Case Report

Acquired Moebius-like palsy in uncontrolled diabetes: An uncommon manifestation of a common metabolic disease

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ABSTRACT

We report the case of a 58-year-old woman with poorly controlled type 2 diabetes mellitus who presented with acute horizontal binocular diplopia due to isolated right abducens nerve (cranial nerve VI) palsy. Laboratory evaluation revealed severe hyperglycemia with glycated hemoglobin of 15.8%. She improved significantly after structured glycemic optimization. This case highlights a rare but reversible presentation of diabetic cranial mononeuropathy.

Key words: Abducens nerve diseases, Cranial nerve diseases, Diabetic neuropathies, Diplopia, Microvascular complications, Moebius syndrome

Abducens nerve palsy is one of the better-known ocular motor neuropathies associated with diabetes mellitus, although its overall frequency remains low compared with other diabetic complications [1-6]. Estimates suggest that diabetic cranial mononeuropathies occur in roughly 0.5–1% of long-standing diabetes cases, with cranial nerve VI being the most susceptible [1]. This vulnerability is attributed to its extended intracranial pathway, its passage through anatomically constrained regions such as Dorello's canal, and its reliance on microvascular perfusion, which is impaired in chronic hyperglycemia [1,2]. Hyperglycemia triggers endothelial injury, oxidative stress, and alterations in the vasa nervorum, which together predispose the sixth nerve to ischemic dysfunction. While Moebius syndrome is primarily understood as a congenital disorder distinguished by facial weakness and impaired abduction of the eyes [7], several reports highlight that similar clinical patterns may arise in adulthood due to secondary causes including vascular insults, inflammation, tumors, and infections [8-10].

This case is noteworthy because such presentations frequently resemble other serious neurological diseases such as brainstem tumors, aneurysmal compression, cavernous sinus pathology, or demyelinating illness. Recognizing diabetic microvascular mononeuropathy early can avoid unnecessary, invasive diagnostic procedures and underscores the critical role of metabolic control in preventing microvascular complications.

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CASE REPORT

A 58-year-old woman presented with a 2-week history of painless horizontal binocular diplopia and mild ptosis, more prominent when viewing distant objects or gazing to the right (Fig. 1a). She had a 20-year history of type 2 diabetes mellitus and acknowledged long-term irregular medication use. She reported no headache, periocular pain, facial numbness, dysarthria, limb weakness, or preceding infection.

She was hemodynamically stable, with a blood pressure of 130/78 mmHg and a body mass index of 22 kg/m². Ophthalmic examination demonstrated intact pupillary responses and preserved visual acuity. Fundus evaluation showed no evidence of diabetic retinopathy. A clear limitation of right lateral gaze indicated isolated sixth nerve palsy, while the remainder of the cranial nerves was normal. Her neurological examination showed normal tone, power, and reflexes. Vibration perception testing was within normal limits.

Laboratory results revealed a random blood glucose of 550 mg/dL and an glycated hemoglobin of 15.8%. Renal, hepatic, thyroid, and inflammatory parameters were normal. Infectious screening for human immunodeficiency virus, venereal disease research laboratory, and hepatitis B was negative. Magnetic resonance imaging of the brain and orbits showed no mass lesion, inflammatory, or demyelinating lesions (Fig. 2).

The clinical picture, normal neuroimaging, and severe hyperglycemia were consistent with diabetic

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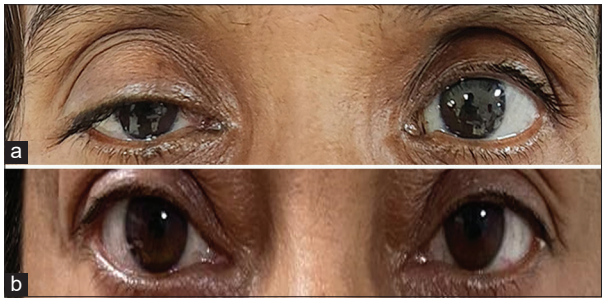

Figure 1: (a) Clinical findings on the day of presentation and (b) resolution of ptosis after 3 months of glycemic optimization in the mentioned patient

