

# Cranial subdural haematoma associated with dural puncture in labour

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A 23-yr-old primigravida sustained a dural puncture during epidural catheter insertion and developed a headache that settled with oral diclofenac and codydramol. On the third day after delivery, she convulsed twice without warning. As plasma urate was increased, the putative diagnosis of an eclamptic fit was made, and magnesium therapy was started. A contrast CT scan revealed that the cause of the patient's symptoms was a subdural haematoma with raised intracranial pressure. A coincidental arteriovenous malformation was noted. This case emphasises the need to consider the differential diagnoses of post-partum headache. The management of acute intracranial haematoma is described.

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Inadvertent dural puncture with subsequent headache is a recognized complication of epidural analgesia.<sup>1</sup> This case report emphasizes the need for careful and ongoing assessment of these patients in order to ensure that less common causes of headache are not overlooked.

## Case report

A 23-yr-old primiparous Indian woman presented in labour following spontaneous rupture of membranes at home. She was distressed and requesting epidural analgesia. The combination of a high body mass index (height 144 cm, weight at last antenatal clinic before delivery 82 kg; body mass index 39.5) and a poorly compliant and distressed patient made epidural catheter insertion difficult, and in the process an inadvertent dural puncture occurred. The catheter was sited at a higher level, and good analgesia was obtained with a test dose of 0.25% bupivacaine 3 ml, followed by a further 10 ml with fentanyl 50 µg. Subsequent top-ups by the anaesthetist with 0.1% bupivacaine 10 ml plus fentanyl 2 µg ml<sup>-1</sup> provided good analgesia without side-effects.

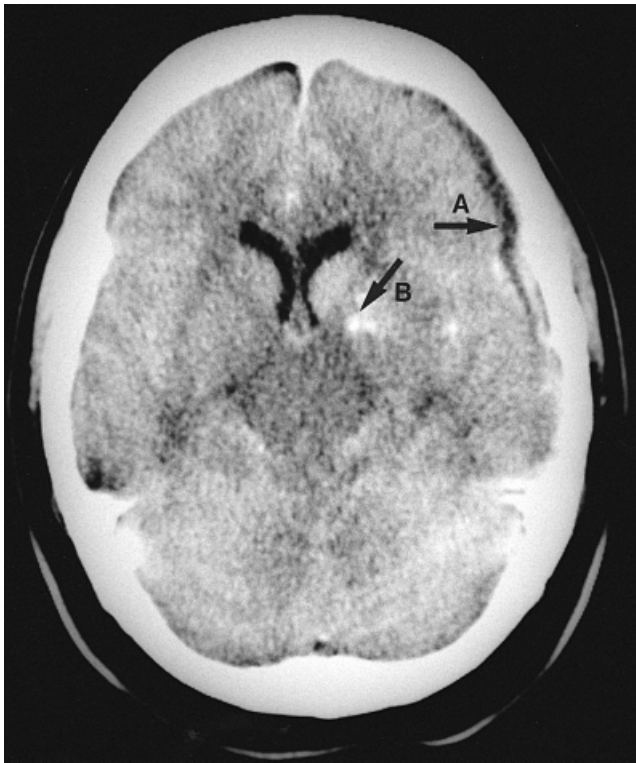
The contractions were augmented with an oxytocin infusion, as meconium was present in the liquor. Four hours after the epidural, the patient developed low-grade pyrexia (37.8°C) and was started on amoxicillin and clavulanic acid (co-amoxiclav 1.2 g) i.v.; a blood culture was taken. After a further 3 h the patient was fully dilated and started to push. At this time she began to complain of a continuous, dull, moderate occipital headache with no radiation. In view

of the pyrexia, meconium and a worsening cardiocograph trace, a forceps delivery was performed.

Overnight, the patient complained of severe occipital headache, although this settled with paracetamol and rest. This was assumed to be a post-dural puncture headache (PDPH) because it had a postural component (it was worse on sitting up or standing). Over the next 2 days the patient was regularly reviewed and her headache improved rapidly with oral codydramol and diclofenac. At no time did she complain of nausea, vomiting, photophobia or neck stiffness. Her pyrexia settled within a day of delivery. On the third postnatal day, it was agreed that a blood patch was not indicated and discharge was planned for the next day.

That evening, the patient had two self-limiting 30 s tonic-clonic convulsions. After 30 min of post-ictal drowsiness, the patient was fully alert with a Glasgow coma score of 15. The only abnormal findings on examination were symmetrical, moderately brisk reflexes. A putative diagnosis of eclamptic fits was made, strengthened by a raised plasma urate level of 438 µmol litre<sup>-1</sup> (normal range 100–400 µmol litre<sup>-1</sup>). Blood pressure was normal, and there was no significant proteinuria or oedema. A raised white cell count of 17.5×10<sup>9</sup> ml<sup>-1</sup>, consisting mainly of neutrophils, was noted. High dependency care was initiated and magnesium therapy started as per unit protocol (magnesium sulphate 4 g i.v. as a slow bolus followed by an infusion of 1 g hourly).

After a stable night during which the occipital headache recurred, the obstetric anaesthetic consultant reviewed the



**Fig 1** Contrast-enhanced cerebral CT scan showing (A) small subdural haematoma with an outer, posterior layer of fresh blood and (B) AVM arising from the basal ganglia.

patient. It was felt that the onset of PDPH during labour, combined with a tonic-clonic seizure 3 days after delivery, warranted an urgent computerized tomography (CT) scan to exclude intracranial pathology. Contrast-enhanced CT revealed a left-sided subdural haematoma, evidence of raised intracranial pressure without midline shift, and an arteriovenous malformation (AVM) in the basal ganglia (see Fig. 1).

