OPEN ACCESS

Middle East Journal of Medical Case Reports
ISSN: 2789-8660 (Print) & ISSN: 2958-2121 (Online)
Frequency: Bi-Monthly
DOI: https://doi.org/10.36348/merjmcr.2026.v06i03.001

KUWAIT SCHOLARS PUBLISHER
Website: http://www.kspublisher.com/
Email: office@kspublisher.com

Severe Hypernatremia and Acute Kidney Injury in an Elderly Patient

Konstantinos S. Mavromatidis[1]*, Anastasia K. Georgoulidou, Nephrologist (MD, PhD)[2], Alexandra D. Demka, (MD, PhD)[3], Irini M. Kalogiannidou, Nephrologist (MD, PhD)[4]

[1]Director, Renal Unit «Dimokrition», Komotini, Thrace, Greece
[2]Director Nephrologist, Nephrology Department, General Hospital of Komotini, Thrace, Greece
[3]Nephrologist, Consultant, Nephrology Clinic, General Hospital «Papageorgiou», Thessaloniki, Greece
[4]Consultant, Renal Unit Dimokrition, Komotini, Thrace, Greece

Abstract: We present 87-year-old woman with moderate hypovolemia, severe hypernatremia (serum sodium 184 mmol/L) and hyperosmolarity (serum osmotic pressure 416 mOsm/L), due to reduced fluid intake and diuretic use, accompanied by acute kidney injury (pre-renal). However, the patient had very mild symptoms upon her admission (communication disorders, drowsiness). She was successfully treated in a common nursing ward, with careful assessment of the fluid status, as well as with careful restoration of her deficits (mainly water and much less sodium). Reference is made to the causes, frequency, clinical picture and outcome of these patients, as well as review of the literature.

Keywords: Diuretics, Hypovolemic Hypernatremia, Osmotic Pressure, Hypernatremia Treatment, Elderly Patients.

Case Report

***Corresponding Author:**
Konstantinos S. Mavromatidis
Director, Renal Unit «Dimokrition», Komotini, Thrace, Greece

How to cite this paper:
Konstantinos S. Mavromatidis *et al* (2026). Severe Hypernatremia and Acute Kidney Injury in an Elderly Patient. *Middle East Res J. Case Rep, 6*(3): 26-29.

Article History:
| Submit: 22.06.2026 |
| Accepted: 25.07.2026 |
| Published: 29.07.2026 |

INTRODUCTION

The amount of the body water depends on gender (women have less water, due to their bigger fat content, which is hydrophobic) and age (muscles contain mainly water something men have more, nevertheless muscle mass of the elderly people is reduced). Water in women constitutes 50% of body weight, which decreases even more after the age of 75-80 years [1]. Hypernatremia is defined as the increase of serum sodium (>145 mEq/L) because mainly the reduce of water in relation to the sodium of the body, which is caused by pure loss of water or hypotonic fluids [2], by an inability to take water due to movement problems that are usually present in the elderly, but also iatrogenically by the administration of hypertonic solutions (usually sodium bicarbonates 8.4%) or by taking salt orally for the purpose of suicide [3].

Serum sodium concentration and serum osmolality are regulated by water homeostasis, which is controlled by the sense of thirst and the presence and action of antidiuretic hormone (ADH) [2-4]. Hypernatremic patients are usually elderly or very elderly with motility problems, who often also have dementia [5, 6]. Elderly people are less sensitive to disturbances of water balance, and often also they are unable to move to the place where there is water to drink. These are the more common causes of hypernatremia in the elderly, although we should not forget the use of diuretics by many of them, which also contribute to higher water loss in relation to the amount of sodium remove through urine [7], as well as the fact that in old age there is a disorder of urine concentration, as well as hormonal disorders, such as a decrease in ADH secretion [8].

