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Acute binocular diplopia with left abducens palsy and intermittent ptosis in a young adult: A diagnostic challenge

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Abstract

Introduction: Diplopia is a distressing neuro-ophthalmic symptom with a broad differential diagnosis. We present a case that illustrates the challenge of distinguishing a microvascular cranial nerve palsy from ocular myasthenia gravis.

Case presentation: A 32-year-old male with poorly controlled diabetes presented with acute horizontal binocular diplopia. His double vision worsened with leftward gaze and fatigue, and he also experienced intermittent right eyelid ptosis. Neurologic examination revealed impaired left eye abduction, suggestive of an abducens (VI) nerve palsy, and an ice-pack test produced slight subjective improvement in the ptosis and diplopia. Brain MRI, CT angiography, and lumbar puncture were unrevealing for stroke, multiple sclerosis, aneurysm, or infection. Serologic tests for myasthenia gravis were negative, and a trial of pyridostigmine yielded no improvement. With no evidence of myasthenia or structural lesions, the diplopia was attributed to a microvascular left sixth nerve palsy due to diabetes. The patient was managed with vascular risk factor control and a temporary prism for symptomatic relief.

Discussion: This case demonstrates the importance of thorough evaluation when diplopia presents with atypical features. His vascular risk factors pointed to an ischemic cranial neuropathy, whereas the fluctuating, fatigable diplopia and ptosis suggested myasthenia gravis. A comprehensive workup was required to resolve the diagnostic ambiguity.

Conclusion: Thorough clinical evaluation and targeted investigations are critical when diplopia presents with overlapping features, to ensure accurate diagnosis and appropriate management.

Keywords: Binocular diplopia; Abducens nerve palsy; Ocular myasthenia gravis; Microvascular cranial neuropathy; Diabetic mononeuropathy.

Abbreviations: AChR: Acetylcholine Receptor; CN: Cranial Nerve; CN VI: Cranial Nerve VI (Abducens Nerve); CRP: C-Reactive Protein; CSF: Cerebrospinal Fluid; CT/CTA: Computed Tomography / Computed Tomography Angiography; EMG: Electromyography; ESR: Erythrocyte Sedimentation Rate; GCA: Giant Cell Arteritis; GBS: Guillain–Barré Syndrome; INO: Internuclear Ophthalmoplegia; LRP4: Low-Density Lipoprotein Receptor-Related Protein 4; MG: Myasthenia Gravis; MRI: Magnetic Resonance Imaging; MuSK: Muscle-Specific Kinase; OMG: Ocular Myasthenia Gravis; SFEMG: Single-Fiber Electromyography; SIH: Spontaneous Intracranial Hypotension.

Introduction

Diplopia, the perception of a single object as double, is a distressing neuro-ophthalmic symptom that often leads to significant disability [1]. Even though the symptom is subjective, persistent diplopia can severely impair a patient's quality of life that can lead to eye strain (asthenopia), disorientation, and difficulty with daily activities such as driving or work. Monocular diplopia (persisting in one eye even when the other is covered) usually indicates an ocular problem (e.g. corneal or lens pathology) rather than a neurological issue [1]. Binocular diplopia (resolving when either eye is closed) arises from a misalignment of the visual axes or a failure of fusion at the brain level, reflecting misalignment of the eyes due to a neuromuscular or neurogenic cause [1]. Diplopia may in fact be the first manifestation of serious, life-threatening intracranial disorders, including strokes, aneurysms, tumors, or infections, which necessitate prompt evaluation of the patient [1]. Among ocular motor cranial neuropathies, an isolated abducens nerve (sixth cranial nerve) palsy is reported as the most frequent cause of binocular diplopia in adults [2]. The differential diagnosis of an acquired cranial nerve palsy is broad, ranging from benign ischemic neuropathy to compressive lesions [2]. In addition to ocular motor nerve palsies, disorders of the neuromuscular junction like Myasthenia Gravis (MG) are important considerations in patients with diplopia or ptosis. Notably, nearly half of all MG patients initially present with purely ocular symptoms, a form known as Ocular Myasthenia Gravis (OMG) [3]. Unlike structural nerve palsies, MG-related ophthalmoplegia is characteristically transient and fatigable, with symptoms that can fluctuate from day to day or even hour to hour [3]. While OMG by definition remains confined to the extraocular muscles, it is not benign: about 20-60% of patients with initial OMG will progress to generalized disease affecting bulbar or limb musculature [3]. Through a combination of patient history, targeted bedside tests and focused lab investigations, clinicians can differentiate between a peripheral nerve palsy and a neuromuscular junction disorder guiding appropriate management for the patient's diplopia. We present a case in which a young diabetic male's neurological exam with intermittent gaze-dependent binocular diplopia, fluctuating ptosis, and impaired left eye abduction.

