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## Reversible blindness in severe preeclampsia: A case report

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#### Abstract

Visual disturbances are common with severe preeclampsia, however, blindness either alone or accompanying convulsions is uncommon. Most women with varying degree of amaurosis are found to have radiographic evidence of extensive occipital lobe hypodensities. In this case report, we present the clinical course of two women in whom preeclampsia was further complicated by blindness. These were managed by standard pre-eclampsia regimen used at our institute.

**Keywords:** Reversible blindness, severe preeclampsia, convulsions is uncommon

#### Introduction

Preeclampsia remains one of the leading causes of maternal and perinatal mortality and morbidity. Such patients may present with headache and visual disturbances in the terms of scotoma, sudden inability to focus, blurred vision, and in severe cases to complete blindness. Although visual disturbances develop in perhaps 25% of women with severe pre-eclampsia, blindness is rare and an incidence of 1-3% is reported with eclampsia [1]. In past most cases of blindness were attributed to retinal abnormalities that include edema, vascular changes and detachment. Retinal detachment may cause altered vision although it is usually one sided and seldom causes total visual loss [2].

In this case report, we present the clinical course of a woman in whom preeclampsia was further complicated by blindness. The woman was managed by standard pre-eclampsia-regimen used at our institute which included inj. Labetalol and magnesium sulfate, nifedipine to lower elevated blood pressure, and intravenous fluid restriction and termination of pregnancy. Abnormal findings in these women were observed by ultrasound and Doppler techniques, and blood investigations for pre-eclampsia, and fundus examination for visual defects.

#### Case report

A 36-year-old multigravida presented with headache, hypertension (190/110 mmHg), and generalized edema in the  $36^{th}$  +2 week of pregnancy. She had headache, blurring of vision, and epigastric pain for the last 5 days. There was no previous history of diabetes, hypertension, thyroid disorders, and seizure disorders. There was no family history of such medical/surgical significant illness.

Her per abdomen examination revealed fetal heart rate of 140 beats/min regular which was present on left spino-umblical line.

On admission, investigations were done. Complete hemogram was normal except platelets count of 96000/mm³. LDH was 660 U/L, serum aspartate aminotransferases and Alanine transferases were 120 IU/L and 140 IU/L respectively, and serum creatine was 1.0 mg/dl. Urine examination showed proteinuria 2+. Fundus examination indicated cortical blindness likely resulting from vascular spasm and also bilateral edematous retinal detachments.

She was provisionally diagnosed with G2P1001 at PoG 36+2 weeks with severe pre-eclampsia. She was treated with inj. Labetalol to control her BP and inj magnesium sulfate started as per Prichard regime after Foley's catheterization of the patient. Her Bishop's score was poor (3); hence, the patient was planned for emergency caesarean section.

The visual impairment lasted 2 days, after which there was gradual resolution over the next 3 days. By the time of her discharge, after her initial admission, her visual impairment was completely resolved and the blood investigations had all returned to within normal prepregnancy values.

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#### Discussion

The neuropathologic mechanism for eclampsia is unclear except when overt hemorrhage is identified. Some factors that have been implicated including cerebral vasospasm, hemorrhage, ischemia, edema as well as hypertensive and metabolic encephalopathy. Recently Schwart *et al.* proposed that the findings of preeclampsia can be explained by the loss of autoregulation of the posterior cerebral circulation <sup>[3]</sup>. Aside from convulsions other dramatic neurologic effects, albeit uncommon, include blindness, an altered state of consciousness and coma. In the past most cases of blindness were attributed to retinal abnormalities that include edema, vascular changes and detachment. More recently case reports have emphasized cortical blindness, which is characterized by intact pupillary response and normal ophthalmoscopic findings.

The management guidelines are straightforward for women with severe preeclampsia or eclampsia in whom cortical blindness develops. Generally, they are the same as for women without this sequeale and include anticonvulsants, (Magnesium Sulfate, Phenytoin and Diazepam) for seizure prophylaxis, control of severe hypertension, and fluid restriction to avoid worsening of cerebral edema. Ophthalmologic and neurologic consultation along with neuroimaging is undertaken; however, delivery should not be delayed unnecessarily. On the basis of previously published experiences with computed tomography in women with eclampsia, as well as the experience described here, we conclude that reversible visual impairment associated with preeclampsia may result from petechial hemorrhages and focal edema in the cerebral cortex.

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