

Rhabdomyolysis due to Severe Hyponatremia Caused by Dehydration, in a Child with Gastroenteritis: a Case Report

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Abstract

Background

Rhabdomyolysis is considered a rare medical condition in pediatric population.

Case Report

We report our experience on a one year old girl referred to Shiraz Nemazee Hospital, Southern Iran with rhabdomyolysis due to severe hyponatremia, secondary to gastroenteritis.

Discussion

Rhabdomyolysis should be taken in to consideration in hyponatremic states, as it may lead to severe consequences. Treatment of underlying cause and proper management of hyponatremia could be helpful while handling this complicated situation.

Key Words: Child, Hyponatremia, Gastroenteritis, Rhabdomyolysis.

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1-INTRODUCTION

Rhabdomyolysis is a medical condition in which, damage to the cells of skeletal muscle leads to release of intracellular components (including myoglobin) in to the general circulation(1). Although muscular pain along with weakness and dark urine are considered the typical characteristics of this condition, clinical presentation of rhabdomyolysis can vary from asymptomatic rise in creatine phosphokinase (CPK) levels to acute renal failure and life threatening hypovolemic shock (2). Rhabdomyolysis can arise secondary to a wide range of disorders, trauma, medications, toxins, electrolyte imbalances, etc. In pediatric cases the most common causes are infection and inherited disorders (2, 3). Hypernatremia is an electrolyte disorder that is considered as a cause of rhabdomyolysis but this sequence is rarely reported, particularly in children (4-6). Here we report our experience on a pediatric patient with rhabdomyolysis due to severe hypernatremia, secondary to gastroenteritis.

2- CASE REPORT

A one year old girl was referred to Shiraz Nemazee Hospital, Southern Iran due to severe hypernatremia (serum sodium level > 190 meq/L) which was detected in primary health care unit. She had a positive history of diarrhea and vomiting prior to her admission. Patient's parents reported that diarrhea had started almost 20 days ago and had become more severe after one week, meanwhile vomiting was added to the situation. Recently her mother had noticed that patient's urine has become dark in color but there was negative history of oliguria. She had received medications including oral rehydration solutions (ORS), Ondansetron, Zinc sulfate and Cefixime before admitting to hospital. Taking past medical history did not reveal any previous illnesses and/or congenital disorders.

Family history didn't show any great significance. Physical examination was consistent with moderate-severe dehydration. Patient was not febrile with pulse rate of 120/minute, respiratory rate of 24/minute and systolic blood pressure of 90 mmHg. Her weight was 9kg. She was irritable with dry mucosal surfaces. Mild periorbital edema was noticed. Abdominal examination showed mild abdominal tenderness and distention. Mild pitting edema (1+) was present in both lower extremities. Digital rectal exam was consistent with some fecal impaction.

On laboratory examinations, serum sodium level was 197meq/L. other lab tests showed a serum hemoglobin level of 11.6 g/dl, total white blood cell count of 11,200/mm³, and platelet count of 140,000/mm³. Other lab findings were: blood urea nitrogen (BUN) of 26 mg/dl, creatinine of 1.2 mg/dl, potassium 4 meq/l, serum calcium 8.7 mg/dl, serum phosphorus 3.9 mg/dl, erythrocyte sedimentation rate (ESR) was normal and C-reactive protein (CRP) level was mildly elevated. Also lab findings showed elevated serum CPK and liver enzymes level: CPK 8072 U/L, aspartate aminotransferase (AST) 1890 U/L, alanine aminotransferase (ALT) 2060 U/L, and lactate dehydrogenase 9540 U/L. Total bilirubin and direct bilirubin levels were 0.3 and 0.1 mg/dl respectively and serum albumin level was 4.3 g/dl. Urinalysis showed 28-30 red blood cells per high-powered field, protein 2+ and blood 3+. Stool exam showed many fatty droplets with trace occult blood. Peripheral blood smear revealed moderate hypochromic anemia with adequate platelet count and normal white blood cell count.

Normal Level of immunoglobulin M (IgM) antibody against hepatitis A virus was used to rule out acute hepatitis A infection. Patient was admitted and management of hypernatremic dehydration started with hypotonic intravenous fluid in order to

decrease the serum Na 10 meq/l daily. By 2nd and 3rd days of treatment the serum sodium level was decreased to 186 and 175 meq/l respectively. A 24-hour urine collection showed proteinuria which was consistent with acute kidney injury (urine protein 12600 mg/24hours, and urine creatinine 880 mg/24hours).

Due to patient's severe irritability, an abdominopelvic ultrasonography was done that was ruled out the possibility of intussusceptions. However, radiologist reported that liver was normal in size and echogenicity without bile duct dilatation. Kidneys were both approximately 6.6 cm in size without stone, stasis or collection.

Brain CT scan and lumbar puncture were also performed and they showed normal results. Laboratory findings on 4th day of hospital admission showed a decrease in elevated liver enzymes: alanine aminotransferase 679 U/L, aspartate aminotransferase 758 U/L, and Creatine phosphokinase (CPK) decreased to 1219 U/L. Meanwhile serum lactate dehydrogenase level was 2,500 U/L.

During the following days of treatment, as the serum sodium level gradually decreased towards normal levels, proteinuria was resolved and CPK levels were dropped along with liver enzyme levels. Patient was discharged by one week with serum sodium level of 142- meq/l, AST 64 U/L, ALT 320U/L, LDH 1672. All laboratory values were normal in second week of patient's follow up and total recovery was achieved.

4- DISCUSSION

Rhabdomyolysis is considered a rare medical condition in pediatric population (7). Still the mechanism of rhabdomyolysis caused by hypernatremia is not well understood, however one theory implies it could be associated with inhibition of electrogenic sodium pumps due to hyperosmolar state. This will further

impair sodium calcium transport (6). Increased intracellular calcium ions can damage the muscular cell via several pathways including persistent contraction, activation of phospholipase A₂ (PLA₂) and free oxygen radical production (1). On the other hand, rhabdomyolysis itself can lead to a more severe hypernatremia (8). This phenomenon could partly contribute to the severely high serum sodium level in this case in addition to gastroenteritis and dehydration.

Released myoglobin from the damaged muscular cells might deposit in renal tubules and therefore cause acute tubular necrosis (9). Actually this fact along with dehydration was the most likely causes of acute renal failure and subsequent proteinuria in the patient presented herein. It is worth mentioning that in the patient the more common causes of rhabdomyolysis such as crush injury, toxins and drugs were excluded using history, physical examinations or laboratory findings. In this case rhabdomyolysis and acute renal failure were resolved after treatment of hypernatremia.

Rhabdomyolysis as a result of hypernatremia, such as in this case, is reported rarely. One similar case was reported by Mastro-Martinez et al., sharing the experience on an infant with diarrhea who developed rhabdomyolysis secondary to hypernatremia (7).

5- CONCLUSION

Rhabdomyolysis should be taken in to consideration in hypernatremic states, as it may lead to severe consequences. Treatment of underlying cause and proper management of hypernatremia could be helpful while handling this complicated situation.

6- CONFLICT OF INTEREST: None.

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