



Case report

Total Knee Arthroplasty Complicated by Posterior Dislocation of a Polyethylene Insert and Neurovascular Compromise

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ABSTRACT

Dislocation of the polyethylene insert is a rare complication of total knee arthroplasty (TKA), which has only been described in a handful of case reports. Here, we describe a report of a 55-year-old woman presenting 13 months after the primary TKA with signs of neurovascular compromise. A magnetic resonance image showed posterior extrusion of her polyethylene insert causing a mass effect on the gastrocnemius muscle and the popliteal neurovascular bundle. A multidisciplinary team including a joint reconstruction surgeon, vascular surgeon, and nerve specialist performed a revision TKA with peroneal nerve decompression. The polyethylene insert was noted to be dislocated, rotated 90 degrees, and incarcerated in the posterior knee.

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Introduction

Although total knee arthroplasty (TKA) has a relatively high success rate in the United States, it has several known complications, most common of which are infection, instability, and aseptic loosening [1]. Dislocation of the polyethylene insert (DPI) is a rare complication of TKA, which results in 0.3% to 1% of TKA revisions [1,2]. At this time, DPI has only been described in a handful of case reports. The immediate danger of DPI stems from its propensity to cause direct mechanical damage to surrounding structures such as the lateral retinaculum or patellar tendon [1,3]. Such damage has required revision TKAs or, in more extreme cases with irreparable damage to the extensor mechanism, arthrodesis [3]. Here, we report a case of TKA complicated by posterior DPI with both neurologic and vascular compromise, a combination of complications not yet reported in the literature.

Case history

Written informed consent for the publication of this case was obtained from the patient. A 55-year-old woman presented to our

institution for a second opinion at approximately 13 months postoperatively from a right TKA performed at an outside facility. Past medical history was most significant for osteoarthritis, diabetic neuropathy, and history of a previous deep venous thrombosis. Her chief complaint was ongoing numbness and weakness in the right lower extremity. She also reported difficulty with ambulation, claudication, muscle spasms, gait imbalance, and recurrent falls.

The patient underwent a right knee primary TKA at an outside hospital for Kellgren-Lawrence Grade III medial compartment osteoarthritis after failing conservative treatment measures. A mid-vastus approach was used, and DJO EmPowr implants were placed without any complications per the operative report (DJO Surgical, Austin, TX). She had been recovering well until 6 months postoperatively when she developed right lower extremity pain and knee instability without a specific inciting event. Per outside hospital records, she underwent knee aspiration, which was negative for infection and without micro-organism growth. Inflammatory markers were also nonconcerning for infection; her erythrocyte sedimentation rate was 12, and C-reactive protein was 3. Magnetic resonance imaging (MRI) and computed tomography (CT) angiogram (CTA) were previously obtained at the outside hospital and available for review. A radiograph of the right knee taken at our institution was notable for right knee effusion (Fig. 1). The MRI revealed displacement of the polyethylene insert extending approximately 2.8 cm beyond the margin of the tibial plateau with

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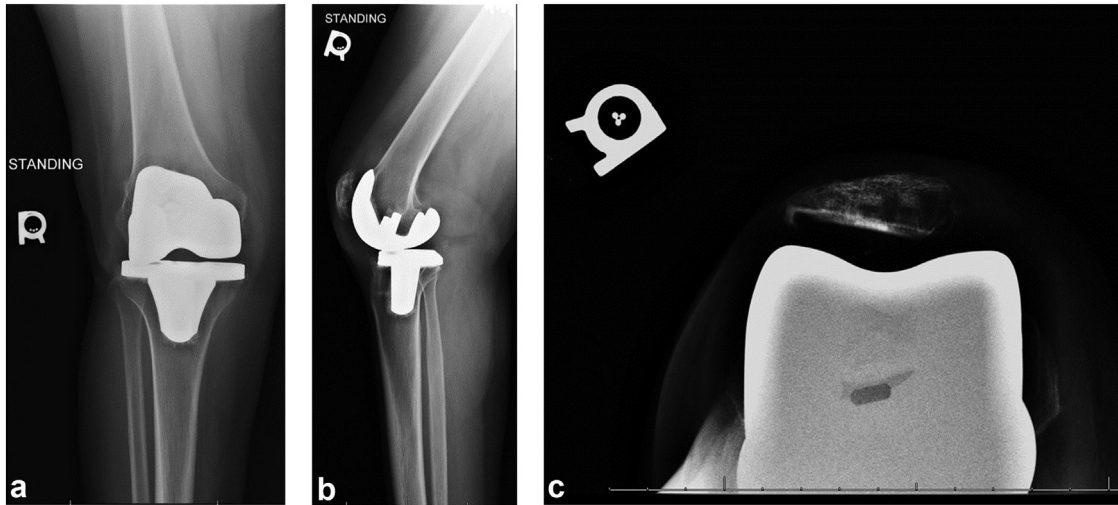


