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Pathogenesis, Risk Factors and Recent Evidence on Physical Therapy Interventions in Knee Pain

Seyed Massood Nabavi*

Department of Neurology and Neuro-physiology, Tehran University of Medical Sciences, Tehran, Iran

Introduction

Knee osteoarthritis is a degenerative joint disease that is the leading cause of knee joint replacement surgery in the United States, with 4.7 million people undergoing surgery in 2010 at a cost of USD 29,488 per surgery. The high frequency of knee OA results in significant social and personal costs, as well as a strong desire to avoid surgery by preventing OA progression. Knowing a patient's risk factors can help them understand their prognosis and clinicians must tailor a patient's treatment plan to their specific needs in order to keep them functional.

Description

Physical therapy is the first step in the patient's treatment, which includes medication (such as acetaminophen, nonsteroidal anti-inflammatory medications AND duloxetine) (e.g., diathermy, exercise therapy, ultrasounds, knee brace and electrical stimulation). Because the exact causes of knee OA progression are not completely understood, determining the optimum therapy for individual patient is difficult. This narrative review includes the most recent information on the pathophysiology of OA, risk factors for developing OA and the most recent evidence for physical therapy to assist doctors in treating persons living with knee OA and preventing knee replacement [1].

Osteo Arthritis has been depicted as the result of articular cartilage degeneration over time. Patients with OA, despite the cartilage's ability to resist biomechanical damage induced by excessive loading, obstruct attempts at healing and disrupt cartilage homeostasis. For example, changes in the content and structure of cartilage cells (i.e., chondrocytes)-such as hypertrophy owing to ageing or oxidative stress-trigger the production of catabolic substances, which accelerate cartilage degeneration. Catabolic factors such cytokines, chemokines and proteolytic enzymes-cytokines (e.g., IL-6, IL-8), chemokines metalloproteases AND heat-shock proteins (e.g., HSPA1A)-have been discovered as quantifiable biomarkers for predicting the start and progression of knee OA. As a result, cartilage degradation as a result of extracellular matrix destruction has been shown as one of the major biological initiators of the OA pathological process for decades [2].

The articular cartilage of the knee begins to breakdown when the pathological process of knee OA is activated by catabolic factors generation, rendering it unable to adequately absorb physiological and physical pressures. This causes joint conformational changes to compensate for the loss of articular cartilage, demonstrating that OA is a healing process. Subchondral bone (SB) sclerosis thickening and hardening as well as the production of bone cysts and marginal osteophytes—are examples of these alterations (coming from bone remodelling). As a result of all of these SB changes, the joint space narrows, hastening the course of OA. Due to synovial inflammation and fibrosis of the joint capsule, OA eventually affects the entire joint, causing loss of range of motion/stiffness, discomfort and pain.

When one experience triggers the next, this pathological pattern has been defined as a vicious cycle of OA. In OA disease, pain is caused by a combination of peripheral and central processes. Nerve sensitizations, for example, are important aspects of pain transmission in OA patients and may contribute to the pain-joint pathology mismatch [3]. The discomfort comes from the synovium, subchondral bone and periosteum, which are innervated by small-diameter nociceptive neurons since hyaline cartilage is not innervated. Tissue damage caused by joint degradation produces nociceptive sensations. Previous research has linked pain to a variety of anatomical issues, such as bone marrow lesions, synovial thickening (synovitis) and knee effusion.

The synovium and chondrocytes release inflammatory mediators that augment the stimulation of nociceptive neurons, resulting in an increased painful response. SB is also a starter of the OA vicious circle, according to new evidence. Indeed, instead of being viewed as a disease of cartilage deterioration, OA is viewed as a joint failure induced by aberrant joint stress. Changes in the SB-which can cause pain through nociceptive stimuli predispose the cartilage to further wear and tear because the SB is less able to absorb forces/load placed on the joint mechanical properties of the SB occur during remodelling, such as bone hardening, which causes increased stiffness and leads to cartilage loss cartilage [4].

Obese people had a 66 percent likelihood of developing symptomatic knee OA, while people of normal weight have a 45 percent chance. Furthermore, the Framingham OA trial found that losing roughly 5 kg 2 units of BMI cut the incidence of knee OA in half for women. Through systemic and biomechanical variables, obesity raises the chance of developing OA. A force of three to six times the body weight is exerted over the knee during a single-leg stance in walking, which is based on the multiplier effect of lever arms outside the body's central axis.

As a result, a rise in weight increases the force across the knee during walking in an obese person, placing the joint's tissue at danger of harm. Regardless of whether weight has a limited effect on the course of knee OA in moderately misaligned knees (2–7 degrees), highly misaligned knees will develop an OA joint regardless of the weight added to it. Furthermore, the growth of adipose tissue that secretes adipokines further strengthens the link between obesity and OA. Indeed, this biologically active chemical causes joint inflammation, which disrupts cartilage homeostasis and makes them more vulnerable to OA [5].

It's becoming increasingly clear that it's largely a mechanical issue, with all of the risk variables affecting the joint's biomechanical stress and contributing to disease progression. Although there are numerous therapies for OA, their efficacy is inconsistent. The WOMAC (Western Ontario and McMaster Universities Osteoarthritis Index) score is one of the most effective techniques to assess the effectiveness of physical therapy interventions. For each of the WOMAC sub-scales, a 20% reduction in clinical efficiency is the minimum. We conducted a review of the key physical therapy interventions utilised by clinicians specialising in knee OA rehabilitation, such as diathermy, exercise therapy, ultrasound knee brace and electrical stimulation, in this study.

***Address for Correspondence:** Seyed Massood Nabavi, Department of Neurology and Neuro-physiology, University of Medical Sciences, Tehran, Iran, E-mail: seyedmassod@gmail.com*

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Conclusion

This evaluation of the research assists physicians in making evidence-based decisions for lowering knee pain and managing persons with OA to avoid knee replacement. Looking at the relative risk reduction in pain perception using the WOMAC scale for diathermy, ultrasounds, knee braces and electrical stimulation, our findings show that only the brace interventions consistently met the clinical efficiency minimum, making the intervention not only significant, but also valuable for the patients' quality of life. Furthermore, over half of the exercise and diathermy therapies met the clinical efficiency threshold for pain reduction.

Conflict of Interest

None.

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