# 3. Tendons and Muscles

**BOOK CHAPTER**

# Tendons and Muscles

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## How to Image Tendons

• *Coils and patient position:* Whether or not a coil should be used is based entirely on the anatomy to be imaged. Generally, surface coils improve images and should be used. For large areas, such as the thighs or pelvis, this approach is not practical, and surface coils are not used. Patients should be positioned as if the nearest joint were being imaged.

• *Image orientation:* Generally, tendons are best imaged transversely (perpendicular to their long axis). Occasionally, other planes are helpful to image tendons in their entire length. The triceps, quadriceps, and Achilles tendons are depicted well on axial and sagittal images. The hamstring tendons in the pelvis and the supraspinatus tendon in the shoulder are shown

well on coronal and axial images. Given only one option for an imaging plane through a tendon, axial images are generally the most useful.

• *Pulse sequences and regions of interest:* T1-weighted (T1W) and some types of T2-weighted (T2W) images are required for complete evaluation of tendons. T2W sequences are useful for showing abnormal fluid surrounding the tendon (tenosynovitis) and tears and most other kinds of tendon pathology. We prefer fast spin echo with fat saturation or short tau inversion recovery (STIR) sequences for T2W images of tendons. Slice thickness and fields of view are determined by the size of the body part being imaged. A good rule of thumb is that the same field of view and slice thickness that are used to image the adjacent joint would suffice to image a tendon in that same region.

• *Contrast:* There is no need to do contrast-enhanced studies for tendon evaluation, except to evaluate possible active tendon disease in inflammatory conditions (e.g., rheumatoid arthritis).

## Normal Tendons

## Anatomy

Tendons are avascular structures that attach muscles to bones. They are made of dense fascicles of collagen fibers. The fascicles of collagen are composed of smaller units, called *microfibrils* . Microfibrils interdigitate with one another in a regular and structured fashion to form extremely tight bonds, giving tendons their strength. The microfibrils are made of a protein called *tropocollagen* , which consists of three polypeptide chains arranged

in a triple-helix configuration. The helical configuration of the protein tightly binds molecules of water so that tendons have low signal intensity because the hydrogen ions in water are not mobile.

Many tendons are invested within a tendon sheath, which either partially or completely covers the tendon. Tendon sheaths are present where tendons pass through fascial slings, beneath ligamentous bands, or through fibro-osseous tunnels. They exist where closely apposed structures move relative to one another to decrease friction. The microscopic structure of a tendon sheath is similar to the synovial membrane that lines joints. During fetal development, the tendon invaginates the tendon sheath so that there are inner (visceral) and outer (parietal) layers of the sheath that are closely apposed to each other. A mesotendon is formed where the tendon invaginated the sheath. The mesotendon carries blood vessels and is located on the nonfrictional surface of the tendon. A thin layer of synovial fluid exists between the visceral and parietal layers of the tendon sheath and allows for smooth gliding of the tendon.

Some tendons are located mainly outside of the muscle, such as the distal biceps tendon at the elbow. Other tendons have long segments that are surrounded by muscle and have very little exposed tendon, such as the brachialis at the elbow ( Fig. 3.1 (f0010) ).

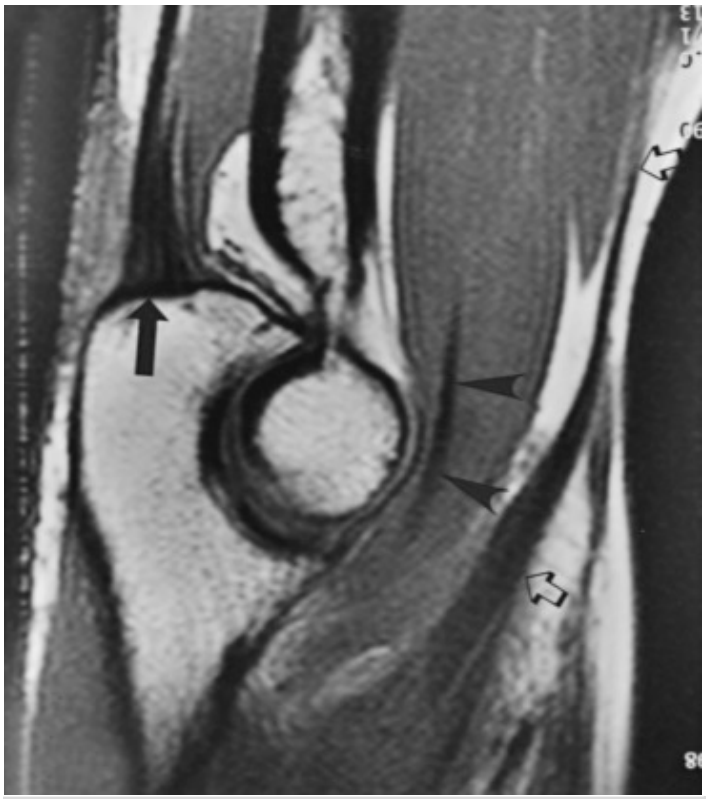


**Fig. 3.1**

**Normal tendons.** T1 sagittal image of the elbow. The anteriorly located biceps tendon ( *open arrows* ) is low signal and has a long segment that is not surrounded by muscle. The brachialis tendon ( *arrowheads* ) is also a typical low signal tendon, but it is surrounded by muscle with little exposed tendon. The triceps ( *solid arrow* ), in contrast to most tendons, normally has vertical striations of low and intermediate signal.

## MRI of Normal Tendons

Normal tendons have so few mobile protons that they are usually low signal intensity on all pulse sequences. The major exceptions to this rule include the quadriceps tendon at the knee and distal triceps tendon at the elbow and the Achilles tendon, which have a striated appearance with alternating areas of linear low and intermediate signal intensity (similar to the distal anterior cruciate ligament in the knee) (see Fig. 3.1 (f0010) ). This striated appearance is caused by the longitudinal arrangement of coarse fasciculi and by

the fact that several tendons are fusing to form a single, conjoined tendon. The longitudinal striations in the triceps, quadriceps, and Achilles tendons must not be mistaken for pathology. Similarly, there is a solitary vertical line of high signal intensity in the midsubstance of many normal Achilles tendons, which probably represents the site where the soleus and gastrocnemius tendons (which make up the Achilles tendon) are apposed to one another, or else a vascular channel in the tendon.

There are certain other exceptions to the rule that normal tendons are low signal intensity on all pulse sequences ( Box 3.1 (b0010) ). Many tendons may show slightly increased signal intensity near their osseous insertions (the posterior tibial tendon, for example). This increased signal intensity occurs because tendons may fan out as they come to attach to a bone, and nontendinous fatty material is interposed between tendon fibers ( Fig. 3.2 (f0015) ).

**BOX 3.1**

**High Signal Within Tendons**

## Normal Causes

- Coarse fascicles or several tendon layers fusing
  - Quadriceps and distal triceps tendons
- Osseous insertions
  - Tendons spread, change orientation
- Magic angle phenomenon
  - Tendon orientation at 55 degrees to bore of magnet

## Abnormalities

- Myxoid degeneration
- Partial or complete tears
- Xanthoma, tumor, gout deposits

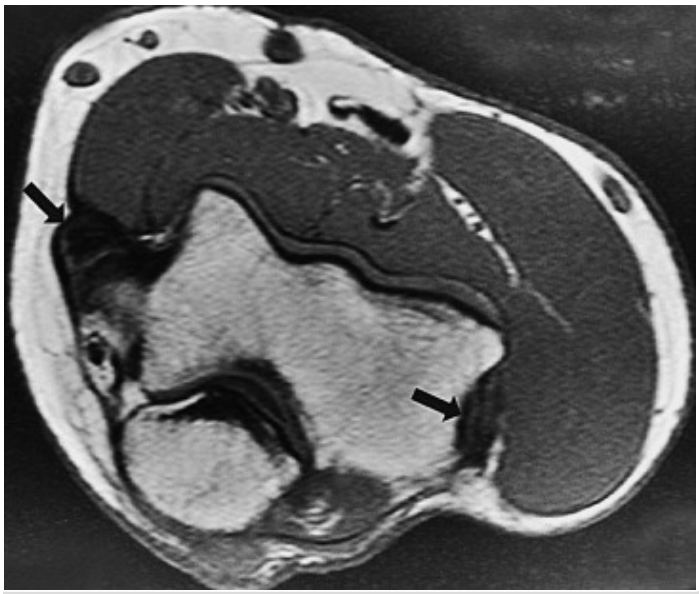


**Fig. 3.2**

**Normal tendon intermediate signal.** T1 axial image of the elbow. The attachment sites of some tendons to bone ( *arrows* ) result in areas of intermediate signal within the tendon, which is normal and is caused by diverging collagen fascicles, rather than partial tendon tears.

A second major reason for a normal tendon having increased signal intensity is the result of the magic angle phenomenon. The magic angle phenomenon results from the fact that tendons are anisotropic structures. When tendons are oriented at an angle of about 55 degrees to the bore of the magnet, there is high signal intensity within the tendon on short TE sequences (e.g., T1W, proton density, and gradient echo sequences). Whether high signal on short TE sequences is from the magic angle phenomenon or from pathology can generally be determined in various ways:

1. Use a pulse sequence with a long TE, in which case the high signal intensity disappears and the tendon appears normal.

2. Observe that the tendon is of normal caliber.

3. Reposition the body part being imaged so that the tendon is imaged at a different angle relative to the bore of the magnet.

Most tendons are round, oval, or flat when imaged transversely. Tendon sheaths are not normally seen on magnetic resonance imaging (MRI) unless fluid is present in the sheath. Small amounts of fluid may be seen in certain tendon sheaths, particularly in the ankle and wrist. Our general rule is that the fluid should not be considered abnormal unless it completely surrounds the circumference of the tendon.

## Tendon Abnormalities ( Box 3.2 (b0015) )

The major abnormalities that may affect tendons include tendon degeneration, tenosynovitis, partial tears, complete tears, subluxation or dislocation, xanthoma formation, deposits of calcium hydroxyapatite or calcium pyrophosphate crystals, gouty tophi, and clear cell sarcoma. Giant cell tumor of the tendon sheath is a common mass that arises from the tendon sheath. The most common tendon abnormalities seen on MRI are degeneration, tenosynovitis, and tears ( Fig. 3.3 (f0020) ).

**BOX 3.2**
**Tendon Abnormalities**

- Degeneration * (fn0010) * Most common.

- Tenosynovitis * (fn0010)
- Partial or complete tears * (fn0010)
- Subluxation or dislocation
- Xanthoma formation
- Gout, hydroxyapatite, or other crystals
- Giant cell tumor of tendon sheath
- Clear cell sarcoma

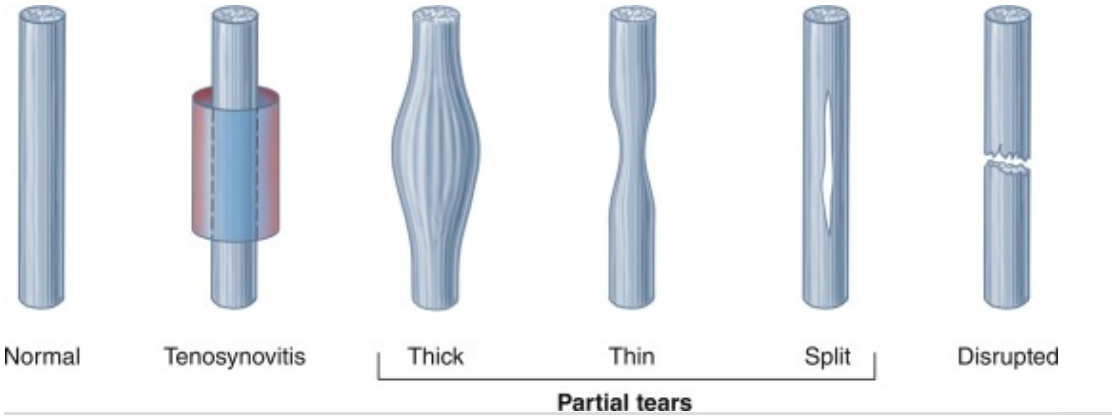



**Fig. 3.3**

**Tendon inflammation/tears.** Diagram showing the changes that occur in tendons from inflammatory tenosynovitis through different types of partial tears and complete tendon disruption.

## Degeneration

Myxoid degeneration of tendons occurs with aging or from chronic overuse ( Fig. 3.4 (f0025) ). This is a painless process, but it weakens the tendon so that it is predisposed to partial or complete tears with

minimal trauma. The quadriceps, patellar, and Achilles tendons are good examples of tendons that may rupture with no or minimal trauma because of preexisting underlying tendon degeneration.

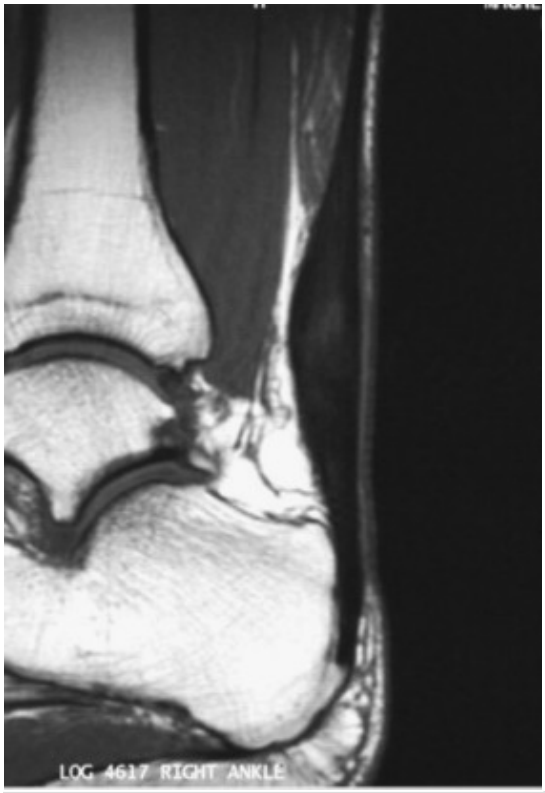



**Fig. 3.4**

**Tendon degeneration/partial tears.** T1 sagittal image of the ankle. The Achilles tendon is markedly thickened in this middle-aged patient and shows intermediate signal within its fibers representing myxoid degeneration and partial tears.

On MRI examination, a degenerated tendon has intermediate signal intensity within the substance of the tendon on all sequences. The tendon is generally normal or enlarged in caliber; this cannot be distinguished from partial tears of a tendon. Many clinicians use terms such as *tendinitis, tendinopathy,* or *tendinosis* to indicate that such abnormal signal intensity of a tendon exists but that it is impossible to give the precise cause for the findings. The term *tendinitis* should not be used because an inflammatory response in

the tendon does not occur. Another way to describe the nonspecific findings of an intrasubstance high signal in a tendon is simply to state that it is compatible with degeneration or partial tears because they generally coexist anyway.

## Tenosynovitis ( Box 3.3 (b0020) )

Fluid that completely surrounds the circumference of a tendon indicates an inflammatory process of the tendon sheath, called *tenosynovitis* . The underlying tendon may be normal or abnormal. The abnormal presence and amount of fluid are required to make this diagnosis, regardless of the status of the tendon fibers. Tenosynovitis may occur from chronic repetitive motion or stress on the tendon from overuse, from an inflammatory arthritis, or from a purulent infection.

**BOX 3.3**

**Tenosynovitis**

### Causes

- Overuse, increased stresses
- Inflammatory arthritis
- Infection

### Stenosing Tenosynovitis

- Focal, loculated fluid collections in tendon sheath, often with septations in fluid

### MRI

- Underlying tendon may be normal or abnormal
- Fluid (low signal, T1; high signal, T2) must surround the entire circumference of a tendon—meaningless if tendon sheath communicates with adjacent joint
- Septations in loculated fluid of stenosing tenosynovitis are thin, linear, low signal structures; do not confuse with mesotendon
- Pannus may be present in tendon sheaths in rheumatoid arthritis

Stenosing tenosynovitis can occur when there are focal, loculated collections of fluid in the tendon sheath. This is a common finding in the flexor hallucis longus tendon around the ankle in patients with os trigonum syndrome and in the wrist from de Quervain's stenosing tenosynovitis.

MRI of tenosynovitis shows a round collection of fluid that is low signal intensity on T1W and high signal intensity on T2W images, completely surrounding a tendon on images obtained transversely through it. The mesotendon may be identified as a thin, low signal intensity line extending from the tendon to the outer layer of the tendon sheath ( Fig. 3.5 (f0030) ). The underlying tendon may be normal or abnormal in signal intensity and caliber. Stenosing tenosynovitis can be diagnosed on MRI by the presence of focal distention of a tendon sheath with fluid and thin, linear low signal intensity septations that course through the fluid in the sheath ( Fig. 3.6 (f0035) ).

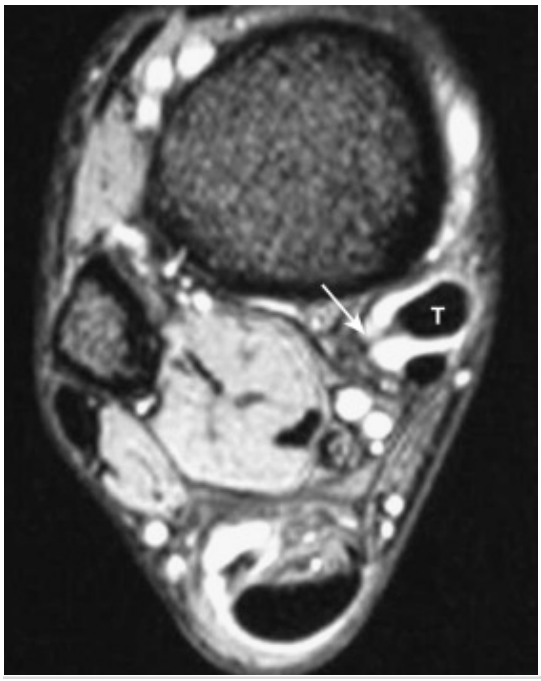



**Fig. 3.5**

**Tenosynovitis.** T2* axial image of the ankle. The posterior tibial tendon (T) is mildly enlarged but of normal signal intensity. It is surrounded by high signal fluid, representing tenosynovitis. The thin line within the fluid ( *arrow* ) is the mesotendon, where the tendon invaginated the tendon sheath during fetal development.

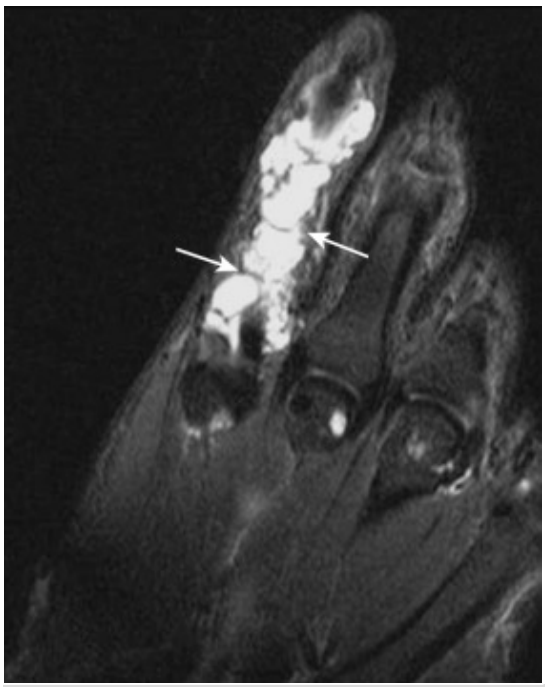


**Fig. 3.6**

**Stenosing tenosynovitis.** Fat-saturated T2 coronal image of the finger. High signal fluid from tenosynovitis surrounds the flexor tendons of the index finger. There are lobulated margins of the tendon sheath and small, linear septations within it ( *arrows* ), indicating this is stenosing tenosynovitis with scarring and inflammatory changes of the sheath.

Tendon sheaths that communicate directly with an adjacent joint (e.g., the long head of the biceps tendon in the shoulder, the flexor hallucis longus tendon at the ankle, and the iliopsoas tendon at the hip) should not be considered to have tenosynovitis simply because of the finding of fluid surrounding the circumference of the tendon. If there is an effusion in the joint, fluid can surround the tendon without the tendon or its sheath being abnormal. We consider fluid around these specific tendons to have possible clinical significance only if there is no adjacent joint effusion.

Tendons that do not have a sheath may have inflammatory changes surrounding the tendon, which is called *paratendinitis* ( Fig. 3.7 (f0040) ). MRI shows abnormal signal intensity typical of edema (low signal intensity on T1W and hyperintense on T2W images) in the soft tissues surrounding the tendon. The Achilles tendon (Kager's fat pad) is a classic location for these findings because it has no tendon sheath.

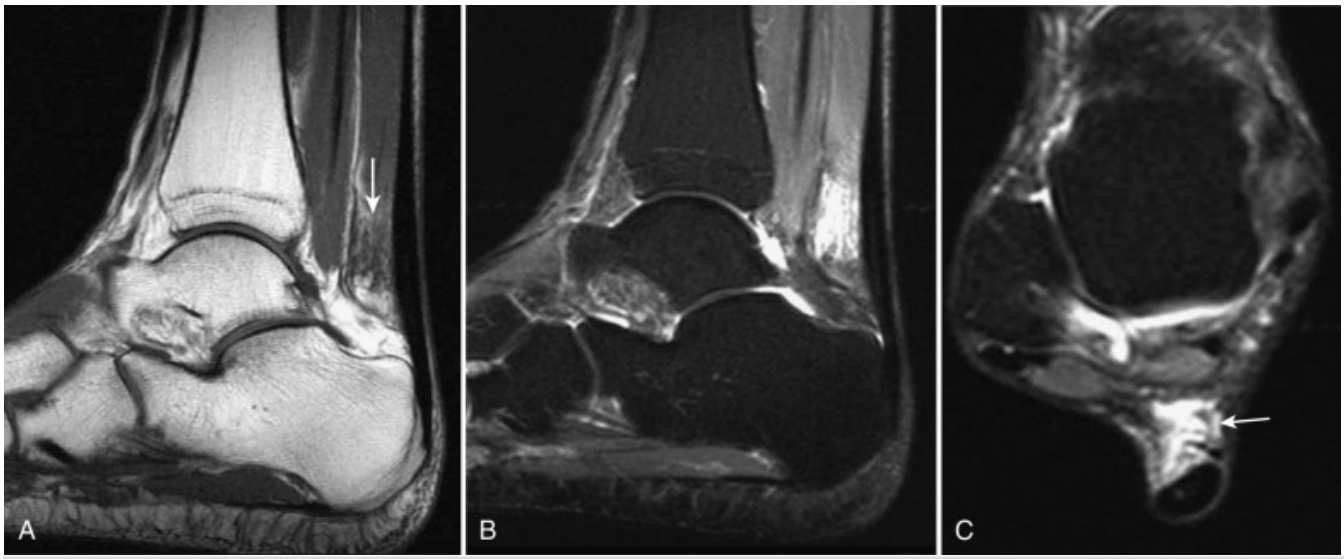



**Fig. 3.7**

**Paratendinitis. A** , T1 sagittal image of the ankle. The Achilles tendon does not have a tendon sheath, so inflammatory changes appear as edema in the fat adjacent to the tendon ( *arrow* ). **B** and **C** , STIR sagittal and axial images of the ankle. Edema anterior to the Achilles tendon manifests as high signal in Kager's fat ( *arrow* in **C** ), indicating paratendinitis.

