EDORIUM Journals
www.edoriumjournals.com

CASE REPORT PEER REVIEWED | OPEN ACCESS

Lower extremity acute compartment syndrome secondary to inferior vena cava thrombus: A case report

Keegan Devon Bradley, Hao Wang

ABSTRACT

Introduction: Acute compartment syndrome (ACS) occurs when pressure within a muscle compartment builds to dangerous levels that exceed the perfusion pressure supplying blood to that compartment, which can lead to nerve and muscle tissue injury. It is uncommon to have an acute compartment syndrome suspected to be secondary to a clot of the inferior vena cava (IVC).
Case Report: A 51-year-old female, without recent trauma presented to an outside hospital emergency department with symptoms of acute onset left lower extremity pain and swelling. She was admitted for ACS, and subsequently required emergent fasciotomy. While in a rehabilitation facility, she had worsening leg pain and presented to the hospital, where she was found to have an occlusive thrombus in her IVC that extended down to her lower extremities. It was later noted the anterior compartment of the patients left lower leg had become necrotic, and the patient could no longer dorsiflex her foot. After reviewing the report and outcome of the patient, it was concluded that she had developed compartment syndrome without any obvious instigating cause.
Conclusion: It is important to have a low threshold and be vigilant for compartment syndrome in any patient presenting with suspicious symptoms, even patients who seem to have no history or related trauma typically associated with compartment syndrome.

All Articles Open Access

International Journal of Case Reports and Images (IJCRI)

International Journal of Case Reports and Images (IJCRI) is an international, peer reviewed, monthly, open access, online journal, publishing high-quality, articles in all areas of basic medical sciences and clinical specialties.

Aim of IJCRI is to encourage the publication of new information by providing a platform for reporting of unique, unusual and rare cases which enhance understanding of disease process, its diagnosis, management and clinico-pathologic correlations.

IJCRI publishes Review Articles, Case Series, Case Reports, Case in Images, Clinical Images and Letters to Editor.

Website: www.ijcasereportsandimages.com

(This page in not part of the published article.)

CASE REPORT PEER REVIEWED | OPEN ACCESS

Lower extremity acute compartment syndrome secondary to inferior vena cava thrombus: A case report

Keegan Devon Bradley, Hao Wang

ABSTRACT

Introduction: Acute compartment syndrome (ACS) occurs when pressure within a muscle compartment builds to dangerous levels that exceed the perfusion pressure supplying blood to that compartment, which can lead to nerve and muscle tissue injury. It is uncommon to have an acute compartment syndrome suspected to be secondary to a clot of the inferior vena cava (IVC). Case Report: A 51-year-old female, without recent trauma presented to an outside hospital emergency department with symptoms of acute onset left lower extremity pain and swelling. She was admitted for ACS, and subsequently required emergent fasciotomy. While in a rehabilitation facility, she had worsening leg pain and presented to the hospital, where she was found to have an occlusive thrombus in her IVC that extended down to her lower extremities. It was later noted the anterior compartment of the patients left lower leg had become necrotic, and the patient could no longer dorsiflex her foot. After reviewing the report and outcome of the patient, it was concluded that she had developed compartment syndrome without any obvious instigating cause. Conclusion: It is important to have a low threshold and be vigilant for compartment syndrome in any patient presenting with suspicious symptoms, even patients who seem to have no history or related trauma typically associated with compartment syndrome.

Keywords: Atraumatic, Compartment syndrome, Deep venous thrombosis, Inferior vena cava

How to cite this article

Bradley KD, Wang H. Lower extremity acute compartment syndrome secondary to inferior vena cava thrombus: A case report. Int J Case Rep Images 2017;8(5):317–321.

Article ID: Z01201705CR10793KB

****

doi:10.5348/ijcri-201754-CR-10793

Keegan Devon Bradley[1], Hao Wang[1]

Affiliations: [1]MD, Resident Physician, Emergency Medicine, John Peter Smith Hospital, Fort Worth, TX, USA.

