Insufficiency fracture of the unresurfaced patella following total knee arthroplasty

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Abstract

Atraumatic patellar fractures after Total Knee Arthroplasty (TKA) are rare in unresurfaced patella. Previous reports are few and have highlighted association of these fractures with rheumatoid arthritis and gout along with interference with patellar blood flow after lateral release and Hoffas’ fat pad excision. We report a case of insufficiency fracture of patella in a patient following TKA after post-operative haematoma and arthrofibrosis that was managed conservatively.

Introduction

Patellar fractures after Total Knee Arthroplasty (TKA) occur in 0.2 and 21% of patients undergoing patellar resurfacing [1]. However, patellar fractures without replacement of patellar component are rare and occur in only 0.05% of patients [2,3]. Medial parapatellar arthrotomy combined with lateral release and excision of Hoffas’ fat pad compromise the patellar blood supply by division of geniculate and infrapatellar arteries [4,5] and this factor has usually been ascribed to causing devascularisation of patella and pathological fracture. Contributing factors include mainly rheumatoid arthritis, corticosteroid intake, gout, excessive knee flexion, obesity, age more than 60 years and these are the main causes in previously reported cases [2]. However, patellar fracture without resurfacing is rare in osteoarthritis. We describe a Case with patellar fracture without resurfacing and without any trauma after TKA due to post-operative arthrofibrosis.

Case report

In February 2010 we performed hybrid total knee arthroplasty (TKA) in a 66-year-old woman due to severe osteoarthritis of the right knee (Figure 1). No patellar resurfacing was done, but careful lateral release was done to protect lateral geniculate artery. Preoperative range of motion was extension/flexion 0/5/90°. One week later we performed open revision due to deep haematoma. Intraoperative samples were sterile with no signs of infection. Further follow-up was uneventful. The patient was recommended to use 2 crutches with partial weight bearing of 20 kg for 6 weeks. The patient received intensive physiotherapy. Nevertheless, the maximum postoperative range of motion was 0/0/90°. X-ray taken after 1 week showed the status after TKR with correct position of the femoral and tibial component (Figure 2). The patient did not wish further revision due to arthrofibrosis. 1.5 years later we saw the patient again for regular clinical and radiologic follow-up. The patient was out of any complaints. Range of motion was 0/0/90°. The patient was able to raise her leg even against strong resistance. X-ray of the right knee showed a correct position of the femoral and tibial component with no radiolucencies but lengthening of the patella with multiple fragments compatible to an insufficiency fracture of the patella (Figure 3). As the patient had a good function of the knee especially of the extensor mechanism we recommended conservative treatment with regular clinical and radiologic follow-ups.

Figure 1. X-ray of the right knee ap view a. and lateral view: b. shows to severe osteoarthritis of the right knee with a varus leg axis.

Figure 3. X-ray of the right knee a. and lateral view: b. shows to severe osteoarthritis of the right knee with a varus leg axis.
Anterior knee pain and patellar fracture are significant complications following Total Knee Arthroplasty (TKA). Patellar fractures without trauma are rare in unresurfaced patella following TKA. Disruption of blood supply has been one of the important reasons causing devascularisation of patella, nevertheless some studies suggest routine patellar resurfacing [1,3,6]. However, the complications increase manifold with resurfacing which can be due to incorrect patellar tracking, metal component, uncemented component and patellar clunk syndrome; that too can cause fracture of patella or patellofemoral problems after TKA the percentage of which is quite high [1]. It is noteworthy to mention that involvement of extensor mechanism is the most common complication after TKA [1,7].

In a recent study of finite element models analysis of strain pattern of patellofemoral (PF) joint in the natural and implanted knee, the volume of bone experiencing strains >0.5% in the implanted condition was approximately 200% larger, on average, than the natural condition. It was also found that highly strained bone was evenly distributed between medial and lateral regions in the natural knee whereas the implanted specimens demonstrated significantly larger volumes of highly strained bone medially as a result of substantially lower modulus bone in the medial compartment. This points to high stress and strain in patella after TKA that predisposes it to insufficiency fracture [8].

Previous case reports accused RA as the most common cause of fracture of the patella and majority of these patients had steroid intake that contributed to Osteonecrosis [9,10]. Reed et al. [2] described a patellar stress fracture as a complication of knee joint arthroplasty without patellar resurfacing. In this case the patient suffered from gout and a previously avascular necrosis of the talus. In that way Reed et al. [2], blamed gout in combination with lateral release as the underlying condition of patellar stress fracture. Our case is different with no underlying metabolic disease but arthrofibrosis. We performed carefully lateral release sparing lateral geniculate artery. In that way we are sure that lateral release did not compromise blood supply of the patella. Fracture of patella was detected 1.5 years after TKA. Fracture of the patella occurred within 2 months in the case reported by Reed et al. [2] and less than 3 years in the cases reported by Seijas Roberto et al. [7]. Ortiguera et al. [11] described that about 80% of patellar fractures occur within 3 years.

Parvizi J et al. [1] described most of the patellar fracture after TKA as asymptomatic; treatment is conservative in most cases which was similar in our case. Despite patellar fracture our patient was asymptomatic with no quadriceps weakness but limited range of motion; the fracture was detected only on routine follow-up. The probable reason for missing weakness of the extensor mechanism despite of the patella stress fracture with lengthening of the patella could be intact patellar retinaculum that maintained the quadriceps function [12]. Hence conservative treatment is recommended in such insufficiency fractures of patella with good functional results. As the strain on patella after TKA is quite high, we think that lateral release of patellar retinaculum should be performed very judiciously in unsurfsaced patella and care should be taken to avoid the division of lateral superior genicular artery.

**Conclusion**

In our case insufficiency fracture occurred due to arthrofibrosis following TKA with a maximum flexion of 90°. In cases of asymptomatic fractures of the patella with good function of the extensor mechanism we recommend conservative treatment.

**References**


