

Hamstring Muscle Complex: An Imaging Review¹

George Koulouris, FRANZCR • David Connell, FRANZCR

ONLINE-ONLY CME

See www.rsna.org/education/rg_cme.html.

LEARNING OBJECTIVES

After reading this article and taking the test, the reader will be able to:

- Describe the basic anatomic and biomechanical features of the hamstring muscle complex.
- Correlate the underlying pathophysiologic features of this anatomic region with clinical and imaging findings.
- Discuss the role of US and MR imaging in the diagnosis of hamstring muscle complex injury.

Increasing activity in the general population and the high demands placed on athletes have resulted in injuries to the hamstring muscle complex (HMC) being commonplace in sports. Imaging of HMC injuries can form a considerable part of a sports medicine practice, with a wide spectrum of such injuries being reflected in their varied imaging appearances. Magnetic resonance (MR) imaging and ultrasonography (US) are the imaging modalities of choice in this setting. Both MR imaging and US provide exquisitely detailed information about the HMC with respect to localization and characterization of injury. Optimization of MR imaging involves the use of a surface coil and high-resolution techniques, allowing the musculoskeletal radiologist not only to diagnose injury and assess severity but also to provide the clinician with useful clues with respect to prognosis. The portability and availability of US make it an attractive modality for the diagnosis of acute hamstring injuries, although its effectiveness is dependent on operator experience. A thorough knowledge of the HMC anatomy and of the spectrum of imaging findings in HMC injury is crucial for providing optimal patient care and will enable the musculoskeletal radiologist to make an accurate and useful contribution to the treatment of athletes at all levels of participation.

©RSNA, 2005

Abbreviations: ACL = anterior cruciate ligament, HMC = hamstring muscle complex, MTJ = musculotendinous junction

RadioGraphics 2005; 25:571–586 • **Published online** 10.1148/rg.253045711 • **Content Codes:** MR MK US

¹From the Department of Radiology, The Alfred Hospital, Melbourne, Australia (G.K.); and the Department of Radiology, Royal National Orthopaedic Hospital, Brockley Hill, Stanmore, Middlesex HA7 4LP, England (D.C.). Received May 13, 2004; revision requested June 23 and received October 12; accepted October 14. All authors have no financial relationships to disclose. **Address correspondence to** D.C. (e-mail: david.connell@moh.nhs.uk).

©RSNA, 2005

Introduction

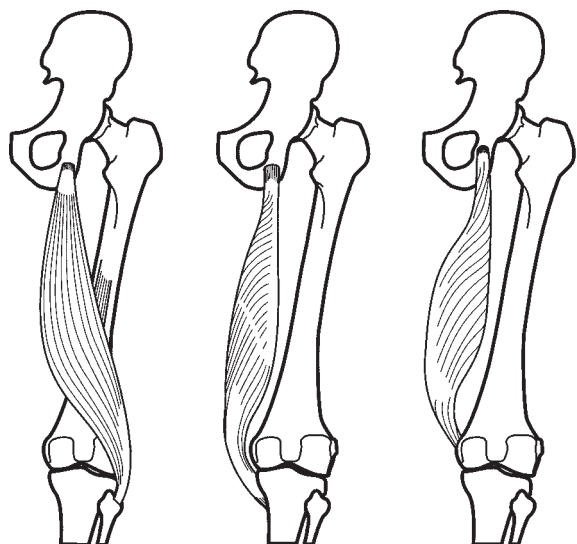
Assessment of muscle injury is a routine part of the daily workload for the musculoskeletal radiologist. The hamstring muscle complex (HMC) is by far the most frequently injured muscle (1–3) and is often recalcitrant to even the most meticulous rehabilitation, making HMC injury a significant contributor to athletic morbidity.

Clinicians are now turning to imaging tests to confirm injury as well as to provide information about a proposed period of convalescence. Minimizing the amount of time spent out of training and competition is not only critical for the professional athlete but also important for an active general population in whom injury can often limit leisure activity. The goals of imaging are to confirm injury, provide a comprehensive assessment of the nature of the injury, and identify which patients may benefit from surgery. Imaging may not be necessary in all cases, and clinical data can provide valuable information about the nature of an injury; hence, a close working relationship with the sports medicine physician or orthopedic surgeon is advantageous. Tendon avulsion generally requires surgical reattachment, whereas strain patterns of injury are managed conservatively. A detailed knowledge of the anatomic, biomechanical, and pathophysiologic features of the HMC and of the various imaging manifestations of hamstring injuries is therefore necessary for providing the referring clinician with an accurate diagnosis and report.

History and clinical examination will help diagnose a hamstring strain in most cases. The patient typically describes sudden excruciating pain in the posterior thigh, resulting in the immediate cessation of competitive activity. However, not all posterior thigh pain is the result of hamstring strain or, indeed, of hamstring disease (Table). Furthermore, not all strain injuries of the HMC manifest with this classic history. Differentiating between injury and muscle soreness, identifying recurrent tears in the rehabilitating athlete, or

Differential Diagnosis for Posterior Thigh Pain

- Hamstring strain
 - Acute
 - Recurrent
 - Chronic
- Ischial tuberosity disease
 - Avulsion fracture
 - Acute
 - Chronic or nonunited
 - Apophysitis
 - Painful nonunited apophysis
- Hamstring enthesopathy
 - Proximal
 - Distal (tenosynovitis)
- “Hamstring syndrome” (pain localized to the ischial tuberosity and radiating down the hamstring muscle during or after exercise)
- Referred pain
 - Lumbar spine
 - Sacroiliac joints
 - Pubic symphysis
 - Nerve tissue, meninges
 - Gluteal muscles
- Hamstring contusion
- Myositis ossificans
- Bursitis
 - Ischiogluteal
 - Pes anserinus
 - Semimembranosus
 - Trochanteric
- Ligament strain
 - Sacrotuberous
 - Sacrospinous
- Posterior compartment syndrome
 - Acute
 - Chronic
- Sciatic nerve
 - Hematoma compression
 - Entrapment
 - Radiculopathy
 - Tumors (schwannoma, neurofibroma)
- Bone tumors
- Spondyloarthropathies or sacroiliitis
- Stress fractures or insufficiency fractures
 - Pelvis
 - Femoral neck
- Vascular claudication



BICEPS FEMORIS SEMITENDINOSUS SEMIMEMBRANOSUS

Figure 1. Drawings illustrate the three muscles in the posterior compartment of the thigh that together constitute the HMC. The short head of the biceps femoris muscle is deep to the long head. The tendinous nature of the semitendinosus muscle inferiorly is appreciated, as is its raphe. The origin of the semimembranosus muscle is noted to be superolateral to the conjoint tendon.

diagnosing an acute injury against a background of prior chronic strain can be difficult clinically. The latter situation is often clouded by the presence of scar tissue within the muscle. Furthermore, an acute intramuscular hemorrhage following direct impact, which is not uncommon in contact sports, can be difficult to differentiate from a muscle tear on the basis of imaging findings alone. Referred pain, most commonly from the lumbar spine and sacroiliac joint, may further complicate the clinical picture and commonly coexists with HMC strain injury in the highly trained athlete.

Overall, the prognosis for HMC injury is good, even in the setting of avulsion injury, provided the injury is diagnosed and treated early. Many athletes return to professional competition following tendon reattachment; however, a few may have chronic disabling symptoms or recurrence of

avulsion (3,4). Strain injury has a far better prognosis, although recurrence of strain is common and may result in the need for further rehabilitation and time out from competition. Even upon the athlete's return, strain injury to the HMC can often result in reduced levels of fitness, strength, confidence, and even skill, inevitably threatening an elite athlete's career.

In this article, we review the normal anatomy and biomechanical features of the HMC as well as the pathophysiologic features of HMC injury. We also discuss and illustrate the imaging appearances of both the normal HMC and injuries to this anatomic structure. In addition, we discuss the correlation between these imaging appearances and clinical findings.

Normal Anatomy

The three muscles that constitute the HMC are the biceps femoris, semitendinosus, and semimembranosus muscles (Fig 1). Some anatomists consider the adductor magnus muscle to be a hamstring muscle, but for the purposes of this review it will not be considered as such.