Figure 1. Preoperative radiographs of the right knee. Anteroposterior (a), lateral (b), and sunrise (c) views of the right knee show a right knee replacement without loosening or failure of the prosthetic components with a small knee effusion.

a mass effect on the head of the gastrocnemius and popliteal neurovascular bundle with concern for rotation of the spacer (Fig. 2). The CTA demonstrated a popliteal artery occlusion with reconstitution distally. Her ankle-brachial index score was 0.6, consistent with occlusion.

She presented to our institution to discuss surgical options. Her physical examination at that time was significant for a quadriceps atrophy, extensor lag of 20 degrees, and limited range of motion at the knee, approximately 20–70 degrees. Neurologically, she demonstrated weakness in tibialis anterior (2/5), extensor hallucis longus (EHL) (2/5), and extensor digitorum longus (3/5). The dorsalis pedis (DP) pulse was not palpable. She reported decreased sensation to light touch on the dorsum of the foot and in the first web space. Given the chronicity of her symptoms and the adequate reconstitution on her CTA maintaining perfusion to her extremity, continued outpatient workup was deemed appropriate. She was referred to a nerve specialist and a vascular surgeon within our institution for further evaluation and preoperative planning for revision arthroplasty.

Her physical examination at our nerve specialist was significant for an obvious deformity of the knee with prominence along the anterolateral aspect of the joint line. The posterior knee was tender to palpation. She demonstrated full strength (5/5) with dorsiflexion and plantar flexion at the ankle and toes. Sensation along the dorsum of the foot was intact, but she reported paresthesias in the

tibial nerve distribution. Tinel sign test was negative on her peroneal nerve and tibial nerve. Throughout the physical examination, she developed intermittent, severe spasms of her gastrocnemius and soleus muscles. These findings were concerning for concomitant tibial nerve pathology. The decision was made to release the peroneal nerve during the revision TKA and to monitor tibial nerve function postoperatively.

Vascular surgery recommended proceeding with revision arthroplasty without concomitant bypass given the reconstitution noted on CTA and the increased risk for complication such as periprosthetic joint infection and hematoma formation. Vascular surgery was readily available during the revision arthroplasty, however, in the case of intraoperative limb ischemia. Informed consent was obtained for a peroneal nerve release and a revision TKA arthroplasty, as well as potential vascular bypass. The aforementioned MRI was vital in determining the proximity of the neurovascular structures for operative planning.

The procedure was performed without tourniquet inflation because of the known popliteal artery occlusion. Doppler examination of the DP and the posterior tibialis arteries preoperatively revealed monophasic signal. The peroneal nerve release was performed first. Several tight bands existed at the muscle compartments, but there was no evidence of nerve compression or abnormality. A checkpoint nerve stimulator was used to stimulate the nerve with 2 mA and resulted in good muscle contraction.

