Ain-Shams University
Faculty of Medicine
Anesthesia, Intensive Care and
Pain Department

Updates of Chronic Muscle Pain

Essay
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By

Dr. Ibrahim Mamdouh Esmat

MB.B.Ch, MSc, M.D.
Lecturer of Anesthesia
Faculty of Medicine –Ain-Shams University

Under Supervision of

Prof. Dr. Ayman Moktar Kamaly

Professor of Anesthesia, Intensive Care and Pain Management
Faculty of Medicine –Ain-Shams University

Dr. Amr M. AbdElfattah

Ass .Prof. of Anesthesia, Intensive Care and Pain Management
Faculty of Medicine-Ain-Shams University

Faculty of Medicine
Ain-Shams University
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بسم الله الرحمن الرحيم

وقُلِ اعْمَلُوا فَسَيَرَى اللَّهُ عَمَلَكُمْ
ورَسُولُهُ والْمُؤْمِنُونَ

صدق الله العظيم

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List of Abbreviations

Ach	:	Acetylcholine
ADP	:	Adenosine diphosphate
ATP	:	Adenosine triphosphate
BMI	:	Body mass index
COX-2 inhibitors:		Cyclooxygenase -2 inhibitors
ESR	:	Erythrocyte sedimentation rate
FM	:	Fibromyalgia syndrome
MEPPs	:	Miniature end plate potentials
MPS	:	Myofascial Pain Syndrome
MRI	:	Magnetic Resonance Imaging
NGF	:	Nerve growth factor
NMDA receptor	:	N- methyl- D- aspartate receptor
NMR	:	Nuclear magnetic resonance
NSAIDs	:	Nonsteroidal anti-inflammatory drugs
RA	:	Rheumatoid Arthritis
RF	:	Rheumatoid factor
SLE	:	Systemic Lupus Erythematosus
TrPs	:	Trigger points

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Introduction:

Myofascial pain syndrome (MPS) is one of the causes of chronic muscle pain. MPS is more common in patients with chronic tension-type headache, tempro-mandibular disorders and pain the face-jaw region and in post-whiplash syndrome. It is more common in women than in men. Bio-mechanical and soft tissue pathologies are more common in older adults with chronic low back pain than in pain-free patients with myofascial pain ***(Couppe et al., 2007).***

Fibromyalgia (FM) is another cause of chronic muscle pain. It is estimated that 2-4% of the general population suffers from FM, making it the second most common rheumatic disorder behind osteoarthritis. Symptoms of FM are more common in women than in men ***(Bennett et al., 2007)***.

Aim of The Work:

The aim of this work will be to highlight the updates in management of myofascial pain syndrome and fibromyalgia.

Muscle Pain:

Muscle pain is most commonly caused by tension, overuse or muscle injury from exercise or over-work. The pain in such cases tends to involve specific muscles and starts during or just after the activity. Muscle pain also may be due to systemic diseases e.g. flu like- infections and connective tissue diseases. Myofascial pain and fibromyalgia syndromes are two common causes of chronic muscle pain.

(Basford and An, 2009)

Myofascial Pain Syndrome (MPS)

Muscle Anatomy:

The structure and activity of a motor unit must be understood to recognize a disruption of normal function. This disruption or pathology is the cause of myofascial pain and dysfunction. Each muscle, in descending order of magnitude, consists of muscle fibers, myofibrils, sarcomeres, and myofilaments (Fig.1). The sarcomeres are the main contractile mechanisms of the muscle. They are made up of thick and thin filaments. The thick filaments are composed of proteins called myosin. The thin filaments are composed of proteins called actin, tropomyosin, and troponin (Fig.2). ***(Anthony, 2013)***

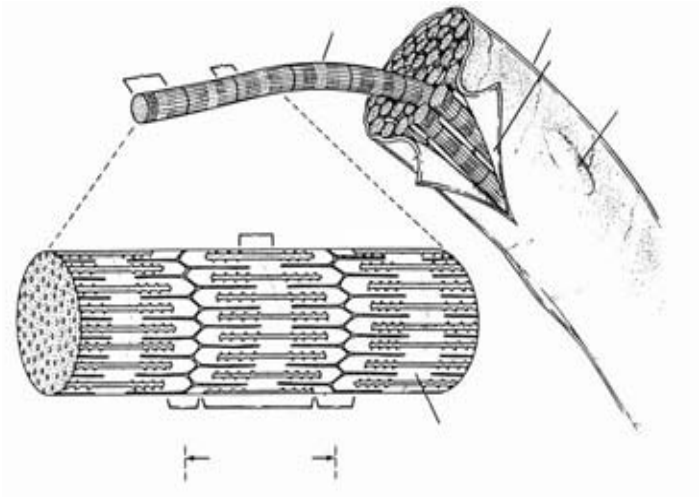

Fig. 1: Anatomy of a muscle fiber. Microscopic anatomy of an individual skeletal muscle fiber (cell). Note the striated (striped) appearance of the muscle fiber and the myofibrils. ***(Anthony, 2013)***

