Case Report
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Intraligamentous Calcification of the Medial Collateral Ligament Mimicking Pellegrini-Stieda Syndrome in a Lower-Extremity Amputee

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Abstract

Soft-tissue calcification is characterized by the accumulation of calcium in damaged collagen fibrils. The pathogenesis of calcium deposition is not fully understood. Inflammatory changes, trauma, and rheumatological diseases have been reported as possible risk factors. Pellegrini-Stieda syndrome (PSS) is post-traumatic calcification or ossification of the medial collateral ligament with a nonspecific etiology. It may occur after trauma or inflammation or it could be idiopathic. Here we present a case of posttraumatic intraligamentous calcification of the medial collateral ligament (MCL) mimicking Pellegrini-Stieda syndrome (PSS) and review the current related literature.
Keywords: Intraligamentous calcification, Pellegrini–Stieda syndrome, postamputation pain, trauma

Introduction

Soft-tissue calcification is caused by large deposits of calcium between or within degenerated collagen fibrils. Shoulder is the common region for calcification, and one in every fifth healthy person has a calcification of the rotator cuff. Histological evaluation has shown nodular deposition of calcium in the collagen fibrils, vascular proliferation, and inflammatory changes (1).

Humoral factors; neural factors; local factors, including hypoxia and hypercalcemia; changes in the sympathetic nervous system activity; long-term immobilization; and mobilization process with frequent exercise periods after the long-term immobilization have been attributed to cause some syndromes with neurogenic ectopic bone formation and calcification, including Pellegrini–Stieda syndrome (PSS) (2). PSS is characterized by the calcification and ossification of the medial collateral ligament (MCL), usually associated with a history of direct or indirect trauma and repetitive microtrauma (3-6).

Here we present the case of a posttraumatic lower-extremity amputee who had also intraligamentous calcification of MCL, mimicking PSS. We also review the related current literature.

Case Report

A 31-year-old male had undergone left knee disarticulation surgery on the same day of his admission to the emergency department because of a motor vehicle accident-related crush injury on his left lower extremity. After the surgery, he had been transferred to the neurosurgery intensive care unit for the treatment of a temporal fracture and cerebral edema. His condition was stabilized without any complications in the follow-up period. Shortly after his discharge from the hospital, the patient

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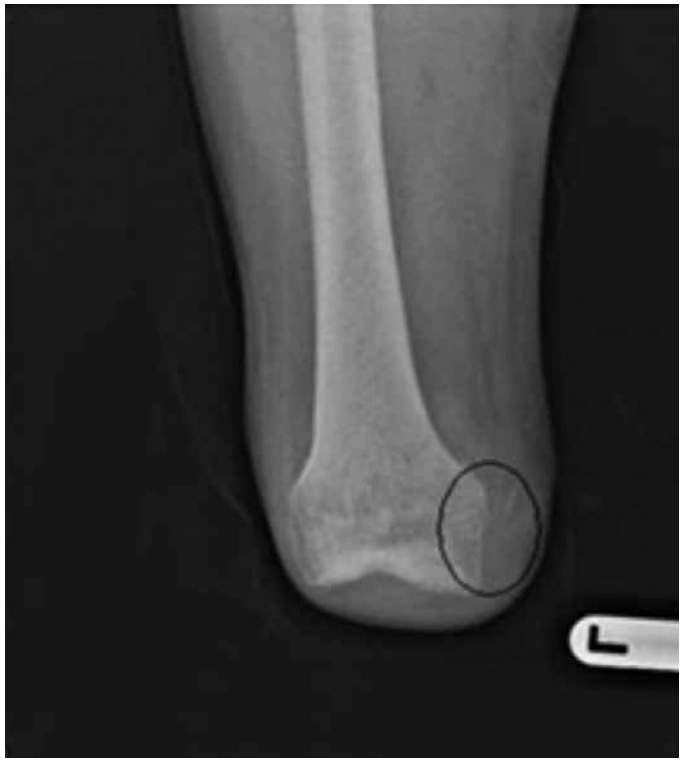

Figure 1. Anteroposterior X-Ray of the left femur demonstrates the linear calcification of the medial collateral ligament

