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CASE REPORT

Intracranial subdural hematoma after spinal anesthesia

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SUMMARY. Intracranial subdural hematoma is an exceptionally rare but life-threatening complication of spinal anesthesia. We report a case of intracranial subdural hematoma following spinal anesthesia for cesarean section in a 27-year-old woman. She developed a diffuse headache after surgery with a blood pressure of 220/140 mm Hg which was followed by generalized seizure activity. Her blood pressure remained high after medication with diazepam, nifedipine and magnesium sulfate. She remained unconscious with a Glasgow coma scale of 5. The cranial tomography revealed a subdural hematoma with diffuse cerebral edema and cerebral tentorial herniation. When a patient complains of postdural puncture headache and then has seizure activity, one should consider alternative diagnoses, including that of a subdural hematoma, and carry out a careful examination, including magnetic resonance imaging or computerized tomography scan.

Keywords: Anesthetic techniques, Subarachnoid; Anesthesia, Obstetric; Complications, Cranial subdural hematoma

INTRODUCTION

Intracranial subdural hematoma is an exceptionally rare complication of regional anesthesia and headache is generally the first symptom. Severe headache, in combination with pregnancy and dural puncture, has a broad differential diagnosis, including post dural puncture headache (PDPH), preeclampsia, migraine, drug-induced headache and intracranial pathologies.[1,2] We describe a case that illustrates the importance of careful ongoing assessment of symptoms of headache because the headache may be more than a PDPH and may herald a life-threatening intracranial problem.

CASE REPORT

A 27-year-old, 162-cm, 69-kg normotensive primigravida with a 40-week pregnancy presented to the labor and delivery unit in labor. After 7 h of labor, vaginal examination revealed 4-cm cervical dilatation, complete effacement, and vertex presentation. She had been receiving oxytocin augmentation for 2 h. The obstetricians decided on cesarean section for delivery of the baby because of failure to progress. There was no notable medical history before or during pregnancy. All laboratory data including coagulation status were within normal limits on admission.

After an 800-mL i.v. bolus of crystalloid solution, a spinal block was performed with a 22-gauge Quincke needle at L3-4 using the midline approach. At the first attempt, clear cerebrospinal fluid (CSF) was obtained, and was followed by slow injection of 0.5% hyperbaric bupivacaine 2.5 mL. After 5 min there was sensory block to T6 using pin-prick and the operation proceeded. The parturient's arterial pressure was maintained within 95–130/55–80 mm Hg without any hypotensive episodes and no vasopressor was used. The intraoperative heart rate was stable at 75–100 beats/min. A healthy, normal baby (3900 g, 53 cm) was delivered with Apgar scores 5 at 1 min and 9 at 5 min. Hartmann's solution (1000 mL) was given during the operation, to which oxytocin 10 units was added after delivery. During the operation the patient was alert and communicating with the anesthetist. Her vital signs were normal in the post-operative care unit and four hours after spinal insertion the block had totally worn off with the return of good motor function.

Forty-five minutes after the end of the operation she complained of pain; paracetamol 1 g was given orally

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and meperidine 0.5 mg/kg i.v. and she was transferred to the ward. Her postoperative course was initially uneventful. She had no evidence of preeclampsia but had two measurements of high blood pressure (135–140/90–100 mm Hg) believed to be due to pain. She then developed a severe diffuse headache 22 h after surgery when she was out of bed. She had had headache episodes earlier, but they were not as severe as on this occasion, when she ultimately complained to the nurse. Her systolic blood pressure was 220/140 mm Hg and her heart rate 82 beats/min at the onset of headache. The on-call obstetrician was informed. When, 5 min later, there was generalized seizure activity, the obstetrician was present and gave diazepam 10 mg i.v. after which the patient was transported to the obstetric high dependency care unit. Her blood pressure was 210/140 mm Hg after the diazepam; nifedipine one capsule was given sublingually. Another arterial pressure of 200/140 mm Hg was measured after 5 min, eclampsia was diagnosed and magnesium therapy started as per unit protocol (magnesium sulfate 3 g i.v. as a slow bolus followed by an infusion of 1 g hourly).

