

Postpartum seizure and ischaemic stroke following dural puncture and epidural blood patch

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SUMMARY

A 33-year-old parturient experienced seizures, then an ischaemic stroke after caesarean section, while undergoing an epidural blood patch for dural puncture. A diagnosis of normotensive late postpartum eclampsia, with either a posterior reversible encephalopathy syndrome or postpartum vasculopathy, leading to stroke, was made – based primarily on a temporal relationship to the postpartum period and consistent findings on magnetic resonance imaging and angiography scans and an electroencephalogram. The difficulties in definitively elucidating the cause of seizures and cerebral infarction in the postpartum period and the impact of anaesthetic interventions in this case are discussed.

Key Words: late postpartum eclampsia, posterior reversible encephalopathy syndrome, cerebral ischaemia, dural puncture, caffeine, epidural blood patch

Patient permission was obtained for publication of this report. A 33-year-old primigravid woman at 39 weeks gestation was admitted with ruptured membranes and was induced with oxytocin. She was generally well with no history of pre-eclampsia or epilepsy. On admission, she was normotensive with 1+ of proteinuria on dipstick analysis.

Successful placement of a lumbar epidural catheter for labour analgesia occurred, but only after two dural punctures with an 18-gauge Tuohy needle. Six hours later, failure to progress necessitated an emergency caesarean which was performed after an epidural ‘top-up’; the epidural catheter was removed at the end of the operation.

On the second postoperative day she complained of posturally-related symptoms of nausea, pressure across the temples and severe neck and shoulder pain. She had developed ankle oedema, but neurological examination was unremarkable and she remained normotensive. Initial treatment with paracetamol, ibuprofen and oxycodone was ineffective and caffeine 100 mg twice a day was commenced for presumed postdural puncture headache.

On day three, although the patient’s neck stiffness was improving, she felt mildly confused. Two generalised tonic-clonic seizures, 30 minutes apart, ensued. Her blood pressure was 102/52 mmHg. Following the second seizure she was given diazepam 10 mg intravenously (IV), clonazepam 1 mg IV and magnesium sulphate 4 g IV loading dose, followed by an infusion. She was transferred to the network tertiary hospital, where her blood pressure was noted to be elevated at 160/100 mmHg, although laboratory screening for markers of eclampsia were negative.

A computed tomography brain on day four post-caesarean excluded an intracranial haemorrhage or mass. On day six magnetic resonance imaging (MRI) and magnetic resonance angiography excluded acute infarction and dural venous sinus thrombosis but showed features suggestive of both intracranial hypotension and cortical oedema in the posterior regions of the brain (Figure 1). An electroencephalograph on day five demonstrated posterior slowing.

Concurrently, the postural headache and neck pain continued to hamper the patient’s ability to ambulate such that, on day seven post-caesarean (and dural puncture), an epidural blood patch was performed injecting 38 ml of blood without sequelae.

On day eight the headache had improved markedly although the patient had become unsteady on her feet, forgetful and disorientated in time and place. She displayed mild dyspraxia, dysarthria, inappropriate affect and right-sided neglect (visual

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and sensory) with mild right-sided weakness and brisk reflexes. Her blood pressure was 150/100 mmHg.

Repeat MRI and magnetic resonance angiography on day nine now revealed increased hyper-

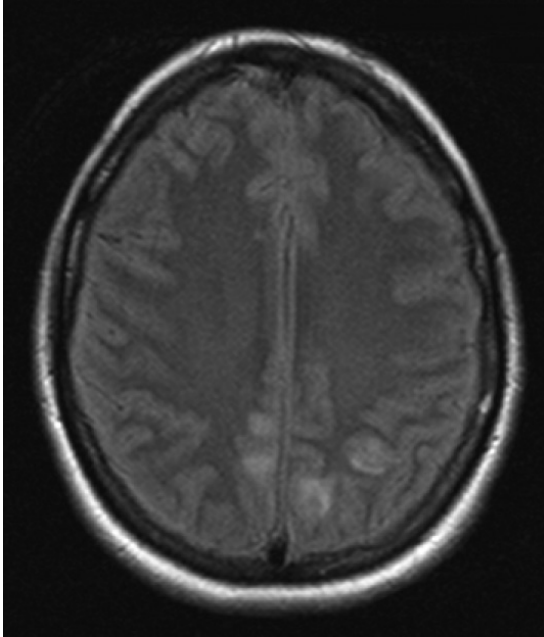


FIGURE 1: Fluid-attenuated inversion recovery windows on day six display slight hyperintensity in the posterior cerebrum (L > R), representing vasogenic white matter oedema.

