Case Report

Treatment of accidental ethanol intoxication with hemodialysis in a dog

Lisa A. Keno, DVM, DACVIM and Cathy E. Langston, DVM, DACVIM

Abstract

Objective – To describe the successful treatment of accidental ethanol intoxication with hemodialysis in a dog.

Case Summary – A 1.5-year-old female intact mixed breed dog was presented in a comatose state believed to be due to ethanol intoxication. The initial 9 hours of supportive care treatment were complicated by multiple seizures and hypothermia, and resulted in only minimal improvement in the dog’s level of consciousness. Hemodialysis was implemented and resulted in rapid clinical recovery, corresponding to a rapid decline in serum ethanol concentration.

New or Unique Information Provided – To the authors’ knowledge, this is the first reported case of using hemodialysis to treat accidental ethanol intoxication in a dog. The patient’s initial serum ethanol concentration was higher than those previously reported for cases of accidental ethanol intoxication in dogs, and the serum ethanol concentration was shown to rapidly decline during hemodialysis. Treatment with hemodialysis for severe ethanol intoxication was effective in this case and may be able to decrease morbidity and mortality in some cases.

Keywords: dialysis, ethyl alcohol, extracorporeal, toxicity, toxicosis

Introduction

Ethanol intoxication is common in people and has been reported infrequently in dogs. Treatment for both species is generally limited to supportive care.1–3 Hemodialysis is an effective means of enhancing elimination of ethanol from the body and can therefore be beneficial in cases expected to be potentially life threatening or associated with high morbidity.1,3–9 There are several reports of successfully using hemodialysis to treat ethanol intoxication in people,6–9 and 1 report describing the use of hemodialysis in an experimental model of ethanol intoxication in dogs.10 To the authors’ knowledge, this is the first reported case of using hemodialysis to treat a case of accidental ethanol intoxication in a dog.

Case Summary

A 1.5-year-old female intact mixed breed dog (Poodle/Yorkshire Terrier cross) weighing 3.76 kg was presented for acute ethanol intoxication. The owners reported returning home after being out for several hours to find the dog recumbent next to an open bottle of 80 proof generic vodka. The exact volume ingested was unknown, but the owners reported that the dog may have ingested as much as 375 mL. The dog had no prior medical history and was not currently being administered any medications.

On presentation, the patient was laterally recumbent, tremoring, unresponsive to noxious stimuli, and therefore assessed as comatose. Oral mucous membranes were pink and tacky with a prolonged capillary refill time (42 s). Pupils were miotic, palpebral reflexes were intact, and menace response was absent. The dog was tachycardic (180 beats/min), tachypneic (60 breaths/min), and hypothermic at 34.7°C (94.5°F). The remainder of the physical exam was unremarkable. The dog was hypotensive at 56 mm Hg (assessed via ultrasonic Doppler technique, which was interpreted as an approximation of mean arterial pressure6), and this responded to a 100 mL crystalloid fluid bolus and heat support. Blood pressure increased to 130 mm Hg (again using ultrasonic Doppler technique) following the fluid bolus and warming. Venous blood gas parameters and electrolytes were compatible with a respiratory acidosis, but were otherwise normal (see Table 1). The patient was admitted to the intensive care unit at approximately midnight for continued monitoring and...
supportive care. Monitoring included continuous ECG, blood pressure (q 2–4 h), rectal temperature (q 2 h), heart rate (hourly), respiratory rate (q 2 h), and frequent examination by the attending veterinarian (q 1–2 h depending on the patient’s status). Treatments included famotidine\(^{c}\) (0.5 mg/kg, IV), maropitant\(^{d}\) (1 mg/kg, SC), and IV crystalloid fluids\(^{b}\) (3.9–5.3 mL/kg/h) with 20 mEq KCl/L\(^{2}\).

At 1 AM the patient had a generalized seizure consisting of chewing activity, rapid blinking, and limb paddling with simultaneous sinus tachycardia (180/ min). Diazepam\(^{f}\) was administered to effect (1 mg/kg) to stop the seizure. At 2 AM, a second generalized seizure with simultaneous sinus tachycardia (210/min) occurred. Approximately 1.3 mg/kg diazepam was required to stop the seizure. At that time, pupil size was normal with intact pupillary light reflexes. Within 5 minutes of administering diazepam, seizure activity recurred. The patient was again treated with a diazepam bolus (0.66 mg/kg, IV) and then started on a continuous rate infusion (CRI) of diazepam at 0.5 mg/kg/h. At 3:50 AM, pupils were miotic, palpebral reflexes were intact, swallow reflex was absent and muscle tone was flaccid. At 4 AM, another generalized seizure resolved following 0.5 mg/kg diazepam. The diazepam CRI was increased to 0.66 mg/kg/h. At this time 1 g/kg mannitol\(^{b}\) was administered IV. At 5:35 AM, palpebral reflexes were intact, pupillary light reflexes were absent, swallow reflex was absent, and muscle tone was flaccid. At 6:45 AM, there had been no further seizure activity and the patient was again assessed as comatose. At this point, the diazepam CRI was decreased slightly to 0.6 mg/kg/h. At 7:40 AM, the patient had moderate jaw tone, an absent swallow reflex, absent palpebral reflexes, and pinpoint pupils.

