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ROLE OF PHYSICAL THERAPY IN REDUCING PAIN AND IMPROVING FUNCTION IN OSTEOARTHRITIS: A NARRATIVE REVIEW

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ABSTRACT

Background: *Osteoarthritis (OA) is the most common progressive joint disorder and a leading cause of disability worldwide. Pharmacotherapy for OA alleviate symptoms, but carry potential health risks and do not modify structural progression of the disease*

Purpose: *To integrate existing evidence regarding Physical Therapy interventions such as strengthening exercises, aerobic exercise, flexibility training, Osteoarthritis manual therapy; focusing on clinical effectiveness, dosing, safety, and application in knee, hip, and spine Osteoarthritis.*

Summary: *High-value physical therapy care – including patient education, exercise therapy (covering strength training; neuromuscular control; aerobic exercise; and flexibility exercises), weight management support, and behaviour change – consistently reduces pain and improves function for people with knee or hip osteoarthritis (OA). Adjunct interventions such as manual therapy, taping/bracing, and aquatic therapy can help further enhance outcomes for selected patients. Whether modalities (e.g. transcutaneous electrical nerve stimulation (TENS), ultrasound, heat/cold therapy) provide relief on a short-term basis in individual cases is open to question; but, generally speaking, they are considered secondary to active care. Telerehabilitation and mixed digital programs deliver results that are just as good as those from face-to-face treatment, provided the programs are well-structured and supervised.*

Conclusion: *Physical Therapy as a first-line treatment of OA, offers clinically meaningful short-to medium-term benefits with excellent safety and cost-effectiveness. To guarantee positive outcomes, it is important to ensure compliance and adjust dose along with lifestyle modifications and psychosocial strategies.*

Key words: *Osteoarthritis (OA) Physical therapy (PT) Exercise therapy, Manual therapy, Aquatic exercise, Patient education, Multimodal interventions, Knee osteoarthritis.*

INTRODUCTION

Osteoarthritis (OA) is a chronic, degenerative joint disorder characterized by progressive loss of articular cartilage, subchondral bone remodelling, synovial inflammation, and formation of osteophytes(1). It

results in joint pain, stiffness, swelling, and reduced mobility, mainly affecting weight-bearing joints such as the knee and hip joints(2).OA is the leading cause of disability worldwide. About 7 percent of the world's population is suffering from OA and the prevalence is increasing with advancing age and high obesity rates(1). OA is a significant socioeconomic burden, it is either due to direct healthcare costs including medications, surgeries, rehabilitation or indirect costs from reduced productivity, and loss of independence(1, 3). Quality of life is compromised(1-3). Multiple risk factors for OA are : Non-modifiable including Advancing age, female sex, genetic predisposition, abnormalities in joint shapes and Modifiable which includes obesity, previous joint injury, joint overuse, poor muscle strength, and physical inactivity (4). Biomechanical stress, metabolic syndrome, and low-grade systemic inflammation further contribute to disease progression(2, 4).If left untreated or poorly managed, OA leads to chronic pain, progressive functional decline, and reduced participation in daily life activities. Ultimately many patients require joint replacement surgery(5). Therefore, emphasis is laid on Preventive strategies such as maintaining healthy body weight, regular physical activity as walking, cycling, swimming, avoiding repetitive joint loading and early treatment of joint injuries(1). Pharmacological management focuses on symptom relief rather than disease modification. Commonly used medications for OA are analgesics, nonsteroidal anti-inflammatory drugs (NSAIDs) ,intra-articular injections (corticosteroids or hyaluronic acid) .They are effective for pain reduction but have side effect such as gastrointestinal bleeding, cardiovascular events, or local joint irritation, and do not stop disease progression(6). Due to multiple factors involved in the complex pathogenesis of OA and the unclear underlying mechanism, the development of new drugs and treatment modalities for OA is yet to be done. Therefore, it is very important to clarify the underlying mechanism of its pathogenesis and discover new approaches to prevent and treat OA.(3, 6)

