

# Amaurosis fugax in primigravida woman precursor of posterior reverse encephalopathy syndrome concurrent with macular edema

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Dear Editor,

Posterior reverse encephalopathy syndrome (PRES), manifests as a confusional state/delirium, convulsion, or acute blindness which illustrates in magnetic resonance imaging (MRI) typical bilateral white matter lesions. These clinical and radiological changes are reversible in two to three weeks, usually generated by acute hypertension, preeclampsia, eclampsia, immunosuppression, septicemia, and end-stage renal disease. PRES is commonly diagnosed in patients in their thirties. Retrospective review showed that preeclampsia patients who present PRES were free of pregnancy-associated comorbidities, of late term gestational age with eclampsia, postpartum seizures, hypertension managed with labetalol and in patients who underwent cesarean section<sup>[1]</sup>.

Here, we present a rare case of PRES, generated by preeclampsia without previously announced related statistical important incidences associated with unilateral macular edema. We investigated a myopic patient, free of myopic retinopathy, presented with ocular symptoms precursor of PRES with simultaneous signs related to systemic hypertension. Fundus examination revealed unilateral macular edema, subretinal hemorrhage and cotton wool spot. With this case we aim to advance our understanding of possible side effects of preeclampsia on ophthalmic physiology and simultaneously

provide a source for clinicians in the handling of patients with preeclampsia/eclampsia syndrome with ocular involvement.

## CASE REPORT

**Ethical Approval** The study was approved by the Ethics Committee of the General Hospital of Patras "O Agios Andreas" (approval number:2024p.n.12), and conducted in accordance with the guidelines of the Declaration of Helsinki. Written informed consent for publication was obtained from the patient.

**Initial Ophthalmic Admission** A 22-year-old primigravida during the 30<sup>th</sup> week of gestation who presented with bilateral persistent painless loss of vision was brought to the ophthalmology emergency room of a tertiary care hospital after being referred from a private practitioner. At admission, the best corrected visual acuity was hand motion in both eyes. The intraocular pressure was normal. Examination of the eye's didn't show any discomfort, redness, photophobia, lacrimation or anterior segment inflammatory reaction. Fundus examination revealed normal optic nerves, a cup-to-disk ratio of 0.4, retinal vessels appeared like a corkscrew, beaded pearl necklace and with arteriovenous crossings. Solitary cotton-like exudate and retinal hemorrhage were observed. Patient underwent macular optical coherence tomography (OCT) examination which displayed macular edema (Figure 1C). Consequently, patient condition deteriorated presenting headache, nausea, vomiting, altered mental status, lower extremity edema. Glasgow Coma Scale score assessment presented mild drop. Clinical examination and medical record cross-check revealed new onset of hypertension, systolic blood pressure 170 mm Hg and diastolic blood pressure 100 mm Hg, accompanied by proteinuria. The patient was diagnosed as preeclampsia syndrome. Complete understanding of patient's medical record, the natural history of the disease and health status along with detailed systematic examination is essential in order to confirm the diagnosis of imminent preeclampsia. Auxiliary examinations, such as OCT, and computerized tomography (CT) are crucial for the diagnosis of this disease, and CT is the key examination to its early diagnosis. Patient underwent CT scan to exclude gross central nervous system lesions such as infarction hemorrhage. CT scan at presentation

exhibited bilateral multiple low signal lesions, hypodensities, in the white matter located in the occipital and parietal lobes (Figure 1A, 1B) with sulcal effacement (Figure 1A, 1B) related to vasogenic edema, suggestive of PRES.

Due to the critical state of the patient's health, useful diagnostic examinations with electroretinogram and evoked potentials were not possible to perform.

The rest of the systemic examination discovered no notable neurological findings. Laboratory reports revealed normal hemoglobin, total leukocyte count, platelet counts. Urinalysis was remarkable for the presence of proteinuria +3 (normal: nil). Erythrocyte sedimentation rate, liver and renal function tests were within the normal range. Other blood tests, arterial blood gas analysis, viral markers, autoantibodies, neoplastic markers, chest radiographs and cerebrospinal fluid analysis were normal. Ultrasonography of the kidney, ureter, bladder, and pelvis was unremarkable.

