Tis the season: festive toxicological hazards for cats and dogs

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ABSTRACT: Christmas can be a very busy time in the veterinary clinic and there are many hazards within the home that may put pets at risk. Some popular Christmas treats are well publicised as being toxic to dogs and cats, such as chocolate, but there are some lesser known toxins that owners may not be aware of e.g. onions or mouldy leftovers. This article examines some of the edible toxicological hazards that may be encountered, explaining the mechanism of action, clinical signs, the toxic dose, treatment options available and patient prognosis.

Keywords: Christmas; toxins; chocolate; raisins; xylitol; ethanol; *Allium* species; mycotoxins

Introduction

The festive season is an exciting, busy time full of indulgence, celebration and fun for all the family including pets. But this period can present a range of dangers to dogs and cats including toxic foods, dangerous plants and gastrointestinal obstruction hazards. As registered veterinary nurses (RVN) we can play a huge role in not only the treatment of these patients but also in the education of owners with the hope of preventing the need for treatment. This article will focus on the edible toxins that are a risk during the festive season, discussing the mechanism of action, toxic dose, clinical symptoms, treatment and prognosis.

Chocolate

Chocolate is a product from the beans of *Theobroma cacao* which are processed into cocoa solids and butter (Bates, 2015) and chocolate will be found in many households over the festive period in some form. Chocolate contains two methylxanthines of toxicological significance – theobromine and caffeine. The concentration of theobromine is 3–10 times that of caffeine but both contribute to the clinical symptoms seen (Gwaltney-Brant, 2013).

Methylxanthines work by inhibiting cellular adenosine receptors, stimulating catecholamine release (adrenaline and noradrenaline) and increasing calcium entry into muscular cells (Babyak & Lee, 2018). Clinical signs of chocolate toxicity include gastrointestinal (GI) effects including vomiting and diarrhoea, excitability/hyperactivity, tachycardia, tremors/seizures, hypertension, hyperthermia and less commonly tachypnoea and cardiac arrhythmias. These signs usually occur 2–4 hours post ingestion but may occur after 6–12 hours (Bates, 2015).

Toxic effects of theobromine may occur at doses of 20 mg/kg in dogs with severe signs reported at 40–50 mg/kg and seizure activity reported at 60 mg/kg (Bates et al., 2015). To establish if a toxic dose has been ingested, it is essential to understand the quantity of cocoa solids present (Table 1) and online 'chocolate toxicity calculators' are available as a quick reference source for practices. Although theobromine toxicity is not considered a risk with white chocolate, it is important to realise that the high fat content may pose a chance of pancreatitis developing and clinical signs of this may develop up to 72 hours post-ingestion (VPIS, 2019).

Initial treatment involves patient decontamination including inducing emesis if the patient is neurologically stable. In dogs, apomorphine (available in a licensed version Emedog©) is administered at 0.1 mg/

kg subcutaneously as a single injection (NOAH, 2020). It has been recommended that this can be performed up to 6 hours post ingestion as chocolate remains in the stomach for a prolonged period (Babyak & Lee, 2018). Activated charcoal (Figure 1) is indicated in cases of chocolate ingestion. Methylxanthines undergo enterohepatic circulation meaning they are metabolised in the liver, excreted into bile and released back into the GI tract where they are reabsorbed and will have further toxic effect. It is recommended to repeatedly dose activated charcoal as a result of enterohepatic circulation and current recommendations for repeated dosing are to administer 1–2 g/kg without a cathartic orally every 6 hours for 24 hours post-ingestion (Babyak & Lee, 2018). If the patient is showing signs of seizure activity, the veterinary surgeon (VS) may decide to prescribe muscle relaxants and anticonvulsants and if tachycardic, may be prescribed beta-blockers. Methylxanthines can be reabsorbed across bladder epithelium so frequent walks or placement of a urinary catheter to allow bladder emptying should be facilitated (Babyak & Lee, 2018).

Most dogs recover fully but prognosis is more guarded if seizure activity or cardiac arrhythmia are present (Bates, 2015).

Table 1. Approximate theobromine content of chocolate products (Bates et al., 2015).

Type of product	Definition	Theobromine content per gram	Approximate dose of product equivalent to 20 mg of theobromine
Milk chocolate	Minimum 20% cocoa solids in UK/Ireland (min 25% in EU)	1.0–2.1 mg	9.5–20 g
Dark (plain) chocolate	Minimum 35% cocoa solids (can be much higher)	4.4–8.8 mg	2.3–4.5 g
Cocoa Powder	Non-fat part of the cacao bean ground into powder	4.6–38 mg	0.2–1.9 g
White chocolate	Contains no cocoa solids	Insignificant	Minimal toxicity risk

Figure 1. Activated charcoal is used for adsorption in some toxicology cases.

