Assessment of Recovery of Cranial Nerve Palsies in Diabetes Mellitus

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ABSTRACT

BACKGROUND

Diabetic cranial neuropathies usually involve cranial nerves III, IV and VI causing acute onset of ophthalmoplegia. These result from diabetes, hypertension, hyperlipidaemia and advanced age. The incidence of cranial nerve palsies in diabetic patients was significantly higher than in non-diabetic patients. We wanted to evaluate the recovery of cranial nerve palsies in relation to the duration and severity of diabetes mellitus and determine the factors associated with diabetes mellitus attributing to the cranial nerve palsies.

METHODS

30 patients with diabetes mellitus were enrolled in the study. Detailed medical history was taken and ocular examination was carried out. Degree of ophthalmoplegia, residual muscle deviation, and ocular movements at each visit were recorded. Blood pressure, RBS, HbA1c and lipid profile were recorded.

RESULTS

Of the 30 patients, males contributed 53.3% and females contributed 46.7%. 67% patients had sixth cranial nerve palsy and 33% patients had third cranial nerve palsy showing that the most common affected nerve is abducens nerve. The mean duration of diabetes mellitus was 6.8 years, mean RBS level was 236 mg/dL and the mean HbA1c level was 8.3 gm%. Majority of the patients had association of hypertension which accounted for 40% of patients.

CONCLUSIONS

The most commonly affected cranial nerve in our study was abducens nerve. The most common association of diabetes mellitus noticed was hypertension. Majority of cases of ischemic ocular motor nerve palsies showed spontaneous recovery by 3 months with medical treatment and with good control of blood glucose level.

KEYWORDS

Cranial Nerve Palsies, Ophthalmoplegia, Glycosylated Haemoglobin, Abducens Nerve, Diabetes Mellitus (DM), Hypertension (HTN), Hypothyroid, Cerebrovascular Accidents (CVA), Dyslipidemia CKD (chronic kidney disease), ER (early recovery), R (recovery), SD (standard deviation)

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BACKGROUND

Ophthalmoplegia is the paralysis or weakness of the eye muscles. It can affect one or more ocular muscles at the same time and the result is either gaze palsy or paralytic strabismus depending on the cause and severity. Isolated cranial mononeuropathies in patients over age fifty most commonly result from vasculopathic risk factors including diabetes, hypertension, hyperlipidaemia and advanced age. Diabetic cranial neuropathies usually involve cranial nerves III, IV and VI causing acute onset of ophthalmoplegia. Sparing of the pupil with paralysis of the third cranial nerve is the characteristic of diabetic ophthalmoplegia.[1]

The incidence of cranial nerve palsies in diabetic patients was higher than that in non-diabetic patients.[2] With the use of imaging, it is now possible to say that isolated oculomotor nerve palsies in a diabetic individual may uncommonly be due to midbrain infarction or haemorrhage. If the third nerve palsy in a diabetic patient fails to recover, it is likely due to a co existent compressive lesion such as a tumour or an aneurysm.[3] Early neuroimaging with MRI may be performed during initial evaluation or some studies have shown observation without neuroimaging unless spontaneous resolution has not occurred by 3 to 6 months.

Hypertension does not increase the risk of ophthalmoplegia, but it worsens the effect of diabetes in ischemic nerve palsies; especially the combination of diabetes and hypertension was associated with increased risk of sixth nerve palsy.[4] The symptoms of the diabetic cranial neuropathies usually occurs suddenly. A proper clinical evaluation is thus important for the diagnosis, treatment strategy and prognosis.

METHODS

This is a hospital based prospective interventional study on 30 patients with diabetes mellitus who visited the outpatient department and squint clinic at Minto Ophthalmic Hospital, Regional Institute of Ophthalmology, Bangalore Medical College and Research Institute conducted between November 2017 to May 2019.

Inclusion Criteria

- Patients willing to give written consent.
- All the patients with Type 1 and Type 2 diabetes mellitus with cranial nerve palsies.
- Associated comorbidities like hypertension and dyslipidaemia.

Exclusion Criteria

- Patients with traumatic nerve palsies.
- Intracranial space occupying lesions causing nerve paralysis.
- Cranial nerve palsies with infective etiology.

After obtaining institutional ethical committee approval, the outpatients in the department of ophthalmology fulfilling the inclusion and exclusion criteria were enrolled in the study. After explaining the need for relevant investigations, and their role in further management, patients were included in the study.

Informed written consent was obtained from patient or a responsible attendant before including the patient in the study. Detailed history was taken from the patients with specific reference to clinical symptoms and all patients were subjected to comprehensive ocular examination. In this study, degree of ophthalmoplegia, residual muscle deviation, ocular movements at each visit was recorded with Hess charting and diplopia charting at squint clinic. Degree of Ophthalmoplegia was recorded by relative limitation of ocular ductions of inferior Oblique, medial, superior, inferior and lateral rectus using 0 to 4 scale. 0 represented full duction, 4 complete absence of duction whereas 1, 2 and 3 indicated 25%, 50% and 75% impairment of duction respectively. Blood pressure measurement, random blood sugar, Glycosylated haemoglobin, lipid profile were recorded. All patients were advised to control diabetes and other associated systemic disorders, patching of the eye and to undergo ocular physiotherapy. Patients were advised to undergo regular follow up 2 weeks, 4 weeks, 8 weeks and 12 weeks.

