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A Comprehensive Approach to Stiffness in Total Knee Arthroplasty

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ABSTRACT

This article is a summary of a symposium on knee stiffness in primary and revision total knee arthroplasties presented at the 2024 American Association of Hip and Knee Surgeons annual meeting. The purpose of this article was to provide an overview of the genetics of arthrofibrosis, preoperative and perioperative risk factors associated with arthrofibrosis, ways to optimize modifiable risk factors, and early and late treatment options for stiffness and when it fails to improve with initial interventions.

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Knee stiffness remains a common and challenging problem for patients and arthroplasty surgeons after primary and revision total knee arthroplasty (TKA) [1,2]. Causes of stiffness may be multifactorial, including host soft-tissue factors such as high thigh-calf index, implant coronal or rotational malalignment, patellofemoral maltracking, or arthrofibrosis from pathologic fibroproliferative processes [3–7]. The reported incidence of knee stiffness in the literature varies from 1.3 to 6.9% [8]. The incidence of manipulation under anesthesia (MUA) is as high as 9.9% in some series, and stiffness accounts for 3% of all revision TKAs in the American Joint Replacement Registry [9,10]. Despite this common problem, there remain several limitations in our current understanding of the disease state [7]. Furthermore, there is a lack of consensus on how best to identify patients at risk, best practices for perioperative and early postoperative prevention and management, and the best surgical treatment options when stiffness becomes persistent.

The Genetics of Arthrofibrosis

Arthrofibrosis, a debilitating complication of TKA, is characterized by excessive scar tissue formation and reduced knee range of motion (ROM). Despite advances in surgical techniques and postoperative management, arthrofibrosis remains a major challenge that negatively impacts patient outcomes and quality of life and increases the need for revision surgery. Emerging research highlights the role of genetics in predisposing individuals to this condition, offering potential avenues for identifying high-risk patients, patient-specific targeted prevention strategies, and nonoperative treatment options.

These genetic predispositions for arthrofibrosis are related to the genetic tendency to create excessive extracellular matrix (ECM) proteins in the knee, which results in less motion [7]. The pathophysiology of arthrofibrosis begins with an exaggerated response to surgical trauma or injury, which leads to a local cascade and fibroblast activation. Following this exaggerated tissue response, there is an imbalance between matrix synthesis and degradation, leading to reduced activity of matrix metalloproteinases (MMPs). Key genetic variations that play a role in arthrofibrosis include transforming growth factor-beta (TGF- β 1), MMPs, and interleukin

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(IL)-6 [11]. Transforming growth factor-beta 1 is a crucial regulator of fibrosis and is found to have increased expression and activity in some patients following TKA [12]. Another area of interest is the role of MMPs, enzymes responsible for ECM remodeling. Dysregulation of MMP activity, influenced by genetic variants, can contribute to the pathological accumulation of scar tissue.

Key among these are polymorphisms in genes encoding cytokines such as IL-6 and tumor necrosis factor-alpha (TNF- α), which are pivotal in the inflammatory response [13]. Elevated expression of these cytokines can lead to prolonged inflammation, promoting fibroblast proliferation and excessive ECM deposition. In addition, single-nucleotide polymorphisms in genes related to collagen synthesis and degradation may further influence susceptibility to arthrofibrosis.

Understanding the genetic predisposition to arthrofibrosis has important clinical implications. Dagneaux et al. [14] sought to identify the presence of similar gene variants in other organ systems that have idiopathic fibrotic disease processes. Despite shared genetic variations in both the hand and lung, there is still limited information on which specific genes cause fibrosis in other musculoskeletal joints. Moreover, targeted therapies aimed at modulating specific genetic pathways, such as TGF- β 1 inhibitors and cytokine blockers, hold promise for mitigating fibrotic responses in susceptible patients. Several experimental therapies have targeted these pathways, including TGF- β 1 inhibitors and specific cytokine blockers such as IL-6 or TNF- α inhibitors [15,16]. While early laboratory studies suggest they may curtail or prevent the fibrotic process, currently, there is no robust clinical data supporting their routine use in TKA patients [16]. Further research is needed to fully elucidate the complex genetic mechanisms involved, and future prospective studies are needed to determine if prophylactic or early postoperative administration of these biologics can reduce the incidence or severity of arthrofibrosis and guide clinical practice for total knee arthroplasty patients.

