"CLINICAL PRESENTATION AND ETIOLOGICAL PATTERN IN PATIENTS WITH OCULAR MOTOR NERVE PALSY IN KOLAR REGION"

By

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Dissertation Submitted to the

SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION AND RESEARCH

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In partial fulfillment
Of the requirements for the degree of

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IN
OPHTHALMOLOGY

Under the Guidance of
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Date: **Signature of the Candidate**

LIST OF ABBREVIATIONS USED

SL. NO.	ABBREVIATION	FULL FORM
1.	BCVA	Best corrected visual acuity
2.	CT	Computed tomography
3.	CN	Cranial nerve
4.	CNP	Cranial nerve palsy
5.	DM	Diabetes mellitus
6.	DOV	Diminution of vision
7.	D	Diopter
8.	HM +	Hand movements present
9.	HIV	Human immunodeficiency virus
10.	HTN	Hypertension
11.	LVH	Left ventricular hypertrophy
12.	LP	Lumbar puncture
13.	MRA	Magnetic resonance angiography
14.	MRI	Magnetic resonance imaging
15.	MLF	Medial longitudinal fasciculus
16.	OMNP	Ocular motor nerve palsy
17.	PPRF	Paramedian pontine reticular formation
18.	RTA	Road traffic accident
19.	SPSS	Sastical program for social science software

20.	SAH	Subarachnoid hemorrhage
21.	SCH	Subconjuctival hemorrhage
22.	SDH	Subdural hemorrhage
23.	UCVA	Uncorrected visual acuity
24.	VDRL	Venereal disease research laboratory test
25.	Yrs	Years

ABSTRACT

TITLE OF THE TOPIC: CLINICAL PRESENTATION AND ETIOLOGICAL PATTERN IN PATIENTS WITH OCULAR MOTOR NERVE PALSY IN KOLAR REGION

NEED FOR THE STUDY: Ocular motor nerve palsies are commonly encountered in clinical practice and usually express an underlying local or systemic disease. The etiology could vary from a simple benign lesion to life threatening neurological disorders; delayed treatment may result in permanent ocular and neurological deficits

The clinical manifestations may differ according to the type and localization of the lesion involving the third, fourth and sixth nerves. There exists scarcity of knowledge on these neuro-ophthalmological conditions which is limited to few of treatment options. Hence we undertook this study to find out the clinical presentation and etiological factors of ocular motor nerve palsies with clinical approach, appropriate investigations and advanced imaging modalities, addressing its role in diagnosis and proper management.

Objectives of the Study:

1) To document the clinical presentation of the ocular motor nerve palsies in Kolar region.
2) To determine the etiological factors of ocular motor nerve palsies.

MATERIAL AND METHODS:

Source of Data: All patients with ocular motor nerve palsies at R. L. JALAPPA HOSPITAL attached to SRI DEVARAJ URS MEDICAL COLLEGE, Tamaka Kolar were taken up for this observational study from the period of december 2013 – august

2015. Patients were asked history in detail and underwent an ophthalmic and neurological examination with appropriate investigations and imaging depending on the necessity for individual case after taking informed consent. The etiology of the ocular motor nerve palsies were classified into categories: ischaemic, traumatic, aneurysmal, neoplastic demyelination, raised intracranial pressure and idiopathic.

The clinical presentation were described in terms of laterality of palsies, single or multiple ocular motor nerve palsies, frequency of involvement of nerves, isolated lesions or associated with other neurological signs & symptoms.

RESULTS The mean age of the patients was 40.68 ±17.43 (range 7 years to 75 years) 60% of male patients and 40% female took part in this study, male to female ratio was 1.4:1.

Most of the patients with cranial nerve palsies were between 31 years and 40 years old. The most common presenting symptom was diplopia affecting 32(43.2%) patients, followed by diminution of vision in 22(29.7%), ptosis 19(25.7%), headache in 19(25.7%), Fever in 9(12.2%).

The frequency of distribution of ocular motor nerve palsy was the 3^{rd} cranial nerve was most commonly affected in 31 patients(41.89%), 4^{th} cranial nerve was involved in 4 patients(5.4%) and 6^{th} cranial nerve was involved in 25 patients(33.78%). Multiple cranial nerve palsy were seen in 14 patients (18.91%).

Neuroimaging (CT, MRI or MRA) was performed for 53 (71.8%) patients and revealed an abnormality in 36 cases (48.6%) which included intracranial tumors, non-neoplastic space occupying lesions, ischemic and demyelinating lesion. Neuroimaging was normal in the remaining 17 patients (23%).

The final diagnosis was classified as traumatic in 27(36.5%), ischemic in 16(21.6%), tumor in 5 (6.8%), demyelinating in 2 (2.7%), inflammatory and idiopathic in 6 (8.1%) cases.

INTERPRETATION AND CONCLUSIONS:

We studied the clinical presentation and etiological pattern of ocular motor nerve palsies in Kolar region, diplopia was the most common symptom at presentation followed by ptosis.

Isolated cranial nerve were more frequently involved in our study among them third nerve was seen in majority of cases and etiology causing third nerve palsy was trauma in most cases, second most common nerve involved was sixth cranial nerve and most cases of 6^{th} cranial nerve palsy were related to systemic disorders such as diabetes mellitus, hypertension, trauma was found to be the major etiological factor for multiple cranial nerve palsies. CT/MRI have been performed in 70% of our cases and the probable diagnosis was made in 49%. Hence proving the usefulness of these imaging modalities in ocular motor nerve palsies.

Ocular motor nerve palsy patients with systemic risk factors like diabetes, hypertension can be managed initially with close observation. Imaging is needed for all those OMNP who do not show improvement after the acute stage >2weeks, or develop neurologic findings, or in those patients without resolution after 12 to 16 weeks.

Keywards – Diplopia, ptosis, diabetes, hypertension, trauma, imaging,neurologic finding

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INTRODUCTION

Ocular motor nerve palsies are commonly encountered in clinical practice and usually express an underlying local or systemic disease. The etiology could vary from a simple benign lesion to life threatening neurological disorders; delayed treatment may result in permanent ocular and neurological deficits.

The muscles of the eye are innervated by the third, fourth and sixth cranial nerves. Ocular motor nerve palsies (OMNPs) may be unilateral or bilateral, may involve one or several nerves at the same time and may be obvious or sub clinical. Lesions in one or more of these cranial nerves results in failure of one or both eyes to move in concert with other eye resulting in characteristic forms of strabismus.

The clinical manifestations may differ according to the type and localization of the lesion involving the third, fourth and sixth nerves. In order to find out the etiology, it is important to carry out a detailed history taking, a careful clinical examination, as well as complementary investigations. To increase the chance of identifying the causes of OMNPs, a close collaboration between different specialists has been recommended.

MRI has clearly shown that brain stem lesions (infarct in nucleus and fasciculus of 3 cranial nerve) mimic as peripheral nerve palsy, the treatment of both of which is entirely different

In this regard, there exists scarcity of knowledge on these neuro-ophthalmological conditions which is limited to few of treatment options. There is also a huge lacunae in the literature in understanding the exact etiology, clinical features and also in utility of advanced neuro-imaging modalities to better delineate these clinical entities.

Hence we undertook this study to find out the clinical presentation and etiological factors of ocular motor nerve palsies with clinical approach, appropriate investigations and advanced imaging modalities, addressing its role in diagnosis and proper management.

OBJECTIVES

Objectives of the study:

1) To document the clinical presentation of the ocular motor nerve palsies in Kolar region.

2) To determine the etiological factors of ocular motor nerve palsies.

REVIEW OF LITERATURE

Cranial mononeuropathies have been recognized for centuries. Until the early and middle part of the twentieth century, most palsies were thought to arise as a result of tumors, trauma, infections or nutritional deficiencies. Syphilis, tuberculosis and vitamin deficiencies were felt to be important causes. The first important pathologic papers on this topic were written by Dreyfus et al[1], and Asbury, et al[2, 3]. The Asbury paper[2], details the histopathology of an 88 year old woman with diabetes who had recovered from third nerve palsy on one side and a pupil sparing third nerve palsy on the other. The postpartum findings were highlighted by a noninflammatory, presumably ischemic, demyelinating focus in the intracavernous portion of the third nerve on the acutely involved side.

There have been many studies done to know the peak incidence of the 3rd, 4th and 6th cranial nerves [28]. The mean age of 41±14 years was found in patients with ocular motor nerve palsy [8] and the age range of 4 months to 71 years with a mean age of 41.61 years were seen in another study which involved sixth nerve palsy cases [9].

Two young female patients, aged 14 and 3.5 years, presented at pediatric ophthalmology section due to diplopia. Both of these girls had suffered 1-2 weeks earlier from otitis media, which had been treated with antibiotics[17]. Mean age of 50.5 years was enrolled in a study that was conducted to know the etiology[18].

Between 1979-1994, among 120 patients of acquired paralysis of cranial nerves of ocular muscles, majority of them were males (84-70.0%) aged 21-40 years (65-54.2%) [7] similarly two thirds (62) of the total patients were male (64.6%) in Richard et al study [4], 81 patients were male and 29 were female of 110 with sixth nerve palsy seen in a tertiary care centre[19].

Diplopia is one of the most vexing problems to confront a physician. When diplopia is binocular, it is commonly seen due to dysfunction of one or more of the ocular motor nerves[20]. Patients with oculomotor, trochlear or abducent nerve palsies mainly complain of binocular double vision, but sometimes merely blurred vision or vertigo might be present. The clinical signs comprise of strabismus, pathologic head posture and disturbed saccades [21].

Ptosis was observed in 41.9% and diplopia in 11 patients as chief complaint [8, 18]. Diplopia was the main neuro-ophthalmic manifestation of patients with intracranial cavernomas[22]. Complete ptosis and full mydriasis was seen in majority of cases with isolated third nerve palsy7.

In Richards BW et al, 96 diplopia patients concerned the ophthalmologist regarding common types and causes in order to develop early and proper management. The result revealed that the common types of diplopia were horizontal, vertical and torsional diplopia respectively[4].

Male patients between the age group of 21-30 years presented with diplopia as main complaint and most common cause of palsies were due to road traffic accidents and the Mwanza JC et al study concluded saying that patients with ocular motor nerve palsy should be carefully examined in close collaboration with other specialists [8].

Among Indian subpopulation, isolated 6th nerve palsy were seen in 44.6%, isolated 3rd nerve palsy in 32.0%, isolated 4th nerve palsy in 6.1% and multiple in 17.3%.[2].

A total of 165 cases, VI nerve palsies accounted for the majority of patients (57%), with IV nerve palsies (21%) occurring more frequently than III nerve palsies (17%) and multiple palsies (5%) [23]. Over a period of 4 years study duration a total of 1050 patients were examined in the Neuroophthalmology clinic, out of which 48 were cases of isolated sixth nerve palsy, which accounted for 4.6% of total clinic attendance[24].

Sixth CNP was most common, followed by third and fourth CNP accounting for 18% of eye movement abnormalities in stroke sub-population[14]**.** In a retrospective study of 412 patients with isolated or combined ocular nerve palsies. Palsies of the oculomotor nerve (41.7%) and of the abducens nerve (40.0%) were more frequent than trochlear nerve (6%). Combined ocular nerve palsies (12.1%) were generally the combinations of the third, and sixth cranial nerve (5.1%) or pareses of all three ocular nerve (4.1%) [25].

The potential risk factors which were studied include diabetes mellitus, hypertension, hypercholesterolemia, coronary artery disease, left ventricular hypertrophy, adiposity,

tobacco use, prior ocular motor nerve palsy and hematocrit appeared to be important determinants of ischaemic ocular motor nerve palsy[9].

In adults, the most common cause of acute ocular motor mononeuropathies were due to microvascular ischemia[4, 5]. Tumour, vascular disease and trauma were the prime considerations when a patient presented with isolated sixth nerve paresis in another study[11].

In cases of isolated fourth nerve paresis, mild frontal head trauma and vascular disease were the most common etiologies[11]. Neoplasm accounted for the largest group, aneurysm was least in Rucker CW et al's study and concluded by saying that sixth nerve was commonly involved when compared to others [26].

Pain was the common feature and can be quite severe especially in the young vasculopathy making it an unreliable method of distinguishing ischemic palsies from more ominous causes[15]. However isolated third nerve palsies can arise in many different circumstances.

Isolated and relative pupil sparing third nerve palsies have also been described in patients with meningioma, metastases, and neurocysticercosis. Severe internal carotid artery stenosis has been reported as manifestations of midbrain stroke or mass lesions[10].

It was seen that surgical excision of Rathke's cleft cyst resulted in near complete resolution of the bilateral 6th nerve palsy[12] Aneurysm of the circle of Willis was found to be

the cause of third nerve paralysis in 11% of the cases [27]. Sinus vein thrombosis is a rare intracerebral complication of mastoiditis presenting with sixth nerve palsy [13].

In 1967, Zorilla and Kozak reported pupil sparing in 16 of 20 patients with oculomotor palsy in association with diabetes mellitus. When the astute physician could recognize the pupil sparing feature of a third nerve palsy and pronounce that this was unlikely to be a compressive or vascular lesion, a patient could be spared an invasive test such as an angiogram. Investigations may be tailored to each patient according to clinical findings and probability of finding a cause, and judicious clinical judgement should be exercised[15].

The primary imaging modality used for patients with TNP is MRI because of its high soft-tissue contrast and ability to show the entire course of the third nerve. CT scanning is limited to patients with spontaneous subarachnoid haemorrhage (SAH) to exclude cerebral aneurysm and in patients of head trauma with suspected skull fracture[34].

Despite the high prevalence of peripheral microvascular ischemia like diabetes mellitus, hypertension, hypercholesterolemia, or coronary artery disease as an etiology, other causes were identified by magnetic resonance imaging (MRI) or computed tomography (CT) scanning in 14% of patients[32].

A patient with an incomplete, third cranial nerve palsy requires MRI/MRA to rule out possible aneurysm localizing to the internal carotid, posterior communicating, posterior

cerebral arteries, or the cavernous sinus. With isolated muscle involvement, the cause is more likely a neoplastic mass in the cavernous sinus or orbit, and less likely an aneurysm [35].

Clinical judgment and detailed history are necessary to determine on a case-by-case basis whether neuroimaging is needed in the initial evaluation of a CN VI palsy, risk factors such as microvascular ischemic disease such as diabetes mellitus and arterial hypertension, patients are managed initially with close observation and medical optimization of the risk factors in concert with the primary care physician[33].

CLINICAL ANATOMY

OCULOMOTOR NERVE CN. III

This is the largest of the cranial nerves and supplies all the extraocular muscles except the lateral rectus and superior oblique muscles. Sympathetic motor fibres to sphincter papillae and ciliaris muscle are also supplied by this nerve through its connections with the ciliary ganglion.

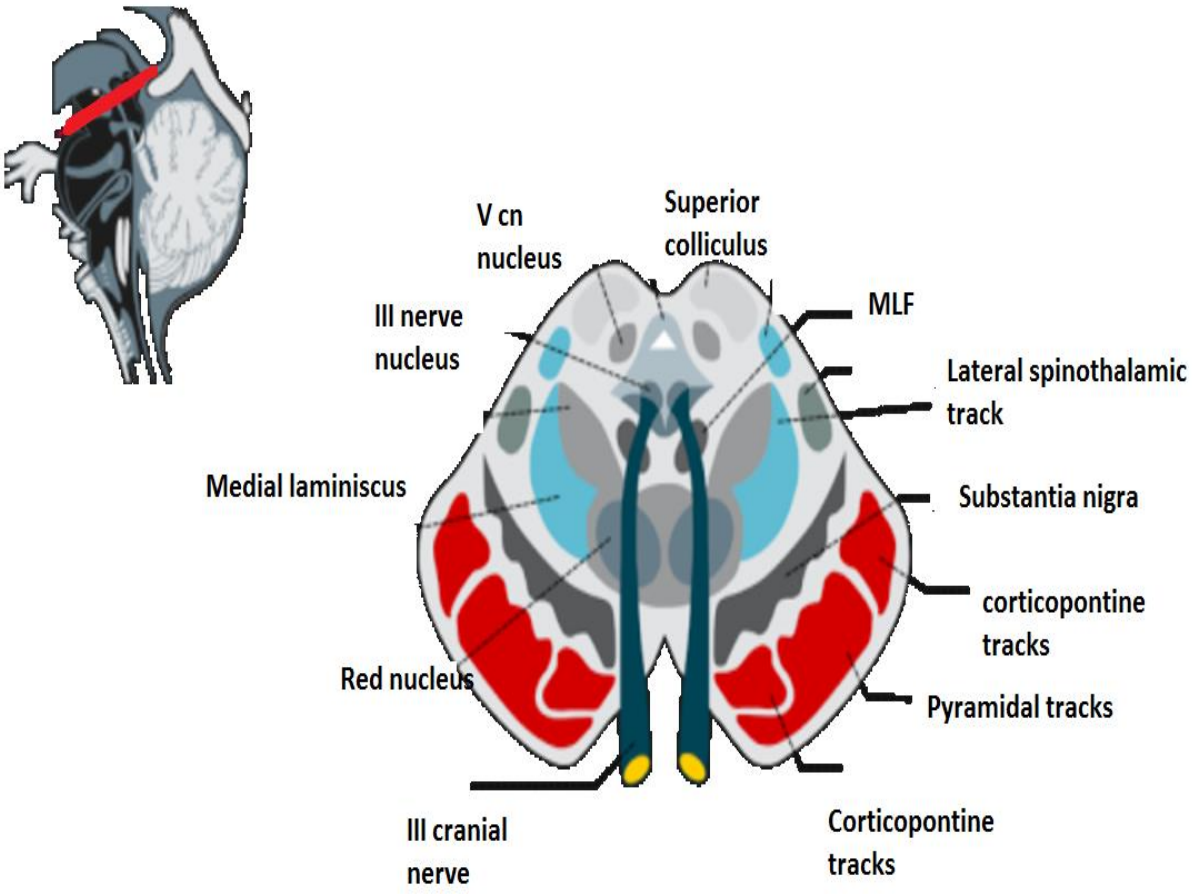
The oculomotor nuclei lies at the level of the superior colliculus in the ventral region of the periaqueduct grey matter and extends cranially for a short distance into the floor of the third ventricle. The medial longitudinal fasciculus lies lateral to the nucleus and contains axons of internuclear neurons that pass ventrally between brainstem nuclei of III, IV, and VI nerves. The oculomotor nerve nuclei consists of two main types: (1) a complex of five individual motor (somatic efferent) nuclei containing the cell bodies of the multipolar motor neurons whose axons directly innervate their respective extraocular muscles; and (2) a general visceral efferent nucleus, the Edinger-Westphal nucleus containing preganglionc parasympathetic neurons.

The fibers emerge from the oculomotor nuclei, pass anteriorly through the tegmentum of the midbrain and red nucleus, and emerge medial to the cerebral peduncle at the upper border of the pons.

In the **Intracranial and intracavernous course** the nerve passes forward, laterally and slightly downward in the interpenduncular fossa lateral to the posterior communicating artery. It passes between the posterior cerebral artery (above) and superior cerebellar artery

(below). It groves the posterior clinoid process and courses forward before it passes through the dural roof of the cavernous sinus. The nerve runs in the upper part of the cavernous sinus and enters the intraconal space of the orbit through the superior orbital fissure within the tendinous ring where it divides into superior and inferior divisions.

In the **Intraorbital course** the superior division (smaller) supplies the superior rectus, which it pierces to reach levator palpabrae superioris. The inferior division (larger) splits into several branches which supplies the medial rectus and inferior rectus, and a long branch passes forward on the lateral aspect of the inferior rectus to reach inferior oblique. It is from this latter branch that the stout motor root passes to the ciliary ganglion, the site of postganglionic parasympathetic neurons. Axons from the postganglionic neurons travel in the short ciliary nerves to supply the choroid, sphincter papillae of iris, and the ciliary muscle.



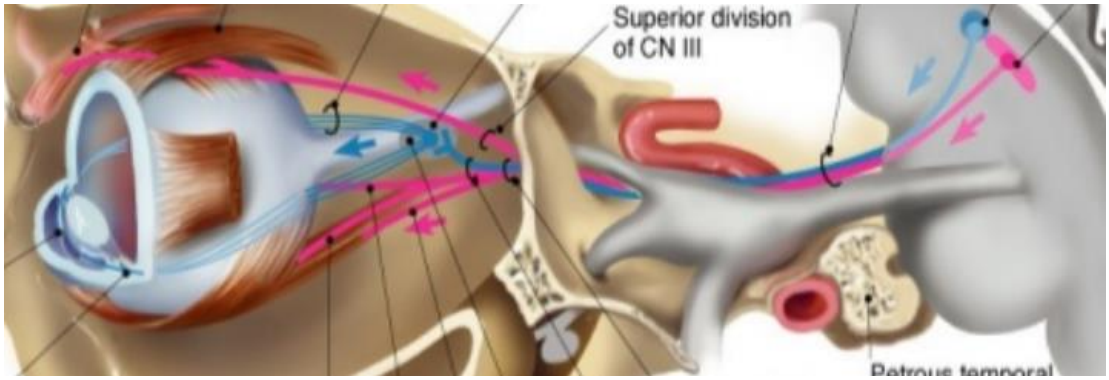


Fig – 1: Course of Oculomotor nerve

Signs - Right 3rd nerve palsy is characterized by the following

a. Weakness of the levator causing profound ptosis, due to which there is often no diplopia.

b. Unopposed action of the lateral rectus causing the eye to be abducted in the primary position. The intact superior oblique muscle causes intorsion of the eye at rest which increases on attempted downgaze.

c. Normal abduction because the lateral rectus is intact.

d. Weakness of the medial rectus limiting adduction.

e. Weakness of superior rectus and inferior oblique, limiting elevation.

f. Weakness of inferior rectus limiting depression.

g. Parasympathetic palsy causing a dilated pupil associated with defective accommodation.

h. Partial involvement will produce milder degrees of ophthalmoplegia

Applied anatomy

Nuclear complex – Levator caudal midline structure which innervates both levator muscles. Lesions confined to this area will therefore give rise to bilateral ptosis.

Superior rectus subnuclei are paired: each innervates the respective contralateral superior rectus. A nuclear 3rd nerve palsy will spare the ipsilateral, and affect the contralateral, superior rectus.

Medial rectus, **inferior rectus** and **inferior oblique subnuclei** are paired and innervate their corresponding ipsilateral muscles. Lesions confined to the nuclear complex are relatively uncommon. Involvement of the paired medial rectus subnuclei cause a wall-eyed bilateral internuclearophthalmoplegia (WEBINO), characterized by exotropia, defective convergence and adduction. Lesions involving the entire nucleus are often associated with involvement of the adjacent and caudal 4th nerve nucleus.

