Arthrofibrosis After Total Knee Arthroplasty

Pathophysiology, Diagnosis, and Management

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KEYWORDS

• Total knee arthroplasty • Arthrofibrosis • Manipulation under anesthesia
• Arthroscopic debridement • Quadricepsplasty

KEY POINTS

- Arthrofibrosis is the pathologic stiffening of a joint caused by an exaggerated inflammatory response causing hyperplasia of the connective tissue around the knee.
- Arthrofibrosis following total knee arthroplasty can cause significant knee pain and restricted range of motion, severely hindering postoperative rehabilitation and basic activities of daily living.
- Disease prevention is most successful and is accomplished with preoperative patient education programs, aggressive postoperative physical therapy regimens, and anti-inflammatory medications.
- When necessary, operative techniques, including manipulation under anesthesia, arthroscopic debridement, and quadricepsplasty, can be used with varying degrees of success.

INTRODUCTION

Arthrofibrosis is the pathologic stiffening of a joint caused by an exaggerated inflammatory response. Proliferation of metaplastic fibroblasts and the excessive deposition of extracellular matrix (ECM) proteins lead to the development of thick, noncompliant, fibrous scar tissue.[1,2] As a common complication following total knee arthroplasty (TKA), this benign-appearing connective tissue hyperplasia is a cause of significant disability among patients, because the concomitant knee pain and restricted range of motion (ROM) severely hinder postoperative rehabilitation, clinical outcomes, and basic activities of daily living (ADL).[1,3,4] It is conservatively estimated that nearly 85,000 cases of arthrofibrosis occur following knee surgery in the United States per annum, with 25% of these cases requiring additional surgery in an attempt to restore adequate knee motion.[5] Moreover, for patients undergoing TKA, arthrofibrosis is estimated to be responsible for 28% of 90-day hospital

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readmissions and 10% of revision surgeries within the first 5 years, placing a significant burden on societal costs.[1,6] This article provides a comprehensive review of the pathophysiology of arthrofibrosis following TKA, associated risk factors, diagnostic pearls, and current management strategies in the published literature.

PATHOPHYSIOLOGY OF ARTHROFIBROSIS

Fibroblasts are the most abundant cells of the connective tissues.[1] Through their production and maintenance of the ECM's repertoire of structural, adhesive, and ground substance proteins, fibroblasts are a heterogeneous population of cells that play a major role in tissue development, architecture, and local cellular differentiation.[1,7] In the setting of tissue injury, as in TKA, fibroblasts become a vital component of scar tissue formation. In the early phases of wound healing, fibroblasts from the adjacent tissue layers produce a rich supply of collagen and adhesive proteins while providing the tractional forces needed to close the wound.[8] The tractional forces can then simulate fibroblasts to differentiate into protomyofibroblasts, upregulating the production of stress fibers.[7,8] It has been well documented that fibroblasts are also intimately linked with the inflammatory and immune response pathways, making them responsive to a wide array of inflammatory cytokines, including transforming growth factor (TGF) β1, interleukin (IL)-1β, IL-6, IL-13, IL-133, prostaglandins, and leukotrienes.[9–11] These signals induce protomyofibroblasts to undergo myofibroblastic differentiation, further promoting wound contraction and the upregulation of ECM protein production.[7–9] Concurrently, fibroblasts secrete cellular signaling factors, including reactive oxygen species, TGFβ1, IL-1β, IL-33, and CXC and CC chemokines, to promote immune cell extravasation and migration to the site of injury and inflammation. Healing of the wound is typically marked by the subsidence of inflammation and disappearance of myofibroblasts, most often through apoptosis.[7,12] However, aberrant inflammatory–wound healing interactions are thought to be the source of chronic inflammation and pathologic arthrofibrosis.[7,8,11]

DIAGNOSIS

Arthrofibrosis is diagnosed primarily on clinical assessment and ultimately confirmed with histopathologic analysis.[13] The impermeability of the joint synovium precludes the systemic circulation of potential serum-based biomarkers.

