

CASE REPORT : POSTPARTUM REVERSIBLE CEREBRAL VASOCONSTRICTION SYNDROME IN THE CONTEXT OF PREECLAMPSIA

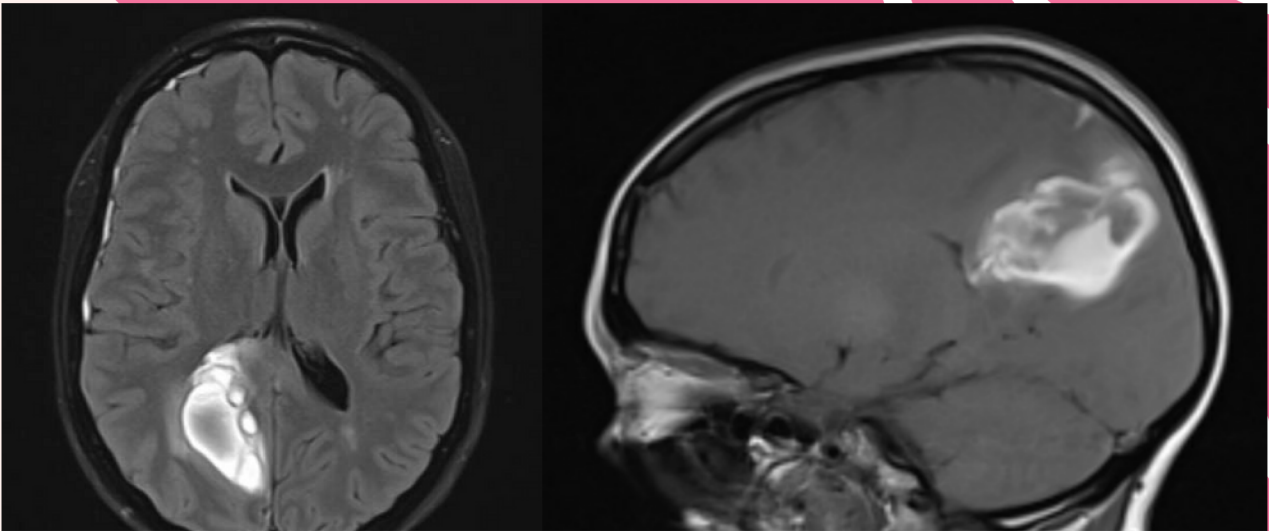


DR HANANI BAHRUDIN¹ , A/PROF DANIEL WARDMAN², DR MURAD ALRABADI¹, DR IAN FULCHER¹ , & PROF ANGELA MAKRIS³ .

1. Obstetrics & Gynaecology Department, Liverpool Hospital 2. Conjoint Associate Professor of UNSW Liverpool Clinical School & Senior Staff Specialist Neurologist 3. Renal & Obstetric Medicine Specialist, Liverpool Hospital

BACKGROUND

Preeclampsia (PET) is a hypertensive disease with potential cerebrovascular complications such as hemorrhagic and ischemic stroke, posterior reversible encephalopathy syndrome (PRES), reversible cerebral vasoconstriction syndrome (RCVS), cerebral small vessel disease, and vascular dementia^[1,2,4]. We report a case of reversible cerebral vasoconstriction syndrome in the postpartum period, with radiological findings of intracranial haemorrhagic stroke secondary to cerebral vasospasm.



MRI Brain of patient revealing a right sided parietal haemorrhage and right subdural haematoma

