

A CLINICAL STUDY OF OCULAR MOTOR NERVE PALSIES

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This is submitted to The Tamil Nadu Dr. M.G.R. Medical University, Chennai, in partial fulfillment of the requirement for the award of M.S., (Ophthalmology) Branch – III Degree Examination to be held in March 2010.

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A CLINICAL STUDY OF OCULAR MOTOR NERVE PALSIES

INTRODUCTION

The designation “Ocular motor system” refers to the entire somatic motor system that controls the position and movements of the eyes. This system includes the extraocular muscles, the cranial nerves and nuclei that innervate them and the forces that stimulate and inhibit their actions. Restricted Ocular mobility could be the result of paralysis of the nerves supplying the extra ocular muscles (neurogenic) or due to the pathology in the muscle itself (myogenic) or there may be the pathology at the myoneural junction (neuro muscular).

Since the treatment for these conditions is different from one another, it becomes clinically important to differentiate them, thus avoiding elaborate neurological investigations it is not necessary.

Movements of the eye ball is brought about by six extraocular muscles – the four recti and the two obliques. The cranial nerves supplying the extra ocular muscles are - the Oculomotor nerve (third), the Trochlear nerve (fourth) and the Abducens nerve (sixth). The Abducens nerve supplies the lateral rectus muscle and the trochlear supplies the Superior oblique and the third nerve supplies the remaining muscles along with levator palpebrae superioris and the intrinsic muscles of the eye.

Palsy is the term used to denote either paresis (partial) or Paralysis (total) of that particular nerve. These nerve palsies could be either Congenital or acquired. Most congenital palsies occur as isolated defects in an otherwise healthy individual,

presumed to be due to developmental defects in the nucleus or the nerve pathway. Acquired palsies are more common and the etiology varies. Ocular motor system involvement can be divided anatomically and physiologically into infranuclear or peripheral (commonest) nuclear, internuclear (MLF) and supranuclear (brainstem, cerebral, vestibular and cerebellar) components.

ANATOMY

OCULOMOTOR NERVE OR THE III NERVE

The oculomotor nucleus is located in the periaqueductal gray matter inferior to the sylvian duct at the level of the superior colliculus. It consists of 2 main somatic lateral nuclei source of somatic fibres to the ocular muscles and the forked anterior group of small cells- Edinger-Westphal nucleus supplying parasympathetic innervation to the eye. A single caudal midline group of cells innervates both levator muscles. The medial rectus, inferior rectus and inferior oblique muscles receive their innervation from the appropriate ipsilateral subnucleus while superior rectus receives innervation from the contralateral superior rectus subnucleus.

The fascicular portion, starting from the ventral side of the nucleus runs through the parenchyma of the midbrain and pass laterally through the red nucleus to exit above the medial aspect of the cerebellar peduncles into the interpeduncular fossa.

The basilar portion of the nerve leaves the midbrain as 15,000 fibers at the sulcus oculomotorius between 2 cerebral peduncles and then coalesce to form the main trunk. It passes through the subarachnoid space, pierces the durometer lateral to the posterior clinoid process and enters the cavernous sinus. Within the subarachnoid space, the nerve passes between the superior cerebellar artery and the posterior

cerebellar artery and is located parallel and lateral to the posterior communicating artery.

The cavernous part in the lateral wall of the cavernous sinus superior to the other nerves and divides into the superior and inferior branch in the anterior part.

The nerve enters the orbit through the superior orbital fissure. The superior division innervates the levator and superior rectus muscles. The larger inferior division innervates the medial rectus, inferior rectus and inferior oblique muscles and parasympathetic fibres to sphincter pupillae and ciliary muscle through the ciliary and episcleral ganglion.

The location of the parasympathetic, pupillomotor axons within the third nerve is important. The pupillomotor fibres are peripheral and located in the superomedial part of the nerve as it leaves the brain stem. Within the cavernous sinus the pupillomotor fibres which are located centrally descend through the substance of the oculomotor nerve in the middle third of cavernous sinus. As the nerve enters the orbit, they are located in the inferior peripheral portion. The pupillomotor fibres are smaller and more thinly myelinated than the somatic fibres of the oculomotor nerve.

THE FOURTH NERVE OR TROCHLEAR NERVE

The fourth nerve nucleus is located in the periaqueductal gray matter ventral to the sylvian aqueduct at the level of inferior colliculus, caudal to the oculomotor nucleus. Each nerve innervates contralateral superior oblique muscle. The fasciculus leaves the nucleus laterally and curves in a dorsal and caudal direction to decussate completely in the anterior medullary velum. The nerve leaves the brain stem just below the inferior colliculus on the dorsal surface.

The subarachnoid portion of the nerve (containing approximately 2000 fibers) curves forward around the brainstem, runs beneath the tentorial free edge and passes between the posterior cerebral and the superior cerebellar arteries. It passes in a rostral direction, pierces the dura and enters the cavernous sinus, lateral and inferior to the third nerve. In the anterior portion of the cavernous sinus the fourth nerve rises and enters the orbit through the superior orbital fissure above the annulus of Zinn to innervate the superior oblique muscle.

ABDUCENS OR SIXTH CRANIAL NERVE

The nucleus of VI nerve (Abducens nerve), is located in the pons inferior to the floor of IV ventricle and slightly lateral to the midline anterior to the pontomedullary junction. Adjacent and medial to each nucleus is the PPRF. The MLF is located superior medial to the VI nerve nucleus the nucleus is closely related to the fasciculus of VII nerve – the facial colliculus. The nerve (approximately 4000 – 6000 fibers) emerges ventrally at the pontomedullary junction just lateral to the pyramidal prominence and enters the prepontine basilar cistern. The nerve which has the longest subarachnoid course passes upward along the base of pons on either side of basilar artery above the clivus. It lies adjacent to or passes through Dorello's canal formed by the petroclinoid ligament (Gruber's ligament) within the canal, VI nerve travels with the inferior petrosal sinus and enters the cavernous sinus by perforating the dura matter lateral and inferior to the III and IV nerves. It is the most medial cranial nerve in the cavernous sinus and is closest to the carotid artery. The nerve enters the orbit through the superior orbital fissure through the annulus of Zinn and terminates at the lateral rectus muscle.

OCULAR MOTOR NERVE PALSIES

Oculomotor system can be anatomically and physiologically divided into

- Supranuclear component (brain stem, cerebral, ventricular and cerebellar lesions).
- Nuclear and internuclear lesions.
- Intranuclear component or peripheral component:

Commonest and is the bulk of the cases under study. Lesions of the infranuclear oculomotor system may affect the III, IV & VI nerve at any location from their fascicular portion to the termination in the extraocular muscles. The lesion may affect two or more of the nerve in combination in one or both eyes and may present as follows:

TOTAL OPHTHALMOPLEGIA

It is the paralysis of all the EOM and LPs and the intraocular muscles (Ciliary muscle and pupillary muscle). This is characterized by complete ptosis, slight proptosis of the eyeball, in a position of slight divergence with a large dilated pupil and absence of accommodation.

Causes

1. Wide spread inflammation or vascular lesions of brainstem – usually bilateral.
2. Extramedullary course, the cavernous sinus or superior orbital fissure or orbit – unilateral.

External Ophthalmoplegia

Paralysis of EOM & LPS where pupillary activity and accommodation are spared.

Internal Ophthalmoplegia

Palsy of only internal muscles (Ciliary and Pupillary muscles).

Partial Ophthalmoplegia

Palsy of EOM where one or more of the external muscles have escaped. Usually orbital causes and unilateral.

Ocular Motor Palsies

May present in one of the 4 ways.

1. Isolated partial or complete nerve palsies without any other neurologic signs and without other symptoms.
2. In associated with symptoms (Pain, dysesthesia, paraesthesia etc.) but without any signs of neurologic or systemic disease.
3. In association with other ocular motor nerve palsies but without any other neurologic signs.
4. In association with neurologic signs.

CONGENITAL VS ACQUIRED PALSIES

Importance

1. Cause must be found and treated in acquired palsies.
2. No further investigation is needed if congenital.
3. Immediate treatment in congenital, at least 6 months allowed for spontaneous recovery if acquired.

CHARACTERISTICS OF MUSCLE PARALYSIS

Objective signs of nerve palsies are (i). Abnormal deviation of eyes.
(ii). Compensatory head posture (iii) Limitation of Movements.

Subjective symptoms are (i). Diplopia (ii). False orientation (iii). Ocular vertigo.

1. Compensatory head posture

In general, any ocular motor nerve palsy will result in limitation of ocular movement leading to incomitant or paralytic strabismus causing diplopia. The direction of deviation depends on the nerve affected. Paralytic strabismus increases as the eyes are turned in the direction of limitation of movement. This leads to compensatory head posture comprising of face turn, head tilt and altered chin position mainly to alleviate diplopia. Usually symptom free in children with congenital palsies.

Muscle Sequelae

Determined by Hering's and Sherrington's laws. They are (1) Underaction of the affected muscle (2) Overaction of the contralateral synergist (Hering's law) (3) Contracture of ipsilateral antagonist (Sherrington's law) 4. Secondary inhibition of the contralateral antagonist (Hering's law).

These developments depend upon the duration of muscle palsy, degree of limitation of ocular movement and the eye which is fixing. Because of these muscle sequelae, the secondary deviation (the angle measured with the affected eye fixing) exceeds the primary deviation (the angle measured with the unaffected eye fixing).

CLINICAL EVALUATION OF A CASE WITH OCULAR MOTOR NERVE PALSIES

After careful history including the medical and neurological conditions, the patient should be examined in general to rule out vascular diseases like diabetes, hypertension and arteriosclerosis. If there is history of cerebrovascular accident, a total neurological examination is needed to find out the associated neurological signs.

Diplopia

If there is horizontal diplopia – Horizontal muscle palsy. As isolated medial rectus palsy is rare, it is most likely due to lateral rectus palsy. Vertical diplopia especially with a tilt, most likely to be superior oblique palsy.

Cover Test

One should look for the type of deviation comparing the amount of movement for near and distance.

Ocular Movements

Both underaction and overaction are to be noted by doing alternate cover test in different positions of gaze. Maximum deviation is noted in the direction of the action of affected muscle. The examiner should remember that both the eyes may be affected, particularly after severe head injury. Other signs like nystagmus especially in certain positions, or retraction of globe on horizontal or vertical gaze can occur in case of direct injury to muscles or their connection.

Diplopia Chart

1. Maximum separation of images in the direction of action of the affected muscle.
2. Abductors – lateral rectus, superior and inferior obliques produce uncrossed diplopia while others produce crossed diplopia.
3. Tilt though produced in vertical palsies is more common with superior oblique palsies.

Hess Chart

Not only diagnosis but also for follow up of a case of muscle palsy. Points to be remembered are

1. The smaller field always belongs to the paretic eye-inward displacement of dots indicates underaction of the paretic muscles and outward displacement indicate overaction of antagonist. Equal sized fields indicate muscle sequelae have developed denoting either congenital or longstanding palsy.
2. The outer field should be examined for small underaction and overaction which may not be apparent on inner fields. If the outer field is very close to the inner field, a mechanical restriction of muscle movement rather than a neurogenic one is suspected.

Forced duction test and its applications

It is useful to differentiate a neurogenic palsy from restrictive palsy which may be due to contraction or fibrosis of a muscle, tightness of a muscle following excessive resection, scarring of conjunctiva, trapping of muscle fibres as in blow out fractures, symblepharon etc.

After application of local anaesthetic, the eye is grasped at the limbus with a forceps and is moved in the direction opposite to that in which mechanical restriction is suspected, taking care not to press the globe. When the test is done patient is asked to look at this hand which is held the direction in which the eye is moved. This is to prevent the influence of the patient's innervation which may otherwise counteract the passive movement of the globe simulating a mechanical restriction.

Bielschowsky Head tilt test

To find out the vertical muscle involved, especially in cases of superior oblique palsies where on tilting, the eye becomes hypertropic on the affected side.

THIRD NERVE PALSY

Clinical Signs

In complete palsy, the following signs will be present

- a. Ptosis of the affected side.
- b. Eye divergent, may be slightly depressed.
- c. Ocular movements – Limitation of adduction, elevation and depression especially on abduction will be present.
- d. If ptosis is complete, there is no diplopia and hence there may not be any abnormal head posture.

- e. Pupil may be dilated or normal. It is very important to note pupillary reaction since it may give a clue about both etiological and anatomical location of lesion. This is very important since pupillary involvement is one of the sign of aneurysm which is very dangerous. In longstanding palsies, signs of aberrant regeneration have to be noted.

Superior division palsy

There will be underaction of superior rectus along with partial ptosis and this is mostly congenital. The affected eye is hypotropic with absence of elevation. The chin is elevated.

The muscle sequelae will be overreaction of contralateral inferior oblique, contraction of ipsilateral inferior rectus and secondary inhibition palsy of other eye superior oblique.

Differential diagnosis of the condition are:

1. Double elevator palsy: Usually congenital and nuclear.
2. Blowout fracture of orbit: With typical findings and history.
3. Thyroid myopathy: Especially of inferior rectus muscle.
4. Paralysis of vertical gaze (Parinaud's syndrome).

Inferior division palsy

Very rare. As it contains the pupillary fibres, pupillary dilatation along with underaction of medial rectus, inferior rectus and inferior oblique muscles. As a result, the eye will be exotropic, intorted and hypertropic; face will be turned to the affected side and head tilted to the side of hypotropic eye.

Isolated single muscle involvements are very rare and mostly myogenic. But if there is medial rectus involvement an internuclear ophthalmoplegia is to be ruled out.

TROCHLEAR NERVE PALSY

It supplies the superior obliques muscle. Commonest cause of palsy is trauma followed by ischemia.

It can be congenital or acquired. Being the longest and slenderest nerve emerging from dorsal aspect of midbrain it is more vulnerable to head trauma can be bilateral in severe head injury. Rarely involved in ischemic injury due to diabetes. Commonest symptom will be diplopia on looking down especially while climbing down the stair case.

Clinical Signs

Compensatory head posture

The chin is depressed and head tilted to the unaffected side. This position moves the eyes away from field of action of the superior oblique to overcome diplopia.

Ocular Signs

The affected eye will be hypertropic & slightly convergent. Hypertropia will be more for near.

Muscle Sequelae

There will be underaction of superior oblique, that is depression in adduction with overaction of inferior rectus of the opposite eye and the inferior oblique on the

same side. Diagnosis is by Bielschowsky head tilt test in which the hypertropia is greatest on looking to the affected side.

Diplopia Chart

Shows uncrossed diplopia with maximum separation of images on **levodepression** in case of right superior oblique palsy.

Hess Chart

According to muscle sequelae. Mostly found in outer segment if minimal.

The binocular field

Will be displaced upwards to the affected side.

SIXTH NERVE OR ABDUCENS NERVE PALSY

The sixth cranial nerve supplies the lateral rectus muscle and its palsy results in lateral rectus weakness. Though it may be different causes the most common cause is only idiopathic neuritis.

Clinical Signs

Esotropia which is greater for distance than for near.

Compensatory head posture

Face turn to the affected side in most cases. Sometimes, only for distant fixation.

Muscle Sequelae

Overaction of the contralateral medial rectus, contracture of ipsilateral medial rectus and secondary inhibitional palsy of contralateral lateral rectus.

Diplopia Chart

Uncrossed diplopia with maximum separation of images towards the affected side.

Hess Chart

Inward displacement of the field in lateral rectus area while outward displacement of field in medial rectus area in other eye. Later on according to other muscle sequelae.

Field of BSV

Displaced towards the unaffected side.

EVALUATION OF NERVE PALSY

It forms one of the false localizing signs along with bilateral papilledema in cases of increased ICT. As the horizontal deviation can be overcome better than vertical it is usually evident in distant gaze. It is the only thing that is allowed apart from papilledema in cases of pseudo tumor cerebri. Anatomic localization of lesion in an isolated VI nerve palsy is difficult sometimes and may require CT & MRI in some cases. Tumors are more common in children if VI nerve palsy is not following post viral or post vaccination in children. Apart from routine CBC, ESR, collagen vascular studies, tests to rule out diabetes or HT, and thyroid (VI is very rarely involved in thyroid ophthalmopathy) and may even sometime require a Tensilon test

to clinch the diagnosis. CT and MRI are indicated if there is associated ICT and if there is no improvement even after 3 months of onset.

ETIOLOGY &PATHOGENESIS

The sites of the lesions of various etiology may be in the nucleus, fascicle, subarachnoid space, cavernous sinus or orbit.

OCULOMOTOR OR THIRD NERVE PALSIES

The oculomotor palsies are either of congenital or acquired type.

Congenital

It is a rare, often unilateral palsy without any neurologic or systemic abnormalities generally. Usually due to perinatal trauma or hypoplasia of the nucleus or muscles. Pupillary involvement is variable, aberrant regeneration is common and divisional palsies may occur. Though rare it accounts for almost 50% of III nerve palsies in children. Slowly progressive oculomotor palsy is an indication for repeat MRI in spite of previous negative studies, as neurilemmoma can remain cryptic.

Lesions of Oculomotor Nucleus

Nuclear lesion of III nerve is rare. Any nuclear lesion is characterized by irregularity, bilaterality, loss of parallelism and presence of diplopia.