Case Presentation

A 32-year-old male with a medical history of poorly controlled type 2 diabetes mellitus (HbA1c 9.9%), primary hypertension, migraines, and generalized anxiety disorder managed with sertraline presented with acute onset binocular diplopia. Associated symptoms included dizziness, intermittent blurry vision, slurred speech, and subjective visual field deficits, pronounced in the left eye. He denied prior episodes of diplopia, head trauma, or childhood strabismus. There was no recent infectious illness aside from a recent dental procedure, for which he was empirically treated with ampicillin. Family history was notable for cerebral aneurysms. On further history, the diplopia was intermittent, binocular, and described

as horizontal and gaze-dependent, particularly exacerbated when looking to the left and at a distance. The patient reported that symptoms improved with rest and worsened with fatigue, raising concern for a neuromuscular junction disorder.

Neurological examination revealed impaired abduction of the left eye, raising suspicion for an isolated left sixth nerve palsy versus an incomplete presentation of OMG. There was noticeable right eye ptosis and no facial droop. An ice pack test demonstrated minor subjective improvement in both ptosis and diplopia. Ophthalmology was consulted for further evaluation of visual symptoms and performed a fundoscopic examination, which revealed no evidence of papilledema. Lumbar puncture with opening pressure and cerebrospinal fluid analysis for inflammatory and infectious etiologies were within normal limits. The patient was started on a corticosteroid pulse regimen and pyridostigmine for presumptive diagnosis of MG with close neurological monitoring. The patient was continued on aspirin and statin therapy for stroke risk reduction and advised to optimize glycemic control.

Initial evaluation included CT and CTA of the head and neck, which were negative for acute pathology or aneurysm. MRI of the brain without contrast, MRI of the orbits, and cervical spine imaging showed no evidence of stroke, no acute intracranial lesions or evidence of demyelination. Cervical imaging demonstrated moderate spondylosis without cord involvement. A trial of pyridostigmine (Mestinon) of 60 mg of 3 doses for 1 day was initiated without any improvement in diplopia or ptosis. Acetylcholine receptor antibody, anti-MuSK testing and anti-LRP4 antibodies were sent; all returned negative (anti-MuSK <1:10). Lyme serologies returned negative and immunoglobulin A levels were also within normal range. Thyroid function tests and transthoracic echocardiogram with bubble study were normal.

Given the presumptive diagnosis of a microvascular sixth nerve palsy, the patient was discharged with counseling on strict glycemic control. He was referred to neuro-ophthalmology for Fresnel prism fitting to alleviate diplopia during recovery and will be monitored for alignment stability before considering any surgical intervention.

Discussion

Approach and workup of diplopia

The evaluation of diplopia begins by distinguishing monocular from binocular double vision. Monocular diplopia persists in one eye even when the other eye is occluded, and it is almost always due to an ocular problem (optical media or retinal issue) rather than a neurologic cause [4]. Common causes of monocular diplopia include refractive errors (astigmatism), dry eye, cataract or lens abnormalities, and corneal irregularities (e.g. keratoconus) [5]. These cause light diffraction within one eye, producing a “ghost” image that often improves with pinhole viewing [5]. In contrast, metamorphopsia from macular changes (macular edema or epiretinal membrane) frequently presents as monocular diplopia; it is distinct from light-diffraction diplopia by the fact that it remains unchanged with pinhole viewing [6]. Binocular diplopia, on the other hand, disappears when either eye is closed and results from misalignment of the eyes (strabismus) so that images do not fall on corresponding retinal points [4]. A thorough history and neurologic exam are critical: one should document the direction of image separation (horizontal vs vertical

