Chapter

Stiffness after Primary Total Knee Arthroplasty

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Abstract

Total knee arthroplasty remains the definitive treatment for end-stage osteoarthritis of the knee. Despite being a very successful intervention in terms of relieving pain and returning a patient's function, it is not without complications. Post-operative stiffness after total knee arthroplasty is one of those complications that can be puzzling for physicians and debilitating for patients. While the etiology of stiffness is multifactorial, the treatment options are essentially limited to manipulation under anesthesia, removal of adhesions and revision total knee arthroplasty. With patient outcomes directly related to relief of pain and post-operative range of motion, it is paramount that surgeons do all that is necessary to minimize risk of post-operative stiffness.

Keywords: total knee arthroplasty, stiffness, manipulation under anesthesia, revision total knee arthroplasty

1. Introduction

Total knee arthroplasty (TKA) remains the mainstay of treatment in terms of pain relief, restoring mobility, and quality of life improvement for patients with end-stage osteoarthritis of the knee. Pain relief and postoperative range of motion (ROM) have consistently been the two variables of most importance to patients [1–3]. Stiffness after TKA can be debilitating due to pain and functional limitations in daily activities such as going up or down the stairs and sitting or arising from a chair. This chapter discusses the prevalence, etiology, and management of stiffness after primary TKA.

2. Prevalence

The reported incidence of stiffness after TKA varies greatly in literature with rates ranging from 1.3 to 12% [4, 5]. This wide range of incidence results largely due to lack of a consistent widely accepted definition of stiffness after TKA. Laubenthal et al. reported in their quantitative analysis of knee range of motion during activities of daily living (ADL) that patients required a mean of 83 degrees of knee flexion to climb stairs, 93 degrees to sit in a chair without using their hands and 106 degrees for tying their shoes while seated [6]. In fact, many authors use a cut-off of around 95 degrees of flexion to define stiffness as that allows patients to do most of their ADLs [7]. What is less clear is perhaps, at what time point in the postoperative period must a patient obtain 95° [7–9]. Based on the international consensus definition for stiffness

according to restriction in ROM, the severity may be graded according to loss of movement based on the deviation from full flexion or extension as mild, moderate, and severe extension restriction (5–10, 11–20, >20) or flexion range (90–100, 70–89, <70) [10]. However, no consensus statement was made on time frame.

3. Etiology

The etiology of stiffness is multifactorial and the associated risk factors can be evaluated by dividing them into three categories: preoperative, intraoperative, and postoperative.

3.1 Preoperative risk factors

There are several preoperative risk factors that may contribute to stiffness after TKA and can be further subcategorized into modifiable versus nonmodifiable.

3.1.1 Modifiable

The major modifiable risk factor is preoperative ROM. Preoperative ROM has consistently been shown to be one of the best predictors of postoperative ROM. Patients with decreased preoperative ROM often have decreased postoperative ROM as well as lower functional scores compared to those without decreased preoperative ROM [11, 12]. With respect to flexion, studies have shown that patients with poor preoperative flexion (<90 degrees) tend to gain flexion postoperatively and those with good preoperative flexion (>105 degrees) experience a net loss in flexion, yet retain a greater ROM overall [13, 14].

3.1.2 Nonmodifiable

Certain patients, that senior author calls "scar-formers", may be at increased risk of stiffness due to their genetic makeup. Several studies have implicated the role of genetics in the formation of arthrofibrosis [15, 16]. It is unclear how to identify these Scar-formers as literature is lacking on whether or not patients with previous keloids or hypertrophic scars go onto to develop stiffness after TKA. At the very least, these findings may serve as a reminder to the treating physician to pay particular attention to these patients as they progress through their postoperative rehabilitation.

While a history of previous surgery and/or trauma has certainly been shown to adversely influence outcomes after TKA, whether or not previous surgery predicts postoperative ROM or stiffness is less clear [17]. In a study by Scranton et al., 85% of the patients with a stiff knee after TKA had previous surgery or diabetes mellitus [9]. Another study evaluating the results of total knee arthroplasty after failed proximal tibial osteotomy for osteoarthritis, reported average arc of motion to be 8 degrees less in TKA patients with prior history of failed proximal tibial osteotomy than those without it. Despite the small difference in arc of motion, the final average arc of motion was 95 degrees in the osteotomy group and there were no differences in rate of people undergoing manipulation for stiffness when compared to those without the osteotomy [18]. Similarly, Harvey et al. showed previous proximal tibial osteotomy had no effect on ultimate ROM [13]. Patients that underwent TKA after a failed unicompartmental knee arthroplasty have demonstrated mean postoperative arcs of motion between 104 and 115 degrees [19]. When comparing patients undergoing TKA for primary osteoarthritis versus post-traumatic osteoarthritis, literature demonstrates that overall there is significant improvement in

postoperative ROM when compared to preoperative ROM in both cohorts [20, 21]. However, the improvements were significantly inferior in the post-traumatic cohort [20]. Ultimately, we suspect that the specific type of prior surgery and/or trauma may play a significant role in determining its effect on postoperative ROM.

