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Muscle Injury: Pathophysiology, Diagnosis, and Treatment*

Lesão muscular: Fisiopatologia, diagnóstico e tratamento

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Abstract

Skeletal muscle tissue has the largest mass in the human body, accounting for 45% of the total weight. Muscle injuries can be caused by bruising, stretching or laceration. The current classification divides these injuries into mild, moderate and severe. The signs and symptoms of grade I lesions are edema and discomfort; grade II, loss of function, gaps and possible ecchymosis; and grade III, complete rupture, severe pain and extensive hematoma. The diagnosis can be confirmed by ultrasound, which is dynamic and cheap, but examiner dependent; and magnetic resonance imaging (MRI), which provides better anatomical definition. The initial phase of the treatment consists in protection, rest, optimal use of the affected limb, and cryotherapy. Nonsteroidal anti-inflammatory drugs (NSAIDs), ultrasound therapy, strengthening and stretching after the initial phase and range of motion without pain are used in the clinical treatment. On the other hand, surgery has precise indications: hematoma drainage and muscle-tendon reinsertion and reinforcement.

Keywords
- musculoskeletal system/ physiopathology
- musculoskeletal system/injury
- musculoskeletal system/surgery
- regeneration

Introduction

Muscle injuries are the most frequent cause of physical disability in sports practice. It is estimated that between 30 and 50% of all sports-associated injuries are caused by soft tissue injuries.[1] This incidence may be higher according to the sport. In athletics and soccer, ~ 30 to 41% of all injuries are muscular,[2–4] while in weightlifting, muscle injuries account for up to 59%.[5]

Although nonsurgical treatment results in a good prognosis in most athletes with muscle injury, the consequences of treatment failure can be dramatic, postponing the return to physical activity for weeks or even months.[6] Knowledge of

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Resumo

O tecido muscular esquelético possui a maior massa do corpo humano, correspondendo a 45% do peso total. As lesões musculares podem ser causadas por contusões, estiramentos ou lacerações. A atual classificação separa as lesões entre leves, moderadas e graves. Os sinais e sintomas das lesões grau I são edema e desconforto; grau II, perda de função, gap e equimose eventual; grau III, rotura completa, dor intensa e hematoma extenso. O diagnóstico pode ser confirmado por ultrassom (dinâmico e barato, porém examinador-dependente); e ressonância magnética (RM) (maior definição anatômica). A fase inicial do tratamento se resume à proteção, ao repouso, ao uso otimizado do membro afetado e crioterapia. Anti-inflamatórios não hormonais (AINHs), ultrassom terapêutico, fortalecimento e alongamento após a fase inicial e amplitudes de movimento sem dor são utilizados no tratamento clínico. Já o cirúrgico possui indicações precisas: drenagem do hematoma, reinserção e reforço musculotendíneos.

Palavras-chave

- sistema musculoesquelético/fisiopatologia
- sistema musculoesquelético/lesões
- sistema musculoesquelético/cirurgia
- regeneração

some basic principles of skeletal muscle regeneration and repair mechanisms can help prevent imminent dangers and accelerate the return to sport.

Anatomy and biomechanics

Muscle fibers usually originate in a bone or dense connective tissue and insert themselves into another bone through a tendon insertion.

There are muscles that go through one or more joints to generate movement. Muscles with tonic or postural function are usually uniarticular, wide, flat, with low contraction speed and with the ability to generate and maintain large contractile strength. They are usually located in the deepest compartments.

Biarticular muscles have greater contraction speed and ability to change length; however, they have less ability to withstand tension. They are usually located in surface compartments.

Regarding shape, the fusiform muscles allow a greater range of motion, while the feathered muscles have greater contractile strength.

Fiber length is an important determinant of the amount of contraction possible in muscles. Because muscle fibers usually have oblique distribution within a muscle belly, they are usually smaller than the total length of the muscle.

Mechanisms of injury

The cause of muscle injury can be considered indirect or direct.[7] Indirect injury is related to lack of contact. It may be of functional cause, due to mechanical overload or neurological injury,[8] or structural, which occurs when there is a partial or complete muscle rupture, such as the lesion in an eccentric concentration. Direct injury occurs at the contact site, which may cause a laceration or contusion. More than 90% of all sports-related injuries are bruises or stretches.[9] Muscle lacerations are the least frequent injuries in sports.

The tensil strength exerted on the muscle leads to an excessive stretching of myofibrils and, consequently, to a rupture near the myotendinous junction. Muscle stretches are typically observed in the superficial muscles that work crossing two joints, such as the recurrent femoral, semitendinous and gastrocnemius muscles.

Hamstrings, for example, show increased muscle tension as the hip and knee extend, either at the beginning of a *sprint* or of a kick, into a classic mechanism of muscle injury by eccentric contraction of a biarticulate muscle.[10]

Classification

Currently, there are several classification systems described for muscle injuries. In the last 10 years only, 5 different systems have been published.[11] Classically, the systems describe muscle injury at 3 different levels, mild, moderate and severe (or grade I, II and III) from imaging evaluation[12,13] or from the clinical aspects revealed.[14] New systems stage the lesions in a more complex way,[8,15,16] using, in addition to the characteristics described above, aspects related to the etiology and anatomical location of the lesion. ►**Table 1** shows some of the current classification models.[11]

The classification proposed by Mueller-Wohlfarht et al.,[8] known as the Munich Consensus, and the system described by Mafulli et al.,[16] also consider etiological aspects. These classify muscle injury as direct, caused by contusion or laceration, and indirect, subclassified into functional (non-structural) or structural.

The system described by Pollock et al.[15] (*British athletics muscle injury classification)* uses the anatomical location and extension of the lesion. It evaluates, through imaging, whether the damage is superficial (myofascial tissue), if it affects the myotendinous junction, or if there is a tendon injury.

The classification published by Valle et al.[7] seeks to group four characteristics of muscle injury into a system formed by the initials MLG-R, related to each letter as follows: mechanism of injury (M), location (L), degree of injury (G) and number of re-injuries (R).

The systems described above also consider clinical aspects, such as intensity, time of onset, and location of pain, to define the type of lesion and provide an adequate prognosis.

