



Acute Salt Toxicity Caused by Whey Ingestion in Mixed-breed 6-months-old Puppy: A case study

Erdem GÜLERSOY^{1*}, Süleyman Serhat İYİGÜN²

¹Department of Internal Medicine, Faculty of Veterinary Medicine, Harran University, Şanlıurfa, Turkey.

²Department of Internal Medicine, Faculty of Veterinary Medicine, Selçuk University, Konya, Turkey.

*Corresponding Author, Erdem GÜLERSOY, +905333344042; E-Mail: egulersoy@harran.edu.tr

ABSTRACT

This case report aims to emphasize the acute management of a dog with suspected salt intoxication, which consists of a treatment protocol with hematological and Modified Glasgow Coma Scale (MGCS) changes and electrocardiographic (ECG) evaluation. A 6-month-old male mixed-breed dog was admitted to the emergency clinic with complaints of weakness, hypersalivation, vomiting, diarrhea and gait disturbance. It was learned from the owner that the dog was fed on whey for the first time on the morning of the admission day. Salt intoxication was suspected based on history and clinical symptoms. The diagnosis was confirmed by laboratory analysis (pH: 7.185, Na: 178 mmol/L, Cl: 155 mmol/L, K: 8.3 mmol/L, HCO₃: 16.1 mmol/L, BE: -8.8 mmol/L, BUN: 105.7 mg/dl, creatinine: 5.4 mg/dl, AST: 513 U/L, amylase: 2324 U/L, LDH: 794 U/L, calcium: 11.8 mg/dl, phosphorus: 13.9 mg/dl, cholesterol: 515 mg/dl, magnesium: 3.6 mg/dl, GGT: 62 U/L CPK: 450 U/L and CK-MB: 324.5 U/L), ECG (sinus tachycardia (180 bpm) and mild ST depression) and MGCS evaluation (pre-treatment score; 7, grave). A treatment protocol including fluid therapy, antiemetic, diuretic, anticonvulsant, beta-blocker to prevent sodium retention and oxygen therapy was administered to the dog during the hospitalization period. It was observed that the clinical findings and MGCS score improved and some blood parameters returned to their reference range on Day 2. Since the dog died on Day 3 after the hospitalization period, which was terminated at the owner's request, it was concluded that an adequate hospitalization period is important. The presented treatment protocol with MGCS evaluation in salt toxicity was successful enough only in acute management.

Keywords: Dog, fluid therapy, hypernatremia, modified Glasgow coma scale, salt toxicity.

Case Study:

DOI: <https://dx.doi.org/10.21608/javs.2021.77045.1081>

Received :21 May, 2021.

Accepted :16 June, 2021.

Published in July, 2021.

This is an open access article under the term of the Creative Commons Attribution 4.0 (CC-BY) International License . To view a copy of this license, visit:

<http://creativecommons.org/licenses/by/4.0/>

INTRODUCTION

The key factors for the development of hypertension, renal failure and coronary heart disease are considered to be medical conditions such as severe water loss and intake of iatrogenic sodium (Arambewela *et al.*, 2016) or excessive consumption of processed food which meets more than 75% of daily salt need (Titze and Ritz 2009). While the recommended daily sodium consumption in humans is 1500-2400 mg/day (Kim *et al.*, 2012), the sodium requirement in adult dogs is reported as 5 mg/kg/day or 0.2 g / 1000 kcal (metabolisable energy) (Morris *et al.*, 1976, Smith *et al.*, 2016).

The clinical signs of salt toxicity are lethargy, depression, dehydration, vomiting and diarrhea (Angelos *et al.*, 1999), followed by neurologic findings such as muscle stiffness, opisthotonus, tremors, convulsions, seizures, and loss of consciousness (Hardy, 1989). Laboratory findings of salt toxicity are metabolic acidosis, hypernatremia, hypoglycemia, and serum biochemistry abnormalities such as azotemia, increasing some liver enzyme levels due to tissue hypoperfusion resulting from dehydration (Kazanji *et al.*, 2015, Arambewela *et al.*, 2016). The most prominent findings on ECG are QT prolongation, PR shortening, ST depression, decrease in P and QRS amplitudes and sinus tachycardia (Fisher *et al.*, 2006).

