Iliotibial band traction syndrome in guided motion TKA
A new clinical entity after TKA

Lucas Luyckx, Thomas Luyckx, Johan Bellemans, Jan Victor

From the University Hospitals UZ Pellenberg, KUL, Leuven, Belgium

INTRODUCTION

Outcomes research after TKA has unveiled several functional deficits in the activities of daily living (17). In recent years, several new knee implants have been introduced aiming at better stability and higher flexion (1, 2, 10, 20, 21). One implant (Journey®, Smith & Nephew, Memphis, TN, USA) followed the principle of guided motion through asymmetric tibiofemoral surface geometry and an asymmetric cam and post design, in an attempt to reproduce a specific kinematic model (21). Clinical research and fluoroscopic analysis on a large patient cohort confirmed the validity of the concept in showing femoral posterior translation and tibial internal rotation with increasing flexion (23).

However, concerns have been raised regarding potential adverse effects of high-flexion guided motion designs (4, 9).

Keywords: guided motion TKA; ITB traction syndrome; lateral knee pain.
As the incidence of lateral knee pain associated with the Journey® implant appeared to occur far more frequently compared to other implants used by the senior authors (JB, JV), a dedicated study was undertaken. The goal of the study was to document the incidence of lateral knee pain and subsequent associated treatment modalities in a consecutive cohort of 1085 patients and to provide a biomechanical explanation of the observed symptoms.

### MATERIAL AND METHODS

Relevant data of 1102 consecutive cruciate substituting, guided motion TKA’s (Journey®, Smith and Nephew, Memphis TN, USA) implanted in 1085 patients, were extracted from the database and patient files. All patients underwent surgery between April 2005 and January 2009. Thirty-two knees (2.9%) were lost to follow-up, leaving 1070 knees available for review. The minimum follow-up was one year and the mean follow up 2.5 years (range, 1-4.7; standard deviation [SD] : 1.0).

The average age for all patients included was 66.5 years (range : 22-90 ; SD : 9.83). There were 36% (378) men and 64% (678) women (table I). Of the 1056 patients, 550 received a TKA on the right side, 492 on the left side and 14 had a bilateral TKA.

Center 1 (AZ-St-Lucas, Brugge) provided 355 TKA’s, center 2 (UZ-Pellenberg, Leuven) provided 715 TKA’s. There were no significant differences between the two centers with respect to revision ratios (2% in center 1 (7 knees), 2% in center 2 (15 knees)), the male-female

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ratio (both centers 36% men and 64% women), the incidence of ITB friction like symptoms (9.2% in center 1, 6.6% in center 2) or demographic variables, allowing the data to be pooled.

We systematically reviewed the patient files, aiming at documentation of symptoms mimicking the well known ITB friction syndrome: symptoms of focal tenderness over the lateral femoral epicondyle and lateral knee pain between 20° and 80° of motion, known as a painful arc (19). Interventions such as oral medication, lateral steroid injection and lateral release were recorded. In an attempt to quantify the severity of complaints, we registered the number of follow-up visits. Also, the time of onset of symptoms, relative to the date of the index operation was noted.

Statistical analysis

Statistical analysis was undertaken using Student’s t-test and the Chi² test. This was performed using SPSS statistical software (version 16.0; SPSS Inc., Chicago, Illinois). All p-values were two-sided and considered significant when smaller than 0.05.

RESULTS

Symptoms mimicking the ITB friction syndrome were observed in 81 knees (7.6%). Four patients were excluded from subsequent analysis because further investigation revealed an occult infection, leaving us with 77 knees (7.2%). The mean age for all patients included in this subgroup was 66 years (range, 44-83; SD: 9.42). There were 36 (47%) men and 41 women (53%). The right knee was concerned in 38 of them, the left in another 38 and one was the left knee of a bilateral TKA (table II).

The mean number of follow-up visits in this group was 7.1 (range, 2-29; SD: 4.37) compared to 2.8 (range, 1-8; SD: 1.15) in the group without problems. This was a statistically significant difference. The time of onset of ITB friction-like symptoms was on average 6.3 months after TKA (SD: 5.59).