Physical therapy (PT) is considered as a keystone non pharmacological and non-surgical treatment of OA and is recommended in all major international clinical guidelines. PT interventions include strengthening exercises, aerobic conditioning, flexibility training, manual therapy. The aim is to improve pain, mobility, and quality of life, while having an excellent safety profile compared to long-term pharmacotherapy(7).Although the benefits of physical therapy for OA cannot be denied but several research gaps remain. Long-term adherence, optimal exercise dosage and personalized therapy based on OA patient require further investigation(4). Comparative effectiveness trials between different PT modalities, and between PT and pharmacologic or surgical interventions are needed. Furthermore, studies evaluating technology-assisted PT (e.g., tele-rehabilitation, wearable feedback devices) are needed to provide accessible care models.(1, 3)

In this review, the prevalence and clinical symptoms of OA, the modifiable and nonmodifiable risk factors for OA, pathogenesis and the current clinical treatments for OA are reviewed. Physical therapy as a promising treatment of OA is discussed.

Osteoarthritis (OA), a major public health concern

A significant shift in the burden of disease from communicable to non-communicable disease (NCD) is observed in Low- and middle-income countries (LMICs). Musculoskeletal conditions form a significant proportion of NCDs, with osteoarthritis (OA) contributing most to this burden. OA carries a risk of mortality and financial burden both societally and to individuals suffering from it. Osteoarthritis affects about 10% of people aged 60 years and above worldwide. It is a leading contributor to global disability, with a 9.6% rise in age-standardized years lived with disability (YLD) from 1990 to 2017, and ranks among the fastest-growing causes of disability alongside diabetes(8).Osteoarthritis (OA) is a highly prevalent and disabling joint disorder, that primarily affect the hip and knee joint. Its incidence increases with age, and is more common in women over 50 years of age (9). While most research originates from high-income countries, evidence suggests OA prevalence may be higher in low- and middle-income countries (LMICs), particularly affecting the knees, hips, hands, feet, and, less commonly, the shoulders and spine.(8, 9)Global estimates from the Global Burden of Disease studies shows variability in prevalence due to differences in population and risk factors such as age, sex, obesity, and genetics. While most epidemiological data come from Western Europe and the US, there is limited information from the Middle East. (9)

Pathophysiology of osteoarthritis (OA)

Osteoarthritis (OA) is a multifactorial, complex disease that is characterized by progressive degeneration of articular cartilage, subchondral bone, and synovium. Osteoarthritis development is driven by metabolic disturbances, inflammatory processes,

and mechanical load that eventually results in cartilage degradation, subchondral bone remodeling, synovial proliferation, and osteophyte formation figure (10). Six different aspects of OA pathogenesis include cartilage matrix degradation, inflammation, fibrosis, failed cartilage repair, bone remodelling and ageing.(11)

Cartilage Matrix Degradation: Matrix metalloproteinases (MMPs) and ADAMTS enzymes degrade the extracellular matrix (ECM) of cartilage composed of collagen (COL2A1) and proteoglycans (ACAN), is progressively broken down, weakening the tissue that ultimately results in its inability to withstand mechanical stress. This degradation leads to thinning of cartilage, surface fissures, and loss of joint cushioning.

Inflammation: OA is a low-grade chronic inflammation in the joint. Pro-inflammatory cytokines such as IL-1β, TNF-α, and IL-6 activate chondrocytes and synovial cells to produce more cytokines and matrix-degrading enzymes. Inflammation contributes to pain, swelling, and further accelerates the process of cartilage and bone damage.

Fibrosis / Synovial Changes: The synovial membrane undergoes proliferation and ultimately fibrosis, that results in stiffening of the joint capsule. These Fibrotic changes restrict joint movement, exacerbate pain, and contribute to functional limitations.

