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Authors: Zabka, Tanja S., Haulena, Martin, Puschner, Birgit, Gulland, Frances M. D., Conrad, Patricia A., et al.

Source: Journal of Wildlife Diseases, 42(3) : 651-657

Published By: Wildlife Disease Association

URL: https://doi.org/10.7589/0090-3558-42.3.651

Journal of Wildlife Diseases, 42(3), 2006, pp. 651–657

Acute Lead Toxicosis in a Harbor Seal (*Phoca vitulina richardsi*) Consequent to Ingestion of a Lead Fishing Sinker

Tanja S. Zabka,[1,5,6] Martin Haulena,[2] Birgit Puschner,[3] Frances M. D. Gulland,[2] Patricia A. Conrad,[4] and L. J. Lowenstine[1,4] [1] Anatomic Pathology Service, Veterinary Medical Teaching Hospital (VMTH), University of California, Davis, California 95616, USA; [2] The Marine Mammal Center, 1065 Fort Cronkhite, Sausalito, California 94965, USA; [3] California Animal Health and Food Safety Laboratory System–Toxicology Laboratory, University of California, Davis, California 95616, USA; [4] Department of Pathology, Microbiology and Immunology, School of Veterinary Medicine, University of California at Davis, Davis, California 95616, USA; [5] Current address: The Marine Mammal Center, Golden Gate National Recreation Area, Building 1044, Sausalito, California 94965, USA; [6] Corresponding author (email: tszabka@yahoo.com)

ABSTRACT: An adult female harbor seal (*Phoca vitulina richardsi*) stranded in northern California on 25 June 2004, exhibited progressive weakness, disorientation, and seizures, and despite therapy, died within 4 days. On pathologic examination, a lead fishing sinker was in the stomach, and changes in the brain, heart, kidney, liver, lymph nodes, and spleen were supportive of acute lead toxicosis. The diagnosis was made on the basis of concentrations of lead in the sinker (90–98% lead), antemortem whole blood (0.66 ppm), and postmortem tissues (84 ppm, wet weight liver). This first documented case of lead toxicosis in a wild marine mammal demonstrates an additional way in which human fishing activities can harm marine mammals.

Key words: Brain, heart, hemosiderosis, lead sinker, pathology, *Phoca vitulina richardsi*, pinniped, toxicology.

Significant mortality due to lead poisoning is well documented in waterfowl and waterbirds following ingestion of lead shot and sinkers, respectively (Franson, 2003; Wilson et al., 2004). Fish containing lead tackle can be a source of lead poisoning in piscivorous predators, as documented in bald eagles (*Haliaeetus leucocephalus*), common loons (*Gavia immer*), and common and red-breasted mergansers (*Mergus merganser* and *M. serrator*, respectively) (USFWS, 1994). The concentration of lead has been detected in a variety of tissues from wild marine mammals; however, nearly all concentrations were within ranges considered normal for other mammalian species (Holsbeek et al., 1998; O'Shea, 1999; Yeats et al., 1999; Julshamn and Grahl-Nielsen, 2000; Cardellicchio et al., 2002; Ikemoto et al., 2004; Ninomiya et al., 2004). In marine mammal species, concentrations of lead in bone, liver, and kidney are higher than in skeletal muscle, blubber, and other soft tissues, but a consistent trend in tissue concentrations with age or sex has not been established. Additionally, associated alterations in clinical and anatomic pathology have not been documented, except for a single report of fatal lead toxicosis in a captive, bottlenose dolphin (*Tursiops truncatus*) that ingested lead air gun pellets (Shlosberg et al., 1997).

