Gousiekte in African buffalo (Syncerus caffer)

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ABSTRACT

Three African buffalo (*Syncerus caffer*) that died after capture and translocation from Mutirikwe Recreational Park in southern Zimbabwe showed macroscopic and microscopic lesions of cardiomyopathy compatible with a diagnosis of gousiekte. The buffalo had had access to *Pavetta schumanniana*, a plant that is known to cause gousiekte. Death was attributed to cardiac failure as a result of previous consumption of the plant, exacerbated by the stress of translocation.

Keywords: African buffalo, cardiomyopathy, gousiekte, Mutirikwe Recreational Park, Zimbabwe, *Pavetta schumanniana*, *Syncerus caffer*.

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INTRODUCTION

Three African buffalo (Syncerus caffer) that died after capture and translocation showed macroscopic and microscopic lesions of cardiomyopathy. The commonest cause of cardiomyopathy in freeranging ruminants in southern Africa is gousiekte, a sub-acute to chronic intoxication by members of the plant family Rubiaceae that contain the polyamine pavetamine⁴. The previous assumption that the myocardial lesions described in the literature are pathognomonic has been discarded⁵, but a diagnosis of gousiekte is justified in cases of cardiomyopathy where one of the plants known to cause the condition is present. This paper describes an outbreak in Mutirikwe Recreational Park in Zimbabwe and considers possible epidemiological factors responsible for the outbreak.

MUTIRIKWE RECREATIONAL PARK

Mutirikwe (Kyle) Recreational Park in southern Zimbabwe (20°10′S, 31°01′E) includes a wildlife area of approximately 3800 ha enclosed by Lake Mutirikwe and a game-proof fence. In the north of the Park the Beza Range rises to a height of 1485 m. Between this range and Lake Mutirikwe is an undulating plain averaging 1070 m above sea level. The plain terminates in an area of broken hills in the south of the Park between the 2 arms of

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the lake. The Park is heavily stocked with more than 25 species of wild mammals, including 3 large grazing species, namely African buffalo, white rhinoceros (Ceratotherium simum) and hippopotamus (Hippopotamus amphibius). The buffalo were introduced as a foot-and-mouth diseasefree breeding herd in the late 1970s. The vegetation in the park is predominantly miombo woodland (Brachystegia spiciformis and Julbernadia globiflora), with thickets dominated by Peltophorum africanum, Terminalia sericea, Combretum spp. and Acacia karoo, small patches of evergreen riverine species and areas of middle veld grassland. A barren zone along the lake shore, which is subject to periodic flooding, supports only a sparse cover of annual herbs and grasses.

CASE HISTORIES

Twenty-seven buffalo were captured on 31 May 2007 in the Park and loaded in trucks for translocation to Mazowe, north of Harare. Two died in the truck the 1st night before they were transported. Post mortem examination of 1 revealed subacute suppurative pericarditis and pleurisy of undetermined aetiology; the other was not examined. The remaining 25 were unloaded at Mazowe on 2 June; 3 from a single truck died over the next 2 days. Post mortem examinations were performed by a layman on 2 cows (Cases B and C), and specimens in formalin were submitted to the Wildlife Unit of the Department of Veterinary Services in Harare. The last animal to die, a heifer (Case A), was examined post mortem by one of the authors (CMF). Histological

sections from the 3 animals were examined by all 3 authors.

Pathology

Case A

This animal was a 2-tooth heifer, 2-3 years old, in very good condition. The lungs were severely congested and oedematous, with froth mixed with rumen contents in the trachea and bronchi. The liver was slightly swollen and congested, the kidneys were slightly swollen and pale, the spleen was swollen with prominent lymphoid follicles. There were approximately 500 ml of slightly turbid, brownish fluid in the abdominal cavity. The myocardium was pale, with the cut surface slightly mottled and there were 50 ml of clear, yellow fluid containing fibrin clots in the pericardial sac. The mucosa of the small intestine was very congested and haemorrhagic. There was superficial bruising of the lower limbs, tearing of the fascia in the right axilla and prominent gelatinous oedema of the neck, especially adjacent to the trachea, probably resulting from trauma during capture and transportation. The superficial lymph nodes were moderately swollen.

Microscopic examination of the myocardium revealed marked variation in the width of the myofibres, indicating both hypertrophy and atrophy. The myofibre nuclei were hypertrophic and sometimes arranged in chains. There were numerous small foci in which the myofibres had disintegrated and been replaced by fibrosis of varying degrees of maturity. Oedema spaces and lymphocyte/macrophage infiltration were variably present between fibres. Significant features in other organs were marked pulmonary congestion and protein-rich oedema, marked hepatic congestion, congestion of the small intestine mucosa and protein in the glomeruli and tubules of the kidney.

