Case Report

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Successful treatment of severe salt intoxication in a dog

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Abstract

Objective: To report successful treatment of severe salt intoxication and hypernatremia in a dog.
Case summary: A 5-year-old intact female Doberman Pinscher was admitted to the intensive care unit with a history of seizures and coma. The owner had administered approximately 100 g of cooking salt to induce vomiting following ingestion of a nontoxic dose (10 g) of chocolate. Upon admission, the dog was comatose with intermittent seizures and vomiting. Diagnostic tests confirmed salt intoxication (Na: 200 mEq/L, Cl: 180 mEq/L) and metabolic acidosis (pH: 7.18; pCO_2: 39 mmHg; HCO_3: 14.3 mmol/L). Immediate treatment included intravenous fluid therapy, an anticonvulsant, antiemetic, diuretic, low molecular weight heparin, and supplemental oxygen. A fluid therapy protocol was initiated to decrease serum sodium concentration by approximately 2 mEq/L/hr. After 24 hours of intensive care, the patient regained consciousness and volume and acid-base abnormalities improved. The patient developed a variety of abnormal clinical signs as a result of the severe hypernatremia. After 5 days of treatment, the serum sodium concentration returned to the established reference range. The patient recovered completely in 10 days.
New information provided: Severe hypernatremia due to salt ingestion is a rare condition in dogs. All dogs in previous case reports of salt intoxication have died. This case report is the first to report survival of a dog with severe salt intoxication.

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Case Summary

A 5-year-old 30 kg intact female Doberman Pinscher was admitted to the intensive care unit for treatment of acute onset of seizures and the development of a coma. One hour before presentation, the owners administered 5–6 tablespoons (weighing approximately 100 g) of cooking salt to the dog to induce vomiting following accidental ingestion of 10 g of chocolate (nontoxic dose). Initially, the patient began to exhibit signs of vomiting and diarrhea. Clinical signs rapidly worsened and included ataxia followed by seizures.

On admission, the patient was moribund and was actively seizuring. Diazepam[a] (2 mg/kg per rectum) was administered and resulted in a reduction of seizure activity and facilitated the placement of an intravenous (IV) catheter. Shortly after admission, seizures were controlled and a complete physical examination was performed. The dog had no voluntary movements and was not responsive to painful stimuli. In addition, the dog had mydriasis, absent pupillary light reflexes, and absent menace responses in both eyes. The cardiovascular system examination revealed moist, pink mucous membranes, normal capillary refill time (CRT), sinus tachycardia (200 beats/min [b.p.m.]), and normal femoral pulse quality. The respiratory rate was tachypneic (54 breaths/min) but no abnormal breathing pattern was noted. The dog continued to vomit. The patient was not clinically dehydrated at this time.

Based on the history of salt ingestion and the clinical signs, salt intoxication was suspected. Pre-fluid therapy blood electrolytes and blood gas analysis revealed severe hypernatremia (serum sodium, 200 mEq/L), severe hyperchloremia (serum chloride, 180 mEq/L) and severe metabolic acidosis (pH 7.18). Venous blood gas measurements are presented in Table 1. This analysis confirmed the suspicion of salt intoxication.

The initial treatment goal was to dilute the sodium concentration, therefore, 5% Dextrose[b] in water (D5W) was chosen for IV administration. This solution is

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Table 1: Changes in electrolytes, blood gas and renal analyses and arterial blood pressure (BP) during the dog's hospitalization

Day	D0	D1	D2	D3	D4	D5	D6	D7	D8	D9
Electrolytes (mEq/L)										
Na	200	178	181	166 160	152		144		143	
K	4.4	3.04	3.34	3.42 3.60	3.92		3.10		4.25	
Cl	180	146	119	121 124	103		110		109	
Blood gas										
pH	7.18	7.33	7.38	7.40			7.43			
pCO_2 (mmHg)	39	30	35	39			36			
HCO_3 (mEq/L)	14.3	15.2	20.4	23.3			23.0			
Urea mg/dL (mmol/L)		24.6 (8.8)	27.44 (9.8)	62.4 (22.3)	103.6 (37.0)	103.0 (36.8)	84.5 (30.2)	67.2 (24.0)		61.6 (22.0)
Creatinine mg/dL (μmol/L)		1.2 (107)	2.0 (181)	4.1 (378)	3.6 (330)	4.7 (427)	4.6 (422)	4.4 (397)	4.4 (404.0)	4.1 (374)
Systolic BP (mmHg)	120	120	100	140	130	100	120	110		

