

MORTALITY OF A WHITE RHINOCEROS (*CERATOTHERIUM SIMUM*) SUSPECTED TO BE ASSOCIATED WITH THE BLUE-GREEN ALGA *MICROCYSTIS AERUGINOSA*

M.D. SOLL* and M.C. WILLIAMS**

ABSTRACT: Soll M.D.; Williams M.C. **Mortality of a white rhinoceros (*Ceratotherium simum*) suspected to be associated with the blue-green alga *Microcystis aeruginosa*.** *Journal of the South African Veterinary Association* (1985) 56 No. 1, 49-51 (En). Private Bag 3, 1685 Halfway House, Republic of South Africa.

Three of four white rhinoceroses died within 3 months of introduction into a game reserve. Post-mortem examination of one of the animals revealed marked hepatomegaly with haemorrhage and severe necrosis of the liver as well as numerous ecchymoses and petechiae in the subcutaneous tissue and subserosa of the thorax, abdomen and diaphragm. Histologically, severe hepatic necrosis was the most significant finding. Algae recovered from the dam from which the animals drank were identified as *Microcystis aeruginosa*. A diagnosis of suspected *Microcystis* poisoning was made.

Key words: White rhinoceros, *Microcystis aeruginosa*, algal toxin, mortality, hepatic necrosis.

INTRODUCTION

Poisoning of domestic animals by toxic "waterblooms" has been an infrequent but repeated occurrence in several countries of the world since the first case was reported from Australia in 1878⁴. Clinical signs and mortalities occur when livestock ingest wind-concentrated surface blooms of toxic strains of freshwater cyanophytes, which develop in eutrophic freshwater dams, pans and lakes.

Three toxic genera, *Microcystis*, *Anabaena* and *Aphanizomenon* are most commonly implicated in these poisonings, although others may also cause toxicity⁶. *Microcystis toxica*, responsible for the death of thousands of cattle and sheep around the Vaal Dam in the 1940's¹⁴⁻¹⁶ is considered to be synonymous with *M. aeruginosa*⁸. At least two toxins are produced by *Microcystis* spp., a fast death factor and a slow death factor, which may cause the death of livestock from minutes to days after ingestion of a critical dose⁵.

A variety of clinical signs are manifested as a result of algal poisoning including incoordination, muscular weakness and fibrillation, unsteady gait, recumbency, laboured respiration, salivation, lacrimation, diarrhoea and, in cases surviving for longer periods, icterus, photodermatitis and loss of condition^{3,9,16}.

On post-mortem examination of acute cases the liver shows striking changes, with severe congestion, hepatomegaly and necrosis of hepatic cells being frequent findings^{7,9,12,16}. Haemorrhages in the intestine and on the abdominal serosa, splenomegaly, pulmonary oedema and haemorrhage and hyperaemia of the kidneys may also be found¹⁶. In chronic cases, lesions may vary according to the severity of the condition¹¹.

In South Africa, mortality of a number of domestic species including horses, mules, cattle, sheep and dogs has been attributed to the toxin produced by blooms of *Microcystis* in dams and pans¹⁶.

Recently, mention has been made of deaths of wild animals in the William Pretorius Game Reserve caused

by a toxic bloom of *Microcystis*¹. In this case, a number of black wildebeest (*Connochaetes gnou*) were suspected to have succumbed to a toxic strain of *M. aeruginosa*, although the diagnosis was not confirmed (P le Roux 1984 Division of Nature Conservation, Orange Free State, personal communication).

The present report deals with mortality of a white rhinoceros associated with the blue-green alga, *M. aeruginosa*.

CASE HISTORY

In May 1979, four white rhinoceroses (*Ceratotherium simum*) were released at the Barakologadi Game Reserve in the Odi district of Bophuthatswana. The reserve, some 9 000 ha in extent, is situated on the banks of the Klipvoor Dam (26°57' E, 25°5' S), approximately 100 km north of Brits).

The animals originated from the Umfolozi Game Reserve in Natal where they had been captured and boma trained for approximately 3 months prior to transport by road to Bophuthatswana. After release, the animals paired off, the older pair remaining closer to the dam and the two younger animals higher up in the reserve.

