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Advances and Current Concepts of Cartilage Repair in the Patellofemoral Joint

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The patellofemoral (PF) joint poses a unique challenge to the cartilage surgeon owing to its physiologically high forces (compression and shear) and multiple degrees of mobility. The recognition of malalignment and instability in patients with chondral lesions, is imperative and plays a significant role in the cartilage treatment algorithm. The goal of PF restoration is to return the articular surface as close to the natural state as possible while correcting malalignment to prevent abnormal loading and at the same time effecting a stable environment. Although cartilage repair outcomes in the tibiofemoral joint have improved over the years, the PF joint reported outcomes remain more variable. Many authors continue to work to optimize comfort and function at the PF joint with specific emphasis on malalignment, stability, and articular cartilage repair on the patella. Technological advances in microfracture, particulated juvenile cartilage allograft transplantation, and autologous chondrocyte implantation are providing the necessary catalyst for research to include well-designed studies, which focus on the PF joint. The purpose of this article is to inform the reader about the current state of the art in chondral repair using cell-based approaches, beyond osteochondral allografts, which are discussed elsewhere in this issue.

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Introduction

The patellofemoral (PF) joint, just as the tibiofemoral joint, is prone to chondral injuries.[1,2] Chondral defects in the PF joint can vary from small, defined defects of 1 patellar facet to large bipolar defects affecting the entire PF joint. There is no clear correlation of defect size to clinical symptoms, which makes the diagnosis and treatment of these defects difficult. In addition, PF defects, even more so than defects in the tibiofemoral joint, are often ill-defined, do not exist in isolation and have a complicated geometric pattern. Indeed, most patients (~95%) presenting with clinically symptomatic cartilage lesions in the PF joint do not fit clear-cut treatment criteria.[3] These articular cartilage injuries are common, occurring from acute traumatic injuries, osteochondritis dissecans, early degenerative changes or overload, or avascular necrosis. Articular lesions located on all chondral surfaces throughout the knee are frequently encountered during routine arthroscopy with several studies reporting a prevalence between 57% and 62%.[1,2,4] The ranges of patella and trochlea cartilage lesions vary slightly, but have been reported to be present in 11%-36% in the patella and 6%-16% of the time in the trochlea.[1,5] Cartilage lesions are more prevalent in athletes, and PF defects have a prevalence of 37%, with 64% of these isolated to the patella.[6] Although these studies document the prevalence of lesions, they do not determine how many of those lesions are symptomatic clinically. Large cross-sectional studies have shown that the mere presence of a cartilage lesion does not coincide with symptomatic osteoarthritis; however, these lesions do lead to rapid progression of radiographic osteoarthritis.[7,8] Both prevention and diagnosis of clinically symptomatic articular cartilage lesions remain difficult and often rely on the process of "diagnosis of exclusion" as the defect and cartilage are aneural. That is, the pain response

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"caused" by the lesion is mediated through local bone, synovium, soft tissues, or nerves. It follows that the mere presence of a chondral defect does not constitute a clinically symptomatic defect requiring treatment. Cartilage lesions in the PF joint most frequently occur in conjunction with PF malalignment and frank patellar instability or both.[9] Henceforth, we will focus this article on cell-based treatment options for articular cartilage defects in the PF joint, whereas recommending concomitant procedures that are essential for their successful outcome. We will discuss traditional and established concepts along with newer options. These treatments will include microfracture with and without augmentation, DeNovo NT juvenile allograft chondrocyte implantation (Zimmer Inc, Warsaw, IN), and autologous chondrocyte implantation (ACI) first, second, and third generations as they pertain to the PF joint.

