• Letter to the Editor •

Isolated unilateral abducens nerve palsy as a neurologic complication of severe preeclampsia: a case report

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Dear Editor,

We report a case of a patient with isolated left abducens nerve palsy and bilateral resolving serous retinal detachment (SRD) following a recent pregnancy complicated by severe pre-eclampsia. Extraocular motility testing revealed an abduction deficit in the left eye and 25 prism diopters of esotropia. Patching of the left eye was done to relieve the diplopia and the patient was observed. Over subsequent months, the patient showed gradual improvement.

CASE PRESENTATION

Ethical Approval This case was approved by the Research Ethics Committee of Armed Forces Hospitals-Jeddah (Ref: REC 784; Application No. 2025-21). Written informed consent has been obtained from the patient to publish this paper.

A 35-year-old Filipino woman presented to the clinic 10d postpartum complaining of diplopia, inward deviation of the left eye, and flashes of light in both eyes. Her symptoms began during the delivery. She had a recent history of a complicated pregnancy and severe pre-eclampsia. She had a known history of hypertension but was not on any medications; she had no history of traumatic head injuries, or other relevant medical conditions. Upon examination, she had an uncorrected visual acuity of 6/15 in her right eye and 6/12 in her left eye. Her intraocular pressures were normal. The pupils were round, regular, and reactive in both eyes, with a trace relative afferent pupillary defect in the left eye. Extraocular motility testing revealed an abduction deficit left eye and 25 prism diopter esotropia (Figure 1). The remaining cranial nerves were intact. The anterior segment examination results were unremarkable. Fundus examination revealed a flat retina with areas of resorbed subretinal fluid inferiorly in both eyes, which were also visible on optical coherence tomography of the macula (Figure 2). She had a healthy-looking optic nerve with clear disc margins, healthy neuro-retinal rim, and a cup-to-disk ratio of 0.3 in both eyes. Humphrey's visual field test results were unremarkable. Contrast computed tomography (CT) and magnetic resonance imaging (MRI) of the brain were performed and the findings were unremarkable. MRI sequences included T1-weighted, T2-weighted, FLAIR, diffusion-weighted imaging (DWI), contrast-enhanced images, and an MR venogram to rule out other potential causes such as ischemia, demyelination, or venous sinus thrombosis. The patient was diagnosed with isolated left abducens nerve palsy and bilateral resolving SRD. Patching of the left eye was recommended to relieve the diplopia. The patient was monitored over the following months.

The patient was evaluated at the 18th week of gestation when she experienced mild headaches. Blood pressure readings at the time ranged from 140/90–150/100 mm Hg. She was started on methyldopa 250 mg every 8h. At 20wk of gestation, she developed a severe headache and bilateral lower limb edema. Her blood pressure had reached 170/100 mm Hg, and urinalysis revealed +3 proteinuria. She was diagnosed with pre-eclampsia and started on nifedipine 20 mg every 8h in addition to the methyldopa. Laboratory test results, including a complete blood count and renal, liver, and coagulation profiles, were unremarkable. At 22wk of gestation, the patient travelled to the Philippines, where she received prenatal care and treatment for pre-eclampsia until delivery. At 24wk of gestation, her headache recurred, in addition to a decrease in urine output and increased abdominal distention. The blood pressure reading at that time was 170/90 mm Hg. The patient received intravenous hydralazine and was admitted. At 25wk of gestation, she underwent epidural anesthesia and delivered a

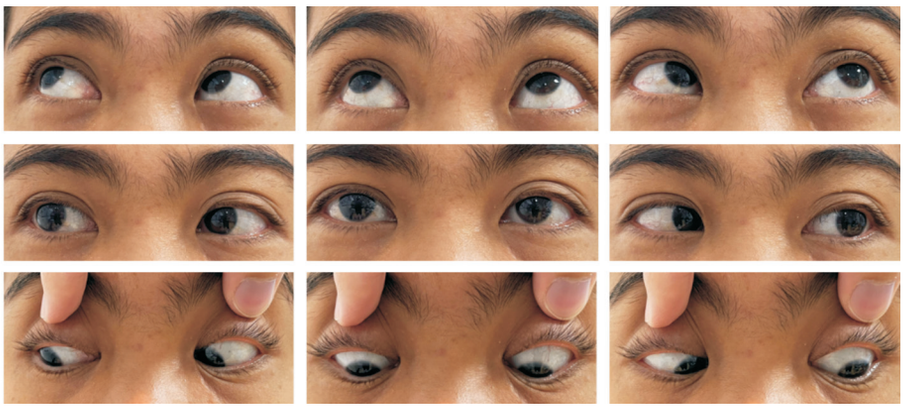

Figure 1 The patient has an esodeviation in primary position with a mild restricted abduction of the left eye on left gaze.

