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SHORT COMMUNICATION

CLINICAL AND PATHOLOGICAL FINDINGS IN A DWARF RED BROCKET *MAZAMA RUFINA* (MAMMALIA: CETARTIODACTYLA: CERVIDAE) ATTACKED BY DOGS

Eduardo Alfonso Díaz, Gustavo Donoso, Carolina Sáenz, Ivette Dueñas & Francisco Cabrera

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Clinical and pathological findings in a Dwarf Red Brocket *Mazama rufina* (Mammalia: Cetartiodactyla: Cervidae) attacked by dogs

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Abstract: Capture myopathy is a common fatal syndrome in wild ungulates resulting from anthropogenic stressful events such as the capture or transport of specimens. There are, however, few published data on this issue due to predator attacks. The present report describes for the first time the capture myopathy syndrome in a Dwarf Red Brocket *Mazama rufina* following dog *Canis familiaris* attack. Clinical signs included pale mucous with increase capillary refill time, tachycardia, tachypnea, hypertension, hypothermia, hypoglycemia, and red brown urine. Muscle tremors, ataxia, prostration, paralysis, and opisthotonus were progressively observed. Laboratory tests showed increased levels of cortisol, creatinine, creatine kinase, lactate dehydrogenase, and potassium; decreased blood urea nitrogen-creatinine ratio; and myoglobinuria. The animal died 72 hours after hospital admission. At necropsy, findings included injuries on both hindlimbs with edema, emphysema, and soft-friable texture in affected muscles, dark kidneys and brown urine in bladder. Histopathological exams were indicative of skeletal-cardiac muscle degenerative lesions and myoglobinuric nephrosis. Immuno-histochemistry revealed myoglobin depletion in degenerate muscles and myoglobin accumulation in renal tissues. We strongly recommend that treatment for capture myopathy be initiated when a wild ungulate is admitted with history of predator attack, since the syndrome may have already established. This report adds to the instances of negative impacts caused by domestic dogs on threatened wildlife species.

Keywords: Capture myopathy, exertional rhabdomyolysis, myoglobin depletion, myoglobinuric nephrosis, predator attacks, wild ungulates

The Dwarf Red Brocket *Mazama rufina* is a member of the order Cetartiodactyla, family Cervidae, native from montane forest and paramos at altitudes between 1,500–3,500 m of Colombia, Ecuador and Peru. Currently, however, the species is restricted to remnant forest patches and paramos, and population trend is decreasing. The IUCN Red List of Threatened species classifies it as Vulnerable due to habitat destruction, hunting and predation, including domestic dog *Canis familiaris* attacks (Lizcano & Alvarez 2016).

Domestic dogs are considered a potential threat to 15 threatened species of the order Cetartiodactyla, including the Dwarf Red Brocket *Mazama rufina* (Doherty et al. 2017). Specifically, in Ecuador, Zapata-Ríos & Branch (2016) have documented decreased abundance and altered activity patterns of this species where dogs were present. The real impacts of dogs on native species, however, are not currently recognized in Ecuador, and studies that identify threats to wildlife are needed (Zapata-Ríos & Branch 2018).

Capture myopathy, or exertional rhabdomyolysis,

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is a potentially fatal syndrome commonly documented in wild ungulates (Paterson 2014). The existing reports mainly describe cases attributed to stressful events such as captures or transport of specimens (Montané et al. 2002b; Hamidieh et al. 2011; Nuvoli et al. 2014; Zahid et al. 2018). Although interactions with domestic dogs contribute substantially to admissions of wild mammals at rescue centers around the world (Wimberger & Downs 2010; Loyd et al. 2017; Romero et al. 2019; Taylor-Brown et al. 2019), as far as we know, there are no published data on capture myopathy triggered by dog attacks.

This report describes the diagnosis of capture myopathy in a Dwarf Red Brocket attacked by dogs in an Ecuadorian mountain forest, based on the history, clinical signs, laboratory tests, gross necropsy, histopathology, and immuno-histochemistry findings.

CASE HISTORY

On 20 January 2019, an adult male Dwarf Red Brocket was found in a state of shock after dog attacks in a mountain forest of Pichincha, Ecuador. The specimen was transported to the wildlife hospital of the Universidad San Francisco de Quito (USFQ) for evaluation. No chemical restraint was required for the clinical examination as the patient was depressed. Blood and urine samples were collected for laboratory analysis.