Microfracture With and Without Augmentation

Marrow stimulation techniques (MST) have been available for over 6 decades being recently popularized by Steadman as microfracture in the 1990s. These techniques were probably inspired by the early work of Pridie on marrow stimulation in the 1950s with later arthroscopic drilling and Johnson abrasion arthroplasty in the 1970s and 1980s. The described technique of marrow stimulation drilling has made a significant resurgence in the recent literature owing to the concern of possible subchondral bone vertical compression, weakening of the subchondral plate, and scarring that occurs when using a microfracture awl, as well as the obvious incitement of a bone healing response when the goal is cartilage repair. With arthroscopic techniques, the concern for heat necrosis with drilling has been shown not to be unwarranted.[10] Each technique, regardless of the specifics, such as use of Kirschner wires, drills, or microfracture awls, attempts to create channels of migration for mesenchymal stem cells through the subchondral plate (Fig. 1). MST are ideally indicated for full thickness articular cartilage defects (International Cartilage Repair Society grade 3), measuring less than 2 cm^2, on the femoral condyles. Steadman et al[11] have reported good results in all compartments of the knee with minimization of pain at follow-up. Mithoefer et al[12] evaluated over 3100 patients who underwent microfracture noting that all patients did on average better during the first 24 months postoperatively, but found deterioration in patient results in several studies at or approximately 18 months of follow-up. These data were corroborated by Goyal et al,[13] who did a critical review of 15 level I and II studies on microfracture. None of the studies analyzed focused solely on PF cartilage defects and although successful in the tibiofemoral joint, MSTs seem to be universally less effective on PF articular defects when taken as a separate group, with some studies such as Kreuz et al[14] reporting significant clinical failure of the procedure at the patella.[15] According to the inventor of the technique, favorable outcomes after MSTs are dependent on correct technical execution. This is particularly difficult in the PF joint and owing to the inverted position, patellar chondral lesions are difficult to access and visualize arthroscopically. Additionally, the subchondral bone of the patella is harder than in other parts of the knee joint making penetration with an awl more difficult. Orientation of the awl (even the 90° awl specifically designed for the PF surface) or the drill perpendicular to the subchondral bone can be tedious, sometimes leading to gouging rather than perpendicular piercing of the subchondral bone. Even when done open or mini-open these technical aspects are difficult to overcome. The microfracture depends on the formation of a blood clot, sometimes called the "superclot" as a scaffold for pluripotent mesenchymal cells from the marrow. Owing to the prone orientation of the articular surface it is difficult to contain this clot in the defect during the early rehabilitation phase and the number of pluripotent cells may be less in a small sesamoid bone compared with the femur. The problems with a suitable scaffold may be possibly addressed by using additional adjuncts such as fibrin glue and powdered cartilage

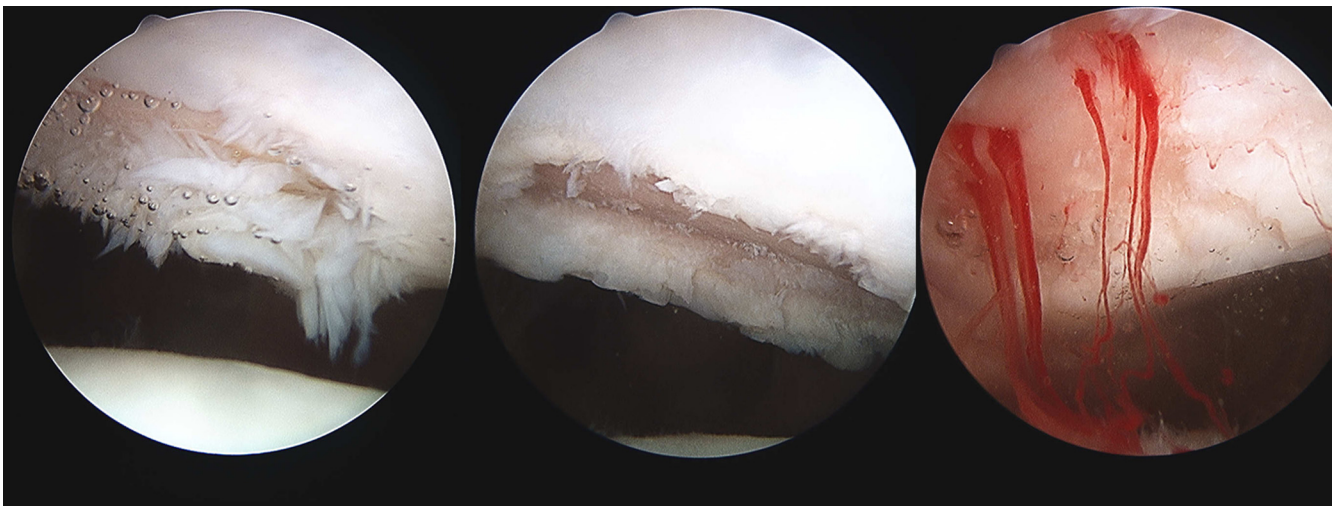

Figure 1 Debridement and microfracture of an isolated patellar defect with subsequent bleeding at the microfracture sites. (Color version of figure is available online.)

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