CASE REPORT

We present an 87-year-old woman (body weight 40 kg), with a history of atrial fibrillation (for the last 4 years) and ischemic stroke with right hemiplegia and emission aphasia (for the last 3 years), who was transferred to the Emergency Department (ED) of the Hospital, from her relatives who noted communication disorders for the last 10 days and reduced blood pressure the last 2 days. The oral water intake of this woman was estimated to be approximately 300 ml/24h (stable for a long period of time), while the daily urine output was satisfactory, as assessed by her relatives from her diapers-pants that they were change. Her food was given every day in the form of pureed foods by mouth. Her medications included furosemide tablets (20 mg/24h)

and losartan (50 mg/24h), which were discontinued upon her admission to the hospital.

The patient was hemodynamically relatively unstable upon examination in the ED, with blood pressure of 70/40 mmHg, 100/min pulses, decreased of skin turgor (supraclavicular and in inner surface of thighs), dry mucous membranes (inner surface of cheeks), and with bulbs that are compressible and dented under gentle pressure (indications of intracellular dehydration). Neurological assessment revealed pre-existing chronic neurological symptoms plus lethargy with drowsiness. Serum glucose was 6.94 mmol/L, urea 113.6 mmol/L, creatinine 477.5 mmol/L, potassium 5.9 mmol/L, sodium 184 mmol/L, chloride 135 mmol/L, ratio Na^+/Cl^- 1.36 (slightly higher the normal, indicating that the increased serum sodium was mainly due to dehydration). Also, total serum calcium was 2.1 mmol/L, phosphate 1.0 mmol/L, albumin 27 g/L, oxaloacetic transaminase (SGOT) 36 IU/L (normal <18 IU/L), pyruvate transaminase (SGPT) 81 IU/L (normal <18 IU/L), creatine phosphokinase (CPK) 286 IU/L (normal range 24 - 198 IU/L), lactate dehydrogenase (LDH) 227 IU/L (normal range 115-220 IU/L), urate 594.8 umol/L (normal range 208 – 428 umol/L), erythrocyte sedimentation rate (ESR) 71 mm/1st hour and C-reactive protein (CRP) 35 mg/L (normal <10 mg/L). The complete blood count revealed leukocytosis (white blood cells 11,900/mm^3), with a normal pattern. The general urine examination showed a specific gravity of 1015, pH 7.0, nitrites positive (+), albumin 500 g/L, leukocytes > 200/ml per high-power field of view and red blood cells 30 - 50 high-power field of view. From the arterial blood gases, high anion gap metabolic acidosis was found [anion gap = Na^+ - (Cl^- - HCO_3^-) = 184 - (135 + 28.4) =184 - 163.4 = 20.6 mmol/L], apparently due to acute kidney injury and metabolic alkalosis (delta gap = Na^+ - Cl^- - 39 = 184 - 135 - 39 = +10 mmol/L), apparently due to diuretic intake and to hypoalbuminemia (pH 7.38, $PaCO_2$ 48 mmHg, PaO_2 72 mmHg, HCO_3^- 28.4 mmol/L). Serum osmolality at admission was 416 mOsm/L and urine osmolality was 486 mOsm/L. Urine sodium on day one was 67 mmol/L.

Was diagnosed hypovolemic hypernatremia with hypotension and acute kidney injury, as well as urinary tract infection. The renal ultrasound showed normal findings for the patient's age. Urine culture performed form sample which was taken on the first day showed the presence of E. Coli at a density of 10^5 colonies/ml after 3 days.

During the first two hours, 500 ml of 0.9% NaCl solution was administered, at which time was observed an improvement in the patient's blood pressure (90/50 mmHg from 70/40 mmHg) and heart pulses (85/min from 100/min), but also in drowsiness. Amoxicillin (500 mg/12h intravenously) was given for the urinary tract infection that she clinically appeared to have (something was confirmed with urine sample culture as mentioned). This was followed by the administration of 5% dextrose solution and tap water orally (but also 500 ml of 0.9% NaCl which were administered on the 4th day of her hospitalization). This was done because from the history, clinical picture and high serum sodium levels, there was certainly water deficit, which was calculated of about 4.3 L, which should be administered along with a small amount of sodium, given that she was relatively hypovolemic.

