CASE REPORT

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Lumbar plexopathy following transforaminal interbody fusion: a rare complication

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Postoperative radiculopathy has previously been reported as a common complication of transforaminal lumbar interbody fusion (TLIF). However, no data has been published on lumbar plexopathy following TLIF. We present a rare case of lumbar plexopathy occurring following TLIF (L5-S1) in a patient with spondylolisthesis. Although initially a diagnostic challenge, sinister causes of neuropathy such as bleeding or neoplastic growths were excluded by imaging. A diagnosis of acute lumbar plexopathy of left L2 to L4 was eventually made after electromyographic studies was performed in consultation with a senior neurologist. The patient was treated supportively with good functional recovery.

Key words: Fusion; interbody; lumbar; plexopathy; spine; transforaminal.

The lumbar plexus originates from the anterior primary rami of L1 to L4. The principal branches of the plexus are the femoral and obturator nerves. Functionally, the lumbar plexus provides muscular innervation to the abdomen, inguinal region and thighs as well as cutaneous innervation to the abdomen, groin, buttocks, and thighs. The plexus also provides articular innervations to the hip and knee joints.[1]

Any disorder of the lumbar plexus gives rise to a plexopathy. Diverse causes of lumbar plexopathy including trauma,[2] hemorrhage,[3-5] surgical procedures,[6] neoplastic growths,[7] radiation,[8] compressive effect,[9] and diabetes[10] have been reported in the medical literature.

Postoperative radiculopathy has previously been reported as a common complication of transforaminal lumbar interbody fusion (TLIF).[11,12] However, no data on lumbar plexopathy post-TLIF has been reported to date.

We present a rare case of lumbar plexopathy occurring following a TLIF (L5-S1) in a patient with spondylolisthesis.

Case report

A 14-year-old Indian male with a past history of well-controlled asthma presented in August 2006 with severe lower back pain. Symptoms began in May 2005 and were intermittent in nature and associated with right sciatica. The patient also complained of non-specific numbness in the right leg.

Examination of his back showed a good range of movement with no focal tenderness. Neurological examination of both lower limbs was normal, with a preoperative ASIA score of E.

Radiographs and computed tomography (CT) of the lumbar spine showed a Grade 1 spondylolisthesis of the

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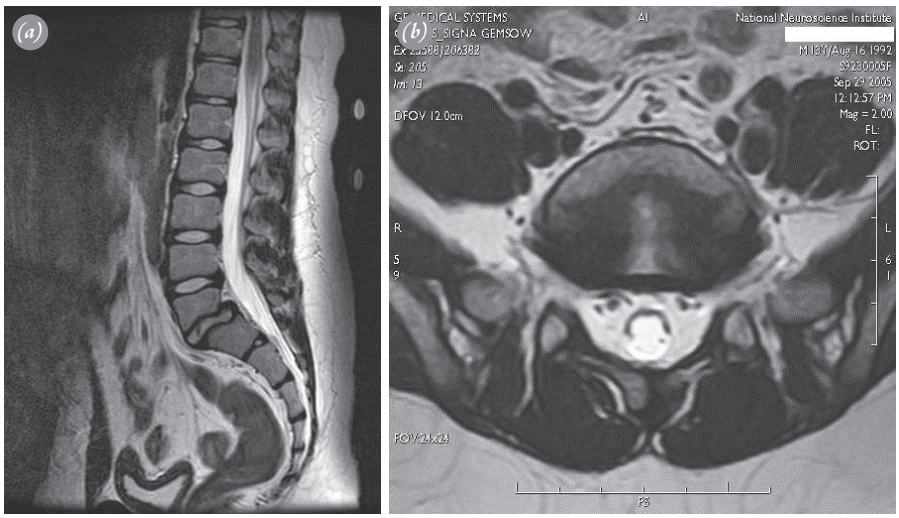

Fig. 1. **(a)** Sagittal and **(b)** axial MR images of the lumbar spine showing no significant spinal canal stenosis.

L5-S1 due to bilateral spondylolysis. Magnetic resonance imaging (MRI) showed L5-S1 Grade 1 spondylolisthesis with mild narrowing of the bilateral L5-S1 exit foramina and no significant spinal canal stenosis (Figs. 1 and 2).

