Muscle Injuries: Strains, Contusions, and Ruptures

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Abstract

Muscle injuries among athletic population are common and often occur during sportive activities or training. Most sports-related muscle injuries involve strains, contusions, and uncommonly ruptures. These injuries are responsible for a large proportion of time lost to competition, and particularly for all professional athletes, rapid return to training and competition is a priority. This chapter will discuss the etiology, risk factors, classification, treatment, and possible complications of common types of muscle injuries under the lights of current literature and recent trends.

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Introduction

Muscle injuries are commonly seen in athletic population. A muscle injury was defined as "a traumatic distraction or overuse injury to the muscle leading to a player being unable to fully participate in training or match play" (Ekstrand et al. 2011). During Summer Olympic Games in London 2012, muscle injuries (21.8 %) were reported the second common injury type after ligament sprains (27 %) which resulted with absence from training or competition for more than 1 week. Most of these muscle problems (63.1 %) were related with thigh strains (Engebretsen et al. 2013). In an epidemiological study among elite soccer players, it was reported that almost one third of all injuries in professional soccer are muscle injuries and a player sustained 0.6 muscle injuries per season. Each season, 37 % of soccer players missed training or match play due to muscle injuries. This means an average of 90 days and 15 matches missing per club per season from hamstring injuries alone (Ekstrand et al. 2011). Besides, 92 % of all muscle injuries affected the four major muscle groups of the lower limbs: hamstrings (37 %), adductors (23 %), quadriceps (19 %), and calf muscles (13 %). By the day the muscle injury rate increases progressively probably due to higher physical demands and increase of fixture congestion especially in team sports, enabling players' short recovery time (Bengtsson et al. 2013).

Etiology and Risk Factors

Although muscle injuries occur commonly during sportive activities, our understanding of the pathophysiology and the factors that predispose athletes to muscle injury is still limited. To establish effective prevention programs, it is important to identify risk factors associated with the occurrence of injury. It should be noted that almost all muscle injuries occurred in noncontact situations and only 5 % occurred during foul play. Several intrinsic risk factors were identified for muscle injuries such as previous injury, older age, poor flexibility, decreased muscle strength or strength imbalances, increased muscle stiffness, failure to stretch, and inadequate of warm-up (Table 1) (de Visser et al. 2012; Freckleton and Pizzari 2013; Hägglund et al. 2013). The strongest risk factor of muscle injury is a recent history of that same injury, and the next strongest risk factor is a past history of the same injury. It was introduced that previously injured soccer players have more than twice as high a risk of sustaining a new hamstring injury (Engebretsen et al. 2010). Furthermore, previous history of one type of muscle strain was shown to increase the risk for certain types of other muscle strains (Orchard 2001). These repetitive injuries cause significantly longer absences than do the first injuries. Muscle fatigue may be also related with the etiology, since it was found that muscle injuries occur more frequently toward the end of matches and increased fixture congestion affects the rate of muscle injuries. The injury incidence is usually higher during match play compared with training. Moreover, limb dominance, playing position for team sports may be related with injury rate. Quadriceps muscle injuries are significantly more common in the dominant kicking leg, whereas other muscle group injuries of lower leg expose no difference in frequency for the dominant and nondominant legs.

There is limited information about the genetic predisposition on muscle injuries. The relationship between single nucleotide polymorphisms in the following genes, IGF2, CCL2, and COL5A1, and

Table 1 Risk factors for muscle injuries

Certain risk factors for muscle injuries	Possible risk factors for muscle injuries
Previous injury	Type of sports
Older age	Artificial turf
Poor flexibility	Limb dominance
Decreased muscle strength or strength imbalances	Playing position
Increased muscle stiffness	Genetic predisposition
Muscle fatigue	
Failure to stretch	
Inadequate of warm-up	
Competitive activities	
Doping abuse or using anabolic agents	

muscle injury risk has been introduced (Ricard et al. 2013). Future studies will prove us to predict each athlete's injurability according to their genetic profiles based on the single nucleotide polymorphisms. From a biological perspective, it seems reasonable to suggest that explosive athletes with a dominant fast muscle fiber type would be more prone to sustain strain injuries (Engebretsen et al. 2010). The effects of increased body mass index and weight and height on muscle injury are still controversial and require further investigations. Doping abuse or using anabolic agents were shown to be related with spontaneous rupture at the musculotendinous junction and within the muscle belly, since they cause dysplasia of collagen fibrils in musculotendinous junction (Nikolopoulos et al. 2011).

In the context of muscle injuries, intrinsic factors related to individual features seem more predictive of injury than extrinsic ones, which are principally environment related. Recently numerous studies have attributed a greater risk and incidence of sport injury playing on artificial turf when compared to natural grass, but it is still not clear if new generation artificial turfs predispose for the risk of muscle injury (Dragoo et al. 2013).

Classification

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 a great interest in optimizing the diagnostic, therapeutic, and rehabilitation process after muscle injuries, to minimize the absence from sport and to reduce recurrence rates. Clinical assessment, site of injury, and pattern of the lesion can all provide prognostic information regarding convalescence and recovery time following both an acute and recurrent muscle injuries. However, little information is available in the literature about muscle injury definitions and classification systems. Various classification systems have been published in the literature to date, but there is no consensus within studies and in daily practice for grading muscle injuries. It is also crucial to provide a common and standardized terminology as well as a comprehensive classification system for athletic muscle injuries in order to improve communication among sports physicians and obtain comparability.

The current classification system of muscle injuries were most widely utilized according to severity of injury and defined muscle injuries as mild, moderate, and severe injuries (Peetrons 2002).

- Grade 1 injury: Edema, but no architectural distortion, focal fiber rupture less than 5 % of the muscle involved
- Grade 2 injury: Architectural disruption indicating partial tear with more than 5 % of the muscle involved
- Grade 3 injury: Complete muscle rupture/tear with retraction of the muscle ends

Recently two studies have been published for novel classification systems about muscle injuries (Chan et al. 2012; Mueller-Wohlfahrt et al. 2013). Chan et al. proposed an imaging nomenclature, which considers the anatomical site, pattern, and severity of the lesion in the acute stage of muscle strain injuries. With regard to the site of injury, they classified muscular injuries as proximal (A), middle (B), and distal (C). Moreover, they differentiated involved muscular structures as intramuscular (a), myofascial (b), myofascial/perifascial (c), musculotendinous (d), and combined injuries (e).

