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Case Report

Severe Bilateral Leg Pain Extending to the Buttock/Saddle Region with Persistent Dense Motor Block after Spinal Anesthesia: A Case Report

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Abstract: Neurologic symptoms during recovery from spinal anesthesia may reflect residual neuraxial block, but severe pain or persistent motor block can also indicate a compressive neuraxial emergency. A 49-year-old man underwent right knee arthroscopic surgery under spinal anesthesia with 14 mg of 0.5% hyperbaric bupivacaine. The neuraxial procedure was uncomplicated, and perioperative coagulation status was normal. At 225 minutes after intrathecal injection, he developed severe bilateral lower-extremity tightening and twisting pain extending to the buttock/saddle region, with Medical Research Council grade 0 motor strength in bilateral hip, knee, and ankle movements. Gross distal sensation in both feet was documented as present. Because cauda equina syndrome, spinal epidural hematoma, or another acute compressive neuraxial lesion could not be excluded clinically, urgent lumbosacral magnetic resonance imaging was obtained. Magnetic resonance imaging demonstrated left-sided disc extrusion at L4-L5 and L5-S1 with compression of the left L4 and S1 nerve roots; no epidural hematoma, central canal compression, or acute cauda equina compression was reported. The patient was managed conservatively with close observation and symptomatic treatment. Motor function recovered within hours, and at outpatient follow-up 29 days after surgery, no specific abnormality was documented. Severe bilateral leg pain with persistent motor block after spinal anesthesia should prompt structured neurologic reassessment and urgent imaging when compressive neuraxial pathology cannot be excluded clinically.

Keywords: Anesthesia, Spinal, Cauda Equina Syndrome, Bupivacaine, Magnetic Resonance Imaging, Postoperative Complications.

INTRODUCTION

Neurologic symptoms after spinal anesthesia may result from residual neuraxial block, compressive neuraxial pathology, surgical or positioning-related nerve injury, preexisting spinal disease, or, less commonly, local anesthetic-related injury [1, 2]. Because early clinical findings may overlap, severe pain, delayed motor recovery, or new neurologic signs during recovery from neuraxial anesthesia require structured evaluation and timely exclusion of treatable causes.

Diagnosis of structural cauda equina syndrome (CES) requires clinical interpretation together with imaging evidence of neural compression. Contemporary guideline reviews emphasize urgent magnetic resonance imaging (MRI) when CES is suspected, particularly with perianal or saddle sensory disturbance, bladder or bowel dysfunction, bilateral radicular pain, or new motor weakness [3]. Transient neurologic symptoms (TNS) after spinal anesthesia usually occur after apparent recovery from block and are characterized by pain or dysesthesia in the back, buttocks, thighs, or legs, typically without dense motor deficit [4, 5].

We report severe bilateral lower-extremity pain with persistent motor block during regression of hyperbaric bupivacaine spinal anesthesia. Urgent MRI excluded surgically actionable neuraxial pathology, and motor function recovered within hours.

CASE REPORT

A 49-year-old man (height, 160 cm; weight, 65 kg; American Society of Anesthesiologists physical

status II) with hypertension and hyperlipidemia was scheduled for right knee arthroscopic surgery for medial and lateral meniscal tears. He had no known neurologic disease or previous anesthetic complications. Preoperatively, he reported right knee pain with an NRS score of 6/10, but neurologic examination was otherwise unremarkable. Preoperative platelet count was 309 × 10^3/μL, prothrombin time-international normalized ratio was 0.88, and activated partial thromboplastin time was 32.2 seconds. He had no antiplatelet or anticoagulant exposure, perioperative anticoagulation, renal dysfunction, traumatic spinal puncture, or bloody tap.

Spinal anesthesia was performed at 12:15 (defined as T0) at the L3-L4 interspace using a 23-gauge Quincke needle via a midline approach in the right lateral decubitus position. After free flow of clear cerebrospinal fluid was confirmed, 14 mg of 0.5% hyperbaric bupivacaine was injected intrathecally at approximately 0.5 mL/s. No paresthesia or pain occurred during needle placement or injection. After supine positioning, bilateral pinprick loss to T10 was confirmed at 12:25.

