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Case Report

Possible Role of Local Anesthetics in Permanent Lower Limb Motor Paralysis after Epidural Anesthesia: A Case Report

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We present a case of permanent bilateral lower limb paralysis after epidural anesthesia. A 71-year-old woman (height 159 cm; weight 48.5 kg; American Society of Anesthesiologists Physical Status 2) with a history of hypertension (treated with nifedipine), benign goiter (under surveillance), surgeries for appendicitis, and a previous left humerus fracture had received general anesthesia with epidural anesthesia during two surgical procedures, namely, laparoscopic-assisted low anterior resection with colostomy and laparoscopic-assisted colostomy closure. She developed left-predominant lower limb paralysis after the first epidural anesthesia (using ropivacaine and levobupivacaine). The symptoms had no identifiable cause, persisted after removal of the epidural catheter, and gradually resolved during rehabilitation. Her lower limb paralysis recurred and progressed, however, after the second epidural anesthesia (using levobupivacaine alone), and she has abnormal spinal reflexes and elevated myelin basic protein in cerebrospinal fluid. Although these findings suggested that bilateral lower limb paralysis was caused by a lesion in the central nervous system (thoracolumbar spinal cord), postoperative MRI scans of the vertebrae/spinal cord and head failed to identify the site of the damage. We concluded that permanent bilateral lower limb paralysis was likely caused by epidural anesthesia, but the mechanism could not be identified.
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Keywords: epidural anesthesia, local anesthetics, permanent motor paralysis

Introduction

Epidural anesthesia is a well-established procedure for intraoperative and postoperative pain management. Its complications include hypotension[1], bradycardia[2], numbness of the legs[3], muscle weakness[4], paresthesia[5], and urinary retention[6], which generally resolve after cessation of epidural anesthesia. However, serious complications such as permanent motor paralysis, sensory abnormalities, epidural abscess[7], epidural hematoma[8], spinal cord infarction[9], and epidural compartment syndrome[10] may persist.

We report a case of permanent bilateral lower limb paralysis apparently related to epidural anesthesia; however, the mechanism responsible could not be identified despite careful examination, including magnetic resonance imaging (MRI).

Case Report

A 71-year-old woman (height 159 cm; weight 48.5 kg; American Society of Anesthesiologists Physical Status 2) had a history of hypertension treated with nifedipine, benign goiter under follow-up, surgeries for appendicitis, and a left humerus fracture. The patient later died of an unrelated condition, and written informed consent for publication of this report was obtained from her hus-

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Table 1 Results of manual muscle testing

		Results (right/left)				
		First surgery		Second surgery		
Muscle	Innervation	POD 4	POD 35	POD 4	POD 32	Transfer day
Iliopsoas	L1–3	3/2	3/1	2/1	3/1	3/2
Quadriceps	L2–4	5/2	4/1	4/1	4/1	3/2
Hamstring	L5–S2	5/5	4/2–3	4/1	4/1	5/2
Anterior tibialis	L4–5	5/5	3–4/3	5/1	5/2	5/1
Extensor hallucis longus	L4–S1	5/5	No data	No data	No data	No data
Gastrocnemius	S1–2	No data	3–4/2–3	5/1	5/3	5/1

L: lumbar vertebra, S: sacral vertebra, POD: postoperative day.

band.

The patient had undergone two operations. The first involved scheduled laparoscopic-assisted low anterior resection and colostomy with general anesthesia and epidural anesthesia. Epidural anesthesia was administered via the vertebral space between thoracic vertebrae (Th) 11 and 12. A catheter was placed 10 cm beneath the skin and 5.0 cm in the epidural space in the cranial direction. No bleeding, radiating pain, or dural puncture were observed during the procedure.

General anesthesia was induced with 80 mg propofol, 100 μg fentanyl, and 50 mg rocuronium, with remifentanil administered at approximately 0.15 μg/kg/min, followed by tracheal intubation. At 23 min after induction of anesthesia, a single dose of 3 mL of 0.75% ropivacaine with 3 mL of physiological saline was administered into the epidural space, followed 64 min later by another single dose of 2 mL of 0.75% ropivacaine with 2 mL of physiological saline. Anesthesia was maintained with remifentanil 0.06–0.12 μg/kg/min and desflurane 5%, with rocuronium as required. The duration of the operation was 6 h 17 min, with the patient in the lithotomy position.