Causes

Vascular, neoplastic and lesser number of demyelinating toxic and inflammatory lesions are the most common causes of nuclear III nerve dysfunction.

Pupillary involvement in a nuclear lesion is variable. Based on these features, Daroff has formulated certain rules.

Obligatory nuclear lesions

- a) Ipsilateral III nerve without contralateral superior rectus palsies with bilateral partial ptosis.
- b) Bilateral III nerve palsy (without or with internal ophthalmoplegia) associated with spared levator function.

1. Conditions which may be nuclear

- a) Bilateral total III nerve palsy.
- b) Bilateral ptosis.
- c) Bilateral internal ophthalmoplegia.
- d) Bilateral Medial rectus palsy.
- e) Isolated single muscle involvement (except LPS & SR).

2. Conditions that cannot represent nuclear lesions

- a) Unilateral external ophthalmoplegia associated with normal contralateral superior rectus function.
- b) Unilateral internal ophthalmoplegia.
- c) Unilateral ptosis.

Lesions of the Oculomotor Nerve Fasciculus

Any fascicular lesion may produce an ipsilateral palsy which may be total and often associated with pupillary involvement and cannot be differentiated from a palsy

caused by lesions outside brainstem but the topical diagnosis made by the coexistence of other neurologic signs which may present as any of the following syndrome.

Causes

Intraparenchymal, vascular, neoplastic and demyelinating lesions

Syndrome	Principle lesions	Signs	Site of Lesion
Nothnagel's	Tumor of pineal body	Ipsilateral III N palsy contralateral ataxia	Superior cerebellar peduncle
Benedict's	Bony tumors vascular lesions solitary tubercles Hemorrhages	Ipsilateral III N Palsy contralateral hemiathetosis	Red nucleus and tegmentum of midbrain.
Claude's	Thrombosis of the medial interpeduncular branch of the posterior cerebral artery	Ipsilateral III N Palsy contralateral ataxia and asynergia	Red nucleus and brachium conjunctivum.
Weber's	Occlusion of PCA gumma TB, tumor	Ipsilateral III N palsy contralateral paresis	Cerebral peduncle

Lesions of the oculomotor IV in the subarachnoid space

The lesions in the interpeduncular fossa may be located anywhere from its emergence from the brainstem to the point where the nerve penetrates the dura beside the posterior clinoid process to enter the cavernous sinus. It may present in 3 ways.

1. Ipsilateral pupillary dilation as a sole manifestation

Any compression of the oculomotor nerve from above and medial may produce only pupillary dilation. May progress to total III nerve palsy and hence to be followed closely. Most important lesions included – Aneurysms at junction of basilar and superior cerebellar artery and at head of basilar artery, tumors & Basal meningitis.

2. Oculomotor nerve palsy with pupillary involvement

Commonest form of presentation of III nerve paralysis. It may present as isolated palsy without any neurological signs.

The basal type of III nerve palsy may be

- a. Isolated III nerve palsy without associated symptoms.
- b. Associated with syndrome involving other nerves.
- c. Syndrome of interpeduncular space – Bilateral III nerve palsies with bilateral hemiplegia.

Causes

Vascular – Intracranial aneurysm arising at the junction of ICA and PCA is the commonest or aneurysm of basilar artery by direct compression or during rupture.

Trauma – Either severe head injury or during surgery, tumour and other compressive

lesions. Infections – Basal meningitis usually in association with other cranial nerves including V, VI & VII nerves. Diabetes Mellitus – very rarely cause pupillary involvement and hence in every case of pupillary involvement an MRI or arteriography is indicated to the treatment of potentially dangerous cause of aneurysm.

Kernohan Notch Syndrome

When there is a supratentorial pressure from a tumor or subdural hematoma, compressing III nerve as it crosses the tentorial edge, the pupillary fibres which are in the peripheral superior medial part of the nerve are affected first without significant involvement of other parts of III nerve. This is a neurological emergency and depending on this ipsilateral pupillary dilatation with or without contralateral hemiplegia (due to involvement of cerebral peduncle) the site of burhole to be put is decided in neurological emergencies. Also known as acute or progressive clivus ridge syndrome.

Isolated III Nerve Palsy with Pupil Sparing

Ischemic form the frequent cause of this type of palsy which includes – diabetes mellitus, systemic HT< atherosclerosis, migraine, temporal giant cell arteritis and SLE. Often associated with severe pain, presumably there is a central ischemic infarct that spares the more peripherally placed parasympathetic pupillary fibres, 40% of diabetic III nerve may involve the pupil. However, comprehensive lesions can cause III nerves palsy without pupillary involvement due to following reasons.

1. They grow so slowly that the pressure is evenly distributed sparing the relatively pressure resistant pupillary fibres.

2. Compression from below sparing the dorsally located pupillary fibres.

Rule of thumb is that an isolated oculomotor palsy with pupil sparing is diabetes mellitus and that an isolated oculomotor palsy with pupil involved is aneurysm at the junction of ICA and PCA. However, there are exceptions. In general if the patient is over 50 and has complete oculomotor nerve palsy and a totally normal pupil arteriography as a routine is not recommended. If the III nerve palsy is incomplete, with or without pupillary involvement and with aberrant regeneration arteriogram is to be done without fail.

Lesions of oculomotor nerve within the cavernous sinus

Usually it will be a polyneuropathy than an isolated involvement. Further divided into anterior cavernous sinus syndrome when V1 of V alone is involved and posterior cavernous sinus syndrome when both first and second divisions of V nerve are involved.

It is characterized by

Partial ophthalmoplegia involving both III, IV & V nerves along with first or first and II division of V nerve.

Oculosympathetic paresis with small, poorly reacting pupil, which is pathognomonic or the pupil is usually spared.

May be associated with proptosis, edema of lids and congestion of conjunctivae.

Commonly leading to aberrant regeneration of III nerve.

May be associated with involvement of Optic nerve in anterior lesions.

Major etiologic lesions are

- Infections : Specific granulomatous infections like tuberculosis, syphilis, etc – Basal Meningitis.
- Inflammatory : Giant cell arteritis, rheumatoid arthritis, SLE, Herpes Zoster, cavernous sinus thrombosis.
- Vascular : caroticocavernous fistula, Intracavernous aneurysm of internal carotid artery, arteriovenous fistulae either spontaneous or traumatic.
- Neoplastic : Metastatic tumors – common
- Intrinsic tumors – multiple myeloma, extension from nasopharyngeal carcinoma.
- Extrinsic tumors – Pituitary adenoma, craniopharyngioma, supra and parasellar meningioma.
- Miscellaneous : Following the irritation of pituitary for extensive PDR, persistent primitive trigeminal artery.

Lesions of oculomotor nerve in the superior orbital fissure

This is always associated with IV and VI nerve involvement with or without II nerve involvement along with I division of V nerve. If it is associated with decreased vision due to II nerve involvement along with light proptosis it is called apical syndrome described later in painful ophthalmoplegia.

Pseudoorbital apex syndrome

Results when large inter cranial masses like giant aneurysm, compressing the cavernous sinus and optic nerve to impede drainage from the orbit. The oculomotor palsy is incomplete involving either divisions of the nerve.

Tolosa-Hunt syndrome

Idiopathic granulomatous inflammation of SOF leading to painful ophthalmoplegia which will be described later.

Lesions of III nerve in orbit

As the nerve has divided into 2 divisions, partial III nerve palsy is frequent and in case of superior division involvement, it has to be differentiated from a nuclear lesion.

Causes

Orbital tumors, contiguous sinus disease such as mucocele, tumor or infection. Infection may not be obvious or orbital trauma and orbital fracture.

Ophthalmoplegic Migraine

Differentiating features from aneurysms are (1) Transient nature of ophthalmoplegia. (2) Recurrent rapidly clearing palsy (3) Frequent personal or family H/o migraine.

Recovery from III nerve Palsy

4 possible mechanisms are present.

1. Complete recovery: Usually within 6 months – Noted in ischemic condition. However, aneurysms take nearly 2 years and traumatic cases from 6 months to 1-2 years.
2. No recovery or change in Palsy: Usually in cases where the nerve is transacted by trauma or chronic compression or has been infiltrated by a tumor.
3. Partial recovery: Especially if there is fascicular lesion.
4. Partial recovery characterized by oculomotor nerve synkinesis: Usually becomes apparent 9 weeks after injury.

Aberrant regeneration of III nerve:

Often after trauma and aneurysms; occasionally after tumour or syphilis; Never after ischemic causes especially diabetes.

Types

Primary – without apparent III nerve palsy in slow growing lesions of cavernous sinus such as meningiomas and aneurysm or birth trauma.

Secondary – following apparent III nerve palsy.

TROCHLEAR NERVE PARALYSIS

Like III nerve, it may be either congenital or acquired.

Congenital trochlear nerve palsy

Not very uncommon. Unmasked when one eye undergoes surgical correction. In adults, it may be presented at 5th or 6th decade, when it decompensates after minor injury without any cause.

Two diagnostic signs are:

1. Large vertical fusion amplitude (10-15 diopters).
2. FAT scan (Family Album Tomography) look at old photographs to detect longstanding head tilt indicative of congenital etiology.

Acquired paralysis

Trauma is the commonest cause of acquired palsies which may be following a direct orbital, frontal or oblique cranial blows or a contrecoup contusion at the tentorial notch may be unilateral or bilateral. Though 68% of cases are due to trauma, the trauma may not be very severe at first instance – Mostly following a rear end automobile collision. Usually, there will not be any loss of consciousness and there will be just a bump on head without redness or swelling of eye. Usually recovers in 3 to 6 months.

Lesions of the trochlear nerve nucleus

Lesions of trochlear nerve nucleus cannot be localized with certainty unless there are other neurologic signs suggesting intrinsic mesencephalic damage. It can be obscured by associated gaze palsy. It is due to either intrinsic lesions or extrinsic compression or dorsal mesencephalon, which may be due to vascular causes leading to hemorrhage, infarction, brainstem AV malformation, medulloblastoma, ependymoma or metastatic tumour.

Extrinsic lesions include – pinealoma, aqueductal stenosis, hydrocephalus associated with ipsilateral or contralateral III nerve palsy.

Lesions of trochlear nerve fasciculus

Difficult to diagnose and differentiate from nuclear lesions but usually without any associated neurological lesion. Diagnosis is by neuroradiological investigations like CT or MRI. May be associated with contralateral Horner's syndrome as the sympathetic pathways run through the dorsolateral tegmentum of mesencephalon adjacent to trochlear fasciculus.

LESIONS OF THE TROCHLEAR NERVE IN SUBARACHNOID SPACE

The nerve is susceptible to trauma or compression as it emerges from the dorsal surface of brainstem by avulsion of emerging rootlets or contusion injury due to hemorrhage within the nerve. These are usually bilateral.

As the trochlear nerve passes forward the cavernous sinus, it is protected from trauma and compression from tumor or aneurysm by the overlying rigid margin of the tentorium. It can however be affected by neurological misadventure and also as part of meningitic process due to syphilis or tuberculosis or even due to minor trauma as this nerve is the thinnest cranial nerve. It can also be involved in cases of tentorial meningiomas, pinealoma, sphenoidal sinusitis, encephalitis, migraine and even Schwannomas. Schwannomas trochlear nerve palsies due to tumor may be associated with intracranial tumor and may need careful check up of ocular and neurological examination. Rarely it is involved in aneurysm in posterior fossa and also chronic mastoiditis and pseudotumor cerebri associated with papilledema.

Lesions of Trochlear nerve within the cavernous sinus and superior orbital fissure

Usually involved with other cranial nerve palsies. However, isolated IV nerve palsy occurs in this area due to ischemic condition like diabetes or due to local granulomatous angiitis from Herpes zoster involving either division of IV nerve or even the geniculate ganglion. Rarely due to compression from persistent primitive trigeminal artery. Etiologies similar to III nerve.

Lesions of trochlear nerve within the orbit

Commonest cause is trauma. May also be due to inflammation and ischemia but it is impossible to ascertain whether the damage has occurred to the nerve, trochlea or the superior oblique muscle and/or tendon. May also follow neurological and dental anaesthesia – probably due to ischemia. Superior oblique muscle can be affected due to Paget's disease or hypertrophic arthritis. Tenonitis, neoplastic infiltration and inflammatory process can also decrease the motility.

Lesions of trochlear nerve of uncertain and variable location

It may be due to progressive sclerosis or allergic granulomatous angitis called Churg-Strauss syndrome. This is due to vasculitis involving the vasa nervorum of trochlear nerve.

Superior oblique myokymia is a rare phenomenon leading to oscillopsia. All cases appear to be benign. First reported by Hoyt. Cause is unknown and may respond to Tegretol.

Brown's syndrome which involves restriction of the muscle and sheath as it slides through the trochlea. It is not innervational but mechanical and it is therefore an entirely different problem.

Recovery from IV Paralysis

1. Complete recovery – Following ischemia or closed head injury or after relief of compression from tumor or aneurysms.
2. Incomplete recovery – Leaving the patient with mild persistent vertical and torsional diplopia.
3. No recovery – Primarily after mesencephalic injury or with transection of the trochlear nerve by trauma or compression.

ABDUCENS NERVE PARALYSIS

The 6th cranial nerve is affected in 30-50% of all ocular muscle palsies. Lesions of the nerve produce different syndromes from those seen with oculomotor and trochlear nerve palsies. It may be either congenital or acquired.

Congenital

Can occur rarely following birth trauma and disappear in 6 weeks may be associated with horizontal gaze palsy which occurs as an isolated sporadic or a familial condition may be associated with other neurologic and/or systemic signs or present as syndromes. Two important syndromes are,

- i. Mobius syndrome – Associated with other congenital malformations.
- ii. Duane's syndrome – The exact etiology not known; not classified under neurologic palsies.

Acquired Paralysis

Depending on the nature and location of lesion, the VI nerve may be involved separately or in association with other cranial nerves or neurological signs.

Lesions of abducens nerve nucleus

As the abducens nucleus is close to the pontine horizontal gaze centre, a lesion here produces conjugate gaze palsy to the ipsilateral side while conjugate horizontal gaze is totally abolished in bilateral cases. In most cases it is associated with ipsilateral peripheral facial palsy as the facial nerve facial loops around the abducens nerve nucleus. This is often asymmetric and is worse in abducting eye because the abducens motor neurons are more vulnerable than those of the internuclear neurons. Common causes include ischemia, infiltration, inflammation, trauma and compression or a combination of several factors.

Lesions of abducens nerve fascicle

When associated with gaze palsy it is difficult to differentiate from nuclear lesion. The common causes include –ischemia, tumour compression or infiltration, infection, inflammation especially dysmyelination.

Syndromes associated with fascicular lesions

Name	Site	Damage to	Lesion Produced
Foville's syndrome (or) syndrome of anterior – inferior cerebellar artery	Pontine tegmentum	<ol style="list-style-type: none"> 1. Facial & abducens nerve fascicles. 2. Nucleus of the tractus solitarius. 3. Central tegmental tract. 4. Spinal tract of trigeminal nerve and its nucleus. 5. And/or superior olivary nucleus. 	<ol style="list-style-type: none"> 1. Ipsilateral abduction paralysis. 2. Ipsilateral LMN facial palsy. 3. Loss of taste from anterior 2/3rd of tongue. 4. Ipsilateral Horner's syndrome. 5. Ipsilateral analgesia of face. 6. Ipsilateral peripheral deafness.
Millard Gubler's syndrome	Ventral paramedian pons	<ol style="list-style-type: none"> 1. Abducens fascicle. 2. Corticospinal tract. 3. And/or ventral part of facial fascicle. 	<ol style="list-style-type: none"> 1. VI and VII cranial nerve palsies. 2. Contralateral hemiplegia.
Raymond's syndrome	Lower pons	<ol style="list-style-type: none"> 1. Abducens fascicle 2. Corticospinal tract. 	Abduction palsy & contralateral hemiplegia.

Lesions of abducens nerve in the subarachnoid region

Because of the long course of the nerve, it is frequently involved due to varied causes in the subarachnoid region.

The lesions include

Vascular : The basilar artery lies in between the two abducens nerves and the anterior inferior cerebellar artery claps around the nerve, along with posterior cerebellar artery which lies closer. The nerve may be compressed by the atherosclerosis, dolichoectasia or aneurysm of these arteries.

Compressive lesions : Space occupying lesions above the tentorium (transtentorial masses) posterior fossa masses, structural malformation like Arnold Chiari malformation.

Trauma : Either direct including neurological trauma or indirect from blunt trauma in closed head injury.

Inflammation : Meningitis – Bacterial, viral, syphilitic, tuberculous, neoplastic associated with AIDS; unilateral or bilateral VI nerve palsy along with other cranial nerve palsies are noted.

Basal tumors : Meningiomas and chordomas may produce abducens palsy without any other neurologic signs and symptom, frequently bilateral. Other tumors include subarachnoid trigeminal neuroma and exophytic spread of intrinsic posterior fossa tumors such as glioma, medulloblastomas or ependymoma. C.P. angle tumors like acoustic neuromas cause hearing loss and impairment of corneal reflex as first symptom along with VI nerve and VII nerve palsy and ataxia.

