Bilateral Sciatic Neuropathy after an Autologous Breast Reconstruction in a Massive Weight Loss Patient

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INTRODUCTION

Effective surgical positioning combines maximal exposure, minimal tension, and comfort for the operator and emphasizes patient’s safety. Operative positions such as lithotomy, supine, or prone can lead to numerous combinations of stretch and compression forces. Compression forces cause a spectrum of nerve injuries during long surgical procedures. Ulnar, radial, lateral femoral cutaneous, and peroneal nerve injuries are well described in the literature. However, sciatic postoperative neuropathy has rarely been reported. Furthermore, injuries can range from low-grade intraneural blood vessel disruption to hemorrhage or myelin necrosis. Recovery may take hours, weeks, or even months. In addition, one of the rarely described risk factors for an intraoperative positional nerve injury includes a history of weight loss. This risk factor is of contextual importance because the incidence of bariatric surgery has been stable at approximately 113,000 cases per year in the United States alone. There has been only 2 prior case reports of a bilateral sciatic neuropathy in a weight loss patient. We report a rare finding of a bilateral sciatic neuropathy in a weight loss patient after an autologous breast reconstruction procedure.

CASE

A 34-year-old woman presented for right supercharged pedicled (transverse rectus abdominis) flap breast reconstruction. Her history was marked for sleeve gastrectomy 2 years earlier resulting in 105 pound weight loss 1 year before breast reconstruction. During the procedure, the patient was in the supine position for 8 hours and in the semirecumbent position for an additional 2 hours with the torso flexed at 30 degrees and knees flexed at approximately 45 degrees in addition to standard padding. Postoperatively, the patient was found to have loss of sensation and motor paralysis distal to her knees bilaterally. Pain sensation was preserved distally and no other neurological abnormalities were noted. Laboratory tests, magnetic resonance imaging, electromyography, and nerve conduction studies all revealed no evidence of neurological lesions and peroneal or lumbosacral radiculopathy. Motor strength gradually returned to her lower extremities over 4–5 weeks, whereas sensory function continued to improve over 7 weeks. The patient had complete neurological recovery 2 months postoperatively.

Disclosure: The authors have no financial interest to declare in relation to the content of this article. The Article Processing Charge was paid for by the authors.
eratively the patient was found to have bilateral motor and sensory loss from her knees and extending distally bilaterally. Physical examination revealed 2/5 power bilaterally in the quadriceps and 0/5 power on dorsiflexion and plantar flexion of the ankle joints bilaterally. Pain sensation was preserved distally in both lower extremities. Deep tendon reflexes were 2+ bilaterally. No muscle rigidity or involuntary movements were noted.

An electrolyte panel was ordered and did not reveal any abnormalities. Magnetic resonance imaging of the spine demonstrated no lesions. Electromyography (Table 1) and nerve conduction (Table 2) studies 6 weeks postoperatively showed no evidence of a peroneal or lumbosacral radiculopathy.

Over the course of 4–5 weeks postoperatively, bilateral lower extremity motor strength returned gradually with eventual progression to full recovery. Sensory function continued to improve over 7 weeks and returned to the normal preoperative level.

**DISCUSSION**

Postoperative sciatic neuropathy is a rarely reported complication in the literature—with bilateral sciatic neuropathy reported only twice. To the best of our knowledge, the cal complication was peripheral neuropathy followed by encephalopathy and even some reports of neurological emergencies.6 Dr. Clark led a discussion about the complications, pathogenesis, prevention, and treatment of neuropathy in bariatric surgery patients.7 Essentially, the etiology of neuropathy in these patients is not fully understood, although the 2 leading mechanisms are thought to be deficiency of micronutrients and vitamins versus inflammatory changes leading to axonal damage.

With a significant number of patients undergoing both bariatric and autologous breast reconstructive surgeries, there is a potential for an increased incidence of sciatic neuropathy. We believe that the identification of this potential complication and its prevention begins at the physician’s office during the preoperative evaluation. It is important to have a detailed discussion with the patient about what type of weight loss surgery he or she received. Mason et al. point out in their publication that the type and severity of metabolic complications resulting from bariatric surgery depend on the particular operation performed, with gastric banding having lower complications than a Roux-en-Y gastric bypass. If in fact such patients are at higher risk for neuropathy due to vitamin deficiency, then it may be valuable to include vitamin levels in the preoperative work-up and replacing them as a necessary part of optimizing the patient for surgery. Unfortunately, there are no particular nutritional supplementation protocols to recommend or follow as much of the knowledge we have on this subject is still theory.

Furthermore, to decrease the risk of compressive neuropathy intraoperatively, we recommend frequent repositioning or shifting the patient on the table every 3 hours to change the points of pressure and additional padding.

Table 1. Electromyography Study 6 Weeks Postoperatively Was Normal, Showing No Fasciculations or Spikes and Read as Normal (Needle Electromyography Examination)

| Muscle Pattern                  | Spontaneous Activity |
|---------------------------------|----------------------|
|                                | Fibs/PSW | Fascics    |
| Biceps femoris (short head) R normal | None      | None       |
| Gastrocnemius (medial head) R normal | None      | None       |
| Vastus medialis R normal        | None      | None       |
| Tibialis anterior R normal      | None      | None       |
| Peroneus longus R normal        | None      | None       |
| L4 paraspinal R normal          | None      | None       |
| L5 paraspinal R normal          | None      | None       |
| Biceps femoris (short head) L normal | None      | None       |
| Gastrocnemius (medial head) L normal | None      | None       |
| Vastus medialis L normal        | None      | None       |
| Tibialis anterior L normal      | None      | None       |
| Peroneus longus L normal        | None      | None       |
| L4 paraspinal L normal          | None      | None       |
| L5 paraspinal L normal          | None      | None       |

PSW, positive sharp waves.

Table 2. Nerve Conduction Study 6 Weeks Postoperatively (Normal Velocity: 50–60 m/s)

| Nerve and Site | Velocity (m/s) |
|----------------|----------------|
| Peroneal R     |                |
| Ankle          | —              |
| Fibular head   | 58             |
| Knee           | 55             |
| Tibial R       | —              |
| Ankle          | —              |
| Popliteal fossa| 48             |
| Peroneal L     |                |
| Ankle          | —              |
| Fibular head   | 47             |
| Knee           | 45             |
It is important to mention that our recommendations are based on expert opinion and not evidence based. Much of our knowledge about neuropraxia secondary to compression comes from tourniquet use during orthopedic surgeries. The major problem is that there are no clear guidelines as to appropriate inflation pressures and durations because the data are scattered and unclear. Adding to our dilemma are the physical constraints associated with repositioning a patient to relieve pressure while still maintaining adequate positioning for good cosmesis and not prolonging the operation time when compared with the relative ease of inflating and deflating a tourniquet cuff.

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