

**Canine ethanol toxicosis associated with accidental wine exposure: clinical findings and literature review**

**Intoxicação canina por etanol associada à exposição acidental ao vinho: achados clínicos e revisão de literatura**

**Toxicosis etanólica canina asociada a la exposición accidental al vino: hallazgos clínicos y revisión de la literatura**

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Júlia Nardi Rodrigues<sup>1</sup>  
Isabela Bazzo da Costa<sup>2</sup>  
Laura Beatriz Sabino<sup>3</sup>  
Beatriz dos Santos Bueno<sup>4</sup>  
Rodrigo Prevedello Franco<sup>5</sup>  
Carlos Eduardo de Siqueira<sup>6</sup>

**ABSTRACT:** Ethanol poisoning in dogs is uncommon but potentially serious, occurring mainly after accidental ingestion of alcoholic beverages, fermented foods, or alcohol-containing products. We report the case of a three-month-old Border Collie bitch weighing 6.5 kg, treated after accidental ingestion of wine in a home environment. The patient presented ataxia, disorientation, motor incoordination, alcoholic halitosis, and superficial traumatic injuries due to contact with glass fragments. Laboratory tests showed discrete alterations compatible with stress and mild dehydration. The diagnosis was established based on anamnesis and clinical signs, without serum ethanol dosage. The treatment consisted of intravenous fluids, antiemetics, and management of skin lesions, resulting in complete clinical recovery in less than 24 hours. The case highlights the importance of early diagnosis, immediate supportive therapy and preventive guidance to owners.

**Keywords:** alcohol; canine; emergency; veterinary toxicology.

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<sup>1</sup> Bachelor of Veterinary Medicine, Universidade de Marília (UNIMAR), Marília, São Paulo, Brazil.  
E-mail: julianardir@gmail.com

<sup>2</sup> Postdoctoral Fellowship in Genetics, Universidade de Marília (UNIMAR), Marília, São Paulo, Brazil.  
E-mail: isabelabazzo@unimar.br

<sup>3</sup> Bachelor of Veterinary Medicine, Universidade de Marília (UNIMAR), Marília, São Paulo, Brazil.  
E-mail: vetlaurasabino@gmail.com

<sup>4</sup> Bachelor of Veterinary Medicine, Universidade de Marília (UNIMAR), Marília, São Paulo, Brazil.  
E-mail: buenobeatriz1999@gmail.com

<sup>5</sup> Doctor of Veterinary Medicine, Universidade de Marília (UNIMAR), Marília, São Paulo, Brazil.  
E-mail: vetrpf@yahoo.com.br

<sup>6</sup> Ph.D. in Animal Science, Universidade de Marília (UNIMAR), Marília, São Paulo, Brazil.  
E-mail: carlossiqueira@unimar.br

## 1 INTRODUCTION

Ethanol poisoning in dogs is considered a relatively uncommon occurrence in veterinary clinical routine, but it has a high potential for severity due to the important systemic and neurological effects promoted by alcohol. Exposure can occur by different routes, most often related to the accidental ingestion of alcoholic beverages, fermented masses, cleaning products, cosmetics, medicines and food waste containing alcohol or undergoing fermentation. In recent years, the growing coexistence of domestic animals within the family environment has increased the risk of access to potentially toxic substances, making domestic accidents an important cause of emergency care in veterinary medicine (Spinosa; Górnaiak; Bernardi, 2017; Plumlee, 2013).

Ethanol has rapid gastrointestinal absorption and high tissue distribution due to its high solubility in water and lipids. Once absorbed, it easily crosses the blood-brain barrier, promoting depression of the central nervous system through the potentiation of GABA receptors and inhibition of NMDA glutamate receptors (Spinosa; Górnaiak; Bernardi, 2017). As a consequence, poisoned animals can show clinical signs ranging from mild behavioral changes to coma, respiratory failure, and death. In addition to the neurological effects, alcohol intoxication can trigger important metabolic changes, including metabolic acidosis, hypoglycemia, dehydration, and electrolyte disturbances, especially in puppies and small patients (Plumlee, 2013).

Although alcoholic beverages are often associated with alcohol poisoning, several reports show that fermentative foods represent an important source of exposure in dogs. Thrall *et al.* (1984) described a severe case of poisoning after ingestion of sourdough containing *Saccharomyces cerevisiae*, in which the patient presented severe respiratory depression and high serum ethanol concentration. Similarly, Suter (1992) reported an outbreak of poisoning in dogs fed raw pizza dough in fermentation, reinforcing the toxic potential of endogenous ethanol production in the gastrointestinal tract. In these cases, in addition to the systemic effects of alcohol, there is an accumulation of carbon dioxide, increasing the risk of gastric dilation and torsion (Osweiler *et al.*, 2011).

Other less common sources have also been described in the veterinary literature. West and Rusbridge (2021) reported alcohol poisoning in a Labrador Retriever after ingestion of gin-soaked fruit, showing that even improperly disposed of food waste can pose a significant risk to domestic animals. The authors also point out that many cases end up being initially confused

with primary neurological diseases or other intoxications, mainly due to the absence of immediate laboratory confirmation and the difficulty in obtaining a detailed anamnesis.