Meanwhile, Munich muscle injury classification system was introduced as a new terminology and classification system of muscle injuries to present a more precise definition of the English muscle injury terminology and to facilitate diagnostic, therapeutic, as well as scientific communication. This clinical comprehensive system classifies muscle injuries into functional and structural–mechanical injuries, where ***functional muscle disorders*** are fatigue-induced or neurogenic injuries causing muscle dysfunction without macroscopic evidence of fiber tear, while ***structural–mechanical injuries*** are tears of muscle fibers with macroscopic evidence of fiber tear, that is, structural damage (Mueller-Wohlfahrt et al. 2013). Previously the term ***muscle strain*** was defined as acute distraction injury of muscles

and tendons. However, Munich muscle injury classification system did not recommend using the term "strain" anymore, since strain is a biomechanical term, which is not defined and used indiscriminately for anatomically and functionally different muscle injuries. Instead, they proposed to use the term tear for structural injuries of muscle fibers/bundles, leading to loss of continuity and contractile properties.

Muscle Strains

The majority of muscle injuries (more than 90 %) are caused either by contusion or by excessive strain of the muscle. Musculotendinous strains and tears are primarily created by a single traumatic event from excessive stretching on musculotendinous fiber (e.g., in high-speed runners), from movements involving excessive range over sequential joints (e.g., in dancers), or as a result of explosive eccentric contractions (e.g., in football players). Muscle strains, very common in sports that require sprinting or jumping, usually arise from an indirect insult, from application of excessive tensile forces. Muscles that are frequently involved in injuries are often biarticular that extend across two joints or are those with a more complex architecture (e.g., adductor longus), undergo eccentric contraction, and contain primarily fast-twitch type 2 muscle fibers and fusiform shape (Fig. 1). Overall, strains and complete tears are usually located at the muscle–tendon junction, since these areas present biomechanical weak points. The hamstrings, quadriceps, and calf muscles, in that order, are the most commonly strained muscles (Järvinen et al. 2000).

The diagnosis is easy when a typical history of muscle contusion or strain is accompanied by objective evidence of swelling and/or ecchymosis distal to the lesion. Ecchymosis may not develop until 24 h after the injury (Kary 2010) (Fig. 2a). Pain is typically felt by the patient with resisted muscle activation, passive stretching, and direct palpation over the muscle strain. Although the diagnosis is usually clinical, imaging tools are often advocated to better understand extent and site of lesion, the relevant prognostic factors predictive of recovery time, return to pre-injury sport activity, and risk of recurrence. Ultrasonography has traditionally been considered the method of choice for clinical diagnosis of muscle injuries, as it is relatively inexpensive in nature (Aydoğ et al. 2008; Tok et al. 2012). However, it has the clear disadvantage of being highly dependent on the experience of the radiologist. Ultrasonography has the ability to image the muscles dynamically and assess for bleeding and hematoma formation via Doppler. Magnetic resonance imaging, in turn, should be preferred if a clear discrepancy exists between the patient's symptoms, the physician's findings, and/or the ultrasonography, particularly

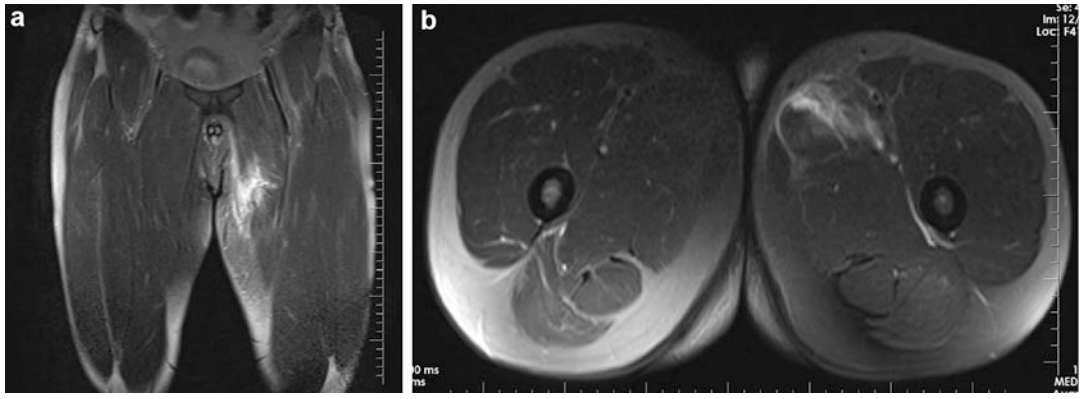

Fig. 1 MR images of an amateur soccer player with acute groin pain. (**a**) T2-weighted coronal image showing left adductor longus muscle strain with mild feathery edema along the intramuscular myotendinous junction. **(b)** Edema of myotendinous strain of adductor longus muscle in axial T2-weighted image

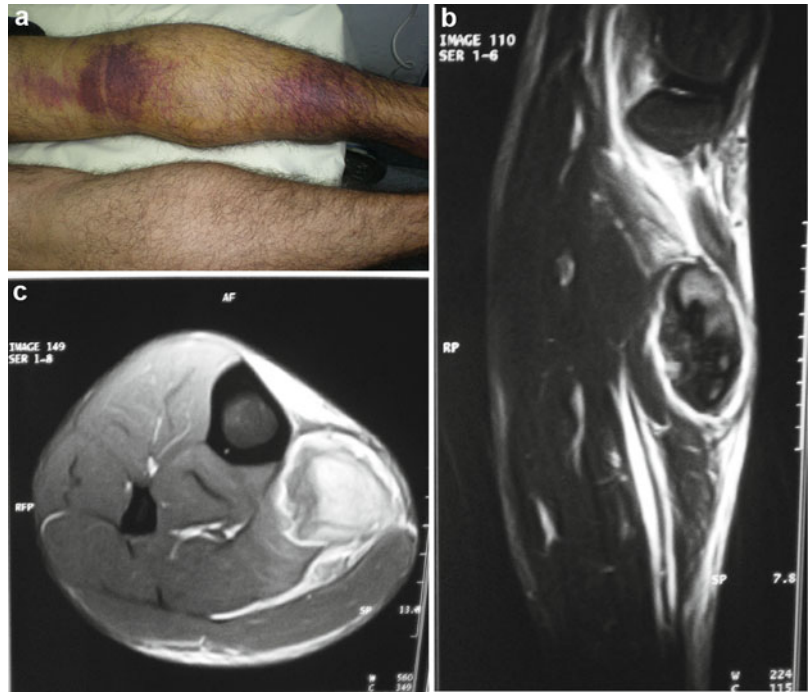

Fig. 2 Tennis leg of an athlete. **(a)** Posterior view of the leg with widespread ecchymosis. **(b)** Intramuscular hematoma formation of medial gastrocnemius muscle in PD-weighted fat saturation coronal view. **(c)** Axial view of the same lesion with effusion around

