Assessment Of Outcome Of Acute Stroke Using National Institute Of Health Stroke Scale (Nihss)

by

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LIST OF ABBREVIATIONS USED

ABC's	-	AIRWAY, BREATHING, CARDIAC STATUS.
ACA	-	ANTERIOR CEREBRAL ARTERY
AoCA	-	ANTERIOR COMMUNICATING ARTERY
ADL	-	ACTIVITIES OF DAILY LIVING
AF	-	ATRIAL FIBRILLATION
BBB	-	BLOOD BRAIN BARRIER
BP	-	BLOOD PRESSURE
CVA	-	CEREBRO VASCULAR ACCIDENT
DBP	-	DIASTOLIC PRESSURE
DM	-	DIABETES MELLITUS
DSY	-	DYSLIPEDEMIA
GCS	-	GLASGOW COMA SCALE
HDL	-	HIGH DENSITY LIPOPROTEINS
ICP	-	INTRA CRANIAL PRESSURE
IHD	-	ISCHEMIC HEART DISEASE
ICA	-	INTERNAL CAROTID ARTERY
ICH	-	INTRA CEREBRAL HEMORRHAGE
LDL	-	LOW DENSITY LIPOPROTEINS
MCA	-	MIDDLE CEREBRAL ARTERY
MMSE	-	MINI-MENTAL STATE EXAMINATION
NIHSS	-	NATIONAL INSTITUTE OF HEALTH STROKE SCALE
NINDS	-	NATIONAL INSTITUTE OF NEUROLOGICAL DISORDERS AND STROKE
OCP	-	ORAL CONTRACEPTIVE PILLS
PACS	-	PARTIAL ANTERIOR CIRCULATION STROKE
PCA	-	POSTERIOR CEREBRAL ARTERY

PoCA	-	POSTERIOR COMMUNICATING ARTERY
POCS	-	POSTERIOR CIRCULATION STROKE
rtPA	-	RECOMBINANT TISSUE PLASMINOGEN ACTIVATOR
RHD	-	RHEUMATIC HEART DISEASE
SAH	-	SUB ARACHNOID HEMORRHAGE
SHT	-	SYSTEMIC HYPERTENSION
SBP	-	SYSTOLIC BLOOD PRESSURE
TACS	-	TOTAL ANTERIOR CIRCULATION STROKE
TG	-	TRIGLYCERIDES
TIA	-	TRANSIENT ISCHEMIC ATTACK

ABSTRACT

NEED FOR THE STUDY: In view of the long-term disabilities caused by stroke the need for an accurate early prediction of future functional abilities is paramount for setting therapeutic goals, starting early rehabilitation planning, implementing home adjustments and community support tailored to patients' needs, and informing patients about their prospects and prognosis.in this study we have assessed significance of the national institute of health stroke scale (NIHSS) score on the day of admission in predicting the severity and outcome on 30^{th} day ,in acute stroke patients.

METHODS: It is a observational prospective study,study conducted on 93 patients of stroke who were admitted in Shri B M Patil Medical College hospital who were diagnosed and admitted with acute stroke on the basis of the History, Clinical examination and proved on CT/MRI scan. Patients were selected on the basis of the inclusion and exclusion criteria. NIHSS score is noted on the day of admission and then after 30days of stroke and the patient is independent at home or requires assistance is also noted and statistically analyzed. This study was conducted between December 2017 to July 2019.

RESULTS: In this study, after 1 month of stroke among 3 patients who had baseline NIHSS score 1-4, all 3 (100%) are independent at home, among 73 patients who had baseline NIHSS score 5-15, 47 (64.4%) are independent and 26 (35.6%) required assistance, among 7 patients who had score 16-20, 1 (14.3%) patient was independent at home, 6(85.7%) required assistance, and among 10 patients who had score more than 20, 7 (70%) died , 3 (30%) required assistance and none of them are home independent. With the p value <0.001 which is statistically significant.

CONCLUSION: Baseline NIHSS score helps in predicting the outcome of the patient. Lesser the baseline score better will be the outcome.

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INTRODUCTION

DEFINITION:

"Acute stroke is defined as abrupt onset of focal neurological deficit that is attributable to a focal vascular cause." [1]

IMPACT OF STROKE: The direct and indirect cost of acute stroke in united states alone was approximated to be $56.8 million in the year 2005. Every year in us more than 7,00, 000 people have stroke, one third in that are recurrent events .There was about 6.2million stroke death in the year 2015, making it the second leading cause of death worldwide.[1] Strokes are even more important because of prolonged disability they cause. The history of world has undoubtedly been altered by stroke. Many important leaders in science, medicine and politics have had their productivity cut permanently or prematurely short by stroke.[2] Among the stroke survivors around 15% and 30% become permanently disabled, while 20% of them remain in institutional care three months after the stroke. The economical and psychological costs of stroke are enormous.

Incidence of stroke has increased considerably in India and other developing countries. On an average, in comparison with high income countries stroke occurs 15 yr earlier in and causes more deaths in low and middle economic countries. The increasing incidence of stroke may be a reflection of - increased longevity, adoption of high fat diet, sedentary life style, increasing incidence of diabetes and hypertension, central obesity.

STROKE DIAGNOSIS AND OUTCOME PREDICTION–

STROKE MIMICS Diagnosis of stroke is not easy always. Diagnosis is difficult if patient presents with altered level consciousness. Many conditions can present like TIA or stroke. Seizures, infection, neoplasms, intracranial haemorrhage ,hypoglycemia and other metabolic abnormalities are some of the conditions mimicing a stroke and TIA.[3,4,5] National Institutes of Health stroke Scale (NIHSS) was found to be helpful both in diagnosis of stroke and in stratifying patients, so that outcome could be predicted and also to decide for acute intervention. Among various stroke scales, NIHSS has been studied extensively and its reliability and validity are well documented in scientific literature.[6] So NIHSS was selected for this study and used on patients diagnosed with stroke and its consistency with the diagnosis of stroke and its usefulness in assessing the outcome was studied and confirmed.

NATIONAL INSTITUTE OF HEALTH STROKE SCALE (NIHSS)

Table No:1 NATIONAL INSTITUTE OF HEALTH STROKE SCALE (NIHSS)

NATIONAL INSTITUTE OF HEALTH STROKE SCALE		
CATAGORY		**SCORE**
1a.Levelof Consciousness(LOC):	**0 = Alert;** **1=Drowsy** **2=Stuporous** **3=Coma**	
1b. LOC Questions: (Month, Age)	**0=Answers both question correctly** **1=Answers one correctly** **2=Answers both incorrect**	
1c.LOC Commands: (eyes close/open, make fist & let go)	**0=Obeys both correctly** **1=Obeys one correctly** **2=Both incorrect**	
2.Best gaze: (Eyes open- pt follows examiner's fingers or face)	**0=Normal** **1=Partial gaze palsy** **2=Forced deviation**	
3.Visual: (Introduce visual stimulus/threat to pt's visual field quadrants. Cover 1 eye and hold up fingers in all 4 quadrants.)	**0=No visual loss** **1=Partial hemianopsia** **2=complete hemianopsia** **3=Bilateral hemianopsia**	
4.Facial Palsy: (Show teeth, raise eyebrows and squeeze eyes tightly shut.)	**0 = Normal symmetrical movements.** **1 = Minor paralysis** **2 = Partial paralysis** **3 = Complete paralysis of one or both sides**	
5. Motor Arm:	**0 = No drift; limb holds 90 (or 45) degrees**	

("Elevate extremity to 90 degrees and score drift/movement. Count to 10 out loud and use fingers for visual cue".)	**for full 10 seconds.** **1 = Drift** **2 = Some effort against gravity** **3 = No effort against gravity.** **4 = No movement.** **UN = Amputation or joint fusion,** **5a. Left Arm** **5b. Right Arm**	
6. Motor Leg: ("Elevate extremity to 90 degrees and score drift/movement. Count to 10 out loud and use fingers for visual cue".)	**0 = No drift; leg holds 30-degree position for full 5 seconds.** **1 = Drift** **2 = Some effort against gravity** **3 = No effort against gravity** **4 = No movement.** **UN = Amputation or joint fusion** **6a. Left Leg** **6b. Right Leg**	
7. Limb Ataxia: (Finger to nose, heal down shin)	**0 = Absent.** **1 = Present in one limb.** **2 = Present in two limbs.** **UN = Amputation or joint fusion**	
8. Sensory: (Pin prick to face, arms, trunk, and legs- compare sharpness side to side, or no feeling at all.)	**0 = Normal; no sensory loss.** **1 = Mild-to-moderate sensory loss** **2 = Severe to total sensory loss**	

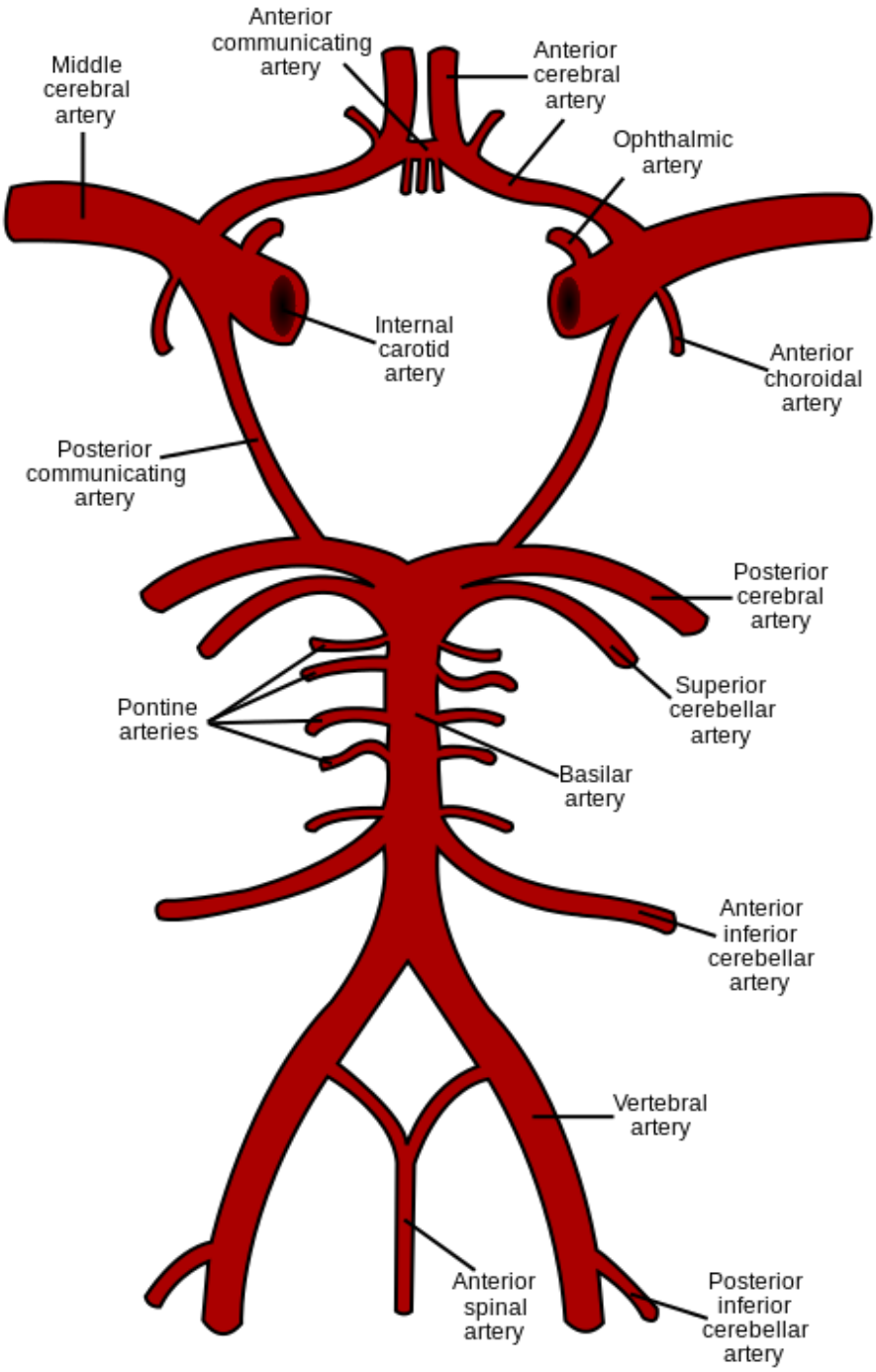
9. Best Language: ("Name items, describe picture, and read sentences. Don't forget glasses if they normally wear them".)	**0 = No aphasia; normal.** **1 = Mild-to-moderate aphasia** **2 = Severe aphasia** **3 = Mute, global aphasia; no usable speech or auditory comprehension.**	
10. Dysarthria: (Evaluate speech clarity by pt reading or repeating words on list.)	**0 = Normal.** **1 = Mild-to-moderate dysarthria** **2 = Severe dysarthria** **UN = Intubated or other physical barrier,** **explain:_____**	
11. Extinction and Inattention (formerly Neglect): ("Use information form prior testing or double simultaneous stimuli testing to identify neglect face, arms, legs and visual fields".)	**0 = No abnormality.** **1 = Visual, tactile, auditory, spatial, or personal inattention or extinction to bilateral simultaneous stimulation in one of the sensory modalities.** **2 = Profound hemi-inattention or extinction to more than one modality; does not recognize.**	
NT= NOT TESTABLE		
TOTAL SCORE		

LITERARY RIVIEW OF ACUTE STROKE

ANATOMY OF CEREBRAL CIRCULATION

Blood supply to the brain is delivered by two internal carotid and two vertebral arteries, which anastamose at the base of the brain to form the circle of Willis.

Figure No:1 CIRCLE OF WILLIS



The anterior and vertebrobasilar system supplies the posterior portions of the brain.At rest,the brain which is 2% of total body weight,receives 20% of cardiac output and consumes 20% of total inspired oxygen.

The internal carotid artery (ICA) starts at the level of thyroid cartilage at the carotid sinus at the bifurcation of common carotid artery. It gives no branches in the neck, it runs in the neck till skull base and then passes through foramen lacerum and enter carotid canal then runs through cavernous sinus and finally divides in to middle and anterior cerebral artery.

Anterior Cerebral artery (ACA) and the larger Middle cerebral artery (MCA).

First branch of ICA is Ophthalmic artery, and It supplies the eye and other structures of orbit.

Next artery from the the ICA is the Posterior communicating artery (PoCA) .It joins posterior Cerebral Artery (PCA) at its first division.

From last section of the ICA arises the anterior choroidal artery.

The ACA runs horizontally and enter the interhemispheric fissure medially, via anterior communicating artery (AoCA) it anastomoses with the opposite side, hoops along the genu of corpus callosum and give blood supply to cerebral hemisphere medially and anteriorly.

The MCA enters the sylvian fissure and divides into 2-4 branches which supply the lateral supply the lateral parts of the cerebral hemisphere.The penetrating (lenticulostriate arteries) branches arises from the proximal MCA(M1segment) that supply the major part of caudate nucleus, internal capsule posterior limb ,adjacent corona radiata, outer globus pallidus, putamen. MCA divides in to superior and

inferior division in sylvion fissure, superior supply frontal and parietal cortex superior part, inferior division supply temporal cortex and parietal lobe inferior part.

The vertebral artery ,a branch of subclavian artery travels upwards in transverse foramina of the sixth to second vertebrae. At pontomedullary junction it forms basilar artery by joining opposite vertebral artery.The vertebral artery gives rise to the anterior and posterior spinal arteries, the posterior inferior cerebellar artery and small penetrating arteries to the medulla. The posterior inferior cerebellar artery gives branches to brainstem, cedrebellar hemisphere posterior and inferior surface and inferior vermis.

The basilar artery ascends and divides into two PCA at ponto midbrain junction in the interpeduncular cistern.The anterior inferior cerebellar artery which arises from the basialar artery gives blood supply to brainstem,rostral cerebellum, cochlea and vestibule. And superior cerebellar artery gives branches to cerebellar hemisphere upper half, dentate nucleus and vermis. Close to 3rd cranial nerve The PCA hoops around the midbrain and supplies the inferior parts of temporal and occipital lobe. The proximal portion of the PCA gives many small perforating arteries to supply the midbrain,thalamus,hypothalamus and geniculate bodies.

In about 15% of the individuals,the PCA is the direct continuation of the PoCA,its main blood supply is from the ICA rather than the basilar artery.

Collateral blood supply to the brain

Normally the anterior 2/3rd of the sameside cerebral hemisphere is supplied by ICA . Little mixing of blood via the PoCA and so the posterior course is supplied by the PCA, basilar and vertebral artery. However there are various ways in which

collateral blood supply to the brain can develop distal to occlusion of major arteries in the neck and head.The actual pattern of collateral blood flow depends on where the major blood vessels are occluded and on which collateral channels are anatomically available in a particular individual and which are free from disease.

Collateral blood flow may develop via

The circle of Willis, formed by the two ACA's proximal part, connected by the ACoA and the two PCA's proximal part, which are connected by PoCA to the distal ICA's.

Other areas of collateral blood flow are

- Around the orbit
- Leptomeningeal anastomoses
- Parenchymal anastomoses

Venous drainage

Venous drainage takes place with deep and superficial cerebral veins from the centre and periphery respectively into the internal jugular vein through dural venous sinuses. The blood flow of cerebral veins is often in the same direction as in neighbouring arteries and they have thin wall with no valves.(7)

EPIDEMIOLOGY OF STROKE

Stroke is the 2nd most common cause of mortality around the globe, with 6.2million people death from stroke in the year 2015, an expansion of 830,000 since the year 2000.In the year 2014 133,000 people died out of stroke in US, and now is the 5th most common cause of mortality in US.

Stroke is also the leading cause of disability in adults. Stroke poses major socioeconomic challenge in rehabilitation of survivors. "Out of thousands of stroke survivors each year, 30% require assistance with activities of daily living, 20% require assistance with ambulation and 16% require institutional care. The human and financial cost of stroke is immense and its estimated annual economic impact in our society, both directly in health care and indirectly in lost income is approximately $41billion." [8]

Variation in the incidence of stroke among patients aged 75 to 84 years ranged from 1054 in France to 2062 in Sweden per 1,00,000 population. In the USA there has been a steady decline to 54% for the past 30 years. In Japan, incidence of cerebral infarction has declined to 34% and cerebral haemorrhage to 29% between periods 1961-66 and 1972-76 respectively. In Finland two studies beginning in 1972 and 1977 found about one third reductions in stroke incidence. It was primarily thought that this reduction was due to widespread control of hypertension. Within past 10 years, the declining annual incidence rate appears to levelled off and some epidemiologists fear that it may begin to rise. In Sweden, there was a 38% increase in stroke among women from 1975- 78 and 1983- 85.[9]

Epidemiology of stroke in India

In India, several cross sectional studies have been carried out in various parts of the country since eighties. The first community based study on stroke was done in and around Vellore during 1969-71 and Rohtak during 1971-74.

Analysis of data from major urban university hospitals suggested that nearly 2% of all hospital cases, 4.5% of medical and 20% of neurological admissions are from stroke.[10]

In early 1980's, in Asia the stroke prevalence was around 500-700 per 1,00,000 in the western countries and 900 per 1,00,000 .[11]

The annual incidence rate of stroke ranged from 105-262/1,00,000 population with the incidence falling within the range reported in the west i.e,100-300/1,00,000 population. The subtypes of strokes were infarction in 68% and haemorrhagic in 32%.The cerebral infarct to haemorrhage ratio was 2.21. Haemorrhage cases were more compared to western countries. [12,13]

The stroke is the cause for 1.2% of total deaths in the country, including all ages with gender ratio M:F=1.24.[14] The stroke death increased with age proportionately and 2.4% of all deaths were in the age group of >70years.