RESULTS

RBS (mg/dL)	N	Minimum	Maximum	Mean	SD
Week 0	30	169.0	472.0	291.60	76.57
Week 2	30	138.0	398.0	258.47	69.95
Week 4	30	137.0	350.0	232.23	58.99
Week 8	30	113.0	361.0	206.23	52.02
Week 12	30	141.0	281.0	193.03	34.97

Table 1. RBS Level with Respect to Cranial Nerve Palsies in 0-, 2-, 4-, 8- and 12-Weeks

p<0.001

HbA1c%	N	Minimum	Maximum	Mean	SD
Week 0	30	6.2	12.6	9.22	1.75
Week 12	30	6.3	9.8	7.39	0.83

Table 2. HbA1c Levels with Respect to Cranial Nerve Palsies in 0- and 12-Weeks

p<0.001

Associated Comorbidities	No. of Patients	Percent
CKD, DYSL	1	3.3
HTN	18	60.0
HTN, CVA	1	3.3
HTN, HYPO	1	3.3
NIL	9	30.0

Table 3. Association and Frequency of Other Systemic Comorbidities

Variables	Remarks	Mean	SD
Duration of DM in years	ER	6.88	5.33
	R	6.18	4.51
RBS (mg/dL) 0 weeks	ER	267.86	53.74
	R	298.83	81.90
RBS (mg/dL) 4 weeks	ER	213.71	52.54
	R	298.83	60.76
RBS (mg/dL) 12 weeks	ER	177.77	12.16
	R	197.74	58.37
HbA1c (%) 0 weeks	ER	9.16	1.18
	R	9.40	1.76
HbA1c (%) 12 weeks	ER	7.26	1.18
	R	7.43	0.73

Table 4. Recovery of Cranial Nerve Palsies with Respect to Various Parameters

Interpretation

Out of 30 patients, 13 were identified in the age group of 51-60 years who contributed maximally to the palsies. Mean age group of 50 years had shown early recovery compared to mean age group of 60 years. Males contributed 53.3% (16 patients) while females contributed to 46.7% (14 patients) showing male predominance. In our study, all 30 patients were diagnosed with type 2 diabetes mellitus. A total of 67% (21) patients had sixth cranial nerve palsy among which 1 patient had bilateral sixth cranial nerve palsy. 33%(9) patients had third cranial nerve palsy among which one patient had recurrent episode of palsy within a span of 2 months period. No cases of fourth nerve palsy were reported. Mean duration of diabetes mellitus was 6.8 years. Mean RBS level 236 mg/dL and the mean HbA1c level was 8.3 gm%. Most common association noted was hypertension (40%), others like CKD (4%), CVA (3%) and hypothyroid (3%) and 40% patients presented with only Diabetes. Early recovery of cranial nerve palsies was noted in 23% (7 patients) as early as 4 to 8 weeks and remaining 77% (23 patients) recovered by 12 weeks. No relation is noted with respect to duration of diabetes mellitus in case of early recovery of palsies. All the patients showed recovery by 3rd month follow up.

DISCUSSION

Oculomotor cranial nerve palsies are more common in diabetes mellitus. Eman et al[4,5] and Suman Adhikari[6] et al in 2013, noticed that sixth cranial nerve palsy was most encountered cause and fourth nerve palsy was the least frequent cause. The likely explanation for the high frequency involvement of sixth cranial nerve is related to its length which exposes it to ischemia secondary to angiopathic changes. Said G[7] in his study concluded that age 45 years or more is the most important and significant risk factor for ophthalmoplegia. Tiffin PA et al[8] in his study shown that average age of onset of ischemic ophthalmoplegia was 37 years and in our study mean age of onset was 55 years. Mean duration of diabetes was 5 years which was significant independent risk factor for cranial nerve palsies unlike in the study done by Boulton AJM[9] diabetes duration of 10 years or more was significant risk factor.

According to study done by Patel et al[10] micro vascular palsies most commonly occur in older age of 50 years or more with existing vascular risk factors like diabetes, hypertension, hyperlipidaemia and smoking. Hypertension was the most common association noticed in our study but is not an independent risk factor and did not increase the risk of ophthalmoplegia but it may worsen the effect of diabetes in cranial nerve palsies. In our study, it is proven that poor glycaemic control is one of the significant risk factor for diabetic ophthalmoplegia which is similar to the findings of study done by Ostricet et al[11] and Kobashi et al.[12] According to study done by Bhanumurthy G[13] concluded that cranial nerve palsies showed slow recovery when the patients presented with uncontrolled diabetes and diabetes of longer duration and recovery was faster when patient presented early which is in correlation with our study where the uncontrolled diabetes showed slow recovery. Ayse Ilsksen Colpak[14] et al in his study showed that mean recovery duration for oculomotor nerve palsy was 35 to 135 days and for abducens nerve palsy was 30 to 163 days which is in correlation with our study where mean recovery time of oculomotor and abducens nerve palsies was noticed from 60 to 90 days. In our study, of 30 patients, 23% (7 patients) recovered early by 6 to 8 weeks and remaining (23 patients) 77% recovered by 12 weeks of which abducens nerve palsies recovered earlier compared to oculomotor nerve palsies.

Limitations

1) Small sample size
2) Short follow up period
3) Patients lost to follow up (10 patients)

CONCLUSIONS

In patients aged 50 years or older, diabetic age of 5 years or more, uncontrolled blood sugar level, and male gender, isolated oculomotor palsies are most likely due to microvascular ischemia. But the presence or absence of diabetic retinopathy and nephropathy was not a significant risk factor in our study. Patients who are not known to have systemic comorbidities should strictly be examined for diabetes, hypertension and hyperlipidaemia and treatment should be started accordingly. The most commonly affected cranial nerve in our study was abducens nerve and the most common association of diabetes mellitus noticed was hypertension. Majority of cases of ischemic oculomotor nerve palsies showed spontaneous recovery by 3 months with medical treatment and with good control of blood glucose level. Worsening of the symptoms, non-improvement for a period of 3 months or greater involvement of other cranial nerves, isolated cranial nerve palsies in the absence of vasculopathic risk factors, neuroimaging done to rule out the other causes.

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