Lastly, obesity itself has not been shown to be a significant risk factor for postoperative stiffness, but patients with large thigh diameters may have reduced range of motion with flexion due to a mechanical block from abutment of soft tissues [22].

3.2 Intraoperative risk factors

Total knee arthroplasty should be thought of as a patient-specific procedure. Each patient's anatomy and deformity presents a unique challenge and no two consecutive knee arthroplasties are the same and therefore, attention to small details is crucial for a successful result. Most of the intraoperative variables that contribute to postoperative stiffness are related to the surgical technique. Improper gap balancing, incorrect component sizing or positioning, excessively elevating or lowering the joint line, incomplete resection of osteophytes, and closure techniques can all contribute to stiffness in both flexion and extension.

Improper gap balancing can lead to a joint that is "overstuffed" in flexion and/or extension, resulting in a stiff joint. Gap balancing can be easily understood by applying McPherson rules: if the tightness is symmetric in both flexion and extension, problem lies in the proximal tibia resection and if the tightness is asymmetric (i.e. tight in flexion but not in extension and vice versa), problem lies in the femoral resection. Excessive tightness in extension is caused by inadequate distal femur resection, tight posterior capsule, and inadequate resection of osteophytes. If tightness is present in both flexion and extension, it is generally due to a polyethylene insert that is too thick or insufficient proximal tibial resection. Excessive tightness in flexion is often caused by inadequate posterior femoral cut, decreased tibial slope, an oversized femoral component, and a femoral component that is shift posteriorly or malrotated. If using a cruciate retaining implant, a tight posterior cruciate ligament can also limit flexion. Furthermore, intimate knowledge of the instrumentation used during TKA is crucial as when an anterior referencing guide is used, the selection of a larger femoral component leads to tightness in flexion if sizing guide measurement is in between sizes.

While gap balancing in the sagittal plane is important, the patellofemoral joint (PFJ) deserves equal attention as overstuffing the PFJ can lead to tightness of the extensor mechanism and stiffness after TKA. PFJ is usually overstuffed due to two reasons: (i) inadequate resection of the patella or (ii) anterior placement of the femoral component. Generally speaking, the amount of patellar bone resected should equal the width of the patellar component while also keeping in mind the thickness of the cartilage that may not be present at the time of TKA. In a study by Alcerro et al., patients in whom the patellar thickness after TKA was restored as close to the native thickness demonstrated the greatest improvements in quality of life, physical measures and Western Ontario and McMaster Universities Arthritis Index stiffness scores [23].

Additionally, their study also showed that patients who reported more stiffness and lower knee active flexion had greater than native patella thickness after surgery [23]. Studies by Daluga et al. and Shoji et al. further show that an increase of 12% in anterior–posterior diameter of the knee and increase of 20% in patellar thickness, respectively, leads to marked increase in postoperative stiffness [8, 22]. In a similar fashion, joint line elevation can lead to issues with PFJ kinematics and cause stiffness after TKA. Elevated joint line, whether due to inadequate resection of tibia, excessive resection of distal femur, or thick polyethylene inserts, leads to patella baja. Patella baja has been associated with decreased postoperative ROM and patient reported outcome measures [24, 25]. The importance of maintaining correct patellar

height is further demonstrated by Vives-Barquiel et al. by showing improvements in flexion and clinical scores after osteotomy of the tibial tuberosity to move it proximally in knees with postoperative patella baja [26]. Of course, there is potential for catastrophic complication with this procedure, including nonunion and escape.

Lastly, several studies have demonstrated that knee position (i.e. flexion, semi-flexed, versus extended) during surgical wound closure may influence postoperative ROM. In a literature review by Faour et al., authors concluded that wound closure in flexion was associated with significant improvement in ROM recovery at earlier follow-ups after TKA and faster physical recovery compared with wound closure in extension. However, no difference was noticed in long-term ROM recovery when comparing closure with knee in flexion versus extension [27]. On the contrary, studies by Motififard et al. and Masri et al. demonstrated no differences in postoperative ROM with knees closed in flexion versus extension [28, 29].