equivalent to pure water and thus allows a relatively rapid decrease in serum sodium. The fluid rate (6 mL/kg/hr) was calculated to reduce serum sodium concentration by <2 mEq/L/hr (Table 2).

Vomiting was controlled with a continuous infusion (CRI) of metopimazine[c] (1 mg/kg/day). Metopimazine antagonizes dopamine and acts on the chemoreceptor trigger zone. Supplemental oxygen was administered via a nasal catheter (150 mL/kg/min). Calcic nadroparine[d] (low molecular weight heparin: 57 IU/kg/day SC) was administered to mitigate the perceived thrombotic risk caused by cell damage induced by the hypertonic state. An experimental study demonstrated that hypernatremia produces changes in hemostatic function consistent with a hypercoaguable state in human volunteers,[1] and a case report describing salt intoxication in a human infant reported the presence of a coagulopathy and venous sinus thrombosis.[2] In both cases, the exact mechanism of thrombosis was unclear.

Initially, the dog's clinical improvement could not be readily explained by significant decreases of serum sodium (Table 1). The dog appeared mentally brighter after 3 hours of intensive care and fluid therapy. The pupillary light reflex returned to normal after 3 hours and the menace response reappeared in 5 hours. The heart rate decreased slowly. The cardiovascular and pulmonary systems evaluations remained normal during initial treatment phase. The serum sodium concentration decreased slowly during the first days (Table 1) to reach the established normal reference range within 5 days. Electrolyte concentrations were monitored frequently and fluid therapy was adjusted as necessary. During the stages of treatment when hypernatremia was severe, D5W was used as the primary fluid. When hypernatremia was present in conjunction with dehydration, D5W was used to decrease sodium, and other fluid types (such as 0.45% saline with 2.5% glucose[e] or LRS[f]) were utilized to correct dehydration. Maintenance fluid therapy (LRS[f]) was continued to correct dehydration, after normalization of serum sodium.

Despite neurological improvement, the dog developed other abnormal clinical signs. Twenty-four hours following admission, an irregular heart rhythm was detected on cardiac auscultation. The electrocardiogram (EKG) revealed a sustained unifocal ventricular tachycardia at 250 b.p.m. Despite the elevated heart rate, ventricular tachycardia was not treated as it did not result in any hemodynamic changes. After 6 hours, the EKG revealed a brief period of paroxysmal ventricular tachycardia followed by a rhythm consisting of supraventricular complexes alternating with very frequent isolated polymorphic complexes of ventricular origin. The R to R interval of the supraventricular rhythm was very irregular with no visible P waves, so atrial fibrillation was suspected. Echocardiographic evaluation was performed and showed increased left ventricular diastolic diameter, systolic left ventricular dimensions in the upper range of normal, marked left atrial enlargement, slightly decreased fractional shortening at 19% (normal should be greater than 20%) and

Table 2: Numerical example of fluid therapy to correct acute hypernatremia for a 30 kg dog with a serum sodium concentration of 200 mEq/L

After the administration of 1 L of D5W, serum sodium decreases by 10.5 mEq/L: $[0–200]/[(30 \times 0.6)+1] = -10.5$ mEq/L.
When the serum sodium rise is acute, its rate of decrease should be 2 mEq/L/hr. Quantity of D5W needed in 1 hour = 190 mL (2 mEq/L/hr ÷ 10.5 mEq/L of sodium/L of D5W).
Thus, 190 mL of D5W should be used every hour, which is equivalent to approximately 6 mL/kg/hr.

increased sphericity (measured sphericity index: 1.3 [normal should be greater than 1.65]). According to the scoring system proposed by the European Society of Veterinary Cardiology Task Force (ESVC),[3] preclinical dilated cardiomyopathy was suspected.

