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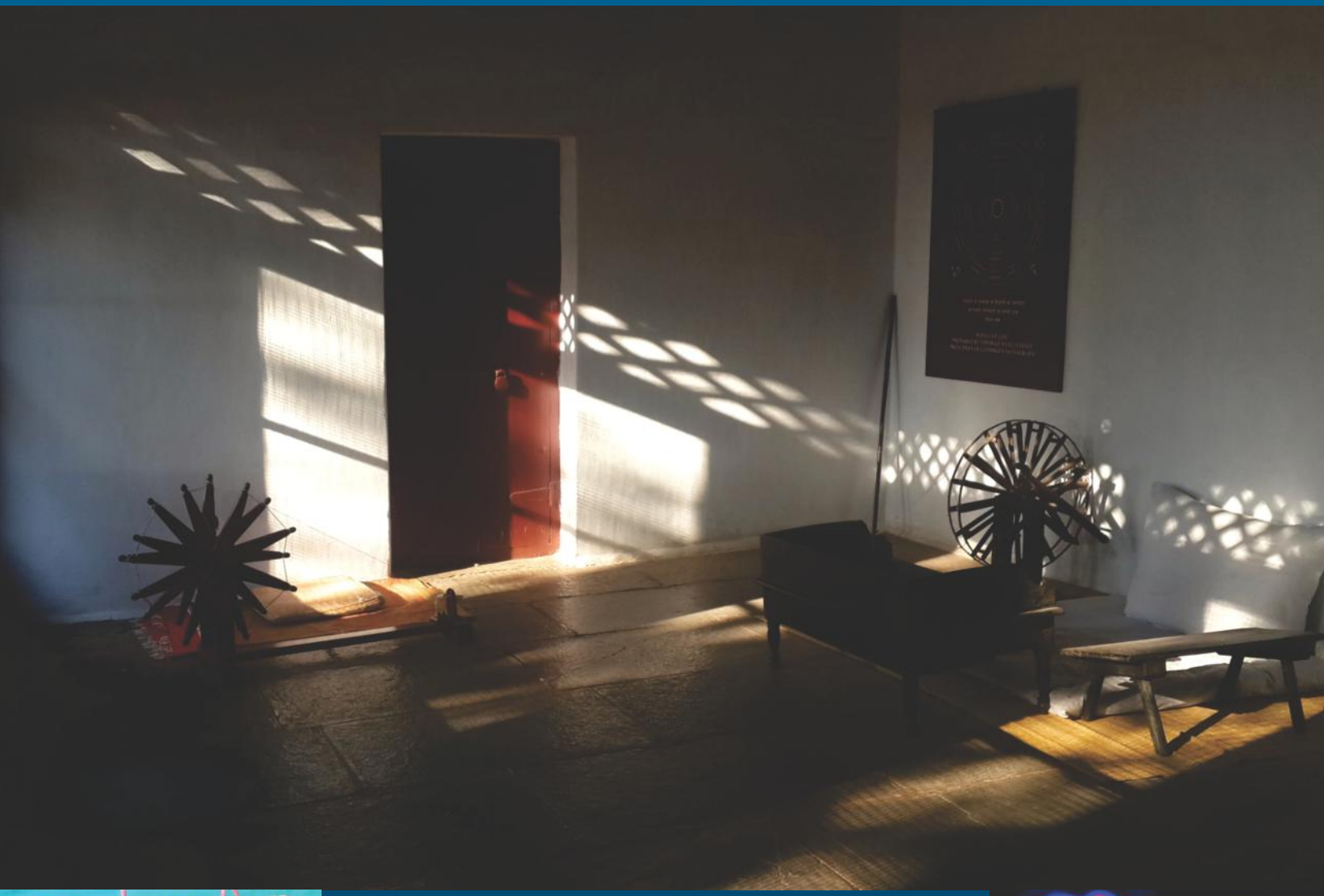