Biceps Femoris Muscle

In morphologic and functional terms, the biceps femoris muscle is considered to be a double muscle, with the long head arising from the medial facet of the ischial tuberosity (Fig 2) and the short head arising from the lateral linea aspera, lateral supracondylar line, and intermuscular septum. The short head is the only component of the HMC that does not span two joints; consequently, it has been postulated that the short head is not a true hamstring (5). Occasionally, the short head may be absent (6). The origin of the biceps femoris muscle on the femur has been used as a consistent landmark in distinguishing between proximal and distal injuries (7). The distal biceps femoris tendon inserts onto the head of the fibula, the lateral condyle of the tibia, and the fascia of the leg, a rather extensive attachment that is thought

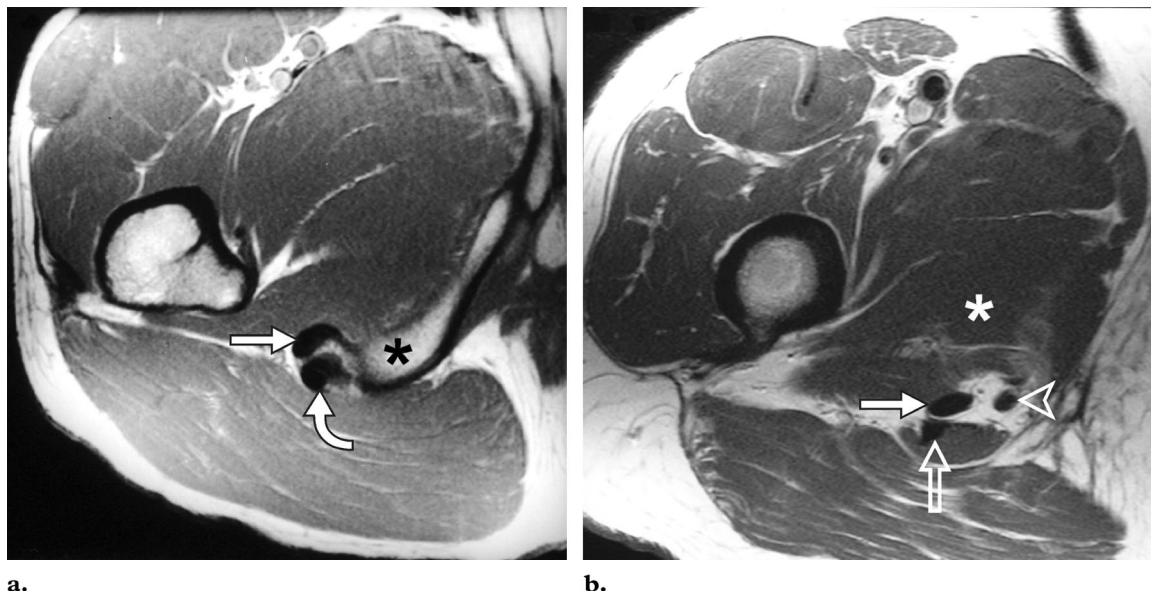


Figure 2. Normal anatomy of the proximal HMC. **(a)** Axial magnetic resonance (MR) image obtained at the level of the ischial tuberosity (*) shows the conjoint tendon of the biceps femoris and semitendinosus muscles posteromedially (curved arrow), with the semimembranosus muscle anterolaterally (straight arrow). **(b)** Axial MR image obtained inferior to **a** at the level of the proximal third of the femur shows the belly of the semitendinosus muscle (solid arrow) and the semimembranosus tendon (arrowhead). Note the laterally placed muscle fibers of the adductor brevis (*). The low-signal-intensity adductor magnus tendon (open arrow) lies anterior to the conjoint tendon.

to predispose it to tears. The proximal and distal tendons with the corresponding musculotendinous junction (MTJ) span the entire length of the biceps femoris muscle, with both the short and long heads contributing to the formation of the distal tendon (1). The long head is innervated by the tibial portion of the sciatic nerve and the short head by the peroneal division. The dual innervation of the biceps femoris muscle may result in asynchrony in the coordination or intensity of stimulation of the two heads, which is also postulated as a cause for this muscle having the highest frequency of tears of the HMC (8,9).

Semitendinosus Muscle

The semitendinosus muscle is a single muscle but is best considered physiologically as a digastric muscle, given that it possesses an intervening raphe onto which the proximal fibers insert. These fibers arise from the inferomedial impression of the upper portion of the ischial tuberosity by way of a conjoint tendon with the long head of the biceps femoris muscle (Fig 2). Caudal to the ischial tuberosity, the semitendinosus muscle becomes bulbous, with the semimembranosus tendon lying anterior to it. The semimembranosus muscle is often mistaken for the semitendinosus muscle because the proximal tendon of the latter

is not always a distinct structure. More distally, the semitendinosus muscle forms a long tendon. This elongated distal tendon may predispose the muscle to rupture (10). The muscle fibers distal to the raphe insert onto the tibia with the gracilis muscle at the Gerdy tubercle (11). Nerve supply is from two distinct branches from the tibial nerve, the lower branch arising in common with the nerve to the semimembranosus muscle.

Semimembranosus Muscle

The semimembranosus muscle originates on the superolateral aspect of the ischial tuberosity, beneath the proximal half of the semitendinosus muscle. The semimembranosus tendon runs medial and anterior to the other hamstring tendons. The proximal tendon is an elongated structure, with connections to the adductor magnus tendon and the origin of the long head of the biceps femoris muscle. The semimembranosus muscle is recognized by its sharp medial border and cordlike appearance (9). More distally, it is mostly composed of muscle, with numerous short unipennate and multipennate fibers, maximizing the number of muscle fibrils per unit area (11). In contrast, the semitendinosus muscle is a largely thin, bandlike tendinous structure after its origin and for most of its course through the thigh (Fig 2). The semimembranosus muscle has multiple insertions (5,11) by way of five tendinous arms,

or expansions, to the medial tibial condyle (anterior, direct, and inferior arms), the posterior oblique ligament (capsular arm), and the posterior joint capsule and arcuate ligament (oblique popliteal ligament). The first three arms are closely related to the tibial collateral ligament, coursing deep to it (12). A U- or J-shaped bursa exists between this ligament and the semimembranosus attachments, which have characteristic morphologic features when pathologically inflamed (10,13,14). In slightly less than one-half of the population, small slips of the semimembranosus tendon insert onto the posterior horn of the lateral meniscus (12,15). The nerve supply is from a single branch arising from the tibial division of the sciatic nerve. As with the biceps femoris muscle, the proximal and distal tendons span the entire length of the muscle (1).

Variations in anatomy may predispose certain patients to injury that may lead to a decrease in the normal glide and flexibility of the muscles. This is true for the short and long heads of the biceps femoris muscle, whose myofascial interface is a common site for injury. For example, slips between the hamstring muscles may be given off and can be quite large (6), resulting in variations in the extent of origin and insertion points and causing a decrease in flexibility by way of tethering. In rare cases, the short head of the biceps femoris muscle may fail to share the same insertion as the long head (11). The semimembranosus muscle can be quite large and occasionally exists as a double muscle (7,16,17), in which case it arises from the sacrotuberous ligament. Conversely, the semimembranosus muscle may be absent (18).

Biomechanical Features

The muscles of the HMC are important hip extensors and flexors of the knee in the gait cycle. They become active in the last 25% of the swing phase just as hip extension begins and continue for 50% of the swing phase to actively produce extension at the hip and actively resist extension of the knee. As the thigh is swung forward, flexion at the knee is largely passive, accounting for the paucity of strains at this stage (19). With heel strike, the HMC also functions to decelerate the forward translation of the tibia during knee extension when foot strike occurs and the weight of the body is shifted forward. The HMC is thus a dynamic stabilizer of anterior tibial translation, working alongside the corresponding static stabilizer, the anterior cruciate ligament (ACL). This occurs particularly when the knee is flexed at 30° and the foot reaches its greatest distance forward

from the body (19). Once foot strike has occurred, the muscles of the HMC are elongated over both hip and knee joints to their optimal length to provide extension of the hip and to once again stabilize the knee. With takeoff, the hamstring muscles again contract with the quadriceps muscle to provide a pushoff from the support leg.

