"STUDY OF SERUM PROCALCITONIN LEVELS IN ACUTE ISCHEMIC STROKE IN RELATION WITH ITS SEVERITY AND SHORT TERM OUTCOME"

By

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IN

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ABSTRACT

BACKGROUND:

Worldwide, stroke is a third leading cause of disability and it ranks first in frequency and importance among all the neurological disorders of adult life[1]. In India stroke incidence ranged from 105-152/100000 persons / year for the previous 2 decades. India also has one of the highest case fatality rate of 42 and 46 % in urban and rural population[6-7]. Inflammatory processes have been considered to have fundamental roles in acute ischemic stroke in both the etiology and pathophysiology[16-17]. It is hypothesized that quantitative rise in serum Procalcitonin is proportional to severity of acute ischemic stoke19 pointing towards greater degree of tissue damage thus poor outcome. This study aims to assess the prognostic importance of Serum Procalcitonin as a biomarker for risk stratification and prediction of functional outcome in acute ischemic stroke.

OBJECTIVES:

To correlate serum procalcitonin levels at the time of presentation with severity and short term outcome of acute ischemic stroke.

MATERIALS AND METHODS:

It is a prospective observational study involving 68 patients who presented within 24 hours of onset of acute ischemic stroke. Blood sample of 2mL was taken and sent for serum procalcitonin levels and severity was calculated using NIHSS and short term outcome was calculated at 4th day using Barthel index.

RESULTS:

In our study mean age of subjects being 62.28 ± 12.303 years with predominant males(61.8%). It was observed that PCT was high among the patients who had more severe stroke as seen by NIHSS(p= <0.001) at the time of presentation. i.e more the NIHSS higher the level of PCT. Higher level of PCT was inversely proportional to Barthel index(p= 0.001) on day 4. Hence PCT at higher level at presentation signifies poor short term outcome.

CONCLUSION:

Serum PCT is a good predictor of severity at the time of presentation and poor functional outcome at day 4 of acute ischemic stroke.

ABBREVIATIONS

GLOSSARY	ABBREVIATIONS
NIHSS	➢ NATIONAL INSTITUTES OF HEALTH STROKE SCALE
BI	➢ BARTHEL INDEX
BNP	➢ BRAIN NATRIURETIC PEPTIDE
PCT	➢ PROCALCITONIN
CVA	➢ CEREBROVASCULAR ACCIDENT
TIA	➢ TRANSIENT ISCHEMIC ATTACK
DALY	➢ DISABILITY ADJUSTED LIFE YEARS
TOAST	➢ TRIAL OF ORG10172 IN ACUTE STROKE TREATMENT
OCSP	➢ OXFORDSHIRE COMMUNITY STROKE PROJECT
TACI	➢ TOTAL ANTERIOR CIRCULATION INFARCTS
PACI	➢ PARTIAL ANTERIOR CIRCULATION INFARCTS
POCI	➢ POSTERIOR CIRCULATON INFARCTS
LS	➢ LACUNAR SYNDROMES
ICA	➢ INTERNAL CAROTID ARTERY
ECA	➢ EXTERNAL CAROTID ARTERY
VA	➢ VERTEBRAL ARTERY
ACA	➢ ANTERIOR CEREBRAL ARTERY
MCA	➢ MIDDLE CEREBRAL ARTERY
PCA	➢ POSTERIOR CEREBRAL ARTERY
PICA	➢ POSTERIOR INFERIOR CEREBELLAR ARTERY
MAP	➢ MEAN ARTERIAL PRESSURE
CPR	➢ CARDIOPULMONARY RESUSCITATION
MRI	➢ MAGNETIC RESONANCE IMAGING
CT	➢ COMPUTED TOMOGRAPHY
HTN	➢ HYPERTENSION
DM	➢ DIABETES MELLITUS
CADASIL	➢ CEREBRAL AUTOSOMAL DOMINANT ARTERIOPATHY WITH SUBCORTICAL INFARCTS AND LEUKOENCEPHALOPATHY
CARASIL	➢ CEREBRAL AUTOSOMAL RECESSIVE ARTERIOPATHY WITH SUBCORTICAL INFARCTS AND LEUKOENCEPHALOPATHY
AF	➢ ATRIAL FIBRILLATION
CNS	➢ CENTRAL NERVOUS SYSTEM

UMN	➢	UPPER MOTOR NEURON
LMN	➢	LOWER MOTOR NEURON
MRA	➢	MEGNETIC RESONANCE ANGIOGRAPHY
CTA	➢	COMPUTED TOMOGRAPHY ANGIOGRAPHY
ASPECTS	➢	ALBERTA STROKE PROGRAMME EARLY COMPUTED TOMOGRAPHY SCORE
CBC	➢	COMPLETE BLOOD COUNT
ECG	➢	ELECTROCARDIOGRAPH
HbA1C	➢	GLYCOSYLATED HEMOGLOBIN
2D ECHO	➢	2 DIMENSIONAL ECHOCARDIOGRAPHY
ASD	➢	ATRIAL SEPTAL DEFECT
IV	➢	INTRA VENOUS
DVT	➢	DEEP VEIN THROMBOSIS
BP	➢	BLOOD PRESSURE
SBP	➢	SYSTOLIC BLOOD PRESSURE
DBP	➢	DIASTOLIC BLOOD PRESSURE
rtPA	➢	RECOMBINANT TISSUE PLASMINOGEN ACTIVATOR
mRS	➢	MODIFIED RANKIN SCORE
FDA	➢	FOOD AND DRUG ADMINISTRATION
AHA	➢	AMERICAN HEART ASSOCIATION
ADL	➢	ACTIVITIES OF DAILY LIVING
SAH	➢	SUBARACHNOID HEMORRHAGE
ESC	➢	EUROPEAN CARDIAC SOCIETY
COPD	➢	CHRONIC OBSTRUCTIVE PULMONARY DISEASE
RBS	➢	RANDOM BLOOD SUGAR
FBS	➢	FASTING BLOOD SUGAR
PPBS	➢	POST PRANDIAL BLOOD SUGAR
SC	➢	SERUM CHOLESEROL
LDL	➢	LOW DENSITY LIPOPROTEIN
TG	➢	TRIGLYCERIDE
HDL	➢	HIGH DENSITY LIPOPROTIEN
RR	➢	RESPIRATORY RATE
GCS	➢	GLASGOW COMA SCALE
HB	➢	HEMOGLOBIN
MCV	➢	MEAN CORPUSCULAR VOLUME
PCV	➢	PACKED CELL VOLUME
ROC	➢	RECEIVER OPERATING CHARACETRISTIC

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INTRODUCTION

INRTODUCTION

Worldwide, stroke is a third leading cause of disability and it ranks first in frequency and importance among all the neurological disorders of adult life[1]. According to world health organization (WHO) over 15 million people suffer from stroke each year worldwide among those 5 million die and 5 million land up in permanent disability[2-5]. In India stroke incidence ranged from 105-152/100000 persons / year for the previous 2 decades. India also has one of the highest case fatality rate of 42 and 46 % in urban and rural population[6-7.]

Stroke is one of the leading cause of death. On survivors it leaves behind significant and permanent residual physical, cognitive and psychological disability. This mandates focus towards tools for accurate prediction of outcome following stroke for better and proper allotment of resource towards treatment and rehabilitation. Hence importance is given to assess stroke severity and predict mortality and morbidity which can dictate treatment protocol and decide counselling strategy for family members.

National Institute of Health Stroke Scale (NIHSS) is the traditional and most accepted tool for predicting severity of Ischemic Stroke[8-9]. Many studies conducted recently showed that NIHSS has limited ability in predicting long term outcome following stroke and the lowest value "0" does not rule out stroke[10-11]. Other widely accepted tools for predicting functional outcome of stroke include Barthel Index[12-13] and modified Rankin Scale[14-15]. These two has its own limitation as these can't be used in acute settings and have no value in assessing severity of stroke. This necessitates for a tool which can be used readily and is easy to use for assessing severity and predicting outcome of acute ischemic stroke.

Inflammatory processes have been considered to have fundamental roles in acute ischemic stroke in both the aetiology and pathophysiology[16-17]. This provides a scope for the use of inflammatory markers in predicting the severity and outcome of acute ischemic stroke16. It was found that

early and transient release of Procalcitonin into the circulation was observed after severe trauma[17]. The amount of circulating Procalcitonin seemed to be proportional to the severity of tissue injury and not related to infection, providing proof of inflammatory role of procalcitonin[18]. Thus it is hypothesized that quantitative rise in serum Procalcitonin is proportional to severity of acute ischemic stoke[19] pointing towards greater degree of tissue damage thus poor outcome.

This study aims to assess the prognostic importance of Serum Procalcitonin as a biomarker for risk stratification and prediction of functional outcome in acute ischemic stroke.

OBJECTIVES OF THE STUDY

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1. To record severity of acute ischemic stroke at the time of presentation using NIH Stroke Scale.
2. To record short term outcome at fourth day of presentation using Barthel index.
3. To measure serum procalcitonin levels at the time of presentation.
4. To correlate serum procalcitonin levels with severity and short term outcome.

REVIEW OF LITERATURE

REVIEW OF LITERATURE

History of Stroke:

Stroke at the earliest was described in ancient India as 'Pakshavadha' in aurvedha which is hemiplegia with detailed clinical presentation and treatment[19]. The father of medicine, Hippocrates recognized stroke over 2,400 years ago and he called it 'apoplexy' a Greek word meaning "struck down by violence"[20].

Galen (AD 131 to 201) proposed that apoplexy was caused by anything which interfered with flow of vital 'spirit' to the brain he also inferred that hemiplegia was caused by lesion in opposite side of brain[20]. The next significant work was done by Vesalius in 1543 with publication of his magnum opus ' De humani corporis fabrica' a set of seven books where he describes structure and functions of brain and its coverings rectifying some of the errors of Galen[21].

During 16th century Ambroise Paré discussed about carotids or soporales calling them 'sleepy arteries' because if they are obstructed or stopped we fall asleep.[22] Thomas Willis and Richard Lower during 17th century mentioned about the circle of arteries at the base of the brain.[20,23]

In 1658, The Swiss physician Johann Jacob Wepfer described carotid thrombosis. In his 'Apoplexia' he describes the carotid and vertebral arteries from their origins to the arterial circle at the base of the brain and noted that patients who died with apoplexy had bleeding in the brain[24]. He also describes about examples of completed stroke, progressing stroke, transient ischemic attack and reversible ischemic neurological deficit in his 'Apoplexia'.[20,24,25]

In 1909, in the 7th edition of 'The Principles and Practice of Medicine' William Osler describes apoplectic stroke largely due to cerebral hemorrhage , no mention of extracranial occlusive disease is made and in the section dealing with thrombosis and embolism he emphasizes blocking of intracranial vessels[26].

Thromboembolism as a major risk factor and its mechanism was first described by Rudolf

Virchow in 1856.[27] This was re emphasized by C. Miller Fischer in his 2 articles during 1950s where he found relationship between frequency of disease of the carotid artery in the neck and cerebrovascular insufficiency. He defined the lesion as atherosclerosis and noted partial and complete occlusion and described many syndromes and even suggested a bypass surgery between external carotid and internal carotid above the occlusion[28,29]

There was continued study on cause, symptom and management of apoplexy and in 1927 the apoplexy was divided into categories based on the blood vessel problem and the term ‘ cerebral vascular accident’ (CVA) or stroke was coined and Apoplexy faded from use. Stroke is also referred as a "brain attack" by American stroke association since 1990 to underline its acute nature and the fact that it is caused by a lack of blood supply to the brain as in heart attack, where there is lack of blood flow to heart.

The next major breakthrough occurred in 1995 when the National Institute of Neurological Disorders and Stroke tissue-type Plasminogen activator (NINDS tPA) trial initiated a paradigm shift in treatment of acute ischemic stroke.[30] Two major trials in 1997 International Stroke Trial (IST) and Chinese Acute Stroke Trial (CAST) found that aspirin within 48 hours of stroke onset reduced stroke recurrence and mortality [31,32]. In 2015, 5 randomized trials showed that endovascular thrombectomy within 6 hours of onset was more efficacious than standard medical treatment in acute ischemic stroke[33] and this time period was extended to 24hrs as recent as 2018.[34]

Even with abundant information on the cause, prevention, risk, and management of stroke there is still lacunae in knowledge to prognosticate stroke.

DEFINITION

STROKE

It is defined as " an abrupt onset of neurologic deficit that is attributable to a focal vascular cause" . By definition stroke is purely a clinical diagnosis and Laboratory studies which include imaging are used as support for the diagnosis[35].

TRANSIENT ISCHEMIC ATTACK (TIA)

It is defined as acute onset of focal neurological deficit which shows full recovery in 24 hours without evidence of brain infarction on imaging [35]. TIAs usually last less than 1 and if TIA lats for longer evidence will be picked up in neuro-imaging as acute infarct.

In many recent studies it was found that 24 hours was too arbitrary as 50% of patient who were diagnosed as TIA initially showed focal lesion on MRI so American stroke association define TIA as "a brief episode of neurological dysfunction caused by focal brain or retinal ischemia, with clinical symptoms typically lasting less than one hour, and without evidence of acute infarction"[36] TIA serve as a warning signal for upcoming major stroke and the risk stratification is done via ABCD2 (Age, Blood pressure, clinical feature, duration of TIA and Diabetes status) score.[37]

EPIDEMIOLOGY

INCIDENCE AND PREVALANCE

Incidence best reflects the burden of stroke on community. One in every four individual over 25 years are at risk of developing stroke , global lifetime risk of stroke above 25 years was around 25% among both men and women in 2016 and the risk was highest in east Asia and central Europe. The risk of developing ischemic stroke is 18.3% and hemorrhagic stroke is 8.2% .[38]

There is 100% increase in stroke incidence from 1970-79 to 2000-08 in low and middle income

countries including India.[39] According to American heart association 4% of all American adults will have had stroke by 2030.[40] In 2016 globally there were 80·1 million prevalent stroke cases, 41·1 million were women and 39·0 million were men and 84·4% of these strokes were ischemic. In 2016 there were around 13·7 million new stroke cases[2].

In different parts of India the crude stroke prevalence ranged from 44.29 to 559/100,000 persons and the cumulative incidence ranged from 105 to 152/100,000 persons/year during the last 2 decades these estimates were much higher than those of high income countries which was reported to be 94/100,000 person-years during 2000-2008.[5]

MORBIDITY AND MORTALITY

Stroke is the most common life threatening neurological disease. It is the second leading cause of death[1,2] and disability[2,3] worldwide. An estimated 6.17 million people died in 2017 due to stroke[1] and 5 million remained permanently disabled, Over 75 % of death and > 85% of disability adjusted life years were in low and middle income countries[1–4].

In 2016, around 5 million were affected by stroke among which 2.7 million people died due to ischemic stroke which was slightly lesser compared to 2.8 million deaths due to hemorrhagic stroke. There was an increase of Disability Adjusted Life Years (DALYs) from 95.3 million in 1990 to 116.4 million due to stroke, it is the second most common cause of DALYs worldwide. DALYs were lower in women (50·8 million) compared to men (65·6 million). DALYs due to ischemic stroke (51·9 million) was lower than hemorrhagic stroke (64·5 million).[2]

There is lack of comprehensive data on morbidity and mortality of stroke in India. A systematic review of epidemiological studies of stroke in India found case fatality rate of 42 and 46 % in urban and rural population which is one of the highest in the world.[5]

CLASSIFICATION OF STROKE

Strokes are broadly classified into

1. Arterial stroke
2. Venous stroke

ARTERIAL STROKE

They are further classified into

1. Ischemic stroke (85%)
2. Hemorrhagic (15%)

ISCHEMIC STROKE

It is further subdivided into 2 types

1. Thrombotic stroke
    1. Large vessel Thrombosis
    2. Lacunar (small vessel thrombosis)
2. Embolic stroke
    1. Cardiogenic embolisms
    2. Artery to artery embolism

Embolic strokes are the most common types of strokes.

HEMORRHAGIC STROKE

They can be further classified into

1. Intracerebral hemorrhage
2. Sub arachnoid hemorrhage

The distinction between Ischemic stroke and Hemorrhagic stroke is crucial to decide the treatment modality. Early and appropriate use of thrombolytic therapy is indicated in ischemic stroke but absolutely contraindicated in hemorrhagic stroke.

VENOUS STROKE

It is a rare type of stroke which affects about 5 million people and accounts 0.5% of all strokes and is caused by Dural venous sinuses (like superior sagittal, Lateral and cavernous sinus) obstruction by thrombosis.[41]

TOAST CLASSIFICATION[42]

It is a system of classification of ischemic strokes based on etiology developed for the Trial of Org 10172 in Acute Stroke Treatment.

1. Large-artery atherosclerosis
2. Cardioembolism
3. Small-vessel occlusion
4. Stroke of other determined etiology
5. Stroke of undetermined etiology (2 or more causes identified, Negative evaluation, Incomplete evaluation)

This system of classification is widely used by clinicians and researchers as it is easy to use with good interobserver agreement and helps in predicting outcome but does not help in making treatment decisions.

OXFORDSHIRE COMMUNITY STROKE PROJECT (OCSP) CLASSIFICATION[43]

It is a simple, easy clinical classification method used in acute ischemic stroke which predicts the site and size of the infarct on imaging. Some trials have used this classification to predict clinical outcome[44,45].

1. Total Anterior Circulation Infarcts (TACI)

   It implies a large cortical stroke in middle or middle and anterior cerebral artery territories.

   Characterised by

   1. New higher cerebral dysfunction
   2. Homonymous visual field defect
   3. A contra lateral motor and or sensory deficit involving at least 2 out of 3 areas of face , arm or leg.

2) Partial Anterior Circulation Infarcts (PACI)

   It implies a cortical stroke in middle or anterior cerebral artery territory.

   Characterized by one of the following

   a) They are characterized by any two of 3 component of TACI

1. New higher cerebral dysfunction
2. Homonymous visual field defect
3. A contra lateral motor and or sensory deficit involving at least 2 out of 3 areas of face , arm or leg

   b) New higher cerebral dysfunction alone

   c) A motor/sensory deficit more restricted than that of TACI

3) Posterior Circulation Infarct (POCI)

Characterized by one of the following

a) Ipsilateral cranial nerve palsy with contralateral motor and or sensory deficit

b) Bilateral motor or sensory deficit

c) Disorder of conjugate eye movement

d) Cerebellar dysfunction without ipsilateral tract involvement

e) Isolated homonymous visual field defects or cortical blindness.

4) Lacunar syndromes

It implies a subcortical stroke due to small vessel disease. Evidence of higher cortical dysfunction or disturbance of consciousness excludes it. It is characterised by

1. Pure motor stroke
2. Pure sensory stroke
3. Ataxic hemiparesis
4. Clumpsy hand – dysarthria syndrome.

PATHOBIOLOGY

Understanding vascular anatomy and its relationships to functional neuroanatomy provide important clues for identifying the cause of symptoms and signs and help guide treatment.

AORTIC ARCH

Paired carotid and vertebral arteries supply the brain. The right subclavian and common carotid artery originates from the brachiocephalic trunk. Right subclavian artery gives rise to right vertebral artery at its proximal part. The left common carotid artery originates directly from the aortic arch. The left subclavian artery arises from the aortic arch distal to the left common carotid artery and also supplies the left vertebral artery[46–48].

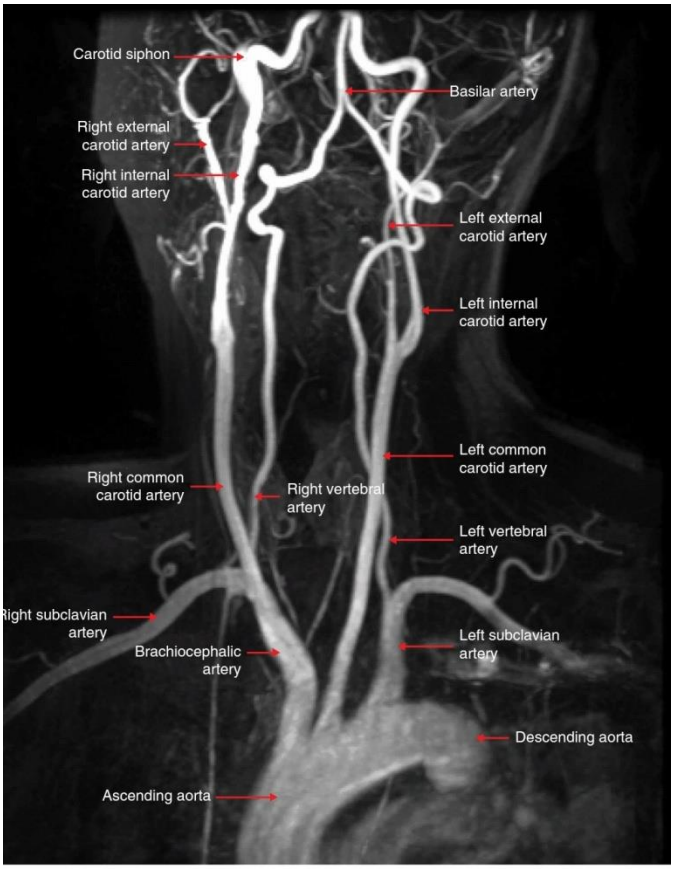


FIGURE 1 : MAGNETIC RESONANCE ANGIOGRAM OF AORTIC ARCH

Source – Harrison's principles of internal medicine 20th edition

INTERNAL CAROTID ARTERIES (ICA)

At the level of superior border of thyroid cartilage corresponding to C3 or C4 vertebrae, common carotid arteries divide into internal and external carotid artery.

The ICA enters the skull through the carotid canal and passes through the Petrous bone near the inner ear, It then passes above the foramen Lacerum to enter the cavernous sinus and ascends in an S shape before penetrating the Dura and finally dividing into the anterior cerebral artery and middle cerebral artery. Opthalmic artery usually arise from the ICA distal to cavernous sinus[46–48].

EXTERNAL CAROTID ARTERIES

The braches of ECA superficial temporal arteries and facial arteries can anastomose with the intracranial circulation through ophthalmic artery branches and it gains significance in proximal internal carotid artery occlusion[46–48].

VERTEBRAL ARTERIES

Vertebral arteries usually arise from the subclavian arteries. They commonly enter transverse process at C6 (V1 segment) and exit at C1 (V2 segment). They turn posteriorly behind the atlantoaxial joint and at the foramen magnum they pass through the dura(V3 segment). Typically at the pontomedullary junction the vertebral arteries join to form a single basilar artery intracanially (V4 segment). Medial branches of vertebral arteries unite to form the anterior spinal artery and lateral branches of vertebral artery supply the dorsolateral medulla, inferior portion of the cerebellum, and vestibular nuclei. Medullary pyramid, inferior olivary nucleus, medial lemniscus, and hypoglossal nerve fibers are supplied by the medial branches. spinothalamic tracts , sympathetic fibers, the sensory nuclei and descending tracts from 5th cranial nerve, fibers from the 9th and 10th cranial nerves are supplied by longer circumferential branches of vertebral and posterior cerebral arteries when they traverse the medulla[44–46].

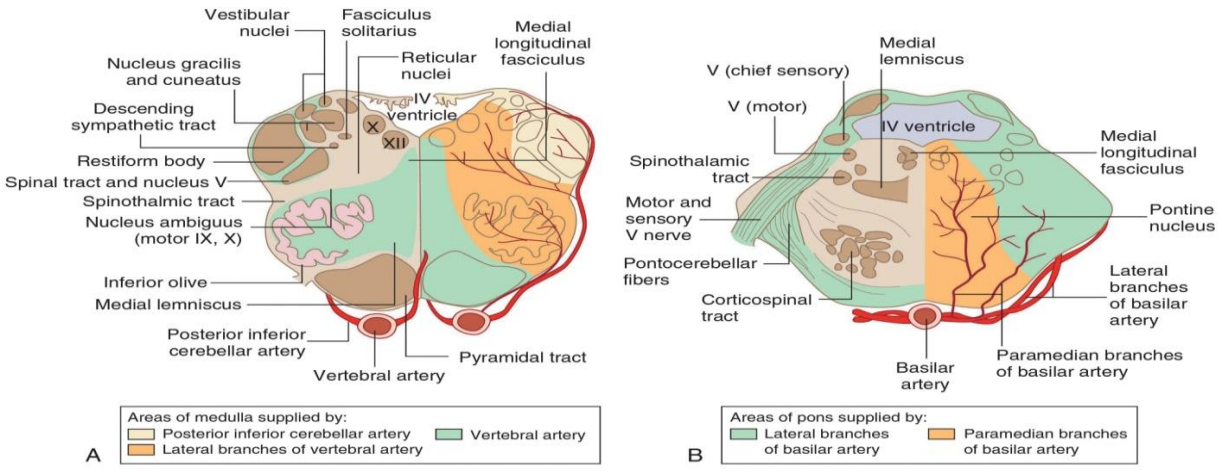


FIGURE 2 : BRAIN STEM BLOOD SUPPLY
CROSS SECTION OF A : MEDULLA OBLONGATA B: MID PONS REGION
Source – Harrison's principles of internal medicine 20th edition

BASILLAR ARTERY

Dorsal portions of the Pons and midbrain are supplied by small penetrating branches which originate from basilar artery. Mid–basilar artery gives rise to the anterior inferior cerebellar arteries which supply portions of the cerebellar hemispheres , lateral pons; 5^{th} ,7^{th} and 8^{th} cranial nerves and pontine portions of the spinothalamic tracts and sympathetic fibers. the distal basilar artery gives rise to two superior cerebellar arteries at the level of the midbrain which supply dorsal midbrain, including the colliculi and the superior portions of the cerebellar hemispheres and vermis. Paramedian vessels which supply the middle portion of the basis pontis and midline pontine structures, including the corticospinal tracts, medial longitudinal fasciculus, and pontine reticular nuclei arise from basillar artery. Paramedian branches also supply the cerebral peduncles, 3^{rd} cranial nerve and its fibres, and medial portions of the red

nucleus and medial lemniscus. ventrolateral pons and midbrain are supplied by short circumferential branches of basilar artery[44–46].

CIRCLE OF WILLIS

Anterior communicating artery connects the two anterior cerebral arteries. Internal carotid artery is connected with the proximal posterior cerebral artery by the posterior communicating artery. A single patent internal carotid artery or vertebral artery can supply the entire intra cranial circulation but most of individuals have an incomplete circle of willis. Sometimes a single intracranial artery can supply both anterior cerebral arteries and sometimes carotid supply the posterior cerebral artery instead of vertebrobasilar arteries[46–48].

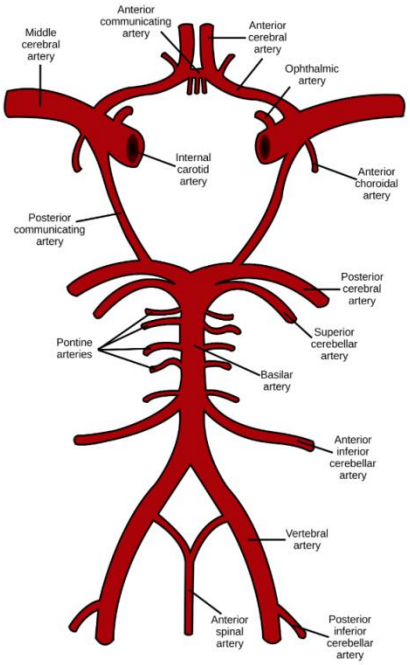


FIGURE 3 : CIRCLE OF WILLIS AND BRANCHES

ANTERIOR CEREBRAL ARTERIES (ACA)

They supply the medial portions of the frontal and parietal lobes. Anterior cerebral artery divides into pericallosal and callosal marginal branches in 50% of individuals. Terminal portion of callosal marginal branch supply the medial cortex between the parietal and occipital lobes. ACA gives rise to series of lenticulostriate branches. One of the most important branch of ACA is the recurrent artery of Heubner a medial striate artery that supply anterior and inferior portions of the anterior limb of the internal capsule and caudate nucleus, anterior globus pallidus, putamen, hypothalamus, olfactory bulbs and tracts, and uncinate fasciculus[46–48].