Raisins/sultanas

Raisins and sultanas are nephrotoxic to dogs but not cats. At Christmas they will be found in a variety of human treats and foods including Christmas cake, Christmas pudding and mincemeat (Figure 2). It is important to remember that if the raisins or sultanas are chocolate covered or soaked in alcohol (for example in a Christmas cake) this poses a double intoxication risk and signs associated with these toxins will need to be taken into account during treatment.

The mechanism of action is currently unknown. Histopathologic examination of necropsy samples has revealed proximal renal tubular necrosis (Norkus, 2019) and some theories include metabolic disruption, a nephrotoxic mycotoxin and an idiosyncratic reaction (McKnight, 2019) but no studies have yet identified a definitive mechanism. The toxic dosage of raisins and sultanas is also unknown. In some cases, a dog can ingest large quantities with no ill effect, whereas in other cases a single raisin has been reported to cause acute renal failure (ARF) (Babyak & Lee, 2018). Clinical signs of intoxication result from ARF and are summarised in Figure 3.

Treatment should be implemented in all cases of raisin and sultana ingestion regardless of quantity and if it is a known recent ingestion, emesis should be induced and activated charcoal administered (Figure 1). Treatment for ARF includes aggressive intravenous fluid therapy (IVFT), blood pressure monitoring for hypertension, frequent assessment of urine output (UOP) to determine if oliguria/anuria develops and regular assessment of renal values (Babyak & Lee, 2018). Dialysis may be considered but the effectiveness of this is unclear (Norkus, 2019).

Prognosis is dependent on the condition of the patient on presentation, success of decontamination and progression of clinical symptoms (McKnight, 2019). If ARF is present, a survival rate of 53% has been reported and negative prognostic indicators, if the patient is diagnosed with ARF, include hyperphosphataemia, hypercalcaemia, decreased UOP and ataxia (Eubig et al., 2005). No factors have yet been identified

