wjpmr, 2020,6(3), 107-124

SJIF Impact Factor: 5.922

WORLD JOURNAL OF PHARMACEUTICAL AND MEDICAL RESEARCH

www.wjpmr.com

Research Article

ISSN 2455-3301

WJPMR

A STUDY OF OCULAR MOVEMENTS AND PUPILS IN ACUTE STROKE WITH ITS CLINICAL CORRELATION AND IMAGING

Dr. Arthi P. S.[1] and Dr. E. Dinesh Ragav[2*]

[1]Assistant Professor, Department of General Medicine, SSSMCRI, Ammapettai, Kancheepuram. Tamil Nadu.
[2]Assistant Professor, Department of General Medicine, SSSMCRI, Ammapettai, Kancheepuram, Tamil Nadu.

***Corresponding Author: Dr. E. Dinesh Ragav**

Assistant Professor, Department of General Medicine, SSSMCRI, Ammapettai, Kancheepuram, Tamil Nadu, India.

Article Received on 26/12/2019 **Article Revised on 16/01/2020** **Article Accepted on 06/02/2020**

INTRODUCTION

Cerebrovascular disease or stroke rank first in frequency and importance, among all the neurological diseases of adult life. It is the third most common cause of death in the world. Every year there are approximately 700,000 cases of stroke-roughly 600,000 ischemic lesions and 100,000 hemorrhages, intracerebral or subarachnoid-with 1,75,000 fatalities from these causes combined.[1]

Just as the "Face is the index of the mind" to a neurologist "the eyes are the index of the brain", the abnormalities of eye movement are frequent manifestations of cerebrovascular disease. Eyes is an important organ which most of us take it for granted. It's a highly specialized sense organ which unlike most organ of body, is available to external examination. About 20% of population have pupils that are slightly unequal in size that respond equally to light.

Different neuroanatomical pathways are involved in the control of pupil, the integrity and the functionality of these neurological pathways can be often be ascertained through the analysis and interpretation of pupillary behavior This makes the pupil size and the pupillary light reflex an important factor to be considered in many clinical conditions. More specifically, the location of the pupillomotor nuclie and efferent occulomotor nerve is important for assessing the onset of descending transtentorial herniation and brainstem compression its has also been shown through blood flow imaging the pupillary changes in neurological patients in ICU are highly correlated with brainstem oxygenated and perfusion or ischemia.[3]

Abnormal ocular movements may occur after injury at several levels of the neuraxis. Unilateral supranuclear disorders of gaze tend to be transient; bilateral disorders more enduring. Nuclear disorders of gaze also tend to be enduring and are frequently present in association with long tract signs and cranial nerve palsies on opposite sides of the body. Nystagmus is a reliable sign of posterior fossa or peripheral eighth nerve pathology.[4] Several of these signs have characteristics which enable the clinician to localize the site as well as the probable nature of the underlying pathology.

One may determine whether the motility disturbance is due to cerebral hemispheric or brainstem disease. Localization is aided by knowledge of central ocular motor anatomy and physiology which is extensively reviewed elsewhere.

Recent research has shown that, the eye movement and pupil examination is more accurate than MRI in predicting stroke and was able to identify all patients in the study who had a stroke, whereas an MRI conducted in the first day or two after hospital admission missed more than one in ten strokes. Hence this study to observe the ocular movements and pupils in acute stroke patients with its clinical correlation and imaging.[5]

WHO define stroke as "rapidly developing clinical signs of focal (or global) disturbance of cerebral function, with symptoms lasting 24 hours or longer or leading to death, with no apparent cause other than of vascular origin".

Strokes are broadly categorized as ischemic or hemorrhagic. Ischemic stroke is due to occlusion of a cerebral blood vessel and causes cerebral infarction. The resultant neurologic syndrome corresponds to a portion of the brain that is supplied by one or more cerebral vessels.

Knowledge of the stroke syndromes, the signs and symptoms that correspond to the region of brain that is supplied by each vessel, allows a degree of precision in determining the particular vessel that is occluded and from the temporal evolution of the syndrome, the underlying cause of vascular occlusion can be deduced.

Ischemic strokes are classified by the underlying cause of the vascular occlusion.

One of three main processes is usually operative:

1. Atherosclerosis with superimposed thrombosis affecting large cerebral or extracerebral blood vessels,
2. Cerebral embolism, and
3. Occlusion of small cerebral vessels within the parenchyma of the brain.

Blood Supply of Brain

Brain is supplied by two internal carotid arteries. Their branches anastamose in base of skull forming "circle of Willis".

Cerebral collateral circulation. Aortic arch, Brachiocephalic artery, Subclavian artery. The four major cerebral vessels, i.e. the carotid system, and the vertebro-basilar system, unite at the base of the brain to form a hexagonal 'Circle of Willis'. These vessels form a network of extensive anastomoses between themselves, in the neck (pre-Willisian anastomoses). Their cerebral branches: the anterior cerebral, the middle cerebral, and the posterior cerebral arteries anastomose with themselves distal to the circle of Willis (post-Willisian anastamoses).

Internal Carotid Artery

Internal carotid artery begins at the bifurcation of common carotid artery at carotid sinus. It enters the base of skull through carotid canal of temporal bone. It runs in cavernous sinus and emerges on the medial side of anterior clinoid process by perforating duramater. It ends by dividing into anterior and middle cerebral arteries in the medial end of lateral cerebral sulcus.

Branches of internal carotid artery

1. **Ophthalmic artery-** It supplies eye, orbital structures, frontal area of scalp, ethmoid and frontal sinuses and dorsum of nose.
2. **Posterior communicating artery** - It supplies posteriorly to join posterior cerebral artery forming circle of Willis.
3. **Choroidal artery** - It supplies curs cerebri, lateral geniculate body optic tract and internal capsule.
4. **Anterior cerebral artery**- Cortical branches supply medial surface medial surface of cortex up to parieto occipital sulcus and 1 inch of cortex in lateral surface. Central branches supply lentiform and caudate nucleus and internal capsule.
5. **Middle cerebral artery** - Cortical branches supply entire lateral surface of cerebral hemisphere except ACA territory and PCA territory. (Supplies all motor areas except leg area). Central branches supplies lentiform and caudate nucleus and internal capsule.

Vertebral Artery

It arises from first part of sub-clavian artery and ascends in foramina of transverse process in upper six cervical vertebrae. It enters skull through foramina magna. It ends in lower surface of pons by joining with vertebral artery of opposite side forming basilar artery.

Branches of Vertebral artery

1. **Meningeal branches** - It supplies bone and dura of posterior cranial fosse.
2. **Posterior spinal arteries** - It supplies posterior one thirds of spinal cord and posterior spinal roots.
3. **Anterior spinal arteries** - It supplies anterior two thirds of spinal cord and anterior spinal roots.
4. **Posterior inferior cerebellar artery** - It supplies vermis, central nuclei of cerebellum, undersurface of cerebellar hemisphere, medulla and choroid plexus.
5. **Medullary artery** - It supplies medulla oblangata.

Basilar Artery

It is formed by union of two vertebral arteries at lower border of pons.

Branches of Basilar artery

1) **Pontine branches** - It supplies the entire pons.
2) **Labrynthine artery** - Internal ear, Internal acoustic meatus and labrynth.
3) **Anterior inferior cerebellar artery** - It supplies anterior and inferior surface of cerebellum and sometimes pons and upper medulla.
4) **Superior cerebellum artery** - Superior surface of cerebellum, pons and pineal gland.
5) **Posterior cerebral artery** - It is the terminal branch of basilar artery. It joins with posterior communicating artery and forms circle of Willis.

Cortical branches supply infero lateral and medial surface of temporal lobe and medial+lateral surface of occipital lobe (visual cortex).

