Muscle Imaging

17

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Learning Objectives

- Review the role of imaging in muscle diseases.
- Detect and classify acute muscle injury by imaging.
- Review unique imaging features in idiopathic inflammatory myopathy.
- Identify the imaging features of other common infectious, traumatic, and vascular muscle pathologies.
- Discuss the differential considerations in muscle lesions.

17.1 Introduction to Muscle Imaging

Evaluation and characterization of skeletal muscle pathology is a frequently encountered indication for musculoskeletal imaging. Causes of muscle pathology are diverse and include traumatic, autoimmune, infectious, inflammatory, neurologic, and neoplastic. Each etiology while dramatically different in the pathophysiology may present with similar imaging features. An understanding of the subtle differences in imaging features between the pathologic conditions may serve to guide diagnosis and treatment in these often complex cases. In this section, we will discuss the various skeletal muscle pathologies and the imaging features associated with each.

17.2 Imaging Modalities in Skeletal Muscle Evaluation

Radiographs, while excellent for bone pathology, has limited utility in the evaluation of muscle. Although the majority of muscle pathology is occult on routine radiographic images, X-ray may be useful in a few conditions. Certain inflammatory or autoimmune myopathy, for example, is characterizedby unique soft tissue calcifications of which radiographs may be the most reliable modality for detection. Magnetic resonance imaging and ultrasound offer excellent special resolution, allowing for the detailed evaluation of muscle microanatomy. Ultrasound offers an added benefit of dynamic imaging but is less sensitive than MRI for muscle edema and low-grade injury. Because of the superior sensitivity in detecting subtle injury, MR imaging evaluation is largely considered the diagnostic gold standard.

17.3 Traumatic Muscle Injuries

Muscle injury is common among athletes and poses a serious limitation to continued performance. The location and extent of a muscle injury has implications on the recovery and functional outcome.

17.3.1 Muscle Strain/Tear

Key Point

- Acute muscle strains/tears most frequently involve the gastrocnemius, biceps femoris, and rectus femoris muscles of the lower extremity.

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Acute muscle strains/tears are commonly encountered muscle injuries in the athlete, most frequently involving the gastrocnemius, biceps femoris, and rectus femoris muscles of the lower extremity [1, 2]. Muscle strains are characterized into three grades with progressing severity and complexity of the injury and increasing time to recovery (Table 17.1).

Table 17.1 Grades of muscle strain [3–6]

Injury	MR findings	Extent of injury	Example
Grade 1	• T2 high signal edema with feathered appearance, usually at the myotendinous junction. No architectural distortion • Thickened or edematous tendon without tear or laxity	<10% fiber disruption	Figure 17.1
Grade 2	• T2 high signal edema with focal/partial fiber disruption • ± intramuscular hematoma	10–50% fiber disruption	Figure 17.2
Grade 3	• Complete muscle fiber/tendon disruption with laxity with/without retraction • ± intramuscular hematoma	50–100% fiber disruption	Figure 17.3

17.3.2 Delayed Onset Muscle Soreness

Subacute or delayed muscle injury can be seen in the setting of overuse and is referred to in radiology literature as delayed onset muscle soreness (DOMS). DOMS is thought to occur as a result of muscle fiber microtrauma followed by a delayed inflammatory phase. This injury, while painful, results in no permanent muscle damage or functional deficit. Ultrasound interrogation often yields diffuse hyperechoic foci in an enlarged muscle belly but may be insensitive in mild injury [7]. Fluid-sensitive MR imaging is the gold standard diagnostic exam, demonstrating diffuse muscle edema involving all utilized muscles of a compartment [8] (Fig. 17.4).

17.3.3 Muscle Herniation

Muscle hernias, or myofascial defects, represent focal protrusion of the muscle fibers through a focal defect in the overlying superficial muscle fascia. Muscle hernias are most commonly seen in the lower extremity with the anterior compartment of the lower leg the most common location. Ultrasound is the

