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Case Report

Myositis ossificans circumscripta in the psoas muscle with femoral neuropathy

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ABSTRACT

Myositis ossificans circumscripta is a benign, self-limiting, ossifying soft-tissue mass that typically occurs in skeletal muscle of the extremities after trauma. Occurrence in the psoas muscle is rare with no reports of this condition presenting as femoral neuropathy. A 54-year-old woman visited our hospital because of progressive left leg pain and weakness, which she had experienced for 2 years. Physical examination indicated left femoral neuropathy, and a 6 cm × 5 cm mass was palpable in the left groin. Plain radiographs showed an irregular calcification over the left hip. Computed tomography of the pelvis showed a fusiform mass within the left psoas muscle. A modified ilioinguinal approach was used to excise the mass, and histologic examination confirmed the diagnosis. After surgery, the patient's symptoms resolved and there was no recurrence at 4 years' follow-up. Excision may be considered when a patient has a large, painful mass with neuropathy and significant functional impairment after conservative treatment.

1. Introduction

Myositis ossificans circumscripta (MOC) is a benign, self-limiting, solitary, and ossifying soft-tissue mass that occurs in the skeletal muscle of the extremities after injury.[1,2] Patients with these lesions may or may not recall a history of antecedent trauma.[3,4] The pathogenesis of MOC remains unknown. In the literature, there are only a few case reports concerning MOC in the psoas muscle.[3,5–8] Here, we report a rare case of MOC affecting the unilateral psoas muscle presenting with femoral neuropathy.

2. Case report

A 54-year-old woman with no history of trauma presented with progressive left leg pain and weakness, which she had suffered from for 2 years. Upon admission, the visual analog scale (VAS) for pain read 8 points (8/10). On physical examination, a sensation of numbness was mainly distributed over the left anterior thigh. In addition, strength of the left hip flexion was diminished [Medical Research Council (MRC) scale 4/5]. The active ranges of motion of the left hip, which were 70° for flexion, 15° for extension, 40° for abduction, and 30° for adduction, were decreased in flexion and extension as compared with the ranges of the right hip. There was left hip pain on performance of the straight leg-raising test, and severe pain occurred upon passive stretching at 15° of the left hip extension. The deep tendon reflexes were normal at the knees and ankles. A 6 cm × 5 cm tumor of rubber-like consistency was palpable at the left inguina. Plain films of the left hip showed an irregular calcification over the anterior aspect of the left hip with preservation of the joint space (Fig. 1). Computed tomography (CT) of the pelvis showed a focal, well-defined, fusiform mass-like lesion over the left pelvic wall and the anterior aspect of the left hip, mainly along the left psoas muscle, from S1 level to the lesser trochanter of the left femur (Fig. 2A). The lesion consisted of irregular fat (low attenuation value) and soft tissue densities (isodense to muscles), and with multiple rims of irregular calcifications (high attenuation value) (Fig. 2B). The soft-tissue density of the lesion had mild contrast enhancement. These findings favored focal fatty change with MOC of the left psoas muscle.

The patient underwent excision because her pain was still agonizing after a watchful waiting period of 2.5 months. A modified ilioinguinal incision was used to excise the mass, which was clearly visible after splitting the psoas muscle (Fig. 3A) and was easily enucleated with blunt dissection (Fig. 3B). On the next day, the patient's pain had decreased and the VAS for pain read three points (3/10). Her symptoms resolved and the muscle power of her left hip

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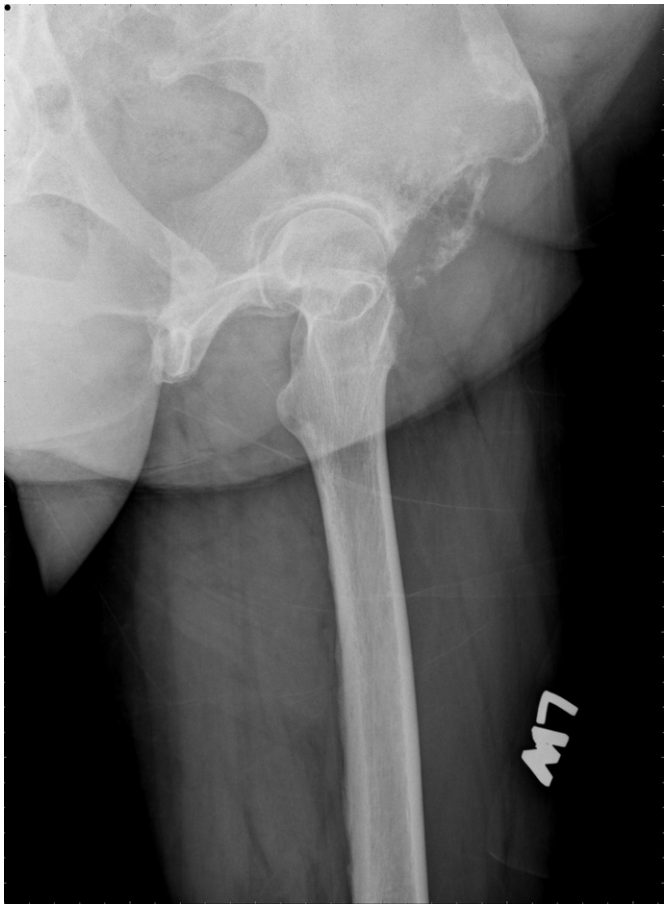

