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Thirdnerve Palsy in Preeclampsia and Eclampsia A Red Flag Complication

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Abstract: ***A 25-year-old primigravida at 37+4 weeks of gestation with severe preeclampsia and previously corrected pulmonary stenosis developed acute right third nerve palsy following emergency caesarean section. Clinical findings included diplopia, ptosis, and restricted ocular movements. Neuroimaging demonstrated narrowing of the cavernous segment of the right internal carotid artery and radiological features suspicious for right cavernous sinus thrombosis. Aneurysmal and other life-threatening structural causes were excluded. Management included blood pressure control, anticoagulation, corticosteroids, and multidisciplinary care. Progressive neurological recovery was observed during follow-up. This case highlights the importance of considering both preeclampsia-related vasculopathy and cavernous sinus thrombosis in the differential diagnosis of peripartum third nerve palsy and emphasises the value of prompt neuroimaging and coordinated multidisciplinary management.***

Keywords: Third nerve palsy; oculomotor nerve; preeclampsia; pulmonary stenosis; hypertensive disorders of pregnancy; peripartum period

1. Introduction

Preeclampsia is a pregnancy-specific hypertensive disorder complicating 3–5% of pregnancies worldwide and contributes to maternal mortality and morbidity [1,2]. The diagnosis is established by new-onset hypertension after 20 weeks of gestation, often accompanied by proteinuria and/or evidence of systemic end-organ dysfunction [1,2]. Neurological manifestations are well recognised and include headache, visual disturbance, seizures (eclampsia), hypertensive encephalopathy, and posterior reversible encephalopathy syndrome (PRES) [3,4].

Cranial nerve involvement in the context of preeclampsia are, however, exceptionally rare. While, Facial nerve palsy (Bell's palsy) is the most predominantly reported, in hypertensive disorder of pregnancy. Isolated abducens (sixth nerve) palsy has been documented occasionally series, In the peer reviewed literature while third nerve (oculomotor) palsy complicating preeclampsia represents one of the rarest cranial neuropathies in pregnancy [5,6,7].

The pathophysiological basis for cranial nerve palsy in preeclampsia is not fully understood but postulated to involve disordered cerebral autoregulation, focal reactive vasoconstriction with ischaemia, or hyperperfusion with blood–brain barrier disruption [6,7]. Third nerve palsy is marked by diplopia, ptosis, exotropia with hypotropia, and ophthalmoplegia affecting adduction, elevation, and depression of the affected eye. Parasympathetic involvement causes pupillary dilatation and impaired accommodation [8]. When pupil-involving, the differential diagnosis mandates urgent exclusion of posterior communicating artery aneurysm or cavernous sinus pathology [8]. Preeclampsia is also associated with a prothrombotic state that can precipitate cerebrovascular thrombosis, including cavernous sinus thrombosis, adding further complexity to the differential diagnosis.

We present an unusual case of right third nerve palsy in a primigravida with severe preeclampsia and background corrected pulmonary stenosis, with CT angiographic and venographic findings suspicious of right cavernous sinus thrombosis. The case is notable for its diagnostic complexity, multi-specialist management, and the additional comorbidity of structural heart disease.

2. Case Report

A 25-year-old primigravida (POG 37+4 weeks) with a history of severe pulmonary stenosis treated by balloon pulmonary valvuloplasty 10 years earlier presented at term with a 2-day history of right-sided headache and 5 episodes of vomiting non projectile. She had gestational hypertension diagnosed at 21 weeks and was onTab. labetalol 100 mg 1-0-1daily.

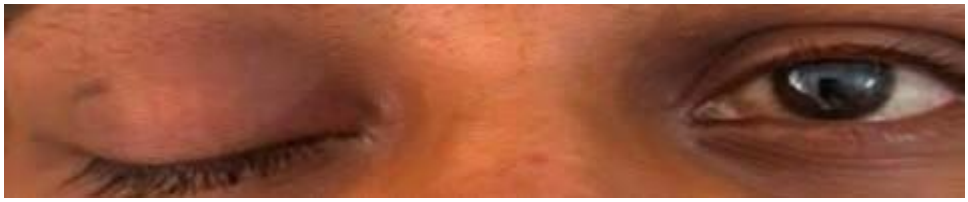
On admission, blood pressure was 150/100 mmHg with symptoms suggestive of imminent eclampsia. Cardiac evaluation during pregnancy had demonstrated residual pulmonary valve stenosis with a gradient of 43–47 mmHg, trivial tricuspid regurgitation, preserved biventricular function (LVEF 60%, TAPSE 20 mm), and no pulmonary hypertension. Laboratory investigations showed normal Haemogram, platelet count, liver and renal function tests, coagulation profile, and LDH levels.

