Postpartum post-dural puncture headache: is your differential diagnosis complete?

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We describe a patient with an intracerebral haemorrhage following an accidental dural puncture during an attempted epidural for pain relief in labour. Anaesthetists need to include intracerebral haemorrhage in the differential diagnosis of post-dural puncture headache in the puerperium.

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Epidural analgesia in labour, complicated by an accidental dural puncture and a post-dural puncture headache (PDPH) occurs not uncommonly.1 The prevention and treatment of PDPH is a therapy ladder of bed rest, fluids, caffeine and other medication leading, if necessary, to an epidural blood patch.1 The combination of pregnancy, labour, dural puncture and headache may suggest PDPH, but this is not the only cause of such a headache. In this sort of situation, the headache poses a differential diagnostic challenge.

We describe a patient with an accidental dural puncture, treated with an epidural blood patch, who was later diagnosed with an intracerebral haemorrhage. The differential diagnosis of the headache and the possible relationship of the intracerebral haemorrhage to the dural puncture and the epidural blood patch are discussed. We believe this is the third patient of its kind described in the literature.2 3

Case report
A previously healthy 30-year-old primigravid, Asian woman, weight 70 kg, height 1.65 m, was admitted for hyperemesis when she was 7 weeks pregnant. She complained of headaches and dizziness. No neurological abnormalities were seen during the admission. She was
treated with antiemetics with good effect and discharged after 10 days. Throughout her pregnancy, her blood pressure remained normal. At 42 weeks, the patient was re-admitted in active labour and an oxytocin infusion was started to augment it. At the patient’s request, the anaesthesia resident tried to insert an epidural catheter for pain relief. The first attempt with an 18 G Tuohy needle resulted in a dural puncture. No spinal catheter was placed. A second attempt at a higher level resulted in inability to advance the epidural catheter. After consultation with the anaesthetist and the obstetrician, the resident aborted further attempts and advised the patient of the possibility of PDPH. After 1.5 h, because of lack of progress of labour, a Caesarean section was performed and a healthy child was delivered.

During the preoperative interview, the anaesthetist was under the false impression that the patient had insufficient command of the Dutch language, as she did not understand him completely and responded inadequately. This difficulty in communication was noted by the patient’s partner, because of this together with the dural puncture, the anaesthetist on call chose to give the patient a general anaesthetic. During the anaesthetic, which consisted of induction with thiopental and succinylcholine, the arterial pressure ranged from 140–105/90–65 mm Hg. No increases in arterial pressure were recorded using an automatic continuous electronic recording machine with a 3 min interval, at intubation, emergence or after a single dose of oxytocin 5 IU, which the patient received after delivery. In the recovery room, the patient was alert and communicative. After surgery, the patient received nadroparine, a low molecular weight heparin, 2850 IU for thromboprophylaxis. The day after surgery, the patient complained of a severe throbbing headache that was relieved by lying down. There was no nuchal rigidity. The patient was given extra fluids and caffeine 500 mg day \(^{-1}\) orally as treatment for a suspected PDPH. In addition, we offered her an epidural blood patch, which she initially refused. On the third day post partum she was to be discharged but, because of the persisting headache, she requested an epidural blood patch. The headache was of a severe throbbing type, worsening on sitting up. She was apyrexial, with no focal neurology. Muscle strength was equal in both hands and legs. Pupils were equal and reactive to light. An epidural blood patch was performed under aseptic conditions, and 10 ml autologous blood was administered at the level of the dural puncture. Immediately afterwards, the symptoms decreased but did not disappear completely. The patient was observed in the post anaesthesia care unit for an hour and then returned to the ward. One and a half hours later, we were informed that the patient had developed a hemiparesis. On physical examination, the patient had reduced consciousness, with a Glasgow Coma Score of eyes, 3; best motor response, 6; best verbal response, 4. She had a left-sided hemiparesis and focal seizure activity. Arterial pressure was 170/90 mm Hg, with a regular pulse of 60 beats min \(^{-1}\). A CT scan showed a recent and a newer intracerebral haemorrhage in the globus pallidus/putamen area (Fig. 1). The patient was admitted to the intensive care unit, as she required ventilatory support because of a further reduction in her level of consciousness. A CT scan with contrast and MR angiography failed to show any intracerebral aneurysm. Full clotting studies were performed and the results were normal. After 3 days the patient started to improve. She was discharged after one month with a left arm and leg paresis of 4 (on a scale of 1–5 with 5 as best score), a slight central facial nerve paresis and cognitive dysfunction. Two months later, the patient was continuing to improve, with only the cognitive dysfunction remaining. The patient has subsequently had a second Caesarean section under general anaesthetic. Her only outstanding neurological problem is now epilepsy.

