CASE REPORT

Atraumatic Posterior Thigh Compartment Syndrome Presenting as an Acute Sciatic Nerve Palsy. A Case Report

Brian M. Katt, MD[1]; Nailah F. Mubin, BS[2]; Pedro K. Beredjiklian, MD[1]

Research performed at Rothman Orthopaedic Institute, Philadelphia, PA, USA

Received: 07 October 2019 *Accepted: 01 March 2020*

Abstract

Atraumatic posterior thigh compartment syndrome is a rare but serious condition. We document a case of atraumatic posterior thigh compartment syndrome that presented as an acute sciatic nerve palsy and required emergent fasciotomy. Based on this case, one should keep in mind the diagnosis of posterior thigh compartment syndrome when encountering a patient presenting with an acute sciatic nerve palsy.

Level of evidence: V

Keywords: Atraumatic, Compartment syndrome, Posterior thigh, Rhabdomyolysis, Sciatic nerve

Introduction

Increased pressures in a fascial compartment causing compromised circulation results in acute compartment syndrome (1). Compartment syndrome in the thigh is rare due to its inherently compliant borders (2) and most commonly occurs after severe trauma to the area such as with femur fractures (2-4). To our knowledge, only one other case has documented atraumatic posterior thigh compartment syndrome that presented as an acute sciatic nerve palsy (5). The patient was treated with emergent fasciotomy however his course was complicated by rhabdomyolysis induced acute renal failure that required hemodialysis.

The patient was informed that data concerning his case would be submitted for publication and he provided consent.

Case presentation

A 38 year old man was brought in by his family to the Emergency Department at 4:30 in the morning. He was complaining of sudden onset severe left groin and anterolateral thigh pain that woke him from sleep. He complained of numbness and pain that radiated down his leg to his toes. He also complained of weakness in the left leg being unable to bear weight when he awoke from sleep. He denied any recent trauma, overuse of his muscles or a previous occurrence of his current symptoms. He had been dealing with diarrhea for one month's time along with recurrent eruptions of boils on his body for the past two and half months. Some of the boils were located on his left buttock and were recently self-lanced. He had a history of IV opioid abuse but reported he had been drug free for 8 months. Currently, he denied any alcohol use but did admit to smoking fifteen cigarettes day. Two weeks prior to arrival he was employed cleaning crawl spaces.

Upon evaluation in the ED he was found to be hypotensive with a blood pressure of 106/40 mmHg, tachycardic at 125 bpm. He had no fever with a temperature of 98.5. Physical examination revealed that he was tender to palpation on the left thigh with no significant swelling. He had diffuse pain with range of motion of the left lower extremity, an intact dorsalis

Corresponding Author: Brian M. Katt, Rothman Orthopaedic Institute, Philadelphia, PA, USA
Email: briankatt@gmail.com

pedis pulse and brisk symmetric capillary refill of bilateral lower extremities. Laboratory analysis found a WBC of 39,000/uL, hemoglobin of 16 gm/dL, hematocrit of 49.2%, CPK of 36,954 iu/L, BUN/Cr of 30/2.17. Toxicology screen was negative for blood alcohol, opiates or cocaine, while urinalysis was positive for blood but negative for RBCs. Imaging tests ordered included a bedside echocardiogram which showed reduced ejection fraction and venous and arterial Dopplers which were both negative for thromboses.

The patient was given intravenous antibiotics, fluids, electrolyte repletion, furosemide with subsequent increasing blood pressures. However, it was noted that throughout evaluation his thigh continued to increase in size. At this point the underlying etiology for his high creatine kinase was unknown since the patient had no history of trauma, crush injury, burn or overt infection. Urgent CT without contrast revealed diffuse hypodensity and swelling of the left hip and thigh musculature. Follow up MRI showed extensive fluid in the thigh musculature with inter-fascial edema [Figure 1]. Fluid collection was also noted around the sciatic nerve [Figure 2]. At this point, the Orthopedic Service was consulted for the increasing leg pain with associated numbness and weakness.

