

Popular Article

Chocolate Poisoning in Pets

Utpal S. Boro¹, Swatantra Kumar Singh¹, Rajeev Ranjan¹,
Arpita Shrivastav¹, Neelam Tandia², Priya Singh^{2*}

¹Department of Veterinary Pharmacology, College of Veterinary Science and Animal Husbandry, Rewa, M.P.,
486001, India

²Department of Veterinary Surgery and Radiology, College of Veterinary Science and Animal Husbandry, Rewa,
M.P., 486001, India

*Corresponding author: Priya Singh, Email: priyasinghvets@gmail.com

charcoal @ 1-4 g/Kg orally.

Abstract

Chocolate poisoning/toxicosis is a syndrome resulting from ingestion of excessive amount of methylxanthine containing chocolate products. Chocolate is derived from roasted seeds of *Theobroma cacao*. The seeds contain substances known as methylxanthines, which includes the CNS stimulants theobromine (3, 7-dimethylxanthine) and caffeine (1, 3, 7-trimethylxanthine), both of which are toxic to dogs. Chocolate poisoning occurs most commonly in dogs, but many other species are also susceptible. The LD50 of both theobromine and caffeine is 100-200mg/kg. clinical signs include vomiting, restlessness, agitation, hyperthermia, tachycardia and in severe cases seizures. Treatment is aimed at reducing the cardiovascular and CNS effects via administration of antiarrhythmics (e.g. Propranolol @ 0.02-0.06mg/Kg slow I/V) and sedatives (e.g. Diazepam @ 0.5-2mg/Kg slow I/V), maintenance of fluid & electrolyte balance and if presented to clinics within 1 hour of ingestion of chocolate, the animal should be decontaminated via inducing emesis (apomorphine) and administering activated

Introduction

Chocolate is derived from the roasted, ground seeds of *Theobroma cacao* tree and contains the methylxanthine theobromine and caffeine. Theobromine acts as a diuretic, heart stimulant, blood vessel dilator and smooth muscle relaxant in animals and humans. Dogs metabolize theobromine very slowly as well as undergoing enterohepatic cycling, thereby enhancing their effects and leading to toxicity. The amount of methylxanthines contained depends on the type of chocolate: Chocolate with a high cocoa content, such as baking chocolate and dark chocolate, contains a significantly higher concentration of methylxanthine than milk chocolate (Zoumas *et al.*, 1980; Dolder, 2013). The content of methylxanthines in white chocolate is considered too low for inducing intoxication. Chocolate ingestion is a common cause of canine poisonings and can lead to illness and death. The first report on chocolate intoxication in dogs was published in 1942 (Clough, 1942). Of tracking the effectiveness and safety of pharmaceuticals while also monitoring the reaction to treatment. They also offer a fantastic opportunity to create and develop

these combination agents, enabling the delivery of medicines and the application of detection techniques both prior to and during the course of therapy. One of the most promising and advantageous applications of nanotechnology is in the field of nano-pharmaceutics, which has many benefits for veterinary science.

Sources of Chocolate Poisoning

It is obvious that a dog cannot go to a shop and buy a chocolate themselves, so it often an owner who most of the time is unaware of the toxic effects of chocolate bar, chocolate cake, coffee, etc. on dogs that give it to their pets as a treat or the pets may consume it accidentally while scavenging for food in garbage bins eating left over chocolate bars, wrappers, cocoa powder or rarely cocoa bean hulls.

Pharmacokinetics of Theobromine and Caffeine

Caffeine reaches maximum serum levels within 30 to 60 minutes after oral intake. Theobromine, on the other hand, is absorbed more slowly compared to caffeine (maximum plasma concentration after about 2 hours). Furthermore, caffeine is metabolized to theobromine (maximum plasma concentration after 6 to 8 hours) (Löffler *et al.*, 2000a, 2000b). Both methylxanthines are metabolized in the liver, excreted via the bile ducts and undergo enterohepatic circulation (Dolder 2013). Methylxanthines are excreted in the urine as metabolites and unchanged parent compounds. The half-lives of theobromine and caffeine in dogs are 17.5 hours and 4.5 hours, respectively. The exact amount of methylxanthines in chocolate varies because of the natural variation of cocoa beans and variation within brands of

chocolate.

The LD₅₀ of both caffeine and theobromine is reportedly 100–200 mg/kg, but severe signs and deaths may occur at much lower dosages, and individual sensitivity to methylxanthines varies. In general, mild signs (vomiting, diarrhea, polydipsia) may be seen in dogs ingesting 20mg/kg, cardiotoxic effects may be seen at 40–50 mg/kg, and seizures may occur at dosages ≥ 60 mg/kg.