Decompression laminectomy of the L5-S1, TLIF with cage and bone graft and posterior spinal stabilization was performed with the patient under general anesthesia one month after first presentation.

Intraoperative findings were of a Grade 1 isthmic spondylolisthesis of the L5-S1, bilateral pars defect of the L5, and L5 root compression on both sides.

After adequate exposure, pedicle screws were inserted into the L5 and S1 vertebrae using a C-arm image intensifier. Laminectomy of the L5 and S1 were performed and both pars defects were debrided. Both L5 nerve roots were identified and decompressed from the shoulders to the exit foramina. Discectomy was then performed on the left, followed by curettage of the disc space. A 10 mm cage was inserted along with laminar bone autograft and additional allograft. A check C-arm image confirmed good placement of the implants. The wound was then irrigated and closed.

Intraoperative electromyographic monitoring was done throughout the surgery covering L2 to S4 bilaterally.

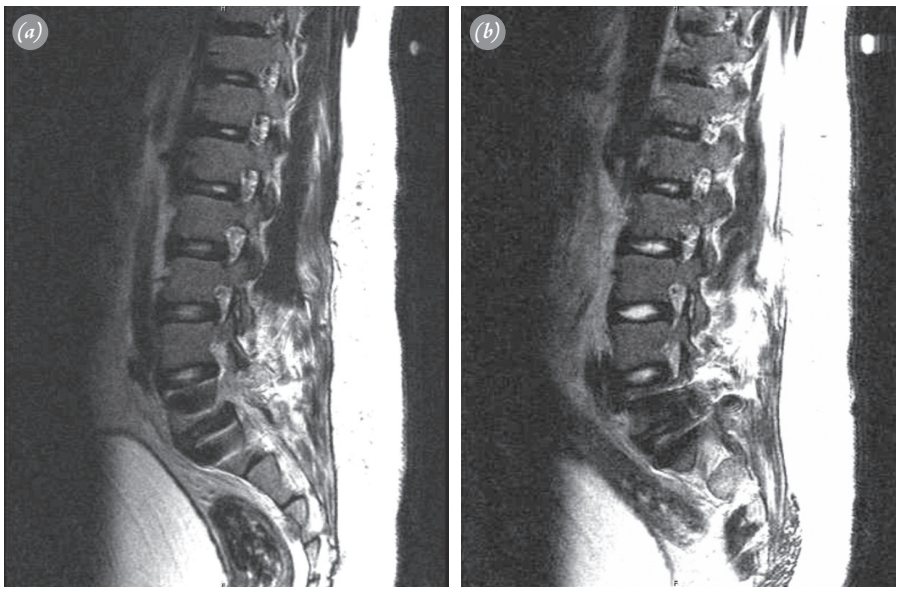

Fig. 2. **(a, b)** Sagittal MR images of the lumbar spine showing mild bilateral narrowing of the L5-S1 exit foramina.

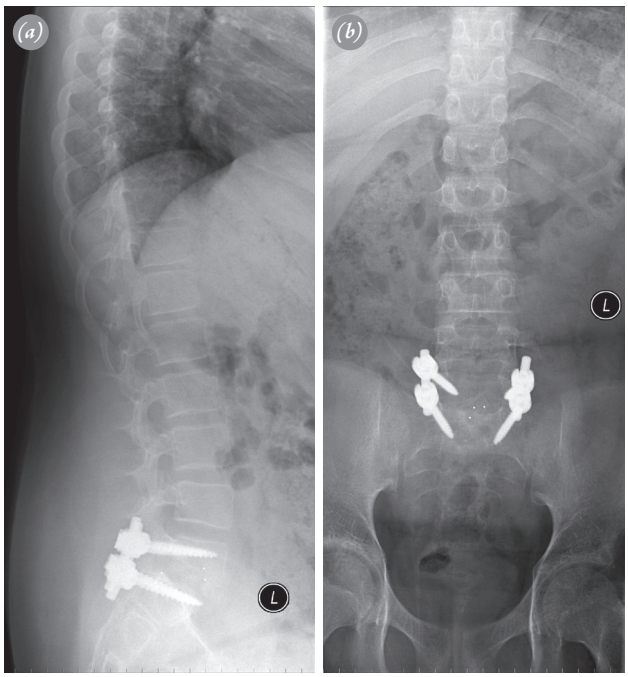

Fig. 3. Immediate postoperative radiographs in **(a)** lateral and **(b)** anteroposterior views.

