

DEFINITION OF ACUTE MUSCLE INJURY

WHAT HAVE WE LEARNT?

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Muscle strains occur most often at the musculotendinous junction as a consequence of indirect trauma. Two-joint muscles, muscles contracting eccentrically and with a higher percentage of type II fibres are most predisposed to injury. Since structural and biochemical changes occur after the initial insult, the overall goal is to assist the body with its natural healing process by respecting the inflammatory, repair and remodelling phases. To better define acute muscle strain injuries, an imaging (MRI or ultrasound [US]) classification can be used, which considers the anatomical site, pattern and severity of the lesion in the acute stage.

INTRODUCTION

Muscle lesions are common in sports, especially among high demand and elite athletes¹. Short-term outcome and long-term prognosis are difficult to predict². The

main site of injury is the musculotendinous junction of the hamstring, rectus femoris and medial head of the gastrocnemius. These muscles all contain a relatively high percentage of type II fibres, are arranged in a pennate muscle architecture and cross two joints. Eccentric contraction is the typical mechanism of injury, in which great tensions are developed and the muscle is susceptible to being injured³. In this article we describe a state of the art method for the treatment of acute muscle injuries, specifically acute strains. We present predisposing factors, mechanisms of injury and recent models of classification to aid prevention, diagnosis and management of such lesions.

MUSCLE INJURY CLASSIFICATION

Acute injuries occur after direct and indirect trauma. When the trauma is direct, the injury is typically a contusion, evident at the point of contact. This is common

“Direct trauma is common in contact sports such as soccer, rugby, football and martial arts”



in contact or combat sports such as soccer, rugby, football and martial arts after application of large compressive forces. Rarely, lacerations are observed when a blunt trauma is directly applied to the epimysium and underlying muscles⁴. On the other hand, there is no contact in indirect injuries, but some myofibres are nevertheless stretched both passively and actively and then disrupted. Passive injuries are produced by flexible overstretching loads, without any contraction; active lesions result from eccentric overloading to the muscle⁵. In this case, muscle strains, which are very common in sprinters and jumpers such as high jumpers, long jumpers, sprinters, long distance runners and tennis players, occur because of an indirect trauma and application of excessive tensile forces⁶. Muscle lesions are classified as grade I, II and III, based on the amount of fibres disrupted according to clinical and imaging investigations.

Grade I injury (STRAIN)

Characteristics:

- Swelling and discomfort of muscle fibres.
- Minimal impairment of strength and function.

US findings:

- May be normal.
- Some perifascial fluid may be evident in around 50% of patients.

MRI findings:

- A classic 'feathery' oedema-like pattern.

- Feathery pattern may appear on fluid-sensitive sequences, with some fluid appearing in the central portion of the tendon and along the perifascial intermuscular region.
- No discernible disruption of muscle fibres or architectural distortion⁷.

Grade II injury (PARTIAL TEAR)

Characteristics:

- Macroscopically evident at imaging assessment with some continuity of fibres appreciable at the injury site.
- <1/3 of fibres are torn in low grade injuries.
- 1/3 to 2/3 fibres are torn in moderate lesions.
- >2/3 fibres are torn in high-grade injuries.

US findings:

- Muscle fibres are discontinuous.
- The disruption site is hypervascularised.
- Echogenicity is altered in and around the lesion⁸.

MRI findings:

- A varied appearance with both the acuity and the severity of the partial tear.
- Changes are time-dependent.
- Oedema and haemorrhage of the muscle or musculotendinous junction (MTJ) may extend between muscle groups along the fascial planes.

- MRI can assist in predicting return to play timelines for high performance athletes.

Clinical findings:

- Clinical and functional assessment shows muscle strength and high speed/high resistance athletic activities are impaired with marked loss of muscle function.

Grade III injury (COMPLETE TEAR)

US and MRI findings:

- Complete discontinuity of muscle fibres, haematoma and retraction of the muscle ends⁸.

Clinical findings:

- Muscle function is lost.
- Difficulty distinguishing partial from complete tears if extensive oedema and haemorrhage fill the defect between the torn edges.
- At this stage, real-time dynamic US imaging may be helpful⁴.

PREDISPOSING FACTORS

The injury pattern changes for directions and angle movements of forces applied. Contusions, strains or lacerations may be distinguished⁹.

Three types of muscle are at possible risk of injury:

1. **Two-joint muscles.** Specifically, the motion at one joint increases the passive tension of the muscle and leads to an overstretching injury.
2. **Muscles contracting eccentrically.** Eccentric contractions, frequent during the deceleration phase of activity, may change the muscle tension and induce myofibre overload injury¹⁰.
3. **Muscles with a higher percentage of type II fibres.** These are fast-twitch muscles which develop high speed contractions. In this way, the muscle is more predisposed to injury. Most of the muscle activity in running and sprinting is eccentric, explaining why such strains most often occur in sprinters or 'speed athletes'. The hamstrings, gastrocnemius, quadriceps, hip flexors, hip adductors, erector spinae, deltoid and rotator cuff are most commonly injured.

