Journal of Case Reports and Scientific Images

E-ISSN: 2708-0064
P-ISSN: 2708-0056
JCRSI 2024; 6(1): 16-20
www.allcasereports.com
Received: 13-12-2023
Accepted: 21-01-2024

Fjouji Salaheddine
Department of Anesthesia and Critical Care, Military Mohamed V Training Hospital of Rabat, Morocco, Hassan II University of Casablanca, Morocco

Houba Abdelhafid
Department of Anesthesia and Critical Care, Military Mohamed V Training Hospital of Rabat, Morocco, Hassan II University of Casablanca, Morocco

Nawfal Doghmi
Department of Anesthesia and Critical Care, Military Mohamed V Training Hospital of Rabat, Morocco, Hassan II University Of Casablanca, Morocco

Balkhi Hicham
Department of Anesthesia and Critical Care, Military Mohamed V Training Hospital of Rabat, Morocco, Hassan II University of Casablanca, Morocco

Corresponding Author:
Fjouji Salaheddine
Department of Anesthesia and Critical Care, Military Mohamed V Training Hospital of Rabat, Morocco, Hassan II University of Casablanca, Morocco

Rhabdomyolysis induced by hyperosmolarity secondary to severe hypernatremia in a patient with trauma brain injury: A case report

Fjouji Salaheddine, Houba Abdelhafid, Nawfal Doghmi and Balkhi Hicham

DOI: https://doi.org/10.22271/27080056.2024.v6.i1a.73

Abstract
Hypernatremia is defined by an increase in blood sodium concentration greater than 145 mmol/l. The incidence of this disorder is 3 to 10% in intensive care patients, and varies from 10 to 50% in brain-injured patients. Severe hypernatremia greater than 190 mmol/l may be associated with rhabdomyolysis in rare cases described in the literature. The authors describe a case of a patient with severe head trauma in intensive care who presented this combination of disorders whose diagnosis must be early and correction must be careful in order to maintain hydro-electrolyte homeostasis, especially in patients with a compromised neurological prognosis.

Keywords: Hypernatraemia, hyperosmolarity, rhabdomyolysis, acute renal failure, trauma brain injury

Introduction
Hypernatremia is defined by an increase in blood sodium concentration greater than 145 mmol/l. The incidence of this disorder is 3 to 10% in multipurpose intensive care patients [1], and varies from 10 to 50% in brain-injured patients [2] high serum sodium levels may be responsible for rhabdomyolysis and secondary acute renal failure. The authors report an observation of a patient with severe head trauma who presented these metabolic disorders which complicated the treatment and influenced the prognosis.

Case report
19-year-old patient with no particular history was admitted to intensive care following serious head trauma. The patient was the victim of a public road accident. When he was crossing the road a car hit him. The impact was cranial. he was transported by firefighters to the emergency room. Examination upon admission to the emergency room found an unconscious patient, a Glasgow coma score of 6, a left fronto-temporal scalp wound with subgaleal hematoma and a left eyelid ecchymosis. Blood pressure was 100/75, heart rate was 85 bpm, respiratory rate was 30 cpm with oxygen saturation 97%. After intubation and mechanical ventilation, deep sedation with midazolam and fentanyl infusion was started associated with a bolus of 20% mannitol at a dose of 1g/kg to counter intracranial hypertension objectified by the initial transcranial Doppler (diastolic velocity at 18 m/s). Cerebral tomography showed two bilateral hemorragic edematous contusions, the largest of wich is left fronto-temporal, left subfalcine herniation, diffuse cerebral odema, subarachnoid hemorrhage, and hemorrhagic filling of the facial sinuses (FIGURE1). The chest x-ray revealed an appearance of left basal lung disease related to probable aspiration. The biological assessment was marked by an inflammatory syndrome (white blood cells at 12,500/mm3, CRP at 75 mg/l) and by moderate hypernatremia at 150 mEq/l. The patient underwent an initial decompressive craniectomy in the emergency operating room due to the inability to monitor intracranial pressure. In intensive care, the patient had intracranial hypertension demonstrated by transcranial Doppler (Diastolic velocities less than 20 m/s) requiring maintenance of deep sedation. Peaks of intracranial hypertension responsible for Cushing's reflexes and reversible bilateral mydriasis required the use of mannitol boluses in an iterative manner. Anticonvulsant prevention was provided by sodium valproate with electroencephalogram monitoring. The control of secondary cerebral accidents of systemic origin was ensured by invasive hemodynamic monitoring (radial arterial line and right

