

Clinical Profile of Ocular Motor Nerve Palsies at Tertiary Eye Care Centre in South India

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Abstract

Introduction: The diagnosis and management of ocular motor nerve dysfunction varies according to the age of the patient, characteristics of the ocular motor nerve palsies, and presence of associated symptoms and signs.

Purpose of the Study: To analyse the various etiology, clinical presentations, recovery pattern in patients with third, fourth, and sixth cranial nerve palsies in Indian population.

Methods: This is a prospective study of patients diagnosed with ocular motor nerve palsies presented at tertiary eye centre within period of 2012 - 2015.

Results: We identified 150 patients diagnosed with involvement of third, fourth, sixth and multiple cranial nerve palsies. Mean age of onset was 54.7 years (23 - 81 years), with female preponderance 51.3% (77 females). Majority of our patients presented with double vision in 87 (58%) cases, followed by ptosis in 27 (18%) cases; headache and pain in eyes in 19 (12.7%) cases and 11 (7.3%) cases respectively. The most common cranial nerve palsy was the sixth cranial nerve with 58 cases (38.7%). Of 150 cases, the causes were ischemia 67 (44.7%), trauma 37 cases (20.7%), idiopathic 18 (12%). Pupil was spared in 26 cases (78.8%) in ischemic palsies, while pupil was involved in 3 cases (66%) in neoplasms. We found no case of aneurysms causing third nerve palsy. Cranial nerve palsy patients from ischemic cause spontaneously improved 49 of 67 cases (73%) in 6 months.

Conclusions: Sixth cranial nerve palsy was the most common cranial nerve palsy. Most common etiology was ischemia. Majority of patients with cranial nerve palsy from ischemic cause spontaneously improved in 6 months. Neuroimaging plays important role to evaluate, diagnose and treat acute ocular motor palsies.

Keywords: Diplopia; Cranial Nerve Palsy; Recovery; Diagnostic Yield; Neuroimaging

Introduction

Extraocular movements are controlled by third, fourth, and sixth cranial nerves. Acquired palsy of these nerves can result from various causes such as trauma, vascular disease, intracranial tumours, or aneurysm, and so on. A determination of the cause and treatment of a cranial nerve dysfunction is critical because without treatment such cases can be fatal. The diagnosis and management of ocular motor nerve dysfunction varies according to the age of the patient, characteristics of the ocular motor nerve palsies, and presence of associated symptoms and signs. There have been several extensive statistical studies on cranial nerve dysfunction [1-5].

However, studies showed variety in the etiology and affected nerve distribution and wide range of recovery rates [2-7]. The present study was conducted at tertiary eye centre in South India to analyse cases of acquired ocular motor palsies. We also determined the incidence of pupillary abnormalities and various clinical presentations associated with ocular motor nerve palsies, neuroimaging and the recovery of each type of nerve dysfunction.

Materials and Methods

It is a prospective study, consisting of 150 cases diagnosed with acquired third, fourth, and sixth cranial nerve palsy, presenting to neurophthalmology and strabismus department, between November 2012 to December 2015 were included. Institutional Review Board Ethics Committee approval was obtained; it was carried out in adherence to the tenets of the Declaration of Helsinki. Demographic, etiological, diagnostic and recovery data was obtained. A detailed medical history and past history of the subjects was noted. All patients were subjected to a comprehensive ocular examination, which included visual acuity and slit lamp biomicroscopy. Particular attention was given towards lid examination, pupillary reflexes, and extraocular movements. All patients were reviewed again after 1 month, 3 months and 6 months from the baseline visit. Lid position, extra-ocular movements, pupil and reaction to light were recorded at every visit. Degree of ocular motor restriction in the worst motion direction, the deviation angle at each visit, presence of other related ophthalmic or neurological signs, and the results of neuroradiological tests were also recorded. Deviation angle was measured with the prism cover test in the primary position at distance fixation. Cases were categorized into six etiological groups: ischemic, trauma, aneurysm, neoplasm, undetermined, or others.

Ischemia was determined to be the cause when other causes had been ruled out and the patient had a positive history of vascular disease such as diabetes mellitus, hypertension, or arteriosclerosis. Congenital palsy and conditions that mimic cranial nerve palsy, such as orbital wall fracture, myasthenia gravis, and thyroid orbitopathy, were excluded. Based on clinical diagnosis appropriate investigations were performed and treatment was given. Cases were followed up to maximum of 6 months for any signs of recovery.

