Acta Clin Croat **2026**, vol. **65**, no. **2**: 414-424

Acute Severe Euvolemic Hyponatremia Caused by Segmental Pulmonary Thromboembolism and Complicated by COVID-19 Infection: A Case Report

Bojan Mitrović[1,3,5], Milan Obradović[2], Esma R. Isenović[2], Zoran Gluvić[1,3,5], Milena Lacković[1,5], Sandra Pekić Djurdjević[4,5] and Milan Petakov[4,5]

[1] Department of Endocrinology and Diabetes, Zemun Clinical Hospital, Belgrade, Serbia;
[2] Department of Radiobiology and Molecular Genetics, Vinča Institute of Nuclear Sciences, National Institute of the Republic of Serbia, University of Belgrade, Belgrade, Serbia;
[3] Medicine Intensive Care Unit, Zemun Clinical Hospital, Belgrade, Serbia;
[4] Department of Endocrinology, Diabetes and Metabolic Diseases, Division of Neuroendocrinology, Clinical Center of Serbia, Belgrade, Serbia;
[5] School of Medicine, University of Belgrade, Belgrade, Serbia

CORRESPONDENCE TO Milan Obradović, Vinča Institute of Nuclear Sciences, National Institute of the Republic of Serbia, University of Belgrade, Department of Radiobiology and Molecular Genetics, P.O.Box 522, 11000 Belgrade, Serbia
obradovicmilan@hotmail.com

RECEIVED December 16, 2021
ACCEPTED July 11, 2022

DOI 10.20471/acc.2026.65.02.19

SUMMARY

Hyponatremia is frequently encountered in clinical practice. Euvolemic hyponatremia (syndrome of inadequate antidiuresis, SIAD) represents diagnostic and management challenges regarding etiology unraveling, gradual acute substitution, and later causal treatment. Here we present a case of severe SIAD caused by segmental pulmonary thromboembolism and manifested as acute psychosis. COVID-19 complicated the course of the disease acquired in hospital settings. By simultaneous treatment of pulmonary thromboembolism, gradual substitution of hyponatremia, and treatment of COVID-19, the patient completely recovered biochemically and mentally. Even experienced clinicians are confused about diverse clinical manifestation of SIAD. Attentive correction of hyponatremia and treatment of its etiology are the mainstays of SIAD management.

KEYWORDS

Hyponatremia; Hypertonic saline; Pulmonary thromboembolism

Introduction

Hyponatremia is an electrolyte disorder that is often encountered in clinical practice. It is defined as a condition where the sodium level is <135 mEq/L. Sodium ions, as the main extracellular cations, significantly determine osmolarity of the plasma. Therefore, the disorder of serum sodium concentration is at the same time a disorder of plasma osmolarity. Normal values of sodium concentration in adult serum are 137-146 mmol/L[1-3].

The first step in evaluating hyponatremia is assessing patient volume status (presumably by clinical estimation of hypervolemia, normovolemia, or hypovolemia). The syndrome of inadequate antidiuresis (SIAD) is a syndrome of euvolemic hyponatremia, characterized by permanently altered secretion of antidiuretic hormone (ADH) and excretion of concentrated urine (>100 mOsm/kg) despite serum hypoosmolarity (275 mOsm/kg), natriuresis >30 mmol/day, and absence of renal, adrenal, or thyroid insufficiency[4, 5]. The causes of SIAD are illustrated in Figure 1, while differential diagnosis of hyponatremia is shown in Table 1[3, 6, 7]. Hyponatremia usually becomes clinically manifest with a sodium level of <125 mmol/L[8]. It is most commonly presented by neurological and digestive symptoms such as headache, nausea, vomiting, confusion, incontinence, aggression, arrhythmias, and, less often, convulsions and coma[8-10]. Treatment of