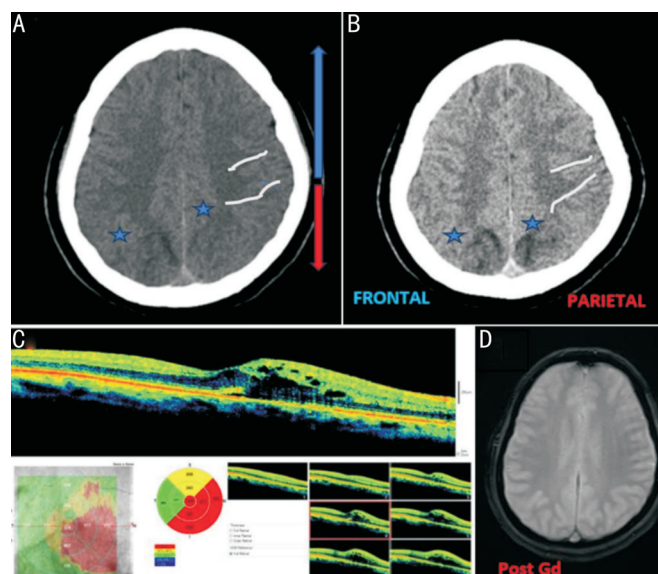
We treated our patient with intravenous administration of fluids, and mannitol 10% w/v and subthreshold photocoagulation for macular edema. Mannitol was given in order to both improve cerebral blood flow, decrease intracranial pressure and improve retinal perfusion. Anti-hypertensive (calcium-channel blockade) agent was given to manage the patient's elevated blood pressure (170/100 mm Hg).

Clinical improvement wasn't seen with supportive treatment. Fetal status evaluation led to intervention, delivering fetus, to reverse the dire consequences. Subsequently, patient underwent MRI which showed complete resolution (Figure 1D).

## DISCUSSION

Patient underwent a meticulous physical examination, primarily with vital signs evaluation, blood pressure in particular. Suspicion for preeclampsia increased due to high systolic blood pressure of 170 mm Hg and diastolic pressure of 100 mm Hg, with blood pressure measurements interval of no less than 4h, repeated in more than two occasions.

Preeclampsia is a clinical syndrome that afflicts 3%-5% of pregnancies, is a new-onset hypertensive disorder after 20wk of gestation, determined as a systolic and diastolic blood pressure equal or greater than 140 mm Hg and 90 mm Hg respectively, on two no sequential measurements no less than 4h from each other; or shorter interval timing coinciding with systolic blood pressure of  $\geq 160$  mm Hg or diastolic blood pressure of  $\geq 110$  mm Hg associated simultaneously with urinary protein excretion  $>300$  mg/d. Other significant clinical findings at presentation involve, severe impairment of major body organs, such as liver failure, epigastric or acute persistent right upper quadrant pain, low blood platelet count, excluding all other possible diagnoses, new-onset treatment resistant headache, pulmonary congestion, or impaired renal function with abnormal laboratory results<sup>[2]</sup>.



**Figure 1 Multimodal imaging** A, B, stars: CT scan at presentation revealed white matter bilateral multiple low signal lesions located in the parietal and occipital lobes; White lines showed sulcal effacement; C: OCT macular map illustrated cystoid macular edema; D: Late MRI scan displayed complete resolution of white matter lesions. CT: Computerized tomography; OCT: Optical coherence tomography; MRI: Magnetic resonance imaging.

Preeclampsia has been illustrated to originate from atypical placentation, resulting to remodeling of aberrant spiral arteries, hypoxia, ischemia and oxidative stress<sup>[2-3]</sup>.

Patient's thorough medical record and investigation wasn't connected to any of preeclampsia predeterminant risk factors including multi-gestation pregnancy, nulliparity, maternal age over than 35 years old, forms of assisted reproductive technology, maternal comorbidities (chronic hypertension, diabetes mellitus, chronic kidney disease, thrombophilia, obstructive sleep apnea, obesity with pre-pregnancy body mass index greater than 30), family history, previous pregnancy history of placental abruption or preeclampsia, or intrauterine fetal growth restriction<sup>[2]</sup>.

