CASE REPORT

Delayed presentation of gluteal compartment syndrome presenting with peroneal palsy secondary to superior gluteal artery pseudoaneurysm following ballistic injury

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ABSTRACT

Gluteal compartment syndrome (CS) secondary to the superior gluteal artery (SGA) injury and pseudoaneurysm formation is a very rare condition. When it does occur, it usually manifests with acute and life-threatening hemorrhage resulting in early hypovolemic changes. Delayed presentation of the gluteal CS (GCS) after trauma has been described in the literature seldom and these cases were demonstrated with sciatic nerve palsy, hemodynamic instability, decreased hemoglobin levels, increasing buttock pain, and a large gluteal hematoma. In this report, we present a case of GCS presenting with the palsy of the peroneal division of the sciatic nerve secondary to SGA pseudoaneurysm following ballistic injury, with a delay of nearly 20 days in diagnosis and treatment with normal hemodynamic findings. The patient required emergent angiographic embolization and then fasciotomy which were approximately 13 days after the onset of the symptoms. The patient made a positive recovery with no further neurologic deterioration and none local wound or systemic complications. This case emphasizes the importance of early diagnosis and treatment of this rare condition.

Keywords: Ballistic injury; delayed presentation; fasciotomy; gluteal compartment syndrome; peroneal nerve palsy; pseudoaneurysm; superior gluteal artery injury.

INTRODUCTION

Compartment syndrome (CS) generally occurs at the extremities, but it has also been described at rare areas such as the gluteal region. Gluteal CS (GCS) is one of the rarest forms and most of the cases result from prolonged immobilization and local pressure on the gluteal muscles due to altered consciousness, improper surgical positioning, blunt or penetrating trauma to pelvis and buttocks, and post vascular procedures.[1–8] Given the rarity of the GCS and lack of knowledge about this entity among health-care professionals and difficulties at detailed history taking and physical examination in the presence of altered consciousness at the patients, the diagnosis is often delayed or unavailable.

Injury to the arterial wall causes continued extravasation of intraluminal blood into the extra luminal space and may result in pseudoaneurysm formation, which is contained within the surrounding soft tissues. Gluteal artery pseudoaneurysms comprises <1% of all the arterial aneurysms and the majority of them are associated with the superior gluteal artery (SGA).[9] Injury to the SGA is usually associated with pelvic fractures and could cause life-threatening brisk hemorrhage which usually manifests with acute hypovolemic changes. Its early detection may prevent dreadful complications such as the GCS, hypovolemic shock, rhabdomyolysis, acute kidney failure, and even death.

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Clinical presentation of the GCS and SGA or inferior gluteal artery (IGA) pseudoaneurysms are generally acute. Delayed presentation of GCS after trauma has been described in the literature seldom, occasionally after trauma to IGA.[9–15] Furthermore, a few cases of delayed presentation of GCS after SGA injury and pseudoaneurysm were reported in the literature.[13–18] In this report, we present a case of GCS presenting with isolated palsy of the peroneal division of the sciatic nerve secondary to SGA pseudoaneurysm with a delay of nearly 20 days in diagnosis and treatment with normal hemodynamic findings and none significant rhabdomyolysis. To the best of our knowledge, this is the first case presenting with isolated palsy of the peroneal division of the sciatic nerve. Despite this delay, the patient underwent emergent angiographic embolization and then fasciotomy approximately 13 days after the onset of the symptoms and he made a positive post-operative recovery with no further neurologic deterioration and none local wound or systemic complications.

CASE REPORT

A 24-year-old male soldier had been referred to our institute 20 days after his injury. He had involved in a conflict and sustained a ballistic injury to his right gluteal region. His bleeding and pain were controlled at the operation theater by application of direct pressure and local hemostatic powder and morphine. He was then transferred to a local state hospital where the wound was irrigated, debrided and sutured. He received daily wound dressings, intravenous (IV) fluids, and antibiotics and was "cleared" for any neurological, vascular or osseous pathologies. Hence, the limited resources of a local state hospital and absence of any further bleeding, the patient did not receive any vascular study and was not referred to a trauma center. He was discharged after 5 days to his combat unit with oral antibiotics and analgesics. He claimed that he was able to move his right hip, knee, and ankle for this period. A few days after his discharge from the hospital, he felt increasing pain and tenderness in his right gluteal region, inability to move his right hip, gradual inability to dorsiflex his right ankle, and inability to walk properly. He was otherwise healthy with no history of allergies or any medication. Due to safety regulations, transfer to a trauma center through air or road was not possible for a long period.