Early Identification of Risk Factors Associated With Stiffness and Perioperative Optimization

Knee ROM is essential for activities of daily living, and varying degrees of knee motion are necessary for certain activities. For example, around 65 degrees of motion is required for flat ground walking, while over 100 degrees is required for descending stairs. Loss of terminal extension is particularly limiting, as flexion contractures of $> 5^\circ$ are associated with gait changes and increased low back pain. For this reason, knee stiffness after TKA is a major cause of patient dissatisfaction. Although less common in the knee than the hip, heterotopic ossification can lead to major loss of motion after TKA [17,18]. Early recognition with advanced imaging and prophylactic measures, such as low-dose irradiation or nonsteroidal anti-inflammatory drugs (NSAIDs) in high-risk patients, can mitigate the detrimental effects of heterotopic ossification on postoperative motion [19]. The pathogenesis of arthrofibrosis is related to a cascade of events that begins with surgery-induced tissue damage that, in some patients, leads to dysregulated production of ECM, leading to excessive fibrosis and knee joint contracture. Several risk factors exist for arthrofibrosis, including joint-specific, patient, and surgical factors. Some knee-specific risk factors include posttraumatic osteoarthritis (OA), prior open knee surgery, patella baja, and poor preoperative ROM, which are associated with reduced ROM after TKA [20]. These factors may warrant more extensive preoperative counseling, as these conditions can predispose to more challenging exposures, scarring, and limited motion postoperatively. Surgeons often discuss with these patients the possibility of requiring additional releases or soft-tissue

procedures to manage the preexisting deformities or scars, emphasizing the need for diligent postoperative therapy.

Subacute or culture-negative infections can present with minimal systemic signs and knee stiffness or arthrofibrosis as the principal clinical sign [21]. A high index of suspicion for infection is warranted, and the workup should include a complete blood count, inflammatory markers, including erythrocyte sedimentation rate and C-reactive protein, and potentially synovial fluid aspiration and culture for early detection [21,22]. Extra-articular damage to the femur, quadriceps muscle, or extensor mechanism, whether from prior trauma or extensive scarring, is also a critical risk factor for the development of a stiff knee [23]. Dense scar tissue of the joint capsule or quad tendon can severely limit postoperative motion and complicate surgical management [23,24]. Metal artifact reduction sequence magnetic resonance imaging can be useful in identifying pathologic scar formation in arthrofibrosis. In addition, a computed tomography scan can be obtained if there is suspicion of component rotational malalignment. Surgeons should also remain vigilant for extra-articular causes, including severe hip or spine pathology.

Patient risk factors include a history of undergoing a MUA after a contralateral TKA, as this carries a 14 $\times$ greater risk for MUA after the second side TKA [25]. This highlights the systemic risk that some patients have for postoperative fibrosis. Pain-related risk factors are both behavioral, such as pain catastrophizing, systemic, as in fibromyalgia, and local, such as cutaneous hypersensitivity or complex regional pain syndrome. Operating on patients who have active local pain syndromes should be avoided, as this may lead to an exacerbation of pain symptoms and a poor outcome. Surgical risk factors for postoperative stiffness include overstuffing the anterior compartment or an excessively tight extension or flexion gap, a tight posterior cruciate ligament with anterior tibial tray lift-off, and internal rotation of the tibial component or femoral component.

How Can We Optimize Patient Risk Factors?

