RESEARCH PAPER

Unravelling the "Long Course": A Case Series of Sixth Nerve Palsy

Dr. Athul M[1], Dr. Arvind L Tenagi[2], Dr. Samyakta Shetti[3], Dr Rohini D K[4], Dr. Goudigama Preethi[5]

[1]Junior Resident, Department of Ophthalmology, KAHER's Jawaharlal Nehru Medical College, Belagavi
[2]Professor, Department of Ophthalmology, KAHER's Jawaharlal Nehru Medical College, Belagavi
[3]Assistant Professor, Department of Ophthalmology, KAHER's Jawaharlal Nehru Medical College, Belagavi
[4]Assistant Professor, Department of Ophthalmology, KAHER's Jawaharlal Nehru Medical College, Belagavi
[5]Junior Resident, Department of Ophthalmology, KAHER's Jawaharlal Nehru Medical College, Belagavi
[1]Email: athulm869@gmail.com, [2]Email: drtenagi@yahoo.com, [3]Email: samyaktashetti@yahoo.com and Email: [4]rohinidk90@gmail.com ,Email: [5]goudigamapreethi3036@gmail.com

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ABSTRACT

Objective: The sixth (abducens) cranial nerve has the longest intracranial course of any cranial nerve, rendering it vulnerable to injury at multiple anatomical sites – from its nucleus in the dorsal pons to its termination in the lateral rectus muscle. We present a case series of five patients with sixth nerve palsy of varied aetiology, to illustrate the spectrum of underlying causes encountered along this "long course" and to outline a structured approach to clinical evaluation.

Methods: This is a descriptive case series of five patients who presented to the Department of Ophthalmology with acute-onset diplopia and restriction of ocular abduction. Each patient underwent a detailed history, complete ocular and systemic examination including cranial nerve assessment, diplopia charting, relevant haematological and biochemical work-up, and neuroimaging (MRI brain, with or without contrast/angiography) as clinically indicated.

Results: The five cases illustrated distinct underlying mechanisms of sixth nerve palsy: presumed microvascular/ischaemic palsy in an elderly patient, a cerebellopontine angle tumour presenting with combined sixth, seventh and eighth cranial nerve involvement, two cases of trauma-related (direct and indirect) abducens nerve palsy, and a case associated with focal dural thickening suggestive of hypertrophic pachymeningitis a young diabetic woman. Diplopia charting and neuroimaging were central to establishing the diagnosis in each case, and management was tailored to the underlying cause, ranging from conservative and medical management to neurosurgical referral.

Conclusion: Sixth nerve palsy is not a single diagnostic entity but a common clinical endpoint of insults occurring anywhere along the nerve's long anatomical course. A systematic evaluation – detailed history, cranial nerve examination, diplopia charting, and judicious neuroimaging – is essential to identify the underlying cause, as this determines both management and prognosis.

Keywords: *Abducens nerve; sixth cranial nerve palsy; diplopia; case series; cranial neuropathy.*

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INTRODUCTION

The abducens (sixth cranial) nerve innervates the lateral rectus muscle and is responsible for abduction of the eye. Its nucleus lies in the dorsal pons, close to the fibres of the facial nerve as they loop around it a relationship that explains why lesions in this region often produce combined sixth and seventh nerve signs [1]. From the pontomedullary junction, the nerve traverses a long subarachnoid (cisternal) course, angles sharply over the petrous apex of the temporal bone, passes through Dorello's canal to enter the cavernous sinus, and finally runs through the superior orbital fissure to reach the orbit and the lateral rectus muscle [2]. Because of this unusually long and vulnerable intracranial trajectory, the sixth nerve is more susceptible than any other ocular motor nerve to a wide range of insults vascular, neoplastic, traumatic, infective, inflammatory, and raised intracranial pressure occurring at very different points along its path [3].

Isolated abducens nerve palsy is reported to occur at a rate of approximately 11.3 per 100,000 population and is the most common isolated ocular motor cranial neuropathy in adults, typically affecting patients between 60 and 70 years of age. Microvascular ischaemia related to diabetes mellitus, hypertension, and other atherosclerotic risk

*Author for Correspondence: Dr. Athul M

factors is consistently reported as the leading identifiable cause in adults, particularly in older patients, while trauma, neoplasms, demyelinating and inflammatory conditions, aneurysms, and raised intracranial pressure account for a substantial proportion of the remaining cases and tend to occur in a younger demographic. Clinically, the presentation is often deceptively uniform horizontal, binocular diplopia worse on gaze towards the affected side, with esotropia and limitation of abduction – regardless of the underlying cause, making a structured diagnostic approach essential [4,5].