## Tendon Tears ( Box 3.4 (b0025) )

Several conditions cause weakened tendons and predispose to tears, including chronic repetitive stresses, tendon degeneration, inflammatory processes of tendons (e.g., from rheumatoid arthritis, seronegative spondyloarthropathies, systemic lupus erythematosus, or infection of the tendon sheath), chronic renal disease, use of long-term systemic steroids and certain other medications, diabetes, and gout. Partial tendon tears represent incomplete disruption of the fibers. Complete tendon tears indicate total disruption of the fibers of the tendon so that there are two separate fragments. Partial tears often are difficult to diagnose on clinical grounds, whereas complete tears are more obvious.

**BOX 3.4**

**Factors Predisposing to Tendon Rupture**

- Tendon degeneration—occurs with age and from chronic stresses
- Chronic, repetitive stresses (overuse)
- Acute major trauma
- Diabetes
- Systemic steroids and other medications
- Rheumatoid arthritis and other inflammatory arthritides
- Chronic renal failure/hyperparathyroidism
- Infection of tendon sheath
- Gout

Partial tendon tears can have a variable appearance on MRI ( Fig. 3.8 (f0045) ). The tendon may be thickened (hypertrophic partial tear) or thinned (atrophic partial tear), or remain of normal caliber with abnormal signal (fluid signal) being the only evidence of the partial tear. An attenuated tendon is closer to complete rupture than a thickened tendon. A classification system has been proposed to describe tears based on the caliber of the tendon; we prefer simply to describe the findings (partial tear with a normal-caliber, thickened, or attenuated tendon), rather than assign a number from a classification system, because we cannot remember the system ourselves and especially because referring physicians may be

unaware of the classification system. Tendons sometimes become partially torn in a longitudinal or vertical manner, rather than transversely ( Fig. 3.9 (f0050) ). A split tendon may be functionally incompetent and act as if it is completely torn, even though it is still in continuity with the muscle and the bone. Another type of partial tear involves delamination of the tendon fibers. This diagnosis is made when fluid is seen to extend through a partial-thickness tear and track within the substance of a tendon, with partial retraction of the fibers along one of its surfaces ( Fig. 3.10 (f0055) ).

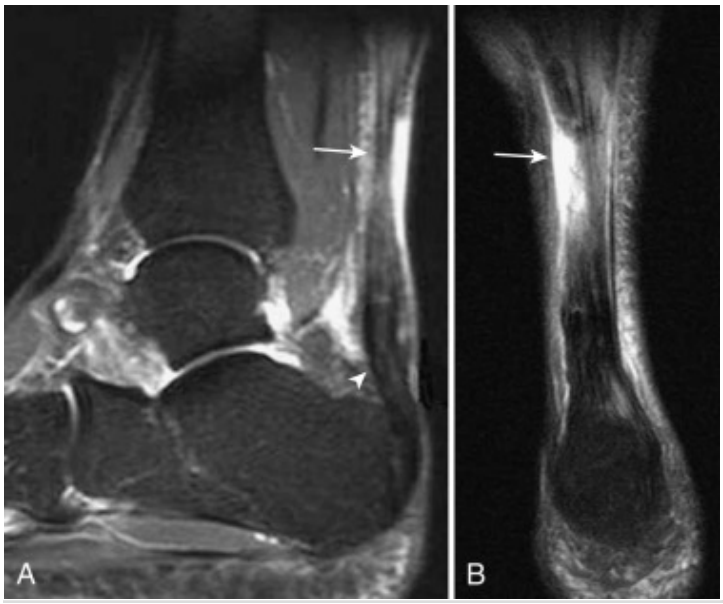



**Fig. 3.8**

**Partial tendon tear. A** , STIR sagittal image of the ankle. There is marked thickening of the distal Achilles tendon related to chronic tendinopathy and hypertrophic partial tendon tears ( *arrowhead* ). The tendon is markedly thinned proximally at the site of a high-grade partial tear ( *arrow* ). **B** , STIR coronal image of the ankle. The degree of tendon disruption is better shown ( *arrow* ).

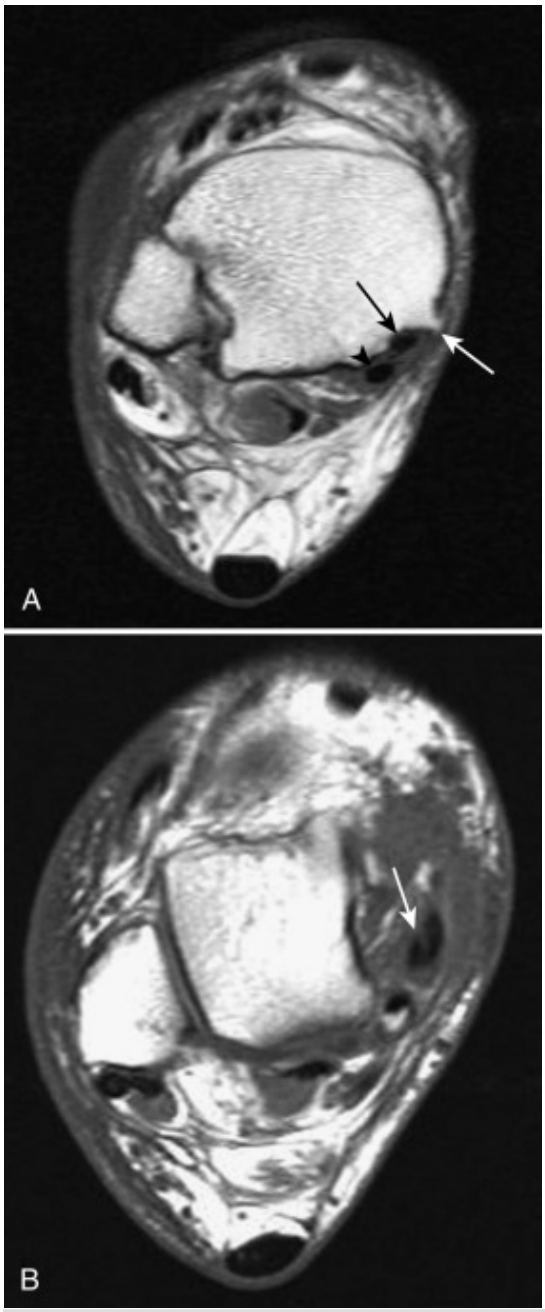



**Fig. 3.9**

**Partial tendon tear: longitudinal split. A** , T1 axial image of the ankle. The posterior tibial tendon ( *black arrow* ) is the same size as the adjacent flexor digitorum longus tendon ( *arrowhead* ), indicating atrophic partial tendon tearing (it should be approximately twice the size of the flexor digitorum tendon at this level). Note also the prominent spur arising from the medial malleolus ( *white arrow* ), a finding often present in cases of posterior tibial tendon dysfunction. **B** , T1 axial image of the ankle. The posterior tibial tendon has a splitlike tear ( *arrow* ), which extended along the length of the tendon, representing a longitudinal split tear.

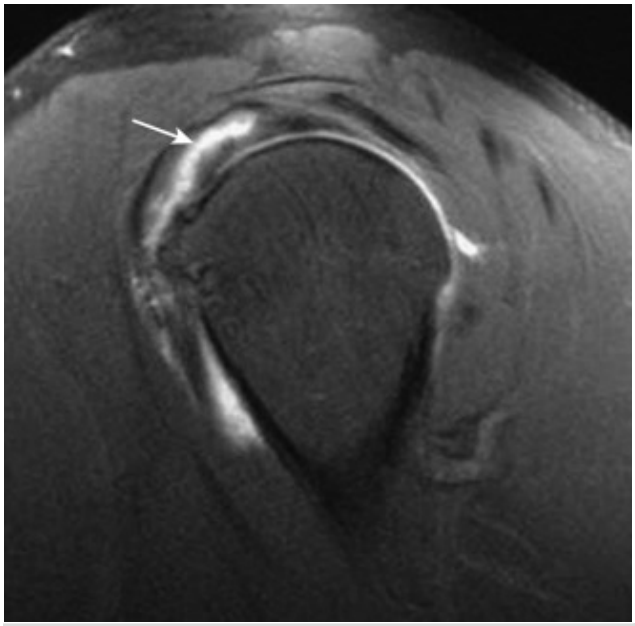


**Fig. 3.10**

**Partial tendon tear: delamination.** Fat-saturated T1 oblique sagittal image of the shoulder (MR arthrogram). There is a large partial-thickness tear of the supraspinatus tendon with horizontal splitting of the tendon ( *arrow* ), indicating delamination.

Usually there is high signal intensity in the tendon on all pulse sequences with partial tendon tears, but with chronic partial tears, there may be low signal intensity because of scarring and fibrosis. An abnormal tendon size and tenosynovitis are the only ways to recognize the tendon as abnormal in this situation. Tenosynovitis often coexists with partial tendon tears.

On MRI, complete tendon rupture appears as a focal disruption with absence of the tendon fibers for variable distances ( Fig. 3.11 (f0060) ). MRI is valuable in documenting the presence of a complete tear, showing the quality of the remaining of tendon, and showing how far retracted the remnants are; all of these features

may contribute to determining how to manage the patient. Careful search for tendons on every image is essential not to overlook tears because abnormalities may be present on only one image.

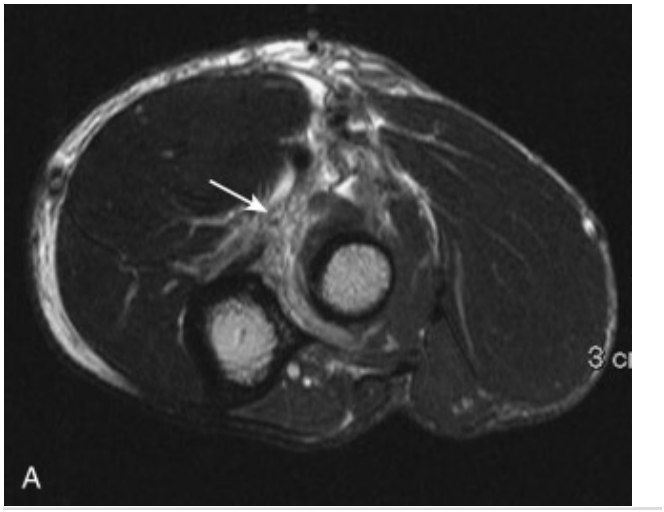
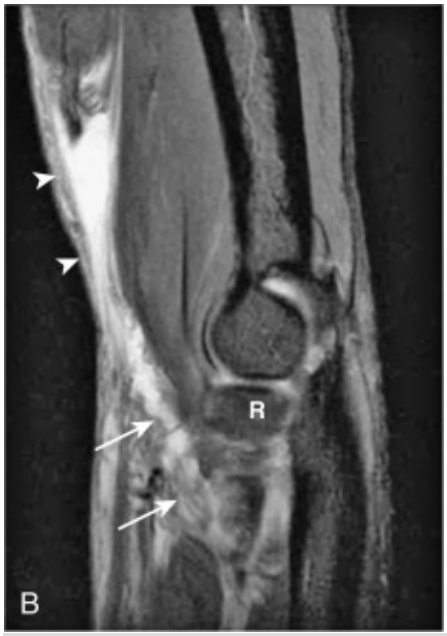
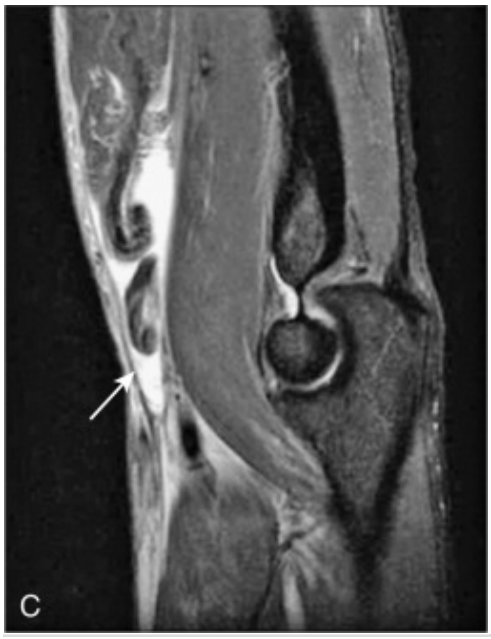



**Fig. 3.11**

**Complete tendon tear. A** , Axial image of the elbow. The distal end of the distal biceps tendon is not present in its expected location ( *arrow* ). **B** , STIR sagittal image of the elbow. Fluid is evident in the expected location of the tendon ( *arrows* ), with high signal fluid and/or hemorrhage proximally ( *arrowheads* ). *R,* Radial head. **C** , STIR sagittal image (adjacent to image in **B** ). The thickened, torn biceps tendon is retracted several centimeters above its insertion site ( *arrow* ).






## Tendon Subluxation/Dislocation ( Box 3.5 (b0030) )

Most tendons maintain a normal relationship to adjacent osseous structures by way of retinacula that hold them in place. If the retinacula become disrupted, the tendons may sublux or dislocate from their normal positions ( Fig. 3.12 (f0065) ). The tendons may have no intrinsic, underlying abnormalities, but partial tears, complete tears, and tenosynovitis are common from irritation with chronic subluxation and wear and tear on adjacent bones. The tendons that may sublux or dislocate include the extensor carpi ulnaris in the wrist, the long head of the biceps in the shoulder, the peroneal tendons over the lateral malleolus at the ankle, and the posterior tibial tendon on the medial side of the ankle. In fact, chronic tendon pathology in the posterior tibial tendon and peroneal tendons may lead to adjacent bone marrow edema in the medial malleolus and cuboid, respectively.

**BOX 3.5**

**Tendons That Sublux or Dislocate**

- Wrist
  - Extensor carpi ulnaris (medial)
- Shoulder
  - Long head of biceps (medial)
- Ankle
  - Peroneal tendons (lateral or medial)
  - Posterior tibial (medial and anterior)

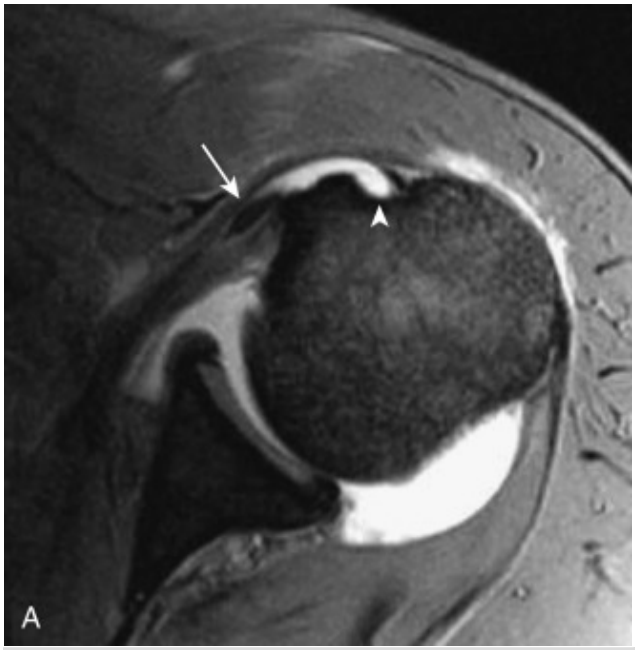
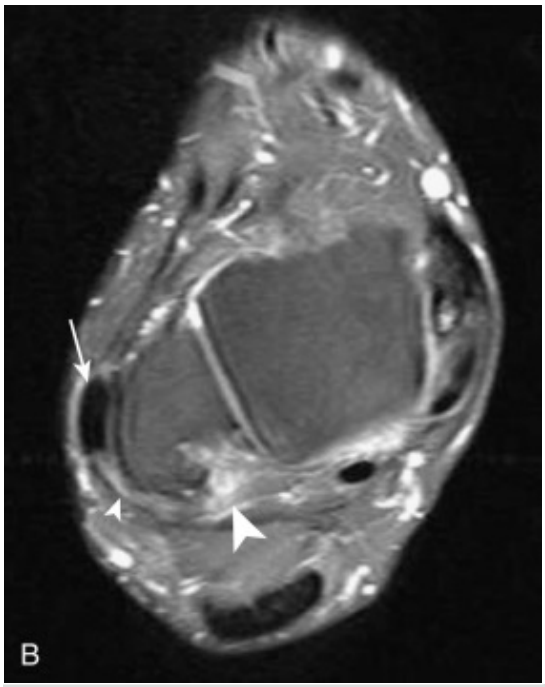


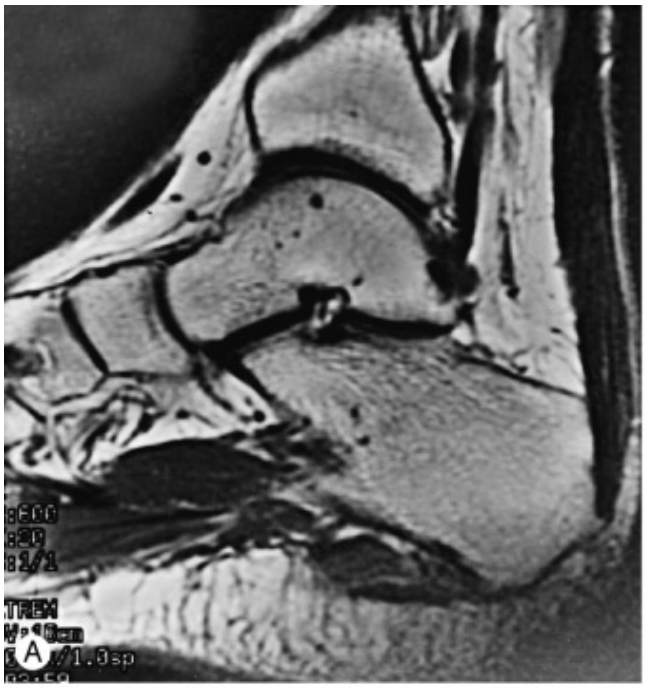

**Fig. 3.12**

**Dislocated tendons. A** , T2* axial image of the shoulder. The long head of the biceps tendon ( *arrow* ) has dislocated medially from the bicipital groove ( *arrowhead* ) and lies within a partially torn subscapularis tendon. **B** , Fat-saturated T1 axial image (postgadolinium administration) of the ankle. The peroneus tendons ( *arrow* ) have dislocated laterally from the retrofibular groove ( *large arrowhead* ) where they normally lie. The superior peroneal retinaculum has stripped away from its attachment on the fibula ( *small arrowhead* ).




## Miscellaneous Tendon Lesions

Abnormalities other than tears, tenosynovitis, and dislocations are uncommon. Xanthomas occur in patients with familial hyperlipidemia syndrome and most commonly affect the Achilles tendon and the extensor tendons of the hand ( Fig. 3.13 (f0070) ).



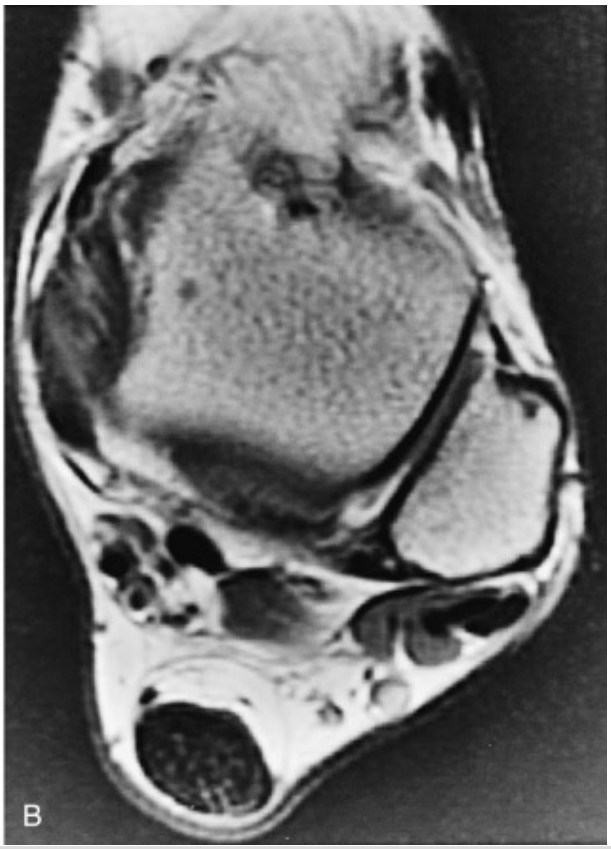



**Fig. 3.13**

**Xanthoma of the tendon. A** , T1 sagittal image of the ankle. The Achilles tendon is thickened and has abnormal high signal striations within it. **B** , T1 axial image of the ankle. The Achilles tendon has a stippled appearance of low and high signal and is thickened with a convex anterior margin. These findings are from familial hyperlipidemia with xanthoma formation but cannot be distinguished from partial tendon tears by MRI.

Deposits of gout crystals also may affect tendons ( Fig. 3.14 (f0075) ). It is usually impossible to distinguish gouty tophi or xanthomas from partial tendon tears by MRI, and they should simply be kept in mind in the appropriate setting. Calcific tendinopathy from deposition of calcium hydroxyapatite crystals is common and easy to diagnose on radiographs, which is a good thing because MRI does not usually show the abnormality well. The hydroxyapatite crystals have low signal intensity on all pulse sequences that are usually difficult or impossible to distinguish from the low signal intensity tendon. If the crystal deposit is large enough, it may show lower signal than the tendon on all pulse sequences, making it visible on MRI ( Fig. 3.15 (f0080) ).

