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Case Report

Intraosseous Tibial Resuscitation After a Total Knee Arthroplasty Leading to Osteonecrosis and Loosening of the Tibial Component

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ABSTRACT

A 51-year-old woman suffered cardiac arrest requiring emergent intraosseous access that abutted the tibial component of her total knee arthroplasty. She developed a wound at the site and knee pain which was concerning for deep infection. Subsequent imaging was consistent with osteonecrosis developing around the tibial component. The component eventually loosened, requiring a revision surgery. Her deep cultures remained negative throughout. Her findings are most consistent with osteonecrosis and aseptic loosening of her prosthesis. While intraosseous access may be beneficial during resuscitation, it has complications. This is the first reported case of osteonecrosis secondary to intraosseous access leading to prosthetic loosening necessitating a revision surgery.

Introduction

Intraosseous (IO) access is an effective method of providing rapid resuscitation where peripheral venous access is not easily obtained [1]. This procedure is reliable with a low rate of complications [1–5]. It has regained popularity recently, and there is a growing body of literature describing known complications and identifying novel ones [6–8]. The most common complication of IO access is extravasation leading to compartment syndrome, followed by osteomyelitis, cellulitis, and skin abscesses [2,5]. While a few cases of osteomyelitis after IO access have progressed to osteonecrosis (ON) [9–11], there are no incidents of isolated ON after IO access reported in the literature.

The prevalence of ON has been shown in a recent study to be 0.17% in patients older than 50 years [12], and the annual incidence worldwide is estimated to be 1.4-3.0 per 100,000 [13]. ON of the knee is commonly defined under 3 categories as spontaneous ON of the knee, secondary ON, and postarthroscopic ON [14]. Causes of ON are classified as traumatic or nontraumatic, with recent studies showing that a nontraumatic cause is almost always associated with metabolic causes such as medical conditions or medications [14]. We present a case of a patient who developed a traumatic bulla at her IO insertion site and subsequent ON below a total knee arthroplasty (TKA) leading to loosening and necessitating a revision surgery. The patient has given written consent for the publication of this case report.

Case history

A 51-year-old female with history of type II diabetes mellitus and severe asthma presented with cardiac arrest necessitating emergent bilateral tibial IO lines. One was placed adjacent to the tibial stem of a TKA performed 2 years earlier. The IO lines were removed the next day. During her admission, she became febrile, and there was a concern for systemic infection. Blood cultures were drawn, and she was started empirically on vancomycin and

Correspondence email: The patient has given consent for the publication of this case report.

