

Case study – chocolate toxicity in a dog

Case history

A 4 year old spayed female Labrador weighing 21kg was presented early on Easter Monday morning for ataxia and muscle tremours. She had been normal the night before and her owners had been woken by sounds of her stumbling and vocalisation. She had vomited a small amount of thick dark brown liquid containing some small pieces of silver wrapper during the night and had urinated in the house.

Clinical examination

On clinical examination the dog was restless, ataxic and exhibiting mild generalised muscle tremours. She had pale mucous membranes, a capillary refill time of approximately 2 seconds and a heart rate of 160 beats per minute with a normal sinus rhythm. She was panting, had a temperature of 39.2°C and discomfort on palpation of the cranial abdomen.

Diagnosis and treatment

The combination of the clinical findings and presence of silver wrapper in the vomitus lead to a presumptive diagnosis of chocolate toxicity. This was later confirmed by the owners noting that two large solid chocolate Easter rabbits and some smaller eggs had disappeared. All were thought to have been milk chocolate but this could not be verified as the wrappers were not available for examination.

Emesis was induced using apomorphine (details? - formulary) and a moderate amount of dark brown material was vomited up. Activated charcoal was given at 2g/kg PO. Tremours were controlled using diazepam at 0.5 mg/kg slow IV. Food was withheld but water offered. In the first 12 hours there was little change in the dog's clinical status, after which time the muscle tremours diminished over a period of approximately six hours, her heart rate returned to 120 beats per minute and her temperature dropped to 38.2°C. Her cranial abdomen became soft and no pain was elicited on palpation. The dog was discharged from the hospital after 36 hours on a bland diet and made a full recovery.

Discussion

Chocolate is derived from the seeds of the plant *Theobroma cacao*. The seeds contain substances known as methylxanthines, which include the CNS stimulants theobromine and caffeine, both of which are toxic to animals. The potential lethality of these substances is illustrated by their investigation as toxicants for the control of pest coyotes in the United States¹.

Although both constituents may contribute to clinical signs of chocolate toxicity, the former is the major cause as its concentration in chocolate is 3-10 times higher than that of caffeine² and its half life significantly longer (17.5 hours and 4.5 hours respectively)

The concentration of theobromine varies depending on the type of chocolate:

White chocolate	0.03 mg/g
Milk chocolate	2.3 mg/g
Dark chocolate	5.5 mg/g
Cooking chocolate	16 mg/g

Individual sensitivity to methylxanthines varies³ but in dogs generally mild clinical signs such as vomiting and diarrhoea can be expected after ingestion of approximately 20mg/kg, effects of cardiotoxicity at approximately 45 mg/kg and seizures after a dose of approximately 60mg/kg. The LD₅₀ for dogs is 100-200mg/kg, which would be approximately 10 standard 150g blocks of milk chocolate for a 10kg dog. The LD₅₀ for cats is lower than that for dogs but as cats are much more discriminating eaters they are less commonly affected.

Mechanism of action

Theobromine is readily absorbed by the GI tract and widely distributed around the body. It is metabolised in the liver and undergoes enterohepatic recycling. Its major effects are thought to be caused by competitive inhibition of cellular adenosine receptors, which results in CNS stimulation, tachycardia through direct myocardial stimulation, and diuresis. Another mechanism of action is inhibition of cellular calcium reuptake which leads to increased strength and contractility of skeletal and cardiac muscle. Theobromine also inhibits phosphodiesterase, leading to an increase in cAMP and thus an increased level of catecholamines⁴.

Clinical findings

Clinical signs usually occur within 6-12 hours of chocolate ingestion². Initial signs usually include vomiting, diarrhoea, abdominal distension, polydipsia and restlessness. Signs may then progress to polyuria, ataxia, hyperthermia, tremours and seizures. Tachypnoea, bradycardia or tachycardia and in more severe cases premature ventricular contractions may be seen. Due to the high fat content of chocolate, pancreatitis is a potential sequela 24-72 hours after ingestion⁴. Death usually results from respiratory failure or cardiac arrhythmias⁴.

Diagnosis

Diagnosis is usually based on the presence of clinical signs coupled with a history of exposure. If there is no known exposure, differential diagnoses may include toxicity caused by the following substances: organophosphates, carbamates, metaldehydes, 1080, strychnine, nicotine and amphetamines⁵.

Treatment

There is no antidote to theobromine and treatment is symptomatic. Prompt removal of chocolate from the stomach, either by emesis or gastric lavage is highly beneficial and may even be useful several hours after the time of ingestion as serum theobromine concentrations measured in a dog were shown to peak approximately 4 hours after ingestion and decline extremely slowly over the following 24 hours⁶. Activated charcoal should be given either orally or by stomach tube. This is beneficial because of the enterohepatic recirculation of theobromine and because chocolate may remain in the stomach for relatively long periods of time due to either being consumed within its wrapper or forming a large mass within the stomach that is not easily removable by vomiting or gastric lavage.

Diazepam is the initial drug of choice for muscle tremours and seizures; barbiturates may be required in refractory cases. Arrhythmias should be treated as needed with propranolol used for tachyarrhythmias, atropine for bradyarrhythmias and lignocaine in cases with persistent premature ventricular contractions. In animals showing abnormalities of cardiac rhythm, electrocardiography should be used to monitor cardiac status.

Administering fluids at twice maintenance will assist with stabilising cardiovascular function and enhance urinary excretion of methylxanthines and their metabolites. As these can be absorbed across the bladder wall, a urinary catheter should be placed in severe cases⁷.

Other symptomatic treatments that may be required include thermoregulation and correction of acid/base and electrolyte abnormalities. Severely affected patients should be monitored for complications such as pancreatitis.

Although the long half-life of theobromine in the dog can result in clinical signs persisting for up to 72 hours, animals given appropriate therapy have an excellent chance of making a full recovery.

References

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