**Discussion**

A successful epidural in the labour room produces a satisfied patient, but a dural puncture with ensuing PDPH results in an unsatisfactory situation. This contrast may be the reason why PDPH receives such detailed investigation and research by anaesthetists.\(^4\)\(^-\)\(^8\) Although the chance of PDPH following dural puncture is 76–85%,\(^9\) it is not the only cause of headache in pregnancy.\(^10\)\(^-\)\(^11\) Other causes are summarised in Table 1.

The symptoms of PDPH are a postural headache, usually fronto-occipital and of a throbbing or dull aching nature and often accompanied by dizziness, nausea and vomiting, visual

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**Fig 1** CT scan with contrast taken after intracerebral haemorrhage, showing new intracerebral bleeding (top arrow) in the globus pallidus/putamen area and an older bleeding site (bottom arrow).
When conservative management fails, the next treatment for PDPH is an epidural blood patch. The optimal response is cessation of the headache. The effect of the blood patch results from a volume or pressure effect of the fluid injected with cranial displacement of the cerebrospinal fluid, rather than a plugging effect, which was the original concept behind an epidural blood patch. The mass effect of the epidural blood patch can cause marked increases in intracranial pressure, with neurological sequelae. A nerve palsy as well as an acute intracranial pressure increase have been described.17 18

Cerebrovascular accidents are an important non-obstetric cause of maternal morbidity and mortality, causing as many as 12% of maternal deaths.19 20 Our patient’s dural puncture alerted the anaesthetist to the possibility of PDPH. Then a headache was diagnosed as a PDPH. This led to conservative treatment followed by an epidural blood patch, before an intracerebral haemorrhage was detected. On CT, our patient showed two sites of intracerebral bleeding, one recent and one older. The timing of her older bleed cannot be determined. Several factors may have contributed to the first intracerebral bleed. The patient had a history of headaches, vomiting and dizziness but these episodes were not accompanied by neurological symptoms. During all her antenatal checkups, arterial pressure was normal and no oedema or proteinuria were detected. She received an infusion of oxytocin to augment the labour. A continuous infusion of oxytocin can cause hypertension that might result in bleeding from aneurysms or weak areas in cerebral vessels. The infusion rate was 10 milliunit min−1, which normally does not produce hypertension. During labour, as well as during anaesthesia for Caesarean section, the patient was monitored and no elevations in arterial pressure were recorded. During anaesthesia, arterial pressure was measured non-invasively every 3 min and might briefly have been higher, but the CT scan with an angiogram failed to show any vascular abnormalities or signs of vasculitis.

It is suspicious that the patient had difficulty communicating just before the anaesthesia but after the dural puncture. It has been reported that dural puncture can cause neurological disorders.12 21–23 The resident failed to place an intrathecal catheter after the dural puncture. This might have decreased cerebrospinal fluid efflux and prevented a PDPH.24 The dose of naloxone used is unlikely to be the cause of bleeding. The headache was typical of a PDPH but may have been due to the older or the recent intracerebral bleed. Or was the headache due to any combination of the three? The onset of the neurological symptoms from the more recent intracerebral bleed was related in time to the epidural blood patch. The epidural blood patch did not relieve the headache completely, possibly because of the small volume of blood (10 ml) used. We do not know if the epidural blood patch caused the rebled because of a pressure effect or if the bleed had already occurred. It may be that the epidural blood patch prevented an even larger intracerebral haemorrhage because of the increase in intracranial pressure from the volume displacement.16