Fasciculus

The fasciculus consists of efferent fibres which pass from the 3rd nerve nucleus through the red nucleus and the medial aspect of the cerebral peduncle. They then emerge from the midbrain and pass into the interpeduncular space. The causes of nuclear and fascicular lesions are similar, except that demyelination may affect the fasciculus.

1 Benedikt syndrome involves the fasciculus as it passes through the red nucleus and is characterized by ipsilateral 3rd nerve palsy and contralateral extrapyramidal signs such as hemitremor.

2 Weber syndrome involves the fasciculus as it passes through the cerebral peduncle and is characterized by ipsilateral 3rd nerve palsy and a contralateral hemiparesis.

3 Nothnagel syndrome involves the fasciculus and the superior cerebellar peduncle and is characterized by ipsilateral 3rd nerve palsy and cerebellar ataxia.

4 Claude syndrome is a combination of Benedikt and Nothnagel syndromes.

Basilar

The basilar part starts as a series of 'rootlets' which leave the midbrain on the medial aspect of the cerebral peduncle, before coalescing to form the main trunk. As the nerve traverses the base of the skull along its subarachnoid course unaccompanied by any other cranial nerve, isolated 3rd nerve palsies are commonly basilar. The following two are important causes:

- Aneurysm of the posterior communicating artery, at its junction with the internal carotid artery
- Head trauma, resulting in extradural or subdural haematoma, may cause a tentorial pressure cone with downward herniation of the temporal lobe.

Intracavernous

- Diabetes
- Pituitary apoplexy

Intracavernous pathology such as aneurysm, meningioma, carotid-cavernous fistula and granulomatous inflammation (Tolosa–Hunt syndrome) may all cause 3rd nerve palsy. Because of its close proximity to other cranial nerves, intracavernous 3rd nerve palsies are usually associated with involvement of the 4th and 6th nerves and the first division of the trigeminal nerve.

Intraorbital

- Superior division innervates the levator and superior rectus muscles.
- Inferior division innervates the medial rectus, the inferior rectus and the inferior oblique muscles, pre-ganglionic parasympathetic fibres from the Edinger–Westphal subnucleus, innervate the sphincter pupillae and the ciliary muscle.

Lesions of the inferior division are characterized by limited adduction and depression, and a dilated pupil. Both superior and inferior division palsies are commonly traumatic or vascular etiology.

CAUSES OF OCULOMOTOR NERVE PALSIES.

1 Nuclear

- Demylination
- Infarctiono Metastatic tumors

2 Fascicular

- Demylination
- Infarction
- Tumor

3 Interpeduncular

- Trauma
- Aneurysm o Meningitis

4 Cavernous sinus

- Carotid –venous fistula
- Granulomatousinflammation (Tolosa hunt syndrome)
- Intracavernous aneurysm o Extrasellar extension of pituitary tumour Meningioma
- Sphenoid sinus carcinoma o Metastatic tumor
- Herpes zoster

5 Orbit

- Trauma
- Tumor
- Pseudotumor

6 Miscellaneouo Cyclic Oculomotorpalsy (Beielschowsky syndrome)

- Migraine , Polyneuritis (Guillain – Barre – Fisher syndrome)

TROCHLEAR NERVE CN. IV

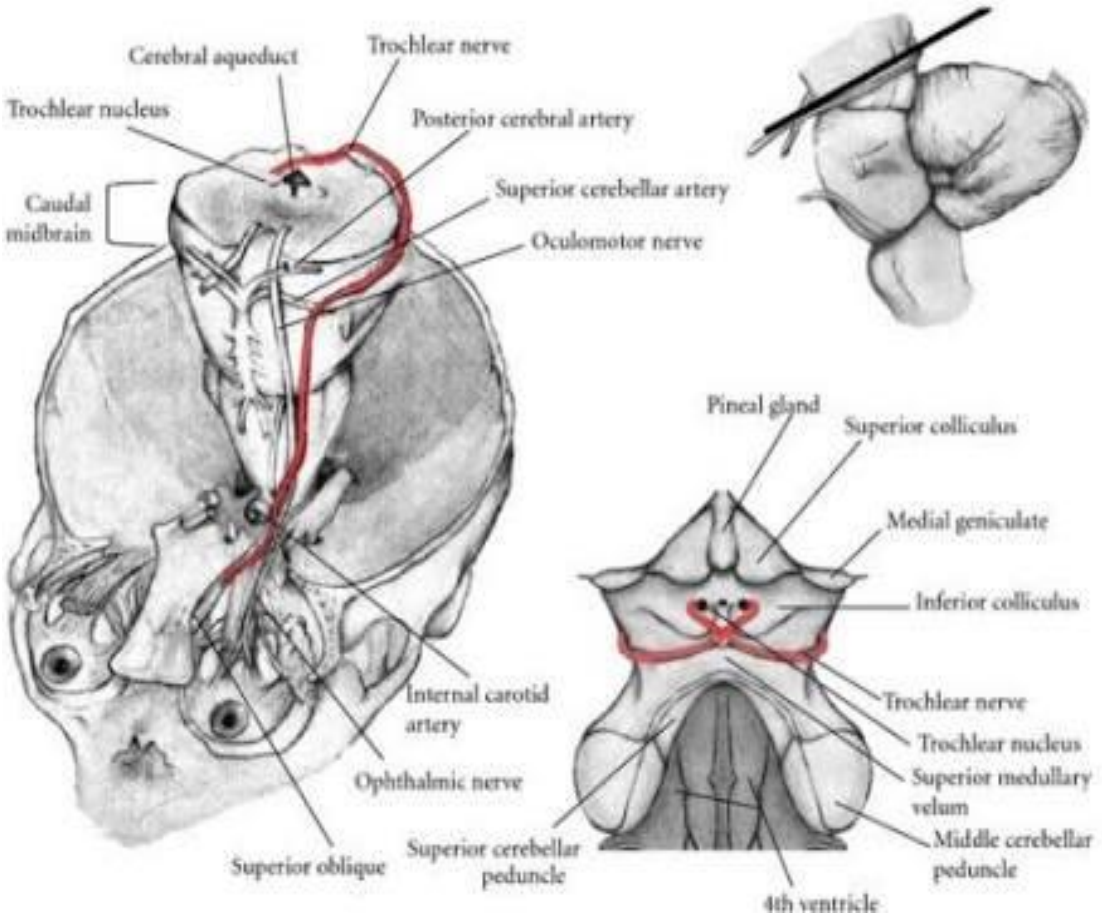
The **Trochlear nerve** is the most slender of cranial nerves [2], it supplies the superior oblique muscle. The nucleus lies in the anterior part of the periaqueductal grey matter at the level of the inferior colliculus in line with the other oculomotor nuclei.

The fibres pass anteriorly and laterally towards the tegmentum before turning and passing posteriorly around the priaqueductal grey matter and into the superior medullary velum where they decussate before emerging from the posterior surface of the brainstem in the posterior craninal fossa.

In the **Intracranial and intracavernous course** the trochlear nerve winds around the crus of the midbrain above the superior cerebellar artery and the pons and below the posterior cerebral artery. It continues anteriorly immediately beneath the
free edge of the tentorium cerebella. It pierces the dura and enters the lateral wall of the cavernous sinus beneath the oculomotor nerve. The trochlear nerve then passes upwards, thus coming to lie above the oculomotor nerve before entering the orbit outside the tendinous ring in the lateral part of the superior orbital fissure.

In the **intraorbital course** it passes medially and passes above the origin of superior palpebral superioris and finally enters the orbital surface of superior oblique.
In the superior orbital fissure it occasionally gives off a branch to the lacrimal nerve. Its gives off a recurrent branch which backward between the layers of the tentorium crebelli and

divides into two or three filaments which may be traced as far as the of the transverse sinus.



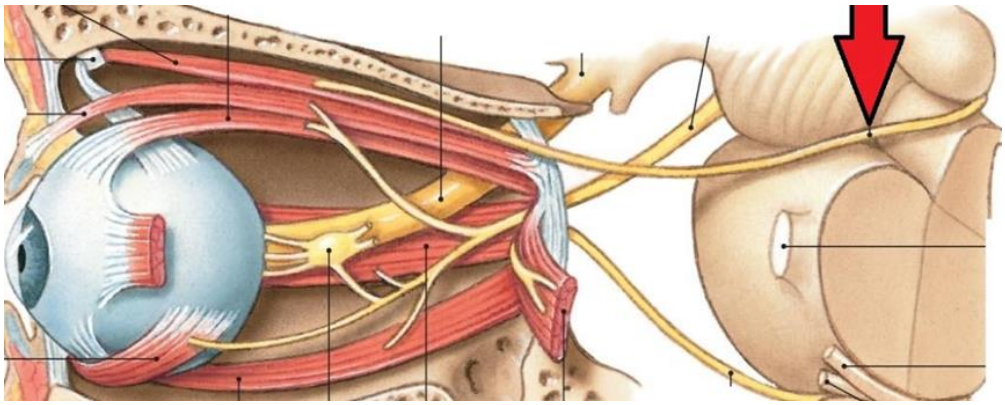


Figure 2- Course of trochlear nerve

Signs

Acute onset of vertical diplopia in the absence of ptosis, combined with a characteristic head posture, strongly suggests 4th nerve disease. The features of nuclear, fascicular and peripheral 4th nerve palsies are clinically identical, except that nuclear palsies produce contralateral superior oblique weakness.

Left 4th nerve palsy is characterized by the following.

a Left hypertropia (‘left-over-right’) in the primary position.

b Increase in left hypertropia on right gaze due to left inferior oblique overaction.

c Limitation of left depression on adduction.

d Normal left abduction.

e Normal left depression.

f Normal left elevation.

CAUSES OF TROCHLEAR NERVE PALSY

- Traumatic (bilateral)
- Vascular mononeuropathy
- Diabetic
- Decompensated congenital paresis
- Posterior fossa tumour
- Neurosurgical procedure
- Herpes zoster

ABDUCENT NERVE CN. VI

Lateral rectus is supplied by the **Abducent nerve.**

The Abducent nucleus lies in the midpons beneath the floor of the upper part of the fourth ventricle, close to the midline beneath the facial colliculus.the nucleus is surrounded by the looping fibers of the facial nerve (genu) and is adjacent to the PPRF and the MLF. The nucleus contains both primary motor neurons and interneurons that cross to the contralateral MLF to reach the third nerve nucleus.

The motor axons exiting the sixth nerve nucleus travel ventrally and slightly laterally, medial to the superior olivary nucleus, to exit on the ventral surface of the caudal pons. As the fascicles pass through the brain stem, they lie adjacent to the spinal tract of the trigeminal nerve and traverse the corticobulbar tracts. Exiting the brainstem, the nerve runs rostrally within the subarachnoid space on the surface of the clivus from the area of the cerebropontine angle to the posterior superior portion of the posterior fossa.

The nerve pierces the dura approximately 1cm below the petrous apex and travel beneath the petroclinoid ligament to enter **Dorello's canal**. Within the canal the nerve travels with the inferior petrosal sinus.

In the **Cavernous sinus** it runs parallel to the horizontal segment of the carotid artery. It is the only cranial nerve within the substance of the cavernous sinus. It is also joined for a short segment by branches of the sympathetic chain, which have been within the wall of the intrapetrous carotid artery.

Reaching the anterior portion of the cavernous sinus, CN VI traverses the **Superior orbital fissure** through the annulus of zinn laterally to enter the medial surface of the lateral rectus muscle.

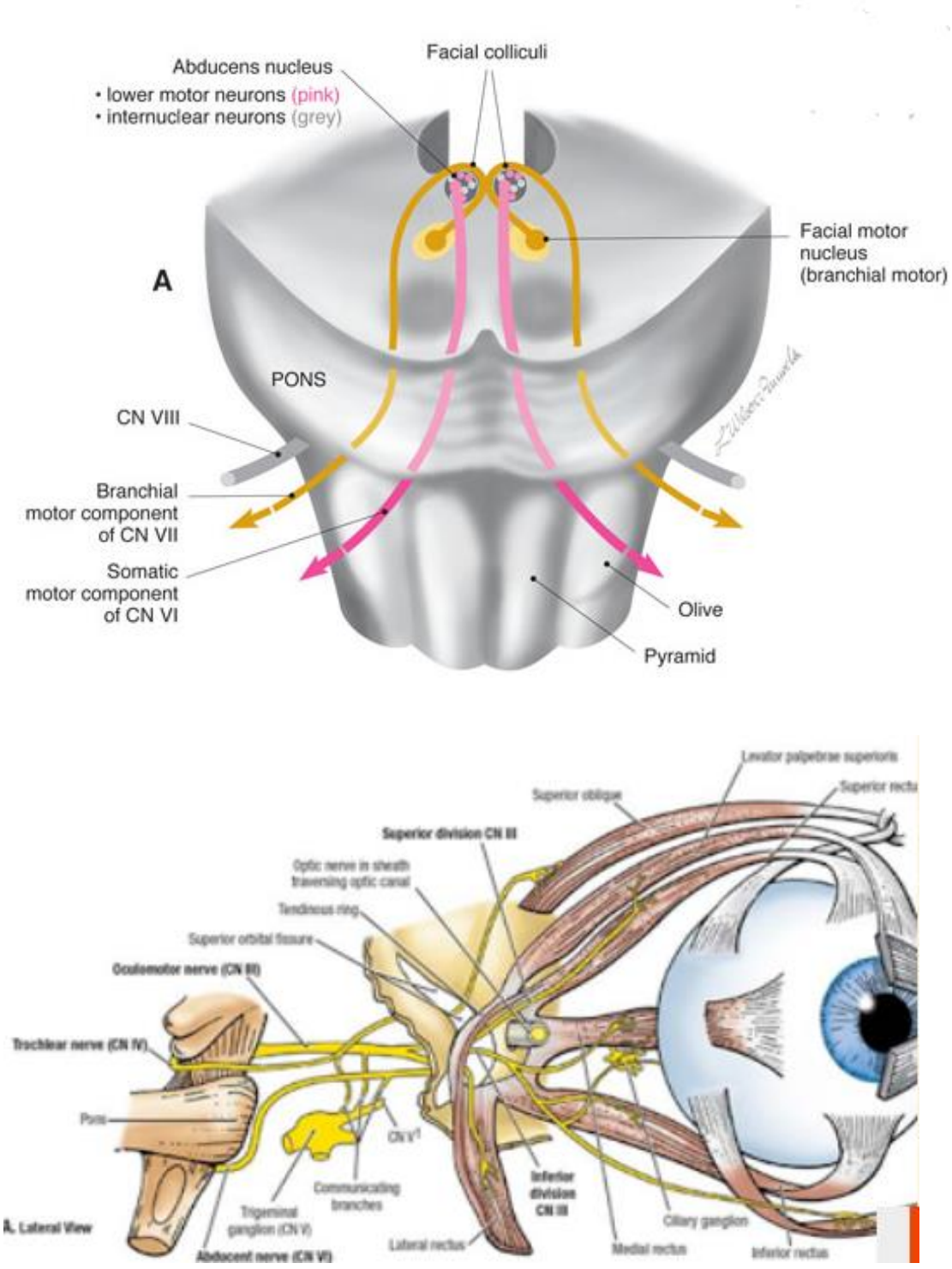


Fig – 3: Course of Sixth Cranial Nerve

Signs: **Acute left 6th nerve palsy**.

a Left esotropia in the primary position.

b Marked limitation of left abduction.

Long-standing left 6th nerve palsy.

a Left esotropia in the primary position due to unopposed action of the left medial rectus. The deviation is characteristically worse for a distant target and less or absent for near fixation.

b Marked limitation of left abduction due to weakness of the left lateral rectus.

c Normal left adduction.

Applied anatomy: The nucleus of the 6th (abducens) nerve lies at the mid-level of the pons, ventral to the floor of the 4th ventricle, where it is closely related to the horizontal gaze centre. The fasciculus of the 7th nerve curves around the abducent nucleus and produces an elevation in the floor of the 4th ventricle Isolated 6th nerve palsy is therefore never nuclear in origin. A lesion in and around the 6th nerve nucleus causes the following sign.

Ipsilateral weakness of abduction as a result of involvement of the 6th nerveFailure of horizontal gaze towards the side of the lesion resulting from involvement of the horizontal gaze centre in the PPRF.

Ipsilateral lower motor neuron facial nerve palsy caused by concomitant involvement of the facial fasciculus is also common.

Fasciculus

The fasciculus passes ventrally to leave the brainstem at the pontomedullary junction, just lateral to the pyramidal prominence.

1 **Foville syndrome** involves the fasciculus as it passes through the PPRF and is most frequently caused by vascular disease or tumours involving the dorsal pons. It is characterized by ipsilateral involvement of the 5th to 8th cranial nerves and central sympathetic fibres.

- 5th nerve – facial anaesthesia.
- 6th nerve palsy combined with gaze palsy (PPRF).
- 7th nerve (nuclear or fascicular damage) – facial weakness.
- 8th nerve – deafness.
- Central Horner syndrome.

2 **Millard–Gubler syndrome** involves the fasciculus as it passes through the pyramidal tract and is most frequently caused by vascular disease, tumours or demyelination. It is characterized by the following:

• Ipsilateral 6th nerve palsy.

• Contralateral hemiplegia (since the pyramidal tracts decussate further inferiorly, in the medulla, to control contralateral voluntary movement).

• Variable number of signs of a dorsal pontine lesion.

Basilar

The basilar part enters the prepontine basilar cistern, passing through or around the inferior petrosal sinus, through the Dorello, to enter the cavernous sinus

1 Acoustic neuroma may damage the 6th nerve at the pontomedullary junction should be emphasized that the first symptom of an acoustic neuroma is hearing loss and the first sign diminished corneal snsitivity.

2 Nasopharyngeal tumours may invade the skull and its foramina and damage the nerve during its basilar course.

3 Raised intracranial stretch the 6th nerve over the petrous tip between its point of emergence from the brainstem and its point of entry into the cavernous sinus. In this situation 6th nerve palsy, which may be bilateral, is a false localizing sign.

4 Basal skull fracture may cause both unilateral and bilateral palsies.

5 Gradenigo syndrome, most frequently caused by mastoiditis or acute petrositis.

Intracavernous

This part more prone to damage than the other nerves. Occasionally, an intracavernous 6th nerve palsy is accompanied by a postganglionic Horner syndrome. The causes of intracavernous 6th nerve and 3rd nerve lesions are similar.

Intraorbital part enters the orbit through the superior orbital fissure within the annulus of Zinn to innervate the lateral rectus muscle

CAUSES OF ABDUCENS NERVE PALSY

Non localizing

- Increased intracranial pressure
- Head trauma
- Intracranial hypertension
- Vascular hypertension
- Spinal anaesthesia
- Parainfectious processes (middle ear infection in children, post viral)
- Diabetes
- Basal meningitis

Localizing

- Cerebropontine angle lesions o Aucostic neuroma o Meningioma
- Pontine syndrome

o Ipsilateral horizontal gaze palsy o Contralateral hemiplegia

- Clivus lesions o Clivuschondroma

o Nasopharyngeal carcinoma

- Cavernous sinus / superior orbital fissure

o Tumour o Inflammation o Aneurysm

- Middle fossa disorders

o Tumous

o Inflammation of medial aspect of petrous

- Carotid – venous or Dural arteriovenous fistula

The intraorbital part enters the orbit through the superior orbital fissure within the annulus of Zinn to innervate the lateral rectus muscle.

SYMPTOMS AND HISTORY

The classical presenting symptoms of patients with abnormality in third, fourth and sixth cranial nerve dysfunction are diplopia, drooping of eyelid, inability to move the eyeball or less frequently, awareness of an enlarged pupil or blurred monocular vision for near.

Onset of diplopia is almost always characterized by patients as sudden in onset. It may be intermittent or may be perceived as blurring of binocular vision. A proper history taking helps to predict the nature and localization of the lesion, point to be asked during history taking include monocularity versus binocularity, direction of separation of images (horizontal, vertical, diagonal, torsional), direction of gaze in which there is greatest separation, change in near versus distant viewing and diurinal variation should be asked.

Abnormality of abducens nerve is mostly likely cause of horizontal double vision, especially if it is worse for distant vision than for near vision, and gets worse on lateral gaze. Trochlear nerve lesions typically manifests as vertical or diagonal diplopia that is worse with near viewing. Ptosis may indicate onset of oculomotor nerve palsy. If ptosis is complete, diplopia may not be recognized. On lid elevation the person may perceive vertical or horizontal diplopia.

MATERIALS AND METHODS

TITLE OF THE STUDY: **CLINICAL PRESENTATION AND ETIOLOGICAL PATTERN IN PATIENTS WITH OCULAR MOTOR NERVE PALSY IN KOLAR REGION**

SOURCE OF DATA:

All patients with ocular motor nerve palsies at R. L. JALAPPA HOSPITAL attached to SRI DEVARAJ URS MEDICAL COLLEGE ,Tamaka Kolar were taken up for this observational study from the period of December 2013 – August 2015.

SAMPLE SIZE:

A total number of 74 patients with ocular motor nerve palsy were selected for the study

INCLUSION CRITERIA:

All conscious patients of ocular motor nerve palsies, isolated III, IV or VI cranial nerve or multiple.

STATISTICAL ANALYSIS

Data were analyzed using the stastical program for social science (SPSS) software. Comparison of variables were done by Chi-Square test. $p<0.05$ will be considered significant.

TECHNIQUES OF EXAMINATION

Patients with paralytic squint who fulfilled the above criteria were enrolled in the study and details regarding their name, age, sex, symptoms, and its duration and any change in the symptoms between their presentation and their onset have been recorded. Detailed history regarding the incidences that preceded the onset of symptoms like trauma [trivial or severe], headache fainting attacks, numbness, etc., was taken.

Past history of any previous episodes of similar nature and the treatment given for the same has been noted. History of systemic illnesses like hypertension, diabetes-mellitus, thyroid abnormalities, and seizure disorder and previous neurological involvement in any other disorders like tuberculosis, syphilis were noted.

History specific to ocular complaints such as double vision, blurring of vision, field defects, and vestibular complaints like vertigo, tinnitus, ear discharge have been recorded.

Personal history regarding smoking, alcohol intake and diet pattern were asked and recorded.

Ophthalmic examination included visual acuity assessment with snellen chart, anterior segment evaluation by slit-lamp biomicroscope, fundus by ophthalmoscopy.

Ocular movements both uniocular and binocular were examined in all cardinal gazes. Amount of deviation of eye were noted and diplopia charting was done. Otorhinolaryngologic examination were performed as and when required.

