STRESS FRACTURE OF THE PROXIMAL TIBIA AFTER TOTAL KNEE ARTHROPLASTY: A CASE REPORT

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Various complications of total knee arthroplasty (TKA) including infection, periprosthetic fractures, implant failure, loosening of implants, and dislocation are becoming more widely recognized as the number of reported patients and the length of follow-up increase [1–3]. Stress fractures of the tibia most often develop in young individuals pursuing athletic or military activities. These fractures usually appear after unaccustomed and repetitive stressful activity [4, 5]. In elderly people, they are much less common but have been reported in association with generalized osteoporosis [4], osteomalacia [6], rheumatoid arthritis (RA) [7], osteoarthritis (OA) [8], calcium pyrophosphate dihydrate deposition disease arthropathy (CPPD) [9, 10], post-traumatic deformity [11], and TKA [12, 13]. This report describes a patient who developed stress fracture of the proximal tibia after TKA.

Case Report

A 76-year-old woman (height 155 cm, weight 102 kg) with a 6-year history of OA of both knees underwent bilateral TKA at another hospital. She received total arthroplasty (Howmedica, Rutherford, NJ, USA) of the right knee in April 1997, and total left knee arthroplasty in May 1997. The right tibial component was cemented because of insufficient primary fixation. There were no abnormal pathologic findings such as RA or CPPD or other metabolic disorders. Her postoperative course was uneventful after both arthroplasties, and within 3 months she had obtained 110° of knee flexion bilaterally. She could walk without crutches or a walker about 4 months after the last operation. In November 1998, approximately 4 weeks prior to admission to our hospital, she developed pain in the right knee and proximal lower leg that severely limited walking distance. The pain had begun spontaneously while she was walking and worsened with activity. She felt right knee medial laxity and varus deformity, which was worsening progressively. Antiinflammatory drugs failed to relieve her symptoms. The pain was so incapacitating that she had begun to use a wheelchair. She had visited a local clinic where roentgenograms of both knees were taken, but no abnormalities had been identified and she was referred to our hospital for further evaluation. She had no history of trauma, treatment with steroids or hormone medication, or any relevant disease history. On examination, the knee was not warm and there was no effusion. There was a 15° varus deformity during stress test at the right knee and tenderness in the medial aspect of the metaphysis. The range of flexion-extension of the right knee was between 10° and 90°. There
was no gross laxity suggestive of a neuropathic joint. The peripheral pulses were normal. Roentgenograms of the right knee taken at the local clinic in November 1998 showed no abnormalities (Fig. 1). However, similar roentgenograms taken in December 1998 at our hospital after her admission showed an obvious stress fracture of the right proximal tibia (Fig. 2). Roentgenograms of the fracture site, ankle, and knees were taken and a skeletal survey was performed to exclude the possibility of osteomalacia. Bone densitometry was not performed, but roentgenographic evidence of osteoporosis was found in the lumbar spine and bilateral hip with a Singh index of 6 [14]. Test of arterial blood gases, automated profiles of electrolytes, calcium, phosphorus, alkaline phosphatase, and liver function, blood-cell count and profiles, parathyroid hormone, rheumatoid factor, erythrocyte sedimentation rate, C-reactive protein, and urinalysis were all within normal limits. Bed rest and continued treatment with analgesics were prescribed, and the leg was immobilized in an above-knee plaster cast. Conservative treatment of the stress fracture provided prompt improvement in her clinical condition without any further surgical treatment. The fracture healed after immobilizing the limb in a plaster cast for 2 months. At 12 months’ follow-up, she was doing well, with full recovery of right knee function and an excellent result of the knee arthroplasty.

Discussion

In 1980, Rand and Coventry reported the first cases of stress fracture of the tibia following TKA in a series of 15 patients [12]. Petje and Landsiedl reported this condition in a patient in 1997 [13]. Our literature review found only these 16 previously reported cases of stress fracture of the proximal tibia in association with TKA. This condition, however, is probably more common than the literature would suggest.

Haider and Storey considered generalized osteoporosis to be the main factor in the pathogenesis of stress fractures of the tibia following TKA [15]. Osteoporosis results in lower fatigue strength of the bone and stiffness of the adjacent joint due to arthritis, which are undoubtedly important etiologic factors [4, 16]. In the present case, although bone mineral density was not measured, generalized osteoporosis of the lumbar spine and bilateral hip region was obvious on the roentgenograms. It is reasonable to assume that a similar degree of osteoporosis was present in the right knee and tibia.