Data Analysis

The data was collected and entered onto the Microsoft excel 2007 spread sheet. Statistical analysis was performed by the statistical software STATA 11.1 (Texas, USA). Continuous variables were represented as 'Mean (SD)', and categorical variables were represented as 'Frequency (percentage)'. Chi square test/Fisher's exact tests was used to assess differences between categorical variables. $P < 0.05$ was considered as statistically significant.

Results

A total of 150 patients were included, fulfilling inclusion and exclusion criteria, with mean age of onset being 54.7 years ranging between 23 years to 81 years with 77 females and 73 males.

Majority of our patients presented with double vision in 87 (58%) cases, followed by ptosis in 27 (18%) cases; headache and pain in eyes in 19 (12.7%) cases and 11 (7.3%) cases respectively. Some patients had other complaints including diminution of vision, defective side vision, blurring of images, inability to read, difficulty in using staircase.

Sixth nerve involvement was most common in 58 (38.7%) cases, while presumed ischemia was the most common etiology in 67 (44.6%) cases.

The involvement of the affected nerves with its etiological distribution is summarized in Table 1.

S.no	Cranial nerves				Total	P value*
	6 th	3 rd	4 th	Multiple nerves		
N	58	54	17	21		0.615
Etiology						
Ischemic	30	25	4	8	67	
Traumatic	8	11	7	5	31	
Aneurysm	1	0	0	0	1	
Neoplasm	0	2	0	1	3	
Idiopathic	7	6	3	2	18	
Others	12	10	3	5	30	

Table 1: Showing distribution of cranial nerve involvement and various etiology.

N: total number of the patients

In patients with third nerve palsy, there was no involvement of pupil in 26(79%) cases with vascular etiology and 1 case of neoplasm. Table 2 describes the involvement of pupil according to the etiology.

Etiology	Pupil		Total	P-value*
	PS	PI		
Ischemic	26 (78.8)	7 (21.2)	33	< 0.001
Traumatic	1 (6.3)	15 (93.7)	16	
Idiopathic	7 (87.5)	1 (12.5)	8	
aneurysm+neoplasm	1 (33.3)	2 (66.7)	3	
Others	2 (13.3)	13 (86.7)	15	
Total	37 (49.3)	38 (50.7)	75	

Table 2: Showing pupil involvement in all cases with isolated third nerve palsy and multiple cranial nerve palsy.

PS: Pupil Sparing; PI: Pupil Involving

Two of our patients had pituitary adenoma with lateral extension resulting in third nerve palsy. Others causes include, a patient of Paget’s disease of skull leading to thickening of skull and resulting in compression of the nerve, similarly another patient had compression resulting due to closely related posterior cerebral artery. One patient had Weber disease; one patient with subarachnoid hemorrhage; five patients with idiopathic orbital inflammation. Some of the patients with fourth nerve palsy were associated with maxillary sinusitis, post viral syndrome. We had only one case of aneurysm of internal carotid artery causing sixth nerve palsy. Other causes for sixth nerve palsy includes carotid cavernous fistula, meningitis, postviral infection and idiopathic intracranial hypertension. Idiopathic inflammatory disease of orbit emerged as a important cause affecting multiple cranial nerves in 5 patients. One of the patient with multiple nerve involvement was diagnosed with carcinoma of nasopharynx.

In forty-three patients, above 50 years of age with history of vasculopathic factors, improving on follow up did not underwent neuroimaging due to presumed ischemic cause. Neuroimaging was done in 107 patients, of which in 53 patients had significant findings in MRI scan, giving diagnostic yield of 50.4%. In our study, diagnostic yield was maximum in detecting inflammatory disorders like idiopathic inflammatory disease of orbit, traumatic cases, neoplasms.

After 6 month of follow up, complete recovery was seen in 69 patients, which was maximum for cases of third nerve palsy (27 cases); followed by sixth nerve palsy (24 cases); ten cases with multiple cranial nerve palsy and 8 cases of fourth palsy recovered completely. Eight (5.3%) cases showed no signs of recovery until 6 months.