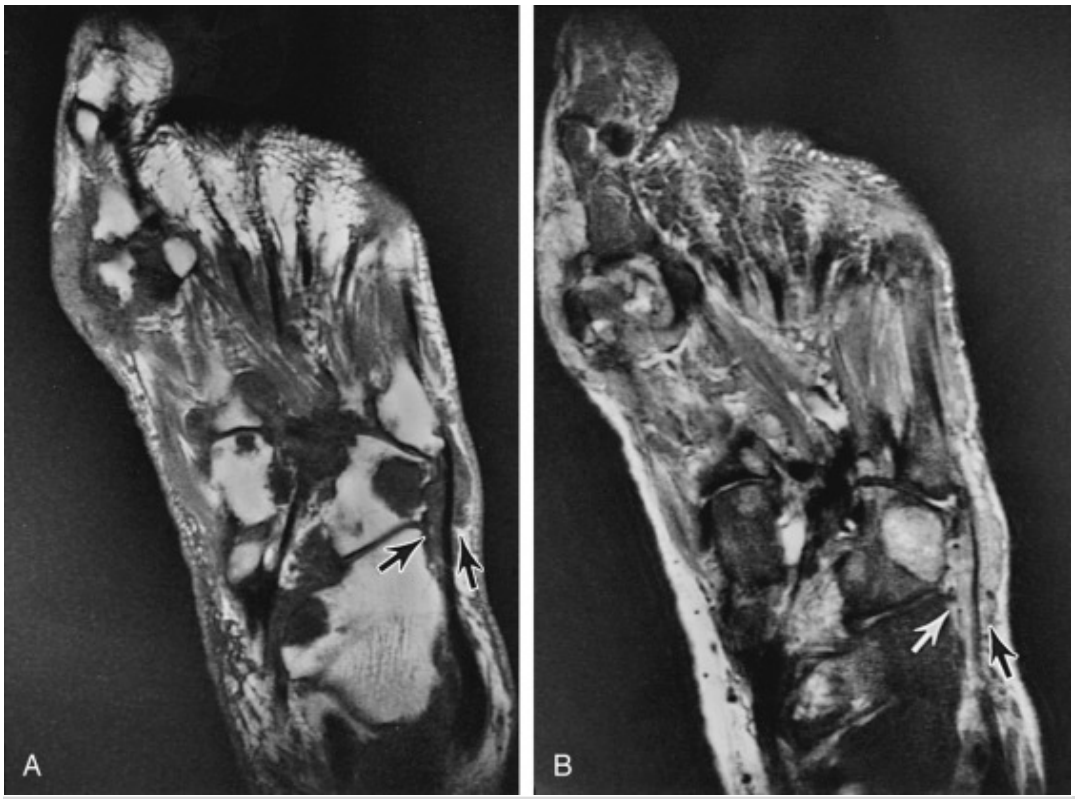



**Fig. 3.14**

**Gout that affects tendons. A** , T1 coronal image of the foot. An intermediate signal lobulated mass ( *arrows* ) representing a gouty tophus surrounds a tendon. Intraosseous gout deposits can be seen in several bones as well. **B** , STIR coronal image of the foot. Findings are the same as in **A** , but the gouty tophi change the signal slightly. They are intermediate signal on this STIR image but may be even lower signal on other types of T2W sequences.

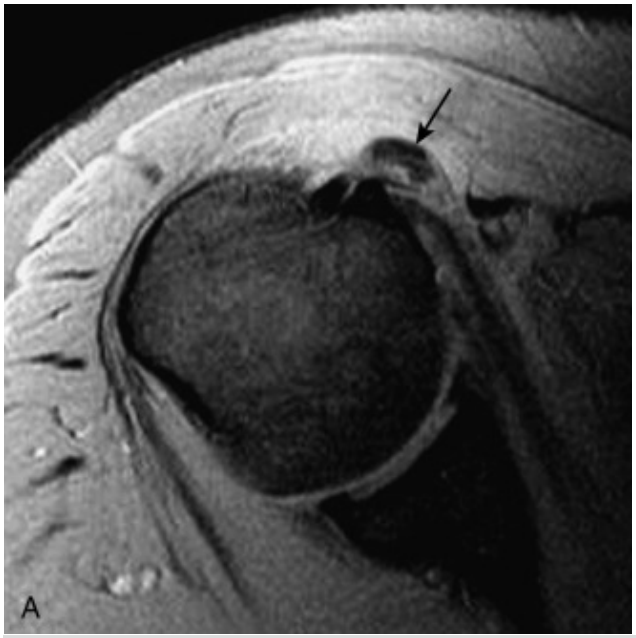
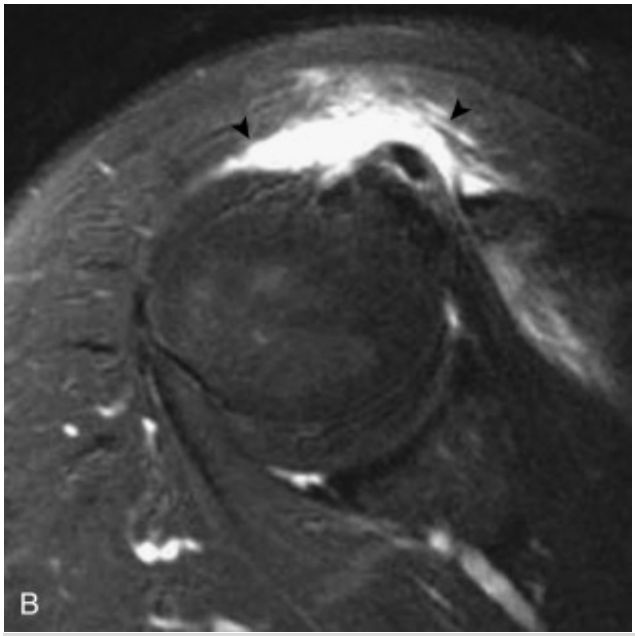


**Fig. 3.15**

**Calcium hydroxyapatite in tendons. A** , T2* axial image of the shoulder. A low signal intensity focus is present within the distal subscapularis tendon ( *arrow* ) from calcium hydroxyapatite crystal deposition (calcific tendinitis). **B** , Fat-saturated T2 axial image of the shoulder. The low signal calcific focus is seen again, and the adjacent inflammatory fluid and edema are better shown ( *arrowheads* ).




Tumors of tendons are exceedingly rare, but clear cell sarcoma (malignant melanoma of soft parts) should be considered if one is considering a tumor at all. Tumors arising from the tendon sheath are much more common than a tumor of the tendon itself. Giant cell tumor of the tendon sheath is a common cause of a mass in the hands and feet. It is a localized and extra-articular form of pigmented villonodular synovitis. It manifests as a nonpainful, soft tissue mass. On MRI, it usually is lobulated with intermediate and low signal on T1W and T2W images due to the presence of hemosiderin and closely apposed to a low signal tendon ( Fig. 3.16 (f0085) ).

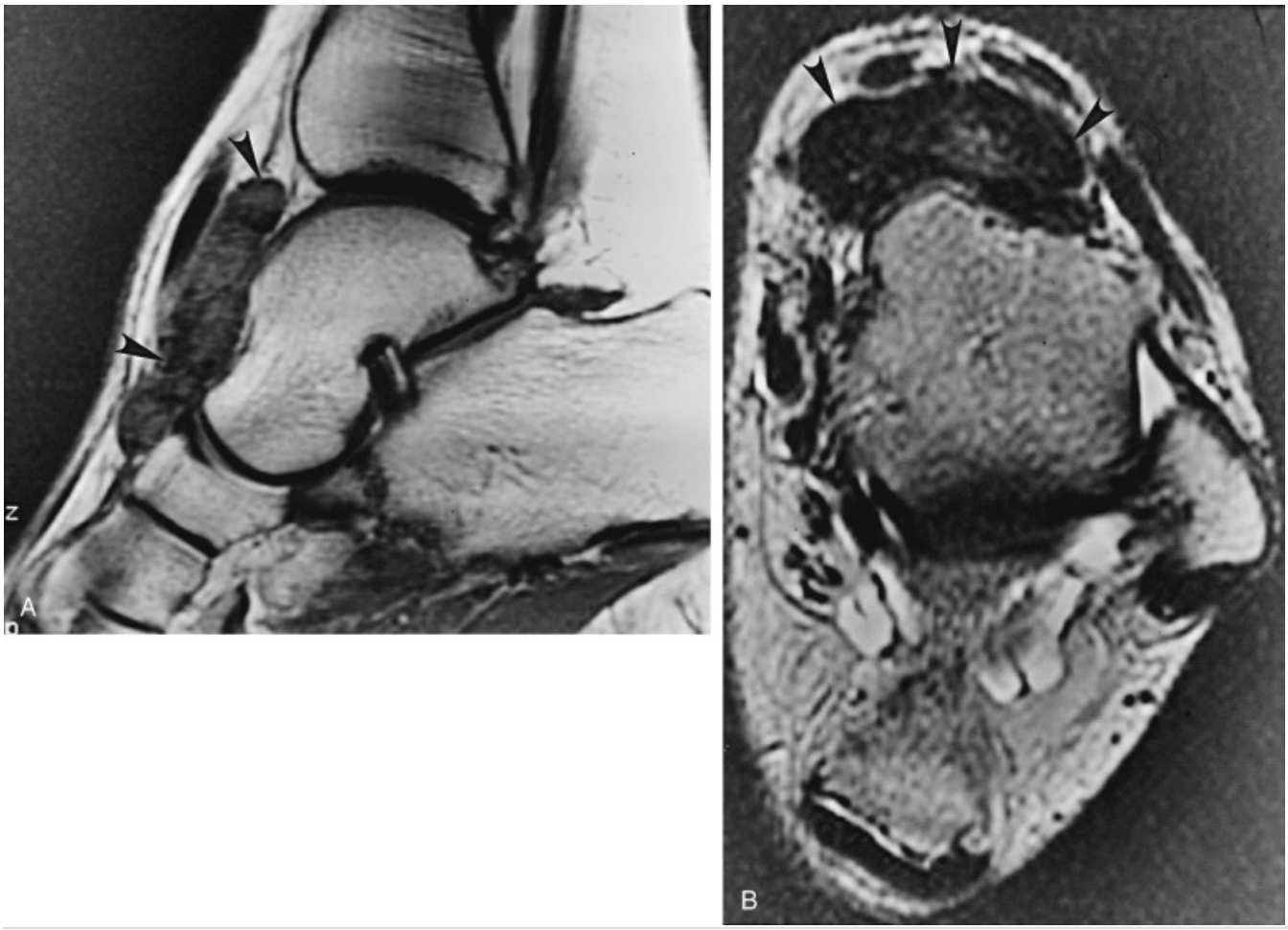



**Fig. 3.16**

**Giant cell tumor of the tendon sheath. A** , T1 sagittal image of the ankle. There is an intermediate signal mass ( *arrowheads* ) anterior to the ankle. **B** , Spin echo–T2 axial image of the ankle. The mass remains intermediate to low signal intensity on this sequence ( *arrowheads* ) and is seen just deep to the extensor tendons.

## How to Image Muscles

- *Coils and patient position:* Injury or other types of pathology often affect large muscles in large body parts where surface coils are not used. The use of coils depends on the size of the region being imaged. Generally, the patient should be imaged in the same position as if the adjacent joint were being imaged.

• *Image orientation:* Generally, most muscles and muscle groups are best evaluated in the axial plane. Longitudinal (coronal or sagittal) planes are used for orientation of abnormalities relative to osseous landmarks and to show the extent of the disease.

• *Pulse sequences and regions of interest:* T1W or some type of T2W imaging is required. For the T2 sequence, we prefer using T2W fat-suppressed or STIR techniques because these sequences are exquisitely sensitive to most muscle pathology. T1 sequences also are necessary to show anatomic detail and configuration of the muscle, subacute hemorrhage, and fatty atrophy of muscle. Fields of view and slice thickness depend entirely on the body part being imaged and the extent of the suspected abnormality. We often do a large field-of-view coronal, fast STIR sequence as a scout view. The edema that is present indicates the region that needs to be covered with a smaller field of view and other pulse sequences and whether a surface coil can be used.

• *Contrast:* Contrast-enhanced sequences are unnecessary, unless one is attempting to identify abscesses or areas of muscle necrosis.

## Normal Muscle

## MRI Appearance

Normal skeletal muscle has intermediate signal intensity on all pulse sequences ( Fig. 3.17 (f0090) ). T1W images show a feathery or marbled appearance because of fat that is interposed between adjacent muscles and between fibers within a muscle. In some

locations, individual muscle groups can be distinguished because of interposed fat. Where intermuscular fat is sparse, such as in the calf, individual muscle groups blend together and are difficult to identify. Low signal intensity tendons may course through a muscle for long distances or may arise near the periphery of a muscle; this myotendinous junction is usually the weakest link in the chain from a perspective of strength of the entire unit. On T2W sequences, normal muscle remains intermediate signal intensity, and if fat suppression is used, no high signal is evident between muscles, with the exception of normal vascular structures.

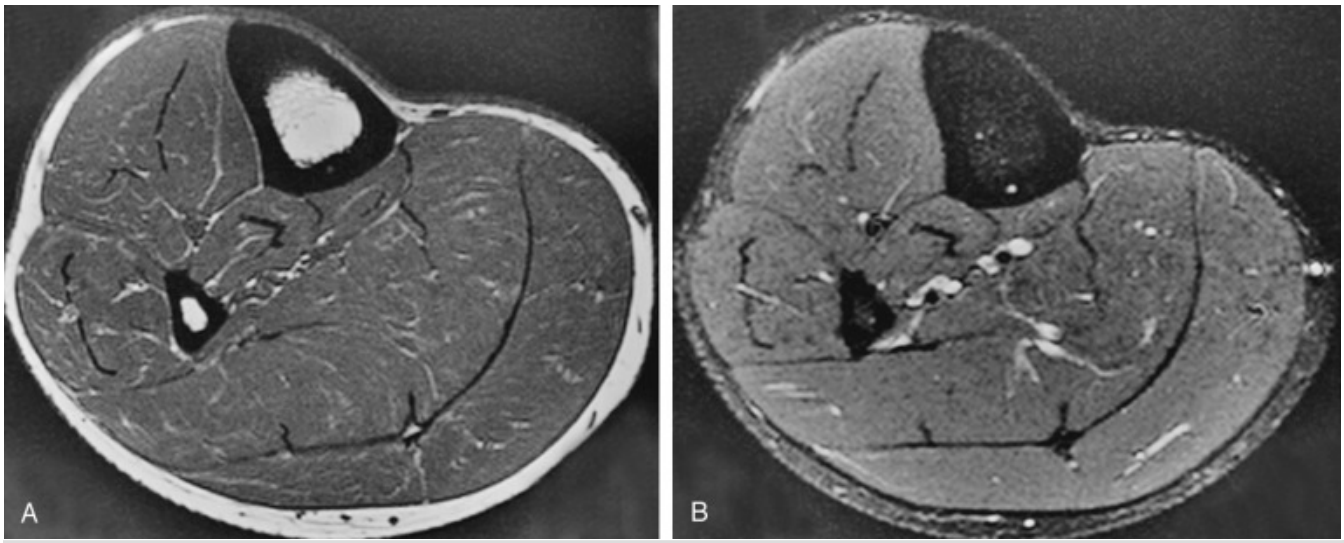



**Fig. 3.17**

**Normal skeletal muscle. A** , T1 axial image of the calf. Muscle has intermediate signal intensity with a feathery appearance created by small amounts of interspersed fat. Little to no intermuscular fat is present in this individual, making identification of specific muscles difficult. **B** , STIR axial image of the calf. Muscle remains intermediate signal on this sequence, but the feathery pattern disappears because of suppression of the fat.

## Muscle Abnormalities

Several abnormalities of muscle can be efficiently evaluated by MRI, including abnormalities from trauma, inflammatory disorders, tumors, denervation, muscular dystrophies, neuromuscular disorders, and ischemia. Despite the wide variety of pathology that may affect it, muscle responds in only a few ways, and this is reflected in the MRI appearances of most of these entities. Generally, *acute* muscle pathology manifests as high signal intensity on *STIR* or fat-saturated *T2W* images (edema, hemorrhage), whereas *chronic* muscle pathology typically results in fatty atrophy that is evident as high signal intensity on *T1W* images. As a result, MRI is exquisitely sensitive to muscle abnormalities but is usually nonspecific. Because of this, it is important to have a list of differential diagnoses available when a muscle abnormality is encountered on MRI (e.g., the abnormalities listed in the outline for this chapter). Clinical findings and biopsy of abnormal tissue play important roles in making a specific diagnosis when abnormalities are detected by MRI, and MRI is useful in directing where a biopsy specimen should be obtained.

Abnormal muscle may be normal, increased, or decreased in size. As mentioned previously, there may be fatty replacement, manifested as high signal intensity in the muscle on T1W images, or there may be areas of high signal intensity on T2W images. A focal mass within muscle is another manifestation of disease.

## Muscle Trauma

Traumatic muscle injuries are extremely common and can be divided into indirect muscle injuries, direct muscle injuries, and miscellaneous muscle injuries. Usually, traumatic injuries are

imaged to assess the exact cause of pain and the extent of the injured area.

## Indirect Muscle Injuries ( **Box 3.6** (b0035) )

Indirect muscle injuries include (1) delayed-onset muscle soreness (DOMS) and (2) strains (muscle tears). When the force of a contracting muscle exceeds the load placed on it, the muscle shortens, and this is called *concentric action* (e.g., the action of the biceps when lifting a weight). Conversely, *eccentric action* is when a muscle lengthens or stretches as it contracts (e.g., the action of the biceps when lowering a weight). Concentric contractions produce fatigue, whereas eccentric contractions are responsible for indirect muscle injuries with muscle strains (partial or complete muscle tears).

**BOX 3.6**

**Indirect Muscle Injuries**

### Delayed-Onset Muscle Soreness (Muscle Stiffness)

- Pain peaks 2 days after unaccustomed activity
- No acute injury or painful event
- Damage is at ultrastructural level and reversible

### Muscle Strains (Partial or Complete Tears)

- Sudden onset of pain occurs during activity
- Occurs during eccentric muscle contraction (i.e., muscle lengthens as it contracts, such as the biceps while lowering a weight)

- Often affects muscles crossing two joints (rectus femoris, biceps femoris, gastrocnemius) and affects myotendinous junction

- Three grades
    - Grade I—few muscle fibers torn, no functional loss, interstitial blood
    - Grade II—more fibers torn, some loss of strength, focal defect and interstitial blood in muscle, blood surrounding tendon from myotendinous junction injury
    - Grade III—muscle completely torn, loss of strength, focal and interstitial blood

## MRI of Muscle Strains

- DOMS and grade I (T2)
    - Feathery, interstitial signal in muscle
    - ± ↑ signal between muscles
- Grade II (T2)
    - Feathery, interstitial ↑ signal in muscle
    - ↑ signal between muscles
    - ± focal muscle defect
    - Tendon thinned, irregular, lax
    - ↑ signal surrounding tendon
- Grade III (T2)
    - Feathery, interstitial ↑ signal in muscle

- Complete muscle disruption with ↑ signal in gap between retracted segments
- ↑ signal between muscle fragments—discontinuity of tendon within muscle

Injuries often occur along the myotendinous junction and are generally sports related. Muscles at highest risk for indirect injuries are muscles that span two joints and are eccentrically activated, such as the hamstrings (biceps femoris, semitendinosis and semimembranosis), gastrocnemius, and rectus femoris muscles in the lower extremity.

## Delayed-Onset Muscle Soreness

DOMS refers to stiff, aching muscles. We all have experienced this problem, which begins hours or days after participating in an unaccustomed exertional activity. Symptoms peak 2 to 3 days after the activity. Serum creatine kinase levels are elevated because of the myonecrosis that is present, and although there is ultrastructural damage to the contractile elements of muscle, no irreversible damage is done. Training prevents or reduces DOMS.

MRI is never done to diagnose DOMS because we all are familiar with what it is and it poses no diagnostic dilemma. We might inadvertently image a patient, however, who happens to have DOMS for unrelated reasons. DOMS is in the differential diagnosis for a certain constellation of MRI findings, and that is the main reason to be aware of it.

T1W images show no abnormality with DOMS. On T2W images, increased signal intensity may be seen around the periphery of muscles or in the perifascial and intermuscular spaces in close proximity to the injured muscle ( Fig. 3.18 (f0095) ). This abnormal rim develops 3 to 5 days after the inciting event. A feathery interstitial pattern of increased signal intensity may also be evident throughout the entire muscle from the extracellular fluid that causes increased intramuscular pressure.

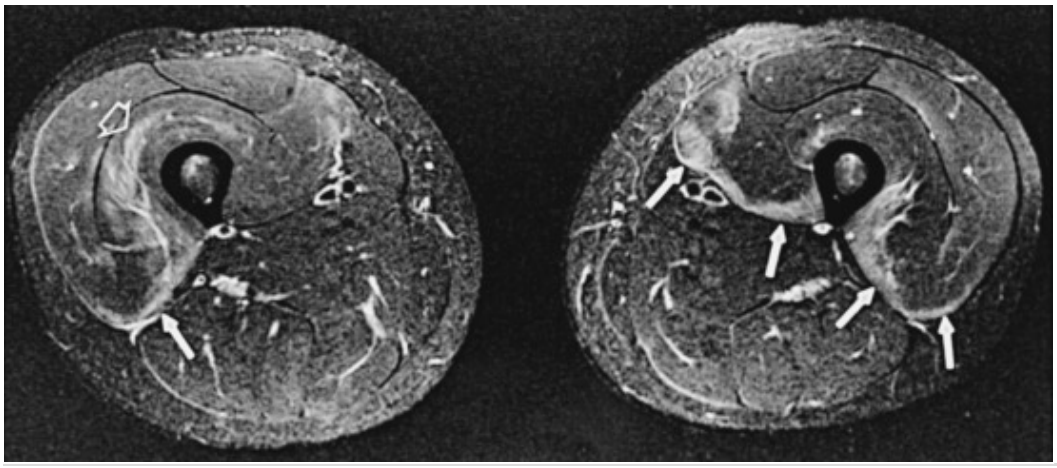


**Fig. 3.18**

**Delayed-onset muscle soreness.** STIR axial image of the thighs. One of our fellows ran a marathon, and these are her thighs less than 2 days later (she said they felt as bad as they look). High signal edema is seen in the periphery of some of the vastus musculature ( *solid arrows* ). More diffuse interstitial muscle edema is seen in the right vastus intermedius ( *open arrow* ). Intermuscular (perifascial) edema was not present at the time of this MRI.

MRI abnormalities persist long after clinical symptoms abate and after creatine kinase levels return to normal; however, the abnormalities on MRI persist for the same period that biopsy-proved ultrastructural damage is identified. Findings may take weeks to resolve on MRI.

## Muscle Strains

Strains are muscle tears, either partial or complete, that result from a sudden event during eccentric muscle contraction. DOMS and certain types of muscle strains can have identical appearances on MRI, but the history serves to distinguish the two entities. Muscle strains occur with an acute onset during activity versus delayed onset of symptoms for DOMS. Muscles can absorb more energy and be protected from muscle strains by warming up and stretching; these activities have no protective effect for DOMS.