Failed Cartilage Repair: Chondrocytes attempt to repair damaged cartilage but fail due to apoptosis, senescence, and hypertrophy. Impaired autophagy and reduced ECM synthesis also play their part, so the damaged areas progressively enlarge.

Bone Remodelling / Subchondral Changes: OA affects the subchondral bone underlying the cartilage. In Early stages, the Trabecular thinning occurs, porosity is increased, and microcracks appear which increases the susceptibility to deformation. In Later stages; Sclerosis, osteophytes, and cyst formation pursues, altering joint movements and further stressing the cartilage.

Ageing / Metabolic Dysfunction: Age-related changes, including cellular senescence and metabolic dysfunction, impair the maintenance of cartilage and bone. Chondrocytes and bone cells lose the ability to sustain ECM turnover, making the joint more vulnerable to stress and inflammation. Ageing amplifies all other pathological processes, accelerating joint degeneration Overall, OA results from a complex interplay of structural, cellular, inflammatory, and metabolic mechanisms, many of which remain incompletely understood, highlighting the need for further research.

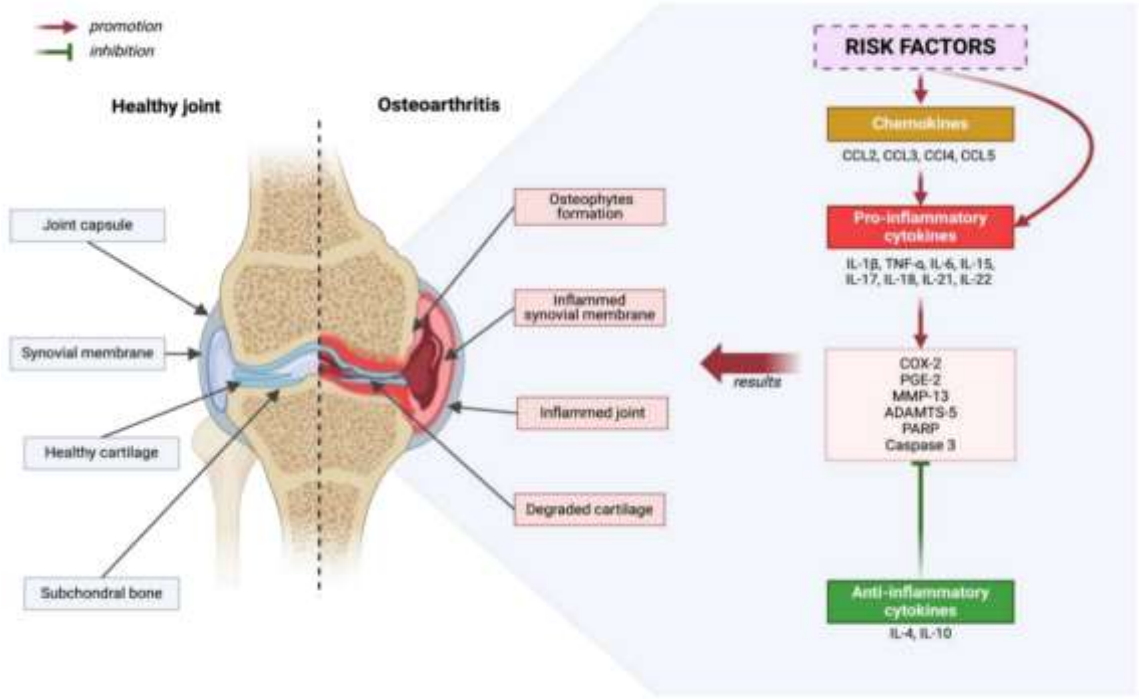

Figure: Diagram depicting a healthy joint in comparison to a joint that has been affected by degenerative disease. Inflammatory processes are outlined, considering the promoting and inhibiting effects of molecular factors.(10)

Symptoms of osteoarthritis (OA)

