

## Case Report

# Reversible blindness in fulminating preeclampsia

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### Abstract

Severe preeclampsia and eclampsia are major causes of maternal and perinatal morbidity and mortality in many parts of the world. One of the uncommon effects of severe preeclampsia on the eye is sudden loss of vision. This blindness can be due to involvement of the occipital cortex or the retina. Although very alarming, this type of blindness is usually reversible following appropriate management of such patients. Here we present the case of a woman in whom fulminating preeclampsia was complicated by blindness. Fundoscopic findings were bilateral papillo edema and macular edema. She responded well to therapy, which included antihypertensives, seizure prophylaxis and operative delivery. Vision was restored by the third day.

**Keywords:** Blindness, cortical, morbidity, preeclampsia, retinopathy, reversible

### Résumé

Sévère preeclampsia et éclampsie sont des principales causes de morbidité maternelle et périnatale et de mortalité dans plusieurs régions du monde. L'un des effets rares de graves preeclampsia sur l'oeil est la perte soudaine de vision. Cet aveuglement peut être due à participation de l'occipital cortex ou de la rétine. Bien que très alarmante, cela Type de la cécité est généralement réversible suite à une gestion appropriée des ces patients. Ici nous présenter la preuve d'une femme qui fulminating preeclampsia a été compliquée par la cécité. Fundoscopic conclusions ont été papilloedema bilatéral et un œdème maculaire. Elle répond bien aux thérapie qui comprenait antihypertensives, prophylaxie de la saisie et conscient livraison. Vision a été restaurée par le troisième jour.

**Mots clés:** réversible, cortical, cécité, rétinopathie, morbidité, preeclampsia

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### Introduction

Severe preeclampsia and eclampsia remain one of the leading causes of maternal and perinatal morbidity and mortality in many parts of the world.<sup>[1]</sup> Visual symptoms in preeclampsia and eclampsia include photopsia, visual field defects, sudden inability to focus, blurred or decreased vision and, in severe cases, complete blindness.<sup>[1,2]</sup> The 3 most common visual complications of preeclampsia and eclampsia are hypertensive retinopathy, exudative retinal detachment and cortical blindness.<sup>[2]</sup>

Here we present a case of a woman in whom severe preeclampsia was complicated by blindness. Fundoscopic findings were bilateral papilloedema and macular edema. Neuroimaging studies were not carried out on our patient due to lack of such facilities at our centre.

### Case Report

A 30-year-old pregnant housewife presented to the Ophthalmology Department of our hospital at 41 weeks gestation complaining of sudden loss of vision in both eyes and severe frontal headache

of 2 weeks' duration. At the time of presentation, she had blood pressure of 200/150 mm Hg. Visual acuity test revealed perception of light with poor projection of light. The eyelids and lashes were normal. The conjunctiva was white; cornea, clear; and anterior chamber, calm, with normal depth. The pupil was mid-dilated with sluggish reaction. The lens was transparent. On fundoscopy, there were bilateral papilloedematous discs with macular edema. Extraocular motility was normal. Intraocular pressure was normal. She was then referred to the Obstetrics and Gynecology Department for urgent attention.

History revealed she was an unbooked gravida 3 para 2 with no previous personal or family history of hypertension, heart disease, diabetes or eye disease. The last time she had been in a hospital was 2 years earlier when she had an emergency cesarean section in her second pregnancy due to poor progress in labor. There was a history of twin birth in her first pregnancy. Otherwise, both previous pregnancies had been uneventful. The patient did not smoke; drink alcohol, coffee; or use illicit drugs. She had attained menarche at 14 years, her menses had been regular before pregnancy and she had never used contraceptives.

