Abdominal wall paresis as a complication of minimally invasive lateral transpsoas interbody fusion

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Object. The minimally invasive lateral transpsoas approach for interbody fusion has been increasingly employed to treat various spinal pathological entities. Gaining access to the retroperitoneal space and traversing the abdominal wall poses a risk of injury to the major nervous structures. Nerve injury of the abdominal wall can potentially lead to paresis of the abdominal musculature and bulging of the abdominal wall. Abdominal wall nerve injury resulting from the minimally invasive lateral retroperitoneal transpsoas approach has not been previously reported. The authors describe a case series of patients presenting with paresis and bulging of the abdominal wall after undergoing a minimally invasive lateral retroperitoneal approach.

Methods. The authors retrospectively reviewed all patients who underwent a minimally invasive lateral transpsoas approach for interbody fusion and in whom development of abdominal paresis developed; the patients were treated at 4 institutions between 2006 and 2010. All data were recorded including demographics, diagnosis, operative procedure, positioning, hospital course, follow-up, and complications. The onset, as well as resolution of the abdominal paresis, was reviewed.

Results. The authors identified 10 consecutive patients in whom abdominal paresis developed after minimally invasive lateral transpsoas spine surgery out of a total of 568 patients. Twenty-nine interbody levels were fused (range 1–4 levels/patient). There were 4 men and 6 women whose mean age was 54.1 years (range 37–66 years). All patients presented with abdominal paresis 2–6 weeks postoperatively. In 8 of the 10 patients, abdominal wall paresis had resolved by the 6-month follow-up visit. Two patients only had 1 and 4 months of follow-up. No long-term sequelae were identified.

Conclusions. Abdominal wall paresis is a rare but known potential complication of abdominal surgery. The authors report the first case series associated with the minimally invasive lateral transpsoas approach.

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KEY WORDS • eXtreme Lateral Interbody Fusion • abdominal wall paresis • direct lateral interbody fusion • pseudohermia • complication

Minimally invasive spinal procedures such as the lateral retroperitoneal transpsoas approach are being increasingly used to treat various spinal disorders including degenerative disc disease, spondyloolisthesis, spinal deformities, and traumatic spinal injuries. In addition to the inherent risks to the lumbar plexus due to the proximity of the surgical pathway, there is also risk of injuring nerves that travel outside the psoas muscle. Gaining access to the retroperitoneal space and traversing the abdominal wall poses a risk of injury to the major nervous structures and adds significant morbidity to the procedure.

Nerve injury of the abdominal wall results in paresis and bulging of the abdominal musculature. Abdominal wall paresis following laparoscopic surgeries, although rare, has been described.11,28 To our knowledge, abdominal wall paresis following minimally invasive spinal surgery has not yet been reported in the literature. We present a case series of abdominal wall paresis following the minimally invasive lateral retroperitoneal transpsoas approach and review the relevant anatomy.

Methods

The authors retrospectively reviewed data obtained in all consecutive patients who underwent a minimally invasive lateral retroperitoneal transpsoas approach for interbody fusion and who subsequently developed abdominal paresis. The data were culled records at 4 institutions between 2006 and 2010. All patient data were recorded prospectively including demographics, diagnosis, operative procedure, positioning, hospital course, follow-up, and complications. Each institution was responsible for the collection and maintenance of their databases. The
surgeons were responsible for the reliability and validity of their own database. The onset and resolution of the abdominal paresis were noted.

**Results**

Between 2006 and 2010 at the 4 centers, we identified 10 consecutive patients in whom abdominal paresis developed after minimally invasive lateral transpsoas spine surgery of a total of 568 patients—an incidence rate of approximately 1.8% (Table 1). These cases were identified on routine physical examinations during follow-up visits. Each patient was examined by his/her surgeon. A total of 29 interbody levels were fused (range of 1–4 levels/patient). There were 4 men and 6 women whose mean age was 54.1 years (range 37–66 years). Primary diagnoses included degenerative disc disease, spondylosis, pseudarthrosis, and adult degenerative and idiopathic scoliosis. All patients presented with complaints of low-back pain, whereas only 4 had a component of radiculopathy. A minimally invasive lateral retroperitoneal approach for interbody fusion was successfully performed in all patients.

All patients later presented with fullness and bulging of the anterior abdominal wall, away from their incisions, and were diagnosed with abdominal paresis 2–6 weeks postoperatively (Table 1). Four patients underwent abdominal CT scanning to confirm that there was no evidence of an abdominal wall defect or hernia. All patients were treated conservatively, with no acute intervention. Eight of the 10 patients had complete resolution of the abdominal wall paresis by the 6-month follow-up visit. Two patients had persistent abdominal wall paresis at last follow-up, which in one was only 1 month and in the other 4 months after presentation. The mean follow-up period was 18.3 months (range 1–40 months). No long-term sequelae were identified.

