

WHEN IN “DEEP WATERS”! PSYCHOGENIC POLYDIPSIA CASE REPORT

Camer Salim¹, Cristina Ramona Tase¹

¹ Emergency Department, Clinical County Emergency Hospital of Constanta, „Sf. Apostol Andrei”, Romania

Cristina Ramona Tase

email: tasecristinaramona@yahoo.com

ABSTRACT

We report a case of a 50-year-old female patient presented to the Emergency Department (ED) in respiratory arrest preceded by an inaugural generalized seizure with traumatic lingual mark and sphincter relaxation. Biological: metabolic acidosis, severe hyponatremia (115 mEq/L), osmolarity 253 mOsm, lactate 2 mmol/L. Acute hyponatremia correction treatment was initiated by reducing infusion solutions, administration of hypertonic sodium molar solution on continuous infusion in 30 minutes (30 ml saline and 20 ml natrium molar solution 58.5 mg/ml), loop diuretic 40 mg iv, cerebral depletive 150 ml and administration of desmopressin. After Foley catheterization, 3-hour diuresis was 6000 ml. The patient was known to have polynodular goiter on thyroazole treatment. Emergency CT scan result- absence of acute intracerebral pathology; presence of a large amount of gastric fluid that depletes including the esophagus. After the insertion of nasogastric tube, we evacuated approximately 4000 mL gastric contents. Toxicology tests were negative. The patient was admitted to the intensive care unit where, after sodium correction and appropriate management, her clinico-biological evolution was favourable. Diagnosis of diabetes insipidus and Syndrome of inadequate antidiuretic hormone secretion (SIADH) were ruled out. After 5 days, the patient was conscious, hemodynamically stable and she kindly told the story about her severe depressive disorder diagnosed one year ago in the US, treated with selective serotonin reuptake inhibitors and about her diet with a water intake of 6-8 litres daily for 6 months.

Keywords: hyponatremia, seizures, psychogenic polydipsia

Introduction

Not infrequently we are confronted with the same questions, both we, as health professionals, and patients in particular: what do you do when you are faced with situations from which you can't find a “way out”, when you can't communicate or explain what is happening to you, when your life is in danger and you can't “cry” for help? Psychogenic polydipsia is a pathological condition often found in patients with psychiatric illness that can end in hyponatremic coma, generalized seizures with subsequent respiratory arrest.

Hyponatremia (natrium <135 mmol/L)

occurs in up to 20% of patients with compulsive water consumption. Neuropsychiatric manifestations of hyponatremia include headache, nausea, myalgia, hyporeflexia, dysarthria, lethargy, temporo-spatial disorientation. If untreated, it can rapidly progress to water intoxication (a severe form of hyponatremia) and can cause grand mal seizures, cerebral oedema and death (1).

The diagnosis is mainly one of exclusion, so other medical causes of polydipsia, polyuria and/or hyponatremia must be rapidly excluded in the emergency department.

Effective therapeutic management involves fluid restriction, correction of hyposodemia

started in the ED, pharmacologic and psychiatric therapy. Serum sodium should be monitored frequently, with prudent use of hypertonic saline in the treatment of symptomatic hyponatremia. (2)

Material and Method

At 12:45 - a car brakes and stops abruptly at the door of the Emergency Reception Unit. In the back seat is a female patient, brought by family from home for altered general condition for approximately 6 hours, with subintractant convulsive seizures during transport to the hospital. Personal pathological history: hyperthyroidized polyendocrinodular goiter, on Thyrozole treatment.

On admission: respiratory arrest, biting tongue with bleeding at the oral commissure, secondary to generalized tonic-clonic convulsive seizures, sphincter relaxation.

Case management in the Emergency Room

A V P U - unconscious patient. A: Respiratory arrest, blood in oral cavity - Upper airway clearance, Saffar triple maneuver, Gudel's pipe is practiced, B: Look, feel, hear- no chest expansion, IOT+VM, C: central pulse present in carotid arteries, CRRT" 2 seconds, peripheral pulse absent in radial arteries. 2 peripheral venous access routes are obtained, biological samples are collected, arterial puncture-EAB, Normal ECG, AV= 60 bpm, sinus rhythm, D: GCS 3 points, glucose on glucotest- 195 mg/dL, myopic pupils are active, corneal reflex abolished, oculomotor reflex abolished, spontaneous Babinski on the right, CPR indifferent on the left, Brudzinski and Kernig negative, E: biting tongue, sphincter relaxation.

