Calcific myonecrosis misdiagnosed as right leg abscess: a case report

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Case presentation

A 63-year-old man attended hospital with a 2-year history of a slow-growing painful mass in his right leg. He had undergone successful surgical drainage of an abscess. Plain X-ray of the right leg at that time (Fig 1a) revealed a longitudinally distributed fusiform mass (29.4 cm×5.1 cm) with plaque-like amorphous calcifications at the right anterolateral aspect. No obvious periosteal reaction or cortical destruction of the tibia or fibula was noted. There was varus deformity of the tibia with medial cortex thickening and angulation that suggested old fracture. He had sustained a crush injury to the right leg 10 years previously and developed peroneal nerve injury with foot drop.

Physical examination of the patient revealed a mass at the anterolateral aspect of the right leg. There were no skin changes, redness or tenderness. The patient was afebrile. Biochemical and haematological investigations were normal. The range of motion of the right lower limb was not impaired but obvious muscle atrophy at the posterolateral compartment of the right leg was noted. Further imaging was requested due to concerns about malignancy.

The plain film of the right leg 2 years later (Fig 1b) showed a similar but smaller calcified mass at the anterior aspect. A 23.4 cm×3.1 cm×4.1 cm corresponding hypoechoic mass with internal and peripheral calcification foci in the muscle layer of the lateral aspect of the same leg and paucity of vascularity was observed under lower extremity sonography examination using Toshiba Aplio 500 (Canon Medical Systems, Tustin [CA], US) [Fig 1c]. Further magnetic resonance imaging study using Discovery MR750 (GE Healthcare, Chicago [IL], US) revealed an elongated and lobulated intramuscular cystic mass with variable internal T1- or T2-weighted signal intensity and low-signal peripheral

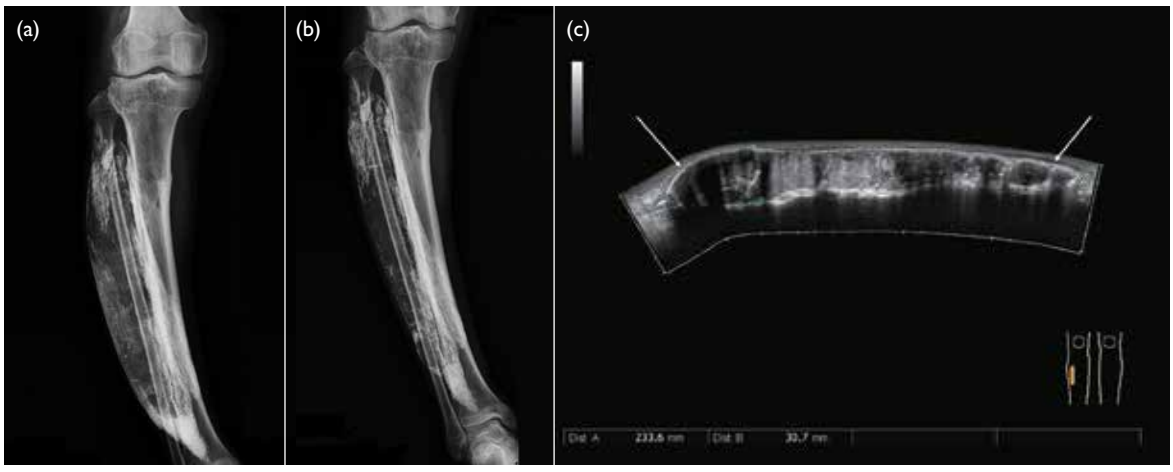

FIG 1. (a) Anteroposterior view of the right leg of the patient at presentation disclosed one longitudinal fusiform-shaped mass (29.4 cm×5.1 cm) at the anterolateral aspect with plaque-like amorphous calcifications and no obvious osteoid or chondroid bone matrix. No associated periosteal reaction and bone density change over the fibula and tibia was noted. There was varus deformity of the tibia with medial cortex thickening and angulation that suggested an old fracture. Two years later, plain film of the right leg (b) showed a similar calcified mass with smaller size at the anterior aspect, while sonography of the same leg (c) showed a 23.4 cm×3.1 cm×4.1 cm hypoechoic mass with internal and peripheral calcification in the muscle layer of the lateral aspect (arrows). Under colour doppler, calcification-related twinkling artifact is depicted without obvious internal or peripheral vascularity (not shown)

