

Postpartum headache: diagnosis and management



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Key points

Anaesthetists need to be aware of the differential diagnoses of postpartum headache as they are often the first to review these patients.

Symptoms and signs can overlap so if in doubt early neuroradiological imaging can ensure diagnostic accuracy.

In obstetric patients, conservative and pharmacological management of post-dural puncture headache (PDPH) is often ineffective.

Epidural blood patching (EBP) remains the gold standard treatment of PDPH.

Controversy still exists over when to perform an EBP, how much blood to inject, and how to manage patients post-EBP.

Postpartum headache is described as a complaint of headache and neck or shoulder pain in the first 6 weeks after delivery.¹ It is one of the most common symptoms with up to 39% of parturients experiencing headache in the first postpartum week.²

Regional anaesthesia has become the preferred choice for analgesia and anaesthesia in obstetric patients. With increasing awareness among our non-anaesthetic colleagues that post-dural puncture headache (PDPH) is a complication of regional anaesthesia, it is common for obstetric anaesthetists to be asked to review women with postpartum headache. It is very important therefore that anaesthetists are familiar with the possible differential diagnoses of postpartum headache and the management options available.

In this article, we will discuss the causes of postpartum headache, their diagnosis, and management. In particular, we will focus on PDPH as this continues to be a significant cause of postpartum headache which poses particular challenges for the obstetric anaesthetist.

Differential diagnosis: what should we be looking for?

Table 1 lists most of the possible causes of headache in the postpartum period taken from a prospective cohort study by Stella and colleagues.³ It highlights the presumed cause of postpartum headache in a group of 95 patients. Tension and migraine headache was considered the cause in 47% of women, pre-eclampsia or eclampsia in 24%, and PDPH in 16% of the study group.³

With such a range of possibilities, a good history, physical examination, and the recognition for the need for early neuroradiological imaging should ensure diagnostic accuracy.

doi:10.1093/bjaceaccp/mkr025

Non-specific/tension headaches

Changes associated with pregnancy and motherhood should always be considered when looking for a cause of headache. These changes can result from fluctuating hormone and hydration levels, caffeine withdrawal as well as various life changes associated with motherhood. An accurate history can highlight simple aggravating factors such as sleep deprivation, or indeed relieving factors such as food or fluid intake and sleep.

Tension headache is characterized by a mild-to-moderate 'band-like' headache that lasts from 30 min to 7 days. It is not aggravated by physical activity and is usually self-remitting. There can be associated neck and shoulder pain resulting in a musculoskeletal component. Treatment includes simple analgesia, massage, or physiotherapy.

Migraine

Migraine headache is usually described as a recurring, unilateral headache lasting 4–72 h. It may be pulsating in nature and associated with nausea and photophobia. There may be focal neurological signs immediately preceding the headache, often described by the patient as 'aura'. Migraine may be associated with visual disturbances such as flashing/flickering lights, zigzag lines, and even temporary blindness, or the experience of numbness, tingling sensations, and slurred speech.

Patients with a history of migraine may notice a reduction in symptoms during pregnancy as a result of hormonal changes. However, immediately postpartum, they may quickly experience a recurrence, with 34% suffering a migraine within the first week postpartum and 55% within the first month. Generally, symptoms are milder and follow their typical pattern. It is rare for migraine to manifest for the first time during the postpartum period.

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Table 1 Differential diagnosis and incidence of postpartum headaches

Headache aetiology
Non-specific/tension headache
Migraine
Pre-eclampsia/eclampsia
Post-dural puncture headache
Cortical vein thrombosis
Subarachnoid haemorrhage
Posterior reversible leucoencephalopathy syndrome
Space-occupying lesion—brain tumour, subdural haematoma
Cerebral infarction/ischaemia
Sinusitis
Meningitis

Treatment includes simple analgesia, non-steroidal anti-inflammatory drugs (NSAIDs) and 5-HT agonists such as sumatriptan.

