
Case report**ACUTE HYPONATRIEMIA IN A PATIENT WITH SCHIZOPHRENIA: CASE REPORT
WATER INTOXICATION INDUCED ACUTE HYPONATRIEMIA**

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Abstract

Introduction: Hyponatremia is defined as a serum sodium level higher than 135 mmol/L, while serum sodium level lower 125 mmol/L is considered as severe hyponatremia and can lead to coma, death, rhabdomyolysis, and neurologic damage. **Case report:** We present the case of a 34 year old male with history of schizophrenia with multiple seizures followed by loss of consciousness after intake of 6 liters of water. Diagnostic CT scan revealed cerebral edema. Laboratory tests revealed severe hyponatremia (109 mmol/l), hypokalemia and hypocalcemia. The patient was treated with 10% hypertonic NaCl 120 ml per day, 7.4% KCl and calcium gluconate. He was sedated and mechanically ventilated. Antiedematous therapy with mannitol 20% was started. The biochemical results improved slowly with gradual correction of the sodium level: 112 mmol/l, 119 mmol/l and 127 mmol/l respectively. CT scan showed cerebral edema regression was showed on repeated brain scan. On the 6th day sodium level was 131 mmol/l, and the patient was awake, oriented and extubated. **Discussion:** Psychogenic polydipsia occurs in 20% of the psychiatric patients which could lead to severe hyponatremia. Second generation antipsychotics intake could also lead to severe hyponatremia. According guidelines hyponatremia treatment consists of hypertonic NaCl 3% 150 ml infusion and frequent sodium levels measurements. The sodium level correction should be gradual and should not exceed more than 10 mmol/l for the first 24 hours neither more than 8 mmol/L for every next 24 hours. **Conclusion:** In our patient, serum sodium level correction was successfully and safely performed with hypertonic saline (NaCl 10%) in absence of hypertonic NaCl 3%. Strict control of serum sodium levels is a must in order to avoid osmotic demyelination and rhabdomyolysis.

Key words: hyponatremia; water intoxication; hypertonic saline

Introduction

Water intoxication could be a fatal medical condition which includes brain function impairment. It could be defined as a hypoosmolar syndrome resulting from an excessive intake of water, followed with dilutional hyponatremia formed principally by the retention of water exceeding renal free water excretion or impaired free water excretion from the kidneys¹.

Hyponatremia is defined as a serum sodium level below 135 mmol/L². A serum sodium level below 125 mmol/L is considered as severe hyponatremia and can have irreversible clinical consequences such as coma, death or even rhabdomyolysis, and neurologic damage³. The hypo-osmolar

syndrome, with the subsequent translocation of a massive amount of extracellular water into the cells generates an increase in the cellular volume which is leading to brain edema development, demonstrating a variety of neurological signs, from appetite loss or emesis to convulsion or consciousness disturbance, depending on the severity and rapidity of development of hyponatremia⁴.

Acute and chronic form of hyponatremia can be seen in daily practice while hyponatremia caused by water intoxication is always an acute medical condition which demands an Intensive Care Unit (ICU) treatment. We present the case of male patient with schizophrenia with water intoxication.

Case report

A 34 years old male with history of schizophrenia and irregular intake of his medications with multiple seizures followed with lost off consciousness after intake of six liters of water during the last 24 hours. The patient was admitted in the emergency center with GCS of 6 and was immediately intubated.

Diagnostic computerized tomography (CT) scan was performed following the intubation and revealed cerebral edema without CT signs of hemorrhage or presence of any intracranial mass. The patient was transferred to the intensive care unit (ICU) for further treatment and diagnostics. Initial laboratory tests has shown very low levels of sodium (109 mmol/l) and low levels of potassium (3.4 mmol/l) and calcium (1.76 mmol/l). Electrolyte substitution was started with low volume of fluids calculated for period of 24 hours. The patient was treated with 10% hypertonic NaCl 40 ml (B. Braun, Hesse, Germany) three times per day. Correction of potassium levels was made with giving 40 ml of 7.4% KCl (B. Braun, Hesse, Germany) per 24 hours and the levels of calcium was corrected with 10 ml calcium gluconate (B. Braun, Hesse, Germany) twice per 24 hours.

The patient was on mechanical ventilation and sedated with midazolam and fentanyl (Panpharma, Beignon, France), (continuous infusion with infusion rate of 2 mcg/kg/min midazolam and 0.03 mcg/kg/min fentanyl). Neurosurgeon was consulted and antiedematous therapy with mannitol 20%, 125 ml, four times per 24 hours was started.

The next day the results of the laboratory test revealed mild correction of the sodium levels (112 mmol/l). Potassium (3.9 mmol/l) and calcium (2.1 mmol/l) levels were within the normal ranges. The following two days the lab results were getting better with sodium levels of 119 mmol/l and 127 mmol/l respectively.

The brain CT scan was performed on the 5th day and regression of cerebral edema was seen. Sodium level was 131 mmol/l on the sixth day and no other laboratory abnormalities were seen. The patient was awake and oriented with GCS 14. No abnormalities were detected on arterial blood gases analysis before and after extubation. The patient was transferred to the Clinic of Neurology for further treatment.