ANTERIOR CHOROIDAL ARTERY

It originates from from the supraclinoid internal carotid artery and enters the brain at the choroidal fissure. It supplies the optic tract, anterior hippocampus, amygdala, tail of the caudate nucleus, geniculate body, and inferior portion of the posterior limb of the internal capsule[44–46].

MIDDLE CEREBRAL ARTERY (MCA)

It supplies the major portion of the frontal, parietal, and lateral portions of the temporal lobes. MCA at sylvian fissure bifurcates in 20 to 30% and in 70% individuals trifurcates . Frontal and parietal lobes are supplied by superior division and lateral portion of the temporal lobe is supplied by inferior division. lateral lenticulostriate and few medial lenticulostriate arteries originate from M1 segment of MCA which is the portion between its origin and distal branches. These striate arteries supply head and body of the caudate nucleus, the putamen, and the globus pallidus , anterior limb of genu, and the superior portions of the posterior limb of the internal capsule[46–48].

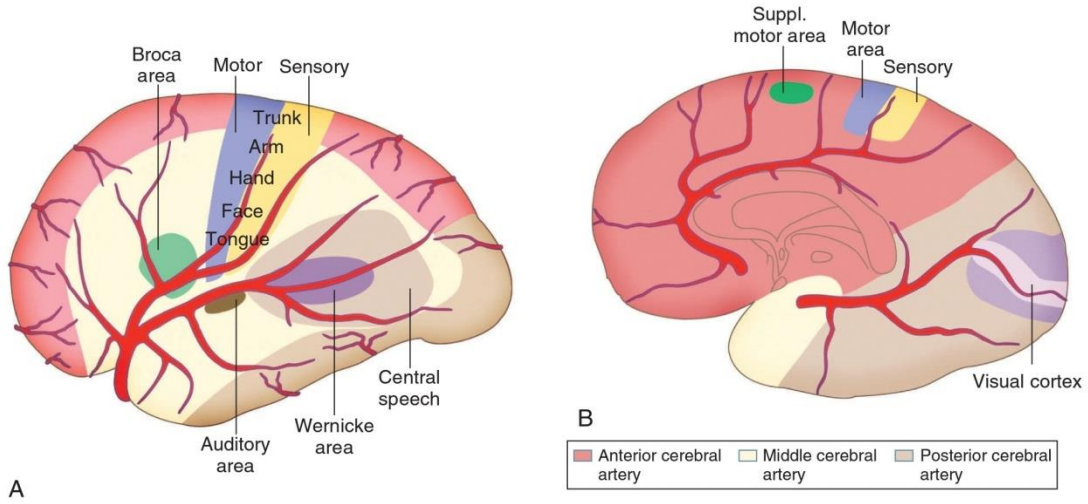


FIGURE 4 : CEREBRAL ARTERY ANATOMY : DISIBUTION OF ANTERIOR , MEDIAL AND POSTERIOR CEREBRAL ARTERY . A : LATERAL AND **B** MEDIAL CEEBRAL HEMISPHERE

POSTERIOR CEREBRAL ARTERY (PCA)

PCA divides into anterior and posterior divison. Inferior and medial portions of the temporal lobe is supplied by the anterior division whereas the occipital lobe and calcarine cortex is supplied by the posterior division. The terminal branches of anterior division anastomose with MCA and posterior division anastomose with MCA and ACA. Thalamus is supplied by small

penetrating arteries branches which originate from the proximal portions of both the posterior cerebral artery and the posterior communicating artery .In few; both the thalami are supplied by a single common artery of Percheron originating from PCA. Choroid plexus, posterior thalamus, fornix, and midbrain tectum are supplied by the two posterior choroidal arteries which originate separately from the PCA. The medial portions of the cerebral peduncles, substantia nigra, red nuclei, hippocampus, and posterior hypothalamus are supplied by PCA perforators[44–46].

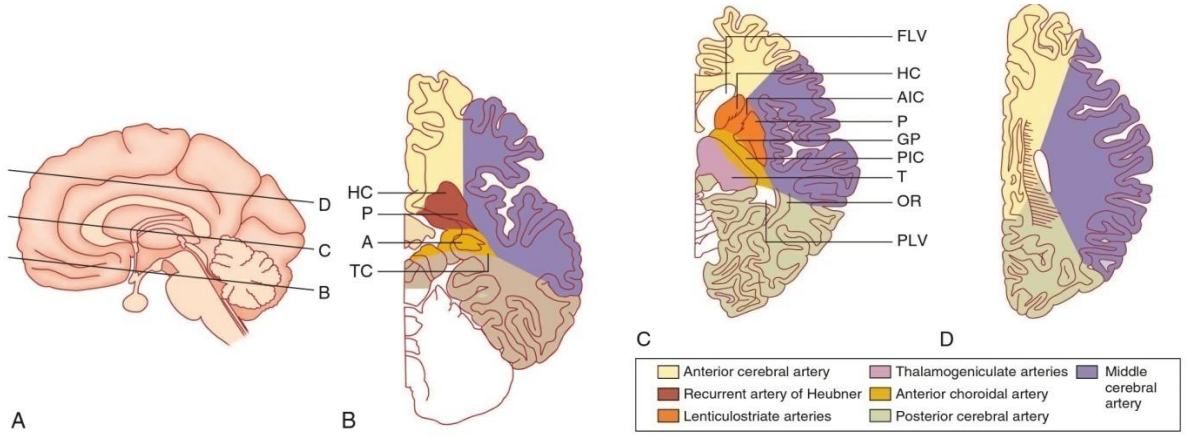


FIGURE 5 : ARTERIAL SUPPLY OF DEEP BRAIN STRUCTURES

A : SAGITTAL VIEW OF BRAIN WITH PLANES PASSING THROUGH VIEW B , C AND D ARE TAKEN.

(HC- head of the caudate nucleus, P- putamen, A- amygdala, TC- tail of the caudate nucleus,

FLV- frontal horn of the lateral ventricle, AIC , PIC -anterior and posterior limbs of the internal capsule, GP- globus pallidus, T- thalamus, OR- optic radiations, and PLV- posterior horn of the lateral ventricle)

PHYSIOLOGY

Human brain constitutes only 2% of body weight but consumes 20% of oxygen and receives 14% of cardiac output. Glucose is the main substrate of brain. At rest brain tissue requires 140 μmol of oxygen and 24 μmol of glucose per 100 g of tissue per minute for metabolism. 80% of glucose is used to generate energy and the rest is metabolized to lactate or used for synthetic activities, no glucose is stored in brain[47].

Brain is the most metabolically active tissue of body and very vulnerable to reduction in oxygen or blood supply. Normal cerebral flow range from 50-100 ml/100gram/minute.if there is a decrease in this flow normal brain function is reduced and results in neural injury.

Cerebral blood flow remains constant normally and is regulated by various autoregulatory mechanisms. When the mean arterial pressure (MAP) decreases, there is dilation of cerebral arterioles leading to a compensatory decrease in cerebrovascular resistance to maintain a constant cerebral blood flow. If MAP increases, constriction of cerebral arterioles occurs and there is a compensatory increase in cerebrovascular resistance manintaing the cerebral blood flow. At MAP > 150 mm Hg, cerebral arterioles are maximally constricted and cerebral blood

flow rises and MAP < 50 mm Hg, cerebral arterioles are maximally dilated resulting in decreased cerebral blood flow(47). Cerebral blood flow is also affected by metabolic factors like hypercapnia which causes cerebral vasodilation and hypocapnia which results in cerebral vasoconstriction. There is a decrease in cerebral blood flow by 2% for every 1 mmHg decline in PCo2. In patients with increased intracranial pressure and threatened herniation,

a short period of hyperventilation can be used as a temporary measure until more definitive treatment can be instituted[47].

PATHOPHISIOLOGY OF ISCHEMIC STROKE

Brain is one of the most metabolically active tissue in body and its functioning is completely dependent on blood and oxygen supply. Clinical symptoms occur when blood flow falls < 50ml/100gm/min. If cerebral blood flow decreases to 0 it causes brain death within 4-10 minutes, if blood flow falls <16-18 ml/100g/min infarction occurs within 1 hour and <20ml/100g/min results in ischemia without infarction unless prolonged for hours or days.[49]

Hypoxic ischemic injury can be focal, diffuse or global. Regions of the hippocampus, cerebellar

Purkinje cells, and neocortical layers III, V are highly metabolic and vulnerable to hypoxia and ischemia.

GLOBAL ISCHEMIC INJURY

It occurs in complete cardiovascular collapse as in ventricular fibrillation, asystole, electromechanical disassociation, hypotension and cardiac arrest . In hypotension watershed areas between the arteries like frontal cortex and adjacent subcortical white matter between ACA and MCA, the parieto-occipital cortex and adjacent subcortical white matter between MCA and PCA, and deep hemispheric white matter and centrum semiovale between MCA and lenticulostriate arteries are vulnerable to injury. The duration of cardiac arrest, anoxia, its cause and duration of CPR are related to prognosis after CPR. It might lead to vegetative state when cerebral cortex is irreversibly damaged but the resistant brain stem which controls respiration and cardiovascular regulation is functioning[47].

DIFFUSE HYPOXIC INJURY

It is caused by high altitudes, severe anemia, and pulmonary disease. Symptoms like altered cognition, confusion, impair consciousness, coma can occur which can be irreversible and

present when the Pao2 abruptly falls < 40 mm Hg[47].

FOCAL ISCHEMIC INJURY

It is caused by occlusion of artery supplying the brain. Although It can occur from infection, inflammation, metabolic disorders, trauma, and hematologic disorders, the majority of strokes are due to thrombotic or embolic occlusion. If blood flow is restored before significant infarction develops patient may experience only transient symptoms resulting in transient ischemic attack

(TIA). Ischemic penumbra is the ischemic but reversible dysfunctional area surrounding infarction which can be seen by perfusion imaging by MRI or CT scan, if there is no change in flow the penumbra will progress to infarction therefore saving the ischemic penumbra is the main goal of revascularisation treatment[49].

Permanent occlusion of a cerebral artery results in necrosis of its supplied neurons, glia, and endothelial cells.

Focal ischemia occurs by 2 pathways

1. Necrotic pathway - energy failure of the cell leads to cellular cytoskeletal breakdown
2. Apoptotic pathway - cells become programmed to die

Ischemia causes necrosis of neurons due to lack of glucose and oxygen, which results in mitochondrial failure. Failure to produce ATP causes membrane ion pumps to stop functioning and neurons depolarize which raises intracellular calcium level. Cellular depolarization also leads to release of glutamate from synaptic terminals; excess extracellular glutamate activates postsynaptic glutamate receptors which increases neuronal calcium influx that results in neurotoxicity. Mitochondrial dysfunction and degradation of membrane lipids produce free radicals which cause catalytic destruction of membranes[47,49–51].

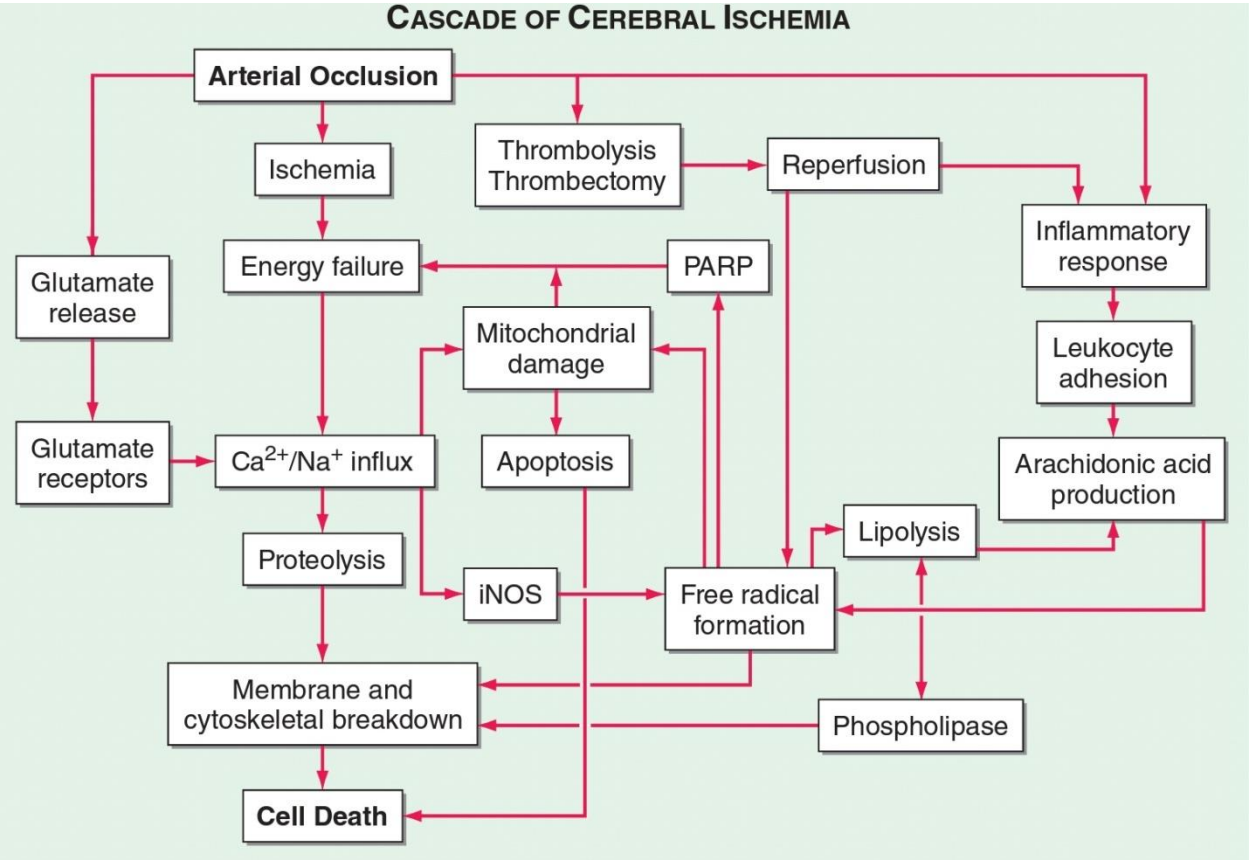


FIGURE 6 : CASCADE OF CEREBRAL ISCHEMIA (iNOS, inducible nitric oxide synthase; PARP, poly-A ribose polymerase.)

Source – Harrison's principles of internal medicine 20th edition

RISK FACTORS FOR ISCHEMIC STROKE

Modifiable and non modifiable factors increase the risk of stroke.[52]

NON MODIFIABLE FACTOR

1. Age
2. Gender
3. Race/ethinicity
4. Family history
5. Genetics

MODIFIABLE RISK FACTORS

1. Arterial hypertension
2. Obesity
3. Cigarette smoking
4. Diabetes mellitus
5. Transient ischemic attacks
6. Physical inactivity
7. Dyslipidemia
8. Asymptomatic carotid bruit/stenosis
9. Cardiac disease
10. Aortic arch atheromatosis
11. Alcohol consumption
12. Increased fibrinogen
13. Elevated homocystine
14. Low serum folate
15. Elevated anticardiolipin antibodies
16. Oral contraceptive use

The INTERSTROKE study, a international case control study in 22 countries found that the 5 risk factors hypertension, current smoking, abdominal obesity, diet, and physical activity accounted for more than 80% global risk of all stroke[53].

AGE

It is the strongest risk factor for stroke. Stroke is a disease of elderly, after the age 55 the incidence of stroke almost doubles for each decade (54). According to global burden of disease study risk of developing of stroke is 25% for individuals more than 25 years , there is a decrease in lifetime risk of stroke among adults after age of 70 reaching incidence of 13.4% among 95 years of age[38].

SEX

The association of sex to stroke risk is dependent on age. Women have the same or high risk as of men of developing stroke at young age but the relative risk is slightly higher for men at older age. Pregnancy, post-partum state and hormonal factors like contraceptives use are the likely cause for higher risk of stroke among women at young age[55]. In 2016 out of 80·1 million stroke cases, 41·1 million were women and 39·0 million were men(2) may be because of the longer life span of women compared to men[54].

RACE/ETHINICITY

The relationship between stroke risk and race is well documented. According to REGARDS study compared to Caucasians, African Americans have a two times high risk of developing stroke may be due to high prevalence of other risk factors like hypertension, Diabetes and obesity in them[56]. According to global burden of disease study risk of developing stroke is highest in east Asia and central Europe[38].

FAMILY HISTORY

Parental and family history of stroke increases the propensity for stroke. There is a 3 fold increase of stroke in offspring of parents who had a documented stroke by 65 years even after adjusting for other risk factors.[57] Other causes of stroke like vascular anomalies, connective tissue disorders, familial hypercholesterolemia which run in the family increases of stroke in offspring.

GENETICS

There is increased stroke risk with expression of genetic variability, there are many mechanisms.[55]

1. A Single gene disorders that primarily cause stroke

1. Cerebral Autosomal Dominant Arteriopathy with Subcortical Infarcts and Leukoencephalopathy (CADASIL) is a autosomal dominant disorder involving NOTCH3 gene and protein they present with ischemic stroke, leukoencephalopathy, migraine, psychiatric manifestations, dementia.
2. Cerebral Autosomal Recessive Arteriopathy with Subcortical Infarcts and Leukoencephalopathy (CARASIL) is a autosomal recessive disorder involving *HTRA1 gene which codes for* HtrA serine peptidase-1 protein which presents with Ischemic stroke, leukoencephalopathy, premature baldness, spondylosis.
3. Familial amyloid angiopathy and Collagen 4 (*COL4A1*) mutations are autosomal dominant disorders which cause rupture of cortical vessels causing hemorrhages .

2) Genetic Disorders that include stroke as manifestation

A single gene disorder can cause multisystem disorder where stroke is a manifestation

1. Ehlers Danlos type 4 is a autosomal dominant disorder can lead to arterial dissection

2. Smooth Muscle Alpha-Actin (ACTA2) mutation associated disorders and Marfan syndrome are autosomal dominant disorders involving ACTA2 and fibrillin1 gene which result in ischemic stroke
3. Fabry disease is a X linked disorder involving α-galactosidase Aprotein which can cause ischemic stroke.
4. Mitochondrial encephalopathy with lactic acid and stroke like episodes (MELAS) is a mitochondrial disorder which cause energy failure leading to metabolic stroke , here ischemia does not follow vascular boundaries.
5. Sickle cell anemia is a autosomal recessive disorder which can cause ischemic stroke along with other manifestations.

3) Few common variants of genetic polymorphisms like TSPAN2 , FOXF2 have been associated with stroke risk
4) Genetic causes for other risk factors like hypertension, diabetes mellitus, Atrial fibrillation are also associated with stroke risk.

MODIFIABLE RISK FACTORS

They are very important as early identification and modification of these factors help in preventing stroke.

HYPERTENSION

It is the major risk factor for stroke and the relationship between blood pressure and stroke is well documented.[58] 54% of strokes were attributable to hypertension in the INTERSTROKE study.[53] Hemorrhagic stroke had higher blood pressure compared to ischemic stroke. Hypertension can lead to stroke by various mechanisms. An increased intraluminal pressure will cause altered function of endothelium and smooth muscle in cerebral arteries which increase permeability over the blood-brain barrier and lead to brain oedema; these can also cause focal

thrombi formation and ischaemic lesions. fibrinoid necrosis can lead to local stenosis and blockage causing lacunar infarcts. Intracerebral haemorrhages are caused by degenerated smooth muscle cells and endothelium. Hypertension also increases the arteriosclerotic process leading to stroke.[59]

DIABETES MELLITUS

Diabetics have greater susceptibility to coronary, femoral and cerebral artery atherosclerosis. It is a independent risk factor for stroke with 2 times increased risk and accounting to 20% of stroke mortality[55,60]. Duration of diabetes and also prediabetes increases the risk of stroke the risk of stroke increases by 3% with each year of diabetes and becomes 3 times with > 10 years of diabetes mellitus[61].

DYSLIPIDEMIA

The relationship between blood lipids and stroke is complex and much lower compared to coronary artery disease. Elevated total cholesterol has increased risk where as elevated HDL cholesterol has decreased risk for stroke[62,63]. There is an inverse relationship between hemorrhagic stroke and total cholesterol. There is a strong association between large artery ischemic stroke and total cholesterol compared to other stroke subtypes[63,64].

OBESITY / SEDENTARY LIFE / DIET

Sedentary life is associated with many disorders including stroke. physical activity reduces risk of diabetes mellitus, hypertension and obesity there by reducing the risk of stroke[55].

There is significant relationship between diet and stroke, diet also increases risk of other conventional risk factors like diabetes, hypertension, dyslipidemia and obesity[65]. Salt rich diet increases the risk of hypertension and stroke[66] and whereas low potassium diet decreases the risk of stroke[67]. Mediterranean diet, dietary approach to stop hypertension (DASH) diet or diet with

increased fruit intake are found to decrease risk of stroke by almost 20%[68].

Obesity increases the risk factors of hypertension and diabetes. Obesity also increase the risk of stroke, abdominal obesity , waist hip ratio is stronger risk factor than body mass index for developing ischemic stroke[53,69].

Metabolic syndrome is a collection of risk factors hypertension, obesity, dyslipidemia and pre diabetes. Risk of stroke doubles in patients with metabolic syndrome.

ALCOHOL CONSUMPTION / SMOKING AND SUBSTANCE ABUSE

Alcohol consumption and stroke risk have a J shaped relation with 2 -3 drinks per day being protective and heavy drinking increasing the risk of stroke[55,70,71]. Alcohol increases the risk of hypertension , dyslipedemea there by increasing risk of stroke. Alcohol has a linear association with hemorrhagic stroke even a small amount of alcohol increases risk of bleed.

Cigarette smoking is a major risk factor for stroke, increasing the risk 2 fold and contributes 15% of all deaths due to stroke[72]. Passive smoking is also associated with increased risk of stroke[73]. Cocaine, heroin, amphetamines, and ecstasy abuse increases the risk of ischemic and hemorrhagic strokes[74,75].

CARDIAC DISORDERS

Cardiac diseases are a major risk factor for stroke. Heart failure increases the risk of stroke by 2-3 times. 10-24% of stroke patients had heart failure and 9 % of stroke was caused by heart failure[76,77] heart failure is often associated with other risk factors like diabetes, hypertension, dyslipidemia and obesity.

Atrial fibrillation(AF) is one of the important risk factor for stroke, the incidence of stroke due to AF has tripled in past 3 decades[78] one of the proposed mechanism for AF causing stroke is formation of thrombus in atrium resulting in embolisation to cerebral vessels but this model is

incomplete as the association between AF and stroke is not convincingly proven[79]. The possible mechanism is thromboembolism caused by AF and abnormal systemic and atrial tissue substrate or 'atrial cardiopathy[79]'. There is also evidence that stroke per se causes AF by changes in autonomic tone and post stroke inflammatory state[80,81]. Supra ventricular tachycardia also predisposes to stroke. The risk of stroke in patient with AF can be calculated by CHA2DS2-VASc score.[82]

Coronary artery disease and stroke have similar risk factors. Coronary artery disease almost tripled the risk of stroke[83] independent of other risk factors. Valvular heart disease like mitral stenosis and mitral regurgitation predispose for development of stroke[84]. In patients with left ventricular hypertrophy risk of atherothrombotic stroke was almost 3-4 times[85]. Carotid artery bruit and stenosis predisposes to develop stroke. Congenital heart disease like cyanotic heart disease, atrial septal defect patent foramen ovale increases the risk of cardioembolic stroke[52].

TRANSIENT ISCHEMIC ATTACK (TIA)/ PREVIOUS STROKE

Individual who had previous stroke or a TIA is having a high risk of recurrent stroke, risk following TIA is calculated by ABCD score[37].

HOMOCYSTEINAEMIA

Patients with rare inherited deficiency of cystathionine syntase develop severe homocysteinaemia and homocystinuria and a tendency to develop venous and arterial thrombosis.

ETIOLOGY OF ISCHEMIC STROKE

The clinical features and examination findings often establish the cause of stroke and judicious use of laboratory testing and imaging studies completes the initial evaluation 30% of strokes can remain unexplained despite extensive evaluation[49].

COMMON CAUSES

1. Thrombosis
    1. Lacunar stroke (small vessel)
    2. Large-vessel thrombosis
    3. Dehydration
2. Embolic occlusion
    1. Artery-to-artery
        1. Carotid bifurcation
        2. Aortic arch
        3. Arterial dissection
    2. Cardioembolic
        1. Atrial fibrillation
        2. Mural thrombus
        3. Myocardial infarction
        4. Dilated cardiomyopathy
        5. Valvular lesions
        6. Mitral stenosis

7. Mechanical valve
8. Bacterial endocarditis

3. Paradoxical embolus
    1. Atrial septal defect
    2. Patent foramen ovale

3. Atrial septal aneurysm
4. Spontaneous echo contrast
5. Stimulant drugs: cocaine, amphetamine

UNCOMMON CAUSES

1. Hypercoagulable disorders
    1. Protein C deficiency
    2. Protein S deficiency
    3. Antithrombin III deficiency
    4. Antiphospholipid syndrome
    5. Factor V Leiden mutation
    6. Prothrombin G20210 mutation
    7. Systemic malignancy
    8. Sickle cell anemia
    9. β Thalassemia
    10. Polycythemia vera
    11. Systemic lupus erythematosus

12. Homocysteinemia
13. Thrombotic thrombocytopenic
14. purpura
15. Disseminated intravascular
16. coagulation
17. Dysproteinemias
18. Nephrotic syndrome
19. Inflammatory bowel disease
20. Oral contraceptives

2) Venous sinus thrombosis
3) Fibromuscular dysplasia
4) Vasculitis
    1. Systemic vasculitis (PAN, granulomatosis with polyangiitis [Wegener's], Takayasu's, giant cell arteritis)
    2. Primary CNS vasculitis
    3. Meningitis (syphilis, tuberculosis, fungal, bacterial, zoster)
5) Noninflammatory vasculopathy
    1. Reversible vasoconstriction syndrome
    2. Fabry's disease
    3. Angiocentric lymphoma
6) Cardiogenic
    1. Mitral valve calcification

2. Atrial myxoma
3. Intracardiac tumor
4. Marantic endocarditis
5. Libman-Sacks endocarditis

7) Subarachnoid hemorrhage vasospasm
8) Moyamoya disease
9) Eclampsia

There are 3 major mechanism that cause ischemic stroke

1. Occlusion of an intracranial vessel by an embolus that arises at a distant site (e.g., cardiogenic sources such as atrial fibrillation or artery-to-artery emboli from carotid atherosclerotic plaque), often affecting the large intracranial vessels;
2. In situ thrombosis of an intracranial vessel, typically affecting the small penetrating arteries that arise from the major intracranial arteries;
3. Hypoperfusion caused by flow-limiting stenosis of a major extracranial (e.g., internal carotid) or intracranial vessel, often producing “watershed” ischemia.

