Case Report

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Gluteal Compartment Syndrome, Sciatic palsy and Renal Failure Following Heroin Injection- Case Report and Literature Review

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Abbreviations: GCS: Gluteal Compartment Syndrome; ED: Emergency Department; AKI: Acute Kidney Injury; MRI: Magnetic Resonance Imaging

ABSTRACT

Background: We report a case of Gluteal compartment syndrome with sciatic palsy, and acute kidney injury, with late presentation and surgical intervention 72 hours after the onset; followed by prolonged recovery after 16 months of physical therapy.

Case presentation: A previously healthy young male has presented to the emergency department with history of right hip pain, with progressive weakness of the ipsilateral limb distally, along with impaired sensation over the distribution of the sciatic nerve, accompanied by generalized weakness, nausea and vomiting. Initially he denied any significant trauma, direct injection, recent fever or any similar history before, while his Blood work up at the ED identified elevated creatinine, myoglobin, and rising serum potassium levels; acute kidney injury (AKI) was diagnosed, and the nephrology team was involved. He was first referred to General surgery, as suspected necrotizing fasciitis, then vascular surgeons were involved due to the suspicion of saddle embolus; and finally Orthopedics referral was made 48 hours after the onset of symptoms; when only then an update for the history was made by the patient's relative; reporting intravenous drug [heroin] abuse. By then he was started on Hemodialysis for a progressively decreasing urine output, then underwent surgical decompression of GCS; almost over 75 hours after onset of symptoms. On follow up, renal function improved gradually and returned to norm by 5 weeks postop, while the lower limbs function has improved gradually, with residual weakness and numbness lasting to 16 months, yet he was able to perform his daily activities and play football.

Conclusion: The recovery of the sciatic palsy is expected to progress slowly, given the nature of the nerve injury [mechanical + chemical], and the relatively late diagnosis; which warrants a low threshold for suspecting such diagnosis in similar cases. Urgent surgical intervention, along with restoration of renal function and prolonged rehab, are essential to improve the final outcome.

Background

Compartment syndrome is considered as a surgical emergency, a favorable outcome is greatly based on earlier surgical intervention, preferably within 24 hours, as further delay is linked to permanent neurological deficits. We wish to address here the role of surgical intervention even in delayed diagnosis, along with supportive care, could prove beneficial even in such a case of mechanical-chemical etiology for sciatic palsy pathophysiology; as the history of drug addiction is usually concealed on presentation to hospitals, this may add further delays to the appropriate intervention, in addition to the systemic effect of renal impairment, manifested in uremic neuropathy, as another reason for nerve injury.

Case Presentation

A young male in his 20s, with no known comorbidities, and Unbeknown to us, he was a heroin addict, who has been on use

over the last week, and hence couldn't clearly discern the onset of symptoms, instead only became aware of it as he sobered up a day prior to the presentation to the ED, as he stated months later on a follow up visit in the clinic. He presented to the Emergency department (ED) with history of severe pain in right gluteal area, that has started suddenly; as per the first history taking, one day prior to presentation. Pain awoke him from sleep with associated with generalized weakness, nausea and vomiting. Later in the same day he developed right hip and thigh swelling and pain, associated with progressive right leg numbness and weakness. He denied any history of similar condition or intramuscular injection. Clinical examination then reported: mixed reduced sensation yet severe pain throughout the lower limb, with weakness, and tender swelling over the glutei, blood work up done at ED reveal an elevated creatinine, white cell count, and later hyperkalemia and myoglobinaemia. He was admitted under General surgery service as suspected necrotizing fasciitis, with progressively decreasing kidney output, eventually culminated in renal shutdown.