Pimobendan[g] (0.25 mg/kg PO q 12 h) and benazepril[h] (0.25 mg/kg PO q 24 h) were administered. Twenty-four hours after admission, sequential pulmonary auscultation detected the development of abnormal respiratory sounds. The presence of pulmonary edema was clinically suspected. No thoracic radiographs were taken at this time because the dog was too unstable. Pulmonary edema could have been cardiogenic in origin, secondary to fluid overload associated to the presence of cardiomyopathy in this dog[4–6] or noncardiogenic due to cell damage related to the severe hypernatremic state increasing pulmonary capillary leak. Furosemide[i] (2 mg/kg IV q 4 h for 3 days) was added to treat the suspected pulmonary edema. An additional benefit of furosemide[i] administration was the potential increase in renal sodium output.

On the first day of hospitalization, salt irritation of the digestive tract led to vomiting, regurgitation and diarrhea. Despite administration of an antiemetic (CRI of metopimazine[c]) and gastromucosal protectants (sodium alginate and sodium bicarbonates[j] [500 mg/kg PO q 8 h], kaolin and pectine[k] [2 mg/kg PO q 8 h]) digestive signs responded slowly to therapy. Oral ulcers were also present and may have been caused by salt irritation and intracellular fluid dehydration due to hyperosmolarity. Improvement was observed after the local application of an oral gel (choline salicylate and cetalkonium chloride[l] [applied on lesions q 8 h]). Diarrhea and melena suggested the presence of mucosal ulcers elsewhere in the digestive system, however endoscopy was not performed. Such clinical signs can be related to desquamation of surface epithelium of intestinal villi,[6] severe gastritis[7] or severe ulceration of digestive system.[8]

On the third day, acute renal insufficiency was diagnosed (blood urea nitrogen [BUN]: 22 mmol/L [61.6 mg/dL], reference range: 2–7 mmol/L [15–32 mg/dL]; creatinine: 378 μmol/L [4.1 mg/dL], reference range: 0–200 μmol/L [1–2 mg/dL], specific gravity: 1.010). We concluded that renal parenchymal lesions were present because the elevation of BUN and creatinine persisted until release of the patient, even after correction of dehydration and hypovolemia. A dietary modification (prescription diet k/d[m]) was added to the fluid therapy in an attempt to further preserve renal function. Placement of a urinary catheter and sterile delivery set allowed a closed monitoring of diuresis. Urine production was consistent with hydration status and fluid administration. Renal parameters and arterial blood pressure were monitored during hospitalization (Table 1).

The patient was discharged after 10 days with complete resolution of clinical signs and abnormal electrolyte values attributable to salt intoxication. Pimobendan[g] and benazepril[h] were continued to treat the dilated cardiomyopathy. A follow-up cardiac evaluation was performed 2 days later. The patient was asymptomatic at this evaluation. Echocardiographic examination revealed no improvement in the cardiac function, and digoxin[n] (5 μg/kg q 12 h) was added to the previous treatments because atrial fibrillation was still present. Follow-up cardiac examinations 2 and 7 months later revealed stable cardiac function.

Discussion

Hypernatremia is a common electrolyte abnormality caused by net solute gain (salt intoxication), sodium-free water loss (diabetes insipidus or heat stroke), or hypotonic fluid loss (e.g., vomiting, diarrhea, osmotic diuresis). Severe hypernatremia is an unusual and life-threatening condition. Hypernatremia due to free-water or hypotonic fluid losses are of acute or chronic presentation and dehydration and hypovolemia are consistently present.[9] On the contrary, when hypernatremia is due to a gain of sodium, the clinical onset is acute. There is no clinical dehydration and animals are normo- or hypervolemic.[4,9–11] The increase in serum sodium creates a hypertonic state in the extracellular fluid. Initially, water shifts from the interstitium to the vascular space and then, from the intracellular space to the extracellular space to maintain equilibrium. As more water leaves the intracellular space and enters the extracellular fluid space, a state of hypervolemia develops.[4] The main goal in the treatment of hypernatremia due to a gain of sodium is a rapid correction of hypernatremia. The speed at which hypernatremia is corrected should parallel the speed at which the hypernatremia developed.[5,10,12]