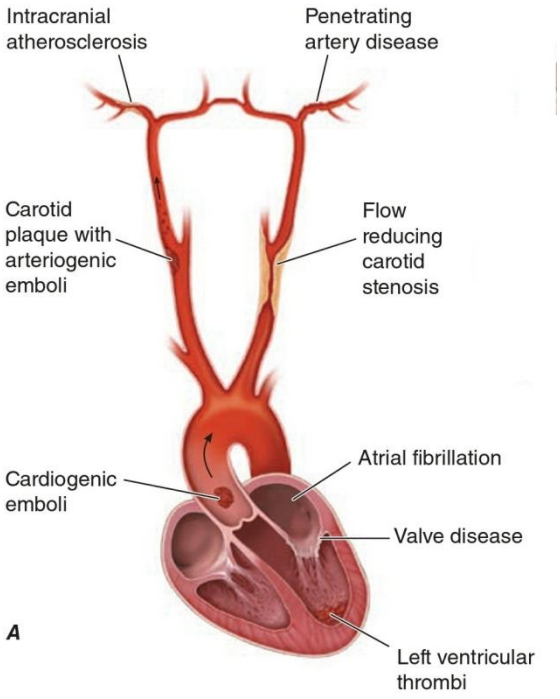


FIGURE 7 : PATHOPHYSIOLOGY OF ISCHEMIC STROKE

Source – Harrison's principles of internal medicine 20th edition

CARDIOEMBOLIC STROKE

It is responsible for 20% of all strokes, cardiac disease cause stroke mainly due to embolism of thrombotic material which forms on the atrial or ventricular wall or the left heart valves. They are sudden in onset with maximum neurological deficit at the time of onset which improvement in deficit. The most important causes for cardioembolic stroke are non valvular atrial fibrillation, myocardial infarction, prosthetic valves, rheumatic heart disease and ischemic cardiomyopathy. The risk of stroke in patient with AF can be calculated by CHA2DS2-VASc score[82]. When anteroapical ventricular wall is involved Myocardial infarction can cause transmural embolisation leading to stroke.

Paradoxical embolisation occurs when there is right to left shunt due to patent foramen ovale or atrial septal defect. Venous clot, fat and tumor emboli, bacterial endocarditis, IV air, and amniotic fluid emboli can migrate to arterilal circulation causing ischemic stroke. Bacterial

endocarditis lead to septic emboli which can cause stroke.

ARTERY-TO-ARTERY EMBOLIC STROKE

Stroke can be caused by emboli from the thrombus formed on atherosclerotic plaque.

CAROTID ATHEROSCLEROSIS

It causes around 10% of all strokes, common carotid bifurcation and proximal internal carotid artery are very vulnerable to atherosclerosis. It can be either symptomatic or asymptomatic. Symptomatic stenosis means that the patient had a stroke or TIA and is associated with a higher risk of subsequent stroke than asymptomatic where stenosis is found during screening.carotid artery disease risk factors are same as that of stroke.

Intracranial atherosclerosis and dissection of internal carotid or vertebral arteries are other causes of artery to artery embolism.

SMALL-VESSEL STROKE

They account for 20% of all strokes and are due to atherothrombotic or lipohyalinotic occlusion of a small artery in brain and are referred to as lacunar stroke, ‘lacunes’ in Latin means ‘lake’ because at autopsy they look like fluid and the size of infarct range from 3 mm to 2 cm in diameter They can present with pure motor, sensory, ataxic hemiplegia and dysarthria or clumpsy hand symptoms[49].

LESS COMMON CAUSES OF STROKE

HYPERCOAGULABLE DISORDERS

Mainly increases the risk of cerebral venous sinus or cortical vein thrombosis. Various mutations in homocysteine pathway lead to homocysteinemia which cause arterial stroke.

Venous sinus thrombosis can occur as a complication of oral contraceptive use, pregnancy and the postpartum period, inflammatory bowel disease, intracranial infections (meningitis), and

dehydration. It is also seen in thrombophilia individuals including antiphospholipid syndrome, polycythemia, sickle cell anemia, proteins C and S deficiencies, factor V Leiden mutation, antithrombin III deficiency, homocysteinemia,cand the prothrombin G20210 mutation[49].

They usually come with headache, seizures and neurological deficits like paraperesis.

In children commonest cause for stroke is Sickle cell anemia. Fibromuscular dysplasia involves the cervical arteries and occurs predominantly in females, there are many rings of segmental narrowing which alternate with dilatation in carotid and vertebral arteries.

Temporal (giant cell) arteritis usually affects elderly individuals, where there is subacute granulomatous inflammation of external carotid system especially the temporal arteries with giant cells. carotid or vertebral thrombosis can result from Idiopathic giant cell arteritis involving the great vessels of the aortic arch (Takayasu's arteritis).

Distal small branches (<2 mm diameter) of the main intracranial arteries are involved in granulomatous (necrorizing) arteritis which can occur along with generalized polyarteritis nodosa or granulomatosis with polyangiitis (Wegener's) causing infarcts in the brain, optic nerve, and spinal cord.

Primary central nervous system vasculitis are extremely rare, they involve small or medium sized vessels and no involvement of systemic vasculitis.

Drugs especially amphetamines and cocaine, can cause stroke because of drug induced vasculopathy due to vasospasm or atherosclerosis.

Moyamoya disease involves large intracranial arteries, primarily the distal ICA , the stem of the MCA and ACA. On conventional x-ray angiography, the collateral circulation around the occlusion formed by lenticulostriate arteries appears as "puff of smoke" (moyamoya in Japanese).

Head injury, seizure, migraine, sympathomimetic drug use, eclampsia, and postpartum period

can cause Posterior reversible encephalopathy syndrome (PRES). It involves a hyperperfusion state where blood pressure exceeds the upper limit of cerebral autoregulation resulting in cerebral edema. Patients presents with headache and fluctuating neurologic symptoms and signs, especially visual symptoms. Sometimes cerebral infarction occurs, but usually the clinical and imaging findings reverse completely.

New onset hypertensives can have reversible cerebral vasoconstriction syndrome (RCVS) patient come with sudden, severe headache. These patients can have infarction and hemorrhage.

Multiple small-vessel infarcts within the subcortical white matter can lead to Leukoaraiosis, or periventricular white matter disease. Chronic hypertension causes lipohyalinosis of small penetrating arteries within the white matter.

CADASIL (cerebral autosomal dominant arteriopathy with subcortical infarcts and leukoencephalopathy), CARASIL (cerebral autosomal recessive arteriopathy with subcortical infarcts and leukoencephalopathy), HERNS (hereditary endotheliopathy, retinopathy, nephropathy, and stroke), Fabrys disease are few genetic disorders causing stroke[49].

CLINICAL MANIFESTATIONS OF ISCHEMIC STROKE

Clinical symptoms, neurological deficit following ischemic stroke depends on the involved artery and underlying cause for stroke.

Embolic stroke usually occurs when individual is active with maximum neurological deficit at onset where as thrombotic stroke occurs early in the morning or during sleep and the deficit progress more slowly in step ladder or stuttering pattern [49,86].

Individuals with ischemic stroke present with history of higher function disorder like altered consciousness, language disturbance, hemineglect or agnosia. Motor symptoms like decreased movements or paralysis, involuntary movements, stiffness or heaviness of limbs. Sensory

symptoms like reduced sensation or increased sensation especially in thalamic strokes. They may also come with cranial nerve involvement like deviation of mouth, slurring of speech in 7th nerve or gaze palsy as in 3rd nerve involvement. They might present with other symptoms like headache, vomiting, blurring of vision, seizure which suggest raised intra cranial pressure and symptoms like palpitation, chest pain and breathlessness give clue towards aetiology of stroke.

CORTICAL INFARCT SYNDROMES

1. Internal carotid artery
    1. Ipsilateral visual loss
    2. Ipsilateral middle cerebral artery syndrome
2. Anterior choroidal artery
    1. Contralateral hemiparesis
    2. Contralateral sensory impairment
    3. Contralateral visual field defect
3. Anterior cerebral artery
    1. Contralateral leg > arm paresis
    2. Contralateral leg > arm sensory deficit
4. Middle cerebral artery
    1. Contralateral hemiparesis affecting face and arm > leg
    2. Contralateral sensory deficit affecting face and arm > leg
    3. Contralateral visual field defect
    4. Aphasia (dominant hemisphere)

5. Contralateral hemispatial neglect (nondominant or dominant hemisphere)

5. Posterior cerebral artery
    1. Contralateral homonymous hemianopia (or homonymous superior or inferior quadrantanopia)
    2. Contralateral sensory deficits (thalamic involvement)
6. Basilar artery tip
    1. Bilateral central visual loss
    2. Confusion
7. Basilar artery
    1. Ipsilateral cranial nerve deficit
    2. Contralateral hemiparesis
    3. Contralateral sensory impairment affecting arm and/or leg
    4. Coordination deficit
8. Vertebral artery, posterior inferior cerebellar artery
    1. Ipsilateral sensory impairment over the face
    2. Dysphagia
    3. Ipsilateral Horner syndrome
    4. Ataxia
9. Superior cerebellar artery
    1. Gait ataxia
    2. Ipsilateral limb ataxia
    3. Variable contralateral limb weakness

BRAIN STEM SYNDROMES

MID BRAIN SYNDROMES

1. Weber Syndrome

   Involving 3rd cranial nerve, ventral midbrain and corticospinal tract

   1. ipsilateral 3rd nerve palsy
   2. contralateral hemipaeresis

2. Benedict syndrome

   Involving 3rd cranial nerve, ventral midbrain, corticospinal tract and red nucleus

   1. ipsilateral 3rd nerve palsy
   2. contralateral hemipaeresis
   3. contralateral ataxia and intentional tremors

3. Claude's syndrome

   Involving 3rd cranial nerve, dentatorubral fibres and red nucleus

   1. Ipsilateral 3rd nerve palsy
   2. Contralateral cerebellar ataxia

PONTINE STROKE SYNDROME

1. Foville syndrome

   Involving 7th cranial nerve, ventral pons, paramedian pontine reticular formation.

   1. Ipsilateral 7th nerve palsy
   2. Contralateral hemipaeresis
   3. Ipsilateral conjugate gaze palsy

2. Millard-Gubler Syndrome

   Involving 6th and 7th cranial nerves and ventral pons

   1. Ipsilateral LMN 7th nerve

b) Ipsilateral 6th nerve

c) Contralateral hemiparesis

d) +/– contralateral pain/temperature

3. Locked-in Syndrome

Involving ventral pons, basi pontis

Occlusion of basilar artery leads to locked in syndrome where patient is awake and alert but unable to move or communicate except for vertical eye movements.

1. Bilateral LMN 7th nerve
2. Bilateral 6th nerve
3. Quadriplegia
4. +/– bilateral light touch/proprioception

4. Raymond Syndrome

Involving 6^{th} cranial nerve, cortico facial fibres and corticospinal tract

1. Ipsilateral internuclear ophthalmoplegia
2. Contralateral hemiparesis
3. Contralateral UMN 7th nerve
4. Contralateral light touch, proprioception
5. +/– contralateral pain/temperature (with ipsilateral Horner)

5. Marie-Foix syndrome

Involving 7^{th} and 8^{th} cranial nerves, spinothalamic tracts, cerebellar and corticospinal tract

1. contralateral hemiplegia/hemiparesi
2. contralateral loss of pain and temperature sensation
3. ipsilateral limb ataxia
4. ipsilateral facial paralysis
5. ipsilateral hearing loss, vertigo and nystagmus

MEDULLARY STROKE SYNDROMES

1. Medial medullary syndrome (Dejerine syndrome)

   involving corticospinal tract, medial leminiscus and 12th nerve

   1. Ipsilateral tongue palsy
   2. Contralateral hemiparesis
   3. Contralateral light touch, proprioception

2) Lateral medullary syndrome (Wallenberg Syndrome)

   Involving spinothalamic tract, 5th and 8th nerve nucleus, nucleus ambigus, cerebellum and inferior cerebellar peduncle

   1. Ipsilateral facial anesthesia
   2. Contralateral pain, temperature
   3. Ipsilateral Horner syndrome
   4. Ipsilateral IX, X palsy- dysphagia
   5. Ipsilateral ataxia (olivocerebellar tract)

DIAGNOSIS

Stroke is a clinical diagnosis. Thorough history, physical and neurological examination diagnoses stroke.

HISTORY

An accurate history should be obtained including the onset of deficit, progression, rapidity of development of symptoms, time of onset and other associated symptoms along with past medical illness, risk factors including smoking, alcohol history and personal history has to be elucidated. History may require inputs from informant as most of the time stroke patient cannot communicate effectively.

PHYSICAL EXAMINATION

General physical examination can give clue regarding etiology, type of stroke.Irregularly irregular pulse suggests atrial fibrillation, absent pulse may suggest takayasu arteritis , pulsus bisferans, collapsing pulse suggest valvular heart disease as risk factor and cause of stroke, where as elevated blood pressure in the setting of sudden onset neurological deficit in the area localisable to thalamus, basal ganglion or Pons suggest hemorrhagic stroke. Signs of atherosclerosis like frank sign, xanthelasma suggest atherothrombotic stroke. A detailed general physical examination has to be done to look for causes of stroke especially in stroke in young patients.

Carotid artery examination is a must in all stroke patients. Absent carotid pulses suggests decreased amplitude of pulse and in equality between two carotids suggest atherosclerosis, aortic dissection, arteritis or embolus. Carotid bruit suggests partial obstruction of carotid artery or conducted cardiac murmur. Usually bruit develops when the lumen of artery us decreased to <50% of its cross sectional diameter.

A general neurologic examination including evaluations of cognition, language, spatial neglect, cranial nerves, motor function, sensory system, coordination, gait, and reflexes is important both for documenting stroke related deficits and for localising the affected brain area and the severity of the injury.

The National Institutes of Health Stroke Scale is a standardized graded neurologic impairment assessment tool for measuring the severity of the stroke, determining the risks and benefits of

management, assessing prognosis, and observing patients objectively over time(86).

Other system examination especially cardio vascular system plays a significant role in evaluating stroke. Presence of murmur, third heart sound, and irregular rhythm give clue regarding cardioembolic stroke. Respiratory system examination gives clue regarding infection,

tuberculosis, wegeners as a cause of stroke also one has to examine respiratory system for complications like aspiration pneumonia. Abdominal system examination may suggest any malignancy, organomegaly giving clue for possible etiology of stroke including autoimmune disorders, coagulopathies, sickle cell anemia.

NATIONAL INSTITUTES OF HEALTH STROKE SCALE (NIHSS)

National institutes of health stroke scale is a neurological examination tool widely used to evaluate stroke patients on parameters like level of consciousness, language, neglect, visual field loss, motor strength , extra ocular movements, ataxia, dysarthria and sensory loss[8,87]. The reliability of NIHSS to predict severity of stroke and outcome is well documented[7]. NIHSS is the most frequently used scale in clinical trials and is easily reproducible. Doctors depend upon NIHSS to evaluate acute stroke patients and decide regarding the treatment[88].

NIHSS was first developed by combining several stroke deficit rating scales like Canadian neurological scale, Edinburgh-2 coma scale , Oxbury initial severity scale and university of Cincinatti scale for trial of naloxone in acute stroke sponsored by National institutes of health[8]. This scale was complex and had poor reliability. During NINDS r-tPA Trial, significant changes were made to it[30]. After successful use in NINDS r-tPA trial NIHSS has become the gold standard for rating stroke severity. Due to critisicm of poor reliability, complexity and not having clear information modifications were made to original NIHSS, 4 questions were dropped from the 15 parameters and non reliable parameters were removed, the score was further validated and found that its reliability increased with video training[89].

The maximum score possible is 31 when compared to 42 of NIHSS. Video training has improved the reliability of NIHSS[89]. To further increase the reliability modifications to the current NIHSS scale are made and poor reliability items like level of consciousness, facial weakness, ataxia, and dysarthria are removed and sensory system choice was reduced to 2 from 3, mNHISS is identical

to NIHSS clinically with fewer items and might be simple to use in clinical trials but no prospective studies are done to validate it[90].

NIHSS is reliable even when used by trained non-neurologists and predict outcome or the presence of large vessel occlusions[91]. Not all the symptoms and signs of stroke are considered as deficits in NIHSS, It outweighs deficits in patients with left compared to right brain strokes, with strokes of similar size left sided strokes scored 4 more points than right and for the given NIHSS score the median volume of right sided stroke was much higher than the left also It is highly biased toward deficits due to anterior circulation strokes, posterior circulation stroke deficits receive lesser points compared to anterior deficits[93].

Recent studies have shown that NIHSS is not a reliable and fails to detect stroke, 0 on NIHSS does not mean absence of stroke as found in a study[9]. It is also shown that NIHSS has limitation to predict outcome in chronic stroke and lacks association with impairment and activities of daily living measurements[10].

Even though NIHSS is designed for clinical trials and not advised as bedside tool for clinician day to day practice[94] it is used worldwide as a prognostic tool and to make treatment decisions.

NIHSS system requires training and certification to assure reproducibility across clinical trials, it does not accurately consider patient's coordination; gait impairment; cortical sensory function; distal motor function; memory; or cognition this is done intentionally to achieve reproducibility[95]. The rule of NIHSS is to 'score what you see, not what you think' but sometimes in a case of aphasia patient might fail to answer question about orientation and which might be scored as points by non neurologist even though aphasia is the problem not stupor or delirium[95]. A recent study found that NIHSS has a very weak correlation with motor dysfunction, functional limitation, and health status measurements and its use in predicting motor outcomes in real life scenarios is not beneficial[96].

Because of these limitations, NIHSS cannot be used to predict prognosis and outcomes of patient

with acute ischemic stroke and there is a need of a simple , reliable and easily reproducible tool to prognosticate these patients.

NIHSS ITEM INSTRUCTIONS & SCALE DEFINITIONS (97)

Administer stroke scale items in the order listed. Record performance in each category after each subscale exam. Do not go back and change scores. Follow directions provided for each exam technique. Scores should reflect what the patient does, not what the clinician thinks the patient can do. The clinician should record answers while administering the exam and work quickly. Except where indicated, the patient should not be coached (i.e., repeated requests to patient to make a special effort).

INSTRUCTIONS	SCALE DEFINITION	SCORE
1a. **Level of Consciousness:** The investigator must choose a response if a full evaluation is prevented by such obstacles as an endotracheal tube, language barrier, orotracheal trauma/bandages. A 3 is scored only if the patient makes no movement (other than reflexive posturing) in response to noxious stimulation.	0 = **Alert**; keenly responsive. 1 = **Not alert**; but arousable by minor stimulation to obey, answer, or respond. 2 = **Not alert**; requires repeated stimulation to attend, or is obtunded and requires strong or painful stimulation to make movements (not stereotyped). 3 = Responds only with reflex motor or autonomic effects or totally unresponsive, flaccid, and areflexic.	_____
1b. **LOC Questions**: The patient is asked the month and his/her age. The answer must be correct—there is no partial credit for being close. Aphasic and stuporous patients who do not comprehend the questions will score 2. Patients unable to speak because of endotracheal intubation, orotracheal trauma, severe dysarthria from any cause, language barrier, or any other problem not secondary to aphasia are given a 1. It is important that only the initial answer be graded and that the examiner not "help" the patient with verbal or nonverbal cues.	0 = **Answers** both questions correctly. 1 = **Answers** one question correctly. 2 = **Answers** neither question correctly.	_____
1c. **LOC Commands:** The patient is asked to open and close the eyes and then to grip and release the nonparetic hand. Substitute another one-step command if the hands cannot be used. Credit is given if an unequivocal attempt is made but not completed due to weakness. If the patient does not respond to command, the task should be demonstrated to him or her (pantomime), and the result scored (i.e., follows none, one, or two commands). Patients with trauma, amputation, or other physical impediments should be given suitable one-step commands. Only the first attempt is scored.	0 = **Performs** both tasks correctly. 1 = **Performs** one task correctly. 2 = **Performs** neither task correctly.	_____
2. **Best Gaze:** Only horizontal eye movements will be tested. Voluntary or reflexive (oculocephalic) eye movements will be scored, but caloric testing is not done. If the patient has a conjugate deviation of the eyes that can be overcome by voluntary or reflexive activity, the score will be 1. If a patient has an isolated peripheral nerve paresis (CN III, IV, or VI), score a 1. Gaze is testable in all aphasic patients. Patients with ocular trauma, bandages, preexisting blindness, or other disorder of visual acuity or fields should be tested with reflexive movements, and a choice made by the investigator. Establishing eye contact and then moving about the patient from side to side will occasionally clarify the presence of a partial gaze palsy.	0 = **Normal**. 1 = **Partial gaze palsy**; gaze is abnormal in one or both eyes, but forced deviation or total gaze paresis is not present. 2 = **Forced deviation**, or total gaze paresis not overcome by the oculocephalic maneuver.	_____
3. **Visual:** Visual fields (upper and lower quadrants) are tested by confrontation, using finger counting or visual threat, as appropriate. Patients may be encouraged, but if they look at the side of the moving fingers appropriately, this can be scored as normal. If there is unilateral blindness or enucleation, visual fields in the remaining eye are scored. Score 1 only if a clear-cut asymmetry, including quadrantanopia, is found. If patient is blind from any cause, score 3. Double simultaneous stimulation is performed at this point. If there is extinction, patient receives a 1, and the results are used to respond to item 11.	0 = **No visual loss**. 1 = **Partial hemianopia**. 2 = **Complete hemianopia**. 3 = **Bilateral hemianopia** (blind including cortical blindness).	_____
4. **Facial Palsy:** Ask—or use pantomime to encourage—the patient to show teeth or raise eyebrows and close eyes. Score symmetry of grimace in response to noxious stimuli in the poorly responsive or noncomprehending patient. If facial trauma/bandages, orotracheal tube, tape, or other physical barriers obscure the face, these should be removed to the extent possible.	0 = **Normal** symmetrical movements. 1 = **Minor paralysis** (flattened nasolabial fold, asymmetry on smiling). 2 = **Partial paralysis** (total or near-total paralysis of lower face). 3 = **Complete paralysis** of one or both sides (absence of facial movement in the upper and lower face).	_____
5. **Motor Arm**: The limb is placed in the appropriate position: extend the arms (palms down) 90 degrees (if sitting) or 45 degrees (if supine). Drift is scored if the arm falls before 10 seconds. The aphasic patient is encouraged using urgency in the voice and pantomime, but not noxious stimulation. Each limb is tested in turn, beginning with the nonparetic arm. Only in the case of amputation or joint fusion at the shoulder, the examiner should record the score as untestable (UN) and clearly write the explanation for this choice.	0 = **No drift**; limb holds 90 (or 45) degrees for full 10 seconds. 1 = **Drift**; limb holds 90 (or 45) degrees, but drifts down before full 10 seconds; does not hit bed or other support. 2 = **Some effort against gravity**; limb cannot get to or maintain (if cued) 90 (or 45) degrees, drifts down to bed, but has some effort against gravity. 3 = **No effort against gravity**; limb falls. 4 = **No movement**. UN = **Amputation** or joint fusion, explain: _____ 5a. **Left Arm** 5b. **Right Arm**	_____
6. **Motor Leg:** The limb is placed in the appropriate position: hold the leg at 30 degrees (always tested supine). Drift is scored if the leg falls before 5 seconds. The aphasic patient is encouraged using urgency in the voice and pantomime, but not noxious stimulation. Each limb is tested in turn, beginning with the nonparetic leg. Only in the case of amputation or joint fusion at the hip, the examiner should record the score as untestable (UN) and clearly write the explanation for this choice.	0 = **No drift**; leg holds 30-degree position for full 5 seconds. 1 = **Drift**; leg falls by the end of the 5-second period but does not hit bed. 2 = **Some effort against gravity**; leg falls to bed by 5 seconds, but has some effort against gravity. 3 = **No effort against gravity**; leg falls to bed immediately. 4 = **No movement**. UN = **Amputation** or joint fusion, explain: _____ 6a. **Left Leg** 6b. **Right Leg**	_____

INSTRUCTIONS	SCALE DEFINITION	SCORE
7. **Limb Ataxia**: This item is aimed at finding evidence of a unilateral cerebellar lesion. Test with eyes open. In case of visual defect, ensure testing is done in intact visual field. The finger-nose-finger and heel-shin tests are performed on both sides, and ataxia is scored only if present out of proportion to weakness. Ataxia is absent in the patient who cannot understand or is paralyzed. Only in the case of amputation or joint fusion, the examiner should record the score as untestable (UN) and clearly write the explanation for this choice. In case of blindness, test by having the patient touch nose from extended arm position.	0 = **Absent**. 1 = **Present in one limb**. 2 = **Present in two limbs**. UN = **Amputation** or joint fusion, explain: _____	_____
8. **Sensory**: Sensation or grimace to pinprick when tested, or withdrawal from noxious stimulus in the obtunded or aphasic patient. Only sensory loss attributed to stroke is scored as abnormal, and the examiner should test as many body areas (arms [not hands], legs, trunk, face) as needed to accurately check for hemisensory loss. A score of 2, "severe or total sensory loss," should only be given when a severe or total loss of sensation can be clearly demonstrated. Stuporous and aphasic patients will, therefore, probably score 1 or 0. The patient with brain stem stroke who has bilateral loss of sensation is scored 2. If the patient does not respond and is quadriplegic, score 2. Patients in a coma (item 1a = 3) are automatically given a 2 on this item.	0 = **Normal**; no sensory loss. 1 = **Mild-to-moderate sensory loss**; patient feels pinprick is less sharp or is dull on the affected side; or there is a loss of superficial pain with pinprick, but patient is aware of being touched. 2 = **Severe to total sensory loss**; patient is not aware of being touched in the face, arm, and leg.	_____
9. **Best Language**: A great deal of information about comprehension will be obtained during the preceding sections of the examination. For this scale item, the patient is asked to describe what is happening in the attached picture, to name the items on the attached naming sheet, and to read from the attached list of sentences. Comprehension is judged from responses here, as well as to all of the commands in the preceding general neurological exam. If visual loss interferes with the tests, ask the patient to identify objects placed in the hand, repeat, and produce speech. The intubated patient should be asked to write. The patient in a coma (item 1a = 3) will automatically score 3 on this item. The examiner must choose a score for the patient with stupor or limited cooperation, but a score of 3 should be used only if the patient is mute and follows no one-step commands.	0 = **No aphasia**; normal. 1 = **Mild-to-moderate aphasia**; some obvious loss of fluency or facility of comprehension, without significant limitation on ideas expressed or form of expression. Reduction of speech and/or comprehension, however, makes conversation about provided materials difficult or impossible. For example, in conversation about provided materials, examiner can identify picture or naming card content from patient's response. 2 = **Severe aphasia**; all communication is through fragmentary expression; great need for inference, questioning, and guessing by the listener. Range of information that can be exchanged is limited; listener carries burden of communication. Examiner cannot identify materials provided from patient response. 3 = **Mute, global aphasia**; no usable speech or auditory comprehension.	_____
10. **Dysarthria**: If patient is thought to be normal, an adequate sample of speech must be obtained by asking patient to read or repeat words from the attached list. If the patient has severe aphasia, the clarity of articulation of spontaneous speech can be rated. Only if the patient is intubated or has other physical barriers to producing speech, the examiner should record the score as untestable (UN) and clearly write an explanation for this choice. Do not tell the patient why he or she is being tested.	0 = **Normal**. 1 = **Mild-to-moderate dysarthria**; patient slurs at least some words and, at worst, can be understood with some difficulty. 2 = **Severe dysarthria**; patient's speech is so slurred as to be unintelligible in the absence of or out of proportion to any dysphasia, or is mute/anarthric. UN = **Intubated** or other physical barrier, explain:_____ _____	_____
11. **Extinction and Inattention (formerly Neglect):** Sufficient information to identify neglect may be obtained during the prior testing. If the patient has a severe visual loss preventing visual double simultaneous stimulation, and the cutaneous stimuli are normal, the score is normal. If the patient has aphasia but does appear to attend to both sides, the score is normal. The presence of visual spatial neglect or anosognosia may also be taken as evidence of abnormality. Since the abnormality is scored only if present, the item is never untestable.	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 own hand or orients to only one side of space.	_____

From http://www.ninds.nih.gov/doctors/nih_stroke_scale.pdf. Accessed February 26, 2015.