Hypertension/pre-eclampsia

Gestational hypertension is commonly associated with headache. However, when the headache is associated with hypertension, proteinuria, or both, the diagnosis of pre-eclampsia should be considered. In pre-eclampsia, headache is a serious premonitory sign, being present in over 50% of women who go on to develop eclampsia.⁴ Physical examination may reveal hypertension, peripheral oedema, and brisk reflexes. It can be associated with HELLP (haemolysis, elevated liver enzymes, and low platelets) syndrome and so it is important to check liver function for elevated alanine aminotransferase and aspartate transaminase, platelet count, serum urate, and proteinuria, before establishing a diagnosis.

Eclampsia is a hypertensive encephalopathy characterized by headache, visual disturbance, nausea and vomiting, seizures, and stupor which may progress to coma. Headache is often bilateral, pulsating in nature, and aggravated by physical activity. The diagnosis of eclampsia postpartum may be difficult in a parturient that has a PDPH, as eclampsia first manifests postpartum in 11–44% of affected women.⁴

In both eclampsia and pre-eclampsia, headache can be managed with simple analgesia but control of the underlying condition is imperative to prevent harm to the mother.

Cortical vein thrombosis

The incidence of cortical vein thrombosis is increased in pregnancy and is estimated to be between 10 and 20 per 100 000 deliveries in developed countries and higher in developing countries.¹ Patients complain of a non-specific headache often accompanied with focal neurology and seizures. The headache can often be difficult to distinguish from PDPH as it may have a postural component. Indeed, several cases of cortical vein thrombosis have been associated with PDPH, possibly secondary to cerebral vasodilatation after cerebrospinal fluid (CSF) leak and prolonged dehydration.

Diagnosis is best confirmed by magnetic resonance imaging (MRI) and MR venography. Symptom control is the mainstay of treatment with the focus on seizure prevention. The use of anticoagulant therapy remains controversial.

Subarachnoid haemorrhage

The incidence of subarachnoid haemorrhage is increased in pregnancy occurring in 20 per 100 000 deliveries,¹ usually presenting in patients with arteriovenous malformation, cerebral aneurysms, and hypertensive encephalopathy. The classic presentation is of an acute onset of intense, incapacitating unilateral headache, accompanied by nausea, neck stiffness, and altered consciousness. Diagnosis is confirmed with urgent computed tomography (CT) and urgent neurosurgical opinion should be sought.

Posterior reversible leucoencephalopathy syndrome

First described in 1996 after recognition of a consistent syndrome presentation in a diverse group, patients describe a severe, diffuse headache, of an acute or gradual onset, occasionally associated with focal neurological deficit such as loss of vision, seizures, and altered level of consciousness.⁵ There may be an association with pre-eclampsia, as the pathophysiology of posterior reversible leucoencephalopathy syndrome is similar to that of a hypertensive encephalopathy where loss of the cerebrovascular autoregulation is thought to compromise the blood–brain barrier resulting in oedema. This process can be reversed by prompt recognition and supportive therapy which includes aggressive treatment of hypertension and seizure prophylaxis.

Neuroradiological imaging shows symmetrical areas of cerebral oedema, predominantly in white matter regions of the posterior circulation.¹

Space-occupying lesions

Headaches are usually dull in nature and associated with symptoms of raised intracranial pressure such as nausea and vomiting. Occasionally, focal neurology and altered level of consciousness may be present. Diagnosis is dependent on history, examination, and neuroimaging. If there is confirmation of a tumour or bleed, urgent neurosurgical opinion should be sought.

Cerebral infarction/ischaemia

This is one of the causes of stroke in pregnancy and can occur in the peripartum period. Patients present with a sudden onset of headache, vomiting, seizures, and focal neurological deficit. Diagnosis requires cerebral angiography as CT or MRI is often normal. Specialist opinion should be sought on appropriate management.

Sinusitis

Headache is frontal, particularly over the sinuses, and is worse in the morning. Pain is secondary to inflamed paranasal sinuses and associated with nasal congestion, purulent nasal discharge, anosmia, and fever. Initial management includes antibiotics, decongestive, and antipyretics, although some patients may require referral to ENT for a nasal endoscopy and CT/MRI of the sinuses.

