

Authors: Dr M Vyas (ST5 O&G)
Dr N Navaneetham (Consultant O&G)

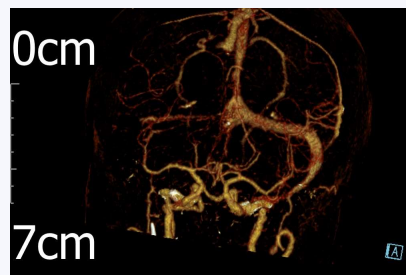
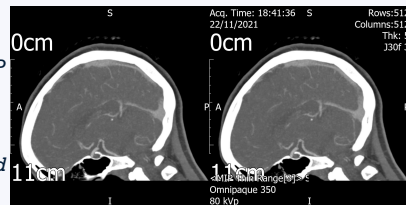
Sequela of severe hypertension in post natal women

Case Study: 29 y/o lady with a low risk pregnancy underwent Kiwi delivery for pathological CTG. Both her parents were treated for hypertension, however booking BP and BP throughout intrapartum period and postnatal period was within range. She presented with severe hypertension 11 days post-partum along with nephrotic range proteinuria. BP 188/016mmHg, uPCR 1074. Associated with sudden onset frontal and retro-orbital headache and photophobia. Commenced on magnesium sulphate infusion and oral antihypertensives.

CT scan showed left parietal lobe and insular sulci subarachnoid haemorrhage. GCS remained 15/15 throughout. Neurosurgeons advised Nimodipine 60mg 4 hourly, fluid resuscitation and bedrest. CT angiogram did not identify any intracranial aneurysms. Treated aggressively with antihypertensives in tertiary unit.

Cerebral angiogram did not show any evidence of intracranial aneurysms, AVMs or AV fistulas, however there were multiple short segment stenoses of the small cortical arteries bilaterally. This was suggestive of a diffuse vasospasm process such as reversible cerebral vasoconstriction syndrome (RCVS) or posterior reversible encephalopathy syndrome (PRES).

There was also tortuous remodelling of the vertebral, basilar and internal carotid arteries suggestive of chronic hypertension.



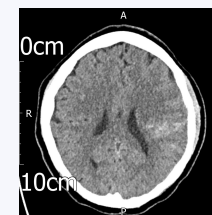
Discussion: Severe postnatal hypertension can result in severe morbidity in young women. Us as obstetricians are well versed with the management of eclampsia and pre-eclampsia, however with patients getting more complicated, one must always assess for other differential diagnoses; especially when a patient presents with headache and signs of meningeal irritation.

Acute subarachnoid haemorrhage is a medical emergency, in need of prompt treatment such as with calcium channel blockers, to prevent ischaemic neurological defects. In this case, this had to be cautiously balanced with severe pre-eclampsia and risk of pulmonary oedema.

RCVS presents as thunderclap headache and imaging can show watershed infarcts associated with vasospasm, vascular narrowing and beading. These findings are completely reversible within 4-8 weeks. There is no clear approach to the optimal treatment of RCVS. The vasospasm can be treated with calcium channel blockers however this was lead to vascular steal phenomenon which can potentially cause harm. Although features of RCVS are not true vasculitis, some cases have been empirically treated with high dose glucocorticoids for a month, followed by tapering of dose over several weeks.

PRES is a well-known complication of pre-eclampsia. Hypertension and endothelial injury causes vasoconstriction which results in vasogenic and cytotoxic oedema. Severe PRES can present with subarachnoid haemorrhage. RCVS can be a precipitant of PRES. High index of suspicion and prompt antihypertensive treatment, expedition of delivery can help reduce morbidity, mortality and aid in early recovery

Conclusions: There should be a low threshold for intracranial imaging in cases of severe hypertension. Multidisciplinary team involvement early on, improves outcome and optimises management of complex patients. Aggressive treatment of blood pressure is necessary to reduce morbidity and mortality, and long term complications. Close follow up after the resolution of initial presentation is advised. These women should be offered pre-conception counselling and close management of their next pregnancy in a medical obstetric antenatal clinic.



References:

The Systemic Vasculitides ; John H. Stone; Goldman-Cecil Medicine, 254, 1750-1758.e2
Reversible cerebral vasoconstriction syndrome; Anne Ducros Dr; Lancet Neurology, The, 2012-10-01, Volume 11, Issue 10, Pages 906-917, Copyright © 2012 Elsevier Ltd

