Recurrent neurological symptoms in a patient following repeat combined spinal and epidural anaesthesia

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A healthy woman developed neurological symptoms after two consecutive Caesarean sections under combined spinal and epidural anaesthesia. Amethocaine was used for spinal anaesthesia and mepivacaine for epidural anaesthesia on both occasions, and a combination of fentanyl and bupivacaine was continuously infused for pain relief after the second. Her symptoms on both occasions were similar, including pain in the buttocks of 7–11 days duration and numbness in the sacral area of 5–6 months.

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Regional anaesthesia for Caesarean section is generally held to be inherently safe. Spinal and epidural blocks are therefore used widely, with the more recently introduced combined spinal and epidural technique gaining popularity.

Since Schneider and colleagues1 reported four patients in whom pain in the buttocks or lower extremities developed following recovery from spinal anaesthesia, it has been realized that such symptoms after uncomplicated spinal anaesthesia are not as rare as thought previously. However, the symptoms usually disappear within a week or two, and have been termed transient neurological symptoms (TNS). In addition, despite its name, pain is seldom accompanied by neurological deficits. In contrast, prolonged or persistent neurological symptoms, including cauda equina syndrome, have been associated with continuous spinal anaesthesia2 or intended epidural anaesthesia,3 and rarely with uneventful single shot spinal anaesthesia4 or combined spinal and epidural anaesthesia.5 Recently, we became aware of a patient who developed pain and persistent hypoaesthesia in the buttocks and legs after two consecutive Caesarean sections under uneventful combined spinal and epidural anaesthesia.

Case report

A 28-yr-old, woman (weight 70 kg, height 154 cm) was admitted at term for elective Caesarean section because of cephalopelvic disproportion. She had neither a remarkable medical history nor abnormal routine laboratory investigation. She was premedicated with atropine 0.5 mg i.m. With the patient in the left lateral decubitus position, combined spinal and epidural anaesthesia was performed using a double-needle, double-interspace method. The lumbar area was cleansed with an iodine-containing solution and wiped dry; then, approximately 3 ml of a 1% lidocaine solution was infiltrated at the L1–2 level and the L3–4 level for epidural anaesthesia and spinal anaesthesia, respectively. Using a sterile technique, an 18-gauge Tuohy needle was placed in the epidural space with the bevel directed cephalad via the midline approach using the loss of resistance to saline technique. An epidural catheter (Portex, Kent, UK) was advanced 5 cm into the epidural space, and aspirated to exclude intrathecal or i.v. placement. A 25-gauge Quincke needle was then introduced into the subarachnoid space at the first attempt without difficulty. After aspiration of approximately 0.2 ml of clear cerebrospinal fluid, 2 ml of 0.5% amethocaine in 10% glucose was injected over approximately 15 s. No paresthesias were elicited during needle insertion with either technique, catheter insertion, or intrathecal anaesthetic administration. The patient was then immediately turned to the supine position with left uterine displacement for surgery.

Since an upper dermatomal level of block for pinprick discrimination 20 min after the injection was T-10 bilat-
During both procedures, appropriate doses of amethocaine and pain after combined spinal and epidural anaesthesia. Throughout the intraoperative period. The epidural catheter was removed in the operating room and the immediate postoperative course was uneventful. Two days after surgery, the patient reported a sacral area of numbness and a moderate burning pain in the buttocks radiating to the dorsolateral sides of both thighs. There were no signs of bladder or bowel dysfunction. A diclofenac sodium suppository was administered to relieve pain. Although the painful sensation resolved within a week, the numbness continued for 5 months.

At age 31 yr, she was again admitted at term for her second Caesarean section because of the previous surgery. Preoperative neurological examination revealed no sensory-motor or muscle-tendon reflex abnormalities. Combined spinal and epidural anaesthesia was again performed using the same method. Intrathecally, 2 ml of 0.5% amethocaine in 10% glucose was injected over approximately 20 s, and a sensory block to pinprick was obtained at T-8 bilaterally. No clear fluid was aspirated from the epidural catheter before the injection. The surgical procedure was uneventful and lasted 30 min. An additional 10 ml of 1.5% mepivacaine was administered epidurally, immediately before the completion of surgery, and the continuous epidural infusion of 0.001% fentanyl and 0.2% bupivacaine was started thereafter at a rate of 2 ml h⁻¹ for 48 h postoperatively. Haemodynamic values remained stable throughout the intraoperative period.

