When Moving Hurts

Assess Understand Take Action

GLOBAL YEAR AGAINST MUSCULOSKELETAL PAIN

OCTOBER 2009 – OCTOBER 2010

Musculoskeletal Pain

Introduction

Musculoskeletal pain is a known consequence of repetitive strain, overuse, and work-related musculoskeletal disorders. These injuries include a variety of disorders that cause pain in bones, joints, muscles, or surrounding structures. The pain can be acute or chronic, focal or diffuse. Low back pain is the most common example of chronic musculoskeletal pain. Other examples include tendonitis and tendinosis, neuropathies, myalgia, and stress fractures.

Epidemiology and Economics

- Musculoskeletal pain from overuse affects 33% of adults and accounts for 29% of lost workdays due to illness.
- Low back pain is most prevalent and most common work-related injury in Western society, and it is the most costly work-related musculoskeletal disorder.
- While incidence rates for overexertion injury due to lifting are 1.3 times greater in males, rates are higher in females for the following conditions: 3.0 times greater for carpal tunnel syndrome, 2.3 times greater for tendonitis, and 2.0 times greater for injuries caused by repetitive motion.
- The economic burden of musculoskeletal pain is second only to that of cardiovascular disease.

Pathophysiology

The pathophysiology of musculoskeletal pain is not completely clear, but inflammation, fibrosis, tissue degradation, neurotransmitters, and neurosensory disturbances have been implicated.

- *Inflammation:* Injury induces increased pro-inflammatory cytokines and mediators in affected tissues and systemically. This increase leads to peripheral nociceptor sensitization.
- *Fibrosis:* Inflammation can induce fibrotic scarring (i.e., increased collagen within and between cells and tissues), which reduces gliding of tissues during movement, leading to stretch injuries and more pain.
- *Tissue degradation:* Increased inflammatory mediators induce increases in matrix metalloproteinases (enzymes that degrade extracellular matrices), lowering tissue load tolerance and leading to further injury and more pain.
- *Neurotransmitters:* Levels of substance P, calcitonin-related related peptide, and *N*-methyl-D-aspartate (NMDA) are elevated in tendons, dorsal root ganglia, and spinal cord dorsal horns.
- *Neurosensory/neuroimmune factors:* Hypersensitivity, with increased levels of neurotransmitters, inflammatory mediators and cytokines, causes peripheral nociceptor sensitization or central amplification of pain. Hyposensitivity occurs with nerve compression from fibrosis.

Clinical Features

- The pain can be acute or chronic, focal or diffuse, in musculoskeletal or associated neural tissues.
- Clinical symptoms include:
  - local symptoms of pain or widespread and persistent pain
  - tenderness
  - peripheral nerve irritation
  - weakness
  - limited motion and stiffness

- Symptoms progressively increase with greater tissue injury and inflammation, with an increase in affected anatomical sites, i.e., increased tender points.
- Symptoms are exacerbated by work-related or personal stress, for example, poor control over one's work, difficult relationships, and time pressure.
- Nerve conduction velocity decreases in an involved peripheral nerve.
- Symptoms have diurnal fluctuation. At first, symptoms subside with cessation of work (i.e., between shifts, over weekends, and during vacations). As exposure persists and tissue injury progresses, symptoms may be insufficiently alleviated by rest, and constant pain may develop.

Diagnostic Criteria

- Local and then later intermittent or persistent pain in musculoskeletal tissues can be tested using a visual analogue pain scale; disability (e.g., weakness), can be tested using the Roland Morris Disability Questionnaire (RMDQ). These tests are recommended by the Multinational Musculoskeletal Inception Cohort Study.
- The UBMA (upper-body musculoskeletal assessment) instrument developed by Kramer can be used to diagnose upper-extremity musculoskeletal pain and disorders.
- A systemic inflammatory response during the early phase can be confirmed by increased serum C-reactive protein, interleukin-6, or tumor necrosis factor alpha. Increases are associated with increased UBMA scores, chronic low back pain, and pain associated with a range of musculoskeletal disorders.

Diagnosis and Treatment

- Diagnoses include peripheral neuropathies; lateral or medial epicondylitis/tendonitis; rotator cuff, bicipital, or wrist tendonitis; wrist sprain or strain; Achilles tendonitis; myositis and myalgia; osteoarthritis; cervical strain; and lower back pain.
- Management is typically multimodal:
    - Physical therapy, primarily with an exercise program (aerobic, strengthening, stretching), together with physical modalities, such as heat or ice
    - Splinting and/or orthoses
    - Use of nonsteroidal anti-inflammatory drugs (NSAIDs), e.g., ibuprofen
    - Reduction in workload or increased rest
    - Stress management/behavioral intervention
- Unfortunately, recovery from inflammation-induced fibrotic tissue changes is negligible, even with complete cessation of strain/activity for up to 12 months. Thus, the pain resulting from fibrotic scarring is chronic.

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