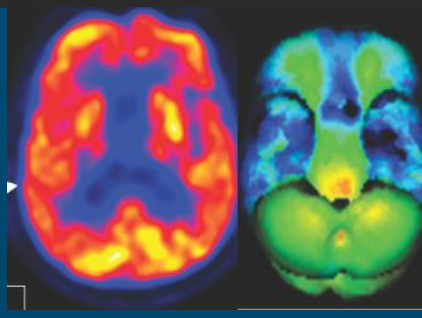

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Permanent spinal cord injury during lumbar spinal anesthesia: A report of two cases

Sir,

Spinal anesthesia is a commonly performed regional anesthesia with several advantages and few complications. The common complications encountered are hypotension, postural headache, and hypothermia. Rarely, serious complications are encountered such as cardiac arrest, seizures, paraplegia or cauda equina syndrome and radicular deficits. Transient neurological deficits are more common than permanent neurological complications. Myelomalacia due to lumbar spinal anesthesia is extremely rare with a few cases reported in literature. We report two cases of spinal cord injury demonstrated by magnetic resonance imaging (MRI) following lumbar spinal anesthesia. In both cases, motor power and dorsal tract sensations improved during the follow-up period, whereas spinothalamic tract sensations failed to improve.

Our first case refers to a 40-year-old female patient who sustained an ankle fracture following a fall; subsequently, she underwent an open reduction and internal fixation under spinal anesthesia. She experienced acute shock-like sensation of the entire right lower limb during spinal anesthesia. On recovering from the anesthesia, she noticed total loss of sensation for all modalities in the entire right lower limb up to the hip region with severe weakness. She received several courses of steroid therapy but without gaining any benefit. With prolonged neurorehabilitation, her weakness and numbness improved slowly. She became ambulant after 4 months. However, loss of ability to appreciate pain or temperature sensation of the entire right lower limb below hip region remained the same. At the time of presentation to our institute, 3 years after the ictus, her lower limb power was normal. She could not perceive pain and temperature on the affected side; however, rest of the sensations were normal. Previous MRI was reported as normal; however, images were not available for review. She underwent a repeat MRI, which revealed a T2 hyperintensity within the right paramedian hemiconus [Figure 1A-E and A1-E1, arrows]. The conus was ending at its normal position at the lower margin of L1 vertebra. Rest of the spinal cord was normal. There was no evidence of kyphoscoliosis or any other spine abnormality.

Our second case referes to a 41-year-old female patient who underwent elective lower segment cesarean section under spinal anesthesia at the age of 31 years. She experienced electric shock-like sensation of the entire left lower limb during the procedure of spinal anesthesia. After recovery from anesthesia, she noticed complete numbness and weakness

