International Journal of Orthopaedics Sciences

E-ISSN: 2395-1958
P-ISSN: 2706-6630
IJOS 2023; 9(1): 361-365

https://www.orthopaper.com
Received: 01-11-2022
Accepted: 05-12-2022

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Total knee replacement in knee arthritis with fixed flexion deformity

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DOI: https://doi.org/10.22271/ortho.2023.v9.i1f.3317

Abstract
Fixed flexion deformity of knee secondary to osteoarthritis occurs mainly due to posterior soft tissue tethering and/or contracture especially posterior capsule rather than bony changes. A flexion deformity of the knee leads to functional, biomechanical and gait impairments that interferes with the normal daily activities of patients and decrease their satisfaction after TKR. Correction of FFD is mandatory during TKR replacement to optimize postoperative results. The intraoperative correction steps differ among knee surgeons and no consensus is adopted regarding the ideal method. So, for good correction a sequential stepwise approach should be adopted which mainly concentrates on soft tissues rather than bone cuts.

Keywords: FFD, osteoarthritis deformity, correction, TKR

Introduction

Patho-anatomical changes in knee Osteoarthritis with Flexion deformity

Osteoarthritis is considered as a disease of the all joint with a multifactorial etiology. The pathological changes include cartilage degeneration, ligament derangements, subchondral bone changes, muscular impairments and so increased mechanical stress on the joint. Moreover, the problem is made worse by additional synovial inflammation, especially at the beginning. The mechanical aspects in particular have an effect on the disease's degenerative cascade. Genetic predisposition, age, obesity, joint stability and congruency, increased mechanical stress, and higher bone density were the risk factors that epidemiological studies attempted to identify. Congenital or developmental abnormalities, intra-articular fractures and ligamentous injuries, as well as systemic diseases like rheumatoid arthritis, haemophilia, hemochromatosis, post-infectious arthritis, and osteochondrosis dissecans, can all be causes of OA. It can also result from congenital or developmental abnormalities [1, 2].

When the dynamic equilibrium between forces that cause damage and systems that repair it is disturbed, OA begins to form in the joint. Despite the fact that many individuals with radiographic evidence of OA don't exhibit any symptoms associated with the condition, this imbalance is believed to be the primary factor contributing to the illness' degenerative nature and may cause pain and incapacity [2-4].

The stability, usability, and alignment of the knee joint are determined by the interaction of the dynamic structures of the knee's ligaments and muscles, which respond differently in knee flexion and extension. If there is imbalance, the knee starts to obtain a deformed position in coronal plane (Varus or valgus). Varus is more commonly occurring and it is caused mainly by the presence of osteophytes that tent over medial collateral ligaments leading to ligamentous contractures, the abnormal mechanical load over the joint leading to bone loss medially, and the lateral collateral laxity. THE varus deformity may occur alone or combined with sagittal plane deformity like flexion more commonly or hyperextension [5, 6].

In osteoarthritis or rheumatoid arthritis, synovial inflammation causes fluid to build up in the joint, which causes a rise in intraarticular pressure and edoema, both of which are chronic conditions. The joint then moves into flexion, which is both its position of greatest accommodation and a means of pain relief.

This flexion deformity is a positional deformity and not a contracture yet. If the osteophytes formation is more in posterior femur and tibia, they start to tent over the posterior capsule. Moreover, the anterior tibial or "anvil" osteophyte and the intercondylar osteophytes mechanically restrict extension. As the posterior capsule contracts alongside other soft structures like the posterior oblique ligament, semimembranosus, and popliteofibular ligament over time, positional flexion evolves into a permanent flexion deformity. [6-9].

Osteophytes and loose bodies must be removed from the posterior knee area in order to properly measure the extension gap following total knee replacement surgery. According to what will be covered subsequently, this clearing might be adequate on its own without further posterior soft-tissue release. On the other hand, improper flexion-extension gap balancing may result from insufficient excision of the posterior osteophytes. In knees with flexion deformity, the extension gap is often smaller because the posterior soft-tissue components are more taut. This disparity typically increases if a coronal plane deformity is present, necessitating more extensive soft tissue releases. To help close the significant flexion gap and bring it into equilibrium with the extension gap, further measures may also be required, such as upsizing, posteriorly moving, and minor flexing of the femoral component. So, to perform a TKR surgery in knee with flexion deformity is a challenge and should be performed with meticulous technique [6, 7].