No abnormal electromyographic discharge was detected.

Immediate postoperative radiographs are given in Figure 3.

After surgery, the right lower limb symptoms did not return. However, on the 1st postoperative day, the patient complained of numbness and severe pain (visual analog scale score of 8) over the left leg. He described his pain to be of a throbbing nature, located mainly over the knee, calf and ankle. On examination, sensation was decreased over the left L2 to L4 dermatomes. Power from the L2 to L4 was reduced on the left due to pain. Reflexes were symmetrical but brisk bilaterally. Spinal drain was charted at 90 ml of hemoserous fluid.

The clinical impression was radicular pain and the patient was treated symptomatically with oral analgesia (paracetamol and tramadol) in addition to the intravenous morphine as patient-controlled analgesia (PCA). Oral prednisolone (for 5 days duration) as an empirical anti-inflammatory agent and gabapentin was also commenced. A radiograph of the lumbar spine and left knee did not reveal any abnormality.

Due to the persistent nature of his symptoms, a CT scan of the lumbar spine was performed on the 3rd postoperative day. The CT did not reveal any abnormality and it was observed that the pedicular screws were well placed and there was no paraspinal hematoma.

An MRI of the lumbar spine performed on the 5th postoperative day did not reveal any paraspinal or epidural collections. At this stage, the patient's pain had improved significantly (VAS score: 2) and the PCA morphine was stopped by the pain team.

Ambulatory physiotherapy was initiated although progress was slow due to pain, hypersensitivity and weakness.

A neurology consult was made on the 12th postoperative day in view of the persistent neurological symptoms. Although now much more bearable, the patient continued to complain of pain and left lower limb weakness (hip flexion power: 2, hip extension: 5, knee flexion: 1, knee extension: 3, ankle flexion and extension: 5). Nerve conduction studies performed on the same day revealed absent left saphenous sensory nerve action potentials (Table 1). Electromyography (EMG) showed action denervation changes in the left vastus medialis, vastus lateralis and adductor longus muscles, corresponding to L2 to L4 myotomes (Table 2). Impression was postoperative left lumbar plexopathy affecting the L2 to L4 levels. As no traumatic, mechanical or infective cause was apparent, autoimmune neuritis (similar to postoperative neuralgic amyotrophy) was considered as a diagnosis of exclusion. The recommendation was to continue symptomatic treatment, inpatient physiotherapy and MRI of the lumbosacral plexus to exclude any organic lesions.

An MRI of the lumbosacral plexus was performed on the advice of the neurologist on the 14th postoperative day. The result was normal with the lumbosacral plexus exhibiting a normal appearance.

The patient was continued on inpatient physiotherapy and showed steady progress. Although hip flexion (3) and knee extension (3) was still weak, he was able to ambulate independently with crutches. The patient was discharged on the 25th postoperative day.

The patient underwent outpatient physiotherapy and follow-up on discharge. He showed slow but steady recovery of the proximal power of his left lower limb. He regained full power of his left lower limb 1 year after surgery. The numbness over L4 dermatome resolved completely. The numbness over his left thigh (L2) and knee (L3), though still present, also improved significantly. He was able to participate in all school activities and the gradual but progressive recovery continued. Follow-up was performed yearly and neurology had completely resolved by the 5th year follow-up.

Discussion

Etiology of neuralgia following lumbar spinal fusion are varied, including misplaced pedicle screw, conjoint nerve

Table 1. Nerve conduction study on the 12th postoperative day.