We describe five patients with sixth nerve palsy evaluated in our department, chosen to illustrate this diversity of aetiology along the nerve's long course, and discuss the clinical clues, investigative approach, and outcomes relevant to each.

Case Reports

Case 1: Presumed Microvascular (Ischaemic) Sixth Nerve Palsy

A 55-year-old woman, a manual labourer by occupation, presented with sudden-onset, painless, non-progressive inward deviation of the right eye of one month's duration, associated with diminution of vision and a mild dull-aching right-sided headache. She was a known case of seizure disorder for the past 10 years, maintained on tablet phenobarbitone 20 mg once daily, but had no history of hypertension, diabetes mellitus, thyroid disease, trauma, or ocular surgery [6].

On examination, she was conscious, oriented, and haemodynamically stable. Cranial nerve examination revealed isolated limitation of abduction of the right eye, with the rest of the cranial nerves, including facial and auditory nerves, being normal [7]. Ocular examination showed a head turn towards the right, right esotropia of approximately 35–45 prism dioptres on prism bar cover test, and grade -4 limitation of abduction of the right eye on extraocular movement assessment. Diplopia charting confirmed uncrossed horizontal diplopia, maximal in dextroversion. Visual acuity was reduced in the right eye due to an underlying immature senile cataract (nuclear sclerosis grade II); anterior segment and fundus examination were otherwise unremarkable bilaterally [8].

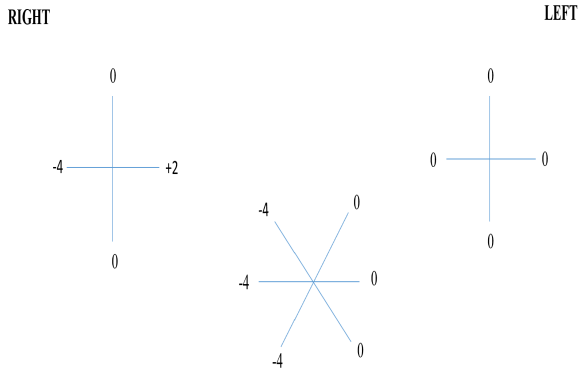
Investigations, including complete blood count, liver and renal function tests, thyroid function, HbA1c (6.1%), random blood sugar, and viral markers, were essentially within normal limits, without an alternative systemic explanation. MRI of the brain (plain) did not reveal an intracranial structural lesion. Neurology opinion was sought, and the patient was managed with low-dose aspirin, a short tapering course of oral corticosteroid, neuroprotective and lubricating agents, alongside continuation of her antiepileptic medication [9].

A working diagnosis of right sixth cranial nerve palsy, probably secondary to microvascular/ischaemic aetiology, was made in the setting of an unremarkable neuroimaging and metabolic work-up, with age being the principal identifiable risk factor. She was planned for close outpatient follow-up to monitor for spontaneous recovery, typically expected over 2–3 months.

Palpebral Aperture

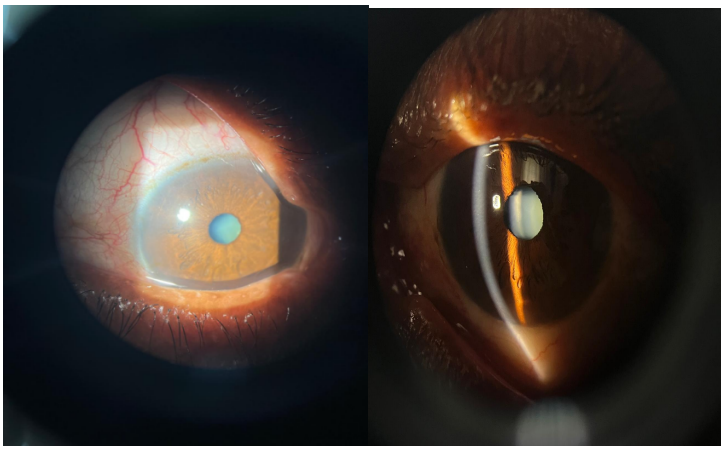
	OD	OS
VERTICAL	10 mm	10 mm
HORIZONTAL	28 mm	28 mm

