

Thallium Poisoning in the Dog

by

Virginia L. Marshall, B.S., M.S.*

Thallium compounds have been used as rodenticides for many years. Reports of poisoning of domestic animals by thallium were published as early as 1920. The general use of these compounds by pest control companies, and their availability to the general public has increased the frequency of accidental poisoning of domestic animals.

Thallium, a heavy metal, has an atomic weight similar to both lead and mercury, and has many characteristics in common with these metals. As is true of many other heavy metals, thallium is a general cellular poison, although its most prominent effects are in those cells localized in a few areas of the body. Following ingestion, thallium is rapidly distributed throughout the body, being found in all tissues and body fluids. It is excreted in urine, feces, and milk. Using spectrographic methods, thallium has been detected in the urine of dogs less than 24 hours after ingestion. Peak blood levels are found within a few hours after oral administration of thallium compounds. Like lead, thallium is very slowly eliminated by the body. It has been found present in the urine months after administration. As might be expected, thallium can be a cumulative poison; several individually non-lethal doses administered over a period of time, can result in the accumulation of a lethal quantity in the tissues.

Thallium compounds, particularly thal-

lous sulfate and acetate, have many advantages over other compounds when used as a rodenticide. Since they are odorless and tasteless, they are more readily taken by rodents than are those with an odor or distinctive taste. Rats in particular are known to avoid baits containing rapid-acting poisons such as strychnine. Since thallium salts rarely cause death in less than 3-5 days after ingestion, rats do not associate the death of other rats with the consumption of the bait. The main disadvantage of thallium as a rodenticide is its toxicity to all other mammals. Since heavy metals are not metabolized, domestic animals can be poisoned by consuming rats which have died from thallium poisoning, as well as by direct consumption of thallium baits.

SYMPTOMS

The symptoms of thallium toxicity in the dog may present a confusing diagnostic picture for the clinician. Poisoning can generally be classed as per-acute, acute, or chronic, although these terms can also be very misleading. It is very rare when any symptoms are observed less than 24 hours after ingestion of thallium. In chronic cases, the onset of symptoms may be delayed for 3-5 days or more.

In per-acute poisoning, severe gastroenteric signs, including vomiting, severe abdominal pain, loss of appetite, and diarrhea are observed (7,8). Occasionally a hemorrhagic enteritis will follow. Dyspnea is also seen, with death occurring in 2-5

* Mrs. Marshall is an assistant professor in the Veterinary Diagnostic Laboratory.

days. In some cases, motor paralysis and/or trembling may also be observed. Some authors have reported that the body temperature remains normal unless secondary infection is present (6). In experimental cases of per-acute thallium toxicity in dogs, the body temperature was elevated 36–40 hours after ingestion to 108° F in one dog, 103° F in a second, and 102.8° F in a third dog. The temperatures remained at this level until death, which followed within 3 days (1).

The course of the acute form is somewhat slower than the per-acute, lasting from one to several weeks. As with the per-acute form, gastroenteric disturbances are usually the first symptoms observed. This is combined with, or followed by, muscular tremors and evidence of pain in the extremities. Conjunctivitis and injection of the sclera are observed in many cases. The oral mucous membranes are frequently congested, often becoming brick-red or mahogany in color. Bronchitis and pneumonia may also develop. If death is delayed, severe motor disturbances and blindness occur. Even if the acute stage is survived, death may occur later. Animals which survive the initial effects of the thallium may succumb to secondary bacterial infections or to extensive renal damage incurred during the process of excretion of the thallium.

The signs of chronic poisoning are similar to those of the acute form, but are generally milder and of longer duration. The symptoms described perviously are not indicative of thallium poisoning alone, but are similar to those of most heavy metal poisonings. Perhaps the most characteristic signs of thallium intoxication in the acute form are the congested sclera and brick-red oral mucous membranes while the hair and skin changes are most characteristic in the chronic form, although they may take from one to three weeks to develop. The most obvious initial change is the rapid loss of hair beginning in the areas subject to friction and progressing at times over the entire body. Close examination will often show this change first in the interdigital and axillary spaces where the skin may become dry and cracked. Fol-

lowing hair loss, the skin becomes dry and crusted. Necrosis of the superficial layers may also appear. Necrosis is sometimes preceded by a reddening of the skin, particularly in areas subject to friction. Should the animal survive, the normal growth of hair will resume, although several months delay may be involved. These gross skin changes can be found in other disorders, and it is often advisable to eliminate the possibility of infectious or parasitic agents.