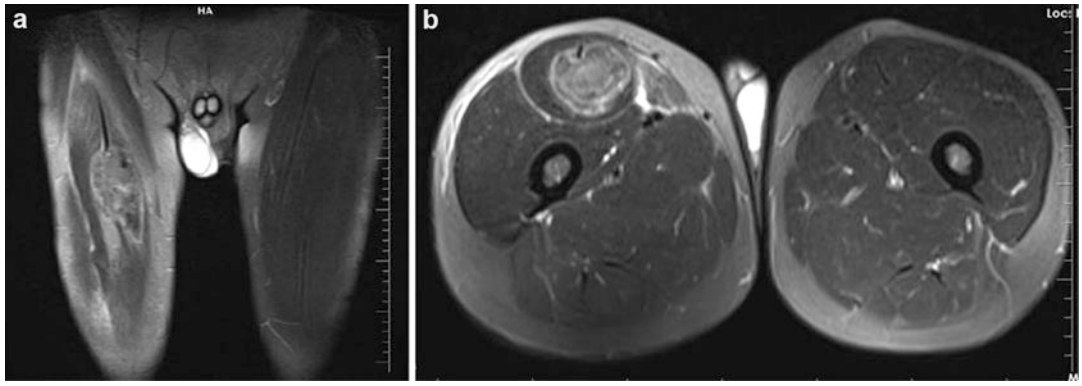

Fig. 3 Coronal and axial PD-weighted images reveal subacute rectus femoris muscle tear and intramuscular smooth-surfaced organized hematoma

in injuries near and/or at the groin area or close to the MTJ, where MRI has shown its superiority over ultrasonography (Järvinen et al. 2005). Recently discrepancy between clinical and radiological classification was introduced in thigh muscle injuries, which demonstrates that a negative MRI does not rule out clinically relevant muscle injury and that clinical diagnosis and management should be based on a combination of clinical history, physical examination, and possibly radiographic evaluation. Among injuries classified both clinically and radiographically, 77 % were clinically classified as structural tears, but radiological grading on MRI showed evidence of muscle tears in only 29 % of injuries (Ekstrand et al. 2013).

Moderate or severe muscle strains often result in a large hematoma, because the intramuscular blood vessels are torn (Fig. 3). In the injured muscle, two different types of hematomas, intramuscular

and intermuscular hematomas, can be identified (Järvinen et al. 2000). In intramuscular hematomas, the extravasation of blood increases the intramuscular pressure, which compresses and finally limits the size of hematoma. Increased intramuscular pressure may result with compartment syndrome as one severe complication of muscle injury. However, intermuscular hematomas develop if the muscle fascia is ruptured and the extravasated blood spreads into the intermuscular spaces without a significant increase in the pressure within the muscle.

It is often difficult to predict both short-term outcome and long-term prognosis following a muscle strain, although these injuries may have a significant impact on the athletes and their teams. It was indicated that the prognosis significantly depends on both the site and the size (cross-sectional area and the length independently predictive) of the muscle strain injury. Of all the common sports injuries, muscle strains have one of the highest recurrence rates (incidence of reinjury) after return to play. Sometimes, a muscle strain that is treated improperly could become a chronic and career-threatening condition. The recurrence rate for hamstring strains (the most common muscle strain) is around 12 % in professional soccer (Woods et al. 2004) and around 30 % (cumulative recurrence rate for the remainder of the season) in professional Australian football despite concentrated rehabilitation and prevention efforts (Orchard et al. 2005).

Delayed Onset Muscle Soreness

Most people experience muscle soreness following unaccustomed physical exertion. Delayed onset muscle soreness (DOMS) is a common myogenic condition with main symptoms of pain, tenderness, and loss of range of movement that usually peaks approximately 24–48 h after intensive activity and gradually subsides in 5–7 days. Associated symptoms include muscle shortening, increased passive stiffness, swelling, decreases in strength and power, localized soreness, and disturbed proprioception. DOMS is most prevalent at the beginning of the sporting season when athletes are returning to training following a period of reduced activity, during the progression from low-intensity to high-intensity training due to periodization, or when unaccustomed or novel types of eccentric exercise are introduced such as downhill running, plyometrics, and resistance training. DOMS is also common when athletes are first introduced to certain types of activities regardless of the time of year.

Several causative factors were suggested for DOMS including lactic acid, muscle spasm, connective tissue damage, muscle damage, inflammation, enzyme efflux, and free radicals. DOMS is often attributed to eccentric muscle contractions known to more severely induce damage of the muscle cell membrane and microinjury of the muscle fibers than static and concentric contractions. This damage is characterized by decreased muscle force production, increased serum creatine kinase activity, ultrastructural disruption, inflammation, and increased proteolytic activity (Lewis et al. 2012).

Many interventions have been used in an attempt to prevent DOMS such as massage, stretching, or low-intensity exercise administered before or immediately after strenuous activity. If massage is rendered during the early stages of inflammation, the mechanical pressure applied with the massage might decrease neutrophil margination, thereby reducing inflammation and DOMS. Aerobic warm-up prior to resistance exercise prevents muscle soreness at the central muscle belly. However, cooldown exercises may be less effective than the warm-up in attenuating muscle soreness for the first 24 h after resistance exercise. Whole-body vibration training before eccentric exercise may protect the muscle fibers against further damage and alleviate the symptoms of DOMS. Eccentric exercises or novel activities should be introduced progressively over a period of 1 or 2 weeks at the beginning of, or during, the sporting season in order to reduce the level of physical impairment and/or training disruption.

Treatment of DOMS currently includes the use of anti-inflammatory analgesics, massage

techniques, cold or warm whirlpools, contrast baths, cryotherapy, transcutaneous electrical nerve stimulation (TENS), vibration training, ultrasound, and laser. Tender point acupuncture may be an effective alternative option to relieve muscle pain of DOMS. Massage administered 48–72 h after unaccustomed physical exertion appears to reduce DOMS, but few well-designed studies exist. Recently it was concluded that stretching neither prevents nor relieves exercise-induced DOMS (Engebretsen et al. 2010).