NATIONAL INSTITUTE OF HEALTH STROKE SCALE

INVESTIGATIONS

Stroke is a clinical diagnosis and investigations including imaging of brain are done to support the diagnosis.

BRAIN IMAGING

Once diagnosis of stroke is made imaging of brain is necessary to detect the type of stroke whether it is an ischemic or hemorrhagic stroke. Imaging can also locate the area of injury and rule out stroke mimics like tumour, abscess or subdural hematoma. Both Computed tomography (CT) and Magnetic Resonance Imaging (MRI) plays essential part in assesing patients with ischemic stroke.

Brain CT is widely and rapidly available and provides the information necessary for the treatment of most patients with acute stroke. CT may be normal in patients presenting very early and examination of early CT changes like loss of gray/ white-matter differentiation, sulcal effacement, effacement of the Sylvian fissure, and obscuration of the lentiform nucleus, hypodensity an swelling of insular cortex (insular ribbon sign) in the area of MCA territory is essential. Sometimes the large intracranial vessels appear hyperdense before infarction appears in CT like the dense MCA sign which indicates thrombotic or embolic occlusion of MCA and often predicts a large cortical infarct. Area of infarction appear hypodense in CT whereas area of hemorrhage appear hyperdense. CT is easily available, cheap and rapid gold standard test in the evaluation of stroke but posterior fossa structures cannot be visualised as it is often obscured by artefacts from petrous bones[52,86].

MRI plays a significant role in patients who present with acute stroke and in posterior circulation stroke. MRI is more sensitive in visualizing acute ischemic infarct and brain stem, cerebellar structures, but it is not available widely, costly and time consuming and patients with metallic implants and devices like cardiac pacemaker, orthopedic implants cannot undergo MRI. It is superior to CT in acute ischemic stroke because diffusion, perfusion and gradient echo MR

sequences can rapidly detect early ischemic and hemorrhagic lesions and also describe the amount of at-risk tissue (the "ischemic penumbra"). The sensitivity of MRI to differentiate infarct from normal tissue mainly depends on changes in tissue T1 and T2 relaxation times. Magnetic resonance angiography (MRA) is used to generate pictures of arteries to look for stenosis, occlusion or aneurysms and the specificity and sensitivity of MRA can be increased by administrating gadolinium. DW-MRI detects acute cerebral ischemia very early and also differentiates acute from chronic stroke[52,86].

Advanced imaging techniques like multifocal imaging with diffusion/perfusion or perfusion CT and CTA are being used to detect ischemic penumbra[98]. Recent study found that diffusion/perfusion mismatches are indicative of brain at risk still the utility of diffusion/perfusion MRI to select patients for thrombolytic therapy is to be determined[99]. There is increasing trend of CT perfusion imaging being used for early diagnosis of stroke along with routine non contrast CT and Ct angiography but there is risk of radiation and contrast dye exposure to patients[98].

Alberta Stroke Programme Early CT Score (ASPECTS) is a 10 point CT scan scoring system which has been described for assessing prognosis and identifying patients who are not likely to have good outcomes after IV thrombolysis[100].

LABORATORY INVESTIGATIONS

They help to determine conditions that may mimic, complicate or cause acute ischemic stroke.

A case of suspected ischemic stroke should undergo complete blood count (CBC) and platelet count, blood glucose level, serum electrolytes, renal function tests, lipid analyses, urinalysis, chest x ray and 12-lead ECG. Glycosylated hemoglobin (HbA1c) in cases of diabetes or suspected diabetes. Laboratory tests for thrombophilia, erythrocyte sedimentation rate,

prothrombin time, aPTT and INR and connective tissue disorder, 2D echo are done as and when necessary.

Elevated total count in CBC suggests infection as a cause of stroke, polycethemia can result in increased viscosity causing blockage of vessels leading to stroke, and thrombocytopenia either primary or secondary can lead to platelet thrombi. Deranged PT, aPTT and INR suggest underlying coagulation disorder and they might not be considered for treatment with intravenous recombinant tissue plasminogen activator. Both hypo and hyperglycemia can mimic as stroke. Hyperglycemia is a risk factor and can worsen brain injury during stroke. Deranged renal function test can cause stroke and also interfere with treatment. Dyselectrolytemia can mimick as stroke and also be the cause and effect, stroke per se can cause syndrome of inappropriate anti diuretic hormone secretion (SIADH) leading to hyponatremia.[49,52,86]

CARDIAC EVALUATION OF THE STROKE PATIENT

It plays an important role determining whether emboli have cardiac source and has to be evaluated thoroughly in suspected cases. 12 lead Electrocardiogram (ECG) is the first investigation and may reveal changes like ST elevation in myocardial infarction or irregular rhythm with absent P waves in atrial fibrillation which is the most common cause for embolic stroke. Stroke itself can lead to arrhythmias and cardiac dysfunction.

Acute myocardial infarction can cause stroke by mural thrombi and stroke can also precipitate MI so in suspected cases there is need for cardiac enzymes like troponin I and Brain natriuretic peptide, recent studies have suggested there is elevation of BNP in stroke patients[15].

Non invasive cardiac imaging like two-dimensional echocardiography is used to screen for variety of cardiac conditions like left ventricular hypertrophy, right to left shunts as in ASD, patent foramen ovale, to look for regional wall motion abnormality following myocardial infarction and ventricular dysfunction these disorders can cause stroke.

2D echo can also detect left ventricular thrombi, thrombi which have protruding edge and mobile appearance are more likely to embolise and transthoracic or trans esophageal echo view may be required to evaluate it. Atrial fibrillation cause atrial thrombus by due to blood stasis in left atrium or atrial appendage and are not visible in routine 2D echo and trans esophageal echocardiography(TEE) is the gold standard technique to detect cardiac source of emboli. Continuous ECG monitoring is advised routinely in cases of suspected paroxysmal arrhythmias and 24 hour holter monitoring may also be required in selected cases[52].

OTHER IMAGING TECHNIQUES

Doppler imaging, B-mode scanning, duplex (combined B-mode and Doppler) scanning, and transcranial Doppler (TCD) imaging are adviced in primary screening and rarely used in acute setting.

Cerebral angiography techniques like CT and MR angiography are used in making treatment decision like endovascular therapy.

DIFFERENTIAL DIAGNOSIS OF ACUTE ISCHEMIC STROKE

The hallmark of acute ischemic stroke is the sudden onset of a focal neurologic deficit, usually attributed to an area of brain supplied by a specific artery.

Other neurological conditions which present acutely can mimic stroke

1. Migraine with aura

   Patient can have focal neurologic deficits, including aphasia, visual changes, vertigo, weakness, numbness, and incoordination

2. Partial seizures

   Patient can have negative symptoms, including aphasia and paresis, and a patient with a

postictal Todd paralysis may looks like hemiplegic patient.

Also stroke itself can cause seizures complicating diagnosis.

3. First episode of multiple sclerosis
4. Mass lesion – Neoplasm and Brain abscess

They usually have slowly worsening symptoms but can manifest acutely

5. Metabolic disorders like hypoglycemia or hyperglycemia
6. Toxin exposures and drug intoxications
7. Malingering or other psychiatric illness

TREATMENT OF ACUTE ISCHEMIC STROKE

The main goal of treatment is to prevent further damage or reverse brain injury and improve clinical outcome. After diagnosis of acute ischemic stroke CT brain is done to rule out hemorrhage.

Treatment categories

1. Medical support
2. IV thrombolysis
3. Endovascular revascularization
4. Antithrombotic treatment
5. Neuroprotection
6. Stroke centers and rehabilitation.

MEDICAL SUPPORT

The primary goal is to optimize cerebral perfusion in ischemic penumbra region.

1. Patient's airway, breathing and circulation (ABCs) are assessed and treated;

   Airway support and ventilator assistance are advised to patient who have low consciousness and bulbar stroke that compromises breathing.

   Saturation to be maintained > 94%

2. Care is taken to prevent complication of stroke like infections (pneumonia, skin or urine infections), deep vein thrombosis and pulmonary embolism.

   Infections are treated with antibiotics

   Subcutaneous heparin can be used as prophylaxis and pneumatic compressive stockings have proven benefit in preventing DVT

3. Collateral blood flow in brain may be dependent on blood pressure

   1. Low Blood pressure and hypovolemia has to be corrected to maintain brain perfusion
   2. If patient has high BP and is a candidate for thrombolysis BP should be carefully lowered to systolic BP < 185mmHg and diastolic < 110mmHg before treatment is initiated

   Labetalol and Nicardapine are the drugs recommended to lower BP and BP to be maintained <180/105mmHg for first 24 hours after IV thrombolysis

   3. If patient has elevated BP and is not a candidate for IV thrombolysis and BP <220/110mmHG there is no evidence that initaing treatment is of any mortality benigit and for those with pre existing HTN , antihypertensives has to be reinitiatied.
   4. If patient has elevated BP and is not a candidate for IV thrombolysis and BP >220/110mmHG, BP has to be lowered by 15% in first 24 hours.[101]

4. Fever is detrimental and should be treated with antipyretics and surface cooling, hypothermia is a good neuroprotective agent but its use in acute stroke is not proven and it may increase risk of infection like pneumonia.
5. Serum glucose should be maintained between 60 to 180 mg /dl by using insulin or glucose infusion whenever necessary.
6. IV Mannitol and fluid restriction therapy can be used if patient develops significant edema. Hemicraniectomy is a proven stratergy and reduces mortality by 50% and outcome is significantly improved in stroke survivors.
7. Extra care has to be taken in patients with cerebellar stroke as even small edema increases intracranial pressure by obstructing CSF and causing hydrocephalus and compressing brain stem which can cause coma and respiratory arrest which require decompression surgeries.[49,101]
8. Nutrition – enteral diet has to be begun within 1 week of stroke onset, ryles tube feeding in patients with dysphagia and percutaneous gastrostomy tubes for patients who fail to improve is adviced[101].
9. Depression screening with structured tool and treatment with antidepressant is recommended by AHA[101].

INTRAVENOUS THROMBOLYSIS

There is clear benefit of intravenous recombinant tissue plasminogen activator(rtPA) in acute ischemic stroke.[30] Administration of rtPA is approved between 3 – 4.5 hours in Europe and Canada where as in USA it is approved only for 0-3 hours.

INDICATIONS

1. Clinical diagnosis of stroke
2. Onset of symptoms to time of drug administration ≤4.5 Hours

3. CT scan showing no hemorrhage or edema of >1/3 of the MCA territory
4. Age 18 ≥ years

CONTRAINDICATION

1. Sustained BP >185/110 mmHg despite treatment
2. Bleeding diathesis
3. Recent head injury or intracerebral hemorrhage
4. Major surgery in preceding 14 days
5. Gastrointestinal bleeding in preceding 21 days
6. Recent myocardial infarction

ADMINISTRATION OF rtPA

1. IV access with two peripheral IV lines (avoid arterial or central line placement)
2. Review eligibility for rtPA
3. Administer 0.9 mg/kg IV (maximum 90 mg) of IV alteplase as 10% of total dose by bolus over 1 minute, followed by remainder of total dose over 60 minutes.
4. Frequent cuff blood pressure monitoring
5. No other antithrombotic treatment for 24 hours
6. For decline in neurologic status or uncontrolled blood pressure, stop infusion give cryoprecipitate, and reimage brain emergently
7. Avoid urethral catheterization for ≥2 hours

ENDOVASCULAR REVASCULARIZATION

Ischemic strokes which involve large vessels like Middle Cerbebral Artery, Internal Carotid and Basilar artery have poor prognosis and high risk of mortality and morbidity, they have high clot

volume and IV rtPA alone fails to open up the occlusion so intraarterial thrombolytics were used to increase the chance of clot lysis and decrease systemic bleeding complication, PROCAT II trial found significant improvement in outcome for intra-arterial pro-urokinase even upto 6 hours after onset in MCA strokes[102]. Intra-arteial thrombolysis is not approved by FDA many studies suggest it can be considered when mechanical thrombectomy fails.[49]

In patients who have contraindication or failed IV thrombolysis, Endovascular mechanical thrombectomy has been tried as adjuvant or alternate treatment. The HERMES meta analysis study which included the 5 studies MR CLEAN, ESCAPE, REVASCAT, SWIFT PRME and EXTEND IA found that mechanical thrombectomy within 6 hours of stroke after large vessel occlusion improved the outcome and the number needed to treat to decrease disability by 1 point in modified rankin score was 2.6[33]. The DAWN and DEFUSE 3 trials have reported good clinical outcome in patients undergoing mechanical thrombectomy with 24 and 12 hours of onset of stroke respectively (34,103). If the patient has good collaterals in CT or MRI perfusion imaging patient can be treated with mechanical thrombectomy for upto 24 hours[49,101].

Patient who meets following criteria should undergo mechanical thrombectomy with stent retriever.[101]

1. Pre stoke mRS score 0-1
2. Internal carotid artery or Middle Cerebral artery (M1 Segment) occlusion
3. Age > 18 years
4. NIHSS score > 6
5. ASPECTS >6
6. Management can be started (groin puncture) within 6 hours of onset of symptoms

ANTITHROMBOTIC TREATMENT

Platelet Inhibition

Aspirin is the only drug proven to effective in treatment of acute ischemic stroke. The 2 trials IST and CAST where aspirin was given 300mg/ day and 160 mg/ day respectively reduced mortality and stroke recurrence(31,32). American heart association recommends initial Aspirin dose of 325mg.[101]

ANTICOAGULATION

There is no clear benefit of anticoagulation following acute ischemic stroke, routine use of heparin or other anticoagulants are not recommended and have shown high risk of hemorrhage.[101]

NEUROPROTECTION

Even though many drugs have shown positive results in animal studies in prolonging brain's tolerance to ischemia, no neuroprotectors have been approved by FDA in treatment of ischemic stroke. There is no benefit of either pharmacological or non pharmacological treatments according to AHA.[101]

Hypothermia is beneficial neuroprotective agent after cardiac arrest but studies have failed to prove its efficacy in acute ischemic stroke and patients are prone to develop pneumonia which impacts stroke outcome.[49,52]

STROKE CENTERS AND REHABILITATION

Many centres have a dedicated stroke team which provide emergency services round the clock like medical management, IV thrombolysis or thrombectomy in cases of acute ischemic stroke.

Studies have proven the benefit of stroke centres in improving neurological recovery and decreasing mortality.[49,52]

Rehabilitation of stroke patients includes early physical, occupational and speech therapies. Patient and family are educated about the neurological deficit and complications of stroke and its prevention are explained including prevention of DVT, back care to prevent bed sores, physiotherapy to prevent contractures, bowel and bladder care to prevent infections. The aim of rehabilitation is to increase recovery by giving a safe, progressive regimen.[49]

POST STROKE ASSESMENT

In spite of advances in early diagnosis and treatment of acute ischemic stroke many patients experience some functional disability or mortality following stroke. 'To be able to walk again' is the goal of patients after stroke; many studies have been done to asses stroke outcome.

Morbidity following stroke has many modalities like impairment (sign of underlying pathology), disability (functional result of impairment) and handicap (social impact of disease).

Stroke is a difficult case to study because of its highly variable clinical presentation, variety of causes and the patients who survive have disability which is highly variable. The traditional neurological examination is suited for only description of a single patient and cannot be used in large scale trial descriptions hence many impairment scales were developed which involved scoring different modalities of neurological examination and adding the scores to get the neurological condition of the patient.

Stroke is the second most common cause of disability worldwide efficacy of its treatment is usually described by measuring disability, ie, functional assessment. Barthel Index (BI) and modified Rankin score (mRS) are two of the most extensively used functional assessment scales which measure basic activities of daily living (ADL), these are the tasks the individual has to perform to achieve functional independence. Barthel index measures performance where as Rankin score measures independence.[104,105]

BARTHEL INDEX

It was first developed in 1965[11] by modifying 'Maryland disability index' which was used in Baltimore's chronic disease hospitals to measure recovery.[104,106] Barthel index had 10 tasks which the patient had to perform like feeding, grooming, care of bowel and bladder, dressing, bathing, getting up from bed and climbing stairs and scores were given with 5 points increment and minimum score is 0 and maximum being 100 which tells that patient is completely independent. It was modified by Collin et al who scored increments in 1 point and maximum score of 20 was used and also found that asking a trained nurse or relative of patient is as reliable as direct examination.[107]

Barhel index has been used extensively in many disorders like spinal cord injuries, burns, rheumatoid arthritis, elderly patients and also in cardiac disorders.[104,108] It has been proven to be reliable after stroke.[109] There are other modified Barthel scores with truncated and expanded versions depending on the disorder and need of the study and the BI has been found reliable and validated for stroke and is the most commonly used scales for stroke to asses ADL.[104]

Barthel index was originally described by interview and distant observation, this still remains the standard. BI should be graded only on what the patient does and not on what the examiner thinks patient can do. There is no recognized training course for Barthel index unlike NIHSS.

BI is a scale used to measure physical dependence and cannot be used to measure to evaluate speech, cognition or mood of the patient. Change in BI over a set period of time can be a better predictor of clinical intervention than measuring a single time and it is more sensitive to change in scores than other scales used in stroke the limitation of Barthel index is that it is not sensitive at extremes of ability because of 'floor effect' and 'ceiling effect'[104] and making it less useful in minor or severe stroke but these effects are not seen in modified Rankin scale. The worst outcome after stroke is death but unlike mRS there is no scoring to represent mortality.

Criteria to classify patients based on Barthel Index vary widely and the cut-off values are chosen arbitrarily and not validated. It is an ordinal scale and many studies have dichotomized it and it has been suggested that at score of < 40 patient is completely dependent, at score >60 patient becomes independent for personal care like feeding, bowel and bladder continence but still need assistance and at score > 85 patients are reasonably independent with minimal aid. The landmark trials NINDS and ECASS II have taken score of >95 as minimal or no disability to define good or favourable outcome[104,105] but this approach has been criticised as inefficient as a patient with minimal disability can make good recovery and score >85 and have no impact on trial where as another patient with major disability can recover substantially but have a score of less than 85 hence analytical methods that measure changes are considered.

Because of its easiness to perform, simplicity, fastness it is one of the most widely used scale to measure outcome following stroke.

THE BARTHEL INDEX SCORING[110]

Activity	Score
FEEDING 0 = unable 5 = needs help cutting, spreading butter, etc., or requires modified diet 10 = independent	_____
BATHING 0 = dependent 5 = independent (or in shower)	_____
GROOMING 0 = needs to help with personal care 5 = independent face/hair/teeth/shaving (implements provided)	_____
DRESSING 0 = dependent 5 = needs help but can do about half unaided 10 = independent (including buttons, zips, laces, etc.)	_____
BOWELS 0 = incontinent (or needs to be given enemas) 5 = occasional accident 10 = continent	_____
BLADDER 0 = incontinent, or catheterized and unable to manage alone 5 = occasional accident 10 = continent	_____
TOILET USE 0 = dependent 5 = needs some help, but can do something alone 10 = independent (on and off, dressing, wiping)	_____
TRANSFERS (BED TO CHAIR AND BACK) 0 = unable, no sitting balance 5 = major help (one or two people, physical), can sit 10 = minor help (verbal or physical) 15 = independent	_____
MOBILITY (ON LEVEL SURFACES) 0 = immobile or < 50 yards 5 = wheelchair independent, including corners, > 50 yards 10 = walks with help of one person (verbal or physical) > 50 yards 15 = independent (but may use any aid; for example, stick) > 50 yards	_____
STAIRS 0 = unable 5 = needs help (verbal, physical, carrying aid) 10 = independent	_____
TOTAL (0–100):	_____

SERUM PROCALCITONIN (PCT) IN STROKE

PROCALCITONIN

The existence of precursor of Calcitonin was first suggested by Moya F et al. in 1975. A large biosynthetic molecule which splits intracellularly to form a molecule, they named this hormone as Procalcitonin.[111] PCT is a precursor peptide of the hormone calcitonin which is produced in C Cells of thyroid gland and is involved in calcium homeostasis. In 1981 a study by Allison et al. showed the synthesis of calcitonin as precursor protein in human. Later studies showed that calcitonin is a product of sequential co and post transitional modification[112,113]. CALC-1 gene located in chromosome 11 is responsible for the production of PCT from thyroid C cells in a healthy individual. preprocalcitonin is a mRNA product which is further modified to procalcitonin which has 116 amino acids. 3 distinct molecules are formed after its cleavage;

1. Active calcitonin (32 amino acid)
2. Kata- calcitonin (21 amino acid)
3. N-terminal procalcitonin (57 amino acid)

Calcitonin is released when CALC-1 gene in thyroid C Cells is induced via elevated calcium, calcium gene related peptide, glucocorticoid, gastrin, glucagon or beta adrenergic stimulation. Normally no PCT is released into the circulation as all of it is converted to calcitonin. This results in very low levels of PCT in blood (less than 0.01ng/mL), but when PCT is released secondary to inflammation it becomes independent of above mechanism resulting in its increased level in the blood. PCT is produced in inflammation mainly by two alternative mechanisms; direct pathway induced by lipopolysaccharide (LPS) or other toxic metabolite from microbes and indirect pathway induced by various inflammatory mediators like IL-6, TNF-α, etc.

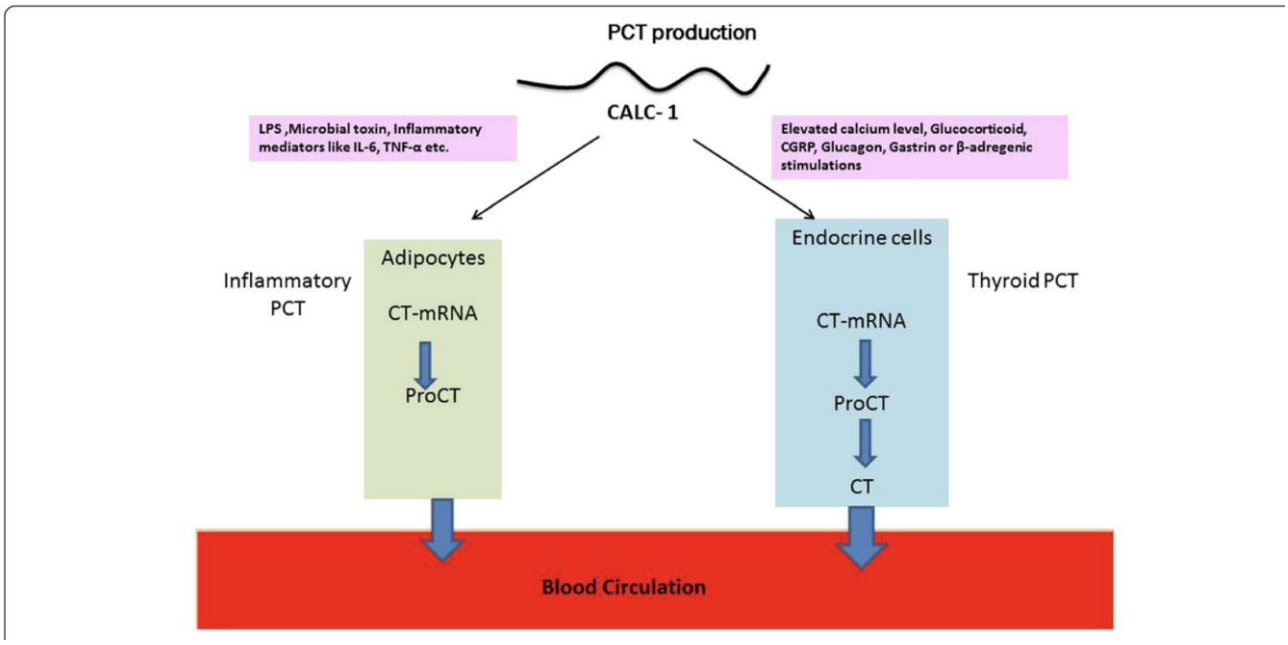


FIGURE 8 : FATE OF PCT DURING INFLAMMATION AND NORMAL CIRCULATION

- *Source – Vijayan, A.L., Vanimaya, Ravindran, S. et al. Procalcitonin: a promising diagnostic marker for sepsis and antibiotic therapy. j intensive care* ***5,*** *51 (2017).*

Medical uses of PCT

1. Sepsis - major use of PCT is in diagnosis and risk stratification of severe sepsis due to bacterial infection as it shows 90% and 91% sensitivity and specificity respectively.[114]
2. Respiratory illness - Use of PCT as a guide to initiate antibiotic therapy has drastically reduced overall misuse of antibiotics and is also helpful in initiating antibiotics in acute exacerbation of Bronchial asthma and chronic obstructive pulmonary disease.[115,116]
3. Cardiovascular disease - Use of PCT has been helpful in differentiating the cause of dyspnoea form infective aetiology or Severe inflammatory respiratory illness. It also has prognostic value in coronary artery disease and atherosclerosis as its level correlate with the severity.[117]

4. Meningitis - PCT is increased in bacterial meningitis and it serves as a guide to start antibiotic therapy.[118]
5. Gastrointestinal disease - Raised PCT is noticed in inflammatory bowel disease, abdominal abscess, bacterial enterocolitis etc.[119]
6. Kidney disease - PCT serve as a marker in diagnosis of pyelonephritis and other renal infections but its role is limited during dialysis as PCT is dialysed hence no specific cutoff value is established.[120]
7. MELD score can be corroborated by PCT together with CRP in Hepatitis.[121,122]
8. Septic arthritis - PCT has a sensitivity of 54% a specificity of 95% in diagnosis of Septic arthritis.[123]

Inflammation plays an important role in both ethology and progression of stroke, which signifies the importance of inflammatory molecules as both prognostic and therapeutic targets. Laura R et al. studied the role of inflammatory molecules in stroke. Post ischemia there is reduction of fuel to brain in the form of oxygen and glucose resulting impaired ATP production which ultimately results in bioenergetic failure and ionic imbalance. This leads to production of catabolic enzymes and increased production of reactive oxygen species which triggers the pro inflammatory genes, such as cytokines and chemokine by the brain tissue which is injured[124,125]. Acute ischemic stroke not only results in local inflammatory response but also results in triggering of systemic inflammatory response which results in increased levels of inflammatory molecules. Main inflammatory biomarkers which are studies in stroke include IL-1b/IL-1Ra, VCAM-1/VLA-4, ICAM-1, TNF-a, TGF-b, MMP-9/MMP-2, IL-6, IL-10, IL-4, P-Selectin, HMGB-1, ANXA-1[126].