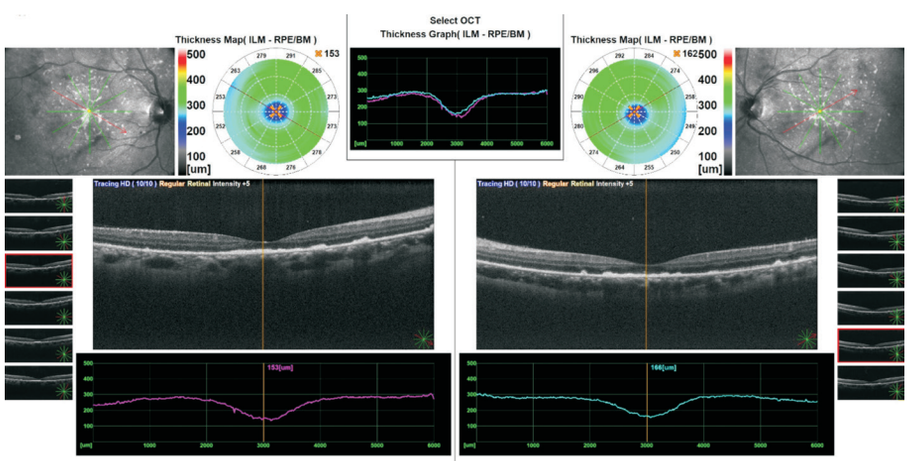

Figure 2 Spectral-domain optical coherence tomography (SD-OCT) scans of both eyes showing resorbed subretinal exudates/fluids ILM: Inner limiting membrane; RPE: Retinal pigment epithelium; BM: Bruch's membrane.

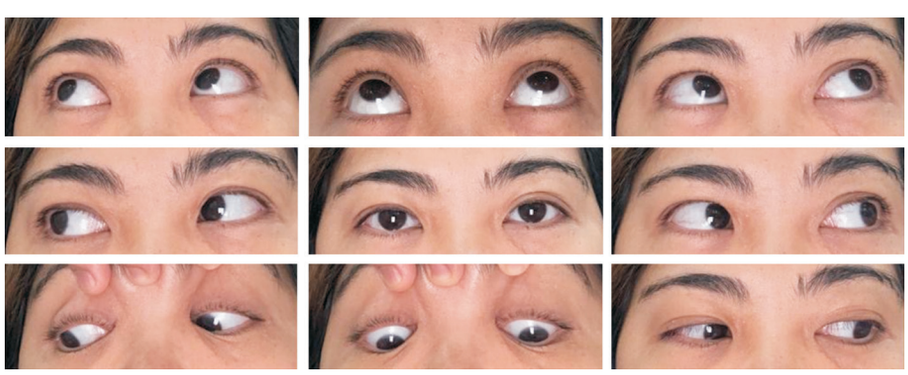

Figure 3 The patient one year after initial presentation demonstrating normal ocular alignment and full range of extraocular movements.

girl *via* cesarean section. Unfortunately, the preterm infant did not survive, likely due to complications of extreme prematurity or maternal medical factors. Two days after delivery, the patient developed flashes of light and floaters. She was examined by an ophthalmologist who reported bilateral SRD. After delivery, the patient returned to Saudi Arabia, where she continued postpartum care and further treatment.

Two months after the initial visit, the patient returned to the clinic for a follow-up visit. Her diplopia persisted and she still had an abduction deficit in her left eye. She received botulinum toxin (Onabotulinum toxin A) injection into the medial rectus muscle of the left eye. One month after the procedure, the patient reported a marked improvement in her vision, with resolution of the inward deviation in her left eye. Extraocular motility was complete in both eyes, and the left abduction deficit had fully resolved. The patient did not report any recurrence of the flashes or floaters. She was scheduled for follow-up after three months and advised to monitor for signs of recurrence. One year after presentation the patient reported no recurrence of symptoms, with stable ocular alignment and full extraocular motility (Figure 3).

DISCUSSION

Hypertensive disorders of pregnancy (HDP) include a spectrum of conditions estimated to occur in around 5% to 10% of pregnancies. These include chronic hypertension, gestational hypertension, pre-eclampsia, and eclampsia[1].

HDP are well-known for their multi-systemic involvement. They can affect the central nervous, renal, and visual systems and may progress to multiple organ failure. Common ocular manifestations of HDP include diplopia, headache, and blurred vision[2]. The neurologic manifestations of HDP include seizures and strokes. However, isolated ocular motor nerve palsies are rare.

The abducens nerve innervates the lateral rectus muscle and is responsible for ipsilateral abduction of the eye[3]. It travels a long course from the brainstem to the lateral rectus muscle in the orbit. Nerve damage may occur at any point along this path from the pons to the orbit. In young adults, isolated abducens nerve palsy is primarily caused by vasculopathies, intracranial tumors, multiple sclerosis, idiopathic inflammation, or trauma[4]. In our patient, brain imaging was performed to exclude secondary causes, such as intracranial lesions or demyelinating diseases. Although cranial nerve palsy rarely occurs during pregnancy, a few isolated cases have been reported (Table 1). Most cases are caused by hypertension and pre-eclampsia, with almost all cases reporting complete recovery; however, the recovery time varies between cases[5-12].

