Third Cranial Nerve

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Abstract- *Third cranial nerve also known as Oculomotor Nerve. It supplies major part of human eye. It has parasympathetic and motor supply. This article looks at the anatomical aspect, functions and clinical correlation of nerve.*

Keywords- Eye, Oculomotor nerve, Parasympathetic, Palsy.

I. INTRODUCTION

Primary efferent nerve fibers:Nucleus lie at of superior colliculus in mid brain that is part of brainstem.It exits ventrally and enters subarachnoid space and courses forward and laterally between posterior cerebral artery and superior cerebellar artery. It then runs alongside posterior communicating artery. Then into the cavernous sinus where it splits into superior and inferior branches. It enters the orbit through the superior orbital fissure inside annulus of zinn.
Secondary efferent nerve fibres come from accessory nucleus of the oculomotor nerve also known as the Edinger – Westphal nucleus which is also situated in midbrain.

II. APPLIED ANATOMY

NUCLEAR PORTION Nuclei lie centrally, close to midline.Pretectal nuclei lie dorsally and receive innervation from the optic tracts as part of pupil light reflex. Most rostral and dorsal nuclei are visceral nuclei that give parasympathetic innervations to sphincter pupillae and ciliary muscles

Most Caudal and midline is Levator sub nucleus which is unpaired and supply both levator muscles. Lesion at this level gives rise to bilateral ptosis. Superior rectus sub nucleus is paired and supply contralateral superior rectus. Lesion lead to contralateral elevation deficit. Inferior rectus, medial rectus and inferior oblique sub nuclei are paired and supply respective muscle of same side. These then pass fibres bilaterally to the Edinger westphal nucleus.

FASICULAR PART these fibres pass ventrally through red nucleus and cerebral peduncle and form Fascicular portion of this nerve

SUBARACHNOID PART The cisternal portion of the nerve courses in the subarachnoid space anterior to the midbrain after exiting between the superior cerebellar artery and posterior cerebral artery and running in close proximity to the posterior communicating artery.Parasympthetic fibres run superficially within the nerve and are typically spared by micro vascular ischaemia.Circle of Willis lies immediately besides the subarachnoid nerve which is supplied by single basilar artery and two internal carotid arteries. The major branches of these are the anterior cerebral, middle cerebral, and posterior cerebral arteries. These are connected into a circle by single anterior communicating artery (site of berry aneurysm), and two posterior communicating arteries.

CAVERNOUS SINUS PORTION In cavernous sinus occulomotor nerve sits against the lateral wall along with 4th nerve and upper two branches of trigeminal nerve.

ORBITAL PORTION The Oculomotor nerve enters the orbit through the superior orbital fissure adjacent to the fourth cranial nerve. Superior division supplies Levator Palpabre Superioris and Superior Rectus while inferior division supplies Medial Rectus, Inferior Rectus, and Inferior oblique. Secondary fibres branch off at the level of ciliary ganglion and synapse there. They then continue to innervate the ciliary body, the iris and sphincter pupillae muscle and dilator muscle through nerve to inferior oblique. Nerve to Inferior Oblique carries preganglionic parasympathetic fibres to ciliary ganglion

III. THIRD NERVE PALSY

CLINICAL MANIFESTATIONS

General features — Patients with an acute acquired third nerve palsy usually complain of the sudden onset of binocular horizontal, vertical, or oblique diplopia and a droopy eyelid

Nuclear lesion –complete ipsilateral IIIrd nerve palsy plus contralateral ptosis and SR dysfunction.[1]

Follow Darroff's rules

Conditions that obligate nuclear involvement

- -Bilateral IIIrd nerve palsy without ptosis (bilaterally spared LPS function)

- -Unilateral IIIrd nerve palsy with contralateral superior rectus weakness and bilateral partial ptosis
- Conditions that exclude a nuclear lesion
- Unilateral ptosis Unilateral internal ophthalmoplegia
- Unilateral external ophthalmoplegia associated with normal contralateral superior rectus function

Conditions that neither exclude nor obligate a nuclear lesion

- Bilateral total IIIrd nerve palsy
- Bilateral ptosis Bilateral internal ophthalmoplegia
- Bilateral medial rectus palsy in nuclear lesions, the clinical picture is that of complete ipsilateral IIIrd nerve palsy plus contralateral ptosis and SR dysfunction.

These tend to be small lesions with few associated neurologic symptoms or signs. Rostral lesions lead to pupillary involvement and muscles are usually spared. Caudal midline lesions give rise to bilateral ptosis. The most common cause of nuclear complex involvement is infarction secondary to the occlusion of small perforating twigs from basilar arteries or occlusive disease of basilar artery. Intraparenchymal haemorrhage and metastasis are other significant causes.

Fascicular lesions. Because of the proximity of the fascicular portion of the nerve to other structures in the midbrain, lesions typically produce neurologic symptoms associated with the damaged structures. Several syndromes have been recognized and are described below

- Weber's syndrome: IIIrd nerve palsy + contralateral hemiparesis. Site of lesion is cerebral peduncle.
- Benedikt's syndrome: IIIrd nerve palsy + contralateral tremors. Site of involvement is red nucleus.
- Nothnagel's syndrome: IIIrd palsy + ipsilateral ataxia. Site of lesion is superior cerebellar peduncle.
- Claude's syndrome is a combination of Benedikt's and Nothnagel's syndrome.
- Causes of fascicular lesion include vascular, inflammatory, infiltrative causes and demyelination disorders

- **Subarachnoid space** is the most likely site of involvement in isolated IIIrd nerve palsies. Aneurysm is the most common lesion in subarachnoid space. The signs and symptoms of subarachnoid haemorrhage include sudden severe headache, stiff neck, and loss of consciousness. It may be due to compression or infarction secondary to micro vascular involvement in diabetics and hypertensive. Because of the dorsal and peripheral position of pupillary fibres, a dilated pupil may be the first sign of nerve compression[2]. Aneurysm is usually situated at the junction of posterior communicating artery and internal carotid artery. Diabetic micro vascular IIIrd nerve palsies are commonly painful and usually pupil-sparing. Other

Causes include infiltrating or compressive neoplasms, meningitis, compression by dolichoectatic vessels, trauma or midline shift by expanding supratentorial lesion or edema.

