Case in Point

Imaging Use in Focal Rhabdomyolysis of the Left Shoulder

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Magnetic resonance imaging and sonography are useful tools for the diagnosis and assessment of this rare musculoskeletal condition.

Rhabdomyolysis involves the breakdown of skeletal muscle with the release of intracellular contents into the extracellular space and circulation.[1] Diffuse rhabdomyolysis has been found in athletes due to overexertion. However, focal rhabdomyolysis is rare.[2,3] The clinical presentation of focal rhabdomyolysis is subtle and nonspecific, with swelling, vague pain, weakness, fatigue, and tea-colored urine.

Early recognition and prompt management are crucial to prevent complications such as compression syndrome, acute renal failure, disseminated intravascular coagulation, cardiac dysrhythmia, or even cardiac arrest. Sonography and magnetic resonance imaging (MRI) can, therefore, be a complementary part of the diagnosis and assessment of the extent of rhabdomyolysis.[4-7]

CASE HISTORY

The patient was a 34-year-old white man with a history of polysubstance abuse who presented to the emergency department (ED) with numbness and weakness in the left arm and hand, pain in the left side of his neck, and 3 days of intermittent amnesia with confusion. He had used IV heroin about 2 weeks prior to admission and used tobacco and alcohol daily. He reported no current medications or known allergies. The patient was in a monogamous relationship with a same-sex partner.

On physical examination, vital signs were within normal limits. He was in distress, confused, and disoriented as to time and place. An extremity examination revealed 1/5 strength in the extensors of the left elbow, left wrist, and left fingers with normal strength noted in the right upper extremity as well as the lower extremities. No sensory deficits were noted. The patient's skin was warm and dry. Remarkable laboratory findings included creatine kinase (CK) 1,744 U/L, creatinine (Cr) 1.9 mg/dL, ALT 1,065 U/L, AST 319 U/L, ALP 159 U/L. A urine toxicology screen was positive for cocaine and opiates, and the urine analysis dip was negative for red blood cells, white blood cells, and protein. A differential diagnosis favored a left arm inflammatory reaction to IV drugs, although rhabdomyolysis was questioned.

A neurology consult was obtained, and a bedside electroencephalography test was performed in the ED by the neurologist, showing mild left occipital slow wave abnormality with no epileptiform discharges. A chest X-ray and computed tomography (CT) scan of the head and cervical spine were unremarkable, other than incidental mild prominence of the ventricles.

Over the next 24 hours, the patient was hydrated with IV normal saline without bicarbonate. His altered mental status, urine output, and biochemical abnormalities returned to normal, except for the serum CK, which decreased to 917 U/L. He had minimal improvement in his left upper extremity nerve palsy symptoms; however, he was deemed to be stable for discharge with follow-up in the clinic.

Instead of a clinic follow-up, the patient returned to the ED 7 days later, with progressive weakness of the left arm, forearm, and wrist. The patient noted that his weakness was so significant that he had to move his left arm with his right arm. He also reported extremity swelling and increasing paresthesias involving the lateral aspect of his left arm and

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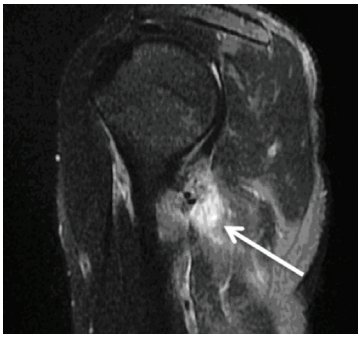

Figure 1A. MRI Sagittal T2 fat-saturated image of the left shoulder. Hyperintense area (arrow) in the triceps muscle.

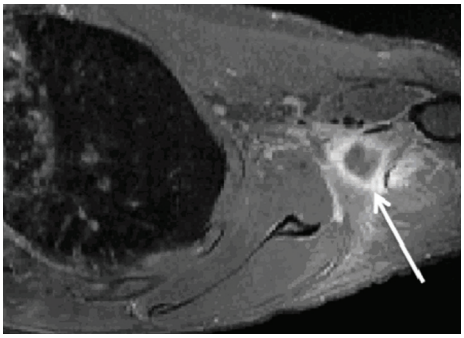

Figure 1B. MRI Axial T1 fat-saturated postcontrast image of the left shoulder. This area is peripherally enhancing and centrally nonenhancing (arrow).