A complete haemogram, urine analysis for albumin, sugar, and deposits, blood sugar VDRL, Mantoux test, X-ray of skull (both anteroposterior and lateral) and paranasal sinuses has been done in patients as and when indicated . CT-scan was done in almost all trauma cases and in other cases whenever needed. MRI-scan was done depending on necessity of individual case. Certain special tests like icepack test has been done in certain cases to diagnose and to differentiate myasthenia gravis from nerve palsies

Follow up of these cases has been done at the end of 4 weeks, 8weeks, 12weeks, and 6 months.

The etiology of the ocular motor nerve palsies were classified into different categories like ischemic, traumatic, aneurysmal, neoplastic, demyelinating , raised intracranial pressure and idiopathic.

The clinical presentation of ocular motor nerve palsies were described in terms of laterality of palsies, single or multiple ocular motor nerve palsies, frequency of involvement of nerves, isolated lesions or associated with other neurological signs & symptoms.

OBSERVATION AND RESULTS

A prospective, observational study titled Clinical presentation and etiological factors in patients with ocular motor nerve palsy in Kolar region was conducted at R.L.Jalappa hospital. A total of 74 patients presented with cranial nerve palsies. There were 44 males and 30 females and the male to female ratio was 1.4:1. The mean age of the patients was 40.68 ±17.43 (range 7 years to 75 years)

The age and gender distribution of the study subjects is as presented in Table 1.

Most of the patients with cranial nerve palsies were between 31 years and 40 years.

Table 1: Age distribution of patients studied

Age in years	No. of patients	%
<10	4	5.4
11-20	6	8.1
21-30	14	18.9
31-40	15	20.3
41-50	13	17.6
51-60	11	14.9
61-70	10	13.5
>70	1	1.4
Total	74	100.0

Graph 1-Age distribution of the study

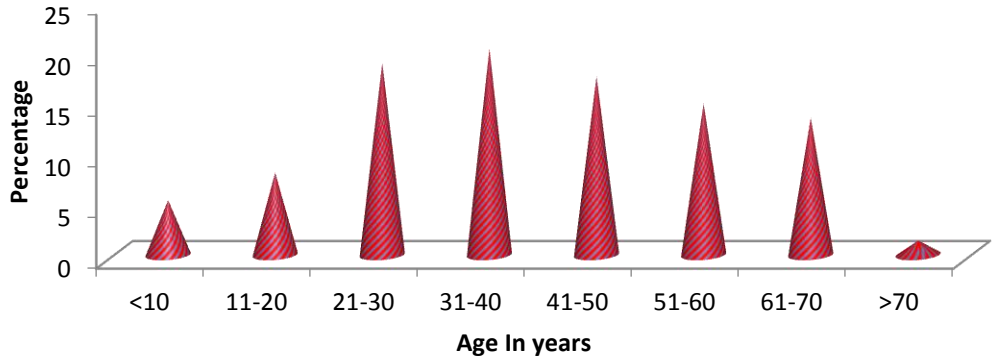


Table 2: Sex distribution of the study

Gender	No. of patients	%
Female	30	40.5
Male	44	59.5
Total	74	100.0

Graph 2- Sex distribution of the study

Male accounted for 59.5% and rest were females

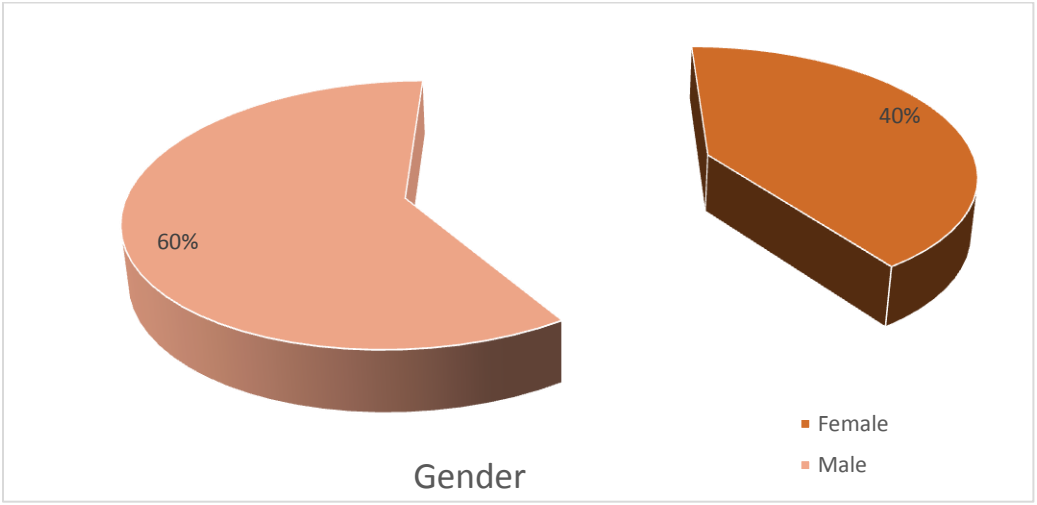
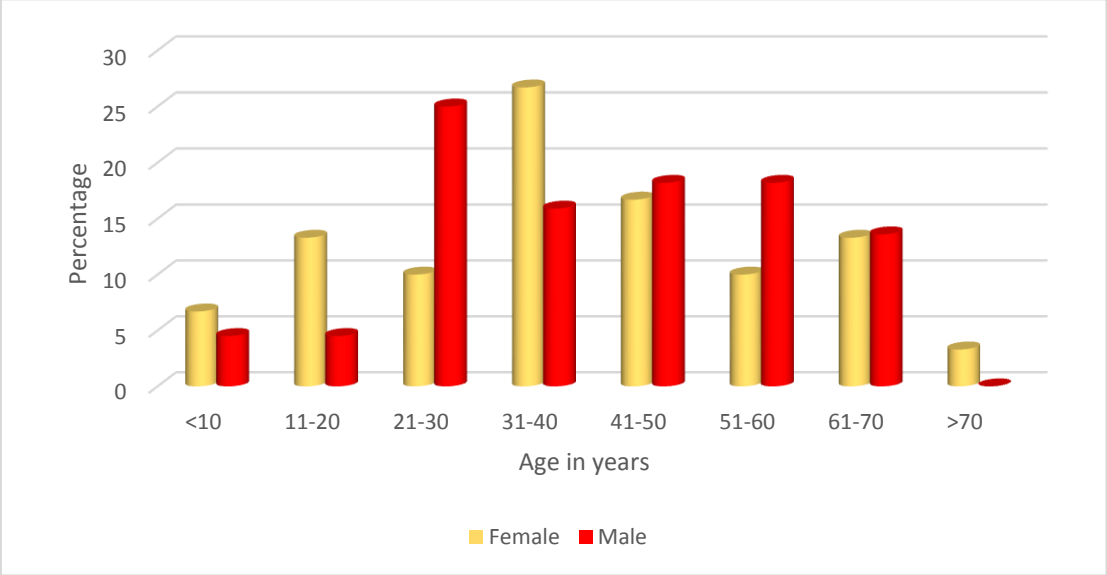


Table 3-Age and Sex distribution of the study

Age in years	Gender		Total
	Female	Male	
<10	2(6.7%)	2(4.5%)	4(5.4%)
11-20	4(13.3%)	2(4.5%)	6(8.1%)
21-30	3(10%)	11(25%)	14(18.9%)
31-40	8(26.7%)	7(15.9%)	15(20.3%)
41-50	5(16.7%)	8(18.2%)	13(17.6%)
51-60	3(10%)	8(18.2%)	11(14.9%)
61-70	4(13.3%)	6(13.6%)	10(13.5%)
>70	1(3.3%)	0(0%)	1(1.4%)
Total	30(100%)	44(100%)	74(100%)

Graph 3-Age and Sex distribution of the study



Graph 4 and Table 4 – Chief complaints of the study

The most common symptom presented was diplopia seen in 32(43.2%) patients; this was followed by diminution of vision in 22(29.7%), drooping of upper eyelid in 19(25.7%), headache in 19(25.7%), fever in 9(12.2%).

Chief complaint	Gender		Total (n=74)
	Female (n=30)	Male (n=44)	
Double vision.	14(46.7%)	18(40.9%)	32(43.2%)
Drooping of eyelids	5(16.7%)	12(27.3%)	17(23%)
DOV	13(43.3%)	9(20.5%)	22(29.7%)
Fever	6(20%)	3(6.8%)	9(12.2%)
Headache	10(33.3%)	9(20.5%)	19(25.7%)
Pain in eye	1(3.3%)	6(13.6%)	7(9.5%)
Deviation of the eye	2(6.7%)	4(9.1%)	6(8.1%)

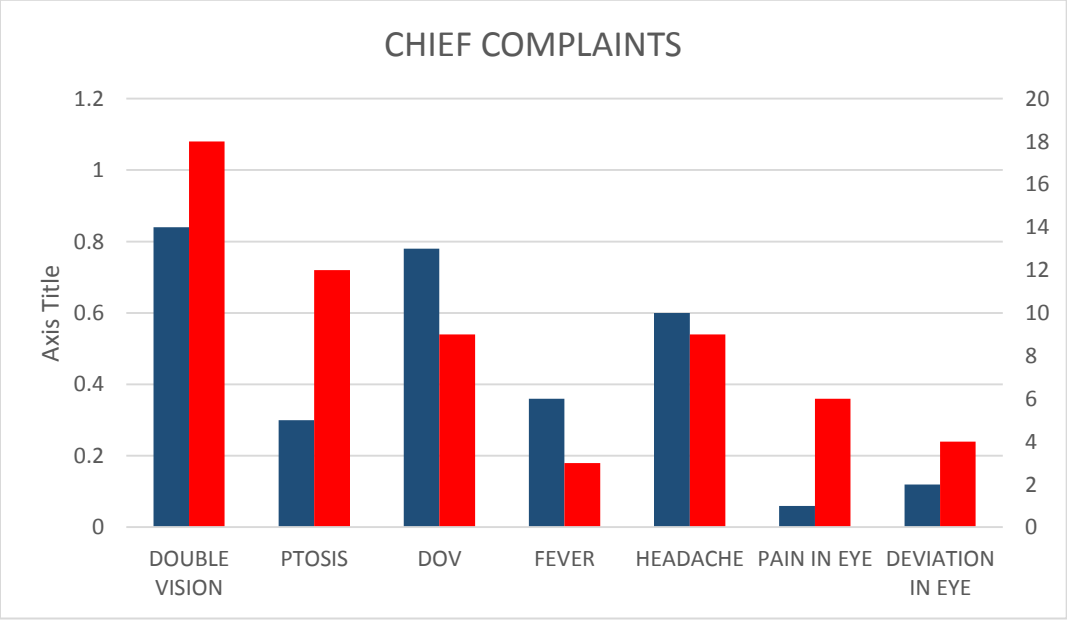
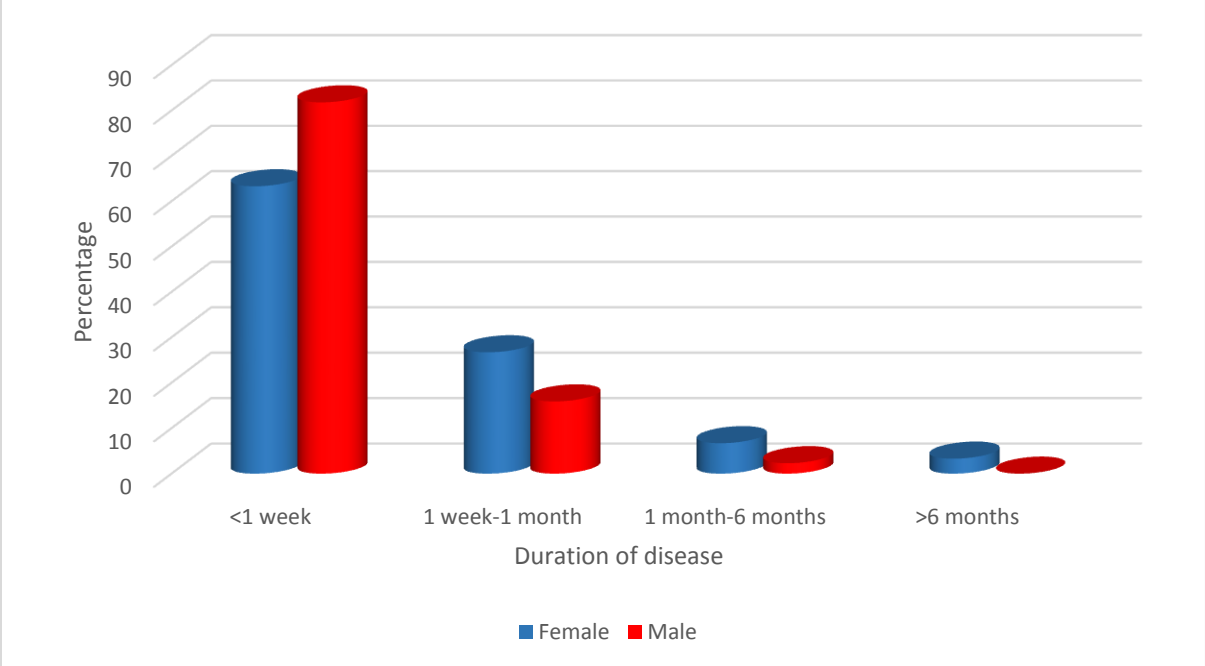


Table 5- Duration at presentation of the study

Most of the patients in our study presented with in one week after onset of symptoms.

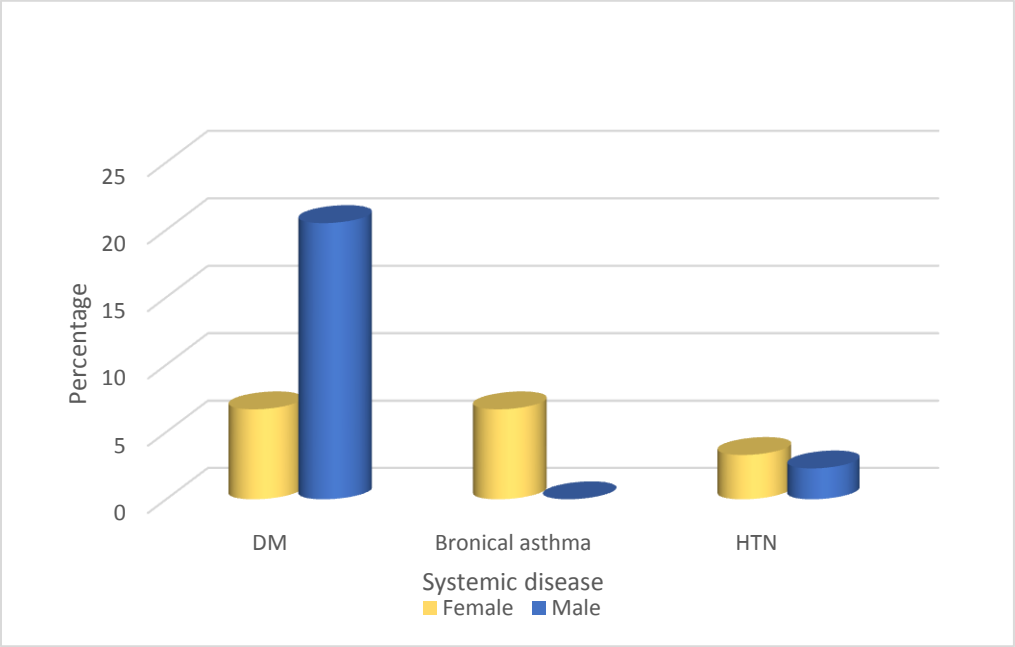
Duration of disease	Gender		Total (n=74)
	Female (n=30)	Male (n=44)	
<1 week	19(63.3%)	36(81.8%)	51(74.4%)
1 week-1 month	8(26.7%)	7(15.9%)	15(20.3%)
1 month-6 months	2(6.7%)	1(2.3%)	3(4.1%)
>6 months	1(3.3%)	0(0%)	1(1.4%)
Total	30(100%)	44(100%)	74(100%)

Graph 5- Duration at presentation of the study



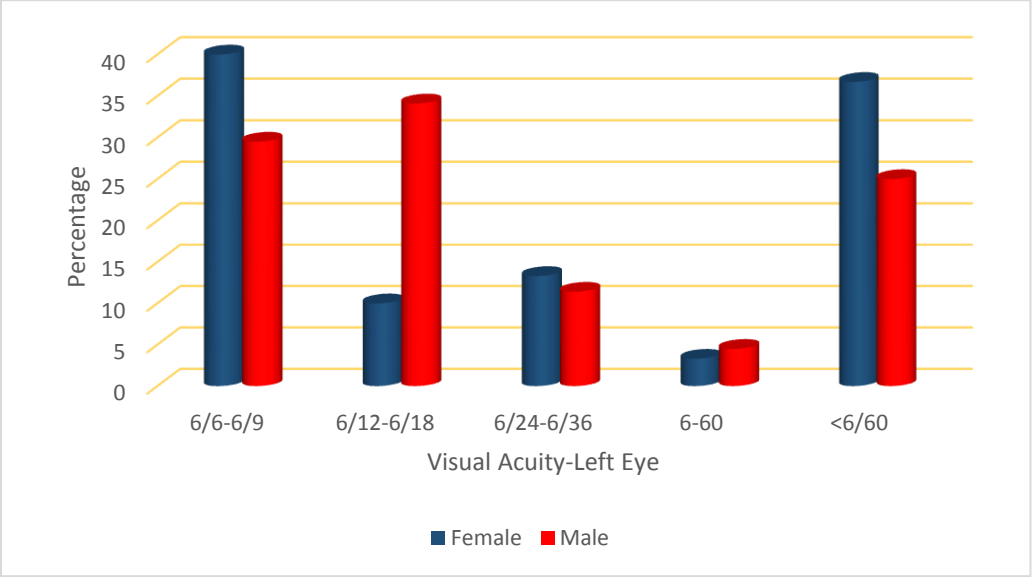
Graph 6- Systemic association of the study

The relationship between the Underlying disorders / systemic association with cranial nerve palsy included diabetes mellitus 11 patients (14.9%), systemic hypertension 2 patients (2.7%) and bronchial asthma 2 patients(2.7%)



Graph 7 – Visual acuity at presentation of the study.

Visual acuity at presentation was normal (VA = 6/6) in 99 eyes (66.89 %), impaired (VA <6/18) in 14.86 % (*N* = 22) eyes and 27 eyes (18.24%) were having low vision (VA = <3/60).



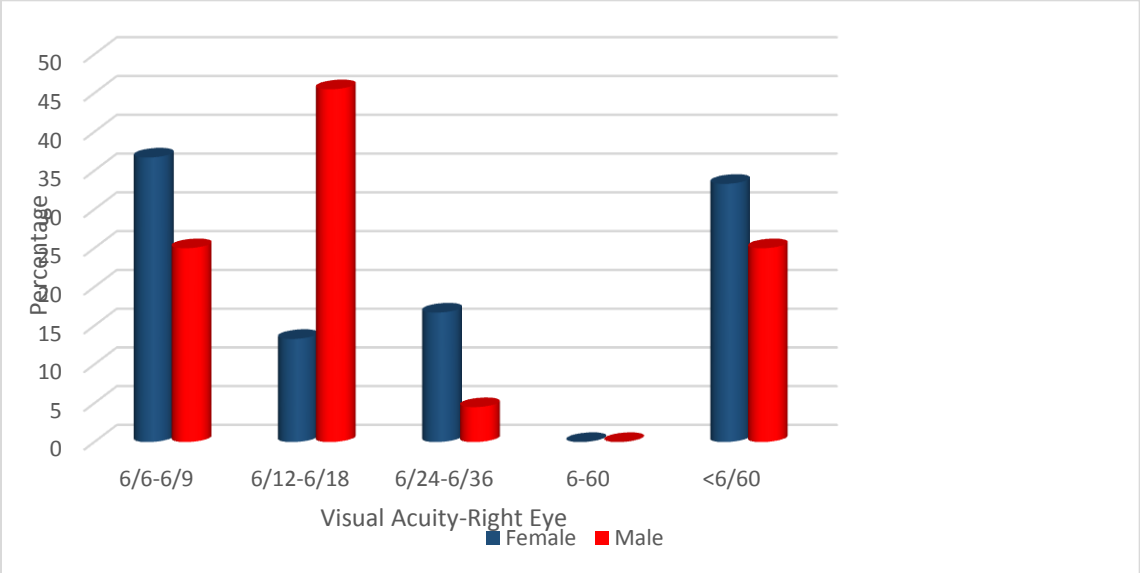
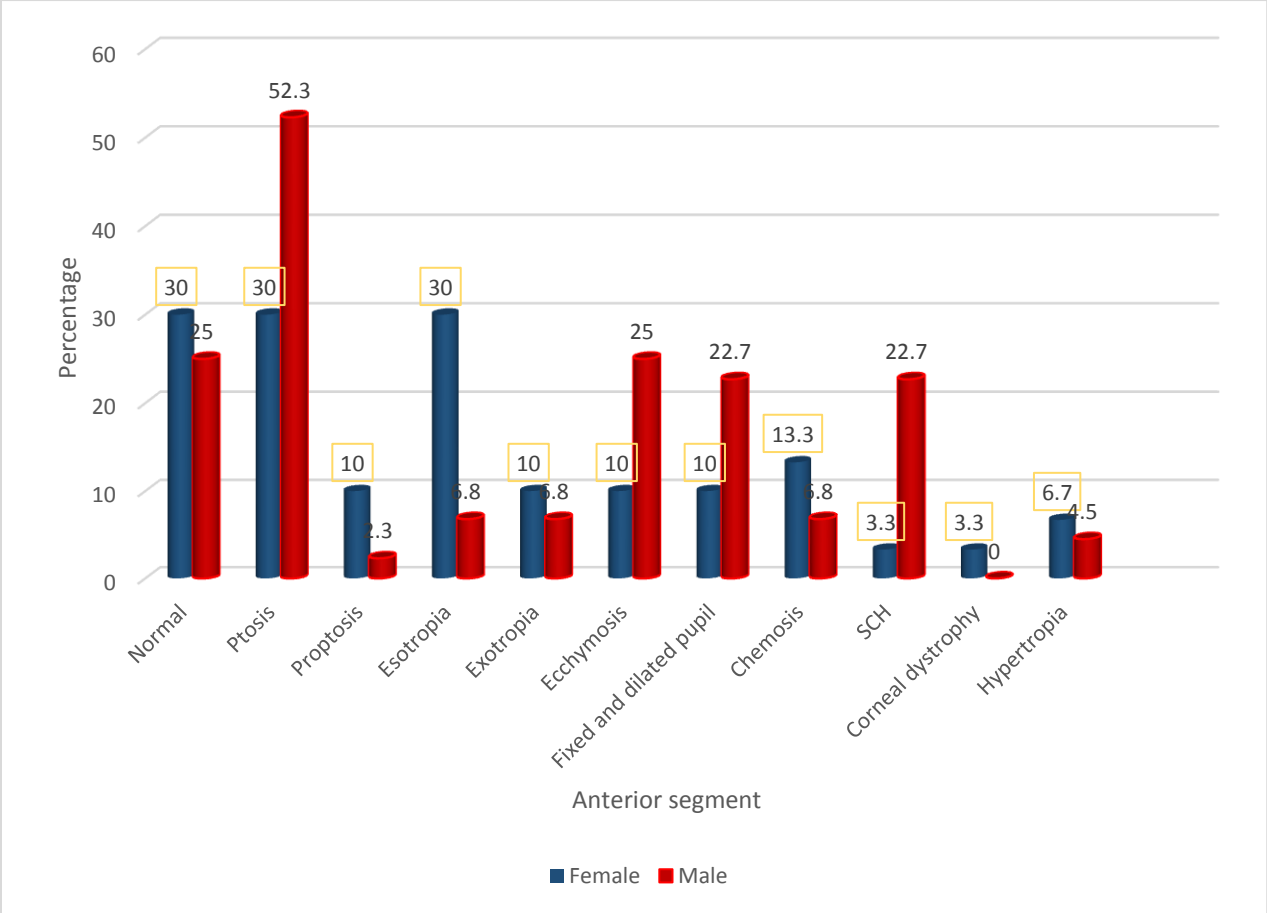


Table 6 - represents the ocular findings.