The clinical signs observed in ethanol poisoning are widely variable and depend on the amount ingested, the rate of absorption, the presence of food in the stomach, the alcohol concentration of the substance and the individual susceptibility of the patient. Among the most frequently described findings are ataxia, disorientation, vomiting, drooling, tremors, hypothermia, hypoglycemia, hypotension, bradycardia, pupillary changes, respiratory depression, and coma. In severe cases, seizures, bronchoaspiration, respiratory failure, and death may occur (Peterson; Talcott, 2013; Kahn; Line, 2016; Plumlee, 2013; Osweiler *et al.*, 2011). Keno and Langston (2011), when reporting a severe case of vodka poisoning in a dog, observed recurrent seizures, severe hypotension and coma, requiring hemodialysis to reverse the clinical picture.

The diagnosis of alcohol intoxication is mainly based on the association between detailed anamnesis, compatible clinical signs and complementary tests. When performing complementary tests, changes in liver markers and lactate elevation may occur. Serum ethanol measurement is an important confirmatory tool, but its unavailability in the veterinary clinical routine limits its use in many cases. Thus, the early recognition of clinical signs and the identification of the possible source of exposure become fundamental for the rapid establishment of therapy (Peterson; Talcott, 2013; Kahn; Line, 2016; Plumlee, 2013; Osweiler *et al.*, 2011).

Treatment is predominantly symptomatic and supportive, including hemodynamic stabilization, hydroelectrolyte correction, body temperature control, ventilatory support when necessary, and intensive monitoring of neurological and respiratory functions (Peterson; Talcott, 2013; Kahn; Line, 2016). Most patients have a satisfactory recovery when care occurs early, however severe cases can quickly progress to respiratory failure and death (Plumlee, 2013; Osweiler *et al.*, 2011).

To avoid these cases, it is of paramount importance to educate the tutor to store alcoholic beverages and cleaning products correctly and out of the reach of pets, also avoid offering raw pasta to animals and always keep them out of reach. As soon as the ingestion of any of the products mentioned later is noticed, it is recommended to immediately go to the veterinarian (Spinosa; Górnjak; Bernardi, 2017; Bowman, 2021).

In view of the scarcity of information and reports involving alcohol poisoning in dogs, especially related to the ingestion of alcoholic beverages in the home environment, the present study aims to describe a case of acute ethanol poisoning in a puppy dog after accidental

ingestion of wine, addressing the main clinical, diagnostic and therapeutic aspects involved, in addition to discussing the findings in the light of the current veterinary literature.

## **2 THEORETICAL FRAMEWORK**

### **2.1 Etiology**

The most common sources of alcohol poisoning in Brazilian homes are represented by alcoholic beverages and cleaning products, which end up being attractive to dogs due to the often sweet smell and taste. Cosmetic products such as perfumes also attract the animal due to their distinctive smell. In the case of perfumes and cleaning products, the intake becomes more worrisome, as only a low dose can cause several risks to the patient's health. The rapid ingestion and size of the animal, in addition to the contents in its stomach, also contribute to the potency that the intoxication will have (Peterson; Talcott, 2013; Plumlee, 2013).

One of the little-known processes that cause alcohol intoxication is the endogenous production of ethanol, which occurs through fermentation within the patient's body. This fact occurs due to the ingestion of raw bread dough, the process of releasing a bacterium called *Saccharomyces cerevisiae*, this bacterium has ethanol and CO<sub>2</sub> as its main products, so the patient who ingested the pasta will manifest the clinical signs of alcohol intoxication along with distention and severe abdominal pain caused by CO<sub>2</sub> (Osweiler *et al.*, 2011; Kahn; Line, 2016).

There are several patient-related factors that alter the intensity of the clinical conditions such as age, low glycemic stock, patient weight and patients with liver diseases that lead to less ability to biotransform alcohol. The speed of ingestion and the type of product ingested also change the severity of the case, products that contain a higher dose of alcohol have a higher intoxicating power. Co-exposure is also a risk factor for patients, making patients who already ingest some type of central nervous system depressant medication more severe (Spinosa; Górnaiak; Bernardi, 2017; Peterson; Talcott, 2013).

### **2.2 Pathophysiology**

Canine alcohol intoxication begins at the time of ingestion of the toxic agent, when it reaches the gastric tract it is easily absorbed, due to its high solubility in water and lipids, after which there is high dispersion to the tissues, one of them being the central nervous system where alcohol crosses the hetoencephalic barrier. In the central nervous system, alcohol has the

ability to potentiate GABA receptors at the same time that it inhibits the NMDA glutamate receptors, which causes depression of the central nervous system, leading to ataxia and sedation of the patient. In high doses, alcohol can cause respiratory depression, which will culminate in low peripheral perfusion as a compensatory mechanism, this low perfusion leads to a drop in temperature, especially in the extremities (limbs) (Peterson; Talcott, 2013; Kahn; Line, 2016).

