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George Koulouris
David Connell

Imaging of hamstring injuries: therapeutic implications

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G. Koulouris
Division of Musculoskeletal Imaging and General Diagnostic Imaging, Thomas Jefferson University Hospital, Philadelphia, Pennsylvania, USA

D. Connell (✉)
Royal National Orthopaedic Hospital, Brockley Hill, Stanmore, Middlesex, HA7 4LP, UK
e-mail: david.connell@rnoh.nhs.uk

Abstract Though recent research into the diagnosis and management of hamstring disorders has resulted in early and accurate recognition of injury, hamstring strain remains the most common form of muscle injury in the active population. With prompt recognition of hamstring strain, an appropriate rest and rehabilitation routine may be devised by the sports clinician in the hope of avoiding future and possibly more debilitating injury. As such, imaging has played a pivotal role in assisting athletes, both elite and recreational, in returning to activity expeditiously.

Keywords Magnetic resonance (MR) imaging · Ultrasound · Hamstring muscle complex (HMC) · Strain · Avulsion · Enthesopathy · Myositis ossificans

Introduction

Hamstring muscle strain remains a significant contributor to athletic morbidity, resulting in a significant amount of time out of competition, with no sporting activity immune [1–3]. Since the hamstring muscle's primary role is that of locomotion, as opposed to postural control, it is utilised for intense bursts of speed. Therefore, for successful sporting activity, the hamstring muscle complex (HMC) must contract forcefully and repeatedly, a factor heavily dependent on the fitness of the individual. As the demands of sport often exceed those of which many are physically capable, it is not surprising that muscles such as the HMC eventually fail. Failure of muscle ultimately results in disruption, as the forces transmitted through the muscle can no longer be absorbed or dissipated effectively and the tensile properties of individual myofibrils is overcome. Muscle failure, or strain, represents a severity of injury spectrum that ranges widely in the clinical manifestations, imaging features and management. Despite the fact that muscle tissue has little regenerative capacity, most hamstring muscle injury has a favourable prognosis, as most strains are partial and muscle possesses excellent healing properties through its high vascularity and ability to form strong granulation (scar) tissue. Hence, most strains are effectively managed conservatively. Surgical repair is reserved for avulsion injury [4, 5], particularly if displaced [6], which most commonly occurs proximally [7]. Distal avulsion is a rare injury [8]. Surgery may also be reserved for recalcitrant myositis ossificans or recurrent/extensive partial strain. Occasionally, radiological intervention may be sought in an effort to expedite the healing process when surgery is considered to be unwarranted.

The mainstay of radiological investigation of hamstring strain is with either magnetic resonance (MR) imaging or ultrasound (sonography), where injury manifests itself as either alteration in muscle signal intensity [usually T2, short-tau inversion recovery (STIR) hyperintense] or echotexture, respectively [9]. In certain circumstances (avulsion fractures and myositis ossificans), plain radiographs (and, less commonly, computed tomography) may be indicated.

Basic science concepts

Correct radiological interpretation of HMC strain requires precise identification of the muscle involved and localisa-

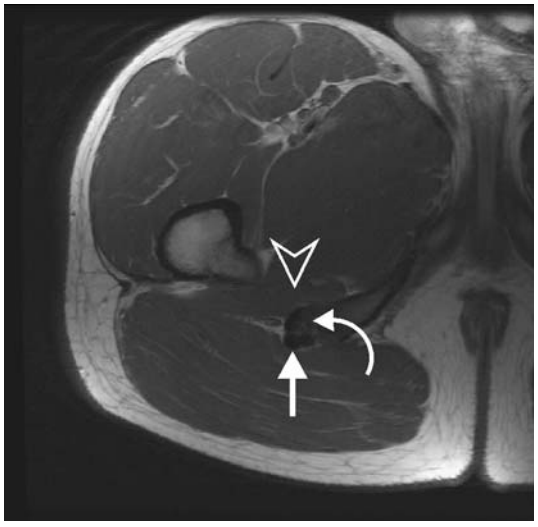

Fig. 1 Proton-density axial images through a normal proximal origin of the right HMC. The larger tendon origin anteriorly is the semimembranosus (*curved arrow*) and posteriorly, the conjoint tendon (*straight arrow*). The muscle fibres crossing the semimembranosus origin anteriorly are those of the adductor magnus (*arrowhead*)

tion to the portion of the muscle–tendon–bone unit. The HMC lies within the posterior compartment of the thigh, with the muscles arising from the ischial tuberosity crossing both the hip and knee joint (bi-articular). Only the short head of biceps femoris is a mono-articular muscle.

The classic and accepted relationship of the hamstring muscles is that of the biceps femoris lying lateral to the semitendinosus and semimembranosus, with the former superficial to the latter. This configuration is not the case proximally. The origin of the HMC is the ischial tuberosity, which possesses four facets, three bearing a ligamentous attachment. Inferomedially is the facet for the common origin of the long head of biceps femoris and semitendinosus, the *conjoint tendon*. Most of the semitendinosus arises from the ischial tuberosity directly as a muscle belly, with the tendon it shares with the biceps lying laterally (Figs. 1 and 2). Semitendinosus rapidly increases in size, accounting for most of the muscle bulk of the proximal third of the HMC. Inferiorly, just proximal to the inferiormost fibres of gluteus maximus inserting onto the tensor fascia lata, the biceps femoris muscle fibres start to form (Fig. 3).

