

Research Letter

Bilateral retinal detachment with subsequent blindness in a pregnant woman with severe pre-eclampsia

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Visual changes or blindness, an unusual complication of hypertension during pregnancy, is a result of several mechanisms. The possible mechanisms, either central or peripheral, include retinal arteriolar vasospasm, thrombosis of the central retinal arteries, reversible cortical pathology, and retinal detachment [1]. Although retinal arteriolar vasospasm is the most common mechanism of visual disturbance, cortical blindness has an incidence of 1–15% in the setting of pre-eclampsia [2]. Retinal detachment, reported in 1–2% of patients with severe pre-eclampsia, is usually bilateral and serous, and its pathogenesis is often related to choroidal ischemia secondary to an intense arteriolar vasoconstriction [3–5]. The majority of patients have a complete recovery of vision with clinical management, and surgery is usually unnecessary [3–9]. Over the past 40 years, the incidence of visual problems has been declining, which probably reflects the lower incidence of severe pre-eclampsia due to better antenatal care. The following report is on a rare case of bilateral retinal detachment with subsequent blindness in a patient with severe pre-eclampsia, without the findings of arteriolar vasoconstriction, infarction, or cortical pathological changes.

A 28-year-old nulliparous woman at 38 weeks of gestation presented to the emergency department due to an episode of syncope. On arrival, she complained of headache, pedal edema, and decreased urine output, with a higher blood pressure of 191/112 mmHg. Neurological examination was grossly normal and the confrontation visual fields were intact. An initial laboratory examination revealed 3+ proteinuria, a hemoglobin level of 146 mg/L, total white cell count of $14.9 \times 10^9/L$, platelet count of $106 \times 10^9/L$, alanine aminotransferase level of 11 IU/L, a creatinine level of 8.2 mg/L, fibrinogen level of 5.19 g/L, and FDP-D dimer level of

2.87 mg/L. The patient had normotension throughout pregnancy. Under the impression of severe pre-eclampsia, she underwent labor induction, which was unsuccessful and was followed by cesarean delivery. The infant's Apgar score was 9 at both 1 min and 5 min without any neonatal complications. Serial examinations of blood tests, liver enzyme levels, and renal function revealed no worsening of the condition. Twenty-four hours after delivery, the patient started complaining of blurred vision. Her blood pressure was around 140/90 mmHg, and upon consultation with the ophthalmologist, the best-corrected visual acuity was determined to be 20/200 in the right eye and 20/2000 in the left eye. The anterior segment was generally normal, whereas the color fundography disclosed an extensive bilateral serous retinal detachment (Fig. 1). Computed tomography (CT) of the brain was performed to exclude simultaneous cerebral edema or infarct (Fig. 2A). With fluorescein angiography, normal arterial filling, which was initiated 14 seconds after fluorescein injection, excluded the possibility of retinal arterial occlusion (Fig. 2B). Fluorescein angiography in the middle phase showed that the retinal vasculature was relatively spared without any evident signs of vascular occlusion (Fig. 2C and D) or retinal vasoconstriction. In the late venous phase, confluent patches were evident, indicating choroidal hyperpermeability and dye leakage, which is compatible with the diagnosis of severe choroiditis (Fig. 2E and F). A diagnosis of severe pre-eclampsia complicated with a bilateral serous retinal detachment was made. The patient was prescribed amlodipine (5 mg orally/day) combined with furosemide (20 mg orally/day) to control the blood pressure, and prednisolone (5 mg orally twice a day) for alleviating choroiditis. Despite the treatment mentioned above, the patient's visual symptoms progressed to a completed bilateral blindness over the next 24 h. She was discharged 1 week later with an adequate control of blood pressure and good physical condition, but limited recovery of visual acuity. The condition of both of her eyes did not improve with time at follow-up examinations. The best-

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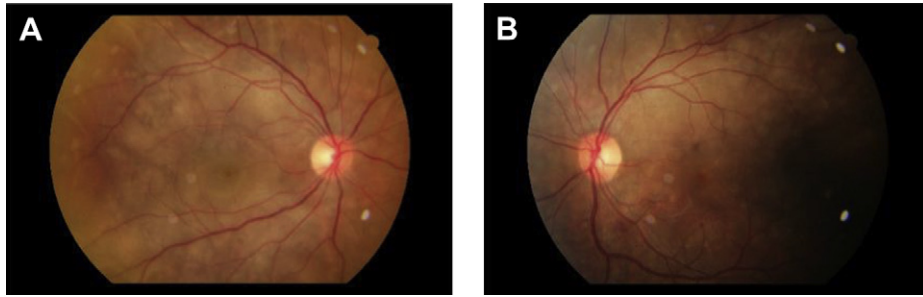


Fig. 1. Fundus photographs of the (A) right and (B) left eyes revealing extensive serous retinal detachment.

