Scientific Research Publishing

Open Journal of Internal Medicine, 2025, 15(4), 347-353
https://www.scirp.org/journal/ojim
ISSN Online: 2162-5980
ISSN Print: 2162-5972

Recurrent, Contralateral Pupil-Sparing Oculomotor (CN III) Mononeuropathy in Poorly Controlled Type 2 Diabetes Mellitus: A Case Report

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How to cite this paper: Kohli, J., Kermanshah, A., Jose, A. and Zainah, H. (2025) Recurrent, Contralateral Pupil-Sparing Oculomotor (CN III) Mononeuropathy in Poorly Controlled Type 2 Diabetes Mellitus: A Case Report. *Open Journal of Internal Medicine*, **15**, 347-353.
https://doi.org/10.4236/ojim.2025.154031

Received: October 15, 2025
Accepted: December 6, 2025
Published: December 9, 2025

Abstract

Diabetic cranial neuropathies are neurological complications that usually involve cranial nerves (CN) III, IV, and VI, causing acute onset ophthalmoplegia. Recurrence is less common, especially when affecting alternating nerves. The disease course is typically benign, with recovery over weeks to months. Recurrent, contralateral, pupil-sparing CN III palsy in a patient with poorly controlled diabetes supports a microvascular ischemic mechanism once compressive lesions are excluded. Microvascular cranial neuropathy occurs when there is blockage of blood flow to the cranial nerves, leading to nerve damage. This case highlights the importance of early neuroimaging, strict metabolic control, and clear documentation to prevent mislabeling as transient ischemic attack (TIA). We present a 53-year-old man with left periorbital headache, ptosis, and diplopia. The examination revealed a left pupil-sparing CN III palsy. He had an identical right-sided episode four years earlier that resolved spontaneously. Both episodes coincided with poor glycemic control. This case adds to the limited literature describing recurrent, alternating, pupil-sparing CN III palsy in diabetic patients.

Keywords

Diabetes Mellitus, Oculomotor Nerve Palsy, Pupil-Sparing, Microvascular Ischemic Neuropathy, Recurrent Cranial Neuropathy, Neuro-Ophthalmology

1. Introduction

Diabetic neuropathies are among the most common chronic complications of diabetes mellitus and encompass a heterogeneous spectrum of clinical syndromes.

They are broadly classified into diffuse neuropathies, including distal symmetric sensorimotor polyneuropathy and autonomic neuropathy, and focal or multifocal neuropathies, which involve individual nerves or nerve groups [1]. Focal cranial neuropathies are less common than diffuse forms, but they are often acute in onset, painful, and typically self-limited. Even though diabetic neuropathy is a common cause, it is important to rule out other non-diabetic etiologies, including neoplasms, giant cell arteritis, brainstem infarctions, infections, and inflammatory conditions.

The pathophysiology of diabetic neuropathy is multifactorial, involving hyperglycemia-induced oxidative stress, advanced glycation end-products, and activation of polyol and protein kinase C pathways. These lead to endothelial dysfunction, reduced neurovascular blood flow, and ischemic injury. In cranial nerves, focal ischemia of the vasa nervorum (small nutrient vessels which supply the innermost parts of the nerve) causes segmental demyelination and axonal degeneration, while sparing peripheral pupillomotor fibers due to their distinct vascular supply [2]. As glycemic control improves, collateral circulation and remyelination may allow functional recovery, usually over several weeks to months.

Among diabetic cranial neuropathies, oculomotor (CN III) palsy is the most frequent, classically presenting with ptosis, impaired adduction, and ophthalmoplegia. Pain is common, and pupillary function is usually preserved in microvascular ischemia due to the peripheral location of pupillomotor fibers [3]-[5]. This pupil-sparing pattern is a key clinical clue distinguishing microvascular ischemic palsy from compressive causes such as aneurysm. Nevertheless, early neuroimaging with MRI/MRA or CTA should be strongly considered, as aneurysms and other compressive lesions can rarely present without anisocoria, particularly in patients without vascular risk factors whose deficits progress or do not improve by 6 to 12 weeks of follow-up or in those with signs of aberrant regeneration, and to rule out infarctions or neoplasms [6]-[8].

The natural history of microvascular CN III palsy is generally favorable, with most cases resolving spontaneously within 6 - 12 weeks [9]. However, recurrent cranial neuropathies are uncommon and can cause significant disability. Recurrence may affect the same nerve or alternate cranial nerves, complicating diagnosis and prolonging recovery. Risk factors for recurrence include poorly controlled diabetes, hypertension, hyperlipidemia, and older age, with recovery often delayed in patients with multiple vascular risk factors [9].

We present a case of a middle-aged man with poorly controlled type 2 diabetes who developed a recurrent, contralateral, pupil-sparing CN III palsy. This case highlights the importance of distinguishing microvascular ischemia from compressive lesions, the role of strict metabolic control in prevention, and the clinical relevance of recognizing recurrence patterns to avoid misdiagnosis as stroke or TIA.