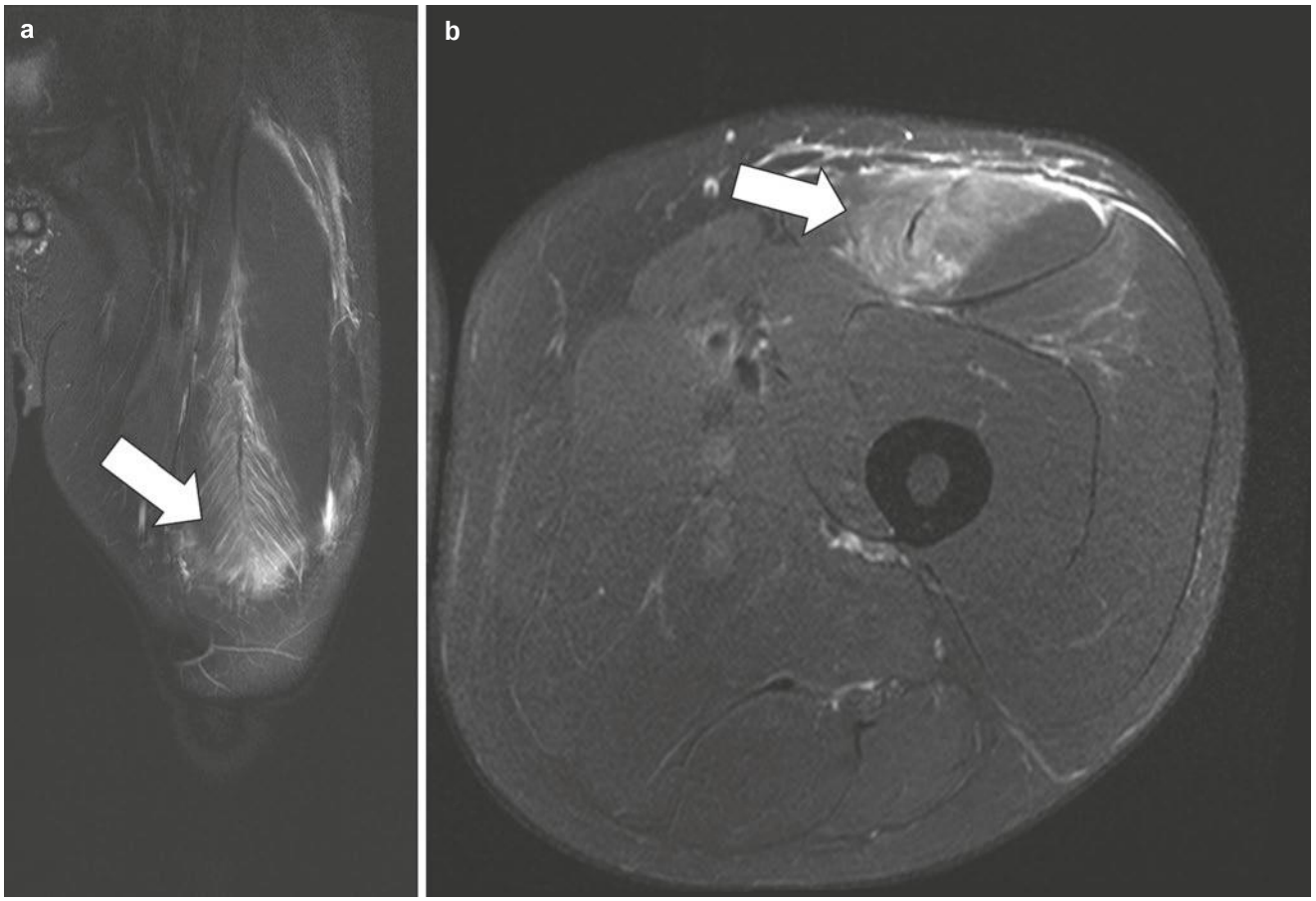

Fig. 17.1 Coronal (**a**) and axial (**b**) fat-saturated STIR MR images of the left thigh demonstrate feathered edema along the myotendinous junction of the rectus femoris muscle. No fiber disruption is identified in keeping with a grade 1 muscle injury

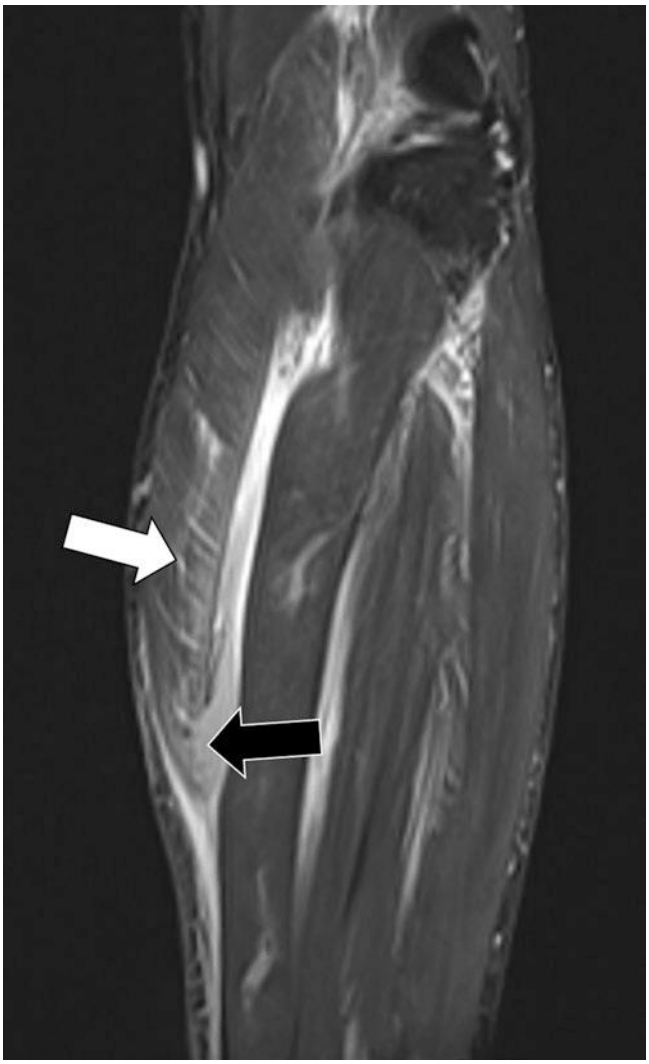

Fig. 17.2 Coronal fat-saturated STIR MR image of the lower leg demonstrates extensive intramuscular and fascial edema at the distal gastrocnemius muscle (white arrow). Partial-thickness fiber disruption at the distal myotendinous junction (black arrow) is in keeping with grade 2 muscle injury

modality of choice for the evaluation of muscle hernia as it may provide dynamic visualization of a reducible protrusion. Herniated muscle on US appears hypoechoic and mushroom-shaped with a smaller neck at the location of the fascia [9] (Fig. 17.5). MRI may be useful in the quantification of the fascial defect and herniated muscle bulk [10].

17.4 Inflammatory Myopathies

Inflammatory myopathy is classified as idiopathic or secondary. Secondary inflammatory myopathy is most frequently associated with autoimmune/connective tissue disorders such as Sjogren's disease or systemic lupus but can also be seen as a consequence of endocrine disorders or paraneoplastic syndromes. Inflammatory myopathy is a subset of heterogeneous muscle diseases that share similar pathophysiology, imaging features, and clinical presentation. Progressive and symmetric proximal to distal muscle pain and weakness in the setting of elevated muscle enzymes and abnormal EMG is suggestive of idiopathic myositis. MRI is the gold standard modality for the identification and characterization of these disorders.

17.4.1 Polymyositis/Dermatomyositis

Polymositis and dermatomyositis are related idiopathic inflammatory diseases of muscle that are characterized by muscle pain, weakness, and edema in a proximal to distal progressive distribution often involving the anterior compartment musculature of the thigh [11]. On MRI, both entities are defined by muscle edema involving one or more muscles in a symmetric distribution [12]. These entities while similar differ slightly in clinical and imaging features. Dermatomyositis, for example, is often seenin conjunction with esophageal dysfunction and maypresent with classic "sheet-like" cutaneous/dermal calcifications [13] (Fig. 17.6). The microanatomy of muscle involvement between these two entities also differs, with the endomysium (lining between small muscle fibers) preferentially involved in polymyositis and the perimysium and epimysium (around the larger muscle fasciclesand muscle belly) preferentially involved in dermatomyositis. While both entities are characterized by feathered muscle edema in the proximal muscle groups, the differences in preferential involvement may help to differentiate these two on MR imaging. Muscle edema in polymyositis is seen centrally within the muscle belly, often sparing the peripheral muscle fascia and myotendi- nous junction (Fig. 17.7). Dermatomyositis by contrast presents as muscle edema predominantly peripheral with notable myotendinous and myofascial involvement [14, 15] (Fig. 17.8).

Key Point

- Muscle edema in polymyositis is seen centrally within the muscle belly, often sparing the peripheral muscle fascia and myotendinous junction.