Muscle strains are the most frequent injuries in sports. Powerful, eccentric muscle contraction while a muscle is being stretched tears the muscle fibers at and just proximal to the myotendinous junction. With healing, the muscle can regain most of its strength. Until full recovery, the injured muscle is at increased risk for a second injury because of the decreased strength and stiffness. The myotendinous junction is the weakest point because it has less ability to absorb energy than either the muscle or the tendon. At the myotendinous junction, the muscle cells have multiple projections that form intervening recesses, into which collagen fibrils from the tendon insert. This ultrastructural arrangement allows for increased contact area between the muscle and tendon that helps dissipate forces and lessen the risk of injury; however, the myotendinous junction remains the area most susceptible to injury.

One exception to muscle strains manifesting as an acute painful event relates to the rectus femoris muscle in the anterior thigh. Chronic repetitive stresses, usually in runners, may result in a

nonpainful muscle strain that manifests as a palpable mass. Many of these patients come to medical attention for work-up of a tumor mass ( Fig. 3.19 (f0100) ).

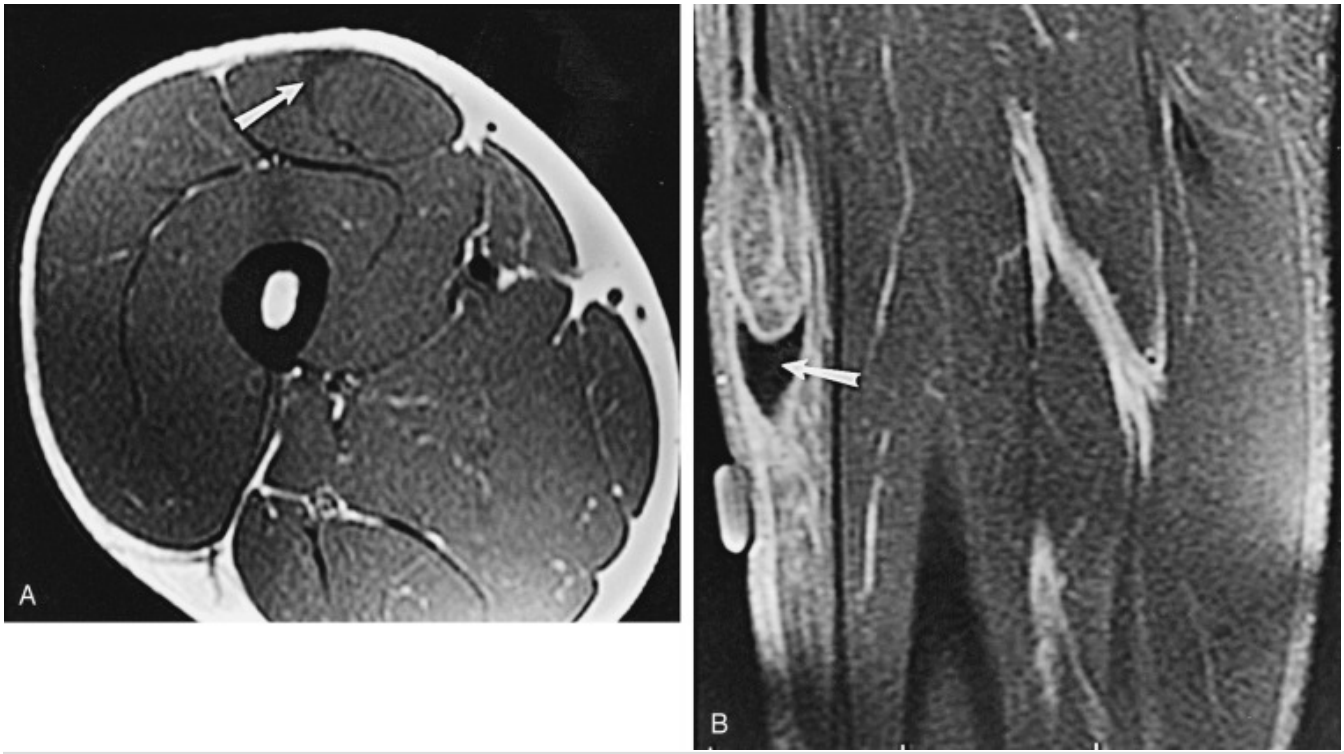



**Fig. 3.19**

**Muscle tear masquerading as a tumor. A** , T1 axial image of the thigh. This patient was referred for work-up of a tumor (painless mass) in the anterior thigh. The rectus femoris muscle is enlarged focally, and there is a defect within it ( *arrow* ) indicating a grade II muscle strain (tear), rather than a tumor mass. **B** , T1 sagittal image of the thigh, with fat suppression after gadolinium enhancement. The gap in the muscle ( *arrow* ) from the tear is seen again. Hyperemia surrounds the torn muscle. No mass is evident. This patient was a runner, which is typically the inciting event for (often painless) tears of this muscle.

Strains are divided into three grades. Grade I and grade II strains can be difficult to distinguish from each other, or from DOMS or a direct blow to muscle, on MRI. A grade I strain consists of tearing

of only a few muscle fibers, with no loss of function or permanent defect in the muscle (essentially, the muscle was stretched). There is edema, which causes enlargement of the muscle, and a feathery, interstitial pattern of increased signal intensity on T2W images, as well as perifascial edema ( Fig. 3.20 (f0105) ). We rarely seem to do MRI for grade I strains, probably because the symptoms are not severe enough to warrant the imaging examination. A grade II strain is a larger partial tear of the muscle with some loss of muscle strength. On MRI, there is the same feathery increased signal intensity from edema and hemorrhage in muscle as in grade I strains; additionally, a focal, masslike lesion or stellate defect may be found in the muscle as the result of a focal disruption of muscle fibers, and perifascial edema or hemorrhage is evident between muscles ( Fig. 3.21 (f0110) ). A grade II strain has a myotendinous junction that is partially torn, so that mild thinning, irregularity, or laxity of the tendon may be evident ( Fig. 3.22 (f0115) ). Hematoma involving the myotendinous junction is diagnostic of a grade II partial tear, indicating a true defect in the muscle tissue.

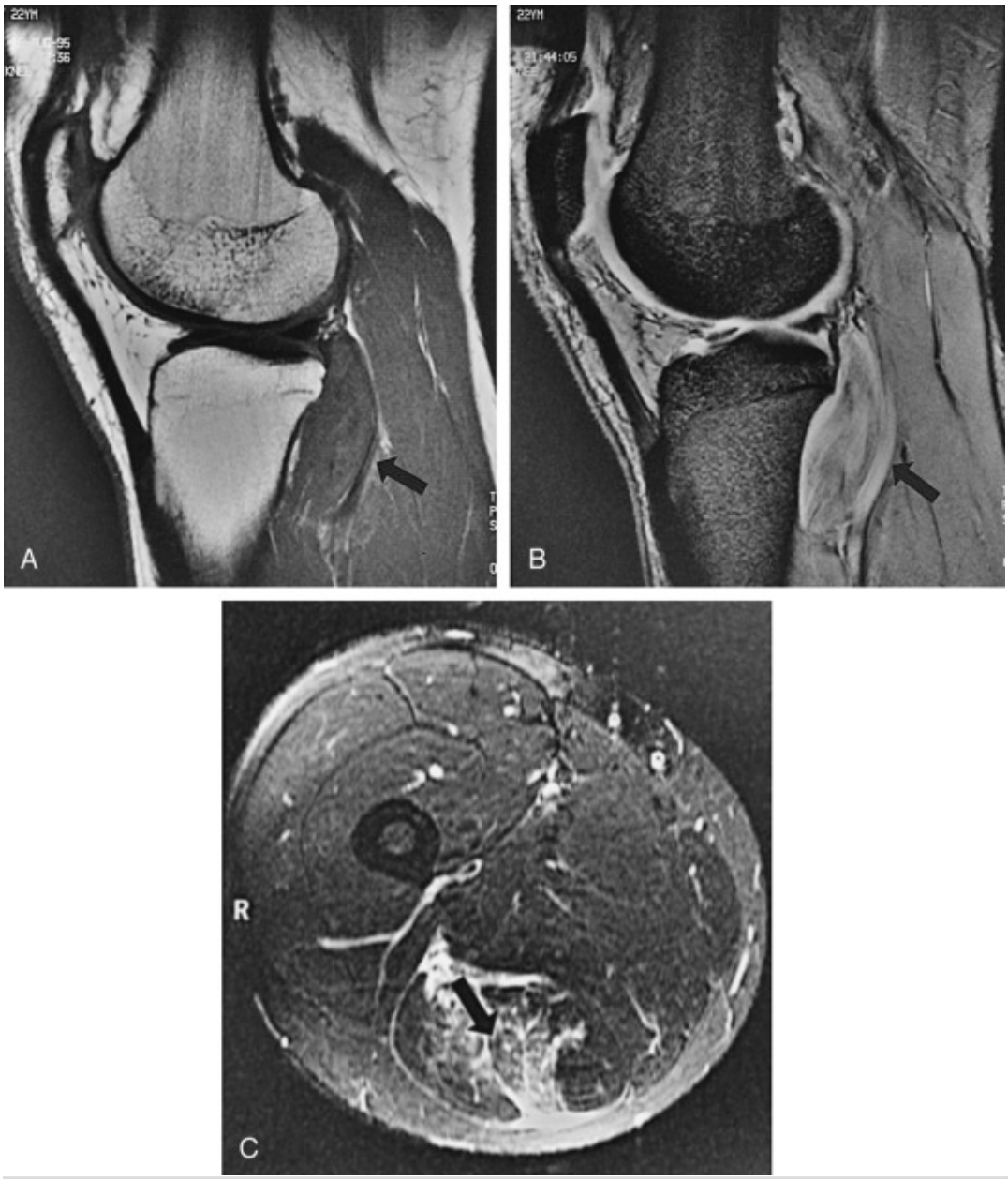



**Fig. 3.20**

**Grade I muscle strains.** **A** , T1 sagittal image of the knee. The popliteus muscle ( *arrow* ) is enlarged, and the feathery fat pattern is absent compared with adjacent muscles. **B** , T2* sagittal image of the knee. High signal within the enlarged popliteus muscle ( *arrow* ) has a feathery pattern from interstitial edema or hemorrhage, and there is perifascial edema anterior and posterior to the muscle. **C** , STIR axial image of the thigh (different patient than in **A** and **B** ). The interstitial feathery pattern from edema or hemorrhage is seen well in this grade I strain of the hamstrings ( *arrow* ). There is also intermuscular (perifascial) edema.

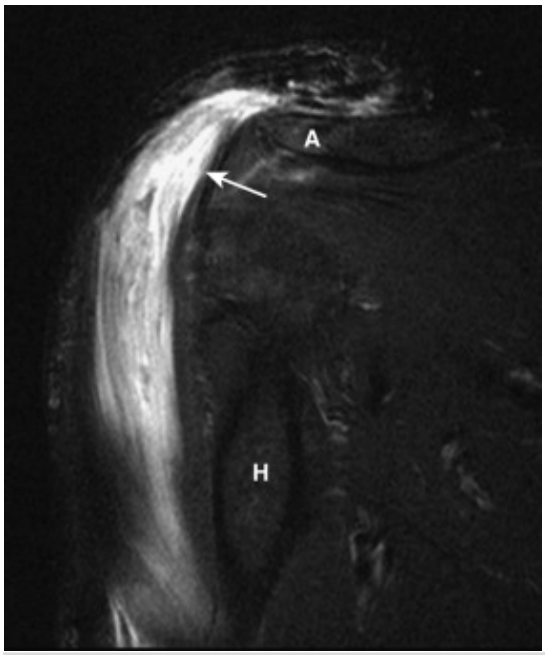



**Fig. 3.21**

**Grade II muscle strain.** STIR oblique coronal image of the shoulder. There is diffuse high signal compatible with edema or hemorrhage, or both, within the deltoid muscle after an injury. There also is an area of focal disruption of the muscle fibers ( *arrow* ) compatible with a grade II strain. *A* , Acromion; *H* , humeral shaft.

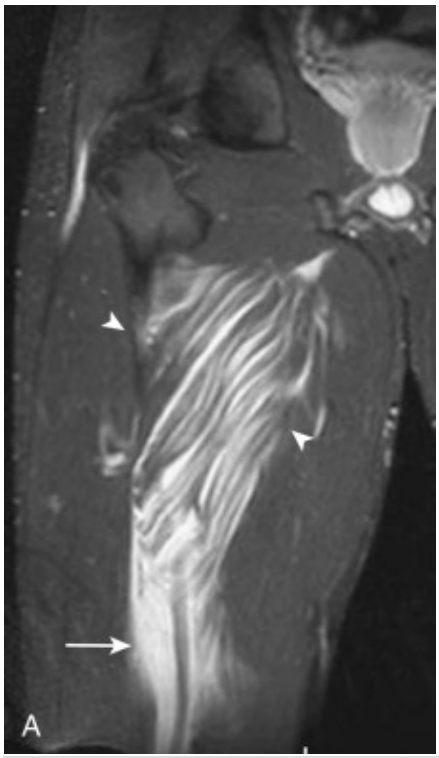
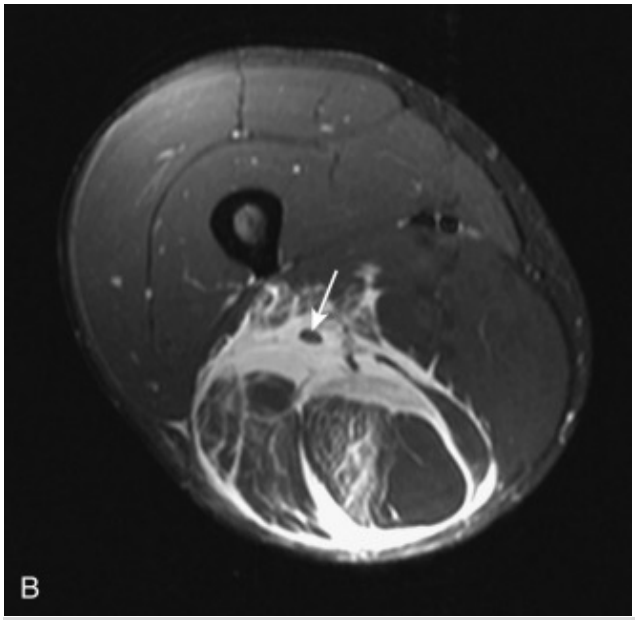


**Fig. 3.22**

**Grade II muscle strain. A** , STIR coronal image of the thigh. High signal intensity infiltrates the hamstring musculature ( *arrowheads* ) in this college football player who felt a “pop” while running. Note the focal disruption of muscle fibers along the myotendinous junction ( *arrow* ). **B** , STIR axial image of the thigh. Intramuscular high signal intensity is seen as extensive fascial fluid, which surrounds the sciatic nerve ( *arrow* ).




A grade III strain is a complete rupture of the muscle with near-complete loss of function (strength). MRI shows discontinuity of muscle and of the tendon traversing the muscle, retraction of fragments with wavy margins, and often a hematoma that forms between the fragments ( Fig. 3.23 (f0120) ).

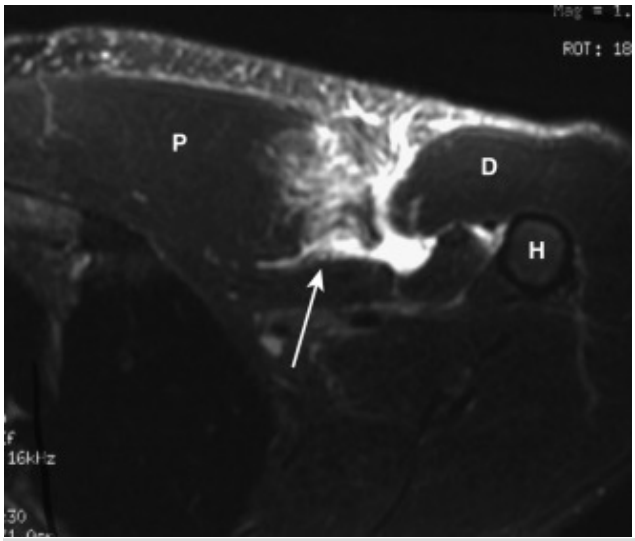



**Fig. 3.23**

**Grade III muscle strain.** STIR axial image of the anterior chest wall. There is complete disruption of the pectoralis major muscle at the point indicated by the *arrow* . High signal hematoma fills the gap. *D* , Deltoid muscle; *H* , humeral shaft; *P* , pectoralis major muscle.

MRI is useful for defining the extent and location of the injury within the muscle, two features that have been correlated with prognosis for recovery. Generally, muscle strains take a long time to heal because it is so difficult to put a muscle at rest for purposes of healing. Persistent contractions of the injured muscle can result in repeated microtears and hemorrhage months after the initial injury. For this reason, it is not unusual to see fatty infiltration from muscle atrophy secondary to disuse or injury, with superimposed blood products of varying age at the site of the muscle strain.

## Direct Muscle Injuries ( Box 3.7 (b0040) )

Direct trauma to muscles can be blunt or penetrating. Blunt trauma results in muscle contusions with intraparenchymal (interstitial) bleeds, hematoma formation, or myositis ossificans as a late sequela. Direct muscle lacerations can occur from penetrating

injuries. Blunt trauma and lacerations produce hemorrhage within the muscle substance at the point of insult ( Fig. 3.24 (f0125) ). Lacerations also may cause denervation with muscle abnormalities distal to the injury.

**BOX 3.7**

**Direct Muscle Injuries**

## Muscle Contusions

- Interstitial hemorrhage
- Intraparenchymal bleeding
- High signal on T2 with muscle fibers coursing through the abnormal area
- No focal defect; muscle may be enlarged

## Hematoma

- Confined, masslike collection of blood
- No muscle fibers coursing through mass
- Focal, heterogeneous mass with muscle enlargement
- Signal of blood on T1 and T2 is age dependent

## Myositis Ossificans

- Intramuscular granulation tissue that may ossify or calcify
- Acute (< 8 wk)
    - Heterogeneous mass
    - T1: isointense to muscle

- T2: mixed high and low signal lesion; surrounding high signal edema

- Chronic (> 8 wk)
  - Variable appearance
  - Fatty marrow—T1 and T2: low signal rim, center isointense with fat
  - T1: diffuse, intermediate signal center
  - T2: slight increased signal in center
  - Low signal rim, all sequences

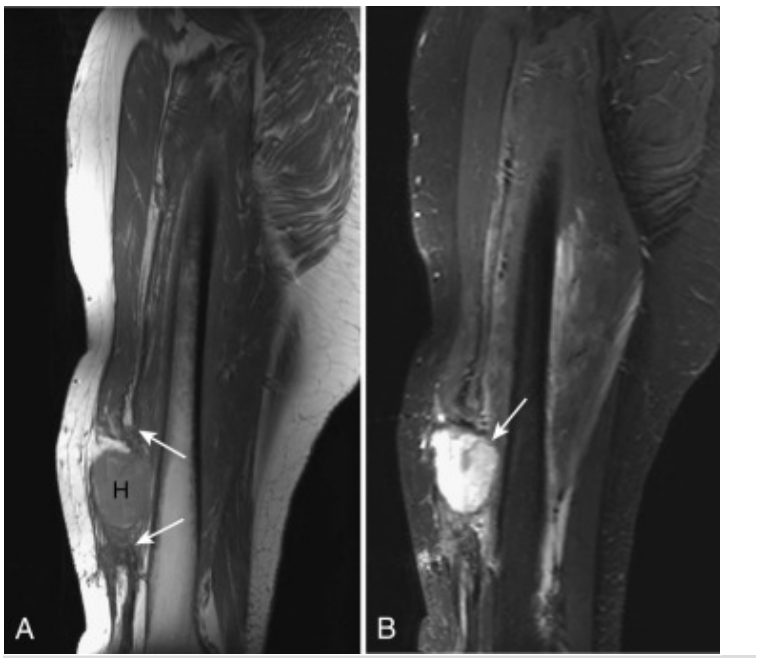



**Fig. 3.24**

**Muscle laceration with hematoma. A** , T1 sagittal image of the thigh. The quadriceps muscles are divided at the site of a prior chainsaw injury ( *arrows* ). The intermediate signal mass at the site of the laceration represents a post-traumatic hematoma (H). **B** , STIR sagittal image of the thigh. The hematoma displays heterogeneous signal, especially in its dependent portion ( *arrow* ).

Acutely, T2W images show increased signal intensity within the muscle as a result of the blood, edema, and inflammation that accompany bleeding from a contusion. This signal intensity may have a feathery pattern within the muscle. Hematomas demonstrate a masslike appearance, and if less than 48 hours old, are usually isointense to muscle on T1W images. Subacute hemorrhage characteristically displays high signal intensity on T1W images.

## Intramuscular (Intraparenchymal or Interstitial) Hemorrhage

When blood dissects freely between muscle fibers, it is referred to as *intraparenchymal, interstitial* , or *intramuscular hemorrhage* . The integrity of the muscle is not violated. Interstitial hemorrhage is caused by direct injury to the muscle with a contusion, usually by a blunt object, rather than from exertional physical activity (indirect muscle injury).

MRI shows focal muscle enlargement, which may be subtle, and separation of fibers by blood, creating a feathery pattern on T2W images ( Fig. 3.25 (f0130) ). T1W images may appear normal or may show the enlarged muscle. No focal collections of blood or edema are evident. Muscle fibers are always evident coursing through the abnormal area in the muscle. This hemorrhage can have an appearance similar to grade I muscle strains, and the history is necessary for reliable differentiation of the two entities.

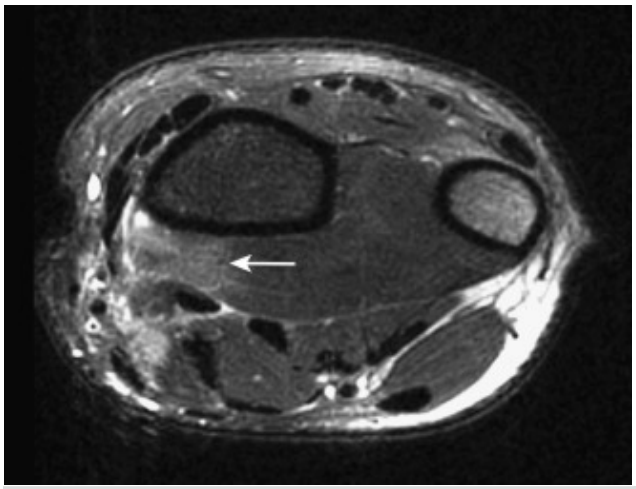


**Fig. 3.25**

**Intramuscular (interstitial) muscle hemorrhage (direct blow).** Axial T2 image with fat saturation. Faint high signal intensity is present in the lateral aspect of the pronator quadratus muscle ( *arrow* ) compatible with interstitial hemorrhage (contusion) at the site of a direct blow from a lacrosse stick.

