CASE REPORT

Spontaneous acute compartment syndrome of the forearm in a patient on oral anticoagulant therapy

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ABSTRACT
Acute compartment syndrome (ACS) in the upper extremity may present with many different signs and symptoms such as swelling, severe pain or paresthesia. These patients require an emergent fasciotomy to decompress the compartment. It is commonly caused by major traumas however a few cases of spontaneous ACS is associated with diabetes, oral anti-coagulation therapy, hypothyroidism or nephrotic syndrome. The report’s focus is to identify the sign/symptoms of ACS in patients taking anticoagulant therapy and encourage prompt interventions to prevent ischemia and necrosis. A 33-year-old right hand dominant Hispanic male presented to the emergency department with severe pain and swelling in his right forearm. The patient did not recall any trauma to his arm but stated that he was taking Warfarin for a venous thrombosis diagnosed 3 months ago. A venous duplex was performed which revealed a 3.2 x 2.2 x 2.2cm avascular mass in the anteriomedial forearm suggestive of an intra-compartmental hematoma. Although uncommon, it can lead to spontaneous acute compartment syndrome. Studies have suggested the correlation between warfarin usage and spontaneous ACS. The causative agent should be discontinued or managed and an emergent fasciotomy may be needed to prevent further ischemia, septicemia, limb loss or possible death.

Key words: acute compartment syndrome, anti-coagulant therapy, fasciotomy, international normalized ratio

INTRODUCTION
Acute compartment syndrome (ACS) of the upper extremity is a rare diagnosis. ACS can be defined as the increase in compartment pressure which exceeds tissue perfusion pressure leading to ischemia and necrosis.1 Often times the cause is due to traumatic events such as suprachondylar humerus fractures, vasculature injury or circumferential full thickness burns. Spontaneous ACS can be defined as having the symptoms of ACS in the absence of trauma to the area. Although less frequent, non-traumatic causes include ischemia-reperfusion injury, thrombosis, bleeding disorders, vascular disease, prolonged limb compression or anticoagulant therapy.2 Common signs and symptoms of ACS are pain out of proportion to the clinical presentation, paresthesia within an hour of ACS, tense/firm compartment, diminished sensation, pallor due to vascular insufficiency and paralysis.1 Many studies have shown that patients on an anticoagulant therapy regimen, more specifically Warfarin, have a 0.4%-7.2% increased chance of a hemorrhage compared to baseline that may lead to ACS.1 Major and minor traumatic events can easily disrupt the vasculature and cause ACS. Hay et al. described the correlation between Warfarin use and the development of ACS. The study followed seven patients on Warfarin who suffered from ACS due to minor injuries to the lower extremities.4 This report presents a case of spontaneous acute compartment syndrome due to the development of an intra-compartmental hematoma in the anteriomedial forearm. Reported cases of spontaneous ACS are associated with diabetes, hypothyroidism, anti-coagulation therapy, influenza virus induced or nephrotic syndrome.5 The patient was taking Warfarin and does not recall any trauma or injury prior to the onset of symptoms. Radiologic images are also negative in revealing any possible injury.

CASE REPORT
A 33-year-old right hand dominant Hispanic male presented to the Emergency Department with the chief complaint of severe pain in his right forearm. He stated that the pain started about 12 hours prior to presenting to the hospital and has rapidly worsened. He rated the pain 10/10 and denied any recent trauma. The review of systems were within normal limits. He had a history of hypertension, epilepsy and was diagnosed with a DVT in the left lower extremity about 3 months ago, for which he was prescribed Warfarin.
was immediately cleared and consented for an emergency venous duplex. Imaging revealed a 3.2 x 2.2 x 2.2 cm avascular mass in the anteriomedial forearm suggestive of an intra-compartmental hematoma. Ultrasonography showed no deep venous thrombosis in the brachial, ulnar or radial vein. Laboratory values revealed a white blood cell- 11.9x1000/ul, Hemoglobin- 15.1g/dl, Hematocrit- 46.4%, Platelet count- 234x109/L. Upon physical exam his right forearm was swollen, tense and painful to palpation. Neuromuscular exam showed positive paresthesia on the palmar and dorsal surfaces and decrease range of motion (flexion/extension) in all of the right digits. Radial pulses were still palpable with rapid capillary refill <2sec. Negative findings on upper right arm exam. There was no sign of cellulitis or open wounds. Vascular surgery was consulted and elected to perform a venous duplex. Imaging revealed a 3.2 x 2.2 x 2.2 cm avascular mass in the anteriomedial forearm suggestive of an intra-compartmental hematoma. Ultrasoundography showed no deep venous thrombosis in the brachial, ulnar or radial vein. Laboratory values revealed a white blood cell- 11.9x1000/ul, Hemoglobin- 15.1g/dl, Hematocrit- 46.4%, Platelet count- 183x109/L, INR-1.8 and PTT-42.2 sec. The patient was immediately cleared and consented for an emergent fasciotomy based upon the clinical findings for acute compartment syndrome of the forearm.

