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# Imaging Findings in the Setting of Rhabdomyolysis

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**R**habdomyolysis is a syndrome characterized by the disruption of skeletal muscle leading to the release of intracellular muscle constituents, including myoglobin and creatine kinase, into the circulation and extracellular spaces. The clinical presentation is broad, ranging from asymptomatic to life-threatening.

The most significant complication is acute kidney injury, which occurs in up to 40% of patients. Other serious complications include disseminated intravascular coagulation and compartment syndrome.<sup>1</sup> Risk factors that predict a poor outcome (eg, a need for renal replacement therapy, or death) include advanced age, female sex, initial creatine, creatinine phosphokinase, phosphate, calcium, and bicarbonate levels.<sup>2</sup> Characteristic symptoms of

rhabdomyolysis include muscle pain, weakness, and dark urine, although over half of patients do not report muscle symptoms.<sup>3</sup> Physical findings include muscle tenderness and weakness. Muscle swelling typically occurs later in the hospital course during fluid repletion.

Creatine kinase, the most sensitive lab abnormality for muscle injury, is released into the bloodstream within 12 hours of injury. Although no specific threshold has been established for the diagnosis of rhabdomyolysis, a creatine kinase level of at least five times the upper limit of normal (ie, 1000 IU/L) is commonly used. A creatine kinase level of >5000 IU/L generally indicates a degree of muscle injury sufficient to cause acute kidney damage. Myoglobinuria is responsible for the brown to red urine of patients with this condition.

Additionally, the accumulation of myoglobin, a nephrotoxin, is responsible for the acute kidney injury of rhabdomyolysis. However, myoglobin has a short half-life and therefore may not be detected in the serum or urine of patients experiencing significant rhabdomyolysis.<sup>4,5</sup>

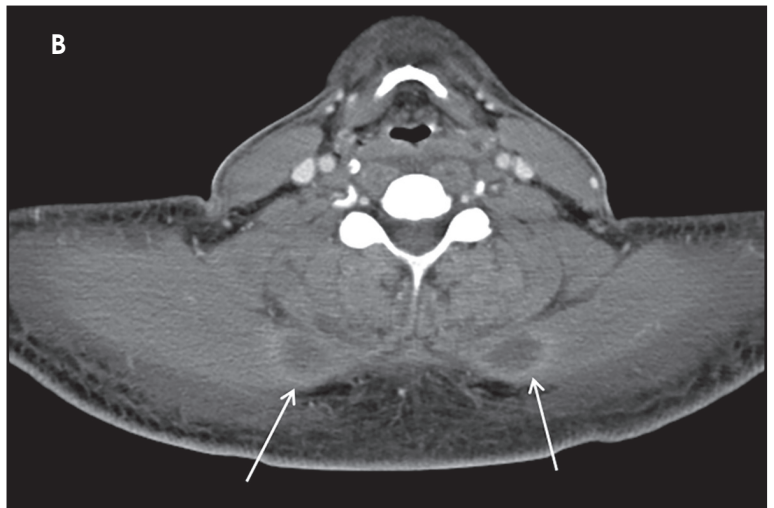
## Imaging Considerations

Rhabdomyolysis is not diagnosed specifically with imaging, but rather through a final pathway shared by numerous etiologies resulting in muscle damage. Imaging findings depend on the underlying cause, although typically the muscles will display signs of edema and/or hemorrhage.