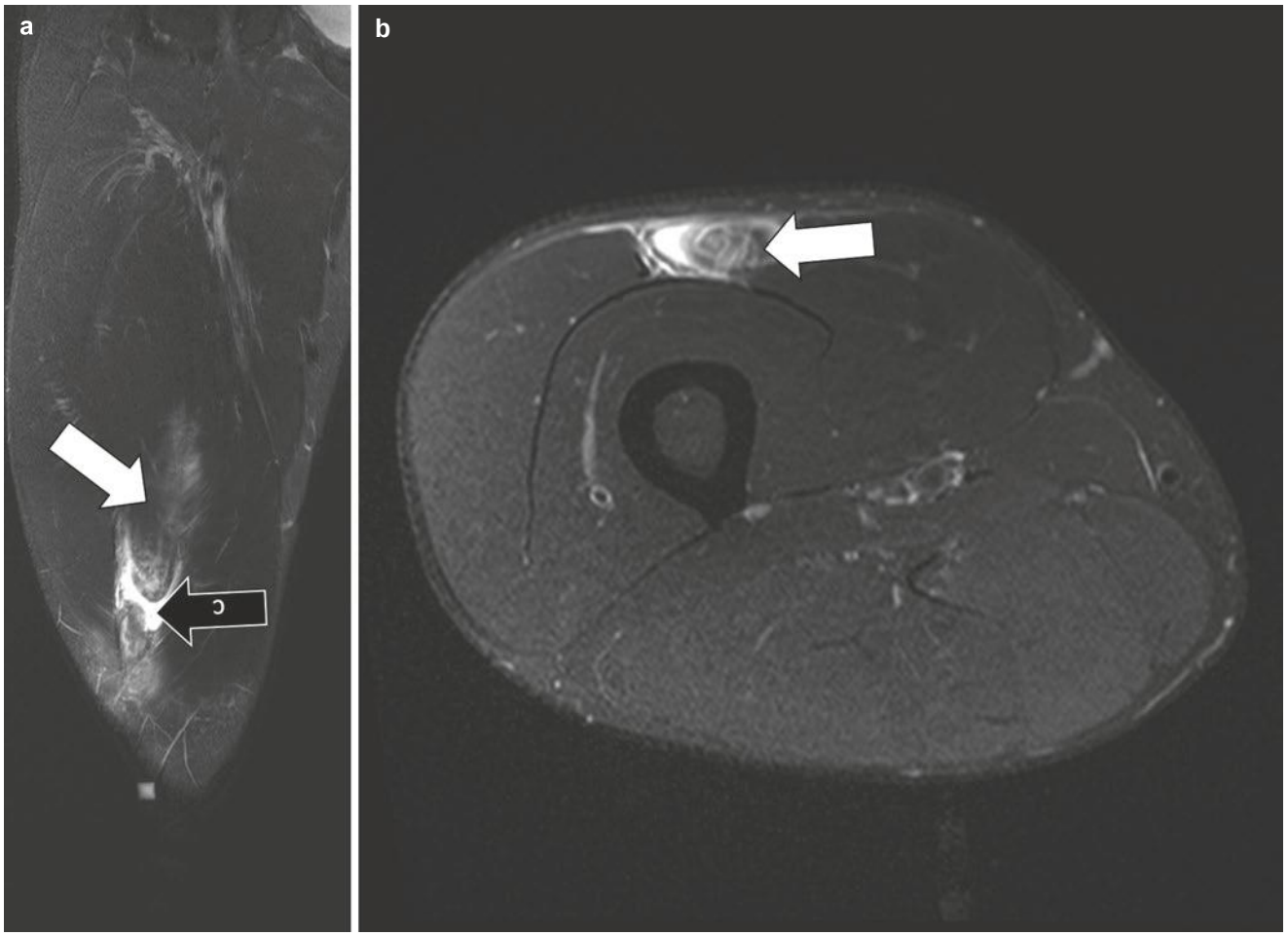

Fig. 17.3 Coronal (**a**) and axial (**b**) fat-saturated STIR MR images of the right thigh demonstrate extensive edema in the distal rectus femoris muscle belly (white arrows). This is a grade 3 injury given the complete fiber disruption and retraction apparent on the coronal image (black arrow)

17.4.2 Inclusion Body Myositis

Inclusion body myositis (IBM) is the most common idiopathic myopathy and is distinctly different in its pathophysiology compared to other idiopathic conditions. IBM is defined by inclusions of amyloid-B-protein within the skeletal muscle [16]. The onset of IBM is more common in the elderly and is often a distal to proximal distribution. MR imaging can easily differentiate IBM through the classic muscle involvement and the presence of often profound fatty infiltration [17, 18] (Fig. 17.9). The medial head of the gastrocnemius and flexor digitorum profundus muscles are commonly involved. IBM also frequently involves the anterior compartment muscles of the thigh, but unlike polymyositis and dermatomyositis, the rectus femoris muscle is notably spared in IBM [14] (Fig. 17.10 and Table 17.2).

17.5 Myonecrosis

Myonecrosis is the infarction of skeletal muscle. It has many etiologies such as trauma (e.g., crush injury), compartment syndrome, prolonged immobilization, poorly controlled diabetes, radiation treatment, and toxin (e.g., snake venom) [19]. On MRI, the infarcted muscle typically demonstrates nonspecific swelling and edema with heterogeneity that may suggest the presence of necrosis [20]. The feathery, striated pattern of muscle architecture usually remains visible. After contrast administration, infarcted muscle does not enhance (Fig. 17.11). It may not be feasible to inject contrast, however, if acute myonecrosis is complicated by rhabdomyolysis. Myonecrosis is sometimes incomplete. Enhancing linear and curvilinear foci indicate residual viable muscle tissue along vascular pedicles [21]. Once the healing process begins, contrast MRI can show

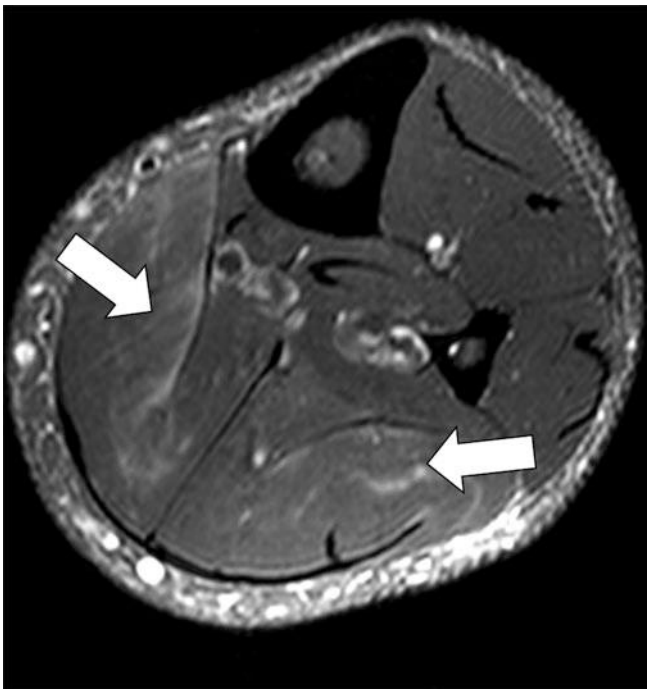

Fig. 17.4 Axial fat-saturated T2 MR image of the left calf in a 31-year-old male with severe calf pain after cross-fit workout. The feathered edema in the posterior compartment musculature (white arrows) is consistent with delayed onset muscle soreness (DOMS). This finding may persist for months following the initial insult

