

Prevention strategy for post dural puncture headache

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Abstract : We report the anesthetic management of a parturient after an unintentional dural puncture while performing epidural anaesthesia for caesarean section and the strategy to prevent postdural puncture headache (PDPH). We injected the cerebrospinal fluid (CSF) back into the subarachnoid space and then administered intrathecal 1.5 mL 0.5% hyperbaric bupivacaine and fentanyl 20 µg to maintain CSF volume via epidural needle. The epidural catheter was inserted following re-identification of the epidural space for possible epidural top-up requirement and postoperative pain relief. After adding 3 mL of 0.5% isobaric bupivacaine via epidural catheter, sensory block level reached at T4 bilaterally. No PDPH was observed.

Key words : Epidural anaesthesia ; caesarean section ; prevention : dural puncture ; postdural puncture headache.

The postdural puncture headache (PDPH) due to accidental dural puncture is the most common major complication of epidural analgesia or anaesthesia and can be extremely disabling for the mother who is trying to nurse her newborn in the postnatal period. Therefore, prevention of accidental dural puncture is still important in teaching hospitals.

The incidence of epidural needle induced PDPH was reported to be between 76-85% according to the text book (4) but it has been recently reported as 6.6% by KUCZKOWSKI (8). In order to prevent this distressing complication, KUCZKOWSKI and BENUMOF (9) suggested to maintain cerebrospinal fluid (CSF) volume. We, describe the conduction of spinal anaesthesia and the strategy to prevent PDPH of a parturient complicated by an unintentional dural puncture with an 18-gauge epidural needle during epidural anesthesia performed by reviewing the literature.

CASE REPORT

A healthy 26-year-old primigravida of 38 weeks' gestation (69 kg and 167 cm) was admitted for caesarean section (C/S). Following discussion of the anaesthesia techniques with the patient,

she requested epidural anaesthesia for C/S. Heart rate, non-invasive blood pressure and peripheral oxygen saturation were monitored. Approximately 650 mL of Ringer's lactate solution was infused. Epidural anaesthesia was performed with an 18-gauge Tuohy needle with its bevel facing cephalad was advanced using loss of resistance technique including saline in the sitting position between L2-3 intervertebral space by midline approach. When loss of resistance to saline occurred approximately at a depth of 4.5 cm, sudden spontaneous backflow was noted by the senior anesthetist soon after the junior anesthetist stopped advancing the epidural needle with one hand simultaneously interrupting the continuous pressure on the attached loss of resistance syringe with the other hand. CSF in the loss of resistance syringe was reinjected and followed by intrathecal injection of 7.5 mg hyperbaric bupivacaine (1.5 mL of 0.5% heavy marcaine) with 20 µg fentanyl (0.4 mL in 50 µg/mL) before removing the Tuohy needle. Thereafter, epidural anaesthesia was performed in the first attempt between L3-4 intervertebral space and epidural catheter with side ports was placed and left in 3.5 cm by the senior anesthetist. Loss of pinprick sensation was found to be at T10 bilaterally. Administration of prepared test dose containing 3 mL lidocaine 2% and 5 µg/mL epinephrine revealed any signs of intravascular CSF injection. Following the test dose, an additional of 3 mL isobaric bupivacaine 0.5% via epidural catheter provided an upper sensory level at T4 bilaterally. Hemodynamic parameters (arterial blood pressure and heart rate) remained within normal clinical limits. The parturient had an uneventful C/S and a healthy female infant was delivered with Apgar scores of 9 and 9 at 1 and

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5 min, respectively. The patient was given a detailed explanation of the possibility of development of PDPH the morning after the procedure. No prophylaxis was undertaken and no other supportive therapy like caffeine or analgesics was initiated. When postoperative bilateral analgesia level decreased to T10, patient controlled epidural analgesia (PCEA) pump was set to deliver a continuous basal infusion of 4 mL/h with a lock out interval of 10 min and 6 mL of bolus on demand with a 4 h limit of 35 mL. The solution in the PCEA pump was prepared in 250 mL 0.9% saline with 0.125% bupivacaine and fentanyl 2 µg/mL. Postoperatively, the epidural catheter was removed after 48 h. Before removing the catheter, 10 mL of saline was injected. Total of 370 mL (21.75 mg bupivacaine and 740 µg fentanyl) solution was infused via PCEA. PDPH was not observed.

DISCUSSION

We have illustrated our anaesthesia management of an accidental dural puncture in a case requested to undergo C/S under epidural anaesthesia and prevention strategy of probable PDPH due to epidural needle insertion by maintaining CSF volume.

Although there have been accepted treatment modalities for PDPH like administration of theophylline, caffeine, sumatriptan and, epidural saline, dextran or autologous blood, maintaining CSF volume deserves consideration as a newer development in the prevention of PDPH (10). It has been demonstrated that the maintenance of CSF volume by injecting CSF 3-5 mL back into the subarachnoid space through the epidural needle for labor analgesia followed by accidental dural puncture prevented PDPH (7, 9). Subsequently, they inserted the epidural catheter into the subarachnoid space through the dural hole and initially administered 3-5 mL intrathecal saline (9). However, NORRIS and LEIGHTON (12) found that the incidence of PDPH after unintentional dural puncture was not significantly different in pregnant patients who had an intrathecal catheter followed by continuous spinal analgesia throughout labor and delivery than in those who received continuous lumbar epidural analgesia following re-identification of the epidural space.

