Case Report

Osteolysis and Wear Debris After Total Knee Arthroplasty Presenting With Extra-Articular Metallosis in the Calf

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Abstract: Component wear after total knee arthroplasty (TKA) with extruded metallosis in the extra-articular tissue of the calf secondary to a periprosthetic fracture is a rare complication. A 77-year-old man with a failed Insall-Burstein II TKA prosthesis presented with calf cellulitis after a fall. Radiologic evaluation revealed severe osteolysis and loosening of prosthetic components and an intramuscular abscess communicating with the medullary canal of the tibia through an undisplaced periprosthetic fracture. The patient developed rhabdomyolysis with acute renal failure. Drainage of the calf abscess showed staining of the muscles with wear debris and metallosis. The patient subsequently had debridement and excision of the infected TKA implant. Prompt diagnosis of this condition should be suspected in cases of failed arthroplasty with osteolysis and periprosthetic fracture. **Key words:** metallosis, osteolysis, rhabdomyolysis, periprosthetic fracture, total knee arthroplasty.

Component wear failure with polyethylene and metal debris is a recognized complication in joint arthroplasty. However, there are limited reports and studies on the immediate and long-term effects of wear debris and metallosis on host tissues, especially extra-articular metallosis. Atypical presentation of metallosis presenting as masses around the joint may take the unsuspecting clinician by surprise, as delayed diagnosis may result in dire consequences to the patient. Failure of total knee arthroplasty (TKA) components presenting as thigh and calf cystic masses [1,2], popliteal cyst [3], bursitis [4], granuloma [5], and abscess [6] has been reported. To the authors' knowledge, no report of a calf swelling and complications caused by extra-articular extruded metallosis from a periprosthetic fracture after failed TKA has been made.

Case Report

A 77-year-old Asian Malay man had a TKA with Insall-Burstein II constrained condylar knee system prostheses (Zimmer, Warsaw, Ind) with stem extensions and tibial augment done 9 years ago at another hospital. Records from his previous surgery showed that he had synovial chondromatosis of his left knee with osteoarthritis and varus deformity. There was bone loss and erosion in the anteromedial corner of the medial tibial plateau, which was built up with bone graft and screws during the primary

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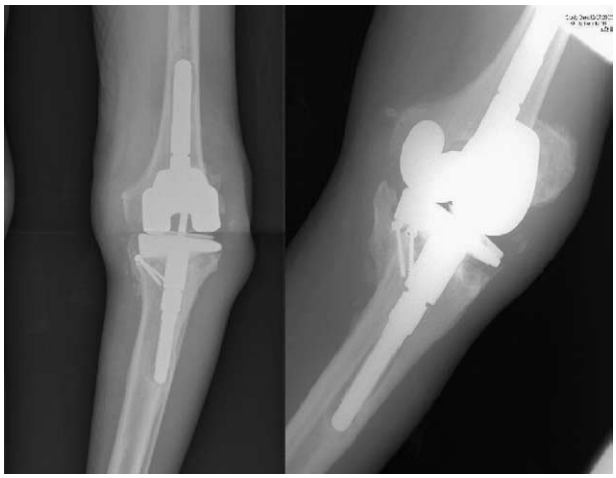

Fig. 1. Radiographs of the prosthesis 8 years after primary TKA.

arthroplasty 9 year ago. He had defaulted follow-up after surgery and now presented with a swelling of his knee and calf after a fall. Clinical examination revealed a warm generalized effusion of his left knee with decreased range of movement secondary to tenderness. There was also cellulitis and mild swelling of his calf. Erythrocyte sedimentation rate was 103 mm/h, C-reactive protein level was 97 mg/L, and total white blood cell count was 18.1×10^9/L (with neutrophil differential of 90.7%). Radiographs showed severe osteolysis and loosening of both the tibial and femoral cemented long-stem implants. The tip of the long-stemmed tibial implant was abutting against the tibial cortex (Fig. 1). Ultrasound duplex scan of the limb did not reveal any evidence of deep venous thrombosis.

The patient had fever secondary to beta-hemolytic group G *Streptococcus* bacteremia and gave a history of having recently recovered from an upper respiratory traction infection with sore throat. Aspiration of his knee revealed 60 mL of ink-colored fluid with a metallic tinge. The fluid white blood cell count was 4600/mm^3 with a neutrophil differential of 75% and protein levels of 35 g/L. Gram stain, bacterial and fungal cultures, and cytologic analysis of the fluid yielded negative results. The patient was given intravenous penicillin and cloxacillin. The initial working diagnosis was cellulitis of the calf with a background of osteolysis and component wear failure (metallosis) of the TKA. However, septic infection of the arthroplasty implants cannot be excluded at this stage.

