Reumatología Clínica

Editorial

Osteoarthritis: Something is moving

Artritis: algo se mueve

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Osteoarthritis (OA) is the most common rheumatic pathology and classically is associated with ageing. Prevalence studies demonstrate that most individuals above 65 years present evidence of this pathology, giving an idea of its enormous social impact. Global estimates are that 9.6% of men and 18.0% of women over 60 have symptomatic OA. OA is the first cause of permanent job incapacity; it is one of the most frequent causes of incapacity in the elderly, and also one of the most common reasons for primary care visits. Between 2002 and 2007, OA moved from the twelfth to the sixth leading cause of years lost by disability or morbidity (World Health Organization (WHO) data). US studies confirm that OA is responsible of 4 million hospitalizations and the loss of 68 million labour days per year. Given the current rate/tempo of population ageing, it is estimated that the number of people who suffer from this disease will double in the next three decades. Moreover, they estimate the costs of an OA patient (drugs, medical visits, radiographies, etc.) at around 2000 dollars/year.

However, although the social, economic and health impact of OA is very high, therapies are symptomatic and pursue only pain alleviation, but have no effect on slowing down the progression of the disease. At least three causes could explain the actual limitation of treating the OA progression. One is the classical OA definition. Although OA has a multifactorial aetiology, for a long time it has been primarily associated with the breakdown of cartilage in joints. A classical definition of OA is referred as a degenerative joint disease involving cartilage degradation, synovial inflammation and subchondral bone sclerosis. Nevertheless, according to our actual knowledge any definition of OA must include degradation of the articular cartilage, thickening of the subchondral bone, osteophyte formation, variable degrees of synovial inflammation, degeneration of ligaments and, in the knee, the menisci, and hypertrophy of the joint capsule. There can also be changes in periarticular muscles, nerves, bursa, and local fat pads that may contribute to OA or the symptoms of OA. The findings of pathological changes in all of the joint tissues are the impetus for considering OA as a disease of the joint as an organ, resulting in an organic dysfunction or joint failure.[1]

Another explanation is regarding the diagnostic criteria of OA. Currently, the diagnosis of OA relies on the description of pain symptoms, stiffness in the affected joints, and radiography, used as the reference technique for determining the grade of joint destruction. Nevertheless, X-ray offers only indirect information about the state of the cartilage, such as narrowing of the joint space or the appearance of bony spurs (osteophytes). Moreover, this procedure lacks sensitivity in detection of slight changes in the joint, making it necessary to wait for several years in order to obtain feasible information about the progression of the disease. Thus, efficient strategies in detecting early phases of OA are essential for the development of new OA modifying therapies and for the evaluation of therapeutic answers. These include the standardization of imaging techniques (magnetic resonance imaging (MRI) and ultra-sound (US)) and the identification of early biomarkers and molecular players of OA. Both MRI and US are more sensitive than radiography in detection of cartilage degradation, sinovitis, subchondral bone modifications and any damaged tissue in the joint. In the past few years, new approaches in OA research such as genomic, proteomic and metabolomic technologies are increasing the number of potential molecular biomarkers for the diagnosis and prognosis of OA.[2] Some of these biomarkers (after clinical qualification) in combination with the OA risk factors such as ageing, heredity, obesity, and mechanical influences, including joint injury or joint overuse could be parameters to define an index to predict the risk of developing OA similar to the cardiovascular index or FRAX. All of these new approaches must help us with diagnosis of early OA (symptomatic and asymptomatic OA), and early diagnosis will permit earlier treatment to modify the course of this disease.

Finally, OA has been considered and it has been treated as a unique disease, without taking into account the different OA phenotypes. Given our current understanding of OA pathogenesis this concept must change. OA is emerging as a disease that has a variety of phenotypes including metabolic, age-related, inflammatory, hormonal and injury-related phenotypes. In addition, although in some cases the patients present a clear phenotype,[3,4] in some patients the phenotype is overlapped. From all phenotypes the metabolic phenotype is demonstrating high interest among the scientific community. Evidence from both epidemiological and biological studies support the concept of metabolic OA, defined as a

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broad clinical phenotype that includes obesity-related OA. Interestingly, studies have demonstrated associations linking OA to several components of the metabolic syndrome, such as hypertension and type 2 diabetes, independently from obesity or any of the other known risk factors for OA. Thus, it is clear that if we want to improve the health of patients with OA, we must treat each OA phenotype with an appropriate therapy.

In conclusion, OA is a prevalent, disabling disease, involving an organ (the joint), resulting in an organic dysfunction or joint failure that currently lacks disease-modifying treatments. The ability to detect early OA as well as to characterize the OA phenotypes are crucial for understanding the disease process, identifying potential disease-modifying treatments, and evaluating the effectiveness of new therapies.

References

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