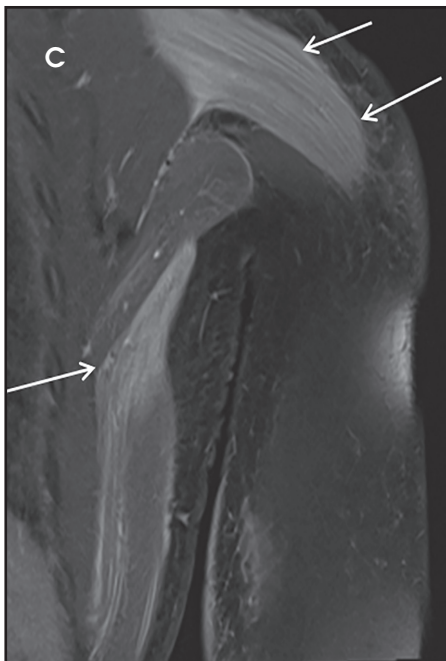
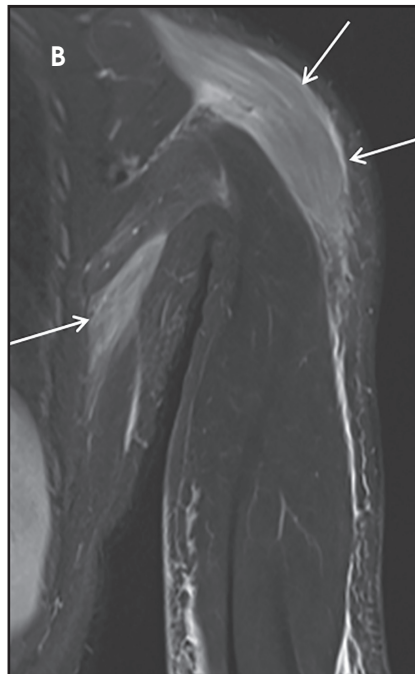
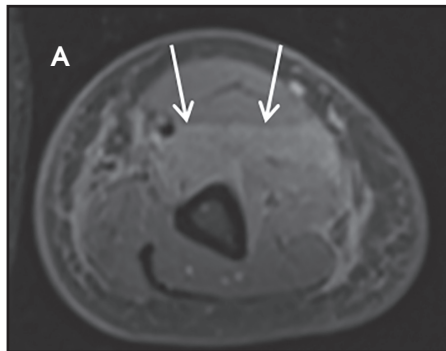
On computed tomography (CT), edematous muscles are typically hypodense and may be enlarged (Figure 1A). As muscle damage progresses, peripheral enhancement surrounding areas of infarcted or necrotic tissue may be demonstrated on postcontrast images (Figure 1B).

Magnetic resonance imaging (MRI) is more sensitive than CT to muscle damage.<sup>6</sup> Involved muscle is usually isointense on T1 sequences, although mild T1 hyperintensity owing to hemorrhage or protein concentration may be demonstrated, particularly on fat-saturated sequences (Figure 2). The affected musculature typically displays high signal on T2 or short tau inversion recovery (STIR) images (Figure 2).<sup>7</sup> The broad differential diagnosis of muscle edema can be narrowed by separating

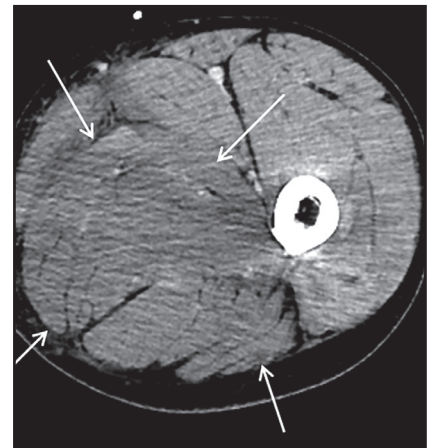
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**FIGURE 1.** Rhabdomyolysis (CK = 26,538IU/L) from prolonged neck extension after IV drug intoxication. (A) Axial CECT of the neck demonstrates hypodense, edematous splenius capitus and semispinalis capitus muscles (arrows). (B) Axial CECT of lower neck demonstrates myonecrosis with peripheral enhancement within the trapezius muscles (arrows).