The semimembranosus tendon arises from the superolateral facet of the ischial tuberosity and is approximately twice the size of the conjoint tendon. It shares an inverse relationship with the semitendinosus and so is largely only a tendon from the ischial tuberosity and for quite some length distally. It flattens and then gives rise to a small flat muscle belly with a characteristic triangular shape (Fig. 4). Infero-laterally arises the adductor magnus muscle, which borders the posterior and medial (adductor) compartments of the thigh and is, in essence, a transitional muscle. The adductor magnus is usually not considered a true hamstring, reflected in its low incidence of injury. It borders the medial (adductor) and posterior (flexor) compartments of the thigh, which are not separated by a distinct fascial boundary.

The short head of biceps femoris arises from the linea aspera, fusing distally with the more posteriorly (superficially) positioned long head. An extensive attachment to the linea aspera has been theorised to limit the mobility of biceps and account for the occurrence of strain [10]. Its innervation by the common peroneal division of the sciatic

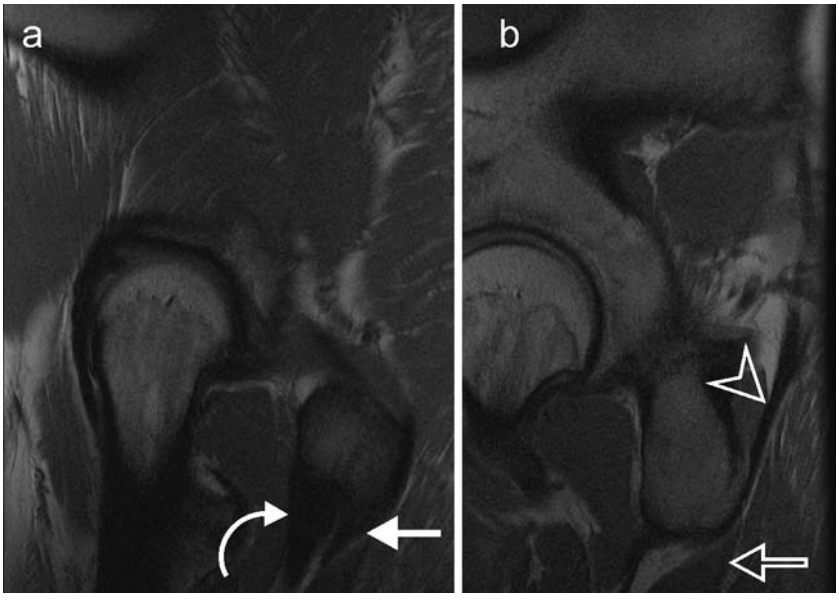

Fig. 2 Forty five-degree oblique sagittal proton-density images through the right ischial tuberosity. **a** The larger semimembranosus tendon (*curved arrow*) lies anterior to the conjoint (*straight arrow*). **b** The conjoint tendon continues superiorly as the ischiotuberous ligament (*open arrowhead*). Note the muscular origin of the semitendinosus (*open arrow*)

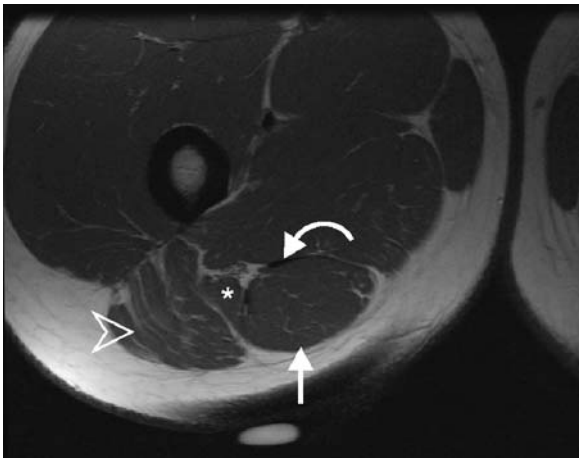

Fig. 3 At the inferior-most aspect of the right gluteus maximus (*open arrowhead*), the biceps femoris starts to emerge (*asterisk*), an important landmark. The semimembranosus tendon at this point has flattened (*arrowhead*), and the semitendinosus (*straight arrow*) forms most of the bulk of the HMC muscle mass at this point. Note the large adductor magnus muscle anterior to the semimembranosus tendon and the lack of a clear plane between the medial and posterior compartment of the thigh

nerve may account for the high frequency of strain within the biceps femoris muscle due to asynchrony in either the timing and/or intensity of the contraction of both heads. The biceps femoris finally inserts onto the fibular head, the semitendinosus onto the anterior tibia (*pes anserinus*) and the semimembranosus into the medial structures of the knee as a complex of five tendinous insertions that become intimately blended with a capsular thickening of the knee joint, the posterior oblique ligament [11].

