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# A Clinical and Aetiological Study of Ocular Motor Nerve Palsy

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**Abstract**

*Aim:* To clinically evaluate cases of ocular motor nerve palsy and to diagnose their possible aetiological causes as they are often perceived as a sign of serious underlying pathology such as intracranial aneurysms.

*Purpose:* To clinically evaluate cases of ocular motor nerve palsy so as to make anatomical localization and also to make possible aetiological and pathological diagnosis of the neurological lesion. Anatomical localization and aetiopathological diagnosis of the neurological lesion will help the attending neurologist in better management of the case.

*Introduction:* Ocular motor nerve palsies may be congenital or acquired, complete or partial, pupil sparing or pupil involving, and isolated or multiple accompanied by signs of more extensive neurological involvement. Precise knowledge of its origin and course from nuclear level to terminal muscles along with associated clinical features helps in localization and management of neurological lesions.

*Materials And Methods:* 50 consecutive cases of ocular motor nerve palsy attending the outpatient clinic of the department of ophthalmology Malla Reddy Hospital, Malla Reddy Institute of Medical Sciences or referred from other specialties are evaluated and investigated thoroughly as a Prospective, observational, non – interventional and Hospital study study from 1st January 2018 to 31st December 2018. All underwent complete ophthalmological, medical, neurological, otorhinolaryngological and general examination along with complimentary investigations and neuroradiological imaging where ever possible.

*Inclusion Criteria:* Acquired ocular motor nerve palsy with a recent onset (within two weeks), all age groups and both sexes included, ocular motor nerve palsies associated with other neurological signs and symptoms other than the palsy itself and acceptance of the patients to undergo investigations wherever needed.

*Exclusion Criteria:* Congenital ocular motor nerve palsy, patients with incomitant squint due to myogenic, myasthenic and restrictive causes, patients who were terminally ill and those palsies secondary to neurosurgical causes were excluded from the study.

*Results:* Paralyses of the sixth and third cranial nerve were the most common. Complete ptosis and full mydriasis were mostly seen in isolated cases of third carnial nerve palsy. Majority of them are pupil sparing. Common causes were vascular, otorhinolaryngological and trauma. Micro vascular ischemia group as an aetiological factor has good recovery rate and so is the case with pupil sparing oculomotor nerve palsy.

*Conclusion:* Proper evaluation of cases of ocular motor nerve palsy in close collaboration with other specialists will go a long way not only in localizing the serious neurological lesion and also help in reducing the mortality, morbidity and better management of ocular motility disorder.

**Keywords:** Isolated Ocular Motor Nerve Palsy; Pupillary Sparing Microvascular Ischemia.

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## Introduction

The oculomotor nerve is entirely motor in function; it supplies the superior rectus, inferior rectus, inferior oblique, medial rectus and levator palpebrae superioris, as well as autonomic pupillary sphincter and ciliary muscles of the eye. The most common etiology is ischemia due to diabetes

mellitus. Etiologies causing multiple cranial nerve palsies include head trauma, vascular pathology, tumours and inflammation due to infectious and non-infectious origin. Interaction between various specialists is very essential for the evaluation and management of the oculomotor nerve palsy.

## Materials and Methods

After obtaining the approval of the institutional ethics committee, the informed consent from the patients were obtained. The study was conducted for a period of 12 months from January 01.2018 to 31st December 2018. The sample size consisted of 50 cases of oculomotor nerve paralysis, presenting themselves directly to the department of ophthalmology or referred by various other departments were studied and analysed. A standard case protocol was maintained, in the history emphasis was given for drooping of eye lid(s), headache, diplopia, fever, trauma, vomiting, convulsions, history of exposure etc. In past history emphasis was given to diseases such as diabetes mellitus, hypertension, and tuberculosis. Current or past medications if any was noted, any history of addictions was also noted. The visual acuity and colour vision was measured using conventional methods, detailed slit lamp examination, thorough clinical examination was done including symmetry of face, head posture, and extraocular movements in all nine cardinal positions of gaze, Hirschberg's test, cover test, prism cover test and aberrant regeneration were performed. Ptosis evaluation and grading of ptosis if present was done as follows: mild (< 2 mm), moderate (2-4mm) and severe (4-8mm). Size, shape and light reflexes of the pupils were noted, the normal pupillary diameter was standardized to 3 mm. Miosis and mydriasis were considered when the diameter was less than 3mm and greater than 7mm respectively. The degree of anisocoria, if present, was recorded. Physiological anisocoria was ruled out after repeating the measurements in dim light. Detailed fundus examination was done and the findings were recorded. Detailed systemic examination was done with special emphasis to central nervous system, cardiovascular and endocrinological examination. Blood pressure measurement, routine blood investigations including blood sugar levels, erythrocyte sedimentation rate and serum cholesterol, VDRL and HIV antibodies, radiological examination of skull, orbital fissures, optic foramina, paranasal sinuses, computed tomography and MRI–scan were performed wherever indicated. They also underwent otorhinolaryngological examination. All patients were reviewed after two weeks, eight weeks, twelve weeks and at the sixth month.

