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FATAL POISONING BY EXOGENIC INTAKE OF SODIUM CHLORIDE

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Summary. *The aim of this study is to point to the dangers of hypernatremia by exogenic intake of sodium chloride through ingesting excessive amounts of common salt or through aggressive correction of electrolytic disorders by using hypertonic solutions of sodium chloride, which may lead to heavy intoxication followed by metabolic encephalopathy (ME) and, very often, to a fatal outcome. Toxicity of common salt makes this substance a possible means of poisoning, accidental, suicidal or homicidal in origin. There is a report of fatal hypernatremia caused by per oral ingestion of a certain amount of common salt solution by a 70-year old woman, who was unsuccessfully treated at the Neurological Clinic in Niš and whose body was autopsied at the Institute for Forensic Medicine in Niš. Clinically, cerebral symptoms, febricity and hypernatremia (up to 192 mmol/l) were diagnosed and the patient died in comma with brain edema in ME.*
At autopsy, general congestion of the internal organs, brain and pulmonary edema, subarachnoid and intraparenchimal brain bleeding were present. Histology revealed disseminated micro-hemorrhages and demyelination in basal ganglia, vein micro-thrombosis in the brain and pulmonary tissue, and a characteristic finding of crenated erythrocytes in the bone marrow. Taking into account the results of both clinical and post-mortem investigations, and having ruled out other possible causes of the fatal outcome, the death was put down to excessive common salt intake.

Key words: *Sodium chloride, hypernatremia, intoxication, metabolic encephalopathy*

Introduction

The toxicity of excessive amount of salt has been known for ages and reports on it date back to the ancient China, when saturated salt solutions were used as a traditional suicide method. Despite the fact that this home provision is widespread and easily accessible, exogenic intake of excessive amounts of common salt which would cause vital clinical symptoms or death by hypernatremia can rarely be seen in adult healthy persons. However, due to excessive salt intake, hypernatremia is far more common in young babies and children, retards and persons with psychic deficiency, as well as in elderly people, most probably within a specific geriatric psychopathology (1,2).

Even though using saline solutions as emetics in first aid for acute poisoning has been abandoned, there are case reports, even today, on some sporadic fatal and non-fatal cases of ingestion of salt which was used for washing out the throat or raising low blood pressure. There are some known cases of hypernatremia of sailors who were forced to swallow a significant amount of seawater under specific circumstances. There are also some case reports of fatal poisoning by violent and unwilling intake of common salt solutions during exorcism (the ritual of getting rid of an evil spirit from a place or a person's body by prayers or magic) (2).

Accidental poisoning induced by excessive sodium intake in cases of infants is often due to inadvertent preparation of food. There was a dramatic case of mass poisoning of 14 babies in a clinical setting in England, March 1962 (3,4,5). Several cases of intentional excessive common salt intake and/or deprivation of exogenic intake of water as a result of the criminal act of child abuse were reported within the scope of Munchausen syndrome by proxy (6,7).

Some cases of accidental iatrogenic fatal poisonings during inadequate intravenous application of hypertonic saline solutions have been described in medical literature. Hypertonic solutions, especially 20% Mannitol and hypertonic saline solutions (sodium chloride 3-30%), are routinely used in curing brain edema and intracranial hypertension syndrome in numerous diseases (traumatic brain damage, artery and vain infractions, intra-cerebral and subarachnoid hemorrhages, tumor progression, hepatic encephalopathy, post-operational edema, etc.), during which their uncritical and aggressive application may lead to aggravation of the disease and to death itself due to hypernatremia (8).

Patients and Methods

In order to present the case, we used the medical files collected during hospital treatment of a 70-year old

female patient, as well as complete autopsy findings taken during the examination. The autopsy of her body was done 20 hours after her death. Routine histology of the sample was carried out after fixation in 10% buffered formalin. After that, the tissues were embedded in paraffin, and the paraffin samples were stained with hematoxylin-eosin (HE). The toxicological analysis of the body material (parts of the organs, blood and urine) was done using the method of thin-layered chromatography (TLC), whereas the chemical analysis of alcohol in the blood was performed by McNally method.