Severe hypernatremia due to salt ingestion in dogs is a very uncommon toxicosis. Very few case reports are described in veterinary literature.[4,11] The animal antipoison center of Lyon (CNITV) documented 260 cases of salt toxicosis in dogs during the time period of 1995–2006. The majority of clinical signs recorded in these cases were digestive and neurological disorders. Patients with neurological signs usually died. To the authors' knowledge, no previous case of deliberate administration of pure table salt to a veterinary patient has been reported in the literature. This case followed the typical clinical presentation of salt intoxication. Signs of toxicosis appear after ingestion of 2–3 g of salt per kg of body weight. Ingestion of 4 g/kg of salt is

considered lethal.[4] Clinical signs typically develop when serum sodium concentration reaches or exceeds 180 mEq/L. In this case, the ingested dose was 3.5 g/kg and the initial serum sodium level was 200 mEq/L. The onset of clinical signs in this dog occurred approximately 60 minutes after salt ingestion.

Salt is an acute mucosal and gastric irritant and thus, the initial signs of intoxication are associated with the gastrointestinal tract.[4,11] The neurological signs develop later. The severity of the neurological symptoms is related to both the degree and, more importantly, the rate of increase of serum sodium concentration.[13] In salt intoxication, rapid sodium concentration changes in the extracellular compartment act to draw water from the intracellular space by osmosis, leading to a decrease in brain volume. Some authors have reported that this decrease in brain volume induces rupture of the cerebral capillaries, resulting in focal intracerebral and subarachnoid hemorrhage and neurologic dysfunction that may be irreversible.[13] When hypernatremia develops slowly or has been present for several days, the brain is able to produce intracellular solutes called idiogenic osmoles. Idiogenic osmoles act to protect the brain from hyperosmolar fluid shifts and the damage that results.[4,13] However, production of protective levels of idiogenic osmoles typically requires at least 4–7 days.[4] This physiological adaptation has 2 major consequences. First, patients with chronic hypernatremia may be relatively asymptomatic. Second, overly rapid correction of chronic hypernatremia may lead to cerebral edema.[13]

Severe hypernatremia due to a gain of sodium is extremely difficult to manage. Survival after salt intoxication in a dog has not been previously described in case reports in the veterinary literature. The goal of the treatment is to address the main clinical signs upon admission, then decrease serum sodium concentration. As a rule of thumb, a 0.5 mEq/L/hr rate of decrease in sodium concentration is considered safe.[4,9] However, this rate can be modified to parallel the speed with which the hypernatremia developed.[9] In cases of acute ingestion, hypernatremia can be corrected safely at a rate of 2 mEq/L/hr.[11] If correction of hypernatremia is too slow, sustained hypernatremia can result in ongoing neurological damage.[11]

IV fluid administration is the primary component of therapy to treat hypernatremia caused by salt ingestion. The choice of fluid and rate of administration should be made based on the cause of hypernatremia. In salt intoxication, the main goal is to administer free water orally if the patient can drink, or IV using D5W in more severe cases.[13] In addition, treatment to speed sodium elimination such as administration of a loop diuretic can be helpful. Potential complications should be anticipated and mitigated. In cases of severe hypernatremia, dilution of the intravascular sodium concentration can occur in 2 ways. Normal physiologic compensation allows water shifting to the intravascular compartment.[5,14] Hypotonic fluid therapy can also be used to dilute the intravascular sodium concentration. The use of D5W is equivalent to administration of pure water. The following formulae represent guidelines to calculate fluid therapy requirements.[9,15] To determine the amount of water required to correct sodium to the desired level in hypernatremia due to water loss or hypotonic fluid loss, the following formula can be utilized:

$$\text{Free water deficit} = 0.6 \times \text{body weight} \times (Na^{+}_{present}/Na^{+}_{desired} - 1)^{9}$$

A second formula determines the effect of 1 L of a solution on the decrease in the rate of sodium: decrease in sodium concentration per L of fluid (mEq/L per L) = $[Na^{+}_{solute} - Na^{+}_{present}]/[(\text{body weight} \times 0.6) + 1]$.[9]

This formula can be used to determine the rate of fluid therapy required to decrease the serum sodium concentration by approximately 2 mEq/L/hr (Table 2).