2. Case Presentation

The patient is a 53-year-old male with a past medical history of right cranial nerve III palsy in 2021, which was initially misdiagnosed as a transient ischemic attack

and resolved after 10 days with steroid treatment, obesity (BMI 35.6 kg/m^2), hypertension, hyperlipidemia, poorly controlled insulin-dependent type 2 diabetes mellitus (glycated hemoglobin [HbA1c] 14.0% [~130 mmol/mol] at diagnosis in 2019, later 9.1% [~76 mmol/mol] in 2021), and Bell's palsy. He presented to the emergency department with blurred vision in the left eye and a headache localized behind the left eye, both of which began abruptly three days prior to presentation. There was no associated weakness, numbness, slurred speech, gait instability, photophobia, or neck stiffness. There was no reported recent illness. The headache worsened with right-sided gaze.

On physical examination, the patient was afebrile, hypertensive at 169/82 mmHg, with a heart rate of 67 beats per minute (bpm), and an oxygen saturation of 97% on room air. He was awake, alert, and oriented to person, place, and date. Heart, lung, and abdominal exams were unremarkable. The neurologic exam was remarkable for left eye ptosis (Figure 1) with diplopia. Left eye extraocular movement showed impaired upward gaze, impaired downward gaze, impaired adduction, and intact abduction (Figure 2). Diplopia was present with a rightward gaze. Downward gaze remained restricted both on unassisted and assisted attempts (Figure 3). Pupils were equal, round, and reactive to light. Right eye extraocular movements were intact (Figure 2). No focal sensory or motor deficits or facial droop were noted.

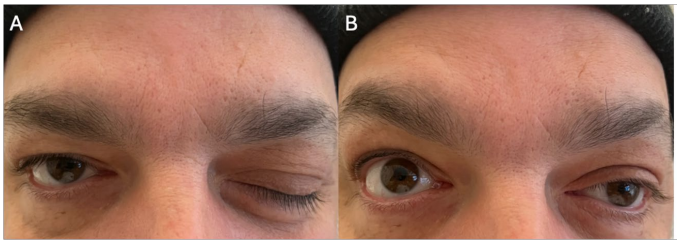

Figure 1. Primary position & ptosis. Primary gaze with left-sided ptosis (A). On attempted upward gaze (B), the patient engages the frontalis muscle and elevates the eyebrow to assist eyelid opening, but incomplete elevation of the left upper eyelid persists, consistent with oculomotor nerve involvement.

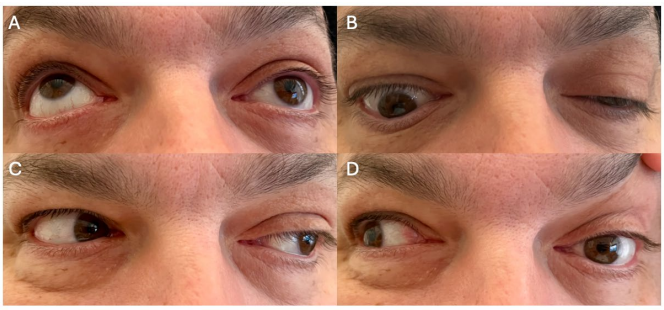

Figure 2. Ocular motility testing: Panel of gaze positions (up, down, left, right). Ocular motility examination. (A) Attempted upgaze demonstrates impaired elevation of the left eye. (B) Attempted downgaze demonstrates restriction. (C) Abduction is preserved. (D) Impaired adduction of the left eye is evident, consistent with a pupil-sparing CN III palsy.

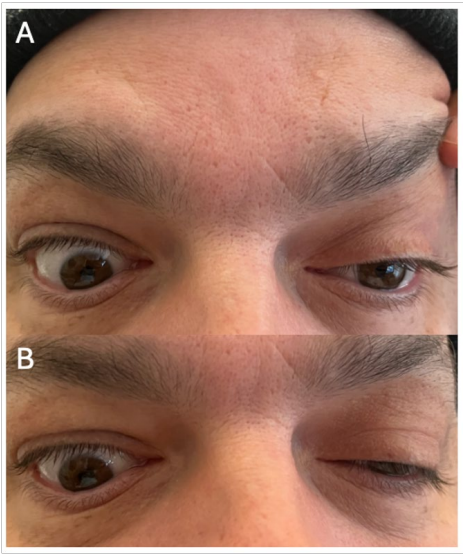

Figure 3. Downward gaze testing. (A) Assisted maneuver: the patient elevates the brow and eyelid, but downgaze remains restricted in the left eye. (B) Unassisted attempt also demonstrates impaired depression of the left eye with eyelid droop, confirming persistent limitation despite preserved pupillary reactivity.

Laboratory testing demonstrated an unremarkable CBC (WBC 6500/uL, HGB 15 g/dL, platelets 184,000/uL). Electrolytes were normal. Blood glucose ranged from 236 - 313 mg/dL, with HbA1c 11.7%. Liver function tests were unremarkable. Lipid panel: LDL 75 mg/dL, HDL 35 mg/dL, TG 80 mg/dL. TSH 2.31 uIU/mL. ESR 8 mm/hr, CRP < 0.3 mg/dL.

