Gino M.M.J. Kerkhoffs
Elvire Servien
Editors

Acute Muscle Injuries

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Foreword I

Access to medical education, where basic sciences together with medical innovations and applied technology are indispensable, represents a challenge of which we are aware and strive to provide worldwide. With this exact purpose, this book is an excellent update of the state of the art in acute muscles injuries. It intends to improve and level the education playing field for sports medicine around the world.

Both access to technology and education are essential to provide equal education opportunities. Uneven realities described and emphasized by fellows and residents from all around the world, along with their extraordinary learning skills and strong motivation, made the authors realize the responsibility to provide an educational umbrella in which all agents would collaborate and profit. Therefore, in one unparalleled determination and effort supported by many, this book can bring you into high-performing educational sets, no matter the zip code you live in. They will join you or you will be joining them in this priceless educational mission. Consequently, allowing you to be in contact and to train with the most advanced therapeutic strategies with ultimate technologies and under guidance of globally renowned experts. This is the way to high-quality patient care.

In the subsequent pages of this book dedicated to muscles injuries, the reader will be able to get acquainted with the last novelties on that subject. This outstanding and generous share of knowledge conveys a comprehensive resource to education. It is a secure value and an important reflex of the authors' commitment to educational mission. The intelligibility, interest and actuality of the reading you are about to begin consubstantiates a text from the best to all that have the drive to catch up an intrinsic responsibility to provide the best health care to our patients. Science and skills brought to you by this book's authors arise from talented and passionate personalities that bring sports medicine nobility. This ESSKA group strives to give no less.

João Espregueira-Mendes

Foreword II

Dear Readers,

This special edition about muscular injuries provides current knowledge to orthopaedic surgeons.

Acute muscular injury is the most frequent trauma encountered by sport physicians and surgeons.

These injuries must be well understood: physiology, biomechanics, healing process, but also epidemiology, nutrition, and psychology may explain not only the onset of the injury but also how to manage these lesions.

The consequences of these lesions are sometimes dramatic when a professional or a very motivated leisure athlete is not able to return to sports due to sequelae of the muscle injury or when he or she is unable to play the next final due to a non-optimized management.

After a global and transversal approach, ten chapters cover different localizations of acute muscle injuries. The chapter will address the definition of the injury, clinical aspects, complementary exams, preoperative findings, assessment and therapeutic options.

Elvire Servien and Gino Kerkhoffs asked the most recognized experts in this field to share their knowledge and their experience.

Due to the excellent contribution of the authors and editors, with the support of ESSKA and ISAKOS, the up to date knowledge in acute muscle injury is now available.

Philippe Neyret

Preface

Aim

Acute muscle injuries present a high burden for all sports medicine physicians, physiotherapists, players, coaches and paramedics involved worldwide in and around the sport fields.

This book creates a unique platform that covers the latest evidence on the main acute muscle injuries.

Evidence-based medical content is combined with experience from medical experts from all around the globe in order to accommodate the reader with a full picture on the latest insights in terminology, basic healing aspects and treatment options for acute muscle injuries.

Hence, the book *Acute Muscle Injuries* contains interesting content to both medical and nonmedical individuals involved in sports participation.

Scope

Sports participation, both on an amateur as well as a professional level, has taken an enormous thrive in the last decades. National health programs are keen to advise people to participate in sports, because it is widely proven that sports participation has a positive influence on all aspects of health except for musculoskeletal injuries.

Acute muscle injuries present approximately 20 % of all injuries in sports and put forward an increasing challenge to the athlete and his/her staff.

As sports have become increasingly important, the physical and psychological impact on the players' health, together with the upgraded multidisciplinary care for the players, has become a growing issue. The challenge for the technical and medical staff in amateur as well as professional sports nowadays is to prevent and treat injuries, according to their best possible standards.

The aim of the book is to document the current standards for acute muscle injuries.

Most commonly, injuries are described from trauma mechanism, physical examination findings and diagnostic and treatment algorithms towards rehabilitation programs and full return to sports. The book is structured in a fashion that allows people to use it as a reference manual.

Therefore, this book is directed to orthopaedic surgeons, sports medicine physicians, physiotherapists, general practitioners, sports managers, athletes and coaches.

The authors are all scientifically active in the field of sports but are also well known for their great clinical experience in the care of acute muscle injuries, with often more than 20 years of expertise in the field.

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Collaboration Links

European Society for Sports traumatology Knee surgery and Arthroscopy (ESSKA) www.esska.org
International Society for Arthroscopy Knee surgery and Orthopaedic Sports medicine (ISAKOS) www.isakos.com

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Terminology and Classification of Athletic Muscle Injuries

1

Peter Ueblacker, Lutz Hänsel, Hans-Wilhelm Müller-Wohlfahrt, Kai Mithoefer, and Jan Ekstrand

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Abstract

Although athletic muscle injuries are very frequent, a consistent and comprehensive classification system as well as a clear terminology were so far missing. In order to facilitate effective communication among medical practitioners and to support the development of systematic treatment strategies, we developed

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NOTE: Since this book chapter is based on consensus statements published not before October 2012 (Epub ahead of print), the new nomenclature and classification used in this chapter is not yet reflected consistently in all chapters of this book.

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practical and systematic definitions of muscle injuries as well as a new and comprehensive classification system, both based on an international consensus meeting.

The classification system differentiates basically between (*A*) *indirect* and (*B*) *direct* muscle injuries and within the *indirect muscle injuries* between (1) *functional muscle disorders*, describing disorders without macroscopic evidence of fiber tear, and (2) *structural muscle injuries* with macroscopic evidence of fiber tear, i.e., structural damage. Subclassifications are presented for each type.

This comprehensive classification system is proven in the daily practice and scientifically validated. It will help to improve clarity of communication for diagnostic and therapeutic purposes and can serve as the basis for future comparative studies to address the continued lack of systematic information on muscle injuries in the literature.

1.1 Introduction

Muscle injuries are a substantial problem for athletes. They constitute 31 % of all injuries in elite football (soccer) [12]; thigh muscle injuries are the most common diagnosis in track and field athletes with 16 % [24, 25]. Their relevance has also been documented in many other sports like rugby (10.4 %) [23], basketball (17.7 %) [5], and American football (46/22 % practice/games) [14].

Muscles that are frequently involved in injuries are often biarticular [19] or have a more complex architecture (e.g., adductor longus). They usually undergo eccentric contraction and contain primarily fast-twitch type 2 muscle fibers [1, 30]. Ninety-two percent of all muscle injuries affect the four major muscle groups of the lower limbs: hamstrings (37 %), adductors (23 %), quadriceps (19 %), and calf muscles (13 %) [12]. Sixteen percent of muscle injuries in soccer are reinjuries and cause significantly longer absence times than the initial injuries [12].

Particularly in elite athletes, where decisions regarding return to play and player availability have significant financial or strategic consequences for the player and the team, there is an enormous interest in optimizing the diagnostic, therapeutic, and rehabilitation process after muscle injuries in order to minimize the absence from sport and to reduce recurrence rates.

However, little information is available in the international literature about muscle injury definitions and classification systems. Since injury definitions are not standardized and guidelines are missing, proper assessment of muscle injury and communication between practitioners are often difficult to achieve [29]. Moreover, it has been documented that variability between definitions creates significant differences in study results and conclusions [7, 17, 20]. Thus, it is critically important to establish a standardization. *Muscle strain*, for example, presents one of the most frequently used terms to describe athletic muscle injury, but this term is still without clear definition and used with high variability [29].

The aim of this book chapter is to present a standardized English terminology for muscle injuries in order to improve diagnostic, therapeutic, and scientific communication. The standardized definitions were established in a consensus meeting of international sports medicine experts working in the field of muscle injuries.

In addition, an empirically based comprehensive and practical classification system is presented that reflects the differentiated spectrum of muscle injuries seen in athletes. This classification was recently published as a consensus statement [29]. It is based on an extensive, long-term experience and has been used successfully in the daily management of athletic muscle injuries.

It has to be pointed out that this book chapter is mainly based on the 2013 publication by Mueller-Wohlfahrt et al. in *British Journal of Sports Medicine*.

1.2 Terminology

Even among sports experts, considerable inconsistency exists in the use of muscle injury terminology. There is no clear definition, differentiation, and use of several terms like *strain*, *tear*, *pulled muscle*. To evaluate the currently used English terminology of athletic muscle injuries, a survey of sports medicine experts was conducted by the authors. The results confirmed that even among experts working frequently with athletic muscle injuries at the elite level, considerable inconsistency exists in the use of muscle injury terminology.

After this survey, a consensus meeting of 15 international experts on the basic science of muscle injury as well as sports medicine specialists involved in the daily care of premier professional sports and national teams was established. The meeting was endorsed by the International Olympic Committee (IOC) and the Union of European Football Associations (UEFA).

In the consensus meeting, practical and systematic scientific terms of muscle injuries were defined. In addition, a new comprehensive classification system was developed.

The following consensus definitions were established:

1.2.1 Functional (=non-structural) Muscle Disorder

"*Acute indirect muscle disorder* <u>*without macroscopic*</u> *evidence (in MRI or ultrasound) of muscular tear.*

Often associated with circumscribed increase of muscle tone (muscle firmness) in varying dimensions and predisposing to tears. Based on the etiology several subcategories of functional muscle disorders exist."

According to Fuller et al., a sports injury is defined as "*any physical complaint sustained by an athlete that results from a match/competition or training, irrespective of the need for medical attention or time loss from sportive activities*" [15].

That means also irrespective of a structural damage. By this definition, *functional muscle disorders*, irrespective of any *structural* muscle damage, are *injuries* as well. However, the term *disorder* better differentiates *functional disorders* from *structural injuries*. Thus, the term *functional muscle disorder* was specifically chosen by the consensus conference.

Functional muscle disorders are *indirect* injuries, i.e., not caused by external force, and present a distinct clinical entity. They result in a *functional* limitation for the athlete, e.g., painful increase of the muscle tone which can represent a risk factor for *structural* injury. However, they are not readily diagnosed with standard diagnostic methods such as MRI since they are without macroscopic evidence of *structural* damage, defined as absence of fiber tear on MRI.

A recent UEFA muscle injury study has demonstrated the relevance of *functional muscle disorders* in football/soccer [13]. This study included data from a 4-year observation period of MRI obtained within 24–48 h after injury and demonstrated that the majority of injuries (70 %) were without signs of fiber tear. However, these injuries caused more than 50 % of the absence of players in the clubs [13].

1.2.2 Structural Muscle Injury

"*Any acute indirect muscle injury <u>with macroscopic</u> evidence (in MRI or ultrasound) of muscle tear.*"

It must be pointed out that MRI is usually precise enough to determine if there is a relevant tear or not. However, MRI alone is not appropriate to determine the diagnosis and extent of a muscle injury. Careful combination of diagnostic modalities including medical history, inspection, clinical examination, and imaging will most likely lead to an accurate diagnosis, not imaging alone.

For example, the history of a sharp acute onset of pain, experience of a snap, and a well-defined localized pain with positive MRI for edema but indecisive for fiber tear strongly suggest a minor partial muscle tear, below the detection sensitivity of the MRI. Edema, or better the increased fluid signal on MRI, would be observed with a localized hematoma and would be consistent with the working diagnosis in this case. The diagnosis of a small tear (*structural* defect) that is below the MRI detection limit is important in our eyes, since even a small tear is relevant because it can further disrupt, e.g., when the athlete sprints.

1.2.3 Strain and Tear

Strain represents certainly one of the most frequently used terms to describe athletic muscle injury. Hägglund et al. defined it as "*acute distraction injury of muscles and tendons*" [17]. However, this definition is rarely used in the literature and in the day-to-day management of athletic muscle injuries.

Strain is a biomechanical term which is not defined and used indiscriminately for anatomically and functionally different muscle injuries. Some authors use *strain* exclusively as a term for “grade I injuries” or “minor muscle injuries” involving only a few muscle fibers [9], whereas others subsume different grades of injuries ranging from mild to severe [25, 30]. Again other authors differentiate *strain* from *ruptures.*

Thus, we do not recommend the use of this term. Instead, we propose to use the term *tear* (which has the same meaning like *rupture*) for *structural injuries* of muscle fibers/bundles leading to loss of continuity and contractile properties. *Tear* better reflects *structural* characteristics as opposed to a mechanism of injury.

The following terms are without specific recommendation:

Strain – See above.

Pulled muscle – A lay term for different, undefined types or grades of muscle injuries and cannot be recommended as a scientific term.

(Further recommended consensus definitions are presented below together with the new classification system.)

1.3 Classification

Usually, researchers compare the results from their study with results from other published studies. But comparisons of different muscle injuries and layoff times can only be made between studies with essentially the same injury definitions and classification system. Since so far no universal and comprehensive classification existed that includes all types of athletic muscle injuries, the significant methodological differences between studies do not allow for comparison between study results [10].

Different classification systems are published in the literature (Table 1.1), but no system is consistently used within studies and in daily practice [8]. Most of the grading systems classify acute muscle injuries as grade 1, 2, and 3. However, this does not accurately reflect the occurrence of muscle injuries in athletes. Previous systems are either based upon clinical signs or upon imaging.

All previous grading systems lack subclassifications within the grades or types. In consequence, injuries with a different etiology, treatment pathway, and prognostic relevance are categorized in the same group. Moreover, no terminology or grading system (sub)classified disorders without macroscopic evidence of structural damage, even though a muscle injury study of the *Union of European Football Associations (UEFA) has* emphasized their high clinical relevance in professional athletes, as mentioned above [13].

The most recently published classification system by Chan et al. is imaging based, even though many authors have stated that diagnosis and prognosis of muscular injuries are normally mainly based on clinical findings and radiological methods such as MRI or ultrasound are used for additional information in order to confirm a diagnosis [13, 22].

Table 1.1 Previous muscle injury classification systems

	O'Donoghue 1962 [31]	Ryan 1969 (*initially for quadriceps*) [36]	Takebayashi et al. 1995, Peetrons 2002 (*ultrasound based*) [35, 40]	Stoller 2007 (*MRI based*) [39]	Chan et al. 2012 (*MRI/ultrasound based*) [9]
Grade I	No appreciable tissue tearing, no loss of function or strength, only a low-grade inflammatory response	Tear of a few muscle fibers, fascia remaining intact	No abnormalities or diffuse bleeding with/without focal fiber rupture less than 5 % of the muscle involved	MRI negative = 0 % structural damage, hyperintense edema with or without hemorrhage	Includes Site of injury: Proximal Middle Distal Pattern: Intramuscular Myofascial Myofascial/perifascial Musculotendinous Combined Severity: Comparable to Stoller
Grade II	Tissue damage, strength of the musculotendinous unit reduced, some residual function	Tear of a moderate number of fibers, fascia remaining intact	Partial rupture: focal fiber rupture more than 5 % of the muscle involved with/without fascial injury	MRI positive with tearing up to 50 % of the muscle fibers. Possible hyperintense focal defect and partial retraction of muscle fibers	
Grade III	Complete tear of musculotendinous unit, complete loss of function	Tear of many fibers with partial tearing of the fascia	Complete muscle rupture with retraction, fascial injury	Muscle rupture = 100 % structural damage. Complete tearing with or without muscle retraction	
Grade IV	X	Complete tear of the muscle and fascia of the muscle–tendon unit	X	X	X

Modified after Mueller-Wohlfahrt et al. [29]

1.3.1 Diagnosis

Our approach is to include the combination of the currently best available diagnostic tools. Careful combination of diagnostic modalities including medical history, inspection, clinical examination, and imaging will most likely lead to an accurate diagnosis, rather than relying on imaging alone.

In accordance with Askling et al. and Järvinen et al., we recommend to start with a precise history of occurrence, circumstances, symptoms, and previous problems, followed by a careful clinical examination with inspection, palpation of the injured area, comparison to the other side, and testing of the function of the muscles [2, 19]. Palpation serves to detect (more superficial and larger) tears, perimuscular edema, and increased muscle tone. An early post-injury ultrasound provides helpful information about any existing disturbance of the muscle structure and reveals if further MR imaging is needed.

1.3.2 Imaging

Imaging (ultrasound and MRI) definitely provides additional information about a muscle injury. It helps to localize the site of injury to reveal if there is any hematoma and a defect/tear including its approximate size in the muscle tissue and if the tendon is involved. Especially MRI is helpful to determine edema incidence and pattern. However, diagnosis based only on imaging is not appropriate. Even the best imaging reveals no information about the muscle tone, pain, functional loss, and other information such as previous injuries, which has significant relevance for the management of the athlete.

In daily practice ultrasound is sufficient in many cases to localize the site of injury and to exclude higher grade of injury. Ultrasound is easily available and cost-effective which makes it superior to MRI for follow-up examinations. However, it must be pointed out that examination of skeletal muscle takes time. With a little practice, the examiner can distinguish a *functional muscle disorder* without evidence of structural damage from a *structural injury* with a tissue defect. We recommend to use a 7.5 or 10.0 MHz transducer and to start with a transversal section. A complete scan through the muscle should be performed to obtain anatomical orientation. The longitudinal section is added on locations where a disturbance of the muscle structure or a gap is suspected. Ultrasound can also assess the need for further investigation by MRI. We recommend MRI in every case that is suspicious for *structural* injury.

High-resolution imaging is required for precise diagnosis. However, quality of MR imaging differs a lot since many radiologists choose a large field of view demonstrating, for example, both thighs including the pelvis in one examination (even though the clinical question is, e.g., only to search for a partial muscle tear in the biceps femoris muscle). Combination with clinical examination is critical to make MRI more sensitive.

The argument that large field-of-view MRI is the best initial test in order not to miss some muscle injuries is only relevant if no clinical and ultrasound examination

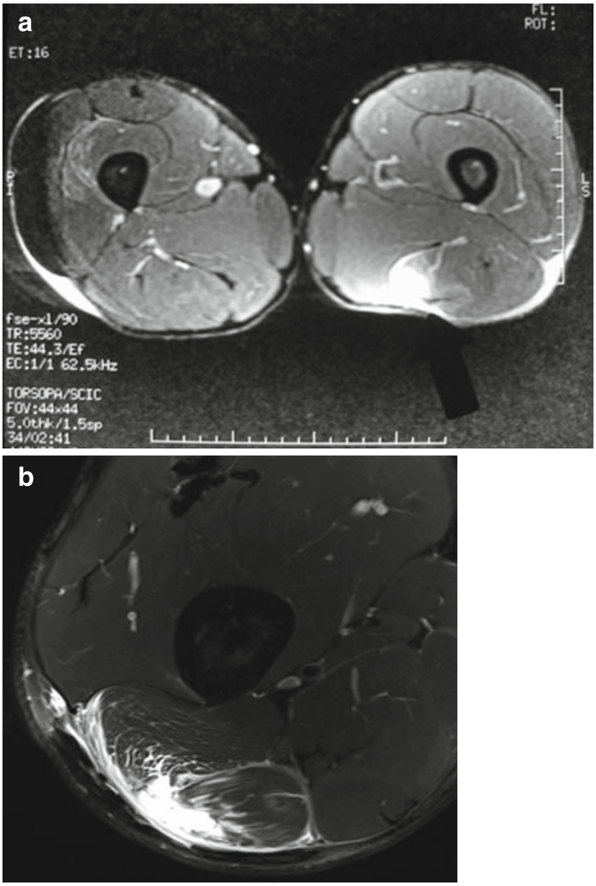

Fig. 1.1 Demonstrates clearly how important resolution and field of view are for interpreting MR imaging (Reprinted with permission of UEFA [41]). In (**a**) it is not possible to judge from the images if there is a structural defect or not, due to overlaying bright signal. (**b**) Performed in high-resolution technique (3 T, coil, limited field of view, 3 mm slice, etc.) demonstrating more clearly the actual defect in the muscle structure

was done before MRI, what usually should not happen. With clinical examination, the field of view can easily be limited. This will lead to a much higher spatial resolution.

We recommend a high field strength with a minimum of 1.5 or better 3 T, the use of surface coils, a limited field of view based on clinical examination and ultrasound, the use of skin markers to localize the center of injury, and a multiplanar orientation. Three millimeter slices must be postulated for MR imaging in muscle injuries. Otherwise smaller tears could be missed. With a limited field of view in MRI, this does not cause additional examination time.

But even in best quality, MRI alone is not sensitive enough to measure the extent of muscle tissue damage accurately. For example, it is not possible to judge from the scans where edema/hemorrhage (seen as high signal) is obscuring muscle tissue that has not been structurally damaged (Figs. 1.1 and 1.2).

Imaging technology to detect muscle injuries continues to evolve, and future techniques may allow for more sensitive and specific visualization of muscle injury and pathology.

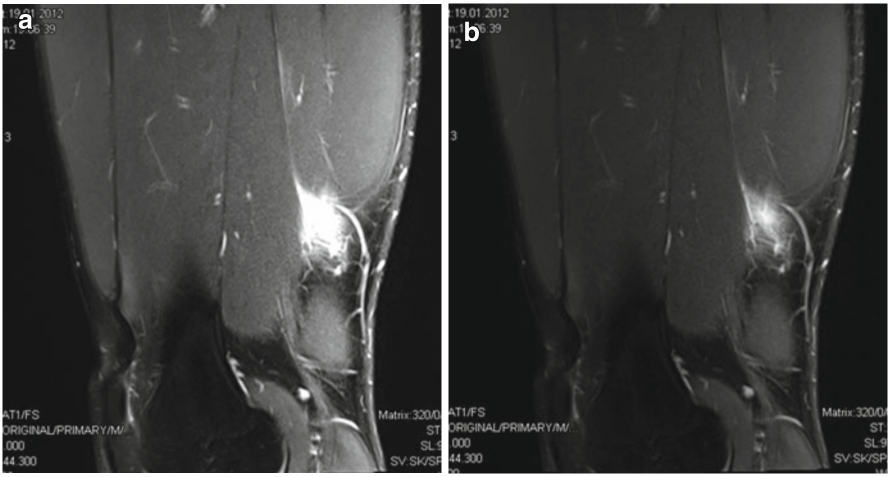

Fig. 1.2 Demonstrates that contrast and brightness (adjustable on the radiological monitors) play a crucial role for interpretation of muscle injuries (Reprinted with permission of UEFA [41]). (**a**) High brightness and little contrast demonstrating a large muscle tear. (**b**) After adjusting contrast and brightness, the actual defect in the muscle appears much smaller

1.3.3 New Comprehensive Classification System

The presented muscle injury classification is based on an extensive, long-term experience and has been used successfully in the daily management of athletic muscle injuries. The classification is empirically based and includes several original aspects of athletic muscle injuries that have not yet been described in the literature, specifically the frequently observed *functional muscle disorders*. Distinguishing these injuries as separate clinical entities has great relevance for the successful management of the athlete with muscle injury and represents the basis for future comparative studies since scientific data are limited for muscle injury in general.

This advanced comprehensive classification system differentiates between *indirect* and *direct* muscle injuries. *Indirect* muscle injuries are divided into *functional* and *structural* ones. *Functional muscle disorders* (type 1, overexertion-related, and type 2, neuromuscular muscle disorders) describe disorders without macroscopic evidence of fiber tear. *Structural muscle injuries* (type 3, partial tears, and type 4, (sub)total tears/tendinous avulsions) are injuries with macroscopic evidence of fiber tear, i.e., structural damage. Subclassifications are presented for each type.

1.3.3.1 Functional (=non-structural) Muscle Disorders

Functional muscle *disorders* are multifactorial. They can be grouped into subgroups reflecting their clinical origin: “overexertional” and “neuromuscular” muscle disorders. This is important since the origin of muscle disorder influences their treatment pathway. A spine-related muscle disorder associated with a spine problem (e.g., spondylolysis) will respond better to treatment by addressing not only the muscle disorder itself but also the original back problem. One could argue that this presents mainly a back problem with a secondary muscle disorder. However, this secondary muscular disorder prevents the athlete from sports participation and will require comprehensive treatment that includes the primary problem as well in order to facilitate return to sport. Thus, a differentiation is important not only because of the different pathogenesis but more importantly because of different therapeutic implications.

1.3.3.1.1 Overexertion-Related Muscle Disorders

Weakened or fatigued muscles absorb less energy and are therefore more likely to get injured [16]. Previous data has shown that muscle fatigue predisposes the athlete to muscle injury [33, Wilson AJ and Myers PT, 2005, unpublished data]. Thus, muscle fatigue due to overexertion and other factors, which frequently presents as firmness of a muscle (bundle or part), must be recognized and treated.

Delayed onset muscle soreness has to be differentiated from fatigue-induced muscle injury [4]. DOMS occurs several hours *after* unaccustomed deceleration movements while the muscle is stretched by external forces (eccentric contractions), whereas fatigue-induced muscle disorder can also occur *during* athletic activity. DOMS resolves spontaneously usually within a week. In contrast, fatigue-induced muscle disorder can – if unrecognized and untreated – persist over a longer time and may cause *structural* injuries such as partial tears.

1.3.3.1.2 Neuromuscular Muscle Disorders

The term *neuromuscular* was chosen by the consensus group to describe the specific pathogenesis of these muscle disorders. Two different types of neuromuscular disorders can be differentiated: a spinal or spinal nerve-related (central) and a neuromuscular end plate-related (peripheral) type.

Muscles act as a target organ and their state of tension is modulated by electrical information from the motor component of the corresponding spinal nerve. Thus, irritation of a spinal nerve root can cause an increase in muscle tone. It is known that back injuries are very frequent in elite athletes [32] and lumbar pathology such as disc prolapse at the L5/S1 level may present with hamstring and/or calf pain and limitations in flexibility, which may result in or mimic a muscle injury [34]. It is logical that this type of injury would require variable forms of treatment beyond simple treatment of the muscle–tendon injuries [18]. Thus, it is important that assessment of muscle injury should include a thorough biomechanical evaluation, especially that of the lumbar spine, pelvis, and sacrum. Negative *structural* findings on the lumbar spine do not exclude nerve root irritation. *Functional* lumbar dysfunctions, like lumbar or iliosacral blocking, can also cause spine-related muscle disorders [28]. The diagnosis is then established through precise clinical functional examination. The spine-related muscle disorder is usually MRI negative or shows muscle edema only [34]. Verrall et al. showed that footballers with a history of lumbar spine injury had a higher rate of MRI-negative posterior thigh injury, but not of actual *structural* hamstring injury [42].

We differentiate muscle-related neuromuscular disorders from the spine-related ones because of different treatment pathways. Dysfunction of neuromuscular control mechanisms can result in a painful muscle firmness which can prevent an athlete from sportive activities, when inhibition of antagonistic muscles is disturbed and agonistic muscles over-contract to compensate this [6].

It has to be stated that it remains an area for future research to definitely describe the *functional muscle disorders* and other risk factors for muscle injuries.

1.3.3.2 Structural Injuries

1.3.3.2.1 Partial Muscle Tears

Most *indirect structural* injuries are partial muscle tears. Clinical experience clearly shows that most partial injuries can be assigned to one of two types, either a minor or a moderate partial muscle tear, which ultimately has consequences for therapy and absence time from sports. Thus, *indirect structural* injuries should be subclassified. Since previous graduation systems refer to the complete muscle size, they are relative and not consistently measurable. In addition to this, there is no differentiation of grade 3 injuries with the consequence that many *structural* injuries with different prognostic consequences are subsumed as grade 3.

Anatomical facts should be considered while discussing muscle injury and classification: The individual muscle fiber presents a microscopic structure with an average diameter of 60 μm [38]. Therefore, an isolated tear of a single muscle fiber remains without clinical relevance. Muscle fibers are anatomically organized into primary and secondary muscle fascicles/bundles. Multiple secondary bundles constitute the muscle.

The extent of the injury to the anatomic landmarks determines the difference between minor and moderate partial muscle tear. However, it definitely remains a challenge for future studies to determine the exact cutoff.

Besides size, the involvement of adjacent connective tissue (endomysium, perimysium, epimysium, and fascia) distinguishes partial muscle tears from each other. Concomitant injury of the external perimysium seems to play a special role: This connective tissue structure somehow has an intramuscular barrier function in case of bleeding. It may be the injury to this structure (with optional involvement of the muscle fascia) that differentiates a moderate from a minor partial muscle tear.

However, drawing a clear differentiation between partial muscle tears seems difficult because of the heterogeneity of the muscles that can be structured very differently. Technical diagnostic tools today (MRI and ultrasound) are not precise enough to ultimately determine and prove the effective muscular defect within the injury zone of hematoma and/or liquid seen in MRI, which can [19] lead to overestimation of the actual damage. It will remain a challenge for future studies to exactly define the size of the injury which describes the distinction between a minor and a moderate partial muscle tear.

The great majority of muscle injuries heal without formation of scar tissue. However, greater muscle tears can result in a defective healing with scar formation [19] which has to be considered in the diagnosis and prognosis of a muscle injury. Our experience is that partial tears of less than a muscle fascicle usually heal completely, while moderate partial tears can result in a fibrous scar.

Table 1.2 Classification of acute muscle disorders and injuries

A. Indirect muscle disorder/injury	Functional muscle disorder	Type 1: Overexertion-related muscle disorder	Type 1A: Fatigue-induced muscle disorder
			Type 1B: Delayed onset muscle soreness (DOMS)
		Type 2: Neuromuscular muscle disorder	Type 2A: Spine-related neuromuscular muscle disorder
			Type 2B: Muscle-related neuromuscular muscle disorder
	Structural muscle injury	Type 3: Partial muscle tear	Type 3A: Minor partial muscle tear
			Type 3B: Moderate partial muscle tear
		Type 4: (Sub)total tear	Subtotal or complete muscle tear Tendinous avulsion
B. Direct muscle injury		Contusion	
		Laceration	

Modified after Mueller-Wohlfahrt et al. [29]

1.3.3.2.2 (Sub)Total Muscle Tears and Tendinous Avulsions

Complete muscle tears with a discontinuity of the whole muscle are very rare. Subtotal muscle tears and tendinous avulsions are more frequent. Clinical experience shows that injuries involving more than 50 % of the muscle diameter (subtotal tears) usually have a similar healing time compared with complete tears.

Tendinous avulsions are included in the classification system since they are biomechanically complete tears of the origin or insertion of the muscle. The most frequently involved locations are the proximal rectus femoris, the proximal hamstrings, the proximal adductor longus, and the distal semitendinosus.

Intratendinous lesions of the free or intramuscular tendon also occur. Pure intratendinous lesions are rare. The most frequent type is a tear near the musculotendinous junction (e.g., of the intramuscular tendon of the rectus femoris muscle). Tendinous injuries are either consistent with the partial (type 3) or (sub)total (type 4) tear in our classification system and can be included in that aspect of the classification.

1.3.3.2.3 Muscle Contusions

In contrast to *indirect* injuries (caused by internal forces), lacerations or contusions are caused by external forces [3, 21] like a direct blow from an opponent's knee. Thus, muscle contusions are classified as acute *direct* muscle injuries (Tables 1.1 and 1.2).

