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CASE REPORT

Ethanol toxicity in a dog due to gin-soaked sloe berry (*Prunus spinosa*) ingestion

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Abstract

A 10-month-old male Labrador retriever was presented for acute onset altered mentation and inability to walk. The dog was presented with a modified glasgow coma scale (MGCS) of 12 with tachycardia (148 bpm) and hypothermia (37°C). The dog had been normal the previous night then found semi-comatose at the following morning. Investigations included haematology, biochemistry, C-reactive protein, magnetic resonance imaging, urine toxicology and cerebrospinal fluid analysis. Ten hours after being found the dog passed faeces containing a significant volume of berries. The owner later determined that 750 g of sloe berries soaked in gin were missing from the garden compost heap. Serum ethanol concentrations measured approximately 20 h after the suspected consumption were 310 mgD/L. The dog was treated with intralipid, intravenous fluid therapy and paracetamol. He was neurologically normal (MGCS = 0) 24 h later, following diagnosis and treatment. Differential diagnosis and management of the comatose dog and management of ethanol toxicity are discussed.

KEYWORDS
emergency medicine, neurology, neurotoxicologym, toxicology

BACKGROUND

Ethanol intoxication has been sporadically reported in the veterinary literature. Causes include ingestion of sourdough containing yeast product, rotten apples, dermal absorption from shampooing puppies with detergent and ingestion of human over-the-counter medications.[1–4] There is one other case report in the veterinary literature of assumed ethanol poisoning via ingestion of sloe berries that had been used to make sloe gin,[5] however this was not confirmed with blood ethanol concentrations.

Identifying the inciting toxin in a suspected toxicity case in veterinary medicine can be challenging. Owners are often either unaware or embarrassed to admit the knowledge of the ingested substance. Ethanol toxicity can be overlooked as a differential especially as testing requires serum concentrations to be quantified, which is not routinely included on a toxicology panel. This case presented with slightly asymmetrical neurological signs, which may reduce the clinical suspicion for encephalopathy due to metabolic disease or toxin ingestion. Ethanol toxicity was only suspected after dog passed faeces containing berries. The realization of this ingestion earlier would have avoided numerous expensive diagnostic tests for the patient. This is an interesting case example of ethanol toxicity emphasizing the importance of gathering a thorough history and the value of supportive care in these patients.

CASE PRESENTATION

A 10-month-old male entire Labrador retriever (Figure 1) was presented to a primary care practice then referred to a specialist neurology service following an acute onset of non-ambulatory tetraparesis with concurrent signs of central nervous system depression. The dog was vaccinated with no history of travel and received appropriate parasitic prevention (including protection against *A. vasorum)*. There was no other relevant medical history. The evening before presentation the dog was allowed outside to urinate at 9 pm and then crated overnight. The next morning the dog was found unable to stand, and his demeanour was noticeably subdued. He was immediately presented to his local veterinary surgeon who found asymmetrical tetraparesis with more marked postural deficits on the left. He also had a sluggish pupillary light reflex bilaterally and an inconsistently absent menace response bilaterally. Blood work was relatively unremarkable; a slightly low urea at 1.7 mmol/L (2.5–9.6 mmol/L), and a very slight monocytopenia of 0.25×10^9/L ($0.30\text{-}2.00 \times 10^9$). Following questioning of the owners, it was determined that there was no history of known toxin ingestion. The dog was referred and arrived 7 h after being found by the owner.