In the liver, alcohol is transformed into acetaldehyde through the alcohol dehydrogenase process, after which acetaldehyde is converted into acetate through the aldehyde dehydrogenase process. As a result of these processes, it leads to an increase in free radicals. After these facts, there is an increase in NADH/NAD<sup>+</sup>, which leads to the deviation of functions, redirecting the metabolic pathways and compromising glycogenesis, thus leading the patient to hypoglycemic crises more easily, with more consequence in animals less than six months old because they already have a low glycogen stock. In addition, all this occurs together with the accumulation of lactate and ketone bodies, further increasing the risk of metabolic acidosis (Plumlee, 2013; Spinosa; Górnjak; Bernardi, 2017).

Regarding hypoglycemic crises caused by alcohol intoxication, these occur due to the failure to convert lactate into pyruvate and malate into oxaloacetate, reducing the availability of the substrates necessary for the glycogenesis process, without this process the patient becomes more prone to neurological dysfunctions such as coma and seizures. Along with these processes there is the accumulation of acetaldehyde, which promotes oxidative stress and mitochondrial dysfunction, which can lead to liver and neurological damage. In the urinary tract, osmotic diuresis and inhibition of vasopressin secretion lead to dehydration, along with myocardial depression caused by electrolyte disturbances (Peterson; Talcott, 2013; Soto-Blanco, 2021).

In the respiratory tract, depression of the bulbar centers and protective reflexes cause serious risks of bronchial aspiration to the patient. The effective sympathetic tone of the patient is also reduced, impairing the cardiovascular system, which can lead to hypotension, being moderate to severe depending on the dose ingested, in severe cases it can even lead to shock, this process leads to the need for hemodynamic correction therapy (Kahn; Line, 2016; Plumlee, 2013).

In the case of endogenous ethanol production by fermentation after ingestion of raw dough, there is in addition to all the damage caused by ethanol poisoning, the risk of gastric volvulus torsion and the presence of severe abdominal pain due to the accumulation of CO<sub>2</sub> caused by the fermentation of the dough. This type of absorption adds the injuries caused by

ethanol to those caused by fermentation, leading the patient to a much more critical case (Osweiler *et al.*, 2011; Kahn; Line, 2016).

### 2.3 Clinical Signs

The onset of the manifestation of clinical signs occurs shortly after the ingestion of ethanol, the changes linked to the central nervous system are the first to be noticed as momentary hyperactivity followed by lethargy and ataxia, over time the presence of nystagmus, dysmetria and hyporeflexia is noted in addition to drowsiness, in more serious cases the patient may present coma. Staggering gait, low pupillary response are also important findings during the patient's physical examination (Peterson; Talcott, 2013; Kahn; Line, 2016).

Other common signs are drooling, nausea and vomiting, in addition to abdominal distension when intoxication occurs from the ingestion of dough in the fermentation process. When presenting these signs, patient monitoring is of paramount importance due to the high risk of vomiting bronchoaspiration, which occurs due to the lack of reflex due to central nervous system depression. Along with these signs, it is possible to observe alcoholic halitosis and abdominal cranial hypersensitivity (Osweiler *et al.*, 2011; Plumlee, 2013).

On performing the detailed physical examination, it is also possible to notice tachycardia or bradycardia, hypotension and bradypnea, with a risk of hypoventilation. Low temperature is a classic sign that occurs due to low peripheral perfusion, along with this cause pale mucous membranes, and increased capillary filling time, in addition to low oximetry. In cases with greater severity, especially in the case of puppies, the patient presents changes in behavior, extreme weakness, tremors and seizures, these signs occur mainly due to hyponatremia and hypokalemia (Kahn; Line, 2016; Plumlee, 2013; Peterson; Talcott, 2013; Soto-Blanco, 2021).

In cases of greater severity, represented by high ethanol intake or frequent ingestion, there may be signs of aspiration such as cyanosis, rales, and respiratory effort. Other clinical signs present are oliguria, slow mydriasis, and lack of response to painful stimuli in addition to high-grade dehydration and coma (Osweiler *et al.*, 2011; Kahn; Line, 2016).

### 2.4 Diagnosis

The diagnosis of canine alcohol poisoning has a detailed anamnesis and a physical and neurological examination whose results point out the main clinical signs of intoxication, in addition to the collection of data such as access to products containing alcohol, the animal's

habit of ingesting objects and others that may cause damage to health. It is possible to add these data to the serum ethanol dosage that will close the diagnosis of alcohol intoxication. The importance of a detailed anamnesis is due to the number of differential diagnoses that exist, such as other poisonings by chemical products (Peterson; Talcott, 2013; Kahn; Line, 2016).

The collection of complementary tests is also necessary to aid in diagnosis and guide for better treatment choice. Immediate capillary glucose was collected, lactate dosage (high) and blood gas analysis (evidence of metabolic acidosis due to high lactate). In the blood count, hemoconcentration is noted due to dehydration, in the biochemical one a slight increase in liver enzymes and hypoglycemia is noted. Urinalysis shows high urine concentration, pigmenturia and ketonuria. In the case of abdominal augmentation, an abdominal ultrasound is indicated that will show the accumulation of gas and gastric distension. There are respiratory signs that recommend a chest X-ray to rule out bronchial aspiration (Plumlee, 2013; Soto-Blanco, 2021).

