

Atraumatic Compartment Syndrome of the Dorsal Compartment of the Upper Arm

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Compartment syndrome is caused by fascial compartment pressures that exceed capillary perfusion, causing ischemia and edema within the compartment. This has been well characterized in the lower leg following excessive muscle strain, tibia fracture, or other blunt trauma and in the forearm following blunt trauma. There are few reports of dorsal compartment syndrome of the upper arm. A literature review found only one previous case without known preceding trauma.

We present a patient in whom dorsal compartment syndrome of the upper arm was likely caused by insidious hemorrhage secondary to enoxaparin and aspirin therapy.

CASE REPORT

A 74-year-old left-hand-dominant woman presented with a 3-day history of right arm swelling, pain, and weakness that began the night after receiving an injection of epoetin in the right shoulder. Her swelling worsened, and her pain increased progressively over the next 2 days until the morning prior to her arrival, when she began to have numbness, partial loss of function in the hand, and severe pain. Approximately 24 hours after the start of these symptoms, she was seen in the emergency room.

She denied any antecedent trauma or history of bleeding episodes. Her medical history included a 7-year history of non-small cell lung cancer treated with lobectomy and chemotherapy. She had been found to have residual disease and was started on gefitinib, a trial cancer drug, approximately 4 weeks prior to this incident. She had a history of pulmonary embolism 6 years previously, which her oncologist attributed to a hypercoagulable state secondary to her cancer. Although she was treated therapeutically with warfarin for approximately 10 years, she sustained multiple transient ischemic attacks. Her anticoagulation regimen was changed to enoxaparin 60 mg SQ bid approximately 6 weeks prior to her admission. Other medical history

was notable for hypertrophic cardiomyopathy, depression, osteoporosis, and chronic obstructive pulmonary disease, for which she took prednisone 5 mg/day. She also took aspirin (81 mg) daily and received epoetin for anemia. She reported that the epoetin was given intramuscularly.

On physical examination, her vital signs were all normal, with the exception of a systolic blood pressure of 147 mm Hg and a diastolic pressure of 82 mm Hg. She held her arm in 90° of elbow flexion. Ecchymosis extended over the entire posterior upper arm and anterolateral forearm. The posterior compartment of the arm was firm, swollen, and painful to palpation (Figure 1). The anterior arm was less tense and not as painful. She had no active elbow extension, wrist

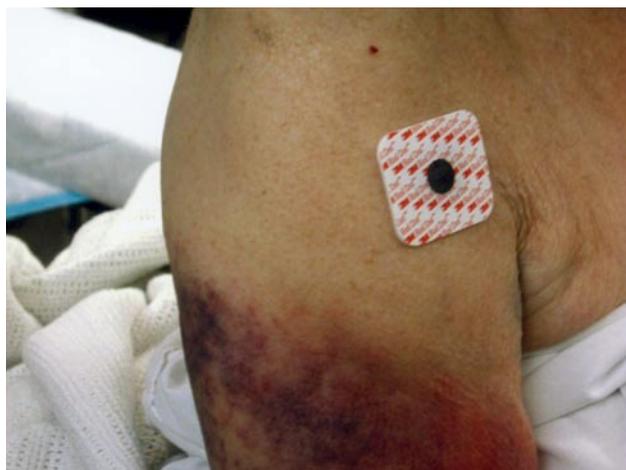


Figure 1. The arm was tense and painful posteriorly.

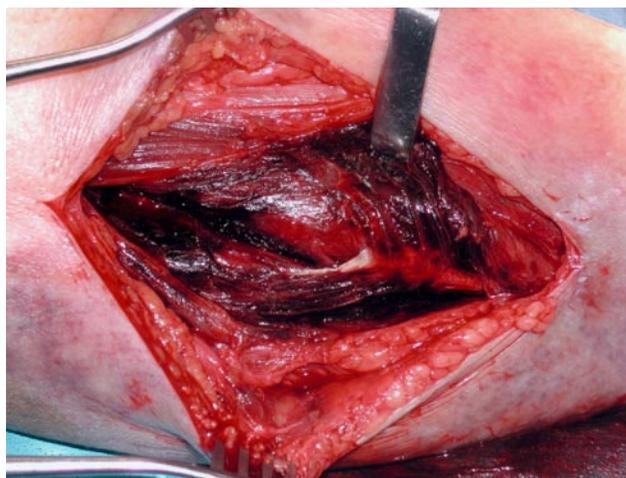


Figure 2. Muscle in the dorsal compartment appeared grossly viable.

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extension, or extensor pollicis longus function. Function of her digital extensors, including the extensor digitorum communis, abductor pollicis longus, and extensor digiti minimi muscles also was diminished. Deltoid muscle, elbow flexors, flexor digitorum profundus, wrist flexors, and interossei were graded 4/5. She was able to flex her elbow from 90° to 120°, and passive flexion beyond this arc caused severe pain. The remainder of her examination was significant for decreased sensation in the radial nerve distribution. Sensation was normal in the axillary, ulnar, and median nerve distributions. Her radial pulse was present and equal to the unaffected side. Radiographs of the right humerus and shoulder revealed only osteopenia and osteoarthritis of the glenohumeral joint. Despite a regimen of 60 mg of enoxaparin twice daily, her prothrombin time, partial thromboplastin time, and international normalized ratio were within normal limits. Her platelet count was 357,000, and her hemoglobin level was 8.5 mg/dL.