Fluid overload was one of the principal complications in this case. Loop diuretics are routinely used to decrease free water and increase renal sodium output. In patients with cardiac or renal dysfunction, loop diuretics can also minimize the risk of pulmonary edema.[4,10,13] The precise effect of diuretics on the decrease in sodium concentration cannot be accurately predicted, but in patients with acute hypernatremia, the rate at which the sodium concentration decreases is less a problem than in patients with chronic hypernatremia.[9] Monitoring cardiovascular, respiratory and electrolyte parameters is an important part of hypernatremia management. Fluid overload should be anticipated and avoided if possible. Measurement of central venous pressure is the best way to monitor intravascular volume status but was not used in this case because of the potential thrombotic risk. Electrolytes should be measured every 2 or 3 hours until they are within the reference interval.

The origin of the cardiac abnormalities in this patient remains uncertain. The echocardiographic changes (according to the ESVC Task Force scoring system) suggest the presence of preclinical dilated cardiomyopathy. However, given the paucity of literature regarding cardiac consequences of salt intoxication, it is difficult to make conclusions about the mechanisms that led to myocardial dysfunction in this case. The clinical suspicion of pulmonary edema during fluid administration in this patient could support the hypothesis of cardiac disease. Indeed, with underlying cardiac dysfunction, pulmonary edema is one of the first clinical signs to appear as a consequence of fluid overload.[5]

No data is available to determine if the renal insufficiency in this patient was pre-existing. Salt intoxication has been associated with acute renal tubular necrosis in other patients.[6,11] In this case, no renal biopsy was performed, but we can speculate that acute renal necrosis might explain acute renal insufficiency.

In dogs, the prognosis in cases of salt intoxication depends on the quantity of ingested salt, serum sodium concentration, and the severity of neurological signs upon admission, the speed of serum sodium rise, the age of the patient, and associated concurrent illness. The prognosis of this patient was guarded upon admission because of the high quantity (3.5 mg/kg) of ingested salt, high serum sodium, severe neurological signs, rapidity of increase in sodium serum and pre-existing condition(s). This case report is, to the authors' knowledge the first to report survival of a dog with salt intoxication. To the authors' knowledge, all dogs from previous case reports with salt intoxication and associated neurological involvement died. Salt intoxication is a true emergency condition requiring rapid patient assessment and aggressive yet precise fluid therapy. A close and continual reassessment of physiologic parameters allows the emergency clinician to anticipate and respond to complications due to volume overload and intracellular dehydration and damage.

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Footnotes

[a] Valium Roche, Roche, Neuilly-sur-Seine, France.
[b] Glucose 5% isotonique, Aguettant, Laboratoire Aguettant, Lyon, France.
[c] Vogalène, Laboratoires Schwarz Pharma, Boulogne-Billancourt, France.
[d] Fraxiparine, Sanofi Winthrop, Gentilly, France.
[e] Glucose 2.5%-NaCl 0.45%, Aguettant, Laboratoire Aguettant.
[f] Ringer Lactate, Aguettant, Laboratoire Aguettant.
[g] Vetmedin, Boehringer Ingelheim France, Reims, France.
[h] Fortekor, Novartis santé animale SAS, Reuil-Malmaison, France.
[i] Dimazon, Intervet SA, Beaucouze, France.
[j] Gavison, Reckitt Benckiser Healthcare, Hull, UK.
[k] Kaopectate, Pharmacia, Guyancourt, France.
[l] Pansoral, Pierre Fabre Oral Care, Boulogne Billancourt, France.
[m] Hill's Pet nutrition SNS, Sophia Antipolis, France.
[n] Digoxine Nativelle, Procter and Gamble Pharmaceuticals France, Neuilly-sur-Seine, France.

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