Cortical branches supply thalamus, lentiform nucleus, midbrain, pineal gland, medial geniculate bodies, choroid plexus of 3rd ventricle.

Venous Drainage

Venous drainage of brain is by external and internal cerebral veins. External cerebral vein is formed by fusion of superior and middle cerebral vein which drains for blood from lateral surface and anterior artery territory which drains into superior saggital, cavernous and straight sinus.

Internal cerebral veins are formed by fusion of thalamostriate and choroid veins draining blood into great cerebral vein and then into straight sinus.

Pupils

Sympathetic impulses dilate the pupils. Fibres in the nasociliary nerve pass to *dilator pupillae.* These arise from the superior cervical ganglion at C2.

Sympathetic preganglionic fibres to the eye (and face) originate in the hypothalamus, pass uncrossed through the midbrain and lateral medulla, and emerge finally from the spinal cord at Tl (close to the lung apex) and form the superior cervical ganglion at C2.

Post ganglionic fibres leave the ganglion to form a plexus around the carotid artery bifurcation. Fibres pass to the pupil in the nasociliary nerve from part of this plexus surrounding the *internal* carotid artery. Those fibres to the face (sweating and piloerection) arise from the part of the plexus surrounding the *external* carotid artery. This arrangement has some clinical relevance in Horner's syndrome. Parasympathetic impulses cause pupillary constriction. Fibres in the short ciliary nerves arise from the ciliary ganglion and pass to *sphincter pupillae*, causing constriction.

The light reflex
Afferent fibres (1) in each optic nerve (some crossing in the chiasm) pass to both lateral geniculate bodies (2) and relay to the Edinger-Westphal nuclei (4) via the pretectal nucleus (3). Efferent (parasympathetic) fibres from each Edinger- Westphal nucleus pass via the third nerve to the ciliary ganglion (5) and thence to the pupil (6). Light constricts the pupil being illuminated (direct reflex) and, by the consensual reflex, the contralateral pupil.

Afferent pathway
(1) A retinal image generates action potentials in the optic nerve.
(2) These travel via axons, some of which decussate at the chiasm and pass through the lateral geniculate bodies.
(3) Synapse at each pretectal nucleus.

Efferent pathway
(4) Action potentials then pass to each Edinger – Westphal nucleus of III.
(5) Then, via the ciliary ganglion.
(6) Lead to constriction of pupil.

The convergence reflex
Fixation on a near object requires convergence of the ocular axes and is accompanied by pupillary constriction.

Afferent fibres in each optic nerve, which pass through both lateral geniculate bodies, also relay to the convergence centre. This centre receives la spindle afferent fibres from the extraocular muscles - principally medial recti - which are innervated by the third nerve.

The efferent route is from the convergence centre to the Edinger-Westphal nucleus, ciliary ganglion and pupils.

Voluntary or reflex fixation on a near object is thus accompanied by appropriate convergence and pupillary constriction. A darkened room makes all pupillary abnormalities easier to see.

Physiological changes and old age
A slight difference between the size of each pupil is common (physiological anisocoria) at any age. The pupil tends to become small (3-3.5 mm) and irregular in old age (senile miosis); anisocoria is more pronounced. The convergence reflex becomes sluggish with ageing and a bright light becomes necessary to demonstrate constriction.

Afferent pupillary defect
A blind left eye, for example following complete optic nerve section, has a pupil larger than the right.

The features of a left afferent pupillary defectare
- The left pupil is unreactive to light (i.e. the direct reflex is absent).
- The consensual reflex (constriction of right pupil when the left is illuminated) is absent. Conversely, the left pupil constricts when light is shone in the intact right eye, i.e. the consensual reflex of the right eye remains intact.

Relative afferent pupillary defect (RAPD)
When there has been incomplete damage to one afferent pupillary pathway (i.e. of one optic nerve *relative* to the other), the difference between the pupillary reaction, and the relative impairment on one side is called a relative afferent pupillary defect (RAPD). The sign can provide evidence of an optic nerve lesion, when, for example, retrobulbar neuritis occurred many years previously and there has been complete clinical recovery of vision.

After previous left retrobulbar neuritis
1) Light shone in the left eye causes both left and right pupils to constrict.
2) When light is shone into the intact right eye, both pupils again constrict (i.e. right direct and consensual reflexes are intact).
3) When the light is swung back to the left eye, its pupil dilates slightly, relative to its previous size.

A left RAPD by the *swinging light test*, showing that the consensual reflex is stronger than the direct, indicates residual damage in the afferent pupillary fibres of the left optic nerve.

3,4,6 Cranial Nerves
This group of three cranial nerves controls the upper eyelid, eye movements and pupils. Each has a long intracranial course.

The Extra Ocular Muscles
The names, positions and actions of the extra ocular muscles are illustrated and discussed in below mentioned figure. The main points to note are:
1) The medial rectus muscle of one eye and the lateral rectus muscle of the other work as a yoked pair to produce lateral eye movements.
2) The vertically acting rectus muscles are at their most effective when the eye is abducted, that is, looking outwards, as in this position the line of pull of the muscles is along the vertical axis of the eye.
3) In the same way the oblique muscles are maximally effective when the eye is adducted, that is, looking inwards, as their line of pull is then along the vertical axis of the eye.

Abnormalities of eye movements in Cerebrovascular Disease

Abnormalities of eye movement are frequent manifestations of cerebrovascular disease. Several of these signs have characteristics which enable the clinician to localize the site as well as the probable nature of the underlying pathology. One may determine whether the motility disturbance is due to cerebral hemispheric or brainstem disease.

Localization is aided by knowledge of central ocular motor anatomy and physiology which is extensively reviewed elsewhere. For the purpose of this discussion, it is important to recall that the cranial nerve ocular motor nuclei (III, IV, VI) are the final common pathways in the brainstem for information arising at several levels of the neuraxis. Disorders of ocular motility may, therefore, reflect pathological processes which do not directly involve these nuclear groups.

Horizontal Gaze

Rapid horizontal eye movements (saccades) are generally believed to originate in the frontal lobes. The pathway for horizontal eye movement control (frontomesencephalic pathway) is probably polysynaptic, composed of many neurons, and passes near the internal capsule and basal ganglia to the midbrain. The pathway crosses at the junction of the midbrain and upper pons to terminate in the contralateral pontine paramedial reticular formation (PPRF). The PPRF lies ventral to the sixth nerve nucleus and ventral and lateral to the medial longitudinal fasciculus (MLF) in the tegmentum of the pons.

From the PPRF, horizontal eye movement commands are relayed to interneurons and motor neurons within the sixth nerve nucleus on the same side. The interneurons within the sixth nerve nucleus subsequently project to the contralateral medial rectus subdivision of the oculomotor nucleus via the medial longitudinal fasciculus. A fast eye movement "command" generated in the left frontal lobe will result in a horizontal saccade to the right with activation of the right lateral rectus and left medial rectus via the pathways described.

Smooth pursuit or "following" eye movements, which allow accurate visual tracking of moving targets, are initiated in the occipitoparietal region and presumably descend in a polysynaptic occipitomesencephalic pathway to the PPRF on the same side. A slow eye movement "command" generated in the right occipitoparietal region will result in horizontal pursuit to the right with activation of the right lateral rectus and left medial rectus.

In addition to saccadic and pursuit eye movements, vestibuloocular reflexes are channeled through the PPRF, making it an important relay for several different modalities affecting ocular motor function. An acute, destructive lesion involving the right frontal lobe will cause a left hemiparesis and leftward gaze palsy.

The eyes, "driven" by the remaining normal left hemisphere, will be deviated to the right (i.e., the eyes look toward the side of the lesion). The patient will at first be unable to generate rapid eye movements to the left. Because the pursuit pathways originating in the occipitoparietal region may not be involved with a small frontal lesion, an alert patient will be able to follow slowly moving targets in either horizontal direction.

