

# Permanent Neurological Complication After Central Neuraxial Blockage: A Case Report

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## Citation

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## Abstract

Cauda equina syndrome after regional anesthesia is a serious and devastating complication. Its occurrence after epidural anesthesia is very rare. We reported a case in which regional anesthesia using combined spinal-epidural anesthesia set was associated with cauda equina syndrome postoperatively. We discussed the possible causes of this complication but believe that the etiology of cauda equina syndrome in this case remains unknown.

## INTRODUCTION

Cauda equina syndrome (CES) is a serious neurologic disorder that is caused by damage of the conus medullaris or the spinal nerve roots comprising cauda equina. It is associated with varying degrees of signs and symptoms including loss of bowel and bladder function, insensate perineal areas and lower extremity muscle weakness (1, 2). Permanent neurological complications like CES after central neuraxial blockades occur significantly more often after spinal blockage with especially intrathecal administration of lidocaine (3). However, severe neurological complication following epidural anesthesia is very rare (4, 5). We reported a case in which regional anesthesia using combined spinal-epidural anesthesia set was associated with cauda equina syndrome postoperatively.

## CASE REPORT

A 56 year old woman (76 kg, 155 cm, Body mass index: 31.63) was scheduled for bilateral knee replacement. Her medical history included an uneventful general anesthesia. The only medication she took was analgesic agents for knee pain. Besides the abnormal levels of glucose and activated partial thromboplastin time (APTT) (glucose: 133 mg/dL, and APTT: 52.1 sec) the physical examination and all laboratory studies were normal. The operation was planned under combined spinal epidural anesthesia after coagulation parameters were seen normal levels. No premedication was given before the operation. The patient was monitored with electrocardiography, pulse oxymetry, invasive blood pressure (radial artery), central venous (basilica vein) pressure and urine output. Consequently, she was given 10

mL/kg % 0.9 NaCl solution immediately prior to anesthetic for prehydration. With the patient in the sitting position, the lumbar area was disinfected with a solution 10% povidone-iodine (Poviodex; Kimpa İlaç Laboratuvarı ve Ticaret Lmt Şti, İstanbul, Türkiye). After removing excess moisture from the disinfected site, a 18-G Tuohy needle (Portex® Combined Spinal/Epidural Minipack with Lock Pencil Point Needle) was used to identify the L<sub>2</sub>- L<sub>3</sub> epidural space by using a loss of resistance to air technique via midline approach. A 27-G pencil point spinal needle was placed through the Tuohy needle into the subarachnoid space with the needle through needle technique. After flow of clear cerebrospinal fluid, patient received 10 mg 0.5 % isobaric bupivacaine and 25 µg fentanyl (2.5 mL) combination intrathecally. During intrathecal injection, the local anesthetic dropped from the locked place. The spinal needle knocked and locked again but dropping of local anesthetic continued. The spinal needle was removed and an epidural catheter was inserted 4 cm into the epidural space. After withdrawal of the Tuohy needle, the patient was placed supine position. The bladder was catheterized. After 15 minutes, there was no sensorial and motor blockage observed. Consequently a test dose was given using 2% lidocaine 2 mL, then, no spinal effect have been observed and 3 mL 2% lidocaine was given by epidural catheter. After 10 minutes the sensorial level was at the L<sub>1</sub> level which was thought to be inadequate for the operation. In addition, the mixture of 5 mL prilokain 2% and 5 mL plain bupivacaine 0.5% administered epidurally. The sensorial block level reached T<sub>10</sub> whereas Bromage score was 0. The operation was started and totally 10 mL 5% plain bupivacain was

given within two hours during the operation. The surgery lasted 4 hours. 1 unit erythrocyte suspension, 1800 mL colloid and 3050 mL crystalloid was given in the intraoperative period. The patient tolerated the procedure very well without complication. At the end of the surgery, the patient was transported to the postanesthesia care unit (PACU). After 90 minutes, sensorial block regressed to T<sub>12</sub>, and Bromage score was 0, then she was discharged from the PACU to the ward. Postoperative analgesia was maintained via patient controlled analgesia pump with epidural bupivacaine 0.125 % with fentanyl 2 µg/mL (bolus dose: 5 mL, lockout interval: 20 min). Low molecular weight heparin was given subcutaneously as a prophylaxis for thromboembolia.

