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# Multiple Cranial Nerve Neuropathy in Uncontrolled Type 2 Diabetes Mellitus

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**Abstract:** ***This Article presents a Case of 62 Years Old Female, a Known Case of Type 2 Diabetes Mellitus who presented with Left Eye Ptosis and range of restricted movement of Left Eye. Patient had remarkable improvement in symptoms after initiation of corticosteroids.***

**Keywords:** Cranial Nerve Palsy, Diabetes Melitus

## 1. Introduction

Neuropathy is an important complication of diabetes with a reported prevalence of around 50% for patients with 25 years of diabetes.1while the vast majority of such patients have distal symmetrical neuropathy, some patients develop focal and multi-focal neuropathies, including cranial nerve palsies. It is important for the clinician to think about a wide variety of differential diagnoses in patients with diabetes presenting with cranial nerve palsy, as there are significant implications in treatment and prognosis depending on the cause we report a case of multiple cranial nerve neuropathy due to uncontrolled diabetes mellitus.

## 2. Case Report

62 Year Old Female Came to OPD With Complaints of Binocular Double Vision, Drooping of Left Eye Lid, Range of Restriction Movements of Left Eye Complaints of Lacrimation of Left Eye for 1 Month. No History of Headche, No History of Vomiting, No History of Fever, No History of Gidiness. She is a known case of Diabestes on Regular Medication for Past 25 Years.

On Examination Patient is concious oriented and a febrile. On systemmic examination patient had complete ptosis, impairment of depression, adduction and elevation of With Presserved Adduction of Left Eye. Pupils Were Bilateral Reactive Equal in Size and Constricted to light visual Acuity of both eyes were 6/6 and visual fields were full. Fundoscopy was Done and it Showed Non-Proliferative Diabetic Retinopathy. No Loss of Sensation over Face. Both Upper Limb And Lower Limb Tone, Power, Reflesx appears to be normal. With Above Clinical Features Suggestive of Third And Fourth Cranial Nerve Palsy of Left Eye. Fasting Blood Sugar Level Was 285mg/Dl Post Prandial Blood Sugar Level Was 345mg/Dl And Hba1c Was 9.86%.

MRI Brain With MRA / MRV Done It Showed Small Vessel Ischaemic Changes And No Other Significant Abnormality. Mri. Brain With Cavernous Sinus And Orbital Cuts Done And It Showed Mild Enlargementin Cvavernous Sinus. Csf Analysis Done And All Were In Normal Limits. Anti Nuclear Antibody Was Done And It Was <1.2 which was within normal limits.

Patient was started on IV Methylprednisolone 500mg for 5 Days Followed by Oral Prednisolone for 40mg and Tappered. Patient was kept on Strict Glycemic Control. There was mild improvement in her ptosis. Follow up after two weeks showed complete resolution of left eye ptosis and ophthalmoplegia

## 3. Discussion

Multiple cranial nerve neuropathies can occur in diabetic population and sometimes it may be a presenting sign of newly diagnosed diabetes or glucose intolerance. From institute of diabetic care and research in Tokyo conducted retrospective study and concluded that incidence of cranial nerve palsy is higher in patient diabetes than non diabetes. (3). The incidence of multiple cranial nerve neuropathy with ophthalmoplegia in diabetes is rare (5, 10). Ross at published three cases with diabetes mellitus had recurrent cranial nerve palsies, with variable combination of the seventh, sixth, fifth, fourth, third cranial nerve (12). Those cases have variable sugar control. One hospital-based case series enrolling 29 patients with diabetic ophthalmoplegia demonstrated only 4 patients had recurrent or alternate cranial nerve involvement on retrospective review (13). In our patients, four recurrent episodes including two peripheral facial palsies, one right oculomotor and another left external ophthalmoplegia occurred with-in two years draw us extra attention. From literature review of diabetic cranial neuropathies, spontaneous recovery is expected within five months in most patients (4, 5). In our patient, the complete recovery of the right oculomotor nerve palsy, followed in two months by a similar external ophthalmoplegia on the opposite side, militates against aneurysm and favors relatively reversible pathology in subarachinoid space.

The pathogenesis of diabetic cranial neuropathy remained obscured due to paucity of pathological reports. Although there‘s no unanimity on the direct relationship of vascular lesions with neuropathy, there had been more attention directed to the probable existence of diabetic angiopathy (14). Dreyfus et al. Postulated ischemic neuropathy as the likely pathological change, although no occluded arterioles or venues and no evidence of hemorrhage or inflammation were found (14). In their pathology report, they remarked

that only one-fourth to one-fifth of the nerve fibers disappeared microscopically, yet almost all of the function of the oculomotor nerve had been affected. Combing with clinical self recovery trait, a functional rather than an irreversible structural change was proposed. Asbury et al. Identified hyalinization of the intra-neural arterioles (30-80 μm) with endothelial proliferative degeneration and resultant stenosis in the lesioned nerves that provided additional support of ischemic pathology (15). Focal non-inflammatory demyelination in the intracavernous portion of third cranial nerve was also observed in autopsy (14). This may provide explanation of pupillary sparing as the oculomotor parasympathetic fibers are small myelinated or unmyelinated.