## **2.5 Treatment**

There is currently no drug available that is considered a reverser or antidote to the effects caused by ethanol ingestion, so treatment involves controlling and maintaining the clinical signs presented by the patient. This maintenance begins based on the ABCs of the trauma, checking and clearing the airways and if necessary performing ventilatory support, then performing volume correction using crystalloids and then evaluating the neurological signs presented, giving more attention to animals that have coma. The beginning of treatment should prioritize normalizing the clinical signs that threaten the patient's life, in any case of intoxication (Gwaltney-Brant, 2025; Lee, 2013).

In the case of ingestions that occurred recently, where the service is carried out within 40 minutes after ingestion of the risk products, and the patient has not yet shown clinical signs regarding intoxication, it is possible to induce vomiting in order to eliminate the toxic content before metabolism, in these cases a safe emetic should be used and the procedure should be performed in a hospital or clinical environment assisted by a veterinarian. It is also possible to perform stomach washing, which is performed in the operating room, with the animal in orotracheal intubation and under general anesthesia, this procedure is evasive because it requires general anesthesia, so it is recommended only in severe cases of ingestion of a high volume of risk products (Almeida *et al.*, 2014; Gwaltney-Brant, 2025).

According to Peterson and Talcott (2013) and Plumlee (2013), the first step in the clinical management of these patients consists of the evaluation of cardiorespiratory and

neurological functions, followed by immediate hemodynamic stabilization. Because ethanol is rapidly absorbed gastrointestinally, decontamination procedures are usually of limited benefit, especially when the patient already has neurological signs. Even so, in recent ingestions and in conscious patients, some authors describe emesis induction or gastric lavage as therapeutic possibilities (Almeida *et al.*, 2014; Gwaltney-Brant, 2025). Thrall *et al.*, (1984), when reporting severe poisoning secondary to the ingestion of sourdough, used apomorphine to induce vomiting soon after the patient's admission. However, the authors emphasize that severe neurological depression considerably increases the risk of bronchial aspiration, making emesis induction contraindicated in patients with reduced level of consciousness. In these situations, airway protection and ventilatory support become therapeutic priorities.

One of the main elements used to correct the damage caused by ethanol ingestion is fluid therapy using isotonic crystalloids, which aims to correct dehydration, increase tissue perfusion, accelerate the process of ethanol elimination via the kidneys, regulate acid-base and electrolyte imbalances. Hypoglycemia is also one of the biggest concerns when stabilizing the patient, it must be corrected with glucose solutions, which can be oral or in continuous infusion together with fluid therapy, which must be monitored frequently during the patient's treatment period (Osweiler, 2011).

Suter (1992), in a report involving dogs poisoned by fermented raw pizza dough, observed significant clinical improvement after intravenous administration of balanced electrolyte solutions associated with vitamin supplementation. Similarly, West and Rusbridge (2021) reported complete neurological recovery in less than 24 hours using primarily intravenous fluids and intensive monitoring.

In addition to volume replacement, continuous monitoring of body temperature is essential. Hypothermia is one of the most frequently observed alterations in ethanol intoxication, due to peripheral vasodilation and depression of the central nervous system (Plumb, 2023; Lee, 2013).

Thrall *et al.* (1984) described a significant reduction in body temperature in a dog intoxicated by sourdough, while Keno and Langston (2011) observed persistent hypothermia in a comatose patient intoxicated by vodka. In these cases, active warming measures such as thermal blankets and circulating heating systems are recommended for patient stabilization.

Neurological alterations represent one of the main therapeutic challenges in alcohol intoxication. Central nervous system depression can quickly progress to stupor, coma, and respiratory failure (Plumb, 2023; Lee, 2013). Keno and Langston (2011) reported multiple seizures in a dog with severe vodka intoxication, requiring repeated use of intravenous

diazepam followed by continuous infusion of the drug for seizure control. The authors also highlight that frequent neurological monitoring is indispensable due to the rapid clinical progression observed in these patients.

Respiratory failure is one of the main causes of death associated with alcohol intoxication. Ethanol promotes depression of the respiratory center, leading to hypoventilation and secondary respiratory acidosis (Plumb, 2023; Lee, 2013). Thrall *et al.* (1984) reported the need for prolonged manual ventilation in a sourdough-intoxicated dog due to the patient's inability to maintain adequate spontaneous ventilation. Thus, patients with severe respiratory depression may require oxygen therapy, orotracheal intubation, and mechanical ventilation.

Another frequently reported complication refers to metabolic and glycemic alterations. Puppies and small dogs are more predisposed to hypoglycemia due to lower hepatic glycogen reserves. According to Plumlee (2013), ethanol metabolism promotes inhibition of hepatic gluconeogenesis, favoring a reduction in serum glucose levels. Thus, frequent monitoring of blood glucose is recommended, especially in pediatric patients, and supplementation with intravenous dextrose may be necessary.

Metabolic acidosis and electrolyte disturbances may also be present, particularly in severe or prolonged cases. Keno and Langston (2011) observed persistent respiratory acidosis associated with hypoventilation in a dog with severe intoxication, reinforcing the need for serial evaluation of blood gas analysis and electrolytes. Continuous laboratory monitoring aids in the early identification of complications and directs individualized therapeutic adjustments.