vs oblique/torsional), the gaze dependency of the diplopia (which directions worsen or improves it), if the diplopia is prominent at near or far distances, and any associated signs (ptosis, ophthalmic pain, pupil changes, etc.) [1,7]. Patient clinical history questions should have emphasis on previous episodes of diplopia, presence of a childhood "lazy eye" or strabismus, and any recent or remote history of head trauma [7]. It is essential to ask about associated symptoms such as headache, pain with eye movement, ptosis, dysphagia, dyspnea, or generalized weakness, and in patients over the age of 55, to consider systemic signs of giant cell arteritis including scalp tenderness, jaw or tongue claudication, fever, chills, unexplained weight loss, or muscle aches [7]. On examination, diplopia that remains constant across all directions of gaze (comitant misalignment) generally reflects a longstanding strabismus or, when vertical, a skew deviation; by contrast, gaze-dependent variation in image separation (incomitant diplopia) most often signifies extraocular muscle or cranial nerve dysfunction [7]. Objective data with a comprehensive neuro-ophthalmological examination is essential in patients with diplopia, focusing on extraocular movements (smooth pursuit and saccades), ocular alignment in all gaze positions, and saccadic function to localize pathology and guide management. Examination should note the speed, accuracy, and symmetry of saccades when the patient shifts gaze rapidly between targets, as well as the quality of smooth pursuit when following a moving object [8]. Ocular alignment must be tested in the nine cardinal positions of gaze [8]. The cover-uncover and alternate cover tests help differentiate comitant from incomitant misalignment, crucial for distinguishing congenital phorias from nerve palsies [8]. O'Colmain (2014) demonstrated that effective history-taking and thorough neurological examination accurately identified the cause of diplopia in 70.5% of cases, while only 4.7% harbored underlying pathology requiring urgent management [9]. Our patient's presentation and workup highlight several unique and instructive aspects in the context of diplopia. First, his profile featured a mix of clues that initially pointed in different directions: on one hand, he had significant vascular risk factors (long-standing poorly controlled diabetes and hypertension) that could predispose him to a microvascular cranial nerve palsy, an isolated VI nerve palsy in a diabetic patient is classically due to ischemic mononeuropathy. On the other hand, the nature of his diplopia was not typical for a microvascular VI palsy: he described fluctuating, fatigue-induced double vision with variable right eye ptosis, which is much more suggestive of OMG. A diabetic VI nerve palsy would usually cause constant or intermitted diplopia that is maximal on gaze to the affected side, and it would not cause ptosis (ptosis implies either CN III involvement or a neuromuscular junction defect as in MG) [4]. His diplopia was also intermittent and gaze-dependent rather than fixed pointing away from a static nerve lesion. This case highlights that meticulous history-taking and examination are important to properly describe diplopia in a systematic fashion in order to determine a differential diagnosis even with atypical features.

Given the broad differential of diplopia, the workup often requires neuroimaging and targeted laboratory tests. Brain MRI with orbit views is typically performed to rule out structural lesions in the brainstem or orbit (such as stroke or tumor, demyelinating disease) and can be combined with MR or CT angiography if a vascular cause (like aneurysm) is suspected [2]. Acute diplopia can result from serious intracranial pathology including intracerebral hemorrhage, ischemic stroke, or aneurysmal compression, so urgent imaging is indicated if any red flags (acute neurologic deficits, severe headache, etc.) are present (Table 1) [2]. In this patient's case, for example, CT/CTA was done (given a family history of aneurysms)

to exclude an aneurysm or vascular malformation as the cause of diplopia. MRI was also crucial to look for evidence of demyelination or brainstem lesions (e.g. multiple sclerosis plaques or infarcts) that might cause internuclear ophthalmoplegia or nuclear cranial nerve palsies. In this patient the MRI of the brain with gadolinium was unremarkable, effectively ruling out MS or stroke and the MRI of the orbits to exclude orbital causes like a compressive mass or thyroid eye disease was normal.

