Emerging Options for Treatment of Articular Cartilage Injury in the Athlete

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INCIDENCE OF ATHLETIC CARTILAGE INJURY

Injuries of the articular cartilage surfaces of the knee are frequently observed in athletes. Although no study has systematically investigated the incidence of sports-related articular cartilage injury, an increasing number of chondral injuries in high-impact sports has been observed, particularly at the competitive collegiate, professional, and world-class level.[1] Besides this rising incidence in high-level competitive sports, increasing recreational participation in pivoting sports such as football, basketball, and soccer has been associated with a rising number of sports-related articular cartilage injuries.[2–4] Injuries of the articular cartilage surface of the knee in the athlete frequently result in association with other acute injuries, such as ligament or meniscal injuries, traumatic patellar dislocations, and osteochondral injuries.[4–6] Articular cartilage defects of the femoral condyles have been observed in up to 50% of athletes undergoing anterior cruciate ligament reconstruction, with increased propensity in female athletes.[6,7] Besides acute injury, articular cartilage defects can develop in the high-impact athletic population from chronic pathologic joint-loading patterns, such as joint instability or malalignment.[4–6] Irrespective of their origin, articular cartilage injuries in athletes will frequently limit the ability of the affected athletes to continue participation in their sport and predispose them to progressive joint degeneration.[8,9]

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NATURAL HISTORY OF ATHLETIC CARTILAGE INJURY

The limited spontaneous repair following acute or chronic articular cartilage injury is well documented.[10–12] The lack of vascularization of articular cartilage prevents the physiologic inflammatory response to tissue injury and resultant repair. This failure of recruitment of extrinsic undifferentiated repair cells combined with the intrinsic inability for replication and repair by the mature chondrocytes results in a repaired cartilage that is both qualitatively and quantitatively insufficient. Furthermore, repetitive loading of the injured articular cartilage, such as in impact and pivoting sports, results in further cellular degeneration with accumulation of degradative enzymes and cytokines, disruption of collagen ultrastructure, increased hydration, and fissuring of the articular surface. These biochemical and metabolic changes are similar to the changes seen in early osteoarthritis.[13]

Although experimental studies have provided much insight into the mechanisms involved in the progression of cartilage injury to osteoarthritis, there is still limited prospective clinical information about the natural history of articular cartilage lesions, particularly in athletes. In a long-term study of 28 Swedish athletes with isolated severe chondral damage in the weight-bearing condyles, 75% of athletes returned to their sport initially, but a significant decline in athletic activity was observed 14 years after the initial injury, with radiographic evidence of osteoarthritis in 57% of these athletes.[14] Similar poor results were reported in a prospective study of untreated osteochondral defects in 38% of athletically active patients, with moderate to severe radiographic evidence of osteoarthritis in 45% at 34 years after diagnosis.[15] One recent report in athletes with anterior cruciate ligament (ACL) injuries demonstrated that hyaline cartilage defects in these patients resulted in significant pain and swelling and were associated with marked lifestyle changes and limitation of athletic activity.[16] Similarly, other studies have shown that untreated articular cartilage defects in patients with ACL deficiency resulted in significantly worse outcome scores up to 19 years after the original injury.[17] These results are supported by the 4- to 5-fold increased risk of knee osteoarthritis in high-demand, pivoting athletes established by the National Institute of Health (NIH) and in several other independent studies.[8,9,18–21]

ATHLETIC ACTIVITY AND CHONDROPENIA

Intact articular cartilage possesses optimal load-bearing characteristics and adjusts to the level of activity. Increasing weight-bearing activity in athletes and adolescents has been shown to increase the volume and thickness of articular cartilage.[22] In the healthy athlete, a positive linear dose–response relationship exists for repetitive loading activities and articular cartilage function. However, recent studies indicate that this dose–response curve reaches a threshold and that activity beyond this threshold can result in maladaptation and injury of articular cartilage.[23] High-impact joint loading above this threshold has been shown to decrease cartilage protoglycan content, increase levels of degradative enzymes, and cause chondrocyte apoptosis.[12,13,24] If the integrity of the functional weight bearing unit is lost, either through acute injury or chronic microtrauma in the high-impact athlete, a chondropenic response is initiated, which can include loss of articular cartilage volume and stiffness, elevation of contact pressures, and development or progression of articular cartilage defects. Concomitant pathologic factors, such as ligamentous instability, malalignment, meniscal injury, or deficiency, can further support progression of the chondropenic cascade. Without intervention, chondropenia contributes to the deterioration of articular cartilage function in high-impact athletes and may ultimately progress to osteoarthritis.