Among the patients studied thirty two patients (43.2%) had ptosis, six patients(8.1%) had exotropia, twelve patients(16.2%) had esotropia, four patients(5.4%) had proptosis, fixed and dilated pupil was seen in 13 patients(17.6%), hypertropia was seen in 4 patients (5.4%), ecchymosis, chemosis and subconjuctival hemorrhage were seen more associated with trauma history in 14, 7 and 11 patients respectively.

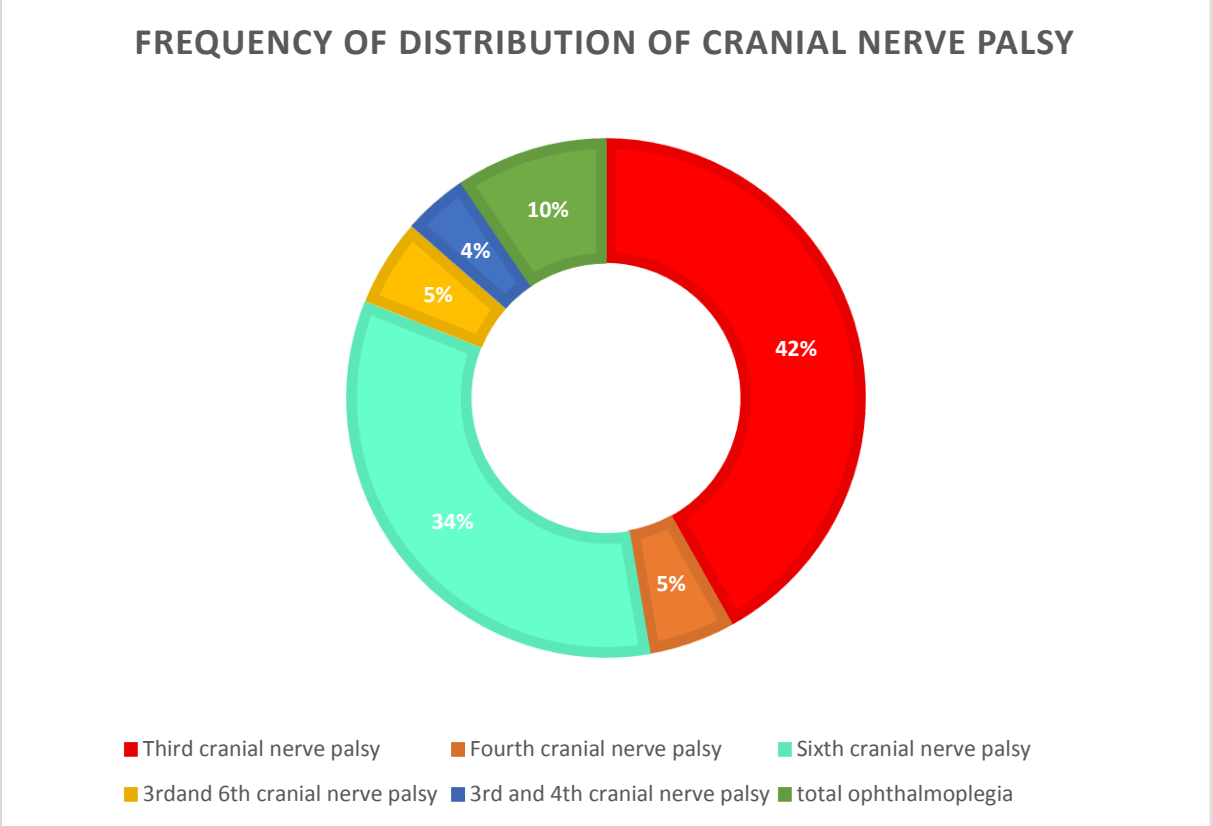
Ocular findings	Gender		Total (n=74)
	Female (n=30)	Male (n=44)	
Normal anterior segment	9(30%)	11(25%)	20(27%)
Ptosis	9(30%)	23(52.3%)	32(43.2%)
Proptosis	3(10%)	1(2.3%)	4(5.4%)
Esotropia	9(30%)	3(6.8%)	12(16.2%)
Exotropia	3(10%)	3(6.8%)	6(8.1%)
Ecchymosis	3(10%)	11(25%)	14(18.9%)
Fixed and dilated pupil	3(10%)	10(22.7%)	13(17.6%)
Chemosis	4(13.3%)	3(6.8%)	7(9.5%)
SCH	1(3.3%)	10(22.7%)	11(14.9%)
Corneal dystrophy	1(3.3%)	0(0%)	1(1.4%)
Hypertropia	2(6.7%)	2(4.5%)	4(5.4%)

Graph 8- Anterior segment findings at presentation of the study



Graph 9 - Represents the frequency of distribution of cranial nerve palsy.

The 3rd cranial nerve was affected in 31 patients, 4th cranial nerve was involved in 4 patients and 6th cranial nerve was involved in 25 patients .The 3rd and 6th cranial both nerves were affected in 3 patients, 3rd and 6th cranial nerves both involvement were observed in 4 patients, all the three cranial nerves (3rd, 4th, 6th) were involved in 7 patients.



Most cranial nerve palsies were associated with normal pupillary reaction. Only 30 patients were associated with pupil involvement. Patients with 3rd and multiple cranial nerve palsies had sluggishly reactive pupils and in some pupil was dilated and fixed.

Graph 10 - Laterality of cranial nerve palsy

Right eye was involved in majority of the patients accounting for 48(64.9%) and left eye in 31(41.9%) and both eye was involved in 6 patients(8.1%).

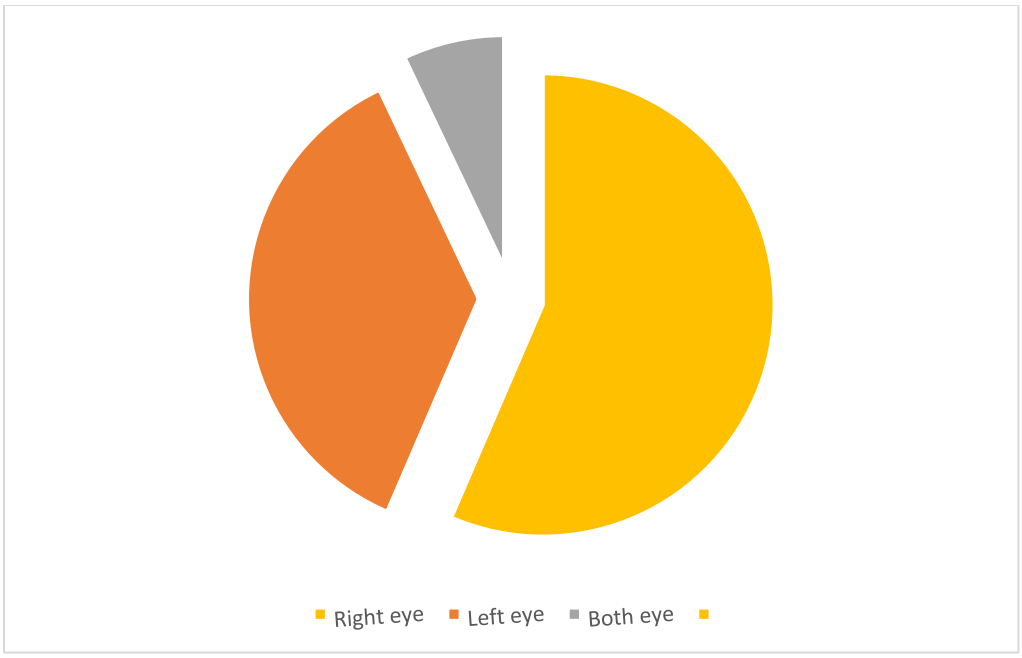
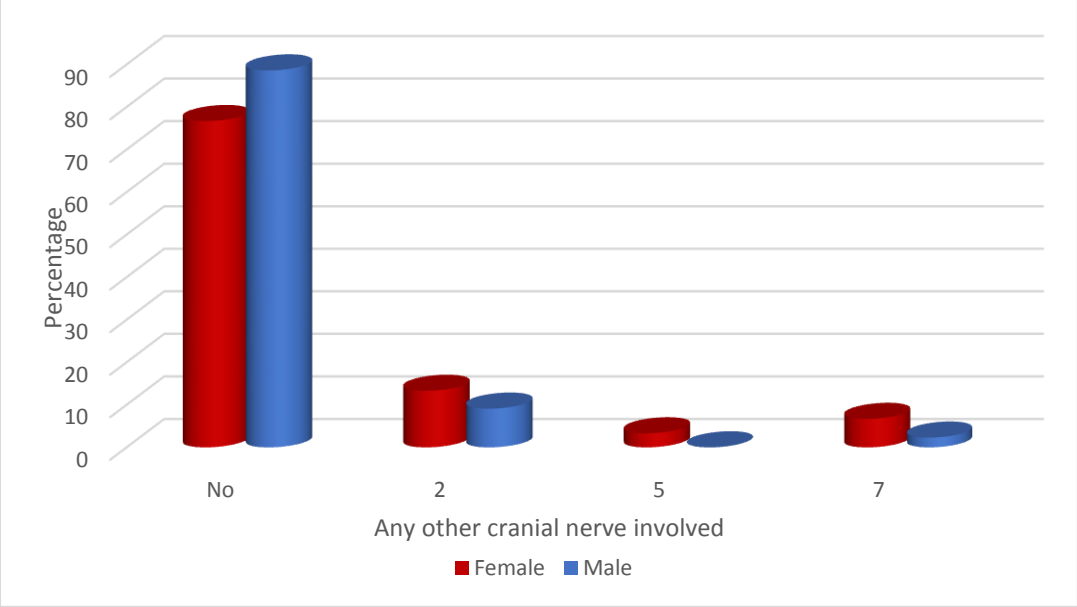


Table 7: Other cranial nerve involvement

Ocular motor nerve palsy associated with other cranial nerve involvement at the time of presentation include the following - optic nerve was involved in 8 patients(10.8%) and these patients had associated vision loss , trigeminal nerve was involved in one patient (1.4%) and facial nerve in 2 patients (2.7%).

Any other cranial nerve involved	Gender		Total
	Female	Male	
No	23(76.7%)	39(88.6%)	62(83.8%)
2	4(13.3%)	4(9.1%)	8(10.8%)
5	1(3.3%)	0(0%)	1(1.4%)
7	1(2.3%)	1(2.3%)	1(1.4%)
8	-	1(2.3%)	1(1.4%)
Total	30(100%)	44(100%)	74(100%)

Graph 11 – Other cranial nerve involvement



Graph 12 – Fundus changes of the patients in the study.

Fundus examination appeared normal in 53 cases (71.6%) and abnormal in 21 cases (28.4%) which include papilloedema, primary optic atrophy, secondary optic atrophy, diabetic & hypertensive retinopathy.

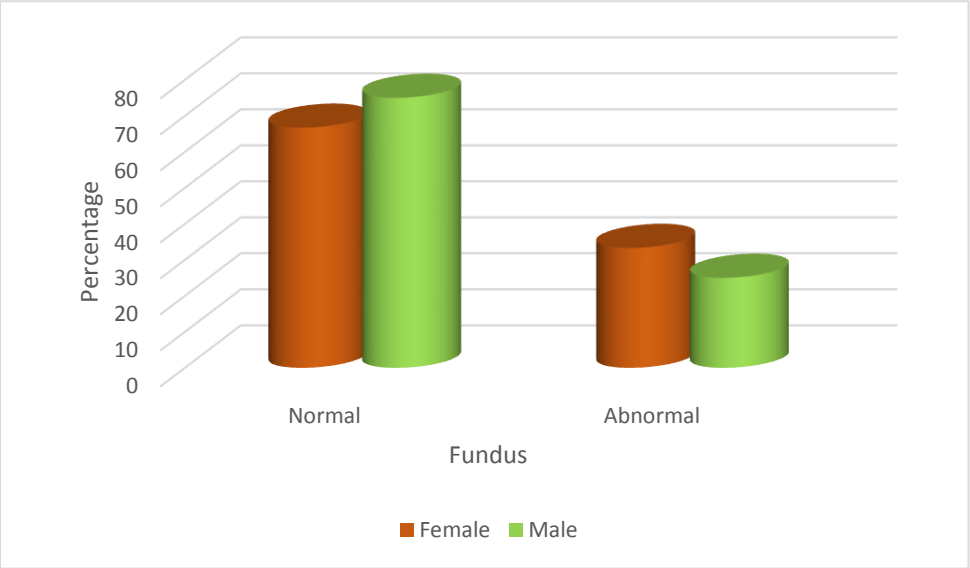


Table 8 –Abnormal fundus findings of the study

Fundus	Gender		Total (n=74)
	Female (n=30)	Male (n=44)	
Papilledema	2(6.7%)	3(6.8%)	5(6.8%)
Primary Optic atrophy	1(3.3%)	1(2.3%)	2(2.7%)
Diabetic retinopathy	2(6.7%)	5(11.4%)	7(9.5%)
Cherry red spot	0(0%)	1(2.3%)	1(1.4%)
Hypertensive retinopathy	1(3.3%)	1(2.3%)	2(2.7%)
Secondary optic atrophy	0(0%)	1(2.3%)	1(1.4%)
Vasculopathic	1(3.3%)	0(0%)	1(1.4%)

Graph 13– Abnormal Fundus findings of the study

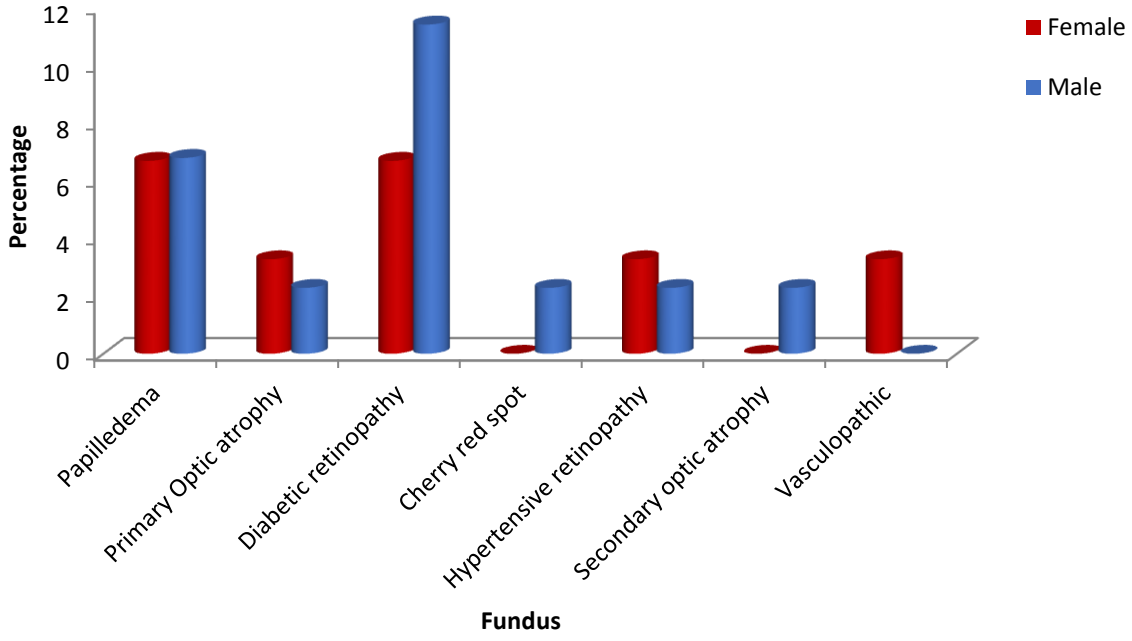


Table 9– Isolated/Multiple cranial nerve palsy of the study.

Isolated/Multiple	Gender		Total (n=74)
	Female (n=30)	Male (n=44)	
Isolated	24(80%)	36(81.8%)	60(81.1%)
Multiple	6(20%)	8(18.2%)	14(18.9%)

Graph 14 –Isolated/Multiple cranial nerve palsy of the study

Majority of the patients had isolated nerve involvement in our study

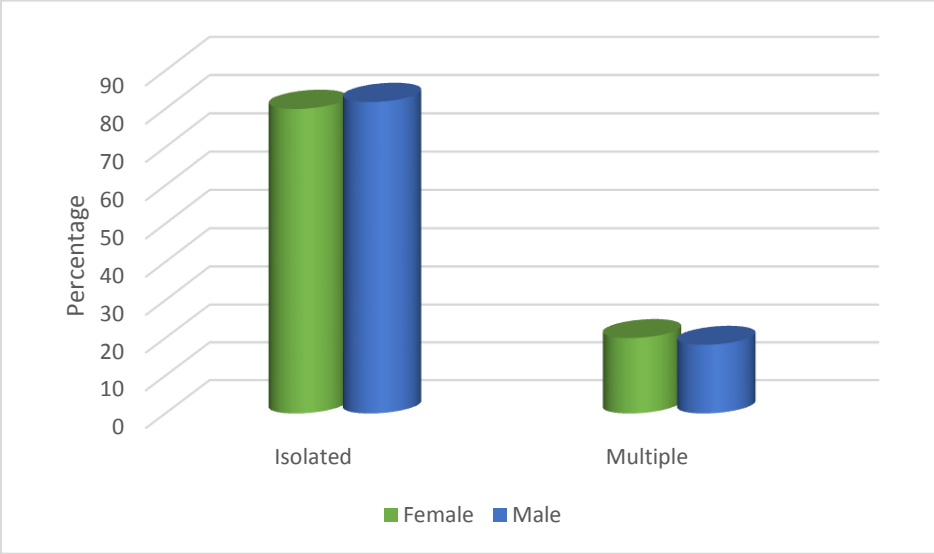


Table 10 – CT/MRI of the study.

Neuroimaging (CT, MRI or MRA) was performed for 53 (71.8%) patients and revealed an abnormality in 36 cases (48.6%) it included intracranial tumors, non-neoplastic space occupying lesions, ischemic and demyelinating lesion. Neuroimaging was normal in the remaining 17 patients (23%).