## Hematoma ( Table 3.1 (t0010) )

Injury to soft tissues can cause subcutaneous or intramuscular hematomas. Hematomas are confined collections of blood that are well defined with a masslike character and no interspersed muscle parenchyma or stroma.

Table 3.1

Progression of Hematoma Signal on MRI

| | Blood Products | T1 Signal | T2 Signal | Mnemonic |
|---|---|---|---|---|
| Hyperacute | Oxyhemoglobin/serum | *I* ntermediate | *B* right | It Be |
| | | | | |
| Acute | Deoxyhemoglobin | *I* | *D* ark | IdDy |

| | Blood Products | T1 Signal Intensity | T2 Signal | Mnemonic |
|---|---|---|---|---|
| Subacute, early | Intracellular methemoglobin | *B* right | *D* ark | BiDdy |
| Subacute, late | Extracellular methemoglobin | *B* right | *B* right | BaBy |
| Chronic | Hemosiderin | *D* ark | *D* ark | Doo Doo |

The appearance of hematomas on MRI is highly variable, age dependent, and follows the same changes as blood in the brain and spinal cord. However, the time course tends to be longer and less predictable because of the lower oxygen tension outside of the brain ( Figs. 3.26 and 3.27 (f0135) ). What is considered an isointense signal relative to the brain or spinal cord can be considered intermediate signal intensity in the extremities, similar to muscle. Hyperacute blood is rarely imaged, but is intermediate signal intensity on T1W (similar to muscle) and high signal intensity on T2W images. Acute blood has intermediate signal intensity on T1W and T2W images. Subacute hematomas show hyperintensity on T1W images, similar to that of fat signal. Early subacute hematomas have low signal intensity on T2W images, whereas older subacute hematomas and chronic hematomas have high signal intensity on T2W images. Chronic hematomas may have low signal intensity, especially around their rims, because of hemosiderin deposition and fibrosis (see Fig. 3.27 (f0140) ).

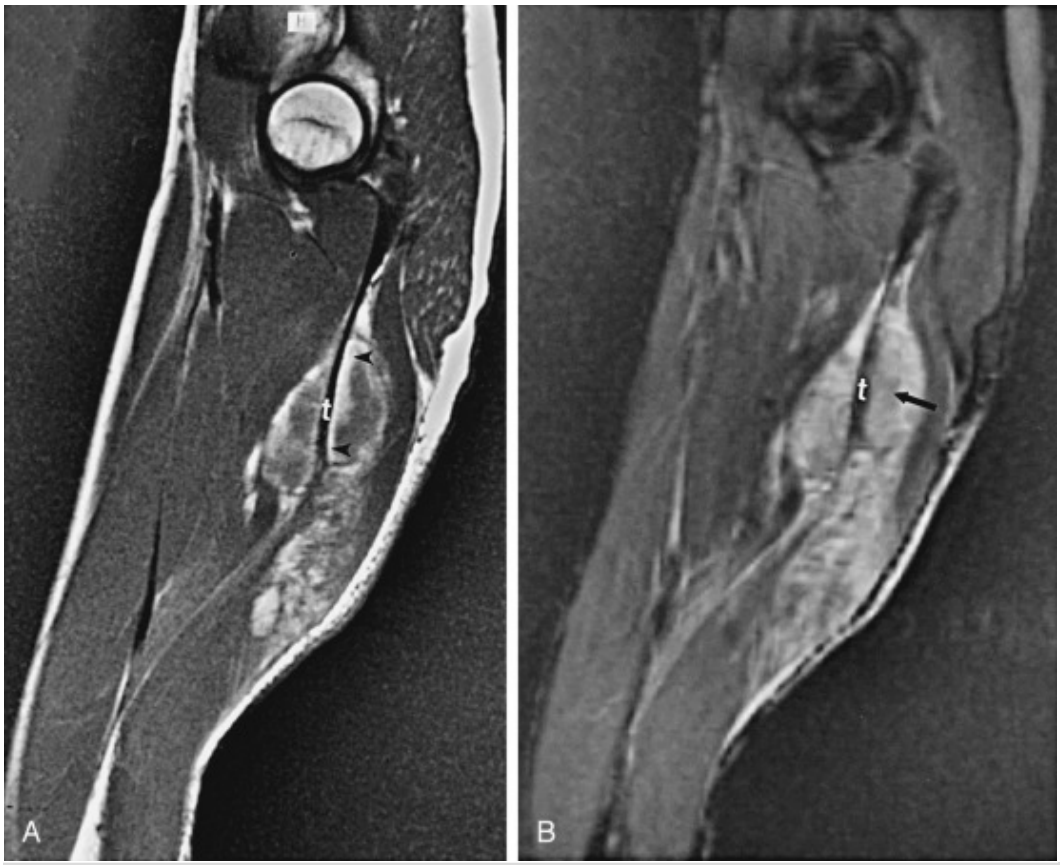



**Fig. 3.26**

**Hematoma with blood at different stages. A** , T1 sagittal image of the thigh. There is a hematoma surrounding the biceps femoris tendon (t) from a large grade II muscle strain. The outer rim of the hematoma ( *arrowheads* ) has high signal from late subacute blood products. The inner portion of the hematoma is intermediate signal. **B** , STIR sagittal image of the thigh. The hematoma around the biceps femoris tendon (t) has a heterogeneous pattern. The areas that were high signal on T1 remain high signal on STIR (late subacute blood). Some of the area that was intermediate signal on T1 became high signal on STIR (hyperacute blood), and other areas were intermediate on T1 and remained low signal on STIR ( *arrow* ) (acute blood).

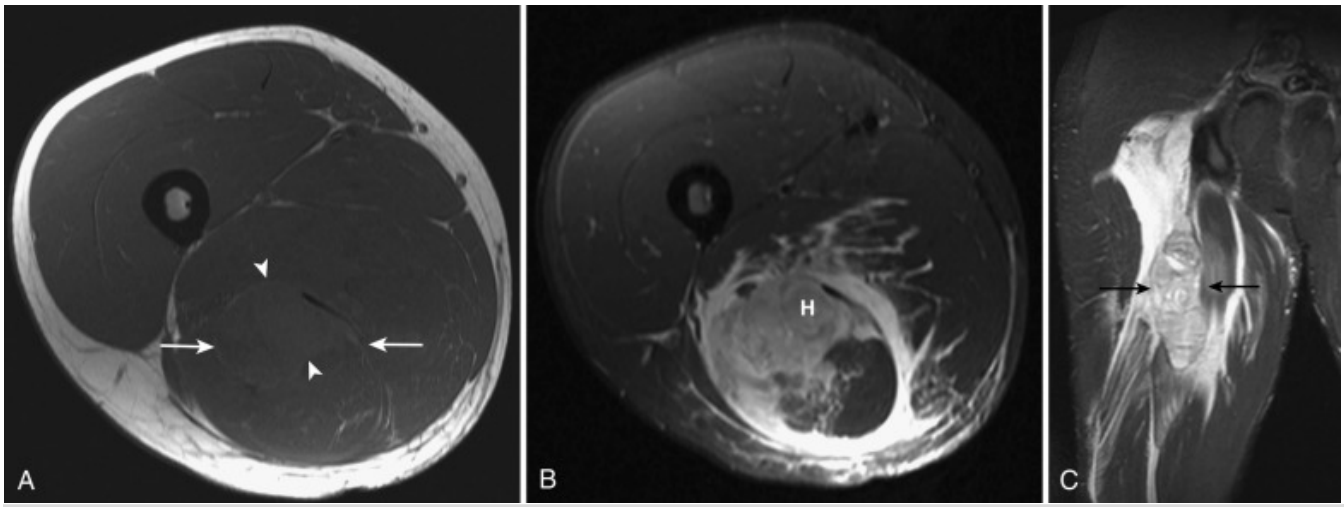



**Fig. 3.27**

**Hematoma with blood at various stages (same patient as Fig. 3.22 (f0115) ). A** , T1 axial image of the thigh. The biceps femoris muscle is enlarged ( *arrows* ) and contains faintly increased signal intensity adjacent to its tendon ( *arrowheads* ). **B** , STIR axial image of the thigh. Masslike tissue replaces the normal muscle architecture at the site of intramuscular hematoma formation (H). **C** , STIR coronal image of the thigh. Note the heterogeneous signal within the hematoma ( *arrows* ) indicating blood products of various ages. The extent of the hematoma is better defined in this imaging plane.

The easiest way we know to remember the progression of signal intensity changes in hematomas that occurs over time is by using a sophisticated mnemonic imported by one of our fellows from Canada (who clearly had nothing better to do on those long, cold northern nights): “ **I** t **B** e **I** d **D** y **B** i **D** dy **B** a **B** y **D** oo **D** oo.” This phrase helps organize the sequence of events: I, intermediate signal; B, bright signal; and D, dark (low) signal; and the bold letters in the phrase refer to the signal intensity on T1W and T2W images in each of the five stages of hematoma progression (hyperacute, acute, early subacute, late subacute, and chronic) (see Table 3.1 (t0010) ).

There often is heterogeneity of the hematoma, probably related to repeated bleeding from recurrent injury, because placing muscles at rest is difficult ( Fig. 3.28 (f0145) ; see Figs. 3.26 and 3.27 (f0135) ). Fat and subacute blood can cause high signal intensity on T1W images in the musculoskeletal system and could have a similar appearance; fat-suppressed images allow differentiation of fat (which suppresses) from blood (which does not).

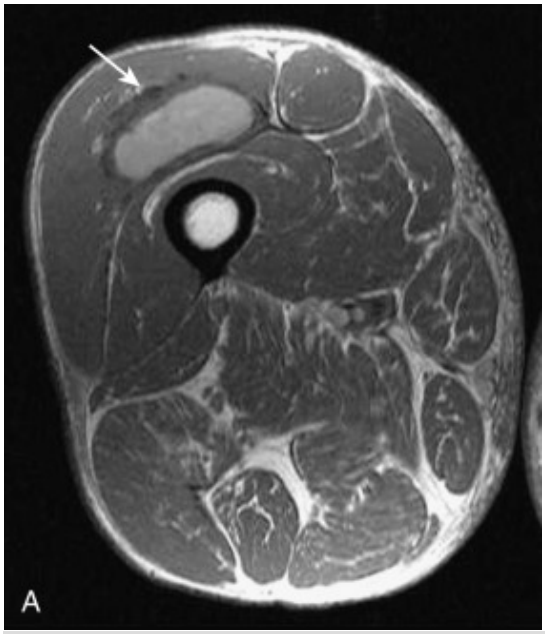
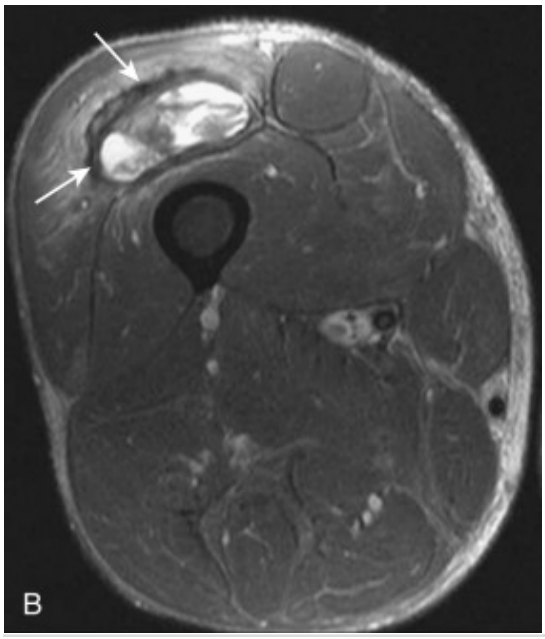



**Fig. 3.28**

**Chronic hematoma.** **A** , T1 axial image of the thigh. Diffusely increased signal is seen within this quadriceps hematoma surrounded by a thick rim containing foci of low signal intensity ( *arrow* ). **B** , STIR axial image of the thigh. The internal blood products show more pronounced heterogeneous signal intensity, and the marginal low signal intensity is more apparent ( *arrows* ), typical of a chronic hematoma with fibrosis and hemosiderin deposition.




In healing, scarring with fibrosis is produced along with muscle regeneration. The amount of fibrosis produced, which can be monitored by MRI examination, predicts whether full tensile strength and functional recovery will occur. Chronic scar tissue and fibrosis have low signal intensity on all MRI pulse sequences. A defect in muscle from a tear may eventually fill in with fat, rather than fibrosis ( Fig. 3.29 (f0150) ).

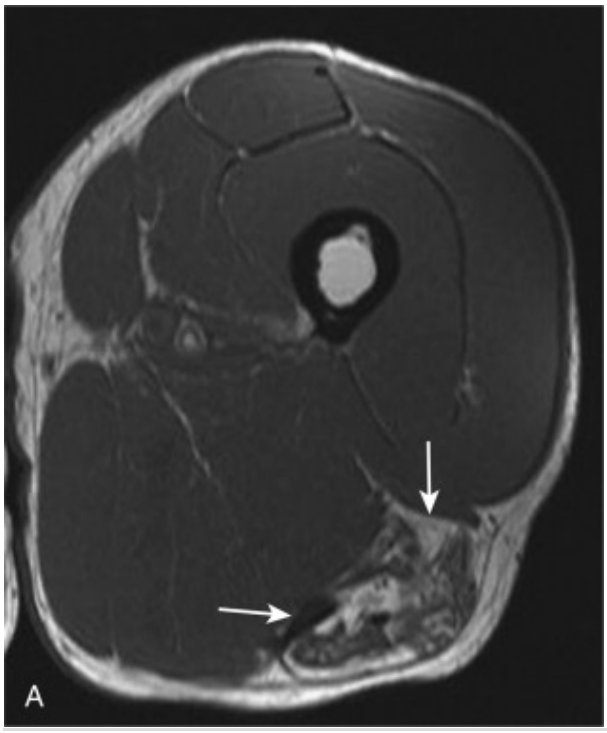
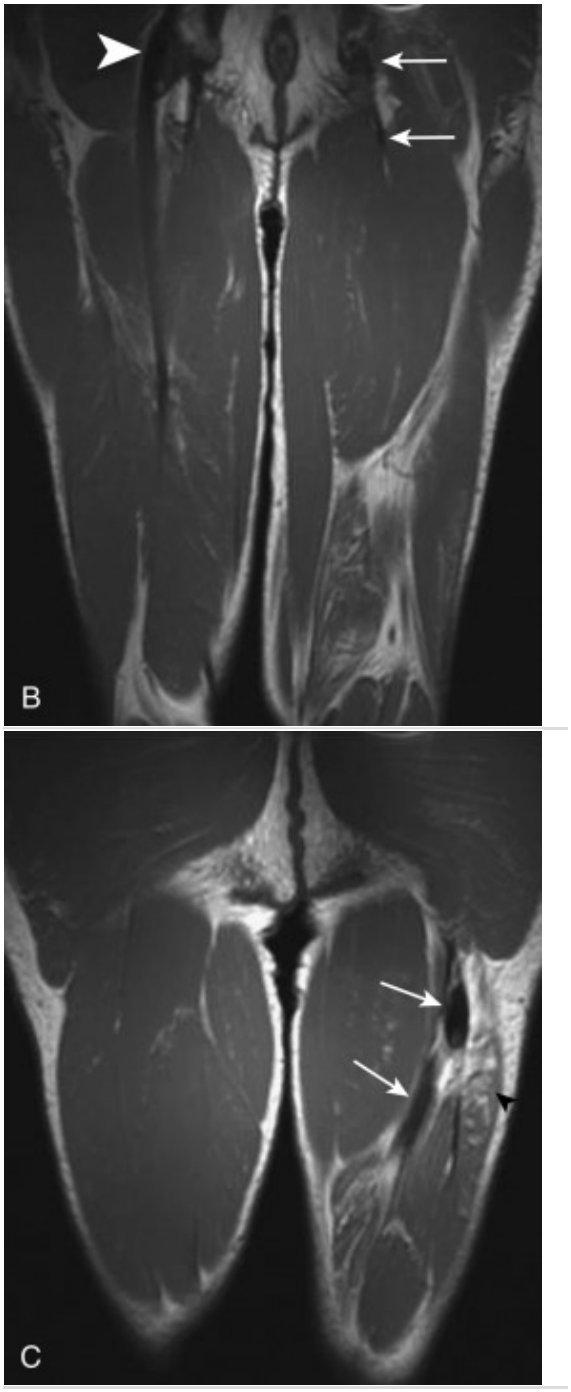


**Fig. 3.29**

**Chronic muscle tear. A** , T1 axial image of the thigh. Focal fatty atrophy of the biceps femoris muscle is evident ( *arrows* ). **B** , T1 coronal image of the thighs. The proximal hamstring tendon is absent ( *arrows* ), compatible with a prior tear. Compare the normal tendon on the right ( *arrowhead* ). **C** , T1 coronal image of the thighs. The thickened, retracted tendon is evident ( *arrows* ) adjacent to the area of muscle atrophy ( *arrowhead* ).




## Hemorrhage Into Tumor

The presence of a hematoma in or between muscles may be the manifestation of bleeding into a necrotic soft tissue tumor, rather than simply being a benign post-traumatic hematoma ( Fig. 3.30 (f0155) ). It can be difficult sometimes to determine whether a hematoma exists within or between muscles because intermuscular masses may significantly displace and thin overlying muscle so that the appearance in both circumstances is very similar. If the history is inconsistent in that the patient reports minimal or no trauma and is not anticoagulated but a large hematoma is present, or if there is any solid-appearing component in the hematoma, one must suspect a soft tissue neoplasm and evaluate it by other means, such as angiography or biopsy. Other helpful signs to differentiate a hematoma from a hemorrhagic neoplasm are that neoplasms are not associated with complete or partial tears of the tendon, and the mass effect of the neoplasm causes displacement of the tendon, rather than abnormal signal surrounding it, with the latter finding being more typical of a traumatic hematoma at the myotendinous junction.

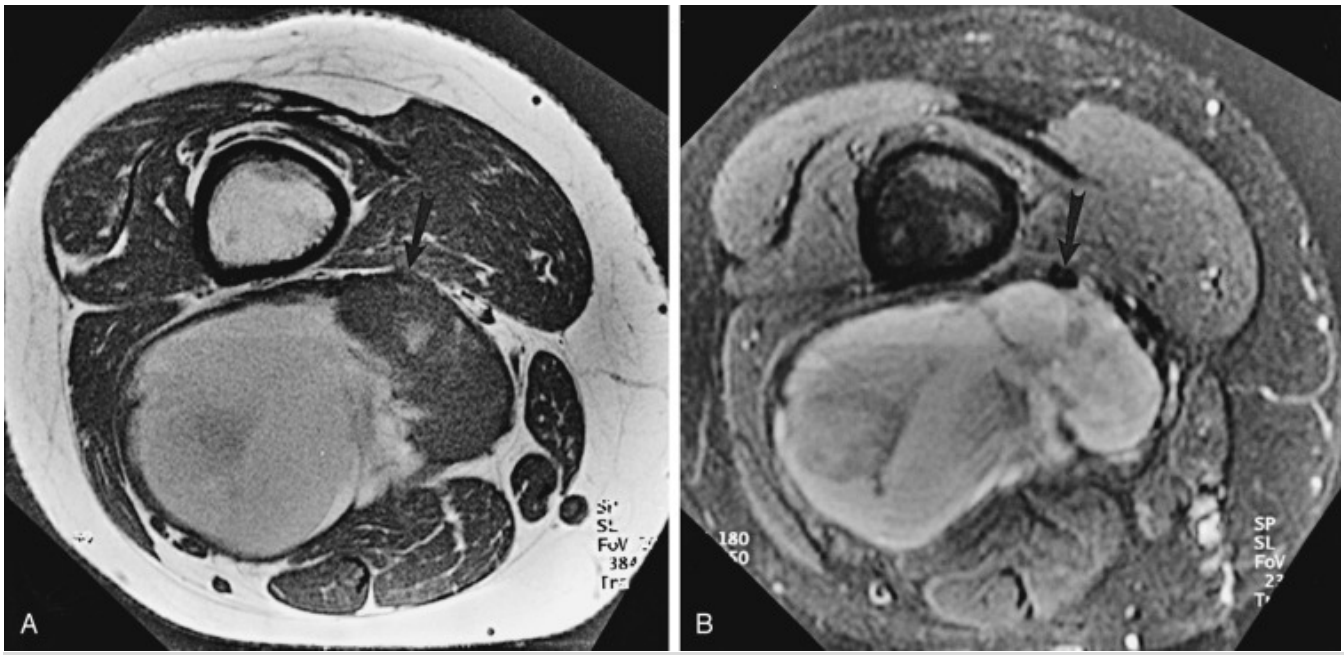


**Fig. 3.30**

**Hemorrhage into a tumor. A** , T1 axial image of the distal thigh. There is a large mass posteriorly with high signal throughout most of it, indicating blood. There is a tendon displaced by the hemorrhagic mass ( *arrow* ). **B** , STIR axial image of the distal thigh. The posterior mass is heterogeneous, but mainly high signal, and consistent with blood. The displaced tendon is seen ( *arrow* ). This initially was diagnosed elsewhere as a hematoma and left alone for 1 year. The patient came to see our orthopedists because the mass did not decrease in size. The fact that the blood does not surround the tendon is a good clue that this is not a traumatic hematoma. The medial portion of this mass (on the side where the tendon is displaced) is actually solid, and a synovial sarcoma was shown at biopsy.

## Myositis Ossificans

Blunt trauma to muscle can cause myositis ossificans, which is a circumscribed mass of granulation tissue that may calcify or ossify with time. If the trauma is not recalled and no calcification is seen on radiographs, this entity is generally not thought of, and patients undergo a work-up for a soft tissue mass.

The MRI appearance depends on the histology and the stage of evolution of the lesion ( Fig. 3.31 (f0160) ). Generally, the appearance varies and is nonspecific, and it can easily be confused with a soft tissue tumor, especially early on. Acute lesions less than 2 months old are isointense to muscle on T1W images and mixed signal (mainly high signal) intensity on T2W images, especially centrally, because of proliferating cellular fibroblasts and myofibroblasts. There may be a large area of surrounding edema.