An adult female harbor seal was presented to The Marine Mammal Center (TMMC), Sausalito, California, on 25 June 2004. The animal previously was admitted to TMMC as a neonate on 6 March 1999, and after a relatively uneventful rehabilitation period, was released on 23 May 1999. On second admission, the seal had intermittent seizures and was weak and disoriented, but could ambulate. On physical examination, the animal was moderately underweight (51.5 kg) and moderately dehydrated. Initial therapy included 200 mg phenobarbital given per os (p.o.) via gavage every 12 hr (bis in die [b.i.d.]) and ketoprofen (50 mg, p.o., b.i.d). In addition, Ponazuril (250 mg, p.o., every 24 hr) and clindamycin (300 mg, p.o., b.i.d.) were administered, because protozoal encephalitis is common in southern sea otters (*Enhydra lutis neresis*) and occurs in harbor seals in California (Miller et al., 2001; Kreuder et al., 2003).

Blood was drawn the following day, 26 June, using the method described by Bossart et al. (2001) and submitted for a complete blood cell count (Vet ABC®

hematology analyzer, Heska Corporation, Fort Collins, Colorado, USA), manual 200-cell differential count, and clinical chemistry profile (AU5200®, Olympus America Inc., Melville, New York, USA). Results were compared against published reference values (Bossart et al., 2001). No abnormalities were identified.

Despite phenobarbital therapy, a seizure occurred on 27 June, and the seal was treated with lorazepam (2.5 mg, intramuscularly). Opisthotonus, muscle fasciculations, and extended vibrissae characterized seizure activity, which became increasingly frequent by 28 June and seemed temporally associated with handling or restraint. Despite additional therapy of diazepam (7.5 mg, intravenously, b.i.d.), the seal continued to be poorly responsive and occasionally exhibited open-mouth breathing. The animal was found dead on 29 June.

Necropsy was performed within 12 hr of death, and representative tissue samples were immersed in 10% neutral buffered formalin, routinely processed for paraffin embedding, sectioned at 4–5 μm, and stained with hematoxylin and eosin (H&E). Selected tissue sections (liver, spleen, and kidney) also were stained with Prussian blue for iron to confirm hemosiderosis, and sections of kidney and liver were stained with Ziehl-Neelson and Fite's acid fast to facilitate detection of lead inclusion bodies (Sheehan and Hrapshak, 1980). Fresh liver tissue was frozen at −20 C and submitted for metal analysis along with the fishing sinker, described below, and an antemortem blood sample, which was taken on 26 June, drawn into ethylenediaminetetraacetic acid and frozen at −80 C. Cerebrospinal fluid (CSF), brain tissue, and blood were collected aseptically and submitted for protozoal culture (brain, CSF) and antibody analysis (serum).

To determine the concentration of lead in blood, whole blood was analyzed using Zeeman graphite furnace atomic absorption (Zeeman/GFAA) (PerkinElmer, Analyst 800 atomic absorption spectrophotometer, Shelton, Connecticut, USA) (Galey et al., 1990). For tissue metal analysis, the frozen liver sample was digested with nitric acid and analyzed for the following metals by inductively coupled argon plasma emission spectrometry (ICP) (ARL, Accuris Model, Thermo Optek Corporation, Franklin, Massachusetts, USA): arsenic, cadmium, iron, mercury, manganese, molybdenum, lead, and zinc (Galey et al., 1990). Accuracy of ICP was determined by analyzing standard reference materials (SRM), such as bovine liver (National Bureau of Standards and Technology, SRM 1577b) and lobster hepatopancreas (National Research Council of Canada TORT-2). Data were accepted if values obtained from SRM were within two standard deviations of the certified reference values. Metal concentrations were determined on a wet weight basis. Based on ICP analysis, confirmation analysis for lead was performed by Zeeman/GFAA. Brain tissue and CSF were cultured and serum was analyzed for antibody titers for *Toxoplasma, Sarcocystis*, and *Neospora* spp. using techniques previously described (Miller et al., 2001).

Significant findings on gross examination were limited to the stomach, spleen, and lymph nodes, and on histological examination additional changes were observed in brain, heart, kidney, and liver. Grossly, the fundic stomach contained a 112-g, 2.75-cm-long × 0.75-cm-diameter, torpedo-shaped, metallic-colored lead sinker attached to a ball of 50-cm-long monofilament line and braided stainless steel wire leader with multiple attachment sites for hooks, which either were absent or rusted. The spleen and lymph nodes were enlarged and congested with multifocal hemorrhage in the lymph nodes.

