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Case Report

Isolated sixth nerve palsy as the first manifestation of preeclampsia ☆,☆☆

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ABSTRACT

Preeclampsia constitutes one of the leading causes of maternal morbidity and mortality in the United States. Preeclampsia-related neurological disorders are well-established and associated with a broad spectrum of manifestations, including headaches, visual symptoms like blurred vision, photopsia, field defects, and other major clinical events. However, cranial nerve disorders are rare in preeclampsia, which is inadequately researched. Here, we present a 26-year-old primigravida woman with an isolated abducens nerve palsy as the first sign of preeclampsia at 35 weeks of gestation.

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Introduction

Preeclampsia is a multisystem hypertensive disorder that affects 3%–5% of pregnancies with clinical spectrum including severe preeclampsia, eclampsia, hemolysis, elevated liver enzymes, and low platelets (HELLP) syndrome, and HELLP syndrome with eclampsia [1]. Based on the evidence, there are several maternal risk factors involved in the development of preeclampsia, including advanced maternal age, nulliparity, previous history and family history of preeclampsia, obesity, comorbid medical conditions including hyperglycemia in pregnancy, pre-existing chronic hypertension, renal disease,

and autoimmune diseases, such as systemic lupus erythematosus and antiphospholipid syndrome [1].

The primary neurologic symptoms of preeclampsia include headache, blurring of vision, and confusion. The spectrum of ocular symptoms seen in preeclampsia comprises blurring of vision, scotoma, hemianopia, and even total cortical blindness, whereas abducens nerve palsy is seldom documented as a presenting symptom in preeclampsia [2]. As per the literature, the most reported etiologies for abducens nerve palsy are vasculopathy (29%), tumors (16%), multiple sclerosis (12%), presumed inflammation (8%), trauma (6%), post-lumbar puncture (4%), and orbital amyloidosis (2%) [3]. The exact pathogenesis of abducens nerve palsy associated with high blood pres-

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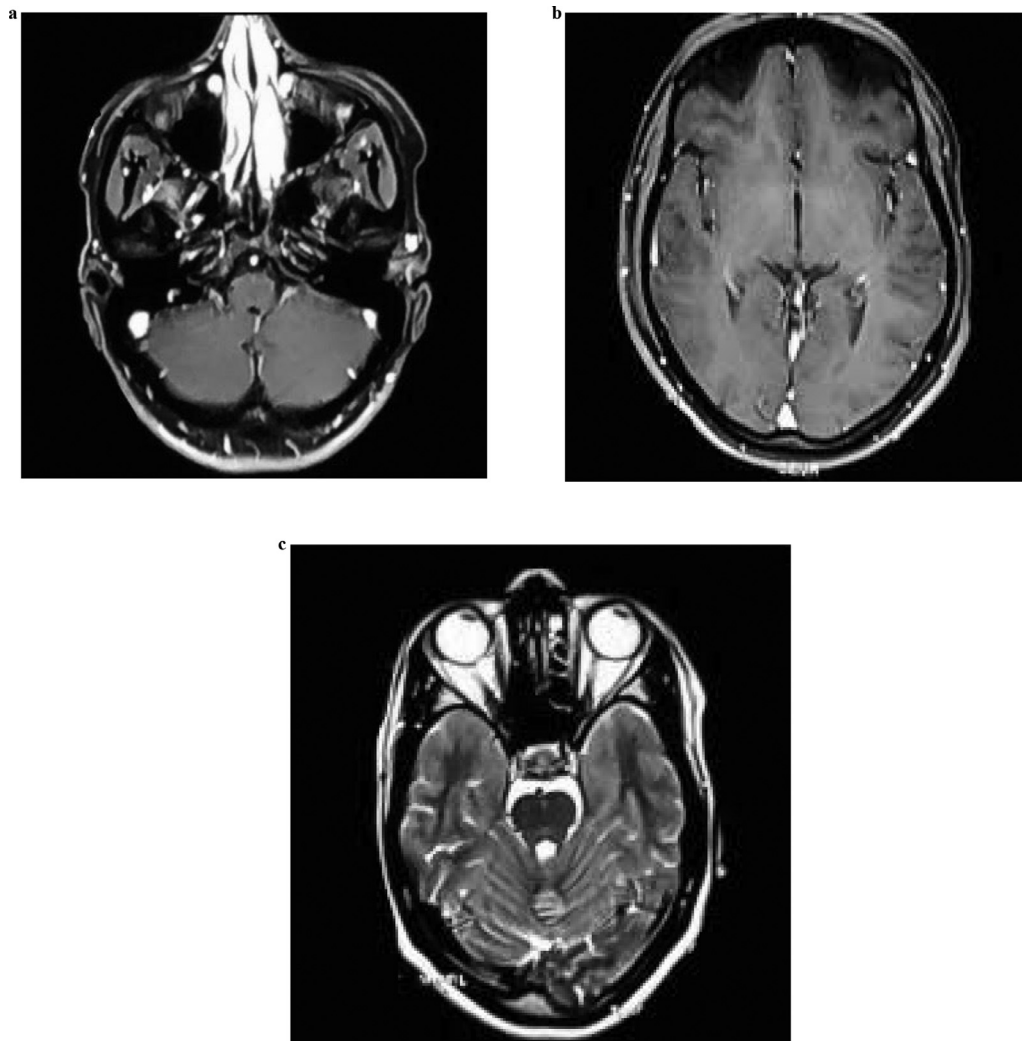