His vital signs were normal and hemodynamically stable at his presentation. Physical examination revealed a tense and tender non-pulsatile mass which measured approximately 12×11×7 cm at the right gluteal region. There were diffuse ecchymosis and a sutured 1×1 cm wound compatible with bullet entry. There were no other wounds, which suggested a retained bullet. There were no signs of local infection. He was unable to dorsiflex his right toe and ankle. Further examination revealed local tenderness at the anteromedial aspect of the mid femoral shaft. Neurological examination confirmed a peroneal nerve lesion with sensory deficit and a foot drop at the right side (0/5 on the Medical Research Council [MRC] scale) with intact tibial nerve motor and sensory functions. Distal pulses in bilateral lower extremities were strong with good capillary refill.

Laboratory assessments were as followed: White blood cell count 8.7 (×10^3 cells/uL), hemoglobin 10.7 g/L, platelet 543 (×10^3 cells/uL), urea 22 mg/dl, creatinine 0.83 mg/dl, sodium 138 mmol/L, potassium 5.9 mmol/L, aspartate transaminase 59 U/L, alanine transaminase 98 U/L, lactate dehydrogenase 978 U/L, and creatine phosphokinase 1787 U/L. Plain radiographs of the pelvis and right femur revealed a retained bullet at anteromedial aspect of the mid femoral shaft without any fractures and a large soft-tissue mass at the right gluteal region (Fig. 1). Computerized tomography angiography revealed right SGA pseudoaneurysm with a large hematoma formation and contrast leakage within the gluteal muscles (Fig. 2).

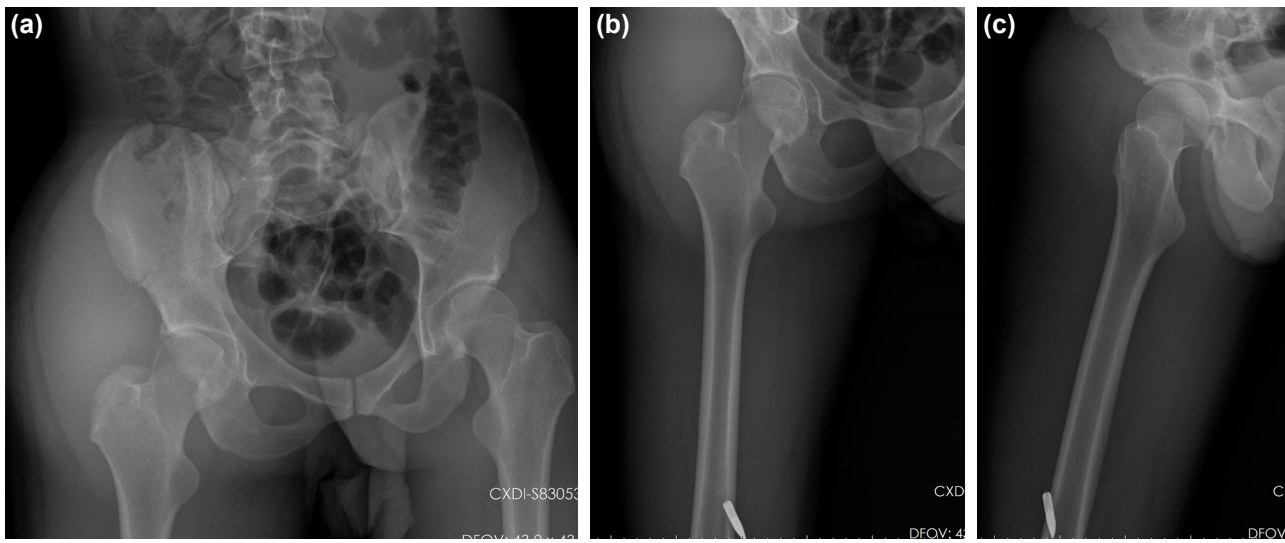

Figure 1. Anteroposterior pelvis and anteroposterior and lateral views of the right femur revealing large soft tissue mass at the right gluteal region **(a)** and a retained bullet **(b, c)** at the anteromedial aspect of the mid femoral shaft without any fractures.