The sudden change in HMC function from a stabilizing role in flexion to rapid activity in extension has been postulated as a cause for injury (19). The biarticular nature of these muscles implies that their contraction cannot be localized to only one joint. Therefore, it is crucial that one joint be stabilized to act on the other. This stabilization is brought about by the contraction of antagonists, the disproportionately larger quadriceps muscle, or ground reaction forces (2). The HMC must therefore create sufficient force to absorb or counteract these forces. Failure to do so ultimately results in strain. A relative imbalance between the strength of the hamstring and quadriceps muscles in which the former is less than 60% that of the latter (20,21), or a significant difference (10% discrepancy) between the two sides of the HMC, have also been proposed as additional biomechanical factors contributing to HMC injury.

Pathophysiologic Features of Hamstring Muscle Strain

The prime function of the HMC is to contract eccentrically, thereby absorbing kinetic energy so as to protect the knee and hip joints. Eccentric contraction occurs when a muscle contracts while being passively stretched. Injury is more likely to occur during eccentric contraction than during concentric contraction, since the tension contributed by stretch is superimposed on that brought about by contraction. Indeed, altered muscle signal intensity is noted at MR imaging following intense eccentric exercise, a finding that is absent after concentric exercise (22). Muscle strain can be viewed as part of a spectrum of muscle disruption of increasing magnitude, ranging from the least severe (delayed onset muscle soreness) to varying degrees of partial strain to complete tear or avulsion (23,24). Any condition that diminishes the ability of a muscle to contract (eg, fatigue, weakness) will make the muscle susceptible to injury because it impairs the muscle's ability to absorb force. For example, even a history of minor hamstring strain will result in incomplete disruptions at the microscopic level and, consequently, a weaker muscle, increasing the risk of

further deleterious injury. Thus, the benefits of adequate rest and an aggressive strengthening rehabilitation program cannot be overstated (8,25,26).

Hamstring muscle injury typically occurs in the region of the MTJ, which, as opposed to being a distinct point, is really a 10–12-cm transition zone in which myofibrils contribute to form the tendon. Highly folded membranes at the muscle-tendon interface increase junctional surface area, an adaptation designed to dissipate energy (27). Tears have been demonstrated microscopically to occur near, although not actually at, the MTJ (28). This region adjacent to the MTJ is more susceptible to injury than any other component of the muscle unit (29). Injury occurs independent of (*a*) the rate or direction of strain and (*b*) differences in muscle architecture. Such injury results in ultrastructural change in which torn myofibrillar Z bands cause protein degradation with release of protein-bound ions leading to edema, which, if of sufficient magnitude, can be visualized at imaging (30–32).

At microscopic analysis, hemorrhage is also seen at these sites of disruption in the acute phase (<24 hours after disruption), followed by an inflammatory reaction whose time of occurrence is variable (usually at day 2) (22) with laying down of fibrous tissue by day 7 to commence the formation of scar tissue (23,33). Such tissue first becomes visible as early as 14 days following initial insult, principally manifesting with low signal intensity (34). At this point, the muscle has regained over 90% of its function. Nevertheless, given that fibrosis results in retraction, the optimal muscle length is altered, and, consequently, so is the ability of the muscle to maximally contract. This phenomenon has been postulated as a mechanism for recurrent hamstring strain despite a rehabilitation period lagging behind histologic resolution (35).

Myofibrillar damage is also most pronounced in muscles with a large proportion of type 2 (fast twitch) fibers (36), which are capable of producing more tension at a greater rate. The HMC possesses a large proportion of such fibers (2). In addition, biarticular muscles, whose role is to limit joint range of motion because of their intrinsic tightness, have passive tension increased by physiologic joint motion and, again, are more susceptible to tear (23,29) and recurrent tear (2).

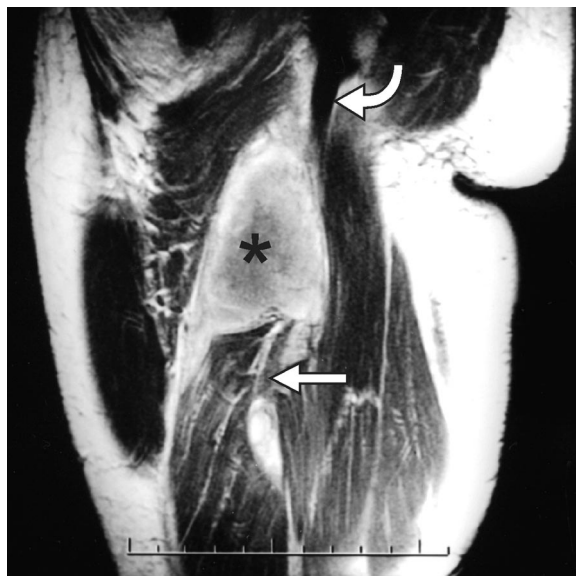


Figure 3. Avulsion injury in a 29-year-old athlete with a hyperextension injury and persistent disability. Oblique coronal MR image demonstrates a large hematoma (*) with retracted fibers of the semitendinosus muscle and the long head of the biceps femoris tendon (straight arrow), findings that are consistent with an avulsion injury. The semimembranosus muscle (curved arrow) remains intact.

Imaging Findings

Understanding the normal MR imaging and ultrasonographic (US) anatomy is essential for interpreting hamstring disease. At MR imaging, two rounded areas of low signal intensity at the region of the origin of the ischial tuberosity with all pulse sequences are consistent with the semimembranosus muscle superolaterally and the conjoint tendon of the biceps femoris and semitendinosus muscles inferomedially (Fig 2). Sometimes these two structures are difficult to separate, reflecting anatomic variation. The semitendinosus is not usually seen as a distinct tendon slip and quickly forms a muscle as it passes into the leg, with the biceps femoris tendon coming to lie on its anterior and lateral surface. The separate and smaller tendon slip of the adductor magnus muscle is situated in front of the semitendinosus, and injury to this structure is rare (37–39).

The most serious acute injury of the HMC is avulsion, which in adults usually involves the tendon but not the bone (Fig 3). Tendon avulsion is important to identify because it necessitates prompt surgical repair. This pattern of injury occurs more commonly at the ischial tuberosity than

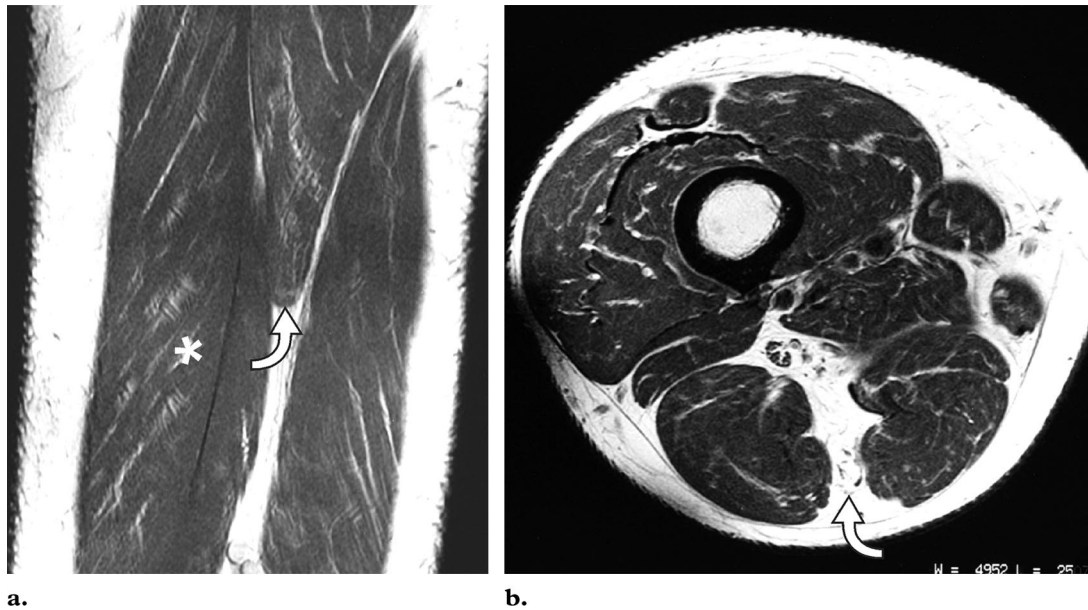


Figure 4. Distal avulsion in a 44-year-old physical therapist who presented with acute distal posterior knee pain during rehabilitation following ACL reconstruction. **(a)** Coronal MR image shows avulsion of the semitendinosus tendon (arrow), with retraction of the muscle. The long head of the biceps femoris muscle is located laterally (*), with the semimembranosus muscle on the medial side. **(b)** Axial MR image shows absence of the normal uniformly low signal intensity of the semitendinosus muscle between the biceps femoris and semimembranosus muscles (arrow), a finding that is consistent with retraction.

at the distal ligamentous insertion (40). In such a case, avulsion almost always involves the conjoint tendon (biceps femoris and semitendinosus muscles) and often results in either complete or incomplete tearing of the semimembranosus. This is the most common form of proximal avulsion. The biceps femoris can arise as a separate and distinct tendon from the semitendinosus as an anatomic variant. In this case, avulsion of the biceps femoris alone carries a better chance of successful surgical repair.