Contusion injuries are common in athletes and present a complex injury that includes defined blunt trauma of the muscular tissue and associated hematoma [3, 21]. The severity of the injury depends on the contact force, the contraction state of the affected muscle at the moment of injury, and other factors. Contusions can be graded into mild, moderate, and severe [37]. The most frequently injured muscles are the exposed rectus femoris and the intermediate vastus, lying next to the bone, with limited space for movement when exposed to a direct blunt blow. Contusion injury can lead to either diffuse or circumscribed bleeding that displaces or compresses muscle fibers causing pain and loss of motion. It happens that muscle fibers are torn off by the impact or by shear forces, but muscle fibers are not typically torn by longitudinal distraction. Therefore, contusions are not necessarily accompanied by a *structural* damage of muscle tissue. For this reason athletes, even with more severe contusions, can often continue playing for a long time, whereas even a smaller *indirect structural* injury often forces the player to stop at once. However, contusions may lead to persistent intramuscular bleeding and hematoma formation with the potential for severe complications such as acute or delayed compartment syndrome and possible resultant long-term functional limitation [26, 27].

Conclusion

Consensus definitions of the English terminology of athletic muscle injuries as well as a new comprehensive and empirical classification system for acute muscle injuries are presented. Both will help to improve effective communication among medical practitioners and development of systematic treatment strategies and can serve as the basis for future comparative studies to address the continued lack of systematic information on muscle injuries in the literature.

Key component of the new classification system is the differentiation between indirect and direct muscle injuries and within the indirect injuries between *functional muscle disorders* from *structural injuries*. The use of the term *strain* is no longer recommended, since it is a biomechanical term, not well defined, and used indiscriminately for anatomically and functionally different muscle injuries. Instead, we propose the term *tear* for *structural injuries*, graded into (minor and moderate) partial and (sub)total tears, used only for muscle injuries with macroscopic evidence of muscle damage (*structural injuries*). While this classification is most applicable to lower limb muscle injuries, it can be translated also to the upper limb.

Scientific data supporting the presented classification system can be found in the publication "Return to play after thigh muscle injury in elite football players: implementation and validation of the Munich muscle injury classification" by Ekstrand J, Askling C, Magnusson H, and Mithoefer K in the *British Journal of Sport Medicine* [11]. Further scientific data are still missing. We hope that the suggested standardized terminology and the new classification system will stimulate research to prospectively evaluate the prognostic and therapeutic implications of the new classification. Furthermore, future studies have to define the exact size threshold between a minor and a moderate partial muscle tear.

Note: Further information about the consensus definitions and the new classification system can be found in

Mueller-Wohlfahrt HW, Haensel L, Mithoefer K, Ekstrand J, English B, McNally S, Orchard J, van Dijk N, Kerkhoffs G, Schamasch P, Blottner D, Swaerd L, Goedhart E, Ueblacker P. Terminology and classification of muscle injuries in sport. A consensus statement. Br J Sports Med. 2013;47(6):342–50.

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Basic Principles of Muscle Healing

2

Thomas Laumonier, Jacques Menetrey, and Johnny Huard

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Abstract

Skeletal muscle has the ability to regenerate following injury, and this response implicates a specific type of resident muscle stem cell, the satellite cell. Three main phases have been identified in the process of muscle regeneration, including (I) a destruction phase with the initial inflammatory response, (II) a repair phase with the activation of satellite cells, and (III) a remodeling phase with the maturation of the regenerated myofibers. Nevertheless, in severe muscle injuries, we also observed the formation of fibrosis that impairs muscle function. Various

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strategies, including the use of growth factors, transplantation of muscle stem cells, or antifibrotic therapies, may become therapeutic alternatives to improve functional recovery after severe muscle injuries.

2.1 Introduction

Human skeletal muscle is about 40 % of the body mass and is formed by bundle of contractile muscle fibers. Muscle fibers are multinucleated cells resulting from the fusion of myoblast, the muscle progenitor cells. Myofibers are surrounded by the sarcolemma, the plasma membrane of muscle fibers. Located between the plasma membrane and the basal lamina, we find satellite cells, i.e., the reserve adult muscle stem cells, which play a key role in the muscle regeneration process [19, 26]. After muscle injury, satellite cells are activated and form myoblasts, then fuse into myotubes, and mature into new myofibers that participate in the muscle regeneration process.

2.2 Muscle Injury

Muscle injuries can stem from a variety of events, including direct trauma (i.e., muscle lacerations, contusions, or strains) and indirect causes (i.e., ischemia or neurological dysfunction) [10, 15, 18, 23, 31]. Muscle injury is one of the most common injuries in professional and recreational sports. In fact, muscle injuries constitute between 10 and 55 % of all injuries sustained by athletes, depending on the type of sport [33]. Whereas relatively minor muscle injuries, such as strains, can heal completely without intervention, severe muscle injuries typically result in the formation of dense scar tissue that impairs muscle function and can lead to muscle contracture and chronic pain. Injured muscle undergoes a sequential cycle of healing phases. Three phases have been identified in this process (Fig. 2.1):

I. Destruction phase, including muscle degeneration/inflammation: Characterized by the rupture and then necrosis of the myofibers, formation of a hematoma, and an important inflammatory reaction.
II. Repair phase: In this phase, we observed phagocytosis of the damaged tissue, followed by regeneration of the myofibers, leading to activation of the satellite cells.
III. Remodeling phase: A period during which we observed maturation of the regenerated myofibers with recovery of the functional capacity of the muscle (III b) but also a period where we can observed fibrosis deposition (III a).

2.2.1 Muscle Degeneration and Inflammation

Active muscle degeneration and inflammation occur within the first few days after injury. In injured muscle, mechanical trauma destroys the integrity of the myofibers. The injured ends of the myofibers undergo rapid necrosis. Similar to cell necrosis, the inflammation starts with invasion of mononuclear cells, activated macrophages,

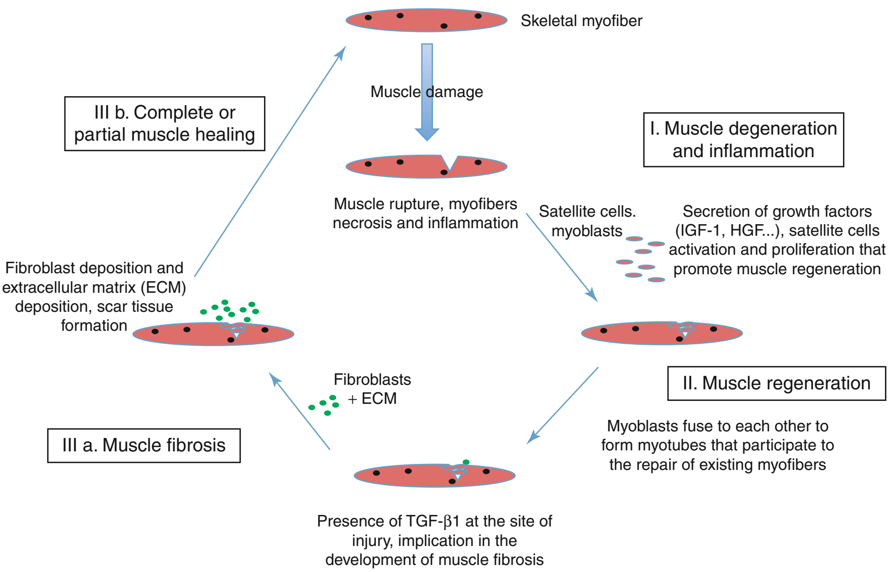

Fig. 2.1 Sequential cycle of muscle healing phases

and lymphocytes at the injury site [45]. Necrotic debris of the damaged myofibers are phagocytized by macrophages, which simultaneously secrete growth factors that enhance muscle regeneration by favoring satellite cells activation and proliferation [7].

Nonsteroidal anti-inflammatory drugs (NSAIDs) are often prescribed to relieve pain after muscle injury. However, the effect of this group of drugs on the muscle healing process remains largely controversial. Human studies are lacking, but some studies have been performed in animal models. It appears that short-term use of different NSAIDs had no major adverse effect on muscle healing [28].

2.2.2 Muscle Regeneration

Muscle regeneration usually starts during the first week after injury, peaks at 2 weeks, and then gradually diminishes 3–4 weeks after injury. Regeneration is linked to the activation of the satellite cells. Satellite cells proliferate, form myoblasts, and fuse with each other to form new multinucleated myotubes that will participate in the muscle regeneration process.

2.2.3 Muscle Fibrosis

Despite the fact that the majority of skeletal muscle lesions heal without formation of an extensive scar tissue, we often observe formation of a dense scar tissue that can prevent the skeletal muscle regeneration process in severe muscle injuries or muscle re-ruptures. Fibrosis usually starts between the second and third week after muscle injury. The amount of scar tissue increases in size over time due to excessive fibroblast proliferation and an increase in production of type I collagen [22].

2.3 Improving Muscle Healing

2.3.1 Growth Factors

Many reports have shown that growth factors play a variety of roles during muscle regeneration [16, 30]. Although hepatocyte growth factor (HGF), fibroblast growth factor (FGF), and platelet-derived growth factor (PDGF) are of interest because of their capacity to stimulate satellite cells [1, 39, 47], insulin-like growth factor-1 (IGF-1) appears to be of particular importance for the muscle regeneration process notably because IGF-1 stimulates myoblasts proliferation and differentiation [13]. IGF-1 is implicated in the regulation of muscle growth [38]. In a mouse model, direct injections of human recombinant IGF-1 at 2, 5, and 7 days after injury have enhanced muscle healing in lacerated, contused, and strain-injured muscle [20, 30]. However, the efficacy of direct injection of recombinant proteins (growth factors) is limited by the high concentration of the factor typically required to elicit a measurable effect. This is mainly due to the bloodstream's rapid clearance of these molecules and their relatively short biological half-lives. Gene therapy may prove to be an effective method by which to deliver high, maintainable concentrations of growth factor to injured muscle [2, 3, 32]. Although we observed improved muscle healing, histology of the injected muscle revealed muscle fibrosis within the lacerated site, despite the production of a high level of IGF-1 [21]. Some studies suggest that the stimulatory action of IGF-1 on myofibroblast proliferation and the deposition of extracellular matrix (ECM-scar tissue) might interfere with the ability of this growth factor to improve muscle healing after injury, even at high concentrations [11].

2.3.2 Stem Cell Therapy

Transplantation of myogenic precursor cells represents a promising therapeutic strategy for treatment of extensive skeletal muscle destruction. Myogenic precursor cells can participate directly in the muscle regeneration process but also create a reservoir of secreting molecules that may impact the different stages of muscle healing. Despite encouraging results obtained in animal models [36], the subsequent clinical trials of myoblast transfer in human patients have been disappointing due to rapid death, limited spread of the injected cells, and rejection of transplanted myoblasts [17, 29, 40]. Although the use of myoblasts for cell therapy applications is prevalent, concerns associated with myoblast proliferation, cell migration, and the limited life span of these cells have brought the usage of stem cells to the forefront of such applications. Stem cells are defined as cells that can both self-renew and

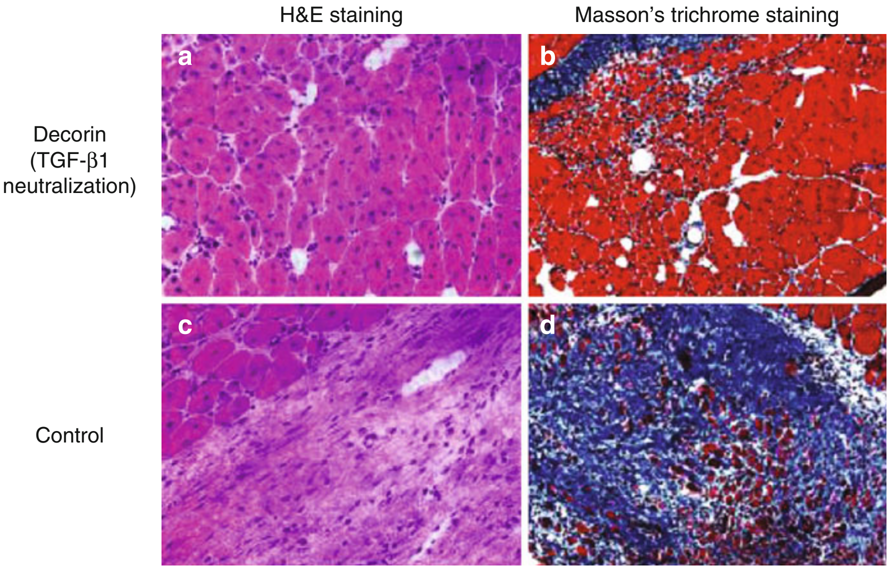

Fig. 2.2 Four weeks after injury, decorin-treated muscle (**a**, **b**) exhibits a greater number of regenerating myofibers (significantly higher numbers of centronucleated myofibers) and contained significantly less fibrosis (less collagen deposition, area in *blue*) than the control muscle (**c**, **d**) (Adapted from Li et al. [25])

give rise to clonal progeny with the ability to differentiate [46]. Isolation of muscle stem cells that can overcome these limitations would enhance the success of muscle cell transplantation significantly.

A population of murine muscle-derived stem cells (MDSC) displayed a high transplantation capacity in both skeletal and cardiac muscles [34, 37]. The MDSCs' ability to proliferate in vivo for an extended period of time combined with their capacity for long-term proliferation, strong capacity for self-renewal, resistance to stress, ability to undergo multilineage differentiation, and ability to induce neovascularization at least partially explains the high regenerative capacity of these cells in various musculoskeletal tissues including skeletal muscle [12, 34, 37]. Recently, it has been demonstrated that blood vessel progenitors (including myo-endothelial cells and pericytes) share a number of features with MDSC [9, 42]. In particular, they share cell-type marker profiles and have high myogenic potentials in vitro and in vivo. The use of such myogenic progenitors cells for improving muscle healing may become an interesting therapeutic alternative [8, 43, 44].

2.3.3 Antifibrotic Therapy

Some reports indicate that scar tissue formation precludes complete regeneration of muscle tissue. Although various studies have implicated TGF-β1 in the onset of fibrosis [24, 41], very few reports have examined the role of this cytokine in skeletal muscle fibrosis. It has been demonstrated that TGF-β1 is expressed at high levels and is associated with fibrosis in the injured skeletal muscle [6, 24]. These results support the hypothesis that TGF-β1 expression in skeletal muscle plays an important role in the fibrotic cascade that occurs after the onset of muscle injury. Therefore, neutralization of TGF-β1 expression in injured muscle could inhibit the formation of scar tissue. Indeed, the use of antifibrotic agents (i.e., decorin, relaxin, antibody against TGF-β1) that inactivate TGF-β1 appears to reduce muscle fibrosis and, consequently, improves muscle healing, leading to a near-complete recovery of the lacerated muscle [14, 25] (Fig. 2.2). Losartan, an angiotensin II receptor antagonist, has recently been demonstrated to neutralize the effect of TGF-β1 and reduce fibrosis, making it the treatment of choice, since it already has FDA approval to be used clinically [4, 35].

2.4 Clinical Implementation After Muscle Injury: From the Bench to the Sport Field

Muscle injuries constitute one of the most frequent sports lesions. Prevention of muscle strain includes proper conditioning and warm-up and good management of fatigue. However, most muscle strains occur in sports competition requiring velocity and force. Muscle injuries are currently identified as mild, moderate, and severe injuries based on muscle impairment (from few muscle fibers contusion to the entire muscle with complete loss of muscle function). In clinical practice, treatment regimens have been designed based upon empiricism and experience.

The objective in the treatment of a muscle strain is to create the best mechanical and biological environment to allow rapid and complete healing and thereby prevent a re-tear.

Treatment must start within minutes after the injury, following the algorithm known as PRICE (Protection, Rest, Ice, Compression, Elevation) to prevent further damage and limit hematoma formation. Protection is a crucial step for the first 2–3 days (crutches or even immobilization) to prevent excessive scar formation and re-rupture at the injury site. In the coming years, the use of IGF-1 injection may improve and accelerate the healing process. Recently, a new treatment approach came from basic science research. From days 3 to 5, the athlete is advised to perform a light exercise for 20′ per day (Fig. 2.3). Berg and Bang [5] have demonstrated a 27 % increase of IGF-1 after 10′ moderate exercise (10–28 μg/l), favoring thus the environment of the initial healing. Moreover, such exercise may increase satellite cell numbers and, thus, appear as an efficient strategy to improve muscle function and repair after injury [27].

After this protective phase, which can extend up to 5 days in severe injuries, controlled isometric, isotonic, and isokinetic contractions of the injured muscle group are performed with increasing intensity. At the same time, one should begin general reconditioning of the athlete, either by activation of the upper extremity in the presence of a lesion of the lower extremity or by activation of the contralateral limb. Reconditioning of the injured muscle group is mandatory. Gentle, progressive,

Fig. 2.3 From days 3 to 5 post-injury, the athlete performs 15–20 min of light exercises (50 % VO_2 max) using the uninjured limbs to enhance circulating IGF-1

and pain-free sports-specific reprogramming is rapidly begun. The criteria for time to return to sports include: (a) the ability to stretch the injured muscle as much as the contralateral healthy muscle, (b) pain-free use of the injured muscle in sports-specific movements, (c) comparable strength between injured and healthy muscles, and (d) the recovery of the proprioceptive and coordination capacity in the injured segment as well as the reprogramming of the sports movement. There is an obvious lack of evidence in determining these criteria, and these guidelines are mostly empirical.

In patients with a true muscle rupture, surgical reinsertion and repair should be considered, particularly with lesions in the proximal hamstrings or distal pectoralis major. The surgical management of these injuries permits a reduction in the length and degree of functional disability. The means to reduce the length of disability in athletes with muscle strains are the following: (a) Take them off the sports field; do not even permit them to play; (b) apply the proper treatment immediately and protect the injured muscle; (c) start controlled motion and general reconditioning; (d) recondition the injured muscle and rapidly begin sports-specific reprogramming; (e) surgically reinsert and repair a muscle rupture (especially hamstrings proximally); and (f) consider the use of hyperthermia which appears to be a promising technique to reduce the length of disability.

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Hamstring Muscle Injury

3

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Abstract

Hamstring muscle injury is common among athletes and can range in severity from mild strains to complete ruptures of the hamstring muscle complex. Injury to the hamstring muscles is commonly sustained during eccentric contraction of the muscle while running or stretching carried out to extreme joint positions. Diagnosis is based on patient history, physical examination, and imaging by means of ultrasound and MRI. The best way to treat a hamstring muscle injury depends on the extent, precise location, and function the muscle is required to regain. This chapter will address relevant anatomy, function, epidemiology, symptoms and signs, imaging, treatment, and prevention.

3.1 Introduction

The term "hamstrings" is derived from the butcher's practice of hanging a pig's ham by its distal tendons or "strings."

Hamstring muscle strains are common in many kinds of sports and often account for extensive absence from training and competition. Injuries range in severity from mild strains to complete ruptures of the hamstring muscle complex. This chapter will address relevant anatomy, function, epidemiology, symptoms and signs, imaging, treatment, and prevention.

3.2 Definition of the Injury

3.2.1 Anatomy

The hamstrings, or ischiocrural muscles, consist of the three muscles in the posterior thigh region that span both the hip and knee joints: semitendinosus, semimembranosus, and biceps femoris (long head) (Fig. 3.1) [18]. Originating from the ischial tuberosity, the semitendinosus and (long head of the) biceps femoris have a common proximal tendon. Anterior to that, the semimembranosus muscle originates on the ischial tuberosity as well. The semitendinosus and semimembranosus extend approximately 44.3 and 38.7 cm [25], respectively. They are attached to the medial condyle of the tibia via the superficial pes anserinus in case of the semitendinosus and to the posteromedial aspect of the medial condyle of the tibia in case of the semimembranosus.

The semitendinosus separates from its common origin with the biceps femoris at approximately 9 cm [25] from the ischial tuberosity at a greater pennation angle than the biceps femoris. A recent study suggested that this angle could make the proximal part of the semitendinosus susceptible to injury [5]. The semitendinosus muscle is divided into two separate parts by a tendinous section, known as the raphe, effectively making the semitendinosus a "digastric" muscle [15].

The long head of the biceps femoris muscle, after separating from its common origin with the semitendinosus muscle, is joined by its short head originating just medial to the linea aspera of the distal femur to traverse the knee joint at the lateral side and attach in a common tendon to the fibular head. Just proximal to the

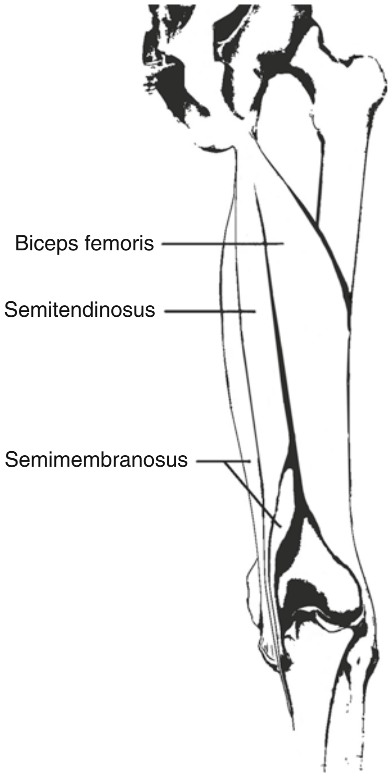


Fig. 3.1 Schematic view of hamstring muscle complex

fibular head, this common tendon is divided into two components, namely, a direct arm and an anterior arm, and several fascial components. The direct arm is inserted on the posterolateral edge of the fibular head, whereas the anterior arm is inserted on the lateral edge, its lateral side terminating as an aponeurosis covering the anterior compartment of the leg. Other insertions of the short head include attachments to the posterolateral joint capsule and to the iliotibial tract [23]. The long and short head of the biceps femoris extend approximately 42.0 and 29.8 cm, respectively [25].

The tibial part of the ischial nerve supplies nerves of the semitendinosus, semimembranosus, and long head of the biceps femoris, whereas the short head of the biceps is innervated by the peroneal part of the ischial nerve. This difference in innervation is often thought to contribute to difficulty in coordination that might lead to injury within the biceps femoris muscle [15].

3.2.2 Function

The hamstring muscles are extensors of the hip and flexors of the knee, absorbing kinetic energy and protecting the hip and knee joints during the gait cycle by limiting knee extension just before and during heel strike. During the gait cycle, there is interplay between the hamstrings and the quadriceps in an antagonizing way [18].

3.2.3 Injury Mechanism

Hamstring muscle injury occurs during eccentric contraction rather than concentric contraction. Eccentric contractions take place in a state of muscle stretching, in which the contracting muscle fibers put even more tension on the stretched muscle. Because the prime function of the hamstring muscle complex is to absorb kinetic energy by eccentric contraction, it is vulnerable to strain injury, especially the region adjacent to the MTJ (musculotendinous junction) [15, 18].

Recent studies pointed out that different sports lead to lesions in different regions of the hamstrings muscle complex through different injury mechanisms. Two distinct types of injuries are known: the high-speed running type [3] and the stretching type [4]. The high-speed running type, incurred during sports as football or athletics, is strongly associated with injuries in the long head of the biceps femoris muscle [3]. Injuries of the stretching type occur during stretching exercises carried out to an extreme joint position (hip flexion and knee extension), as is the case with dancing or kicking during rugby. They are often complex, but mainly involve the semimembranosus muscle and its proximal free tendon close to the ischial tuberosity [4]. These types of injury have different prognoses, which is illustrated by the time that is needed to recover to pre-injury level, which is longer for the stretching type [2].

Apart from the injury mechanism, the precise location of the injury within each muscle also corresponds with different prognoses. Hamstring muscle strains, generally occurring at the proximal sections of a muscle, prove to be more problematic as they are located closer to their origin at the ischial tuberosity [1]. Involvement of the free proximal tendon is also associated with longer time to return to the pre-injury level [3].

3.3 Epidemiology

Incidence rates of hamstring muscle injury vary widely, depending on the type of competition an athlete competes in. In soccer, the incidence rate is 52/1,000 players per year [9]. In rugby, incidence rate has been reported as injuries per 1000 player training or match hours. The incidence of hamstring muscle injury is 0.27/1,000 player training hours and 5.6/1,000 player match hours [6]. Similarly, In the National Football League (NFL), also known as American Football, incidence is calculated as injuries per athlete exposure (AE), with every training and match counting as an exposure. The total injury rate in the NFL for hamstring muscle injuries is 0.77/1,000 AE, 0.47/1,000 AE during practice, and 2.70/1,000 AE during matches [11]. Overall, the biceps femoris muscle is most often injured, followed by the semitendinosus muscle and the semimembranosus muscle [15, 16].

3.4 Symptoms and Signs

While people with a mild hamstring lesion seldom seek medical attention, athletes incurring a moderate or severe injury will typically do so. The patient's history will relate a sudden onset of pain in the posterior thigh region, most likely during sprinting or extreme stretch. Hamstring muscle injuries of the high-speed running type occur during sprinting when speed is (close to) maximal, causing the athlete to stop running at once [3]. Injuries of the stretching type are often experienced as acute pain close to the ischial tuberosity, accompanied by an audible "pop" [4]. Predisposing factors include inadequate warm-up, muscle fatigue, poor muscle strength, poor lumbopelvic strength and stability, older age, muscle imbalance between quadriceps and hamstrings, muscle imbalance between left and right hamstrings, previous hamstring muscle injury, and L5 nerve root entrapment [18].

3.4.1 Physical Examination

Although the value of a physical examination in (suspected) hamstring muscle strains remains controversial [1], expert opinion and a review of the available literature seem to agree on several issues. The initial assessment should take place within 2 days post-injury to ensure a reliable medical history and possibility of a quick intervention when necessary, yet allow possible signs of swelling and hematoma to develop [14].

Inspection should mainly focus on posture and gait examination, focal swelling, and hematoma. Both inspection and palpation of the injury site can help identify the muscle involved as well as determine the proximity of the injury. For instance, in case of a true hamstring avulsion, extensive bruises can be seen on the posterior thigh and a defect in the tendon may be felt if there has been sufficient retraction [8].

During palpation, athletes with hamstring injuries of the high-speed running type report their highest pain in the lateral part of the posterior thigh, about 11–12 cm (±7, 4–24) distal to the ischial tuberosity. With this type of injury, there is a significant correlation between the location of the point of highest pain and the length of the convalescent period. The more proximal the point of highest pain, the longer the convalescent period. Length of the painful area however does not correlate with the duration of the convalescent period [3].

Athletes with the stretching type hamstring injury report their highest pain in the proximal posterior thigh, 2 cm (±1, 0–5) distal to the ischial tuberosity. The convalescent period of the stretching type injury does not correlate significantly with the location of the highest pain [4].

Despite possible inaccuracies due to pain, range of motion (ROM) testing should reveal decreased flexibility in the affected leg. The best way to establish a deficit in the posterior thigh region is by using passive or active straight leg raise and passive or active knee extension tests. The "sit-and-reach" test should be omitted from standard ROM testing due to confounding factors such as spinal mobility (i.e., lumbar flexion), leg length, scapular abduction, and stretch on the peripheral nerves by dorsiflexion of the ankle joint.

With the patient lying supine, the strength of the hamstring muscles can be examined by comparing knee flexion and hip extension between both legs. In this way, a possible decrease in strength of the affected leg can be determined, although it is good to realize that this could also be caused by pain instead of fiber disruption [2]. The use of the "hamstring drag" test remains controversial and needs further validation [14].

Table 3.1 Clinical classification for hamstring muscle injury [1]

Classification	Degree	Symptoms	Architectural damage
Mild	First	Minor discomfort, minor swelling, no/minimal loss of strength, and restriction of movement	Microscopic muscle fiber disruption
Moderate	Second	Marked discomfort, diminished ability to contract	Macroscopic muscle fiber disruption
Severe	Third	Distinct pain, total loss of muscle function	Complete rupture of muscle-tendon unit, possibly with avulsion fracture

3.4.2 Clinical Classification

As with other muscle injuries, hamstring strains are conventionally classified according to the impairment they cause [1], which generally is a direct consequence of the extent of damage to the muscle-tendon unit. In this way, hamstring strains are defined as mild (first degree), moderate (second degree), or severe (third degree) [1] (Table 3.1). This classification system, however, is quite indistinct and does not take into account that the precise location of a hamstring injury can influence the duration of symptoms and time to return to full activity to a large extent. A minor tear in the proximal biceps femoris, for example, can have greater consequences than a larger injury located more distally [1]. Preferably therefore, a future clinical classification system will take these factors into account.

3.5 Imaging

Imaging can provide detailed information on the nature and extent of hamstring muscle injuries [15]. Magnetic resonance imaging (MRI) and ultrasound (US) are the modalities of choice [14, 15]. Nevertheless, MRI is preferred over US because it is more sensitive in identifying minimal injury [14]. Moreover, it is more difficult to use US to accurately assess an injury that is located deeper within the muscle, and its interpretation is subject to greater interobserver variability [13, 15]. Edema and hemorrhage along disrupted muscle fibers show as a hyperintensity in a characteristic feather-shaped appearance on STIR (short tau inversion recovery) and T2-weighted MR images [15]. Because the amount of edema reaches its maximum after 24 h and decreases after 48 h, MRI is preferably performed 1–2 days post trauma [10, 13, 14]. To grade hamstring muscle injury based on radiological findings, Peetrons [19] classification modified for MRI [10] is reproduced below (Table 3.2).

In professional football, radiological grading is highly associated with the duration of the convalescent period and correlates with clinical severity. This indicates that an MRI examination can be used to verify the diagnosis, provide an accurate prognosis regarding convalescent period, and monitor healing strains [10, 15, 16, 18, 22].

