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Case Report

Can McArdle disease be induced by infection associated with rhabdomyolysis case report?

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Abstract

Introduction: Rhabdomyolysis is the clinicopathologic presentation that is caused by striated muscle damage with leaking of toxic pigments into the blood. Clinical findings of rhabdomyolysis ranges from asymptomatic increase of muscle enzymes to the fatal forms of acute renal failure (ARF) and electrolyte disturbance. The causes of rhabdomyolysis can be classified under several groups: (a) direct injury from trauma, (b) overuse of muscle, (c) congenital muscle enzyme errors, and (d) uncommon pathogens like drugs and toxins, ischemia of muscle, metabolic and endocrine disorders, and finally infections. The diagnosis of rhabdomyolysis is based on myalgia and weakness, tea-colored urine (myoglobinuria), an increase in muscle enzymes that is not caused by myocardial infarction (MI) or polymyositis (PM). The most important goal of treatment is to diagnose and cure complications like electrolyte disturbances and acute tubular necrosis (ATN).

Case Report: The case in the present study was a young man who was admitted with fever and chill and generalized myalgia with tea-colored urine. Finally, he died because of acute adult respiratory distress syndrome (RDS), acute renal tubular necrosis, and hyperkalemia. Among congenital muscle enzyme disorders, the most common type is the carnitine-palmitotransferase defect (CPT), and after that, the second most common is myophosphorylase deficiency. Among muscle carbohydrate metabolism defects, McArdle disease (MD), or glycogenosis type V (GSD-V), is the most prevalent, which is caused by a deficiency in glycogen-phosphorylase enzyme. In this disorder, only striated muscles are affected, which leads to rhabdomyolysis in the primary steps, and exercise intolerance, muscle cramps, and myoglobinuria, when reaches more developed levels. This predisposition to rhabdomyolysis in patients with MD prevents us from administrating certain drugs in these cases.

Conclusion: Rhabdomyolysis may develop among individuals with abnormal muscles as in congenital diseases of glycogenolysis, glycolysis, purine, and lipid metabolism. Considering the rare causes of rhabdomyolysis like MD permits physicians to diagnose and treat this potentially fatal occult causes.

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Introduction

Rhabdomyolysis is the clinicopathologic presentation caused by striated muscle damage and leaking of toxic pigments into the blood.[1] Clinical finding of rhabdomyolysis ranges from asymptomatic increase of muscle enzymes to the fatal forms of acute renal failure (ARF) and electrolyte disturbance. The causes of rhabdomyolysis can be classified under many groups: (a) direct injury from trauma, (b) overuse of muscle, (c) congenital muscle enzyme errors, and (d) uncommon pathologic etiologies like drugs and toxins, ischemia of muscle, metabolic and endocrine disorders, and finally infections.[2] The diagnosis of rhabdomyolysis is based on myalgia and weakness, tea-colored urine (myoglobinuria), increase of muscle enzymes that is not caused by myocardial infarction (MI) or polymyositis (PM). The important treatment objective of rhabdomyolysis is to diagnose

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and treat its complications likely electrolyte disturbances and acute tubular necrosis (ATN). Rhabdomyolysis may develop among individuals with abnormal muscles as in congenital diseases of glycogenolysis, glycolysis, purine, and lipid metabolism.[3] Enzyme deficiencies like carnitine palmitoyltransferase (CPT) were the most common followed by myophosphorylase deficiency. This is called the glycogenosis type V (GSD-V). It is historically labeled as McArdle disease (MD).[4,5]

Case Report

The 20-year old man referred from Ajabshir City (one of the cities of East Azerbaijan Province of Iran) hospital with a diagnosis of acute respiratory tract infection with dry cough and dyspnea. A few days prior to this presentation, he had history of fever and chill and generalized myalgia with tea colored urine. 2 weeks ago, he was well without history of previous respiratory and urinary tract symptoms and icterus. He had not any history of use of prescribed over-the-counter (OTC) illicit drug herbal medicines like nonsteroidal anti-inflammatory drugs (NSAIDs), no history of joint, and skin findings like arthralgia or jaundice. The physical examination performed disclosed generally healthy looking man. His vital signs were as follows blood pressure = 120/85 mmHg, pulse rate = 82 beats per minute, respiratory rate = 16 per minute, body temperature = 38.5 °C, and trace leg oedema. His mucous membranes were dehydrated with generalized tender muscles without any muscle atrophy or weakness.

Rheumatological examination for evaluation of connective tissue disorder was inconclusive. Respiratory system examination showed shallow breathing bilaterally and the pleural effusions were not detected. Cardiovascular system examination revealed no sign of cardiac disease. Point of maximal impulse was in five intercostal space at mammillary line, S1, S2 of heart sounds were present with no murmurs, and no pericardial friction rub. Abdomen examination was performed which revealed soft distended tender on deep palpation diffusely without organomegaly or ascites. Bowel sounds were present. Central nervous system examination was performed and showed that he was alert and orientated without any focal neurologic finding. Urine dipstick revealed dark urine with moderate blood without protein or white blood cell (WBC). Intake and output control showed that urine had been gathered in urine bag with volume of 150 ml of dark urine.

His problem list included a 20-year-old man, previously healthy, with tea colored urine possibly due to hematuria accompanied by ARF along with acute respiratory tract infection with dyspnea. Clinically, he was dehydrated with Kussmaul breathing and evidence of acute PM.