CASE REPORT

Antenatal / Delivery	We report a case of a 32 year old P2 with who had an uncomplicated antenatal course and delivered via a repeat Caesarean Section. She recovered well post operatively, was normotensive throughout pregnancy and was otherwise well with no other medical history.
1st presentation 7 days postpartum	She was admitted from the emergency department 5 days postpartum due to a new diagnosis of late onset preeclampsia. She presented with bilateral upper neck discomfort and a sudden onset generalised headache, hypertension up to 170/100 mmHg and proteinuria (PCR of 230mg/mmol). She required IV Labetalol, IV Hydralazine, PO Nifedipine IR in the emergency department. She otherwise examined normally without focal neurological signs, or clinical signs of preeclampsia such as clonus, or hyperreflexia. Her investigations revealed new liver derangements with a normal abdominal ultrasound. Her symptoms improved following blood pressure control with antihypertensives, thus a CT Brain was not pursued during that admission. She was discharged home on PO Enalapril twice daily with a plan for antihypertensives titration and a Renal Outpatient follow up, as well as 6 weeks of Enoxaparin for VTE prophylaxis. She was otherwise not on any other regular medications.
2nd presentation 2 weeks postpartum	She represented 1 week following her discharge with ongoing headache, blurred vision and new onset altered depth perception, resulting in clumsiness and bumping into walls and objects. She was normotensive on presentation with a normal neurological examination. However, her CT Brain revealed a subacute 4 x 2 x 3 cm right parietal intracerebral haematoma with an early midline shift as well as a subdural haematoma. Her CT Brain Venogram and Angiogram was normal without evidence of cerebral venous sinus thrombosis, arterial dissections or stenoses. Her MRI Brain revealed that the changes are consistent with RCVS. The impression was that her right parietal haematoma was secondary to cerebral vasospasm in the context of her preeclampsia, likely consistent with RCVS. Other potential causes of her intracranial pathology was excluded with a normal vasculitis screen and a normal transthoracic echocardiogram. There was also no possible iatrogenic causes of her symptoms found. During her admission, she had multidisciplinary care involving the neurosurgeons, neurologist, ophthalmologist, cardiologist and the obstetric medicine team. She was discharged following monitoring and physiotherapy, with a plan for progress imaging 3 months postpartum.
3 months follow up	Her progress MRI showed residual gliosis but no further haemorrhage. She has been asymptomatic since discharge and her vision has returned to baseline. She remained normotensive and was completely weaned off her antihypertensives. She was not planning for further pregnancies, but should she fall pregnant she was advised on preeclampsia prophylaxis such as aspirin and calcium.

CONCLUSION

Preeclampsia is known to have significant cardiovascular and cerebrovascular complications, such as RCVS. RCVS causes vasospasm of the cerebral arteries and occurs most often in the postpartum period and can be complicated by ischaemic or haemorrhagic stroke^[4]. While headache is a symptom of preeclampsia, patients should be evaluated for other intracranial conditions, as early recognition of intracranial pathologies such as ischemic or haemorrhagic stroke or RCVS may be life-saving. Although based on little evidence, calcium channel blockers are prescribed in RCVS as well as oral magnesium to prevent recurrent headaches following RCVS^[3]. Future studies are needed to determine whether it should be considered as a first-line agent for RCVS in the postpartum period. Given that the patient’s blood pressure was controlled, she did not have any intravenous prophylactic magnesium infusion, which is usually prescribed to prevent progression to eclampsia while managing hypertension crisis in preeclampsia, especially in the antepartum period. It is difficult to ascertain whether or not receiving intravenous magnesium could have changed her outcomes in this situation.

REFERENCES

1. Lee, D. E., Krishnan, A., & Collins, R. (2023). Reversible cerebral vasoconstriction syndrome in the postpartum period: A case report and review of the literature. *International Journal of Gynecology & Obstetrics*, 162(3), 823–828. <https://doi.org/10.1002/ijgo.14756>
2. McDermott, M., Miller, E. C., Rundek, T., Hurn, P. D., & Bushnell, C. D. (2018). Preeclampsia: Association with Posterior Reversible Encephalopathy Syndrome and Stroke. *Stroke*, 49(3), 524–530. <https://doi.org/10.1161/strokeaha.117.018416>
3. Roth J, & Deck G. (2019). Neurovascular disorders in pregnancy: A review. *Obstet Med*. 2019 Dec;12(4):164–167. doi: 10.1177/1753495X19825699. Epub 2019 Mar 21. PMID: 31853255; PMCID: PMC6909296.
4. Skeik, N., Porten, B. R., Kadkhodayan, Y., McDonald, W., & Lahham, F. (2015). Postpartum reversible cerebral vasoconstriction syndrome: Review and analysis of the current data. *Vascular Medicine*, 20(3), 256–265. <https://doi.org/10.1177/1358863x14567976>