarma, Turkey), IV) was applied. Systolic blood pressure (SBP) and mean arterial pressure (MAP) increased to 110 mmHg and 85 mmHg, respectively *(Table 1)*.

The dog received oxygen therapy, with a nasal oxygen mask (100 mL/kg/min) according to SpO_2. Concurrent administration of triple antimicrobial therapy with ceftriaxone (Eqiceft®, Tüm Ekip, Turkey) 30 mg/kg, IV, every 12 h, metronidazole (Polgyl®, Polifarma, Turkey) 10 mg/kg, IV, every 12 h, enrofloxacin (Dufafloxacin®, Holland) 5 mg/kg, IM, every 12 h and, theophylline (Biofleks Teosel®, Osel, Turkey) 10 mg/kg, IM, every 12 h, dexamethasone (Dexaveto-0.2®, Belgium) 0.2 mg/kg, IM, every 12 h and enoxaparin sodium (Oksapar®, Koçak Farma, Turkey) 1.5 mg/kg, IM, every 12 h were administrated. The diagnosis of septic shock was based on the presence of severe sepsis and hypotension that could not be reversed with fluid resuscitation [9]. Decreased ionized calcium and glucose concentrations were corrected by IV administration of calcium (Calcium Picken 10%®, Adeka, Turkey) 0.1 mL/kg, IV and dextrose (30% Dextrose® (Polifarma, Turkey) 0.5-1 mL/kg, IV. The chest radiograph and abdominal X-ray and ultrasound examinations were unremarkable.

The ECG was normal. Transthoracic echocardiography using right parasternal long and short-axis windows was performed to evaluate LV systolic function [10-12]. The echocardiographic findings included a normal LV systolic function with ejection fraction (EF) (82%) and stroke volume (SV) (12.74 mL).

The CK level was very high (13.690 UI/L) on the 1st day of hospitalizations and increased to 31.587 UI/L on the 2nd day. CK values during the 4th and 9th days were 15.796 UI/L and 1.064 UI/L, respectively *(Table 3)*.

Venous acid-base status and azotemia were normalized by the 2nd day of hospitalization *(Table 3)*.

On the fourth day of the treatment, the dog started to walk and receive food orally, however, liver enzymes (ALT, ALP) *(Table 3)* and C-RP values were still high *(Table 2)*. His urine was still normal.

On the ninth day of illness, the dog was in a sternal paralyzed position with a diffusely tender abdomen and depressed. Despite intensive therapy, the cyanotic mucosal membranes, prolonged CRT, low body temperature *(Table 1),* increased granulocyte count *(Table 2)* and high lactate concentration persisted *(Table 3)*. There was no azotemia *(Table 3)*. However, liver enzymes (ALT, ALP) and total bilirubin concentration were high *(Table 3)*. pCO_2 increased to 49.00 mmHg in the venous acid-base analysis. Despite restoration to a normal hydration status (dehydration degree <5%), the BP was still hypotensive *(Table 1)*. The acute phase response never responded to treatment, with the C-RP 3 times upper limit of normal *(Table 2)*. He became acutely worse, with no oral intake, persistent severe abdominal pain, and severe myalgias that made movement difficult.

Despite aggressive shock treatment and adequate treatment of secondary infections and the complication of rhabdomyolysis (azotemia and liver failure), the patient developed progressive myalgia, progressive respiratory failure, and low compliance, resulting in death on the 9th day of hospitalization. Because of the emotional reason, the patient owner was reluctant to consider necropsy.

