Case Report

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Website: www.joas.org.in

DOI: 10.4103/joas.joas_19_18

Acute compartment syndrome of the foot: An unusual case caused by Graston technique

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Abstract:

The purpose of this case report is to describe an unusual cause of compartment syndrome of the foot that developed subsequent to the use of Graston technique in a patient who had recently underwent revision total hip arthroplasty (THA). A 60-year-old female presented for orthopedic follow-up care in the office, 10 days postoperatively following an explant of her right hip Prostalac® spacer (DePuy) for previous infection and conversion to right THA (DePuy CORAIL® Hip System) complaining of severe right foot pain, swelling with pressure in foot, paresthesia, and inability to bear weight. The patient had undergone Graston technique for bilateral foot swelling by her home physical therapist. The patient was clinically diagnosed with acute compartment syndrome (ACS) of her right foot and underwent intervention. The treatment consisted of acute fasciotomy of her right foot with vacuum-assisted closure application. Delayed closure was initiated once soft-tissue decompression occurred. Due to acute fasciotomy, ambulation was inhibited and physical therapy delayed. The postoperative course was uneventful following the release of tense compartments, weight bearing was resumed once incisions healed, and the patient was pain free while deep-venous thrombus prophylaxis was continued. No further issues occurred postoperatively, and the patient showed overall clinical improvement. Residual soft swelling can be expected following hip surgery, especially at the caudal end of limbs. Immobilization enhances this engorgement. However, due to the aggressive Graston technique performed by the therapist, the patient developed ACS of the foot. Graston technique has been documented to increase blood flow to treated areas. Early and aggressive intervention should be initiated if ACS is clinically or objectively found, as late detection may have disastrous results.

Keywords:

Compartment syndrome, Graston technique, level of evidence – 5

Introduction

Acute compartment syndrome (ACS) is described as a decline in perfusion pressure to tissue within a compartment due to a critical pressure increase in a confined space.[1,2] When Richard von Volkmann first documented this phenomenon in the hand, he considered it a simultaneous paralysis and contracture due to an interruption of blood supply to a local area of muscles.[3] Because the amount of tissue perfusion is directly proportional to the difference between capillary perfusion pressure and interstitial fluid pressure, any osseofascial compartment interstitial pressure increase can lead to arteriolar compression and cause ACS.[4] This can occur from a decrease in compartment volume (e.g., tight cast), an increase in the contents of a compartment (e.g., bleeding), or both.

Compartment syndrome most commonly affects the leg, with tibial shaft fractures being the most common etiology in these documented cases.[5] However, ACS can also easily affect the arm, thigh, buttock, hand, abdomen, and foot.[6-12] Classic signs of compartment syndrome include pulselessness, paralysis, paresthesia, pallor,

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Received: 01 May 2018
Revised: 05 October 2018
Accepted: 05 November 2018
Published: 13 December 2019

How to cite this article: Lands VW, Malige A, Delong WL. Acute compartment syndrome of the foot: An unusual case caused by Graston technique. J Orthop Spine 2019;7:68-71.

and pain.[13] Pain that is out of proportion to the eliciting stimulus or pain on passive stretch of the affected compartment should alert the physician to possible compartment syndrome.[14,15] However, eliciting these subjective symptoms can be hard, especially in unconscious patients and those with regional anesthesia.[16] By the time pulselessness is present, however, it may already be too late to act. Diagnosis of ACS is primarily clinical, but intracompartmental pressure monitoring or noninvasive near-infrared spectroscopy can help confirm the diagnosis.[17,18] An absolute value of 30–45 mmHg or a change in delta pressure <30 mmHg is usually enough to make the diagnosis.[19-21] When ACS is suspected, the treating physician should treat the situation as a surgical emergency. Unless the affected compartment is quickly decompressed, necrosis of the soft tissue can occur, with permanent disability or even death as a possible sequela.[7] Treatment guidelines outline the need to remove any external compressive forces along with maintaining a normotensive state in order to avoid further ischemia to the affected area,[22,23] with fasciotomy being an important course of action if surgically indicated.[24]

This phenomenon usually arises secondary to crush fractures,[25] but many other uncommon causes have been documented.[26-28] In the foot, specifically, calcaneal, Lisfranc, Pilon, and metatarsal/phalangeal fractures have been documented as the most likely instigators.[29-31] However, we present a curious case of ACS in the foot following Graston technique, a patented instrument-assisted therapy aimed at breaking apart scar tissue and fascial restrictions, sometimes even indicated in the treatment of ACS.[32]

Case Report

A 60-year-old female presented to the orthopedic office for routine postoperative care following a revision right total hip arthroplasty (THA) (Depuy-Synthes CORAIL® PINNACLE® Total Hip System, Warsaw, Indiana, USA). The patient was 10 days status post explant of her right hip Prostalac® spacer (DePuy) and conversion to a right THA. During the visit, the patient had a new complaint of right foot pain, swelling, and inability to bear weight. She initially presented to the emergency department for evaluation to rule out deep-venous thrombosis. D-dimer and lower-extremity duplex ultrasound were both negative for the presence of venous thrombi She was discharged and instructed to follow up on an outpatient basis. According to the patient, she had undergone Graston technique to both lower extremities by her home physical therapist twice, most recently 3 days prior for postoperative foot swelling. Graston technique, which was done at physical therapist's discretion, was done using a stainless steel tool to break up the scar tissue using direct manipulation of feet and lower-extremity muscles. The patient denied any foot pain, paresthesias, or discomfort postoperatively, but had some mild dorsal foot swelling, and she is currently taking thrombi prophylaxis for prevention. Physical examination during the visit revealed the patient to have significant dorsal foot swelling with tense compartments, paresthesias, pain with range of motion, and refusal to bear weight. Due to a high index of suspicion of ACS, the patient was taken to the operating room for emergent right foot fasciotomy without the need for compartment pressure measure. The risks versus benefits were discussed and consent was obtained for surgical intervention.