Extra ocular movements

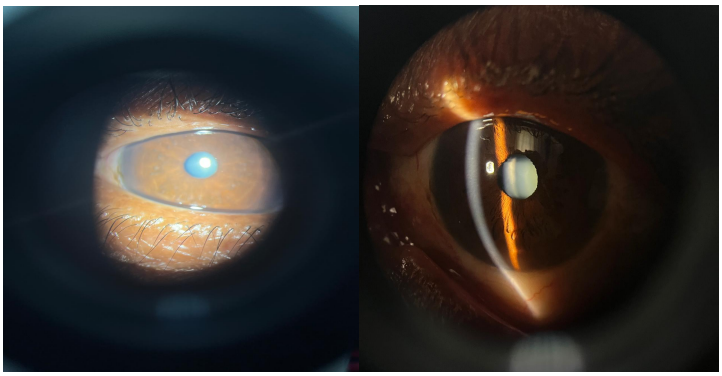


Extra ocular movements

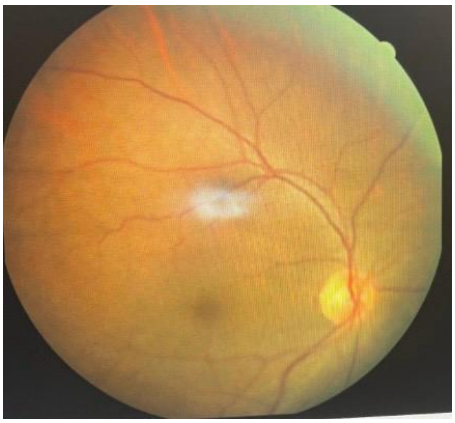
Anterior segment : OD



Anterior segment:OS



Fundoscopic examination by indirect ophthalmoscopy: OD



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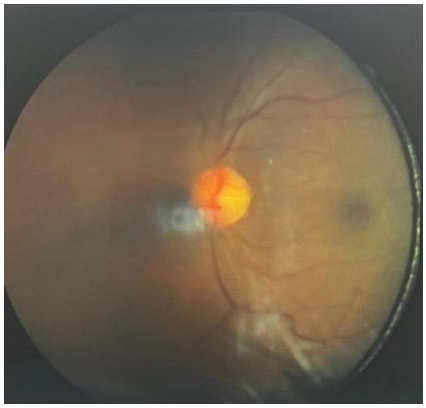


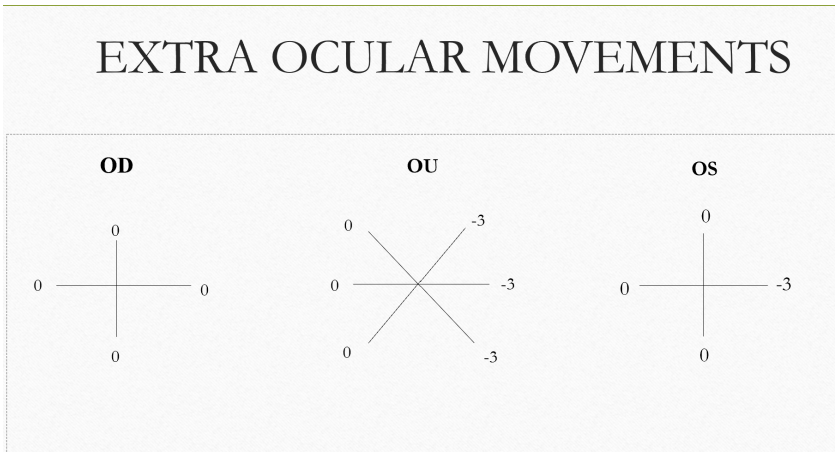
Case 2: Cerebellopontine Angle Tumour with Combined VI, VII, VIII Nerve Palsy

