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Case Report

CT of rhabdomyolysis as a sequela of drug abuse ☆,☆☆,★

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ABSTRACT

Rhabdomyolysis is distinguished by the breakdown of skeletal muscle and the subsequent release of intracellular components into the bloodstream. Necrosis, which commonly manifests as muscle swelling, weakness, and myalgia, is a symptom associated with both traumatic and non-traumatic rhabdomyolysis. The etiology may be considered acquired, including trauma, infections, and drugs, or genetic, such as myopathies or enzyme deficiencies. Diagnosed by exceedingly high creatine kinase levels, rhabdomyolysis can lead to increased serum levels or more worrisome complications such as life-threatening acute kidney injury. In this article, we report the case of a 56-year-old male with a diagnosis of severe rhabdomyolysis and acute renal failure. We focus on optimizing diagnosis through the application of radiological modalities.

Introduction

Rhabdomyolysis is characterized by the dissolution of skeletal muscle and the subsequent release of intracellular content into circulation [1]. Extensive necrosis, evidenced by muscle weakness, inflammation, myalgia, and gross pigmenturia without hematuria, is a symptom of rhabdomyolysis [2]. Consequently, patients are susceptible to increased serum levels of enzymes such as creatine kinase (CK), aspartate transaminase (AST), as well as lactate dehydrogenase (LDH), electrolyte and metabolic abnormalities, and, most alarming, acute kidney injury (AKI). In addition to traumatic sources of injury, non-traumatic causes include alcohol, illicit drugs, and pharmaceutical agents [3]. Given that skeletal muscle constitutes approximately 40% of body mass, the breakdown of the sarcolemma membrane and the ensuing release of high concentration muscle-cell content into the bloodstream can easily overcome the body's response mechanism [4,5].

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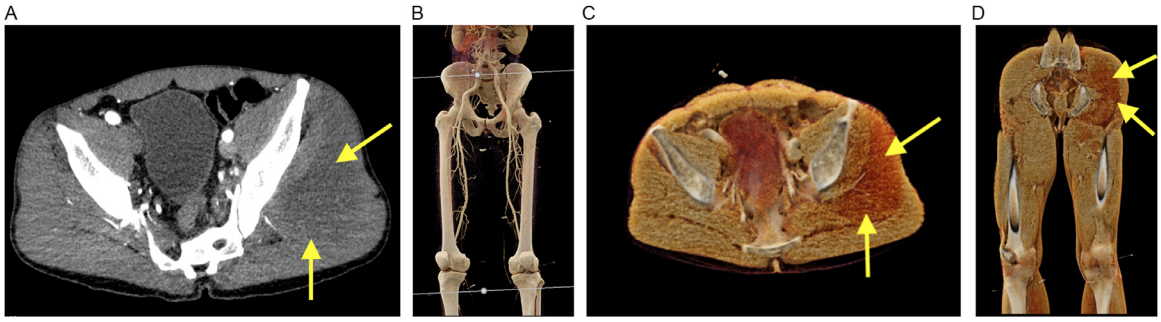

Fig. 1 – A 56-year-old male with a past history of polysubstance abuse presented with left lower extremity numbness and pain following heroin use. A CT angiogram to rule out any vascular process including aneurysm or pseudoaneurysm was performed. (A) Axial CT demonstrates enlargement of the left gluteus muscles (arrows) with decreased enhancement especially of the gluteus medius noted. (B) CT angiogram using cinematic rendering demonstrates no evidence of vasculitis or the presence of aneurysm. (C) Cinematic rendering of the pelvis demonstrates the enlargement best defined in the gluteus medius muscle (arrows) with changes in texture mapping of the muscle due to rhabdomyolysis. (D) Cinematic rendering in coronal plane defines the extent of involvement of the gluteal muscles (arrows) with decreased attenuation of the affected muscles defined.

