Acta Orthop. Belg., 2007, **73**, 118-121

CASE REPORT

Posterior knee dislocation after total knee arthroplasty in a patient with multiple sclerosis A case report

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A patient with multiple sclerosis (MS) and severe osteoarthritis of the right knee with valgus deformity developed early in the postoperative period a posterior dislocation of the knee after total knee arthroplasty (TKA), due to severe spasm of her hamstring muscles. At revision surgery the posterior cruciate retaining prosthesis was exchanged for a non-linked constrained knee prosthesis. Technical considerations are described for this rare complication, which remains a serious clinical challenge for the orthopaedic surgeon.

Keywords : knee arthroplasty ; multiple sclerosis ; muscle spasm ; dislocation ; posterior stabilised.

INTRODUCTION

Multiple sclerosis (MS) is a progressive disease of unknown origin, affecting the white matter of the central nervous system. Besides a wide range of neurological symptoms, typically evolving in alternating periods of exacerbation and remission, the disease is often characterised by severe muscle spasms. Limb surgery in patients with MS is associated with local exacerbation of symptoms (*1*). A less favourable outcome of total knee arthroplasty (TKA) due to spasm of the hamstrings has been reported in such patients (*2, 3*). However, the orthopaedic literature provides scarce documentation on outcomes after TKA in patients with MS. Therefore, the treatment of symptomatic osteoarthritis of the knee in patients suffering from MS presents a serious clinical challenge to the orthopaedic surgeon.

We present a case of posterior dislocation of the tibia relative to the femur following primary TKA in a patient with multiple sclerosis.

CASE REPORT

A 64-year-old female, with a 30-year history of MS, was referred to our hospital with progressive complaints at the right knee. The patient's history and physical examination were suggestive of osteoarthritis, which was confirmed by plain radiographs (fig 1). Due to pain, the patient had become wheelchair-bound. Because of the severity of the complaints and the relatively mild course of the MS in this patient, TKA was judged feasible and was

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No benefits or funds were received in support of this study

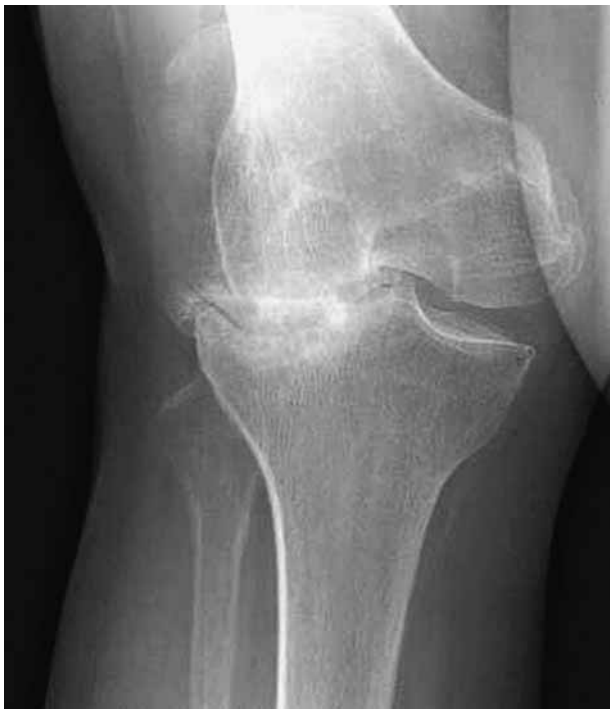

Fig. 1. — Preoperative antero-posterior radiograph of the right knee showing evident signs of osteoarthritis.