Serum sodium reached 150 mmol/L on day 4th of hospitalization (from 184 mmol/L on admission), while the serum osmolality also decreased to around 300 mOsm/L H_2O on day 4th (from 416 mOsm/L on admission). Serum urea and creatinine during her hospitalization showed a progressive decrease from day 1st (serum creatinine 477.5 mmol/L and serum urea 113.6 mmol/L), reaching to normal levels (serum creatinine 115 mmol/L and serum urea 13.9 mmol/L) on day 6th of hospitalization. On this day of hospitalization, serum glucose was noted to be 6.94 mmol/L, potassium 3.6 mmol/L, sodium 139 mmol/L, urate 255.6 umol/L, and CRP 45 mg/L. The patient was discharged on day 7th, after her renal function, hypernatremia, hyperosmolality, and her clinical picture had been restored.

DISCUSION

Community-acquired hypernatremia is often found in extreme ages (infants and the elderly) [3]. Patients with mental disorders are also at increased risk for hypernatremia [9], because they are often unable to drink water alone or to reach to the water on their own.

Studies have shown that hypernatremia diagnosed in 1% of people living in nursing homes [8], and in 9% of those admitted to intensive care units [10]. As for the ED, Batalile *et al*., out of 54,753 visits, within a year, they found hypernatremia in 216 patients (0.4%) and of those, the 32% had serum sodium between 161 - 187 mmol/L (one of them had 183 and another one 187 mmol/L), while the mean osmotic pressure of all hypernatremics was 347 ± 25 mOsm/L, meaning that severe hypernatremia was very rare [5]. In general, the frequency of severe hypernatremia (serum sodium > 160 mmol/L) is low [11], and very severe (serum sodium > 190 mmol/L) even lower [12], since few cases with very elevated serum sodium levels have been published to date [13], with the maximum value reaching 254 mmol/L in a patient with anorexia nervosa) [14].

The most common cause of hypernatremia is the loss of hypotonic fluids from the gastrointestinal tract or from the kidneys (from taking diuretics), but also from reduced oral water intake, as happened to our patient, who, in addition to the diuretic, was drinking daily a relatively small amount of water, while she had a constant and rather normal diuresis. This was apparently due to the diuretic intake, the acute kidney injury (which was non-oliguric) and the increased serum urea levels,

according to the confirmation of her relatives. In a study of hypernatremic patients, 47% had as a cause condition with water loss, who in fact showed hypernatremia paradoxically by 39% in winter and by 20% in summer [5], as happened to our patient, who had the hypernatremia episode in the winter.

All forms of hypernatremia represent hypertonic states that lead to ADH secretion and activation of the feeling of thirst. This increase in plasma osmolality due to hypernatremia leads to water movement from brain cells (and generally from all cells), leading to their shrinkage. The brain then adaptively accumulates osmoles in its cells, aiming to maintain their volume. This is why the findings and symptoms of hypernatremia are mainly due to central nervous system dysfunction, which are predominates when the serum sodium concentration increases sharply [2]. However, elderly people with hypernatremia have few symptoms, until the serum sodium levels reach at 160 mmol/L [15]. Thus, intense thirst is usually absent in the elderly people, who are generally hypodipsic. The level of consciousness is correlated with the severity of hypernatremia [15], and the rapidity with which the serum osmotic pressure increased [4], however, our patient, despite very severe hypernatremia and particularly the very elevated serum osmotic pressure, had only a mild clinical semiology (drowsiness). It is worth noting that non-specific lethargy and weakness are frequent symptoms of hypernatremia. Muscle weakness, confusion and coma are sometimes present and are manifestations of the coexisting's disorder and not of the hypernatremia itself. Our patient had few of the above symptoms and this may have played a role in the delayed transfer to the hospital of her by her relatives.

