Pellegrini-Stieda Disease in a Patient with Spinal Cord Injury

Tuzun S, Uludag M, Atarod N, Ugurlu S.

Istanbul University,Cerrahpasa Medical Faculty, Department of Physical medicine & Rehabilitation, Istanbul, Turkey

A 44-year-old female with complete paraplegia, resulting from an L1 fracture following a car accident 20 years ago, presented to our clinic with swelling of the left knee. Her swelling had manifested 6 min previously and worsened gradually. The patient had no history of systemic disease, such as gout, rheumatoid arthritis, systemic sclerosis, sarcoidosis, or hyperparathyroidism, and there had been no recent trauma to the knee. On physical examination, the lower extremities were found to be in stage 2 spasticity, according to the Ashworth scale. Superficial sensation had been lost bilaterally, beginning at the L1 level. Deep tendon reflexes had increased, and she tested positive for Babinski's sign and clonus. The patient had no anal sensation or voluntary anal contraction. Her neurological level was classified as T12 American Spinal Injury Association Impairment Scale (AIS) A. In addition to the swelling in the left knee, limited and passive range of motion was observed in both knee joints, but particularly in the left. An anteo-posterior bilateral radiograph of the knee joints revealed the drop-like ossification of the left medial collateral ligament (Figure).

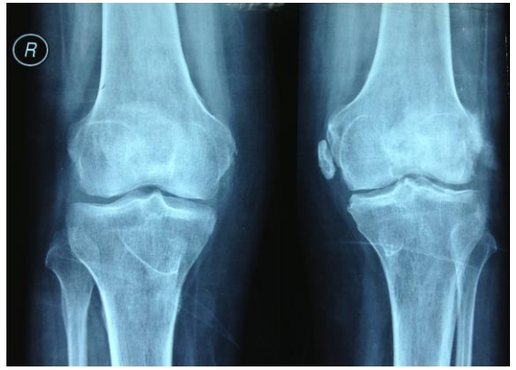

Figure*: Knee anteroposterior radiography reveals the drop-like ossification of the tibial collateral ligament of the left knee.*

Paralysis and sensory deficits predispose patients with SCI to traumatic knee effusions. Microtrauma, due to exercise, the positioning of the joint, or joint capsule distension (due to decreased muscular support) may result in knee effusion. Physiotherapists may exert up to six times the force tolerated by neurologically intact individuals when effecting hamstring stretches on flaccid patients. Altered biomechanics of the joint may irritate the tissues or lead to diminished nutrition supply to the joint cartilage. Either or both mechanisms may lead to effusion [1].

Pellegrini–Stieda (PS) disease is characterized by calcification or ossification, or both, in the tibial collateral ligament, usually related to a history of trauma [2]. Patients typically present with pain in the femoral condyle, which is aggravated by activity, but lesions can also be coincidental. Several theories have been proposed to explain the pathogenesis of PS disease, none of which have been widely accepted. First described in the 1900s, pathogenesis probably involves calcification of a posttraumatic hematoma, but other postulated etiologies include metaplasia of the ligamentous tissue, an avulsion fracture of the medial femoral epicondyle, periosteal proliferation, a type of myositis ossification, or traction tear of the tendon of the ischiocondylar portion of the adductor magnus muscle, resulting in periosteal new-bone formation [2,3].

Several factors, such as tissue hypoxia, hypercalcemia, changes in sympathetic nerve activity, prolonged immobilization, and mobilization with frequent periods of exercise following prolonged immobilization, may precipitate neurogenic ectopic bone formation [4].
The symptom course of PS disease is generally self-limiting. Treatment is symptomatic and comprises local corticosteroid injections and range-of-motion exercises for mild-to-moderate cases. Surgical excision of calcifications and tear reparation in the medial collateral ligament should be considered for refractory cases [4,5]. PS disease can be observed in neurologic rehabilitation patients such as spinal cord injury during aggressive range-of-motion exercises, and it must be considered for the differential diagnosis of knee pain, swelling,
and range-of-motion impairments.

REFERENCES

1.Kupfer M, Dholakia M, O'Neill BJ. Effusion of the hips in a patient with tetraplegia. J Spinal Cord Med 2006;29:160-162.

2.Mendes LF, Pretterklieber ML, Cho JH, et al. Pellegrini-Stieda disease: a heterogeneous disorder not synonymous with ossification/calcification of the tibial collateral ligament-anatomic and imaging investigation.Skeletal Radiol 2006;35:916-922.

3.Majjhoo A, Sagar H. Pellegrini-Stieda disease: calcification of the medial collateral ligament. J Clin Rheumatol 2011;17:456.

4.Altschuler EL, Bryce TN. Images in clinical medicine. Pellegrini-Stieda syndrome. N Engl J Med 2006;354:e1.

5.Yildiz N, Ardic F, Sabir N, Ercidogan O. Pellegrini-Stieda disease in traumatic brain injury rehabilitation. Am J Phys Med Rehabil 2008;87:514.