Fig. 1. Plain radiograph (lateral view) of the left hip shows an area of irregular calcification over the anterior aspect of the left hip.

returned. Pathology revealed a zonal phenomenon pathognomonic of MOC,[9] in which lesional maturation progressed from peripheral to central, nonossified cellular foci to osteoid to peripheral rims of mature lamellar bone (Fig. 4). Ten days after surgery, the patient was pain free (VAS 0/10) and there was no limitation of range of motion of her left hip. She was given celecoxib[10] for 1 month to prevent recurrence, and she did not undergo low-dose radiotherapy. Plain films of the left hip at 2.5 years' follow-up showed no signs of local recurrence (Fig. 5). At 4 years' follow-up, she remained free of symptoms and signs.

3. Discussion

Femoral neuropathy presents with varying symptoms and signs depending on both severity and location of the injury, and is characterized typically by groin or thigh pain, weakness of the iliopsoas, paralysis of the quadriceps femoris, loss of knee jerk, and sensory loss over the anteromedial aspect of a lower extremity.[11] The differential diagnosis includes lumbar radiculopathy, lumbar polyradiculopathy, lumbar plexopathy, avascular necrosis of the femoral head, and polymyalgia rheumatica.[12] Diabetic amyotrophy is the most common cause of focal neuropathy, but hemorrhage is also common.[12] Other culprits include trauma, surgery, infection, cancer, pregnancy, and radiation.[11,12] Reports of psoas MOC in the literature mainly described symptoms of groin pain and limited range of motion of the hip joints without addressing issues of neuropathies.[3,5–8]

The appearance of MOC on images varies with its stage of development, and the findings on conventional radiography, ultrasonography, scintigraphy, CT, and magnetic resonance imaging are well documented.[13] CT is the best imaging modality for the diagnosis of MOC.[14] In the early stage of the disease, plain radiographs are normal except for a localized, slight increase in soft-tissue density.[13] CT shows an enlarged muscle group with normal attenuation with or without faint calcification.[13] In the intermediate stage, plain radiographs show faint, irregular, floccular calcification (within 2–6 weeks after onset of symptoms), and later the lesion becomes sharply circumscribed (by 6–8 weeks).[13,15] In addition, noncontinuous or continuous peripheral calcification with a central lucent core and possible radiolucent cleft between the lesion and adjacent bone may present. CT shows a rim of calcification (after 4–6 weeks) with varying thickness at the periphery (zonal phenomenon) with a central area of the same attenuation as normal muscle.[15] In the late stage, plain radiographs show a heavily calcified lesion (by 5–6 months) with trabecular bone formation, and the lesion may merge with adjacent cortex mimicking an osteochondroma.[13,15] CT shows a heavily calcified lesion.

The magnetic resonance appearance also varies, reflecting the histologic changes.[2] Early lesions are poorly defined and isointense on T1-weighted images, heterogeneously T2 hyperintense, and have diffuse surrounding soft-tissue edema.[2] As peripheral calcification develops, a peripheral low signal intensity may became visible.[2] Mature lesions are well-defined masses that are isointense to fat centrally and have low signal intensity peripherally, without surrounding edema, on both T1- and T2-weighted images.[2]

Serum alkaline phosphatase levels have been reported to parallel the activity of ossification. Typically, serum alkaline phosphatase levels rise approximately 2 weeks after injury, and reach