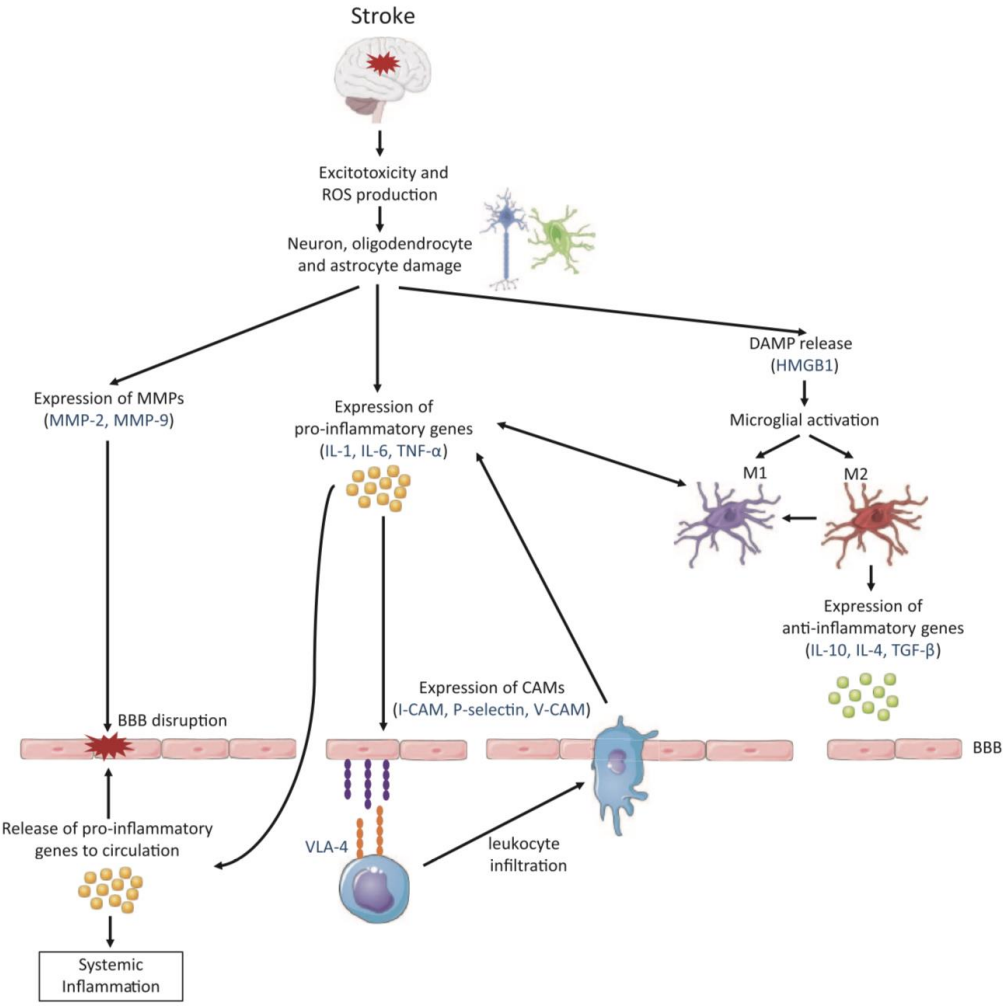


FIGURE 9 : THE INFLAMMATORY CASCADE FOLLOWING A STROKE

Source - Laura R, Alba S, Teresa G-B, et al. Inflammatory molecules might become both biomarkers and therapeutic targets for stroke management. Ther Adv Neurol Dis. 2018

Despite improvement in protocol for management of ischemic stroke has improved and there have been many inflammatory markers studied for diagnosis and prognostication appropriate diagnostic and prognostic tool is still missing. PCT was not widely used diagnostic marker as its major use was documented in its role in diagnosing bacterial infection. It was observed that PCT was elevated in post trauma signifying its role in inflammation secondary to tissue damage independent of infection. In a Chinese study by Da Tian et al first time demonstrated that there is proportional increase in serum PCT

levels in Acute ischemic stroke compared to controls in the lack of previous studies[127]. In another study Serum PCT was seen to be independently associated with increased 30 day mortality in acute ischemic stroke and its increased value carried poor prognosis. But further data regarding the role of PCT as diagnostic and prognostic marker in India is lacking.

Even though there are recent advances in diagnosing and managing acute stroke there is paucity of knowledge regarding its prognosis. Stroke is a debilitating disease and 2nd most common cause of mortality and disability. There is a need for biomarker which is easily available, cheap, validated and fast to stratify stroke patients. The aim of this study is to evaluate the role of Procalcitonin as prognostic biomarker in clinical practice.

MATERIALS AND METHODS

MATERIALS AND METHODS

SOURCE OF DATA

Patients admitted with history and clinical features suggestive of acute ischemic stroke admitted at R.L. Jalappa Hospital and Research Centre, a tertiary care hospital, Tamaka, Kolar.

Prospective observational study from JULY 2018 to MAY 2020

SAMPLE SIZE:

Regression methods - Sample size for correlation coefficient analysis

Sample correlation coefficient = .265

Population correlation coefficient = .55

Power (%) = 80

Alpha Error (%) = 5

Sided = 2

Required sample size = 68

INCLUSION CRITERIA:

1. Patients > 18 years of age with acute ischemic stroke presenting within 24 hours of onset

EXCLUSION CRITERIA:

1. Systemic Infections
2. Previous history of stroke
3. Head injury
4. Intracerebral bleed or hemmorhagic stroke

5. Patients with any evidence of heart failure, left ventricular systolic or diastolic dysfunction, myocardial infarction and acute coronary syndromes, hypertensive heart disease, hypertrophic cardiomyopathy, dilated cardiomyopathy, Atrial fibrillation and other arrhythmias, Valvular and other structural heart disease.
6. Collagen vascular diseases
7. Neoplastic diseases
8. Pregnancy
9. Patients who are thrombolysed as a part of treatment.
10. Patients who have lost follow up before 4 days

METHOD OF COLLECTION OF DATA

All patients with features suggestive of acute ischemic stroke presenting within 24 hours of onset to outpatient and inpatient department of General Medicine department, R.L Jalappa Hospital and research centre, Tamaka , Kolar were screened and patients who met the inclusion and exclusion criteria were recruited and followed up for three months.

Written and Informed consent were taken from all patients or responsible next of kin patient attendant. Acute ischemic stroke was diagnosed clinically and CT Brain or MRI Brain was done to confirm the diagnosis and rule out hemorrhagic stroke.

Detailed history was taken and a thorough clinical examination including detailed neurological examination was done in all patients and data collected were entered into the proforma. Signs of atherosclerosis like xanthelasma, arcus senalis, thickened vessels and History of risk factors for stroke like hypertension, diabetes, smoking, alcohol and tobacco chewing were obtained. Patients

with history of cardiac disorders, seizure, renal and liver diseases were excluded from the study. Based on history and examination patients were also classified into groups according to Oxfordshire community stroke project classification as total anterior circulation stroke, partial anterior circulation stroke, lacunar stroke and posterior circulation stroke.

Venous blood was collected EDTA vacutainers for hemotological and clot activated vacutainers for biochemical analysis. Routine investigations like complete blood count, blood urea, serum creatinine, serum electrolytes, random blood sugar, fasting and post prandial sugars, glycated Hba1c and lipid profile were done in all patients by using standard techniques. A RBS value of >200mg/dl with symptoms, FBS of > 126mg/dl, PPBS of >200 mg/dl or HbA1c of > 6.5 % was considered as diabetic. Patients with blood urea > 60mg/dl, serum creatinine > 1.5mg/dl and haemoglobin < 10 gm% were excluded from the study. Patients with serum cholesterol > 200mg/dl, LDL > 130mg/dl , triglyceride > 150mg/dl and HDL cholesterol < 40mg/dl were considered dyslipidemic.

All patients underwent standard 12 lead Electrocardiogram and standard 2D echocardiography and patients who had structural or valvular changes, ischemic changes, arrhythmias or atrial fibrillation were excluded from the study. Chest x ray was done in all patients. Carotid Doppler was done in 53 patients and patients were classified based on stenosis into low grade (1-49%), moderate grade (50-69%) and high grade (70-99%). None of the patients presented within window period and all patients received standard medical therapy.

Stroke deficit was calculated by National institutes of health stroke scale (NIHSS) on the day of admission, at 4^{th} day of admission, at end of one and 3^{rd} month. Patients were classified into groups based on NIHSS score.

GROUP	NIHSS SCORE	DESCRIPTION
1	0	NO STROKE
2	1-4	MINOR STROKE
3	5-15	MODERATE STROKE
4	16-20	MODERATE/SEVERE STROKE
5	21-42	SEVERE STROKE

Table 1 – CLASSIFICATION OF NIHSS

Stroke disability and functional outcome was measured using Barthel Index (BI) on 4^{th} day, at end of 1 months and 3 months following stroke. They were classified into group based on BI scores.

GROUP	BARTHEL INDEX	DESCRIPTION
1	<40	DEPENDENT
2	40-85	SLIGHTLY INDEPENDENT
3	>85	INDEPENDENT

Table 2 – CLASSIFICATION OF BARTHEL INDEX

2ml of venous sample was collected in EDTA vacutainers from all the patients at time of presentation. Blood was immediately centrifuged and serum sample was stored in EDTA vacutainers at -20^0 Celsius and serum assay of PCT was carried out by PCT fast test kit using AGAPPE - MISPA REVO immunofluorscence quantitative analyser by fluorescence immunochromatography.

Principle Test - The test uses an anti-human PCT monoclonal antibody conjugated with fluorescence latex and another anti-human PCT monoclonal antibody coated on the test line.

Total duration of assay is 15mins with measuring range 0.1-50.0 ng/mL and for normal individuals <0.1ng/mL is considered within normal limit based on 99th percentile concentration in normal individuals.

FIGURE 10 : IMMUNOFLURESCENCE QUANTITATIVE ANALYSER

STATISTICAL ANALYSIS

Data was entered into Microsoft excel data sheet and was analyzed using SPSS 22 version software. Categorical data was represented in the form of Frequencies and proportions. **Chi-square test** was used as test of significance for qualitative data. Continuous data was represented as mean and standard deviation. **Paired t test** is the test of significance for paired data such as before and after surgery for quantitative data.

Spearman's correlation was done to find the correlation between two quantitative variables.

Correlation coefficient (r)	Interpretation
0 - 0.3	Positive Weak correlation
0.3-0.6	Positive Moderate correlation
0.6-1.0	Positive Strong correlation
0 to (-0.3)	Negative Weak correlation
(-0.3) to (-0.6)	Negative Moderate Correlation
(-0.6) to – (1)	Negative Strong Correlation

Table 3 - CORRELATION COEFFICIENT AND ITS INTERPRETATION

Graphical representation of data: MS Excel and MS word was used to obtain various types of graphs such as bar diagram, Pie diagram and Scatter plots.

p value (Probability that the result is true) of <0.05 was considered as statistically significant after assuming all the rules of statistical tests.

Statistical software: MS Excel**,** SPSS version 22 **(**IBM SPSS Statistics, Somers NY, USA) was used to analyze data.

RESULTS

RESULTS

During the study period 79 patients with acute ischemic stroke were recruited among those 11 lost follow up and total of 68 patients were included in the study.

AGE DISTRIBUTION

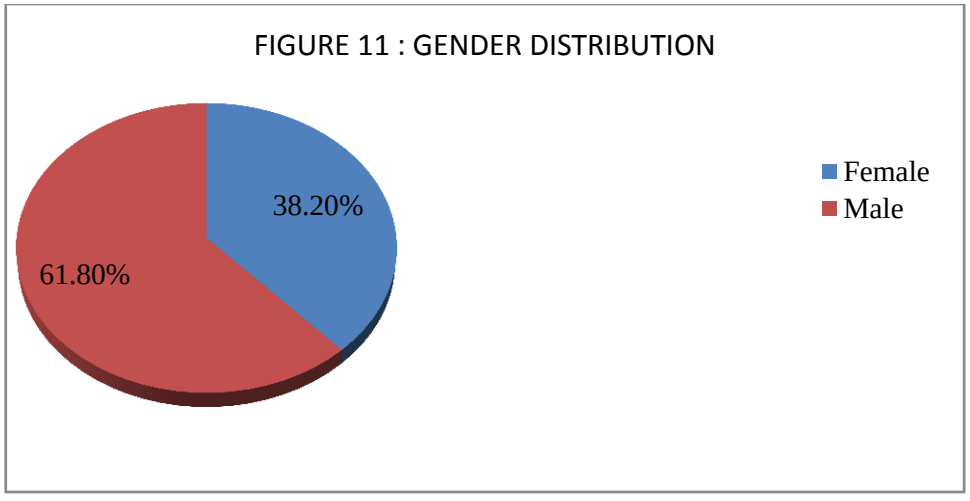
Study subjects were in age group 18-80 years with mean age of subjects being 62.28 ± 12.303 years. Majority of subjects were in the age group >70 years. No subject was below 40 years of age group in our study

		Frequency	Percentage
Age	40 to 49 years	14	20.6%
	50 to 59 years	11	16.2%
	60 to 69 years	16	23.5%
	>70 years	27	39.7%
	Total	68	100.0%

Table 4 – DISTRIBUTION OF AGE

GENDER DISTRIBUTION

Among the 68 patients studied 42(61.8%) were males and 26(38.2%) were female. Ischemic stroke was more common in Males compared to Females.



CLINICAL FEATURES

In this study, the most common presenting complaint was acute onset weakness of one half of the body (hemipaeresis) or a single limb (monopaeresis) in 53 patients followed by loss of consciousness in 17, giddiness in 12, speech deficit in 9, headache in 5, vomiting in 6, altered sensorium in 7, deviation of angle of mouth in 24 patients and sensory disturbance in 1 patient.

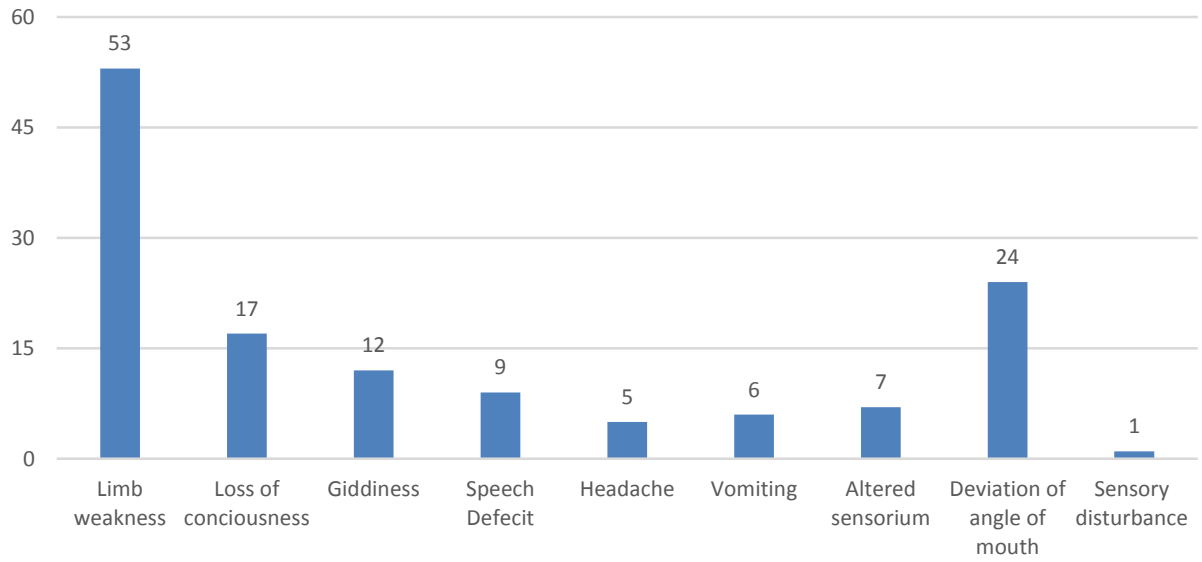


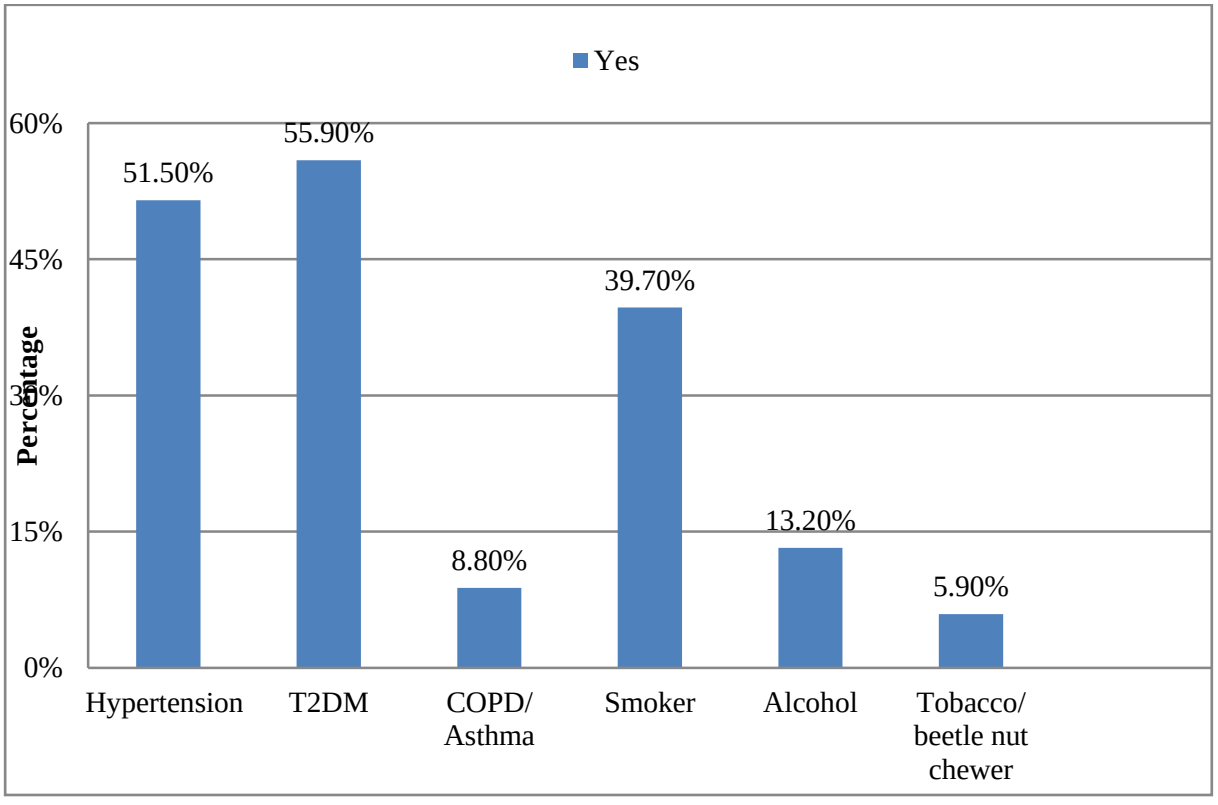
FIGURE 12 : DISTRIBUTION OF PRESENTING COMPLAINTS

RISK FACTORS

In the study 35 (51.5%) had Hypertension, 38 (55.9%) had Type 2 Diabetes Mellites, 6 (8.8%) had COPD/Asthma, 27 (39.7%) were smokers, 9 (13.2%) were Alcoholics, and 4 (5.9%) were Tobacco / beetle nut chewers.

	No		Yes	
	Count	%	Count	%
Hypertension	33	48.5%	35	51.5%
T2DM	30	44.1%	38	55.9%
COPD/ Asthma	62	91.2%	6	8.8%
Smoker	41	60.3%	27	39.7%
Alcohol	59	86.8%	9	13.2%
Tobacco/ beetle nut chewer	64	94.1%	4	5.9%

TABLE 5 – DISTRIBUTION OF RISK FACTORS



In the study out of 68 patients 34 (53.1%) had signs of atherosclerosis in the study like thickened peripheral vessels and arcus senalis. 2 patients had clubbing .The average duration of hospital stay was 8.11±5.17 days. The average GRBS at presentation was 178.6mg/dl average systolic blood pressure was 146.32±26.67 mmHg and diastolic blood pressure was 86.91±13.07 mmHg. The average pulse rate was 90.76±12.06 beats/min and respiratory rate was 19.10±3.54 cycles/minute and saturation at room air was 95.53±2.56 %. All patients were afebrile at presentation.

	Mean	**Std. Deviation**
PULSE	90.76	12.06
SBP	146.32	26.67
DBP	86.91	13.07
TEMPERATURE	97.65	.49
RR	19.10	3.54
SPO2	95.53	2.56
GRBS	178.6	97.37

TABLE 6 – CHARRACTERISTICS OF VITALS IN PATIENTS

Cardiovascular, respiratory and abdominal system examinations were normal. The average Glasgow coma scale at presentation was 11.97±3.85. 14 patients had a score of <8(severe), 19 had GCS score between 9-13(Moderate) and 35 patients had >13 (mild). 32 patients were either unconscious, drowsy or had altered sensorium at time of presentation. 2 patients had aphasia on examination.

CNS FINDINGS IN THE STUDY SUBJECTS

		Count	%
HMF	Aphasia	2	2.9%
	Drowsy	13	19.1%
	Normal	34	50.0%
GCS	Not Oriented	7	10.3%
	Unconscious	12	17.6%
	Severe (3 to 8)	14	20.6%
	Moderate (9 to 13)	19	27.9%
	Mild (14 to 15)	35	51.5%
Pupils	BERL	67	98.5%
	Post Cataract	1	1.5%

TABLE 7 – DISTRIBUTION OF CNS FINDINGS IN SUBJECTS

Cranial nerve examination showed, 17.6% had Left UMN 7th facial palsy, 8.8% had Right UMN 7TH facial N Palsy and Gaze palsy respectively. 3 had Nystagmus, 1.50% had dysarthria and 11 patients had cerebellar dysfunction. Plantar response on Right side, 35.3% had extensor, 45.6% had flexor, 17.6% had mute and 1.5% had withdrawal reflex. On Left side, 35.3% had extensor, 41.2% had flexor, 22.1% had mute and 1.5% had withdrawal reflex. 53 patients had either hemiparesis or mono paresis on either side. In the study on Right side, 1 had Hypotonia and 2 had spasticity and on left side, 2 had Hypotonia and 7 had spasticity. 2 patients had sensory disturbance, 11 patients had cerebellar dysfunction like impaired dysdiodokinesia, impaired finger-finger or finger nose test and impaired knee heel or tandem walking and in 20 patients sensory examination and cerebellar examination could not be completed because of altered sensorium.

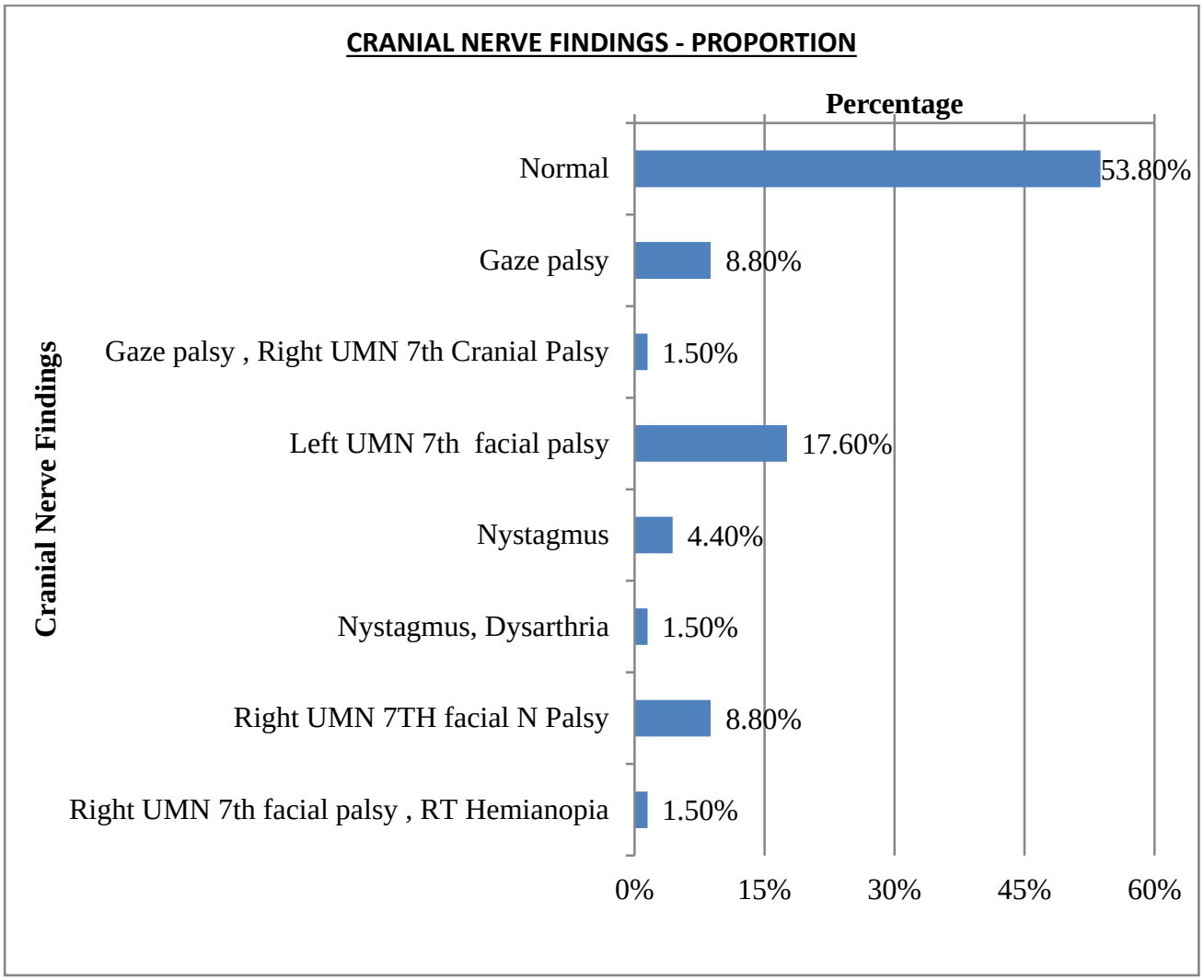


FIGURE 14 : DISTRIBUTION OF CRANIAL NERVE FINDINGS IN SUBJECTS

All patients had normal 12 lead ECG with No ST T changes or arrhythmias. 2D echocardiography and chest x ray was normal in all patients. Hematological and biochemical laboratory tests were done and mean and standard deviation calculated. 4 Patients had hyperkalemia and 27 patients had hyponatremia. Patients with deranged renal function and low haemoglobin were excluded from the study.