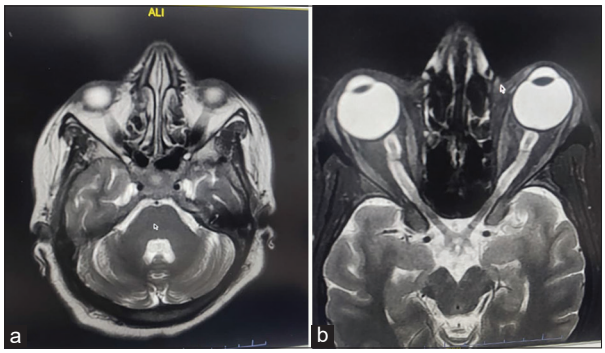

Figure 2: Magnetic resonance imaging brain section showing (a) normal costophrenic angle and no compression of sixth cranial nerve was seen; (b) normal prepontine area and no compression of optic nerve was seen

microvascular sixth nerve palsy with an acquired Moebius-like phenotype. She was started on basal insulin glargine and oral antihyperglycemic agents. Intensive dietary counseling, diabetes education, and self-monitoring of blood glucose were initiated. Diplopia was managed temporarily with alternate eye occlusion.

Over the next 3 months, glycemic control improved markedly, accompanied by gradual and near-complete resolution of her eye movement abnormality (Fig. 1b).

DISCUSSION

Isolated sixth nerve palsy in adults generates a broad differential diagnosis. Diabetes remains one of the most common non-compressive causes due to its propensity to induce microvascular ischemia. The long intracranial course and anatomical configuration of the abducens nerve make it particularly susceptible to metabolic vascular compromise. Chronic hyperglycemia contributes to endothelial dysfunction, impaired autoregulatory capacity, sorbitol accumulation, and basement membrane changes, which collectively culminate in focal ischemic neuropathy.

Several reports have documented similar presentations. Bayrak *et al*. described bilateral sixth nerve palsy in a patient with diabetes, emphasizing the role of microangiopathy [1]. Alroughani and colleagues identified isolated sixth nerve palsy as the initial sign of undiagnosed diabetes [2]. Garay *et al*. and Sarkar *et al*. described cases in which severe hyperglycemia directly produced isolated abducens involvement, with eventual recovery after glycemic stabilization [3,4]. Larger population-based analyses by Patel and Richards indicated that more than half of acute ocular motor nerve palsies in adults stemmed from diabetic or microvascular mechanisms [5,6].

Acquired Moebius-like presentations in adulthood introduce diagnostic complexity. Verzijl *et al*. highlighted ischemic and inflammatory processes as mechanisms capable of producing Moebius-like phenomena [7]. Carrera *et al*. reported cases where adult patients developed combined sixth and seventh nerve involvement resembling Moebius syndrome but recovered once the precipitating cause was corrected [8]. Reports by Dutta *et al*. and Steffen *et al*. further underscore that poorly controlled diabetes can result in simultaneous cranial nerve VI and VII dysfunction that closely imitates Moebius syndrome, again with reversibility following metabolic correction [9,10].

The differential diagnosis of acute sixth nerve palsy includes vascular aneurysms, brainstem gliomas, idiopathic intracranial hypertension, cavernous sinus thrombosis, demyelinating disease, thyroid-related orbitopathy, myasthenia gravis, and various vasculitic neuropathies [11-18]. In this case, normal imaging, preserved pupillary function, absence of systemic symptoms, and severe hyperglycemia strongly supported diabetic microvascular neuropathy as the underlying cause.

Most patients with diabetic sixth nerve palsy experience significant improvement within 6–12 weeks once metabolic control is restored. A similar recovery trajectory was observed in our patient, consistent with the reversible nature of ischemic mononeuropathies associated with diabetes.

CONCLUSION

Acquired Moebius-like palsy represents an unusual but clinically important neurological complication of poorly controlled diabetes mellitus. Because its presentation can closely resemble more ominous intracranial pathology, prompt identification is essential to avoid unnecessary investigations and initiate appropriate metabolic treatment. Strict glycemic control remains the cornerstone of management, with excellent potential for complete reversal of neurological deficits. This case reinforces the need for heightened clinical awareness of diabetic cranial mononeuropathy in patients presenting with acute ocular motor deficits.

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