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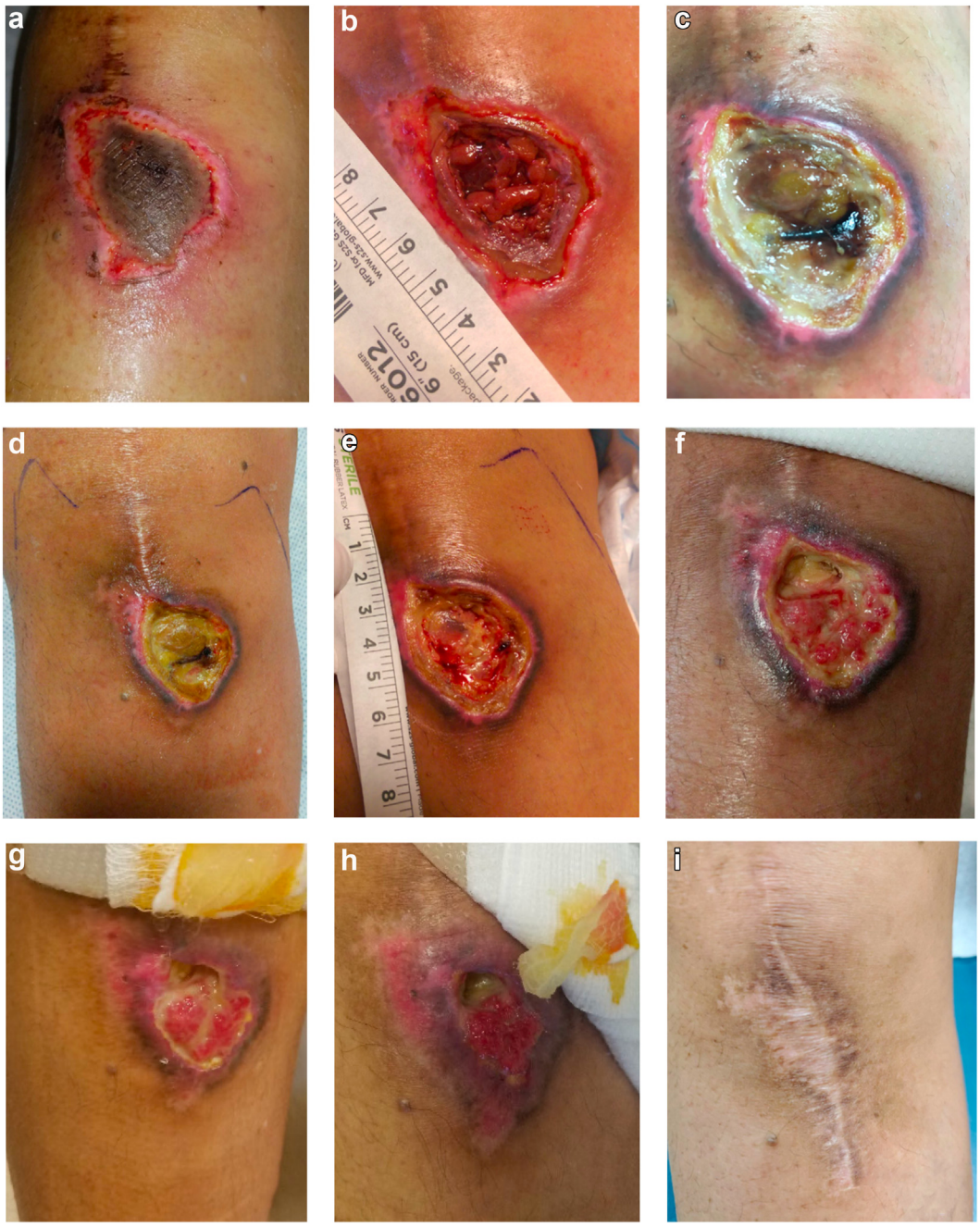

Figure 1. Wound progression over time. (a and b) During the initial hospitalization when the IO needle was placed. (c-e) During the second hospitalization during which the wound culture was positive for *Streptococcus agalactiae*. (f-h) At follow-up appointments with orthopedics prior to the operative debridement. (i) From a year and a half after the debridement and complex wound closure.

cefepime. Six days after admission, a blister consistent with a traumatic bulla was described, and 4 days later, the patient developed an open wound at the right IO access site (Fig. 1a and b). She then complained of right knee pain. Radiographs of her right TKA remained normal compared to radiographs taken 6 months after her primary TKA (Fig. 2). The knee was aspirated for concern of infection despite actively receiving antibiotics. The white cell count was 610 with 45% polymorphonuclear cells (PMNs), and cultures were negative. Erythrocyte sedimentation rate (ESR) was 56 mm/h (reference range 4-30), and C-reactive protein (CRP) was 81 mg/L (reference range <50); however, these elevations were attributed to her post-cardiac-arrest state without other evidence for a periprosthetic joint infection (PJI).

