A Comprehensive Review on the Management of III Nerve Palsy

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Abstract
The third cranial nerve is a motor nerve chiefly involved in execution of movements of the eye. The paresis or paralysis of the one or more of these muscles due to oculomotor nerve palsy, leads to ptosis, anisocoria and ocular motility defects. This article highlights the origin and course from nuclear level to terminal branches along with associated clinical symptoms and signs that help in localizing the site of lesion and planning appropriate management.

Keywords: oculomotor nerve, paralysis, management

Introduction
The third cranial nerve is a motor nerve chiefly involved in execution of movements of the eye. Also known as the oculomotor nerve, it supplies all the extraocular muscles except for lateral rectus and superior oblique. Thus it helps in carrying out the extraocular movements efficiently improving the binocular field of vision. The chief muscles being supplied by the third nerve are the Superior Rectus, Inferior Rectus, Medial Rectus and Inferior Oblique which are responsible for the elevation, depression, adduction and extorsion of the eye respectively. In addition, the ciliary muscle and the sphincter pupillae are supplied by the parasympathetic fibres from the Edinger-Westphal nucleus and are responsible for the accommodation and pupillary constriction. The levator palpebrae superioris (LPS) which elevates the eyelid is also supplied by the oculomotor nerve. The paresis or paralysis of the one or more of these muscles causes ptosis, anisocoria and ocular motility defects. The unopposed action of the lateral rectus and superior oblique muscles results in fixed eye in a down and out position in cases with complete paralysis.

The involvement of the third nerve could be congenital or acquired in nature. The main causes of acquired third nerve palsy include: infections (CNS or local), trauma, direct or indirect compression of the nerve anywhere along its path, vascular conditions (ischemic/aneurysms), neoplastic, inflammatory or demyelinating diseases. It could be complete or partial, pupil-sparing or involving, isolated or associated with other neurological symptoms. Precise knowledge of its origin and course from nuclear level to terminal branches along with associated clinical symptoms or signs helps in localizing the site of lesion and planning appropriate management.

The search of published literature for this review article had been completed using Ovid, Medline, Embase, Pubmed over the last 5 decades along with the checking of cross references also. English language articles with full text access were included and electronic literature search was performed using oculomotor nerve, palsy and management as key words. While reviewing the literature, parameters evaluated were applied neuroanatomy, related syndromes, medical management and surgical management modalities for oculomotor nerve palsy.

Applied Neuro-Anatomy
Nuclear Complex - The location of nuclear complex of the third nerve is in the midbrain at the level of the superior colliculus ventral to the Aqueduct of Sylvius, the right and left components straddling the midsagittal plane. The central caudal nucleus (unpaired) in the midline innervates both the right and left levator palpebrae muscles. Thus lesion at this level results in bilateral ptosis. The superior rectus fascicles decussate within the nuclear complex and innervate the contralateral superior rectus. The medial rectus, inferior rectus and inferior oblique muscles and the parasympathetic pathways are supplied by the ipsilateral subnuclei which are paired. Caudal nuclear lesions may spare the pupil and rostral lesions may present without ptosis. Lesions involving the nucleus, due to infarction, demyelination, inflammation, primary tumours or metastasis are uncommon.

Fasciculus - The fascicles travel ventrally through the tegmentum, passing through the red nucleus and the medial aspect of the cerebral peduncles, emerge from the midbrain and pass into the interpeduncular fossa. Fascicular involvement occurs in compressive lesions (primary tumor or metastasis), infarction, hemorrhage and demyelinating diseases. Classically described midbrain syndromes arising out of lesions affecting the fasciculus are as mentioned in the Table 1.

Subarachnoid space (Basilar) - The rootlets emerge from the brainstem medial to the cerebral peduncle and unite to form the main trunk. This trunk traverses between the posterior cerebral and superior cerebellar arteries passing lateral to the posterior communicating artery along the base of the skull, in the subarachnoid space. Aneurysms arising at the junction of posterior communicating artery and middle cerebral arteries can affect the nerve by compression or acute hemorrhage resulting in acute painful ophthalmoplegia. Lesions at this level typically result in isolated third nerve palsy. Involvement of nerve at this level may be idiopathic in 25% of the cases. Pupillomotor fibres lie superomedial in the periphery of the nerve making them prone to injury
The downward herniation of the uncus 7,16,17,18

