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Capture myopathy accompanied with severe enteritis in a female lion

Case Report

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ABSTRACT

A one-year-old female lion was presented with a history of lethargy, unability to stand, incoordination, unsteady, swaying gait and diarrhoea to Istanbul University Veterinary Faculty Internal Medicine Clinics. Anamnesis was followed by physical examination. The lion was immobilized with ketamine (Ketasol; Interhas) and xylazine hydrochloride combination (Rompun; Bayer, West Germany). Blood was collected from the cephalic vein for routine haematology and biochemistry analysis. The patient died during the night. A slight decrease in platelet account, a mild decrease in triglyceride level with a mild elevation in glucose and urea and a severe augmentation in aspartate aminotransferase (AST), lactate dehydrogenase (LDH) and creatine kinase (CK) levels were observed. Pathologically, cause of death was determined as dehydration and hypovolemia due to severe hemorrhagic chronic-atrophic enteritis. Anamnesis, biochemical data and clinical findings suggested also capture myopathy (CM). In this case study it is aimed to give detailed knowledge about diseases seen in both free-ranging and captive lions and to discuss the death of this female captive lion due to severe diarrhoea and CM.

Keywords: lion, free-ranging lion, captive lion, enteritis, capture myopathy

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Introduction

Capture myopathy (CM) is a non-infectious, shock-like hyper metabolic myopathic disease triggered by stress due to pursuit, capture, restraint and transportation of animals. The disease leading to serious morbidity and mortality may also exist as secondary to other diseases or environmental situtations (Paterson, 2007; Hamidieh et al., 2011). The condition, formerly named as muscular distrophy, white muscle diseas, capture disease, cramp, overstraining disease, leg paralysis, spastic paresis, stress myopathy, transport myopathy, incipient myopathy, degenerative polymyopathy, (idiopatic) muscle necrosis is referred as "capture myopathy", "exertional myopathy" or "exertional rhabdomyolysis" recently (Paterson, 2007; Fowler, 2011; Blumstein et al., 2015). The disease shares similarities with the myodegenerative disorders of domestic cattle, sheep, horses, swine as well as exertional rhabdomyolysis in humans. CM is reported in birds, wild African ruminants, antelopes, deers, Rocky mountain goats, Rocky mountain bighorn sheeps. CM is very similar to human exertional rhabdomyolysis which is one of eight forms of rhabdomyolysis, having hundreds of underlying causes (Paterson, 2007; Hamidieh et al., 2011). Exertional rhabdomyolysis/CM, being a multifactorial disease in animals differs from other similar conditions by its pathophysiology as it influences both cardiac and skeletal muscles. Predisposing factors are listed as "species", "signalement", "nutrition" and "drugs" (Paterson, 2007). The stress of capture causes an increase in energy metabolism. This augmentation helps an animal escape,

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but also cause a homeostatic imbalance which may damage physiological functions (Ganhao et al., 1988; Kock et al., 1987; Cattet et al., 2003). CM results from altered blood flow to the tissues and exhaustion of normal aerobic energy especially in skeletal muscle. Exhaustion of ATP in muscle cells ends by decreased rate of oxygen and nutritions delievered, an overproduction of lactic acid and failure to removal of cellular waste products. Lactic acid in the bloodstream drops the body pH; this affects cardiac output. Myoglobin released from muscle cells undergoing damage and necrosis, cause tubular necrosis in kidneys and acute renal failure. Similar process occurs also in cardiac tissue (Paterson, 2007; Fowler, 2011).