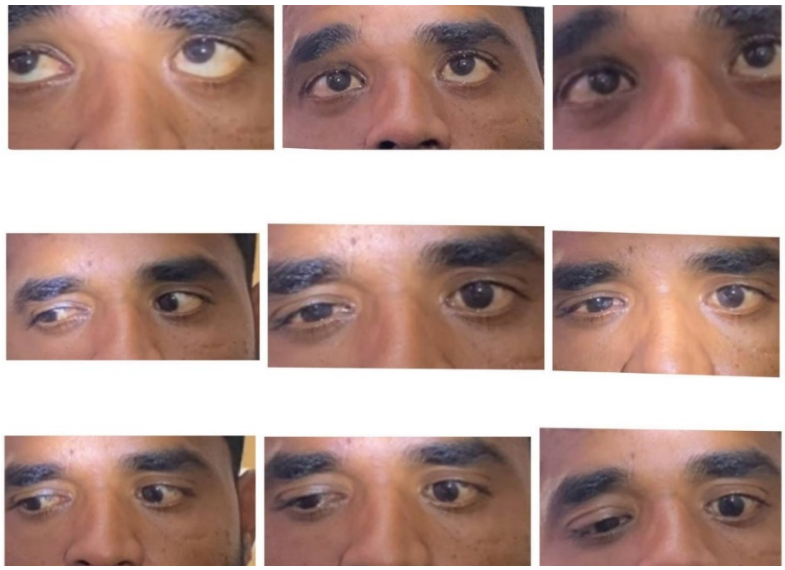
A 36-year-old male lighting decorator presented with a 15-day history of occipital headache and neck pain, progressing over the following days to left-sided ear pain, and subsequently, over the preceding week, to horizontal diplopia on left gaze, inability to close the left eye, right-sided deviation of the angle of the mouth, and left-sided hearing impairment. There was an associated episode of raised blood pressure and projectile vomiting for which he was evaluated locally; an MRI brain performed at that time raised suspicion of a nerve sheath tumour, prompting referral to our institution [10].

On examination, cranial nerve assessment revealed a left lower-motor-neuron type facial palsy (reduced forehead wrinkling, incomplete eye closure, flattened nasolabial fold, absent corneal reflex on the left), and left-sided sensorineural hearing loss with an abnormal Weber's and Rinne's test pattern and a positive Romberg's sign with swaying to the left. Ocular examination showed a left head tilt, left esotropia, and grade 3 limitation of abduction of the left eye with a corresponding grade 3 limitation on levoversion. Diplopia charting confirmed uncrossed horizontal diplopia, worse in levoversion [11].

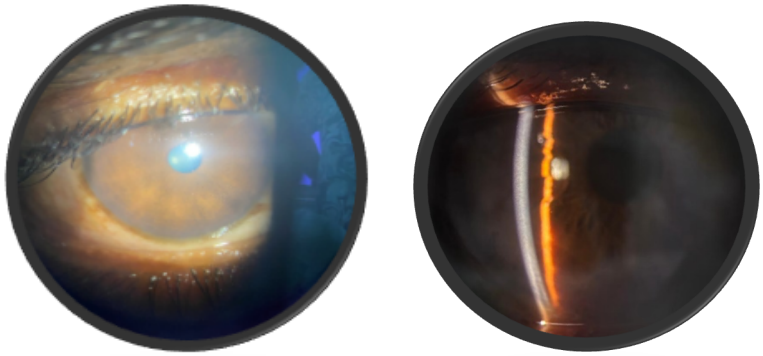
Contrast-enhanced MRI of the brain demonstrated a peripherally enhancing soft-tissue-intensity lesion in the left cerebellopontine angle with irregular haemorrhagic areas, for which tumour bleed and an arteriovenous malformation were considered as differentials, and MR angiography was recommended for further characterisation. Brainstem auditory evoked response testing confirmed an abnormal central pathology pattern on the left, with no measurable response even at high intensity, corroborating the profound sensorineural hearing loss [12].

A diagnosis of left lateral rectus (sixth cranial nerve) palsy with ipsilateral seventh and eighth cranial nerve involvement, secondary to a left cerebellopontine angle tumour, was made. The combination of the sixth, seventh, and eighth nerve deficits localised the lesion to the cerebellopontine angle/pontine region, and the patient was managed conservatively in the interim with ocular lubrication and lid taping at night, pending neurosurgical evaluation and definitive management of the tumour, with planned monthly follow-up [13].