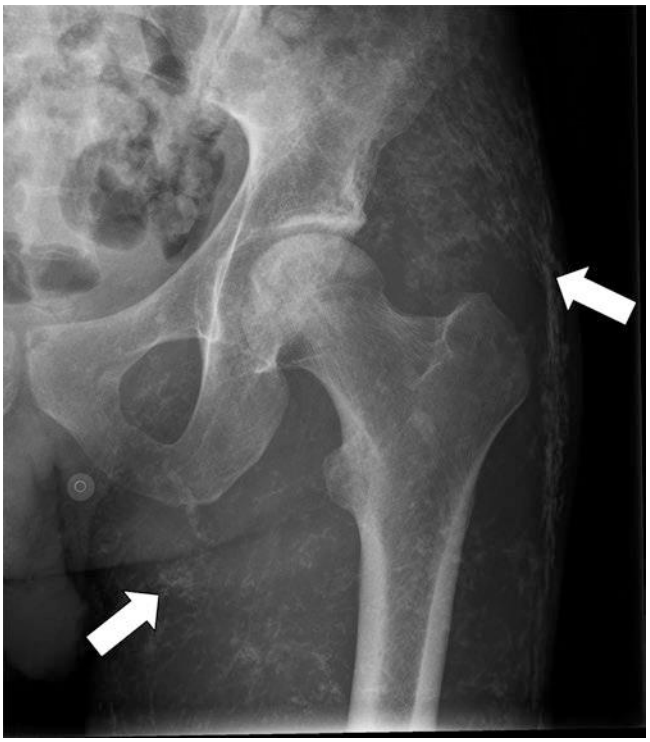

Fig. 17.6 Anteroposterior radiograph of the left hip demonstrates "sheet-like" linear calcifications (white arrows) within the soft issues in a patient with known dermatomyositis

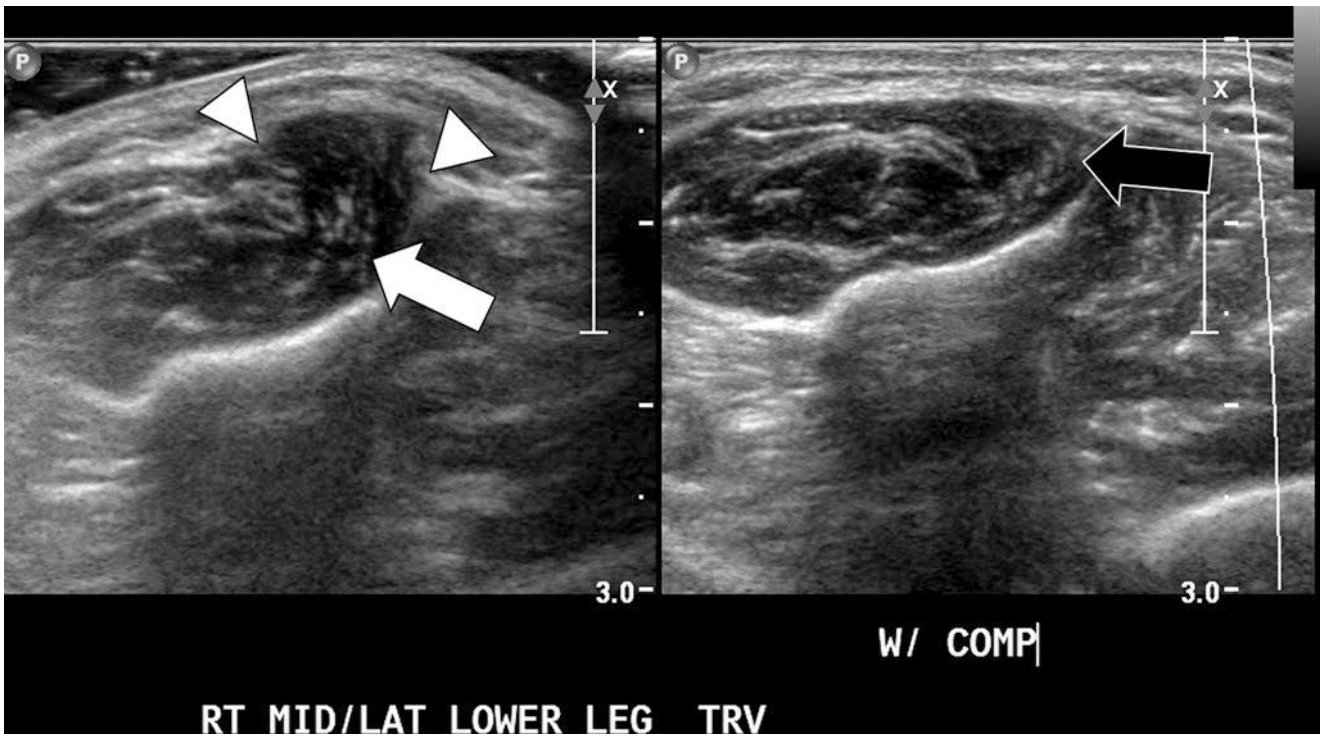

Fig. 17.5 Transverse gray-scale sonographic image of the right lower leg anterior compartment demonstrates a short segment defect in the superficial fascia (white arrowheads) with associated hypoechoic mushroom-shaped herniation of the peroneus longus muscle belly (white arrow). Comparison images of the normal left peroneus longus muscle are provided (black arrow)

rim enhancement due to granulation tissue at the interface of viable and nonviable tissue. This appearance can be confusing because it can mimic a phlegmon or abscess. In the late stages of healing, chronic sequelae of myonecrosis include cystic cavitation and dystrophic calcification.