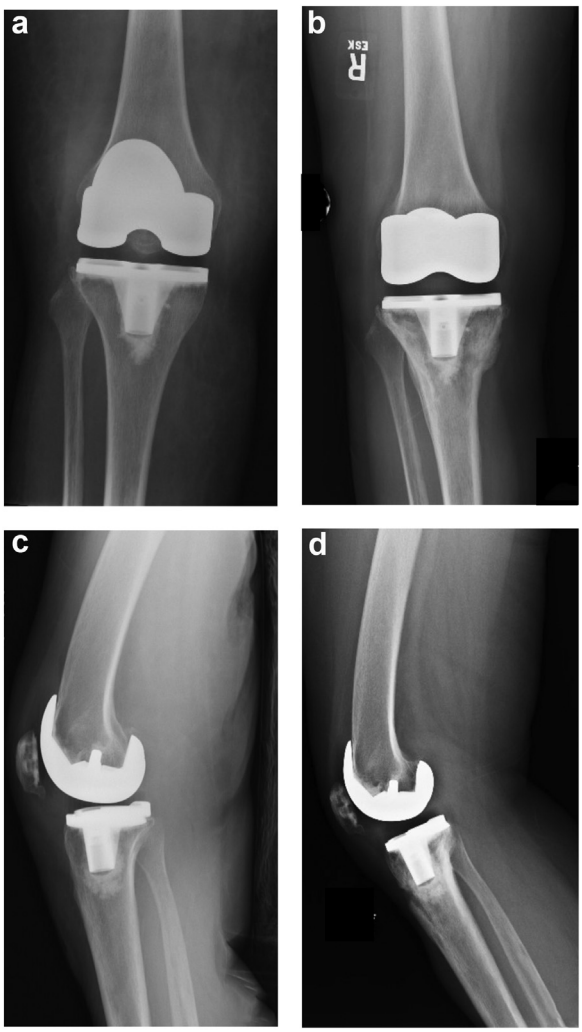

Figure 2. Anteroposterior (a and b) and lateral (c and d) radiographs with changes. (a and c) Images taken during the initial hospital stay. (b and d) Images taken a month after the patient's negative bone biopsy when she returned with excruciating pain, demonstrating evidence of loosening.

A week after discharge, her knee pain worsened, and she developed chills after finishing her antibiotic course. She presented to the emergency department and was admitted to observation for an elevated peripheral white blood cell count of 15.5 cells/mm^3. The wound care service was consulted for concerns of infection of the tibial wound with worsening surrounding cellulitis but no purulence (Fig. 1c-e). The wound was cultured prior to being started on vancomycin and cefepime. Radiographs were unchanged, and an arthrocentesis performed after initiating antibiotics showed a white cell count of 1510 with 42% PMNs, with the ESR and CRP down to 54 mm/h (reference range 4-30) and 40 mg/L (reference range <50), respectively. The tibial wound cultures grew *Streptococcus agalactiae*, and intra-articular knee cultures remained negative. She was transitioned to ampicillin/sulbactam, which was then switched to cefazolin after an adverse reaction. Computed tomography of the knee was performed and showed an effusion with edema and fat stranding near her tibial wound site. The path of the IO needle was identified abutting the tibial keel (Fig. 3). A subsequent metal artifact reduction sequence magnetic resonance imaging (MRI) demonstrated heterogenic T1 intensity in the proximal tibia with questionable hardware involvement (Fig. 4). She was treated empirically with ciprofloxacin and vancomycin, and she was discharged home.

Two weeks after discharge, the patient stopped taking her antibiotics due to abdominal pain. She was switched to daptomycin, but continuing gastrointestinal issues caused her to stop after 4 days. She was referred to the orthopedic clinic for evaluation of her tibial wound and knee pain (Fig. 1f and g). Her knee was aspirated once again 8 days after the last antibiotic dose and remained negative for infection. Her white cell count was 1128 with 44% PMNs. Nine days later, drainage was noted at the site of the tibial wound (Fig. 1h).

The patient was taken to the operating room for an exploration of the wound. Intraoperatively, the infection was localized to the subcutaneous tissue with no tract to the bone and no communication with the prosthesis. The wound was then closed by plastic