Five days later, the patient developed increased erythema, tenderness and swelling of his left calf, and rhabdomyolysis with resultant acute renal failure. Serum creatine kinase and creatinine levels were both elevated with deranged liver function tests and decreased urine output. He required short-term hemodialysis support.

A computed tomographic scan of the left calf revealed a 24 × 4-cm intramuscular abscess communicating with the medullary canal of the tibial shaft and the lower end of the tibial prosthesis through a small periprosthetic fracture (Fig. 2). An emergency operation to drain the calf abscess was done. At surgery, there was a 25 × 5-cm abscess in

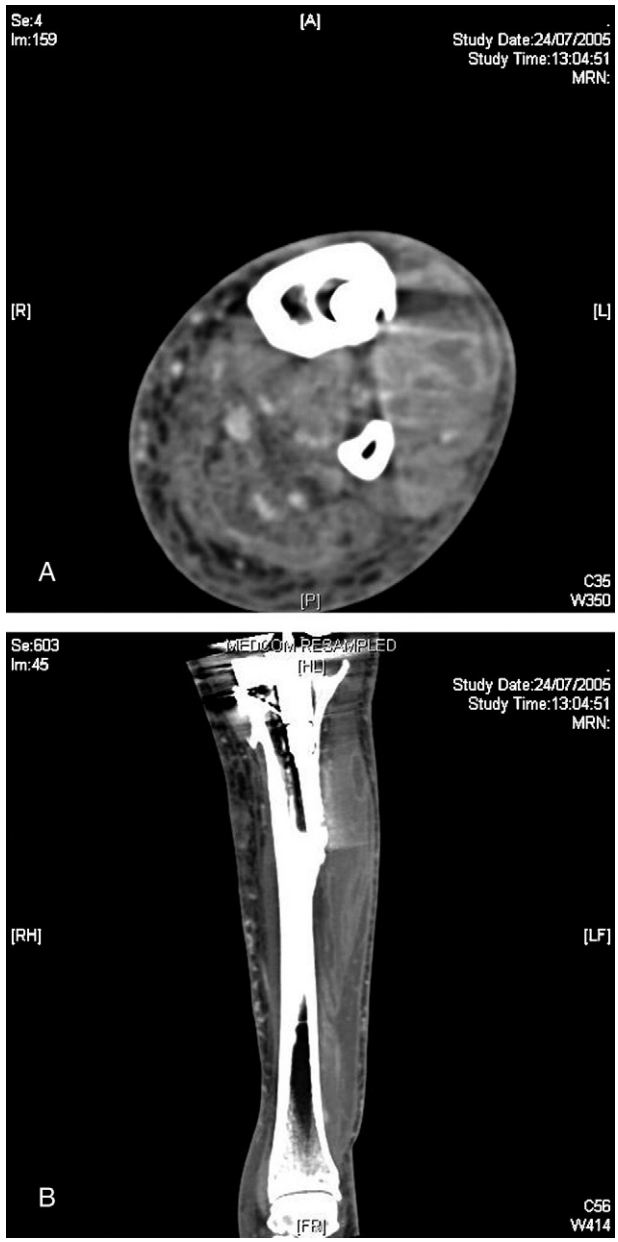

Fig. 2. Computed tomographic scan of the calf showing communication of the intramuscular abscess with the intramedullary canal of the tibia through an undisplaced periprosthetic fracture. (A) Coronal view. (B) Sagittal view.

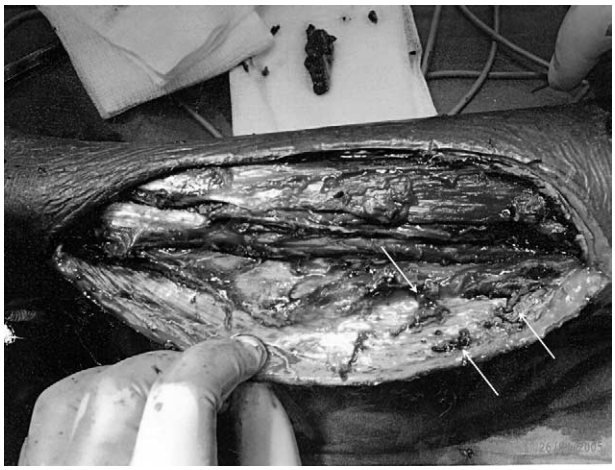

Fig. 3. Photograph of the intramuscular calf abscess during surgery showing staining of the muscle compartment with metallosis debris (arrows).