Biological: on ASTRUP, metabolic acidosis, pH 7.05, severe hyponatremia (115 mEq/L), potassium 4.7 mEq/L, osmolarity 253 mOsm, lactate 2 mmol/L. Toxicology - negative, urinalysis: urinary osm towards the lower limit of normal reference range, rest of tests (CBC, biochemistry)- within normal parameters. Acute hyponatremia correction treatment was initiated by: orotracheal intubation and mechanical ventilation; administration of hypernatremic sodium molar solution with continuous infusion

in 30 minutes (30 ml saline and 20 ml sodium molar solution 58.5 mg/ml), sodium bicarbonate 8.4% 100 mL, reducing infusible solutions, loop diuretic 40 mg iv, cerebral depletive 150 ml Mannitol 20%. Post resuscitation patient reassessment: A V P U- Patient responds to verbal stimulation, A: OTI, B: patient resumes spontaneous breathing; mechanical ventilation in SIMV mode, C: central pulse present in carotid arteries, CRT < 2 seconds, peripheral pulse present in the radial arteries, BP= 110/70 mmHg, AV=78 bpm, sinus rhythm, repeat the ASTRUP, D: GCS 7 points, blood glucose on glucotest- 169 mg/dL, pupils intermediate, equal, reactive, psychomotor agitated-" analgesia of the patient Fentanyl (I F) and Midazolam (I F) on continuous infusion (8 mL/H), E: t=36.7, abdomen distended by volume, no signs of acute surgical abdomen, after Foley catheterization Polyuria (5500 mL/ 2H), hypochromic urine. Stage diagnosis at this point was: respiratory arrest, severe mixed acidosis, severe hyponatremia, in observation inadequate secretion of ADH. We started to administer desmopressin (Minirin Melt 60 mcg, sublingual, 2 tablets) and indicated CT scans (cerebral, thorax, abdominal)- Figure 1. Nasogastric tube then is fitted - about 4000 mL gastric contents are evacuated.

Diagnosis: 1. Respiratory arrest. 2. Status post inaugural seizure. 3. Coma. 4. Severe hyponatremia. 5. Severe metabolic acidosis. 6. Observing water intoxication. 7. Observing psychogenic polydipsia.

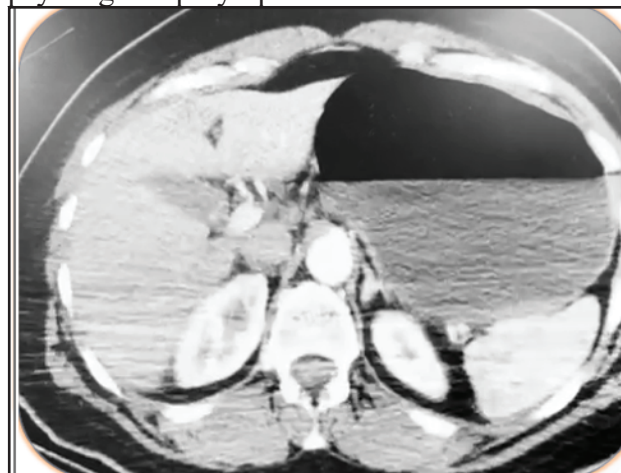


Figure 1: CT scans: Absence of acute intracerebral pathology; presence of a large amount of gastric fluid distending also the oesophagus!

The patient was admitted to the intensive care unit where, after sodium correction and appropriate management, her clinical and biological evolution was favourable.

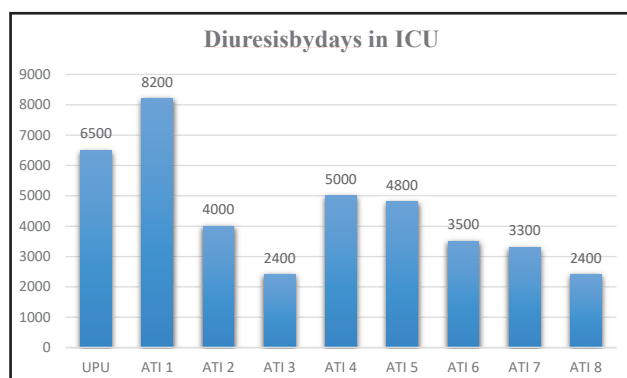


Figure 2 diuresis monitoring in the intensive care unit (ATI/ICU)