J. Appl. Vet. Sci., 6(3) : 31 – 37.

Clinical findings of salt toxicity are associated with pathological changes in the brain and the level of consciousness depends on the severity of the hypernatremia (Braun *et al.*, 2015). In cases that affect the level of consciousness such as hypoglycemia, hypotension, hypernatremia and trauma (Bowins, 2018), the Modified Glasgow Coma Scale (MGCS) is useful in observing the progression of neurological disorders and the effects of therapeutic interventions (Platt *et al.*, 2001).

In this case report, based on historical information of the dog such as iatrogenic whey consumption (0.7-1.7 M NaCl; maximum 99.34 g per 1 liter) and clinical signs, salt toxicity was suspected and confirmed by laboratory analyzes. Acute management and changes in hematochemical parameters, ECG and MCGS score findings during the hospitalization period are presented.

Case Description:

A 6-month-old male mixed-breed dog weighing 20 kg was brought to Selcuk University Faculty of Veterinary Medicine Animal Hospital Emergency Clinic with complaints of sudden weakness, respiratory distress, hypersalivation, vomiting, diarrhea and gait disturbance (Fig. 1). It was learned from the owner that the dog had two seizures lasting less than 1 minute at 15-minute intervals at home and one seizure on the road before admitting to the hospital. The dog was vaccinated, had no history of the disease, was fed commercial dry dog food, and was fed on whey for the first time as a supplement on the morning of the admission day (Day 0, approximately 3 hours before admission). Clinical and laboratory tests were performed for further diagnosis.

Physical examinations including palpation of lymph nodes, measurement of heart and respiratory rate and temperature, auscultation of lung and heart, evaluation of color of mucous membranes, capillary refill time, degree of dehydration, ECG (Biocare ECG 300G, China) and MGCS assessment to determine the level of consciousness were performed. Laboratory analysis included blood gases (ABL80 Flex, Denmark), complete blood count (CBC) (MS4 Autoanalyzer, France) and serum biochemistry (BT3000 Autoanalyzer, Italy) measurements.



Figure 1: Hyper salivation (black arrow) and lethargic status of the dog with labored respiration

RESULTS

During a physical examination, a considerable loss of skin turgor and severe dehydration (~10%) with enophthalmos, pale mucous membranes, prolonged capillary refill time (> 4 sec), high rectal temperature (39.8 °C), weak femoral pulse, mildly depressed respiratory sounds and normal palpable lymph nodes were detected. ECG measurement revealed sinus tachycardia (180 bpm) and mild ST depression (Fig 2). Blood pressure was measured as 220 mmHg (systolic) and 130 mmHg (diastolic) and urine specific gravity as 1.040 (URIT-31, China). The MGCS score at the bedside was determined to be seven and evaluated as severe (Table 1).

In blood gases, metabolic acidosis (pH: 7.185), hypernatremia (Na: 178 mmol / L), hyperchloremia (Cl: 155 mmol / L), hyperkalaemia (K: 8.3 mmol / L), hyperlactaemia (lactate: 6.8 mmol / L), low bicarbonate

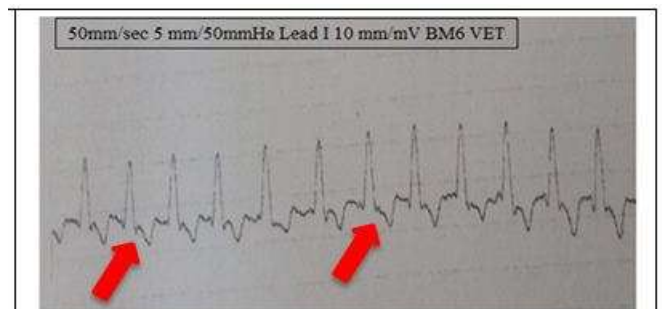


Figure 2: Sinus tachycardia and mild ST depression (red arrows)

level (HCO_3^- : 16.1 mmol / L), base deficit (BE: -8.8 mmol / L), low partial pressure of carbon dioxide (pCO_2 : 31.9 mmHg) and high partial pressure of oxygen (pO_2 : 59.7 mmHg) were determined. In CBC analysis, parameters except secondary polycythemia (RBC: 10.41 M / mm^3 ; Hct: 50.5%; Hb: 15.1 g / dl) were within reference range.

