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Chinese Journal of Traumatology

journal homepage: <http://www.elsevier.com/locate/CJTEE>

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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ARTICLE INFO

Article history:

Received 23 November 2015

Received in revised form

2 March 2016

Accepted 30 March 2016

Available online 13 May 2016

Keywords:

Compartment syndromes

Achilles tendon

Tibial fractures

Angiography

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.

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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} McQueen⁸ 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 detection 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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Peer review under responsibility of Daping Hospital and the Research Institute of Surgery of the Third Military Medical University.

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/m²). 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.

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



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.

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 paracetamol) 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 paracetamol 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

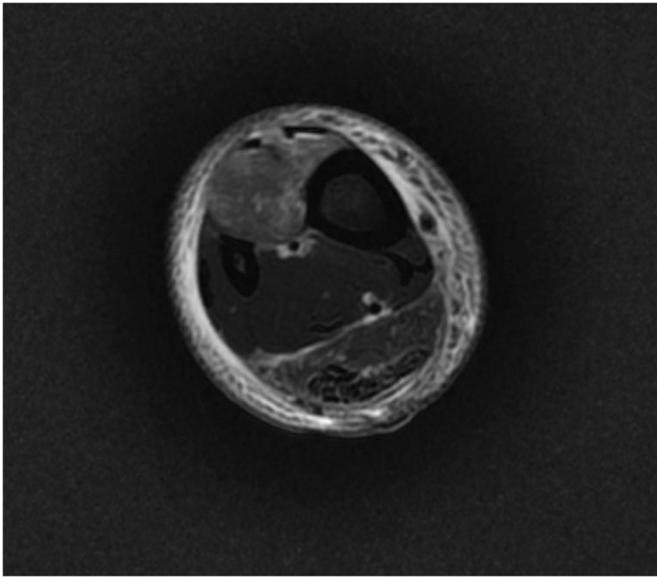


Fig. 2. Coronal MRI of right lower limb shows enhanced signal and swelling of pre-tibial area, limiting in the anterior compartment.

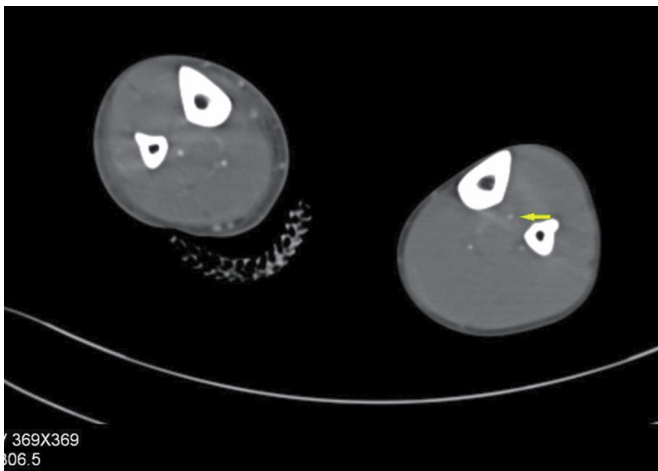


Fig. 3. Coronal CTA shows occlusion of left limb tibial artery, the yellow arrow displays left tibial artery, comparing with the right one.



Fig. 4. CTA 3D reconstruction displays occlusion of right anterior tibial artery and peroneal artery.

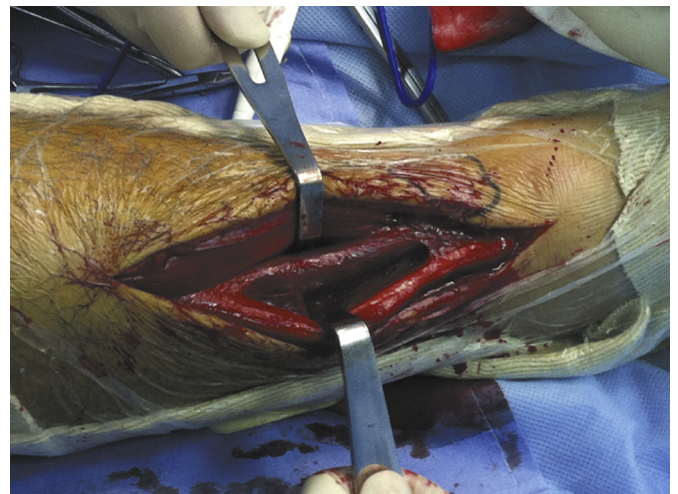


Fig. 5. Operation of fasciotomy shows section of EHL and EDL necrosis with ashen gray in color corresponding with MRI performance.