CT/MRI	Gender		Total (n=74)
	Female (n=30)	Male (n=44)	
Normal	5(16.7%)	12(27.3%)	17(23%)
Abnormal	13(43.3%)	23(52.3%)	36(48.6%)
Not done	10(33.3%)	9(20.5%)	19(25.7%)

Table 11 and Graph 15 – CT/MRI findings

Etiology	3rd nerve	4th nerve	6th nerve	3rd and 4th nerve	3rd and 6th nerve	All three nerve
Post traumatic contusion	1(1.4%)	0(0%)	1(1.4%)	1(1.4%)	0(0%)	0(0%)
Neoplastic space occupying lesion	0(0%)	0(0%)	2(2.7%)	0(0%)	0(0%)	2(2.7%)
Non – neoplastic space occupying lesion	5(6.8%)	1(1.4%)	2(2.7%)	0(0%)	1(1.4%)	0(0%)
Vasculopathic/ischemic	1(1.4%)	0(0%)	5(6.8%)	1(1.4%)	1(1.4%)	1(1.4%)
Inflammatory	0(0%)	2(2.7%)	3(4.1%)	0(0%)	0(0%)	1(1.4%)
Demyelinating	0(0%)	0(0%)	0(0%)	0(0%)	0(0%)	1(1.4%)
Post traumatic orbital fracture	2(2.7%)	2(2.7%)	2(2.7%)	0(0%)	0(0%)	1(1.4%)

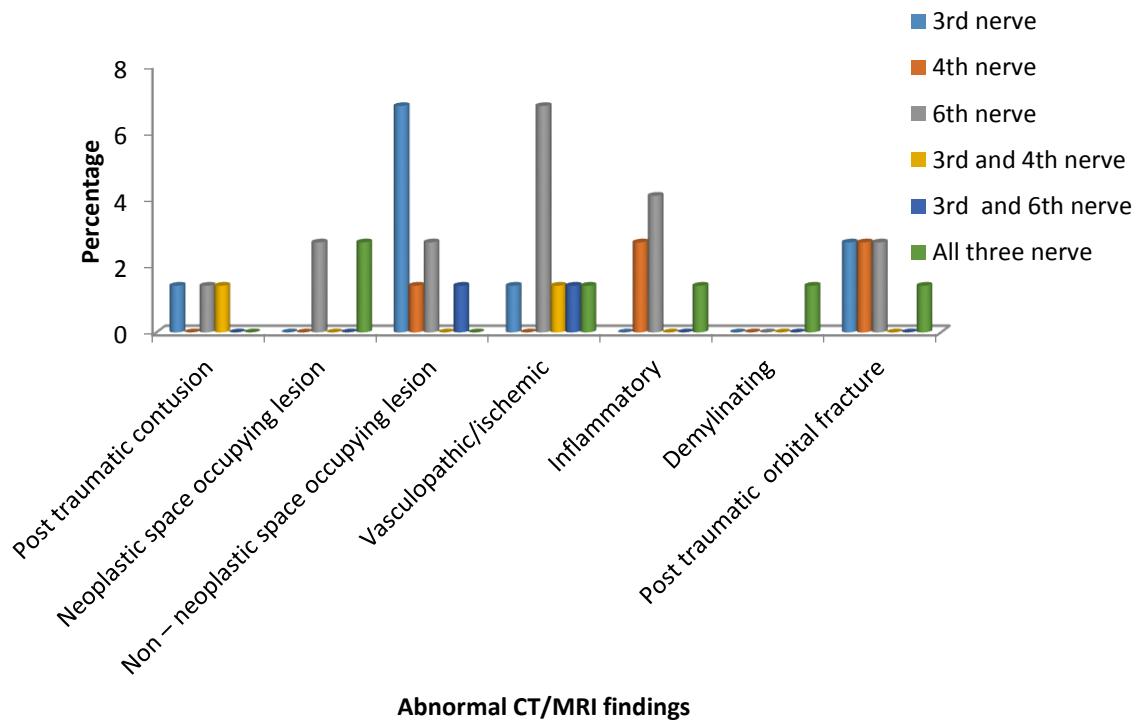
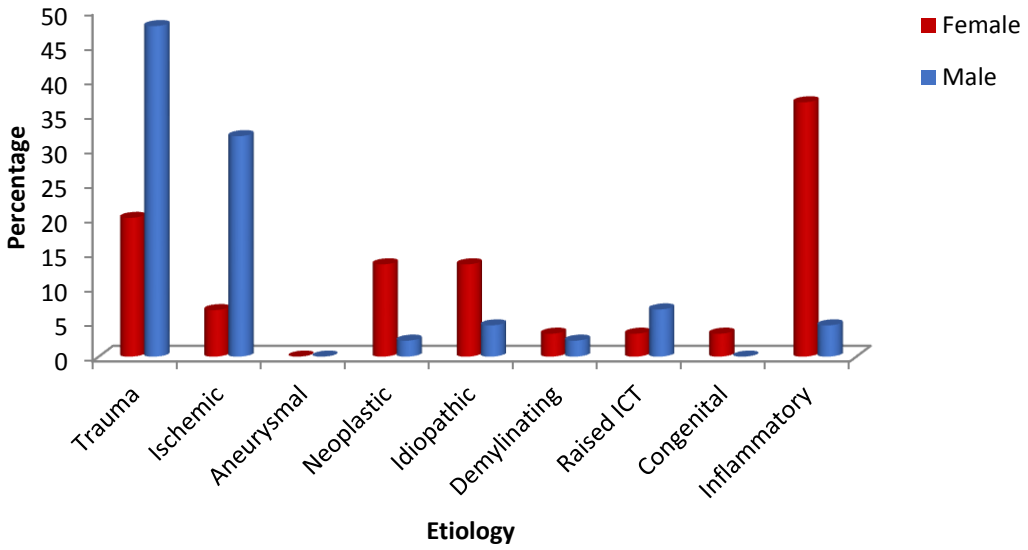


Table 12– Etiology of ocular motor nerve palsy

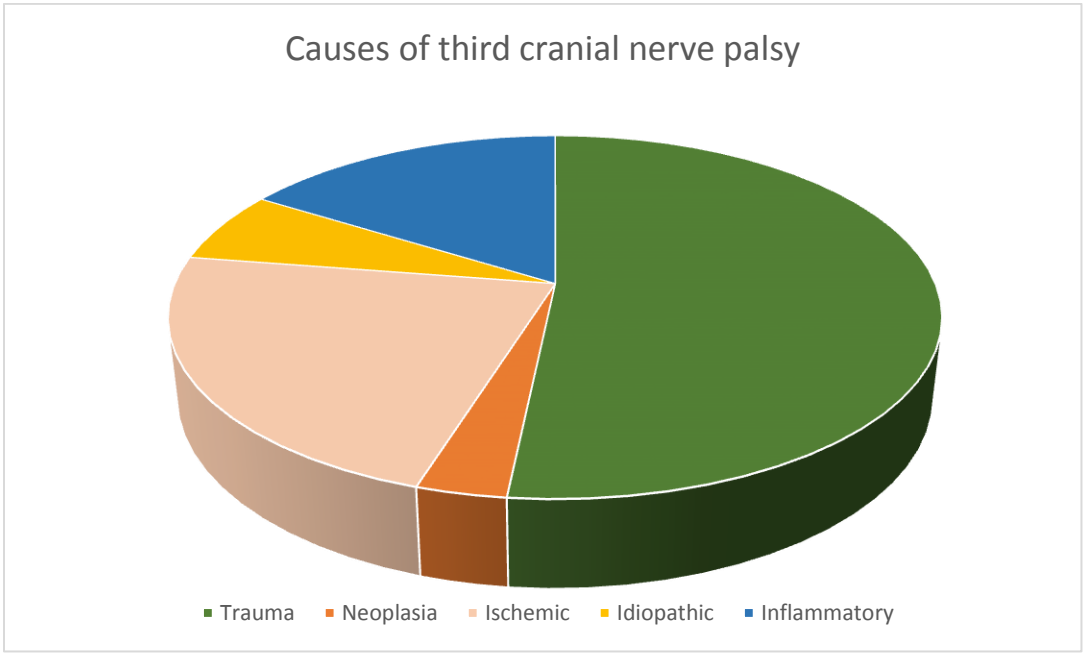
The final diagnosis was classified as traumatic in 27(36.5%), ischemic in 16(21.6%), tumor in 5 (6.8%), demyelinating in 2 (2.7%), inflammatory in 13 patients and idiopathic in 6 (8.1%) cases. Among patients with ischemic etiology, 9 (32.1%) had diabetes mellitus and 3(10.7%) had systemic hypertension, one patient had history of a cerebrovascular accident.

Etiology	Gender		Total (n=74)
	Female (n=30)	Male (n=44)	
Trauma	6(20%)	21(47.7%)	27(36.5%)
Ischemic	2(6.7%)	14(31.8%)	16(21.6%)
Aneurysmal	0(0%)	0(0%)	0(0%)
Neoplastic	4(13.3%)	1(2.3%)	5(6.8%)
Idiopathic	4(13.3%)	2(4.5%)	6(8.1%)
Demyelinating	1(3.3%)	1(2.3%)	2(2.7%)
Raised ICT	1(3.3%)	3(6.8%)	4(5.4%)
Congenital	1(3.3%)	0(0%)	1(1.4%)
Inflammatory	11(36.7%)	2(4.5%)	13(17.6%)

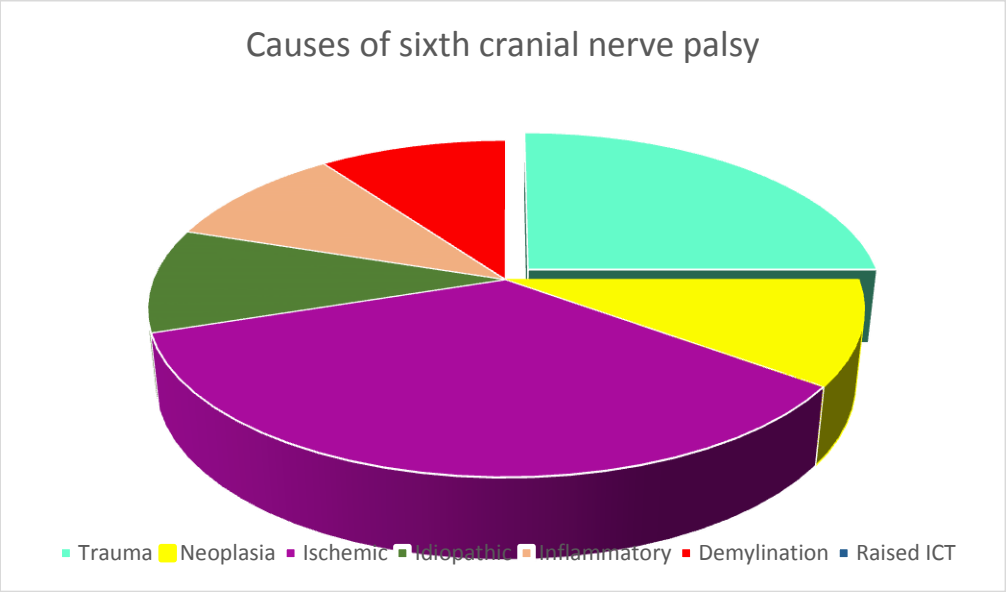
Graph 16– Etiology of ocular motor nerve palsy



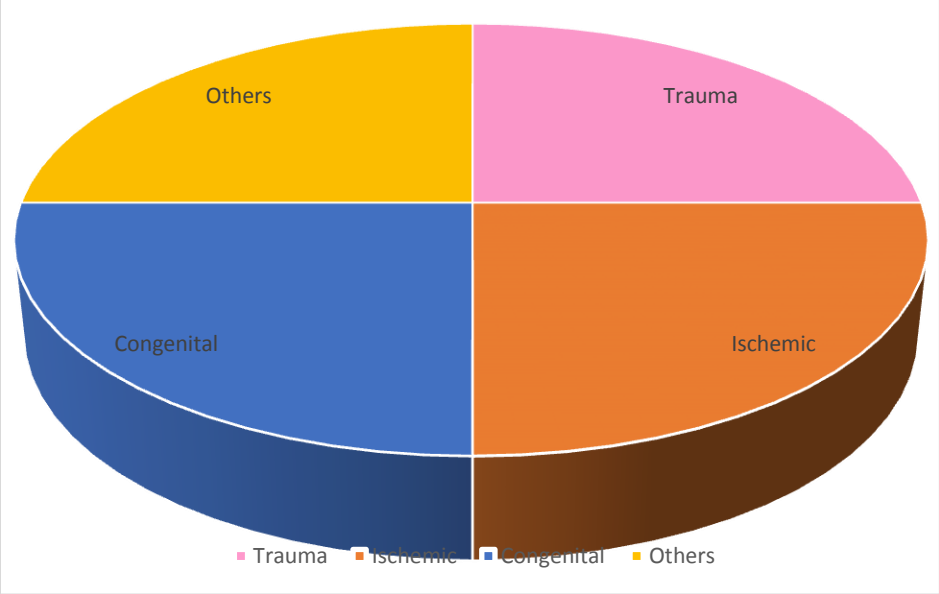
Graph 17– Etiology of Third cranial nerve palsy



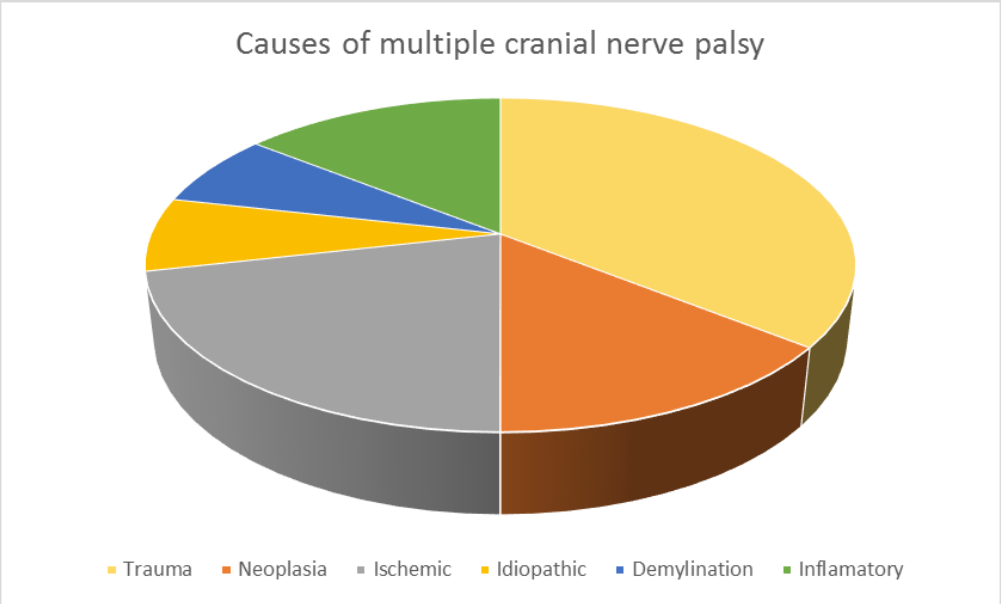
Graph 18– Etiology of Sixth cranial nerve palsy



Graph 19– Etiology of fourth cranial nerve palsy.



Graph 20– Etiology of Multiple cranial nerve palsy



DISCUSSION

Over a period of one and half year of study duration, 74 cases of ocular motor nerve palsies were seen. Our results show that most of the patients were between 31 and 40 years of age with a mean of 40.68±17.43 years (range, 7-75 years).This was similar to study done in Nigeria by **pedro-edge et al** where mean age of presentation was 34.50 ± 18.41 years (range, 4-75 years). This differs from a study by **Rowe et al[4]** where the mean age of patients was 69.18 ± 14.19 years (range, 1-94 years). This difference is not surprising since **Rowe et al[4]** study involved only stroke patients which may have accounted for the older age group as the risk factors to developing a stroke are greater in older individuals.

	Pedro edge et al[36]	**Rowe et al[4]**	**Present study**
Mean age and Range	34.50 ± 18.41 years (range, 4-75 years)	69.18 ± 14.19 years (range, 1-94 years).	40.68±17.43 years (range, 7-75 years)

Male to female ratio of ocular motor nerve palsy

The male to female ratio in our study was 1.4:1 , similarly it was 1.5:1 in a study by **Tabassi et al[18]** done at Iran which was carried out to know the etiology of third nerve, The results differs from a study by **Pedro edge et al**, where the male to female ratio was 0.8:1

	Pedro edge et al[36]	**Tabassi et al[18]**	**Present study**
Male : Female ratio	0.8:1	1.5:1	1.4:1

Chief complaints at presentation of ocular motor nerve palsy

The most common symptom at presentation were double vision, defective vision, fever, headache and ptosis. Diplopia and ptosis are in keeping with the functions of some of the affected cranial nerves. The patient with fever, eye pain, and headache and neck stiffness had inflammatory causes like meningitis, neurotuberculosis, herpes zoster ophthalmicus, HIV.

Chief compliant	**Mwanza et al[8]**	**Tabassi et al[18]**	**Pedro edge et al[36]**	**Present study**
Diplopia.	11(35.4%)	3(10.7%)	7(21.2%)	32(43.2%)
Drooping of eyelid.	7(22.5%)	22(78.6%)	4(12.2%)	17(23%)
Diminution of vision.	4(12.9%)	3(10.7%)	2(6%)	22(29.7%)
Headache.	-	13(46.7%)	7(21.2%)	19(25.5%)

This study showed that diplopia32(43.2%), which is an extremely disturbing phenomenon, remains the most common symptom at presentation which is comparable with **Mwanza et al** were diplopia was seen in 11(35.4%) patients . In our study, most patients (87.5%) had horizontal diplopia. This was expected as the 3rd and 6th cranial nerves were the most affected

This results differ from study by **Tabassi et al** were drooping of upper eyelid was the chief complaint seen in 22(78.6%) patients and diplopia was seen in least number of cases. This can be accounted as in our study as patients with mild ptosis may not be aware of the abnormality and they would not come to seek treatment for it, so any doubt arise on the presence or absence of ptosis in a case of third cranial nerve palsy, objective measurements

should be done. In our study headache was seen in 19(25.5%)cases it is comparable with both the study by **Pedro edge et al** and **Tabassi et al.**

Physical symptoms of patients with cranial nerve palsies

Anterior segment finding	**Pedro et al[36]**	**Present study**
Exotropia	13.3%	8.1%
Esotropia	26.6%	16.2%
Hypertropia	6.7%	5.4%
Severe Ptosis	13.3%	16.2%
Proptosis	-	5.4%
Complete ophthalmoplegia	13.3%	9.4%

Table presents the ocular findings. Six patients(8.1%) had exotropia in comparison with pedro et al study which was 13.3%, esotropia was seen in 12 patients (16.2%) it was less in comparison to 26.6%, hypertropia (5.7%) was almost similar to 6.7% in previous study, even though total number of patients with ptosis were 32, but only twelve patients had severe ptosis accounting for 12(16.2%), four patients had proptosis (5.4%) in contrast there were no case of proptosis in previous study and complete ophthalmoplegia were seen in 7 patients (9.4%) were comparable with the previous study(13.3%).

The frequency of distribution of oculor motor nerve palsies was compared with both Indian and western countries

Cranial nerve	Menon [5]	Mwanza[8]	Rama[28]	Rucker[26]	Rucker[27]	Rush[5]	Present study
III	63(31.9%)	11(35%)	28(40%)	33.5%	27.4%	29.0%	31(41.89%)
IV	12(6%)	04(12.9%)	1(1.4%)	6.7%	8.4%	17.2%	04(5.4%)
VI	88(44.6%)	12(38%)	29(41%)	40.9%	51.5%	41.9%	25(33.78%)
Multiple	24(12%)	06(19.3%)	32(8%)	18.9%	12.7%	11.9%	14(18.9%)

The incidence of isolated III cranial nerve involvement was similar to all the studies previously done, even though paralysis of the sixth cranial nerve is recognized as the most common type in most of the series throughout the literature (**Menon ,Mwanze, Rama,)** , the third cranial nerve was the most affected in our study

The sixth nerve affection seen in 33% was lower in our study when compared to other series. The IV nerve was involved in 4 patients (5.4%) affection were similar to **Rama et al, Menon et al, Rucker1 et al**. The incidence of combined nerve palsies in various combinations were seen in 14 patient (18.9%)was higher in our series compared to other studies previously done.

Pupil involvement of ocular motor nerve palsy

Pupil involvement	Pedro edge[36]	Present study
3rd nerve	12.5%	33.8%
4th nerve	-	-
6th nerve	-	-
Multiple cranial nerves	16.7%	18.2%

The pupil was involved in many cases of third cranial nerve palsy and multiple cranial nerve palsy, third nerve showed involvement of 33.8% was higher compared to 12.5% of **Pedro edge[26]** study, but the pupil involvement were same in cases of multiple cranial nerve palsy in both the study.

Fundus examination of ocular motor nerve palsy patients revealed fifty three patients had normal fundus and twenty one patients showed abnormal findings like diabetic retinopathy present in seven people which suggest more of ischemic etiology in OMNP patients, five patients had papilloedema suggestive of raised intracranial pressure, few cases had optic atrophy of both primary and secondary type and cherry red spot was seen in one case of trauma more suggestive of berlins edema. These findings direct us towards identifying the etiological factors of OMNP.

Laterality of ocular motor nerve palsy

Laterality	Number of cases
Right eye	48(64.86%)
Left eye	31(41.6%)
Both eye	6(8.1%)

Right eye was involved in 48(64.86%) of patients, 6(8.1%) patient had both eye involvement these people had involvement of VI cranial nerve in four patients and two patients had total ophthalmoplegia, etiology of them were demyelination, inflammatory and raised intracranial pressure Thus in more than half the cases, bilateral sixth cranial nerve was involved it was similar to **Rucker**[27]

Involvement of other cranial nerve in ocular motor nerve palsy

Any other cranial nerve involved	**Menon et al**[6]	**Present study**
2^{nd}	20(10.15%)	8(10.8%)
5^{th}	16(8.1%)	1(1.4%)
7^{th}	14(7.1%)	2(2.7%)
8^{th}	1(0.50%)	1(1.4%)
12^{th}	1(0.50%)	-
Total	197(100%)	74(100%)

In present study optic nerve was involved in higher number in 8(10.8%) patients similar to study by **Menon et al** 10.15%, and seventh nerve was involved in two patients (2.7%) less in number compared to the previous study coated, and fifth nerve and eighth nerve were involved in one case each .No case of XII nerve involvement was documented in our study.

Etiology of the patients with ocular motor nerve palsy

Etiology	**Rama et al[28]**	**Present study**
Inflammatory	19(21.11%)	13(17.6%)
Intracranial neoplasia	16(17.77%)	5(6.8%)
Head injury	16(17.77%)	27(36.5%)
Vascular	13(14.44%)	16(21.6%)
Idiopathic	6(6.66%)	6(8.1%)
Miscellaneous	13(14.44%)	7(9.5%)

Head injury 27(36.5%) is one of the common causes of ocular motar palsy in our study in comparison to the study by **Rama et al** and **Rucker** in which head trauma accounted only in 18% of cases ,our study had higher incidence of trauma as this region is more close towards the highway and prone for road traffic accident.

The mechanism of traumatic abducent nerve palsy may be direct mechanical injury to the nerve or an indirect injury. The indirect injury results from nerve ischemic change due to vessel compression or vasospasm.[38, 39] The three cranial nerves (3^{rd}, 4^{th} and 6^{th}) are fed by a comprehensive network of arterial blood vessels, and thus are susceptible to vascular

compromise particularly localized lesions and disturbances at the level of the brainstem cranial nerve nuclei as well as in the cavernous sinus just before innervating the extraocular muscles.[40]

Paralysis of the 6th cranial nerve has no localizing value because it may be affected by, almost any type of cerebral lesion. Many theories have been proposed for this observation.[41] **Collier** thought that this happens because of the shifting backwards of the brainstem since the direction of the nerve is mostly fronto-caudal. Affection of the 6th nerve is followed by the 3rd, 7th and 8th cranial nerves. The more fragile 4th nerve with its longer intracranial course (75 mm) is less affected because it is thought to be protected by, the free margin of the tentorium cerebelli. Alternately, the 6th nerve, has the longest extradural course (even though its entire length is 1/3 that of the 4th nerve), thereby making it vulnerable to intracranial insults as seen in meningitis and subarachnoid hemorrhage[4]

Inflammatory etiology like neuroinfection, tuberculoma , herpes zoster ophthalmicus, Human immunodeficiency virus infection , meningitis were seen in 13 patients .In comparison to Rama et al were inflammatory cause was the most common casuse as the study was conducted at Rayalaseems area i.e., former Ceded Districts of Andhra Pradesh Is an entity which accounts for over a third of neurological problems in the region.

Vascular lesions like 16(21.6%) diabetes was the underlying cause in most of the OMNP(80%) 11 out of 16 had diabetes, two patients had hypertension., It is comparable with **Berlit et al and Moster**[37] were OMNP of vascular origin (29.7%) was the most common, followed by inflammatory disease (19.4%) and tumors (10.9%). **Moster** also said that the vascular group was involved in older compared to the younger group.