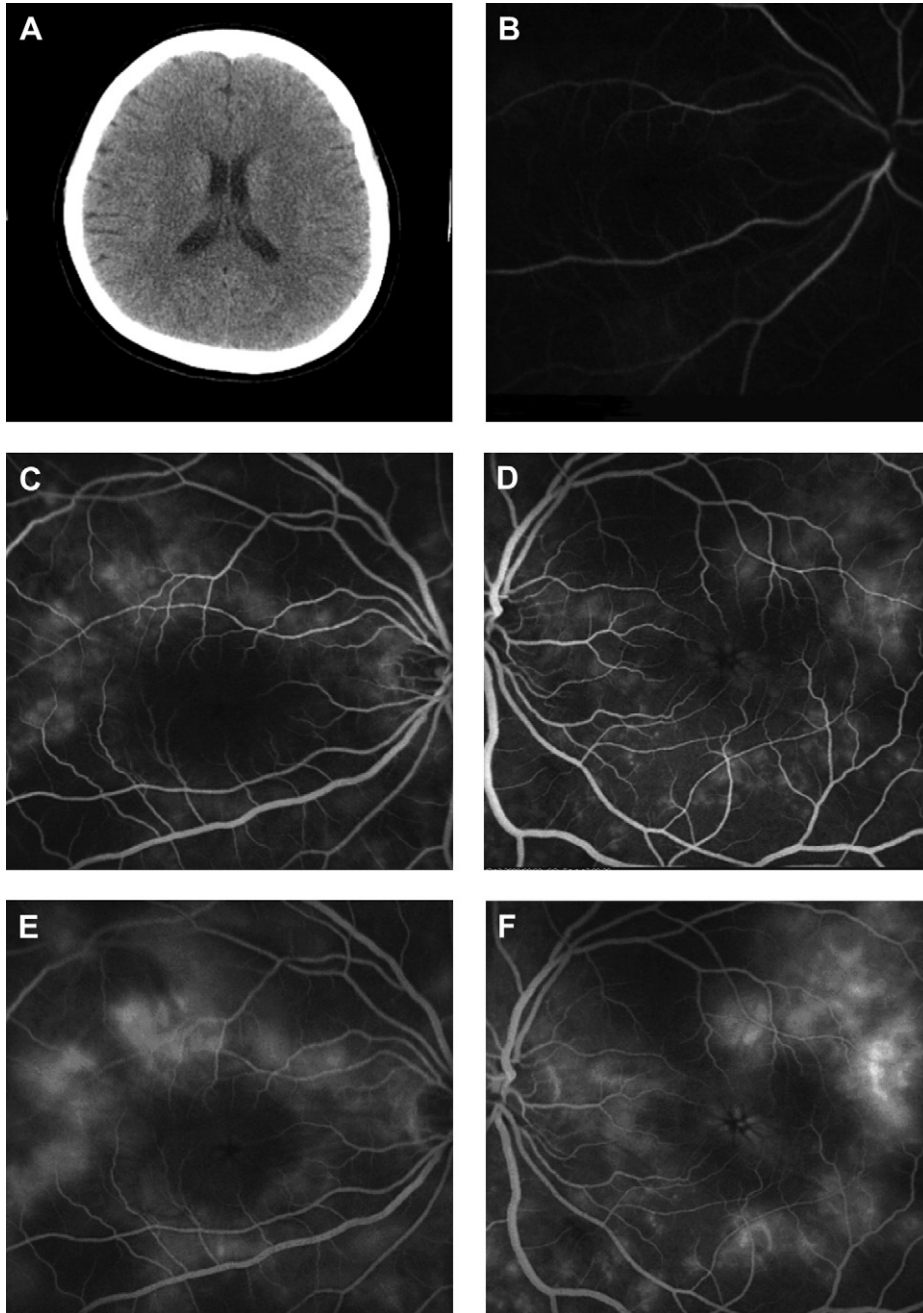


Fig. 2. (A) Computed tomography of the brain reveals no evidence of cerebral edema or infarct. (B) Fluorescein angiography (initiated at 14 seconds after fluorescein injection) reveals normal arterial filling, excluding the possibility of retinal arterial occlusion. (C) Right and (D) left fluorescein angiography in the middle phase reveals relatively sparse retinal vasculature without evident signs of vascular occlusion. (E) Right and (F) left fluorescein angiography in the late venous phase reveals confluent patches, indicating choroidal hyperpermeability and dye leakage, compatible with the diagnosis of severe choroiditis.

corrected visual acuity achieved was 20/2000 in the right eye and 20/2000 in the left eye.

Spontaneous retinal detachment is an uncommon complication of pre-eclampsia, occurring either *antepartum* [10,11] or *postpartum* [3,4,6–9,12]. Most patients with retinal detachment in pregnancy-induced hypertension and associated complications have full spontaneous resolution within a few weeks, and they do not have any sequelae [3,4,6–9,13]. Retinal and choroidal vascular damage have been implicated in the pathogenesis. In addition to the possible mechanisms mentioned above, Hutchings et al hypothesized that retinal detachment is secondary to choroidal vascular damage [10]. In many cases, fundoscopic findings are bilateral papilledema and macular edema. Previous case reports have suggested a higher incidence in primigravidas, and most cases occurred *postpartum*. These detachments are usually bilateral, serous, and nonrhegmatogenous (i.e. atraumatic), and they often only involve the posterior pole. Apart from one report describing a patient with severe pre-eclampsia who suffered from blindness secondary to retinal arteriole occlusions, with permanent visual loss noted at follow-up [14], a literature review using Medline indicates that most cases of pre-eclampsia-related blindness are transient, with a full recovery after 48–72 h [2–9,11,13]. In many cases, visual impairment can be attributed to cerebral edema secondary to pre-eclampsia, with resulting transient cortical blindness [2,11,13,14]. CT or magnetic resonance images of the brain often reveal areas of signal shifts in the occipital lobes, demonstrating cortical pathological changes of edema or infarct [2,7,11,13,15]. In our case of severe pre-eclampsia with bilateral retinal detachment and subsequent blindness, there was no such finding