Based on his symptoms, physical examination and radiologic findings, patient was diagnosed with peroneal nerve palsy due to GCS secondary to SGA injury and pseudoaneurysm. Given the prolonged history and marked clinical and radiological findings, gluteal compartment pressure measurement was not decided. The patient was taken to interventional radiology for emergent embolization, approximately 13 days after the onset of the symptoms. The patient underwent selective right internal iliac angiography using a left transfemoral approach approximately 2 h after his admittance. SGA branch of the right internal iliac artery demonstrated pseudoaneurysm. The vessel was selected with a catheter and then embolized using coils (Fig. 3). Post intervention, the patient was taken to operation room immediately for compartment release. He was placed in prone position under general anesthesia and a Kocher-Langenbeck approach was utilized to decompress the right gluteal compartments. Fascia was divided and a large amount of hematoma (nearly 1 L) was evacuated from deep to gluteus maximus. None hemorrhage was encountered after removing the tamponading hematoma. Nonviable gluteus maximus parts were debrided down to healthy muscle tissue, which amounted to approximately one third of the muscle. The gluteus mediusminimus and tensor fascia lata compartments were soft, healthy and contractile to electrocautery stimulation. The sciatic nerve was partially bruised but there was no evidence of intraneural hemorrhage. The patient was consulted to neurosurgery team intraoperatively for possible neurolysis and none further intervention was offered. The wound was closed primarily over a suction drain, which was used to monitor possible bleeding and reformation of hematoma for 48 h. Retained bullet was also removed utilizing a 3 cm incision at the anteromedial aspect of the mid femoral shaft. A small pillow was put under his right buttock postoperatively. Tension and pain at the gluteal region were immediately resolved postoperatively, but the peroneal palsy remained. At the post-operative 3rd day, electromyography was performed. It revealed high grade partial axonal damage at the peroneal branch of sciatic nerve with intact tibial nerve conduction. He was observed for 10 days with adequate hydration, IV cefazolin for 5 days and nonsteroidal anti-inflammatory analgesics. No complications were observed and then the patient then discharged to rehabilitation unit. At postoperative 1st month, local ecchymosis, edema end tension was completely resolved (Fig. 4). At post-operative 1st year the peroneal palsy persisted, and the patient was using Ankle Foot Orthosis during daily activities. He refused tendon transfer for the foot drop.

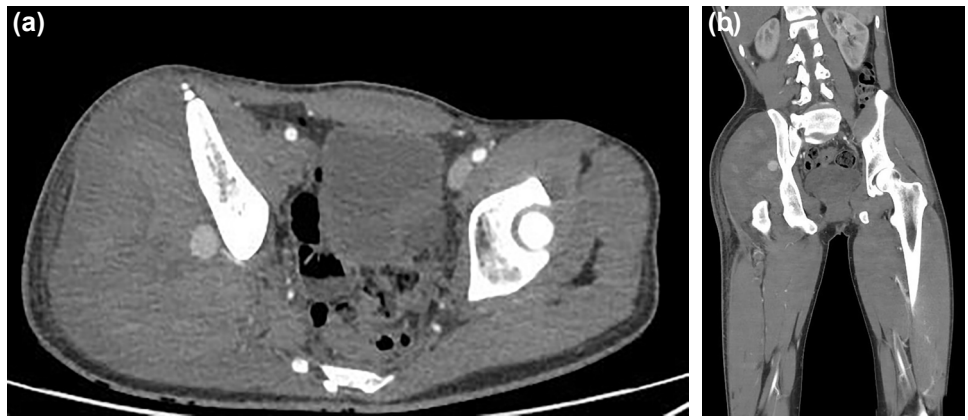

Figure 2. Axial **(a)** and coronal **(b)** computerized tomography angiography images showing right superior gluteal artery pseudoaneurysm with a large hematoma formation and a blush of intravenous contrast within the gluteal muscles.