subclavicular venous access), blood gas control to optimize oxygen values and capnia through parameters ventilators of the respirator. The respiratory infection was controlled with antibiotics (ceftazidime and amikacin), biological assessments were carried out to ensure hydro-electrolyte and metabolic balance. The stay was marked by the worsening of the initial hypernatremia reaching figures of 195 mEq/l on the third day and significant polyuria (8 liters/day). Blood osmolarity was 396 mosm/l. Urine biological analyzes were not carried out. The patient was put on hydration in plain water by nasogastric tube and Dextrose 5% in the absence of dextrose 2.5% as well as administration of desmopressin (dose of 2 mcg/day). The evolution was marked by a regression of polyuria and a progressive regression of hyperosmolarity and hypernatremia (155mEq/l on the sixth day). In order to eliminate a neurological complication linked to hypernatremia or the rapid correction of this disorder, a brain CT scan was performed on the fourth and seventh days which did not show specific lesions of metabolic origin. However, areas of ischemia around the initial traumatic contusions have set in. During the stay in intensive care, rhabdomyolysis set in from the fourth day objectified by the dosage of creatine phosphokinase which reached a peak of 42,000 IU/l and which was complicated by acute renal failure from the seventh day (peak urea and creatinine 1.30 g/l and 26 mg/l respectively) (FIGURE 2). Faced with the deterioration of renal function and the drop in diuresis, Desmopressin was stopped. Urine alkalinization was difficult to achieve with sodium bicarbonate solutions given the risk of worsening hypernatremia. The use of hemodialysis sessions was necessary using a dialysate rich in sodium (150mEq/l). Neurologically, the evolution was not favorable. Refractory intracranial hypertension led to a state of brain death (bilateral mydriasis and reverse flow appearance on transcranial Doppler) and the death of the patient on the eighth day.