Orthopedic evaluation was performed eleven hours after initial presentation to the ED. At this time, the posterior compartment was firm while the medial and anterior compartments were soft. He was tender to palpation at the left groin and medial thigh. He had pain with hip flexion but not with internal or external rotation of the hip. Edema of the left lower extremity was noted to extend from the level of the groin to the knee. Sensation was decreased along the left lateral thigh, the entire leg below the knee including the foot. Muscle strength on the right extremity was 5/5 while left hamstrings and quadriceps were 4/5. Left plantar flexion was 3/5, while dorsiflexion and extensor hallucis longus were rated 0/5. The patient had no back tenderness. He was extremely firm over his posterior thigh and this area was the most painful to palpation. A repeat CPK was at 127,503 iu/L. The diagnosis of compartment syndrome was suggested and measurement of the intra-compartmental pressure was performed. Intra-compartmental pressure was measured using the Stryker© Intra-Compartmental Pressure Monitoring System (Stryker; Kalamazoo, MI) at two different locations in the posterior compartment and was found to be 70 and 75 mmHg confirming the diagnosis of compartment syndrome. The patient was then emergently taken to the operating room for a decompressive posterior thigh fasciotomy.

Intraoperatively, a single incision fasciotomy approach was taken starting at the level of the greater trochanter and carried distally with dissection through the skin and subcutaneous tissue. The iliotibial fascia was released anterior to the intermuscular septum gaining access to the anterior compartment. The vastus lateralis was retracted anteriorly exposing the intermuscular septum. This intermuscular septum was then released decompressing the posterior thigh compartment. It was noted that the gluteus fascia was extremely tight and this was also released [Figure 3]. The Iliotibial band fascia was pie crusted to prevent laceration of the underlying vastus lateralis musculature. There was no gross purulence or necrosis noted. The sciatic nerve was palpated along its course in the posterior thigh. No collection or constriction was encountered. A muscle biopsy and tissue cultures for aerobic and anaerobic bacteria, acid-fast bacilli and fungus were taken for possible identification of etiology. The wounds were copiously irrigated with saline and then partially closed with the placement of a wound vacuum.

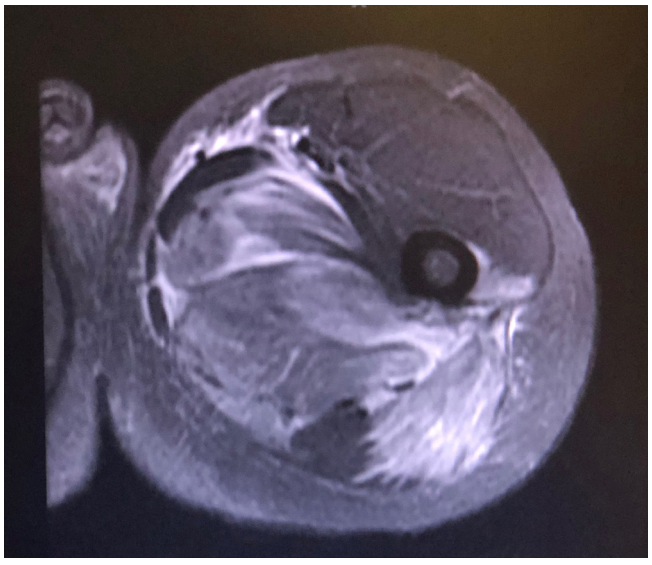

Figure 1. Axial STIR sequence MRI of proximal left thigh showing inter-fascial edema in posterior and medial compartments.

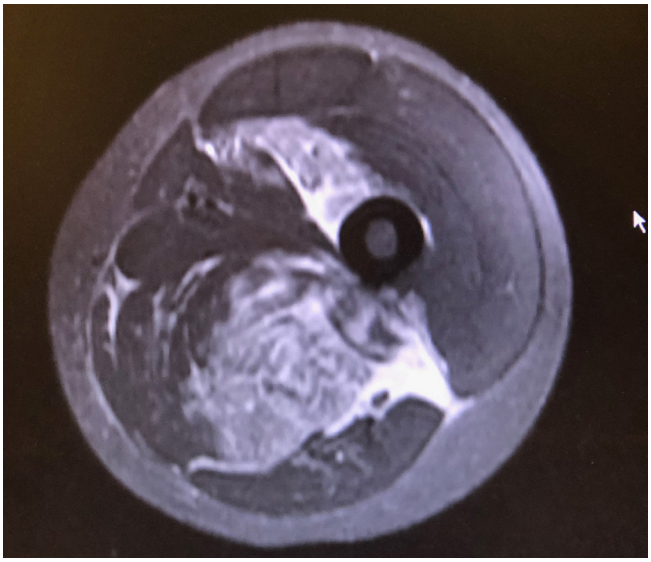

Figure 2. Axial STIR sequence MRI of middle left thigh showing extensive edema in posterior compartment and fluid collection around sciatic nerve.