At 1 h 12 min after the initial ropivacaine administration, continuous infusion of 100 mL 0.25% levobupivacaine, 100 mL physiological saline, 20 mL fentanyl (1,000 μg), and 2 mL droperidol (5 mg) was administered into the epidural space at a rate of 4 mL/h. The patient was aroused immediately after the end of anesthesia and was returned to the ward with normal vital signs. The total anesthesia time was 7 h 36 min.

The patient reported weakness and hypoesthesia in her left thigh on postoperative day (POD) 1, and continuous epidural infusion was discontinued on POD 2. She continued to notice weakness of the right thigh even after discontinuation of the infusion, and the epidural catheter was thus withdrawn on the same day. Iliopsoas and leg muscle weakness, predominantly on the left side, appeared on POD 3. The patient underwent manual muscle testing (MMT) by an orthopedist on POD 4, and the results suggested that the left first to fourth lumbar nerves were damaged (**Table 1**). Hypoalgesia was evident at the level of left lumbar vertebrae (L) 2 and 3, with attenuation of the left patellar and left Achilles reflexes.

Lumbar spinal MRI on POD 7 showed bulging of the L3/4 and L4/5 intervertebral discs but no obvious stenosis of the bilateral intervertebral foramina or vertebral canal. The diagnosis by radiologists was intervertebral disc degeneration.

The patient continued rehabilitation in hospital. Her right thigh muscles gradually became stronger, but there was no improvement in the sensory deficit of the left leg. She was transferred to another hospital on POD 38 to continue rehabilitation.

The patient's second operation was an open colostomy closure, scheduled for day 116 after the first operation. Preoperative investigations before the second operation showed persistent sensory loss in the front of the left thigh and numbness of the left lower leg and foot, but improved leg muscle strength, which enabled her to walk with a cane. Open surgery was selected, and epidural anesthesia, in addition to general anesthesia, was planned to lessen expected postoperative pain. Although the procedure was switched from open to laparoscopic surgery on the day of the procedure, the method of anesthesia was unchanged, to reduce postoperative pain.

Epidural anesthesia was performed via the vertebral space between Th12 and L1 by placing a catheter 12 cm beneath the skin and 8.0 cm in the epidural space in the cranial direction. No bleeding, radiating pain, or dural puncture was observed during the procedure. A single

Table 2 Neurological reflexes

		Results (right/left)				
		First surgery		Second surgery		
	Innervation	POD 4	POD 35	POD 4	POD 32	Transfer day
Pathologic reflexes						
Babinski's	L4–S1	No data	No data	+/+	+/+	No data
Chaddock's	L4–S1	No data	No data	+/+	+/+	No data
Deep tendon reflexes						
Achilles tendon	S1–S2	No data	No data	−/−	−/+	No data
Patellar tendon	L4	No data	No data	−/−	−/−	No data

L: lumbar vertebra, S: sacral vertebra, POD: postoperative day.

dose of 7 mL of 0.25% levobupivacaine was administered into the epidural space. General anesthesia was induced 1 min later with 80 mg propofol, 100 μg fentanyl, and 50 mg rocuronium, with remifentanil administered at approximately 0.27 μg/kg/min, followed by tracheal intubation. Anesthesia was maintained with remifentanil 0.06–0.1 μg/kg/min and sevoflurane 1.5%, with rocuronium as required.

At 28 min after the initial administration of 0.25% levobupivacaine, a further 5 mL 0.25% levobupivacaine was administered into the epidural space, and 1 h and 29 min after the initial administration of 0.25% levobupivacaine, a continuous infusion of 100 mL 0.25% levobupivacaine, 100 mL physiological saline, and 10 mL fentanyl (500 μg) was administered into the epidural space at a rate of 4 mL/h.

With the patient in supine position, surgery was performed and completed with no unexpected events, with an operating time of 1 h 14 min. The patient was aroused immediately at the end of anesthesia and was returned to the ward with normal vital signs. The total anesthesia time was 1 h 59 min.