Fig. 1. A) Anteroposterior and B) lateral roentgenograms of the right knee showed no abnormalities after symptoms began (November 1998).
Severe varus deformities would lead to repetitive abnormal stresses on the proximal tibial metaphysis [17]. Deformity of the knee will produce abnormal loading of the tibia. Eccentric loading of a tibia causes tension stresses on its convex side and compresses the concave aspect [7]. Although TKA is designed to return function to a damaged knee, no one would claim that it completely restores normal biomechanics. Reynolds notes that alignment in the coronal plane is an important biomechanical factor in the etiology and healing of these stress fractures [6]. Martin et al [17] and Satku et al [8] also reported stress fractures of the proximal tibia in patients with RA and OA affecting the knees who had flexion deformity and varus or valgus deformity of the knees. Our patient had a 4-week duration of pain and increasing deformity in the right knee, and varus deformity of the knee may therefore have been one of the causes of this complication.

Restoring a painless range of movement to a knee after TKA increases the patient’s mobility [8]. In the present case, the symptoms developed about 18 months after TKA. During the first 4 months after TKA, she had been able to walk without the aid of crutches or a walker, and did not take any analgesics for knee pain.

CPPD is a general term applied to a disorder that typically manifests roentgenographically as chondrocalcinosis and severe destruction [9, 10]. RA patients who develop stress fracture of the proximal tibia usually have a long history of RA, involving the peripheral joints in a symmetrical fashion [6, 7]. Cartilaginous destruction, bony erosions, and joint deformation are hallmarks of persistent synovial inflammation [6, 7]. In the present case, bilateral TKA was done at another hospital, and OA was the only bony pathology identified during surgery. The patient had no history of steroid treatment, and had normal concentrations of parathyroid hormone. Laboratory tests did not suggest a diagnosis of underlying osteomalacia. Skeletal surveys showed that the patient had generalized osteoporosis.

Severe discomfort localized to the medial aspect of the proximal tibial metaphysis of the right leg was the most important symptom in the present case. Symptoms developed when the patient was walking or weight-bearing, and the pain in the fracture site was mechanical in character [16].

Diagnosis of stress fracture is often delayed [8, 18]. The delay in diagnosis is often attributed to a delay in the patient being seen by the examining physician. The diagnosis is a challenging one, and in the present case, it was established 4 weeks after the symptoms began. Roentgenologic diagnosis is often difficult because early roent-
The roentgenograms may be negative. The minute hairline crack may initially be overlooked and callus may not be apparent for several weeks [7], as in the present case. If there is no other explanation for the acute pain, a stress fracture should be suspected and new roentgenograms of the knee and of the adjacent tibia and femur should be made. The roentgenograms should include the proximal half of the tibia. The present case reminds us of the need for careful examination of the knee and tibia and full length roentgenograms in possible cases of stress fracture.

Since roentgenographic evidence of stress fractures may not be obvious until 2 to 3 months after the onset of symptoms [4], a bone scan and computerized tomography is the recommended diagnostic procedure because results are often positive even before roentgenographic changes are visible. These examinations may also be useful in the diagnosis of avascular necrosis of the femoral or tibial condyle [18].

Devas suggested that compression stress fractures are stable and have little tendency to become displaced [19]. If diagnosed early and treated with rest or reduced activity, they will heal uneventfully. Some authors have also suggested that in elderly patients, stress fractures of the tibia should usually be treated with rest and reduced activity and sometimes with cast, but rarely with surgery [6, 20]. In the present case, the diagnosis of stress fracture of the proximal tibia was recognized early, and the patient was treated with bed rest, above-knee plaster cast, and avoidance of further weight-bearing for 8 weeks. Good healing of the fracture without displacement was achieved and the patient remained asymptomatic at 12 months’ follow-up.

The present case of stress fracture of the proximal tibia as a complication of TKA occurred in a patient who had been walking independently for more than 1 year following TKA. The main causes of this complication in our patient may have involved multiple factors including an increased level of activity after TKA, generalized osteoporosis, and varus deformity of the right knee. The possibility of stress fracture of the tibia should be included in the list of potential complications following TKA.

**References**