IOSR Journal of Dental and Medical Sciences (IOSR-JDMS)
e-ISSN: 2279-0853, p-ISSN: 2279-0861.Volume 23, Issue 5 Ser. 7 (May. 2024), PP 20
www.iosrjournals.org

Uncontrolled Diabetes Mellitus Presenting As Isolated Abducens Nerve Palsy

Raju H Badiger, Dhruv Madaan, Arush Vedwa
Kles Dr Prabhakar Hospital And Mrc,Jawahar Lal Nehru Medical College,Belagavi,Karnataka

Date of Submission: 07-05-2024 Date of Acceptance: 17-05-2024

I. Introduction :

Isolated sixth nerve(Abducens Nerve) palsy is a type of mononeuropathy resulting in the weakness of lateral rectus muscle. Neoplasia, vascular disease, and trauma are among the most common causes. Microvascular ischemia related to hypertension and / or diabetes is a commonly identified cause of sixth nerve palsy in older adults.

Diabetes mellitus is a rare but benign cause of Cranial neuropathy. Extra ocular motility disorders may occur in patients with diabetes, secondary to diabetic neuropathy, involving the third, fourth, or sixth cranial nerve.Third cranial nerve is most commonly affected due to diabetes induced micro-vasculopathy. Rarely, sixth cranial nerve palsy can occur. We report a 48-year-old man, a known case of diabetes mellitus for 10 years, who presented with binocular diplopia, secondary to sixth nerve palsy. Neuropathy of the cranial nerves, despite being a rare entity in diabetes mellitus, appears to be a serious problem from a diagnostic and therapeutic point of view. Although the prognosis is excellent, it remains a diagnosis of exclusion and retrospection.

Aim :

To promote the recognition of Diabetes as a cause of Abducens nerve palsy.

II. Case presentation

A 54-year-old Indian male, a known case of diabetes mellitus since 10 years, presented to the emergency department with a 1 week history of sudden onset diplopia and right sided frontal headache.

The headache was constant over the right frontal and retroorbital area, radiating to the nasal side of the right eye, throbbing in nature, not aggravated with light or sound and not associated with any redness or lacrimation. No history of fever, neck stiffness, photophobia, phonophobia, pulsatile tinnitus, seizures, nausea and vomiting. The horizontal diplopia was more prominent upon right lateral gaze and resolved on covering one eye (Binocular diplopia). Left lateral vision was unaffected. No pain on eye movement. Both the headache and horizontal diplopia began simultaneously for the first time.

There was no history of neurologic or cardiac disease, hypertension, cigarette smoking, or alcohol consumption. He had been taking oral hypoglycemic agents for approximately 10 years.

Patient denied any recent history of travel, viral illness and head injury. There was no similar history of headaches or attacks in the family. Review of systemic and rheumatological systems was insignificant.

Physical examination was significant only for right lateral rectus palsy and left sided horizontal nystagmus. Pupils were equal, round, reactive to light and accommodating. No proptosis, ptosis or facial numbness present. No papilledema appreciated on fundoscopy. Visual acuity, other cranial nerves and upper and lower motor neuron examinations were normal.

III. Investigations

On investigation, full blood count displayed raised WBC's (13.7 × 109/L) with a left sided neutrophil shift (80.4%), normal lymphocytic count (13.2%), normal hemoglobin (149 g/L) and normal platelets level (246). No elevated inflammatory markers (CRP 0.2 mg/L; ESR 3). Creatinine kinase levels were 742 U/L (normal

35–232).There was an elevated fasting plasma glucose level of 145 mg/dl (normal, 70-130 mg/dl) and HbA1C of 10.1 (normal<6.5%). Rheumatological screening showed normal ANA, rheumatoid factor and complement levels. Infectious work up including Lyme and syphilis serologies and CSF analysis were not performed, because patient was not sexually active and no other neurological signs were present.A pyridostigmine test for myasthenia gravis was negative. Repetitive nerve stimulation test of the trapezius and orbicularis oculi muscles was normal.Skull x-rays and MRI/Magnetic resonance angiography including the neck and head were normal.

	FBS(mg/dl)	PPBS(mg/dl)
At presentation	145	240
1st week	130	260
2nd week	124	236
3rd week	113	208
4th week	112	198
5th week	101	174
6th week	110	180
7th week	98	168
8th week	98	164

IV. Treatment outcome

Given the normal examinations, we tentatively attributed the ocular motor findings to diabetes mellitus. The patient was prescribed Injection human mixtard, and a controlled diet. An occluder was prescribed for his left eye to prevent diplopia and he is adviced to do extraocular muscle exercises. 8 weeks later, his fasting glucose level is 98 mg/dl the ocular motor findings had disappeared.

V. Discussion

Oculomotor cranial nerve palsies are common in diabetes, but our case represents an uncommon event in

diabetes: isolated uniocular sixth nerve palsy. In 1976, Keane analyzed 125 cases of bilateral sixth cranial nerve palsies; none was classified as diabetic. In 1981, Rush and Younge analyzed 1,000 patients with ocular motor cranial nerve palsies and found bilateral involvement of the sixth cranial nerve in 33. None was caused by vascular disorders. In more than half of the cases, the bilateral sixth cranial nerve involvement was associated with head trauma, pontine neoplasm, or aneurysm of the posterior circulation. In that series, 8 patients had bilateral third cranial nerve palsies, 2 of which remained idiopathic. Thirteen patients had bilateral fourth nerve palsies, none from diabetes. In another large study of patients with ocular motor cranial nerve palsies there were 53 cases of bilateral sixth nerve palsies; none was attributed to diabetes. Nine patients had bilateral third nerve palsies, 3 of undetermined cause and none from diabetes. Of the 21 bilateral fourth nerve palsies, none was attributed to diabetes. Considering both studies together there were 15 (1.5%) cases with multiple cranial nerve palsies that were attributed to a vascular cause, 3 of them (0.3%) with diabetes. Two of these diabetic cases had asymmetric third and fourth cranial nerve palsies; the other was not further described. Sergott et al reported two cases of bilateral third and fourth cranial nerve palsies associated with diabetes. In both cases, the ophthalmoplegia resolved completely within a few months. Jay and Nazarian described a patient with bilateral sixth nerve palsy associated with temporal arteritis and diabetes. The cranial neuropathy was attributed to diabetes.

VI. Conclusion :

Isolated Sixth Nerve palsy is infrequently associated with Diabetes which is sequelae of microvascular disease. Majority of patients with microvascular sixth nerve palsies improve spontaneously after 8 - 10 weeks.Insulin aids in recovery much faster than compared to Oral Antidiabetic agents.