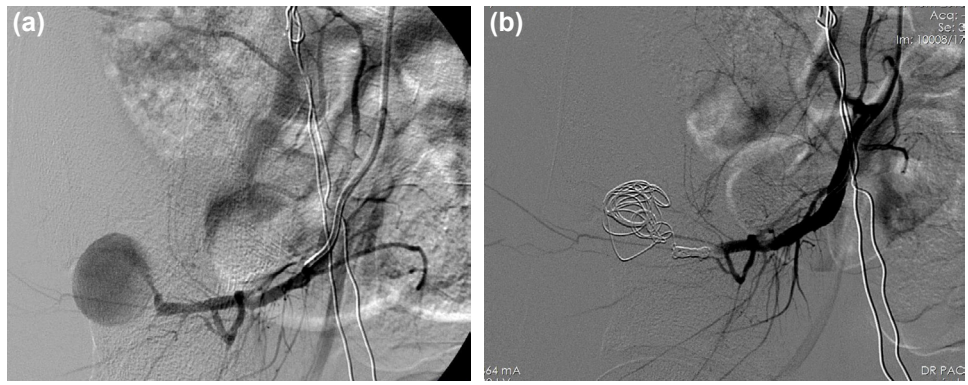

Figure 3. Selective right internal iliac angiography showing dye leakage and pseudoaneurysm formation at the right superior gluteal artery **(a)**. Angiogram after embolization showing successful embolization **(b)**.

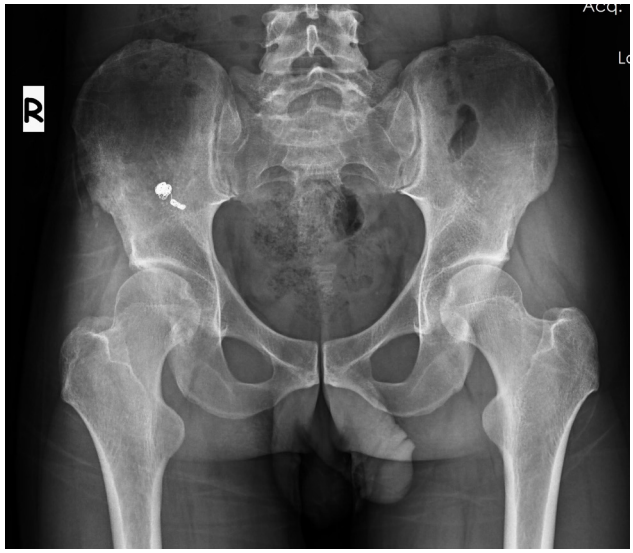

Figure 4. Anteroposterior pelvis view revealing normal gluteal soft-tissue contours and coils at the right superior gluteal artery region.

DISCUSSION

The descending aorta bifurcates at the level of the fourth lumbar vertebra and the common iliac artery bifurcates into internal and external iliac arteries in front of the sacroiliac joint. The internal iliac artery supplies the pelvic wall and the pelvic visceral organs, as well as the buttock, the genitalia, and the medial thigh. The visceral branches of the internal iliac artery are the superior vesical, inferior vesical, middle rectal, umbilical, uterine, and vaginal arteries. The superior gluteal, inferior gluteal, obturator, and internal pudendal arteries supply the limb and perineum and the iliolumbar and lateral sacral arteries are the somatic segmental branches of it.[19]

Injury of the arterial wall secondary to trauma, inflammation, or infection may result in pseudoaneurysm formation. Gluteal artery pseudoaneurysm comprises <1% of all the arterial aneurysms and the majority of them are associated with the SGA.[9,12,20] Injury to the SGA is usually associated with pelvic fractures and could cause life threatening brisk hemorrhage which usually manifests with acute hypovolemic changes or rapidly progressing hematoma formation, GCS, and sciatic nerve palsy.