Diabetes mellitus, hypertension, tobacco use and low haemoglobin were the most Important risk factors for ischemic stroke.[10]

RISK FACTORS FOR STROKE

1. **Non modifiable risk factors**

    a) Age- it is the single most important risk factor for stroke. Risk increases by 2times for each decade after 55years.

    b) Gender-Males suffer from stroke 1.25 times more commonly than females, who have greater mortality than men for stroke each year.

    c) Heredity- Increased incidence of stroke is noted in families.

    d) Prior stroke or TIA- A person who has had one or more TIAs is almost 10times more likely to have a stroke than someone of same age and sex.

2. **Modifiable risk factors**

    a) Blood pressure – Hypertension is the single most important modifiable risk factor for ischemic stroke. Prevalence of stroke is 45% at 50 years, 60% at 60 years in patients having hypertension.

    The incidence is approximately three times greater in persons with elevated BP than in normal, irrespective of age and sex.

    BP causes 46% decline in stroke and treatment of isolated systolic hypertension reduces stroke risk by 36%.About 40% of strokes can be attributed to systolic blood pressure of >140mmhg.[10,15,16]

    b) Smoking – The Framingham study showed compared to non smokers smokers have three fold increase of ischemic strokes .After the study data shows that smoking is hazardous to all forms of stroke like it is to the coronary heart disease. [17,18]

    c) Diabetes mellitus- It increases the risk of stroke by 1.8-3.5 times.

Most of the ischemic strokes in them are lacunar strokes. It was the sixth most important predictive factor for stroke according to Framingham study.[19]

d) Blood lipids- Dyslipidemias increase the risk of stroke by 1-2 times. There is a higher incidence in patients with low level of high density lipoproteins (HDL) and high levels of low density lipoproteins(LDL) and triglycerides(TG).[16] The 20-30% relative risk reduction in stroke that occurs within 1-2 years after institution of statins is due to its pleotropic effect which improves the endothelial function, Plaque stabilisation, anti thrombotic properties, diminished inflammation and improved hemorrheologic environment.

e) Obesity- The Whitehall study showed that "body mass index (BMI) was predictive of stroke in both smokers and non smokers. It was estimated that having a BMI above 25kg/m2 and smoking accounts for 60% strokes in up to 65years".[7]

f) Cardiovascular diseases- According to Framingham study, "ECG changes of LVH increases the risk of ischemic stroke by tenfold; non-specific ST and T changes by four fold and congestive cardiac failure by nine fold."[17,20] Mitral valve prolapse, prosthetic valves, endocarditis, peripheral vascular disease, MI, cardiac arrhythmias are the risk factors for embolic stroke.[21]

g) Alcohol-The risk is variable. In low to moderate consumption lowers overall mortality, while heavy consumption increases the risk of haemorrhage. Embolic strokes result from cardiac arrythmias and cardiac wall motion abnormalities, hypertension, enhanced platelet

aggregation and activation of clotting cascade, which are common in alcoholics. [22]

h) Anticoagulant therapy- Anticoagulant therapy increases the risk of intracranial haemorrhage.

i) Illicit drug use- Use of cocaine, heroin, amphetamine, LSD etc has been found to be associated with increased risk for stroke.

j) Oral contraceptives- Young women consuming oral contraceptives have risk for stroke and much more in those whose oestrogen content is more than 50mcg. [23]

k) Miscellaneous- Migraine, decreased serum fibrinogen levels, polycythemia, increased homocysteine levels etc are associated with risk of ischemic stroke.

CLASSIFICATION OF STROKE

1) According to pathogenesis

    a) Ischemic strokes

        I. With cerebral infarction

            i. Cerebral thrombosis with or without atherosclerosis

            ii. Cerebral embolism

            iii. Cerebral venous thrombosis

            iv. Arteritis

            v. Coagulopathy disorders

            vi. Cerebral anoxia

            vii. Dissecting aneurysm of brachiocephalic vessels

            viii. Angiographic complications

        II. With cerebral ischemia

            i. Transient ischemic attacks

            ii. Local embolism from proximal atheromatous plaques

            iii. With cardiac arrhythmias

            iv. Arterial hypotension

            v. Vasospasm with migraine

            vi. Idiopathic types(drugs and oral contraceptives)

    b) Haemorrhagic stroke

            i. Hypertensive cerebral haemorrhage

            ii. Ruptured aneurysm

            iii. Ruptured angioma

            iv. Trauma

            v. Complications of anticoagulant therapy

c) Stroke of undetermined origin

i. Multi infarct dementia in lacunar syndrome

ii. Fibro muscular disease

iii. Winiwarter-Buerger disease

iv. Aortic arch syndrome

2) Etiological classification

i. Atherosclerotic thrombosis

ii. Transient ischemic attacks

iii. Embolism

iv. Ruptured or unruptured saccular aneurysm

v. Arteritis

vi. Cerebral thrombophlebitis

vii. Amyloid angiopathy

viii. Dissecting aortic aneurysm

3) Clinical classification

I. Arterial territories (Oxfordshire stroke subtype classification)

i. Anterior circulation syndrome

- Anterior cerebral artery syndrome (ACA)
- Middle cerebral artery syndrome (MCA)

ii. Posterior circulation syndrome

- Vertebrobasilar artery syndrome
- Posterior cerebral artery syndrome

II. Clinical manifestations

i. TIA : Focal neurological deficit with complete recovery within 24 hrs

ii. Reversible ischemic neurologiocal deficit (RIND): Neurological deficit with complete recovery within a period of one week.

iii. Evolving stroke: Gradual stepwise development of neurological deficit.

iv. Complete stroke : sudden onset persisting neurological deficit not progressing beyond 96hrs.[10]

PATHOGENESIS OF STROKE

Cerebrovascular disorders are due to either ischemia or haemorrhage within the central nervous system. The site and size of the lesion depicts the neurological deficit.

Ischemic stroke is more common, and is due to cerebral artery occlusion due to thrombosis or embolism.

Atherosclerotic or thromboembolic arterial occlusions account for around 80- 85% of all the cases of stroke. Cerebral atherothromboembolism involves predominantly middle cerebral artery, followed by the posterior cerebral artery, anterior cerebral artery and basilar artery which are involved less commonly.

Haemorrhage may be epidural, subdural, subarachnoid, intra parenchymal or intraventricular in location. Haemorrhage could result from arterial hypertension, saccular aneurysm, arteriovenous malformations, blood dyscrasias, vasculitis, sympathomimetic drugs, cerebral amyloid angiopathy, trauma and neoplasms.[23,24]

Pathophysiology of cerebral infarction

The pathogenesis of the cerebral infarction can be separated into two sequential processes

1. Vascular and hematological events that cause reduction of local cerebral blood flow.
2. Ischemia induced alteration of cellular chemistry that lead to necrosis of neurons, glia and supportive brain cells.

The molecular outcome of brain ischemia are changes in cell signalling (neurotransmitters, neuromodulators) in signal transduction, in metabolism and in gene regulation/expression

Cerebral blood flow at rest in adult is approximately 50-55ml/100gm/min. The cerebral microcirculation distributes blood to brain. Any decrease in blood supply to the microcirculation leads to cerebral ischemia. The magnitude of flow reduction is a function of collateral blood flow which depends on individual vascular anatomy as well as the site of occlusion.

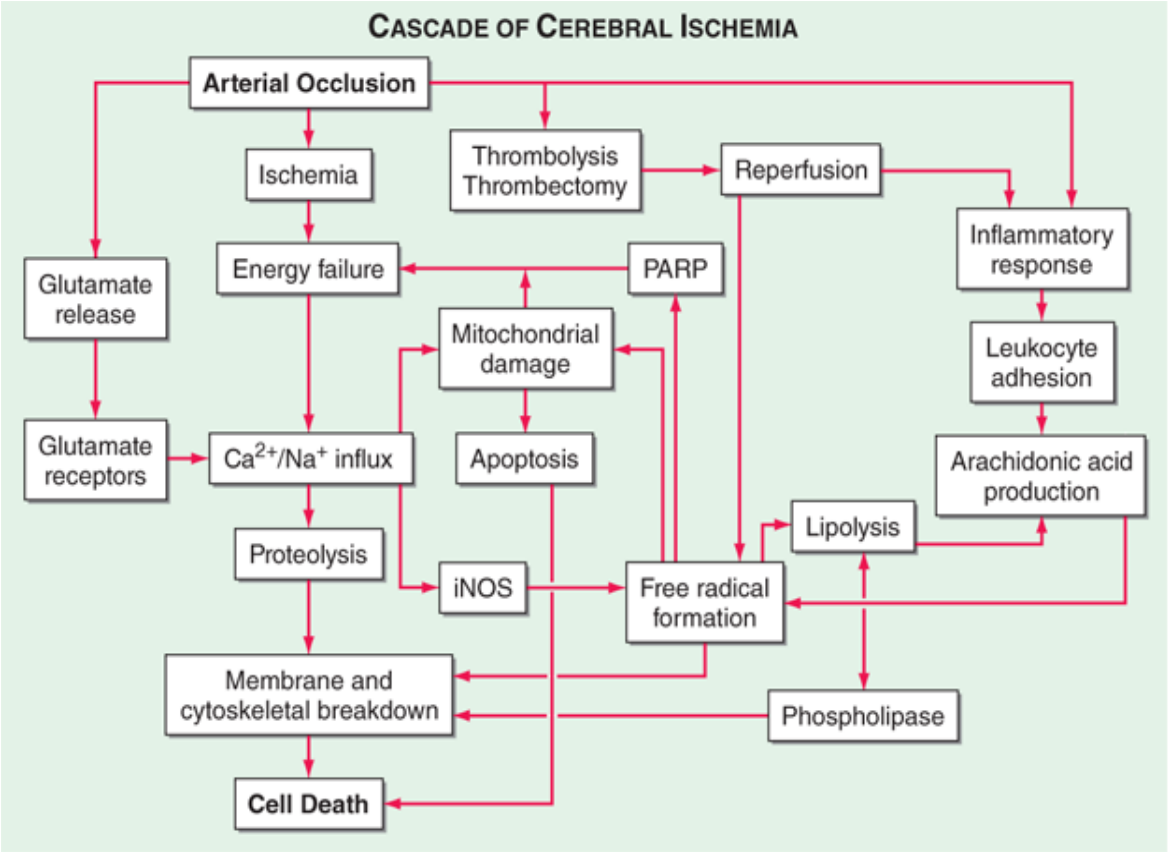
Complete interruption of cerebral blood flow suppresses electrical activity within 12-15 seconds, inhibits synaptic excitability of cortical neurons after 2-4 minutes and inhibits electrical excitability after 4-6 minutes. As the blood flow reduces to 18ml/100gm per minute , the brain reaches a threshold for electrical failure. The neurons still have the capacity for recovering though they are not functioning normally. When the blood flow reduces to 8ml/100gm per minute, membrane failure occurs. This can result to cell death. Fall in cerebral flow to zero causes death of brain tissue within 4-10minutes.

The upper threshold of blood flow i.e., 18ml/100gm per minute and lower threshold of blood flow i.e., 8ml/100gm per minute mark the limits of the ischemic penumbra. The area of misery perfusion or the ischemic penumbra is the area of the ischemic brain between these two flow thresholds in which there are some neurons that are functionally silent but structurally intact and potentially salvageable. This ischemic penumbra will eventually infarct if blood flow is not restored. Thus saving the penumbra is the goal of thrombolytic therapy and newer therapies under investigation.

Cellular death via two distinct mechanisms:

1. A necrotic pathway: cellular cystoskeletal breakdown is rapid, principally because of failure of energy of the cell.
2. An apoptotic pathway: where programmed cell death occurs.

Figure no 2: Cascade of cerebral ischemia



CARDIOEMBOLIC STROKE

Out of all ischemic strokes, Cardio embolism is responsible for 20%, mainly because of dislodgement of thrombus formed on the wall of atrium or ventricle or mitral or aortic valves. Embolic strokes occur during excertional activity and are sudden in onset, having maximum neurologic deficit at onset. The fragmentation of thrombus or quick lysis produces only transient ischemic attack (TIA).

Embolism usually occurs in PCA territory and middle cerebral artery; less commonly, it involves the anterior cerebral artery (ACA) territory.

Large emboli of 3-4mm is sufficient to block the stem of the MCA. A small embolus may block penetrating arterial branches. The size of an infarct depends on the collateral circulation within vascular territory.

The important causes of cardio embolic stroke are atrial fibrillation, ischemic cardiomyopathy, MI, prosthetic valves, RHD.

Artery to artery embolism appears to be the dominant vascular mechanism causing ischemia. It is secondary to distal embolization to intracranial arteries from thrombus on atherosclerotic plaques. Atherosclerosis of Carotid bifurcation is the most common source of artery to artery embolus. Arch of aorta, common and internal carotid arteries are other sources.

An estimated 5% of ischemic stroke are due to carotid atherosclerosis and the risk of stroke increases with the increase in carotid narrowing.

It is estimated that the risk of a recurrence is about 15% per year following stroke or TIA from intracranial atherosclerosis.

Internal carotid dissection or vertebral arteries dissection or even dissection of vessels beyond the circle of willis is a common source of embolic stroke in young (age 45years) patients. Usually it is painful and precedes the stroke by several hours or days.[24,25]

CLINICAL FEATURES OF ISCHEMIC STROKES

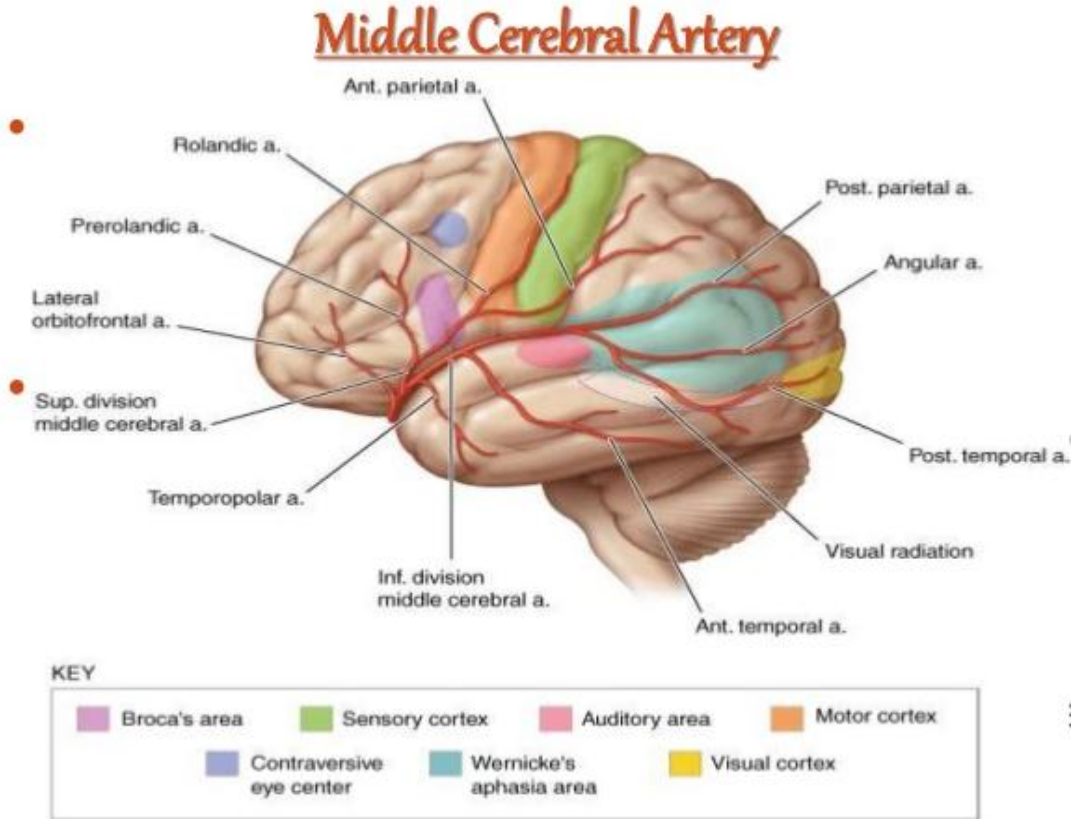
Anterior Circulation Stroke : Internal carotid artery and its branches supplies the anterior part of brain.

A. Middle Cerebral Artery territory involvement

The lateral surface of the hemisphere is supplied by the cortical branches except for

1) The lower temporal and occipital pole convolutions which is supplied by the PCA.
2) ACA supplies the frontal pole and strip of the superomedial border of the parietal and frontal lobes.

Figure No:3 Diagram showing lateral aspect of cerebral hemisphere showing distribution and branches of middle cerebral artery



Signs and symptoms of MCA territory involvement

1. Somatic motor area for face and arm and the fibres for leg descend and enter the corona radiata and corresponding somatic sensory system.
   - Opposite side face , arm and leg palsy.
   - Sensory defect in the same area (fine touch , pinprick, vibration, joint position, and all cortical sensations.)
2. Dominant hemisphere motor speech area:
   - Motor aphasia
3. Cental, suprasylvian speech area and parietooccipital cortex of the dominant hemisphere -Gerstmann syndrome comprising right left

confusion, finger agnosia, alexia, acalculia , anomia, jargon speech, sensory agraphia, Cental aphasia, word deafness.

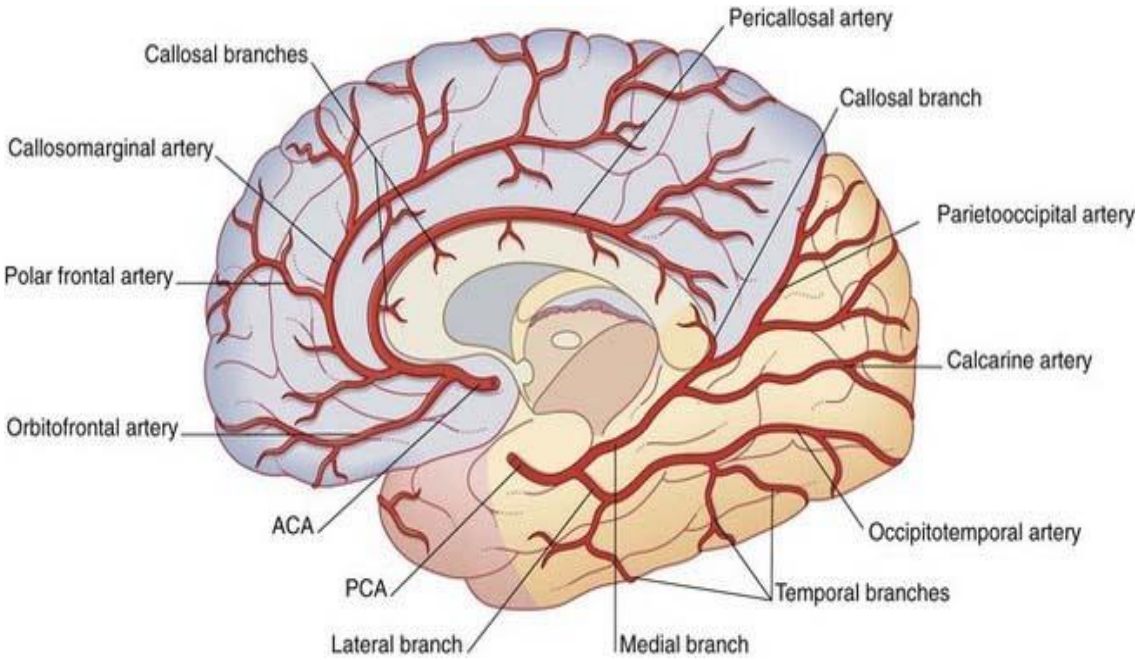
4. Central speech area (parietal operculum): conduction aphasia
5. Non dominant parietal lobe:
   - Apractognosia of the non dominant hemisphere, anosognosia, hemiasomatognosia, unilateral neglect, agnosia for the left half of external space, dressing "apraxia", constructional apraxia, distortion of visual coordinates, impaired reading, visual illusions.
6. Optic radiation deep to second temporal convolution: Homonymous hemianopia.
7. Frontal contraversive eye field or projecting fibres: conjugate gaze paralysis to the opposite side.

