



Subdural Hematoma Following Post-Dural Puncture Headache After Combined Spinal-Epidural Anesthesia for Cesarean Section: A Case Report

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Abstract

Aortic dissection (AD) is a life-threatening condition that can mimic acute myocardial infarction (MI), highlighting the importance of maintaining a broad differential diagnosis in acute chest pain. We present a case of a 43-year-old male with acute chest pain and electrocardiographic findings consistent with ST-segment elevation myocardial infarction (STEMI), including ST elevations in leads V1-V4. Despite elevated troponin levels, classical signs of AD—such as mediastinal widening on imaging and discrepancies in pulse or blood pressure—were absent. Coronary angiography revealed no significant coronary obstruction, but an immediate aortogram identified a dissecting flap at the root of the ascending aorta, just distal to the right coronary artery (RCA) ostium. The patient underwent emergent surgical repair with an uncomplicated postoperative course. This case highlights the critical need for vigilance in atypical presentations of MI and reinforces the importance of incorporating advanced imaging early in the diagnostic process. Prompt recognition of AD in such scenarios is vital, as delays can result in catastrophic outcomes. This report underscores the educational value of recognizing nuanced presentations and integrating multidisciplinary approaches to optimize patient care.

Keywords: Subdural hematoma; Post-dural puncture headache; Neuraxial anesthesia; Intracranial hypotension

Introduction

Post-dural puncture headache (PDPH) is a common complication of neuraxial anesthesia, typically presenting as a postural headache in the frontal and occipital regions, often accompanied by additional symptoms such as neck stiffness and tinnitus. While most cases resolve spontaneously within one week, persistent symptoms may require epidural blood patch (EBP) therapy [1]. Although PDPH is usually self-limiting, excessive cerebrospinal fluid (CSF) loss may, in rare cases, result in severe intracranial hypotension, increasing the risk of life-threatening complications such as subdural hematoma (SDH) [2]. We report a rare case of PDPH complicated by SDH following an unintentional dural puncture during anesthesia for a cesarean section. Fortunately, the patient's symptoms improved with conservative management, and no significant neurological sequelae were observed.

Case Presentation

The patient was a 43-year-old female, gravida 2, para 1, with a height of 152 cm and a weight of 57.5 kg. A previous cesarean section had been performed 10 years ago due to prolonged labor; she experienced no anesthesia-related complications. The patient was admitted for an emergency cesarean

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section due to a scarred uterus and irregular contractions. Preoperative laboratory studies revealed mild anemia (hemoglobin 10.9 g/dL) with normal platelet count and coagulation parameters. Her medical history was negative for lumbar spine disorders, cranial trauma, cerebrovascular malformations, or neurological disorders.

The patient was positioned in the right lateral decubitus position for epidural puncture. Under aseptic conditions, 3 mL of 1% lidocaine was administered for local infiltration anesthesia, followed by a midline approach epidural puncture at the L3-4 interspace using a 17G Touhy needle. The dura mater was inadvertently punctured, and clear cerebrospinal fluid (CSF) was observed. Subsequently, 15 mg of ropivacaine and 20 mcg of fentanyl were administered intrathecally through the epidural needle. The needle was then retracted into the subcutaneous tissue, and a second successful epidural puncture was performed at a depth of 4 cm. An epidural catheter was inserted, left 4 cm in place, and secured at 8 cm at the skin, with no aspiration of blood or CSF.

The sensory block level was determined to be at T4. The cesarean section proceeded without complications, resulting in the delivery of a female infant weighing 3030 g, with APGAR scores of 10/10 at both 5 and 10 minutes. The total intraoperative fluid intake was 1800 mL, with an estimated blood loss of 1200 mL and urine output of 350 mL. After the procedure, the patient and the obstetric team were informed of the unintentional dural puncture and the associated risk of PDPH.