In addition, appropriate tonic ocular deviations may be produced by either caloric irrigation or the oculocephalic (doll's head) maneuver (the tendency of the eyes to maintain their direction of gaze when the head is passively rotated or flexed).

When the destructive lesion is isolated to one frontal lobe, the paralysis of horizontal saccades is *transient*, resolving in a matter of days, usually before any improvement is noted in the hemiparetic extremities.

The role of making rapid eye movements is eventually taken over by the remaining intact hemisphere.

An irritative lesion of the frontal lobe, such as that associated with a seizure focus, will drive the eyes to the opposite side and is accompanied by clonic movements of the opposite extremities.

Tonic deviation of the eyes lasts only as long as the seizure, and once the patient becomes fully alert, a full range of horizontal extraocular movements returns.

A lesion in the brainstem which involves the PPRF will result in a pontine gaze palsy. These lesions are most often secondary to vascular occlusive or demyelinating disease and are often bilateral and asymmetrical, encompassing several brainstem nuclei and tracts.

Since the PPRF is the final prenuclear substrate for ipsilateral gaze, a unilateral lesion involving this area will result in a loss of *all* horizontal movements (saccades and pursuit) to the affected side.

A patient with a *left-sided* PPRF lesion will be unable to execute voluntary rapid eye movements or to pursue a slowly moving target to the *left*. In addition, cold caloric irrigation of the left ear, which normally causes a right bearing nystagmus, will evoke no response. Similar stimulation on the right will cause a slow tonic deviation of the eyes to the right with an absent fast phase of nystagmus to the left. An incomplete lesion of the PPRF will cause a gaze paresis which may or may not be associated with abnormal caloric responses.

Bilateral infarcts in the tegmentum of the brain-stem involving the PPRF will result in complete paralysis of all voluntary and reflex horizontal eye movements. This

condition is most commonly observed after massive hypertensive pontine hemorrhage, but it can be seen as an isolated acute sign in a small infarct of the PPRF. In contrast to the transient nature of gaze palsies caused by lesions of the cerebral hemispheres, a destructive lesion of the PPRF usually results in *lasting* paralysis of ipsilateral gaze.

Because of the proximity of the PPRF to other important ocular motor structures, there may be involvement of the adjacent sixth nerve nucleus and/or the medial longitudinal fasciculus. A lesion which involves the PPRF and ipsilateral MLF results in what has been designated a "one and a half" syndrome. In this disorder a right-sided lesion will cause a horizontal gaze palsy to the right and an internuclear ophthalmoplegia during gaze to the left.

The ophthalmoplegia is characterized by failure or slowing of adduction of the right eye during leftward gaze as well as nystagmus of the abducting left eye. Thus, in a one and a half syndrome, the only remaining movement may be abduction of the eye on the side opposite the lesion. Although a distinction has been made in the past between "anterior" (midbrain) and "posterior" (pontine) involvement of fasciculus fibers in an internuclear ophthalmoplegia, it is often not possible clinically to localize the level at which the MLF is interrupted. Unilateral involvement of the MLF is secondary to vascular disease in approximately 70% of cases.

The onset is often sudden in an older individual and associated with other brainstem symptoms, such as vertigo, ataxic gait, or diplopia. The deficit generally resolves over a period of two to three months. Bilateral internuclear ophthalmoplegia has been reported to occur more frequently in demyelinating disease, although other authors have found vertebrobasilar disease to be more common.

Certainly, any patient with a history of transient loss of consciousness who is found to have bilateral ophthalmoplegia should be suspected of having vascular disease since transient loss of consciousness is extremely rare in demyelinating disease. Disturbance of ocular motor function secondary to brainstem pathology may also include nystagmus with or without prominent symptoms of vertigo. The characteristics of nystagmus which help distinguish peripheral eighth nerve from central dysfunction have recently been reviewed.

Although nystagmus is considered to be one of the most reliable signs of posterior fossa pathology, it may also occur with vascular lesions of the cerebral hemisphere. During recovery from a gaze palsy secondary to a frontal lobe lesion, patients pass through a phase when they are able to make a gaze movement away from the side of the lesion but cannot sustain the deviated position. As the eyes drift back toward the side of the lesion, a corrective saccade may reposition them in an eccentric position. Repetition of this pattern results in nystagmus with the fast phase toward the side opposite the lesion, a pattern which has been termed "gaze paretic nystagmus."

Vertical Gaze

Eye movements in the vertical plane are probably generated by simultaneous bilateral activation of the frontal or occipital cortex. The exact route taken by impulses involved in vertical gaze is not known in detail, although there is evidence that vertical gaze pathways converge on the periaqueductal region just ventral to the collicular plate.

Perhaps the most frequently encountered disorder of vertical gaze is the dorsal midbrain syndrome (Parinaud's syndrome), a constellation of neuroophthalmological signs which include abnormalities of vertical gaze, pupillary responses, accommodation, and vergence. It is commonly seen in association with pineal, midbrain, or third ventricular tumors and midbrain infarction.

Upward saccades and pursuit are affected first, then downward movements. Attempts at upward saccades may elicit convergence-retraction nystagmus which appears as rhythmic bilateral retraction and/or convergence of the eyes.

The abnormality may be demonstrated to best advantage by utilizing downgoing optokinetic targets, each target providing a stimulus for an upward saccade. The pupils are often large in the dorsal midbrain syndrome and react better to near than to direct light (light-near dissociation). In addition, there may be pathological lid retraction (Collier's sign). The paralysis of downgaze frequently observed in the dorsal midbrain syndrome may also be seen as an isolated ocular motility defect on rare occasion. The pathology in these cases is located bilaterally, dorsal and medial to the red nuclei near the ventral periaqueductal gray.

AIM AND OBJECTIVES

Aims

To study the ocular movements and pupils in acute stroke patients with its clinical correlation and imaging**.**

Objectives

To study the comprehensive clinical profile of the acute stroke patients with particular importance to ocular movements and pupils in this sample of patients and its clinical correlation and imaging.

MATERIALS AND METHODS

This prospective study will be conducted in Rajah Muthiah Medical College and Hospital (RMMCH) during the period from October 2013 to October 2015. The study sample will include 50 patients with acute stroke confirmed by CT/MRI findings of both sexes and who belong to age group of 20 to 80 years from

RMMCH. A detailed clinical history will be taken for these patients who are included in this study. All these patients will be examined thoroughly with particular importance to ocular movements and pupils. All these patients will undergo a CT brain or MRI brain.

Inclusion Criteria

1) Patients in age group of 20-80 years of both sexes.
2) Patients who gets admitted within 48 hours of stroke onset.
3) All the patients will be examined thoroughly with particular importance to ocular movements and pupils.
4) All the patients will undergo a CT Brain or MRI Brain.
5) All the patients will be re-examined every 12 hours for 72 hours.

Exclusion Criteria

1. Patients admitted after 48 hours of stroke onset
2. Patients who are in age group less than 20years and more than 80years
3. Patients with previous history of stroke
4. Patients who comes with any acute neurological syndrome other than stroke
5. Patients with previous or present ophthalmological lesions.

Investigations

CT Brain/MRI Brain

OBSERVATIONS AND RESULTS

Table 1: Age Wise Distribution.

Age	No. of patients (n=50)	Percentage (%)
31-40	7	14
41-50	6	12
51-60	18	36
61-70	12	24
71-80	7	14

Table 2: Gender Wise Distribution.

Gender	No. of patients (n = 50)	Percentage (%)
Male	**36**	**72**
Female	**14**	**28**

Table 3: Neurological findings.