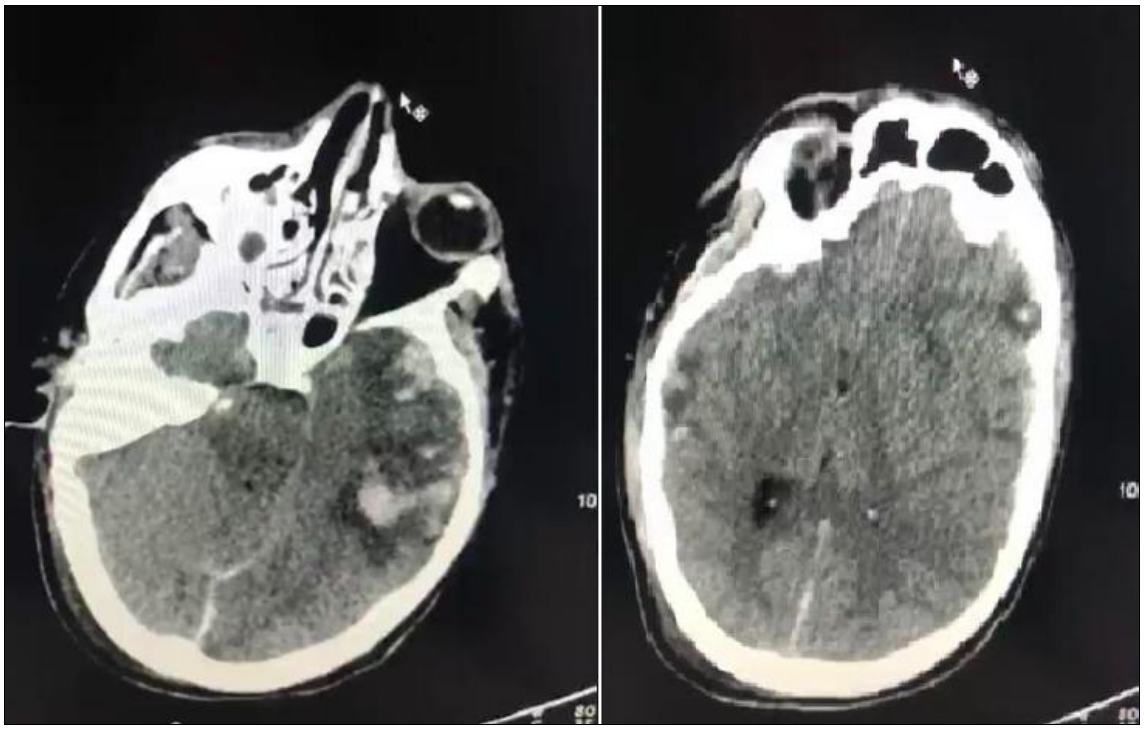
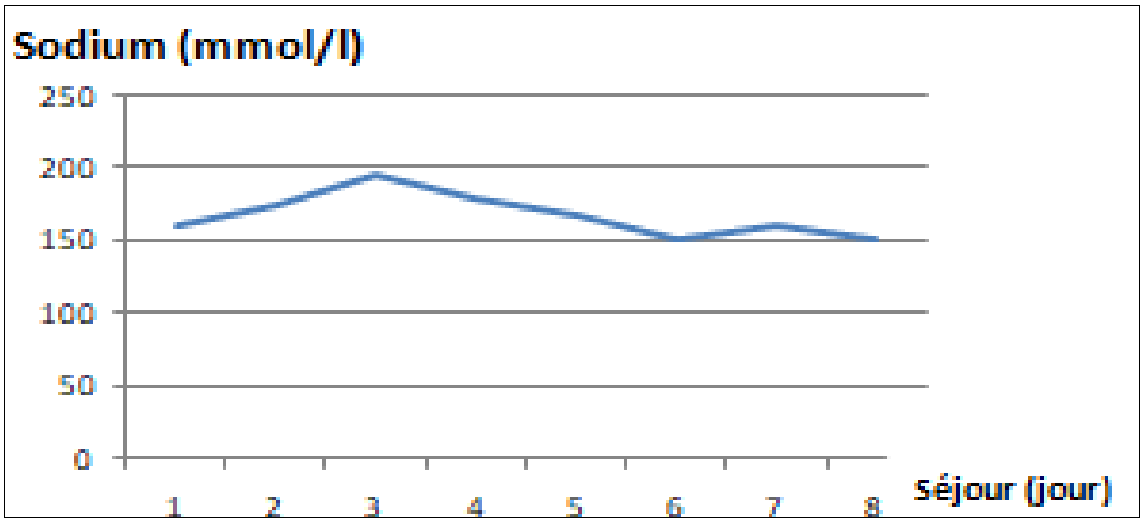


Fig 1: Cerebral tomography showed two bilateral hemorragic edematous contusions, the largest of wich is left fronto-temporal, left subfalcine herniation, diffuse cerebral odema, subarachnoid hemorrhage, and hemorrhagic filling of the facial sinuses