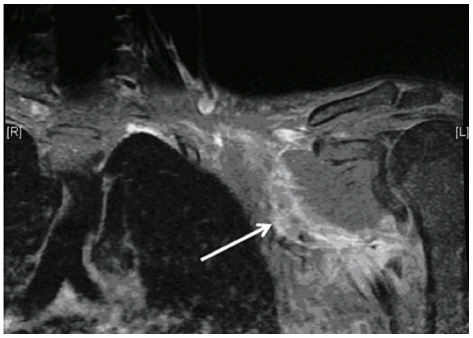

Figure 1C. MRI Coronal T1 fat-saturated postcontrast image of the left shoulder. Enhancing tissue in the region of the brachial plexus can be seen (arrow).

hand, dizziness, and left neck pain. A physical examination revealed 3/5 strength at the left deltoid and left triceps, and 0/5 strength in the left fingers and grip. Remeasurement of CK was 54 U/L and Cr was 0.9 mg/dL. Compartment pressures were not measured.

Magnetic resonance imaging using multiplanar spin echo T1 and fast spin T2 weighted and post-IV 16cc Omniscan contrast sequences of the left shoulder were performed, showing multiple patchy T2 hyperintense focal areas with peripheral enhancement in the muscles of the posterior shoulder and in the tissues adjacent to the brachial plexus in the neck and shoulder (Figures 1A, 1B, and 1C). Sonography with matrix array linear 6-15 MH3 transducer was performed, which demonstrated patchy focal hypoechoic areas of muscle with enlarged, thickened, and disrupted muscle, representing devitalized muscle without any drainable fluid collection or abscess (Figures 2A, 2B, 2C, and 2D).

Magnetic resonance imaging and magnetic resonance angiogram scans of the brain and cervical spine with and without contrast were unremarkable. At that time, a definitive diagnosis was made of focal rhabdomyolysis and compressive neuropathy of the brachial plexus posterior cord, leading to brachial plexopathy of the left shoulder.

The patient was treated with hydration, a left arm sling, elevated left arm, and ibuprofen 600 mg qid to reduce inflammation. His swelling decreased markedly, and there was a slight improvement in pain and mobility at a 2-week neurology clinic follow-up. The patient lost contact after that.

DISCUSSION

Rhabdomyolysis is caused by diverse etiologies. Most commonly, it is generalized and occurs due to overexertion, crush injury, steroid use, metabolic abnormalities, and certain medications and illicit drugs.[1,2] The most likely etiology of rhabdomyolysis in patients presenting to the ED without significant trauma is of substance abuse, especially with ethanol, heroin, amphetamines, cocaine, and other sedatives or stimulants.[1-3] The patient presented in this case study had a history of drug abuse, with a positive urine toxicology screen for cocaine and opiates. He had been intermittently confused and amnesic for 3 days prior to presentation, during which he may have been lying on his shoulder for a prolonged period.

Focal rhabdomyolysis and acute compression at the posterior shoulder leading to compressive brachial plexopathy is rare, with only 3 cases reported in the literature, all occurring with IV drug use.[1-3] This patient had compression of the brachial plexus posterior cord from rhabdomyolysis and prolonged immobilization. Intravenous drug abusers may delay medical care due to perceived illicit drug effects and frequently present to the ED confused, agitated, or obtunded. Acute extremity swelling, a palpable lump, and pain can be due to various etiologies, such as trauma, fluid collection, muscle tear, myopathy, venous thrombosis, neoplasm, or rhabdomyolysis.

Diagnosis of nontraumatic rhabdomyolysis depends on clinical history and biochemical tests, such as serum CK and urine myoglobin.[1,8] Creatine kinase is present in large quantities in the myocytes and is 100% sensitive as a marker for rhabdomyolysis.[1,8] Creatine kinase may increase acutely > 1,000 U/L, suggesting muscle lysis and necrosis as etiology for pain as opposed to other causes such as hematomas, abscesses, or venous thrombi.[1,9] Serum CK decreases rapidly at a rate of 39% per day, and it may normalize by the time a patient presents for medical care.[1,10-12]