	Mean	SD
HB (gm%)	13.38	2.36
PCV (%)	39.0	6.0
MCV (fl)	81.53	7.65
TOTAL COUNTS(Thousand/mm^3)	13.39	5.2
PLATELETS (Thousand/mm^3)	270.13	97.05
BLOOD UREA (mg/dl)	31.3	10.55
SERUM CREATININE (mg/dl)	0.86	0.6
SODIUM (meq/L)	134.75	5.43
POTASSIUM (meq/L)	4.13	0.85
RBS (mg/dl)	156.53	86.23
FBS (mg/dl)	153.897	99.72
PPBS (mg/dl)	193.82	99.1
HBA1C	8.075	2.955
SERUM CHOLESTEROL (mg/dl)	184.04	42.35
TRI GLYCERIDES(mg/dl)	142.35	58.62
HDL CHOLESTROL(mg/dl)	41.54	9.99
LDL CHOLESTEROL(mg/dl)	111.35	37.59s

TABLE 8 – BIOCHEMICAL PARAMETERS

Carotid Doppler was done in 54 patients. 16 patients had normal study. Atherosclerotic changes were present in rest of the patients. 2 patients had atherosclerotic changes of vertebral artery. Increased intimal thickness was seen in 29 patients. 11 patients had plaques in internal carotid artery.

GRADE	STENOSIS	NUMBER OF PATIENTS
LOW	<50%	19
MODERATE	50-69%	6
HIGH	>70%	12

TABLE 9 – CAROTID DOPPLER CLASSIFICATION

CT or MRI brain was done in all patients to confirm the diagnosis of stroke and rule out hemorrhagic stroke. Lacunar strokes were present in 2 patients. 12 patients had cerebellar involvement, 3 patients had hyperdense MCA. 2.9% had ICA involvement, 19.1% had ACA involvement, 75% had MCA involvement, 20.6% had PCA involvement, 1.5% had SICA involvement and 4.4% had vertebral artery involvement.

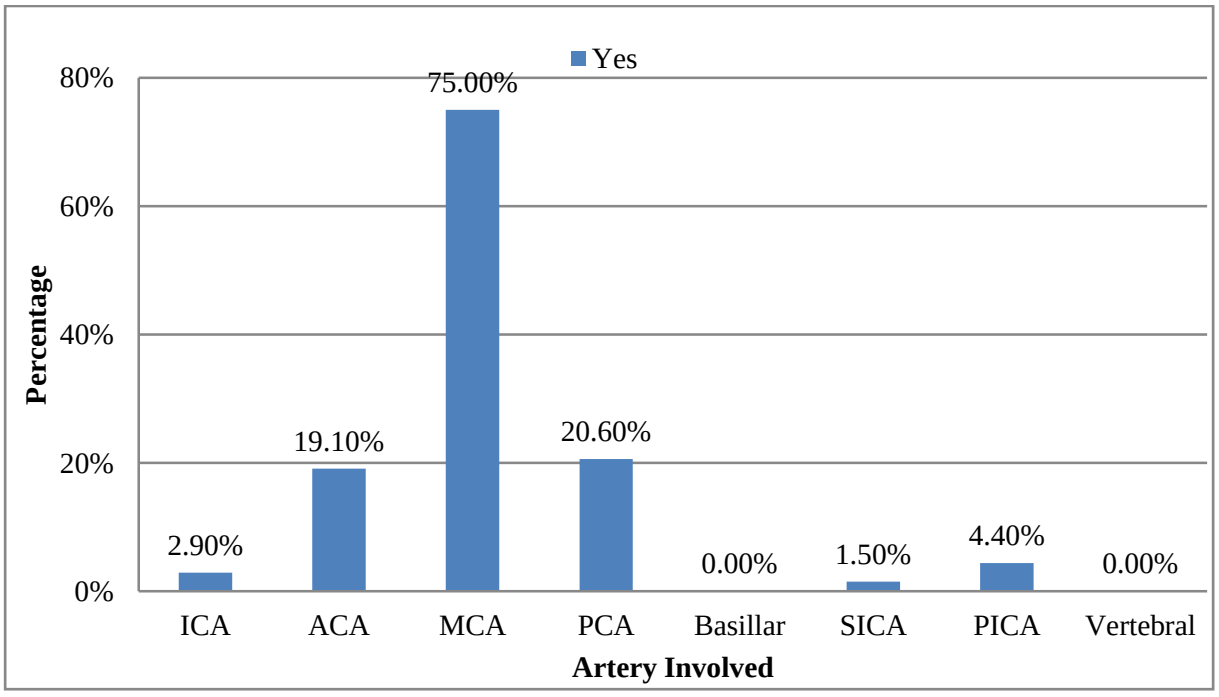


FIGURE 15 : DISTRIBUTION OF INVOLVED ARTERY

Patients were also classified as per Oxfordshire Community Stroke Project(OCSP). Total Anterior Circulation Infarcts (TACI) were seen in 23 (33.8%) of patients, 27 (39.7%) had Partial Anterior Circulation Infarcts (PACI), Posterior Circulation Infarct (POCI) was seen in 11 (16.2%) and Lacunar syndromes (LS) in 8 (11.8%)

	No		Yes	
	Count	%	Count	%
TACS	45	66.2%	23	33.8%
PACS	41	60.3%	27	39.7%
LAC	60	88.2%	8	11.8%
POC	57	83.8%	11	16.2%

TABLE 10 - OXFORDSHIRE COMMUNITY STROKE PROJECT DISTRIBUTION AMONG SUBJECTS

Average NIHSS at the time of admission was 13.235±7.4 there was no significant gender variation. Average serum PCT at the time of admission was 5.723±9.10 this was compared with the NIHSS at day 0, day 4 and Barthel Index day 0 and day 4 and was found to have significant correlation.

Correlations

			Procalcitonin levels at Day 0	**NIHSS score at Day 0**	**Barthel Index at Day 4**
Spearman's rho	**Procalcitonin levels at Day 0**	Correlation Coefficient	1.000	0.697**	-0.729**
		P value	.	<0.001*	<0.001*
		N	64	64	64

**. Correlation is significant at the 0.01 level (2-tailed).

TABLE 11 - CORRELATION BETWEEN PROCALCITONIN LEVELS AT DAY 0 WITH NIHSS SCORE AT DAY 0 AND BARTHEL INDEX AT DAY 4

In the study there was significant positive correlation between Procalcitonin levels at Day 0 and NIHSS score at Day 0. I.e. on Day 0 with increase in Procalcitonin levels there was increase in NIHSS score and vice versa.

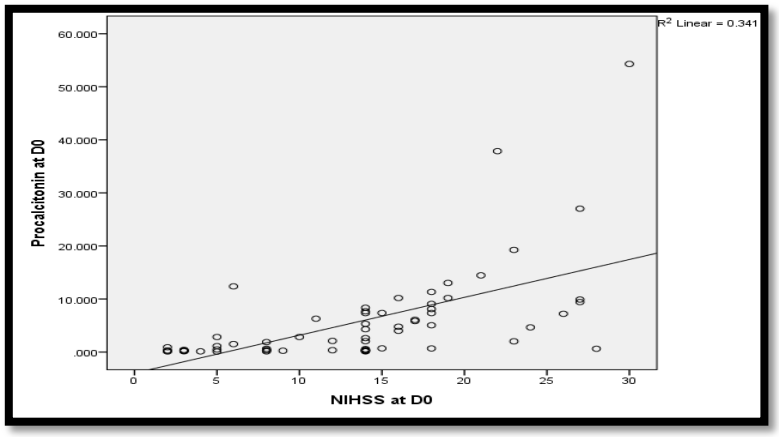


FIGURE 16: SCATTER PLOT SHOWING POSITIVE CORRELATION BETWEEN PROCALCITONIN LEVELS ON DAY 0 WITH NIHSS AT DAY 0

There was significant negative correlation between Procalcitonin levels at Day 0 and Barthel Index at Day 4. I.e. with increase in Procalcitonin levels on day 0 there was decrease in Barthel Index at Day 4 and vice versa.

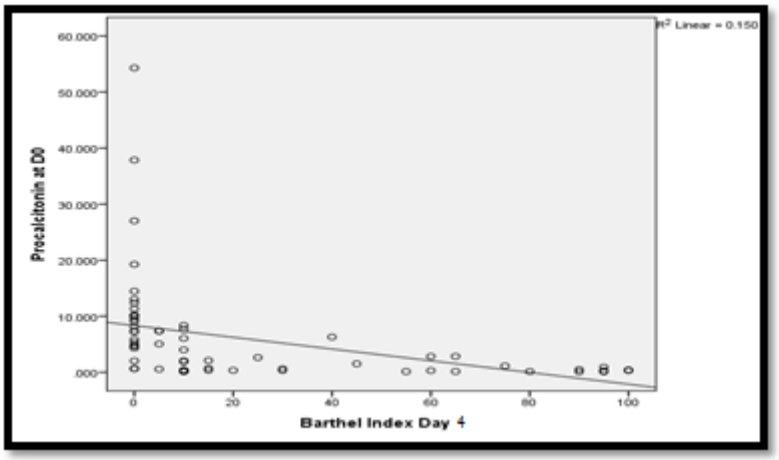


FIGURE 17: SCATTER PLOT SHOWING NEGATIVE CORRELATION BETWEEN PROCALCITONIN LEVELS ON DAY 0 WITH BARTHEL INDEX AT DAY 4

		N	Mean	SD	Median	P value#
Barthel Index	Barthel Index D 0	68	19.56	33.776	0	
	Barthel Index Day 4	68	25.51	33.593	10	<0.001*
	Barthel Index at Discharge	66	28.48	33.305	10	<0.001*

TABLE 12 - BARTHEL INDEX COMPARISON B/W DAY 0 AND DAY 4, AT DISCHARGE

Wilcoxon Signed rank test

In the study at Day 0, Median Barthel Index was 0, at Day 4, Median Barthel Index was 10 and at discharge, Median Barthel Index was 10. There was significant increase in Median Barthel Index at Day 4 and at discharge compared to Day 0 value.

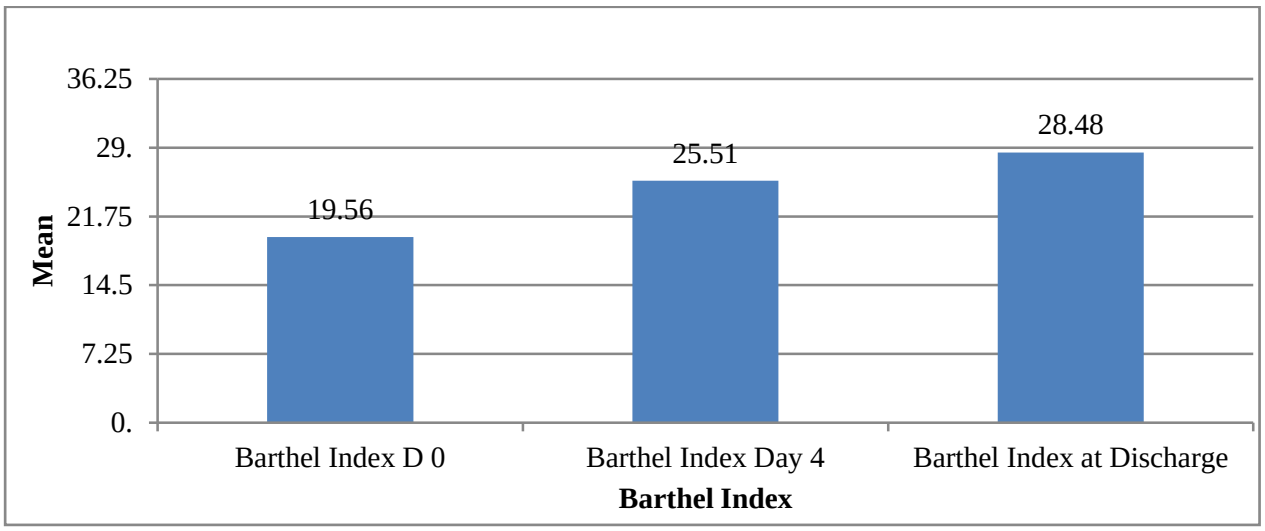


FIGURE 18: BAR DIAGRAM SHOWING BARTHEL INDEX COMPARISON B/W DAY 0 AND DAY 4, AT DISCHARGE

		N	Mean	SD	P value	Deaths
NIHSS	D0	60	11.85	6.525		
	D4	52	11.58	6.634	0.312	8
	Discharge	49	10.44	5.846	0.022*	8+3 = 11
	1 Month	46	8.69	5.525	<0.001*	11+5 = 16
	3 Month	40	7.21	5.099	<0.001*	16 + 4 = 20

TABLE 13 - NIHSS SCORE AT DAY 0 AND COMPARISON WITH DAY 4, AT DISCHARGE, 1 MONTH AND 3 MONTHS SCORE

In the study NIHSS score at Day 0 was 11.85 ± 6.525, at Day 4 was 11.58 ± 6.634, at discharge was 10.44 ± 5.846, at 1 month was 8.69 ± 5.525 and at 3 months was 7.21 ± 5.099.

There was significant decrease in NIHSS score at discharge, 1 Month and 3 Month compared to NIHSS score. At Day 4 there was no significant decrease compared to Day 0 value.

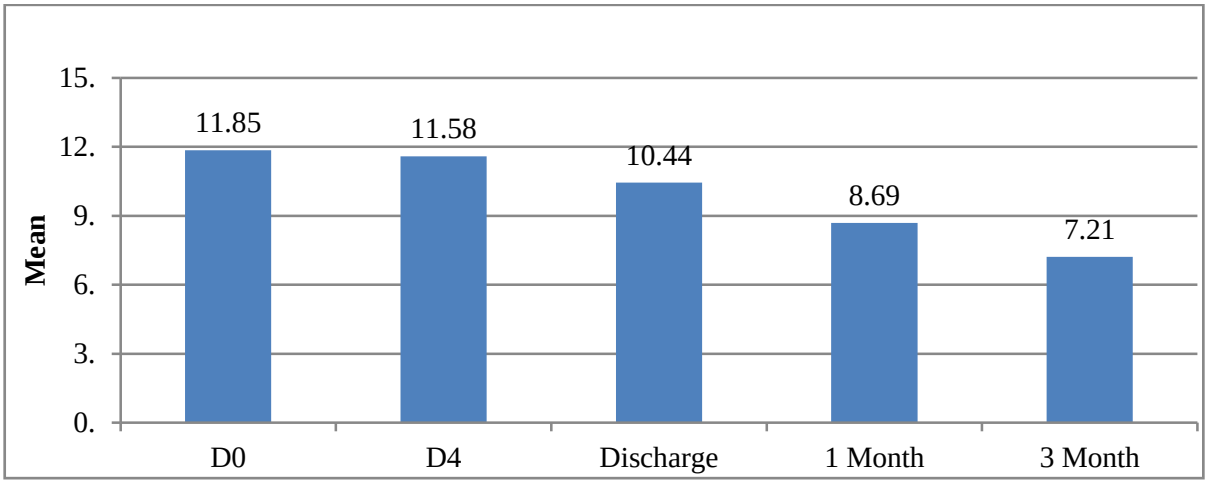


FIGURE 19: BAR DIAGRAM SHOWING NIHSS SCORE AT DAY 0 AND COMPARISON WITH DAY 4, AT DISCHARGE, 1 MONTH AND 3 MONTHS SCORE

		Day 0						P value
		<5 (Mild)		5 to 20 (Moderate)		>20 (Severe)		
		Count	%	Count	%	Count	%	
Day4	<5 (Mild)	10	100.0 %	4	8.5%	0	0.0%	<0.001 *
	5 to 20 (Moderate)	0	0.0%	41	87.2%	0	0.0%	
	>20 (Severe)	0	0.0%	0	0.0%	5	45.5%	
	Death	0	0.0%	2	4.3%	6	54.5%	
Discharge	<5 (Mild)	10	100.0 %	4	8.5%	0	0.0%	<0.001 *
	5 to 20 (Moderate)	0	0.0%	41	87.2%	1	9.1%	
	>20 (Severe)	0	0.0%	0	0.0%	1	9.1%	
	Death	0	0.0%	2	4.3%	9	81.8%	
1 Month	<5 (Mild)	10	100.0 %	6	12.8%	0	0.0%	<0.001 *
	5 to 20 (Moderate)	0	0.0%	35	74.5%	1	9.1%	
	>20 (Severe)	0	0.0%	0	0.0%	0	0.0%	
	Death	0	0.0%	6	12.8%	10	90.9%	
3rd Month	<5 (Mild)	9	90.0%	11	23.9%	0	0.0%	<0.001 *
	5 to 20 (Moderate)	0	0.0%	26	56.5%	0	0.0%	
	>20 (Severe)	0	0.0%	0	0.0%	0	0.0%	
	Death	1	10.0%	9	19.6%	11	100.0 %	

TABLE 14: COMPARISON OF DAY 0 NIHSS GRADE WITH DAY 4, DISCHARGE, 1 MONTH AND 3 MONTH GRADE

In the study there was significant association between Day 0 NIHSS grade with Day 4, Discharge, 1 Month and 3rd Month. There was significant variation of NIHSS as it decreased significantly over the course as there was significant improvement among the patients as shown by decrease in mean NIHSS.

Among the 68 patients included in the study 8 patients died with 4 day of admission and total of 11, 16 death at 1 month and 20 deaths at 3rd month. Higher PCT levels was associated with poor mortality benefit as there was significantly higher mortality (p= <0.001) in patients with higher PCT levels. Receiver operating characteristic (ROC) of NT PROBNP at Day 0 in predicting death (P value <0.001, area under the curve 0.995, 95% confidence interval 0.984 to 1.000)

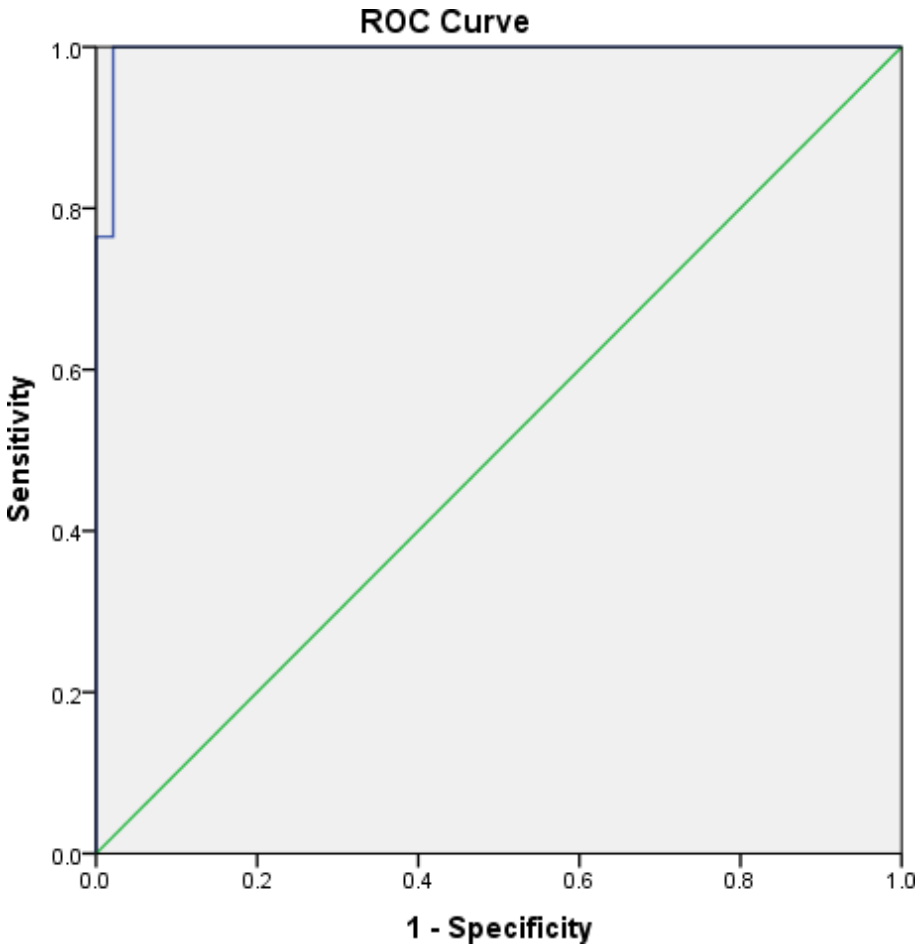


FIGURE 20 : ROC CURVE OF PCT IN PREDICTING DEATH

Receiver operating characteristic (ROC) of NIHSS at Day 0 in predicting death (P value <0.001, area under the curve 0.926, 95% confidence interval 0.841 to 1.000).

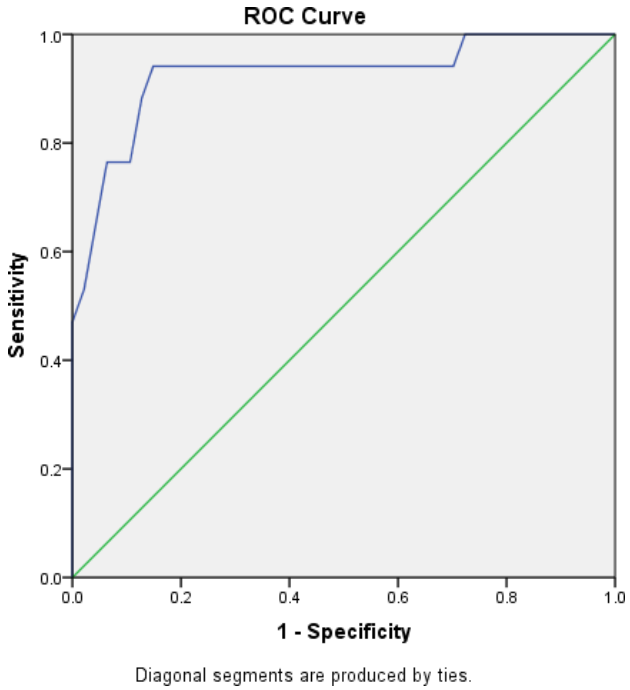


FIGURE 21 : ROC CURVE OF NIHSS IN PREDICTING DEATH

Area under the ROC curve above 0.8 indicates fairly good prediction. Area under the ROC curve for both NIHSS and PCT on admission is almost similar with 0.926 and 0.995 suggesting they can predict mortality. The sensitivity for both NIHSS and PCT was 94.1% but PCT had a higher specificity of 97.9% compared to 86% of NIHSS;

Area under the ROC curve for PCT was 0.946 in predicting dependency at 3 months with sensitivity of 90% and specificity of 81% and NIHSS failed to predict dependency hence PCT is better predictor of mortality and functional disability compared to NIHSS.

DISCUSSION

DISCUSSION

The aim of this study was to assess the levels of serum Procalcitonin in patients presenting with acute ischemic stroke and to study its association with severity and short term outcome on 7th day assessed by Barthel index. This was accomplished by taking 68 patients with acute ischemic stroke who presented within the 24 hours of the onset of symptoms in whom serum Procalcitonin levels were checked at the time of presentation and severity at the time of presentation was assessed using NIHSS scale. The patient was followed up for seven days and the outcome at 4th day was recorded using Barthel index. The major findings of this study are: 1. Serum Procalcitonin levels were significantly higher in patients who had severe stroke assessed using NIHSS i.e more the NIHSS higher was the serum Procalcitonin levels, 2. Higher serum Procalcitonin levels at the time of presentation was associated with lower Barthel index at day 4 i.e poor outcome, 3. High serum Procalcitonin levels at the time of presentation was associated with higher mortality incidence at 4th day and 1 month.

Prior to this study, four studies were carried out where serum Procalcitonin was studied in relation with acute ischemic stroke as both diagnostic marker and short term outcome predictor. The results of these studies are summarised in the table 14. Overall two studies showed association between serum Procalcitonin with severity and short term outcome and one study where serial monitoring of serum Procalcitonin was done showed no association between the outcome and serial measurement. However the way of association of serum Procalcitonin in stroke is not same in the three studies. Ling Yan et al. 2020 showed that Procalcitonin is a significant independent prognostic marker of 30 day mortality after one set of acute ischemic stroke. Da Tian et al. 2015 study demonstrated that serum Procalcitonin levels at admission was associated with severity and volume of stroke. Both of these were Chinese based studies and no Indian studies are yet available. Another study Myakis S et al.

2004 where serial monitoring of serum Procalcitonin were done in first ever stroke patients showed no significant association. With this utility of serum procalcitonin levels in assessing severity and short term outcome still remains questionable. The result of this study is in agreement with the two Chinese based studies as there was significant association between serum procalcitonin levels with short term outcome and severity of patients presenting with acute ischemic stroke.

Population	**S.PCT (median)**	**Severity**	**Outcome at Day 4**	**Reference**
n=422	1.04ng/ml	p=<0.001	-	Da Tian et al.[127]
n=748	1.37ng/ml	p-<0.001	p=<0.001	Ling Yan et al.[128]
n=378	0.88ng/ml	p=<0.001	p=<0.001	Wen Jing Deng et al.[129]
n=68	2.36ng/ml	p=<0.001	p=<0.001	Current study

TABLE 15 – COMPARISON OF SIMILAR STUDY WITH CURRENT STUDY

In our study higher levels of serum procalcitonin was significantly associated with the severity of stroke at the time of presentation as assessed by NIHSS at the presentation and it had a significant positive correlation with the level of serum procalcitonin. In various literatures serum procalcitonin was a significant marker for diagnosis of bacterial infection later it was showed serum procalcitonin levels elevation independent of bacterial infection as

it was found in trauma cases. In our study we conclude that serum procalcitonin as a significant marker in predicting severity of acute ischemic stroke as defined by NIHSS scale as a result of stroke related brain damage resulting in increase in inflammatory markers like Serum procalcitonin. Our study also found that serum procalcitonin levels at the time of admission had negative correlation with the Barthel index I.e higher the serum procalcitonin levels lower the Barthel index which shows poorer functional outcome. It was not a surprising result as the more severe stroke via higher NIHSS scale means more damage to brain which in-turn signifies poor outcome. Serum procalcitonin levels were significantly higher in patients who died as a result of acute ischemic stroke as compared to patients who survived in our study which signifies serum procalcitonin not only predicts poor functional outcome but it also is an independent marker of mortality prediction in first ever acute ischemic stroke.

This study has inherent limitations, as it requires more extensive work on interpretation of the procalcitonin levels as it is expected that the levels of inflammatory markers at the site of lesion in brain may no be same as that found in the peripheries. Serial monitoring of serum procalcitonin levels were not done which denies us the opportunity to study the duration for which its level remain elevated in the blood and its changes during the coarse of illness. The volume of infarct with the cause of mortality was not evaluated in the study.

Further studies are required with involvement of larger population with multicenter involvement to study further role of serum procalcitonin as an inflammatory marker and its role in acute ischemic stroke to assess the severity and short term outcome along with other inflammatory markers before considering serum procalcitonin as a routine biomarker in acute ischemic stroke.

CONCLUSION

CONCLUSION

Serum procalcitonin is significantly elevated in patients after acute ischemic stroke and it is strongly associated with the severity and functional outcome at 4th day and it can predict all cause mortality in patients presenting with acute ischemic stroke.

SUMMARY

- 68 cases of acute ischemic stroke were studied and followed up until discharge or 4th day whichever is the later.
- Majority of patients presenting with acute ischemic stroke belongs to age group >70 years with male predominance.
- Diabetes was the most common risk factor in this study followed by hypertension.
- GCS was significantly associated with severity and short term outcome. Low GCS was associated with significantly higher mortality.
- MCA was the most common artery involved in acute ischemic stroke and 13 patients had significant stenosis of ICA >70%
- PACI was the most common type of stroke in our study
- Average NIHSS on the day of admission 13.235$\pm$7.4 and among deceased it was 21.26$\pm$5.88. NIHSS at the time of admission was significantly associated with the short term functional outcome, Barthel index and all cause mortality.
- The median Procalcitonin at the time of admission was 5.723+9.1 ng/ml. It was significantly associated with the NIHSS at the time of presentation as higher serum Procalcitonin represented more severe stroke. It was also significantly associated with Barthel index and GCS as it showed negative correlation with Barthel index where higher serum procalcitonin levels signifying poor functional outcome and it was also an independent predictor of all cause mortality in patients presenting with acute ischemic stroke.