Results

A 70-year old female patient who was a long-term hypertonic and a 5-year diabetic had a positive psychiatric anamnesis and suicidal tendencies in the previous period. According to the hetero-anamnestic data, she felt dizzy and unstable so she drank a solution of an unspecified amount of common salt unsolicited which, after vomiting and diarrhea, only aggravated her previous state. On her admission to the Neurological Clinic, she was conscious, with a rhythmic and rapid heartbeat, and she had normal findings on both of the lungs, the stomach, and the ocular fundus. During the first hours, she was hypertensive (170/90mmHg), tachycardic (SF 120-130/min.) and then hypotensive (80/65mmHg) up to the immeasurable values of blood pressure. She developed the body temperature up to 39,4°C. Soon after she had been admitted, her state of consciousness changed from somnolent to comma, with general hypotony and without pathological muscle-tendon reflexes. The level of serum sodium chloride ions was 177-192-183 mmol/l; chloride ions = 152 mmol/l; glucose = 17.1–14.1-13.1 mmol/l; acid-basic state: pH=7.3; pCO_2 = 24.9-41.0; pO_2 = 61.6-95.2; HCO_3 = 14.2-19.8; $satO_2$ = 91.7-96.6%.

Although she was given intensive medical treatment, including hypotonic infusions to correct hypernatremia, the lethal outcome occurred 15 hours after she had been hospitalized for ME.

The autopsy confirmed a high level of brain edema, general congestion of the internal organs, disseminated micro-bleedings (Fig. 1) and demyelination in the brain parenchyma of basal ganglia (Fig. 2), subarachnoid hemorrhages (Fig. 3), and venous micro-thromboses in the brain and pulmonary tissue.

The microscopic examination of the bone marrow specimen revealed crenated erythrocytes (Fig. 4). Apart from some degenerative changes in the heart muscle and the kidneys, which are normal at a certain age, other pathological conditions in the internal organs have not been found. Injuries to the body have not been found either, except for injection needle wounds. The toxicological analysis was negative, and endogen alcohol was only found in the blood (0.02%). On the basis of the clinical and post-mortem findings, it was concluded that the death in this case was a direct consequence of excessive common salt intake.

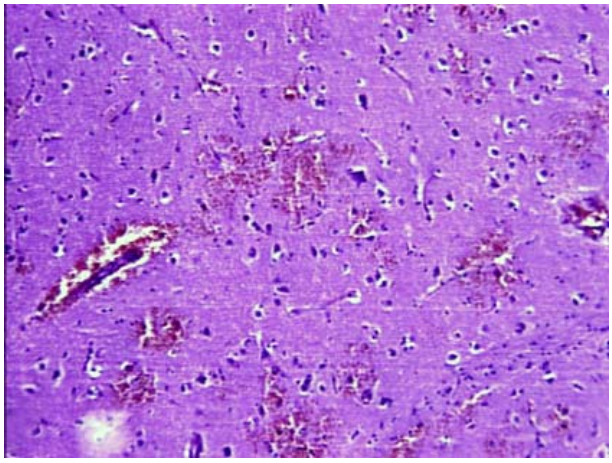

Fig. 1. Histopathology in fatal salt poisoning: perivascular and disseminated micro-bleedings in the swollen brain parenchyma. (HE staining, original magnification 20×)

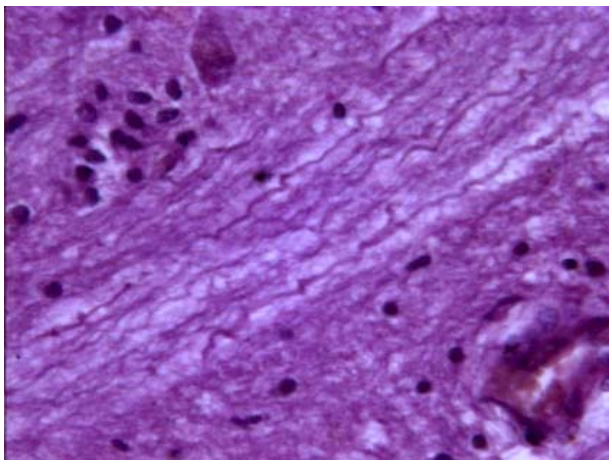

Fig. 2. Histopathology in fatal salt poisoning: demyelination in the brain parenchyma of basal ganglia. (HE staining, original magnification 100×)