Initial conservative treatment for the ITB friction like symptoms consisted of anti inflammatory medication. All patients with the 77 knees exhibiting ITB friction like symptoms received oral NSAID’s. In 46% of these (35 knees), pain persisted and symptoms were prominent, so that a local steroid injection was given. In 22 knees (28.6%) the pain still persisted, leading to a surgical release of the ITB.

The overall survivorship with partial or total implant revision as an endpoint was 97.9%. Twenty-two knees needed partial or total implant revision, consisting of revision for infection (6 knees, 0.5%), revision for tibial component loosening (6 knees, 0.5%), revision for tibiofemoral dislocation (3 knees, 0.3%), revision for patellar component loosening (5 knees, 0.4%), revision for instability (1 knee, 0.1%) and secondary patellar resurfacing (1 knee, 0.1%).

DISCUSSION

This study aimed at documenting a specific lateral pain syndrome after implantation of a guided-motion knee implant, which we defined as the ITB traction syndrome.

The study has several limitations. First, it was conducted in a retrospective manner. Second, pain is a subjective outcome variable and pain rating scales were not available. We decided to quantify the severity of symptoms by the number of the follow-up visits, which was significantly larger in the ITB traction subgroup (mean of 7.1 follow-up visits, compared to 2.8 in the group without problems). Also the use of anti inflammatory medication, corticosteroid injections and surgical release of the ITB, as objective outcome parameters were noted.

Seventy seven patients (7.6%) were identified with a specific lateral pain syndrome, mimicking ITB friction syndrome. This syndrome was previ-
ously unreported in association with TKA. Tendonitis of the patellar tendon or pes anserinus has been reported after TKA, but lateral knee pain is rather uncommon. Pandher et al reported a case of posterolateral knee pain after TKA, but they stated that the pain was caused by biceps femoris tendonitis and did not resemble the ITB friction syndrome (18). Symptoms of the ITB traction syndrome are lateral and anterolateral knee pain, occurring with motion, and leading to a painful arc. Symptoms started at a mean of 6.3 months post-operatively, suggesting the guided motion pattern imposed by the implant was generating the pain syndrome as patients became more active after the operation.

The ITB is a complex structure, receiving the insertions from the gluteus maximus and tensor fasciae latae muscles. The ITB does not have a single insertion at Gerdy’s tubercle, as is generally thought, but has a wide periarticular insertion: an insertion at the linea aspera, one at the lateral epicondyle, a patellar insertion, which is wide and fuses itself to the lateral retinaculum, a direct insertion, which widely inserts at Gerdy’s tubercle and a capsular-osseous insertion, also known as the femorotibial ligament (25). Especially the patellar insertion, which connects the ITB to the patella and forms the lateral retinaculum, has been described by many authors, but the literature includes different interpretations of the tissue bands, the layers and their denominations.

Earlier studies, as those by Fulkerson and Gosseling described the anatomy of the lateral retinaculum in two separate layers: the superficial oblique layer and the deep layer (7). Currently it is agreed that there are three distinct layers: a superficial, an intermediate and a deep layer. The superficial layer consists of the deep fascia of the thigh. The deep fascia is not attached to the patella but is adherent to deeper tissues laterally, thus acting as a brace. The ITB is a derivative of the deep fascia and by its expansions contributes to the retinaculum. The longitudinal fibres of the ITB merge with those of the quadriceps aponeurosis lateral and adjacent to the patella, forming an intermediate layer. These longitudinal fibres are reinforced by the transverse retinaculum, which consists of superficial fibers of the ITB, the iliopatellar or arciform fibres, and the deeper transverse fibres of the ITB. The most substantial structure consists of the deeper transverse fibres which are dense and anchor the lateral edge of the patella and the tendon of vastus lateralis obliquus to the ITB. They are not a distinctly separate layer, but adhere to the deep aspect of the ITB.

The deep layer consists of the joint capsule. The joint capsule is thickened laterally and forms the lateral patellofemoral ligament. Similarly, another condensation of the joint capsule is termed the meniscopatellar or patellomeniscal ligament. Some studies noted however that these capsular ligaments vary considerably and are not always found (13).

