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REVIEW

Delayed onset muscle soreness: No pain, no gain? The truth behind this adage

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Abstract

The purpose of this article is to provide brief insight into delayed onset muscle soreness (DOMS), a phenomenon that is often experienced by recreational and elite athletes. The negative implications of DOMS include pain, decreased motivation to continue training, and decreased performance. While performance issues may be more relevant to the elite athlete, pain and decreased motivation are particularly relevant to recreational athletes wishing to sustain a regular level of physical activity. The article is aimed at general practitioners (GPs) who may encounter athletes presenting with DOMS, and who will benefit from understanding the proposed mechanisms, signs and symptoms of the condition. Numerous researchers have hypothesised that certain interventions may prevent or minimise the symptoms thereof, and all GPs could benefit from understanding the available options for athletes, and the scientific evidence that supports these options.

Keywords: delayed onset muscle soreness, mechanism, symptoms, treatment, athletes, management

Introduction

Delayed onset muscle soreness (DOMS) is muscle pain and stiffness that develops 24-72 hours after exercise involving unaccustomed muscle loading[1] (Table I). It is classified as a type of exercise-induced muscle damage, but is different to muscle fatigue or pain that develops during or immediately after exercise. While the aetiology and exact mechanisms remain unknown, most research acknowledges that DOMS is initiated by eccentric exercise.[1-5] This has been demonstrated in a number of studies that have tested the relationship between muscle pain and different types of eccentric, concentric and static activities. Since it was first described, many theories have been proposed to explain the mechanisms of DOMS, including lactic acid build-up, muscle spasm, damage to connective tissue, mechanical muscle damage, cellular infammation and enzyme efflux theories.[5] It is most likely that the best explanation and understanding of DOMS derives from a combination of two or more of these theories.[5] A variety of treatment modalities have been tested, with post-exercise nonsteroidal anti-inflammatory drugs (NSAIDs), massage and light aerobic activity showing limited success in reducing the symptoms.[1,4-6] The DOMS phenomenon, which affects both elite and recreational athletes, remains topical as researchers continue to refine aetiological models and investigate effective preventative strategies and treatment modalities.

Table I: Delayed onset muscle soreness explained[1]

Examples of common activities known to cause delayed onset muscle soreness include:
- Eccentric strength-training exercises
- Walking or jogging downhill
- Step aerobics.

Causes of muscle pain associated with delayed onset muscle soreness include:
- Structural damage to the muscle fibre and plasmalemma
- An inflammatory reaction.

Prevention is achieved with:
- Graduated intensity and duration of novel activities and eccentric exercise over a period of 1-2 weeks.

Treatment strategies include:
- Nonsteroidal anti-inflammatory drugs.
- Massage administered shortly after the eccentric exercise.
- Light aerobic exercise.
- Nutritional supplementation (antioxidants and L-carnitine) shows promise, but needs to be investigated further in order for the optimal dosage and timing of intake to be defined.

Proposed mechanisms

Research suggests that eccentric muscle activity is the trigger activity for DOMS.[3,5,7] Eccentric activities are those which cause the muscles to lengthen while contracting.[7] Examples include lowering a dumbbell or performing a calf press off a ledge.[4] Mechanical stress is greater in eccentric exercise, compared to concentric exercise, as this type of activity recruits fewer muscle fibres, which results in a greater mechanical load per fibre and a higher propensity for cellular damage.[8] Although the exact cellular mechanisms involved in DOMS are unknown, a model was proposed by Armstrong in 1984,[3] and has continued to be validated and refined by researchers in more recent years.[8,9]

The proposed cellular mechanisms occur as follows:

- High tension in the muscle fibre results in microtrauma.

- Structural damage of the cell membrane disrupts calcium homeostasis, causing necrosis, that peaks 48 hours post exercise.
- Intracellular contents, such as histamine, kinins and potassium, and the products of the inflammatory process accumulate in interstitium, stimulating the free nerve endings, which results in the pain associated with DOMS.[3,8,9]

Histology and biochemistry

Biopsies taken from the affected muscles show structural damage to the muscle fibres at the Z-line (Z-line streaming), as well as leukocyte infiltration, mast cell degranulation and increased plasma substrates in the extracellular space.[10,11] The neutrophil count was shown to peak at six hours, while the monocyte count decreased 48 hours post exercise in studies that investigated changes in serum haematology and biochemistry.[11] A significant correlation was found between DOMS and elevated myoglobin and creatinine kinase 24-96 hours post exercise.[3,7,10]