Muscle Hernias

Muscle herniations of athletes especially in lower legs are not rare entity in sports medicine practice, however commonly neglected by the physicians. Herniation of muscle occurs through defects in the deep fascial layer of the lower extremity, particularly over the anterior tibial compartment. Most hernias occur in the lower leg and tibialis anterior is the most likely muscle of the lower leg to be herniated. The posterior tibial compartment, peroneal compartment, and quadriceps fascia are other possible sites. These defects typically occur following local blunt trauma or muscle hypertrophy so muscle hernias have been classified into traumatic and constitutional (Gupta et al. 2008). Therefore, elite athletes tend to face with a muscle hernia during their careers more than any individual.

Muscle hernias are usually asymptomatic, although they may present with cramping or pain after prolonged activity. Patients may present with intermittent painful swelling that becomes more prominent with muscle contraction. The diagnosis of a muscle hernia is generally a clinical one, based on symptoms and physical examination. A small superficial bump may be noted with the limb at rest, which will become more prominent with contraction of the associated muscle. Typically, a palpable soft-tissue mass is found and the fascial defect can often be palpated. Radiologic imaging techniques, including MRI, CT, and USG, have been used to identify the fascial defect and confirm the diagnosis. The sonographic features of muscle hernias are characteristic and exclude alternative clinical diagnoses such as tumors and muscle tears (Fig. 4). Advantages of sonography include the ability to examine the patient dynamically or erect and to show the nature of the lesion to the patient during the examination (Beggs 2003). The differential

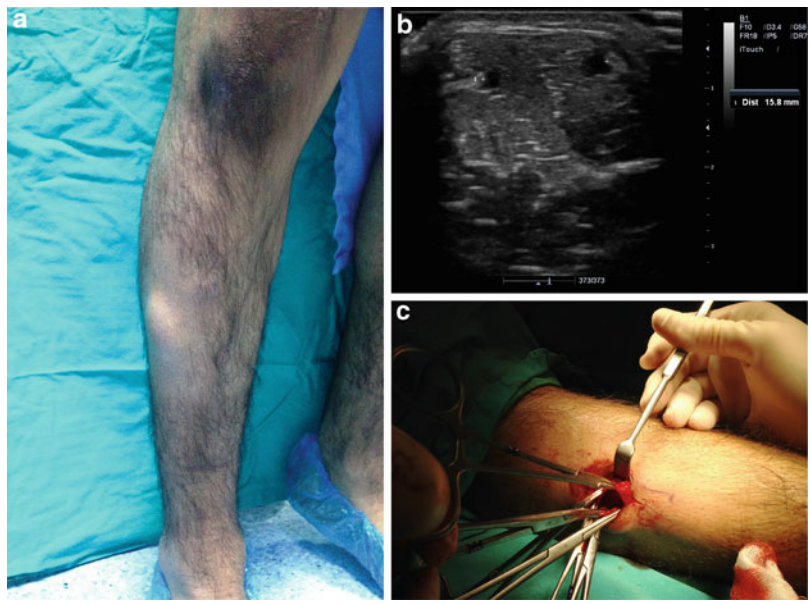

Fig. 4 Tibialis anterior muscle hernia. **(a)** Gross appearance of the protruding mass on the anterolateral aspect of the right leg. **(b)** Longitudinal ultrasonography showing the bulging of tibialis anterior muscle. **(c)** Fascial defect easily seen during surgery

diagnosis includes varicosities, lipomas, angiolipomas, hematomas, thrombophlebitis, fibromas, varices, neurilemmoma, arteriovenous aneurysms, and soft-tissue sarcoma.

Treatment depends on the symptomatology of the herniation and varies from conservative therapy to surgical repair. Many patients with muscle hernias are asymptomatic and may not seek treatment or may seek medical care out of concern for appearance or to rule out an underlying neoplastic process. For symptomatic muscle herniation, nonoperative treatment options include activity modification, compression stockings, physical therapy, deep tissue massage, and anti-inflammatory medications. For patients with moderate to severe symptoms or those in whom conservative treatment has failed to alleviate symptoms, surgical methods can be considered. Available surgical procedures were reported as direct repair, fascial grafting, fasciotomy, and more recently mesh grafting. Direct repair is possible when the defect is small and the laxity of the borders permits approximation. Patients with a postsurgical muscle herniation may have a worse clinical outcome in terms of lower satisfaction with surgery, delayed or incomplete return to sports, and more significant residual symptoms after fasciotomy (Kramer et al. 2013). Cosmetic reasons usually are not an indication for surgery, because complications can be serious.

Muscle Contusions

Muscle contusion injury is a frequent and debilitating condition in athletic competitions and traditionally seen in contact sports. Muscle contusions result from direct trauma, usually caused by the opponent player. Contusion injuries are frequent in contact or combat sports as a result of large compressive forces applied directly on the muscle. It is more likely to occur during games than during practice and more frequently seen in male athletes than in female athletes (Trojian 2013).

Unlike strains, these traumas usually occur deep in the muscle belly and tend to be less symptomatic than strains. The injury consists of a well-defined sequence of events involving microscopic rupture and damage to muscle cells, macroscopic defects in muscle bellies, infiltrative bleeding, and inflammation. Bleeding deep within the involved muscle group causes swelling, pain, and stiffness (Beiner and Jokl 2002).

Treatment of muscle contusions is usually nonoperative and in a staged manner. Initial treatment should start with rest, ice, and compression for the first 24–48 h to limit the hemorrhage and control the size of the secondary area of injury. Rest with the knee in a flexed position helps to limit muscle stiffness especially in quadriceps muscle. Placing and holding the knee in 120° of flexion immediately following a quadriceps contusion appears to shorten the time to return to unrestricted full athletic activities. In these cases, it is important to limit the hematoma by controlling bleeding through maintaining knee flexion. Measurement of knee flexion has been used as a prognostic indicator in quadriceps contusions. The patient will need to use crutches and should maintain this position of knee flexion for 24 h. After 24 h, the brace or wrap should be removed, and gentle, active, pain-free range of motion at the knee should be instituted along with stretching and isometric quadriceps strengthening. The active phase of treatment, including functional rehabilitation, can begin when pain-free, active knee flexion of at least 120° is attained. Physical therapy must be applied as pain permits and then functional rehabilitation program should be planned specific for the patient. Cryotherapy should be used with continuation of NSAIDs until 48–72 h. Experimental and theoretical evidences support that therapeutic ultrasound is clinically effective on treatment of muscle contusion injuries.