Table 3.2 MRI classification of hamstring muscle injury [10, 19]

Grade	Findings	Clinical equivalent
0	No visible pathology	Mild
I	Edema without architectural distortion, <5 % of muscle volume involved	Mild
II	Partial rupture with 5–50 % of muscle volume involved	Moderate
IIIa	Complete rupture (Fig. 3.2a– c)	Severe
IIIb	Complete rupture with avulsion fracture	Severe

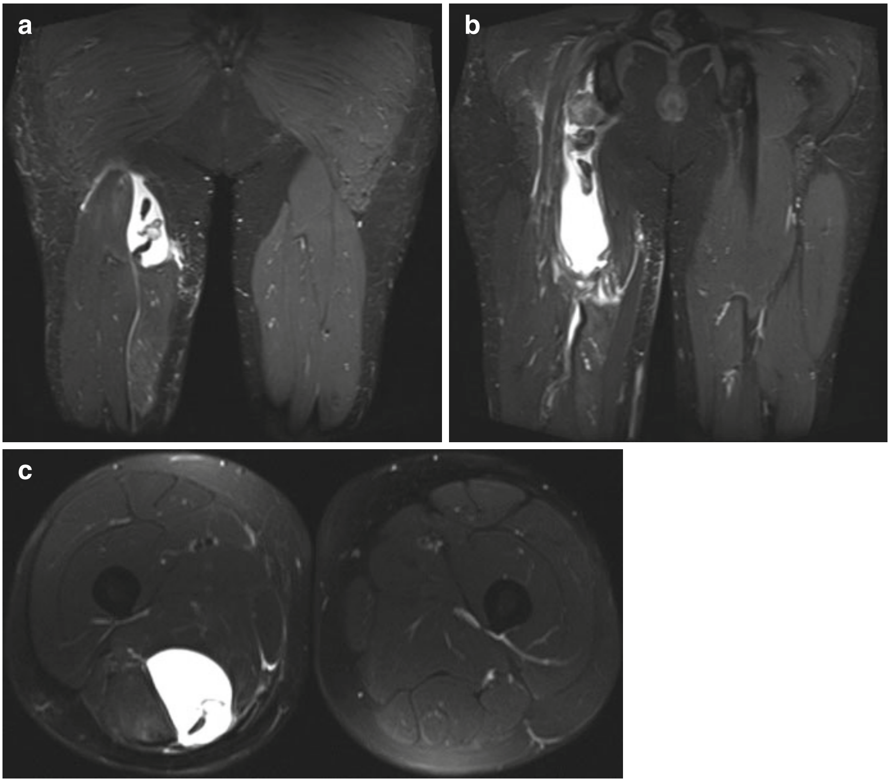


Fig. 3.2 (**a**) Preoperative MRI (coronal T2 fat sat) of a proximal avulsion of the semitendinosus muscle with hematoma. (**b**) Preoperative MRI (coronal T2 fat sat) of a proximal avulsion of both the semitendinosus muscle and biceps femoris muscle (long head) with hematoma. (**c**) Preoperative MRI (axial T2 fat sat) of a proximal avulsion of both the semitendinosus muscle and biceps femoris muscle (long head) with hematoma

The greater part of hamstring injuries are grade 0 or grade 1, without any signs of fiber disruption on MR images [10]. Even though only 26 % of hamstring injuries are objectified by imaging at the initial assessment, nearly 90 % of clinicians use imaging to try and diagnose a hamstring injury in (elite) athletes. When imaging is requested, MRI is used in 40–58 %, US in 29–53 %, and both MRI and US in 7–40 % of cases [14].

3.6 Treatment

The best way to treat a hamstring muscle injury depends on the extent, precise location, and function the muscle is required to regain.

3.6.1 Nonoperative

Mild or moderate hamstring strains are to be treated conservatively [8, 13]. The treatment consists of a phased rehabilitation program [13, 24], as shown in Table 3.3. The first phase focuses on hamstring protection and consists of rest to avoid stretching of the hamstrings and application of ice to minimize pain and inflammation, followed by low intensity pain-free exercises within a limited ROM. These low intensity exercises involve the entire lower extremity and lumbopelvic region in order to develop neuromuscular control and minimize atrophy. Avoiding stretching of the hamstring prevents the development of dense scar tissue that can prohibit muscle regeneration [13].

Table 3.3 Rehabilitation schedule [13, 21]

Phase	I	II	III
Rehabilitation goal	Hamstring protection	Normalize gait Regain of strength Regain of neuromuscular control	Return to sports/work with good control and no pain
Restrictions	No pain No hip flexion coupled with knee extension Limited ROM	No dynamic stretching No running or impact Limited ROM (avoid end range lengthening)	No sprinting or explosive acceleration movements
Exercises	Balance exercises Isometric exercises of lumbopelvic musculature (core stability)	Neuromuscular control exercises Agility drills Core stability exercises (Submaximal) eccentric strengthening exercises	Sport-specific drills Impact control exercises Agility drills Core stability exercises Eccentric strengthening exercises, with increasing resistance
Interventions	Crutches Application of ice postexercise	Application of ice postexercise	Application of ice postexercise
Progression criteria	Normal gait without pain Low speed jogging without pain Isometric contraction against resistance without pain	Full strength during 1 repetition isometric manual muscle test (prone) without pain Forward/backward jogging at 50 % of maximum speed, without pain	Full ROM, strength and regain of functional abilities without pain or stiffness

Further phases focus on allowing the muscle to heal and regain proper function as rapidly as is reasonably achievable [13]. Phase II consists of therapeutic exercises targeting neuromuscular control, agility, core stability, and eccentric strengthening with increasing speed and intensity [13, 21]. These exercises should be started as soon as the patient is able to walk, jog at very low speed, and perform isometric contraction against resistance without pain. Exercise intensity and ROM are increased based on tolerance and improvement of the patient [13]. It deserves mention that despite a lack of evidence, electrical stimulation is sometimes used. Because stretching exercises may lead to re-injuries [21], they should be performed while avoiding end range lengthening of the hamstrings [13]. Phase III is started when full strength during a 1 repetition maximum effort isometric manual muscle test is reached and the patient is able to jog forward/backward at 50 % of maximum speed. It focuses on return to sports/work, with sport-specific exercises, impact control exercises and increasingly challenging agility drills, core stability exercises, and eccentric strengthening exercises. ROM is no longer limited, but sprinting and explosive acceleration movements should be avoided. Phase III is continued until return to sports/work with full strength, ROM, and regain of functional abilities without pain or stiffness is achieved. Application of ice after rehabilitation exercises is advised to help decrease eventual pain and inflammation [13].

To avoid re-injury, the rehabilitation program will also focus on balancing strength in opposing limbs and muscle groups. High-level clinical trials are needed to further validate the effect of all exercises in the rehabilitation program [20, 21].

Limited evidence suggests that local administration of autologous blood products containing platelet-derived growth factors may enhance healing of muscles and tendons. This platelet-rich plasma (PRP) is best administered by ultrasound-guided injection and is recognized by the World Anti-Doping Agency (WADA) as treatment for tendinopathies while intramuscular injections remain prohibited [18].

Both Actovegin [18, 21] (deproteinized calf blood hemodialysate) and Traumeel S [18] (homeopathic formulation) injections are increasingly popular for the management of acute muscle strains. However, evidence to support the use of these substances is limited to low-quality reports. Controlled trials are needed for confirmation [18, 21].

Non-steroidal anti-inflammatory drugs (NSAIDs), despite their widespread use, have not been proven effective in management of both pain and hamstring function. NSAIDs should not be administered due to the possibility of adverse effects [13, 18, 21].

Intramuscular glucocorticoids are also not recommended. Intramuscular use has been associated with atrophy, disruption of normal muscle architecture, and reduction in tendon collagen strength. Intramuscular use may lead to negative long-term consequences on both muscle regeneration and function [18].

3.6.2 Operative

Surgical treatment is reserved for severe hamstring injuries like avulsions and extensive/recurrent hamstring strains that are resistant to conservative therapy [7, 8, 12, 16]. For avulsions, a reattachment procedure is needed to achieve proper function in the affected leg. Compared to a conservative approach, surgical repair of tendinous avulsions leads to higher patient satisfaction, higher rate of return to pre-injury level, and greater strength/endurance [12], whereas a conservative approach is associated with functional impairment for a longer period of time [7]. Acute surgical repair is preferred over delayed repair, with higher patient satisfaction, pain relief, strength/endurance, a higher rate of return to pre-injury level, and lower rates of complication and re-rupture [12]. Besides better results, delayed surgical repair is known to be technically more challenging than acute surgical repair [8]. Not infrequently, the ischial nerve lies in the middle of major scar tissue and must be meticulously dissected using a surgical microscope and a nerve stimulator [8].

Surgical repair consists of the use of suture anchors or an interference screw. Auto- or allografts can be used in cases where a large defect between the tendons and the ischial tuberosity prevents anatomic reinsertion [17].

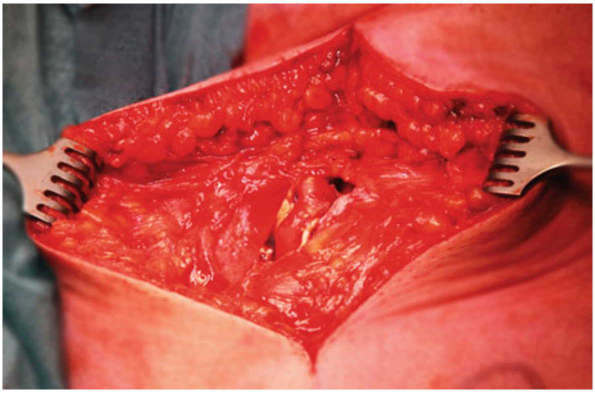
Postoperative rehabilitation is similar to a conservative approach. The difference lies in an additional phase. The postoperative rehabilitation program consists of four phases and is not started until 7–10 days after surgery. Phase I focuses on protecting the repaired hamstring and consists merely of ROM exercises and gait training. As with the conservative approach, application of ice after rehabilitation exercises is advised to help decrease eventual pain and inflammation. Phase II is started 6 weeks after surgery and aims at progression of both speed and amplitude of movement. Core stability and hamstring strengthening exercises are started. Exercises are done with both legs simultaneously in a short arc of motion either at the hip or knee, thereby avoiding lengthened hamstring positions. This means that only concentric and isometric exercises can be performed. Impact and running should also be avoided. Phase II is completed when gait is normalized and functional movements are controlled and painless. Phase III also aims at progression of both speed and amplitude of movement, now one leg at a time with force occurring at both the hip and knee simultaneously. Core stability and hamstring strengthening exercises are continued, carefully supplemented with eccentric strengthening exercises, impact control exercises, and running drills. Phase III is completed when dynamic neuromuscular control is regained and multiplane activities can be carried out without pain or swelling. Also, deficit in leg comparison on Biodex testing (60° and 240° per second) should be less than 25 %. Phase IV is then started and focuses on return to work/sport. All core stability, hamstring strengthening exercises, impact control exercises, and running drills are continued. Sprinting drills, sport-/work-specific balance drills, and proprioceptive drills are started, as well as stretching exercises to correct muscle imbalances. Phase IV is completed when sport-/work-specific movements can be carried out at high velocity with good control and without pain. Also, deficit on both Biodex testing and functional testing profile should be under 10 %. If these criteria are met, the rehabilitation program has successfully been completed [24].

3.6.3 Surgical Technique

The patient should be in prone position, fully anesthetized (Fig. 3.3). The incision is made in line with the hamstring muscle tendons, extending distally from the ischial

Fig. 3.3 Preparations for surgery with patient in prone position

Fig. 3.4 Spreading of craniocaudal incision, incised through dermis, subcuticular tissue, and fascia



tuberosity (approximately 5–7 cm) (Fig. 3.4). A larger incision may be needed to identify retracted muscles. A horizontal incision in the gluteal rim results in a more cosmetically acceptable scar but makes surgery more challenging [8]. The inferior edge of the gluteus maximus is retracted proximally, and the deep fascia of the posterior thigh is subsequently incised parallel to the skin incision. After palpation of the ischial tuberosity, the retracted tendons and the sciatic nerve are identified and mobilized (Figs. 3.5, 3.6, and 3.7) [8]. The ischial tuberosity is then exposed and cleared of scar tissue to insert suture anchors into the tuberosity (Figs. 3.8, 3.9, 3.10, and 3.11) [8, 24]. Sutures are used to attach the retracted tendons to the suture anchors (Fig. 3.12) [8, 24] using a modified Mason-Allen technique. The wound should then be closed in layers, with subcuticular skin closure [8]. Figure 3.13 shows the scar 6 weeks postoperatively. The postoperative position of the suture anchors can be verified by an x-ray (Fig. 3.14).

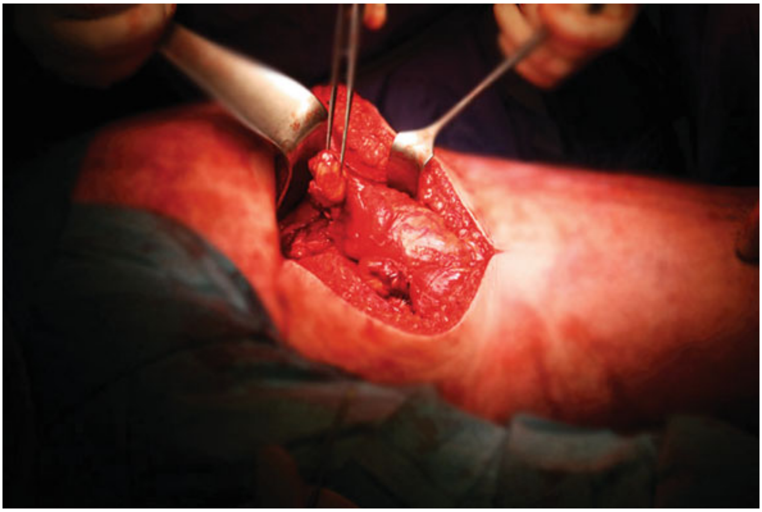


Fig. 3.5 Inspection of posterior thigh compartment, with avulsed hamstrings displayed (both a vertical and horizontal incision was used to be able to identify the avulsed hamstrings)

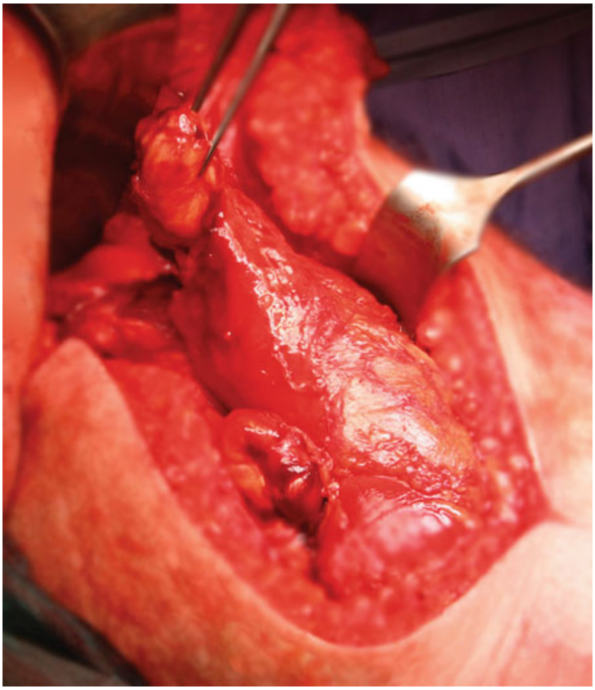


Fig. 3.6 Mobilization of avulsed biceps femoris muscle (long head)

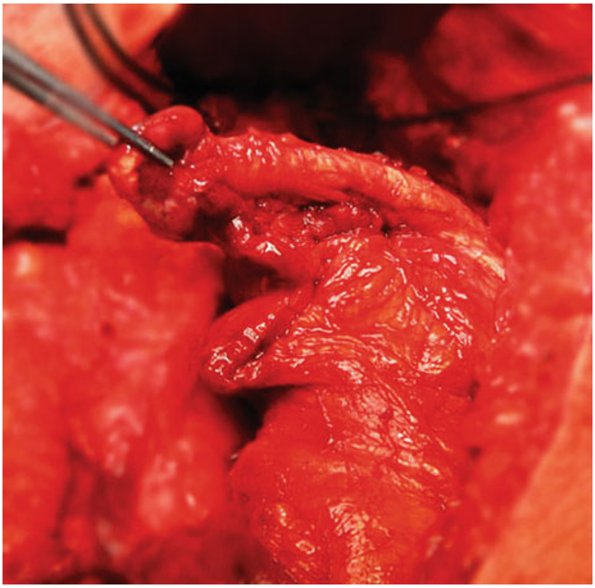


Fig. 3.7 Closer inspection of avulsed biceps femoris muscle (long head)

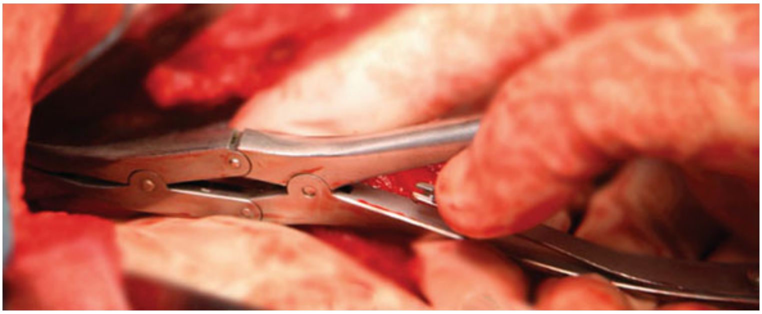


Fig. 3.8 A bone nibbler is used to prepare the ischial tuberosity for reattachment of the avulsed tendons

Fig. 3.9 A surgical drill is used to drill holes for the suture anchors

Fig. 3.10 Ischial tuberosity with holes for suture anchors

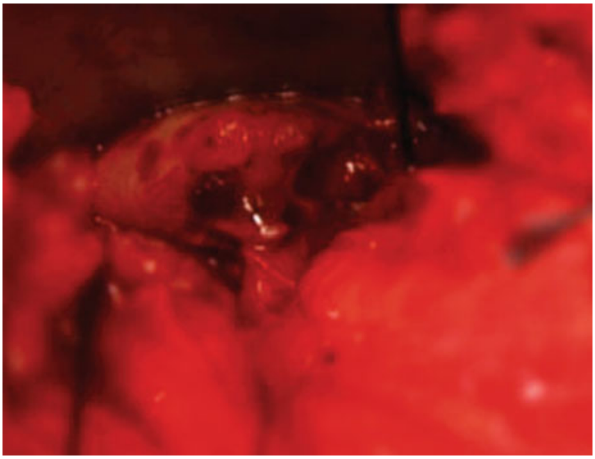


Fig. 3.11 Placement of the suture anchors

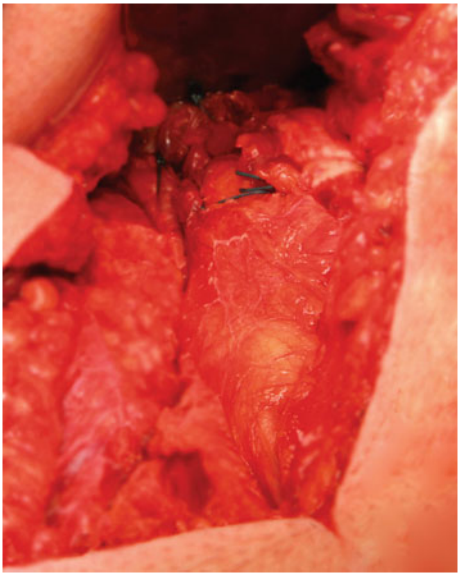


Fig. 3.12 Reattached hamstrings in situ

Fig. 3.13 Scar 6 weeks after operation

Fig. 3.14 Postoperative x-ray to ensure the correct position of the suture anchors (3pcs) into the ischial tuberosity

3.6.4 Findings at Surgery

As mentioned above, surgical repair is mainly reserved for bony or tendinous avulsions. Logically, the tendons are retracted and may be bound by a pseudocapsule or scar tissue. This pseudocapsule is formed by the remains of a hematoma in an acute setting. Scar tissue is formed over a longer period of time in cases where surgery has been delayed. Because the sciatic nerve lies alongside the hamstring origin and muscle bellies, there is risk of entrapment of the nerve by scar tissue. In that case, neurolysis during surgery is required for symptom relief and prevention of iatrogenic injury. Due to this scar tissue, surgery will be substantially more difficult and time consuming than early surgery. On the other hand, if surgery is performed too soon, the surrounding tissue will be edematous and fragile, also resulting in a difficult repair [8].

3.7 Prevention

Hamstring muscle injuries account for extensive absence from competition and training. Prevention of these injuries can therefore be extremely valuable, both to (elite) athletes and their clubs.

Hamstring flexibility programs do not lead to a decrease in incidence of hamstring muscle injuries, whereas eccentric hamstring exercises do lead to a substantial reduction in incidence of hamstring muscle injuries [13, 20]. Other risk factors that can be dealt with are insufficient warming-up and muscle fatigue [20]. Although studies point in the same direction, these findings cannot be generalized due to a lack of high-level clinical trials.

Re-injuries occur frequently and should be prevented by a proper rehabilitation program [13].

Conclusion

Hamstring muscle injury is common in many kinds of sports and often accounts for extensive absence from training and competition. Injury is commonly sustained during eccentric contraction. It is most frequently located proximally and can be divided into the high-speed running type and the stretching type injury. Physical examination should take place within 2 days post-injury and may reveal bruising, focal swelling, pain, and a decrease in both muscle strength and range of motion. MRI and ultrasound can provide detailed information on the nature and extent of the injury. MRI is the best choice to confirm the diagnosis, provide an accurate prognosis, and monitor healing strains. Mild or moderate hamstring strains are preferably treated conservatively, whereas surgical treatment is reserved for severe hamstring injuries including avulsions and recurrent hamstring injuries that are resistant to a conservative approach. Adequate warming-up and eccentric stretching exercises lead to a decrease in risk of injury, while muscle fatigue increases that risk.

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Acute Adductor Muscle Injury

4

Tomasz Piontek, Kinga Ciemniewska-Gorzela, Marcin Dzianach, and Andrzej Szulc

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Abstract

The main sign of the adductor muscle injury is intense pain in the groin area. The diagnosis of adductor syndromes is sometimes difficult to determine, as numerous patients may experience symptoms similar to those of adductor muscle injury.

In this chapter, we give a detailed overview of thigh adductor muscle-tendon complex injuries (adductor syndrome), which are typically acute in character. The main signs, imaging techniques, and differential diagnosis of groin pain in athletes have been described. The role of surgery and recommended procedures for specific pathological conditions have been presented.

4.1 Introduction

Tomasz Piontek

Musculotendinous injuries to the groin are the most common cause of groin pain in athletes [6]. These injuries are mainly a consequence of cumulative microtraumas (overuse trauma, repeated minor injuries), leading to chronic groin pain. Although, in some cases, groin pain is due to acute injury. A direct injury to the soft tissues typically results in muscle hematoma. The underlying injury is most often a muscle or tendon strain at the tendinous insertion of the adductor muscles to the bone. A chronic tendinitis of the adductor muscles/tendons, especially that of the adductor longus, is the most frequently diagnosed cause of groin pain. Acute tears of the adductor tendons are a rarely reported injury, and the best treatment for this injury is unknown.

This chapter focuses on the thigh adductor muscle-tendon complex injuries (adductor syndrome), which are typically acute in character.

4.2 Definition of the Injury

Andrzej Szulc

4.2.1 Anatomy

The hip adductors comprise the adductor longus (AL), adductor magnus (AM), adductor brevis (AB), gracilis (GR), and pectineal muscles. The adductors are composed primarily of superficial (pectineus, gracilis, and adductor longus), middle (adductor brevis), and deep (adductor magnus) muscle structures. As shown in Figs. 4.1, 4.2 and Table 4.1, the adductors all originate on the pubic ramus and almost all insert on the linea aspera of the posterior femur [12]. The posterior head of the adductor magnus has a proximal attachment on the ischial tuberosity anteroinferiorly and attaches distally on the medial distal femur at the adductor tubercle. The gracilis insertion is on the medial border of the tuberosity of the tibia [3, 18].

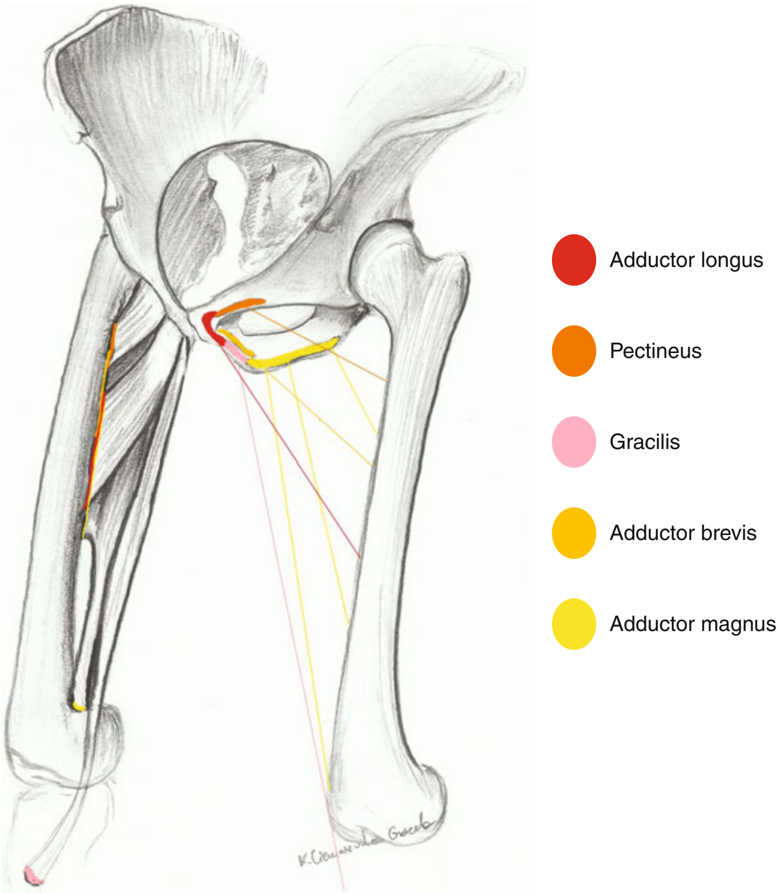

Fig. 4.1 Anterior view of muscle origins and insertions. Note that all adductors originate on the pubic ramus. The adductors all insert on the linea aspera of the posterior femur. Anterior view of the pubic ramus with schematic direction of the many forces acting on the pubic joint. Each muscle is highlighted with another color according to the legend

4.2.1.1 Vascularity

The AL muscle usually attaches to the pubic bone through a tendinous insertion, but occasionally this tendinous unit is very short, or the muscle may even insert directly into the pubic bone [12]. In such cases, the proximal tendons of AB and gracilis are fused, forming a common tendinous insertion. The AL and AB both contain extensive intramuscular tendons of variable length. The vascularity of AL and AB tendons decrease significantly toward the enthesis, and their entheses were significantly less vascular than those of gracilis. The arrangement and fusion of these muscles, their fibrocartilaginous entheses, and differences in the vascularity of their proximal tendons may be important anatomical considerations in the pathogenesis and pattern of adductor-related groin pain [7].

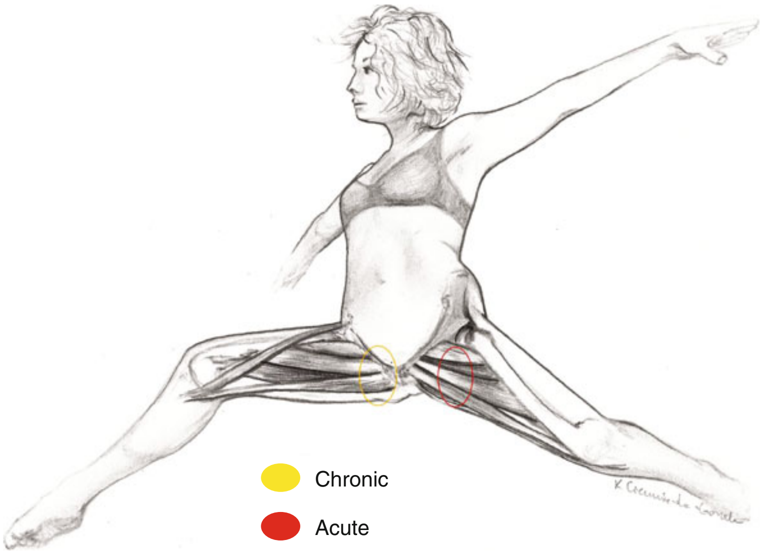

Fig. 4.2 A different type of stress injury is thought to be related to repetitive avulsive stresses at tendinous insertions on a bone, leading to a traction periostitis at these sites. This situation is also localized around the pubic ramus and can cause chronic adductors injury (*yellow ellipse*). The acute adductor injuries are most often localized in the myotendinous junction (*red ellipse*)

4.2.1.2 Innervation

The anterior head of the adductor magnus (as well as other muscles in the adductor group) is innervated by the obturator nerve. The posterior head is innervated by a branch of the sciatic nerve, similar to the hamstrings, and functionally resembles a hamstring muscle [3].

4.3 Symptoms and Signs

Kinga Ciemniewska-Gorzela

4.3.1 History and Physical Examination

Patients often describe lower abdominal pain when exerting themselves physically, particularly when kicking, skating, shooting, and sprinting [10, 11]. It has been established that 70 % of patients remember a distinct injury during exertion that resulted from trunk hyperextension and thigh hyperabduction [11, 12]. The same patients will describe pain with coughing, sneezing, or other Valsalva-type maneuvers; this is more indicative of an inguinal hernia. The diagnosis of adductor syndromes is sometimes difficult to determine, as numerous patients may experience symptoms similar to those of adductor muscle injury. The most frequent diagnoses associated with groin pain are listed in Table 4.2 [9, 16]. This type of pain is characteristic in chronic cases;

Table 4.1 The functions of adductor muscles are described

	Origin	Insertion	Action
Pectineus	Pecten pubis	Pectineal line and proximal linea aspera of the femur	Adduction, external rotation, and slight flexion of the hip joint Stabilizes the pelvis in the coronal and sagittal planes
Adductor longus	Superior pubic ramus and anterior side of the symphysis	Linea aspera: medial lip on the middle third of the femur	Adduction and flexion (up to 70°) of the hip (extends the hip past 80° of flexion) Stabilizes the pelvis in the coronal and sagittal planes
Adductor brevis	Inferior pubic ramus	Linea aspera: medial lip on the upper third of the femur	Adduction and flexion (up to 70°) of the hip (extends the hip past 80° of flexion) Stabilizes the pelvis in the coronal and sagittal planes
Adductor magnus	Inferior pubic ramus, ischial ramus, and ischial tuberosity	Deep part ("fleshy insertion"): medial lip of the linea aspera Superficial part ("tendinous insertion"): medial epicondyle of the femur	Adduction, external rotation, and extension of the hip joint (the tendinous insertion is also active in internal rotation) Stabilizes the pelvis in the coronal and sagittal planes
Adductor minimus (upper division of the adductor magnus)	Inferior pubic ramus	Medial lip of the linea aspera	Adduction, external rotation, and slight flexion of the hip joint
Gracilis	Inferior pubic ramus below the symphysis	Medial border of the tuberosity of the tibia (along with the tendons of the sartorius and semitendinosus)	Hip joint: adduction and flexion Knee joint: flexion and internal rotation

the symptoms of groin pain are complex and nonspecific. Pain can be more or less diffuse, localized over the tendon area of the adductor longus. Pain can also diffuse along the medial side of the thigh or toward the rectus abdominis at the hip joint [9, 14]. In acute grades I and II strains of the adductor muscle, there is intense pain in the groin area. Hemorrhage and swelling can be seen a few days after the injury. Complete muscle tears (grade III) are most often found in the distal musculotendinous junction located in the proximal part of the muscle and toward the insertion on the femur (Fig. 4.2) [9, 14]. Acute ruptures are seen often after a long history of groin pain,

Table 4.2 Differential diagnosis of groin pain in athletes

Localization	Differential diagnosis
Muscle, tendons	Tendinitis of adductors, partial or complete tear of adductors, tendinitis, or tear of rectus femoris Ileopsoas tendinitis, tear (snapping hip), rectus abdominis muscle tear, external oblique muscle tear, fascia tear
Hip joint	Labral tear, chondral lesions, FAI – femoral acetabular impingement, loose bodies, synovitis, osteonecrosis
Symphysis	Osteitis pubis, symphysis disruption
Bursae	Iliopsoas bursitis (snapping hip), bursitis to rectus femoris muscle insertion
Hernias	Inguinal hernia, femoral hernia
Nerve entrapments/irritations	Lateral cutaneous femoral nerve, femoral nerve, obturator nerve, ilioinguinal nerve, genitofemoral nerve, perineal nerves, Th–L2 spinal nerve (facet syndrome, lateral stenosis, anomaly, disc injury, spondyloarthrosis)
Fractures	Compression fractures (Th XII–L2), posterior vertebral apophysis fracture with nerve compression, pelvic fractures, symphysis disruption, avulsion fracture of the anterior iliac spine, lower corner of the symphysis pubis joint, stress fractures of the femoral neck, pubic arches, subtrochanteric femoral shaft fracture
Ectopic calcifications	Insertion of adductor muscles, anterior and inferior superior iliac spine, insertion of the rectus femoris muscle, calcifications in the muscle after injury
Tumors	Prostate cancer, metastasis of the spine and pelvis, osteoid osteoma, lymphoma, osteosarcoma
Infections	Inguinal lymphadenopathy, urinary tract infections, appendicitis, osteomyelitis

especially after adductor tendinitis has been diagnosed [4]. These strains are usually easily diagnosed on physical examination by pain on palpation of the involved muscle and pain on adduction against resistance. If the adductor longus muscle is injured, pain will be transmitted to the injured area resisting leg adduction and in passive stretching at full abduction of the hip [9]. These strains must, however, be distinguished from osteitis pubis and "sports hernias" and others disorders shown in Table 4.1, which can present with pain in similar locations.