Neurological findings	No. of patients (n=50)	Percentage (%)
Level of Consciouness	**16**	**32**
Best Language	**21**	**42**
Dysarthria	**9**	**30**
Best Gaze	**18**	**36**
Facial Palsy	**50**	**100**
Motor	**50**	**100**

Table 4: **Level of Consciousness.**

Level of Consciousness	No of Patients(n=50)	Percentage (%)
0	**34**	**68**
1	**12**	**24**
2	**4**	**8**
3	**-**	**-**

Table 5: Speech Disturbances.

SPEECH	No. of Patients (N=50)	Percentage (%)
Normal	20	40
Abnormal	30	60

Table 6:

Types		No. of Patients (N=30)	Percentage (%)
Dysphasia	Broca	7	23.3
	Wernicke	1	3.3
	Global	1	3.3
Aphasia	Broca	3	10
	Wernicke	4	13.3
	Global	4	13.3
Dysarthria		9	30
Dysphonia		1	3.3

Table 7: Cranial Nerve Involvement

Cranial Nerve	No. of patients (n=50)	Percentage (%)
7th cranial nerve	50	100
3rd cranial nerve	9	18
4th cranial nerve	1	2
6th cranial nerve	3	6

Table 8: **Pupillary changes.**

Pupils	No. of patients (n=50)	Percentage(%)
Normal	32	64
Abnormal	18	36

Table 9: Gender wise distribution of Pupillary Changes.

Pupils	Gender	No. of patients (n=50)	Percentage (%)
Normal(32)	Male	24	48
	Female	8	16
Abnormal(18)	Male	12	24
	Female	6	12

Table 10: Pupillary changes observed every 12 hours for 72 hours.

S. No.	0- 12 hrs.	Pupils	N	Percentage (%)
1		DNRL	9	18%
		CRLA	9	18%
		ERLA	32	64%
S. No.	12- 24 hrs.	Pupils	N	Percentage (%)
2		DNRL	9	18%
		CRLA	7	14%
		ERLA	34	68%
S. No.	24-36 hrs.	Pupils	N	Percentage (%)
3		DNRL	9	18%
		CRLA	2	4%
		ERLA	39	78%
S. No.	36 - 48 hrs.	Pupils	N	Percentage (%)
4		DNRL	9	18%
		CRLA	2	4%
		ERLA	39	78%
S. No.	48- 60 hrs.	Pupils	N	Percentage (%)
5		DNRL	9	18%
		CRLA	2	4%
		ERLA	39	78%
S. No.	60 - 72 hrs.	Pupils	N	Percentage (%)
6		DNRL	9	18%
		CRLA	2	4%
		ERLA	39	78%

Table 11: Ocular movements.

Ocular Movements	No. of patients (n=50)	Percentage (%)
Normal	30	60
Abnormal	20	40

Table 12-Gender Wise Distribution of Ocular Movements.

Ocular movements	Gender	No. of patients (n=50)	Percentage (%)
Normal(30)	**Male**	23	46
	Female	7	14
Abnormal(20)	**Male**	13	26
	Female	7	14

Table 13: Abnormal Ocular movements observed every 12hours for 72hours.

S. No		Ocular changes	N	Percentage (%)
1	0-12hours	Full range	32	64%
		Left 3rd Cranial Nerve palsy	5	10%
		Conjugate eye deviation	3	6%
		Right3,6 Cranial Nerve palsy	1	2%
		Nystagmus on Lateral Gaze	4	8%
		Left Horizontal Nystagmus	1	2%
		3,4,6 Cranial Nerve Palsy	1	2%
		Right 3rd Cranial Nerve Palsy	2	4%
		Right 6th Cranial Nerve Palsy	1	2%
S. No		Ocular changes	N	Percentage (%)
2	12-24hours	Full range	32	64%
		Left 3rd Cranial Nerve palsy	5	10%
		Conjugate eye deviation	3	6%
		Right3,6 Cranial Nerve palsy	1	2%
		Nystagmus on Lateral Gaze	4	8%
		Left Horizontal Nystagmus	2	4%
		3,4,6 Cranial Nerve Palsy	1	2%
		Right 3rd Cranial Nerve Palsy	1	2%
		Right 6th Cranial Nerve Palsy	2	4%
S. No		Ocular changes	N	Percentage (%)
3	24-36hours	Full range	30	60%
		Left 3rd Cranial Nerve palsy	5	10%
		Conjugate eye deviation	3	6%
		Right3,6 Cranial Nerve palsy	1	2%
		Nystagmus on Lateral Gaze	4	6%
		Left Horizontal Nystagmus	2	4%
		3,4,6 Cranial Nerve Palsy	1	2%
		Right 3rd Cranial Nerve Palsy	2	4%
		Right 6th Cranial Nerve Palsy	2	4%
S. No		Ocular changes	N	Percentage (%)
4	36-48hours	Full range	32	64%
		Left 3rd Cranial Nerve palsy	4	10%
		CED	2	6%
		Right3,6 Cranial Nerve palsy	1	2%
		Nystagmus on Lateral Gaze	4	8%
		Left Horizontal Nystagmus	2	4%
		3,4,6 Cranial Nerve Palsy	1	2%
		Right 3rd Cranial Nerve Palsy	2	4%
		Right 6th Cranial Nerve Palsy	2	4%
S. No		Ocular changes	N	Percentage (%)
5	48-60hours	Full range	32	66%
		Left 3rd Cranial Nerve palsy	5	10%
		Conjugate eye deviation	2	4%

		Right3,6 Cranial Nerve palsy	**1**	**2%**
		Nystagmus on Lateral Gaze	**3**	**6%**
		Left Horizontal Nystagmus	**2**	**4%**
		3,4,6 Cranial Nerve Palsy	**1**	**2%**
		Right 3rd Cranial Nerve Palsy	**2**	**2%**
		Right 6th Cranial Nerve Palsy	**2**	**4%**
S. No		**Ocular changes**	**N**	**Percentage (%)**
6	**60 – 72 hrs**	**Full Range**	**36**	**72%**
		Left 3rd Cranial Nerve palsy	**5**	**10%**
		Conjugate eye deviation	**1**	**2%**
		Right3,6 Cranial Nerve palsy	**1**	**2%**
		Nystagmus on Lateral Gaze	**2**	**4%**
		Left Horizontal Nystagmus	**1**	**2%**
		3,4,6 Cranial Nerve Palsy	**1**	**2%**
		Right 3rd Cranial Nerve Palsy	**2**	**4%**
		Right 6th Cranial Nerve Palsy	**1**	**2%**

Table 14: Motor Deficit.

Weakness	Side	No. of Patients (N=50)	Percentage (%)
Hemiparesis	**R**	**24**	**48**
	L	**12**	**24**
Hemiplegia	**R**	**7**	**14**
	L	**7**	**14**

Table 15-Area of Infarct.

Area of infarct	No of patients	Percentage
LMCA	27	54%
RMCA	9	18%
LPCA	5	10%
RPCA	6	12%
LACA	2	4%
RACA	1	2%

Table 16: Area wise distribution of pupillary changes and abnormal ocular movements.

	Area of Infarct	Pupillary Changes(n=18)	Abnormal Ocular Movements (n=20)
Anterior circulation	LMCA	6 (33%)	7 (35%)
	RMCA	2 (11%)	2 (10%)
	LACA	-	-
	RACA	1 (6%)	1 (5%)
Posterior circulation	LPCA	4 (22%)	5 (25%)
	RPCA	5 (28%)	5 (25%)

Statistical Analysis

All the data were evaluated using a statistical package for social science (SPSS 17.0). Chi-square test with Yate's adjustment was performed to determine associations between ocular movements and pupillary changes and other neurological findings and CT Brain reports (hemisphere involvement, type of stroke, area of involvement, age and gender). The confidential interval was considered at 95% level. When *p* value was equal to or less than 0.05, the finding was considered significant.