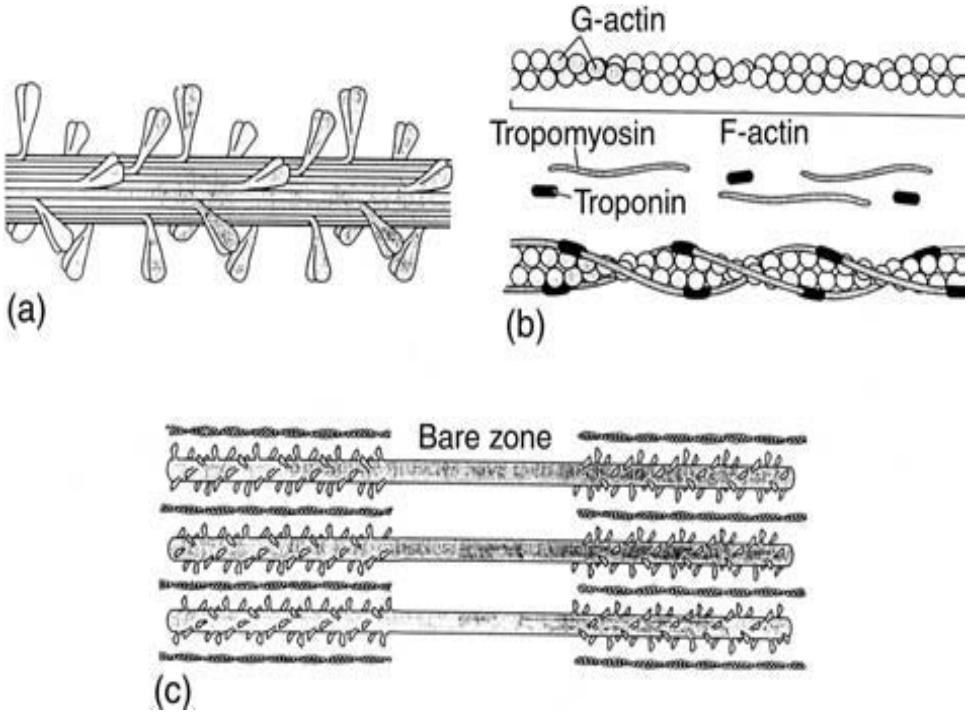

Fig. 2: Myosin and actin filaments. (a) Thick filament, (b) thin filament, (c) longitudinal section of filaments. ***(Anthony, 2013)***

It is the sliding action of these two filaments that actually causes the contraction. The sliding is a result of a series of rowing actions between the projections or heads on the myosin filament and attachment sites on the actin filament. Relaxation occurs when the myosin heads detach from the actin filament. ***(Mense et al., 2001)***

The site where the terminal branch of the alpha-motor axon links with the muscle fiber is called the motor end plate or neuromuscular junction. When an action potential arrives at this junction, the axon releases acetylcholine (ACh). The ACh diffuses across the synapse to the muscle cell membrane and causes a change in the membrane at the junction of the muscle cell. This change results in the generation of a stimulatory impulse that spreads over this plasma membrane and into the interior of the muscle cell by way of t-tubules. This impulse excites the sarcoplasmic reticulum causing it to release calcium ions ($Ca^{2+)}$. This constitutes the initiating event for muscle contraction. ***(Anthony, 2013)***

In the resting muscle, the tropomyosin masks the binding sites (for the myosin heads) on the surface of the actin molecule. The rise in Ca^{2+} concentration causes the tropomyosin chain, which is wrapped around the actin, to move from its blocking position. The myosin heads can then

bind with actin in a rowing motion. This movement pulls the actin filament toward the middle of the sarcomere thereby shortening it. Adenosine triphosphate (ATP) provides the energy required for muscle contraction. In a resting muscle cell, an ATP molecule binds to the projection (club-shaped head) on the myosin molecule. The myosin head also contains ATPase, an enzyme that splits ATP into adenosine diphosphate (ADP) and phosphate (P) plus energy. Actin activates the ATPase in the presence of magnesium (Mg) ions. Consequently, as soon as the tropomyosin chain moves and the myosin heads make contact with the actin filaments, ATP is split. ***(Mense et al., 2001)***

This released energy then separates the myosin heads from the actin. If intracellular Ca^{2+} remains high, the myosin heads will again attach to the actin with the flexing motion. When the concentration of Ca^{2+} drops, the attachments no longer occur and tropomyosin once again blocks the binding sites. The calcium pump returns the Ca^{2+} to the sarcoplasmic reticulum, until ACh spikes again. ***(Mense et al., 2001)***

Anatomy and physiology of a trigger point:

Trigger points (TrPs) have been difficult to understand because there has been no method of studying them. Often, differences in terminology have made it difficult to know if

investigators were even dealing with the same condition. The current understanding of trigger points results from the convergence of two independent lines of investigation, one electrodiagnostic and the other histopathology. Fitting together the lessons from each led Mense and Simons to postulate a theory called the Integrated Hypothesis. It is now becoming clear that the region we are accustomed to calling a TrP, or a tender nodule, is a cluster of numerous microscopic loci of intense abnormality. These loci are scattered throughout the nodule. The critical TrP abnormality now appears to be neuromuscular dysfunction at the motor end plate of an extrafusal skeletal muscle fiber, making myofascial pain caused by TrPs a neuromuscular disease. ***(Mense et al., 2001)***

