

Review Article on Pain and Other Compliance of Osteoarthritis

R. Ramani* and Dr.V. Hemavathy

Abstract--- *Current evidence suggests that although persistent arthritic pain is initiated and maintained by articular pathology, it is also heavily influenced by a range of other factors. Strategies for treating arthritic pain are therefore different from those adopted for acute pain. Although published guidelines offer general assistance, the complexity of underlying mechanisms requires that measures designed to relieve pain must take into account individual biological, psychological and societal factors. It follows that a combination of both pharmacological and non-pharmacological approaches offers the best opportunity for therapeutic success, although determining the effectiveness of such complex interventions remains difficult. Pharmacological therapy is often prolonged, and safety and tolerability issues become as important as efficacy over time.*

Keywords--- *Nociceptive, Neuropathic Pain, Neuroplastic Pain, Non-Steroidal Anti-inflammatory Drugs NSAIDs.*

I. INTRODUCTION

Arthritic pain is common and is associated with worse functional outcomes and poorer quality of life when compared with a range of other chronic conditions. A bewildering array of guidelines and other evidence-based resources are available, but the variability of therapeutic responses can lead to frustration and disappointment for both patients and health professionals¹. This review categorizes different pain states associated with arthritis and discusses the extent to which an understanding of underlying mechanisms can be used to inform the choice of analgesic therapy. Although a detailed and systematic evaluation of specific interventions is beyond the scope of the review, evidence for the utility of general approaches is presented. The limitations of current approaches to assessment and management are discussed along with the rationale for use of integrated care in patients with persistent pain.

II. MECHANISMS OF PAIN

Pain Classification

Traditionally, pain has been regarded as being either nociceptive (arising in response to tissue injury) or neuropathic (arising in response to nerve injury). Nociception is the physiologic process by which information about tissue damage is communicated to the central nervous system. Learning about the neurophysiologic mechanisms of pain is necessary to understanding the guidelines for pain assessment and treatment: nociception involves four processes: 1. Transduction 2. Transmission 3. Perception 4. Modulation².

Although this distinction has had some therapeutic utility, it has served to maintain the Cartesian concept of a

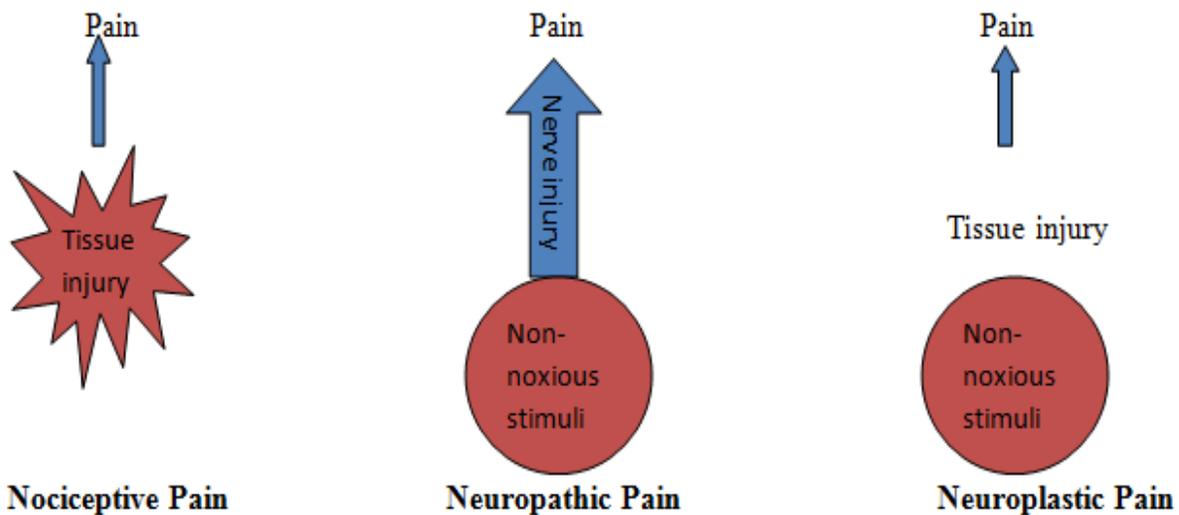
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fixed immutable pain system that faithfully transmits information from a site to pain centers within the brain. Although this largely true after acute injury, it is clear from epidemiological studies that in the presence of persistent disease a range of additional factors, often unrelated to the musculoskeletal system serve to modify activity within pain pathways.

In recent classification the acute and chronic pain are different and functional changes within the nociceptive system are important in determining the signs and symptoms experienced by individual with somatic disease. Four different pain states are recognized.

The first of this nociceptive pain refers to those transient symptoms and signs that arise in response to acute injury and reflects that activation of specialized pain receptors and corresponding activity in more central pathways³.