B. Anterior Cerebral Artery (ACA) territory involvement

The ACA it categorised into two parts:

- Precommunal (A1) segment; connecting the internal carotid artery to the anterior communicating artery.
- Postcommunal (A2) segment; distal to the anterior communicating artery.

Figure No:4 Diagram showing medial aspect of cerebral hemisphere showing distribution and branches of anterior cerebral artery



Signs and symptoms of ACA territory involvement

1. Motor area for leg: Paralysis of contralateral side foot and leg.
2. Area of arm in the cortex or descending fibres to corona radiata: paresis will be of lesser severity of opposite side.
3. Sensory area for lower limb: Cortical sensory loss over toes, foot and leg.
4. Paracentral lobule sensorimotor area: urinary incontinence
5. Posterior frontal lobe medial surface : contralateral grasp reflex, sucking reflex
6. Leg motor area: Impairment of gait and stance
7. Corpus callosum: Dyspraxia of left limbs, tactile aphasia in left limbs anterior choroidal artery territory involvement- contralateral hemiplegia, homonymous hemianopia and hemianesthesia .

Internal Carotid Artery Territory Involvement:

Occlusion of ICA often goes unnoticed because of the competent circle of willis. It shows similar symptoms as that of proximal MCA obstruction if the thrombus progress up the internal carotid artery into the MCA or embolizes it.

Repeated amaurosis fugax is seen In about 25% of internal carotid artery disease.

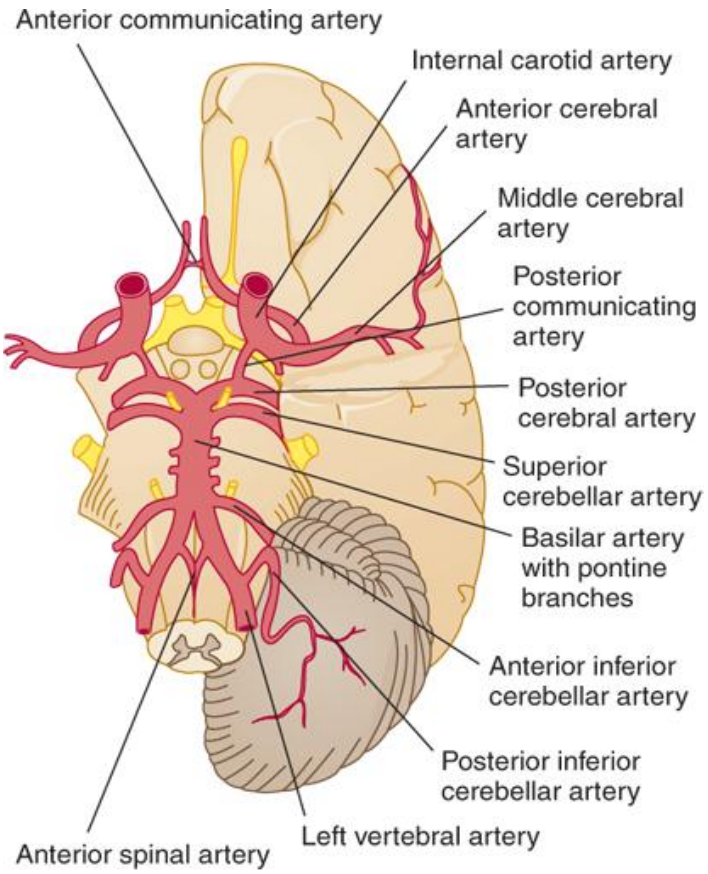
Stroke within The Posterior Circulation

The occlusion of the posterior cerebral artery involvement leads to two syndromes

P1 syndrome: Thalamic, mid brain and sub thalamic signs.

P2 syndrome: occipital lobe signs, cortical signs and temporal lobe signs.

Figure No:5 Diagram showing inferior aspect of the Brain with the distribution and branches of posterior cerebral artery



Signs and symptoms of PCA territory involvement

Peripheral territory

(1) Optic radiation or Calcarine cortex: Homonymous hemianopia (often upper quandrantic).

(2) Bilateral occipital lobe with the parietal lobe involvement: cortical blindness, Bilateral homonymous hemianopia, awareness or denial of blindness; tactile naming, achromatopia (colour blindness), failure to see to- and- fro movements, inability to perceive objects not centrally located, apraxia of ocular movements, inability to count or enumerate objects, tendency to run into things that the patient sees and tries to avoid.

(3) Bilateral Hippocampal lesion or on the dominant side only: Verbal dyslexia without agraphia, colour anomia, memory deficit.

(4) Non dominant calcarine and lingual gyrus lesions: Topographic disorientation and prosopagnosia.

(5) Dominant visual cortex: Simultanagnosia, hemi visual neglect.

(6) Calcarine cortex: peduncular hallucinosis, Unformed visual hallucinations, , teleopsia , central photophobia, metamorphopsia, palinopsia, illusory visual spread, distortion of outlines.

(7) Non dominant hemisphere: Complex hallucinations

Central Territory:

1. Posteroventral nucleus of thalamus; adjacent subthalamus body or its afferent tracts involvement; Thalamic syndrome: sensory loss (all modalities), choreoathetosis , intention tremor, hemiparesis, spasms of hand ,spontaneous pain and dysesthesias.

2. Dentothalamic tract and issuing third nerve: Thalamoperforate syndrome: (Claude's syndrome) contralateral cerebellar ataxia with ipsilateral third nerve palsy.

3. Third nerve and cerebral peduncle: Weber's syndrome: occulomotor nerve palsy and opposite side hemiplegia.

4. Cerebral peduncle: Contralateral hemiplegia

5. Supranuclear fibers to third nerve, interstitial nucleus of Cajal, nucleus of darkschewitsch, and posterior commissure : Paralysis or paresis of vertical eye movement, skew deviation, sluggish pupillary responses to light, slight miosis and ptosis (retraction nystagmus and tucking of the eyelids may be associated)

6. Dentothalamic tract: contralateral rhythmic, ataxic action tremor: rhythmic postural or holding tremor (rubral tremor)

Vertebral and Posterior Inferior cerebellar arteries

1. First part of vertebral artery (V1) ascends till 6^{th} or 5^{th} transverse vertebral foramen from the site of origin.

2. Second part of vertebral artery (V2) ascends from C6 to C2 in vertebral foramina.

3. Third part of vertebral artery (V3) travels along transverse foramen and circle and enter the dura at foramen magnum.

4. Fourth part of vertebral artery (V4) travels upwards and unites to further vertebral artery. This part gives branches that supply brainstem and cerebellum.

The lateral medulla is supplied by the proximal segment of the posterior inferior cerebellar artery and its distant branches supplies base of the cerebellum.

The first vertebral division gets affected by atherothrombotic lesions at the site of its vessel origin and leads to posterior circulation emboli, usually lesions affect V1 to V4 parts. Stenosis near to the PICA origin can damage the lateral medulla and posterior inferior part of the brain.

There is retrospective blood flow in ipsilateral vertebral artery if obstruction in subclavian artery is near the point of origin of vertebral artery. Ipsilateral arm exercise may increase demand on vertebral flow, producing TIAs, or subclavian steal of posterior circulation.

Thrombus or embolus involving the V4 segment leads to lateral medullary ischemia. leading to the lateral medullary (or Wallenberg's)syndrome comprising involvement of vestibular nucli, nucleus ambiguous affecting vagus and glosopharyngeal nerve causing causing absent gag reflex and absent gag reflex dysphagia and hoarseness of voice, spinal trigeminal tract and nucleus causing ipsilateral pain and temperature loss of the face, inferior cerebellar peduncle causing ipsilateral cerebellar symptoms and signs, lateral spinothalamic tract leading to loss of pain and temperature of contralateral body, descending sympathetic fibres causing ipsilateral Horner's syndrome.

Medial medullary syndrome rarely occurs, which causes infarction of the pyramid leading to contralateral hemiparesis sparing the face. Joint position sense of

contralateral side and ipsilateral tongue weakness occurs when the medial lemniscus and hypoglossal nerve emerging fibres are involved.

Small vessel Stroke:

The term lacunar infarction refers to infarction due to occlusion of a small artery (30-300μm) in the brain, now termed as small vessel stroke.

The arteries forming circle of Willis gives rise to 30 to 300 micrometre branches that penetrate the deep gray and white matter of the cerebrum or brainstem. These small arteries can obliterate either by atherothrombotic disease or by lipolinotic thickening. The infarct formed by Thrombosis of these vessels known as lacunes. They vary in size from 3 mm to 2 cm. Hypertension and age are the major risk factors.

The most common lacunar syndromes:

(1) Pure motor hemiparesis.

(2) Pure sensory stroke.

(3) Ataxic hemiparesis.

(4) Dysarthria and a clumsy hand or arm.

(5) Pure motor hemiparesis with motor (Broca's) aphasia.

Small-vessel infarct may manifest as TIA.

INITIAL ASSESMENT OF A STROKE PATIENT:

(A) Immediate evaluation

1. Stabilisation of airway, breathing and circulation.

2. Neurologic examination to define the neurologic deficits to classify the event into one of the clinical stroke syndromes.

3. Stroke mimics like hypoglycaemia, conversion disorder, hypertensive encephalopathy, seizures, etc. to be excluded..

4. Other conditions requiring immediate intervention to be excluded.

5. Potential causes of the stoke to be determined for early secondary prevention.

(B) History taking

1. Time of onset- The time of stroke onset is when the patient was last seen to be symptom free.

2. Circumstances during neurological symptoms like any excertional activity, sleeping.

3. History of features of other potential causes of the symptoms.

4. History of use of medications, like oral anticoagulants and antiplatelet drugs.

5. Determine risk factors for cardiac disease, atherosclerosis, drug abuse, migraine, seizures or pregnancy.

6. Determine eligibility for therapeutic intervention like revascularisation.

(C) Systemic examination

1. Assessment of airway, breathing and circulation including pulse oximetry, blood glucose and body temperature.

2. Head and neck examination to see for signs of trauma, carotid disease (bruits), seizure activity (contusions, tongue bite) , congestive heart failure (distension of jugular vein)

3. Cardiavascular system examination to identify valvular diseases, irregular rhythm, associated ischemic heart disease, etc.

4. Examination of skin and extremities to detect coagulopathies, platelet disorders, etc.

5. "The initial NIHSS score provides important prognostic information. Approximately 60% to 70% of patients with an acute ischemic stroke and a baseline NIHSS score <10 will have a favourable outcome after 1 year as compared with only 4% to 16% of those with a score >20. In the NINDS trial of rtPA, those with a score of 20 or greater on the NIHSS had a 17% chance of intracranial haemorrhage, whereas the risk of bleeding was only 3% among those with a score <10." [(26)]

(D) Investigations

1. Basic work-up to be done in all patients of acute ischemic stroke:

-Complete blood count

-Prothrombin time, bleeding and clotting profile, partial thromboplastin time.

-Plasma glucose level.

-Blood ureanitrogen and serum creatinine.

-Lipid profile.

-Urine analysis.

-Chest X-Ray

-Electrocardiogram.

2.Non enhanced cranial CT (computed tomography)

-Primary modality is the brain imaging study for evaluation of stroke.[27,28]

(a) Hyperacute infarcts (<12 hours)

Early CT signs of ischemic stroke in MCA territory: Sulcal effacement, effacement of the sylvian fissure, obscuration of the lentiform nucleus, loss of grey-white matter differentiation and Horizontal part of MCA may be hyperdense (dense MCA sign) before infarction becomes visible.

(b) Subacute infarcts

Most large vessel infarcts are seen as wedge-shaped areas of decreased attenuation involving both grey and white matter in a typical vascular distribution seen after first 24-48hrs on non enhanced CT.

(c) Chronic infarcts

Focal encephalomalacic areas appear on CT scans. Iipsilateral ventricle enlarges and adjacent sulci become prominent.

"Scan negative infarcts usually occur with lower brainstem infarcts,lacunar infarcts, early scans(60% cases within 12 hours of ictus may not pick up the lesion), scans done after 2-3 week may not show or may underestimate the size of the infarct." [29]

Figure No:6 CT showing Hyper dense MCA sign- high attenuation within the expected location of the right MCA(arrow),consistent with an acute thrombus

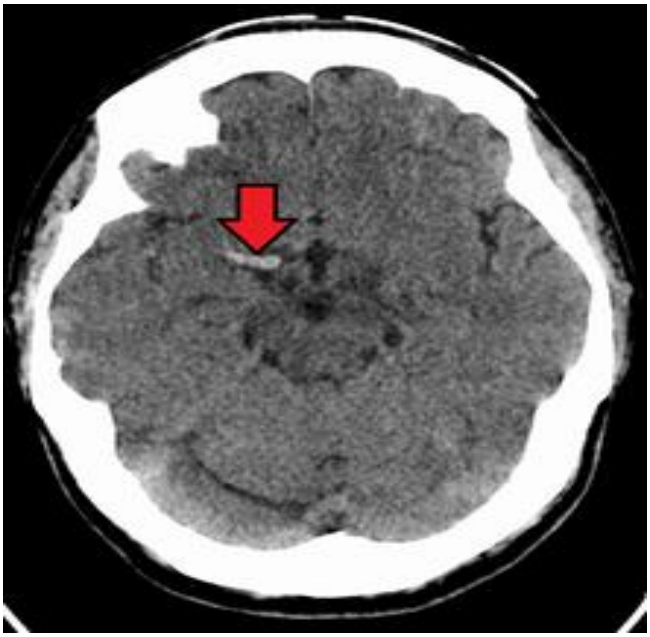


Figure No:7 CT showing Left Basal Ganglia Infraction

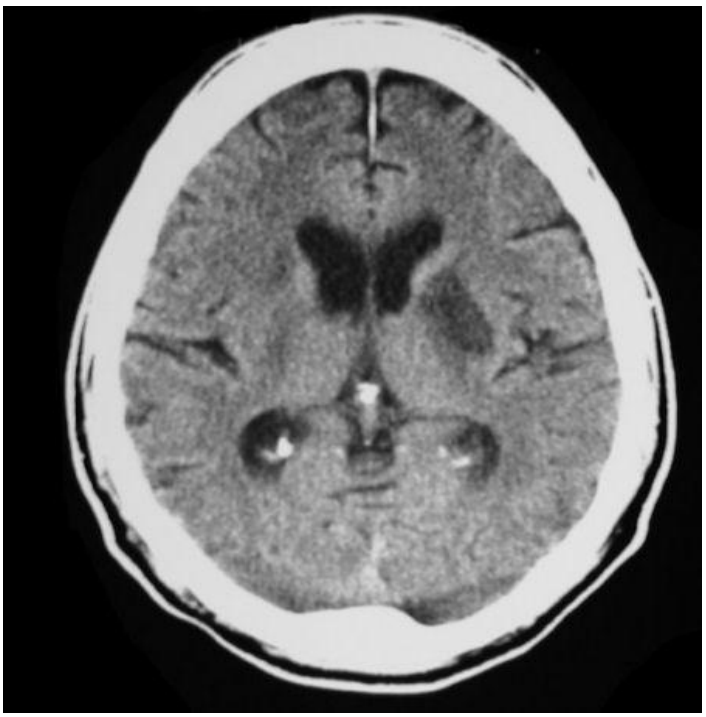
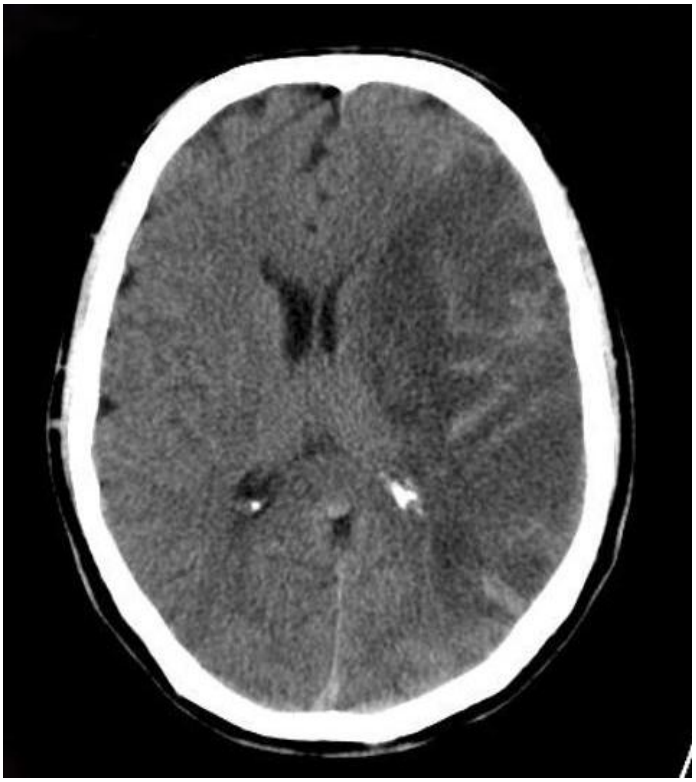


Figure No:8 CT showing Left MCA Territory Infarction



3. Multimodal CT

i. Whole brain perfusion CT- gives a map of brain blood volume and areas of hypo attenuation depicting the ischemic core.

ii. Dynamic perfusion CT- gives accurate amount of blood flow to brain.

iii. Helical CT angiography- rapidly and non invasively assesses the vasculature both intracranially and extracranially and provides information regarding vessel occlusion or stenosis.

4. Magnetic Resonance Imaging (MRI)

-Superior to CT for detecting cerebral ischemia.

- It reveals flow voids and hemosiderin and also alterations resulting from ischemic necrosis and gliosis.

5. Multimodal MRI

i. Diffusion weighed MRI (DWI) – detects ischemic areas as early as minutes of stroke.

ii. Perfusion weighed MRI (PWI) – gives relative measurement of brain hemodynamics.[30]

6. MRI angiography

Maps the blood flow and vascular lesions.useful mainly in identifying acute proximal large-vessel occlusions.

7. Other brain imaging methods

i. Oxygen-15 positron-emission tomography (PET) helps identify the first manifest of a penumbra in stroke patients.

ii. Single photon-emission computed tomography (SPECT) identifies "thresholds for reversible ischemia and could be helpful in predicting outcomes or monitoring responses to treatment."[31]

iii. Duplex Doppler Ultrasonography

For detection of >50% diameter stenosis. Sensitivity is 87-96% and specificity is 81-96%. Can detect stenosis of large vessels especially carotids and atheromatous plaques.

iv. Transcranial Doppler sonography evaluates blood flow velocity and patency of the main intracranial arteries and in identification of high intensity transient micro embolic signals. [32]

v. Digital Subtraction Angiography (DSA) –helps in location of atherosclerotic lesions and collateral circulation.