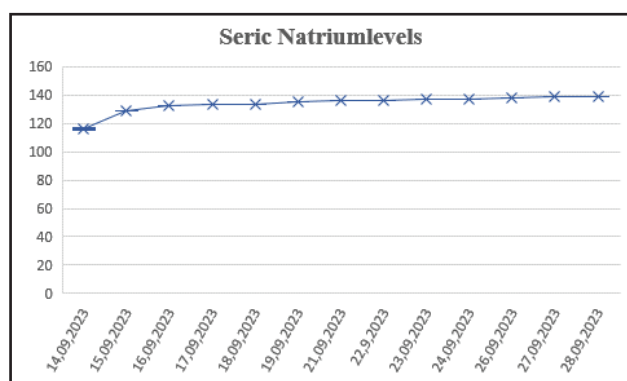


Figure 3 correction of sodium values in the ICU

After 5 days, our patient is detubated, GCS 15 pct, conscious, cooperative, hemodynamically and respiratory stable. Endocrinologic tests were within normal limits throughout. (TSH, T3, T3, FT3, FT4). Diagnosis of diabetes insipidus was ruled out (normal glycaemic values, PT=4,9, NH3=33,8, ATG=13, PRO-vasopressin=15 pmol/L- reference range 2,4-28,2 pmol/L). She reports that she has been on a diet with a hydration intake of 6-8 liters of water daily for about 6 months. She was diagnosed a year ago with depressive disorder, for which she was being treated with selective serotonin reuptake inhibitors (Paroxetine) in the USA. She recently returned to the country to visit her mother, who was recently diagnosed with an incurable disease.

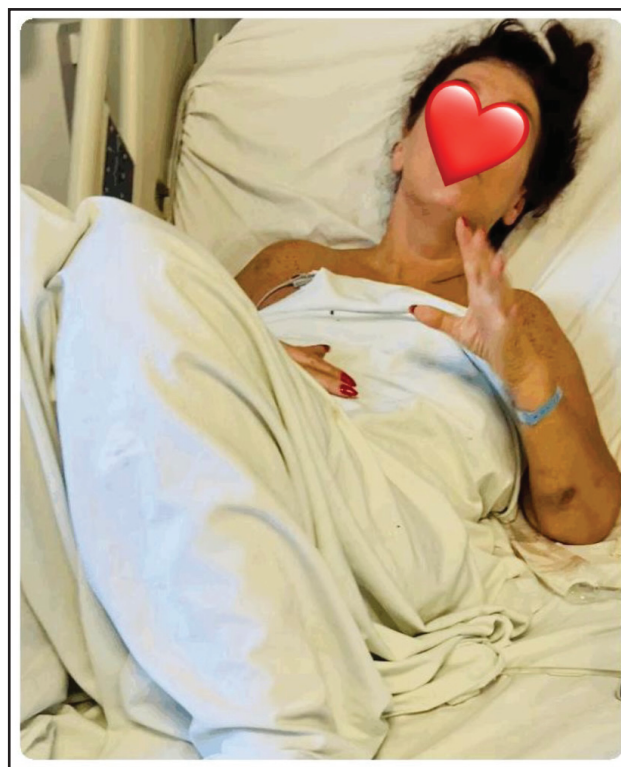


Figure 4 Detubated patient, conscious after 5 days

All necessary investigations were ordered, and the patient's vital signs were closely monitored. The target serum sodium level was not higher than 125 mmol/l in six hours to avoid overcorrection symptoms. Syndrome of inadequate antidiuretic hormone secretion (SIADH) was ruled out by the endocrinology team, according to the patient's urine analysis. Further investigations excluded all medical conditions that are known to be etiologically related to hyponatremia. A diagnosis of severe hyponatremia induced by psychogenic polydipsia was made as he had low serum osmolality. In addition, a psychiatric consultation was requested. A nutritional assessment revealed that the patient was normoweight. Diagnosis of certainty: Psychogenic polydipsia aggravated by antidepressant treatment with paroxetine.

Discussions

Severe acute hyponatremia is a medical emergency that, if not recognized in time and untreated, can lead to death within hours.