0-12 hours pupil and CT Brain

0- 12 hrs pupil	CT BRAIN						Total
	1	2	3	4	5	6	
1	**22**	**6**	**0**	**1**	**2**	**0**	**31**
2	**5**	**2**	**0**	**2**	**0**	**0**	**9**
3	**0**	**0**	**5**	**3**	**0**	**1**	**9**
4	**0**	**1**	**0**	**0**	**0**	**0**	**1**
Total	**27**	**9**	**5**	**6**	**2**	**1**	**50**

Chi square Test

	Value	Df	Asymp. Sig (2 sided)
Pearson Correlation – chi square	47.398	15	.000 (pValue)
No of Valid Cases	50	15	.000 (p value)

If the p value is < .005 it is statistically significant

12-24 hours pupil and CT BRAIN

12- 24 hrs pupil	CT BRAIN						Total
	1	2	3	4	5	6	
1	223	8	0	1	2	0	34
2	4	1	0	2	0	0	7
3	0	0	5	3	0	1	9
Total	27	9	5	6	2	1	50

Chi square Test

	Value	df	Asymp. Sig (2 sided)
Pearson Correlation – chi square	43.911	10	0.000
Likely hood ratio	43.549	10	(pValue)
Linear- by Linear Association	16.066	1	
No of Valid Cases	50	15	

If the p value is < .005 it is statistically significant

The above mentioned table shows the statistical significant between clinical correlation of pupil reaction of 12 – 24hrs and CT Brain findings.

24 – 36 hours pupil and CT BRAIN

24-36 hrs pupil	CT BRAIN						Total
	1	2	3	4	5	6	
1	27	9	0	1	2	0	39
2	0	0	0	2	0	0	2
3	0	0	5	3	0	1	9
Total	27	9	5	6	2	1	50

Chi square Test

	Value	df	Asymp. Sig (2 sided)
Pearson Correlation – chi square	57.265	10	
Likely hood ratio	50.985	10	0.000 (p Value)
Linear- by Linear Association	20.979	1	
No of Valid Cases	50		

If the p value is < .005 it is statistically significant

The above mentioned table shows the statistical significant between clinical correlation of pupil reaction of 24-36hrs and CT Brain findings.

36-48hrs hours pupil and CT BRAIN

36- 48 hrs pupil	CT BRAIN						Total
	1	2	3	4	5	6	
1	27	9	0	1	2	0	39
2	0	0	0	2	0	0	2
3	0	0	5	3	0	1	9
Total	27	9	5	6	2	1	50

Chi square Test

	Value	df	Asymp. Sig (2 sided)
Pearson Correlation – chi square	57.265	10	0.000 (pValue)
Likely hood ratio	50.985	10	
Linear- by Linear Association	20.979	1	
No of Valid Cases	50	15	

If the p value is < .005 it is statistically significant

The above mentioned table shows the statistical significant between clinical correlation of pupil reaction of 36 – 48 hrs and CT Brain findings.

48 - 60hrs hours pupil and CT BRAIN

36- 48 hrs pupil	CT BRAIN						Total
	1	2	3	4	5	6	
1	27	9	0	1	2	0	39
2	0	0	0	2	0	0	2
3	0	0	5	3	0	1	9
Total	27	9	5	6	2	1	50

Chi square Test

	Value	df	Asymp. Sig (2 sided)
Pearson Correlation – chi square	57.265	10	0.000 (pValue)
Likely hood ratio	50.985	10	
Linear- by Linear Association	20.979	1	
No of Valid Cases	50	15	

If the p value is < .005 it is statistically significant.

The above mentioned table shows the statistical significant between clinical correlation of pupil reaction of 36 – 48 hrs and CT Brain findings.

0-12hrs hours Ocular movements and other CN

0- 12 hrs Occular	OTHER CN			Total
	1	2	3	
1	21	11	0	32
2	0	2	0	2
3	4	1	0	5
4	0	1	0	1
5	0	0	1	1
6	4	0	0	4
7	1	0	0	1
8	1	2	0	3
9	0	1	0	1
Total	31	18	1	50

Chi square Test

	Value	df	Asymp. Sig (2 sided)
Pearson Correlation – chi square	61.865	16	0.000 (pValue)
Likely hood ratio	24.235	16	
Linear- by Linear Association	0.674	1	
No of Valid Cases	50		

If the p value is < .005 it is statistically significant

The above mentioned table shows the statistical significant between clinical correlation of 0-12 hrs Occular movements and Other Cranial nerve involvements.

0-12hrs hours Ocular movemnets and CT brain

0-12 hrs Occular	OTHER CT Brain						Total
	1	**2**	**3**	**4**	5	6	
1	21	7	0	2	2	0	32
2	0	0	0	2	0	0	2
3	0	0	4	0	0	1	5
4	0	0	0	1	0	0	1
5	0	0	1	0	0	0	1
6	4	0	0	0	0	0	4
7	1	0	0	0	0	0	1
8	1	2	0	0	0	0	3
9	0	0	0	1	0	0	1
Total	27	9	5	6	2	1	50

Chi square Test

	Value	df	Asymp. Sig (2 sided)
Pearson Correlation – chi square	90.812	40	0.000
Likely hood ratio	63.337	40	(pValue)
Linear- by Linear Association	0.323	1	
No of Valid Cases	50		

If the p value is < .005 it is statistically significant

The above mentioned table shows the statistical significant between clinical correlation of 0-12 hrs Occular movements and CT Brain.

12- 24hrs hours Occular and other CN.

12-24hrs Occular	OTHER CN			Total
	1	**2**	**3**	
1	21	11	0	32
2	0	2	0	2
3	4	1	0	5
4	0	1	0	1
5	0	0	1	1
6	4	0	0	4
7	1	0	0	1
8	1	2	0	3
9	0	1	0	1
Total	31	18	1	50

Chi square Test

	Value	df	Asymp. Sig (2 sided)
Pearson Correlation – chi square	61.865	16	
Likely hood ratio	24.235	16	0.000
Linear- by Linear Association	0.674	1	(pValue)
No of Valid Cases	50		

If the p value is < .005 it is statistically significant

The above mentioned table shows the statistical significant between clinical correlation of 12- 24 hrs Occular movements and Other Cranial nerve involvements.

24 - 36hrs Ocular movements and other CN

0- 12 hrs Occular	OTHER CN			Total
	1	**2**	**3**	
1	21	11	0	32
2	0	2	0	2
3	4	1	0	5
4	0	1	0	1
5	0	0	1	1
6	4	0	0	4
7	1	0	0	1
8	1	2	0	3
9	0	1	0	1
Total	31	18	1	50

Chi square Test

	Value	Df	Asymp. Sig (2 sided)
Pearson Correlation – chi square	64.289	16	0.000 (pValue)
Likely hood ratio	27.228	16	
Linear- by Linear Association	0.991	1	
No of Valid Cases	50		

If the p value is < .005 it is statistically significant

The above mentioned table shows the statistical significant between clinical correlation of 24 - 36hrs Occular movements and Other Cranial nerve involvements.

24 - 36 hours Ocular movements and CT brain

0-12 hrs Occular	OTHER CT Brain						Total
	1	**2**	**3**	**4**	5	6	
1	21	7	0	2	2	0	32
2	0	0	0	2	0	0	2
3	0	0	4	0	0	1	5
4	0	0	0	1	0	0	1
5	0	0	1	0	0	0	1
6	4	0	0	0	0	0	4
7	1	0	0	0	0	0	1
8	1	2	0	0	0	0	3
9	0	0	0	1	0	0	1
Total	27	9	5	6	2	1	50

Chi square Test

	Value	Df	Asymp. Sig (2 sided)
Pearson Correlation – chi square	100.179	40	0.000 (p Value)
Likely hood ratio	70.259	40	
Linear- by Linear Association	0.849	1	
No of Valid Cases	50		

If the p value is < .005 it is statistically significant.