Although not common, more severe cases could result in significant tightness and shortening of the gastrocnemius heads and hamstring tendons. On the posteromedial side of the knee joint, chords can be felt where the hamstring shortened tendons are, which often get taut during knee extension. Flexion contracture is primarily caused by soft tissue contracture, with just a few osteophytes present, with the exception of a few unusual situations including hemophilic arthropathy, extended immobilisation, and neuromuscular problems [7, 10, 11].

Gait changes in knee Osteoarthritis with Flexion deformity

Many symptoms of ambulation problems in people with knee arthritis include the inability to stand for extended periods of time, increased energy consumption, shorter and slower strides, and fixed flexion deformity. Also, there is a quadriceps weakness and extension lag that may be not obvious in the set of the fixed flexion contracture but it may manifest later after total knee replacement and so requires a meticulous physiotherapy program. If there is a bilateral flexion deformity with one side being more severe than the other, or if there is a unilateral flexion deformity, the increased aberrant forces at the joint while standing and walking result in an irregular gait pattern. The difference in leg length that results from flexion more clearly demonstrates this aberrant gait pattern. The body's attempt to compensate for these stresses by walking may start spreading pathological anomalies upward to the pelvis and spine, exacerbating the problem [7, 12].

In cases of fixed flexion contracture, gait modifications may include shorter walking distances, no heel striking, knee flexion at the start of the stance phase and throughout the gait cycle, placing the foot flat on the ground when there is less than 15 degrees of extensor lag, and toe walking when there is more than 15 degrees of extensor lag. Knees that are stiff when walking popliteal angle is shortened. Circumduction gait and stiff knee gait are further options. The foot may start to move forward at a less acute angle and imprint more. During the swing phase, hip flexion increases as the body is driven forward. Walking with a progressively hunched posture and limping causes stride length to shorten [13-15].

Other flexion contracture symptoms, such as anterior knee pain and compensatory behaviours like hip flexion deformity with the lumbar lordosis, have even more negative effects on gait. The spinal imbalance caused by knee flexion contracture has a significant impact on the three-dimensional trunk kinematics during level walking and relaxed standing. Chronic pressure on the popliteal fossa may cause the tibial nerve, common peroneal nerve, and other contents to swell. Hence, knee flexion contractures have a variety of functional effects, making it difficult to undertake weight-bearing activities or sit or lie down comfortably. Daily chores get harder as they require more effort to execute. The patient's social and personal elements of life are impacted [13-15].

Total Knee Arthroplasty in Flexion Deformity

Fixed flexion deformity (FFD), which is usually linked to varus deformity, is present in about 60% of knees undergoing TKA. Surgical techniques like the excision of the posterior osteophyte, the release of the posterior cruciate ligament, the posterior capsular release or capsulotomy, and the requisite femoral bone resections are required in order to achieve the intraoperative full repair of this deformity [9, 16]. As previously mentioned, residual flexion deformity affects gait kinematics, adding to the quadriceps' burden and increasing pressure on the patellofemoral joint. This ongoing functional impairment causes patients' unhappiness [16]. Most small flexion contractions are permanent, while some may gradually go away following surgery. As a result, it is essential to pursue a strict, precise physical therapy programme that seeks to repair all intraoperative issues and maintain recovery after surgery [9, 16].

Normally, knee extension should reach $180^{o} \pm 5$ degrees. The limited full active extension is called extension lag and it may be associated with FFD. A severe flexion deformity, according to some writers, is defined as a fixed flexion contracture with an angle more than or equal to 20° [17]. More severe flexion contractures are typically seen in rheumatoid arthritis compared to osteoarthritic knees [18]. The phrase "stiff knee," which also refers to knees with a range of motion of less than 50 degrees and is associated with a permanent flexion deformity, is another that appears frequently in literature [19].

