Journal of Biosciences and Medicines, 2026, 14(11), 381-391
https://www.scirp.org/journal/jbm
ISSN Online: 2327-509X
ISSN Print: 2327-5081

Severe Hyponatremia in Hospitalized Patients in Non-Surgical Wards. Causes, Clinical Symptoms and Outcome

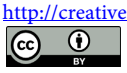
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How to cite this paper: Mavromatidis, K.S. and Kalogiannidou, I.M. (2026) Severe Hyponatremia in Hospitalized Patients in Non-Surgical Wards. Causes, Clinical Symptoms and Outcome. *Journal of Biosciences and Medicines*, **14**, 381-391.
https://doi.org/10.4236/jbm.2026.1411027

Received: October 15, 2026
Accepted: November 13, 2026
Published: November 16, 2026

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Abstract

All patients who had serum sodium ≤ 120 mmol/L upon admission or during their hospitalization in the Internal Medicine and Cardiology Departments of our 210-bed hospital were prospectively studied over a 5-year period (from November 2020 to October 2025). The cause of hyponatremia, the clinical picture at admission were recorded in all patients, and the levels of serum sodium, glucose, cholesterol, triglycerides, lactate, and blood gases were determined and monitored. Also, immediately after the diagnosis of hyponatremia, fundoscopy and brain CT scan were performed to investigate the presence of cerebral edema. The method of therapeutic treatment (conservative or hemofiltration), the duration of hospitalization, and the outcome of the patients were recorded. A total of 26 patients (8M, 18F), with a median age of 73 years, were enrolled. In 10 cases, the main cause was iatrogenic, in 20 patients' diuretics were involved (in 10 of them thiazides) and in 10 there was also heart failure. 23/26 had more than one cause of hyponatremia. Although all patients had serum sodium ≤ 120 mmol/L, 14 had serum sodium levels ≤ 115 mmol/L. The clinical signs were severe in 12 (coma, seizure, disorientation, confusion, depression) which were the cause of the diagnosis. CT revealed cerebral edema in 4 patients, who had serum sodium levels of 101, 98, 116 and 98 mmol/L, respectively. Regarding fundoscopy findings, only 3/17 cases tested (aged 72, 62 and 98 years) showed optic disc edema (the first and last patients also had CT findings of cerebral edema). The 24 patients were treated conservatively and two with hemofiltration. The duration of hospitalization ranged from 4 - 10 days (median 6 days). Of the 26 patients, 2 died, 18 improved and 6 recovered completely. It is concluded that severe hyponatremia is diagnosed mainly by clinical findings, the main causes are iatrogenic (diuretics), it mainly affects elderly women, and its prognosis is good if diagnosed and treated promptly.

Keywords

Severe Hyponatremia, Diuretics, Iatrogenic Causes, Elderly Patients, Heart Failure, Hemofiltration, Central Pontine Myelinolysis, Cerebral Edema

1. Introduction

Hyponatremia is the most common electrolyte disorder in hospitalized patients [1]-[3]. Its most common causes are diuretics (thiazide or loop), congestive heart failure and liver disease, while 75.3% of patients have more than one cause [4]. Recently, it was found that severe hyponatremia (serum sodium less than 120 mmol/L) affected 1% of hospitalized patients [5] [6]. Hyponatremia is also more common in older women.

In hyponatremia, symptoms are usually not noted unless the serum sodium decreases below 120 mmol/L, which are basically non-specific (headache, lethargy, nausea, etc.). In our study, severe cases of hyponatremia (serum sodium ≤ 120 mmol/L) were recorded, and their causes, clinical-laboratory data, type of treatment and outcome were analyzed.

2. Patients-Methods

2.1. Patients

The study included patients who had serum sodium levels ≤ 120 mmol/L upon admission or during their hospitalization in Internal Medicine and Cardiology Department of our Hospital. The cause of hyponatremia, the presence or absence of heart failure, the medications they were taking, other possible causes and their clinical icon (clinical sings and objective findings) were recorded in all of them. From the laboratory test, the levels of serum sodium, glucose, cholesterol, triglycerides, lactate, as well as blood gases were monitored at the diagnosis of hyponatremia, and during the treatment.

