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Gluteal Hematoma Causing Compression of the Common Peroneal Branch of the Sciatic Nerve

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ABSTRACT

Lower extremity injuries causing significant neurovascular compromise can be an iatrogenic or traumatic complication. Early diagnosis and intervention is the key to restoration of neurologic function of the affected lower extremity. The authors present a case of gluteal hematoma, due to a fall, causing a common causing a common peroneal nerve compression and foot drop. Common peroneal nerve palsy is usually caused by compression at the femoral head. In our case common peroneal nerve palsy was caused due to compression by hematoma at both the femoral head as well as at the level of the gluteus muscle. It was also unusual to see this diagnosis in a patient with blunt trauma due to a fall.

KEYWORDS

Gluteal Hematoma and Compression of the Common Peroneal Branch of the Sciatic Nerve; Trauma and Gluteal Hematoma and Compression of the Common Peroneal Branch of the Sciatic Nerve; Gluteal Hematoma; Compression of Common Peroneal Branch of the Sciatic Nerve.

INTRODUCTION

Lower extremity injuries causing significant neurovascular compromise can be a iatrogenic or traumatic complication. Common peroneal nerve palsy is usually caused by compression at the femoral head. In this case common peroneal nerve palsy was caused due to compression by hematoma at both the femoral head as well as at the level of the gluteus muscle.

CASE PRESENTATION

A previously healthy 22-year-old male presented to an Emergency Department (ED) via Emergency Medical Services (EMS) for the evaluation of a suspected overdose. The patient was found on the ground by his mother. Multiple bags of suspected heroin were in the vicinity of the patient. The patient's mother called 911. His mother had Narcan at home and administered it. The patient subsequently became responsive. Per EMS patient was unable to move his right lower extremity in the field. Patient did not have any significant past medical history. He did not take any medications at home. He denied having any surgeries in the past. Patient denied any history of smoking or alcohol use. He did admit to using drugs including marijuana and heroin. He did admit to injecting drugs but stated he used clean needles. Patient denied any significant family history. The patient had nausea and a few episodes of vomiting prior to arrival. He noted a loss of sensation of his right lower extremity below his knee. On physical exam, the patient was tachycardic at 110 bpm, respiratory rate 16 breaths per minute. He was noted to be hypotensive at 85/60 torr but was alert, awake and oriented X 3. He was afebrile. His O2 sat was 98% on 2L nasal cannula. Upon inspection of his back he was noted to have a large gluteal hematoma measuring 4 cm x 4 cm on his right gluteal region. The area was severely tender to palpation. His neurologic exam showed decreased muscle strength of 2/5 at his right thigh area and 0/5 on dorsiflexion and plantar flexion of his right foot. He had no sensation to light touch on his right lower extremity below his right knee.

Lab studies showed WBC: 45.5 g/dL, Hgb 15.1 g/dL, Hct 42.9

g/dL, and platelet count of 456. His basic metabolic profile showed (mEq/L): sodium of 134, potassium of 6.4, chloride of 99, bicarbonate of 16, BUN of 22 (mg/dL), Creatinine of 2.87 (mg/dL) with an anion gap of 26.4. His lactate was 7.0. His liver function test showed a total bilirubin of 0.9µmol/L, AST of 159, ALT of 365. His CK was 41,154 U/L. His toxicology screening was negative for alcohol, positive for opiates and cannabinoids. A computed tomography (CT) scan without contrast of his chest, abdomen and pelvis with thoracic and lumbar 2-Dimensional reconstruction was performed. The CT showed patchy ground glass densities in both upper lobes, right greater than left, possibly secondary to aspiration. There was also an asymmetric enlargement of right gluteal muscle with underlying areas of decreased attenuation significant for possible myositis versus gluteal hematoma (Figure 1).

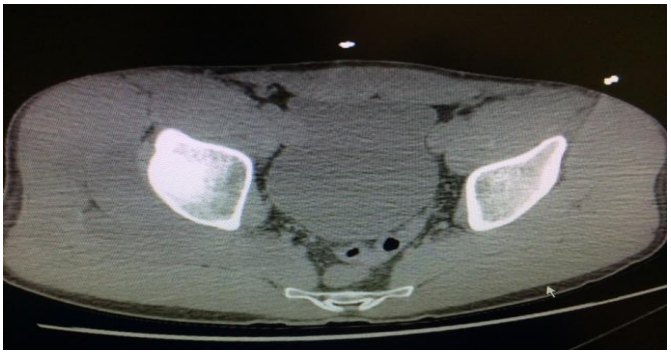

Figure 1: Asymmetric enlargement of right gluteal muscle with underlying areas of decreased attenuation significant for possible myositis versus gluteal hematoma.

Due to his significant leukocytosis, elevated creatinine, elevated lactate level, hypotension and tachycardia, the patient was treated as sepsis secondary to possible aspiration pneumonia. The patient also had underlying rhabdomyolysis, acute renal failure and hyperkalemia. The LFTs were also elevated. The patient was given antibiotics and an intravenous (IV) fluid bolus of 30 cc/kg in the ED and was admitted to the ICU. His tachycardia and hypotension improved with the fluid bolus. His neurologic deficits were discussed with the neurologist on call. No acute neurological intervention was recommended.

