



An Interesting Case Of Diabetic Cranial Mononeuropathy

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Type of Publication: Case Report

Conflicts of Interest: Nil

Abstract

Unilateral oculomotor nerve palsy is a common clinical condition with various etiologies ,including aneurysm ,diabetes mellitus ,CNS infections,Pituitary tumours .Due to the plethora of possible causes early and thorough investigation is essential for treatment .

Here we present a case of a 55 year old male known case of type II diabetes mellitus presented with ptosis,double vision along with pupillary involvement ,who initially thought to have a compressive lesion and after ruling out other causes finally diagnosed as diabetic mononeuropathy .Patient was treated with antiplatelets ,novel oral anticoagulants and oral hypoglycemic agents .Patient had an almost complete recovery in 4 weeks .

Keywords: third nerve palsy ,pupillary involvement,diabetes mellitus,aneurysm

Introduction

A 55 year old male known case of type II Diabetes mellitus came with complaints of inability to open the right eye and on opening the eyelid manually had double vision for the past 10 days .C/o inability to open the right eyelid was acute in onset and showed no diurnal variation.C/o double vision on manually opening the right eyelid and double vision was not present on looking with one eye and aggravated on focusing near objects.No h/o eye pain,fever,headache,vomiting .No h/o trauma .No history suggestive of other cranial nerve involvement .No h/o limb weakness/sensory disturbances .Pateint was vitally stable with Blood pressure of 150 mmHg.On examination of cranial nerves III,IV,VI on the right eye primary gaze was down and out ,and in extraocular movements adduction and elevation were absent .Complete ptosis was present (palpebral fissure not visible) and Pupil size was 4 mm sluggish reacting to light and in left eye pupil size was 3 mm and extraocular movements full in range .All other cranial nerve examination and motor and sensory

system examination was found to be normal .Patient was provisionally diagnosed as isolated right third cranial nerve palsy with pupillary involvement - probably aneurysmal lesion / diabetic cranial mononeuropathy .Routine investigations were done showed Hb -16 g/dl,total counts of 10,500 with 50% neutrophils ,ESR - 15 mm ,serum urea 16 mg\dl ,serum creatinine was 0.9 mg\dl with eGFR of 101 ml/min/1.732 m2 ,RBS -99 mg\dl ,FBS - 130 mg\dl,PPBS - 160 mg\dl ,HbA1c- 7.1%,urine protein negative ,urine glucose - 3 +Total cholesterol -132 mg\dl ,Triglycerides - 118 mg\dl,HDL - 41 mg\dl,LDL - 67 mg\dl [ASCVD : 10.8%],VLDL - 24 mg\dl,Non HDL - 91 mg\dl.MRI brain with MRA was done was found to be normal .Patient was diagnosed as isolated diabetic cranial mononeuropathy .

Treatment given - Patient was started on T. Metformin SR 500 mg BD,T. Vildagliptin 50 mg OD ,T. Telmisartan 40 mg 1-0-0,T. Aspirin 75 mg 0-

1-0,T. Rivaroxaban 2.5 mg BD ,T. Rosuvastatin 40 mg 0-0-1.

Patient was discharged and asked to review after 4 weeks and was found to have an almost complete recovery.

Figure 1.1 MRI brain (T2)

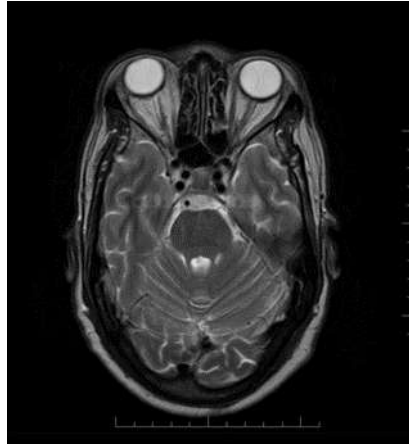


Figure 1.2 (T2 flare)

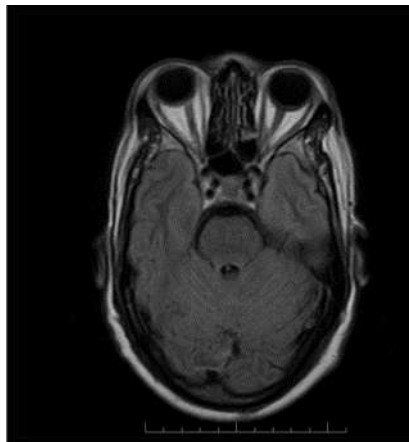


Figure 1.3 MRA



Figure 2.1 :Presentation on the day of admission



Figure 2.2 : After 4 weeks



Discussion:

Diabetic mononeuropathy is one of the rare forms of diabetic microvascular involvement, but oculomotor nerve palsy is the most frequent manifestation of diabetic mononeuropathy. Paresis may go unnoticed if not revealed by a diplopia that is disturbing to the patient therefore a careful examination of ocular motility must be systematically carried out.¹

The pathophysiology of diabetic Oculomotor nerve palsy include alterations in the blood-nerve barrier;hypertrophy of the microvascular basement membrane, and hypoxia in endoneurial space;cranial nerve dysfunction may arise from conduction block or demyelination, as opposed to axonal damage, thus explaining the favorable natural history for recovery from microvascular ischemic insults.