Dexmedetomidine was administered for sedation as a loading dose of 1 μg/kg followed by an infusion at 0.5 μg/kg/h. Ramosetron 0.3 mg was administered immediately after spinal anesthesia. Surgery began at 12:40 and ended at 13:10. A pneumatic tourniquet was inflated on the operative limb at 250 mmHg from 12:40 to 13:05. No intraoperative hypotension, hypoxia, or major blood loss was recorded.

The patient arrived in the postanesthesia care unit at 13:10 and received acetaminophen 1,000 mg intravenously, ibuprofen 300 mg intravenously, and patient-controlled analgesia (PCA) containing ketorolac 90 mg, nefopam 50 mg, and ramosetron 0.3 mg. At discharge to the ward at 13:40, his Numeric Rating Scale (NRS) pain score was 0/10, and no nausea or vomiting was reported. Circulation in both feet was intact, but gross distal sensation and motor function in both feet remained absent, and active ankle dorsiflexion and plantar flexion were absent bilaterally. At 14:00, spontaneous voiding was observed; voided volume and postvoid residual volume were not documented. The postoperative course is summarized in Table 1.

At 15:00, circulation in both feet remained intact, but gross distal sensation and motor function in both feet remained absent. At 16:00, 225 minutes after intrathecal injection, the patient developed severe bilateral lower-extremity tightening and twisting pain extending to the buttock/saddle region, became markedly distressed, and requested additional treatment. The NRS pain score was 7–8/10. Circulation and gross distal sensation in both feet were documented as present. Motor strength was Medical Research Council grade 0 in bilateral hip, knee, and ankle movements, consistent with persistent dense motor block. Formal dermatomal sensory mapping, reflex testing, objective saddle sensory assessment, anal sphincter tone assessment, and postvoid residual volume measurement were not recorded.

Because CES, spinal epidural hematoma, or another acute compressive neuraxial lesion could not be excluded clinically, urgent lumbosacral MRI was requested. Tramadol 50 mg intramuscularly and piroxicam 20 mg intramuscularly were administered. At 16:30, the anesthesia and orthopedic teams evaluated the patient, explained the concern for spinal epidural hematoma or another compressive neuraxial lesion to the patient and family, and urgent MRI was performed.

Emergent lumbosacral MRI showed no epidural hematoma, no central canal compression, and no acute cauda equina compression. The MRI report also described L3–4 disc bulging, left extraforaminal L4–5 disc extrusion compressing the exiting left L4 nerve root, and left subarticular L5–S1 disc extrusion compressing the left S1 nerve root. Representative images are shown in Fig. 1.

Because MRI identified no surgically actionable compressive lesion, the patient was managed conservatively with close observation and symptomatic treatment. At 18:00, severe pain had resolved, although lower-extremity numbness persisted; pregabalin 50 mg was administered orally. Methylprednisolone sodium succinate 62.5 mg was administered intravenously at 18:30. By 19:30, circulation, motor function, and gross distal sensation in both feet were intact; active ankle dorsiflexion and plantar flexion were present bilaterally; and the NRS pain score was 3/10, although numbness persisted in the operative right leg. By 09:00 on postoperative day 1, only surgical-site pain was reported. At outpatient follow-up 29 days after surgery, no specific abnormality was documented.

DISCUSSION

This case is best understood as a diagnostic emergency rather than as a proven specific neurologic syndrome. The key event was new severe bilateral lower-extremity pain extending to the buttock/saddle region at 225 minutes after intrathecal injection, accompanied by MRC grade 0 motor strength in bilateral hip, knee, and ankle movements. This combination created a CES-like warning presentation rather than routine postoperative pain or uncomplicated block regression. The absence of pain at postanesthesia care unit discharge made routine surgical-site discomfort less likely as the sole explanation. Because this presentation overlapped clinically with possible CES, spinal epidural hematoma, and other compressive neuraxial lesions, urgent MRI was appropriate and excluded surgically actionable pathology. The differential diagnosis and interpretation are summarized in Table 2.

The apparent dissociation between documented gross distal sensation and persistent dense motor block should be interpreted cautiously. Bedside documentation

did not include dermatomal mapping, reflex testing, sacral sensory assessment, anal sphincter tone assessment, or postvoid residual volume measurement. Therefore, severe bilateral pain with persistent motor block could not be safely attributed to routine block regression before imaging.