First, standard patient preop optimization of modifiable risk factors should be performed, including body mass index (BMI) reduction below 40, hemoglobin A1C $< 7.5\%$, hemoglobin (Hb) > 12 g/dL, and smoking cessation > 4 weeks before surgery [26]. Preoperative diagnoses of obesity (BMI > 40) and unmanaged diabetes (hemoglobin A_{1C} $> 7.5\%$) are associated with increased risk of infection, dislocation, wound dehiscence, implant failure, and revision, along with decreased postoperative ROM in total joint arthroplasties [26–30]. Medical optimization reduces the overall inflammatory burden associated with metabolic syndrome, which is a combination of obesity, hypertension, diabetes, and high cholesterol. Additional preoperative steps that may be taken to specifically reduce the risk of developing arthrofibrosis include education on the value of a low-carbohydrate diet combined with increased lean protein, fruit, and vegetable intake. Since surgery induces oxidative stress, and uncontrolled oxidative stress can lead to persistent inflammation, this may trigger a cascade of inflammation-related events ending in arthrofibrosis. The goal in the perioperative period is to provide the body with sufficient antioxidants to scavenge and neutralize free radicals and potentially reduce the oxidative stress of surgery [31,32]. Many patients who have knee OA already have uncontrolled oxidative stress that negatively impacts cartilage quality and, as such, are a high-risk group for increased oxidative stress postoperatively [33]. More research is needed, but emphasizing dietary vitamin C intake or oral supplementation (1,000 mg daily) in addition to a multivitamin and glycine/N-acetyl choline (approximately 1,000 mg of each daily) are low-cost, low-risk interventions that could improve

perioperative outcomes and reduce fibrosis risk; however, more research is needed to confirm efficacy.

Quadriceps muscle atrophy is commonly encountered in patients, particularly deconditioned individuals who have long-standing knee OA. The arthrogenic muscle inhibition score is a way to quantify quadriceps muscle inhibition. In a study of 272 patients undergoing primary TKA, Parratte et al. [34] found the incidence of preoperative quadriceps muscle inhibition to be present in 13% of patients undergoing TKA, and these patients were at an increased risk of developing a flexion contracture postoperatively. Clinicians and physical therapists intuitively recognize and emphasize the importance of the quadriceps muscle to drive the knee into terminal extension, and the results of this study highlight how quadriceps muscle inhibition needs to be addressed preoperatively to avoid this dreaded complication [34]. Quadriceps muscle atrophy and inhibition can be optimized with preoperative physical therapy, either supervised or with a home exercise program. Straight leg raises are a simple and effective way to engage the quadriceps muscle complex without causing knee pain. A stationary bike or seated pedaling device is also a crucial aspect of recovery, both pre- and post-operatively. Increasing patients' preoperative activity level increases muscle activation, which releases myokines that have positive systemic anti-inflammatory effects. A home-based stationary bicycle designed for early postoperative recovery after TKA demonstrated improved ROM and statistically fewer MUAs (1.5 versus 4.4%) compared to standard physical therapy [35]. The results of this study reinforce the important benefit low-impact pedaling have on limiting fibrosis postoperatively. The key is patient education and engagement throughout the surgical episode to avoid postsurgical fibrosis and flexion contracture development.

In regard to anti-inflammatories, increasing literature supports the use of NSAIDs or corticosteroids to reduce the risk of stiffness and MUA after TKA. In a recently published study, Salmons et al. [36] found that NSAID use reduced the risk of stiffness after TKA, and corticosteroids reduced the risk of MUA. However, they did not find a protective effect with some other medications thought to reduce the risk of fibrosis, including statins, angiotensin receptor blockers, and angiotensin-converting enzyme inhibitors. At the Hospital for Special Surgery, a similar analysis was performed that looked at the risk of MUA in patients who did and did not take NSAIDs postoperatively [37]. When matched for type of deep vein thrombosis prophylaxis, age, sex, and BMI, patients who were unable to take NSAIDs had a significantly increased risk for MUA within the first 3 months after TKA (5.3 versus 12.6%, $P < 0.001$) [37]. These two studies taken together reinforce the impact anti-inflammatory medications have on reducing postsurgical inflammation that may lead to early fibrosis and the need for MUA after primary TKA.

The importance of some type of anti-inflammatory coverage in the postoperative period has changed our perioperative protocols in this higher-risk cohort. For a patient who is unable to take NSAIDs postoperatively, we will prescribe a tapered corticosteroid regimen (Medrol Dosepak, Pfizer, New York) for the first week postoperatively. Due to limited high-quality evidence, the dosage and duration of corticosteroids necessary to reduce the risk of MUA in this higher-risk cohort are unknown. However, sufficient evidence does exist that the use of corticosteroids in the perioperative period is safe, as Palmer et al. [38] recently published a review on the use of corticosteroids after hip and knee arthroplasty, showing efficacy in improving early outcomes without associated complications.