Rise in ICP : Compresses the nerve between the pons and basilar artery or stretches it along the petrous temporal bone leading to unilateral or bilateral VI nerve palsies. May also follow spinal anaesthesia or lumbar picture or myelography.

Lesions of extradural portion of VI nerve at petrous apex

Gradenigo's syndrome : As the abducens nerve penetrates the dura overlying the clivus, it passes beneath the petroclinoid (Gruber's) ligament and close to mastoid air cells. In patients with severe mastoiditis, the inflammation extends to the tip of the petrous bone, producing local inflammation of meninges. There is VI and VII nerve paralysis along with hearing loss; and irritation of gasserian ganglion leading to severe ipsilateral pain in the face, around eye etc. associated with lacrimation, photophobia and diminished corneal sensation.

Pseudo Gradenigo's syndrome : Sometimes occur due to tumor infiltration at this region especially by nasopharyngeal tumor extension or compression due to aneurysms of intrapetrous part of internal carotid artery.

Other causes include involvement of VI nerve following lateral sinus thrombosis or phlebitis extending into inferior petrosal sinus or due to dural arteriovenous malformation in the superior petrosal sinus or due to compression by the persistent trigeminal artery. Trauma leading to longitudinal fractures of petrous temporal bone may lead on to abducens palsy along with V, VI, VII nerve palsy with hemotympanum, Battle's sign and CSF otorrhea.

Lesions of abducens nerve in the cavernous sinus and superior orbital fissure. –same as that of III nerve involvement.

Lesions of abducens nerve in the orbit

Due its short course in the orbit isolated involvement is rare. May be involved in orbital schwannoma or may be involved following dental anesthesia. It may be difficult to clinically differentiate the neural and muscular involvement of abduction.

Lesions of abducens nerve of uncertain or variable location

Though it can occur in all ages, it is more common in children. Causes in children include a post vaccination viremia or in new born following nonspecific febrile illness or respiratory illness. Usually recovers within 10 weeks but may recur in the same eye.

SYNDROME OF PAINFUL OPHTHALMOPLEGIA

This constitutes the combination of single or multiple oculomotor palsies with pain in and about the eye. They may present as various syndromes depending on the location of the lesion.

Tolosa-hunt syndrome

It is an idiopathic nonspecific inflammation of cavernous sinus and pachymeningitis of the superior orbital fissure. The syndrome is self-limiting with dramatic response to corticosteroid therapy. It is characterized by sharp boring pain in and about the eye, ophthalmoplegia, sensory deficit in the first division of trigeminal nerve, dilated and fixed pupil, with optic nerve rarely involved. There may be even spontaneous remissions with complete or partial regression of deficits which may occur at intervals of months or years. Rarely bilateral.

Superior orbital fissure syndrome of Rochon-Duvigneaud

Any lesion involving the orbit from the posterotemporal aspect leading to involvement of the three ocular motor nerves along with I division of trigeminal. May extend to cavernous sinus also.

Clinical features include slight exophthalmos, total ophthalmoplegia, severe pain, sensory loss in the distribution of I division of trigeminal and oculosympathetic fibres. Lesions: 1) Inflammation in the sphenoidal and ethmoidal air sinuses (2) tumors (3) Rheumatic periostitis (4) Aneurysm (5) trauma (6) periosteal hematoma due to trauma (7) bony changes (8) blood cysts.

Orbital apex syndrome

Here along with ocular motor nerves, optic nerve, lacrimal and frontal nerves are involved. Ophthalmic artery with sympathetic plexus and orbital veins are also involved. Trauma leading to incomplete palsy and if due to hemorrhage it is reversible. May be involved in tumor invasion also where it may lead to compression of orbital vein – pseudoorbital apex syndrome.

Cavernous sinus syndrome

The third, fourth, sixth along with sympathetic and ophthalmic trigeminal leading on to multiple ocular motor palsies pain, numbness along II division of trigeminal with “apparent pupillary sparing”, VI nerve is most commonly affected as it forms content of cavernous sinus along with internal carotid artery.

Divided into

Anterior cavernous sinus syndrome – III, IV, VI & V1 of V

Posterior cavernous sinus syndrome – III, IV, VI, V1 & V2 of V – described earlier.

Diabetic ophthalmoplegia

20-45% of the mononeuropathies of III, IV & VI nerves are due to diabetes. In this, palsies of III & VI nerve are more frequent while the IV nerve is the least affected in diabetes. It can also lead to simultaneous involvement of two or more cranial nerves rarely. In these cases, CT & MRI are indicated to rule out compressive lesions. Pupillary sparing is an important feature of diabetic third nerve palsy as the lesion is mainly due to ischemic demyelination due to obstruction of the vasonervorum of the affected cranial nerve. Remyelination is thought to be responsible for recovery without aberrant regeneration.

MANAGEMENT OF OCULAR NERVE PALSIES

Acquired cranial nerve palsy is symptomatic during the first few months, after onset. Diplopia in many patients is controlled by compensatory head posture and requires no treatment.

Some patients who are unable to control the diplopia the treatment options include the use of Fresnel prism's if the angle of deviation is small, uniocular occlusion and botulinum toxin injection into the uninvolved rectus muscle especially in lateral rectus muscle in case of oculomotor nerve palsy.

AIM OF STUDY

- To find out the common etiology and incidence of the infra nuclear neurological lesions of III, IV and VI cranial nerves.
- To study the pattern of recovery in each type of nerve palsy in these cases.
- To emphasize the importance of non invasive techniques in these cases to clinch the localization of involvement and probable aetiology according to localization.
- To find out the mode of treatment to be followed in cases of both recent and established cases of neurological palsies involving III, IV and VI cranial nerves.

MATERIALS AND METHODS

This study was conducted in department of ophthalmology, Coimbatore Medical College Hospital, Coimbatore. The study period was from august 2007 to august 2009. All patients attending to ophthalmology department and referred cases from other departments, who are having neurologic lesion of III, IV and VI cranial nerves mainly infra nuclear neuropathic ocular movement disorder were included in this study.

Patients with supra nuclear, nuclear, inter nuclear and myogenic types were excluded from the study by doing suitable examination and investigations.

Patients with any ocular cranial nerve palsies were indentified and their age, sex, duration of paralysis was noted. After this, a detailed history of the mode of onset, progression, and treatment if any received, or any previous surgeries was recorded. History or relevant associated diseases such as diabetes, hypertension, tuberculosis, syphilis, past meningitis, stroke, hypothyroidism, hyperthyroidism, myasthenia and migraine were noted. Then history specific to ocular complaints such as diplopia, blurring of vision, defective visual field, drooping of upper lid, vestibular complaints of vertigo, oscillopsia and tilt, were recorded. History of trauma, any history suggestive of raised intracranial tension, fever, ear discharge, and deafness was elucidated and recorded. Any other history that the patient has volunteered which was found relevant to the diagnosis was also noted. Patients were also questioned regarding family history of similar complaints and consanguinity. Personal history regarding smoking, alcohol intake and the diet history was also noted.

GENERAL EXAMINATION

Each patient was subjected to a detailed general examination with particular reference to anaemia, generalized lymphadenopathy and nutritional status and blood pressure in adult patients.

OCULAR EXAMINATION

Thorough ocular examination is done in all cases. Any abnormality in head posture, any head tilt, face turn, and chin position were noted. Any skull abnormalities also noted. The lids were examined for ptosis, retraction, lid lag, presence of Marcus Gunn phenomenon, or any other synkinetic movement. Amount of ptosis and presence or absence of levator action was also noted.

Regarding eye position, presents of deviation was first noted in all positions of gaze with either eye fixating. Ocular movement were tested in all positions in one eye (ductions) and also in both eyes (Versions and vergence). The primary and secondary deviations were tested by cover and un cover test.

A thorough examination of anterior segments using slit lamp was done. Pupils were tested for size, shape and reaction both direct and consensual, vergence and accommodation. Fundus examination was done meticulously to find out any associated fundus abnormalities.

The unaided visual acuity, retinoscopic value, corrected visual acuity, colour vision, examination of the fields- both central and peripheral as well as recording of intra ocular pressure were done as a routine.

Specific tests include Diplopia charting and Hess screen to find out both the defective muscle action also its sequelae. These tests were repeated during each review for the same reason.

NEUROLOGICAL EXAMINATION

A full neurological examination was done on each patient. Examination of higher functions, other cranial nerves, motor and sensory systems and cerebellar functions were examined.

INVESTIGATIONS

A complete haemogram, urine analysis, blood sugar estimation, Mantoux and Blood VDRL were done. X-ray of chest, orbit, paranasal sinuses and skull was done in indicated cases. CT scan and MRI were done in selected cases.

If necessary, neurologist's opinion, ENT opinion, diabetologist's opinion and any other specialist's opinion to come to definite clinical diagnosis was sought. After these appropriate treatment was started. Patient was asked to come for follow up once in two weeks.

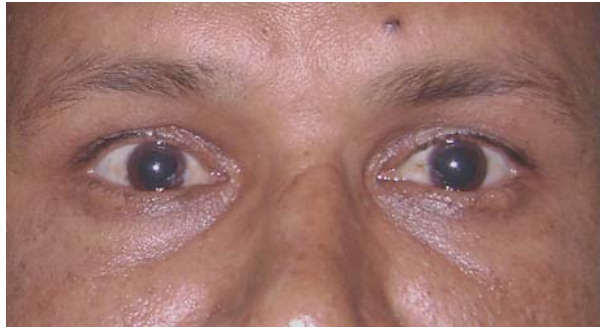
TREATMENT

In all cases where a specific cause is found, such as diabetes, hypertension, tuberculosis or any other specific infection, the treatment was directed to the treatment of the cause. In cases of diabetic neuritis, diabetologist's for control and further usage of drugs was got.

For patients who had any specific neurological problems, opinion from neurologist was got and so also from ENT surgeon for suspected ENT causes. In case of non specific neuritis which proved to be the commonest cause, steroids in the form

of oral prednisolone in divided doses was given along with neuro vitamins and analgesics if there was associated pain. Steroids were tapered off after 6 weeks or in cases where there was partial recovery it was given for some more time in the same dosage and then tapered off.

Right Sixth Nerve Palsy



Primary Position



Limitation of right Abduction

After Recovery



Normal Abduction



Normal Adduction

Right Sixth Nerve Palsy



Slight Right Esotropia
in the Primary Position



Normal Right adduction



Limitation of Right Abduction



Normal Elevation

Recovery after Six Months



Primary Position



RE Normal Abduction



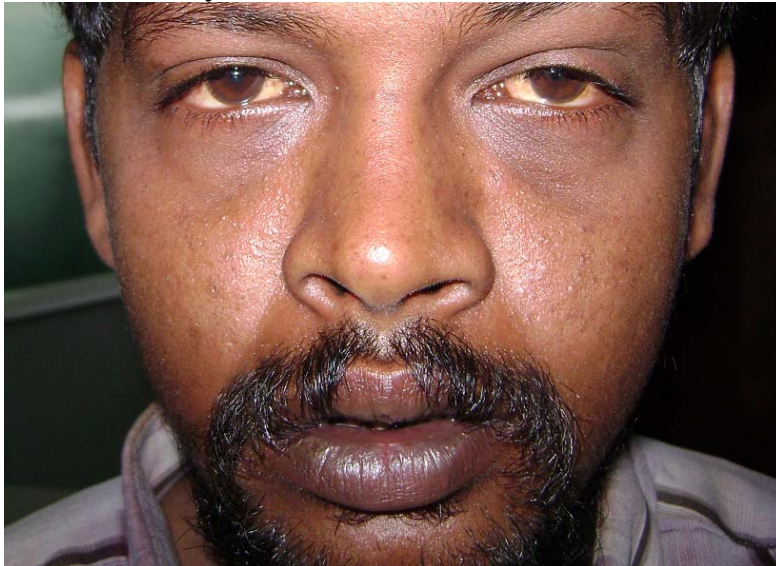
RE Normal Adduction

Left Fourth Nerve Palsy



Head Tilt to
Right

After Recovery



Normal
Head Posture

Non-Recovered Right Sixth Nerve



Primary Position
Right Esotropia

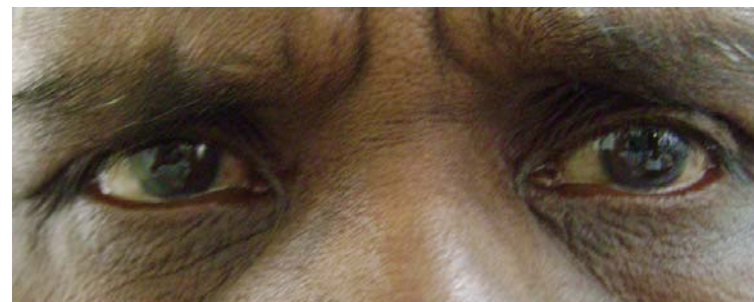


Absent Right
Abduction

Diabetic Partial Third Nerve Palsy



After Recovery



RE III Nerve Palsy (Diabetic)