On admission, she was hypertensive (150/100 mmHg) with clinical features concerning for impending eclampsia. Laboratory evaluation excluded HELLP syndrome and other major end-organ dysfunction. Following stabilization with antihypertensive therapy and magnesium sulphate seizure prophylaxis, a multidisciplinary decision was made to proceed with emergency LSCS under spinal anaesthesia,

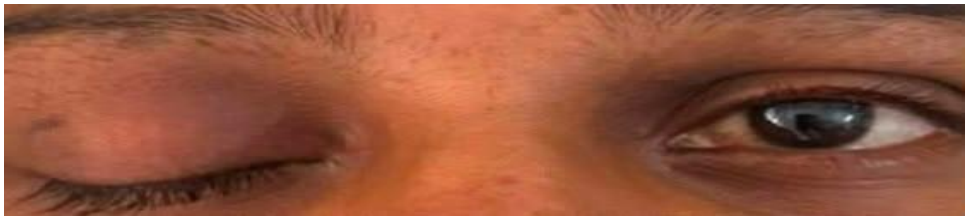
delivered a healthy neonate weighing 2.34 kg with favourable neonatal outcomes.

The early postoperative period was notable for the onset of right-sided ptosis, periorbital discomfort, diplopia, lacrimation, and progressive ophthalmoplegia. Neurological assessment confirmed a complete right oculomotor nerve palsy with pupillary involvement. Given the atypical neurological presentation, urgent neuroimaging was undertaken. MRI and angiography demonstrated asymmetrical prominence of the right cavernous sinus with associated attenuation of the cavernous segment (C3–C6) of the right internal carotid artery. Subsequent CT angiography and venography revealed a bulky hypoenhancing right cavernous sinus with ipsilateral ICA narrowing, radiologically favouring cavernous sinus thrombosis.

The patient underwent multidisciplinary management involving obstetrics, neurology, ophthalmology, cardiology, and internal medicine teams. Treatment comprised systemic corticosteroids, therapeutic anticoagulation with low-molecular-weight heparin followed by rivaroxaban, antiplatelet therapy, neurotrophic supplementation, and supportive ophthalmic care. Her clinical course remained haemodynamically stable without progression of neurological deficits. She was discharged on postoperative day 11 with partial symptomatic improvement and scheduled for close multidisciplinary follow-up. A favourable clinical course was noted during followup. By POD-27 the patient exhibited marked recovery and remained stable on conservative management. Written informed consent was obtained from the patient for publication of this case report and the accompanying clinical photographs.



Photograph on Post operative day -5



Photograph at the time of Discharge day -11

Photograph on Post operative day- 27

3. Discussion

Course Of Third cranial nerve: Midbrain origin > Subarachnoid course alongside PCoA> Cavernous sinus wall > Superior Orbital fissure > Orbital branches and pupil fibres

Why Prone in Pre Eclamsia?

1) Vasospasm/ Microvascular ischaemia
2) Hyperperfusion/ PRES Oedema
3) Cavernous Sinus thrombosis
4) PCoA Aneursyms.

