

Reversible Blindness in Severe Preeclampsia

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Abstract

Severe preeclampsia and eclampsia are predominant causes of maternal and perinatal morbidity and mortality in many parts of the world. The sudden loss of vision is less common and worrying effect of severe preeclampsia.¹

Involvement of occipital cortex or the retina leads to temporary blindness. However, it is reversible with return of vision within a week. We report a case of patient with severe preeclampsia followed by complete loss of vision. She had emergency C-Section. In the post-operative period vision started improving within 24 hours and completely recovered in one week.²

Introduction

Preeclampsia is a multisystem disorder, with incidence of 5–7%. In developed countries, it is one of the main cause of maternal and fetal morbidity and mortality. The etiopathogenesis of preeclampsia is still not completely understood, as many genetic, immunologic, and environmental factors are involved.³

The most common presenting symptoms are headache, epigastric pain, pedal oedema and visual disturbances. The scotoma, blurred vision and in severe cases blindness are main visual symptoms. In severe pre-eclampsia, nearly one quarter of women present with visual disturbances, as complete blindness is seen in only 1%-3% of cases. Now it is thought that cortical blindness is leading pathology in severe preeclampsia and eclampsia patients as compared to old theories of vascular abnormalities, oedema or detachment.^{3,4,5} We report a rare case of a patient with severe preeclampsia who developed reversible cortical blindness, underwent CS and vision recovered within a week.²

Case Report

Thirty years` old second gravida with no alive issue presented at 31+2 weeks of gestational amenorrhoea on January 2016 with c/o epigastric pain since night. She was booked at antenatal clinic at 29 weeks gestation. On presentation, her BP was 190/130 mmHg, urine albumin trace.

On examination marked pedal oedema was present, brisk reflexes. On abdominal examination, fundal height was 32 weeks size, longitudinal lie with cephalic presentation and normal foetal heart rate. On urine routine examination protein was in traces. Her serum biochemical examination showed Haemoglobin 8gm%, ALT 48 iu/ml, uric acid 6.9. Her platelet count and coagulation profile were within normal limits. On pelvic examination os was closed, Cervix was firm, posterior. She developed loss of vision within 30 minutes of presentation. BP was controlled with Inj Labetolol, MgSO₄ therapy started and in view of poor Bishop's score, severe pre-eclampsia, her emergency caesarean section was performed and baby boy delivered, weighing 1.4 kg with APGAR score of 6 and 7 at 1 and 5 minutes respectively. In postoperative period, her BP remained 160/110 to 180/120 mmHg despite oral antihypertensive medication. She became drowsy. Both pupils were reactive to light. MRI Brain was advised, which showed multiple basal infarcts in basal ganglia and external capsule. Intraparenchymal bleed in frontal and occipital lobe. Initially IV antihypertensive were given along with MgSO₄ therapy. Later on she was put on oral medication. Vision slowly improved with complete recovery of vision in 7 days. She was discharged on the 13th post-operative day with BP of 126/72 mm of Hg and 6/6 vision.

Discussion

Complications of preeclampsia include renal failure, hepatic failure, cerebral hemorrhage, intrauterine growth retardation, and fetal death. Proteinuria and hypertension are most common presentations of preeclampsia as the kidney is one of target organ of syndrome. The severe preeclampsia includes blood pressure $\geq 160/110$ mmHg, proteinuria ≥ 5 g/24 h, oliguria, lung edema, cerebral symptoms, epigastric pain, elevation of liver enzyme levels, microangiopathic hemolysis, thrombocytopenia, delayed fetal intrauterine growth, or oligohydramnios.

The hypertensive retinopathy, exudative retinal detachment and cortical blindness are three most common visual complications of severe preeclampsia. Bilateral damage to visual pathways leads to cortical blindness. Ophthalmic examination is usually unremarkable, including a normal pupillary light reflex. Only 15% of preeclampsia and eclampsia patient develop this complication. Cerebral edema may occur due to cerebral vasospasm caused by severe hypertension leading to cerebral ischemia and cytotoxic edema. The other theory is that sudden increase in blood pressure leads to loss of autoregulation, dilatation of cerebral arterioles and increase in vascular permeability, and vasogenic edema. Management of

preeclamptic patients who develop cortical blindness is the same as for women without this visual complication.

Gestational age, fetal wellbeing, and presenting complaints of patient are kept in view when deciding delivery or conservative management of patient. The goal in these patients is to minimize the end organ damage and prevent eclampsia

The aim of Antihypertensive medication is to prevent cerebrovascular complications. New-onset seizures, headache, and visual disturbances are main presenting features of PRES (posterior reversible encephalopathy) Syndrome. The other visual problems may be hemianopia and cortical blindness. The syndrome may not be completely reversible, is not always limited to the posterior regions, and may involve gray as well as white matter.^{3,4,5}

References

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Corrigendum

In volume 6(4)2016, page no 182-186, the citation (authors name) of the published article titled "An Audit of Caesarean Section in Fauji Foundation Hospital Rawalpindi" was typed as "Imran F, Alia N. Zaman Z", which was typo error. The exact citation of this published article should be read as:

Cite this article as: **Akhtar N, Hayat Z, Ashraf M. An Audit of Cesarean Section in Fauji Foundation Hospital Rawalpindi. J. Soc. Obstet. Gynaecol. Pak. 2016; Vol 6(4):182-186.**

The article citation was also corrected in online edition.