Down and out: acquired oculomotor nerve palsy

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DESCRIPTION

A 50-year-old male presented to the ophthalmology emergency room with a complaint of sudden drooping of his left eyelid and double vision in the left eye (figure 1). The patient had a history of diabetes mellitus for the past 9 years. The records showed that he was poorly compliant to the prescribed oral hypoglycaemics. The patient did not report any history of recent headache associated with the presentation.

On orbital and adnexal examination, there was exotropia with hypotropia and findings typically suggestive of oculomotor (isolated third cranial nerve) palsy in the left eye. The cover test revealed that the patient could not adduct (figure 1A,D), supraduct (figure 1B) or infraduct (figure 1G,H) his left eye. The patient could perform lateral rotation (figure 1C,F and I) and depression (figure 1H,I), as well as abduct his left eye (figure 1C,F and I), thus confirming functional trochlear and abducens nerve, respectively. The pupil of the left eye had sluggish reaction to direct light and measured 4 mm, compared with 3 mm in the right eye with a normal reaction to light (figure 1E). The patient's visual acuity was 6/9 in both eyes. The remaining cranial nerves were intact, and there was no evidence of sensory or motor weakness. The right eye was pseudophakic, and the left eye had immature senile cataract (Nuclear Sclerotic Cataract-II with Posterior Sub-capsular Cataract). The patient's posterior segment examination was grossly normal, and there were no signs of diabetic retinopathy.

CT and gadolinium-enhanced brain MRI scans with contrast were essentially normal. There was no evidence of intracerebral aneurysm, and this was confirmed by magnetic resonance angiography of the head and neck.

A series of blood tests showed raised blood sugar (between 330 and 350 mg/dL), and glycated haemoglobin A1c to be 12.2%. The patient was diagnosed with third nerve palsy secondary to diabetes caused by pathological microvascular ischaemia. The patient was advised strict glycaemic control. A comprehensive management plan was prepared with the opinion of an endocrinologist. The patient was prescribed basal-bolus insulin therapy that involved administering long-acting, basal insulin twice daily in combination with a rapid-acting insulin analogue injected before every meal.

The patient's hyperglycaemia was controlled to 180–200 mg/dL at his first-month follow-up. The patient made a complete recovery from the palsy. No exotropia or hypotropia was observed, and he could adduct, infraduct or supraduct his left eye well. He did not have residual ptosis of the left eye as well.

Cranial nerve neuropathies are rare long-term complications of type II diabetes mellitus, and the prevalence depends on the severity as well as duration of hyperglycaemia. Different studies

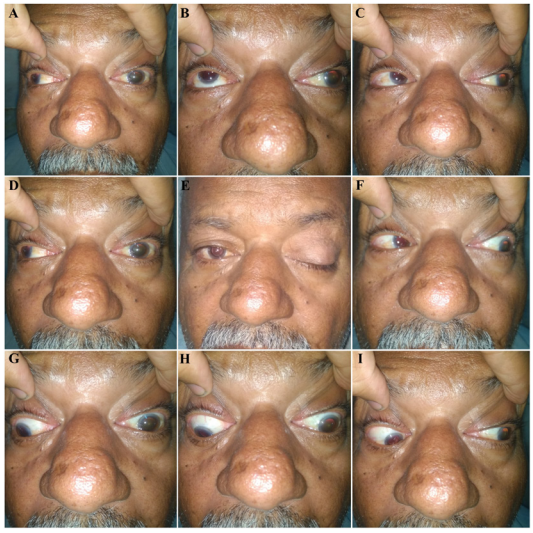

Figure 1 The patient presented to the ophthalmology emergency room with a complaint of inability to adduct (A,D), infraduct (B) or supraduct (G,H) his left eye, thus resulting in diplopia, and sudden onset ptosis (E) of his left eyelid. The montage shows the position of the left eye in comparison to the right eye while examining the different cardinal positions of gaze.

Learning points

- The patients with a long history of uncontrolled diabetes mellitus should be counselled thoroughly and informed about the consequences of persistently high blood sugar, specifically catastrophic outcomes like cranial nerve palsies.
- A high clinical suspicion, detailed history taking and clinical examination should be performed to rule out different causes of cranial nerve neuropathy such as arterial aneurysms and space-occupying lesions.
- The use of neuroimaging, that is, gadolinium-enhanced MRI, is necessary for a conclusive diagnosis, before initiating the management plan for the patient.
- Aggressive control of hyperglycaemia by ensuring compliance through close monitoring of the patient is necessary. In a majority of cases of oculomotor nerve palsy due to diabetes, the symptoms resolve if the patient is compliant and successfully control the blood sugar levels.

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have shown about 0.75%–1% of patients with diabetes mellitus eventually develop cranial nerve neuropathy.[1 2] The commonly encountered cranial mononeuropathies secondary to diabetes involve cranial nerves III (oculomotor), VI (abducens) and IV (trochlear).[3]Among the patients with mononeuropathies, it has been reported that oculomotor nerve palsy secondary to diabetes occurs in 11% patients.[4] Typically, the palsy occurs as a result of microvasculopathy from uncontrolled hyperglycaemia.

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