Meningitis

The severe headache of meningitis may manifest in the first few days postpartum and is classically associated with neck stiffness, photophobia, and fever. On examination, Kernig and Brudzinski signs are elicited and a petechial rash may be present.

Diagnosis is confirmed by examination and culture of CSF after exclusion of other pathology by CT scan.

Post-dural puncture headache

PDPH occurs after intentional dural puncture with a spinal needle or unintentional dural puncture with an epidural needle. In parturients, the collective risk of unintentional dural puncture with an epidural needle is ~1.5%, and of these, 52.1% will experience PDPH.⁶ After spinal injection, the incidence of headache ranges from 1.5% to 11.2%, depending on the size and type of the needle.⁶ Interestingly, up to 38% of PDPH can arise after a seemingly uneventful procedure.⁷

The cause of PDPH is believed to be secondary to intracranial hypotension caused by CSF leak through the puncture site. Pain is thought to be a result of either traction on intracranial structures or from compensatory cerebral vasodilatation. The postural component is thought to be triggered by vascular distension that occurs on standing followed by an increased hydrostatic gradient forcing more CSF leakage. Adenosine receptors may be activated to compensate for the sudden loss of intracranial volume, resulting in cerebral vasodilatation and causing headache. The physiology is reversed when the patient lies down.

Headaches normally occur in the first 72 h after dural puncture. Patients complain of a frontal or occipital headache, characterized by its postural component. The severity increases on sitting or standing, coughing or straining, and improves on lying down.

Associated symptoms include: neck stiffness, nausea, vomiting, visual disturbances, photophobia and auditory symptoms, such as hearing loss, hypacusis, and tinnitus. In severe cases, cranial nerve palsy of the abducens nerve may occur as this is susceptible to traction when CSF volumes are low.

Abdominal compression over the liver with the patient lying at 45° may sometimes improve the headache but should be undertaken with care in patients who have had a recent Caesarean section (Gutsche's test).

Prevention of PDPH

Unintentional dural puncture may be witnessed by the anaesthetist when performing an epidural. Various strategies to prevent the onset of headache have been used such as prophylactic epidural blood patch (EBP) or intrathecal saline injection. However, the most widely practiced is that of threading the epidural catheter into the intrathecal space at the time of dural puncture. Study results are conflicting but suggest that most benefit occurs when the catheter is left in place for 24 h. A recent meta-analysis demonstrated a significant reduction in the incidence of PDPH from 66% to 51% and requirement for EBP from 59% to 33% after intrathecal catheter placement.⁷ The safety of this practice in individual units must also be carefully considered.

Treatment of PDPH

Most treatment options relieve the symptoms of PDPH by attempting to replace lost CSF, minimize cerebral vasodilatation, or seal the dural puncture site. Treatment options can be divided into conservative, pharmacological, and EBP.

Conservative treatment

Symptoms of PDPH can be controlled in the hope that the hole in the dura will seal spontaneously. Patients are advised to bed rest, maintain hydration, and to take simple analgesics, such as paracetamol and NSAIDs. When a dural puncture is made by a small-bore spinal needle, conservative treatment may be effective. However, the young obstetric patient is particularly at risk of debilitating PDPH and conservative measures may not be effective, especially when a dural puncture is made with a large-bore epidural needle.

Pharmacological treatment

Caffeine

Caffeine is a cerebral vasoconstrictor and has been prescribed for the treatment of PDPH. However, it remains controversial with no strong evidence to support its use and its benefits may only be transient. In addition, it is not without risk. It is a potent central nervous stimulant which reduces seizure threshold and it should not be prescribed for long-term therapy. Nevertheless, many anaesthetists encourage parturients with PDPH to drink highly caffeinated beverages in the hope that this will provide some symptomatic relief.

5HT-Agonists

Sumatriptan is a serotonin-receptor agonist and a cerebral vasoconstrictor. It is widely used for the treatment of migraine. Findings from case reports suggest that it may be effective when given as a subcutaneous injection, but randomized controlled trials have not shown benefits from using this agent. More recent data suggest that 5-HT_{1b/1d} agonists such as frovatriptan may be more effective cerebral vasoconstrictors.⁸ The study was small, non-randomized,

and showed effectiveness in prevention as opposed to management of the headache and further studies are required to establish the effectiveness of this agent.