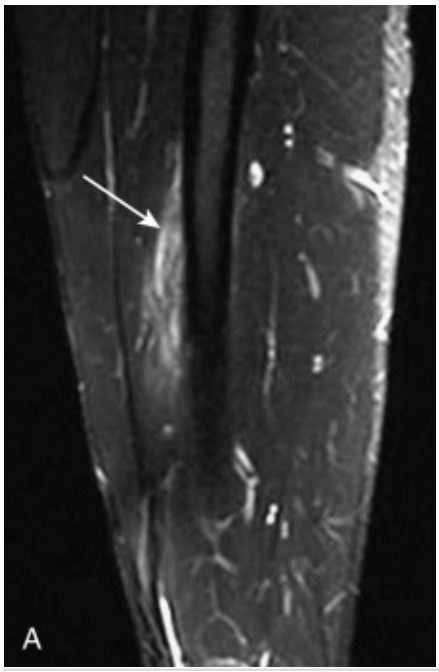
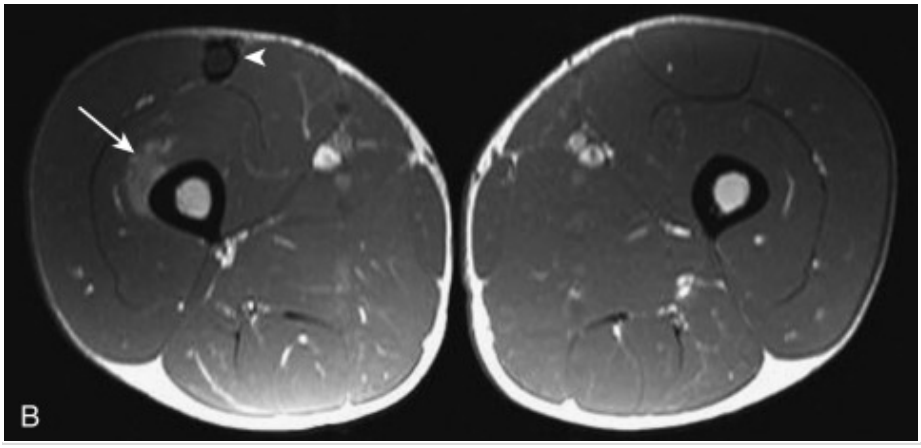
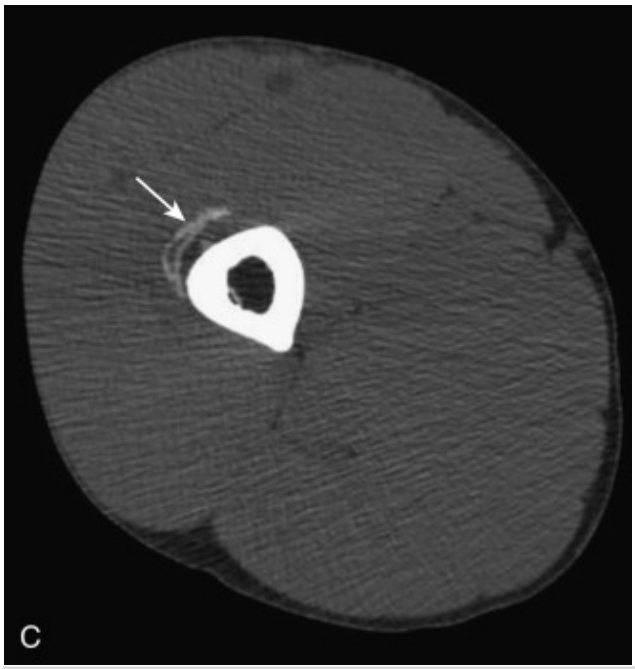



**Fig. 3.31**

**Myositis ossificans. A** , T2 fat-saturated sagittal image of the thigh. This college basketball player presented with thigh pain. There is diffusely increased signal within the vastus intermedius muscle ( *arrow* ). **B** , T1 axial image after IV gadolinium administration. There is diffuse enhancement within that portion of the muscle ( *arrow* ). **C** , Axial computed tomography scan of the thigh (obtained subsequently). Mature shell-like ossification is present at the site of the MRI abnormality ( *arrow* ) compatible with a diagnosis of myositis ossificans.






Lesions of myositis ossificans more than 8 weeks old may have two different patterns on MRI: (1) central signal intensity that is isointense with fat on T1W and T2W images, representing bone marrow surrounded by a low signal intensity rim of lamellar bone, or (2) diffuse intermediate signal intensity on T1W images from fibrosis that is slightly high signal intensity on T2W images. Edema generally is not present surrounding the mass after the first few weeks postinjury. Radiographs and computed tomography can be helpful in making the specific diagnosis of myositis ossificans if it is unclear on MRI.

## Miscellaneous Traumatic Injuries

Compartment syndromes and fascial herniation of muscle are considered here. Denervation of muscle and rhabdomyolysis may occur from trauma, but are discussed elsewhere.

## Compartment Syndromes ( **Box 3.8** (b0045) )

Acute traumatic compartment syndrome can affect an extremity after a fracture. Compartment syndrome occurs when hemorrhage or edema within closed fascial boundaries leads to increased pressure with compromise of the circulation. MRI can show the extent of edema and rhabdomyolysis because necrotic muscle is much higher signal intensity than normal muscle on T2W images. This is a surgical emergency, and MRI generally does not play a role in the work-up of this entity.

**BOX 3.8**

**Compartment Syndromes**

## Traumatic

## Acute

- Fracture, hemorrhage, edema, usually in calf
- ↑ pressure, ↓ circulation
- Muscle necrosis—T2: increased signal diffusely in and between muscle of affected compartment

## Chronic

- Muscles atrophied, fibrotic, or necrotic with calcified rim (calcific myonecrosis)

## Exertional

- Exercise normally causes extracellular, intramuscular fluid that disappears 10 minutes after cessation of exercise
- If the intramuscular edema persists > 15-25 minutes after exercise, acute or chronic exertional compartment

syndromes may occur from the increased pressure

• T2: increased signal in and between muscles, 25 minutes after provocative exercise

In *chronic compartment syndrome,* muscles are atrophied and may be densely fibrotic. Calcification of the involved muscle compartment may exist, and this is especially common in the peroneal compartment. A chronic compartment syndrome may rarely lead to calcific myonecrosis. There is typically a remote history of trauma, usually several decades before the development of calcific myonecrosis. Patients have a painless mass, usually in the calf. The mass consists of liquefied necrotic muscle surrounded by a thin shell of calcification ( Fig. 3.32 (f0165) ). Peripheral peroneal nerve damage is commonly associated with this condition. MRI is useful for showing the anatomic extent of the abnormalities and the extent of muscle loss that exists.

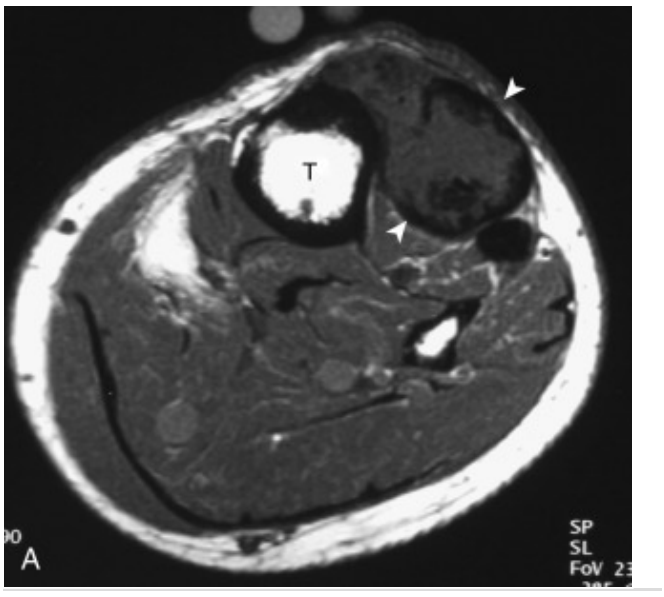
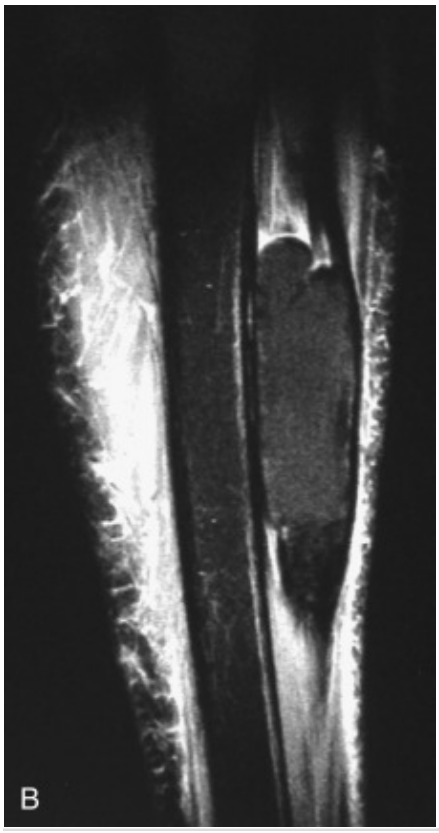
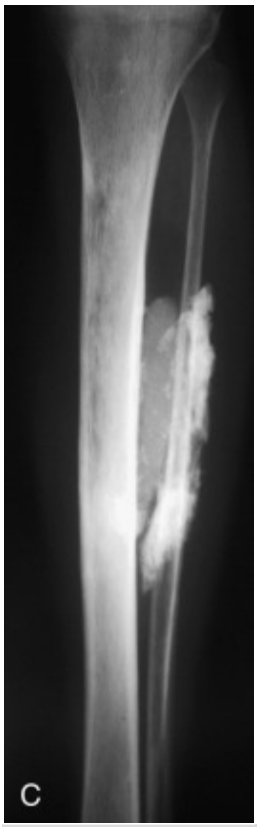


**Fig. 3.32**

**Calcific myonecrosis from chronic compartment syndrome. A** , T1 axial image of the lower leg. The patient presented with a palpable mass. There is enlargement of the anterior compartment muscles with a masslike area of intermediate signal intensity bordered by a thick, low signal intensity rim ( *arrowheads* ). *T* , Tibia. **B** , STIR coronal image of the lower leg. The mass shows similar signal characteristic along with diffuse edema within the soft tissues of the calf. **C** , Anteroposterior radiograph of the lower leg. The mass is shown to contain extensive, mature calcification compatible with calcific myonecrosis.






*Exertional compartment syndrome* occurs as a result of intense activity. Extracellular free water increases in the affected muscles, particularly in concentrically exercised muscles. This increased intramuscular fluid leads to increased pressure in the anatomic compartment that contains the muscles. MRI can show normal, exercise-induced changes within muscle, which cause no abnormality on T1W images, but increased signal intensity on T2W images immediately after exercise. The signal intensity returns to baseline by 10 minutes after cessation of exercise. The increased signal intensity is seen diffusely throughout the affected muscles.

Intramuscular pressure increases more than normal in some patients after exercise and does not quickly return to baseline. Such changes may lead to an acute or chronic exertional compartment syndrome (CECS), requiring fasciotomy for cure. CECSs are difficult to diagnose because symptoms abate between episodes of exertion. Intramuscular wick pressure measurements before and after

exercise may be useful for diagnostic purposes, but it is an invasive test, and pressure criteria for the diagnosis are not universally accepted.

On MRI, exertional compartment syndromes are characterized by swelling within a compartment, which manifests as intramuscular diffuse high signal intensity on T2W images ( Fig. 3.33 (f0170) ). Performing the MRI examination immediately after a provocative exercise may be necessary to identify the changes and support the diagnosis. Failure of the edematous muscles to return to a baseline normal appearance by 15 to 25 minutes after the completion of exercise is diagnostic.

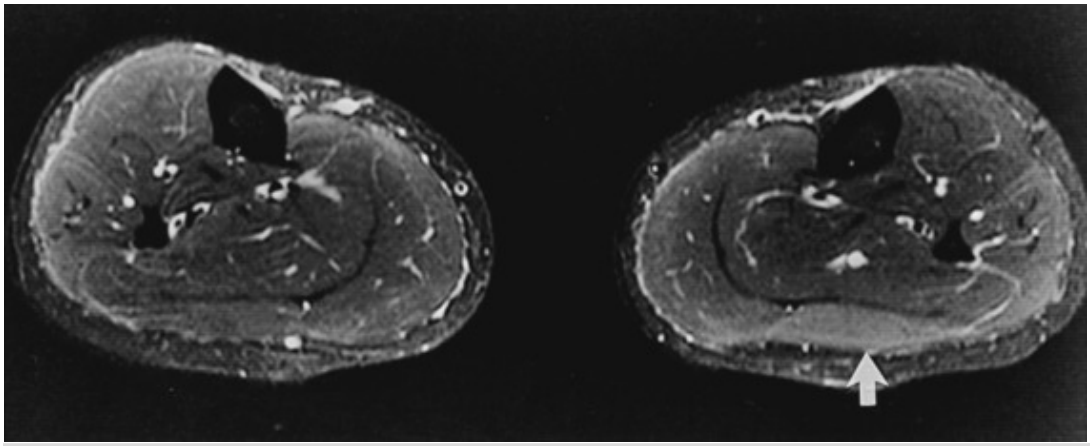


**Fig. 3.33**

**Exertional compartment syndrome.** STIR axial image of the calves. There is diffuse intramuscular high signal in the lateral head of the left gastrocnemius muscle 30 minutes after provocative exercise ( *arrow* ). The T1 images were normal, as were the STIR images obtained before exercise (not shown). Postexercise muscle edema normally disappears within about 10 minutes of the activity.

## Fascial Herniation of Muscle

Muscle can herniate through a traumatic fascial defect and manifest clinically as a soft tissue mass ( Fig. 3.34 (f0175) ). The lesions are usually asymptomatic, although pain or cramping may occur with activity. It sometimes is difficult to show the mass during the MRI examination without contracting the muscle by placing the foot in dorsal and plantar flexion, but this may lead to motion and degraded images. Very short pulse sequences during muscle contraction may show muscle tissue protruding in the region of the patient's palpable mass. The mass may disappear on images obtained without muscle contraction. In these cases, ultrasound is a useful adjunct, given its ability to provide dynamic information.

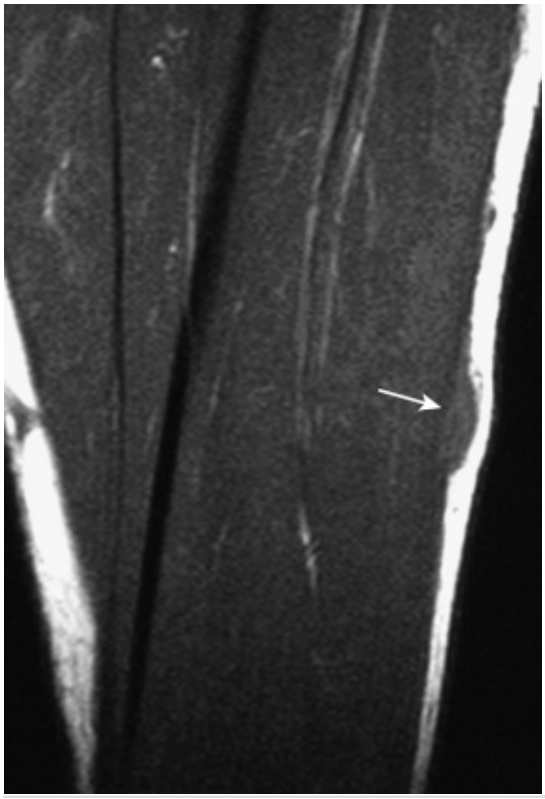


**Fig. 3.34**

**Muscle herniation.** T1 coronal image of the calf. There is a soft tissue mass ( *arrow* ) protruding into the subcutaneous fat at the site of a painless, palpable abnormality. The signal intensity followed that of muscle on all pulse sequences, and this represents herniation of the peroneus longus muscle through fascia.

Even if the herniated muscle is not shown on the study, MRI can suggest the diagnosis by showing normal muscles with no mass. In other cases, the muscle remains herniated at all times and is not significantly affected by muscle contraction; the diagnosis is easier to make in this circumstance.

Occasionally, MRI can reveal a defect in the low signal intensity fascia through which the muscle herniates. This is a traumatic injury and most commonly is seen in the anterior lower leg or thigh in athletes, may affect multiple sites, and usually is asymptomatic.

## Inflammatory Myopathies

Bacterial or viral pyomyositis, necrotizing fasciitis, sarcoidosis, and the autoimmune idiopathic inflammatory polymyopathies are all unusual diseases that may affect muscle. MRI has proved to be useful for evaluating these conditions.

## Pyomyositis ( Box 3.9 (b0050) )

Infection of muscle may be introduced either during a penetrating injury or by hematogenous spread and must be considered as a cause for abnormal size and signal intensity in musculature by MRI.

**BOX 3.9**

## Infection

### Pyomyositis

- Penetrating injury or hematogenous
- Rare; usually in immunocompromised host
- Nonspecific MRI with high signal on T2 images in and around muscle

### Necrotizing Fasciitis

- Infection of intermuscular fascia, pyomyositis rarely associated
- Severe systemic toxicity, high mortality
- Difficult clinical distinction from cellulitis, which affects only subcutaneous fat
- MRI
  - T2: increased signal between muscles; possible increased signal in muscles (from hyperemia)
  - Easy distinction from cellulitis that shows strandlike abnormal signal (low on T1, high on T2) limited to subcutaneous fat

Bacterial myositis is unusual except in immunocompromised hosts, such as transplant patients and patients with AIDS.

Pyomyositis generally occurs where there was blunt trauma, with a coexisting source in the body for bacteremia, usually in someone who is immunocompromised. There is diffuse muscle involvement,

sometimes with a focal abscess ( Fig. 3.35 (f0180) ). Nothing specific is seen on MRI in pyomyositis. There is increased signal intensity throughout the affected muscle on T2W images. The muscle often is enlarged, and there may be high signal intensity fluid in fascial planes surrounding the abnormal muscle.

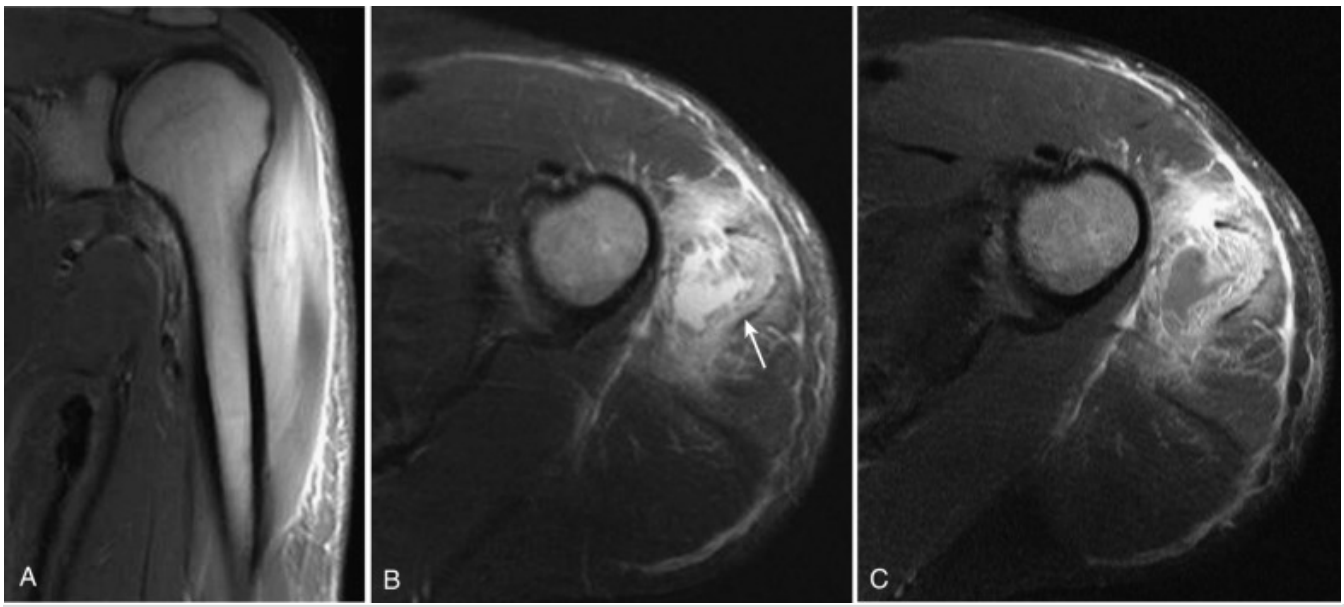



**Fig. 3.35**

**Pyomyositis. A** , STIR coronal image of the shoulder. There is diffuse edema within the deltoid muscle of this immunocompromised patient. **B** , STIR axial image of the shoulder. A thick-walled focal fluid collection is present within the muscle, compatible with an intramuscular abscess ( *arrow* ). **C** , T1 axial image with fat saturation after IV gadolinium administration. There is a lack of central enhancement within the abscess with prominent enhancement of its irregular wall and adjacent soft tissues.

Muscle abscesses are fluid-filled cavities that are bright on T2W images, with a thick rim ( Box 3.10 (b0055) ). The center does not enhance with intravenous (IV) contrast material, whereas the rim

typically does. Such a pattern is typical of an abscess, but it may be seen in other entities, including ischemic foci in muscle and necrotic soft tissue tumors.

**BOX 3.10**

**Rim Enhancement of Focal Muscle Lesion**

### MRI

- Low signal on T1
- High signal on T2
- Rim enhancement postcontrast

### Differential Diagnosis

- Abscess
- Necrotic tumor
- Ischemic foci (diabetics)

## Necrotizing Fasciitis

Necrotizing fasciitis is a rare, rapidly progressive infection characterized by extensive necrosis of subcutaneous tissue and the fascia between muscles, and is usually accompanied by severe systemic toxicity (see Box 3.9 (b0050) ). The mortality rate is greater than 70% if not recognized and appropriately treated.

Early clinical recognition of necrotizing fasciitis may be difficult, and the differentiation between this entity and cellulitis may be impossible on clinical grounds. Cellulitis involves infection of only

subcutaneous fatty tissue and can be treated adequately with antibiotics in most cases. Necrotizing fasciitis requires early surgical intervention in addition to antibiotics. Early fasciotomy and débridement in necrotizing fasciitis have been associated with improved survival compared with delayed surgical exploration.

Before MRI, the only way to diagnose necrotizing fasciitis was at the time of surgery, when no resistance to probing was discovered in the fascial planes between muscles. MRI is able to make the differentiation between cellulitis and necrotizing fasciitis in a noninvasive manner and constitutes a legitimate reason for an emergency MRI examination ( Figs. 3.36 and 3.37 (f0185) ).

