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CASE REPORT
Cosmetic

Bilateral Sciatic Neuropathy following Gluteal Augmentation With Autologous Fat Grafting

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Summary: As gluteal augmentation continues to gain in popularity among patients seeking aesthetic enhancements, a thorough knowledge of the postoperative complications associated with this procedure is crucial. This case report concerns a 31-year-old woman who suffered bilateral foot drop secondary to sciatic neuropathy and as a result was wheelchair-bound for several months, following gluteal autologous fat grafting in the Dominical Republic. One year later, the patient had persistent left foot drop and sensory deficits. This is a devastating but seldom reported complication that all plastic surgeons need to be aware of when performing this operation. *(Plast Reconstr Surg Glob Open 2018;6:e1696; doi: 10.1097/GOX.0000000000001696; Published online 19 March 2018.)*

Augmentation gluteoplasty is a rapidly growing aesthetic procedure. In the pursuit of beauty and cultural aesthetic ideals, numerous techniques of gluteal augmentation and contouring have been described in the literature, among which autologous fat grafting is widely implemented with excellent results and very high patient satisfaction rates.

CASE PRESENTATION

A 31-year-old healthy woman underwent autologous gluteal augmentation with fat grafting and abdominal liposuction in the Dominican Republic. Following the procedure she experienced significant bilateral foot drop and paresthesia and was wheelchair-bound.

She presented to our emergency room on postoperative day 8, and on examination, her gluteal areas were soft bilaterally, with mild induration and moderate swelling and tenderness. There was no evidence of infection or hematoma. She demonstrated full active motor function in her bilateral lower extremities up to the level of the ankles. Her foot dorsiflexion, plantarflexion, inversion, and eversion were markedly weak, worse in the left than the right. Sensation to light touch was globally reduced in her feet. Achilles and plantar reflexes were unable to elicit.

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An electromyogram revealed bilateral axonal sciatic neuropathy. The magnetic resonance imaging of the lumbar spine and ultrasound of bilateral lower extremities were unremarkable. The abdominal CT scan revealed a right lower abdominal hematoma, which was percutaneously drained using an 18-gauge needle. The pelvic CT scan revealed significant edema of the gluteus maximus muscles bilaterally. In addition, large well-demarcated hypodense areas and circumferential calcifications were seen in the bodies of the gluteus maximus muscles bilaterally, representing sites of intramuscular fat injections and necrosis, respectively (Fig. 1).

Given the delayed presentation and resolving gluteal edema, gluteal compartment fasciotomies and sciatic nerve decompression were not performed. The patient was hospitalized for 5 days and was treated with high-dose steroids and physical therapy. The motor function of her feet improved only mildly to dorsiflexion of 3 of 5 and 4 of 5 in the left and right, respectively, plantarflexion of 4 of 5 bilaterally, also inversion and eversion of 3 of 5 bilaterally. Her sensory examination was unchanged. She was wheelchair-dependent at the time of her discharge to an acute rehabilitation facility.

Following a 3-month course of outpatient physical therapy, the patient was able to walk without assistance. One year from her presentation to the emergency room, the sensory and motor function of the right foot had recovered completely. In the left, the sensory deficits and foot drop had persisted, and her motor function was 4 of 5 throughout.

DISCUSSION

Despite the relative paucity of data, the complication profile of gluteal augmentation has been reviewed. Sinno

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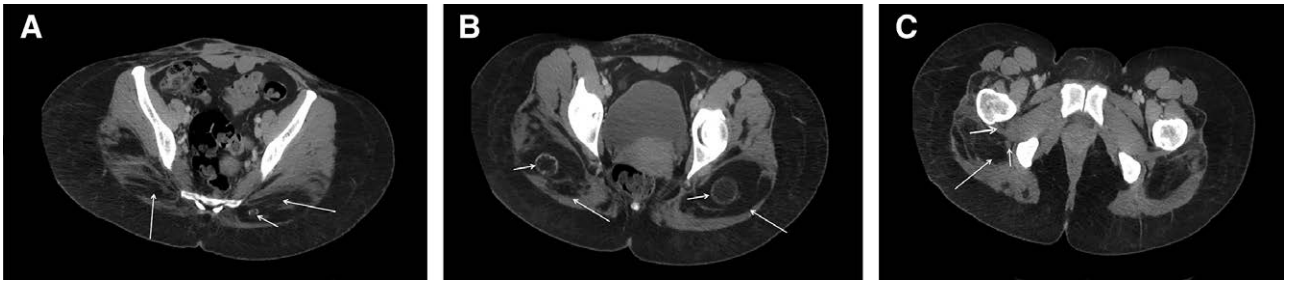

Fig. 1. CT scan of the pelvis on postoperative day 8. A, Imaging of bilateral proximal gluteus maximus muscles demonstrating gross edema (long arrows) with a focal area of calcification on the left (short arrow). B, Imaging of the of the midportion of the gluteus maximus muscles demonstrating well-demarcated areas of hypoattenuation (long arrows) indicative of intramuscular fat injections and calcifications within the deposits (short arrows). C, The close relationship of the right sciatic nerve and inferior gluteal vessels (short arrow) with the deep surface of the indurated gluteus maximus muscle (long arrow) in the lower pelvis. The sciatic nerve courses posterior to the obturator internus muscle (thick arrow).