Regarding advanced therapies, hemodialysis represents a promising alternative for severe cases of alcohol intoxication. Ethanol has low molecular weight, reduced protein binding and small volume of distribution, characteristics that favor its extracorporeal removal. Keno and Langston (2011) described the first report of successful use of hemodialysis for the treatment of accidental ethanol poisoning in a dog. In the case reported, the patient had refractory coma, recurrent seizures, and high serum ethanol concentration. After the dialysis session, there was a rapid reduction in serum alcohol levels associated with significant neurological improvement.

## **2.6 Prognosis and Prevention**

Most patients have a favorable prognosis when treatment is instituted early. West and Rusbridge (2021) highlight that the rapid identification of poisoning reduces the occurrence of serious complications and reduces the need for invasive therapies. However, patients who

develop respiratory failure, severe hypotension, severe acidosis, or prolonged coma are at higher risk of mortality.

Prevention continues to be one of the main pillars to reduce the occurrence of alcohol poisoning in dogs. Guidance from owners regarding the safe storage of alcoholic beverages, sourdough and food waste is essential, especially in home environments with puppies and curious animals. Many of the cases described in the literature occurred after accidental access to apparently harmless products, reinforcing the importance of preventive education. When adopting or buying a new animal, this tutor must be warned to leave products that cause a risk to the animal's health out of its reach to avoid accidents. In addition, if any case occurs, consult the veterinarian immediately (Osweiler, 2011; Lee, 2013).

### **3 CASE REPORT**

A female Border Collie dog, 3 months old and weighing 6.5 kg, was taken for emergency consultation at the Araújo Veterinary Hospital (HVA), located in Jaú, São Paulo – Brazil, on 09/14/2025. When starting the consultation, the tutor reported that when she arrived home, she found the patient with a strong odor of wine and dirty, when walking further through her residence the tutor found several broken wine bottles and little liquid scattered on the floor. When checking the surveillance camera, the cub was seen jumping over the sideboard where the wines were stored, knocking over numerous bottles and injuring herself with them, in addition to ingesting much of the wine that was on the floor. In the physical examination performed at a medical appointment on 10/17/25 where it was found: heart rate (HR) of 180 bpm; respiratory rate (*f*) of 44 mpm; rectal temperature (RT) of 38.7°C, capillary filling time (TPC) of 2 seconds; systolic blood pressure (SBP) of 100 mmHg; normocormatic mucous membranes; non-reactive lymph nodes. During inspection, the animal was hydrated and sensitive to abdominal palpation, presenting staggering gait, incoordination and disorientation, strong alcoholic halitosis and cuts caused by shards of glass on its snout and front limb pads.

Due to the clinical signs, a complete blood count (Table 1) and a biochemical blood count (Table 2) were requested. In the erythrogram there is a slight tendency to hemoconcentration, compatible with dehydration or stress, rouleaux + is linked to an increase in proteins or artifacts, platelets are in adequate quantity and normal morphology. Radiographic examination (Figures 1 and 2) was also requested to rule out possible injuries caused during the fall of the bottles. Where it was found that there was no bone injury.

The diagnosis of alcohol intoxication was given by combining the clinical signs presented by the patient and the detailed anamnesis performed in consultation. Although complementary tests have been requested, they do not have the ability to prove a definitive diagnosis. The animal was then sent for hospitalization, for treatment and monitoring.

Table 1. Serum haematological values of the patient

| <b>ERYTHROGRAM</b> | <b>Value</b> | <b>Unit</b>          | <b>References</b> |
|--------------------|--------------|----------------------|-------------------|
| Red blood cells    | 5,18         | x10 <sup>6</sup> /μl | 3,5 – 6,0         |
| Hemoglobin         | 11,30        | g/dL                 | 8,5 – 13          |
| Hematocrit         | 35           | %                    | 26 – 39           |
| VCM                | 67,80        | fl                   | 69 – 83           |
| CHCM               | 32,10        | g/dL                 | 31 - 33           |
| <b>LEUKOGRAM</b>   |              |                      |                   |
| Leukocytes         | 8.700        |                      | 8.500 - 17.300    |
| <b>Totals</b>      | <b>Value</b> | <b>Value</b>         | <b>References</b> |
|                    | Relative (%) | Absolute/μl          | (Absolute/μl)     |
| Myelocytes         | 0            | 0,0                  | 0                 |
| Metamyelocytes     | 0            | 0,0                  | 0                 |
| Rods               | 1            | 87                   | 0 - 200           |
| Segmented          | 64           | 5568                 | 3.900 - 11.800    |
| Lymphocytes        | 31           | 2697,0               | 2.550 - 8.300     |
| Monocytes          | 4            | 348,0                | 100 - 1.750       |
| Eosinophils        | 0            | 0                    | 100 – 885         |
| Basophils          | 0            | 0,0                  | rare              |
| Protein            | 6,5          | g/dL                 | 4,0 – 6,0         |
| Plasma             |              |                      |                   |
| Jaundic Index      | Discreet     | U                    | 2 - 5 U           |
|                    | lipemia      |                      |                   |

Citology: Rouleaux +. Discrete anisocytosis and polychromasia.

