Suspected case of Angel's near 'death by chocolate' experience

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MARTIN ATKINSON discusses the toxicity of chocolate to dogs and the history, diagnosis and treatment of a dog presenting with vomiting and diarrhoea

Acute chocolate poisoning in dogs is relatively common – following a history of consuming large amounts of milk or dark chocolate.

The toxic ingredients are methylxanthines, mainly theobromine and caffeine, which cause an increased release of catecholamines and increased muscle contractility by facilitating cellular entry of calcium and inhibition of sequestration by the sarcoplasmic reticulum. Benzodiazepine receptors in the brain are competitively antagonised.

Toxicity

The amount of methylxanthines in chocolate varies depending on the degree of refinement and its formulation. Theobromine is contained in higher quantities than caffeine (three to 10 times) and, in practice, is more toxic, because it is metabolised more slowly in dogs.

Raw cocoa beans contain 1.2 per cent theobromine by weight. Far smaller quantities are found in milk chocolate (1.2-1.8g/kg) than in dark chocolate, cocoa powder and bakers' chocolate, which contain 12-20g/kg. White chocolate contains insignificant amounts. LD50 for theobromine in dogs is around 200mg/kg bodyweight but death may occur at much lower rates. Cardiotoxicity may occur at 40-50mg/kg, while signs of mild toxicity can be seen after the ingestion of as little as 16mg/kg.

Therefore, in practical terms, one ounce of milk chocolate per pound of bodyweight (60g per kilo) may be fatal – or a tenth of that amount in cooking chocolate. In contrast, it would be virtually impossible for a human to eat enough chocolate to induce methylzanthine poisoning.

Classical signs of theobromine poisoning in dogs are:

• vomiting;

• diarrhoea;

• polyuria;

• cardiac arrhythmias;

• excitability;

• seizures;

• coma; and death.

Other less frequently documented symptoms are:

• cyanosis;

• internal haemorrhage;

• right atrial enlargement; and

• cardiomyopathy.

Case study

Angel, a nine-year-old, regularly vaccinated, neutered female West Highland white terrier, weighing 7.5kg, was presented as a night-time, out-of-hours emergency at a sole-charge, first-opinion clinic. She had been vomiting intermittently for a few hours and had diarrhoea.

On examination she was found to be moderately hypothermic (T99F). Her respiration was shallow and rapid, but there were no abnormal respiratory sounds and she had a fairly rapid weak pulse, which was not considered tachycardic or arrhythmic. However, the most striking presenting symptom was severely cyanotic oral mucosal membranes. Capillary refill time (CRT) was not measurable due to the degree of cyanosis. Given the apparent state of her cardio- vascular compromise, she was bright and alert. Poisoning was initially suspected but was ruled out after

discussion with the owners.

Other differentials considered for immediate emergency treatment were:

• addisonian crisis;

• internal organ haemorrhage with pulmonary or cardiac infarct; and

• acute heart failure.

The patient was admitted for observation and investigation. Shortly after admission she suffered a seizure and was given oxygen by mask. The seizure ceased after a few seconds and she was then placed in an oxygen therapy unit, but her mucous membranes remained cyanotic. Her cardiovascular compromise was considered too severe to risk restraint or sedation for radiography. Normal saline was administered intravenously. Acute cardiac failure was considered to be the primary differential and approximately 1cm of glyceryl trinitrate, two per cent cream, was applied on the underside of the pinna.

Her condition improved within two hours and, by the morning, her pulse quality was greatly improved. The mucous membrane colour was normal with a CRT of one second.

Haematology revealed a raised haematocrit, a marked leucocytosis with neutrophilia and a borderline eosinophilia, which may have reflected dehydration. Biochemistry showed a slightly raised total bilirubin and mild hyperglycaemia. Electrolytes were within normal range. Radiography of the chest and ECG examination showed a rightsided cardiac enlargement, but there was no evidence of pulmonary congestion or pathology.

Abdominal radiography was unremarkable and peritoneal lavage/ paracentesis for abdominal haemorrhage was unproductive. She was given one pimobendan 1.25mg capsule, and 0.5ml amoxycillin/clauvulonic acid subcutaneously. Glyceryl trinitrate cream was re-applied when she started to become cyanotic again and her condition improved within an hour following its application. Fluid therapy was continued with Hartmann’s solution at maintenance levels for a further 12 hours. After which, she was clinically normal and, at the client’s request, discharged with Synulox 50mg tablets two bid, Vetmedin 1.25mg capsules bid and Percutol gel to be applied in case of a relapse.