In Mid-July 1979 the younger animals were found dead. Interpretation of post mortem findings could not be made because the animals were in an advanced state of decomposition. It was estimated that they had been dead for 7-10 days at the time of examination.

Both older animals appeared clinically normal on subsequent daily observation, but their condition remained poor. On July 25, 1979 the female was found dead in sternal recumbency, although she was reported to have been active and lively the previous day. Apart from signs of mild diarrhoea, the male animal appeared to be otherwise healthy.

The dead animal was inspected and, apart from signs of excessive lacrimation, dried soft faeces around the anus and moderate infestation with *Amblyomma* ticks, no other external abnormalities were evident. A full necropsy was conducted.

*State Veterinarian, Phokeng, Bophuthatswana. Present address Private Bag 3, 1685 Halfway House, Republic of South Africa.

**Faculty of Veterinary Science, University of Pretoria.

MACROSCOPICAL PATHOLOGY

The most striking finding was severe hepatomegaly. The organ was markedly swollen with rounded edges and was a deep red-purple colour. When incised, the cut surface bulged and red fluid seeped from the incision. The parenchyma itself was soft and friable. The consistency of the organ resembled a liver with advanced autolytic changes even though the animal had only been dead for a few hours and the other organs showed no signs of autolysis.

Widespread petechiae and ecchymoses occurred in the subcutaneous tissues and serosa of the thorax, abdomen and diaphragm. Severe subendocardial and subepicardial haemorrhage was evident as well as severe focal disseminate subcapsular haemorrhage in the spleen.

The blood vessels supplying the intestines were congested. Approximately 5l of serous fluid was present in the abdominal cavity. The condition of the animal was poor with serous atrophy of fat in the mesenterium and pelvic area. Localized areas of congestion about 1cm in diameter were scattered throughout the lung.

MICROSCOPICAL PATHOLOGY

Samples of liver, spleen, kidney, heart and lung were collected and fixed in 10% buffered formalin. Appropriate tissue blocks were embedded in paraffin wax and 3-5 μm sections were cut and stained with haematoxylin and eosin for light microscopic examination.

In the liver all of the hepatic lobules examined showed that 90-100 per cent of the hepatocytes had undergone lysis necrosis. A few small groups of 3-10 viable hepatocytes were randomly scattered through the parenchyma. The connective tissue framework which had supported the liver cell plates was distorted or absent and the resulting confluent sinusoids were filled with large numbers of erythrocytes admixed with cytoplasmic debris from lysed hepatocytes. No inflammatory cells were observed and the portal triads showed no lesions. These lesions are similar to those reported in rats fed an extract of *M. aeruginosa*² as well as those reported in cattle and sheep dying from *Microcystis* toxicity^{7 9 12 16}. The spleen showed severe subcapsular haemorrhage with a marked atrophy of the white pulp.

The red patches seen in the lungs macroscopically were confirmed to be the result of congestion, probably due to hypostasis.

ALGAL IDENTIFICATION

At the time the animal died, the Klipvoor Dam in the park was covered with large quantities of blue-green algae which floated on the surface of the water forming a porridge-like green scum 4-12 cm thick.

Samples of water containing algae were collected and submitted to the Department of Botany, University of Pretoria, for identification. The water was shown to contain an almost pure culture of *M. aeruginosa*. (J C Coetzee and A Eicker Department of Botany, University of Pretoria, personal communication).

DISCUSSION

The literature on toxic freshwater blooms dates back to 1878 when the occurrence of a green scum was reported on Lake Alexandrina in Southern Australia by Francis⁴ who described the phenomenon as follows:

"A conferva that is indigenous and confined to the lakes has been produced in excessive quantities, so much as to render the water unwholesome.

It is, I believe *Nodularia spumigena*, allied to *protoptoccus*. Being very light, it floats on the water, except during breezes when it becomes diffused. Thus floating, it is wafted to the lee shores, and forming a thick scum like green oil paint, some two to six inches thick and as thick and pasty as porridge, it is swallowed by cattle when drinking, especially such as suck their drink at the surface like horses. This acts poisonously and rapidly causes death".