Athlete may be allowed for return to sport when pain-free full range of motion, equal muscle size with uninjured side, and muscle strength is achieved. Most of the muscle contusion injuries recover uneventfully within a few weeks. Improved protective equipment seems to be decreasing the frequency and severity of this injury in many sports; however, there is no research to support this apparent trend. In sports without padding for the thigh and upper leg, such as soccer and rugby, muscle contusions continue to be a major disabling injury (Kary 2010).

A more severe contusion can result in a palpable, hard mass in the involved muscle, indicating the presence of myositis ossificans. Although it is not necessary to measure compartment pressures for all cases unless there is suspicion of a complicating condition such as a fracture or injury to a large blood vessel, compartment syndrome should be always kept in mind and patient should be checked frequently for symptoms and signs of compartment syndrome.

Compartment Syndrome

Compartment syndrome occurs when increased intramuscular pressure (fluid hydrostatic pressure in the interstitial space of a skeletal muscle tissue) impedes local muscle blood flow, thereby impairing neuromuscular function of tissues within the specific compartment. Chronic, acute-on-chronic, and rarely acute compartment syndrome has been recognized in athletes and soldiers who undergo rigorous training, which is due to an increase in volume of muscle in a tight fascial compartment or repeated trauma.

Acute compartment syndrome is a rare entity and the causal factor is usually a blow/direct trauma to the thigh (with or without fracture) or acute muscle overuse. Such trauma increases the amount of extravascular fluid, via either edema or blood, and compresses the perfusing capillaries leading to tissue ischemia. This is an emergency condition and prompt recognition in this syndrome is imperative to prevent irreversible ischemic insult.

Chronic compartment syndrome has been described in athletes who undergo rigorous training regimes presenting with pain induced by exercise, swelling, and impaired muscle function. It is usually bilateral and can arise from insufficient compliance of the surrounding fascia or exercise-induced muscle hypertrophy, which can increase the muscle volume by 20 % within the confined nonelastic space of the compartment (Aweid et al. 2012). Physiopathologically, elevated intracompartmental pressure impedes blood flow, leading to ischemia and temporary neurological deficits.

Although compartment syndrome can exist wherever a compartment is present (thigh, forearm, upper arm, foot, hand, lumbar spine, abdomen, buttock), the anterior and lateral compartments of the lower leg are most commonly affected (Schubert 2011). In many cases, the diagnosis can be made from clinical symptoms and signs alone. The main features are weakness and severe pain on passive stretching of the muscles of involved compartment, paresthesias in the distribution of the nerves running through the compartment, and tenseness of the fascial envelope surrounding the compartment. Other diagnostic findings are swelling, induration of the involved compartment, and increased extremity circumference. Diminished pulses and paresis within the extremity are late findings and signify significant tissue ischemia and cell necrosis. Ultrasound and MR imaging are both possible tools to identify either circumscribed fluid collections, such as hematoma, or diffuse bleeding within a muscle.

The diagnosis is usually confirmed by intracompartmental pressure measurements. At present, no consensus has been reached on the optimal regimen and optimal moment to measure pressures, a large range of compartmental pressure values are considered pathological for compartment syndrome, and normal ranges are still debated. Normal resting intracompartmental pressures range between 0 and 8 mmHg, and capillary perfusion deficits are noted with compartment pressures exceeding 30 mmHg. Muscle necrosis occurs with 4 h of compartment pressures greater than 50 mmHg, and 6–12 h of such increased pressures yields irreversible nerve damage and functional muscular deficits. Elevated white blood cell count, temperature, and creatine kinase were acute indicators of ongoing tissue ischemia and subsequent muscle necrosis.

The most commonly recognized threshold for surgical decompression is a recorded compartment pressure within 30 mmHg of diastolic blood pressure. Fasciotomy is suggested in cases of compartment pressures exceeding 40 mmHg or in patients with above symptoms and signs worsen or do not resolve. Fasciotomy is generally a safe and effective treatment for compartment

syndrome with success rates greater than 90 % in patients unresponsive to conservative measures and complaining of progression of symptoms and paresthesia. Complications following compartment release exist and include hemorrhage, hematoma, deep vein thrombosis, wound infection, ulceration, skin breakdown, nerve entrapment or injury, muscle herniation, swelling, vascular injury, residual weakness, and lymphocele. Incidence of complications ranges from 4.5 % to 13 %; recurrence of symptoms occurs in 7–17 % of surgical cases (Schubert 2011).

Myositis Ossificans

Posttraumatic myositis ossificans (MO) also known as posttraumatic heterotopic ossification, nonhereditary heterotopic ossification, and myositis ossificans circumscripta is defined as nonneoplastic proliferation of bone and cartilage tissue within a muscle after the formation of an intramuscular hematoma. It is encountered most frequently in the sporting community as a complication of muscle contusions and strains by either a major trauma or repeated injury (overuse). The incidence of myositis ossificans after muscle strains and contusions has not been well documented but has been reported to be 9–17 % (Beiner and Jokl 2002) and highest in the high-contact sports in which the use of protective devices is uncommon (e.g., rugby). The incidence also is thought to be related to the severity of injury.

Any muscle can be affected, but the most frequent locations are the anterolateral aspect of the thigh and upper arm. The mechanism of MO has not been clearly established. It is believed to be formed after prolonged macrophage invasion of myonectoric tissue with osteogenic mediator release after traumatic injury to muscle. Trauma to the muscle triggers proliferative repair with activation of the perimysial tissue-inducible osteoprogenitor cells.

Clinically, myositis ossificans should be suspected if the pain and swelling after a traumatic injury do not respond to conservative treatment within 10–14 days after initial trauma, and patients begin to demonstrate symptoms including decreased range of motion, local tenderness, stiffness, erythema, soft-tissue swelling, and a palpable mass 2–3 weeks after the trauma.

The diagnosis is confirmed by the characteristic radiologic finding of "zoning," which describes a distinct peripheral margin of mature ossification and a radiolucent center of immature osteoid and primitive mesenchymal tissue. These findings help to distinguish this benign disorder from osteosarcoma in which the central bone is mature. The radiologic features of myositis ossificans evolve as the lesion matures with evidence of ectopic bone formation being detected 3–4 weeks after the initial trauma. At about 10 weeks onward, radiographs will show a zoning pattern of peripheral maturation, producing an "eggshell" appearance. At approximately 4–6 weeks, CT demonstrates a rim of calcification around the lesion and a central core with decreased attenuation.

