

Case Report: A Severe Hyponatremia with Multiple Organ Failure, about a Clinical Observation

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Abstract

A hyponatremia severe is often associated with a poor prognosis, especially if it is associated with multiple organ failure. In a country with very limited resources, the prognosis may be favorable in the absence of renal replacement. We report the case of a 63 years old woman, hypertensive and diabetic, admitted to the ICU for unconsciousness. Clinically, neurological examination notes a Glasgow of 8/15 (Y2, V2, M4) and a left hemiplegia. Temperature was 39°C. Diuresis was 100 ml during the first 24 hours. Blood pressure was 90/60 mmHg, tachycardia at 133/min. Cardiac auscultation is normal. Vascular axes were weakly perceptible. Oxygen saturation was 95%. The skin examination notes a dry skin and mucous membranes, a flattening of the superficial veins, sunken eyes and a persistent skin fold. There is no hepatomegaly or splenomegaly, or jaundice. Biological point of view note natremia: 176 mmol/L; osmolarity: 390 mosmol/kg; creatinin: 300 µmol/L; glycemia > 6 g/L; transaminases 217 UI. Diagnostics: malignant hyponatremia with a high plasma osmolarity associated with an acute anuric renal failure, hydro electrolytic disorders, an abnormal liver function, a fever of central origin and a stroke. The treatment consisted of a correction of the electrolyte disorders by infusion of isotonic and hypotonic fluids with insulin. In a country with very limited resources, the severe hyponatremia prognosis associated with anuric acute renal failure may be favorable in the absence of renal replacement.

Keywords

Hyponatremia, Hyperosmolar Coma, Multiple Organ Failure, Acute Renal Failure, Diabetes

1. Background

Severe hypernatremia is often associated with a poor prognosis; however, some cases of extreme hyponatremia successfully treated have been reported in the literature [1] [2]. Indeed, there is a positive association between the severity of hypernatremia and mortality [3]. More generally, it is estimated that acute hypernatremia onset, greater than 160 mmol/l, is associated with a mortality rate that varies between 42% and 60%. This rate is lower when it is a chronic hypernatremia onset [4].

We report the case of a patient with extreme hypernatremia associated with acute anuric renal failure and multiple organ failure treated with hypo and isotonic solutions.

2. Clinical Case

This is a 63 year old woman admitted to the emergency services of the Donka University Hospital Center for a gradual onset of coma.

His medical history is marked with type II diabetes, diagnosed ten years ago and treated with 5 mg of glibenclamide. It is not complicated of macro and microangiopathy. She also has an old Hypertension discovered in the same year, and was treated with 10 mg Amlodipine.

Clinically, neurological examination notes a Glasgow of 8/15 (Y2, V2, M4) and an isolated left hemiplegia. There was neither meningeal syndrome nor attacks of cranial nerves. She was febrile at 39°C without development of specific infectious appeal. Lumbar pits are free and painless. Diuresis was almost zero in 100 ml during the first 24 hours. Blood pressure is rather low for a known and treated hypertensive patient. She was 90/60mm Hg with no signs of shock, but tachycardia at 133/min. Cardiac auscultation is normal without perceived cardiovascular breath. Vascular axes were weakly perceptible. It was stable on the respiratory plan with Oxygen saturation of 95% within room air without auscultatory and radiological home. The skin examination notes a dry skin and mucous membranes, a flattening of the superficial veins, sunken eyes and a persistent skin fold. There is no hepatomegaly or splenomegaly, or jaundice.

The electrocardiogram is normal, no disturbance of repolarization or conduction.

From a biological point of view we noted to admission:

A hypernatremia to 176 mmol/l with high plasma osmolarity to 390 mosmol/kg of water. The corrected natriemia is 186.5 mmol/l. The estimated water deficit is about 10 kg for a typical weight of 70 Kg. There is metabolic acidosis with an alkaline reserve to 16 mmol/l and potassium levels to 5 mmol/l. There was a blood volume contraction with proteins to 85 g/l without argument for a monoclonal peak. The calcium and phosphate was correct. They were a biological inflammatory syndrome with leukocytosis 16,000/mm³ and CRP 30 mg/l; the rest of the blood count notes hemoglobin to 15 g/dl and a hematocrit to 45%. The thick film (research of trophozoites of malaria) comes back positive, 0 - 1 trophozoite/microscopic field. Diabetes is at a high blood sugar > 6 g/l. The renal

function was altered with a serum creatinine to 300 mmol/l knowing that the basis of serum creatinine was normal six months ago to 80 mg/dL. Blood cultures were negative. Liver function tests noted cytolysis without cholestasis. ASAT and ALAT were to 10 times the normal, 217 UI/l. The dipstick is positive for the protein and for leukocytes. It was negative for ketones and nitrites. Natriuresis on sample is less than 20 mmol/l with a ratio of Na/K⁺ reversed.