The classification of muscle injury in 3 levels is still well-known and used. It is usually based on clinical findings that

Table 1 Summary of 17 currently existing muscle classification models, divided between systems based on clinical and imaging findings, systems based on image evaluation, and systems based on clinical presentation[11]

1. Classifications based on clinical and imaging findings					
Author	**Description**				
Lopes, A. 1993.	**Classification based on etiology and ultrasound findings** Type I: muscle injury caused by extrinsic factors: muscle contusion Type II: muscle injury caused by intrinsic factors without muscle rupture Type III: muscle injury caused by intrinsic factors with muscle rupture				
Verrall, J. 2003.	**Clinical parameters**			**Image findings - MRI** Classification of the lesion	
	Beginning	Insidious	Abrupt		
	Circumstance	Playing	Training	Positive	Negative
	Pain	(0–10) visual analog scale			
Malliaropoulos, N. 2010.	**Clinical Grade - ROM Deficit**			**Image findings (US)**	
	I	< 10th		Degree 0 to 3 (based on Peetrons)	Injury area:
	II	10th–19th			< 25%
	III	20th–29th			25–50%
	IV	> 30th			> 50%
Pollock, N.[15] (British athletics muscle injury classification)	**Degree of injury**		**Description**	**MRI**	
	Grade 0: referred pain				
	0a		Local pain	Normal	
	0b		Generalized muscle pain	normal or with signs of delayed pain	
	Grade 1: small muscle injuries (< 5 cm or < 10% of total muscle area)				
	1a		Fascial pain	Intermuscular fluid	
	1b		Muscle or JMT pain	Intermuscular fluid	
	Grade 2: moderate muscle injuries (5–15 cm or 10–50% of total muscle area)				
	2a		Fascial pain	high perspherical signal	
	2b		Muscle or JMT pain	high signal in JMT	
	2c		Tendon pain	high sign on tendon	
	Grade 3: extensive muscle injuries (> 15 cm or > 50% of total muscle area)				
	3a		Fascial pain	high perspherical signal	
	3b		Muscle or JMT pain	high signal in JMT	
	3c		Tendon pain	high sign on tendon	
	Grade 4: complete muscle injuries				
	4a		Fascial pain	high perspherical signal	
	4b		Muscle or JMT pain	high signal in JMT	
	4c		Tendon pain	high sign on tendon	

(*Continued*)

Table 1 (*Continued*)

<table>
<tr><th colspan="5">1. Classifications based on clinical and imaging findings</th></tr>
<tr><td rowspan="4">Mueller-Wohlfahrt, H.[8]
(The Munich consensus statment)</td><td colspan="4">A. Indirect muscle injury</td></tr>
<tr><td colspan="4">Functional muscle injury
Type 1: Overload-related muscle disorder
Type 1A: Fatigue-induced muscle disorder
Type 1B: Late-onset muscle pain (DMIT)
Type 2: Neuromuscular disorder
Type 2A: Related to the spine
Type 2B: Related to muscles</td></tr>
<tr><td colspan="4">Structural muscle injury
Type 3: Partial muscle injury
Type 3A: Minimal partial muscle injury
Type 3B: Moderate partial muscle injury
Type 4: Injury (sub)total
Subtotal or complete muscle injury
Tendinous avulsion</td></tr>
<tr><td colspan="4">B. Direct muscle injury
Bruise
Laceration</td></tr>
<tr><td rowspan="3">Maffulli, N.[16]</td><td colspan="4">- Direct muscle injury
Bruise
Laceration</td></tr>
<tr><td colspan="4">- Indirect muscle injury
Nonstructural muscle injury
Type 1: Fatigue muscle injury
Type 1A: Fatigue-induced muscle disorder
Type 1B: Late-onset muscle pain (DMIT)
Type 2: Neuromuscular disorder
Type 2A: Related to the spine
Type 2B: Related to muscles</td></tr>
<tr><td colspan="4">- Indirect muscle injury
Structural muscle injury
Type 3: Partial muscle injury
Type 3A: Minimal partial muscle injury
Type 3B: Moderate partial muscle injury ($< 50\%$)
Type 4: Injury (sub)total
Subtotal or complete muscle injury
Tendinous avulsion
Structural lesions can be proximal (P), middle (M), and distal (D)</td></tr>
<tr><td rowspan="3">Valle, X.[7]</td><td colspan="4">Clinical findings</td></tr>
<tr><td>Injury mechanism (M)</td><td>Location of the lesion (L)</td><td>Degree of injury (G)</td><td>Rescan number (R)</td></tr>
<tr><td>T - Direct lesion of the hamstrings</td><td>P Lesion located in the proximal third of the muscle belly
M Lesion located in the middle third of the muscular belly
D Lesion located in the third of the muscular belly</td><td>0–3</td><td>0: 1st episode
1^{s}t reinjury
2: 2nd reinjury</td></tr>
</table>

Table 1 (*Continued*)

1. Classifications based on clinical and imaging findings				
	I - Indirect injury of the hamstrings, plus index s if it is by stretching (stretching), or index p if it is run.	P Lesion located in the proximal third of the muscle belly. The second letter is index p or d, describing whether the lesion is proximal or distal to JMT, respectively M Lesion located in the middle third of the muscle belly, plus the corresponding index D Lesion located in the middle third of the muscle belly, plus the corresponding index	0–3	
	N - Negative MRI injury	N p Lesion in the proximal third N m Injury in the middle third N d Lesion in the distal third	0–3	
	Magnetic resonance findings			
	Grade 0	Negative MRI		
	Grade 1	Hyperintense muscle fiber edema without intramuscular hemorrhage or change in architecture		
	Grade 2	Hyperintense edema of muscle fiber and/or paratendon with minimal intramuscular hemorrhage without gaps or minimal alteration in muscle architecture.		
	Grade 3	Any gap between muscle fibers in the craniocaudal or axial plane. Hyperintense focal defect with partial retraction of muscle fibers ± intermuscular hemorrhage.		
	(r) code overwrite	Used when there is intratendinous injury or affecting JMT or intramuscular injury with retraction or loss of normal tension.		
2. Classifications based on image findings				
Pomeranz, S. 1993.	**MRI assessment**			
	Muscle group involved	Injury area	Location	Superficial Involvement
	Semimembranosus	< 50%	Tendineous	Yes
	Semitendinosus	> 50%	JMT	No
	Femoris biceps	Total		
	Femoris square			
Takebayashi, S.[12]	**US findings**			
	Type 1		Normal	
	Type 2		Hyperecoic infiltration	
	Type 3		Mass	
	Type 4		Complete lesion (Infiltration + mass)	
Peetrons, P.[13]	**US findings**			
	Grade 0	Normal		
	Grade 1	Hyperecoic area, < 15 mm on the longest axis; < 5% of muscle.		
	Grade 2	5–50% of muscle. Partial muscle rupture.		
	Grade 3	Complete muscle or fascia injury, with collection extravasation from the injured muscle.		