Following TKA, a low index of suspicion should be held for arthrofibrosis, particularly among patients with clinically significant loss of knee extension and/or flexion (<90° of passive flexion and <10° of full extension).[14] Although physical examination findings show substantial extension and flexion ROM deficits, the loss of knee extension is more disabling for the patients.[15] Additional findings include anterior knee pain, flexed-knee gait, quadriceps weakness, and patellofemoral painful crepitation.[16–18] Furthermore, diffuse edema, warmth, tenderness localized to the fat pad, and limited patellar mobility are characteristic findings.[16] A firm, nonfluctuant and nonedematous knee with limited patellofemoral mobility and a low-lying patella (patella baja) are also supportive findings for arthrofibrosis.[19] In contrast, a stiff knee with appropriate patellofemoral mobility typically places arthrofibrosis lower on the differential diagnosis, and may be suggestive of other disease processes, such as component malpositioning or lack of proper soft tissue balancing.[14]

Although there are no widely accepted diagnostic criteria for arthrofibrosis, there have been several attempts at defining it based on ROM deficits. Shelbourne and colleagues[19] characterized and graded arthrofibrosis into 4 categories (types 1–4) based on loss of flexion and extension ROM compared with the native contralateral knee (Table 1). More recently, Mayr and colleagues[20] defined arthrofibrosis as the presence of scar tissue in any compartment of the joint leading to restricted ROM.

Imaging

Diagnostic imaging is a useful tool in the diagnosis of arthrofibrosis.[21] Advancements in metal artifact

Table 1
Classification of arthrofibrosis based on degree of extension and flexion loss

Type	Extension and Flexion Deficit
1	<10° of extension loss in the absence of flexion loss
2	>10° of extension loss in the absence of flexion loss
3	>10° of extension loss and >25° of flexion loss with a tight patella
4	>10° of extension loss, ≥30° flexion loss, and patella baja with marked patellar tightness

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reduction sequences (MARS) for MRI can significantly reduce artifacts generated from implanted metal components, allowing physicians to assess the soft tissue within the periarticular region with high resolution.[22] In patients with stiff knees, MARS-MRI identification of nonparticulate densities of low-intensity and intraarticular adhesions within the knee are highly suggestive for arthrofibrosis.[23,24] Periarticular ultrasonography may also be a useful modality in assessing fibrotic tissue around the knee. A study performed by Boldt and colleagues[25] evaluated sonographic findings in patients with arthrofibrosis following TKA. Synovial membrane thickness was increased and the Hoffa fat pad was more pronounced; however, there were no differences among case and control cohorts with regard to the size of the joint effusion and patellar tendon thickness. The investigators concluded that synovial membrane thickening and neovascularity are unique sonographic findings that are suggestive of arthrofibrosis in the setting of TKA.[25] Radiographic examination can also identify indirect variables that may contribute to arthrofibrosis, including the use of plain film radiographs to determine joint-line elevation and computed tomography to evaluate femoral and tibial component malrotation.[14]

RISK FACTORS

There is a paucity of information evaluating patients' risk for developing arthrofibrosis, thus this article briefly summarizes the relevant risk factors, with attention to preoperative and postoperative variables predisposing TKA candidates to arthrofibrosis (Table 2).

Preoperative Risks

Several risk factors place patients at an increased risk for post-TKA stiffness, including previous knee surgery,[26] smoking,[27] diabetes mellitus,[27–29] and preoperative ROM.[30] Of these, preoperative ROM remains the most important.[30] In a study by Lizaur and colleagues,[31] patients with a preoperative flexion less than 90° had an average post-TKA flexion of 88°, significantly lower than the average 103° of flexion in patients with a preoperative flexions greater than 90°. The ability to walk, climb stairs, run, sit in a chair, and perform the most basic ADLs requires 10° to 120° of active knee flexion and is considered a tolerable ROM following TKA.[5] Accordingly, arcs of flexion less than 90° after TKA have been shown to correlate with significant patient frustration and dissatisfaction.[32]

Patient motivation and state of mind also play a critical role in patients' participation in physical rehabilitation after TKA. In a study by Fisher and colleagues,[33] patients who were depressed or had a low pain tolerance were less likely to properly perform rehabilitation activities, resulting in delayed recovery and an increased likelihood of developing arthrofibrosis.