4.4 Imaging

Marcin Dzianach

Imaging techniques, including plain radiographs, MRI, and ultrasound (US), provide a more comprehensive view of the injured area. Plain radiographs are useful for the differential diagnosis of groin pain, for example, in detecting stress fractures or

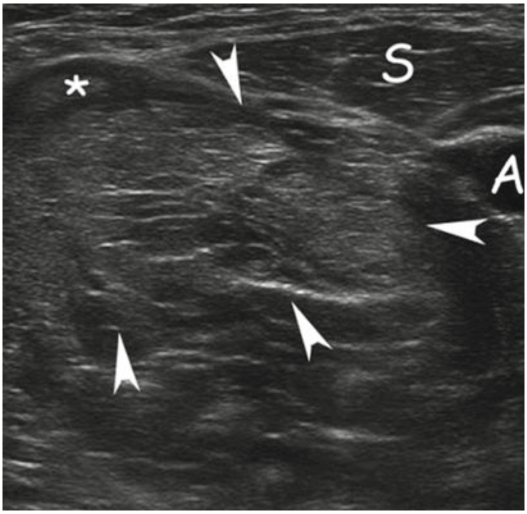

Fig. 4.3 Acute grade I tear (strain) of the myotendinous junction of the superficial part of the distal aponeurosis of the adductor longus. Sonographic transverse scan on the level of the distal part of the adductor longus. Between *arrowheads* there is affected, swollen part of the muscle belly with subtle fluid collection located in the area of the myotendinous junction (*asterisk*). *S* sartorius muscle, *A* femoral artery

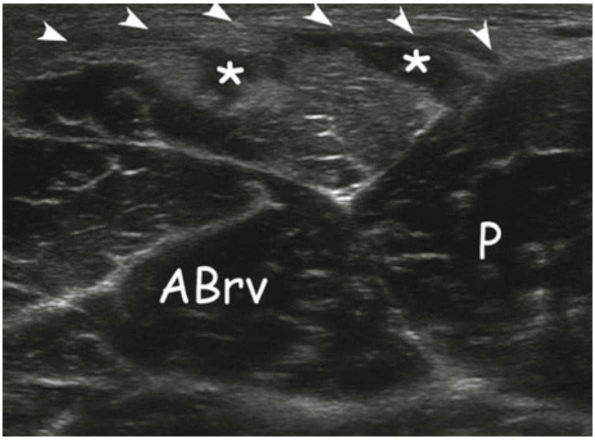

Fig. 4.4 Acute grade II tear (partial tear) of the myotendinous junction of the proximal tendon of the adductor longus. Sonographic transverse scan on the level of the proximal part of the adductor longus. *Arrowheads* indicate localization of the myotendinous injury with hematoma (*asterisks*) and extensive muscle belly edema. *ABrv* adductor brevis muscle, *P* pectineus muscle

avulsion fractures [9, 17]. US is also helpful in the diagnosis of adductor injury and chronic groin complaints (Figs. 4.3, 4.4, 4.5, 4.6, and 4.7). Tendon injuries, especially partial tears, are well demarcated [8]. Muscle injuries are diagnosed based on bundle disruption and fluid accumulation [9]. Scarring and fibrosis of muscle-tendon tissues have characteristic features that are easily demonstrated by US examination. US may also provide valuable information before surgery, such as the exact extension and location of the tendinous injury, especially in case of partial rupture [19] (Figs. 4.3 and 4.4). MRI presents significant advantages due to the high contrast and therefore resolution of soft tissues and the possibility of direct multiplanar imaging of bone and soft tissues (Figs. 4.5 and 4.7) [3].

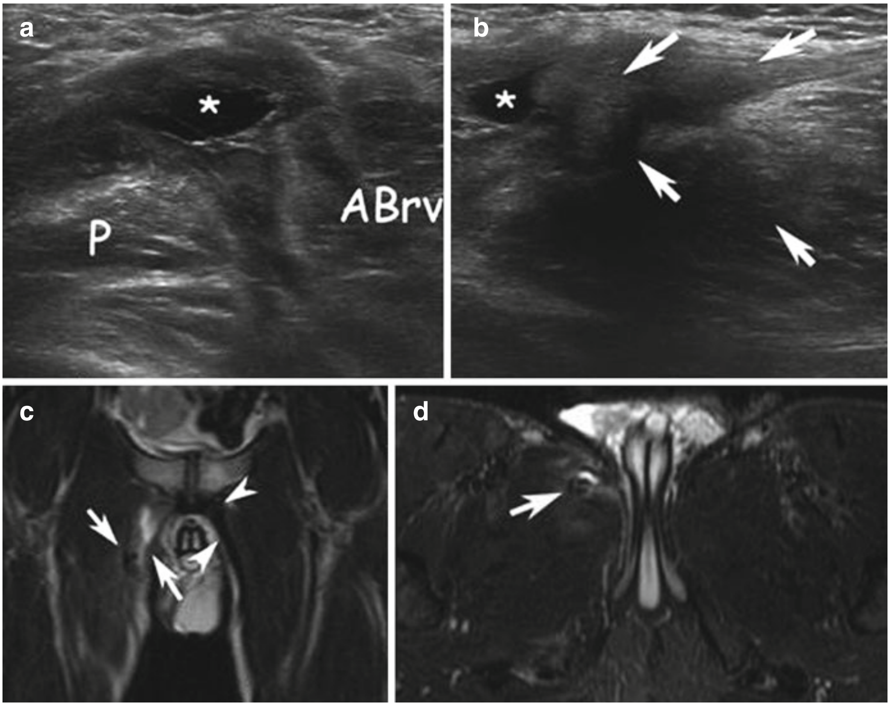

Fig. 4.5 Acute grade III tear (complete tear) of the tendon of the adductor longus. Sonographic scans, transverse (**a**) and longitudinal (**b**), on the level of the proximal part of the adductor longus show hematoma (*asterisk*) in the space between adductor brevis (*ABrv*) and pectineus (*P*) muscles and retracted tendon stump of the avulsed adductor longus from the pubic tubercle (*arrows*). The same patient in transverse short tau inversion recovery (STIR) MR image and coronal T2-weighted image (**c**, **d**). Images show stump of the tendon surrounded by hematoma (*arrows*). On the left side, the normal tendon is clearly defined

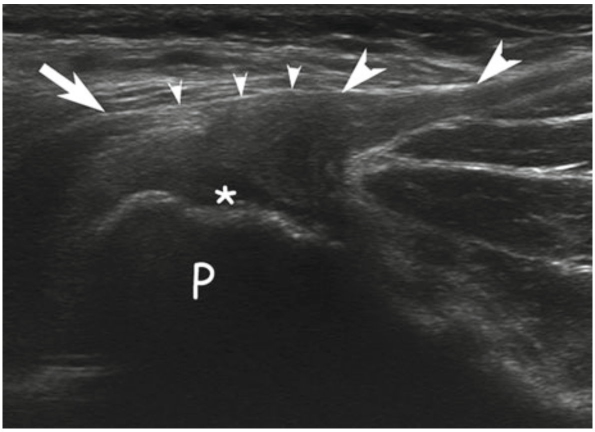

Fig. 4.6 Acute or chronic injury. Sonographic scan along adductor longus tendon (*arrowheads*) and their connection with the ipsilateral rectus abdominis (*arrow*) show tendinosis of their aponeurosis and origin of the adductor longus (*small arrowheads*) with osteolytic changes on the surface of the pubic tubercle (*P*). The cleft filled with fluid (*asterisk*) is a sign of partial avulsion of this complex tendinous structure

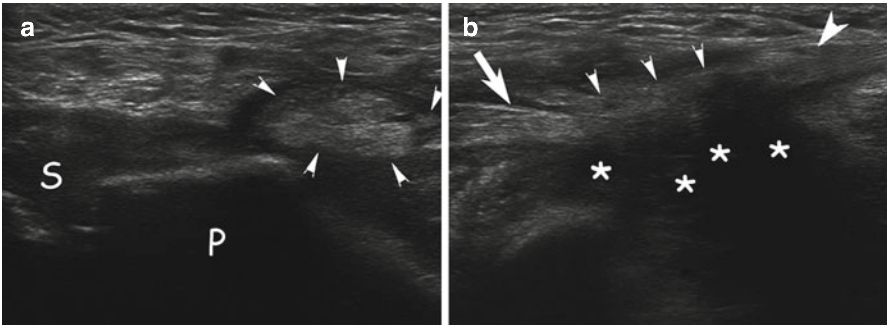

Fig. 4.7 Casual acute complete avulsion of the whole aponeurosis with preserved continuity of both the adductor longus and rectus abdominis tendons. On the transverse sonographic scan (**a**) on the level of pubic symphysis (*S*), the cross-section of the avulsed and laterally (from the "bare" pubic tubercle (*P*)) displaced aponeurosis is visible (*arrowheads*) (**b**) shows a sonographic scan along the displaced complex of the adductor longus tendon (*arrowheads*), aponeurosis (*small arrowheads*), and ipsilateral rectus abdominis (*arrow*) surrounded by hematoma (*asterisks*)

4.5 Treatment

Tomasz Piontek

Prevention training including stretching of the adductors should be included in every training session. A history of acute groin injury and weak adductor muscles are significant risk factors for new groin injuries [5]. Immediate rest after injury is advised until a diagnosis is determined. The primary goal of the treatment program is to minimize the effects of immobilization, regain a full range of motion, and restore muscle strength, endurance, and coordination [1, 15].

Surgical treatment is rarely indicated in acute groin injuries. Surgical treatment should be considered if a muscle or tendon unit is completely torn or if a partial rupture results in intramuscular hematoma that affects function or interferes with the healing process. Only a few cases of proximal adductor longus rupture have been reported in the literature. Management of these injuries has included nonoperative treatment, surgical excision of the entire muscle, and repair with suture anchors [15]. The most common indication for surgical treatment is chronic injury.

4.5.1 Surgical Technique

There are some important points that should be made concerning the operative technique for adductor tenotomy in chronic cases. The procedure involves a partial tenotomy, which is only performed on the superficial fibers of the adductor longus tendon [2, 5]. The adductor longus tendon is much longer on the superficial side than on the deep side. The tenotomy is performed at a level below the inguinal skin fold, at a distance of 2–4 cm from the tendon insertion (Fig. 4.8) [13].

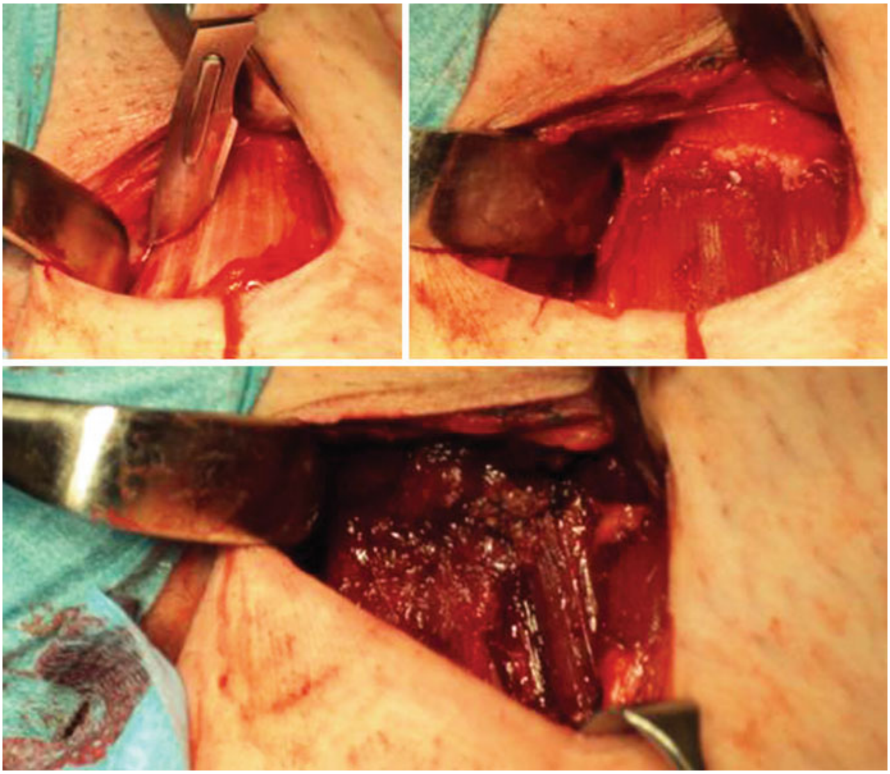

Fig. 4.8 Partial tenotomy, which is only performed on the superficial fibers of the adductor longus tendon

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Quadriceps Muscles

5

Casey M. Pierce and Robert F. LaPrade

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Abstract

Quadriceps muscle injuries are among the most common muscle injuries. The typical injury mechanism is a direct blow to the thigh. Other injuries include strains, tendon tears, and fascial ruptures. While most injuries can be diagnosed on physical exam, imaging studies may be beneficial to verify a diagnosis or to evaluate for structure damage or myositis ossificans. Immediate treatment recommendations generally include the principles of decreasing bleeding, maintaining motion, and avoiding hematoma formation. Proper diagnosis and classification of the injury pattern is essential to assist with selection of nonoperative or operative treatment regimens.

5.1 Definition of the Injury

Acute quadriceps injuries are those affecting the quadriceps femoris musculature and its tendons. The quadriceps femoris is comprised of four distinct muscles: the rectus femoris, vastus medialis, vastus lateralis, and vastus intermedius, which act as the major extensor apparatus for the knee. The rectus femoris also imparts hip flexion activity to the quadriceps due to its origin on the pelvis. Some authors argue for the definition of a fifth quadriceps muscle, the vastus medialis obliquus, which is thought to have a horizontal orientation to its muscle fibers and help to medially stabilize the patella, but it is typically described as simply part of the vastus medialis [5, 23, 52].

5.1.1 Anatomy

The quadriceps muscles are found in the anterior compartment of the thigh along with the femoral artery and nerve. All four muscles of the quadriceps converge to form a common tendon insertion which attaches on the patella. The quadriceps tendon itself has three distinct layers. The most superficial layer is derived from the rectus femoris, the middle layer from the vastus lateralis and medialis, and the deepest layer from the vastus intermedius. The rectus femoris originates on the ilium and is the most superficial of the four muscles. Its anterior position, superficial to the other three quadriceps muscles, makes it more susceptible to blunt force trauma, and, henceforth, it is the most often injured. The three deep components, from lateral to medial, are the vastus lateralis, the vastus intermedius, and the vastus medialis, all of which have their origins on the femur [53].

5.1.2 Function

The quadriceps muscles, patella, and patella tendon make up the extensor mechanism of the knee. Tension produced in the quadriceps is transmitted down through the patella, which acts as a pivot point to lever the force, through the patella tendon and to the tibial tubercle. The quadriceps muscles work together to control knee stability and minimize impact forces during loading of the knee joint and are critical in active movements of the lower limb including walking, running, jumping, and cutting [32, 52]. This is in addition to the patella stabilizing activity of the vastus medialis during normal gait and the hip flexion activity of the rectus femoris which helps swing the leg forward when taking a step [18].

Forces in the quadriceps tendon vary depending on the degree of knee flexion. Patella and quadriceps tendon forces are equal at 50° of flexion, while at 30° of flexion, the force experienced by the quadriceps tendon is 30 % less than that in the patellar tendon. This change in forces shifts the patellofemoral contact area to a more distal point on the patella which gives the quadriceps tendon a mechanical advantage during late knee extension. Conversely, when the knee is flexed to 90° and beyond, the force in the quadriceps tendon is 30 % greater than the patella, and the patellofemoral contact area is more proximal on the patella giving the patellar tendon a mechanical advantage during early knee extension [20, 21]. The quadriceps muscles can contract both concentrically and eccentrically, although higher forces are generated during eccentric contractions when the majority of quadriceps injuries occur [46]. Pain models, reported by Torry et al. and Henricksen et al., reported reduced quadriceps activation and decreased early stance knee flexion, which demonstrated the effects of quadriceps dysfunction on knee biomechanics [19, 50, 52].

5.1.3 Injury Mechanism

There are several common injuries which affect the quadriceps musculature. The most common is a quadriceps contusion, usually the result of a direct blow to the anterior thigh, commonly seen in contact sports such as football, basketball, ice hockey, and soccer [25]. Colloquially referred to as “Charlie Horses” in the United States, quadriceps contusions result from forces sufficient to cause tissue damage and disrupt the vasculature leading to two types of injury. The first injury is an intermuscular hematoma that is contained within the fascia and is more likely to spread out causing an ecchymosis. The second is an intramuscular hematoma that takes longer to heal and can lead to myositis ossificans and other complications [23, 25].

The quadriceps are the most common site for muscle contusion in the body, likely due to their anterior position in the lower limb and the fact that the quadriceps muscles are adjacent to the femur, making them highly susceptible to compressive forces and injuries from direct trauma. Interestingly, the quadriceps muscles are most resistant to impact injuries when they are struck while maximally contracted. Contraction decreases the compression of the muscle against the bone and protects the muscle fibers from more severe compression. Muscle contusions usually occur deep in the muscle belly and tend to cause less disability than strains, which are usually confined to the superficial muscle layers [12, 16, 23, 52, 53].

Quadriceps strains occur most commonly at the musculotendinous junction, the weakest point of the musculotendinous unit, and are due to indirect trauma from an

Fig. 5.1 Sagittal view T2 MRI of a knee demonstrating increased signal at the distal insertion of the quadriceps tendon (*yellow arrow*) secondary to quadriceps tendinopathy

excessive stretch [6, 16, 22, 54]. A quadriceps strain, commonly referred to as a "pulled muscle," typically takes place following a single major active extension activity such as sprinting or jumping where the patient did not properly warm up to prevent injury due to tensile overload [22]. Several components of the quadriceps muscles place them at a higher risk for strains and tears: higher constitution of fast-twitch (type II) muscle fibers, extension of the musculotendinous unit across two joints, eccentric action (lengthening during contraction), and shape (fusiform has the highest risk) [5, 16, 46, 54]. Because the rectus femoris is the only one of the quadriceps muscles that crosses two joints, it is the most commonly strained of the four quadriceps muscles [5].

Other injuries to the quadriceps include tendinopathy, tendon tears, and fascial ruptures leading to muscle hernias. Quadriceps tendinopathy (Fig. 5.1) is significantly less common than patellar tendinopathy due to the superior vascularity, strength, and mechanical advantage of the quadriceps tendon compared to the patellar tendon [52]. Quadriceps tendinopathy tends to be the result of an overload/overuse injury in runners and endurance athletes. It most commonly occurs at the distal attachment on the patella [33]. Tendon ruptures (Fig. 5.2) are seen mostly in the middle-aged and elderly population and are often associated with systemic illnesses such as diabetes, gout, rheumatoid arthritis, and kidney disease [23]. Quadriceps tears typically occur when a patient is attempting to regain their balance to avoid a fall. Rapid eccentric contraction against the falling body weight while the knee is partially flexed imparts maximum tensile stress on the quadriceps tendon. In younger patients, tears are usually the result of forced contraction or blunt trauma [22, 23].

Quadriceps muscle hernias have been found to occur most often in soccer, basketball, and rugby and result from a portion of the muscle protruding through a focal fascial defect. Fascial defects commonly occur following muscle hypertrophy and increased compartmental pressure at weak areas within the fascia, such as portions where blood vessels penetrate the fascia [34, 47].

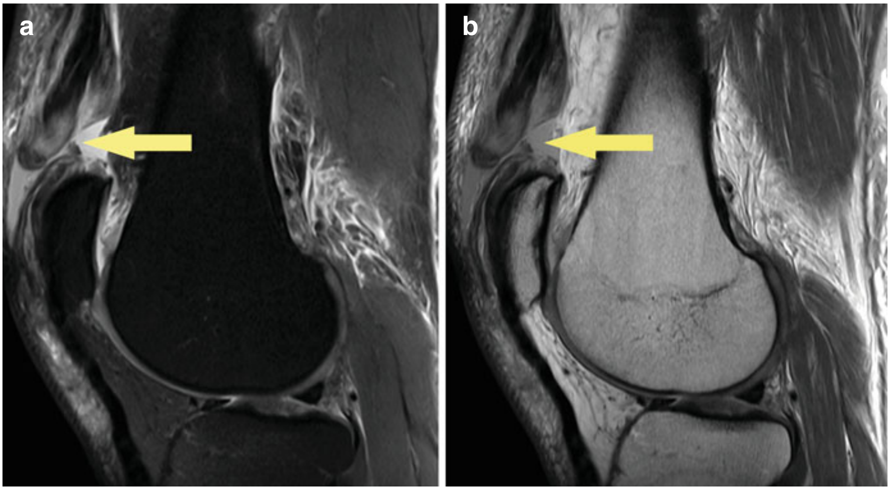

Fig. 5.2 Sagittal view T2 (**a**) and T1 (**b**) MRIs of a knee demonstrating complete disruption of the quadriceps tendon above the patella (*yellow arrows*)

5.2 Symptoms and Signs

5.2.1 Quadriceps Contusions

The typical history includes a direct trauma from an object or person to the anterior, medial, or lateral thigh, for example, a hockey player who blocks a hard shot of the puck with his thigh. The impact from the puck causes bleeding into the quadriceps muscles and a contusion [3, 25]. A study from the West Point Military Academy reported that the most common activities leading to quadriceps contusions were sports including football, hockey, wrestling, and baseball; however, quadriceps contusions can also result from simple accidents, falls, car crashes, and crush injuries [44].

Patients who have suffered a quadriceps contusion will typically complain of swelling and tightness in the thigh, difficulty walking, local tenderness to palpation, tenderness with straightening, decreased range of motion, and occasionally a palpable mass in the muscle body depending on the severity of their injury. Rarely, patients can suffer femoral nerve damage from the impact, due to either the direct impact or resultant swelling in the anterior compartment after the injury [3]. Although the mechanism of injury is a forceful impact, quadriceps contusions are rarely accompanied by fractures of the underlying femur. Hemorrhaging is common due to local capillary disruption and tissue edema, and a major trauma causing a large contusion can lead to compartment syndrome [25]. This is a rare occurrence, typically only seen with large contusions resulting from a major trauma, but due to its severe consequences (necrosis, fibrosis, and contractures), practitioners should remain mindful of it. Symptoms of compartment syndrome include pain

Table 5.1 Quadriceps contusion clinical classification system and typical symptoms at presentation

Grade	Active knee flexion	Gait pattern	Typical presentation
Mild (grade I)	More than 90°	Normal	Difficulty walking Mild/absent swelling Minimal pain with resisted knee straightening Full prone ROM ± Effusion ± Increased thigh circumference
Moderate (grade II)	Between 45° and 90°	Antalgic (slight limp)	Difficulty walking Occasional pain with activity Mild/moderate swelling Pain with palpation Pain with resisted knee straightening Limited ROM ± Effusion ± Increased thigh circumference
Severe (grade III)	Less than 45°	Severe limp	Assisted ambulation Severe pain Immediate swelling Pain with static contraction ± Bulge in the muscle ± Effusion ± Increased thigh circumference

Adapted from Jackson and Feagin [24]

out of proportion to the injury, paralysis, paresthesia, pallor, and a palpable pulse (the "5 Ps"). Since the anterior compartment houses the femoral nerve, compartment syndrome can often be ruled out with normal testing of the femoral cutaneous nerves.

Quadriceps contusions can be assessed using a variety of tests, including direct palpation described by Brukner and Khan, a firmness rating and thigh circumference described by Bull, passive knee flexion ROM described by Jackson and Feagin, and the brush-swipe and tap tests described by Ryan to determine the presence of knee effusion. Of these, only the reliability of the thigh circumference measurements has been verified [24, 56].

Inspection of the affected area may demonstrate ecchymosis, deformity, or swelling depending on the extent of the injury. Strength testing may show deficits in resisted knee extension and hip flexion compared to the uninjured side, and patients may also have limited knee flexion. Straight leg raises should be normal, unless there is concurrent damage to the extensor mechanism. The patient's gait might also be altered in more severe contusions [50]. Knee effusion can be present in any grade, as can increased thigh circumference on the affected side [27, 56].

Based on the clinical exam findings, Jackson and Feagin described an initial classification system for quadriceps contusions in 1973, which was later modified by Ryan et al. in 1991 (Table 5.1). This classification system has been shown to be

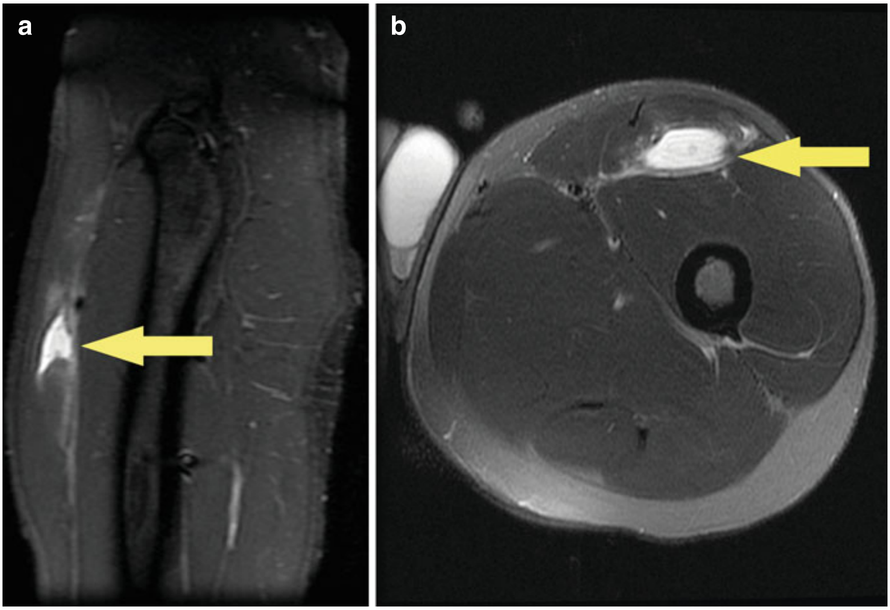

Fig. 5.3 Coronal (**a**) and axial view (**b**) MRIs of an anterior thigh demonstrating a partial tear of the rectus femoris muscle (*yellow arrows*)

useful in predicting disability times associated with each grade of contusion. Mild contusions have an average disability time of 13 days, 19 days for moderate contusions, and 21 days for severe contusions [24].

5.2.2 Quadriceps Strains

Quadriceps strains are a common injury encountered in sporting events which require rapid cutting movements, kicking, or jumping. The typical history of a quadriceps strain will reveal a patient who attempted to jump, kick, or suddenly changed directions while running. An excessive tensile force rapidly acting on the muscle strains the fibers past their ability and causes a tear near the musculotendinous junction; however, recent imaging-based studies have demonstrated tears proximal in the rectus femoris (Fig. 5.3), which is the muscle most often injured [12]. Fatigue has been reported to be a major factor in this type of injury, so injuries often occur toward the end of sporting events [16, 48].

The affected patient will often be immediately aware that they have suffered some sort of injury and will report a sharp, tearing pain in their anterior thigh. The pain may have a delayed onset, but the patient will typically experience rapid loss of function. Pain can be felt anywhere down the quadriceps muscle but is most commonly located toward the musculotendinous junction of the rectus femoris. An obvious deformity may be seen following the injury which can be palpated along the anterior thigh (Fig. 5.4). Strength testing of hip flexion and resisted knee flexion

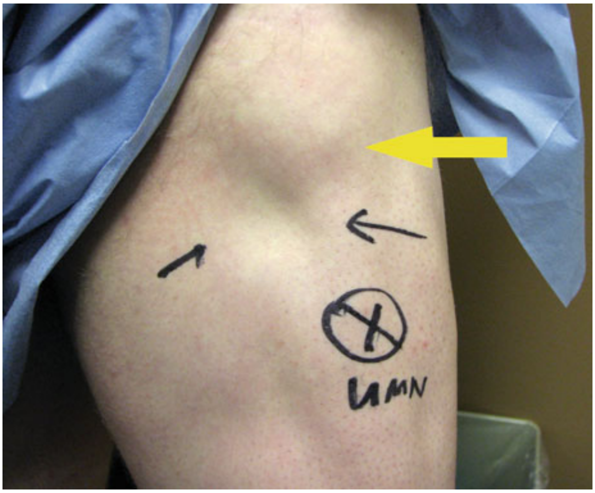

Fig. 5.4 Photograph of an obvious deformity in the anterior left thigh as the result of a mid-substance rectus femoris muscle tear (*yellow arrow*)

Table 5.2 Quadriceps strain clinical classification system

Degree	Pain	Strength loss	Palpable muscle defect
I	Mild	None or minimal	No palpable defect
II	Moderate	Moderate	± Small palpable defect
III	Severe	Complete loss of strength	Palpable defect

Adapted from Kary [27]

may elicit pain and demonstrate loss of strength/function. Passive stretching and direct palpation will also cause pain. The clinical grading system for quadriceps strains is based on pain, strength, and palpable muscle defects detected during physical examination (Table 5.2) [27].

