# Traumatic Pedal Compartment Syndrome Caused by Dorsal Venous Arch Injury

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## Statement of Purpose

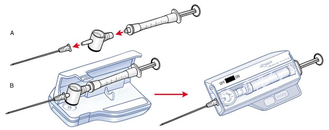
To present a dorsal venous arch injury in the setting of a traumatic pedal compartment syndrome, and to review its significance to the overall pathology.

## Literature Review

Compartment syndrome occurs when pressure builds inside an enclosed space, resulting in insufficient blood flow to neighboring tissues and nerves. In the foot this is very rare, accounting for <5% of all limb compartment syndrome cases [1]. Diagnosis should be based on the characteristic development of disproportionate, uncontrolled pain in the presence of swelling and ecchymosis. In acute compartment syndrome, trauma is often, but not exclusively, the mechanism of injury, with crush mechanism alone doubling the risk of developing a pedal compartment syndrome[2]. Other causes include: reperfusion of an ischemic limb, fracture, and severe muscle contusion. Literature shows that in the anticoagulated patient, compartment syndrome is still possible and should be highly considered in those with lower limb pain, even without a history of major trauma[3].Treatment consists of fasciotomy when compartment pressures are > 30 mmHg or if diastolic pressure minus compartment pressure is less than 30 mmHg[4].

## Case Report

A 56 year old female presented to the emergency room on August 4, 2016 with right lower extremity pain after falling from her wheelchair a day prior. The patient's past medical history was significant for atrial fibrillation, paraplegia, mechanical aortic valve, CVA with left-sided deficits, and polysubstance abuse.



Intra-compartmental Pressure Monitor

## Case Report Continued

On physical exam, pedal pulses were non palpable on the right, but dopplerable. Light touch sensation was absent, and she had tenderness upon palpation to the dorsum of the right foot. There was no increased pain elicited with passive dorsiflexion of the toes. Radiographs revealed no evidence of fracture. An intra-compartmental pressure monitor was used in the first interspace in the foot and was found to be 6 mmHg. It was suspected that the patient had a hematoma to the dorsum of the right foot and she was monitored closely. Of note, she also presented with an INR of 3.2. She was seen the next morning and her pain was well controlled with pain medication. Pedal pulses were now palpable. She was found to have a frank hematoma on the dorsum of the right foot that was then drained through a 1cm stab incision over the 1st interspace and packed bedside without complications. She was subsequently discharged and given follow- up. The patient failed to keep her scheduled appointment and returned to the emergency room one week later for increased pain and swelling to the right foot. Her INR at this admission was 5.4 and her toxicology screen was positive for cocaine. Her blood pressure at the time was 154/82mm Hg. Upon examination, she was found to have an edematous right foot with blister formation overlying the first ray. Skin tension lines were absent and the skin appeared shiny and atrophic. There was weeping serosanguinous drainage noted from the dorsum of the foot. Her pulses were dopplerable and she had pain with light touch to the dorsum of the right foot. Also, she was found to have pain with passive dorsiflexion of the lesser digits on the right. She exhibited tenderness with compression of the right calf. The intra-compartmental pressure monitor was used in the second interspace and the pressure was found to be 62 mm Hg. Prothrombin complex concentrate was administered for supra-therapeutic INR and the patient was taken for an emergent fasciotomy of the right foot. Intraoperative compartment pressures were measured and all compartments except for the calcaneal compartment were >30 mm Hg. The compartments were released through a dorsal lazy-S incision extending from the second to the fourth metatarsals, as well as a medial forefoot incision. Intraoperatively there was extensive hematoma. It was also noted that the lateral dorsal venous arch was lacerated, which was clamped and tied off with 3-0 Vicryl ties. Following surgery a venous duplex of the right leg was performed which revealed deep venous thrombosis of the right peroneal vein. The patient was anticoagulated with Heparin and bridged to Coumadin. Wound VAC therapy was initiated to both fasciotomy sites. The patient healed uneventfully. The patient was followed for a total of 14 months at the time of this poster submission.

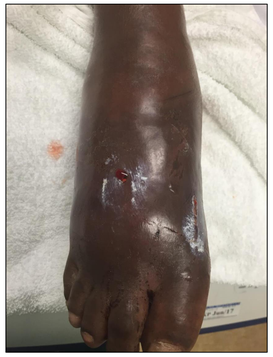


Figure 2: 8/11/15

Figure 3: 8/22/16

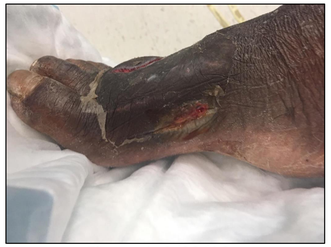


Figure 4: 8/22/16

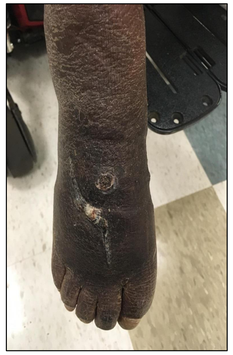


Figure 5: 10/31/16

## Discussion

We report a case of minor trauma sustained to the foot of an anticoagulated patient. The patient was diagnosed with a deep venous thrombosis of the right lower leg as well as compartment syndrome of the right foot. It was discovered intraoperatively while undergoing fasciotomy that the dorsal venous arch was lacerated and there was significant surrounding hematoma. To the best of our knowledge, there are no reported cases of venous arch injury causing pedal compartment syndrome. However, literature does provide some insight into the significance of these venous findings.

Tissue hemodynamics was extensively reviewed in a study on compartment syndrome with arterial and venous injuries of the lower extremity. They found that in all the patients that developed compartment syndrome, there was vascular compromise and concomitant venous injury. Vascular compromise was repaired within 5 hours, but venous injury was either ligated or the repair occluded. When recreating these injuries with animal models, similar results were reported. The authors concluded that short-term ischemia and reperfusion in combination with persistent venous occlusion significantly increased compartment pressure[4]

Brockman and Vasco in 1966 demonstrated that after massive venous thrombosis in the lower extremity, there was a resultant increase in interstitial pressure in the corresponding muscle compartment. They stated that this rise in interstitial pressure occurred because hydrostatic pressure on the venous capillary rose above colloid osmotic pressure, resulting in movement of fluid into the interstitial space and therefore an increase in compartment pressure[5].

We hypothesize that the pathogenesis of the patient's compartment syndrome was likely multifactorial. The findings of popliteal venous thrombus and dorsal venous arch laceration with surrounding hematoma cannot be ignored. The patient suffered occlusion of both their superficial and deep venous system, successfully occluding venous outflow. It is not possible to conclude how or when the dorsal venous arch injury occurred, but it is evident from the literature that venous injury can and will increase compartment pressure. This has a major impact, especially in the setting of anticoagulation with likely additional vascular compromise from foot contusion.

**References**

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