In cases where hypernatremia is due to diuretics and insufficient oral water intake, typically the urine sodium is > 20 mmol/L and the urine osmotic pressure is < 700 mOsm/L [9], as was the case in our patient, who had a urine sodium of 67 mmol/L and a urine osmotic pressure of 486 mOsm/L. The fact that our patient responded therapeutically, both in terms of hypernatremia and acute kidney injury, to the administration of mainly water (with a minimal amount of sodium) confirms the diagnosis of relatively hypovolemic hypernatremia (water deficiency). It is noted that the urinary tract infection was also easily controlled and the CRP began to decrease from the second day of the antibiotic treatment, while her white blood cells also normalized soon.

To estimate the water deficit, two parameters are needed, the serum sodium levels and the patient's body weight. It is also help the application of the Adrogué-Madias equation for the expected change in patients' serum sodium levels from fluid administration which is the following: Expected sodium change = (solution sodium - patient's sodium) / (body water + 1) [15], which contributes to the smooth restoration of serum sodium levels, so the change of serum sodium levels to not exceed the of 8-10 mmol/L daily, as happened in our patient, in which of course this formula was not applied, since the greatest water deficit was administered orally.

Because hypernatremia is not a homogeneous situation, in the initial steps of treatment it is necessary to determine the water volume status and treat the underlying disease, so as not to worsen the patient's general condition. Thus, the treatment of hypernatremia is considered appropriate when it aims: a) initially at restoring the volume when there is a disorder and b) at treating the underlying cause and hypertonicity (preferably with intravenous administration of hypotonic fluids). In patients with hypernatremia who have relative hypovolemia (moderate or mild), as our patient had, the administration of hypotonic fluids (NaCI 0.2% or dextrose 5%) is required [15], of course is needed frequently checking serum glucose levels (to avoid osmotic diuresis from possible hyperglycemia).

Rapid diagnosis and timely application of therapeutic treatment in patients with hypernatremia can lead to complete recovery of the patient, during which the water deficiency is gradually corrected. This minimizes the side effects of the treatment (cerebral edema), which are also serious and usually irreversible [15]. In chronic hypernatremia when there are no clinical manifestations of increased intracranial pressure, the reduction of serum sodium levels should be done at a rate of <0.5 mmol/L/hour [5-15], which we also followed, and perhaps more conservatively in our patient.

Severe hypernatremia is accompanied by clinical manifestations from the central nervous system and has an increased mortality (serum sodium levels > 180 mmol/L are usually fatal) [3]. In adults, hypernatremia has a mortality of 20 - 60%, depending on the underlying disease, its severity, its management and whether it is accompanied by complications (subdural hemorrhage, venous sinus thrombosis) [3-12].

Mortality of elderly people with hypernatremia is also high. However, Bataille *et al*., did not find an association between the severity of hypernatremia and mortality in outpatients, although they found that the main risk factors for death were the findings of hypovolemia (hypotension, weak pulse) and acute renal injury [5], as was the case of our patient, which of course survived. Also, the mortality of hypernatremia in the intensive care unit is increased, but it is not known whether this is due to the underlying disease or to hypernatremia itself and its severity [3-11]. Therefore, despite the increased mortality of this disorder, our patient who had very severe hypernatremia and serum hyperosmolality and despite her hospitalization in a general ward survived. This is attributed to the timely and correct diagnosis that was made, the correct

assessment of her fluid balance, and the very close monitoring that she had.

CONCLUSION

It is concluded that early diagnosis of severe hypernatremia in elderly patients is critical and careful restoration of the water deficit is needed, so that the disorder can be safely restored and the lives patients can be saved.