Laboratory investigations are guided by clinical suspicion. In a comprehensive study of diplopic patients, a broad lab panel included thyroid function tests (and thyroid antibodies) to assess for thyroid eye disease, fasting blood glucose/HbA1c (since diabetes can cause microvascular cranial neuropathies), and infectious serologies when appropriate (e.g. Lyme disease in endemic areas, since neuroborreliosis can cause cranial nerve palsies) [4]. In our patient, Lyme titers were negative, thyroid functions tests were normal, and the most recent hba1c was 9.9%. When an autoimmune neuromuscular junction disorder is suspected, specific testing for MG is essential. This includes serum assays for acetylcholine receptor (AChR) antibodies, muscle-specific kinase (MuSK) antibodies and antibodies to LRP4 (a less common MG antigen), the combination of these tests identifies the majority of MG cases [3]. Our patient was found to be seronegative for AChR, MuSK, and LRP4 antibodies, essentially ruling out all the common immunological markers of myasthenia. Seronegative MG is a well-documented entity, but it can be diagnostically vexing. Approximately half of patients with purely OMG are seronegative for AChR antibodies, and among those without AChR or MuSK antibodies (so-called double-seronegative MG), some 2–30% may have LRP4 antibodies but many still have no detectable antibodies at all (termed triple-seronegative MG) [10]. The absence of antibodies makes the diagnosis rely on clinical criteria and response to therapy [10]. In his case, the diagnosis was slightly supported by the exam and minor improvement on ice-pack test, but did not have any improvement on Mestinon and steroid treatment. If there is high clinical suspicion of MG despite triple negative antibody testing, consideration for Anti-striated muscle antibodies can be tested [11]. Anti-striated muscle (striational) antibodies, directed against skeletal muscle proteins such as titin, the ryanodine receptor, and Kv1.4, are detected in a subset of MG patients and serve primarily as markers of thymoma and late-onset disease [11]. Cytometric cell-based assays have demonstrated that anti-titin and anti-Kv1.4 antibodies are present in over 90 % of MG patients with thymoma or with concurrent myositis/myocarditis, whereas these antibodies are rare in early-onset or purely ocular MG [11]. Although purely ocular MG less commonly harbors these antibodies, their presence (prevalance of 22.7% in MG) would have reinforced the autoimmune etiology and prompted consideration of mediastinal imaging despite negative standard serologies [12]. Nonetheless, this illustrates an important point emphasized in recent literature: seronegative MG must be identified based on clinical features and electrophysiology, and treatment should not be withheld simply because antibody tests are negative [10]. It's also noteworthy that his acetylcholinesterase inhibitor trial (pyridostigmine) did not yield immediate improvement. In OMG, cholinesterase inhibitors alone may not control diplopia adequately, and corticosteroids are "frequently required" when extraocular muscle weakness (ophthalmoplegia) is present [3]. If high clinical suspicion remains for OMG, Single-Fiber EMG (SFEMG) is indicated despite negative antibody tests. It is particularly useful in patients with isolated ocular symptoms (diplopia, ptosis) where other diagnostics may be normal [3]. SFEMG is also recommended when differentiating neuromuscular junction disorders from cranial

nerve palsies, as it directly measures neuromuscular transmission instability [13]. Orbicularis oculi muscle SFEMG demonstrates a sensitivity of 94% and specificity of 79% in OMG, outperforming the ice-pack and repetitive nerve stimulation tests [14]. In a large prospective series of patients with suspected OMG, SFEMG of the orbicularis oculi achieved a sensitivity of 79% (95% CI 73-85%) and specificity of 80% (95% CI 71-90%) overall, with higher diagnostic yield when ptosis was present [15]. Our patient exhibited ocular MG features (fatigable diplopia and ptosis) but remained seronegative for AChR, MuSK, and LRP4 antibodies and did not respond to a pyridostigmine trial, so single-fiber EMG was deferred. The case underlines the importance of proper history taking and clinical workup especially when symptoms and signs point towards multiple differential diagnoses.