2.2. Methods

Hyponatremia was considered iatrogenic when the patient was receiving intravenously hypotonic solutions or if a doctor recommended increased fluid intake (a common erroneous instruction given by many doctors to reduce serum urea, e.g. in patients with heart failure), while his kidneys could not handle it. Diuretics were considered responsible when their intake began at least 6 months before the episode of hyponatremia.

When diagnosing hyponatremia, computed tomography (CT) of the brain was performed to investigate the presence of cerebral edema, as well as fundoscopy to detect possible papilla edema. The therapeutic treatment used (conservative or hemofiltration), the duration of hospitalization and the outcome of the patients were recorded.

Patients presenting with hyponatremia and manifestations of encephalopathy received at least two doses of 150 mL 3.0% NaCl solution—administered 30 minutes apart—to reduce cerebral edema. Other conservative management measures included the discontinuation of causative factors (specific medications such as diuretics), the cessation of fluid consumption (mainly tap water), and the improvement of cardiac function, among others.

Serum electrolytes were determined with an ion-selective analyzer, serum glucose, cholesterol and triglycerides with an automatic biochemical analyzer, blood gases and lactate were determined directly with a GEMPremier 3000 gas analyzer from blood taken from the radial artery.

2.3. Ethical Committee

This prospective study was conducted in accordance with the Declaration of Helsinki and the Ethical Guidelines for Medical and Health Research Involving Human Subjects and was approved by the Scientific Council of the General Hospital of Komotini, Greece (18/11/2020, registration number 4/2020). The patients included in the study agreed to sign their consent to participate, after being thoroughly informed about the protocol.

3. Results

A total of 26 patients (8M, 18F), aged 42 to 98 years (median 73) (12 were over 75 years), were included. The demographic data of the patients, the causes of hyponatremia and its type (hypovolemic, hypervolemic or euvolemic) and their clinical signs and findings (mainly neurological) are shown in Table 1.

Table 1. Age, sex, causes of hyponatremia, neurological clinical sings and findings (ACE = Angiotensin converting enzyme).

Sex	8M, 18F
Ηλικία	71.3 ± 12.4 (range 42 - 98 years, median 73)
Causes of hyponatremia	Diuretics 20 (13 from thiazides)
	Taking ACE inhibitors 12
	Heart failure 10
	Iatrogenic 10 (high intravenous infusion hypotonic fluids or per os given high volume of tap water)
	Hyperglycemia 2
	Potomania 1
	Acute kidney injury 1
Kind of hyponatremia	Hypervolemia 18
	Hypovolemic 3
	Euvolemic 5
Neurological signs and findings	Weakness 12

Continued

	Fever 7
	Headache 6
	Nausea 6
	Confusion 5
	Drowsiness 4
	Vomiting 3
	Disorientation 3
	Sedation 2
	Seizure 1
	Coma 1

Twenty-four patients had hyponatremia upon admission to the Emergency Department (ED), and 2 developed it while hospitalized. 23/26 patients had more than one cause of hyponatremia (heart failure in 10 patients and diuretic use in 20), except for 3 patients who had one each (acute kidney injury, psychogenic polydipsia, potomania). It is noteworthy that in 10 cases the main cause was iatrogenic (incorrect recommendation to take large amounts of tap water, in people whose kidneys were unable to manage it satisfactorily or administration of large amounts of hypotonic fluids intravenously). Of those who taken diuretics, all had used them for more than 6 months.

Serum laboratory findings (biochemistry), blood gases, lactate, findings of CT of the brain and fundoscopy are shown in Table 2.

Although all patients had serum sodium ≤120 mmol/L, 14 had serum sodium ≤115 mmol/L. Only in two cases could serum glucose explain part of the hyponatremia, since it was 44.4 and 22.4 mmol/L, respectively. Both cholesterol and triglycerides were not significantly increased to justify a decrease in serum sodium levels. Blood gases did not reveal any disorder that would explain cerebral edema, such as hypercapnia ($PaCO_2$ ranged from 19 to 44 mmHg) or acidosis (pH ranged from 7.28 to 7.56). Lactate was not significantly elevated in any patient, both before and after treatment.