On presentation, the dog was semicomatose with occasional periods of reduced responsiveness to auditory stimuli. He was tachycardic with a heart rate of 148 bpm, hypothermic with a rectal temperature of 37.0°C. He was slightly

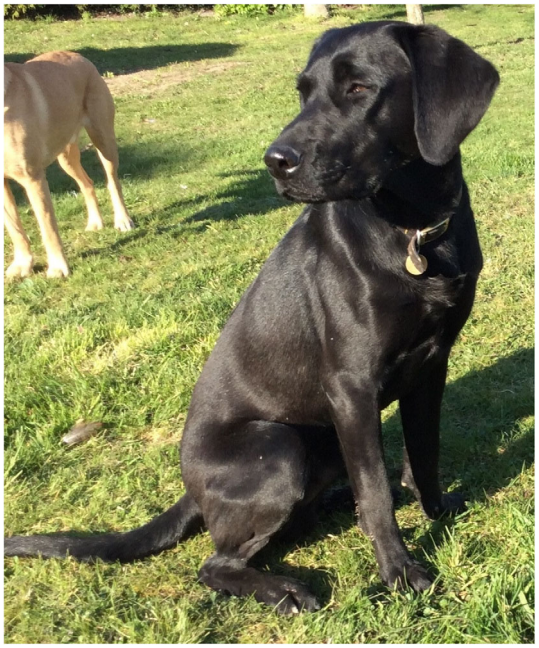

FIGURE 1 The dog who consumed the gin-soaked sloe berries

hypertensive on oscillometric blood pressure measurement, with a mean arterial pressure of 110 mmHg. He was non-ambulatory tetraparetic, with a right sided body turn and intermittent high extensor tone in all limbs. There was absent proprioception in both thoracic limbs and the left pelvic limb, with intact spinal reflexes. There was a bilateral inconsistently sluggish menace response, slightly more delayed on the right side and a right sided ventrolateral strabismus. The pupillary light reflex was also sluggish bilaterally, the pupils were symmetrical and miotic. There was a sluggish vestibulo-ocular reflex bilaterally. The dog scored 12 using the modified glasgow coma scale (MGCS) (Figure 2).[6] The neuroanatomical localisation was multifocal, with brainstem, forebrain and diencephalon involvement.

INVESTIGATIONS

Initial investigations included haematology, serum biochemistry, C-reactive protein and resting bile acids. All were within normal limits other than a very mild hypernatremia suggesting dehydration (Na 162 mmol/L ref.:144–160 mmol/L). An MRI of the brain was performed, no abnormalities were noted. Cisternal cerebrospinal fluid was collected for analysis, the results of which were within normal limits.

Given the initial diagnostic findings and the clinical suspicion for a toxin, Naloxone (Narcan) was given intravenously at a dose of 0.02 mg/kg, in addition to intravenous fat emulsion (Intralipid 20%; Fresenius Kabi) at a dose of 0.5 mL/kg/min. Urine was taken for toxicology for 'drugs of abuse'. Upon recovery from his general anaesthetic, the dog passed dark faeces, a significant proportion (approximately 80%) consisted of a red berry (Figure 3). When the owner was further questioned, she did report that her husband had been decanting sloe berries (750 g) from home-made gin the previous evening (Figure 4). He had deposited the berries at approximately 8 pm on the compost heap. The dog was reported to be allowed outside to urinate an hour later, it was at this time the owner suspected he could have ingested the berries. The owner checked the compost heap and found that the berries were missing.

Given this new information a tentative diagnosis of ethanol toxicity was made, and serum was submitted to an outside laboratory for ethanol concentration and was determined to be 3.1 g/L. In humans a toxic concentration is dependent on individual tolerance and usage; concentration greater than 3.0–4.0 g/L can be fatal due to respiratory depression. In the UK the drink driving limit is 0.8 g/L. Urinalysis was negative for drugs of abuse.

LEARNING POINTS/TAKE-HOME MESSAGES

- Toxin ingestion should be high on the list of differentials with an acute onset of encephalopathic signs.
- Ethanol toxicity is characterised by varying neurological deficits including ataxia, tetraparesis, bilateral miosis and altered mentation. Other effects include dehydration with resulting tachycardia, hypothermia, gastrointestinal signs (vomiting) and, in severe cases, respiratory depression.
- When toxicity is suspected, appropriate samples should be taken for testing (vomitus, blood, urine, faeces).
- Ethanol toxicity is managed supportively with decontamination in the acute phase of ingestion, IVFT, correction of acid-base derangements, maintaining body temperature, supportive oxygen therapy as required. Intravenous lipid infusion and charcoal have not been shown to be effective.
- This case presented with severe neurological signs but made a full recovery after time and IVFT. If toxicity is suspected but financial restraints prevent full investigation, then it is worthwhile providing simple supportive care.
- In cases of suspected toxicity, it is important for the owners to search the home for evidence of ingestion and to ask everyone in the family or who has had contact with the animal, whether they are aware of any toxin ingestion.