Inpatient and outpatient management of diplopia

Management of diplopia depends entirely on the underlying cause, as there is no single treatment for "double vision" itself, aside from symptomatic measures. Patients who present with diplopia due to an acute neurological insult (such as stroke or hemorrhage in the brainstem) require hospitalization and appropriate acute therapy (e.g. IV thrombolysis or anticoagulation for stroke, neurosurgical evaluation for hemorrhage) [16]. In general, diplopia accompanied by other brainstem signs (e.g. weakness, ataxia, altered consciousness) or by papilledema (raised intracranial pressure) should be managed in hospital until life-threatening processes are excluded or treated (Table 1) [2]. Our patient's workup was largely done inpatient: given the concern for possibly serious causes (like aneurysm or stroke at onset), he was observed closely while undergoing neuroimaging and other tests. Another indication for inpatient management is if diplopia is caused by a condition requiring intensive treatment or monitoring. MG is an important example with bulbar weakness or risk of respiratory failure (myasthenic crisis) must be admitted to an ICU for monitoring, and even purely ocular MG cases may be admitted for initiation of therapy if symptoms are disabling [10]. In this case, although the patient's suspected MG was limited to ocular muscles, he was started on high-dose corticosteroid in the hospital due to the disability caused by the diplopia. Other acute therapies for severe autoimmune causes of diplopia include plasmapheresis or IV immunoglobulin (for example, in Miller–Fisher syndrome or Guillain-Barré if ophthalmoplegia is part of a broader paralytic illness) [17,18]. If an infectious cause is identified (e.g. Lyme meningitis, neurosyphilis), appropriate IV antibiotics would be initiated [19,20]. In summary, inpatient management is indicated for urgent treatment of the underlying cause (be it vascular, infectious, or inflammatory) and for cases where close neurological monitoring is required. Notably, one study of ocular motor palsies emphasized that prompt diagnosis and treatment are critical, as some etiologies (like aneurysm or stroke) carry significant morbidity and mortality if missed [2]. This underscores that diplopia that causes disability is not just an eye problem when due to a neurologic cause, it often demands a multidisciplinary and emergent approach [1,2].

While many diplopia causes are benign or self-limited, certain features (red flags) should prompt urgent evaluation and neuroimaging (Table 1). If binocular diplopia has any accompanying brainstem or cerebellar symptoms (e.g. vertigo, severe headache, ataxia, dysarthria, hemiparesis, sensory loss, etc.), immediately obtain neuroimaging with MRI of the brain with diffusion-weighted imaging to look for ischemic stroke in the brainstem [4]. In an emergency setting, a non-contrast head CT has limited utility

for diplopia evaluation, but CT can rule out hemorrhage if stroke is suspected and facilitate urgent therapy [4]. An acute third nerve palsy with a dilated pupil or pain mandates urgent vascular imaging to exclude a posterior communicating artery aneurysm, and even pupil-sparing palsies in vasculopathic patients deserve close monitoring and MRI or MRA if there is any uncertainty [4]. When multiple ocular motor nerves are affected or diplopia occurs with vision loss or proptosis, an urgent contrast MRI of the brain and orbits is warranted to evaluate for cavernous sinus or orbital apex lesions, with CT/MR venography added if cavernous sinus thrombosis is suspected [4]. In patients over sixty with new diplopia with an isolated ocular motor palsy, jaw claudication, scalp tenderness, temporal headache, systemic symptoms, or raised ESR or CRP should prompt immediate high dose corticosteroids and urgent evaluation to prevent stroke or vision loss [21]. Bilateral abducens palsies or optic disc edema signal increased intracranial pressure and mandate prompt neuroimaging, with lumbar puncture considered if idiopathic intracranial hypertension is suspected [4]. In older patients with vascular risk factors, an isolated third, fourth or sixth nerve palsy usually resolves within six to twelve weeks; if there is no improvement by six to eight weeks, atypical features arise, or the patient is under fifty, neuroimaging should be pursued [4].

Table 1: Red flag features in diplopia and recommended urgent management.

Red flag feature	Clinical signs	Urgent management
Brainstem or cerebellar symptoms	Binocular diplopia accompanied by any of these acute neurologic signs: vertigo, severe headache, ataxia, dysarthria, hemiparesis, sensory loss)	Obtain MRI of the brain with diffusion-weighted imaging to evaluate for brainstem or cerebellar stroke
Acute third nerve palsy with pupil dilation or pain	"Down and out" eye position with ptosis and a dilated pupil, often painful	Perform urgent CT angiography or MR angiography to exclude a posterior communicating artery aneurysm
Multiple ocular motor nerve involvement or diplopia with vision loss or proptosis	Simultaneous III, IV, and VI palsies, or diplopia plus optic disc edema or proptosis	Obtain contrast-enhanced MRI of the brain and orbits; add CT/MR venography if cavernous sinus thrombosis is suspected
New-onset diplopia in order patients with sign of Giant cell arteritis (GCA_	Isolated ocular motor palsy in an older patient (> 60 yrs) with features of Giant cell arteritis: jaw claudication, scalp tenderness, temporal headache, systemic symptoms, or elevated ESR/CRP	Corticosteroids should be initiated promptly if clinical suspicion for GCA is high, even with normal ESR and CRP, and clinical prediction tools may aid decision-making in uncertain cases [21,22].
Bilateral sixth nerve palsies, optic disc edema, tinnitus	Horizontal diplopia on both sides or papilledema on fundoscopic exam	Perform prompt neuroimaging (MRI or CT) to assess for raised intracranial pressure; consider lumbar puncture if idiopathic intracranial hypertension is suspected
No improvement in an isolated III, IV, or VI palsy by 6-8 weeks, atypical features develop, or patient is under 50	Persistent diplopia beyond expected microvascular recovery period, early-onset palsy without vascular risk factors, or new alarming signs	Pursue neuroimaging (MRI/MRA) to exclude compressive, inflammatory, or demyelinating causes