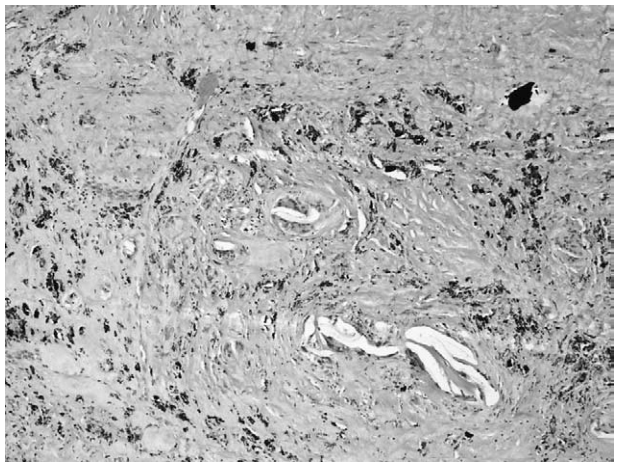

Fig. 5. Synovial biopsy specimen (hematoxylin-eosin stain ×10 magnification) showing foreign body–type multinucleated giant cells engulfing metallic particles and surrounding inflammatory infiltrates.

the anterior compartment of the calf, which was extensively stained with metallosis (Fig. 3). The tissue culture did not yield any positive growth. Histologic examination of the left calf tissue showed fibrous granulation tissue with both acute and chronic inflammatory cells and macrophages infiltrating in between muscle fibers (Fig. 4).

Postoperatively, the patient continued to require fluid resuscitation and support for his acute renal failure. His renal failure subsequently improved, and secondary suture was done for the calf wound. He was eventually well enough to undergo a definitive operation of debridement, synovectomy, and removal of the TKA implant.

At operation, there was extensive dark staining of the synovium with surrounding metallic debris and surrounding granulation tissue. Scanty turbid pus-like deposit was found in the parapatellar gutters. There was hypertrophy of the synovium and loosening of the femoral and tibial components as well as severe wear of the polyethylene insert. The implants were removed, and a Simplex P (Stryker, Mahwah, NJ) with tobramycin antibiotic cement spacer was inserted.

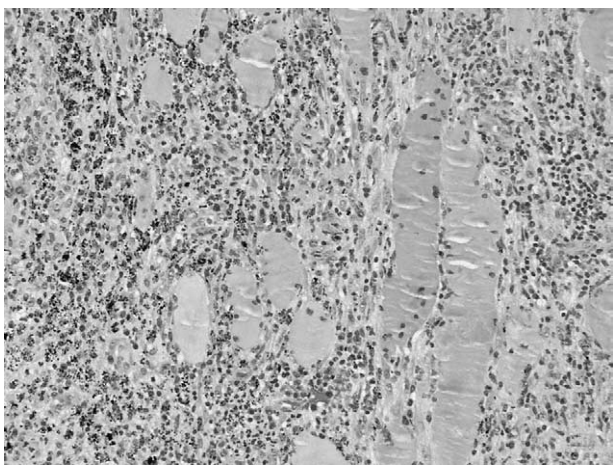

Fig. 4. Histologic specimen taken from intramuscular calf abscess ×20 magnification, hematoxylin-eosin stain showing acute and chronic inflammatory cells infiltrating the skeletal muscles. Wear debris are seen as black pigments.

The synovial fluid and tissue cultures were negative; histologic examination of the synovium showed large numbers of foreign body giant cells engulfing a black pigment substance and birefringent strandlike particles on polarized light. There was thickened fibrous tissue with fibrin and acute inflammatory cells on the surface (Fig. 5).

The patient was able to ambulate full weight bearing in a locked knee brace with a walking stick at 2 months postoperatively. He completed 6 weeks of intravenous antibiotics. His surgical wounds had healed, and he is currently being planned for a staged revision arthroplasty at a later date.

