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Review Article

Arthrofibrosis in ATR

Jesús Ignacio Cardona Medina*, Jesús Ignacio Cardona Muñoz and Ariel de la Rosa Guerrero

Orthopedic Surgeon Knee Surgery, Center for Orthopedics and Sports Medicine (CETRAMED), Jalisco, CP, Mexico

***Corresponding Author:** Jesús Ignacio Cardona Medina, Orthopedic Surgeon Knee Surgery, Center for Orthopedics and Sports Medicine (CETRAMED), Jalisco, CP, Mexico.

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Abstract

Stiffness is a relatively uncommon complication after total Knee Arthroplasty. It has been defined as a painful limitation in the range of movement (ROM) Its pathogenesis is still unclear even if some risk factors have been identified. Patient-related conditions may be difficult to treat. Preoperative ROM is the most important risk factor, but an association with diabetes, reflex sympathetic dystrophy, and general pathologies such as rheumatoid arthritis and ankylosing spondylitis has been demonstrated. Moreover, previous surgery may be an additional cause of an ROM limitation. Postoperative factors include infections, arthrofibrosis, heterotrophic ossifications, inadequate control of postoperative pain and incorrect rehabilitation protocol. Finally, surgery-related factors represent the most common cause of stiffness; they include errors in soft-tissue balancing, component malpositioning and incorrect component sizing. Although closed manipulation, arthroscopic and open arthrolysis have been proposed, they may lead to unpredictable results and incomplete ROM recovery. Revision surgery must be proposed in the case of well-documented surgical errors. These operations are technically demanding and may be associated with high risk of complications; therefore, they should be accurately planned and properly performed.

Keywords: *Arthrofibrosis; Rigidity Post-operative; Total Knee Arthroplasty; Arthroscoritching Arthrosis*

Overview

The main objective of a Total Knee Arthroplasty (ATR) is to relieve pain and restore knee joint function. To achieve adequate postoperative function the knee must be stable and have an adequate range of mobility (RDM). Fortunately, joint stiffness in ATR is a rare complication, with an incidence of 1.3 - 5.3% [1], however it results in severe disability for the patient and frustration for both the patient and the surgeon when it occurs. One of the frequent causes of stiffness after an ATR is arthrofibrosis with an incidence of 1.2 to 17%. It is an entity characterized by the loss of joint mobility due to severe intra and peri-articular proliferation of fibrous tissue, particularly type I, III collagen. and IV. Histopathological findings show the development of a metaplasia consisting of calcified tissue, myofibroblasts and excessive fibrosis, adding to a synovial membrane hyperplasia and an increase in the number of macrophages and lymphocytes in the periarticular tissue. Although there is no gold standard for diagnosis, physical examination is currently considered the clearest way to diagnose it. Recently, inflammatory markers have been identified that could be auxiliary in the diagnosis of arthrofibrosis such as Growth Transforming Factor B (FTC-B), which plays an important role in the pathophysiology of the disease. Bone morphogenetic protein (BMP-2) is also identified [2].

There is usually a previous event of surgery, trauma, infection or prolonged immobilizations. There are different clinical forms of arthrofibrosis that have been classified by Shelbourne (Table 1) depending on the clinic and the degree of severity [3]. Its treatment rep-

resents a therapeutic challenge that requires an immediate and aggressive rehabilitation protocol adding to an adequate management of postoperative pain. However, if in the short term such treatment fails or it is not possible to have a desired range of mobility, more aggressive treatments such as MBA or arthroscopic arthrolysis should be considered.

Types	Characteristics
I	< 10º loss of extension and normal flexion
II	> 10th loss of extension and normal flexion
III	> 10º extension loss and> 25º flexion loss, patellar mobility limitation, without low patella
IV	> 10º extension loss and> 25º flexion loss with low patella.

Table 1: *Shelbourne arthrofibrosis classification.*

We conducted a retrospective study of our cases with postoperative stiffness in ATR where patients who developed type III arthrofibrosis of the Shelbourne classification were included, including cases that did not respond favorably to non-surgical treatment and were treated by arthroscopic arthrolysis. All patients were operated by the same surgical team and completed the same postoperative rehabilitation program with a 24-month postoperative follow-up. Patients with a diagnosis of arthrofibrosis type I-II-IV, patients with regional painful syndrome or without a clear diagnosis were excluded. For this study, 12 patients were included who met the inclusion criteria that we had performed an arthroscopic arthrolysis after an ATR during the period from January 2009 to December 2016. 7 were women and 5 men. The average age 65 years (range 56 - 81 years) with a post-operative follow-up at 3, 6, 12 and 24 months. The average pre-surgical mobility range was 15º/ 50º, being the loss of extension by inclusion criteria greater than 10º in all patients and the loss of flexion greater than 25º.

All patients underwent the same arthroscopic surgical procedure with sequential arthrolysis of the knee compartments and in the cases that were required, the medial posterior capsulotomy was performed by accessory post-medial portal. All patients underwent the same rehabilitation program and pain management protocol with peridural catheter and intravenous analgesia. All patients were installed with a continuous passive mobilization machine in the first 24 hours after surgery and a rehabilitation program with patellar mobilization and extension and assisted flexion exercises in the first 72 hours. We perform an analysis with pre and post-operative evaluation with WOMAC functional scale. The visual analog scale was used for post-operative pain and the IKDC range of mobility was used (< 3º, 3 - 5º, 6 - 10º and 10º) to assess the loss of the extension. The average post-operative passive mobility arc increased extension / flexion (5º/110º) compared to passive preoperative extension/flexion mobility (15º / 50º). The WOMAC pre-operative average functional scale was 43 and the post-operative improved significantly to 75. Regarding the classification of IKDC, it showed a loss of the extension 6 cases < 3º, 3 cases 3 - 5º, 2 cases 6 - 10º and no case > 10º flexion contracture. On average the VAS pain was 4/10 in post-operative pain.

