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Magnetic Resonance Imaging of Musculoskeletal Emergencies

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Musculoskeletal trauma and infections are commonly encountered in the emergency department. Magnetic resonance imaging (MRI) is rarely employed in true emergencies and most musculoskeletal studies can be deferred to the outpatient setting. This article seeks to address the urgent conditions in which MRI can play a role in diagnosis, management, and treatment. This article outlines MRI's role in the evaluation of posterolateral corner injuries and other soft-tissue pathologies such as rhabdomyolysis, diabetic myonecrosis, septic arthritis, cellulitis, necrotizing fasciitis, and compartment syndrome.
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Introduction

True emergencies in musculoskeletal (MSK) imaging are usually secondary to trauma. Traumatic emergencies can be easily diagnosed using plain radiography or computed tomography (CT). Magnetic resonance imaging (MRI) is sparingly employed in the emergent setting because of limitations related to the image acquisition time, availability, and cost. Given the exquisite contrast resolution and high degree of anatomical detail provided by MRI, it can play a vital role in the assessment of urgent conditions such as soft-tissue and bone infections. MRI's usefulness in detecting small foci of infection, necrosis, and pathologic fluid collections is well established and in doing so, has proven itself superior to other cross-sectional imaging techniques such as CT and ultrasonography. This article addresses the urgent conditions in which MRI can play a vital role not only in diagnosis but also in management and treatment of MSK emergencies.

MR sequences useful for the diagnosis include T1-weighted fast spin-echo (FSE) and T2-weighted fat-suppressed (FS) FSE. Contrast-enhanced T1-weighted FSE sequences using gadolinium-based contrast media are typically performed in patients who do not have a contraindication to using such agents (ie, patient with significant renal disease). At our institution, contrast is administered only if the estimated glomerular flirtation rate is greater than 30 mL/min/1.73 m^2. In the presence of magnetic field inhomogeneities, T2-weighted fat-suppressed fast SE sequence can be replaced with inversion recovery sequences such as short tau inversion recovery (STIR) or turbo inversion recovery magnitude. Inversion recovery sequences are less susceptible to magnetic field inhomogeneity and are extremely sensitive in detecting edema and fluid collections. Non–fat-saturated FSE sequences are performed with increased bandwidth to reduce susceptibility artifact. Gradient-echo sequence may be employed to demonstrate the presence of gas, calcium, and metal, which are seen as hypointense foci of blooming. Gadolinium contrast is not essential for most trauma-related emergent studies. The identification of viable tissue from nonenhancing necrotic tissue is particularly useful in destructive infectious processes where patterns of enhancement and tissue characteristics on particular imaging sequences can differentiate abscess from soft-tissue edema and phlegmon.

At our institution, standard sequences acquired in the axial (AX) plane are used for the assessment of soft-tissue and bony pathology. Sagittal or coronal (COR) sequences are employed depending on the location of the pathology. Our standard sequences include the following: sagittal or COR T2 STIR, sagittal or COR T1, AX T2 FS, and AX T1. When gadolinium contrast is administered, precontrast AX T1 FS are acquired first, followed by T1 FS postcontrast administration images, each generated in at least 2 planes.

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Rhabdomyolysis

Approximately 26,000 cases of rhabdomyolysis are reported in the United States annually.[1] Etiologies for rhabdomyolysis include traumatic causes (crush injuries), nontraumatic exertional causes (over exercise, hyperthermia, and metabolic myopathies), and nontraumatic nonexertional causes (drugs or toxins, infections, electrolyte disorders, endocrine disorders, and inflammatory myopathies).

Rhabdomyolysis occurs secondary to striated muscle damage with subsequent release of intracellular toxins into the blood stream. The initial insult causes disruption of the sarcolemma and release of calcium. The increase in intracellular calcium results in activation of destructive enzymes, including proteases, which ultimately cause skeletal muscle death and the release of toxic intracellular contents such as myoglobin, creatine kinase, electrolytes, and other enzymes into the blood stream. Damaging oxidative free radicals are also produced leading to cellular damage and leakage of contents into the blood stream. Elevated levels of intracellular toxins within the blood stream lead to systemic complications such as cardiac arrhythmia (due to electrolyte abnormalities), renal failure (due to myoglobinuria), and disseminated intravascular coagulation (late complication). Of note, the acute kidney injury is caused by the combination of intracellular myoglobin release and renal vasoconstriction due to a loss of extracellular fluid.