The Etiology and Presentation of acquired third-nerve lesions include

Supranuclear lesions: Lesions at the level of the cerebral cortex or the supranuclear pathway cause conjugate paresis of both the eyes.

Nuclear lesions: Vascular diseases, demyelination, and tumors are the main cause of third-nerve palsy.

Basilar portion: In this region, isolated third-nerve palsy is very common.The primary causes of isolated palsy - aneurysms, diabetes mellitus, and extradural hematoma.The palsy results from either direct

compression of the nerve by an aneurysm or due to subarachnoid hemorrhage in the vicinity of an aneurysm. This causes isolated and painful third-nerve palsy.Extradural hematoma - causes increased tentorial pressure and herniation of the temporal lobe - third nerve gets compressed by the herniation .

Intracavernous portion: Any lesion in the cavernous sinus will result in multiple nerve palsies of the cranial nerve IV, VI, and the first division of cranial nerve V.The common etiology is diabetes, pituitary apoplexy, aneurysm, or carotid-cavernous fistula.

•**Intraorbital portion:** Trauma, tumors, and Tolosa-Hunt syndrome are the main causes of intraorbital third-nerve palsy.

Miscellaneous causes like

Diabetes:Usually painful/painless,with pupil sparing ;recovery around 12 weeks;

Cranial arteritis : >60 yrs,nocturnal headache,scalp tenderness;

Meningovascular syphilis ;

Acute migranous headache:Transient,incomplete 3 rd nerve palsy repeated attacks

Ocular Myasthenia :Usually bilateral involvement 2

The risk factors associated with development of vasculopathic oculomotor nerve palsy include - hypertension was seen most frequently

(42.8%),hypercholesterolemia (40%),smoking (28.57%), coronary artery disease (14.2%),alcoholism (11.3%),obesity.

Majority of the patients with pupillary involvement showed no diabetic retinopathy changes or had less severe grades of diabetic retinopathy.A study by Acaroglu et al., in which presence and level of diabetic retinopathy was found to be significantly lower in diabetics with cranial nerve palsy .3

Pupil involvement in patients with diabetes associated oculomotor nerve palsy occurs in about 1/4th of all cases.Pupillary involvement is common in aneurysmal lesion due to the blood supply to the nerve ,where the vasovarium of the nerve which lies centrally (affected by diabetes) and the pupillary fibres run in the periphery of the nerve is supplied by pial vessels which is easily involved in a compressive lesion.Although pupil may be involved in both - characteristics like an incomplete involvement and anisocoria < 2 mm may help to distinguish diabetic (ischemic) from aneurysmal (compressive) injury).Another differentiating point is that in diabetes as etiology there is a fast recovery between 4 to 12 weeks .Imaging may not be required in pupil-sparing oculomotor nerve palsies in patients over 50 years with known vasculopathic risk factors .Imaging should be considered in those cases of pupil involved oculomotor nerve palsies if patient presents with additional cranial nerve palsy or neurological abnormalities and pupil shows characteristics of a compressive lesion even if history is suggestive of an ischemic lesion.Majority of cases of ischemic oculomotor nerve palsy show spontaneous resolution with medical treatment alone in contrast to nerve palsy due to aneurysmal injury where earliest possible surgical intervention is required .4

Conclusion :Diabetic mononeuropathy is one of the rare forms of diabetic microvascular involvement.

Oculomotor nerve palsy is the most frequent manifestation of diabetic mononeuritis.MRI can eliminate the neurosurgical causes of 3 rd nerve palsy and must be included in the evaluation.Several cardiovascular risk factors associated with diabetes are in favor of ischemic origin of 3rd nerve palsy. The natural history is that it is usually spontaneously resolving, and the management being preventive and consisting of control and cardiovascular risk factors.The occurrence of ocular motor CN palsy is a significant risk factor of subsequent stroke even after adjusting for demographic factors and confounders in the general population. Hence we must educate patients with ocular motor CN palsy regarding the higher risk of future stroke.

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