This table summarizes critical red flag features in patients presenting with diplopia that warrant urgent evaluation or intervention. It categorizes each red flag by its clinical scenario and outlines the recommended immediate actions to exclude life- or vision-threatening conditions such as brainstem stroke, aneurysmal compression, cavernous sinus syndromes, giant cell arteritis, and raised intracranial pressure. The guidance is based on current neurology and neuro-ophthalmology literature, emphasizing timely neuroimaging, vascular studies, and empiric therapy to optimize patient outcomes [4].

For less acute causes or after initial treatment, diplopia is often managed outpatient with a combination of addressing the underlying condition and providing symptomatic relief. In many cases of isolated cranial nerve palsy due to presumed microvascular ischemia, no specific acute treatment is required as the condition usually resolves spontaneously within 3-6 months as the nerve recovers [23]. Guidelines

often suggest an observation period of about 6 months for ischemic ocular motor palsies, since roughly 80-85% will fully recover in that time, and controlling vascular risk factors (tightening diabetes and blood pressure control) [2,23]. Independent of etiology, outpatient symptomatic treatment for diplopia is crucial to improve a patient's quality of life. Patching one eye (or using opaque tape on glasses) to eliminate the second image can immediately solve double vision, though at the cost of depth perception [1,23]. Another strategy, which preserves binocular vision to some degree, is the use of prism lenses which is the mainstay for symptomatic relief of diplopia in patients with acute cranial nerve palsies [1,23]. Press-on Fresnel prisms can be applied to the patient's glasses to optically realign the two images, thereby reducing or abolishing the diplopia in primary gaze [1,23]. Both neuro-ophthalmologists and orthoptists are qualified to select appropriate prism strength based on measurement of ocular deviation in primary gaze, ensuring optimal symptom relief [24]. After a microvascular cranial nerve palsy the alignment may change over 3-6 months after onset and during recovery, Fresnel prisms can be trimmed and repositioned easily to match the patient's current deviation [25]. Surgical correction is indicated when significant diplopia remains at six months despite optimal prism correction [24]. Long-term outcomes for surgical correction of chronic sixth nerve palsy are highly favorable, with sustained binocular single vision achieved in up to 85.7% of patients, underscoring the role of surgical intervention in cases with persistent diplopia despite optimal non-surgical management [26]. For MG, long-term outpatient management may include steroids or steroid-sparing agents (azathioprine, mycophenolate, etc.) to keep the disease in remission and pyridostigmine can be continued as needed for symptom control as many ocular MG patients do not get complete relief from pyridostigmine alone [3]. It's worth noting that while immunosuppressive therapy in ocular MG is common, there is still debate about how much it alters the long-term course of disease (some ocular MG patients will generalize despite treatment) [3]. In summary, outpatient management of diplopia revolves around treating the cause and providing optical or surgical solutions for persistent misalignment (prisms, patching, or muscle surgery).