(*Continued*)

Table 1 (*Continued*)

<table>
<tr><td colspan="8">1. Classifications based on clinical and imaging findings</td></tr>
<tr><td colspan="8">2. Classifications based on image findings</td></tr>
<tr><td rowspan="5">Slavotinek, J. 2002.</td><td colspan="7">MRI image of hamstring injury</td></tr>
<tr><td colspan="3">Affected muscle</td><td colspan="2">Location</td><td colspan="2">Total area of the lesion</td></tr>
<tr><td colspan="3">Femoris biceps</td><td colspan="2">Proximal to short biceps head</td><td colspan="2">0–100%</td></tr>
<tr><td colspan="3">Semitendinosus</td><td colspan="2">Distal to short biceps head</td><td colspan="2"></td></tr>
<tr><td colspan="3">Semimembranosus</td><td colspan="2"></td><td colspan="2"></td></tr>
<tr><td rowspan="2">Bordalo-Rodrigues, M. 2005</td><td colspan="7">MRI image of Proximal Rectus Femoris - anatomical location</td></tr>
<tr><td colspan="7">Avulsion injury of the apophysis
Musculotendinous junction injury (JMT)
Muscle contusion and laceration</td></tr>
<tr><td rowspan="8">Cohen, S. 2011.</td><td colspan="7">MRI-based graduation system</td></tr>
<tr><td>Item</td><td colspan="2">Description</td><td>0 points</td><td>1 point</td><td>2 points</td><td>3 points</td></tr>
<tr><td>1</td><td colspan="2">N° of muscles involved</td><td>No</td><td>1</td><td>2</td><td>3</td></tr>
<tr><td>2</td><td colspan="2">Location</td><td>–</td><td>Proximal</td><td>Middle</td><td>Distal</td></tr>
<tr><td>3</td><td colspan="2">Insertion</td><td>No</td><td>–</td><td>Yes</td><td>–</td></tr>
<tr><td>4</td><td colspan="2">Total area of injury in % of the muscle involved</td><td>0%</td><td>25%</td><td>50%</td><td>≥ 75%</td></tr>
<tr><td>5</td><td colspan="2">Retraction</td><td>No</td><td>–</td><td>> 2 cm</td><td>–</td></tr>
<tr><td>6</td><td colspan="2">Longitudinal axis involvement</td><td>0 cm</td><td>1–5 cm</td><td>6–10 cm</td><td>> 10 cm</td></tr>
<tr><td rowspan="5">Chan, O. 2012</td><td colspan="7">Graduation based on imaging findings and lesion site</td></tr>
<tr><td colspan="2">Degree</td><td colspan="2">MRI</td><td colspan="2">US</td><td>Local</td></tr>
<tr><td colspan="2">I (distension)</td><td colspan="2">< 5% fiber rupture;</td><td colspan="2">Normal; without distortion of architecture</td><td>. Proximal to JMT</td></tr>
<tr><td colspan="2">II (Partial loom)</td><td colspan="2">< 5% fiber rupture; high intramuscular signal; edema and bleeding of the muscle or JMT extending to the fascial planes between the muscle groups</td><td colspan="2">Discontinuity of muscle fibers</td><td>Muscle
A. Proximal B. Medium
C. Distal</td></tr>
<tr><td colspan="2">III (Complete loom)</td><td colspan="2">Complete discontinuity of muscle fibers, hematoma, and muscle retraction</td><td colspan="2">Comparable with MRI</td><td>Distal to JMT</td></tr>
</table>

Table 1 (*Continued*)

1. Classifications based on clinical and imaging findings							
2. Classifications based on image findings							
Corazza, A. 2013.	**Combined US-MRI assessment**						
	Degree	**MRI**			**US**		
	0	No pathological findings			No pathological findings		
	I	Muscle edema without tissue alteration			Altered echotexture at the site of pain, without rupture		
	Ii	Partial muscle injury			Lesion with associated hematoma		
	Iii	Complete muscle injury			Complete muscle injury		
3. Classifications based on clinical findings							
Bass, A. 1969.	**Classifies muscle injuries by etiology and location**						
	Type	Etiology			Location		
	I	Direct external contact			Intramuscular		
	Ii	Twitch			Intermuscular		
Wise, D. 1977	**Classification based on cause, severity, and location of leg muscle injury**						
	Indirect lesions - inflammation						
	Direct injuries - trauma						
	Degree	Pain	Circumference difference	Arc of motion	During contraction		
					Pain	Loss of strength	Function disorder
	I	Minimum;	< 6 mm	100%	Minimum	No	Moderate
	Ii	Substantial	6–12 mm	50%	Middle	Middle	Important
	Iii	Intractable	> 12 mm	<5 0%	Serious	almost total	Don't step

Abbreviations: JMT, myotendinous junction; MRI, magnectic resonance imaging; US, ultrasound.

are related to the extent of muscle tissue rupture, as described below.

Stretches and mild contusions (grade I) represent an injury of only a few muscle fibers with small edema and discomfort, accompanied by no or minimal loss of strength and movement restriction. It is not possible to palpate any muscle defect during muscle contraction. Although pain does not cause significant functional disability, maintenance of the athlete in activity is not recommended due to the high risk of increasing the extent of the injury.[4]

Moderate stretches and bruises (grade II) cause greater damage to the muscle, with evident loss of function (ability to contract). It is possible to palpate a small muscle defect, or gap, at the site of the lesion, and a slight local hematoma with eventual ecchymosis occurs within 2 to 3 days. The evolution to healing usually lasts from 2 to 3 weeks and, in ~1 month, the patient can return to physical activity slowly and carefully.[14]

An injury extending throughout the transverse session of the muscle and resulting in virtually complete loss of muscle function and severe pain is determined as severe stretch or contusion (grade III). The failure in the muscle structure is evident, and the ecchymosis is usually extensive, often distant to the site of rupture. This type of injury requires intense rehabilitation and for long periods of up to 3 to 4 months. The patient may remain with some degree of pain for months after the occurrence and treatment of the lesion.[14]

Pathophysiology

Skeletal muscle healing follows a constant order, with no major changes depending on the cause (contusion, stretch or laceration).

Three phases were identified in this process: destruction, repair, and remodeling. The last two phases (repair and remodeling) overlap and are closely related.

Phase 1: destruction – characterized by rupture and subsequent necrosis of myofibrils, by the formation of hematoma in the space formed between the ruptured muscle, and by the proliferation of inflammatory cells.