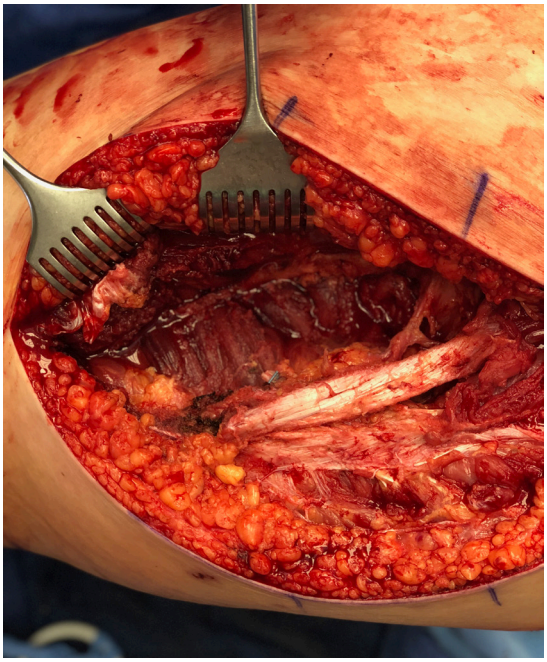

Figure 3. Left thigh s/p compartment release.

Post-operatively the patient reported significant relief compared to his pain prior to the fasciotomy. However, his hospital course was considerably complicated by acute renal failure due to rhabdomyolysis induced acute tubular necrosis. Nephrology initiated dialysis on hospital day 4 as his creatinine had increased to 7.54. The patient was taken to the OR on hospital day 5 for attempted wound closure, however the tissue was found to still be under excessive tension so only the edges were able to be closed. In addition to another central wound vacuum, a vessel loop was placed to provide tension on the wound edges so as to facilitate closure on the next attempt. On hospital day 12, the swelling on his left thigh had further decreased and the tissue was able to be fully closed without significant tension. During his hospital stay the patient continued to receive dialysis three times per week as his renal function showed no signs of improvement until hospital day 18. The patient was eventually discharged on hospital day 19 with complete return of sensation in the lower extremity, strength of 4/5 in his EHL, 5/5 in all other muscles in his lower extremity and mild pain only around his incision site. He was also given instructions to begin physical therapy and to follow up with orthopedics in 1 week. He planned to continue outpatient hemodialysis thrice weekly for at least the next two weeks.

At four weeks from initial procedure the patient presented to the outpatient clinic for follow up. He walked in under his own volition without the use of an assistive device. He no longer needed dialysis but the catheter was still in place as a precaution should his renal function decline. He had no pain in his left leg but felt weak secondary to muscle atrophy. His incision was completely healed. His sensation was equal and symmetric to the opposite side. His extensor hallucis longus was rated as a 5-/5 and all other muscles in the left leg were rated 5/5. He did not ask for pain medication.

At final follow-up 10 weeks after this index procedure he had a complete recovery. His kidney function improved to baseline and he had excellent strength in his left lower extremity. He had no pain at the incision site and his sensation was equal and symmetric to the opposite side.

Discussion

Compartment syndrome occurs when intra-compartmental pressures exceeds to a point where arterial, venous and lymphatic circulation of local tissues, muscles and nerves is compromised (1). The complications from mismanaged compartment syndrome can be severe, ranging from death, limb amputation to persistent strength and neurological deficits (4,6). Compartment syndrome is most common in the leg or forearm and less commonly found in the foot, gluteal area or thigh (7). The proposed reason for the rarity of thigh compartment syndrome is that the compartment is more accommodating due to its very nature of being larger and having more compliant borders (2). Furthermore, the majority of thigh compartment syndrome cases are provoked by some sort of trauma while a minority are due to underlying physiological anomaly such as toxic substance exposure, extended compression, exertional overuse, or coagulation disorder (3,5,8-11).

The largest systematic review of thigh compartment syndrome by Okije, et al. looking at acute cases of traumatic thigh compartment syndrome found that 90% were due to blunt trauma (3). Almost half of these injuries were associated with ipsilateral femur fractures. Eighty-six percent of fasciotomies were performed through a single incision and over half were able to be closed by delayed primary closure. Sixteen percent of these patients had a neurological complication.

A retrospective review by Schwartz et al., looked at 17 patients with acute compartment syndrome at their institution (4). Over half of these patients presented with a femur fracture of which 50% were open.

Myoglobinuria and renal failure developed in seven patients secondary to the compartment syndrome. Two other patients had myoglobinuria without renal failure. Only one of the patients who developed renal failure survived. This has been termed the “crush syndrome” manifest by myoglobinuria, acidosis and hyperkalemia and has an extremely poor prognosis.