Investigations were performed and showed complete blood count as follows: WBCs, platelet, and hemoglobin (Hb) with values of 12000 /mm^3, 368 × 1000/mm^3, and 12.6 gm/dl, respectively. Arterial blood gas (ABG) analysis revealed partial pressure of oxygen (PaO_2), partial pressure of carbon dioxide ($PaCO_2$), arterial blood pH, oxygen saturation (SaO_2), and bicarbonate (HCO3) with rates of 75 mmHg, 55 mmHg, 7.25, 75%, and 12 mEq/l, respectively. An electromyography (EMG) with nerve conduction study (NCS), and nerve conduction velocity (NCV) test were performed, which recorded the electrical signals passing through muscles. Interpretation of EMG-NC showed that some compound muscle action potentials (CMAPs) were low amplitude and the others were normal. Myogenic changes were noted in some sampled muscles. These findings were compatible with acute myogenic process in majority of muscles which was mostly suggestive of generalized myositis. The underlying causes of myositis causing rhabdomyolysis including hypokalemic periodic paralysis (hypoKPP), endocrine and metabolic causes like myopathies such as MD disease, neuroleptic malignant syndrome (NMS), and viral infection were subjected.

Briefly, the case study was a 20-year-old man with ARF secondary to rhabdomyolysis associated with upper respiratory tract infection (URTI). Clinically, he was acidotic, dehydrated and oliguric. Paraclinic workup showed metabolic acidosis, hyperkalemia with electromyographic changes mostly suggestive of generalized myositis. He was placed on for hemodialysis. Ultrasound study showed kidneys which were grossly normal with increased cortex echogenicity without hydronephrosis. Urine analysis showed many red blood cells with epithelial cell casts. Medical management of the patient was began with following orders starting with his hemodynamic status evaluation. It was performed by central venous pressure (CVP) line insertion. In the study case, his CVP reading was unmeasurable. He was prescribed physiologic normal saline infusion.

The goal of the study was to stabilize CVP line pressure at 14 cm. The second objective was correcting electrolyte imbalances, including hyperkalemia. Intravenous calcium was began to prevent cardiac complications of hyperkalemia, intravenous glucose and insulin infusion were performed to enhance potassium uptake by cells and correct severe metabolic acidosis with sodium bicarbonate. Intake and output monitoring were requested. Urine sample was sent for microscopy, culture and sensitivity, and searching for pigmented crystals. Renal ultrasonography was asked to evaluate renal size and rule out obstructive uropathy.

Follow up result of the case was as follows: he died with acute respiratory failure against aggressive medical treatment with adult respiratory distress syndrome (RDS), acute renal tubular necrosis, and hyperkalemia.

Discussion

Rhabdomyolysis is the clinicopathologic presentation caused by striated muscle damage and leaking of toxic pigments into the blood. Clinical findings of rhabdomyolysis ranges from asymptomatic increase of muscle enzymes to the fatal forms of ARF and electrolyte disturbance. The causes of rhabdomyolysis can be classified under many groups, including: (a) direct injury from trauma (b) overuse of muscle, (c) congenital muscle enzyme errors, and (d) uncommon pathologic etiologies like drugs and toxins, ischemia of muscle, metabolic and endocrine disorders, and finally infections. The diagnosis of rhabdomyolysis is based on the myalgia and weakness, tea colored urine (myoglobinuria), increase of muscle enzymes that is not caused by MI or PM. Important treatment aim was to diagnose and treat complications like electrolyte disturbances and ATN.[4,5]

The study case differential diagnosis included primarily ARF due to hypovolemia secondary to acute respiratory infection (ARI) with dyspnea, which was less acceptable due to his history and clinical findings. Secondly, the pigment-induced acute kidney injury (AKI) was possible diagnosis. Kidney injury may be due to myoglobinuria. Rhabdomyolysis was final consideration. Rhabdomyolysis causes includes acute versus chronic muscle injury, trauma, pressure necrosis, and electric shock. Other conditions may be considered like myofiber exhaustion states with severe seizures, excessive exercise, and thermal exhaustion. Exposure to chemicals and medicines included toxins including alcohol, cocaine, heroin, amphetamines, ecstasy, snake bite, etc.[6-8] Infectious agents like viral or bacterial agents with pneumonia infections should be ruled out. Rare diagnosis like familial malignant hyperthermia, MD, hypothyroidism, PM, and dermatomyositis can be suggested. Myofiber exhaustion occasionally can be seen with subclinical myoglobinaemia, myoglobinuria, and increased creatine phosphokinase (CPK) titer.[6,9,10] It is may be observed even after usual activities. Rhabdomyolysis has been associated with a variety of electrolyte disorders, particularly hypokalemia, hypophosphatemia, and hyponatremia. Patients with idiopathic myoglobinuria must be sent for muscle

biopsy. Enzyme deficiencies like CPT were the most common cases, followed by myophosphorylase deficiency.[9-11]

Rhabdomyolysis may develop among individuals with abnormal muscles as in congenital diseases of glycogenolysis, glycolysis, purine and lipid metabolism. Enzyme deficiencies like CPT was the most common cases, followed by the myophosphorylase deficiency. This was called GSD-V. It was historically labeled as MD.[12,13]

Conclusion

The case in the present study was a young man who presented fever and chill and generalized myalgia with tea colored urine. Interpretation of EMG-NC showed some CMAPs indicating generalized myositis. The underlying causes of myositis leading to rhabdomyolysis included metabolic causes like myopathies with enzyme deficiencies like MD. Finally, he was expired with acute respiratory failure with adult RDS, acute renal tubular necrosis, and hyperkalemia.[2]

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Conflict of Interest

There was no conflict of interest.

Ethical Approval

Not indicated.

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