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Abducens Nerve Palsy: A Case Report

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ABSTRACT:

The most prevalent ocular motor nerve palsy, abducens nerve palsy, is frequently associated with microvascular reasons in diabetics. We describe a 52-year-old man who developed hazy vision, binocular horizontal diplopia, with worsening on right gaze one week prior. The patient had a known history of diabetes mellitus but no hypertension or recent infection. Ocular examination revealed restricted right lateral gaze and correctable refractive errors. Fundoscopy showed bilateral drusens and dull foveal reflexes, indicating early macular changes. MRI revealed a calcified granuloma in the left frontal lobe, an incidental finding. The patient was managed with neuroprotective agents, antiplatelets, statins, and medications to improve microcirculation. Clinical signs resolved over four months, consistent with a microvascular aetiology. This case highlights the need for thorough systemic and ocular evaluation in cranial nerve palsies and supports the potential for full recovery with appropriate medical management in diabetic patients.

Keywords: Abducens nerve palsy, Drusens, Diplopia, Sixth cranial nerve, Granuloma, Motoneurons

1. Introduction:

Among ocular motor nerve palsy, abducens nerve palsy is the most common.(1) Although these palsies typically go away, they frequently reoccur. In older persons, sixth cranial nerve palsy is most commonly caused by presumed microvascular disease. The hallmarks of these microvascular mononeuropathies include function recovery within two to three months of the event's acute onset, normal neuroimaging findings if any, and usually a history of hypertension, diabetes, hypercholesterolemia, tobacco use, or a combination of these conditions.(2)

Eye abduction is accomplished by the ipsilateral lateral rectus, which is innervated by the sixth cranial nerve, the abducens nerve. Motoneurons begin in the caudal dorsal pons' abducens nucleus, leave the nucleus ventrally to generate the brainstem's abducens fascicle, and then exit the brainstem at the pontomedullary junction to enter the subarachnoid space. The nerve then abruptly twists and climbs over the clivus and petrous apex. It passes beneath the Petro clival ligament (including the Gruber ligament) and pierces Dorello's canal. It is susceptible to injury and changes in intracranial pressure because of dural tethering. Before proceeding down the ophthalmic division of the trigeminal nerve, sympathetic fibers momentarily follow the abducens nerve in the cavernous sinus. When the abducens nerve is injured, the damaged lateral rectus acts in a poorer direction, causing unilateral impairment of abduction, binocular horizontal diplopia, and eso-deviation (eye tilted in toward the nose).(3)

2. Case Presentation:

After experiencing binocular diplopia for a week, a 52-year-old man arrived at the emergency room. When one eye was rested or the left eye was looked at, the diplopia disappeared. He also reported blurred vision over the same duration. His visual issues were related to both myopia and hypermetropia and were not associated with redness, tearing, or itching. The patient had previously used eyeglasses but had been noncompliant for the past two months.

There was no history of fever, neck stiffness, tinnitus, nausea, vomiting, head trauma, recent infections, or travel. He was a known diabetic for the past six months and was on regular medication. He denied having a history of asthma, TB, convulsions, or hypertension. He reported a history of bilateral, intermittent headaches over the past year, which had worsened in the past week and were associated with photophobia and phonophobia.

General Examination:

Well-built and nourished

• BP: 120/80 mmHg

• Pulse: 80 bpm

• Respiratory Rate: 18/min

There was no family history of similar symptoms.

Right lateral rectus deficit was suggested by diplopia charting, which showed horizontal binocular diplopia that increased with right gaze (dextroversion). Diplopia reduced in left gaze and disappeared on monocular viewing.

Table 1: OCULAR EXAMINATION FINDINGS

Parameters	Right Eye	Left Eye
Visual Acuity (VA)	6/18	6/36
Pinhole (PH)	6/9	6/12
Near Vision (NV)	N12	N18
Aided VA	6/12	6/12
Aided NV	N10	N12
Autorefraction (AR)	+2.00 DS, +0.25 DC @ 2°	+2.25 DS
BCVA	+1.50 DS	+2.25 DS
BCNV Add	+2.50 DS	+2.50 DS
Lids	MGD, papillae	MGD, papillae
Cornea	Clear	Clear
Conjunctiva, Iris, Pupil	Normal	Normal
Lens	SIMC	SIMC
Extraocular Movements	Restriction in dextro-elevation, dextro-version, dextro-depression	Full and free in all directions

Table 2: DILATED FUNDUS EXAMINATION

Parameter	Right Eye	Left Eye
Media	Clear	Clear
Optic Disc	Normal size, hyperemic, distinct margins	
CDR	0.3	0.3
NRR	Healthy	Healthy
Blood Vessels	Normal	Normal
Macula	Foveal reflex dull	Foveal reflex dull
Background	Drusens	Drusens

Table 3: DIPLOPIA CHARTING

II	II	I I
II	II	I I
II	II	I I



Figure 1: Right eye abducens nerve palsy

NEUROIMAGING AND TREATMENT

MRI of the brain showed a **calcified granuloma in the left frontal lobe**—likely a sequela of an old granulomatous infection. Although not directly linked to the abducens palsy, it holds neurological significance and warrants monitoring.

Treatment Initiated:

- Ginkgo biloba plus Piracetam (800 mg BD): Neuroprotective and blood flow-supporting.
- Rosuvastatin 20 mg + Aspirin 75 mg + Clopidogrel 75 mg OD to prevent stroke.
- Betahistine 16 mg BD: For vertigo-related symptoms.
- Pentoxifylline 400 mg OD: To improve microcirculation and reduce blood viscosity.

3. DISCUSSION:

This case presents a middle-aged male with known diabetes mellitus and no significant systemic vascular comorbidities, who developed right sixth nerve palsy presenting as binocular horizontal diplopia. A calcified granuloma in the left

frontal lobe was seen by MRI. Fundus examination revealed early signs of age-related macular degeneration (drusens and dull foveal reflex), not directly related to the cranial nerve dysfunction but warranting regular follow-up. Diplopia charting confirmed malfunction of the right lateral rectus, which is compatible with palsy of the right abducens nerve. Given the patient's diabetic status and spontaneous resolution of symptoms over four months, the likely aetiology is microvascular ischemic mononeuropathy. Patients with diabetes usually recover from microvascular sixth nerve palsies in three to six months. This patient followed the expected course, with complete resolution of diplopia and extraocular movement restriction. However, refractive errors persisted, requiring ongoing correction with eyeglasses.

ACKNOWLEDGEMENT:

The patient consented to the publication of his case for educational purposes.

CONFLICT OF INTEREST:

The study was conducted without any financial or commercial ties that might be seen as a potential conflict of interest, according to the authors.

ETHICS STATEMENT:

The study involving human volunteers did not require an ethical assessment or permission, as per local laws and institutional requirements. To take part in this study, the patients/participants provided written informed consent. The dissemination of any potentially identifying information used in this study was approved by the subject in writing.

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