Treatment of Acute Ischemic Stroke:

(A) General supportive care

a. Maintaining adequate tissue oxygenation is important to prevent hypoxia and potential worsening of the neurological injury. Monitoring with pulse oximetry should be done to the Patients with acute stroke.

b. Fever- Increased body temperature in acute stroke is associated with poor neurological outcome, possibly due to increased neurotransmitters release, increased metabolic demands and increased free radical production. [33] Antipyretic medications and cooling devices can be used to treat fever.

c. Arterial hypertension: Stressfull outcomes of stroke are raised blood pressure , completely filled bladder, pain, response to decrease O2 levels or raised CSF pressure.In most circumstances, the blood pressure should generally not be lowered, except in acute renal failure, acute pulmonary oedema, hypertensive encephalopathy, acute myocardial infarction or aortic dissection . Antihypertensive agents should be

with held unless the unless the systolic blood pressure is >220 mmHg or diastolic blood pressure is >120 mmHg .[34]

d. Arterial Hypotension : Treat with normal saline and management of irregular heartbeats. If the above treatment is unsuccessful dopamine can be given. Correction of cardiac output and hypovolemia are essential factors within an hour of stroke.

(B) Measures to Restore or Improve Perfusion

(i) Thrombolytic therapy

"The NINDS study used IV rtPA (0.9 mg/kg to a 90-mg max; 10% as a bolus, then the remainder over 60 min) within 3 hr of onset resulted in good functional outcome though incidence of haemorrhage increased after thrombolysis. Those with NIHSS score <20) had the greatest possibility for a favourable response to treatment. In ECASS-II, intravenous rtPA was not more effective than placebo in improving neurological outcomes at 3 months after stroke." [35]

In the multicentre acute stroke trial Europe study group (MAST-E), streptokinase in the dose of 1.5 million units over one hour was associated with haemorrhagic transformation of ischemic infarct and hence not recommended.[36]

Intra-arterial thrombolysis can be used in selected patients with severe stroke of time <6 hours due to occlusion of large vessel of the middle cerebral artery. It is not FDA approved.

(ii) Anticoagulants

According to the Joint Guideline Statement from the AHA and AAN, urgent routine anticoagulation with the goal of improving neurological outcomes or preventing early

recurrent stroke is not recommended as it is associated with increased risk of bleeding complications. [37]

(iii) Antiplatelet Agents

a) Aspirin

The International Stroke Trial (IST) demonstrated a significant lowering in ischemic events recurrence by using aspirin within first 2 weeks, but acute mortality was not reduced. At 6 months, patients assigned aspirin had a significantly lower incidence of dependency and death. [38]

The Chinese Acute Stroke Trial (CAST) showed that mortality was significantly reduced with aspirin, but the rates of long term complete recovery or death and disability were not significantly improved.[39]

A combined result suggested that aspirin was effective in reducing recurrence of ischemic stroke, death, or dependency.

b) Ticlopidine

In the ticlopidine aspirin stroke study, the risk of non-fatal stroke or death from any cause at 3 years was lower in ticlopidine group as compared to aspirin group (17% vs 19%). The 3 year risk of fatal or non-fatal risk was also lower (10% vs 13%). Thus it was concluded that ticlopidine was more effective than aspirin. The Canadian American ticlopidine study concluded that an exclusive benefit cannot be claimed for ticlopidine over aspirin in treating patients with stroke.[40]

c) Clopidogrel

It is a theinopyridine derivative which is a potent inhibitor of platelet aggregation caused by ADP. In a trial recent stroke/TIA patients were randomized to receive clopidogrel 75 mg/day with low dose aspirin 75 mg/day, showed no statistically significant difference in outcome between the two treatment groups. Clopidogrel can be given to patients allergic to aspirin. [41]

(C) Neuroprotective agents

Hypothermia is probably the most powerful neuroprotectant. Six variant drugs have been tried like Calcium channel antagonists (nicardipine, nimodipine), NMDA receptor agonist (selfolate, eliprodil), ICAM-1 antibodies (Enlimomab), GABAergic antagonists (diazepam, Clomethiozole), glutamate antagonists (leleluzole), free radical scavengers (tirilazed, dihydrolipoate), lipid peroxidation inhibitors, required before routine use. [42]

(3) Surgical Interventions

The MERCI (Mechanical Embolus Removal in Cerebral Ischemia) single-arm trial investigated endovascular thrombectomy to restore patency of occluded intracranial vessels within 8 hours of ischemic stroke symptoms. Recanalization of the target vessel occurred in 48% of treated patients and in 60% following use of adjuvant endovascular methods, and successful recanalization at 90 days correlated well with favourable outcome. The North American Symptomatic Carotid Surgery Trial (ECST) evaluated patients with symptomatic stenosis of carotid arteries found a substantial benefits in patients with a stenosis of >70%.[40]

(4) Treatment Of Acute Neurological Complications

(a) Brain oedema and increased intracranial pressure- The goals of management to reduce brain oedema are:

(1) Decrease the pressure in brain tissue and cerebrospinal fluid.

(2) maintainance of adequate cerebral perfusion to prevent worsening of cerebral ischemia.

(3) Avoid cerebral damage from herniation.

Osmotherapy and hyperventilation are helpful for patients who have raised intracranial pressure features. Corticosteroids are not recommended Surgical decompression and evacuation is recommended in large cerebellar infarctions that are leading to hydrocephalus and brain stem compression . It is life saving procedure, but survivors have neurological impairments. [43]

(b) Seizures

Seizures are with or without generalisation and most commonly occur within first 24hrs of stroke. In about 20% to 80% of patients recurrent seizures develop.Till date there is no data regarding administration of anticonvulsants prophylactic for stroke.

(c) Haemorrhagic Transformation

Antithrombotics agents, especially anticoagulants and thrombolytic agents, increases the likelihood of serious haemorrhagic transformation. The early use of aspirin also is associated with a small increase in the risk of clinically detectable haemorrhage. [38]

Management of patients with haemorrhage infarction depends on the amount of bleeding and its symptoms.

Motor recovery from stroke

Most recovery of the stroke happens in the first 2-3 months. greater than 90% of recovery has occurred at 2-3 years. 75-85% of patients are ambulatory at one year post stroke, 48-58% regain independence in performance of activities of daily living and 10-29% require nursing home care.

Recovery of leg movements is at greater extent compared to arm.

Conditions contributing for the lack of good outcome are –Raised blood glucose levels, cardiac manifestations ,ECG irregularity , past history of stroke , functional dependence, lack of sensory, visual and cognitive activities.

Language does not improve evenly across all components. Recovery from aphasia and hemiparesis appears to occur independently.

Neurophysiologic mechanism for recovery from stroke

Reduce in the amount of edema and repair of ischemic shade from the dissemination . The above procedures are analysed and reported by Brodal that these procedures will not help for rehabilitation after 4-6 weeks of stroke .

Factors contributing to brain reorganization after stroke are increased glutamatergic activity, removal of inhibition, loss of perilesional GABA-ergic inhibition, improved synaptic transmission and changes in neuronal -membrane excitability.

Certain clinical observations shows the bilateral representaion of the brain. Cortical reorganization through formation of new synapsis and freeing from inhibition may lead to stroke recovery.

Cortical reorganization through formation of new synapsis and freeing from inhibition may lead to stroke recovery.

By activation of inactive synapses spontaneous restore of functions takes place by development of denervation hypersensitivity, reorganisation can be axonal or dendritic sprouting in later phase.

Inappropriate progress of new synapses and neuronal development will lead to spasticity , seizures. Early physiotherapy is useful in dendritic progress.

The reappearance of inhibitation after stroke may play an vital role in recovery. Loss of fine movements and increased reflexes shows the loss of inhibition. The reappearence of inhibitory mechanism may be responsible for the regaining of fine coordinated movements.

STROKE ASSESSMENT

NEUROLOGICAL SCALES AND INDEXES USED IN STROKE[43,44,45]

1) Neurological Impairment Scales

In a study "Comparison of neurological scales and scoring systems of acute stroke prognosis". Compared the use of NIHSS' Canadian neurological scale' Prognostic score and middle cerebral artery. Best results were produced by NIHSS at 3 months . NIHSS is consistent 'easy and fast to carryout and corresponds with ischemic size and 3 months outcome after the episode of stroke.

2)Cognitive Scale For the stroke patients MMSE is extensively used for the screening of cognitive dysfunction.[46] The statistical study that evaluates memory ,language , orientation ,attention ,and creating activities. MMSE scores remain accordant with educational level . Degree of dementia is calculated by neurobehavioral cognitive

status examination . followed by acute ischemic episode 15%-25% of the stroke population progress evident cognitive impairment.

3)Language Scales

Functional assessment of communication skills for adults ,this tool measures adequacy appropriateness and promptness of verbal responses. Others language evaluation tools are available including the Baston diagnostic apasia examination. American speech language hearing association documents language disabilities ,language dysfunction is seen in 30% of stroke population who survive. Independent life of person is interrupted by speech and language disorders.

4)Depression scale

For the screening of depression in stroke patients two testing scales are introduced

- CESD scale
- Geriatric depression scale

Cause of depression is mostly due to biological effect of cerebral ischemia , corresponding to the lesions of basal ganglia, left frontal cortex or major dropping corresponding to stroke. Depression and Dementia have indistinguishable symptoms hence it is tough to differentiate.

- Symptoms-Unperceptive , disorientation, loss of memory
- Incidence- 11%-68% is noted depression value after stroke, and it is very common but unfortunately least observed and treated , a third of which is classified as major depression.

5)Basic Activities of Daily Living Scales

The degree of disability is assessed by Barthel Index guidelines.[47] Assesssment is done on following points to live independently-

- Degree of disability after the stroke episode
- Self care tasks
- Capability

BADL is commonly used measure. There are certain limitations ,since it does not include

- Physical function loss at higher levels
- Tasks necessary to survive independently in home and community

Other most commonly used disability measure is Functional Independence Measure.

6)Instrumental Activities of Daily Living Scales (IADL)

"The Philadelphia Geriatric Center (PGC) Instrumental Activities of Daily Living Scale"

NIHSS –LITERARY REVIEW

THOMAS BROTT et al first designed the original scale in 1989 as 15-item neurologic examination stroke scale for use in acute stroke Therapy trials for measurement of acute cerebral infarction. It consists of 15 test items. On studying this scale it was found that the most interrater reliable item (pupillary response) had low validity. While less reliable items such as upper or lower extremity motor function were more valid. Therefore, the scale was altered by National Institutes of Health (NIH) and is called as NATIONAL INSTITUTES OF HEALTH STROKE

SCALE (NIHSS). [48] The NIH Stroke Scale is an 11-item (expandable to 15 items) with 13 specific tests being performed. This graded neurologic examination stroke scale was used to evaluate the effect of acute cerebral infarction like neurologic outcome and degree of recovery for stoke patients. The person who is trained will observe and rates the ability of the patient to answer to the questions and perform activities. Each item are scored as 0 to 5 grades taking 0 as normal, and there is an allowance for untestable (UT) items. The examination needs less than 10 min to perform. The maximum NIHSS score is 42. Depending on the score severity of stroke is graded as

score	**Severity of stroke**
1-4	Minor stroke
5-15	Moderate stroke
16-20	Moderate to severe stroke
21-42	Severe stroke

Important areas evaluated by NIHSS score are:

- Cranial nerve (visual)
- Motor
- Level of consciousness
- Language/neglect

BY evaluating the grade of particular area regarding the disorder (mild/moderate/severe) and a score of two, four, and eight based on the severity it is possible to calculate NIHSS score.

The features, which were found to predict functional dependence or death, included; older age, complete limb paralysis, depression of conscious level and the combination of hemiplegia and hemianopia with higher cerebral dysfunction. Hemiparesis uncomplicated by hemianopia or higher cerebral dysfunction predicted a return to functional independence. Thus, NIH Stroke Scale is a critical component of acute stroke assessment.

TRAINING [49]

Minimal amounts of training are required to reliably administer NIHSS,even by non-neurologists, which take less than 10 minutes to complete. A trained person will be able to Identify stroke patients and to stratify according to severity of stroke and to take necessary measures.

NIHSS IN ACUTE STROKE DIAGNOSIS [50,51]

NIHSS can also be used to diagnose stroke apart from assessing stroke outcome and disability. Three items identified 100% of patients with stroke they were; facial palsy, motor arm, and dysarthria. An Abbreviated NIH Stroke Scale based on these items had a sensitivity of 100% and a specificity of 92%.Based on a prospective observational cohort study, the presence of any of three physical examination finding(facial palsy,limb weakness, language) selected from the NIHSS is found to be useful. Analysed by statistical techniques, diagnosed patients with stroke with 100% sensitivity and 88% specificity.

NIHSS AND OTHER STROKE SCALES – A COMPARISON [51,52,53,54,55]

In a study, three scales were compared to see which of them is best predicted outcome. The stroke scales compared were the National Institutes of Health Stroke Scale (NIHSS), the Canadian Neurological Scale, and the Middle Cerebral Artery Neurological Score. Outcome at 2, 3, 6, and 12 months was taken as good (alive at home) or poor (alive in care or dead). Of the 408 patients studied, 373 had confirmed acute stroke and completed follow-up. The three-stroke rating

Scales each predicted 3-month outcome with an accuracy of 0.79 or greater. The NIHSS provided the most prognostic information. Implementation of NIHSS added significantly to the predictive value of all other scores. No score was useful to the NIHSS. A cutoff point of 13 on the NIHSS best predicted 3-month outcome.

THE NIHSS ADVANTAGE

The effect of stroke analysis showed that elevated NIHSS score was a strong and unrestrained predictor of discharge to rehabilitation or nursing facilities, doubling for each 5 point increase. The interrater reliability for NIHSS is also good. [57]

RETROSPECTIVE ANALYSIS USING NIHSS [58,59,45]

The NIH Stroke Scale (NIHSS) a stroke impairment scale originally designed for prospective scoring, which is used to evaluate retrospective severity of initial stroke. It is also helpful to know the prognosis. Its outcome studies of retrospective application will allow direct comparisons with data from prospective trials. The NIHSS was notified to be accurate and valid when used retrospectively in study of patients applied in clinical trials who were prospectively evaluated. A retrospective application of NIHSS on the basis of data taken from patients' medical records in an

academic hospital setting also found to be accurate and valid studies have been shown that the NIHSS (and other scoring systems) can be calculated retrospectively if there is appropriate documentation of the neurological exam. Retrospective NIHSS scoring with the algorithm is definitive and unbiased even when physical examination segment is missing from the written medical records.

NIHSS IN ACUTE STROKE MANAGEMENT [60,51,55,48]

Five phase III trials of intravenous rtPA have been reported. In 1996 this treatment was accepted by FDA relaying on the outcome of Neurological Disorders and Stroke (NINDS) rt-PA Stroke Study. Amongst which 624 patients with ischemic stroke were treated with rtPA 0.9 mg/kg IV maximum 90mg or with placebo, within 120 minutes of onset of the symptoms and approximately one half were treated within 90 minutes. The study was concluded in two sections

Section 1- In NIH stroke scale, improvement of 4 points or more.

Section 2- complete neurological recovery at 24 hours as indicated.

Patients age and degree of disorder also contributed to the outcome. Patients with NIHSS<14 and whose age was <75 years showed better response to the treatment. There was evident improvement among sever stroke patients (NIHSS score >20) also with regular treatment. Overall improvement was less in severely ill patients. With or without treatment of rtPA, prognosis was poor for major stroke patients (NIHSS>22).

The current guidelines is to thrombolyse patients suspected of stroke with NIHSS score not less than 4 and not more than 20. The NIHSS score can predict discharge disposition when thrombolysis is used.

The presence and site of vessel occlusion and NIHSS score are associated,

- NIHSS score≥10, vessel occlusion is likely to be seen on angiography.
- Vessel occlusion location is mostly in the central if NIHSS score ≥12.
- On angiography vessel occlusions is seen 97% of carotid and 96% of vertebasilar stoke in patients with NIHSS score ≥10.
- PPV of NIHSS score for central occlusion was 91% in score ≥12
- In variable analysis predictors of central occlusions were based on NIHSS elements such as :
    - Level of consciousness questions
    - Gaze
    - Motor leg
    - Neglect

PROGNOSIS OF STROKE [52,61,55]

The initial score of NIHSS gives important prognostic data. Neurological evaluation is used to assess the degree of stroke and it is a good prognostic measure. Patient with stroke whose NIHSS score is more than 20 have poor beneficial outcome compared to patients with NIHSS score less than 10, about 60% to 70% of stroke patients with NIHSS score less than 10 will have better result after an year.

NIHSS score gives numerical brief of neurological shortfall that makes observation easier for the changes in degree of disablement. It helps in prognostic outcome and use of appropriate treatment. There was difference of 24% of 7 days outcome for 24hrs of stroke onset. There is less than 20% chance of recovery with patients score of NIHSS is >15. On bases of NIHSS scores gathered within 24hrs of stroke will predict the outcome for 3 months. The NIHSS is made by number of few definitive scoring systems which are extensively used.

AIMS AND OBJECTIVES

To study the clinical profile, note the baseline NIHSS score and to find out the significance of the national institute of health stroke scale (NIHSS) score on the day of admission in predicting the severity and outcome on 30^{th} day ,in acute stroke patients

MATERIALS AND METHODS

Source of data :

The information for the study will be collected from Patients with Acute Stroke admitted to BLDEU'S Shri B. M. Patil medical college and hospital and Research centre, Vijayapur between December 2017 to June 2019.

METHOD

Observational prospective study using National institute of health stroke scale to diagnose and assess outcome of acute stroke using it. NIHSS applied on patients diagnosed with stroke, two scores were obtained for each patient, one on day of admission another after 30 days . NIHSS score at the day of admission and after 30 days of admission were noted and statistically analyzed.

TYPE OF STUDY - observational prospective study

SAMPLE SIZE

- With 95% confidence level and margin of error of ±7.5%, a sample size of 93 subjects will allow the study to determine the predictive value of NIHSS in diagnosis and outcome of stroke with finite population correction.
- By using the formula:
- $n = \frac{z^2 p(1-p)}{d^2}$

where

- Z= z statistic at 5% level of significance

- d is margin of error
- p is anticipated prevalence rate

STATISTICAL ANALYSIS

All characteristics will be summarized descriptively. For continuous variables, the summary statistics of N, mean, standard deviation (SD) will be used. For categorical data, the number and percentage will be used in the data summaries and data will be analyzed by Chi square test for association, comparison of means using t test, ANOVA and diagrammatic presentation.

INCLUSION CRITERIA:

- All male and female cases of acute stroke.
- Patients of age more than 18yrs.

EXCLUSION CRITERIA:

- Patients of age less than 18yrs.
- Transient ischemic attacks.
- Subdural/Epidural haematomas.

STUDY DESIGN

1. Patients diagnosed to have stroke by CT/DW MRI , NIHSS scoring is done on the day of admission.
2. Based on the NIHSS score severity is assessed at the time of admission, 1-4 indicates minor stroke, 5-15 indicates moderate stroke, 16-20 indicates moderate to severe stroke, 21-42 indicates severe stroke.

3. Estimation of Complete hemogram ,Urine routine, Renal function test, ECG, Chest X-ray, rbs, HbA1C and 2D Echo,CT/MRI scan done at the time of admission.
4. Patients are followed up after one month, NIHSS score after 30 days of stroke is noted.
5. After 30 days of stroke the patient is independent at home or requires assistance is noted.