Continuous monitoring and support was provided throughout the night. Heat support was provided with a circulating-water blanket but the temperature ranged from 35.5\(^{1}\) to 37.5\(^{1}\) (96–99.5\(^{1}\)). After the initial crystalloid fluid bolus, blood pressure varied from normal to hypertensive (indirect mean blood pressure\(^{h}\) between 96 and 192 mm Hg). Two additional panels of venous blood gas parameters and electrolytes were evaluated (see Table 1). They were similar to the initial measurements; a respiratory acidosis persisted with no other abnormalities. A continuous electrocardiogram was used to monitor for cardiac arrhythmias and a sinus rhythm was noted throughout the night. Additional supportive care included ocular lubrication, urinary bladder expression, and rotation of the patient’s down side.

At 8:45 AM, approximately 9 hours after presenting to the hospital, the patient responded minimally to noxious stimuli and was therefore assessed as stuporous. The dog was recumbent with elevated nictitans membranes, miotic pupils, and an absent swallow reflex. Because of the severity of signs overnight and the persistent decreased level of consciousness, hemodialysis was recommended to hasten removal of ethanol from the body and potentially decrease morbidity associated with the intoxication. Obtaining client consent, making financial arrangements, placing the dialysis catheter, and setting up the dialysis machine delayed the actual start of dialysis for several additional hours.

A temporary 7-Fr double-lumen 20 cm dialysis catheter\(^{1}\) was placed in the right jugular vein via the seldinger technique and hemodialysis was initiated at 2 PM, approximately 14 hours after presentation to the hospital. A hemodialysis machine\(^{1}\) was used with a low-volume extracorporeal circuit\(^{b}\) (40 mL volume) and dialyzer\(^{l}\) (28 mL volume, 0.4 m\(^{2}\) surface area, polysulfone membrane). The extracorporeal circuit was primed using 3% dextran (6% dextran 70 in 0.9% sodium chloride\(^{m}\) diluted 50:50 with 0.9% sodium chloride\(^{m}\)). Unfractionated heparin\(^{n}\) was used as the anticoagulant. The average blood flow rate was 50 mL/min, and a total of 11.525 L of blood were processed during the 3-hour, 53-minute treatment. Mannitol (1.875 g) was administered 1 hour after initiating dialysis. The sodium profile of the dialysate was increased gradually from an initial concentration of 145 to 150 mmol/L over the treatment. Although dialysis treatment was prescribed for 5 hours, the treatment was discontinued early due to excellent patient response. The patient regained a normal state of consciousness during dialysis and was eating and drinking before the treatment was finished. Immediately after the dialysis treatment ended, the patient was able to walk unassisted. IV fluids were discontinued overnight. The following morning, the dialysis catheter was removed, and the patient was discharged from the hospital. The patient

---

**Table 1: Venous blood gas and electrolyte panels from a dog with severe ethanol intoxication assayed at presentation, 1.5 hours after presentation, and 8 hours after presentation**

<table>
<thead>
<tr>
<th>Venous sample measurement (U)</th>
<th>Presentation</th>
<th>1.5 hours after presentation</th>
<th>8 hours after presentation</th>
</tr>
</thead>
<tbody>
<tr>
<td>pH</td>
<td>7.25</td>
<td>7.344</td>
<td>7.309</td>
</tr>
<tr>
<td>pCO(_2) (mm Hg)</td>
<td>49.3</td>
<td>45.1</td>
<td>48.4</td>
</tr>
<tr>
<td>Na (mmol/L)</td>
<td>147.5</td>
<td>140.4</td>
<td>142.5</td>
</tr>
<tr>
<td>K (mmol/L)</td>
<td>3.65</td>
<td>3.08</td>
<td>3.65</td>
</tr>
<tr>
<td>HCO(_3) (mmol/L)</td>
<td>21.2</td>
<td>24.0</td>
<td>23.8</td>
</tr>
<tr>
<td>Anion gap (mmol/L)</td>
<td>23</td>
<td>14.5</td>
<td>16.4</td>
</tr>
<tr>
<td>Glucose (mmol/L)</td>
<td>5.1 [91]</td>
<td>6.6 [120]</td>
<td>6.7 [122]</td>
</tr>
<tr>
<td>Lactate (mmol/L)</td>
<td>1.57</td>
<td>0.43</td>
<td>0.78</td>
</tr>
</tbody>
</table>

