

Case Report

Two cases of visual impairment associated with preeclampsia

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Case 1

Cortical Blindness (Posterior Reversible Encephalopathy Syndrome)

A 20 year old primigravida was referred to our hospital at 35 weeks of gestation with severe pregnancy included hypertension with chief complaints of sudden loss of vision. On examination she had pedal edema and a BP of 170/110 mm hg. She had absent far and near vision. Her pupils were normal in size and reacting to light. Fundus examination revealed findings within normal limits and there was no evidence of any retinal detachment.

Sine she was already in labor and 3 cm dilated, an artificial rupture of membranes was performed and labor was augmented as retinal detachment was ruled out by fundus examination. The BP was controlled with use of antihypertensives and the patient was also started on prophylactic magnesium sulphate regime. The patient delivered normally uneventfully after 6 hours of labor.

Postnatally a contrast computed tomography scan was performed which showed bilateral symmetrical parietooccipital white mater hypo-densities. Her vision had started improving right from the 1st postnatal day. In view of the above and associated severe PET the findings were opined to represent 'Posterior Reversible Encephalopathy Syndrome' secondary to severe preeclampsia.

The patient regained vision completely by the 3rd postnatal day and was later discharged on treatment with antihypertensives.

Discussion

Cortical blindness associated with preeclampsia-eclampsia results from petechial hemorrhages and focal edema in the occipital cortex. These lesions are likely to be stimulated by disparity in cerebral regional blood flow that is characterized by vasospasm and diminished flow primarily affecting the posterior circulation¹. The ocular findings in eclampsias are fairly common and they are those of hypertensive retinopathy. However, the cerebral complications are not as common, and less common is reversible cortical blindness following eclampsia. Hinchey J et al ², between 1988 and 1994, found only three cases that had reversible cortical blindness following eclampsia. They also pointed out that neuroimaging showed the findings to be consistent with those of subcortical edema without infarction, and

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the cortical blindness reversed in two weeks following antihypertensive therapy. Some other studies suggest vascular endothelial damage as the underlying mechanism in this case of preeclampsia related transient cortical blindness³. Sometimes residual cerebral damage may remain shown by persistent electroencephalogram abnormalities and transient episodes of blindness as depicted by Grimes et al⁴. It has also been shown that RPLS can occur in pregnancy without preeclampsia. Two hypotheses are conceived to explain the emergence of RPLS without hypertension. The first suggests that an immunotolerant condition such as pregnancy can easily cause vasogenic edema without the elevation of blood pressure. The second suggests that hypertension exists but cannot be detected because it is extremely acute and transient⁵.

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Case 2. Central serous retinopathy

A 25 year old primigravida at 37 weeks was referred to our institution with preeclamptic toxemia on treatment with antihypertensives and complaining of unilateral central loss of vision only in the right eye. She did not have any other symptoms suggesting an imminent eclampsia and her blood pressure was within reasonable control. All her blood investigations were within normal limits. Fundus examination revealed findings suggestive of central serous retinopathy. As there was no evidence of any retinal detachment the

patient had an induction of labor and subsequently delivered uneventfully. The patient was later referred to ophthalmology for visual acuity and other tests. After discharge the patient followed up and she slowly had a complete vision recovery in around 2 months. This is the first case of preeclampsia encountered by us in our institution associated with central serous retinopathy. The case is presented because of the rarity of the two conditions being associated.

Discussion

Central serous retinopathy is usually seen as an idiopathic, self limiting, non inflammatory leakage of fluid in the central macula, which results in blurred or distorted vision (metamorphopsia). A blind or gray spot in the central vision is common, along with flashes of light (photopsia). The leakage, of unknown origin is through the retinal pigment epithelium. Ocular changes during pregnancy predispose the eye to pathologic conditions including hypertensive and vascular disorders, central serous chorioretinopathy, uveal melanoma and others¹. Diagnosis of serous neuroretinal detachments of the macula in severe preeclamptic patients with optical coherence tomography have been reported². Some authors have described peculiar choroidal lesions associated with central serous chorioretinopathy (CSC) in pregnancy and ischemic exudative choroidal lesions during severe toxemia³. A retinal pigment epithelium (RPE) tear which presumably followed a RPE detachment (PED) was described in the macular region in one study³. Disease is mostly self limiting and vision improves in 2 to 3 months in most of the cases. No medical treatment is proved to be of any help. Steroids are contraindicated. Only treatment is laser treatment of the leaks.

References

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