Corresponding Author: Keegan Devon Bradley, Resident Physician, Emergency Medicine, John Peter Smith Hospital, Fort Worth, TX, USA; Email: kbradley02@jpshealth.org

Received: 15 January 2017
Accepted: 21 February 2017
Published: 01 May 2017

INTRODUCTION

The purpose of this case report is to demonstrate a novel case of suspected atraumatic acute compartment syndrome (ACS) secondary to a proximal obstructive venous thrombus. This is a potentially devastating condition when, for multiple reasons, an osseofascial compartment pressure rises to a level that decreases perfusion and may lead to permanent neuromuscular damage in that compartment. Acute compartment syndrome can occur anywhere in the body that muscle is surrounded by fascial tissue, and most commonly follows acute trauma involving long bones fractures [1]. Among all long bone fractures, tibia fractures are the most common fracture type with high ACS risks in adults [2]. In addition, ACS occurs more commonly in patients who sustain comminuted fractures [2]. One study showed that out of 164 patients, 69% of those cases were secondary to fractures in which tibial diaphyseal fractures accounted

for the majority (36%) and distal radius fractures were the second most common (9.8%) [3]. Similar findings were reported in other study with tibia fractures of diaphysis being the most common one [4]. Acute compartment syndrome, however, can also occur in non-trauma patients. Hynes et al. reported a case of ACS in a female with a presumed DVT after a prolonged episode of drug induced sleep without known trauma [5]. Gutfraynd and Philpott reported a 24-year-old male who developed ACS of the left thigh after being at a concert for the entire day. The patient denied any trauma, but still required fasciotomy [6].

Another study found that in 113 ACS patients, those who sustained no fracture had a significant delay in diagnosis and treatment. An average of 12.4 hour delay from onset of symptoms resulted in muscle necrosis in 20% of patients who eventually required debridement during fasciotomy [3]. Some other non-traumatic causes of ACS include bleeding disorders, vascular disease, nephrotic syndrome (or other conditions that decrease serum osmolarity), certain animal envenomation and bites, extravasation of intravenous fluids, injection of recreational drugs, and prolonged limb compression (e.g., following severe drug or alcohol intoxication or poor positioning during surgery). Postischemic compartment syndrome phenomenon has also been seen commonly following many procedures such as; bypass surgeries, embolectomies, or thrombolysis. Increasing flow into an already swollen compartment puts the patient at high risk for ACS. Dr. Newman et al. demonstrates this with a case report of a 57-year-old male with non-traumatic ACS of three compartments of the left lower extremity [7]. The patient had been recently started on anticoagulation for multiple pulmonary emboli and a deep venous thrombosis of the left posterior tibial and peroneal veins. Patient was found to have 3 of the 4 compartment syndromes with >70 mmHg, which required fasciotomies that resulted in significant blood loss but no deficits postoperatively.

The clinical symptoms of ACS include any combination of: pain out of proportion to the clinical scenario, pain with passive stretch, paresthesias, peripheral swelling, and late findings including paralysis and absent peripheral pulses. However, different patients can present differently with very subtle symptoms. Cohen et al. reported pediatric patients can present more often with paresthesias than pain, and 12% of these patients with ACS will not have any pain at all in their course [8]. Stollsteimer et al. present a case of a young, unconditioned, football player who attempted to run a mile twice under 7.5 minutes. The patient then began to have persistent leg pain, but was able to achieve the run day-3. By this time the patient was diagnosed by the trainer with shin splints, but then was sent to the team physician day-4 due to ongoing pain and new inability to dorsiflex the foot. Patient was sent to hospital and diagnosed in the emergency department with ACS. He underwent fasciotomy, but unfortunately was unable to regain function in that extremity [9]. Another example of a different presentation of atraumatic ACS is presented by Parisa et al. which demonstrates a patient that develops thigh compartment syndrome with an unknown cause secondary to a hematoma in his leg. The patient presented with 10/10 pain in his right thigh, and was found to have a large spontaneous thigh hematoma that required fasciotomy and evacuation. The only presenting symptom was ongoing pain without numbness or weakness [10].