REFERENCES

1. Fulop, T. Jr, Worum, I., Csongor, J., Leövey. A. (1985) Body composition in elderly people. I. Determination of body composition by multiisotope method and the elimination kinetics of these isotopes in healthy elderly subjects. Gerontology, 31, 6-14. doi: 10.1159/000212676.
2. Yun, G., Baek, S.H., Kim, S. (2023) Evaluation and management of hypernatremia in adults: clinical perspectives. The Korean Journal of Internal Medicine, 38, 290-302. doi: 10.3904/kjim.2022.346.
3. Mittal, R., Lai, A.M.W., Coskin, R. Kaplan JM, Doyle J. (2025) Comprehensive overview of hypernatremia: Pathophysiology, diagnosis, and management. British Journal of Hospital Medicine. (Lond), 86(12), 1-32. doi: 10.12968/hmed.2024.0822.
4. Lima, E.Q., Aguiar, F.C., Barbosa, D.M., Burdmann, E.A. (2004) Severe hypernatraemia (221 mEq/l), rhabdomyolysis and acute renal failure after cerebral aneurysm surgery. Nephrology Dialysis and Transplantation, 19(8), 2126-2129. doi: 10.1093/ndt/gfh324.
5. Bataille, S., Baralla, C., Torro, D., Buffat C, Berland Y, Alazia, M. et al. (2014) Undercorrection of hypernatremia is frequent and associated with mortality. BMC Nephrology, 15, 37-46. doi: 10.1186/1471-2369-15-37.
6. Begg, D.P. (2017) Disturbances of thirst and fluid balance associated with aging. Physiology & Behavior, 178, 28-34. doi: 10.1016/j.physbeh.2017.03.003.
7. Phillips, P.A., Bretherton, M., Johnston, C.I., Gray, L. (1991) Reduced osmotic thirst in healthy elderly men. American Journal of Physiology, 261(1 Pt 2), R166-R171. doi: 10.1152/ajpregu.1991.261.1.R166.
8. Beck, L.H., Lavizzo-Mourey, R. (1987) Geriatric hypernatremia. Annals of Internal Medicine, 107(5), 768-769. doi: 10.7326/0003-4819-107-5-768.
9. Lin, M., Liu, S.J., Lim, I.T. (2005) Disorders of water imbalance. Emergency Medicine Clinics of North America, 23(3), 749-770. doi: 10.1016/j.emc.2005.03.001.
10. Lindner, G., Funk, G.F., Schwarz, C., Kneidinger, N., Kaider, A., Schneeweisset, B. al. (2007) Hypernatremia in critically ill is an independent risk factor for mortality. American Journal of Kidney Disease, 50(6), 952-957. doi: 10.1053/j.ajkd.2007.08.016.
11. Kolmodin, L., Sekhon, M.S., Turgeon, A.F., Eg Griesdale, D. (2013) Hypernatremia in patients with severe traumatic brain injury: a systematic review. Annals of Intensive Care, 3, 35-42. doi: 10.1186/2110-5820-3-35.
12. Kamatam, S., Waqar, A., Chatterjee. T. (2023) Extreme hypernatremia due to dehydration. J Med Cases, 14(7), 232-236. doi: 10.14740/jmc4124. Epub 2023 Jul 31.
13. Arambewela, M.H., Somasundaram, N.P., Garusinghe, C. (2016) Extreme hypernatremia as a probable cause of fatal arrhythmia: a case report. Journal of Medical Case Reports, 10(1) 272. doi: 10.1186/s13256-016-1062-9.
14. Kazanji, N., Al Assad, W., Gjeorgjievski, M., Thalla, R.K (2015) Extreme hypernatremia (254 mmol/L) and electrocardiogram findings. International Urology and Nephrology, 47(5), 871-872. doi: 10.1007/s11255-015-0969-1.
15. Adroguė, H.J., Madias, N.E. (2000) Hypernatremia. New England Journal of Medicine, 342(20), 1493-1499. doi: 10.1056/NEJM200005183422006.