

Lead intoxication in a puppy – a case report

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ABSTRACT: Lead intoxication in a puppy is described. The puppy was submitted to surgical procedure for suspicion of acute abdominal pain. Diagnosis was established on the basis of the presence of eroded lead weights in stomach, biochemical and hematological blood tests as well as by elevated tissue lead concentrations.

Keywords: lead poisoning; dog

A puppy of the Labrador breed, aged 9 months, was admitted to surgical department during the night for the symptoms of acute abdominal pain. Clinical examination revealed shallow and short breathing, weak and accelerated pulse of unmeasurable rate, pale mucosa, and distended, tympanic and sensitive abdomen. The stomach could not be reached by gastric probe. Because of such a condition, the puppy was immediately submitted to laparotomy, showing the stomach to be rotated by 180 degrees, enlarged and of tight walls. The stomach was moved to the normal position, whereafter gastrostomy was performed. On gastric fundus exploration, 16 lead weights of 1 150 g total weight, a half of them partially eroded, were found. Gastric mucosa was of pink-grayish to occasionally gray color. Upon the removal of lead weights, the stomach and gastric wall were closed by standard procedure, and gastropexy was performed (Meyer-Lindenberg, 1993). During the first two days, the postsurgical course was satisfactory, and the owner was instructed to refer to the Clinic for Internal Diseases for further treatment of the dog, which he failed to do. The owner came to the Clinic 10 days later, complaining of the dog's poor appetite and occasional vomiting. Clinical examination showed 5% dehydration, the dog looked unhappy, of tight abdomen with distended stomach filled with gases. Behind the stomach, empty, loose intestinal curvatures were palpated. Other clinical findings were within the normal limits. Blood samples were obtained for hematology and biochemistry

tests. Lead concentration was determined in the blood and urine, and therapy was initiated (Fikes and Dorman, 1994). Hematology tests produced the following findings: red blood cells 6.7 (normal $5.5\text{--}8.5$) $\times 10^{12}$; hemoglobin 158 (normal $120\text{--}180$) g/l; hematocrit 51 (normal $37\text{--}55\%$); leukocytes 35.3 (normal $6.9\text{--}17.0$) $\times 10^9$; MCV 76 (normal $6\text{--}77$) fL. Biochemistry tests showed the following findings: ALT 630 (normal < 50) U/l; AST 22 (normal < 40) U/l; AP 3020 (normal < 190) U/l; GGT 67 (normal < 6) U/l; urea 25.4 (normal $3.3\text{--}8.3$) mmol/l; creatinine 138 (normal < 115) $\mu\text{mol/l}$; total protein 83 (normal $55\text{--}75$) g/l; albumin 43 (normal $22\text{--}37$) g/l. The δ -ALAD activity on days 1, 3 and 5 of treatment was 44.73 , 15.44 and 37.76 nm PBG/ml RBC/h, respectively. (In two healthy dogs, δ -ALAD activities of 153.79 and 169.17 nm PBG/ml RBC/h were measured). Blood concentration of lead was 0.14 , < 0.04 and < 0.04 mg/kg on days 1, 3 and 5 of treatment, respectively. Urine concentration of lead was < 0.04 on day 1 and 7.25 mg/kg on day 2 of treatment. Therapy consisted of infusion of 1500 ml physiological saline; CaEDTA (50 ml *s.c.* 4 times daily or a total daily dose of 2 g dissolved in 200 ml glucosaline); thiamine (125 mg twice daily); Synulox[®] – Pfizer (amoxicillin + clavulanic acid) 225 mg; Reglan[®] – Alkaloid (metoclopramide) 10 mg *s.c.* twice daily; Peptoran[®] – Pliva (ranitidin) 50 mg *s.c.* twice daily; C vitamin 3 ml. This therapy was administered to the dog for five days. On treatment days 2 and 3, x-ray examination showed severe

esophageal hypotonia and gastric dilatation. On day 6, CaEDTA was discontinued, and the dog was referred to primary veterinarian for symptomatic therapy, with instructions to the owner on the possible need of repeat chelating therapy. On the same day, hematology and biochemistry tests were repeated, with the following results: RBC 4.97; hemoglobin 116; hematocrit 38; leukocytes 12.6; ALT 250; AST 23; AP 598; GGT 36; urea 4.6; creatinine 134; total protein 53; albumin 31. During hospital stay, the dog had poor appetite with occasional vomiting. Upon discharge, the dog condition failed to improve, vomiting persisted, the owner decided to abandon further therapy, and the dog was euthanatized. On autopsy, pale gingiva, lung perfusion, parenchymatous degeneration and perfusion of the liver, nephrotic kidneys, grayish urinary bladder mucosa, catarrhal enteritis, and hyperemia and edema of the brain were observed. Histologic examination showed catarrhal, occasionally necrotic enteritis, chronic interstitial hepatitis with degenerative alterations, tubular nephrosis, cerebral hemorrhage and encephalopathy. The brain, kidney and liver contained 0.37, 1.39 and 7.39 mg Pb/kg w/w, respectively.

On subsequent interview, the owner said that some 20 days before aggravation of the dog's condition, he had been looking for his lead weights that were subsequently removed from the dog's stomach. He could not find the weights and did not think about it anymore.

DISCUSSION

The case presented was a drastic example of lead poisoning in a puppy, confirmed by the inhibition

of δ -ALAD activity, a specific biomarker of lead exposure. The diagnosis was additionally confirmed by the increased level of lead in the dog's organs, especially liver, where the normal lead concentration is up to 1.22 mg/kg (Humphreys, 1991). On interpretation of biochemical and particularly hematologic parameters, attention should be paid to the hemoconcentration which was considerable on the initial blood sampling, ascribed to dehydration of the animal. This fact was confirmed by normalization of the levels of total protein and albumin, and overt anemia upon rehydration. Biochemical changes pointed to major liver damage, which was histologically verified on autopsy. The dog's condition would have probably improved considerably had the owner followed the surgeon's advice and referred to the Clinic for Internal Diseases after the operation, where the above mentioned therapy would have been administered on time.

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