MRI pelvis was done one day later, reported: thickening and abnormal heterogeneous bright signal on the T2 fat-sat sequence in the gluteus muscles bilaterally, but more on the right side. Also left obturator internus muscle and right adductor muscles shows similar changes. There was also mild deep fascia fluid collection [thickness 9mm] is seen between the right hamstring muscles and vastus lateralis muscle extending to the superficial fascia, mild fluid collection is seen [thickness 7mm] over the vastus lateralis muscle, slightly extending to the deep fascia mainly on right side, slight on left side. Subcutaneous tissue edema mainly on the right hip posterior aspect. Mild fluid collection is seen in the posterior aspect of the spine deep to subcutaneous fat thickness 7mm. He was also seen by Vascular surgeon, as suspected saddle embolus, but was excluded by intact distal pulses, and normal vascularity of both lower limbs; concluding that such findings in the patient are not explained by an acute vascular condition. He was referred to orthopedics surgery following the MRI, about 72 hours from alleged onset of symptoms; the history was refined later; as the patient's relative reported history of addiction to heroin. Preoperative examination then revealed minimal or no pain in the described region, with firm- hard swelling over the gluteal region, anesthesia of gluteal region, posterior aspect of the ipsilateral thigh and leg, slightly intact over the anterior aspect of the thigh. He could extend the knee, yet not able to do any active movements of the hip and ankle and toes. The updated history then was in favor of a clinically progressing right Gluteal compartment syndrome, with sciatic nerve palsy; yet without completely ruling out the possibility of infections, such as necrotizing fasciitis.

Nephrology team inserted Internal jugular catheter, and started him on Hemodialysis, prior to surgery, which was done by Orthopedics oncology surgeon. Intraoperatively; through a Langenbeck approach, subcutaneous fat was found pale, fibrotic and of abnormal integrity on cutting with diathermy. The right gluteus maximus muscle was found to be under severe tension and sides its fascial covers; release of the compartments was done, along with shaving and excision of the superficial part of the right gluteus maximus muscle, which was not contractile and not bleeding [necrotic] and sent for histopathology. Severe congestion of the superior and inferior gluteal veins, yet arteries were pulsatile, as that was confirmed by sterile doppler. Dissection and release of the sciatic nerve from its exit at the greater sciatic notch, tracking its course behind the cervical trochanteric junction of the femur; tight semitendinosus fibrous bands were released over the sciatic nerve which was compressed because of the edema of the surrounding structures. Thorough wash of the wound with meticulous hemostasis was carried out. One suction drain was put inside and secured to the wound, which was closed primarily. Histopathology reported: Benign skeletal muscle bundles, fibrovascular and adipose tissue showing focal acute suppurative inflammation and focal degeneration of the skeletal muscles with focal interstitial hemorrhage, negative for granulomas or malignancy. He was discharged after his renal markers showed improvement, with Ankle and Foot Orthosis (AFO) to compensate for the foot drop and followed regularly at in the clinic. Wound healing was achieved as expected, and lower limb function was reported gradually upon physical therapy sessions. Renal function as evident by blood testing has returned to norm by 5 weeks.

He underwent Nerve conduction Study after 6 weeks from the operation, reported evidence of severe right sciatic nerve neuropathy proximal to the origin of semimembranosus muscle; with the right peroneal, superficial peroneal and sural showing no repressibility, in comparison to the left. The study was reported as "limited", since the patient was in pain, and refused to permit needle testing for more than two muscles innervated y the sciatic nerve; those had positive waves and fibrillation with single polyphasic MUAPs. By 5 months follow up, surgical scar was healed with healthy appearance, no gluteal region swelling but mild tenderness, power grade 4 in hip and knee flexion, and sciatic nerve recovery showed improvement in the form of (+3/5) toe flexion, and grade 4 out of 5 for both ankle dorsiflexion and plantar flexion, which rendered him ambulating without AFO, minimal flickering of toe extensors (1/5), and neuropathic pain over the calf and the foot. He lost follow up at 16 months; by then had the toe extensors power grade (4/5), yet with residual hypoesthesia over the anterolateral and posterior aspect right leg and foot, and occasional neuropathic sciatica pain.