CM manifestation differs according to species, the individual, the tissue damage and the circumstances. Different classifications are explained but it is very important to remember that the disease is a continuum and animals may exhibit overlapping syndromes. The disease is defined by four syndromes cited as "capture schock syndrome" (CSS), "ataxic myoglobinuric syndrome" (AMS), "ruptured muscle syndrome" (RMS) and "delayed-peracute syndrome" (DPS). The first may occur either during capture or post-capture; death comes 1-6 hours after capture. Depression, hyperpnea, tachycardia, augmented body temperature, weak pulses are clinical symptoms. Elevations in aspartate aminotransferase (AST), lactatedehydrogenase LDH and creatine kinase (CK) exist. AMS is represented several hours or days post capture. Clinical signs are mild or severe ataxia, torticollis or myoglobinuria. AST, CK, LDH and blood urea nitrogen (BUN) elevation. RMS which doesn't manifest until 24-48 hours postcapture, is accompanied by a marked drop in hindquarters and hyperflexion of of the hock. Extreme elevations in AST, CK and LDH is noted while BUN is slightly or not elevated. In case DPS occurs, animals may seem healthy until they are disturbed; and when disturbed, they remain still and die due to ventricular fibrillation. Elevated AST, CK and LDH levels are expressed (Rose, 2005; Paterson, 2007). Liberation of huge amount of myoglobin into the bloodstream related to muscle damage causes myoglobinuric nephrosis. This myoglobin filtered by glomeruli is toxic for proximal tubules' and loop of Henle's epithelial cells. Ischemic damage and shock with acute tubular necrosis occur (Rose, 2005). The diagnosis is generally based on histologic findings (Blumstein et al., 2015).

Though treatment of CM is ineffective, cure with the use of analgesics, muscle relaxants (i.e. dantrolene), benzodiazepines, cortisone, multivitamines, hyperbaric oxygen application, sodium bicarbonate, vitamin E, selenium and fluid therapy may be performed (Paterson, 2007; Fowler, 2011). The treatment strategy is based on reverse the ongoing shock, metabolic acidose and stabilise cellular membranes (Rose, 2005).

The prevention of disease is important by minimal handling of wildlife, and the most appropriate transportation method, most appropriate anesthesic drug choice. The most important of all is understanding the behaviour and physiology of species (Paterson, 2007; Fowler, 2011; Hamidieh et al., 2011).

Differantiation from Takotsubo disease, CM which comes after metabolic acidosis due to lactic acid accumulation and typically affects muscle tissue while specifically referring to a stress-induced cardiac muscle effects only, CM in wildlife can be a model for human stress cardiomyopathy including Takotsubo/broken heart cardiomyopathy related to the surge of cathecholamines followed by left ventricular dysfunction, where emotional stress triggers heart attack-like synmptoms and sudden death in humans (Blumstein et al., 2015).

Long-living species appear to be more predisposed to CM; this is thought to be due to rapid response to autonomic response to threats. Apart from adaptations associated with reduced predation risk, sociality and larger brains are related to more complex autonomic systems. More social species have larger brains. Moreover, long-living species seem to be better adapted to avoid predation and more likely to be susceptible for CM. Thus, CM may be an unavoidable result of being social and long-living species. This comparative finding can be converted into human medicine and also be useful in understanding whether similar factors play part in similar human diseases (Blumstein et al., 2015).
Joubert and Stander (1990) report CM in an African lion severely stressed prior to immobilization in spite routine lion capture procedures involving also ketamine HCL and xylaxine HCL combination application was performed. The lion had died due to respiratory failure (Joubert and Stander, 1990).

Case Presentation

A one-year-old female lion, weighing approximately 100-150 kg, presented with a history of lethargy, incoordination, and diarrhoea for one week to Istanbul

University Internal Medicine Clinics. The lion originated by a private collection within Istanbul and previous medical history was not available. Parasite prophylaxy nor any vaccination was not performed. She was staying alone, in a room-like cage, and fed with liver, cut pieces of red meat containing bones, being or not uncooked. The lion was alert on physical examination. The patient was not anorexic. No blood was observed in the gaita.The lion was sedated with the use of ketamine (Ketasol; Interhas) andxylazine hydrochloride combination (Rompun; Bayer, West Germany) administered by hand injection for physical examination (Figure 1). Ketamine-xylazine combination

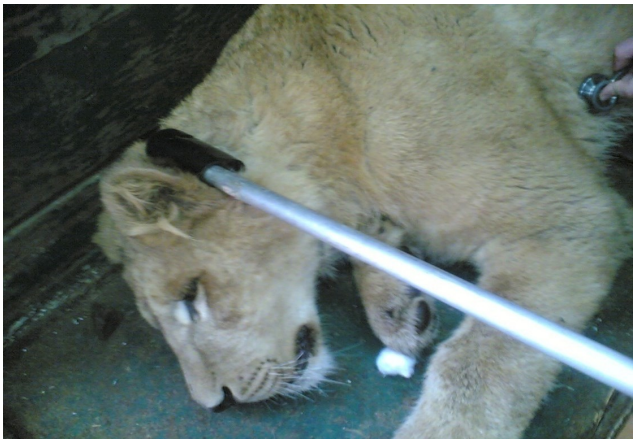

Figure-1. Lion when sedated, controlled with an examination bar; during general examination