Elevated levels of BUN (105.7 mg / dl), creatinine (5.4 mg / dl), AST (513 U / L), amylase (2324 U / L), LDH (794 U / L), calcium (11.8 mg / dl), phosphorus (13.9 mg / dl), cholesterol (515 mg / dl), magnesium (3.6 mg / dl), GGT (62 U / L), CPK (450 U / L) and CK-MB (324.5 U / L) levels were observed in serum biochemistry. The diagnosis of acute salt toxicity due to whey ingestion was confirmed on the basis of history, physical and laboratory examinations. Blood analysis results were presented in Table 2.

Table 1. The change of Modified Glasgow Coma Scale score at first admission and before discharge

Modified Glasgow Coma Scale (MGCS) (Platt <i>et al.</i> , 2001)		Score	First admission (Day 0)	Before discharge (Day 2)
Motor Activity	Normal gait, normal spinal reflexes	6		
	Hemiparesis, tetraparesis, or decerebrate rigidity	5		
	Recumbent, intermittent extensor rigidity	4		
	Recumbent, constant extensor rigidity	3		3
	Recumbent, constant extensor rigidity with opisthotonus	2	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		4
	Pinpoint pupils with reduced to absent oculocephalic reflexes	3		
	Unilateral, unresponsive mydriasis with reduced to absent oculocephalic reflexes	2	2	
	Bilateral, unresponsive mydriasis with reduced to absent oculocephalic reflexes	1		
Level of Consciousness	Occasional periods of alertness and responsive to the environment	6		
	Depression or delirium, capable of responding but the response may be inappropriate	5		5
	Semicomatose, responsive to visual stimuli	4		
	Semicomatose, responsive to auditory stimuli	3	3	
	Semicomatose, responsive only to repeated noxious stimuli	2		
	Comatose, unresponsive to repeated noxious stimuli	1		
MCGS Score	3-8: Grave, 9-14: Guarded, 15-18: Good		7 (Grave)	12 (Guarded)

Table 2. Blood gases, hemogram and serum biochemistry results at the first admission

Venous Blood Gases	Values	Reference*	Hemogram	Values	Reference*
pH	7,185	7.31-7.42	WBC	15,75	6-17 m/mm ³
pCO ₂	31,9	29-42 mmHg	Lym	4,47	0.6-5.1 m/mm ³
pO ₂	59,7	85-95 mmHg	Mon	1,2	0.1-1.7 m/mm ³
K	8,3	3.6-5.5 mmol/L	Gra	9,05	3-13.6 m/mm ³
Na	178	139-154 mmol/L	RBC	10,41	5.5-8.5 M/mm ³
Ca	0,95	2.2-3 mmol/L	MCV	67	58-73 fl
Cl	155	102-120 mmol/L	MCH	22,6	19.5-24.5 pg
Lactate	6,8	0-2 mmol/L	MCHC	32	28-40 g/dL
Base excess	-8,8	-4-4 mmol/L	Hct	50,5	35-55 %
HCO ₃	16,1	17-24 mmol/L	Hb	15,1	10-18 g/dL
Biochemistry	Values	Reference*	Biochemistry	Values	Reference*
BUN	105,7	4.70-7.30 mg/dl	Phosphorus	13,9	1.8-6.4 mg/dl
Creatinine	5,4	0.8-1.8 mg/dl	Albumin	2,8	2.1-3.9 g/dl
AST	513	10-80 U/L	Cholesterol	515	90-205 mg/dl
ALT	23	10-80 U/L	Calcium	11,8	8-10.7 mg/dl
ALP	76	10-80 U/L	Triglycerides	96	10-114 mg/dl
Amylase	2324	500-1800 U/L	Magnesium	3,6	1.5-3.5 mg/dl
Glucose	131	70-150 mg/dl	GGT	62	1-10 U/L
LDH	794	75-490 U/L	Total Protein	7,2	5.4-7.8 g/dl
Total Bilirubin	0,4	0.1-0.6 mg/dl	CPK	450	50-450 U/L
Direct Bilirubin	0,2	0-0.3 mg/dl	CK-MB	324,5	78-134,5 ng/ml

pH: Power of hydrogen, pCO₂: partial pressure of carbon dioxide, pO₂: partial pressure of oxygen, K: potassium, Na: sodium, Ca: calcium, Cl: chlorine, HCO₃: bicarbonate, WBC: leukocyte, Lym: lymphocyte, Mon: monocyte, Gra: granulocyte, RBC: red blood cells, MCV: mean corpuscular volume, MCH: mean corpuscular haemoglobin, MCHC: mean corpuscular haemoglobin concentration, Hct: hematocrit, Hb: haemoglobin, BUN: Blood urea nitrogen, AST: aspartate aminotransferase, ALT: alanine transaminase, ALP: alkaline phosphatase, LDH: lactate Dehydrogenase, GGT: gamma-glutamyl transferase, CPK: creatine phosphokinase. *Reference values (**Lumsden *et al.*, 1979**).