### Table 1

<table>
<thead>
<tr>
<th>Origin of headache</th>
<th>Typical features</th>
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<tr>
<td>Post-dural puncture headache</td>
<td>Throbbing fronto-occipital headache, relieved by lying down, interscapular pain, nuchal rigidity, often accompanied by dizziness, nausea and vomiting, visual disturbances, photophobia and auditory symptoms, cranial nerve palsy, upper and lower limb pain12</td>
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<tr>
<td>Non-specific coincidental headache</td>
<td>None</td>
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<tr>
<td>Pregnancy-induced hypertension, (pre-) eclampsia</td>
<td>Throbbing headache, often hypertension and proteinuria developing during pregnancy, transient focal disturbances such as amaurosis, aphasia, hemiplegia. Can progress to convulsions25</td>
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<tr>
<td>Meningitis</td>
<td>Acute occipital headache, neck stiffness, fever, photophobia26</td>
</tr>
<tr>
<td>Cerebral tumour</td>
<td>Dull, deep intermittent headache, elevated intracranial pressure, drowsiness, unequal pupils, papilloedema, convulsions</td>
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<tr>
<td>Cerebral vein thrombosis</td>
<td>Generalized or focal neurological symptoms and signs. Headache in 80% of cases, nausea, vomiting. Psychiatric symptoms. There may be an alteration of consciousness or cerebellar uncoordination. Other neurological signs include papilloedema, focal deficits, or seizures. Papilloedema may be associated with transient visual abnormalities, while seizures may be focal or generalized. Most deficits are motor and sensory, usually unilateral, and involve mostly the lower extremities27</td>
</tr>
<tr>
<td>Migraine</td>
<td>Generalized or unilateral paroxysmal throbbing headache, nausea, vomiting, dizziness, visual disturbances, aura and prodromas: changes in mood, anorexia, scintillating scotomas28</td>
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<tr>
<td>Intracranial bleed—Intracerebral</td>
<td>Sudden severe headache (‘the worst in my life’). Weakness and/or numbness of one side of the body. Slurred speech or language difficulties. Loss of vision in one or both eyes; double vision. Incoordination, unsteadiness, giddiness. Drowsiness, coma28</td>
</tr>
<tr>
<td>Intracranial bleed—Subdural</td>
<td>Headache from mild to severe, localized or generalized. Intermittent with slow onset, often a history of trauma. Fluctuating changes in consciousness28</td>
</tr>
<tr>
<td>Intracranial bleed—Subarachnoid</td>
<td>Occipital headache with sudden onset, severe, constant. Prodomal pain in one eye, ptosis, blunting of consciousness, vomiting, stiff neck28</td>
</tr>
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</table>

Disturbances, interscapular pain, nuchal rigidity, photophobia and auditory symptoms. The cause of the headache is related to loss of cerebrospinal fluid and lowering of the cerebrospinal fluid pressure. The actual mechanism, however, is not clear. Two explanations have been offered. First, the lowering of cerebrospinal fluid pressure causes descent of the brain, leading to traction on pain-sensitive anchoring structures in the cranium. The second explanation is that the loss of cerebrospinal fluid produces a compensatory dilatation of the cerebral veins and venous sinuses, which may also play a role in the production of these headaches.12 With enough traction, dural vessels may tear, leading to subdural haematoma. Subdural haematoma and cranial nerve involvement following dural puncture have been extensively described.13–15 However, in our patient the bleeding was intracerebral.
This report emphasises the need for a broad differential diagnostic approach when confronted with neurological symptoms following dural puncture; not all headaches are just a PDPH.

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Use of nitrous oxide causing severe visual loss 37 days after retinal surgery

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A case of severe visual loss following nitrous oxide anaesthesia in the presence of an intraocular perfluoropropane (C3F8) gas bubble is described. The diabetic patient had previously undergone vitreoretinal surgery at which time the gas had been inserted. The case highlights the use of