Case report

A 56-year-old male with a history of polysubstance abuse presented to his local hospital due to lower left extremity numbness and pain following intranasal heroin use during the previous night, 3 months after using heroin following 14 years of abstinence. The patient was admitted with a severe episode of rhabdomyolysis as labs were notable for a CK of 84,825 U/L, a sticking variance from the 24-195 U/L reference range, and consequential acute renal failure (ARF) and myositis. A computed tomography angiograph scan showed an asymmetric enlargement and hypodensity of the patient's left gluteus muscles (Fig. 1), especially gluteus medius muscle, as well as adductor and vastus lateralis muscle, which was consistent with myositis and rhabdomyolysis. Initial imaging demonstrated absent blood flow in the distal tibial and peroneal arteries; however, a repeat run displayed patency of these vessels, which evidenced slower flow on the left leg, likely due to compression by muscle edema. Additionally, there was also edema and expansion of the adductor muscle compartment, including the left quadratus obturator, piriformis, and femoris muscles. Ultimately, the individual was placed on intravenous fluids, which stabilized his CK blood serum levels, and a follow-up kidney biopsy revealed mild acute tubular injury and mild arteriolar hyalinosis. The patient was discharged exactly a week after admission.

Findings of rhabdomyolysis in CT include heterogeneously enlarged asymmetric muscles with hypodense, poorly enhancing foci, which may calcify in the subacute or chronic phases of the disease [6]. Mild, linear enhancement may be observed after contrast administration. Similarly, our case showed enlargement, edema, and decreased enhancement of the gluteus medius and minimus muscles as well as the adductor magnus and vastus lateralis muscles. However, these findings are nonspecific and may be seen in other disease processes such as pyomyositis and autoimmune myositis.

Discussion

This article reviews a case of rhabdomyolysis. Rhabdomyolysis is characterized by the release of exceedingly concentrated intracellular content, including myoglobin, electrolytes, and other sarcoplasmic proteins, from highly necrotic muscle tissue into the blood stream. Symptoms generally include non-specific features such as swelling, stiffness, and pain, which could be present in any part of the body, but typically presents in the proximal leg muscles [7]. Clinical presentations are contingent on the severity of muscle damage. Therefore, patients can present with asymptomatic features including increased serum levels or more alarming complications including hypocalcemia, hyperphosphatemia, hyperkalemia, and AKI. The incidence of AKI in patients with rhabdomyolysis ranges from 13% to 50%, as individuals that have undergone trauma and had a history of illicit drug use or alcohol abuse have been found to be more susceptible to AKI [2,5,8].

We report a 56-year-old male who presented with lower left extremity weakness and pain following intranasal heroin use. Subsequently, the patient was found to have severe rhabdomyolysis with CK levels of 84,825 U/L, ARF, and left gluteal myositis. Definite rhabdomyolysis diagnoses are ascribed to serum CK levels >1000 U/L or 5 times the upper baseline limit. Rising CK levels, or failure of levels to diminish following the onset of therapy, indicate muscle injury or renal failure. Thirty-three percent of patients experience rhabdomyolysis-induced ARF, which occurs as a result of the nephrotoxic effects of myoglobin, tubular obstruction, acidosis, aciduria, or hypovolemia. Urine myoglobin and serum CK, creatinine, calcium, and potassium levels are possible predictors of ARF [9]. Furthermore, the etiology of rhabdomyolysis may be attributed to acquired sources such as trauma, infections, and drugs, or genetic factors including myopathies and enzyme deficiencies. More specifically, as seen in this case, recreational drug use may lead to the dissolution of skeletal muscle either by enhancing the permeability of the sarcolemma,

thus releasing intracellular contents, or by altering adenosine triphosphate (ATP) production. Recreational drugs implicated in rhabdomyolysis include heroin, cocaine, hallucinogens, lysergic acid diethylamide, methamphetamines, and amphetamines [5].

Rhabdomyolysis is a clinical diagnosis that involves a thorough history, physical exam, and serum chemical workup. Elevated serum CK 5 times the normal value is 100% sensitive for the diagnosis of rhabdomyolysis [10]. Thus, imaging plays a secondary role and instead supports the diagnosis of rhabdomyolysis and rules out other pathologies that may present with similar clinical findings.

Patient consent

The patient reported in the manuscript signed the informed consent/authorization for participation in research which includes the permission to use data collected in future research projects including presented case details and images used in this manuscript.

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