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Case Report

Abdominal wall paresis after posterior spine surgery: An anatomic explanation

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1. Introduction

Abdominal wall paresis from denervation (pseudo-hernia) may be caused by damage to the lower thoracic intercostal, iliohypogastric (IH), or ilioinguinal (IG) nerves [1]. This complication has been reported in anterior and lateral spine surgery [2]. However, there has not been an association in posterior-only approaches to the spine for decompression and fusion. In this manuscript we report two instances of unilateral abdominal wall paresis after revision posterior thoracolumbar decompression and fusion. This is the first reported incidence of abdominal wall paresis after a posterior-only approach. Utilizing cadaveric anatomy of the L1-2 and L2-3 lumbar foramina and their contribution to nerves that supply the abdominal wall, we present an anatomic explanation for this complication and highlight the proximal anastomoses of motor rami contributing to the innervation of the anterior abdominal wall.

2. Case report

Patient #1 is a 73 year-old man with a history of previous L2-5 decompression and fusion who presented with neurogenic claudication and back pain. His imaging revealed adjacent segment disease above the level of his previous fusion and severe canal stenosis (see Fig. 1). The decision was made to proceed with an extension of his fusion to T9 and decompression from T11-L2. Intraoperatively, there was extensive epidural scar and severe foraminal stenosis at the L1-2 level on the left. The decompression at the L1-2 level was tedious and performed carefully in order to fully decompress the L1 nerve root exiting in the foramen. Intraoperative MEP and SSEP monitoring was utilized, and there were no changes throughout the procedure. An intraoperative CT scan was performed which showed satisfactory instrumentation placement. The patient awoke with no immediate complications. He had complete relief of his preoperative neurogenic claudication and significant improvement in his back pain. He noticed a slight protrusion of his abdomen immediately after surgery, which was not concerning at the time. At his follow up visit three weeks later, he still complained of abdominal bulging on his left side which had worsened (see Fig. 1). He underwent a general surgery consult and was diagnosed with left sided abdominal muscular paresis. He elected to proceed with conservative therapy with an abdominal binder. The patient had no improvement in the bulging at his latest follow up three months postoperatively.

Patient #2 is a 69-year-old female with a history of a previous L2-S1 decompression and fusion. She presented with neurogenic claudication and back pain due to adjacent segment disease above the level of her previous fusion. Her imaging revealed severe L1-2 lateral recess and foraminal stenosis on the left side due to facet hypertrophy and a calcified disc (see Fig. 1). The decision was made to proceed with extension of her fusion to T11 with decompression from T11-L2. Intraoperatively, her left L1 nerve root was severely compressed. Tedious dissection and separation of the nerve from the calcified disc was

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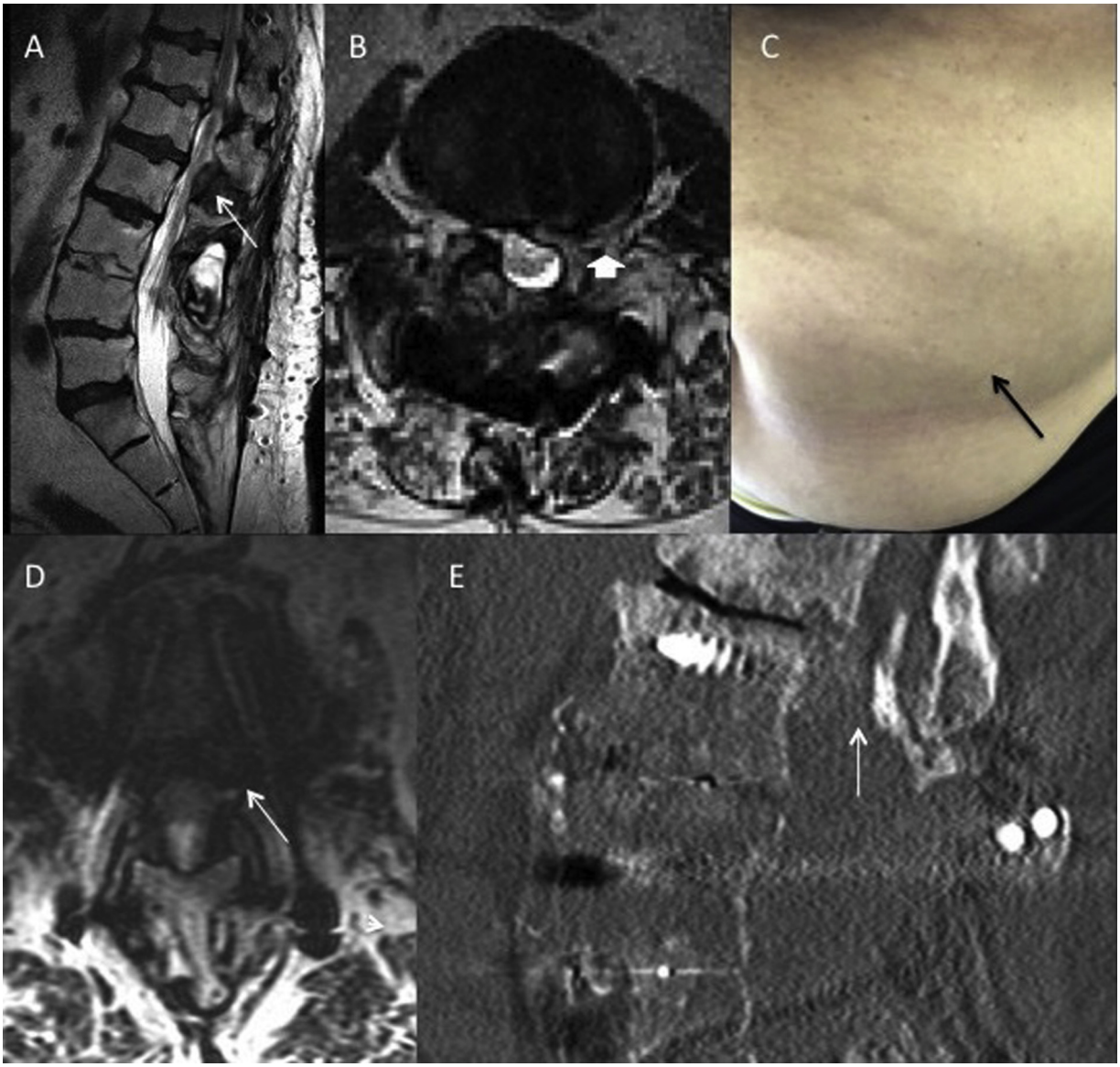

