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Review Article

Cartilage lesions and ankle osteoarthrosis: review of the literature and treatment algorithm☆,☆☆

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ABSTRACT

The main etiology of ankle osteoarthrosis is post-traumatic and its prevalence is highest among young individuals. Thus, this disease has a great socioeconomic impact and gives rise to significant losses of patients' quality of life. The objective of its treatment is to eliminate pain and keep patients active. Therefore, the treatment should be staged according to the degree of degenerative evolution, etiology, joint location, systemic condition, bone quality, lower-limb alignment, ligament stability and age. The treatment algorithm is divided into non-surgical therapeutic methods and options for surgical treatment. Joint preservation, joint replacement and arthrodesis surgical procedures have precise indications. This article presents a review on this topic and a proposal for a treatment algorithm for this disease.

Lesão de cartilagem e osteoartrose do tornozelo: revisão da literatura e algoritmo de tratamento

Palavras-chave:
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RESUMO

A principal etiologia da osteoartrose (OA) do tornozelo é pós-traumática e sua maior prevalência está entre indivíduos jovens; assim, essa doença apresenta grande impacto socioeconômico e significativo prejuízo na qualidade de vida dos pacientes. O objetivo do tratamento é eliminar a dor e manter os pacientes ativos. Dessa forma, o tratamento deve ser estagiado de acordo com o grau de evolução da degeneração, a etiologia, a localização articular, a condição sistêmica, a qualidade óssea, o alinhamento do membro inferior, a estabilidade ligamentar e a idade. O algoritmo de tratamento é dividido nas modalidades

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de terapia não cirúrgicas e nas opções de tratamento cirúrgico. As cirurgias de preservação articular, as cirurgias de substituição articular e as artrodeses apresentam indicações precisas. O presente artigo apresenta uma revisão sobre o tema e uma proposta de algoritmo de tratamento para essa doença.

Introduction

Osteoarthrosis (OA) is a syndrome characterized by degeneration of the joint cartilage, alterations to subchondral bone, intra-articular inflammatory alterations and periarticular bone growth, and it causes pain and functional loss in the affected limb.[1–3] There is still no effective cure for this syndrome today, through any methods for prevention, diminution of its progression or treatment of symptoms that have been proposed.[1,2,4,5]

OA affects 15% of the worldwide adult population, which makes it a disease of high socioeconomic impact both for individuals and for all of society.[6] For example, in the United States, this represents annual costs of 60 billion dollars for the direct treatment.[1,6,7]

The most important universal risk factors are age, excessive joint loading, joint injuries, fractures and ligament injuries.[1,6]

OA of the tibiotarsal joint is present in 4.4% of the patients who seek orthopedic attendance because of OA of the lower limbs.

Differently from hip joint degeneration (58%) and knee joint degeneration (67%), OA of the ankle is of primary origin in only 9% of the cases. The secondary causes (rheumatoid arthritis, hemochromatosis, hemophilia or osteonecrosis) are present in 13% of the cases. The main etiology is post-traumatic and this is the reason for tibiotarsal joint degeneration in 78% of the cases, while fractures around the ankle are the cause in 62% and ligament injuries are the cause in 16%.[7–9]

Individuals with ankle arthrosis tend to be younger than other patients with joint degeneration in the lower limbs and present faster functional loss, with progression to the final stages of the disease between 10 and 20 years after the start of the lesion.[9]

Physiopathogenesis

A variety of anatomical and biomolecular characteristics of the ankle are determinants for understanding the susceptibility of the cartilage of this joint to degeneration.

The total area of the tibiotarsal joint is 350 mm^2 and it is subjected to a force of 500 N, while the hip and knee, with joint areas of respectively 1100 mm^2 and 1120 mm^2, are subjected to the same force.[10–12] Thus, the pressure on the joint cartilage of the ankle may be up to three times greater than the pressure on the other joints of the lower limbs. On the other hand, the load distribution on the congruent joints, i.e. the ankle and hip, differs from the load distribution on the knee, such that the compressive forces are distributed over a larger area. This possibly allows the ankle cartilage to be thinner than that of the knee. The thickness of the ankle joint cartilage ranges from 1 to 1.62 mm and is thinner than that of the hip (1.35–2 mm) and knee (1.69–2.55 mm).[13]

Comparative biomolecular studies on humans have shown that the ankle cartilage has higher density of glycosaminoglycan sulfate and lower modulus of equilibrium, dynamic rigidity, water component and hydraulic permeability than those of the knee cartilage. The properties influence the capacity to deform under compression during the load cycle.[14,15] The way in which the collagen of the ankle cartilage is organized resembles that of the knee, but the chondrocyte distribution differs. In the ankle, in the superficial layer of the cartilage, the chondrocytes are presented in groups.[16]

Along with these characteristics, the cartilage tissue of ankles that are subjected to injury presents increased collagen synthesis. The chondrocytes of the ankle are metabolically more active than those of the knee and present greater aggrecan turnover and greater sensitivity to anabolic stimuli, followed by removal of interleukin-1, and greater response of the chondrocytes to inflammatory stimuli.[17–20]

The following are also determinants for the physiopathogenesis: poor structural or acquired alignments of the lower limb, muscle imbalance and weakness around the tibiotarsal joint, age, gender, ethnicity and genetic predisposition.[2,6]

Diagnosis and classification system

The clinical presentation consists of pain in the region of the joint interline, with or without an associated increase in volume (joint effusion) and limitations on the range of joint motion, functioning, work and recreational activities. These conditions may diminish the quality of life of individuals with diseases like hip OA, dialytic kidney failure, congestive heart failure or radiculopathy.[21] Other associated clinical alterations include leg muscle atrophy and alterations to gait patterns, particularly changes to kinematics and kinetics.[22–25]

The initial investigation by means of imaging is conducted using radiographs with weight-bearing. These may show different degrees if diminution of the joint space and formation of osteophytes, sclerosis and subchondral cysts. The Morrey and Wiedeman classification system is based on these radiographic findings.[26,27]

Magnetic resonance imaging (MRI) is the most sensitive and specific noninvasive imaging examination for evaluating the joint cartilage. By means of specific protocols for image acquisition and analysis, it also enables access to the morphology and biochemical composition of this tissue.[28]