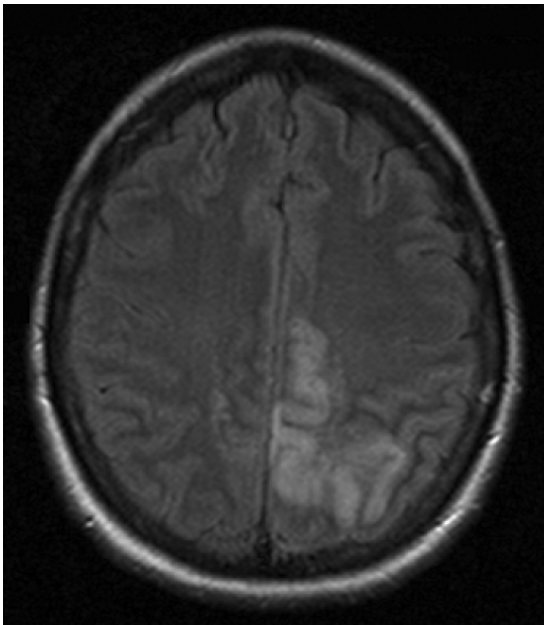


FIGURE 2: Axial fluid-attenuated inversion recovery windows on day nine show progressing hyperintensity (oedema) in the left posterior parietal and occipital lobes compared with day six images. Note also the improvement in the right parieto-occipital area, consistent with reversibility of the posterior reversible encephalopathy syndrome.

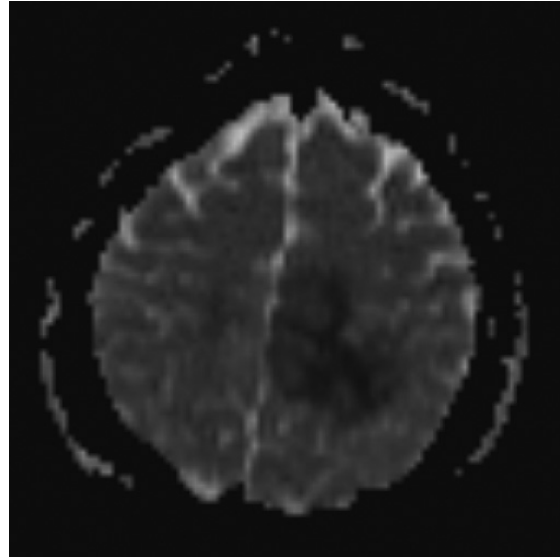


FIGURE 3: Axial apparent diffusion coefficient map on day nine, showing left parietal hypointensity indicating cerebral infarction. Note that the opposite pattern occurs when there is only vasogenic oedema present.

intensity on T2 and fluid-attenuated inversion recovery images, in the left posterior parietal lobe, consistent with a recent left middle cerebral artery infarct (Figures 2 and 3). In addition, multiple focal areas of narrowing affecting bilateral anterior, middle and posterior cerebral arteries indicated possible vasculitis. The neurologists believed the clinical picture was consistent with a postpartum vasculopathy leading to an ischaemic cerebral infarct, despite her vasculitic screen being negative for anti-nuclear, double-stranded deoxyribonucleic acid, extractable nuclear and anticardiolipin antibodies, rheumatoid factor and lupus anticoagulant. Treatment with intravenous fluid and nimodipine 60 mg orally three times a day was commenced for one week but aspirin was not prescribed.

Over the next 10 days, slow neurological improvement occurred and she was discharged to rehabilitation on day 19. A repeat MRI performed three months later showed a persisting left parietal infarct but the absence of vessel narrowing. Apart from some residual right-sided 'clumsiness' there had been almost complete resolution of the patient's neurological deficits. This was consistent with the vasospasm resolution one sees in a posterior reversible encephalopathy syndrome (PRES).