Acute care of a severely injured muscle during the initial two weeks aims to reduce local inflammation, which is hypothesized to reduce the chance of developing MO. During the first 15 days after trauma, the lesions are vulnerable to further trauma, and therefore, excessive activity, forceful stretching, and massage should be avoided to limit bleeding. If MO is thought to be present, then even greater caution needs to be taken to make certain that the injured muscle does not worsen by the rehabilitation program.

Treatment includes pain control and physical therapy with weight bearing as tolerated and passive range of motion (ROM) exercises once the patient is pain-free at rest. Rehabilitation of patients with MO should include restoration of flexibility, strength, ROM, and proprioception with special attention to avoid maladaptive compensation patterns during the rehabilitation phase and on return to sport. Therapy has been largely based on the RICE principle of rest, ice, compression and elevation, and non-painful, passive stretching and strengthening routine. NSAIDs (indomethacin) prophylaxis have been shown to be effective for HO after spinal cord injury and joint replacement and may be advised for trauma-induced MO; however, it has not been validated

for the prevention and/or treatment of myositis ossificans yet. Researches for medical treatment with local and oral magnesium therapy, injections of xylocaine and dexamethasone, bisphosphonates, calcitonin, and warfarin are still needed to be confirmed. Low-dose radiotherapy can be helpful in impeding ossification, although the strict evidence for that is lacking. A recent study has demonstrated that ESWT may be a promising treatment modality for the management of pain and loss of ROM that result from MO (Torrance and Degraauw 2011). Complete resolution of symptoms may require up to 2 years. In most cases, spontaneous resorption is the rule, and therefore surgical excision of the lesion is not necessary. Surgical excision may be warranted in rare cases, where there is persistent pain, limitation of joint movement, or neurological involvement. Surgery is reserved until approximately 12–14 months post-injury, when the lesion becomes stable and the periosteum has formed. It is universally accepted that surgical removal should be delayed until the bone has fully matured and no longer shows increased uptake on a bone scan. Surgery is contraindicated for immature lesions due to the high recurrence rates.

Muscle Ruptures

Comparing with subtotal tears or tendon avulsions, complete muscle ruptures, with a discontinuity of the whole muscle, are very rare. The most frequent type of muscle tears occurs near the muscle–tendon junction (e.g., of the intramuscular tendon of the rectus femoris muscle). Muscle ruptures may be caused by active contraction of the muscle, contraction of an antagonist, increase of tearing over cohesive power, asynchronous contraction, or additional muscle force provided by another muscle. However, the most common cause of muscle rupture is an overload of an actively contracting muscle group due to the application of a resisting load or external force that exceeds tissue tolerance (Krogsgaard et al. 2003). The injury usually results in tearing of muscle fiber and the muscle sheath as well as a palpable defect in the muscle. The healing of the ruptured muscle is slow and fibrosis, degeneration, and inadequate functional recovery may be developed.

Complete musculotendinous rupture is commonly accompanied by a hematoma. The diagnosis is usually made on clinical grounds, i.e., complete loss of muscle function, with palpable gap and muscle fiber retraction (Fig. 5). Extensive acute edema and hemorrhage may limit accurate evaluation of the injured muscle. Immediate repair of ruptured muscle is a common and accepted treatment strategy to reestablish and improve the function. Clinical experiences showed that injuries involving more than 50 % of the muscle diameter (subtotal tears) usually have a similar healing time compared with complete tears (Trojian 2013). If the tears are left untreated, the ends may become rounded and tether to adjacent muscles or fascia. Minimally invasive repair of the muscle ruptures may decrease the risk of myositis ossificans (Kaya et al. 2013).

The frequency of acute hamstring tears appears to be increasing, both clinically and by reports in the literature, as middle-aged patients continue to be physically active and as the recognition and potential for treatment has become better established. Surgical repair was indicated in young (aged <50 years), active individuals, especially those still participating in athletics who have retraction of the ruptured tendons of ≥ 2 cm. Surgeons are more likely to recommend repair of proximal hamstring-origin complete avulsions because of the risk for tendon retraction and progressive functional impairment with time, whereas the majority of hamstring muscle injuries respond well to nonoperative treatment. Surgical treatment also reported as beneficial in selected cases of distal hamstring tears (Lempainen et al. 2007).

Rectus femoris muscle tears may appear as a discrete soft-tissue mass in the anterior thigh. The mechanism of injury is usually a forceful contraction of the quadriceps against excessive resistance, such as landing from a jump or forcibly kicking the ball. The rectus femoris muscle is at increased risk of injury because these strains occur more frequently in muscles undergoing eccentric

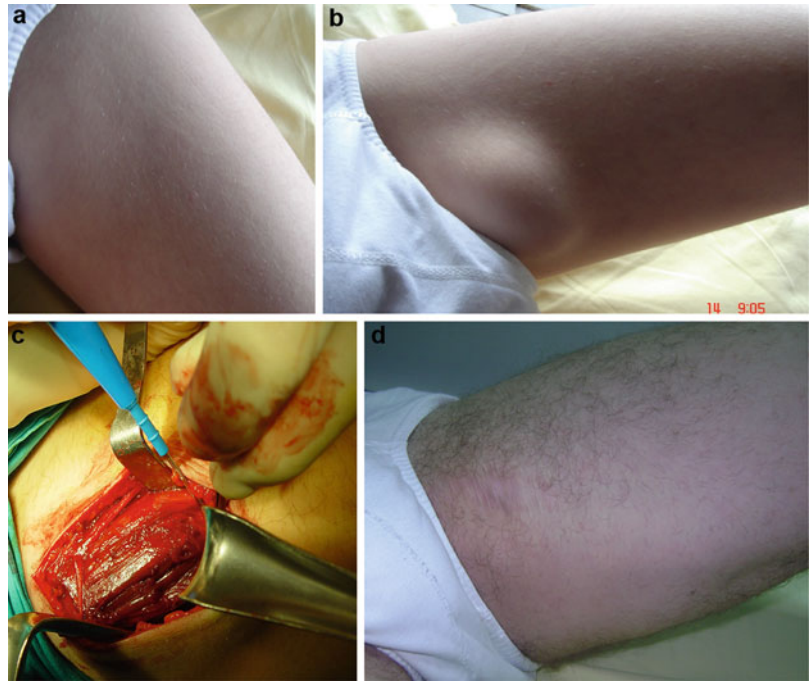

Fig. 5 Complete rupture of adductor magnus muscle. (**a**) When the patient was examined in the prone position, the left adductor muscles group had a normal appearance compared to the opposite side. **(b)** The defect was clearly visible as a soft-tissue mass when the patient contracts the adductor group of muscles, retraction. **(c)** Surgical repair of ruptured part of adductor magnus muscle at the myotendinous level. **(d)** Symptomatic relief and no visible soft-tissue mass after 18 months (All figures are used with the permission of senior surgeon Mahmut Nedim Doral MD)