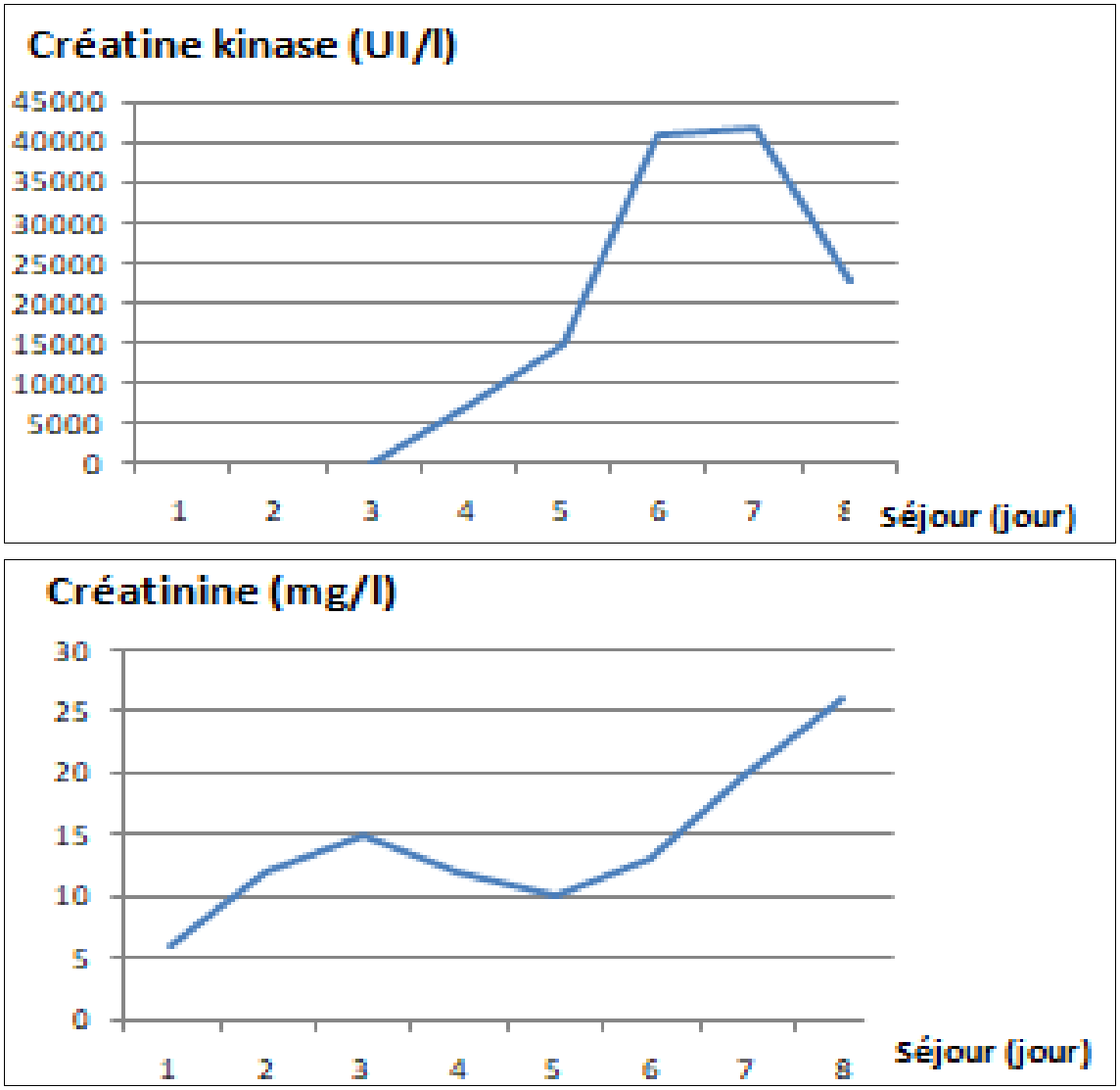


Fig 2: The figure shows the variation in intensive care of biological parameters: sodium, creatine phosphokinase and creatinine according to the days of hospitalization. After a peak in serum sodium on the 3rd day (195 mmol/l), there was a progressive regression of the figures following therapeutic correction means while maintaining supranormal values (greater than 150 mmol/l). From the 3rd day, the creatine phosphokinase figures begin to increase until a peak between the 6th and 7th day of hospitalization (42,000 IU / l) at the time when acute renal failure appeared with progressive increase (value maximum of 26 mg/l) thus compromising the patient's prognosis and his death on the 8th day.

