TREATING LEAD POISONING IN DOGS

Author : Lisa Gardbaum

Categories : Vets

Date : December 6, 2010

Lisa Gardbaum discusses diagnosis and treatment of lead toxicity in companion animals – an issue that is, thankfully, rarer now than in previous years

A THREE-YEAR-OLD female neutered collie-cross dog had been referred with a six-week history of vomiting, diarrhoea, weight loss, dullness, anxiety and frequent adoption of the "praying" stance (Figure 1).

Two weeks earlier, the owners had reported that the animal had undergone a generalised tonic-clonic seizure. One of the owners also mentioned that he worked at a lead plant and took the dog to work each day.

Routine blood tests for haematology and biochemistry showed mildly reduced cholesterol levels at 2.1mmol/L (2.5-0.6), but were otherwise unremarkable. Bloods were submitted for canine pancreatic lipase, and basal and postprandial bile acids, and were all within normal ranges. Levels of B12 and folate were both reduced at 142pmol/L (200– 408) and 7.6nmol/L (12-30) respectively and blood levels of lead were markedly raised at 5.13µm pmol/L (0-1.2).

Abdominal radiographs revealed the presence of small radiodense particles in the small intestine (Figure 2). Stomach and duodenum endoscopy had been carried out while awaiting the results of the lead levels – and was grossly unremarkable. Endoscopic gastric and intestinal biopsies showed evidence of a moderate lymphocytic-plasmacytic gastritis and enteritis.

Treatment with intravenous fluids, amoxycillin-clavulanate (20mg/kg three times daily intravenously), ranitidine (2mg/ kg twice daily intravenously), maropitant (1mg/kg once daily

subcutaneously) and vitamin B12 (0.5mg subcutaneously weekly for four weeks) was given.

Once lead toxicity had been confirmed from the blood lead levels, treatment with sodium calcium edetate at 75mg/kg day, divided into four doses and provided as a one per cent solution (10mg/ml diluted with five per cent dextrose saline) subcutaneously for five days, was also started.

Within two days there was a rapid improvement in demeanour and appetite and no further seizures occurred. After five days the dog was discharged from the hospital, with oral ranitidine (2mg/kg) prescribed. Repeat lead levels taken three weeks after treatment were reduced markedly to 1.75µmol/L (0-1.2). The dog remained symptom-free, so no further treatment was given.

Lead toxicity

The incidence of lead toxicity has reduced in frequency over the past 30 years, due to the removal of lead from paints, petrol and other household items and is now reported to account for less than one per cent of reported accidental toxicities.

It may still result from ingestion of old metal tubing, golf balls, curtain weights, linoleum, or paint chips and dust caused by renovation of buildings painted with high-lead-content paint.

The degree to which lead is absorbed will vary, depending on the form of lead and age of the animal. Young animals absorb up to five times more lead following ingestion than older animals. Lead absorption will also be increased in animals deficient in calcium, zinc, iron and vitamin D. Lead pellets embedded in muscle and other soft tissue are not significantly absorbed and are unlikely to result in lead toxicity.

Once absorbed, lead is carried in the blood, predominantly on red blood cells, with a smaller amount bound to albumin or free in the plasma. Lead is distributed in the tissues, especially bone, teeth, liver, kidney, brain and spleen. Bone serves as a storage depot for lead. It can cross the blood-brain barrier and concentrate in the grey matter, especially in younger animals. The majority of ingested lead is excreted unchanged in faeces. The lead in the blood is excreted in the kidneys through the glomeruli and is taken up by the tubules, which are sloughed into the urine over time.

The predominant clinical signs are non-specific gastrointestinal problems, such as vomiting, diarrhoea, weight loss, inappetence and abdominal discomfort.

Neurological signs are less common and are thought to occur by decreasing cerebral blood flow and altering neuronal energy metabolism. The most common neurological signs are behavioural changes, agitation, ataxia, tremors and seizures. Less common clinical signs seen with lead toxicity include aggression, blindness, pica, polyuria, polydipsia and megaoesophagus (in cats)[1].

Diagnosis

The diagnosis of lead poisoning will largely depend on a combination of a history of possible lead ingestion, radiography to identify radiodense opacities in the gastrointestinal tract, haematological findings, blood lead levels and compatible clinical signs.