Phase 2: repair and remodeling – consists of the phagocytosis of the necrotic tissue, the regeneration of myofibrils, and the concomitant production of connective scar tissue, as well as vascular neoformation and neural growth.

Phase 3: remodeling – maturation period of regenerated myofibrils, contraction and reorganization of scar tissue, and recovery of muscle functional capacity.

Since myofibrils are fusiform and very long, there is an imminent risk that the necrosis initiated at the site of the lesion extends throughout the length of the fiber. However, there is a specific structure, called a contraction band, which is a condensation of the cytoskeletal material that acts as an "antifire system".[17]

Once the destruction phase decreases, the present repair of muscle injury begins with two simultaneous and competitive processes: the regeneration of the myofibril route and the formation of the scar connective tissue. A balanced progression of these processes is a prerequisite for optimal recovery of contractile muscle function.[17]

Although myofibrils are generally considered nonlytic, the regenerative capacity of skeletal muscle is guaranteed by an intrinsic mechanism that restores the injured contractile tract. During embryonic development, an undifferentiated cell reserve pool called satellite cells is stored below the basal lamina of each myofibril. In response to the lesion, these cells first proliferate, then differentiate into myofibrils, and finally join each other to form multinucleated myobribules.[18]

Over time, the formed scar decreases in size, leading the edges of the lesion to a greater grip with each other. However, it is not known whether the transection of the myofibrils from the opposite sides of the scar will definitely merge with each other or if it will form a septum of connective tissue between them.[19]

Immediately after the muscle injury, the interval formed between the rupture of muscle fibers is filled by hematoma. From the 1st day, inflammatory cells, including phagocytes, invade the hematoma and begin to organize the clot.[20]

Blood-derived fibrin and fibronectin intersperse to form granulation tissue, an initial frame and anchoring of the site for the recruited fibroblasts.[17] More importantly, this new formed fabric provides the property of initial tension to resist the contractions applied against it.

Approximately 10 days after the trauma, the maturation of the scar reaches a point in which it is no longer the most fragile site of the muscle injury.[21]

Although most skeletal muscle lesions heal without the formation of disabling fibrous scar tissue, fibroblast proliferation may be excessive, resulting in the formation of dense scar tissue within the muscle lesion.

A vital process for the regeneration of the injured muscle is the area of vascularization. Restoration of vascular supply is the first sign of regeneration and is a prerequisite for subsequent morphological and functional recoveries.[21]

Diagnosis

The diagnosis of muscle injury begins with a detailed clinical history of the trauma followed by a physical examination with inspection and palpation of the muscles involved, as well as function tests with and without external resistance.[23] The diagnosis is easy when a typical history of muscle contusion is accompanied by an evident edema or ecchymosis distal to the lesion.

Complementary exams

Imaging tests such as ultrasound (US), computed tomography (CT), and magnetic resonance imaging (MRI) provide useful information to verify and determine the lesion more accurately. New methods have been studied to detect physiological changes related to muscle injury, such as thermography.

Ultrasonography is traditionally considered the method of choice for initial evaluation of muscle injury. It is a relatively inexpensive and easily accessible imaging method. It is possible to dynamically evaluate muscle contraction and rupture. Renoux et al.[24] demonstrated a correlation between the severity of the acute muscle injury assessed by US with the time of return to sports activities. This examination presents the disadvantage of being examiner-dependent, having limited field of vision and reduced sensitivity for morphological evaluation.[25]

Computed tomography has already been shown to be able to identify changes related to muscle injuries, such as the presence of edema.[26] But the fact that CT generates radiation and produces a static image with little definition in relation to MRI[26] caused this evaluation method to be replaced.

Magnetic resonance imaging allows detailed evaluation of muscle morphology due to the ability to generate multiplanar and high-resolution soft tissue images.[25] It is the method of image evaluation used by many authors to define the classification of muscle injury.[8,15,16] The ability to differentiate ruptures and edemas and to perform calculation of the size of the hematoma proved to be useful in guiding the return time and the risk of re-injury in athletes.[27] In chronic lesions, it has the ability to show signs of tissue healing and fatty degeneration.[25] Advanced MRI techniques allow the evaluation of microstructure and muscle composition.[25]

Infrared medical thermography enables a noninvasive and nonradioactive assessment of body temperature. It allows the detection of physiological changes that mean increased risk of muscle injuries, such as inflammatory reactions by overload.[28,29] Thermography does not show data from deep surfaces and should not be used as a single diagnostic tool. Its use has been shown to be effective in preventing muscle injuries, reducing the incidence of injuries in professional soccer players by $> 60\%$.[30]

Treatment

The current principles of treatment of muscle injury are lacking in solid scientific foundations.

Early mobilization induces an increase in local vascularization in the lesion area, better regeneration of muscle fibers, and better parallelism between the orientation of regenerated myofibrils when compared with movement restriction.[31] However, reruptures at the original site of the trauma are common if active mobilization begins immediately after the injury.[33]

A short immobilization period with firm or similar adhesive bandage is recommended. This period of rest allows the scar tissue to reconnect to the muscle failure.[9]

The patient should use a pair of crutches for the most severe muscle injuries of the lower limbs, especially in the initial 3 to 7 days.

Acute phase

Immediate treatment for skeletal muscle injury or any soft tissue injury is known as the Protection, Rest, Ice, Compression, and Elevation (PRICE) principle. The justification for using the PRICE principle is because it is very practical, since the five measures cry out to minimize bleeding from the site of the injury.[23] Some authors advocate the use of the POLICE protocol, which presents as the main innovation the orientation for the optimized use of the injured limb in the acute phase, avoiding the adverse effects of long periods of rest.[33]

Putting the injured limb at rest soon after the trauma prevents a late muscle retraction or the formation of a larger muscle gap by reducing the size of the hematoma and, subsequently, the size of the scar connective tissue. Regarding the use of ice, it was shown that the early use of cryotherapy is associated with a significantly smaller hematoma in the gap of ruptured muscle fibers, with lower inflammation,[34] and with accelerated regeneration.[35]

The combination of ice application and compression in shifts of 15 to 20 minutes, repeated within intervals of between 30 and 60 minutes is recommended, since this type of protocol results in a decrease in 3° to 7°C of intramuscular temperature and in a 50% reduction of intramuscular blood flow.[37]

Finally, the elevation of the limb above the level of the heart results in decreased hydrostatic pressure, reducing the accumulation of fluid in the interstitial space.