dosage ranged between 3-10 mg/kg of ketamine and 0.3- 1-4 mg/kg of xylazine in large felids/wild lions (Currier and Russel, 1982; Melton et al., 1987; Gunkel and Lafortune, 2007; Larsson et al., 2015). The lion was fully sedated (lying down and unable to lift its head and/or consciously bite) after 15-20 minutes and, her eyes were covered with a serviette to keep calm, prevent damage and limit unnecessary excitement (Erasmus, 2008). Body temperature was between physiologic values. No mucosal colour alteration was noted. Abdominal sensitivity couldn't be evaluated since the patient was under general anestheisa. Neither abnormal heart sound nor pulmoner pathologic sound was detected.

Dermatologic inspection showed diffuse echymosis and petechiaes. 5-6 mL. of blood were collected from the cephalic vein for routine haematology and biochemistry analysis (Kaiser et al., 2014; Larsson et al. 2015). Gaita was also taken from rectum. The patient died during the night and death was not observed.

Pathologic Findings: A systemic necropsy was performed to the cub. The animal's body condition was low. Area around the tail was smudged with dark red matter. All organs in the body cavities observed congested. Foamy fluid inside the trachea detected. Liver surface was hackly and atrophy in the spleen was observed. Most remarkable finding was in the intestines. Intestinal walls were thickened, in the lumen of the organ there was a crimson red matter. Tissue samples from each organ collected, they were fixed in 10% formaldehyde overnight and embedded in paraffin. 3-4µ sections were cut and stained with hematoxylin-eosin (H&E) routinely. All sectioned were investigated under light microscope. In microscopic evaluations; distinct edema in lung with congestion determined. In some areas alveolar walls were thickened. Edema and peripheral fibrosis in liver was observed (Figure 2B). In paranchymatous cells extensive degenerative changes were detected. Apparent lymphoid atrophy and lymphoid necrosis were observed in spleen. Intestinal villi were atrophic and there was distinctive epithelial loss on their surfaces, number of the mucosal glands was diminished and there were small number of chronic inflammatory cells, erythrocytes in the lumen and between those glands (Figure 2C). In some areas the mucosa was totally atrophic without glands or villi (Figure 2D). Pathologically, cause of death was determined as dehydration and hypovolemia due to severe hemorrhagic chronic-atrophic enteritis.

Anamnesis, biochemical data and clinical findings suggested CM.

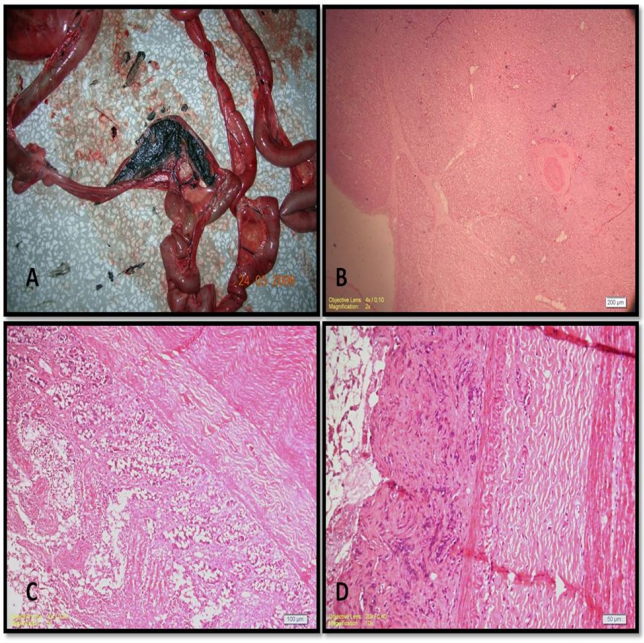

Figure-2. A. Crimson red matter in the intestines. **B**. Distinct peripheral fibrosis in liver. **C**. Atrophic villi, desquamative epithelial cell, erythrocytes, inflammatory cells in lumen and

Table 1: Reference values of haemogram resulted from various researches and measured value in this case.