There are several prescription medications that have been studied for the prevention of postsurgical fibrosis (Table 1). However, there is currently limited evidence on efficacy, as most of

Table 1

Prescription Medications That Have Been Studied for the Prevention of Postoperative Stiffness After Total Knee Arthroplasty.

Corticosteroids
Nonsteroidal anti-inflammatory drugs
Ketotifen (anti-histamine)
Colchicine
Metformin
Atorvastatin
Angiotensin-converting enzyme inhibitors (losartan)
Angiotensin II receptor blockers
Leukotriene receptor antagonist
Colchicine
Glucagon-like peptide-1 receptor agonists

these medications have been studied in flexion contracture animal models, and the few human trials that have been published include heterogeneous definitions and treatment protocols. For that reason, the authors currently prioritize the use of corticosteroids and NSAIDs over other prescription medications to prevent postsurgical fibrosis until further studies are available [39].

Another recent publication that won the clinical award for best paper at the AAHKS 2023 annual meeting focused on the use of tranexamic acid (TXA) to improve early outcomes after TKA. Tranexamic acid has become the standard of care to limit surgical bleeding and has led to a dramatic reduction in transfusion rates. However, TXA also has an anti-inflammatory component related to a reduction in the release of fibrin split products. In this double blind randomized controlled trial of 40 patients undergoing TKA, patients who received 2 grams of oral TXA on the day of surgery and postoperative days one, two, and three had 10 degrees better ROM at 6 weeks, in addition to less pain and higher Knee Injury Osteoarthritis Outcome Score Joint Replacement, but no difference at 3 months in regard to mean ROM [40]. This study was not powered to assess for differences in MUA rates, but the data support the use of extended oral TXA to optimize early postoperative outcomes, including ROM [40].

Regarding pharmacologic adjuncts for pain control and potentially limiting pain-associated arthrofibrosis, gabapentin has been used to reduce neuropathic pain and opioid consumption in some TKA protocols [41]. While there is no direct evidence that gabapentin alone prevents arthrofibrosis, improved pain control may enhance patient participation in therapy, thereby indirectly reducing the risk of stiffness [41].

In summary, stiffness after TKA is often multifactorial. Preoperative interventions that may limit postoperative fibrosis include medical optimization and nutrition counseling and implementation of a targeted prehabilitation program that minimizes quadriceps muscle inhibition and encourages stationary bicycle use. Surgical principles should be followed that ensure a well-balanced, rotated, and sized knee that achieves full extension intraoperatively. Perioperatively, NSAIDs and steroids reduce the risk of early fibrosis, and additional research is needed to identify the need for additional pharmacologic adjuncts. There is some early data to support the use of extended oral TXA, and consider the use of antihistamines and antioxidants to control inflammation and the oxidative stress from surgery, but further prospective trials are necessary to better delineate the efficacy of each of these modalities on improving knee ROM.

Early Postoperative Management: Within 90 Days

Early intervention within the first 90 days postoperatively is crucial to prevent long-term stiffness and inferior patient outcomes. However, there is little consensus on best practices for

postoperative rehabilitation and early postoperative management, aside from MUA.

Physical Therapy and the Quiet Knee Protocol

Postoperative physical therapy protocols vary widely, ranging from solely self-directed exercises at home without formal therapy to regular, aggressive formal physical therapy regimens [42]. However, there is a paucity of data in the literature on best practices.

We have evolved our postoperative rehabilitation protocol after several decades of performing TKAs as well as treating many patients who have developed postoperative arthrofibrosis. All patients should undergo primary and revision TKA visits with a physical therapist. At this visit, setting proper expectations and education is the focus. Patients are taught self-directed exercises for terminal extension and flexion, educated on how to using walking aids and other adaptive equipment, and given a host of physical and online resources to assist with their postoperative rehabilitation. Physical therapy starts immediately when patients' neuraxial anesthesia fades, with a therapist mobilizing patients and again educating them on exercises to perform at home.