From POD 1, the patient experienced weakness of the legs, predominantly on the left side, and sensory loss at the front of the left thigh, similar to her experience after the previous operation, and the epidural catheter was withdrawn on POD 2. She was examined by a neurologist on POD 4, who confirmed muscle weakness in the left leg. Atrophy of the left quadriceps muscle was also observed. MMT findings are shown in **Table 1**. Babinski's and Chaddock's reflexes were positive on the left and right sides, but Achilles and patellar reflexes were absent on both legs (**Table 2**). Pain sensitivity (right/left [R/L]) was 5/0 for Th7–L2 and 10/0 for L3–4 (full sensitivity = 10). The position sense of the lower extremities was normal. The vibration sensitivity (R/L) was 20/16 s for the head of the radius and 12/8 s for the medial malleolus. Bladder/bowel dysfunction was also noted.

Atrophy of the left quadriceps muscle remained on POD 32. Babinski's and Chaddock's reflexes remained positive on both sides, and Achilles reflex was positive on the left side but absent on the right side, while the patellar reflex remained absent for both legs (**Table 2**). Pain sensitivity was 5/5 for Th7–L2 and 10/0 for L3–4. The vibration sensitivity was 16/13 s for the head of the radius, 5/5 s for the iliac crest, 10/7 s for the patella, and 10/7 s for the medial malleolus.

Vertebral/spinal cord MRI was performed on POD 6. T2 prolongation was observed at the level of the lower thoracic spine (**Figure 1**), suggesting spinal cord damage such as myelitis and spinal cord ischemia. There were no clear signal changes on T1-weighted images (**Figure 1**) and no sign of acute fracture or inflammatory changes of the spine.

Repeat vertebral/spinal cord MRI on POD 10 showed loss of the T2 prolongation observed at the lower thoracic spine observed on POD 6 (**Figure 2**). T2 prolongation was not observed in contrast-enhanced MRI performed on POD 32, and there were no other abnormal findings.

The loss of T2 prolongation observed on POD 6 and elevation of the myelin basic protein level suggested possible transient myelitis; however, we considered it unlikely that this was the cause of the symptoms in our patient because T2 prolongation had resolved in 4 days.

Lumbar puncture for cerebrospinal fluid (CSF) testing was performed on POD 12. CSF pressure was 18 cm H_2O at the beginning and 12 cm H_2O at the end. The CSF was clear yellow and the cell count was 7/μL (upper limit 5 μL), with 100% mononuclear cells. The CSF glucose level was 53 mg/dL (reference value 50–75 mg/dL) and the blood glucose level was 89 mg/dL (reference value 73–

