Heterotopic ossification in right popliteal fossa causing arterial insufficiency

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ABSTRACT

Heterotopic ossification (HO) is the abnormal formation of extra-skeletal bone in soft tissue, which can occur after trauma or surgery. HO in joints can cause pain, hinder mobility, and compress surrounding nerves and blood vessels. We present an unusual case of arterial insufficiency caused by HO in the right popliteal fossa. (J Vasc Surg Cases Innov Tech 2023;9:101360.)

Keywords: Arterial insufficiency; Heterotopic ossification; Popliteal artery; Popliteal fossa; Vascular compression

Heterotopic ossification (HO) is a pathologic condition in which abnormal bone formation occurs within the soft tissue outside the normal skeleton.[1] The causes of HO include genetic bone disorders, musculoskeletal trauma, spinal cord and central nervous system injury, and a complication after surgery.[2,3] Although the exact pathophysiology of HO is unknown, it is hypothesized to be due to repeated microtrauma. This leads to the accumulation of mesenchymal spindle stem cells, which eventually transform into osteoblasts.[4] The symptoms of HO are often related to impingement of the surrounding structures, including deep vein thrombosis, neurogenic pain, joint impingement, and, rarely, arterial insufficiency.[5,6] We report an unusual case of HO causing significant arterial insufficiency secondary to popliteal artery compression. The patient provided written informed consent for the report of his case details and imaging studies.

CASE REPORT

A 63-year-old male patient presented in July 2020 for vascular consultation for evaluation of right knee pain, after a referral from orthopedic surgery. He had a history of hypercoagulability with prior deep vein thrombosis. The patient was experiencing pain due to a compressive mass in the popliteal fossa. Magnetic resonance imaging ordered by the orthopedic surgery team in June 2020 showed a 3-cm nidus of HO impinging on the popliteal artery and vein (Fig 1). The patient underwent duplex ultrasound, which showed mild compression of the popliteal artery on knee extension, causing 50% to 74% stenosis with dorsiflexion, with a normal ankle brachial index (ABI). His physical examination at his initial presentation showed warm and well-perfused lower extremities, with palpable femoral popliteal and pedal pulses. There were no changes with active plantar flexion or passive dorsiflexion. After the patient was informed, he elected for conservative management, given his overall mild symptoms. Approximately 2 years after his first vascular surgery appointment, he returned for follow-up evaluation of new claudication symptoms but denied rest pain or edema. The physical examination showed a new, obvious deformity of the right popliteal fossa compared with the physical examination findings at his initial presentation. The area of the right popliteal fossa was firm to palpation. Physiologic testing was performed at the follow-up evaluation, including ABIs, segmental limb pressures, and pulse volume recordings (Fig 2). The patient had an ABI of 0.55, without palpable pulses. Arterial compression from the HO was further reflected by the damping of waveforms below the level of the knee. Repeat arterial testing showed progression of the popliteal artery impingement, with blunting of the waveforms distally. After discussion with the patient, surgical resection was planned for November 2022.

The patient's popliteal fossa was exposed via a posterior approach (Fig 3) with a standard "lazy S" incision. The popliteal vein was identified first, and all the small branches along the lateral aspect of the vein were dissected and ligated, allowing for retraction of the vein medially to obtain exposure of the deeper popliteal artery. After the tibial nerve was carefully preserved, the 4 × 2-cm heterotopic ossified lesion was successfully dissected in its entirety (Fig 4) off the popliteal artery, taking care to not injure any of the neurovascular structures. The artery was noted to be healthy in appearance, without obvious signs of damage. There was an obvious plane between the HO and the artery, and it was easily shelled out. Hemostasis was achieved, and the distal extremity was checked for palpable pulses, which were present just as before surgery. His postoperative course was unremarkable, and his duplex ultrasound showed normal popliteal and tibial artery anatomy without compression. He was discharged from the hospital after 1 day with a prescription for rivaroxaban. One week after the procedure, the patient's noninvasive testing showed resolution of the arterial compression,

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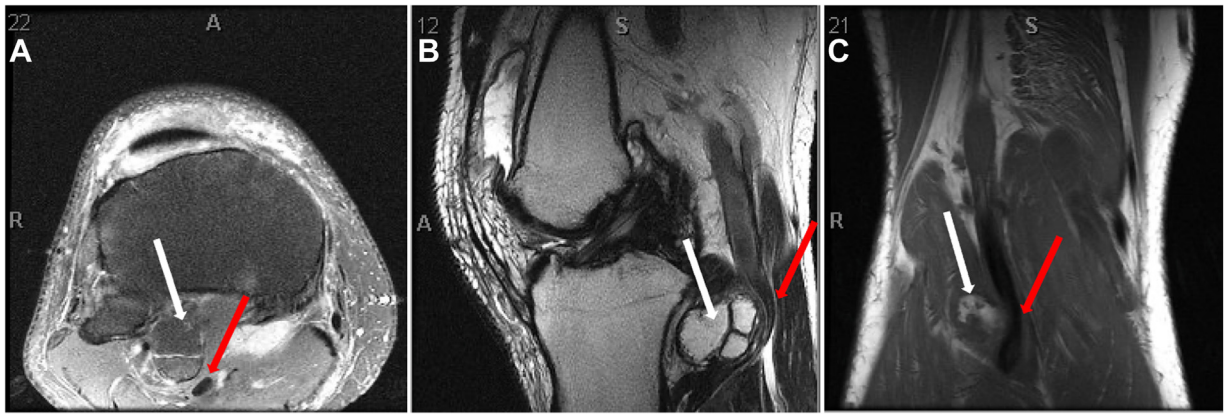