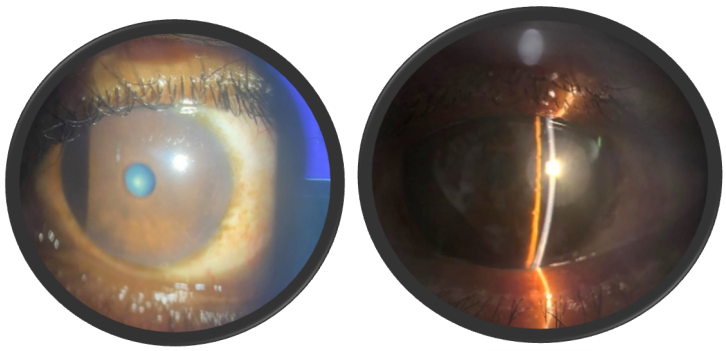




ANTERIOR SEGMENT – OD



ANTERIOR SEGMENT – OS



Case 3: Traumatic Sixth Nerve Palsy Following Road Traffic Accident

A 32-year-old male manual labourer presented with diplopia and inward deviation of the right eye of one month's duration, following blunt trauma to the right eye sustained in a road traffic accident approximately one month earlier. There was no history of fever, diurnal variation of symptoms, or other neurological deficits, and systemic examination was unremarkable [14].

Ocular examination revealed a face turn towards the right side, symmetrical facial features, and right esotropia of 45 prism dioptres with limitation of abduction of the right eye; the left eye was orthophoric with full ocular movements. Visual acuity was 6/6 in both eyes with correction. Anterior segment examination of the right eye showed the residual esotropia, while the fundus was within normal limits bilaterally. Diplopia charting demonstrated uncrossed horizontal diplopia, more pronounced in dextroversion [15].

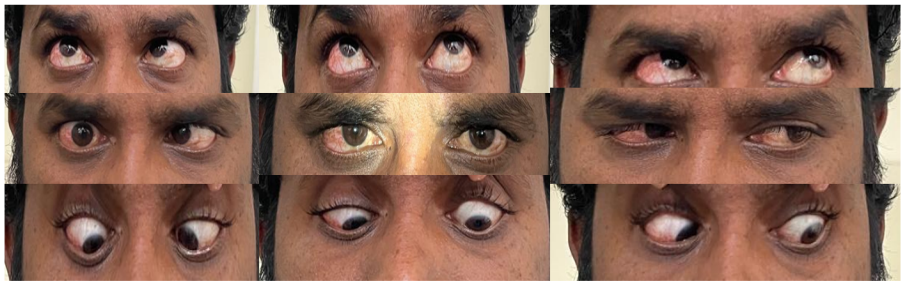
A diagnosis of right sixth cranial nerve palsy secondary to trauma was made on the basis of the temporal relationship to the road traffic accident and the absence of any alternative vascular, infective, or neoplastic cause on evaluation. Traumatic abducens nerve palsy is thought to result from stretching or contusion of the nerve along its long, fixed intracranial course – particularly at the point where it angles over the petrous apex and passes through Dorello's canal – making it particularly susceptible to shearing forces during head trauma even without an associated skull-base fracture [16].

Ocular Examination

Head posture	Face turn toward right side
Facial symmetry	symmetrical
Visual axis	unparallel ,RE:45 degree esotropia
Ocular posture	RE :inward deviation(esotropia) LE :normal

Visual acuity	6/6(p) →6/6	6/6(p) →6/6
Anterior Segment	45” esotropia	Within normal limit
Fundus	Within normal limit	Within normal limit

Diplopia charting:Uncrossed horizontal diplopia, more in dextroversion.



Case 4: Indirect Traumatic Sixth Nerve Palsy Following Ocular Trauma

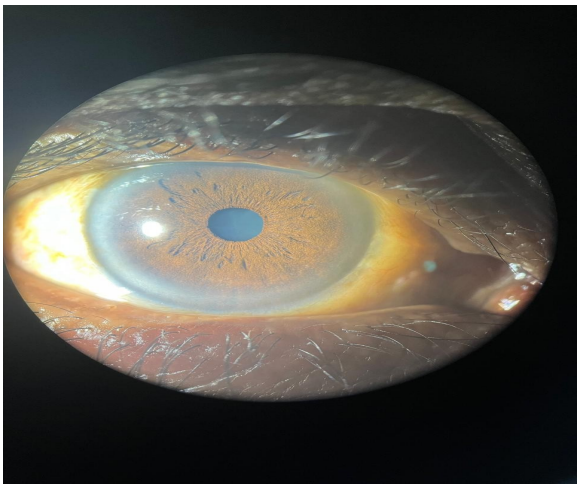
A 50-year-old male presented with deviation of the left eyeball and diminution of vision in the left eye of one month's duration. He gave a history of blunt trauma to the left eye with sugar cane leaves approximately one month prior, associated with pain that was managed with oral analgesics and multivitamins at a local facility, following which the pain subsided; there was no associated diplopia at the time of injury, no history of ocular surgery, and he

was not a known case of hypertension, diabetes, or thyroid disorder. He reported a long-standing history of tobacco chewing [17].