This description of the algae on the lake corresponds closely to what was observed at Klipvoor Dam at the time of the mortalities. The Klipvoor Dam is a large dam, 880 ha in extent with a capacity of 44×10^6 cubic metres. Some years ago, a large amount of fertilizer was added to the dam as part of a fish breeding scheme. This resulted in the development of highly eutrophic water which is known to stimulate the growth of blue-green algae¹⁰.

The cyanophyte responsible for the first reported mortalities of livestock in South Africa was named *Microcystis toxicata*¹⁴. It was considered to be a distinct though closely allied species to *M. aeruginosa*, differing in its larger size, peripheral network of cells, toxicity, odour and colour in decay¹⁴.

Although cell size plays an important role at the species level in the taxonomy of *Microcystis*, numerous morphological deviations, which are not genetically stable, exist¹⁰. Comparison of cell size and cell size distribution of a toxic and a non-toxic isolate of *Microcystis* grown under different environmental conditions showed that cell size varied to such a large extent that its use as a taxonomic criterium was invalid, unless environmental conditions were carefully taken into consideration¹⁰. It has further been noticed that waterblooms of *Microcystis* may vary in toxicity without any noticeable differences in microscopic morphology¹³. Colony habit as a taxonomic criterium is also considered to be suspect, since many variations exist even under natural conditions¹³.

Lewin, cited by Smit et al.¹³, typified the chaotic state of blue-green algal taxonomy in that *Anacystis nidulans* has been assigned to four different genera in a four year period. According to Stanier et al.¹⁵ there is little value in the older taxonomic treatments. Because of this and the fact that *M. toxicata* has been given as a synonym for *M. aeruginosa* in a taxonomic review of blue-green algae⁸, it appears that livestock mortalities in South Africa as a result of ingestion of toxic algae may be attributed to a single species of cyanophyte viz. *M. aeruginosa*.

No mortalities attributable to *Microcystis* poisoning were observed in any other species of game found in the reserve. These include giraffe, zebra, wildebeest, impala, kudu, jackal and warthog. Occasionally carcasses of these species are found, but they are usually decomposed or have been partially consumed by scavengers and the cause of death is therefore seldom established.

The specific susceptibility of the rhinoceros to algal toxins is unknown. Despite the wide variety of species

(including horses, mules, cattle, sheep, dogs and laboratory animals) affected by the toxin^{2 7 9 12 16} there is apparently a wide interspecies variation in susceptibility to *Microcystis* poisoning. For a certain strain of

The toxicity is further determined by dominance of toxic strains of algae, concentration of toxic organisms, release of toxin and consumption of toxin in sufficient amounts by susceptible animals before appreciable dilution, absorption or destruction occurs⁵. Experiments in sheep revealed a remarkably sharp dose response curve in that up to 90 % of the lethal dose of bloom could be ingested in a single administration without measurable effect⁷. Relative to other species found in the reserve, the rhinoceros would drink a large volume of water and therefore ingest more toxic algae, which may be a further explanation as to why they were specifically affected. Apart from a small number of dried *Senecio* plants (which had not been eaten), no other known hepatotoxic plants were found in the reserve.

M. aeruginosa, the LD 100 for mice was found to be 19 mg of dried algae/kg whereas a dose of more than 950 mg/kg was required to induce symptoms in sheep⁷.

It has been shown that variations in the toxicity of *Microcystis* may occur over short periods of time due to the influence of environmental factors such as light intensity, temperature, age of cells and pH^{5 17}. The algae in the dam are therefore probably not always toxic and the mortalities may have occurred because the rhinoceros happened to drink at a time when the correct environmental factors prevailed for the production of toxin.

On the basis of the liver lesions, which are similar to those described for laboratory animals exposed to a toxic extract of *M. aeruginosa*^{2 9} and those described for domestic animals ingesting the toxin^{7 9 12}, mortality of the rhinoceros was suspected to be the result of *Microcystis* algal poisoning. This diagnosis was supported by confirmation that the dam from which the animals drank was heavily contaminated with *M. aeruginosa*, although no tests were conducted to establish the toxicity of this particular strain.

The remaining male rhinoceros was captured, successfully transported and released in the Pilanesberg Game Reserve in August 1979.

This report emphasizes the important role that toxic flora may play in the successful introduction or reintroduction of indigenous and exotic game to reserves and farms which may be unnatural habitats or disturbed ecosystems.

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