contraction and in muscle groups functioning over two joints. The quadriceps muscle is composed largely of type II fibers used in rapid forceful activity, which predisposes this muscle to repeated strains and tears in young, active people. In cases of avulsion of the anterior inferior iliac spine, it clearly appeared that orthopedic treatment was successful by rest, initial immobilization and subsequent rehabilitation, reserving surgery for cases with large initial retraction of the bone fragment, or patients for whom nonoperative treatment had not been successful. Recently, surgical treatment was indicated in professional athletes for a complete functional recovery with lower recurrence rate (García et al. 2012).

Rupture of the distal musculotendinous junction of the medial head of the gastrocnemius is also called “tennis leg” (Fig. 2b, c). The classic clinical manifestation is that of a middle-aged, poorly conditioned patient who complains of acute sports-related pain in the middle portion of the calf that is associated with a snapping sensation. Tennis leg typically occurs in connection with plantar flexion of the foot with simultaneous extension of the knee, which implies active contraction and passive stretching of the gastrocnemius muscle. A sudden pain is felt in the calf, and the patients often report an audible or palpable “pop” in the medial aspect of the posterior calf, or they have a feeling as though someone has kicked the back of their leg. The treatment of this condition is usually conservative with elevation of the lower extremity, application of ice, and use of supportive elastic dressings. Surgical treatment (fasciotomy) is indicated only when an associated compartmental syndrome has complicated the evolution of the signs and symptoms.

Acute avulsion injuries result from extreme, unbalanced and often eccentric muscular contractions, and patients with such injuries present with severe pain and loss of function. Adolescents are particularly vulnerable to avulsion injuries because of the inherent weakness of the apophyses (Doral et al. 2005). Apophysitis describes a chronic traction injury at the tendon insertion site. The many apophyses in the pelvis and hip are common sites of avulsion injuries and the single most common site is at the ischial tuberosity. Cheerleaders, sprinters, gymnasts, track athletes, American football players, and baseball players are commonly affected. Most avulsion injuries are

generally treated by conservative means and the prognosis is good, but nonunion may occur.

Treatment

Treatment of a muscle injury should be focused on to return the player to training and matches as soon as possible with lower recurrence risk. When muscle is acutely damaged by strain, contusion, or laceration, there is bleeding and hematoma formation among the ruptured muscles cells followed by an inflammatory reaction. Clinically, the first aid of muscle injuries follows the RICE principle (rest, ice, compression, and elevation) initially to minimize intramuscular bleeding and thereby limit the progression of the muscle injury. Ice or cold application is thought to lower intramuscular temperature and decrease blood flow to the injured area. It has been shown that the early use of cryotherapy has beneficial effects; it is followed by a significantly smaller hematoma between ruptured myofiber stumps, less inflammation and tissue necrosis, and a slight accelerated early regeneration response. Compression may help decrease blood flow and accompanied by elevation will serve to decrease both blood flow and excess interstitial fluid accumulation. The elevation of the injured extremity above the level of the heart to decrease capillary hydrostatic pressure and thereby promote resorption of extravasated fluid is recommended. Thereby, this approach may limit the size of the hematoma via decreasing blood flow to the injury site and increasing venous return.

During the first few days after the injury, a short period of immobilization allows the formation of granulation tissue at the site of injury, but it should be noted that the duration of reduced activity (immobilization) should be limited only until the scar reaches required tensile strength to withstand the contraction-induced forces applied on it without a rerupture. At this point, gradual mobilization should be started to optimize the healing by restoring the strength of the injured muscle and preventing muscle atrophy, loss of strength, and extensibility (Järvinen et al. 2005). It has been shown that early mobilization induces more rapid and intensive capillary ingrowth into the injured area as well as better muscle fiber regeneration and more parallel orientation of the regenerating myofibers in comparison to immobilization. However, mobilization started immediately after the injury often causes reruptures at the original injury site. Therefore, the immobilization period should be kept as short as possible, i.e., only the first few days immediately after the injury. The optimal length of immobilization depends on the grade of the injury and should not be longer than needed for the scar to bear the pulling forces without rerupture.

Nonsteroidal anti-inflammatory drugs (NSAIDs) can be useful for reducing pain and allowing earlier return to activity. NSAIDs appear to have a paradoxic effect on the healing of muscle injuries, with early signs of improvement and subsequent late impairment in functional capacity and histology. NSAID use in the first 3 days appears not to have a detrimental effect, but continued use seems to be detrimental to the regenerating skeletal muscle (Trojian 2013). Besides, the use of corticosteroids is definitely contraindicated based on research demonstrating delayed healing and reduced biomechanical strength of injured muscle.

The acute phase of treatment is subsequently followed by an active phase of management including carefully structured rehabilitation program, stretching, strengthening, range of motion, maintenance of aerobic fitness, proprioceptive exercises, and functional training. Stretching should be done carefully and always to the point of discomfort, but not pain. Ballistic stretching is discouraged due to the risk of re-tearing muscle fibers. Strengthening exercises should be performed through a pain-free range of motion and should progress sequentially through isometric, isotonic, isokinetic, and functional exercises (Kary 2010). It is of particular importance to note that all physical rehabilitation activities should start with warming-up of the injured muscle to minimize the risk of reruptures, which reduces muscle viscoelasticity and relaxes muscles neutrally (Järvinen et al. 2005).

If the symptoms caused by the injured muscle fail to improve 3–5 days after the trauma, this is

the stage at which it is necessary to reconsider the existence of an intramuscular hematoma or extensive tissue damage that might require special attention. The imaging modalities (ultrasonography or especially MRI) are highly recommended under these circumstances. The puncture and aspiration of the injured area (if fluctuation is present) are among the procedures that are sometimes required.