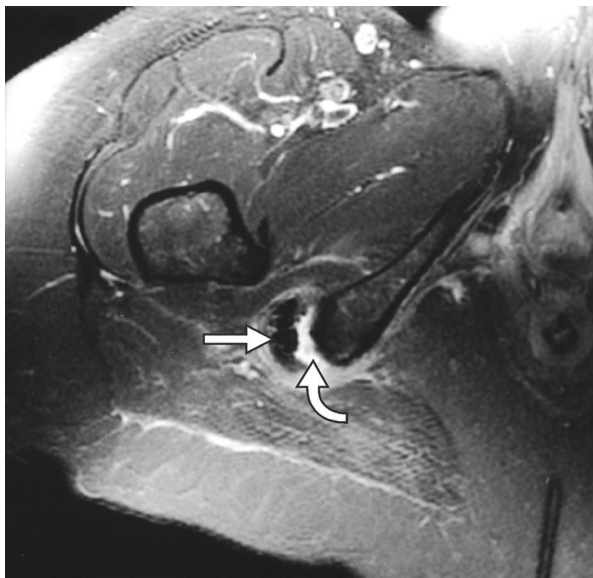
Avulsion injury in the adult is usually without an osseous fragment (40). Conversely, in adolescents, the apophysis forms the weakest link in the musculotendinous unit due to its incomplete ossification, resulting in osseous avulsion. MR imaging is more reliable than US for documenting this injury (10), which can be difficult to detect in the presence of extensive hematoma of varying age. The challenge is compounded by the depth of the injury and by the absorption of US waves by the overlying and, in the athletic setting, often large gluteal muscles covering the proximal hamstring tendons. Conventional radiography allows exclusion of a bone fragment, which has important

clinical and prognostic ramifications. MR imaging allows accurate assessment of the degree of tendon retraction and of tendon edge morphologic features for the surgeon contemplating primary surgical repair.

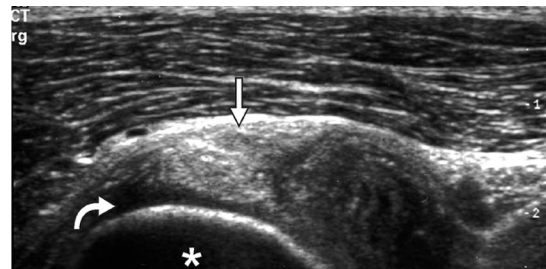
Distal avulsions are uncommon injuries (10,38,39,41) but are most often seen in water skiers and football players (Fig 4). Avulsions of each tendon insertion have been reported, although avulsion of the semitendinosus is probably the most common. Avulsion usually occurs in the setting of prior or chronic injury, with abnormal tendon morphologic features or degeneration being the most likely predisposing factors, as in Achilles tendon rupture (39). A past history of an ACL repair made with the semitendinosus and gracilis tendons from the same side is another causative factor.

MR imaging accurately displays distal tendinous avulsion and the degree of retraction. However, US has superior spatial resolution, which, in combination with the superficial nature of the

Figure 5. (a–c) Chronic tendinopathy in a 29-year-old Olympic marathon runner who presented with a recent injury following a history of chronic posterior thigh pain. (a) MR image through the pelvis demonstrates thickening of the HMC origin with loss of the normal hypointensity of the tendons (straight arrow), findings that are compatible with repetitive microtears. A band of free fluid is also visualized (curved arrow). These findings are typical of partial tear set against a background of enthesopathy. Compare the normal morphologic features of the ischial tuberosity as shown in Figure 2a. (b) Sagittal (longitudinal) US image shows loss of the normal bright fibrillar echotexture of the muscle origin, which instead appears heterogeneous and thickened (straight arrow), findings that are consistent with enthesopathy. The low-echogenicity band deep to the tendon (curved arrow) is consistent with fluid and corresponds to the partial tear seen at MR imaging. * = ischial tuberosity. (c) US image demonstrates the normal appearance of the HMC (straight arrow), which inserts at the ischial tuberosity (*) and demonstrates uniform fibrillar echotexture. Superior to the ischial tuberosity, the HMC tendon blends with and continues as the sacrotuberous ligament (curved arrow). (d) Chronic tendinopathy in a different athlete. MR image shows ill-defined thickening of the HMC origin (arrow) with no discernible tear. There is no evidence of edema in the ischial tuberosity (*).



a.



b.

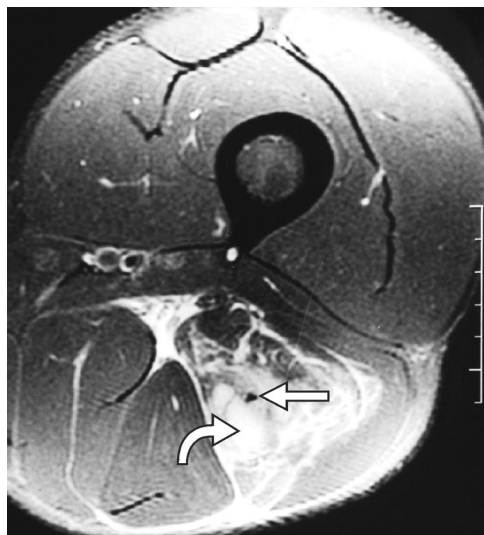


c.

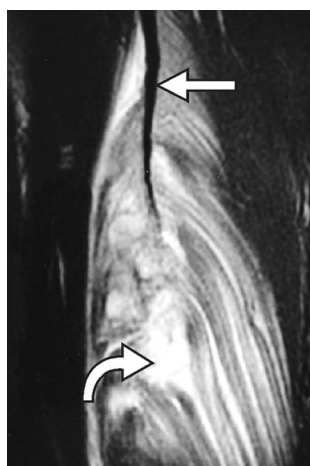


d.

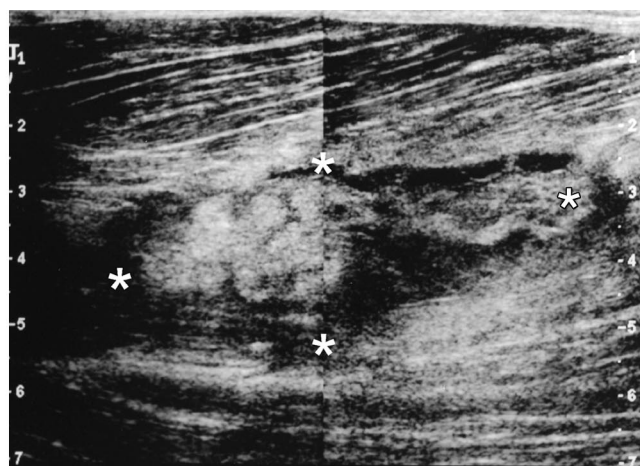
tendon, makes application of this modality ideal. Dynamic assessment can provide additional information about tendon integrity, and color or power Doppler US can be used to assess neovascularization, inflammation, and healing. Diffuse and focal thickening with hypoechoic change is characteristic of chronic tendinopathy. Minor degrees of fibril disruption and partial tearing can be detected, as can fluid collections around the tendon (Fig 5). Contralateral evaluation may be useful for the inexperienced ultrasonographer.



a.



b.