Variable Cranial Nerve Palsy May Happen Onto Subjects with DM or Glucose Intolerance. Associated Perior-Bital aching Commonly Happens. The ”Pupil Sparing Rule” is not an unfailing sign in a diabetic patient with incomplete third nerve palsy or multiple cranial neuropathies. Exception does occur. Thus, prudent neurological assessment and appropriate imaging study should be performed in these patients. Recurrence of diabetic ophthalmoplegia may occur occasionally, albeit its prognosis is relatively benign.

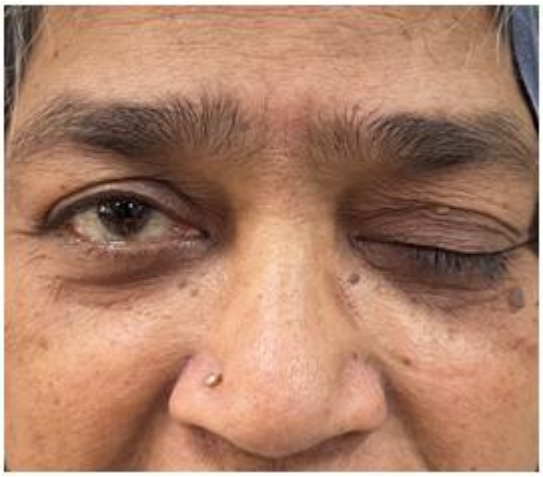

**Figure 1:** At the time of admission

**Figure 2:** At the time of discharge

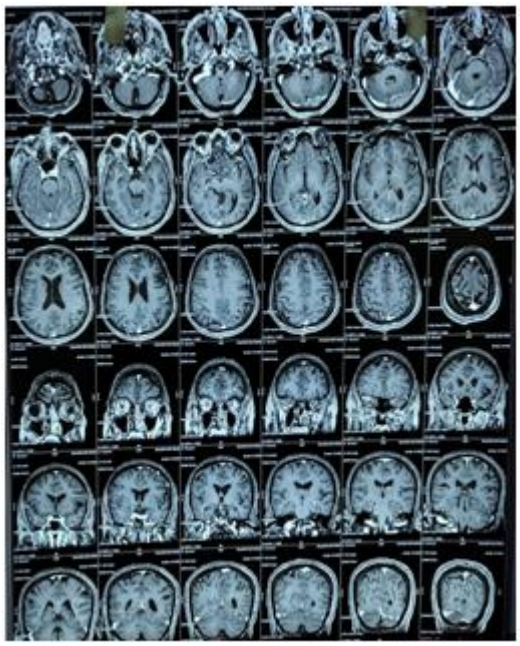

**Figure 3:** MRI BRAIN

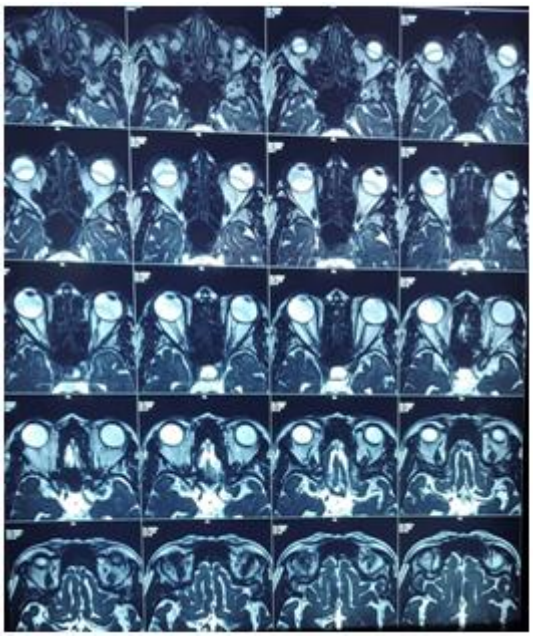

**Figure 4:** MRI brain with orbital cuts

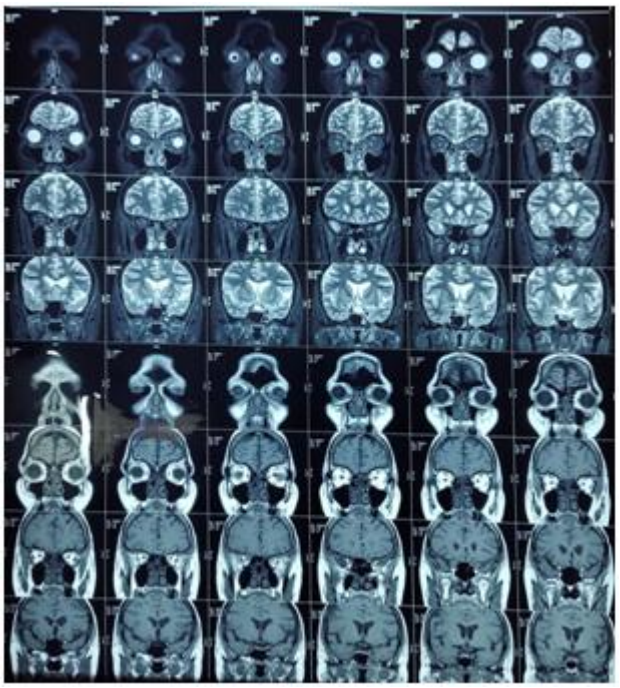

**Figure 5:** MRI brain with cavernous sinus

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