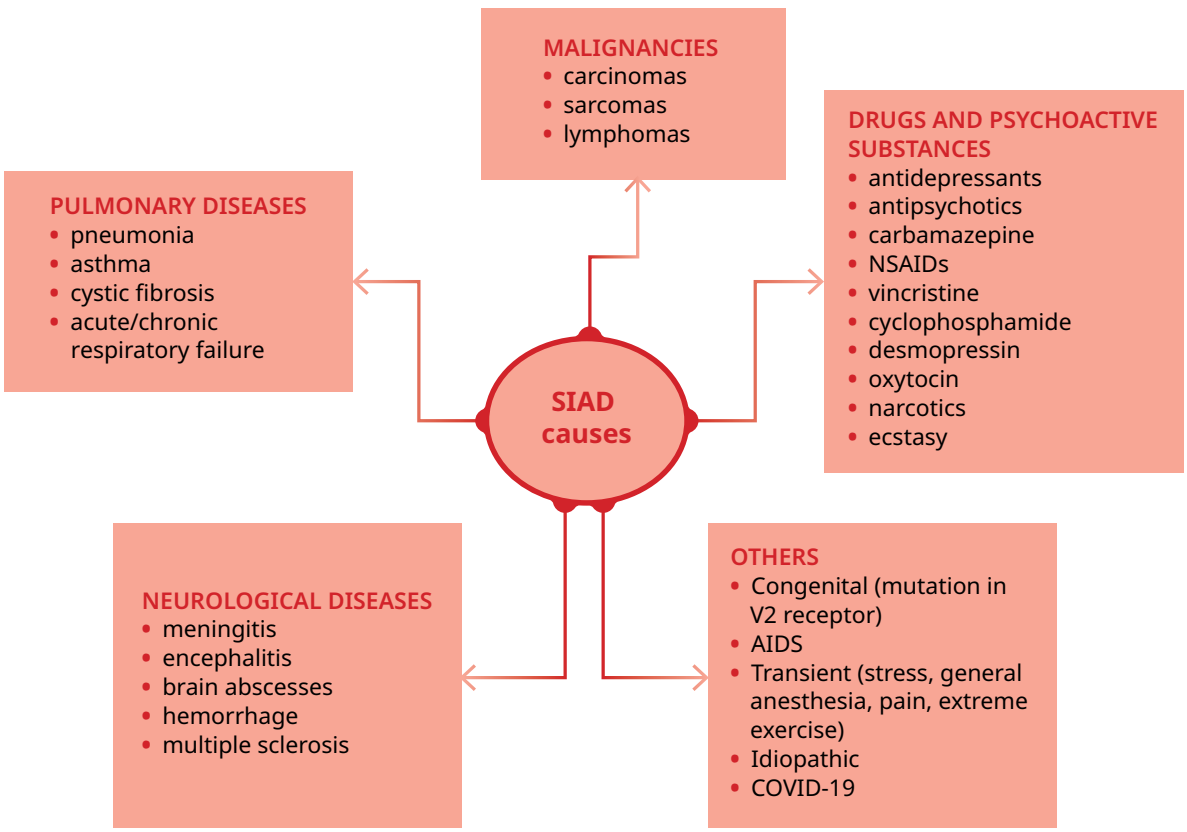

Fig. 1. Causes of the syndrome of inadequate antidiuresis.
AIDS = acquired immune deficiency syndrome; SIAD = syndrome of inadequate antidiuresis; COVID-19 = coronavirus disease caused by SARS-CoV-2 virus; NSAIDs = non-steroidal anti-inflammatory drugs

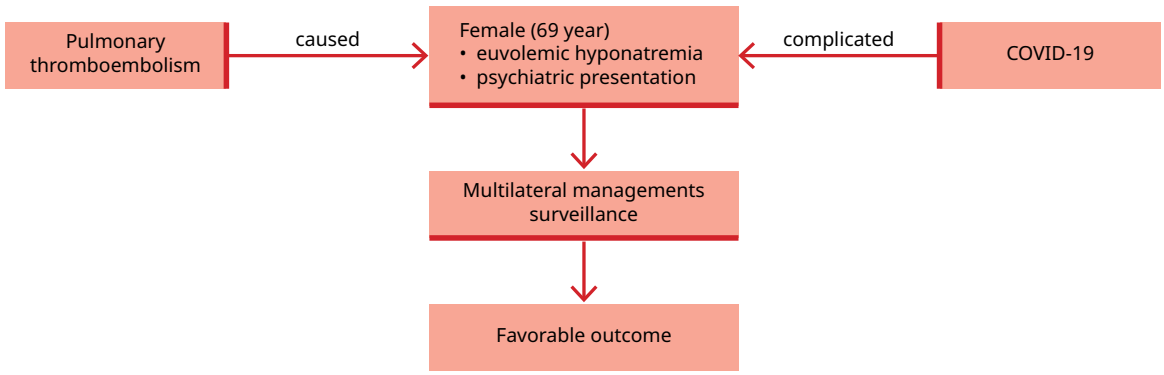

Fig. 2. Pulmonary thromboembolism-induced syndrome of inadequate antidiuresis complicated by COVID-19.

SIAD includes careful correction of hyponatremia with treatment of the causative disease[8, 11-14]. One should have in mind that too rapid correction of hyponatremia can lead to pontine myelinolysis[15].

This article presents a patient with euvolemic hyponatremia caused by segmental pulmonary thromboembolism (PTE) complicated by COVID-19 (Fig. 2).

