

Case Report

Addisonian crisis complicated by rhabdomyolysis and acute kidney injury

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ABSTRACT

Addisonian Crisis is an endocrine emergency requiring hospitalization and urgent management. The diagnosis is usually complicated by nonspecific sign and symptoms of primary adrenal insufficiency. A high degree of suspicion is required in such cases. Rhabdomyolysis caused by addisonian crisis is a rare complication which was seen in our patient.

Keywords: Addisonian crisis, Rhabdomyolysis

INTRODUCTION

Patients with Addison disease may have musculoskeletal symptoms including muscle weakness, flexion contractures, and hyperkalemic myopathy.¹ However, the association between rhabdomyolysis and acute adrenal insufficiency is unusual and is almost always observed in patients with severe hyponatremia. Rhabdomyolysis is a well-recognized etiology of acute renal failure and hence it accentuates the adversity of acute adrenal insufficiency. We herein report the case of a 40 years old patient with acute primary adrenal insufficiency, which was complicated by rhabdomyolysis.

CASE REPORT

A 68-years-old female with the past medical history of adrenal insufficiency, ischemic heart disease status post stenting, COPD, hypothyroidism and hypertension who presented to emergency with the complaints of nausea, vomiting and severe generalized abdominal pain. She started having abdominal pain two days ago which progressively got worst. Abdominal pain was followed by

severe nausea and vomiting. She also had 2 episodes of dark colored vomit along with malena. She denied any fever, chills, seizures or fall. She was found to have low blood pressure (70/30mm Hg), for which 2 liters of normal saline was given.

Her baseline investigations showed Creatinine of 7.4mg/dl, BUN of 72 mg/dl, bicarbonate of 14 meq/l and sodium of 120 meq/l. She had normal sodium level and kidney function tests 6 months ago. Rest of the labs including, CBC, liver function tests, lipase, amylase and urine drug screen were unremarkable. Her CK total was more than 5000 IU/L for which myoglobin was ordered which was also significantly high (>5000 ng/ml). She was admitted in the ICU and given aggressive intravenous fluids. Pancultures were ordered which did not grow any bacteria. CT scan of abdomen showed gallstones only without any evidence of pancreatitis or cholecystitis (Figure. 1).

Detailed history revealed that she was not taking fludrocortisone for the last 9 months. She was started on hydrocortisone 50 mg IV four times a day for addisonian crisis. She was continued on intravenous fluids for

rhabdomyolysis and acute kidney injury. She underwent upper GI endoscopy and colonoscopy for hematemesis and positive occult blood test respectively; both endoscopic procedures were unremarkable. Her kidney function improved to normal and hyponatremia resolved within 1 week. No underlying cause of her rhabdomyolysis was identified. She denied any fall, trauma or ambulatory dysfunction. Medication review indicated that she was on Lipitor 40 mg daily but she had been taking this medication for 5 years with no side effects. Also, her CK total and liver functions 6 months ago were normal. Her rhabdomyolysis was considered secondary to Addisonian crisis induced hyponatremia.



Figure 1: CT scan of abdomen without contrast showed no acute intra-abdominal pathology.

DISCUSSION

Addison's disease is an endocrine disease characterized by the slow development of a constellation of nonspecific symptoms such as muscle weakness, fatigue, abdominal pain, salt craving, light-headedness, nausea, vomiting, diarrhea and changes in personality.^{1,2} In addition to the deficiency of adrenocorticotropic and hypothalamic hormones, adrenal insufficiency can be caused by immunologic, vascular and infectious diseases.

Patients with untreated Addison's disease can develop Addisonian crisis. It can be triggered by rapid withdrawal of exogenous steroids or with conditions requiring increased endogenous steroids such as infections, trauma, and other concomitant severe medical illnesses.³ Sudden and intense development of symptoms like penetrating pain in legs, vomiting, diarrhea, hypoglycemia, hyponatremia, and syncope are characteristic of Addisonian crisis. There is excessive salt wasting in the urine of patients due to deficiency of aldosterone and cortisol, which leads severe hyponatremia.⁴ Consequences of hyponatremia range from salt craving to convulsions and rhabdomyolysis but are highly variable, depending on its severity.

Hyponatremia in Addisonian crisis is mediated by an increase in the release of anti-diuretic hormone (ADH), which results in the reduction in the plasma sodium concentration due to water retention. The hypersecretion of ADH seen in cortisol deficiency is due to increase in hypothalamic secretion of corticotropin-releasing hormone (CRH) and may be due in part to the reduction in systemic blood pressure and cardiac output (mechanism unknown).⁵ The mechanism of rhabdomyolysis due to hyponatremia is thought to be a result of hypo-osmolality of extracellular fluid causing myocytes swelling. This leads to extrusion of intracellular potassium leading to its depletion in myocytes. In potassium-depleted cells, the transmembrane potential is decreased which leads to the release of creatine kinase and myoglobin. Hypokalemia also leads to diminished muscle perfusion as potassium is a vasodilator and mediates hyperemic response during exercise, resulting in cramps, ischemic necrosis, and rhabdomyolysis.⁶ Second, the Na-Ca exchange pumps are disturbed due to the decreased extracellular sodium. This leads to increased intracellular calcium levels because of reduced extracellular sodium to be exchanged causing the cell death by the release of lipase and proteases.⁷⁻⁹

Rhabdomyolysis is a syndrome characterized by muscle necrosis leading to the release of intracellular constituents into the circulation. The severity of disease ranges from asymptomatic serum elevation of muscle enzymes to life-threatening disease associated with electrolyte imbalances and acute kidney injury. Traumatic causes include crush injuries, strenuous exercise, and non-traumatic causes are mainly metabolic and genetic disorders. Statins, fibrates, ciclosporin and antipsychotic medications increase rhabdomyolysis risk.¹⁰ Myoglobin accumulation in renal tubules leads to acute kidney injury.¹¹ Complications of rhabdomyolysis include acute tubular necrosis, acute renal failure, and shock; however, management depends on its cause but the main goal is to prevent kidney injury. High volumes of intravenous fluids are given along with diuretics to excrete myoglobin and to reduce contact time of myoglobin in renal tubules because of toxicity of myoglobin to tubules. Bicarbonate can be used to minimize myoglobin's toxic effects. The inadequacy of supportive treatment warrants uses of renal replacement therapy and modalities of which include hemodialysis, peritoneal dialysis and continuous hemofiltration.¹⁰ Hyponatremia due to water intoxication and SIADH is reported to cause rhabdomyolysis.^{12,13} Many conditions are associated with rhabdomyolysis however it is rarely associated with the Addisonian crisis. Hyponatremia due to any cause can result in convulsions, which in turn may lead to muscle breakdown and ultimately renal failure.

Our patient presented with Addisonian crisis that was complicated by rhabdomyolysis. Her sodium level was found to be 120 meq/l, which we believe was the cause of rhabdomyolysis in our patient. Detailed history, physical exam and lab testing could not provide an alternated

explanation of rhabdomyolysis. She was given IV steroids and supportive treatment with complete return of kidney functions and sodium levels within 1 week.

CONCLUSION

Acute adrenal insufficiency due to any etiology may cause severe hyponatremia, which can rarely lead to rhabdomyolysis. Muscle enzymes might be monitored in patients with acute hyponatremia who develop myalgias and muscle weakness. Hyponatremia-induced rhabdomyolysis might be a possibility in such cases. A high degree of suspicion and low threshold for ordering myoglobin levels are required to diagnose such complications to prevent grave consequences.

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