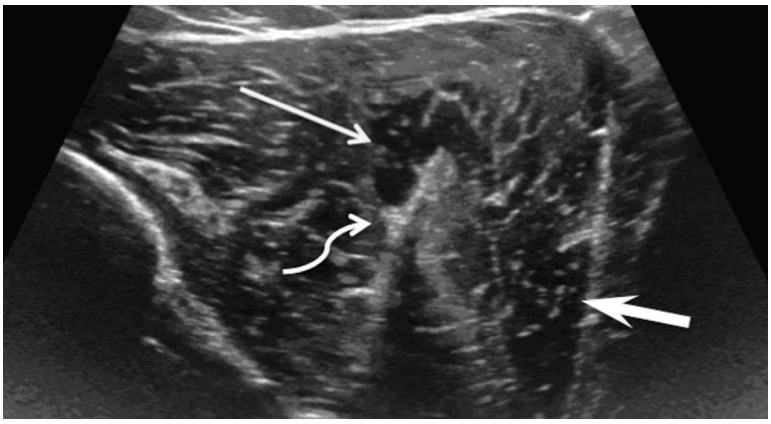

Figure 2A. Transverse ultrasound view of the posterior left shoulder demonstrates a hypoechoic area (arrow) and triangular area with peripheral hyperechogenicity and decreased intramuscular echogenicity (curved arrow). Local loss of muscle texture with preservation of muscle boundary (thick arrow) is noted. No evidence of a fluid collection visualized.

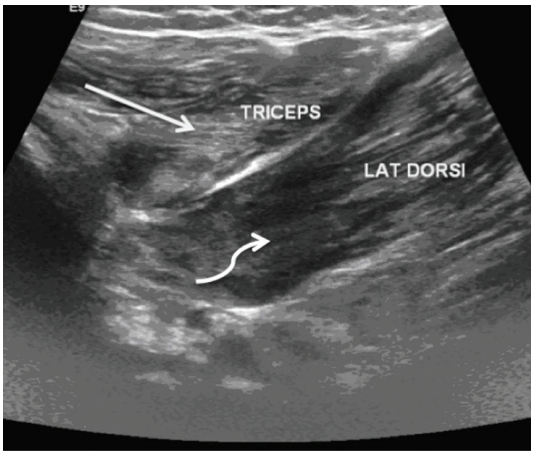

Figure 2B. Longitudinal ultrasound of left posterior shoulder. Hypoechoic and hyperechoic areas in long head of triceps muscle (arrow). Hypoechoic area and thickening of the lattisimus dorsi (curved arrow) is noted.

Imaging plays a significant complimentary role. During the patient's second ED presentation, the CK was normal at 54 U/L, whereas ultrasound and MRI findings were suggestive of focal muscle abnormalities.

Although there are diverse etiologies of rhabdomyolysis, the ultimate consequences of rhabdomyolysis are muscle cell membrane injury, metabolism malfunction, and destruction of the myofibril, resulting in inflammatory changes, such as muscle edema, hemorrhage, and myonecrosis and disruption of muscle fibers.[1,2,8,9,13] This may cause an alteration in muscle size, shape, and echogenicity on sonography and abnormal signal intensity on MRI.[13] The sensitivity of MRI in the detection of muscle involvement is higher than that of CT or ultrasound due to the high soft tissue contrast.[4,13,14] Specificity of all 3 modalities is low and not reported.

Although the sensitivity of ultrasound is lower than that of MRI, use of ultrasound in neuromuscular evaluation has been increasing recently due to technical refinements. Ultrasound can be effectively used as a first-line screening modality, especially in an emergency.[5] Magnetic resonance imaging best assesses the distribution and extension of the affected muscles, especially when fasciotomy is considered for treatment, and initially reveals edema, inflammation, and findings of myonecrosis; muscle atrophy and fatty degeneration occur later.[4,13-15] Typical MRI findings include increased signal intensity on T2-weighted and STIR (short-tau inversion recovery) sequences and variable enhancement on T1 postcontrast images, as was seen in this case, which indicated edema, inflammation, and necrosis of the muscle tissue.

