Muscle Soreness and Delayed-Onset Muscle Soreness

Paul B. Lewis, MD, MS\textsuperscript{a,*}, Deana Ruby, APN, ACNP-BC\textsuperscript{b}, Charles A. Bush-Joseph, MD\textsuperscript{b}

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- Muscle soreness
- DOMS
- Delayed-onset
- Muscle ache
- Stiffness

The novice and elite athlete is familiar with the postexercise muscle discomfort known as delayed-onset muscle soreness (DOMS) after unfamiliar exercises. While common in occurrence, most patients will self-treat the condition unless symptoms are progressive in nature. The sports medicine clinician needs to maintain this diagnosis among their active differential diagnoses. Associated symptomology of muscle soreness can be quite debilitating and the presentation of this phenomenon is as diverse as the population that experiences it.

Immediate or delayed-onset muscle soreness with a nonuniform intramuscular distribution may portray itself as a nonmuscular injury with an unrecalled or vague traumatic event. The prudent clinician is to base their advisory guidance, medical management and/or surgical treatments on sound medical and/or surgical principles.

The purpose of this communication is to describe the clinical presentation, cellular mechanisms, preventative measures, and management options related to muscle soreness and DOMS for the sports medicine clinician.

CLINICAL PRESENTATION

Muscle soreness is classified as a type I muscle strain\textsuperscript{1} and refers to the immediate soreness perceived by the athlete while or immediately after participating in exercises. Muscle soreness presents with muscle stiffness, aching pain, and/or muscular tenderness. These symptoms are experienced for only hours and are relatively transient compared to those of DOMS. The symptomatology of DOMS shares similar

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\textsuperscript{a} Department of Diagnostic Radiology & Nuclear Medicine, Rush University Medical Center, 1653 West Congress Parkway, Chicago, IL 60612, USA
\textsuperscript{b} Department of Orthopedic Surgery, Rush University Medical Center, Third Floor, 1611 West Harrison Street, Chicago, IL 60612, USA
\textsuperscript{*} Corresponding author.
E-mail address: paul_lewis@rsh.net

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quality and intensity to that of immediate exercise-induced muscle soreness but symptom onset is at about 24 hours after the athlete has completed their exercise.\(^2,3\) In the ensuing days, the related symptoms peak within 72 hours and slowly resolve in 5 to 7 days.\(^2,3\) While the presentation of these conditions differs temporally, there is a shared subtly that may lead one to the clinical question of more concerning pain generators (eg, stress fracture, ligamentous sprain, or tendon tear).

Muscle soreness may begin as a concerted area sensitive to passive manipulation and active movement.\(^4,5\) This early perception may later be perceived as broad muscle soreness with focal points of tenderness referred from the active process within the musculotendinous junction.\(^6\) This latter presentation is more ambiguous, and accordingly more concerning. The clinical responsibility is to discern DOMS from a non-muscular injury with an unrecalled or vague traumatic event (eg, ligamentous rupture, chondral defect, stress fracture). To do so, it requires an understanding of the characteristics of DOMS and competing diagnoses.

Clinically, focal exercise-induced soreness can be separated from a nonmuscular etiology with reproducible active and passive, weight-bearing and non–weight-bearing range of motion. Moreover, the clinician can use specific tests to the possible surrounding nonmuscular etiologies. Expanding the diagnostic thought process to include recent changes in workout intensity, exercise duration, and the individual’s baseline exercise tolerance will further direct one toward or away from an etiology. Finally, consideration of new activities the patient may have performed is warranted, particularly in patients who were already well conditioned.

Independent of the patient’s previous condition, there is an associated muscle weakness after an episode of acute or delayed muscular soreness.\(^5,7–11\) This decline in muscular performance is intuitively related to the associated cell damage and subsequent inflammatory response.\(^12–14\) The identified cellular damage related to unaccustomed exercise is loss of membrane integrity\(^15,16\) and excitation-contraction coupling.\(^12,14\) Restoration of muscle strength from the causative exercise may take up to 2 weeks to occur.\(^4\) The delay in recovery has been attributed to the inflammatory cell infiltration and accumulation.\(^61,63\) Clinically, the strength deficit and its duration deserve to be included when advising patients whom are considering beginning or increasing their exercise program.

Concurrently, as focus turns to “return to play” during or after the resolution of muscle soreness, the clinician is to remain grounded with injury risk factors that include but are not limited to subtle deficiencies in joint stabilization and cushioning,\(^17\) gross coordination,\(^17,18\) and, as above, strength.\(^12–14\)

**CELLULAR MECHANISMS**

Paralleling the diverse population and clinical presentation of muscle soreness, there are 6 competing theories for the mechanism of DOMS: lactic acid accumulation,\(^19,20\) muscle spasm,\(^21,22\) microtrauma,\(^23–25\) connective tissue damage,\(^25\) inflammation,\(^5,8,26\) and electrolytes and enzyme efflux.\(^19,27,28\) While these 6 theories were presented independently from one another, the current consensus is that a single theory alone is insufficient to explain the process; instead, they work in concert.\(^19,20,29–31\)

The mechanism of muscle soreness, immediate and delayed onset, begins at the time of exercise with strain on the muscle’s functional unit—the sarcomere. Subsequent to breakdown of the functional unit, there is an intracellular accumulation of calcium, causing further degradation of the sarcomere. This intracellular damage increases the demands on surrounding connective tissue. Following this is an inflammatory response with recruitment of inflammatory cells and cytokines that
potentiate the nerve endings and perception of pain. Passive manipulation and active movement alter intramuscular pressures and stimulate mechanoreceptor nerve endings, contributing to the perception of soreness.\textsuperscript{19,20,29–31}

