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Letter to the Editor

An extraordinary complication of spinal anesthesia: Brain edema

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Dear Editor,

Postspinal headache (PSH) is the most common iatrogenic complication of spinal anesthesia due to puncturing the duramater. Findings and symptoms of PSH result from the loss of cerebrospinal fluid (CSF), tension in the intracranial content, and reflex vasodilatation (1). The postural component should also be considered in the diagnosis of PSH. Other causes of headache can be ruled out with a carefully obtained medical history (2). A severe headache starting immediately after an invasive procedure or a prolonged headache for more than 7 days should remind the following potential causes including intracranial hemorrhage, meningitis, cerebral infarct, uncal herniation or an attack ofmigraine (3). In this letter, we present our experience in a patient who underwent a C-section under spinal anesthesia and subsequently developed cerebral edema postoperatively.

The 25-year-old patient was admitted to the hospital during her second pregnancy at term. The body weight of the patients was 117 kg and her ASA score was II. The patient was informed and her consent was obtained in a signed form before she underwent an elective C-section under spinal anesthesia. The findings of the preoperative examination were non-specific excluding the morbid obesity and anemia based on the laboratory results. She did not undergo a previous surgery before. Before the operation, a venous cannula was inserted and 0.9% NaCl at a dose of 10 ml/kg was administered intravenously. In the sitting position, 25G Quincke spinal needle was inserted between the L3-L4 vertebrae. After observing the clear CSF coming from the spinal needle, a 12.5 mg dose of hyperbaric bupivacaine was injected into the intrathecal space. Following the confirmation of block at the level of T6 dermatome by the pinprick test, the operation was initiated. During the operation, 1000 ml of 0.9% NaCl and 500 ml of modified liquid gelatin (Gelofucine®) were given to the patient. The operation ended uneventfully. In the postoperative care unit, the level of the resolving block and the hemodynamic parameters were monitored. Then, the patient was transferred to the inpatient unit under appropriate conditions, providing the necessary information to the healthcare team.

The patient was discharged from the hospital on the postoperative second day; however, she presented to the clinic again, this time with the complaints of head and neck pain on the postoperative 3rd day. The pain and nausea aggravated while the patient was standing up but the neurological examination was normal and there was no neck stiffness. The patient was diagnosed with a postspinal headache. Intravenous hydration was applied along with the administration of paracetamol, theophylline, and antiemetics. She was observed and monitored; then, bed rest, oral hydration, caffeine, and analgesics were recommended.

The headache was still persistent on the 7th postoperative day. The patient was re-evaluated and consulted with the neurology department. Neurologists identified cerebral edema on the CT images (Figure 1). The patient then was hospitalized and given dexamethasone 4x4 mg and furosemide 2x20 mg. On the next day, the patient's headache improved and she was discharged on the 3rd day of hospitalization.

The diagnosis of PSH is made based on a comprehensive physical and neurological examination and medical history. Meanwhile, clinical conditions such as spinal hematoma or abscess, septic or aseptic meningitis, intracranial mass or hemorrhage, aneurysm, thrombophlebitis, and drug-related neurotoxicity should be considered in the differential diagnosis (4). In the case of clinical suspicion, imaging tests should be carried out in patients with neurological findings or with characteristic features of

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PSH. The patient should be consulted to the neurology department if deemed necessary. This patient in this current case report presented to our clinic several times with similar complaints. Despite the treatment, the clinical signs and symptoms did not resolve. A neurology consultation was performed and the CT images revealed cerebral edema in the patient (Figure 1).

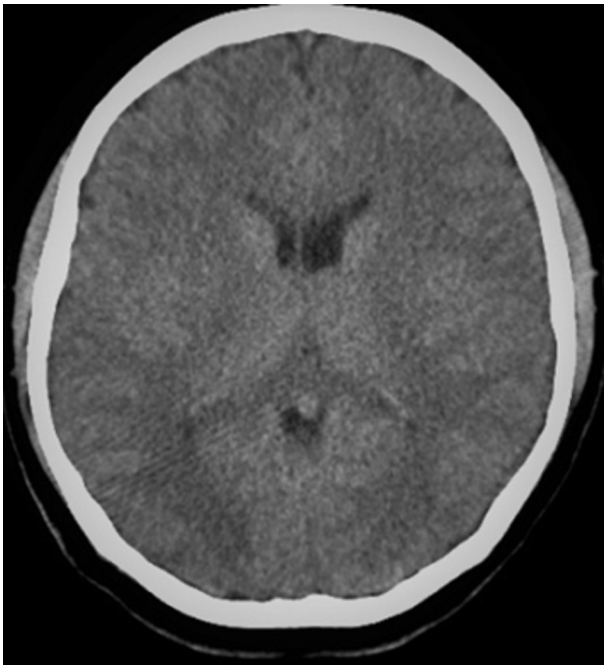

Figure 1. Cerebral CT (Postoperative first week)

As a consequence of endothelial injury and venous congestion after a puncture to the dura, tension may develop in the cerebral veins, potentially resulting in sinus venous thrombosis (SVT) or venous stasis. Furthermore, intracranial venous congestion and pushing during childbirth may cause endothelial injury in the vessels (5). Development of PSH has been associated with hypotension, a lowered CSF pressure, and CSF leakage in the literature. In addition, physiological effects such as vasodilatation and tension in the bridging veins suggest damage to the vascular endothelium, which in turn will be potentially related to a long-lasting PSH due to fluid leakage and brain edema developing indirectly.

In 70-80% of patients with cerebral vein thrombosis, headache can be seen, most likely accompanied with increased intracranial pressure symptoms such as papill edema, nausea, and vomiting. In deteriorating patients; cognitive dysfunction, convulsion, paresis, loss of sensation, and coma might be observed, respectively (5). SVT was ruled out in the differential diagnosis of our patient but it was considered that there might bevascular endothelial damage developing secondary to the delivery process.

Mannitol is an osmotic diuretic and it transfers the fluid in the neurons and glial cells to the extracellular space. Therefore, it is suggested that headache is relieved as a result of reduced brain weight and decreased strain on the meninges and bridging veins (6). In the currently presented case, not mannitol but furosemide was used as a diuretic. We believe that the tension on the meninges was relieved by transferring some of the intracellular fluid to the extracellular space. To the best of our knowledge, no reports are available in the literature informing that cerebral edema occurred after spinal anesthesia. We are of the opinion that this clinical picture developed in our patient due to endothelial injury. As it is observed in our patient, administration of diuretics is a basic treatment, which is reliable and associated with favorable tolerability. Therefore, with diuretic treatment, the clinical signs and symptoms can be resolved appropriately. Although it is rare, cerebral edema should be remembered in patients with persistent PSH and the patients may benefit from radiologic imaging.

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