This case does not establish structural CES. CES descriptions emphasize sacral sensory disturbance and bladder or bowel dysfunction, often with bilateral radicular pain and variable motor weakness, and suspected cases warrant urgent MRI to establish or exclude compression [3,6]. In this patient, the extension of pain to the buttock/saddle region and bilateral MRC grade 0 motor weakness increased the clinical resemblance to CES. However, spontaneous voiding was observed and MRI did not report acute cauda equina compression. Because objective saddle sensory testing, anal sphincter tone assessment, and postvoid residual volume measurement were not documented, incomplete CES could not be fully excluded at the bedside before imaging.

The presentation was also atypical for classic TNS, which generally appears after recovery from spinal anesthesia and causes back, buttock, thigh, or leg pain or dysesthesia without profound persistent motor block [4, 5]. Bupivacaine is associated with lower TNS risk than lidocaine [4]. Here, severe bilateral pain developed before complete block regression while ankle dorsiflexion and plantar flexion remained absent bilaterally.

Prolonged or atypical regression of spinal anesthesia remains plausible. The patient received 14 mg of hyperbaric bupivacaine, and intravenous dexmedetomidine was administered; intravenous dexmedetomidine can prolong sensory and motor block during bupivacaine spinal anesthesia [7]. Persistent motor block alone therefore would not establish neurologic injury. The concerning feature was the superimposed severe bilateral tightening and twisting pain during persistent motor block.

MRI demonstrated unilateral left-sided disc extrusion at the L4-L5 extraforaminal zone and L5-S1 subarticular zone, with compression of the left L4 and S1 nerve roots. These findings may have represented a vulnerable substrate, because neuraxial anesthesia in patients with spinal canal or nerve-root pathology can be difficult to interpret and may rarely be associated with worsening neurologic symptoms [2,8]. Nevertheless, the unilateral abnormalities did not show central canal or cauda equina compression and did not adequately explain the abrupt bilateral symptoms with rapid motor recovery.

The mechanism remains undetermined. Possible explanations include atypical block regression or prolongation, transient lumbosacral nerve-root irritation, local anesthetic distribution effects, or another reversible neuraxial phenomenon in the setting of lumbar degenerative disease. The rapid motor recovery and absence of a documented persistent abnormality at follow-up argue against established local anesthetic neurotoxicity or structural nerve-root injury. Empiric methylprednisolone was administered during observation, so its contribution cannot be determined.

This case is limited by incomplete objective neurologic documentation at symptom onset. Although bilateral hip, knee, and ankle motor strength was documented as MRC grade 0, formal dermatomal sensory mapping, reflex testing, objective saddle sensory assessment, anal sphincter tone assessment, and postvoid residual bladder volume measurement were not recorded. These limitations restrict causal inference but do not alter the central clinical message: severe bilateral pain with persistent motor block after spinal anesthesia warrants urgent exclusion of compressive neuraxial pathology when it cannot be excluded clinically.

Table 1: Clinical timeline of postoperative neurologic symptoms and recovery

Time	Clinical event	Key findings and management
12:00	Operating room entry	The patient entered the operating room and anesthesia care began.
12:15 (T0)	Intrathecal injection completed	Spinal anesthesia was performed at L3-L4 using a 23-gauge Quincke needle via a midline approach in the right lateral decubitus position. After free flow of clear cerebrospinal fluid was confirmed, 0.5% hyperbaric bupivacaine, 14 mg, was injected intrathecally. No paresthesia, pain, or bloody tap occurred.
12:25 (T+10 min)	Block level confirmed	Bilateral pinprick loss to T10 was confirmed.
12:40-13:10 (T+25-55 min)	Surgery and tourniquet	Right knee arthroscopic surgery was performed supine. The tourniquet was inflated at 250 mmHg from 12:40 to 13:05, for 25 minutes. No intraoperative hypotension, hypoxia, or major blood loss was recorded.
13:10 (T+55 min)	PACU arrival	Surgery and anesthesia ended. Acetaminophen 1,000 mg IV and ibuprofen 300 mg IV were administered. PCA contained ketorolac 90 mg, nefopam 50 mg, and ramosetron 0.3 mg.
13:40 (T+85 min)	PACU discharge and ward transfer	NRS pain score was 0/10, and no nausea or vomiting was reported. Circulation in both feet was intact, but gross distal sensation and motor