CS occurs due to increased pressure within a confined space (osseo-fascial compartment), which compromises the circulation and viability of the tissues within that space. The increased pressure may originate externally, such as tight cast or dressings, prolonged positioning, and circumferential burns or internally such as tissue edema, fluid or blood extravasation or the combination of both internal and external factor.[21] It generally occurs at the extremities, but it has also been described at rare areas such as the gluteal region. Owen et al.[22] reported the first GCS case in 1978, which the patient sustained blunt trauma to buttocks and resolved spontaneously. The incidence of the GCS is not clear, though Ritenour et al.[23] reported 0.6% rate of fasciotomy for GCS amongst 336 United States military personnel who underwent fasciotomy for combat related injuries. Most of the cases result from prolonged immobilization and local pressure on the gluteal muscles due to altered consciousness, improper surgical positioning such as exaggerated dorsal lithotomy, lateral decubitus and sitting with long operative time, and epidural anesthesia. GCS cases, even bilaterally, had been reported after sedative or drug overdoses (ecstasy, heroin, etc.), carbon monoxide poisoning, hip and knee arthroplasty, arthroscopic posterior cruciate ligament repair, prolonged urologic surgeries, bariatric surgery, open or endovascular abdominal aortic aneurysm repair and open or endovascular internal iliac artery aneurysm repair. Various GCS cases had been reported after blunt or penetrating trauma to pelvis and buttocks such as pelvic or acetabular fractures, simple hip dislocation, SGA and/or IGA injury, low energy blunt traumas at the patients on anti-platelet agents or low-molecular-weight heparins or statins; and iatrogenic injuries during several procedures such as improper portal placement at hip arthroscopy, IM gluteal injection, iliac bone graft harvesting, and bone marrow biopsy. And other causes include spontaneous gluteal artery rupture in Ehlers-Danlos syndrome, necrotizing fasciitis of gluteal muscles and sickle cell anemia.[1–8,24] The gluteal compartment is composed of three separate sub compartments: The gluteus maximus, the gluteus medius and minimus, and the tensor fascia lata.[22] Sciatic nerve and the superior and inferior gluteal neurovascular bundles are extra compartmental and lays deep to the gluteal compartment but the sciatic nerve and its blood supply are prone to external compression.

Clinical findings of the GCS and SGA pseudoaneurysms are kindred: Tense and/or ecchymotic swelling at the gluteal region, disproportional pain to known injury which is resistant to opioid analgesics and exacerbated by passive hip motions. Neurological signs of sciatic nerve compression such as sensorial or motor deficits are the late findings.[8,11,21] Sensory deficits generally occur before the motor deficits sets in and some authors believe that the loss of two-point discrimination is more typical in CS than in raised intracompartmental pressure alone without CS.[25,26] Pseudoaneurysms may demonstrate thrills, bruits, or pulsations during palpation and auscultation. Occasionally pseudoaneurysms may be mistaken for soft tissue tumors, or an abscess because of the resembling clinical findings.[12] Clinical presentation is generally acute. Delayed presentation of GCS after trauma has been described in the literature seldom, occasionally after trauma to IGA and these cases were demonstrated with the symptoms of sciatic nerve palsy,[9–11] hemodynamic instability or dramatically decreased hemoglobin levels,[9,10,12] increasing pain and large gluteal hematoma,[13,14] hip and pelvic pain.[15] Furthermore, a few cases of delayed presentation of GCS after SGA injury and pseudoaneurysm were reported in the literature and these cases were demonstrated with sciatic

nerve palsy,[16,17] hemodynamic instability or dramatically decreased hemoglobin levels,[16–18] increasing pain and large gluteal hematoma.[13] The delay between trauma and diagnosis can vary from a few days to several years.[11,12,15]