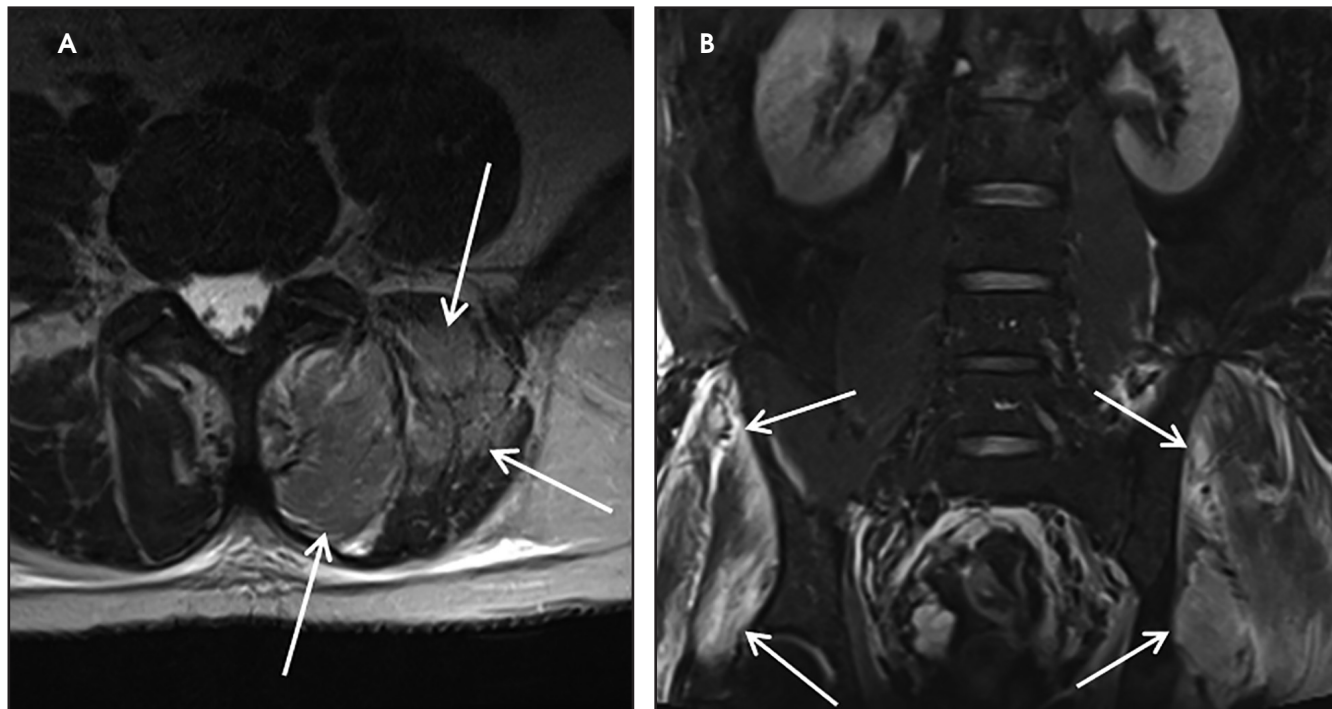
**FIGURE 2.** Exertional rhabdomyolysis (CK=26,365IU/L). Patient experienced upper-extremity pain and dark urine after an intense workout. (A) T1 fat-saturated axial MRI of upper arm demonstrates high signal in the brachialis muscle (arrow). (B) Coronal STIR image of left shoulder displays high signal throughout the deltoid and latissimus dorsi muscles (arrows). (C) Coronal fat-saturated T1 postcontrast image displays homogeneous enhancement of the left deltoid and latissimus dorsi muscles (arrows).



**FIGURE 3.** Rhabdomyolysis (CK= 310,541 IU/L) and compartment syndrome due to prolonged immobilization while intoxicated. CECT of the left thigh demonstrates decreased enhancement of multiple muscles, including the adductor longus, adductor magnus, biceps femoris, and semitendinosus (arrows). The patient underwent emergency fasciotomy and debridement.

cases into symmetric or asymmetric patterns.

Symmetric causes of muscle edema are typically secondary to inflammatory (polymyositis, dermatomyositis, human immunodeficiency viral associated (HIV), myositis) and drug-related myopathies. Asymmetric edema is more likely to be caused by trauma,



**FIGURE 4.** Patient found unconscious after fentanyl overdose (CK 155,355). (A) Axial T2 MRI of the lumbar spine demonstrates high signal edema in the left paraspinal musculature (arrows). (B) Coronal STIR MRI of the pelvis displays high signal throughout the gluteal muscles (arrows).

pyomyositis, radiation, myonecrosis, denervation, and compartment syndrome.<sup>8</sup> Following gadolinium-based contrast administration, involved muscles may demonstrate homogeneous enhancement prior to liquefaction or heterogeneous enhancement (stippled sign), reflecting viable tissue intermixed with necrotic muscle (Figure 2).<sup>8</sup> Scintigraphy is not a primary imaging modality in the setting of rhabdomyolysis, although muscle injury may be demonstrated on bone scans.