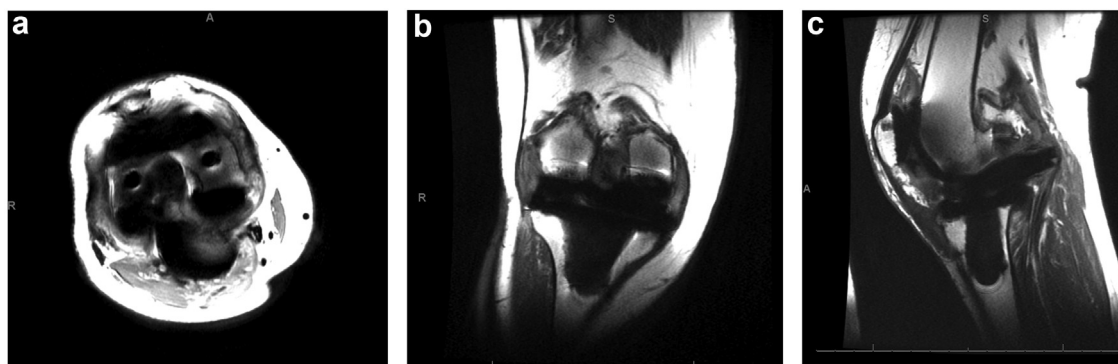


Figure 2. Axial (a), coronal (b), and sagittal (c) views from preoperative magnetic resonance imaging of the right knee. The sagittal view (c) demonstrates a 2.8-cm displacement of the polyethylene insert posteriorly with impingement of the neurovascular bundle and the head of the gastrocnemius muscle.

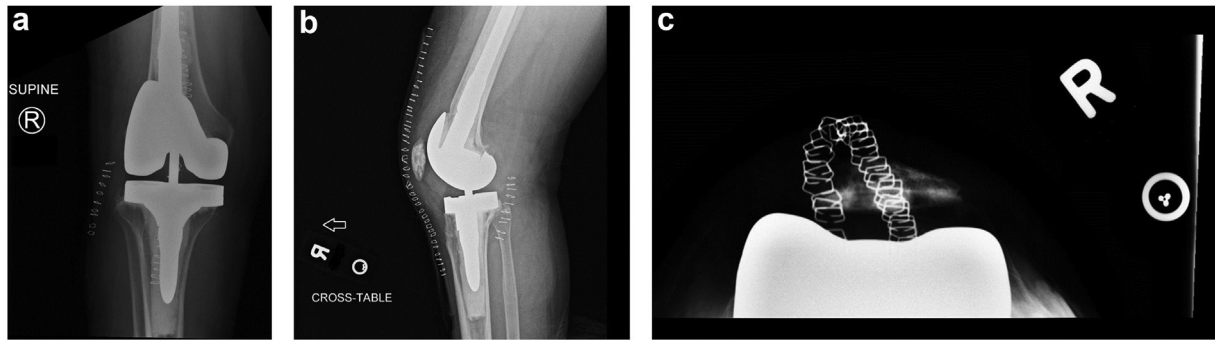


Figure 3. One-week postoperative radiographs of the right knee. Anteroposterior (a), lateral (b), and sunrise (c) views show expected overall alignment without immediate complications. Mild anterior soft-tissue swelling and a small joint effusion were noted in the lateral (b) view.

The nerve release was immediately followed by the revision TKA. A standard medial parapatellar approach was used. The polyethylene insert was noted to be dislocated, rotated 90 degrees, and incarcerated in the posterior knee. It was subsequently removed with a rongeur. No polyethylene wear or issues with the locking mechanism were noted. There were no significant flexion or extension gap imbalances noted. The femoral and tibial components were well fixed but able to be removed with minimal bone loss. Intraoperative tissue cultures were obtained. The tibia was sized to 2 Triathlon TS 5-mm medial and lateral augments used for mild deficiencies on the plateau (Stryker Corporation, Kalamazoo, MI). A 12 × 50-mm Triathlon TS cemented stem was assembled and placed. A size 3 Triathlon TS femoral component with 5-mm posterolateral augment and 12 × 100-mm stem was placed. A 29-mm Triathlon asymmetric patellar component was used. The decision was made to use a constrained nonhinged Triathlon X3 TS tibial insert size 2, 11-mm polyethylene liner.

