



Chlorate poisoning in animals: A brief overview

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In the form of a spray or dry salt, sodium chlorate, a potent oxidizing agent, is employed as a weedicide and defoliant. Despite the low likelihood of chlorate poisoning, animals that crave salt may consume large amounts of the substance due to its salty flavor and appearance, or it may be mistaken for common salt and added to animal feed. Sheep are poisoned less frequently than cattle. For cattle, sheep, and poultry, the lethal doses of sodium chlorate are 1, 2, and 5 g/kg body weight, respectively. But for dogs, the lethal dose ranges from 1.5 to 3.5 g/kg. In 6 to 48 hours, death frequently happens. It is easily absorbed from the intestines and substantially unaltered when eliminated.

Mechanism of toxicity

In a manner similar to that of nitrite poisoning, chlorate is a potent oxidizing agent that causes methaemoglobinemia. However, unlike nitrite poisoning, the process of met-Hb formation is slower and continues even after the animal has died because chlorate is not inactivated. Moreover, it leads to severe hemolysis.

There is no mechanism in the erythrocytes for the reversal of methaemoglobinemia, and chlorates also cause the development of low amounts of sulphaemoglobin, which is a partially oxidized and denatured mixture of the pigments as a result of nonspecific oxidation. But it never occurs in concentrations that pose a threat to life. Chlorate immediately irritates the digestive tract, which results in vomiting and pain in the abdomen.

Clinical signs

The general symptoms of chlorate toxicity are similar to those of nitrite poisoning, with the exception of the delayed start. In chlorate poisoning, the respiratory indicators are the first to be noticed, whereas in nitrite poisoning, the gastrointestinal symptoms, such as vomiting, diarrhea, colic, etc., show earlier and the respiratory distress is observed later. Additionally, symptoms of chlorate poisoning



include hematuria, hemoglobinuria, and the efflux of dark, tarry blood that clots easily from the body's natural orifices. In severe cases, poisoning may still be present even though there are no outward symptoms.

Post-mortem lesions

1. Generalized cyanosis.
2. Brownish discoloration of the organs and tissues.
3. Exudation of blood from natural orifice.
4. Blood is of dark chocolate color.
5. Gastroenteritis.

Diagnosis

1. History.
2. Clinical symptoms.
3. Oozing out of dark tarry blood from the natural orifices.
4. Post-mortem findings if death.
5. Muscle tissue is dark or nearly black, and when exposed to air, its surfaces become slightly lighter.
6. Heart is dark flabby and liver is almost black.
7. Chlorate is detected through the analysis of blood, urine, tissues, and ruminal fluids.
8. Estimation of met-Hb in blood during the onset of clinical symptoms, during the death, and after. Due to the fact that met-Hb creation continues even after an animal dies, the concentration of met-Hb rises with time.
9. Response to treatment.

Differential diagnosis

Chlorate poisoning should be distinguished from other poisonings because respiratory distress is one of the primary symptoms, followed by mortality in acute instances.

- I. Nitrite poisoning: No oozing out of blood from the natural orifices.
- II. Cyanide poisoning: Blood is bright red in color.
- III. Hydrogen sulphide: Blood is dark in color.
- IV. Carbonmonoxide: Blood is bright red in color.



- V. poisoning caused by various hemolytic substances such as medicines, copper, dimethyl sulfoxide, gossypol, and snake venom.
- VI. Warfarin poisoning.

Treatment

Similar to nitrite poisoning, the goal of treatment for chlorate poisoning is to lessen met-Hb production from Hb as a result of chlorate poisoning. methylene blue is the preferred medication. In nitrite poisoning, specifics of the method of action, doses, and safety measures for using methylene blue have been described. The creation of met-Hb continues in the body for as long as chlorate is present, therefore methylene blue treatment is more time-consuming and frequent than treating nitrite poisoning. The next step should be to flush the poison out of the digestive tract using gastric lavage or saline purgatives.