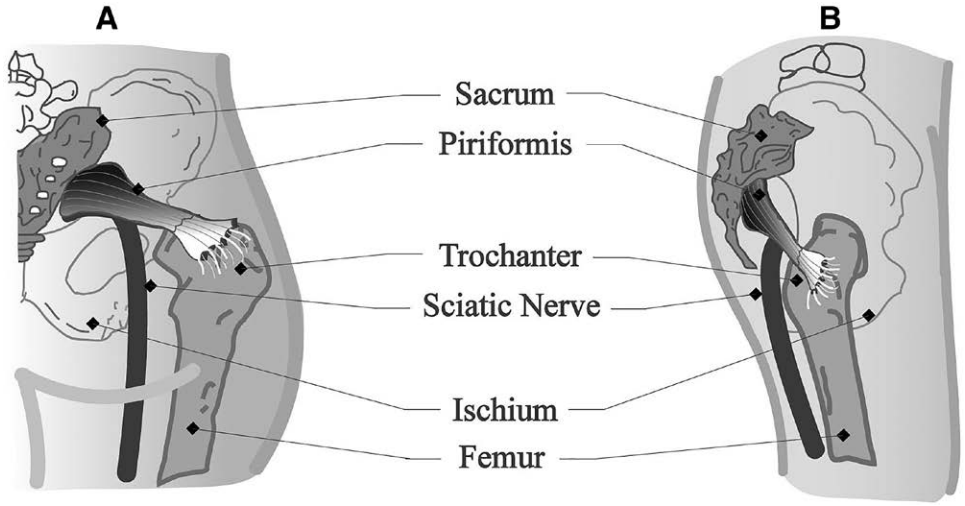

Fig. 2. The sciatic nerve is most susceptible to direct trauma at the sciatic "cutaneous projection" midway between the ischium and the greater trochanter (A). The narrow anatomic relationship of the key landmarks of the inferior gluteal aspect is shown (B).

et al.[1] conducted an extensive database analysis and found the overall complication rate of gluteal fat grafting to be 9.9%, whereas the rate of sciatic pain was reported at 1.7%. In their world literature review, Oranges et al.[2] reported a complication rate of 10.5% and 1 incident of bilateral sciatic nerve axonotmesis (0.04%) following autologous fat grafting. In addition, they reported 14 instances of sciatic nerve symptoms (0.3%) and 1 case of neuroapraxia (0.02%) after gluteal augmentation with implants.[2]

The pathogenesis of sciatic nerve injury in this case is not entirely understood, as at the level of the gluteus, the sciatic nerve is susceptible to both direct trauma and compressive injury. As illustrated by many reports of sciatic neuropathy following gluteal intramuscular injections in the literature, the anatomic understanding of the sciatic nerve is crucial when intervening in the gluteal region.[3,4] In the gluteal region, the sciatic nerve is reliably and entirely covered by the gluteus maximus, and it is most commonly found in the subpiriformis canal. It courses distally through the ischio-trochanteric channel where it is most susceptible to injury, at the sciatic "cutaneous projection" midway between the ischium and the greater trochanter, in the lower gluteal region (Fig. 2).[4,5] Respecting these anatomic landmarks by good technique is essential to avoiding injury to the sciatic nerve.

With regard to compressive injury, the association between compartment syndrome and sciatic neuropathy is well known.[6,7] Both the nerve and its blood supply are vulnerable to compressive forces.[6,8] Any delay in decompressing the gluteal compartments and sciatic nerve may lead to irreversible ischemia. Gluteal intracompartmental pressures are often unreliable; therefore, the diagnosis of gluteal compartment syndrome is based on the clinical findings and examination.[9,10] Clinical suspicion should lead to prompt exploration in the operative room and fasciotomies of the 3 compartments (gluteus maximus, gluteus medius–minimus, and tensor fascia lata) to assess the viability of the musculature. Two common approaches described in the literature for adequate exposure are the Kocher-Langenbech and the "question mark" incisions.[10,11]

Following the fasciotomies, release of the midline insertion of the fascia to the sacrum would expose the sciatic nerve for decompression.[10]

The reported sequela of sciatic neuropathy is variable. It ranges from mild neuropathy of the peroneal distribution to paralytic foot drop and more proximal lower extremity paralysis. However, our patient presented with signs of partial peroneal neuropathy, which is not uncommon in this setting.[12] The peroneal is the most commonly and severely affected division of the sciatic nerve in cases of traumatic and compressive injury.[13] This is attributed to its innate neurologic structural characteristics—for instance, fewer fascicles—smaller blood supply, and gross anatomic features such as its superficial location and multiple fixation points, all of which render it highly prone to injury.[13]

To the best of our knowledge, there is no current literature on the management of sciatic neuropathy in the setting of late presentation following gluteal fat grafting. On a broader topic, several authors advocate against fasciotomies in the treatment of compartment syndrome with delayed presentation (longer than 35 hours from the onset of compartment syndrome) due to high complication rates and limited evidence of subsequent functional improvement.[8,14] We, therefore, recommend to attempt nonoperative management in this setting with aggressive physical therapy and a steroid regimen.

Although data are limited, in cases where the ischemic injury to the sciatic nerve is reversible, some degree of recovery can be expected, such as here. Lastly, successful sciatic neurolysis and scar release several months following gluteal compartment syndrome has been reported in the orthopedic literature; however, it was not attempted in our patient as the evidence is yet again limited.[15]

CONCLUSIONS

A young woman suffered bilateral foot drop following gluteal augmentation with autologous fat grafting in the Dominican Republic. Although with time and physical therapy her symptoms improved and she was able to ambulate independently, she has persistent left foot motor and sensory deficits 1 year following the operation. This is a devastating and potentially preventable complication of a popular aesthetic procedure, which all surgeons need to be aware of.

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