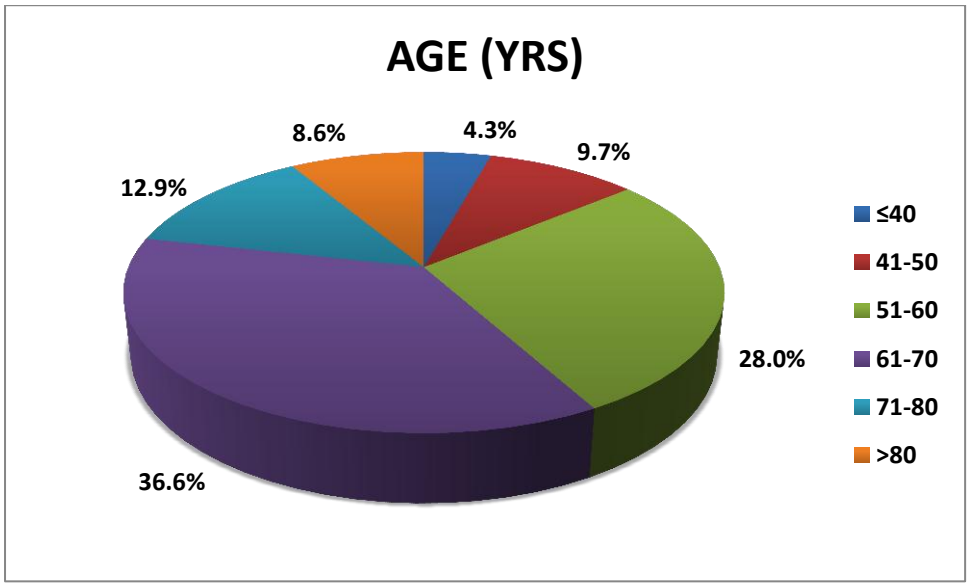
RESULTS AND OBSERVATION

Table No 2: DISTRIBUTION OF CASES ACCORDING TO AGE

AGE (YRS)	N	%
≤40	4	4.3
41-50	9	9.7
51-60	26	28
61-70	34	36.6
71-80	12	12.9
>80	8	8.6
Total	93	100

	Min	Max	Mean	SD
AGE (YRS)	26	90	63.3	11.8

Figure No 9: DISTRIBUTION OF CASES ACCORDING TO AGE

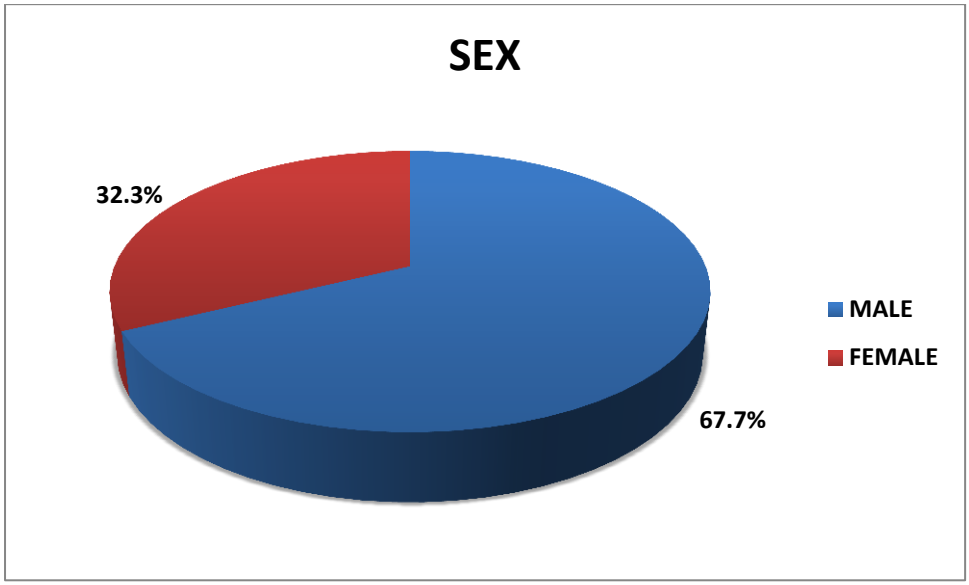


Age group of the patient ranged from 26yrs to 90 years, with mean age group 63.3±11.8, maximum number of patients were in the age group of 60-70 years

Table No 3: DISTRIBUTION OF CASES ACCORDING TO SEX

SEX	N	%
MALE	63	67.7
FEMALE	30	32.3
Total	93	100

Figure No 10: DISTRIBUTION OF CASES ACCORDING TO SEX

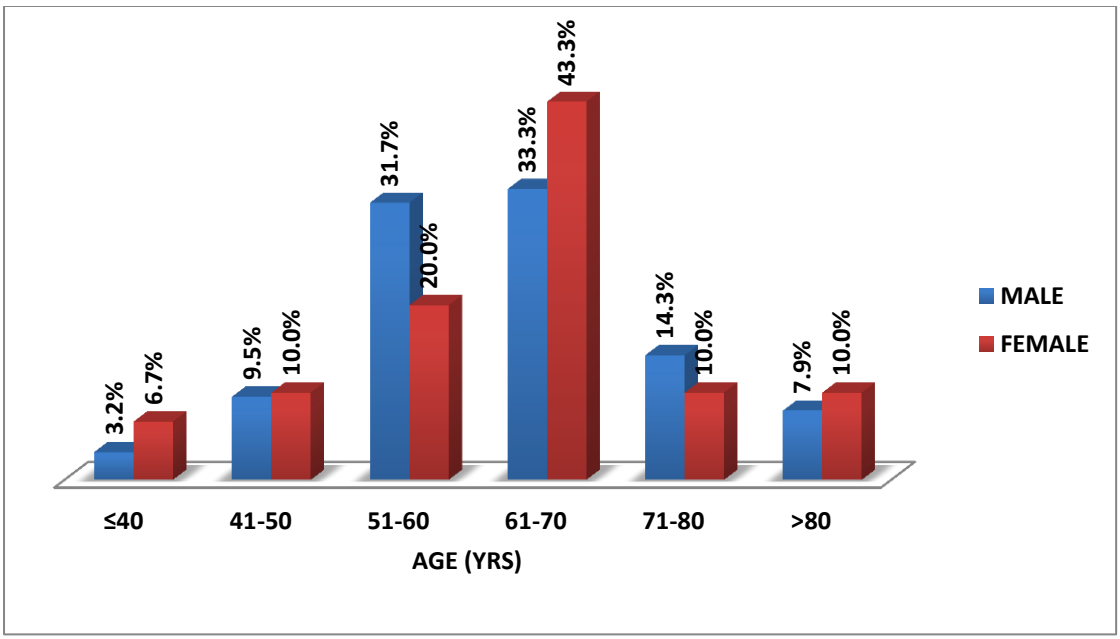


In this study, There were 63 (67.7) male patients and 30(32.3) female patients **.**there is male preponderance with male to female ratio 2.1:1 respectively.

Table No 4: ASSOCIATION OF AGE AND SEX

AGE (YRS)	MALE		FEMALE		p value
	N	%	N	%	
≤40	2	3.2%	2	6.7%	0.772
41-50	6	9.5%	3	10.0%	
51-60	20	31.7%	6	20.0%	
61-70	21	33.3%	13	43.3%	
71-80	9	14.3%	3	10.0%	
>80	5	7.9%	3	10.0%	
Total	63	100.0%	30	100.0%	

Figure No 11: ASSOCIATION OF AGE AND SEX

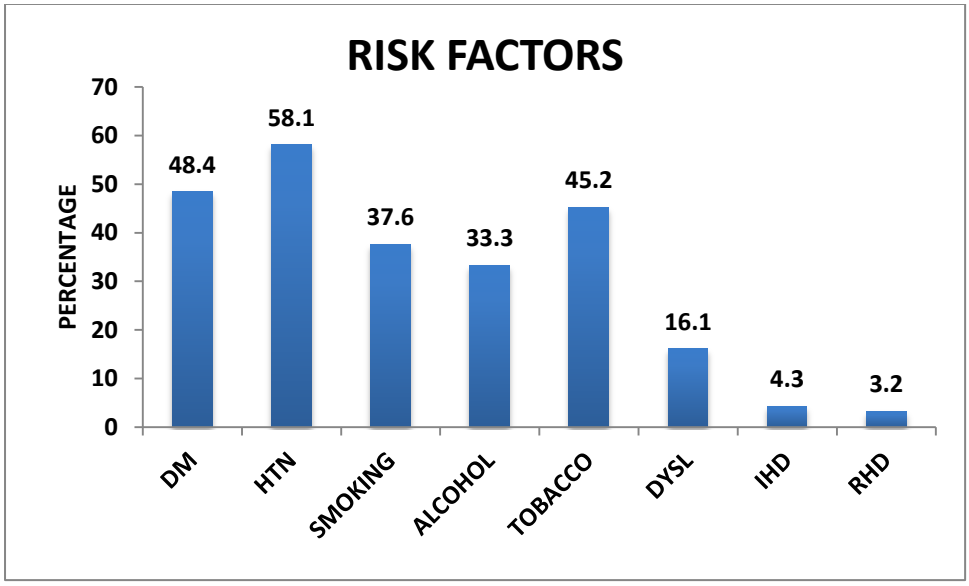


In this study, stroke in less than 60 year age group is common in males than females.

Table No 5: DISTRIBUTION OF CASES ACCORDING TO RISK FACTORS

RISK FACTORS	N	%
DM	45	48.4
HTN	54	58.1
SMOKING	35	37.6
ALCOHOL	31	33.3
TOBACCO	42	45.2
DYSL	15	16.1
IHD	4	4.3
RHD	3	3.2

Figure No 12: DISTRIBUTION OF CASES ACCORDING TO RISK FACTORS

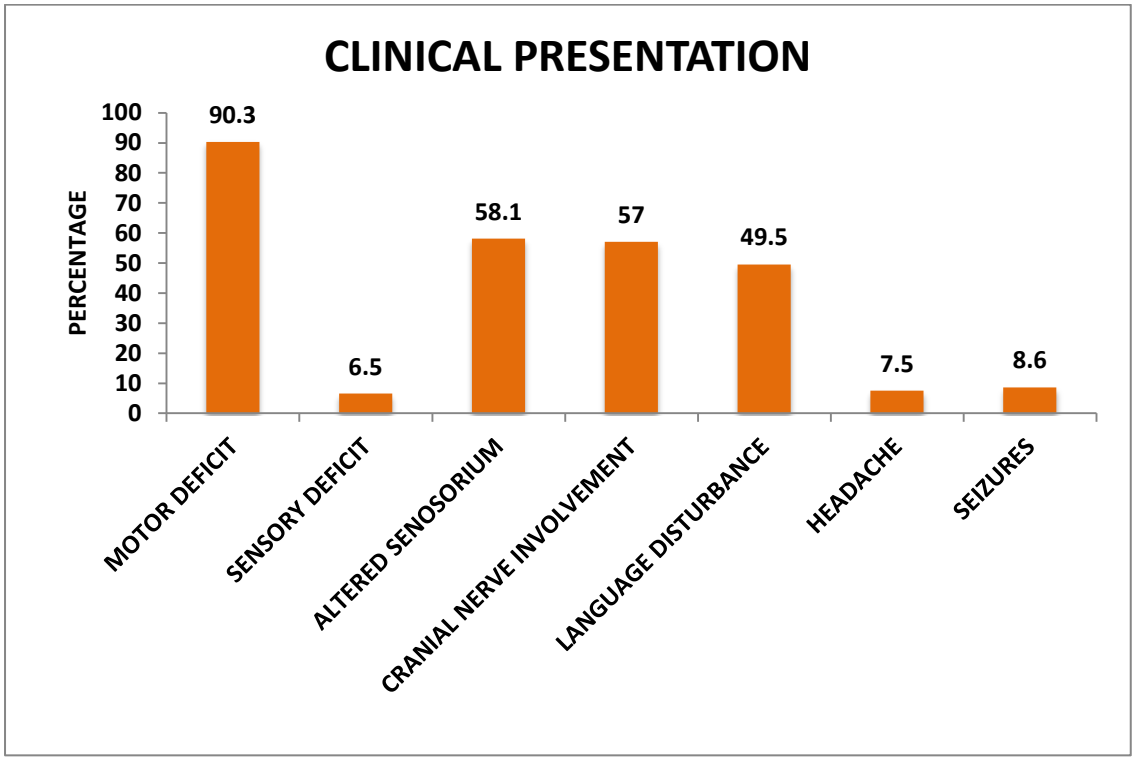


In this study, hypertension is the major risk factor, followed by diabetes mellitus, tobacco chewing and smoking etc.

Table No 6: DISTRIBUTION OF CASES ACCORDING TO CLINICAL PRESENTATION

CLINICAL PRESENTATION	N	%
MOTOR DEFICIT	84	90.3
SENSORY DEFICIT	6	6.5
ALTERED SENOSORIUM	54	58.1
CRANIAL NERVE INVOLVEMENT	53	57
LANGUAGE DISTURBANCE	46	49.5
HEADACHE	7	7.5
SEIZURES	8	8.6

Figure No 13: DISTRIBUTION OF CASES ACCORDING TO CLINICAL PRESENTATION

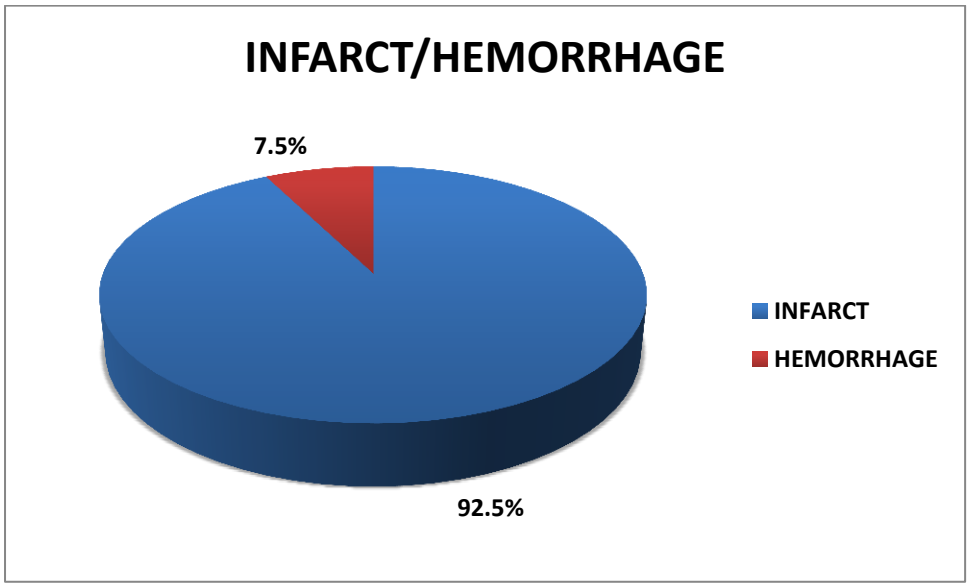


In this study most common presentation is with motor deficit, followed by altered sensorium, cranial nerve involvement etc.

Table No 7: DISTRIBUTION OF CASES ACCORDING TO INFARCT/HEMORRHAGE

INFARCT/HEMORRHAGE	N	%
INFARCT	86	92.5
HEMORRHAGE	7	7.5
Total	93	100

Figure No 14: DISTRIBUTION OF CASES ACCORDING TO INFARCT/ HEMORRHAGE

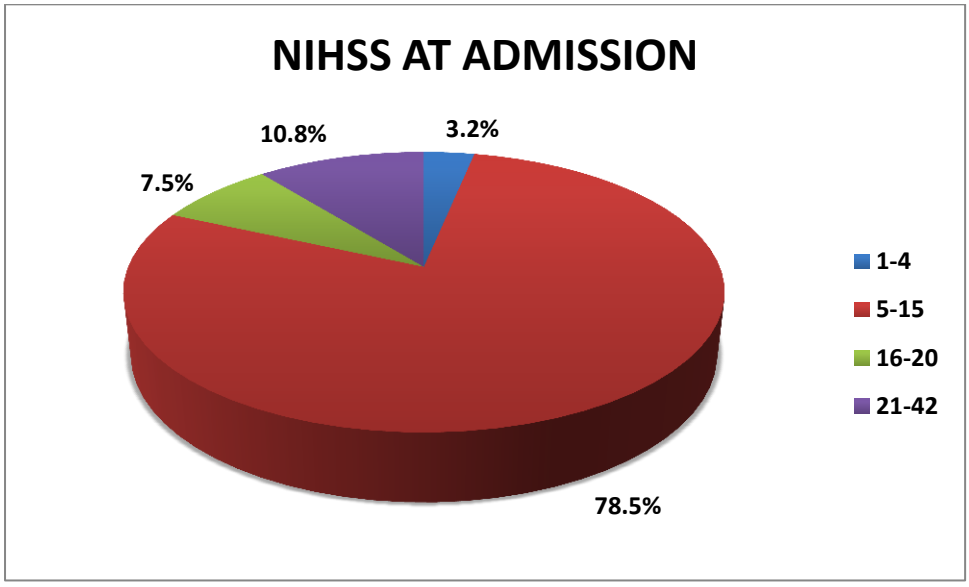


In this study, 7.5% patients had hemorrhagic stroke and 92.5% patients had ischemic stroke**.**

Table No 8: DISTRIBUTION OF CASES ACCORDING TO NIHSS AT ADMISSION

NIHSS AT ADMISSION		N	%
1-4	**Minor stroke**	3	3.2
5-15	**Moderate stroke**	73	78.5
16-20	**Moderate to severe stroke**	7	7.5
21-42	**Severe stroke**	10	10.8
Total		93	100

Figure No 15: DISTRIBUTION OF CASES ACCORDING TO NIHSS AT ADMISSION



In this study, 3 patients had minor stroke, 73 patients had moderate stroke, 7 patients had moderate to severe stroke and 10 patients had severe stroke.