Time	Clinical event	Key findings and management
		function in both feet were absent. Active ankle dorsiflexion and plantar flexion were absent bilaterally.
14:00 (T+105 min)	Spontaneous voiding	Spontaneous voiding was observed after ward transfer. Voided volume was not documented, and postvoid residual volume was not measured.
15:00 (T+165 min)	Persistent block documented	Circulation in both feet remained intact, but gross distal sensation and motor function in both feet were absent. Active ankle dorsiflexion and plantar flexion were absent bilaterally.
16:00 (T+225 min)	Severe bilateral leg pain	The patient reported severe tightening and twisting pain in both legs extending to the buttock/saddle region, with an NRS pain score of 7-8/10. Circulation and gross distal sensation in both feet were documented as present. Motor strength was MRC grade 0 in bilateral hip, knee, and ankle movements. Formal dermatomal, reflex, objective saddle sensory, anal sphincter tone, and bladder assessments were not recorded.
16:30 (T+255 min)	Urgent MRI and counseling	The anesthesia and orthopedic teams evaluated the patient, explained the concern for spinal epidural hematoma or another compressive neuraxial lesion, and proceeded with urgent lumbosacral MRI. Tramadol 50 mg IM and piroxicam 20 mg IM were administered.
MRI result	Compressive lesion excluded	MRI reported left extraforaminal L4-L5 disc extrusion compressing the left L4 nerve root, left subarticular L5-S1 disc extrusion compressing the left S1 nerve root, and L3-L4 disc bulging. No epidural hematoma, central canal compression, acute cauda equina compression, or other surgically actionable compressive lesion was reported.
18:00 (T+345 min)	Pain resolved; numbness persisted	Severe pain had resolved, but subjective lower-extremity numbness persisted. Pregabalin 50 mg PO was administered.
18:30 (T+375 min)	Corticosteroid administered	Methylprednisolone sodium succinate 62.5 mg IV was administered.
19:30 (T+435 min)	Motor recovery documented	Circulation, motor function, and gross distal sensation in both feet were intact. Active ankle dorsiflexion and plantar flexion were present bilaterally. NRS pain score was 3/10, and numbness persisted in the operative right leg.
POD 1, 09:00	Neurologic symptoms improved	Only surgical-site pain was reported.
POD 29	Outpatient follow-up	No specific abnormality was documented.

T0, completion of intrathecal bupivacaine injection at 12:15; NRS, Numeric Rating Scale; MRI, magnetic resonance imaging; PACU, postanesthesia care unit; PCA, patient-controlled analgesia; IV, intravenous; IM, intramuscular; PO, oral; POD, postoperative day.

Table 2: Differential diagnosis and interpretation

Differential diagnosis	Interpretation in this case
Spinal epidural hematoma or other compressive neuraxial lesion	Severe bilateral pain with persistent motor block made this the main emergency diagnosis to exclude. Coagulation status and puncture history were reassuring, but imaging was still required. Urgent MRI reported no epidural hematoma, central canal compression, or acute cauda equina compression.
Cauda equina syndrome/incomplete CES	Severe bilateral pain extending to the buttock/saddle region with bilateral MRC grade 0 motor strength increased concern for CES-like presentation. Structural CES was unlikely after MRI and spontaneous voiding, but objective saddle sensory examination, anal sphincter tone assessment, and postvoid residual volume measurement were not documented; therefore, incomplete CES could not be excluded clinically before imaging.
Prolonged or atypical regression of spinal anesthesia	Hyperbaric bupivacaine and intravenous dexmedetomidine may have contributed to persistent dense motor block. However, new severe bilateral tightening and twisting pain extending to the buttock/saddle region was atypical for routine block regression alone.
Transient neurologic symptoms	TNS was considered because postoperative bilateral leg pain occurred after spinal anesthesia. The timing before complete block regression and the presence of persistent dense motor block made classic TNS an incomplete explanation.
Preexisting lumbar disc disease	MRI showed unilateral left L4 and S1 nerve-root compression, which may have represented a vulnerable substrate. The unilateral findings did not show central canal or cauda equina compression and did not adequately explain the abrupt bilateral symptoms.