17.6 Compartment Syndrome

In compartment syndrome, elevated pressure reduces arterial blood flow to muscles. The clinical diagnosis is confirmed by measuring intracompartmental pressure. Decreased circulation causes ischemia that can be reversed if the pressure is relieved. If intracompartmental pressure rises above perfusion pressure, blood flow is arrested causing irreversible myonecrosis. Infarcted muscle cannot be salvaged. The mostcommon etiologies include trauma (crush injury, fracture), burn, overexertion, infection, and prolonged compression as might result from overly tight bandaging. The anterior compartment of the lower leg is particularly susceptible to compartment syndrome due to its confinement by surrounding

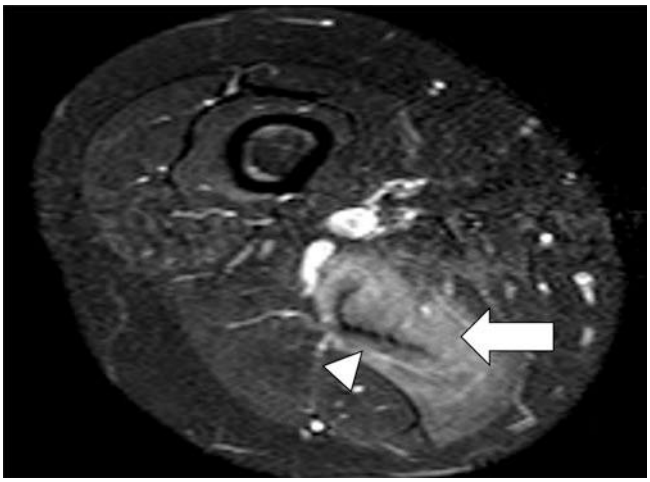

Fig. 17.7 Fat-saturated STIR MR image of the right thigh in a patient with known Sjogren's disease and polymyositis demonstrates marked diffuse edema isolated to the semimembranosus muscle (white arrow). Notice the diffuse endomysial pattern of muscle involvement with sparing of the fascia and myotendinous junction (white arrowhead)

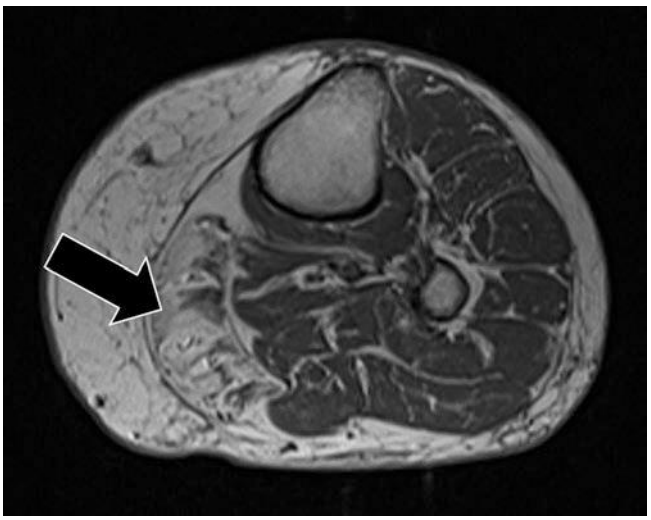

Fig. 17.9 Axial T1 MR image of the left lower leg in an 82-year-old male demonstrates profound isolated fatty infiltration of the medial head of the gastrocnemius muscle (black arrow). This pattern when symmetric is in keeping with chronic changes of inclusion body myositis

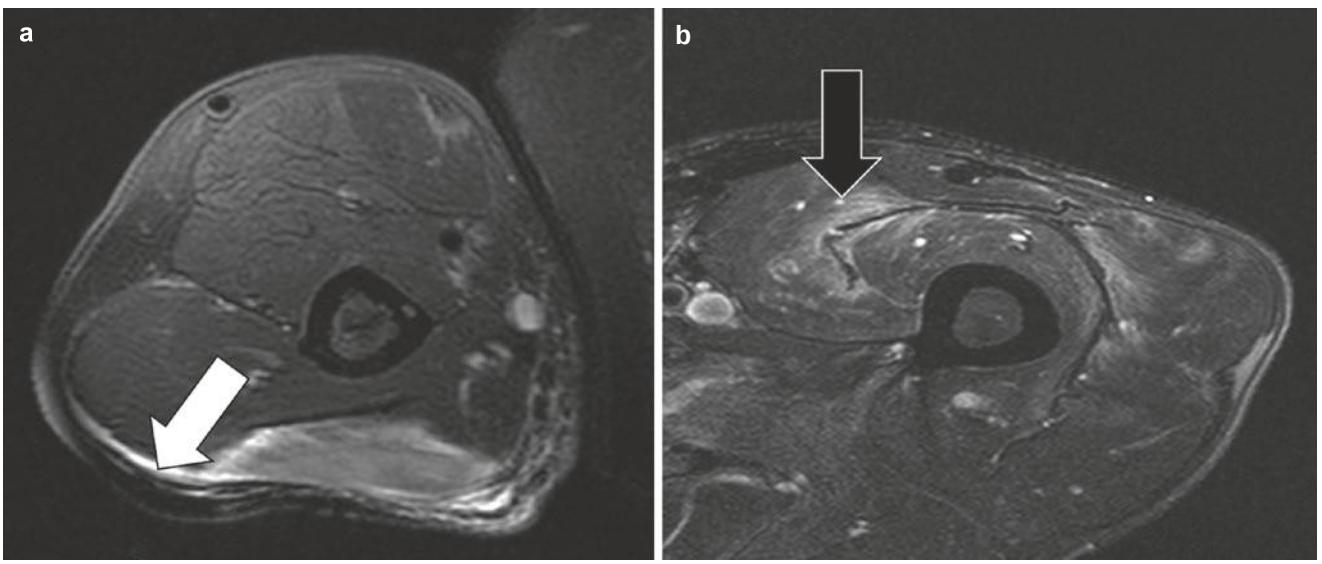

Fig. 17.8 Fat-saturated STIR MR images of the upper right arm (**a**) and right thigh (**b**) in a 27-year-old patient with dermatomyositis. Notice the perimysial pattern of edema with preferential involvement of the superficial fascia (white arrow) and myotendinous junction (black arrow)

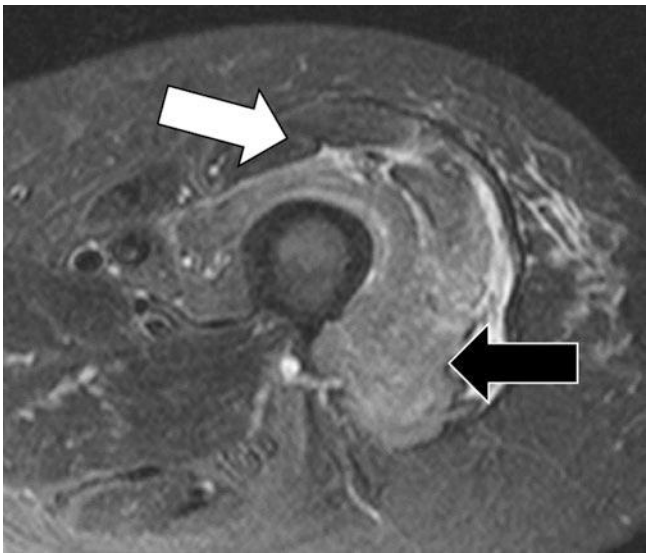

Fig. 17.10 Axial fat-saturated STIR MR image of the left thigh in a patient with inclusion body myositis and acute pain. Notice the diffuse edema in the anterior compartment musculature (black arrow) with notable sparing of the rectus femoris (white arrow)