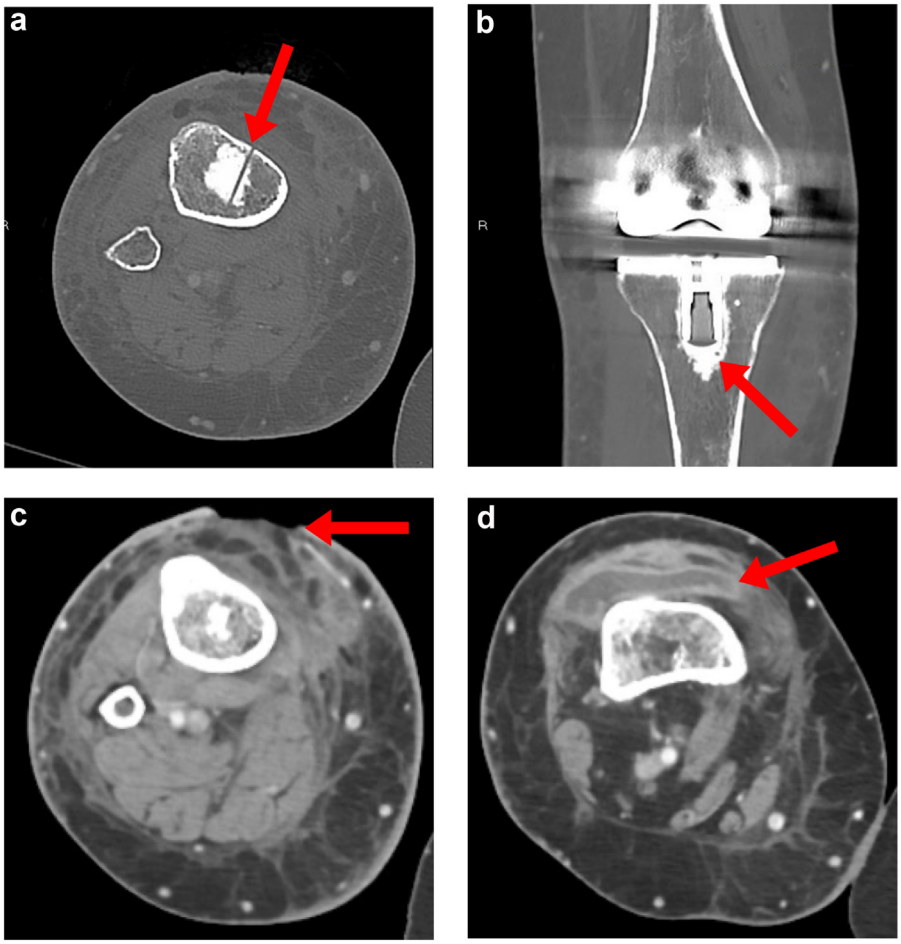

Figure 3. Computed tomography scan. (a) An axial image of the proximal tibia demonstrating a cortical breach (arrow) that is further shown in (b), with the coronal image also demonstrating the proximity of the needle tract to the tibial stem and cement (arrow). (c) The pretibial defect (arrow) with adjacent cellulitis, myositis, and fasciitis. (d) Suprapatellar effusion and synovial thickening with enhancement (arrow).

surgery. Intraoperative tissue cultures grew *S. agalactiae* and methicillin-sensitive *Staphylococcus aureus*. The soft tissue infection was treated with amoxicillin and clavulanate. Her tibial wound closure healed uneventfully.

She remained free of knee pain for a year and a half, during which time she was on corticosteroids and immunomodulating therapy for various periods of time due to severe asthma. She then returned to the clinic for right knee pain after a ground level fall onto that knee. Radiographs were unchanged. At this time, her wound had healed completely (Fig. 1i). Her ESR was slightly elevated at 52 mm/h (reference 0-40), and CRP was at the upper limit of normal at 10 mg/L (reference range 0-10). As she was taking antibiotics for a respiratory infection at the time, her knee aspiration was delayed. When she returned 2 weeks after stopping the antibiotics, an aspiration was sent for culture, finding no growth. Given her history, a metal artifact reduction sequence MRI was ordered to assess for possible osteomyelitis. This demonstrated extensive edema with superimposed ON around the tibial component (Fig. 5). A bone biopsy was ordered for concerns of deep osteomyelitis, but it was deferred due to the COVID-19 pandemic. During this time without treatment, her symptoms improved, and her inflammatory markers normalized. A percutaneous biopsy and culture were eventually performed 3 months later, which showed no evidence of active inflammation on pathology and negative cultures (Fig. 6).

Two months later, she returned with worsening pain. Radiographs now demonstrated lucency under the tibial baseplate and varus subsidence (Fig. 2c and f). A revision was planned. Ongoing cardiac issues delayed surgery for 8 months. At the time of the revision surgery, the tibial component was loose with extensive ON of the metaphysis without gross purulence. Given the concern for underlying septic loosening, a 2-stage revision with aggressive debridement and placement of an antibiotic spacer was performed (Fig. 7a and c). Pathology noted chronic bone inflammation, and cultures were negative from frozen sections sampled from the medial and lateral gutters and the medullary canal intraoperatively (Fig. 8a and b). Postoperatively, she received vancomycin and ceftriaxone empirically for 6 weeks. Three months later, her inflammatory markers were normal, she had no symptoms of active infection, and a second-stage reimplantation was completed (Fig. 7b and d). Surgical pathology and cultures were negative for any organism (Fig. 8c).