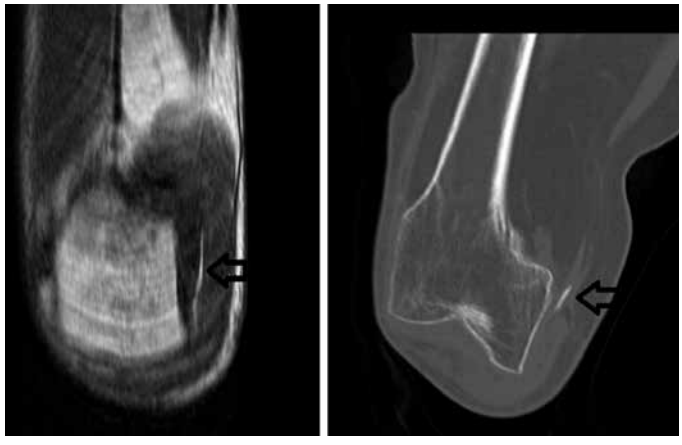

Figure 2. a,b. Intraligamentous calcification of the medial collateral ligament on MRI (a), intraligamentous calcification of the medial collateral ligament on CT (b)

was admitted to our outpatient clinic with the complaint of local tenderness on the medial side of his left knee.

Physical examination revealed a below-knee amputation on the left side. Atrophy was evident in the stump muscles, but no significant motor deficit was present. Both hip joints had a full range of motion. Left femoral medial condyle was tender, but there was no palpable nodule or signs of warmth, discharge, or open wound. Laboratory findings revealed an erythrocyte sedimentation rate of 49 mm/h (0-25 mm/h), CRP of 7.61 mg/dL (0-M0.8 mg/dL), and a white blood cell count of 16.67 µL (4.3-10.03 µL). The levels of serum Ca, P, and ALP were within their normal ranges. The patient was diagnosed with a pulmonary infection on the basis of chest radiography findings and a consultation with the Chest Diseases Department; he was administered an antibiotic regimen for 2 weeks.

Plain X-rays of femur and knee revealed a fine linear calcification on the medial aspect of the distal femur. At baseline, only plain X-Rays were obtained because of the serious general status of the patient (Figure 1). In the second year of the follow-up, magnetic resonance imaging (MRI) and computerized tomographic (CT) evaluation revealed a linear calcification of the MCL with a similar size as that observed in the baseline X-Ray (Figure 2 a, b). The orthopedic surgeon who performed the amputation reported that MCL was preserved but the insertion site of the ligament was pulled upward during the operation. Non-steroidal anti-inflammatory drugs were administered to the patient, and the regression of the symptoms was confirmed during the follow-up.

Discussion

In regular orthopedic practice, the incidence of symptomatic calcium deposits in ligaments and tendons is best manifested as tendinitis or tendinosis of the rotator cuff. The cause of calcium deposits in soft tissues remains ultimately unclear (7). The accepted theory is that the trauma results in a hydroxyapatite or calcium pyrophosphate dihydrate crystal deposition (8).

Calcification presents with pain and thus may mimic other abnormalities associated with inflammation, such as joint infection. Heterotopic ossification can be so painful that it may mimic acute knee trauma, such as meniscal or ligamentous tear, fracture, or PSS.

In the current case, although wound-site infection was suspected initially because of the increased levels of acute phase reactants, further investigation revealed a pulmonary infection.

Heterotopic ossification was considered in the differential diagnosis. However, the MRI and CT findings of the patient at the second year follow-up revealed a nonprogressive intraligamentous calcification; hence, heterotopic ossification was ruled out.

This case mimicked PSS because of the location of calcification at the medial knee on MRI and CT images. There is no accepted consensus regarding a classification system for PSS, but the radiological imaging findings in the current case resembled type 2 PS (9). Although trauma plays a major role in the etiology, symptomatic cases of PSS with no identifiable underlying cause have also been reported (10). In our case, we consider that the preservation of the ligament with relocation of the insertion site during the posttraumatic amputation surgery may have led to a decrease in blood supply, resulting in hypoxia and the development of linear calcifications. Rare causes of PSS, including traumatic brain injury and cauda equina syndrome, have been described in the literature (3,4). Strenuous exercise has been proposed as the initiating factor in this group of patients. Our patient has not performed any exercise protocol, disproving this hypothesis.

In fact, it is suggested that the pathology does not develop only in the ligament. Mendes et al. (9) showed that calcifica-

tions may occur not only in MCL but also within the fibers of adductor magnus muscle, which join the ligament at its superior aspect. In our case, the calcification of MCL has was purely intraligamentous.

Conclusion

Here we presented a case of intraligamentous calcification of MCL after lower-extremity amputation, which mimicked PSS. The present report emphasizes the consideration of soft-tissue calcification in the differential diagnosis of medial knee or lower thigh pain following knee disarticulation, apart from other well-known causes of post-amputation pain.

Informed Consent: Written informed consent was obtained patient who participated in this case.

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