Table 17.2 Clinical and imaging features of idiopathic myopathies [11, 12, 14, 17, 18]

		Inclusion body
Polymyositis • Symmetric • Proximal > distal • Female > male • Adults peak 30–60 • Endomysial involvement (diffuse muscle edema on MR) • Anterior thigh compartment	Dermatomyositis • Symmetric • Proximal > distal • Female > male • Juvenile and adult onset • Classic dermal calcifications • Perimysial involvement (facial or myotendinous edema on MR) • Anterior thigh compartment	myositis • Symmetric • Proximal = distal • Male > female • Elderly predisposition • Fatty infiltration • Anterior compartment with rectus femoris sparing • Classic muscle involvement (medial head of the gastrocnemius, flexor digitorum profundus)

bone and fascia. Compartment syndrome can be acute or chronic. Acute compartment syndrome is a surgical emergency that requires fasciotomy for decompression and preservation of remaining viable tissue. Chronic and exertional compartment syndromes are aggravated by intense activity. On US and MRI, edema and swelling efface the normal muscle striations [22]. Following contrast administration, ischemic muscle enhances heterogeneously, whereas infarcted muscle lacks enhancement. Long-term complications include scarring, atrophic change, and mineralization. Calcific myonecrosis represents a severe complication of post-traumatic compartment syndrome and typically affects the anterior compartment of the lower leg.

17.7 Myositis Ossificans

Heterotopic ossification is biologically and histologically identical to normal bone but is located in soft tissues [23]. Many different insults can damage tissue and trigger a physiological reaction that leads to the formation of heterotopic bone. For example, orthopedic surgeries may be complicated by postoperative heterotopic ossification following hardware implantation, arthroplasty, and fracture fixation. Juxtaarticular heterotopic ossification may develop in immobilized patients after severe burn or brainor spinal cord injury. Surgical resection may be necessaryto relieve debilitating pain and mechanical symptoms. On radiographs, mature heterotopic ossification demonstrates a pathognomonic cortical shell. On CT, low attenuation of the central cavity follows the attenuation of fat in cancellous bone. On MRI, heterotopic ossification can be misdiagnosed as lipoma because the cortical shell can have the appearance of a fibrous pseudocapsule, and the cancellous bone can demonstrate reticulations similar to adipose tissue [24].

The most frequent type of heterotopic ossification is myositis ossificans (also known as myositis ossificans circumscripta and myositis ossificans traumatica) whichusually results from traumatic injury [25]. Myositis ossificans evolve in three general stages [26]. During the first 3–4 weeks, tissue injury causes organizing fibroblastic reaction, osteoblastic differentiation, and osteoid formation. During the second 3–4 weeks, the osteoid matrix becomes mineralized and produces immature lamellar bone. Finally, after 8–10 weeks, immature bone progresses to mature bone with characteristic cortex and intramedullary cavity. The imaging findings evolve with each of the three general stages. Radiographs are negative at first. If the patient presents with a painful, palpable mass, MRI is requested because of the concern for neo-plasm. MRI features depend on the degree of tissue damage and surrounding inflammation [27]. MRI can show a mass-like lesion with enhancement following contrastadministration simulating sarcoma. If the lesion is biopsied at this early stage, histopathological analysis can leadto the misdiagnosis of pseudo-malignancy [28]. In the middle stages of evolution, radiographs and CT show azonal arrangement of perimeter mineralization. After full maturation, myositis ossificans exhibits a pathognomonic rind of cortex that is better characterized by radiographsand CT than MRI (Fig. 17.12).

Key Point

- In the early stage of myonecrosis, MRI can show an enhancing mass-like lesion simulating sarcoma.