Medication

There are few controlled studies using non-hormonal anti-inflammatory drugs (NHAIDs) or glucocorticoids in the treatment of muscle injuries in humans. O'Grady et al.[37] reported that the use of anti-inflammatory drugs in the treatment of in situ necrosis, the mildest type of muscle injury, in the short term, results in a transient improvement in the recovery of exercise-induced muscle injury. Despite the lack of evidence, the effects of NHAIDs have been well-documented. Järvinen[19] argued that short-term use in the early stages of recovery decreased the cellular inflammatory reaction without side effects on the healing process, on the tensil strength, or on the ability to contract muscle.

Furthermore, INAD does not delay the abilities activated by satellite cells in the proliferation or in the formation of myotubules.[38] However, chronic use seems to be harmful in the model of eccentric contraction in stretch lesions, as discussed by Mishra et al.[39]

Regarding the use of glucocorticoids, delays in the elimination of hematoma and necrotic tissue were reported, as well as delay in the regeneration process and reduction of the biomechanical strength of the injured muscle.[40]

Acute postphase treatment

1. Isometric training (muscle contraction in which the muscle length remains constant and tension changes) can be started without the use of weights and, later, with the addition of them. Special attention should be taken to ensure that all isometric exercises are performed painlessly.
2. Isotonic training (muscle contraction in which the muscle size changes and tension is maintained) can be initiated when isometric training is performed painlessly with resisted loads.
3. Isokinetic exercise with minimum load can be initiated once the two previous exercises are performed painlessly.

Local application of heat or "contrast therapy" (hot and cold) can be of value, accompanied by careful passive and active stretching of the affected muscle. It is emphasized that any rehabilitation activity should be initiated with the proper heating of the injured muscle.[41]

Another reason for stretching is to distend the mature scar tissue during the phase when it is still plastic. Pain-free scar stretches can be acquired by gradual stretches, starting with shifts of 10 to 15 seconds and then progressing to periods of up to 1 minute.

However, if the symptoms caused by the lesion do not improve in between 3 and 5 days after the trauma, the possibility of an intramuscular hematoma or of extensively injured tissue that will require special attention should be considered. Puncture or aspiration of the hematoma may be necessary.

Ultrasound

Therapeutic US is diffusely recommended and used in the treatment of muscle injury; some authors argue that there is vague scientific evidence of its effectiveness.[42] The fact that US produces micromassages by high frequency waves apparently makes it work for pain relief. Engelmann et al.[43] showed a reduction in inflammatory activity with the use of pulsed US. Ultrasound may also be useful for the performance of therapeutic procedures and in the surgical treatment of muscle injuries.[44,45]

Surgical treatment

There are precise indications in which surgical intervention is required. These indications include patients with large intramuscular hematomas, complete lesions or ruptures (grade III) with little or no associated agonist musculature, and partial lesions in which more than half of the muscle is ruptured.[46,47]

Surgical intervention can also be considered if the patient complains of persistent pain when stretching for > 4 to 6 months, particularly if there is an extension deficit. In this case, scar injuries should be suspected, restricting muscle movement at the site of the injury.

After surgical repair, the muscle should be protected by an elastic bandage around the limb to promote relative immobility and compression. Naturally, the duration of immobilization depends on the severity of the trauma. Patients with complete rupture of the quadriceps or of the gastrocnemius muscle are instructed not to load the limb for at least 4 weeks.

If the gap or muscle failure is exceptionally wide, the denervated part can generate a permanent neurological deficit and consequent muscle atrophy.[21] Surgical repair in these circumstances increases the chance of reinnervation, and the development of thick scar tissue can be avoided.

New perspectives

The therapeutic use of growth factors and gene therapy, alone or in combination, and the application of stem cells provide the latest and most promising existing therapeutic options. However, there is currently a need for greater scientific validation for its intensification in the treatment of skeletal muscle injuries.

Growth factors and cytokines are potent mitogenic activators for numerous cells, including myogenic precursor cells (MPCs) during the regeneration of injured muscle cells.[48] Therefore, they are promising therapeutic options to aid in the recovery of skeletal muscles.

In relation to stem cells, it has recently been shown that, in response to the injury, not only tissue-specific cells, but also nonmuscle stem cells participate in the repair process.[49]

The first steps of gene therapy have already been taken. Successful studies have shown good results of the use of stem cells in muscle tissue in the treatment of muscular dystrophy, of cardiac muscle injuries, and of urinary incontinence.[10,50] Future studies will demonstrate in which sphere gene therapy can fulfill the current expectations regarding the treatment of muscle trauma scans.

Clinical Presentation

Quadriceps muscle injury

Distal quadriceps injury is an unusual lesion, occurring more frequently in individuals > 40 years old.[51] The injury may occur due to direct trauma, but it is classically reported as a forced eccentric contraction in a position of slight flexion of the lower limb in an attempt to regain balance at the time of a fall.

Spontaneous ruptures and bilateral ruptures have been described in athletes with systemic metabolic disorders and steroid use.[51]

The diagnosis of rupture is based on clinical findings. The patient typically presents, after a fall with flexed knees, acute pain above the patella and the inability to remain in the orthostatic position without assistance.

During physical examination, the patient is not able to actively extend the knee and, often, there is a palpable interval above the patella, known as the "groove sign" or gap test. Patients can actively flex the knee and have total passive knee flexion and extension.

Plain radiography is an inexpensive tool for the diagnosis of breakage. Although it does not show a specific alteration of the lesion, it shows indirect signs of rupture. Soft tissue edema, joint effusion, calcifications, shadow of quadriceps rupture, and low patella are all indirect signs seen on plain radiography.[53]

Ultrasound is another inexpensive method for diagnosing muscle injury. Magnetic resonance imaging is particularly useful for better visualization, accuracy of lesion location and extent, and anatomical details for preoperative programming.

For complete muscle ruptures, the treatment is surgical. Early surgical treatment in these cases is associated with better functional results.[54] The delay in surgical repair is associated with a period of prolonged physiotherapy, with inadequate flexion, and with loss of total knee extension.[54] After surgical repair, patients have the knee immobilized for 4 to 6 weeks.

Injury of the hamstring muscles

The hamstring muscles are the least elongated of the lower limb and, for this reason, more easily injured during eccentric muscle contraction.

The severity of the injury is usually neglected, especially in the acute phase.

Hamstring stretching is the most common lesion in athletes.[55]

The diagnosis of the lesion is usually made from a high rate of clinical suspicion and careful clinical examination. Magnetic resonance imaging is valuable for differentiating between a complete or incomplete lesion and for treatment planning.