Flexibility imbalances between agonists and antagonists may also predispose an athlete to injury. Flexible muscles are most likely to be injured¹¹. A previous injury makes muscles more vulnerable to re-injury, explaining why sprinters with recent hamstring injuries have tighter and weaker hamstrings than those with no previous injury⁹.

When rehabilitation is inadequate, strength, flexibility and endurance may not be completely restored when an athlete returns to activity. Therefore, residual weakness and impairment may predispose the muscle to a new injury. Since cold or tight muscles are more predisposed to muscular strain, proper stretching exercises and warm-up may prevent muscular injury¹². After warm-up, muscle elongation before failure is increased¹³.

STRUCTURAL CHANGES

The musculotendinous junction, the weakest link within the muscle tendon unit, is the most vulnerable site in indirect strain injuries¹⁴. When muscle tension increases suddenly, as it occurs in eccentric contractions, the damage is in the area beneath the epimysium and the site of muscle attachment to the periosteum⁵. In injuries of the fascia, which are often found in the medial calf and biceps femoris, differential contractions of adjacent muscle bellies can produce aponeurotic distraction injuries^{15,16}. Hamstring strain muscle injuries, which are the most widely studied, typically occur in the region of the MTJ. This region is a transition zone organised in a system of highly folded membranes, designed to increase the junctional surface area and dissipate energy. The region adjacent to the MTJ is more susceptible to injury than any other component of the muscle. Susceptibility is independent of type and direction



of applied forces and muscle formation¹⁷. This strain may weaken the muscle and predispose it to further injury. Disruptions in the fibres cause biochemical changes both from direct injury to the fibres and from the inflammatory reaction.

BIOCHEMICAL CHANGES

Released in response to an insult, serum creatine kinase and lactate dehydrogenase enzyme levels are indirect markers of muscle damage after eccentric exercise¹⁸. Inflammatory reactions also occur. Acute inflammation protects, localises and removes injurious agents from the body and

promotes healing and repair¹⁹. Chemical inflammatory mediators such as histamine, serotonin, bradykinin and prostaglandin appear in acute muscular strain. After a strain, the capillary membrane permeability increases, blood vessels change in diameter and pain receptors are stimulated. Therefore, the accumulation of proteins and transudate in the interstitial space produces oedema²⁰. The acute phase of inflammation lasts up to 3 to 4 days after the initial insult. Proliferation of fibroblasts, increased collagen production and degradation of mature collagen weaken the tissue. In this way, stretching the tissue induces

progressive irritation and limitation, up to predisposing to chronic muscle strains.

Healing

When the inflammatory phase subsides, repair is started and continues for 2 to 3 weeks. Specifically, capillary growth and fibroblast activity to form immature collagen are promoted. This immature collagen is easily injured if overstressed. The final stage of healing is maturation and remodelling of collagen, occurring from 2 to 3 weeks after the insult, until patients are pain-free. In the healing phase, if fibres are not properly stressed, surrounding adhesions and scarring resilience to remodelling may form.

TREATMENT OF ACUTE STRAINS

Management varies with the severity of the injury, the natural healing processes of the body and the response of the tissue to new demands.

The overall goal is to assist and respect the body with its natural healing processes. Therefore, the athletic trainer must not return the athlete to activity too soon. Activity should be restricted for 2 to 3 weeks to allow collagen formation and prevent re-injury.

Inflammatory phase

Rest, ice, compression and elevation (RICE) are suggested for at least 48 hours. Rest protects the injured tissue, but immobilisation may be detrimental to healing and uninjured tissues. Ice slows the inflammatory process, decreases pain and muscle spasm; compression and elevation reduce oedema. Crutches are also recommended. When the inflammation subsides, passive range of motion (ROM) and gentle mobilisation should be initiated to maintain soft-tissue and joint integrity. Submaximal isometric muscle sets may be used at multiple angles to maintain strength and keep the developing scar tissue mobile. Aggressive stretching and strengthening should be avoided. Electrical stimulation and pulsed ultrasound should be used during both the inflammatory and repair phases to reduce pain and oedema.