others, corresponding with the MRI findings. No contraction of the EHL or EDL occurred upon stimulation with a diathermy needle (Fig. 5). Within a few minutes of compartment release, the color of the EHL and EDL muscle bellies had improved, but to a minimal extent. Finally, the wound surface was temporarily closed with negative-pressure wound therapy. The patient's pain was relieved immediately after surgery, and sensation in the first toe web area was slightly improved. However, he was still unable to perform active dorsiflexion of the first toe. One week later, the fasciotomy wound was closed. Intraoperatively, the EHL and EDL muscle bellies were found to be slightly gray and showed no reaction to the diathermy needle (Fig. 6).

The patient recovered well and was discharged home 3 days later in a plaster back slab. He had no pain at the 1-month postoperative visit to our Orthopaedics Clinic. His numbness resolved completely, and dorsiflexion of the first toe was moderately improved. Extension and dorsiflexion strength of the EHL and EDL were rated 4+/5. The patient was instructed to

remove the plaster and train the first toe. He returned 3 months later, at which time he had recovered well, with no motor or sensory deficit in the right lower extremity. He walked with no discomfort. After 6 months, he could perform some light running and jumping activities.

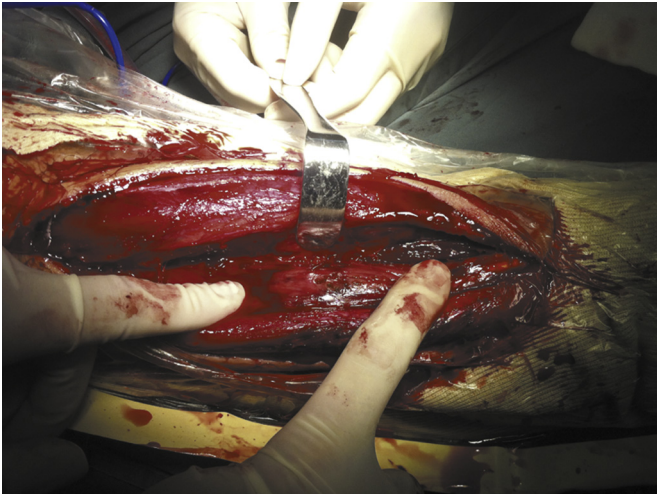


Fig. 6. The operation for fasciotomy wound close. The muscle belly of EHL and EDL are slightly gray in color and no reaction to electrical stimulation.

Discussion

ACS is usually caused by traumatic injuries, such as extremity fracture, crush injury, prolonged tourniquet use under anesthesia, compression by casts, burns, hematoma, anticoagulation therapy, or, in chronic compartment syndrome, exercise.¹⁵ The anterior compartment is the most frequently involved site.¹⁶ Swelling and tenderness are usually present over the tibialis anterior muscle. Patients complain of pain over the anterior region of the lower leg and feel weakness when trying to pull the foot upward against resistance. Pain occurs when the foot and toes are bent downward. Our case involved an extremely rare condition that occurs only in segmental ACS. Furthermore, no obvious preoperative traumatic factor was found that could have resulted in ACS.

