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ISOLATED, PUPIL-SPARING OCULOMOTOR NERVE PALSY SECONDARY TO MIDBRAIN INFARCTION IN A DIABETIC PATIENT: A CASE REPORT

Neurology

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ABSTRACT

Background: Isolated oculomotor nerve palsy (ONP) is an atypical neurological manifestation in diabetes, typically attributed to microvascular ischemic injury of the peripheral nerve. Pupil-sparing is a classic feature of diabetic ischemic ONP. However, central lesions such as midbrain infarction can mimic this presentation, though such presentations are exceedingly rare. **Case Presentation:** We report the case of a 59-year-old man with a history of insulin-dependent type 2 diabetes mellitus, hypertension, and peripheral vascular disease who presented with acute-onset left eyelid ptosis and diplopia. Examination revealed pupil-sparing, partial left oculomotor nerve palsy with isolated medial rectus weakness. Neuroimaging revealed acute infarct in the left midbrain. The patient was managed with antiplatelet therapy, high-intensity statin, optimized glycemic control, and vascular support. Gradual improvement in diplopia occurred over several weeks, with residual mild ptosis. **Conclusion:** This case highlights that pupil-sparing third nerve palsy in diabetics, though often peripheral in origin, may occasionally result from a central lesion such as a midbrain infarct. Detailed neuroimaging should be pursued in atypical or partial presentations, and aggressive vascular risk factor control is imperative to prevent future ischemic events.

KEYWORDS

Oculomotor Nerve Palsy, Diabetes Mellitus, Pupil Sparing, Midbrain Infarct, Cranial Neuropathy, Ischemic Stroke

INTRODUCTION

Cranial nerve palsies involving the extraocular motor nerves (III, IV, and VI) are uncommon in diabetic patients, with an incidence of approximately 0.32–1%[1]. When they occur, they usually present with acute onset ophthalmoplegia and binocular diplopia, most often unilateral. Among these, the oculomotor (III) and abducens (VI) nerves are most involved. Pupil sparing is a defining feature of ischemic lesions of the oculomotor nerve, distinguishing them from compressive etiologies such as aneurysms or neoplasms[1].

The oculomotor nerve contains two functional fiber groups: centrally located somatic motor fibers, which innervate the extraocular muscles and levator palpebrae superioris, and peripherally located parasympathetic fibers from the Edinger–Westphal nucleus that control pupillary constriction. In microvascular ischemia, the centrally located fibers are primarily affected, sparing the peripheral parasympathetic fibers; hence, the typical pupil-sparing pattern seen in diabetic ischemic third-nerve palsy. In contrast, compressive lesions (e.g., posterior communicating artery aneurysm) usually affect the peripheral fibers early, leading to pupillary dilatation[1].

Most adult-acquired oculomotor nerve palsies are ischemic in nature, secondary to systemic vascular conditions such as diabetes mellitus, hypertension, atherosclerosis, hypercholesterolemia, and cardiac disease, all of which collectively contribute to involvement of the extraocular muscles[2]. However, isolated pupil-sparing third-nerve palsy may occasionally originate in the midbrain, where focal infarction of the oculomotor fascicle can clinically mimic peripheral diabetic neuropathy[3,4]. Recognitionof this central cause is essential, as it implies an underlying cerebrovascular process with implications for secondary stroke prevention.

Case Presentation

A 59-year-old man with a 15-year history of insulin-dependent type 2 diabetes mellitus and essential hypertension presented to the emergency department with sudden-onset, non-progressive, 3-day history of drooping of the left eyelid and binocular diplopia. Diplopia was more pronounced on leftward gaze. There was no diurnal variation, fatigability, headache, vomiting, sensory loss, limb weakness, or altered consciousness. His past medical history was notable for peripheral arterial disease, for which he had undergone plain old balloon angioplasty (POBA) of the right superficial femoral artery and a left below-knee amputation two years earlier. He was on insulin and telmisartan, with suboptimal glycemic control. He was a chronic smoker (25 pack-years) and regular alcohol consumer. There was no history of any prior neurological illness.

Examination

On admission the patient was alert, oriented, and hemodynamically stable (BP 150/80 mm Hg, HR 92/min, SpO_2 99% on room air). Systemic examination was within normal limits. Neurologically, he showed left-sided ptosis and restricted adduction of the left eye, while abduction and vertical gaze were preserved. Pupillary reactions were equal and reactive bilaterally, indicating pupil-sparing partial third-nerve palsy. Fundus examination is normal. No other cranial nerves were involved, and motor, sensory, and cerebellar examinations were normal.

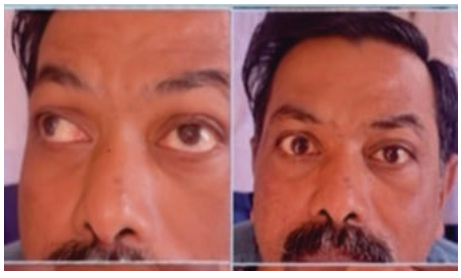

Figure1: Showing Adduction Defect.

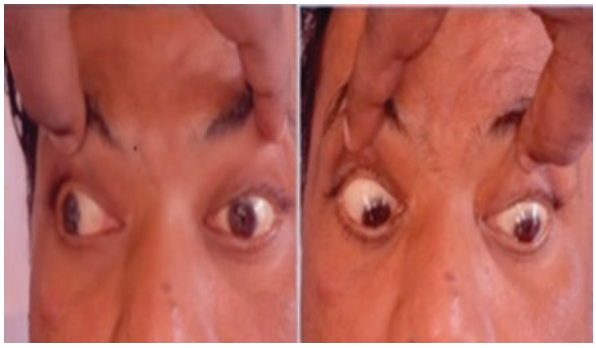

Figure 2: Showing Convergence Defect.