### *Inclusion Criteria*

Aquired oculomotor nerve palsy with a recent onset (within two weeks), all age groups and both sexes were included, oculomotor nerve palsy associated with other neurological signs and symptoms other than the palsy itself and acceptance of the patients to undergo investigations where ever needed.

### *Exclusion Criteria*

Congenital oculomotor nerve palsies, patients with incomitant squint due to myogenic, myasthenic and restrictive causes, patients who were terminally ill, oculomotor nerve palsy secondary to neurosurgery were excluded from the study.

## Results

**Table 1:** Percentage of incidence of oculomotor nerve Palsy

| **Total No. Of patients seen in OPD from January 2018 to December 2018.** | **40151** |
|---|---|
| Total No. Of oculomotor nerve palsies examined | 50 |
| Percentage Of Incidence | 0.12453 |

The percentage of incidence of oculomotor nerve palsy in the present study is 0.12453% (Table 1).

**Table 2:** Gender incidence

| **Sex** | **Number** | **Percentage** |
|---|---|---|
| Male | 18 | 36 |
| Female | 32 | 64 |

There is a female preponderance in incidence (Table 2).

**Table 3:** Age incidence

| **Age Group** | **Number** | **Percentage** |
|---|---|---|
| 21-30 | 6 | 12 |
| 31-40 | 8 | 16 |
| 41-50 | 9 | 18 |
| 51-60 | 14 | 28 |
| 61-70 | 10 | 20 |
| >70 | 3 | 6 |

Highest incidence is seen in 51-70 age group (48%) (Table 3).

**Table 4:** Laterality

| Laterlity | Number | Percentage |
|---|---|---|
| Unilateral | 41 | 82 |
| Bilaterral | 9 | 18 |

Unilateral incidence is more (82 %). All the cases with bilateral oculomotor nerve palsy had lesions located in the midbrain involving the oculomotor nucleus complex (Table 4).

**Table 5:** Mode of presentation of oculomotor nerve palsy

| Type of Invovement | Number | Percentage |
|---|---|---|
| Isolated Nerve Palsy | 38 | 76 |
| Multiple cranial nerve palsy | 12 | 24 |

Incidence of isolated third nerve palsy is more (76%) (Table 5).

**Table 6:** Classification of patients by pupil involving / pupil sparing

| Pupil Involving / Pupil Sparing | Casess | |
|---|---|---|
| | Number | Percentage |
| Pupil involving | 18 | 36 |
| Pupil sparing | 32 | 64 |

Incidence of pupil sparing palsy is more (64%) (Table 6).

**Table 7:** Oculomotor nerve palsy according to aetiology

| Aetiology | Number | Percentage |
|---|---|---|
| Microvascular ischemia | 24 | 48 |
| Posttraumatic | 7 | 14 |
| Intracranial anesurysm | 1 | 2 |
| Neo plasm | 4 | 8 |
| Orbital inflammatory group | 7 | 14 |
| Undertermind cause | 7 | 14 |

Incidence of microvascular ischemia as a aetiological factor is more (48%) (Table 7).