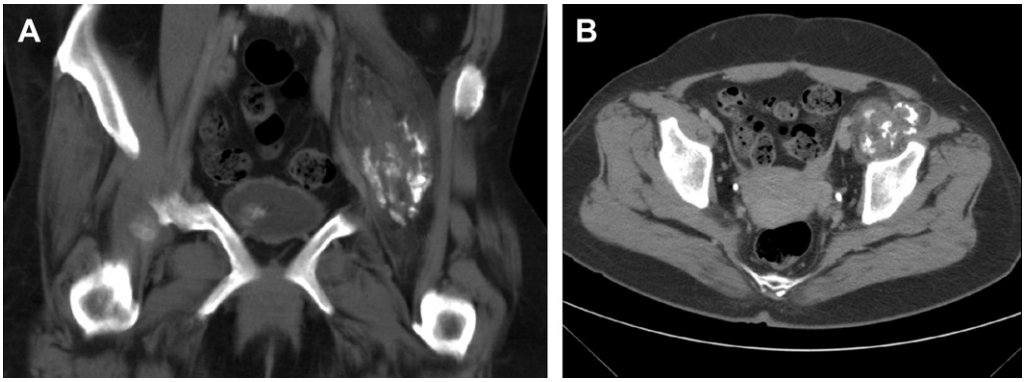

Fig. 2. (A) Three-dimensional reconstructed computed tomography (CT) of the pelvis shows a focal, well-defined, fusiform, mass-like lesion, with calcification and a low attenuation region, mainly along the left psoas muscle, from S1 level to the lesser trochanter of the left femur. (B) Cross-sectional CT of the pelvis shows multiple rims of calcifications inside the left psoas muscle with varying thickness at the periphery (zonal phenomenon).

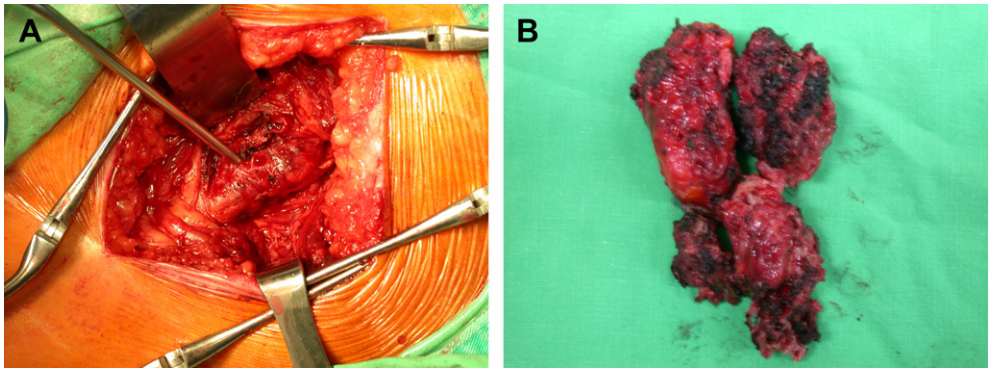

Fig. 3. (A) Intraoperative photograph shows the calcified mass after splitting the psoas muscle. (B) Photograph shows the enucleated mass was lobulated and calcified.

approximately 3.5 times the normal value 10 weeks after the inciting trauma before returning to normal at approximately 18 weeks.[16] Serum alkaline phosphatase levels, however, cannot be used to predict maturity or recurrence of heterotopic ossification, and in many patients, values may be normal in the presence of active disease or may remain elevated for years.[17] In addition, the elevation can be nonspecific because of healing fractures, occult biliary disease, or normal bone growth in younger patients.[16]

Nontraumatic MOC usually occurs after muscle injury such as repeated microtrauma, and in a small number of cases, possible causes include infections, burns, neuromuscular disorders, hemophilia, tetanus, and drug abuse.[18] Early to intermediate MOC may be confused with sarcoma or infection especially when there is no history of trauma.[3] In MOC of the psoas muscle, as in our case, the main differential diagnostic considerations are calcified lipoma, sarcoma, and infection.[3,6]

Current initial treatment of MOC includes restriction of activity and gentle stretching exercises to prevent contractures.[19] The full course of growth of MOC is approximately 7–8 weeks from inception, and this information can help in making a diagnosis and judging whether or not the ossification is mature in order to reduce the risk of recurrence after surgery.[4,13] Surgical removal may be indicated when a large, painful mass of mature bone is associated with significant functional impairment or with compression neuropathy, and surgical excision is generally delayed for 6–12 months until the lesion has matured and bone scan activity has decreased.[20,21] Järvinen et al[22] suggested the excision should be performed 12–24 months after the onset of symptoms because the excision of immature bone often results in local recurrence. Ogilvie-Harris and Fornasier[23] suggested the nontraumatic MOC may be excised at any time in its evolution, with minimum risk of recurrence.[23] Indomethacin and other nonsteroidal anti-inflammatory drugs are commonly used in orthopaedics to prevent heterotopic ossification, but they have not been validated for the prevention and treatment of MOC.[22] Buselli et al[24] reported using