Post-ATR arthrofibrosis is characterized by the excessive formation of scar tissue and fibrous adhesions that restrict the range of mobility involving different anatomical structures and compartments of the knee. Although its incidence is low (< 5%), it is postulated that it may be due to a deregulation in the cascade of inflammatory events, combined with the abnormal local increase in growth factors (platelet growth factor, fibroblastic growth factor, insulin type I and growth factor beta transformation), also recently high concentration of type VI collagen has been isolated, suggesting disorder in the regulation of its synthesis. These processes could be secondary to infection, hemarthrosis, prolonged immobilization, post-surgical trauma. Holger has also proposed a genetic basis for said inflammatory disorder [4].

Histological analysis of the synovial membrane reveals chondrometaplasia with dense fibrovascular tissue, less frequent findings with hemosiderin deposits, endochondral ossification and bone necrosis. In the pathophysiology of this disorder, fibrous proliferation leads

to progressive loss of mobility and irreversible damage. Quadriceps atrophy and flexion contracture aggravates the picture [5-7]. Van Eijden postulated in mathematical models an increase in the anterior translational forces of the tibia during the extension produced by the shortening of the patellar tendon, with an increase in the quadricipital force necessary for the extension which generates an increase of patellofemoral contact pressures the same effect produced by arthrofibrosis with the adhesions of the patellar tendon aggravates the chondral lesions [8]. This biomechanical alteration would be one of the causes of the association between arthrofibrosis and degenerative changes. Cosgarea observed degenerative changes in 89% of patients. Waugh desbribe 82% of degenerative changes in knees that show flexion contracture compared to 9% in those who achieved full extension, recommending that flexion contracture should be treated immediately and the extension restored using all possible techniques. This could be related to early loosening of the implants due to alteration of the loads [9-1].

The treatment of arthrofibrosis in ATR has clear objectives that are to restore the mobility arc to improve function and avoid overload that generates an early loosening of the implants especially when patella is replaced. Generally, non-surgical treatments are insufficient to restore the mobility arch. On the other hand, mobilization under anesthesia can have complications such as fractures and injuries of the extensor mechanism. In addition, it is not possible to dry the adhesions under this technique and extraction of foreign bodies. Arthroscopic evaluation allows direct vision of adhesions as well as resection and removal of foreign bodies [1,12,13].

The loss of flexion after an ATR is associated with intra-articular adhesions mainly in the extensor apparatus such as adhesions in suprapatellar recess, patellar aileron fibrosis as well as in the medial and lateral recesses. The anterior region can also be affected at the level of infrapatellar adipose tissue with adhesions of the patellar tendon to the Hoffa pouch [14-16]. Loss of extension in ATR is associated with adhesions at the level of intercondylar notch, but mainly the retraction and fibrosis of the posterior capsule is associated. However, there is no consensus on when it is the ideal time for surgery, it is recommended to do so before three months after surgery to prevent the process from becoming irreversible [17].

We only included patients with type III arthrofibrosis of the Shelbourne classification, all patients presented extension and flexion deficits with significant adhesions in the extensor apparatus that limited the range of motion. In all cases, an arthroscopy was performed with debridement and resection of patellofemoral adhesions and the suprapatellar space using shaving and radiofrequency to break adhesions, as well as in lateral and medial recess and in the anterior region. It is a difficult procedure since care must be taken not to damage the implants, mainly polyethylene. It is essential to recover the entire extension intraoperatively if it is not achieved, it should be assessed to perform posterior capsulotomy through the medial postero portal or with an open technique since, no matter how aggressive the rehabilitation program is, it will be difficult to recover what was not achieved in the operating room. Recovery should be prioritized for full extension (0º), even hyperextension, given that with only 5º of flexion contracture, it may be sufficient to alter gait and generate a patellofemoral syndrome while loss of flexion to a lesser degree (< 10º) does not alter ordinary activities. We can help ourselves in the post-operative with the continuous passive mobilizer and it is essential to mobilize the patella.

Given that there is really no evidence of the predisposing factors or the pathophysiology of arthrofibrosis, it is difficult to compare the results with the literature, in addition to the fact that there are very few studies and the number of patients in each study, as well as these, there is little that makes them Statistically not validable. When analyzing our results we found an increase in the range of mobility both in the extension (from 15º to 5º average) and in the flexion being more significant the improvement in the flexion (50º to 110º average), in the WOMAC functional scale there was an improvement However, we see that the average is still low, which we can interpret that although there was clinical improvement in the patient there is still limitation and dissatisfaction in the patient. In our experience, although there are few cases, arthroscopic arthrolysis may be a tool to obtain good functional results in the recovery of flexion in arthrofibrosis, however in flexion contracture a posterior capsulotomy may be required to achieve the goal of total extension of the knee.

Conclusion

In conclusion, I consider that prevention is the best treatment to prevent arthrofibrosis, from the appropriate selection of patients with identification and control of systemic inflammatory diseases, their range of pre-operative mobility given the expectations of the patient and the surgeon to achieve full extension, Avoiding unnecessary post-operative immobilization, adequate post-operative pain control and specific rehabilitation therapy programs can decrease its frequency and avoid its severity since this can be a tragedy for the patient and headache for us as surgeons.

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