Diagnosis

DOMS may occur in recreational athletes embarking on a new training programme, in elite athletes at the beginning of a new season, or as a result of repetitive, high-intensity contractions.[5,12] Patients typically present with complaints of dull, aching pain in the affected muscle, often combined with tenderness, stiffness and the loss of strength.[13] Pain is not felt at rest, but rather when the affected muscle is activated by either being stretched, contracted or placed under pressure.[13] The initial symptoms usually start at the musculotendinous junction, and thereafter spread throughout the rest of the muscle.[13] The severity of the symptoms depends on the duration, intensity and type of exercise performed.[5,9] The symptoms usually increase in intensity in the first 24 hours after exercise, peak between 24 and 72 hours, then subside without intervention 5-7 days after exercise.[13] Other than pain, DOMS also causes a reduction in range of motion, shock attenuation and peak torque.[5] This reduced function adversely affects athletic performance and causes distress to many athletes. The diagnosis of DOMS is made clinically, based on the patient history and symptoms. It seldom requires special investigations unless complications of rhabdomyolysis are suspected. Practitioners should have a high index of suspicion of complications in athletes presenting with persistent muscle pain, weakness and myoglobinurea (cola-coloured urine).[14] Biochemistry often demonstrates a raised plasma creatine kinase and myoglobinemia, and raised aspartate aminotransferase, hyperkalaemia and hypocalcaemia in some case reports. These athletes may need admission for intravenous hydration and monitoring.[14]

Management

A number of treatment strategies have been tested to decrease severity, improve muscle function and expedite return to play.[5] Although numerous practices exist for the management of DOMS, few are based on scientific evidence.[6] Interestingly, muscle activity is the best known treatment for DOMS.[3,5] Endorphins released in the body during exercise increase the pain threshold and pain tolerance. However, this analgesic effect is temporary and the discomfort may return following exercise.[3,5] Athletes who train daily should be encouraged to decrease the intensity of their training for 1-2 days following DOMS-induced activity, or alternatively participate in cross-training that targets other unaffected muscle groups so that the affected muscle can recover.[5] Massage administered shortly after exercise has been shown to decrease the amount of pain and stiffness felt.[15] Despite a reported improvement in the analgesic symptoms, massage does not have an effect on muscle function or enzyme activity in the damaged muscles.[15]

Some evidence supports the use of NSAID medication, although the effects have been shown to be dependent on the dose and timing of ingestion.[5] NSAIDs work by inhibiting cyclo-oxegenase (COX) enzymes, suppressing prostaglandin production.[9] A theoretical risk exists that NSAIDs may impair the adaptive response to exercise as COX activity and prostanoid-mediated signalling are key processes involved in achieving maximum skeletal muscle hypertrophy in response to functional overload.[9] Current evidence suggests that the occasional short-term use of NSAIDs does not negatively effect muscle growth, while the effects of chronic use need further investigation.[9] In a comprehensive literature review in which the role of nutritional supplements in the prevention and treatment of exercise-induced muscle damage was investigated, Bloomer found evidence which supported the use of certain supplements (vitamin C, vitamin E, flavonoids and L-carnitine) in DOMS. However, the optimal "prophylactic" and treatment dosage has yet to be defined.[16] While supplementation may have shown promise in attentuating some signs and symptoms of DOMS, it did not eliminate muscle injury.[16] Studies conducted in this area are limited in number and quality, and further research is needed before the use of nutrients can be recommended with absolute confidence when treating DOMS.[16]

Modalities, such as stretching, warming up, cryotherapy, homeopathy and electrical current treatments, have been repeatedly tested, but have failed to demonstrate efficacy in alleviating the symptoms or severity of DOMS.[1,5,6] There are conflicting results with respect to hydrotherapy. A reduction in functional deficit after cold water immersion and contrast water therapy has been reported in some studies,[12] while little or no evidence to support hydrotherapy has been found in others.[6]

DOMS can be prevented or reduced by gradually increasing the intensity of a new exercise programme over 1-2 weeks.[5] The symptoms usually resolve within 3-7 days if no active intervention is introduced.[13]

Conclusion

DOMS has an impact on both recreational and elite athletes, resulting in pain and functional limitation, which adversely affect athletic performance. Numerous studies have been conducted that have investigated the mechanisms and management of DOMS. However, definitive models that outline aetiology and treatment are yet to be established. As athletes respond differently to treatment, a combination of strategies should

be employed when managing this condition. There is scope for further research to clearly define structured protocols for treatment intervention and preventative strategies.

Declaration

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Conflict of interest

The authors declare that there were no competing interests.

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