Features

Level of Lesion

Cerebral peduncle

Fasciculus + Superior

Cerebral Peduncle

Combination of Benedikt and Nothnagel syndrome

| Syndrome                  | Level of Lesion          | Features                        |
|---------------------------|--------------------------|---------------------------------|
| Weber Syndrome            | Cerebral peduncle        | Ipsilateral III nerve palsy + Contralateral hemiparesis |
| Benedikt Syndrome         | Red Nucleus              | Ipsilateral III nerve palsy + Contralateral extrapyramidal signs |
| Nothnagel Syndrome        | Fasciculus + Superior    | Ipsilateral III nerve palsy + Cerebellar Ataxia |
| Claude Syndrome           | Combination of Benedikt and Nothnagel syndrome |

by external compression or trauma and are spared by the ischemic events.8 The downward herniation of the uncus in the temporal lobe by extradural or subdural hematoma caused by head trauma or an expanding supratentorial mass can stretch or compress the third nerve against the tentorial edge, initially causing irritative miosis followed by pupillary dilatation (Hutchinson’s pupil) and later total oculomotor nerve palsy. “The rule of the pupil” suggests that if a compressive lesion causes a third nerve palsy, the chances of pupil involvement are 95-97%.9,10 In 32% cases of ischemic lesions pupil involvement may be seen.11

Cavernous sinus (Intracavernous) - The nerve lie in the superolateral wall of the sinus along with the first two divisions of the trigeminal nerve. Here it divides into the superior and inferior divisions in the anterior part of the sinus or at the superior orbital fissure passing into the orbit through the annulus of Zinn. However, there is evidence that the functional bifurcation of third nerve occurs more proximally within the brainstem.12-14 Intracavernous involvement may be seen in cases of diabetes mellitus, pituitary apoplexy, aneurysmal dilatations, Tolosa hunt syndrome, meningiomas, etc.

Orbit (Intra-orbital) - Innervation of the superior rectus and levator palpebrae muscles is by the superior division and the inferior division innervates the medial rectus, inferior rectus, inferioroblique and provides parasympathetic fibres to the ciliary ganglion. These divisions then enters the orbital apex through the superior orbital fissure, where they travels with the optic nerve, ophthalmic artery, nerve VI, and the nasociliary branch of V1.15 Orbital lesions are usually associated with visual impairment, proptosis and chemosis. Causes mentioned in literature are trauma, neoplasm, vascular malformation and inflammation.

**Aberrant Regeneration**

This usually occurs in congenital third nerve palsy or following trauma, compression of the nerve by slow growing tumour (meningioma) or aneurysm. The breached myelin sheath and the perineurium cause misdirection of the regenerating axons and innervate the surrounding muscles such as the LPS, superior oblique and occasionally iris sphincter muscle. This results in clinical manifestations such as elevation of the eyelid during attempted adduction (Inverse Duane syndrome) or depression (Pseudo-Von Graefe’s sign) or miosis in an otherwise non-reactive pupil (Pseudo-Argyll-Robertson pupil). A patient with CN III dysfunction can present with doubling of vision, drooping of upper eyelid, ocular pain, headache, glare, monocular blurred vision, or any combination.16

When a patient presents with acute onset of limitation of movements in one eye, the categorization of the defect as complete or partial with or without the involvement of the pupil is useful in coming to a diagnosis and further evaluation. Isolated oculomotor nerve palsy is idiopathic in 25% of the cases and is commonly due to basilar lesions. Pupil-sparing third nerve paresis in an elderly patient with known systemic vascular disease can be considered to be ischemic mono neuropathy, which is a common cause. However the possibility of vascular inflammation such as giant cell arteritis should be kept in mind in elderly patient and may be excluded by history, complete hemogram, erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP). These patients can be managed conservatively and reassessed after a week to rule out pupil-involvement or increase in pain. Weekly follow-ups initially followed by monthly evaluation are important to document recovery and plan any intervention accordingly. Ischemic mono neuropathies usually start recovering by 8-12 weeks.

Acute palsy in individuals less than 40 years of age suggests need for neuroimaging and a complete neurological workup.

Common cause of isolated oculomotor nerve involvement is intracranial aneurysms. Therefore, its thorough evaluation is a must.17,18 If a patient presents with sudden onset of severe headache, meningismus and photophobia with oculomotor nerve involvement, a non-contrast head computed tomography (CT) scan should be performed to rule out spontaneous subarachnoid hemorrhage; if CT is non-diagnostic, further lumbar puncture should be performed.