The most prominent structure of the lateral retinaculum and the strongest, as shown by Merican et al consists of the ITB-patellar fibres (16). The location, bulk and orientation of those fibers suggests that they may play an important role in the lateral restraint of the patellofemoral joint. However these transverse fibres lack a direct connection to the femur, and they are more prominent and are consistently found, as compared to the lateral patellofemoral ligament.

Moreover, the part of the ITB to which the deeper transverse band is attached is relatively fixed in a longitudinal direction as a short segment. Proximally, it is anchored to the proximal aspect of the femoral condyle and distally to Gerdy’s tubercle on the tibia. In view of its longitudinal orientation, tension in the ITB would not be thought to affect patellofemoral tracking. However, when the bony fixation mentioned above and the transverse fibres are taken into account, it is likely that tension in the iliotibial band plays a role in the function of the lateral retinaculum (13).

This was confirmed by Merican et al, who demonstrated that with increasing ITB loads, the patella tracked and tilted more laterally in the mid flexion range and rotated more laterally later in the flexion cycle. Simultaneously, the tibia rotated externally up to 13° (90N load) (14).

The function of the ITB is complex. Together with the tensor fasciae latae and the gluteus maximus fibers, it plays a role in stance and monopodal balance. It also plays a role in the external rotation of the tibia and it is considered as a primary lateral...
stabilizing ligament of the knee. This stabilizing effect is provided by its attachments to the patella preventing the ITB from shifting posteriorly during flexion, creating a higher tension in the transverse fibres of the ITB. This tension provides a lateral stabilizing effect on the knee during flexion (26).

Some authors have stated that the ITB friction syndrome is not caused by friction between the ITB and the lateral femoral epicondyle, but rather by compression of the ITB against a layer of highly innervated fat tissue that intervenes between the ITB and the epicondyle. This compression occurs mainly at 30° of flexion, probably as a result of the internal rotation of the tibia at that point (5,6).

Gosh et al demonstrated that a conventional cruciate retaining TKA (Genesis 2, Smith & Nephew, Memphis, Tennessee) did not cause significant elongation of the lateral retinaculum. The lateral retinaculum of the native knee was longest in the flexed position and slackened in the final 70° of knee extension, which is consistent with the anatomy. After TKA no significant changes could be found (8).

In contrast, ex vivo experimental data comparing case to case tibial rotation in the native knee to tibial rotation after Journey TKA clearly showed excessive internal rotation of the prosthetic knee in the loaded setting (24). Where the axial kinematic pattern of the prosthetic knee mimicked well the passive kinematics of the native knee, the prosthetic knee failed to adapt to loading as did the native knee in the experimental setting. These findings were confirmed in vivo, based on fluoroscopic kinematic analysis (23). In some patients posterior femoral translation exceeded 23 mm, creating internal tibial rotation over 10.8° with increasing flexion. This kinematic pattern is imposed by the cam-post geometry of the implant and partially dependent on the axial alignment of the tibial component. In patients with excessive internal rotation of the tibia during flexion, the length of the ITB is greater than in patients with a natural rotational pattern (fig 2). In the native knee, contraction of the biceps femoris reduces internal rotation of the tibia with increasing flexion and thereby reduces traction on the ITB (11,12,22). Excessive internal rotation, as observed in some patients after Journey TKA could lead to eccentric loading of the ITB and induce the observed symptoms.

We noted a tendency to a higher percentage of men amongst the patients with ITB traction syndrome: 46% compared to 36% in the overall group (not statistically significant). Possible explanations for this tendency could be related to gender associated general ligament laxity, activity level or muscular force.

Patients presenting with the ITB traction syndrome were initially treated conservatively, using oral medication and local injections with steroids. This treatment was significantly helpful in 71.4% of the patients. Symptoms persisted in 28.6% and those patients were treated with a surgical release of the ITB. This release was performed through a lateral skin incision and transected the anterior fibres of the ITB extending into the lateral retinaculum (3,15,26) (fig 3).

Based on the observed symptoms and the incidence of occurrence after TKA, we believe that a modification of the prosthetic design is desirable. The amount of posterior translation of the lateral
femoral condyle during flexion needs to be reduced, resulting in less guided internal rotation of the tibia.

REFERENCES