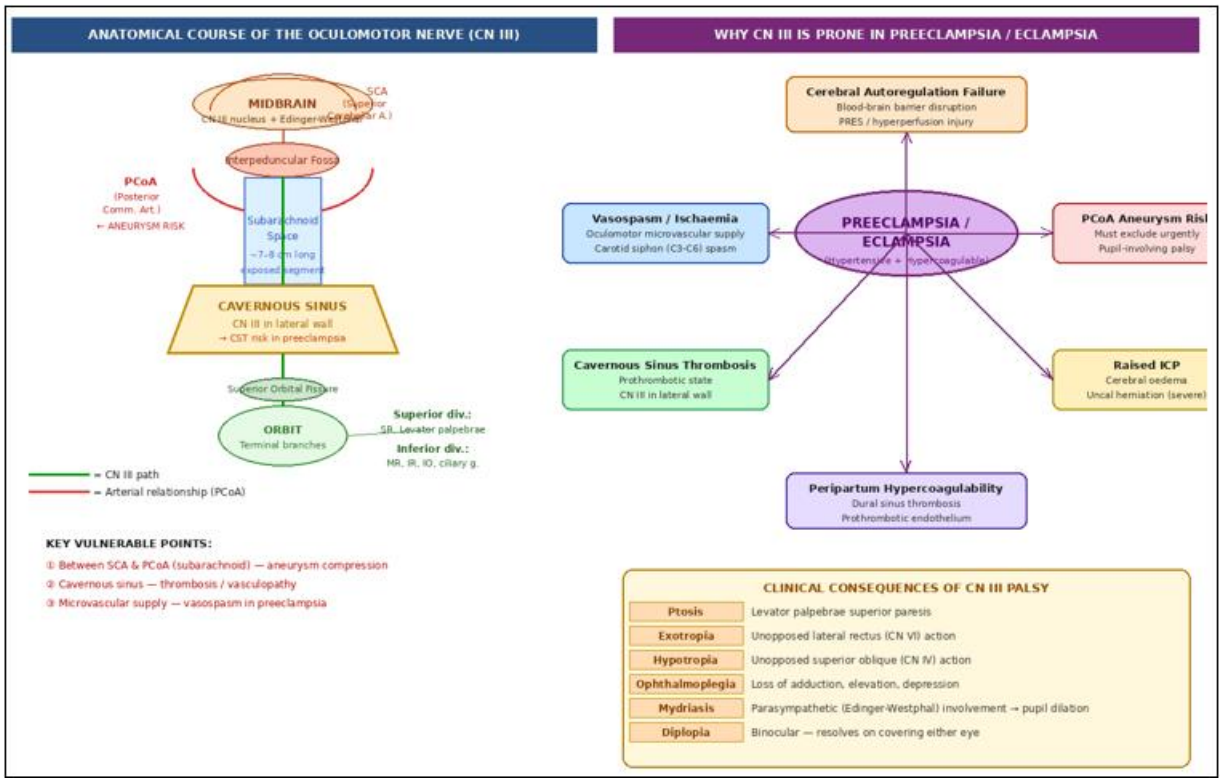


Figure 1: Anatomical course of the oculomotor nerve (CN III) and pathophysiological mechanisms of injury in preeclampsia/eclampsia. *Left panel:* Stepwise anatomical course from midbrain nucleus through the subarachnoid space, cavernous sinus, and superior orbital fissure to terminal orbital branches, highlighting key vulnerable points (numbered). *Right panel:* Pathophysiological mechanisms by which preeclampsia/eclampsia predisposes to CN III palsy, with resultant clinical signs. CN III = oculomotor nerve; PCoA = posterior communicating artery; PCA = posterior cerebral artery; SCA = superior cerebellar artery; CST = cavernous sinus thrombosis; ICA = internal carotid artery; PRES = posterior reversible encephalopathy syndrome.

Several features make this case particularly noteworthy in the context of the published literature. Third nerve palsy complicating preeclampsia is exceedingly rare; fewer than fifteen cases have been reported in the peer-reviewed literature [8,11,13,14]. Shenhav and Khalifeh described an isolated oculomotor palsy resolving after delivery, attributing it to microvascular vasospasm of the nerve's nutrient vessels [12]. Wilkinson and Sivakumar reviewed available cases and concluded that vasospasm, compounded by disordered cerebral autoregulation, is the predominant mechanism, though they acknowledged that thrombotic and oedematous contributions may coexist [13]. Kumar and Prasad noted that cranial neuropathies in hypertensive disorders of pregnancy are under-recognised and may involve ischaemic or hyperperfusion injury depending on which mechanism predominates in the individual case [14]. Neuroimaging — including MR angiography and CT angiography/venography — was therefore essential and appropriately performed, allowing aneurysmal causes to be excluded and cavernous sinus pathology to be identified. Sebastian et al. similarly highlighted the importance of systematic neuroimaging in cranial nerve palsies complicating hypertensive disorders of pregnancy, noting that atypical features such as pupil involvement or failure to resolve promptly should always prompt dedicated vascular imaging [15].