Despite limited research into the genetic factors driving arthrofibrosis, possible correlations have been found between specific human leukocyte antigen (HLA) subtypes.[2] Skutek and colleagues[2] found an association between postoperative arthrofibrosis and patients with negative HLA-Cw*07, negative DQB1*06, and positive HLA-Cw*08 haplotypes. However, these findings were performed in patients following autologous anterior cruciate ligament reconstruction, and were limited to a small sample size of 17 patients.

Table 2
Risk factors for development of knee arthrofibrosis following total knee arthroplasty

Perioperative Period	Risk Factor
Preoperative	Limited preoperative ROM
	History of previous knee surgeries
	Smoking
	Systemic disease (eg, diabetes)
	Patient state of mind; depression
	Genetic predisposition
Intraoperative	Inappropriate soft tissue balancing
	Component malpositioning
	Incorrect component sizing
	Excessive femoral component hyperflexion
	Excessive patellofemoral thickness
	Incorrect joint-line height
	Errors in bony resection
Postoperative	Length of immobilization
	Infection
	Complex regional pain syndrome

Surgical Risks

Iatrogenic surgical errors, such as improper soft tissue balancing, component malpositioning, and incorrect component sizing, are a common cause of postoperative stiffness following TKA.[30,34] More specifically, these surgical errors can include an inappropriately tightened posterior cruciate ligament (PCL),[35,36] excessive femoral component hyperflexion,[31] and errors in bony resection leading to so-called component overstuffing[37] and inappropriate joint-line elevation.[38,39] These surgical errors may lead to alterations in normal knee kinematics, resulting in repetitive microtrauma that is thought to trigger an inflammatory response with subsequent progression to arthrofibrosis.[39]

It is important to address methods for preventing these surgical errors in the hope of decreasing the risk for arthrofibrosis. In patients with a fixed varus deformity greater than 15°, the PCL is likely to be engaged and tight, resulting in reduced anterior tibial translation during femoral rollback, a concomitant increase in anteromedial tibial contact pressures, and a reduced flexion ark of the knee.[30,39–41] In the setting of posterior cruciate–retaining prostheses, it may be beneficial to partially release the PCL. However, caution should be exercised to not over-release the PCL because it may result in a paradoxic roll forward and anterior displacement of the femoral component, causing posterior impingement and tightening of the extensor mechanism, effectively limiting flexion.[30] Alternatively, the surgeon can opt to resect the PCL and use a posterior stabilized construct instead.

Femoral components that are placed in hyperextension or hyperflexion can limit the knee's ability to fully flex and extend, respectively.[39] To avoid excessive sagittal rotation of the femoral component, proper use of intramedullary guides or navigation systems is essential to align the femoral component with respect to the proper axis of the femur.[39,42] For axial alignment in a measured resection technique, the femoral component axis should be placed parallel to the epicondylar axis and the tibial component with the middle one-third of the tibial tubercle.[43] In the gap balancing method, femoral rotation is set parallel to the tibial cut, after the extension gap is balanced, to recreate a rectangular space, matching extension gap.[44,45]

Component overstuffing in TKA occurs when the inserted implants create a suboptimal flexion, extension, or patellofemoral space leading to reductions in the joint's arc of motion.[42] To avoid component overstuffing and inappropriate joint-line positioning, adequate tibial resections that position the joint line 1 cm proximal to the fibular head or 2 cm distal to the medial epicondyle are suggested. Furthermore, insufficient patellar resection can lead to a thick patellar bone–implant construct and cause tightness in flexion.[43] Errors in distal and posterior femoral resection can also lead to inappropriate flexion and extension gaps, further hindering postoperative knee ROM.[43] Femoral resections should be designed to restore neutral mechanical alignment with 3° to 6° valgus, and 0° to 4° of flexion.[43]