DIFFERENTIAL DIAGNOSIS

Toxic or metabolic encephalopathy was considered the most likely differential for a young previously healthy dog presented with acute onset semi-coma. The most common causes of neurotoxicity seen in our practice are metabolic problems such as hepatic encephalopathy and ingestion of illicit drugs such as cannabis and cocaine. The dog was considered unusual in his presentation in that some of his signs were slightly lateralising whereas encephalopathies are usually symmetrical. The next most likely differentials were trauma or haemorrhagic and ischemic stroke. Infectious and non-infectious encephalitis were considered unlikely differentials as given the patient signalment and history. Space occupying lesions (e.g. neoplasia or abscessation) were considered unlikely because signs were: acute in onset; only slightly

Modified Glasgow Coma Scale	
	Score
Motor activity	
Normal gait, normal spinal reflexes	6
Hemiparesis, tetraparesis, or decerebrate rigidity	5
Recumbent, intermittent extensor rigidity	4
Recumbent, constant extensor rigidity	3
Recumbent, constant extensor rigidity with opisthotonus	2
Recumbent, hypotonia of muscles, depressed or absent spinal reflexes	1
Brainstem reflexes	
Normal PLR and oculocephalic reflexes	6
Slow PLR and normal to reduced oculocephalic reflexes	5
Bilateral unresponsive miosis with normal to reduced oculocephalic reflexes	4
Pinpoint pupils with reduced to absent oculocephalic reflexes	3
Unilateral, unresponsive mydriasis with reduced to absent oculocephalic reflexes	2
Bilateral, unresponsive mydriasis with reduced to absent oculocephalic reflexes	1
Level of consciousness	
Occasional periods of alertness and responsive to environment	6
Depression or delirium, capable of responding, but response may be inappropriate	5
Semicomatose, responsive to visual stimuli	4
Semicomatose, responsive to auditory stimuli	3
Semicomatose, responsive only to repeated noxious stimuli	2
Comatose, unresponsive to repeated noxious stimuli	1
MCGS Score	**Score**
3-8	**Grave**
9-14	**Guarded**
15-18	**Good**

FIGURE 2 The modified glasgow coma scale

FIGURE 3 *Prunus spinosa* (sloe) passed in the dog's faeces

asymmetrical; and suggested both forebrain and brainstem involvement but with no signs suggesting elevated intracranial pressure such as cervical pain or vestibular disease.

TREATMENT

After diagnosis of ethanol toxicity, the dog was hospitalised for 12 h for monitoring, intravenous fluid therapy (IVFT) and paracetamol for anticipated headache. The dog initially received intensive one to one care with the multiparameter monitor at bedside monitoring ECG, pulse oximetry and blood pressure. Within an hour from extubation post-general anaesthesia, the dog improved rapidly. He was alert, responsive with normal brainstem reflexes and ambulatory within 1 h (MGCS = 16). He was discharged the following morning, he was neurologically normal (MGCS = 18).