Fig 1. Axial proton-density fast-spin echo fat-suppressed **(A)**, sagittal T2-weighted magnetic resonance image **(B)**, and coronal T1-weighted fast spin echo **(C)** magnetic resonance images of the lower extremity, showing three large ossicles in the popliteus tendon sheath, measuring ≤3 cm anteroposteriorly. The ossicles (*white arrows*) resulted in deformity of the popliteal artery (*red arrows*) with a mildly narrowed lumen.

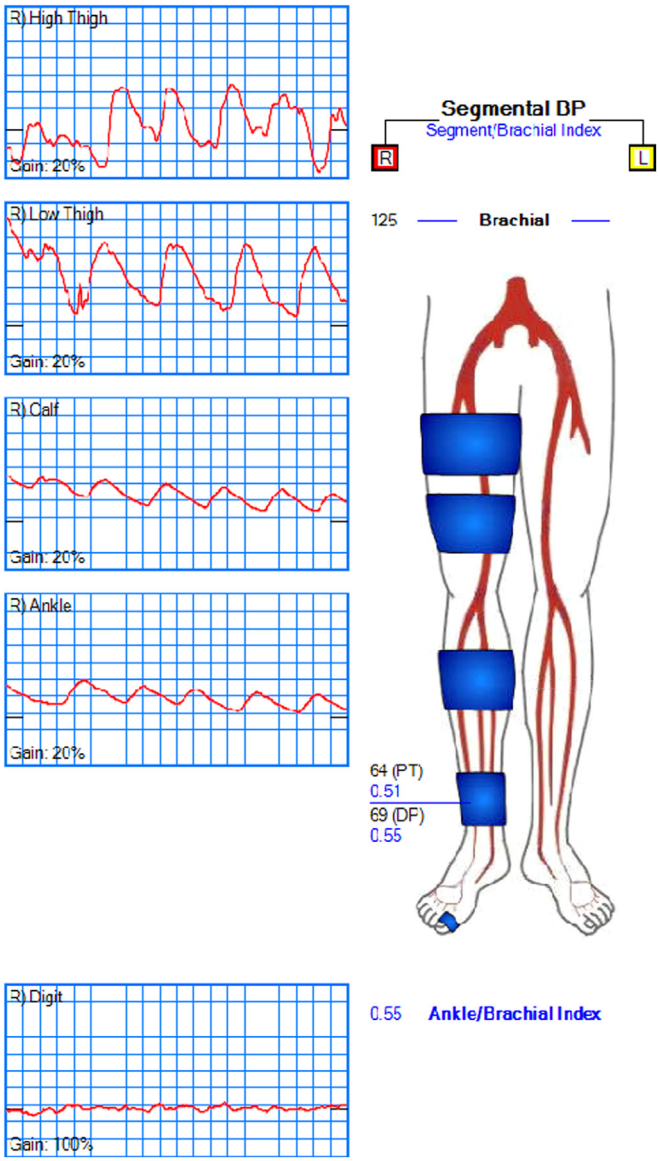

Fig 2. Ankle brachial indexes (ABIs), segmental limb pressures, and pulse volume recordings, showing a low ABI of 0.55, with dampening of the waveforms below the level of the knee due to compression from the heterotopic ossification (HO). *DP,* Dorsalis pedis; *L,* left; *PT,* posterior tibial; *R,* right.