CASE REPORT

A 51-year-old Caucasian female who presented to study hospital for chief complaint of left anterior leg pain, numbness between first and second toes, and inability to dorsiflex her left foot. Prior to being seen at study hospital the patient had presented to an outside hospital with a chief complaint of acute onset left lower extremity pain and swelling without trauma or an inciting event. She was admitted for ACS monitoring, where she subsequently developed increasing pain, swelling, and numbness in her left foot the following night. Imaging at the outside hospital showed bilateral deep vein thromboses and small distal pulmonary emboli. She was taken to the operating room the same night for emergency medial and lateral incision fasciotomy of her left lower extremity. Patient was monitored in hospital for another week before she was discharged to a rehabilitation facility. While in rehabilitation facility, patient daughter noticed that patient's left leg pain had become worse and she was subsequently diagnosed cellulitis and antibiotics were given. However, patient's leg pain has not improved and patient thus presented to emergency department of the study hospital for further evaluation.

Patient's past medical history included diabetes and hyperlipidemia. Past surgical history included tubal ligation, cesarean section, and recent left lower extremity fasciotomy. No family history of bleeding problems or cancer. Home medications of note include: oral contraceptive pills for a year, as well as Coumadin started at the initial outside hospital.

Physical examination was remarkable for a diffusely swollen left lower extremity with wound vacuum in place over medial and lateral previous fasciotomy sites. Upon removal of the wound vacuum, there was foul smelling serous drainage from both wounds. The medial incision showed viable muscle tissue that contracted with sudden stimulation. The lateral incision revealed a posterior compartment with muscle that contracts on stimulation, but the anterior compartment did not respond to stimulation. Patient was able to plantarflex, invert, and evert her left foot, but unable to dorsiflex. There is a loss of sensation in the deep peroneal nerve distribution, but sensation remained intact in the saphenous, sural, and superficial peroneal nerve distributions. Dorsalis pedis and posterior tibial signals were appreciable on doppler, but unable to be palpated. No pain with palpation of her thigh (Figure 1A–D).

Patient's X-ray of left lower extremity showed no acute fractures, and laboratory examinations were significant C-reactive protein (CRP) 125, lactate 3.5, white blood cell count (WBC) 19, and international normalized ratio (INR) of 2.3.

Ultrasound revealed thrombi extending from distal inferior vena cava to the level of the popliteal veins on left and calf veins on the right. Computed tomography angiography demonstrated occlusive thrombus within femoral and iliac veins extending into the IVC. The extension included the infrarenal, juxtarenal, suprarenal and up to the intrahepatic portion of the IVC. The suprarenal IVC measures 32 mm in diameter, which made it too large from any filter devices to be placed. The patient's subsequent hospital course began with vascular surgery recommending heparin therapy with a hematology consult for hypercoagulability workup, as well as to guide future long-term therapy. The patient was taken to the operating room by orthopedic surgery for fasciotomy and replacement of a wound vacuum. During the hospitalization, the patient also developed heparin induced thrombocytopenia (HIT) and heparin was discontinued. For the remainder of her hospital course her treatment was focused on medical anticoagulation via argatroban bridging to Coumadin with an INR goal of 2–3. Following discharge the patient would require lifelong Coumadin therapy given her extensive clot burden, as well as outpatient follow-up with orthopedics and continued physical therapy. She was most recently seen in the orthopedic clinic approximately six months following discharge from the hospital. She had been continuing outpatient physical therapy, but has not regained any ability to dorsiflex her left foot. (Figure 2A–D).