---

1. L.A. Keno & C.E. Langston
Discussion

Ethanol intoxication is a common problem in people. Ethanol is a selective CNS depressant at low doses and a general depressant at high doses. Initially, ethanol depresses areas of the brain involved with highly integrated functions. Signs associated with mild cases of inebriation are highly variable, ranging from energetic or gregarious to ill-tempered behavior. As the degree of intoxication increases, successive impairment of neuronal activity causes a variety of signs including lethargy, disorientation, and aggression. With severe intoxication, there is loss of airway protective reflexes, coma, and increasing risk of death from respiratory depression. Common clinical signs in people include tachycardia, hypotension, hypothermia, hypoventilation, mydriasis, nystagmus, vomiting, dysarthria, muscular incoordination, ataxia, altered consciousness, and coma. Ethanol-induced seizures have been reported in adults but are more common in children and may be associated with hypoglycemia in some cases. Hyperosmolarity is expected, and a high anion gap metabolic acidosis can occur with chronic exposure, especially if combined with starvation.

Treatment for ethanol intoxication in people consists mostly of monitoring and supportive care with supplemental oxygen and IV fluids. Supplementation with thiamine, folate, magnesium, and dextrose is sometimes provided. Occasionally, a severely intoxicated or comatose patient requires intubation and respiratory support. Hemodialysis is an effective means of enhancing ethanol clearance and can therefore be used in cases expected to be associated with high morbidity or mortality.

Blood ethanol concentrations of 87–109 mmol/L (400–500 mg/dL) and above are considered severe and potentially life-threatening in people. In dogs, ethanol intoxication may result in behavioral changes (eg, excitability and vocalization), a depressed state of consciousness (including coma), vomiting, ataxia, hypothermia, seizures, and respiratory or cardiac arrest. Treatment is usually supportive. The reported oral lethal dose of ethanol in dogs is 5–8 g/kg.

A small number of studies have evaluated the effects of ethanol intoxication on dogs following measured administration of toxic doses of ethanol. In a 1958 study by Kingman et al, dogs were administered a lethal dose of ethanol via stomach tube (12 g/kg). Most dogs died of respiratory failure (65%) with the remainder dying of progressive hypotension and resultant circulatory failure. In a 1967 study by Garriott et al, dogs were administered ethanol IV at a rate of 1.25 g/kg hourly until death occurred. In this study, signs of circulatory failure (eg, decreased heart rate and hypotension) began 3 hours after initiation of ethanol infusion, at a mean blood alcohol concentration of 115 mmol/L (530 mg/dL); these signs of circulatory failure preceded respiratory difficulty by at least 1 hour. The mean venous alcohol concentration was 150 mmol/L (690 mg/dL) at 5 hours, which was 45 minutes before the average time of death.

Prior reports of accidental ethanol intoxication in dogs have been secondary to ingestion of ethanol containing beverages, rotten apples, sourdough bread, uncooked pizza dough, and bread dough. In previous case reports, most patients recovered with supportive care and various treatments including gastric lavage, IV fluids, vitamin injections, dextrose, and antimicrobial administration. One prior report described a dog that became comatose and died following ethanol intoxication; however, hepatic damage secondary to chronic ingestion of rotten apples (presumed to reflect chronic ethanol toxicity) was suspected to play a role in the death of this dog. There is a previous case report of a dog that survived severe ethanol intoxication following mechanical ventilation.

Ethanol intoxicated patients can suffer from a variety of derangements in their acid-base status, electrolytes, and osmolarity, some of which may be further impacted by the patient’s clinical manifestations of intoxication, nutritional status, and the presence or absence of chronic exposure. Ethanol is expected to cause an
increase in the osmolar gap; however, serum osmolarity was not measured in the current case. When alcohol is metabolized to acetyl-CoA, NAD+ is reduced to NADH + H+. The increase in NADH/NAD+ in addition to a contraction in the extracellular fluid volume can lead to lactic acidosis. Unlike some of the other alcohols (such as methanol and ethylene glycol), high anion gap metabolic acidosis is not a key feature of acute ethanol intoxication. A high anion gap can be seen in people with chronic alcohol exposure, especially if chronic exposure is combined with starvation. The resultant alcoholic ketoacidosis and high anion gap thus is unlikely to be a feature of acute intoxication in veterinary patients. The current patient demonstrated a persistent respiratory acidosis, likely secondary to respiratory depression and hypoventilation, which is a known complication of ethanol intoxication both in people and animals. A variety of other clinical findings have the potential to further impact the acid-base status of ethanol-intoxicated patients, including but not limited to dehydration, hypovolemia, hypoxia, seizures, and vomiting. Hypoglycemia has been reported in cases of ethanol intoxication in people and dogs but was not noted in the current case.