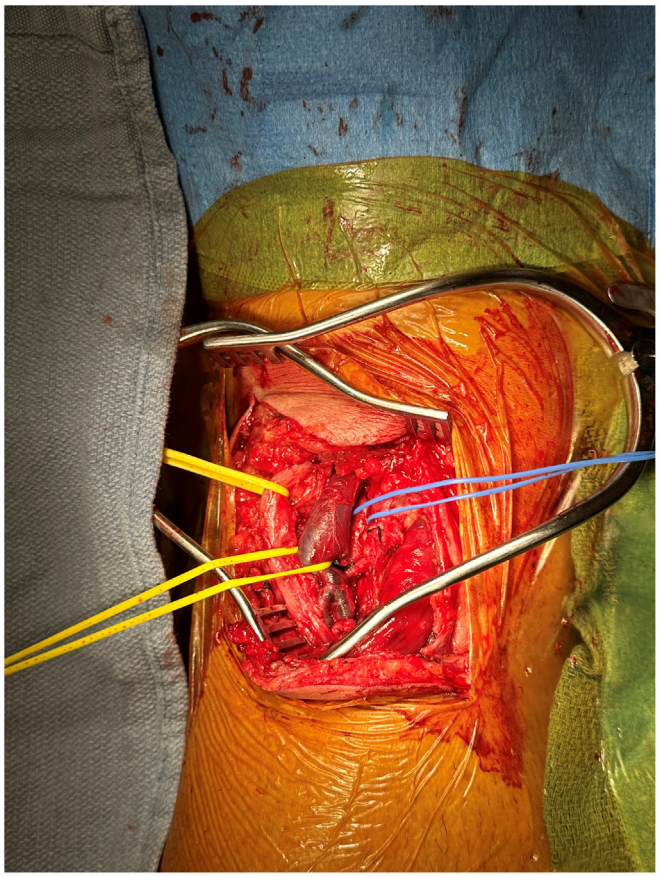

Fig 3. Photograph of the surgical incision. A posterior approach to the popliteal artery was taken with a "lazy S" incision over the popliteal fossa.

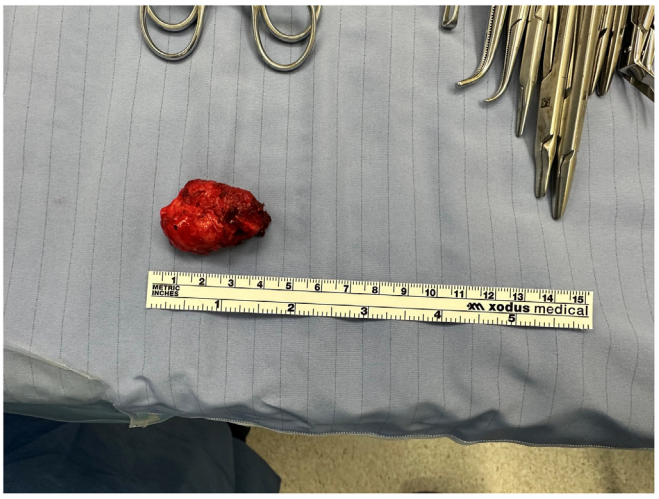

Fig 4. Photograph of the heterotopic ossification (HO), an unoriented, 3.9 × 1.9 × 2.8-cm portion of tan-red, calcified tissue encased in a minimal amount of red-tan, focally cauterized soft tissue.

with a measured ABI of 0.94 and normal arterial waveforms distally. His magnetic resonance imaging study in March 2023 indicated that the previous HO in the popliteal space was completely excised. No new ossicles posteriorly in the popliteal fossa or along the popliteus were noted. The patient has been doing well, with complete resolution of his claudication. In addition, he had improvement of his ABI to 0.97, although he still experiences knee pain from his arthritis.

DISCUSSION

HO is an aberrant bone formation in nonosseous tissue found most often between muscle planes. Specific sites are more susceptible to HO, such as the brain, hip, elbow, and spinal cord.[7] The underlying pathophysiology surrounding HO's predilection for these regions is not well understood. The most common clinical presentation is an acquired singular lesion in a young adult, with preceding localized trauma identified in ~75% of cases.[7] Other risk factors include male sex, previous surgical manipulation, orthopedic injury, spinal cord injury, thermal injury, and neurogenic or metabolic conditions.[8] Rare hereditary forms exist, such as fibrodysplasia ossificans progressiva and progressive osseus heteroplasia.[4] In both forms, the usual presentation involves multiple HO lesions in the soft tissues beginning in childhood.[9] Previous case reports have illustrated HO lesions in unusual locations such as the patellar tendon and calcaneal region, both cases also without obvious risk factors or genetic predisposition.[10,11] Complications of HO include decreased mobility, nerve entrapment, vascular compression, pressure ulcers, ankylosis, and lymphedema, although these have been more extensively studied in the setting of traumatic neurologic injury or after joint replacement.[4,12] Treatment of HO is typically conservative with physical therapy, nonsteroidal anti-inflammatory drugs, or bisphosphonates. However, the use of bisphosphonates has been primarily studied in the context of HO occurring after burns or spinal cord injury.[13] Surgical excision becomes necessary for the treatment of symptomatic HO that has failed conservative treatment, as was the case for the present patient.

To the best of our knowledge, this is an uncommon case of HO occurring in the popliteal fossa and causing arterial compression in a patient devoid of risk factors apart from male sex, without prior trauma or surgery to the area. We further highlight the importance of identifying arterial compression secondary to HO. We have previously encountered another case of HO in our institution that caused severe bleeding in the thigh and required endovascular covered stent placement to address the bleeding. This provides anecdotal evidence regarding using endovascular methods to potentially address any urgent bleeding issues with HO and arterial compression if the patient is too unstable for open surgical intervention.

CONCLUSIONS

Idiopathic HO is a rare clinical entity. We report a case of popliteal HO causing worsening arterial insufficiency secondary to popliteal artery impingement. Although most cases of HO can be treated conservatively, aggressive management in the present case prevented progression of our patient's arterial insufficiency.

DISCLOSURES

None.

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