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Lead Poisoning in Dogs

by
John Bengfort, D.V.M.*
Dr. Robert W. Carithers†

SUMMARY
Lead poisoning is seen primarily in dogs less than one year old; the highest percentage of those are less than five months old. Many sources of lead are available to young dogs, although lead-based paint and linoleum are the principal problems. The characteristic clinical signs involve the gastrointestinal system (vomiting, colic and diarrhea), nervous system (hysteria, convulsions and nervousness) or both. Generally the signs are followed by a period of normalcy. Dogs are unique in that clinical pathological analysis showing many nucleated red blood cells and basophilic stippling in the absence of severe anemia is strongly suggestive for lead poisoning. However, analysis for blood lead with over 0.35 ppm. is the best diagnostic tool. Chelation therapy with Ca EDTA is the treatment of choice.

CASE REPORT
On August 1, 1975, a one-year-old male beagle was admitted to Stange Memorial Clinic as a lead poisoning suspect. The dog's history included vomiting and abdominal pain for the past 10 days and nervous signs of two days duration manifested by convulsions, running, crying and apparent blindness. The dog reportedly had been tethered adjacent to an old building from which the owner had been scraping paint. On outpatient examination, the beagle appeared depressed and emaciated and vomited a yellow foamy material. He had reportedly not eaten any significant amount of food for about three weeks. When left to himself, he would stretch out his front legs and rest his head on them while keeping the hind legs erect. Abdominal radiographs revealed numerous flecks in the colon which could have been lead-based paint chips. Blood samples for hematologic examination and lead analysis were taken. Chelation therapy was initiated on the basis of the radiographs. He was given 12 mg/lb. of Ca EDTA subcutaneously. The blood analysis showed a hemoglobin of 13.6, PCV of 42, and a corrected WBC of 8,625. There were 387 NRBC/100 WBC, along with basophilic stippling, polychromasia and anisocytosis. The blood lead level was 1.34 ppm. The dog was given several enemas initially and was continued on Ca EDTA, lactated ringer's and glucose, periodically, for 14 days. He had several convulsions and went into more severe depression at the onset of Ca EDTA.

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therapy, but when it was stopped for one to
two days, he would appear very bright and
alert. As the treatment regime progressed,
the Ca EDTA didn’t have as much lead
available to mobilize and therefore no
effects were noted.

Periodic monitoring of blood and urine
lead indicated that the blood lead was 0.44
pm. and the urine lead 15.4 pm. on
August 8th. On August 15th, the blood lead
was 0.5 pm. and urine lead 1.4 pm.

The dog was hospitalized a total of 15
days, with a total of 10 days Ca EDTA
treatments. Upon his release, he was very
active and playful, eating well and
regaining lost weight.

DISCUSSION

In a study at Angell Memorial Animal
Hospital in Boston, it was found that about
one in every 25 dogs under six months of
age had lead poisoning. They also noted a
higher incidence in the summer months.
This may be due to increased Vitamin D
produced because of more exposure to
sunlight aiding in intestinal absorption of
ingested lead. Also, there are more sources
of lead available in summer (2,5,6).

Due to the uncontrollable chewing habits
of young dogs, anything containing lead
can be a potential source. Lead-based paint
has been the primary agent when the
source was known. Unfortunately, in 70% of
the documented cases, the source was
unknown (6). Other lead containing items
include linoleum, putty, toys, golf balls, rug
pads, solder, batteries, glazed ceramic
water bowls, lead weights, fishing sinkers,
and plumbing materials.

Automobile exhaust from leaded gasoline
in areas of heavy traffic has been in­

criminated in those cases where the source
was unknown (6).

Increased incidence of lead poisoning is
usually in slum areas where there is a high
amount of natural lead available in the
soil, such as around lead mines (3,5).

Clinically, the dog exhibits gastroin­
testinal and nervous symptoms. The

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gastrointestinal signs usually are apparent
first and consist of vomiting, diarrhea (or
constipation), anorexia, and marked
emaciation. Colic or pain is the most
consistent finding and is manifested by

whining, restlessness, abnormal positions,
tensing of abdominal muscles, and crying
(1,2). This is followed by a period of
quiescence, after which the nervous signs
develop. The nervous signs consist of
hysteria, convulsions, champing, ner­
vousness, and behavioral changes, par­
ticularly depression (1,2,5).

Blood lead analysis is the best diagnostic
method for lead poisoning. Levels above
0.35 pm. are indicative of unusual ex­
posure. Normal levels are in the range of 0-
0.15 pm. (1,7). Since blood analysis for
lead is not rapidly accomplished, other
diagnostic methods have proven to be quite
definitive. The hematologic finding of many
nucleated red blood cells along with
basophilic stippling of red blood cells in the
absence of anemia is nearly pathognom­
ic. The number of nucleated RBC’s varies
directly with the amount of lead in the blood
and hyperplasia of erythroid elements in
bone marrow. One will see nucleated RBC’s
in severe anemia and hemangiosarcoma,
but in lower numbers and rarely ac­
accompanied by stippled RBC’s. In man and
other animals, stippled RBC’s are not
uncommon in many conditions, but in dogs
they are rarely seen to the same degree as
in lead poisoning. Polychromatophilia,

anisocytosis, poikilocytosis, and target cells
are also numerous. Lead causes the RBC to
be more fragile and shortens its life span.
The fragile RBC’s are slowly destroyed and
replaced in part by immature and abnormal
RBC’s which account for the nucleated
RBC’s and basophilic stippling. Lead in­
terrupts hemoglobin synthesis at several
steps and interferes with normal
maturation process of the RBC’s. A slowly
developing anemia may occur in the face of
hyperplastic bone marrow in a long
standing involvement (1,2,5,6).