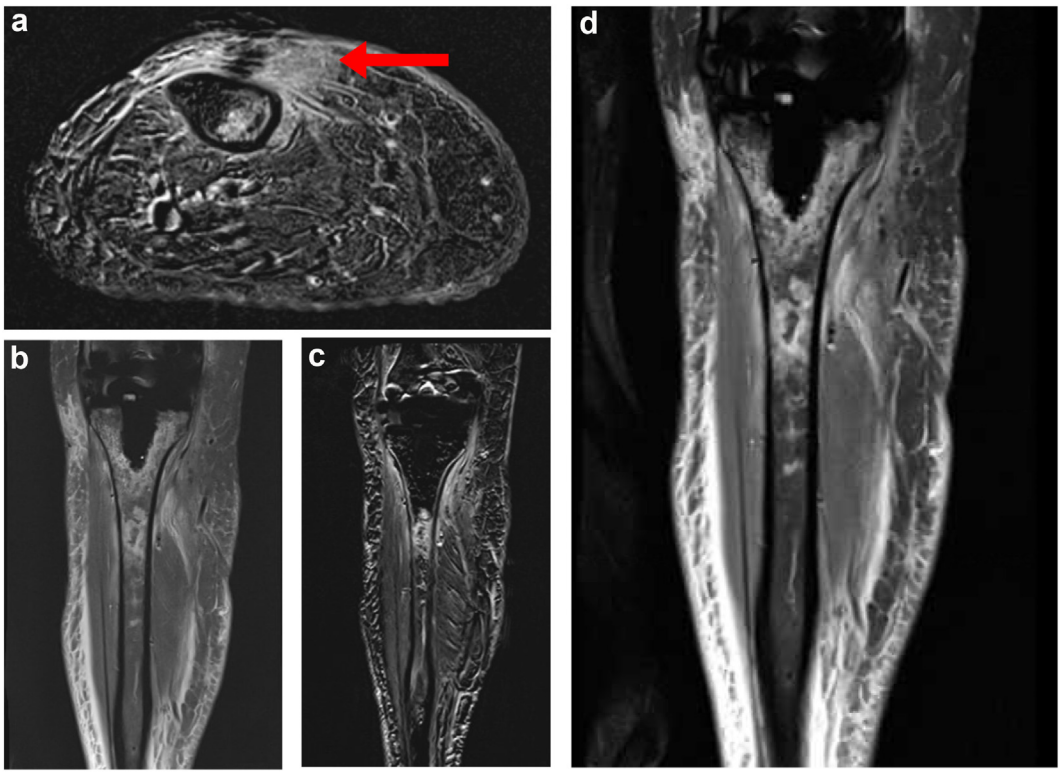

Figure 4. First metal artifact reduction sequence MRI scan with contrast that was suspicious for osteomyelitis. (a) A postcontrast axial T1 Total imaging matrix image demonstrating the pretibial defect with surrounding abnormal soft tissue signals associated with cellulitis, myositis, and fasciitis (arrow). (b) A coronal Short-T1 inversion Rshowing hyperintensity in the proximal and mid-tibia regions with questionable hardware involvement. (c) A postcontrast coronal T1 image demonstrating heterogenic intensity in the proximal and mid-tibia regions. (d) A coronal T1 Turbo inversion recovery magnitude sequence image with high bandwidth further demonstrating the abnormal hyperintensity in the proximal and mid-tibia regions surrounding the tibial stem of the TKA.

After the second stage of the revision, the patient had a normal recovery. At 6 months after revision, she felt that her pain was well controlled and was happy with the results. Her physical examination demonstrated full knee extension to 110 degrees of flexion without pain and no signs of loosening or instability. At the time of publication, she has recovered without any complications.

Discussion

IO access provides a rapid, reliable method for vascular access during emergencies. The ideal location for insertion is the proximal tibia, as it has a combination of flat and thin cortical bone with a thin layer of tissue with sparse neurovascular structures [1,2]. IO access combines a high rate of successful placement with a low rate of complications at less than 1% [1]. The most common complication is compartment syndrome, followed by osteomyelitis, cellulitis, and skin abscesses [2,5]. A novel complication recently reported is the formation of traumatic bullae [6]. Our case describes a second occurrence of this complication, along with progression to ON of the proximal tibia and subsequent aseptic loosening of the tibial component requiring revision arthroplasty. Currently, there are no case reports detailing isolated ON as a complication after the use of IO resuscitation.

Although the etiology of ON is not fully understood, 1 mechanism involves impaired blood supply due to traumatic disruption or intravascular/extravascular occlusion, leading to cell death [14]. Implicated as an extravascular cause many years ago, increased IO pressures have been measured in ON [14–18]. For the diagnosis of ON, MRI is the most used modality. Necrotic tissue will appear hypointense on both T1- and T2-weighted images, while the hypervascular granulation tissue will have low signal on T1-weighted images with higher signal on T2-weighted images [19,20]. Similar findings were seen in our case. Although secondary and spontaneous ON of the knee are well recognized, there is no known literature showing ON of the knee after a primary TKA or after IO resuscitation.

