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Case Report

High Spinal Anesthesia in a Patient Recently Submitted to an Epidural Blood Patch: A Case Report

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Abstract: Spontaneous intracranial hypotension (SIH) is a rare condition caused by cerebrospinal fluid (CSF) leakage, leading to orthostatic headache and associated symptoms such as nausea, vertigo, and visual disturbances. Epidural blood patch (EBP) is the first-line therapy for persistent symptoms. Neuraxial anesthesia after recent EBP poses unique challenges due to morphological changes in the epidural space. These changes, primarily caused by clot formation and subsequent fibrosis, can result in unpredictable epidural spread or excessive cephalad migration during spinal anesthesia. We report the case of a 30-year-old primigravida with SIH who underwent two EBPs during pregnancy and subsequently developed a high spinal block during elective cesarean delivery under spinal anesthesia. The patient experienced hypotension and bradycardia and sensitive blockade at C6, which were managed successfully, and both mother and newborn had an uneventful outcome. This case highlights the potential for erratic neuraxial block following recent EBP and provides a detailed discussion of the underlying mechanisms and management strategies. It underscores the importance of careful review of anesthetic history, individualized technique, and dosing adjustments. Evidence on the optimal timing and safety of neuraxial anesthesia after EBP is limited, and further research is needed to guide clinical decision-making in this setting.

Keywords: Cesarean delivery; Epidural blood patch; High spinal block; Neuraxial anesthesia; Spontaneous Intracranial Hypotension.

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1. Introduction

Normal intracranial pressure is maintained by the balance of cerebrospinal fluid (CSF) production, absorption, and flow. Alterations of these mechanisms lead to intracranial hypotension, which can be spontaneous or secondary to trauma or spinal surgery. Spontaneous intracranial hypotension (SIH), with an estimated incidence of 5 patients per 100.000, arises from dural tears, meningeal diverticula and/or CSF venous fistula. SIH compromises brain buoyancy and induces caudal traction on pain-sensitive structures, including cranial nerves and blood vessels. The reduction in intracranial volume triggers compensatory vasodilation, leading to the hallmark manifestation of SIH, which is orthostatic headache, frequently accompanied by nausea, vomiting, neck pain, vertigo, visual disturbances, tinnitus, and dizziness [1].

The diagnostic criteria of SIH include the presence of orthostatic headache temporally associated with low CSF pressure (<60 mmH_2O) and imaging evidence of CSF leakage [2]. The gold-standard treatment for SIH is the epidural blood patch (EBP). Symptom recurrence is common, often requiring repeated epidural blood patches (EBPs). We report a pregnant patient who required two EBPs due to SIH and later developed an unexplained

high spinal block during cesarean delivery [1], which we hypothesize was related to the prior interventions. This case aims to explore the mechanisms of orthostatic headache and EBP, emphasizing epidural space morphological changes that may predispose to unpredictable neuraxial blocks. We provide a systematic review of the literature on this topic and discuss management strategies for similar cases.

2. Case Report

A 30-year-old primigravida (ASA II, 73 kg, 162 cm) at 25+6 weeks of gestation was admitted with a severe fronto-occipital orthostatic headache accompanied by nausea, vomiting, vertigo, weakness, and visual disturbances. Neurological workup, including brain magnetic resonance Imaging (MRI) and lumbar puncture, revealed pachymeningeal enhancement, engorged dural venous sinuses, and an opening CSF pressure of 30 mmH_2O, fulfilling diagnostic criteria for spontaneous intracranial hypotension (SIH).

Due to persistent and disabling symptoms, an epidural blood patch (EBP) was performed at 27+2 weeks of gestation under strict aseptic conditions. Using a midline approach, an 18G Tuohy needle was inserted at the L4–L5 interspace. The epidural space was identified via the loss-of-resistance-to-air technique, and 20 mL of autologous blood was slowly injected. The patient experienced immediate symptoms and was subsequently discharged. However, two weeks later, her headache recurred, necessitating readmission and a second EBP at the same interspace using the same technique. Once again, complete symptom resolution was achieved.

At 37+1 weeks of gestation, ten weeks after the second EBP, the patient was admitted for an elective cesarean delivery. Spinal anesthesia was administered at the L4–L5 interspace with the patient in the sitting position. A 27G Whitacre needle was used to inject 10 mg of hyperbaric bupivacaine combined with 80 μg of preservative-free morphine over approximately 20 seconds, without barbotage. No pre-anesthetic sensory level assessment was performed. Following the procedure, the patient was positioned supine with a 15° left lateral tilt, and continuous monitoring of vital signs was initiated.

