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CASE REPORT

Antenatal blood patch in a pregnant woman with spontaneous intracranial hypotension

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ABSTRACT

Spontaneous intracranial hypotension is a condition that presents with postural headaches similar to those caused by accidental dural puncture. The diagnosis is based on clinical presentation, cerebrospinal fluid evaluation and magnetic resonance imaging scanning. We present a case of spontaneous intracranial hypotension with typical clinical and magnetic resonance imaging findings in a pregnant patient who was treated with an epidural blood patch. The blood patch, performed at 32 weeks of gestation, produced transient improvement in symptoms but failed to completely cure the headache, which worsened over the next few days. Symptoms resolved over the subsequent three weeks with conservative therapy.

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Introduction

In 1938, spontaneous intracranial hypotension (SIH) was initially described by Schaltenbrand as a spontaneous postural headache frequently combined with nausea and vomiting, neck stiffness, tinnitus and vertigo.¹ The pathophysiology and symptoms are identical to those of post dural puncture headache (PDPH) as they are also caused by cerebrospinal fluid (CSF) leakage, although it may be difficult or sometimes impossible to identify the site of leak. It is considered most likely to result from rupture of a spinal perineural cyst. The incidence of SIH is 1 in 50 000,² and the condition affects women more frequently than men.³ Many patients improve spontaneously, with or without conservative treatment, and some benefit from epidural blood patch (EBP).^{4,5} Surgical repair is seldom required. To our knowledge there are no published reports of treatment of SIH with EBP in pregnancy.

Case report

A 33-year-old Caucasian woman, (G2, P1) presented to our antenatal clinic at 32 weeks of gestation with a two-

week history of severe postural headache with associated symptoms of mild photophobia, nausea and vomiting, making her unable to tolerate food or fluids. She had no recent history of severe headache, lumbar puncture, head trauma or other relevant medical problems. Her first child had been delivered six years earlier by elective caesarean section for breech presentation, with surgery performed under uneventful spinal anaesthesia.

On examination she was 170 cm tall and weighed 54 kg (body mass index (BMI) 18.7 kg/m²). She appeared dehydrated and pale, with no neurological deficit; her urine dipstick revealed ketonuria. Her blood pressure was 110/66 mmHg and her pulse 88 beats/min. Full blood count, liver and renal function tests were all normal. She was admitted to the antenatal ward for re-hydration and analgesia. A neurological opinion was requested for the unusual presentation of headaches and spontaneous intracranial hypotension was diagnosed on clinical grounds. A magnetic resonance imaging (MRI) scan was requested to exclude other possible causes of headache such as sagittal sinus thrombosis.

Intracranial and spinal MRI confirmed the diagnosis of SIH (Figs. 1 and 2); thickening of the meninges with some extra-cerebral and extra-spinal fluid collections could be clearly identified. At the patient's request, she was given conservative management including bed rest, oral codeine, paracetamol and i.v. and oral fluids.

When her symptoms did not improve over the following week, EBP was offered. A consultant anaesthetist

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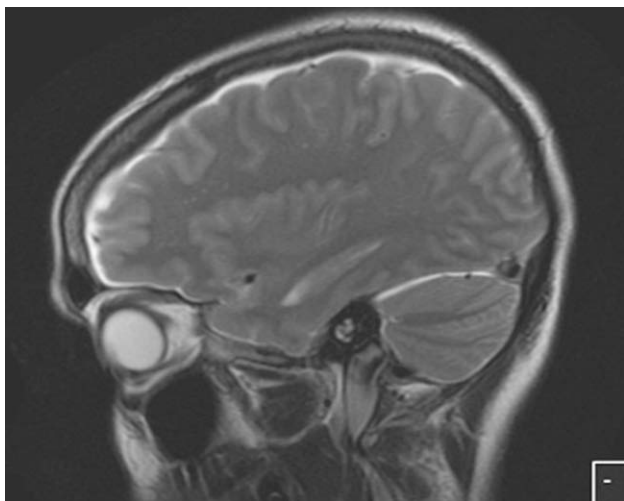


Fig. 1 MRI scan of the brain showing thickening of meninges with some extracerebral fluid collections.