Postoperative Risks

Postoperative management is as important as proper surgical technique in preventing knee arthrofibrosis. Early motion of the knee has been hypothesized to reduce the incidence of postoperative arthrofibrosis through the breakdown of existing scar tissue, inhibition of fibrotic deposition, and adhesion formations.[46,47] It has therefore been proposed that postoperative physical therapy (PT) protocols emphasizing early motion may reduce arthrofibrosis of the knee.[39]

NONOPERATIVE MANAGEMENT

Preoperative Education

Preoperative patient education (PPE) programs are designed to improve patient adherence and outcomes through patient motivation, the encouragement of patients to take an active role in their health before and after TKA.[48] In doing so, patients are educated on proper techniques of home exercises and outpatient rehabilitation, while simultaneously setting realistic functional expectations. Patient participation is of particular value because most of these protocols include programs that demand a high degree of patient-led therapy. In a case-control study using the Danish Knee Arthroplasty Registry, PPE was associated with a decreased risk of arthrofibrosis following TKA (odds ratio, 0.16; $P = .02$).[49]

Physical Therapy

Many studies have shown clear evidence correlating decreased preoperative ROM and an increased risk for poor postoperative ROM following TKA.[29,31,50] In a randomized controlled trial (RCT) of 131 patients undergoing TKA, Beaupre and colleagues[51] evaluated the utility of a 4-week combined PPE and exercise program in improving postoperative knee ROM and function. Postoperative functional recovery was equivocal in patients who

underwent this program compared with patients who did not, further disputing the effectiveness of preoperative PT.[51] It is possible that the surgery itself negates any benefits derived from preoperative PT or that the improvement in pain and function as a result of the TKA overshadow the modest improvements achieved from preoperative PT.[48,52]

Patients with arthrofibrosis after TKA pose a unique challenge to physical therapists because they require rehabilitation that focuses on restoration of ROM and the management of inflammation, pain, and swelling.[21] In the absence of arthrofibrosis, post-TKA rehabilitation tends to place more stress on building quadriceps muscle strength; however, in the setting of arthrofibrosis, it is important to prioritize targeting ROM deficits promptly because the fibrotic tissue can mature and develop resistance to exercise.[53,54] Treating ROM deficits requires a high level of patient compliance and activation, necessitating optimal pain management and an aggressive PT regimen.[55] Labraca and colleagues[56] showed that PT that began within 24 hours following TKA was associated with greater joint ROM in flexion (16.29° ± 11.39°; $P = .012$) and extension (2.12° ± 3.19°; $P = .035$). Although PT should be aggressive to achieve optimal ROM, it is important to avoid an overly aggressive protocol because it can precipitate an inflammatory reaction that can worsen pain and further joint contracture or cause patella fracture or tendon rupture.[39]

The use of continuous passive motion (CPM) machines has been a debated topic regarding the prevention of knee stiffness following TKA because there has been inconclusive evidence of its ability to improve ROM and reduce the need for manipulation under anesthesia (MUA).[57] In a Cochrane Review of 24 RCTs with a cumulative 1445 TKA patients, CPM enacted only a modest difference in active knee flexion.[58] The gain in ROM was clinically insignificant because the mean active ROM in patients without CPM was 78° compared with 80° in patients using CPM machines. In addition, Boese and colleagues[57] conducted a 160-patient RCT evaluating the ability of CPM devices to improve postoperative ROM. The investigators compared outcomes among a group of patients who received a CPM device moving from 0° to 110°, a group of patients who received a CPM device that was fixed at 90° in flexion, and a group of patients who did not receive a CPM device.[57] There was no difference found between the 3 groups with regard to postoperative ROM, further disputing the benefit of using CPM in TKA patients to improve knee ROM.[57]

In cases in which PT fails to improve arthrofibrosis, noninvasive assistive devices, such as various knee orthotics, have shown promise.[59–61] The hinged metal brace uses the principle of static progressive stretching, a technique that holds the joint at a position near the end of ROM followed by incremental increases in displacement between the thigh and leg over time.[59] Bonutti and colleagues[59] reported on outcomes in 25 patients who were refractory to PT and were treated with this device. After a median treatment interval of 7 weeks, the investigators showed a median 25° (range, 8°–82°) increase in ROM, median 19° (range, 5°–80°) increase in knee active flexion, and 92% satisfaction in results among the patients.[59]