Common differential diagnosis of binocular diplopia

Diplopia has a broad differential diagnosis, and a key initial step is to localize the problem. Monocular diplopia (one eye alone sees double) is usually due to an ophthalmologic issue such as corneal irregularities, cataract, refractive errors, or retinal disease and is not neurological [7]. In contrast, binocular diplopia (double vision only when both eyes are open) implies misalignment of the visual axes and can result from a variety of neurological or neuromuscular conditions (Table 2) [7]. An isolated palsy of an ocular motor cranial nerve will cause diplopia in the direction of action of that nerve's muscle [2]. For example, abducens (VI) nerve palsy causes horizontal diplopia worse on gaze to the side of the lesion (as the lateral rectus muscle is weak) [2]. Such palsies are often due to microvascular ischemia in older patients with diabetes or hypertension, but can also result from compressive lesions (tumors along the nerve course), aneurysms (especially a pupil-involving III nerve palsy from a posterior communicating artery aneurysm), trauma, or inflammation [2]. Across a large case series of diplopia, microvascular ischemia is the single most common cause of isolated ocular motor nerve palsies, especially in patients over age 50 [2]. Notably, epidemiologic studies show that in patients <50 years old with an isolated ocular motor palsy, only ~49% are due to microvascular ischemia, whereas in age ≥50 about 83% are ischemic, suggesting younger patients have

a higher proportion of inflammatory or other causes [2]. On the spectrum of neuromuscular disorders, the top differential would include MG. OMG often presents with asymmetric ptosis and diplopia that worsen with prolonged use of the eyes (e.g. toward end of day or with sustained upgaze) and improve with rest [3]. Our patient's intermittent horizontal diplopia, which worsened with fatigue and improved with rest, and the fact that it was binocular (resolved when either eye was covered), strongly pointed to a neuromuscular junction etiology. Other neuromuscular causes of diplopia to consider include the Miller-Fisher syndrome, a variant of Guillain-Barré syndrome characterized by ophthalmoplegia, ataxia, and areflexia. Miller-Fisher syndrome can cause acute bilateral cranial nerve III, IV, or VI palsies (often with ptosis), but it typically presents with obvious systemic features (unsteady gait, generalized areflexia) and is confirmed by anti-GQ1b antibodies [17]. Extraocular muscle diseases can cause diplopia by mechanically limiting eye movements. The most common example is thyroid eye disease (Graves' ophthalmopathy), in which autoimmune inflammation hypertrophies the extraocular muscles (classically the inferior and medial rectus), causing restrictive strabismus (often vertical diplopia or inability to elevate the eyes) [4]. Orbital myositis, orbital tumors, or trauma resulting in muscle entrapment (e.g. an orbital blowout fracture) are other considerations. These often present with signs such as proptosis, pain, or visible conjunctival inflammation [4]. Limitation of eye movement can also be present in a microvascular cranial nerve palsy, MG, or Miller-Fischer [3,27,28]. This patient had a normal thyroid panel, no orbital pain or proptosis, and his orbital MRI was normal (no enlargement of muscles or masses), making an orbital myopathy unlikely. Diplopia can arise from lesions in the brainstem that disrupt the coordinated movement of both eyes. For instance, an Internuclear Ophthalmoplegia (INO) from a medial longitudinal fasciculus lesion will cause impaired adduction of one eye and nystagmus of the other eye on lateral gaze, resulting in horizontal diplopia [4]. INO in a young patient strongly suggests MS, whereas in older patients' stroke is a more common cause [4]. There was no evidence of an addiction deficit with associated abducting nystagmus to suggest internuclear ophthalmoplegia, and the patient's horizontal diplopia pattern which was worse on leftward gaze was not consistent with INO or a vertical skew deviation, making a brainstem localization unlikely. Lastly Spontaneous Intracranial Hypotension (SIH) classically presents with an orthostatic headache, a headache that worsens on standing and improves on lying flat, and may cause horizontal diplopia due to bilateral abducens (VI) nerve palsies producing horizontal diplopia [29]. On MRI, SIH typically shows diffuse pachymeningeal enhancement, subdural fluid collections, brain "sagging," and pituitary enlargement, and lumbar puncture reveals a low opening pressure (<60 mm H_2O) [30]. In our patient, SIH was effectively excluded by the absence of orthostatic headache, as his headaches did not vary with posture, and by normal findings on contrast-enhanced MRI of the brain and orbits (no meningeal enhancement or brain sagging). Furthermore, his opening CSF pressure was within normal limits and cerebrospinal fluid analysis showed no evidence of a leak or inflammatory changes, ruling out SIH as a cause of his bilateral VI palsy and diplopia [29]. In summary, having excluded neuromuscular junction disorders, inflammatory and orbital causes, brainstem lesions, and spontaneous intracranial hypotension, the clinical picture most closely fits a microvascular ischemic sixth nerve palsy in our patient.

Table 2: Differential Diagnosis of Binocular Diplopia: Clinical Clues and Diagnostic Approach.