The patient was diagnosed with an acute compartment syndrome of the posterior arm and was scheduled for urgent fascial release. Her compartment pressures were measured by Whitesides technique.¹ The posterior compartment measured 93 mm Hg, a level so high that upon extrusion of the needle, serosanguinous fluid was forcefully ejected through the puncture site. The anterior compartment measured 11 mm Hg. A straight lateral longitudinal incision approximately 10 cm in length was made overlying the interval running from the lateral head of the triceps to the brachioradialis. When the fascia of the posterior compartment was entered, approximately 250 mL of hematoma escaped from within the posterior compartment (Figure 2), and this immediately decompressed the entire arm. The fascia was released along its entire length. No active site of bleeding was encountered. The wound was irrigated thoroughly, and the skin was approximated loosely with a vessel loop in a crisscross pattern through staples along the wound edge. The posterior compartment pressure measured 4 mm Hg at the end of the procedure.

“Pain with passive stretch or neurologic symptoms mark the beginning of the ischemic window...”

Postoperatively, the patient was given cefazolin, and her arm was kept elevated. Her creatine phosphokinase, myoglobin, and serum creatinine values were closely monitored, and she was well hydrated. She returned to the operating room 48 hours later for inspection, irrigation and débridement, and primary closure. Very little necrosis was found, and closure was performed easily. Although elevated initially, the creatine phosphokinase and myoglobin levels normalized, and there was no evidence of reperfusion injury or renal impair-

The Upper Arm's Posterior Compartment: An Unlikely Site of Compartment Syndrome

The posterior compartment contains the triceps brachii, the anconeus, the deep brachial artery, and the radial nerve. Why upper arm dorsal compartment syndrome is so rare is unclear, but several hypotheses are feasible¹⁰:

- (1) Muscle hypoxia that occurs in the leg from both over use and acute trauma rarely occurs in the arm.
- (2) Occlusive arterial disease leading to ischemia is much less frequent in the arm than in the lower leg.
- (3) Fractures that lead to vascular injury or hemorrhage into a compartment occur less often in the arm than in the lower leg.
- (4) The brachial fascial compartments frequently blend anatomically with those of the shoulder, creating a potentially larger space for blood to track⁴.
- (5) The brachial fascia has fewer taut, broad tendons and ligaments blended with it and therefore is more compliant with rising intracompartmental pressures.¹¹

ment. She was placed in a cock-up wrist splint and began occupational therapy for a persistent radial nerve palsy. The palsy lasted for several weeks and slowly began to improve. Currently, 9 months postoperatively, she has recovered nearly full motor function of her radial nerve but is left with a mild sensory deficit.

DISCUSSION

Compartment syndrome is characterized by increasing interstitial pressure in rigid osseofascial envelopes, surpassing capillary perfusion pressures such that myoneural tissue becomes hypoxic and at risk for ischemic necrosis. Compartment syndrome has classically been described in the compartments of the lower leg and forearm^{2,3} but has only rarely been reported to occur in the posterior arm. Brumback⁴ reported a case of dorsal arm compartment syndrome following avulsion of the lateral head of the triceps. Cases of compartment syndrome of the upper arm have been reported following blunt trauma in a football game,⁵ an altercation,⁶ a surgical neck fracture of the humerus,⁷ thrombolytic therapy,⁸ and in a professional power weight lifter.⁹ This is the second reported case of posterior arm compartment syndrome without known preceding trauma, and the only known case caused by hemorrhage secondary to enoxaparin and aspirin therapy. The factors that may contribute to the rarity of compartment syndrome's developing in the posterior arm are enumerated in the Box above.

Clinical signs and symptoms of compartment syndrome include pain out of proportion, with severe pain on passive stretch of the muscles in the involved compartment¹²; a tense compartment on palpation; and paresis or paresthesias in the distribution of the nerves running through that compartment.^{3,13,14} When clinical signs are equivocal, direct compartmental pressure

can be measured. Our patient demonstrated each of the hallmarks of compartment syndrome, with some weakness of the ulnar nerve as well. Based on the clinical diagnosis, the patient was taken for emergent operative decompression.

The direct cause of this patient's hemorrhage into her posterior arm compartment is unknown. Although she recalled an intramuscular injection of epoetin in the superior deltoid several days prior to admission, this was away from the site of hemorrhage. More likely a subclinical traumatic event, coupled with her aggressive anticoagulation status, caused a slow bleed that accumulated in the dorsal compartment over several days, causing progressive pain and ultimately a radial nerve palsy. Pain with passive stretch or neurologic symptoms mark the beginning of the ischemic window,¹¹ and decompression within 8 hours^{15,16} to 12 hours^{2,17} of the onset of ischemia will usually avert muscle necrosis. Operative decompression in this case revealed healthy muscle, indicating ischemia had only recently begun.

The risk of a clinically significant bleeding episode is 0.9% in patients treated with enoxaparin for deep venous thrombosis prophylaxis (30 mg SQ bid) following hip replacement.¹⁸ At higher doses (1 to 1.5 mg/kg) bleeding rates have been reported at 1.3%,¹⁹ 2%,²⁰ 3.3%,²¹ and 4%.²²

CONCLUSIONS

Regardless of the initiating factor or the compartment involved, the common end point of increased pressure within the compartment is consistent. As with other compartment syndromes, emergent decompression is appropriate. Although arm compartment syndromes are only rarely reported, the physician must be vigilant when a patient presents with the combination of increased pain, severe pain with passive stretch, palpably tense compartments, and possibly neurologic findings, particularly when the patient is on a therapeutic anticoagulation regimen.

AUTHORS' DISCLOSURE STATEMENT AND ACKNOWLEDGMENTS

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