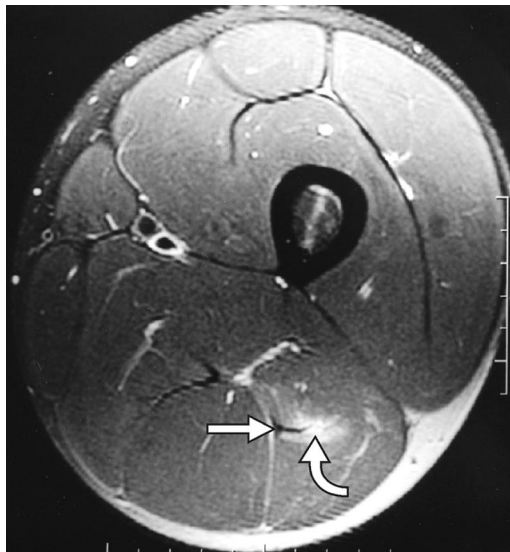
c.

Figure 6. MTJ strain in a 26-year-old professional football player who presented with recurring hamstring strains and prolonged rehabilitation periods. **(a, b)** Axial **(a)** and oblique coronal **(b)** MR images demonstrate hyperintensity (curved arrow) in the region of the MTJ of the biceps femoris muscle (long head) in keeping with myofibrillar disruption and retraction from the central tendon slip (straight arrow). Note the hyperintensity around the fascial sleeve. **(c)** Sagittal US image demonstrates an abnormality with mixed echogenicity that corresponds to the MR imaging findings. * = boundaries of the area of disruption.

Curiously, the semitendinosus tendon has been known to regenerate with histologically demonstrable tenocytes following harvesting for ACL reconstruction, although this may take up to 2 years. Awareness of this fact helps avoid potential confusion as to the donor site for the reconstructed ligament in the postoperative knee, since the semitendinosus appears normal at imaging (42–44).

Partial tearing of the HMC is often referred to as a strain. Most strains occur in the region of the

MTJ (7), which is the weakest link in the muscle complex (Fig 6). However, the MTJ is not a distinct area but a 10–12-cm zone of transition in which muscle fibrils intersect with the tendon origin or ligamentous insertion (1,11). The proximal MTJ is more commonly strained than the distal MTJ, with the biceps femoris disproportionately

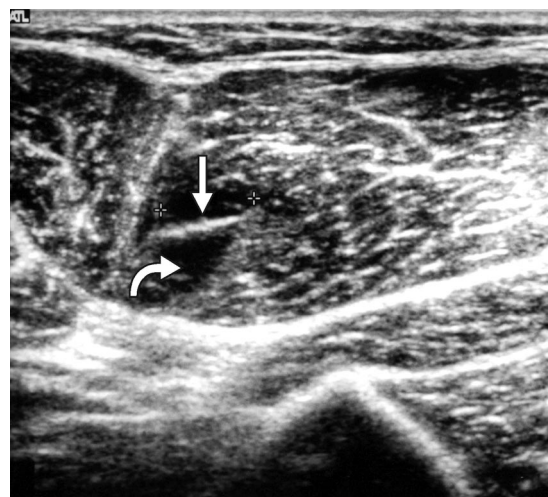


a.

Figure 7. Proximal MTJ strain in an elite athlete. Differentiation between muscle soreness following training and a small hamstring tear was difficult at clinical examination. (a, b) Axial (a) and coronal oblique (b) MR images demonstrate a small region of hyperintensity in the biceps femoris (curved arrow), a finding that is consistent with edema as a result of a subtle MTJ tear (straight arrow). (c) Corresponding transverse US image demonstrates a hypoechogenic area of edema (curved arrow) around the MTJ (straight arrow).



b.

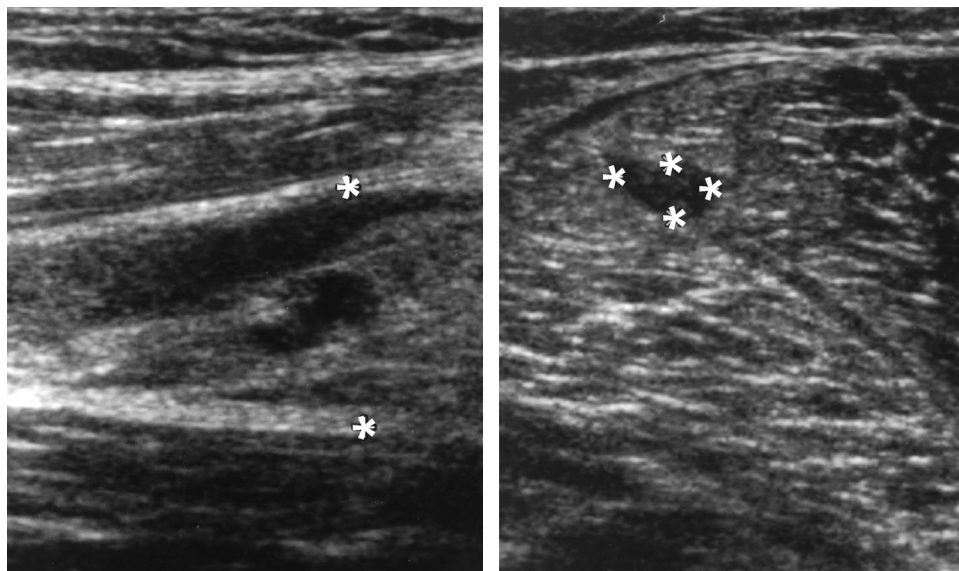


c.

represented (Fig 7) (7). Injury rates to the semimembranosus and semitendinosus vary. In the largest study to date on the prevalence of HMC strain, semimembranosus injury exceeded semitendinosus injury (10), a finding that has been supported by other studies (45–47). Other research, however, has shown the semitendinosus to be more frequently strained (7,48). The high signal intensity of edema, fluid, and blood products characteristically dissects along disrupted fibrils and lies between the fibrils of isointense but intact skeletal muscle near the MTJ, creating a feathered appearance (49,50). Such tears can be

subtle at MR imaging and even more so at US. The low echogenicity of muscle edema in a minor strain can be difficult to appreciate at US because it contrasts poorly with the low to intermediate echotexture of skeletal muscle. Nevertheless, US is a sensitive imaging modality in the presence of blood products and edema, which increase the conspicuity of muscle disruption (Fig 8).

With increasing degrees of muscle disruption, hemorrhage becomes more prevalent, involving a greater cross-sectional and longitudinal area.



a.

b.

Figure 8. Distal MTJ strain in an athlete with posterior thigh pain who had experienced pain during sprinting. Longitudinal (**a**) and transverse (**b**) US images demonstrate a focal area of retraction (*), a finding that is consistent with a macroscopic tear in the distal MTJ of the semitendinosus.