Cavernous sinus lesions are usually painful .Usually associated with the dysfunction of IVth and VIth nerves, first or second division of trigeminal nerve. Affection in anterior part of the cavernous sinus may lead to selective involvement of superior or inferior division. The pupil may be small sized due to involvement of oculosympathetics. Causes include neoplasms (pituitary tumours, craniopharyngioma, meningioma, nasopharyngeal carcinoma, and metastasis), Tolosa-Hunt syndrome, sarcoidosis, aneurysmal compression, ischemia, cavernous sinus thrombosis and arteriovenous fistula.

Ophthalmoplegic migraine presents in childhood with recurring bouts of unilateral headache and ipsilateral third cranial nerve palsy that can last several weeks at a time. Pupillary involvement with a 'full blown pupil' is a rule in children; however, pupil-sparing may be observed in adults. Rarely associated with 5^{th} and 6^{th} nerve palsy. Resolution of ophthalmoplegia is a rule .The link to migraine in general has come under question because many or most of these patients demonstrate thickening and enhancement of the cisternal portion of the third cranial nerve on MRI with gadolinium.

IV. DIFFERENTIAL DIAGNOSIS

— several conditions can mimic the extra ocular dysfunction of third nerve palsy.

- isolated mydriasis-Tonic pupil, iris sphincter damage, and pharmacologic mydriasis should be specifically excluded by careful neurophthalmologic evaluation and pharmacologic testing in this setting
- Muscle paralysis-Orbital disease, e.g., orbital fracture, tumour, and inflammation, can affect the extra ocular muscles directly.
- Myasthenia gravis can mimic any painless, pupil-sparing ophthalmoplegia and should be considered in

every patient with pupil-sparing third nerve palsy. Other signs of myasthenia gravis, such as ptosis, variability, and fatigue, usually are present.

- Skew deviation is a vertical misalignment caused by disruption of the vestibulocular connections and can result in hyperopia. Usually it is associated with other posterior fossa signs (e.g., other cranial neuropathies

V. EVALUATION

— The evaluation of the patient with a third nerve palsy depends on associated symptoms and signs and the pattern of oculomotor nerve involvement, both of which help to localize the lesion anatomically. The age of the patient and other historical features also help to distinguish congenital, traumatic, or infectious conditions. Inflammatory and neoplastic conditions have a more insidious onset compared with cerebral infarction.

Non isolated third nerve palsy — Third nerve palsies that are accompanied by other neurologic deficits, orbital signs, or meningismus require a neuroimaging. A lumbar puncture (LP) may also be required .A brain magnetic resonance imaging study (MRI) is indicated for patients with findings suggesting a lesion of the nerve's nuclear or fascicular course within the brainstem. These findings include bilateral ptosis, contralateral superior rectus palsy, hemiparesis, ataxia, and tremors.

The emergent work-up should include head computed tomography (CT) without contrast looking for blood in the subarachnoid space. Signs that localize to the cavernous sinus or orbital apex indicate a brain MRI with gadolinium. The table below shows the need for urgency of neuroimaging in acute onset third nerve palsy.SAH, subarachnoid haemorrhage.

Urgent (immediate)	Routine (next working day)
	Age >55
	Diabetes
	Hypertension
Pupil Involved (compression)	
Reduced Consciousness (SAH)	Headache, but no neck stiffness or drowsiness
Neck stiffness (SAH)	Isolated 3rd nerve (or isolated 4th or 6th nerve palsy)
Photophobia (SAH)	intermittent or longstanding
Age <55 (unless diabetes and/or hypertension)	previous squint surgery
Other cranial nerves affected	

VI. TREATMENT AND PROGNOSIS

— Therapy is directed at the underlying aetiology of the third nerve palsy.

In most cases, third nerve deficits, diplopia, and ptosis recover over weeks to months.

Intervention for aneurysmal third nerve palsy is primarily directed at preventing subarachnoid haemorrhage which includes either neurosurgical clipping or endovascular embolization. The majority of ischemic third nerve palsies, including those related to migraine headaches usually improves over three to six months. Vascular risk factors should be treated; antiplatelet therapy is usually provided. Traumatic third nerve palsies, particularly those who have partial palsies, may also experience spontaneous resolution

Persistent deficits — patching one eye is useful in alleviating diplopia, particularly in the short term. Prism therapy may be employed for small, comitant, long-standing deviations. A temporary press-on (Fresnel) prism of sufficient power to align the eyes is placed on the spectacle lens of the affected child. Permanent prisms can be ground into the spectacle lens if the patient has a stable but symptomatic deviation alleviated with prism.

Strabismus surgery may be helpful in patients who fail prism therapy. However, this surgery is difficult to perform, particularly in those with complete third nerve palsies, because multiple muscles are involved; ocular alignment in primary position might be achievable but might require multiple procedures. These include transposition of Superior oblique muscles anterior and medial to Superior Rectus (Scott's procedure), large Lateral Rectus recessions and occasionally recession-resection of horizontal recti muscles. The role of botulinum toxin is is still not defined[4].

REFRENCES

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