Explanation of disproportionate (nonuniform) exercise-induced soreness begins with the intramuscular architecture.\textsuperscript{32–34} Each imposed stress on muscles (eg, concentric versus eccentric actions) recruits different branches of a structural, intramuscular organization. Demands on selected parts within muscle develop selective pathways for electrical activity, hypertrophy, and generation of forces.\textsuperscript{35–41} The gross manifestation of this intramuscular architecture is the orientation and shape of muscle. Had such intramuscular architecture not existed, the gastrocnemius would be a homogeneous, uniform cylinder of muscle tissue between the tendons. The demanded contractions during unaccustomed exercises may overload underdeveloped muscle fibers and lead to cellular damage.\textsuperscript{42–44} Subsequent inflammation remains specific to those posits of underdeveloped myocytes within that architecture.\textsuperscript{5,9,26} Ultimately, this translates into focal points of tenderness overlying typical locations of long bone stress fractures or superficial to a ligament’s site of insertion.

**PREVENTIVE MEASURES**

Effective prevention of muscle soreness is difficult; it is a physiologic response to activity. The most effective prophylaxis of muscle soreness would be abstaining from prolonged, intense unfamiliar physical exercises. Identifying such activities before participating often carries a commensurate degree of difficulty. When such tasks are identified or anticipated however, there are inherent modalities—physical preparation, demand reduction and nutritional resources—can minimize anticipated muscle soreness.

Prevention of muscle soreness through stretching is supported by the rationale of viscoelastic and stress-relaxation behaviors of muscle.\textsuperscript{20} The benefit of stretching, however, is marginal.\textsuperscript{45,46} The 2011 Cochrane Database review by Herbert and colleagues\textsuperscript{46} found the reduction of DOMS is maximized with stretching before and after activity.

Use of assistive devices may alleviate the demand on muscle fibers and decrease subsequent soreness.\textsuperscript{47} When desired to maintain muscle demand and recruitment, the athlete is to be advised regarding nutritional supplements.

Carbohydrate and protein supplement drinks are seen to be most beneficial when consumed after, not prior to, muscle-damaging exercise activity.\textsuperscript{48,49} In a sophisticated, double-blind study by Matsumoto and colleagues,\textsuperscript{50} protein supplement surpassed the placebo drink in reducing muscle soreness and fatigue after prolonged physical exercise.

The use of supplements, assistive devices, and stretching is to be done cautiously and with a confirmation of understanding from the athlete on instructions. Additional preventative measures are also included next because of the crossover between prevention and symptom management.

**SYMPTOM MANAGEMENT**

Completing the physiological mechanism of muscle soreness is the only effective treatment. Each clinician is to approach the most compatible options for the athlete with a sound understanding of existing basic science that supports or refutes the selected modalities. The primary responsibility of the clinician is to prevent the athlete from injuring himself or herself with the chosen management(s).

The outcomes from clinical research on massage are too variable\textsuperscript{28,51–55} to confidently support its gainful benefit; its use should be directed empirically by athlete
perception. As suggested by Cheung and colleagues, the variability of outcomes from massage are likely related to the variations in timing and methods of the studied tissue massage. Additionally, it is unclear if massage increases local blood flow to affected muscle.

Recommendations of cryotherapy, vibration treatment, and nutriceutics (eg, pomegranate juice) carry the same degree of neutrality. Sample sizes, scheduling, and treatment administration have limited the value of studies of such methods.

The rationale and outcomes of pharmaceutical intervention with nonsteroidal anti-inflammatory drugs (NSAIDs) are supported consistently and thus are recommended when provided with a reasonable guidance on the dangers of their use. Clinically, NSAIDs decrease perceived muscle soreness associated with DOMS but fail to impact the length or degree of muscle weakness.

Further research is needed but supported in the use of mechanical lower extremity compression as it has been shown to reduce swelling and decrease perceived muscle soreness. The same study by Kraemer and colleagues showed continuous compression also allowed the maintenance of elbow range of motion and promotion of strength reconditioning.

The most effective, and highly recommended, modality to treat muscle soreness is continued exercise. The basic principle supporting exercise is the increase in local blood flow and endorphin release it produces and its subsequent analgesic effects. While effective, the athlete is more likely to cause muscular injury while exercising and benefits diminish at the cessation of activity.

Surgical intervention is not recommended as a prophylactic or treatment modality.

**SUMMARY**

Immediate and delayed-onset muscle soreness differ mainly in chronology of presentation. Both conditions share the same quality of pain, eliciting and relieving activities and a varying degree of functional deficits. There is no single mechanism for muscle soreness; instead, it is a culmination of different mechanisms. The developing pathway of DOMS begins with microtrauma to muscles and then surrounding connective tissues. Microtrauma is then followed by an inflammatory process and subsequent shifts of fluid and electrolytes. Throughout the progression of these events, muscle spasms may be present, exacerbating the overall condition.

There are a multitude of modalities to manage the associated symptoms of immediate soreness and DOMS. Outcomes of each modality seem to be as diverse as the modalities themselves. The judicious use of NSAIDs and continued exercise are suggested to be the most reliable methods and recommended. This review article and each study cited, however, represent just one part of the clinician’s decision-making process. Careful affirmation of temporary deficits from muscle soreness is not to be taken lightly, nor is the advisement and medical management of muscle soreness prescribed by the clinician.

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