		Reference values		
Parameters	Measured value	Erasmus, 2008	Maas et al., 2013	Larsson et al., 2015
RBC (1 x 10^{6}/ µl)	7.75	6.3 - 9.8	5.1 - 8.3 / 5.7 - 8.7 (males) 5.0 - 8.4 / 5.2 - 8.5 (females)	8.97
HGB (g/dl.)	11.2	5.4 - 20.5	8.9 - 14.6	14.11
HCT (%)	32	29.2 - 45.6	26.8 - 44.1	42.38
WBC (1 x 10^{3} / µl.)	16.7	-	7.2 - 25.6 / 9.7 - 28.2	9.37
PLT (1 x 10^{3}/ µl.)	246	355.41	-	-
MCV (fl)	42	43 - 50	46.6-55.9 (males) 48.0-56.7 (females)	47.70
MCH (pg)	14	9.6 - 22.4	14.8-18.5 (males) 15.5-19.1 (females)	15.84
MCHC (%)	35	-	-	33.33

Discussion

Despite no specific viral identification had been established, anamnesis, physical examination findings, and histopathology seem to be in concordance with literature.

In our case, a slight decrease in platelet count and a mild decrease in trigliseryde level were noted. On the other hand, a mild elevation in glucose (GLU) and urea accompanied a severe augmentation in AST, LDH and CK levels.

Apart from textbooks generally representing blood values of captive lions, Maas et al., explain hematologic and biochemical values for free-ranging and captive lions (Maas et al., 2013). In a study which aims to generate biochemical data, alanine aminotransferase (ALT), AST, alkaline phosphatase (ALP), albumin (ALB) calcium, creatinine (CRE), GLU, lactate dehydrogenase (LDH), phosphorus, total bilirubin (TB), total cholesterol, total protein (TP), triglyceride, urea and uric acid were estimated by standard laboratory methods and it was concluded that GLU, protein, urea and ALP varied significantly with sex and season (Behera et al., 2013).

GLU is the chief metabolic fuel for energy yield in carnivores. Its blood level depends on several factors such as type of diet and interaction of insulin, glucagon, epinephrine, thyroid, glucocorticoid, pituitary and sex hormones. The secretion of these hormones varies between season and sex. There is a positive co-relation between blood glucose level and temperature. Basal metabolic rate (BMR) is regulated by environmental temperature and sex. In summer season, significantly higher level of blood glucose in female lions is due to less utilization of glucose at low BMR and high temperature. The normal body temperature of female is more than that of male. More energy is required to maintain it in cold environment of winter season at low BMR. Here more glucose is spared for conservation of heat. Therefore female lions exhibit low blood glucose in winter season (Behera et al., 2013). Serum glucose levels were positively related to increasing group size (Melton et al., 1987). Elevation in glucose level during general anesthesia pursuing induction with ketamine as well as dose-dependent changes in glucose concentrations are reported in Bengal tigers immobilized with xylazine (Seal et al., 1987; Reilly et al. 2014). The elevation observed in this case was thought to be related to augmented metabolic rate during capture and/or ketamine use.

In lions, BUN is reported to be significantly affected by three variables; the main association is for nomadic animals to have elevated levels. BUN levels will rise within a normal range as the quality or quantity of protein ingested increases, abnormal elevation can occur if protein is catabolized during food shortage or owing to renal dysfunction. The results may also show protein catabolism following an inadequate diet (Melton et al., 1987). The elevation in this case might be due to an inadequate diet, a renal dysfunction beginningand/or CM; CM being the strongest possibility.

Intracellular liver specific serum enzymes like ALT, AST and ALP take part in cell metabolism and in different metabolic pathways. Generally their elevated

Table 1: Reference values of haemogram resulted from various researches and measured value in this case.