The etiology of HDP-associated cranial nerve palsy remains unclear. Three mechanisms have been postulated to explain this phenomenon: The first theory suggests that in cases of cerebral edema, an increase in intracranial pressure results in displacement of the abducens nerve[13]. This theory is supported by studies on animal models of preeclampsia, which have shown that cerebral vasogenic edema results from impaired cerebral blood flow autoregulation and increased blood-brain barrier (BBB) permeability. Also, circulating factors, likely released from the placenta, may target brain endothelial cells and increase BBB permeability[14]; the second suggests that in cases of cerebral bleeding or infarction, the cerebral center of the abducens nerve may become involved, leading to nerve palsy[15]; the third suggests that this disorder occurs as a result of the hypertension, which leads to vasospasm of the blood vessels supplying the nerve, resulting in ischemia and palsy[15]. Lastly, the fourth theory suggests that preeclampsia-induced systemic inflammation and endothelial dysfunction disrupt protective barriers such as the blood-brain and blood-nerve barriers, leading to an increase in permeability and allowing inflammatory mediators to enter neural tissue[16]. Our patients' findings are likely due to a combined mechanism between the third and fourth theories, as our patient showed no signs of organic brain injury or cerebral edema, with brain imaging revealing no trans-tentorial herniation or midline shift.

Furthermore, our patient's clinical progression from persistent uncontrolled hypertension with SRD to the onset of abducens nerve palsy supports the hypothesis that hypertension-induced vasospasm results in localized ischemia and nerve palsy. SRD

Table 1 Reported cases of abducens nerve palsy associated with pregnancy

Reference	Age	Primiparity	Pre-eclampsia in previous gestation	Involved cranial nerve	Palsy time onset	Diagnosis	Recovery time	Comments
Negreanu *et al*[5]	28	No	Yes, one previous pregnancy	Isolated sixth nerve palsy	36^{+3}wk of gestation	Pre-eclampsia	4d postpartum	Her previous pregnancy was also complicated by Preeclampsia with isolated sixth nerve palsy
Caputo *et al*[6]	35	Yes	None	Isolated sixth nerve palsy	37wk of gestation	Medically free	Not mentioned	The patient was treated with dexamethasone after inducing labor; mild improvement was noticed 3d later
Sebastian *et al*[7]	26	Yes	None	Isolated sixth nerve palsy	35wk of gestation	Pre-eclampsia	Not mentioned	Sixth nerve palsy was the first manifestation of preeclampsia
Nieto-Calvache *et al*[8]	28	No	None	Isolated sixth nerve palsy	37^{+2}wk of gestation	Gestational hypertension	3mo postpartum	The patient's medical background is significant for obesity and hypothyroidism
Yevale[12]	34	No	None	Isolated sixth nerve palsy	Day 7 postpartum	Pre-eclampsia	Day 12 postpartum	The patient was observed with no specific treatment for the sixth nerve palsy
Yousefi[11]	40	No	Yes, 2 previous pregnancies with preeclampsia	Isolated sixth nerve palsy	39wk of gestation	Gestational hypertension	Day 5 postpartum	Apart from uncontrolled blood pressure, the patient presented with diplopia and headache only
Vallejo-Vaz *et al*[9]	36	No	Yes, one previous pregnancy with preeclampsia	Isolated sixth nerve palsy	36wk of gestation	Preeclampsia	3mo postpartum	The patient was initially diagnosed with gestational hypertension, then preeclampsia developed
Turnbull *et al*[10]	28	Yes	None	Isolated sixth nerve palsy	33wk of gestation	Preeclampsia	2mo postpartum	The patient's liver function tests, urate, and proteinuria were rising; however, her diplopia was stable

is a rare but recognized ophthalmic complication associated with HDP, particularly pre-eclampsia[17]. The pathophysiology remains unknown; however, it is thought to be due to choroidal ischemia caused by intense arteriolar vasospasm leading to retinal pigment epithelium (RPE) damage and subsequent subretinal fluid accumulation[18]. In our patient, bilateral SRD developed during an acute pre-eclamptic episode and resolved spontaneously with blood pressure control alone. This favorable outcome is consistent with the usual clinical course of patients with SRD in the context of HDP. In most cases, conservative management leads to complete resolution[17]. The prognosis of HDP-associated cranial nerve palsy is generally favorable. As in our case, most patients make a full recovery. Nonetheless, a thorough workup, including brain imaging with CT or MRI, should be performed to exclude any underlying pathology such as intracranial lesions, vasculopathy, tumors, multiple sclerosis, and inflammation. Interventions such as botulinum toxin injections may promote faster functional or cosmetic recovery in persistent cases.

In summary, a young female patient with pregnancy complicated by severe pre-eclampsia developed left isolated abducens nerve palsy and bilateral SRD. Although rare, further investigation into the pathophysiology behind HDP-associated isolated cranial nerve palsy is warranted to better our understanding of the disease, as well as to improve overall patient care and management.

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Conflicts of Interest: Rajab A, None; **Melebary R,** None; **Badeeb N,** None.

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