

Spontaneous Recovery from Recurrent Cranial Nerve Palsies in a Diabetic Patient with History of Ischaemic Stroke: A Rare Clinical Entity

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Abstract: *Diabetes mellitus commonly causes involvement of cranial nerves and third nerve being the most common, however recurrent and multiple cranial nerve involvement is rare. We present here a case of recurrent cranial nerve palsy and spontaneous recovery in a patient with diabetes mellitus.*

Keywords: diabetes mellitus, diabetic neuropathy, recurrent cranial nerve palsy, cranial neuropathy, recovery

1. Introduction

Diabetes Mellitus has been associated with many macro and microvascular complications and neuropathy is one of the most debilitating amongst them. Cranial nerve involvement has been seen very often in these patients and Oculomotor being the most commonly involved. Recurrent cranial nerve palsies have been rarely reported and poses diagnostic dilemma for the physician and hence leading to increased monetary burden over the patient. We present a case with recurrent cranial nerve palsies showing spontaneous recovery in a diabetic patient with history of ischemic stroke.

2. Case Report

A 47 year old Diabetic male presented to OPD with complaints of drooping of left eyelid and double vision. He had left antidiabetic drugs for last three month. He was on Metformin 500 mg twice a day, Glimepride 2 mg twice a day, and a combination of Pregabalin 75mg and Methylcobalamin 750mcg once a day before he left treatment. He had past history of left sided seventh cranial nerve palsy six months back from which he has completely recovered.



Figure 1: Ptosis of the left eye due to Oculomotor palsy

He was diagnosed to be diabetic 6 yrs back when he presented to OPD with complaints of left sided hemiparesis and was found to have multiple lacunar infarcts on MRI scan. On examination, the refraction was normal and visual field was also normal on perimetry. On fundus examination retina showed nonproliferative diabetic changes. Palpebral fissure width and margin reflex distance was decreased in the left eye.



Figure 2: Inability to move eyeball upwards and medially

Left eye was deviated downward and outwards and patient was having difficulty in moving left eye inward and upward. Diplopia decreased when patient was asked to look on his left hand side in downward direction. Pupil on both sides was equal in size and was equally reactive to light. On further examination, there was no chemosis or proptosis of the affected eye, and visual acuity was normal. Cranial nerves II and IV–XII were normal. Neurological examination including power, sensation, reflexes, and cerebellar function were normal. And hence a diagnosis of pupil sparing oculomotor nerve palsy was made. On evaluation his fasting blood sugar was 377 and HbA1c was 9.8%. Complete blood count, renal function test and liver function tests, lipid profile, serum electrolytes were normal. His ANA and viral markers were negative. His blood pressure was 132/80mm Hg in right arm supine position. MRI head with MR Angiogram was done and did not show

any infarct or aneurysm. Patient made complete recovery of the symptoms within 10 weeks of reinstatement of oral antidiabetic treatment.

3. Discussion

Diabetic Neuropathy is one of the most dreaded complications of diabetes; with prevalence of nearly 50% for patients having diabetes for more than 25 years.¹ Most patients usually have distal symmetrical neuropathy, whereas some patients develop focal as well as multifocal neuropathies that also include cranial nerve palsies. Generally, cranial nerve involvement occurs in individuals suffering from diabetes from longer duration², though there have been cases, reported with shorter duration of diabetes having cranial neuropathies³

Cranial nerve palsies are much more common in patients with diabetes than non diabetic subjects. In a study of 5082 patients, the incidence of cranial nerve involvement was tenfold higher in patients with diabetes than in those without diabetes (0.97% versus 0.13%).⁴ Cranial neuropathies in diabetic patients usually involve the third, fourth and sixth cranial nerves. The Oculomotor nerve is involved more than the other counterparts. Greco D et al in a retrospective study of 8150 hospitalized diabetic subjects, found out that isolated third nerve palsies occurred in the majority of patients with cranial nerve involvement, followed by seventh and then sixth nerve.⁵

The common differential diagnosis for third nerve palsy in a diabetic patient includes a large number of pathologies, like structural causes, including inflammation, trauma, infection, cerebrovascular disease and ophthalmoplegic migraine. The most frightening amongst this group is the possibility of an acutely enlarging intracranial aneurysm. Oculomotor palsy in diabetes may or may not be associated with intense retro-orbital. Therefore, presence or absence of pain does not make any significance in differentiating etiologies though aneurysmal rupture leading to subarachnoid hemorrhage may complain of severe pain. However, normal pupillary reaction (pupillary sparing) is usually consistent with ischaemic third nerve lesion, as occurs in diabetes. This is probably due to the fact that fibres supplying the pupil pass superficially in the oculomotor nerve and are spared from the ischaemic effect.^{6,7} whereas aneurysmal compression of superficial pupillary fibres usually leads to a dilated fixed pupil. However, a 'pupillary sparing' sign is not absolute, as intracranial aneurysms can also sometimes present without pupillary involvement and ischaemic third nerve lesions as in diabetes can present with incomplete pupillary signs.⁸ It is therefore important to consider all aspects of history, neurological signs, biochemistry and neuroimaging before coming to a final conclusion.

The finding of simultaneous involvement of multiple cranial nerves or recurrent cranial neuropathies in patients with diabetes is rare although a very few cases have been reported⁹⁻¹¹. Involvement of cranial nerves in these patients recurrently poses difficulty for the doctor and lead to exhaustive workup to search the cause. However there is spontaneous recovery in most of the patients¹². In our patient the occurrence of left facial palsy and an ischaemic stroke 6

years back, followed by right Oculomotor palsy with pupillary sparing, rule out the possibility of more sinister diagnosis and suggest recurrent diabetic cranial neuropathy. Spontaneous recovery on both occasions also supports the ischaemic etiology. Thus, a proper history along with examination and neuroimaging makes it easier to clinch the diagnosis of recurrent cranial neuropathy in patients with diabetes.

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