Her condition remained apparently normal until she was re-presented four days later. She was vomiting and collapsed with pale and icteric mucous membranes. Biochemistry showed a slight increase in total bilirubin than the previous occasion, a mild hypocalcaemia and a moderate, but strongly regenerative, anaemia with a normal platelet and leukocyte count. There was no evidence of internal haemorrhage, melaena or haematemesis, and intravascular haemolysis was indicated from the profile. She was given intravenous metoclopramide and dexamethasone at an

immunosuppressive dose, as auto-immune haemolytic anaemia was now suspected, and maintained on Hartmann's solution. A sample for Coombe's test was taken and subsequently reported negative. Unfortunately, her owners were unable to afford continued treatment or further investigation and Angel was discharged at their request despite a guarded prognosis.

On further questioning of the clients as to any other possible causes of Angel's condition, they revealed their daughter had admitted to habitually giving chocolate to Angel – both in the form of candies and cooking chocolate. They were, however, adamant that the amount of chocolate being fed was just a few grams – far less than that considered necessary to cause acute theobromine poisoning. Questioning also revealed a history of intermittent vomiting, diarrhoea and lethargy over several weeks or months.

Angel's condition continued to improve and within a few days she was clinically normal, despite no further treatment, and remained so for an additional 24 months with no medication. During this time the client's daughter had also moved out of the home and no more chocolate had been given to Angel. Furthermore, Angel's demeanour had reportedly improved considerably over the corresponding period of time prior to her initial presentation.

Discussion

Considering the apparent frequency of chocolate poisoning, reliable research on this subject appears to be sparse. Documented cases of chocolate poisoning in dogs mostly relate to acute ingestion of large quantities, with the onset of symptoms within six to 12 hours. In this case it is speculated that chronic ingestion of small quantities had accumulated to become toxic.

Although the owners insisted the amount of chocolate being fed was small, there is the potential of mild toxic effects in a 7.5kg dog after as little as two grams of cooking chocolate and serious symptoms with just 20 grams. Theobromine is partly metabolised in the liver and metabolites undergo hepatobiliary recycling. Metabolites and the basic compound are excreted via the kidneys. Whereas the half life of theobromine in humans is two to three hours, in dogs it is 17 to 18 hours, which could lead to an accumulative effect. It is further speculated that subclinical poisoning over a longer period of time resulted in cardiomyopathy, which culminated in acute heart failure.

So far, as the author is aware, intravascular haemolysis is not reported as a consequence of theobromine poisoning, but may have occurred in this case. Unfortunately, however, it was not possible to rule out the possibility of pre-existing cardiac disease, and although there was evidence of rightsided cardiac enlargement, the continued health of the patient without specific treatment appears to rule out primary heart disease as a cause of the presenting condition. Symptoms of acute cardiac failure may have been attributable to the profound hypoxia – likely from the initial cyanotic state.

Of further interest is the role of glyceryl trinitrate and pimobendan, which were initially used to treat

the suspected right-sided heart failure in this case when there was no knowledge of chocolate ingestion. Glyceryl trinitrate is a systemic vasodilator and may appear contraindicated in a case of apparent reduced cardiac output. However, it is also a potent coronary vasodilator, and it is this property that was utilised in this case. One of the modes of action of pimobendan is to increase the contractility of cardiac muscle by sensitising it to calcium. As theobromine increases the entry of calcium into the muscle and reduces sequestration, there could be a synergistic effect.

In retrospect, the use of this medication was probably contraindicated, despite the potential benefit to be derived from the positive inotrophic effect of pimobendan on cardiac muscle. In this case, there appears to have been no detrimental effect. However, pimobendan was withdrawn after initial treatment. Standard treatment protocols for chocolate ingestion include induction of emesis and gastric lavage with adsorbents. However, as this appears to have been a chronic case, this treatment would not have been useful, even with knowledge of the history of chocolate ingestion at first admission.