Due to the patient's extended antimicrobial treatments throughout her clinical course, 1 possible explanation for her ON is culture-negative (CN) PJI. The frequency of CN PJI is most likely around 7%-15% of cases, but there have been reports of incidences up to 35% [21–23]. Possible causes of CN PJI include prior antimicrobial therapy hindering the ability to isolate an organism, presence of an unrecognized cause of PJI, or that it may not truly be infectious [23]. CN PJI tends to have a delayed onset, with only 15% occurring in the first 3 months after arthroplasty and a median time to diagnosis of 3 and a half years shown in a large study [24,25]. We tested her multiple times during her treatment, and our assumption was she had no infection. Additionally, aspirations and intraoperative findings at multiple points in time also failed to demonstrate the presence of osteomyelitis or other infectious causes.

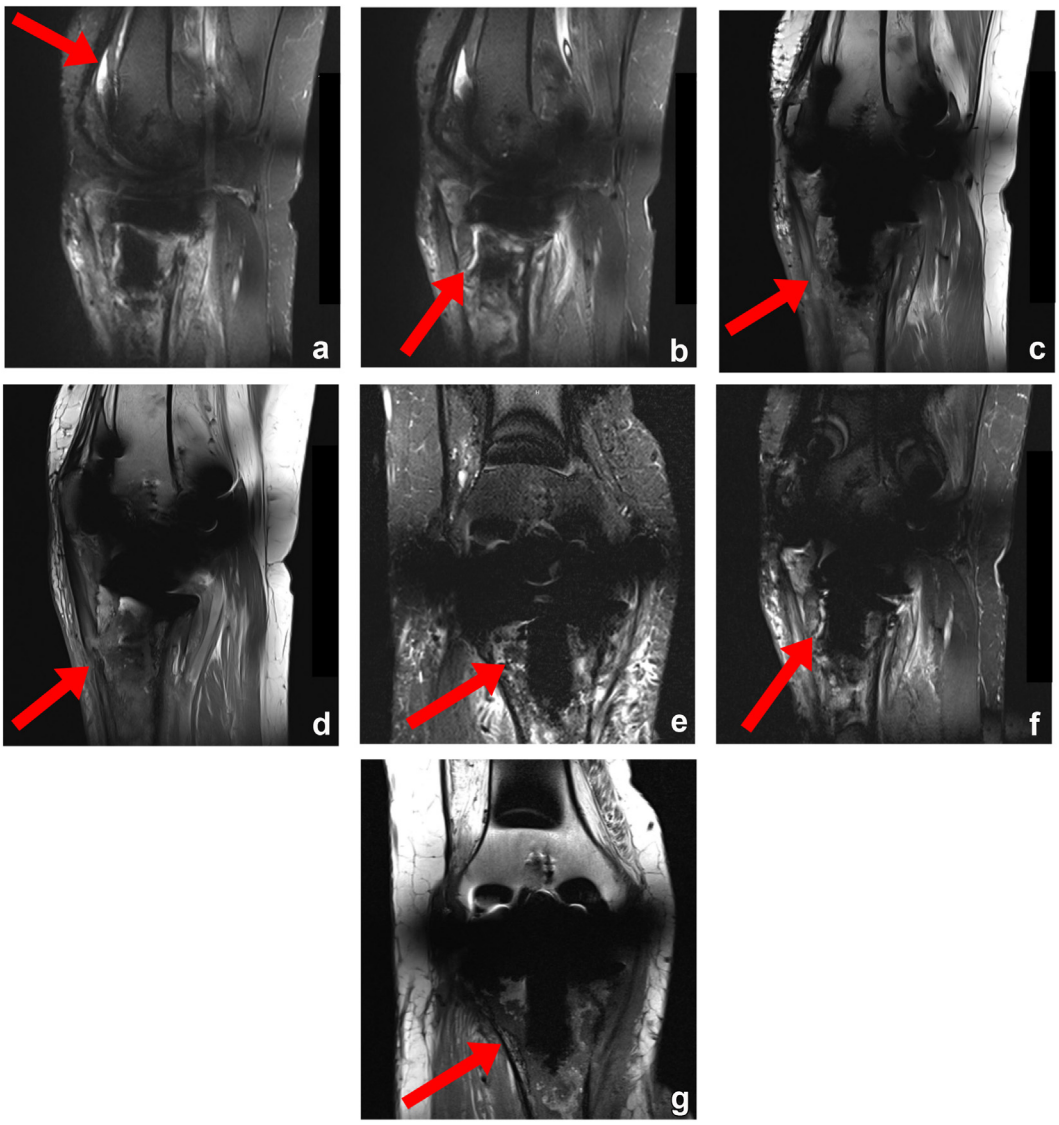

Figure 5. Repeat metal artifact reduction sequence MRI scan without contrast 1 and a half years after the first one. (a and b) Sagittal T1 turbo inversion recovery magnitude sequence images demonstrating abnormal bone marrow signal in the proximal tibia with a fluid collection in the retropatellar soft tissue and bursa (arrows in a) and the double-line sign pathognomonic for osteonecrosis (arrow in b). (c and d) Sagittal proton density–weighted images with high bandwidth showing cortical breakthrough involving the proximal tibia at the level of the tibial tuberosity (arrows). (e [coronal] and f [sagittal]) Proton density–weighted short-T1 inversion recovery sequence images with high bandwidth demonstrating abnormal bone marrow signal in the proximal tibia with hyperintense periprosthetic cysts in (e) (arrow) and the double-line sign in (f) (arrow). (g) A coronal T1 with high bandwidth image demonstrating abnormal bone marrow signal in the proximal tibia with hypointense periprosthetic cysts (arrow).