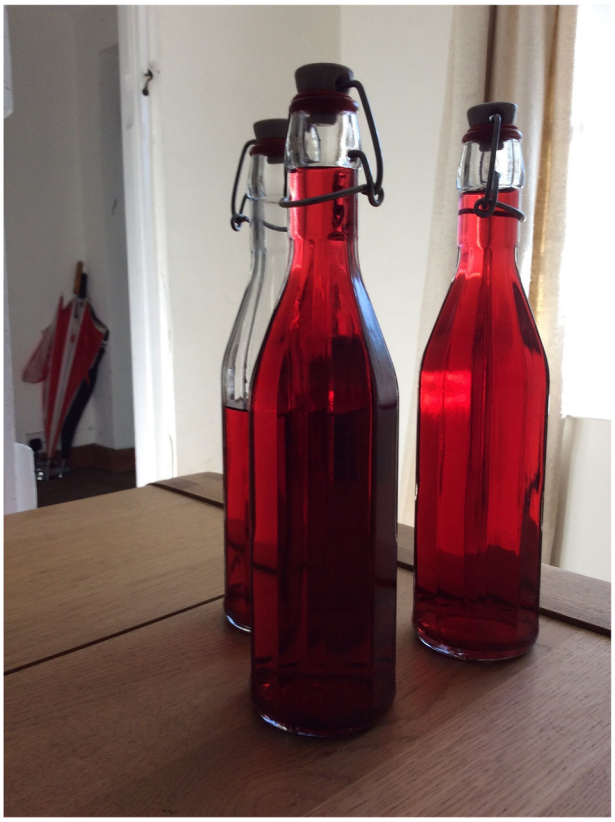

FIGURE 4 Sloe gin distilled by the dog's owner

FIGURE 5 *Prunus spinosa* (sloe) flowering plant

OUTCOME AND FOLLOW-UP

The owners reported that the dog was bright at home, eating well, and they had no concerns (Figure 4). There were no long-term adverse effects from the intoxication.

DISCUSSION

Sloe berries (sloes) are the fruit of *Prunus spinosa*, a species of flowering plant (Figure 5). Sloes ripen in autumn and tend to be harvested in October or November in the UK. Sloes and gin are combined to make the British red liqueur 'Sloe Gin'.[7]

Ethanol has a rapid absorption via the gastrointestinal tract following oral ingestion.[8,9] It rapidly crosses cell membranes resulting in rapid equilibration between intra and extra-cellular concentrations.[10] it's hyperosmolality can result in osmotic diuresis, resulting in profound dehydration.[8,9,11] It also acts as an irritant to the gastric mucosa directly stimulating vomiting, which in turn further exacerbates dehydration and electrolyte disturbances. Other effects of ethanol include peripheral vasodilatation with resulting hypothermia.[8] There can also be loss of urinary continence.[12]

From a neurological point of view, ethanol has a low molecular weight and can cross the blood-brain barrier to exhibit a neurotoxic effect.[11] This can manifest as excitation, vomiting, poor coordination, and in toxic doses can result in potent central nervous system depression and neuronal death. Death from ethanol toxicity is primarily associated with respiratory depression, metabolic acidosis or aspiration pneumonia.[8,13]

The lethal dose of pure alcohol in veterinary literature has been reported to vary from 5500–8000 mg/kg in a single dose.[10,13,14] The percentage alcohol by volume (mL of ethanol per 100 mL of solution) in gin is variable based on different production methods, ranging from 37.5-96%.[15] The density of 95% ethanol is approximately 816 g/L.[16] This would mean that for a 27 kg dog, the lethal dose of ethanol would be 182–265 mL of pure alcohol. Clinical features of alcohol toxicity are associated with blood alcohol concentration (BAC). A BAC of >1 g/L neurological signs such as ataxia, nystagmus and hyperreflexia can be expected. At >2 g/L nausea, vomiting and hypothermia respiratory depression occurs at 400–500 mg/dl. Coma at 4.5–5.0 g/L and death above 6.0 g/L.[8,10,17] This dog had BAC of 3 g/L at the time of sampling (16 h post ingestion). In human medicine this BAC would be associated with stupor, blackout and total loss of consciousness,[18] it would also be almost four times above the drink driving limit (0.8 g/L).[19] In both the dog and humans, the rate of ethanol detoxification ranges from the rate of ethanol detoxification ranges from 0.15–0.2 g/L/h. In a human study, the time taken to achieve peak BAC was on average 36 ± 10 min post consumption of neat spirits on an empty stomach.[20] Should this theory be applied, and the dog's BAC be achieved from an initial single dose of ethanol, peak ethanol concentration could have been as high as 6.2 g/L. This concentration would likely be fatal; thus, it therefore seems likely that the presence of the berries may have slowed the ethanol absorption. By lowering the rate of absorption, the alcohol concentration gradient is lowered; this is why peak BAC is higher if ethanol is ingested in a single dose rather than several smaller dose.[9]