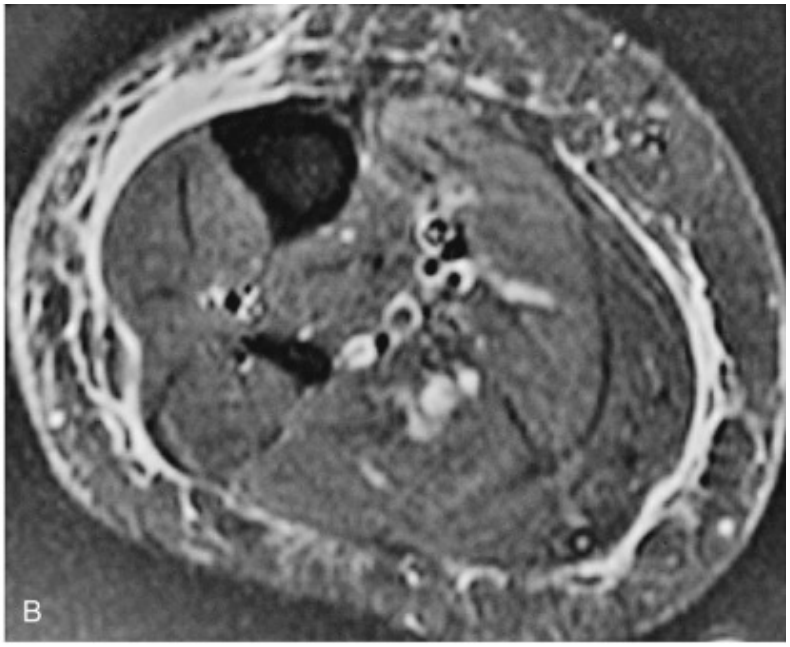



**Fig. 3.36**

**Cellulitis. A** , T1 coronal image of the calf. There is a low signal reticular pattern throughout the subcutaneous fat from edema as the result of cellulitis. **B** , STIR axial image of the calf. The reticular edema pattern in the subcutaneous fat becomes high signal on this sequence. Of key importance is that there is no high signal in or between muscles.

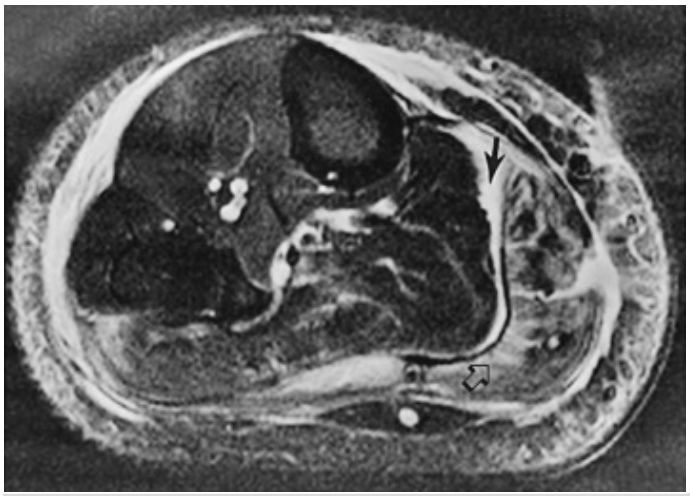


**Fig. 3.37**

**Necrotizing fasciitis.** STIR axial image of the calf. There is edema in the subcutaneous fat. There is also high signal between muscles ( *solid arrow* ) in deep fascial planes and diffuse interstitial edema in the gastrocnemius muscle ( *open arrow* ) from hyperemia and edema as a result of the adjacent fascial infection.

The MRI appearance in necrotizing fasciitis is that of high signal intensity and thickening between muscles along deep fascial sheaths on T2W images. The adjacent muscles also may have high signal intensity secondary to hyperemia and edema from the adjacent inflammatory process. Contrast-enhanced images show high signal intensity on T1W images in fascial planes because of the hyperemia associated with the infection; occasionally, muscle abscesses or focal necrotic tissue with ring enhancement can also be

seen. Generally, there is no need to do contrast-enhanced imaging for this diagnosis, especially because muscle pyomyositis is rarely associated with this disease.

MRI has a high sensitivity for detecting necrotizing fasciitis and showing its extent, but the findings are nonspecific. This lack of specificity is not a real problem, however, because the clinical setting in conjunction with the typical MRI appearance allows the diagnosis to be made, even though the MRI findings are seen in other entities.

## Idiopathic Inflammatory Polymyopathies

The common diseases among the idiopathic inflammatory polymyopathies are polymyositis and dermatomyositis. Active myositis shows increased signal intensity on T2W images, which is more conspicuous with fat-suppression techniques ( Fig. 3.38 (f0195) ). Burned-out disease shows fatty replacement of muscles that is high signal intensity on T1W images.

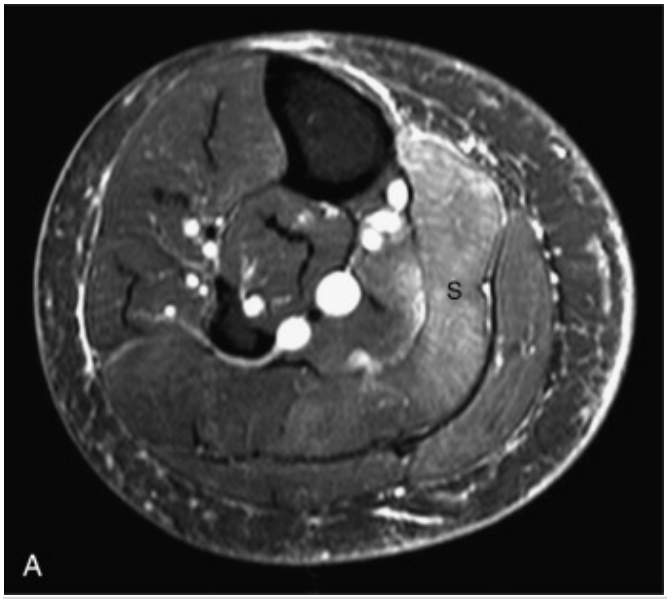
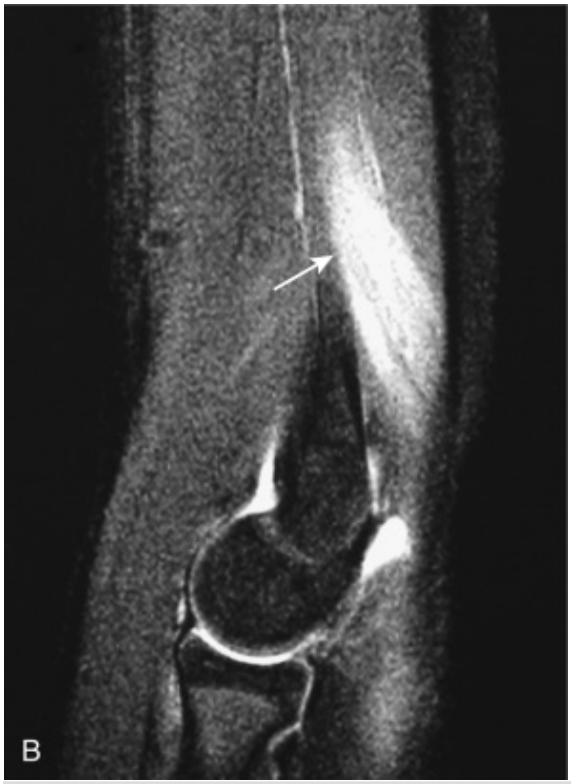


**Fig. 3.38**

**Idiopathic inflammatory polymyopathies. A** , STIR axial image of the lower leg. There is patchy high signal within multiple muscles, most prominent in the medial portion of the soleus muscle (S) in this patient with known polymyositis. **B** , STIR sagittal image of the elbow. A focal area of diffusely increased signal intensity is evident within the triceps musculature ( *arrow* ). This child presented with elbow pain and a skin rash. Subsequent biopsy of the rash revealed dermatomyositis.




Muscle involvement is typically nonuniform in these diseases, and MRI is useful to direct a biopsy to an abnormal muscle early in the disease process to make a diagnosis since non–image-guided biopsies can have a 25% false-negative rate because of sampling error.

When disease is established, increased weakness in these patients can be from several sources, such as an inflammatory flare involving the muscles (which responds to steroid therapy), a

steroid-induced myopathy, or progressive muscle atrophy. MRI is useful to distinguish among these possibilities.

## Primary Muscle Diseases

## Dystrophies and Myopathies

Muscular dystrophies and congenital myopathies can sometimes be differentiated on the pattern of muscle involvement. Duchenne's muscular dystrophy tends to have symmetrical involvement progressing from proximal to distal, whereas myotonic dystrophy tends to progress from distal to proximal.

MRI can show the pattern of muscle involvement, which may help confirm a diagnosis; MRI also can be valuable in guiding a biopsy to an involved muscle. Similarly, MRI can identify muscles that are selectively spared by the disease process and can be used for muscle transfer operations. MRI more accurately depicts progression of disease than serum enzyme levels.

Early in the disease, MRI generally shows increased signal intensity on T2W images as a result of edema and inflammation in the abnormal muscle. Atrophy of the muscle is the predominant finding late in the disease, and high signal intensity from fatty infiltration of the atrophied muscle is best seen on T1W images.

## Denervation ( **Boxes 3.11 and 3.12 (b0060)** )

Muscles and the nerves that supply them can be considered as a single motor or neuromuscular unit. Damage to a nerve causes changes in the muscle supplied by that nerve. Several traumatic, vascular, congenital, metabolic, and infiltrative processes can disrupt the nerve supply to muscle.

**BOX 3.11**

## Fatty Atrophy of Muscle

- High signal intensity on T1 images
- Differential diagnoses
  - Denervation
  - Burned-out dermatomyositis or polymyositis
  - Disuse
  - Muscular dystrophies
  - Congenital myopathies

**BOX 3.12**

## MRI Appearance of Denervation

- Acute (< 2 wk)
  - Normal signal in muscle
- 1-12 mo
  - High signal in affected muscles on T2 from extracellular, intramuscular edema
- > 12 mo
  - High signal on T1 from fatty infiltration of atrophied muscle

The MRI changes after denervation follow a predictable course. Acutely, MRI may be normal. After about 2 weeks, extracellular water increases within muscle, resulting in increased signal intensity on T2W images, especially fat-suppressed images such as STIR ( Fig. 3.39 (f0200) ). This appearance tends to persist for about 1 year after the insult. If the nerve heals, the signal intensity returns to normal. If reinnervation does not occur, fatty atrophy develops in the muscle, which is easily detected on T1W images as high signal intensity replacing muscle fibers ( Fig. 3.40 (f0205) ). These early and late changes in muscle are not specific unless the pattern of muscle involvement correlates with a known nerve distribution, which would suggest the diagnosis.

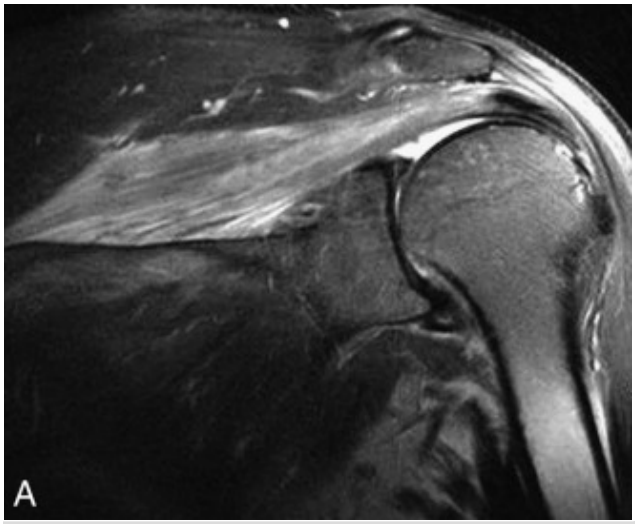



**Fig. 3.39**

**Subacute denervation. A** , Fat-saturated T2 oblique coronal image of the shoulder. Diffuse edema is present within the supraspinatus muscle (SS) in this patient who sustained a traumatic injury to the suprascapular nerve. **B** , T1 oblique coronal image of the shoulder. Mild, streaky increased signal is noted within the muscle indicating minimal fatty atrophy.

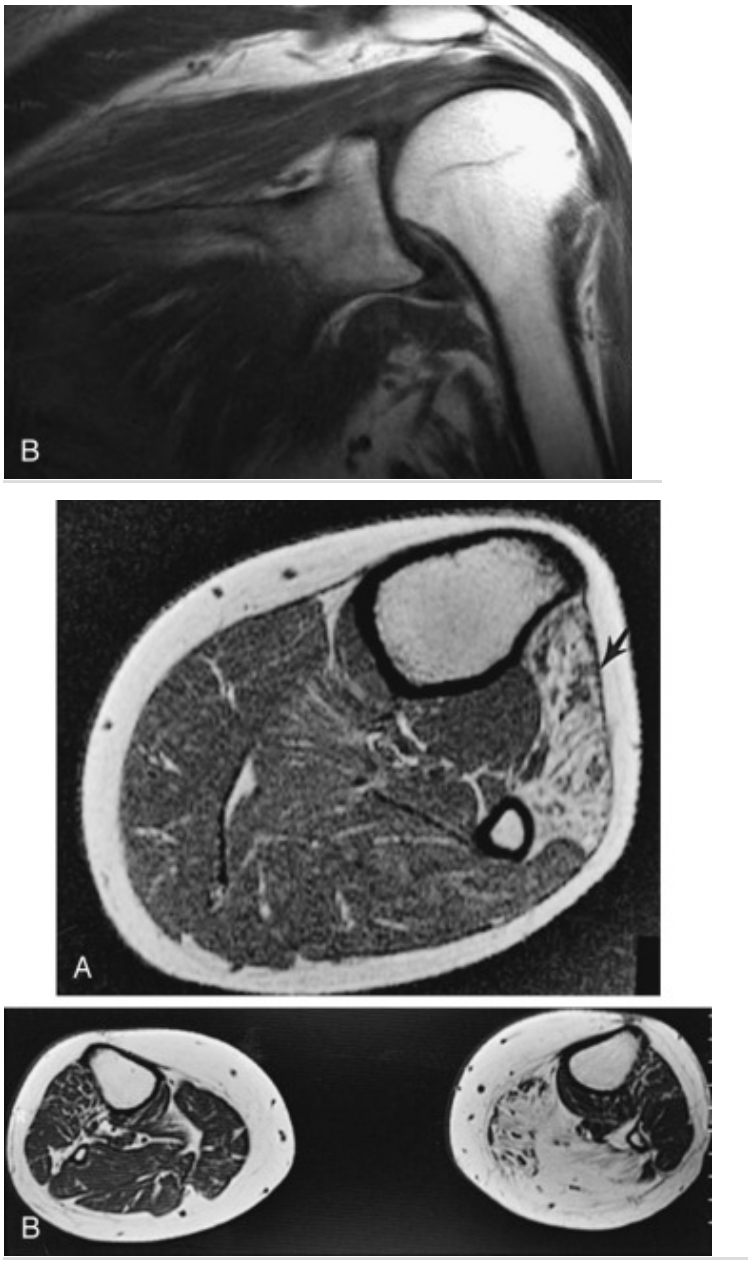



**Fig. 3.40**

**Late denervation.** **A** , T1 axial image of the calf. The tibialis anterior muscle shows fatty atrophy ( *arrow* ) compared with the normal adjacent muscles. **B** , T1 axial image of the calves (different patient than in **A** ). Profound fatty replacement of the posterior calf musculature on the left is evident from long-standing denervation in this patient with diabetes.

MRI can predict whether a muscle would not be salvageable from reinnervation or nerve grafting by showing that the muscle has undergone fatty atrophy, which is irreversible at that point. MRI can also be useful to map out muscles that are spared and could be

used in muscle–tendon transfer operations. Occasionally, the cause of the neuropathy can be shown with MRI. MRI has several advantages over electromyographic evaluation: it is noninvasive, has excellent resolution, and can show pathology in muscles with aberrant or dual nerve supplies.

## Tumors ( Table 3.2 (t0015) )

MRI is frequently used in the work-up of soft tissue masses, many of which arise in muscle. The MRI appearance of some masses, such as intramuscular lipomas, hematomas, and hemangiomas, is sufficiently specific so that no further tests are necessary. Most disorders have a nonspecific appearance, however, with increased signal on T2W images and variable enhancement. Although biopsy is often necessary, MRI is still useful to localize the most viable and diagnostic tissues for biopsy.

Table 3.2

Intramuscular Tumors

| Lesion | Discriminators |
|---|---|
| Hemangioma | Contains fat |
| Lipoma | Is fat |
| Myxoma | Many cyst characteristics |
| Sarcoma | No surrounding edema |
| Metastases | Often surrounding edema |
| Lymphoma | Infiltrates entire muscle |
| Neurofibroma | Target sign possible; resembles myxoma, but diffuse enhancement |

Common intramuscular tumors include hemangioma, lipoma, myxoma, neurofibroma, sarcoma, metastases, and lymphoma. Hemangiomas usually have serpiginous channels surrounded by fat that can be diagnosed by MRI ( Fig. 3.41 (f0210) ). Lipomas also are easily diagnosed as masses with fat signal intensity on all pulse sequences ( Fig. 3.42 (f0215) ). Lymphoma often infiltrates an entire muscle but also may appear as a rounded mass ( Fig. 3.43 (f0220) ); metastases to muscle are rounded masses that often have large areas of edema surrounding them ( Fig. 3.44 (f0225) ). Metastases from malignant melanoma may have a classic MRI appearance, consisting of focal lesions in muscle that are high signal intensity on T1W images and very low signal on STIR or T2W images as a consequence of melanin (see Fig. 3.44 (f0225) ). Soft tissue sarcomas are rounded masses, but they generally do not have edema surrounding them; they may become necrotic, and hemorrhage into the necrotic tumor could be mistaken for a traumatic hematoma. Myxomas have signal characteristics that resemble fluid (very low signal on T1W [lower than muscle] and very bright signal on T2W images [fluid signal]) with heterogeneous or only peripheral enhancement when contrast material is administered ( Fig. 3.45 (f0230) ) (not diffuse enhancement and not exclusively peripheral enhancement [“sort of, kind of enhances”]). Intramuscular neurofibromas generally have a nonspecific appearance—a mass that is low signal on T1W images (usually isointense or slightly higher intensity than muscle) that may be difficult to distinguish from normal surrounding muscle and diffuse increased signal on T2W images (mimicking fluid signal). A target sign may be evident on T2W or contrast-enhanced images (low signal in the center of the mass with high signal periphery).

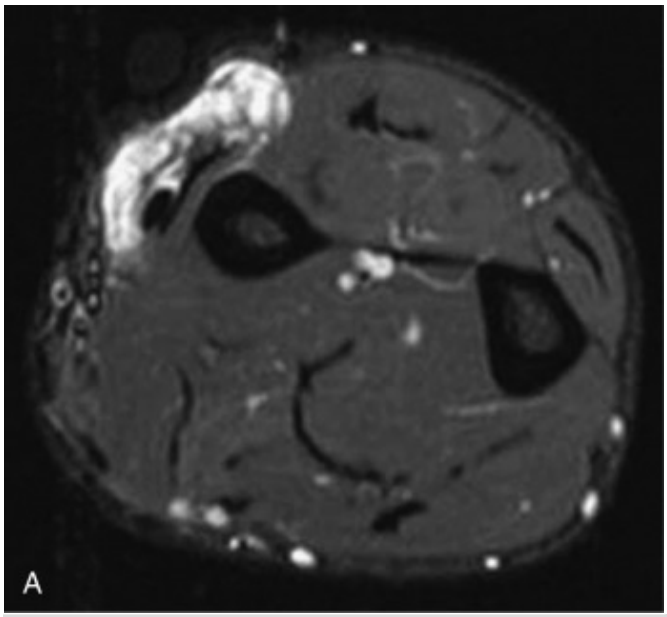
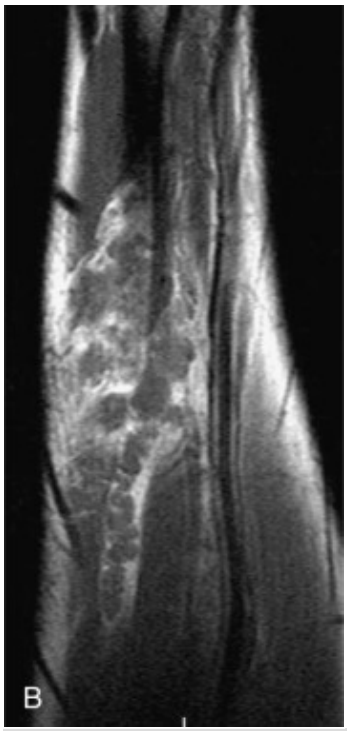



**Fig. 3.41**

**Intramuscular tumor: hemangioma. A** , STIR axial image of the forearm. A mass showing lobular areas of high signal is present within the extensor carpi radialis brevis muscle. **B** , T1 coronal image of the forearm. The lobular components are separated by fat, an appearance compatible with a soft tissue hemangioma.



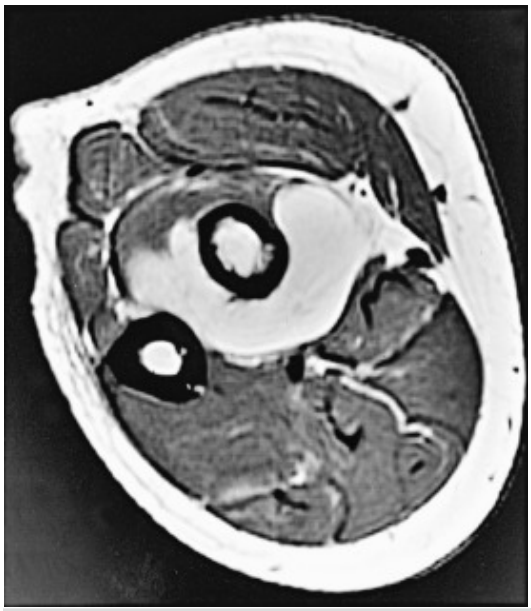
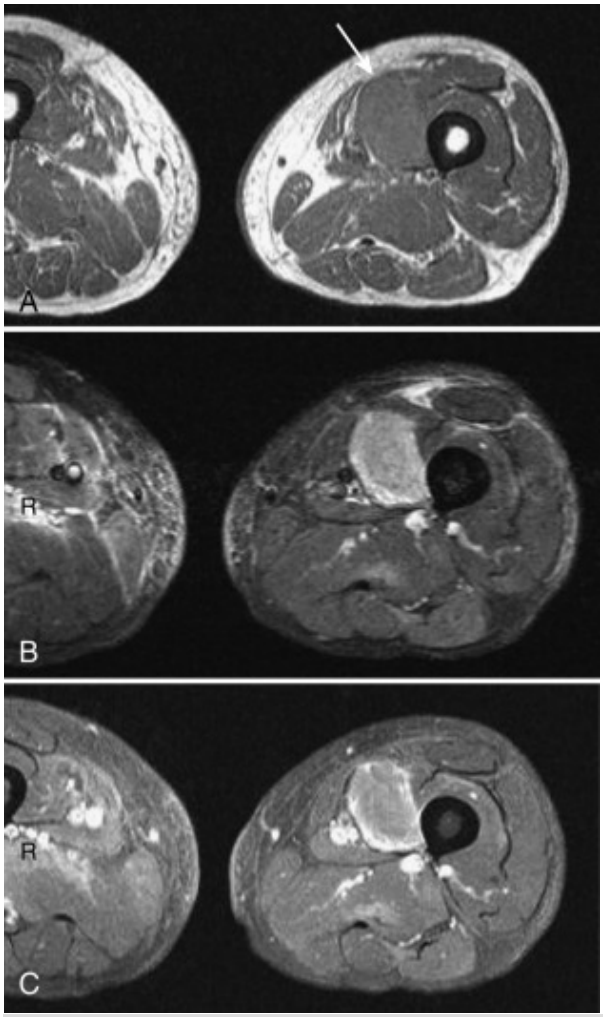


**Fig. 3.42**

**Intramuscular tumor: lipoma.** T1 axial image of the forearm. There is a high signal fatty mass replacing most of the supinator muscle and extending between the radius and the ulna.