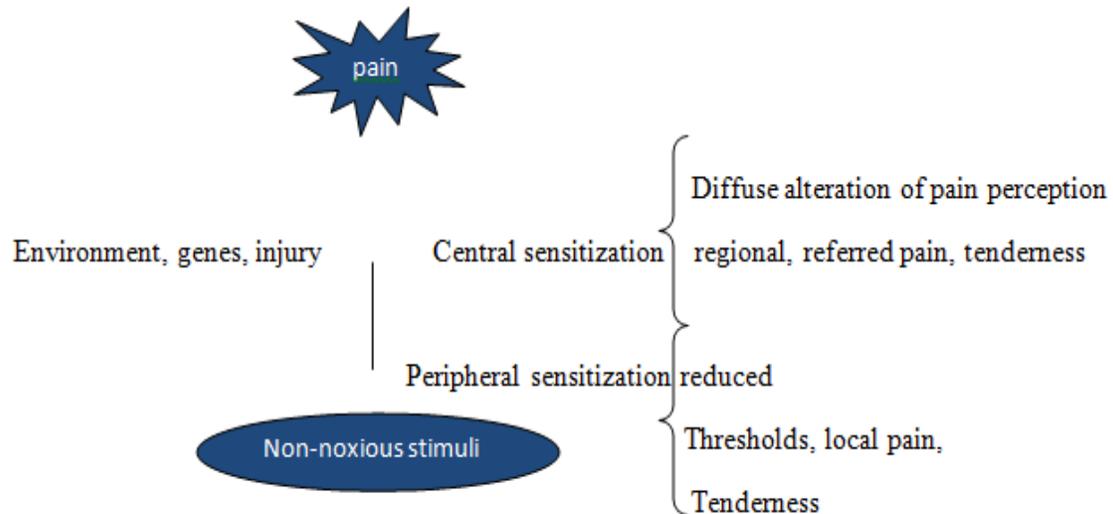
Neuroplastic pain (also called inflammatory pain) occurs in response to more persistent tissue injury and is the most common pain state associated with musculoskeletal disease. It arises as a result of mediators released from damaged tissues acting to increase the excitability of the nociceptive pathway and has the effect of making everyday activities such as standing or walking painful.

Neuropathic pain occurs in the presence of nerve injury, as might occur in association with carpal tunnel syndrome or after lumbar disc prolapsed⁴.

Arthritic Pain

At a local level mediators released from synovium, bone or other tissues will induce the sensitization of articular pain receptors.

The clinical correlate of sensitization at this peripheral level is that musculoskeletal symptoms will be localized with a relatively close relationship to mechanical stimuli such as walking or standing⁵. Treatment with systemic or topical therapies designed to reduce inflammatory mediators might be expected to have a beneficial effect.



General Approaches to Pain Management

Few patients ----- surgical interventions: joint replacement.

Advanced nonsurgical interventions: injections, behavioral modification.

Many patients ----- simple interventions: non steroidal anti inflammatory drugs, other drugs, Physical therapies.

Self-help: simple analgesics, topical agents, and lifestyle

How to Reduce the Pain

1. Arthritis pain. Arthritis is a group of painful and degenerative conditions marked by inflammation in the joints that causes stiffness and pain.
2. Lose weight, Get more exercise, Use hot and cold therapy.
3. Try acupuncture, Use meditation to cope with pain.
4. Include the right fatty acids in your diet.
5. Get a massage

Pharmacological Therapies

Paracetamol (acetaminophen): it is effective in many arthritic conditions and across all age groups. It has been recommended as the oral analgesic choice for mild moderate pain in osteoarthritis and is generally well tolerated in osteoarthritis patients for periods of up to 12 months. In general, paracetamol has a good tolerability profile and overall record, although recently the frequency of use has been reported to be independently associated with a moderate increase in the risk of incident hypertension⁷.

Tramadol

Tramadol is central acting oral analgesic that has a unique dual mechanism of action involving both a weak μ -agonist action as well as inhibition of the reuptake of noradrenalin end serotonin. It has received widespread

approval for use in both moderate and severe pain.

Non-Steroidal Anti Inflammatory Drugs

- NSAIDs have been shown to be highly effective for treating acute pain.
- Opioids: codeine for chronic arthritic pain
- Antidepressants: The ant nociceptive action of antidepressants is independent of their effect on depression and occurs at lower doses and after a shorter duration of treatment. Prescribed for neuropathic pain⁸.
- Anti-cytokine therapies: Cytokines released from immune cells as part of the inflammatory cascade, anti-inflammatory and analgesic effects to be seen.

Additional Approaches

- Topical therapy-NSAID
- Intra articular injections and other local therapies-steroids
- Acupuncture
- Transcutaneous electrical nerve stimulation

Complication/Side Effects

- No steroidal anti-inflammatory drugs(NSAIDs): edema(swelling in the feet), heart burn, stomach upset and ulcer heart attack and stroke⁹
- Corticosteroids: its elevate the blood sugar levels, increased appetite and bone loss.
- Disease modifying antirheumatic drugs(DMARDs): Stomach Upset And Increased Infection
- Biologic agents: redness,swelling,difficulty breathing, nausea, vomiting, weak pulse rate

Other Side Effects

- Acute Liver Failure, Acute Pustular Eruptions On Skin, Decreased Blood Platelets, Decreased Neutrophils A Type Of White Blood Cell, Decreased White Blood Cells¹⁰.
- Deficiency Of Granulocytes A Type Of White Blood Cell, Discolored Spots And Small Elevations Of The Skin, Giant Hives, Hepatitis Caused By Drugs, Inflammation Of Skin Caused By An Allergy, Stevens-Johnson Syndrome, Toxic Epidermal Necrolysis, Vocal Cord Swelling

III. CONCLUSION

Knee OA is a degenerative joint disease and one of the leading causes of disability in the US and worldwide. Although disease pathophysiology is still poorly understood and is under current investigation, it is accepted that knee OA is multifactorial in origin. Multiple risk factors related to the development of knee OA are described as either nonmodifiable or modifiable. Treatment designed for knee OA should be aimed at relieving pain, improving function, and limiting disabilities. It must focus on relieving symptoms and improving quality of life for patients.

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