Case B

Putrefaction in the myocardium was advanced. There was no evidence of disintegration of myofibres or fibrosis, but hypertrophy and atrophy of adjacent fibres were present in some areas.

Case C

Microscopic myocardial lesions were similar to those in Case A.

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In all 3 animals there was evidence of mild degeneration in skeletal muscle affecting less than 5 % of fibres, characterised by variation in width, loss of striation and occasional fragmentation of sarcoplasm, with a light interstitial macrophage infiltration. These lesions were attributed to the effects of capture and transport but were not considered clinically significant.

Diagnosis

The pathological changes in the animals that died were those of cardiomyopathy in the early stages in Case B and more advanced in Cases A and C. The history of sudden death after being subjected to the stress of capture and transport were characteristic of gousiekte in domestic ruminants. Of the plants known to cause gousiekte, Pavetta schumanniana occurs widely in the area. The plant is a slender shrub or small tree that has a preferred habitat in woodland in association with granite koppies and outcrops², such as occurs in parts of the Park that were frequented by the buffalo. The plant is known to be browsed by cattle on occasion.

DISCUSSION

The combination of death from cardiac failure after stress, lesions of subacute cardiomyopathy and the presence of a known toxic plant in the area where the buffalo grazed is sufficient evidence for a diagnosis of gousiekte. Cases A and C were 'typical' of the disease, while Case B was 'atypical'⁵. Degenerative changes in skeletal muscle were much less severe than in myocardium and of so limited extent as to rule out an alternative diagnosis of capture myopathy.

The unusual feature is the occurrence of plant toxicosis in free-living wildlife in a location in which they had lived for several generations. It is generally assumed that animals in such situations become adapted to their environment and either learn to avoid or develop the ability to degrade or detoxicate potentially lethal toxic plants³. For example, plant toxicosis does not feature as a cause of mass die-offs in large mammals in a 1994 review⁶. Nevertheless, plant poisoning may occur in situations of shortage of food, which make avoidance difficult and reduce the capacity for degradation and detoxication3.

Gousiekte in wildlife has been reported on only 1 previous occasion¹. On that occasion springbok (*Antidorcas marsupialis*) were confined to overgrazed camps in which there was very little vegetation. When the gousiekte plant, *Pachystigma pygmaeum*, sprang up after the first rains, the animals ate it for want of any other green feed and succumbed (T W Naudé, University of Pretoria, pers. comm. 2009).

No such obvious precipitating factor was present in the outbreak described in this paper, and the following explanation for the outbreak is speculative. The capture operative observed that, rather than frequenting the less wooded areas of the Park as in previous years, the buffalo spent most of their time in 2007 in the densely wooded areas amongst the granite outcrops, which is where the toxic plant is most common (C van der Riet, Harare, pers. comm. 2009). Possible reasons for this are 2-fold. One is the competition for grazing in the more open veld by white rhino and, at night, hippopotamus. The other is that the buffalo may have been sheltering in the dense woodland to

escape unauthorised hunting, which was suspected to be occurring in the Park at the time. For whatever reason, it appears likely that the buffalo were forced into a habitat with which they were not familiar and ate the plant because they failed to recognise it and because of shortage of other, more familiar, food. The reason for the fatalities being confined to animals in the same truck is also unclear. The herd bull was in that truck, and he may have caused more disturbance during transport than the animals in the other trucks.

There is no way of knowing whether gousiekte had occurred previously on a smaller scale in buffalo in the park, or in any other species, or what proportion of the animals that survived this incident had subclinical lesions from which they recovered. Veterinarians and wildlife management specialists need, however, to be aware of the possible occurrence of this disease in animals under their care in situations where grazing is scarce.

REFERENCES

- 1. Basson P A 1987 Poisoning of wildlife in southern Africa. *Journal of the South African Veterinary Association* 58: 219–228
- Coates Palgrave K 2002 Trees of Southern Africa (3rd edn) (Revised by Coates Palgrave M). Struik Publishers, Cape Town
- 3. Fowler, M E 1983 Plant poisoning in freeliving wild animals: a review. *Journal of Wildlife Diseases* 19: 34–43
- 4. Kellerman T S, Coetzer J A W, Naude T W, Botha C J 2005 Plant poisonings and mycotoxicoses of livestock in southern Africa (2nd edn). Oxford University Press, Cape Town
- Prozesky L 2008 A study of the pathology and pathogenesis of myocardial lesions in gousiekte, a cardiotoxicosis of ruminants. PhD thesis, University of Pretoria, Pretoria
- 6. Young T P 1994 Natural die-offs of large mammals: implications for conservation. *Conservation Biology* 8: 410–418