Persistent Knee Pain In A Collegiate Track And Field Athlete
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Background: A 22-year old male multi-event intercollegiate track and field athlete presented with moderate persistent left anterior knee pain secondary to various athletic activities without any unusual sounds or sensations. The patient reported being able to perform ADLs, running, and lower extremity conditioning exercises; however, the pain significantly increased with these activities. Visual examination revealed a slight deformity in the patellar tendon, as well as a medially rotated patella at rest between the femoral condyles. Physical inspection elicited palpable tenderness and edema over the patellar tendon and specifically, the inferior patellar pole. Functional inspections including ROM, special, and neurological testing were WNL; however, active and resisted extension and passive flexion were painful. The patient’s medical history was significant for bilateral Osgood-Schlatter’s Disease at age 13, which required extensive rest and a gradual return to activity. Throughout high school, the patient was able to participate in athletic activities without significant left knee pain. The patient was not currently taking any medication; however, reported being hypersensitive to all NSAIDs. Differential Diagnosis: Infrapatellar tendinopathy, fat pad impingement, patellofemoral instability, patellar malalignment, infrapatellar bursitis; patellar osteochondrosis, osteochondritis dissecans, osteogenic sarcoma, patellar tendon calcification. Treatment: After the initial evaluation, the patient received conservative treatment encompassing ice, compression, and elevation and was referred to the team physician who ordered radiographs. Radiographs revealed a 4.5 cm bony calcification disconnected from the tibial tuberosity that appeared to have grown into the patellar tendon. The patient was diagnosed with a non-union patellar tendon calcification derived from Osgood-Schlatter’s Disease with inferior pole patellar tendinopathy and infrapatellar bursitis as a secondary diagnosis. The physician attributed the tendinopathy and bursitis to the size, location, and instability of the calcification. The patient was removed from activity for one week to control the inflammation and pain and was prescribed a neoprene sleeve to assist with compression, patellar alignment, and patellar stability. Following initial rest, a rehabilitation program consisting of partial weight bearing isotonic exercises and aquatic therapy was implemented to restore and improve muscular strength and cardiovascular fitness. After one week of the conservative therapeutic exercise program, a progressive rehabilitation program was incorporated emphasizing calf and eccentric extensor mechanism strengthening, core stabilization and strengthening, and quadriceps and hamstring flexibility. The patient also returned to limited team activities that included running, but no jumping. Status post four weeks, the patient reported being pain free and was conditionally cleared by the team physician for unrestricted athletic activities, pending continuation of rehabilitation. The patient continues to address core and lower extremity strength and flexibility deficiencies. Uniqueness: Osgood-Schlatter’s disease is a traction apophysitis causing a tibial osteochondrosis that traditionally responds well to restricted activity, stretching, and NSAIDs. A long-term sequella of the condition is typically a thickening and prominence fusion of the tibial tubercle; however, this case is unique because a 4.5 cm non-union calcification formed at the tibial tuberosity which appeared to have grown into the patellar tendon itself, and the calcification size, location, and instability resulted in poor static control of the patella. Also, this calcific instability in the tendon contributed to the secondary patellar tendinopathy and bursitis. Conclusion: Skeletally mature individuals may continue to be symptomatic from pathologies associated with Osgood-Schlatter’s disease. An unstable patellar tendon attachment on the tibial tubercle resulting from a non-union secondary to Osgood-Schlatter’s disease contributed to the development the patient’s disability. Unfortunately, a calcification within the patellar tendon formed during the bony fusion of the tibial tubercle caused lingering symptoms. In addition to the initial treatment of rest and a gradual return to activity, management should include core, calf, and knee extensor mechanism stretching, strengthening, and patient education. Word Count: 598