Parameters	Measured value	Reference values			
		Currier and Russel, 1982	Erasmus, 2008	Behara et al., 2013	Miller, 2015
Glucose (mg/dl)	191	135-154 (all)	-	77.61 ± 2.60 (M) 102.78 ± 5.52 (F)	132
Urea (mg/dl)	77	30-36.1	-	143.20 ± 5.69 (M) 133.79 ± 6.24 (F)	31
Creatinin (mg/dl)	1.3	2.46-3.07	-	1.60 ± 0.07 (M) 1.48 ± 0.09 (F)	-
AST (IU/L)	319	58-79 (wild) 40-47 (captive)	25.79	20.02 ± 2.20 (M) 18.29 ± 1.53(F)	-
ALT (IU/L	94	48-57 (all)	42.58	12.39 ± 1.35 (M) 13.09 ± 1.06(F)	32
ɣ-GT (IU/L)	4	-	1.78		-
T. Bilirubine (mg/dl)	0.3	0.2-0.3 (wild) 0.5-0.7 (captive)	-	0.56 ± 0.05 (M) 0.47 ± 0.07 (F)	0.3
LDH (IU/L)	1343	133-197 (wild) 128-153 (captive)	81.98	118.10± 14.92(M) 103.73± 11.35 (F)	-
CK (IU/L)	632	82-135 (wild) 46-78 (captive)	-	-	-
Ccholesterol (mg/dl)	189	148-185 (wild) 192-214 (captive)	-	145.23± 7.79(M) 150.32±12.8 (F)	-
Trigliserid (mg/dl)	31	-	-	85.23 ± 13.71 (M) 61.43 ± 11.67 (F)	-
T. Protein (g/dl)	6.9	7.1-7.3 (all)	-	7.86 ± 0.11 (M) 7.62 ± 0.23 (F)	7.5
Albumin (IU/L)	3.8	-	-	-	-

M: male , **F:** females

levels in serum is attributed to the alteration in architectural integrity of hepatocytes and muscle fibers (Behera et al., 2013).

ALT is found in high amounts in feline hepatocytes' cytoplasm. The enzyme enters the blood when liver cell damage occurs. In spite of indicating liver cell damage, this enzyme doesn't explain the cause or reversibilty of the damage; but only recent or ongoing hepatic cell alteration. An increase of three times may be seen as normal (Sodikoff, 2001).

AST which is a mitochondria-bound enzyme, is found in different body tissues, especially high values in liver and striated muscle. Augmented serum AST levels indicates skeletal muscle necrosis and/or hepatocellular necrosis (Sodikoff, 2001). In domestic cats, AST levels can go up to 100 IU/L. without being any pathologic cause (Currier and Russell, 1982). In lions, an elevated AST measurement in addition to BUN is evaluated as a result of tissue damage related to a disease state or perhaps trauma during capture (Melton et al., 1987). Serious elevation of AST accompanied by BUN augmentation in this case were in concordance with capture oriented trauma findings revealed in litterature, despite other BUN elevation causes must also be taken into consideration.

CK, found in high levels in the CNS and striated muscle, increases in muscle trauma, IM injection, myositis and occasionally CNS damage. Increased levels of both CK and AST mean muscle necrosis (Sodikoff, 2001). Since wild lions are subject to more strenuous physical activity before tranquilization, CK and LDH elevation is expected (Currier and Russell, 1982).

Moreover, since AST, LDH and CK levels gradually rise after muscle damage, a slight augmentation in these parameters may be related to time elapsed between tranquilizing and withdrawing blood. Another reason might be the injection procedure with the use hand-held syringe even this application causes less stress (Currier and Russell, 1982). In this case, serious elevation in AST accompanied by BUN, LDH and CK augmentation were in concordance with CM findings.

Hypotriglyseridemia is not specifically associated with any disease (Willard and Tvedten, 2012).

It is well known that this shock-like, hypermetabolic and myopathic disease may lead to mortality secondary to both other diseases and environmental factors; nutrition being one of them as a predispositive cause. The patient represented in this case had been living in inappropriate conditions for a wildlife animal. It is highlighted that stress of capture causes energy metabolism to accelerate, homeostatic and physiologic imbalance to occur. These changes end by acute renal failure which were supported by blood analysis findings in this case. It is known that death may occur 1 -6 hours or several hours after capture (Rose, 2005; Paterson, 2007; Blumstein et al., 2015).

According to what cited above, both clinical and biochemical findings seemed also to be in concordance with vitamin E deficiency strongly related to capture living and feeding conditions, in which case weakness, difficulty standing, muscle detoriation occur apart from augmentation of ALT, LDH and CPK levels (Bouts and Gasthuys, 2003).