Case report

A 69-year-old female was initially treated at the Department of Endocrinology, Division of Neuroendocrinology, Clinical Center of Serbia (CCS), due to severe hyponatremia (101 mmol/L) with psychiatric presentation (psychotic behavior, hallucinations). During hospital stay, the patient came in contact with a COVID-19 positive patient. After a polymerase chain reaction (PCR) SARS-CoV2 test, which was positive, she was transferred to the Zemun Clinical Hospital, a COVID-19 center, to continue treatment of hyponatremia and COVID-19.

According to the manufacturer's instructions, hematologic and biochemical analyses were

Table 1. Differential diagnosis of hyponatremia

Hypovolemic	**Euvolemic**
Osmotic diuresis	SIAD
Salt-losing nephropathy	Lung diseases
Diuretic use	Intracranial diseases
Renal volume loss	Primary hypoadrenalism
Cerebral salt loss	Hypothyroidism
Diarrhea	Psychogenic polydipsia
Vomiting	Non-psychogenic polydipsia
Burns	Water intoxication
Excessive sweating	Excessive infusion treatment
Extrarenal volume loss	After transurethral prostatectomy
Hypervolemic	During early postoperative period
Cirrhosis with ascites	Vomiting and nausea
Congestive heart failure	Severe pain or emotional stress
Nephrotic syndrome, chronic renal failure	Medications including general anesthesia

SIAD = syndrome of inadequate antidiuresis

Table 2. Laboratory findings during hospital stay at the Department of Endocrinology, Clinical Center of Serbia (on admission and discharge)

Hematologic analysis	Biochemistry	Hemostasis parameter	Blood gases	Urinalysis
WBC (x10^9/L) 7.6...2.5	BG (mmol/L) 6.8...4.8	D-dimer (mg/L) 8.11... 2.03	pH 7.52	Specific weight 1.018
RBC (x10^{12}/L) 3.84...3.9	CRP (mg/L) 6.5...2.6	PT (s) 12... 31.1	PaCO2 (kPa) 4.73	pH 6
Hb (g/L) 117...118	Urea (mmol/L) 4.0...3.9	INR 0.89...2.42	PaO2 (kPa) 12	Microscopy: 10-15 RBC, plenty of bacteria
MCV (fL) 95...94	Creatinine (µmol/L) 72...64	aPTT (s) 24.4...36.9	HCO3 (mmol/L) 29	
Ht (L/L) 0.365...0.367	Total proteins (g/L) 62	Fibrinogen (g/L) 3.7...3.3	SaO2 (%) 98%	Natriuresis (mEq/D) 13...14
Plt (x10^9/L) 304...344	Albumin (g/L) 45	Antithrombin 106%		Kaliuresis (mEq/D) 25...42
ESR (mm/h) 6/	Chol (mmol/L) 4.92	Protein C 132%		Chloriuresis (mEq/D) 23...24
	HDL-Chol (mmol/L) 1.57	Factor XI 137%		Urea (mmol/D) 512
	LDL-Chol (mmol/L) 3.0	Factor X 1		Creatinine (mmol/D) 17.9
	TG (mmol/L) 0.77	Factor IX 140%		Morning urine osmolality 167...551 mOsm/kg
	Iron (µmol/L) 10.1...11.7	Factor VIII 149%		
	Ferritin (µg/L) 175...155			
	TIBC (µmol/L) 45			
	Sodium (mmol/L) 103...108...124 ...130...139...144			24h urine osmolality 573 mOsm/kg
	Potassium (mmol/L) 3.2...3.8			Bacteriology: *E. coli*
	Chloride (mmol/L) 65...108			
	Calcium (mmol/L) 2.20...2.21			
	Phosphate (mmol/L) 0.71...1.24			
	AST (U/L) 39			
	ALT (U/L) 20			
	ALP (U/L) 64			
	GGT (U/L) 20			
	Plasma osmolarity (mmol/L) 218... 276			

WBC = white blood cell count; RBC = red blood cell count; Hb = hemoglobin; Ht = hematocrit; Plt = platelet count; ESR = erythrocyte sedimentation rate; BG = blood glucose; CRP = C-reactive protein; Chol = cholesterol; HDL = high-density lipoprotein; LDL = low-density lipoprotein; TG = triglycerides; AST = aspartate aminotransferase; ALT = alanine transaminase; ALP = alkaline phosphatase; GGT = gamma-glutamyl transferase; PT = prothrombin time; aPTT = activated partial thromboplastin time; INR = international normalized ratio; PaCO2 = partial pressure of carbon dioxide in arterial blood; PaO2 = partial pressure of oxygen in arterial blood; HCO3 = bicarbonates; SaO2 = oxygen saturation in arterial blood