Soft-tissue injuries and muscle tears occur frequently in athletes.^{17,18} Muscle swelling can reportedly also result from hematoma due to minor injury.¹⁹ In this case, the patient was trying to turn a somersault when his right heel was injured. His anterior tibial muscle might have been strained during sports activities, causing a buildup of pressure in the anterior compartment. Muscle swelling may have been delayed by increased muscle ischemia. On the other hand, he had practiced martial arts for more than 6 years, and his muscles were strong with large muscle volumes, while his compartment size was limited. Chronic exertional compartment syndrome is commonly seen in physically active military service members and athletes.^{20,21} Affected individuals have recurrent, activity-related leg pain of a consistent duration or intensity that is relieved only by rest. As the interval between injury and surgery was only 3 days in our patient, we cannot rule out the possibility of an acute episode of chronic exertional compartment syndrome.

Some authors have considered ACS to be related to the operative position, especially when the muscles are compressed, as in the lithotomy position.¹² In our case, a prone position was adopted. We used a pneumatic tourniquet around the proximal leg and placed soft surgical linen under the ankle; these procedures were performed carefully and checked by several members of the surgical team. We observed no marked compression force on the anterior tibialis during the operation. The operation time was 57 min, within the permissible range of lower limb tourniquet application. In this patient, ACS appeared to have a minor relationship with the operative events. Thrombi and vasospasm resulting from the use of a tourniquet have been implicated as etiologies of compartment

ischemia.²² Therefore, based on the patient's muscular factors and the mechanism of ischemia-reperfusion injury, the tourniquet increased the risk of muscle ischemia and swelling, resulting in elevated pressure in the compartment.

An anterior short leg cast was applied to this patient to maintain the lower leg in a neutral position after surgery. Cast removal can reportedly decrease pressure in the anterior and deep posterior compartments.²³ Masten²⁴ considered venous obstruction aroused by a long leg brace to be an etiology of anterior tibial compartment syndrome. We replaced the anterior short leg cast with a posterior short leg cast, but the symptoms did not improve. However, external pressure secondary to the cast must not be dismissed as a possible cause. Previous studies have found that casts potentially restrict compartment expansion and decrease venous flow, ultimately resulting in ACS.^{25,26} Throughout this patient's clinical course, we speculated that the use of the cast was the main factor related to the onset of ACS.

ICP monitoring is an important diagnostic approach for ACS. Controversy exists about the identification of critical factors leading to muscle ischemia or tissue necrosis. Many researchers have proposed an ICP threshold of 30 mmHg as an indication for fasciotomy.²⁷ In modern medicine, MRI has a wide variety of diagnostic applications. Compared with the invasiveness of ICP monitoring, MRI can precisely demonstrate the region of swelling and provide accurate guidance for the operation. In our case, CTA and MRI results were obtained incidentally. Thus, MRI or CTA findings should be taken into account if conditions permit. Because ACS was considered with the help of radiographic information, we did not monitor the ICP to gain time to surgery. In most cases of compartment syndrome, no time is available for the acquisition of MRI data. Muscle edema caused by ACS is sometimes difficult to distinguish from that caused by trauma. Although some authors consider that MRI is not useful and may even be detrimental because of the resultant delay in surgical treatment, it aided the diagnosis of this rare condition in the present case. Therefore, it was indeed fortunate that CTA and MRI data were obtained in our case.

To sum up, acute segmental single-compartment syndrome is a rare but serious complication encountered in several fields of medicine, especially orthopedics, sports medicine, and traumatology. High awareness must be maintained at all times when patients complain of pain in the extremities. Our report presented the detailed imaging data used for the diagnosis of ACS in a patient with extremity pain. A literature review found no such detailed information for ACS in previous reports. Our goals are to provide information that will aid the development of additional diagnostic methods and highlight the importance of vigilance for ACS.