The incidence of myositis ossificans has been reported to be between 9 and 17 %. Myositis ossificans should be suspected if symptoms worsen after 2–3 weeks accompanied by loss of knee flexion and persistent swelling [4].

5.2.3 Quadriceps Tendon Tears

Tears of the quadriceps tendon are a rare occurrence, but they are more common than tears of the patellar tendon. Patients who have suffered a complete or partial tear of the quadriceps tendon are typically older (>40 years old) and often have conditions that can lead to degeneration in the tendon (gout, obesity, rheumatoid arthritis, diabetes, infections, etc.) [48]. Other patients who are at risk for quadriceps tendon tears are those that use performance-enhancing substances such as anabolic steroids and creatine. These drugs lead to increased muscle strength, and steroids have been reported to weaken tendons, change collagen fibril structure, and decrease tendon elasticity in animal studies. The combination of amplified muscle strength and a potentially weakened tendon increases the likelihood of suffering a tendon rupture. Quadriceps tendon tears can also be the result of repeated micro-traumas and strong, sudden decelerations [41].

The common mechanism of injury in quadriceps tendon tears is a sudden eccentric contraction of the quadriceps muscle with the foot planted and the knee partially flexed (typically more than 60°). This is typically the result of a fall or missed step but can also be due to a direct blow, laceration, or iatrogenic causes (following total knee replacement, anterior cruciate ligament reconstruction, meniscectomy, or steroid injections).

If seen in the acute phase, the patient will usually present with a painful, swollen knee with loss of function following a fall or buckling of the knee. Younger patients often have a history of activity-dependent pain above the patella. A careful history is critical in these patients to understand the underlying mechanism or disease which contributed to the tendon tear. Ambulation will be difficult, if not impossible, for the patient, and they will likely have tenderness and swelling above the patella with ecchymosis. With complete tears, the patella can be low-lying, and there will be a palpable defect or gap above the patella [23].

Extensor strength testing in full active extension against gravity in supine and flexed positions will reveal the degree of tear and retinaculum damage based on the amount of extension lag. Patients with complete tears will be unable to complete a straight leg raise or maintain a passively extended knee position. Patients with a partial tear can have some degree of extension intact but will be unable to maintain extension against force and will demonstrate an extensor lag. In addition, partial ruptures may demonstrate full extension in supine position, while being limited from the flexed position. Flexion should be intact in both complete and partial tears and should be compared to the contralateral normal side to rule out bilateral tears [23].

Patients with a delayed presentation or chronic tear of the quadriceps tendon may have the gap in their tendon filled in with fibrotic tissues or a hematoma. These patients will still have weakness in extension and an antalgic gait [23, 28, 44].

5.2.4 Quadriceps Tendinopathy

Quadriceps tendinopathy has been classified as a variant of “jumper’s knee” because it has a propensity for affecting athletes involved in jumping sports such as volleyball and basketball. Those affected patients complain of anterior knee pain at the superior pole of the patella. Compared to the sharp or deep pain associated with other common quadriceps ailments, the pain of quadriceps tendinopathy is dull and often described as “achy.” The onset of pain is slow and activity dependent [9, 11, 13, 33].

Physical examination will reveal tenderness to palpation at the suprapatellar pole. The stability of the patella and knee should be normal, but patients may demonstrate hamstring and/or quadriceps tightness. Patients will usually have a full range of motion of their knee, although there may be joint effusion [9, 11, 13].

Table 5.3 Jumper's knee classification system

Stage	Symptoms
1	Pain occurs only after activity and does not cause functional impairment
2	Pain occurs during and after activity but does not cause functional impairment
3	Pain is prolonged during and after activity and causes increasing functional impairment
4	Loss of function due to completely torn tendon that will require surgical repair

Adapted from Blazina et al. [9]

Jumper's knee is classified into four different stages based on the timing and duration of symptoms. The classification of jumper's knee is seen in Table 5.3 [9].

5.2.5 Quadriceps Muscle Hernia

Quadriceps muscle hernias usually occur at the anterior mid-thigh region where a previous injured area of fascia has become weakened. Fascial weakening can be the result of muscle overuse or hypertrophy and commonly occurs at the structurally weak points where vessels and nerves penetrate the fascia. The typical history for quadriceps hernias is a sudden forceful kick followed by a bulge at the site of the herniation. Muscle hernias are common in professions where a high demand is placed on the legs such as athletes and members of the military. A soft mobile mass can be palpated at the site, and it becomes more prominent and solid when the quadriceps muscles are contracted [47]. When the quadriceps are relaxed, sometimes a fascial defect can be palpated. Most muscle hernias do not cause symptoms, but with prolonged activity some cramping and pain can develop. The defect can lead to nerve entrapment or incarceration of the muscle tissue, which can cause substantial pain and discomfort [7, 47].

5.3 Imaging

5.3.1 Quadriceps Contusions

Imaging of quadriceps contusions is typically limited to ruling out other complications of the injury. Radiographs of the femur can be obtained to rule out a fracture and the development of myositis ossificans. Magnetic resonance imaging (MRI) can be used to obtain information about the early phases of healing in contusions; however, MRI is typically only used to evaluate patients who are not responding to therapy and those whose diagnosis is in doubt. MRI is also widely used to assess the degree of injury in professional athletes [27, 30].

The appearance of a quadriceps contusion on MRI varies depending on the extent and severity of the lesion. A "feathery," infiltrative signal intensity on STIR (short-tau inversion recovery) and T2-weighted images is indicative of focal edematous changes in the muscle and can have a similar appearance to grade I strains [12].

Contusion-induced hematomas are typically found deeper in the muscle belly as compared to those caused by muscle strains, and while increased muscle girth may be noted, discontinuity of the fibers or major disruption of the muscle architecture should not be present. As the lesion heals, it will demonstrate lower-signal intensity as hemosiderin deposits within the healing hematoma. Compared to ultrasound of a contusion, which is initially echobright before becoming echopoor as it liquefies and then echobright again as the clot organizes, MRI allows for the identification of muscle atrophy, fibrotic muscle tissue, and any heterotopic bone formation as the lesion heals [1, 12, 16, 30].

Myositis ossificans is a relatively uncommon complication encountered following a single or repeated muscle contusion. This condition most often affects young male athletes who engage in contact sports such as football, basketball, ice hockey, and wrestling. Following some contusions, heterotopic bone formation occurs in the soft tissues of the affected muscle or tendon. Bone formation can also been seen in the fascia and can be attached to the periosteum, causing it to be confused with a parosteal osteosarcoma in some cases [36]. To differentiate between the two conditions, the patient history is important because sarcomas will typically not have a significant history of trauma to the affected limb. If the diagnosis remains in doubt, an excisional biopsy may be warranted to confirm the pathology and direct the proper treatment course [4, 27, 31, 36].

Thus far, the cause of myositis ossificans is not yet understood, but animal studies have suggested the cause to be mesenchymal connective tissue cells undergoing trauma and/or osteogenic protein-induced metaplasia to osteoblasts and fibroblasts. The osteoblasts and fibroblasts deposit cancellous, osteoid material which eventually matures into lamellar bone around the edges of the lesion. The lesion formed is typically solitary and well defined and will often regress without treatment [4].

The risk of developing myositis ossificans was defined by Jackson and Feagin in 1973 and then again by Rothwell in 1982 [43]. With mild (grade I) contusions, the risk of developing myositis ossificans was reported to be between 0 and 9 %, with a moderate (grade II) or severe (grade III) contusion the risk jumped to 17–72 %, and with recurrent quadriceps contusions the risk was between 54 and 100 % [24, 27].

Ultrasound and MRI are both highly sensitive for diagnosing myositis ossificans. Ultrasounds can differentiate myositis ossificans from bony tumors and will reveal an oval hypoechoic mass without infiltrative borders. Early signs of myositis ossificans on ultrasound are a center of less echogenicity accompanied by an outer hyperechoic peripheral rim. Calcifications may be seen in mature myositis ossificans lesions leading to distal acoustic shadowing [1, 30, 31].

The MR appearance of early (immature) myositis ossificans lesions typically shows a heterogeneous, high T2 signal and an enlargement of the tissues where the mass is growing. There may be a rim of low-signal intensity surrounding the mass. The addition of intravenous gadolinium will result in an intense heterogeneous enhancement of the lesion(s). Older myositis ossificans lesions (weeks or months following the initial injury), which have had the chance to begin to mature, will show a different appearance on MR imaging. A low-signal rim can be appreciated which corresponds to mature cortical bone. The same heterogeneous, high T2 signal lesion will still be appreciated, along with persistent edema surrounding the lesion. Further imaging as the lesion matures will show decreased edema in and around the lesion and can show signals mimicking fat tissue located centrally within the lesion which likely represents marrow fat [31].

5.3.2 Quadriceps Strains

Quadriceps strains are classified via MRI based on the amount of disruption to the muscle fibers and muscle architecture. First-degree strains can show the same “feathery” appearance on MRI as contusions, indicating edema and hemorrhage in the fibers and musculotendinous junction; however, strains will show at least a minimal amount of fiber disruption. Second-degree strains will show a partial tear that has not retracted back into the muscle. Fluid collections around the fascia and hematomas are often observed in conjunction with the tear. Complete rupture of the muscle fibers is seen in third-degree strains, and the muscle will typically retract from the site of the tear [5, 12]. MRI is usually only utilized for preoperative planning to assess the degree of muscle retraction [16, 30].

5.3.3 Quadriceps Tendon Tears

Complete tears of the quadriceps tendon are typically diagnosed clinically and confirmed by ultrasound or less expensive modalities than MRI. However, magnetic resonance imaging is the best choice for an imaging study if there is any remaining doubt regarding the diagnosis. T2-weighted MR images will clearly demonstrate clear, full, and continuous disruption of all three layers of the tendon if a complete rupture has occurred. Characteristically, with complete tears, the proximal remnant will retract, as well as giving the patellar tendon a “wrinkled” appearance [8, 30]. Heterogeneous signal intensity on T1-weighted images often indicates the presence of an accompanying hematoma. When looking at the entire extensor mechanism on MRI, an undulating outline of the patellar tendon should not be considered diagnostic for a complete tear because it can also be the result of knee hyperextension and is therefore not diagnostic. Partial tears will have single layers that are discontinuous, but the remaining layers will be intact which will rule out a complete tear. Partial tears are often confined to the anterior rectus femoris tendon fibers at the insertion on the patella [5, 30]. This makes MRI quite useful for differentiating complete from partial quadriceps tendon tears.

5.3.4 Quadriceps Tendinopathy

Although it occurs less often, quadriceps tendinopathy has a similar appearance to that seen in patellar tendinopathy on MRI. Quadriceps tendinopathy will reveal fusiform thickening of the tendon and altered signal intensity, most commonly at the insertion of the distal quadriceps tendon to the patella. The normal lamellated architecture of the tendon will be lost, and indistinct changes can be appreciated within the fatty tissue planes. If extension strength is normal, MRI may demonstrate signs of degeneration at the posterior insertion of the quadriceps tendon [9, 11, 13, 33].

MRI is quite sensitive for detecting tendinopathies in both symptomatic and asymptomatic athletes, and symptomatic tendons can have the appearance of being normal. To prevent false-positives, it is very important that physicians order MRIs for quadriceps tendinopathy in the face of positive clinical findings and not as a routine screening test. Older patients suffering from quadriceps tendinopathy may exhibit degenerative changes, including tendon calcification or bony spurs on the superior pole of the patella [9, 11, 13, 18].

When evaluating an MRI for signs of tendinopathy, one should be aware of a phenomenon known as the “magic angle,” which affects tissues composed of well-ordered collagen fibers, seen in hyaline cartilage, menisci, and tendons. When exposed to a magnetic field, these tissues can appear hyperintense when the fibers are oriented at a so-called magic angle of approximately 55° to the static field of the magnet. The hyperintense artifact this creates can be easily mistaken for tendinopathy or other forms of pathology. The magic angle phenomenon can be avoided with an echo time of 37 ms or more on T2-weighted sequences [39].

Ultrasound examination is quite sensitive to diagnose and demonstrate the extent of tendinopathy. Signs of chronic tendinopathy include a quadriceps tendon thickness greater than 6.1 mm, erosion of the superior pole of patella, patellar osteophytes, and tendinous calcification. Ultrasound imaging of quadriceps tendinopathy can also reveal hypoechoic areas masking the normal fibrillar appearance of the tendon [11, 13].

5.3.5 Quadriceps Muscle Hernia

Ultrasound and MRI are both valuable tools to detect or confirm muscle hernias. MRI can often show the fascial defect through which the muscle is protruding. While most hernias can be diagnosed based on clinical signs, MRI is very useful in confirming the diagnosis, preoperative planning, and postoperative monitoring if necessary [34].

5.4 Treatment

5.4.1 Nonoperative

5.4.1.1 Quadriceps Contusions

If possible, a patient who has suffered a quadriceps contusion should immediately have the knee put in 120° of flexion for approximately 10 min. This has been reported to compress the injury to limit hemorrhage and muscle spasm. Research has shown that patients who are put in 120° of flexion immediately following a quadriceps contusion return to normal range of motion more quickly than those who do not and have a lower chance of developing myositis ossificans [2–4, 27]. If the leg is left extended, the quadriceps can start to heal in a shortened position which makes for a more difficult recovery [3].

The use of a hinged brace or a compression wrap is helpful to maintain the leg in 120° of flexion, and ice and compression should be actively employed during the first 24 h. Twenty-four hours following the initial injury, patients should discontinue immobilization at 120° of flexion and begin electrical stimulation and/or passive stretching, still accompanied by icing and compression [2]. Active pain-free quadriceps stretching and strengthening should be performed several times a day. Patients should apply ice for approximately 20 min every 2–3 h to reduce swelling and inflammation. Crutches may be needed for more severe contusions. Typically, patients will be able to return to sports following a mild contusion within a week or two. More severe contusions can cause disability lasting up to 3–4 months but typically resolve within 3 weeks [24, 27]. Development of myositis ossificans or compartment syndrome will significantly increase the length of disability, especially if surgery is indicated.

Contusions that do not receive immediate medical attention and already have limited knee flexion due to intramuscular bleeding should be managed differently. The patient should be kept in the prone position, and the knee should be positioned in the maximum amount of flexion without experiencing pain. In the flexed position, the patient should isometrically contract the quadriceps muscles until the patient feels fatigued. This fatiguing contraction should relax the spastic muscle and allow the practitioner to passively stretch the muscle further. This entire process is repeated approximately three more times, and then the patient is immobilized in their maximum amount of pain-free flexion and placed on crutches. This process should be repeated twice daily until 120° of flexion is achieved, at which time the crutches may be discontinued once the patient can ambulate without a limp [2].

The use of nonsteroidal anti-inflammatory drugs (NSAIDs) is not recommended for mild contusions, but their use is thought to prevent the development of myositis ossificans following severe or repeated contusions based on studies in hip replacement patients [14, 35]. Myositis ossificans should be suspected if symptoms worsen 2–3 weeks following the initial injury or in patients with persistent knee swelling [4].

To return to sports, players should have no pain and be able to flex the knee to 120° with the hip extended, in addition to being able to complete functional field testing without limitations. Players with myositis ossificans can still compete in sports, but they may experience a limited range of knee motion and some occasional pain and/or swelling.

Surgical excision of heterotopic bone should not be performed until the bone has matured (typically 12–24 months). Surgical excision of immature bone should not be attempted because it has been reported to lead to a more severe local recurrence of the bone formation.

5.4.1.2 Quadriceps Strains

Treatment goals for muscle strains are aimed at minimizing the bleeding and hematoma formation following injury to the muscle. There is a scarcity of literature on the specific treatment of muscle injuries, including strains. Because of this, the treatment protocols have not changed drastically in recent years. Acute treatment for strains complies with the PRICE (Protection, Rest, Ice, Compression, and Elevation) protocol for the first 24–72 h following an injury. Ice and compression should be used for approximately 10–20 min at a time in hour-long intervals [10]. Grade 2 and 3 strains may require crutches to allow proper rest and immobilization of the quadriceps. Protection and rest are aimed to prevent further damage to the muscle, while ice decreases blood flow, bleeding, and inflammation at the damaged area. The use of ice following acute muscle injuries has been shown to be effective in decreasing pain caused by the injury, but as of yet there is no definitive proof that it leads to faster healing and a quicker return to sports [10, 17, 25, 37]. Compression and elevation both aid in decreasing blood flow and swelling in the injured area.

The use of NSAIDs can help to reduce pain and facilitate earlier to sports, but the long-term effects on strains is unknown, so a short course (between 3 and 7 days) is only recommended by some centers. Some centers believe that NSAIDs should be contraindicated due to the increased risk of local bleeding and the potential for slower healing of the injury. Therefore, their use is controversial [35].

Following acute treatment with the PRICE protocol, active management of a quadriceps strain should begin 3–5 days following the injury. Active management consists of stretching, strengthening, range-of-motion exercises, cardiovascular fitness, proprioceptive exercises, and functional training to restore function of the injured muscle [12].

Cross et al. reported a 4-stage active management phase to return athletes to sports that involved sport-specific exercises, combined with intense physiotherapy focused on soft tissue therapy with stretching and strengthening exercises. They allowed athletes to perform staged running drills once they had achieved full pain-free passive knee range of motion and could complete single leg hops with three sets of ten repetitions while pain free. Athletes were not allowed to run on consecutive days because this was considered to be potentially harmful. When players were able to successfully complete all four of the stages, they were slowly integrated back into team practices [12].

Stretching should be performed to the point where it becomes uncomfortable but should not cause pain. Passive, active-passive, dynamic, and proprioceptive neuromuscular facilitation stretching are all acceptable forms of stretching, but ballistic stretches should not be used because they risk re-injuring the healing muscle fibers.

Strengthening exercises should include isometric, isotonic, isokinetic, and functional exercises and should be conducted through a pain-free range of motion. There is not an established set of guidelines to safely return to sports after a quadriceps strain; however, athletes should have restored knee range of motion and normal or close to normal strength compared to the uninjured side while being pain free. Performance on functional tests is also helpful to gauge readiness to return to sports, and isokinetic muscle strength testing may be useful for assessing strength and preventing re-injury [37].

The time required to return to play following a quadriceps strain is not only dependent on the severity or grade but also the location of the strain. Cross et al. reported that strains affecting the rectus femoris took significantly longer to heal (9–27 days) compared to those occurring in the vastus muscles (4–5 days), especially those that affect the central tendon, known as the "bull's eye lesion" [12, 37].

5.4.1.3 Quadriceps Tendon Tears

Incomplete or partial tears of the quadriceps tendon can often be managed nonoperatively. The patient's knee should be immobilized in full extension for a period of up to approximately 6 weeks depending on the size of the tear. Use of the PRICE protocol has yielded positive results, and reports have indicated that hemarthrosis evacuation, which is common after a quadriceps tendon tear, can aid in reducing pain and speeding recovery [10]. It has been reported that even small knee effusions can affect quadriceps strength, which further supports the need for early and aggressive knee effusion treatment [26, 50]. The effect of early hematoma evacuation has not been studied to determine its possible benefits; however, evacuation is thought to be useful to reduce pain and may allow for earlier recovery. It should be noted that hematoma evacuation, if attempted, should be done early on, prior to consolidation, which would make evacuation more difficult [3]. Practitioners should attempt to avoid prescribing anti-inflammatory medications for patients with quadriceps tendon tears because they have been shown to impair tendon healing. Patients can be allowed to discontinue immobilization once their ability to perform a straight leg raise has been restored, and they can begin progressive knee range of motion and strengthening exercises.

Surgical intervention may be required if patients fail conservative management. The choice of surgical procedure is typically dependent on the degree of the tear and how many tendon layers have been ruptured. If one of the three tendon layers remains intact, repairs can proceed with removal of scar tissue and side to side-to-side closure of the tendon; however, if the majority of the tendon is torn off the bone, suture anchors or sutures placed through the patella may be necessary to reattach the tendon [28, 29, 40, 45].

When a complete quadriceps tendon rupture occurs, patients will require surgical repair or reconstruction to reestablish function of the extensor mechanism because conservative treatment most often results in poor outcomes. Early interventions (between 24 h and 1 month) for complete tears are desirable to prevent tendon retraction, patella baja, and muscle atrophy, which can lead to poor outcomes. Several different techniques have been described for surgical repair of quadriceps tendon ruptures. The most commonly used technique involves a transosseous suture repair through vertical patellar drill holes, although other surgeons recommend positioning the transosseous suture through the patella with a transverse drill hole. To anchor the quadriceps tendon, a locking or figure-of-eight suture technique is used, and repair of the medial and lateral retinaculum should be performed when necessary. Ruptures that occur more proximally with part of the tendon remaining intact can sometimes be repaired with end-to-end sutures. Ruptures occurring at the insertion on the patella will often have bony avulsion fragments still attached to the tendon which should be retained to allow for improved bone healing. Another repair technique uses a circumferential suture placed with the knee at 30° of flexion, which aims to protect the repair and add additional strength to allow for early postoperative mobilization. Alternative repair techniques involved the use of wire cerclages, screw augmentation, or suture anchors [28, 29, 41, 42, 45, 55].

Studies have reported that early postoperative mobilization results in improved tendon healing and earlier return of joint motion. As a result, passive flexion to between 30° and 90° is typically initiated on postoperative day one [55]. Most surgeons recommend partial or non-weight-bearing for the first 6 weeks following surgery [25, 28, 29].

Chronic quadriceps tendon ruptures are typically more difficult to treat due to scarring and retraction of the tendon which can prevent direct repairs. The tendon must be mobilized by elevating the quadriceps muscles off of the femur and adhesions must be released. Once the tendon is released, improved outcomes are reported if the tendon can be reapproximated to the patella. If reapproximation is not possible, the tendon will require reconstruction, typically with an autologous hamstring graft. Alternatively, a procedure to lengthen the quadriceps tendon, described by Scuderi, can be used when direct repair is not possible. This procedure entails cutting a full-thickness inverted "V" in the proximal quadriceps tendon stopping approximately 2 cm proximal to the rupture. A triangular flap can then be turned down and attached to the patella. The portion of the tendon where the flap was cut can then be repaired in a side-to-side manner [28, 29, 41, 42].

5.4.1.4 Quadriceps Tendinopathy

Conservative therapy is the treatment of choice for quadriceps tendinopathy and should include rest with activity modification and a physiotherapy program aimed at aggressive hamstring strengthening. Ice, massage, ultrasound, iontophoresis, and phonophoresis have also been used with positive results [25]. NSAIDs and local steroid injections around the tendon are relatively safe and can be a helpful addition to conservative treatment [35]. As a patient's pain decreases, the intensity of physical activity can be gradually increased [9, 11, 13].

Platelet-rich plasma (PRP) treatment is based on the premise that growth factors in concentrated plasma can aid in tendon healing. The use of serial injections of platelet-rich plasma into an injured tendon has been reported to improve outcomes, especially in refractory cases of quadriceps tendinopathy [15].

Sclerosing agents, if properly injected, can obliterate the hypervascularity associated with tendinopathy. Improved results have been reported when ultrasound is used to find a "feeder" vessel, which is obliterated by injection of a sclerosing agent leading to a decrease in the vasculature surrounding the affected tendon. Serial injections of sclerosing agents have been reported to yield significant reductions in pain and disability [11, 38].

Surgery to treat quadriceps tendinosis is not often needed and is typically only considered in patients who have failed various modalities of conservative therapy and steroid injections. Following MRI, the degenerative tissue and calcifications in the affected tendon can be surgically removed. If a large portion of the tendon is affected and requires excision, reattachment to the patella may be necessary [11, 13].

5.4.1.5 Quadriceps Muscle Hernia

Quadriceps muscle hernias typically do not meet the criteria for surgery and only present an aesthetic problem. For those that become strangulated and require repair, fasciotomy with a simple closure of the fascial defect has reported good results [47].

5.4.2 Operative Treatment and Findings at Surgery

5.4.2.1 Quadriceps Contusions

Surgical intervention for quadriceps injuries is typically only indicated for hematoma removal, compartment syndrome, complete muscle ruptures, quadriceps tendon tears, and, rarely, muscle hernias. Occasionally, with the development of myositis ossificans following a contusion, surgery will be necessary to restore knee movement or relieve pressure on a nerve or blood vessel [27].

Operative findings for muscle contusions treated with fasciotomy typically show increased compartmental pressures with a large hematoma confined within the anterior compartment of the thigh. Published reports have not reported any occurrences of necrotic muscle, fibrosis, or loss of function. Rarely, contusions can cause muscle tears which can be appreciated upon operation [27].

Myositis ossificans can be excised, but surgical excision of the ectopic bone is typically reserved for cases in which symptoms have progressed or remained debilitating over a period of 12 months despite physical therapy and pain management. Reports suggest that excision of the mass should be held until the bone has fully matured, typically within 1 year of symptom onset and with a negative uptake on a bone scan [4].

Operative findings for myositis ossificans vary depending on the location of the lesion. In the thigh, one will typically see an osseous mass extending from the muscle itself. One type commonly seen is the stalk type with a thin bony stalk connecting the femur to the overlying ossified bone. Another is the periosteal type, where a larger region of the ossified muscle contacts the underlying bone. The third common type shows no connection between the ossified muscles and bone beneath [4].

5.4.2.2 Quadriceps Tendon Tears

Quadriceps tendon tears can be complete or partial and tend to occur either midsubstance or near the insertion on the patella. Operative findings can be as simple as a torn tendon or can show signs of degenerative changes which are common in quadriceps tendon ruptures. Grossly degenerative or friable tissue should be removed to allow for the fresh end of the tendons to be repaired [22, 23]. Biopsies taken from 22 operative cases reported evidence of degenerative changes, including organized scar tissue, calcifications, neovascularization, chondrogenic metaplasia,

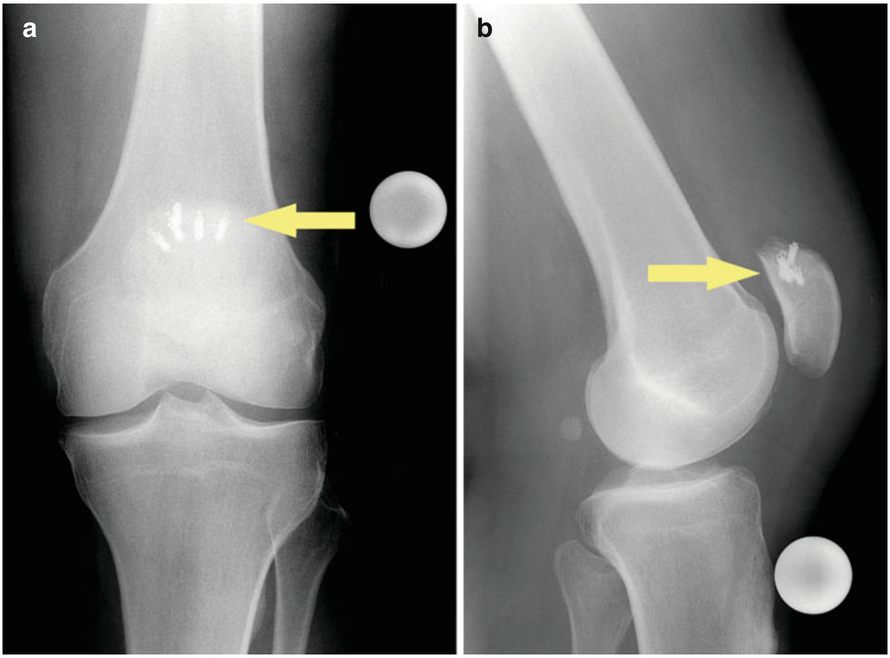

Fig. 5.5 Anteroposterior (**a**) and lateral (**b**) radiographs of a left knee showing a repaired quadriceps tendon tear with suture anchors (*yellow arrows*) in the proximal pole of the patella

and severe mucoid collagen structure changes in all cases [51]. Inflammatory changes, such as granulation tissue and damaged collagen, should be expected when repairing a torn tendon and are not indicative of degeneration but rather the inflammatory process occurring after the initial rupture. In older patients, signs of collagen degeneration are expected and are likely a cause of the increased incidence of tendon rupture in the older population. A lack of signs of tendon degeneration is common in younger patients with a tendon rupture. Hematomas are a common finding during operative repair of a torn quadriceps tendon, and Hardy et al. reported that nearly 80 % of patients with a complete quadriceps rupture also had a patellar osteophyte typically located on the anterior superior patellar pole [18, 44, 51].

Upon operative repair, a complete tear can show a “bald patella” where the tendon has torn completely off of the proximal patella. A firm and secure repair of the torn tendon to its bony attachment via suture anchors or sutures placed into the bone and tendons should be performed (Fig. 5.5). Ideally a firm and secure repair which allows for early knee range of motion is desired [28, 29].

5.4.2.3 Quadriceps Muscle Tears

Operative findings for quadriceps muscle tears typically show damage to the muscle body (Fig. 5.6). On histological examination, Temple et al. in 1998 reported finding patients with large areas of chronic inflammation mixed in with normal muscle,

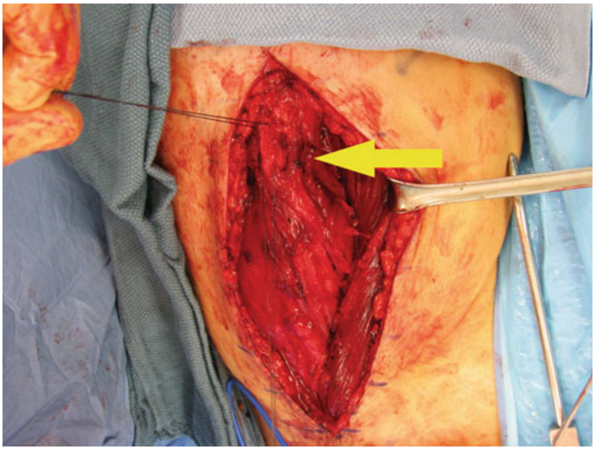

Fig. 5.6 Intraoperative photograph showing a chronic mid-substance tear of the left rectus femoris muscle (*yellow arrow*)

degenerating muscle, and reactive fibrous tissue. They also noted areas of small focal hemorrhage and neovascularity. Normal-appearing muscle was also seen, and they postulated that the time frame between repair and the initial injury could account for operative findings [49, 51].