Pharmacodynamics of Theobromine

Theobromine and caffeine inhibit cellular adenosine receptors, which leads to stimulation of the CNS, tachycardia and diuresis. Furthermore, methylxanthines increase intracellular calcium concentration by increasing intracellular calcium influx and decreasing intracellular sequestration of calcium into the sarcoplasmic reticulum of striated muscles. This results in increased contractility of the skeletal muscles. A further effect of methylxanthines is an inhibition of the phosphodiesterase and an increase in cAMP concentration (sympathomimetic effect). Methylxanthines also increase the concentration of epinephrine and norepinephrine in the blood.

Clinical Signs of Chocolate Poisoning

Vomiting and Diarrhea

These two problems are the earliest signs of chocolate toxicity in dogs. In light cases, dogs may simply vomit or have diarrhea a few times and then feel better. However, in severe cases, these symptoms may progress further to the others on the list below.

Excessive thirst and Urination

Since both theobromine and caffeine are diuretics, dogs may show both excessive thirst and urination after ingesting too much of either of these substances.

Restlessness

Caffeine, in particular, may cause dogs to behave restlessly. Darker chocolates with higher caffeine contents may lead to this symptom even without the dog ingesting very much of it.

Tachycardia/Arrhythmia

An elevated heart rate is a dangerous symptom that could lead to cardiac arrest, especially in older dogs or those with underlying conditions.

Seizures

Seizures are one of the most severe symptoms of extreme chocolate toxicity in dogs. This may only occur when a dog has ingested a large quantity of chocolate, but it can sometimes mean the toxicity will become fatal without veterinary treatment. If a dog has ingested a potentially toxic amount, they will begin to show signs and symptoms of poisoning between four to 24 hours after eating the chocolate.

Early signs are mild and include:

- Gastrointestinal upset
- Vomiting
- Diarrhoea
- Excessive urination
- Increased thirst

Signs can progress to include:

- Hyperactivity
- Increased heart rate
- Hyperthermia
- Pancreatitis

The more severe signs include:

- Depression
- Heart arrhythmias and heart failure
- Tremors
- Seizures
- Coma

Diagnosis of Chocolate Toxicosis

Diagnosis of chocolate toxicosis is based upon history of chocolate consumption and display of clinical signs. A thorough history needs to be taken to determine whether the animal has gotten into trash, cocoa shell yard mulch, or other toxic substances or whether another underlying cause of symptoms is present. Differential diagnosis must be made from amphetamine toxicosis, cocaine toxicosis and ingestion of anti-histaminic, antidepressants or other CNS stimulants.

A complete physical exam will help in diagnosis. Blood analysis (complete blood cell count and chemistry) and urinalysis will aid in detection of disease or organ failure. Electrocardiography (EKG) can detect heart arrhythmias and abnormalities. Radiographs may aid in ruling out other causes for symptoms.

Treatment of Chocolate Toxicosis in Animals

- Management of potentially life-threatening clinical signs
- Symptomatic and supportive care

Stabilization of symptomatic patients is a priority in treating chocolate toxicosis. Methocarbamol (50–220 mg/kg, slow IV; no more than 330 mg/kg per day) or diazepam (0.5-2 mg/kg, may be used for tremors and/or mild seizures; barbiturates

or other general anesthetics may be required for severe seizures. Arrhythmias should be treated as needed: administration of propranolol (0.02–0.06 mg/kg, slow IV) or metoprolol (0.2–0.4 mg/kg, slow IV) for tachyarrhythmias, atropine (0.01–0.02 mg/kg) for bradyarrhythmias and lidocaine (1–2 mg/kg, IV, followed by 25–80 mg/kg/minute infusion) for refractory ventricular tachyarrhythmias. Fluid diuresis may help stabilize cardiovascular function and hasten urinary excretion of methylxanthines.

Once a patient is stable, or when presented before clinical signs have developed (e.g. within 1 hour of ingestion), decontamination should be performed. Induction of emesis via administration of apomorphine or hydrogen peroxide should be initiated; in animals that have been sedated because of seizure activity, gastric lavage may be considered. Activated charcoal (1–4 g/kg, PO) should be administered; because of the enterohepatic recirculation of methylxanthines, repeated doses should be administered every 12 hours in symptomatic patients for as long as signs are present (vomiting may be controlled with administration of metoclopramide, 0.2–0.4 mg/kg, SC or IM, every 6 hours as needed).

Other treatments for symptomatic patients include cardiac status monitoring via electrocardiography, correction of acid-base and electrolyte abnormalities, management of body temperature and urinary catheter placement (methylxanthines and their metabolites can be reabsorbed via the bladder). Clinical signs may persist for up to 72 hours in severe cases.

Conclusion

It is clear from the above inscriptions that since the metabolism of pets like dogs and cats are different from humans, any type of chocolate consumption (whether milk chocolate, dark chocolate, chocolate cake etc.) needs to be prohibited in pet animals.

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