Acute compartment syndrome resulting from elevated compartmental pressure is a complication of rhabdomyolysis. The diagnosis is clinical and supported by an abnormal intracompartmental pressure measurement. Imaging plays a limited role in diagnosing acute compartment syndrome, but it may demonstrate normal or increased T1 signal within muscle, owing to hemorrhage and increased T2 signal secondary to edema. Decreased enhancement on CT or MRI within the compartment can be demonstrated on postcontrast images (Figure 3).<sup>8</sup>

### Causes of Rhabdomyolysis

Any condition that leads to extensive muscle damage may theoretically result in rhabdomyolysis. A study of 425 adult patients with rhabdomyolysis found drug or alcohol abuse, medicinal drug use, and immobility to be leading causes.<sup>9</sup> The multitude of etiologies can be organized into three main categories:

- Trauma or long-standing compression of muscle (prolonged immobilization, crush injury, electric shock, muscle tear);
- Nontraumatic (strenuous exercise, seizures, delirium tremens, muscular dystrophy, hyperthermia, heat stroke, diabetic ketoacidosis, myopathy); and,
- Muscle toxins (illicit drugs; prescription drugs, including statins; influenza, HIV, and herpes virus; bacterial infection; snake venom).<sup>1,5</sup>

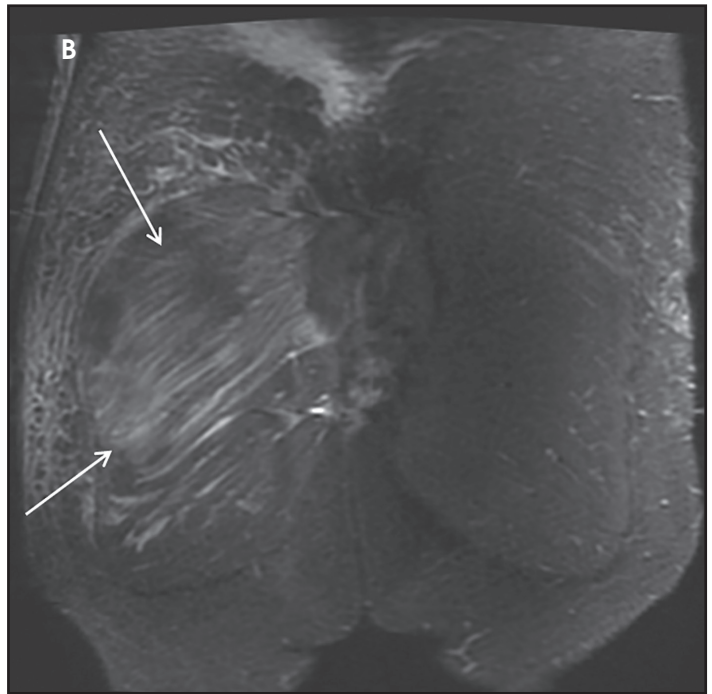
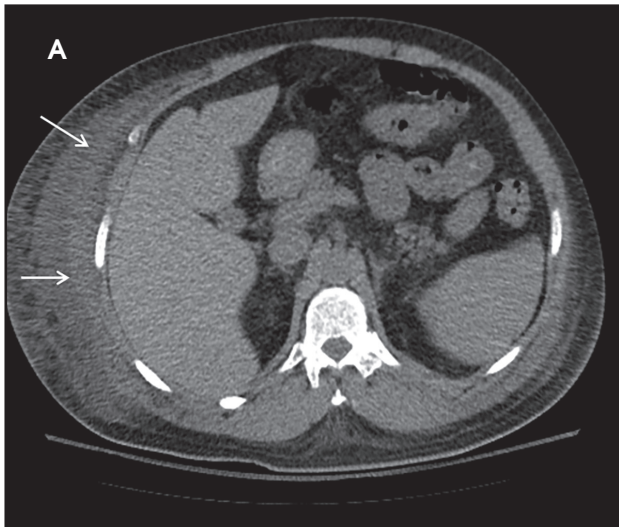
### Muscle Compression or Trauma