**Table 8:** Oculomotor nerve palsy due to systemic vascular deseases.

| Systemic Desease | Number | Percentage |
|---|---|---|
| Diabetes | 23 | 46 |
| Hypertension | 05 | 10 |

Incidence of diabetic palsy is more (46%) among systemic aetiological factors (Table 8).

**Table 9:** Symptoms

| Symptoms | Number | Percentage |
|---|---|---|
| Visual impairment | 7 | 14 |
| Ptosis | 22 | 44 |
| Diplopia | 16 | 32 |
| Ocular deviation | 36 | 72 |

Ocular deviation as a symptom is of higher incidence (72%) (Table 9).

**Table 10:** Ptosis

| Symptom | Number | Percentage |
|---|---|---|
| Total number of cases with ptosis | 23 | 46 |
| Mild ptosis | 10 | 43.43 |
| Moderate ptosis | 6 | 26.08 |
| Complete ptosis | 7 | 30.43 |

Incidence of ptosis is about (46%) among which milder ptosis incidence is higher (43.43%) (Table 10).

**Table 11:** Recovery Pattern in Isolated OMNP according to Aetiology

| Aetiology | Complete Recovery | | Partial Recovery | | Lost During Followup | |
|---|---|---|---|---|---|---|
| | No. | % | No. | % | No. | % |
| Micro Vascular Ischemia | 18 | 47.36 | 6 | 16.78 | - | - |
| Post Traumatic | - | - | 6 | 15.78 | - | - |
| Intracranial Aneurysm | - | - | - | - | 01 | 2.63 |
| Benign Third Nerve Palsy | 02 | 5.26 | - | - | - | - |
| Undetermined Group | 03 | 7.89 | 03 | 7.89 | - | - |

Complete recovery is seen more in microvascular ischemia as an aetiological factor (Table 11).

**Table 12:** Recovery pattern in OMNP associated with multiple cranial nerve palsy according to aetiology of palsy

| Aetiology | Complete Recovery | | Partial Recovery | | Lost During Followup | |
|---|---|---|---|---|---|---|
| | No. | % | No. | % | No. | % |
| Orbital inflam-mantory group | 5 | 41.66 | 03 | 25 | - | - |
| Post traumatic | - | - | 02 | 16.66 | - | - |
| Neoplasm | - | - | - | - | 02 | 16.66 |

Complete recovery in multiple cranial nerve palsy is seen more in orbital inflammatory group as aetiological factor (Table 12).

**Table 13: R**ecovery pattern according to pupillary involvement

| Pupillary Involvement | Complete Recovery | | Partial Recovery | | Lost During Followup | |
|---|---|---|---|---|---|---|
| | No. | % | No. | % | No. | % |
| Pupil spared | 23 | 46 | 9 | 18 | - | - |
| Pupil involved | - | - | 12 | 24 | 6 | 12 |

Pupil sparing palsys show higher complete recovery incidence (46%) (Table 13).

## Discussion

**Table 14:** Age incidence

| Study | Age Group | Percentage |
|---|---|---|
| Present Study | 51 To 70 Years | 48 |
| Vimala Menon And Co-Workers | 11 To 40 Years | 71 |

In the present study maximum number of patients belonged to between age group 51 to 60 years 28% (fourteen patients) and 61 to 70 years 20% (ten patients), while VIMALA MENON and co-workers found maximum incidence of oculomotor nerve palsy (71%) in the 11 to 40 years age group [1]. (Table 14).

**Table 15:** Gender incidence

| Study | Male | Percentage | Female | Percentage |
|---|---|---|---|---|
| Present Study | 18 | 36 | 32 | 64 |

Out of 50 patients, 64% (32patients) were females and 36% (eighteen patients) were males. Green and co-workers have reported equal sex distribution [2].

Unilateral oculomotor nerve palsy was seen in 82% (Forty one) patients, out of which right eye oculomotor nerve palsy was observed in 75.60% (thirty one) patients, left eye oculomotor nerve palsy was observed in 24.39% (ten) patients and 18% (nine) patients had bilateral oculomotor nerve palsy, the results could be compared to earlier studies by Green and co-workers and Rush and Younge [2,3]. (Table 15).