Our focus is on gradual, gentle ROM protocols with a focus on self-directed exercises but also formal physical therapy one to two times per week for 2 to 3 months postoperatively. We strongly discourage overly aggressive therapy involving aggressive early passive knee ROM; this can perpetuate a cycle of knee trauma, with the resulting inflammation causing frequent setbacks to knee ROM. As such, we have developed a “quiet knee” protocol for primary TKA that many surgeons at our institution follow (Table 2). The goal is to minimize knee trauma and thereby reduce knee swelling, pain, and inflammation and ultimately facilitate a better patient experience and better ultimate knee ROM. Patients should be reminded that terminal extension exercises are crucial, as adequate knee extension, even more than knee flexion, facilitates a normal gait pattern [43,44].

We also encourage aggressive cryotherapy postoperatively, recommending patients ice/elevate and work on terminal extension exercises for at least 20 to 30 minutes every 2 to 3 hours, if not more, for the first several weeks. There is some limited data that a compressive cryotherapy machine may be more beneficial than ice packs alone in diminishing swelling and pain and facilitating early knee motion [45].

Early non-MUA interventions include the following: steroid taper regimen, terminal extension bracing, and frequent patient monitoring to detect early difficulties with knee extension and/or flexion are important to facilitate early intervention. In the modern era, there is a plethora of enabling technology, including smart-watch applications, smart braces, text messaging applications, and even smart implants that can assist with patient monitoring. Simple phone calls to patients or in-office visits at 2 and 4 weeks to ensure patients are on track can also be effective.

For patients struggling with early flexion at three to 6 weeks, many surgeons use a steroid taper regimen to reduce inflammation

and hopefully prevent dense scar tissue formation [36]. While several regimens exist, one common regimen is prednisone 40 mg $\times$ 5 days, 20 mg $\times$ 5 days, and 10 mg $\times$ 5 days. Anecdotally, many surgeons have had successful results in patients, preventing the need for an MUA, but a prospective study is pending. Ensuring patients are also on gastroprotection is recommended. Contraindications to these protocols include brittle diabetics or patients who have tenuous wounds.

For patients developing more than a 10° flexion contracture beyond 2 to 3 weeks, initiating a dynamic extension orthosis can be effective in assisting patients in achieving better terminal extension [46–48]. We recommend that patients wear it at night and for 1 to 2 hours, 3 to 4 times per day if tolerated. Early intervention of a flexion contracture is crucial, as MUA is much less effective for regaining terminal extension than flexion [49,50].

Manipulation Under Anesthesia: When, How, and Outcomes

If early interventions to prevent stiffness are ineffective, MUA remains the gold standard to improve knee flexion within the first 3 months postoperatively [51]. The incidence of MUA after primary TKA varies from 0.5 to 10% in the literature [9,50]. Patients who cannot achieve 90° of knee flexion by 6 to 8 weeks should be considered for an MUA, depending on their preoperative knee ROM. As previously discussed, preoperative knee flexion is the best predictor of postoperative ROM. As such, a patient who has preoperative 80° of flexion should be treated with different expectations than a patient who has preoperative 120° of flexion. A number of studies have shown improved results if MUA is performed within 3 months compared to a later MUA [50–52]. Akhtar et al. [51] reported in a systematic review and meta-analysis that patients undergoing MUA < 3 months from surgery had better improvement in knee flexion than those undergoing MUA > 3 months from surgery (30 versus 19°) and had a lower incidence of subsequent revision TKA.

The technique of an MUA is familiar to the majority of knee surgeons. We recommend slow and gentle pressure with one hand supporting the distal femur and the other near the proximal tibia, maintaining a short lever arm to minimize the risk of periprosthetic fracture. As scar tissue yields, the surgeon should listen for the palpable and audible release of adhesions. We perform MUAs in our post-anesthesia care unit under intravenous sedation. Patients receive perioperative dexamethasone 8 mg and a post-MUA corticosteroid taper regimen outlined above, with the same physical therapy philosophy outlined above. We strongly encourage surgeons to take pre- and post-MUA photographs for patient encouragement and documentation purposes. We see patients in follow-up with new radiographs at 2 to 3 weeks post-MUA.