Table 3. Hormones, tumor markers, and immunology analyses during hospital stay at the Department of Endocrinology, Clinical Center of Serbia (on admission and discharge)

Hormone	Immunologic analysis	Tumor marker
TSH (μIU/mL) 0.7	ANA HEP2 IgG - nucleoplasm + homogeneous (1:40)	CA 125(U/mL) 37
FT4 (pmol/L) 20.9	IgG-AMA negative	CA 15-3 (U/mL) 18
FT3 (pmol/L) 2.64	IgG ANA negative	CA 19-9 (U/mL) 9
Cortisol (morning) (nmol/L) 486...497	IgG-ASMA negative	CEA (μg/L) 2.8
ACTH (pg/mL) 39.3	LKM1 Abs negative	Cyfra 21-1 (ng/mL) 3.2
LH (IU/L) 19.8	Antiparietal Abs positive	
FSH (IU/L) 54.6	IgG ACLA negative	
Prolactin (mIU/L) 492	IgM ACLA negative	
	ENA screen negative	

TSH = thyroid-stimulating hormone; FT4 = free thyroxin; FT3 = free triiodothyronine; ACTH = adrenocorticotropic hormone; LH = luteinizing hormone; FSH = follicular stimulating hormone; IgG = immunoglobulin G; ANA = antinuclear antibodies; AMA = anti mitochondrial antibodies; ASMA = anti smooth muscle antibodies; LKM1 = liver kidney microsomal 1 antibody; Abs = antibodies; ACLA = anticardiolipin antibodies; ENA = extractable nuclear antigen; CA = cancer antigen; CEA = carcinoembryonic antigen; Cyfra = cytokeratin-19 fragments

performed on Access-2 and DxC 800 Beckman Coulter automatic analyzers. Ultrasonographic examinations were performed on a Toshiba Xario device (Japan), using appropriate probes. Radiographic recordings were made on an AGFA DX-D 100+ device (Belgium, Germany). Computed tomography (CT) of organs and organ systems was performed on a Toshiba 128 slice (Japan), while magnetic resonance imaging (MRI) of the pelvis was done by use of the 1.5 Tesla Canon Titan (Malaysia).

Results

Tables 2 and 3 present the results of hematologic, biochemical, and hemostatic parameters and urinalyses performed at the Department of Endocrinology and Diabetes, CCS. No pathologic findings were observed in the hematologic analyses performed. Biochemical results revealed severe hyponatremia (101 mmol/L), hypochloremia (65 mmol/L), hypoosmolarity (218 mmol/L), as well as moderate hypokalemia (3.2 mmol/L). The other biochemical parameters observed, including the general and organ-specific tumor marker, were within the normal range. Spot-urine natriuresis was under 30 mmol/L. *Escherichia coli* (*E. coli*) was cultered in urine. Out of hemostatic parameters, elevated D-dimer (8.11 mg/L) was detected. The hypothalamic-pituitary-thyroid and hypothalamic-pituitary-adrenal axes maintained functional integrity, while the levels of gonadotropins were in the menopausal range. Apart from indirect immunofluorescence detected ANA HEP2 IgG homogeneous nucleoplasmic activity (1:40), other immunologic tests were without pathologic findings. To detect pathologic substrates causing hyponatremia, numerous imaging procedures were performed. Apart from the finding of a

mildly elevated right hemidiaphragm on the initial and repeated chest x-ray scans, no other pathologic findings were found on radiographic (cranial and breast x-rays), ultrasonographic (abdomen, pelvis, breast, Doppler scan of lower limb blood vessels) or MRI procedures (pelvis).

Due to elevation of the right hemidiaphragm, a CT scan of the chest and abdomen indicated contrast filling defects characterized as acute thromboembolism in the branches for the anterior segment of the upper left and right lobes, as well as branches for the apical segment of the upper right lobe and branches of the anterobasal and laterobasal segment of the lower left lobe. No other radiologic changes (i.e., consolidation, infiltration, or collapse) of the lungs were seen (Figs. 3 and 4).

Due to epidemiologic reasons (contact with a SARS CoV2 positive person), our patient was tested for PCR SARS CoV2, and the result was positive. According to the COVID-19 rules of the health care authorities, the patient was transferred to the COVID-19 Zemun Clinical Hospital for continued monitoring of electrolyte metabolism and treatment of PTE and COVID-19. Throughout the COVID-19 hospital stay, the patient was afebrile with no respiratory complaints, stable vital parameters, and mentally inconspicuous. Mild leukopenia and anemia, as well as moderate hypokalemia were verified by laboratory findings. Sodium values were stable throughout the stay in the COVID-19 center, without the need of substitution. She was treated with ceftriaxone, multivitamin therapy, and low molecular weight heparin. After the inconsistent INR values were observed during warfarin administration, direct oral anticoagulant (apixaban 10 mg/day) was introduced.