After 3 days, urinary catheter was withdrawn and the patient started to complain of unable to micturate and urinary incontinence. The next day the patient complained of the difficulty in defecation with urinary incontinence. She had to take an enema in order to evacuate the bowels and was consulted by an anaesthesiologist. PCA was stopped and the epidural catheter was withdrawn. Total drug using via epidural catheter was 200 mL (250 mg bupivacaine). Neurological examination revealed loss of sensation under T<sub>11</sub> sensorial level on the left side and weakness of left lower extremity. Laboratorial findings were normal except white blood cell count (9.900/mm<sup>3</sup>) and minimal decrease in Ca<sup>++</sup> level (7.5 mmol/L). In the postoperative 5<sup>th</sup> day, difficulty in defecation and urinary incontinence were still remained while loss of sensation under L<sub>1</sub> sensorial level and hyperesthesia at right hypochondria and anaesthesia in S<sub>2-4</sub> dermatomes were obtained. During this period, the patient on several occasions expressed the sensation of urination and she was repeatedly unable to urinate and an urinary catheter had to be inserted each time. The voiding sistometry was seen as normal. After neurology and neurosurgery consultations, a lumbar magnetic resonance imaging (MRI) was performed and there were antelystesis at the level of L<sub>5</sub>-S<sub>1</sub>, discal protrusion and degeneration of facet joints at the level of L<sub>2</sub>-L<sub>3</sub>, wide discal protrusion at the level of L<sub>4</sub>-L<sub>5</sub> and subcutaneous oedema from T<sub>12</sub> to S<sub>1</sub>.

The patient received synacten 1 mg/day, intramuscularly, an initial dose of dexamethasone 4 mg, intravenously, followed by 4 mg intravenous dose every six hours for four days than the dose was reduced day by day and stopped at 8<sup>th</sup> day and vitamin B<sub>1+6</sub> was given every twelve hours. She was able to walk with help. On the 10<sup>th</sup> day after the operation, she was able to feel the miction and defecated spontaneously on the

11<sup>th</sup> day of the operation. An electromyogram (EMG) was performed on 12<sup>th</sup> day and there were the findings of early period associated with bilateral radiculopathy/plexopathy at the level of L<sub>5</sub>-S<sub>1</sub>. One month later, control EMG was performed and there were electrophysiologic influences at the level of L<sub>3-5</sub> and S<sub>1</sub>. She was discharged from hospital without improving clinical symptoms. One year after surgery, the patient was able to walk with help, although the gait was broad and slow; she was not able to run. She has regained sensation in perineum. The patient was able to urinate, but initiating the urination still required effort.

### DISCUSSION

The incidence of permanent or transient neurologic complications after central neuraxial blockade (CNB) is estimated between 1/1000 and 1/1000000 (6-8). Among neurologic complications, severe root and spinal complications are more frequent than cerebral complications (6). Cauda Equina syndrome was first described in four cases after continuous spinal anesthesia (CSA) in 1991 (9). Three of these cases were associated with the administration of delivery through a 28 gauge catheter specifically marketed for CSA; in the fourth case, 0.5% tetracaine was administered through a standard epidural catheter. Rigler et al (9) postulated that the combination of trauma, maldistribution and a relatively high dose of local anesthetic resulted in neurotoxic injury. Administration of hyperbaric local anesthetic through a sacrally directed catheter resulted in restricted distribution of anesthetic with a relatively high peak concentration, accumulated to sacral area and caused neurotoxicity. One year after than the first reported cases, American Food and Drug administration reported 8 more cases with similar etiology (3). Aria et al (2) composed experimental intrathecal model to explain the etiology of the damage. Their results suggest that local anesthetic neurotoxicity are closely associated with local anesthetic concentration (2, 3). In addition, case reports showed that not only the high concentration of lidocaine but also the dose of local anesthetic agent (relatively high dose of lidocaine >= 75 mg) is also important factor in CES. When compared with bupivacaine, the incidence of neurotoxicity is more often with lidocaine (1, 2, 10). However, Moen et al (7) reported that CES was observed in 32 patients of the 1260000 spinal blockades (SB). In the cases of SB, hyperbaric 5% lidocaine was used in eight cases, bupivacaine 5% in 11 cases (six hyperbaric and five isobaric) and in one case a mixture of both drugs was used. Spinal stenosis was most frequently found in these cases.