Since she remained unconscious and laboratory investigations did not support preeclampsia, after 20 min she was examined by a neurologist. Her Glasgow Coma Scale (GCS) was 5. Her left arm was in abnormal flexion and the right arm responded to deep pain with extension. Both her pupils were dilated and unreactive to light. The neurologist recommended cranial tomography (CT), but before the test could be performed she stopped breathing. The anesthesiologist began resuscitation and the trachea was intubated without any drugs. After intubation, the GCS was 3, diffuse rhonchi and rales were noted, her arterial pressure was 40/20 mm Hg and heart rate 96 beats/min. An infusion of Gelofusine 500 mL and dopamine 10 $\mu g \cdot kg^{-1} min^{-1}$ was started and she was transported for a CT scan.

The cranial CT revealed a subdural hematoma over the right temporoparietal region of 1.5 cm with diffuse cerebral edema, an intracranial shift and cerebral tentorial herniation (Fig. 1). Emergency surgery and evacuation of the hematoma by craniotomy were planned. During the operation her blood pressure was maintained at 80–120/40–60 mm Hg and whole blood, crystalloid, colloid, and fresh frozen plasma transfusions were given with supplementary dopamine infusion. The surgeons could not locate the main cause of the hematoma because of diffuse cerebral edema. In the postoperative period her GCS was 3 with no pupillary response to light. She was immediately taken to the intensive care unit intubated and brain edema treatment was started. Her follow-up CT scan at 48 h showed no progress in brain edema with cortical contusions, lacerations and ischemic changes. She died on the 7th postoperative day. We could not perform an autopsy since her family did not consent to it.

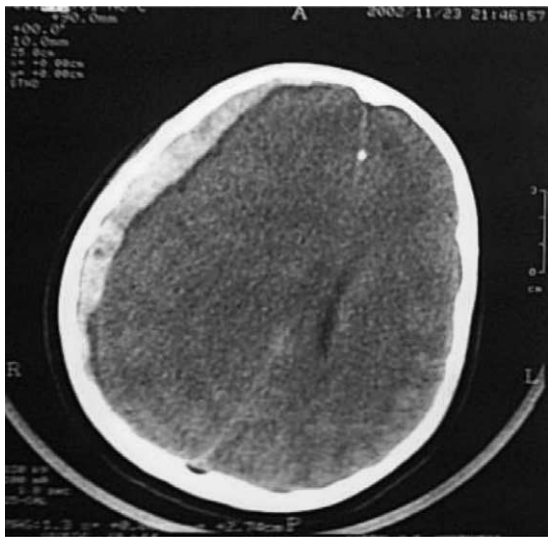

Fig. 1 CT scan before craniotomy, showing 1.5 cm right temporoparietal subdural hematoma and displacement of midline structures.

DISCUSSION

Subdural hematoma is a serious but rare complication of dural puncture. Cases have been reported following accidental dural puncture with an epidural needle and even after spinal anesthesia, myelography, discography, and diagnostic lumber puncture.[2,3] The needles used ranged in size from 16 to 25 gauge.[3–5] Scott and Hibbard[6] reported an incident rate of 1/500000.

Severe headache in combination with pregnancy and dural puncture has a broad differential diagnosis to include PDPH, preeclampsia, migraine, drug-induced headache and intracranial pathologies. The latter include hemorrhages, venous sinus thrombosis and post-partum cerebral angiopathy.[7] The difficulty in diagnosing a hematoma was evident as definitive diagnosis took between 8 and 42 days post partum.[8] Headache after dural puncture is most commonly ascribed to just a routine PDPH.[9]

The presumed mechanism for intracranial hematoma is the loss of CSF with low CSF pressure leading to traction and tearing of the intracranial subdural veins.[10,11] Excessive leakage of CSF through the dural puncture ($\geqslant$ 250 mL) may cause caudal displacement of intracranial structures, which may result in subdural hematoma formation.[12,13]

We used a 22-gauge Quincke needle which was all that was available. It is well known that the incidence and severity of PDPH are directly related to the size of the needle, and the nature of the tip. When using a