Fig. 1 – (A-C) Brain magnetic resonance imaging (T2-weighted image) showing no specific pathology.

sure during pregnancy remains unknown. We report a case of a young primigravida female presenting with unilateral abducens nerve palsy as the first manifestation of preeclampsia.

Case report

A 26-year-old primigravida woman at 35 weeks of gestation was referred to our emergency department with diplopia and high blood pressure. She reported progressive diplopia for the past 6 days associated with a mild right-sided headache. There was no history of nausea, vomiting, loss of vision, neck stiffness, epigastric pain, or shortness of breath. Her medical history was unremarkable, with no history of hypertension, diabetes mellitus, or neuro-ophthalmologic disorders.

On general examination, the patient was alert and verbal communication was normal. She had hypertension with a blood pressure of 170/110 mmHg. Cranial nerve evaluation documented limited abduction in the right eye and diplopia charting confirmed isolated right-sided sixth cranial nerve

palsy. Extraocular movements were full in the left eye with visual acuity of 6/6 in both eyes. The fundoscopic examination was normal. Neurological examination of the upper and lower limbs showed normal muscle tone, power, and reflexes. Gait and coordination were normal, with no signs of meningeal irritation. Examination of other systems was unremarkable. The initial laboratory investigations revealed the following; hemoglobin: 9.8 g/dL (normal: 13.8-17.2 g/dL), total leukocyte count: 9600/mm³ (normal: 4000-11,000/mm³), platelet count: 230,000/mL (normal: 150,000-450,000/mL), creatinine: 0.9 mg/dL (normal: 0.59-1.04 mg/dL), uric acid: 5.2 mg/dL (normal: 3.5-7.2 mg/dL), serum bilirubin: 0.9 mg/dL (normal: 0.1-1.2 mg/dL), aspartate aminotransferase: 45.20 U/L (normal: 8-33 U/L), alanine aminotransferase: 37.0 U/L (normal: 4-36 U/L), serum total protein: 5.9 g/dL (normal: 6-8.3 g/dL), serum albumin: 4 g/dL (normal: 3.4-5.4 g/dL), lactate dehydrogenase: 331 IU/L (normal: 105-333 IU/L), prothrombin time: 15 seconds (normal: 11-13.5 seconds). On urine analysis, trace albuminuria and a few pus cells and epithelial cells were reported with no proteinuria. Chest X-ray and electrocardiogram (ECG) were normal.