Haematological abnormalities that may be seen in lead toxicity cases include anaemia, increased nucleated red blood cell numbers and basophilic stippling (Figure 3).

These findings, however, are not consistently found, especially in acute cases, and are not pathognomonic for lead toxicity. They are also less commonly seen in affected cats than dogs. Blood lead levels greater than 2.9µmol/L are considered diagnostic for lead toxicity. However, fluctuating blood levels can occur. They may not be indicative of total body levels and the level of lead may not correlate with the degree of clinical signs.

Treatment

Treatment of lead toxicosis involves removal of the source of lead, supportive care and chelation therapy. Removal of any lead from the gastrointestinal tract is essential before starting chelation treatment, since most chelators (except succimer) will enhance the absorption of lead from the gastrointestinal system.

Sulphate-containing cathartics, such as magnesium or sodium sulphate, may help empty the gastrointestinal tract and cause the lead to precipitate as lead sulphate, which is not readily absorbed. Surgical or endoscopic removal of larger leadcontaining objects may be necessary. Cats, which often succumb to lead toxicosis through grooming lead-containing materials from their coat, should be thoroughly bathed.

Chelation therapy works by binding the lead into a soluble form that can be rapidly excreted by the kidneys. Most of the chelators and also chelated lead are potentially nephrotoxic. It is, therefore, vital to check renal parameters before and during treatment, and to ensure that hydration is maintained.

It is suggested that animals with high blood lead levels that are asymptomatic should not be treated with chelators since they can cause temporary elevation of blood lead levels and precipitate clinical signs. In these cases removal of the source of lead will allow the animal to gradually eliminate lead on its own. Also, this effect of chelation treatment may cause a temporary worsening of clinical signs in symptomatic animals, and more intensive supportive treatment may be necessary.

Chelators include sodium calcium edetate, penicillamine and succimer. Sodium calcium edetate is the drug most commonly used due to its effectiveness and appears to be the easiest to obtain (Figure 4). The doses for dogs are as outlined above and are given for two to five days. Improvement is typically seen within 24 to 48 hours. If signs persist after five days of treatment the course can be repeated after a five-day rest period.

Potential side effects include nephrotoxicity, gastrointestinal signs and pain at the injection site. Oral zinc supplementation may reduce gastrointestinal side effects.

The dose for cats is 27.5mg/ kg every eight hours for five days diluted in 15ml of five per cent dextrose subcutaneously.

Penicillamine can be given orally but is associated with side effects, such as vomiting, anorexia, pyrexia, lymphadenopathy and blood dyscrasias. Reported doses for dogs are 100mg/kg/day orally before food for seven days. The course can be repeated if necessary after a seven-day rest period. The dose for cats is 125mg/cat orally every 12 hours for five days.

Succimer has the advantage of being given orally, is much less likely to be nephrotoxic and it does not bind essential nutrients, such as copper, zinc and iron, as penicillamine does. It also does not enhance the absorption of lead from the gastrointestinal system and is associated with fewer gastrointestinal side effects. The dose is 10mg/kg orally every eight hours for 10 days, but unfortunately it appears to be difficult to obtain[2].

Thiamine (2mg/kg/day to 4mg/kg/day subcutaneously) is reported to alleviate clinical manifestations and reduce tissue deposition of lead. It can be combined with calcium sodium edetate.

Prognosis

The prognosis is good in animals with mild to moderate signs that are treated appropriately and promptly, but is worse in animals showing severe central nervous system signs.

Despite its rarity, lead toxicity should always be considered a differential with unexplained gastrointestinal signs and abdominal pain in cats and dogs. This is especially true if the history suggests the possibility of exposure to lead containing materials, if radiodensities are seen in the gastrointestinal tract on radiographs or any concurrent neurological signs are present, as early treatment can be very effective and rewarding and prevent life-threatening complications from developing.

References

- 1. Maddison J E, Allen G S (1990). Megaoesphagus attributable to lead toxicosis in a cat, *JAVMA***197**(10): 1,357-1,358.
- 2. Ramsey D T et al (1996). Use of orally administered succimer (meso-2,3-dimercaptosuccinic acid) for treatment of lead poisoning in dogs, *JAVMA***208**(3): 371-375.