A fasciotomy was performed in the operating room in the anterior (flexor) and posterior (extensor) compartments. A 26 cm incision was made in the anterior compartment, extending distally from the antecubital region to the anterior portion of the wrist. An 18 cm incision was made in the posterior compartment. The muscles were found to be edematous but no injuries were noted upon exploration. The intra-compartmental hematoma was evacuated and drained; the wound was left open. Xeroform petrolatum wound dressing, 4x4 gauze, ABD pads and 6” cling were used to bandage the wound.

The patient was seen the next day with no new complaints. Patient denied any loss of sensation/numbness or tingling. Radial pulses were palpable but range of motion in the hand was still limited due to the pain. Vital signs were stable temperature-98.7°F, Pulse-95 beats/min, Respiration-20 breathes/min, blood pressure-112/64 mmHg. Laboratory values revealed slight changes most like due to recent surgery: white blood cell- 14.9x1000/ul, Hemoglobin-13.2g/dl, Hematocrit-46.4%, Platelet count- 234x109/L, INR-1.8 and PTT-42.2 sec. Warfarin was discontinued during his hospital stay. He returned to the OR 6 days later for a washout, primary closure and split thickness skin graft to the forearm. The grafts were taken from the anterior thigh bilaterally. The distal portions of the wound were closed with sutures. A 15x7 cm skin graft was placed on the anterior compartment and a 19x8 cm skin graft was placed on the posterior compartment.

DISCUSSION
Anticoagulant therapy should be periodically monitored to lower the possibility of hemorrhaging due to any major or minor insult. INR values should be maintained between 2.0-3.0 and dosages should be adjusted accordingly.3 In patients taking anticoagulant therapy and presenting with pain out of proportion to clinical findings and swelling, ACS should be high on the differential diagnosis. Anticoagulant therapy associated ACS was first documented in 1992 by Hay. He reported six cases of ACS in the lower extremity and one case in the upper extremity in patients who were taking anticoagulant therapy for atrial fibrillation, aortic valve replacement or DVT prophylaxis. These patients had undergone various injuries: fibular fracture, pulled muscles or minor impacts.4 More recently Titolo reported a 66 year old female taking anticoagulant therapy and developing ACS after sustaining a minor shoulder elongation. Similar to the previous study, the patient’s INR was within the therapeutic range.5 Major traumas associated with anticoagulant therapy usage have also been reported. Fung reported a 77 year old male on warfarin who developed ACS after sustaining a bicep tendon rupture. Fasciotomy was immediately performed to evacuate the hematoma and decompress the arm.5

In our case, major/minor trauma was not noted however the use of Warfarin and low INR levels may have contributed to the development of a spontaneous ACS. Other spontaneous causes include diabetes, which was reported by Jose, anti-coagulation use, hypothyroidism and nephrotic syndrome.5 Spontaneous ACS related to warfarin usage is rare but should quickly be ruled out with clinical presentation or imaging. Zimmerman et al. first report spontaneous ACS in a 75 year old man taking anticoagulant therapy for atrial fibrillation. The patient’s INR was 2.8 and did not report any recent trauma nor did radiologic images show any injury to the site.8 Our patient presented very similarly however an intra-compartmental hematoma was discovered upon imaging, highly indicating for fasciotomy.

The initial clinical presentation of the patient is essential in properly diagnosing and treating acute compartment syndrome. Despite the absence of classical mechanisms of ACS (ie. Fractures), and abnormal lab values suggestive of hemorrhage, anticoagulant therapy usage in patients presenting with pain in the upper and lower extremity should lead to the suspicion of ACS. Emergent fasciotomy is a definitive treatment and should be performed within 24h to avoid ischemia, septicemia, limb loss and possibly death.9

CONCLUSION
This case report presents the rare occurrence of a spontaneous acute compartment syndrome due to the use of anti-coagulant therapy and the importance of rapid clinical diagnosis and treatment. Despite the absence of any significant trauma, ACS should remain high on the differential diagnosis because of the clin-
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ical signs and symptoms. After proper imaging and labs are performed to rule out other causes, such as a venous duplex scan, an emergent fasciotomy should be performed to prevent further ischemia and necrosis.

REFERENCES