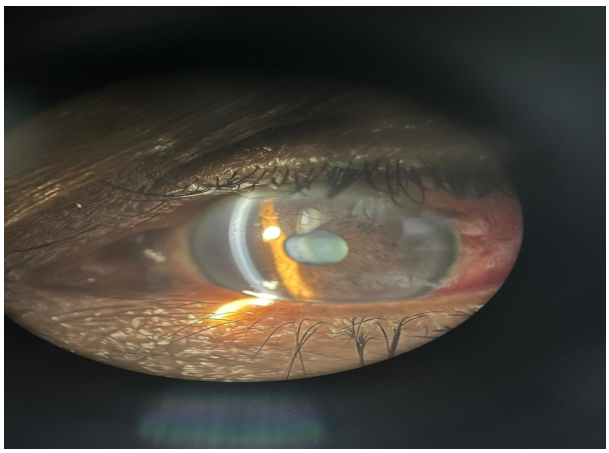
On examination, he had a face turn/head tilt towards the paralysed eye with asymmetrical facial posture; the visual axis was not parallel, with left esotropia and additional upward deviation. Extraocular movement assessment confirmed a grade -4 limitation of abduction of the left eye. The Worth four-dot test showed the patient perceiving three green dots, with the red target misinterpreted as white, in keeping with suppression. Ice-pack and fatigue tests were negative, arguing against a myasthenic component [18].

Haematological and biochemical work-up, including HbA1c (5%), random blood sugar, renal and liver function tests, thyroid profile, and viral markers, were unremarkable. MRI of the brain (plain and contrast) did not identify any significant intracranial pathology. Neurosurgical opinion was sought, and in the absence of a demonstrable structural or vascular cause, a diagnosis of indirect traumatic abducens nerve palsy/neuropathy was made, attributable to the antecedent blunt ocular trauma. Steroids were not advised in view of the time elapsed since injury; the patient was managed with intramuscular and intravenous multivitamin/neurobion supplementation and ocular lubricants, with plans for continued outpatient follow-up [19,20].