General anesthesia influences different organ systems and may exacerbate various non-detected homeostatic imbalences. It is well known that depression of the CNS and cardiopulmonary system due to general anesthesia may end by decreased tissue perfusion, cellular metabolism and changes in the endocrine responses in cats and dogs. Despite possible physiologic changes related to general anesthesia remain unknown, similar changes may be expected during general anesthesia in lions. While no statistically significant difference was noted during anesthesia in CBC, BUN, creatinine, CK, globulins, ionized calcium, sodium, chloride, or lactate concentrations for any animal, most important clinical changes were simultaneous increase in plasma potassium and glucose concentrations with a decrease in initial plasma insulin concentration(Reilly et al., 2014). Furthermore, sudden death due to respiratory failure after ketamine HCL and xylaxine HCL combination application exists in a report (Joubert and Stander, 1990). According to the results, minimizing the duration of anesthesia, continous monitoring of clinical and biochemical changes during general anesthesia and evaluation of the effects of anesthesic drugs, drug comnbinations, a very careful evaluation of biochemical parameters and clinical situation in large wild felids is warranted (Reilly et al., 2014).

According to researchers, no significant difference exists between mortality rate of wild-born animals and captive-born animals and excess mortality of wild-born animals brought to zoo doesn't seem to be a general phenomenon. Yet, the mortality rate and situation for wild species is expected to differ between animals living in wild and captivity. In spite of researchers explaining the benefits of captive-living conditions such as veterinary care, lack of predators, regular food supply, the risk of obesity, injuries from exhibits, poor adaptation to captivity conditions and to zoo's climate as well as higher perinatal mortality rate because of inbreeding must also be highlighted. Another risk is the facilitation of infection spreading due to close quarters. Even those who argue that no difference exists between free-living and captive living, are highlighting that the error of commentary occurs when birth date or origin of animals were not recorded. Reports explain excess mortality when brought into a zoo relative to animals born and reared in a zoo, beacuse of emotional trauma and exposure to new diets (Kohler et al., 2006).

Conclusion

As a result, it is believed that the lion had severe complaints due to severe hemorrhagic chronic-atrophic enteritis which played a very important role in sudden death. On the other hand very inappropriate living conditions including environmental and nutritional factors must have been efficient in both enteritis and CM to occur. CM must have been the final cause of sudden death.

Declined population of wild fauna, extensive deforestation, destructive man made activities, nutritional deficiency, metabolic and infectious diseasesmay result in extinction of many species in near future. Environmental and genetic mutations of pathogens also adversely affect the population growth (Behera et al., 2013). To prevent animal species from extinction, augmented aid to breeding programs, exchange between institutions, greater support for veterinary care, research programs and conservation are supported. Yet the difficulty in detecting signs of diseases in captive animals remains problematic. Especially keeping individuals requiring large

territories under artifical conditions is necessary (Larsson et al., 2015).

Despite it is very important to establish serious attempts to prevent wildlife species extintions; these serious attempts should definitely performed by conscious welfare, healthcare and similar other professionals which have enough scientific knowledge to handle both living and health needs and conditions of such animals. Beyond being a medical obligation, this condition seems to be a very improtant ethical one.

References

Behera, P. C., Dash, D., Senapati, M. R., Bisoi, P. C., & Parija, S. C. (2013). Effect of sex and season on serum biochemical constituents and enzymes of Panthera leo of Nandankanan Zoological Park. *IOSR-Journal of Agriculture and Veterinary Science, 4*(2), 68-72.

Blumstein, D. T., Buckner, J., Shah, S., Patel, S., Alfaro, M. E., & Natterson-Horowitz, B. (2015). The evolution of capture myopathy in hooved mammals: A Model for human stress cardiomyopathy? *Evolution, Medicine, and Public Health, 1*, 195-203.

Bouts, T., & Gasthuys, F. (2003). The importence of vitamin E in zoo mammals. *Vlaams Diergeneeskundig Tijdschrift, 72*, 125-129.

Cattet, M. R. L., Christison, K., Caulkett, N. A., & Stenhouse, G. B. (2003). Physiological responses of Grizzly Bears to different methods of capture. *Journal of Wildlife Diseases, 39*(3), 649-654.

Currier, M. J., & Russel, K. R. (1982). Hematology and blood chemistry of the mountain lion (Felis concolor). *Journal of Wildlife Diseases, 18*(1), 99-104.

Erasmus, H. L. (2008). Determination of some blood parameters in the African lion (Panthera leo). *Magister Scientiae Agriculturae thesis*, University of the Free State, Bloemfontein, South Africa.