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ANNEXURES

PROFORMA FOR DATA COLLECTION

IP No.:

Date:

Serial No:

Name:

Age:

Gender:

Occupation:

Date of admission:

Date of discharge:

Address with Phone No:

Chief Complaints:

Past History:

Drug/ Treatment History:

Personal History:

General physical examination(at presentation)

PR

BP

Temp

Respiratory rate

SpO2:

GRBS:

Pallor:

Icterus:

Cyanosis:

Clubbing:

Oedema:

Systemic Examination

CVS:

RS:

PA:

CNS:

Diagnosis:

Duration of Hospital Stay:

Investigations:

- Complete Hemogram
- Chest X Ray
- A Standard 12 Lead ECG
- Blood Urea
- Serum Creatinine
- Serum Electrolytes NA+ K+ CA+
- FBS PPBS RBS HBA1CS
- URINE ROUTINE
- CT SCAN BRAIN PLAIN/MRI BRAIN
- SERUM PROCALCITONIN
- CAROTID DOPPLER
- LIPID PROFILE:
- OTHERS:
- NIHSS SCORE AT PRESENTATION:
- BARTHEL SCORE ON 4^{TH} DAY:

PATIENT INFORMATION SHEET

TITLE: "STUDY OF SERUM PROCALCITONIN LEVELS IN ACUTE ISHCHEMIC STROKE IN RELATION WITH ITS SEVERITY AND SHORT TERM OUTCOME".

Principal Investigator: Dr. Dhruvanandan K

Post Graduate, Department of Medicine

SDUMC Kolar

Ph No 8095995698

E-mail id: dhruva.nandan.dn@gmail.com

Study site: R.L Jalappa Hospital and Research Center attached to Sri Devaraj Urs Medical College, Tamaka, Kolar.

Aim of the study: The aim of this study is to study the Serum Procalcitonin levels in acute ischemic stroke in relation to its severity and short term outcome.

Voluntary Participation: Your participation in this study is entirely voluntary. There is no compulsion to participate in this study. You will be in no way affected if you do not wish to participate in the study. You are required to sign only if you voluntarily agree to participate in this study. Further you are at a liberty to withdraw from the study at any time. We assured that your withdrawal will not affect your treatment by the concerned physician in any way.

Procedure: We will take 2ml of blood from your arm with a syringe. This blood will be tested for Serum Procalcitonin levels.

Confidentiality: All information collected from you will be strictly confidential and will not be disclosed to anyone except if it is required by the law. This information collected will be used only for research. This information will not reveal your identity. We would not compel you any time during this process; also we would greatly appreciate your cooperation to the study. We would like to get your consent to participate in the study. For any information you are free to contact investigator. This study has been approved by the Institutional Ethics Committee & has been started only after their formal approval. The sample collected will be stored in the institute and I request you to permit us to store and use this sample for any future study.

Subject name: (Parents / Guardians name)

DATE: **SIGNATURE /THUMB**

INFORMED CONSENT FORM

STUDY NUMBER:

SUBJECT'S NAME:

HOSPITAL NUMBER:

AGE:

If you agree to participate in the study we will collect information (as per proforma) from you or a person responsible for you or both. We will collect the treatment and relevant details from your hospital record. This information collected will be used for only dissertation and publication. This study has been reviewed by the institutional ethical committee. The care you will get will not change if you don't wish to participate. You are required to sign/ provide thumb impression only if you voluntarily agree to participate in this study.

I understand that I remain free to withdraw from the study at any time and this will not change my future care. I have read or have been read to me and understood the purpose of the study, the procedure that will be used, the risk and benefits associated with my involvement in the study and the nature of information that will be collected and disclosed during the study. I have had the opportunity to ask my questions regarding various aspects of the study and my questions are answered to my satisfaction. I, the undersigned agree to participate in this study and authorize the collection and disclosure of my personal information for publication.

Subject name:

(Parents / Guardians name)

DATE: **SIGNATURE /THUMB IMPRESSION**

ರೋಗಿಯ ಮಾಹಿತಿ ಮತ್ತು ಸಮ್ಮತಿ ಪತ್ರ

ಸಂಶೋಧಕರ ಹೆಸರು: ಡಾ॥ ಕೆ. ಧ್ರುವನಂದನ್., ಸ್ನಾತಕೋತ್ತರ ಪದವೀಧರ., ಡಿಪಾರ್ಟ್‌ಮೆಂಟ್ ಆಫ್ ಜನರಲ್

ಮೇಡಿಷನ್.,ಎಸ್ ಡಿ ಯು ಎಂ ಸಿ ಕೋಲಾರ್

ಫೋನ್ ನಂ :8095995698

ಪಾಲ್ಗೊಳ್ಳುವವರ ಹೆಸರು :

ಕ್ರ.ಸಂ :

ಶ್ರೀ/ಶ್ರೀಮತಿ ----------ಆದ ನಾನು ಆರ್ ಎಲ್ ಜಾಲಪ್ಪ ಆಸ್ಪತ್ರೆಯಲ್ಲಿ ನಡೆಸಲಾಗುತ್ತಿರುವ ಅಧ್ಯಯನ **"ತೀವ್ರವಾದ ಸ್ಟ್ರೋಕ್ ಸೀರಮ್ ಈಔಅಂಐಅಐಖಿಔಓಐಓ"** ದಲ್ಲಿ ನನ್ನನ್ನು ಸೇರಿಸಲ್ಪಡಲಾಗುವುದು ಎಂದು ನನಗೆ ಅರ್ಥವಾಗುವ ಭಾಷೆಯಲ್ಲಿ ವಿವರಿಸಲಾಗಿದೆ. ಈ ಸಂಶೋಧನಾ ಅಧ್ಯಯನದಲ್ಲಿ ಪಾಲ್ಗೊಳ್ಳಲು ನನ್ನನ್ನು ಆಹ್ವಾನಿಸಲಾಗಿದೆ. ಈ ದಾಖಲೆಯಲ್ಲಿರುವ ಮಾಹಿತಿಯು ಅಧ್ಯಯನದಲ್ಲಿ ಪಾಲ್ಗೊಳ್ಳಬೇಕೇ ಅಥವಾ ಬೇಡವೇ ಎಂಬುದನ್ನು ನಿರ್ಧರಿಸಲು ನನಗೆ ನೆರವಾಗುವುದು.

ಪ್ರಧಾನ ಸಂಶೋಧಕರೊಂದಿಗೆ ನಾನು ಈ ಅಧ್ಯಯನಕ್ಕೆ ಸಂಬಂಧಿಸಿದಂತೆ ನನ್ನ ಅನುಮಾನಗಳನ್ನು ಸ್ಪಷ್ಟಪಡಿಸಿಕೊಂಡಿದ್ದೇನೆ. ಈ ಅಧ್ಯಯನದಲ್ಲಿ ಪಾಲ್ಗೊಳ್ಳುವಂತೆ ನನಗೆ ಸೂಚಿಸಲಾಗಿದೆ. ಏಕೆಂದರೆ ನಾನು ಅರ್ಹತಾ ಮಾನದಂಡಗಳನ್ನು ಪೂರೈಸುತ್ತೇನೆ. ನನ್ನ ರಕ್ತದ ಮಾದರಿಯನ್ನು ಗೊತ್ತುಪಡಿಸಿದ ಪರೀಕ್ಷೆಗಳಿಗೆ ನಿರ್ವಹಿಸಲು ನಾನು ಡಾ॥ ಕೆ. ಧ್ರುವನಂದನ್ ಅವರನ್ನು ವಿನಂತಿಸುತ್ತೇನೆ ಮತ್ತು ಅವರಿಗೆ ಅಧಿಕಾರವನ್ನು ನೀಡುತ್ತೇನೆ. ಕೆಳಗಿನ ನನ್ನ ಸಹಿಯು ಅರ್ಹಆರೋಗ್ಯ ವೃತ್ತಿಪರರಿಂದ ಪರೀಕ್ಷೆಯ ಅನುಕೂಲಗಳು, ಅಪಾಯಗಳು ಮತ್ತು ಮಿತಿಗಳನ್ನು ನನ್ನ ತೃಪ್ತಿಗೆ ವಿವರಿಸಲಾಗಿದೆ ಎಂದು ನನ್ನ ಅಂಗೀಕಾರವನ್ನು ರೂಪಿಸುತ್ತದೆ ಮತ್ತು ಭಾಗವಹಿಸುವಿಕೆ

ಸಂಪೂರ್ಣವಾಗಿ ಸ್ವಯಂಪ್ರೇರಿತವಾಗಿರುತ್ತದೆ ಹಾಗೂ ಮಾದರಿ ಸಂಗ್ರಹಣೆಗೆ ಯಾವುದೇ ಹಣಕಾಸಿನ ಪಾವತಿಯಿಲ್ಲ. ಎಲ್ಲಾ ಪರೀಕ್ಷಾ ಫಲಿತಾಂಶಗಳನ್ನು ವೈದ್ಯಕೀಯ ಗೌಪ್ಯತೆಯೊಂದಿಗೆ ಪರಿಗಣಿಸಲಾಗುತ್ತದೆ ಮತ್ತು ಕಾನೂನಿನ ಅಗತ್ಯವಿದ್ದರೆ ಅದನ್ನು ಹೊರತುಪಡಿಸಿ ಯಾವುದೇ ಹೊರಗಿನವರಿಗೆ ಬಹಿರಂಗಪಡಿಸುವುದಿಲ್ಲ. ನನ್ನ ಗೌಪ್ಯತೆ ನಿರ್ವಹಿಸಲ್ಪಡುವವರೆಗೆ ವೈದ್ಯಕೀಯ ಪರೀಕ್ಷೆ, ಪರೀಕ್ಷೆಯ ಮೌಲ್ಯಮಾಪನ ಅಥವಾ ಶಿಕ್ಷಣಕ್ಕಾಗಿ ನನ್ನ ಮಾದರಿಯನ್ನು ಬಳಸಲು ನನ್ನ ಒಪ್ಪಿಗೆಯನ್ನು ನೀಡುತ್ತೇನೆ. ನಾನು ಈ ಅಧ್ಯಯನದಿಂದ ಯಾವುದೇ ಸಮಯದಲ್ಲಿ ಹಿಂತೆಗೆದುಕೊಳ್ಳಲು ಮುಕ್ತವಾಗಿರುತ್ತೇನೆ ಮತ್ತು ಇದು ನನ್ನ ಮುಂದಿನ ಕಾಳಜಿಯನ್ನು ಬದಲಿಸುವುದಿಲ್ಲ ಎಂದು ಅರ್ಥಮಾಡಿಕೊಂಡಿದ್ದೇನೆ. ರೋಗಿಯ ಮಾಹಿತಿ ಪತ್ರವನ್ನು ನಾನು ಓದಿದ್ದೇನೆ ಮತ್ತು ಪ್ರತಿಯನ್ನು ಸ್ವೀಕರಿಸಿದ್ದೇನೆ. ಈ ದಾಖಲೆಯಲ್ಲಿ ಒದಗಿಸಿದ ಮಾಹಿತಿಯನ್ನು ನಾನು ಅರ್ಥಮಾಡಿಕೊಂಡಿದ್ದೇನೆ ಮತ್ತು ಪರೀಕ್ಷೆ, ಪ್ರಕ್ರಿಯೆಗೆ ಸಂಬಂಧಿಸಿದ ಅಪಾಯ ಮತ್ತು ಪರ್ಯಾಯಗಳ ಬಗ್ಗೆ ನಾನು ಹೊಂದಿರುವ ಪ್ರಶ್ನೆಗಳನ್ನು ಕೇಳಲು ನನಗೆ ಅವಕಾಶ ಕಲ್ಪಿಸಲಾಗಿದೆ.

ಹೆಸರು ಮತ್ತು ಸಹಿ/ ಹೆಬ್ಬೆರಳು ಗುರುತು

ದಿನಾಂಕ:

ಪೋಷಕರ/ಪಾಲಕರ ಹೆಸರು/ಹೆಬ್ಬೆರಳು ಗುರುತು

ದಿನಾಂಕ:

ಒಪ್ಪಿಗೆ ತೆಗೆದುಕೊಳ್ಳುವ ವ್ಯಕ್ತಿಯ ಸಹಿ

ದಿನಾಂಕ:

SL NO	UHID NO						PRESENTING COMPLAINTS	RISK FACTORS							VITALS							GPE	
SL NO	UHID NO	GENDER	AGE	DOA	DOD	DURATION	PRESENTING COMPLAINTS	HTN	T2DM	COPD/ ASTHMA	SMOKER	ALCOHOL	TOBACCO/ beetle nut chewer	OTHERS	PULSE	SBP	DBP	TEMP	RR	SPO2	GRBS	PIICKLE	ATHEROSCLEROSIS SIGNS
2	595598	M	67	29/07/2018	04/08/2018	7	Weakness of right UL and LL	1	0	0	1	0	0	0	90	150	90	97.6	16	98	120	0	P
3	610202	M	45	30/07/2018	06/08/2018	7	Slurring of speech, weakmess of lt UL and LL	1	0	0	1	1	0	0	102	180	100	97.2	18	96	176	0	A
4	610204	M	51	30/07/2018	07/08/2018	8	loss of speech, weakness of right upper and lower limb	0	1	0	1	0	0	0	96	130	90	97.2	18	96	296	0	P
5	605410	M	40	01/08/2018	24/08/2018	24	LOSS OF CONSCIOUSNESS	1	0	0	1	0	0	0	90	180	90	97.2	28	90	170	0	P
6	618936	F	70	22/08/2018	29/08/2018	8	Weakness of LEFT UL and LL	0	1	0	0	0	0	0	90	130	80	97.2	16	94	110	0	P
7	454460	M	78	29/08/2018	10/09/2018	12	Weakness of LEFT UL and LL	0	0	0	1	0	0	0	100	130	80	97.8	18	96	108	0	P
8	652127	F	75	29/08/2018	08/09/2018	10	Weakness of right UL and LL	1	0	0	0	0	1	0	96	130	80	97.2	18	96	118	0	P
9	621363	M	60	29/08/2018	07/08/2018	9	Weakness of right UL and LL , SLURRING OF SPEECH, DEVIATION OF ANLGLE OF MOUTH	1	1	0	0	0	0	0	96	130	80	97.6	18	90	126	0	A
10	622502	F	55	31/08/2018	06/09/2018	7	Numbness and weakness of left ul and ll	0	0	0	0	0	0	0	90	110	70	97.6	18	96	127	0	A
11	622783	M	45	01/09/2018	09/09/2018	8	Weakness of LEFT UL and LL	0	1	0	0	0	0	0	96	130	80	97.2	24	96	200	0	A
12	622813	F	75	01/09/2018	10/09/2018	9	WEAKNESS OF RIGHT UL	1	1	0	0	0	0	0	98	130	80	98.2	18	96	108	0	P
13	623934	F	45	04/09/2018	10/09/2018	7	WEAKNESS OF RIGHT UL AND LL	0	0	0	0	0	0	0	100	130	80	97.4	20	98	106	0	A
14	625614	F	55	08/09/2018	18/09/2018	11	LOSS OF SPeech, weakness of right upper and lower limb, difficulty in swallowing	0	1	0	0	0	0	0	90	140	80	97.6	18	99	298	0	P
15	626098	F	75	10/09/2018	19/09/2018	9	Weakness of LEFT UL and LL	1	1	0	0	0	0	0	82	110	70	99	14	94	173	0	P
16	626016	M	65	10/09/2018	15/10/2018	35	SLURRING OF SPEECH ,WEAKNESS OF LEFT UL and LL	0	0	0	1	0	0	0	86	130	80	97.6	18	96	110	0	P
17	626700	M	58	12/09/2018	19/09/2018	7	Weakness of LEFT UL and LL	0	0	1	1	1	0	0	100	110	80	97.4	24	96	205	C	P
18	627178	M	72	14/09/2018	16/09/2018	3	GIDDINESS	1	1	0	0	1	0	0	96	160	100	97.6	18	96	136	0	A
19	629487	F	75	19/09/2018	28/09/2018	10	WEAKNESS OF RIGHT UL AND LL	1	1	0	0	0	0	0	96	190	80	97.6	18	96	390	0	P
20	648052	M	75	06/11/2018	07/11/2018	1	LOSS OF CONSCIOUSNESS	1	1	0	1	0	0	0	96	140	90	98.2	26	90	110	0	P
21	654081	F	65	23/11/2018	30/11/2018	8	Slurring of speech, weakmess of lt UL and LL	1	1	0	1	0	0	0	90	160	80	97.4	18	98	139	0	P
22	652174	M	65	17/11/2018	25/11/2018	9	WEAKNESS OF RIGHT UL AND LL	0	1	1	1	0	0	0	86	130	80	97.2	18	96	210	0	P
23	653644	M	48	21/11/2018	25/11/2018	4	HEADACHE, WEAKNESS OF RIGHT UL AND LL	0	1	0	1	0	0	0	86	140	90	97.2	28	94	382	0	P
24	654847	M	42	25/11/2018	02/12/2018	8	DROWSINESS , SLURRING OF SPEECH, WEAKNESS OF LEFT UL AND LL	1	1	0	1	0	0	0	90	130	90	97.6	18	98	226	0	P
25	655766	M	47	28/11/2018	05/12/2018	8	ALTERED SENSORIUM, , VOMITTING	0	1	0	1	0	0	0	80	130	80	97.4	18	96	293	0	P
26	653607	F	65	29/11/2018	05/12/2018	7	SLURRING OF SPEECH, GENERALISED WEAKNESS	1	1	0	0	0	0	0	86	140	100	99	18	96	226	0	A
27	657010	M	72	01/12/2018	10/12/2018	10	HEADACHE, WEAKNESS OF RIGHT UL AND LL, LOSS OF SPEECH, ALTERED SENSORIUM	1	1	0	1	0	0	0	90	160	80	97.8	18	96	195	0	P
28	657508	F	60	03/12/2018	08/12/2018	5	VOMITTING , 1 E/O LOSS OF CONSCIOUSMESS	1	1	0	0	0	0	0	96	160	100	98.2	18	96	160	0	A
29	667388	M	60	28/12/2018	30/12/2018	2	LOSS OF CONSCIOUSNESS	0	0	0	1	0	0	0	110	130	80	97.4	16	90	100	0	P
30	670618	F	70	06/01/2019	16/01/2019	10	ALTERED SENSORIUM, WEAKNESS OF RIGHT UL AND LL	1	0	0	0	0	1	0	100	160	100	97.6	18	96	110	0	A
31	671249	F	80	08/01/2019	16/01/2019	9	WEAKNESS OF LEFT UL AND LL	1	0	1	0	0	0	0	88	170	100	98.6	18	90	86	0	A
32	672803	M	60	11/01/2019	25/01/2019	15	SLURRING OF SPEECH, WEAKNESS of RIGHT UL and LL	1	0	0	1	0	0	0	98	140	90	97.8	30	90	106	0	P
33	672780	M	52	11/01/2019	17/01/2019	7	LOSS OF CONSCIOUSNESS, WEAKNESS OF LEFT UL AND LL	0	1	0	1	0	0	0	84	120	80	97.6	18	94	410	0	A
34	672406	F	48	11/01/2019	16/01/2019	6	Weakness of LEFT UL and LL	1	0	0	0	0	1	0	72	110	80	97.6	22	98	90	P	A
35	673091	F	45	12/01/2019	18/01/2019	7	deviation of angle of mouth to left, slurring of speech , weakness left UL and LL	0	0	0	0	0	0	0	90	110	70	97.8	12	98	106	P	A
36	673207	F	60	13/01/2019	21/01/2019	9	SLURRING OF SPECH, WEAKNESS OF RIGHT UL AND LL	1	1	0	0	0	0	0	98	160	80	97.4	18	96	242	0	A
37	680282	M	72	31/01/2009	16/02/2019	17	GIDDINESS , VOMITTING, TREMORS OF RIGHT UL	1	1	1	1	0	0	0	90	210	100	97.8	30	96	118	0	P
38	681211	M	75	03/02/2019	10/02/2019	7	LOSS OF CONSCIOUSNESS	1	0	0	0	0	0	0	90	90	60	97.6	18	98	140	0	P
39	681163	M	53	03/02/2019	06/02/2019	4	LOSS OF CONSCIOUSNESS	1	1	0	1	1	0	0	68	150	80	97.5	16	98	97	0	P
40	691370	M	46	01/03/2019	14/03/2019	14	HEADACHE, ALTERED BEHVIOUR,WEAKNESS OF LEFT SIDE	1	1	0	1	0	0	0	90	170	90	97.4	18	96	220	0	P
41	693680	F	75	07/03/2019	14/03/2019	8	LOSS OF SPEECH , WEAKNESS OF RIGHT UL AND LL	0	0	0	0	1	0	0	74	180	90	97.2	18	98	110	0	A
42	694055	F	40	08/03/2019	18/03/2019	11	ALTERED SENSORIU, WEAKNESS OF LEFT ULA ND LL	0	1	0	0	0	0	0	80	100	60	97.6	18	96	367	P	A
43	694722	F	50	11/03/2019	18/03/2019	8	GIDDINESS, VOMITTING	0	1	0	0	0	0	0	86	140	80	97.6	18	98	88	0	A
44	697139	F	80	16/03/2019	16/03/2019	1	LOSS OF CONSCIUOSNESS	1	1	0	0	0	0	0	96	160	90	97.8	16	96	201	0	P
45	703621	M	60	03/04/2019	14/04/2019	12	LOSS OF CONSCIUOSNESS, PAUCITY OF MOVEMENTS OF RIGHT UL AND LL	0	0	0	1	0	0	0	46	150	90	97.2	18	97	140	0	A
46	703598	M	70	03/04/2019	06/04/2019	4	GIDDINESS	1	0	0	0	0	0	0	88	150	100	97.4	18	96	100	0	A
47	707885	F	75	14/04/2019	20/04/2019	7	LOSS OF CONSCIOUSNESS, WEAKNESS OF RIGHT UL AND LL	1	1	0	0	0	0	0	98	160	100	98	18	96	198	0	A
48	710772	M	80	23/04/2019	30/04/2019	7	WEAKNESS OF RIGHT UL AND LL	0	0	0	0	0	0	0	90	130	90	97.4	18	96	105	0	A
49	713696	M	42	30/04/2019	02/05/2019	3	GIDDINESS	1	1	0	0	0	0	0	96	200	100	97.6	18	98	190	0	A
50	714458	F	70	02/05/2019	09/05/2019	8	GIDDINESS, VOMITTING	1	1	0	0	0	0	0	80	160	100	97.4	18	90	534	0	A
51	714471	M	67	02/05/2019	07/05/2019	3	GIDDINESS, HEADACHE	1	0	0	0	0	0	0	80	180	100	97.4	16	96	118	0	A
52	718342	F	50	12/05/2019	15/05/2019	4	WEAKNESS OF RIGHT UL AND LL	1	0	0	0	0	0	0	120	190	130	97.2	18	97	130	0	P
53	721900	F	80	22/05/2019	30/05/2019	9	LOSS OF CONSCIOUSNESS, PAUCITY OF MOVEMETNS OF RIGHT UL AND LL	0	0	0	0	0	0	0	78	160	80	97.4	18	94	118	P	A
54	726078	M	70	02/06/2019	12/06/2019	10	GIDDINESS, WEAKNESS OF RIGHT UL AND LL	1	1	0	0	0	0	0	90	110	70	97.2	18	94	87	0	P
55	727022	M	62	05/06/2019	08/06/2019	4	LOSS OF CONSCIOUSNESS	0	0	0	0	0	0	0	130	140	90	98.2	22	94	126	0	A
56	727332	M	65	05/06/2019	16/06/2019	12	LOSS OF CONSCIOUSNESS, PAUCITY OF MOVEMETNS OF LEFT UL AND LL	0	1	0	0	1	0	0	68	130	90	97.6	18	96	290	0	A
57	728133	M	73	07/06/2019	14/06/2019	8	Weakness of LEFT UL and LL	1	0	0	0	0	0	0	96	200	100	97.4	18	96	109	0	A
58	730116	M	75	12/06/2019	20/06/2019	9	LOSS OF CONSCIOUSNESS	0	1	0	1	0	0	0	80	190	100	97.2	18	96	307	0	A
59	730101	M	50	13/06/2019	15/06/2019	3	GIDDINESS	0	1	0	1	0	0	0	80	140	90	98.3	18	96	240	0	P
60	731322	M	48	16/06/2019	16/06/2019	1	LOSS OF CONSCIOUSNESS	0	1	0	1	0	0	0	110	130	80	100	30	90	437	0	P
61	736048	M	55	28/06/2019	01/07/2019	4	GIDDINESS , SPEECH DISTURBANCE	0	1	0	0	0	0	0	90	130	90	97.2	18	96	110	0	P
62	736570	M	65	29/06/2019	05/07/2019	7	GIDDINESS	0	0	0	0	0	0	0	80	150	70	97.4	20	96	114	0	A
63	738927	M	78	05/07/2019	12/07/2019	8	LOSS OF CONSCIOUSNESS , WEAKNESS OF LEFT UL AND LL	1	1	0	1	1	0	0	84	200	110	97.6	20	96	106	0	P
64	742225	F	70	16/07/2019	17/07/2019	2	LOSS OF CONSCIOUSNESS	0	0	0	0	0	1	0	100	110	70	97.6	18	96	110	0	P
65	744547	M	75	19/07/2019	26/07/2019	8	WEAKNESS OF LEFT UL and LL , DEVIATION OF ANGLE OF MOUTH TO RIGHT	0	0	0	1	1	0	0	90	140	80	97.6	18	96	114	0	P
66	835474	M	40	03/08/2020	15/3/2020	7	SEIZURES, WEAKNESS OF RIGHT UPPER AND LOWER LIMB	0	0	0	0	0	0	0	118	130	70	97.4	17	99	223	0	A
67	828158	M	80	18/2/2020			GIDDINESS	0	1	1	0	0	0	0	90	140	80	98	18	94	179	P	P
68	837595	M	59	13/03/2020			GIDDINESS, WEAKNESS OF RIGHT UL AND LL	0	1	1	1	1	0	0	92	190	130	97.7	23	96	237	0	A