On Day 1 of his hospital stay, the patient's clinical condition was suggestive of severe sepsis with end organ dysfunction as indicated by his LFTs and elevated creatinine levels. His lactate, creatinine and LFT function improved with IV fluid boluses. He was placed on IV antibiotics for possible aspiration pneumonia. His rhabdomyolysis improved with IV fluids. An orthopedic consult was obtained to address the patient's right lower extremity weakness and gluteal hematoma. At the time of their consult, the patient's neurologic exam showed 2/5 muscle strength on dorsiflexion and plantar flexion of the right ankle. He continued to demonstrate decreased sensation to light touch below his right knee. On exam, the right leg compartments were soft and there was no noticeable increase in the size of the hematoma. The patient's hemoglobin remained stable. The patient was diagnosed with right lower extremity weakness secondary to peroneal nerve palsy, neuropraxia from hematoma compression and from lying on the affected extremity overnight. The Orthopedics team recommended a conservative approach and did not suggest evacuation of the hematoma. They recommended physical therapy and encouraged passive range of motion of the affected extremity.

The patient was discharged to home on day 12. He was able to ambulate without any difficulty and his gait resolved to normal as the hematoma resolved. His renal function also improved. Outpatient follow-up with orthopedics and physical therapy was arranged.

DISCUSSION

This case involves a nerve injury. There are two classification systems that apply. Seddon's classification dates from 1943, and defines three categories (neuropraxia, axonotmesis, and neurotmesis). This system is essentially anatomic. Considerably later (1978), Sunderland subdivided neurotmesis into three additional grades. Sunderland's work adds additional prognostic and treatment implications (Table 1) [1].

Table 1: Nerve injury: Seddon/Sunderland Classification.

Grade	Category
1	***Neuropraxia***
	Conduction disruption, intact axon, preserved supportive structures. Blunt trauma, stretch injury
	Prognosis: Generally full recovery without surgical intervention
2	***Axonotmesis***
	Disrupted axon, intact endoneurium; Wallerian degeneration after 1-2 weeks
	Prognosis: Variable recovery, worse prognosis for proximal injuries, worse prognosis for injuries that do not successfully re-implant in the muscle within 18 months
3	***Neurotmesis with preserved perineurium***
	Endoneurium is disrupted
	Prognosis: 60-80% recovery
4	***Neurotmesis with preserved epineurium***
	Prognosis: Nerve graft needed
5	***Neurotmesis with complete transection of nerve trunk***
	Prognosis: Bypass/jump grafting needed

Injury secondary to sharp object lacerations or evolving hematoma need to be monitored closely for possible neurotemesis requiring early surgical intervention [2]. In the case of our patient, he seemed to have signs of neuropraxia with his evolving improvement of neurologic function over time. However, serial physical exams were recommended since there was a

concern for possible axonotemesis. Nerve injuries are also classified as open vs. closed based on integrity of the overlying skin involved. Usually neuropraxia and axonotemesis are considered closed injuries. Surgical interventions are therefore indicated after observation of 3 months in grade 3 and 4 axonotemesis injury with no recovery since axonal growth rate is 1-3mm/day [3].

The sciatic nerve is formed in the pelvis by the joining of the ventral rami of roots L4-S3. It emerges from the greater sciatic foramen and enters the gluteal region by remaining beneath the gluteus maximus and passing below the piriformis muscle as an epineural sheath. The sciatic nerve then divides into the common peroneal and tibial nerve at the superior angle of the popliteal fossa. Many studies show variation in this division such as a high level of division at the levels of the pelvis and the piriformis. Such studies are usually done on cadavers; exposing the sciatic nerve at the level of the level of the piriformis by elevation of the gluteus maximus muscle [4]. This study emphasizes the relationship of the gluteus muscle to the sciatic nerve and possible points of nerve compression.

The common peroneal nerve is also susceptible to injury more so than the tibial nerve for various reasons. It is tethered proximally at the sciatic notch and distally at the popliteal fossa giving it limited mobility. It has less connective tissue and fewer and larger fascicles then the tibial division [2].

There are many traumatic and iatrogenic causes of peroneal nerve palsy. Operative causes of peroneal nerve palsy include ischemia, mechanical irritation, traction, crushing injury, and laceration [5]. According to research at a level 1 trauma center, most peripheral nerve injuries are associated with head injuries. The majority of these cases are involved in motor vehicle crashes (MVCs) and most common peripheral injuries involve the upper extremity. Most of these injuries are sustained from sharp objects penetrating through the extremity causing trauma leading to possible neurotemesis [6]. Sciatic nerve injuries are commonly seen with procedures such as intragluteal hip injections and hip arthroplasties [7]. Common peroneal nerve palsies can be seen with knee dislocations usually in cases with complete rupture of anterior cruciate ligament, posterior cruciate ligament and posterolateral corner. This was usually due to the fixed attachment of the peroneal nerve at the neck of the fibula [8]. Other causes of common peroneal nerve injury were due to spiral fractures at the distal part of the leg and high tibial osteotomy procedures [1,2].

It is unusual to see common peroneal nerve injury in patients with blunt trauma secondary to a fall. There could be multiple reasons for our patient to have symptoms associated with common peroneal nerve injury. The gluteal hematoma from the fall could have caused sciatic nerve compression at the level of the pelvis. The patient also admitted he had been lying on his right side and could have possibly compressed the nerve at the level of the fibular neck. He was unresponsive on the ground for unknown amount of time. This could have caused nerve compression at multiple sites. As discussed earlier on, the common peroneal nerve is tethered at two points, at the sciatic notch and at the popliteal fossa; both sites were affected in the case of our patient. His nerve injury was classified as closed type neuropraxia since his symptoms resolved with the decompression of the nerve. Further evaluation of nerve function could have been performed with nerve conduction studies but these studies were not available at the hospital onsite facility and would need to be performed as an outpatient.

CONCLUSION

Common peroneal nerve palsy is usually caused by compression at the femoral head. In our case common peroneal nerve palsy was caused due to compression by hematoma at both the femoral head as well as at the level of the gluteus muscle. It was also unusual to see this diagnosis in a patient with blunt trauma due to a fall.

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