Therapeutic ultrasound is widely recommended and used in the treatment of muscle strains, although no clinical evidence exists on its effectiveness and experimental studies are not encouraging. Hyperbaric oxygen therapy has been proposed as another fascinating possible option to improve the regeneration of the injured skeletal muscle during the early phase of the repair. The therapeutic use of growth factors and gene therapy, alone or in combination, and the application of stem cell-based therapies provide the latest, most promising, but currently also the most poorly validated therapeutic options for the enhancement of the healing of an injured skeletal muscle. In recent years, injection therapies, such as Actovegin, Traumeel S, and growth factors, have gained popularity for the treatment of muscle injuries. Platelet-rich plasma (PRP) therapies may help athletes to hasten restoration of muscle function by promoting muscle regeneration after strain or contusions. Actovegin is a deproteinized hemodialysate obtained from filtered calf blood. It is suggested that it contains active components with muscle regenerating promoting effects (Reurink et al. 2012). Significantly shorter rehabilitation time for treatment of acute hamstring injuries with Actovegin compared with no injection therapy was reported. D Traumeel is a homeopathic formulation containing botanical and mineral components to which anti-inflammatory effects are ascribed. Despite the lack of evidence, the use of Traumeel S injections for the management of acute muscle strains and other inflammatory musculoskeletal conditions remains popular in many countries. The combined injection of Traumeel S and Actovegin immediately after a hamstring muscle injury and again at days 2 and 4 post-injury to the area of the muscle strain and the lumbar spine has been anecdotally reported to be common practice in sports medicine in Germany and is becoming increasingly popular among elite athletes elsewhere (Linklater et al. 2010). Local administration of autologous conditioned serum treatment (Orthokine therapy) has already been shown to enhance the healing of muscle injuries in animal models as well as in human athletes (Wehling et al. 2007). Suramin, a heparin analogue, is not part of the current standard treatment of muscle injuries in humans, but suramin-injected animals after 2 weeks of contusion injury showed increased strength with more regenerating myofibers and less fibrotic scar at 4 weeks. Further research is needed before it should be used in humans with muscle contusions.

Surgical interventions should be considered if an athlete has a large intramuscular hematoma(s), a complete (III degree) strain, or tear of a muscle with few or no agonist muscles, or a partial (II degree) strain if more than half of the muscle belly is torn (Järvinen et al. 2005). The surgical repair of the injured skeletal muscle is usually easier if the injury has taken place close to the MTJ, rather than in the middle of the muscle belly, because the fascia overlying the muscle is stronger at the proximity of the MTJ, enabling more exact anatomical reconstruction.

Return to Play

The first and the most common question of players, coaches, and even media after a muscle injury is when the player will return training back again. The time taken to walk pain-free after injury may be a simple prognostic indicator for clinicians. For instance, a player who takes longer than 24 h to achieve pain-free walking is four times more likely to need more than 3 weeks to return to competition compared with players who are able to walk pain-free within 24 h after a lower extremity muscle injury. Furthermore, MRI parameters of the longitudinal length and the cross-sectional area of the muscle injuries show good correlation with injury severity and

convalescence time. Several studies found that MRI negative hamstring strains have a significantly faster rehabilitation time compared with MRI positive strains and are less likely to recur. According to Munich muscle injury classification system, structural injuries are associated with longer average layoff times than functional muscle disorders (Mueller-Wohlfahrt et al. 2013).

There are no established consensus guidelines or agreed-upon criteria for safe return to sports following muscle strains that completely eliminate the risk for recurrence and maximize performance. Sport physicians use several criteria in order to assess player's ability to return to competitive soccer after a muscle injury. The decision to return a player to competition is a collaborative one and should be made by the sports medicine/sports science members and was based on the player successfully completing following tasks:

- Complete pain relief
- Normalized isokinetic muscle strength assessment
- Normalized flexibility equal to uninjured side
- Subjective feeling reported by the player
- Recovery in radiological findings (ultrasound or/and MRI)
- Achievement of sports-specific functional field tests

Strength deficit in injured muscle significantly cause greater risk of recurrence. Previously Heiser et al. suggested the players to begin jogging when the peak torque of hamstrings equaled 70 % of baseline level in isokinetic strength test. Players were allowed to return to play when peak torque reached a level of 95 % of the baseline score or a hamstrings-quadriceps ratio of 0.55 or greater. This protocol provided lower rates of hamstring injury and hamstring recurrence (Heiser et al. 1984).

Correcting any flexibility deficit is equally important to a strength deficit in terms of determining return to play. However, studies examining hamstring injury risk have consistently failed to show decreased flexibility as a risk factor for hamstring injury. With respect to groin strains, decreased range of hip abduction has been associated with groin injury.

Improved prognostic assessment of muscle strains with injury identification (MRI) and injury assessment (isokinetic testing) may assist practitioners to lower, but not eliminate, recurrent injuries. It was reported that 15 % of the players experienced a recurrence of their hamstring injury within the first 3 weeks of returning to play, with the majority of these occurring in the first match. Therefore, return to play criteria after a sport injury should represent a key point in order to decrease the risk of subsequent reinjury. Since some muscle groups are in high-risk location, physicians should consider the injured muscle for return to play decision. Biceps femoris, central tendon of rectus femoris, medial head of gastrocnemius, and adductor longus or magnus muscles are susceptible to reinjury (Orchard et al. 2005).

Despite the lack of high-quality evidence, our ability to manage return to play may be improving due to better understanding of prognosis, ability to identify risk factors for recurrent injury, an improved understanding of the mechanism of injury, identification of risk management strategies, and improved rehabilitation programs.

Cross-References

- ▶ Acute Hamstring Muscle Injury: Types, Rehabilitation, and Return to Sports
- ▶ Anesthesia Managements for Sports-Related Musculoskeletal Injuries
- ▶ Compartment Syndromes of Thigh and Lower Leg
- ▶ Exertional Rhabdomyolysis
- ▶ Lower Extremity Muscle Injuries in Professional Football Players: Overview
- ▶ Platelet-Rich Plasma in Muscle Injuries: When and How It Can Be Used
- ▶ Prevention of Hamstring Muscle Injuries in Sports
- ▶ Surgery of Muscle Injuries in Sports: When and Why?
- ▶ Usage of Growth Factors in Acute Muscle Injuries

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