Fig. 1. Patient images. (A) Patient #1 imaging showing a sagittal T2 MRI with facet hypertrophy from adjacent segment disease and resulting central canal stenosis (white arrow). (B) Axial T2 MRI showing epidural scar tissue and calcification at the left L1-2 foramen (thick white arrow). (C) Left sided abdominal bulge from unilateral flaccid abdominal wall musculature (black arrow). (D) Patient #2 imaging showing an axial T2 MRI with adjacent segment disease at the L1-2 level with resulting foraminal stenosis and impingement of the exiting left L1 nerve root (white arrow). (E) Sagittal CT showing calcified disc causing lateral recess and foraminal stenosis at the L1- 2 level (white arrow).

required in order to achieve complete decompression. Intraoperative MEP and SSEP monitoring was utilized, and there were no changes throughout the procedure. An intraoperative CT scan was performed which showed satisfactory instrumentation placement. She had complete relief of her preoperative neurogenic claudication. At her postoperative visit three weeks later, during her physical exam it was noticed that she had a left sided abdominal bulge, consistent with muscular paresis from abdominal wall dennervation. Retrospectively, the patient did notice the bulge during her hospitalization. This was not bothersome to the patient. She had no improvement in the left sided bulging at her latest follow up 12 months postoperatively.

3. Discussion

Abdominal pseudo-hernia is a known consequence of lateral spine surgery and can significantly impact quality of life. This complication is also documented after cases of thoracotomy and large abdominal wall incisions in order to access the retroperitoneal space [3]. The most commonly implicated nerve in cases of thoracotomy is damage to the subcostal nerves that contains motor fibers from T12 and L1 [3]. The ilio-inguinal and ilio-hypogastric nerve also serve as a major contribution to innervation of the oblique muscles and transversus abdominis [1]. These nerves receive motor fibers from T12-L2 [4]. Disruption of any of these peripheral branches may lead to abdominal wall atrophy. There is existing literature describing unilateral abdominal wall paresis due to singular impingement of the L1 nerve root from an acutely herniated disc [5]. This finding in conjunction with our experience implies a significant motor contribution to the anterior abdominal wall from the L1 nerve root. There are connections between the L1 and L2 nerve roots that occur in close proximity to the foramen (see Fig. 2). This corroborates the previously mentioned T12-L2 contributions to the subcostal, IG, and IH nerves that have been shown in other cadaveric studies [1]. With these anatomic considerations, we formulated a hypothesis for the development of unilateral abdominal wall paresis. Both cases involved extensive scar and adjacent segment disease at the L1-2 level on the left side, and required some