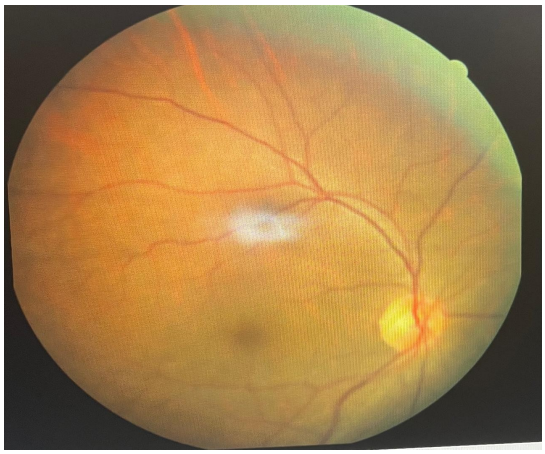
Anterior segment OD



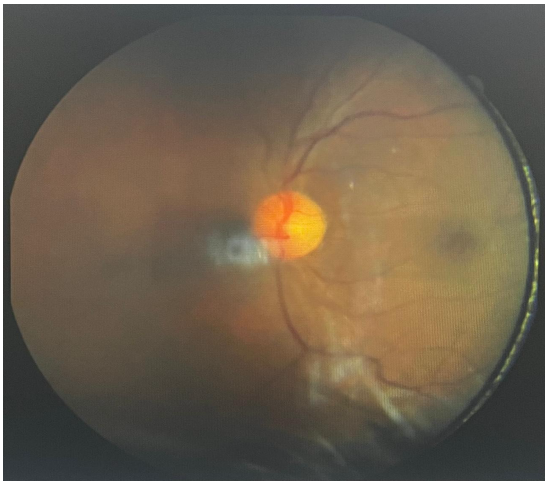
Anterior segment OS



Fundoscopic examination by indirect ophthalmoscopy: OD



Fundoscopic examination by indirect ophthalmoscopy: OS

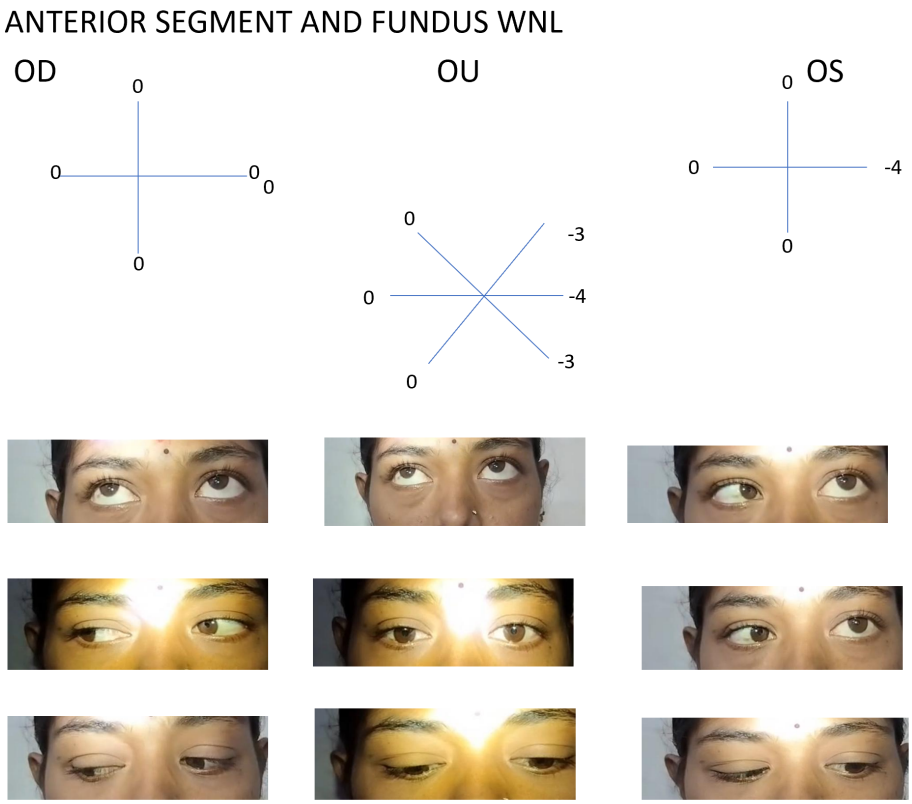


Case 5: Sixth Nerve Palsy Associated with Focal Dural Thickening in a Young Diabetic

A 29-year-old woman, a known case of type 2 diabetes mellitus of four years' duration on treatment, presented with diplopia in the left eye of one month's duration, associated with headache for the preceding five days. She gave a history of spectacle use but no history of ocular trauma or surgery [21].

Visual acuity was 6/12 improving to 6/6 with pinhole in the right eye and 6/6(p) in the left eye, with normal colour vision bilaterally. Anterior segment and fundus examination were within normal limits in both eyes. Extraocular movement assessment revealed restriction of abduction of the left eye, and diplopia charting confirmed uncrossed horizontal diplopia [22].

MRI of the brain revealed sheet-like focal thickening with enhancement of the dura in the right fronto-temporal region, along with a few non-specific ischaemic changes in the bilateral peritrigonal, periventricular, and fronto-parieto-temporo-occipital white matter findings suggestive of focal hypertrophic pachymeningitis, a recognised though uncommon cause of isolated ocular motor nerve palsy through direct dural/nerve involvement or secondary compressive/inflammatory effect [23,24]. A diagnosis of left lateral rectus (sixth cranial nerve) palsy, in the setting of focal dural thickening and background diabetic microvascular changes, was made, and the patient was planned for further work-up to characterise the dural process (including inflammatory and infective screening) and glycaemic optimisation, with close interval follow-up [25].