Table No 9: DISTRIBUTION OF CASES ACCORDING TO NIHSS AFTER 1 MONTH

NIHSS AFTER 1 MONTH	N	%
1-4	11	12.8
5-15	69	80.2
16-20	4	4.7
21-42	2	2.3
Total	**86**	**100**

Figure No 16: DISTRIBUTION OF CASES ACCORDING TO NIHSS AFTER 1 MONTH

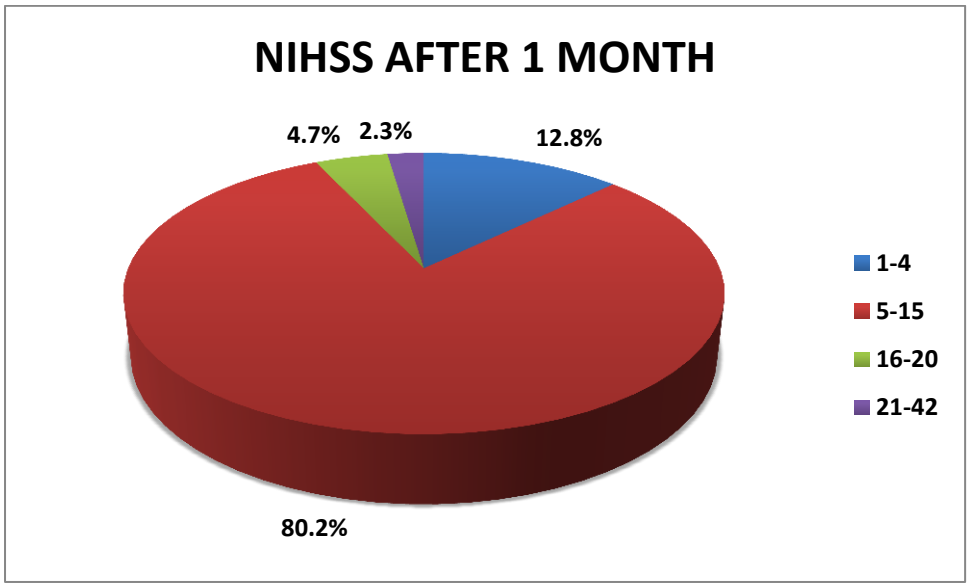


Table No 10: MEAN NIHSS BETWEEN ADMISSION AND AFTER 1 MONTH

NIHSS	Min	Max	Mean	SD	p value
AT ADMISSION	4	29	11.7	5.7	<0.001*
AFTER 1 MONTH	0	23	8.9	4.4	

Note: * significant at 5% level of significance (p<0.05)

Figure No 17: MEAN NIHSS BETWEEN ADMISSION AND AFTER 1 MONTH

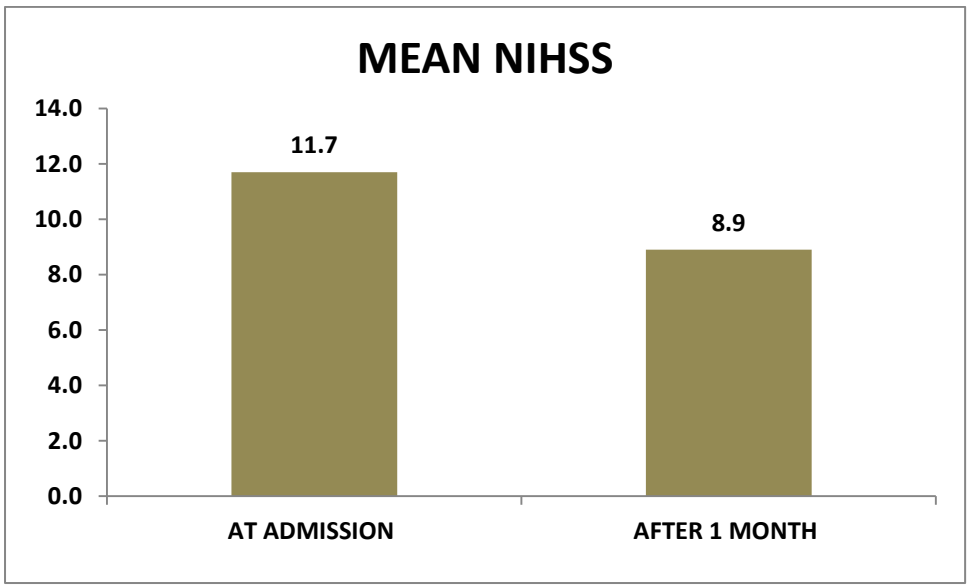
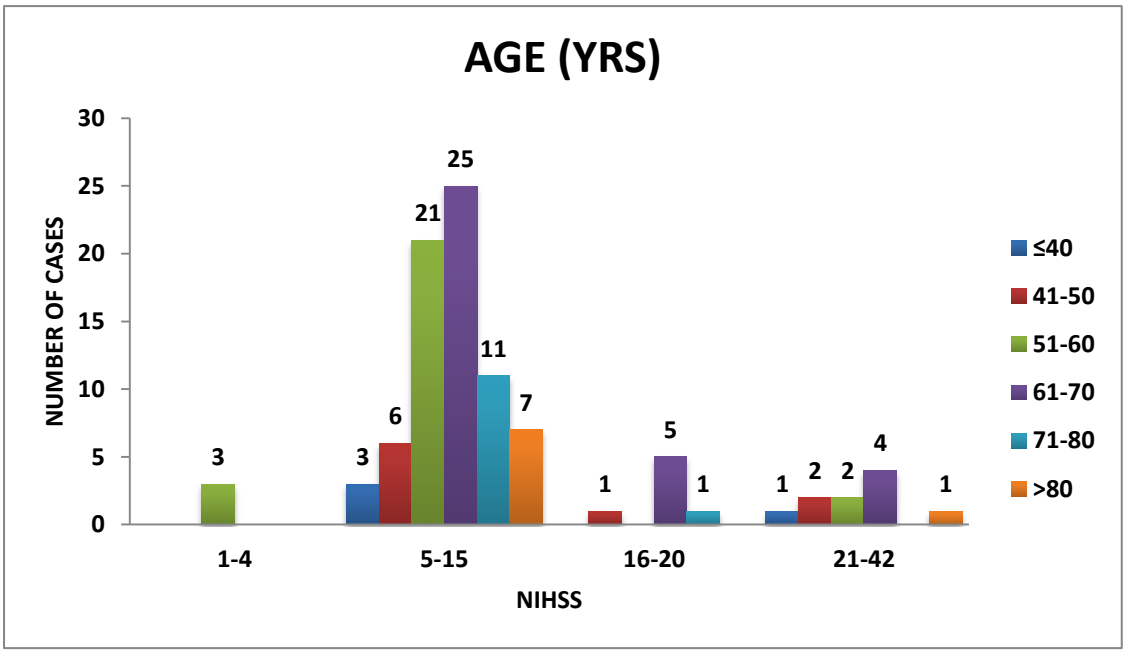


Table No 11: ASSOCIATION OF AGE AND NIHSS AT ADMISSION

AGE (YRS)	NIHSS AT ADMISSION								p value
	1-4		5-15		16-20		21-42		
	N	%	N	%	N	%	N	%	
≤40	0	0.0%	3	4.1%	0	0.0%	1	10.0%	0.310
41-50	0	0.0%	6	8.2%	1	14.3%	2	20.0%	
51-60	3	100.0%	21	28.8%	0	0.0%	2	20.0%	
61-70	0	0.0%	25	34.2%	5	71.4%	4	40.0%	
71-80	0	0.0%	11	15.1%	1	14.3%	0	0.0%	
>80	0	0.0%	7	9.6%	0	0.0%	1	10.0%	
Total	3	100.0%	73	100.0%	7	100.0%	1089	100.0%	

Figure No 18: ASSOCIATION OF AGE AND NIHSS AT ADMISSION



In this study, as the age increases the percentage of patients having moderate and severe stroke increases.

Table No 12: ASSOCIATION OF AGE AND NIHSS AFTER 1 MONTH

AGE (YRS)	NIHSS AFTER 1 MONTH								p value
	1-4		5-15		16-20		21-42		
	N	%	N	%	N	%	N	%	
≤40	0	0.0%	3	4.3%	0	0.0%	0	0.0%	0.576
41-50	0	0.0%	7	10.1%	0	0.0%	0	0.0%	
51-60	4	36.4%	20	29.0%	0	0.0%	2	100.0%	
61-70	3	27.3%	24	34.8%	3	75.0%	0	0.0%	
71-80	3	27.3%	9	13.0%	0	0.0%	0	0.0%	
>80	1	9.1%	6	8.7%	1	25.0%	0	0.0%	
Total	11	100.0%	69	100.0%	4	100.0%	2	100.0%	

Figure No 19: ASSOCIATION OF AGE AND NIHSS AFTER 1 MONTH

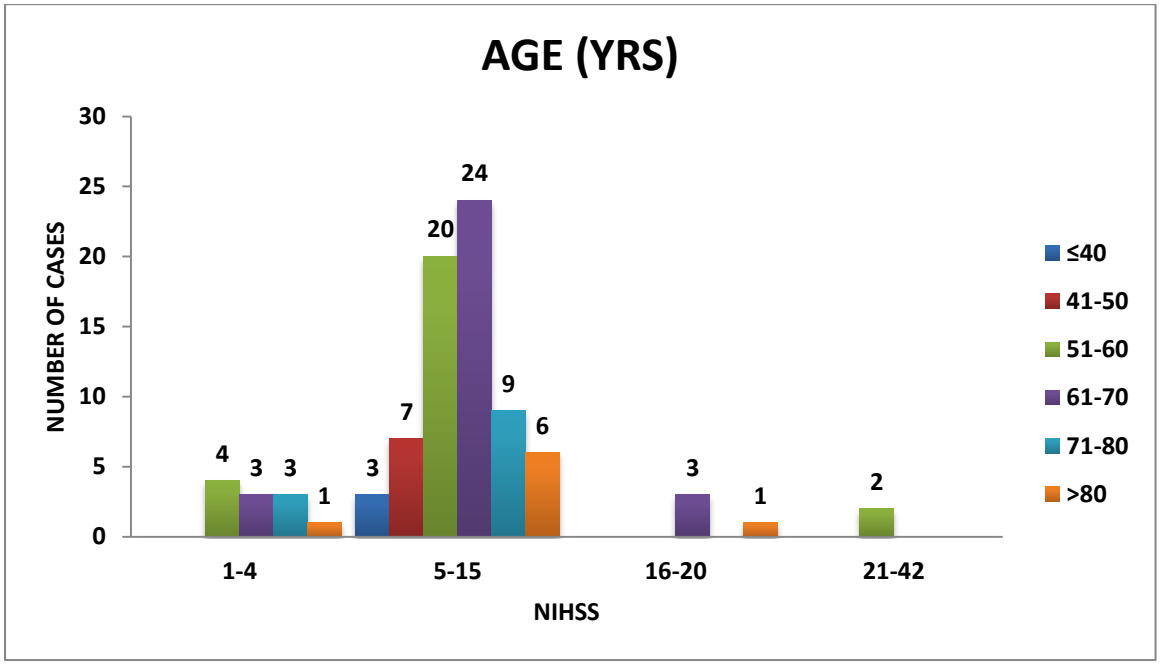
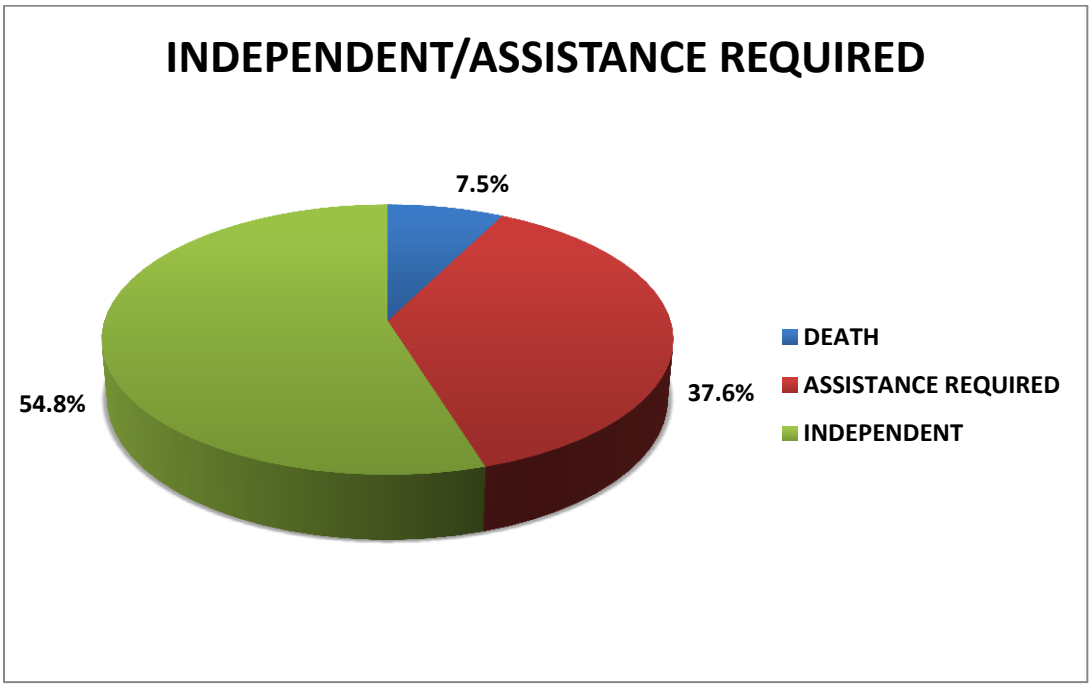


Table No 13: DISTRIBUTION OF CASES ACCORDING TO INDEPENDENT/ASSISTANCE REQUIRED

INDEPENDENT/ASSISTANCE REQUIRED	N	%
DEATH	7	7.5
ASSISTANCE REQUIRED	35	37.6
INDEPENDENT	51	54.8
Total	93	100

Figure No 20: DISTRIBUTION OF CASES ACCORDING TO INDEPENDENT/ ASSISTANCE REQUIRED



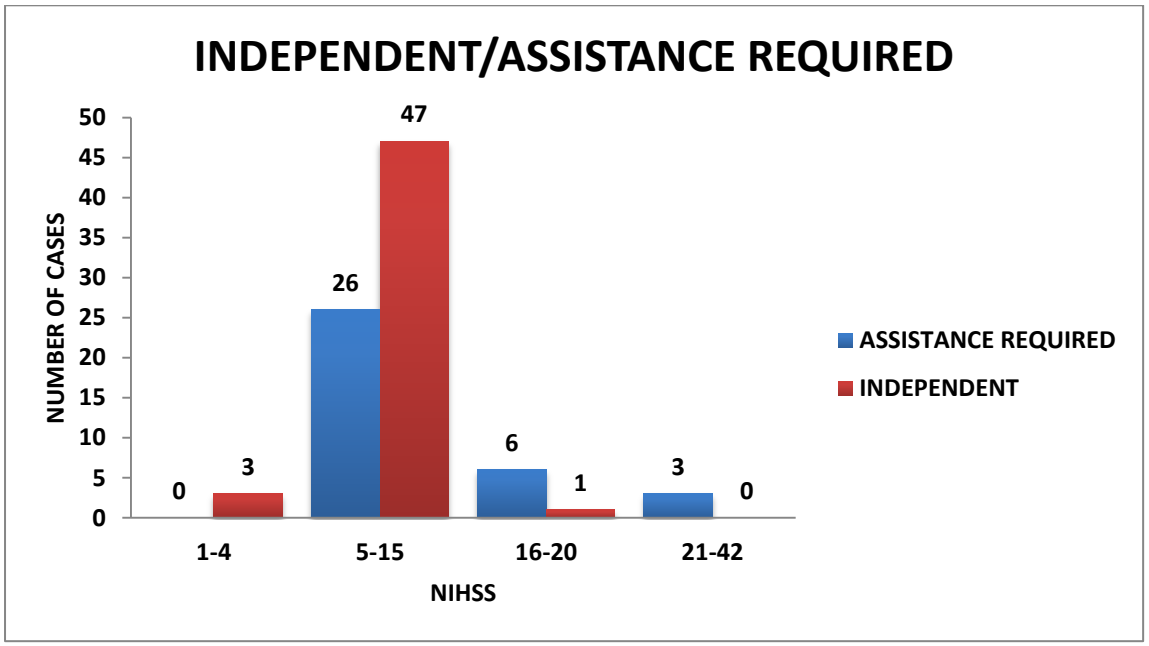
In this study, 7.5% had died at one month, 54.8% were independent at home and 37.6% patients required assistance at the end of one month.

Table No 14: ASSOCIATION OF INDEPENDENT/ASSISTANCE REQUIRED AND NIHSS AT ADMISSION

INDEPENDENT/ASSISTANCE REQUIRED	NIHSS AT ADMISSION								p value
	1-4		5-15		16-20		21-42		
	N	%	N	%	N	%	N	%	
ASSISTANCE REQUIRED	0	0.0%	26	35.6%	6	85.7%	3	100.0 %	<0.001*
INDEPENDENT	3	100.0 %	47	64.4%	1	14.3%	0	0.0%	
Total	3	100.0 %	73	100.0 %	7	100.0%	3	100.0 %	

Note: * significant at 5% level of significance (p<0.05)

Figure No 21: ASSOCIATION OF INDEPENDENT/ASSISTANCE REQUIRED AND NIHSS AT ADMISSION



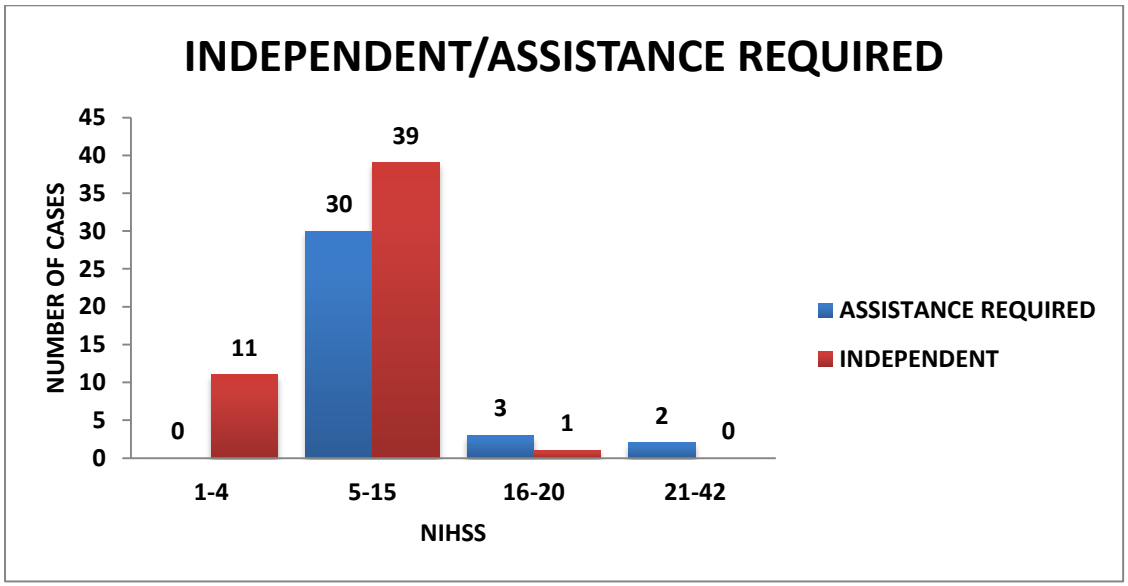
In this study, after 1 month of stroke among 3 patients who had baseline NIHSS score 1-4, all 3 (100%) are independent at home, among 73 patients who had baseline NIHSS score 5-15, 47 (64.4%) are independent and 26 (35.6%) required assistance, among 7 patients who had score 16-20, 1 (14.3%) patient was independent at home, 6(85.7%) required assistance, and among 10 patients who had score more than 20, 7 (70%) died , 3 (30%) required assistance and none of them are home independent.

Table No 15: ASSOCIATION OF INDEPENDENT/ASSISTANCE REQUIRED AND NIHSS AFTER 1 MONTH

INDEPENDENT /ASSISTANCE REQUIRED	NIHSS AFTER 1 MONTH								p value
	1-4		5-15		16-20		21-42		
	N	%	N	%	N	%	N	%	
ASSISTANCE REQUIRED	0	0.0%	30	43.5%	3	75.0%	2	100.0%	0.005 *
INDEPENDENT	11	100.0%	39	56.5%	1	25.0%	0	0.0%	
Total	11	100.0%	69	100.0 %	4	100.0%	2	100.0%	

Note: * significant at 5% level of significance ($p<0.05$)

Figure No 22: ASSOCIATION OF INDEPENDENT/ASSISTANCE REQUIRED AND NIHSS AFTER 1 MONTH



In this study, the patients who were independent at home after one month had less NIHSS score compared to patients who required assistance.