The patient was transferred to a neurosurgical centre where she returned to a symptom-free state without intervention. A cerebral magnetic resonance imaging (MRI) scan revealed no further information. It has been decided to manage her conservatively with regular neurological follow-up.

## Discussion

This case illustrates the difficulties of diagnosing the cause of headache and convulsions in the peripartum period. The differential diagnosis of post-delivery headache in this case includes PDPH, pre-eclamptic headache, subdural or subarachnoid haemorrhage, simple tension or migraine headache, meningitis and cortical vein thrombosis.<sup>1</sup> With the knowledge that dural puncture had occurred earlier in labour and from the postural component of this headache, it was assumed that PDPH was the most likely diagnosis. However, the onset of the headache was early, coinciding with the second stage of labour and the initiation of pushing,

making the diagnosis of PDPH less likely. The diagnosis of pre-eclampsia as the causal condition was discounted, as no other symptoms were present. Subdural or subarachnoid haemorrhage were not considered at the time of onset of the headache, as these are extremely rare causes to present *de novo*. It was only when the convulsions occurred that this omission from the differential diagnosis was corrected. Other than an initial neutrophilia, there were no features to support a diagnosis of meningitis at this time; the pyrexia settled and the white count normalized. The history was not suggestive of cortical vein thrombosis, and this was excluded later on MRI scanning.

The differential diagnosis of the convulsions in this case includes eclampsia, intracranial haemorrhage, embolic episodes and epilepsy. Local anaesthetic toxicity can be excluded as the convulsion occurred on the third day after delivery. Subdural haematoma following dural puncture is a very rare complication of epidural analgesia in labour, and may be cranial or spinal.<sup>2-5</sup> The cause is low cerebrospinal fluid pressure following dural puncture leading to traction and tearing of thin-walled dural blood vessels.<sup>6</sup> In this case the onset of the headache was very early in relation to the time of dural puncture. It is unusual for a large CSF leak to have occurred in this short time. We have assumed that the tear and subsequent bleeding occurred at or around the time of onset of headache in labour. However, this may have been a tension or stress-related headache, and the pathological events may have occurred later that evening. It is not possible to pinpoint the time of vessel rupture definitively.

Cranial subdural haematomas may present acutely, subacutely or chronically with a variety of complaints including headache, altered level of consciousness or even psychiatric symptoms.<sup>7</sup> Acute subdural haematomas have been described as lesions causing signs or symptoms within 7 days of the bleed. Spinal subdural haematomas following epidural anaesthesia are extremely rare and of complex presentation.<sup>6, 8</sup> The presence of a suspected PDPH complicated by neurological deterioration, as in this case, should prompt a search for intracranial pathology.

The plain CT scan did not reveal the underlying pathology, and the subdural haematoma and AVM were only seen after contrast enhancement. CT may not show subdural haematomas without contrast, and this has previously been reported as a cause of delayed diagnosis.<sup>9</sup> Haemorrhage from the AVM at the time of pushing is a possible cause of the subdural haematoma but, more commonly, ruptured AVMs cause subarachnoid bleeding.

It was considered by the neurosurgeon and neuro-radiologist that the AVM seen on CT was an incidental finding and that the subdural haematoma was secondary to the dural puncture. The raised intracranial pressure was a result of the haematoma and its increasing space-occupying effect because of oedema. This is in marked contrast to the low CSF pressure believed to have caused the vessel tear.

Haemorrhage from an AVM is most common in women

under 25 yr and is most likely during the second trimester and in the immediate post-partum period.<sup>10</sup> The clinical presentation is sudden onset of headache, often accompanied by nausea and vomiting, photophobia, seizure activity, and focal motor and sensory disturbances.<sup>11</sup> In this case, the onset of headache coincided with initiation of pushing, arousing suspicion—in retrospect—of an intracranial event. Subarachnoid haemorrhage only occurs in one in 10 000 pregnancies, but is associated with a high maternal mortality.<sup>12</sup>

The management of subdural haematoma is either conservative (clinical observation and possible intracranial pressure monitoring) or surgical evacuation. For this reason the patient was transferred to a neurosurgical unit. In view of the small size of the haematoma coupled with a rapid return to normal neurological status, conservative management was deemed appropriate. Haematomas under 5 mm often spontaneously resolve. In cases requiring surgery, preoperative neurological status affects postoperative survival. Early haematoma evacuation and young age are favourable prognostic factors. This patient had no midline shift on CT scan although there was evidence of raised intracranial pressure. Parenchymal injury causes midline shift and is associated with decreased survival.<sup>13</sup>

This case also raises the issue of when a blood patch for PDPH should be performed. Reynolds recommends that, 'Headache after dural puncture with a large needle should be treated promptly with an epidural blood patch'.<sup>14</sup> Our unit policy is to assess all known dural punctures for blood patching after 24 h bed rest, hydration and analgesia. In this case, the patient improved rapidly with conservative measures and it was felt that a patch was not necessary. It could be argued that early blood patching may decrease the risk of subdural bleeding by preventing a fall in CSF pressure. In this patient such action probably would not have helped, as we believe the bleeding occurred during labour. It is our practice to discharge patients with PDPH 24 h after a successful blood patch, or when they are mobile in conservatively managed cases. All patients are reviewed in the obstetric anaesthetic clinic 1–2 weeks later to ensure that symptoms have not recurred or worsened, and are advised to contact the obstetric anaesthetist if any symptoms occur or recur. We feel that this follow-up minimizes the risk of late complications being missed.

The management of PDPH is often complex and is an important role for the obstetric anaesthetist. While we emphasize the rarity of subdural haematoma as a complication of dural puncture, this case highlights the importance of including it in the differential diagnosis. We suggest the urgent use of contrast-enhanced CT in all cases of seizure after dural puncture to exclude intracranial bleeding.

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