Platelets: 527,000/uL (reference: 150,000 a 500,000)

Leukogram: Eosinopenia

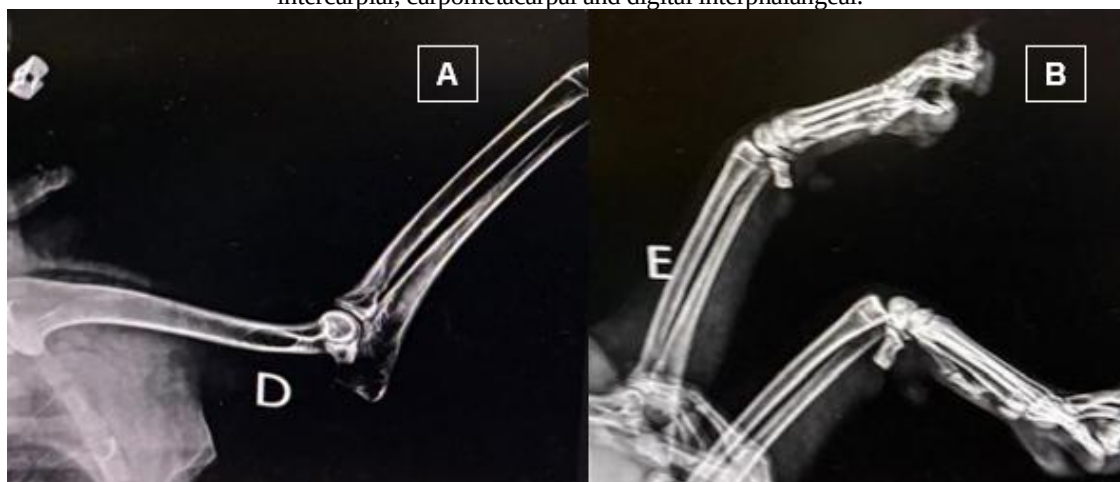
Source: H.V.A. Archive; Weiss, Wadrop, 2010.

Table 2. Patient's biochemical values.

|                   | <b>Value</b> | <b>Unit</b>  | <b>References</b>   |
|-------------------|--------------|--------------|---------------------|
| Albumin           | 3,70         | G/DL         | 2,6 – 3,3           |
| ALT               | 36,2         | U/L          | 21 - 102            |
| Total Cholesterol | 209,9        | md/ dl       | 135 - 270           |
| Creatinine        | 0,4          | mg/dL        | 0,5 - 1,5           |
| Phosphatase       | 244,1        | U/L          | 20 – 156            |
| Alkaline          |              |              |                     |
| Glucose           | 128          | mg/dl        | 65 - 118            |
| Globulin          | 1,59         | g/dl         | 2,7 – 4,4           |
| GT Range          | 9,4          | U/L          | 1,2 – 6,4           |
| Proteins          | 5,29         | g/dl         | 5,4 – 7,10          |
| <b>Totals</b>     |              |              |                     |
| <b>Urea</b>       | <b>53,0</b>  | <b>mg/dL</b> | <b>21,4 - 59,92</b> |

Source: H.V.A. Archive; Kaneko, Harvey, 2008.

Figure 1. (A) Radiographic examination in the mediolateral lateral position of the right thoracic limb encompassing the humerus, radius and ulna and the humerus-radio-ulnar joint. (B) Radiographic examination in the mediolateral position of the left thoracic limb, encompassing carpus, metacarpals, phalanges and joints of the left carpus, intercarpal, carpometacarpal and digital interphalangeal.



Source: HVA imaging sector, 2025.

Figure 2. Radiotherapy examination in the bilateral dorsopalmar position of the thoracic limbs, encompassing radius, ulna, carpals, metacarpals, phalanges, and radiocarpal, intercarpal, and carpometacarpal joints.



Source: HVA imaging sector, 2025.

The patient was monitored for 24 hours, and at hospitalization, venipuncture was performed to perform maintenance fluid therapy using lactated ringer's at the rate of 14 ml per hour for 24 hours. Intravenous ondansetron 0.3 ml, SID, was performed. The wounds caused by the glass were cleaned with saline solution and 2% chlorhexidine degerming. The patient slept most of the time in the hospitalization and as upon arrival, she presented disorientation and incoordination of movements, to prevent the animal from accidentally injuring herself in the stall, an intern was assigned to observe her attentively. After a few minutes, the patient fell

asleep. After waking up, he ate a large amount of dry food and ingested a lot of water, urinated (his urine had a normal smell and color). Fluid therapy followed as prescribed by the doctor. At night, the patient ate again and ingested water, urinated and defecated within normal limits and did not present vomiting, during this period she was already active and no longer had behaviors that represented disorientation and incoordination.

The next day the patient woke up, ate and ingested water in large quantities, urinated and defecated, was released by the hospitalization during the change of her bed, walked and ran, and no longer presented disorientation. This was examined in detail, where it was found that his vital parameters were within normal limits and the critical period of intoxication had passed. With this, the patient was sent for medical discharge.

For home cleaning, only cleaning the wounds using saline solution and the application of local topical antiseptic (1% chlorhexidine digluconate) were recommended. It was recommended that the owner leave all objects that pose a risk to the health of her pets out of their reach, or that she restrict the access space that dogs have in periods when there is no supervision to avoid new accidents.