Treatment and Outcome:

Following diagnostic tests immediate fluid therapy (0.9% NaCl, 154 mEq / L Na content to reduce serum sodium more slowly), antiemetic (metoclopramide 0.4 mg / kg IV), diuretic (furosemide 6 mg / kg IV), anticonvulsant (diazepam 1 mg / kg IV), beta-blockers (atenolol 1 mg / kg sublingual) to prevent sodium retention and heart rate control and oxygen therapy (50 milliliters / kg / minute) with nasal tube was initiated and the dog was hospitalized for continuous cardiac monitoring. Idwelling urinary catheter was inserted. After 24 hours of hospitalization. It was observed that convulsions disappeared and urine output was started. MGCS score was re-evaluated at the bedside (Day 2) and was determined as 12 (guarded). In the second blood analysis (Day 2), the sodium concentration was measured as 158 mmol / L (Table 3). As a result of the significant clinical improvement observed in the dog, the owner did not accept the longer hospitalization period and the patient was discharged on Day 2. It was learned that the dog was standing but vomiting and diarrhea continued in the evening of Day 2, after discharge. The next day, it was learned that the dog died on the morning of Day 3 (considering the first admission as Day 0).

Table 3. Blood gases result in Day: 2

Venous Blood Gases	Values	Reference*	Hemogram	Values	Reference*
pH	7,36	7.31-7.42	WBC	12,08	6-17 m/mm ³
pCO ₂	34,9	29-42 mmHg	Lym	5,16	0.6-5.1 m/mm ³
pO ₂	78,16	85-95 mmHg	Mon	0,2	0.1-1.7 m/mm ³
K	4,2	3.6-5.5 mmol/L	Gra	6,78	3-13.6 m/mm ³
Na	158	139-154 mmol/L	RBC	6,56	5.5-8.5 M/mm ³
Ca	0,89	2.2-3 mmol/L	MCV	58,8	58-73 fl
Cl	110	102-120 mmol/L	MCH	23,8	19.5-24.5 pg
Lactate	2,1	0-2 mmol/L	MCHC	32,45	28-40 g/dL
Base excess	-5,7	-4-4 mmol/L	Hct	44,78	35-55 %
HCO ₃	20,6	17-24 mmol/L	Hb	14,9	10-18 g/dL

pH: Power of hydrogen, pCO₂: partial pressure of carbon dioxide, pO₂: partial pressure of oxygen, K: potassium, Na: sodium, Ca: calcium, Cl: chlorine, HCO₃: bicarbonate, WBC: leukocyte, Lym: lymphocyte, Mon: monocyte, Gra: granulocyte, RBC: red blood cells, MCV: mean corpuscular volume, MCH: mean corpuscular haemoglobin, MCHC: mean corpuscular haemoglobin concentration, Hct: hematocrit, Hb: haemoglobin.

DISCUSSION

The sodium content of a regular commercial dry dog food is 0.5 - 2.5 g / 1000 kcal equal to 2-10 g / kg dry matter (Beynen, 2017). Therefore, extra salt, especially sodium, originated from milk, milk permeate, or whey consumption causes excessive salt intake (Smith *et al.*, 2016). In this case report, the cause of salt toxicity was evaluated as iatrogenic whey use.

The majority of clinical signs recorded in salt toxicity cases were gastrointestinal and neurological disorders (Bagley *et al.*, 1993, Pouzot *et al.*, 2007). In this case report, neurological findings such as seizures and gait disturbances, hypersalivation, vomiting and diarrhea due to mucosal irritation of salt were the most prominent clinical symptoms.

Hypernatremia is a common electrolyte abnormality caused by salt intoxication, diabetes insipidus or heat stroke, or hypotonic fluid loss such as vomiting, diarrhea, osmotic diuresis (Pouzot *et al.*, 2007). Extracellular volume abnormalities and hypernatremia, hyperchloremia and metabolic acidosis resulting from endocrine changes such as renin-angiotensin-aldosterone system and sympathetic nervous system due to excessive sodium intake have been reported as hematological changes (Dibartola, 2006). The hematochemical abnormalities detected in our case were consistent with previous reports (Pouzot *et al.*, 2007, Arambewela *et al.*, 2016).