**Illustrative Case**

This 66-year-old woman underwent a left-sided minimally invasive lateral retroperitoneal approach to L1–2 and L2–3 for interbody fusion. The patient’s hospital course was uneventful and she was discharged to home. Two weeks postoperatively, she had a noticeable fullness and bulging of the anterior abdominal wall on the side of the surgical approach, but away from the incisions (Fig. 1). There was no associated pain or discomfort. This bulge became more pronounced at 4 weeks, and abdominal wall denervation was diagnosed. A CT scan of the abdomen was acquired to confirm that there was no abdominal wall defect or hernia. The patient was treated conservatively with a corset. By 6 months postoperatively, the abdominal wall paresis had completely resolved and the abdomen had returned to a normal appearance. There were no long-term sequelae due to the abdominal wall dysfunction.

**Discussion**

The minimally invasive lateral retroperitoneal transpsoas approach is increasingly being used to treat various spinal disorders. Although this procedure is safe and reproducible, approach-related neural complications have been reported.4,10,27,29 The majority of neural complications, secondary to lumbar plexus injuries, occur while traversing the psoas muscle. However, there is inherent risk of direct injury to the main motor nerves that supply the anterior abdominal muscles during the early stages of the minimally invasive lateral retroperitoneal approach while gaining access to the retroperitoneum, traversing the abdominal wall, or bluntly dissecting the retroperitoneum.3 Injury to these nerves may result in denervation, paresis, and bulging of the anterior abdominal wall. This paresis is typically characterized by swelling and is often associated with pain, hyperesthesia, or other sensory abnormalities. It is sometimes referred to as a “pseudohermia.”6,7,16,21

One of the main causes of abdominal wall paresis is iatrogenic injury during surgery. It has been reported after abdominal and pelvic surgery.5,8,11,19,28 Nerve root compression from a herniated disc has also been reported in the literature.1,18,22 Diabetic neuropathy has also been implicated as an etiological factor of abdominal wall paresis.3,12,13 One of the main causes of diabetic neuropathy is herpetic zoster and

### TABLE 1: Summary of patient, disease, and treatment characteristics*

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Age (yrs), Sex</th>
<th>Diagnosis/Pathology</th>
<th>Presenting Sign/ Symptom</th>
<th>Lat Interbody Fusion Levels</th>
<th>No. of Levels</th>
<th>FU (mos)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>37, F</td>
<td>adult idiopathic scoliosis</td>
<td>LBP</td>
<td>L1–5</td>
<td>4</td>
<td>36</td>
</tr>
<tr>
<td>2</td>
<td>56, F</td>
<td>adult idiopathic scoliosis</td>
<td>LBP with radiculopathy</td>
<td>L2–5</td>
<td>3</td>
<td>40</td>
</tr>
<tr>
<td>3</td>
<td>54, F</td>
<td>adult idiopathic scoliosis</td>
<td>LBP with radiculopathy</td>
<td>L1–4</td>
<td>3</td>
<td>33</td>
</tr>
<tr>
<td>4</td>
<td>66, F</td>
<td>ASF/ DDD/degenerative scoliosis</td>
<td>LBP</td>
<td>L1–3</td>
<td>2</td>
<td>24</td>
</tr>
<tr>
<td>5</td>
<td>65, M</td>
<td>postaminectomy syndrome, kyphotic deformity, DDD</td>
<td>LBP with radiculopathy</td>
<td>L1–5</td>
<td>4</td>
<td>4</td>
</tr>
<tr>
<td>6</td>
<td>54, M</td>
<td>pseudarthrosis</td>
<td>LBP</td>
<td>L2–3</td>
<td>1</td>
<td>9</td>
</tr>
<tr>
<td>7</td>
<td>53, F</td>
<td>adult degenerative scoliosis</td>
<td>LBP with radiculopathy</td>
<td>L2–5</td>
<td>3</td>
<td>6</td>
</tr>
<tr>
<td>8</td>
<td>57, F</td>
<td>DDD</td>
<td>LBP</td>
<td>L3–5</td>
<td>2</td>
<td>12</td>
</tr>
<tr>
<td>9</td>
<td>46, M</td>
<td>lumbar spondylosis</td>
<td>LBP</td>
<td>L2–5</td>
<td>3</td>
<td>18</td>
</tr>
<tr>
<td>10</td>
<td>53, M</td>
<td>adult degenerative scoliosis</td>
<td>LBP</td>
<td>L1–5</td>
<td>4</td>
<td>1</td>
</tr>
</tbody>
</table>