She was discharged from the COVID-19 Zemun Clinical Hospital in a good general and mental condition, afebrile, well-oxygenated (ambient SO2 99%), with normal sodium levels, and satisfying other laboratory findings listed in Table 4.

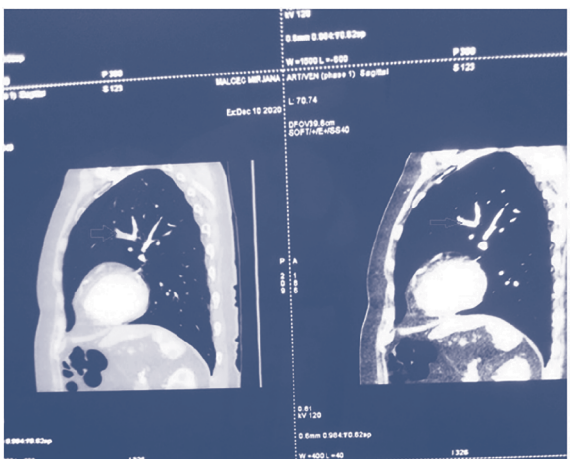

Fig. 3. Multi-slice computer tomography pulmoangiography 1 (arrows indicate pulmonary thromboembolism in the branches of the anterior segment of the upper left and right lobes).

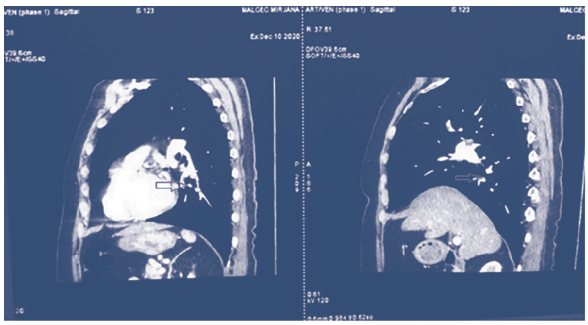

Fig. 4. Multi slice computed tomography pulmoangiography 2 (arrows indicate pulmonary thromboembolism in the branches of anterobasal and laterobasal segment of the lower left lobe).

Table 4. Laboratory findings during hospital stay in COVID-19 Zemun Clinical Hospital (on admission and discharge)

Hematologic analysis	Biochemistry	Hemostasis parameter
WBC (x10^9/L) 2.5...3	BG (mmol/L) 5.1	D-dimer (mg/L) 1...0
RBC (x10^{12}/L) 3.49...3.72	Bilirubin direct (µmol/L) 1.0	aPTT (s)38.7...36
Hb (g/L) 107...114	Bilirubin total (µmol/L) 5.6	PT (s) 40.4...26.5
Ht (L/L) 0.326...0.348	Total proteins (g/L) 54	INR 3.25...2.08
Plt (x10^9) 263...183	Albumin (g/L) 35	TT (s) 22.2...18
	CRP (mg/L) 1.5...6.0	Anti Xa (U/mL) 0.3
	Potassium (mmol/L) 3.4...3.5	
	Sodium (mmol/L) 143...141	
	Calcium (mmol/L) 2.08	
	Chloride (mmol/L) 106...106	
	Phosphate (mmol/L) 1.36	
	Magnesium (mmol/L) 0.93	
	Ferritin (µg/L) 172	
	AST (U/L) 34	
	ALT (U/L) 43	
	GGT (U/L) 20	
	CK (U/L) 86	
	Urea (mmol/L) 2.9...4.3	
	Creatinine (µmol/L) 68...59	

WBC = white blood cell count; RBC = red blood cell count; Hb = hemoglobin; Ht = hematocrit; Plt = platelet count; BG = blood glucose; CRP = C-reactive protein; AST = aspartate aminotransferase; ALT = alanine transaminase; GGT = gamma-glutamyl transferase; CK = creatine kinase; PT = prothrombin time; aPTT = activated partial thromboplastin time; INR = international normalized ratio; TT = thrombin time

Discussion

Hyponatremia (Na <135 mmol/L) is among the most common electrolyte disturbances in clinical practice and is recorded in 15%-30% of hospitalized patients[2]. If hyponatremia persists even after repeated measurement, its etiology must be clarified.

In addition to often being a humoral manifestation of malignancy, it is a well-known marker of neurohumoral activation in patients with left ventricular heart failure. Additionally, it reflects the non-osmolar release of ADH due to activation of the sympathetic and renin-angiotensin-aldosterone systems[16, 17].

