

Posterior Reversible Encephalopathy Syndrome in Severe Pre-eclampsia

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Abstract

Pre-eclampsia is an unpredictable, multiorgan disorder unique to human pregnancy. Focal neurological deficits in a preeclamptic woman must be rapidly assessed and treated to minimize damage to both mother and foetus. We report a case of antenatal mother presenting with headache, visual loss, mental status changes and severe preeclampsia, diagnosed as posterior reversible encephalopathy syndrome. This syndrome manifests as symmetric bilateral hyperintensities of parieto-occipital white matter in T2 weighted images on MRI and is usually reversible with prompt treatment.

Introduction

Posterior reversible encephalopathy syndrome (PRES) is a recently recognized syndrome characterized, clinically, by headache, confusion, seizure and visual loss associated with imaging findings of bilateral cortical and subcortical oedema.¹ Since its first description in 1996, PRES has been reported to occur in a number of clinical conditions characterized by either an acute elevation of blood pressure (BP) or treatment with certain drugs.^{2,3}

Imaging modalities demonstrate symmetric lesions, usually of posterior white and gray matter. Prompt treatment of hypertension or removal of offending drug is found to be critical in ensuring reversibility of deficits.^{4,5} The following case report features a case of preeclampsia that developed PRES.

Case Report

A 25 year old gravida 4, abortion 3 was admitted with complaints of headache and pedal oedema at 31 weeks of gestation. She had no significant past medical

history. Her only medication was prenatal vitamins and had no known drug allergies. Patient was apparently asymptomatic 10 days back when she noticed gradually increasing bilateral pedal oedema. Patient complained of severe throbbing headache over last two hours. On presentation, patient was conscious and coherent, with a arterial BP of 160/120 mmHg, respiration 18 breaths/min. Her cardiopulmonary examination was normal. She had pedal oedema 3+, proteinuria 4+ and normal deep tendon reflexes.



Fig. 1 : MRI showing hyperintensities in occipitoparietal white matter, etc. on T2 weighted image.

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Examination revealed a 32 weeks relaxed uterus with a normal foetal heart rate. She was started on antihypertensives. IV access was secured and blood was sent for investigations. An ophthalmic examination revealed vasospasm with no evidence of papilloedema. Liver and renal profiles were within normal limits with Hb 12.9 gm% and platelet count of 2.9 lakh/cumm.

In the meanwhile, patient developed severe epigastric pain, lethargy, somnolence, disorientation and visual loss over an hour. Patient was started on IV Labetalol to stabilize her arterial BP. In light of her acute neurologic deterioration an urgent MRI was done. MRI demonstrated T2-weighted hyperintensities in bilateral occipitoparietal white matter, posterior frontal lobe, bilateral thalami, posterior limb of internal capsule. There was no abnormalities of ventricular system and no evidence of midline shift or mass effect. The neuroradiologist made a diagnosis of PRES.

Patient was immediately proceeded to operating room for a caesarean delivery. The patient's arterial BP decreased after induction of anaesthesia from 158/118 to 100/80 mmHg. Patient was shifted to medical intensive care unit and treated with supportive therapy directed by the cardiology team. Patient regained her vision on postoperative day 1. She was oriented and coherent, without any recollection of the previous day events. Postoperative recovery was uneventful and patient was discharged with baby in her arms.

Discussion

A preeclamptic patient presenting with acute mental status changes and visual loss suggest a broad differential diagnosis. In face of severity of the hypertension and loss of vision, intracranial haemorrhage was of greater concern and prompted an urgent MRI. Distinguishing between thromboembolic stroke and haemorrhagic stroke is paramount when considering treatment of arterial BP.

PRES is a recently recognized syndrome, typically presents with symptoms of headache, visual changes, seizures and mental changes. PRES is associated with an acute increase in arterial BP, acute renal failure and immunosuppressive or cytotoxic drugs (such

as methylprednisolone, cyclosporine, tacrolimus). Treatment of arterial BP or withdrawal of the offending drug(s) is treatment of choice.

Appropriate therapy usually results in complete resolution of the deficits over several days to weeks, although partial resolution has been reported and the disease can be fatal.

Lesions are generally bilateral and parieto-occipital, but may involve temporal or frontal lobes, brainstem or cerebellum. T2 weighted MRI shows areas of hyperintense signal and is thought to capture the images with best quality,⁶ but fluid attenuated inversion recovery (FLAIR) sequences may improve detection of cortical / subcortical areas of injury.⁷ With rapid improvement in the patient's clinical status, a subsequent MRI was deemed unnecessary, as clinical resolution corresponds with radiographic resolution.

The pathophysiology of PRES is most likely, and most often reported as, vasogenic oedema secondary to an acute increase in arterial blood pressure, which overwhelms the autoregulatory capacity of the cerebral vasculature, causing arteriolar vasodilation and endothelial dysfunction, leading to interstitial extravasation of fluid. The posterior circulation is thought to be more susceptible to this type of damage, because there is less sympathetic innervation of the ventrobasilar vasculature to protect the parenchyma from rapid increases in arterial blood pressure.^{6,8,9}

Conclusion

Acute focal neurologic changes should prompt rapid investigations, including imaging of the brain. The characteristic findings on MRI helps to differentiate PRES from other conditions like intracranial bleeding and infarct, which require different

line of management. PRES, especially in patient presenting with typical symptoms of headache, seizures, visual deficits and mental changes should be considered and treated without delay to maximize the potential for reversibility.

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SHOULD THE CD4 THRESHOLD FOR STARTING ART BE RAISED?

Current British and American guidelines recommend that, in the absence of an AIDS –defining illness, ART should be started in patients with blood CD4 cell counts in the range 200-350 cells per μL . It might be time for the pendulum to swing once more towards earlier treatment.

Frequency of death, or combined AIDS and death, in patients receiving and not receiving ART was used to identify a minimum threshold of 350 cells per μL for starting ART.

Those who deferred treatment had a far higher rate of major morbidity and all-cause mortality than did those treated immediately.

At high CD4 cell counts, differences in absolute risk of AIDS and death between early and deferred ART are small and uncertainty about the risk to benefit ratio remains. Even when benefits outweigh risks, cost-effectiveness is unclear. Data are needed on serious complications of ART that might negate the benefits, such as cardiovascular, renal and hepatic disease.

When considering both high-income and resource-limited settings, the question of when to start ART might have more than one right answer. WHO guidelines for resource-limited settings currently recommend initiation of ART before blood CD4 cell counts fall below 200 cells per μL with an upper threshold of 350 cells per μL .

Robin Wood, Stephen D Lawn, The Lancet, 2009; 373 : 1315-16.