Adrenocorticotrophic hormone

Adrenocorticotrophic hormone may increase CSF production but has yet to be recommended as a therapy for PDPH. A small randomized clinical trial has shown no difference in the severity of headache or requirement of EBP.⁹

Epidural saline or dextran 40

Historically, these have been infused via the epidural catheter and used with some benefit in the past, especially in patients in whom EBP was contraindicated. Theoretically, the effects are thought to be similar to blood, raising the epidural pressure, reducing CSF leakage, and resolving headache. Recent reviews have concluded treatment to be transient or ineffective for the management of PDPH.

Epidural blood patch

A recent Cochrane review¹⁰ has concluded that therapeutic EBP is beneficial compared with conservative treatment for PDPH. It remains the gold standard treatment for persisting PDPH, but success rates are often exaggerated. Although initial pain relief from an EBP is quite high, many women experience a recurrence. Consequently, women should be advised that the chance of complete cure from a single EBP is ~50%, and in up to 40% of cases, a second EBP may be required.¹¹

The mechanism of action is thought to be by a tamponade effect of blood in the epidural space, resulting in an increased intracranial pressure and relief from headache. Formation of a clot seals the puncture site preventing further CSF leak.

Table 2 outlines the procedure for performing an EBP. Anaesthetists should explain the procedure, risks, and benefit of the blood patch and ask the patient to give written and informed consent. Contraindications include: known coagulopathy, local or

systemic infection (patient should be afebrile), increased intracranial pressure, and patient refusal.

The optimal timing for the procedure has not been studied adequately, although observational studies have shown a higher failure rate if performed in the first 24 h of the dural puncture.¹² In practice, most obstetric anaesthetists wait 24–48 h before performing an EBP.

The optimum volume of blood to inject has not been the subject of a randomized control trial. Commonly, 20 ml of blood is the standard volume used, although smaller volumes have been used with success. Some anaesthetists inject up to 30 ml or as much as can be tolerated until the patient experiences the sensation of pain or pressure in the back, buttocks, or legs, secondary to nerve root irritation.

There is no clear consensus in the literature about how to manage patients after the EBP procedure. Some anaesthetists admit patients overnight for bed rest, prescribe stool softeners, and advise avoidance of straining or too much physical exertion in the belief that this regime will maximize the chance of the EBP sealing the hole in the dura. Others, however, observe the patient for a few hours and then discharge home allowing them to return to normal duties.

Traditional teaching advises that blood cultures should be taken at the time of EBP to detect any infection. This practice varies among units, but most no longer routinely perform blood cultures in apyrexial patients as the results are mostly negative.

Early complications include backache during injection, fever, bradycardia, and seizures. Late complications include meningitis; subdural haematoma, arachnoiditis, and radicular pain.

Most patients report almost instantaneous relief of headache but may complain of neck or backache for 24 h. If the first procedure fails, a second may be attempted but it is important to consider other diagnoses and when in doubt organize appropriate investigations and referral.

Conflict of interest

None declared.

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Table 2 Performing an EBP

Obtain consent
I.V. access
Complete asepsis by both anaesthetists
Position the patient—usually laterally for patient comfort
The EBP should be performed at or one space below the original site of dural puncture with local anaesthetic infiltration
Once the epidural space has been identified, the assistant should aseptically withdraw ~40 ml of blood from a peripheral vein
10–30 ml should be injected into the epidural space. Should radicular pain occur, slow or stop injecting
Post-procedure, the patient should lie flat for 1–2 h after which they can ambulate with the advice to refrain from vigorous activity or lifting for a few days
Consider prescribing stool softeners to avoid constipation and the need for the Valsalva manoeuvre which may dislodge the patch
Before leaving hospital, patients should be counselled to report fever, severe back pain, or radicular pain immediately

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Please see multiple choice questions 25–28.