References

- Matsen 3rd FA, Winquist RA, Krugmire Jr RB. Diagnosis and management of compartmental syndromes. *J Bone Jt Surg Am.* 1980;62:286–291.
- Whitesides TE, Heckman MM. Acute compartment syndrome: update on diagnosis and treatment. *J Am Acad Orthop Surg.* 1996;4:209–218.
- Mausser N, Gissel H, Henderson C, et al. Acute lower-leg compartment syndrome. *Orthopedics.* 2013;36:619–624.
- Jepson P. Ischemic contracture: experimental study. *Ann Surg.* 1926;84:785–795.
- Kostler W, Strohm PC, Sudkamp NP. Acute compartment syndrome of the limb. *Injury.* 2004;35:1221–1227.
- Elliott KG, Johnstone AJ. Diagnosing acute compartment syndrome. *J Bone Jt Surg Br.* 2003;85:625–632.
- Shadgan B, Menon M, Sanders D, et al. Current thinking about acute compartment syndrome of the lower extremity. *Can J Surg.* 2010;53:329–334.
- McQueen MM, Gaston P, Court-Brown CM. Acute compartment syndrome. Who is at risk? *J Bone Jt Surg Br.* 2000;82:200–203.
- Erdos J, Dlaska C, Szatmary P, et al. Acute compartment syndrome in children: a case series in 24 patients and review of the literature. *Int Orthop.* 2011;35:569–575.
- Gourgiotis S, Villias C, Germanos S, et al. Acute limb compartment syndrome: a review. *J Surg Educ.* 2007;64:178–186.

11. Singh S, Trikha SP, Lewis J. Acute compartment syndrome. *Curr Orthop*. 2004;18:468–476.
12. Ikeya E, Taguchi J, Ohta K, et al. Compartment syndrome of bilateral lower extremities following laparoscopic surgery of rectal cancer in lithotomy position: report of a case. *Surg Today*. 2006;36:1122–1125.
13. Mubarak SJ, Hargens AR. Acute compartment syndromes. *Surg Clin North Am*. 1983;63:539–565.
14. McQueen MM, Duckworth AD, Aitken SA, et al. The estimated sensitivity and specificity of compartment pressure monitoring for acute compartment syndrome. *J Bone Jt Surg Am*. 2013;95:673–677.
15. Tzioupis C, Cox G, Giannoudis PV. Acute compartment syndrome of the lower extremity: an update. *Orthop Trauma*. 2009;23:433–440.
16. Ojike NI, Roberts CS, Giannoudis PV. Compartment syndrome of the thigh: a systematic review. *Injury*. 2010;41:133–136.
17. Best TM. Soft-tissue injuries and muscle tears. *Clin Sports Med*. 1997;16:419–434.
18. Williams P, Shenolikar A, Roberts RC, et al. Acute non-traumatic compartment syndrome related to soft tissue injury. *Injury*. 1996;27:507–508.
19. Aliano K, Gulati S, Stavrides S, et al. Low-impact trauma causing acute compartment syndrome of the lower extremities. *Am J Emerg Med*. 2013;31:890.
20. George CA, Hutchinson MR. Chronic exertional compartment syndrome. *Clin Sports Med*. 2012;31:307–319.
21. Davis DE, Raikin S, Garras DN, et al. Characteristics of patients with chronic exertional compartment syndrome. *Foot Ankle Int*. 2013;34:1349–1354.
22. Reed J, Hiemstra LA. Anterior compartment syndrome following an Achilles tendon repair: an unusual complication. *Clin J Sport Med*. 2004;14:237–241.
23. Weiner G, Styf J, Nakhostine M, et al. Effect of ankle position and a plaster cast on intramuscular pressure in the human leg. *J Bone Jt Surg Am*. 1994;76:1476–1481.
24. Matsen 3rd FA. Compartmental syndrome. An unified concept. *Clin Orthop Relat Res*. 1975;113:8–14.
25. Olson SA, Glasgow RR. Acute compartment syndrome in lower extremity musculoskeletal trauma. *J Am Acad Orthop Surg*. 2005;13:436–444.
26. Younger AS, Curran P, McQueen MM. Backslabs and plaster casts: which will best accommodate increasing intracompartmental pressures? *Injury*. 1990;21:179–181.
27. Zweifach SS, Hargens AR, Evans KL, et al. Skeletal muscle necrosis in pressurized compartments associated with hemorrhagic hypotension. *J Trauma*. 1980;20:941–947.