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Review Article

Current concepts in articular cartilage repair

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ABSTRACT

Articular cartilage is a specialized connective tissue covering vrious joint surfaces. Due to its poor repair potential and no nerve supply early injuries can be easily missed. Articular cartilage injury poses a challenge to treating orthopaedic surgeons and with various treatment options available it becomes difficult to treat due to the limited self-healing capacity, affliction of a young active patient and risk of progression to secondary osteoarthritis. There is no universally accepted successful treatment for these lesions. The ideal treatment should provide good repair fill with hyaline cartilage and maintain quality of subchondral bone. There is an increasing need for high quality studies to evaluate and compare outcomes between different techniques currently available. This article discusses articular cartilage injury and the various treatment options available to the treating surgeon along with the future upcoming treatment options.

1. Introduction

Articular cartilage is a specialized connective tissue covering joint surfaces. It also has no nerve supply and is therefore not sensitive to early injuries. It also has poor repair properties, because there are relatively few cells in the tissue, the metabolic rate is low, and the matrix fibres restrict the capacity of chondrocytes to divide and migrate in the articular cartilage.[1] As a consequence, it is generally agreed that articular cartilage does not repair significantly after injury.[2]

Articular cartilage injury poses a major challenge to the treating orthopaedic surgeons due to the limited self-healing capacity, affliction of a young active patient and risk of progression to secondary osteoarthritis.[3] There is no universally accepted successful treatment for these lesions. The ideal treatment should provide good repair fill with hyaline cartilage and maintain quality of subchondral bone. There is an increasing need for high quality studies to evaluate and compare outcomes between different techniques currently available. This article discusses articular cartilage injury and the various treatment options available to the treating surgeon along with the future upcoming treatment options.

2. Response to injury

Deep lacerations of articular cartilage extending beyond the tidemark heal with fibrocartilage produced by undifferentiated mesenchymal cells. Superficial lacerations do not heal, although some proliferation of chondrocytes may occur.[4] Immobilization of joints leads to atrophy of the articular cartilage and therefore continuous passive motion is believed to be beneficial to healing. In arthritic cartilage, chondrocytes are recovered in clusters of upto thirty cells, which probably represents an attempt at tissue regeneration.[5]

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3. Why do we need hyaline cartilage repair tissue?

In fibrocartilage the matrix component is minimal, and the fibrous one greatly predominates. The chondrocytes are less numerous and much more widely separated than in other types, but most of them are still enclosed in lacunae.[5] Repair tissue that fills osteochondral defects is less stiff and more permeable than normal articular cartilage. The orientation and organization of the collagen fibrils in even the most hyaline-like chondral repair tissue do not follow the pattern seen in normal articular cartilage. The decreased stiffness and increased permeability of repair cartilage matrix may increase loading of the macromolecular framework during joint use and result in progressive structural damage, thereby exposing the repair chondrocytes to excessive loads. The remaining cells often assume the appearance of fibroblasts as the surrounding matrix comes to consist primarily of densely packed collagen fibrils. This fibrous tissue usually fragments and often disintegrates, thus leaving areas of exposed bone. The inferior mechanical properties of chondral repair tissue may be responsible for its frequent deterioration.[6]

4. Natural history of focal chondral defects

The natural progression of untreated chondral defects is still unclear.[7] Linden noticed 55% of patients who were diagnosed with osteochondritis dessicans after the closure of distal femoral physis progressed to osteoarthritis compared to zero percent of patients who were diagnosed as osteochondritis dessicans before the closure of distal epiphyseal line.[8]

Widuchowski retrospectively analysed 25, 124 arthroscopies. Cartilage lesions were classified in accordance with the Outerbridge classification.[9] Focal cartilage lesions were localized in 67%, osteoarthritis in 29%, osteochondritis dessicans in 2% and other types in 1% of the patients in this study. The patellar articular surface (36%) and the medial femoral condyle (34%) were the most frequent sites of the cartilage lesions. Curl noticed that patients under 40 years of age with grade IV lesions accounted for 5% of all arthroscopies.[9]

Lars Engebretsen in a prospective study on 993 knee arthroscopies noticed articular cartilage pathology in 66% and a localized cartilage defect was found in 20%.[10] A localized full-thickness cartilage lesion (ICRS grade 3 and 4) was observed in 11% of the knees. Of the localized full-thickness lesions, 55% of lesions (in 6% of all knees) had a size above 2 cm. Brittberg[11] in another prospective study of 1000 arthroscopies noticed focal chondral defects (ICRS grade 3 and 4) in 19% of patients with average size 2.1 cm^2. The medial femoral condyle was the commonest site for articular cartilage pathology in this study.

5. Clinical diagnosis

The spectrum of chondral pathologies seen in practice are osteochondral traumatic injuries, focal chondral defects and early osteoarthritis.[5] Traumatic osteochondral defects are common with patella dislocations and other significant knee trauma. Patello-femoral joint assessment should include an assessment of hypermobility and maltracking. These should be suspected by the presence of acute onset of significant swelling soon after the injury with lipo-haemarthrosis and with or without osteochondral fragment on radiographs.

Chondral defects have to be differentiated from early OA and the duration of symptoms could help in the decision making.[12] Patients with articular cartilage defects commonly present with knee pain often exacerbated by impact or weight-bearing. These can commonly be misinterpreted clinically with the meniscal injury in the presence of generalized degeneration.

Plain radiographs are essential in the initial assessment especially to rule out early osteoarthritis. Weight bearing long-leg alignment X-rays to assess normal knee alignment is mandatory before consideration of cartilage repair.

MRI scans using cartilage-sensitive sequences like fast spin echo or spoiled gradient-recalled echo are useful to estimate the cartilage loss, fissuring and delamination, underlying subchondral bone and the other structures in the knee.[12] In addition to diagnosing the location and size of defects, detailed cartilage MRI can identify reduction in cartilage volume, changes to GAG (Glycosaminoglycans) and collagen content and can also assess repair tissue. Standard MRI using a cartilage-sensitive sequence (e.g., spoiled gradient-recalled echo or fast spin echo) can show cartilage fissuring, delamination, and focal loss as verified by arthroscopy.[13,14] Quantitative and semi quantitative cartilage imaging techniques are now available and include dGEMRIC (delayed gadolinium-enhanced MRI of cartilage), sodium-23 imaging, T1rho, T2*, and T2 mapping techniques.[13] In comparison with traditional MRI, which emphasizes morphology, these additional techniques help to evaluate cartilage composition. In broad terms, dGEMRIC, sodium, and T1rho are sensitive to proteoglycan content, while measurement of T2 or T2* relaxation times are sensitive to collagen architecture, specifically collagen orientation. To assess the collagen orientation and free water content of repair tissue, T2 mapping techniques can be used.[15]

6. Arthroscopy

Arthroscopy is still the gold standard for assessment of cartilage lesions especially to assess lesion grade and edges and also to identify those suitable for repair.[16]

Numerous cartilage defects classification systems are in place including Insall, Outerbridge, Beuer and International cartilage repair society (ICRS) grading system.[9,11,17] ICRS grading system is more comprehensive and is increasingly used by the surgeons.[18]

The ICRS grading system is graded into 4 grades with each grade further subgraded to accurately evaluate the cartilage injury.

Grade 0 Normal (Fig. 1)
Grade 1 Superficial lesions
  A Soft indentations (Fig. 2)
  B Superficial fissures/cracks (Fig. 3)
Grade 2 Abnormal lesions extending down to <50% of cartilage depth (Fig. 4)
Grade 3 (Fig. 5)