On physical examination, she was anxious, afebrile and not pale; she had bilateral pitting pedal edema to the level of the ankle, and urinalysis revealed proteinuria 2+. Her weight was 97 kg; and height, 158 cm (pre-pregnancy values were unknown), with a body-mass index of 39. Abdominal examination revealed a midline scar from the previous cesarean section and mild epigastric tenderness. Symphysio-fundal height was 35 cm, and a single fetus was palpated in longitudinal lie, cephalic presentation and right occipito-anterior position, with a regular fetal heart rate of 140 beats per minute. Repeat blood pressure was 240/170 mm Hg. Pelvic examination revealed a Bishop score of 5. A diagnosis of fulminating preeclampsia was made, and she was admitted to the eclamptic room. Intravenous (IV) hydralazine 10 mg and diazepam 10 mg were given. Her packed cell volume was 34%. The client and her husband were counseled on the need for immediate delivery, and informed consent was obtained for emergency cesarean section. She was delivered of a live male infant weighing 2.3 kg with Apgar scores of 4 and 8 in the first and fifth minutes, respectively. The baby was reviewed by the neonatologist. The immediate post-operative status of the patient was stable. On the morning after the cesarean section, the patient's blood pressure was found to be 200/120 mm Hg. This was controlled with administration of IV hydralazine and maintained with oral nifedipine and methyldopa. The patient then complained

of blurred vision in both eyes. This gradually improved, and she finally regained vision in both eyes on the third day. Visual acuity was 6/9 and 6+/9 in the right and left eye, respectively. Fundoscopy revealed resolving disc edema with clearer disc margins. Blood pressure also normalized after 4 days on oral antihypertensives. Urinalysis revealed a proteinuria 1+ up to the 10<sup>th</sup> day. The patient was finally discharged after 12 days with complete vision in both eyes and normal blood pressure. Proteinuria had also resolved completely. A follow-up visit a week after discharge showed that both mother and baby were doing fine.

## Discussion

Although visual disturbances develop in perhaps 25% of women with severe preeclampsia, complete blindness, as seen in this patient, is rare, with an incidence of 1%- to 3%.<sup>[1,3,4]</sup> However, Cunningham *et al.* have shown that blindness is much more common, with an incidence of about 15%.<sup>[2,4,5]</sup> This blindness can be due to involvement of the occipital cortex or the retina.<sup>[6]</sup> Most cases of blindness in preeclampsia and eclampsia were, in the past, commonly attributed to retinal pathology: vascular abnormalities, edema or detachment.<sup>[1,4]</sup> Nowadays, more emphasis is being placed on cortical blindness, following numerous case reports of such blindness. Current opinion suggests that blindness in severe preeclampsia is mostly associated with cortical etiology.<sup>[1,5]</sup> Cortical blindness is a clinical syndrome characterized by intact pupillary reflexes and normal fundoscopic findings.<sup>[1,4,5]</sup> The lost vision is usually regained within 4 hours to 8 days.<sup>[5,7]</sup> In the case presented, the patient regained vision completely on the third day. One of the limitations of our case report is the lack of neuroimaging facilities at our center. As such, cortical blindness cannot be ruled out in our patient. However, the fundoscopic findings of bilateral papilloedema and macular edema in our patient strongly call attention to the retina as a possible etiologic site. Grimes *et al.*, in 1980, reported the first case in which computed tomographic scanning was used to demonstrate reversible cortical lesion in a woman with preeclampsia and temporary blindness.<sup>[1,8]</sup> Neuroimaging findings in cortical blindness range from normal to typical findings such as bilateral cortical occipital lesions with hypodensity on CT or hyperdensity on T2-weighted MRI.<sup>[1,2,4,9]</sup>

There is 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. Thus the nature and duration of these

reversible focal neurological lesions and transient cortical blindness are in concordance with the characteristic reversible pathophysiologic changes of preeclampsia and eclampsia.<sup>[1,10]</sup>

The management of patients with fulminating preeclampsia, as seen in this patient, is the same whether it is complicated with blindness or not.<sup>[1,2,4,5]</sup> The patient had IV diazepam (for seizure prophylaxis) in the absence of magnesium sulfate; antihypertensives (IV hydralazine, oral nifedipine and methyldopa); and emergency cesarean section as a rapid method of delivery.

In conclusion, we have presented a case of reversible blindness complicating fulminating preeclampsia, possibly as a result of cortical blindness with some degree of retinopathy. Health care providers should draw lessons from this experience when confronted with similar cases.

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