Neurohumoral activation has also been demonstrated in patients with pulmonary hypertension and occurs in proportion to the degree of right ventricular dysfunction[18, 19]. Therefore, hyponatremia may indicate neurohumoral activation in the context of acute pulmonary hypertension, associated with PTE, and consequent right heart dysfunction, a known prognostic factor in patients with PTE[20]. A prospective study in patients with chronic pulmonary arterial hypertension showed a significant association between hyponatremia, the presence of right ventricular dysfunction, and a shorter survival rate[21]. Current recommendations advise conducting detailed echocardiographic and biohumoral examinations (i.e., measurement of brain natriuretic peptide)[22]; however, our patient did not perform them due to circumstances related to COVID-19, as well as the favorable clinical course.

Table 1 shows some of the causes of euvolemic hyponatremia. It is sometimes ominous, as it occurs due to altered ADH secretion or the effect on its receptors in the tubules, as part of malignant diseases, most often in the lungs. It is also known that vascular lesions and hereditary or acquired thrombophilia can often cause PTE. Likewise, hyperhomocysteinemia is a risk factor for venous thromboembolism because it interferes with the activation of coagulation factor Va by activating protein C. This factor also increases tissue factor expression and suppresses heparin sulfate, an endogenous anticoagulant[23-26]. Unlike malignancy-induced ADH secretion, which lasts as long as there is malignancy, the transient nature of presented hyponatremia that responds well to substitherapy supports the benign nature of the cause of hyponatremia, PTE[4, 6]. As one of the SIAD criteria, the absence of elevated natriuresis could be explained by shorter duration of hyponatremia-causative disease (acute PTE).

The clinical presentation spectrum of hyponatremia is impressive. The patients with hyponatremia are most often seen in traumatology, orthopedics, neurosurgery, and neurology. However, acute hyponatremia is most often accompanied by severe neurological disorders, often coma, primarily caused by brain edema. If hyponatremia is chronic, adaptive mechanisms allow patients to adapt gradually to low sodium levels. Likewise, rapid correction of hyponatremia can lead to central pontine myelinolysis, which dramatically affects the disease outcome[4, 27, 28].

Regarding COVID-19, the real prevalence of hyponatremia in patients with lung affection, more infectious than vascular, is still unknown. Based on clinical experience, the prevalence of hyponatremia in COVID-19-associated pneumonia appears to be relatively low. There still are insufficient data on the impact of complicated pneumonia and massive PTE on SIAD development. However, it seems that the most severe forms of the disease contribute to the development of hyponatremia, especially in patients who received mechanical ventilation, which itself, especially when applied with high positive end expiration pressures (PEEP >10 cm H_2O), contributes to hyponatremia[21, 28-30]. Recommendations for treating hyponatremia in COVID-19 do not differ from previous recommendations, i.e., treatment of the etiology of hyponatremia with its gradual and careful correction. Recommendations for using vaptans do not differ from the currently available ones[4, 28, 31-34].

Clinicians should always consider hyponatremia etiologically and correct it appropriately. Vascular lung lesions may be associated with hyponatremia but also with COVID-19, which in itself is a risk factor for PTE.

ACKNOWLEDGMENT This work was funded by the Ministry of Education, Science and Technological Development of the Republic of Serbia (contract no. 451-03-33/2026-03/200017).

