Editorial

Menopause and Osteoarthritis: Any Association ?

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Osteoarthritis (OA) is progressive joint disease characterized by joint inflammation and a reparative bone response and is one of the top five most disabling conditions that affects more than one-third of persons >65 years of age, commonly affecting hands, feet, spine, hips, and knees. Approximately 100 million people worldwide suffer from OA. In a recent statement quoted by Piramal Healthcare Limited in a nationwide campaign against chronic diseases, "India is expected to be the chronic disease capital, with 60 million people with arthritis, by 2025."[1]

OA strikes women more often than men and it increases in prevalence, incidence, severity with preponderance to polyarticular involvement, with increased hand and knee OA after menopause. The effects of age on both hip and knee OA risk in women follow similar patterns, increasing rapidly between the age of 50 and 75 years. Conversely, risk of hand OA peaks in women after menopause with ≥3.5-fold higher rates in women aged 50–60 years when compared to men of similar age.[2] A large epidemiological study was conducted in Italy supporting the hypothesis that estrogen deficiency may increase the risk of OA.[3]

OA affects all articular tissues and finally leads to joint failure. Although articular tissues have long been considered unresponsive to estrogens or their deficiency, there is now increasing evidence that estrogens influence the activity of joint tissues through complex molecular pathways that act at multiple levels.[4] Several experimental studies have shown that estrogens are implicated in the regulation of cartilage metabolism. Indeed, 17 β-estradiol enhances glycosaminoglycan synthesis in cultures of rabbit joint chondrocytes through the upregulation of the uridine diphosphate glucose dehydrogenase gene.[5] The effects of estrogen on joint tissues have primarily been studied in ovariectomized animal models. Despite these studies, the influence of estrogen deficiency on cartilage remains unclear, even though there is significant evidence of the detrimental effect of estrogen loss in mature female animals.[6]

Although most of the reviews on OA usually focus on the statement that OA is no longer a disease limited to cartilage only but despite of this maximum resources are diverted to develop the chondroprotective drugs, so there is a huge lacuna in this perspective. Pharmacological treatment in the form of drugs which can relieve symptoms such as acetaminophen, salicylates, and traditional nonsteroidal anti-inflammatory drugs, opoids such as tramadol, topical analgesia, and intraarticular, glucocorticoid injections can be helpful. Besides there are drugs with symptom-modifying potential in OA which include glucosamine sulfate, chondritin sulfate, and diacerein.[7] The lack of a complete understanding of the complex processes involved in the etiopathogenesis, and subsequent appropriate phenotyping makes it difficult to find therapies that may be efficacious in most patients with OA. To some extent, OA may be considered an orphan disease. Therefore, there is an urgent need to identify effective and safe new pharmacologic modalities for treating OA.[8]

Due to the poor self-healing capacity of articular cartilage and lack of specific diagnostic biomarkers, OA is a challenging disease with limited treatment options. The current research focuses on the development of new OA drugs (such as sprifermin/recombinant human fibroblast growth factor-18, tanezumab/monoclonal antibody against β-nerve growth factor), which aims for more effectiveness and less incidence of adverse effects than the traditional ones. Furthermore, regenerative therapies (such as autologous chondrocyte implantation (ACI), a new generation of matrix-induced ACI, cell-free scaffolds, induced pluripotent stem cells [iPS cells], and endogenous cell homing) are also emerging as promising alternatives as they have potential to enhance cartilage repair, and ultimately restore healthy tissue. However, despite currently available therapies and research advances, there remain unmet medical needs in the treatment of OA.[9]

In conclusion, the menopause is associated with the onset and progression of OA in women, and HRT can render help in such patients by reducing symptoms and progression, increasing bone mineral density, reducing bone, and radiological abnormalities in OA, but presently it cannot be recommended the first-line treatment with the current level of evidence.

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