Radiographs may be of considerable help
in making an immediate diagnosis. The
radiopaque lead-based paints will be
visualized as small flecks in the
gastrointestinal tract of the dog. Other lead
objects will also show up. These must not
be confused with gravel or chips of bone.
Examination of feces for chips of paint or
bone may suggest the nature of the sub­
stances in the gastrointestinal tract that
were seen radiographically. Lead lines may
develop in the metaphysis of the long bones

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in immature dogs with poisoning of over 10 days' duration. These lines are seen in many bones and the ribs, but are best seen proximal to the open epiphysis of the distal radius, ulna and metacarpal bones. These changes, called metaphyseal sclerosis, are the result of incorporation of lead into sites of active bone formation, causing a dense zone of mineralized cartilage and bone. However, these changes are also seen in phosphorus and Vitamin D intoxication (2).

Urinalysis may be normal but usually hyaline and granular casts are found and sometimes small amounts of protein.

Once diagnosis is made, treatment should be started immediately to remove the lead from the body. Enemas can effectively remove small lead-containing objects from the large intestine. Emetics will remove lead from the upper gastrointestinal tract. Sedatives may be given to control convulsions and other nervous signs. Lead that has been absorbed by the body can be effectively removed by chelation with Ca EDTA given subcutaneously at the rate of 12 mg/lb four times daily for five days, but not to exceed two grams per day. Caution must be used to prevent the dog from going into convulsions and showing other nervous signs by mobilizing too much lead too fast. In the severe cases, the treatment should be repeated in five days, especially if neurological signs recur (1,2,3,6). Therapy should be accompanied by urine lead analysis to monitor success of treatment.

It has been suggested that lead stored in the bones may be mobilized spontaneously by infection, strenuous exercise, severe trauma or metabolic disorders. Therefore, lead ingested by a young puppy may be stored in the body in a low level, not causing immediate clinical signs (4). A type of chronic, low level lead poisoning may appear similar to canine distemper since more often than not no source of exposure to lead is found or no evidence of exposure is found except by blood lead analysis and hematologic examination (5). In one study of 27 cases of distemper diagnosed in dogs, 14 had liver Pb of 75 ppm (4). Samples of liver obtained post mortem provide the best single diagnostic test for lead poisoning, the upper limit of normal being 3.5 ppm.
The cortex of the kidney is also a good post mortem source with normal lead level being below 1-3 ppm (1).

Lead is removed slowly from the body primarily by the kidneys. Clinical pathology shows that in the kidney, lead causes degeneration and necrosis of renal tubules which may result in chronic nephritis (5). The nervous signs come as a result of segmented degeneration which results in un-unified motor nerve conduction resulting in ataxia and uncoordinated musculoskeletal movements (1). The brain lesions include degeneration and fibrosing of vessels, hemorrhages and gliosis. Consequently, as a result of the various effects of lead poisoning, a differential diagnosis must include distemper, hepatitis, rabies, chronic nephritis, and poisoning by other toxic agents.

**BIBLIOGRAPHY**


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**Canine Vasectomy**

by

Sally McCreery*

Many people are starting to consider vasectomy as a substitution for castration in the canine species. Vasectomy is indicated in cases where owners do not want to breed the dog due to hereditary problems such as hip dysplasia. Vasectomy does not alter the male hormones as does castration. Young dogs still develop normal male characteristics and there is no problem of weight gain from decreased metabolic rate due to loss of testosterone. Intact male dogs have more drive to exercise and therefore keep in shape. Many owners also prefer not to castrate their dogs due to psychological indentification problems.

A nine month old German Shorthair was admitted to Stange Memorial Clinic, Iowa State University August 30, 1976 to undergo the vasectomy procedure. After inducing general anesthesia the area of the prepuce was surgically prepared. A 2 cm incision was made on the ventral midline of the prepuce just cranial to the scrotom. This incision was midway between and parallel to both spermatic cords permitting blunt dissection of these structures. The spermatic cords and their respective blood supply were drawn into the incision site one at a time. The ductus deferens, a white cordlike structure 3 mm in diameter, was identified. The tunica vaginalis parietalis was incised and the ductus deferens with its separate fold of tunica vaginalis was exposed. Two crushing forceps were placed on the ductus, 1 cm apart. The portion in between the forceps was excised and the severed ends were ligated with 2-0 chromic gut. The incision in the tunica vaginalis was carefully

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