Conclusion

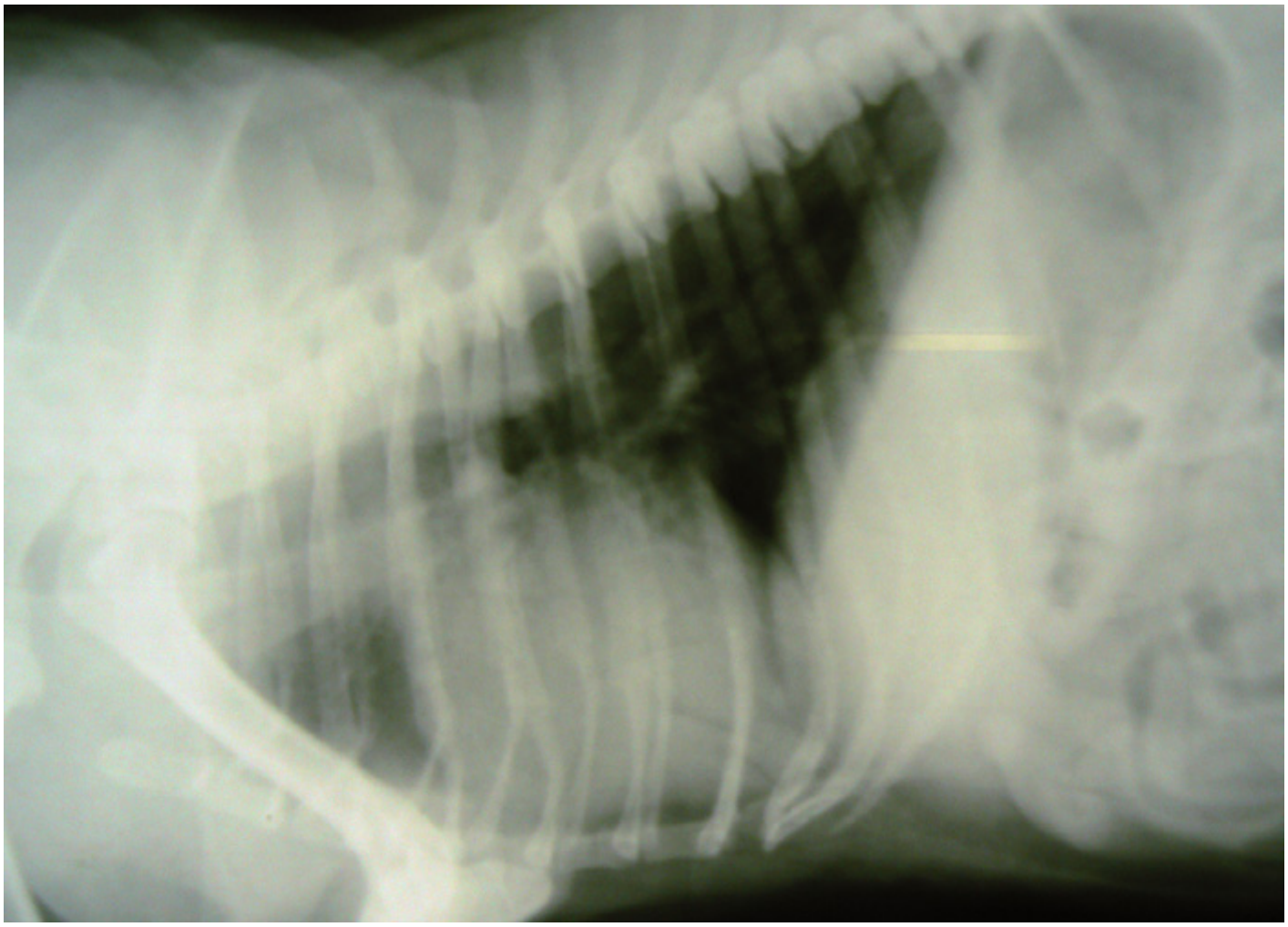
Although many of the symptoms of this case were typical of theobromine poisoning, the lack of initial evidence of chocolate ingestion was misleading. Theobromine poisoning remains the most likely cause, given her recovery with no further treatment following the cessation of chocolate ingestion.

Other factors relating to the daughter’s relationship with the patient, such as a restrictive diet and lack of exercise, cannot be ruled out as contributing factors, which, if removed, may have led to an improvement in Angel’s quality of life.