Complete rupture of the hamstring muscles proximally in their origin is rare. The conduction of the case varies between conservative treatment with an immobilizer in flexion and surgical repair in a second moment. Although surgical repair in a second moment may show good results, early repair allows for faster functional rehabilitation and avoids the potential neurological symptom of gluteal sciatica.

Adductor muscle injury

The adductor muscle group acts in conjunction with the low abdominal muscles to stabilize the pelvis during activities involving the lower limbs. Athletes who participate in activities that require repetitive kicks, starts, or frequent changes of direction have a higher incidence of chronic pain in the topography of the adductors.[5]

There is evidence that athletes with imbalance between the adductor musculature and the abdominal wall are more likely to acquire pubalgia during the season.[57] Weakness of the adductor muscles and decreased amplitude of hip movement are also related to pubalgia.[58]

Patients typically present with a sore groin area or medial pain in the thigh and may or may not report a triggering factor. On physical examination, pain is presented on palpation with focal edema along the adductor muscles and decreased muscle strength and pain in resistance exercise of hip adduction.

The diagnosis can be made with the findings of the physical examination. However, contrast-enhanced MRI may be useful to confirm the diagnosis or to make the differential diagnosis between pubic osteitis and sports hernia.[59]

The initial treatment is conservative. Infiltration of the long adductor entese may be useful for refractory treatment. In cases of acute rupture, open surgical repair with anchor placement and suture has been described with good results.[60]

Patients may resume the sport after returning to the previous pattern of strengthening and range of motion of the hip and resolution of pain. Due to the predisposition of the adductor injury to be caused by muscle imbalance, attention should be paid to strengthening the musculature to prevent further injuries.

Injury of gastrocnemius muscles

Like the hamstring and quadriceps muscles, the gastrocnemius is prone to injury because it crosses two joints.

The medial head of the gastrocnemius is more commonly injured than the lateral head, since it is more active.[61] Deep vein thrombosis may be associated with or be a differential diagnosis of calf pain, as well as thrombophlebitis.[62]

The term tennis leg has been used to describe calf pain and injury. The term is attributed to the movement of the serve in tennis, in which there is a complete extension of the knee associated with an abrupt dorsiflexion of the ankle, causing maximum stretching of the calf. However, this injury has also been described in young athletes during periods of strenuous exercises such as basketball, running, and bodybuilding.[63]

The onset of pain is sudden, with focal edema and ecmosis of the calf. Classically, tennis leg is referred to as a lesion of the distal myotendinous junction, although proximal injury may occur.

Because of the superficial nature of the lesion, US evaluation is reliable, makes it possible to easily exclude the presence of deep vein thrombosis, and provides aspiration of image-guided liquid collections.

The treatment of most gastrocnemius lesions is conservative. Occasionally, surgery should be performed to drain hematomas, to repair a grade III lesion, or to perform compartmental decompression in cases of compartment syndrome.

Pectoral muscle snare injury

The pectoralis major (PM) muscle presents a complex anatomy. The tendon is bilaminar (anterior and posterior layers) and the muscular belly is composed of the clavicular head and of the sternal head, divided into 7 segments.[65]

Cases of PM muscle injury have become more common in recent years. The main reason is the increase in the practice of weightlifting. The most common mechanism is indirect injury during the eccentric phase in weightlifting in supine.[65] This injury is also frequent in sports such as gymnastics, Greco-Roman wrestling, and windsurfing.[66]

Loss of upper limb adduction strength leads to the need for surgical treatment, both for acute (up to 3 weeks) and chronic (after 3 weeks) lesions. Treatment in the acute phase is usually repair near the humeral insertion. In chronic lesions, reconstruction of the PM tendon with the use of flexor tendons may be necessary.[66]

Minor pectoral muscle injuries are rare, and are often confused with PM injury. Conservative treatment was effective in the few reported cases of this type of lesion.[67]

Distal lesion of the brachial biceps muscle

The brachial biceps muscle is composed of the long head, which originates in the supraglenoidal tubercle and acts on the supination, and of the short head, which originates from the coracoid process and presents a greater performance in elbow flexion. The distal insertion is in the radial tuberosity.[68]

Distal rupture is uncommon, occurring in 10% of all lesions of the biceps. It happens mainly in the dominant limb, in male patients. The mechanism is eccentric contraction during elbow extension.[9]

Biomechanical studies show reduced supination strength and resistance and a lower loss of elbow flexion strength. Conservative treatment is usually indicated for sedentary or low-demand patients.[68] Surgical treatment is performed by reinsertion into the radial tuberosity with the use of cortical buttons, anchors, interference screw, or transosseous suture.[68]

Final Considerations

Understanding the pathophysiological mechanisms that regulate muscle repair and its adaptation to physical training are essential for the professional who proposes to treat these patients. They are the basis for the development of means of injury prevention and for the proper treatment and rehabilitation of installed injuries.

Regarding the appropriate time of return to training specific to the sport, the decision can be based on two simple and inexpensive measures: the ability to lengthen the injured muscle as much as the healthy contralateral side, and absence of pain in the injured muscle in basic movements.

When the patient refers to reaching this point in recovery, permission to gradually start the exercises specific to the sport is guaranteed. However, it should always be emphasized that the final phase of rehabilitation should be carried out under the supervision of a qualified professional.

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Conflict of Interests
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References