Even though our patient was free of organic changes of arteriolosclerosis she was presented with severe arteriolar spasm, probably being the primary response of the retinal vasculature to increased blood pressure which consequent led to vasoconstriction. Moreover, our patient, being myopic, was involved with unilateral fluid extravasation to the extravascular spaces due to increased vascular permeability. Other retinal changes observed, were decreased retinal to vein ratio, single hemorrhage and cotton wool spot, no serous retinal detachment was seen. Due to aforementioned signs subthreshold laser photocoagulation was applied, we used recommended settings, 200ms pulse duration, 100-200  $\mu$ m spot size and 5% duty cycle<sup>[4-5]</sup>. Placental abnormal arteriole remodeling and vascular sclerosis gradual results to ischemia, free inflammatory like, pro

inflammatory antiangiogenic factors, distress markers, thus initiating an imbalance of existing competition with binding regions for crucial angiogenic and growth molecules. This subsequently causes downregulation and insufficient vascular accommodation for various organ systems, especially renal, cardiovascular, and hepatic<sup>[2,6]</sup>.

Patient new-onset headache was unresponsive to medication, not attributed to any other possible diagnosis (*i.e.*, history of headaches or migraines) that is.

Due to shortness of breath, percussion and auscultation of the lungs was tested for pulmonary disturbances. Abdominal symptom was further investigated for tenderness. Followed by evaluation of dependent, gravity-related lower extremities edema.

Also, we assessed patient's medical history prior to physical exam and meticulous diagnostic testing, including a complete assessment for liver function, renal markers to identify excessive protein level in urine, daily urine collection sample was greater 300 mg, protein to creatinine ratio was 0.3, renal failure factors, and complete blood count to evaluation for thrombocytopenia.

Even though visual manifestations are quite common, complete blindness is rare, with a reported incidence of 1%-3%<sup>[7-8]</sup>. Blindness in our case was attributed to the involvement of both retina and parietal - occipital cortex which is a rarely reported combination in bibliography.

Even though proteinuria with coexisting increased blood pressure is considered to be required for establishing the diagnosis of preeclampsia, it is not always sine qua non condition. In such instances, first detectable episode of other symptoms such as impaired liver or renal failure, thrombocytopenia, pulmonary congestion, or new-onset cephalalgia frequently involving visual symptoms may be used for diagnosis. This is noted as preeclampsia free of severe features, encompassing newly initiated high blood pressures (systolic pressure equal or greater than 160 mm Hg, diastolic pressure equal or greater than 110 mm Hg on two measurements of no less than 4h difference), not including previously mentioned findings<sup>[1,7-8]</sup>.

Early diagnosis and intervention are the gold standard in the management of preeclampsia, aiming at adequate blood pressure control and ocular manifestations, calcium-channel blockade, was used for pressure control.

Fetal status evaluation was the major factor in determining delivery versus expectant management in our patient.

Fetus delivery is the definitive treatment of preeclampsia. Observation is only acceptable in patients with well-controlled preterm gestations or preeclampsia free of severe features in the setting of normal antenatal fetus surveillance, the possibility of expectant management exists. Patients diagnosed

with preeclampsia or gestational hypertension without severe features at 37 0/7wk gestation should be managed with delivery rather than expectant management as recommended by the American College of Obstetricians and Gynecologists.

Maternal factors are persistent cephalalgia, visual distress, unresponsive blood pressure, localized epigastric or right upper quadrant pain resistant to repeated medical treatment, hemolysis, liver failure, thrombocytopenia, myocardial infarction, eclampsia, stroke, pulmonary congestion, or placental bleeding or abruption without other diagnosis. Intravenous and oral administration of anti-hypertensive agents, hydralazine, labetalol, and nifedipine respectively are utilized to control increased blood pressures. Intravenous magnesium sulfate is considered to be indicated for seizure prophylaxis in cases with severe features preeclampsia<sup>[2,7-8]</sup>.

In conclusion, the iatrogenic treatment of arterial blood pressure with the administration of specific agents may improve visual prognosis in preeclamptic cases, which can implicate visual function. Cases complicated with hypertensive retinopathy may require rescue therapeutic interventions. Therefore, ophthalmologists must be vigilant for pregnancy related hypertension and ophthalmic disturbances. Our results may assist clinicians deal with possible complications in the handling of pregnancy hypertension, with medical records similar to the patient in our case. This study should alert clinicians to the significance of visual signs during pregnancy and hypertension management.

## ACKNOWLEDGEMENTS

**Authors' Contributions:** Christodoulou P diagnosed and treated this case; wrote the manuscript; collected data; Katsimpris I reviewed the manuscript. All authors have read and approved the final version of the manuscript.

**Conflicts of Interest:** Christodoulou P, None; Katsimpris I, None.

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