Antiinflammatories and Other Medications

Although the exact cause of arthrofibrosis is poorly understood, a strong relationship with inflammatory markers, postoperative pain, and pain during rehabilitation has been observed.[9–11,62,63] Multiple studies have correlated increased perioperative pain with arthrofibrosis and decreased ROM in total joint arthroplasty patients.[64–67] Therefore, a multimodal approach to decreasing inflammation and controlling pain can improve patient mobilization and prevent arthrofibrosis.

Nonsteroidal anti-inflammatory drugs (NSAIDs) inhibit 2 of the most common inflammatory pathways, cyclooxygenase (COX)-1 and COX-2, effectively decreasing prostaglandin synthesis. Although, prostaglandins do not directly mediate pain, they perpetuate the inflammatory cycle and increase the excitability of nociceptors in injured tissues.[68] Feng and colleagues[69] noted significantly decreased levels of regional cytokines and leukocytes in knee drainage fluid from patients who received a preoperative dose of rofecoxib (a selective COX-2 inhibitor) compared with a placebo. In the control group, the total number of leukocytes increased approximately 2 to 4 times above the baseline, starting 2 hours after TKA, and continuing for 48 hours.[69] However, patients who received 25 mg of rofecoxib before surgery only showed a 2-fold increase in leukocytes. The measured levels of IL-6 and tumor necrosis factor alpha were also significantly lower in joint fluid by 50% to 60% up to 48 hours. Patients using rofecoxib showed improved pain scores at rest and with activity. At rest, the mean modified numerical pain rating scales were significantly lower in the rofecoxib group at 24 hours (0.3 ± 0.1 vs 0.9 ± 0.1; $P<.05$) and at 48 hours (0.1 ± 0.1 vs 0.7 ± 0.1; $P<.05$) following surgery.

A similar outcome was noted when patients were active in the rofecoxib group with lower pain scores at 24 hours (0.8 ± 0.5 vs 1.8 ± 1.2; $P<.05$) and 48 hours (0.7 ± 0.4 vs 1.6 ± 1.0; $P<.005$) following surgery.[69] Buvanendran and colleagues[70] found that patients who received postoperative rofecoxib were able to achieve higher joint ROM with shorter time in PT after TKA. A significant increase in both active (84.2° vs 73.2°; $P = .03$) and passive knee flexion (90.5° vs 81.8°; $P = .05$) was noted as early as discharge compared with the placebo group. Furthermore, mean flexion continued to be significantly superior in the patients receiving rofecoxib at 1 month postoperatively (109.3° vs 100.8°; $P = .01$). As such, perioperative NSAID use may reduce inflammation, minimize the pathogenesis of fibrotic scar formation, and decrease pain in the early rehabilitation phase, allowing more aggressive physiotherapy regimens following TKA.

In an effort rehabilitate TKA recipients, an emphasis has been placed on narcotic-sparing pain protocols, which include NSAIDs, acetaminophen, peripheral nerve blocks, long-lasting local anesthetics, periarticular infiltration, epidural infusions, oral and intravenous opioids, steroids, and anticonvulsants.[71–73] Lavernia and colleagues[66] retrospectively examined the effects of a multimodal pain management protocol, consisting of PPE, perioperative pain cocktails, femoral nerve blocks, and intraoperative analgesic injections, on outcomes following TKA. The investigators showed a reduced incidence of MUA from 4.75% (37 out of 778) to 2.24% (8 out of 357) compared with the traditional pain protocol, comprising patient-controlled analgesia pumps and opioid medications.[66] Ranawat and colleagues[74] described improved outcomes with their perioperative pain protocol for TKA patients. Using a multimodal protocol, Ranawat and colleagues[74] reported improved recovery and a minimum of 90° ROM with more than 85% of patients reporting 110° ROM. Furthermore, patients had higher rates of ambulation (98% vs 80%) and quicker recovery in PT starting on postoperative day 1. More recently, they have used a combination of epidural infusions and femoral nerve blocks with or without intravenous pain-control analgesia, as well as a transition from general to regional anesthesia. In the evolving Bundled Payments for Care Improvement environment, an increased use of pain services and a multidisciplinary team care approach has emerged as an important part of proper pain management following TKA.[65]