Flexibility, strength, endurance and proprioception should be assessed pre-season



Repair phase

The inflammatory and repair phases overlap during the first week after injury. An early accelerated rehabilitation programme may prolong the inflammatory phase and lead to chronic muscle strain. When collagen is formed, it must be appropriately stressed in the normal lines of tension. Signs of inflammation (pain, swelling, redness, warmth) are signs of tissue overstress and allow assessment of the rehabilitation programme. Frequency, intensity and duration of exercises are altered to allow for healing and to prevent inflammation for the next 1 to 2 weeks. Cold may be beneficial initially to allow for pain-free exercise and aid in the formation of the scar tissue. Gentle, pain-free stretching and pain-free submaximal isometrics can be incorporated into contract-relax techniques to help align collagen fibres. A cardiovascular conditioning programme should be incorporated for any athlete not capable of full athletic participation.

Maturation and remodelling phase

When collagen is mature, tension should be applied in the line of normal stresses for proper remodelling. This stage presents at about 2 to 3 weeks after injury and is characterised by:

1. The absence of inflammation.
2. Full, pain-free ROM.
3. Pain after tissue resistance (passive ROM).

The athlete is progressed as tolerated with limited participation in his/her sport. More vigorous stretching, closed- and open-chain strengthening, cardiovascular training and

sport-specific activities are allowed. Muscles must be stressed and overloaded in the manner in which they are used functionally, following the principle of specificity. Specifically, type of contraction (eccentric vs concentric), metabolism (aerobic vs anaerobic) and functional pattern (diagonal vs cardinal plane) of the muscle should be respected. Eccentric exercise is functional in most athletic activities, develops greater tension than concentric exercise and may be more comfortable in the early stages of rehabilitation¹¹.

Proprioceptive and endurance training are used in the advanced stages of rehabilitation. After the athlete has regained full, pain-free active ROM and over 90% strength bilaterally, full participation is allowed. Maintenance programmes should be continued to avoid any dysfunctional adaptation or compensation.

PREVENTION OF ACUTE STRAINS

Prevention of acute muscular strains implies adequate pre-season screening of flexibility and strength balances in major joints (knee, shoulder and ankle). Flexibility, strength, endurance and proprioception should be also assessed. Adequate agonist/antagonist ratios for strength and flexibility should be attained for major muscle groups and muscles must be strengthened in the mode in which they are used functionally. Warm-up and stretching before activity are recommended. Specifically, active warm-up such as jogging or biking should be helpful before specific muscle stretching, especially in two joint muscles at high risk for strain, muscles with high percentages of fast-

twitch fibres (hamstrings, gastrocnemius, quadriceps, biceps) and those with high incidence of strain (hip flexors, hip adductors, erector spinae, rotator cuff). Muscles which contract eccentrically or decelerate in functional high-speed activities such as the posterior rotator cuff in throwing athletes or the hamstrings in sprinters should be stretched for 15 to 20 seconds and repeated four times²¹.

NEW FRONTIERS OF CLASSIFICATION

Mueller-Wohlfahrt et al have recently highlighted the need for a more standardised definition and universal classification that reflects both functional and structural features of muscle injuries²². Our belief is that the term 'functional' is not well-defined and is used with various ambiguous and at times inconsistent meanings. Both functional and structural disorders may lead to functional limitations in athletes and these latter may also hide misunderstood structural changes of the muscle. Therefore, from a diagnostic viewpoint, both the terminology and the ability to distinguish these two entities are challenging. For structural changes, Mueller-Wohlfahrt et al suggest that the term 'tear' better reflects the structure of the muscle, recommending that the term 'strain' not be used. The latter, conversely, implies the biomechanics of the injury²². The term 'strain' reflects some radiological features detectable on MRI and US. A strain is a grade I injury, in which less than 5% of muscle fibres are disrupted, with a feathery oedema-like pattern and intramuscular high signal on the fluid-sensitive sequences at MRI. This condition

is well differentiated from grade II (partial tear) and III (complete tear) lesions²³. In addition, it is unlikely that MRI is sensitive enough to detect the presence of microscopic disruptions which may be decisive in differentiating 'functional' from 'structural' muscle injuries. We have proposed a recent anatomic classification of acute muscles strain injuries, suggesting that the proper identification and description of the injury site could be prognostic for muscle recovery. We classified the lesion as type I, when involving the proximal MTJ, type 2, for muscle belly injuries and type 3, when the distal MTJ is torn. Considering the anatomy, muscular lesions can be further classified as:

- intramuscular,
- myofascial,
- myofascial/perifascial,
- musculotendinous or
- a combination²³.

The severity of the muscular and musculotendinous injuries is classified according to a 3-grade classification system from MRI and US^{4,24}.

CONCLUSION

Clinical assessment, site of injury and pathophysiology can all provide prognostic information regarding rehabilitation and recovery time after an acute muscle strain injury. The classification system outlined in this article can be a valuable tool when applied in clinical practice. However, thorough research and investigation should be executed to assess appropriateness for practitioners before the system is adapted. Similarly, the anatomical system we propose must be assessed in different muscles to determine its more general applicability.

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