On microscopic examination, there was marked histiocytosis with erythrophagocytosis and hemosiderosis, endothelial cell hypertrophy of small blood vessels, and diffuse congestion in the lymph nodes and spleen, as well as multifocal hemorrhage

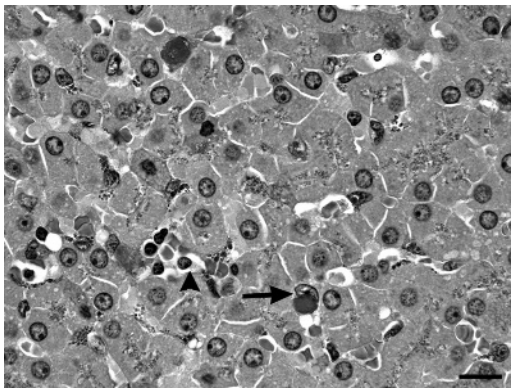

FIGURE 1. Liver from a harbor seal with lead toxicosis demonstrating diffuse hepatocellular hemosiderosis (dark stippling), Kupffer cell erythrophagocytosis and hemosiderosis (arrow), and hypertrophy of endothelial cells lining sinusoids (arrowhead), which are suggestive of hemolysis and hepatotoxicity. H&E staining. Bar=20 μm.

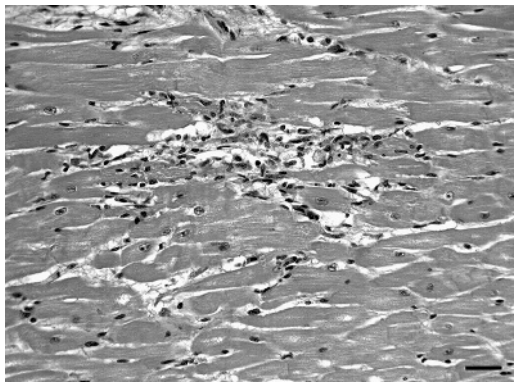

FIGURE 2. Ventricular myocardium from a harbor seal with lead toxicosis demonstrating segmental myocyte necrosis and degeneration, similar to that described in children with acute intoxication and in birds with experimental intoxication. HE. Bar=40 μm.

in the lymph nodes. In the liver, diffuse hepatocellular hemosiderosis, marked Kupffer cell hyperplasia with erythrophagocytosis and hemosiderosis, and endothelial cell hypertrophy of sinusoids were present (Fig. 1). The heart had generalized congestion and interstitial edema. The left ventricle and interventricular septum of the heart were moderately disrupted by acute, segmental myocyte necrosis and degeneration, and mild endocardial to subendocardial hemorrhage was observed (Fig. 2). Myocardial damage extended from the apex to the subvalvular region, but was most prominent from the apex to midventrical.

The meninges, choroid plexus, white matter, and to a lesser extent gray matter of the brain, had generalized interstitial and intramyelinic edema, and congestion and endothelial cell hypertrophy of small to medium blood vessels. The meninges and choroid plexus also had acute, perivascular microhemorrhage and minimal, multifocal, nonsuppurative inflammation. In the cerebrum, neurons of the laminar cortex sometimes were shrunken and darkly stained. It was uncertain whether this change was artifact due to brain removal or an early manifestation of neuronal injury (Zsombok et al., 2005).

Renal proximal tubular epithelial cells contained small amounts of hemosiderin. Lead inclusion bodies were absent on H&E-stained sections of liver, kidney, and brain and on acid-fast–stained sections of liver and kidney. Other findings were mild and considered common background lesions in this species.

CSF and brain tissue culture for protozoa were negative, and serology yielded titers for *Sarcocystis* sp., *Toxoplasma* sp., and *Neospora* sp. at dilutions of 1:640, 1:40, <1:40, respectively. Based on information in this and other marine mammals, the latter two titers are negative and the former suggests exposure to *Sarcocystis* sp. In this case, the pathologic examination did not indicate an active infection with *Sarcocystis* sp., and in another harbor seal with an active infection, a higher titer was found (Miller et al., 2001, 2002).