Supplemental cryotherapy is sometimes suggested in an effort to reduce swelling and inflammation.[75] It is a treatment that uses cold compression in the postoperative period and is thought to help with pain management, ROM, and knee function. In a recent RCT by Kullenberg and colleagues,[76] 86 patients were randomized to receive either cryotherapy or no treatment following TKA. The investigators showed that patients who received cryotherapy in the immediate postoperative period showed improved ROM measurements 3 weeks following TKA (98.9 vs 87.6; $P = .0045$), further underscoring its potential utility.

OPERATIVE MANAGEMENT

Manipulation Under Anesthesia

For TKA patients that fail PT and continue to experience functionally limiting knee flexion, MUA is the first-line operative treatment of choice.[77,78] Performed in the operating room, patients are placed under conscious sedation and maximal muscle relaxation is obtained.[77,79] The ipsilateral hip is subsequently flexed to 90° while the surgeon grasps the proximal third of the tibia and the knee is flexed slowly until audible and palpable separation of adhesions no longer occur. Use of the distal third of the tibia should be avoided to prevent excessive leverage on the joint and potential supracondylar fractures.[79]

In a prospective cohort study by Esler and colleagues,[80] patients who consented to MUA showed an average gain in active knee flexion of 33° that was sustained from 6 weeks to 1 year. In contrast, those who declined were only able to recover 3.1° of knee flexion ($P = .23$).[80] These findings are congruent with more recent studies and systematic reviews, which have reported a 30° to 47° recovery of knee flexion following MUA.[77,78,81] However, current indications for MUA vary between studies, with cutoffs ranging widely between 80° and 110°, potentially concealing the true ROM gained following MUA.[77,80] Clinically, patients should be evaluated on a case-by-case basis for functional limitations secondary to subjective stiffness and restricted knee ROM. Patient background, culture, and religion should also be accounted for because these factors may involve kneeling or cross-legged sitting, requiring deeper knee flexion angles. Although the risks and benefits of increased ROM should also be weighed against potential complications, such as supracondylar fractures and patellar tendon ruptures, their incidences have been inadequately reported in the literature.[81–83]

MUA timing also plays a critical role in the extent of knee flexion regained.[77,84] In a retrospective study by Issa and colleagues,[77] patients who had received MUA within a 12-week window after surgery showed a significantly higher recovery of knee flexion (36.5° vs 17°; $P<.0001$) and Knee Society objective (89 vs 84 points; $P<.05$) and function (88 vs 83 points; $P<.045$) scores than those after 12 weeks.[77] Subset analysis of these groups showed worsening outcomes for MUAs between 13 to 26 weeks versus greater than 26 weeks (21° vs 12°; $P<.01$), but no significant differences between 0 to 6 weeks versus 6 to 12 weeks following surgery (36° vs 38°; $P<.89$). In summary, despite the lack of high-quality RCTs comparing the outcomes of MUA with non-MUA intervention, the current orthopedic literature strongly supports MUA as an effective first-line intervention in the setting of unsatisfactory knee flexion and function, and should ideally be performed within 12 weeks of surgery (Fig. 1).[77,78,80] For patients with ongoing infection, component malalignment, or an elevated joint-line, MUA is contraindicated and the patient should instead be evaluated for revision TKA to address the primary underlying condition.[77]