Most hamstring strain occurs in muscles where the forces that the muscle attempts to absorb exceed those that it is able to be generate and thus attempt to counteract. The

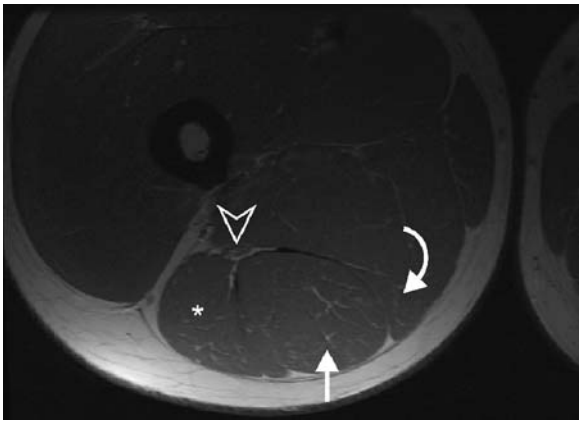

Fig. 4 All three muscle bellies are now demonstrated. The flattened tendon of the semimembranosus has given rise to a triangular shaped muscle (*curved arrow*), whose base points medially. The conjoint tendon remains a single structure, with the long head of biceps femoris (*asterisk*) and semitendinosus (*straight arrow*) on either side. Note the position of the sciatic nerve (*open arrowhead*)

muscles of the HMC act principally as agonists of the anterior cruciate ligament; they prevent anterior tibial translation during walking. As the nearly extended knee contacts the ground, the quadriceps contracts, which otherwise unopposed, would pull the tibia anteriorly via the forces imparted when contracting the instant the foot contacts the ground (and the knee is almost fully extended [12]). To counteract this, the HMC contracts *eccentrically*, that is, its muscles contract whilst being lengthened. It is this additional force (that is, being stretched whilst contracting) that is thought to be a principal cause for the high HMC strain rate [13]. Recently, athletic training has focused on strengthening the HMC and training it under the same condition it fails, that is, with eccentric overloading exercises [14]. Such exercises simulate match conditions when the HMC is at most stress [15, 16] and when most strains occur [17]. This has been shown to safely increase the HMC strength, with a decrease in injury rates and improved performance [18, 19]. The high percentage of fast twitch (type II) fibres in the HMC [20] has also been implicated as a risk factor, as the muscles can generate forces rapidly, which, when also simultaneously stretched, may result in failure [21, 22].

The HMC has a dual role, acting as either a hip extensor or knee flexor. Since the muscles cross two joints, they can only localise their contraction to one joint if either of its antagonist contracts so movement can occur at the other. As the antagonists (quadriceps and iliopsoas) are exceedingly larger than the HMC, it is likely that this strength imbalance is a factor in injury. Rapidly switching from acting on one joint and then to the other is also a likely aetiological factor. The biceps femoris is known to be stretched more than the other two hamstring muscles [23], despite the fact that the muscle possesses two double attachments: superiorly with the semitendinosus and inferiorly with the short head of biceps. This increased stretch during the gait cycle with double attachments may be a predisposing factor.

Causes of HMC strain can be divided into *extrinsic* (related to the activity, or sport-specific) or *intrinsic* (individual athlete factors) [19, 24]. Intrinsic factors include "pathological" muscle contraction, that is, contraction which is less effective and hence weaker, due to fatigue [25], prior injury [13] or impaired by scar tissue [19, 24]. Ultimately, pathological muscle contraction renders the HMC muscles unable to absorb forces effectively, particularly eccentrically, and as such they are at increased risk of strain. Hence, initial injury prevention is paramount in order to avoid a re-injury cycle. Poor flexibility [26], inherent muscle weakness [1], poor core muscle and truncal stability [27], and a past history of anterior cruciate ligament (ACL) repair with use of hamstring tendon graft, independent of tendon regeneration [28, 29], are other well-known risk factors. Adverse neuromeningeal tension, and multiple biomechanical and postural hypotheses have also been implicated.

Imaging concepts

Armed with a detailed knowledge of the functional anatomy of the HMC, a thorough understanding of the imaging manifestations of injury utilising the various imaging modalities available is necessary [30]. The importance of imaging has increased, as not all causes of posterior thigh pain are the result of HMC strain [31]. It is likely that previously recurrent HMC strain may have been clinically misdiagnosed, or, if diagnosed correctly, underestimated with respect to the extent of myofibrillar damage. It is here that imaging is anticipated to provide the clinician with valuable information with respect to pathology, and, when present, precise anatomical localisation. As such, it can be anticipated that the morbidity of HMC strain will be minimised; players otherwise thought to have a strain but subsequently found to have normal imaging may be allowed to return to competition with confidence. Those with minimal or confusing clinical findings, who may have returned to competition, can be now be excluded when imaging confirms an injury. It is important to appreciate that the radiological features vary with the timing of imaging following injury, reflecting the dynamic process of healing, and is one with which the radiologist must be familiarised.

Strain

Strains can range from microscopic foci of myofibrillar disruption, which may be beyond the resolution of imaging, to large areas of separation of fibres, with areas of intra-muscular haematoma formation, just short of avulsion injury. Strain injury occurs consistently in a predictable location within the muscle unit. Histopathologically, this occurs just proximal to the musculotendinous (myotendinous) junction (MTJ) [32–34], though, from a macroscopic (and hence radiological) perspective, the injury is essentially at this junction. The injury occurs irrespective of the type of muscle structure [35]. If the disruption is severe enough, haemorrhage occurs, which may be restricted to the muscle belly or extend beyond the muscle perimeter, into the subcutaneous tissues, extending into the subcutaneous tissue layer [36] and/or encasing other structures, such as the adjacent sciatic nerve [7], to result in neural irritation.