Initial treatment included oxygen via facemask and fluid therapy with warmed lactated Ringer's solution (3.5ml/kg/hour) through a catheter placed in the cephalic vein. Additionally, a bolus of dextrose 50% was administered (1ml/kg intravenous once). Antibiotic treatment was also initiated with ceftriaxone (25mg/

kg intravenous twice a day) and metronidazole (15 mg/kg intravenous once a day). For analgesia, meloxicam (0.2 mg/kg intravenous once a day) was used. We also injected a prokinetic, metoclopramide (0.1 mg/kg intravenous once a day) and supplementation with selenium, phosphorus, and vitamins A, D & E (0.02ml/kg subcutaneous once) were administered. The patient was then transferred to an isolation room to monitor the recovery. The patient died 72 hours after admission.

A complete necropsy was performed two hours later. Samples from the affected as well as apparently healthy muscles, heart, and kidneys were collected, fixed in 10% neutral buffered formalin. After seven days, we embedded the specimen in paraffin wax, and sections of 4µm were cut to be stained with hematoxylin and eosin for routine histopathology evaluation. In addition, immunohistochemical examination was performed on skeletal and cardiac muscles, and renal tissue sections using the protocol described by Herráez et al. (2013).

CLINICAL RESULTS

Upon admission, the Dwarf Red Brocket presented multiple penetrating fresh wounds in its hindlimbs muscles: gluteus, quadriceps, semimembranosus, and semitendinosus. Clinical examination revealed pale mucous membranes, increased capillary refill time, tachycardia, tachypnea, hypertension, hypothermia and hypoglycemia. Laboratory tests revealed increased levels of cortisol, creatinine, creatine kinase, lactate dehydrogenase, potassium, decreased blood-urea/nitrogen-creatinine ratio, and myoglobinuria (Table 1).

Muscle tremors, ataxia, prostration, paralysis and opisthotonus were progressively documented in the

Table 1. Biochemical values of a *Mazama rufina* (post dog attack blood samples). Normal range values for *Mazama temama* were obtained from the Zoological Information Management System database (Species 360 2018), with the exception of cortisol, which pertains to *Mazama gouazoubira* (Munerato et al. 2010).

Biochemical parameter	First sample (12h post-admission)	Second sample (72h post-admission)	Reference values ^a
AST (U/L)	3.9	7.8	47–174
BUN (mmol/L)	5.41	5.32	4.5–13.5
BUN/C (ratio)	6.3	1.2	16.6 (M)
CRE (µmol/L)	214.2	1109.7	53–184
CK (U/L)	1870	1085	72–725
K (mmol/L)	4.64	12.87	3.3–7.1
LDH (U/L)	ND	29818	366 (M)
COR (ng/ml)	73.2	25.69	10.04

AST—aspartate-aminotransferase | BUN—blood-urea-nitrogen | BUN/C—blood-urea nitrogen/creatinine | COR—cortisol | CRE—creatinine | CK—creatine-kinase | K—potassium | LDH—lactate-dehydrogenase | M—mean | ND—not determined. ^a There are no published reference values for *Mazama rufina*, reference from *Mazama temama* and *Mazama gouazoubira* were used for this reason

