Compartment Syndrome Following the Use of Statins: A Case Presentation

Gabriel Santamarina, DPM
Valeria Alfano, BS
Noelis Rosario, DPM
Hani Badahdah, DPM, MD
Mario Cala, DPM
Thomas Merrill, DPM

INTRODUCTION

Compartment syndrome is a medical emergency. The consequences of this condition can be devastating. Ignoring this disease can result in acute muscle necrosis, nerve damage, life-long disability or loss of limb (1, 2). Compartment syndrome is defined as a condition of elevated intramuscular compartment pressure that is greater than the diastolic blood pressure, resulting in decreased perfusion to the compartment. The most common area of presentation is the lower extremity, below the knee (3, 4). Typically, this condition is associated with trauma in the foot, especially after fracturing multiple metatarsal bones. The trauma generally associated with compartment syndrome in the leg, is a fracture of the tibial bone (1, 5, 6). Regardless of etiology, it is imperative to recognize the condition as early as possible to avoid complications. This is a unique case of compartment syndrome that presented without any obvious trauma to the right lower extremity.

Statins are a very popular class of drugs taken long-term and used to lower high cholesterol. A well-documented serious side effect of these medications is rhabdomyolysis, nontraumatic breakdown and bleeding in muscle tissue. This can produce a nontraumatic, acute compartment syndrome with elevated compartment pressures. Our patient developed elevated superficial posterior compartment pressures following an acute episode of rhabdomyositis that compromised the patient’s medial head of the gastrocnemius muscle. This injury bled into the superficial posterior compartment of the right lower extremity, becoming a very painful hematoma. The elevated compartment pressures required an emergency superficial posterior fasciotomy and evacuation of the hematoma.

CASE PRESENTATION

The patient is a 47-year-old male with a history of diabetes mellitus type 2, hypertension, and obesity, who presented to the emergency department with acute pain to the posterior aspect of the right lower extremity. The patient stated that the pain began at 8:00 a.m. when he was standing and felt a sharp pain to his posterior right lower extremity over the calf area. Immediately his calf swelled up “like a balloon.” The patient denied any previous heavy exertional activity or trauma to the area. Icing and anti-inflammatory medications were not effective on alleviating the pain. The patient sought emergency medical treatment at 7:00 p.m. that night after he could no longer tolerate the pain to the right lower extremity. At the time of examination, the patient described the pain as being a 10 (on a scale of 1-10) and sharp, throbbing, and localized to the posterior aspect of the calf. He denied any nausea, vomiting, fevers, chills, shortness of breath, or chest pain. A complete history and physical examination was performed and was unremarkable outside of the history of present illness. The patient’s medication list included hypertension medication (losartan), diabetic medication (metformin), gastric acid medication (omeprazole), and a cholesterol-lowering statin (simvastatin). The patient denied any previous surgeries or known drug or food allergies. There was no reported alcohol, tobacco, or illicit drug use.

The patient was examined in the emergency department while in a supine position on the examination bed. The patient was diaphoretic and exhibited grimacing and signs of pain. Focusing on the right lower extremity, diffuse areas of ecchymosis to the proximal posterior aspect of the calf area were identified. No open lesions or scaring were noted. The dorsalis pedis and posterior tibial artery
Pulses were fully palpable with a capillary refill time less than 3 seconds to all the right lower extremity digits. Non-pitting edema was noted to the posterior aspect of the right lower extremity with significant musculature tightness and tenderness on palpation (Figure 1). Protective and epicritic sensations were grossly diminished to the most distal aspect of the extremity. The patient was unable to perform any range of motion to the right knee, ankle, or pedal joints due to excruciating pain.

Radiographic examination to the right proximal tibia and fibula revealed significant soft tissue swelling with no cortical interruptions. The venous ultrasound studies were negative for right lower extremity deep vein thrombosis. There was however, a 9.7 cm heterogeneous lesion noted along the right calf musculature suggesting a gastrocnemius muscle tear and hematoma.

Due to the nature of the history, physical examination, and imaging studies, compartment pressure evaluation was performed to the right lower extremity. A local infiltration block of 1% lidocaine plain local anesthetic was used to each area. The compartment pressures were taken using a Stryker brand Wick Catheter. The normal compartment pressures in the lower extremity are <30 mm Hg. The superficial posterior compartment initially demonstrated a reading of 106 mm Hg, a repeat examination revealed a pressure of 65 mm Hg (Figure 2). The deep posterior compartment demonstrated 62 mm Hg of pressure and the anterior compartment 15 mm Hg of pressure. Patient refused lateral compartment pressure evaluation stating that it was too painful.

Magnetic resonance imaging (MRI) of the right lower extremity showed evidence of a hematoma to the proximal right calf musculature medially, with associated myositis and subcutaneous fluid that extended to the distal calf (Figure 3). Based upon the clinical findings, pressure measurements and MRI results the patient was diagnosed with compartment syndrome to the right lower extremity and gastrocnemius muscle tear.

