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Ataxia & Vomiting in a German Shepherd

Trigger, a 1.5-year-old neutered male German shepherd dog, was presented for ataxia and vomiting.

History. Trigger was clinically normal when his owner left for work in the morning and he had no history of medical problems. When the owner returned 8 hours later, Trigger was ataxic and multiple piles of vomitus were present. The owner rushed Trigger to a local emergency clinic.

Physical Examination & Laboratory Results. On presentation, the clinician noted hypersalivation, mild muscle tremors, and ataxia. Vital signs were within normal limits. Serum biochemistry was unremarkable except for a blood glucose level of 45 mg/dL.

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ASK YOURSELF...

What are potential causes of hypoglycemia in an adult dog?

A. Ingestion of diabetic medications, such as sulfonylureas
B. Ingestion of food, gum, or drinks containing xylitol
C. Ingestion of supplements, such as alpha-lipoic acid (ALA) or cinnamon
D. Addison's disease (hypoadrenocorticism)
E. All of the above

Correct Answer: E
All of the above

Hypoglycemia can result from medical conditions, such as liver disease, insulin overdose in a diabetic animal receiving exogenous insulin, Addison's disease, or an insulinoma. Signs can be gradual or vague. However, acute onset of ataxia, weakness, and seizures with profound hypoglycemia is frequently a clue that the dog may have ingested a hypoglycemic agent.

Sulfonylureas are frequently prescribed medications to treat type 2 diabetes. Xylitol is a sweetener that does not affect insulin levels in humans but causes insulin release in dogs. Neutraceuticals and holistic products are used by many people to treat their own medical conditions; they often turn to supplements such as ALA (an antioxidant used to treat human immunodeficiency virus, diabetes, liver ailments, diabetic polyneuropathy, cataracts, and other conditions) or cinnamon (for treating type 2 diabetes and fungal diseases).

After reviewing the results of the blood analysis, the clinician asked the owner about potential hypoglycemic agents in the house. The owner, a type 2 diabetic, was taking metformin (a diabetic medication that does not normally cause hypoglycemia), ALA, other supplements, and vitamins. The owner called home and her husband confirmed that Trigger had ingested 10 to 12 300-mg ALA capsules.

Toxicity. ALA, also known as thioctic acid, is a naturally occurring substance found in spinach, broccoli, potatoes, yeast, liver, and skeletal muscle. ALA is an antioxidant in both fat- and water-soluble tissues. It is synergistic with insulin, resulting in reduced blood sugar and increased liver glycogenesis; it also facilitates uptake of glucose into cells.

ALA has been used to treat diabetes in dogs and cats in clinical practices that use alternative therapies for disease. It is also used to treat cognitive dysfunction in aging dogs. The therapeutic

dose for cats is 1 to 5 mg/kg body weight, with a maximum dosage of 25 mg/day. The therapeutic dosage in dogs is up to 200 mg per day, although large dogs should receive no more than 80 mg. Cats are 10 times more sensitive to the effects of ALA than dogs.

Diagnosis. Clinical signs of ALA toxicity or overdose are consistent with hypoglycemia. Hypersalivation, vomiting, and ataxia are the most commonly reported clinical signs; tremors and seizures may also be seen. Clinical signs have been reported to develop 30 minutes to several hours after ingestion.

Clinical pathology generally reveals hypoglycemia, which may be profound. Increased liver enzyme levels may develop 24 to 72 hours after ingestion, and acute renal failure may also occur.

Differential Diagnoses. If a patient presents with profound hypoglycemia, consider other medical and nutraceutical agents, including sulfonylurea diabetic agents (glipizide, glyburide), nutraceuticals (cinnamon), and nutritional additives (xylitol). Toxins that can manifest as hypoglycemia include *Amanita* species mushrooms or sago palms (*Cycas* species). Hypoglycemia has multiple causes, including insulinoma, hypoadrenocorticism, malnutrition, parasitism, and end-stage liver disease.

Treatment. If a dog is not showing clinical signs and ingestion of ALA occurred less than an hour prior to presentation, induce emesis. The effectiveness of the administration of activated charcoal to treat ALA overdose is unknown. Baseline serum biochemistry levels, especially of glucose, liver enzymes, phosphorus, potassium, blood urea nitrogen, and creatinine, should be obtained.

If emesis is effective (ie, pills are recovered) and the calculated dose of ALA is low, the dog can be managed at home with frequent small meals and owner observation. If, however, the dog is showing clinical signs and the calculated dose of ALA is greater than 30 mg/kg, start 2.5% to 5% dextrose as an IV continuous rate infusion (CRI).

If a cat has ingested ALA and the calculated dose is greater than 5 mg/kg, start 2.5% to 5% dextrose as an IV CRI. In both dogs and cats, measure blood glucose levels every 2 to 4 hours for 12 hours and reevaluate liver enzymes in 48 hours. Hepatoprotective agents can be used, including S-adenosylmethionine (SAMe), milk thistle, and N-acetylcysteine. Fluid diuresis maintains renal perfusion and helps with drug elimination.

High doses of ALA (20 mg/kg) have been reported to be fatal to mice and rats with a

ALA = alpha-lipoic acid; CRI = constant rate infusion, SAMe = S-adenosylmethionine

known thiamine deficiency. Deaths were prevented when thiamine was given with toxic doses of ALA. It is unknown whether thiamine is protective in other species or helpful in animals with normal thiamine levels, but addition of it to IV fluids may be of benefit.

Treatment end point is achieved when clinical signs have resolved and blood glucose levels have normalized

Case Follow-Up. Trigger was treated with an IV CRI of dextrose and his blood glucose levels were normal 10 hours after exposure. He was released to the owner 24 hours after presentation and SAMe and silymarin were given for 2 weeks. Follow-up blood analysis obtained 72 hours after exposure was within normal limits.

See Aids & Resources, back page, for references, contacts, and appendices.
Article archived on www.cliniciansbrief.com.

Tx at a glance

Patients Without Clinical Signs

- Emesis (in dog not showing clinical signs and if less than 60 min after exposure)
- Activated charcoal (effectiveness unknown at this time)
- Frequent, small meals if emesis is successful
- Recheck blood glucose in 3–4 hours because signs may manifest a few hours after exposure

Patients With Clinical Signs

- Baseline serum biochemical panel
- Dextrose bolus followed by CRI
- Monitor blood glucose Q 2–4 H
- Hepatoprotection
  - SAMe: 18 mg/kg
  - Silymarin: 50–250 mg/kg
  - N-acetylcysteine: 140 mg/kg loading dose, 70 mg/kg Q 6 H for 6 treatments
- Thiamine: 2 mg/kg
- Fluid diuresis