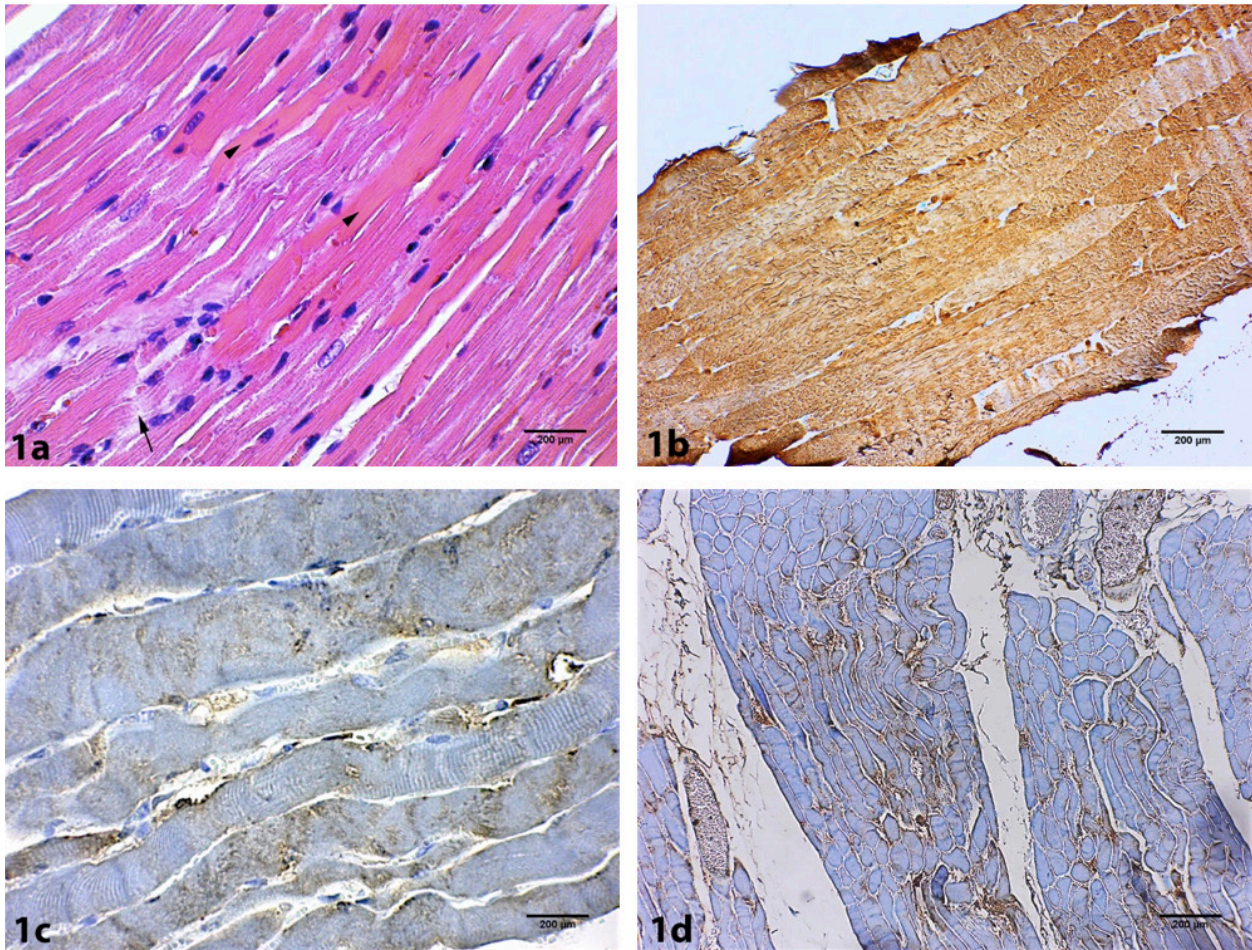


Image 1. Skeletal muscle: a—acute degenerative changes with loss of striation (arrowhead) and cytoplasmic fragmentation (arrow) (hematoxylin and eosin method) | b—positive control for myoglobin. Note the strong immuno-reaction and normal distribution in the tissue (avidin-biotin-peroxidase method) | c—depletion of myoglobin in the skeletal fibers and degeneration. Depletion denoted by the weak immuno-reaction (avidin-biotin-peroxidase method) | d—almost complete depletion of myoglobin in the affected tissue of *Mazama rufina* compared with the positive control (b). Accumulation of myoglobin is present in the extracellular space (avidin-biotin-peroxidase method).

patient. During the gross post-mortem examination, multiple lacerations and puncture injuries were observed macroscopically over the gluteus, quadriceps, semimembranosus, and semitendinosus muscles. The affected muscles were edematous and emphysematous with soft friable texture, but no gross lesions were evident in other muscles. Kidneys appeared swollen and dark, and the bladder contained brown urine. Microscopically, both affected and apparently normal muscles showed signs of degeneration (Image 1). Aseptic myositis and severe necrosis were seen in the skeletal muscles. Myocardial degeneration adjacent to the pericardium was also observed (Image 2). Kidneys showed signs of tubular and glomerular degeneration and necrosis, with intra-tubular and glomerular accumulation of eosinophilic proteinaceous material (Image 3). The immuno-histochemistry exams corroborated depletion

of myoglobin in degenerated muscles (Image 1) and presence of intra-tubular and glomerular myoglobin where eosinophilic proteinaceous material was present (Image 3).

DISCUSSION

The clinical signs detected in the present case are in agreement with those described as characteristic of capture myopathy in wild ungulates by Paterson (2014); however, although hyperthermia and hyperglycemia have been described after capture in White-tailed Deer *Odocoileus virginianus* (Boesch et al. 2011), our patient showed hypothermia and hypoglycemia upon examination. Decrease in body temperature and blood glucose has been documented during transport (Montané et al. 2002a) and one hour after capture (Montané et al. 2007) in Roe Deer *Capreolus*