Satisfactory postoperative range of motion is a key indicator of TKA success and a critical criterion in most knee grading systems. For daily tasks, a minimum of 90 degrees of active flexion is needed [20]. Particularly, the typical walk demands 67° of knee flexion, and climbing or descending stairs calls for 83°, 90°, or 93° of knee flexion, respectively [20, 21]. Several religious and cultural traditions, such as Muslim prayer, which requires the motion of prostration (Sujood), have greater demands than the approximately 110-115 degrees of flexion necessary by older people's lifestyles in western countries [21].

The root causes of issues in flexion contracture situations will be reviewed before the surgical approaches and postoperative care are presented.

Knowing the connection between a permanent deformity and tightness of the soft tissue structures at the back of the knee, which corresponds to the concave side of the deformity, is necessary to comprehend the therapeutic philosophy. In fact,

enough bone can be removed from the proximal tibia and distal femur to allow the knee to fully extend. On the surface, it would seem that treating fixed flexion contracture in this way would be adequate to entirely resolve the issue. It would be necessary to remove a lot of bone, take into account any probable quadriceps lag, and perform a successful postoperative rehabilitation [8, 9, 20, 22, 23].

Examining the flexion contracture issue so quickly is wrong. Without first paying close attention to the soft tissues, it is insufficient to repair the deformity by removing bone while extending the knee. It is accurate to claim that the anterior bulging of the surrounding soft tissue sleeve. The bulk of the bulge is actually in the anterior direction, despite their being a bulge or surplus of soft tissue in the medial and lateral directions. The anterior soft tissues expand when bone is removed to enable full extension, or a relative quadriceps laxity appears. Yet, with this kind of bone excision, the locations on the medial and lateral sides of the knee converge. Also, leaving the tight posterior structures increase incidence of residual deformity [8, 9, 20].

Many surgeons utilise the measured resection technique in traditional total knee arthroplasty, in which the bone is removed at the same size as the prosthesis. Moreover, knees are generally sufficiently deflexed to allow for surgical exposure, especially when under anaesthesia, so that it rarely poses a difficulty. Osteophytes are meticulously eliminated from all knee segments following the precise resections. With a curved osteotome and curette, osteophytes are removed from the posterior femoral condyles and circumferentially around the tibial plateau (Fig. 1). This crucial phase must be a part of every surgical procedure used to address abnormalities of every kind. The knee's flexion and extension gaps will be assessed next. The degree of the deformity, the firmness of the fixation, and the patient's age will all have a significant impact on the discrepancy [24]. It's time to adjust the extension gap between soft tissue releases that are performed sequentially to balance the flexion-extension space. It was believed that this represented a shift in the gap balancing strategy. After that, the soft tissue capsular attachments on the posterior femur of the patient are removed of the remains of the anterior and posterior cruciate attachments from the intercondylar notch of the femur. A periosteal elevator is then used to lift the posterior femur capsule (Fig.2) [9, 16, 20, 22, 24, 25].

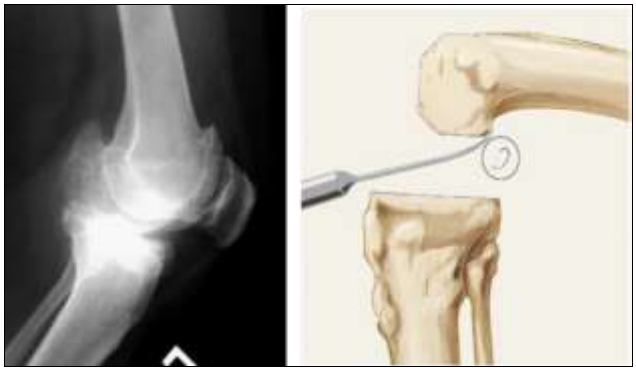

Fig 1: Posterior osteophytes affect flexibility contracture. These osteophytes must be removed to prevent the onset of a protracted flexion contracture brought on by their anchoring to the posterior capsule [16]