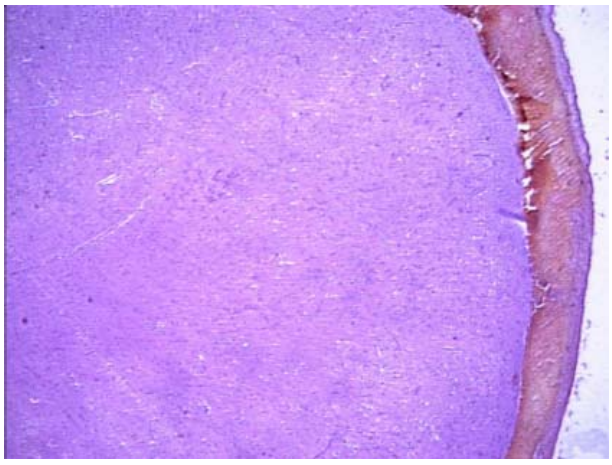

Fig. 3. Histopathology in fatal salt poisoning: subarachnoid hemorrhage. (HE staining, original magnification 4×)

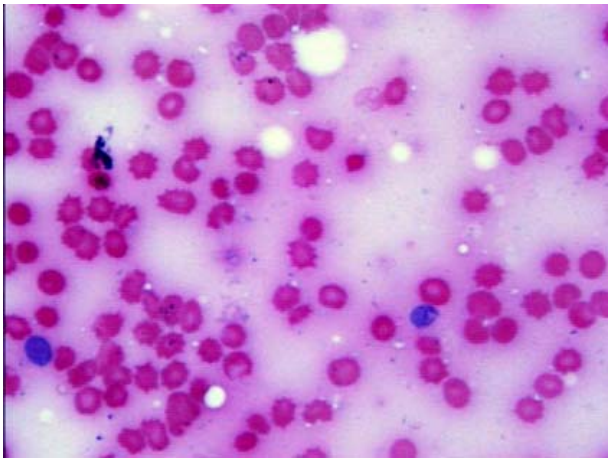

Fig. 4. Histopathology in fatal salt poisoning: crenated red blood cells in the bone marrow specimen. (HE staining, original magnification 100×)

Discussion

Hypernatremia occurs when there is a deficiency of water in the body compared to solute sodium chloride ions. This state can occur as a consequence of insufficient liquid intake or its extreme loss (burns, the effect of osmotic laxatives, patients with changed consciousness, excessive insensible losses-febrile state, tachypnea, etc.) or increased exogenic sodium chloride intake (9,10,11).

The toxic oral dose of salt is 0.5-1 g/kg of body weight, whereas the fatal dose amounts to about 1-3 g/kg of body weight, depending on the age, gender, general health state, and other individual characteristics. It has been concluded that 1 g/kg of body weight sodium chloride raises the level of sodium chloride ions in blood to about 30 mmol/l. In the case presented, there were no precise data as to the amount of the ingested salt. However, according to the clinically established level of sodium chloride ions in our patient, the ingested amount of salt must have been greater than 1.5 g/kg of body weight (10,11).

The pathophysiological mechanism of hypernatremia is seen in hyperosmolarity of extracellular (interstitial and intravascular) liquid, with intracellular dehydration as its consequence. Metabolic disorders originating from misbalance of water and electrolytes are most vivid in the central nervous system and are expressed in ME (10). The brain starts to decrease its volume five minutes after developing the hypertonic state. Partial restitution of the brain volume occurs within a few hours, when electrolytes enter the brain cells (fast adaptation), and normalization of the brain volume finishes after a few days as a result of intracellular accumulation of organic osmolytes (slow adaptation). A slow correction of the hypertonic state reestablishes normal osmolarity of the brain because the organic osmolytes keep balance with the decrease in the natremia level during hypernatremia correction. As opposed to this, a fast correction results in brain edema since water penetration in the brain cells is faster than is sodium decrease in the extra cellular area (10,11).