The earliest manifestation of muscle injury is following unaccustomed exercise, known as delayed onset of muscle soreness (DOMS) [21]. This results in an appearance similar to that of minor muscle strain injury only on MR imaging; however, it is reliably differentiated from the latter clinically. With DOMS, muscle soreness occurs *after* exercise, is usually noticed the following day, and peaks in severity on day 2. In contrast, most muscle strains occur at the precise moment at which the athlete can recall immediate cessation of activity.

The characteristic features of muscle strain are demonstrated elegantly with either MR imaging or ultrasound. On MR imaging, the small foci of microscopic haemorrhage at the MTJ region incite an inflammatory reaction as part of the healing response, or oedema, resulting in an ill-defined area of T2, proton-density (PD) fat-saturated or STIR hyperintensity. The muscle fibres contrast against this hyperintense background, giving an alternating low and high signal pattern, the characteristic feathered appearance when imaged in the sagittal or coronal plane (Figs. 5 and 6). T1-weighted imaging is less sensitive for depicting muscle injury and is rarely used in the clinical setting. T1 images are best utilised to demonstrate areas of hyper- or hypointensity if haemorrhage has occurred, which is age

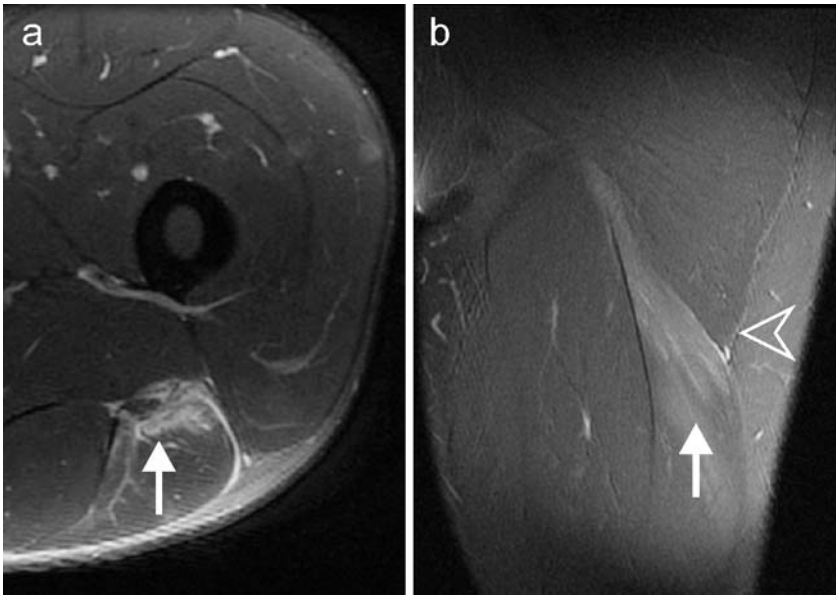

Fig. 5 Proton-density fat-saturated sequences in the **a** axial and **b** coronal plane of the proximal left HMC. The hypointense line of the conjoint tendon separates the two muscle to which it gives origin, the semitendinosus medially and biceps femoris laterally. The characteristic feathered appearance of a musculotendinous junction strain injury to the biceps (*straight arrow*) is typical. Note that the gluteus maximus insertion onto the ilio-tibial band (*open arrowhead*) serves as a guide as to where to expect some reasonable long head of biceps femoris muscle bulk to start appearing

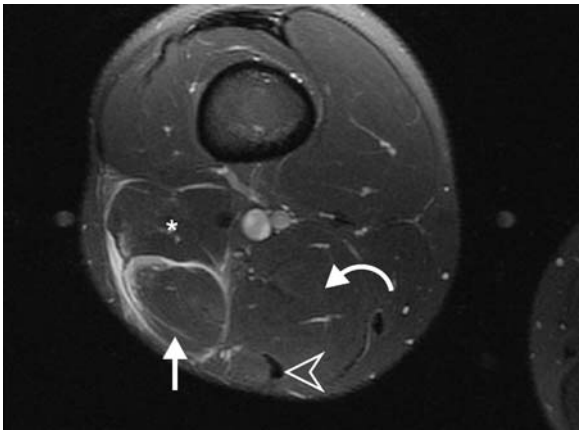

Fig. 6 Axial proton-density fat-saturated images through the distal right leg. The semitendinosus has little muscle bulk, now mainly composed of tendon (*open arrowhead*) and lies superficially to the semimembranosus (*curved arrow*). A tear of the long head of the distal biceps femoris muscle (*straight arrow*) is demonstrated with epimysial extension, clearly outlining the plane between the long and (at this level) larger short head (*asterisk*)

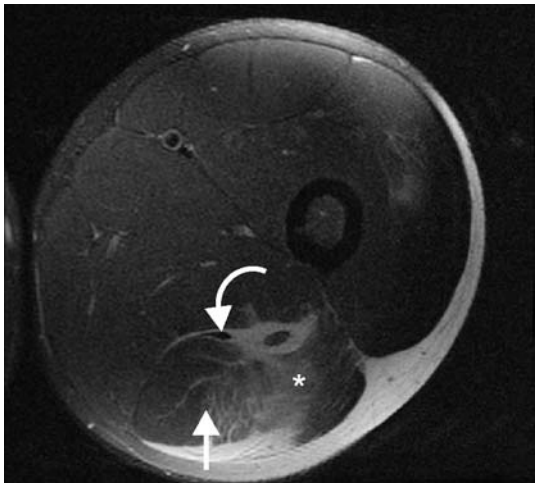

Fig. 7 Axial proton-density fat-saturated MR images through the left leg, demonstrating the most common tandem strain injury, that of the semitendinosus (*straight arrow*) and long head of biceps femoris (*asterisk*). The centrally placed conjoint tendon is indistinct due to the hyperintensity of the strain. The semimembranosus remains a tendon at this point (*curved arrow*)