BAC depends on the amount of alcohol consumed, the presence of food in the stomach and factors which affect gastric emptying and the rate of alcohol oxidation.[9] The volume of berries consumed will have caused gastric distension, reducing the rate of gastric emptying and alcohol absorption, thus

limiting the dogs BAC. There was one reported case of a dog that died from ethanol toxicity following rotten apple ingestion with a serum ethanol concentration of 3 g/L.[3] The severity of toxic effects secondary to alcohol toxicity depends on both the BAC and the rate of elimination. In the dog in the present case report, alcohol metabolism was likely elevated due to his high nutritional state. Following a meal, human studies have demonstrated higher levels of alcohol dehydrogenase, enhanced mitochondrial oxygen update and increased hepatic blood flow.[9] Another factor to consider is basal metabolic rate, for instance, the lower the body weight the greater the rate of alcohol metabolism.[9]

Treatment for ethanol toxicity consists of supportive care, including correcting any acid-base arrangements, rehydration and oxygen supplementation in the event of respiratory compromise. This dog was given an intravenous fat emulsion before the realization occurred that ethanol was likely to be the inciting toxin. The use of an intravenous lipid emulsion is unlikely to be useful for ethanol toxicity because it is a hydrophilic molecule making it a water-soluble compound with poor lipid solubility.[21] In addition, alcohol related toxicity is secondary to the metabolism of ethanol via alcohol dehydrogenase into the reactive metabolite; acetaldehyde.[9] Acetaldehyde contributes to oxidative stress with resulting tissue damage and pathology.[22]

Gastrointestinal decontamination, achieved gastric lavage via a stomach tube, is indicated in the early management of many toxicities, however as the dog was defaecating the ingested berries when ethanol toxicity was considered likely then this was inappropriate and may have increased the risk of aspiration pneumonia. The use of activated charcoal was considered, however from the literature it is reported that due to the rapid absorption of ethanol from the gastrointestinal tract, it has been shown to be ineffective in binding ethanol.[23] Charcoal can also be an aspiration risk and may exacerbate the adverse gastrointestinal effects of alcohol.[24]

It was upon defaecation of the berries that the owners realized that the intoxication was likely secondary to the gin-soaked sloe berries. It is therefore important in cases with a very high toxin suspicion, to ensure that everyone that has been in contact with the animal is asked whether there is any possibility with contact with toxic substances and that the home is thoroughly searched. Owner education is crucial as part of history taking as they may not perceive some common toxins as toxic (such as raisins, onions, macadamia nuts and mold).

OWNER'S PERSPECTIVE

I left my young Labrador in perfect health and shut in his pen in the kitchen at nine in the evening and went to bed leaving my husband decanting his sloe gin. When I came down at twenty past five the next morning, to my horror, the dog was unable to stand and was extremely distressed. I contacted my emergency vet, and he was in their surgery by 6.30 am, if anything, in an even worse state. We left him there for assessment, but by around 9.30 we were contacted and told he was being referred to the neurological referral vets, who fortunately, were not far away. He was in their surgery by around 11.00 in a truly desperate state. We were completely unable at that stage to think of any reason for his dramatic and sudden collapse. Leaving him for further investigations, we were telephoned at around 4.30 pm to be told that upon waking from his anaesthetic he had passed a huge amount of what looked like berries…only then did the awful truth become apparent, that he had been let out for a late night pee, only to discover the gin soaked sloe berries that had been left on the compost heap. We will not be making any more sloe gin……ever.

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