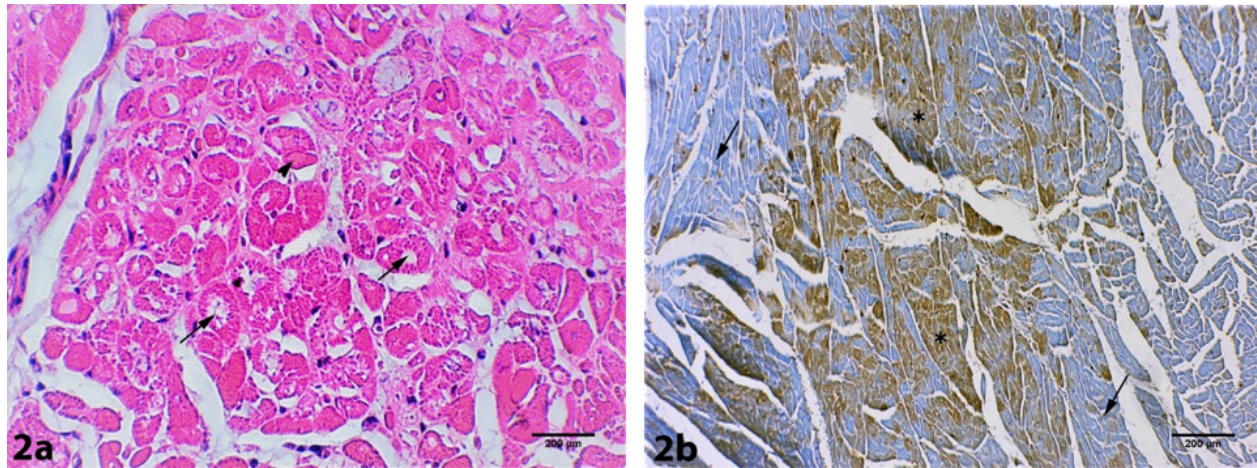


Image 2. Heart: a—myocardial degeneration adjacent to the pericardium, with perinuclear vacuolation (arrows). Note some fibers are hypereosinophilic and others are pale, necrosis is also seen (arrowhead) (hematoxylin and eosin method) | b—less injured fibers (asterisks) have a homogenous reaction for myoglobin, while injured fibers do not react to myoglobin (arrows) (avidin-biotin-peroxidase method).