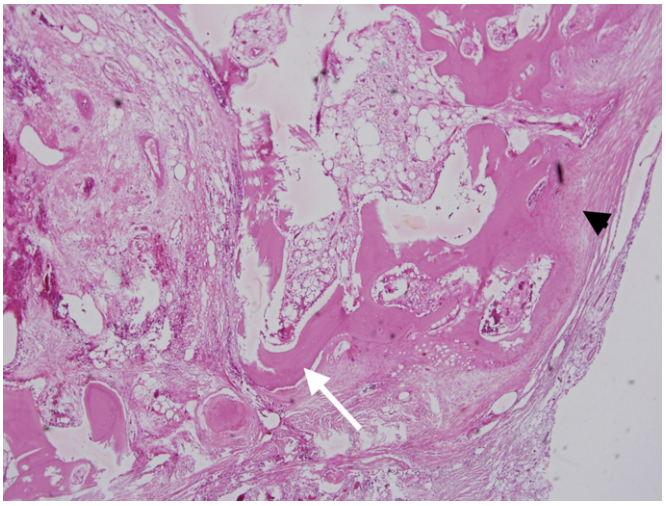

Fig. 4. Photomicrograph of the ossified mass shows muscle fibers, fat cells, and proliferating fibroblasts (arrowhead) giving rise to woven bone (arrow). Hematoxylin and eosin, ×40.

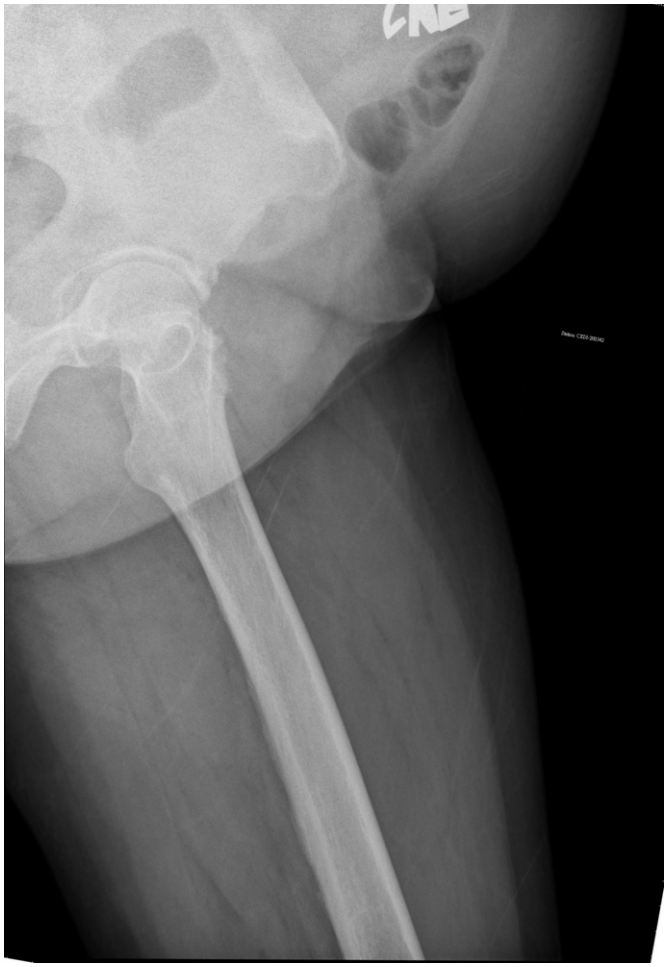

Fig. 5. Plain radiograph (lateral view) of the left hip shows no calcification over the anterior aspect at 2.5 years' follow-up.

extracorporeal shock wave therapy to treat 24 athletes with traumatic MOC, and 21 of the athletes (87.1%) returned to previous athletic activity levels 3 months after treatment. Improvement in a patient with persistent asymptomatic calcification has been reported with bisphosphonate therapy.[14] Most lesions remain stable and asymptomatic after maturation.[18]

MOC that develops in the psoas muscle may manifest as a femoral neuropathy, and direct compression of the femoral nerve can cause symptoms. Surgical excision may be considered when a large painful mass causes significant functional impairment or compression neuropathy.

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