Five cases of intracranial neoplasia were seen in the present study accounting for 6.8% the incidence were less when compared to **Rama**[28]**Rucker**[27] while it was similar to **Krishna's** series[43]

Not even a single case of aneurysm was detected in our study as an etiology of ophthalmoplegia. The incidence was very high (7.7%) in **Rucker's** series **Krishna and Mehkri**[43]also reported only 4 cases (2.2%). This might not reflect the true incidence of aneurysm in general as there have been other cases which manifested mainly as subarachnoid haemorrhage.

In every case series a proportion of patients remained whose pathogenesis was unknown. In our study seven patients, the etiology could not be established. The third nerve alone was affected in three of them, while the third and sixth were involved in one case and total ophthalmoplegia were seen in two patients of unknown etiology. **Rucker** found 211 of 1000 cases (21%) while **Krishna and Mehkri** [43]found 18% of undetermined etiology. The incidence in our study was lower. No definite explanation could be offered for this.

Associated vision loss was found in 8 patients, they had optic atrophy due to optic nerve involvement clinically evident as relative afferent pupillary defect, V, VII and VIII nerve were involved in three patients with multiple cranial nerve palsies. These associated findings were correlating with **Menon's study.**

Etiology of third cranial nerve palsy

Etiology 3rd nerve	**Rucker[26]**	**Rush[5]**	**Menon[6]**	**Tabassi[18]**	**Present study**
Head trauma	18.6%	16.2%	22,2%	14.3%	51.61%
Neoplasia	12.7%	11.7%	9.5%	7.1%	3.2%
Ischemic	22.99%	20.7%	-	42.8%	22.58%
Aneurysm	23.35%	13.8%	-	7.1%	-
Undetermined	34.67%	23%	30%	17.8%	6.4%
Others	9.8%	14.5%	9.5%	10.7%	16.12%

In the present case study, head injury (51.61%) and microvascular ischemia (22.58%) were significant causes for isolated oculomotor nerve palsy.

Post-traumatic oculomotor nerve palsy accounted for 51% of total cases, incidence was high compared to **Rucker et al** (18.6%) and **Rush et al** (16.2%). The pathogenesis of oculomotor palsy in head injury is sufficiently well documented [50]and ischemic etiology were seen in 22.58% similar to the studies **Rucker** 22.99%, **Rush** 20.7% **,** but differed from **Tabassi et al's** study as it accounted for 42.8%

The etiology of third cranial nerve palsy remained unknown in about 6.4% of cases in our study, it differed from **Rucker et al who** found 34.67**%, Rush - 23%, Menon -30%, Krishna et al** - 18%. This might be because the earlier studies were carried out in the pre imaging era .[26, 43]

In most of the earlier studies, these etiologies were considered as miscellaneous causes except for series by **Vimala[28] et al**. who used ultrasonography as an investigative tool in the diagnosis of these cases and found results similar to the our study (16.5%). **Rama et al**(17.5%) had comparable results owing to high prevalence of tuberculosis in the area [28].

Trobe et al suggested an approach to manage pupil sparing oculomotor nerve palsy based the relative deficit in pupillomotor and extraocular muscle function. He divided patients into three groups: (1) patients with a normal pupillary sphincter and completely palsied extra-ocular muscles. Such patients should not have cerebral angiography if they are aged 50 or more; (2) patients with a normal pupillary sphincter and incompletely palsied extraocular muscles. Such patients should have angiography, particularly if the inferior oculomotor division is spared; (3) patients with a subnormal pupillary sphincter and completely palsied extraocular muscles

("relative pupil-sparing"). Such patients should have angiography unless they have clear-cut vasculopathic findings[51].

Etiology of sixth cranial nerve palsy

Etiology 6th nerve	Rucker[26]	Rush[5]	Menon[6]	Present study
Head trauma	10.67%	16.7%	10.2%	20%
Neoplasia	30.8%	14.6%	12.5%	8%
Aneurysm	2.9%	3.6%	-	-
Vascular	8.9%	17.4%	10.2%	28%
Undetermined	21.70%	29.6%	40.56%	8%
Others	24.8%	17.9%	24.4%	32%

Majority of the patients with sixth nerve palsy had ischemic etiology seen in 28% of people, which is comparable with study done by **Rush** 17.4% Diabetes mellitus and arterial hypertension are widely held to be the most common risk factors for microvascular ischemic CN VI palsy.

Studies have suggested other risk factors including hyperlipidemia, coronary artery disease, or alternative signs of hypertensive end-organ damage such as left ventricular hypertrophy (LVH) Further investigations have recently sought to specifically address the degree to which these risk factors are associated with microvascular ischemic CN VI palsy. Patel et al[29]reviewed 137 cases of CN VI palsy in patients of all ages, and found the most frequent associations to be unknown (26%), hypertension alone (19%), diabetes mellitus and hypertension (12%), and diabetes mellitus alone (4%) [49]. The authors found that arterial hypertension alone was not associated with an increased likelihood of CN VI palsy as

compared with diabetes mellitus alone, which carried a six-fold increase. Coexistent diabetes mellitus and arterial hypertension conferred an eight-fold increase.

Head trauma accounted for second most common cause of sixth nerve palsy which is about 20% ,which is comparable with **Rush** findings in his study

With regard to neoplasm as the cause of isolated, sixth nerve palsy, our study showed previously undiagnosed tumor, primary or metastatic; to be the causative lesion in 8% compared to higher percentages seen in other studies **Rucker, Rush , Menon** of 30.8%, 14.6%, 12.5% respectively. **Bendszus**[52] found a high proportion of cases (60.4%) to be caused by tumors, tumor like lesions and metastases On the contrary, **Patel et al** have did not find even a single case of sixth nerve palsy. It is difficult to explain such vast variations in the incidence of tumor related isolated abduces palsy.

Other causes mainly include inflammatory and those leading to raised intracranial pressure which press on the sixth nerve giving a false localizing sign, it accounted for 32% higher when compared to previous studies. , the 6th nerve, has the longest extradural course (even though its entire length is 1/3 that of the 4th nerve), thereby making it vulnerable to intracranial insults as seen in meningitis and subarachnoid hemorrhage[52]

Cushing[53]postulated that when the anterior inferior cerebellar artery ran ventral to the 6th nerve it may press on and groove the nerve and the underlying pons due to increased intracranial pressure and thus cause lateral rectus palsy.

Another author suggested that the bend over the sharp apex of the petrous temporal bone exposes the 6th nerve to insult when there is an increase in intracranial pressure with resultant coning of the brain. If the 6th nerve is fixed to the pons and more or less held in the cavernous sinus, it will therefore be pressed up against the sharp border of the petrous temporal bone causing an interruption in conduction and palsy of the lateral rectus.

A similar condition may follow compression of the skull in difficult labor with or without forceps and may explain 6th nerve palsy at birth. A review of the medical literature[55, 56] has shown that abducent nerve vulnerability results from factors other than its intracranial length.

Etiology of fourth cranial nerve palsy

Etiology 4th nerve	**Rucker[27]**	**Rush[5]**	**Menon[6]**	**Present study**
Head trauma	27.38%	32%	41.66%	25%
Neoplasia	8.3%	4.1%	-	-
Aneurysm	-	1.7%	-	-
Vascular	15.47%	18.6%	8.3%	25%
Undetermined	33.33%	29.6%	33.33%	-
Others	15.47%	7.6%	25%	25%

There were 4 cases of fourth nerve palsy in our study, closed head injury was responsible for one case (25%) of fourth nerve palsy, the incidence remained similar to previous studies. Remaining one case each of ischemic, congenital, inflammatory etiology.

Etiology of multiple cranial nerve palsy

Etiology- multiple nerve palsy	**Rucker[27]**	**Rush[5]**	**Menon[6]**	**Present study**
Head trauma	12.90%	21%	26.47%	35.71%
Neoplasia	41.93%	33%	20.58%	14.28%
Aneurysm	4.8%	10.9%	-	-
Vascular	-	5.04%	-	21.42%
Undetermined	14.51%	8.4%	-	7.14%
Others	25.80%	20%	38.22%	21.42%

Multiple cranial nerve palsies accounted for 14.3% of all cases. This figure is much lower than reported in the other two reports from India. Krishna and Mehkari (1973) found multiple cranial nerve involvement in 25% of their cases while Rama et al (1980) quoted an even higher incidence (35.5%) amongst their patients. The incidence of multiple nerve palsies in the present series goes well with that quoted in western literature (**Rucker, 1958 [27]; Rush and Younge[5], [5]1981**). We observed the same combination pattern of multiple nerve palsies as described by **Rush and Younge[5] (1981).**

Cases of head trauma (35.7%) causing multiple OMNP were encountered more frequently in our study, when compared to reports by other Indian authors **Krishna and Mehkari[46] 1973, Rama[28] et al. 1980**. In our study , there was a decline in neoplasm(14.2%) causing sixth cranial nerve palsy compared to 30.9% of **Rucker's[26]** (1966) series. A similar trend was noted **Rush and Younge5**in 1981.

Imaging of ocular motor nerve palsy

Of the 74 patients of ocular motor nerve palsy patients, only 53 underwent neuro-imaging. Of these seventeen imaging were normal. The observation that the neuro-imaging results were normal should not give a false sense of hope. This was shown in a case reported by **Hoenig[44]**of a 62-year-old male with facial nerve palsy who was managed over an 18-month period with initial normal magnetic resonance imaging results. **Hoenig[44]** therefore cautioned that normal neuro-imaging results might be falsely misleading in identifying the cause of cranial nerve palsy.

The indications of neurological imaging in 3rd nerve palsy are –

a). All patients less than 10years irrespective of pupillary findings.

b). Children more than 10 years with pupillary involvement

c). If pupil becomes dilated after 5-7 days of onset

d). Multiple cranial nerves affected

e). No improvement is seen within 3 months

f). Signs of aberrant regeneration

e). other neurological signs

Cranial nerve palsies are one of the most common indications for neuroimaging. Although MRI scans are generally felt to be more sensitive and specific than CT scans in the evaluation of cranial neuropathies, no conclusive evidence demonstrates an increased yield from performing an MRI scan rather than a CT scan for the specific evaluation of fourth nerve palsy[57].

The etiology of OMNPs remained unknown in more than 25% of cases[3, 5] it is however worth stressing that in the recent past, there has been a marked progress in investigative procedures, mainly in the field of imaging. This has helped increase the chances of arriving at an etiological diagnosis in cases of OMNP[13]

Traumatic isolated third nerve palsy should undergo computed tomography (CT) scanning to evaluate for associated central nervous system damage (subdural or intracerebral hematoma) as indicated by associated neurologic signs and symptoms.[58] Neuroimaging may also warranted in patients with third nerve palsy after minimal or trivial trauma to exclude mass lesions or cerebral aneurysms[58] .**Walter et al[59]** described two patients with third nerve palsies precipitated by minor head trauma with negative brain CT scans; both were subsequently discovered to have ipsilateral posterior communicating artery aneurysms.
MRI is recommended in all patients with congenital third nerve palsies, mainly to investigate for associated structural abnormalities of the brain.

Ischemic third nerve palsy with pupillary sparing has, however, also been reported due to fascicular damage with mesencephalic infarcts documented on MRI.[60, 61]

Some authors recommend noninvasive vascular studies (MRI with MR angiography [MRA] or CT angiography) in all patients with third nerve palsy— regardless of whether they have diabetes or any other systemic vasculopathy[62]

Patients with a ‘‘relative pupil sparing’’ third nerve palsy should have an MRI scan to rule out the possibility of a compressive lesion. **Cullom et al[63]** published a small prospective study of 10 patients with ‘‘relative pupillary sparing’’ third nerve palsy, and none of the patients demonstrated aneurysms. **Jacobson and Trobe** recommend MRI followed by catheter angiography if the MRI scan does not disclose a nonaneurysmal cause.[64]

Patients with third nerve palsy that worsen after the acute stage (> 2 weeks) or who develop new neurologic finding, no vasculopathic risk factors, those with aberrant regeneration Patients without resolution of third nerve palsy after 12 to 16 weeks require MRI and MRA or standard angiography.

Multiple retrospective studies of traumatic fourth nerve palsy have recommended that isolated, traumatic, unilateral, or bilateral fourth nerve palsies do not require additional neuroimaging or further evaluation.[65, 66]Fourth nerve palsy after mild head trauma has been observed in underlying asymptomatic basal intracranial.[67, 68]

Vasculopathic and congenital fourth nerve palsies are not associated with intracranial lesions, they do not require neuroimaging. Isolated fourth nerve palsy is more commonly due to stretching of the superior medullary velum of the fourth ventricle, where the trochlear nerve decussates, by an intraventricular mass.[45,48]

Richards et al[3]reported that ‘‘multiplanar CT may be a sufficient noninvasive study, especially when clinical suspicion is high . . . (or) in patients with other neurologic findings. . . ’’[3] Nevertheless, MRI is the study of choice for patients with fourth nerve palsy.

In acute traumatic sixth nerve palsy and Isolated vasculopathic sixth nerve palsy may be observed for improvement for 4 to 12 weeks. [69] In a prospective clinical study of 66 patients with ocular motor cranial neuropathies, **Chou et al.**[32] attributed 57 (86%) of these to a microvascular ischemic etiology.

Nonvasculopathic sixth nerve palsy should undergo neuroimaging. Younger patients, or those without vasculopathic risk factors could also undergo a more extensive evaluation, these have a significant (27%) chance of harboring an underlying malignant neoplasm.[7]

In our study abnormal CT/MRI findings were as follows post traumatic contusion were seen in three cases, among them one case had third nerve palsy, one case of sixth nerve palsy, and a one case of combined 3rd and 6th nerve palsy. Neoplastic space occupying lesion were seen in two cases of sixth nerve palsy and two cases of total ophthalmoplegia, Non neoplastic space occupying lesion these include of Sub arachinoid hemorrhage(SAH) Subdural hemorrhage(SDH) were seen in five cases of third nerve palsy, one case of fourth nerve palsy, two cases of sixth nerve palsy , one case of combined third and sixth nerve palsy are seen.

Ischemic/vasculopathic lesions were seen in one case of third nerve, five cases of sixth nerve palsy, one case of combined nerve palsy each. Inflammatory cause like tuberculoma, meningitis, mucormycosis,were seen one case of fourth nerve palsy, three cases of third

nerve palsy, and there was one case of fungal sinusitis of maxillary sinus that had total ophtthalmoplegia. Demyelinating lesions were evident in two patients one having multiple sclerosis with sixth nerve palsy and other case of total ophthalmoplegia having demyelinating plaque at the level of brain stem.

The traumatic nerve palsy cases were associated with fracture of medial wall, lateral wall floor of orbit involved one wall or in various combinations in number of cases, These are seen in two cases of third nerve palsy, one case of fourth nerve palsy , two cases of sixth nerve palsy, and one case of superior orbital fissure syndrome having total ophthalmoplegia

CONCLUSION

We studied the clinical presentation and etiological pattern of ocular motor nerve palsies in Kolar region, diplopia was the most common symptom at presentation followed by ptosis.

Isolated cranial nerve were more frequently involved in our study among them third nerve was seen in majority of cases and etiology causing third nerve palsy was trauma in most cases, second most common nerve involved was sixth cranial nerve and most cases of 6^{th} cranial nerve palsy were related to systemic disorders such as diabetes mellitus, hypertension. Trauma was found to be the major etiological factor for multiple cranial nerve palsies followed by neoplasia and demyelination, optic nerve was involved in majority of the patients in our study.

CT/MRI have been performed in 70% of our cases and the probable diagnosis was made in 49%. Hence proving the usefulness of these imaging modalities in ocular motor nerve palsies.

Ocular motor nerve palsy patients with systemic risk factors like diabetes, hypertension can be managed initially with close observation. Imaging is needed for all those ocular motor nerve palsy patient who do not show improvement after the acute stage >2weeks, or develop neurologic findings, or in those patients without resolution after 12 to 16 weeks.

SUMMARY

This study was done to know the clinical presentation and etiological factors in patients with ocular motor nerve palsy in Kolar region

74 patients of ocular motor nerve palsy, who attended outpatient department of ophthalmology, R.L.JALAPPA HOSPITAL attached to SRI DEVARAJ URS MEDICAL COLLEGE, TAMAKA, KOLAR, took part in this observational study between december 2013 to august 2015.

The mean age of presentation was 40.7 years and the incidence of nerve palsy were more between the age group of 31 years to 40 years.

60% of male patients and 40% female took part in this study and male to female ratio was 1.4:1.

Most of the patients came with chief complaints of diplopia (43%) followed by ptosis(23%), headache(19%), diminution of vision(8.1%), fever(12.2%) and eye pain (9.5%). Majority of patients about 74% presented within a week after onset of symptoms to the hospital.

The patients had associated systemic risk factors (19%) like diabetes mellitus hypertension, bronchial asthama. Diabetes was seen in 14.9 % patients and it is consider to be a significant risk factor for ocular motor nerve palsy of ischemic etiology, this is supported by fundus changes of different degree of diabetic retinopathy.

Visual acuity was normal (VA = 6/6) in 66.89% of ocular motor nerve palsy, impaired (VA <6/18) in 22 (14.86%) and 27 eyes (18.24%) had low vision(VA = <3/60) at presentation.

Isolated cranial nerve palsy were seen in 60 patients(81%) and 14 patients(19%) had multiple cranial nerve palsy

The frequency of distribution of ocular motor nerve palsy was the 3rd cranial nerve was most commonly affected in 31 patients(41.89%), 4th cranial nerve was involved in 4 patients(5.4%) and 6th cranial nerve was involved in 25 patients(33.78%) .The 3rd and 4h cranial nerves were affected in 3 patients(3.8%), 3rd and 6th cranial nerves were involved in 4 patients(5.2%), all the three cranial nerves (3rd, 4th,6th) total ophthalmoplegia was seen in 7 patients (9.4%).

Patients with 3rd and multiple cranial nerve palsies had sluggishly reactive pupils and in some pupil was dilated and fixed seen in about 40.54%.

Right eye was involved in majority of the patients accounting for 48(64.8%) and left eye in 31(41.8%) and both eye was involved in 6(8.1%) patients.

Optic nerve was involved in majority of the patients in our study seen in 8 patients(10.8%) they came with diminution of vision.

Neuroimaging (CT, MRI or MRA) was performed for 53 patients(71.8%) and revealed an abnormality in 36 cases(48.6%) which included intracranial tumors, non-neoplastic space occupying lesions, ischemic and demyelinating lesion, neuroimaging was normal in the remaining 17 patients(23%).

Trauma was the major etiology seen in 36.5% of ocular motor nerve palsy followed by inflammatory(17.6%), and ischemic(21.6%). No cases of aneurysm as etiology were detected in our study.

Trauma seen in 51.61% accounted for the maximum number of isolated third cranial nerve palsies, diabetes mellitus seen in 28% was the most common diagnosis of isolated sixth cranial nerve palsy. Multiple cranial nerve palsies were most associated with trauma(35.71%) followed by neoplasia and demyelination disorders.

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ANNEXURE 1: PROFORMA

SI No :
Date :
HISTORY

Particulars of the patient:

- **Name :**
- **Age :**
- **Sex :**
- **Address :**
- **Hospital No.:**

Chief complaint:

History of presenting illness

Past history:

Family history:
Personal history:
Examination

Ophthalmic examination

	RE	LE
(a) Visual Acuity		
Distance		
Near		
NPA / NPC		
(b) Confrontation visual fields		
(c) Head posture		
(d) Forehead		
(e) Ocular posture		

(i) Slit lamp examination

(i) Lids

(ii) Conjunctiva

(iii)Cornea

(iv) A/C

(v) Iris

(vi) Pupil

(vii) Lens

(viii) Intraocular pressure

(Applanation)

(j) Gonioscopy

(k) Ophthalmoscopic examination

(a) Direct

(b) Indirect

(i) Ocular motility

Inspection

Duction

Vergence

Assessement of ocular misalignment

Hirchberg corneal reflex test

Cover test

Cover-uncover test

Alternate cover-uncover test

Prismbar cover test

Tests for stereopsis

Nystagmus

Forced duction test / Force generation test

(k) Assessement of Double vision

Diplopia charting

Hess screening

Maddox rod test

Double Maddox test

(l) Fatigueability test

Neurologic examination

Mental status

Sensory system

Motor system

Cerebellar function

Gait / Reflexes

Examination of other cranial nerves

General systemic examination

Ht / Wt

Pallor / Icterus/Clubbing/Edema/Lymphadenopathy

<u>Provisional Clinical diagnosis:</u>

Lab Investigations-

Imaging

Follow-up studies

ANNEXURE 2: CONSENT

CONSENT TO PARTICIPATE

I, the undersigned, agree to participate in this study and authorize the collection and disclosure of my personal information as outlined in this consent form.

I have read or had read to me and understand the purpose of this study, the procedures that will be used, the risks and benefits associated with my involvement in the study and the confidential nature of the information that will be collected and disclosed during the study.

I have had the opportunity to ask questions regarding the various aspects of this study and my questions have been answered to my satisfaction.

I understand that I remain free to withdraw from this study at any time and this will not change my future care.