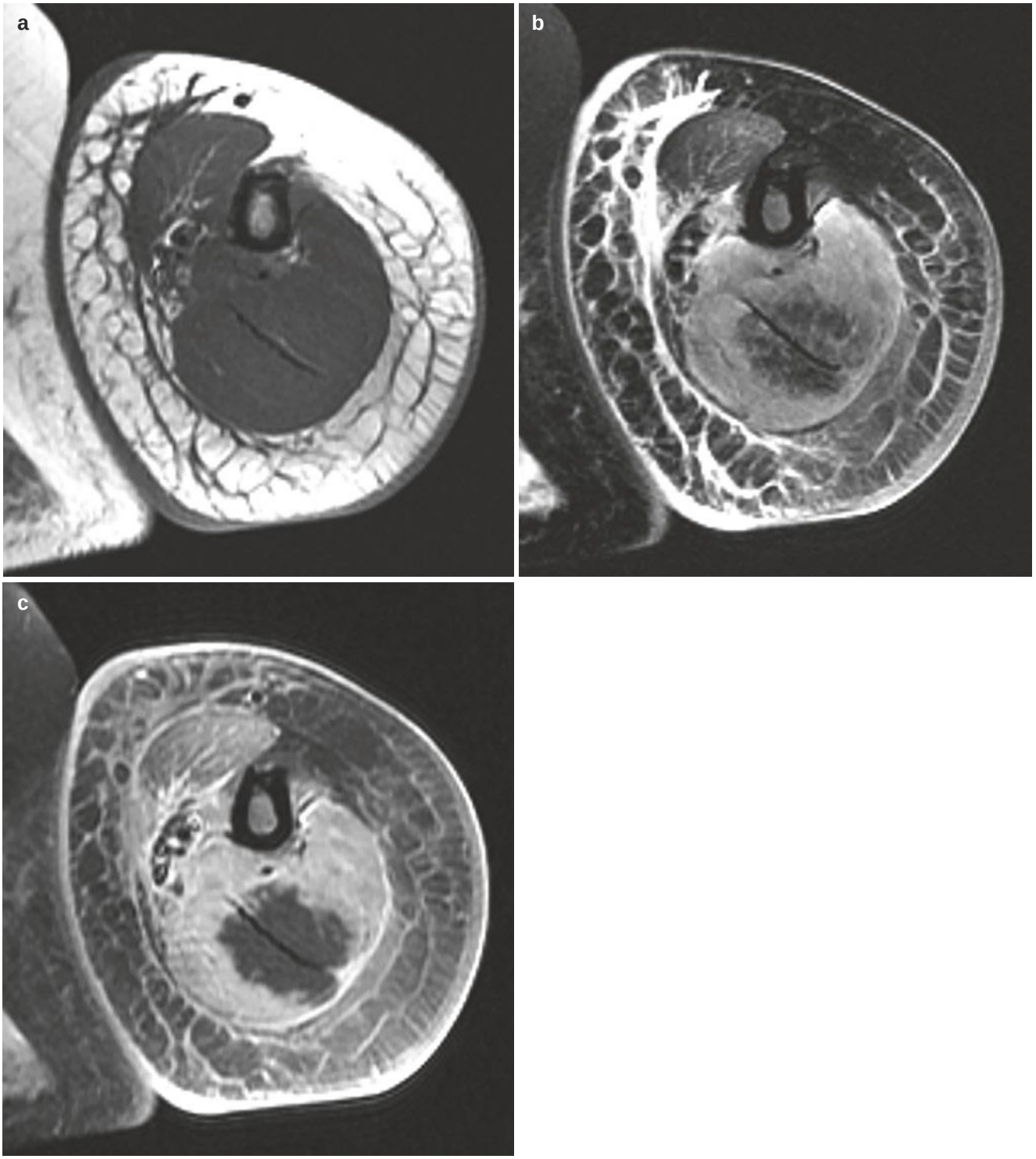

Fig. 17.11 Myonecrosis in a 73-year-old woman complaining of arm pain following drug overdose and stroke. (**a**) T1-weighted axial image shows swelling of the triceps muscle. (**b**) T2-weighted, fat-suppressed axial image shows diffuse edema of triceps muscle and low-signal region surrounding the central tendon. (**c**) Following intravenous contrast administration, T1-weighted, fat-suppressed axial image shows non-enhancing infarcted muscle corresponding to the low-signal T2-weighted region

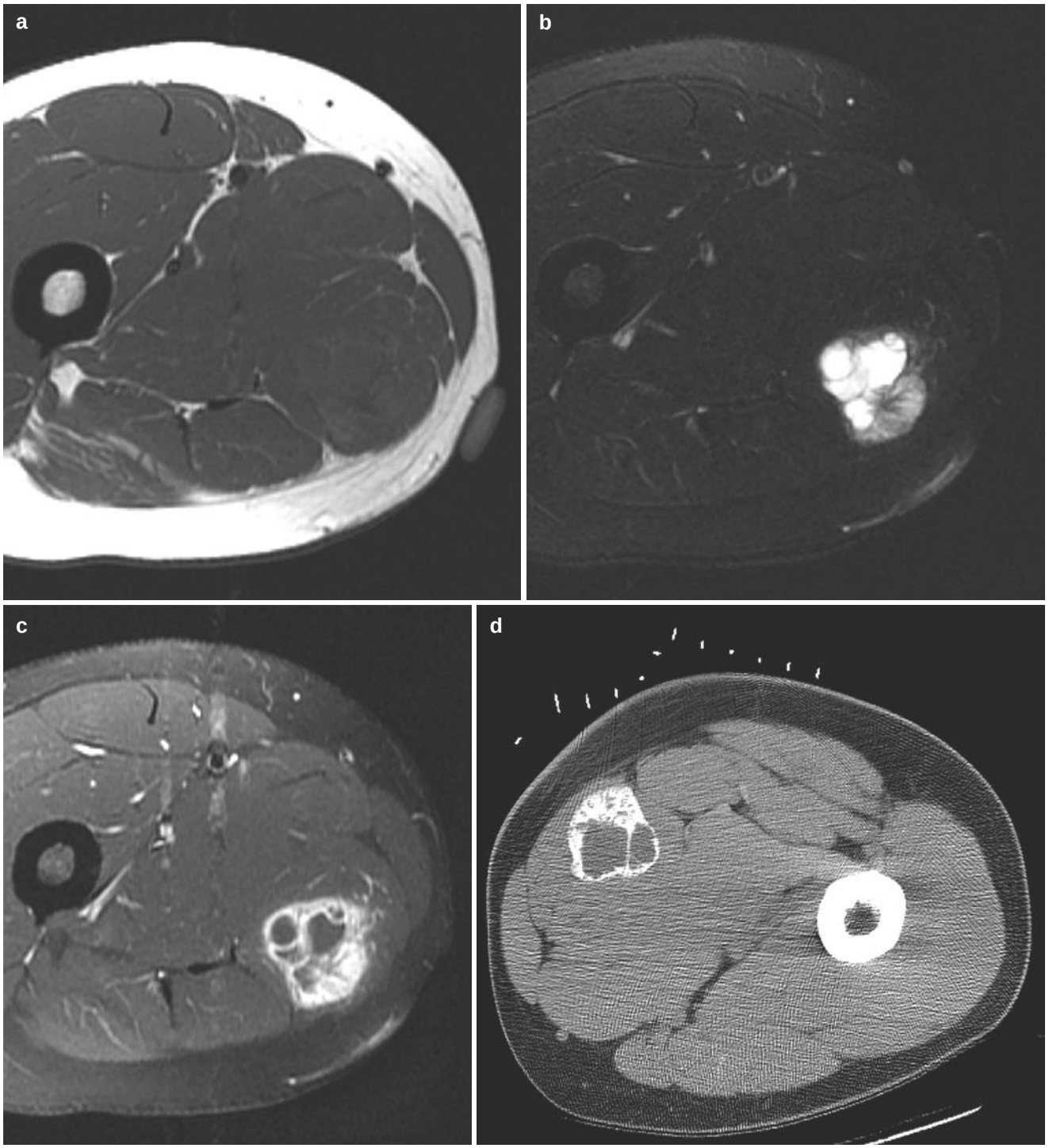

Fig. 17.12 Myositis ossificans in a 31-year-old man presenting with a painful, palpable thigh mass. (**a**) T1-weighted axial image demonstrates skin marker and swelling of underlying adductor muscle. (**b**) T2-weighted, fat-suppressed axial image demonstrates a sharply demarcated mass with cystic and solid regions. (**c**) Following intravenous contrast administration, T1-weighted, fat-suppressed axial image demonstrates dense enhancement of periphery, septations, and solid regions. (**d**) The patient was referred for CT-guided biopsy 2 weeks after the MRI study. With the patient prone, axial image obtained for procedural planning demonstrates grid placement and mineralized lesion corresponding to the MRI abnormality. The biopsy was canceled due to predominantly peripheral mineralization in pattern typical for myositis ossificans

17.8 Myopathy

Myopathy refers to the dysfunction of skeletal muscle and encompasses a spectrum of diseases. Symptoms include myalgias, muscle tenderness, and weakness. Hereditary myopathies result from enzymatic deficiencies and inborn errors of metabolism (e.g., glycogen storage disease). Endocrine myopathies are caused by adrenal, thyroid, parathyroid, and pituitary disorders. Myopathies may be induced by numerous different drugs (e.g., statin, steroid). Myositis is a subcategory of myopathy and refers to the presence of inflammation on histopathological analysis. Inflammatory myopathies are idiopathic, immune-mediated diseases such as dermatomyositis, polymyositis, and inclusion body myositis. They include conditions that affect multiple organs such as sarcoidosis. Myositis can be associated with viral infection (e.g., HIV) or bacterial infection. A complication of bacterial infection is pyomyositis. On MRI, edema-like signal, fatty muscle infiltration, and atrophic changecan correlate with disease activity, intensity, and chronicity [19]. On fluid-sensitive MRI sequences, active disease locations show edema-like signal that can provide targets for biopsy [29]. Regions of active inflammation enhance after contrast administration. T1-weighted MRI images are useful for estimating the degree of muscle atrophy and fat replacement related to long-standing or burned-out myositis. Whole-body MRI provides an assessment of disease distribution and helps to gauge therapeutic response. PET can demonstrate corresponding regions of FDG uptake due to hypermetabolism.