The life-threatening consequences are seizures, cerebral edema, brain stem herniation, aspiration pneumonia, respiratory arrest and

in some cases rhabdomyolysis, compartment syndrome by striated muscle necrosis. (3)

There are 3 elements can characterize 'water intoxication': a) polydipsia, which is defined as water consumption of more than three litres per day and up to 10 or 15 litres, b) inability to excrete water due to renal impairment or inadequate secretion of antidiuretic hormone, c) cerebral sensitivity to hyponatremia. (4)

Correction of serum sodium levels should be done slowly, a rapid rate leading to other complications of neuronal osmotic demyelination, e.g. central pontine myelinolysis. (5)

The differential diagnosis is mainly made with diabetes insipidus and syndrome of inappropriate antidiuretic hormone secretion (SIADH).

Dilutional hyponatremia (hypotonic hyponatremia) is most commonly referred to in the literature, occurring when water intake exceeds the excretory capacity of the kidneys, usually in the context of renal failure.(3)

Paroxetine (Seroxat) is an antidepressant in the class of selective serotonin reuptake inhibitors that causes hyponatremia through inadequate ADH secretion.

In prehospital, these patients may have only signs of severe neurologic dysfunction with generalized, inaugural tonico-clonic seizures secondary to hyponatremia and cerebral edema who are given anticonvulsant therapy without symptomatic coupling! cause-effect

Other particular situations in which the prognosis of psychogenic polydipsia is a dramatic are mentioned in the literature such as: unmanageable subintractant seizures leading to rhabdomyolysis, cerebral edema, aspiration pneumonia and crural compartment syndrome following water intoxication (6)

The differential diagnosis is made with diabetes insipidus and syndrome of inadequate antidiuretic hormone secretion (SIADH), as shown in Table 1. (7)

Table 1: differential diagnostic elements of psychogenic polydipsia.

	Psychogenic polydipsia	Diab. Insipidus	STADH
Plasma osmolality	↓	↑	↓
Serum sodium	↓	↔ or ↑	↓
Urine osmolality	↓	↓	↑
Urine sodium	↔	↔	↑

Conclusions

The most valuable treasure is life so give meaning to it and don't live shallow! Compulsive water consumption has no specific cause, but complications can be fatal (polydipsia, polyuria, seizures, coma, respiratory arrest). The diagnosis of this pathology in the emergency room is sometimes difficult to make, especially in comatose patients, in cardiorespiratory arrest, or extreme psychomotor agitation, with impossible anamnesis, without the possibility of obtaining medical history data.

The possibility of performing ASTRUP in pre-hospital or in the Emergency Department is absolutely necessary for a correct diagnosis.

References

1. Gill M, McCauley M. 2015. "Psychog Psychogenic polydipsia: the result, or cause of, deteriorating psychotic symptoms? A case report of the consequences of water intoxication. ." *Case Rep Psychiatry*. 2015:846459.
2. No year. https://www.researchgate.net/figure/Biochemical-differences-between-psychogenic-polydipsia-diabetes-insipidus-and-SIADH_tbl1_13602951.
3. J. J. M. Ligtenberg, Anke N Machteld Wymenga, Jaap E Tulleken. 1998. „A lethal complication of psychogenic polydipsia: Cerebral edema and herniation." *Intensive Care Medicine* 24(6):644-5.
4. Ligtenberg, J. & Wymenga, Anke & Tulleken, Jaap & van der Werf, Tjip & Zijlstra. Jan. (1998). „A lethal complication of psychogenic polydipsia: Cerebral edema and herniation. ." *Intensive care medicine*. 24. 644-5.
5. Nauwynck E, Van De Maele K, Vanbesien

- J, Staels W, De Schepper J, Gies I. 2021, . „Psychogenic polydipsia in a female adolescent without a psychiatric background: a case report. .” *Clin Case Rep.* 9:1937-42. .
6. Quinn CJ, Iyegha UP, Beilman GJ, Cerra FB. 2012. „Acute correction of hyponatremia secondary to psychogenic polydipsia.” *Am J Case Rep.* 13:69-71. .
 7. Sharma P, Shah B, Sangroula M, Jirel R. 2019. „Psychogenic polydipsia complicated to hyponatremia induced seizure in schizophrenia: a case report from Nepal. .” *Case Rep Psychiatry.* 2019:6021316. .
 8. Weingarden H, Curley EE, Renshaw KD, Wilhelm S. 2017. „Patient-identified events implicated in the development of body dysmorphic disorder.” *Body Image.* 21:19-25. .