After the final inserts were placed, Doppler was used to recheck the distal pulses. The DP pulse was now undetectable, but a monophasic posterior tibial pulse remained present with an intraoperative mean arterial pressure of 76. At this time, a vascular surgery team was consulted intraoperatively and recommended no acute intervention as the extremity remained perfused and any further intervention would unnecessarily increase the risk of hematoma formation or periprosthetic joint infection. The

arthrotomy was then closed, and the patient was woken from general anesthesia without complications.

At her 2-week postoperative examination, she demonstrated 2+ DP pulses with mildly weak ankle dorsiflexion, plantar flexion, and EHL strength with decreased plantar sensation. Intraoperative tissue cultures were held for 14 days and found to have no growth. Her 6-week postoperative examination was notable for 4/5 strength in ankle dorsiflexion, plantar flexion, and EHL. There was continued altered tibial nerve distribution sensation and calf atrophy. Otherwise, she had a well-healed incision, with improved range of motion from 10 to 100 degrees of flexion with <5 degrees of varus/valgus laxity and <5 mm of anterior-posterior laxity. These findings suggest continued, but objectively improved, peroneal and tibial nerve function as well as resolution of her vascular compromise. A radiograph obtained at 1 week postoperatively demonstrated well-positioned components (Fig. 3).

From her 1- to 12-month follow-up visits, her strength remained improved from her prerevision examinations, but she continued to report paresthesias and demonstrated weakness and foot drop. During this period, she also received multiple periarticular needle-based therapies by an outside care provider for her residual symptoms. Unfortunately, her postoperative course was complicated by a periprosthetic joint infection approximately 2 years after her revision surgery, which was managed with explant and placement of a static antibiotic spacer (Fig. 4). Due to extensive

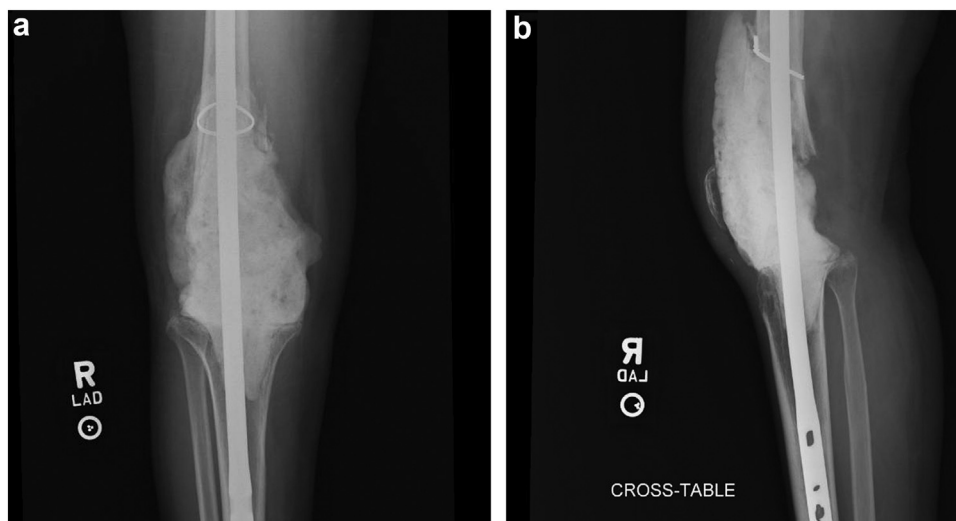


Figure 4. Three-month postoperative radiographs after explant and placement of antibiotic spacer. Anteroposterior (a) and lateral (b) views demonstrate continued overall neutral alignment with a long intramedullary rod, now effectively a functional arthrodesis.

bone loss, a non-articulating spacer was chosen for management. While awaiting her second-stage revision for infection, she was admitted to an outside hospital with sepsis. Given her ongoing medical comorbidities, treatment options including nonoperative management, knee arthrodesis, femoral-replacing hinge prosthesis, and above-knee amputation were considered. After careful consideration of the risks and benefits, the patient elected to manage her symptoms nonoperatively with observation and intermittent follow-up every 6 months, as she had a functional arthrodesis and acceptable ambulatory function.