DISCUSSION

The most probable diagnosis in this case was late postpartum eclampsia with a posterior reversible

encephalopathy syndrome, and whether anaesthetic interventions could have contributed to the clinical picture needs to be considered.

In late postpartum eclampsia, the onset of convulsions occurs more than 48 hours but less than four weeks after delivery. It accounts for 5 to 17% of all cases of eclampsia and is by far the most common cause of seizures in this period¹. However late postpartum eclampsia frequently presents without the classic triad of eclampsia, namely hypertension, proteinuria and oedema². Lubarsky et al³ reported that of 54 cases of late postpartum eclampsia, 44% had not been identified as pre-eclamptic before seizure onset. However Veltkamp et al⁴ highlighted that both the patients they described with late postpartum eclampsia, with no pre-eclamptic prodrome, exhibited severe headaches and visual disturbance during the hours preceding the seizure. Our patient also complained of a headache and feeling 'fuzzy', perhaps representing visual disturbance.

Characteristic MRI findings of eclampsia and late postpartum eclampsia include reversible hyperintensities on T2 weighted images in the cortex, white matter and adjacent to the grey-white matter junction⁴. When PRES is present, up to half of these cases have a predilection for vasogenic oedema in the posterior circulatory territories².

PRES is a clinical and radiological syndrome of heterogeneous aetiologies grouped together because of similar findings on neuroimaging⁵. Eclampsia is a common aetiology, but there are also associations with hypertensive encephalopathy, renal failure and the use of cytotoxic and immunosuppressant drugs⁶. Clinically, PRES is characterised by headaches, altered consciousness, visual disturbances, seizures and frequently (though not always) hypertension⁵. T2 or fluid-attenuated inversion recovery MRI images typically show symmetrical parieto-occipital hyperintensities which appear hypodense on computed tomography; this represents white matter oedema in the posterior cerebral hemispheres^{6,7}. With treatment, resolution of neuroimaging abnormalities within days to weeks is expected⁵.

Despite its name, the syndrome is not always reversible, with cerebral infarction a potential consequence^{6,8,9}. Apparent diffusion coefficient maps and diffusion weighted imaging help distinguish the reversible vasogenic oedema of PRES from acute infarction⁵. For instance, vasogenic oedema is usually visualised as increased signal on apparent diffusion coefficient maps and hypo- or isointense signal on diffusion weighted imaging^{5,10,11}.

Acute cerebral infarction produces the opposite effect⁵.

The increased signal on our patient's day six apparent diffusion coefficient MRI mapping suggested vasogenic oedema underlying the seizure activity. Furthermore, the improvement in T2 hyper-intensity in right parieto-occipital areas on day nine (Figures 1 and 2) was consistent with the reversibility of PRES. Unfortunately, the left parietal area became infarcted, as confirmed by diffusion restriction or hypointensity on apparent diffusion coefficient (Figure 3) and hyperintensity on diffusion weighted imaging on the same MRI scan (not shown).

Although the concurrent magnetic resonance angiography demonstrated widespread vasospasm in the intracranial arteries, the negative screens for extractable nuclear antibodies and lack of evidence for systemic vasculopathy indicate that the diagnosis of postpartum vasculopathy or vasculitis was less likely. Nevertheless, nimodipine was given because of cerebral infarction, having been shown to reduce vasospasm and the rate of infarction in circumstances such as subarachnoid haemorrhage¹². Incidentally, nimodipine would have also controlled PRES, because the mainstay of PRES treatment (after treating the underlying condition) is control of hypertension, even though most patients only have moderately elevated blood pressure⁵.

Adding further weight to the diagnosis of PRES was the day five electroencephalograph findings, which were consistent with an underlying posterior lesion rather than a generalised eclamptic seizure, and the resolution of vasospasm of all intracranial arteries noted on MRI at three months.