CT revealed cerebral edema in 4 patients aged 58, 72, 98 and 42 years, with serum sodium levels 101, 98, 116 and 98 mmol/L, respectively (*i.e.* very low). Regarding fundoscopy findings, only 3/17 patients tested had papilla edema, aged 72, 62 and 98 (the first and last also had cerebral edema on CT).

The clinical symptoms were severe in 12 patients (coma, seizure, disorientation, confusion, shallowness). In two cases there were no symptoms of hyponatremia (both simply had low serum sodium levels [120 and 116 mmol/L] on routine examination, in the first due to heart failure, diuretics and ACE inhibitors, and in the second due to acute kidney injury).

24/26 patients were treated conservatively (fluid deprivation where required,

Table 2. Laboratory findings of patients (biochemical, radiological) and fundoscopy (CT = computer tomography).

	At the diagnosis of hyponatremia
Serum sodium (mmol/L) (n = 26)	112 ± 7.6 (range 98 - 120)
Glucose (mmol/L) (n = 26)	8.9 ± 8.2 (range 4.7 - 44.4)
Cholesterol (mmol/L) (n = 22)	5.0 ± 2.0 (range 2.0 - 8.3)
Triglycerides (mmol/L) (n = 22)	1.9 ± 1.0 (range 0.6 - 5.4)
Blood gases	
pH (n = 24)	7.47 ± 0.07
HCO_3^- (mmol/L) (n = 24)	24.3 ± 5.7
$PaCO_2$ (mmHg) (n = 22)	33.2 ± 6
Blood lactates (mmol/L) (n = 22)	1.7 ± 1.3
CT brain (n = 14)	Cerebral edema 4
	Normal findings (not edema) 10
Fundoscopy (n = 17)	Papilla edema 3
	Normal fundus 13
	Cataract 1

discontinuation of drugs responsible for hyponatremia, administration of diuretics [in hypervolemic cases], administration of NaCl 0.9% intravenously in hypovolemic cases and 3.0% in most cases for the clinical findings from the central nervous system, in doses depending on the case) and two with hemofiltration (3 sessions of 18 L of substitution fluid for first patient, and 4 sessions of 18 L for the second). The duration of hospitalization ranged from 4 - 10 days (mean ± SD = 6.3 ± 1.6, with a median value of 6 days). Of the 26 patients, 2 died (the first, aged 80, from uncontrolled upper gastrointestinal bleeding in the context of acute kidney injury, and the second, who was alcoholic, from pontine myelinolysis, despite the fact that the change in his sodium during treatment never exceeded 8 - 10 mmol/L over 24 hours), 18 improved and 6 recovered completely.

4. Discussion

The incidence of hyponatremia in hospitalized patients reaches up to 30% of [7]. Many noted an increased incidence among women compared to men, in percentages ranging from 71% [8] to 82.7% [9]. Others found that women with hyponatremia were approximately 2 [10] or even 3 times more in comparison to men [9]. We also agree with these results, as we found that 69.2% of our patients were women.

The reason why more hypertensive women experience hyponatremia from diuretics than men is because more of them use these drugs [11] and because women are more symptomatic than men for a similar degree of hyponatremia (they are

diagnosed more often than men) [12]. Estrogens also inhibit the activity of Na^+-K^+-ATPase, which is responsible for the adaptive removal of sodium from brain cells in cases of hyponatremia with cerebral edema. This means that when estrogen is present, this pump does not work, resulting in women being more susceptible to cerebral edema from hyponatremia [1] [13]. Vasopressin (ADH) also facilitates the movement of water into brain cells, independent of the effect of hyponatremia.

Regarding the role of age, it is emphasized that with aging process, physiological changes occur in the regulation of water balance, so that elderly individuals become more susceptible to the occurrence of hyponatremia. Many diseases that cause inappropriate ADH secretion are also common in the elderly [14] [15], such as vascular lesions, trauma, tumors, infections, schizophrenia and psychogenic polydipsia. In addition, the elderly often use ACE inhibitors, which are associated with hyponatremia.