DISCUSSION

In humans, signs and symptoms of rhabdomyolysis may be hard to pinpoint. This is largely true because the course of rhabdomyolysis varies, depending on its cause. And, symptoms may occur in one area of the body or affect the whole body. Also, complications may occur in early and later stages [4]. The "classic triad" of rhabdomyolysis symptoms in humans are (1) muscle pain in the shoulders, thighs, or lower back; (2) muscle weakness or trouble moving arms and legs; and (3) dark red or brown urine or decreased urination. However, half of the people with the condition may have no muscle-related symptoms [4]. In this case, there was no history of trauma, seizures, surgery, snake or insect bite, contact with chemical agents. He did not take any drugs regularly. We agree that the course of rhabdomyolysis may vary because we did not observe dark red or brown urine and most prominent symptoms were muscle-related. Khan [2] informed that the definitive diagnosis of rhabdomyolysis should be made by laboratory tests including serum CK and urine myoglobin. In our case, there was no myoglobinuria, however significant increments in serum CK were determined *(Table 3)*. So, severe myalgia, unexplained muscle weakness, and elevated CK were the key to diagnosis.

Serum CK concentration, mainly the CK-MM subtype, is the most sensitive indicator of muscle damage. Serum CK begins to rise approximately 2 to 12 h after the onset of muscle injury, peaks within 24 to 72 h, and then decline at a relatively constant rate of 39% per day [13]. In this dog, the CK level was very high (13690 UI/L) on the 1st day of hospitalizations and increased to 31587 UI/L by the 2nd day of hospitalizations. CK values at the 4th and 9th days were 15796 UI/L and 1064 UI/L, respectively. In this case, the peak value of 31587 UI/L was during the 2nd day of hospitalizations and declined at the constant rate to 1064 UI/L on the 9th day of the treatment *(Table 3)*. This could be a result of intense volume repletion, followed by the administration of diuretics (Furosemide).

Although various values of CK have been postulated to define rhabdomyolysis, the magnitude of elevation is rather arbitrary; and there is no cut-off value that conclusively diagnoses rhabdomyolysis in humans. A serum CK activity greater than five times the normal value (in the absence of heart or brain diseases) was accepted as a criterion for the diagnosis of rhabdomyolysis [14]. However, the Clinical Advisory on Statins defined statin-induced rhabdomyolysis as muscle symptoms with marked CK elevation typically substantially greater than 10 times the upper normal limit, with a creatinine elevation consistent with pigment nephropathy and usually with brown urine with myoglobinuria [6,15]. In veterinary medicine, marked CK elevation in exertional rhabdomyolysis and toxication has been determined as high as 187380 U/L [8]. There is no data concerning septic shock. In this dog, serum CK activity was ten times greater than the normal value (13.690 UI/L) seen on the 1st day of admission *(Table 3)*.

Myoglobin is normally bound to plasma globulins and has a rapid renal clearance which maintains a low plasma level up to a certain serum concentration (0 to 0.003 mg/dL). After the occurrence of muscle damage, the circulating myoglobin levels exceed the plasma protein binding capacity, reach the glomeruli, and are eventually excreted in the urine [2]. We failed to detect myoglobinuria in the initial and following evaluations. This could be explained by studies in human medicine where Cervellin et al.[3] informed that myoglobinuria is detected in a varying proportion (28-70%) of patients with rhabdomyolysis. Khan [2] and Minnema et al.[16] also stated that serum myoglobin precedes the rise in CK and drops rapidly. Serum myoglobin usually increases before a rise in CK and drops more rapidly than the decline in CK concentration (in one to six hours). Moreover, myoglobinuria may not be visible or may resolve early in the course of rhabdomyolysis. These facts make this parameter less sensitive and therefore should not be relied upon to rule out the diagnosis of rhabdomyolysis. Thus, myoglobinuria may be undetectable in a patient presenting with muscle weakness and high CK. Our patient had a 2 days history of weakness and myalgias thus myoglobinuria may be undetectable at presentation [17]. Finally, we may say that myoglobinuria does not occur without rhabdomyolysis, but rhabdomyolysis does not necessarily lead to visible myoglobinuria (tea or cola-colored urine).