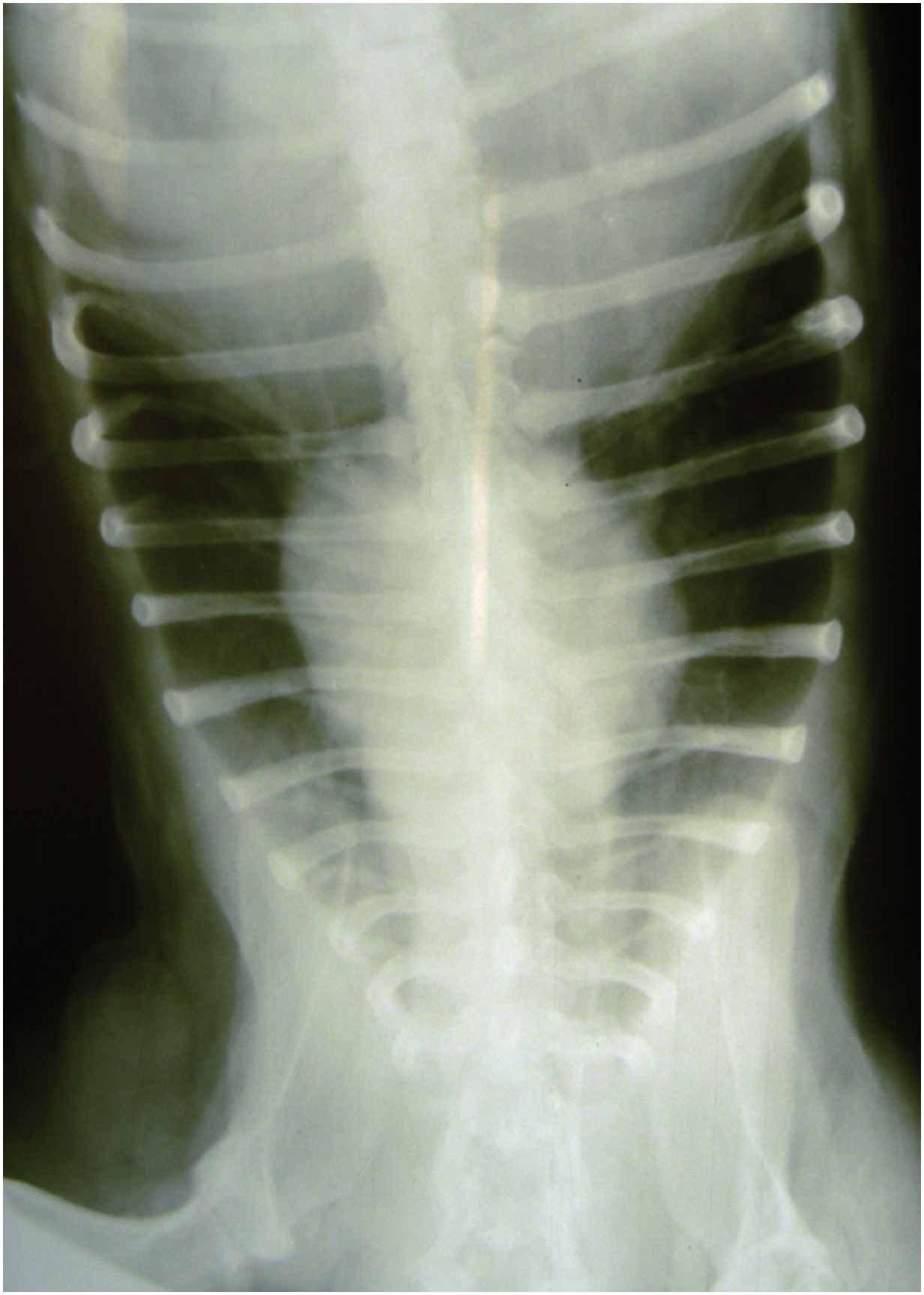
However, chronic theobromine poisoning should be considered as a differential diagnosis, even in cases where there is no history of acute ingestion of large quantities of chocolate, and may need to be considered as a differential cause of acute intravascular haemolysis.

• References available on request.

Dark chocolate contains significantly more toxic theobromine than white chocolate.



Lateral view radiographs of the patient revealed right-sided cardiac enlargement.



Dorsal view radiographs of the patient revealed right-sided cardiac enlargement.

Actual values	Reference range
HCT 58.6 per cent	37-55 per cent
WBC 39.1 x 10^9/L	6-16.9 x 10^9/L
Neutrophils 35 x 10^9/L	2.8-10.5 x 10^9/L
Eosinophils 1.6 x 10^9/L	0.5-1.5 x 10^9/L
Total bilirubin 14µmol/L	2-10µmol/L
Glucose 8.1mmol/L	3.3-6.1mmol/L
Other parameters normal	

Table 1. Relevant haematological and biochemical parameters of Angel on first visit

Actual values	Reference range
RBC 3.49 x 10^{12}/L	5.5-8.5 x 10^{12}/L
Other RBC parameters in proportion to this anaemia	
Total bilirubin 17μ/L	2-10μmol/L
ALT 438μ/L	10-118μ/L
Ca 2.11mmol/L	2.15-2.95mmol/L
Other parameters normal	

Table 2. Relevant haematological and biochemical parameters of Angel on second visit