Etiologically, another key factor in causing and worsening hyponatremia is the administration of hypotonic fluids (oral or intravenous), which is found in 78% of elderly people living in institutions [16], as well as the intake of diuretics (20 of our patients [77%] were taking diuretics). Prostaglandins are also known to antagonize the action of ADH in the collecting tubules and reduce the osmotic force of water reabsorption [17], whose synthesis in the elderly is reduced, resulting in the above effects on urine production.

Thiazides predispose to hyponatremia through a variety of mechanisms including increased urinary sodium loss, non-osmotic stimulation of ADH secretion, their effect on the maximum diluting capacity of the kidneys [18], and through a decrease in glomerular filtration rate and increase in proximal tubular water reabsorption. Recently, it has been suggested that the pathogenesis of thiazide-induced hyponatremia involves overexpression of aquaporin-2 in the collecting duct (and thus increased water reabsorption) in patients taking them [19]. Of course, the effect of the amount of salt ingested (which is usually limited in such cases) and the greater amount of water ingested should not be forgotten [20].

Of course, in the elderly taking thiazides, the amount of water consumed also plays a role in the occurrence of hyponatremia, which should be reduced [21], and the use of non-steroidal anti-inflammatory drugs should also be limited. At the same time, it is worth noting that hospitalized patients have and other causes that cause increased ADH levels (nausea, vomiting, taking diuretics, pain, stress, postoperative periods, pneumonia, asthma, meningitis, encephalitis, neoplasms, etc.), due to which any administration of hypotonic fluids leads to hyponatremia [22].

In a prospective study of 100 hospitalized elderly patients, hyponatremia (serum sodium of 109 - 129 mmol/L) was iatrogenic in 30% [23]. In a study of 35 patients with serum sodium < 125 mmol/L, the cause of hyponatremia was iatrogenic in 62.8% of cases (37.1% from diuretics and 22.8% from hypotonic saline infusion) [24]. Similarly, others found iatrogenic causes of hyponatremia (intravenous fluids or diuretics), with serum sodium < 130 mmol/L in 73% of patients.

Also, out of 44 hospitalized patients with serum sodium < 125 mmol/L, the cause of hyponatremia was iatrogenic in 44% of cases [25]. Finally, out of 108 patients who had hyponatremia (serum sodium < 125 mmol/L), 75.3% had more than one cause [4], as we also noted (23/26, 88.5%). However, beer, which usually contains less than 5 mmol/L sodium, is also a cause of hyponatremia (in cases of potomania) [26], as happened to one of our patients.

Euvolemic hyponatremia is caused by inappropriate ADH secretion, potomania, psychogenic polydipsia and hypothyroidism. Patients with thiazide-induced hyponatremia are between euvolemic and hypovolemic type [27]. Of our patients receiving thiazides, 6 had hypervolemic, 2 hypovolemic and 5 euvolemic hyponatremias. In 35 hyponatremic patients, 48.5% were euvolemic, 42.8% hypervolemic, and 8.5% hypovolemic [24], while others found that 34% of their patients were euvolemic, 19% hypervolemic, and 17% hypovolemic [10]. In our material, most had hypervolemic (69.2%) and the few had hypovolemic hyponatremia (11.5%).

Usually, chronic and moderate hyponatremia is asymptomatic [28] or is accompanied by mild to moderate symptoms. This is because in these cases the brain volume and intracranial pressure do not increase significantly (since the brain is given time to adapt), as was characteristically seen in most of our patients on brain CT and fundoscopy. Thus, in the study by Misra and Mansharamani, 67% were asymptomatic or with non-specific symptoms [23].

Therefore, since symptoms are not detected when serum sodium is >120 mmol/L, we set this as the limit in our study. Thus, Sharabi *et al.* found in 180 patients with hyponatremia (of whom 86 had serum sodium less than 120 mmol/L), 17.2% were asymptomatic [9]. Our symptomatic patients (24/26) had basically neurological clinical signs and from this the suspicion and diagnosis of hyponatremia was established in most of them, while only 2/26 (7.7%) were asymptomatic. Others also agree that the most frequent clinical manifestations of severe hyponatremia are neurological [4] and are due to cerebral edema. And of course, in most cases when serum sodium levels are significantly low, e.g. with sodium around 101 mmol/L, symptoms range from confusion to coma, as we have also found.