1 Herring SA, Nilson KL. Introduction to overuse injuries. Clin Sports Med 1987;6(02):225–239
2 Edouard P, Branco P, Alonso JM. Muscle injury is the principal injury type and hamstring muscle injury is the first injury diagnosis during top-level international athletics championships between 2007 and 2015. Br J Sports Med 2016;50(10): 619–630
3 Ekstrand J, Hägglund M, Waldén M. Epidemiology of muscle injuries in professional football (soccer). Am J Sports Med 2011; 39(06):1226–1232
4 Jones A, Jones G, Greig N, et al. Epidemiology of injury in English Professional Football players: A cohort study. Phys Ther Sport 2019;35:18–22
5 Aasa U, Svartholm I, Andersson F, Berglund L. Injuries among weightlifters and powerlifters: a systematic review. Br J Sports Med 2017;51(04):211–219
6 Pedrinelli A, Fernandes TL, Thiele E, Teixeira W. Lesão muscular - ciências básicas, fisiopatologia, diagnóstico e tratamento. In: Alves Júnior W, Fernandes T, eds. Programa de Atualização em Traumatologia e Ortopedia (PROATO). Porto Alegre: Artmed; 2006:10
7 Valle X, Alentorn-Geli E, Tol JL, et al. Muscle Injuries in Sports: A New Evidence-Informed and Expert Consensus-Based Classification with Clinical Application. Sports Med 2017;47(07): 1241–1253
8 Mueller-Wohlfahrt HW, Haensel L, Mithoefer K, et al. Terminology and classification of muscle injuries in sport: the Munich consensus statement. Br J Sports Med 2013;47(06):342–350
9 Järvinen MJ, Lehto MU. The effects of early mobilisation and immobilisation on the healing process following muscle injuries. Sports Med 1993;15(02):78–89
10 Almeida A, Dorileo C, Thiele E, SantAnna JPC, Costa PHP. Lesões musculares. In: Cristante AF, Brandão GF, editores. Programa de Atualização em Traumatologia e Ortopedia (PROATO): Ciclo 12. Porto Alegre: Artmed; 2015:85–110
11 Santanna JPC, de Almeida AM, Pedrinelli A, Hernandez AJ, Fernandes TL. Quality assessment of muscle injury classification in sports: A systematic literature review. Muscles Ligaments Tendons J 2018;8(02):206–221
12 Takebayashi S, Takasawa H, Banzai Y, et al. Sonographic findings in muscle strain injury: clinical and MR imaging correlation. J Ultrasound Med 1995;14(12):899–905
13 Peetrons P. Ultrasound of muscles. Eur Radiol 2002;12(01):35–43
14 Hernandez AJ. Distensões e rupturas musculares. In: Camanho GL, editor. Patologia do Joelho. Sao Paulo: Sarvier; 1996:132–138
15 Pollock N, James SL, Lee JC, Chakraverty R. British athletics muscle injury classification: a new grading system. Br J Sports Med 2014; 48(18):1347–1351
16 Maffulli N, Oliva F, Frizziero A, et al. ISMuLT Guidelines for muscle injuries. Muscles Ligaments Tendons J 2014;3(04):241–249
17 Hurme T, Kalimo H, Lehto M, Järvinen M. Healing of skeletal muscle injury: an ultrastructural and immunohistochemical study. Med Sci Sports Exerc 1991;23(07):801–810
18 Rantanen J, Hurme T, Lukka R, Heino J, Kalimo H. Satellite cell proliferation and the expression of myogenin and desmin in regenerating skeletal muscle: evidence for two different populations of satellite cells. Lab Invest 1995;72(03):341–347
19 Äärimaa V, Kääriäinen M, Vaittinen S, et al. Restoration of myofiber continuity after transection injury in the rat soleus. Neuromuscul Disord 2004;14(07):421–428
20 Cannon JG, St Pierre BA. Cytokines in exertion-induced skeletal muscle injury. Mol Cell Biochem 1998;179(1-2):159–167
21 Kääriäinen M, Kääriäinen J, Järvinen TL, Sievänen H, Kalimo H, Järvinen M. Correlation between biomechanical and structural changes during the regeneration of skeletal muscle after laceration injury. J Orthop Res 1998;16(02):197–206
22 Järvinen M. Healing of a crush injury in rat striated muscle. 3. A micro-angiographical study of the effect of early mobilization and immobilization on capillary ingrowth. Acta Pathol Microbiol Scand A 1976;84(01):85–94
23 Fernandes TL, Pedrinelli A, Hernandez AJ. Dor na coxa e na perna. In: Nobrega A, editor. Manual de Medicina do Esporte. Sao Paulo: Atheneu; 2009:140–141
24 Renoux J, Brasseur J-L, Wagner M, et al. Ultrasound-detected connective tissue involvement in acute muscle injuries in elite athletes and return to play: The French National Institute of Sports (INSEP) study. J Sci Med Sport 2019;22(06):641–646
25 Crema MD, Yamada AF, Guermazi A, Roemer FW, Skaf AY. Imaging techniques for muscle injury in sports medicine and clinical relevance. Curr Rev Musculoskelet Med 2015;8(02):154–161
26 Davis KW. Imaging of the hamstrings. Semin Musculoskelet Radiol 2008;12(01):28–41
27 Ekstrand J, Healy JC, Waldén M, Lee JC, English B, Hägglund M. Hamstring muscle injuries in professional football: the correlation of MRI findings with return to play. Br J Sports Med 2012;46 (02):112–117
28 Côrte ACR, Hernandez AJ. Termografia Médica Infravermelha Aplicada à Medicina do Esporte. Rev Bras Med Esporte 2016;22 (04):315–319

29 Bandeira F, Neves EB, Barroso GC, Nohama P. Métodos de apoio ao diagnóstico de lesões musculares. Rev Bras Inov Tecnol Saúde 2013;3(03):27–44

30 Côrte AC, Pedrinelli A, Marttos A, Souza IFG, Grava J, Hernandez AJ. Infrared thermography study as a complementary method of screening and prevention of muscle injuries: pilot study. BMJ Open Sport Exerc Med 2019;5(01):e000431

31 Järvinen M. Healing of a crush injury in rat striated muscle. 2. a histological study of the effect of early mobilization and immobilization on the repair processes. Acta Pathol Microbiol Scand A 1975;83(03):269–282

32 Lehto M, Duance VC, Restall D. Collagen and fibronectin in a healing skeletal muscle injury. An immunohistological study of the effects of physical activity on the repair of injured gastrocnemius muscle in the rat. J Bone Joint Surg Br 1985;67(05):820–828

33 Bleakley CM, Glasgow P, MacAuley DC. PRICE needs updating, should we call the POLICE? Br J Sports Med 2012;46(04):220–221

34 Puntel GO, Carvalho NR, Amaral GP, et al. Therapeutic cold: An effective kind to modulate the oxidative damage resulting of a skeletal muscle contusion. Free Radic Res 2011;45(02):125–138

35 Hurme T, Rantanen J, Kaliomo H. Effects of early cryotherapy in experimental skeletal muscle injury. Scand J Med Sci Sports 1993; 3(01):46–51

36 Thorsson O, Hemdal B, Lilja B, Westlin N. The effect of external pressure on intramuscular blood flow at rest and after running. Med Sci Sports Exerc 1987;19(05):469–473

37 O'Grady M, Hackney AC, Schneider K, et al. Diclofenac sodium (Voltaren) reduced exercise-induced injury in human skeletal muscle. Med Sci Sports Exerc 2000;32(07):1191–1196

38 Thorsson O, Rantanen J, Hurme T, Kalimo H. Effects of nonsteroidal antiinflammatory medication on satellite cell proliferation during muscle regeneration. Am J Sports Med 1998;26(02): 172–176

