Tibial Avascular Necrosis After Conversion From High Tibial Osteotomy to Total Knee Arthroplasty

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Proximal or high tibial osteotomy (HTO) is a well-established procedure for managing unicompartmental osteoarthritis of the knee.[1-3] Successful HTO may delay or prevent the need for eventual unicompartmental or total knee arthroplasty (TKA). However, clinical outcomes of HTO have been found to deteriorate because of progression of joint disease and pain, with arthroplasty required as a result.[4-7] Up to 40% of patients require conversion to TKA 10 to 15 years after HTO.[8]

In a study by Meding and colleagues,[9] 85% of patients who were converted from HTO to TKA were pain-free at a mean follow-up of 7.5 years. Other favorable clinical outcomes after TKA have been reported in patients who had previously undergone HTO.[10-12] However, conversion from HTO to TKA is technically demanding, and the outcomes reported in the literature, accordingly, have been mixed.[13-15] Complications associated with the procedure include patella baja,[13,15-17] tibial stem impingement,[10] decreased range of motion,[10,18] instability,[12] tibial polyethylene wear,[19] and tibial component subsidence.[19,20] In addition, conversion surgery involves longer operative times, difficulty in obtaining adequate exposure (often requiring extended soft-tissue releases),[13] a scarred posterior cruciate ligament (rendering the need for a posterior stabilized implant),[10] patellar maltracking,[10,21] difficulty in adequate ligament balancing,[18,22] neurovascular injury,[23,24] and bone defects requiring augmentation.[14,25] Some authors have compared this conversion surgery with revision TKA.[14,25]

Several investigators have studied the complications associated with conversion from HTO to TKA. A relatively underexamined complication is the potential for disruption of the blood supply to the proximal tibia. This vascular insult may result in avascular necrosis (AVN) of the proximal tibial bone stock. Parvizi and colleagues[26] reported radiolucent lines and a high rate of radiographic loosening on postoperative radiographs after TKA conversion from HTO, but they did not relate them to a vascular etiology.

We believe that some patients who undergo HTO and subsequent TKA develop AVN. Until now, this phenomenon has not been described in the literature.

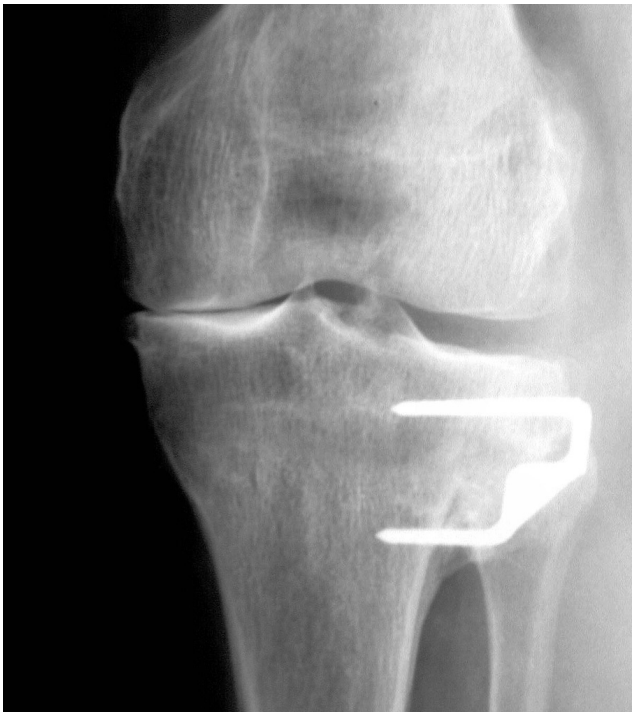

Figure 1. Case 1. Anteroposterior radiograph after closing-wedge high tibial osteotomy.

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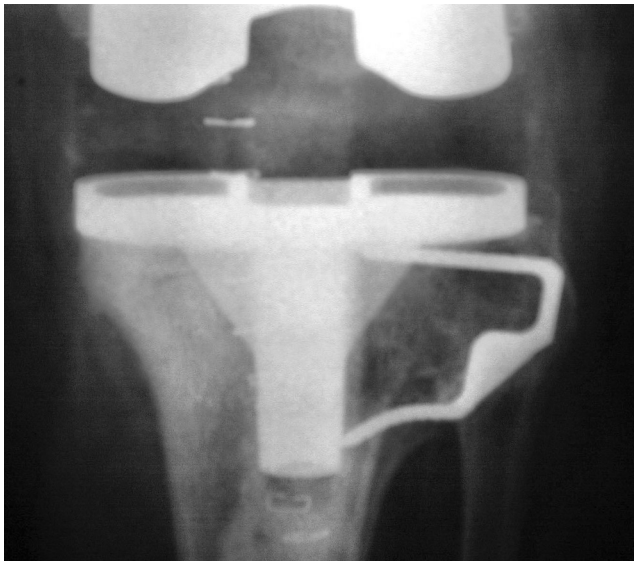

Figure 2. Case 1. Anteroposterior radiograph after total knee arthroplasty, with early avascular necrosis of lateral proximal tibial metaphysis.