Summary of Cases

Case	Age/Sex	Side	Presentation	Key Association	Aetiology
1	55F	Right	Painless esotropia, diminution of vision, headache – 1 month	Seizure disorder (10 yrs); no vascular risk factors identified	Presumed microvascular/ischaemic
2	36M	Left	Headache, ear pain, diplopia, facial weakness, hearing loss	Left CPA lesion (nerve sheath tumour with haemorrhage)	Neoplastic (CPA tumour) – combined VI, VII, VIII palsy
3	32M	Right	Diplopia, esotropia – 1 month post road traffic accident	History of blunt ocular/head trauma	Traumatic (direct)
4	50M	Left	Ocular deviation, diminution of vision – 1 month post blunt trauma	Trauma with sugar cane leaves; tobacco chewer	Traumatic (indirect)
5	29F	Left	Diplopia, headache – 1 month	Type 2 diabetes mellitus (4 yrs); dural thickening on MRI	Hypertrophic pachymeningitis / dural process

DISCUSSION

The five cases in this series illustrate why the sixth cranial nerve has been described as having a particularly “long course” from a diagnostic standpoint not merely anatomically, but in the breadth of pathology that must be considered before a cause can be assigned [6,7]. In a prospective series of 36 isolated abducens nerve palsy patients, Erdal et al. found microvascular causes to be the single most common aetiology, accounting for 44.4% of cases, with the microvascular group being significantly older (mean age 62.8 vs. 44.5 years) and more likely to have diabetes mellitus than patients with non-vascular causes; notably, diabetes rather than hypertension emerged as the dominant vascular risk factor in that cohort, and clinical recovery did not differ significantly between microvascular and other causes, with an overall mean recovery time of about 2.5 months [2]. Our Case 1, an elderly woman with an unremarkable metabolic and imaging work-up, fits this well-recognised presumed-microvascular pattern, where a normal MRI and the

absence of an alternative cause rather than a single confirmatory test – support the diagnosis [8,9].

By contrast, Cases 3 and 4 represent the trauma end of the spectrum, one following a road traffic accident and the other an indirect ocular injury without a fracture [14-17]. The abducens nerve's fixed course over the petrous apex and through Dorello's canal renders it especially prone to stretch injury during acceleration-deceleration trauma, and indirect traumatic abducens palsy without a demonstrable skull-base fracture on imaging is a recognised, if under-reported, entity, as seen in Case 4. Both patients were managed conservatively, in keeping with the generally favourable natural history of traumatic abducens palsy once intracranial and orbital structural causes have been excluded [18-20].

Case 2 illustrates the importance of a complete cranial nerve examination in every patient with abducens palsy: the co-existing facial and vestibulocochlear involvement immediately localised the lesion to the cerebellopontine angle rather than the orbit or cavernous sinus, and neuroimaging confirmed a mass lesion requiring neurosurgical input. This underscores a broader point echoed in the literature that neuroimaging is warranted even when a vascular risk-factor profile is present, since a proportion of patients presumed to have vascular disease on clinical grounds alone are subsequently found to harbour a structural cause [10-13]. Case 5 similarly reinforces this principle: despite a background of diabetes mellitus that could plausibly explain an isolated sixth nerve palsy on clinical grounds, MRI identified focal dural thickening and enhancement, redirecting management towards evaluation for an inflammatory or infiltrative dural process rather than presumed microvascular disease alone [21,22].

Across this series, diplopia charting was uniformly useful in confirming an uncrossed horizontal pattern worse in the direction of the affected lateral rectus, supporting the clinical diagnosis of a sixth nerve palsy in each case, while neuroimaging rather than laboratory testing alone proved decisive in distinguishing presumed microvascular disease from structural, traumatic, and dural aetiologies. Taken together, these cases support a low threshold for MRI brain (with contrast where indicated) in patients presenting with sixth nerve palsy, particularly outside the classic elderly, multiple-vascular-risk-factor demographic, or where atypical features additional cranial neuropathies, headache out of proportion, or a history of trauma are present [23-25].

CONCLUSION

This case series highlights the diverse aetiological spectrum of sixth nerve palsy encountered along the nerve's long and vulnerable intracranial course – spanning presumed microvascular disease, neoplasia, direct and indirect trauma, and dural pathology. While presumed microvascular palsy remains the most common cause, particularly in older patients with vascular risk factors, a systematic evaluation combining detailed history, complete cranial nerve examination, diplopia charting, and judicious neuroimaging remains essential in every patient, as clinical presentation alone cannot reliably distinguish between these aetiologies. Early identification of the underlying cause is critical, as it directly determines management – ranging from conservative observation to urgent neurosurgical referral and ultimately, visual and neurological outcome.

ETHICS STATEMENT:

Informed consent was obtained from all patients for the use of their clinical details and relevant images for academic and educational purposes, with confidentiality of identity maintained throughout.

CONFLICT OF INTEREST:

The authors declare no conflict of interest.

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