Once the diagnosis of rhabdomyolysis is established, a search must be instituted for a cause. In our case, there was no history of trauma, seizures, surgery, snake or insect bite, contact with chemical agents, and no medication regularly. Our dog had all the criteria for septic shock including *S. pseudintermedius* isolate in blood culture [9,18]. Rhabdomyolysis may occur as part of the septic syndrome in which hemodynamic instability and elaboration of bacterial toxins and other cytokines that may either selectively or collectively contribute to muscle necrosis. It is noteworthy in this regard that both the tumor necrosis factor-a (TNF-α) and interleukin-1β, elaborated by septic patients, are capable of causing acute proteolysis in the skeletal muscle cells [19]. Cytokines are known to activate branched-chain a- ketoacid dehydrogenase, the rate-limiting enzyme in branched-chain amino acid oxidation in the muscle, leading to a severe catabolic state. TNF-α is also known to cause an acute reduction in the cross skeletal muscle cell plasma membrane, implying direct injury to the muscle cell or an increase in Na permeability of the muscle cell. An increase in cytosolic Ca rapidly follows the increased Na permeability of the cell, resulting in swelling and eventual death of the muscle cell [10,19]. So, decrements in calcium and albumin concentrations in our case could be the result of muscle cell death. The accumulation of substantial amounts of fluid into the affected muscle cause

hypovolemia. At the same time, high intra-compartmental pressure provokes additional damage and necrosis [20]. This further muscle damage is manifested as the 'second wave phenomenon', with persistent elevation or rebound elevation in CK levels at 48 to 72 hours after the initial insult [21] *(Table 3)*.

High liver enzymes (ALT, ALP) and total bilirubin concentration, azotemia at admission, high lactate, high LDH hypothermia, hypobasemia, hypoglycemia, low blood pressure, decreased SpO_2 and thrombocyte count, and increased WBC, and C-RP could be explained by septic shock or/and rhabdomyolysis, and dysfunctional organ systems *(Table 3)*.

Blood gas analysis allows the interpretation of acid-base status as well as respiratory function, including both oxygenation and respiration. In our dog, there was hypobasemia (HCO_3: 18 mmol/L; BE: -7 mmol/L) and respiratory alkalosis due to decreased pCO_2 (31.80 mmHg) despite normal blood pH (7.37) on the day1 *(Table 3)*. The decreased pCO_2 can be assumed to be respiratory compensation of the hypobasemia. However, pCO_2 increased to 49.00 mmHg on the day9. A high pCO_2 is compatible with respiratory acidosis. This could be the result of sepsis-associated ARDS. Increased lactate concentration (7.37 mmol/L) on day9 supports this conclusion.

Our patient presented with severe rhabdomyolysis with a peak CK level of 31587 UI/L complicated by renal, respiratory, and hepatic failure. It should be emphasized that the risk of renal, respiratory, and hepatic failure could be decreased by early detection of rhabdomyolysis through routine measurement of CK level in patients with sepsis. Established shock and elevated CK level subsequently resulted in a cascade of renal failure, hepatic failure, secondary infections, and respiratory failure due to progressive ARDS with eventually a fatal course. Perhaps the clinical outcome would have been different if alarming signs had been recognized on time and shock could have been prevented.

In conclusion, we describe an unusual case of severe rhabdomyolysis associated with septic shock. This unusual case may further add to the understanding of rhabdomyolysis with sepsis.

Statement of Author Contributions

KT: Conceptualization, Methodology, Writing - review & editing; AN, HS, ME and MEI: Writing - review & editing..

Conflict of Interest

None

REFERENCES

1. Buyuktas D, Tascilar K, Ugurlu S, Unalan H, Ozdogan H: Rhabdomyolysis as the presenting symptom of lung carcinoma: A case report. *Lett Ed Rheumatol*, 1 (2): e110009, 2011. DOI: 10.2399/ler.11.0009

2. Khan FY: Rhabdomyolysis: A review of the literature. *Neth J Med*, 67 (9): 272-283, 2009.

3. Cervellin G, Comelli I, Lippi G: Rhabdomyolysis: Historical background, clinical, diagnostic and therapeutic features. *Clin Chem Lab Med*, 48 (6): 749-756, 2010. DOI: 10.1515/CCLM.2010.151