In addition to the clinical and pathologic findings, lead toxicosis was confirmed and was at a high level according to elevated concentrations of lead in the antemortem blood sample at 0.66 ppm and postmortem liver sample at 84 ppm wet weight. In most animal species, a blood lead concentration of less than 0.06 ppm is

nontoxic, 0.1–0.35 ppm is suggestive of subclinical exposure, and greater than 0.35 ppm is diagnostic for acute lead toxcosis (Gwaltney-Brant, 2004). Liver lead concentration in most domestic animals with lead toxicosis is greater than 10 ppm wet weight and in most aquatic birds with exposure and toxicosis is greater than 2 and 6 ppm wet weight, respectively (Franson, 2003; Gwaltney-Brant, 2004). The source of lead in this harbor seal was apparently the lead sinker, which contained between 90% and 98% lead.

After gastrointestinal absorption, lead initially is bound to erythrocytes, then is distributed to a number of tissues, including liver, kidney, lung, brain, and spleen, and later is distributed to bone. In most species, a concentration of lead in the blood greater than 0.3 ppm is diagnostic of lead poisoning and associated with clinical signs of poisoning. Due to the widespread distribution of lead within various tissues, however, the concentration of lead in blood is not necessarily reflective of the total body burden and may not correlate with the severity of poisoning. Thus, the substantially elevated concentration of lead in the liver of this harbor seal is an important finding to support acute lead toxicosis. Bone is a long-term, relatively sequestered reservoir for lead; however, lead can be released under conditions that induce bony resorption, including pregnancy, hyperthyroidism, renal disease, fractures, advanced age, and calcium deficiency. These conditions were not present on clinical and pathologic examination of this harbor seal. Accordingly, chronic exposure to lead with accumulation in and subsequent release from the bone probably was not a contributory factor to toxicosis in this harbor seal. Lead analysis of the bone would be required to confirm this inference.

In humans and animals, lead toxicity can affect every organ system, because it can inhibit and mimic the action of calcium, thus disrupting calcium-dependent pathways. Lead also interacts with proteins, including those with sulfhydryl, amine, phosphate, and carboxyl groups (Prentice and Kopp, 1985; Locke and Thomas, 1996; ATSDR, 2000). The symptoms and lesions of lead toxicosis are related to the content of lead in soft tissues; particularly the hematopoietic system, liver, kidneys, and central nervous system (CNS), where it tends to concentrate (Goyer and Rhyne, 1973). There can be species and individual variation in susceptibility and systemic response to acute lead toxicity that may be related to the dose, duration, and form of exposure to lead, the abrasiveness or protein and mineral content of the diet, gastric acidity, age, sex, stage of the reproductive cycle, body condition, metabolism, and concurrent disease (Goyer and Rhyne, 1973; Locke and Thomas, 1996). This harbor seal was a 5-yr-old, multiparous female in the progestational phase of her reproductive cycle. She was in fair body condition, had digested fish in the stomach and intestines, and was without evidence of major concurrent disease, including hyperthyroidism, renal disease, or bone fractures. The pathology, clinical, and toxicology results corroborate that this harbor seal had high-dose, acute lead toxicosis; however, any relationship between the susceptibility to lead toxicosis and the physiologic, health, and life history status of this harbor seal is speculative and would require controlled studies to discern.

Anemia is a consistent, early manifestation of acute and chronic lead toxicosis, and ultimately, but perhaps not initially, is characterized by basophilic stippling, microcytosis, hypochromasia, and reticulocytosis (Goyer and Rhyne, 1973; ATSDR, 2000). Acute toxicosis has been associated with hemolysis, but not always frank anemia (ATSDR, 2000). In this harbor seal, the hematologic profile and cytology within 1 day of presentation was nonremarkable. Four days later when the animal died, there was histologic evidence of massive, ongoing erythrocyte clearance in

the spleen, lymph nodes, and liver. This apparent discrepancy between antemortem and postmortem findings suggests that there was acute intoxication and that anemia initially was masked by a rapid, short-term contribution of erythrocytes from the splenic storage pool (Ponganis et al., 1992).