Discussions

Hypernatraemia is a common disorder in intensive care. It was found in more than 50% of head trauma patients, and was associated with a three times greater risk of death [3]. During hypernatremia, the increase in the osmolality of the extracellular fluid leads to a movement of water from the cells towards the extracellular sector, causing dehydration and shrinkage of especially neurological cells. This effect of brain volume contraction may be beneficial by lowering intracranial pressure in the early phase. This osmotherapy induced by hypernatremia or mannitol decreases in effectiveness over time due to the new osmotic balance established by the brain. Indeed, an early accumulation of inorganic osmoles allows brain tissue a new adaptation towards a state of intracellular hypertonicity limiting water transfer and variations in brain cell volume. If hypernatremia becomes symptomatic, the signs of hypernatremia are mainly neurological. Their severity is related to the degree and rate of increase in serum osmolality. They can vary from a slight confusional state, nausea and vomiting, muscle weakness, drowsiness to the appearance of serious signs such as convulsions or coma [4].

In brain-damaged patients the distinction of the clinical impact of the The causative pathology hypernatremia is often difficult. In our patient, the brain scan did not show any change in images or worsening of cerebral edema during hydration correction. Contraction of brain volume can pull and tear intracerebral veins, causing subarachnoid or intracerebral hemorrhages. In our case, there was no additional bleeding on the CT scan compared to the initial appearance. The etiological search for hypernatremia in brain-injured patients depends on the status of their blood volume. Thus, in the case of hypovolemia, hypernatremia would be due to insufficient water intake, particularly in comatose patients, to water losses of renal origin (diabetes insipidus, osmotic diuresis, diuretics) or extra-renal (respiratory, digestive, fever). In case of normo- or hypervolemia, hypernatremia would be due to excessive administration of sodium, Cushing's syndrome or primary hyperaldosteronism [2]. Our case presented hypernatremia following a loss of water of renal origin through repeated use of mannitol as well as probable post-traumatic diabetes insipidus (The dosage of antidiuretic hormone was not carried out but the diagnosis of diabetes insipidus was based

on a favorable clinical response to desmopressin administration). Hypernatraemia associated with rhabdomyolysis has been reported as a rare event, especially if it is also complicated by renal failure [5, 6, 7]. The causes of rhabdomyolysis apart from Crush syndrome which remains the most common are medications (neuroleptics, halogenated, etc.), toxic (alcohol), infections and more rarely a state of hyperosmolarity. The latter would be responsible for muscle lysis in our patient favored by prolonged bed rest. The medications given to the patient (midazolam, fentanyl, ceftazidine, amikacin, enoxaparin, etc.) are not known to cause rhabdomyolysis. The exact mechanism of hypernatremia causing rhabdomyolysis is not well known. It is hypothesized that a hyperosmolar state inhibits the electrogenic sodium pump in muscle cells and impairs sodium calcium transport, activating protein kinases and leading to muscle cell lysis [8]. On the other hand, rhabdomyolysis also generates new osmoles in skeletal muscle cells and moves water from the extracellular to the intracellular compartment. This further increases serum sodium levels and worsens hypernatremia [9]. Symptomatic treatment aims first to restore blood volume when hypernatremia is hypovolemic. Correction of plasma hypertonicity involves the administration of hypotonic solutions such as serum glucose at 5%, sometimes at 2.5%. In neuro-intensive care, the correction of hypernatremia must not exceed 0.5 mmol/l/h, or 12 mmol/l/24h, the objective being to reduce the serum sodium to around 150 mmol/l. Urinary losses due to diabetes insipidus are compensated with solutions containing potassium and the blood ionogram will be monitored every 6 hours. Substitution with desmopressin also allows correction of antidiuretic hormone deficiency. The objective of treatment is to maintain an effective diuresis between 1 and 1.5 ml/kg/h [10]. Rhabdomyolysis and resulting acute renal failure are treated with adequate and early hydration and alkalinization. The use of sodium bicarbonate solutions may worsen hypernatremia. Renal function improves within a few days to one to two months depending on the reversibility of tubular necrosis lesions. In the event of oligoanuria, major hyperkalemia or severe acidosis, assistance with hemodialysis would be necessary. In the case of acute hypernatremia, as in this patient, hemodialysis or peritoneal dialysis, which causes a rapid drop in serum sodium, can induce cerebral edema and a seizure with high morbidity and mortality. The use of higher sodium or dextrose dialysate could slow the decline of serum osmolarity in patients with hypernatremia and hyperosmolality [11]. In any case, the management of these metabolic disorders remains a major challenge and involves collaboration between intensivists, nephrologists and neurosurgeons.