After medical discharge, no return was scheduled. The patient returned to the hospital after two weeks for the application of the polyvalent vaccine, was examined and was in good health and with healed wounds.

#### **4 DISCUSSION**

Ethanol poisoning in dogs represents a potentially serious clinical condition that is often underdiagnosed in veterinary medicine, especially in young and small patients, in whom lower body mass, hepatic immaturity and reduced metabolic reserves favor the rapid development of neurological and systemic alterations. In the present report, the patient presented classic signs compatible with acute alcohol intoxication, including ataxia, disorientation, staggering gait, motor incoordination, and neurological depression, corroborating the findings described by several authors, who highlight the rapid gastrointestinal absorption of ethanol and its high ability to cross the blood-brain barrier (Peterson; Talcott, 2013; Plumlee, 2013; Van Wuijckhuise; Cremers, 2003; West; Rusbridge, 2021).

The clinical presentation observed in this case has important similarities with other reports described in the veterinary literature. West and Rusbridge (2021), when describing a Labrador Retriever intoxicated after ingesting gin-soaked fruits, reported acute neurological signs such as tetraparesis, altered level of consciousness, hyporeflexia, and hypothermia,

similar to those observed in the patient in this study. Similarly, Van Wuijckhuise and Cremers (2003) described a dog intoxicated after acute ingestion of whiskey, presenting depression of the central nervous system and neurological alterations compatible with acute alcohol intoxication. Keno and Langston (2011), in turn, reported a dog in a comatose state after ingesting vodka, presenting hypothermia, hypotension, miosis and seizures, demonstrating that alcohol intoxication can quickly evolve to critical conditions depending on the dose ingested, speed of absorption and individual susceptibility of the patient.

Although the patient in the present report did not develop seizures, coma, or respiratory failure, her age of only three months represented an important predisposing factor for worsening of the clinical condition. Puppies have lower hepatic glycogen reserve, greater susceptibility to hypoglycemia, and lower capacity to metabolize ethanol, making pediatric patients particularly vulnerable to the toxic effects of the substance (Plumlee, 2013; Spinoso; Górnaiak; Bernardi, 2017). The hepatic metabolism of ethanol promotes an increase in the NADH/NAD<sup>+</sup> ratio, leading to the inhibition of gluconeogenesis, reduction of the conversion of lactate to pyruvate and favoring the accumulation of lactate and ketone bodies. These alterations predispose to metabolic acidosis, neurological depression, and hypoglycemia, complications often associated with severe cases of alcohol intoxication (Plumlee, 2013; Soto-Blanco, 2021).

The absence of significant hypoglycemia in the patient in this report was probably related to the rapid referral to veterinary care and the early institution of intravenous fluid therapy. Even so, the slightly elevated blood glucose observed in laboratory tests can be interpreted as a physiological response to acute stress. This aspect has important clinical relevance, as it demonstrates that pediatric patients may initially present inexpressive laboratory parameters, even in the face of evident neurological signs, reinforcing the need to value anamnesis and clinical examination in the diagnostic approach.

Another relevant aspect refers to the variety of sources of exposure to ethanol in dogs. Although alcoholic beverages are frequently associated with cases of intoxication, the literature shows that fermentative products represent an important cause of clinical occurrence. Thrall *et al.* (1984) reported severe poisoning secondary to ingestion of sourdough containing *Saccharomyces cerevisiae*, with a serum ethanol concentration of 328 mg/dL and the need for manual ventilation due to severe respiratory depression. Similarly, Suter (1992) described an outbreak of poisoning in dogs fed raw pizza dough while still fermenting, showing signs such as stupor, ataxia, hypothermia and fermentative diarrhea. Kammerer *et al.* (2001) also reported fatal poisoning after ingestion of fermented apples, reinforcing the lethal potential of alcohol poisoning in dogs. These reports demonstrate that continuous fermentation in the

gastrointestinal tract represents an important mechanism of endogenous ethanol production, often also associated with gastric distension due to the accumulation of carbon dioxide.

In the present case, however, exposure occurred directly by ingestion of wine in a home environment, a situation less frequently described in the veterinary literature. The association between alcohol intake, secondary trauma caused by glass fragments, and acute neurological signs makes this report particularly relevant for emergency clinical practice, especially considering the patient's age profile. In addition, the case reinforces how domestic accidents can result in potentially serious poisoning in curious and unsupervised puppies.

The diagnosis was established mainly through detailed anamnesis associated with the clinical signs observed. The identification of the accentuated alcohol odor, associated with the visualization of broken bottles and the report of wine ingestion, was essential to quickly direct clinical suspicion. According to West and Rusbridge (2021), anamnesis plays a decisive role in diagnosis, since many owners are unaware of or omit the animal's access to the toxic substance. The authors also point out that several cases of alcohol intoxication end up being initially confused with primary neurological diseases, metabolic encephalopathies or other intoxications.

Although serum ethanol measurement is considered the confirmatory method for diagnosis, its unavailability in the veterinary clinical routine often limits its use. Keno and Langston (2011) observed extremely high serum concentrations in a dog intoxicated with vodka, reaching 610 mg/dL at hospital admission. West and Rusbridge (2021) identified a serum concentration of 3.1 g/L approximately 20 hours after ingestion of gin-soaked fruits. Despite the absence of this analysis in the present report, the clinical findings, the compatible anamnesis, and the favorable outcome after supportive therapy were highly suggestive of acute alcohol intoxication.