Red flag signs such as no improvement within 3-4 weeks, pupillary involvement, associated headache and jaw claudication, history of trauma and features of raised intracranial pressure require neuro-imaging and blood investigations on an urgent basis.
The aneurysms in the posterior part of the Circle of Willis, posterior communicating and basilar tip aneurysms have the highest rates of ruptures between 2.5-50% depending upon their size. Thus, these aneurysms are the most commonly associated with isolated third nerve palsy. Trauma is also a common cause. However trivial trauma without loss of consciousness should suggest the possibility of pre-existent mass lesion at the base of skull. When diurnal variation or fluctuations in clinical findings are associated with a pupil-sparing palsy, myasthenia gravis should be excluded. Evaluation for tuberculosis and sarcoidosis would, at a minimum, require chest radiograph, skin testing for the former and angiotensin converting enzyme level for the latter. Signs of increased intracranial hypertension warrant neuroimaging to rule out space occupying lesions. Multiple sclerosis should also be considered in the differential diagnosis. Oculomotor nerve palsies in children are usually congenital or traumatic. Other causes include brainstem tumours, infarction, meningitis, post-infectious/post vaccination neuropathies and ophthalmoplegic migraine.

### Medical Management

Treatment initially involves medical management of systemic predisposing factors and conservative measures to obviate symptoms followed later by surgical intervention in non-resolving oculomotor nerve palsy. The patient should be evaluated at each follow up with a complete squint work-up, diplopia charting and Hess-charting. Ischemic neuropathies usually resolve in 6 weeks to 3 months and can be treated conservatively. Underlying systemic conditions should be adequately controlled and managed to prevent further ocular morbidity. Ptosis in a complete oculomotor palsy usually limits the diplopia. Occlusion of the normal eye during the period of observation if ptosis is not present eliminates the troublesome diplopia and past-pointing associated with acute palsies. Botulinum toxin injection to the only functioning rectus muscle, lateral rectus has been described to prevent contracture during the period of conservative management. The exotropia shows marked recovery early in the course. Though temporary it provides short term diplopia free vision. It could provide an alternative treatment modality in a patient who is unfit for neurosurgical intervention or strabismus surgery. Mono neuropathies with aneurysmal compressions or other compressive lesions require neurosurgical referral and intervention. An alert patient may notice anisocoria and increased light sensitivity resulting due to pupil involvement. Pilocarpine can be used if there are no other contraindications. Diminished accommodation, can also occur in pupil involving oculomotor palsy, and the clinician should consider near vision addition in optical prescriptions. In non-recovering or residual third nerve palsies surgical intervention may be considered in cases with stable deviation and minimum follow-up of 6-12 months.

### Surgical Management

The surgical correction of large angle exotropia and hypotropia in the oculomotor nerve palsy is a formidable challenge for a strabismologist. It varies according to the number of extraocular muscles involved, their recovery and the presence or absence of aberrant regeneration (Figure 1). The surgical procedure employed aims at achieving adequate alignment of the two eyes in the primary gaze. The goals and limitations of the surgery should be clearly understood by the patient to avoid disappointment. Several staged procedures are required to achieve an optimal correction, to assess the results prior to considering further options and to avoid anterior segment ischemia. Surgical outcome is often better and predictable in partial oculomotor nerve palsy. Paralysis of the upper division of the oculomotor nerve can be corrected by Knapp procedure (transposition of medial and lateral rectus adjacent to the insertion of the superior rectus) on the affected eye. In large angle hypotropia (>15Δ) the above can be combined with recession of the contralateral superior rectus muscle. Isolated palsy of the inferior division of the oculomotor nerve is rare. A procedure has been described by Kushner that involves simultaneous transposition of the superior rectus muscle to lie adjacent to the medial rectus and of the lateral rectus muscle toward the insertion of inferior rectus muscle, with tenotomy of the ipsilateral superior oblique. Isolated palsy of the medial rectus is even rarer. Lateral rectus disinsertion and periosteal fixation to the lateral wall with or without medial globe anchoring.

Complete oculomotor palsy is difficult to correct because of involvement of most of the extraocular muscles. Several surgical options are available with acceptable results.

