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## **Postpartum cerebral ischaemia after accidental dural puncture and epidural blood patch**

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Puerperal women are reported to have a rate of cerebral infarction 13 times greater than non-pregnant females. We report a case of cerebral ischaemia in a 30-yr-old healthy parturient after epidural analgesia for labour, complicated by dural puncture treated with two epidural blood patches. Investigations showed the development of cerebral ischaemia on postpartum

day 14. A transcranial Doppler ultrasonography showed vasospasm of the left middle cerebral artery still present at 3-month follow-up. At 1-yr follow-up, the patient had homonymous hemianopsia. We discuss the possible causative mechanism of the cerebral ischaemia in relation to the dural puncture and epidural blood patch.

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The incidence of ischaemic stroke in women of child-bearing age is 3.5–18 per 100 000 per year in western countries.<sup>1</sup> The risk is slightly increased during pregnancy, particularly during the postpartum period.<sup>1</sup> Puerperal women are reported to have a rate of cerebral infarction that is 13 times greater than non-pregnant females.<sup>2</sup> We report a case of postpartum cerebral ischaemia after epidural analgesia complicated by postdural puncture headache (PDPH) and epidural blood patch (EBP).

## Case report

A 30-yr-old gravida 2 para 1 woman was admitted to hospital in active labour. She had no history of neurological or haematological disease, migraine, lupus, malignancy or infection, except for pulmonary tuberculosis treated medically 2 yr before admission. She had no oedema, proteinuria, hyperuricaemia or hypertension. She took no medication, did not use alcohol or tobacco and reported no allergies.

Epidural analgesia was required when the cervical dilatation was 5 cm. During the attempt at epidural needle placement at L3–4, a dural puncture occurred with a 16-gauge Tuohy needle. After a new attempt at L2–3, the epidural catheter was correctly positioned and epidural analgesia for labour was conducted until the parturient delivered a healthy term infant.

Seven hours after delivery, the patient suffered from a dull, throbbing headache, relieved only by supine positioning, but recurring immediately after sitting or standing. Headache was so severe that we performed an EBP, injecting 18 ml of autologous blood, at the same level of dural puncture. The procedure was effective in relieving headache; the patient felt well and started lactation without discomfort.

On postpartum day 2, postural headache recurred and gradually became extremely severe and disabling. This prompted us to perform a second EBP on postpartum day 3, with injection of 20 ml of autologous blood at L3–4 level. After the procedure, she developed mental confusion and agitation. She still suffered with occipital headache, not relieved by recumbent position and not associated with other neurological symptoms, nausea or vomiting. Arterial pressure remained in the normal range throughout the puerperium. Because of the continuing headache, which

was no longer postural, a cranial computed tomography (CT) scan and, subsequently, a magnetic resonance imaging (MRI) were performed disclosing no abnormalities. In particular there was no sign of subarachnoid blood.

No medication, such as ergotamine or bromocriptine, was administered to the patient. On postpartum day 9, she developed generalized seizures followed by right hemiparesis, mainly affecting the face and arm, associated with visual disturbances. Again a CT scan showed no abnormalities except for a subtle effacement of cortical sulci. A transcranial Doppler ultrasonography (TCD) showed a left middle cerebral artery (MCA) mean flow velocity ( $V_m$ ) of 221  $\text{cm s}^{-1}$  and right MCA  $V_m$  of 165  $\text{cm s}^{-1}$ ; normal  $V_m$  was found in basilar artery. Follow-up TCDs were performed by the same examiner at 2, 5, 15 days, and 3 and 6 months after symptom onset (Table 1). The patient was transferred to ICU; nimodipine and phenobarbital were commenced.

Eventually, on postpartum day 14, a repeat MRI disclosed multiple hyperdense areas on T2-weighted images, involving the left internal capsule and the parieto-occipital white matter. These findings were consistent with ischaemic lesions involving the left MCA territory. Conditions such as hereditary antithrombin III deficiency, proteins C and S deficiencies were excluded. Laboratory studies, including vasculitis screening, electroencephalography and echocardiography, were normal.

