TREATMENT OF PATIENTS WITH INTRA-ARTICULAR FRACTURES OF THE KNEE JOINT

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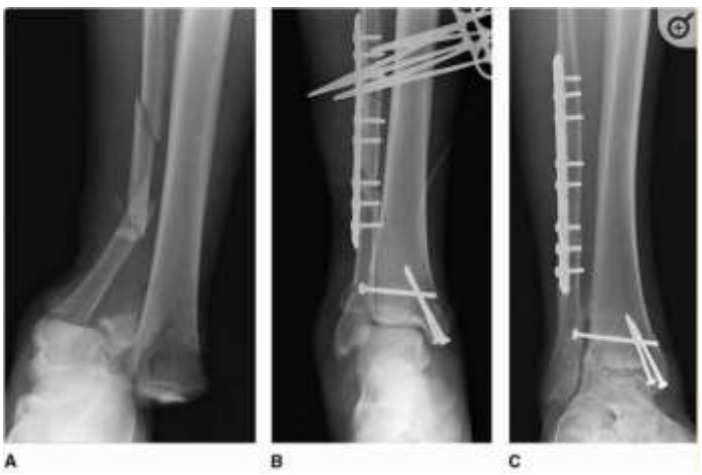
Abstract: Posttraumatic osteoarthritis (PTOA) occurs after traumatic injury to the joint. It is most common following injuries that disrupt the articular surface or lead to joint instability. The reported risk of PTOA following significant joint trauma is as high as 75%; articular fractures can increase the risk more than 20-fold. Despite recent advances in surgical management, the incidence of PTOA following intra-articular fractures has remained relatively unchanged over the last few decades.

Keywords: intra-articular fracture, knee joint, transverse osteosynthesis, restorative treatment, quality of life.

INTRODUCTION

Posttraumatic osteoarthritis (PTOA) occurs after traumatic injury to the joint and is most commonly associated with fractures that disrupt the articular surface or injuries that lead to joint instability (Figure 1). Approximately 12% of the overall symptomatic osteoarthritis burden can be attributed to PTOA of the hip, knee, or ankle, and the annual healthcare costs associated with the disease in the United States is approximately $3 billion.

MAIN PART



Radiographs of the ankle demonstrating the progression of posttraumatic osteoarthritis over 2 years. A, AP radiograph of the ankle demonstrating a fracture-dislocation. B, Postoperative AP radiograph of the ankle following fracture fixation. C, Follow-up AP radiograph of the ankle demonstrating severe degenerative

changes at 2 years postoperatively.

The risk of PTOA following significant joint trauma has been reported to be as high as 75%; articular fractures can increase the risk by more than 20-fold (Table 1). Despite changes in surgical management, including improved fracture fixation and management of chondral injuries, the incidence of PTOA following intra-articular fractures has remained relatively unchanged over the last few decades.

TABLE 1

Reported Risk of Posttraumatic Arthritis by Joint Involved

Joint	Risk of Posttraumatic Arthritis (%)
Tibial plafond	70–75
Tibial plateau	23–44
Distal radius	24–65
Distal femur	23–35
Distal humerus	44
Acetabulum	11–38

RESULTS AND DISCUSSION

The mechanisms of injury and factors that contribute to the development of PTOA following intra-articular fractures are not well understood; therefore, the ability to intervene clinically and delay or prevent the progression of PTOA is limited. Current data suggest that multiple factors contribute to the development of PTOA, including acute mechanical injury to the cartilage at the time of impact, biologic response (eg, bleeding, inflammation), and chronic cartilage overload secondary to incongruity, instability, and malalignment. Other factors, including patient age and injury severity, also may contribute to worse clinical outcomes and progressive degeneration following intra-articular fractures.

One of the proposed mechanisms that trigger this cascade is the release of reactive oxygen species and/or proinflammatory mediators following injury, which may lead to progressive chondrocyte damage and matrix degeneration. Some in vitro studies of impact injuries on cartilage explants showed that injury induced the release of oxygen free radicals from chondrocytes, possibly secondary to mitochondrial injury, which led to chondrocyte death and matrix degeneration. Severe high-impact injuries have been shown to result in greater local tissue damage (as measured by a higher proportion of cells releasing reactive oxygen species) and a higher rate of chondrocyte death and matrix disruption than that associated with less severe injuries. Intra-articular fracture has also been shown to result in elevated synovial levels of proinflammatory cytokines and mediators, including tumor necrosis factor-α, interleukin (IL)-1, nitrous oxide, matrix metalloproteinases, and fibronectin fragments that can stimulate further cell and matrix degradation.

CONCLUSION

The development of PTOA after intra-articular fracture is likely multifactorial and may be the combined result of initial cartilage injury and the associated chondrocyte death, matrix disruption, and release of proinflammatory cytokines and reactive oxygen species as well as chronic joint overload secondary to instability, incongruity, and malalignment. Additional studies are needed to better elucidate the relative contributions of these factors in the development of PTOA and to develop advanced treatment algorithms. Based on available clinical data, future treatment modalities may consist of acute biologic interventions targeted at decreasing inflammation and cellular death in response to injury as well as improved surgical methods to better restore stability, congruity, and alignment following intra-articular fractures to reduce the individual and societal burden of PTOA.

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