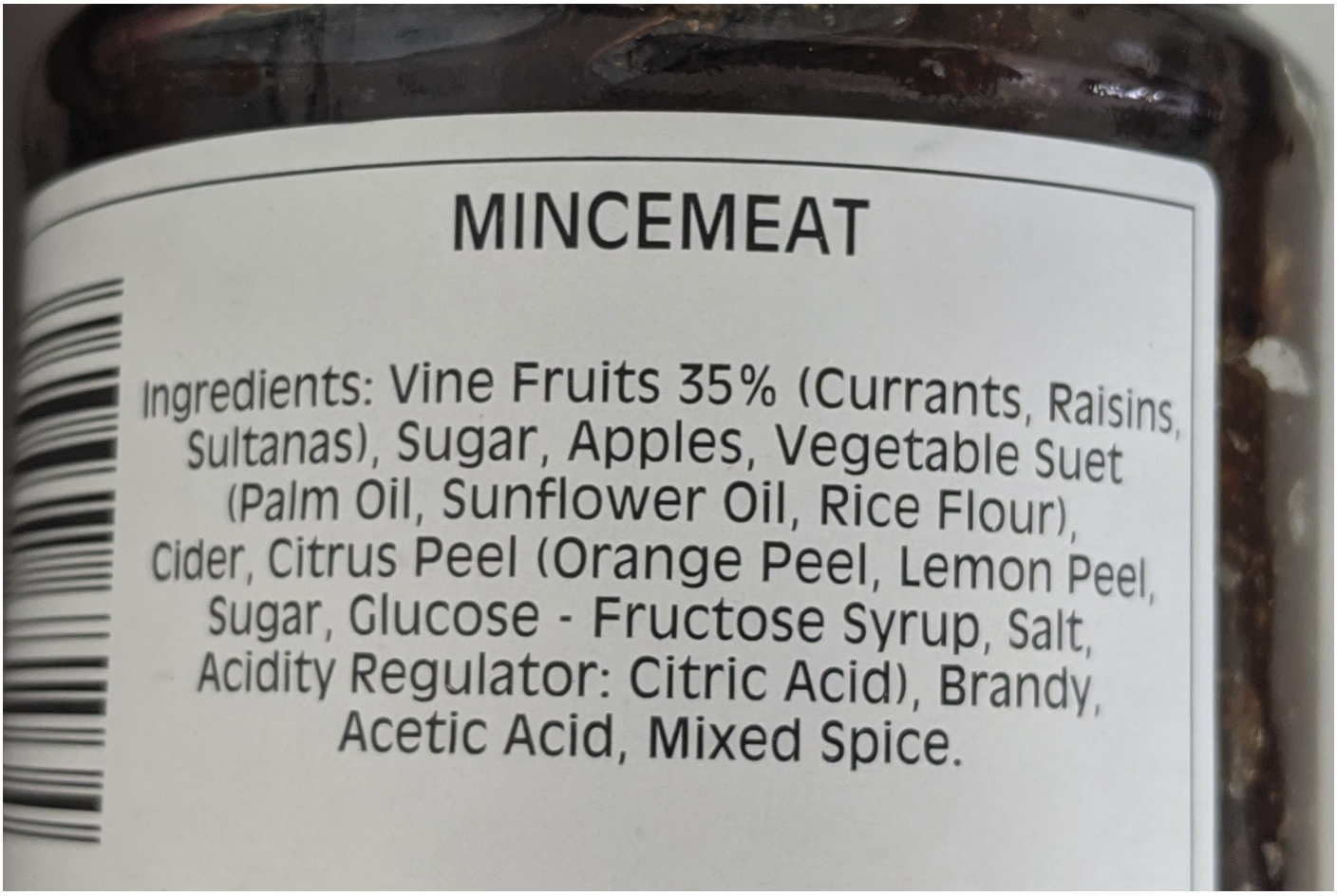

Figure 2. Mincemeat is a common Christmas treat that contains toxic ingredients and may be used as filling in other treats.

- Vomiting
- Lethargy
- Anorexia
- Abdominal pain
- Diarrhoea
- Dehydration
- Polyuria and polydipsia (PUPD)
- Uraemic breath

Figure 3. Signs and symptoms of ARF that may develop in cases of grape and raisin toxicity (Babyak and Lee, 2018).

to predict which patients will develop ARF (Reich et al., 2020).

Xylitol

Xylitol is a sugar substitute and may be found in sugar-free or calorie-free treats over the Christmas period, or in the New Year as everyone starts to make their resolutions to eat better. It is toxic to dogs but cats do not appear to be sensitive to it (Norkus, 2019). Xylitol can be found in peanut butter, sugar-free sweets and mints, jams and low-sugar puddings, if anything contains artificial sweeteners it may contain xylitol.

Ingestion of xylitol stimulates a large secretion of insulin leading to an insulin spike and severe hypoglycaemia which occurs rapidly within 15–30 min of ingestion (Norkus, 2019). Higher doses of xylitol ingestion can cause an acute hepatic necrosis, the cause of which is not known (Schmid & Hovda, 2016). Doses as low as 0.05–0.1 g/kg can cause hypoglycaemia while doses >0.5 g/kg) can lead to acute hepatic necrosis (Babyak & Lee, 2018).

Clinical signs of xylitol toxicity typically relate to hypoglycaemia and include lethargy/weakness, vomiting, ataxia, seizure activity and patients ingesting hepatotoxic doses may exhibit signs of hepatic failure (Babyak & Lee, 2018). The main aim of treatment is to normalise blood glucose levels starting with dextrose boluses and continuing with dextrose continuous rate infusion (Table 2). Emesis is only indicated if there is a potential for GI obstruction from the product eaten, it is unlikely to prevent hypoglycaemia due to the rapid absorption rate of xylitol (Babyak & Lee, 2018). Activated charcoal does not bind to xylitol so is not recommended in these cases. Treatment for hepatotoxicity includes hepatoprotectants such as SAMe and supportive care including IVFT. Hepatic enzymes should be rechecked 72 hours post exposure for evidence of early hepatic failure (Norkus, 2019).

Prognosis in these cases is generally positive if rapid intervention is implemented and hepatic failure does not develop but prognosis in cases with evidence of hepatic failure is poor (Norkus, 2019).

Alcohol

The active ingredient in alcoholic beverages that is toxic to dogs and cats is ethanol (Lundgren, 2020). Although a less common toxicity to present at clinic, it is still possible if alcoholic drinks are left unsupervised or if the pet has eaten some rotting/fermenting fruit (Lundgren, 2020). The pet may also eat alcohol filled chocolates or alcohol-soaked sultanas or raisins in products such as Christmas cake/pudding (Wismer, 2017) and this will

Table 2. Quick reference guide on making a dextrose solution for infusion.