At the time of discharge, the patient still showed right arm weakness and homonymous hemianopsia. One year after discharge, she had completely recovered from the motor deficit but still had homonymous hemianopsia.

**Table 1** Transcranial Doppler ultrasonography measurements. Data are mean flow velocity ( $\text{cm s}^{-1}$ )

Days after symptom onset	Right MCA	Left MCA	BA
0	165	221	65
2	170	215	53
5	171	241	55
15	156	170	49
90	125	142	64
180	65	60	55

MCA=middle cerebral artery; BA=basilar artery.

## Discussion

To our knowledge, no report has documented the development of cerebral ischaemia after epidural analgesia complicated by PDPH and EBP, after a normal pregnancy. Most of the reported cases of peripartum cerebral ischaemia were associated with a history of eclampsia and pre-eclampsia.<sup>3,4</sup>

In our case, diffuse arterial vasospasm, shown by TCD, caused the cerebral ischaemia. In obstetrics, vasospasm has reportedly been associated with either eclampsia or postpartum cerebral angiopathy (PPCA).<sup>5-7</sup> In our patient, however, eclampsia can be excluded. She was normotensive throughout pregnancy, labour and delivery, and the puerperium. PPCA is a rare clinical syndrome comprising headache, vomiting, seizure and focal neurological deficits after a normal pregnancy. It has always been reported as a reversible syndrome.<sup>7</sup> However, in our patient, vasospasm was still present at 3-month follow-up and homonymous hemianopsia was present at 1-yr follow-up. The patient refused any further diagnostic procedures. In our opinion, the sequence of events suggests a definite causal relationship between the development of vasospasm and what happened after the dural puncture. Whether the vasospasm was related to the dural puncture or to the subsequent EBP is unknown.

Perhaps the cerebral vasospasm was caused by the lumbar puncture *per se*. Shearer and colleagues<sup>8</sup> described eight women with postpartum PDPH who developed seizures and visual disturbances. Angiography performed in two of them revealed diffuse cerebral artery vasospasm. They concluded that cerebral vasoconstriction, possibly due to anatomical brain displacement, could provide an explanation for the development of both headache and seizures after dural puncture. However, our case is noteworthy for the severity of neurological impairment. Also, it is the first to demonstrate definite MRI evidence of ischaemic lesions as a result of vasospasm after dural puncture.

Alternatively, vasospasm could be primarily related to the EBP. Subdural and extradural haematomas are powerful stimuli for cerebral vasoconstriction.<sup>9</sup> We may hypothesize that blood, spreading through the dura mater, could have caused cerebral vasoconstriction. Griffiths and colleagues<sup>10</sup> have shown a significant subarachnoid spread of blood after EBP with MRI. In a recent study carried out in pigs,<sup>11</sup> cerebral blood flow (CBF) increased after removing 9 ml of cerebrospinal fluid (CSF) with a cisternal puncture. CBF returned to baseline after injecting 10 ml autologous blood in the lumbar epidural space and decreased, by as much as half the baseline value, after subdural injection of 10 ml autologous blood. Of note, three pigs had blood-stained CSF at puncture of cisterna, probably as a result of needle injury

of an epidural blood vessel, and CBF remained unchanged, after CSF drainage, in all three animals. The author suggested that EBP relieves PDPH through vasoconstriction caused by subarachnoid spread of the epidurally injected blood. In our case, the time elapsing between EBP and the onset of neurological symptoms matches the latency time after which typical post-subarachnoid haemorrhage vasospasm usually develops.

In conclusion, this case is of considerable complexity and difficult interpretation. Our two hypotheses are not mutually exclusive. Nevertheless, neither of them alone can consistently give an explanation of the series of events and the long-lasting cerebral vasospasm that occurred in our patient. As a last option we could speculate that dural puncture and/or EBP might have caused an irreversible case of PPCA where sustained cerebral vasospasm has been responsible for the development of cerebral ischaemia.

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