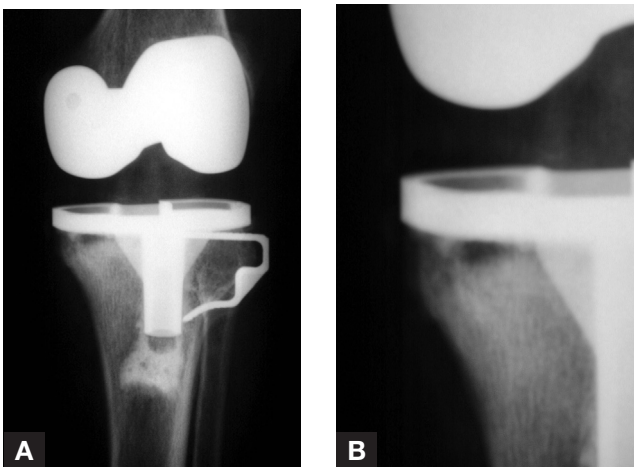

Figure 3. Case 1. (A) Anteroposterior radiograph shows radiolucency under medial tibial baseplate and interval progression of osteonecrotic lesion in lateral tibial proximal metaphysis. (B) Medial tibial radiolucent region, magnified.

In this article, we report 3 cases of AVN in the proximal tibia of TKA patients who had previously undergone HTO. The patients provided written informed consent for print and electronic publication of these case reports.

Case Reports

Case 1

In 1993, a 62-year-old man with no significant medical risk factors for AVN underwent closing-wedge HTO of the left tibia for isolated medial compartment osteoarthritis. His postoperative course was uncomplicated. Five years after surgery, he reported pain and crepitus with activities of daily living. Radiographs showed evidence of tricompartmental osteoarthritis without radiographic findings of AVN (Figure 1). Six years after the index HTO, he underwent TKA without complications (Figure 2).

Eight months after TKA, he reported isolated, progressively worsening pain along the proximal medial tibia. Radiographs showed a radiolucent zone with sclerosis underneath the medial tibial tray as well as a large area of radiolucency under the lateral tibial tray involving the prior osteotomy site (Figures 3A, 3B). Preoperative cultures, cell count, and serologic studies showed no evidence of infection. The patient underwent joint exploration. During surgery, a large osteonecrotic defect was noted beneath the medial tibial tray (Figure 4). As previously described,[27] the defect was filled with tobramycin-impregnated cement after being curetted down to viable bone (Figure 5). The tibial tray was well-fixed and thus was not revised. Intraoperative cultures and frozen and permanent sections were negative for infection, but histopathologic findings were suggestive of AVN (Figures 6A, 6B). The patient was last evaluated 9 years after revision arthroplasty. There were no radiographic or clinical signs of failure.

Case 2

A 43-year-old man underwent TKA 8 months after opening-wedge HTO using a medial-offset tibial stem. The patient did well for 7 months after TKA but then began to experience medial-side knee pain. Radiographs showed a medial tibial lucency. The preoperative index of suspicion, based on clinical scenario, inflammatory markers, and radiographic evaluation, was an aseptic bony defect after prior osteotomy. The plan was to address the limited bony defect with retention of prosthetic components.

During surgery, a necrotic defect was noted on the proximal medial tibial plateau between the site of the healed HTO and the tibial tray. Visual and manual inspection revealed that the components, including the bone–prosthesis interfaces, were well-fixed. This defect was filled with tobramycin-impregnated cement after being curetted down to viable bone. Reactive

Table. Patient Characteristics, Operative Time Line, and Development of Avascular Necrosis

Pt	Sex	HTO Type	HTO–TKA Interval	Age at TKA, y	AVN Location	TKA–AVN Diagnosis Interval
1	M	Closing wedge	6 y	50	Medial > lateral	8 mo
2	M	Opening wedge	8 mo	43	Medial	7 mo
3	F	Closing wedge	7 y	51	Medial	5 y

Abbreviations: AVN, avascular necrosis; HTO, high tibial osteotomy; TKA, total knee arthroplasty.