Discussion

There is paucity in the English literature describing the local and systemic effects of extruded extra-articular metallosis after TKA failure with osteolysis and particulate wear debris. Detrimental effects of metallosis such as metallic poisoning [7], metal hypersensitivity [8], risk of carcinogenesis [9-11], granuloma formation [5], and popliteal cysts [3] have been described. To the authors' knowledge, no previous report of a calf swelling caused by extruded metallosis from periprosthetic fracture of a failed TKA has been made.

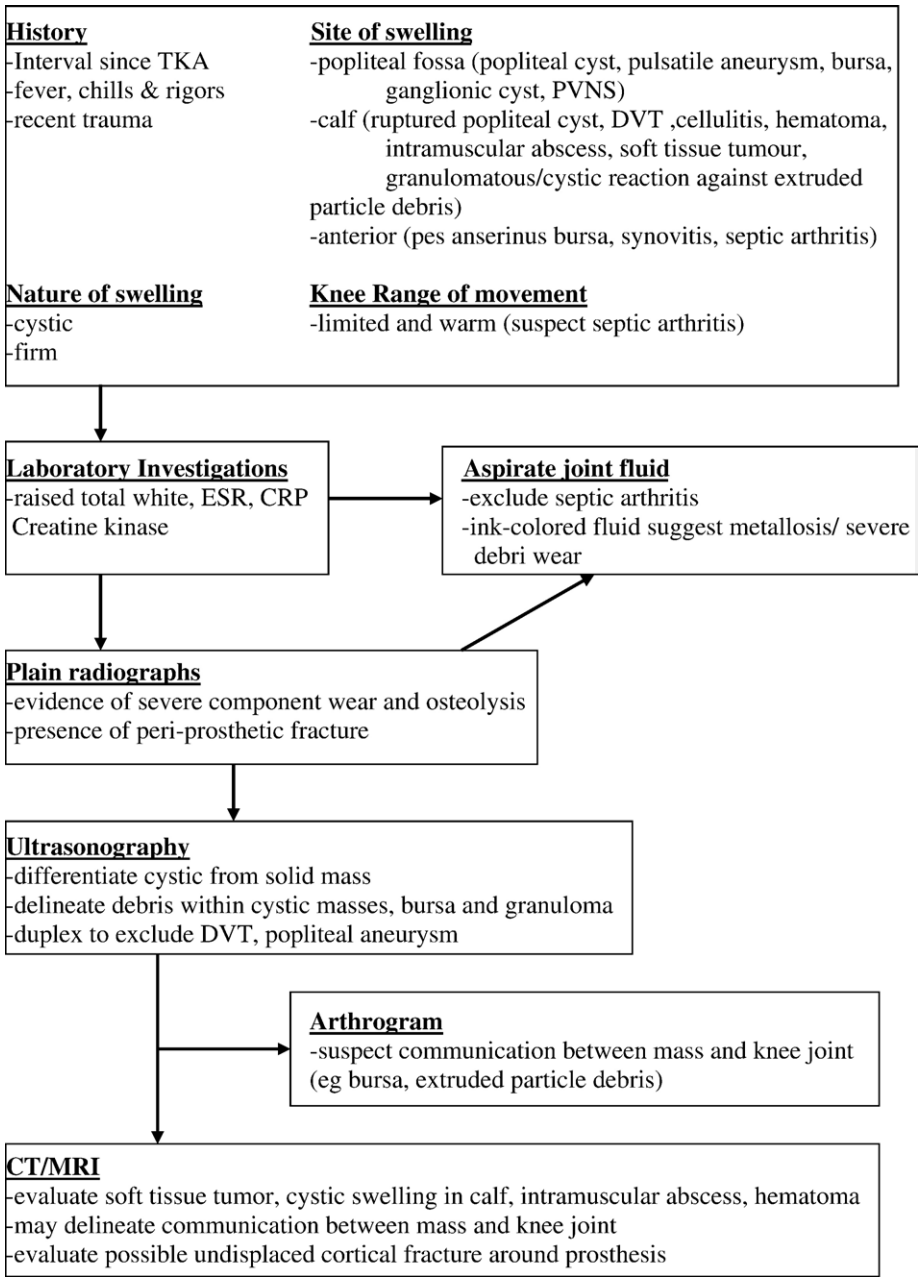

Fig. 6. Clinical algorithm for evaluation of swelling around the knee and calf in patients with TKA. DVT indicates deep venous thrombosis; ESR, erythrocyte sedimentation rate; CRP, C-reactive protein; PVNS, pigmented villo-nodular synovitis.