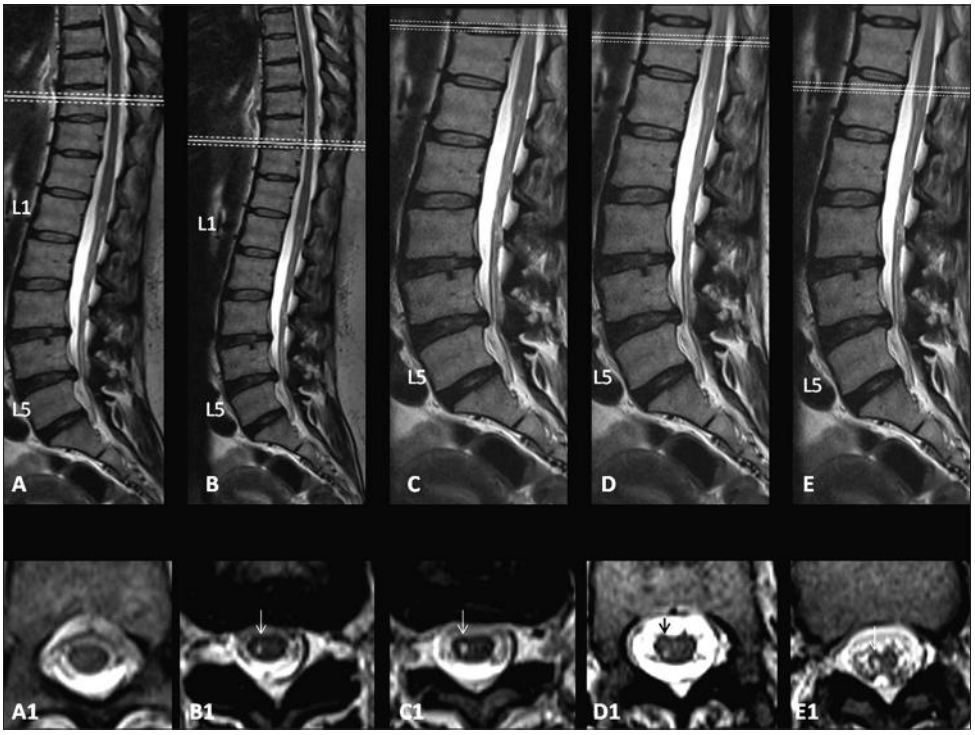

Figure 1: (A-E) Sagittal T2 thoracolumbar spine of patient 1 and (A1) Axial T2 image at T10 vertebral level shows normal cord signal intensity. Serial axial T2 sections at T10-11 disc (B1 and C1), T12 vertebral body (D1), and L1 upper vertebral body (E1) show a well-defined slit-like focal T2 hyperintensity in right hemicord/conus medullaris

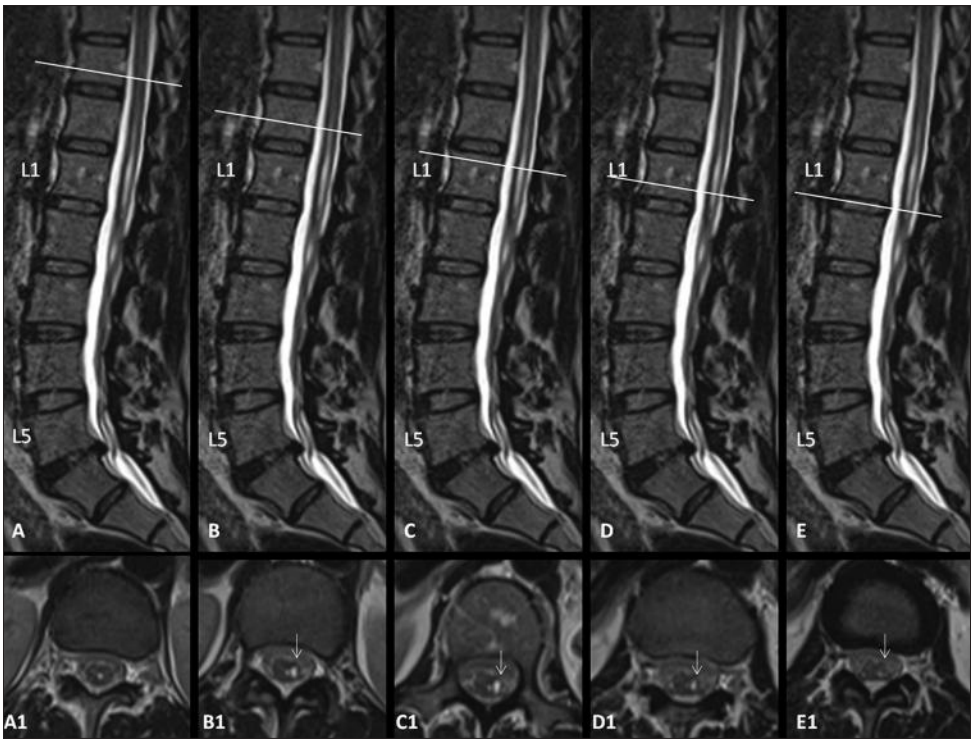

Figure 2: (A-E) Sagittal T2 images of thoracolumbar spine of patient 2. (A1-E1) Serial axial T2-weighted images at the level of lower spinal cord and conus showing slit-like T2 hyperintensity within left hemicord