RE Ptosis



Primary Position
RE Exotropic



RE Absent Adduction



RE Absent Elevation

Recovery After Three Months



Primary Position

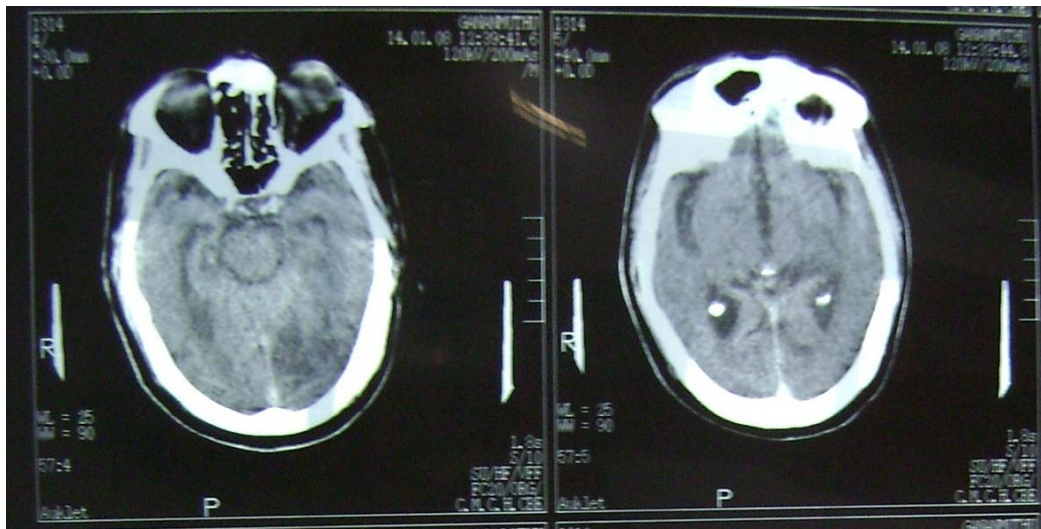


RE Normal Adduction



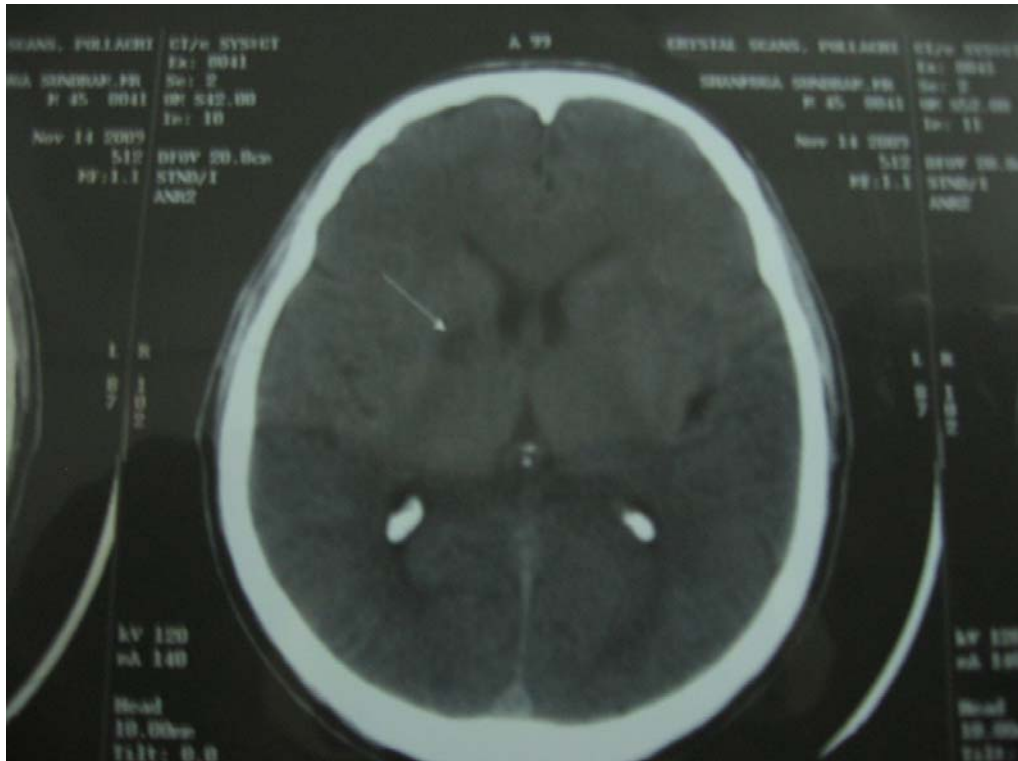
RE Normal Elevation

CT-BRAIN



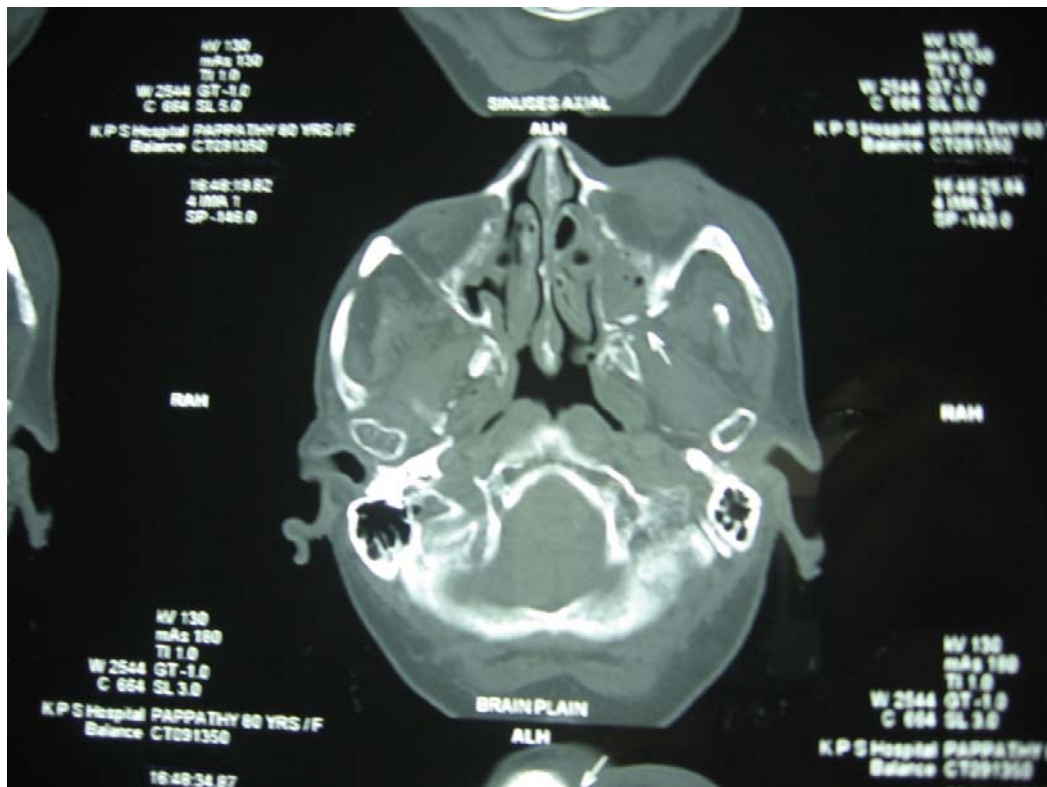
Non Hemorrhagic infarct in Left Half of Brain stem and Left Parieto Occipital Region

CT-BRAIN



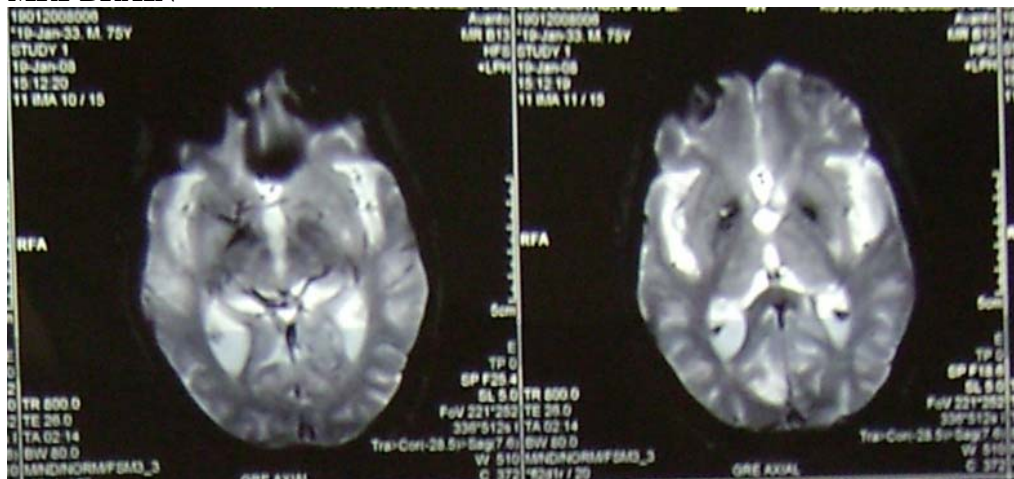
Right capsulo ganglionic infarct

CT-BRAIN WITH ORBIT



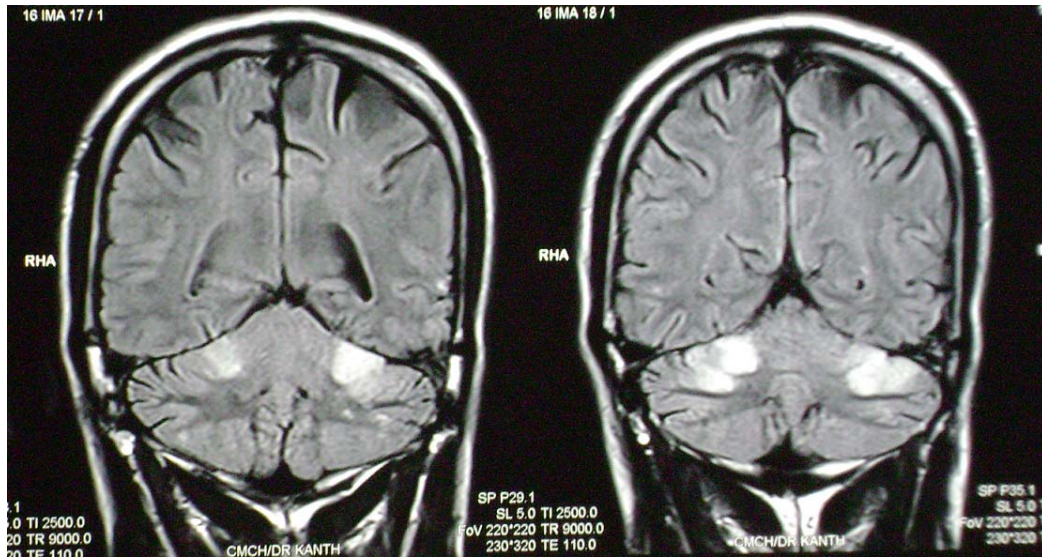
Fracture left frontal and temporal bone with pneumo encephalocele

MRI-BRAIN



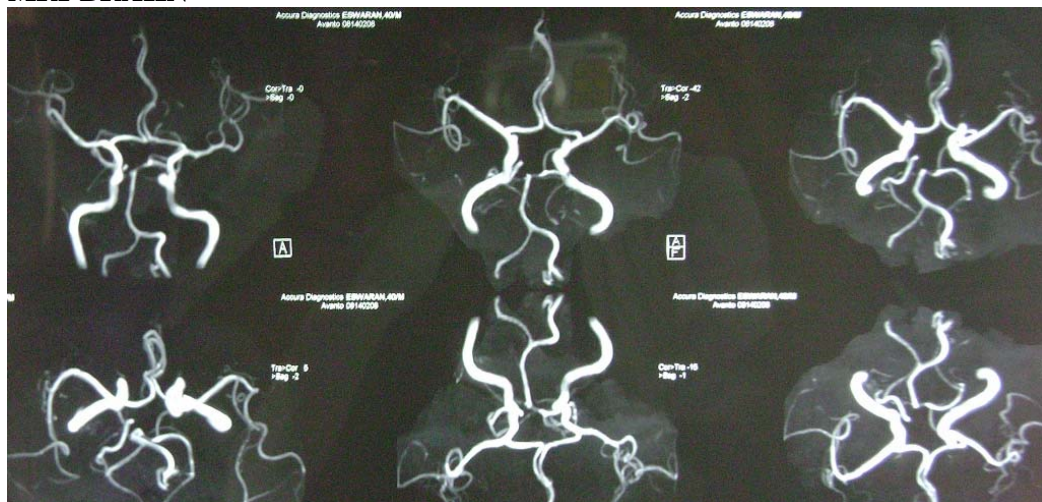
Sub-acute Infarct Involving Left Cerebellar Hemisphere, Brain Stem and Periventricular Ischemic Changes

MRI-BRAIN



Subacute infarcts in the posterior circulation involving the cerebellar hemispheres, brainstem and the medial thalami.

MRI-BRAIN



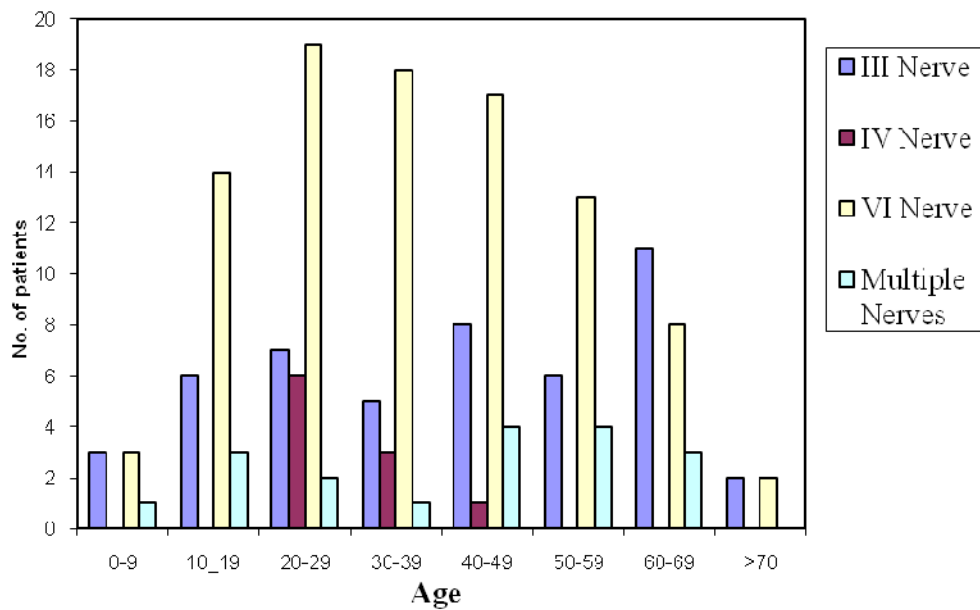
Normal MRA of Brain

RESULTS

AGE

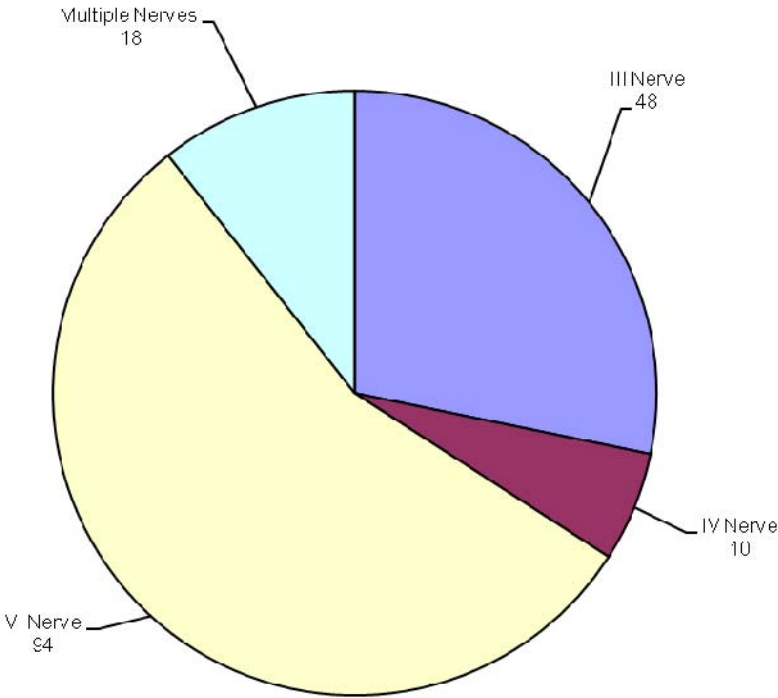
Age in years	III Nerve	IV Nerve	VI Nerve	Multiple Nerves	Total
0-9	3	-	3	1	7
10-19	6	-	14	3	23
20-29	7	6	19	2	34
30-39	5	3	18	1	27
40-49	8	1	17	4	30
50-59	6	-	13	4	23
60-69	11	-	8	3	22
>70	2	-	2	-	4
Total	48	10	94	18	170

Age Distribution



Of the 170 cases of Ocular cranial nerve palsies, 82.35% occurred between 20-70 years, the range being 1 ½ - 70 years. In this study maximum number of III nerve palsies were found only between 60 – 69 years i.e. 11 cases (22.17%) followed by 40 – 49 years –8 cases (16.67%). All trochlear nerve palsies are between 20 – 44 years of age. There is wide distribution of cases of VI nerve palsies from 2nd to 7th decade, with 71.28% falling between 20 – 60 years of age. In case of multiple cranial nerve palsies 50% of the cases fell between 40 and 60 years of age. In case of III nerve palsies, 8 cases out of 10 cases due to diabetes and all 5 cases due to hypertension belonged to the age group between 45 to 70 explaining large number of cases in this age group who are prone for microangiopathic lesions due to these illnesses. In cases of IV nerve, trauma either closed head trauma or severe head injuries seemed to be the commonest cause involving adult males between 20 and 40 years.

Distribution of Ocular Cranial Nerve Palsies

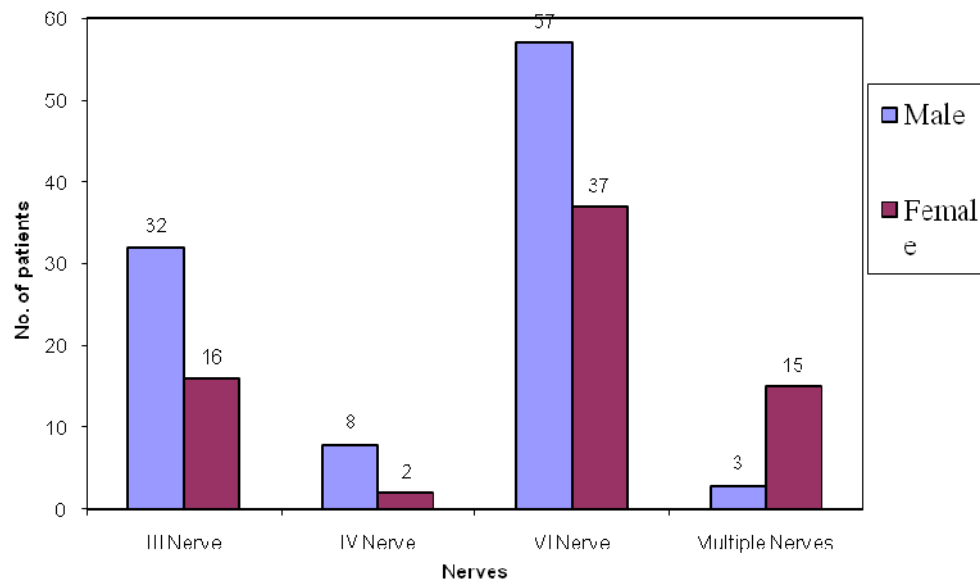


GENDER

Gender	III Nerve	IV Nerve	VI Nerve	Multiple Nerves	Total
Male	32	8	57	3	100 (58.82%)
Female	16	2	37	15	70 (41.18%)

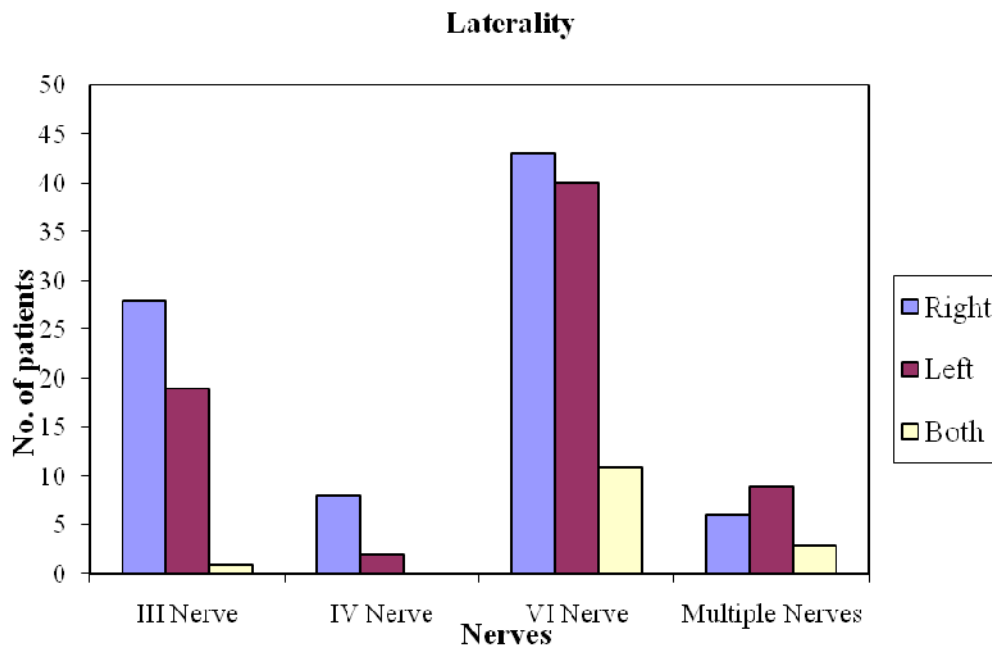
In general Males are more frequently affected i.e. 100 patients out of 170 (58.82%). In case of trochlear palsies 80% of the cases were found in males while in multiple cranial nerves affect more than 80% is in females.

