# A CASE OF POST-PARTUM ANGIOPATHY

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## Background:

A 38 yo woman presented 7 days after cesarean section, complicated by pre-eclampsia, after she was witnessed to collapse at home and have tonic/clonic activity.

## Clinical Course:

Initially, the patient was given an IV bolus of 4g magnesium, a loading dose of phenytoin and her trachea was intubated for airway protection. Her head CT was normal, initial blood pressure was 170/100 and CSF studies were normal. She was transferred to our neuroscience critical care unit where the brain MRI showed bilateral, scattered infarcts in the posterior circulation. Her initial neurological exam consisted of GCS 1/t/2 with sluggishly reactive 3mm pupils, no blink to threat, and flickering extension in all 4 extremities. Angiography on hospital day 4 revealed Rt MCA/Rt ACA/Rt PCA/Lt proximal superior cerebellar artery constriction and intra-arterial (IA) verapamil was injected into these fields. She went for additional IA verapamil treatments on hospital day 10 and 17 where there was improvement of vasoconstriction from initial imaging. Final physical exam had the patient giving thumbs up, wiggling toes bilaterally, shaking head yes/no, and mouthing words. She appeared to have persistent right pupillary dilation, could follow with all 4 extremities, but had left arm spasticity (likely due to her thalamic infarcts) and chorea-like movement with the right arm being extended and abducted. She was discharged to an LTAC for further rehabilitation.

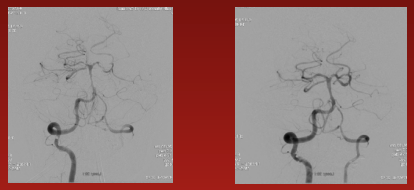

**Figure 1:** Pre and post-verapamil treatment

## Discussion:

Post-partum angiopathy (PPA), a variant of reversible cerebral vasoconstriction syndrome (RCVS), usually occurs within 1-3 weeks post-partum and presents with thunderclap headaches, a constellation of neurological signs based on affected regions of the brain, seizures, and ischemic stroke. Brain imaging is normal in approximately 70% of patients with RCVS while others have border-zone ischemic strokes, parenchymal hemorrhage, vasogenic edema, and non-aneurysmal SAH.

Even though it is thought vasoconstriction resolves within 3 months, it was determined with this patient that intervention was needed to prevent further neurological complications. The decision to administer IA verapamil was based on clinical exam, TCDs, and imaging. The mechanism of action of PPA is not quite understood at this point. Recent studies have shown placental growth factor (P1GF), soluble P1GF receptor and soluble endoglin correlate with the presence of eclampsia and prediction of its development, but it is unsure of whether these play a true role in post-partum angiopathy. Overall, goals of management were MAP ~ 100, SBP > 160 mmHg, [Mg] > 2.5mg/dL, ASA 325 mg (spasm antiplatelet affect), and verapamil 80 mg TID. The risk with repeated angiographies and IA verapamil included hemorrhagic transformation of brainstem strokes, but the risk of a disabling stroke without treatment, such as further pontine strokes which could lead to a locked in state, was deemed to outweigh the risk/ Thus multiple doses of IA verapamil were deemed to be the appropriate choice to give the patient a chance at a reasonable recovery.

## References:

1. Singhal, A.B., et al (2009) NEJM; 360:1126-1137
2. Chen (2010) Theory of Advanced Neurological disorders 3(3): 161-171