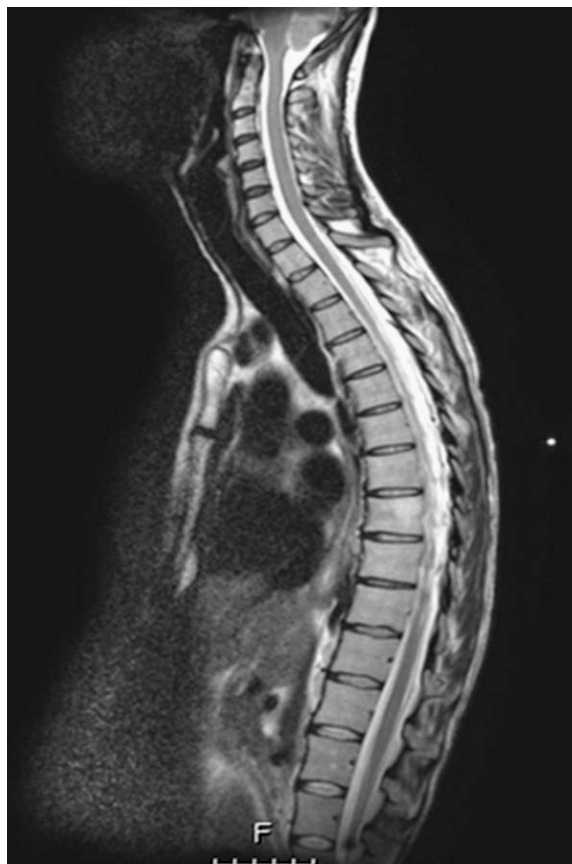


Fig. 2 MRI scan of the spine. No overt CSF leak can be seen in the epidural space.

discussed the procedure in detail with the patient outlining the potential benefits and possible complications such as failure, accidental dural puncture and worsening of symptoms, infection and temporary or permanent

nerve damage. On the following day EBP was performed in the obstetric theatre without complication. The epidural space was identified using loss of resistance to saline and 20 mL of autologous blood were injected under strict aseptic conditions. The injection was stopped when the patient experienced increasing pressure in the lumbar region. There was no immediate improvement of her headache and she was subsequently transferred to the recovery area where she was monitored for 2 h.

The patient was told to lie flat for the next 12 h and after a period of nearly 16 h she noted marked improvement of her symptoms. When she mobilised for the first time after the EBP, her headache had almost completely resolved, enabling her to be discharged home later that day. Unfortunately her symptoms reappeared gradually over the following days and were as disabling as before. The option of a second EBP was discussed but she declined. Conservative management was continued and over the next three weeks her symptoms gradually improved and when she attended the antenatal clinic at 36 weeks she was free from symptoms. Despite her previous caesarean section and the potential risk of scar rupture she was keen to try for vaginal delivery. At 41 weeks and five days she presented to the delivery suite with painful contractions. After 12 h of active labour without epidural analgesia, she gave birth to a male infant weighing 4.02 kg with Apgar scores of 9 and 10 at 1 and 5 min respectively. She was discharged home the following day and suffered no complications. Three weeks after the delivery she was seen by the anaesthetist who performed the EBP, and was found to be completely asymptomatic with no recurrence of headaches.

Discussion

Orthostatic headache which is thought to result from loss of CSF and intracranial hypotension is a well known, relatively frequent and an easily recognized complication in obstetric anaesthesia. It occurs either after deliberate dural puncture during spinal anaesthesia or after accidental dural puncture associated with epidural analgesia.⁶ Causes of spontaneous CSF leak are, however, not well understood. Fragility of the spinal meninges at the level of the radicular nerve root sleeves is suspected to predispose to the formation of meningeal diverticulae.^{3,7} In other cases dural tears following a traumatic event are sometimes reported in the patient's history.⁸

The diagnosis of SIH is based on clinical presentation, lumbar puncture revealing low CSF pressure and MRI scanning.⁹ Our patient presented with classical symptoms of postural headache combined with nausea and photophobia. The spinal anaesthetic she had received six years earlier appeared an extremely unlikely

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