3.3 Postoperative risk factors

Postoperative risk factors that can contribute to stiffness include lack of patient participation/compliance with therapy, uncontrolled pain, complex regional pain syndrome (CRPS), heterotrophic ossification (HO), infection, patellar complications, and arthrofibrosis. Postoperative physical therapy is an integral component of recovery after TKA, as patients often have issues with gait, balance, strength, and ROM. It requires significant commitment on the part of the both the physician and the patient to come up with an individualized plan meet postoperative rehabilitation goals. Poorly motivated patients are less likely to mobilize after surgery, comply with postoperative ROM and more likely to have a prolonged hospital stay. It is pertinent to identify these patients early (often even before surgery) and intervene early.

Uncontrolled pain or CRPS can also prohibit patients from exerting their maximum effort at therapy and must be correctly identified early and correct interventions including a possible referral to pain management be instituted. The incidence of postoperative infection after TKA is around 2% and should be considered in any patient with postoperative stiffness. If suspicion for infection is high appropriate labs including ESR, CRP and possible aspiration must be obtained to rule out an infection. Patellar complications such as unresurfaced patella, avascular necrosis of patella, patellar fracture, or mal-tracking can also cause also cause pain and stiffness. Lastly, while the incidence of radiographic HO after TKA may be as high as 26%, it is rare to find HO significant enough to limit ROM.

4. Management

The most important aspect of management for a stiff TKA is identifying the underlying etiology.

There are several treatment options available for stiffness after TKA including observation with more aggressive physical therapy, manipulation under anesthesia (MUA), surgical debridement, and revision total knee arthroplasty (rTKA). All of these strategies are only successful if done for the right indications. For example, a patient with stiff knee due to component misalignment or underlying infection is not likely to respond to a MUA.

4.1 Manipulation under anesthesia (MUA)

MUA should be considered when stiffness persists despite an aggressive program to gain motion.

Since MUA is not a benign procedure given the risk of fracture, extensor mechanism disruption, and hemarthrosis, correct patient selection is crucial. Anterior femoral notching is considered an absolute contraindication to MUA due to increased risk of femoral fractures. When it comes to MUA, one is faced with two questions: (i) which patients to manipulate? And (ii) what is the best time for manipulation? The answer to the first question lies in how one defines post-operative stiffness. Some physicians may stick to a strict number (i.e. flexion <90 by 6 weeks) and offer to manipulate everyone who fails to meet those criteria. However, the issue with a strict-number definition of TKA is that a patient who may not be considered to have stiffness (based on the aforementioned criteria, for example) might in fact be the one who needs the MUA as his/her activity requirements may consist of kneeling and hence greater need for flexion, as is often the case in Middle Eastern cultures. Therefore, the decision to proceed with a TKA should be centered on a joint conversation between the patient and the physician. We have patients in our practice who are very content with a ROM of 0–85 degrees, as they are able to do all the activities that they desire to do and therefore, do not need a MUA.

With respect to the timing for MUA, there is no consensus in the literature. Studies demonstrate both increased and no additional benefit with early MUA. In their review of patients undergoing MUA for stiffness, Issa et al. report that patients who underwent early MUA (<12 weeks postoperatively) had significantly higher mean gain in flexion (36.5 versus 17), higher final range of motion (119 versus 95 degrees) and higher function scores (88 versus 83) than those who had late (>12 weeks postoperatively) MUA [30]. Furthermore, when they sub-stratified outcomes based incremental time to MUA demonstrated that there was significant drop in range of motion gained after MUA as more time elapsed postoperatively. While some range of motion was gained with MUA at all periods postoperatively, authors reported that best results were obtained when MUA was done within 12 weeks postoperatively and significantly worse at 26 weeks (36.5 versus 12 degrees). Other authors who found higher gains in flexion with early MUA reported similar results [7, 22, 31–33]. Yercan et al. reported a study of 46 patients that underwent MUA for stiffness after TKA had mean flexion arc improvement from 67+/−11 to 114+/−16 degrees. Furthermore, patients that underwent a MUA within the first 3 weeks after TKA had significantly higher final range of motion compared with those who underwent after 3 months (121+/−11 versus 112+/−16). Similarly, Namba et al. reported that although both early and late MUA result in significant gains in flexion arc, early manipulation resulted in approximately twice the mean flexion gains [31].