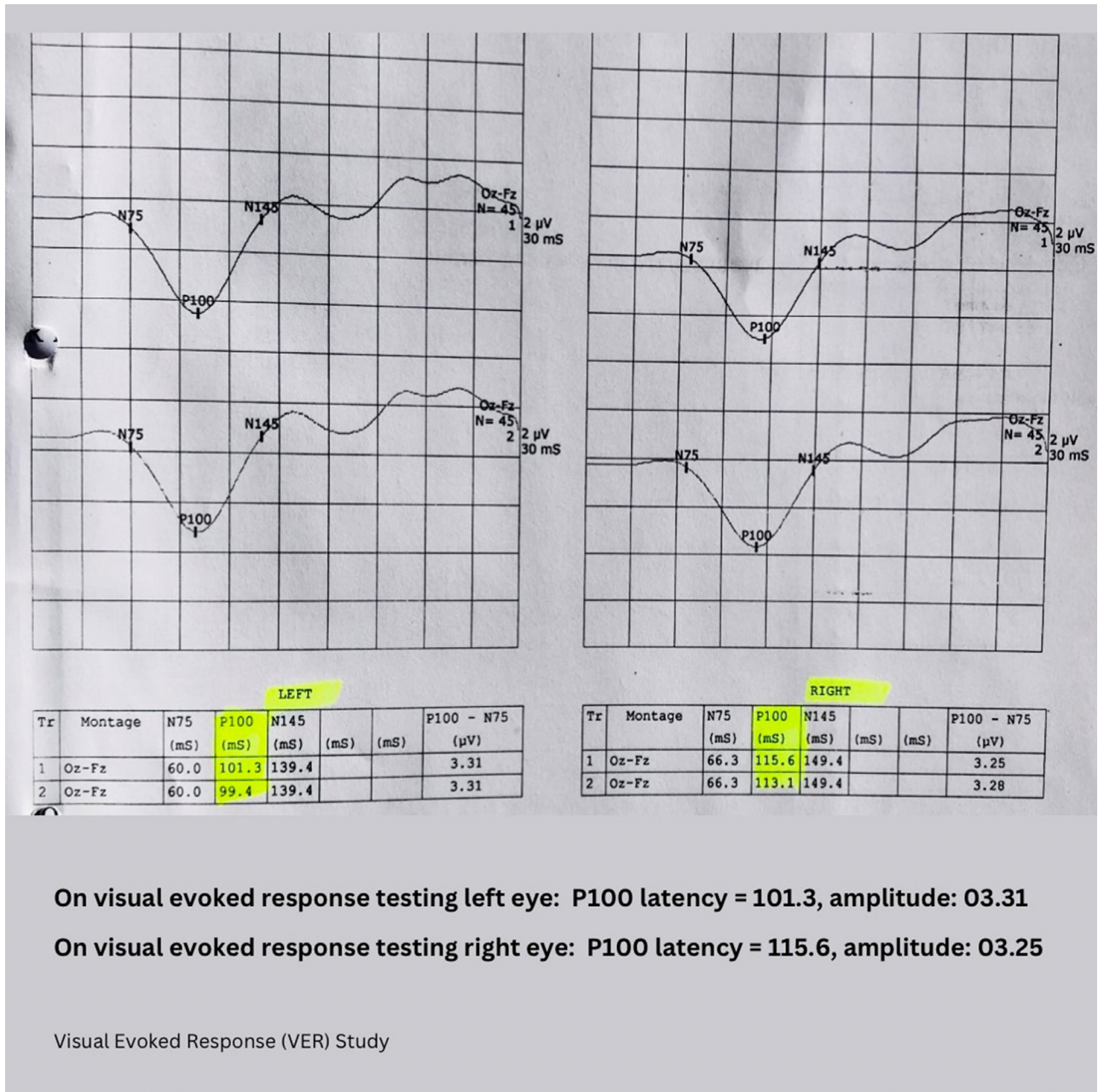


Fig. 2 – Findings of visual evoked response (VER) study.