Gender distribution



LATERALITY

Affected Eye	III Nerve	IV Nerve	VI Nerve	Multiple Nerves	Total
Right	28	8	43	6	85 (50%)
Left	19	2	40	9	70 (41.18%)
Both	1	-	11	3	15 (8.82%)

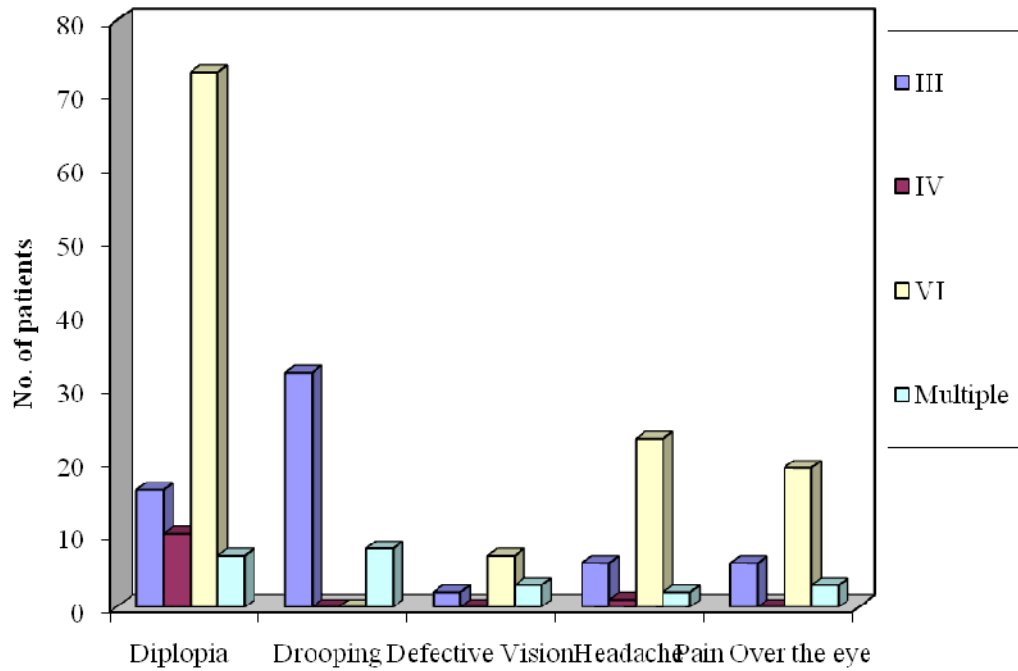


Right eye was the commonly affected eye i.e. in 50% of the cases except in cases of multiple cranial nerve palsies where half the number of cases occurred in LE while 16.66% occurred bilaterally and 33.33% affected in right eye. Of the 15 bilateral cases, one occurred in III nerve, 11 in VI nerve, and 3 with multiple cranial nerve involvement, while there are no bilateral involvement with IV nerve palsies.

SYMPTOMS

Nerve Affected	Diplopia	Drooping of Lid	Defective Vision	Headache	Pain Over the eye
III	16	32	2	6	6
IV	10	-	-	1	-
VI	73	-	7	23	19
Multiple	7	8	3	2	3

Symptoms



General: Commonest systemic association was diabetes Mellitus in 27 patients (15.88%) followed by fever in 14 patients (8.23%) Nausea and vomiting were found in few patients only. Most of the diabetes were aware of the disease and were under treatment. Only 4 had evidence of retinopathy.

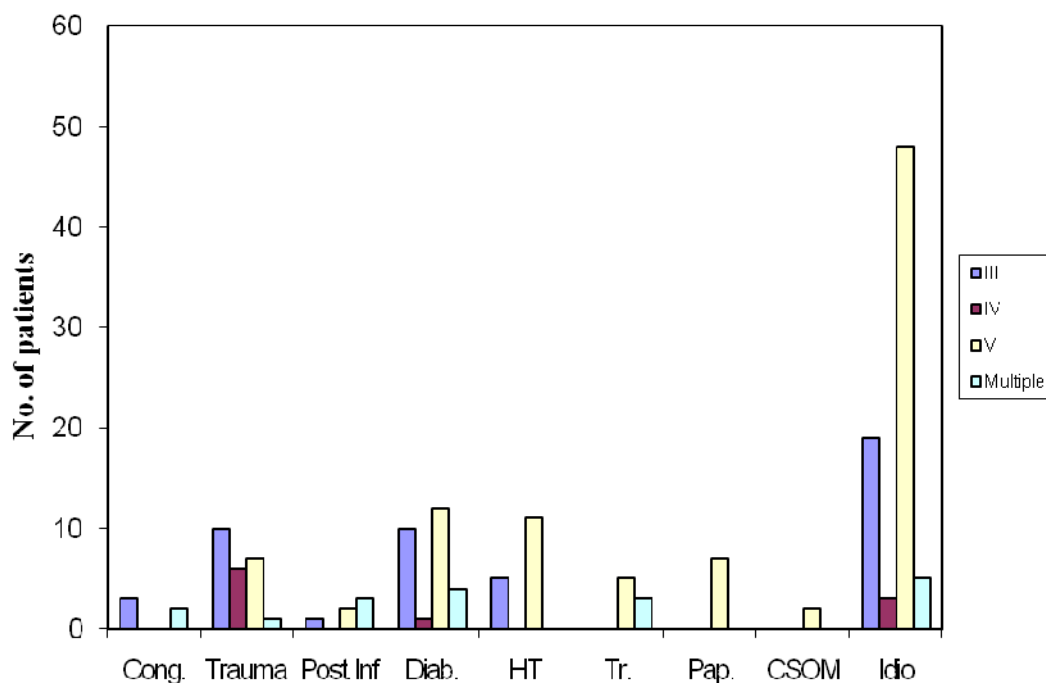
Ocular: Commonest ocular symptom is diplopia especially almost in all cases of VI nerve and also in cases of IV nerve. In case of IV nerve reporting is mainly due to difficulty in climbing down stairs and reading. In patients with III nerve palsies the main complaint was drooping of the eyelid and diplopia was noted only during the recovery stage by many of them. In multiple cranial nerve palsies, depending on the involvement of nerves diplopia or drooping of lids formed the commonest presenting symptoms.

AETIOLOGY

The various etiologies leading to affection of the ocular motor cranial nerves are given in the following tables.

Nerve	Cong.	Trauma	Post. Inf	Diab.	HT	Tr.	Pap.	CSOM	Idiopathic
III	3	10 (20.83%)	1	10 (20.83%)	5	-	-	-	19 (39.58%)
IV	-	6	-	1	-	-	-	-	3
VI	-	7	2	12	11	5	7	2	48
Multiple	2	1	3	4	-	3	-	-	5
Total	5	24 (14.11%)	6	27 (15.88%)	16	8	7	2	73 (42.94%)

Etiology



III Cranial Nerve Palsies

Of the total 48 cases, the largest number i.e. 19 belonged to non specific neuritis (39.58%). This diagnosis is arrived at after eliminating other possible causes by doing relevant investigations. Next is the microangiopathy due to diabetes – 10 cases (20.83%) and traumatic –10 cases (20.83%). 8 of the diabetic cases and the 5 hypertensive patients belonged to the age group between 45 and 70 years. In patients with Trauma only 2 people belonged to this age group while others were below 40 years where trauma is common.

IV Cranial Nerve Palsies

Commonest cause is trauma i.e. 6 cases (60%). One diabetic (10%) and 3 non specific neuritis. (30%).8 cases in males – 6 of them due to trauma and 1 due non specific neuritis. All between 20 and 40 years.

VI Cranial Nerve Palsies

Of the 94 cases more than 50% i.e. 48 cases (51.06%) belonged to non specific neuritis. Here too, all the relevant investigations and neurological examination were done to eliminate the other possible causes. 12 cases were due to diabetes followed by 11 cases due to hypertension, especially in older age group between 50 and 70 years. Two cases of children had CSOM associated with VI nerve palsy. Two cases one with III nerve and another with VI nerve involvement were thought to be due to demyelination after investigations and sent to neurology department for opinion.. 11 cases had bilateral VI nerve palsies. In these 4 cases had evidence of papilledema – 1 case following postpartum eclampsia improved well with treatment; 2 cases had hyperemic discs following fever with neck rigidity and were sent to paediatric neurology for further treatment; 1 case was due to uncontrolled hypertension in 31 years old male. In other cases, 1 was due to diabetes, 1 was due to non specific neuritis 1 was due to suspected sellar tumour and 1 suspected demyelination.

Multiple Cranial Nerve Palsies

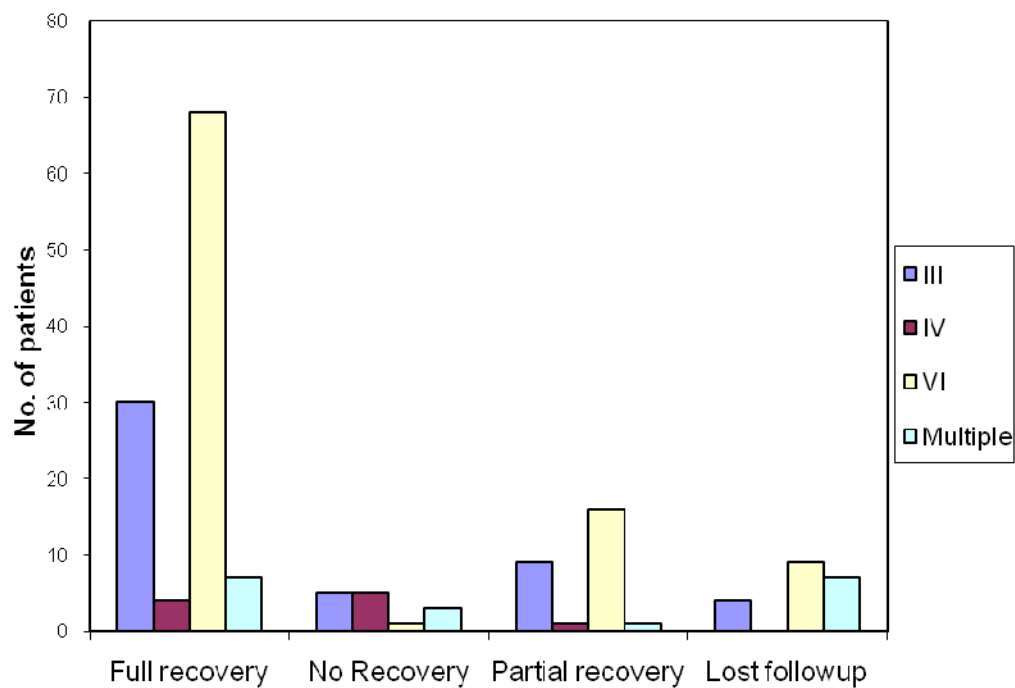
Of the 18 cases, 4 were due to diabetes, 1 following trauma, 3 following tumours, 3 following specific inflammation, 2 were congenital, 5 due to non specific inflammation. 3 cases of tumour were following astrocytoma, maxillary growth and juvenile nasopharyngeal angioma. 3 cases following specific inflammation were post Tuberculous meningitic, post typhoid, and post exanthematous fever

RECOVERY PATTERN

All the ocular motor cranial nerves showed full recovery in more than 50% of cases but the recovery is poor if the lesion is due to trauma especially head injuries.

Nerve affected	Full recovery	No Recovery	Partial recovery	Lost followup
III	30	5	9	4
IV	4	5	1	-
VI	68	1	16	9
Multiple	7	3	1	7
Total	109	14	27	20

Recovery Pattern



All the cases with non specific neuritis and also microangiopathy due to diabetes or hypertension showed mostly full recovery or at least partial recovery in 4 months. The average recovery time for IV nerve palsies due to non specific neuritis and diabetes ranged from 6 to 12 weeks. Average recovery time in VI nerve palsies ranged from 6 to 16 weeks. In III nerve palsies the recovery was noted within 4 weeks and the 1st muscle to recover was the LPS leading to diplopia if it was not present already. This was followed by medial rectus, superior rectus, inferior oblique and inferior rectus. Inferior rectus took longer time to recover. There was no discrepancy if the palsy was total or partial and age and sex had no correlation with the recovery pattern. Recovery was not good in posttraumatic cases. These were the cases along with those with organic lesions sent for higher investigations usually lost for followup.

DISCUSSION

Ocular Cranial nerve palsies are always a diagnostic and therapeutic dilemma to the ophthalmologists. The acquired cranial nerve palsies in this study are compared with similar studies done earlier to analyze the various etiologies and also general recovery pattern of these nerve afflictions.

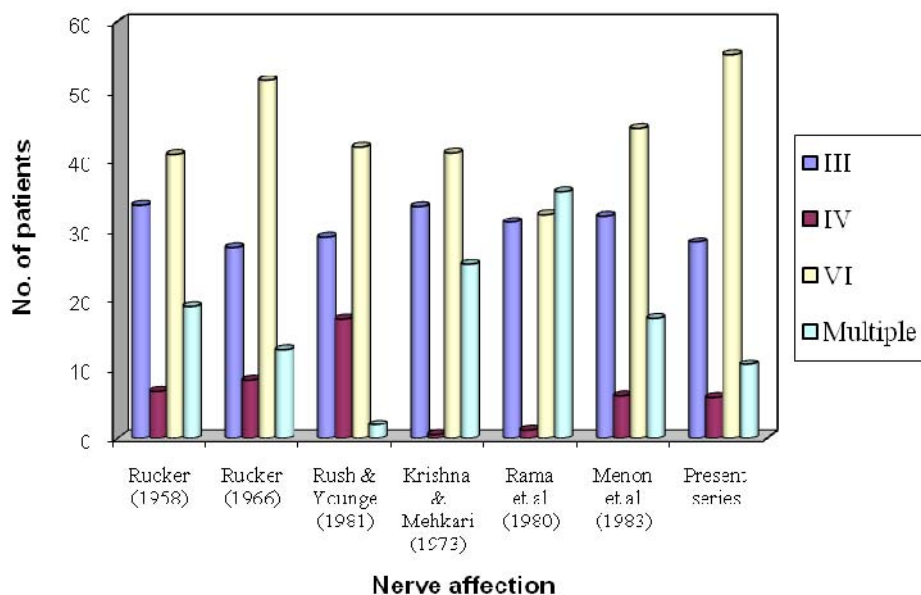
Nerve Affection

Comparison cases of acquired extra ocular muscle paralysis Nerves Affected

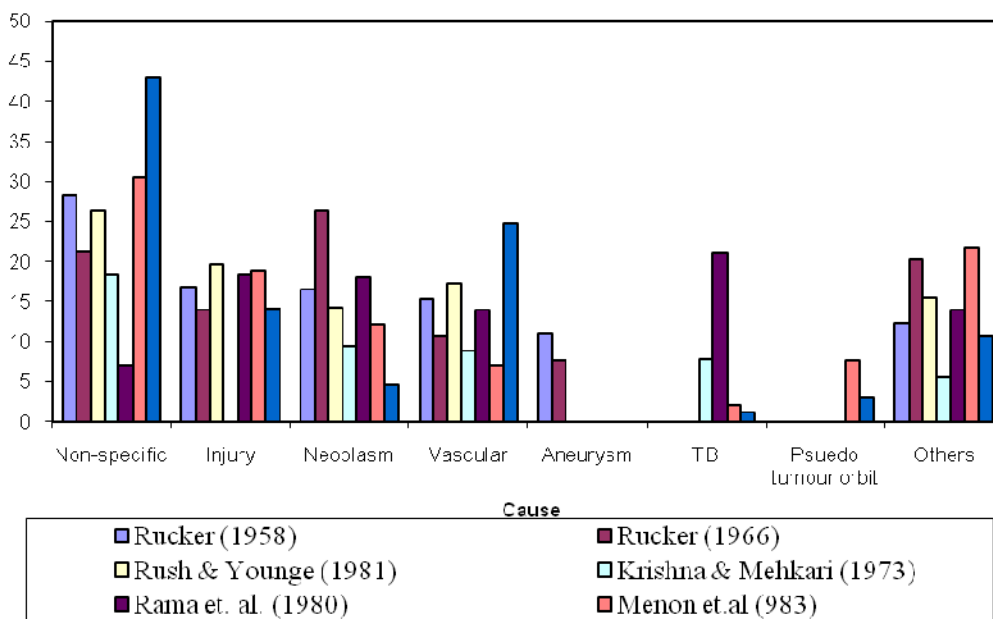
Cranial Nerve	Rucker (1958)	Rucker (1966)	Krishna & Mehkari (1973)	Rama Et.al (1980)	Rush & Younge (1981)	Menon Et.al (1983)	Present series
III	33.5	27.4	33.3	31.1	29.0	32.0	28.22
IV	6.7	8.4	0.5	1.1	17.2	6.1	5.88
VI	40.9	51.5	41.1	32.2	41.9	44.6	55.29
Multiple	18.9	12.7	25	35.5	1.9	17.3	10.59

In this study IV nerve was the commonly affected nerve (55.29%). This is in accordance with the studies quoted in the above table comparing both Western and Indian studies. As per the study conducted on 4298 cases of ocular motor paralysis by Richards, Jones and Younge from Mayo clinic in 1992, the largest group had involvement due to undetermined causes and the most frequently affected nerve was the sixth nerve. But according to the study by Berlitz P in 1992 in a series of 412 patients the III nerve was the most commonly affected nerve. According to study by

Comparison cases of acquired extra ocular muscle paralysis Nerves Affected



Causes of paralysis of cranial nerves comparison of previous reports and present series (%)



Tiffin et.al also 57% were VI nerve but after that IV nerve in 21% while III nerve palsies is only 17%. VI nerve is more affected may be because of its long intracranial course with bends.