dependent [37]. The same area of oedema is demonstrated with ultrasound. Muscle fibres form a regular pattern of alternating hypo- and hyperechogenicity. The oedema of a minor strain is visualised as an area of hypo-echogenicity, which results in increased prominence of the hyperechoic tendon, and, similarly, the probe, when rotated in the correct plane, will result in a feathered appearance. Basic concepts with respect to hamstring muscle imaging are as follows. Most injuries are mild strains, resulting in oedema and, hence, the characteristic feathered appearance. Most of these occur at the musculotendinous junction of the long head of biceps femoris [7, 38–40] muscle proximally. If more than one muscle is involved, it is usually the long head of biceps and the semitendinosus muscle (Fig. 7). Semimembranosus injury is rare.

Increased muscle disruption results in larger dimensions of abnormality on MR imaging and ultrasound. Associated with increasing volume of injury is the increased probability of visualising actual fibre disruption. On MR imaging, the normal low signal of the muscle fibres becomes disrupted and disorganised, with a wavy pattern the result. Associated haemorrhage results in obvious focal fluid collections that separate muscle fibres and are of varying signal intensity. This disruption is seen sonographically as an interruption of the normal monotonous muscle structure, which often may be correlated with the point of maximal discomfort. Often, a haematoma may be subcutaneous and clinically evident, which is, classically, *inferior* to the site of tear, due to the effects of gravity. The haematoma, as with MR imaging, is of mixed echo-texture, usually hyperechoic when acute and, with time, becomes hypo-echoic, which, if it persists, forms an anechoic seroma. In the hyperacute stage, the area may be hypoechoic as the collection has yet to coagulate.

Most haematomas are small and have invariably resolved by the time the athlete is deemed suitable for competition. This is always the case with intermuscular haematomas, which resolve quickly and have never required intervention, according to our experience. This type of haematoma does not form a mass-like collection, instead, dissipating rapidly between muscle, fascia and fat. Given its large surface area, the haemorrhage is absorbed quickly. On the other hand, intramuscular haematomas are slower to resolve and may require intervention. A persistent intramuscular haematoma may impair the healing process, acting as a chemical irritant to muscle to cause spasm and even result in reflex inhibition of normal muscle contraction and, thus, atrophy, further prolonging time out of competition. Occasionally, haemorrhage may be severe enough to warrant intervention, particularly in the athletic setting. Though evidence for this does not exist, it is felt that the draining of a large haematoma is beneficial, hastening recovery. Ultrasound is the ideal modality in this instance, as it is perfectly placed to dynamically visualise the haematoma whilst a drainage catheter is temporarily placed to remove the collection [41]. Our practice is to wait for the haematoma to liquefy, so that it can be aspirated with ease, and hence maximise the amount that is able to be aspirated. This also minimises the small, but real, risk of infection and thus possible abscess formation. Rarely can a haematoma be so extensive as to result in an acute compartment syndrome necessitating surgery [42].

By the seventh day following injury, fibrosis and scar tissue formation is well underway and predominates histologically [43, 44]. This is not usually seen radiologically until some time after this date, until the scar tissue has become more mature and larger so that it is macroscopi-

cally visible. On MR imaging, scar tissue is of low signal intensity, and, since it forms at the site of injury, is seen as areas of nodularity adjacent to, or actually on, the normal low signal of the tendon [36]. This may be confused with an area of focal, discrete, tendon disruption, with associated retraction. The appearances are such that a small focus of the tendon is retracted upon itself to result in an identical radiological finding of tendon nodularity secondary to scar formation. Both may result in recurrent strain injury. Future research may provide a greater role for the radiologist, such as directly injecting antifibrotic agents into the focus to prevent exuberant scar tissue formation [45], though at this early stage, this procedure must be tempered with the risk of a weakened muscle. Further research is clearly required.

Imaging prognostic indicators

The dimensions of a strain are paramount in determining the period of convalescence. The cross-sectional area has been a lesser-used parameter, as the longitudinal length is quicker, easier to measure, and is more reproducible. Nevertheless, a strain injury involving more than 50% of the cross-sectional area correlates well with a convalescence period of greater than 6 weeks [46]. Increasing length of a strain has also been correlated with an increased period of convalescence. As the length of a strain is best demonstrated on MR imaging, this makes MR imaging the modality of choice. In fact, data are emerging that MR imaging assists in stratifying which athletes are also at higher risk of re-tear. Again, this is associated with an increased strain longitudinal length [38]. Other prognostic indicators include increased cross-sectional area, injury to the biceps femoris, and the presence of intermuscular haematoma. Though ultrasound has a high sensitivity for confirming the presence of a hamstring strain, it does not have the ability to differentiate which athletes are at high risk of re-strain or a prolonged period of convalescence, as the lengths measured are not discriminatory. In another study [39], the MR imaging presence of a strain did not predict accurately the period that an athlete would be restrained from training. However, the presence of a tear was significant, resulting in a prolonged period of convalescence (20.2 days). The best prognostic indicator for a patient with a clinically suspected hamstring strain is normal imaging (ultrasound or MR). In this instance, the period of convalescence ranges from 6.6 days [39] to 7 days [38].