below hip region in the left lower limb. Following prolonged neurorehabilitation, her weakness and numbness improved. However, she noticed that she could not appreciate pain and temperature sensations below the hip on the left side and had suffered multiple painless burns over the left lower limb. At the time of presentation to our institute, 10 years after the ictus, she was ambulant but had subtle pyramidal weakness in the form of mildly exaggerated deep tendon reflexes and a positive Babinski sign. There was complete loss of pain and temperature sensations below L1 level but the sensation for other modalities was normal. She had undergone many MRIs before her current presentation; however, all of them were reported to be normal. The MRI scans available for review, however, were poor quality and did not show any gross abnormality. There were no dedicated, thin, axial sections at the level of conus. An MRI was repeated, which showed a long segment T2 hyperintensity within the left hemiconus extending into the lower spinal cord [Figure 2]. The spinal cord was ending at the upper margin of L2 vertebral body. No kyphoscoliosis or any other spine abnormalities were seen.

Transient neurological signs and symptoms are well known during spinal anesthesia, ranging in frequency from 0.01% to 6.8%; however, none of these studies reported the MRI findings.[1-3] This transient neurological deficit was attributed to nerve trauma or intraneural injection. Persistent neurological deficits are rare (0.01–0.8%). The exact etiology of such injuries during lumbar puncture remains elusive. Direct needle injury to the cord due to the higher level of lumbar/lower thoracic puncture are known,[4-6] whereas permanent sensory deficit and transient/permanent motor deficit due to presumed 'below L2 level' puncture are extremely rare, and only a few cases have been reported in the literature.[7-9]

In both the cases in the current report, a course of variable duration of steroids was given immediately following spinal cord injury. Although both patients had undergone MRI of spine and spinal cord, it was reported as being normal. One patient's prior MRIs were reviewed and found to be of low quality and not part of a dedicated study to look for spinal cord damage. Both patients had severe weakness of the involved limb which completely improved with neurorehabilitation; however, their severe inability to perceive pain and temperature sensations remained static. Few of previously reported cases showed a variable improvement in the motor power without significant changes in the sensory symptoms[7-9] similar to our case, whereas some patients did not show any improvement. The MRI appearance is also similar in all the previously reported cases including the two case reports from the current study.

The possible proposed etiological hypothesis included spinal cord ischemia, trauma, direct toxic effect of the injected agent, and cord hemorrhage.[7] However, none of the previous

case reports demonstrated an existing MR abnormality in the spinal cord immediately after the spinal anesthesia. Significant improvement in the motor power and dorsal tract sensations without improvement in the spinothalamic tract sensations in both cases described in this study, as well as few of the previously published cases, would argue against cord hemorrhage, ischemia or even infection [Table 1]. Several common peculiar features are seen in both of our cases as well as in a few of the previously published cases. These include: (i) sudden severe pain during injection of the anesthetic agent that was found to be much more severe than that seen during needle introduction, (ii) strict unilateral cord and

Table 1: Summary of case reports/series of permanent spinal cord injury following lumbar puncture