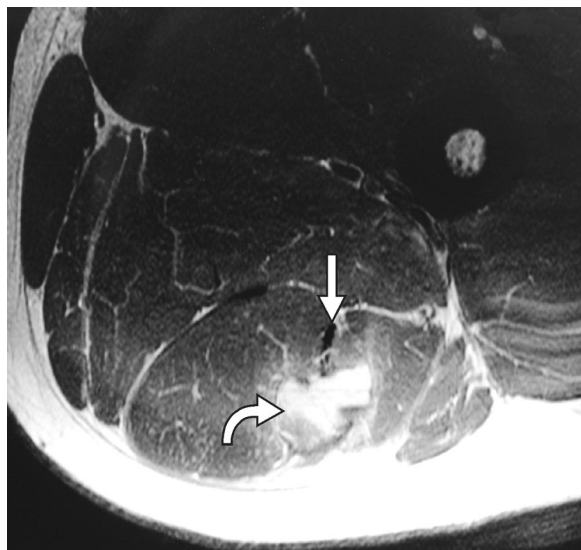
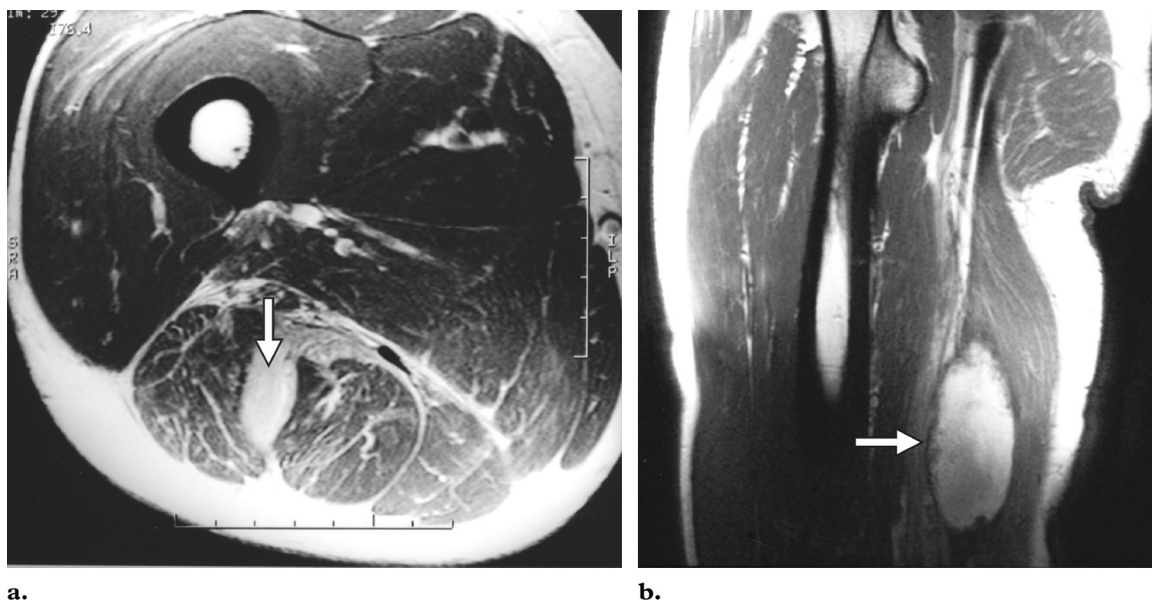


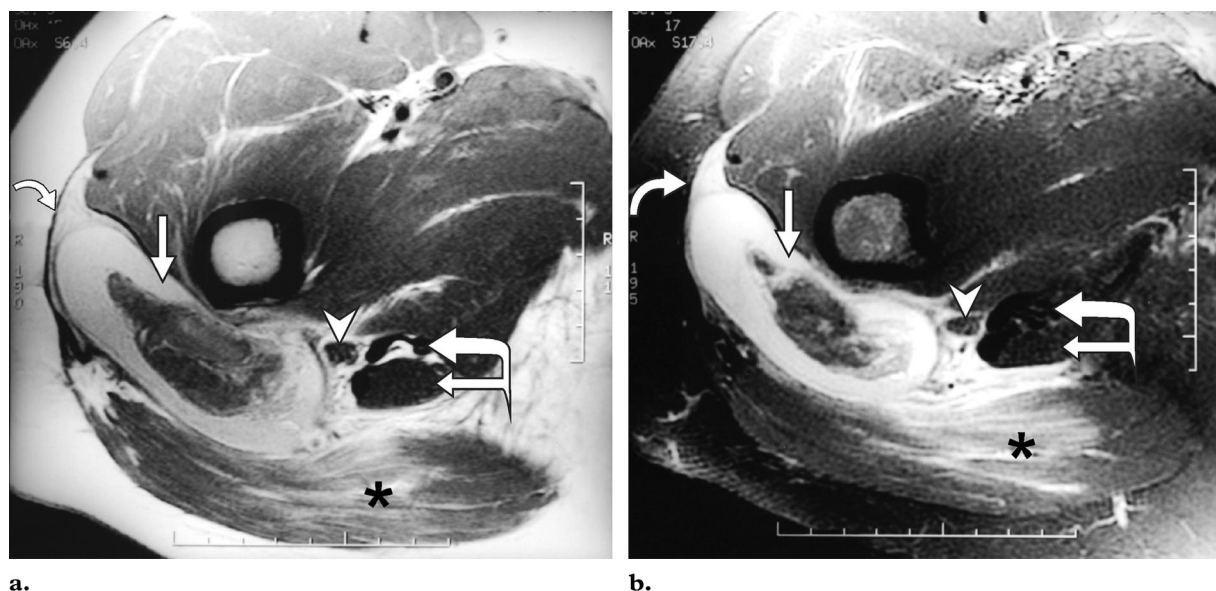
Figure 9. Epimysial fascial strain in an elite athlete with a hamstring strain and evidence of a visible ecchymosis. Axial MR image shows an area of abnormal hyperintensity in the biceps femoris muscle (curved arrow), with subtle fluid-fluid levels predominantly in a myofascial distribution. There is relatively little involvement near the tendon (straight arrow). Hematoma and tearing resulted in disruption of the most superficial aspect of the muscle at the fascial boundary, accounting for the clinical findings.

Hemorrhage is hyperintense in the acute setting and may track around the sciatic nerve. Injury also manifests at US as fluid gliding between the muscle planes as well as disruption and disorganization of the skeletal muscle architecture adjacent to the hyperechoic tendon. The appearance of hemorrhage varies at both MR imaging and US according to its age. Hematoma may predominate within the muscle or lie outside the epimysial covering between muscles. Intramuscular fluid-fluid levels may be seen.

Strains of the epimysial fascia (Fig 9) and within the muscle belly alone may also occur. Strain at the epimysial boundary is eccentric and is most commonly seen in the biceps femoris muscle proximal to where the short and long heads fuse. Such injuries are thought to occur due to the differential contraction of the two muscles, which contributes to decreased efficiency of overall muscle function. This applies an additional distracting force to the muscle bellies, thereby increasing susceptibility to tear. The second most common site of epimysial tearing is the posterior



a. **b.**
Figure 10. Muscle belly injury. Axial proton-density-weighted (**a**) and coronal (**b**) MR images show an intramuscular hematoma in the biceps femoris muscle (arrow). The central location of the hematoma is unusual.



a. **b.**
Figure 11. Hematoma in an aerial skier who presented with persistent posterior thigh pain and swelling with focal posterolateral thigh tenderness after suffering a fall during training. Axial proton-density-weighted MR images obtained without (**a**) and with (**b**) fat saturation demonstrate a large hematoma of varying intensity (straight arrow) within the fascia of the thigh (curved arrow). The hematoma is located deep to the gluteus maximus muscle, which also contains an area of high signal intensity (*), a finding that is consistent with a contusion. However, the collection is superficial to the proximal hamstring tendons (double arrow) and the sciatic nerve (arrowhead), both of which appear normal.

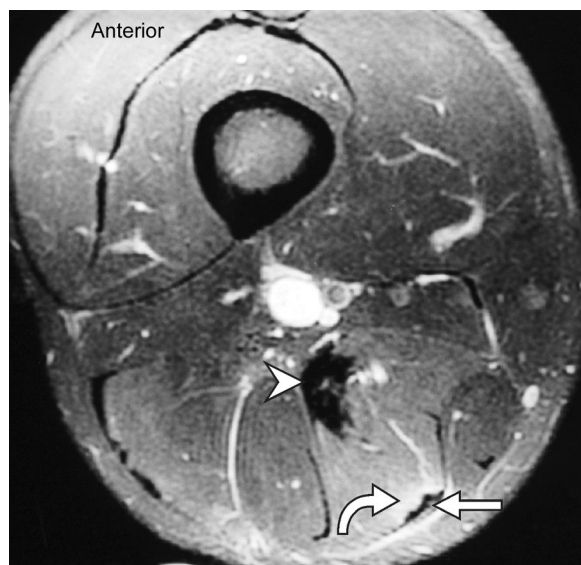


Figure 12. Return HMC in a 32-year-old football player with recurrent hamstring strain and a limited range of hip motion. MR image shows an area of hyperintensity (curved arrow) near the biceps femoris tendon (straight arrow), a finding that is consistent with an MTJ tear. However, an irregular area of low signal intensity deep within the muscle (arrowhead) is characteristic of scar tissue following a prior myofascial tear, which may have contributed to the decrease in hamstring flexibility, ultimately leading to tear. Recurrent tears at the site of prior scar tissue are uncommon owing to the greater strength of the fibrous tissue compared with the surrounding musculature.

boundary of the biceps femoral muscle proximal to the formation of the distal tendon slip.