with a CT scan. Additionally, fluorescein angiography revealed confluent patches indicating choroidal hyperpermeability without changes of occlusion, infarction, or vasoconstriction of retinal vessels (Fig. 2), which is unlike the typical pattern. Although the changes in choroidal ischemia and increased permeability are similar to those of the patient with idiopathic central serous retinopathy described by Hutchings et al. [10], in our case, the presentation of retinal detachment in both eyes is different from that of the patient with idiopathic central serous retinopathy, which usually involves one side. Moreover, the visual changes with retinal detachment and subsequent blindness developed after delivery, without an adequate recovery noted on follow-up, is also different from previously described cases.

Our findings suggest that a different mechanism may be responsible for the bilateral retinal detachment with subsequent blindness in pre-eclampsia when compared to the known traditional pathways mentioned above; however, the true pathophysiology remains unknown. Based on the clinical presentation, we propose that the primary etiology of the bilateral retinal detachment with subsequent blindness was due to a delayed immunochemical reaction in response to the insults of severe pre-eclampsia. It is well known that the control of ocular blood flow occurs predominantly at the level of the retinal and choroidal vessels. According to Scholfield et al, the regulation of retinal and choroidal vessel tone or permeability is primarily mediated via Ca^{2+} handling mechanisms and

plasmalemmal ion channels. The local Ca^{2+} transients (Ca^{2+} sparks) play an important excitatory role, which act as the building blocks for more global Ca^{2+} signals that can initiate the regulation of associated vessels [16]. Thus, an immunochemical reaction may be mediated by the cytokines released in severe pre-eclampsia, contributing to endothelial damage and inflammation. Consequently, injury to the choroidal layer may result in an autoregulatory dysfunction of choroid circulation, increase vascular permeability and leakage of protein and fluid into the subretinal space, and hence induce retinal detachment, followed by permanent visual loss. However, this hypothesis remains to be confirmed. Further work that explores the relationship between pre-eclampsia and retinal detachment with subsequent blindness is warranted.

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