Morphologically: Chest X-ray is normal, no focal or radiological sign of overload, no overhang heart, heart within normal limits. The renal ultrasound shows a kidney size of 11 cm on each side, without dilatation of pyelocalical cavities, the cortico-medullary index is slightly reduced; there is no lithiasis. The non-injected scanner is normal, no hematoma.

The diagnosis was a malignant hyponatremia to 176 mmol/l with a diabetic hyperosmolar coma. It is defined as a high plasma osmolarity to 390 mmol/l due to a major hyperglycemia (greater than 33 mmol/l). Ketosis is absent. Dehydration was massive and global as evidenced by the significant weight loss of 10 kg and anuric acute renal failure. Fever is probably of central origin associated with hyponatremia. As to the hepatic cytolysis, it may be related to liver ischemic shock. There is no infectious sign.

The patient is first hospitalized in intensive care for a total of a week before being transferred to ensure a better monitoring and management.

Initially, she has benefited to a prudent parenteral rehydration, two liters of physiological saline solution of 0.9% during three hours to restore the hemodynamic status; then it is replaced with glucose solution of 5% associated with insulin in the absence of other hypotonic solutions. The water deficit has been corrected on 48 h. The correction of natremia over the first twenty-four hours was 13 mmol/l. Along with rehydration she received a rapid insulin treatment to correct her hyperglycemia, 6 UI per hour with a target range between 2.5 and 3 g/l. The correction of blood glucose has been slow to avoid the risk of cerebral edema.

The inflammatory syndrome normalizes with rehydration and diabetes on insulin progressively too.

Clinically, the patient gradually wakes as serum sodium is corrected. This happens in D4 with complete disappearance of the left hemiplegia. The fever disappears within 72 hours in rehydration. The resumption of diuresis was progressive during rehydration. It started on day 1 after 10 hours of rehydration and then normalizes between D2 and D3; In the other hand, we noted the normalization of mucocutaneous signs with a weight of 67 Kg for a typical weight of 70 kg.

The evolution was marked by normalization of laboratory parameters (**Table 1**).

3. Discussion

A natriemia greater than 155 mmol/l at the admission is an independent mortality risk factor with an or between 2.62 and 3.64 [1]. Only a few cases of survival

Table 1. Evolution of laboratory parameters.

Parameters	D1	D2	D3	D4	D6	D7
Natremia (mmol/l)	176	163	151	140	137	138
Creatinine $\mu\text{mol/l}$	300	200	179	127	110	90
Hemoglobin (g/dl)	14.7	13	12.3	10.8	10.8	11
Transaminases (UI)	217	170	141	131	100	72

Natremia normal: 135 - 140 mmol/l; Creatininemia normal: 80 - 90 mmol/l.

of patients with serum sodium greater than 170 mmol/l are described in the literature [1].

In the case of our patient, it is a malignant hypernatremia to 176 mmol/l in association with a plasma hyperosmolarity to 390 mosmol/l in connection with a major hyperglycemia greater than 33 mmol/l. Portel *et al.* reported a case of overall dehydration associated with a serum sodium level among the highest (209 mmol/l) and an osmolarity of 456 mosmol/l with a favorable prognosis under treatment. [2] Hyperosmolar coma is fatal in half of the cases. Prognosis depends on the time of treatment of the causal factor. It is even worse in case of vascular accident or the possible complications of decubitus installation.

A fever and diabetes mellitus are from the most common causes of hypernatremia [5]. Fever in our case would be of central origin related to hypernatremia, like cerebral hemorrhage and hemiplegia as described in the literature [6]. It is responsible in our case study, in addition to the major hyperglycemia, of an overall dehydration associated with acute renal failure anuric. The treatment consisted of a correction of electrolyte troubles by hypotonic solutions (glucose solution 2.5% + insulin) and isotonic (saline 0.9%). In this case, the acute renal failure is functional as evidenced by the rapid improvement of renal function in rehydration associated with the correction of hyperglycemia. I recall that the natriuresis was locked (less than 20 mmol/l) and the Na^+/K^+ ratio reversed. There was not abnormal urinary sediment. All these data support the functionality character. Renal function at the output is normalized to 90 $\mu\text{mol/l}$ of creatinin against a peak of 300 μmol . Water, electrolyte disorders associated with anuric acute renal failure have been successfully treated by renal replacement with hypotonic dialysate as reported by Giabicani M. *et al.* in 2015 [1] and CW Yang in 1995 [7]. In our case, the therapeutic possibilities are very limited.

4. Conclusion

The severe hypernatremia prognosis associated with anuric acute renal failure may be favorable in the absence of renal replacement by a gradual correction of fluid and electrolyte disorders with hypo and isotonic solutions.

Conflict of Interest

There is no conflict of interest. The script I am about to present is original and represents my work and that of my co-authors. No part of the manuscript (text,

tables) has been copied or borrowed from an existing material or without a clear reference to the source.

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