

CASE REPORT

Isolated oculomotor nerve palsy caused by midbrain infarct: A case report

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Abstract

A 70-year-old man with hypertension and diabetes mellitus was admitted to the hospital two days after the onset of acute diplopia. His neurological examination revealed a disconjugate gaze at rest and limited inward gaze in the left eye, as well as an impaired convergence test. There was no ptosis of the upper eyelid, and the rest of the eye movements were normal. The pupils on both sides were equal in size and round, with normal light reflexes. The rest of the neurological examination was normal. High-resolution magnetic resonance imaging showed restricted diffusion in the periaqueductal gray matter of the lower midbrain. Acute stroke patients with visual symptoms may be misdiagnosed and do not receive the appropriate treatment. This is particularly important because potential acute stroke treatments such as intravenous thrombolysis or thrombectomy should be timely administered. We reported an isolated oculomotor nerve palsy caused by central cerebral infarction, which is seldom reported previously.

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1. Introduction

The etiology of oculomotor nerve palsy is complex, involving multiple diseases and pathological mechanisms. Among cranial nerve palsy caused by intracranial aneurysm compression, oculomotor nerve palsy accounts for about 58%.¹ The incidence of oculomotor nerve palsy among diabetic patients is 5 – 10 times that of non-diabetic patients. Moreover, among people over 45 years old, diabetes accounts for approximately 25 – 30% of acute extraocular muscle palsy cases.² Syphilis is one of the causes of isolated oculomotor nerve palsy, accounting for 9.2% of the cases.³ Isolated third cranial nerve stroke syndrome has a relatively low incidence rate in clinical practice. Moreover, there are few cases where high-resolution magnetic resonance imaging (MRI) is detected, and infarction foci are found; prompt recognition is crucial in cases such as this, as patients with acute stroke who present visual complaints may be misdiagnosed and not receive the appropriate treatment. Inappropriate treatment caused by misdiagnosis and missed diagnosis may predispose patients to increased recurrence of stroke. This is particularly important because potential treatments for acute stroke, such as intravenous thrombolysis and thrombectomy, are strictly time-dependent.

2. Case presentation

A 70-year-old man with a history of hypertension and diabetes was admitted to the hospital with a 2-day episode of acute diplopia, which was worse in his peripheral vision but improved when either eye was closed. The distance between the real and virtual images was maximized when the eyes were directed to the right. At initial onset, he had dizziness and nausea that lasted for approximately 6–8 h and then subsided. He was able to live independently without accompanying symptoms, such as headache, regional weakness, or numbness of the limbs, before the onset of the illness. Physical examination revealed his blood pressure to be 167/98 mmHg. Neurological examination revealed a disconjugate gaze at rest, limited adduction at the left eye (Figure 1), and no ptosis; the rest of his eye movements were normal, and his pupils were equal in size and reacted normally to light. The remainder of the neurological examination was normal. He received a score of 1 for partial gaze palsy on the National Institutes of Health Stroke Scale. His laboratory tests showed a hemoglobin A1c of 6.3% (0–6% is within the normal range). Within 24 h of onset, the initial non-contrast computed tomography (CT) scan did not show acute intracranial hemorrhage or ischemia. On the 7th day of onset, a high-resolution MRI (3.0 Tesla, 5-mm slice thickness) showed restricted diffusion in the periventricular gray matter area on the left side. For treatment, the patient started taking 75 mg clopidogrel (for 3 weeks) and 20 mg atorvastatin to control hypertension and diabetes from the 1st day of admission while receiving counseling and education on lifestyle changes. The entire hospitalization process lasted for 11 days. 70 days after discharge, the patient returned to the outpatient clinic for a

follow-up visit. At this time, the patient had fully recovered from diplopia, regaining normal left eye movement, and there was no restricted adduction.

3. Discussion

Isolated medial rectus palsy resulting from a midbrain infarction is exceedingly rare.⁴ In this case, restricted adduction of the left eye suggested the involvement of the medial rectus muscle of the left eye. This could result from lesions of the orbit, eye muscles, oculomotor nerve, oculomotor nucleus, or medial longitudinal bundle. The oculomotor nerve is responsible for innervating the eye muscles, including both motor fibers and parasympathetic fibers. Motor fibers originate from oculomotor nuclei within the superior colliculus of the midbrain. These nuclei can be divided into three types. The lateral nucleus, found on each side, is the motor nucleus and is situated in the ventral gray matter around the conduit at the level of the superior colliculus. It sends out motor fibers of the oculomotor nerve toward the ventral side, passing through the red nucleus. These fibers constitute the oculomotor nerve, which exits the brain from the middle cerebral angle of the fossa. The nerve traverses the space between the posterior cerebral artery and the superior cerebellar artery, proceeds anteriorly to the lateral wall of the cavernous sinus, and subsequently enters the orbit. Within the orbit, the levator palpebrae superioris, superior rectus, medial rectus, inferior oblique and inferior rectus muscles are innervated by the oculomotor nerve. The Perlia's nuclei, situated bilaterally in the midline between the Edinger–Westphal nuclei, exhibit an absence of pairing and project parasympathetic fibers originating from the oculomotor nerve toward the medial