A number of studies have shown MUA to be effective in improving patient knee ROM. Abdel et al. [53] reported an average immediate flexion gain of 46°, an average 6-week flexion gain of 28°, and an average 1-year flexion gain of 37°. However, several studies have shown poorer long-term outcomes in patients undergoing a post-TKA MUA compared to those who did not undergo an MUA. In a review of almost 300,000 primary TKAs in the National Joint Registry, patients who underwent an MUA were at a five-fold risk of subsequent revision TKA compared to patients who did not undergo MUA [54]. Similarly, Crawford et al. [55] reported that patients who underwent an MUA versus those who did not have higher rates of revision TKA for stiffness (9.9 versus 3.0%), lower final follow-up knee ROM, and lower final follow-up patient-reported outcome measures.

Also, in patients who return with continued stiffness and minimal improvement, a repeat MUA can be effective. In one institution's report, between 15 and 31% of patients after primary MUA underwent a second MUA [56]. In another study of patients

Table 2
Postoperative “Quiet Knee” Protocol Aimed to Minimize Knee Swelling and Early Knee Trauma.

For 7 to 10 day after primary total knee arthroplasty, patients should:
1. Self-directed GENTLE active range of motion (ROM) exercises
a. Avoid aggressive passive ROM exercises
2. Focus on terminal extension exercises
3. Ambulate <1,000 feet per day
4. Limit standing to <20 minute at a time
5. Ice and elevate for 20 to 30 minute every 2 to 3 hour

undergoing a second MUA after primary TKA, Issa et al. [57] reported that only 60% of patients achieved acceptable ROM, while the other 40% went on to additional procedures for stiffness.

Late Postoperative Management: Revision Total Knee Arthroplasty

There is a plethora of surgical etiologies that can contribute to chronic stiffness after TKA, including component malalignment, patellar overstuffing, joint line elevation, unresected posterior osteophytes, and increased condylar offset [58,59]. A thorough preoperative workup and surgical correction of these technical etiologies can alleviate stiffness for some patients.

However, for patients who have true arthrofibrosis and severe stiffness (arc of motion $<60^\circ$), full revision TKA, typically to a rotating hinge (RH) construct with a systematic surgical and perioperative protocol (Table 3), has yielded the most promising results [60,61].

A wide and safe surgical exposure is critical to improving ROM and preventing further complications, especially with the extensor mechanism. Start with a full and complete medial release. Next, perform an aggressive, wide synovectomy of the medial and lateral gutters and suprapatellar pouch. If additional exposure is necessary, there should be a low threshold to perform a quadriceps muscle snip; this provides a safe and effective exposure without increasing complication rates or changing postoperative rehab protocols [40]. If planning a hinged device, it is also beneficial to completely peel the collateral ligaments from the femur, as this not only facilitates safer exposure but can improve ultimate knee motion by releasing contracted ligaments and opening up the flexion space to give patients a mechanical advantage to improve their ROM [62,63]. The polyethylene component can then be removed to detension the knee and relax the extensor mechanism.

The femoral and tibial components should then be removed in a safe and efficient manner as has been previously described, with the goal of preserving bone to facilitate the reconstruction [64,65]. On the tibial side, any heterotopic bone should be removed and the implant prepared, correcting any malrotation and/or malalignment. On the femoral side, the posterior capsule should be stripped, and any remaining posterior osteophytes should be removed. Surgeons should aim to correct the joint line, femoral rotation, and condylar offset when preparing the femoral component. In these severe revision scenarios for arthrofibrosis, surgeons have also reported deliberately undersizing the femoral component to help

ensure a more generous flexion space so that patients can more reliably achieve higher degrees of knee flexion postoperatively [66]. In a patient who has stiffness in flexion and patella baja, lowering the joint line can improve patellar position and open the flexion gap with the hinged prosthesis. This space in flexion can facilitate a mechanical advantage for the patella and greater ROM to achieve more flexion in this patient population [67,68]. Before closure, the arthrotomy can be pie-crusted gently in the coronal plane to facilitate more motion and relax the extensor mechanism. We recommend aggressive perioperative and postoperative corticosteroid and anti-inflammatory doses as well as close patient follow-up, with MUA at 4 and 8 weeks as necessary.