The above mentioned table shows the statistical significant between clinical correlation of 24 -36hrs Occular movements andCT Brain.

36- 48 hrs Ocular movements and other CN

0- 12 hrs Occular	OTHER CN			Total
	1	2	3	
1	20	10	0	30
2	0	2	0	2
3	4	1	0	5
4	0	1	0	1
5	0	0	1	1
6	4	0	0	4
7	2	0	0	2
8	1	2	0	3
9	0	2	0	2
Total	31	18	1	50

Chi square Test

	Value	df	Asymp. Sig (2 sided)
Pearson Correlation – chi square	64.289	16	
Likely hood ratio	27.228	16	0.000 (p Value)
Linear- by Linear Association	0.991	1	
No of Valid Cases	50		

If the p value is < .005 it is statistically significant.

The above mentioned table shows the statistical significant between clinical correlation of - 36- 48hrs Occular movements and Other Cranial nerve involvements

36-48hrs hours pupil and CT BRAIN

36- 48 hrs pupil	CT BRAIN						Total
	1	2	3	4	5	6	
1	27	9	0	1	2	0	39
2	0	0	0	2	0	0	2
3	0	0	5	3	0	1	9
Total	27	9	5	6	2	1	50

Chi square Test

	Value	df	Asymp. Sig (2 sided)
Pearson Correlation – chi square	57.265	10	
Likely hood ratio	50.985	10	0.000 (p Value)
Linear- by Linear Association	20.979	1	
No of Valid Cases	50	15	

If the p value is < .005 it is statistically significant.

The above mentioned table shows the statistical significant between clinical correlation of pupil reaction of 36 – 48 hrs and CT Brain findings.

DISCUSSION

- **Age Incidence**

Of the 50 patients in our study, the maximum number of cases were between the age group 51-79 years (28 patients).The percentage of male patients was 72 % to that of female patients which was 28 %.

In our study almost all ages were in their fifth and sixth decade, with youngest at 31 and oldest at 79 years of age with a mean age of 55years, which partly correlates with study done by Abdu Sallam et al.16 (13.6%), Gauri et al.12 (19%), P. Chitrambalam et al.13 (20%), Ukoha Ob et al.11 and Maskey et al.

- **Sex Incidence**

Of the 50 ischemic stroke patients taken into study, 36 were males and 14 were females. Stroke is common among males (72%) compared to females (28%), which correlates with study done by Aiyar et al,14 Pinhero et al.,15 Eapen et al.5

- **Neurological Assessment**

All the 50 patients with acute stroke are assessed with National Institutes of Health Stroke Scale (NIHSS). Of the 50 patients, 16 patients were having altered level of

consciousness(32%), 18 patients were having gaze paresis(36%), all the patients who are included in the study were having facial palsy (100%), all the patients presented with motor deficits (100%), 30 patients were having speech disturbances (72%).

According to NIHSS scale, the score 0- Alert, keenly responsive; 1- Not alert, arousable by minor stimulation to obey, answer, respond; 2- Not alert, requires repeated or painful stimulation to attend; 3- Responds only with reflex motor or autonomic effects,or totally unresponsive, flaccid, areflexic. Of the 50 patients, 34 patients were alert (68%) and 16 patients were having altered level of consciousness.

- **Speech**

In our study group, 30 patients (60%) were having speech disturbances. Of the 30 patients, 9 patients were having dysphasia including 7 patients with Broca's dysphasia(23%), 1 patient with Wernicke's dysphasia (3%,) and 1 patient with Global dysphasia (3%) and 11 patients were having aphasia including 3 patients with Broca's aphasia(10%), 4 patients with Wernicke's aphasia(13%) and 4 patients with Global aphasia(13%) and 9 patients were having dysarthria (30%) and 1 patient had dysphonia (3%). The most common speech abnormality observed was dysarthria (30%) and Broca's dysphasia (23%).

- **Cranial Nerve Involvement**

Cranial nerve involvement in all the 50 cases (100%). The involvement of 3rd cranial nerve was found in 9subjects, involvement of 6th cranial nerve in 3 subjects, 7th cranial nerve is involved in almost all the cases, multiple cranial nerve involvement in 3 subjects, conjugate eye deviation towards lesion side found in 3, nystagmus in lateral gaze was observed in 6 cases, significant pupillary changes were observed in 18 cases.

This study is mostly comparable to other studies on type of ischemic stroke, hemisphere involved (left, 70%), and area involved (cortical, 65%). Conjugate eye deviation towards the affected hemisphere as well as eye movement disorders due to involvement of the third, fourth and sixth cranial nerves are common.[6] The percentage of extra ocular muscle paresis due to stroke in our study (17.5%) has been observed to be quite similar (18%) to that from the study done by Rowe et al. study.[7]

- **Pupils & Ocular Movement Abnormalities**

Pupils

Of the 18 patients with abnormal pupillary findings, there were 9 patients(18%) with dilated pupils which were not reacting to light during the first 12 hours, i,e, from the time of admission, 9 patients (18%) with round, constricted pupils, sluggishly reacting to light and 32 patients(64%) did not show any changes in the pupils.

After 12 hours, 9 patients(18%) were having dilated pupils not reacting to light and 7 patients(14%) with round, constricted pupils, sluggishly reacting to light and 34 patients(68%) were having normal sized round pupils reacting to light.

In 24-36 hours, 9 patients(18%) were having dilated pupils not reacting to light and 2 patients(4%) with round, constricted pupils, sluggishly reacting to light and 39(78%) were having normal sized round pupils reacting to light.

In 36-48 hours, 9 patients(18%) were having dilated pupils not reacting to light and 2(4%) patients with round, constricted pupils, sluggishly reacting to light and 39(78%) were having normal sized round pupils reacting to light.

In 48-60 hours, 9 patients(18%) were having dilated pupils not reacting to light and 2(4%) patients with round, constricted pupils, sluggishly reacting to light and 39(78%) were having normal sized round pupils reacting to light.

In 60-72 hours, 9 patients(18%) were having dilated pupils not reacting to light and 2(4%) patients with round, constricted pupils, sluggishly reacting to light and 39(78%) were having normal sized round pupils reacting to light.

According to Behr C Et al, the stimulation of the cerebral hemisphere, prefrontal, parietal and occipital lobe as well as the corona radiata and internal capsule produce pupillary changes. Dilatation is obtained from the above structures with or without horizontal conjugate eye movements. At times, constriction of the pupil occurs with frontal and occipital lobe stimulation. constriction of the pupil is mostly associated with disjunctive movements such as convergence or divergence.[9]

Ocular Movements

Of the 20 patients who were having abnormal ocular movements, 13 patients were male (26%) and 7 patients were female (14%).

Of the 20 patients(40%) with abnormal ocular movements, the commonly observed ocular changes in the study sample were 3rd nerve palsy, 6th nerve palsy, multiple cranial nerve palsies, conjugate eye deviation to the side of the lesion, nystagmus on left lateral gaze, horizontal nystagmus on lateral gaze to the side of lesion.

In the first 12hours (from the time of admission), 5 patients had left 3rd cranial nerve palsy, which continued to persist till 72 hours, 2 patients had right 3rd cranial nerve palsy which continued to persist till 72 hours. 2 patients had right 6th cranial nerve palsy, of which one patient developed the lesion after 24hours of hospital admission and continued to persist till 72 hours of the hospital stay. Multiple cranial nerve palsies were recorded in 2 patients involving 3,6 cranial nerves in one patient and 3,4,6 cranial nerves in the other patient.

In the first 12hours, conjugate eye deviation was observed to the side of the lesion in 3 patients, of which one patient recovered full range of ocular movements in 48-60 hours and one patient recovered full range of ocular movements in 60-72hours.