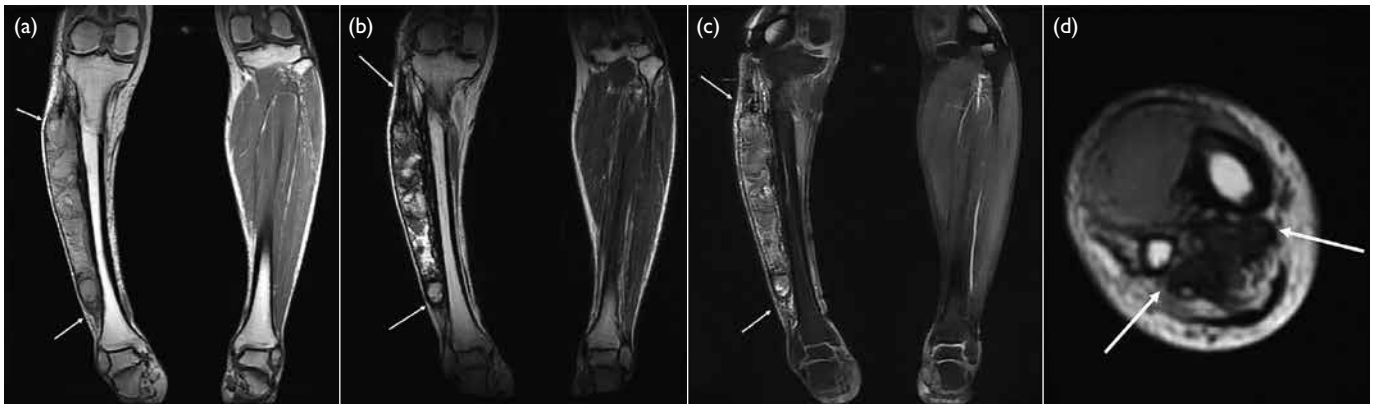

FIG 2. (a) T1- and (b) T2-weighted magnetic resonance imaging of bilateral leg of the patient shows varus deformity with more severe condition on the right side. There is one intramuscular longitudinally elongated and lobulated mass (arrows) at the lateral aspect of the right leg. Internal content with variable T1- and T2-weighted signal intensity implies variable stage haematoma. Thin peripheral low-signal rim under T1- or T2-weighted image is consistent with peripheral calcification under plain radiography. (c) T1-weighted fat-saturated post-gadolinium image shows no obvious internal enhancement, but mild peripheral enhancement is seen (arrows). (d) Axial T1-weighted magnetic resonance image shows severe muscle atrophy with intramuscular fibrosis involving posterior compartments of the right leg (arrows), particularly deep posterior compartment. It is most likely related to ancient post-traumatic compartment syndrome

rim (Fig 2a and 2b). Mild peripheral enhancement after intravenous gadolinium administration was noted (Fig 2c). Additional findings included varus deformity of the right tibia and severe muscle atrophy with muscle fibrosis involving the deep posterior and lateral compartments of the right leg.

The tibial and fibular bones were intact and without involvement. No regional inflammatory change around the calcified cystic mass, prominent vascularity and significant peripheral enhancement was observed. The findings were consistent with calcific myonecrosis and muscle fibrosis, which is most likely related to a remote post-traumatic compartment syndrome (Fig 2d). In the absence of any symptoms that impacted daily living, the patient declined invasive treatment and opted for regular follow-up.