## Fig. 3.43

**Intramuscular tumor: lymphoma.** **A** , T1 axial image of the thigh. A masslike area of intermediate signal intensity is evident within the medial aspect of the vastus intermedius ( *arrow* ). **B** , STIR axial image of the thigh. The mass shows diffusely increased signal intensity. **C** , T1 axial image with fat saturation after IV gadolinium administration. There is prominent enhancement along the margins of the mass with less enhancement centrally. Note the nonspecific signal characteristics and well-defined margins of this intramuscular lymphoma.

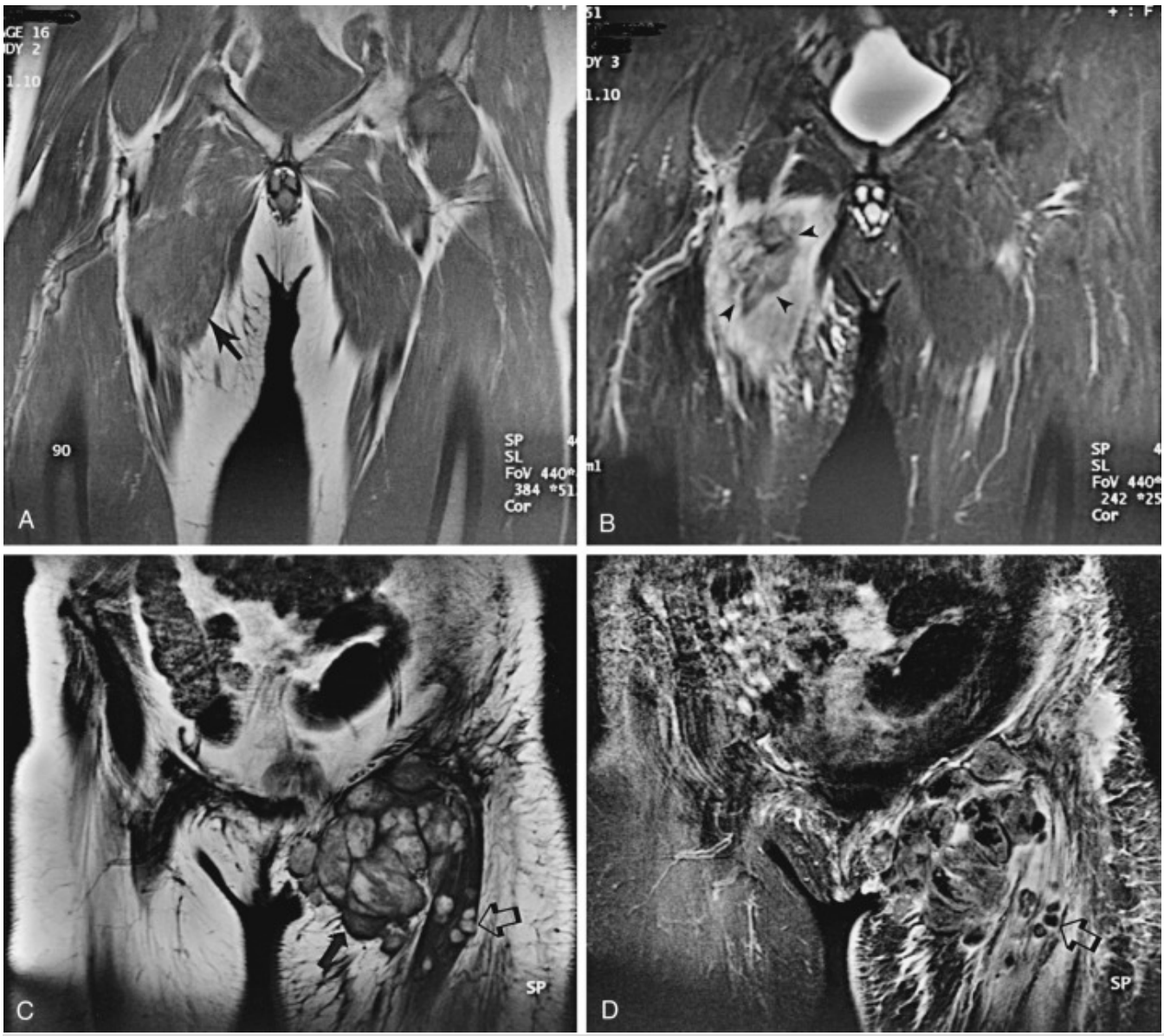


**Fig. 3.44**

**Intramuscular tumor: metastases.** **A** , T1 coronal image of the pelvis. A mass is in the right adductor muscles ( *arrow* ). **B** , STIR coronal image of the pelvis (same patient as in **A** ). The mass is heterogeneous and intermediate to high signal ( *arrowheads* ). The mass is surrounded by a large amount of high signal edema. This was metastatic transitional cell cancer from the bladder. **C** , T1 coronal image of the pelvis (different patient than in **A** and **B** ). A mass of enlarged lymph nodes is present in the left groin. Several rounded high signal masses are evident in the left sartorius muscle ( *open arrow* ). **D** , STIR coronal image of the pelvis (same patient as in **C** ). The intramuscular masses in the sartorius ( *open arrow* ) and several focal areas in the enlarged lymph nodes demonstrate profoundly low signal intensity. In addition, there is edema in the sartorius muscle surrounding the focal lesions. These masses are from metastatic malignant melanoma, and the signal intensity changes are typical for that tumor.

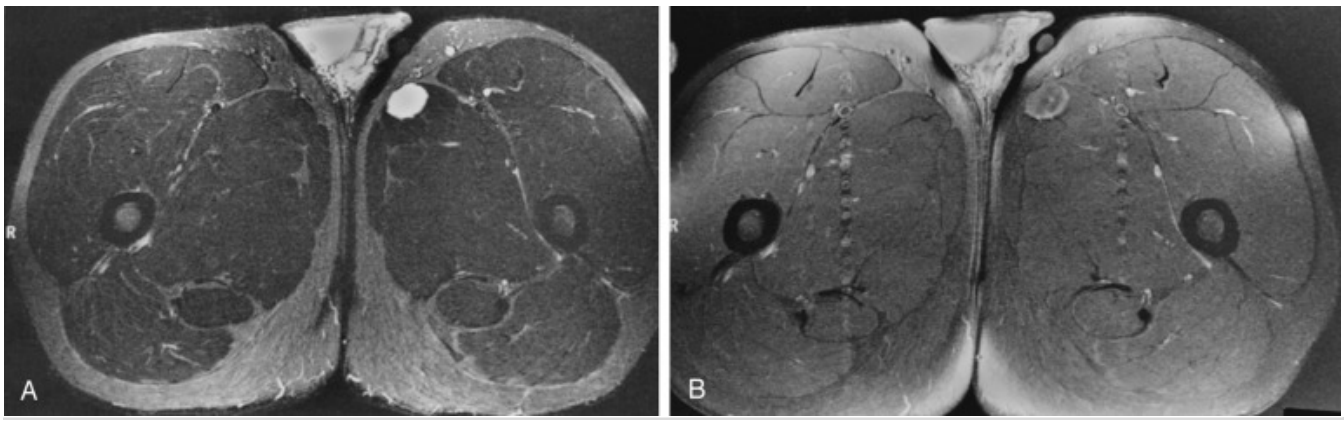



**Fig. 3.45**

**Intramuscular tumor: myxoma.** **A** , STIR axial image of the thighs. There is a very high signal mass in the left adductor musculature. **B** , T1 contrast-enhanced axial image of the thighs with fat suppression. This myxoma shows peripheral enhancement.

## Miscellaneous Muscle Abnormalities

## Rhabdomyolysis

Many entities may cause massive destruction of muscle with increased serum levels of creatine kinase enzyme, including massive trauma, prolonged immobilization, vascular ischemia, excessive exercise, drug or alcohol overdose, and metabolic disorders such as hypokalemia, among others. The involved muscles show diffuse increased signal intensity on T2W images resulting from edema, necrosis, and hemorrhage ( Fig. 3.46 (f0235) ). T1W images do not demonstrate an abnormality, and the muscles usually are not enlarged.

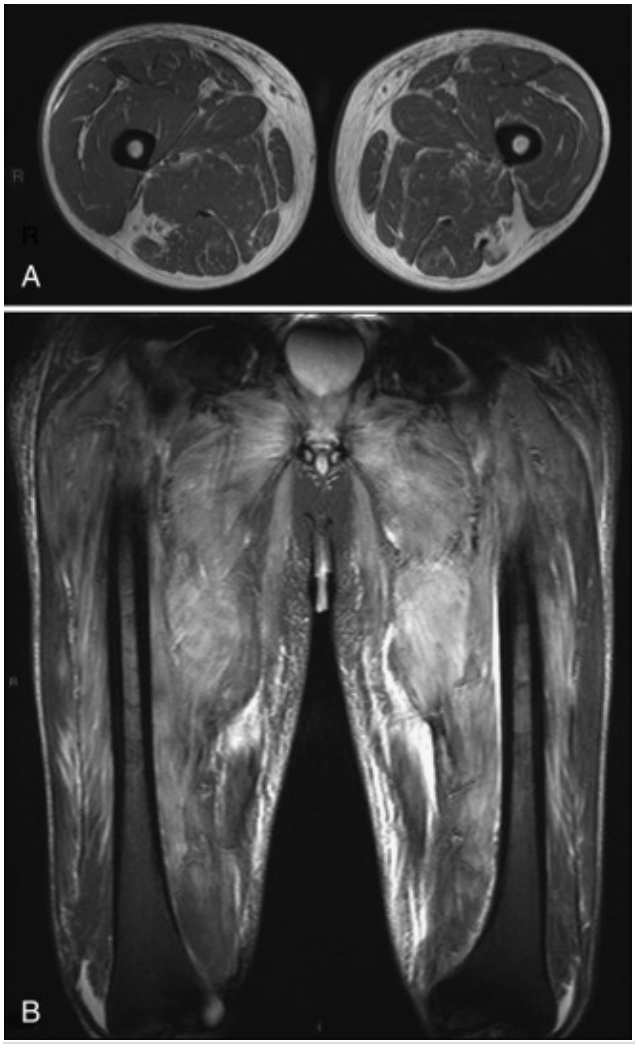


**Fig. 3.46**

**Rhabdomyolysis. A** , T1 axial image of the thighs. No abnormality is evident in this patient with a history of lipid therapy with statin and fibrate drugs. **B** , STIR coronal image of the thighs. Diffuse, high signal intensity is seen throughout the thigh musculature compatible with extensive rhabdomyolysis.

## Muscle Infarction ( Box 3.13 (b0070) )

Muscle ischemia is particularly common in people with diabetes and patients with sickle cell anemia. Ischemic changes are very painful and result in edema with focal and diffuse areas of increased signal intensity in and surrounding muscle on T2W images.

**BOX 3.13**
**Muscle Ischemia**

- Common in diabetes and sickle cell anemia
- Diabetic muscle infarction
  - Severe pain with or without swelling/mass
  - Normal white blood cell count
  - Thigh involved in 80%; calf involved in 20%
  - Bilateral in one third of patients
- MRI appearance
  - T1: muscle swelling, obliteration of fat planes
  - T2: diffuse intramuscular and intermuscular increased signal

- Contrast: diffuse enhancement of affected area or ring enhancement of necrotic foci

Diabetic muscle ischemia (previously referred to as *diabetic muscle infarction/necrosis* ) occurs from thrombosis of medium and small arterioles in patients with atherosclerosis and poorly controlled diabetes. Clinical symptoms consist of severe pain with a history of a palpable mass with or without swelling. The white blood cell count is normal. More than half of patients have coexistent diabetic nephropathy, neuropathy, and retinopathy.

The thigh, especially the vastus musculature, is involved most commonly (≈ 80%); the calf is next most frequently affected (≈ 20%). The ischemia may start in the calf and progress to the thigh. There is bilateral involvement in more than one third of cases.

MRI shows diffuse enlargement of several muscles, and more than one compartment often is involved ( Fig. 3.47 (f0240) ). The intermuscular fatty septa may be obliterated, and subfascial edema often is evident. On T2W images, there is increased signal intensity diffusely in and between muscles. There may be foci of hemorrhage. Gadolinium administration may show diffuse enhancement, but there also may be focal areas of rim enhancement, probably from hyperemia around an area of nonperfused muscle.

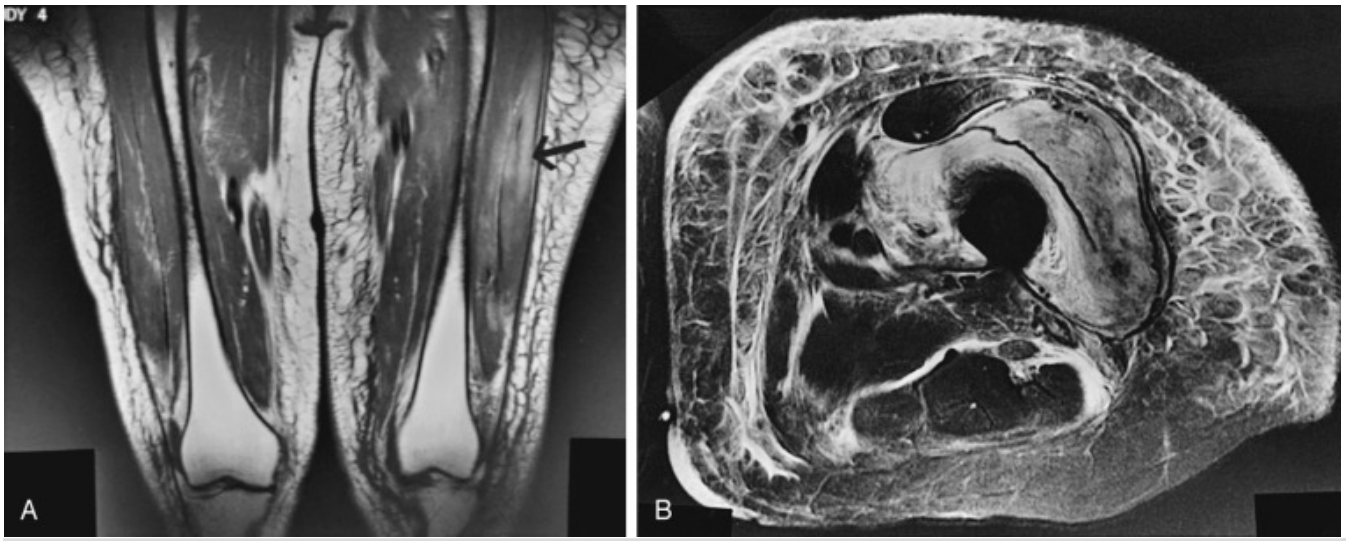



**Fig. 3.47**

**Muscle infarction. A** , T1 coronal image of the thighs. Both thighs are abnormal, but the findings are much more pronounced on the left. There is subcutaneous edema with a reticular pattern. The muscles are enlarged, and high signal in a left thigh muscle ( *arrow* ) indicates hemorrhage from the muscle infarction in this patient with diabetes. **B** , STIR axial image of the left thigh. Subcutaneous edema with reticular high signal is present diffusely. There is increased signal within the vastus musculature, and intermuscular septa show high signal as well.

The diagnosis is not specific based on MRI alone, but in conjunction with the clinical history, a specific diagnosis often can be made. If there is any question about the diagnosis, percutaneous biopsy for culture and histology can be performed.

## Accessory Muscles

Accessory muscles may be an incidental finding on MRI examinations; they also may manifest clinically as a mass or cause compression of an adjacent nerve. Accessory muscles occur in many locations throughout the body. The diagnosis of an accessory

muscle is easy with MRI because the signal intensity and feathery texture are identical to other adjacent muscles on all imaging sequences ( Fig. 3.48 (f0245) ).

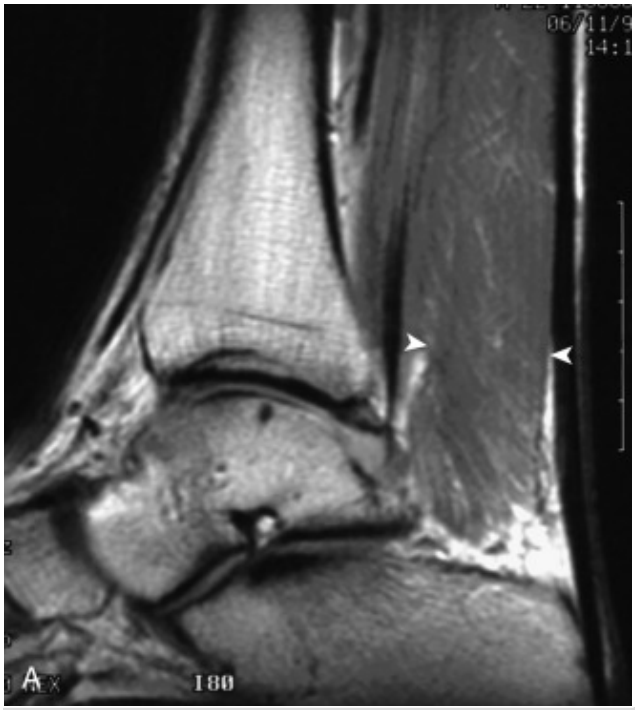
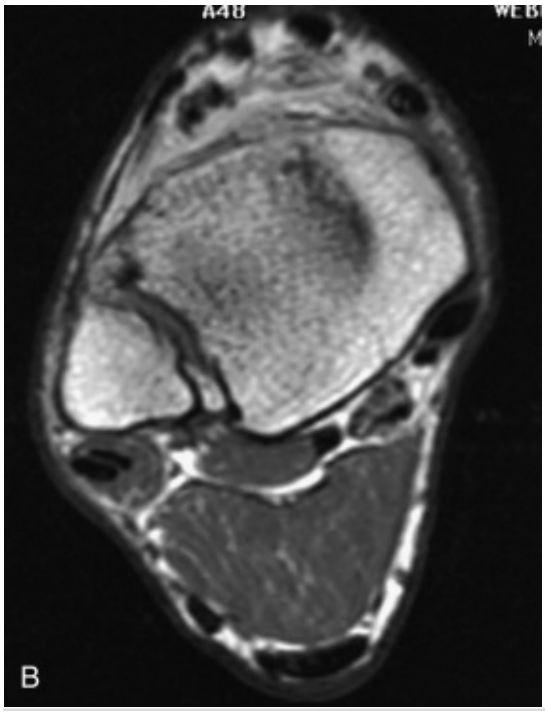


**Fig. 3.48**

**Anomalous muscle. A** , T1 sagittal image of the ankle. This is a basketball player who presented with a pre-Achilles mass. Intermediate signal intensity tissue with fatty striations is present within the pre-Achilles fat ( *arrowheads* ), an appearance compatible with skeletal muscle, in this case, an accessory soleus muscle. **B** , T1 axial image of the ankle. The accessory muscle fills the pre-Achilles space.




## Radiation, Surgery, and Chemotherapy

Lesions in the extremities that are treated with surgery, local radiation therapy, or chemotherapy often develop diffuse high signal intensity in the muscle and subcutaneous fat with the typical feathery appearance in muscle on T2W images ( Fig. 3.49 (f0250) ). These findings are nonspecific, but they should not be confused with persistent or recurrent tumor because it is a common “normal” finding after treatment. Only the presence of a rounded mass should be of concern for persistent or recurrent tumor in the site of previous surgery or radiation. Often, a mass at a surgical site is a hematoma or seroma, rather than tumor; this can be determined by administering IV contrast material to determine whether the mass is cystic or solid, and if any question remains, an image-guided biopsy can be performed.

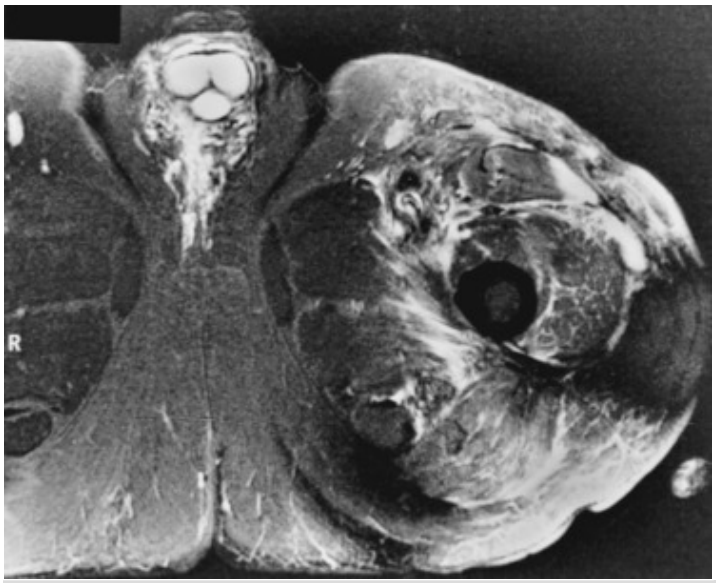


**Fig. 3.49**

**Radiation changes.** STIR axial image of the proximal thigh. There is diffuse intramuscular and subcutaneous high signal. This man received radiation and surgery for a tumor in this region. These findings are expected after therapy. The lack of a round, high signal mass essentially excludes recurrent tumor.

## Suggested Reading

### Tendon Abnormalities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