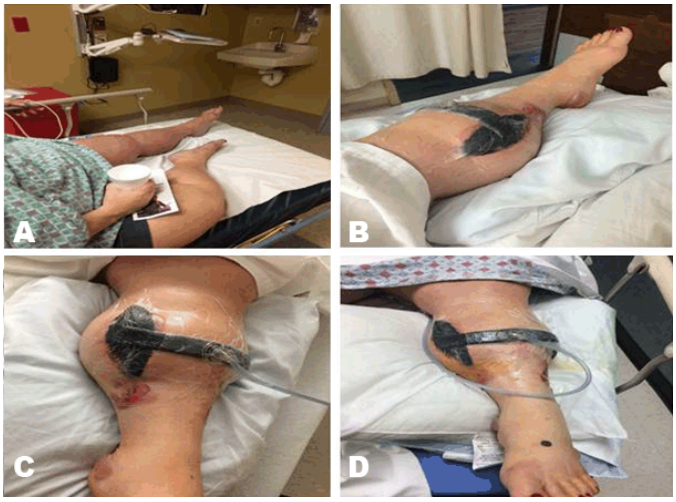

Figure 1: Patient's leg immediately prior and after fasciotomy was performed and a wound vacuum was applied. Left lower extremity on initial presentation February 2nd, 2015 (A) Left lower extremity after initial fasciotomy at outside hospital February 2nd, 2015, (B) Left lower extremity after initial fasciotomy at outside hospital February 2nd 2015, and (C, D) Left lower extremity taken during initial presentation to our hospital February 12th 2015.

Figure 2: Patient's wounds after the vac was removed and the progress of her healing. (A) Left lower extremity following surgery at our hospital February 17th, 2015 , (B) Left lower extremity following surgery at our hospital February 17th, 2015, (C) Left lower extremity while being seen in clinic in April 2015, and (D) Left lower extremity taken at home in April 2015.

DISCUSSION

Here, we report a patient who presented initially with inferior vena cava thrombosis, subsequently developed lower extremity compartment syndrome, which eventually resulted in necrosis and fasciotomy. To the best of our knowledge, this case is unique due to few reported ACS cases that have developed without trauma in the setting of an occlusive proximal thrombus. Therefore, it is important to recognize atypical clinical presentations of such ACS and understand its etiologies.

Incidence of reported cases of unilateral ACS so distal to a bilateral occlusive thrombus without reperfusion is very scarce in current literature. Most cases of ACS reported secondary to a thrombus are typically seen when a thrombus is lysed and reperfusion causes increased inflammation and expansion of muscle tissue in the associated compartment as demonstrated by cases such as those presented by Dr. Newman et al. and Dr. Javedani et al. [7, 10]. It is important to note that this patient's underlying pathophysiology leading to her ACS is very different from the well documented post-ischemic compartment syndrome. The etiology of ACS case reported here is most likely due to a significant VTE, including a bilateral lower extremity DVT which extends proximately to the IVC and pulmonary vessels. The incidence of bilateral lower extremity DVT is rare, and this should warrant more vigilant considerations of 1) the development of ACS; 2) the proximal large vessel VTE involvements; and 3) the potential systemic coagulopathies (such as anti-phospholipid antibody, protein C, protein S deficiency, etc).

Non-traumatic causes of ACS are not extensively reported, and likely predispose the afflicted patient to risk of prolonged time between presentation and diagnosis. Clinicians must have a lower threshold in diagnosing a patient with ACS who presents without a recent history of trauma. A diagnosis of ACS in a non-traumatic patient should be suspected among patients with lower extremity VTE.

The diagnosis of ACS itself is typically clinical, involving the physician looking for symptoms such as: increasing pain out of proportion to the stimulus, altered sensation, pain with passive stretch, muscle weakness, and palpable tightness of muscle compartments [1]. It is important to note that these symptoms are not, by themselves, particularly specific or sensitive in the diagnosis of ACS. For example, the degree of pain a patient reports can differ dramatically from patient to patient, hypoesthesia and muscle weakness can be secondary to direct nerve damage rather than increased compartment pressure, and compartment stiffness is not as appreciable in deeper compartments that are typically not palpable [2]. Symptoms such as muscle weakness or paralysis are signs that are typically seen at a point in the ACS process when permanent damage has been already occurred [11]. Individually, these tests are not diagnostic, but a constellation of multiple positive symptoms should alert the clinician to a case of potential ACS. Other diagnostic tests such as intercompartment pressure measuring can be applied with many agreeing that a difference between the diastolic blood pressure and the compartment pressure (delta pressure) of 30 mmHg or less be used as the threshold for diagnosing ACS [12]. Unfortunately, many of these advanced methods are not always on hand acutely when concern for ACS arises. It falls on the clinician to asses and use their best diagnostic judgement on whether the patient is suffering from ACS and requires immediate surgical intervention.