Acute compartment syndrome of the thigh is uncommon and associated with considerable long term morbidity. Mithoefer et al., reviewed eighteen patients at an average of sixty-two months following treatment (6). Long-term functional deficits were present in almost half of the patients. Only five patients had full recovery of thigh muscle strength. Patients who had symptoms for greater than eight hours had significant more frequent muscle wasting, lower quadriceps strength and worse subjective outcome scores. This adds evidence that compartment syndrome management is a surgical emergency.

The vast majority of reported cases of compartment

syndrome of the thigh involve the anterior compartment. Of an analysis of 35 reported cases of thigh compartment syndrome by Khan et al. in 2011, only five were solely the posterior compartment (8). These five cases were associated with skeletal trauma, muscular trauma, post-surgical procedure or non-steroidal anti-inflammatory drugs. An additional case of posterior thigh compartment syndrome was reported by Zimmerman et al. in 1977 (12). This patient was struck by an automobile while riding his motorcycle. He was found to have a sciatic nerve palsy and compartment syndrome due to a hematoma from active bleeding from a lateral sacral artery diagnosed by aortogram. He underwent decompression of the left buttock and posterior thigh as well as decompression of the lower leg via a fibulectomy. At last reported follow up the patient had decreased hamstring strength, weak plantar flexion and no dorsiflexion of the ankle. He had very slight touch and pinprick hypalgesia which was not troublesome to him.

Spontaneous thigh compartment syndrome is exceedingly rare. Most cases are traumatic as mentioned above. Khan et al. described a case of a patient who underwent decompression of the anterior, posterior and medial compartments after a large hematoma was diagnosed (8). Javendi et al. described a case of a patient who was clinically diagnosed with thigh compartment syndrome and taken for urgent surgical decompression (9). His symptoms began 1 week earlier in the calf and then progressed to the thigh. He reported no change to his activity level, no recent or remote trauma, or alcohol or intravenous drug use. Gutfrayand and Philpott added a case to the literature of a 24 year old patient who developed anterior thigh compartment syndrome after dancing at a concert for several hours (10). McQueen et al. showed that young to middle aged males were more likely to be affected by compartment syndrome after traumatic injuries (13). This was postulated to be due to the relative hypertonicity of the young male's muscles not allowing adequate space for swelling secondary to localized inflammation.

Alobaidi, et al. published a case somewhat similar to ours regarding a patient who had rhabdomyolysis, acute renal failure and thigh compartment syndrome with sciatic nerve palsy (5). The inciting event was thought to be an alcohol binge. The MRI looked similar with significant muscle edema involving the left thigh. The patient stayed in the MICU for 1 month and had dorsiflexion of grade 3 on final follow up.

Compartment syndrome is a known complication that may develop from rhabdomyolysis, however as mentioned above the inverse has also been known to occur (14-17). Rhabdomyolysis is characterized by muscle damage resulting in pain and the release of muscular cell contents into the circulation. Common etiologies include drugs/alcohol, crush injuries, medications and muscle disease. It usually presents with the triad of myalgia, weakness and tea colored urine (16, 17). A high creatinine kinase, five times the normal limit (198 u/L) is diagnostic and myoglobinuria is pathognomonic (17). The interplay between rhabdomyolysis and compartment syndrome highlights the importance of considering each condition during the management of the other. Although it may seem unclear as to which came first in this case, we propose that this was a case of rhabdomyolysis induced compartment syndrome.

In the ED, the patient's initial presentation was myalgias, inability to bear weight and paresthesias. Serum CPK is known to rise with 2-12 hours of muscle injury, and his presenting CPK was 185 times more than the normal limit, indicating that it had been increasing for some time prior (17,18). His urine was amber in color and its analysis was positive for blood but negative for RBCs thus suggesting myoglobinuria Eventually over the course of a couple hours, the patient developed characteristic compartment syndrome symptoms such as pain out of proportion, paralysis, paresthesias and firmness to palpation (4). As demonstrated by his rising CPK, there was continuing muscle damage possibly due to the increasingly building pressures and progression of his compartment syndrome. As identified in reviews by Torres et al., Keltz et al., and Khan, a "second wave phenomenon" of persistently elevated CPK is another indicator of compartment syndrome development in the setting of rhabdomyolysis (16-19).