% dextrose solution to make	Fluid bag size	Amount fluid to remove	Amount dextrose to add	Total amount fluid
2.5%	250 ml	12.5 ml	12.5 ml of 50% dextrose	250 ml
2.5%	500 ml	25 ml	25 ml of 50% dextrose	500 ml
2.5%	1,000 ml	50 ml	50 ml of 50% dextrose	1,000 ml
5%	250 ml	25 ml	25 ml of 50% dextrose	250 ml
5%	500 ml	50 ml	50 ml of 50% dextrose	500 ml
5%	1,000 ml	100 ml	100 ml of 50% dextrose	1,000 ml

N.B. if solution >5% is required this must be administered via a central vein due to the risk of thrombophlebitis.

Table 3. Ethanol concentrations in common alcoholic beverages (Wismer, 2017).

Substance	% proof	% ethanol by volume
Light beer	5–7	2.5–3.5
Beer	8–12	4–6
Ale	10–16	5–8
Wine	20–40	10–20
Amaretto	34–56	17–28
Schnapps	40–100	20–50
Coffee liquers	42–53	21–26.5
Brandy	70–80	35–40
Bourbon	80–90	40–45
Rum	80–82	40–41
Cognac	80–82	40–41
Vodka	80–82	40–41
Whiskey	80–90	40–45
Tequila	80–92	40–46
Gin	80–94	40–47

result in double toxin exposure. It is important to note that although some may think of a drunk pet as amusing, it can lead to serious clinical signs and even death, therefore prompt treatment should be sought.

Ethanol is a central nervous system (CNS) depressant and also stimulates release of other inhibitory neurotransmitters, such as dopamine and serotonin (Wismer, 2017). The oral lethal dose in dogs is 5.5–7.9 g/kg of 100% ethanol and 1 ml ethanol is equal to 0.789 g (Wismer, 2017). The amount of ethanol required to cause intoxication will vary depending on the concentration of the substance ingested (Table 3). Clinical signs occur within 15–30 minutes of ingestion (Lundgren, 2020) and the patient will likely exhibit signs similar to human inebriation such as nausea/vomiting, polyuria and polydipsia, incoordination and disorientation and hypothermia. High levels of intoxication may lead to cardiorespiratory depression, hypotension and hypoglycaemia (Lundgren, 2020).

Treatment of these patients partly depends on their condition on presentation. Emesis is only indicated in asymptomatic patients due to the risk of aspiration and although IVFT has not been shown to increase ethanol clearance but is useful to support the patient (Wismer, 2017). Activated charcoal binds poorly to alcohol so is not indicated in these cases. An important role of the RVN is to monitor cardiorespiratory parameters and blood glucose levels, informing the VS of any changes immediately, they also play an essential role in the protection of the patient as, being inebriated, these patients are predisposed to traumatic brain injury (Wismer, 2017). If the patient is highly intoxicated with severe CNS depression, they may require intervention with intubation and ventilatory support.

In most cases, prognosis is good but in cases where aspiration of GI contents has occurred, or in patients who have a pre-existing disease, they may have a more guarded prognosis (Wismer, 2017).

Allium species

The genus *Allium* includes onion, garlic, leek, chives and shallot (Figure 4) and all are toxic to both dogs and cats (Salgado et al., 2011). Although these may not be intentionally fed to pets, accidental ingestion can occur if they have been used as ingredients in dishes such as stuffing and casseroles or during the preparation of these. The domesticated

Figure 4. Onions and garlic are some of the common *Allium* species that cause a problem in our canine patients.

Table 4. *Allium* species commonly involved in toxicosis (Cope, 2005).

Common name	Scientific name
Onion	*Allium cepa*
Garlic	*Allium sativum*
Leek	*Allium porrum*
Spring onion	*Allium fistulosum*
Chive	*Allium schoenoprasum*

Allium species most commonly involved in toxicosis are summarised in Table 4.

Allium species contain various organosulfoxides which are converted to organosulfur compounds as a result of trauma (chewing or cutting) and many of these compounds appear to be readily absorbed from the GI tract before they are converted to reactive oxidants (Salgado et al., 2011). These oxidants cause oxidative damage to erythrocytes causing a haemolytic anaemia and Heinz body formation.