Conclusion

Hypernatremia is associated with high morbidity and mortality in brain-damaged patients. The benefit of osmotherapy by hypernatremia remains limited to the initial phase of treatment. Correction of this disorder must be careful. The occurrence of rhabdomyolysis following a state of hyperosmolarity is a rare phenomenon in clinical practice. In a brain-injured patient it risks worsening the prognosis, hence the need to consider it in the face of severe hyperosmolarity states by biological assay of creatine kinase with a view to early diagnosis and multidisciplinary care.

Conflict of Interest

Not available

Financial Support

Not available

References

1. Polderman KH, Schreuder WO, Strack van Schijndel RJ, Thijs LG. Hypernatremia in the intensive care unit: an indicator of quality of care? Critical Care Medicine. 1999;27(6):1105-8.
2. Payen JF, Bouzat P, Franconi G, Ichai C. Hypernatrémie chez le patient cérébro-lésé : utile ou dangereux? Annales Françaises d'Anesthésie et de Réanimation (AFAR). 2014;33(6):433-435.
3. Maggiore U, Picetti E, Antonucci E, Parenti E, Regolisti G, Mergoni M, *et al*. The relation between the incidence of hypernatremia and mortality in patients with severe traumatic brain injury. Critical Care. 2009;13(4):R110.
4. Adrogué HJ, Madias NE. Hypernatremia. New England Journal of Medicine. 2000;342(20):1493–1499.
5. Rosa EC, Lopes AC, Filho AW, Schor N. Rhabdomyolysis due to hyperosmolarity leading to acute renal failure. Renal Failure. 1997;19(2):295–301.
6. Lima EQ, Aguiar FC, Barbosa DM, Burdmann EA. Hypernatraemia (221 mEq/l), rhabdomyolysis and acute renal failure after cerebral aneurysm surgery. Nephrology Dialysis Transplantation. 2004;19(8):2126-9.
7. Yang TY, Chang JW, Tseng MH, Wang HH, Niu DM, Yang LY. Extreme hypernatremia combined with rhabdomyolysis and acute renal failure. Journal of the Chinese Medical Association. 2009;72(10):555-8.
8. Shinghal PC, Abramovici M, Ayer S, Desroches L. Determinants of rhabdomyolysis in diabetic state. American Journal of Nephrology. 1991;11(6):447–50.
9. Welt LG, Orloff J, Kydd DM, Oltman JE. An example of cellular hyperosmolarity. Journal of Clinical Investigation. 1950;29(7):935–9.
10. Quintard H, Facchini A, Gentelet C, Ichai C. How to manage dysnatremia disorders in neurointensive care? Praticien en Anesthésie et Réanimation. 2018;22(2):105-109.
11. McBryde KD, Bunchman TE, Dudelka TL, Pasko DA, Brophy PD. Hyper-osmolar solutions in continuous renal replacement therapy for hyper-osmolar acute renal failure: a preliminary report. Pediatric Critical Care Medicine. 2005;6(2):220–5.

How to Cite This Article

Salaheddine F, Abdelhafid H, Doghmi N, Hicham B. Rhabdomyolysis induced by hyperosmolarity secondary to severe hypernatremia in a patient with trauma brain injury. A case report. Journal of Case Reports and Scientific Images. 2024; 6(1): 16-20.