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Case report

Computed tomography angiography and magnetic resonance imaging performance of acute segmental single compartment syndrome following an Achilles tendon repair: A case report and literature review

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ABSTRACT

Acute compartment syndrome of the lower extremity is a serious postinjury complication that requires emergency treatment. Early diagnosis is of paramount importance for a good outcome. Four muscle compartments in the calf (anterior, lateral, deep posterior, and superficial posterior) may be individually or collectively affected. Acute segmental single-compartment syndrome is an extremely rare condition characterized by high pressure in a single compartment space with threatening of the segmental tissue viability. In this case report, we describe a young man with Achilles tendon rupture who complained of postoperative pain in the anterior tibial region. Emergent computed tomography angiography and magnetic resonance imaging revealed local muscle edema. Segmental anterior compartment syndrome was diagnosed and fasciotomy was performed.

Introduction

Acute compartment syndrome (ACS) is defined as high pressure in a closed compartment resulting in decreased capillary blood perfusion.[1] When this high pressure lasts for hours, the circulation and normal function of tissues such as muscles and nerves are disturbed, leading to myoneural necrosis. Muscles and nerves tolerate ischemia for up to 4 h with limited sequelae; however, 8 h of ischemia would result in irreversible damage.[2,3] An internally expanding or externally compressive force can lead to compartment syndrome. Early recognition is critical for successful treatment. Therefore, timely and effective decompression, called fasciotomy, is essential.[4] The most common causes of ACS are orthopedic conditions, such as fractures and fracture surgery, especially tibial diaphyseal fracture.[5–7] McQueen[8] reported that 23% of ACS cases are caused by soft tissue injuries without fracture. In addition, vascular and iatrogenic conditions, and occasionally conditions such as snakebite, can lead to ACS.

Increased intracompartmental pressure (ICP) reduces capillary perfusion, which eventually leads to arteriolar compression and subsequent tissue ischemia. Ischemia-reperfusion and exudation of fluid result in further increases in ICP, developing a vicious cycle.[9] The ultimate outcomes are muscle infarction and nerve damage.[10] The calf contains four compartments (anterior, lateral/peroneal, deep posterior, and superficial posterior). The anterior compartment of the lower leg contains the tibialis muscles and extensors of the toes and is the space in which ACS most frequently occurs.

Timely diagnosis is often difficult because ACS can be caused by minimal trauma or masked by concomitant trauma. A delay in diagnosis leads to delayed treatment and can result in ischemic contracture and severe disability. Therefore, early d 3etection is of pivotal importance. Clinical symptoms and physical signs should be considered in combination for diagnosis,[11] and physical examination findings are particularly important.[12] In ACS of the calf, for example, patients often report pain that is out of proportion to the injury of the anterior tibialis. The deep peroneal nerve lies in the anterior compartment; therefore, the sensory territory over the first web space is deficient, and pain in the corresponding toes occurs during passive plantar flexion. Distal pulses are sometimes weak or absent. However, the “five ps” (pain, pallor, paresthesia, paralysis, and pulselessness) are usually very late signs. When all of these symptoms, especially pulselessness, have developed, the

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condition is irreversible.[13] A high index of suspicion and awareness of the risk of the syndrome may reduce delays in diagnosis.

No gold standard for diagnosing ACS exists. ICP monitoring can facilitate diagnosis based on clinical symptoms and physical examination; it has shown 94% sensitivity, 98% specificity, 93% positive predictive value, and 99% negative predictive value for the diagnosis of ACS.[14] No study to date has reported the performance of imaging modalities in the diagnosis of ACS. Once ACS is diagnosed, urgent treatment should be undertaken. No time is left for imaging examination, and it is thus difficult to obtain imaging data in patients with ACS.

We herein report a case of ACS in a single compartment in which segmental tissue viability threatened the lower extremities following surgical repair of a ruptured Achilles tendon. The diagnosis was almost delayed; fortunately and coincidentally, we obtained valuable computed tomography angiography (CTA) and magnetic resonance imaging (MRI) information in the diagnostic process. We would like to increase awareness and describe the radiographic findings of this uncommon clinical presentation. To our knowledge, this report is the first to provide complete CTA and MRI information for the diagnosis of ACS. The patient provided informed consent to the publication of data concerning his case.

Case report

The patient was a 24-year-old man with a height of 169 cm and weight of 65 kg (body mass index, 22.76 kg/m^2). He was admitted to our Orthopaedic Emergency Department with a 1-day history of pain in the right heel. He felt a sudden “pop” in his right ankle region while trying to turn a somersault. He was a martial arts actor with more than 6 years of experience. Inspection revealed slight swelling along the right heel, but no skin change or ecchymosis. Palpation revealed discontinuation with a palpable defect of the Achilles tendon 4 cm above the calcaneal tuberosity. Physical examination showed that the patient had difficulty with active plantar flexion of the right foot, and passive dorsiflexion was painful. He had easily palpable dorsal pedis and posterior tibial pulses. Motor and sensory testing of his right leg yielded normal results. Another institution's roentgenograms (X-rays) were negative for fracture. Therefore, we performed an MRI examination, which revealed right Achilles tendon rupture (Fig. 1). The patient was admitted to our hospital ward for surgical treatment.