5.4.2.4 Quadriceps Tendinopathy

Surgery should only be considered for patients suffering from quadriceps tendinopathy once conservative measures have failed or a tendon rupture has occurred. Typical findings upon surgical repair are macroscopically normal tendon tissue, but degeneration is seen occasionally, especially with a previous history of steroid injections. On pathological and histological examination, tendon samples from surgically repaired quadriceps tendons demonstrate abnormalities at the junction of the muscle and bone. These include pseudocystic cavities between the mineralized fibrocartilage and the bone. Cavities are filled with necrotic or areolar tissue with signs of neovascularization. Areas of pseudocysts will demonstrate a loss of the so-called blue line, the borderline which separates fibrocartilage from mineralized fibrocartilage. Increased thickness of fibrocartilage at the insertion of the tendon can also be seen due to increased production of myxomatous tissue or hyaline cartilage [50].

5.4.2.5 Quadriceps Muscle Hernia

The fascia overlying the quadriceps muscles is not easily torn. Operative repair of herniation through the fascia typically reveals a small defect in the fascia which has allowed a portion of the muscle to bulge out and cause symptoms or become strangulated. The accepted treatment for such defects is a fasciotomy with extension of the fascial defect to a length of 7–10 cm. This will usually alleviate symptoms. Fascial repair was an option previously thought to hold merit, but as it has been known to cause compartment syndrome, it is no longer performed. MRI can provide valuable preoperative information for the surgeon regarding the size and location of the fascial defect, as well as being useful if postoperative complications are a concern [34, 47].

Conclusion

Injuries involving the quadriceps musculature including muscle contusions, strains, tears, tendinopathy, and herniations are among the most common muscle injuries. A review of the mechanism of injury, combined with physical examination and imaging studies, when indicated, can help to define the type of injury to the quadriceps musculature. Proper diagnosis and classification of injury patterns and grades can assist with selecting nonoperative or operative treatment regimens in the majority of cases.

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The Calf Muscle Complex

6

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Abstract

Calf muscle complex, consisting of the plantaris, soleus, and gastrocnemius muscle, is commonly injured, especially in athletes. Patients typically complain of acute pain in the proximal calf, ecchymosis, and significant swelling.

Based on clinical presentation, injuries of the calf muscle complex are graded 1–3. These grades are used to estimate convalescence period and to elect a suitable rehabilitation program. MRI or ultrasound can be of value in both the initial assessment and follow-up of the injury. Treatment, principally conservatively, is initiated according to the RICE schedule. Rehabilitation focuses on gentle stretching, massage, strengthening exercises, and balance exercises along with core strengthening and general conditioning.

6.1 Introduction

The "tennis leg" was first described in 1883 by Powell [27] and was thought to be a rupture of the plantaris tendon. In reality, this injury of the calf muscle complex is a rupture of the medial head of the gastrocnemius muscle, owing his name to the fact that this injury was originally described in tennis players [5, 6, 10–12, 15, 19, 27].

Nowadays, injuries of the calf muscle complex are regarded as common injuries, especially in athletes. Strains to the calf muscle complex are preceded in frequency by injuries to the hamstrings, groin, and quadriceps muscle group [26].

6.2 Definition of the Injury

6.2.1 Anatomy

The calf muscle complex consists of the plantaris muscle, the soleus muscle, and the gastrocnemius muscle, which can be divided into a medial and a lateral head.

The plantaris muscle originates from the supracondylar ridge of the lateral femoral condyle and courses toward the posteromedial side of the lower leg. It inserts just medial of the Achilles tendon on the calcaneus. The plantaris muscle is located between the more superficial gastrocnemius and the deeper soleus muscle. The general function of this muscle group is plantarflexion of the ankle [29].

The soleus muscle originates from the proximal part (±1/2) of the posterior tibia along the soleal line and the proximal part (±1/3) of the posterior fibula.

The gastrocnemius muscle spans three joints: the knee, ankle, and subtalar joint. The gastrocnemius is a bipennate muscle; the lateral head originates from the posterior aspect of the lateral femoral condyle, whereas the medial head arises from the medial femoral condyle.

The soleus and gastrocnemius muscles together insert on the calcaneus via a common tendon, the calcaneal or Achilles tendon.

6.2.2 Injury Mechanism

A differentiation must be made between a sprain and a strain. A sprain is a ligamentous injury caused by being stretched beyond its capacity. A tear in a muscle caused in the same manner is called a strain and usually occurs at the region adjacent to the musculotendinous junction [13]. In this chapter we focus on the muscle pathology.

Calf strains are often incurred during acceleration or an abrupt change in direction while running [14, 23]. The gastrocnemius muscle is the most frequently injured calf muscle, followed by the plantaris and soleus muscle [10].

The gastrocnemius muscle is especially vulnerable to injury since it spans three joints. Injury commonly occurs during eccentric contraction when the ankle is forced in dorsiflexion with an extended knee. It occurs mainly during racquet sports, running, basketball, football, and skiing.

Soleus and plantaris muscle injuries are less common than gastrocnemius muscle injuries. Similar to gastrocnemius muscle injury, plantaris muscle injury occurs when the ankle is forced in dorsiflexion while the knee is extended. Soleus muscle injuries, on the other hand, occur when the ankle is passively dorsiflexed while the knee is flexed. This striking difference in pathophysiology is due to the fact that the gastrocnemius and plantaris muscle cross the knee joint and the soleus muscle does not. These injuries occur during sports such as running and volleyball, or simply during stepping off a curb without evident trauma [6].

6.3 Symptoms and Signs

The patient typically complains of acute pain in the proximal calf [6, 10, 12, 20, 21, 23, 24, 27]. Other frequent symptoms include cramping, muscle weakness, ecchymosis, and significant swelling [6, 20, 21, 23]. Because of the muscle weakness, patients complain of difficulty to bear weight or stand on the toes with the affected leg.

Clinically, it may be difficult to distinguish among gastrocnemius, plantaris, and soleus injuries. On top of that, a more distal calf injury may present as a proximal Achilles tendon rupture or strain due to the anatomical proximity. This is emphasized by the frequently described sensation of tearing, popping of the tendon or muscle, or the feeling as if the calf has been kicked or was struck [6, 22, 23].

Calf injuries occur more commonly in men than in women [6, 10, 21, 23]. These injuries usually affect athletes and people in the fourth to sixth decades of life.

Medial calf injuries are most commonly seen acutely, but up to 20 % of affected patients have reported calf tightness being present several days before the injury, suggesting a potential chronic predisposition [6, 21, 23].

6.3.1 Physical Examination

6.3.1.1 Gastrocnemius

On inspection, a (horizontal) ecchymosis can be found at the location of the injury [6, 21, 27]. On examination, the patient experiences pain or soreness at the site of injury [6, 21, 22, 27] usually located at the medial belly [5]. A rupture may be palpable if there is retraction of the muscle. As previously mentioned, it may be difficult to clinically distinguish a muscle tear from a proximal tear of the Achilles tendon [6, 12, 23, 24, 27]. The pain is often elicited with passive stretching of the calf or with plantar flexion against resistance. One might differentiate between gastrocnemius and soleus muscle injury by varying the angle of the knee at which muscle strength is tested. With the knee fully flexed, the soleus muscle is the primary generator of force during plantarflexion. Conversely, the gastrocnemius muscle is the primary generator of force when the knee is in full extension. This technique can also be used in testing pain and flexibility during passive movements. Note that tears in both muscles may occur simultaneously [5]. Besides differentiating between the calf muscles, it is essential to rule out compartment syndrome, which typically presents itself as an extremely tense and painful muscle compartment with or without impaired neurovascular function [3, 6].

6.3.1.2 Plantaris and Soleus

A plantaris or soleus injury typically presents itself with an acute swelling and ecchymosis of the calf [6, 16, 18]. The pain is exacerbated on weight-bearing and with passive dorsiflexion of the ankle and palpation over the site of injury, usually located laterally [5]. It has been suggested that soleus and/or plantaris injuries can be differentiated from gastrocnemius injuries based on less extensive pain and swelling. This is due to the size of the muscles [6, 16].

6.3.2 Clinical Classification

Based on clinical presentation, injuries of the calf muscle complex are graded 1–3 (Table 6.1). These grades can be used to estimate convalescence period and to elect a suitable rehabilitation program [17].

All muscle strains are graded 1–3, with a grade 1 injury being a minor tear with up to 10 % of muscle fibers involved. A grade 2 injury involves tearing up to 50 % of the muscle fibers, and a grade 3 injury represents tearing over 50 % of the muscle fibers, including complete ruptures [10].

Table 6.1 Clinical classification of calf muscle injury

Grade 1 (mild)	Sharp pain in the posterior aspect of the leg at time of injury or with activity May be able to continue activity Mild pain, spasm, and swelling Minimal loss of strength and range of motion (ROM) Tightness and aching for up to 2–5 days after injury
Grade 2 (moderate)	Sharp pain in the posterior aspect of the leg Unable to continue activity Swelling Mild to moderate bruising Loss of strength and ROM Tightness and aching for about 2 weeks
Grade 3 (severe)	Severe immediate pain and disability Considerable swelling and bruising Complete loss of function Palpable defect or mass (in case of full thickness rupture)

6.4 Imaging

MRI or ultrasound can be of value in both the initial assessment and follow-up of the injury. Besides proper management decisions, imaging plays a role in providing an accurate prognosis and rehabilitation strategies [4, 17].

Although ultrasound is inexpensive and widely available, it is subject to both intra- and interobserver variability and has lower sensitivity for ongoing muscle healing than MRI, leading to a more inaccurate prediction of convalescence time as well as risk of recurrent injury. MRI is thus preferred over ultrasound (Figs. 6.1 and 6.2). However, ultrasound guidance can be particularly useful in draining large hematomas.

To correlate MR images with clinical findings, a skin marker is placed over the area of symptoms. Acute muscle injuries commonly lead to altered water content in affected musculotendinous units. This can be seen as a hyperintense feather-shaped pattern in the muscle on fluid-sensitive sequences (e.g., fat-suppressed T2-weighted, proton density-weighted, and STIR sequences). T1-weighted sequences are able to differentiate between edema (hypointensity) and hematoma (hyperintensity), along with detection of atrophy and fatty infiltration [17]. Muscle injuries can also be graded based on radiological findings as shown in Table 6.2.

6.5 Treatment

6.5.1 Nonoperative

Calf muscle injuries have a relatively good prognosis and are principally treated conservatively. Treatment is initiated according to the RICE schedule. It includes rest, ice application, compression wrapping, and elevation (Appendix). Additional

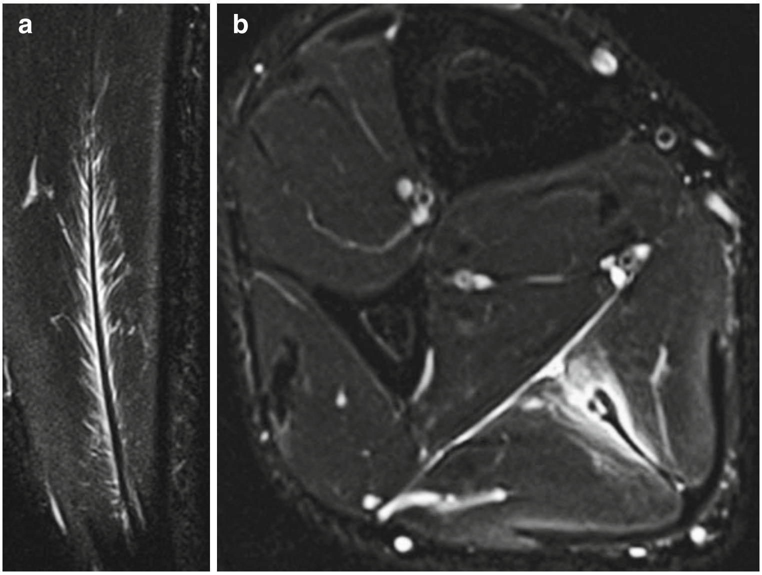

Fig. 6.1 Sagittal (**a**) and axial (**b**) MR images of soleus muscle injury. Edema can be seen at the musculotendinous junction in a characteristic feather-shaped pattern (grade 1 injury) (Reprinted with permission of Thieme [25])

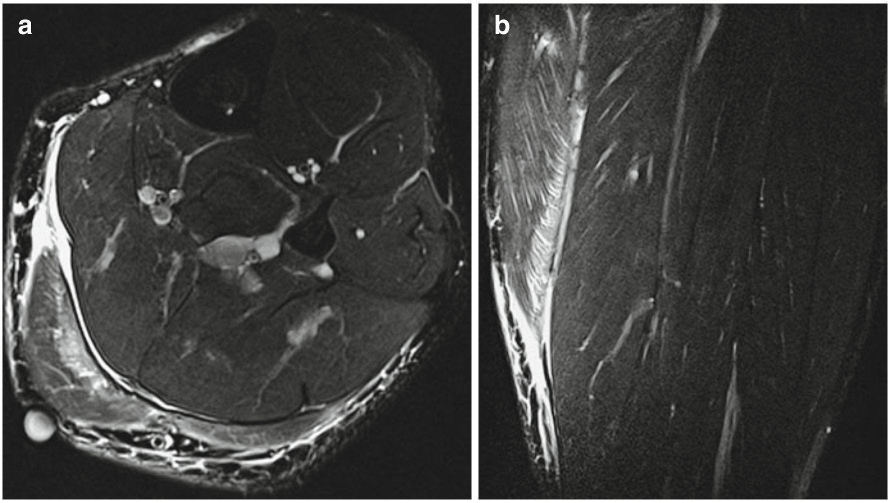

Fig. 6.2 Axial (**a**) and sagittal (**b**) MR images of a partial avulsion of the gastrocnemius muscle (medial head). Note the extension of edema along the fascial planes (grade 2 injury) (Reprinted with permission of Thieme [25])

therapy consists of pain relief and reduction of muscle spasms to facilitate an early start of the rehabilitation program. Weight-bearing and walking are allowed as soon as they are tolerated, and the level of activity is gradually increased as symptoms

Table 6.2 Radiological classification of calf muscle injury

Grade 1 (mild)	Intramuscular feather-shaped hyperintensity without muscle fiber disruption
Grade 2 (moderate)	Hyperintensity (edema and/or hematoma) intramuscular or in the musculotendinous junction with extension along fascial planes between muscle groups Irregularity of tendon fibers
Grade 3 (severe)	Complete discontinuity of muscle fibers with possible retraction of the tendon Extensive edema and/or hematoma

abate. The rehabilitation program focuses on gentle stretching, massage, strengthening exercises, and balance exercises along with core strengthening and general conditioning. Resumption of sports should not be allowed until symptoms have fully subsided and range of motion and strength are symmetrical. This may take up to 3–4 months [5, 6].

6.5.2 Operative

Few have published on operative intervention for injured calf muscle complex and primarily focused on pressure release to prevent limb-threatening pathology (compartment syndrome). A recent report of two cases outlined a surgical treatment of gastrocnemius muscle ruptures. Excision of scar tissue, often seen in chronic injury, is performed, and approximation of the muscle fibers is achieved by means of absorbable and nonabsorbable sutures. Postoperative rehabilitation followed after a 3-week-long leg cast with the ankle plantar flexed (20–30°) and a below-the-knee cast for an additional 3 weeks [7]. In our hands an operation is rarely needed. It is reserved for a severe muscle tear in athletes that is combined with a heavy and painful swelling of the calf due to a severe hematoma. Sometimes, approximation of the muscle to the tendon is needed.

6.6 Prevention

To prevent calf muscle injury, it is advisable to keep the muscle strong so it can absorb the sudden physical stress during activity [6, 14, 21]. Also, we propose an adequate warming-up and stretching of the calf muscles before engaging in activity.

6.7 Future Treatment

The use of treatment modalities based on biologicals is a popular topic for musculoskeletal disorders. Many studies have evaluated platelet- and growth factor-enriched plasma for tendon pathology; results however vary substantially between studies and affected pathology [2, 8, 28]. Hitherto, no studies have focused on biologicals purely for calf muscle pathology. A number of growth factors released by platelets, such as PDGF, VEGF, IGF-1, TGF beta, and FGF, promote repair in various soft tissue models. With the results of enriched plasma for other musculoskeletal pathologies in mind, it is a promising future treatment option. As with enriched plasma modalities, other upcoming treatment options also lack any evidence for the here-described pathology. One of them is mesenchymal stem cell (MSC) therapy: regeneration of healthy muscle tissue involves infiltration of tissue- and vascular-derived cells into the wound area, releasing a cascade of mediators (GFs, BMPs, cytokines, and neuropeptides). The hypothetical benefit of MSC therapy lies in the molecular approaches by which MSC, along with genetically modified cells and gene therapy, can synthesize and deliver the desired growth factor in a temporarily and spatially orchestrated manner to the site of injury [1, 9].

The current lack of knowledge can be regarded as a contraindication because it is unsure whether the used modalities will enhance regeneration of functional musculous tissue or the formation of scar tissue. This is a serious concern and should be studied meticulously before it is ready to be used in daily clinical practice.

Conclusion

Calf muscle injuries are one of the most common sports injuries. Most injuries are sustained during acceleration while running or during an abrupt change of direction. The immediate treatment is according to the RICE scenario: rest, ice, compression, and elevation. It is essential to have the clinical diagnosis confirmed with imaging in order to treat the injury and assess the healing process. The prognosis is generally good, although strongly depending on a gradual rehabilitation program. The treatment of choice is conservative and focuses on stretching and strengthening of the calf musculature. Exercises should be supervised by a physical therapist. Surgical treatment is generally not indicated. Injury prevention is based primarily on muscle strength and flexibility, enabling to cope with (sudden) physical stress during the activity.

Appendix: Rehabilitation Program

	Stage 1	Stage 2	Stage 3
Grade 1 (mild)	Days: 0 RICE Rest as much as possible Ice (15 min every 2–3 h) Compression wrapping Elevation	Days: 3+ Avoid aggravating activities Start with careful stretching of the calf muscles three to five times a day (once it is comfortable to do so) Start with sports massage Continue to wear a compression bandage	Days: 7+ Continue daily stretching Continue with sports massage Start with strengthening exercises (resistance bands can be used initially followed by calf raises and subsequently single leg calf raises) Gradually incorporate some running into your program (once you can achieve 3 sets of 20 single leg calf raises, pain-free) Exercises should be pain-free
Grade 2 (moderate)	Days: 0+ RICE Rest as much as possible Ice (15 min every 2–3 h) Compression wrapping Elevation	Days: 7+ Avoid aggravating activities Start with careful stretching of the calf muscles three to five times a day (once it is comfortable to do so) Start with sports massage Continue to wear a compression bandage Instead of icing, try alternating hot and cold for 5 min each, for 20–30 min three times a day Electrotherapy may be beneficial	Days: 14+ Continue to stretch regularly Continue with sports massage if necessary Start with strengthening exercises (resistance bands can be used initially followed by calf raises and subsequently single leg calf raises) Gradually incorporate some running into your program (once you can achieve 3 sets of 20 single leg calf raises, pain-free) Exercises should be pain-free
Grade 3 (severe)	Stop the activity and rest immediately In case of complete loss of function or considerable swelling and bruising, see a physician immediately In case of a grade 3 injury, the usual treatment involves an operation If the muscle is not completely torn, follow a *grade 2* program		

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Pectoralis Major Rupture

7

Robert A. Magnussen, Matthias Jacobi,
and Elvire Servien

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Abstract

Injuries to the pectoralis major muscle range in severity from mild strains and contusions to complete ruptures. Careful physical examination and a high level of suspicion based on history are keys to accurate and timely diagnosis. Injuries are most common in males in the 20s and 30s and occur when load is applied to an eccentrically contracting muscle. In the pectoralis major, this situation frequently occurs when the arm is maximally loaded in a position of forward flexion, abduction, and external rotation. Injuries are classified based on tear location and severity. Physical examination of an acute rupture typically reveals significant bruising of the chest, axilla, and arm, along with loss of the axillary fold. The diagnosis can be confirmed with ultrasound or MRI. While contusions and partial tears can often be managed nonoperatively, complete ruptures near the distal insertion are generally treated surgically. A variety of repair techniques have been described.

7.1 Introduction

Injuries to the pectoralis major muscle range in severity from mild strains and contusions to complete ruptures. Injuries typically result from eccentric contraction of the muscle with the arm in an extended and externally rotated position [31]. The initial description of the injury dates from 1822 and occurred in a butcher attempting to lift a large piece of beef [28]. The injury remained primarily occupational and quite rare until recent years, when sporting activities have become the most common etiology [21, 31].

Although acute complete ruptures are generally associated with significant bruising and swelling, presentation can be more subtle in more chronic cases and in the elderly. Careful physical examination and a high level of suspicion based on history are keys to accurate and timely diagnosis.

7.2 Definition of the Injury

7.2.1 Anatomy

The pectoralis major is a triangular-shaped muscle overlying the rib cage and pectoralis minor muscle on the superolateral chest. The muscle can be divided into two distinct portions based on their origins: the clavicular head and the sternocostal head, both of which insert on the anterior humerus just lateral to the bicipital groove (Fig. 7.1) [12, 31]. The clavicular fibers attach distally and laterally in the insertion site, while fibers from the manubrium and second through fourth ribs attach in the central portion. Inferior fibers originating from the fifth and sixth ribs and external oblique aponeurosis are multipennate and are noted to twist 180° prior to their insertion proximally and medially in the footprint [41]. With humeral abduction, these fibers undergo significantly more stretch than those originating more superiorly,

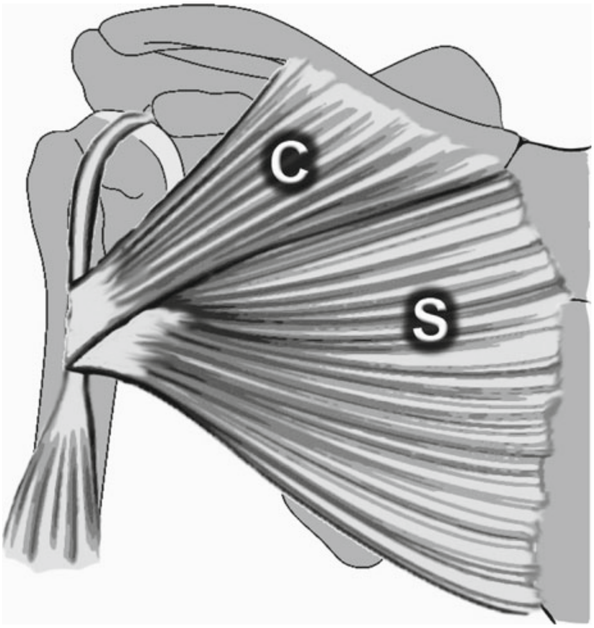

Fig. 7.1 Drawing of pectoralis major origin and insertion on a right upper extremity. The clavicular head (*C*) takes origin on the clavicle and inserts on the humerus just lateral to the bicipital groove. The sternocostal head (*S*) takes origin from the sternum and inserts on the humerus just lateral to the bicipital groove, medial and proximal to the fibers of the clavicular head

which may be related to the propensity of partial tears to involve these inferior fibers [41]. The insertion site measures approximately 5.4 cm in length and 3–4 mm in width with its widest portion located distally [15].

The pectoralis major is innervated by the medial and lateral pectoral nerves, which are named for the respective cords of the brachial plexus from which they originate. The lateral pectoral nerve innervates the majority of the muscle, with the medial pectoral nerve supplying much of the innervation to the pectoralis minor and the inferior portion of the pectoralis major [5, 19, 31]. Its primary blood supply is from the pectoral branch of the thoracoacromial artery, with smaller contributions from the clavicular branch and internal mammary arteries [9].

7.2.2 Injury Mechanism

Injuries to the pectoralis major can occur from several mechanisms. First, a direct blow to the muscle belly can lead to disruption of the fibers and formation of a large hematoma [12]. Traction injury to the muscle is more common and can lead to injury at the muscle's origin on the chest wall, at the myotendinous junction, or near its insertion [6, 25, 41]. The highest forces occur when load is applied to an eccentrically contracting muscle. In the pectoralis major, this situation frequently occurs when the arm is maximally loaded in a position of forward flexion, abduction, and external rotation. This arm position places the inferior fibers of the muscle in a position of mechanical disadvantage and leads to very high forces in this region [41]. Tears frequently begin in this inferior region and progress superiorly if loads persist [41].

Pectoralis major ruptures have traditionally been described as occurring almost exclusively in men in their 20s and 30s, likely related to their involvement in activities that place them at risk [1, 31]. A recent prospective cohort study in a military population confirmed that the injury typically affects males and noted a significantly increased risk in black soldiers relative to white soldiers [40]. By far the most common activity leading to injury of the pectoralis major muscle is weightlifting. The bench press in particular places the muscle at high risk for injury, especially as the muscle contracts eccentrically while lowering the weight to the chest [21]. Anabolic steroid use in such athletes has been noted to predispose to pectoralis major rupture [11, 18]. Pectoralis major rupture has also been reported in football players [27], windsurfers [13], water-skiers [22], elderly [8], and in association with shoulder dislocations [4] and seatbelt injuries [16]. Bilateral ruptures have also been reported [29, 38].

7.3 Symptoms and Signs

Patients suffering a muscle contusion report typical symptoms of pain and discoloration with short-term limitation of function secondary to pain. Patients suffering an acute complete rupture typically describe a sensation of tearing in their axilla or hear a "pop" followed by a significant loss of strength in the affected extremity [3]. Patients presenting with chronic injuries may report weakness in the affected extremity and muscular asymmetry.

7.3.1 Physical Examination

Physical examination of an acute rupture typically reveals significant bruising of the chest, axilla, and arm depending on the location of rupture (Fig. 7.2a). Pain on palpation as well as with motion of the shoulder is to be expected. Weakness in shoulder adduction, internal rotation, and to a lesser extent forward flexion are also common. Unfortunately these signs are common to numerous injuries in this region.

A more specific sign of pectoralis major rupture is loss or thinning of the axillary fold (Fig. 7.2b) [31]. This finding is best noted in comparison with the contralateral side and may be accentuated by abduction [22] or resisted adduction [20] of the shoulder. Additionally, one may note a bulge on the chest wall in cases of more distal rupture or in the axilla in cases of proximal rupture associated with palpable gaps in the tendon or muscle tissue. One should note that associated swelling and pain may limit the effectiveness of physical examination in diagnosing this injury and localizing the tear [41].

Chronic ruptures may present with atrophy of the pectoralis major muscle on the affected side and possibly dimpling of the skin due to an underlying scar tissue in the region [31]. A careful physical exam is often quite successful in making the diagnosis if one maintains a high index of suspicion.

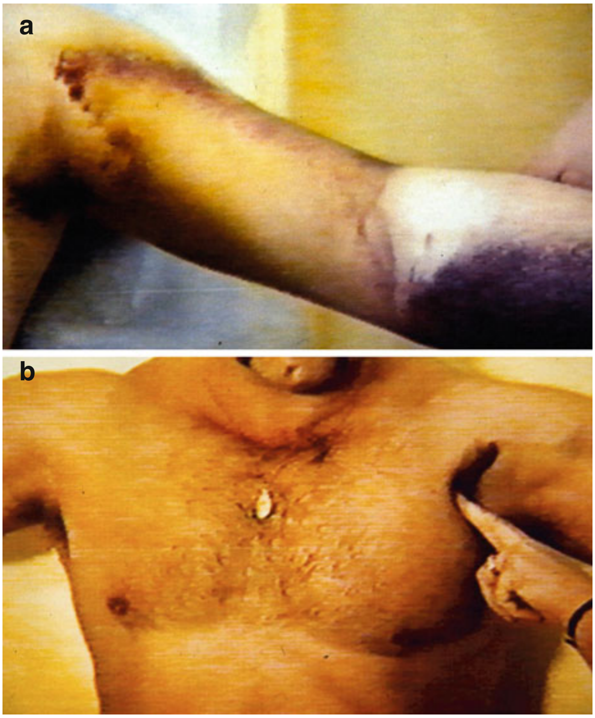

Fig. 7.2 Clinical photographs following pectoralis major rupture. Bruising can be severe, extending from the axilla into the forearm (**a**). Bruising associated with loss or thinning of the axillary fold relative to the contralateral side is also a common finding (**b**) (Image courtesy of Jean-François Luciani)

Table 7.1 Tietjen's [37] classification of pectoralis major injuries as modified by Bak et al. [6]

	Description	Relative incidence
Type I	Contusion	Unknown
Type II	Partial rupture	9 %
Type III	Complete rupture	91 %
A	Muscle origin rupture	1 %[a]
B	Muscle belly rupture	1 %[a]
C	Musculotendinous junction rupture	27 %[a]
D	Muscle tendon avulsion	62 %[a]
E	Bony avulsion of the insertion	5 %[a]
F	Intratendinous rupture	1 %[a]

[a]Represents the percentage of type III Injuries (complete ruptures)

7.3.2 Clinical Classification

Pectoralis major injuries are typically classified according to the system described by Tietjen [37] and modified by Bak et al. [6], in which muscle contusions are classified as type I injuries, partial tears as type II injuries, and complete ruptures as type III injuries. Type III injuries are further classified as type IIIA to IIIF depending on the specific location of the tear (Table 7.1). Complete ruptures are more common than partial ruptures, with avulsion of the tendon insertion (type IIID) representing the majority of injuries (Table 7.1) [6].

7.4 Imaging

Although physical examination is often sufficient for diagnosis, imaging provides additional useful information regarding tear extent and location and allows the physician to rule out associated injuries.

7.4.1 Plain Radiographs

Plain radiographs are generally normal (with the exception of soft tissue swelling) in cases of isolated pectoralis major muscle injuries. Rarely, one can visualize a bony avulsion injury at the humeral insertion [6, 39], which is more common in the skeletally immature [36]. The greatest value of plain radiographs probably lies in ruling out associated injuries. Pectoralis major ruptures have been reported in association with both anterior shoulder dislocations and proximal humerus fractures [23].

7.4.2 Ultrasound

Ultrasound imaging has proven to be an efficient and cost-effective means of evaluating injuries to the pectoralis major muscle and tendon. It is effective in confirming ruptures in the acute setting (particularly when the comparison to the opposite side is performed) and in localizing tear location [7, 31].

7.4.3 MRI

MRI is generally the most useful imaging modality in assessing pectoralis major injuries (Fig. 7.3). In addition to confirming tear location (which significantly impacts treatment strategy), MRI has been shown effective in differentiating partial and complete tears as well as acute versus chronic injuries [10].

7.5 Treatment

7.5.1 Nonoperative

Pectoralis major contusions and other minor injuries are best treated with rest, ice, and anti-inflammatory medication. If a partial tear is confirmed with MRI or ultrasound, some authors recommend a trial of conservative management [6, 33]. Treatment consists of brief immobilization in adduction and internal rotation followed by gradual restoration of motion following resolution of pain from the initial injury. Gradual strengthening then proceeds after full painless range of motion has been restored. Nonoperative management of complete ruptures generally yields poorer functional outcome [6, 12, 35, 41] and is limited to the elderly and those with low functional demands [8, 17].