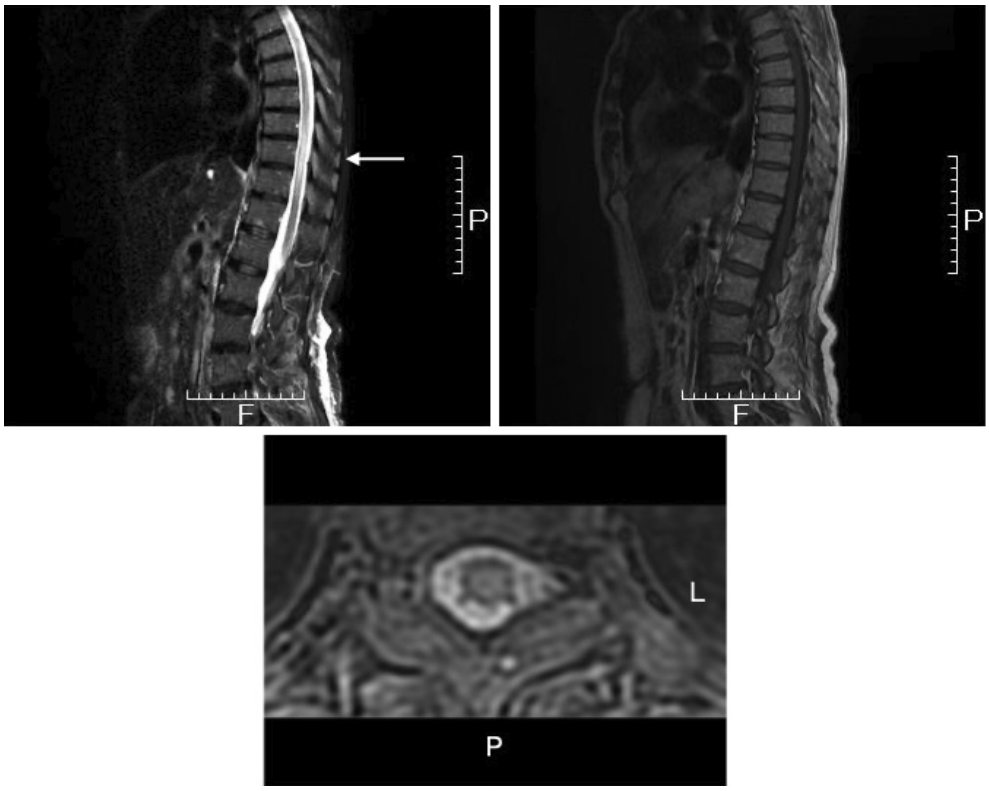

Figure 1 MRI scans of the spinal cord on postoperative day 6 after the second operation
Top: sagittal views of the spinal cord. Top left (T2-weighted imaging): hyperintensity is evident at Th9–12 (white arrow); top right (T1-weighted imaging): no obvious signal changes. Bottom: axial view of T2-weighted imaging; section is indicated by white arrow.
P: posterior side, F: foot side, L: left side.

109 mg/dL). The CSF protein level was 45 mg/dL (reference value 10–40 mg/dL) and the chloride level was 127 mEq/L (reference value 120–125 mEq/L). Notably, the myelin basic protein level was elevated at 747.7 pg/mL (reference value ≤102 pg/mL).

A nerve conduction study on POD 13 showed normal motor nerve conduction velocity (left tibial nerve 44.1 m/s; right tibial nerve 47.6 m/s). The F-waves of the left tibial nerve and left median nerve showed normal minimal latencies for both nerves, but the F-wave of the left tibial nerve showed a low frequency and low waveform diversity (**Figure 3**). These findings suggested a proximal abnormality in the thoracolumbar spinal cord or nerve roots.

Itoh et al.[11] administered steroids to a patient with idiopathic paraplegia after epidural anesthesia. Although treatment was delayed, we administered steroid pulse therapy (methylprednisolone 1,000 mg/day) to our patient from POD 20 to POD 22 after the second operation, with no discernible effect.

The patient was again transferred to another hospital for rehabilitation on POD 38. MMT (R/L) scores at transfer are shown in **Table 1**. Sensory testing showed moderate tactile hypoalgesia in left Th10–L3 and mild tactile hypoalgesia near right L1.

Discussion

Epidural anesthesia is a common procedure for intraoperative and postoperative pain management. Our patient experienced left-predominant lower limb paralysis after her first surgery, which, given the time course, was considered to be potentially caused by epidural anesthesia. Paralysis may be caused by direct nerve injury, radiculopathy, or damage by a Cathelin or Tuohy needle[12], as well as by nerve compression, indwelling catheters, an epidural abscess[7], epidural hematoma[8], or spinal cord infarction[9]. Direct nerve injury was considered unlikely because she did not complain of any radiating pain during administration of the epidural anesthesia (either during the advance of the Tuohy needle or during catheter insertion), and there was no retrograde flow of CSF from the Tuohy needle. There were no indications of epidural puncture during either surgery and no evidence of an epidural abscess, epidural hematoma, or spinal cord in-