On postoperative days one and two, the patient remained in a supine position without headache upon brief sitting or standing. However, she reported insomnia and anxiety on the first postoperative night. In response, she started taking pizotifen (0.5 mg at bedtime), which led to an improvement in sleep quality on the second postoperative night, although anxiety symptoms persisted. On day three, she developed a postural headache localized to the occipital, parietal, and bilateral shoulder regions, accompanied by a distending pain in both eyes. The headache was triggered within one minute of sitting or standing and was relieved upon lying down. PDPH was diagnosed, and an EBP was recommended, but the patient declined due to the concerns and anxiety about the procedure. Conservative management including increased fluid intake, non-steroidal anti-inflammatory drugs (NSAIDs) and bed rest was initiated. Psychological factors such as postpartum anxiety may have influenced her pain perception.

On day five, the patient developed a non-postural pulsating headache in the parietal region, distinct from the previous postural headache that was accompanied by elevated blood pressure (160-165/70 mmHg), and resolved after antihypertensive treatment. Given her perioperative risk factors for deep vein thrombosis (DVT), including significant

blood loss, prolonged surgery, and immobility, enoxaparin (4000 units subcutaneously) was initiated on postoperative day two and continued until day five. The coagulation test results were normal prior to anticoagulation initiation.

On day six, despite partial relief of her headache with NSAIDs and antihypertensive therapy, the non-postural headache persisted. The patient was referred to neurology for consultation due to a history of unintentional dural puncture and progressive headache characteristics. Although anxiety and insomnia were considered potential contributors to symptom persistence, the possibility of an intracranial complication could not be excluded. Due to the absence of focal neurological deficits and partial symptom improvement, MRI was deferred initially; the patient was placed under strict clinical observation. It was emphasized that urgent neuroimaging should be performed if symptoms worsened or new neurological signs emerged.

On day nine, the patient experienced a sudden, severe, non-postural headache with elevated blood pressure, but without nausea or vomiting. An urgent MRI was performed to evaluate for intracranial complications, revealing a subdural effusion in the left frontoparietal region with an associated hematoma measuring 5 mm (Figure 1). Given the small size of the hematoma and the absence of focal neurological deficits, the consulting neurosurgeon recommended conservative management, close monitoring, and regular imaging follow-up.

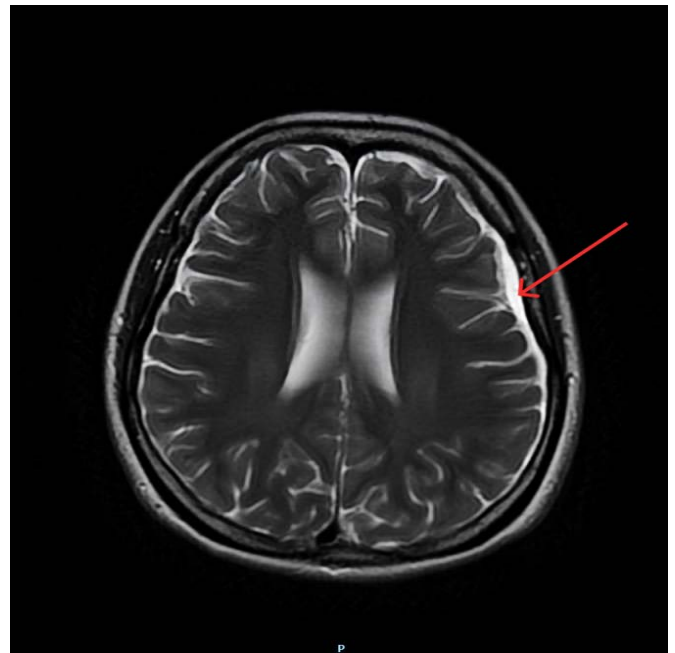


Figure 1: MRI on postoperative day nine revealed a 5 mm subdural hematoma with an associated effusion in the left frontoparietal region (red arrow).



Figure 2: MRI on postoperative day seventeen revealed mild thickening of the left frontoparietal dura mater and significant resorption of the subdural hematoma (red arrow).

By day seventeen, the patient was clinically stable. A follow-up MRI demonstrated mild thickening of the left front parietal dura mater, with significant resorption of the subdural hematoma (Figure 2). At both one-month and four-month follow-ups, the patient did not experience a non-postural pulsating headache in the parietal region and exhibited no neurological deficits.