The toxic dose in cats is as little as 5 g/kg onions while in dogs it is 15–30 g/kg onions (Cope, 2005). It is important to note that cooking does not reduce the potential toxicity. Clinical signs may appear within 24 hours if a large amount of onions have been consumed but it can take up to 7 days for signs to occur if smaller amounts have been ingested. GI signs are common including vomiting, diarrhoea, abdominal discomfort and hyporexia. Signs consistent with anaemia include lethargy/weakness, pale mucous membranes, haemoglobinuria, jaundice, tachycardia and tachypnoea (Salgado et al., 2011).

There is no specific antidote for *Allium* species ingestion. Induction of emesis in asymptomatic patients can be performed within 1–2 hours of ingestion and activated charcoal (Figure 1) is suitable for use in these cases (Salgado et al., 2011). These patients may require a lot of supportive care including anti-emetics if vomiting as a result of the toxicity, IVFT, analgesia and oxygen therapy and it is this area where RVNs can provide a lot of support and care

for the patients. If indicated and depending on the severity of anaemia, the patient may receive a blood transfusion of packed red blood cells (Salgado et al., 2011).

Prognosis is dependent upon the severity of anaemia, the species of *Allium* involved and the effective implementation of supportive care (Cope, 2005).

Mouldy leftovers

Mycotoxins are produced by fungi that are present on mouldy foods and are neurotoxic to dogs (Bough, 2019). These mycotoxins may be referred to as tremorgenic mycotoxins due to the fact tremors are one of the key clinical signs. Around 20 mycotoxins have been determined as tremorgens and those most frequently reported to affect dogs are penitrem A and roquefortine C produced by *Penicillium* spp (Bough, 2019). Table 5 lists the foods penitrem A grows on and that may cause an issue, roquefortine C is a metabolite of the mould on blue cheese (Finoli et al., 2001). Dogs may suffer this type of intoxication due to their sometimes-indiscriminate eating habits involving the raiding of bins and feasting on mouldy leftovers from festive dinners and parties.

It is likely that the mechanism of action differs for each individual mycotoxin and most are not yet fully understood (Bough, 2019). With regard to penitrem A, the exact mechanisms of action are not known but it is known that primary site of action is the CNS. It is most likely that penitrem A has a dual role affecting both inhibitory and excitatory neurotransmission and it is rapidly absorbed with signs appearing between 15 minutes to several hours post ingestion (Waratuke, 2017).

The toxic dose in one study was reported as 0.5 mg/kg purified penitrem A administered intraperitoneally resulted in onset of acute tremors in a dog (Hooser & Talcott, 2016). As mentioned, clinical signs can vary in time of onset but typically include early signs of restlessness, panting and hypersalivation which often progress to mild/moderate body tremors. In high dose toxicity, tremors may be severe and progress to seizure activity and, if untreated, tremors/seizures can lead to hyperthermia, exhaustion and dehydration along with potential metabolic acidosis and rhabdomyolysis (though rare) (Hooser & Talcott, 2016).

Table 5. Penitrem A may grow on these foods and pose a toxicological risk to our patients.

Penitrem A commonly grows on:
Meat
Cereals
Nuts
Cheese
Eggs
Fruits
Processed/refrigerated food
Rubbish
Compost

Treatment for mycotoxicity begins with induction of emesis, gastric lavage and administration of activated charcoal (Figure 1) if the patient is neurologically appropriate (Waratuke, 2017). Diazepam can be used to control tremors and seizure activity while barbiturates can also be administered if there is no response to diazepam (Hooser & Talcott, 2016). The RVN can assist in the provision of supportive care including temperature monitoring with active cooling measures implemented as necessary and IVFT. Intravenous lipid emulsion (ILE) therapy may be useful but information regarding its use is limited, penitrem A is believed to be lipid-soluble which makes it a potential candidate for ILE therapy (Waratuke, 2017).

Prognosis is generally good. In most patients, signs resolve within 24–48 hours but with larger exposures signs may last 4–5 days (Hooser & Talcott, 2016).

Conclusion

Following this brief overview of a selection of festive toxicological hazards, the RVN should feel more confident in providing advice and guidance to owners over the Christmas and New Year period. Good owner education is vital in reducing incidents of accidental exposure and ensuring they seek veterinary attention quickly to achieve a good outcome for their pet. RVNs play an active role in owner education from regular in-practice promotions featuring festive themes to providing accurate advice during the initial communication on the telephone. Toxins work in different ways and patients may present with a variety of different signs so questioning the owners on what the pet may have had access to is crucial in the management of these cases.

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