Shintani and colleagues described the reversibility of the MRI findings, showing that the high-intensity lesions seen on T2-weighted images resolved in parallel with the clinical course.[14,16] Lu and colleagues investigated 10 patients with rhabdomyolysis and found 2 distinct imaging types: Type 1 shows homogenous signal changes and enhancement in the affected muscles, and Type 2 shows rim enhancement on contrast-enhanced MRI, a "stipple sign" indicating areas of myonecrosis.[17] Magnetic resonance imaging signal alterations in the musculature can be nonspecific and overlap with those of inflammatory myopathies such as polymyositis, connective tissue diseases with inflammatory myositis, muscle infection, muscle infarction such as diabetic myonecrosis, muscle contusion, drug-induced myotoxicity, corticosteroids use, and use of cholesterol-lowering agents.[18,19]

Sonography is a useful screening modality for pain and swelling of the extremity, because it can detect a muscle tear, muscle sprain, and fluid collection, especially in emergent cases. There is scant literature about sonographic findings in rhabdomyolysis and compression nerve entrapment. The sonographic findings of rhabdomyolysis are local disorganization of the damaged muscle, decreased muscle echogenicity, and enlargement of the muscle, with preservation of the muscle boundaries.[5-7]

Intramuscular hyperechoic areas are seen due to hypercontractility of injured muscle. In this case, noted findings included patchy, irregular,

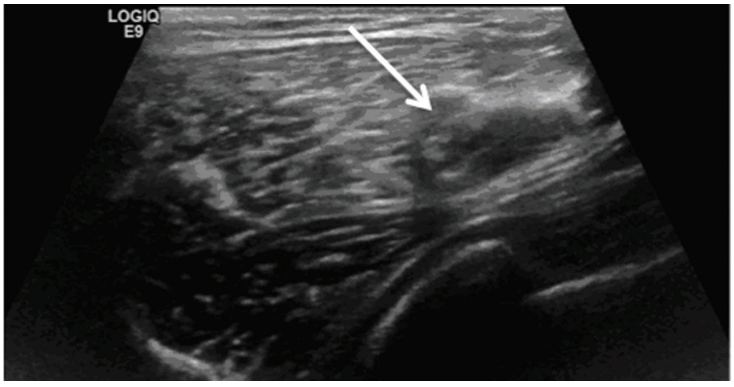

Figure 2C. Longitudinal ultrasound of the left posterior shoulder. A hypoechoic mass with loss of muscle texture in the triceps muscle (arrow).

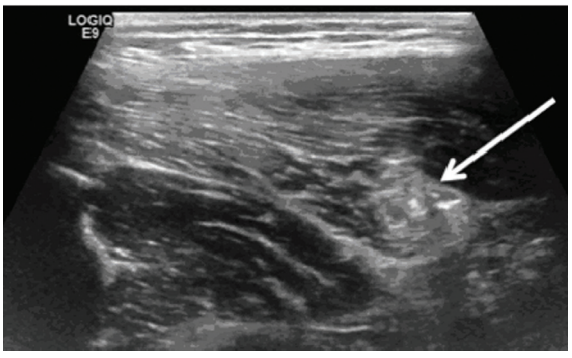

Figure 2D. Longitudinal ultrasound of the left posterior shoulder. A hyperechoic area and disorganized muscle is noted in the teres major (arrow).

hypoechoic areas, enlargement of the muscles and tendons, and irregular hyperechoic areas without focal defects. These findings differentiated an abnormality from a muscle tear or rupture, as these often show a focal muscle gap and focal defect, signifying the ruptured muscle retracting.

A study by Su and colleagues used the large number of crush injuries after an earthquake in China.[5] The characteristic sonographic findings were edema and thickened disrupted striated muscle, good overall muscle continuity, vague muscle texture, and enhanced cloudy or ground-glass-like echo. There was no blood flow signal in the hypoechoic areas.[6] Ultrasound was deemed a cost-effective, easily available modality by the authors.

CONCLUSION

Nontraumatic, focal rhabdomyolysis is rare and should be detected and differentiated from other causes of swelling, lump, pain, or other muscle disorders to prevent late complications. Sonography is an important screening diagnostic modality. MRI is used for assessment of the extent and distribution of injury. Awareness and familiarity with imaging findings can play a significant role, along with clinical and laboratory findings in the diagnosis and management of rhabdomyolysis. ●

Author disclosures

The authors report no actual or potential conflicts of interest with regard to this article.

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