Discussion

PDPH is a common complication of neuraxial anesthesia. The reported incidence of unintentional dural puncture during obstetric anesthesia ranges from 0.51% to 1.5%, with 50%–80% of these cases progressing to PDPH [3,4]. SDH, though rare, is a potentially life-threatening complication that has been documented in case reports. The incidence of PDPH and SDH in parturients is 309 per 100,000 and 1.5 per 100,000, respectively, with the risk of SDH in PDPH patients being significantly higher at 147 per 100,000 [5].

The pathogenesis of PDPH and SDH is closely related. CSF leakage through a dural puncture site creates a negative intracranial pressure gradient, leading to brain sagging and traction on pain-sensitive intracranial structures and bridging veins, ultimately causing headache. In some cases, traction of bridging veins may cause venous rupture, leading to hematoma formation and increased intracranial pressure [6].

Several factors contribute to the risk of PDPH, including younger age, female gender, a history of headaches (chronic, contemporaneous, or prior PDPH), needle type and size, bevel orientation, number of puncture attempts, operator

experience, and patient positioning [7]. Risk factors for SDH include excessive CSF leakage (due to multiple dural punctures or large needle use), pregnancy, coagulopathy, cerebral aneurysms, arteriovenous malformations, tumors, and cerebral hypotension [8]. Notably, large-gauge needles and multiple dural punctures increase the risk of both PDPH and SDH.

PDPH typically presents as a postural headache in the frontal and occipital regions, often radiating to the neck and shoulders. It occurs within 72 hours after dural puncture, and usually resolves within one week with conservative management or EBP [1]. Acute SDH, on the other hand, may present with headache, altered consciousness, or focal neurological symptoms, while chronic SDH can initially mimic PDPH but later evolves into a persistent non-postural headache with new neurological manifestations [2]. Both conditions may also be accompanied by dizziness, nausea, vomiting, sensory disturbances, and diplopia [1,2].

The diagnosis of PDPH is primarily clinical, based on a history of dural puncture and characteristic headache presentation. Other potential causes, such as analgesic or caffeine withdrawal, preeclampsia, intracranial hypertension, cerebral venous sinus thrombosis, brain tumors, and ischemic or hemorrhagic postpartum headaches, must be excluded [9]. The diagnosis of SDH requires neuroimaging. When a patient with a history of neuraxial anesthesia develops severe, persistent headaches or a transition from postural to non-postural headache, intracranial complications should be suspected, warranting brain CT or MRI with neurosurgical consultation. Acute SDH typically appears as a crescent-shaped hyperdense lesion on CT, whereas contrast-enhanced CT or MRI is more sensitive for detecting bilateral hematomas [10].

The choice of treatment strategies for PDPH is determined by the severity of symptoms. Mild cases can be managed with bed rest, hydration, and symptomatic treatment, while moderate to severe cases often require interventions such as EBP. SDH treatment is determined by hematoma size and associated symptoms. Surgical evacuation is indicated for hematomas exceeding 10 mm in thickness or those causing significant midline shift (>5 mm). Smaller, asymptomatic hematomas can be managed conservatively with close monitoring [11].

In this case, unintentional dural puncture with a 17G epidural needle led to significant CSF leakage, resulting in PDPH and subsequent SDH. The patient's refusal to undergo EBP and the administration of enoxaparin for DVT prophylaxis may have contributed to the development of SDH. The delayed recognition of SDH underscores the importance of timely neuroimaging in patients with evolving headache characteristics.

Conclusion

This case highlights the need for vigilance in PDPH patients, as it can rarely progress to SDH. Clinicians should maintain a low threshold for neuroimaging in patients with persistent, worsening, or non-postural headaches after dural puncture, even without focal neurological deficits.

Patients declining an EBP should receive conservative treatment to reduce the risk of intracranial hypotension-related complications. A multidisciplinary approach involving anesthesiologists, neurologists, and neurosurgeons is crucial for timely diagnosis and intervention. Prompt neuroimaging is warranted when headache characteristics change, particularly if a postural headache transitions to a non-postural headache or new neurological symptoms arise. Timely recognition and intervention are critical to optimizing patient outcomes and preventing life-threatening complications.

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Conflicts of interest: None.

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