Discussion

Calcific myonecrosis is a rare, delayed manifestation of post-traumatic muscle injury or sequelae of neurovascular injury that occurs almost exclusively in the lower limbs.[1] It is likely due to previous muscle injury complicated by compartment syndrome.[1] Muscle injury may be a result of trauma, nerve injury with denervation, snake bite complicated by compartment syndrome,[2] chronic inflammation (eg, dermatomyositis),[3] or repeated contraction due to static epilepsy.[4]

The most common site of compartment syndrome in the leg is the anterior compartment followed by the lateral and deep posterior compartment. Compartment syndrome results in chronic vascular insufficiency and ischaemic change to muscular tissue. Following long-term hypoperfusion, muscles become necrotic and fibrosed with a consequent reduction in mass and development of contracture. If extensive necrosis with cystic change, haematoma formation, and calcification are present, calcific myonecrosis may develop, evident as a slow-growing mass.

In most cases, calcific myonecrosis presents as a painful or painless mass. Pain is likely due to pressure on surrounding tissue. Signs of inflammation (skin erythema, tenderness, and warmth) are often absent. Cosmetic problems and concerns of malignancy (eg, soft tissue sarcoma) are common reasons for seeking medical advice after decades of the disease course.

Plain radiography and computed tomography usually reveal lesions confined to muscle compartments in a longitudinally oriented pattern with peripheral fusiform and amorphous calcifications parallel to muscle orientation. Magnetic resonance imaging usually shows a circumscribed fusiform intramuscular cystic mass with the peripheral calcification rim in the longitudinally oriented pattern as muscle bulk. The internal content usually shows variable signal intensities in T1- and T2-weighted images that depend on the internal content (proteinaceous necrotic fluid, serous fluid, and different blood stages). Mild peripheral rim enhancement may be observed following intravenous gadolinium administration. The central cystic part lacks enhancement secondary to extensive necrosis. Post-compartment syndrome–related muscle

atrophy, fibrosis, and contracture usually coexist in these disease entities.[1] Other common differential diagnoses should be excluded including myositis ossificans, soft tissue sarcoma, and musculoskeletal tuberculosis.[5,6]

Calcific myonecrosis is essentially benign although previous reports have revealed postoperative complications such as sinus tract formation,[7] poor wound healing, and secondary infection. This implies that wound healing is difficult, probably due to previous compartment syndrome and chronic microvascular insufficiency. In cases of uninfected calcific myonecrosis, observation is the recommended management given the high risk of postoperative complications. Nonetheless cosmetic problems and symptoms may persist.[1] For calcific myonecrosis with infection, conservative management with wound dressing and antibiotics is usually not sufficient. Extensive debridement, removal of diseased tissue, and flap coverage are recommended.[8] Secondary surgery may be needed if postoperative complications (poor wound healing, secondary infection, and sinus tract formation) are observed. Advanced wound management (eg, negative pressure wound therapy) facilitates healing with a moist environment and topical negative pressure.[8,9]

In our patient, the calcified mass in his right leg 2 years previously had been drained since abscess was suspected. Plain X-ray at that time suggested calcific myonecrosis or other differential diagnoses (myositis ossificans, soft tissue sarcoma, etc.). Thus, further magnetic resonance imaging surveys and more details about a previous crush injury would have been helpful to reach a diagnosis and avoid unnecessary surgery. Knowledge of calcific myonecrosis is important for clinicians and radiologists to optimise patient care.

Conclusion

Calcific myonecrosis is a benign disease process. Generally, 'do not touch' is a treatment strategy. Prompt recognition, proper diagnosis based on imaging, and detailed history taking are important.

Author contributions

Concept or design: CH Lin.
Acquisition of data: CY Lee.
Analysis or interpretation of data: CY Lee.
Drafting of the manuscript: CY Lee.
Critical revision of the manuscript for important intellectual content: Both authors.

Both authors had full access to the data, contributed to the study, approved the final version for publication, and take responsibility for its accuracy and integrity.

Conflicts of interest

Both authors declared no conflicts of interest.

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Ethics approval

The patient was treated in accordance with the Declaration of Helsinki and has provided informed consent for the treatment/procedures, and consent for publication.

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