*ASF = adjacent-segment failure; DDD = degenerative disc disease; FU = follow-up; LBP = low-back pain.
Abdominal wall paresis due to interbody fusion

Lyme disease have been documented to cause neuropathy causing abdominal wall paresis.9,15,16,20,24–26,33

Although there are many causes of abdominal paresis, treatment remains conservative. Spontaneous recovery of nerve function has been reported in several cases.18–20 No patient in our case series required any intervention or treatment. The majority of patients improve. Abdominal CT scanning may be performed to exclude the presence of abdominal hernias. Conservative treatment may consist of increasing abdominal support with a fitted corset. In our series, all 6 patients with greater than 6 months of follow-up had full recovery and resolution of their abdominal paresis.

Although abdominal paresis following the minimally invasive lateral retroperitoneal surgery has not been reported, it is a well-known complication of conventional abdominal and gynecological surgery.2,14,17,23,31 In our series, the overall incidence of this complication was 1.8%. Abdominal paresis has been reported to be the second most common neuropathy following gynecological surgery with a reported incidence of 3.7%.31 Although this approach-related complication is rare, with knowledge of the anatomy and a meticulous technique, it can be entirely avoided.

Regional Abdominal Anatomy

To prevent such complications, knowledge of the regional anatomy is critical. A brief review of the pertinent anatomy is as follows. The abdominal wall is mainly composed of 4 groups of muscles: the rectus abdominis, internal oblique, external oblique, and transverse abdominis. The pertinent nerves that supply these muscles are the subcostal, iliohypogastric, and ilioinguinal. Their anatomical description, trajectory, and clinical significance have been well described.5,32 The subcostal nerve originates from the T-12 nerve root and innervates the rectus abdominis and external oblique muscles. The iliohypogastric and ilioinguinal originate from the T-12 and T12–L1 nerve roots, respectively. They innervate the internal oblique and transverse abdominis muscles. All 3 nerves travel freely in the retroperitoneum and course anteriorly and inferiorly as they pierce the abdominal wall muscles.5,32 When the motor portions of these nerves are injured, it leads to paralysis of the muscles that are supplied by them and subsequent weakening of the abdominal wall.

There are several limitations to this study. As a retrospective review, there is a risk of a selection bias, but the databases were collected prospectively, and all consecutive patients were included in the study. Also, we reviewed a large cohort that included 568 patients from 4 treatment centers.

Complication Avoidance

The location and trajectories of these nerves place them at significant risk during the early stages of the minimally invasive lateral retroperitoneal transpsoas approach. The nerves can be directly injured while accessing the retroperitoneum during the flank or lateral incision, while dissecting the abdominal muscle wall, or while performing blunt digital dissection of the retroperitoneal cavity. We advocate a combination of useful adjuncts to minimize nerve injuries directly or indirectly during the approach. During the early stages of the approach, once the external oblique muscle fascia is sharply dissected, the dissection of the abdominal wall muscles requires special care. We suggest sequential and gentle muscle dilation with blunt instruments (hemostat forceps) until the retroperitoneal cavity is identified. If a nerve branch is found during the dissection, it is possible to dissect it and mobilize it without causing direct injury. Once the retroperitoneal space is accessed, the blunt dissection of the retroperitoneal space requires gentle development of the space from posterior to anterior and superior to inferior to avoid injuring the main nerves that run freely in the retroperitoneal cavity. While dissecting the retroperitoneum bluntly with the finger, one must be careful not to confuse a free-running nerve in the retroperitoneal fat for an adhesion and avulse or injure it.

Conclusions

Abdominal wall paresis is a rare complication of minimally invasive lateral spinal surgery. We report, to the best of our knowledge, the first case series in the literature. Knowledge of the anatomy of the abdominal wall muscles and the traversing motor nerves that supply them.
is very crucial to avoid damaging these nerves. Injury to these nerves may result in paresis and bulging of the anterior abdominal wall.

Disclosure

Dr. A. X. Le is an owner of Phygen Spine and consults for Nuvasive. Dr. Smith consults for Nuvasive. Dr. Akbarnia consults for Nuvasive, K2M, and DePuy Spine; is an owner of Phygen Spine; and receives clinical or research support from K2M, Nuvasive, and DePuy Spine. Dr. Uribe consults for Nuvasive.

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