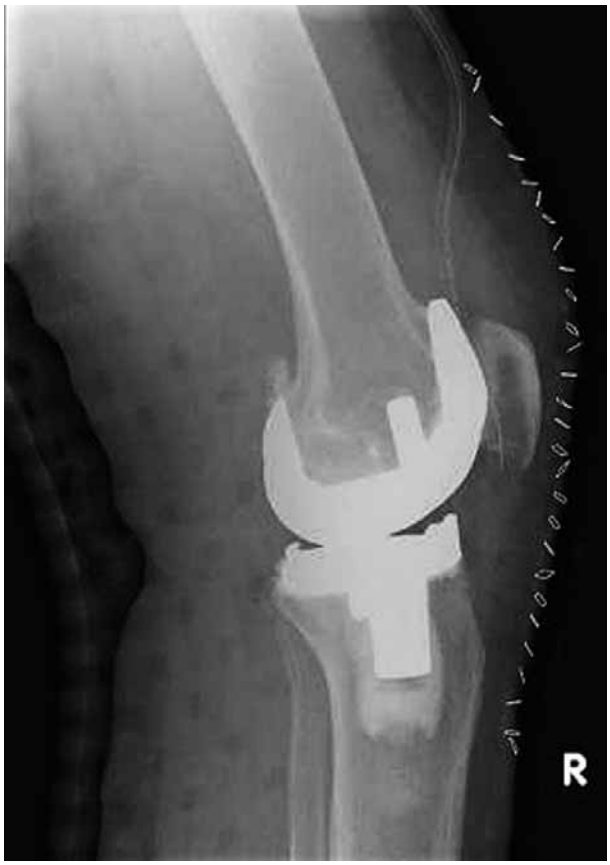

Fig. 2. — Anteroposterior and lateral radiographs of the right knee after implantation of the primary TKA.

performed. A fixed-bearing posterior cruciate ligament retaining cemented knee prosthesis (Kinemax, Stryker, Allendale, New Jersey, USA) was implanted (fig 2) and a correct alignment of the knee in flexion and extension was obtained.

Postoperatively, the patient developed severe spasms of the ipsilateral hamstring muscles. To decrease the spasms and to prevent a flexion contracture, high-dose oral baclofen was administered and the knee was immobilised in an extension splint. However, despite these prophylactic measures, a posterior dislocation of the knee occurred one month postoperatively (fig 3). No neurovascular complications were observed at the lower leg. Closed reduction of the dislocated prosthesis was unsuccessful and a revision of the TKA was planned. In order to prevent muscle spasms after the revision, the patient received prophylactic botulinum toxin type A injections in the right hamstring muscles.

The chosen implant for the TKA revision was a total stabilised non-linked constrained prosthesis (Scorpio TS, Stryker, Allendale, New Jersey, USA) (fig 4). At the time of knee revision we observed a major flexion- extension mismatch, exceeding 40 mm. Complete extension of the knee was only possible after extensive release of the contracted posterior and lateral soft tissues (iliotibial tract, popliteal muscle, biceps femoris tendon, capsule and lateral collateral ligament). Posterior translation of the femoral component was performed to additionally decrease the flexion gap. A sliding osteotomy of the medial epicondyle (*2*) achieved an acceptable re-tensioning of the medial structures.

Unfortunately, we could not avoid elevation of the joint line (resection of 20 mm of the distal femur), which resulted in a significant patella infera. The revision surgery had resulted in multidirectional stability with a nearly full range of

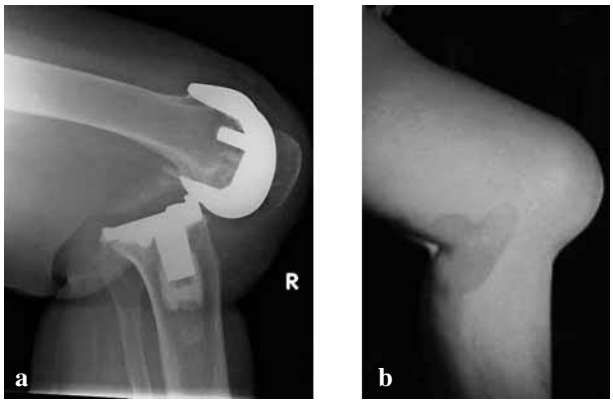

Fig. 3. — a. Lateral radiograph of the posteriorly dislocated TKA ; b. Clinical picture of the knee after TKA dislocation.