The laboratory tests observed in the patient showed slight alterations, mainly a tendency to hemoconcentration, compatible with mild dehydration and stress response. These findings are similar to those described by Suter (1992), who identified hemoconcentration and hyperproteinemia associated with dehydration in dogs poisoned by fermented pizza dough. In addition, metabolic alterations such as hypoglycemia, metabolic acidosis, and lactate elevation are frequently described in the literature, especially in more severe cases (Plumlee, 2013; Keno; Langston, 2011). The absence of severe metabolic alterations in this case probably reflected the lower toxic load ingested and the rapid initiation of supportive treatment.

The rapid clinical recovery observed after intravenous fluids reinforces the central role of supportive treatment in alcohol intoxication. Fluid therapy with isotonic crystalloids helps to

correct hypovolemia, improve tissue perfusion, stabilize hemodynamics, and increase renal ethanol elimination. West and Rusbridge (2021) reported complete neurological recovery in less than 24 hours using only intensive monitoring and intravenous fluid therapy. Similarly, in the present report, the patient presented complete resolution of neurological signs within the hospitalization period.

In severe cases, however, more aggressive therapeutic measures may be necessary. Keno and Langston (2011) demonstrated the successful use of hemodialysis in a dog with severe intoxication and coma refractory to conventional treatment. The authors observed a rapid reduction in serum ethanol concentrations and a significant improvement in neurological status during the dialysis session. Although hemodialysis is not yet widely available in veterinary medicine, the report highlights its therapeutic potential in severe life-threatening poisonings.

Another important point observed in this case refers to the absence of secondary respiratory complications, such as bronchial aspiration. The literature describes that depression of the protective reflexes of the airways is an important risk factor in patients intoxicated by ethanol, especially in the presence of vomiting and reduced level of consciousness. Thrall *et al.* (1984) highlighted that the main cause of death in these patients is related to severe respiratory depression, while Keno and Langston (2011) reinforce the need for continuous ventilatory monitoring in comatose patients.

The favorable clinical evolution observed in this report was probably related to the rapid identification of poisoning and early referral to veterinary care. According to West and Rusbridge (2021), the immediate recognition of the toxic agent significantly reduces the need for invasive diagnostic tests and enables the rapid institution of supportive therapy. In addition, patients treated early are at lower risk of developing severe acidosis, persistent hypoglycemia, and respiratory failure.

From a scientific point of view, this case contributes to the veterinary literature by broadening the discussion about alcohol intoxication in pediatric patients, a condition that is still little described in young dogs. Most of the available reports involve adult animals or poisoning secondary to food fermentation, while the present study describes direct exposure to wine in a home environment associated with complete recovery after intensive support. Thus, the case reinforces the need to include ethanol poisoning among the differential diagnoses of acute neurological alterations in puppies, especially when there is a history of access to alcoholic beverages or fermentative residues.

From a preventive point of view, the case reinforces the importance of educating tutors about the risks of exposing domestic animals to alcoholic beverages and fermentative products.

Many cases occur in a home environment due to improper storage of beverages, raw pasta, or fermented food waste. West and Rusbridge (2021) point out that owners often do not immediately associate clinical signs with alcohol intoxication, making the initial diagnosis difficult. Thus, educational campaigns and preventive veterinary guidance play a fundamental role in reducing the occurrence of these accidents.

Finally, the present report shows that acute alcohol poisoning in dogs, although uncommon, represents a potentially serious clinical emergency, especially in young patients. Detailed anamnesis, early recognition of clinical signs, intensive monitoring and rapid institution of supportive therapy remain fundamental pillars for a favorable prognosis. In addition, the case contributes to the veterinary literature by demonstrating that apparently simple domestic exposures can result in important neurological manifestations, emphasizing the need for tutors' awareness and adequate clinical preparation for the management of these patients.

## **5 FINAL CONSIDERATIONS**

This report describes a case of acute ethanol poisoning in a puppy dog after accidental ingestion of wine, characterized by neurological manifestations compatible with central nervous system depression and diagnosis established through anamnesis associated with clinical findings. The rapid gastrointestinal absorption of ethanol and its systemic effects, including neurological depression, hypothermia, hypoglycemia, and metabolic alterations, reinforce the relevance of understanding the pathophysiology of this intoxication, especially in pediatric patients, who have greater susceptibility due to hepatic immaturity, lower energy reserve, and reduced metabolization capacity.

The favorable clinical evolution observed after supportive therapy shows that early recognition and immediate intervention are determinant for a positive prognosis, even in the face of potentially severe neurological signs. In addition, the case expands the veterinary literature by documenting a common, but often underestimated, household source of ethanol exposure, highlighting the need to include this intoxication among the differential diagnoses of acute neurological changes in young dogs. The present report also contributes to clinical practice by emphasizing the importance of cardiorespiratory stabilization, metabolic monitoring, and intensive support in intoxicated pediatric patients, providing relevant information for the recognition and management of accidental ethanol exposures in small animal veterinary medicine.

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