Muscle compression during prolonged unconsciousness, rehabilitation, or surgery may lead to muscle oxygen

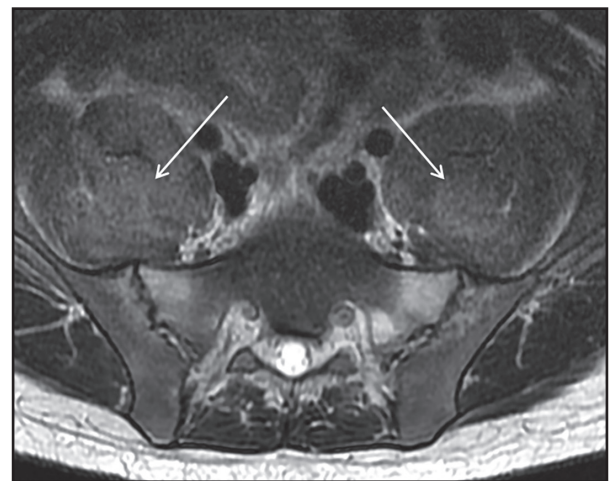
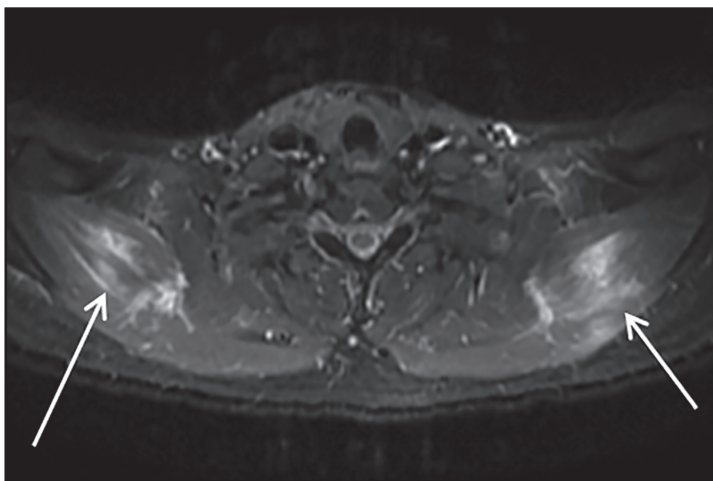
deprivation, eventually resulting in muscle necrosis and rhabdomyolysis. Muscle compression resulting in ischemia can develop after drug or alcohol intoxication in which the patient has been lying or sitting in one position for a prolonged period of time (Figures 4,5). Crush injuries and blunt trauma are also common etiologies of rhabdomyolysis, particularly in the elderly. Amongst 167 patients with rhabdomyolysis, all of whom were over 65 years, 56.9% of cases were caused by falls.<sup>10</sup> In the setting of a crush syndrome, such as from a building collapse or automobile crash, rhabdomyolysis occurs once the muscle compression is relieved. Injuries related to high-voltage electricity (electrocution, lightning strikes) cause rhabdomyolysis in up to 10% of survivors.<sup>1</sup>

### Nontraumatic Muscle Damage

Rhabdomyolysis may occur when the metabolic demands of normal muscle exceed the energy supply. Although serum creatine kinase naturally rises after strenuous exercise, rhabdomyolysis typically occurs when exertion generates



**FIGURE 5.** Patient found unconscious on right side after heroin overdose (CK=140,406). (A) Axial CECT of the upper abdomen demonstrates swelling and edema of the right chest/abdominal wall musculature and adjacent soft tissues (arrows). (B) A STIR coronal MRI of the pelvis demonstrates edema throughout the right gluteal musculature (arrows).



**FIGURE 6.** Patient presented with arm weakness and paresthesias after prolonged, overhead, military-style training (CK=13,645). Axial fat-saturated T2 MRI of the shoulders demonstrates high-signal edema within the deltoid muscles (arrows).