**Table 16:** Isolated oculomotor nerve palsy

| Study | Percentage |
|---|---|
| Present study | 76 |
| Ruker | 68.5 |
| Richards and co-workers | 68.1 |

In the present study the number of isolated oculomotor nerve palsy is 76%, compared to studies by Rucker 68.5% and Richards and co-workers 68.1 % [4,5] (Table 16).

**Table 17:** Predominant cause oculomotor nerve palsy

| Study | Cause | Percentage |
|---|---|---|
| Present Study | Microvascular Ischemia | 48 |

Studies by Green and workers, Rush, Rucker and Younge found similar results. In the present study, Microvascular Ischemia (48%) emerged as the predominant cause for oculomotor nerve palsy where a specific aetiology could be determined, which could be compared to most of the earlier studies who have found similar results [2,5] (Table 17).

**Table 18:** Post – traumatic oculomotor nerve palsy

| Study | Percentage |
|---|---|
| Present study | 14 |
| P.Muthu And P.Pritty | 15 |
| Rush and Younge series | 16.2 |
| Richards and Younge series | 14.7 |

Post-traumatic oculomotor nerve palsy accounted for 14% of total cases in the present study, compared to a study by P. Muthu and P. Pritty in June 2000,they found a 15% occurrence of isolated 3rd nerve palsy attributable to head trauma [6]. Rush and Younge series (16.2%) [3], and Richards and Younge series (14.7%) [5] (Table 18).

**Table 19:** Idiopathic orbital inflammatory group as cause of omnp.

| Study | Percentage |
|---|---|
| Present study | 14 |
| Vimala menon series | 9.5 |

In the present study, idiopathic orbital inflammatory group comprised of 14% cases, compared to vimala menon series (9.5%), [1] only such study where orbital inflammatory diseases were considered as a separate group (Table 19).

**Table 20:** Intracrainal neoplasms as a cause of omnp

| Study | Percentage |
|---|---|
| Present study | 8 |
| Vimala menon series | 9.5 |

Intracranial Neoplasms were suspected in 8% of cases, compared to Vimala Menon [1] series (9.5%). (Table 20).

**Table 21:** Intracranial aneurysms as cause of omnp

| Study | Percentage |
|---|---|
| Present Study | 2 |

Studies by Rush, Rucker and YOUNGE had higher incidence. Intracranial aneurysms causing oculomotor nerve palsy were diagnosed in only 2% of cases in the present study while most of the earlier studies [3,4,5,7] had higher incidence of aneurysms (Table 21).

**Table 22:** Undetermined aetiology as a cause of omnp

| Study | Percentage |
|---|---|
| Present Study | 14 |

In The Present Study, Etiology Was Undetermined In 14% Cases (Table 22).

**Table 23:** Complete recovery of omnp

| Study | Percetnage |
|---|---|
| Present study | 46 |
| V.P.Singh Et Al | 50 |
| Rush and younge | 44.6 |

In the present study complete recovery was seen in 46% of cases compared to earlier studies by V.P. Singh et al. [9] (50%) and Rush and YOUNGE series (44.6%) [3] (Table 23).

**Table 24:** Micro vascular group as a cause for best recovery in omnp.

| Study | Percentage |
|---|---|
| Present Study | 48 |

Study by Rush and Younge series show comparable results. Microvascular ischemia group had the best recovery (48%) which is comparable to previous study by Rush and Younge series [3] (Table 24).

**Table 25:** Pupil sparing/involving for complete recovery of omnp.

| Pupil | Percentage of Complete Recovery |
|---|---|
| Pupil Sparing | 64 |
| Pupil Involving | 36 |

Pupil sparing ocular motor nerve palsy (64%) had better prognosis for complete recovery compared to (36%) pupil involving ocular nerve palsy (Table 25).

## Conclusion

Isolated oculomotor nerve palsy is the predominant mode of presentation which has a good recovery rate, microvascular ischemia appears to be the major etiological factor and the prognosis for complete recovery is good, but when oculomotor nerve palsy is associated with multiple cranial nerve palsies or other neurological features, the chance of complete recovery is less. Even though certain etiological factors such as microvascular ischemia can be done using simple laboratory investigations, interaction with other specialists helps in guiding the diagnosis, evaluation, in predicting prognosis and efficient management.

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