Stress Fracture of the Tibial Tubercle in a Collegiate Volleyball Player

Samir G. Tejwani, MD
Ali R. Motamedi, MD

Tibial stress fractures in young athletes most commonly occur in the proximal and middle thirds of the bone along the medial aspect. The orientation of tibial stress fractures usually is transverse, although longitudinal and spiral patterns have been reported.

Less common tibial stress fractures have been described in the anterior diaphyseal cortex, posterior diaphyseal and metaphyseal cortices, lateral plateau, and medial malleolus. When treated by activity cessation with or without casting in young patients, tibial stress fractures typically heal without sequelae. Midanterior stress fractures are an exception, and often become chronic, with nonoperative management proving unsuccessful.

This article presents a stress fracture of the tibial tubercle in a skeletally mature patient. Internal fixation was ultimately used to alleviate pain and allow expeditious return to competitive sport.

CASE REPORT

A 20-year-old male collegiate volleyball player presented with insidiously progressing right anterior knee pain of 3 months’ duration. No history of trauma to the knee was reported; however, the frequency and intensity of volleyball practice sessions had increased.

Medical history was significant for ipsilateral Osgood-Schlatter disease that resolved early in high school with nonoperative management. Initial examination was consistent with insertional patellar tendinitis of the right knee; palpation revealed point tenderness at the distal insertion of the patellar tendon and proximal aspect of the tibial tubercle. Full range of knee motion without functional motor or sensory deficits was noted.

Nonoperative treatment for patellar tendinitis included cryotherapy, nonsteroidal anti-inflammatory medication, ultrasound, and immediate cessation of volleyball play. Physical therapy with an emphasis on quadriceps and hamstring stretching also was begun. Weight bearing was not restricted. After 6 weeks, the patient continued to experience pain and was unable to return to play.

Radiographs of the right knee demonstrated a small oval focus of calcification in the infrapatellar fat pad 5 mm distal to the inferior pole of the patella, which was interpreted as a phlebolith (Figure 1). Nonoperative modalities were continued for an additional 6 weeks without improvement.

Magnetic resonance imaging (MRI) of the right knee demonstrated linear enhancement at the base of the tibial tubercle and edema in the surrounding cancellous bone and tibial tubercle, consistent with a stress fracture (Figure 2). The patellar tendon was normal in signal and thickness without evidence of tear, degeneration, or calcification (Figure 3).

Tibial tubercle stress fracture should be suspected when a history of running or repetitive jumping, without trauma, is present, particularly in patients with a presumed diagnosis of insertional patellar tendinitis who do not respond to adequate nonoperative treatment.
Closed treatment, consisting of prolonged cast immobilization, versus surgical fixation was discussed with the patient. Because of the patient’s high level of competitive volleyball participation and his desire to return to play as quickly as possible, he elected surgical intervention. Internal fixation of the right tibial tubercle with a 3.5-mm bicortical lag screw, which was countersunk, was performed (Figure 4). No perioperative complications occurred.

Postoperatively, immediate weight bearing as tolerated and an active range of motion and strengthening program were begun. Two weeks postoperatively, the right knee was pain-free. Physical examination 6 weeks postoperatively revealed full range of motion, full motor and sensory function of the lower extremity, and no tenderness to palpation at the tibial tubercle. The patient returned to play without incident. At 18-month follow-up, the patient is playing volleyball at the collegiate level, is pain-free, and has no reported functional deficits.

**DISCUSSION**

Ehrenborg\textsuperscript{12} divided the postnatal development of the tibial tubercle into four stages. The tibial tubercle is a discrete cartilaginous structure by the 15th week of fetal development. This cartilaginous stage occurs before the secondary ossification center appears, and persists in girls until age 9 years and in boys until age 10 years. The apophyseal stage, in which the ossification center appears in the tongue of the cartilage, occurs between ages 8 and 12 years in girls and ages 9 and 14 years in boys. The epiphyseal stage, in which the secondary ossification centers coalesce to form a tongue of bone continuous with the proximal tibial epiphysis, occurs in girls between ages 10 and 15 years and in boys between ages 11 and 17 years. In the final bony stage, the epiphyseal line is closed between the fully ossified tuberosity and tibial metaphysis. Thus, despite variability in the onset and completion of ossification of the tibial tubercle apophysis, by age 17 years the process is completed in the majority of individuals.

In the clinical syndrome first described by Paget\textsuperscript{13} and later by
Osgood\textsuperscript{14} and Schlatter,\textsuperscript{15} now termed Osgood-Schlatter disease, repetitive stress results in a separation of the immature chondro-osseous structures in the anterior aspect of the tibial tubercle in the skeletally immature; the deeper layers of the apophysis and cartilage of the underlying physis remain intact. If the process occurs in the pres ossification phase of development of the physis, or if the avulsed fragments are completely cartilaginous, symptoms will precede radiographic evidence of diagnosis. However, with normal maturation, the avulsed fragments continue to grow and ossify and eventually become visible as separate bony fragments. Typically, the tissue between the avulsed bony fragments and underlying tuberosity also ossifies, creating the appearance of “overgrowth” of the tubercle at skeletal maturity.\textsuperscript{16}

The mainstay of treatment for Osgood-Schlatter disease is symptomatic and supportive, with operative treatment reserved for nonunited ossicles with persistent symptoms after the physes have closed. Activity cessation with or without casting usually is effective in alleviating symptoms, and no clear evidence to date indicates that the tibial tubercle apophysis ossifies in a weakened structural state.

