

ALGAL POISONING IN BEEF CATTLE

D. W. MACDONALD†

ALGAL POISONING due to the blue-green algae, *Microcystis* species, has been recognized for years (5, 2). As a result of the work of the National Research Council, (3, 4, 1) the true nature of the toxic principle is known. A multiplicity of symptoms and lesions may present themselves in this type of poisoning, and this report is written to illustrate some of these phenomena. The poisoning described occurred in a herd of 80 Hereford cattle pastured on lands adjoining a large lake. For the past 50 years cattle had watered from the lake and no previous algal poisoning had occurred. Weather conditions are important in creating the proper conditions for the growth of algae. All but one of the fourteen deaths occurred in cattle pasturing on the south shore of the lake where the algae concentrated.

History

June 28, 1959. One cow was found dead that morning. The owner later reported that the remaining animals appeared normal at that time.

June 29 and 30. The weather was cool with intermittent rain.

July 1. A warm, brisk wind was blowing from the north-west.

July 2. In the morning four cattle were found dead, four were missing, and an additional twelve animals were ill. The latter were treated that afternoon.

July 3. The missing animals were found dead. Several animals that had appeared well the day before were now showing symptoms and were treated. One of the calves treated the previous afternoon had died during the night, but in general the treated animals showed remarkable improvement.

A second group of animals had been brought from pasture for branding and during the night had access to the eastern shore of the lake. The only loss in this group occurred during the evening of July 3.

Symptoms

Most of the deaths were unobserved. The affected animals which were seen before death showed symptoms of prostration, bellowing, kicking and obvious pain. Icterus was quite pronounced on the mucous membranes and lips of all the affected animals. Photosensitization, with severe blistering of the muzzle, was apparent. Muscular tremors were present in certain muscle groups, particularly those of the shoulder. Almond-scented breath was observed in several of the affected animals. Diarrhoea was not a constant feature. The udders of some of the cows became a brilliant red, and at a later date pronounced sloughing was noticeable on the muzzles and udders of the cows. Lactation ceased in those cows nursing calves at the time of the outbreak. The cases which recovered displayed oedema of the face, most notably around the eyes. Later the oedema formed dependent swellings under the jaw. Affected animals which survived showed a very marked loss in weight.

†Lacombe, Alberta.

Post-mortem Findings

Necropsy was performed on one cow and one calf. The calf displayed very little gross change. The cow was examined on July 3 within one hour of death. Prior to death, she had bled profusely from the anus. Post mortem examination revealed profuse haemorrhage from the terminal two feet of the rectum, limited localized areas of inflammation in the colon and haemorrhages on the abdominal serosa. The liver was enlarged and very friable. There was marked distention of the gall bladder and oedema of the adjacent tissues. Samples of liver and kidney were forwarded to the Provincial Veterinary Laboratory for histopathological examination.

Histopathology

The kidney showed a toxic tubular nephritis. There was proliferation of fibrous tissue in the portal areas of the liver with diffuse degenerative changes in the hepatic cells. Haemosiderin was present in large amounts.

Diagnosis

A tentative diagnosis of algal poisoning was made. On July 2, when the initial losses were observed, a heavy paint-like, blue-green surface scum was noticeable along the south-east shore of the lake. Traces of this growth were plainly visible on the muzzles and feet of the dead and sick animals. The following day, the blue-green algae could be readily seen along the shore, but the heavy growth had dissipated.

Following the death in the second group of animals, which did not have access to the south shoreline, a closer examination of the east shore revealed a small bay-like recess with heavy algal growth. Fresh tracks indicating that the place had been used for watering were plainly visible.

Laboratory Investigation

In order to confirm the original diagnosis, samples of algae were collected from three separate locations: (1) dried blue-green algae from the south shoreline; (2) heavy green scum from the south-east shoreline; and (3) heavy green scum from the east shoreline near the buildings. A 0.2 c.c. suspension of each of these samples was injected intraperitoneally into mice. The mouse receiving the first sample died minutes later. The mice receiving the second and third samples remained normal. A 1 gm. portion of the first sample was ground in a mortar and suspended in 30 c.c. of distilled water; additional water was added to facilitate filtering. A Seitz filtrate was prepared and 0.2 c.c. of the filtrate were injected intraperitoneally into two mice. The smaller of the two showed symptoms and died 1½ hours later. The larger mouse displayed symptoms but subsequently recovered. It was sacrificed.

On histopathological examination, the mouse livers showed congestion and haemorrhage in interlobular areas. The liver from one mouse showed diffuse haemosiderin deposition.

Treatment

Access by the cattle to the lake's shoreline was prevented and they were watered by other means.

Treatment for the severely affected adult animals consisted of 50 c.c. of Thiofate* intravenously, 10 ounces of Scarlet Drench† orally, and 10 c.c. of Amfetasul 5 per cent‡ intramuscularly. The mildly affected adult animals were treated with 50 c.c. of Thiofate intravenously. Affected calves were given 20 c.c. of Thiofate intravenously.

Rectal haemorrhage was a feature in only two of the adult animals, both of which died. Liver damage with photosensitization and subsequent sloughing undoubtedly accounted for the marked weight loss which occurred in many of the surviving animals.

Discussion

An outbreak of algal poisoning affecting a herd of range cattle has been described. The poisoning resulted in the rapid death of 14 animals. Another 20 animals exhibited signs of liver damage and photosensitization. The liver damage probably resulted in porphyrinaemia. It has been reported that photosensitization is directly related to porphyrinaemia (6). The question concerning the length of time between the consumption of the poisonous algae and the onset of illness is invariably posed on such an occasion. In the second group of animals, the only loss occurred approximately 24 hours after consumption of the poisonous algae. Undoubtedly the time lapse was considerably less in the some of the more acutely affected cases. Factors such as the degree of fullness of the rumen, the concentration of the poisonous algae in the water and the amount of water containing poisonous algae consumed by the animals would all tend to vary the time lapse between ingestion and the onset of symptoms. There was no indication that calves suffered ill-effects from nursing obviously ill cows.

The author wishes to acknowledge the assistance of Drs. J. G. O'Donoghue and F. E. Graesser of the Alberta Veterinary Laboratory.

REFERENCES

1. BISHOP, C. T., ANET, E. F. and GORHAM, P. R. Isolation and identification of the fast-death factor in *Microcystis aeruginosa* NRC-1. *Canad. J. Biochem. Physiol.* 37: 453-471. 1959.
2. GARNER, R. J. *Veterinary toxicology*. London: Baillière, Tindall and Cox. 1957.
3. GRANT, G. A. and HUGHES, ELWYN O. Development of toxicity in blue-green algae. *Canad. J. Publ. Hlth.* 44: 334-339. 1953.
4. HUGHES, E. E., GORHAM, P. R. and ZEHNDER, A. Toxicity of a unialgal culture of *Microcystis aeruginosa*. *Canad. J. Microbiol.* 4: 225-235. 1958.
5. O'DONOGHUE, J. G. and WILTON, G. S. Algal poisoning in Alberta. *Canad. J. Comp. Med.* 15: 193-198. 1951.
6. SMITH, H. A. and JONES, T. C. *Veterinary pathology*. Philadelphia: Lea and Febiger, 1957.

*Moore-Thompson-Clinger Limited, Hamilton, Ontario.

†Haver-Lockhart Laboratories, Kansas City, Mo.

‡Allied Laboratories (Canada) Ltd., Guelph, Ontario.