Monitoring healing strains

MR imaging can be used to monitor patients in the healing phase, in particular, evaluating lesion resolution. This is reassuring to the clinician; however, it should be noted that at 6 weeks, nearly all athletes would have returned to competition, including those with persisting MR imaging (36%) and ultrasound (22%) demonstrable abnormality. Defects within the muscle may be visualised, as can also the presence of early scar tissue formation. Although some clinicians and radiologists are injecting steroids [47] or other substances into muscles to "promote healing", the practice is not widespread and certainly would benefit from further evaluation. Whether this results in an accelerated healing process is, at this stage, unclear, although this may provide symptomatic relief. It is worthy to note that the persistence of abnormality on imaging is present in a large proportion of athletes and is likely to represent an ongoing subacute to chronic healing/inflammatory response, ultimately forming mature scare formation. Though this commences as early as 7 days histopathologically [43], the persistence of inflammation likely reflects ongoing muscle re-organisation and scar tissue maturation.

Myositis ossificans

Muscle contusion is second only to strain as a cause of athletic morbidity [48]. Though the muscles of the HMC are less frequently contused than the lateral vastus muscle, the HMC and sacrotuberous ligaments [49] are vulnerable. Direct trauma (as opposed to strain, which is an indirect injury) may lead to the feared complication of myositis ossificans, though a strain also may be complicated by this

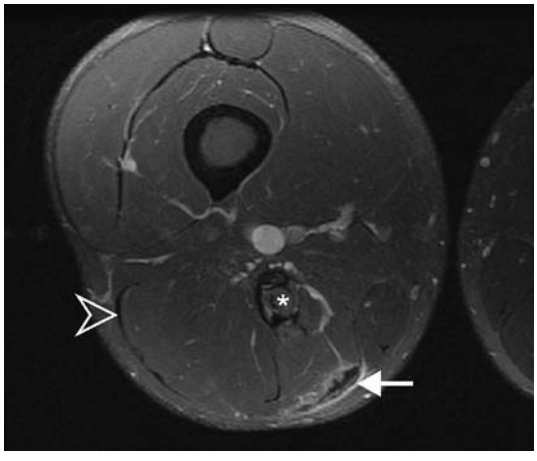

Fig. 8 Note the presence of a tandem injury in the uncommonly injured right semimembranosus on these proton-density fat-saturated sequences. A large, peripherally hypo-intense, centrally iso-intense globular focus is consistent with an area of evolving myositis ossificans (*asterisk*). Nodularity of the superficial tendon is also consistent with prior injury and subsequent scar formation (*straight arrow*). Hyperintensity around this region is consistent with a repeat musculotendinous junction strain. Note the subcutaneous hyperintensity related to this strain, as well as the eccentric position and the extent to which the tendon of the long head of biceps femoris envelops the muscle

[50]. As for haemorrhage, myositis ossificans, a form of heterotopic bone formation, has a variable appearance [51, 52], which is reflective of the complex interplay between the healing and scar tissue formation [48]. In its early phase, the focus is T1 hypo-intense, T2 hyperintense, with a peripheral rim of hypo-intensity, often with striking muscle oedema (Fig. 8). With maturity, the lesion becomes centrally T1 hyperintense, peripherally hypo-intense, consistent with medullary bone formation. CT may demonstrate the zonal distribution of myositis ossificans with improved resolution.

Early identification of evolving myositis ossificans is paramount, as the clinician may decide upon a course of non-steroidal anti-inflammatory medication, which may reduce the amount of heterotopic bone formed [53]. Though rare, the condition may be disabling, despite even drastic measures such as surgery. Knowledge even of the possibility of myositis ossificans avoids activities that may worsen the condition, such as massage, rehabilitation or aggressive rehabilitation [54]. Extensive oedema crossing muscle planes can be a useful diagnostic clue. The patient may then undergo CT evaluation to confirm the presence of early ossification. Furthermore, myositis may be diagnosed first on ultrasound, where the presence of fine (or "soft" calcification) is identified even prior to radiographically evident calcification [51].