Findings				
1. Motor conduction study				
Nerve: Stimulation/Recording sites	**Latency (msec)**	**Amplitude (mV)**	**Conduction velocity (m/sec)**	**F wave latency (msec) & Frequency**
R tibial:				
ankle/abductor hallucis	3.1	8.7	popliteal fossa	
popliteal fossa/abductor hallucis	12.6	6.6	ankle: 46.3	50.8
L tibial:				
ankle/abductor hallucis	3.7	10.5	popliteal fossa	
popliteal fossa/abductor hallucis	15.5	6.9	ankle: 38.1	53.5
R peroneal:				
ankle/extensor digitorum brevis	4.0	8.0	popliteal fossa	
popliteal fossa/extensor digitorum brevis	13.7	6.5	ankle: 45.1	51.6
L peroneal:				
ankle/extensor digitorum brevis	4.8	4.5	popliteal fossa	
popliteal fossa/extensor digitorum brevis	14.6	4.1	ankle: 43.9	56.3
2. Sensory conduction study				
Nerve: Stimulation/Recording sites	**Latency (msec)**	**Amplitude (uV)**	**Conduction velocity (m/sec)**	
R superficial peroneal: leg / ankle	2.4	14	50.0	
L superficial peroneal: leg / ankle	2.6	3.3	46.2	
R sural: leg/ankle	2.5	14	48.0	
L sural: leg/ankle	2.6	12	46.2	
R saphenous	2.3	4.9	52.2	
L saphenous	Absent	–	–	
3. H Reflex				
Nerve: Stimulation/Recording sites	**Latency (msec)**	**Amplitude**		
R tibial: popliteal fossa/soleus	29.7			
L tibial: popliteal fossa/soleus	Absent			

root, bony fragment adjacent to a nerve root, nerve root swelling/relative stenosis, and graft subsidence.[13] However, etiology is usually at the level of the nerve root, i.e. radiculopathy. While plexopathy after spinal surgery is rare, it should be suspected and investigated if the symptoms do not correlate to the level of the surgery. In this case, the level of pathology and surgery occurred at L5-S1 while the neurology the patient complained of was located at a higher level (L2 to L4). Further imaging with MRI should be performed to exclude organic causes of plexopathy such as hemorrhage, neoplastic growths and bony fragments. If such causes are excluded, a diagnosis of neuralgic amyotrophy can be made, allowing for proper treatment. In the current case, the patient eventually made a full recovery of his power and sensation.

While postoperative neuralgic amyotrophy[14] often affects the brachial plexus, lumbar plexus involvement may occur. Neuralgic amyotrophy, also known as Parsonage-Turner Syndrome, is a diagnosis of exclusion. This is a clinical syndrome associated with various etiologies, including virus infections, vaccinations and trauma, and presents with rapid onset of pain, weakness and wasting of the shoulder girdle muscles and, less commonly, the pelvic girdle muscles.[15] The pattern of weakness can be variable, patchy and does not conform strictly to a dermatomal or myotomal distribution. Most cases have an autoimmune pathogenesis, resulting in focal demyelination and axonal degeneration. Electrophysiological studies including nerve conduction and EMG are helpful in its evaluation. In many cases, direct imaging of the plexus does not show abnormalities, therefore suggesting a non-structural cause of plexopathy.

Steroids have not yet proven to be useful in its treatment, which is generally supportive in nature (physio-

Table 2. Electromyography findings on the 12th postoperative day.

Side	Muscle	Nerve	Roots	Insertional activity	Spontaneous activity			Motor unit potentials (MUP)				
					Fib	Fasc	PSW	Amplitude (mV)	Duration	Recruitment	Polyphasic potentials (%)	Interference pattern
Left	Vastus medialis	Femoral	L2, L3, L4				2+			No voluntary units		
Left	Vastus lateralis	Femoral	L2, L3, L4				2+			No voluntary units		
Left	Adductor longus	Obturator	L2, L3, L4									
Left	Tibialis anterior	Peroneal, Deep	L4, L5				Occasional	1-2	Normal	Slightly↓	Occasional	Slightly↓
Left	Gastrocnemius, Medial head	Tibial	L5, S1, S2		Nil	Nil		1-2	Normal	Slightly↓	Nil	Normal

Fib: Fibrillation; Fasc: Fasciculation; PSW: Positive sharp wave. Repeat study. This study gives evidence of axonal left lumbar plexopathy. No voluntary units were elicited in the L3, L4 innervated muscles.

therapy, occupational therapy and pain control). Most cases are expected to recover completely although it may run a protracted course.

For this patient, the rapid onset, lack of correlation between the surgical level of intervention and clinical findings and electrophysiological studies supported the impression of neuralgic amyotrophy in the lumbar plexus. There is no established method to prevent its onset.

In conclusion, we present a rare occurrence of lumbar plexopathy occurring following transforaminal lumbar interbody fusion surgery and its good long-term prognosis.

Conflicts of Interest: No conflicts declared.

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