17.9 Necrotizing Fasciitis

In necrotizing fasciitis, aggressive bacterial infection spreads rapidly between muscle compartments along deep fascial planes and subcutaneous fat. The organisms produce toxins that infarct and liquefy muscle and fat. After a short period of nonspecific symptoms, necrotizing fasciitis swiftly progresses to limb discoloration, systemic toxicity, and sepsis. Although antibiotics can be successful against nonnecrotizing cellulitis and fasciitis, necrotizing infections are often fatal without surgical treatment such as debridement, fasciotomy, or amputation [30]. In patients with suspected necrotizing fasciitis, CT is obtained first because of its availability and sensitivity for gas in the soft tissues. The presence of gas is a specific but insensitive imaging sign. Therefore, the lack of gas cannot exclude necrotizing fasciitis. CT and MRI depict the size and locations of fluid collections [31]. Although intravenous contrast can better delineate muscle liquefaction and tissue infarction, intravenous contrast is contraindicated when necrotizing fasciitis is complicated by rhabdomyolysis and renal failure. Nonspecific imaging features include subcutaneous edema, muscle edema, fascial enhancement, and small intracompartmental fluid collections [32].

Key Point

- In patients with suspected necrotizing fasciitis, the presence of gas on CT is a specific but insensitive imaging sign. The lack of gas cannot exclude necrotizing fasciitis.

17.10 Denervation Myopathy

Denervation occurs when the nerve supply to muscle is partially or completely blocked. This blockade can be symptomatic or asymptomatic and temporary or permanent. More common etiologies include penetrating trauma, surgical transection, and prolonged stretching or compression. Less common etiologies include autoimmune disease, viral infection, and carcinomatosis (nerve infiltration by malignancy). In some locations such as the spinoglenoid notch in the shoulder, slowly growing lesions (e.g., paralabral cyst, intra-neural ganglion) may compress the neurovascular bundle and gradually lead to myopathic symptoms. In the acute stages of denervation, MRI shows diffuse muscle edema with uniform contrast enhancement. Muscle bulk remains normal. In the later stages of denervation, MRI findings canoverlap with other disorders. Denervated muscle can becomeatrophic and infiltrated by fat. Edema-like signal may or may not be present. Parsonage-Turner syndrome, also known as idiopathic brachial neuritis, affects one or more muscles of the shoulder girdle. Progressive pain and weakness can mimic the clinical presentation of other shoulder disorders such as rotator cuff tear or capsulitis and, therefore, lead to adelay in diagnosis [33]. In Parsonage-Turner syndrome, supraspinatus and infraspinatus muscles are more commonly involved than deltoid, teres minor, and subscapularismuscles.

17.11 Muscle Lesions: Differential Considerations

Myotendinous strain shows pathognomonic localization to the myotendinous junction [34]. Other disorders show a random distribution in muscle. Any localization to the myotendinous junction is coincidental. For example, focal myositis may simulate acute strain if it happens to be contiguous with the myo-tendinous junction. However, it is usually located in the muscle

belly separate from the myotendinous junction. Intramuscularneoplasms can displace the myotendinous junction due to mass effect, but they rarely disrupt or encase it.

In myonecrosis, nonviable and adjacent viable muscle can have identical appearances on T1-weighted, T2-weighted, and STIR images because infarcted muscle maintains its normal fiber architecture, similar to necrotic cancellous bone which maintains its normal trabecular architecture.Therefore, the diagnosis of myonecrosis is made with greatest confidence when intravenous contrast is injected. The non-enhancing, avascular tissue stands out prominently against the densely enhancing viable muscle. Although infection (e.g., pyomyositis) can also cause diffuse muscle edema, non-enhancing phlegmon or abscess is distinguishedfrom myonecrosis by the disruption of muscle architecture.

On MRI, hematoma is characterized by a bull's-eye configuration due to degradation of blood products, hemoglobinbreakdown, and hemosiderin deposition. On T1-weighted images, the central region is increased in signal intensity. Neoplasms containing fat, such as hemangioma, angiolipoma, and liposarcoma, may also have a central region that is increased in T1-weighted signal intensity and, therefore, similar in appearance to intramuscular hematoma. On fat-suppressed images, however, the signal from fat is decreased,whereas the signal from hematoma is unchanged. Neoplasmscomplicated by hemorrhagic necrosis also can be mistaken for hematoma, but they have mass affect and displace sur- rounding structures, including the myotendinous junction. Hemorrhagic necrosis can be mistaken for enhancement fol-lowing intravenous contrast administration.

Myositis ossificans can pose diagnostic pitfalls. It may present as a painful, palpable soft tissue mass with vague, remote, or absent history of trauma. Clinically, these symptoms and signs are suspicious for neoplasm. On MRI, myositis ossificans has a variable appearance depending on the age of injury, extent of myonecrosis, and degree of adjacent inflammation. In earlier stages, a mass-like abnormalitycan enhance densely after contrast administration, support the clinical suspicion of malignancy, and result in patient referral to an oncological center for biopsy and surgical resection. Imaging evidence of benign myositis ossificans may not occur until the time of CT-guided biopsy, when thepre-procedural scan demonstrates a characteristic peripheralrim of calcification (Fig. 17.12). In that case, the biopsy canbe canceled.

17.12 Concluding Remarks

Although muscle is susceptible to multiple traumatic, inflammatory, neoplastic, and neuropathic disorders, imaging oftenenables a narrow differential diagnosis. Acute traumatic andactivity-related injuries such as strain, contusion, and overuse may be complicated by myonecrosis, myositis ossificans, orcompartment syndrome. Chronic sequelae are less specific, including muscle atrophy and fatty infiltration. In inflammatory myopathy, imaging is useful clinically in guiding biopsyand assessing treatment response. Imaging has a critical rolein the early diagnosis and characterization of necrotizing fasciitis.

Take Home Messages

- Different imaging modalities have targeted roles in assessing muscle disorders.
- In most muscle disorders, imaging enables a narrow differential diagnosis.
- Clinical and imaging features of early myositis ossificans can simulate sarcoma.
- Contrast administration may be necessary to confirm the diagnosis of myonecrosis.

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