Muscle belly injury can occur anywhere within the muscle. This is a rare injury whose pathogenesis is poorly understood. Hematomas arising from such injury usually remain localized within the deep substance of the muscle belly and are easily recognized (Fig 10). The signal intensity and echogenicity of hemorrhage from myofascial or muscle belly injuries are the same as those for MTJ injury. Blood products may irritate the musculature and cause spasm; therefore, the detection of hematoma may encourage the clinician to seek aspiration under US guidance.

Miscellaneous disease is also observed at imaging. Atrophy of the hamstring muscles with a decrease in muscle mass and fatty replacement is usually the result of a long-standing injury such as chronic tendon avulsions with retraction or recurrent strain in which there is disuse. Signal intensity characteristics typical of fat are demonstrated on T1-weighted MR images, findings that can be

confirmed with fat-suppression techniques. The corresponding US findings consist of a diffuse increase in the echogenicity of the muscle, with a decrease in muscle bulk and loss of the regular organization of muscle fibrils. Hematomas superficial to the muscle, which clinically may be confused with a strain (Fig 11), are also recognized at imaging. Such hematomas are easily localized as being separate from the muscle mass of the HMC and invariably have an excellent prognosis.

Chronic tears of the HMC can be investigated with MR imaging to evaluate scar tissue formation (Fig 12). This scar tissue has low signal intensity with all pulse sequences and is usually treated with a conservative stretching program. However, in a certain proportion of recalcitrant cases, surgical removal may be warranted. At US, areas of scar tissue have irregular morphologic features and display a heterogeneous echotexture. These are important sites to identify, since recurrent strain may occur near these regions where the normal contractility and mobility of the muscle is impaired due to shortening and tethering (33). The neurovascular bundle should also be routinely assessed because chronic injury may cause tethering of the sciatic nerve (51,52).

Clinical Correlation with Imaging Findings

Recent research has focused on correlating radiologic and clinical findings in HMC disease, particularly strain injury (48,52–54). MR imaging has traditionally served as an objective standard for confirming the presence of injury (53). Risk factors for HMC strain include increasing age, a prior history of posterior thigh pain (hamstring strain and back-related referred pain), knee injury, and osteitis pubis (55).

A clear association between the size of a tear (length and volume as determined with MR imaging) and the number of days lost from competition has been reported (56). HMC strain exceeding 50% of the cross-sectional area was associated with a longer rehabilitation time; indeed, all such athletes sustained a return HMC within 2 years. Understandably, higher pain scores were also associated with an increased area of abnormal signal intensity at MR imaging, reflecting significant muscular disruption and thus a prolonged recovery time (56).

Athletes in whom there is no documented evidence of a tear at MR imaging but in whom clinical findings create a strong suspicion for an HMC strain have a better prognosis than those with a detectable abnormality at MR imaging (48). By extrapolating from this data, one can deduce that a small tear carries a better prognosis. No demonstrable tear would certainly be the most favorable scenario. It is possible that athletes actually sustain HMC strain beyond the resolution capability of MR imaging. An alternative diagnosis such as back-related referred pain, spasm, or severe muscle soreness following exercise may have accounted for the complaint of posterior thigh pain that led to imaging in the first place. More than one muscle injured at the time of imaging has been reported (7,10,47).

No prognostic significance has been attributed to either the location of the injury (proximal distal)—regardless of which muscle was involved—or the type of tear (musculotendinous or myofascial) (54). Intermuscular fluid collections, an indirect sign of injury usually resulting from an epimysial tear, correlate weakly with a delayed return to competition. Although injury to the lower third of the HMC is less painful than injury to the upper third (48), the convalescent period is no different. It seems plausible that although the volume of muscle injured is smaller (thus producing less impressive clinical symptoms) in the former scenario, function remains equally poor. Of note is the fact that MR imaging helped confirm the presence of HMC strain in 9% of patients with an atypical history or normal clinical examination. Over a playing season, this figure would represent quite a large number of athletes. The potential risk of reinjury in this setting would be enormous and possibly deleterious to an athlete's success.

The efficacy of US has recently been compared with that of MR imaging (54). Discordance between US and MR imaging findings occurred when injury was subtle, manifesting as edema and hemorrhage without macroscopic myofibril disruption and retraction. When the latter were present, both imaging modalities allowed identification of an HMC strain. It is thought that the more deeply located MTJ is best seen at MR imaging, with its superior contrast resolution. US, on the other hand, can readily delineate epimysial tears, which tend to be located more superficially.

At the time of initial injury, US assessment has a sensitivity equal to that of MR imaging in the depiction of muscle tears. Because US is extremely sensitive in the depiction of fluid collections, this is not surprising. However, as the fluid collections resolve (usually within 2 weeks), depicting a myofibrillar abnormality becomes more difficult with US, although not with MR imaging. This observation may be representative of several processes: the reparative-inflammatory response, persistent (possibly progressing to permanent) abnormality, or even microrecurrence or extension of injury as athletes became progressively more active. Interestingly, the HMC strain reinjury rate in football players is on the order of 30% within 3 months, with the peak prevalence occurring 3–4 weeks following injury (56).

Conclusions

Increasing activity in the general population and the high demands placed on athletes have resulted in injuries to the HMC being commonplace in sports. In turn, as imaging matures and becomes more accessible, the hamstring muscles are increasingly scrutinized. A wide spectrum of hamstring injuries is reflected in the varied appearances of injury at imaging. MR imaging and US are the imaging modalities of choice. Experience, in combination with a knowledge of the HMC anatomy, will assist the musculoskeletal radiologist in making an accurate and useful contribution to the treatment of athletes at all levels of participation. Recent research has correlated imaging abnormalities with clinical findings. The musculoskeletal radiologist must be alert to the significance of certain imaging parameters and findings, especially those relating to prognosis.

References

1. Garrett WE Jr, Rich FR, Nikolaou PK, Vogler JB III. Computed tomography of hamstring muscle strains. *Med Sci Sports Exerc* 1989; 21:506–514.
2. Garrett WE Jr, Califf JC, Bassett FH III. Histological correlates of hamstring injuries. *Am J Sports Med* 1984; 12:98–103.
3. Sallay PI, Friedman RL, Coogan PG, Garrett WE Jr. Hamstring muscle injuries among water skiers: functional outcome and prevention. *Am J Sports Med* 1996; 24:130–136.
4. Kurosawa H, Nakasita K, Nakasita H, Sakasi S, Takeda S. Complete avulsion of the hamstring tendons from the ischial tuberosity: a report of two cases sustained in judo. *Br J Sports Med* 1996; 30:72–74.

