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Pathophysiology of Osteoarthritis

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Introduction

Osteoarthritis (OA) is a common disease of the joints, and it is also known as degenerative joint disease. According to Batt and Mobasheri (2016), OA affects more than 20 million Americans every year. It is considered a priority disease by the World Health Organization, with the prevalence of the disease expected to rise worldwide over the next ten years (Batt & Mobasheri, 2016).

The disease is defined as a progressive breakdown and loss of articular cartilage around joints, as well as deterioration of the structure and function of joints (Batt & Mobasheri, 2016). The extracellular matrix of cartilage allows it to handle repetitive stress loading for decades, but has low regenerative capacity (Fernandes et al., 2019). Changes associated with OA are found in the synovial fluid and cavity, meniscus of the knee, surrounding ligaments, and subchondral bones (Batt & Mobasheri, 2016). Age, obesity, injury/trauma, metabolic disease, innate immunity, and systemic inflammatory responses are all risk factors of development of OA (Batt & Mobasheri, 2016). The aging of articular cartilage has always been a primary culprit for development of OA, but recent studies have found that chronic, low-grade inflammation within the joint has a separate pathologic impact (Batt & Mobasheri, 2016).

Clinical Manifestations

- Pain in the joints
- Generalized fatigue
- Pain with movement, walking, or change of position
- Gradual stiffening of joint
- Redness, swelling, and increased temperature around affected joints
- Immobilization of a joint and muscle atrophy (Koszowska et al., 2014).

Reducing Risk

- Lifestyle modification and exercise are the two key elements in reducing risk
- Weight management
- Avoiding sedentary lifestyle
- Managing comorbidities
- Improving sleep habits

Underlying Pathophysiology:

There are multiple phenotypes and pathological processes that contribute to the development of OA (Batt & Mobasheri, 2016). Age, obesity, injury/trauma, metabolic disease, innate immunity, and systemic inflammatory responses are all risk factors of development of OA (Wang et al., 2015). Inflammation is currently the primary focus of OA pathophysiology, as all of these mentioned factors can lead to chronic, low-grade inflammation. Metabolic inflammation is another pathological process leading to OA. Metabolic surplus results in oxidative stress and inflammation, and begins a stress cycle that leads to cell dysfunction, obesity, and increased incidence of metabolic disease (Wang et al., 2015). This can lead to chronic inflammation due to increased levels of cytokines, C reactive proteins, and other inflammatory mediators (Wang et al., 2015). Excessive metabolites and nutrients, such as lipids and glucose in diabetic patients, can disrupt systemic metabolism and lead to inflammatory responses. (Wang et al., 2015). Chronic inflammation due to obesity, comorbidities, disruption in sleep habits, or repetitive injury results in the activation of complement systems that lead to tissue and cartilage destruction (Wang et al., 2015). Pro-inflammatory, TNF, and pro-catabolic mediators, along with oxidative and mechanical stressors, decrease the function and life cycle of cartilage cells which causes them to hypertrophy, and become more sensitive to inflammatory responses (Batt & Mobasheri, 2016). Programmed cell death also occurs during the development of OA, with chondrocytes undergoing apoptosis during the progression of OA and ossification of chondrocytes (Hughes et al., 2017).

Significance of Pathophysiology

Understanding the pathophysiology and inflammatory process of OA is significant in understanding how to treat, prevent, and educate patients about the disease process. With so many Americans being diagnosed with OA, it is something that a family nurse practitioner will diagnose and treat often, and knowing of the pathophysiology behind OA allows for more targeted and personalized treatment of the disease.

Treatment

- Treatment is aimed at improving quality of life (Koszowska et al., 2014).
- Exercise: Reduces pain, fatigue, inflammation, and can prevent obesity and other diseases that can lead to OA
- Joint replacements
- Tissue engineering to replace cartilage with hydrogels similar to the body’s cartilage (Fernandes et al., 2019).
- Implantation of autologous chondrocyte matrix and chondrocytes to improve regeneration (Fernandes et al., 2019).
- Pharmacological interventions to reduce pain and inflammation
- Steroid or hyaluronic acid injections into affected joint (Koszowska et al., 2014).

Implications for Nursing

OA is a commonly seen and treated disease in the healthcare setting. Both bedside nurses and APNs need to have an understanding of the disease process, prevention, and treatment measures in order to provide education to patients. Educating at risk patients about lifestyle modifications can prevent development of OA.

Providing education about lifestyle modifications to those patients already diagnosed with OA can help to slow progression and improve quality of life. APNs should have knowledge of medications that are used to treat symptoms, and prescribe them as needed, and make referrals to specialists when necessary.

Conclusion

- Osteoarthritis is a debilitating chronic disease that affects many Americans
- The number of cases is expected to increase worldwide in years to come.
- The pathophysiology of OA is multifactorial, but primarily attributed to chronic inflammation, and release of inflammatory biochemistry (Batt & Mobasheri, 2016).
- Risk factors include obesity, aging, repeated joint stress, and comorbidities.
- Treatment is aimed at managing symptoms and improving QOL.
- Prevention and education are critical factors in reducing the incidence and slowing progression of OA.

Additional Sources for Patient Education


References


Wang, Y., et al. (2015). Chronic inflammation due to obesity, comorbidities, disruption in sleep habits, or repetitive injury results in the activation of complement systems that lead to tissue and cartilage destruction. Arthritis Research & Therapy, 17(1), 130.

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