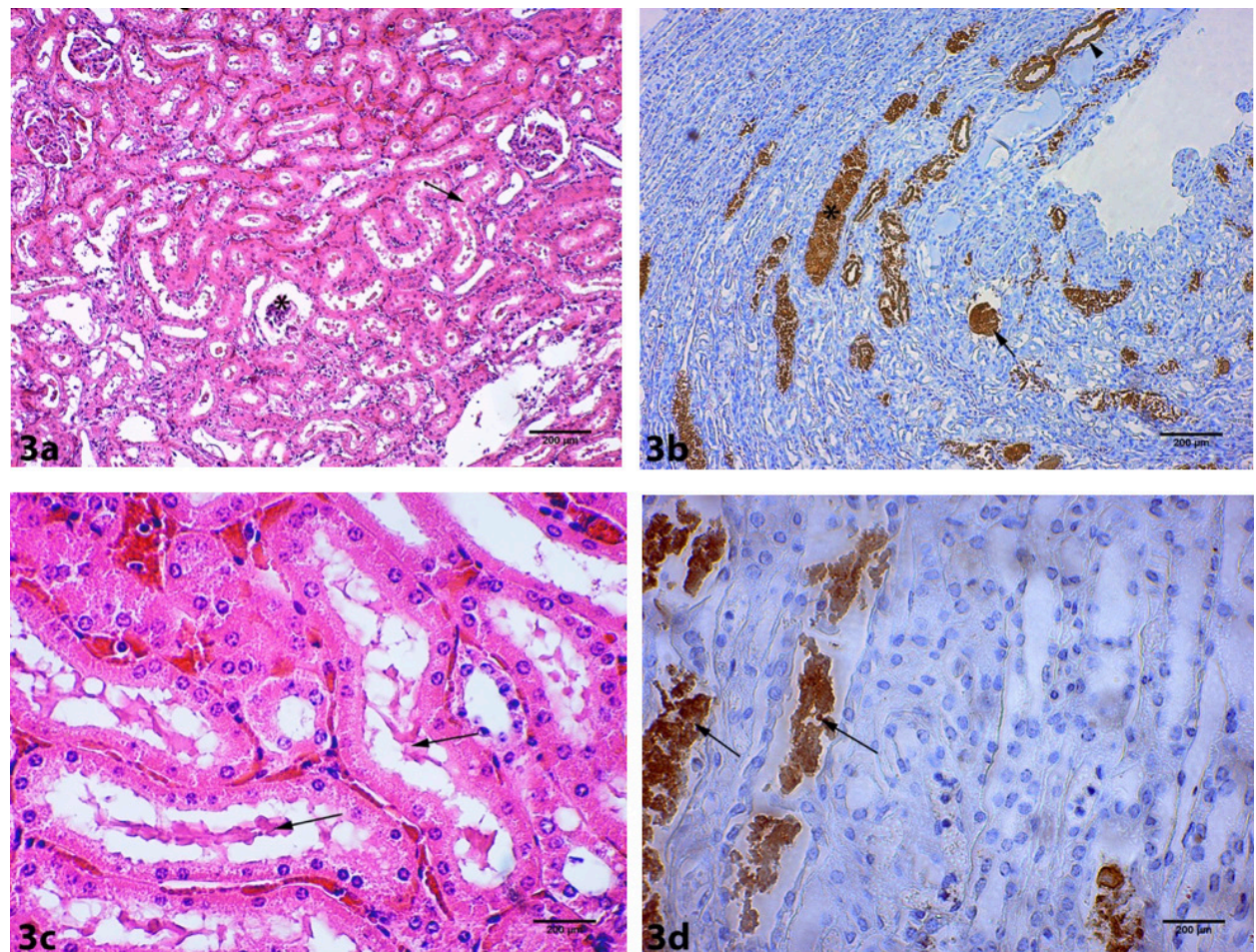


Image 3. Kidney: a—swelling, congestion and degenerative changes with necrosis of the glomerulus (asterisk) and accumulation of eosinophilic proteinaceous material (arrow) (hematoxylin and eosin method) | b—strong immuno-reaction for myoglobin in veins (asterisk), glomerulus (arrow) and tubules (arrowhead) (avidin-biotin-peroxidase method) | c—accumulation of eosinophilic proteinaceous material in the tubules (arrows) (hematoxylin and eosin method) | d—immuno-reaction for myoglobin in the tubules (arrows), where the eosinophilic proteinaceous material was present in (c) (avidin-biotin-peroxidase method).

capreolus. The Dwarf Red Brocket was found just after the dog attacks but the transport to the hospital took approximately three hours, therefore, the low values could be due to the delayed measurement. In fact, 24 hours later the patient reached normal levels of body temperature and blood glucose.

Capture myopathy is characterized by muscle injury resulting in release of myofiber content into the bloodstream. The diagnosis is based primarily on the findings of elevated serum creatine kinase levels and myoglobinuria (Nance & Mammen 2015). The early increase in serum potassium (Bagley et al. 2007) and creatinine (Zimmerman & Shen 2013) levels are also a consequence of muscle injury. These alterations have been described in wild ungulates and are consistent with our findings (Montané et al. 2002b, Nuvoli et al. 2014).

Muscle damage in wild ungulates is also responsible for increase in enzymes lactate dehydrogenase and aspartate aminotransferase (Montané et al. 2007; Casas-Díaz et al. 2010). The patient's laboratory tests corroborated the increase in lactate dehydrogenase, but not that of aspartate aminotransferase. In this context, metronidazole can cause a false decrease in readings of aspartate aminotransferase when ultraviolet absorbance measurement is used (Plumb 2008). The Dwarf Red Brocket received metronidazole as antibiotic, and aspartate aminotransferase concentration was measured by ultraviolet absorbance (Chemray 120 Vet, Rayto Life and Analytical Sciences, Shenzhen 518107, China), which could explain the low aspartate aminotransferase values.

On the other hand, increase in serum cortisol concentration has also been observed in wild ungulates subject to stressful management events (Arzamendia et al. 2010; Carmanchahi et al. 2011; Nuvoli et al. 2014). There are no cortisol values reported in a Dwarf Red Brocket, but the values detected in the present report are higher than previously reported for another species in this genus (Munerato et al. 2010), corroborating that serum cortisol can be used as a stress indicator in the species.

Finally, macroscopic, histopathological and immunohistochemical findings of muscles and kidney necrosis have been previously described by Nuvoli et al. (2014) in Red Deer *Cervus elaphus*, and suggest an acute renal failure associated with myoglobinuric nephrosis as a consequence of capture myopathy.

We have described for the first time a fatal myopathy syndrome in a Dwarf Red Brocket attacked by dogs based on history, physical examination, clinical signs, laboratory tests, gross necropsy, histopathology

and immuno-histochemical findings. We recommend that capture myopathy should be considered as a complicating factor in the diagnosis and treatment of wild ungulates after predatory attacks. This report also adds to the list of negative impacts on wildlife caused by domestic dogs.

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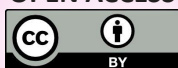
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