1. **Large Recession and Resection** - A forced duction test for adduction should be performed by which the passive mobility of the globe is determined to rule out mechanical restriction of ocular motility. If significant medial rectus muscle function is present so as to cause adduction of the eye past midline, the eye may be aligned in the primary position with a supramaximal recession of the lateral rectus muscle and resection of the medial rectus muscle. It is usually combined with posterior tenectomy of the superior oblique (PTSO). Figure 2a and 2b, showing preoperative and postoperative pictures of patient of partial right III n palsy with aberrant regeneration with good MR function and lid retraction on adduction, in which recession and resection procedure was done in left eye.

2. **Globe anchoring Procedures** - In the absence of significant medial rectus muscle function, even supramaximal recession of the lateral rectus muscle and resection of the medial rectus muscle will almost always result in recurrence of the strabismus. In such a case, an active force generation test and saccadic velocities are useful in assessing the function of apparently paretic medial rectus muscle in the presence of contracture of the lateral rectus. When no or minimal force is generated, a globe fixation procedure to the
medial wall of the orbit at the anterior lacrimal crest can be performed. This can be achieved by using either 5-0 nonabsorbable polyester suture, fascia lata, orbital periosteal flap, superior oblique tendon or silicon bands through a skin incision or through a precaruncular approach. The anchoring of the globe to the nasal periosteum with the use of non-absorbable polyester suture was a new technique developed by Sharma P et al. Similarly, Saxena R et al evaluated the precaruncular approach for globe fixation in the largest case series described till then. The suture/T-plate anchoring platform system anchors the globe by sutures to a titanium T-plate screwed to the orbital wall, advantages being reduced risk of anterior segment ischemia and fewer re-operations, with likely longer durability of the system. Figure 3 showing preoperative and post operative pictures of a patient in which right eye medial anchor was done for congenital right third nerve palsy.

3. Superior oblique transposition procedures
Transposition of the superior oblique muscle, with or without trochleotomy, along with horizontal recti surgery, has been the mainstay of treatment in achieving ocular alignment in third nerve palsy. Scott described the disinsertion and shortening of the superior oblique tendon without trochleotomy and its insertion at the superior end of the medial rectus insertion to improve the alignment. However it is
complete of Bell's phenomenon and aberrant regeneration. Complete management of ptosis depends on the presence or absence of the lateral and superior recti have been reported by Maruo et al.\textsuperscript{46}

4. **Lateral Rectus Transposition Procedures** - Complete transposition of the lateral rectus muscle to the medial globe to facilitate the medial rotation of the eye up to the primary gaze was described by Taylor et al.\textsuperscript{47} Y-splitting of the lateral rectus and its transposition to retro equatorial points 20 mm posterior to the limbus near the nasal superior and inferior vortex veins\textsuperscript{48} or to points 1 mm posterior to the superior and inferior borders of the MR insertion without\textsuperscript{49} or with posterior fixation sutures\textsuperscript{50} have been described. The latter procedure described by Saxena R et al modified the technique described by Gokyigit B et al by placing non-absorbable sutures to fix each split belly of the transposed lateral rectus muscle to the sclera at the equator adjacent to the medial rectus (Figure 4a and 4b). They postulated that the posterior fixation sutures augment the force of the transposed muscles by redirecting the force vectors in the direction of action of the medial rectus resulting in minimum residual exotropia.

Management of ptosis depends on the presence or absence of Bell’s phenomenon and aberrant regeneration. Complete ptosis with poor Bell’s phenomenon can be managed with crutch glasses. Frontalis sling procedures for the ptosis can be done, however keeping in mind that over-correction can lead to corneal exposure. Levator resection may be inadequate as the levator muscle is paralysed to some extent and may show variable response to the procedure. In patients with aberrant regeneration, ptosis can be corrected by performing a recession-resection procedure in the contralateral eye. Similarly, in a case of partially recovered oculomotor nerve palsy with residual superior rectus function, the ptosis can be corrected using the concept of ‘Fixation duress’ in the non-involved eye. The fixation duress is proposed to decrease the elevation of the normal eye thereby creating similar forces of duress in both eyes when fixation takes place.

**Conclusion**

To summarize, correct diagnosis after accurately localizing the causative lesion, is very essential, to avoid life threatening situations. Knowledge of associated syndromes based on different location can be helpful in management. Furthermore, strabismus surgery can correct residual non-recovered part of third nerve palsy and helps in improving the patient’s quality of life.

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