Discussion

We presented a case of patient with schizophrenia who developed severe acute onset hyponatremia and cerebral edema because of intake of six liters of water. Patients with psychiatric disorders, including those with schizophrenia and presence of delusions may develop polydipsia and subsequent hyponatremia because of uncontrolled water intake⁵.

According to Gill M. and McCauley M. psychogenic polydipsia occurs in 20% of the psychiatric patients which could lead to severe hyponatremia⁶. Our patient was diagnosed with schizophrenia when he was 24 years old and not taking medications regularly. The excessive water intake frequently occurs in patients with psychiatric disorders, especially in patients with schizophrenia leading to rapid development of hyponatremia⁸.

Hyponatremia is a common electrolyte abnormality which could be caused by an extracellular compartment fluid excess or an actual sodium deficiency. Signs of neurological and neuromuscular disorders (headache, confusion, stupor, seizures, and coma) are result of brain cells edema⁹.

Developing hyponatremia could be a potentially life-threatening medical condition in patients with schizophrenia. According to Ali and Bazzano the second generation antipsychotics (olanzapine, risperidone, aripiprazole, clozapine and ziprasidone) can be potential cause of severe hyponatremia¹⁰. Hyponatremia is reversible after discontinuing the treatment¹⁰. Rapid and excessive water intake leads to dilutional hyponatremia and subsequent brain edema. Brain edema is the main cause of neurologic symptoms like seizures, coma or even death after water intoxication. In our patient, the brain CT scan performed on admission revealed cerebral edema which was explained later with laboratory results of presence of severe hyponatremia of 109 mmol/L. Severe hyponatremia has been associated with increased mortality, morbidity and length of hospital stay². An acute hyponatremia term defines hyponatremia development during 48 hours, while chronic hyponatremia persists more than 48 hours.

The treatment of the hypervolemic hypoosmotic hyponatremia consists of fluids restriction and sodium supplementation. According to the guidelines, 150 ml of hypertonic saline with concentra-

tion of 3% has to be given to the hyponatremia patient initially in the first 20 minutes after admission in the ICU followed with a check up of the serum sodium level after the bolus infusion^{11,12}. Another dose of 150 ml 3% hypertonic saline should be given in the next 12 hours and close monitoring of serum sodium level on every 4–6 hours should be performed^{11,12}. As a therapeutic measure the predicted dose that the patient should receive is 2 ml per kilogram of 3% hypertonic saline^{11,12}. Our patient was treated with a total amount of 120 ml NaCl 10% for 24 hours. The sodium level correction should not exceed more than 10 mmol/L for the first 24 hours and not more than 8 mmol/L for the next 24 hours^{3,11,12}. Planning therapy to achieve the sodium level correction for less than 6 mmol/L is the best way to avoid iatrogenic brain damage¹³.

Hypervolemic hyponatremia is treated with restriction of volume of fluids given per diem, hypertonic NaCl 3% and giving 20–40 mg of furosemide¹⁴. Our patient was treated with mannitol 20% and furosemide for antiedematous and diuretic effects. From the laboratory results of sodium levels taken every day during the hospital stay in the ICU we can conclude that for the first 24 hours sodium level was corrected for only 5 mmol/L, in the second 24 hours 7 mmol/L, the third 24 hours 8 mmol/L and the last fourth 24 hours the correction of the sodium level was only 4 mmol/L. Comparing the numbers of the sodium level correction per day we can say that we did the correction within the ranges proposed by the guidelines for providing safe treatment and avoiding brain damage due to osmotic demyelination^{3,11}. Using 10% of hypertonic saline instead of 3% hypertonic saline in therapeutic properties was necessary due to unavailability of 3% hypertonic saline as a pharmacologic formulation in our country. In order to provide safe treatment and avoid osmotic demyelination we adjusted the doses of the 10% hypertonic saline to be approximately equivalent of the doses of 3% hypertonic saline. Osmotic demyelination has been described as a brain damage caused because of aggressive, excessive and rapid sodium levels correction¹⁵. Rapid correction of the levels of serum sodium was also associated with rhabdomyolysis in patients with water intoxication. Therefore, in order to avoid complications, precise and vigorous control of the serum sodium levels might be needed in such patients¹⁶.

Conclusion

Polydipsia in psychiatric patients could lead to water intoxication and severe hyponatremia. Antipsychotics in patients with schizophrenia may cause hyponatremia by themselves. Acute onset severe hyponatremia should be managed in ICU. Strict control of serum sodium levels is a must in order to avoid osmotic demyelination and rhabdomyolysis. Correction of the serum sodium level should be performed with hypertonic saline (NaCl 3%) and correction must not exceed 10 mmol/L in the first 24 hours and 8 mmol/L in the next days. If using 10% hypertonic saline in therapeutic measures adjustment of the doses should be performed in order to provide safe treatment and avoid osmotic demyelination and further brain damage.

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