Vulnerability of the Femoral Nerve During Complex Anterior and Posterior Spinal Surgery

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Abstract

Background: Femoral nerve palsy is not a common adverse effect of lumbar spinal surgery.

Objective: To report 3 unique cases of femoral nerve neuropathy due to instrumentation and positioning during complex anterior and posterior spinal surgery.

Methods: Case series

Results: All 3 patients demonstrated femoral nerve neuropathy. The first patient presented postoperatively but after 6 months, the palsy resolved. Femoral nerve malfunctioning was documented in the second and third patients intraoperatively; however, with rapid patient repositioning and removal of offending instrumentation, postoperative palsy was avoided.

Conclusions: Use of motor evoked potential monitoring of the femoral nerve during surgery is vital for the prevention of future neuropathies, an avoidable complication of spinal surgery.

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INTRODUCTION

Although uncommon, femoral nerve neuropathy can be an adverse outcome during spinal surgery. Review of the literature indicates that femoral nerve palsy is consistently related to positioning during anterior lumbar surgery. In this report, we document 3 unique cases of femoral nerve palsy caused by intraoperative retraction and manipulation of the nerve rather than positioning.

CASE REPORTS

Case 1

A 51-year-old woman presented with recurrent intractable low back pain after an anteroposterior lumbar fusion for medically refractory diskitis at L5 to S1. Imaging studies showed pseudoarthrosis at L5 to S1 with concurrent spondylolisthesis at L4 to L5. After failure to improve with nonoperative treatment measures, the patient underwent a revision anterior and posterior fusion

from L4 to S1. Intraoperatively, a retractor was placed on the left psoas muscle laterally, and another retractor blade was placed medially to maintain exposure of the spine. EMG monitoring, both free-running and evoked, showed no abnormalities, but proximal muscles were not being tested.

Immediately postoperatively, the patient had motor and sensory signs of femoral neuropathy: left quadriceps (2/5), iliopsoas (4/5), and decreased sensation over the anteromedial aspect of her left leg. An immediate computed tomography scan of the abdomen/pelvis and spine showed only postoperative changes, with the bone graft and instrumentation in good position (Figure 1). The patient was returned to the operating room to rule out compressive neuropathy, and no evidence was found of either hematoma or extrinsic compression of the femoral nerve. Six months postoperatively, she had complete resolution of her femoral neuropathy and ambulated without assistance.

Case 2

A 24-year-old man presented with pain and progressive weakness of the lower extremities (left > right) with an L5 to S1 grade IV spondylolisthesis. He underwent an

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Figure 1. Postoperative films of case 1 before the revision surgery. Although the films showed good positioning of the instrumentation, the patient developed femoral neuropathy that resolved 6 months later.

anterior/posterior L4 to S1 decompression and fusion. Motor-evoked potentials (MEPs) were recorded throughout the procedure because of the high-grade spondylolisthesis. During the anterior graft placement and manipulation of the spine, MEPs from the left quadriceps and abductor hallucis disappeared, and it was presumed to be caused by the retraction of the femoral nerve to the left psoas muscle. Both irregular MEPs were resolved after release of the retractor. The procedure was completed without further abnormalities, and the patient awoke without signs of femoral neuropathy and with preoperative weakness resolved. Postoperative films showed normal recovery (Figure 2). The patient required no postoperative physical therapy or extra management.

Case 3

A 59-year-old man presented with subacute cauda equina syndrome, neurogenic claudication, and intractable pain from spinal stenosis and adjacent level deterioration several years after an L4 to L5 fusion and kyphotic deformity. After an uncomplicated anterior revision surgery, the patient also underwent a staged delayed posterior arthrodesis and decompression. During this surgery, neurophysiologic monitoring of the quadriceps muscle showed a decrease in the quadriceps responses, but no such abnormality in the anterior tibialis. The surgeon was not manipulating the spinal nerve roots during this decrease and questioned whether it may be

Figure 2. Postoperative films of case 2. The posterior instrumentation of L4 to S1 and the anterior instrumentation of L4 to L5 shows good placement. The patient showed no further neuropathies.

caused by direct compression of the femoral nerve in the inguinal region from patient positioning. The patient was repositioned, all extrinsic compression along the course of the femoral nerve was removed, and the response of the quadriceps returned to the baseline potential. Despite the transient decrease in femoral nerve function, the patient recovered from the surgery with no signs of neurologic deficit and required no postoperative physical therapy.