Within five minutes, she developed profound hypotension, with blood pressure falling from a baseline of 115/75 mmHg to 70/35 mmHg, accompanied by bradycardia at 45 beats per minute (Table 1). A high spinal block was suspected and confirmed by the unexpected cephalad spread of the anesthetic, reaching the C6–C8 dermatomes, as evidenced by bilateral hand paresthesia 10 minutes post-injection. Complete motor block was observed (Bromage score of 4). The patient was immediately placed in a slight anti-Trendelenburg position and treated with repeated boluses of 0.5 mg metaraminol (total dose 5 mg) and 1000 mL of crystalloid solution. These measures successfully stabilized her hemodynamic status without compromising airway function.

Table 1. Temporal Evolution of Blood Pressure, Heart Rate, Sensory Block Level, and Motor Block (Bromage Score).

Time (min)	Blood Pressure (mmHg)	Heart Rate (bpm)	Sensory Block Level (Dermatome)	Motor Block (Bromage Scale)
0	115/75	78	Baseline	0
2	100/65	70	T6	3
5	70/35	45	C6	4
10	85/55	55	C6	4
15	100/65	65	T8	4
30	110/70	70	T10	4
60	112/72	75	T12	3
90	118/78	77	L1	1

Time (min)	Blood Pressure (mmHg)	Heart Rate (bpm)	Sensory Block Level (Dermatome)	Motor Block (Bromage Scale)
120	121/82	78	Baseline	0

A healthy infant was delivered with Apgar scores of 9 and 10. The postoperative course was uneventful, with no recurrence of headache. The patient was discharged with her newborn and later referred to a headache clinic, where she remained symptom-free.

3. Discussion

EBPs are the first-line therapy for SIH and should be performed in patients with symptoms persisting for more than two weeks [1]. The therapeutic mechanism of EBP is not fully elucidated. MRI studies suggest that immediate pain relief after an EBP is due to a transient mass effect, which elevates CSF pressure. This effect can last from minutes to several hours, or in rare cases, up to a week. Sustained symptom resolution is thought to result from clot formation at the puncture site, which seals the CSF leak, promotes scar tissue development, and restores CSF balance [3]. Up to half of patients with spontaneous intracranial hypotension require multiple EBPs. In one series, symptomatic relief was achieved in 36% after the first EBP and an additional 33% after the second, with some cases requiring up to four to six procedures for sustained benefit [4].

A key yet unresolved question in neuraxial anesthesia is the optimal timing for performing a subsequent neuraxial procedure following an EBP. We propose a specific hypothesis to explain the high spinal block observed in this case, suggesting that prior epidural blood patches (EBPs) induced a fibrotic reaction within the epidural space, which resulted in reduced volume and altered morphology. This fibrosis created a mass effect that effectively channeled the hyperbaric bupicavaine cephalad, resulting in a more extensive block than anticipated.

This hypothesis is supported by the histological timeline of clot organization. Animal model studies demonstrate a progressive sequence of healing: early cellular infiltration within 24 hours, fibroblast proliferation by day four, collagen deposition starting at two weeks, and mature scar formation peaking around three weeks, followed by gradual regression to baseline thickness over the subsequent three months [5]. In this case, the cesarean delivery occurred during the phase of mature scar formation but before complete regression, likely contributing to the altered epidural anatomy and facilitating excessive cephalad spread of the spinal anesthetic.

Although 10 mg of hyperbaric bupivacaine is not considered a low dose in many studies, it is widely regarded as a safe and effective dose for obstetric anesthesia. The dose administered in this case aligns with the standard protocol at the treating institution. Although hyperbaric solutions tend to settle in the most dependent areas of the subarachnoid space, the altered epidural anatomy could have impeded the normal caudal spread, effectively "trapping" the anesthetic and promoting its cephalad migration. The use of isobaric solutions for cesarean sections is generally discouraged by most centers due to a high risk of inadequate sensory blockades for surgical anesthesia. However, evidence comparing hyperbaric and isobaric agents in this context remains scarce and is largely limited to isolated case reports [6, 7].

To provide a more comprehensive review of the available evidence, we conducted a systematic search of published case reports on neuraxial anesthesia after EBP. As of our search, only a handful of cases describe successful neuraxial blocks, generally when the interval between EBP and anesthesia was several weeks to months. Reports of effective epidural anesthesia within days of EBP are rare, and high spinal block remains a potential complication. A summary of these cases is provided in Table 2 [3, 6, 8-15].