This case demonstrates how variable the underlying suspected causes of ACS in a patient can be. This patient's initial presentation was relatively stereotypical for ACS. However, she had a nontraditional underlying cause of an occlusive thrombus in her IVC. The thrombus was found to occlude from her IVC, bilateral femoral veins, and further extension down to the popliteal vein on the left and calf vein on the right. The significance of proximal occlusion is unclear, but further research or cases may demonstrate the possibility that proximal impedance of flow may lead to increased pressure in all distal compartments. Investigation for an occlusive proximal DVT would likely be indicated, if it can be further demonstrated in the future that proximal occlusion is in fact, a risk for development of distal ACS. She had a hypercoagulable workup done while being admitted to our hospital which was negative, except for being initially positive for antiphospholipid antibody which will require confirmatory testing later. Given this patients potential to be antiphospholipid antibody positive and the potential that proximal occlusion by a DVT can cause distal ACS, hypercoagulability workup should be considered in a patient with an otherwise non-traumatic unknown cause of ACS and spontaneous DVT formation.

What we learned from this case is that there are still potentially unknown causes of ACS that can be just as devastating. This can also delay a patient's diagnosis and more importantly their intervention. We found that occlusion of venous blood flow can potentially predispose the distal compartments to developing ACS. We suspect the pathophysiology behind the patients ACS occurring in the setting of a clot without reperfusion could possibly be secondary to: increases in pressure from lack of venous circulation return, an injury the patient does not remember that was significant enough to instigate an already pressurized compartment, or simply idiopathic ACS with a large clot being incidentally identified on investigation for underlying causes.

There remain some limitations of this case report. We are unable to confirm the patient had no trauma in her recent past though she did not recall the event. All pertinent information was obtained from this patient and no formal previous outside hospital health record reviewed. We are also unable to find strong evidence based study in literature to support the hypothesis of a proximal venous occlusion causing distal ACS. Further research should be focused towards assessment of the entire limb distal to the occlusion to measure if all compartments pressures increased or simply that single compartment. It should also be investigated if thrombectomy and return of flow will improve this specific cause of ACS by relieving pressure.

CONCLUSION

It is important for clinicians to suspect non-traumatic acute compartment syndrome (ACS) with atypical signs and symptoms such as bilateral lower leg DVT. More importantly, thorough evaluations including proximal venous images and systemic coagulopathy workups should be performed for potential ACS etiologies among these patients.

****

Acknowledgements
We are thankful to Hao Wang MD, PHD, FACEP; Research Director, John Peter Smith Hospital, Department of Emergency Medicine for his help and support in preparing this report.

Author Contributions
Keegan Devon Bradley – Substantial contributions to conception and design, Acquisition of data, Drafting the article, Revising it critically for important intellectual content, Final approval of the version to be published

Wang, Hao – Substantial contributions to conception and design, Drafting the article, Revising it critically for important intellectual content, Final approval of the version to be published

Guarantor
The corresponding author is the guarantor of submission.

Conflict of Interest
Authors declare no conflict of interest.

Copyright
© 2017 Keegan Devon Bradley et al. This article is distributed under the terms of Creative Commons Attribution License which permits unrestricted use, distribution and reproduction in any medium provided the original author(s) and original publisher are properly credited. Please see the copyright policy on the journal website for more information.