We hypothesize that the rapid resuscitation with IO fluids placed beneath the tibial component led to increased IO pressures, and the resulting impairment in blood supply eventually led to ON and collapse with aseptic loosening of the tibial component. As this has never been described previously and she was noted to have a superficial infection initially, the suspicion remained for a septic etiology of her knee failure. However, a number of knee aspirates were performed, and no causative organism was ever found. In addition, all soft tissue cultures performed at the time of the first and second stages of revision were negative for infection. Although CN PJI remains a possibility, we do not suspect this given the combination of serology, knee aspirates, and intraoperative findings.

This case describes a complication of IO line placement and is the first to identify extensive ON under a knee arthroplasty. As the patient was asymptomatic and her knee was functioning well prior to the IO resuscitation, we suspect subsequent ON and collapse likely underlies the aseptic failure of her knee arthroplasty. IO lines

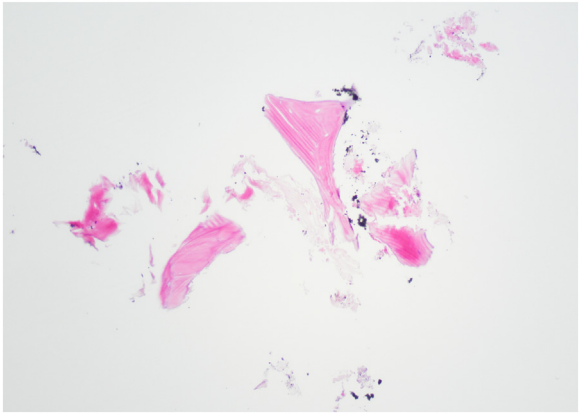

Figure 6. Bone biopsy demonstrates scant fragments of bone and dense fibrous tissue without evidence of inflammation. The presence of black pigmented material is artifactual.

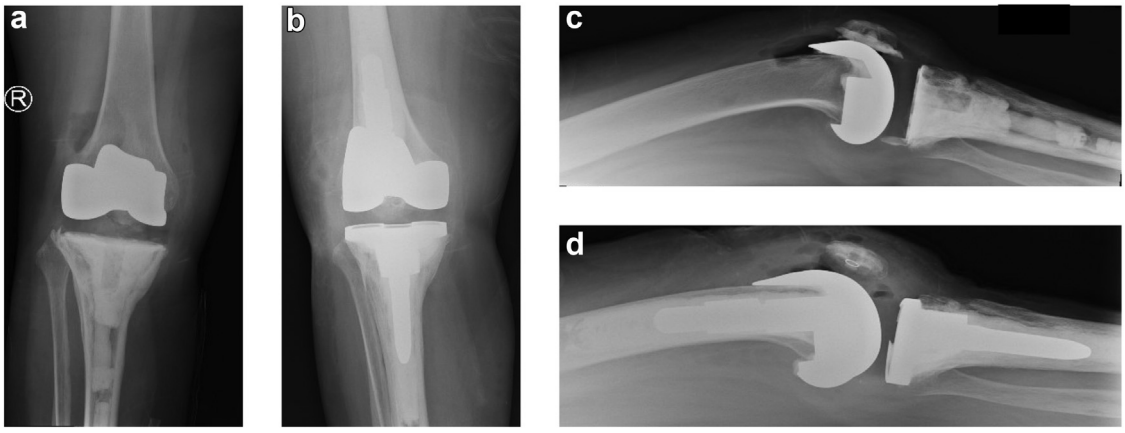

Figure 7. Anteroposterior (a and b) and lateral (c and d) radiographs from revision surgeries. (a and c) From the first stage. (b and d) From the second stage.