Gender Distribution

In this study there is a slight pre ponderance of male patients (58.82%) slightly higher than compared with series by Rush (1981) and Richards Jones and Younge in (1992), where it 52% and 54% respectively. It may too probably due to the early approach of male patient for treatment and also an inherent hormonal protection in females. Laterality has got no significance in any of these studies.

Presenting symptoms

Regarding presenting symptoms diplopia forms the commonest presenting symptom in 106 of 170 patients. (62.35%) same as that of the study by Kubutko-Zielinska et . al in 1995 where diplopia formed the presenting symptoms in 90% of cases.

Causes of paralysis of cranial nerves comparison of previous reports and present series (%)

Cause	Rucker (1958)	Rucker (1966)	Krishna & Mehkari (1973)	Rama Et. Al. (1980)	Rush & Younge (1981)	Menon et.al (1983)	Present series
Non-specific	28.2	21.1	18.3	7	26.3	30.5	42.94
Injury	16.8	13.9	-	18.3	19.7	18.7	14.11
Neoplasm	16.5	26.3	9.5	18	14.3	12.2	4.70
Vascular	15.3	10.7	8.9	14	17.2	7.1	24.7
Aneurysm	10.9	7.7	-	-	-	-	-
TB	-	-	7.8	21	-	2.1	1.176
Pseudo tumour orbit	-	-	-	-	-	7.6	2.941
Others	12.3	20.3	5.6	14	15.4	21.75	10.72

Diabetes mellitus is the commonest general systemic disorder associated with ocular motor nerve palsies in correlation with the studies in the above table and also in the study by Watanabe et.al in 1990 who found out that the incidence of cranial nerve palsies in diabetic patients was significantly higher than in non-diabetic patients. 79/170 patients in this series were above the age of 40 where the incidence of diabetes is expected to be higher than in younger age group. In this study the incidence of diabetes was less than only to non specific causes. One case had pupillary involvement along with diabetes. As the success rate of CT increases above 60% according to the study conducted by Kwan ES et al, hypertension and diabetes

have to be ruled out before ordering CT and a time limit of about 2 to 4 weeks are allowed to note any improvement or worsening to decide about ordering higher investigative aids.

Trauma though commonest cause for trochlear nerve palsies, it can also affect the III and VI cranial nerves. According to the study by Tokuno T. Natkazawa K. et.al. there is high incidence of traumatic Sub Arachnoid Hemorrhage (71%) and skull fractures i.e. (57%). In this study only 14.70% are due to trauma but in these cases the recovery from the palsies is not good even after long followup in correlation with the above study.

OCULOMOTOR PALSIES

Congenital Palsies

In this series there were 3 congenital III nerve palsies. These were included in the study as the patients and parents were not sure of onset of problem and a diagnosis of congenital III nerve lesion was arrived at by excluding all other possible causes. Two cases reported in first decade and 1 in second decade. These cases were uniquely without any other neurological deficits unlike in the study by Tsaloumas and Willshaw HE in 1997 on congenital Oculomotor palsies where 5/12 patients had neurological abnormalities.

Analysis of various etiologies

The results of this series is compared with the western and Indian series regarding etiologies in the above table. As in others non specific neuritis formed the commonest cause in the series also. This is followed by the vascular etiology due to diabetes and hypertension, which formed 24.7% of III nerve palsies in this series.

Here too comparing to that by Rush and Richards Younge, the largest group was non specific neuritis i.e. 39.58%. While vascular, trauma, aneurysm and neoplasms formed other common causes in their studies, there is no case of proved aneurysm in this series. Though pupillary dilatation was found in 6 cases, 4 of them are after trauma and one with diabetes and one with hypertension. In these cases apart from evidence of trauma the CT did not reveal any specific lesion. This is in comparison with the study conducted by Kwan ES et al., who stated that the overall diagnostic yield of CT for isolated III nerve palsies was low (30%) but improved to (60%) if diabetes and hypertension were excluded. It became highly sensitive i.e. 90% if associated with additional neurologic deficits. According to the study, the status of pupil in and of itself can not be the sole determinant as to whether angiography is indicated to exclude aneurysm. The study by JD Trobe in 1985 enumerates the conditions where arteography is indicated. According to the study by Bianchi Marzoli S, Brancato R. MRI has been confirmed to be the most important diagnostic tool, but relative non availability and high cost precluded its routine usage in this study.

VI NERVE PALSIES

It formed the commonest occurring ocular motor palsy in most of the series and also in this series.

Etiologies

Compared to the table above in this series also non specific neuritis form the major cause. According to the study by Miller et.al, vasculopathy followed by non specific neuritis formed the major cause.

Laterality

In this study, RE affection is more than LE (58.23%) with 11 cases of bilateral involvement. According to the study conducted by Berlitz P et.al., it is frequent on left (52%) with 10% bilateral involvement. According to the study by Keane JR, this is almost equal to unilateral affection and these cases have to be thoroughly investigated. According to the study conducted by Galletta SL and Smith JL any chronic isolated VI nerve palsies have to be thoroughly investigated especially to rule out tumours. There were 5 cases of intracranial tumours mostly with papilloedema and bilateral affection.

Recovery

In this study 72% recovered within 3 months. In the study by King A.J et.al it is slightly higher i.e. 78.4%. In those who failed to recover nearly 40% had serious underlying pathology. In this series also patients who have not recovered had serious problems like tumour or traumatic causes.

SUMMARY

1. Of the 170 cases of ocular cranial nerve palsies, 82.35% occurred between 20 – 70 years, the range being 1 ½ - 70 years. The maximum number of III nerve palsies were found only between 60 – 69 years. All trochlear nerve palsies are between 20 to 44 years of age. There is a wide distribution of cases of VI nerve palsies, from 2nd to 7th decade and multiple cranial nerve palsies 50% of cases fell between 40 – 60 years of age.
2. The distribution of cases is as follows:
VI nerve palsies – 94
III nerve palsies – 48
IV nerve palsies – 10
Multiple nerve palsies - 18
3. Number of patients affected.
Male – 100
Female - 70
4. Eye affected
Right Eye - 85
Left Eye – 70
Both Eyes - 15
5. Most of the cases reported within 1 month of affection and generally followed upto 3 months at 2 weekly interval upto a maximum period of 4 months.
6. Commonest systemic association was Diabetes (15.29%) followed by fever.

7. Commonest ocular symptom was diplopia in VI and IV nerve palsies and drooping of eyelid in III nerve palsies.
8. In the 170 cases, aetiology could not be determined in 73 cases i.e. 42.94%.
9. Of the 48 cases of III nerve palsies, the largest number belonged to non specific neuritis. Next is microangiopathy due to diabetes and trauma – 10 cases each followed by five cases of hypertension.
10. Trauma formed commonest etiology in IV cranial nerve palsy occurring in 60% of cases followed by 3 cases of non specific neuritis one case due to diabetes.
11. In the 94 cases VI nerve palsies, 48 cases belonged to non specific neuritis. 12 cases due to diabetes 11 cases due to hypertension. 11 cases were involved bilaterally.
12. In case of multiple cranial nerve palsies, 3 were due to post inflammatory cause, 5 due to non specific neuritis, 4 due to diabetes, 1 due to trauma and 3 due to tumours.
13. There were 27 cases of Diabetes Mellitus (15.88%) and about 15% were diagnosed at the time of palsies. Only four cases had evidence of diabetic retinopathy. Pain or headache on the affected side was present in 82.6% of these patients.
14. Of the 15 cases with bilateral involvement, 11 cases were with VI nerve involvement. 3 cases with multiple cranial nerve involvement and one case of III nerve involvement.

15. Among the 25 cases following trauma, the injury ranged from trivial to severe head trauma and most of them are below 40 years of age.
16. There were 3 cases where there were recurrences. One was in a male with III nerve palsy who did not come to follow up regularly. One is in a female with IV nerve – non specific neuritis and other in male in right eye VI nerve with H/O diabetes under treatment.
17. 109 cases had full recovery and another 27 cases had partial recovery.
18. Post traumatic cases had fewer chances for full recovery. The dropouts in follow up especially after 1 or 2 months belonged to this group.

CONCLUSION

From this study of 170 cases of cranial nerve palsies it is concluded that.

1. The VI cranial nerve is the most commonly affected nerve followed by III nerves and multiple cranial nerves and lastly the IV cranial nerve.
2. All these Ocular motor nerves can be affected by various pathological conditions.
3. Commonest presenting symptoms were diplopia, drooping of eye lid, pain and headache especially on the affected side.
4. Among the systemic conditions, diabetes followed by hypertension form the most commonly occurring association.
5. Closed head trauma even trivial is associated with trochlear nerve palsies while forcible head trauma as in cases of RTA leading III & VI cranial nerve palsies also.
6. There is no specific preponderance of infection in this series.
7. In 73 cases no specific cause could be made out
8. patients belonging to older age group were suffering from ocular motor cranial nerve palsies due to micro angiopathic lesions while trauma was the main cause in younger age group;
9. Non invasive procedures like CT & MRI were invaluable in diagnosis in selected cases.

10. Cases due to non specific neuritis or due to micro angiopathic lesions secondary to diabetes, hypertension etc recovered within 4 months regardless of the nerve affected.

11. Longer follow-up, better facilities to do MRI may help in finding out specific aetiologies in these cases classified as nonspecific neuritis.

BIBLIOGRAPHY

TEXT BOOK REFERENCES

Altered Kesten Baum M.D Clinical methods of Neuroophthalmologi examination II Ed.

Bajandas FK. Lanning Kline B. MD – Neuroophthal mology review manual IV Ed. Ind Jaypee brothers, 1998.

Duane TD., marshall M. Park MD – Vol.2, Chater 12 Duane's Clinical Ophthalmology – William Tasman, Edward A. Jaeger.

Jack J.Kandki- Clinical Ophthalmology – A systematic Approach – VIth Edition.- Ophthalmology – 2006, Page 816-824.

Neil R. Miller MD, Nancy J. Newman M.D.- Walsh and Hoyt's – Clinical 372Neuroophthalmology 1998 Fifth Edition.

Ronald M . Burde MD. Peter J. Savino MD, Jonathan D. Trobe MD-Clinical Decisions in Neuroophthalmology, 1985.

Roy W. Beck M.D. Craig H. Smith – Neuroophthalmology: A problem-Oriented Approach 1988 I Edition.

Sir Stewart Dke – Elder – System of Ophtalmology- Neuroophthalmology henry Kimpton Publishers 1973, Vol.12.

Thomas j. Walsh – Neuroophthamology- Clinical Signs and Symptoms-IVth Edition, 1997.

Wolff's anatomy of the Eye and Orbit- Eighth Edition, 1997.

JOURNAL REFERENCE

Berlit, P, Reinhardt Eckstein J, Krause KH. – isolated abduces paralysis-a retrospective study of 165 patients – *Forschr. Neurol. Psychiatr.* 1989 Jan. 57(1).

Berlit P – Isolated and combined palsies of cranial nerves III, IV and VI. A retrospective study of 412 patients – *J. Neurol. Psychiatr.* 1989 Jan. 57 (1).

Bianchi – Marzoli S, Brancata R – Third, fourth and Sixth nerve palsies. *Curr. Opin. Oph.* 1997 Dec.8, (6) 45 – 51.

Brazis PW – Localization of lesions of the oculomotor nerve: recent concepts – *Mayo Clinic Proc.* 1991 Oct.66 (10), 1029-35.

Brazis PW- Palsies of trochlear Nerve: Diagnosis and Localisation recent-concept – *Mayoclinic Proc.* 1993. May.68(5) 501 – 9.

CapoH, Warren F, Koppersmith MJ – Evolution of Oculomotor nerve palsies *J. Clin. Neuroophthal.* 1992. Mar.12(1) 21 – 5.

Cullom ME, Savino PJ, Sergott RC, Bosley TM, J. *Neuroophthal* 1995 Sep.15(3) – 136-40; Discussion 140 – 1.

Elston JS, Lee JP - Paralytic strabismus: the role of botulinum toxin *BJO* 1985 Dec.69 (12)891 – 6.

Harley RD – Paralytic strabismus in children. Etiologic incidence and management of the third, fourth and sixth nerve palsies – *Ophthalmology* 1980, Jan. 87(1) 24 – 43.

Holmes JM. Mutyala S, Maus TL, Grill R, Hodge DO, Gray DT – Paediatric third, Fourth and Sixth nerve palsies: a population – based study. *AJO* 1999 April 127(4) 388 – 92.

Jacobson, DM, MC, Canna TD, Layde DM. – Risk factors for Ischemic ocular motor nerve Palsies – *Arch. Ophthal.* 1994 Jul. 112 (7), 961 – 6.

Juncos JL, Beal MF – Idiopathic cranial Polyneuropathy. A fifteen year experience – *Brain* 1987 Feb. 110(Pt1) 197-211.

Keane JR – Bilateral sixth nerve palsy. Analysis of 125 cases *Ach. Neurol* 1976 Oct.33(10) 681-3.

Keane JR – Fourth nerve palsy: Historical review and study of 215 inpatients – *Neurology* 1993 Dec.43(12) 2439-43.

King AJ, Staury E, Stephnoson G, Tribble RB – Spontaneous recovery rates for unilateral sixth nerve palsies *Eye* 1995 9(Pt4) 476-8.

Kwan ES, Laucella M, Hedges TR 3d, Wolpert SM – A clinico neuroradiologic approach to third cranial nerve palsies – *AJNR Am. J. Neuroradiol.* 1987, May – June 8(3) 459 – 68.

Laine FJ – Cranial Nerves III, IV and VI – *Top may Reason Imagin* 1996 April (2) 111 – 30.

Tiffin PA, MacEwen CJ, Craing EA, Clayton G.- Acquired Palsy of the Oculomotor, trochlear and abducens nerves – *Eye* 1996 10 (pt 3) 377-84.

Liu GT, Volpe NJ, Galetta SL – Eye movement disorders *Curr Opin ophthalmol* 1995 Dec. 6(6) 27 – 33.

Menon V, Singh J, Prakash P – Aetiological pattern of ocular motor nerve palsies. *IJO* 1984, Sep. – Oct. 32(5), 447-53.

Moster ML, Savino PT, Sergott RC, Bosley TM, Schatz NJ – Isolated sixth – nerve palsies in younger adults.

Richards BW, Jones FR, Younge, BR – Causes and Prognosis in 4278 cases of Paralysis of the Oculomotor, trochlear and abducens cranial nerves – *AJO* 1992 May 15 113(5) 489-96.

Rush JA, Younge BR – Paralysis of Cranial nerves III, IV and VI cause of Prognosis in 1000 cases – *Arch.Ophthal.* 1992.Dec.70(6): 721-4.

Trobe JD – Isolated third nerve palsies – *Semin Neurol* 1986, June 6(2), 135-41.

Trobe JD – Isolated Pupil sparing third nerve palsy – *Ophthalmology* 1985, Jan. 92(1) 58-61.

Tsaloumas MD, Willshaw HE: Congenital Oculomotor Palsy: associated neurological and Ophthalmological findings. Eye 1997; 11(Pt.4) 500-3.

Watanabe K, Hagura R, Akanuma Y, Takasu T, Kajinuma H, Kazuya N, Irie M
– Characteristics of Cranial nerve palsies in diabetic patients – Diabetes Res. Clinical Pract 1990 Aug. Sept. 10(1) 19 – 27.

Younge BR, Sutala F – Analysis of trochlear nerve palsies. Diagnosis, etiology and treatment – Mayo Clinic Proc. 1977 Jan. 52(1) 11 – 8.