In the operating room, the patient's right lower extremity was appropriately scrubbed and draped in a sterile fashion. After appropriate preoperative pause to review consent, two dorsal foot incisions were created; one medial to the second metatarsal bone and one lateral to the fourth metatarsal bone. After subcutaneous dissection, all the fascial layers were decompressed through the layers of the foot to assure that all medial, central, and lateral compartments were adequately released. Upon release of the fascia, the muscular compartments began to bulge out due to intramuscular edema. Hemorrhagic clots were removed, further reassuring that decompression was appropriate. It was also noted that there may have been a Morel-Lavallee lesion. However, all muscular tissues were viable. After appropriate hemostasis and decompression of all compartments, vacuum-assisted closure (VAC) application was placed to the wound dimensions, with Jacob's ladder technique being used to assist in delayed primary closure. Following the assessment of dressing and stable seal, the patient was extubated and delivered to the postanesthesia care unit in stable condition. General anesthesia was used according to hospital and surgeon practices. The patient was initially instructed to be non-weight bearing while the VAC was in place; however, she was allowed to start physical therapy (concentrating on strengthening and endurance) starting the day of surgery as per the surgeon's protocol. Deep-vein thrombosis prophylaxis was started for a 28-day course with Lovenox®. Pain control was achieved using a combination of oral and intravenous agents. On postoperative day 3, irrigation and debridement was repeated, and the patient was able to undergo delayed closure due to decrease in muscle swelling and 48–72 h recommendations for VAC changes. Following closure, the patient's weight bearing status was changed to 50% partial weight bearing. The patient was able to be discharged on postoperative day 5. The postoperative course was unremarkable, without reoccurrence of symptoms. Full weightbearing was resumed 4 weeks following closure. Three-month follow-up demonstrated the patient's ability to ambulate without the use of assisted devices with well-healed incisions.

Discussion

The pathophysiology of compartment syndrome is characterized by a unique type of ischemia that affects a group of muscles enclosed within a relatively nonexpandable fascial sheath and bony structures.[20] There remains uncertainty about the exact mechanism of the ischemia, but there are a number of proposed theories. One theory, described as the critical closing theory, states that there is a critical closing pressure in small vessels when transmural pressure (the difference between intravascular pressure and tissue pressure) drops. When tissue pressure rises, the transmural pressure decreases to a level such that elastic fibers in vessel walls ultimately collapse because they are no longer able to stretch and therefore unable to contribute to elastic tension.[21]

A second theory known as the arteriovenous gradient theory states that an increase in local tissue pressure simply reduces the arteriovenous pressure gradient and thus reduces blood movement into tissues. Finally, a third theory entitled the microvascular occlusion theory which suggests that capillary occlusion is the main culprit in reducing blood movement into the tissue. Regardless of the mechanism, ACS can be devastating if not acutely managed.[21]

The incidence of ACS following arthroplasty has been well documented in literature, more commonly in total knee arthroplasty (TKA) and THA.[19] The most commonly associated compartments to become affected in THA and TKA include the gluteal compartment (buttocks), the thigh, and the calf. Factors associated with ACS include prolonged positioning, tourniquet use, calf compression devices, postoperative analgesia, anticoagulant administration, high-risk patients, and early physiotherapy.[19] In our case, the patient received a number of these modalities and treatments, including Graston techniques specifically focusing on the foot. Graston technique is an instrument-assisted soft-tissue mobilization (IASTM). This technique is performed using six stainless steel tools of varying sizes, shapes, and styles of treatment edges to manipulate tissue. Figure 1a and b illustrates the Graston tools. Graston technique has been documented to increase blood flow to treated areas in order to allow healing.[33] There are no documented studies describing the induction of foot ACS following Graston techniques. Literature review did reveal a selected case in which exertional ACS was treated with Graston technique.[34] Hammer and Pfefer also described a case of subacute lumbar ACS and improvement after the use of IASTM.[32]

ACS is usually a clinical diagnosis characterized by symptoms consisting of pain out of proportion refractory to medication. The pain is worse with passive range of motion. These early signs should lead clinicians to suspect ACS. Late findings include swelling, cool extremities, and paralysis, usually due to lack of detection. Pulselessness may or may not occur, thus the presence of a peripheral pulse does not rule out the presence of ACS.[19,21] Clinical symptoms are usually adequate to make the diagnosis. In this setting, the clinical symptoms of severe right foot pain, swelling with tense compartments, paresthesias, and inability to bear weight allowed the physician to make the diagnosis of ACS without the need for ancillary testing such as intra-compartment pressure due to the presence of clear clinical signs.

Figure 1: (a and b) Graston technique steel rods

Residual soft swelling can be expected following hip surgery, especially at the caudal end of the limbs for up to 6 months due to edema secondary to inflammation and postsurgical changes without good elevation and mobilization technique. Immobilization enhances this engorgement, potentially causing or worsening ACS. To prevent swelling, it is recommended to elevate the affected extremities following arthroplasty while aggressively using compression stockings as per the American Academy of Orthopaedic Surgeons guidelines.[35] However, due to the aggressive Graston technique performed by the therapist, the patient developed ACS of the foot. Early and aggressive intervention should be initiated if ACS is clinically suspected, as late detection may have disastrous results.

In this scenario, prompt fasciotomy possibly spared the patient of irreversible sequelae, such as amputation or even death.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient has given her consent for her images and other clinical information to be reported in the journal. The patient understands that her name and initials will not be published and due efforts will be made to conceal identity, but anonymity cannot be guaranteed.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

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