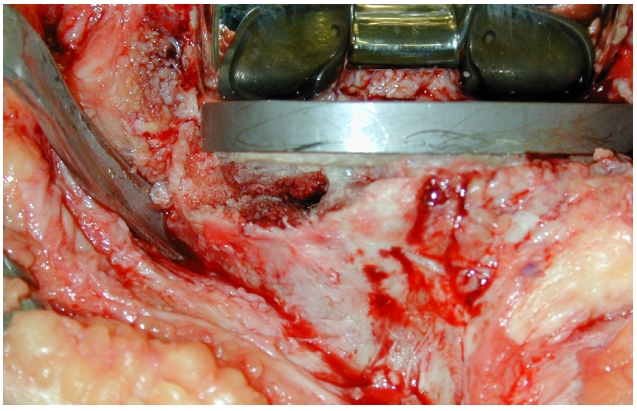

Figure 4. Case 1. Intraoperative exploration revealed medial tibial osteonecrotic lesion.

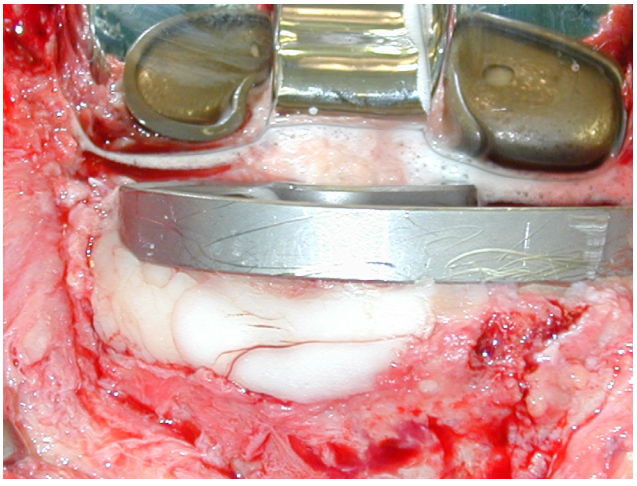

Figure 5. Case 1. Medial tibial lesion after curettage and cementation.

fibrodysplasia was seen on histology, confirming AVN of this metaphyseal segment. The polyethylene insert was replaced secondary to minor wear. The patient was doing well after revision TKA.

Case 3

A 57-year-old woman underwent left proximal closing-wedge tibial osteotomy for severe medial joint osteoarthritis. There were no significant medical risk factors for AVN. As tricompartmental arthritis was advancing 7 years after the index tibial osteotomy, left TKA was performed.

Five years after TKA, the patient returned with a progressively painful left knee and instability. Radiographs showed radiolucency beneath the medial tibial baseplate. During surgery, the femoral and tibial components were stable, and an osteonecrotic defect was noted beneath the medial tibial plateau, between the tibial component and the healed HTO. The defect was curetted back to viable bone, and the void was filled with tobramycin-impregnated cement. Microscopic tissue examination showed AVN. After revision arthroplasty, the patient felt relief from her symptoms and was functioning well.

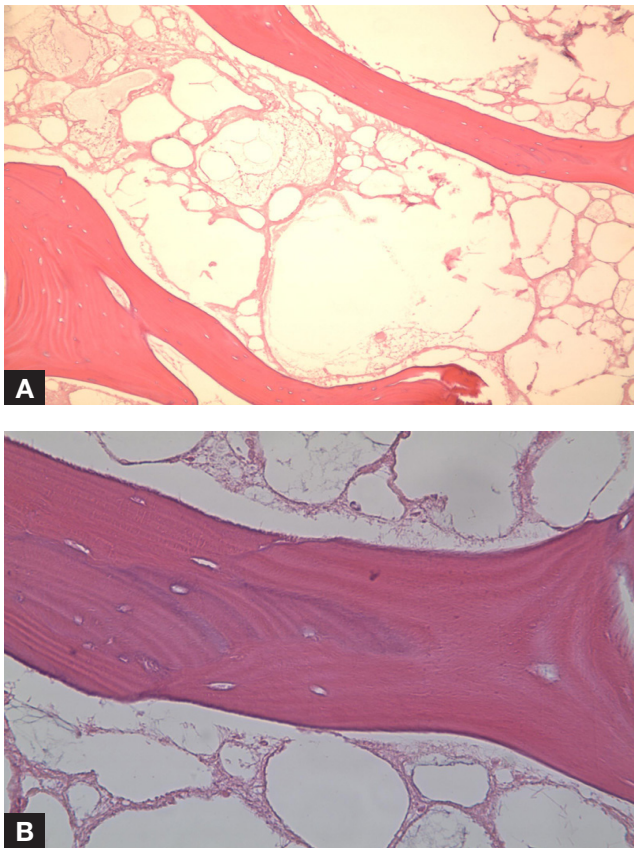

Figure 6. Case 1. (A) Histopathologic analysis of lesion revealed empty lacunae (no osteocytes visible within lacunae) and fatty necrosis, which, with usual lack of inflammatory response in osteolysis, confirmed histologic diagnosis of avascular necrosis (hematoxylin-eosin, original magnification ×10). (B) Histopathologic analysis revealed avascular necrosis of lesion (hematoxylin-eosin, original magnification ×40).