Subject's name and signature /thumb impression Date:

Name and signature of parent /guardian Date:

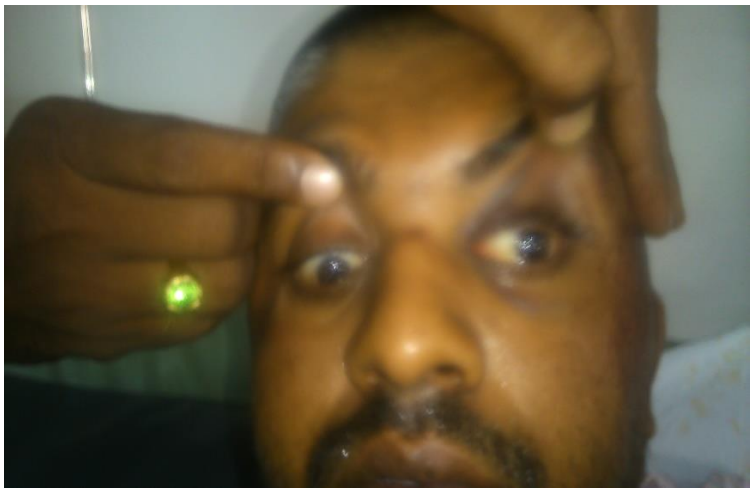
Name and signature of person obtaining consent Date:

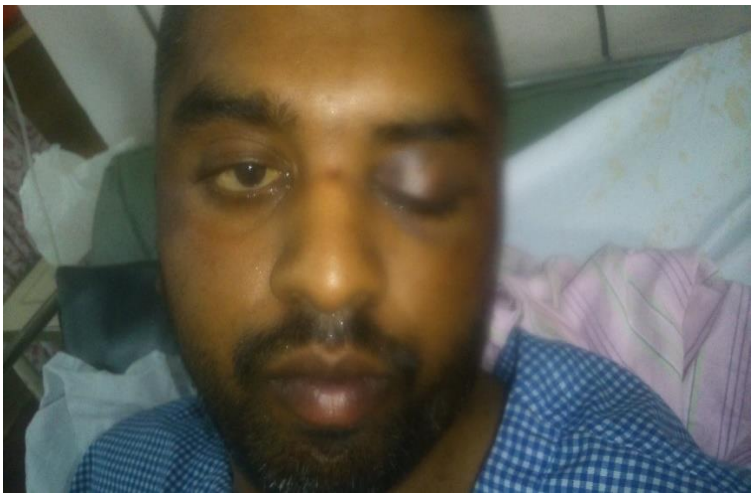
ANNEXURE 3: PHOTOGRAPHS

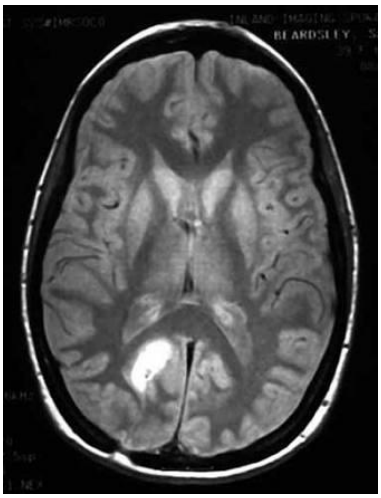
PHOTOGRAPH 1 - EXAMINATION OF THE PATIENT – EXTRA OCULAR MOVEMENTS

PHOTOGRAPH 2 - NEUROLOGICAL EXAMINATION

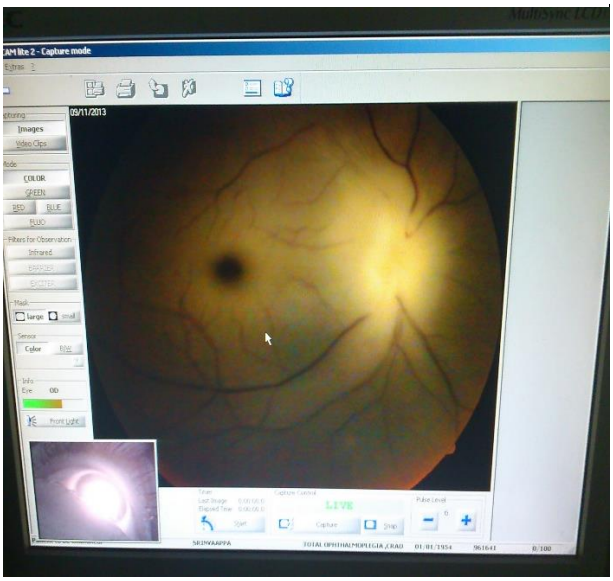
PHOTOGRAPH 3 – STUDYING COMPUTED TOMOGRAPHY IMAGE

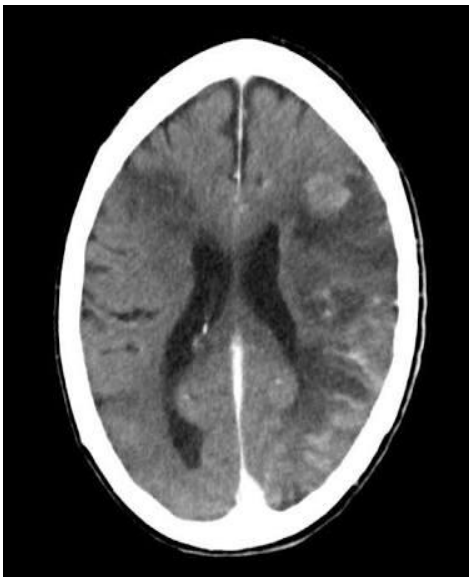




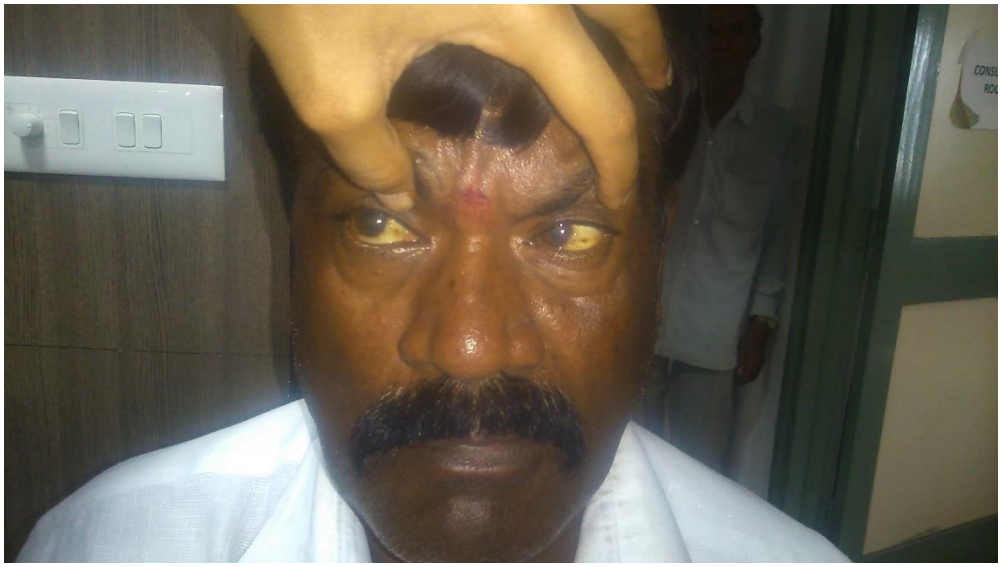


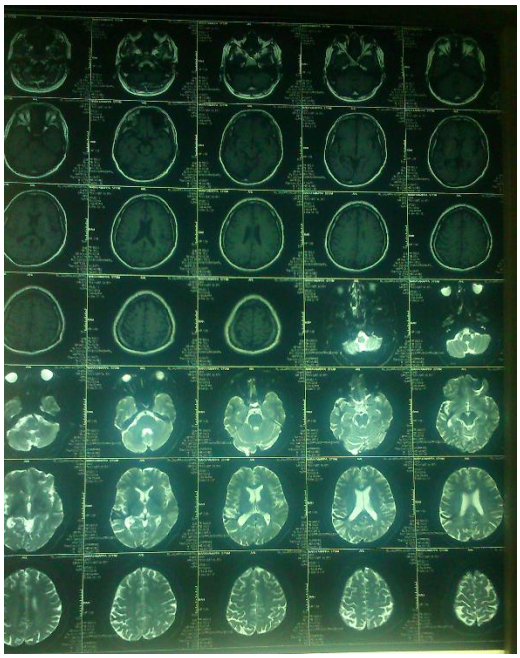
PHOTOGRAPH 4 - CASE OF TRAUMATIC THIRD NERVE PALSY – LEFT EYE



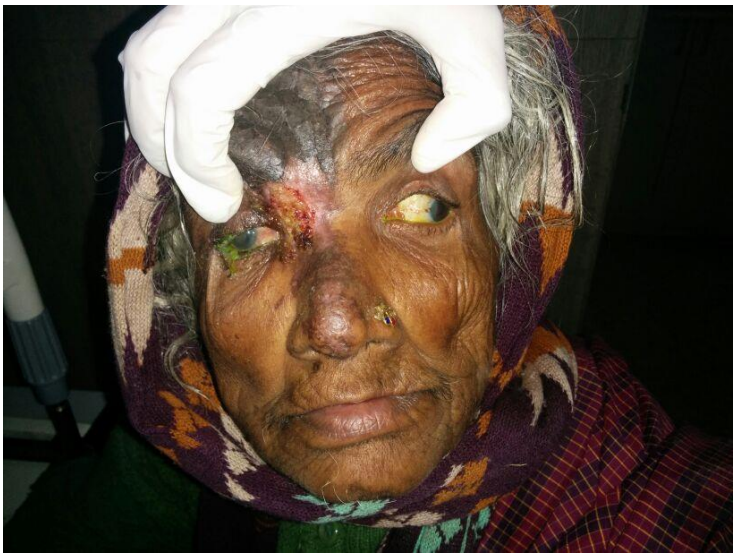


PHOTOGRAPH 5 - CASE OF TOTAL OPHTHALMOPLEGIA – RIGHT EYE DUE TO INFLAMMATORY CAUSE

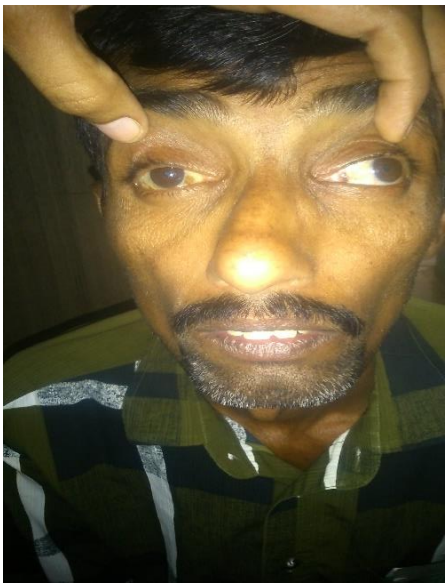




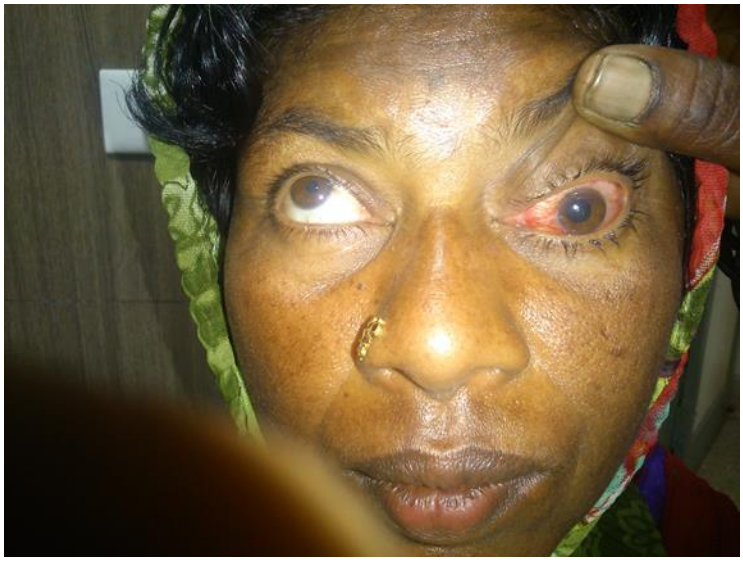
PHOTOGRAPH 6 - CASE OF SIXTH CRANIAL NERVE PALSY RIGHT EYE DUE TO DIABETES CAUSE



PHOTOGRAPH 7 - CASE OF SIXTH AND THIRD CRANIAL NERVE PALSY RIGHT EYE DUE TO HERPES ZOSTER

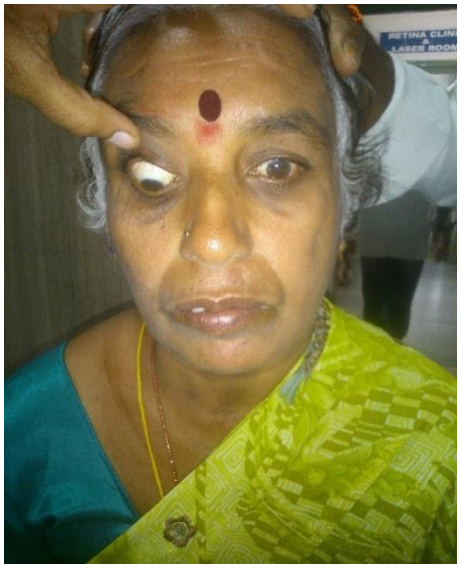


PHOTOGRAPH 8- CASE OF IDIOPATHIC THIRD CRANIAL NERVE PALSY - RIGHT EYE

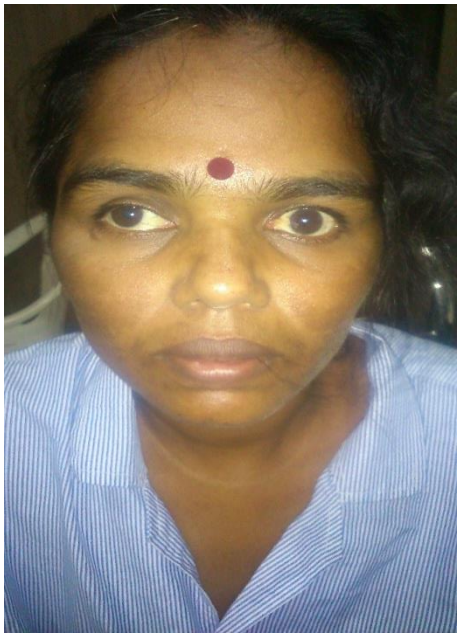


PHOTOGRAPH 9- CASE OF NEOPLASTIC TOTAL OPHTHALMOPLEGIA

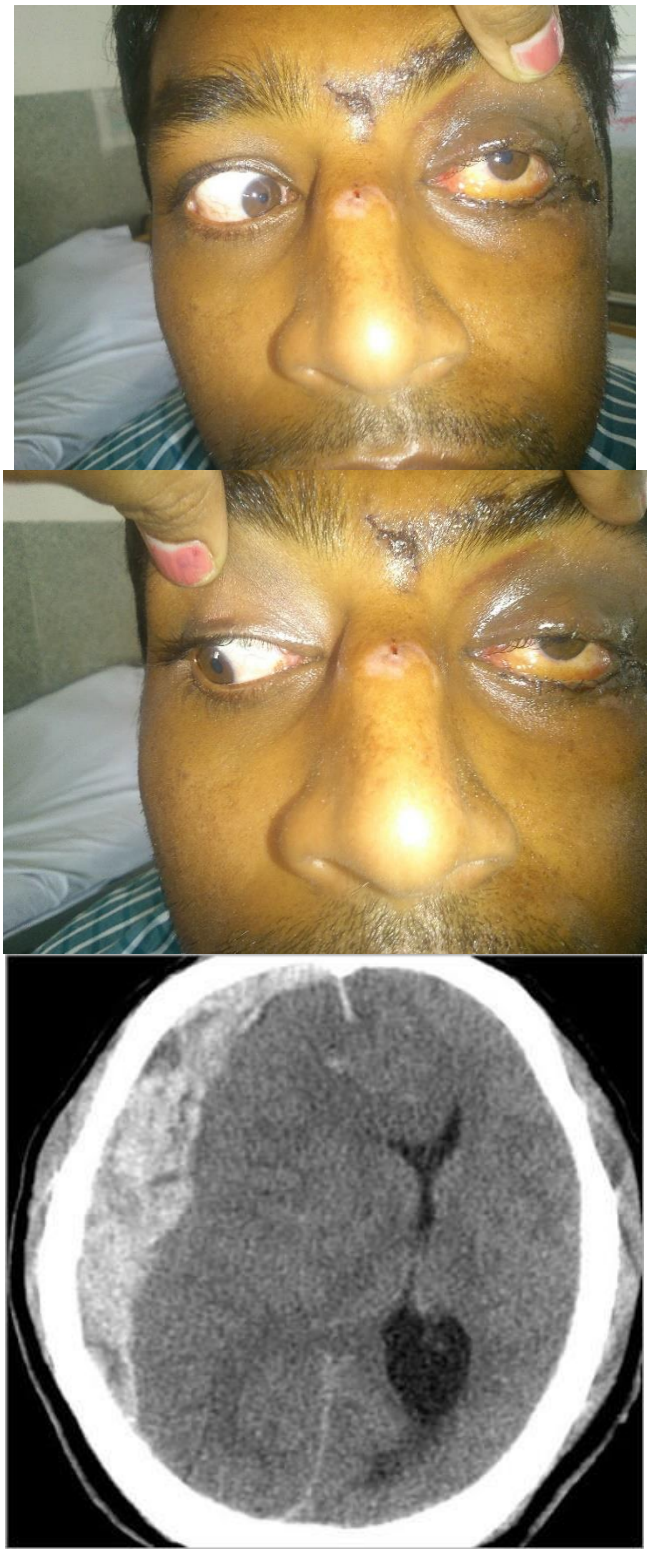
LEFT EYE.



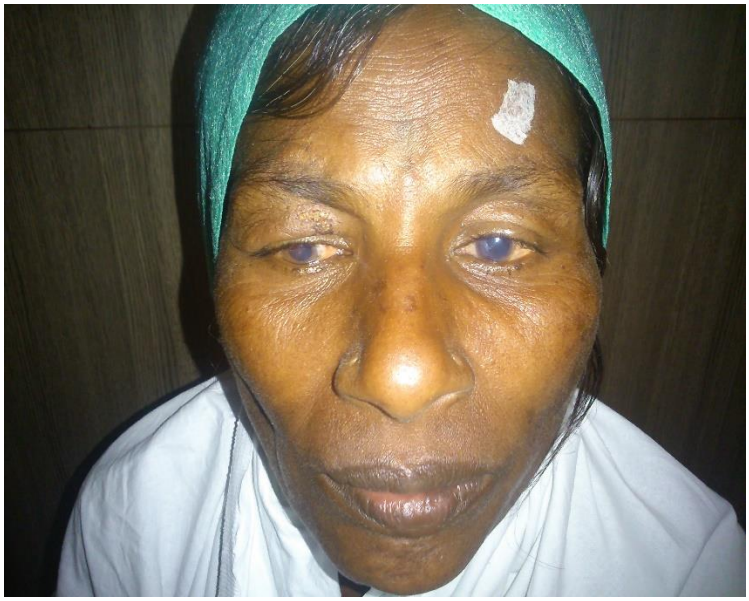
PHOTOGRAPH 10- CASE OF THIRD AND FOURTH CRANIAL NERVE PALSY – RIGHT EYE DUE TO ISCHEMIC ETIOLOGY..

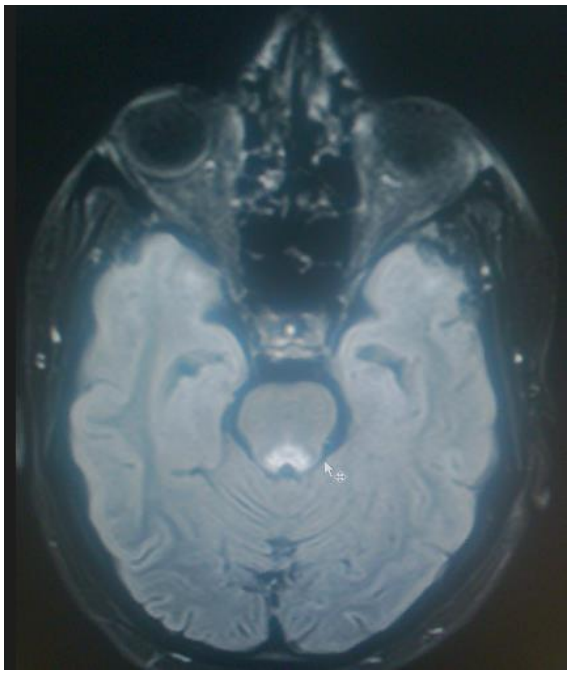


PHOTOGRAPH 11 - CASE OF THIRD CRANIAL NERVE PALSY – RIGHT EYE DUE TO ISCHEMIC ETIOLOGY..



PHOTOGRAPH 12 - CASE OF SUPERIOR ORBITAL FISSURE SYNDROME LEFT EYE DUE TO TRAUMA.





PHOTOGRAPH 13- CASE OF TOTAL OPHTHALMOPLEGIA BOTH EYE DUE TO PONTINE DEMYELINATION.