5. McMinn RMH, ed. Last's anatomy, regional and applied. Edinburgh, Scotland: Churchill Livingstone, 1990.
6. William PL, Warwick R, Dyson M, Bannister LH, eds. Gray's anatomy. Edinburgh, Scotland: Churchill Livingstone, 1989.
7. De Smet AA, Best TM. MR imaging of the distribution and location of acute hamstring injuries in athletes. *AJR Am J Roentgenol* 2000; 174:393–399.
8. Sutton G. Hamstrung by hamstring strains: a review of the literature. *J Orthop Sports Phys Ther* 1984; 5:184–195.
9. Burkett LN. Investigation into hamstring strains: the case of the hybrid muscle. *J Sports Med* 1976; 5:228–231.
10. Koulouris G, Connell D. Evaluation of the hamstring muscle complex following acute injury. *Skeletal Radiol* 2003; 32:582–589.
11. Markee JE, Logue JT, Williams M, Stanton WB, Wrenn RN, Walker B. Two-joint muscles of the thigh. *J Bone Joint Surg Am* 1955; 37-A(1):125–142.
12. Beltran J, Matityahu A, Hwang K, et al. The distal semimembranosus complex: normal MR anatomy, variants, biomechanics and pathology. *Skeletal Radiol* 2003; 32:435–445.
13. Rothstein CP, Laorr A, Helms CA, Tirman PF. Semimembranosus-tibial collateral ligament bursitis: MR imaging findings. *AJR Am J Roentgenol* 1996; 166:875–877.
14. Hennigan SP, Schneck CD, Mesgarzadeh M, Clancy M. The semimembranosus-tibial collateral ligament bursa: anatomical study and magnetic resonance imaging. *J Bone Joint Surg Am* 1994; 76:1322–1327.
15. Kim YC, Yoo WK, Chung IH, Seo JS, Tanka S. Tendinous insertion of semimembranosus muscle into the lateral meniscus. *Surg Radiol Anat* 1997; 19:365–369.
16. Stoane JM, Gordon DH. MRI of an accessory semimembranosus muscle. *J Comput Assist Tomogr* 1995; 19:161–162.
17. Cattysse E, Barbaix E, Janssens V, Alewaeters K, Van Roy P, Clarijs JP. Observation of a supernumerary hamstring muscle: a state of the art on its incidence and clinical relevance. *Morphologie* 2002; 86:17–21.
18. Peterson JE, Currarino G. Unilateral absence of thigh muscles confirmed by CT scan. *Pediatr Radiol* 1981; 11:157–159.
19. Slocum DB, James SL. Biomechanics of running. *JAMA* 1968; 205:721–728.
20. Cooper DL, Fair J. Trainer's corner: hamstring strains. *Phys Sports Med* 1978; 8:104.
21. Safran MR, Seaber AV, Garrett WE Jr. Warm-up and muscular injury prevention: an update. *Sports Med* 1989; 8:239–249.
22. Shellock FG, Fukunaga T, Mink JH, Edgerton VR. Exertional muscle injury: evaluation of concentric versus eccentric actions with serial MR imaging. *Radiology* 1991; 179:659–664.
23. Garrett WE Jr. Muscle strain injuries. *Am J Sports Med* 1996; 24(6 suppl):S2–S8.
24. Rubin SJ, Feldman F, Staron RB, Zwass A, Totterman S, Meyers SP. Magnetic resonance imaging of muscle injury. *Clin Imaging* 1995; 19:263–269.
25. Burkett LN. Causative factors in hamstring strains. *Med Sci Sports* 1970; 2:39–42.
26. Kujala UM, Orava S, Jarvinen M. Hamstring injuries: current trends in treatment and prevention. *Sports Med* 1997; 23:397–404.
27. Heiser TM, Weber J, Sullivan G, Clare P, Jacobs RR. Prophylaxis and management of hamstring injuries in intercollegiate football players. *Am J Sports Med* 1984; 12:368–370.
28. Garrett WE Jr. Muscle strain injuries: clinical and basic aspects. *Med Sci Sports Exerc* 1990; 22: 436–443.
29. El-Khoury GY, Brandser EA, Kathol MH, Tearse DS, Callaghan JJ. Imaging of muscle injuries. *Skeletal Radiol* 1996; 25:3–11.
30. Friden J, Sjostrom M, Ekblom B. A morphological study of delayed muscle soreness. *Experientia* 1981; 37:506–507.
31. Fleckenstein JL, Weatherall PT, Parkey RW, Payne JA, Peshock RM. Sports-related muscle injuries: evaluation with MR imaging. *Radiology* 1989; 172:793–798.
32. Palmer WE, Kuong SJ, Elmadbouh HM. MR imaging of myotendinous strain: pictorial essay. *AJR Am J Roentgenol* 1999; 173:703–709.
33. Nikolaou PK, Macdonald BL, Glisson RR, Seaber AV, Garrett WE Jr. Biomechanical and histological evaluation of muscle after controlled strain injury. *Am J Sports Med* 1987; 15:9–14.
34. Greco A, McNamara MT, Escher RM, Trifillio G, Parienti J. Spin-echo and STIR MR imaging of sports-related muscle injuries at 1.5 T. *J Comput Assist Tomogr* 1991; 15:994–999.
35. Jonhagen S, Nemeth G, Eriksson E. Hamstring injuries in sprinters: the role of concentric and eccentric hamstring muscle strength and flexibility. *Am J Sports Med* 1994; 22:262–266.
36. Friden J, Sjostrom M, Ekblom B. Myofibrillar damage following intense eccentric exercise in man. *Int J Sports Med* 1983; 4:170–176.
37. Brandser EA, el-Khoury GY, Kathol MH, Callaghan JJ, Tearse DS. Hamstring injuries: radiographic, conventional tomographic, CT, and MR imaging characteristics. *Radiology* 1995; 197:257–262.
38. Alioto RJ, Browne JE, Barnhouse CD, Scott AR. Complete rupture of the distal semimembranosus complex in a professional athlete. *Clin Orthop* 1997; 336:162–165.
39. Varela JR, Rodriguez, Soler R, Gonzalez J, Pombo S. Complete rupture of the distal semimembranosus tendon with secondary hamstring muscles atrophy: MR findings in two cases. *Skeletal Radiol* 2000; 29:362–364.
40. Orava S, Kujala UM. Rupture of the ischial origin of the hamstring muscles. *Am J Sports Med* 1995; 23:702–705.

41. Yao L, Lee JK. Avulsions of the posteromedial tibial plateau by the semimembranosus tendon: diagnosis with MR imaging. *Radiology* 1989; 172: 513–514.
42. Cross MJ, Roher R, Kujawa P, Anderson IF. Regeneration of the semitendinosus and gracilis tendons following their transection for repair of the anterior cruciate ligament. *Am J Sports Med* 1992; 20:221–223.
43. Eriksson K, Larsson H, Wredmark T, Hamberg P. Semitendinosus tendon regeneration after harvesting for ACL reconstruction: a prospective MRI study. *Knee Surg Sports Traumatol Arthrosc* 1999; 7:220–225.
44. Ferretti A, Conteduca F, Morelli F, Masi V. Regeneration of the semitendinosus tendon after its use in anterior cruciate ligament reconstruction; a histologic study of three cases. *Am J Sports Med* 2002; 30:204–207.
45. Bencardino JT, Rosenberg ZS, Brown RR, Hassankhani A, Lustrin ES, Beltran J. Traumatic musculotendinous injuries of the knee: diagnosis with MR imaging. *RadioGraphics* 2000; 20:S103–S120.
46. Pomeranz SJ, Heidt RS. MR imaging in the prognostication of hamstring injury: work in progress. *Radiology* 1993; 189:897–900.
47. Speer KP, Lohnes J, Garrett WE Jr. Radiographic imaging of muscle strain injury. *Am J Sports Med* 1993; 21:89–96.
48. Verrall GM, Slavotinek JP, Barnes PG, Fon GT. Diagnostic and prognostic value of clinical findings in 83 athletes with posterior thigh injury: comparison of clinical findings with magnetic resonance imaging documentation of hamstring muscle strain. *Am J Sports Med* 2003; 31:969–973.
49. De Smet AA, Fischer DR, Heiner JP, Keene JS. Magnetic resonance imaging of muscle tears. *Skeletal Radiol* 1990; 19:283–286.
50. De Smet AA. Magnetic resonance findings in skeletal muscle tears. *Skeletal Radiol* 1993; 22:479–484.
51. Topsakal C, Akdemir L, Tiftikci M, Ozercan I, Aydin Y. Malignant schwannoma of the sciatic nerve originating in a spinal plexiform neurofibroma associated with neurofibromatosis type 1: case report. *Neurol Med Chir (Tokyo)* 2001; 41: 551–555.
52. Slavotinek JP, Verrall GM, Fon GT. Hamstring injury in athletes: using MR imaging measurements to compare extent of muscle injury with amount of time lost from competition. *AJR Am J Roentgenol* 2002; 179:1621–1628.
53. Verrall GM, Slavotinek JP, Barnes PG, Fon GT, Spriggins AJ. Clinical risk factors for hamstring muscle strain injury: a prospective correlation of injury by magnetic resonance imaging. *Br J Sports Med* 2001; 35:435–440.
54. Connell DA, Schneider-Kolsky ME, Hoving JL, et al. Longitudinal study comparing sonographic and MRI assessments of acute and healing hamstring injuries. *AJR Am J Roentgenol* 2004; 183:975–984.
55. Orchard JW. Intrinsic and extrinsic risk factors for muscle strains in Australian football. *Am J Sports Med* 2001; 29:300–303.
56. Verrall G. Hamstring injuries: safe return to play from MR studies. Presented at the Australasian College of Sports Physicians Annual Scientific Meeting (incorporating Football Australasia Conference—controversies in 2003), Melbourne, Australia, September 22–25, 2003.