The actual pathophysiology of hypernatremia on cardiac dysfunction is unknown. It is, however, hypothesized that increased extracellular sodium causes more calcium to exit the cell via a sodium-

calcium exchanger on the sarcolemma. Therefore, ECG findings such as QT prolongation, PR shortening, ST depression, decreased P and QRS amplitudes and sinus tachycardia have been reported (**Arambewela et al., 2016**). In our case, sinus tachycardia and mild ST depression were remarkable.

Changes in the brain and the level of consciousness have been reported in cases of salt toxicity, and the severity depends on the level of hypernatremia (**Bowins, 2018**). Observing the progression of neurological disorders and general prognosis, MGCS is useful and is important in evaluating the patient's motor activity, brainstem reflexes and level of consciousness (**Platt et al., 2001**). In our case, the MGCS score assessment was useful in evaluating general prognosis (7 at first admission, 12 before discharge) along with blood analysis .

The main goal of salt toxicity treatment is to correct the hypernatremia to a maximum of 2 mEq / L / hour of sodium (**Pouzot et al., 2007**). The amount of fluid required to correct the sodium concentration in the case of hypernatremia (178 mmol / L) detected in our case was calculated as 24.72 liters using the formulation [Free water deficit = $0.6 \times \text{bodyweight} \times (\text{Na}_{\text{Measured}} / \text{Na}_{\text{desired}} - 1) \times 9$]. It was given intravenously as a slow infusion during hospitalization (24 hours). Although a rate of 10-12 mmol / 24 hours is recommended for correction of hypernatremia by several authors (**Al-Absi et al., 2012, Braun et al., 2015, Arambewela et al., 2016**), it has been reported that slow correction is associated with high mortality (**Alshayeb et al., 2011**). In our case, the correction of hypernatremia was performed at a rate of 23 mmol / 24 hours. A decrease in sodium concentration (Table 3) was obtained and improvement in clinical findings and in MGCS (Table 1) score was observed on Day 2, the end of the hospitalization period.

The prognosis of salt toxicity in dogs depends on the amount of salt ingested, blood sodium concentration, the severity of neurological findings determined at the clinic admission, and the presence of any comorbid disease (**Pouzot et al., 2007**). The salt content of a typical whey ranges from 0.7-1.7 M and this corresponds to 99.34 g salt per 1 liter (**Blaschek et al., 2007**). Since the dog in the presented case is 20 kg, 80 grams of salt is sufficient for the occurrence of severe salt toxicity findings. The origin (type of cheese) of whey given by the owner was unknown. Therefore, it was thought that it contained a maximum of 99.34 gr salt in 1 liter and it was confirmed that salt toxicity was related to iatrogenic whey consumption.

In this case, the duration of hospitalization could not be extended to the recommended period

(**Fisher et al., 2006, Pouzot et al., 2007, Arambewela et al., 2016**) as it was against the will of the owner. It was determined that the pre-discharge blood sodium level was decreased (178 mmol / L at first admission, 158 mmol / L before discharge) and the MGCS score (7 at first admission, 12 before discharge) was found to be increased, but the ECG could not be evaluated. Therefore, blood analyzes, including blood gases and electrolyte measurements with MGCS assessment, were determined to be useful to gain insight into prognosis in the acute management of salt intoxication.

The limitation of this case report is that because of insufficient hospitalization period, the treatment protocol could not be completed and the cause of death could not be determined due to the lack of necropsy examination.

CONCLUSION

It is reported in the previous studies that the management of hypernatremia due to excessive sodium intake is difficult to treat. There is only one dog reported to have survived in cases of salt intoxication with neurological findings. Considering the difficulty of managing salt toxicity and the lack of literature data, this case report focuses on the clinical findings and acute management of salt toxicity. The most prominent findings in salt toxicity are ECG findings such as tachycardia and ST depression, low MGCS score and laboratory findings such as hypernatremia, hyperchloremia and metabolic acidosis. It was concluded that the treatment and management protocol, including fluid therapy, antiemetic, diuretic, anticonvulsant, beta-blocker and oxygen therapy administrations and evaluation of MGCS scoring with appropriate hospitalization period are beneficial.

Declaration of competing interest

On behalf of all authors, I hereby declare that no conflict of interest may interfere with the publication of the manuscript.