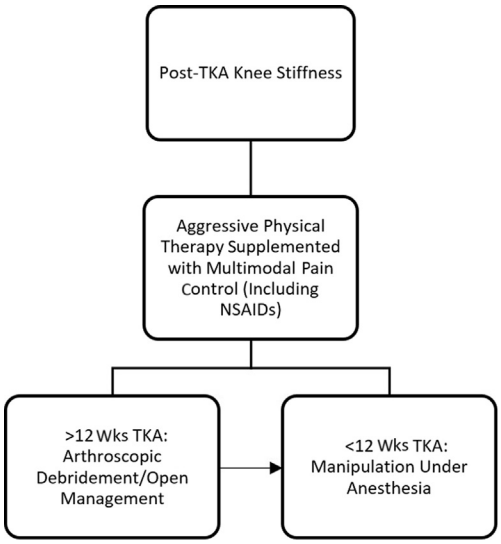

Fig. 1. Intervention protocol for TKA candidates. Patients with knee stiffness should be immediately started on an aggressive physical therapy regimen, supplemented with a multimodal pain control that includes NSAID therapy. Treatment failure should progress to more aggressive management. For patients within 12 weeks of TKA, manipulation under anesthesia should be pursued. Patients failing manipulation, or who are outside the 12-week window, should progress to arthroscopic debridement and/or possible open management.

Arthroscopic Lysis of Adhesions

The formation of adhesions in the arthrofibrotic knee occurs primarily between the capsule and femoral condyles, as well as in the anterior interval, infrapatellar fat pad, and pretibial recess.[85] Arthroscopic lysis of adhesions (LOA) is therefore a minimally invasive surgical approach that allows for direct visualization and treatment of the focal and diffuse pathologic fibrous scar tissue using motorized shaver instruments and radiofrequency ablation devices.[85]

Although there are few studies examining the outcomes of arthroscopic LOA, retrospective studies report good outcomes. A retrospective study by Schwarzkopf and colleagues[86] reported a significant gain of 23.75° of total ROM following arthroscopic LOA with MUA. Higher preoperative WOMAC (Western Ontario and McMaster Universities) scores, shorter patients, and body mass index greater than 30 kg/m^2 were also correlated with greater ROM gains. Several other studies have also shown similar gains ranging from 18.5° to 60° of total ROM.[78,87]

Although posterior capsular adhesions are thought to be the primary contributor of flexion contractures, and access to this region of the knee is arthroscopically limited, previous studies have been reassuring. In a retrospective study of arthroscopic LOA after failed MUA by Tjoumakaris and colleagues,[87] the average extension deficit was significantly decreased from 16° to 4° at final follow-up (minimum 12 months, average 31 months) despite no attempt at posterior release in of any patients in the cohort. Furthermore, in a separate retrospective study of 18 patients with posterior stabilized TKAs, arthroscopic LOA again showed significant reductions in extension deficits, from 9.17° to 3.06°.[88] In addition, unlike MUA alone, the timing of arthroscopy does not seem to affect outcomes, and successful results have been reported up to 1 year following TKA.[78]

Open Scar Excision and Revision Knee Arthroplasty

In a small number of patients, extensive periarticular and intraarticular fibrosis makes it very difficult to use arthroscopic treatments. These refractory cases necessitate an open scar excision with debridement and soft tissue release for better visualization and easier access to the fibrotic tissue.[89] A retrospective study by Millett and colleagues[89] reported improvements in 8 knees, with gains of flexion from 81° to 125° and reductions in extension loss from 18.8° to 1.25°. In addition, Lysholm II scores improved

by 35.5 points per patient and all patients were satisfied with their outcomes.[89]

Once all other treatment options are exhausted or if there is clear evidence of implant malposition, revision TKA should be considered. Outcomes following revision TKA for arthrofibrosis have been modest compared with those following revision TKA for other causes, such as instability or loosening.[90] Kim and colleagues reported improvements in mean Knee Society function scores (40–58 points), mean Knee Society pain scores (15–47 points), and mean knee ROM (55° to 82°) among 52 patients that underwent revision TKA for arthrofibrosis.

SUMMARY

Arthrofibrosis is a common complication following TKA, causing patients significant functional disability. The emergence of rapid rehabilitation protocols and perioperative NSAIDs has reduced the prevalence of arthrofibrosis following TKA. An understanding of the pathophysiologic underpinnings, associated risk factors, and management strategies can aid in the treatment of these patients.

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