To our knowledge, the present case is among the first to provide radiological documentation of cavernous sinus pathology- a bulky right cavernous sinus with hypoenhancement on CT venography- as a probable contributory mechanism, an observation that aligns with the description of CN III's intimate course through the cavernous sinus lateral wall and its consequent vulnerability to compressive or thrombotic injury at that site [8].

The diagnostic approach in this case warrants specific comment. Biousse and Newman stress that any pupil-involving third nerve palsy mandates urgent exclusion of a posterior communicating artery aneurysm before a benign vascular or preeclamptic aetiology is accepted, as aneurysmal third nerve palsy may spontaneously decompress after delivery and be erroneously attributed to the hypertensive disorder [8].

The additional comorbidity of corrected pulmonary stenosis introduced specific management challenges. Continued beta-blockade was necessary to maintain cardiac output across the residual pulmonary gradient, yet this had to be balanced against the haemodynamic demands of preeclampsia management, ultimately necessitating a transition to amlodipine–atenolol combination therapy. The risk of neuraxial anaesthesia was considered acceptable given preserved cardiac function on echocardiography (LVEF 60%, no PAH), and spinal anaesthesia was administered successfully. NICE guidance on hypertensive disorders of pregnancy underscores the need for close multidisciplinary involvement when concurrent cardiac pathology is present, and this case illustrates those principles in practice [9].

From a pathophysiological standpoint, the narrowing of the right internal carotid artery at the C3–C6 (cavernous) segments on both MRI and CT angiography points to focal vasospasm of the carotid siphon, a recognised feature of severe preeclampsia and hypertensive encephalopathy [10]. Simultaneously, the peripartum hypercoagulable state- characterised by platelet activation, endothelial dysfunction, and heightened procoagulant activity- may have precipitated thrombosis within the right cavernous sinus, directly compressing CN III. Sibai has emphasised that the prothrombotic endothelial milieu of preeclampsia can persist into the postpartum period, amplifying the risk of cerebrovascular and venous sinus thrombotic events even after delivery [10]. The coexistence of vasospasm and thrombosis at the cavernous segment likely accounts for the severity and pupil involvement seen in this patient.

Management in this case appropriately integrated multispecialty inputs. Anticoagulation with low-molecular-weight heparin (enoxaparin) followed by oral rivaroxaban addressed the thrombotic component; corticosteroids (dexamethasone and prednisolone) targeted perilesional oedema and inflammation; neurotropic supplementation (B-complex vitamins) supported axonal recovery; and ocular exercises facilitated functional rehabilitation. Kumar and Prasad note that outcomes of cranial neuropathy in hypertensive disorders of pregnancy are generally favourable when blood pressure is controlled and the underlying mechanism addressed promptly [13].

The clinical trajectory at discharge- stable vital signs and ongoing anticoagulation- was encouraging, though long-term follow-up remains necessary. The entire clinical picture underscores Wilkinson and Sivakumar's conclusion that a coordinated multidisciplinary approach encompassing obstetrics, neurology, ophthalmology, and imaging is indispensable in the management of peripartum cranial nerve palsies [14].

All reported cases of preeclampsia-associated third nerve palsy have shown favourable neurological outcomes with spontaneous recovery following blood pressure normalisation and delivery [14]. Future reporting of similar cases with detailed neuroimaging will be important to better characterise the frequency and spectrum of cavernous sinus involvement in peripartum cranial neuropathy [14,15].

4. Conclusion

Third nerve palsy is a rare neurological complication of severe preeclampsia that requires prompt recognition and investigation. This case suggests that both preeclampsia-related vasculopathy and cavernous sinus thrombosis may contribute to oculomotor nerve dysfunction in the peripartum period. Comprehensive neuroimaging is essential to exclude aneurysmal and thrombotic causes, while multidisciplinary management can facilitate favourable neurological recovery. Additional case reports and longitudinal studies are needed to better define the spectrum and outcomes of cavernous sinus involvement in hypertensive disorders of pregnancy.

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