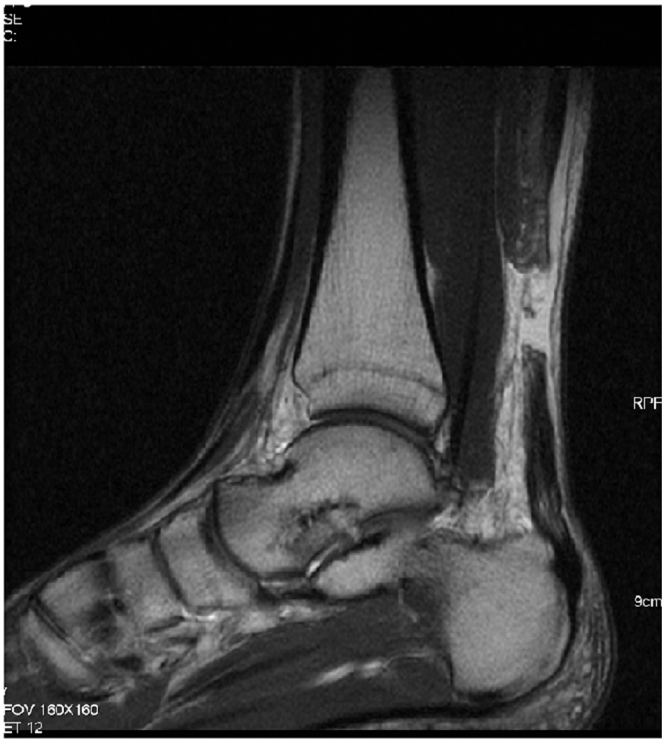

Fig. 1. MRI of Achilles's tendon rupture. MRI appearance of Achilles tendon rupture represents Achilles tendon thickening, hyperintensity in tendon, completely discontinuous fibrous bundle.

Surgical repair of the Achilles tendon rupture was performed 3 days after all preoperative examinations had been completed. After the administration of preoperative antibiotics and general anesthesia, the patient was placed in a prone position. A pneumatic tourniquet was placed on the proximal right thigh during the operation. The tourniquet was inflated to 60 kPa for a total of 57 min. A 10-cm incision was made on the medial aspect of the Achilles tendon, centered over the palpable defect. A 5-# Ethibond suture (Ethicon, Shanghai, China) was used to repair the tendon using the Kessler or Krakow suture technique. The two ends of the tendon were then reinforced with a 2-0 absorbable suture (Vicryl, Ethicon) under no tension. Another 2-0 absorbable suture was used to suture the plantar tendon over the two ends of the Achilles tendon using the Lynn technique. The paratenon and subcutaneous tissues were closed using a 2-0 absorbable suture. The skin was closed using an ordinary 4-0 silk suture. The operation was successfully completed, and an anterior short leg cast was applied to maintain the lower leg in a neutral position. The splint was wrapped loosely with a bandage.

The patient's condition was stable until he complained of pain over the anterior tibialis and around the incision approximately 5 h after surgery. This condition was considered to represent normal postoperative pain at that time. Despite the administration of a 100-mg dose of oral tramcontin, the patient continued to feel mild to moderate pain. He required additional analgesia (40 mg parecoxib) 7 h postoperatively. During rounds the next day, we replaced the anterior short leg cast with a back short leg cast, which eased the patient's pain. Physical examination revealed local mild tension and swelling in the right pretibial region. He denied numbness or tingling of the toes. Neurovascular examination findings were normal. The patient was able to perform active plantar flexion and dorsiflexion of all toes without excessive pain. However, approximately 27 h postoperatively, the patient complained of pain again in the anterior aspect of the lower leg without numbness or toe activity pain. He was given another 40 mg parecoxib intravenously.

Two days later, approximately 38 h after surgery, the patient's symptoms seemed to progress to intense pain over the anterior aspect of the lower leg. He experienced weakness in dorsiflexion of the great toe, and plantar flexion of the toes aggravated the pain. Paresthesia was found in the first toe web area. We considered the possibility of a thrombus in the lower leg. We then performed emergent MRI and CTA, which revealed swelling of the extensor hallucis longus (EHL) and extensor digitorum longus (EDL) with distal anterior tibial artery and peroneal artery stenosis (Figs. 2–4). Acute ACS was diagnosed. Fasciotomy for decompression was emergently performed. During the operation, after a 10-cm incision of the skin was made and the subcutaneous tissue was sharply dissected down to the level of the fascia, tension over the anterior compartment was found. The fascia over the other compartments was normal. Upon fascial incision, the EHL and EDL immediately and prominently herniated through the fascia, and part of the muscle was ischemic and edematous. The sectional muscle's color appeared ashen gray relative to the

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