First author, year	Case number	Age (years)/ sex	Limb involvement	Motor deficit	Sensory deficit	Follow-up	MRI findings
Hamandi, 2002	Case 1	39/female	Right	Weakness of right lower limb	Reduced sensations in L5, S1 dermatome	Neurological status unchanged	Slit-like T2 hyperintensity in right hemicord at conus
	Case 2	81/female	Left	Weakness of left lower limb	Pinprick diminished below T9	Neurological status unchanged	Slit-like T2 hyperintensity in left hemicord at conus
	Case 3	69/female	Left	Weakness of left lower limb	Sensory loss in T11 to S1 distribution	Neurological status unchanged	-"-
	Case 4	41/female	Left	Weakness of left lower limb	Sensory loss up to T9	Motor power improved, sensory deficit unchanged	-"-
	Case 5	57/female	Right	Weakness of right lower limb	Impaired sensation in right L5 to S1 distribution	Neurological status unchanged	Slit-like T2 hyperintensity in right hemicord at conus
Reynolds, 2001	Case 1	-	Right	Weakness of right lower limb	Light touch, pinprick, and vibration sense absent below the level of T8-T9	Persistent foot drop, sensory deficit	-"-
	Case 2	-	Left	Weakness of left lower limb	Sensation was reduced over the lateral side of the left lower calf, dorsum of the foot and the outer three toes	Mild improvement in both motor and sensory deficit	Slit-like T2 hyperintensity in left hemicord at conus
	Case 3	-	Left	Weakness of left lower limb	Sensory loss from L1 downwards	Mild improvement in both motor and sensory deficit	-"-
	Case 4	-	Bilateral	Weakness in both lower limbs	Sensory loss up to T4 on the right and T10 on the left side	Sensation returned to normal on the left over weeks, but remained abnormal on the right from T4 to S2 Motor power completely improved in a week	Normal
	Case 5	-	Right	Weak dorsal and plantar flexion at the ankle but normal reflexes	Sensation on the right side from L4 to S3		Slit-like T2 hyperintensity in right hemicord at conus
	Case 6	-	Left	Weakness of left lower limb	Reduced temperature and vibration sensation below the left knee, allodynia in L5 and S1, and a numb great toe	Incomplete motor recovery, persistant sensory deficit	T2 hyperintensity in left hemicord at conus
	Case 7	-	Right	Weakness of right lower limb	Pain in right lower limb	Not available	Slit-like T2 hyperintensity in right hemicord at conus
Netravathi, 2010	Case 1	21/female	Left	Weakness of left lower limb	Numbness in left lower limb	Incomplete motor and sensory recovery	T2 hyperintensity in left hemicord at conus
Pradhan, 2006	Cases 1-3	-	Bilateral	Weakness of both lower limbs	Pain and numbness of both lower limbs	Incomplete motor and sensory recovery	T2 hyperintensity in hemicord
Present case series, 2016	Case 1	40/female	Right	Weakness of right lower limb	Loss of all sensations in right lower limb	Complete motor recovery, incomplete sensory recovery	Slit-like T2 hyperintensity in right hemicord at conus
	Case 2	41/female	Left	Weakness of left lower limb	Numbness in left lower limb	Partial sensory and motor recovery	Slit-like T2 hyperintensity in Left hemicord at conus

": same as above

limb involvement, as well as shock-like sensations of lower limb, (iii) both motor and sensory involvement immediately after the injection, (iv) peak neurological deficit immediately after the injection, (v) involvement of conus in all the cases, (vi) typical central location of myelomalacia with sparing of the thin peripheral rim of parenchyma, (vii) remarkable absence of volume loss of the involved spinal cord despite myelomalacia, (viii) anteroposterior orientation of the slit-like cavity. These peculiar features favor direct anesthetic toxicity as a possible etiology. However, the pathophysiology behind the improvement of motor power and dorsal tract sensations over a period of time in a few patients as well as the complete lack of recovery of spinothalamic tract sensations remains elusive. It is presumed that the variable recovery of sensory and motor deficits depends on multiple factors including the amount of anesthetic agent injected, type of anesthetic agent or the needle used to inject the medication, and possibly other unknown factors including the genetic influence. Considering the rarity of these permanent spinal cord injuries during lumbar puncture, relative contribution of each of these component is difficult to study. Future high-resolution MRI studies undertaken immediately after the ictus and during the follow-up, and in particular, spinal cord tractography would reveal more information regaridng its etiology.

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Conflicts of interest

There are no conflicts of interest.

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