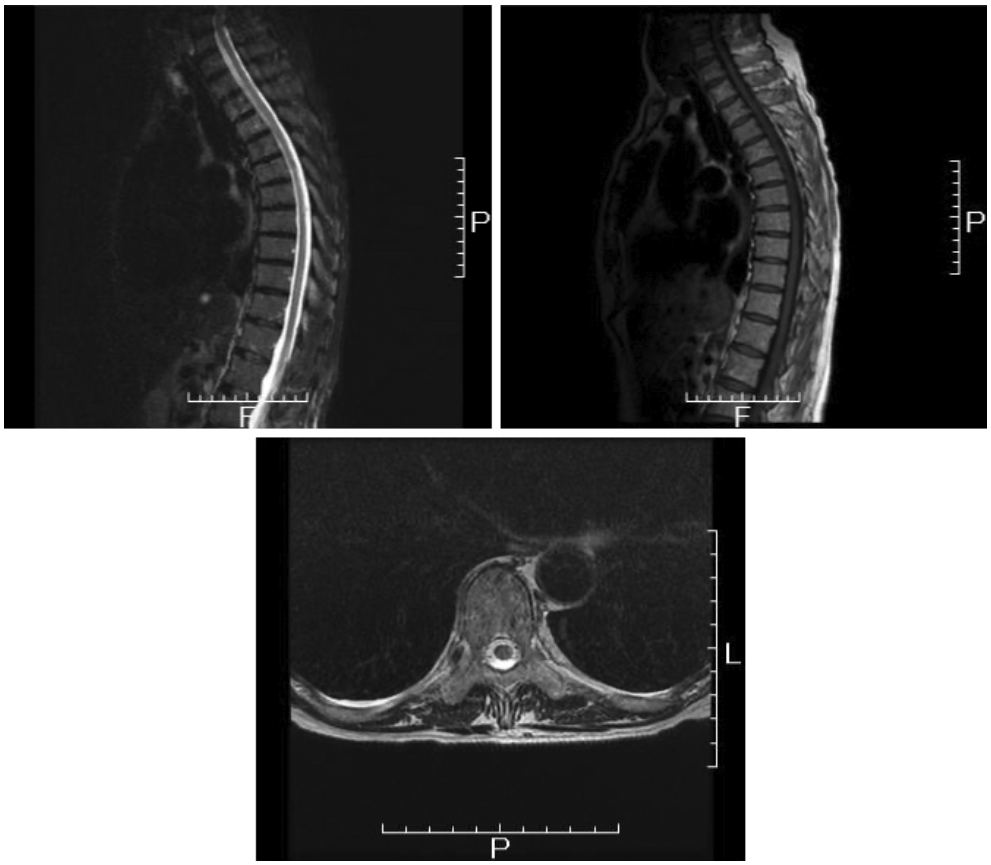

Figure 2 MRI scans of the spinal cord on postoperative day 10 after the second operation
Top: sagittal views of the spinal cord. Top left (T2-weighted imaging): the hyperintense region in **Figure 1** is not visible; top right (T1-weighted imaging): no obvious signal changes. Bottom: axial view of T2-weighted imaging; section near Th11 with no obvious signal changes.
P: posterior side, F: foot side, L: left side.

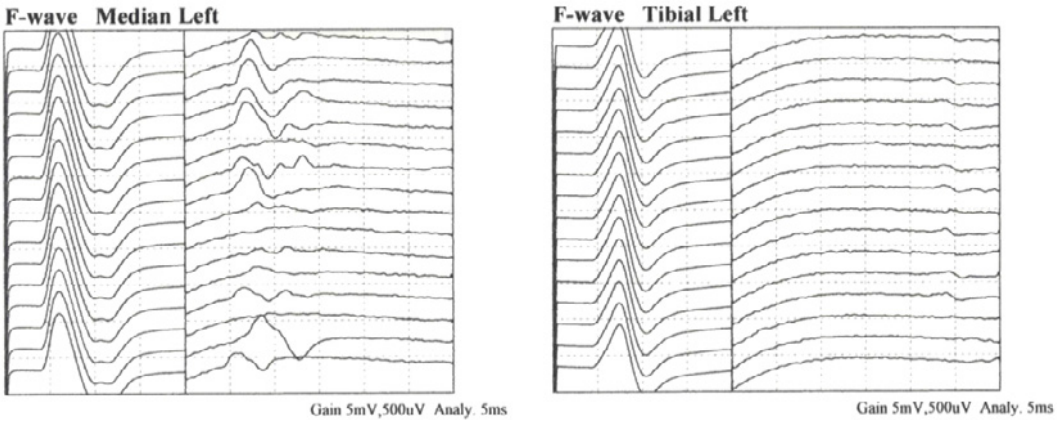

Figure 3 F-waves in the left median nerve (left) and left tibial nerve (right)
Diversity has been lost in the left tibial nerve.

farction on MRI. The cause of paralysis and the other symptoms thus remains unclear.

Regarding the neurological findings, the left nerve roots of left L2–L4 and right L3 appeared damaged on POD 4 after the first surgery. The MMT worsened until the patient was discharged from hospital after the second surgery.

After the second surgery, Achilles and patellar reflexes were absent in both legs, indicating damage to the spinal roots of L2–S1, possibly due to radiculopathy after the

first surgery. Babinski's and Chaddock's reflexes were positive on the left and right sides on POD 4 after the second surgery. Pain sensitivity (R/L) was 5/0 for Th7–L2 and 10/0 for L3–4, and the position sense of both lower extremities was normal. The vibration sensitivity (R/L) was 20/16 s for the head of the radius and 12/8 s for the medial malleolus, with a weaker vibration sense in the medial malleolus than in the head of the radius, and more apparent weakness on the left side. Bladder/bowel dysfunction was also observed. The above findings suggested radiculopathy of the nerve roots of L3–S1, predominantly on the left side, after the first surgery, and spinal cord injury at the level of Th7–L2, possibly due to pyramidal tract disorder after the second surgery.