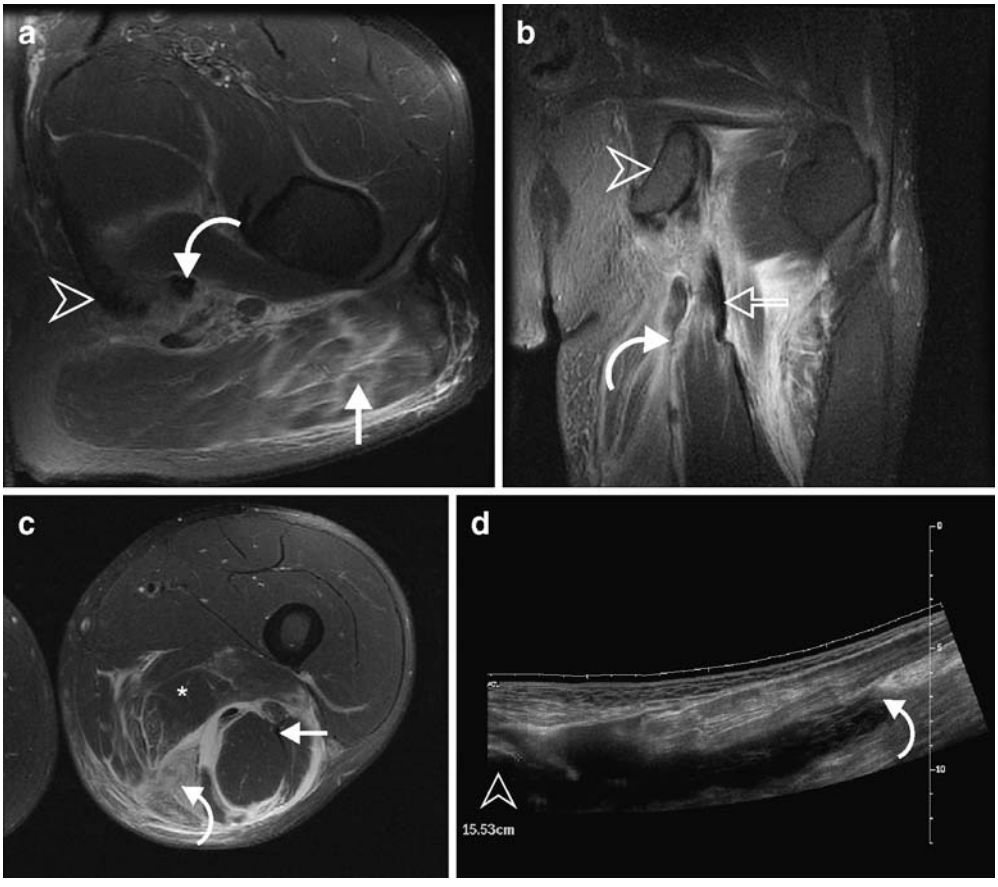

Fig. 9 **a**. Axial PD fat-saturated images through the left proximal origin show avulsion from the ischial tuberosity (*open arrowhead*) of the semimembranosus tendon (*curved arrow*) as well as the immediately posteriorly placed conjoint tendon (not marked). Associated tearing of the fibres of the gluteus maximus (*straight arrow*) in a water-skier is secondary to a severe hyperflexion force. **b**. The coronal images again show the dramatic injury. The conjoint tendon (*open arrow*) and semimembranosus tendon (*curved arrow*) are both recoiled. Note the large amount of haemorrhage and oedema. The cortex of the ischial tuberosity remains intact (*arrowhead*). **c**. Further distally, impressive amounts of haematoma surround all three muscles and tracks into the medial compartment, outlining the adductor muscles, particularly the adductor magnus (*asterisk*), as no fascia separating these compartments exists. The semimembranosus muscle (*curved arrow*) has also sustained a significant strain injury, the entire triangular muscle belly demonstrating hyperintense signal abnormality. Minimal strain injury to the adductor magnus is noted. Subcutaneous haematoma was clinically evident. Note the conjoint tendon (*straight arrow*). **d**. Ultrasound demonstrates a large haematoma filling in the defect left by the retracted avulsed tendons. A gap in excess of 15 cm was measured. Note the retracted end of the tendon distally (*curved arrow*) and its relationship with the ischial tuberosity (*open arrowhead*)

Avulsion

Truncal hyperflexion, which results in tension of the HMC at the hips with simultaneous knee extension resulting in tension at the knee, such as occurs when one is bending over to pick up a ball (particularly at full stride), seriously predisposes the hamstring muscle to rupture. By the time a person has reached the age of 25 years, the ischial apophysis has united with the remainder of the pelvis [55]. In the adult, rupture of the HMC occurs by way of ligamentous disruption (Fig. 9). This contrasts with the immature skeleton, where the un-united ischial apophysis is the commonest avulsion fracture of the pelvis [56]. If presentation by the patient is delayed, eventual imaging assessment may confuse the callus of the healing ischial tuberosity as an aggressive bone lesion. Biopsy is not reassuring, as the immature osteiod may be confused with an osteosarcoma. Hence, appreciation of the location of the injury is paramount to avoid unnecessary intervention.

Avulsion injury is classically proximal, with distal avulsion exceedingly uncommon [8, 57]. Water skiers are at high risk of avulsion injury [58], where gluteal muscle injury, including rupture, is an associated finding [59, 60]. Plain radiographs are initially performed in order to exclude osseous injury. MR imaging is the modality of choice, as it has a higher detection rate than ultrasound has [7]. The striking haemorrhage and oedema associated with avulsion injury contrasts with the low signal intensity of the recoiled disrupted tendons. Ultrasound on the other hand, is difficult in this setting, for a number of reasons. Imaging the proximal hamstrings often requires firm compression in order to maximise visualisation. This may not be possible in the tender limb. The well-developed gluteal musculature in an athlete often results in poor beam penetrance. Finally, the haemorrhage is hyperechoic, which is difficult to distinguish from the avulsed hyperechoic tendon. Surgical repair is best carried out immediately, though it can be scheduled as a delayed procedure.

