Lead Arsenate Toxicosis - A Case Report

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Lead arsenate compounds are used as insecticides in and around orchards. It persists in the soil many years after use and commonly is found in high concentration around filling places for spraying and dusting machinery and in old orchards from which trees have been removed.

Animals poisoned with a compound of lead arsenate manifest clinical signs of acute arsenic poisoning. The lead portion is relatively insoluble and is rapidly excreted from the body; only long periods of exposure to high levels of lead will cause toxicosis. On the other hand, arsenic poisoning can cause an acute or chronic condition, depending on the particular compound involved, dosage, and physical condition of the animal.3

Arsenic compounds have widely varying chemical and physical properties which govern their toxicity and absorption. In general, organic arsenic compounds are much less toxic. Also, the chemical composition, physical state (i.e. solid, coarsely or finely ground, in solution), and solubility contribute to the toxic action. The condition of the animal's digestive organs, nature of the ingesta, and method of application will affect the toxic dose to any species.

Trivalent and pentavalent forms of arsenic exist in nature. The trivalent form is more toxic; it is believed that compounds containing pentavalent arsenic exert a toxic action only after conversion to the trivalent form. The trivalent arsenical compounds are toxic mainly because they combine with sulfhydryl groups and enzymes essential in the oxidative decarboxylation of pyruvic acid and alpha-ketoglutaric acid. In addition, fat and carbohydrate metabolism is blocked, a reduction in tissue concentration of vitamin C, and capillary dilation occurs.

All species of domestic animals are susceptible to arsenic toxicity. However, the bovine and feline are most commonly poisoned by inorganic arsenic compounds. Cattle will seek it out for its salt-like flavor; cats contact arsenicals through ant baits.

The onset of clinical signs of lead arsenate poisoning are sudden, commonly the owner finds one or two animals dead. Extreme weakness and trembling along with violent colicky symptoms, slate-grey fluid type diarrhea, nausea, and vomiting are the most prominent signs in affected animals. Temperatures are usually normal in these animals.

Post mortem lesions are always present in the gastrointestinal tract with arsenic poisoning. The stomach and intestines may be inflamed and edematous. Ruptured blood vessels, necrosis of intestinal epithelium, and fluid contents containing shreds of mucosa may be evident. Non-specific lesions may or may not be present and include congestion of the lungs, endocarditis, and hemorrhages on the surface of the heart. Hemorrhage on the peritoneal surface occurs occasionally. It should be kept in mind, however, that if death occurs rapidly, lesions will be minimal. The most constant post mortem lesion is the large volume of gastrointestinal fluid contents.

Other conditions may produce signs similar to those of arsenic poisoning. Among them are lead poisoning, mucosal disease complex, malignant head catarrh, poisonous plants, organophosphorus insecticides, and hypomagnesemic tetany. However, a careful history, circumstantial evidence, clinical signs, and postmortem examination will serve to differentiate most conditions.

On August 16th, 1969, a northwest Missouri farmer contacted a local veterinarian with a complaint of finding one calf sick...
and another sick and recumbant. Upon arrival at the farm, one of these animals had died—the other was prostrate and breathing rapidly. Severe cyanosis of the epithelial membranes, grey-fluid diarrhea, and a temperature of $104^\circ$ F. were noted upon physical examination.

Careful observation of the other cattle in the pasture revealed several more animals showing clinical signs. In these animals severe weakness, posterior ataxia, profuse fluid diarrhea containing flecks of blood and sloughed epithelial cells were noted. Signs of abdominal pain and colic were evident. Temperatures of these animals ranged from $100^\circ$ F. to $104^\circ$ F.

Post mortem examination of the dead calf revealed severe congestion and edema of the gastrointestinal epithelium. An unusually large amount of fluid was present in the intestinal tract which had the same character as the feces. Ecchymotic hemorrhages on the epicardium and hemorrhage around the thymus gland were also noted.

The affected calves were in a herd of thirty-seven black-white-face and Hereford-Holstein crossbred heifers. These animals ranged in age from fifteen months to two years. Most of these calves were home raised. The remainder of the herd had been purchased before reaching a month of age from a farm in New York. These heifers were triple vaccinated at approximately four months of age. No other vaccinations had been given.

The herd had been grazing in an eighty acre pasture which surrounded the buildings of an old farmstead since June. The improved pasture was a mixture of bluegrass and lespedeza. The water was supplied by a large pond with no history of treatment to kill the moss and weeds around the edge.

The clinical signs, history, and post-mortem exam were indicative of a toxico-sis. A thorough search of the surroundings and repeated questioning of the owner was attempted without success.

Symptomatic treatment of the affected animals consisted of intravenous administration of $500cc$ of calcium gluconate, $500cc$ of $50\%$ dextrose and electrolytes, and four Alkadote boluses.

The veterinarian gave instructions to watch the animals carefully and advised the owner that a retreatment was indicated in twelve hours.

On the morning of August 17th, a second treatment was administered. One of the heifers had died during the night and another of the animals was recumbent so the owner was advised on the possibility of collecting tissues and sending them to a diagnostic laboratory for analysis. He was reluctant to permit this until the calf died so instructions to call the veterinarian when the calf died were made. Also, the owner was urged to search closely again for some clue of the toxicant.

Late that afternoon the herd was re-examined. At this time the owner announced the discovery of several torn paper sacks of abandoned lead arsenate orchard spray powder in one of the old farm buildings. The sacks were on a shelf approximately six feet from the floor of the building. Very recent entry into the building by the calves was evident from fresh droppings on the floor. It was noted that the animals which had become ill had been taller, enabling them to reach the paper sacks containing the lethal compound.

**Summary**

Treatment of the affected animals was unsuccessful, all seven animals treated died within forty-eight hours. No diagnostic work was employed due to the conclusive circumstantial evidence present.

Perhaps more thorough follow-up treatment and the use of sodium thiosulfate intravenously would have been beneficial.

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<tr>
<th>Rx Alkadote-Curts Laboratories</th>
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<tbody>
<tr>
<td>1. Sodium thiosulfate, anhydrous, equivalent to U.S.P. Sodium thiosulfate 150 gr.</td>
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<td>2. NaHCO₃ 40 gr.</td>
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<td>3. Al hydrate 100 gr.</td>
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**REFERENCES**


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