Differential diagnosis	Interpretation in this case
Peripheral or surgical causes	Tourniquet neuropathy, compartment syndrome, positioning-related neuropathy, and ischemic neuraxial injury were considered unlikely because symptoms were bilateral, circulation remained intact, surgery was supine with a 25-minute unilateral tourniquet, and motor recovery occurred within hours.

CES, cauda equina syndrome; MRI, magnetic resonance imaging; TNS, transient neurologic symptoms.

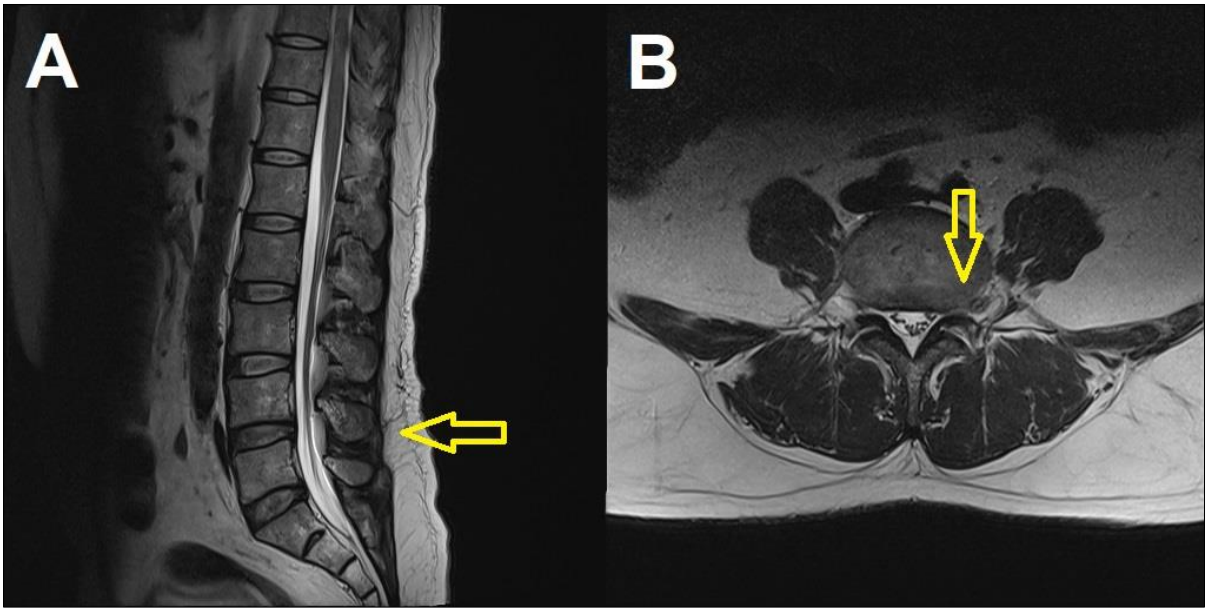

Fig. 1: (A) Sagittal T2-weighted image shows no epidural hematoma, central canal compression, or acute cauda equina compression. (B) Axial T2-weighted image at L4–5 shows left extraforaminal disc extrusion with involvement of the exiting left L4 nerve root. Additional reported MRI findings included L3–4 disc bulging and left subarticular L5–S1 disc extrusion affecting the left S1 nerve root.

CONCLUSION

Severe bilateral lower-extremity pain extending to the buttock/saddle region with persistent dense motor block during recovery from spinal anesthesia should be treated as a potential neuraxial emergency until compressive pathology is excluded. Urgent MRI is warranted when cauda equina syndrome, spinal epidural hematoma, or another surgically actionable lesion cannot be excluded clinically. Rapid recovery after negative imaging may suggest a reversible nonstructural process, but incomplete sacral and bladder assessment limits definitive etiologic interpretation.

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Conflict of Interest: No potential conflict of interest relevant to this article was reported.

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Ethics Statement

This case report was approved by the Institutional Review Board (IRB) of the Public Institutional Bioethics Committee designated by the Ministry of Health and Welfare, Republic of Korea (approval number: 2026-1129-001). The requirement for informed consent was waived by the IRB.

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