References

1. Al-Salman J, Kemp D, Randall D. Hyponatremia. West J Med. 2002;176(3):173-6. https://doi.org/10.1136/ewjm.176.3.173
2. Decaux G. The syndrome of inappropriate secretion of antidiuretic hormone (SIADH). Semin Nephrol. 2009;29(3):239-56. https://doi.org/10.1016/j.semnephrol.2009.03.005
3. Ellison DH, Berl T. Clinical practice. The syndrome of inappropriate antidiuresis. N Engl J Med. 2007;356(20):2064-72. https://doi.org/10.1056/NEJMcp066837
4. Spasovski G, Vanholder R, Allolio B, Annane D, Ball S, Bichet D, *et al.* Clinical practice guideline on diagnosis and treatment of hyponatraemia. Eur J Endocrinol. 2014;170(3):G1-47. https://doi.org/10.1530/EJE-13-1020
5. Janicic N, Verbalis JG. Evaluation and management of hypo-osmolality in hospitalized patients. Endocrinol Metab Clin North Am. 2003;32(2):459-81, vii. https://doi.org/10.1016/s0889-8529(03)00004-5
6. Królicka AL, Kruczkowska A. Hyponatremia in infectious diseases – a literature review. Int J Environ Res Public Health. 2020;17(15):5320. https://doi.org/10.3390/ijerph17155320
7. Verbalis JG, Goldsmith SR, Greenberg A, Schrier RW, Sterns RH. Hyponatremia treatment guidelines 2007: expert panel recommendations. Am J Med. 2007;120(11 Suppl 1):S1-21. https://doi.org/10.1016/j.amjmed.2007.09.00
8. Ball SG, Barber T, Baylis PH. Tests of posterior pituitary function. J Endocrinol Invest. 2003;26(7 Suppl):15-24.
9. Upadhyay A, Jaber BL, Madias NE. Incidence and prevalence of hyponatremia. Am J Med. 2006;119(7 Suppl 1):S30-5. https://doi.org/10.1016/j.amjmed.2006.05.005
10. Schrier RW. Body water homeostasis: clinical disorders of urinary dilution and concentration. J Am Soc Nephrol. 2006;17(7):1820-32. https://doi.org/10.1681/ASN.2006030240
11. Chen S, Jalandhara N, Batlle D. Evaluation and management of hyponatremia: an emerging role for vasopressin receptor antagonists. Nat Clin Pract Nephrol. 2007;3(2):82-95. https://doi.org/10.1038/ncpneph0401
12. Adrogué HJ, Madias NE. Hyponatremia. N Engl J Med. 2000;342(21):1581-9. https://doi.org/10.1056/NEJM200005253422107
13. Decaux G, Soupart A, Vassart G. Non-peptide arginine-vasopressin antagonists: the vaptans. Lancet. 2008;371(9624):1624-32. https://doi.org/10.1016/S0140-6736(08)60695-9
14. Gross P. The vaptans: just landed. But how was the trip? Nephrol Dial Transplant. 2010;25(5):1367-8. https://doi.org/10.1093/ndt/gfq102
15. Gankam Kengne F, Nicaise C, Soupart A, Boom A, Schiettecatte J, Pochet R, *et al.* Astrocytes are an early target in osmotic demyelination syndrome. J Am Soc Nephrol. 2011;22(10):1834-45. https://doi.org/10.1681/ASN.2010111127
16. Schrier RW, Abraham WT. Hormones and hemodynamics in heart failure. N Engl J Med. 1999;341(8):577-85. https://doi.org/10.1056/NEJM199908193410806
17. Levine TB, Francis GS, Goldsmith SR, Simon AB, Cohn JN. Activity of the sympathetic nervous system and renin-angiotensin system assessed by plasma hormone levels and their relation to hemodynamic abnormalities in congestive heart failure. Am J Cardiol. 1982;49(7):1659-66. https://doi.org/10.1016/0002-9149(82)90243-0
18. Velez-Roa S, Ciarka A, Najem B, Vachiery JL, Naeije R, Van De Borne P. Increased sympathetic nerve activity in pulmonary artery hypertension. Circulation. 2004;110(10):1308-12. https://doi.org/10.1161/01.CIR.0000140724.90898.D3
19. Nootens M, Kaufmann E, Rector T, Toher C, Judd D, Francis GS, *et al.* Neurohormonal activation in patients with right ventricular failure from pulmonary hypertension: relation to hemodynamic variables and endothelin levels. J Am Coll Cardiol. 1995;26(7):1581-5. https://doi.org/10.1016/0735-1097(95)00399-1
20. Sanchez O, Trinquart L, Colombet I, Durieux P, Huisman MV, Chatellier G, *et al.* Prognostic value of right ventricular dysfunction in patients with haemodynamically stable pulmonary embolism: a systematic review. Eur Heart J. 2008;29(12):1569-77. https://doi.org/10.1183/16000617.0120-2022