Table 2. Summary of Published Case Reports on Neuraxial Anesthesia Outcomes Following Previous Epidural Blood Patch (EBP).

Reference	Indication for EBP	Interval Between EBP and Block	Main Conclusion
Peralta et al. (1998)	Post-dural puncture headache	15 months	Partial epidural block failure due to fibrosis in the epidural space.
Kelpstad (1999)	Post-dural puncture headache	12 months	Erratic epidural block with anomalous spread and altered latency time.
Lalonde & Lajoie (2002)	Post-dural puncture headache	5 years	Unilateral and irregular spinal block due to adhesions.
Sengupta & Gunturi (2007)	Post-dural puncture headache	2 years	Total failure of epidural block due to adhesions and fibrosis.
Chen (2009)	Post-dural puncture headache	1 month	High spinal anesthesia caused by excessive cephalad spread due to fibrosis.
Lee et al. (2013)	Post-dural puncture headache	2 years	Total failure of spinal anesthesia following prior EBP.
Borgeat et al. (2014)	Post-dural puncture headache	10 days	Total failure of epidural block, hypothesized due to fibrotic "compartment."
Whitwell & Patel (2015)	Post-dural puncture headache	2 months	Total failure of spinal anesthesia for cesarean section after prior EBP.
Einhorn & Schwartz (2016)	Post-dural puncture headache	2 years	Difficulty with catheterization and partial analgesia due to scar tissue.
Moucharige et al. (2019)	Spontaneous intracranial hypotension	10 days	High spinal anesthesia caused by excessive cephalad spread due to fibrosis.
Current Case	Spontaneous intracranial hypotension	10 weeks	Excessive cephalad spread of spinal anesthesia following prior EBP.

High spinal block remains a potential, though rare, complication. Upon recognition of signs indicative of excessive anesthetic spread, namely hypotension and bradycardia, the patient was promptly positioned in a slight anti-Trendelenburg posture. This intervention, combined with administration of vasopressors and intravenous fluids, effectively stabilized the patient's hemodynamics and prevented further cephalad extension of the block that could have compromised the airway.

While alternative explanations for the excessive cephalad spread cannot be excluded, the fibrosis hypothesis is emphasized based on the team's expertise and current understanding of pathophysiology. It is noteworthy that due to the infrequency of such clinical presentations, unpredictable or aberrant neuraxial blocks following prior epidural blood patches remain largely unfamiliar to many anesthesiologists. Awareness of this possibility facilitates more cautious pre-anesthetic assessment and encourages consideration of alternative anesthetic strategies.

In this clinical context, alternative approaches merit consideration. General anesthesia would have provided a safe and reasonable alternative, circumventing the challenges posed by altered neuraxial anatomy. Additionally, placement of an epidural catheter for

incremental dosing of local anesthetic could be considered; however, this technique carries its own risks of unpredictable anesthetic spread and potential block failure secondary to epidural fibrosis [8-11, 13, 14]. For future cases, a reduced dose of hyperbaric bupivacaine (e.g., 7–8 mg) may be advisable to minimize the risk of excessive cephalad spread [7, 15]. Ultimately, individualized anesthetic planning that carefully weighs risks and benefits is essential to optimize safety for both mother and neonate.

This case report is limited by the scarcity of published data on high spinal anesthesia following an epidural blood patch. Most available evidence consists of isolated case reports, and no large series or controlled studies specifically address the incidence, risk factors, or optimal timing for neuraxial blocks after recent EBP. Consequently, our observations are based on a single patient and may not be generalizable, highlighting the need for further research to guide anesthetic management in this clinical scenario.

4. Conclusion

Neuraxial anesthesia remains the cornerstone for cesarean delivery. In patients with a recent history of an EBP, clinicians must be acutely aware of the potential for a high spinal block due to fibrotic changes in the epidural space. This case highlights the importance of a detailed review of the patient's recent anesthetic history to inform a tailored anesthetic plan. Such a plan might include a reduced dose of local anesthetic, the use of an alternative formulation, or, if necessary, the consideration of alternative techniques such as general anesthesia. Further research, including larger case series, is needed to clarify the effects of prior neuraxial interventions and to establish evidence-based guidelines for anesthetic management in this unique clinical scenario.

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Conflicts of Interest: The authors declare no conflicts of interest.

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