Rhabdomyolysis generally presents with muscle pain, weakness, and dark urine (myoglobinuria). Additional symptoms including nausea, vomiting, and fever may also be present. Mild forms may present with myoglobinuria and abnormal laboratory findings. Accompanying swelling of the affected musculature may compromise vascular flow in certain cases causing increased intracompartmental pressure and eventual compartment syndrome. The lower extremity, specifically the thigh or calf muscles, are most commonly involved. More than half of the cases of rhabdomyolysis present with bilateral involvement.

Diagnosis is predominantly based on abnormal laboratory findings such as myoglobinemia and myoglobinuria as well as elevated blood levels of creatine kinase, potassium, and uric acid. The most sensitive indicator of myocyte injury is an elevated serum creatine kinase level,[2] which peaks in approximately 2 days and subsequently declines.

Imaging studies are generally used in an attempt to determine the underlying etiology of rhabdomyolysis. MRI can be useful to assess for underlying myopathies or to evaluate for the presence and extent of myonecrosis. Type I as well as type II rhabdomyolysis demonstrate increased signal intensity on T1, T2, and STIR images. Although the former demonstrates a homogeneous pattern of hyperintensity, the more devastating type II rhabdomyolysis causes widespread muscle damage leading to a heterogeneous pattern of hyperintensity on the image sequences (Figs. 1 and 2). Multiple studies have postulated that the cause of increased signal intensity on T1-weighted images is likely due to methemoglobin, proteinaceous material, and fat.[3,4] T2-weighted and STIR image sequences demonstrate high signal intensity secondary to the presence of increased interstitial water content secondary to edema, necrosis, or active muscle infarction.[4-9] Gadolinium administration is ideal to identify and distinguish necrotic musculature, but extreme caution must be exercised when administering contrast to this patient population, as they usually present with severe renal impairment. A uniform pattern of enhancement without evidence of necrosis is detected on postgadolinium administration image sequences in type 1 rhabdomyolysis. This mimics the imaging findings of many other causes of muscle edema such as myositis and myopathies.[10]

A study was conducted by Lu et al[11] in 2007 on a small cohort of patients presenting with type II rhabdomyolysis. They found that postcontrast imaging in all the patients demonstrated a characteristic rim enhancement. The study also identified and

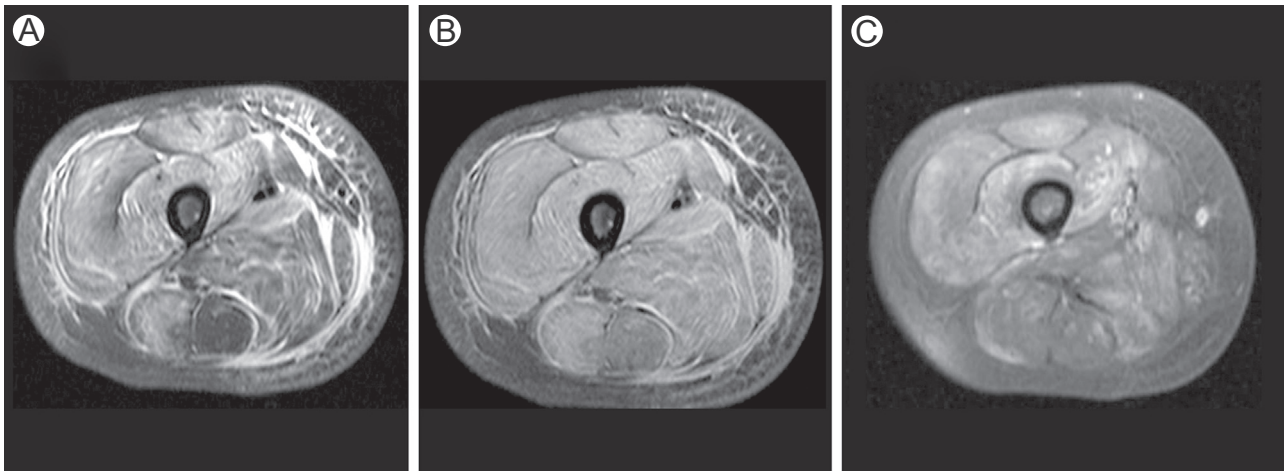

Figure 1 Type II rhabdomyolysis. A 5-year-old boy presenting with elevated creatine kinase and progressive lower extremity weakness status post motor vehicle accident. Axial T2 FS (A) and axial proton density (B) demonstrate diffusely increased signal in muscles of the upper thigh representing edema and fascial thickening. Postcontrast axial T1 FS image (C) demonstrates patchy enhancement in the abductor and quadriceps musculature. Focal and linear areas of nonenhancement identified in the adductor and quadriceps muscle represent necrotic tissue.