The patient in this report presented with Osgood-Schlatter disease in the right knee early in high-school and was successfully treated nonoperatively. He was playing volleyball at the high school and collegiate level for approximately 5 years prior to presenting with complaints of pain at the tibial tubercle. His significant asymptomatic period while returning to play suggests that no immediate sequelae from the management of Osgood-Schlatter disease or any clinically significant structural defects at the tibial tubercle due to the disease were present.

A lesion in Osgood-Schlatter disease may precede an acute avulsion of the tibial tubercle. However, in reports by Deliyannis,\textsuperscript{17} Levi and Coleman,\textsuperscript{18} and Ogden et al,\textsuperscript{19} the patients were all skeletally immature adolescents. The avulsion typically is caused by a severe concentric or eccentric contraction of the extensor mechanism through the tibial tubercle apophysis, between ages 12 and 16 years, prior to closure of the physis. Activities where the knee is forcibly flexed against a contracted or resisting quadriceps, such as in high jump, basketball, and football, typically are responsible.\textsuperscript{20,21} In these cases, open reduction and internal fixation is recommended for all but the smallest undisplaced fragments, which can be treated by closed means if the knee can be extended to 0°. One or two cancellous screws with washers are used, extending through the tubercle, parallel to the joint, into the metaphysis. If the patient is >3 years from skeletal maturity, smooth pins are used.\textsuperscript{22}

In this case, the patient presented with pain in the tibial tubercle at skeletal maturity, and radiographs confirmed full ossification of the tibial tubercle and closure of the proximal tibial epiphysis. Although the most frequently seen and reliable radiographic sign of a stress fracture is periosteal and endosteal cortical thickening,\textsuperscript{23} in a patient with tibial tubercle overgrowth secondary to Osgood-Schlatter disease, these findings can be obscured.

Occult fracture lines can display high signal on T2-weighted MRI sequences.\textsuperscript{24} T2-weighted MRI in this patient demonstrated linear enhancement at the base of the tibial tubercle as well as edema in the surrounding cancellous bone and tibial tubercle, consistent with a stress fracture of the tibial tubercle. This is the first report of a tibial tubercle stress fracture in a skeletally mature patient. It is unclear whether the history of ipsilateral Osgood-Schlatter disease was a predisposing factor in the development of the stress fracture. In this patient, the gross morphology of the tibial tubercle was altered through the repetitive microtrauma that is well described in Osgood-Schlatter disease, leading to moderate overgrowth. It is unknown whether on a histologic level structural insufficiencies were present in the tibial tubercle due to the Osgood-Schlatter disease that predisposed to stress fracture. The patient did, however, progress through high school and on to collegiate volleyball play after conservative treatment of Osgood-Schlatter disease, and was asymptomatic for approximately 5 years while continuing to play. From the patient’s history, it was suggested that the increase in intensity of volleyball practice over the prior 3 months contributed to the development of tibial tubercle stress fracture.

Of interest is whether tibial tubercle stress fractures are more prevalent than thought, particularly in the subset of athletes who are involved in repetitive running or jumping and report anterior knee pain. In these patients, clinical examination may lead to a misdiagnosis of insertion patellar tendinitis. Cessation of the causative activity could heal patellar tendinitis and a tibial tubercle stress fracture, with the true diagnosis evading the treating clinician. As with stress fractures in other parts of the tibia, it is possible that cast immobilization would effectively promote healing of a tibial tubercle stress fracture; this treatment option should be used when other conservative modalities have failed and no urgency for fracture healing exists.

Tibial tubercle stress fracture should be included in the differential diagnosis of a skeletally mature athlete with anterior knee pain. This condition should be suspected when a history of repetitive running or jumping, without trauma, is present, particularly in patients with a presumed diagnosis of insertion patellar tendinitis who do not respond to nonoperative treatment.

Although plain radiographs were unremarkable, MRI was a useful modality in identifying the pathology and making the diagnosis in this patient. An unclear relationship exists between a history of ipsilateral Osgood-Schlatter disease and the subsequent development of a tibial tubercle stress fracture. Theoretically, microstructural insufficiencies in the tibercle could facilitate
mechanical failure under repetitive stress. Unlike our experience with patellar tendinitis, this condition did not respond to conservative treatment. Closed immobilization remains a viable treatment option; however, we did not attempt this due to the desire for expeditious return to sport. Internal fixation of the tibial tubercle stress fracture in this skeletally mature patient was successful in alleviating pain and allowing return to collegiate-level volleyball in 6 weeks; the patient remains pain-free at 18-month follow-up. The operative technique was similar to that used for treatment of acute avulsion of the tibial tubercle in the skeletally immature.

**REFERENCES**


Section Editor: Darren L. Johnson, MD