REFERENCES

- AL-ABSI, A., GOSMANOVA, E. O., WALL, B. M., 2012.** A clinical approach to the treatment of chronic hypernatremia. *Am J Kidney Dis*, 60, 1032–1038.
- ALSHAYEB, H. M., SHOWKAT, A., BABAR, F., 2011.** Severe hypernatremia correction rate and mortality in hospitalized patients. *Am J Med Sci*, 341, 356–60.
- ANGELOS, S. M., VAN METRE, D. C., 1999.** Treatment of sodium balance disorders. *Veterinary clinics of North America. Food Animal Practice*, 15, 587–607.
- ARAMBEWELA, M. H., SOMASUNDARAM, N. P., GARUSINGHE, C., 2016.** Extreme hypernatremia as a probable cause of fatal arrhythmia: a case report. *J Med Case Rep*, 10, 272.

- BAGLEY, R. S., LAHUNTA, A. D., RANDOLPH, J. F., 1993.** Hypernatremia, adipsia, and diabetes insipidus in a dog with hypothalamic dysplasia, *Journal of American Animal Hospital Association*, 29, 267-271.
- BEYNEN, A. C., 2017.** Salt in dog foods. *Creature Companion*, 10, 34-35.
- BLASCHEK, K. M., WENDORFF, W. L., RANKIN, S., 2007.** Survey of salty and sweet whey composition from various cheese plants in Wisconsin. *Journal of dairy science*, 90, 2029-2034.
- BOWINS, B., 2018.** The rational unconscious: Implications for mental illness and psychotherapy. *Am J Psychother*, 71, 28-38.
- BRAUN, M. M., BARSTOW, C. H., PYZOGA, N. J., 2015.** Diagnosing and management of sodium disorders: Hyponatremia and hypernatremia. *Am Fam Physician*, 91, 299-307.
- DIBARTOLA, S. P., 2006.** Disorders of sodium and water: hypernatremia and hyponatremia. In *Fluid*. 47-80. In: SP Dibartola (Ed), *Electrolyte and Acid-Base Disorders in Small Animal Practice* (3rd ed). St Louis MO, Saunders Elsevier.
- FISHER, L. A., KO, N., MISS, J., 2006.** Hypernatremia predicts adverse cardiovascular and neurological after SAH. *Neurocrit Care*, 5, 180-185.
- KAZANJI, N., ASSAD, W. A., GJEORGJIVSKI, M., 2015.** Extreme hypernatremia (254 mmol/l) and electrocardiogram findings. *Int Urol Nephrol*, 47, 871-872.
- KIM, M. K., LOPETCHARAT, K., GERARD, P. D., 2012.** Consumer awareness of salt and sodium reduction and sodium labeling. *J Food Sci*, 77, 307-313.
- LUMSDEN, J. H., MULLEN, K., McSHERRY, B. J., 1979.** Canine Hematology and Biochemistry Reference Values. *Can J Comp Med*, 43, 125-131.
- MORRIS, M., PATTON, R., TEETER, S., 1976.** Low sodium diet in heart disease: how low is low? *Veterinary Medicine*, 71: 1225-1227.
- PLATT, S. R., RADAELLI, S. T., MCDONNELL, J. J., 2001.** The prognostic value of the modified Glasgow coma scale in head trauma in dogs. *Journal of veterinary internal medicine / American College of Veterinary Internal Medicine*, 15, 581-584.
- POUZOT, C., DESCONE- JUNOT, C., LOUP, J., 2007.** Successful treatment of severe salt intoxication in a dog. *Journal of Veterinary Emergency and Critical Care*, 17, 294-298.
- SMITH, S. T., METZGER, L., DRAKE, M. A., 2016.** Evaluation of whey, milk, and delactosed permeates as salt substitutes. *J. Dairy Sci.* 99, 8687-8698.
- TITZE, J., RITZ, E., 2009.** Salt and its effect on blood pressure and target organ damage: new pieces in an old puzzle. *Journal of Nephrology*, 22, 177-189.

How to cite this article:

Erdem GÜLERSOY and Süleyman Serhat İYİĞÜN, 2021. Acute Salt Toxicity Caused by Whey Ingestion in Mixed-breed 6-months-old Puppy: A case study. *Journal of Applied Veterinary Sciences*, 6 (3): 31 – 37.

DOI: <https://dx.doi.org/10.21608/javs.2021.77045.1081>