Discussion

DPIs continue to be a rare complication in TKAs, and only anterior DPIs have been reported. The prevailing mechanism for anterior DPIs has previously been described by Davis et al., in which force projected onto the posterior half of the polyethylene insert causes a subsequent liftoff of the tibial component, culminating in the extrusion of the insert [4]. Existing case reports have somewhat differed on the cause of the posterior force, implicating the angle of the tibial portion [5], unresolved osteophytes [4], failed locking mechanisms [6], ligamentous laxity [7], and polyethylene wear and tear [3,8]. Although the source of the posterior force is different in each of these reports, they corroborate the mechanism proposed by Davis et al. [4].

A posterior DPI has not yet been reported in the literature at the time of this publication. However, it seems rather likely that force may have been projected onto the anterior half of the polyethylene insert, analogous to the mechanism proposed by Davis et al. [4]. Our patient endorsed recurrent falls, gait imbalance, and difficulty ambulating before her visit. It is likely that one of these falls generated the anterior force needed to dislocate the polyethylene insert posteriorly. On the other hand, the absence of inciting events would suggest a more chronic mechanism of anterior force such as polyethylene wear and tear as proposed by Hedlundh et al. [3,6,9]. This is unlikely, as no obvious polyethylene wear or issues with the locking mechanism were noted intraoperatively. It is also possible that the polyethylene insert was never fully engaged into the tray during her initial TKA, in which case, displacement was inevitable.

The posterior DPI caused a rather unique pattern of neurovascular compromise, as the popliteal artery, the bifurcation of the sciatic nerve into the common peroneal and tibial nerves, and the head of the gastrocnemius and soleus muscles were all affected in our report. A high index of suspicion to obtain advanced imaging is warranted to evaluate the component positioning in a patient with a change from the baseline neurovascular status after TKA. After evaluation, a multidisciplinary approach with close communication between the orthopedic, nerve, and vascular specialists was vital to the success of management. Unfortunately, this patient developed a periprosthetic joint infection. Because she had reported chronic knee pain and sought continued periarticular steroid injections, it is possible that these injections, compounded with her longstanding diabetes, were the source of her infection [10].

Summary

We report a novel case of an exceedingly-rare TKA complication: a posterior DPI causing neurovascular compromise. Although the exact mechanism of injury is unknown, existing data and our case suggest a traumatic anterior force caused the dislocation of the spacer. A staged approach with peroneal nerve release and TKA

revision was planned with a multidisciplinary team of orthopedic, nerve, and vascular surgeons. The polyethylene insert was noted to be dislocated, rotated 90 degrees, and incarcerated in the posterior knee. Our carefully planned approach in this rare complication was instrumental in addressing our patient's neurovascular compromise and improving her functional status. Caution is recommended in the use of injection therapies around knee arthroplasty, which can lead to infectious complications.

Conflicts of interest

The authors declare the following financial interests/personal relationships which may be considered as potential competing interests: G. J. Golladay is the Editor in Chief of *Arthroplasty Today*; receives royalties from OrthoSensor (Stryker); receives research support from KCI and Cerus; receives royalties or financial or material support from AAHKS; is in the editorial or governing board of JOA; and is on the AAHKS Publications Committee and Virginia Orthopaedic Society Board. He was recused from the peer review and editorial decision on this manuscript. B. J. Chiang, J. A. Ross, and A. A. Jorgensen have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

For full disclosure statements refer to <https://doi.org/10.1016/j.artd.2022.01.001>.

Informed patient consent

Informed consent was obtained from the patient for this case report. The patient gives her consent for images or other clinical information relating to her case to be reported in a medical publication and understands that her name and initials will not be published and that efforts would be made to conceal her identity. She is in full understanding that the material may be published and may be seen by the general public. Request for the signed patient consent for publication is available upon request from the corresponding author Benjamin J. Chiang at chiangbj@vcu.edu.

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