Fowler, A. (2011). Capture Myopathy. Retrieved from http:// www.fourthcrossingwildlife.com/CaptureMyopathy-AnneFowler.pdf

Ganhao, M. F., Hattingh, J., & Pitts, N. (1988). Physiological responses of blesbok, eland and red hartebeest to different capture methods. *South African Journal of wildlife Research*, *18*(4),134-136.

Gunkel, C., & Lafortune, M. (2007). Felids. In G. West, D. Heard, N. Caulkett (Ed). *Zoo animal and wildlife immobilization and anesthesia. 2nd ed.* (pp. 443-458). Ames Iowa, US: Blackwell Publishing.

Hamidieh, H., Alhami, A.. & Mirian, J. (2011). Capture myopathy in red deer and wild goat. *Archives of Razi Institute, 66*, 147-149.

Joubert, F. G., & Stander, P. E. (1990). Capture myopathy in an African Lion. *Madoqua, 17*(1), 51-52.

Kaiser, C., Wernery, U., Kinne, J., Marker, L., & Liesegang, A. (2014). The role of copper and vitamin A deficiencies leading to neurological signs in captive cheetahs (Acinonyx jubatus) and lions (Panthera leo) in the United Arab Emirates. *Food and Nutrition Sciences, 5*, 1978-1990.

Kock, M. D., Jessup, D. A., Clark, R., & Franti, C. E. (1987). Effects of capture on biological parameters in free-ranging bighorn sheep (Ovis canadensis): evaluation of drop-net, drive-net, chemical immobilization and the net-gun. *Journal of Wildlife Diseases, 23*(4), 641-651.

Kohler, I. V., Preston, S. H., & Lackey, L. B. (2006). Comparative mortality levels among selected species of captive animals. *Demographic Research, 15*(14), 414-434.

Larsson, M. H. M. A., do Espírito Santo, P. L., Mirandola, R. M. S., Fedullo, J. D. L., Ito, F. H., Itikawa, P. H., & Pessoa, R. B. (2015). Hematologic parameters of captive lions (Panthera leo) and Siberian tigers (Panthera tigris altaica). *Acta Scientiae Veterinariae, 43*, 1311.

Maas, M., Keet, D. F., & Nielen, M. (2013). Hematologic and serum chemistry reference intervals for free-ranging lions (Panthera leo). *Research in Veterinary Science, 95*(1), 266-268.

Melton, D. A., Berry, H. H., Berry, C. U. & Joubert, S. M. (1987). Aspects of the blood chemistry of wild lions, Panthera leo. *South African Journal of Zoology, 22*(1), 40-44.

Miller, M. (2015). Tuberculosis In Free-Ranging African Lions. A Case Report From Krugert National Park , South Africa. *Vetcom.* Retrived from. https://vet.abaxis.co.uk/wp-content/uploads/2015/01/WW-TB-in-free-ranging-African-Lions.pdf

Paterson, J. (2007). Capture myopathy. In G. West, D. Heard, N. Caulkett (Ed). *Zoo animal and wildlife immobilization and anesthesia. 2nd ed.* (pp. 171-179). Ames Iowa, US: Blackwell Publishing.

Reilly, S., Seddighi, R. M., Steeil, J. C., Sura, P., Whittemore, J. C., Gompf, R. E., Elliott, S. B., & Ramsay, E. C. (2014). Selected clinical, biochemical, and electrolyte alterations in anesthetized captive tigers (Panthera tigris) and lions (Panthera leo). *Journal of Zoo and Wildlife Medicine, 45*(2), 328-334.

Rose, K. (2005). Common diseases of urban wildlife – myopathy and trauma. Retrieved from http://arwh.org/ sites/default/files/2016-11/Common%20Diseases 20General_with_images.pdf

Seal, U. S., Armstrong, D. L., & Simmons, L. G. (1987). Yohimbine hydrochloride reversal of ketamine hydrochloride and xylazine hydrochloride immobilization of Bengal tigers and effects on hematology and serum chemistries. *Journal of Wildlife Disease, 23*(2), 296–300.

Sodikoff, C. H. (2001). *Laboratory profiles of small animal diseases*. St Louis, US: Mosby Inc.

Willard, M. D. & Tvedten, H. (2012). *Small Animal Clinical Diagnosis by Laboratory Methods, 5th Ed*. St Louis, US: W.B. Saunders. anging-African-Lions.pdf. 52: 40-42.