Kubina et al (11) described two cases of CES following bupivacaine with glucose injected spinally, and bupivacaine without glucose injected in a combined spinal-epidural technique. The first patient had spinal stenosis which could explain this complication; however the explanation for CES in the second patient is uncertain and consequently speculative (11). Similar to this case, Chabbou et al (12) reported that a case with CSE after spinal administration of 0.5% hyperbaric bupivacaine (12.5 mg) but she had no causative factor in the genesis of CES. Röhm et al (13) reported that the amount of aspirated cerebrospinal fluid (“Barbotage”) during spinal anesthesia procedure, seem to be important factor in persistent neurological impairment.

We did not use barbotage during spinal anesthesia (SA) and observe any sensorial and motor blockage after SA in our case, so we thought that SB was ineffective due to technical reasons. Cauda equina syndrome is a very rare and serious neurologic complication after epidural anesthesia. It is usually resulted from the subarachnoid administration of an intended epidural injection of local anesthetic agent. In our case, isobaric bupivacaine with prilocaine was given via epidural catheter and no spinal effect was observed. So, we believed that CES was not resulted from the subarachnoid administration of an intended epidural injection of local anesthetic mixture.

Kirihara et al (4) compared the neurotoxic effects of local anesthetics administered intrathecally or epidurally. They reported that histological damage and functional impairment in the nerve roots and spinal cord was less severe after epidural administration than after intrathecal administration with equipotent dose of local anesthetics. The pharmacokinetics of local anesthetics may differ when administered intrathecally or epidurally. Possible sites of action of local anesthetic administered epidurally include the nerve trunks in the paravertebral space, spinal nerves intradurally and the spinal cord. Epidural local anesthetic spreads to the dural sleeves, where the dura matter is thin with arachnoid proliferations and villi. Subsequently, the drug diffuses into the cerebrospinal fluid and causes nerve blocks on the nerve roots and on the spinal cord. Thus, local anesthetic in the cerebrospinal fluid should play a limited role in producing blocking effects after epidural administration. It was shown that intrathecal concentration of local anesthetic after epidural administration is lower than that after intrathecal administration. Could this lead to high concentrations of bupivacaine in the cerebrospinal fluid for a relatively long time, which resulted in neurotoxicity? It is

recommended that the total dose of bupivacaine should not exceed 2 mg/kg over a 2-hour period in order to prevent local anesthetic toxicity. In this case, the diffusion of local anesthetic to cerebrospinal fluid may lead to neurotoxicity, but the dose of bupivacaine administered epidurally (approximately 1 mg/kg/day) was not high that lead to neurotoxicity.

Other possible factors including trauma (14), ischemia (15), stretching of nerve roots in lithotomy position (16) and vascular pathology (15) should be considered in the etiology of CES. We have no clear explanation for development of CES in this patient. The intervention was performed with first attempt and no paresthesia and pain were observed either at the time of insertion of the epidural and spinal needles or during the insertion of the epidural catheter in our patient. In addition, EMG findings showed a bilateral nature supported that neurotoxicity rather than trauma. Even though, direct needle trauma rarely causes permanent or long term neurological sequelae, it is usually present as an existence of underlying predisposan radiculopathy (17). There was no evidence of spinal cord compression on MRI. Therefore, haematoma, intervertebral disc lesion and narrow spinal channel can be excluded as the causes of the neurological deficit in this patient. However, we don't know exactly whether these degenerative changes in the MRI detection contribute to development of CES in this patient. Another possible contributing factor might be a chemical damage but we carried out this manipulation with a single-use sterile needle and local anesthetic agent. Similarly, we used antiseptic solution for skin cleansing and this area was covered with a sterile wrap before the process. Further, there were no symptoms or signs of central nervous system infection so neurological injury in this patient was unlikely to have been contaminant induced.

Consequently, we could not explain the etiology of CES in our case. Lying in supine position for a long time after the operation in this patient who had degenerative changes on her columna vertebralis and took a local anesthetic solution for a relatively long time may lead to neurotoxicity.

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