ANNEXURE 4 - KEY TO MASTER CHART

1. SI No: Serial number
2. IP No: In patient number
3. RE: Right eye
4. LE: Left eye
5. DV: Double vision
6. DP: Drooping of upper eyelid
7. AHT: Alleged history of trauma
8. DOV: Diminution of vision
9. FV: Fever
10. HA: Headache
11. PE: Pain in eye
12. D: Duration of symptoms
13. SPH: Significant past history
14. VA: Visual acuity
15. P: Ptosis
16. PR: Proptosis
17. ES: Esotropia
18. EX: Exotropia
19. EC: Ecchymosis
20. FAD: Fixed and dilated pupil
21. C: Chemosis
22. SCH: Subconjuctival hemorrhage

23. CD: Corneal dystrophy

24. HY: Hypertropia

25. FN: Fundus normal

26. 4CN: Fourth cranial nerve

27. 6CN : Sixth cranial nerve

28. 3PS: Third pupil sparing

29. 3PI: Third pupil involved

30. 3CC: Third complete

31. 3CI: Third incomplete

32. I/M: Isolated/Multiple

33. CT/MRI: Computer tomography/magnetic resonance imaging

34. CTN: CT/MRI normal

35. CTAB: CT /MRI abnormal

36. CTNT : CT/MRI not done

37. TR: Trauma

38. IS: Ischemic

39. AS: Aneurysm

40. NE: Neoplastic

41. ID: Idiopathic

42. D: Demyelination

43. RICT: Raised Intra cranial tension

44. CG: Congenital

45. IF: Inflammatory

PT NM	AGE	G	IP NO			MATER CHART																																												
													VA																																					
				DV	DP	AHT	DOV	FV	HA	PE	D	SPH	RE	LE	MAL	P	PR	ES	EX	EC	FAD	C	SCH	CD	HY	FN	FAB	4CN	6CN	3CC	3CI	3PS	3PI	FNO	FC	I	M	CAN	CTN	B	T	TR	IS	AS	NE	ID	D	RICT	CG	IF
BALAKRISHNA	60	MALE	986012	+	-	-	-	-	-	-	7 DAYS	DM	6/12p	6/18.	+	-	-	-	-	-	-	-	-	-	-	+	-	-	+	-	-	-	-	-	+	+	-	NO	-	+	-	-	+	-	-	-	-	-	-	-
BYRAMMA	58	FEMALE	5942	-	-	-	-	+	-	-	4 DAYS	BRONICAL ASTHMA	6/18p	6/9p	-	+	-	-	-	-	-	-	-	-	-	+	-	-	-	-	+	+	-	-	+	+	-	NO	-	-	+	-	-	-	-	-	-	-	-	+
CHANDRAKANTHA	51	MALE	962535	-	-	-	-	-	-	+	15 DAYS	K/C/O DM	6/12.	6/24.	-	+	-	-	-	-	-	-	-	-	-	-	+	-	-	+	-	+	-	-	+	+	-	NO	+	-	-	-	+	-	-	-	-	-	-	-
CHANDRAKANTHA	51	FEMALE	984931	+	-	-	-	-	-	-	20 DAYS	DM	6/12.	6/36.	+	-	-	-	-	-	-	-	-	-	-	+	-	-	+	-	-	-	-	+	-	+	-	NO	-	-	+	-	+	-	-	-	-	-	-	-
CHANGAMMA	81	FEMALE	984886	-	-	-	+	-	-	-	15 DAYS	HERPES ZOZTER OPHTHALMICUS	PL NEG	PL NEG	-	-	-	-	-	+	-	-	-	-	-	+	-	-	-	-	+	+	-	+	-	+	-	5	-	-	+	-	-	-	-	-	-	-	-	+
HANUMAKKA	70	FEMALE	1002112	-	-	-	+	-	-	-	3 MONTHS	CA CERVIX ON RADIOTHERAPHY AND CHEMO	CF-1M	PL +	+	-	-	-	-	-	-	-	-	-	-	-	+	-	+	-	-	-	-	-	-	+	-	NO	-	-	+	-	-	-	-	+	-	-	-	-
KOUSOLYAMMA	65	FEMALE	935687	+	-	-	-	-	-	-	1 MONTH	NAD	6/9.	6/9.	-	-	-	-	+	-	-	+	-	-	-	+	-	-	-	-	+	-	+	-	+	+	-	NO	+	-	-	+	-	-	-	-	-	-	-	-
LAKSHMAIAH	66	MALE	1007392	-	-	-	-	-	+	-	3 DAYS	DM	6/12P	6/12p	-	+	-	-	-	-	-	-	-	-	-	+	-	-	-	-	+	+	-	+	-	+	-	NO	-	-	+	-	+	-	-	-	-	-	-	-
LAKSHMI	34	FEMALE	1008940	-	-	-	-	-	+	-	MORNING	HTN	CF- 1M	CF - 1 M.	-	+	-	-	-	-	+	-	-	-	-	+	-	-	-	-	+	-	+	+	-	+	-	PLANTAR	-	+	-	-	-	-	-	-	-	-	-	+
MURLIDHARA	40	MALE	1015164	-	-	-	-	-	+	-	5 MONTHS	DROOPING OF R EYELID SINCE 5 YEARS	CF- 5M	6/18.	-	+	-	-	-	-	-	-	-	-	-	+	-	-	-	+	-	+	-	+	-	+	-	NO	+	-	-	-	-	-	-	+	-	-	-	-
NANJUNDAPPA	60	MALE	5094	+	-	-	+	-	-	-	8 DAYS	DM	6/12.	6/12.	+	-	-	-	-	-	-	-	-	-	-	+	-	-	+	-	-	-	-	+	-	+	-	NO	-	+	-	-	+	-	-	-	-	-	-	-
NARAYANAMMA	42	FEMALE	908957	+	-	-	+	-	+	-	4 MONTHS	NAD	6/6.	CF- 3M	-	-	+	-	-	-	-	-	-	-	-	+	-	+	+	+	-	+	-	-	+	-	+	2,5	-	+	-	-	-	-	+	-	-	-	-	-
NARAYANAPPPA	57	MALE	3171	+	-	-	-	-	-	-	3 DAYS	DM	6/12P	6/60.	+	-	-	-	-	-	-	-	-	-	-	-	+	-	+	-	-	-	-	+	-	+	-	NO	-	+	-	-	+	-	-	-	-	-	-	-
NATARAJ	65	MALE	30651	-	-	-	+	-	-	-	3 MONTHS	MULTIPLE SCLEROSIS	CF-1M	CF -4 M	+	-	-	-	-	-	-	-	-	-	-	-	+	-	+	-	-	-	-	+	-	+	-	NO	-	-	+	-	-	-	-	-	+	-	-	-
PAPAMMA	50	FEMALE	44080	-	-	+	-	-	-	-	2 DAYS	NAD	6/6p	6/12p	+	-	-	-	-	-	-	-	-	-	-	+	-	-	+	-	-	-	-	+	-	+	-	NO	-	-	+	+	-	-	-	-	-	-	-	-
PRITHVIRAJ	19	MALE	996358	-	-	+	-	-	-	-	1 DAY	NAD	6/6P	6/6P	-	-	-	-	-	+	-	-	-	-	-	+	-	-	-	-	+	+	-	+	-	+	-	NO	+	-	-	+	-	-	-	-	-	-	-	-
RAVIKUMAR	30	MALE	1010609	-	-	+	-	-	-	-	1 DAY	NAD	6/6.	6/12p	-	-	-	-	-	+	-	-	+	-	-	+	-	-	-	+	-	-	+	-	+	+	-	NO	-	+	-	+	-	-	-	-	-	-	-	-
SEENAMMA	40	FEMALE	999509		-	-	+	-	-	-	10 YEARS	NAD	CF- 2M	CF- 2M	-	-	-	-	-	-	-	-	-	+	-	-	+	-	+	-	-	-	-	+	-	-	+	2, 8	+	-	-	-	-	-	-	+	-	-	-	-
SHASHI KUMAR	25	MALE	998607	-	-	+	-	-	-	-	1 DAY	NAD	6/12.	6/9p	-	-	-	-	-	+	-	+	-	-	-	+	-	-	-	-	+	+	-	+	-	+	-	NO	-	+	-	+	-	-	-	-	-	-	-	-
SHIV KUMAR	30	MALE	3524	-	-	+	-	-	-	-	1 DAY	NAD	6/6P	PL -	-	+	-	-	-	-	+	-	-	-	-	+	-	-	-	+	-	-	+	-	+	+	-	2	-	+	-	+	-	-	-	-	-	-	-	-
SRIDHAR	30	MALE	28338	-	-	-	-	-	+	-	8 DAYS	PSHCOSOMATIC DISORDER	CF -5M	CF- 2M	+	-	-	-	-	-	-	-	-	-	-	-	+	-	+	-	-	-	-	-	+	+	-	NO	+	-	-	-	-	-	-	-	-	+	-	-
SRINIVAS	28	MALE	7340	-	-	+	-	-	-	-	1DAY	NAD	6/6.	6/6.	-	-	-	-	-	+	-	-	+	-	-	+	-	-	-	+	-	+	-	+	-	+	-	NO	+	-	-	+	-	-	-	-	-	-	-	-
SRINIVASAPPA	42	MALE	961641	-	-	-	-	+	-	-	8 DAYS	NAD	PL-	6/9.	-	+	-	-	-	-	+	-	-	-	-	-	+	+	+	-	+	-	+	+	-	-	+	2	-	+	-	-	-	-	-	-	-	-	-	+
SYED KALIL	40	MALE	991167	-	-	-	-	+	+	-	10 DAYS	DEFAULTED FROM ATT TREATMENT	6/6.	6/6P	+	-	-	-	-	-	-	-	-	-	-	+	-	-	+	-	-	-	-	+	-	+	-	NO	-	+	-	-	-	-	-	-	-	+	-	-
VAISHNAVI	7	FEMALE	5870	+	-	-	-	-	-	-	1MONTH	NAD	6/6P	6/6P	-	-	-	-	-	-	-	-	-	-	+	+	-	+	-	-	-	-	-	+	-	+	-	NO	+	-	-	-	-	-	-	-	-	-	+	-
VENKATASWAMY	35	MALE	513	-	-	+	-	-	-	-	1 DAY	NAD	6/12P	6/12P	+	-	-	-	-	-	-	-	-	-	-	+	-	+	-	-	+	+	-	+	-	-	+	NO	-	+	-	+	-	-	-	-	-	-	-	-
ARUNAGRACY	26	FEMALE	5578	-	+	-	-	-	-	-	5 DAYS	AMENORRHAEA 4 MONTHS	cf - 1M	CF-1M	-	+	-	-	-	-	-	+	-	-	-	-	+	+	+	+	-	-	+	+	+	-	+	NO	-	-	+	-	-	-	-	-	+	-	-	-
CHANDRASHEKAR	30	MALE	983875	-	+	-	+	-	-	-	8 DAYS	NAD	6/6.	HM+	-	+	-	-	-	-	+	-	-	-	-	+	-	+	+	+	-	-	+	-	+	-	+	NO	-	+	-	+	-	-	-	-	-	-	-	-
AMBRISH	26	MALE	37828	+	-	-	-	-	-	-	15 DAYS	NAD	6/18.	6/36P	+	-	-	-	-	-	-	-	+	-	-	+	-	-	-	-	+	-	+	+	-	+	-	NO	+	-	-	+	-	-	-	-	-	-	-	-
ARAB JAAN	65	MALE	9565	+	-	-	-	-	-	-	5 DAYS	DM	6/12P	6/18P	-	-	-	+	-	-	-	-	-	-	-	+	-	-	+	-	-	-	-	+	-	+	-	NO	-	+	-	-	+	-	-	-	-	-	-	-
VENKATESHAPPA	55	MALE	1018396	+	-	-	-	-	-	-	2 WEEKS	SUPRASELLAR MASS WITH HYDROCEPHALOUS	CF -1M	CF-5M	-	+	-	-	-	-	-	-	-	-	-	-	+	-	-	+	-	-	+	+	-	+	-	NO	-	+	-	-	-	-	+	-	-	-	-	-
SYRYANARAYAN	70	MALE	36340	-	+	+	-	-	-	-	2 DAYS	NAD	6/24P	6/12P	-	+	-	-	-	+	+	-	+	-	-	+	-	-	-	+	-	-	+	-	+	+	-	2	-	+	-	+	-	-	-	-	-	-	-	-
SRLAKKALAPPA	60	MALE	122056	-	-	-	+	-	-	-	3 DAYS	DM	CF-5M	CF-5M	-	+	-	-	+	-	+	-	-	-	-	-	+	-	-	-	+	-	+	+	-	+	-	NO	-	-	+	-	+	-	-	-	-	-	-	-
SRINIVAS	55	MALE	88598	-	-	-	-	-	+	-	3 MONTHS	DM	6/18P	6/12P	-	+	-	-	-	-	+	-	-	-	-	+	-	-	-	+	-	-	+	+	-	+	-	NO	-	-	+	-	+	-	-	-	-	-	-	-
SRIDEVI	36	FEMALE	929663	+	-	-	-	-	+	-	2 WEEKS	NAD	6/24P	6/18.	+	-	-	+	-	-	-	-	-	-	-	-	+	-	+	-	-	-	-	+	-	+	-	NO	-	+	-	-	-	-	-	-	-	-	-	+
NARAYANAMMA	60	FEMALE	42278	+	-	-	-	-	-	-	2 WEEKS	CA BUCCAL MUCOSA	CF- 5M	6/60.	+	-	-	+	-	-	-	-	-	-	-	+	-	-	+	-	-	-	-	-	+	+	-	7,8	-	+	-	-	-	-	+	-	-	-	-	-
MANJUNATHREDDY	44	MALE	90740	-	-	+	-	-	-	-	1 DAY	NAD	6/12P	6/9P	-	-	-	-	-	+	-	-	+	-	-	+	-	-	+	-	-	-	-	-	+	+	-	NO	-	-	+	+	-	-	-	-	-	-	-	-
LASHMI NARASHIMA	24	MALE	99706	-	+	+	-	-	-	-	1 DAY	NAD	6/6.	6/6.	-	+	-	-	-	-	-	+	+	-	-	+	-	-	+	-	+	+	-	-	+	+	-	NO	+	-	-	+	-	-	-	-	-	-	-	-
LAKSHMIDEVAMMA	20	FEMALE	85721	-	-	-	-	+	+	-	5 DAYS	NAD	6/24.	6/36.	+	-	-	-	-	-	-	-	-	-	-	-	+	-	+	-	-	-	-	+	+	+	-	NO	-	+	-	-	-	-	-	-	-	-	-	+
KRISHNAPPA	55	MALE	11384	-	-	+	-	-	-	-	4 DAYS	NAD	CF-5M	6/24P	-	+	-	-	-	+	+	-	+	-	-	+	-	-	-	-	+	-	+	+	-	+	-	NO	+	-	-	+	-	-	-	-	-	-	-	-
HANUMAPPA	40	MALE	161840	+	-	+	+	-	-	+	1 DAY	NAD	6/12P	6/12P	+	-	-	-	+	-	-	-	-	-	-	+	-	-	-	-	+	+	-	-	+	+	-	NO	-	+	-	+	-	-	-	-	-	-	-	-
HARI	45	MALE	88147	+	+	-	-	-	+	-	10 DAYS	HTN	6/9P	6/12.	-	+	-	-	+	-	-	-	+	-	-	+	-	-	-	-	+	+	-	+	-	+	-	NO	-	-	+	-	+	-	-	-	-	-	-	-
IAKUMAR	19	MALE	38806	+	+	+	-	-	-	+	3 DAYS	NAD	CF>3M	PL NEG	-	+	-	-	-	+	-	+	-	-	-	-	+	-	+	+	-	+	-	-	+	-	+	2	-	+	-	+	-	-	-	-	-	-	-	-
KANTHA	64	FEMALE	78284	-	-	-	+	-	-	-	2 MONTHS	DM, HTN	CF - 4M	CF - 5M	+	-	-	-	-	-	-	-	-	-	-	+	-	-	-	-	+	+	+	-	+	+	-	NO	+	-	-	-	+	-	-	-	-	-	-	-
GANESH	26	MALE	50535	+	+	-	+	-	-	-	5 MONTHS	DM,HTN	CF- 2M	CF-4M	-	+	-	-	-	-	+	-	-	-	-	-	+	-	-	+	-	-	+	+	-	+	-	NO	-	-	+	-	+	-	-	-	-	-	-	-
GANGAMMA	38	FEMALE	86754	-	-	+	-	-	-	-	2 DAYS	NAD	6/12P	6/9P	-	-	-	+	-	-	-	-	+	-	-	+	-	-	+	-	-	-	-	+	-	+	-	NO	+	-	-	+	-	-	-	-	-	-	-	-
GOPALAPPA	45	MALE	72845	+	+	-	-	-	-	-	4 DAYS	ALCOHOLIC	6/9P	6/12P	-	+	-	-	-	-	-	-	-	-	-	+	-	-	-	-	+	+	-	+	-	+	-	NO	-	+	-	-	+	-	-	-	-	-	-	-
ANAND KUMAR	30	MALE	19978	-	+	+	-	-	-	-	2 DAYS	NAD	6/12.	6/9.	+	-	-	-	-	-	-	-	-	-	-	+	-	-	-	-	+	+	-	+	-	+	-	NO	+	-	-	+	-	-	-	-	-	-	-	-
BABU	63	MALE	38476	-	+	-	-	-	+	-	20 DAYS	SMOKING , ALCOHOLIC	6/9P	6/9P	-	+	-	-	-	-	-	-	-	-	-	+	-	-	+	-	+	-	+	+	-	-	+	NO	-	+	-	-	+	-	-	-	-	-	-	-
CHINNAGANGAMMA	65	FEMALE	91157	-	-	-	+	-	+	-	1 YEAR	NAD	CF -1M	CF - 1M	-	-	-	+	-	-	-	-	-	-	-	+	-	-	+	-	-	-	-	-	+	+	-	NO	-	-	+	-	-	-	-	+	-	-	-	-
MANJUNATH	45	MALE	927767	+	+	+	-	-	-	+	2 DAYS	NAD	6/36.	6/24.	-	+	-	-	-	+	+	-	-	-	-	+	-	-	-	+	-	-	+	-	+	+	-	NO	-	+	-	+	-	-	-	-	-	-	-	-
MUNIRAJU	45	FEMALE	931100	-	+	+	-	-	-	-	2 DAYS	NAD	6/24P	6/24P	-	+	-	-	+	-	-	-	-	-	-	+	-	+	-	-	+	-	+	-	+	-	+	NO	-	+	-	+	-	-	-	-	-	-	-	-
RATHNAMMA	36	FEMALE	857803	-	-	-	+	+	+	-	8 DAYS	HIV AND RT SIDED FACIAL CELLULITIS	CF-4M	6/6P	-	+	+	-	-	+	+	-	-	-	-	-	+	-	-	+	-	-	+	+	-	+	-	2	-	-	-	-	-	-	-	-	-	-	-	+
GAYATHRAMMA	38	FEMALE	858651	+	-	-	+	-	-	-	2 DAYS	CHIKANGUNIYA	HM +	CF- 2M	-	+	-	+	-	-	-	-	-	-	-	-	+	-	-	-	+	+	-	+	-	+	-	NO	-	-	+	-	-	-	-	-	-	-	-	+
NETHRAVATHI	26	FEMALE	908906	-	-	-	+	-	+	-	8 DAYS	NAD	6/6P	6/6P	-	-	-	-	+	-	-	-	-	-	-	-	+	-	+	-	-	-	-	+	+	+	-	NO	+	-	-	-	-	-	-	-	-	+	-	-
CHAITHANYA RAVI	30	MALE	893206	+	-	-	+	-	-	-	1 YEAR	FOOD POISONING	6/12P	6/6P	+	-	-	-	-	-	-	-	-	-	-	+	-	-	+	-	+	+	-	+	-	+	-	NO	-	-	+	-	-	-	-	-	-	-	-	+
MANJULA	25	FEMALE	878213	+	-	-	-	-	-	-	7 DAYS	DENGUE FEVER	6/12P	6/6P	-	-	-	+	-	-	-	-	-	-	-	-	+	-	+	-	-	-	-	+	-	+	-	NO	-	-	+	-	-	-	-	-	-	-	-	+
NARAYANAMMA	42	FEMALE	908957	+	-	+	+	-	-	-	1 MONTH	NAD	6/6P	HM+	-	-	-	+	-	-	-	+	-	-	+	+	-	-	-	-	+	+	-	-	+	+	-	NO	-	-	+	+	-	-	-	-	-	-	-	-
ASIF DOULA	45	MALE	932576	-	+	-	-	-	+	-	3 MONTHS	INFARACTION IN POSTERIOR CIRCULATIONS	6/12P	6/60.	-	+	-	-	-	-	-	-	-	-	-	+	-	+	-	-	+	+	-	-	+	-	+	7	-	+	-	-	+	-	-	-	-	-	-	-
SUDHAKAR	40	MALE	944690	+	-	-	+	-	-	-	3 DAYS	NAD	CF- 5M	CF - 4M	-	-	-	-	-	-	-	-	+	-	+	+	-	+	-	-	-	-	-	+	-	+	-	NO	-	+	-	+	-	-	-	-	-	-	-	-
GOUSIYA	13	FEMALE	947460	-	+	-	+	+	-	+	10 DAYS	NAD	6/6P	PL -	-	+	+	-	-	-	-	-	-	-	-	-	+	+	+	+	-	-	+	-	+	-	+	2	-	+	-	-	-	-	+	-	-	-	-	-
CHANDRAN	34	MALE	947046	-	-	-	+	-	-	+	1 MONTH	DM	6/18P	HM+	-	+	-	-	-	-	-	-	-	-	-	+	-	+	+	-	+	+	-	+	-	-	+	NO	+	-	-	-	+	-	-	-	-	-	-	-
KAMALESH	4	MALE	917774	+	-	+	-	-	-	+	3 DAYS	NAD	6/12.	6/18P	-	-	-	+	-	+	-	-	-	-	-	+	-	-	+	-	-	-	-	-	+	+	-	-	-	+	-	+	-	-	-	-	-	-	-	-
MANJULA	50	FEMALE	920725	+	-	-	-	-	+	-	15 DAYS	BRONCHIAL ASTHAMA	6/60.	6/24P	-	-	-	+	-	-	-	-	-	-	-	+	-	-	+	-	-	-	-	+	-	+	-	NO	-	+	-	-	-	-	+	-	-	-	-	-
RAMADAS	63	MALE	956743	+	-	-	-	-	-	-	10 DAYS	NAD	6/12P	6/24P	-	+	-	-	-	-	-	-	-	-	-	+	-	+	+	+	-	+	-	+	+	-	+	NO	-	-	+	-	-	-	-	+	-	-	-	-
MANJUNATH	8	MALE	170861	-	-	-	-	+	+	-	6 DAYS	NAD	HM+	HM+	-	+	+	-	-	-	-	-	-	-	-	-	+	-	+	-	-	-	-	+	+	+	-	NO	+	-	-	-	-	-	-	-	-	+	-	-
SUMAIAH KHANUM	7	FEMALE	176533	+	-	-	-	+	-	-	8 DAYS	NAD	6/6P	6/6P	-	-	-	+	-	-	-	-	-	-	-	+	-	-	-	-	+	+	-	+	-	+	-	NO	-	-	+	-	-	-	-	+	-	-	-	-
HARITHA	15	FEMALE	917203	+	-	-	-	-	+	-	10 DAYS	NAD	6/6P	6/6P	+	-	-	-	-	-	-	-	-	-	-	+	-	-	+	-	-	-	-	+	+	+	-	NO	-	+	-	-	-	-	-	-	-	-	-	+
VISHNU	34	MALE	917838	-	-	+	-	-	-	-	3 DAYS	NAD	6/12P	6/18P	-	-	-	+	-	-	-	-	+	-	-	+	-	-	+	-	-	-	-	+	-	+	-	NO	+	-	-	+	-	-	-	-	-	-	-	-
SHANTHAKUMAR	45	MALE	809264	+	-	+	-	-	-	-	2 DAYS	NAD	6/6P	6/6P	-	-	-	-	-	-	-	-	-	-	+	+	-	+	-	-	-	-	-	-	+	+	-	NO	-	+	-	+	-	-	-	-	-	-	-	-
POOJA	11	FEMALE	914600	+	-	-	+	+	-	-	7 DAYS	NAD	6/6P	6/6P	+	-	-	-	-	-	-	-	-	-	-	+	-	+	-	-	-	-	-	-	+	+	-	NO	-	+	-	-	-	-	-	-	-	-	-	+
RAMAKRISHNA	42	MALE	903554	+	+	+	-	-	-	-	1DAY	NAD	6/12P	6/12P	-	+	-	-	-	+	+	-	-	-	-	-	+	-	-	+	-	-	+	+	-	+	-	NO	-	+	-	+	-	-	-	-	-	-	-	-
MUNIRATHNAMMA	35	FEMALE	889947	-	+	-	+	-	+	-	1 MONTH	NAD	6/24P	6/6P	-	+	-	-	-	-	+	-	-	-	-	+	-	-	+	+	-	-	+	+	-	-	+	NO	-	+	-	-	-	-	-	-	-	-	-	+
VIJAYA	40	FEMALE	946251	+	+	+	-	-	-	-	2 DAYS	NAD	6/24P	6/12P	-	+	-	-	-	+	-	+	-	-	-	+	-	-	-	-	+	+	-	+	-	+	-	NO	-	+	-	+	-	-	-	-	-	-	-	-