COIMBATORE MEDICAL COLLEGE HOSPITAL

Department of Ophthalmology

Proforma

“A Clinical Study of Ocular Motor Nerve Palsies”

1. Name: _____
2. IP No: _____ Date: _____
3. Age: _____
4. Sex: a) Male [], b) Female [],
5. Educational Qualification: No formal Education[], Schooling[], UG[], PG and above[]
6. Occupation: Studying [], Govt [], Private [], Own Business [], House wife [], Not working [],
7. Income: _____
8. Address: C/o, _____
Door No: _____
Street: _____
Place: _____
City: _____
PIN: _____ / Phone: _____
9. Referred from: _____
10. Presenting Complaints: _____
11. Presenting Illness:

H/o Diminution of Vision	[<input type="checkbox"/>]
H/o Double Vision	[<input type="checkbox"/>]
H/o Deviation of eyes	[<input type="checkbox"/>]
H/o Drooping of Lids	[<input type="checkbox"/>]
H/o Photophobia	[<input type="checkbox"/>]
H/o Watering	[<input type="checkbox"/>]
H/o Fever	[<input type="checkbox"/>]
H/o Head ache	[<input type="checkbox"/>]
H/o Vomiting	[<input type="checkbox"/>]
H/o Disturbance in Smell	[<input type="checkbox"/>]

- H/o Deviation of Mouth []
- H/o Hearing disturbance []
- H/o Tinnitus []
- H/o Vertigo []
- H/o Difficulty in swallowing []
- H/o Disturbance in taste []
- H/o Loss of Weight []
- H/o Loss of Appetite []
- H/o Bowel and bladder disturbance []
- H/o Trauma []
- H/o Contact with Pet Animals []
- H/o Ear discharge []

12. Past History:
- | | Yes | No | Duration |
|-------------------|-----|-----|----------|
| Diabetes Mellitus | [] | [] | [] |
| Hypertension | [] | [] | [] |
| Tuberculosis | [] | [] | [] |
| Seizure disorder | [] | [] | [] |
| Medical History | [] | [] | |
| Surgical History | [] | [] | |

If Yes, Details: _____

13. Personal History:
- a. Diet: Veg: [] Non Veg: [] Mixed: []
 - b. H/o Consumption of Alcohol: Yes [] No: [] If Yes:
Duration: _____
 - c. H/o Smoking: Yes [] No: [] If Yes:
Duration: _____
 - d. H/o Chewing: Yes [] No: [] If Yes:
Duration: _____
 - e. H/o Drug intake: Yes [] No: [] If Yes:
Duration: _____

14. General Examination :

- a) Consciousness []
- b) Orientation []
- c) Higher functions []
- d) Built: Mild [], Moderate [], Well []
- e) Nourishment: Ill [], Moderate [], Well []
- f) Anaemia []
- g) Jaundice []
- h) Cyanosis []
- i) Clubbing []
- j) Generalized lymphadenopathy []

15. Blood Pressure: _____ / _____ mm of Hg.

16. Pulse Rate: _____ / Min.

17. Eye Examination:

a) Head Posture : Normal: [] Abnormal: []

b) Facial Symmetry : Normal: [] Abnormal: []

c) Eye: : Right Eye: Left Eye:

i. Vision: _____ / _____

ii. With correction: _____ / _____

iii. Eye Brows: _____ / _____

iv. Lids: _____ / _____

v. Conjunctiva: _____ / _____

vi. Cornea: _____ / _____

vii. Anterior Chamber: _____ / _____

viii. Iris: _____ / _____

ix. Pupils:

a. Size: _____ / _____

b. Shape: _____ / _____

c. Reaction to Light:

Direct: _____ / _____

Consensual: _____ / _____

d. Reaction to Accommodation: _____ / _____

- x. Lens: _____ / _____
- xi. Proptosis: _____ / _____
- xii. Nystagmus: _____ / _____

d) Movement of the Eye ball:

- Elevation : _____ / _____
- Depression : _____ / _____
- Abduction : _____ / _____
- Adduction : _____ / _____
- Intorsion : _____ / _____

e) Intra Ocular Pressure: _____ / _____ mm of Hg
Schitz

f) Fundus:

- Media : _____ / _____
- Disc : _____ / _____
- Vessels : _____ / _____
- Macula : _____ / _____
- Other findings : _____ / _____

g) Fields: _____

h) Colour Vision: _____ / _____

i) Diplopia Charting:

j) Hess Chart:

18. Other Systems:

1. Cardio Vascular System: S1 S2 Heard
2. Respiratory System: Normal Vesicular Breath Sounds Heard.
3. Abdomen:

	Yes	No
Soft:	[]	[]
Organomegaly:	[]	[]

	Normal	Abnormal
4. Central Nervous System:		
i. Other Cranial Nerves	[]	[]
ii. Motor System	[]	[]
iii. Sensory System	[]	[]
iv. Extra Pyramidal System	[]	[]
v. Cerebellar Functions	[]	[]
5. Examination of Ear, Nose, Throat:	[]	[]
6. Examination of Thyroid:	[]	[]
7. Examination of Breast:	[]	[]
19. Investigation:		

BLOOD

Sugar :

Hb _{A1C} :

T.C :

D.C :

E.S.R :

VDRL :

Mantoux :

URINE: _____

X-ray Skull: _____

X-ray Orbit: _____

C.T.Brain: _____

C.T.Orbit: _____

M.R.I: _____

Other Investigations:

Referred to: _____

20. Provisional Diagnosis:

21. Treatment: _____

Follow up:

ABBREVIATIONS

RE	- Right Eye
LE	- Left Eye
Dip	- Diplopia
Drop	- Drooping
Def.Vn	- Defective vision
Def.Movt	- Defective movement
H/A	- Headache
IMC	- Immature Cataract
Mat Cat	- Mature Cataract
A	- Acting
NA	- Not Acting
POA	- Primary Optic Atrophy
HT	- Hypertension
Gr.	- Grade
Pap	- Papilloedema
PT	- Pulmonary Tuberculosis
ONH	- Optic Nerve Head
#	- Fracture
FR/PR/NR	- Full/Partial/No recovery
LF	- Lost follow up
SOF	- Superior Orbital fissure
ATT	- Anti tuberculous Treatment
RTA	- Road Traffic Accident
LOC	- Loss of Consciousness
PRE. SYM	- Presenting Symptoms
ASS. SYS.DIS	- Associated Systemic Diseases
F	- Fundus
CT	- Computerised Tomography
DEMYELI	- Demyelination
BDR	- Background Diabetic Retinopathy
BS	- Blood Sugar

MASTER CHART

Sl No.	Age	Gen	Lat	Sym & Duration	Ass.Sys.Dis	V/A	A.S	Pupil	Fundus	Nerves	CNS	ESR-mm/hr	B.S	MX	X-ray	CT/MRI	Followup
1	22	F	RE	Dip-1MT	-	6/12	N	A	N	VI	NAD	12	90	-Ve	-	-	4 months-FR
2	60	M	RE	Drop-3D Pain-3D	DM	6/36	IMC- BE	A	NV	III	NAD	11	240	-Ve	NAD	-	3 months-FR
3	13	M	RE	Dip-1MT	RTA	6/6	N	A	N	VI	NAD	5	-	-	# base of skull	-	4 months-FR
4	15	M	LE	Drop-3D	H/O Injury	6/6	N	NA	N	III	NAD	12	-	-Ve	NAD	NAD	3 months-FR
5	59	M	RE	Dip-1D Def VN- 1D	HT	6/9	N	A	Gr.I HTR	III	NAD	10	100	-Ve	NAD	NAD	4 months-FR
6	53	M	LE	Dip10 D	-	6/6	N	A	N	VI	NAD	8	110	-Ve	NAD	NAD	3 months-FR
7	35	F	BE	Dip H/A15D	PP eclampsia	6/6	N	A	BL. PAP	VI BE	NAD	5	81	-Ve	NAD	Enlarge-Ventricle	3 months-FR
8	11	M	RE	DipH/A-10D	Fever/Vomiting	6/6	N	A	N	VI	NAD	14	60	-Ve	NAD	NS	4 months-FR
9	50	F	LE	Defvn Dip 15D	Nil	6/60	IMC- BE	A	N	VI	NAD	10	74	-Ve	NAD	NS	4 months-FR
10	55	F	LE	Defvn Dip 1 Month	Nil	HM	MC	A	NB	VI	NAD	16	85	-Ve	NAD	NS	4 months-FR
11	60	F	LE	Dro 3yrs	Cataract extraction – 3yrs	6/24	N	A	N	III,IV,VI	NAD	9	90	-Ve	NAD	NS	2 months-LF
12	11	M	LE	Dip- 5D	Fever/ATT – irregular	6/6	N	A	N	VI	NAD	20	69	+Ve	?PT	NS	Ref.toTB Hospital-LF
13	64	M	LE	H/A – Dro Def Movt. 10 D	HT	6/18	IMC	A	Gr-II HTR	III	NAD	14	91	-Ve	NAD	NS	3 months-FR
14	20	M	LE	Dip. H/A – 2Wks	Inj. Cricket Ball.	6/6	N	A	N	VI	NAD	5	78	-Ve	NAD	NS	1 months-FR
15	52	M	RE	Dip. H/A – 2wks	Pain	6/18	N	N	Colobama ONH	VI	NAD	26	82	-Ve	NAD	NS	4 months-FR
16	31	M	LE	Dip. HA -1week	-	6/6	N	A	N	VI	NAD	22	79	-Ve	NAD	NS	2 months-PRLF
17	27	M	LE	Dip. Pain – 5D	-	6/6	N	A	N	VI	NAD	27	84	-Ve	NAD	NS	2 months-FR
18	31	M	BE	Defvn Dip – 2wks	-	6/6	N	A	N	VI BE	NAD	31	82	-Ve	NAD	NS	4 months-FR
19	30	M	LE	Dip HA -1week	HT under trt	6/6	N	A	GrI-HTR	VI	NAD	12	90	-Ve	NAD	NS	3 months-FR
20	5	F	RE	Defvn 3yrs	Marcus Gunn +ve	6/18	N	A	N	Cong -III	NAD	-	-	-	NAD	NS	NR
21	28	M	LE	Dip. HA -1 yrs	Trauma	6/6	N	A	N	IV	NAD	6	81	-Ve	NAD	-	1 moth -LF
22	44	M	RE	Dip. -10D	-	6/12	N	A	N	VI	NAD	31	79	-Ve	NAD	-	3 Month - FR
23	35	F	LE	Drop. 3weeks	Trauma	6/6	N	NA	N	III	NAD	9	95	-Ve	Sphenoditis Mastoiditis	NS	4Month-FR
24	53	M	LE	Dip.pain 2 days	HT Under trt	6/9	N	A	GrI-HTR	VI	NAD	10	106	HS	NAD	NS	1 ½ Months-PR LF
25	49	M	RE	Dip 1 months pain 15D	Ht under trt ? Pontine hge	6/12	N	A	GrI-HTR	VI	NAD	8	95	-Ve	NAD	-	LF
26	20	M	LE	Dip. Pain -3D	-	6/6	N	A	N	III	NAD	12	96	-Ve	NAD	NS	2 months-PR
27	55	F	LE	Dip. 1 week	-	6/60	IMC	A	N	III	NAD	14	108	-Ve	NAD	NS	3 months-FR
28	28	M	RE	Dip. 3 months	RTA with inter hemispheric bleed	6/6	N	A	N	IV	NAD	8	91	-Ve	# R-panetal	-	LF
29	35	M	RE	Dip. 1 month	Inj with LOC.	6/18	N	A	N	VI	NAD	-	-	-	# L Temporal	# L- Temporal & concussion	1 Months-NR
30	37	F	RE	Dip. HA -3 days	-	6/9	N	A	N	VI	NAD	5	94	-Ve	NAD	NS	3Month-FR

31	24	M	BE	Loss of vision 1 year	Known case of Parietooccipital astrocytoma	No PL	RE - N LE - Total Leucoma	NA	RE-SOA	Re - VI Le-III, IV, VI	↓ tone power sensation R side	-	-	-	-	-	Referred to neurology - LF
32	42	F	LE	Dip drop 3 days	-	6/6	N	A	N	III	N	7	74	-ve	NAD	NS	3 months -FR
33	28	F	RE	Pain & Dip 3 weeks	Exanthematous Feve +	6/35	Iridocyclitis	NA	N	III,VI	N	11	97	-ve	NAD	-	3 months -FR
34	35	M	RE	Dip.Drop.H/A 1week	-	6/6 p	N	NA	N	III	N	10	89	-ve	NAD	NS	3 months -FR
35	11	M	RE	Pain /Drop 2 yrs	H/O trauma -RTA 2 Years ago	6/18	N	NA	N	III	N	11	-	-ve	NAD	NS	Advised occlusion LE -LF
36	55	F	LE	Dip. Def movt 20 day	Diabetes & HT	6/12	IMC	A	NPDR	III,IV,VI	N	14	345	-ve	NAD	NS	4 months -FR
37	60	M	LE	Pain drop 10 day	-	6/9	N	A	N	III	N	8	101	-ve	NAD	NS	3 months -FR
38	14	M	BE	H/A 3 day	Vomiting. altered sensorium	6/6	N	A	Hyperemic disc	VI BE	N	-	-	-	-	-	Ref. To neurology emergency LF
39	25	F	LE	Dip. 2 day	-	6/9	N	A	N	VI	N	16	78	-ve	NAD	NS	3 months -FR
40	4	F	LE	Dip .drop.4 day	Fever similar com 6 months ago	6/9	N	NA	N	III	N	12	-	-ve	NAD	NS	2 months -PR LF
41	34	F	RE	Dip.partial drop 10 day	-	6/6	N	A	N	III	N	8	74	-ve	NAD	NS	4 months -FR
42	44	F	RE	Dip -10 day	Diabetes - 10 yrs	6/6	N	A	NPDR	IV	N	18	279	-ve	NAD	NS	4 months -FR
43	36	F	LE	Pain Dip H/A -2 day	-	6/18	N	A	N	VI	N	12	88	-ve	NAD	NS	3 months -FR
44	59	M	RE	Dip 10 day	HT on Examination	6/6	N	A	N	VI	N	14	92	-ve	NAD	NS	2 months -PR LF
45	41	F	LE	Dip. 15 day	-	6/6	N	A	N	VI	N	8	84	-ve	NAD	NS	4 months -FR
46	59	F	RE	Dip 1 week	HT on Examination	6/6	N	A	N	VI	N	16	270	-ve	NAD	NS	3 months -FR
47	30	M	RE	Dip 15 day	-	6/6	N	A	N	VI	N	12	78	-ve	NAD	NS	4 months -FR
48	21	F	LE	Dip 2 week	-	6/6	N	A	N	VI	N	10	88	-ve	NAD	NS	3 months -FR
49	48	M	LE	Dip.1 months	H/O RTA	6/6	N	A	N	VI	N	8	92	-ve	# R Panetal	#R Parietal	4 months -FR
50	40	M	RE	Dip.drop 1 week	-	6/6	N	A	N	III	N	14	84	-ve	NAD	NS	2 months -NR
51	18	F	BE	H/A.Pain def.movt 10 day	Suspect demyeli	6/6	N	A	N	VI, BE	N	20	76	-ve	NAD	-	LF
52	47	F	LE	Drop15 day	-	6/18	N	NA	N	III	N	16	85	-ve	NAD	NS	3 months -FR
53	65	M	RE	Drop 1 week	Diabetes	6/6	N	A	N	III	N	7	180	-ve	NAD	NS	3 months -FR
54	28	M	RE	Dip 1 week	Vomiting	6/6	N	A	N	VI	N	9	92	-ve	NAD	NS	1 month -FR2 months later III N-FR
55	40	F	LE	Dip.H/A Pain -2 months	-	6/12	N	A	N	VI	N	11	87	-ve	NAD	NS	2 months -FR
56	55	M	RE	Dip.Partial drop 1 months	-	6/60	IMC	NA	N	III,IV,VI & VI	N	28	106	-ve	SOF - Penosteal Thick	-	3 months -FR
57	18	F	LE	Drop-from childhood	-	1/60	N	NA	N	III	N	15	75	-ve	NAD	NS	NR- Congenital
58	25	F	LE	Dip.Pain-2 week	-	6/9	N	A	N	VI	N	17	98	-ve	NAD	NS	2 months -FR
59	42	F	LE	H/A drop 1 months	-	6/9	N	NA	N	III	N	15	124	-ve	NAD	NS	3 months -FR
60	33	M	RE	Dip 2 months	H/O RTA	6/6	N	A	N	IV	N	7	80	-ve	-	Contusion R frontal Labe	2 months -FR