Myocardial degeneration and necrosis similar to that found in this harbor seal has been associated with acute lead toxicosis in children and in experimental lead toxicosis in birds (Goyer and Rhyne, 1973; Pattee et al., 1981; Beyer et al., 1988; Locke and Thomas, 1996). When measured in children and rats, these changes often were associated with abnormal cardiac function, which may be related to direct interference with calcium-dependent cellular processes and may be exacerbated under conditions of increased metabolic demand (Goyer and Rhyne, 1973; Prentice and Kopp, 1985). Cardiac function tests were not performed on this harbor seal.

The nervous system is the most sensitive target of lead toxicosis (ATSDR, 2000). In human adults, encephalopathy occurs less commonly than in children and usually follows acute and high-dose exposure to lead. In humans and animals, the clinical and histologic manifestations are highly variable and can occur independently of each other (Goyer and Rhyne, 1973). The typical, most prominent histologic changes, of which several were noted in this harbor seal, include cerebral edema, endothelial cell hypertrophy and hyperplasia accompanied by dilation, congestion, and sometimes perivascular hemorrhage in capillaries and arterioles, proliferation of glial cells, and multifocal neuronal necrosis and degeneration, especially in the laminar cortex and basal nuclei. The mechanism or mechanisms by which these changes occur, although they are poorly understood, may involve a direct singular or combinatorial toxic effect on endothelial cells, neurons, or glial cells, or a response to ischemic-hypoxic events and parenchymal damage. In this harbor seal, the visceral lesions appeared of slightly longer duration than the CNS lesions, even when considering the longer delay for CNS versus visceral tissue to manifest cellular insult on histology. This disparity may suggest that there was a component of CNS ischemia or hypoxia from the development of lead-induced hemolytic anemia, cardiac dysfunction, or seizure activity. Finally, although nonsuppurative inflammation in the meninges has been noted with lead toxicosis in humans, its occurrence in this harbor seal is considered unrelated due to the overall short duration of the brain lesions and its frequent occurrence as an incidental finding in pinnipeds.

Notably absent in this harbor seal were acid-fast, intranuclear inclusion bodies, which frequently are found in cases of lead toxicosis within renal tubule epithelial cells, hepatocytes, or astrocytes, or a combination of these (Goyer and Rhyne, 1973). These inclusions are hypothesized to have a protective function, because they represent lead permanently bound by host cell protein and are thus nondiffusible. Their occurrence, however, may depend on the species affected and the concentrations of lead in tissues (Goyer and Rhyne, 1973; Pattee et al., 1981; Beyer et al., 1988).

The clinical presentation, toxicology results, and pathologic findings in this harbor seal are consistent with fatal, acute, high-dose lead toxicosis consequent to ingestion of a lead fishing sinker. Based on the type of fishing tackle and foraging behavior of harbor seals, this seal likely ingested a fish attached to the tackle, which is used by both recreational and commercial fisherman for rock fishing. The dimensions of the tackle presumably prevented its movement through the pyloric sphincter of the seal's stomach with consequent gastrointestinal absorption of lead. Human fisheries interactions, such as gillnet entrapment, are a well-recognized threat to marine mammals

(Julian and Beeson, 1998). Now, similar to the problem originally recognized in waterfowl and waterbirds that ingest lead shot and lead sinkers, respectively, this first documented case of lead toxicosis in a wild marine mammal demonstrates that ingestion of lead fishing tackle can harm marine mammals.

The authors thank the histotechnicians at the UC Davis–Veterinary Medicine Teaching Hospital, the technicians of the California Animal Health and Food Safety Laboratory System, and the staff and volunteers at The Marine Mammal Center, Sausalito, California. Funding for support of this work was provided by the John H. Prescott Marine Mammal Rescue Assistance Grant Program of the National Marine Fisheries Service.

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Received for publication 31 March 2005.