39 Mishra DK, Fridén J, Schmitz MC, Lieber RL. Anti-inflammatory medication after muscle injury. A treatment resulting in short-term improvement but subsequent loss of muscle function. J Bone Joint Surg Am 1995;77(10):1510–1519

40 Beiner JM, Jokl P, Cholewicki J, Panjabi MM. The effect of anabolic steroids and corticosteroids on healing of muscle contusion injury. Am J Sports Med 1999;27(01):2–9

41 Magnusson SP, Simonsen EB, Aagaard P, Gleim GW, McHugh MP, Kjaer M. Viscoelastic response to repeated static stretching in the human hamstring muscle. Scand J Med Sci Sports 1995;5(06): 342–347

42 Wilkin LD, Merrick MA, Kirby TE, Devor ST. Influence of therapeutic ultrasound on skeletal muscle regeneration following blunt contusion. Int J Sports Med 2004;25(01):73–77

43 Engelmann J, Vitto MF, Cesconetto PA, et al. Pulsed ultrasound and dimethylsulfoxide gel treatment reduces the expression of proinflammatory molecules in an animal model of muscle injury. Ultrasound Med Biol 2012;38(08):1470–1475

44 Wood JP, Beaulieu CF. Musculotendinous Injuries: Sonographic-guided Interventions. Semin Musculoskelet Radiol 2017;21(04): 470–484

45 Ballard DH, Campbell KJ, Hedgepeth KB, et al. Anatomic guide and sonography for surgical repair of leg muscle lacerations. J Surg Res 2013;184(01):178–182

46 Almekinders LC. Results of surgical repair versus splinting of experimentally transected muscle. J Orthop Trauma 1991;5 (02):173–176

47 Kujala UM, Orava S, Järvinen M. Hamstring injuries. Current trends in treatment and prevention. Sports Med 1997;23(06): 397–404

48 Best TM, Shehadeh SE, Leverson G, Michel JT, Corr DT, Aeschlimann D. Analysis of changes in mRNA levels of myoblast- and fibroblast-derived gene products in healing skeletal muscle using quantitative reverse transcription-polymerase chain reaction. J Orthop Res 2001;19(04):565–572

49 LaBarge MA, Blau HM. Biological progression from adult bone marrow to mononucleate muscle stem cell to multinucleate muscle fiber in response to injury. Cell 2002;111(04):589–601

50 Maclean S, Khan WS, Malik AA, Anand S, Snow M. The potential of stem cells in the treatment of skeletal muscle injury and disease. Stem Cells Int 2012;2012:282348

51 Siwek CW, Rao JP. Ruptures of the extensor mechanism of the knee joint. J Bone Joint Surg Am 1981;63(06):932–937

52 Liow RY, Tavares S. Bilateral rupture of the quadriceps tendon associated with anabolic steroids. Br J Sports Med 1995;29(02): 77–79

53 Stephens BO, Anderson GVJ Jr. Simultaneous bilateral quadriceps tendon rupture: a case report and subject review. J Emerg Med 1987;5(06):481–485

54 Walker LG, Glick H. Bilateral spontaneous quadriceps tendon ruptures. A case report and review of the literature. Orthop Rev 1989;18(08):867–871

55 Blasier RB, Morawa LG. Complete rupture of the hamstring origin from a water skiing injury. Am J Sports Med 1990;18(04):435–437

56 Cunningham PM, Brennan D, O'Connell M, MacMahon P, O'Neill P, Eustace S. Patterns of bone and soft-tissue injury at the symphysis pubis in soccer players: observations at MRI. AJR Am J Roentgenol 2007;188(03):W291-6

57 Almeida MO, Silva BN, Andriolo RB, Atallah ÁN, Peccin MS. Conservative interventions for treating exercise-related musculotendinous, ligamentous and osseous groin pain. Cochrane Database Syst Rev 2013;(06):CD009565

58 Verrall GM, Slavotinek JP, Barnes PG, Esterman A, Oakeshott RD, Spriggins AJ. Hip joint range of motion restriction precedes athletic chronic groin injury. J Sci Med Sport 2007;10(06): 463–466

59 Schilders E, Bismil Q, Robinson P, O'Connor PJ, Gibbon WW, Talbot JC. Adductor-related groin pain in competitive athletes. Role of adductor enthesis, magnetic resonance imaging, and entheseal pubic cleft injections. J Bone Joint Surg Am 2007;89(10): 2173–2178

60 Vogt S, Ansah P, Imhoff AB. Complete osseous avulsion of the adductor longus muscle: acute repair with three fiberwire suture anchors. Arch Orthop Trauma Surg 2007;127(08):613–615

61 Segal RL, Song AW. Nonuniform activity of human calf muscles during an exercise task. Arch Phys Med Rehabil 2005;86(10): 2013–2017

62 Bianchi S, Martinoli C, Abdelwahab IF, Derchi LE, Damiani S. Sonographic evaluation of tears of the gastrocnemius medial head ("tennis leg"). J Ultrasound Med 1998;17(03):157–162

63 McClure JG. Gastrocnemius musculotendinous rupture: a condition confused with thrombophlebitis. South Med J 1984;77(09): 1143–1145

64 ElMaraghy AW, Devereaux MW. A systematic review and comprehensive classification of pectoralis major tears. J Shoulder Elbow Surg 2012;21(03):412–422

65 de Castro Pochini A, Ejnisman B, Andreoli CV, et al. Exact moment of tendon of pectoralis major muscle rupture captured on video. Br J Sports Med 2007;41(09):618–619

66 Pochini AC, Rodrigues MSB, Yamashita L, Belangero PS, Andreoli CV, Ejnisman B. Surgical treatment of pectoralis major muscle rupture with adjustable cortical button. Rev Bras Ortop 2017;53 (01):60–66

67 Vance DD, Qayyum U, Jobin CM. Rare Isolated Pectoralis Minor Tear from a Noncontact Injury: Case Report and Review of the Literature. Case Rep Orthop 2019;2019:3605187

68 Cerciello S, Visonà E, Corona K, Ribeiro Filho PR, Carbone S. The Treatment of Distal Biceps Ruptures: An Overview. Joints 2019;6 (04):228–231

69 Krych AJ, Kohen RB, Rodeo SA, Barnes RP, Warren RF, Hotchkiss RN. Acute brachialis muscle rupture caused by closed elbow dislocation in a professional American football player. J Shoulder Elbow Surg 2012;21(07):e1–e5