In the present case report, we noted approximately 3 mL CSF in the loss of resistance syringe and we injected this amount of CSF back into the subarachnoid space in a manner similar to

KUCZKOWSKI and BENUMOF (9). We preferred to administer intrathecal bupivacaine and fentanyl (a total volume of 1.9 mL) to maintain CSF volume instead of 3-5 mL saline given by KUCZKOWSKI and BENUMOF (9). Although the intrathecal drug dose we selected was similar with JAIN *et al.*'s (6), we were unable to get an adequate anaesthesia level for C/S with 0.5% bupivacaine 7.5 mg with fentanyl 20 µg. The possible explanation for the inadequate anaesthesia could be the extra time elapsed during re-identification of epidural space for catheter placement leading caudal pooling of hyperbaric bupivacaine in the sitting position and the lowest spinal dose we used.

According in the latest survey of UK practice in the management of accidental dural puncture, the rate of re-siting the catheter (41%) is higher than epidural catheter placement intrathecally (28%) and the remaining consisting of both options (2). Although we have previously reported the successful management of accidental dural puncture and PDPH with continuous spinal analgesia (13), we preferred to re-site the epidural catheter.

The use of intrathecal catheter for continuous infusion of 0.0625% bupivacaine with fentanyl at a rate of 2 mL/h for maintenance of labor analgesia prevented PDPH in 14 out of 15 patients (7). Whereas, we used the epidural catheter that we inserted following re-identification of epidural space for possible top-up administration. After administration of 3 mL epidural test dose of 2% lidocaine, 3 mL 0.5% isobaric bupivacaine was injected before the onset of surgery and the upper sensory block level increased from T10 to T4 bilaterally. We planned to use epidural catheter for postoperative analgesia particularly as continuous basal infusion via PCEA for preventing continuous CSF leakage from the dural hole.

Contrary to the idea of maintaining CSF volume with small amount (3-5 mL), it has been reported that immediate injection of 10 mL intrathecal saline through the epidural needle after accidental dural puncture reduced the incidence of PDPH (3, 9). KUCZKOWSKI and BENUMOF (9) left the intrathecal catheter in situ for a total of 12-20 h whereas we removed the epidural catheter which was left in-situ for 48 h after injecting 10 mL saline as well. Therefore, injection of 10 mL epidural saline before catheter removal might have been a contributing factor in the absence of PDPH in this case report.

The underlying mechanism of the prevention of PDPH was speculated to be the short term plugging effect observed after immediate epidural

catheter insertion into subarachnoid space and long term plugging effect due to prolonged presence of intrathecal catheter (9, 10). In the present case report, there should be a different explanation for the absence of PDPH rather than plugging effect leading to inflammatory reaction promoting dural healing after intrathecal catheter removal. The most likely mechanism seems to be the subsequent prevention of CSF loss initially by re-injecting the CSF and the bupivacaine and fentanyl into subarachnoid space and then initiation of continuous basal infusion with PCEA resulting in decrease of further CSF leak.

AYAD *et al.* (1) reported an asymptomatic CSF leak 35 h after epidural catheter placement for labor without a known dural puncture. Therefore, not every patient necessarily gets headache and repeated epidural bolus top-ups might have contributed to the absence of PDPH in our case.

The effect of bolus and continuous epidural saline administrations in reducing the incidence and severity of PDPH after accidental dural puncture during attempted epidural needle placement has been extensively discussed by HARRINGTON (5). In contrast to use of bolus and continuous intrathecal analgesia in several case reports (9, 11, 13), we used continuous infusion and bolus on demand with PCEA to prevent PDPH. By doing this we produced positive pressure within the epidural space with the continuous basal infusion which could have probably prevented persistent CSF leakage and PDPH. However, the limitation of this hypothesis is the lack of measuring positive pressure created by the continuous infusion through PCEA in this case report.

Incidence of unintentional dural puncture complicating epidural anaesthesia changes according to the experience of the anaesthetist. Although this incidence is considered to be inversely related to experience, sleep deprivation, fatigue, and the effect of night work may have a role in the higher incidence of accidental dural puncture in junior personnel performing epidural analgesia (10). We previously reported a case of accidental dural puncture during epidural anaesthesia at cervicothoracic level for mastectomy which required relatively more experience than performing lumbar epidural analgesia even by a senior anaesthetist (13).

Therefore, the accidental dural puncture occurred during supervising a junior trainee anaesthetist is another important issue not only because of preventing PDPH but also the potential risk of

total spinal if unrecognized. However, in a case report of unrecognized accidental dural puncture followed by subarachnoid administration of 0.1% bupivacaine 15 mL containing fentanyl 2 µg/mL for labor analgesia by a junior anaesthetist did not cause a total spinal due to use of dilute solution and the punctual intervention of the senior anaesthetist (11).

In conclusion, we have shown that resiting epidural catheter in a case of recognized accidental dural puncture successfully prevented PDPH, though arguably intrathecal placement was a preferred option.

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