Figure 1. The neurological examination revealed limited inward gaze in the left eye and no ptosis, and the rest of his eye movements were normal

rectus muscles of the eyes, governing the convergence movements of the eyes. Conversely, the Edinger–Westphal nuclei, positioned dorsally to the Perlia's nuclei within the gray matter surrounding the midbrain ducts, project parasympathetic fibers from the oculomotor nerve, which are accountable for the convergence movements of the eyes. Damage to the oculomotor nuclei frequently results in partial or dissociated oculomotor nerve palsy, with the spatial positioning of the subnucleus playing a significant role in this occurrence. The etiologies of medial rectus muscle palsies encompass a range of conditions. Medial rectus muscle palsies induced by intraorbital lesions (*e.g.*, intraorbital tumors, inflammatory pseudotumors, or sinusitis) may manifest as external ophthalmoplegia with conjunctival hyperemia, exophthalmos, and eyelid edema. Orbital MRI, CT, or ultrasonography can aid in diagnosis (not seen in our case).

Ophthalmopathy (*e.g.*, thyroid-associated ophthalmopathy, ocular muscular dystrophy, chronic progressive external ophthalmoplegia) is a painless syndrome involving the extraocular muscles with preserved pupillary function (unlikely due to normal thyroid hormone levels and acute onset). Disorders of the neuromuscular junction (such as myasthenia gravis or botulism) were also unlikely as there were no symptoms that fluctuated daily or morbid fatigue. Oculomotor nerve palsy can be caused by various conditions, including diabetic neuropathy, aneurysmal pathologies (such as those involving the posterior communicating artery, basilar artery, and internal carotid cavernous segment), brainstem infarction, ischemic microvascular infarction, cavernous sinus arteriovenous fistula, tumors (such as pituitary tumors, parasellar meningiomas, nasopharyngeal carcinomas, and lung cancer brain metastases), inflammatory conditions (such as tuberculous meningitis and cavernous sinusitis), Guillain-Barré syndrome, and painful ophthalmoplegia. The blood supply of the midbrain comes from the multi-branch anastomosis of the posterior cerebral artery, basilar artery, and superior cerebellar artery. This multi-source blood supply feature makes its collateral circulation relatively rich. Therefore, compared with other regions of the brain stem, such as pons and medulla oblongatus, which mainly rely on a single vessel for blood supply, the midbrain has a relatively low risk of ischemic stroke. However, midbrain stroke often leads to ischemic responses in adjacent structures, such as dizziness in this patient.⁵ The blood supply in the midbrain is divided into four areas: anteromedial, anterolateral, lateral, and dorsal. The anteromedial area supplies the oculomotor nucleus located near the midline, primarily through small branches of the distal basilar artery. The involvement of paramedian structures often

results in nuclear damage, and is characterized by limited adduction of eyeball, ptosis, and vertical gaze paralysis. However, pupil movement remains normal.^{6,7} Involvement of the anterolateral area is often accompanied by fascicular damage and oculomotor nerve palsy (the pupil can be affected or normal) or contralateral hemiplegia and contralateral ataxia.⁸ The patient in this particular case showed four consistent signs of involvement of the paramedian structures, which are small branches of the distal basilar artery (Figure 2A and 2B). It is worth noting that the patient also had basilar artery atherosclerosis and basilar artery dolichoectasia (Figure 2C and 2D), both of which have been associated with brainstem stroke.⁹

The patient was discharged home on aspirin at a daily dose of 100 mg, clopidogrel at 75 mg daily (for a duration of 3 weeks), and atorvastatin at 20 mg daily. The comprehensive management of hypertension and diabetes includes not only medical interventions but also counseling and education focused on lifestyle modifications. He recovered completely 2 months after discharge. A study conducted in the Taiwan region found that patients with isolated 3rd, 4th, or 6th nerve palsies have a higher risk of developing ischemic stroke (the patient has received outpatient or inpatient treatment due to cerebrovascular diseases, and the disease code in Taiwan is ICD-9 codes 433.x, 434.x or 436).¹⁰ This study included 657 patients