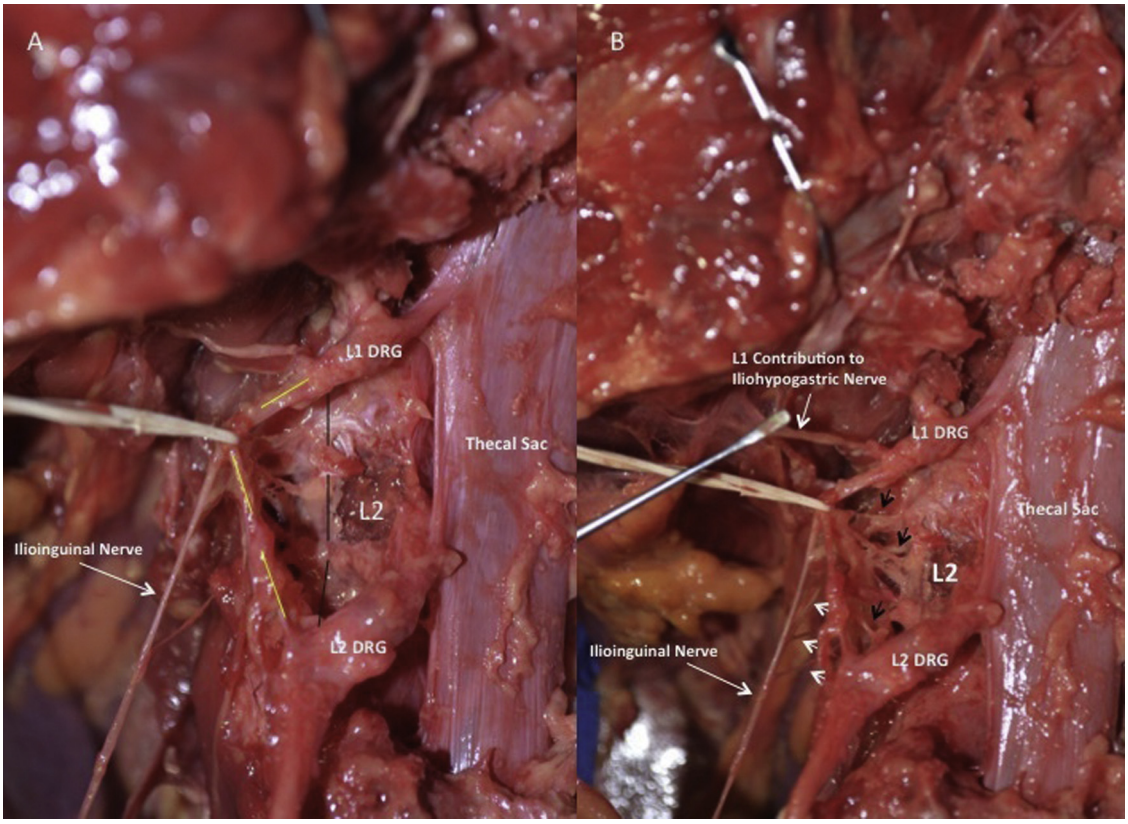

Fig. 2. Magnified posterior view of a cadaveric dissection at the L1-2 level with posterior elements removed. (A) The ilioinguinal nerve is shown arising from the L1 nerve root. The connecting branch between the L1 and L2 nerve roots was isolated (yellow dashes). Its natural course without retraction was adjacent to the lateral border of the L2 pedicle and traveled in the distal portions of the L1-2 and L2-3 foramen (black dashes). (B) The L1 contribution to the iliohypogastric nerve is shown, joining with the T12 nerve root. Anastomotic connections from the ilioinguinal nerve (arising from L1) to the L2 nerve are shown (white arrowheads). The recurrent sinuvertebral nerves arising from the L1 and L2 roots innervating the annulus of the L1-2 disc are shown (black arrows) (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article).

manipulation of the left L1 nerve root during decompression of the lateral recess and foramen. It is possible that during dissection of the L1 nerve root, there was injury to the branching motor rami in the distal portion of the foramen that contribute to the motor innervation of the abdominal wall through the subcostal, IG, and IH nerves. This may have been undetected due to the small size of these branches. Neuropraxic injury of the L1 nerve root from traction during foraminal dissection likely also contributed to some degree, especially if this root was the sole motor contributor to the IH or IG nerve in both cases. There was no psoas or soft tissue retraction below the level of the transverse processes during either surgery, disputing the possibility of direct lumbar plexus injury.

4. Conclusions

This report has significant implications for complication avoidance in posterior spine surgery, and represents the first reported series of patients with abdominal wall atrophy after a posterior only approach for thoracolumbar decompression and stabilization. The anastomotic connections between the T12, L1, and L2 nerve roots that arise in the distal foramen at the proximal end of the post ganglionic segment may be injured through traction or direct mechanical damage during a posterior foraminal approach, leading to abdominal wall paresis. This is an important consideration for preoperative patient counseling and intraoperative safety factors during posterior spine surgery.

Declaration of Competing Interest

The authors have no conflicts of interest to disclose.

All pertinent patient information and images included have been de-identified and anonymized.

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