POSTMORTEM FINDINGS

The necropsy findings in thallium intoxication vary with the severity of dosage and length of illness. In per-acute cases, severe hemorrhagic gastroenteritis may be the only finding, although inflammation of the respiratory mucosa occasionally occurs.

In acute cases lesions in other organs are observed. Congestion and hemorrhage may occur in many organs including spleen, kidneys, and heart. Fatty degeneration and necrosis of the liver has also been described. Petechial hemorrhage, perivascular cuffing and demyelination occur frequently in the brain. In the dog, the cutaneous histopathology of thallium intoxication is unlike any other cutaneous reaction reported in the literature. Examination of sections taken from erythematous areas reveals hyperkeratosis, moderate parakeratosis, hyperemia, and hyaline changes. The cutaneous histopathology of thallium poisoning has been described in great detail by Schwartzman and Kirschbaum (5).

Analysis of tissues from an animal with thallium poisoning has shown the presence of thallium in all tissues. For the laboratory diagnosis of a suspected case of thallium intoxication there are several methods available for analysis of body fluids. In practice, testing of urine by any of the "quick tests" (2, 3) available has been shown to be quite reliable. One such test, described by Gabriel and Dubin (2), requires a minimum of time and can be routinely used by a practitioner with a minimum of expense for chemicals and equipment.

TREATMENT

Many approaches have been tried for the treatment of thallium poisoning. If used within one to two hours after ingestion of the poison, gastric lavage with 1% sodium iodide solution has been shown to be beneficial. Treatment with sodium thiosulfate, BAL or calcium EDTA has been of little benefit. Diphenylthiocarbazonone has been used for treatment of thallium poisoning with somewhat more success than other therapeutic agents. Since this compound has resulted in a serious systemic zinc loss, it must be used with utmost caution. Treatment of acute cases with 70 mg. diphenylthiocarbazonone/kg body weight three times daily, with 2-6 gms. potassium chloride daily has been suggested as being satisfactory (4). In chronic cases it may be inadvisable to administer such high doses since it may cause the rapid transfer of thallium from the tissues into the blood stream and vital organs resulting in acute thallium toxicity.

SUMMARY

Thallium poisoning in dogs is a disorder which is difficult to diagnose by symptoms

alone. The most characteristic clinical changes observed are injection of the sclera and reddening of oral mucous membranes in acute cases and alopecia and skin changes in chronic cases. The detection of thallium in the urine of suspected cases of poisoning by one of the "quick tests" available has been shown to be one of the best diagnostic aids. A specific, effective antidote for thallium toxicity is lacking, although diphenylthiocarbazonone has shown promising results in some cases.

REFERENCES

1. Buck, W. B., Personal Communication.
2. Gabriel, K. L. & Dubin, S., A Method for the Detection of Thallium in Canine Urine, *Am. Vet. Med. Assoc. Journal* 143:722, 1963.
3. Garner, R. J., *Veterinary Toxicology*, William & Wilkins Co., Baltimore p 124-125, 1961.
4. Mather, G. W. & Low, D. G., Thallium Intoxication in Dogs, *Jour. Am. Vet. Med. Assoc.*, 137:544, 1960.
5. Schwartzman, R. M. & Kirschbaum, J. O., The Cutaneous Histopathology of Thallium Poisoning, *Jour. Investigative Dermatology* 39:169, 1962.
6. Skelley, J. R. & Gabriel, K. L., Thallium Intoxication in the Dog, *Annals; N. Y. Acad. Sci.*, 3:612, 1964.
7. Radeleff, R. D., *Veterinary Toxicology*, Lea & Febiger, Philadelphia, p 161-162, 1964.
8. Wildson, J. E., Thallotoxicosis, *Jour. Am. Vet. Med. Assoc.*, 139:1116, 1961.

a case report:

Thallium Toxicity in the Dog

by

Marvin Farley

HISTORY

A two and one-half year old spayed female miniature schnauzer was presented to the university clinic for treatment. The owner reported that the dog had been losing hair for the past four days. No other history was available and the animal had no other observable signs.

* Mr. Farley is a senior student in the College of Veterinary Medicine at Iowa State University.

CLINICAL SIGNS

The patient was treated as an inpatient. Large, multiple, focal areas of erythema were noted on the dog's skin. Hair could easily be pulled out of the skin in tufts. Physical examination revealed no other externally detectable clinical signs, although skin sensitivity seemed to increase over a period of five to seven days.