The diagnosis of late postpartum eclampsia with PRES syndrome, evolving into acute stroke, seems reasonable but other contributions, like the double dural puncture with a large needle, the concurrent use of caffeine and the large volume epidural blood patch need discussion.

Can seizures occur due to dural puncture? In 1995 Shearer et al¹³ published a study of 19,000 puerperal women undergoing subarachnoid analgesia for delivery or puerperal sterilisation. Of these, eight women developed new-onset seizures associated with postdural puncture headache (incidence 1:2375 for all obstetric spinals, increasing to 1:100 if a postdural puncture headache developed). The authors postulated that the seizures were caused by cerebrospinal fluid hypotension and cerebral shift, provoking cerebral vasospasm. Interestingly, two cases described had occipital MRI hyperintensities,

raising the possibility that they had undiagnosed PRES.

One case report⁶ attributed postdural puncture headache as the cause of a PRES. MRI images post-spinal puncture showed characteristic findings of PRES, a situation analogous to our patient. The mechanism proposed was diminished cerebrospinal fluid volume causing collapse of the ventricle, with mechanical stimulation by arterial wall traction inducing vasospasm. There is an association between dural punctures and seizures, as well as PRES, but the evidence is insufficient to implicate dural puncture as the cause in our patient.

Could the seizures have been due to caffeine? Caffeine is a central nervous system stimulant and a cerebral vasoconstrictor which has been reported to cause seizures in neonates, infants and adults¹⁴⁻¹⁷. There are three reported cases¹⁸ of puerperal seizures following the treatment of postdural puncture headache with synthetic adrenocorticotrophic hormone and sumatriptan, both of which exhibit cerebral vasoconstrictive activity. However, in these cases there are many confounders such that the specific aetiology is unclear. The 100 mg twice daily dose of oral caffeine received by our patient represents a relatively small dose (equivalent to two to four cups of coffee in a day) and the doses that have been recommended to treat postdural puncture headache are 300 to 500 mg of oral or IV caffeine, once or twice daily¹⁹⁻²¹. Cases of seizures reported in association with caffeine use involve much higher doses of caffeine, such as 500 mg to 1 g IV over three hours^{22,23}, or 200 mg four times a day orally¹⁴. It seems unlikely that the seizures were solely due to the caffeine, but it may have contributed.

Could the epidural blood patch have contributed to cerebral ischaemia? Mercieri et al²⁴ speculated that cerebral vasospasm may follow dural puncture or follow an epidural blood patch. Subdural and extradural haematomas are known to be powerful stimuli of cerebral vasoconstriction²⁵ but theirs is the only case report attributing epidural blood patch as the cause of ischaemia. Could the large volume of blood used in our patient have magnified the physiological effects of the epidural blood patch, including potentially the degree of cerebral vasoconstriction? While plausible, there is no proven link between epidural blood patch and stroke, a very rare occurrence, and it is known that cerebral infarction is already up to 13 times more frequent in puerperal women compared with non-pregnant women^{24,26}.

CONCLUSION

This case highlights the diagnostic difficulties when seizure and stroke occur in the postpartum period and a number of potential contributors are usually present. Despite the possible role of dural puncture, caffeine and an epidural blood patch, and although this patient did not have a thrombophilia screen, the diagnosis of late postpartum eclampsia with a posterior reversible encephalopathy syndrome progressing to a cerebral infarct appears to be the most likely cause, based on the clinical and radiological findings.

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Errata

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Incidence of difficult intubation in intensive care patients: analysis of contributing factors

JF. Heuer, TA. Crozier, J. Barwing, SG. Russo, E. Bleckmann, M. Quintel, O. Möerer

The seventh author's name should read O. Moerer.

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End-of-life practices in a tertiary intensive care unit in Saudi Arabia

Aldawood AS, Alsultan M, Arabi YM, Baharoon SA, Al-Qahtani M, Haddad SH, Al-Dorzi HM, Jahdali HA, Alatassi A, Rishu AH

The fifth author's name should read S. Al-Qahtani and the eighth author's name should read H. Al-Jahdali.

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Determinants of family satisfaction in the intensive care unit

Sundararajan K, Sullivan TS, Chapman M

The second author's name should read T. R. Sullivan.