In contrast to the above studies, there are several studies that have shown no difference in outcomes when stratified based on timing of MUA. Yeoh et al. report on 48 patients that underwent MUA for stiffness and they noticed that at 1 year there was no difference in gain in ROM between knees that were manipulated within 12 weeks postoperatively versus after 12 weeks [34]. Similarly, Keating et al. report their results of 113 MUAs in 90 patients followed for a mean of 4.6 years and noticed that mean knee flexion improved from 70 to 105 degrees, however, no significant difference was found for patients that underwent MUA before or after 12 weeks after TKA ($p = 0.36$) [35].

4.2 Surgical treatment

Surgical treatment of stiffness in the forms of arthroscopic or open lysis of adhesions with or without MUA after TKA should be considered as the last resort after a patient has failed both physical therapy and MUA (or is outside the time window where MUA alone might not be beneficial). While arthroscopic debridement of adhesions with MUA has shown promising results in patients

with stiffness from procedures other than TKA, this is not always the case for patients who have it done after TKA [36–39]. Campbell reports an increase of only 11 degrees in flexion and 55 degrees in extension for 8 patients in 1 year after arthroscopy. Similarly, Bocell et al. report that only 2 out of 7 patients maintained pain-free improvements in ROM after arthroscopic debridement and MUA after TKA. On the contrary, other authors have reported marked improvements in ROM after arthroscopic lysis. Tjoumakaris et al. report in their study that after arthroscopic lysis with gentle manipulation for stiffness after TKA, mean flexion improved from 79 to 103 degrees and mean extension deficit from 16 to 4 degrees at average of 31 months, leading authors to conclude that arthroscopic lysis of adhesions is a reliable procedure [40]. However, they also noticed that patients achieved approximately half of the improvement that was obtained at the time of surgery. Volchenko et al. report on a matched cohort study of 35 patients treated with MUA and 35 patients treated with arthroscopic lysis of adhesions plus MUA. Arthroscopic lysis with MUA yielded changes in ROM: a 72.7% increase 4 to 12 weeks after index TKA ($p = 0.032$), a 50.0% increase 12+ weeks after TKA ($p = 0.032$), and a 99.8% increase in patients with a pre-manipulation ROM of 0–60 degrees ($p = 0.001$). MUA alone yielded a 49.2% increase 4 to 12 weeks after index TKA ($p = 0.161$), a 27.0% increase 12+ weeks after TKA ($p = 0.161$) and a 68.8% increase in patients with pre-manipulation ROM of 0 to 60 degrees [41]. Authors concluded arthroscopic lysis of adhesions plus MUA led to greater increases in ROM ($p = 0.026$) and final knee flexion ($p = 0.028$) compare with those treated with MUA alone. After arthroscopic lysis of adhesions and manipulation, Diduch et al., Scranton, and Bae et al. also report similar results with mean flexion improvement of 26 degrees, mean gain in ROM of 31 degrees, and mean improvement in arc of motion of 42 degrees, respectively [9, 42, 43]. There is evidence in literature that for patients with a PCL-retaining implant and limitations in ROM (especially flexion), there may be a benefit from arthroscopic release of PCL. Williams et al. report a mean flexion increase of 30 degrees and mean extension improvement from 4 to 1.5 degrees at 20 month follow up 10 knees after arthroscopic release of PCL.

Lastly, revision TKA should be reserved for patients when a clear diagnosis for the cause of stiffness (i.e. malpositioning of components, infection, loosening, etc.) can be made and corrected during surgery as these patients have more predictable results compared to revisions done in patients without a clear-cut diagnosis [44–47]. Hartman et al. report on 35 patients that underwent rTKA for stiffness and at mean of 54.5 months, the mean arc of motion improved by 44.5 degrees. However, 49% (17/35) of the patients required a further intervention for stiffness or sustained a complication. Authors concluded that while rTKA can be performed with reasonable expectation of improvement in ROM, the complication risk is significant [48]. Ries et al. reported better results with rTKA in 6 knees with mean increase in arc of motion of 50 degrees at minimum of 2 year follow up for patients with stiffness secondary to arthrofibrosis only [49]. Generally, results of rTKA specifically for stiffness are less predictable and may be influenced by surgical technique and patient's response to surgical trauma.

5. Conclusion

TKA is an excellent option for patients with end-stage knee osteoarthritis in terms of pain relief. Postoperative stiffness continues to be a challenge for both the physicians and the patients. Due to the multifactorial etiology of stiffness, the interventions to address it are limited to MUA, lysis of adhesions, and revision TKA. The results with each intervention are variable, especially with surgical options.

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