Our patient presented with a delay of nearly 20 days in diagnosis. Increasing pain and a large gluteal hematoma, normal hemodynamic findings, palsy of the peroneal division of the sciatic nerve, decreased hemoglobin level, slightly elevated liver enzymes and elevated lactate dehydrogenase, and creatine phosphokinase levels were the brief findings. Whiteside et al.[27] revealed that muscles and nerves tolerate ischemia for up to 4 h with limited sequelae and 8 h of ischemia results in irreversible damage. Although our patient had not received a fasciotomy and thorough debridement initially, he had not sustained significant muscle necrosis the peroneal nerve palsy. This condition may be attributed to somehow slowly enlarging SGA pseudo aneurysm and the subacute or delayed settling of the GCS. IV fluid replacement at his primary treatment may have prevented acute kidney failure and further systemic complications. A further question raised by this case is what caused the palsy of the peroneal division rather than the both peroneal and tibial divisions. Yuen et al.[28] reported that the peroneal division has greater susceptibility to palsy than the tibial division and related this condition to three causes: First, the peroneal division has larger fascicles with less supportive connective tissue compared to the tibial division. Second, the peroneal division is more securely fixed at the sciatic notch, making it more vulnerable to traction injuries. Third, the peroneal division lies superficial to the tibial division in the hip and proximal thigh regions. To the best of our knowledge, this is the first case presenting with isolated palsy of the peroneal division of the sciatic nerve occurring secondary to GCS.

Various reports suggested the use of the Wick catheter to measure compartment pressures, especially for the unconscious patients, when the detailed history taking impossible and physical examination is ambiguous. The literature is debatable about the threshold for a fasciotomy but pressures greater than 30 mmHg are suggestive of CCS and fasciotomy is indicated.[4–6,13,21,29] Clinical diagnosis remains the optimal way for the diagnosis. Given the prolonged history and marked clinical and radiological findings at our patient, gluteal compartment pressure measurement was not decided.

We have applied endovascular embolization of the SGA followed by open fasciotomy for our patient. Fasciotomy for the GCS can be performed with a posterolateral approach to the hip including Kocher-Langenbeck, Gibson or Henry's modifications.[1,30] It is essential to decompress all three compartments. All approaches require extensive soft tissue dissection and it is generally difficult to control bleeding because of the retraction of the neurovascular bundle in to the pelvis. Angiographic embolization of the injured vessels is an effective technique that may provide lifesaving bleeding control in areas that are difficult to access surgically.[4,14,18,20,31] Smith et al.[21] debated the priority of angiographic or surgical intervention at the setting of GCS and suggested prior angiography in the presence of hemodynamic instability. They had ill-advised angiography in the presence of hemodynamic stability and GCS, as it causes delay in fasciotomy. We recommend prior angiography and embolization should be considered where the optimal facilities and conditions exist and prompt angiographic intervention could be applied to avoid the risk of profuse bleeding, secondary poor visualization, and risk to the sciatic nerve as some authors advocated.[17,32,33]

Lawrence et al.[8] debated fasciotomies at the treatment of cases of CS with delayed presentation and suggested that fasciotomy must be considered where optimal facilities and conditions exist, as in our case. Despite the myonecrosis at the gluteus maximus, which required debridement approximately one third of the muscle, his tension and pain at the gluteal region were immediately resolved postoperatively, which supports the choice of surgical decompression. Furthermore, fasciotomy may have avoided further compression of the sciatic nerve and possible palsy of the tibial division too.