Typical symptoms of OA include joint pain that increases with movement and is relieved by rest, and brief joint stiffness that develops after inactivity(12) . Pain is the predominant symptom in people with knee OA, the pain is usually provoked by load bearing and relieved by rest, but it may become less predictable over time. Patients also frequently report other problems such as dysfunction, joint stiffness, and sleep disturbances (13). The joint Stiffness is worse in the morning or on arising after prolonged sitting.(11, 12)

The radiographic features of OA include joint space narrowing along with degradation of articular cartilage and meniscus, and bony changes such as sclerosis of subchondral bone and osteophytes. synovitis also occurs in OA patients which is triggered by the macrophage-mediated innate immune response. On physical examination, knee effusions of OA patients are generally either absent or small and cool in comparison to other arthritis patients, such as those of rheumatoid arthritis who have warm, easily palpable effusions. The complete understanding of OA symptoms is helpful to distinguish OA from other diseases that can cause joint pain(1).

Risk factors of osteoarthritis (OA)

Include both modifiable and non-modifiable risk factors.(1, 4, 6-9, 11-14)

Non-modifiable Risk Factors include age, gender, genetics, and ethnicity. Incidence and prevalence of OA increase with age, particularly after 50 years. OA is more common in women than men, especially post-menopause. Genetic predisposition contributes to OA susceptibility, especially seen in hand, hip, and knee OA.(13)

Modifiable Risk Factors include obesity, joint trauma, repetitive mechanical stress, muscles weakness and physical inactivity. Excess body weight increases mechanical load on weight-bearing joints (knees, hips) and promotes low-grade inflammation via adipokines. Previous injuries, fractures, or ligament tears increase OA risk. Occupations or sports involving repetitive joint loading can accelerate cartilage wear and tear. Weak muscles also increase joint stress. Sedentary lifestyle contributes indirectly through weight gain and reduced joint protection. Other factors contributing to OA are metabolic disorders such as diabetes, dyslipidaemia; Hormonal changes as reduced estrogenic levels in postmenopausal women and bone and joint alignment abnormalities including malalignment (varus/valgus deformities).

Treatment options for OA

Different treatment modalities are used for OA including non-pharmacological, pharmacological and surgical. Pharmacological treatment includes the use of different medications as acetaminophen (Paracetamol), Nonsteroidal Anti-Inflammatory Drugs (NSAIDs) and Intra-Articular Corticosteroid Injections for short-term relief of inflammation and pain in severe flare-ups and hyaluronic acid Injections with the aim to improve joint lubrication and reduce pain. Disease-modifying OA drugs (DMOADs) targeting inflammation or cartilage metabolism are currently under investigation(2, 3).

Surgical options for osteoarthritis are Arthroscopy, Osteotomy and Joint Replacement (Arthroplasty). Joint replacement is the most commonly used surgical intervention for OA with positive outcomes. (5).

Non pharmacological treatment includes Physical Therapy, Weight Management, Lifestyle Modifications and Patient Education(1).

Physical therapy as a promising treatment for osteoarthritis

Aims

The outcomes of OA include biomechanical overload, impaired shock absorption, low-grade inflammation, and altered pain processing. Consequences include quadriceps/hip abductors and other muscles weakness, reduced range of motion (ROM), gait deviations, proprioceptive deficits, and ultimately the patient avoids physical activity at all. PT targets these areas aiming at Restoring capacity, improving movement quality, reducing pain and stiffness and enhancing self-efficacy.(7)

Core physical therapy (PT) Interventions

Patient Education & Self-Management

Patient education plays an important role and addresses OA mechanisms, prognosis, activity pacing, flare management, sleep, and weight control. Emphasis is laid on **staying active** as most of the patients avoids physical activity due to fear of pain worsening. Brief cognitive-behavioural strategies such as goal setting, action planning also plays role in improving adherence and outcomes.