REFERENCES

1. Elliott KG, Johnstone AJ. Diagnosing acute compartment syndrome. J Bone Joint Surg Br 2003 Jul;85(5):625–32.
2. Stracciolini A, Hemmerberg ME. Acute Compartment Syndrome of the extremities. 13 May 2016. [Available at: http://www.uptodate.com/contents/acute-compartment-syndrome-of-the-extremities]
3. McQueen MM, Gaston P, Court-Brown CM. Acute compartment syndrome. Who is at risk? J Bone Joint Surg Br 2000 Mar;82(2):200–3.
4. Park S, Ahn J, Gee AO, Kuntz AF, Esterhai JL. Compartment syndrome in tibial fractures. J Orthop Trauma 2009 Aug;23(7):514–8.
5. Hynes JE, Jackson A. Atraumatic gluteal compartment syndrome. Postgrad Med J 1994 Mar;70(821):210–2.
6. Gutfraynd A, Philpott S. A Case of Acute Atraumatic Compartment Syndrome of the Thigh. J Emerg Med 2016 Sep;51(3):e45–7.
7. Newman PA, Deo S. Non-traumatic compartment syndrome secondary to deep vein thrombosis and anticoagulation. BMJ Case Rep 2014 Jan 17;2014. pii: bcr2013201689.
8. Cohen E, Truntzer J, Klinge S, Schwartz K, Schiller J. Acute pediatric leg compartment syndrome in chronic myeloid leukemia. Orthopedics 2014 Nov;37(11):e1036–9.
9. Stollsteimer GT, Shelton WR. Acute atraumatic compartment syndrome in an athlete: A case report. J Athl Train 1997 Jul;32(3):248–50.
10. Javedani PP, Spontaneous Compartment Syndrome of the Thigh in the Absence of Trauma. J Emerg Med 2016 Jul;51(1):70–2.
11. Rorabeck CH. The treatment of compartment syndromes of the leg. J Bone Joint Surg Br 1984 Jan;66(1):93–7.
12. Ovre S, Hvaal K, Holm I, Strømsøe K, Nordsletten L, Skjeldal S. Compartment pressure in nailed tibial fractures. A threshold of 30 mmHg for decompression gives 29% fasciotomies. Arch Orthop Trauma Surg 1998;118(1–2):29–31.

Access full text article on other devices

Access PDF of article on other devices

EDORIUM JOURNALS — AN INTRODUCTION

Edorium Journals: An introduction

Edorium Journals Team

About Edorium Journals

Edorium Journals is a publisher of high-quality, open access, international scholarly journals covering subjects in basic sciences and clinical specialties and subspecialties.

Invitation for article submission

We sincerely invite you to submit your valuable research for publication to Edorium Journals.

But why should you publish with Edorium Journals?

In less than 10 words - we give you what no one does.

Vision of being the best

We have the vision of making our journals the best and the most authoritative journals in their respective specialties. We are working towards this goal every day of every week of every month of every year.

Exceptional services

We care for you, your work and your time. Our efficient, personalized and courteous services are a testimony to this.

Editorial Review

All manuscripts submitted to Edorium Journals undergo pre-processing review, first editorial review, peer review, second editorial review and finally third editorial review.

Peer Review

All manuscripts submitted to Edorium Journals undergo anonymous, double-blind, external peer review.

Early View version

Early View version of your manuscript will be published in the journal within 72 hours of final acceptance.

Manuscript status

From submission to publication of your article you will get regular updates (minimum six times) about status of your manuscripts directly in your email.

Our Commitment

Six weeks

You will get first decision on your manuscript within six weeks (42 days) of submission. If we fail to honor this by even one day, we will publish your manuscript free of charge.*

Four weeks

After we receive page proofs, your manuscript will be published in the journal within four weeks (31 days). If we fail to honor this by even one day, we will publish your manuscript free of charge and refund you the full article publication charges you paid for your manuscript.*

Favored Author program

One email is all it takes to become our favored author. You will not only get fee waivers but also get information and insights about scholarly publishing.

Institutional Membership program

Join our Institutional Memberships program and help scholars from your institute make their research accessible to all and save thousands of dollars in fees make their research accessible to all.

Our presence

We have some of the best designed publication formats. Our websites are very user friendly and enable you to do your work very easily with no hassle.

Something more...

We request you to have a look at our website to know more about us and our services.

* Terms and condition apply. Please see Edorium Journals website for more information.

We welcome you to interact with us, share with us, join us and of course publish with us.

Edorium Journals: On Web

Browse Journals

CONNECT WITH US