Prolonged Bowel Dysfunction after Spinal Anesthesia

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Abstract: Spinal anesthesia is a relatively old technique but easy to perform. Serious neurological complications occur infrequently. However, we experienced a case in which prolonged bowel dysfunction occurred after a spinal anesthesia. One possible explanation is a direct neurotoxic effect of the local anesthetic resulting from intrathecal maldistribution.

Key words: Bowel dysfunction, complications, local anesthetic, spinal anesthesia

Spinal anesthesia is a relatively old technique but is easy to perform and provides good pain relief. Hypotension and post dural puncture headache are minor but common complications. Major complications include traumatic lumbar puncture, meningitis and cauda equina syndrome. These serious neurological complications occur infrequently\(^1\)\(^-\)\(^3\). This case report describes a patient in whom prolonged bowel dysfunction occurred after a spinal anesthesia.

CASE REPORT

A 61-year-old man was scheduled for a hemorrhoidectomy in 7 May 1992. His medical history included spinal canal stenosis, duodenal ulcer, and unstable angina. Previous surgery included an appendectomy, an L4–5 laminectomy, and a hemorrhoidectomy. These surgeries were performed at other hospitals under general anesthesia and without complications. Preoperative neurological assessment had not been performed for the previous surgeries. Lumbosacral spine films showed a small abnormal shadow in the anterior space of the S1 spine, which seemed to be the residue of the contrast medium used in the myelography performed more than ten years ago (Fig. 1-A, B). The patient was orally medicated with nifedipine, nicorandil, captopril, and nitroglycerin. Although he previously had a L4–5 laminectomy, he was scheduled for a hemorrhoidectomy under spinal anesthesia.

With the patient in a sitting position, a 23-gauge spinal needle was inserted by a paramedian approach at the L2–3 interspace, and a total of 4.8 mg of 0.24% dibucaine and 2.4 mg of 0.12% T-caine with 9.5% glucose (Neo-percamin \(^\text{®}\), Teikokukagaku Nagase, Osaka, Japan) was administered. No paresthesias were elicited during needle placement. Cerebrospinal fluid was clear. A satisfactory level of decreased sensation to pinprick to the L4 dermatome on the right side and the L2 dermatome on the left side resulted 15 minutes after the injection. Surgery was performed with the patient in the lithotomy position. The procedure (Milligan-Morgan method) lasted approximately 200 minutes and the intraoperative period was uneventful. The patient could move his left leg four hours after the injection, and his right leg eight hours later. On the following day, the patient began to have episodes of fecal incontinence. Neurological assessment confirmed a sensory
deficit to pinprick in the perineal region (S2-S3 dermatomes). The patient did not experience motor weakness of lower extremities or bladder dysfunction. Magnetic resonance imaging demonstrated spinal canal stenosis (Fig. 2-A, B). The patient's symptoms improved gradually after six months and he was able to control his bowel movements to some extents. However, since the patient disliked fecal incontinence, he had a colostomy under general anesthesia on August 1993.

**DISCUSSION**

Complications of spinal anesthesia may be classified as minor or major. Minor complications include hypotension and post dural puncture headache, and these occur commonly. Major complications include traumatic lumbar puncture, meningitis and cauda equina syndrome, but these severe neurological deficits are rare\(^1,2\). In the present case, the patient exhibited sensory deficit in the perineal region and fecal incontinence. There was neither motor weakness of the lower extremities nor bladder dysfunction. The patient experienced some degree of recovery six months postoperatively.

Neurological complications following spinal anesthesia can result from anesthesia, surgery, or exacerbation of preexisting disease. In the present case, the underlying cause is unlikely to be related to the surgical procedure performed. The potential causes of neurological injury associated with the anesthesia itself include trauma from lumbar puncture, infection, spinal cord ischemia, and neurotoxic reaction to the local anesthetic solution\(^2\). The patient did not experience paresthesias with the lumbar puncture, thus, it is unlikely that

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Fig. 1-A. (Anterior-posterior view).
Fig. 1-B. (Lateral view).
Lumbosacral spine films showed a small abnormal shadow at an anterior space of S1 spine. Arrows indicate the residue of myelography that had been performed more than ten years ago.
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Fig. 2-A. Lateral view of Magnetic resonance image.
Fig. 2-B. Transverse section of level 4.
Magnetic resonance imaging demonstrated spinal canal stenosis.

direct traumatic injury to the roots had occurred. The neurological findings were not consistent with the specific patterns of injury characteristic of spinal ischemia or infection.

Cauda equina syndrome has recently been reported in association with continuous spinal anesthesia. Spinal microcatheters can permit maldistribution of local anesthetics. The mechanism of spinal root injury may be related to both the high local concentrations and the duration of exposure to anesthetic. The present patient did not suffer from typical cauda equina syndrome, since he had no bladder dysfunction despite the fecal incontinence.

In the present case, the patient’s medical history included spinal canal stenosis and laminectomy. The lumbosacral spine film showed a small abnormal shadow in the anterior space of the S1 spine (Fig. 1). This shadow may have represented the contrast medium administered in myelography performed more than ten years ago. The presence of spinal canal stenosis could have led poor mixing of drugs. In addition, with the patient maintained in the sitting position after lumbar puncture, it is possible that Neopercamin S® might have accumulated in the sacral space, since it is a hyperbaric solution. These factors suggest that decreased mixing of the local anesthetic and intrathecal maldistribution might have occurred. The spread of local anesthetic might have been restricted and could thus have resulted in a toxic concentration.

Whether local anesthetics in clinical concentrations produce direct toxic effects on nerves is controversial. Recent reviews of local anesthetic tissue toxicity mention the rarity of nerve damage when local anesthetics are administered at the usual clinical concentration. In some animal models, the concentrations of various local anesthetics required to produce irreversible conduction blockade exceed the clinically administered concentrations of the agents. In contrast, in other animal studies, the potential neurotoxicity of clinical
concentrations of local anesthetics has been demonstrated\(^9\). Further, an in vitro experiments has shown the neurotoxicity of dibucaine and tetracaine in clinical concentrations\(^10\). In view of the above findings, we consider that poor mixing of the drug and a higher concentration than that clinically used could have led to the prolonged bowel dysfunction in this patient.

In summary, we presented a case in which the patient experienced prolonged bowel dysfunction after spinal anesthesia. One possible explanation for this occurrence may be a direct neurotoxic effect of the local anesthetic, resulting from intrathecal maldistribution.

**References**


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