4. Elsayed EF, Reilly RF: Rhabdomyolysis: A review, with emphasis on the pediatric population. *Pediatr Nephrol,* 25 (1): 7-18, 2010. DOI: 10.1007/s00467-009-1223-9

5. Willamson JA, Kaelble M, Chisholm A: Acute necrotizing myopathy in a dog. *J Am Anim Hosp Assoc,* 47 (2): 112-116, 2011. DOI: 10.5326/JAAHA-MS-5389

6. Ettinger Sj, Feldman, Eccôté E: Textbook of Veterinary Internal Medicine Expert Consult. 8th ed., 96, Elsevier Saunders, USA, 2017.

7. Holahan ML, Littman MP, Hayes CL: Presumptive hepatotoxicity and rhabdomyolysis secondary to phenazopyridine toxicity in a dog. *J Vet Emerg Crit Care,* 20 (3): 352-358, 2010. DOI: 10.1111/j.1476-4431.2010.00541.x

8. Lechowski R, Sapierzyński R, Bonecka J, Ostrzeszewicz M: Acute rhabdomyolysis in a dog: case report. *Med Weter,* 73 (10): 675-678, 2017. DOI: 10.21521/mw.5765

9. Silverstein DC, Hoper K: Small Animal Critical Care Medicine. 2nd ed., 26, Elsevier Saunders, USA, 2015.

10. Côté E: Clinical Veterinary Advisor: Dogs and Cats. 3rd ed., 929-956, Elsevier Mosby, 2015.

11. McLean AS: Echocardiography in shock management. *Crit Care*, 20:275, 2016. DOI 10.1186/s13054-016-1401-7

12. Turgut K: Klinik Kedi ve Köpek Kardiyolojisi. Nobel Tıp Kitabevleri Tic. Ltd. Şti., İstanbul, 2017.

13. Tintinalli JE, Kelen GD, Stapczynski JS: Emergency medicine: A comprehensive study guide. 6th ed., 122, McGraw-Hill Inc, New York, USA, 2004.

14. Sauret JM, Marinides G, Wang GK: Rhabdomyolysis. *Am Fam Physician*, 65 (5): 907-912, 2002.

15. Antons KA, Williams CD, Baker SK, Philips PS: Clinical perspectives of statin-induced rhabdomyolysis. *Am J Med*, 119 (5): 400-409, 2006. DOI: 10.1016/j.amjmed.2006.02.007

16. Minnema BJ, Neligan PC, Quraishi NA, Fehlings MG, Prakash S: A case of occult compartment syndrome and nonresolving rhabdomyolysis. *J Gen Intern Med,* 23 (6): 871-874, 2008. DOI: 10.1007/s11606-008-0569-1

17. Joshi D, Kumar N, Rai A: Dermatomyositis presenting with rhabdomyolysis and acute renal failure; an uncommon manifestation. *Ann Indian Acad Neurol,* 12 (1): 45-47, 2009.

18. Ford RB, Litster A: Infectious diseases. **In,** Schaer M, Gaschen F (Eds): Clinical Medicine of the Dog and Cat. 3rd ed., 909-923, Taylor & Francis Group, LLC, London, UK, 2016.

19. Visweswaran P, Guntupalli J: Rhabdomyolysis. *Crit Care Clin,* 15 (2): 415-428. 1999. DOI: 10.1016/s0749-0704(05)70061-0

20. Gabow PA, Kaehny WD, Kelleher SP: The spectrum of rhabdomyolysis. *Medicine,* 61 (3): 141-152, 1982. DOI: 10.1097/00005792-198205000-00002

21. Poels PJE, Gabreels FJM: Rhabdomyolysis: A review of the literature. *Clin Neurol Neurosurg*, 95 (3): 175-192, 1993. DOI: 10.1016/0303-8467(93)90122-w