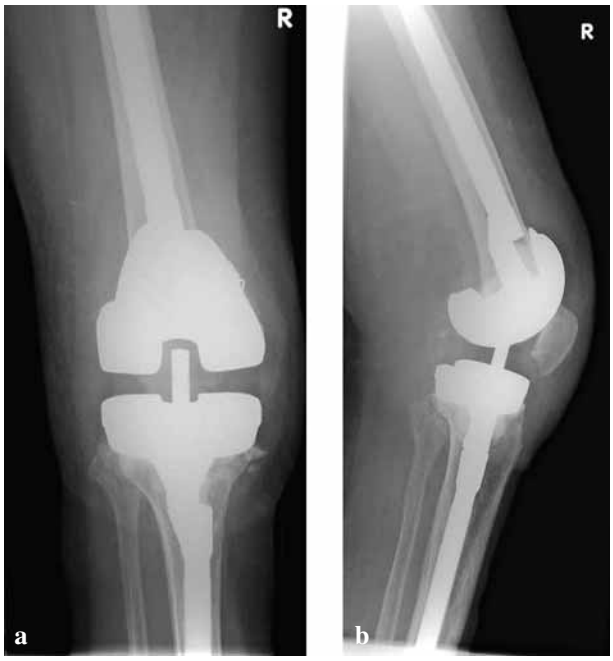

Fig. 4. — Anteroposterior (a) and lateral (b) radiographs of the knee showing the revision knee prosthesis.

motion. Postoperatively, high-dose oral baclofen was again administered. Combined with the muscle release and botulinum injections, this proved to be successful to prevent the severe spasms seen after the first operation. An extension brace was applied allowing gradually more flexion of the knee. One year postoperatively, no dislocation or other adverse events have been noticed.

DISCUSSION

This case illustrates the risk of knee instability and muscle spasm following TKA in a patient with MS. Rao *et al* already reported such instability (*13*). However, complete posterior tibial dislocation in a patient with MS has not been reported before. Erceg *et al* recognised joint laxity in knee flexion as the major cause of posterior subluxation of a TKA in patients with Parkinson's disease (*3*). Furthermore, Pagnano *et al* discussed flexion instability of the knee after TKA as the most important risk factor for posterior dislocation (*12*). In our patient, the posterior tibial dislocation was predominantly caused by excessive spasm of the hamstring muscles during flexion of the lax knee joint. The dislocation might have been further facilitated by the absence of a posterior stabilising mechanism in the primary TKA and an early postoperative rupture of the posterior cruciate ligament (PCL). Several clinical and cadaver studies showed the relative increase in width of the flexion gap in comparison to the extension gap after loss of the PCL (*10, 11*). This is probably due to laxity in flexion of the posterior capsule and other dynamic stabilisers (e.g. gastrocnemius and medial hamstrings muscles and iliotibial band) and tautness in extension (*10*).

There are several known factors which increase the risk of early rupture of the PCL (*5, 8, 9, 16*). These factors include an increased tibial slope, a tight or loose flexion gap, excessive proximal tibial resection or partial release of the PCL. In addition, the PCL may already have been weakened due to degenerative changes (*9*). Furthermore the PCL in our patient with a valgus knee deformity may have been stretched or elongated because of its more medial position after correction (*4*). Therefore, the retained PCL was probably less functional and a posterior stabilised prosthesis would have been a better initial choice. However, even with a PS knee, the "jumping distance" between spine and cam in flexion could still allow posterior dislocation (*1*).

Revision TKA should focus on precise balancing of the flexion and extension gaps in conjunction with a posterior stabilised prosthesis (*3, 12*). During the current revision surgery, we judged that the prosthesis should offer more than only posterior constraint. The aim was to achieve multidirectional stability despite a persisting minor flexion extension mismatch. Furthermore, a fully stabilised prosthesis has a larger and thicker polyethylene intercondylar post offering greater resistance to potential dislocating forces in case of recurrence of hamstrings spasms. Of course, there can be concerns about an increased rate of long-term failure with increased constraint. However, studies by Font-Rodriguez and Trousdale have revealed the opposite. Trousdale showed an 80% survivorship at 15 years, while Font-Rodriguez at 7 years reported a 98% cumulative survivorship (*6, 17*). A fully constrained prosthesis in combination with extensive muscle release proved to be successful in the current case. It is hard to define the contribution of the pre-operative injections with botulinum to the successful outcome in the current case. Shah *et al* recently described the successful treatment of a flexion contracture in a patient with TKA and Parkinson's disease (*14*). The clinical improvement after injections with botulinum toxin typically lasts for three months and we have seen no return of spasms for over a year now.

In conclusion, the risk of TKA instability in a patient with MS should not be overlooked. If conservative treatment fails in an otherwise mobile MS patient, TKA may be considered. In these cases a posterior stabilised prosthesis should be used. Flexion instability should be carefully avoided and flexion of the knee should be gradually built up postoperatively. Patients should be informed about these risks and the possible additional surgical interventions that possibly have to be performed to gain an acceptable outcome.

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