Discussion

Spontaneous or primary osteonecrosis of the knee was first described by Ahlbäck and colleagues[28] (1968) as involving the medial femoral condyle, and then by D'Anglejan and colleagues[29] (1976) as involving the medial tibial plateau. Ecker and Lotke[30] presented the first large series of cases of medial tibial plateau AVN, with the classic radiographic lesion seen as a radiolucent area surrounded by a sclerotic margin. Secondary osteonecrosis of the knee or osteonecrosis with iatrogenic causes has been sporadically described in the literature. Although there have been reports of osteonecrosis of the proximal tibia after tibial osteotomy[31] and knee arthroscopy,[32,33] osteonecrosis of the tibial plateau of a TKA after proximal tibial osteotomy has not been previously described.

TKA after HTO traditionally has had mixed outcomes.[9,15,21,34] Outcomes in our case series of 1 female and 2 male patients (Table) suggest that, besides there being well-known potential mechanical and technical complications, HTO–TKA conversion also may be associated with AVN of the proximal tibia. This rare complication may lead to implant failure requiring revision surgery.

It is unclear why this subset of patients developed AVN. Cellular toxicity, coagulopathy, fat emboli, elevated bone marrow pressures, and compromise of the tibial vascular supply—first at time of HTO and again during TKA—may all play a role. AVN has multiple known risk factors; patient factors, such as alcohol abuse, corticosteroid treatment, cytotoxic agents, and sickle cell disease, play a role in secondary causes.[30]

Although the exact etiologies are unclear in our patients' cases, we speculate that the tenuous vascular supply of the proximal tibial may be disrupted, secondary to soft-tissue or bony trauma in either an opening- or closing-wedge osteotomy, and then further compromised during conversion to TKA. The vascular supply to the proximal tibia is understood to be supplied by the metaphyseal, nutrient, and periosteal systems.[35-38] Cadaveric studies have found that 1 to 2 nutrient arteries originate a mean of 11.4 cm to 16.4 cm from the tibial plateau.[39] Approximately 50% of the nutrient supply is from the posterior tibial artery, and the remaining supply arises from the anterior tibial and popliteal arterial systems.[39]

A rich anastomosis surrounds the knee, and the blood supply to the proximal tibia has contributions from the lower portion of the geniculate anastomoses. Direct blood supply to the proximal tibia may derive from the medial and lateral inferior genicular arteries, the anterior and posterior tibial recurrent arteries, and the circumflex fibular arteries.[38,39] Although contributions from the geniculates exist, it is believed that, because of the point of entry, the nutrient artery does not supply the tibial metaphysis; the proximal extent of the nutrient artery branches end at the proximal tibial diaphysis or metadiaphyseal junction. The periosteal supply to the proximal tibial is almost entirely from the popliteal artery. No perioperative vascular mapping studies were performed in our cohort of patients.

Although most contemporary HTO procedures are performed through a standard midline incision, prior L-shaped incisions, with associated medial soft-tissue dissection, may play a part in disrupting the extraosseous blood supply to the proximal tibia. The possible blood-supply disruption that occurs during initial osteotomy and subsequent TKA is one plausible cause of induction of osteonecrosis in these patients. Another is application of polymethylmethacrylate (PMMA) bone cement to an already devascularized region of bone during TKA. Study results suggest that PMMA particles may affect osteoblastic function and contribute to osteolysis around the prosthesis, eventually resulting in prosthetic failure.[40] Other possibilities are closely spaced tibial bone cuts, excessive temperatures from tibial saw cuts and cementing, and increased pressure beneath the tibial tray leading to elevated marrow pressure.

The initial HTO procedure also may alter both local and regional blood supply, compartment pressures,[41] and venous outflow.[42] In addition, nonunion is an uncommon but potential complication of the index HTO,[43-45] with vascular insult a possible etiology. Although HTO may alleviate joint contact forces and improve cartilage biology on the articular surfaces,[46-50] the potential compromise of subchondral and metaphyseal bony blood supply remains to be further elucidated.

We have presented 3 cases of AVN of the tibial plateau in patients who underwent TKA after prior HTO. Although the exact etiology is unclear, vascular insult is likely. The incidence of AVN after conversion of HTO to TKA is unknown, but this series illustrates that AVN is indeed a risk after this surgery. HTO remains an important surgical alternative to arthroplasty for young, active patients with isolated single-compartment osteoarthritis, but the potential for AVN of the proximal tibia after conversion to TKA must be recognized as a potential complication that may compromise clinical outcomes.

Authors' Disclosure Statement

The authors report no actual or potential conflict of interest in relation to this article.

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