Magnetic Resonance Imaging of Muscle

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Learning Objectives

At the end of this presentation, readers should be able to:

• Identify the normal imaging features of skeletal muscle on magnetic resonance imaging (MRI).
• Recognize that some muscle abnormalities do not produce signal alterations.
• Recognize the common patterns of muscle inflammation.
• Know the most common classification system used for muscle injury.
• Understand the evolution of hemorrhage in muscle tissues.

Normal Muscle

The MRI appearance of normal skeletal muscle is the result of the latter's admixture of muscle fibers and fat. Normal skeletal muscle shows a "striated" and "feathery" appearance, produced by the presence of high-signal fat interlaced within and between the major muscle bundles. Normal muscle is of low signal intensity on all sequences and of decreases in signal on T2-weighted images. The exterior surfaces of the muscle are smooth and typically show a mild convexity. An important anatomic region of the muscle is its myotendinous junction, where muscle fibers interdigitate with the tendon. The myotendinous junction is located at a variable distance from the site of tendon insertion.

Four common patterns of abnormal muscle are visualized with MRI: (1) muscle with abnormal morphology with normal signal; (2) mass within the muscle; (3) muscle atrophy; (4) muscle edema

Abnormal Morphology with Normal Signal

This pattern is most commonly caused by an accessory muscle, a congenital abnormality in which an anomalous extra muscle is present. The accessory muscle may be asymptomatic or present either as a palpable mass due to its effect on adjacent structures, such as nerve compression that results in denervation. Most accessory muscles are identified around the hand and foot (Fig. 1). The best-known accessory muscle is the accessory soleus, seen in the pre-Achilles fat pad. This muscle can be very large and is usually felt by the patient. Other common accessory muscles include the peroneus quartus muscle, located behind the fibula; the accessory anconeus epitrochlearis; the accessory abductor digiti minimi, in the wrist; and the anomalous lumbrical muscle, in the carpal tunnel.

Fig. 1 a, b. Accessory muscle. This middle-aged man palpated a mass on the dorsum of his wrist. Axial T1-weighted magnetic resonance imaging (MRI) of the wrist (a) shows an accessory extensor digitorum manus muscle. The mass remains isointense to normal muscle on the axial T2-weighted image (b)
**Intramuscular Mass**

In this case there is a focal mass within the muscle, due to primary or secondary neoplasms or as part of a large group of non-neoplastic disorders. Metastatic disease within the muscle is relatively rare and is seen most frequently with advanced disease. The most common location for intramuscular metastasis is the paraspinal and retroperitoneal musculature (Fig. 2). Several different primary neoplasms can arise in the muscle. Common benign intramuscular neoplasms include lipoma and hemangioma. Primary intramuscular malignancies can arise from a host of tissues and include liposarcoma, malignant fibrous histiocytoma, and rhabdomyosarcoma. Non-neoplastic masses within the muscle include hematoma, abscess, myositis ossificans (see below), and inflammatory pseudotumors of muscle.

**Muscle Atrophy**

Muscle atrophy is the result or end stage of many muscle abnormalities, and thus is only a finding, not a diagnosis. It reflects numerous disorders that result in a loss of muscle volume and/or increased fatty infiltration of the muscle substance. Spectroscopy is more sensitive than conventional MRI for the detection of early muscle atrophy. Chronic disuse, denervation, and myopathies are the most common causes seen in clinical practice. Atrophy is generally considered irreversible.

**Congenital Myopathies**

Congenital myopathies result in symmetrical muscle atrophy involving multiple muscles, with a predilection in most forms for axial and truncal involvement. There are numerous forms of muscular dystrophy, with Duchenne and Becker muscular dystrophy being the most common. These typically present in childhood or adolescence as progressive proximal muscle weakness. Numerous metabolic myopathies have also been described, typically related to mitochondrial dysfunction or defects in energy metabolism. In the acute phase of muscle damage, symmetrical mild hyperintensity of the muscles can be seen. Unlike inflammatory myopathies, the subcutaneous tissues remain normal. More advanced disease typically shows pseudohypertrophy of lower-extremity muscles, particularly the calf musculature, due to excessive fatty infiltration. Ultimately, severe fatty atrophy of the muscle develops (Fig. 3).

**Denervation**

Muscle denervation also results in muscle atrophy, but the distribution of involvement is limited to muscles innervated by a single nerve. Acutely denervated muscle shows a paucity of findings on MRI, with signal alterations usually seen several weeks following loss of neural innervation. In subacute denervation, the denervated muscle shows high signal on T2-weighted and inversion-recovery sequences. Typically, the size of the muscle remains normal or is slightly diminished due to concomitant atrophy (Fig. 4). In chronic denervation, the muscle edema resolves, and the involved muscles undergo volume loss and fatty atrophy. The presence of fatty change implies an irreversible lesion. Clinical history and the distribution of the muscle abnormalities, which correspond to a specific nerve distribution, allow accurate diagnosis of muscle denervation.

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Fig. 2. Metastatic disease. The axial T2-weighted image of the lumbar spine in a patient with renal cell carcinoma shows an inhomogeneous high-signal mass in the left paraspinal musculature. Biopsy revealed that the mass represented metastatic disease.

Fig. 3. Muscular dystrophy. A coronal T1-weighted image of the pelvis shows profound diffuse muscle atrophy with fatty infiltration of the muscle due to long-standing Duchenne muscular dystrophy.