Hemodialysis can be used to hasten the removal of ethanol as well as many other toxins from the body. Both dialysis-related factors and toxin-related factors (including pharmaceuticals) must be considered when determining whether or not hemodialysis is an option in a particular toxicity. Properties of a toxin that make it amenable for removal by hemodialysis include a low molecular weight, poor protein binding, and a small volume of distribution. Low molecular weight compounds (defined as <500 Da) more readily cross dialysis membranes than larger molecules. Protein binding makes toxins unsuitable for removal via dialysis, so poorly protein bound (<80% bound) compounds are more readily dialyzable than those that are highly protein bound. Toxins with a small or moderate volume of distribution (≤1 L/kg) are more likely to be removed by hemodialysis. The larger the volume of distribution of a toxin, the higher the degree of tissue binding, and less available the compound is for removal from plasma by hemodialysis. Ethanol is readily dialyzed because of its low molecular weight (46 Da), poor protein binding, and small volume of distribution (0.6 L/kg). Charcoal hemoperfusion can also be considered as a treatment option for some intoxications. Compared with hemodialysis, charcoal hemoperfusion can be used to remove larger toxicants that are more highly protein bound and more lipid soluble (have a larger volume of distribution). Combination hemodialysis/hemoperfusion has been used to successfully treat baclofen toxicity in a dog and enrofloxacin overdose in a cat. In the current case, hemodialysis was chosen over charcoal hemoperfusion because the characteristics of ethanol are ideal for removal by hemodialysis.

In a 1960 study by Marc-Aurele et al., dogs were used as a human model to study the efficacy of using hemodialysis in the treatment of experimental ethanol intoxication. Dogs were administered ethanol at a dose of 3.2–6 g/kg. In this study, dialysis achieved removal of ethanol 3.8–4.3 times faster than physiologic elimination. In this study, all ethanol control animals (those not treated with dialysis) remained unconscious and unresponsive during the 10-hour duration of the study, while the dialyzed dogs were conscious by the second hour of treatment.

Although uncommonly required, there are several reports of using hemodialysis to treat ethanol-intoxicated human patients. Various published reports on the use of hemodialysis for ethanol-intoxicated human patients have described hemodialysis in the following clinical situations: coma, marked hyperosmolarity, multiple seizures, and progressive CNS depression. In one report, the authors argued that although many intoxicated victims can recover when properly treated with supportive therapy alone, some individuals with blood alcohol concentrations 87–109 mmol/L (400–500 mg/dL) or higher will die despite the most efficient conservative management. For this reason, they recommended that hemodialysis be considered as a treatment option for severely affected cases of ethanol intoxication. Another author stated that dialysis may be of value in severe intoxications, and defined severe as those cases having a blood ethanol concentration >87 mmol/L (>400 mg/dL).

Although the exact amount of ethanol ingested by the dog in the current case described was unknown, a potentially fatal dose was presumed to have been consumed. If the patient had indeed ingested the amount estimated by the owners (375 mL of 80 proof generic vodka), this would have been approximately 32 g/kg, which is well above the reported lethal dose in dogs. It is unknown whether gastric lavage at presentation would have hastened recovery. Gastric lavage was not performed because the dog’s decreased state of consciousness was thought to increase the risk of aspiration associated with this procedure.

The dog’s initial serum ethanol concentration of 132 mmol/L (610 mg/dL) is well above the level considered to constitute severe and potentially fatal in people (87–109 mmol/L [400–500 mg/dL]). To the authors’ knowledge, this is the highest blood ethanol concentration that has been reported secondary to accidental ethanol ingestion in veterinary medicine. During the dialysis treatment (just under 4 h), the
Hemodialysis was demonstrated to effectively and rapidly decrease blood ethanol concentrations, and this application of hemodialysis may be able to decrease morbidity and mortality in some severe cases of ethanol toxicity.

**Footnotes**

a. Ultrasonic Doppler Flow Detector, Parks Medical Electronics Inc, Aloha, OR.
c. Pepcid, APP Pharmaceuticals, Schaumburg, IL.
d. Cerenia, Pfizer, New York, NY.
e. Potassium Chloride, Hospira Inc, Lake Forest, IL.
f. Diazepam, Hospira Inc.
g. 25% Mannitol, Hospira Inc.
h. Cardell Veterinary Monitor 9403, Sharn Veterinary, Tampa, FL.
i. Arrow-hooves Pediatric multilumen catheterization set, Arrow International Inc, Reading, PA.
j. Cole Century System 3, Gambro Renal Products, Lakewood, CO.
k. Gambro low volume pediatric, Gambro Renal Products.
l. Hemoflow F3, Fresenius Medical Care North America, Waltham, MA.
m. 0% Gentran 70, Baxter Healthcare Corporation.

**References**