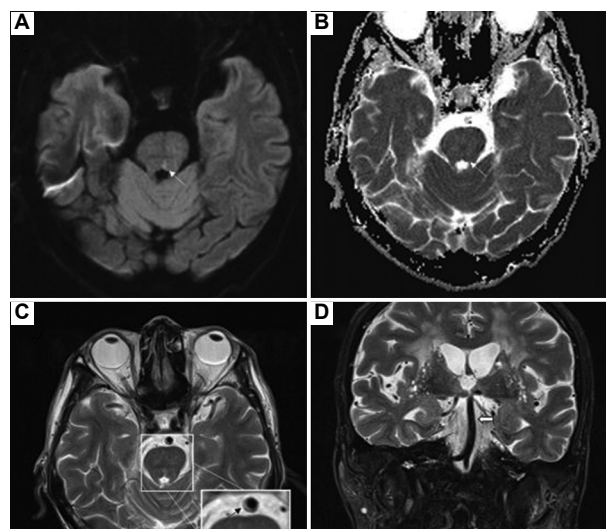


Figure 2. Axial diffusion-weighted imaging (A) Axial diffusion-weighted imaging of the patient shows acute cerebral infarction in the high-signal part indicated by the arrow. (B) The part indicated by the white arrow in the axial apparent diffusion coefficient sequence of the patient shows acute infarction. (C) The axial high-resolution magnetic resonance T2-weighted imaging (T2WI) sequence of the patient showed paracrine infarction (white arrow) and basilar atherosclerotic plaque (black arrow). (D) Coronal T2WI shows a prolonged dilatation of the basilar artery with the top of the basilar artery extending to the bottom of the third ventricle (white blank arrow).

with isolated 3rd/4th/6th cranial nerve palsy, among whom 61.1% were male, with an average age of 54.8 years. Compared with the control group, patients with isolated 3rd/4th/6th cranial nerve palsy had a significantly increased risk of ischemic stroke. The adjusted hazard ratios were as follows: 3.69 (95% confidence interval [CI] 2.20 – 6.19) for the 3rd cranial nerve, 2.71 (95% CI 1.11 – 6.64) for the 4th cranial nerve, and 2.15 (95% CI 1.31 – 3.52) for the 6th cranial nerve.

In this context, timely recognition of symptoms is critical because acute stroke patients with visual problems are at high risk of being misdiagnosed and not receiving appropriate treatment. This is particularly important because potential acute stroke treatments such as intravenous thrombolysis or thrombectomy are strictly time-sensitive. Forty percent of FAST-negative stroke patients may also experience visual symptoms, but these include vision loss, diplopia, and blurred vision. Approximately 33% of the patients will experience gait imbalance.¹¹ With the application of the FAST-modified scale, the recognition rate of stroke has increased from 59% to 67%, and the recognition rate of mild stroke has increased from 30% to 42.2%.¹² Therefore, there is still a gap in visual symptoms and detection rate between patients with FAST negative and those with isolated medial rectus muscle palsy.

The proportion of stroke patients with isolated oculomotor nerve palsy is extremely small. For example, among 2,447 patients, the prevalence of isolated bundle stroke is only 0.25%.⁴ In enhanced MRI, isolated oculomotor nerve palsy caused by cerebral ischemia may present as contrast enhancement of the oculomotor nerve.¹³ For example, a coronal MRI scan of the orbit of a patient infected with the influenza A virus revealed that the right oculomotor nerve was significantly enlarged and enhanced in fat-inhibited T2-weighted images and fat-inhibited gadolinium-enhanced T1-weighted images.¹⁴ For stroke patients, it can also be manifested under MRI as limited diffusion of gray matter around the aqueduct in the midbrain.⁴ This is consistent with the imaging manifestations of this case.

In this specific case, the patient exhibited isolated medial rectus palsy without other neurological signs. However, it is important not to dismiss this case as an ischemic mononeuropathy solely according to the patient's history of diabetes. A comprehensive neurologic examination, including an assessment of adjacent signs (e.g., ataxia or hemiplegia), provides an important basis for determining the need for further advanced imaging. The use of advanced neurovascular imaging should be evaluated with more caution in patients with multiple

vascular risk factors and other cranial nerves or long bundle signs. The National Institute for Health and Care Excellence guidelines in the UK recommend that all stroke patients admitted to the hospital undergo an ophthalmic examination before discharge. For example, within 24 h after admission, an assessment of visual acuity, visual field, and pupil response should be conducted, and emergency problems such as papillary edema should be ruled out. Before discharge, detailed assessments for the visual field, eye movement, visual perception, *etc.*, should be conducted to guide the rehabilitation plan. After discharge, the visual acuity function was reexamined to evaluate the efficacy of rehabilitation, which is critical for adjusting intervention measures and preventing secondary stroke.¹⁵ Early identification and treatment are vital for these individuals, as therapies such as intravenous thrombolysis are typically more effective if administered at an early stage.

4. Conclusions

An isolated oculomotor nerve palsy is a rare presentation of stroke. The fact that patients with acute stroke who present visual complaints may be misdiagnosed and not receive the appropriate treatment afterward underscores the necessity of prompt recognition in such cases. A detailed neurological examination with attention to “neighboring” signs and imaging features is essential during the evaluation of individuals presenting with oculomotor nerve palsy.

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Conflict of interest

Zhaoyao Chen is a Youth Editorial Board Member of this journal, but was not in any way involved in the editorial and peer-review process conducted for this paper, directly or indirectly. Separately, other authors declared that they have no known competing financial interests or personal relationships that could have influenced the work reported in this paper.

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Ethics approval and consent to participate

Not applicable.

Consent for publication

Written informed consent was obtained from the patient. The consent was for publication of the patient's medical history, current presentation, radiological source images, and any other of his pertinent details.

Availability of data

All data is available without restriction from the corresponding author on reasonable request.

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