The primary dysfunction hypothesized here is an abnormal increase (by several orders of magnitude) in the production and release of ACh packets from the motor nerve terminal under resting conditions. The greatly increased number of miniature end plate potentials (MEPPs) produces end plate noise and sustained depolarization of the postjunctional membrane of the muscle fiber. This sustained depolarization could cause a continuous release and inadequate uptake of calcium ions from local sarcoplasmic reticulum and produce sustained shortening (contracture) of sarcomeres. Each of these four italicized changes would increase energy

demand. The sustained muscle fiber shortening compresses local blood vessels, thereby reducing the nutrient and oxygen supplies that normally met the energy demands of this region. The increased energy demand in the face of an impaired energy supply would produce a local energy crisis, which leads to the release of sensitizing substances that could interact with autonomic and sensory (some nociceptive) nerves traversing that region. Subsequent release of neuroactive substances could, in turn, contribute to excessive ACh release from the nerve terminal, completing what then becomes a self-sustaining vicious cycle. (Fig.3). ***(Mense et al., 2001)***

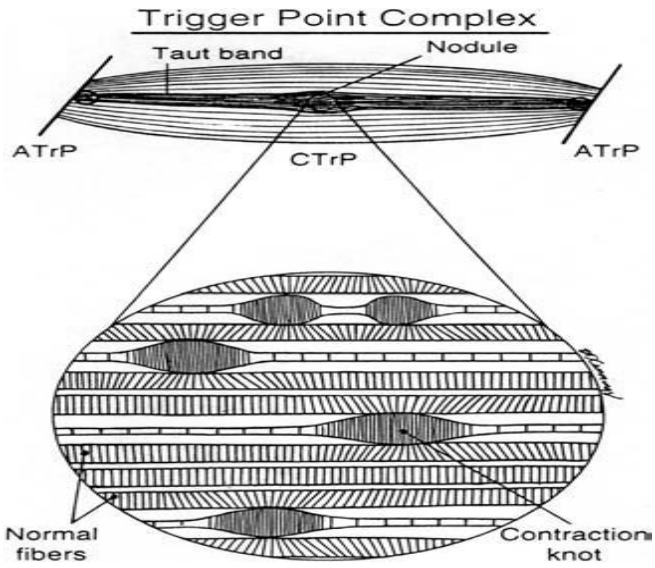

Fig. 3: Sarcomeres with contraction knots. ***(Mense et al., 2001)***

Two types of myofascial trigger points have been identified. The central TrP occurs in the belly of the muscle at

the dysfunctional motor end plate. The attachment trigger points occur where myofascial tissue attaches. The dysfunction at the motor end plate results in a contraction knot, which can produce a palpable nodule, thus the central TrP. The remainder of the sarcomeres is stretched into a taut band of very tense muscle fibers. As the tightened sarcomere pulls on the attachment points, it causes a disruption of these fibers, resulting in tenderness and swelling. ***(Mense et al., 2001)***

Definition of Myofascial Pain (MFP):

The traditional and narrow definition of myofascial pain is that it is pain that arises from trigger points (TRPs) in a muscle. TRPs are small and sensitive areas in a muscle that spontaneously or upon compression cause pain to a distant region, known as the referred pain zone. Tender spots (TSs), in contrast to TRPs, only cause pain locally. Taut bands (TBs) are groups of muscle fibers that are hard and painful on palpation. TB is an objective and consistent palpatory finding in muscles with myofascial pain. Within TB, the most painful and sensitive areas are the TRP and TS. Nowadays, in broader terms, myofascial pain includes muscle pain from TB with TRP and/or TS. The muscles are in spasm, with increased tension and decreased flexibility. It usually presents with regional muscle pain distributed in1 or 2 quadrants of the body. ***(Borg-Stein and Simons, 2002)***

Epidemiology:

MFP is a major cause of musculoskeletal pain. There is a high prevalence of MFP in patients with regional musculoskeletal pain. It is one of the most frequent causes of back pain and neck pain.In a study of 164 patients referred to a pain clinic with chronic head and neck pain of at least 6 months duration, 55% were found to have a primary diagnosis of MFP.In a general medical clinic study, the primary complaint of 30% of patients was due to MFP.The prevalence of MFP pain in pain management centers is higher.In a comprehensive pain centre study on 283 consecutive patients, 2 physicians independently reported MFP as the primary diagnosis in 85% of cases.One physician who examined 96 patients in another pain centre study found MFP to be the primary cause of pain in 74% of cases, and 93% of cases had at least part of their complaint caused by MFP.

(Borg-Stein and Simons, 2002)

Precipitating Factors:

1-Trauma:

A-Macrotrauma:

Contusions, sprains and strains may give rise to MFP acutely.

B-Microtrauma:

The onset is more subtle. Chronic repetitive overloading or overuse of muscles may lead to fatigue and gradual onset of MFP.