

Case Report

CORTICAL BLINDNESS IN PREECLAMPSIA PATIENT AFTER A CESAREAN DELIVERY A CASE REPORT AT FMH

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Abstract: The three most common visual complications of preeclampsia and eclampsia are hypertensive retinopathy, exudative retinal detachment and cortical blindness. Preeclampsia-eclampsia can have neurological sequelae; cortical blindness is one of these. We report case of parturient with mild preeclampsia that subsequently developed transient blindness.

Case Report

A 28 yrs old primigravida booked patient with poor compliance and twin pregnancy presented at 37+5 wks with c/o watery vaginal discharge. On examination her BP was 120/80 mm/Hg, SFH was 39cm. Presented twin was breech, vaginal leaking was confirmed. Baseline investigations were carried out.

1 unit of blood was arranged and category 3 C. Section carried out in spinal anesthesia. There was an episode of transient hypotension after spinal anesthesia which was successfully managed in 3 minutes. First episode of raised BP was noted at the end of c-section on table i.e. Up to 150/100 mm Hg but she was oriented throughout the period. 17 hrs post operatively she complained of blindness. Her BP was 140/90. 1 hour later she became disoriented, agitated and started irrelevant and incomprehensible talk. Eye examination revealed normal disc, macula, blood vessels and no evidence of disc swelling. Psychiatry department advised sedatives. MRI brain was advised by neurologist but pt was not stable enough to leave hospital. BP was persistently raised but not more than 140/90 mmHg. There was +proteinuria and uric acid in post operative period was 6.6mg/dl. She was given intravenous sedatives to control her agitated state in order to carry out CT scan. CT scan showed bilateral areas of white matter hypo density involving occipital region suggestive of acute hypertensive encephalopathy syndrome, mild sulcal effacement suggestive of brain edema. Gradually she became unstable to maintain her oxygen saturation so was ventilated. Inj mannitol 100cc i/v BD given. On 4th post op day, she was weaned off ventilator. Agitation settled, blindness resolved. She was shifted on oral anti hypertensive medication and later discharged on 7th post op day discharged without any antihypertensives.

Discussion

Cortical blindness signifies a loss of vision caused by lesion of geniculocalcarian visual pathways in the

posterior hemispheres.² Cortical blindness has also been reported as a complication of preeclampsia-eclampsia^{3,6} with an incidence as much as 15% in woman with eclampsia⁵. but rarely with otherwise mild preeclampsia. This case report describes a confusing scenario caused by the overlapping clinical and radiological features of preeclampsia and hypotension associated with watershed infarcts, subsequently, subtle radiologic clues in her clinical course helped to establish the diagnosis of preeclampsia induced cortical blindness. A differential diagnosis included puerperal psychosis, symptomatic pre-eclampsia, hypertensive encephalopathy or spinal induced hypoperfusion-related watershed infarcts however these conditions differ in their associated radiologic findings, pathophysiology and natural history.

Bilateral focal to diffuse low density lesion on CT and hyperintense lesion on T2 weighted MRI images are the most common neuroradiologic abnormalities in pt with severe preeclampsia-eclampsia. These changes represent focal increases in brain water contents^{3,5,7}. In severe cases radiologic evidence of gross or petechial hemorrhages may also be present^{8,9}. Hypotension related watershed infarcts demonstrate the same type of edematous changes (hypo density on CT & hyper intensity on T2 weighted MRI) but in very specific "distal field" territories of the middle cerebral artery and posterior cerebral artery.¹⁰ The cause of these focal increase in brain water is different in hypotension related watershed infarcts and preeclampsia-eclampsia. In preeclampsia-eclampsia there is hyper perfusion and loss of normal protective autoregulation (break through) resulting in forced vasodilatation, passive over distention and vasogenic extracellular edema. Ischemia and infarction secondary to hypoperfusion lead to cytotoxic edema in border zone infarcts in this case sodium and water enter damaged neurons producing intracellular edema.¹¹ The prognosis for cortical blindness depends on etiology. Cortical blindness associated with preeclampsia-eclampsia almost always resolves^{5,11} further supporting in their theory the radiologic lesions represent vasogenic edema and are not caused

by vasospasm and ischemia. Bilateral occipital abnormalities caused by hypotension are associated with poor prognosis.^{2,10} In conclusion health care providers should draw lesson from this experience when facing similar cases of blindness complicating pregnancy or its management and should keep the

differential diagnosis in mind because of very different prognosis.

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