**FIGURE 7.** Patient with back pain and rhabdomyolysis following a prolonged seizure (CK=23,002). Axial T2 image of the pelvis demonstrates high-signal edema with the psoas muscles (arrows).

levels of pain and fatigue beyond that which would normally compel an individual to stop exercising. Factors such as competitive athletic or military training, extreme sports, and peer pressure during group exercise may predispose individuals to this level of exertion (Figures 2,6). Compounding risk factors include insufficient physical training and exertion in hot and humid conditions. Additionally, pathologic muscle exertion during

delirium tremens, psychotic agitation, and seizures (Figure 7) may result in rhabdomyolysis.<sup>3</sup> Other causes of nontraumatic muscle damage include malignant hyperthermia, neuroleptic malignant syndrome, and heat stroke.<sup>1,3</sup>

#### Muscle Toxins

Prescription medications and abused substances may produce a myopathy leading to rhabdomyolysis.

Statin medications are a well-known cause of muscle toxicity and rhabdomyolysis (Figure 8), especially in patients with predisposing conditions such as hypothyroidism, those on high doses of these medications, advanced age, and female sex.<sup>1,12,13</sup> Other classes of drugs that can cause rhabdomyolysis include psychiatric agents, non-statin lipid-lowering agents, and antihistamines.



**FIGURE 8.** Patient with neck and extremity pain, amber-colored urine, and rhabdomyolysis. Known risk factors included a statin (atorvastatin calcium) as well as alcohol use. Sagittal T2 image of the right side of the neck demonstrate edema in the longus colli musculature (arrows).

Substance abuse can result in metabolic acidosis and induce rhabdomyolysis. Alcohol, cocaine, heroin, and amphetamines have all been implicated. Myopathy secondary to systemic drug use typically affects the buttocks, quadriceps, adductors, and calf muscles in a symmetric distribution.<sup>8</sup> Toxins, including poisons and venoms, may directly interfere with muscle energy production, cause electrolyte abnormalities, or degrade the muscle cell.<sup>14,15</sup>

A vast number of bacterial and viral infections (eg, influenza, HIV, herpes simplex) can cause muscle damage. Infections that invade the muscle cells may lead to muscle necrosis. Some infections may generate toxins that inhibit metabolic activity, inducing rhabdomyolysis.<sup>1</sup> MRI of pyomyositis-induced rhabdomyolysis demonstrates swelling of involved muscles on T1 images. T2 images display increased muscle signal owing to pus and edema (Figure 9). Following contrast administration, small fluid collections representing myonecrosis or larger, non-enhancing abscesses may be demonstrated.<sup>8</sup>



**FIGURE 9.** Pyomyositis and rhabdomyolysis (CK = 5369) in a patient with sepsis and left hip pain following intramuscular heroin administration. (A) T2 axial MRI of the lower pelvis and (B) STIR coronal MRI of posterior pelvis demonstrate high signal edema in the left gluteus muscles and intermuscular fat planes (arrows).

## Rhabdomyolysis Treatment

The specific cause of muscle damage, such as drugs, toxins, or infection, should be identified and, where possible, eliminated.<sup>15</sup> Intravenous fluid replacement to achieve a urine output of 300 mL/h is important to prevent acute kidney injury. Similarly, bicarbonate administration can prevent acute kidney injury by increasing urine pH, which helps mitigate myoglobin toxicity.<sup>15,16</sup> Closely monitoring patients for fluid and electrolyte abnormalities is essential for preventing metabolic disturbances. Patients with severe kidney damage may require dialysis;<sup>17</sup> those with suspected acute compartment syndrome require urgent surgical consultation.

## Conclusion

Rhabdomyolysis is a complex syndrome characterized by significant muscle damage and subsequent release of muscle components into the bloodstream and extracellular spaces. The most significant complication is acute

kidney injury. The numerous causes can be categorized into three main groups: Muscle compression or trauma, non-traumatic/exertional muscle damage, and toxins. Rhabdomyolysis is a clinical diagnosis and imaging reflects the underlying cause and extent of muscle damage. Involved muscles typically display enlargement, edema, and hemorrhage.

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