Rhabdomyolysis is diagnosed by serum creatine kinase (CK) levels. Rhabdomyolysis is defined as CK levels elevated 10 times the normal, (which is 55-170 units/liter) (5). CK levels are measured on a regular basis to monitor the side effects of the statin drugs, and if the level is elevated, statin therapy should be discontinued immediately. The CK level in our patient was 452 units/liter. Based upon the clinical presentation, venous studies, compartment pressures, and MRI results, emergent compartment release surgery via fasciotomy and hematoma evacuation was recommended.

**Surgical Intervention**

After general anesthesia via laryngeal mask, a 5 cm linear incision was made at the posterior medial aspect of the right lower extremity along the course of the superficial compartment. Utilizing a #10 blade, the incision was then carried down to the level of subcutaneous tissue. Blunt dissection with a mosquito hemostat, was then performed down to the level of the muscle fascia of the gastrocnemius. Utilizing Metzenbaum scissor, a fasciotomy was performed to expose the muscle belly of the gastrocnemius muscle. Inspection of the muscle, demonstrated that the gastrocnemius was partially torn about the medial head of the muscle belly. Compression of the superficial posterior compartment expressed significant amounts of clotted blood (Figure 4). The compartment was then copiously

Figure 1A. Anterior clinical view of the patient’s right lower extremity in the emergency department.

Figure 1B. Posterior clinical view.
irrigated using normal saline until no more hematoma could be expressed. Approximately 30 ml of coagulated blood was evacuated in total from the superficial posterior compartment. Intra-operatively, it was decided the tear to the gastrocnemius muscle did not require repair despite the amount of hematoma. Using #2 nylon, vertical mattress reapproximation sutures were placed in order to begin closure of the skin. Using 4-0 nylon, a continuous locking suture was performed over the incision area for final skin closure. Sterile dressings with compression and a posterior splint were applied to the right lower extremity, both to maintain the foot in a rectus position and to take tension off the gastrocnemius muscle.

Postoperative Care
The patient was admitted to the hospital after surgery for continued care with adequate analgesic and antibiotic treatment. The patient was discharged from the hospital 2 days after admission with diminished pain. The patient was seen in our office for follow-up 1 week after the fasciotomy.

No postoperative signs of infection were noted to the right lower extremity and the calf musculature was now supple on palpation. The patient was transferred from a posterior splint to a long CAM walker 1 week after the procedure. He was kept non-weightbearing for 1 additional week, then increased weightbearing to tolerance by week 2, to assist in maintaining appropriate position for the gastrocnemius muscle to heal without complications. The patient’s overall postoperative course was uneventful, and after 3 weeks, the patient had no symptoms and the skin sutures were removed. The patient transitioned to regular shoes by week 4. To date, no postprocedure complications have been seen.

DISCUSSION
The most common manifestation of compartment syndrome is that of a traumatic etiology. Approximately 75% of the cases of acute compartment syndrome are due to fractures (5). Tibial fractures are associated with 1-10% of the cases of compartment syndrome (5). The incidence of compartment syndrome in the lower extremity after a closed tibial diaphyseal fracture is 1.2% per year (3). Reports of nontraumatic compartment syndrome in the literature are much lower (3, 7). In the case presentation described above; compartment syndrome was caused via a non-traumatic mechanism.

In our case, a 47-year-old diabetic male on simvastatin suffered a non-traumatic gastrocnemius tear. It is well known that one of the most serious side effects of statin therapy is rhabdomyolysis (2, 8, 9). The precise mechanism of how this class of drugs causes rhabdomyolysis is poorly understood. We do know that muscle breakdown results in myoglobin release; this molecule becomes oxidized, creating myoglobin casts that cause renal tubular obstruction and acute renal failure (10). The tear in the medial head of the gastrocnemius muscle in our patient was believed to be
caused by concomitant statin therapy. This tear created a bleed into the superficial posterior compartment; which in turn led to the development of compartment syndrome. Compartment syndrome is defined as an elevation of interstitial pressure in a closed fascial compartment. Bleeding within the compartment creates a hematoma; which increases the pressure and restricts blood flow through the compartment leading to hypoxia (6, 11).

According to Flamini et al, diabetes mellitus type-II and concomitant use of statins for hyperlipidemia; activates a cycle of inflammation, edema, and muscle necrosis. This leads to compartment syndrome (11, 12). Acute compartment syndrome is usually characterized by severe pain, stretch pain, swelling, and sensory abnormalities (7). It is accepted in the medical community that extreme breakthrough pain resistant to analgesics is usually the first symptom of acute compartment syndrome (7). The ultimate surgical therapy for compartment syndrome is an emergency compartment release or fasciotomy, necessary to relieve the elevate pressures. The goal of decompression is restoration of muscle perfusion within 6 hours. Drop in capillary perfusion leading to oxygen deprivation of tissue and muscle necrosis are complications of untreated compartment syndrome (7, 12). In our case, fasciotomy was the procedure of choice to evacuate the hematoma and relieve the increased compartment pressure.

REFERENCES
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