Cause	Suggestive clinical findings	Diagnostic approach
Cerebrovascular disease (pons/ midbrain infarction)	Older patients with vascular risk factors such as hypertension or diabetes; may have accompanying brainstem signs (ataxia, dysarthria) [1].	MRI of the brainstem with diffusion-weighted imaging to detect acute ischemia [1].
Compressive lesion (aneurysm)	Acute pain (especially with aneurysm); possible pupil involvement or other cranial nerve deficits [2].	Urgent CT angiography or MR angiography to identify aneurysm [2].
Idiopathic (microvascular) cranial nerve palsy	Isolated diplopia in older adults with diabetes or hypertension; no other neurologic signs [2].	Observation with vascular risk-factor control; MRI or CT angiography if no improvement by six to eight weeks [2].
Graves' ophthalmopathy (infiltrative)	Progressive diplopia, proptosis, lid retraction; restrictive motility, often of the inferior rectus [1].	Thyroid function tests and orbital MRI demonstrating extraocular muscle enlargement [1].
Orbital myositis	Eye pain exacerbated by movement; possible conjunctival injection; acute onset [1].	MRI of the orbits with contrast showing muscle inflammation [1].
Trauma (fracture, hematoma)	History of facial or orbital trauma; early diplopia with restricted gaze; possible globe displacement [2].	Non-contrast CT to assess orbital fractures and hematoma [2].
Tumors (skull base, sinuses, orbit)	Insidious onset; painless proptosis or mass effect; may involve multiple cranial nerves [2].	Contrast-enhanced MRI or CT of the skull base and orbit to detect neoplasm [2].
Botulism	Acute descending paralysis; bulbar weakness; dilated pupils; often preceded by gastrointestinal symptoms [3].	Serum or stool assays for botulinum toxin; clinical correlation and supportive ICU care [3].
Guillain-Barré syndrome (Miller Fisher variant)	Ophthalmoplegia with diplopia, ataxia, and areflexia [3].	Lumbar puncture showing albuminocytologic dissociation and nerve conduction studies. Test anti-GQ1b antibodies [3].
Multiple sclerosis	Intermittent internuclear ophthalmoplegia; other migratory neurologic symptoms (sensory or motor) [2].	MRI of brain and cervical spine demonstrating demyelinating lesions [2].
Myasthenia gravis	Fluctuating diplopia and ptosis worsened by fatigue; improvement with rest or ice-pack test; serology may be negative [10].	Ice-pack test; anti-acetylcholine receptor (blocking, binding, modulating)/anti-MuSK/anit-LRP4 antibodies antibody assays; single-fiber electromyography if seronegative, anti-striational antibodies [10,11].
Spontaneous Intracranial Hypotension	Orthostatic headache, nausea, neck stiffness, horizontal diplopia (commonly bilateral CN VI palsies), photophobia [29].	MRI brain with contrast shows diffuse pachymeningeal enhancement, brain sagging, subdural fluid collections, pituitary enlargement; Lumbar puncture analysis: low opening pressure (<60 mm H_2O) [30].

This table outlines the major neurologic and ophthalmologic causes of binocular diplopia, along with their hallmark clinical features and recommended diagnostic strategies. It is intended to guide clinicians through an evidence-based approach to localizing and distinguishing among cerebrovascular ischemic events, compressive lesions, microvascular cranial nerve palsies, infiltrative and inflammatory orbital disorders, neuromuscular junction diseases, and other etiologies. The table synthesizes key findings with targeted imaging or laboratory studies that facilitate prompt and accurate diagnosis.

Conclusion

This case highlights the inherent ambiguity that can accompany neuro-ophthalmic presentations and underscores the value of a thorough, methodical approach. Our patient's clinical features initially pointed in two directions. The fatigue-related fluctuation of his diplopia and ptosis suggested myasthenia, whereas his vascular risk factors indicated a microvascular cranial neuropathy. This ambiguity demanded careful history-taking, detailed examination, and an appropriately broad diagnostic work-up. By systematically evaluating each symptom and sign, and ruling out serious mimics, we navigated the uncertainty in order to arrive at a working diagnosis of microvascular cranial neuropathy. For clinicians, a diligent and

comprehensive approach to such cases ensures that critical diagnoses are not missed and that patients receive appropriate management.

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