The CT for diagnosis of cerebral edema is usually difficult to impossible [29]. In fact, there is a better correlation between brain CT findings and clinical symptoms than with serum sodium levels. The reason that severe hyponatremia often has unexpectedly mild symptomatology, especially in elderly patients, is because their relatively atrophic brain provides space for expansion (increase in volume due to cerebral edema) within its cranial vault, in contrast to younger individuals where this space is minimal [30].

In every patient with hyponatremia, before any therapeutic intervention, the following questions must be answered: a) is he symptomatic, b) is the duration of hyponatremia known, c) is he at risk of developing neurological complications? And it is also necessary for each such patient to clearly classify him as hypovolemic, hypervolemic or euvolemic hyponatremia. These parameters will deter-

mine the type and speed of treatment. Despite the very low sodium levels of our patients, only two needed to be treated with hemofiltration and the rest conservatively (discontinuation of the causative factor when it was known and obvious, restriction of fluid intake where necessary, administration of 0.9% NaCl serum in hypovolemic cases, administration of 3.0% NaCl serum in every symptomatic patient with symptoms from the central nervous system, etc.).

Osmotic demyelination is a serious complication of hyponatremia treatment and appears to occur when serum sodium is corrected rapidly in cases of chronic hyponatremia. However, in several groups of patients with chronic hyponatremia, demyelination occurs more frequently and more easily, such as alcoholics (as was the case in one of our patients), malnourished, and patients with hypokalemia or liver disease. These groups are particularly susceptible to the occurrence of myelinolysis during the treatment of hyponatremia [31] and it can even occur in cases where sodium does not enhance by more than 10 mmol/L/24 hours, as was the case in our patient.

CT is diagnostic in myelinolysis, where pontine and extrapontine lesions are usually found symmetrically (hypodense areas) [32], however it should be carried out after 6 - 10 24 hours from the onset of the episode [28]. This was the reason why nothing pathological was found in our patient on the first CT it was done 3 days after the clinical manifestation of myelinolysis, while the next CT performed after a total of 12 days from its hospitalization.

The duration of hospitalization of our patients was from 4 - 10 days, although according to the literature, severe hyponatremia (serum sodium levels less than 120 mmol/L) requires much longer hospitalization days [33].

Regarding prognosis, the etiology of hyponatremia is the most important prognostic factor, but not the absolute serum sodium level [4]. Thus, the prognosis of thiazide-induced hyponatremia is very good, even when severe [34] and most patients recover completely. Mortality is <10%, although some have noted it to be higher [4] and most deaths are not due to hyponatremia, but to the underlying disease [34] [35], as was the case in our patients who died.

In a review of hyponatremia cases from 1975 to 2006 (n = 344), the mortality was 44% [1]. In particular, in the study by Clayton *et al.* that included 108 hospitalized patients, aged 28 - 95 years (median 75 years), with severe hyponatremia (<125 mmol/L), 20% died during their admission and by the end of follow-up [4], while it is worth noting that hospital-acquired hyponatremia has an increased mortality [36]. Accordingly, 17.1% of Farooqui & Ahmed's 35 patients died [24], as did 33% of Tolias' patients [8]. Finally, the mortality of Rahil *et al.*'s patients was 11.3% (6/53), which is rather low, considering that 22 (41.6%) had serum sodium < 115 mmol/L [10]. In our study, the mortality was 7.7%, *i.e.* particularly low, even though more than half (14/26) had serum sodium < 114 mmol/L. Also, the mortality of hyponatremia is increased in patients in whom treatment is delayed or in those with serious underlying diseases.

It is concluded that severe hyponatremia: 1) is very often iatrogenic and of phar-

maceutical etiology, 2) usually affects elderly individuals and mainly women, 3) has symptoms that mainly concern the central nervous system, 4) the diagnosis is usually made mainly from the clinical findings and the patient's history, 5) the findings of CT and fundoscopy are not particularly helpful in the diagnosis, since they are rarely positive for cerebral edema, 6) special attention is needed during the diagnosis, classification and treatment of the disease and 7) early diagnosis and treatment contribute to a good prognosis of the patient's.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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