Table No 16: DESCRIPTIVE PARAMETERS OF BLOOD GLUCOSE AND HbA1C

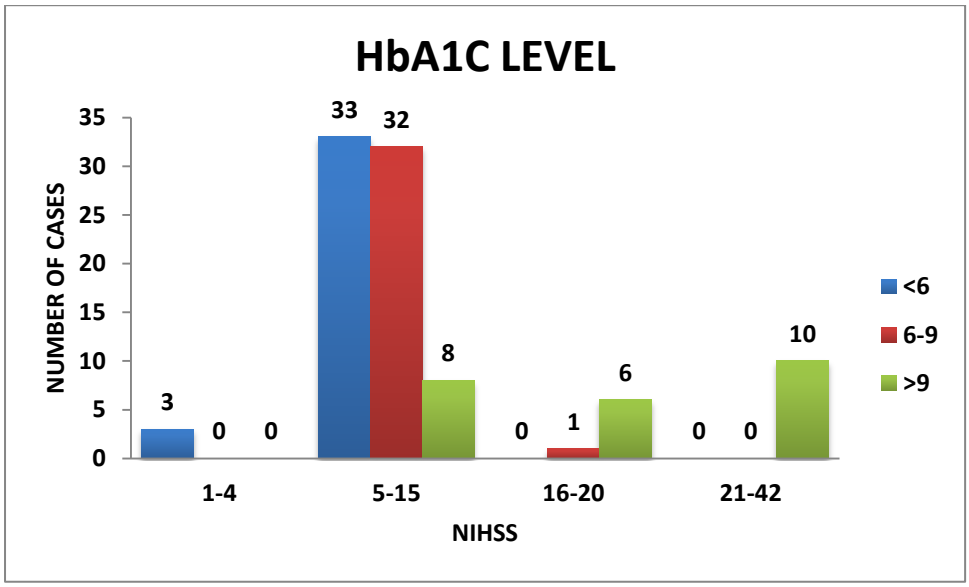
	Min	Max	Mean	SD
BLOOD GLUCOSE ON ADMISSION	80	420	196.1	82.4
HbA1C	4.5	12.8	7.3	2.0

Table No 17: ASSOCIATION OF HbA1C LEVELS AND NIHSS AT ADMISSION

HbA1C	NIHSS AT ADMISSION								p value
	1-4		5-15		16-20		21-42		
	N	%	N	%	N	%	N	%	
<6	3	100.0%	33	45.2%	0	0.0%	0	0.0%	<0.001*
6-9	0	0.0%	32	43.8%	1	14.3%	0	0.0%	
>9	0	0.0%	8	11.0%	6	85.7%	10	100.0%	
Total	3	100.0%	73	100.0%	7	100.0%	10	100.0%	

Note: * significant at 5% level of significance (p<0.05)

Figure No 23: ASSOCIATION OF HbA1C LEVELS AND NIHSS AT ADMISSION



In this study, the severity of the stroke is more with poorly controlled diabetes.

DISCUSSION

Stroke is a global epidemic and an important cause of morbidity and mortality. It is the second most common cause of death and may soon become the leading cause of death worldwide.

Stroke is a medical emergency and can cause permanent neurological damage, complications and death. In view of the long-term disabilities caused by stroke the need for an accurate early prediction of future functional abilities is paramount for setting therapeutic goals, starting early rehabilitation, planning of implementing home adjustments and community support tailored to patients needs, and informing patients about their prospects and prognosis.

The National Institutes of Health Stroke Scale (NIHSS) is a well-validated, reliable scoring system for use specifically with stroke patients. The National Institutes of Health Stroke Scale (NIHSS) can be used as a standard measurement instrument by physicians to evaluate the severity of a patient and outcome.

This study is assessment of outcome of acute stroke using national institute of health stroke scale (NIHSS).

93 patients admitted to Shri B.M.Patil Medical College Vijayapur, who met inclusion criteria were included in the study, age group of the patient ranged from 26yrs to 90 years, with mean age group 63.3$\pm$11.8, maximum number of patients were in the age group of 61-70 years. Age is non modifiable risk factor that correlates best with stroke**.** The study by **Sacco RL, Risk factors, outcomes, and stroke sub types for Ischemic Stroke** shows that majority of ischemic strokes occur in persons older than 65 years.[67]

There were 63 (67.7) male patients and 30(32.3) female patients with male to female ratio 2.1:1 respectively. Stroke is common in men than in women. **Hyvarinen M et al study** reported that there is male preponderance with 55%men and 45%women out of 21706 cases.[62]

In our study 7 patients (7.5%) had hemorrhagic stroke and 86 (92.5%) had ischemic stroke. Analysis of data from large stroke studies shows approximately 80% of all stroke are ischemic and 20%are hemorrhagic.

In this study common risk factors were diabetes mellitus type 2, hypertension, dyslipidemia, smoking, tobacco chewing, alcoholism, ischemic heart disease, rheumatic heart disease.

These risk factors are comparable to other studies of stroke.[63,64]

Commonest presentation was motor weakness seen in 90.3% of patients, while altered sensorium cranial nerve involvement and speech disturbance is seen in 58.1% 57%and 49.5% respectively. Other symptoms include seizures, head ache, sensory deficit.

The clinical severity of stroke and outcome after one month of stroke is measured using NIHSS score on admission and after one month.

In this study the patients diagnosed with stroke are further divided in to minor stroke (NIHSS 1-4), moderate stroke (NIHSS 5-15) moderate to severe stroke (NIHSS 16-20) and severe stroke (NIHSS 21-42) based on baseline NIHSS score.

3.2% patients had minor stroke, 78.5% patients had moderate stroke, 7.5% patients had moderate to severe stroke and 10.8% patients had severe stroke.

Analyzing NIHSS score and age it shows that as the age increases the percentage of patients having moderate and severe stroke increases. For example only 29% of patients had moderate stroke in 41-50 age group while 61-70% had moderate to severe stroke in 61-70 age group.

NIHSS score after 1 month of stroke shows that 11(11.8%) patients had score between 1-4, 69 (74.19%) patients had score between 5-15, 4 (4.3%) patients had score between 16-20, 2 (2.15%) patients had score21-42, and 7 (7.52%) patients died.

After 1 month of stroke, 35 (37.6%) patients were home independent, 51(54.8%) patients required assistance at home and 7(7.5%) patients had died.

Among 35 patients who were home independent at one month of stroke, 3 patients had score 1-4, 47 had score5-15, 1 had score 16-20 and none had score more than 20.

Among 51 patients who required assistance at one month following stroke, 26 patients had score 5-15, 6 had score 16-20, 3 had score more than 20 and none of them had score less than 5. And all the 7 patients who had died had score more than 20.

In other way after 1 month of stroke, among 3 patients who had baseline NIHSS score 1-4, all 3 (100%) are independent at home, among 73 patients who had baseline NIHSS score 5-15, 47 (64.4%) are independent and 26 (35.6%) required assistance, among 7 patients who had score 16-20, 1 (14.3%) patient was independent at home, 6(85.7%) required assistance, and among 10 patients who had score more than 20, 7 (70%) died , 3 (30%) required assistance and none of them are home independent.

No patient with NIHSS score less than 20 died, all the patients who died had severe stroke (NIHSS>20).

In this study the results shows that the patients with the NIHSS score >= 16 have high chance of severe disability or death. Whereas patients with score <16 have chances for better recovery. The results are consistent with the study **(Adams Hp Jr,et.al. Baseline NIHSS score strongly predicts outcome after stroke.)**[52] Shows NIHSS strongly predicts the likelihood of patient recovery after stroke. A score >= 16 shows high probability of death or severe disability where as score <=6 have good recovery.

Similar study done by **Gert kwakkel et al. predictive value of the NIHSS for ADL outcome after ischemic hemispheric stroke: Does timing of early assessment matters?.**[65] Showed similar results**.**

In this study patient HBA1C ranged from 4.5gm% to 12.8gm%. Among 3 patients who had minor stroke (NIHSS 1-4) , all 3 had HBA1C less than 6%. Among 73 patients with moderate stroke (NIHSS 5-15), 33(45.2%) had HBA1C <6%, 32(43.8%) had HBA1C 6-9, 8(11%) had HBA1C >9%. Among 7 patients who had moderate to severe stroke (NIHSS 16-20) 1 had HBA1C 6-9%, 6(85.7%) had HBA1C >9% none had HBA1C <6%. Shows that the severity of the stroke, so as the NIHSS score increases with increasing HBA1C level.

It is consistent with other studies. **Study done by Chinmay J Kulkarni et al** [66] showed the similar inference that the poorly controlled D.M has large size of lesion and high NIHSS score on admission.

SUMMARY

1. As the age increases incidence of stroke increases, age is the independent risk factor for acute stroke.

2. Maximum numbers of patients were in the age group of 60-70.

3. Stroke is more common in males (67.7%) compared to females (32.3%),with male :female ratio of 2.1:1. In the same way multiple risk factors are common in males compared to females.

4. Systemic hypertension was the most common risk factor associated with stroke (58.1%) followed by diabetes mellitus (48.4%) and smoking (37.6%).

5. Motor deficit is the most common presentation followed by altered sensorium and speech disturbance.

6. NIHSS is most helpful in identifying patients with acute stroke.

7. NIHSS is helpful in assessment and stratification and further course of management. Among patients identified with stroke most had moderate stroke compared to moderate to severe stroke and severe stroke.

8. The NIHSS score on day of admission predicts the outcome of stroke, lesser the score better the outcome of stroke. And all the patients who had died had NIHSS score more than 20.

9. Increased severity of the stroke is seen in poorly controlled diabetes mellitus.

10. Medical and paramedical staff can be trained to administer NIHSS for early recognition and effective treatment of acute stroke.

CONCLUSION

1. NIHSS score correlates well with the diagnosis and severity of the stroke.

2. Baseline NIHSS score is helpful in assessment and stratification of the stroke patients and also helps in further course of management of stroke.

3. Baseline NIHSS score helps in predicting the outcome of the patient. Lesser the baseline score better will be the outcome.

4. HBA1C levels correlate well with the severity of the stroke and NIHSS on admission.

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ANNEXURE I

B.L.D.E.UNIVERSITY'S
SHRI.B.M.PATIL MEDICAL COLLEGE HOSPITAL AND RESEARCH CENTRE
VIJAYAPUR – 586103

IEC/Ref/100-142/17
14/11/2017

INSTITUTIONAL ETHICAL COMMITTEE

INSTITUTIONAL ETHICAL CLEARANCE CERTIFICATE

The Ethical Committee of this college met on **13-11-2017** at **03-15pm** Scrutinize the Synopsis of Postgraduate Students of this college from Ethical Clearance point of view. After scrutiny the following original/corrected and revised version synopsis of the Thesis has accorded Ethical Clearance.

Title : UTILIZATION OF NATIONAL INSTITUTE OF HEALTH STROKE SCALE (NIHSS) IN ASSESSING THE SEVERITY AND PREDICTING THE OUTCOME OF ACUTE STROKE,

Name of P.G. Student : **DR. SANJEETH M B**
DEPARTMENT OF GENERAL MEDICINE.

Name of Guide/Co-investigator : **DR. S.S.DEVARMANI**
PROFESSOR DEPARTMENT OF MEDICINE

DR .RAGHAVENDRA KULKARNI
CHAIRMAN, IEC,
Institutional Ethical Committee
BLDEU's Shri B.M. Patil
Medical College,BIJAPUR-586103.

Following documents were placed before Ethical Committee for Scrutinization:

1. Copy of Synopsis/Research project
2. Copy of informed consent form
3. Any other relevant documents

ANNEXURE II

INFORMED CONSENT FORM

TITLE OF THE PROJECT -

"ASSESSMENT OF OUTCOME OF ACUTE STROKE USING NATIONAL INSTITUTE OF HEALTH STROKE SCALE(NIHSS)."

PRINCIPAL INVESTIGATOR -

P.G.GUIDE NAME -

CHAIRMAN ETHICAL COMMITTEE

All aspects of this consent form are explained to the patient in the language understood by him/her.

I) **INFORMED PART**

1) PURPOSE OF RESEARCH:

I have been informed about this study. I have also been given a free choice of participation in this study.

2) PROCEDURE:

I am aware that in addition to routine care received I will be asked series of questions by the investigator. I have been asked to undergo the necessary investigations and treatment, which will help the investigator in this study.

3) RISK AND DISCOMFORTS:

I understand that I may experience some pain and discomfort during the examination or during my treatment. This is mainly the result of my condition and the procedure of this study is not expected to exaggerate these feelings that are associated with the usual course of treatment.

4)BENEFITS:

I understand that my participation in this study will help to patients survival and better outcome.

5) CONFIDENTIALITY:

I understand that the medical information produced by this study will become a part of Hospital records and will be subject to the confidentiality and privacy regulation. Information of a sensitive personal nature will not be a part of the medical records, but will be stored in the investigator's research file and identified only by a code number. The code-key connecting name to numbers will be kept in a separate location.

If the data are used for publication in the medical literature or for teaching purpose, no name will be used and other identifiers such as photographs and audio or videotapes will be used only with my special written permission. I understand that I may see the photographs and videotapes and hear the audiotapes before giving this permission.

6) REQUEST FOR MORE INFORMATION:

I understand that I may ask more questions about the study at any time.

is available to answer my questions or concerns. I understand that I will be informed of any significant new findings discovered during the course of the study, which might influence my continued participation.

If during the study, or later, I wish to discuss my participation in or concerns regarding this study with a person not directly involved, I am aware that the social worker of the hospital is available to talk with me. A copy of this consent form will be given to me to keep for careful reading.

7) REFUSAL OR WITHDRAWAL OF PARTICIPATION:

I understand that my participation is voluntary and that I may refuse to participate or may withdraw consent and discontinue participation in the study at any time without prejudice to my present or future care at this hospital. I also understand that may terminate my participation in the study after he has explained the reasons for doing so and has helped arrange for my continued care by my own physician or physical therapist, if this is appropriate.

8) INJURY STATEMENT:

I understand that in the unlikely event of injury to me resulting directly from my participation in this study, if such injury were reported promptly, the appropriate treatment would be available to me, but no further compensation would be provided. I understand that by my agreement to participate in this study I am not waiving any of my legal rights.

I have explained to _____the purpose of the research, the procedures required and the possible risks and benefits to the best of my ability in patient's own language.

_____ _____

Date

(Investigator)

II) **STUDY SUBJECT CONSENT STATEMENT:**

I confirm that has explained to me the purpose of research, the study procedures that I will undergo, and the possible risks and discomforts as well as benefits that I may experience in my own language. I have read and I

understand this consent form. Therefore, I agree to give consent to participate as a subject in this research project.

Participant / Guardian	Date
Witness to signature	Date

ANNEXURE III

PROFORMA

SCHEME OF CASE TAKING

Name: CASE NO:

Age: OP/IP NO:

Sex: DOA:

Religion: DOD:

Occupation:

Address:

Presenting Complaints:

History of presenting complaints:

Past History:

Family history:

Personal History:

Diet –

Appetite –

Sleep –

Bowel –

Bladder –

Habits –

Obstetric and menstrual history (in females):
Physical Examination:

1. .Built :
2. .State of Nutrition :
3. Hydration status :
4. .Eyes :
5. Ears:
6. .Oral cavity:
7. .Lymphadencopathy :
8. .Pedal Edema:
9. .Examination of peripheral vessels and neck vessels

Vitals:

Pulse rate: Respiratory rate:

Blood pressure: Temperature:

Systematic Examination:

1.Nervous system examination:

a.Handed ness:

b.Mental status examination

i.Memory :

iiOrientation :

iii.Speech and language :

iv intelligence:

v.level of consciousness:

c.Cranial nerves examination

d.Motor system examination:

	Right		Left	
	UL	LL	UL	LL
a.Nutrition				
b.Tone				
c.Power				
d.Coordination				
e.Involuntary movements:				

f.Reflexes:	Right	Left
a.Superficial		
i.Abdominal		
ii.Cremastric		
iii Corneal		
iv Conjunctival		
v .Plantar		
b.Deep		
i.Biceps		
ii.Triceps		
iii.Supinator		
iv.Knee		
v.Ankle		
c.Primitive reflex –		

d.Sensory system examination:

	Right	Left
Touch		
Pain		
Temperature		
Vibration		
Joint sense		
Cortical sensation		

e.Cerebrallar system examination:

	Right	Left
Finger Nose test		
Knee Heel test		
Dysdiadokinesia		

g.GAIT:

h.Signs of Autonomic Disturbances – Present/Absent

i.Signs of Meningeal iriitation

Neck Rigidity/Kernings sign/Brudzenski sign

j.Examination of skull and spine – Deformity Present /Absent

2. Cardiovascular System:

3. Respiratory System:

4. Per Abdomen Examination:

Final diagnosis:

NIHSS score

NATIONAL INSTITUTE OF HEALTH STROKE SCALE		
CATAGORY		**SCORE**
1a. Level of Consciousness(LOC):	**0 = Alert;** **1=Drowsy** **2=Stuporous** **3=Coma**	
1b. LOC Questions: (Month, Age)	**0=Answers both question correctly** **1=Answers one correctly** **2=Answers both incorrect**	
1c.LOC Commands: (Open/Close eyes, make fist & let go)	**0=Obeys both correctly** **1=Obeys one correctly** **2=Both incorrect**	
2.Best gaze: (Eyes open- pt follows examiner's fingers or face)	**0=Normal** **1=Partial gaze palsy** **2=Forced deviation**	
3.Visual: (Introduce visual stimulus/threat to pt's visual field quadrants. Cover 1 eye and hold up fingers in all 4 quadrants.)	**0=No visual loss** **1=Partial hemianopsia** **2=complete hemianopsia** **3=Bilateral hemianopsia**	
4.Facial Palsy: (Show teeth, raise eyebrows and squeeze eyes tightly shut.)	**0 = Normal symmetrical movements.** **1 = Minor paralysis** **2 = Partial paralysis** **3 = Complete paralysis of one or both sides**	
5. Motor Arm: (Elevate extremity to 90 degrees and score drift/movement. Count to 10 out loud and use	**0 = No drift; limb holds 90 (or 45) degrees for full 10 seconds.** **1 = Drift** **2 = Some effort against gravity**	

fingers for visual cue.)	**3 = No effort against gravity.** **4 = No movement.** **UN = Amputation or joint fusion,** **5a. Left Arm** **5b. Right Arm**	
6. Motor Leg: (Elevate extremity to 90 degrees and score drift/movement. Count to 10 out loud and use fingers for visual cue.)	**0 = No drift; leg holds 30-degree position for full 5 seconds.** **1 = Drift** **2 = Some effort against gravity** **3 = No effort against gravity** **4 = No movement.** **UN = Amputation or joint fusion** **6a. Left Leg** **6b. Right Leg**	
7. Limb Ataxia: (Finger to nose, heal down shin)	**0 = Absent.** **1 = Present in one limb.** **2 = Present in two limbs.** **UN = Amputation or joint fusion**	
8. Sensory: (Pin prick to face, arms, trunk, and legs- compare sharpness side to side, or no feeling at all.)	**0 = Normal; no sensory loss.** **1 = Mild-to-moderate sensory loss** **2 = Severe to total sensory loss**	
9. Best Language: (Name items, describe picture, and read sentences. Don't forget glasses if they normally wear them.)	**0 = No aphasia; normal.** **1 = Mild-to-moderate aphasia** **2 = Severe aphasia** **3 = Mute, global aphasia; no usable speech or auditory** **comprehension.**	

10. Dysarthria: (Evaluate speech clarity by pt reading or repeating words on list.)	**0 = Normal.** **1 = Mild-to-moderate dysarthria** **2 = Severe dysarthria** **UN = Intubated or other physical barrier,** **explain:_____**	
11. Extinction and Inattention (formerly Neglect): (Use information form prior testing or double simultaneous stimuli testing to identify neglect face, arms, legs and visual fields.)	**0 = No abnormality.** **1 = Visual, tactile, auditory, spatial, or personal inattention or extinction to bilateral simultaneous stimulation in one of the sensory modalities.** **2 = Profound hemi-inattention or extinction to more than one modality; does not recognize.**	
NT= NOT TESTABLE		
TOTAL SCORE		

INVESTIGATIONS

PATHOLOGY:

1.)Complete blood count:	
Hb	gm/dl
Total count	Cells/cumm
Differential count Neutrophils	%
Lymphocytes	%
Eosinophils	%
Basophils	%
Monocytes	%
2.) ESR	At the end of 1st hour.
3.)Platelet Indices	
Platelet Count	
4.) Urine Routine	
Sugar	
Albumin	
Pus cell	
Epithelial cell	

BIOCHEMISTRY:

B urea	
S creatinine	
Total cholesterol	
Ldl	
Vldl	
Hdl	
S triglycerides	
Rbs	
Serum Electrolytes	
HbA1C	

ECG

2D ECHO

CHEST XRAY PA VIEW

CT BRAIN PLAIN/MRI

Other relevant investigations will be done when required.