In the first 12 hours of hospital admission, horizontal nystagmus was commonly observed in 5 patients on left lateral gaze which disappeared after 24-60 hours in most of the patients and it disappeared in 2 patients in 48 hours and 60-72 hours respeectively. It was commonly seen towards the lesion side when attempted for lateral gaze.

- **Motor**

Motor deficits in all the 50 cases (100%) and Sensory deficits were less commonly observed (10%). Of the 50 acute stroke patients, neurological findings included a clinical presentation of hemiplegia (14 subjects) and hemiparesis in (36 subjects), involvement of left hemisphere of brain in 36 subjects (60%), right hemisphere in 14 cases (40%) and involvement of cortical area in 26 subjects (65%). Cortical areas included frontal, parietal, temporal, and occipital lobes; internal capsule; thalamus and basal ganglion.

- **Site of Infarct**

Among the areas of the infarct of the study group, the left hemisphere involvement (dominant) (70%) is more common than the right (non dominant) hemisphere (30%) involvment. The anterior circulation stroke was the commonest among the study group (78%). 39 patients presented with anterior circulation stroke (78%) and 11 patients presented with posterior circulation stroke (22%).

Among anterior circulation stroke, left middle cerebral artery infarct constitutes 56% (28 patients), followed by right middle cerebral artery infarct 18% (9patients), followed by left anterior cerebral artery infarcts 4%(2patients) and right anterior cerebral artery 2% (1 patient), this was also confirmed in study done by Devichand et al. and Caroli et al.18.

Correlation between Site of Infarct and Pupils and Ocular Movements

Of the 50 acute stroke patients in our study group, the pupillary changes and abnormal ocular movement in anterior and posterior circulation stroke are found to be equal, patients with LMCA territory infarct were having maximum pupillary changes(33%) and abnormal ocular movements(35%), patients with RMCA territory infarct were having 11% pupillary changes and 10% abnormal ocular movements, patients with LPCA territory infarct were having 22% pupillary changes and 25% abnormal ocular movements, patients with RPCA territory infarct were having 28% pupillary changes and 25% abnormal ocular movements, pateints with RACA territory infarct were having 6% pupillary changes and 5% abnormal ocular movements and there were no pupillary changes and abnormal ocular movements found in patients with LACA territory infarct in our study group

Evidence for pathways producing dilatation independent of the peripheral sympathetic system was presented as early as 1900 by Parsons, who obtained dilatation of pupils by cortical stimulation after bilateral cervical sympathectomy.[10] The persistant associated severe deficit(paralysis, sensory changes, field defect) suggests destruction of cortical and subcortical gray and white matter.

The left hemisphere stroke (11 out of 16 cases) has been observed almost to have associated significantly higher level of ocular movement abnormalities than the right hemisphere stroke (5 out of 16 cases) at $p = 0.001$. This finding was comparable with other reports.

In the Vallar et al. study,41 35% right brain damaged subjects and 9% left brain damaged subjects had contralateral visual changes. Similarly, Pedersen et al.[11] have reported right hemisphere lesions in 42% subjects and left hemisphere lesions in 8% subjects. Ocular defects in lesion confined to the left hemisphere usually gives rise to minor and short-lasting spatial impairments in the contralateral side, but bilateral lesions are necessary to produce persistent and severe right visual defects.[12,14] This could probably explain the incidence of abnormal ocular movements in the left hemisphere lesions more than right hemisphere lesions.[13,14]

Summary

On summarizing the observations and results of this study,

- There was male preponderance in our study (78%).
- The most commonly affected age group was 5th (36%) & 6th(24%) decades.
- There were 30% patients with speech defects, of which the more common speech abnormality was aphasia 36% and dysarthria 30%.
- The commonly involved cranial nerves were 7th(Facial) 100%, 3rd(oculomotor) 18%, 4th(trochlear) 2%, 6th(abducens) 6% cranial nerves.
- There were 36% patients with abnormal pupillary findings (with male preponderance 24% than female 12%) and the commonly observed finding was the dilatation of pupils, which predicted the poor outcome.
- There were 40% pateients abnormal ocular movements (with male preponderance 26% than female (14%) and the more common ocular movement observed was horizontal nystagmus.
- The commonest motor deficit observed was hemiparesis 72% (with left sided lesion 62% than the right 38%).
- The left hemisphere involvement (dominant hemisphere) 70% was more common than the right hemisphere (non dominant hemisphere) 30%.
- The Anterior circulation stroke (78%) was more common than the Posterior circulation stroke (22%).

- The abnormal pupillary changes and ocular movements were very common in middle cerebral artery territory (72%%) than in posterior cerebral artery territory(22%).

CONCLUSION

The incidence of stroke in our county is rising. The occurrence rises with age with peak between 5th and 6th decades. Young pts (31-40 years) were 14% of pts which is more dangerous in view of productive year lost. This study showed male predominance in stroke cases. Males were more affected than females. Most common clinical presentation was hemiparesis followed by speech involvement. The pupillary changes and ocular movemnets and were found to be significantly associated with stroke in this study. Patients with stroke were found to require an eye examination at different period of times which could outperform an early CT brain or an MRI brain.