The neurological findings and vertebral levels of epidural anesthesia (Th11/12 for the first surgery and Th 12/L1 for the second surgery) suggest that epidural anesthesia was the most likely cause of our patient's symptoms. However, we found no organic nerve damage or injury, making it difficult to identify the exact level of the spinal cord or nerve root lesions.

Myelin basic protein[13] is present in the myelin sheath, in oligodendroglial cells in central nervous tissue, and in Schwann cells in peripheral tissue. Elevated levels of myelin basic protein in spinal fluid may thus indicate structural nerve damage. In the present case, elevated CSF levels of myelin basic protein suggested that the responsible lesion was located in the central nervous system (CNS) rather than in peripheral nerves. Our patient's pathological reflexes and bladder and rectal dysfunction after the second operation suggested that motor paralysis and paresthesia were caused by CNS damage rather than by peripheral nerve damage; however, postoperative vertebral/spinal cord and cranial MRI failed to identify any CNS damage.

The local anesthetics used in the present case were ropivacaine and levobupivacaine for the first operation and levobupivacaine for the second operation. Local anesthetics are known to be neurotoxic[14–18]. The package inserts note that ropivacaine can cause lower limb paresthesia and motor dysfunction in <1% of cases, and levobupivacaine can cause hypoesthesia in 1% to <5% and motor dysfunction in <1% of cases, as well as paralysis, sensory disturbance, and decreased motor function (incidence rates unknown). However, these symptoms were only present during the use of local anesthetics, suggesting that these drugs were unlikely to cause long-term persistent paralysis, as in the present case. The total volume and concentration of local anesthetics used during surgery were 10 mL of 0.375% ropivacaine as single injections and 200 mL of 0.125% levobupivacaine as a continuous infusion (at 4 mL/h) during the first operation, and 12 mL of 0.25% levobupivacaine as single injections and 200 mL of 0.125% levobupivacaine as a continuous infusion (at 4 mL/h) for the second operation. The possibility of local anesthetic overdose should be considered. The maximum ropivacaine dose is 150–200 mg and the maximum dose of levobupivacaine is 150 mg. In our patient, the initial dose of ropivacaine was 37.5 mg for the first surgery and 30 mg for the second surgery, and continuous-dose administration of levobupivacaine was 250 mg for 2 days for the first surgery and 250 mg for the second surgery. Therefore, it is unlikely that an overdose of local anesthetic caused toxic effects.

This case report has limitations. Although the complications were unexpected, they were initially regarded as temporary symptoms caused by the prolonged effect of local anesthetics used for epidural anesthesia, and the opportunity to perform systematic diagnostic examinations was therefore lost because the observation period was too long. There were therefore insufficient data from clinical tests to make an accurate diagnosis or to determine the mechanism responsible for the paralysis. Timely neurological examinations and MRI examinations of the relevant regions are necessary for precise diagnosis, and the current conclusion is thus purely speculative.

Although residual abnormal neurological function was regarded as a complication of epidural anesthesia at the first surgery, epidural anesthesia was again used to address postoperative pain at the second surgery. Despite the risk of postoperative pain, the second epidural anesthesia was contraindicated. If the second surgery had been conducted with general anesthesia alone, residual neurological dysfunction after the first surgery might have resolved; however, this is also speculative, as the mechanism of the neurological disorder remains unclear.

In conclusion, we presented a case of permanent bilateral lower limb paralysis after epidural anesthesia. Although causes for the onset and persistence of lower limb paralysis have been suggested, the underlying mechanism remains unclear. The presence of elevated levels of myelin basic protein in CSF and pathological spinal reflexes suggests that the lesion responsible for the bilateral lower limb paralysis was located in the CNS (thoracolumbar spinal cord or nerve roots), but no damage could be identified on diagnostic images, and the cause of paralysis could not be determined. The patient's clinical course suggested that epidural anesthesia was the

probable cause of the CNS damage, but we could not identify the mechanism responsible. Although ropivacaine and levobupivacaine toxicity was considered unlikely, the lack of other obvious causes of nerve damage after epidural anesthesia meant that this possibility could not be excluded.

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