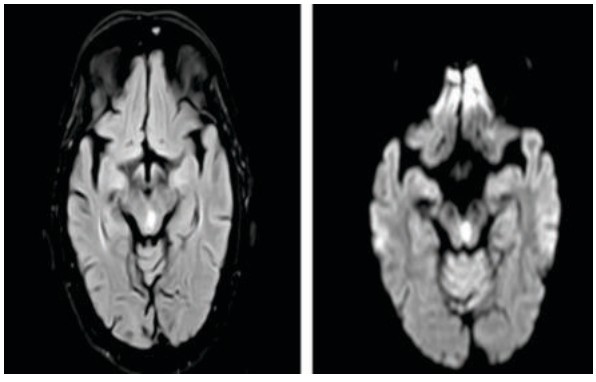

Figure 3: Showing Infarct in the Left Midbrain

Investigations

Brain MRI revealed acute infarct in the left midbrain. Carotid Doppler revealed fibro-calcific plaques causing 60–70% luminal narrowing of the left internal carotid artery and 40–50% narrowing of the left common and external carotid arteries. Echocardiography showed mild concentric left-ventricular hypertrophy, grade 1 diastolic dysfunction, and a preserved ejection fraction of 60%. Taken together, these findings are consistent with atherosclerotic disease. Blood investigations were mostly unremarkable except for an elevated random blood glucose of 198 mg/dL, Hba1c of 9.6%, elevated triglycerides of 230mg/dl

Management

The patient was started on dual antiplatelet therapy (aspirin 150 mg + clopidogrel 75 mg daily), a high-intensity statin (atorvastatin 40 mg daily), and strict glycemic control using short-acting insulin (Actrapid every 8 hours) with basal insulin (Basalog at bedtime). Cilostazol 100 mg twice daily was added for peripheral vascular support, and telmisartan 40 mg was continued. There was no further neurological deterioration.

DISCUSSION

Isolated oculomotor nerve palsy (ONP) is an uncommon but clinically significant manifestation, often associated with systemic vascular diseases such as diabetes mellitus and hypertension[2]. Although most cases in such patients are due to microvascular ischemia, other causes—including compressive, inflammatory, infectious, and intrinsic midbrain lesions—should also be considered. In individuals with vascular risk factors, ischemic mononeuropathy remains the leading cause. However, central fascicular infarction of the oculomotor nerve may, in rare cases, present with features resembling diabetic third-nerve palsy[3,4].

Anatomically, the oculomotor fascicles course through the ventral midbrain before emerging to form the main nerve trunk. Small-vessel infarction in this region can selectively damage motor fibers supplying individual extraocular muscles, resulting in partial oculomotor nerve palsy. The preservation of pupillary function in such cases is explained by the nerve's structural organization—parasympathetic fibers lie peripherally, whereas motor fibers are located centrally. Consequently, ischemic injury limited to the central fascicular region typically spares the pupillary fibers[4]. In our patient, the combination of acute-onset left eyelid ptosis, restricted adduction, preserved pupillary reactions, and absence of other cranial or long-tract deficits suggested a partial, pupil-sparing oculomotor palsy. MRI demonstrated a infarct in the left midbrain, confirming fascicular localization. His long-standing diabetes, hypertension, and peripheral vascular disease support a microangiopathic vascular etiology leading to focal ischemia.

This presentation is rarely reported in the literature, with only a few documented cases describing isolated, pupil-sparing oculomotor nerve palsy secondary to midbrain infarction[3, 4], underscoring the importance of neuroimaging even in cases that appear typical of diabetic cranial mononeuropathy.

The oculomotor nerve receives its vascular supply primarily from the vasa nervorum, small perforating branches of the posterior cerebral and superior cerebellar arteries, which nourish the fascicular portion of the nerve within the midbrain[5]. In diabetes, chronic hyperglycemia induces characteristic microangiopathic changes—hyaline arteriolosclerosis, endothelial proliferation, and thickening of the capillary basement membrane—that compromise the patency of these small vessels[5]. This results in ischemic injury to the centrally located motor fibers while sparing the peripherally situated parasympathetic fibers, explaining the pupil-sparing pattern observed in this case.

Clinical recovery in ischemic cranial mononeuropathies usually occurs spontaneously as nerve fiber conduction and axoplasmic transport are restored. Most patients show improvement within 4–6 weeks, with near-complete recovery by 3 months and complete resolution by 6–12 months[5, 6]. In the present case, the patient showed progressive improvement in diplopia and ptosis over the weeks following treatment initiation. This recovery coincided with stabilization of glycemic levels and optimization of vascular parameters, consistent with the expected course of ischemic cranial mononeuropathy.

This case underscores the importance of maintaining a high index of suspicion for central (fascicular) lesions in diabetic patients presenting with isolated, pupil-sparing oculomotor nerve palsy. Early neuroimaging is essential for accurate localization, as midbrain infarcts can closely mimic peripheral ischemic third-nerve palsy. Moreover, the occurrence of cranial mononeuropathy in a diabetic patient should prompt a thorough evaluation and control of vascular risk factors to prevent future cerebrovascular events.

CONCLUSION

Isolated, pupil-sparing oculomotor nerve palsy in diabetic patients should not automatically be associated with peripheral microvascular ischemia. Central lesions, particularly midbrain fascicular infarctions, though rare,can present with asignificant clinical picture and warrant MRI evaluation. Early recognition and accurate localization through neuroimaging, together with comprehensive vascular risk management, are essential for enhancing recovery and preventing recurrence. This case underscores the importance of considering midbrain infarction as a rare but significant cause of pupil sparing midbrain infarcts.

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