A number of studies have shown encouraging results of revision TKA to a hinged device in patients who have severe arthrofibrosis. Bingham et al. [69] reported an increased arc of motion of 20° on average with revision to a hinged device compared to just 12° with a Varus-Valgus Constrained (VVC) implant ($P < 0.01$). Liow et al. [61] similarly reported significantly improved ROM gains with a hinged device of an average of 41° compared to just 18° in a VVC implant ($P < 0.01$) in a mixed group of patients who have severe and nonsevere arthrofibrosis. Also, Schneider et al. [19] reported an increase in knee flexion of 60° in a cohort of 60 patients revised to a hinged device who were also treated with postoperative low-dose radiation.

Conclusions

Stiffness after TKA remains a multifactorial challenge, requiring a comprehensive, patient-centered approach for prevention and management. Genetic predispositions, including variations in TGF- β 1, MMPs, IL-6, and TNF- α , can heighten the risk of arthrofibrosis and underscore the need for continued research to better identify and target high-risk patients [7,11–13]. By integrating perioperative optimizations such as patient education, nutritional enhancement through diet or supplements, and prehabilitation muscle strengthening, surgeons can mitigate the inflammatory cascade that leads to fibrosis [33–35].

Building on this foundation, multimodal anti-inflammatory strategies, including NSAIDs, corticosteroids, and possibly extended oral TXA, show promise in reducing the incidence and severity of postoperative stiffness [36,40]. Nevertheless, early detection and intervention remain paramount: the “quiet knee” rehabilitation protocol stresses gentle, progressive ROM exercises while minimizing mechanical trauma to foster controlled rather than aggressive therapy [42,43].

When early interventions fail, despite dynamic bracing, oral corticosteroid tapers, and close clinical monitoring, MUA remains a key tool, particularly when undertaken within 3 months postoperatively [51,52,57]. Though MUA can successfully increase knee flexion, patients requiring MUA are at a higher risk of later revision TKA and may exhibit less favorable long-term outcomes compared to those who do not need an MUA [54,55].

Ultimately, in patients who have persistent, severe stiffness (arc of motion $<60^\circ$) or definitive component malpositioning, revision TKA with VVC or an RH construct may provide an effective solution [58,61]. Meticulous surgical exposure, complete synovectomy, component removal, and attention to restoring soft-tissue balance are critical. Modern implants and metaphyseal fixation have demonstrated encouraging survivorship and durable functional gains, particularly with RH designs in complex revision scenarios [69–71].

Moving forward, further research must elucidate the genetic underpinnings of arthrofibrosis and refine risk stratification algorithms, ensuring that targeted prevention and tailored postoperative regimens benefit those most susceptible. Through a

Table 3

Revision Total Knee Arthroplasty Arthrofibrosis Protocol of the Senior Author (MPA).

1. Revision to rotating hinge total knee arthroplasty with wide debridement, lysis of adhesions, and removal of all heterotopic ossifications
2. Coronal pie crusting of quadriceps tendon before arthrotomy closed
3. 8 mg IV dexamethasone before incision assuming no contraindication
4. Intraoperative periarticular injection with corticosteroid assuming no contraindication
5. Hard Robert Jones dressing for 48 hour
6. 8 mg IV dexamethasone on morning of POD#1 assuming no contraindication
7. 15 mg of IV ketorolac every 6 hour $\times$ 4 starting in recovery assuming no contraindication
8. Aspirin 81 mg PO BID for 6 weeks if no contraindication
9. Celecoxib 200 mg PO BID for 4 month to start after ketorolac doses are completed assuming no contraindication
10. Protonix 40 mg PO QD for 4 month
11. GENTLE range of motion with outpatient physical therapy starting 1 week postoperatively
12. Follow-up with surgeon at 2, 4, 6, 8, 10, and 12 weeks
13. Up to two manipulations under anesthesia at 4 weeks and 8 weeks postoperatively

IV, intravenous.

combination of early risk factor modification, thoughtful rehabilitation, judicious use of anti-inflammatory therapies, and, when necessary, MUA's or surgical revision, surgeons can continue improving outcomes for this difficult yet increasingly prevalent problem in contemporary TKAs.

CRedit authorship contribution statement

Brian P. Chalmers: Writing – review & editing, Writing – original draft. **Linda I. Suleiman:** Writing – review & editing, Writing – original draft. **Peter K. Sculco:** Writing – review & editing, Writing – original draft. **Matthew P. Abdel:** Writing – review & editing, Writing – original draft.

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