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Review

Update on the efficacy of extracorporeal shockwave treatment for myofascial pain syndrome and fibromyalgia

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HIGHLIGHTS

- We present the current knowledge on shockwave treatments for myofascial pain syndrome.
- ESWT is an efficient tool for the treatment of myofascial pain síndrome.
- The clinical efficacy of ESWT in fibromyalgia is controversial.
- Promising results have been reported on myofascial pain syndrome.

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ABSTRACT

Chronic muscle pain syndrome is one of the main causes of musculoskeletal pathologies requiring treatment. Many terms have been used in the past to describe painful muscular syndromes in the absence of evident local nociception such as myogelosis, muscle hardening, myalgia, muscular rheumatism, fibrositis or myofascial trigger point with or without referred pain. If it persists over six months or more, it often becomes therapy resistant and frequently results in chronic generalized pain, characterized by a high degree of subjective suffering.

Myofascial pain syndrome (MPS) is defined as a series of sensory, motor, and autonomic symptoms caused by a stiffness of the muscle, caused by hyperirritable nodules in musculoskeletal fibers, known as myofascial trigger points (MTP), and fascial constrictions.

Fibromyalgia (FM) is a chronic condition that involves both central and peripheral sensitization and for which no curative treatment is available at the present time. Fibromyalgia shares some of the features of MPS, such as hyperirritability.

Many treatments options have been described for muscle pain syndrome, with differing evidence of efficacy. Extracorporeal Shockwave Treatment (ESWT) offers a new and promising treatment for muscular disorders.

We will review the existing bibliography on the evidence of the efficacy of ESWT for MPS, paying particular attention to MTP (Myofascial Trigger Point) and Fibromyalgia (FM).

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1. Extracorporeal shockwave treatment and myofascial pain syndrome

Myofascial Pain Syndrome (MPS) is a musculoskeletal disorder with local pain and stiffness, characterized by the presence of hyperirritable palpable nodules in the skeletal muscle fibers, known as myofascial trigger points (MTP) [1–6]. (Fig. 1). MTP produce pain with any activating stimulus (direct or indirect trauma), causing local and referred pain, tenderness, motor dysfunction, autonomic phenomena and hyperexcitability of the central nervous system [7–9]. Recent research argues that MPS offers a simplistic explanatory model, which posits a local (muscular) origin of nociception within the trigger points (TP) and advocates local treatment.

MPS is a common disorder (12% in general population) [10]. Some studies observed an incidence of 30% MTPs in internal

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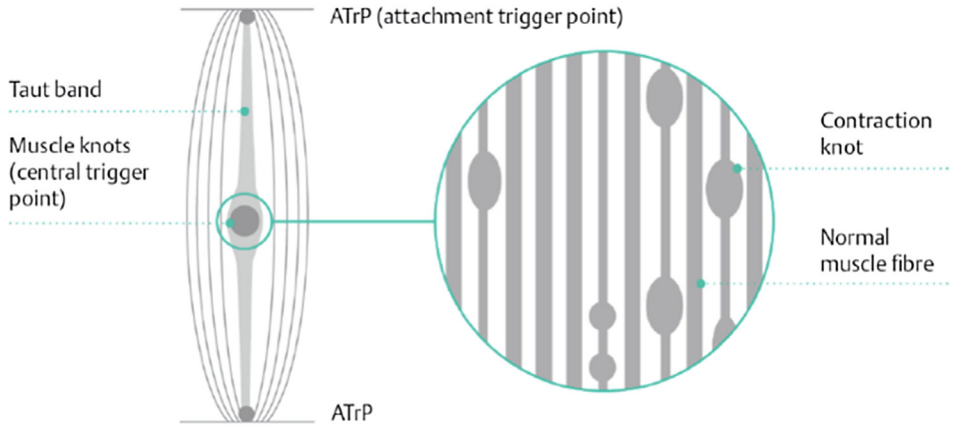

Fig. 1. Myofascial Trigger Point complex: Muscle knot (central trigger point) with taut band and attachment TP [1,2].

medicine practice [11], with a prevalence of 54% in women and 45% in men [2,5,12], although some publications do not report significant differences between the two genders [2,7]. The most common age bracket at onset is 27–50 [8,9].