NIHSS SCORE AFTER 30 DAYS

NATIONAL INSTITUTE OF HEALTH STROKE SCALE		
CATAGORY		**SCORE**
1a. Level of Consciousness(LOC):	**0 = Alert;** **1=Drowsy** **2=Stuporous** **3=Coma**	
1b. LOC Questions: (Month, Age)	**0=Answers both question correctly** **1=Answers one correctly** **2=Answers both incorrect**	
1c.LOC Commands: (Open/Close eyes, make fist & let go)	**0=Obeys both correctly** **1=Obeys one correctly** **2=Both incorrect**	
2.Best gaze: (Eyes open- pt follows examiner's fingers or face)	**0=Normal** **1=Partial gaze palsy** **2=Forced deviation**	
3.Visual: (Introduce visual stimulus/threat to pt's visual field quadrants. Cover 1 eye and hold up fingers in all 4 quadrants.)	**0=No visual loss** **1=Partial hemianopsia** **2=complete hemianopsia** **3=Bilateral hemianopsia**	
4.Facial Palsy: (Show teeth, raise eyebrows and squeeze eyes tightly shut.)	**0 = Normal symmetrical movements.** **1 = Minor paralysis** **2 = Partial paralysis** **3 = Complete paralysis of one or both sides**	
5. Motor Arm: (Elevate extremity to 90 degrees and score drift/movement. Count to	**0 = No drift; limb holds 90 (or 45) degrees for full 10 seconds.** **1 = Drift**	

10 out loud and use fingers for visual cue.)	**2 = Some effort against gravity** **3 = No effort against gravity.** **4 = No movement.** **UN = Amputation or joint fusion,** **5a. Left Arm** **5b. Right Arm**	
6. Motor Leg: (Elevate extremity to 90 degrees and score drift/movement. Count to 10 out loud and use fingers for visual cue.)	**0 = No drift; leg holds 30-degree position for full 5 seconds.** **1 = Drift** **2 = Some effort against gravity** **3 = No effort against gravity** **4 = No movement.** **UN = Amputation or joint fusion** **6a. Left Leg** **6b. Right Leg**	
7. Limb Ataxia: (Finger to nose, heal down shin)	**0 = Absent.** **1 = Present in one limb.** **2 = Present in two limbs.** **UN = Amputation or joint fusion**	
8. Sensory: (Pin prick to face, arms, trunk, and legs- compare sharpness side to side, or no feeling at all.)	**0 = Normal; no sensory loss.** **1 = Mild-to-moderate sensory loss** **2 = Severe to total sensory loss**	
9. Best Language: (Name items, describe picture, and read sentences. Don't forget glasses if they normally wear them.)	**0 = No aphasia; normal.** **1 = Mild-to-moderate aphasia** **2 = Severe aphasia** **3 = Mute, global aphasia; no usable speech or auditory** **comprehension.**	

10. Dysarthria: (Evaluate speech clarity by pt reading or repeating words on list.)	**0 = Normal.** **1 = Mild-to-moderate dysarthria** **2 = Severe dysarthria** **UN = Intubated or other physical barrier,** **explain:_____**	
11. Extinction and Inattention (formerly Neglect): (Use information form prior testing or double simultaneous stimuli testing to identify neglect face, arms, legs and visual fields.)	**0 = No abnormality.** **1 = Visual, tactile, auditory, spatial, or personal inattention or extinction to bilateral simultaneous stimulation in one of the sensory modalities.** **2 = Profound hemi-inattention or extinction to more than one modality; does not recognize.**	
NT= NOT TESTABLE		
TOTAL SCORE		

INDEPENDENT/REQUIRE ASSISTANCE AT HOME?

CONCLUSION

DATE:

SIGNATURE

MASTER CHART

SL NO	NAME	AGE	I.P.NUMBER	SEX	RISK FACTORS						CLINICAL PRESENTATION						INFARCT/ HEMORRHAGE	NIHSS		BLOOD GLUCOSE ON ADMISSION	HbA1C	I/A
					DM	HTN	SMOKING	ALCOHOL	TOBACCO	OTHER	MOTOR DEFICIT	SENSORY DEFICIT	SENOSORIUM	INVOLVEMENT	DISTURBANCE	OTHER		ADMISSION	AFTER 1 MONTH			
1	DURU	68	18/40837	M	P	P	A	P	P	NIL	P	A	P	P	P	NIL	INFARCT	1 9	15	420	9.2	AS
2	TUKARAM	60	19/7891	M	P	P	P	A	A	NIL	P	A	A	P	A	NIL	INFARCT	11	9	280	8.4	I
3	JAGANATH	55	18/2753	M	P	P	P	P	A	NIL	P	A	A	P	A	SEIZURES	HEMORRHAGE	9	9	270	7.5	AS
4	RAJASHEKHAR	55	18/2238	M	A	P	A	P	A	NIL	P	A	P	A	A	NIL	INFARCT	7	5	145	6.1	I
5	ANNAPPA	75	18/18450	M	P	A	A	P	P	NIL	P	A	A	P	P	NIL	INFARCT	12	11	298	9.2	I
6	KALLAPPA	86	20414	M	P	A	P	A	A	NIL	P	A	P	P	P	NIL	INFARCT	22	19	321	9.4	AS
7	SRISHAIL	72	18/19346	M	P	A	P	A	A	NIL	P	A	P	A	A	NIL	INFARCT	13	12	244	8	AS
8	SATTEWWA	75	5985	M	A	P	A	A	P	NIL	P	A	A	A	A	NIL	INFARCT	5	3	180	4.5	I
9	LAXMAN	60	18356	M	A	P	A	P	P	IHD	P	A	P	A	A	NIL	INFARCT	4	0	140	5	I
10	SHRISHAIL BIRADAR	60	18/18383	M	A	A	P	P	P	NIL	P	A	A	A	A	NIL	INFARCT	4	0	166	5.4	I
11	NEELABAI	75	8811	F	A	P	A	A	A	NIL	P	A	A	A	A	NIL	INFARCT	5	3	120	5.7	I
12	PARVATHI	75	16732	F	P	P	P	A	A	NIL	P	A	P	P	P	NIL	HEMORRHAGE	18	15	344	10.2	AS
13	NEELAWWA	68	24463	F	A	A	P	A	P	NIL	P	A	A	A	A	NIL	INFARCT	5	3	114	4.9	I
14	SIDDAWWA	65	25329	F	A	A	A	A	P	NIL	P	A	A	A	A	NIL	INFARCT	11	7	167	6.3	I
15	NAMDEV	70	24781	M	A	A	A	P	A	RHD	P	A	P	A	A	NIL	INFARCT	8	6	160	5.8	I
16	SHAKUNTALA	75	125711	F	A	A	A	P	P	DYSL	P	A	P	A	A	NIL	INFARCT	9	7	82	5.7	I
17	TIPPANNA	75	10442	M	A	A	A	A	A	NIL	P	A	A	A	A	NIL	INFARCT	6	6	191	5.9	I
18	HARISH	48	10645	F	A	P	A	A	A	NIL	P	A	A	A	A	NIL	INFARCT	10	9	88	5.4	I
19	YALLAPPA	40	15294	F	P	P	P	A	P	NIL	P	P	P	P	P	SEIZURES	HEMORRHAGE	22	DEATH	350	12.8	AS
20	MAYAPPA	60	18/15459	M	A	A	P	P	P	NIL	P	A	A	A	A	NIL	INFARCT	5	5	125	5.3	I
21	SANGAYA	64	15972	M	A	A	A	P	A	IHD	P	A	P	A	A	NIL	INFARCT	6	4	102	5.4	I

22	KAMALABAI	60	18/16234	M	P	A	A	A	P	NIL	A	A	A	P	A	NIL	INFARCT	13	13	211	8.1	AS
23	YALLAWWA	70	17488	F	P	P	A	A	P	NIL	P	P	P	P	P	NIL	HEMORRHAGE	19	18	276	9.6	I
24	PANDURAY	68	17774	F	P	A	A	A	A	DYSL	P	A	P	P	P	NIL	INFARCT	21	DEATH	254	9.1	AS
25	SHANTHABAI	66	14884	M	A	A	A	P	P	NIL	P	A	A	A	A	NIL	INFARCT	5	5	80	4.5	I
26	HANUMANTRAY	65	10240	F	P	A	P	A	P	NIL	P	A	P	P	P	NIL	INFARCT	18	17	294	9.4	AS
27	SAHEBGOWDA	70	12953	M	A	P	A	A	P	NIL	P	A	A	A	A	NIL	INFARCT	6	5	140	5.3	I
28	RAJESAB	45	10275	M	P	P	P	A	A	DYSL	P	P	P	P	P	SEIZURES	HEMORRHAGE	25	DEATH	420	11.1	AS
29	KAMALA	50	14888	M	A	P	A	P	P	DYSL	P	A	A	A	A	NIL	INFARCT	12	11	181	5.5	I
30	SIDDAMMA	66	13901	F	P	A	A	A	A	NIL	P	A	A	P	A	NIL	INFARCT	13	13	146	7.1	AS
31	SUMITRA	63	20509	F	P	P	A	A	A	DYSL	A	P	P	P	P	SEIZURES	INFARCT	28	DEATH	358	12.6	AS
32	JAGADISH	45	18237	F	P	A	P	A	A	NIL	P	A	A	P	P	NIL	INFARCT	27	DEATH	325	9.2	AS
33	AMRUTHA	58	18/24484	M	P	P	A	P	P	NIL	A	A	P	P	P	NIL	INFARCT	12	13	232	8.1	I
34	NAGAPPA	51	25249	F	A	P	A	A	P	DYSL	P	A	A	A	A	NIL	INFARCT	4	0	82	5.2	I
35	IRABASAPPA	85	25734	M	A	A	A	A	A	NIL	P	A	A	A	A	NIL	INFARCT	11	11	83	5.9	AS
36	BHIMRAYA	55	25661	M	P	P	A	A	P	NIL	A	A	P	P	P	NIL	INFARCT	8	6	187	7.4	I
37	RAMARAO	68	17373	M	P	A	P	A	P	IHD	P	P	P	P	P	HEADACHE	HEMORRHAGE	17	11	232	9.2	AS
38	SIKANDHAR	35	24775	M	A	A	A	A	A	RHD	P	A	A	A	A	NIL	INFARCT	6	6	123	5.4	I
39	SHANTAPPA	65	18/18450	M	P	P	A	P	A	NIL	A	P	P	P	P	NIL	INFARCT	22	DEATH	240	9.5	AS
40	BASAMMA	80	16590	F	A	A	A	A	P	NIL	P	A	A	A	A	NIL	INFARCT	5	4	151	5.2	I
41	MALLANAGOWDA	50	13848	M	A	P	A	A	P	NIL	P	A	A	A	A	SEIZURES	INFARCT	10	7	90	5.3	I
42	RATNABAI	65	21698	F	P	P	A	A	A	NIL	P	A	P	P	P	NIL	INFARCT	9	9	210	8	I
43	NINGAPPA	55	21699	M	P	A	P	P	P	NIL	P	A	P	P	P	NIL	INFARCT	11	10	250	9.3	I
44	BALU	85	19278	M	A	A	A	P	A	NIL	P	A	A	A	A	NIL	INFARCT	5	5	166	5.9	I
45	SHRISHAIL	72	19346	M	A	P	A	A	A	NIL	P	A	P	A	A	HEADACHE	HEMORRHAGE	12	9	144	4.7	AS
46	PEERAPPA	55	19207	M	A	P	P	A	A	DYSL	P	A	P	A	A	NIL	INFARCT	13	11	115	5.6	AS
47	BHIMAPPA	65	8172	M	P	P	P	P	P	NIL	P	A	P	P	P	NIL	INFARCT	29	DEATH	344	11.2	AS
48	MUTTAPPA	80	7689	M	P	P	A	A	P	IHD	P	A	P	P	P	NIL	INFARCT	9	6	256	9.2	I
49	HAJILAL	65	7223	M	P	A	P	P	A	NIL	P	A	P	P	P	NIL	INFARCT	7	7	278	9.8	I
50	KANTEWWA	70	13369	M	P	A	A	P	P	NIL	P	A	A	P	P	NIL	INFARCT	20	16	267	9.4	AS
51	SIDDAWWA	60	18/25329	F	P	P	A	A	P	DYSL	P	A	P	P	P	NIL	INFARCT	23	23	300	10.1	AS
52	SULOCHANA	90	12072	F	P	A	A	A	P	DYSL	P	A	A	P	A	NIL	INFARCT	12	9	220	8.2	I
53	NEELABAI	48	10897	F	P	P	A	A	P	NIL	P	A	A	P	A	NIL	INFARCT	11	10	196	8.9	AS

54	SARSWATHI	65	9503	F	A	P	A	A	A	NIL	A	A	A	A	A	NIL	INFARCT	13	11	156	4.8	AS
55	HANAMANTH	80	8587	M	P	A	P	A	A	NIL	A	A	P	P	P	NIL	INFARCT	12	13	241	9.4	AS
56	MADARSAB	45	8593	M	P	P	P	A	P	DYSL	P	A	P	P	P	NIL	INFARCT	13	9	292	9.9	I
57	DEVENDRAPPA	45	7941	M	P	P	P	P	P	DYSL	A	A	A	P	P	NIL	INFARCT	17	14	193	8.3	AS
58	PUTALABAI	90	18802	F	P	A	P	A	P	NIL	P	A	A	A	A	NIL	INFARCT	6	6	189	7.4	I
59	KAMALA	55	18/14888	F	P	P	A	A	A	DYSL	P	A	P	P	P	NIL	INFARCT	22	22	367	12.7	AS
60	MALLAPPA	62	17932	M	P	P	P	P	P	NIL	P	A	A	P	P	NIL	INFARCT	11	9	210	8.4	AS
61	ANNAPPA	75	18450	M	A	A	P	P	P	NIL	A	A	A	A	A	NIL	INFARCT	5	3	115	5.5	I
62	MALLAPA	55	21287	M	A	P	A	P	P	NIL	P	A	A	A	A	NIL	INFARCT	13	9	130	4.9	AS
63	SANGANAGOWDA	55	17183	M	P	P	A	A	A	NIL	P	A	P	P	P	NIL	INFARCT	11	8	188	9.7	AS
64	AMBUBAI	70	17265	F	P	P	A	A	P	DYSL	P	A	P	P	P	NIL	INFARCT	10	9	344	11.2	I
65	ANAPPA	78	18/18450	M	A	P	P	A	P	NIL	P	A	P	A	A	HEADACHE	INFARCT	8	7	90	6.2	I
66	VIDYASHREE	26	18/2089	F	A	A	A	A	A	RHD,AF	P	A	P	P	P	HEADACHE	INFARCT	7	6	148	5.8	I
67	RAMARAO	58	18/5134	M	P	P	P	P	A	NIL	P	A	P	A	A	NIL	INFARCT	9	7	136	6.4	I
68	SARASWATHI	68	18/9503	F	A	A	A	A	A	NIL	P	A	P	P	P	NIL	INFARCT	10	10	190	5.6	I
69	SADASHIV	78	18/4469	M	A	A	P	A	A	NIL	P	A	P	A	A	NIL	INFARCT	11	8	186	6.8	AS
70	REKHA	60	18/44125	F	A	P	A	A	A	NIL	P	A	P	P	P	NIL	INFARCT	11	7	154	6.2	AS
71	FULABAI	65	19/7570	F	A	A	A	A	A	NIL	P	A	P	P	P	HEADACHE	INFARCT	5	4	180	5.9	I
72	KAMALABAI	60	18/16234	F	P	P	A	A	A	NIL	P	A	P	P	P	NIL	INFARCT	8	8	220	7.9	I
73	RAMESH	56	19/10464	M	P	P	P	P	A	NIL	P	A	A	A	A	NIL	INFARCT	9	6	190	6.4	I
74	MALLAMMA	60	18/2236	F	P	P	A	A	P	NIL	P	A	A	P	P	NIL	INFARCT	13	9	300	8.6	AS
75	SADASHIVA	56	18/40832	M	P	P	A	A	A	NIL	P	A	P	P	P	NIL	INFARCT	12	12	254	7.7	I
76	CHANDSAB	67	19/7498	M	A	A	P	P	A	NIL	P	A	P	P	P	SEIZURES	INFARCT	12	11	145	6.2	AS
77	SHRISHAIL	60	19/3099	M	A	P	P	A	A	DYSL	P	A	P	A	A	HEADACHE	INFARCT	5	4	155	5.3	I
78	MAHADEVI	65	19/2873	M	A	A	P	A	P	NIL	P	A	P	P	P	HEADACHE	INFARCT	11	10	147	6.1	AS
79	NAGAPPA	68	19/6950	M	P	P	A	A	A	DYSL	P	A	P	P	P	NIL	INFARCT	10	8	150	6.4	I
80	GURAPPA	52	19/3103	M	A	P	P	P	P	NIL	P	A	A	P	P	NIL	INFARCT	11	9	147	5.9	AS
81	SHANKAREPPA	65	18/25501	M	A	P	A	A	A	NIL	P	A	P	A	A	NIL	INFARCT	11	7	132	6.2	I
82	NAMADEV	70	18/24781	M	A	A	A	A	A	NIL	P	A	A	A	A	NIL	INFARCT	12	9	180	6.9	I
83	NAGAPPA	57	18/25249	M	A	P	P	A	A	NIL	P	A	P	A	A	NIL	INFARCT	12	11	122	5.4	AS
84	GIRISH	40	18/3275	M	P	P	P	A	A	NIL	P	A	P	P	P	SEIZURES	INFARCT	13	12	285	8.2	AS
85	NEELAMMA	62	19/650	F	A	A	A	A	A	NIL	P	A	A	P	P	NIL	INFARCT	11	11	133	4.9	I

86	MALLANAGOWDA	70	18/13848	M	A	P	A	P	A	NIL	P	A	P	A	A	NIL	INFARCT	12	9	122	5.6	AS
87	RAMAPPA	75	18/5470	M	A	A	A	A	A	NIL	P	A	P	P	P	NIL	INFARCT	9	8	110	6.4	I
88	SAMBHAJI	66	19/8848	M	A	P	A	A	A	NIL	P	A	P	P	A	NIL	INFARCT	10	7	130	7	I
89	IRAYYA	58	18/13073	M	A	P	P	P	P	NIL	P	A	A	A	A	NIL	INFARCT	12	11	156	6.8	AS
90	CHANDSAB	61	19/7498	M	A	A	A	P	A	NIL	P	A	P	P	P	NIL	INFARCT	13	12	96	5.4	AS
91	SANJEEV	45	18/43064	M	P	P	P	A	P	NIL	P	A	P	A	A	NIL	INFARCT	10	8	170	7.6	AS
92	IRAGANTAPPA	68	18/6818	M	A	P	A	P	A	NIL	P	A	P	P	P	NIL	INFARCT	9	5	122	5.3	I
93	CHANDRAWWA	67	18/9510	M	A	P	A	A	A	NIL	P	A	A	P	P	NIL	INFARCT	15	13	106	5.2	AS