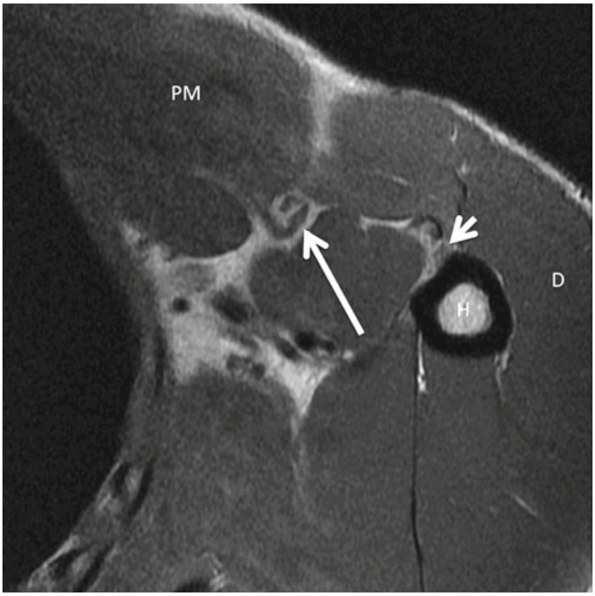

Fig. 7.3 An axial T1-weighted MRI image of a left shoulder of a patient with a complete tear of the pectoralis major (*PM*). The tendon of the pectoralis major has avulsed from the humerus (*H*), where periosteal stripping can be visualized (*short arrow*). The tendon has retracted medially and curled (*large arrow*). The deltoid (*D*) has been labeled (Image courtesy of Ryan L. Hartman)

7.5.2 Operative

Surgical repair is the treatment of choice for complete ruptures of the pectoralis major in active patients. A recent meta-analysis demonstrated that surgical repair yielded less pain and improved function when compared with nonoperative management [6].

Following surgical repair of complete ruptures, the arm should be placed in a sling for 3–6 weeks. Passive range of motion exercises are then initiated and transitioned to light resistance at about 8 weeks postoperative [4, 14, 16, 27, 30, 35, 41]. Full abduction and external rotation should be avoided until at least 6 weeks postoperative [24, 30, 34]. More aggressive strengthening can be initiated 3–4 months postoperative, with an expected return to unrestricted activity at about 6 months [31].

7.5.3 Surgical Technique

Following an approach through the deltopectoral interval and identification of the location of the rupture, the tendon is mobilized to restore its native insertion site. This mobilization can be accomplished through releasing scar around the tendon and muscle belly in all directions. If sufficient length cannot be gained in this manner, one can consider a relaxing incision in the inferomedial portion of the muscle belly to facilitate lateralization [2]. Alternatively, an Achilles allograft can be used to lengthen the pectoralis tendon [20].

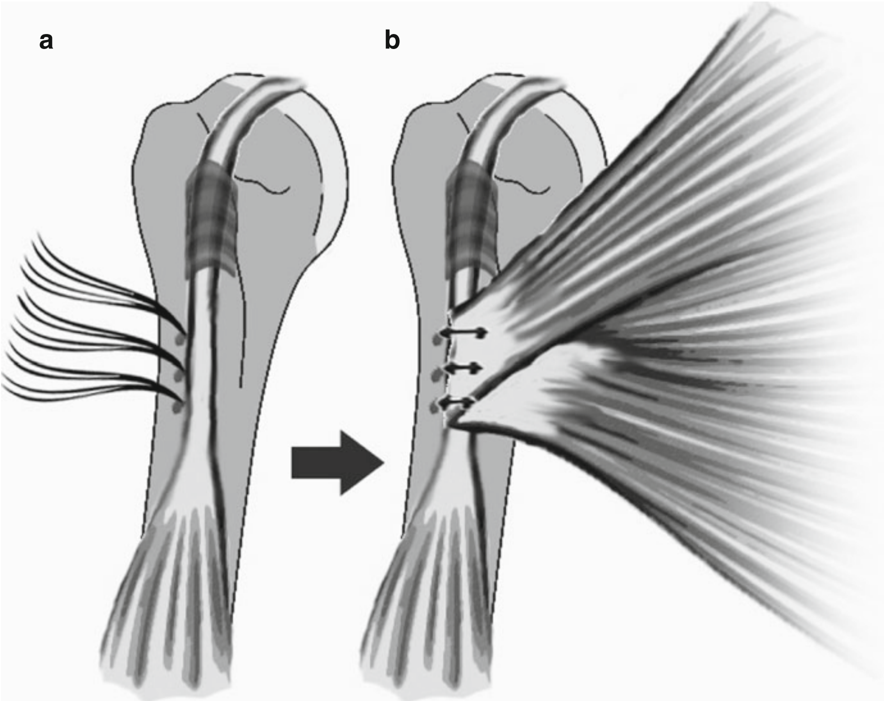

Fig. 7.4 A schematic of the suture anchor technique for repair of a complete pectoralis major tendon avulsion. Suture anchors are first placed in the pectoralis major footprint, just lateral to the bicipital groove (**a**). The sutures are then passed through the tendon and tied, reattaching the tendon to its native position (**b**)

After sufficient tendon length and mobility have been established to restore the humeral insertion of the tendon, a fixation method must be selected. Numerous methods have been described with excellent results including suture anchors (Fig. 7.4) [1, 26], bone tunnels (Fig. 7.5) [31], staples [14], and screws and washers [32].

7.5.4 Findings at Surgery

Ruptures treated acutely are frequently associated with formation of a large hematoma that is encountered following skin incision in the deltopectoral interval. Although tear location varies (Table 7.1), the most common surgical finding is avulsion of the tendon from its humeral attachment with medial retraction [6]. The tendon is generally freely mobile and can easily be reattached to its insertion.

Chronic cases can be significantly more difficult. The formation of fibrous tissue in the gap between the retracted tendon and its insertion may give the false impression that the tendon is intact [35]. Additionally, scarring of the tendon into the muscle belly as well as numerous adhesions to the skin and chest may complicate

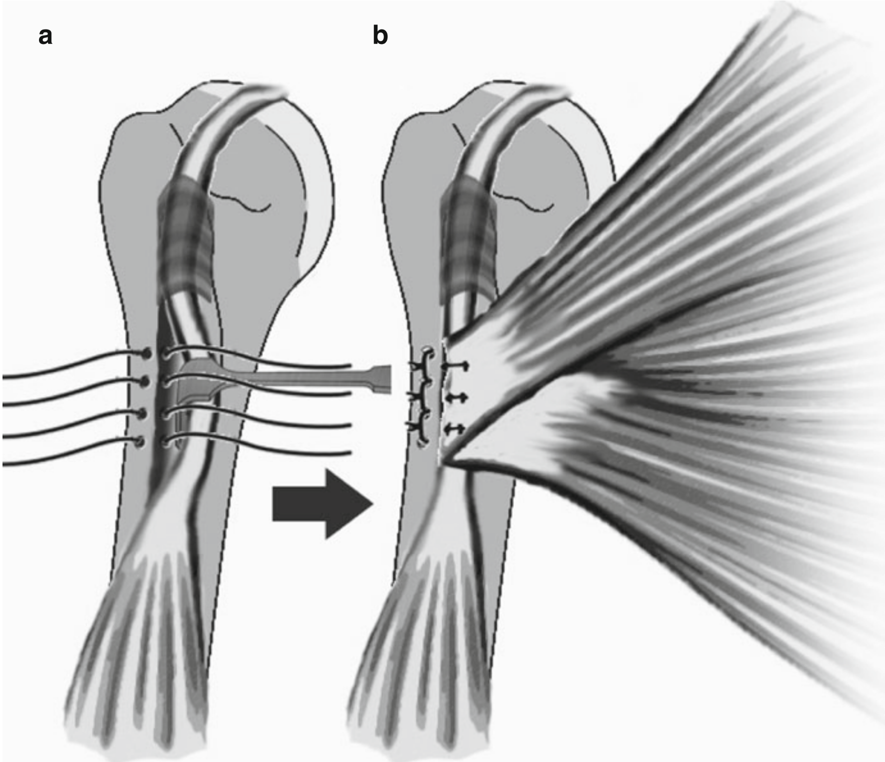

Fig. 7.5 A schematic of the bone tunnel technique for repair of a complete pectoralis major tendon avulsion. Drill holes are created through the pectoralis major footprint, just lateral to the bicipital groove and sutures passed through the holes (**a**). The sutures are then passed through the tendon and tied, reattaching the tendon to its native position (**b**)

identification and mobilization of the tendon prior to reattachment to the humeral insertion [31].

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Acute Biceps Brachii Injuries

8

Robert A. Magnussen, Paul Y. Chong, Luke S. Oh, and Gilles Walch

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Abstract

Acute injuries to the biceps range in severity from mild strains and contusions to complete tendon and muscle belly ruptures. As with many muscles and tendons, injuries tend to occur during eccentric loading of the muscle, such as resisting a force attempting to extend the elbow and pronate the forearm. While distal biceps ruptures are more common, proximal rupture of the long head and more rarely the short head have also been reported. Proximal ruptures can result in a "popeye" deformity, while distal ruptures can be diagnosed on physical examination through the use of the hook test and occasionally the presence of a reverse "popeye" deformity. While plain films are generally negative, MRI or ultrasound can confirm either diagnosis. Complete ruptures of the long head of the biceps tendon may be asymptomatic, particularly in elderly patients and those with lower functional demands. Such injuries are frequently treated nonoperatively. Conservative treatment of complete distal ruptures is generally less favorable, with significant residual weakness and cosmetic deformity noted. Operative treatment is generally indicated, with the exception of the elderly and patients with low functional demands.

8.1 Introduction

The majority of biceps tendon pathology evaluated and treated by orthopedic surgeons is chronic in nature. This pathology most frequently involves the long head of the biceps tendon and is often associated with rotator cuff and labral pathology. This chapter however focuses on acute injuries of the biceps brachii muscle and tendons, both distally and proximally. Acute injuries to the biceps range in severity from mild strains and contusions to complete tendon and muscle belly ruptures. This chapter will address relevant anatomy, injury mechanisms, symptoms, physical exam and imaging findings, and treatment options.

8.2 Definition of the Injury

8.2.1 Anatomy

The proximal origin of the biceps brachii muscle consists of two heads: the long head and the short head. The long head takes its origin on the supraglenoid tubercle inside the shoulder joint capsule where its fibers blend with those of the superior labrum. The tendon passes above the humeral head just posterior to the coracohumeral ligament and enters the bicipital groove on the anterior humeral head [2]. Fibers from the coracohumeral ligament, superior glenohumeral ligament, and subscapularis tendon coalesce to form the biceps pulley that secures the tendon in the groove [43]. The tendon then passes distally in the groove and under the transverse humeral ligament (Fig. 8.1). The total tendon length prior to the myotendinous junction is about 9 cm [20].

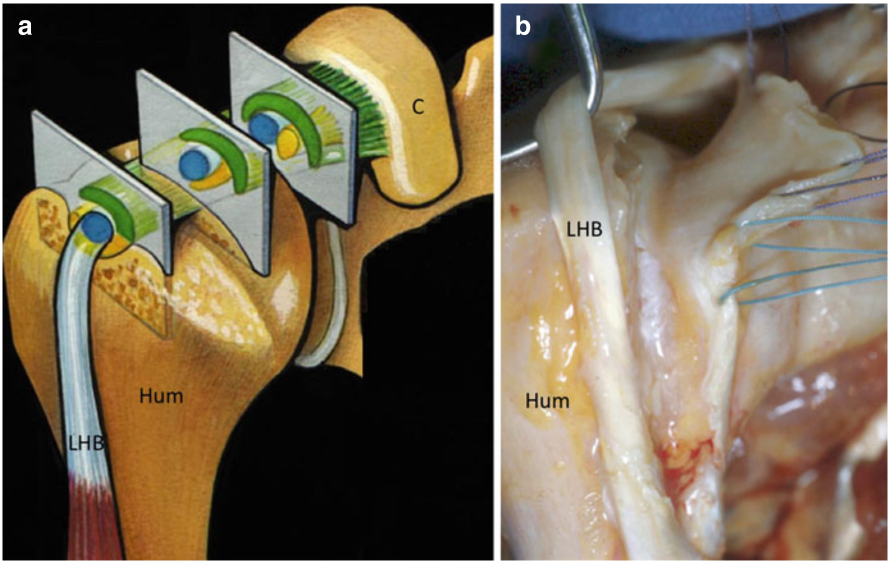

Fig. 8.1 (**a**) Drawing demonstrating the relationship between the long head of the biceps tendon (*LHB*), the coracohumeral ligament (*green*), and superior glenohumeral ligament (*yellow*) as they proceed toward the bicipital groove. The coracoid (*C*) and humerus (*Hum*) are labeled. (**b**) Anatomical dissection of a right shoulder viewed anteriorly. The biceps sling and transverse humeral ligament (*tagged with blue sutures*) have been elevated to reveal the long head biceps tendon (*LHB*), which has been retracted laterally out of its groove on the anterior humerus (*Hum*)

The short head of the biceps tendon takes origin on the tip of the coracoid process just lateral to the attachment of the pectoralis minor tendon. This shared origin with the coracobrachialis is known as the conjoined tendon. Recent work suggests that this origin consists of muscle fibers taking origin directly on the coracoid with an aponeurosis on its anterior surface rather than a true tendon as seen in the long head [10]. The musculocutaneous nerve passes just posterior to the short head prior to entering the muscle belly of the coracobrachialis.

The muscle bellies of both heads of the biceps are located just anterior to the brachialis muscle. Distal to the myotendinous junction, the bicipital aponeurosis (lacertus fibrosus) continues distally and medially while the distal tendon of the biceps crosses the elbow joint and continues toward the radial tuberosity. The fibers rotate as they approach the tuberosity, with medial fibers attaching to the anterior portion of the tuberosity and more lateral fibers attaching more posteriorly [28].

8.2.2 Injury Mechanism

As with many muscles and tendons, injuries tend to occur during eccentric loading of the muscle, such as resisting a force attempting to extend the elbow and pronate the forearm. Injuries can also result from a direct blow to the muscle belly itself.

The long head of the biceps is the most common site of rupture. This injury was first described in 1781 [17, 31]. The vast majority of these injuries are chronic in nature, often found in association with rotator cuff pathology [21, 55]. Acute injuries of the long head of the biceps account for a very small proportion of ruptures [48]. Injuries to the short head of the biceps tendon are also rare. Case reports have associated this injury with a forceful abduction of the shoulder in situations such as waterskiing [12], skydiving [34], and automobile accidents [50]. It has also been reported in cases of minimal trauma in the presence of significant degenerative change in the tendon [42].

Distal ruptures are less common than proximal ruptures, occurring at a rate of 3 per 100,000 people per year and representing between 3 and 10 % of all biceps tendon ruptures [36, 46]. These injuries more commonly affect the dominant arm and are almost exclusively seen in males in their fifth and sixth decades [36]. Distal biceps ruptures are extremely rare in women. Smoking has been noted to increase the risk of a distal biceps rupture sevenfold [46]. Several authors have noted degenerative changes in ruptured tendons, possibly contributing to the risk of a tear [25].

8.3 Symptoms and Signs

Patients suffering a contusion to the muscle belly from a direct blow report typical symptoms of pain and discoloration with short-term limitation of function secondary to pain. Patients suffering an acute complete rupture typically describe feeling or hearing a pop followed by pain and a loss of strength. Bruising and swelling in the arm are commonly reported. Partial ruptures of the long head are often more symptomatic than complete ruptures [48], while more functional loss is typically noted with a distal rupture than a proximal injury.

8.3.1 Physical Examination

Physical examination of an acute rupture may reveal bruising of the upper arm, distal arm, or forearm depending on the location of the rupture. Pain on palpation or with motion of the elbow is to be expected. Weakness in elbow flexion and especially supination is common immediately following injury but may improve with time [45].

Patients with an acute rupture of the long head may experience tenderness over the bicipital groove during palpation, especially in cases of partial tearing [48]. Patients with a complete rupture may present with a "popeye" deformity in which the muscle belly of the long head of the biceps forms a mass in the distal portion of the arm (Fig. 8.2) [22]. Resisted elbow flexion or supination may highlight this finding. Findings following complete rupture of the short head of the biceps are similar. Bruising and tenderness of the proximal medial arm is typically noted along with a palpable gap in the muscle tissue in this same area. A popeye sign is common but is typically located more medial than in cases of rupture of the long head [34, 50].

Fig. 8.2 Clinical photograph of a right arm in a patient with a long head biceps rupture. The classic "popeye" deformity is visible as the muscle belly of the long head of the biceps balls up in the distal portion of the arm

A distal rupture of the biceps tendon often results in significant distal bruising and a palpable gap in the muscle. This gap may not be easily palpable in cases in which the lacertus fibrosus and brachialis tendons are still intact. However, the "hook test" has been demonstrated to be highly specific and sensitive in making the diagnosis [38]. The elbow is flexed to 90° and maximally supinated. The examiner can then hook the lateral edge of the biceps tendon with his or her index finger. Failure to palpate the tendon in this position confirms the diagnosis [38].

8.3.2 Clinical Classification

No formal classification system has been described for acute ruptures of the biceps. Lesions are typically described by location (long head, short head, or distal). Ruptures of the long head of the biceps can either involve the tendon itself or avulse a portion of the superior labrum. This situation represents a type 2 SLAP (superior labrum anterior posterior) tear [51].

8.4 Imaging

Although physical examination is often sufficient for diagnosis, imaging studies often provide additional useful information regarding tear extent and location and allow the physician to rule out associated injuries.

8.4.1 Plain Radiographs

Plain radiographs are generally normal (with the exception of soft tissue swelling) in cases of isolated biceps muscle injuries. Plain radiographs are generally not useful in cases of proximal tendon rupture. Rarely, one can visualize irregularities of the radial tuberosity associated with a distal avulsion injury [44]. The greatest value of plain radiographs probably lies in ruling out associated elbow injury in cases of distal rupture [53].

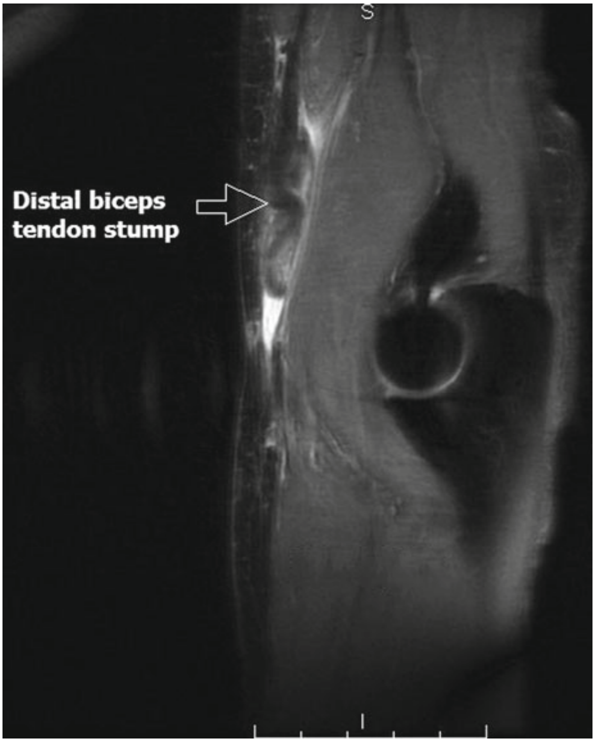

Fig. 8.3 Sagittal plane MRI image demonstrated a complete rupture of the distal biceps tendon. The tendon can be seen retracted proximally in the arm (*arrow*) (© Luke S. Oh, MD, Boston, MA, USA)

8.4.2 Ultrasound

Ultrasound imaging has proven to be an efficient and cost-effective means of evaluating both proximal [35] and distal [4, 19] injuries to the biceps tendon. Advantages include its low cost and rapidity, although its sensitivity and specificity are highly operator dependent [19].

8.4.3 Magnetic Resonance Imaging (MRI)

MRI is frequently the most useful imaging modality when evaluating potential tears of the biceps. Proximally, MRI is used in differentiating partial from complete ruptures, the degree of retraction of complete tears, and in ruling out other intra-articular shoulder pathology. Both SLAP tears [7] and rotator cuff injuries [14, 21] are frequently associated with long head biceps ruptures. MRI can be especially useful in the evaluation of distal ruptures (Fig. 8.3). It has been shown effective in differentiating partial from complete tears and defining other pathology such teno-synovitis and hematomas [15].

8.5 Treatment

8.5.1 Nonoperative

Biceps contusions and other minor injuries are best treated with rest, ice, and anti-inflammatory medication. Complete ruptures of the long head of the biceps tendon may be asymptomatic, particularly in elderly patients and those with lower functional demands [52]. However, more recent comparative studies have demonstrated poorer functional outcomes and cosmetic appearance with nonoperative management, especially in patients with high functional demands [32]. Partial ruptures of the long head of the biceps tendon are generally due to underlying degenerative change and can be quite painful [48]. Natural progression to complete rupture may result in significant improvement of symptoms.

Distally, partial biceps tendon tears are less common than complete tears. Several authors have reported good results of conservative management of partial tears [13, 18] and recommend a trial of conservative management in these patients. Conservative treatment of complete ruptures is generally less favorable, with significant residual weakness and cosmetic deformity noted [8]. Nonoperative treatment is generally limited to the elderly and patients with low functional demands.

8.5.2 Operative

Surgical repair is the treatment of choice for symptomatic complete ruptures of the biceps brachii in active patients.

8.5.3 Surgical Technique

Surgical options for the treatment of ruptures of the long head of the biceps include tenotomy or tenodesis in cases of partial rupture and tenodesis in cases of complete rupture. Tenotomy is less likely to result in persistent pain in the bicipital groove but may lead to variably symptomatic weakness in supination or a popeye deformity [24]. Numerous surgical techniques have been described for performing a tenodesis of the biceps tendon. Tenodesis can be performed either into the bicipital groove or more distally in the humeral diaphysis. Tenodesis into the bicipital groove can be performed via an open technique utilizing suture fixation with [23] or without [30] a periosteal flap. Alternatively, the tendon stump can be fixed within the humerus using either the keyhole technique [16], sutures [41], or an interference screw (Fig. 8.4) [37]. Tenodesis into the humeral shaft can also be performed via a subpectoral approach using the same interference screw technique [33]. Subpectoral tenodesis has the theoretical advantage of removing any degenerative and potentially

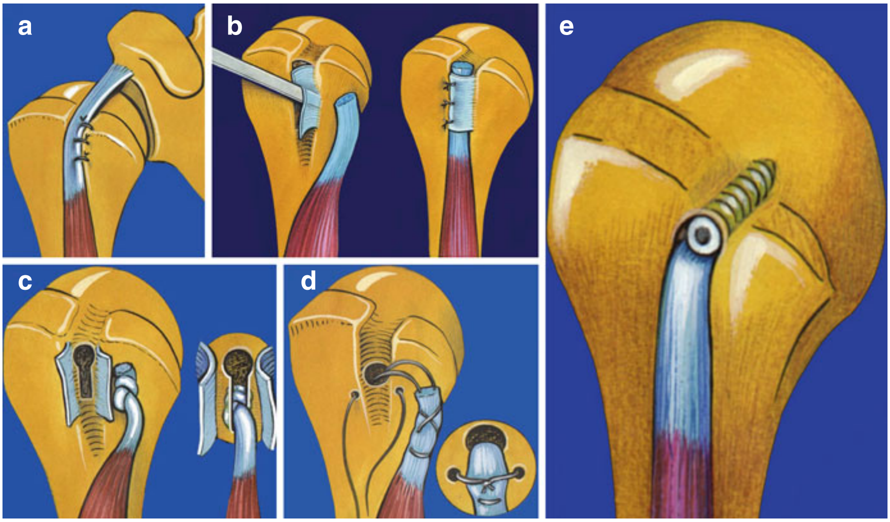

Fig. 8.4 Techniques for long head biceps tenodesis: (**a**) Suture technique of Lippman [30]. (**b**) Suture and periosteal cover technique of Hitchcock [23]. (**c**) Keyhole technique of Fromison [16]. (**d**) Suture technique of Post [41]. (**e**) Interference screw technique

symptomatic tendon from the bicipital groove, but cases of fracture through the tenodesis site have been reported [47]. Arthroscopic techniques for biceps tenodesis have been described in cases of tendon degeneration but are generally not applicable to cases in which rupture has already occurred [5].

There are significantly fewer published reports on surgical treatment of ruptures of the short head of the biceps tendon. Acute primary repair of the ruptured muscle belly is most commonly reported, usually utilizing a Kessler- or Bunnell-type suture technique [34, 50]. One case of tenodesis to the intact coracobrachialis tendon has also been reported with a good outcome [42].

Repairs of distal ruptures generally focus on restoring the insertion of the distal biceps tendon on the radial tuberosity, although tenodesis to the brachialis tendon has also been reported [11, 27]. The tendon and muscle can generally be mobilized to facilitate anatomic repair in acute cases, while augmentation is occasionally required in more chronic situations [45]. Repair was traditionally performed through an extensile Henry approach to the anterior elbow [53]. A high complication rate led Boyd and Anderson to develop the classic two-incision approach to anatomic repair [6]. This approach includes an anterior incision over the distal arm to identify and mobilize the tendon stump and a second approach to the dorsal radius to facilitate fixation (Fig. 8.5). The approach was modified to a muscle-splitting technique by Kelly et al. to avoid damage to the periosteum of the ulna and subsequent high rate of heterotopic ossification [26]. Recent advancements in fixation have also facilitated repair through a single, smaller volar incision [1]. Fixation options for repair of the tendon to the radial tuberosity include the use of bone tunnels [26], cortical buttons [49], suture anchors [29, 54], and interference screws [3, 9].

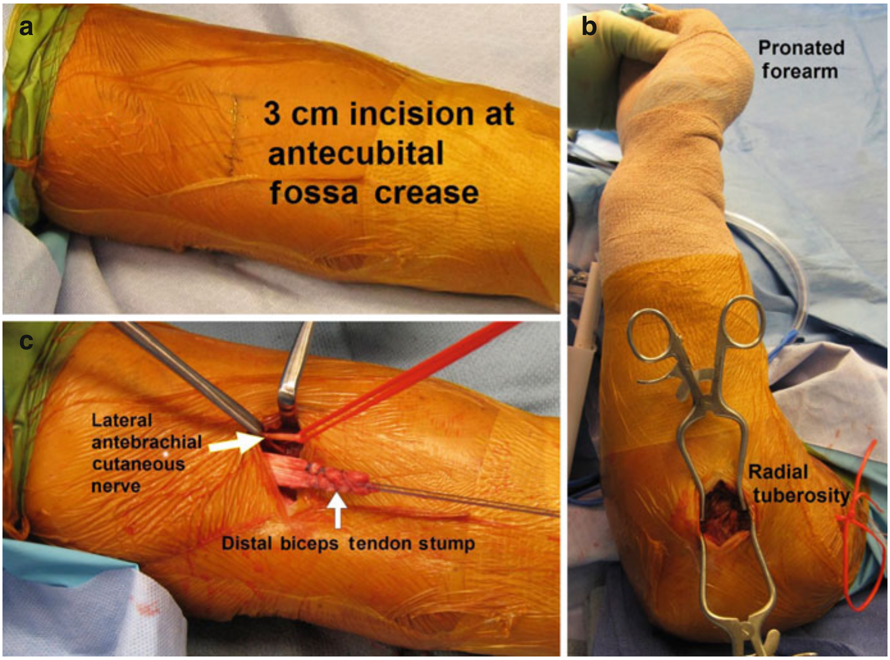

Fig. 8.5 (**a**) Intraoperative photograph demonstrating the small, transverse volar incision to be used for distal biceps tendon repair. (**b**) The distal biceps tendon stump has been identified and prepared for fixation. The lateral antebrachial cutaneous nerve has been identified and protected. (**c**) A small dorsal incision is utilized to access the radial tuberosity for fixation (© Luke S. Oh, MD, Boston, MA, USA)

Biomechanical studies have shown increased stiffness of fixation performed through bone tunnels with a two-incision technique when compared to single-incision repair with suture anchors [40]. However, one comparative clinical study demonstrated no difference in outcome in patients treated with bone tunnels, suture anchors, or cortical buttons [39].

8.5.4 Findings at Surgery

Following rupture of the long head of the biceps, the muscle contracts into the distal part of the arm. The degree of tendon retraction is variable, but the tendon is often quite distal in the bicipital groove and difficult to locate. When it is identified it can generally be mobilized proximally enough to perform a tenodesis into the groove except for some chronic cases where contracture of the muscle prevents tendon mobilization [55].

Distal ruptures of the biceps tendon result in retraction of the muscle and tendon proximally. The status of the lacertus fibrosus and the chronicity of the tear are important predictors of the degree of retraction. If the lacertus fibrosus remains intact, it may limit the degree of proximal retraction and facilitate mobilization of the tendon stump [45]. More chronic cases are generally associated with more retraction and scarring that can complicate identification of the stump.

Conclusion

Acute biceps muscle injuries are relatively rare compared to the more common degenerative conditions associated with lesions of the rotator cuff. Ruptures of the biceps tendon mostly occur during eccentric contraction of the biceps muscle. Physical examination may reveal hematoma and a distalization or proximalization of the muscle belly. Ultrasound and MRI are useful in confirming the diagnosis. Conservative treatment may be effective in cases of proximal rupture and in cases of partial distal rupture. Operative treatment is often preferred in cases of distal ruptures in order to restore muscle function.

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Rectus Abdominis Injury

9

Bruce C. Twaddle and Lucinda Boyer

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9.1 Definition of the Injury

The anatomy of the pubic symphysis region makes it a diagnostic challenge due to the confluence of numerous musculoskeletal attachments, a variety of potential weaknesses in the abdominal wall and its close proximity to both the symphysis pubis and the hip joint.

Injury to the rectus abdominis is one of the areas that fall loosely under the term “groin pain” or “groin injury”. The complex nature of the anatomy of this region and the close proximity of many anatomical structures that could be the source of pain in this area make isolating the exact source of pain felt in the lower abdominal or groin

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region very difficult. The often insidious nature of the onset of pain with a minor initiating injury and the referral or associated pain into the symphysis, the groin, the adductor region and towards the hip makes pain and symptoms in this region a diagnostic dilemma. Consequently, terms such as abdominal strain, athletic pubalgia, sportsman's hernia, Gilmore's groin, hockey goalie syndrome and osteitis pubis can all be attributed in part to injury to the rectus abdominis [1, 4, 5]. As a consequence, "groin strain" has been a curse for trainers, physiotherapists, sports physicians, orthopaedic surgeons and musculoskeletal radiologists alike with individual biases and experiences, often leading to varying schools of thought as to the true origin of pain of the same presenting characteristics and inevitably varying beliefs in "successful" treatment. As is often the case where such a diagnostic dilemma and variation in treatment exists, this suggests that either all treatments have some success, reflective in part of the self-limiting nature of the condition, or none of the treatments are particularly successful. Groin pain in itself can be a very frustrating condition to treat since the difficulties with diagnosis may lead to difficulties in tailoring treatment.