are placed in emergent situations, and the location of placement may be dictated by a number of factors that preclude identifying the ideal location. However, if possible, avoiding placing an IO line below a knee arthroplasty or any orthopedic hardware is advisable to avoid this novel complication and that of PJI.

Summary

While IO access provides many benefits during emergent resuscitation, it is not without complications. This is the first reported case of ON affecting the implanted hardware after IO access. Based on our experience, we recommend avoiding IO access in limbs that have hardware; if that is not possible, the next best recommendation is to attempt the IO access away from the implanted hardware in a way that does not compromise patient safety or delay necessary care.

Conflict of interest

Dr. H. S. Khanuja is a paid consultant for Smith & Nephew, has stock or stock options with Sight Medical, is in the editorial or governing board of the *Journal of Arthroplasty*, and is a board member of American Association of Hip and Knee Surgeons. All other authors declare no potential conflicts of interest.

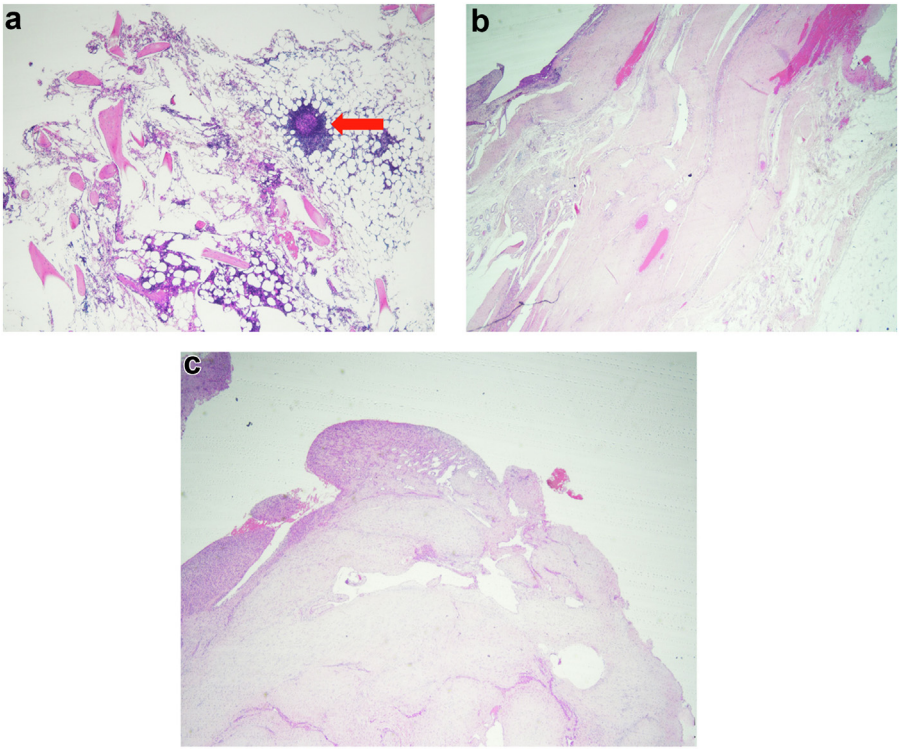

Figure 8. Surgical pathology results. The first stage of the revision (a and b) demonstrates cellular marrow (a) with expected trilineage hematopoiesis and benign lymphoid aggregates. The presence of a small granuloma (arrow) is idiopathic, and further workup with acid-fast Bacilli smear and Grocott's methenamine silver stains was conducted to rule out infectious etiologies. Joint histology (b) demonstrates chronic inflammation with hypertrophied synovium. The second stage of the revision was completed 3 months later (c). Excised tissue reveals unremarkable synovium with rare neutrophils but no significant inflammation.

For full disclosure statements refer to https://doi.org/10.1016/j.artd.2022.101088.

Informed patient consent

The author(s) confirm that informed consent has been obtained from the involved patient(s) or if appropriate from the parent, guardian, power of attorney of the involved patient(s); and, they have given approval for this information to be published in this article.

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