As the number of total joint arthroplasty increases in our local population and with the increase in life expectancy, there will be a sizeable number of patients suffering from neglected osteolysis and wear of arthroplasty components who are lost to regular follow-up. A patient with severe osteolysis of TKA presenting with swelling in and around the knee should be treated with caution and investigated promptly.

The severe osteolysis and loosening of the long-stem femoral and tibial prostheses in our patient predisposed the tibial stem to lever against the relatively osteopenic bone, causing an undisplaced periprosthetic fracture during low-energy trauma. The preexisting metallosis and debris in the knee joint extruded into the calf and incited a local inflammatory response. Case et al [12] found that wear particles from prostheses caused necrosis, fibrosis, and structural changes in regional lymph nodes, liver, and spleen and that the concentration of these particles is higher especially if the prosthesis is worn or loose. Wear particles, namely, metal,

high-density polyethylene and polymethylmethacrylate, stimulates a reaction that leads to necrosis of the surrounding tissues. In addition, polyethylene and polymethylmethacrylate particles are known to stimulate the production of multinucleated giant cells [13]. Previous authors have also reported elevated metal levels in urine and serum from loosened prostheses after joint arthroplasties [14-16]. Particulate metal debris can affect the immune system by inhibition of T-cell activation and increasing the numbers of T- and B-activated lymphocytes [17].

As summarized by Bullough [18], there are 3 effects through which wear particles from articular implants can exert on the body. Firstly, the fragmentation of the particles into smaller fragments increases the surface area through which toxic elements can be exchanged at the interface between the particle and the host tissue. Secondly, the breakdown of particles to a suitable size may allow it to be phagocytosed to affect intracellular metabolism. Thirdly, the dissemination of such particles to distant sites such as lymph nodes, liver, and spleen can interfere with organ functions. Locally, the effect of wear debris can cause necrosis of adjacent periprosthetic tissue, marrow fibrosis and necrosis, cystic destruction of bone, and bone resorption [19].

In our patient's case, we believe the necrosis of the calf muscles serves as a rich nidus or culture medium for hematogenous microbial seeding, possibly from the preceding upper respiratory tract infection. The resultant rhabdomyolysis subsequently tipped the patient over to acute renal failure requiring hemodialysis. The direct toxic and chemical effects of the extruded particulate metallosis could have modulated the immune response during the subsequent microbial attack on the already traumatized muscles and contributed to the development of rhabdomyolysis. There was a lead time of 10 days between presentation and diagnosis. Time should be of the essence in treating and diagnosing this particular condition of metallosis, given its potentially severe complications. To the authors' knowledge, there has been no documented literature report on rhabdomyolysis caused by the noxious effect of short-term exposure of muscle tissue to metallosis.

High index of suspicion should be exercised in the management of such a patient with TKA and severe osteolysis presenting with a calf swelling. The differential diagnoses may include a popliteal cyst (ruptured or unruptured), deep venous thrombosis, granuloma tissue formation, cellulitis, calf abscess, and in this case, tissue reaction to extruded metallosis and wear debris. The loosened long-stem prosthesis and intramedullary osteolysis had allowed the debris of metallosis to extrude at a distance from its origin. Metallosis in itself can mimic infection or septic arthritis [20,21], presenting as a tender, warm, and swollen joint or an abscesslike swelling around the joint. In such an instance, aspiration will help in diagnosis. Other useful investigations include inflammatory markers (erythrocyte sedimentation rate and C-reactive protein), creatine kinase, ultrasound duplex scan, and computed tomography or magnetic resonance imaging to delineate the anatomy of the calf swelling or mass. If in doubt, an arthrogram may reveal abnormal communication with the extra-articular environment via extravasation of the contrast. An algorithm for evaluation of unusual swelling around the knee in TKA patients is shown in Fig. 6.

Although much has been written on the advances in arthroplasty, more work and research remain to be dedicated to the immediate and long-term biologic effects of wear debris and metallosis—a long-known complication of failed joint arthroplasty. Given the potentially severe effects and complications of metallosis as illustrated in our case, the importance of regular orthopedic follow-up, especially of patients at high risk of total joint arthroplasty failure, cannot be overemphasized.

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