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CATTLE POISONING RELATED TO THE BLUE-GREEN ALGA, *POLYCYSTIS AERUGINOSA* KUTZ.

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Numerous isolated incidents of animal poisoning by toxic algae have been reported in the literature (Gorham, 1962, Kingsbury, 1964). However, this condition is not generally represented in human or veterinary medical clinical texts.

The aforementioned episode involved 16 cattle deaths in two herds near Fayetteville, Arkansas, one in which another toxic material (an organophosphate compound) was in use in a research project and was suspected as the lethal agent. Poisoning by the phosphate material was ruled out on the basis of extensive experience with these compounds by one of the authors (J. F. B.). In the search for the cause of the illness, high concentrations of blue-green algae were found in the water supply for the cattle.

As a result of these incidents, a preliminary research project has begun to further investigate the potential implications of this isolated suspected algal poisoning episode. Limnological, phycological and clinical investigations are incorporated in the research program.

A concise presentation of the occurrence, etiology, clinical findings, and control of algal poisoning in animals follows.

OCCURRENCE

A usually acute and highly fatal disease of animals results from drinking water containing high concentrations of toxic strains of blue-green algae. Extensive loss of life and severe sickness of livestock, pets, wild animals and humans have been associated with algal blooms in the northern half of the U. S. (also in Texas), the southern provinces of Canada, Russia, Argentine, Australia, South Africa and other countries.

Poisoning does not occur unless there is a dense bloom of toxic material. The factors leading to the formation of such blooms include warm sunny weather, ample nutrients (especially nitrates), and a gentle prevailing wind which drifts and collects the algae against the windward shore. Such conditions commonly occur during the summer months in drainage ponds and lakes used for watering livestock.

Cattle Poisoning by Blue-Green Algae

ETIOLOGY

Early studies indicated the primary toxic principle to be an alkaloid which affects the central nervous system and liver. A secondary toxic principle was described to be algal phycobilin pigments which accumulate in the skin of animals with a resultant increase in photo sensitivity.

More recent research discounts the alkaloidal nature of the toxic principle and incriminates a seven-amino acid cyclic polypeptide which produces rapid toxic symptomatology. In general, the crude toxic principle has the following characteristics: It can exist outside the cells in the water around the algae; it passes through cellophane and animal membranes by dialysis; it is non-volatile; it is relatively heat stable; it is soluble in water, alcohol (95% or less) and acetone, it is resistant to extreme pH changes. Several toxic fractions have been obtained by chromatographic procedures and one of these may be identified by a characteristic absorption curve having an almost complete absorption from 210-290 millimicrons. This fraction produces paralytic symptoms in mice and is lethal to 20 gram mice at a dosage of 0.7 mg.

CLINICAL FINDINGS

Toxic symptoms appear rapidly, usually within 15 to 45 minutes, after ingestion of poisonous material. Poisoning proceeds rapidly and is severe; death is common, occurring in less than 24 hours, often within one or two hours. The most commonly reported sequence of events are rapid prostration, convulsions and death; although convulsive signs are not always marked. Abdominal pain, muscular tremors, dyspnea, cyanosis and excessive salivation are commonly reported. A moderate number of cases have shown severe gastrointestinal manifestations including diarrhea, bloody feces, and icterus. Photosensitization frequently occurs in animals who survive for several days.

CONTROL

Removal of all animals from the affected water supply is an essential first step to all other measures. Algae growth may be suppressed with copper sulfate or other algacide treatment, but does not remove the toxin already present in the water. If no other water supply is available, animals should be allowed to drink from the clearest part of the water source opposite the windward shore (wind currents tend to blow and accumulate algal growth on the windward shore).

It is essential that animals dying from algae poisoning not be used for food as the toxic principle is quite stable and consistently produces toxic symptoms in the consumer. This is especially true with respect to the liver of diseased animals.

TREATMENT

Following removal from the contaminated water supply, affected animals should be placed in a relatively protected holding area, especially out of direct sunlight. Ample quantities of water and excellent quality feed should be made easily available. Mild to moderate laxatives may be used to move the toxic material out of the body (Caution: Affected animals are usually very weak and a minimum of violent procedures should be employed). In vomiting animals, an emetic may be used to good effect.

Even though the alkaloidal nature of the toxin has been discounted, 1 to 2 oz of sodium thiosulfate intravenously or orally seems to be of benefit. In surviving animals, a long recuperation period is to be expected before normal production is resumed.

It is anticipated that initial research findings of the present study will justify a more intensive investigative effort of the previously described phenomenon.

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