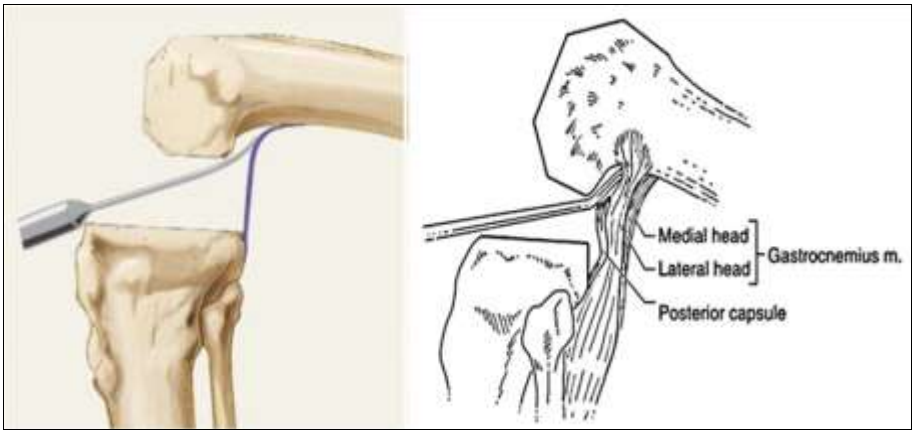

Fig 2: The posterior capsules may need to be separated from the femur as they contract. Although this treatment is rarely required because the capsular release and capsulotomy are often sufficient, the gastrocnemius muscle insertion may also need to be released in situations of significant persistent deformity [16]

In order to release the gastrocnemius muscle origins from the femur, the extension gap is evaluated once more to determine whether further dissection is necessary. The extension gap is subjected to yet another evaluation. A posterior capsulotomy is carried out if extra release is necessary after carefully dissecting the lateral and medial corners all the way to the area directly behind the lateral and medial collateral ligaments. A novel yet technically challenging fusiform shaped capsulotomy has been proposed by several authors. The collateral ligament's attachments are left in place (fig.3). If the extension gap is still too narrow after the posterior capsule, gastrocnemius muscle origins, posterior corners, and posterior parts of the collateral ligaments have all been removed, more of the distal femur will be cut away (Fig.4). The distal femur's further bone resection is done as a last resort rather than prior to soft tissue releases because it has a considerable negative impact on joint mechanics by moving the joint line proximally [9, 16, 20, 22, 25].

The majority of the deformity will be corrected, and equal flexion-extension gaps will be guaranteed, by freeing soft tissues from the posterior aspect of the joint and enabling enough proximal migration of the joint line. In order to improve joint stability, a more restrictive prosthesis with a high central tibial spike may be used if the joint is still too tight in extension and/or too loose in flexion. Full correction is frequently not possible in individuals with severe disability, considerable polyarticular deformity prior to surgery, and refusal to resume to typical activities after surgery. As a result, there won't be a significant functional compromise [20, 24].

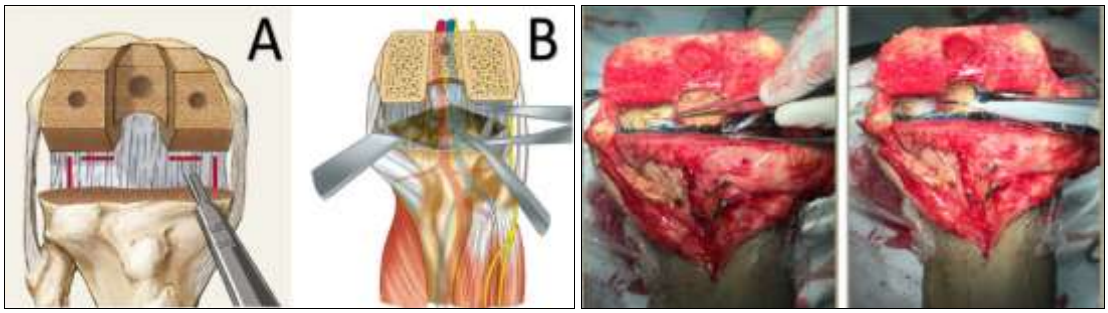

Fig 3: Careful transection of the posterior capsule and release of the collateral ligaments with a medial and lateral longitudinal incision may be necessary for FFD that ranges from moderate to severe (A) [16], a fusiform capsulotomy may be also used as new alternative technique to allow more correction (B), the capsule is grasped by Kocher or forceps and lower output electrosurgical knife. The only structures that can be removed are the lateral and medial collateral ligaments, the posterior femoral condyles, and the tibial plateau (B*) [24]

