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## Chocolate poisoning in golden retriever: A case report

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### Abstract

A 2-year-old dog with severe abdominal cramps, hematochezia, hypersalivation with panting was brought to the Enrich Pet Care Hospital with a history of eating Bournville dark chocolate. It was quickly treated with activated charcoal, and fluid therapy along with the required supportive medications based on a confirmed history from the owner and corresponding clinical indications. The symptoms of the poisoning subsided immediately after the treatment. The animal was discharged by advising the owner to follow Nothing Per Os (NPO) principle along with continuation of fluid therapy for two days for the animal to recover completely.

**Keywords:** Chocolate, poisoning, tachycardia

### Introduction

Most cases of toxicosis were noticed in dogs that ingest concentrated caffeine-containing compounds, such as dietary supplements, over-the-counter stimulants, coffee beans, caffeinated beverages, and chocolate-based products. Depending on the type of chocolate, different levels of theobromine and caffeine are present. Caffeine is included in most chocolate components; however theobromine is the poisonous substance that predominates. Based on the concentration of poisonous chemicals, white chocolate is claimed to be the least harmful, whereas dark chocolate is the most toxic (Zoumas *et al.*, 1980) [6]. Caffeine's half-life in adult canines has been estimated to be between 4.5 and 6.66 hours. Theobromine and caffeine had LD50 of 250-500 mg/kg body weight and 140 mg/kg body weight, respectively, in dogs (Kovalkoviová *et al.*, 2009) [3]. Caffeine and theobromine can penetrate the placenta and enter breast milk, posing a risk to unborn children or nursing infants.

Theobromine and caffeine are two of the toxic methylxanthines found in chocolate (Kovalkoviová *et al.*, 2009) [3]. Methylxanthine toxicosis is caused by to competitive inhibition of cellular adenosine receptors. Hence, caffeine naturally inhibits the activity of adenosine on the central nervous system and resulting in stimulating the neurological system and causing cardiac muscle contraction, smooth muscle relaxation (bronchodilation), and diuresis (Choi *et al.*, 1988) [8]. Methylxanthines also prevent cells from reabsorbing calcium, which raises the concentration of free calcium and improves the contractility of the heart and skeletal muscles (Papsa *et al.*, 1984) [5].

Clinical symptoms often appear six to twelve hours after intake which include Polydypsia, vomiting, diarrhoea, bloating, and restlessness. As the condition progresses, polyuria, ataxia, tremors, and seizures may also occur. Tachycardia, early ventricular contractions, tachypnea, cyanosis, hypertension, hyperthermia, and coma are some of the serious consequences (Kovalkoviová *et al.*, 2009) [3]. Bradycardia and hypotension were observed but with less frequency. Late in the toxicosis, hypokalemia is a possibility. Pancreatitis is one of the possible after effects noticed 24 to 72 hours after consumption because of the high fat content of many chocolate products. Arrhythmias of the heart or respiratory failure are the usual causes of death. Since there is no known cure for chocolate intoxication, conventional therapies include induction of emesis to expel the gastric contents followed by activated charcoal adjuvant therapy to prevent enterohepatic recirculation (Rosendale *et al.*, 2002) [2], antiarrhythmic medications, and intravenous fluid therapy are suggested.

### Case history & clinical findings

A 30-kg, 2-year-old adult female Golden Retriever had intake of Bournville dark chocolate, five hours before the presentation of the dog in Enrich Pet Care Hospital in Hyderabad. The dog was in lateral recumbency with obtunded mentation due to several bouts of hematochezia.

On clinical observation, pyrexia (105.6<sup>0</sup> F) and tachycardia (180 beats per minute) were noted.



**Fig 1:** Case history & clinical findings

### Treatment and Discussion

To remove the toxic material, gastric lavage was carried out, and moderately chilled fluids (5 mL/kg/h) were given over 12 hours in addition to antipyretics to correct mild dehydration and pyrexia. Lidocaine was administered (1-2 mg/kg i.v. till effect, followed by 25-80 mg/kg/min infusion rate to sustain the effect) to deal with tachyarrhythmias caused by ventricular origin which is commonly noticed in chocolate poisoning. No significant abnormalities were detected in the whole blood analysis, however a minor leucocytosis ( $23 \times 10^9/L$ ) was observed. Later, proton pump inhibitor (pantaprazole @ 1 mg/kg b.wt), etamsylate @ 10 mg/kg b.wt, and other supportive such as sucralfate and antibiotic amoxicillin clavulanate @ 11 mg/kg b.wt were administered along with supportive fluid treatment.

Gastric emptying and patient stabilization followed by supportive therapy were the important aspects in the treatment of chocolate poisoning. If the duration of the induction was shorter than 4 hours and the dog was conscious without experiencing any seizure episodes, emesis induction can be performed on the animal (Liuz *et al.*, 2008) [1]. Diazepam or a barbiturate can be used as needed to control seizures. Using electrocardiography, monitor dogs heart condition and treat arrhythmias as needed. Metoprolol succinate or metoprolol tartrate (0.2 to 0.4 mg/kg orally b.i.d.) is the preferred-blocker, if available, because propranolol hydrochloride has been reported to induce a delay in the renal excretion of methylxanthines (Brant *et al.*, 2001) [7].

Once the symptoms of the central nervous system are under control, hyperthermia caused by excessive muscular activity usually subsides naturally and does not require aggressive treatments, such as cold-water enemas or baths. Since erythromycin and corticosteroids impede the excretion of methylxanthines, they should be avoided (Kovalkovičová *et al.*, 2009) [3]. Giving repeated doses of activated charcoal (1-4g/kg PO) is typically beneficial in symptomatic animals as large quantities of chocolate may coalesce in the stomach in some cases, forming a big lump that cannot be readily vomited or removed by gastric lavage. Additionally, if the dog had swallowed wrapped candy, the adverse effects of methylxanthines can be delayed for a few hours or days.

The cardiovascular system will be supported and urine excretion will be improved by administering intravenous fluids at double the maintenance levels. As caffeine can be reabsorbed from the urinary bladder, it is emptied continuously by inserting a urinary catheter in animals that

are moderately to severely affected (Liuz *et al.*, 2008) [1]. Regularly, monitor the serum electrolytes and adjust any imbalances, if necessary. Half-life of Theo bromine in dogs is 17.5 hours in comparison to 4.5 hours for caffeine, which leads to persistence of clinical symptoms for up to 72 hours in extreme instances.

### Conclusion

Chocolate intoxication is a serious condition that requires immediate medical attention. A theobromine and caffeine level, and not the amount of chocolate consumption, determines how serious it can be. In most situations, timely treatment results in a safe recovery.

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