21. Forfia PR, Mathai SC, Fisher MR, Housten-Harris T, Hemnes AR, Champion HC, *et al.* Hyponatremia predicts right heart failure and poor survival in pulmonary arterial hypertension. Am J Respir Crit Care Med. 2008;177(12):1364-9. https://doi.org/10.1164/rccm.200712-1876OC
22. Konstantinides SV, Meyer G, Becattini C, Bueno H, Geersing GJ, Harjola VP, *et al.* 2019 ESC Guidelines for the diagnosis and management of acute pulmonary embolism developed in collaboration with the European Respiratory Society (ERS). Eur Heart J. 2020;41(4):543-603. https://doi.org/10.1093/eurheartj/ehz405
23. Oliveira LR, Fonseca JR. Simultaneous pulmonary thromboembolism and superior mesenteric venous thrombosis associated with hyperhomocysteinemia secondary to pernicious anemia-induced vitamin B12 deficiency. Hematol Transfus Cell Ther. 2018;40(1):79-81. https://doi.org/10.1016/j.htct.2017.11.004
24. Huang HL, Ting MS, Olszyna DP, Quek ST, Sinha AK, Loi HY. Localization of *Staphylococcus lugdunensis* clavicular osteomyelitis using FDG-PET/CT. Am J Med. 2016;129(3):e9-11. https://doi.org/10.1016/j.amjmed.2015.10.040
25. Üzmezoğlu B, Özdemir L, Hatipoğlu ON, Özdemir B, Çakır Edis E. A case of massive pulmonary embolism due to diabetic ketoacidosis and hyperhomocysteinemia. Euras J Pulmonol. 2014;16(2):124-6. https://doi.org/10.5152/ejp.2014.69345
26. Kostić A, Ilić J, Stojanović N, Janković R, Nikolov V, Živković S. Elevated serum C-reactive protein levels in COVID-19 patients with sciatica: implications for prognosis and treatment strategies. Acta Clin Croat. 2024;63(1):149-56. https://doi.org/10.20471/acc.2024.63.01.18
27. Sterns RH, Riggs JE, Schochet SS, Jr. Osmotic demyelination syndrome following correction of hyponatremia. N Engl J Med. 1986;314(24):1535-42. https://doi.org/10.1056/NEJM198606123142402
28. Christ-Crain M, Hoorn EJ, Sherlock M, Thompson CJ, Wass JAH. Endocrinology in the time of COVID-19: management of diabetes insipidus and hyponatraemia. Eur J Endocrinol. 2020;183(1):G9-G15. https://doi.org/10.1530/EJE-21-0596
29. Robertson GL. Regulation of arginine vasopressin in the syndrome of inappropriate antidiuresis. Am J Med. 2006;119(7 Suppl 1):S36-42. https://doi.org/10.1530/EJE-21-0596
30. Balık A, Yagci B, Balik R, Uncu UY. Are COVID-19 pneumonia findings different between comorbid and non-comorbid patients? The high resolution computed tomography features of the 108 follow-up patients. Acta Clin Croat. 2024;63(2):260-74. https://doi.org/10.20471/acc.2024.63.02.2
31. Smith DM, Mckenna K, Thompson CJ. Hyponatraemia. Clin Endocrinol (Oxf). 2000;52(6):667-78. https://doi.org/10.1046/j.1365-2265.2000.01027.x
32. Palmer BF, Gates JR, Lader M. Causes and management of hyponatremia. Ann Pharmacother. 2003;37(11):1694-702. https://doi.org/10.1345/aph.1D105
33. Brunner JE, Redmond JM, Haggar AM, Kruger DF, Elias SB. Central pontine myelinolysis and pontine lesions after rapid correction of hyponatremia: a prospective magnetic resonance imaging study. Ann Neurol. 1990;27(1):61-6. https://doi.org/10.1002/ana.410270110
34. Atik D, Kaya HB. Evaluation of the relationship of MPV, RDW and PVI parameters with disease severity in COVID-19 patients. Acta Clin Croat. 2021;60(1):103-14. https://doi.org/10.20471/acc.2021.60.01.15

SAŽETAK

Akutna teška euvolemijska hiponatremija uzrokovana segmentnom plućnom tromboembolijom i kompliciranа infekcijom COVID-19: prikaz slučaja

Bojan Mitrović, Milan Obradović, Esma R. Isenović, Zoran Gluvić, Milena Lacković, Sandra Pekić Djurdjević i Milan Petakov

Hiponatremija se često susreće u kliničkoj praksi. Euvolemijska hiponatremija (sindrom nedostatne antidiureze, *syndrome of inadequate antidiuresis*, SIAD) predstavlja izazove u dijagnostici i liječenju, kako u pogledu otkrivanja etiologije i postupne akutne supstitucije, tako i kasnijeg kauzalnog liječenja. Ovdje smo prikazali slučaj teškog SIAD-a uzrokovanog segmentnom plućnom tromboembolijom, koji se manifestirao kao akutna psihoza. COVID-19 je zakomplicirao tijek bolesti stečene u bolničkim uvjetima. Istodobnim liječenjem plućne tromboembolije, postupnom supstitucijom hiponatremije i liječenjem COVID-19 bolesnica se biokemijski i psihički potpuno oporavila. Čak i iskusni kliničari mogu biti zbunjeni raznolikom kliničkom manifestacijom SIAD-a. Pažljiva supstitucija hiponatremije i liječenje njezine etiologije temelj je liječenja SIAD-a.

KLJUČNE RIJEČI

Hiponatremija; Hipertonična fiziološka otopina; Plućna tromboembolija