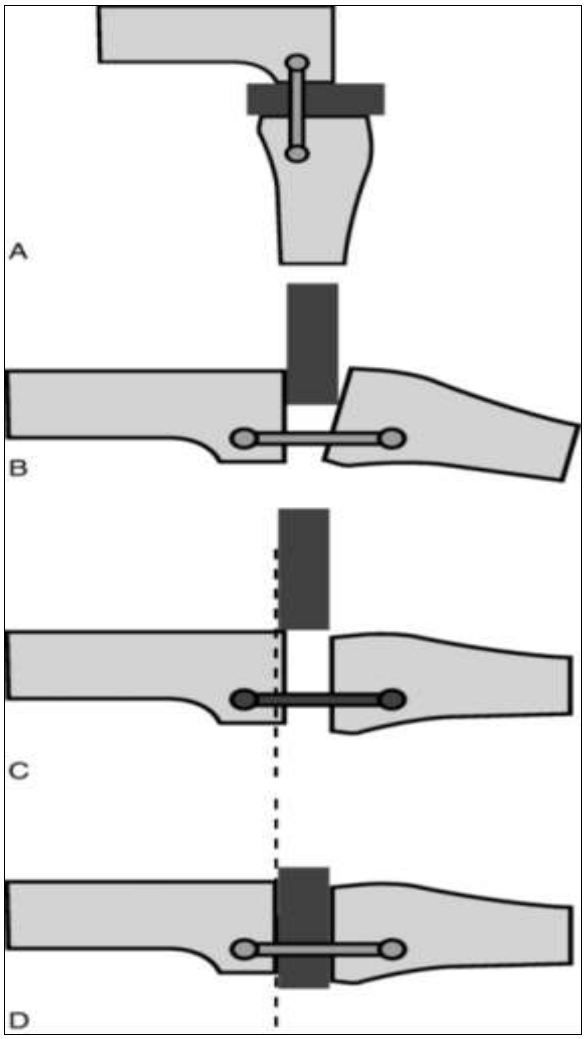

Fig 4: By removing bone, the gap can be balanced. A spacer block can be used to check the flexion gap. B. The gap is narrower in comparison to the block's size. D. To make place for a block the same size as the flexion gap, more bone is taken from the distal femur. D. The space between the flexion and extension is equal [16]

David W. and colleagues assessed the effect of distal femoral resection on the correction of flexion deformity. They adopted a strategy depending mainly on bone resection to correct deformity rather than soft tissue releases. They used navigation system and cruciate retaining prosthesis and found that to correct 10º of FFD you need to resect 3.55 mm more from distal femur according to certain formula [26].

Following surgery, it is desirable to virtually totally rectify the abnormality. With appropriate, individualised postoperative treatment, the knee is expected to be able to extend fully even with the less severe flexion contractures [20, 24].

Postoperative management

After surgery, patients typically adopt the flexion position due to extensor lag and muscle weakness. Physiotherapy programmes must make sure that they passively maintain full extension every day in order to treat any residual deformity and prevent it from developing again. It may take patients several months to reach full active extension [20, 24, 27, 28].

After being discharged, the patient needs to be shown how to put on a graded hinged knee brace and passively completely extend their knee. If patients begin to experience a recurrence of their flexion contracture after the first few weeks, a manipulation should be performed. In some serious cases, a long leg cast in full flexion may be used. Nonetheless, the degree of correction achieved through surgery must always be one that the patient can sustain. It's critical to postpone resolving persistent irregularities. Even though this patient population frequently reacts favourably to motion in flexion, total extension must be maintained at all times [9, 20, 21, 22, 28].

Conflict of Interest

Not available

Financial Support

Not available

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How to Cite This Article
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