## Editorial

### Neural Deficits and Lateral Lumbar Interbody Fusion

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**EDITORIAL**

In lateral lumbar interbody fusion (LLIF), as opposed to a traditional anterior or posterior surgical approach, the lumbar spinal motion segment is accessed via a direct lateral, retroperitoneal, transpsoas approach. The broad spectrum of surgical indications for LLIF includes spondylolisthesis, sciotic deformities, central or foraminal stenosis, and adjacent segment disease.

In fact, besides the avoidance of morbidities associated with traditional anterior or posterior interbody fusion techniques, including injury to visceral, vascular or neural structures, minimally invasive LLIF enables the surgeon to spare the anterior and posterior longitudinal ligaments during the surgical approach, and to implant cages with a large footprint, designed to span the dense apophyseal ring bilaterally [1-5].

However, despite the utilization of intraoperative real-time directional electromyography (EMG) and/or intraoperative neuromonitoring systems, the transpsoas approach to lumbar motion segments has been associated with an increased risk for post-operative neurological deficits, with a reported prevalence of nerve injuries ranging from 0.7% to 23% in current literature [6-12]. When we evaluated neurologic deficits in 451 patients (919 levels) who had undergone LLIF, the prevalence of motor or sensory deficit was 2.3% and 9.6%, respectively, as recorded after a minimum follow-up of 18 months [13]. At the same time point, persistent anterior thigh/groin pain was present in 5.8% of the patients.

The total number of levels operated per patient has been identified as the strongest predictor of neural complications during LLIF, and the inclusion of the L4-L5 level as an independent risk factor for lumbosacral plexus injury following LLIF [8]. On the other hand, some authors support that involvement of L4-L5 does not increase the risk of lumbosacral plexus motor deficit [6]. Operative time has also been proposed as a risk factor for lumbar plexus injury secondary to the neural ischemia caused by the prolonged retraction against the transverse process, particularly at L4-L5 level [6]. However, motor and/or sensory nerve deficits and postoperative pain still occur when levels other than L4-L5 are treated even by the most experienced spine surgeons.

According to our findings, although inclusion of the L4-L5 level was associated with a higher number of motor and sensory deficits, it was not found to be a significant risk factor for developing a neurologic deficit after LLIF [13]. Likewise, no correlation was recorded between operative time and lumbopelvic injury or anterior thigh/groin pain. Multivariate regression analysis revealed a trend of increased persistent neurologic deficits as levels treated were moving from cranial to caudal and increased anterior thigh/groin pain when levels L1-L2 or L4-L5 were fused.

In the same study a correlation between recombinant human bone morphogenetic protein-2 (rhBMP-2) and postoperative neural deficits and pain following LLIF was speculated [13]. A well-matched study that conducted by the same authors, demonstrated a strong association between rhBMP-2 exposure of lumbar plexus and neurologic complications after LLIF [14]. According to this study, the use of rhBMP-2 resulted in a higher number of patients with neural deficits during the immediate postoperative period as well as a higher number of persistent neurologic deficits at the last follow-up. These findings suggested an adverse effect of rhBMP-2 on nerve physiology and an inhibitory action on nerve recovery, respectively.

Blind percutaneous dilation through the psoas muscle carries a high risk for nerve injury. Dissection under direct visualization of the psoas with identification and protection of the lumbar plexus nerve roots is strongly recommended. rhBMP-2 adverse effect on neural tissue may be modifiable with careful drug handling and/or modification of the surgical technique.

**REFERENCES**


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