Any muscular injury can be acute or chronic and can be related to a direct blow, strain or overload of the affected muscle, tendon, origin or insertion. Direct blow injuries are usually easily diagnosed by the history, but the ongoing symptoms that can result from any injury to the rectus abdominis can present a clinical challenge. Modern imaging, particularly with MRI, can help reveal potential abnormalities in the region of the pain that may be of clinical significance [6, 7, 11]. Frequently, more than one subtle abnormality can be present and so treatment may need to be for a variety of possible solutions which can be frustrating to physician and patient alike.

This chapter deals specifically with problems and treatment of the rectus abdominis muscle and its insertions on the pubis. It does not in any way suggest that this is the source of all pain and symptoms in the region. Untangling the variety of potential causes of this pain makes medicine the art (as opposed to the science) it still is today.

9.2 Symptoms and Signs

There are no universally agreed classification systems (or systems specifically for injury to the rectus abdominis), and the differential diagnosis is only limited by one's imagination. It is therefore useful to try and define the variety of conditions in the region that could be considered an "injury" to the rectus abdominis and some of the signs that may be attributable to each of the conditions.

The tendons of the rectus abdominis and the adductor longus effectively form a continuous structure, termed by some authors as the "common adductor–rectus abdominis origin" [6, 7] (Figs. 9.1 and 9.2). Biomechanically this acts as a dynamic stabilizer of the pubic symphysis, so that any dysfunction of either tendon unit predisposes the other to failure due to increased load. Secondary loss or dysfunction of the stabilizers of the symphysis can lead to increased load on this joint as well and as a result symptoms and signs in the region can be distributed over a wide area. Injury to the attachment of the posterior wall of the inguinal canal as part of

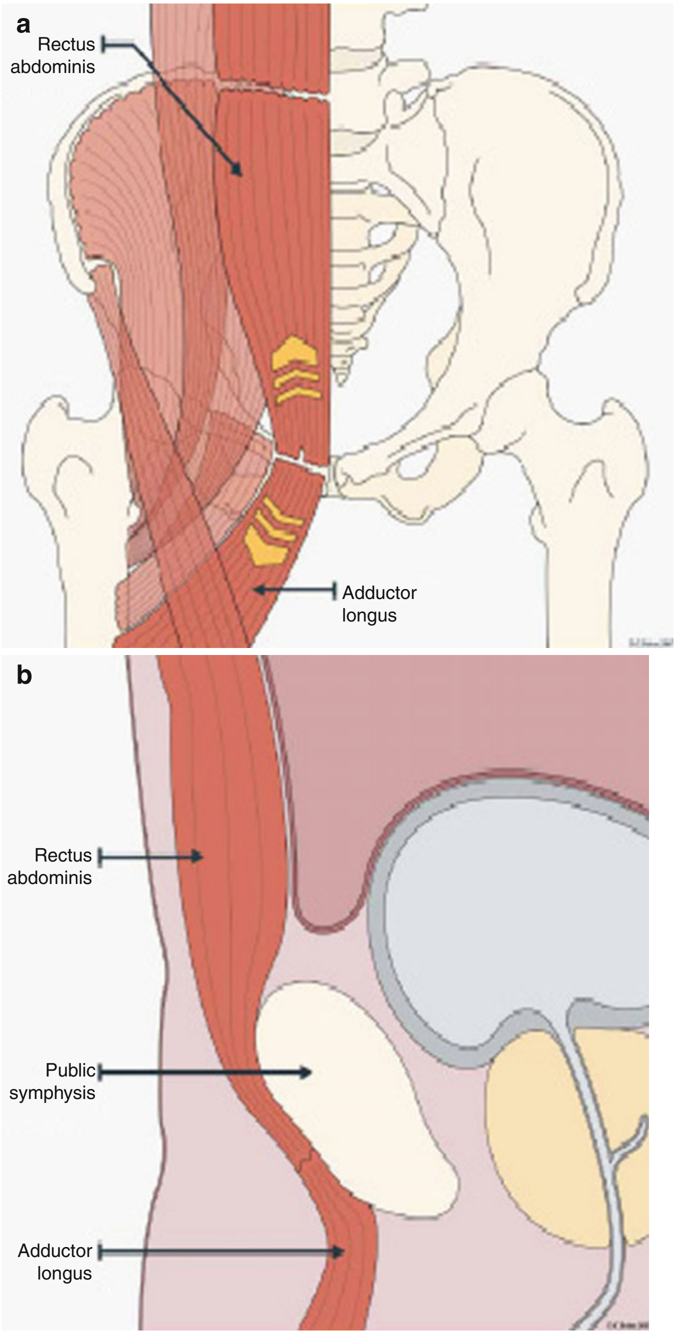

Fig. 9.1 Schematic representation of the confluent but apposing musculotendinous units of rectus abdominis and adductor longus (Imaging provided by Prof Adam C Zoga, Department of Radiology, Thomas Jefferson University, Philadelphia, PA, USA)

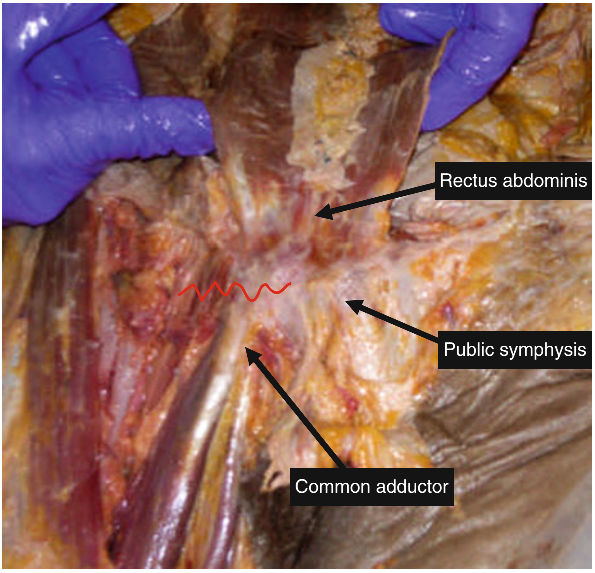

Fig. 9.2 Anatomic dissection of the confluent but opposing musculotendinous units of rectus abdominis and adductor longus (Imaging provided by Prof Adam C Zoga, Department of Radiology, Thomas Jefferson University, Philadelphia, PA, USA)

the anterior rectus sheath can result in hernia formation as part of the spectrum of injuries in this region.

9.2.1 Acute Muscle Tear or Strain

These injuries are the result of a direct blow, rapid twisting or an extension event classically described in elite tennis players. The acute history and localizing tenderness that is aggravated by contraction of the rectus while trying to perform a resisted sit-up, often with associated swelling and bruising, make the initial diagnosis relatively straightforward. The problem for the physician is the ongoing "chronic" symptoms that can result from this injury.

The spectrum of injuries includes local hematoma, partial or complete muscle tear and partial or complete tendon avulsion. The severity of injury is often a reflection of the severity of the trauma with avulsion injuries commonly being associated with pelvic fractures involving disruption of the symphysis and rarely with strains from athletic endeavour.

9.2.2 Rectus Abdominis/Adductor Longus Tendinopathy

The tendon of insertion of the rectus and origin of the adductor are now recognized with anatomical dissection and MRI imaging [9, 11] to be confluent with each other, and their function is antagonistic in stabilizing the pelvis and the symphysis in a variety of activities. Pain in this region can therefore be classically referred into both areas as well as towards the symphysis, and it is almost impossible to accurately

Table 9.1 Blazina grading of patellar tendinopathy

Grade 1	Pain after activity with no functional impairment
Grade 2	Pain at the beginning of activity, disappearing after warm-up, reappearing after activity
Grade 3	Pain during and after activity with functional impairment
Grade 4	Complete tendon rupture

identify the exact source. Such diagnoses fall under the classification of most tendon strains or chronic injuries and can be grouped as the treating physician prefers and finds useful. This can include clinical grading such as that described by Blazina et al. [2] (Table 9.1) in association with the degree of damage defined by imaging studies. Less severe injuries may just have an awareness of pain aggravated by intense loading of the muscles concerned. More symptomatic examples may have pain or discomfort at rest that is initially eased by activity only to return and with increased intensity once the activity has been ceased.

Muscular or tendinous injury in this region is far more common in males than females. Thus, making the diagnosis in a female patient should always make the examiner check for other "pelvic"-related problems as a potential source. The relationship of the pain to the frequency or intensity of activity should allow the examiner to lean towards a tendon/muscle strain or tendinopathy. Aggravation of symptoms by resisted testing of the affected muscle, either the adductor or abdominal "crunch"-type activity, brought on by repeated use or testing can help attribute the symptoms to either one or the other, but often testing of both muscles can bring on the symptoms. Use of local anaesthetic injection may help localize the pathology. However, in more severe examples which have been going on for some time, where the pain can be over a wide area and reference zone, localizing the cause of the pain can be more difficult.

9.2.3 Hernias

A variety of conditions are referred to as "hernias" in this region, and some of them can be associated with weaknesses or injury to the lateral part of the insertion of the rectus abdominis. The term hernia should be reserved for the true definition, which is a protrusion of a loop or knuckle of an organ or tissue through an abnormal opening in the boundary that contains it. By definition, this insertion of rectus abdominis is actually superficial and so it is likely that the other abdominal muscles are at fault. Nevertheless, this weakness and diagnostic category inevitably falls into the broad categorization of rectus abdominis injury. The so-called acquired inguinal wall deficiency [4, 7] is likely to be an overuse condition present in up to 15 % of athletes presenting with groin pain and is really a weakness or deficiency of the inguinal canal that is insufficient to cause discrete hernia formation. It has been called sportsman's hernia, Gilmore's groin, incipient hernia, pubalgia and a variety of other names. Conceptually it can involve the anterior inguinal wall made up of the external oblique aponeurosis, the posterior wall made up of the transversus abdominis and internal oblique or both as they attach to the edge of the symphysis. This condition occurs almost exclusively in males and is usually associated with focal tenderness at the superficial inguinal ring. If the posterior wall is sufficiently disrupted, a small direct hernia may be palpable.

9.2.4 Osteitis Pubis

Use of this term should be reserved for symptoms related to the pubic symphysis joint itself or parasymphyseal rami. These symptoms can often be the result of longstanding dynamic imbalance from injury to the rectus abdominis–adductor unit resulting in increased loading of the symphysis. Characteristically, these patients have a central aching discomfort aggravated by activity and localizing tenderness to clinical examination made worse by bracing manoeuvres or single-leg stance loading. The pain will often linger long after exercise has finished and may create sleep disturbance and early morning symptoms. This central pain is characteristically brought on by resisted adduction of the leg in an abducted position.

9.3 Imaging

9.3.1 MRI Classification and Staging Systems (Table 9.2)

Table 9.2 MRI classification of muscle and tendon injury to the rectus abdominis

Grade I strain Less than 5 % disruption of muscle fibres	
MR	Focal or diffuse increased T2 signal around myotendinous junction Oedema or haemorrhage Feathery appearance within muscle
Grade II strain Partial disruption of muscle or detachment of muscle from aponeurosis or fascia Irregular or thin myotendinous junction	
MR	Increased T2 signal Retraction of torn muscle fibres Myotendinous junction haematoma Fascial fluid
Grade III strain Complete disruption at myotendinous junction ± retraction	
MR	Increased T2 signal Muscle or tendon ends appear rounded Wavy contour of tendons Haematoma within muscle or tendon defect

Table 9.2 (continued)

Muscle contusion	
Direct, blunt trauma	
MR	Increased T2 signal Interstitial oedema Increased muscle size
Hematoma on MR	
Hyperacute (<48 h): haemoglobin T1 isointense to muscle T2 hyperintense	
Subacute: haemoglobin -> methaemoglobin T1 hyperintense T2 hyperintense	
Chronic: hemosiderin or fibrosis T1 hypointense T2 hypointense T2 "blooming" artefact	

In addition, there is a series of features and signs on MRI that are more specific to imaging in this area, and these are outlined in Tables 9.3 and 9.4 [3, 6–11].

9.3.2 Correlation of MRI Findings with Clinical Examination

Zoga et al. [11] published a study reviewing the correlation of MRI and clinical findings in patients with "groin pain"-type diagnoses. Physical examination findings were compared with those at MR imaging in all study patients. Ninety-three (66 %) of 141 patients had physical examination findings that suggested rectus abdominis tendon injury. Of these 93 patients, 60 (65 %) had injury involving the rectus abdominis tendon that was noted at MR imaging. Among the 93 patients with physical examination findings that suggested rectus abdominis tendon injury, 53 (57 %) had bone marrow oedema around the pubic symphysis (regardless of extent or symmetry) and 52 (56 %) had a secondary cleft sign at MR imaging.

Fifteen of 141 patients had physical examination findings that suggested injury isolated to the adductor compartment. Of these 15 patients, nine (60 %) had adductor tendon injury at MR imaging, but three of these nine patients also had MR imaging findings involving the rectus abdominis tendon. Of the 15 patients with isolated adductor tendon injury at physical examination, only three (20 %) had pubic symphysis bone marrow oedema, while four (27 %) had a secondary cleft sign (Fig. 9.3). At physical examination, 71 (50 %) of 141 patients had findings suggestive of injury involving both the rectus abdominis tendon and the adductor compartment. MR imaging revealed both rectus abdominis tendon and adductor tendon injury in only 38 (54 %) of these 71 patients, but 46 (65 %) had bone marrow oedema around the pubic symphysis and 43 (61 %) had a secondary cleft sign (Fig. 9.3).

Table 9.3 Checklist for MRI evaluation of rectus abdominis and adductor aponeurosis injury

Muscle and tendon identification	

Rectus abdominis
  Attachment to superior pubic ramus
  Medial and lateral heads	

Rectus abdominis /adductor aponeurosis Attachment to anteroinferior pubic bone	

Adductor longus
  Tendinous (superficial) and muscular (deep) origin on anterior pubic bone (bone without periosteum)
  Avulsion usually associated with bone fragment	

Adductor brevis Muscular origin posterior to adductor longus	
Adductor tendon injury Myotendinous injury – proximal (or distal)	
Pectineus Origin: lateral pubic symphysis + superior pubic crest	
Obturator externus Origin: posterolateral pubic symphysis	
Gracilis Origin: anteroinferior pubic symphysis and superior pubic arch	
Grading of injury	
Rectus abdominis muscle strain	
Acute	Increased T2 signal
Chronic	Hypertrophic
	Atrophic
Rectus abdominis adductor aponeurosis injury	
(i) Ipsilateral rectus abdominis distal detachment + adductor aponeurosis partial or complete detachment	
(ii) Unilateral rectus abdominis atrophy	
(iii) Unilateral or bilateral adductor aponeurosis injury	
Rectus abdominis/adductor aponeurosis plate disruption	

Secondary cleft sign (Fig. 9.3)
  Anteroinferior pubic bone curvilinear increased T2 signal
  Detachment of the rectus abdominis distal attachment
  Extends across the midline
  Bilateral secondary cleft appearance	

May have associated osteitis pubis	
Maybe associated with injury of pectineus and obturator externus	

At physical examination, 16 patients had findings most compatible with osteitis pubis, without concomitant rectus abdominis insertion or adductor tendon injury. Of these patients, all 16 (100 %) had bone marrow oedema that spanned the pubic symphysis and extended from anterior to posterior at the subchondral region of the symphysis, and none had rectus abdominis insertional injury at MR imaging. At physical examination and MR imaging, osteitis pubis manifests as an arthritis involving the fibrous pubic symphysis joint. However, MR imaging findings of osteitis pubis were not always observed in isolation in this group, as six (38 %) of these

Table 9.4 Imaging of associated pathologies

Symphysis pubis
Bone marrow oedema
Location: Superior = rectus abdominis Anteroinferior = RA/AA Lateral = pectineus Posterolateral = obturator externus
Unilateral or bilateral
Degenerative change Subchondral cystic change Subchondral sclerosis Erosions
Acquired inguinal wall deficiency
Frequent coexistent adductor tendon injury
Anterior inguinal ring insufficiency Tear medial aspect external oblique, resulting in patulous external inguinal ring
Proposed that pain is caused by entrapment of cutaneous branches of the ilioinguinal nerve and genital branch of genitofemoral nerve
Clinical diagnosis
Rare to have findings on MRI or US
MRI – may see hyperintensity of external inguinal ring
Posterior inguinal wall insufficiency
Conjoint tendon or transversalis fascia attachment tear or weakness
Use of imaging debated for diagnosis
US – posterior inguinal wall bulging – not sensitive or specific sign, as can be present in asymptomatic population
MR dynamic imaging – asymmetrical focal protrusion of the posterior inguinal wall without a hernia

16 patients also had adductor compartment injury at MR imaging and seven (44 %) had intrinsic hip disease at MR imaging.

9.4 Findings at Operation

The features at surgery for injuries to the rectus abdominis are in keeping with the specific injuries and their associated pathologies. Surgery to the rectus abdominis itself is rare apart from avulsion reattachments, which are more commonly part of a more significant pelvic injury with symphysis disruption or rami fracture. These may or may not be repairable depending on whether they are a true avulsion or a muscular disruption.

Similarly, surgical repair of sleeve-type avulsion injuries of the adductor origin (which may include one or a series of the adductor muscles) are relatively rare but usually associated with a sleeve of periosteum which can be anchored down to the bone. If performed acutely, the site of this avulsion is usually apparent. Repair/reconstruction in the delayed setting is not a procedure that is commonly described,

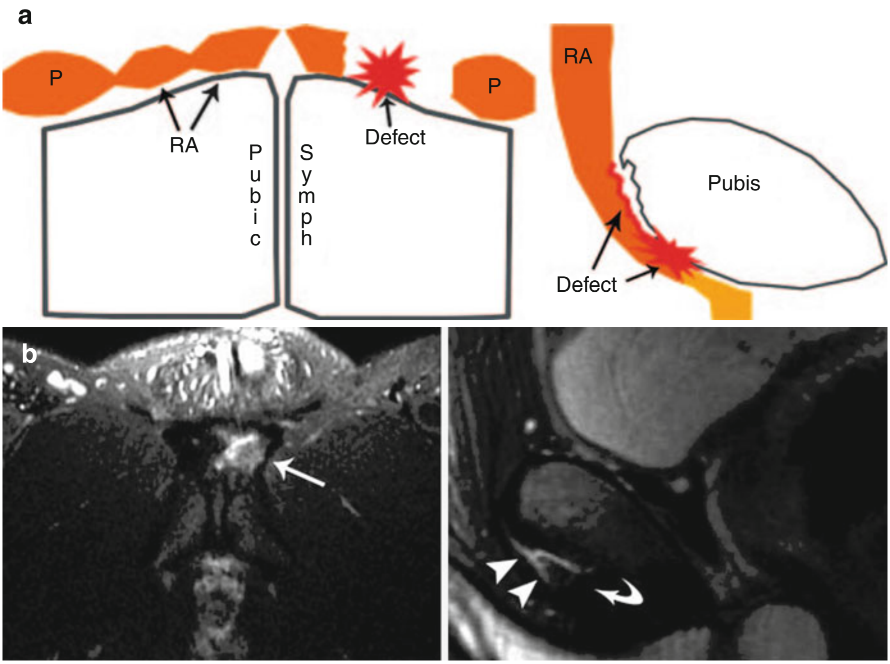

Fig. 9.3 The secondary cleft sign with oedema and fluid identifiable beneath the confluence of the two tendons. (**a**) Schematic representation, (**b**) MRI correlation. *P* psoas, *RA* rectus abdominis (Imaging provided by Prof Adam C Zoga, Department of Radiology, Thomas Jefferson University, Philadelphia, PA, USA)

and few mentions exist in the literature. It seems logical that these injuries (adductor longus avulsions or proximal disruptions) are an extension of the separation and oedema seen in the "secondary cleft" sign described earlier for the rectus abdominis/adductor aponeurosis plate. It is much more common to see disruption of the adductor longus than the rectus abdominis as part of this type of injury. Such a disruption is often associated with preceding symptoms of adductor tendinopathy or "groin" strain. Surgical repair of anything other than a major sleeve avulsion is controversial, and return to sport would appear to be more rapid when conservative treatment is undertaken for adductor longus muscle and tendon ruptures with no documented loss in function. Treatment of symptomatic adductor longus tendinopathy usually involves release of the tendon at its origin from the pubis with reasonably successful reported results and relatively rapid return to sport.

Hernia repair or laparoscopic mesh reinforcement of the abdominal wall are described for the treatment of "sportsman's hernia" or more accurately described "acquired inguinal wall deficiency". It appears that if this is the source of the patient's symptoms, and it is often a diagnosis of exclusion, relief of symptoms can be rewarding.

Unfortunately, due to the already highlighted difficulty of attributing the symptoms of "groin strain" to one particular entity, all surgical treatments for chronic symptoms in this region carry a degree of uncertainty in terms of effecting a reliable and permanent cure.

9.5 Outcome Measures

There are no validated outcome scores for rectus abdominis or groin injuries, and outcomes of this type of injury in terms of return to sport are poorly documented. The outcome of surgical or nonsurgical management has not been compared in any form of robust scientific investigation. It is clear that sports that require “bracing” or a Valsalva manoeuvre such as weightlifting are likely to increase the chance of aggravating the injury after treatment, as are sports that involve rapid trunk rotation such as tennis where rectus abdominis injury is well described. Kicking sports such as football or positions in other sports where kicking is important such as rugby and Australian Rules football can have recalcitrant problems with both adductor tendon and rectus abdominis symptoms as part of “groin strain”. Outcomes of various treatments in terms of return to sport or kicking distance measurements or gains are more the subject of myth and legend than clearly documented results.

As with all tendinopathies, the restoration of strength and the balance between the opposing muscle groups, in this case the adductor longus and rectus abdominis, are the key to the resolution of symptoms. Any treatment must allow some degree of pain relief or increase in loading to allow the patient to gain strength. Any rehabilitation regime must have injury and sports-specific strengthening incorporated into it.

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Muscle Research: Future Perspective on Muscle Analysis

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Abstract

We are still faced with a dearth of scientific knowledge on muscle injuries, as the history of science in this field is relatively short. In the present chapter, the current knowledge on acute muscle injuries is placed in the perspective of this limited scientific history and a reflection is given on which directions to go in muscle injury research in future.

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10.1 Introduction

We are still faced with a dearth of scientific knowledge on muscle injuries. Despite the growing number of publications over the last three decades, our current knowledge on etiology, prognosis, and therapy is based on only 2,000 published injuries, of which the majority is acute hamstring injuries. If we restrict ourselves to level 1 trials, then there are less than 300 injuries examined.

The progress our research has led to for the individual athlete is limited: compared to three decades ago the injury and re-injury rate have not been changed. The relatively short history of acute muscle injury science places our challenges in perspective and should guide us to strengthen our academic base toward a brighter scientific future.

10.2 Risk Factors and Prevention of Muscle Injuries

The device "prevention is better than cure" certainly holds for muscle injuries, as an athlete is not only unable to compete and train for several weeks to months but also charged with a considerable increased risk of recurrent injury after sustaining a first muscle injury. Nonetheless, to date the evidence for preventative measures is limited to only one high-level study, in which the Nordic hamstring exercise was shown to be effective in the prevention of hamstring injury in football [17].

To be able to direct preventative measures to those players at risk for a specific muscle injury, risk factors associated with the injuries need to be identified. Unfortunately, studies published to date on risk factors for muscle injuries have methodological limitations, as they use univariate approaches and have too small sample sizes to detect small to moderate associations. Muscle injuries in sports occur from a complex interaction of multiple risk factors. This multifactorial nature should be taken into account when studying risk factors for muscle injuries by using appropriate multivariate statistical approaches [3, 13]. In addition, sample sizes should be sufficient to study associations of risk factors with injury risk. As clearly depicted by Bahr and Holme [3], to detect moderate associations up to 200 injured subjects are needed. Taking hamstring injuries in football as an example, with a seasonal injury prevalence of 17 %, a sample size of over 1,000 players is needed to study the risk factors with moderate associations. Risk factor studies in the other less prevalent muscle injuries will of course need even larger numbers of athletes.

10.3 Too Short History for Understanding the Injury Mechanism

It is generally assumed that muscle injuries occur as a cause of excessive eccentric action or passive lengthening of the muscle. The passive stretching mechanism is frequently reported in acute adductor longus lesions. In hamstring injuries the mechanisms have been differentiated in sprinting type and stretching type injuries.

This frequently cited assumption is based on only two case series in sprinters [1] and dancers [2] and has not been reproduced. The actual mechanisms of injury are probably more complex than this practical, relevant classification. Kicking for example, a commonly reported hamstring injury situation, comprises both eccentric loading and outer range lengthening of the hamstring and matches with both suggested injury mechanisms.

From biomechanical studies in hamstring injuries, there is some evidence for eccentric loading during the terminal swing phase as a possible injury mechanism during sprinting. However, there are only two cases described in which a hamstring injury occurred during sprinting analysis in a research setting [11, 18]. The increased availability of high-quality recordings by (high-speed) cameras in elite sports might offer an opportunity to study large numbers of injury situations and correlate these with clinical and imaging features. Although this will probably give us some insight into the possible mechanisms, it will always represent an indirect observation of what actually happened at microscopic level.

New innovative imaging techniques should provide insight into muscle injury mechanisms. We dream of high-speed, wireless, mobile, skin-attached ultrasound techniques to catch that single injury moment. That futuristic tool will give us, for example, the answer whether the terminal swing phase is indeed the mechanism of hamstring injury during sprinting.

10.4 Athletes Are Only Interested in the Near Future

After being injured the first question of the athlete, coaching staff, and press is: When can he/she return to play (RTP)? Although our knowledge has improved at a group level for hamstring injuries, previous research has not satisfactorily answered this question for the individual athlete. Documentation of acute adductor and calf muscle lesions is restricted to small case series, and there is no data available on predicting RTP [9].

Multiple clinical and MRI parameters have been shown to correlate with time to RTP in hamstring injuries. Although these prognostic factors are valuable for scientific studies, its limited strength of correlation and variability of time to RTP makes it unsuitable for accurately predicting the time to RTP for an individual athlete.

For example, in the largest series on the prognostic value of MRI in acute hamstring injuries, Ekstrand et al. [8] found that in professional football players, MRI grading was significantly correlated with injury time. This group found, for each grade injury (in days ± standard deviation): grade 0, 8 ± 3, grade I, 17 ± 10, grade II, 22 ± 11, and grade III, 73 ± 60 days. However, by applying these results to an individual athlete with a grade I hamstring injury, we can estimate that there is a 95 % chance that he/she returns to play within 0–37 days (mean 17 days ± two times the standard deviation of 10 days). The athlete, coaching staff, and press will argue that it is a long way from being a satisfactory estimation of the injury time.

A great challenge for future research is to provide tools which can give a more accurate prediction of the injury time. The first step in this research is to identify the relevant prognostic factors. However, as muscle injuries are a complex multifactorial condition, only by combining multiple prognostic factors for injury duration in mathematical models will we ever be able to provide accurate predictions for the individual athlete. Designing and validating an ultimate prognostic model requires a multitude of standardized documented muscle injuries. To answer the athlete's most important and simple question, we are forced to build up a worldwide online muscle injury registration systems.

10.5 Return to Play Decision-Making

It is a major challenge to decide whether an athlete can safely RTP. The high re-injury rate, up to 63 %, reflects this challenge [6]. The re-injuries have been reported predominantly (59 %) to occur in the first month after RTP [4, 7, 16]. Although there is no consensus, in clinical practice an athlete is typically regarded as being ready once full range of motion, full strength, and functional sport-specific activities (e.g., sprinting, jumping, cutting) can be performed asymptomatically [14]. Despite this conventional approach, the decision of functional, physiological, and psychological readiness remains challenging.

The ultimate test whether an athlete is ready to RTP is to mimic the use and loading of the injured muscle as required during (match) play itself. However, failing this ultimate test implies that the athlete suffers a re-injury. Less rigorous tests will reduce the risk of re-injury during testing, but there will always be an uncertainty on the athlete's readiness to RTP. Basic science shows that healing of the injury is incomplete at the time clinical tests indicate recovery and that the majority of athletes can RTP successfully prior to complete tissue healing [14, 15]. A conservative approach by waiting for complete tissue healing would probably decrease the re-injury rate but might unnecessarily extend the RTP.

As stated by Orchard et al. [14, 15], RTP management strategies should not aim at re-injury risk elimination but at re-injury risk evaluation to support RTP decisions. The practical decision-based RTP model of Creighton et al. [5] guides us through three steps. In step 1, medical factors such as age, injury history, psychological state, outcome of clinical tests, and imaging are evaluated. In step 2, sport-specific risk modifiers such as type, level of sport, and player position are evaluated. Finally in step 3, decision modifiers such as timing in season, importance of match (e.g., final), external pressure, and financial conflicts of interest are considered.

Future research should aim at validation of the medical factors and sport risk modifiers to provide mathematical models that can accurately predict re-injury risk to guide RTP decision-making after muscle injury. For example, based on multiple factors such as patient characteristics (e.g., age, sex, previous injuries), injury characteristics (e.g., mechanism, location, extent), and clinical tests (e.g., flexibility, strength, functional field testing), the future ultimate model estimates that a player has a 6 % risk of being re-injured when participating in the upcoming match and 2 % in the second game. The decision to RTP in the upcoming match will differ whether it is a world cup final or a preseason friendly game. Although this model does not provide definitive RTP criteria, it provides an evidence-based risk estimation that fits in a decision-based RTP approach.

10.6 Future Imaging Techniques

10.6.1 Dynamic MRI

Conventional MRI presents a static image, thereby ignoring the dynamic contraction mechanics of the muscles. Dynamic MRI can measure the motion of muscles under different loading conditions. Future studies using dynamic high-resolution MRI can study the musculotendinous morphology and mechanics during contractions and will provide a better insight into contraction mechanics and association with injury risk [12].

10.6.2 Diffuse Tensor Imaging

Diffuse tensor imaging (DTI) is an advanced MRI technique that images detailed muscle fiber structure. DTI measures the movement of water molecules in the tissue. In a healthy muscle tissue, water molecules move more easily along the muscle fibers than in other directions. At the site of muscle fiber disruption, the movement of water molecules is changed. A preliminary study in marathon runners showed that DTI could detect even the lowest grade of muscle fiber disruption due to delayed onset muscle soreness, which could not be detected by conventional MRI [10]. DTI has therefore great potential for quantifying muscle damage and monitoring tissue healing.

10.7 Numbers Needed to Build Up Evidence and History

Our main limitation is that as an individual sports physician, we deal with a too limited number of muscle injuries to justify an experience-based approach. For example, in professional football, with 15 muscle injuries per team per season [7], our most experienced sports physicians will have had managed just 450 muscle injuries in his/her 30 years' career.

As a consequence, to gain expertise and to answer the most important and simple questions, we need to collaborate. Faced with our short research history, a worldwide muscle injury registration system should start today rather than tomorrow.

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