Exercise Therapy (Foundation of Care)

A meta-analysis of 77 randomized controlled trials (6,472 participants) showed significant benefits of exercise on Pain (effect size [ES] = 0.56), Function (ES

= 0.50), Performance (ES = 0.46) and Quality of Life (QoL) (ES = 0.21). peak improvements were noted around 2 months with gradually waning effects after 9 months(15)
Home-based or therapist-led exercise programs lasting at least 8 weeks are effective for alleviating pain, improving function, and better QoL in knee OA. Both home-based exercise and exercise led by a physical therapist improved pain, function and quality of life in patients with osteoarthritis of the knee. To gain positive outcomes the exercises must be carried out for at least 4 weeks. Most of the exercises concentrated on strength training of the quadriceps and used 2-4 repetitions each week(16).
An umbrella review on non-pharmacological interventions for osteoarthritis concluded that diet therapy, patient education, and resistance training are core treatment for OA , while aquatic therapy, balance training, dietary supplements, extracorporeal shockwave therapy, and tai chi are also good treatment options for OA but not as effective as the above mentioned treatment options(17)

Adjunct Modalities and Manual Therapies
A systematic narrative review on PT modalities for hip and knee OA states that 15 out of 30 interventions, most important of which are ultrasound, diathermy, and electrical stimulation successfully relieved pain associated with OA (≥20% improvement in WOMAC or VAS), however no single modality was proved to be better than the other in terms of pain management of OA . The review suggested structured, sequential multimodal protocols including TENS followed by ultrasound and then diathermy for sustained benefit(18). Manual therapy (MT) addressing soft tissues and joint mobilization has shown short-term effectiveness in reducing pain and improving range of motion and functionality in knee OA, although more comparative studies are needed(19)

Aquatic Therapies
In clinical practice, aquatic exercise can be used as an adjunct to medication and other non-pharmacological treatment such as land-based exercise, manual therapy, knee bracing, and physical modalities for the management of OA symptoms. A systematic review and meta-analysis of RCTs assessing the effects of aquatic exercise in osteoarthritis (OA) patients indicate that aquatic therapy significantly reduces pain, improves function, and enhances quality of life in OA patients. However, further high-quality research is needed to address logistical barriers like cost, space, and access(20).

Stationary Cycling (Indoor or Land-Based)
A meta-analysis of 8 RCTs (total 724 participants) concluded that cycling significantly reduces pain (WMD = 12.86) and improves sport-related performance (WMD = 8.06), though the improvements in stiffness, daily function, and QoL were unable to meet minimal clinical thresholds(21)

Final word
Physical therapy remains a cornerstone in the non-surgical management of osteoarthritis, with exercise therapy and patient education forming the foundation of care. Evidence supports the use of multimodal strategies integrating land-based or aquatic exercise with manual therapy and carefully selected physical modalities to achieve superior clinical outcomes. Emerging interventions, such as indoor cycling, demonstrate promising benefits, particularly in improving joint mobility, pain control, and functional performance. However, Therapeutic plans must be in line with individual patient characteristics such as disease stage, comorbidities, and personal preferences in order to achieve sustained adherence and optimize long-term results.

Conclusion: Physical Therapy remains a cornerstone in the non-surgical management of osteoarthritis, offering safe, cost-effective, and clinically meaningful benefits in pain reduction, functional improvement, and quality of life. Exercise therapy encompassing strength training, aerobic conditioning, flexibility exercises, and neuromuscular control forms the foundation of care, complemented by patient education, lifestyle modification, and behaviour-change strategies. Adjunctive interventions, including manual therapy, aquatic exercise, stationary cycling, and selected physical modalities, can further enhance outcomes when tailored to individual patient characteristics.
Despite these advancements, challenges such as long-term adherence, optimal dosages, individualized programming, access limitations, and integration of digital health solutions remain. Future research should focus on rigorous, high-quality trials to refine exercise protocols, evaluate multimodal interventions, and expand accessible care models. Overall, evidence supports positioning physical therapy as a first-line, holistic approach in osteoarthritis management, emphasizing personalized, multimodal strategies to

optimize long-term joint health and patient well-being.

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