CASE REPORT

Reversible Blindness in Severe Preeclampsia and Eclampsia

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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- eclampsia regimen used at our hospital which includes Diazepam /Phenytoin therapy to control seizures, nifedipine to lower elevated blood pressure, intravenous fluid restriction and termination of pregnancy. Abnormal findings in these women were seen by computed tomography and magnetic resonance imaging technique. CT demonstrated low density areas localized predominantly in occipital area. Blindness persisted from 24 hrs to 72 hrs, it subsequently resolved completely in both.

Key words

Eclampsia, Preeclampsia, Blindness, Computed tomography, Magnetic resonance imaging.

Introduction

Severe preeclampsia and eclampsia remains one of the leading causes of maternal and perinatal mortality and morbidity in many parts of the world. Patients may present with symptoms of headache and visual disturbances in the form 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). More recently case reports have emphasized cortical blindness which is characterized by intact pupillary light reflexes and normal ophthalmoscopic findings.

Here we report the clinical course in two women in whom preeclampsia-eclampsia was further complicated by blindness. Abnormal findings in these women were seen by computed tomography and magnetic resonance imaging techniques. None of the patients had the diagnosis of seizure disorder, migraine headaches or collagen vascular disease.

Case Reports

Case 1— 20 yr old primigravida presented with headache, hypertension (160/100 mm of Hg) and generalized edema in the 38th week of pregnancy. Urine

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examination showed proteinuria 3+. Her serum creatinine was 1.2 mg/dl. Serum aspartate aminotransferase was 96u /lt. She was treated with nifedipine to control her BP and intravenous fluid administration was restricted to 60 ml /hr. Since she had good bishops score so labour was induced and augmented with oxytocin infusion. Soon after vaginal delivery she noted severe impairment of vision in both eyes. She was examined by an ophthalmologist and neurologist. Computed tomography scan was advised which showed low density area localized predominantly in the occipital lobe. Cortical blindness lasted for 24 hrs and was followed by complete recovery of vision by 7 days. Patient was normotensive postpartum.

Case 2— 23 yr old, para1 presented with post partum eclampsia. Her BP at the time of admission was 170/90 mm of Hg. Urine examination showed proteinuria 4+. Her serum creatinine was 0.8 mg/dl. Serum aspartate aminotransferase was 93u /lt. Computed tomography showed low density lesions in occipitoparietal area. (Fig.1). Repeat CT scans after one week showed partial resolution at one week. (Fig. 2) Follow up neurological studies 2 weeks later by which time the patient's visual acuity returned to normal showed complete resolution. Patient was normotensive 2 months postpartum. None of the patients had thrombocytopenia.

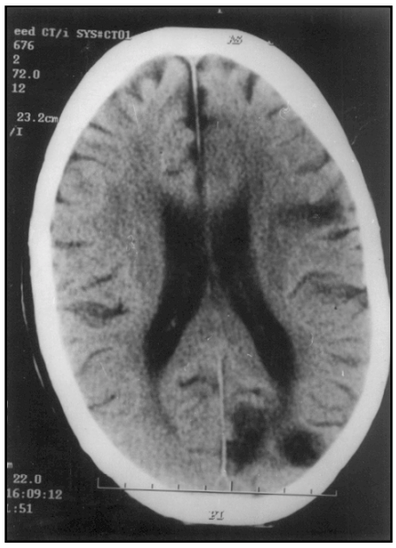

Fig. 1. CT Scan head shows hypodense lesion in rt. occipital lobe suggestive of ischemic changes.

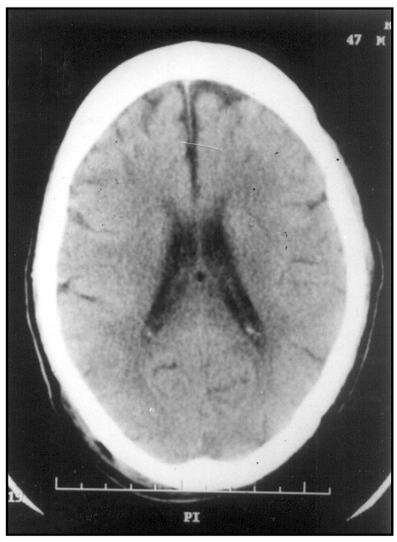

Fig. 1. CT Scan head shows resolution of the ischemic changes after two weeks.

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-eclampsia can be explained by the loss of autoregulation of the posterior cerebral circulation (3). 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.

In 1980, Grimes *et al* reported the first case in which computed tomographic scanning was used to demonstrate

reversible cortical lesion in a women with preeclampsia and temporary blindness (4, 5). Others have reported low density areas predominantly in the occipital lobes of women with blindness associated with severe preeclampsia or eclampsia. Radiologic findings have ranged from normal to documentation of wide spread low density areas. The latter are nonenhancing and have been attributed to localized areas of decreased perfusion associated with arterial spasm, infarction or cerebral edema (6). Computed Tomography in both of our cases with cortical blindness, showed similar occipital lobe hypodensities. Various authors have reported partial resolution of these radiologic hypodensities by 3 to 5 days with complete resolution within 14 days (7, 8). We observed a similar time course for their resolution and observed that clinical recovery precedes normalization of CT findings.

There is other evidence that these lesions are induced by vascular changes. Using doppler velocimetry, Williams and Mclean showed that cerebral blood flow velocity is increased in pregnancy-induced hypertension, suggesting an increased resistance to flow. Velocity increased even more in the immediate puerperium (9). Thus the nature and duration of these reversible focal neurological lesions and transient cortical blindness are in concert with the characteristic reversible pathophysiologic changes of preeclampsia-eclampsia (10).

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 cortical blindness associated with preeclampsia-eclampsia may result from petechial hemorrhages and focal edema in the cerebral cortex.

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