The **mechanism of action** that best explains it is Simons' Integrated Hypothesis of TP Formation (or Energy Crisis Integrated Hypothesis) [1,2]. Fig. 2.

Muscle trauma, strain, repetitive low-intensity muscle overload, or intense muscle contractions may give raise to a vicious circle which ends up damaging the sarcoplasmic reticulum, and leading to an increase of the calcium concentration, a shortening of the actin and myosin filaments, a shortage of adenosine triphosphate and an impaired calcium pump [2,13]. The "energy crisis hypothesis" reflects this vicious circle. This hypothesis has evolved into the "integrated TP hypothesis" [14], which postulates that abnormal depolarization of the post-junctional membrane of the motor endplates causes a localized hypoxic energy crisis, associated with sensory and autonomic reflex arcs that are sustained by complex sensitization mechanisms.

Muscular injury causes a dysfunction of the neuromuscular endplate, which in turn increases the release of Acetylcholine (ACh) in the synaptic gap. This triggers high-frequency miniature endplate potentials, causing permanent depolarization. These endplate potentials can be experimentally revealed as spontaneous electrical activity (SEA) by needle electromyography [2]. The release of ACh in turn increases the release of calcium at the

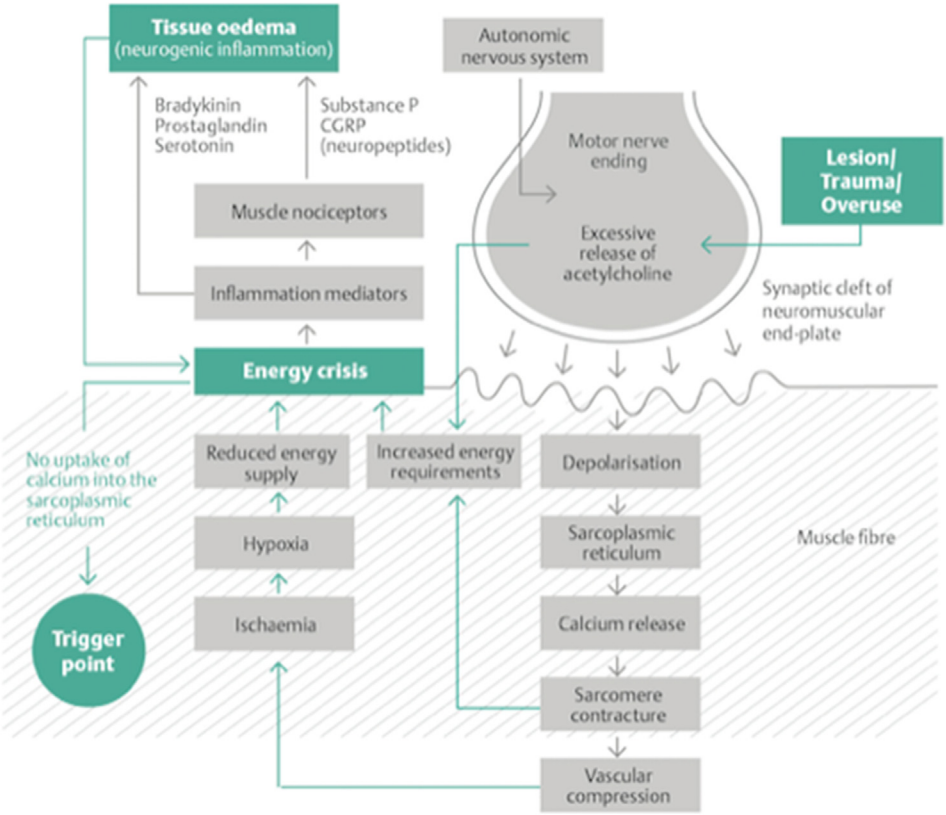

Fig. 2. Integrated myofascial trigger point (MTP) hypothesis [1,2].

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