Head CT, MRI, and MRA showed no acute intracranial hemorrhage, mass effect, significant midline shift, or recent infarct; angiography of the intracranial circulation was unremarkable. Stable minimal chronic microvascular ischemic changes were noted. Carotid Doppler showed no significant stenosis.

During his hospitalization, the patient was treated for his hypertension and hyperglycemia. He was given analgesics for his headaches. His cranial nerve palsy remained stable and unchanged. He was not given steroids due to a lack of evidence that supports them as an effective treatment.

The patient was discharged on hospital day 4 with scheduled follow-up appointments with Neurology, Neuro-ophthalmology, and Endocrinology. His antihypertensive and diabetic medication regimens were adjusted, with increased doses prescribed at discharge. A telephone follow-up three weeks later revealed that he was still experiencing fasting glucose levels in the 200 - 250 mg/dL range and residual blurry vision with diplopia, though ptosis was improving. The patient had poor follow-up with his specialists and primary care physician. A second telephone follow-up 3 months after discharge revealed he was able to see the neuro-ophthalmologist, and his exam indicated complete resolution of the cranial nerve III palsy.

Written informed consent for publication of this case and images was obtained from the patient.

3. Discussion

Pattern recognition is central to diagnosing cranial nerve palsies. In adults with diabetes and hypertension, the combination of acute painful ptosis, ophthalmoplegia, and preserved pupillary function is highly suggestive of a microvascular ischemic third nerve palsy. Although pain is common in ischemic presentations, it is not specific, as compressive lesions can also present with orbital or retro-orbital pain. Therefore, neuroimaging remains essential at least once in every new presentation, particularly if it is the first episode or if features are atypical. In this case, MRI and MRA were negative, and normal inflammatory markers further supported a microvascular etiology [7] [8] [10].

The natural history of microvascular CN III palsy is generally favorable. Most patients improve substantially within 6 - 12 weeks, with near-complete or complete recovery by 3 - 4 months [9]. Less consistently documented is how commonly recurrence occurs. One of the largest available cohort studies and case series by Trigler (2003) reported that 3.9% of patients had consecutive palsies. And even less discussed is the incidence of recurrence on the contralateral side. Nonetheless, recurrence may occur especially in the presence of ongoing vascular risk factors [11].

Our patient exemplifies this pattern, with alternating CN III palsy episodes separated by four years, both coinciding with poor glycemic control.

Management focuses on optimizing modifiable vascular risk factors such as glycemic control, blood pressure, lipid profile, and smoking cessation, while providing symptomatic relief for diplopia with occlusion or prisms, and ensuring corneal protection if exposure occurs [12]. Importantly, there is no evidence that corticosteroids alter outcomes in presumed microvascular oculomotor palsy. Specialty reviews and American Academy of Neurology guidance emphasize that recovery is spontaneous, and perceived improvement after steroids often reflects the natural course rather than a therapeutic effect [13].

Avoiding misdiagnosis is critical. The patient's initial episode was labeled as a transient ischemic attack (TIA), yet the presence of ptosis, impaired adduction, and preserved pupillary responses should have pointed toward a cranial neuropathy. Recognizing such characteristic patterns can prevent unnecessary stroke evaluations and guide appropriate imaging and follow-up.

This case aligns with prior reports of recurrent diabetic cranial neuropathies. Dey (2019) described recurrent, alternating cranial neuropathies in a diabetic patient, including pupil-sparing CN III palsy, and emphasized their typically benign and self-limited course [14]. Similarly, Tu (2010) reported a diabetic patient with four recurrent cranial neuropathies, two episodes of facial palsy and two of external ophthalmoplegia within two years [15]. These cases highlight that recurrent neuropathies, sometimes alternating between nerves, may occur in the setting of poorly controlled diabetes and other vascular risk factors.

Together, these reports reinforce the need for targeted neuroimaging even when the clinical picture suggests a microvascular cause, and they underscore the

potential for misattribution of recovery to corticosteroid therapy when improvement is more likely due to the natural history of ischemic cranial neuropathy.

Strengths of this case include the clearly documented pupil-sparing pattern, comprehensive neuroimaging, and correlation with poor glycemic control, which together support a microvascular mechanism. Limitations include the absence of original documentation from the first episode and the lack of standardized neuro-ophthalmologic measurements, which reduce phenotypic precision. Additionally, delayed outpatient follow-up limited ongoing characterization of recovery.

Cranial neuropathies from uncontrolled diabetes are common; however, recurrent contralateral cranial nerve palsy is a rare presentation. The key difference between diabetic cranial neuropathy and other causes is the pupil-sparing pattern; however, it is important to rule out all other etiologies using labs and imaging prior to making the final diagnosis. This case again reinforces the importance of proper management of diabetes to prevent complications. Additional studies should be undertaken to confirm these findings as well as to understand the risk of ipsilateral versus contralateral nerve involvement.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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