The symptoms of hypernatremia are principally neurological, characterized by an alerted sensorium, decrease in spontaneous activity, lethargy, ataxia, stupor, and comma. As in other metabolic encepalopathies, neurological manifestations correlate with the speed of growth and sodium serum concentration. In adults, the symptoms may develop with serum sodium levels of 150 mmol/l, although a majority of symptomatic patients have the values that are greater than 160 mmol/l. Levels over 180 mmol/l are frequently lethal (13). Due to dehydration of the brain cells and decrease in the brain volume, mechanical damage of small blood vessels occurs with the subarachnoid and intra-cerebral hemorrhages consequence, which was also seen during the autopsy of the case presented (10,12,14,15).

In autopsy findings of fatal sodium chloride poisoning cases, brain edema, venous and capillary congestion, cortical venous thromboses and venous brain infarcts are predominant. Demyelination, which covers a wide area of the thalamus, basal ganglia, external capsules and cerebral vermis, has been described in literature in cases of three deceased children, whose hypernatremia was aggressively treated and whose values of sodium chloride serum were 195, 168 and 177 mmol/l (10,16,17). Recent studies show that a fast growth of sodium chloride ions in patients who have long hypo/hypernatremia is key to the pathogenesis of this disorder although it is still unknown in which way the oscillations of sodium chloride ions lead to demyelination lesions in such sensitive places (18).

Through presenting this case, we are trying to draw doctors' attention to this rare form of intoxication which can be seen at every age. The clinical symptoms within ME caused by hypernatremia may be wrongly understood as being primary brain lesions, thus leading to a failure in detecting a misbalance between water and electrolytes.

A fast diagnosis and an early therapeutic approach to hypernatremia cases may lead to a total recovery, during which intensive but gradual correction of the water-electrolytes misbalance lessens the therapeutic complications and danger of additional aggravation of brain edema (19,20).

The findings in fatal sodium chloride intoxication are highly unspecific, and if no data on the history of illness are available, it might easily escape detection. Besides a high level of brain edema, general congestions of the internal organs, subarachnoid and intracerebral micro-hemorrhages and micro-thrombi, crenated erythrocytes might point to the right diagnosis.

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SMRTNA TROVANJA IZAZVANA EGZOGENIM UNOSOM NATRIJUM HLORIDA

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Kratak sadržaj: *Cilj rada je da ukaže na opasnosti od hipernatremije izazvane egzogenim unosom natrijum hlorida (NaCl) ingestijom prekomerne količine kuhinjske soli ili agresivnom korekcijom elektrolitnih poremećaja hipertonim rastvorima NaCl, usled koje može doći do teške intoksikacije praćene metaboličkom encefalopatijom (ME), neretko i sa fatalnim ishodom. Toksičnost kuhinske soli opredeljuje ovu supstancu kao sredstvo trovanja akcidentalnog, suicidnog ili homicidnog porekla. Prikazn je slučaj fatalne hipernatremije nakon peroralne ingestije rastvora kuhinjske soli od strane 70-godišnje starice, bezuspešno lečene na Neurološkoj klinici Kliničkog centra u Nišu, čiji je leš obdukovan u Zavodu za sudsku medicinu u Nišu. U kliničkoj slici dominirala je cerebralna simptomatologija, febrilnost i hipernatremija (do 192 mmol/l), a pacijentkinja je umrla u komi, pod kliničkom slikom moždanog edemau sklopu ME. Autopsijski su utvrdjeni znaci opšte kongestije unutrašnjih organa, edem mozga i pluća, subarahnoidalna i intraparenhimska moždana krvarenja. Mikroskopskim pregledom su nadjene diseminovane mikrohemoragije i demijelinizacija u nivou bazalnih ganglija, venska mikrotromboza u moždanom i plućnom tkivu, a karakterističan je nalaz erithrocita nazubljenih ivica u razmazu koštane srži. Sagledavajući kliničku sliku i autopsijski nalaz, uz isključenje ostalih mogućih uzroka smrti, u konkretnom slučaju je smrtni ishod pripisan prekomernom unosu kuhinjske soli.*

Ključne reči: *Natrijum hlorid, hipernatremija, trovanje, metabolička encefalopatija*