When the bullet enters within the tissue it advances through at a certain velocity disrupting and necrotizing the surrounding tissues, creating the permanent cavity and decelerates due to counteracting forces. Low velocity bullets, such as the hand-gun bullets, have weaker blast forces and cause their main effect by the permanent cavity. High velocity bullets, such as the rifle bullets, create similar permanent cavities as the low velocity bullets along the first 10–12 cm in the soft tissues. After this distance, the temporary cavity or the blast effect occurs due to the pressure waves produced by the bullet in soft tissue and the radial stretching.[34] The temporary cavity size may be tenfold the diameter of the bullet, but it lasts a few milliseconds and then it collapses towards the permanent track. Because of the high elastic recoil capacity of the soft tissues, there may be reduced tissue damage. This correlates with the theory that identifies the temporary cavity as not always reliable wounding mechanism.[35] If the bullet is retained in tissues, it is called a penetrating wound and if exits the body, it is called a perforating wound.[36] Our patient sustained a penetrating missile wound with a 7.62 mm caliber assault rifle bullet. The distance between the skin entry and the bullet's position was measured 31.3 cm on the CT images. Although this distance is sufficient to create a moderate to severe temporary cavity in the femoral region, there was no significant soft tissue damage. Patient's relatively mild soft-tissue injury at the femoral region could be the result of a long-distance shot, or a ricocheting bullet, which could decrease the velocity of the bullet, thus acting like a low velocity missile injury. It should be kept in mind that thorough irrigation and debridement of the permanent cavity including the removal of devitalized tissues and foreign objects like clothing is essential.

Conclusion

Given the rarity of the GCS and SGA pseudoaneurysms and the lack of knowledge about these entities among the healthcare professionals, the diagnosis is often delayed or unavailable. All trauma surgeons should be aware of the possibility of GCS in patients who have an acute pelvic trauma with pain and swelling of the gluteal region. Delay of the diagnosis and treatment can result in hypovolemic shock, peroneal or sciatic nerve palsy, irreversible loss of gluteal musculature, rhabdomyolysis, renal failure, and even death. Combined endovascular embolization and fasciotomy are a safe and effective option and must be considered where the optimal facilities and conditions exist.

Informed Consent: Written informed consent was obtained from the patient for the publication of the case report and the accompanying images.

Peer-review: Internally peer-reviewed.

Authorship Contributions: Concept: A.B.B.; Design: A.B.B.; Supervision: A.B.B.; Resource: A.B.B., N.S.K.; Materials: A.B.B., N.S.K.; Data: A.B.B., N.S.K.; Analysis: A.B.B., Ç.N.; Literature search: A.B.B., Ç.N.; Writing: A.B.B.; Critical revision: Ç.N., Y.E., D.B.

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OLGU SUNUMU - *ÖZ*

Ateşli silah yaralanması sonrası gelişen süperior gluteal arter psödoanevrizmasına bağlı peroneal sinir lezyonunun eşlik ettiği gecikmiş gluteal kompartman sendromu

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Süperior gluteal arter hasarına ve psödoanevrizma oluşumuna sekonder gluteal kompartman sendromu çok nadir görülen bir durumdur. Meydana geldiğinde, genellikle erken hipovolemik değişikliklere neden olan, akut ve hayatı tehdit edici bir kanama ile kendini gösterir. Literatürde travma sonrası gluteal kompartman sendromunun gecikmiş sunumu nadiren tanımlanmıştır ve bu olgularda genellikle siyatik sinir felci, hemodinamik instabilite, azalmış hemoglobin düzeyleri, artan kalça ağrısı ve gluteal bölgede gelişen büyük bir hematom bulguları bildirilmiştir. Bu yazıda, tanı ve tedavide yaklaşık 20 günlük bir gecikmeyle merkezimize normal hemodinamik bulgular ve siyatik sinirin peroneal bölümünün lezyonu ile başvuran, ateşli silah yaralanması sonrası gelişen süperior gluteal arter psödoanevrizmasına ikincil bir gluteal kompartman sendromu olgusu sunulmaktadır. Semptomların başlamasından yaklaşık 13 gün sonra hastaya acil anjiyografik embolizasyon ve fasyotomi uygulanmıştır. Hasta cerrahi sonrası ek nörolojik hasar yaşamadan hızlı bir iyileşme süreci yaşamış ve bölgesel yara ile ilgili komplikasyonlar veya herhangi bir sistemik komplikasyon görülmemiştir. Bu olgu sunumu, bu nadir durumun erken tanı ve tedavisinin önemini vurgulamaktadır.

Anahtar sözcükler: Ateşli silah yaralanması; fasyotomi; gecikmiş başvuru; gluteal kompartman sendromu; peroneal sinir felci; psödoanevrizma; superior gluteal arter yaralanması.

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