REFERENCES

1. A. K. Khurana, "Book of ophthalmology", 4th edition, Oxford Blackwell Publications Adams and Victor's Principles of Neurology, 10th Edition API Textbook of Medicine, 9th Edition Behr C: Die Lehre von den Pupillen Bewegungen. Berlin, Springer, 6: 224-1924.
2. Daroff RB, Troost, BT: Supranuclear disorders of eye movements.*In* Neuro-Ophthalmology, edited by Glaser JS. Hagerstown, Maryland, Harper & Row, 1978; 201-218
3. David Newman Toker "Eye movement exam more accurate than MRI in Stroke Prediction" John Leigh David S. Zee: The Neurology of Eye Movements, 4th Edition. Macintosh C. Stroke re-visited: visual problems following stroke and their effect on rehabilitation. *Br Orthopt J*., 2003; 60: 10-14. of conjugate eye deviation in acute stroke. *Neurology*., 2003; 60: 135-137.
4. Parsons JH: On dilatation of the pupil from stimulation of the cortex cerebri. J Physiol, 26: 366-379, 1900/01.
5. Pedersen PM, Jorgensen HS, Nakayama H, Raaschou HO Olsen TS. Hemineglect in acute stroke. Incidence and prognostic implications. The Copenhagen stroke study. *Am J Phys Med Rehabil*, 1997; 76: 122-127.
6. Pedersen RA, Abel LA, Troost BT: Anatomy of eye movements. *In* Biomedical Foundations in Ophthalmology, edited by Jacobiec F. Hagerstown, Maryland, Harper & Row, (in press) Rowe F, VIS group UK. Prevalence of ocular motor cranial nerve palsy and associations following Stroke. *Eye (Lond)*, 2011; 25(July (7): 881-887.
7. Simon JE, Morgan SC, Pexman JHW, Buchan AM. CT assessment Stroke, 1981; 12(2).
8. Fisher CM: Some neuro-ophthalmologic observations. J Neurol Neurosurg Psychiat, 1967; 30: 383.
9. Blanke O, Landis T, Seeck M. Electrical cortical stimulation of the human cerebral apoplexy. *Folia Psychiatr Neurol Jpn*., 1952; 6: 125–137. dans certains cas d'he´miple´gie. 1868. Thesis.
10. De Renzi E, Colombo A, Faglioni P, Gibertoni M. Conjugate gaze paresis eye deviation in stroke patients. *Stroke*. 1991; 22: 200 –202. hemispheric asymmetry. *Arch Neurol*, 1982; 39: 482– 486. in stroke patients with unilateral damage. An unexpected instance of Mohr J, Rubinstein LV, Kase C. Gaze palsy in hemispheral stroke: the NINCDS stroke data bank. *Neurology*, 1984; 34(suppl 1): 199.
11. Okinaka S, Toyokura Y, Nakamura H, Kuroiwa Y, Tsubaki T. A contribution Paus T. Location and function of the human frontal eye-field: a selective review. *Neuropsychologia*, 1996; 34: 475– 483.
12. Petit L, Clark VP, Ingeholm J, Haxby JV. Dissociation of saccade-related and pursuit-related activation in human frontal eye fields as revealed by fMRI. *J Neurophysiol*, 1997; 77: 3386 –3390.
13. Prefrontal cortex evokes complex visual hallucinations. *Epilepsy Behav*, 2000; 1: 356 –361.
14. Prevost J. De la de´viation conjugue´e de yeux et de la rotation de la tete Ringman JM, Saver JL, Woolson RF, Adams HP. Hemispheric asymmetry of gaze deviation and relationship to neglect in acute stroke. *Neurology*, 2005; 65: 1661–1662.
15. Tanaka H, Arai M, Kubo J, Hirata K. Conjugate eye deviation with head version due to a cortical infarction of the frontal eye field. *Stroke*, 2002; 33: 642– 643.
16. Tijssen CC, Schulte BP, Leyten AC. Prognostic significance of conjugate to the study of pathogenesis of conjugate deviation of eyes in Daroff RB, Troost BT, Dell'Osso LF: Nystagmus and other ocular oscillation. *In* Neuro-Ophthalmology, edited by Glascr JS, Hagerstown, Maryland, Harper & Row, 1978; 219-240.
17. Ash P, Keltner J: Neuro-ophthalmic signs in pontine lesions. Medicine, 1979; 58: 304.
18. Ashrafi an H. Letters to the Editor: Familial stroke 2700 years ago. Stroke, 2010; 41(4): e187.
19. Cogan D: Paralysis of down-gaze. Arch Ophthal (Chicago), 1974; 91: 192.
20. Kopito, J. A Stroke in Time". MERGINET com, 2001; 6: 9.
21. National Institute of Neurological Disorders and Stroke (NINDS). Stroke: Hope Th rough Research. National Institutes of Health http://www.ninds.nih.gov/disorders/stroke/detail stroke.htm, 1999.
22. R. Barnhart, ed. Th e Barnhart Concise Dictionary of Etymology, 1995.
23. Reese DM. Fundamentals—Rudolf Virchow and modern medicine. West J Med., 1998 August; 169(2): 105-108.
24. Safavi-Abbasi S, Reis C, Talley MC, Th eodore N, Nakaji P, Spetzler RF, Preul MC. Rudolf Ludwig Karl Virchow: pathologist, physician, anthropologist, and politician. Implications of his

work for the understanding of cerebrovascular pathology and stroke. Neurosurg Focus, 2006 Jun 15; 20(6): E1. PMID: 16819807.
25. Schiller F. Concepts of stroke before and after Virchow. Med Hist., 1970; 14(2): 115–3.
26. Thompson JE. Th e evolution of surgery for the treatment and prevention of stroke. The Willis Lecture. Stroke, 1996; 27(8): 1427–34.
27. Tijssen CC, van Gisbergen JA, Schulte BP. Conjugate eye deviation: side, site, and size of the hemispheric lesion. *Neurology*, 1991; 41: 846–850.
28. Gay A, Newman N, Keltner J, Stroud M: Eye Movement Disorders. St. Louis, CV Mosby, M.Gold stein (Chairman) USA, HJM Barnett Canada, J.M.Orgogozo, 1974; 20.
29. Bogousslavsky. J, F. Regli, Maeder, R. Meuli et al; The etiology of posterior circulation infarcts; Neurology, 1993; 43: 1528-1533.
30. E Ratnavalli, D. Nagaraja, M. Veerendrakumar et al: stroke in the posterior circulation territory – A clinical and radiological study, JAPI, 1995; 43(12): 910.
31. France et al Stroke, Recommendations on stroke prevention, diagnosis and therapy: Report WHO task force on stroke and other Cerebrovascular disorders. Stroke, 1989; 20(10): 1407-1431.
32. Gauri LA, Kochar DK, Joshi A, Jain R, Gupta S, Saini G, et al. A study of risk factors & clinical profile of stroke at Bikaner. J API, 2000 Jan; 48(1).
33. Jack P Whisnant Chairman, Jeffery R.B, Eugene F.B, Edward S.C.et al; Special report from National Institute of Neurological Disorders & Stroke; A classification and out line of cerebrovascular disease III, Stroke, 1990; 21(4): 637-676.
34. L.R.Caplan, R.J. Wityk, L.Pazdera et al; New England Medical Center Posterior Circulation Stroke registry II Vascular Lesions, Journal of clinical Neurology, 2005; 1(1): 31-49.
35. Ma. Cristina L., Isagani Jodi G. de los Santos; posterior circulation stroke, Philippine heart center, 2001; 4. (http://www.phc.gov.ph/cgi-bin/res_ complete. cgi).
36. P. Chitrambalam, Dipti Baskar, S. Revathy. A study on stroke in young and elderly in Rajiv Gandhi government general hospital, Chennai. Int J Clin Med, 2012; 3: 184-9.
37. Patrick. K.B., Manuel Ramirez and Bruce. D.Snyder; Temporal profile of vertebrobasilar territory infarction, Stroke, 1980; 11(6): 643-648.
38. R.B. Libman, T.G.Kwiatkowski, M.D.Hansen et al; Differences between Anterior and Posterior Circulation Stroke in TOAST, Cerebrovascular Diseases, 2001; 11: 311-316.
39. Pinhero L, Damodar S, Roy AK. Risk factors in stroke: a prospective study. J Assoc Physician India, 2000 Jan; 48: 72-6.
40. Abdul-Rahman Sallam, Khalid Al-Aghbari. The clinical profile of stroke: a Yemeni experience. J Med J., 2009; 43(2): 115-21.
41. Kaur IR, Agarwal MP, Singh NR. Study of clinical profile & CT correlation in CV stroke. J Assoc Physician India, 2001; 51: 112-7.
42. Devichand, Karoli RK. A study of cerebrovascular strokes. J Indian Med Assoc, 1991 Jan; 36(12): 62-5.
43. Aiyar et al. A study of clinic-radiological correlation in cerebrovascular stroke (A study of 50 cases). Guj Med J., 1999 Mar; 52: 58-63.
44. Maskey A, Parajuli M, Kohli SC. A study of risk factors of stroke in patients admitted in manipal teaching hospital, Pokhara. Kathmandu Univ Med J (KUMJ), 2011 Oct-Dec; 9(36): 244-7.
45. R. P. Eapen, J. H. Parikh, N. T. Patel. A study of clinical profile and risk factors of cerebrovascular stroke. Guj Med J., 2009; 64(2): 47-54.
46. Shah B, Mathur P. Workshop report on stroke surveillance in India. In: Shah B, Mathur P, eds. WHO Report. New Delhi: Division of Non Communicable Diseases, Indian Council of Medical Research, 2006; 1-33.
47. Sotaniemi KA, Pyhtinen J, Myllyla VV. Correlation of clinical and computed tomography findings in stroke patients. Stroke, 1990; 21(11): 243-5.
48. Ukoha OB, Ajaegbu O, Eke CO. A review of stroke cases in a military hospital in Nigeria. AFRIMEDIC J., 2012 July-Dec; 3(2): 30-3.
49. Uma Sundar, R Mehetre, Etiopathogenesis and Predictors of In-hospital Morbidity and Mortality in Posterior Circulation Strokes – A 2 Year Registry with Concordant comparison with Anterior Circulation Strokes, JAPI, 2007; 55: 846-849.