The patient was admitted, and intravenous (IV) labetalol 20 mg stat was administered for elevated blood pressure. Also, she was prescribed twice daily oral labetalol 100 mg with frequent blood pressure monitoring. The blood pressure was controlled adequately 24 hours after admission (125/80 mmHg), and the patient was induced into labor and delivered a baby girl, weighing 2400 g. First and 5-minute Apgar scores were 9 and 10, respectively. The placenta was normal. On the third day of postpartum, magnetic resonance imaging (MRI) brain and orbit was done, and did not reveal any evidence of increased intracranial tension, hematoma, space-occupying lesion, hydrocephalus, or any orbital abnormality (Fig. 1). Also, a visual evoked response test documented prolonged P100 latency (prominent positive deflection) on the right side, reveal-

ing the right visual pathway abnormality (Fig. 2). In the absence of any other cause, a diagnosis of preeclampsia-induced isolated sixth cranial nerve palsy was made. No specific treatment was given for the sixth cranial nerve palsy. Upon follow-up after the delivery, blood pressure was under control, and diplopia began to improve spontaneously with regression in sixth nerve paresis.

Discussion

Isolated abducens nerve palsy is a rare complication of preeclampsia and hypertension in pregnancy. Based on pre-

vious reports, bleeding or infarction in the nucleus of the abducens nerve has been suggested as a cause of the abducens nerve palsy. Since preeclampsia can cause severe vasoconstriction, thus, severe vasospasm supplying vessels of the sixth cranial nerve can cause ischemia and nerve paralysis [2,4]. This is one of the proposed pathogenesis of isolated abducens nerve palsy in preeclampsia after excluding other possible causes discussed in the introduction. A few authors have suggested a downward displacement of the nerve due to increased intracranial pressure or nerve ischemia as a probable cause of sixth nerve palsy in pregnancy [5,6]. As the abducens nerve has a long course from the brainstem to the lateral rectus muscle in the orbit, paralysis may result from any lesion of the nerve between the pons and orbit.

In 1968, Bladé et al. [5] reported the first case of sixth nerve palsy associated with pregnancy in a 22-year-old primigravida. The patient was diagnosed with eclampsia and developed sixth nerve paresis 24 hours after postpartum. She presented in a coma stage with a pronounced neurological deficit. On further evaluation, she had increased intracranial and intraocular pressure along with vasospasms of retinal arterial vessels. And she recovered after 6 months [5]. Park and Kim [6] in 2007 recorded a case of sixth nerve palsy in a 33-year-old multi-gravida with preeclampsia after 24 hours postpartum, and the patient recovered in seven days, which is the shortest recovery period so far reported. Nieto-Calvache et al. [7] recently reported a case of isolated sixth cranial nerve palsy after delivery in a 28-year-old woman with gestational hypertension. There was no specific pathology documented on detailed evaluation, and symptoms of abducens nerve palsy resolved spontaneously. Barry-Kinsella et al. [8] presented sixth nerve palsy in a multigravida with hypertension and proteinuria at 38 weeks of gestation. After inducing labor, hypertension escalated in the early puerperium with concomitant worsening diplopia. They noticed the resolution of the sixth nerve lesion after controlling blood pressure with no evidence of other pathology on extensive investigations [8].

In patients with preeclampsia presenting as abducens nerve palsy, regardless of its rarity, a complete investigation including CT or MRI brain must be undertaken to exclude other possible underlying pathologic conditions like hemorrhage, intracranial lesions, or edema. We also recommend avoiding epidural/spinal anesthesia in patients diagnosed with sixth nerve palsy during delivery, as these procedures can cause downward displacement of the nerve that can worsen the paresis. In conclusion, based on our case and other cases recorded in the literature, the prognosis for recovery is excellent without specific treatment in isolated sixth nerve

palsy associated with preeclampsia and hypertension in pregnancy. The patient can be reassured about the complete recovery of the palsy, although there is significant variability in the duration of recovery.

Patient consent

The case information in this manuscript has been provided with informed consent from the patient presented.

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