DISCUSSION

The femoral nerve arises from rootlets of the L2, L3, and L4 nerve roots, with minor contributions from the L1 and L5 spinal nerves. It follows a retroperitoneal course through the psoas major muscle and travels posteriorly to the lateral border of the psoas major before passing under the inguinal ligament en route to the thigh and leg. Traveling laterally to the femoral artery, the femoral nerve passes through the Hunter canal and gives rise to the saphenous nerve. On its route, it innervates the psoas major, the iliacus, and the sartorius muscles; it provides motor innervation to the pectineus and quadriceps muscles and sensation to the anterior and medial thigh. The saphenous nerve provides sensory innervation to the

medial knee leg and foot. Injury of the femoral nerve, therefore, may result in weakness or paralysis of hip flexion and knee extension, atrophy of the quadriceps muscle, and numbness of the anterior thigh and medial aspects of the leg and foot.

Peripheral nerves can be compressed anywhere along their course. The femoral nerve is particularly vulnerable to penetrating, compressive, and iatrogenic injuries within the body of the psoas muscle, at the iliopsoas groove, and at the inguinal ligament. Iatrogenic injury to the femoral nerve is well documented: a 33-year review of 119 surgically treated femoral nerve injuries found 52 of 89 (58%) traumatic lesions to the femoral nerve to be iatrogenic in nature (1).

Gynecologic procedures were the most frequently implicated (2). When patients are in the lithotomy position, sharp flexion of the hip can compress the nerve at the inguinal ligament, and excessive external rotation and abduction of the hip can result in stretch on the nerve. Herniorrhaphies (3), hip replacement (4), appendectomies (5), and aortic aneurysm repair (6) have also been reported to result in femoral nerve injury. Along with direct transection or ligation, the use of self-retaining retractors has been cited as another cause of iatrogenic femoral nerve injury (7), either through direct or indirect compression of the nerve against the pelvic side-wall when the psoas is retracted laterally. The femoral nerve may also be injured because of traction or compression of the nerve within the psoas compartment caused by patient positioning during surgery or the presence of a compartment syndrome (8,9).

Femoral nerve injury resulting from spine surgery is a rare phenomenon. An iliopsoas hematoma causing compression of the femoral nerve within the psoas compartment has been reported as a complication after a successful posterior spinal decompression (10). Compression neuropathy of the femoral nerve within the psoas muscle during an anterior lumbar interbody fusion was suggested to result from prolonged hip extension during an anterior spinal procedure (9).

In the first case, postoperative imaging, as well as a direct surgical exploration of the femoral nerve, showed neither acute compression nor nerve injury. In addition, the patient's hips and knees were placed in a slightly flexed position during the supine anterior procedure and flexed 90 degrees while on an Andrew's frame during the posterior procedure. Thus, an axial stretching mechanism caused by joint hyperextension as reported by Papstefanou (9) is unlikely in this case. In hindsight, it was noted that, in the first case, the abdominal retractor system was placed in a position that could compress the femoral nerve along the medial pelvic wall, with the medial retractor blade displacing the great vessels medially and the lateral blades displacing the psoas muscle laterally. The force needed to maintain the retractor in an adequate position may have been greater than normal

because of the presence of scar tissue and soft tissue adherence of the surrounding spinal landmarks.

By identifying the probable cause for the femoral nerve palsy in the first case, future neuropathies were prevented. In the second case, the immediate removal of offending psoas retractors when MEPs showed a decrease in the femoral nerve and allowed normalization of nerve function. The third case again showed the need to understand the anatomic course of the femoral nerve, because removing extrinsic compression on the nerve during surgery also allowed normalization of function and prevented neurologic deficit. The use of MEP monitoring during surgery in the cases of the second and third patients alerted the surgeon that the femoral nerve was compromised by the retraction, allowing the surgeon to relieve the compression and prevent further damage. When the third patient presented with isolated quadriceps weakness during surgery, immediate repositioning of the patient and release of extrinsic compression on the nerve resolved the abnormal MEPs (11).

CONCLUSION

Because of an improvement in surgical technique and a better understanding of biomechanical and neural anatomy, the volume of spinal surgery is increasing. The 3 cases reported here illustrate the importance of monitoring the lumbar plexus during these complex surgical procedures. By understanding the etiology of the first patient's nerve palsy, we were able to prevent future femoral neuropathies, thus avoiding a significant post-operative morbidity.

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