An interesting factor to consider is the impact that blood pressure has on the onset of compartment syndrome symptoms. In previously documented cases of thigh compartment syndrome, Okije et al. found the diagnosis and treatment was carried out based on clinical signs in 47% of the cases (3). Intra-compartmental pressures can also be used to confirm compartment syndrome in order to avoid unnecessary surgeries. A delta pressure less than 30 mmHg or a direct intra-compartmental pressure greater 30 mmHg is the gold standard for diagnosis (20). With compartment pressures within 20-25 mmHg of mean arterial pressure, pain develops, and capillary blood flow becomes compromised (21). Therefore, in a hypotensive patient as in this case, it is likely that tissue compromise may have occurred prior to an intra-compartmental pressure of >30mmHg. In fact, Whitesides and Heckmann recommended that a fasciotomy should be carried at any compartmental pressure within 20mmHg of the diastolic pressure (22). These findings stress that the hemodynamic status is important to take into account when determining the urgency of intervention.

Acute renal failure secondary to rhabdomyolysis complicated this patient's hospital course. The reported incidence of acute renal failure in rhabdomyolysis patients ranges from 15%-46% (23-25). Mechanisms such as direct nephrotoxicity of myoglobin, renal vasoconstriction and tubular obstruction contribute to the kidney damage (19, 25, 26). Prevention of kidney injury is focused on early and aggressive fluid repletion (26). The use of loop diuretics in rhabdomyolysis is controversial. While they increase urinary flow and thus reduce myoglobin precipitation, they also acidify the urine and exacerbate the nephrotoxic effects of myoglobin on the renal tubules (23, 26, 27). However, this effect can be offset with close monitoring of urine pH and administration of bicarb (27). Reviews have found that the use of loop diuretics did not reduce risk of mortality

in patients at risk for acute kidney injury (26, 27). Thus, in cases of rhabdomyolysis it is generally recommended to avoid using loop diuretics, or at least exercise caution and close monitoring if used (19, 23, 26, 27).

Although this patient was given IV Lasix, he was also given a bolus of sodium bicarb in the ED for his potassium abnormalities. However, his urinary pH six hours after admission was 6.0, and a pH of 6.5 is the recommended minimum to reduce acidosis induced myoglobin damage (23, 26). Therefore it is possible that while the Lasix was helpful in addressing the patients reduced ejection fraction, it may have contributed to worsening of his kidney injury. Most patients with kidney injury in the setting of rhabdomyolysis recover with no major complications if appropriately hydrated. For patients with kidney injury, the risk of mortality from the rhabdomyolysis increases (23, 26). The patient's kidney damage was one of the major reasons for an extended hospital stay as his kidney function needed to be stabilized prior to discharge. He also required follow up hemodialysis after discharge despite rigorous management of his condition.

While the etiology of the patient's rhabdomyolysis was initially unexplained, imaging was consistent with a myositis. Investigation into the patient's studies found negative blood and intermuscular microbiology cultures thus ruling out a bacterial cause for his presentation. In light of the fact that the patient had been experiencing diarrhea for one month, we hypothesize that a viral myositis triggered his rhabdomyolysis which caused his compartment syndrome. The most commonly identified cause of viral myositis has been Influenza; however, enteroviruses have also been implicated (28, 29). Of the two muscle biopsies taken, one showed focal necrosis and chronic inflammation, consistent with myositis (30). It should be noted however that muscle biopsies are not routinely indicated in the evaluation of viral myositis (28). Although not drawn, inflammatory markers such as an ESR or CRP may have been helpful to confirm an inflammatory response against the musculature. In light of the fact that the patient had a known history of intravenous drug use and a history of unexplained gluteal abscesses, a drug induced period of unconsciousness and immobilization must also be mentioned as a possible explanation to his rhabdomyolysis and subsequent compartment syndrome.

The case presented above is unique in that the patient not only presented with suspected myositis rhabdomyolysis induced compartment syndrome of his posterior thigh, but his course was also complicated by renal failure and sciatic nerve palsy. Though compartment syndrome is known to classically present after trauma to the lower leg, this case shows that it is important to have a low threshold of suspicion for compartment syndrome when patients present with severe symptomatology that is otherwise unexplained. Furthermore, while our patient regained his kidney function and muscle strength, this case reinforces that rapid management should be initiated for rhabdomyolysis and compartment syndrome to avoid the potential disastrous outcomes of end stage kidney disease, limb amputation or death.

Brian M. Katt MD[1]
Nailah F. Mubin BS[2]
Pedro K. Beredjiklian MD[1]
1 Rothman Orthopaedic Institute, Philadelphia, PA, USA
2 Rowan University School of Osteopathic Medicine, Philadelphia, PA, USA

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