Discussion

We studied the clinical profile and neuroimaging of the patients with acute ocular motor nerve palsy, presenting to our tertiary eye care centre. Our mean age of presentation was 54.7 years, which is somewhat younger than 65.6 years [8], however mean age of even 48.1 years have been reported [9]. Due to high prevalence of inflammatory, structural and infectious causes, age at presentation is now become a important consideration in terms of work up for causes. In our study, affection of cases of third nerve, sixth nerve and multiple cranial nerves were above 60 years of age, while affection of fourth nerve was distributed equally in all age groups. But the earlier studies, done by Menon., *et al.* [10] found majority of patients with third nerve palsy and multiple nerve involvement in between 11 - 40 years of age. Our data confirmed the nerve distribution reported in previous studies which found sixth nerve involvement to be most common; while fourth nerve involvement to be least [1,9,11]. In our study patients with multiple nerve involvement (14%) was higher compared to other studies ranging from 9 - 35.5% [1,12]. Various etiologies causing acute onset ocular motor cranial neuropathies, includes presumed microvascular ischemia, inflammation, trauma and compression. Ischemic etiology was most common affecting 44.6% cases vs 34.8% and 31% in previous studies, similarly trauma was the cause in 20% of cranial nerve palsy in our population vs 15% - 21% in other studies [1,9]. The differences in the various etiologies can be attributed to the different and ambiguous criteria used for recruitment of the patients in respective studies.

However, we had only 3(2%) cases of neoplasm and one case of aneurysm while other studies have reported it over varied range. In 79% patients of ischemic third nerve palsy pupil was spared, which was in accordance with other studies by Dhume., *et al.* [13] (74.3%), Lee., *et al.* [14] (73%), Schultz., *et al.* [15] (60%).

Certain causes are more commonly associated with specific cranial nerve involvement. Any lesion resulting in increase in intracranial pressure, commonly leads to sixth nerve paresis; fourth nerve palsy is common following closed head injury. Pupil involving third nerve palsy, aneurysm is the most important consideration and appropriate neuroimaging need to be done. Contrary to this our study showed no case of posterior communicating aneurysm causing third nerve palsy. Rama., *et al.* [12] also found only one case of aneurysmal etiology in their 90 patients and suggested that aneurysms are less common in South India. In our study 10 patients having vacuopathic factors were found to have other non-microvascular causes resulting in paresis.

The causes like idiopathic inflammatory orbital disease, compressive lesions like pagets disease, subdural hemorrhage, compression due to posterior cerebral artery which could have missed if neuroimaging was not performed in these cases.

In a study of 109 cases, neuroimaging and other studies identified a non-microvascular cause in 18 patients (16.5 %, 95% CI 10.7 - 24.6%) [8]. In another prospective study of isolated ocular motor palsies done by Chou., *et al.* [16] found, that over 50% of patients with non-microvascular palsies had vasculopathic risk factors.

Neuroimaging procedures may have a role in the initial evaluation of patients 50 years of age or older with acute ocular motor palsies. In a study done by Tamhankar., *et al.* [8], they suggested that excluding third nerve palsy and giant cell arteritis cases there was approximately 1 in a 20 chance that a patient with vasculopathic risk factors alone had another cause found for the 4th and 6th cranial nerve palsy.

There were few limitations of our study, that we could follow up patients only till 6 months and neuroimaging was not done in all cases due to financial constraints of patients and presumed vascular etiology.

Conclusion

In our study, there was increased incidence of sixth nerve palsy followed by third nerve palsy. The most common etiology was ischemia. Majority of pupil involving palsies were secondary to trauma. There were no cases of third nerve palsy due to aneurysms. Recovery was

maximum in etiologies like ischemia and inflammatory orbital disease. In spite of advanced neuroimaging technique idiopathic cause was still seen. The age of the patient, signs of an improvement, and associated neurological deficits are important diagnostic markers to determine the best type of imaging methods for evaluating neurologically isolated third, fourth, and sixth cranial nerve dysfunction. Neuroimaging procedures may have a role in the initial evaluation of patients 50 years of age or older with acute ocular motor palsies.

Conflict of Interest

Authors don't have any conflict of interest.

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