61	50	F	LF	Dip. one month	H/O RTA With LOC ans bleeding thro mouth and nose	6/6	N	A	N	VI	N	6	78	-Ve	#R Parletal bone	Contusion R Parietal region	2Months NR LF
62	34	M	RE	Dip. 1 week H/A 2 weeks	Fever 1 week	6/6	N	A	Bilateral Papiledema	VI	N	8	86	-Ve	Erosion of Post Clinoid	-	LF
63	29	M	RE	Pain. Dip 20 days	-	6/9	N	A	N	VI	N	12	74	-Ve	NAD	NS	
64	40	F	LE	H/A dip.pain 1 month	-	6/12	N	A	N	VI	N	20	80	-Ve	NAD	NS	
65	50	M	RE	Dip. 2 weeks	HT of Examination	6/9	N	A	N	VI	N	18	76	-Ve	NAD	NS	
66	23	M	RE	Dip. Pain 1 month	-	6/9	N	A	N	VI	N	12	84	-Ve	NAD	NS	
67	67	M	RE	Dip. One month	HT	6/60	IMC	A	N	VI	N	18	98	-Ve	NAD	NS	
68	49	M	LE	Dip. 1 month	-	6/12	N	A	N	III	N	20	76	-Ve	NAD	NS	
69	45	F	RE	Dip. Pain 3 days	Diabetes	6/36	IMC	A	Temporal pallor	III.IV.VI	N	14	312	-Ve	NAD	NS	
70	69	M	RE	Drop.Dip. 15 days	-	6/18	IMC	A	N	III	N	11	84	-Ve	NAD	NS	
71	18	F	LE	Dip. Pain 1week	-	6/6	N	A	N	VI	N	14	83	-Ve	NAD	NS	
72	13	M	BE	Def.vn 10 days	Fever alt sensonum 10 days	6/18	N	Stug gil	Temporal pallor	VI	Planter I	13	80	-Ve	Erosion of Clinoid Mastoditiad	Hypodense mass in Sellar regin	3 months FR
73	18	F	LE	H/A Left ear ache + discharge 1 month	CSOM left	6/6	N	A	N	VI	N	17	74	-Ve	-	-	4 Months FR
74	10	F	RE	H/A 1 week	H/O Epilepsy once	6/6	N	A	N	VI	N	11	76	-Ve	NAD	NS	3 months FR
75	26	F	RE	Dip. 1 month	Mass R nostril irradiation Given	6/6	N	A	N	VI	N	5	85	-Ve	-	-	3 months FR
76	64	M	LE	Drop.RE 1 week	Diabetes	6/60	IMC	A	N	III	N	5	145	-Ve	NAD	NS	3 months FR
77	57	M	LE	Dip.1 ½ months	Diabetes	6/24	IMC	A	N	VI	N	180	-	-	NAD	NS	10 Days LF NR
78	21	F	RE	Dip.partial drop 2 months	Hypothyroid	6/9	N	A	N	III	N	8	90	-	NAD	NS	4 Months FR
79	25	F	RE	Dip.Def.movt. 10 days	-	6/12	N	A	N	VI	N	12	70	-Ve	NAD	NS	4 Months FR
80	30	M	RE	Def. Movt.10 days	Def.VnRE since childhood	1/60	N	A	Highmyopia	VI	N	7	82	-Ve	NAD	NS	3 Months FR
81	47	F	LE	Def.movt.drop. 4 moths	-	3/60	IMC	Stug gil	N	III.IV.VI	N	25	79	-Ve	Sinus maxillary growth	-	Taken by neurology dept
82	21	M	RE	Dip. 10 days	-	6/6	N	A	N	IV	N	6	92	-Ve	NAD	-	3 Months FR
83	30	M	RE	H/A 5 months Dip. 1 months	-	6/6	N	A	N	VI	N	8	88	-Ve	NAD	-	3 Months FR
84	45	F	RE	Dip. 1 ½ moths	-	6/6	N	A	Temporal pallor	VI	N	6	66	-Ve	NAD	-	? Nasopharyngal TR Referred to ENT
85	15	F	RE	Def. Vn pain 1 week	Case of JNA ref. from ENT	PL	N	A	POA	III & III	N	-	-	-	-	-	3 Months FR
86	21	M	RE	Dip. 2 months	H/o injury ref.from ENT	6/6	N	A	N	IV	N	9	81	-Ve	NAD	-	3 Months FR
87	23	F	LE	Drop.2weeks	-	6/6	N	A	N	VI	N	8	78	-Ve	NAD	-	2 Months FR
88	58	M	RE	Dip.4 days	Diabetes	6/12	N	A	N	VI	N	20	194	-Ve	NAD	-	3 ½ months FR
89	52	M	RE	Drop.pain H/A 1 week	HT	6/12	IMC	NA	GR II HT Changes	III	N	11	87	-Ve	NAD	-	3 Months FR
90	19	M	LE	Partial Drop Det. Movt 3 months	H/O Accident 3 months with LOC	NO PL	N	NA	POA	III	N	6	78	-	-	-	3 months FR NR V/A
91	58	F	RE	Def Mov.10days	Nil	NOP L	N	RAPD	Temporal Pallor	III.IV-III	NAD	16	108	-ve	NAD	-	Ref. to Diabetology LF
92	29	M	RE	Dip.drop.pain 3days	H/O ink RE with stick	6/24	N	Sluggish	Macular Edema	III	NAD	6	86	-	NAD	-	3 months FR

93	19	M	Re	Dip.drop.pain 1month	-	6/6	N	Sluggish	n	III	-	8	90	-	NAD	NS	2 months FR
94	35	M	LE	Dip.2 weeks	-	6/6	N	A	Myopic	VI	NAD	6	86	-ve	NAD	NS	3 months FR
95	54	M	LE	Dip Def.Vn.10 days	HT	4/70	IMC	A	GR II HT Change	VI	NAD	10	92	-	NAD	NS	3 months FR
96	20	M	BE	Dip.21/2 weeks	-	6/6	N	A	N	VI	NAD	17	86	-ve	NAD	NS	1 month pr
97	24	F	RE	Dip 2 weeks	-	6/6	N	A	N	VI	NAD	13	79	-	NAD	NS	3 months FR
98	65	M	LE	Dip-3 Days	Diabetes	6/18	IMC	A	N	VI	NAD	15	192	-ve	NAD	NS	3 months FR
99	60	F	LE	Partial drop. Defmovtpain dip 2weeks	-	6/18	IMC	A	N	III,IV & VI	NAD	13	78	-	SOF -2 thickened periestium	NS	3 months FR
100	39	M	RE	Dip.Def.Vin.2Weeks	-	6/6	N	A	N	IV	NAD	8	86	-ve	NAD	NS	3 months FR
101	65	M	RE	Pain H/A Dip.15 days	Diabetes	5/60	IMCA	A	N	VI	NAD	12	214	-	NAD	NS	3 months FR
102	32	M	RE	Dip.31/2 months	H/O Trauma	6/24	N	A	N	VI	NAD	11	82	-ve	-	III Def. hypodense area R occiput	30 days PR LF
103	23	M	LE	H/A Dip.15 days	-	6/6	N	A	N	VI	NAD	8	88	-ve	NAD	NS	3 months FR
104	42	F	RE	Dip.3 years	Diabetes	6/18	IMC	A	N	VI	NAD	10	218	-ve	NAD	NS	3 months FR
105	27	M	RE	Dip.5 years	H/O Trauma	6/6	N	NA	N	III	NAD	6	83	-ve	Old # Temp bone	# Temp. bone	NR
106	70	M	LE	Dip Partial drop 5 days	-	6/6	N	A	N	III	NAD	16	93	-ve	NAD	NS	3 months FR
107	28	F	RE	Drop. Pain 10 days	-	6/6	N	A	N	III	NAD	13	76	-ve	NAD	NS	3 months FR
108	2	FC	LE	Squinting LE 10days	Fever 20 days	-	N	A	N	III	NAD	8	-	-ve	NAD	NS	3 months FR
109	19	F	BE	H/A dip. Pain 10 days	Fever neck rigidify	6/6	N	A	Papilloedema	VI	NAD	22	79	+ve	NAD	NS	2 months PR on ATT
110	40	F	LE	Dip.10 days	-	6/6	N	A	N	VI	NAD	14	83	-ve	NAD	NS	3 months FR
111	65	M	LE	Dip.1 months	HT	5/60	IMC	A	NO View	VI	NAD	9	94	-ve	NAD	NS	3 months FR
112	63	M	LE	Dip. pain 1 week	Diabetes	6/9	N	A	N	VI	NAD	18	270	-	NAD	NS	3 months FR
113	70	M	LE	Dip.10 days	HT	6/24	IMC	A	GR II Change	VII	NAD	7	82	-	NAD	NS	3 months FR
114	60	F	RE	Complete drop 11/2months	-	6/24	IMC	NA	N	III,IV VI	NAD	6	96	-ve	NAD	Basal Meningoma	NR Ref to Neuro
115	10	F	LE	Def.Vn.Pairtical drop crop 2 months	Post TB meningitis	CF CF	N	NA	POA	III,VI	NAD	-	-	+ve	-	-	PR 4 months on ATT
116	34	M	BE	Dip.4 days	-	6/6	N	A	Papilloedema	VI	-	9	112	+ve	NAD	NS	PR 4 months on ATT
117	62	F	LE	Dip. Pain 15 days	-	6/12	IMC	A	N	VI	NAD	8	82	-ve	NAD	NS	3 months FR
118	6	F	LE	Squinting LE with face turn	-	6/6	N	A	N	VI	NAD	12	-	+ve	NAD	-	Ref. paediatric neurology LF
119	35	F	LE	Drop pain 1 month	-	6/6	N	A	N	III IV Vi	NAD	11	100	-ve	NAD	NS	3 months PR
120	35	M	RE	Drop 15 days	-	6/12	N	A	N	IV	NAD	33	82	-ve	NAD	NS	3 months PR
121	14	F	RE	Dip.1 month	-	6/9	N	A	Temporal pallor	VI	NAD	21	-	-VE	NAD	NS	3 months FR
122	24	F	LE	Dip. 1 ½ months	-	6/6	N	A	N	VI	NAD	18	86	-VE	NAD	NS	1 month FR
123	50	F	BE	Drop.Def.mov.since childhood	-	6/60	IMC	A	Temporal pallor	III, IV, Vi	NAD	-	-	-	-	NS	Congenital - NR
124	46	F	LE	Pain Dip. 1 week	Nil	6/60	IMC	A	N	VI	NAD	24	96	-VE	NAD	NS	3 months FR
125	18	M	RE	Pain Dip. 1 day	-	6/60	N	A	N	VI	NAD	38	74	-VE	NAD	NS	3 months FR
126	70	M	RE	Drop. 10 days	Diabetes	3/60	IMC	NA	BDR	III	NAD	32	182	-VE	NAD	NS	3 months FR
127	49	F	LE	Dip. 4 days	L VII palsy 10 years ago	6/60	IMC	A	N	VI	NAD	20	150	-VE	NAD	NS	3 months FR

128	25	M	RE	Def.Mov.partial drop. 1 month	H/O RTA with LOC 1 month go	6/12	N	Sluggish	N	III	NAD	16	96	-VE	# parietal bone	# parietal bone	3 months PR
129	60	M	RE	H/A drop 1 month	Diabetes	1/60	IMC	NA	N	III	NAD	22	185	-VE	NAD	NS	3 months FR
130	52	M	RE	Drop. H/A 1 week	Diabetes, IHD 1 months	6/18	IMC	A	N	III	NAD	7	185	-	NAD	NS	3 months FR
131	27	M	LE	Dip. 40 days	--	6/6	N	A	N	VI	NAD	7	82	-VE	NAD	NS	3 months FR
132	22	M	RE	Dip. 3 weeks	-	6/9	N	A	N	VI	NAD	12	78	-VE	NAD	NS	2 months PR
133	63	M	RE	Drop. 1 month	Diabetes	5/60	Aphakia	A	N	III	NAD	10	178	-VE	NAD	NS	3 months FR
134	50	F	RE	Partial drop dip. 15 days	HT	6/60	IMC	A	GR IHT Changes	III	NAD	60	140	-VE	NAD	NS	4 months FR
135	28	M	RE	Dip. 10 days	-	6/18	N	A	N	VI	NAD	28	88	-VE	NAD	NS	3 months FR
136	48	M	RE	Dip.pain H/A 2 days	-	6/6	N	A	N	VI	NAD	24	190	-VE	NAD	NS	3 months FR
137	55	M	LE	Dip. 1 week	-	6/12	IMC	A	N	VI	NAD	11	126	-	NAD	NS	3 months FR
138	27	F	RE	Dip.pain H/A 10 days	-	6/6	N	A	N	IV	NAD	14	90	-VE	NAD	NS	2 months FR
139	40	M	LE	Def.vn 3 months	-	6/18	N	NA	N	III	NAD	18	90	-VE	NAD	NS	2 months PR LF
140	68	F	LE	Def.vn 1 week	HT	CF CF	MC	A	No View	VI	NAD	7	94	-VE	NAD	NS	3 months FR
141	7	M	RE	H/A Dip. 1 week	Fever	NC	N	A	N	VI	NAD	24	-	-VE	NAD	NS	3 months FR
142	42	M	LE	Dip. 1 month	H/O Trauma left side of face.	6/6	N	A	N	VI	NAD	9	86	-VE	NAD	NS	3 months FR
143	45	M	LE	Drop. 1 week	H/O Trauma	6/6	N	NA	N	III	NAD	26	140	-VE	NAD	NS	2 months PR
144	50	M	RE	Dip. pain 10 days	Diabetes	5/60	IMC	A	N	VI	NAD	21	168	-VE	NAD	NS	3 months FR
145	60	M	RE	Dip. 10 days	Diabetes	6/6	N	A	N	III	NAD	28	80	-VE	NAD	NS	3 months FR
146	30	F	RE	Dip. 1 ½ months	-	6/6	N	A	N	III	NAD	15	70	-VE	NAD	NS	1 ½ months PR
147	38	F	LE	Dip. H/A 15 days	-	6/9	N	A	N	VI	NAD	25	100	+VE	No PT	NS	3 months FR
148	40	F	LE	H/A dip. pain partial drop 15 days	Diabetes	8/60	IMC	NA	N	III,IV,VI	NAD	40	208	+VE	NAD	NS	3 months FR
149	10	F	BE	Def. mov	-	HM	N	A	Pallor	III,IV,VI	-	-	-	-	-	NS	NR – congenital
150	51	M	RE	Pain 10 days	H/O trauma	N	NA	N	N	III	NAD	28	70	-VE	NAD	NS	3 months PR
151	23	F	BE	Loss OF Visio 1 month	Fever – Encephalitis	No peel	N	NA	Bilateral Papiloedema	VI	NAD	40	75	-VE	NAD	-	NR
152	45	M	RE	Dip. 10 days	-	6/24	IMC	A	N	VI	NAD	17	100	-VE	NAD	NS	3 moths FR
153	30	F	RE	Dip. 1 week	-	6/9	N	A	N	VI	NAD	13	75	-VE	NAD	NS	3 moths FR
154	40	M	BE	Dip. H/A 15 days	-	6/18	N	A	N	VI	NAD	7	185	-VE	NAD	NS	Ref. To Diabetology
155	30	F	RE	Dip. Face turn 10 days	-	6/9	N	A	N	VI	NAD	9	74	-VE	NAD	NS	3 moths FR
156	60	M	LE	Dip. 15 days	Diabetes	6/9	N	A	N	VI	NAD	12	180	-	NAD	NS	3 moths FR
157	1 ½	M	RE	Squinting RE dip. 2 days	H/O Trauma R site of face	NC	N	A	N	VI	NAD	-	-	-VE	NAD	NS	2 moths FR
158	28	M	RE	Head tilt dip 2 years	H/O Head inj. 2 years ago	6/6	N	A	N	IV	NAD	-	-	-	-	NS	NR

159	62	M	RE	Dip. Pain 7 days	Diabetes	6/12	N	A	NPDR	VI	NAD	28	248	-	NAD	NS	3 moths FR
160	29	M	RE	Drop. 2 weeks	-	6/6	N	A	N	III	NAD	28	90	-VE	NAD	NS	2 moths PR LF
161	45	M	RE	Partial drop. Dip. 30 days	H/O fall from bike	6/6	N	Sluggish	N	III	NAD	7	86	-VE	NAD	NS	3 moths PR
162	17	F	BE	Dip. 4 days	Fever with neck stiffness 4 days	6/6	N	A	Hyperemia	VI	NAD	14	69	-VE	NAD	-	Sent neurology
163	41	M	RE	H/A Dip. 1 week	-	6/6	N	A	N	VI	NAD	24	96	-VE	NAD	NS	1 months FR
164	14	F	LE	Dip 10 days	-	6/6	N	A	N	VI	NAD	24	72	-VE	NAD	NS	3 moths FR
165	45	F	LE	Drop. 1 week	Diabetes	6/6	N	A	N	III	NAD	18	212	-VE	NAD	NS	3 moths FR
166	32	F	RE	Drop. 1 month	H/O Trauma Over right eyebrow	6/18 BE	N	A	N	III	N	9	87	-VE	NAD	-	Neurology LF
167	64	M	RE	Complete drop 10 days	-	HM	MC	A	No view	III	N	8	103	-VE	NAD	NS	3 moths FR
168	33	M	LE	Dip. Pain 1 week	-	6/9	N	A	N	VI	N	15	79	-VE	NAD	NS	1 moth PR LF
169	60	M	RE	Dip partial drop 1 week	HT	6/18	IMC	A	N	III	N	6	98	-	NAD	NS	2 moths PR
170	55	M	RE	Dip. 1 week	HT	6/9	N	A	N	VI	N	8	84	-VE	NAD	NS	3 moths FR