Although the immediate postoperative course was uneventful, she again complained of numbness in the buttocks 12 h after surgery, when neurological examination revealed no motor disturbance but hypoaesthesia in the S3 dermatomal area. Next day, a tingling pain developed in the buttocks radiating to the dorsal area of the left thigh which became more intense after continuous epidural infusion was stopped. A diclofenac sodium suppository was administered to relieve pain, which lasted 11 days. The patient denied difficulty in voiding or defaecating. The numbness slowly decreased and disappeared 6 months later.

**Discussion**

We have presented a case of recurrent neurological deficits and pain after combined spinal and epidural anaesthesia. During both procedures, appropriate doses of amethocaine were administered for spinal anaesthesia and mepivacaine was used for epidural anaesthesia. A combination of bupivacaine and fentanyl, the doses of which were also reasonable, was continuously infused through the epidural catheter for postoperative pain relief after the second Caesarean section. Symptoms on both occasions were remarkably similar in nature, degree and duration. The pain was characterized as burning or tingling and lasted only 7 or 11 days, respectively. In contrast, hypoaesthesia in the sacral area was rather persistent, lasting 5 or 6 months.

The characteristics of the pain on both occasions were remarkably similar to those of previous cases of TNS after the administration of a single injection of lidocaine, mepivacaine, amethocaine, and bupivacaine. One common feature of all these cases and the findings of prospective studies were moderate-to-severe pain in the buttock, lower back, and/or legs that appeared 1–24 h postoperatively after complete recovery from uneventful spinal anaesthesia. The pain was often characterized as dull, aching, cramping, sharp, or radiating. In most cases, the symptoms resolved fully within 1 or 2 weeks and neurological examination was normal.

Persistent neurological symptoms were initially reported after continuous spinal anaesthesia or intended epidural anaesthesia. Maldistribution, repeated injection, and relatively large doses of local anaesthetics have been implicated. However, Beardsley and colleagues recently described them after uncomplicated spinal anaesthesia. As part of their clinical investigation concerning epidural and spinal anaesthesia, one volunteer developed a minor sensory deficit lasting for 3 months in the sacral area after a slow injection of 2 ml of 5% lidocaine. Subsequently, Gerancher reported cauda equina syndrome, which involved bladder and/or bowel dysfunction, after an uneventful lidocaine spinal. Additionally, Auroy and colleagues performed a prospective multicentre study, in which two out of 40 640 cases incurred permanent sensory deficits. These reports have suggested that even a recommended dose of local anaesthetic can cause neurological injury. Despite no direct evidence, many anaesthetists have proposed that persistent or permanent neurological symptoms are likely to result from a direct toxic effect of local anaesthetics.

In contrast, the aetiology of TNS has been controversial, since neurological deficits have rarely been associated with pain. For example, Freedman and colleagues demonstrated in their recent epidemiological study that 118 out of 1864 patients who had spinal anaesthesia developed TNS and, out of 118, only four patients reported neurological symptoms such as lower extremity weakness, numbness, and/or paraesthesia lasting less than 1 month. We previously showed in our prospective study that only one out of 11 patients who developed TNS after amethocaine spinal anaesthesia had numbness, which lasted for only 3 days.

Although our patient did not incur bladder or bowel dysfunction, the duration of numbness in the sacral area (>5 months) makes the symptoms worthy of note. The
similarity in location between pain and hypoaesthesia in our patient suggests that both TNS and neurological injury may share a common mechanism and that TNS may be related to a direct neurotoxic effect of local anaesthetic solutions.

Although the similarity in symptoms on the two occasions suggests that the repeated events may have resulted from the same mechanism, it is not clear which procedure was responsible for the symptoms; spinal anaesthesia, epidural anaesthesia, and/or postoperative epidural analgesia. It has been demonstrated that TNS can follow amethocaine spinal anaesthesia although the incidence is lower compared with lidocaine. In addition, Freedman and colleagues demonstrated in their clinical study that one patient developed TNS accompanied by numbness in the lower extremity after spinal amethocaine. Epidural anaesthesia alone has been associated with TNS, but not persistent neurological symptoms, as long as the catheter is inside the epidural space. In our patient, although the proper positioning of the epidural catheter was not confirmed on both occasions, the catheter was probably kept in the epidural space because there was no aspiration of cerebrospinal fluid and there was a complete and rapid motor recovery after surgery. Another possible explanation involves diffusion of the local anaesthetic from the epidural into the intrathecal space through the dural hole made by the 25-gauge spinal needle. Since a top-up with mepivacaine had to be given twice and once on the first and second occasions, respectively, the cauda equina incurred a relatively large exposure to mepivacaine. Finally, it is possible that postoperative epidural analgesia was responsible for the symptoms. However, the postoperative pain management described has been used in our institution without causing similar neurological symptoms or muscle weakness. Moreover, many other researchers have recommended using similar dosage of bupivacaine.

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