Enthesopathy

Enthesopathy is an overuse phenomenon, occurring in a different athletic setting, namely that of the long-distance runner. This is in contrast to HMC strain, an injury of sprinters. This usually occurs proximally, with the signal intensity of the tendinous origin vague and ill-defined in size. If the condition is severe enough, the medullary bone may become oedematous, thus T2 or STIR hyperintense. Associated cortical thickening and periostitis may be present, and contrast enhancement may be seen. The ultrasound findings are, again, of those of tendon thickening, with disorganisation of the normal fibrillar echotexture as evidenced by a decrease in its echotexture (Fig. 10). The athlete may correlate this with the area of pain. As ultrasound is extremely sensitive at detecting bursal fluid, this is easily demonstrated as a thin hypo-echoic collection.

The importance of imaging is to identify the presence of a partial tear, which is correlated with a marked increase in rupture. This manifests itself as areas of high intra-tendinous signal on T2-weighted imaging or as discrete clefts of hypo-echogenicity on ultrasound. Partial tears are areas of discrete tendon fibre breakdown, usually secondary to a background of mucoid degeneration. Radiologically guided intervention is now starting to find favour in the treatment of enthesopathy, as it has in other tendon abnormalities. Injection, superficial to the tendon, of corticosteroid and local anaesthesia is performed to control local symptoms, particularly to alleviate the symptoms of bursitis, which is associated with enthesopathy. The tendon is not directly injected itself with corticosteroid, as this may

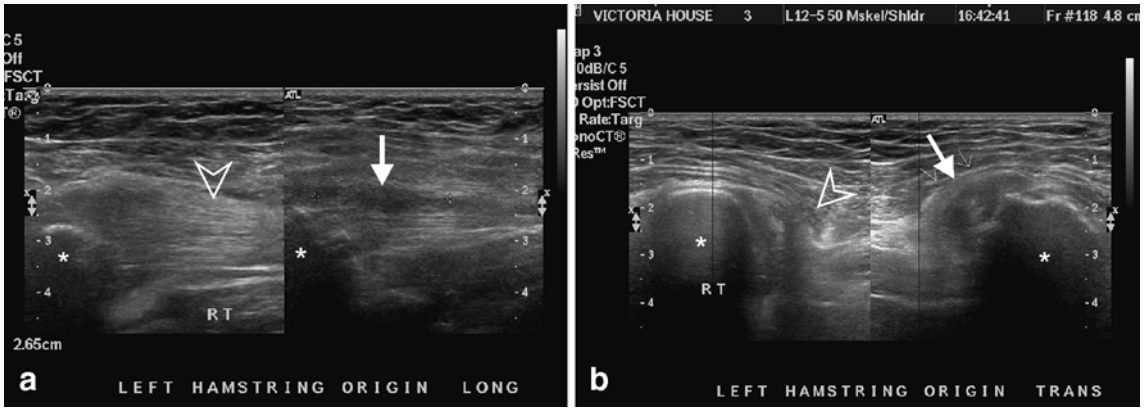

Fig. 10 **a** Longitudinal and **b** transverse ultrasound split-screen images through the ischial tuberosity (*asterisk*); *the images to the left* are those of the right hamstring origin. There is thickening and loss of the normal hyperechoic fibrillar echotexture typically seen in normal tendons (*arrowhead*). A focal area of hypo-echogenicity in the abnormal origin is consistent with mucoid degeneration resulting in a split tear (*straight arrow*). Note the cortical irregularity of the ischial tuberosity on the left side (**b**). The proximal portion of the tendon arising from the ischial tuberosity on the normal side is superiorly hypo-echoic, due to anisotropy as opposed to true pathology on the longitudinal images (**a**)

further weaken it and hence pose an even greater risk of rupture. A "dry needle technique" is then utilised, lancing the substance of the tendon repeatedly in an attempt to disrupt the already disorganised collagen fibres; however, it also causes internal foci of haemorrhage. This usually results in an inflammatory response (and, hence, true tendonitis). The idea behind this is that the reparative inflammatory response is beneficial, as granulation tissue forms and results in strengthening. The presence of increased vascularity, as brought about by the inflammatory response, further assists in the healing potential of the tendon and may prevent further damage. The injection of autologous blood into tendons has also shown promise [32, 34], and it is feasible that this technique may be applicable to enthesopathy of the hamstring origin as it may also have to HMC tendinopathy.

On plain radiographs the appearance of enthesopathy varies. With more recent injury, the ischial tuberosity may be osteopenic, ill defined and have the appearances of an aggressive, destructive lesion, as it does on MR imaging. As the injury heals, the tuberosity becomes asymmetrically enlarged and sclerotic. Uptake on all three phases, or only in the delayed images, is typical on technetium-99m bone scintigraphy. The presence of oedema on MR imaging (or increased uptake on scintigraphy on the delayed images) makes it unlikely that the patient will obtain a favourable response with dry needling. This oedema may take at least 6 months to resolve. The adolescent counterpart of this injury is apophysitis. Rarely is treatment surgical [61]. As for avulsion fracture, recognition of the characteristic location of this injury should alert the radiologist to the diagnosis, and it avoids unnecessary tissue diagnosis [62].

Conclusion

Despite most hamstring muscle injuries healing with conservative treatment, the high frequency, recurrence rate and the often prolonged period out of competition has led to an increase in imaging of this region, particularly in the elite athlete. Both magnetic resonance imaging and ultrasound have been shown to be able to effectively assess the extent of injury, hence providing a useful guide to the clinician as to the anticipated period of convalescence. Intervention is occasionally required to expedite healing, which, subject to further research, is a potential area where further radiological input is sought as an adjunct in the management of hamstring muscle complex injuries.

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