Xylitol Alert

The June, 2011 issue, (Vol 15, No 2) of AHL Newsletter from the Animal Health Laboratory at the University of Guelph has a case report entitled *Xylitol toxicity in a Golden Retriever*.

The report outlines the rapid decline and death of a 6-year-old dog due to hepatic necrosis induced by eating some muffins baked with xylitol in place of sugar. The article includes the following passages in bold print:

"Once thought to only induce hypoglycemia in dogs, xylitol can also produce life threatening acute hepatic necrosis."

"Xylitol doses >0.1 g/kg may cause hypoglycemia, and doses of >0.5 g/kg may result in hepatotoxicity. It is unclear whether hepatic failure is truly dose dependent or idiosyncratic reaction."

"It is imperative to educate dog owners about the potential consequences of xylitol consumption, and to raise awareness of the increased use of xylitol within foodstuffs."

I have attached a copy of the page on which this article appears so that you can read the entire thing for context.

Why am I bothering to mention this at all? Well there are two concerns.

1. I have also attached a promotional flyer that arrived in our Henry Schein order recently, describing an oral rinse for human patients that has, as its main active ingredient, xylitol. If this product gets traction in the human dental field, I can see owners also trying it on their dogs. This would be an exceedingly bad idea and so we need to make sure we are ahead of this issue (pro-active) rather than trying to catch up after a bunch of dogs have died (reactive).

2. You should also be aware that some veterinary products (AquaDent™ and Breathalyzer™) also contain xylitol as the active ingredient. The makers of these products state that the concentration of xylitol in their products, when diluted per label instruction, would not constitute a toxicity risk. But the toxicity may not be dose dependent, so…

3. It would be rather contradictory and confusing for you to inform your clients of the potentially fatal risk of allowing their dogs to have access to xylitol and then turn around and recommend a product whose active ingredient is xylitol!

What is Missing?

healthymouth™ **products contain no xylitol, no alcohol, no sugar, no industrial dyes. What they do contain is a mixture of ingredients that have been proven in multiple studies to been a valuable aid in plaque control. Hence the seven VOHC seals of acceptance for helping to control plaque**

THIS PRODUCT COULD KILL DOGS!

COMPANION ANIMALS

Xylitol toxicity in a Golden Retriever

Kristiina Ruotsalo

A 6-year-old Golden Retriever was presented to the referring veterinarian approximately 24 hours after consuming an unknown number of muffins prepared with xylitol. The dog was seizuring upon presentation and developed hematomas at venipuncture sites. CBC results revealed mild hemoconcentration, mild thrombocytopenia (platelet count 109 x 10^9/L), mild mature neutrophilia and lymphopenia. The serum biochemistry profile revealed numerous alterations in liver parameters including a profound increase in ALT 27,790 U/L (reference interval 19-107 U/L), a mild to moderate increase in ALP 499 U/L (22-143 U/L), moderate increases in both free bilirubin 25 µmol/L (0-3 µmol/L) and conjugated bilirubin 39 µmol/L (0-1 µmol/L), along with a mild increase in GGT 17 U/L (0-7 U/L). Serum bile acids were 560 µmol/L (0-6 µmol/L). Serum glucose was decreased at 2.2 mmol/L (3.3-7.3 mmol/L), as were serum potassium 3.1 mmol/L (3.8-5.4 mmol/L) and chloride 93 mmol/L (104-119mmol/L). Mild azotemia was noted. Prothrombin time and partial thromboplastin time were markedly increased at > 100 seconds. Due to the poor clinical prognosis, the dog was euthanized.

Xylitol is a 5-carbon sugar alcohol which occurs naturally in low levels in fruits and vegetables. It is being increasingly used as a sugar substitute in chewing gum, candy, nicotine gum, toothpastes, baked goods, and is also available in a granulated format for home cooking. The increased popularity of xylitol is based on the fact that it is as sweet as sucrose but contains only two-thirds the calories of sugars. It causes little insulin release in humans, and is thus suitable for low-carbohydrate and low-glycemic-index diets. Xylitol has been shown to inhibit oral bacterial growth, thus exhibiting anticariogenic properties.

Once thought to only induce hypoglycemia in dogs, xylitol can also produce life threatening acute hepatic necrosis. Following ingestion, vomiting is typically noted. Hypoglycemia may then develop within 10 to 60 minutes due to xylitol's potent insulin stimulatory effects. Delayed hypoglycemia may occur with some cases of xylitol gum ingestion. Clinical signs may then progress rapidly from lethargy to ataxia, collapse and seizure activity. **Xylitol doses >0.1 g/kg may cause hypoglycemia, and doses of >0.5 g/kg may result in hepatotoxicity. It is unclear whether hepatic failure is truly a dose dependent or idiosyncratic reaction.** Hepatic damage may be noted as soon as 9-12 hours post-ingestion, or be delayed up to 72 hours. Hepatic necrosis may result in exacerbation of hypoglycemia and secondary disseminated intravascular coagulation. Depression, vomiting, icterus, melena, diarrhea, petechiation/ecchymosis, and hepatic encephalopathy may be noted with clinical disease progression.

It is imperative to educate dog owners about the potential consequences of xylitol consumption, and to raise awareness of the increased use of xylitol within foodstuffs. *AHL*

Cocoa mulch and dogs (theobromine poisoning)

Brent Hoff, Nick Schrier

Cocoa bean shells are frequently used in horticulture as mulch. Cocoa mulch, which is sold by most garden supply stores, contains large amounts of theobromine and caffeine, which are toxic to pets. Due to their indiscriminate eating habits, dogs of any age or breed can be affected by ingesting this material. Cats are less likely to eat the material, because they are not able to sense the sweet taste.

Theobromines are methylated xanthine alkaloids (methylxanthines) that cause a variety of clinical signs, including vomiting, diarrhea, PU/PD, ataxia, cardiac arrhythmias, CNS stimulation and potentially death. Dogs are the most frequently intoxicated species, but cats, pigs, horses, birds and other animals can also be affected.

Domestic animals metabolize theobromine much more slowly than humans. Dogs and other animals can easily consume enough of the mulch to cause serious poisoning.

Theobromine is slowly absorbed, reaching peak plasma levels in approximately 10 hours. It is metabolized within the liver and has a half- life of approximately 17 hours, so in severe cases, **clinical signs of theobromine poisoning can persist for 72 hours**. The LD_{50} of theobromine in dogs is 140 mg/kg. Cocoa beans contain approximately 1.2% theobromine by weight and a handful of this material could cause bradycardia or tachyarrhythmia and possibly death of a pet.

Chemists with the USDA are investigating the use of theobromine as a toxicant to control coyotes that prey on livestock. *AHL*

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