Discussion

Gluteal compartment syndrome (GCS) has been considered of rare frequency, when compared to compartment syndrome in other anatomical regions, yet recent literature shows that it might not be as rare as it is thought of in reality, and warrant changing such idiom in the mind of clinicians; considering the variability of precipitating factors, being masked by the clinical presentation contexts related

to drugs abuse, would result in delayed diagnosis, and likely higher incidence of preventable complications, such as irreversible loss of gluteal muscles, sciatic nerve palsy, permanent disability, as well as overlooked kidney failure, or even death.

Reported causing factors included trauma to, or surgeries on the pelvis, abdominal aorta, superior and inferior gluteal vessels, prolonged bed rest, including such following alcohol intoxication, and drug abuse. As per February 2018; in a single reporting site such as Pubmed, 118 published studies were found to be discussing GCS, with a considerable rising trend in the last 2 decades. in this paper, we would like to shed the light over one particular etiology; drug abuse, which was found in 22 publications, with the first one dating back to 1977 by Evanski [1], attributing the onset to prolonged bed rest, following a drug overdose.

Since then, more attention is being given to the related clinical presentation, which may include history of trauma, surgery, prolonged bed rest, localized pain and or swelling, elevated blood markers for renal dysfunction, as a consequence of rhabdomyolysis, and neurological deficits, either localized for specific nerve distribution, or generalized as impaired level of consciousness. In fact, GCS is common among drug abusers; it was found in about one third of total cases operated for Heroin-related compartment syndrome in a 10-years review for a university center in the US [2]. likely as it is a common site of pressure as a gravity-dependent body part in the state of altered consciousness. However, it was reported also in the hand [3], the forearm [4], and even the brachial plexus [5]. Unfortunately, we found little about the compound effect of uremic neuropathy in the onset and continuum of the neurological findings; as though it's known that it takes a rather prolonged onset to establish the full clinical image; a recent papers had described a rapid onset in as short as 2 days [6]. Another similarity to uremic neuropathy is the two-phase recovery, where there's rapid partial improvement, followed by rather prolonged phase to get the maximal recovery. However, since the classical uremic neuropathy tends to be symmetrical; in contrast to our case, here we propose elevated creatinine, myoglobin and potassium levels, either individually or in combination, play an additive role to the worsening of the neurological symptoms, and the delay in recovery in similar cases of compartment syndrome.

Conclusion

The take home message is to have a low threshold for suspecting compartment syndrome in such cases, as gold standard treatment remains the rapid and proper decompression of the compartment, aided by supportive medical treatment, especially dialysis, [7] and physical rehabilitation. Delayed identification of the "compartment syndrome" should not justify further delays, on the bases of "poor outcome" or "waiting for renal functions to recover", as Rhabdomyolysis may contribute further to the ongoing renal dysfunction. Throughout the follow up, renal function recovery would be expected within weeks, while function of the lower limb muscles may improve partially or completely within 6-18 months. Residual neurological deficits were reported in a spectrum that ranges from foot drop, and sensory deficits at the leg and dorsum of foot, or mere hip localized pain or discomfort while sitting.

Declarations

Ethical Approval: This article describes a formal treatment method, and does not contain any studies with human participants or animals performed by any of the authors.

Informed Consent (to Participate): obtained for the "therapeutic purposes and procedures" during the treatment period, while "consent for publication" was waived; (IRB granted permission for this case report to be published on condition that no patient-identifiable data (including patient name and photograph) are included.

Consent for Publication: Granted by HMC's IRB, on the condition that "only anonymized" Data and material are available for review or publications. Availability of data and materials: data -other than script and MRI cuts- will not be shared; as the relevant contents are copied from the original medical records, which cannot be published nor anonymized.

Competing Interest

The authors declare that they have no conflict of interest.

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Authors contributions

1) Dr. K. M (Khalid mukhtar), MD: Corresponding author, and surgery team member

2) Dr. A.M (Ahmed Munir), MD: Senior surgeon and attending physician; conducted the operation and the follow up visits.

3) Dr. S.I (Sayed Intaikhab): MD, Musculoskeletal radiologist; aided in the manuscript writing and preparation of study material. *All authors have read and approved the manuscript

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