SL NO	SYSTEMIC EXAMINATION			CNS				POWER				PLANTAR		DTR (B/T/S/K/A)		TONE		SENSORY		CEREBELLAR SIGNS	
SL NO	CVS	RS	P/A	HMF	GCS	PUPILS	CRANIAL NERVES	RIGHT		LEFT		RIGHT	LEFT	RIGHT	LEFT	RIGHT	LEFT	RIGHT	LEFT	RIGHT	LEFT
2	S1 S2 +	NVBS	SOFT	NORMAL	15	BERL	NORMAL	5	5	0	0	FLEXOR	EXTENSOR	2	1	NORMAL	NORMAL	NORMAL	NORMAL	NO	NO
3	S1 S2 +	NVBS	SOFT	NORMAL	15	BERL	left UMN 7th facial palsy	5	5	0	0	FLEXOR	EXTENSOR	2	2	NORMAL	NORMAL	NORMAL	NORMAL	NO	NO
4	S1 S2 +	NVBS	SOFT	DROWSY	13	BERL	right UMN 7th facial palsy , RT HEMIANOPIA	0	0	5	5	EXTENSOR	FLEXOR	2	2	NORMAL	NORMAL	NORMAL	NORMAL	-	-
5	S1 S2 +	NVBS	SOFT	DROWSY	10	BERL	gaze palsy	0	0	0	0	EXTENSOR	MUTE	2	2	NORMAL	NORMAL	-	-	-	-
6	S1 S2 +	NVBS	SOFT	NORMAL	15	BERL	NORMAL	5	5	0	0	FLEXOR	EXTENSOR	2	2	NORMAL	NORMAL	NORMAL	NORMAL	NO	NO
7	S1 S2 +	NVBS	SOFT	NORMAL	15	BERL	NORMAL	5	5	0	0	FLEXOR	EXTENSOR	2	2	NORMAL	NORMAL	NORMAL	NORMAL	NO	NO
8	S1 S2 +	NVBS	SOFT	NORMAL	15	BERL	NORMAL	0	0	5	5	EXTENSOR	FLEXOR	3	2	NORMAL	NORMAL	NORMAL	NORMAL	NO	NO
9	S1 S2 +	NVBS	SOFT	NORMAL	15	BERL	right UMN 7th facial palsy	0	0	5	5	EXTENSOR	FLEXOR	2	2	NORMAL	NORMAL	NORMAL	NORMAL	NO	NO
10	S1 S2 +	NVBS	SOFT	NORMAL	15	BERL	NORMAL	5	5	3	3	FLEXOR	EXTENSOR	2	1	NORMAL	NORMAL	NORMAL	REDUCED	NO	NO
11	S1 S2 +	NVBS	SOFT	NORMAL	15	BERL	NORMAL	5	5	0	0	FLEXOR	EXTENSOR	2	3	NORMAL	SPASTICITY	NORMAL	NORMAL	NO	NO
12	S1 S2 +	NVBS	SOFT	NORMAL	15	BERL	NORMAL	0	4	5	5	FLEXOR	FLEXOR	2	2	NORMAL	NORMAL	NORMAL	NORMAL	NO	NO
13	S1 S2 +	NVBS	SOFT	NORMAL	15	BERL	NORMAL	2	2	5	5	EXTENSOR	FLEXOR	2	2	NORMAL	NORMAL	NORMAL	NORMAL	NO	NO
14	S1 S2 +	NVBS	SOFT	APHASIA	13	BERL	NORMAL	0	0	5	5	EXTENSOR	FLEXOR	3	2	NORMAL	NORMAL	NORMAL	NORMAL	NO	NO
15	S1 S2 +	NVBS	SOFT	NORMAL	15	BERL	NORMAL	5	5	0	0	FLEXOR	EXTENSOR	2	2	NORMAL	NORMAL	NORMAL	NORMAL	NO	NO
16	S1 S2 +	NVBS	SOFT	DROWSY	12	BERL	left UMN 7th facial palsy	5	5	0	0	FLEXOR	EXTENSOR	2	3	NORMAL	NORMAL	NORMAL	NORMAL	-	-
17	S1 S2 +	NVBS	SOFT	NORMAL	15	BERL	NORMAL	5	5	0	0	FLEXOR	EXTENSOR	2	3	NORMAL	SPASTICITY	NORMAL	REDUCED	NO	NO
18	S1 S2 +	NVBS	SOFT	NORMAL	15	BERL	NORMAL	5	5	5	5	FLEXOR	FLEXOR	2	2	NORMAL	NORMAL	NORMAL	NORMAL	PRESENT	NO
19	S1 S2 +	NVBS	SOFT	NORMAL	15	BERL	NORMAL	1	1	5	5	EXTENSOR	FLEXOR	3	2	NORMAL	NORMAL	NORMAL	NORMAL	NO	NO
20	S1 S2 +	NVBS	SOFT	UNCONSCIOUS	3	BERL	-	-	-	-	-	MUTE	MUTE	1	1	NORMAL	NORMAL	-	-	-	-
21	S1 S2 +	NVBS	SOFT	NORMAL	15	BERL	left UMN 7th facial palsy	5	5	3	3	FLEXOR	MUTE	2	3	NORMAL	SPASTICITY	NORMAL	NORMAL	NO	NO
22	S1 S2 +	NVBS	SOFT	DROWSY	12	POST CATRACT	NORMAL	0	0	5	5	EXTENSOR	FLEXOR	2	2	SPASTICITY	NORMAL	NORMAL	NORMAL	NO	NO
23	S1 S2 +	NVBS	SOFT	DROWSY	11	BERL	NORMAL	0	0	3	3	MUTE	MUTE	2	2	NORMAL	NORMAL	-	-	-	-
24	S1 S2 +	NVBS	SOFT	DROWSY	10	BERL	left UMN 7th facial palsy	5	5	3	3	FLEXOR	EXTENSOR	2	2	NORMAL	NORMAL	NORMAL	NORMAL	NO	NO
25	S1 S2 +	NVBS	SOFT	DROWSY	9	BERL	gaze palsy	3	3	3	3	MUTE	MUTE	2	2	NORMAL	NORMAL	-	-	-	-
26	S1 S2 +	NVBS	SOFT	NOT ORIENRED	13	BERL	NORMAL	5	5	5	5	FLEXOR	FLEXOR	1	1	NORMAL	NORMAL	NORMAL	NORMAL	PRESENT	NO
27	S1 S2 +	NVBS	SOFT	APHASIA	10	BERL	RIGHT UMN FACIAL PALSY	0	0	5	5	EXTENSOR	FLEXOR	2	2	NORMAL	NORMAL	-	-	-	-
28	S1 S2 +	NVBS	SOFT	DROWSY	13	BERL	NORMAL	4	4	4	4	EXTENSOR	FLEXOR	2	2	NORMAL	NORMAL	NORMAL	NORMAL	NO	NO
29	S1 S2 +	NVBS	SOFT	UNCONSCIOUS	3	BERL	-	-	-	-	-	MUTE	MUTE	1	1	NORMAL	NORMAL	-	-	-	-
30	S1 S2 +	NVBS	SOFT	NORMAL	15	BERL	RIGHT UMN FACIAL PALSY	3	3	5	5	EXTENSOR	FLEXOR	2	2	SPASTICITY	NORMAL	NORMAL		NO	NO
31	S1 S2 +	NVBS	SOFT	NORMAL	15	BERL	RIGHT UMN FACIAL PALSY	5	5	0	0	FLEXOR	EXTENSOR	2	3	NORMAL	SPASTICITY	NORMAL	NORMAL	NO	NO
32	S1 S2 +	NVBS	SOFT	UNCONSCIOUS	5	BERL	NORMAL	0	0	3	3	EXTENSOR	EXTENSOR	2	2	NORMAL	NORMAL	-	-	-	-
33	S1 S2 +	NVBS	SOFT	DROWSY	10	BERL	NORMAL	0	0	4+	4+	EXTENSOR	FLEXOR	2	2	NORMAL	NORMAL	NORMAL	NORMAL	NO	NO
34	S1 S2 +	NVBS	SOFT	NORMAL	15	BERL	left UMN 7th facial palsy	5	5	3	3	FLEXOR	EXTENSOR	2	3	NORMAL	SPASTICITY	NORMAL	NORMAL	NO	NO
35	S1 S2 +	NVBS	SOFT	NORMAL	15	BERL	left UMN 7th facial palsy	5	5	2	2	FLEXOR	EXTENSOR	2	2	NORMAL	SPASTICITY	NORMAL	NORMAL	NO	NO
36	S1 S2 +	NVBS	SOFT	NORMAL	15	BERL	left UMN 7th facial palsy	5	5	3	3	FLEXOR	EXTENSOR	2	2	NORMAL	NORMAL	NORMAL	NORMAL	NO	NO
37	S1 S2 +	NVBS	SOFT	NORMAL	15	BERL	NYSTAGMUS	5	5	4	4	WITHDRAWL	WITHDRAWL	2	2	NORMAL	NORMAL	NORMAL	NORMAL	PRESENT	NO
38	S1 S2 +	NVBS	SOFT	UNCONSCIOUS	6	BERL	-	-	-	-	-	MUTE	MUTE	2	2	NORMAL	NORMAL	-	-	-	-
39	S1 S2 +	NVBS	SOFT	UNCONSCIOUS	5	BERL	-	-	-	-	-	MUTE	MUTE	1	1	NORMAL	NORMAL	-	-	-	-
40	S1 S2 +	NVBS	SOFT	NOT ORIENRED	11	BERL	left UMN 7th facial palsy	4	4	1	1	EXTENSOR	FLEXOR	2	2	NORMAL	NORMAL	NORMAL	NORMAL	NO	NO
41	S1 S2 +	NVBS	SOFT	NOT ORIENRED	13	BERL	RIGHT UMN FACIAL PALSY	0	0	3	3	MUTE	FLEXOR	2	2	NORMAL	NORMAL	-	-	-	-
42	S1 S2 +	NVBS	SOFT	NORMAL	15	BERL	left UMN 7th facial palsy	5	5	0	0	FLEXOR	EXTENSOR	2	3	NORMAL	SPASTICITY	NORMAL	NORMAL	NO	NO
43	S1 S2 +	NVBS	SOFT	NORMAL	15	BERL	NYSTAGMUS	5	5	5	5	FLEXOR	FLEXOR	2	2	NORMAL	NORMAL	NORMAL	NORMAL	NO	PRESENT
44	S1 S2 +	NVBS	SOFT	UNCONSCIOUS	3	BERL	gaze palsy	-	-	-	-	EXTENSOR	MUTE	2	2	NORMAL	NORMAL	-	-	-	-
45	S1 S2 +	NVBS	SOFT	DROWSY	8	BERL	-	-	-	-	-	EXTENSOR	MUTE	2	2	NORMAL	NORMAL	-	-	-	-
46	S1 S2 +	NVBS	SOFT	NORMAL	15	BERL	NORMAL	5	5	5	5	FLEXOR	FLEXOR	2	2	NORMAL	NORMAL	NORMAL	NORMAL	NO	PRESENT
47	S1 S2 +	NVBS	SOFT	DROWSY	7	BERL	-	0	0	3	3	EXTENSOR	FLEXOR	2	2	NORMAL	NORMAL	-	-	-	-
48	S1 S2 +	NVBS	SOFT	NOT ORIENRED	13	BERL	NORMAL	0	0	3	3	EXTENSOR	FLEXOR	2	2	NORMAL	NORMAL	-	-	-	-
49	S1 S2 +	NVBS	SOFT	NORMAL	15	BERL	NORMAL	5	5	5	5	FLEXOR	FLEXOR	2	2	NORMAL	NORMAL	NORMAL	NORMAL	PRESENT	NO
50	S1 S2 +	NVBS	SOFT	DROWSY	14	BERL	NORMAL	5	5	5	5	FLEXOR	FLEXOR	1	1	NORMAL	NORMAL	NORMAL	NORMAL	PRESENT	NO
51	S1 S2 +	NVBS	SOFT	NORMAL	15	BERL	NORMAL	5	5	5	5	FLEXOR	FLEXOR	2	2	NORMAL	NORMAL	NORMAL	NORMAL	PRESENT	NO
52	S1 S2 +	NVBS	SOFT	NORMAL	15	BERL	NORMAL	4	4	5	5	EXTENSOR	FLEXOR	2	2	NORMAL	NORMAL	NORMAL	NORMAL	NO	NO
53	S1 S2 +	NVBS	SOFT	DROWSY	10	BERL	GAze palsy , RIGHT UMN 7TH CRANIAL PALSY	0	0	3	3	EXTENSOR	FLEXOR	2	2	NORMAL	NORMAL	-	-	-	-
54	S1 S2 +	NVBS	SOFT	NORMAL	15	BERL	NORMAL	5	5	2	2	FLEXOR	EXTENSOR	2	0	NORMAL	HYPOTONIA	NORMAL	NORMAL	NO	NO
55	S1 S2 +	NVBS	SOFT	UNCONSCIOUS	4	BERL	-	-	-	-	-	MUTE	MUTE	0	0	HYPOTONIA	HYPOTONIA	-	-	-	-
56	S1 S2 +	NVBS	SOFT	UNCONSCIOUS	9	BERL	GAZE PALSY	3	3	0	0	FLEXOR	EXTENSOR	2	2	NORMAL	NORMAL	-	-	-	-
57	S1 S2 +	NVBS	SOFT	NOT ORIENRED	11	BERL	-	3	3	0	0	FLEXOR	EXTENSOR	2	2	NORMAL	NORMAL	-	-	-	-
58	S1 S2 +	NVBS	SOFT	UNCONSCIOUS	8	BERL	-	0	0	3	3	EXTENSOR	FLEXOR	2	2	NORMAL	NORMAL	-	-	-	-
59	S1 S2 +	NVBS	SOFT	NORMAL	15	BERL	NYSTAGMUS	5	5	5	5	MUTE	MUTE	2	2	NORMAL	NORMAL	NORMAL	NORMAL	NO	PRESENT
60	S1 S2 +	NVBS	SOFT	UNCONSCIOUS	4	BERL	GAZE PALSY	-	-	-	-	EXTENSOR	EXTENSOR	1	1	NORMAL	NORMAL	-	-	-	-
61	S1 S2 +	NVBS	SOFT	NORMAL	15	BERL	NORMAL	5	5	5	5	FLEXOR	FLEXOR	2	2	NORMAL	NORMAL	NORMAL	NORMAL	PRESENT	NO
62	S1 S2 +	NVBS	SOFT	NORMAL	15	BERL	NYSTAGMUS, DYSARTHRIA	5	5	5	5	MUTE	MUTE	2	2	NORMAL	NORMAL	NORMAL	NORMAL	NO	PRESENT
63	S1 S2 +	NVBS	SOFT	NORMAL	15	BERL	left UMN 7th facial palsy	5	5	0	0	FLEXOR	EXTENSOR	2	2	NORMAL	NORMAL	NORMAL	NORMAL	NO	NO
64	S1 S2 +	NVBS	SOFT	UNCONSCIOUS	8	BERL	GAze palsy	-	-	-	-	MUTE	MUTE	1	1	NORMAL	NORMAL	-	-	-	-
65	S1 S2 +	NVBS	SOFT	NORMAL	15	BERL	left UMN 7th facial palsy	5	5	0	0	FLEXOR	EXTENSOR	2	2	NORMAL	NORMAL	NORMAL	NORMAL	NO	NO
66	S1S2 +	NVBS	SOFT	UNCONSCIOUS	4	BERL	RIGHT UMN 7TH facial N Palsy	.	.	.	.	EXTENSOR	EXTEMSOR	2	1	NORMAL	NORMAL	.	.	.	.
67	S1S2 +	NVBS	SOFT	NOT ORIENRED	11	BERL	NORMAL	4	4	4	4	MUTE	MUTE	2	2	NORMAL	NORMAL	.	.	.	.
68	S1S2 +	NVBS	SOFT	NOT ORIENRED	8	BERL	left UMN 7th facial palsy	4	4	0	0	FLEXOR	EXTENSOR	2	1	NORMAL	NORMAL	.	.	.	.

SL NO	CBC							RFT				SUGAR				LIPID PROFILE				CT SCAN / MRI BRAIN
SL NO	HB	PCV	MCV	TC	N	L	PLATELETS	BLOOD UREA	SERUM CREATININE	SODIUM	POTASSIUM	RBS	FBS	PPBS	HBA1C	SERUM CHOLESTEROL	TG	HDL	LDL	
2	12.9	38	77.4	10.96	72	14.5	260	13	0.7	135	4.9	87	100	126	5.4	196	194	37	120	HYPODENSITY IN RIGHT CORONA RADIATA
3	15.6	47	83.6	10.3	89.3	4.6	334	38	1.2	142	5.2	109	123	140	6.2	160	91	62	80	HYPODENSITY IN RIGHT INTERNAL CAPSULE
4	15.1	45	92	10.21	75.6	15.8	262	17	0.9	139	5	179	190	230	8.6	191	88	64	109	HYPODENSITY IN LEFT INTERNAL CAPSULE
5	16.2	46	81	28.33	94.9	1.4	222	42	1.2	138	5	81	90	110	5.6	170	94	68	88	HYPODENSITY IN LEFT FRONTOTEMPORAL AREA
6	11.3	33.5	81.1	10.71	69.4	18.6	261	22	0.5	136	3.5	97	96	170	6.9	180	100	50	90	HYPODENSITY IN RIGHT CAPSULO GANGLIONIC REGION
7	12.9	39.8	81.7	7.08	58.9	26.6	265	47	1.2	137	4.9	92	98	110	5.4	157	211	31	84	HYPODENSITY IN RIGHT CAPSULO GANGLIONIC AREA
8	12.2	34.7	75.9	12.01	73.7	18	250	45	0.6	138	2.5	86	18	197	5.8	164	123	33	107	HYPODENSITY IN LEFT INTERNAL CAPSULE
9	16.1	45.2	85.5	22.2	89	4.5	459	29	1.1	137	3.4	52	261	146	15.2	117	89	44	55	HYPODENSITY IN LEFT INTERNAL CAPSULE
10	12.4	40	90.9	25.06	91.5	33	264	33	1.2	143	4.4	105	69	126	5.4	120	90	42	84	HYPODENSITY IN RIGHT CAPSULO GANGLIONIC AREA
11	13.8	37.7	84.7	10.19	50.19	28	268	29	0.7	138	4.4	206	89	152	11.7	158	210	30	82	HYPODENSITY IN RIGHT CAPSULOGANGLIONIC REGION
12	12.8	34.5	82	12.34	72	20.8	187	18	0.6	134	4	226	223	256	12.8	255	128	40	189	HYPODENSITY IN LEFT INTERNAL CAPSULE
13	11.4	34.5	82	7.6	69.4	18.8	186	17	0.6	135	3.6	93	70	126	5.8	238	145	38	171	LACUNAR INFARCT IN RIGHT INTERNAL CAPSULE
14	11.4	33.5	79.6	13.5	81.9	7.5	219	30	1.1	135	2.2	149	110	200	8.6	186	98	37	97	HYPODENSITY IN LEFT FRONTOTEMPORAL AREA
15	12.9	37.8	94	13.26	85.5	6.5	327	30	0.8	130	4.2	117	133	135	7.6	136	90	46	67	HYPODENSITY IN RIGHT CAPSULOGANGLIONIC REGION
16	17.2	43.2	75.3	14.83	75.9	15.3	3.1	39	0.6	103	3.3	56	68	110	5.6	131	93	46	67	HYPODENSITY IN RIGHT MCA AND PCA TERRITORY
17	16.2	45.1	76.4	9.04	66.5	16	224	24	0.6	138	4.2	94	90	126	5.4	175	145	38	108	HYPODENSITY IN RIGHT MCA TERRITORY
18	13.8	38.6	77.8	9.78	83.2	13	204	20	0.6	134	4.8	206	225	349	12.2	202	242	44	109	HYPODENSITY IN RIGHT CEREBELLAR REGION
19	12.8	39.6	79.8	10.78	83.1	14	224	39	0.6	132	4.1	210	172	205	13	202	140	46	128	LACUNAR INFARCT IN LEFT INTERNAL CAPSULE
20	15.6	44.8	84.2	19	90	5.9	406	23	0.8	129	4.8	207	130	336	14.6	220	110	38	190	HYPODENSITY IN LEFT CEREBELLAR HEMISPHERE
21	12.2	35.2	86.1	7.96	80.7	12.7	265	16	0.8	136	4.1	127	100	212	7.6	249	212	43	163	HYPODENSITY IN LEFT INTERNAL CAPSULE
22	16.1	48.7	87.9	10.34	73.9	16.4	231	36	1.2	138	5.8	324	598	502	12.7	251	180	40	174	HYPODENSITY IN LEFT FRONTOPAREITAL REGION
23	15.6	45.7	81.6	10.94	61.7	29.6	320	32	0.5	127	3.7	213	315	276	11.4	172	128	45	102	HYPODENSITY IN LEFT TEMPORO PARIETAL REGION
24	13.4	38	79.3	6.7	50.3	33.7	169	10	0.4	138	4	208	204	116	10.3	180	230	27	107	HYPODENSITY IN RIGHT HIGH PARIETAL REGION
25	16.1	46.6	81.6	15.23	61.7	29.6	235	41	1	137	4.8	423	123	233	10.6	151	154	43	78	HYPODENSITY IN LEFT HIGH PARIETAL REGION
26	11.9	34.5	82.4	14.2	79.9	12.7	226	55	1.2	135	3.8	226	279	270	9.2	153	135	20	106	HYPODENSITY IN RIGHT CEREBELLAR AND THALAMIC REGION
27	14.7	39.1	80.5	25.78	88	5.5	373	49	1.3	129	5.5	173	80	180	5.7	205	103	48	136	HYPODENSITY IN LEFT HIGH PARIETAL REGION
28	13.5	38.6	81.8	14.63	87.2	9.6	338	32	0.5	136	3.6	190	311	255	12.7	242	117	63	155	HYPODENSITY IN LEFT PARIETO OCCIPITAL REGION
29	15.1	45	80.6	17.99	87.7	7.3	304	42	1	138	5.6	132	88	146	5.8	201	148	40	131	HYPODENSITY IN LEFT FRONTO PARIETAL REGION
30	15.9	44.9	75.7	12	86.6	9.2	352	35	0.1	132	3.1	110	110	136	5.8	169	123	56	89	HYPODENSITY IN RIGHT CAUDATE AND LENTIFORM NUCLEUS
31	11	32.7	80.9	14.03	81.2	12.3	226	34	0.9	137	4.5	91	81	110	5.6	160	110	42	98	HYPODENSITY IN LEFT CAPSULO GANGLIONIC REGION
32	15	41.8	83.8	17.21	80.5	9.2	398	38	0.9	130	3.8	90	66	114	5.8	249	235	47	156	HYPODENSITY IN LEFT INTERNAL CAPSULE AND HIGH PARIETAL REGION
33	15.7	45.5	79.5	17.18	98.4	3.8	214	30	0.7	134	6.5	286	421	520	10.1	281	104	52	208	HYPODENSITY IN RIGHT CAPSULOGANGLIONIC REGION
34	8.8	29.1	64.5	6.5	72.6	18.9	206	24	0.5	136	3.9	98	129	94	6.5	131	103	17	93	HYPODENSITY IN RIGHT CAPSULO GANGLIONIC AREA
35	8.5	30.2	64.14	16.8	82.8	8.7	324	27	0.6	137	4	100	72	130	5.9	189	126	38	126	HYPODENSITY IN RIGHT TEMPORO PARIETO OCCIPITAL REGION
36	12.9	39.9	81.8	11.12	72.6	19.2	466	24	0.5	135	4.8	190	220	320	10.7	231	64	52	166	HYPODENSITY IN RIGHT CAPSULO GANGLIONIC AREA
37	9.9	29.5	85.5	9.15	93.6	4.2	132	27	1.1	132	4.3	100	107	210	6.8	278	196	45	193	HYPODENSITY IN BILATERAL CEREBELLAR REGION
38	10.1	29.9	90.1	15.71	80.6	6.2	193	39	0.7	147	2.1	96	70	130	5.8	198	132	44	127	HYPODENSITY IN LEFT FRONTOTEMPORAL AREA
39	13.3	40.6	72.8	9.2	75	16.6	325	33	0.6	132	3.9	97	90	126	5.8	202	131	46	103	HYPODENSITY IN RIGHT FRONTOTEMPRAL REGION
40	16.5	45.5	79.4	17.49	70.5	20.7	349	26	0.7	133	4.1	311	201	236	9.8	176	148	48	98	MCA DOT SIGN IN RIGHT MCA
41	15.3	45.5	82.9	12.7	79.3	11.5	366	18	0.6	134	2.9	140	78	136	5.8	170	184	38	95	HYPODENSITY IN LEFT BASAL GANGLION REGION
42	9	30.3	63.4	10.22	69.4	21.3	577	39	0.6	130	4.2	90	90	110	5.7	162	110	42	108	HYPODENSITY IN RIGHT CAPSULOGANGLIONIC REGION
43	13.3	38.7	83	11.9	76	18	392	25	0.4	134	4.2	136	221	244	10	187	156	38	117	HYPODENSITY IN PICA TERRITORY
44	12.9	38	77.4	10.96	72	14.5	260	18	0.6	136	4.8	188	220	360	14.5	202	198	35	124	HYPODENSITY IN LEFT CAPSULO GANGLIONIC REGION
45	16	48.7	90.4	8.23	41	43.5	189	48	1.2	142	3.8	73	78	160	5.6	171	168	33	105	HYPODENSITY IN LEFT CAPSULO GANGLIONIC REGION
46	15.7	44.4	81.3	10.5	90.4	5	183	42	0.7	135	4	100	90	130	5.8	134	95	50	68	HYPODENSITY IN LEFT CEREBELLAR HEMISPHERE
47	14.9	44.1	84.5	16.8	87.1	10.71	169	18	0.9	136	4.1	191	197	178	6.8	240	307	44	178	HYPODENSITY IN FRONTO PARIETAL AND TEMPORAL REGION
48	13.1	36.7	85.2	8.51	57.5	23.3	234	26	0.7	138	3.8	67	70	116	5.2	153	134	35	91	HYPODENSITY IN LEFT CAPSULO GANGLIONIC REGION
49	16.8	47.9	85.8	12.55	60.8	30.8	396	50	0.6	131	3.8	281	259	298	8.6	154	120	30	96	HYPODENSITY IN RIGHT CEREBELLAR REGION
50	13.1	35.2	77.9	15.6	87.5	7.4	220	44	0.5	139	2.6	477	235	139	14.9	160	98	48	90	HYPODENSITY IN RIGHT CEREBELLAR REGION
51	13.1	37.1	84.9	9.04	43.5	28.2	216	35	1.2	141	3.2	87	96	130	5.2	233	327	28	129	HYPODENSITY IN RIGHT CEREBELLAR REGION
52	12.2	36.1	74	21.6	93.5	2.7	223	27	1.2	134	2.4	170	138	166	5.9	237	126	48	164	HYPODENSITY IN LEFT CAPSULO GANGLIONIC REGION
53	5.2	19.2	60.2	12.4	92.4	3.9	407	21	0.4	133	4.3	96	90	124	5.8	166	94	49	90	HYPODENSITY IN LEFT FRONTOTEMPRAL REGION
54	12.3	36.7	75.4	9.95	50	8.5	299	42	1.1	133	3.5	111	210	330	9.2	138	82	46	75	HYPODENSITY IN RIGHT MCA ACA WATERSHED
55	13.6	39	85.9	18.7	68.3	16.1	144	27	0.9	133	4	90	78	130	5.8	61	141	14	19	HYPODENSITY IN B/L CEREBELLAR HEMISPHERE
56	14.9	42.7	81.3	31.6	94.8	2.4	257	25	1	131	4.3	78	90	106	9.3	139	67	46	80	HYPODENSITY IN LEFT PARIETO TEMPORAL REGION
57	13.8	39.1	84.6	8.78	83.5	9.2	319	27	0.7	136	3.7	85	98	110	5.6	174	110	39	69	HYPODENSITY IN LEFT CAPSULO GANGLIONIC AREA
58	12.8	35.6	70.6	11.2	65.1	23.8	200	43	0.5	135	4.4	208	268	320	9.3	139	67	46	80	HYPODENSITY IN FRONTO PARIETAL AND TEMPORAL REGION
59	12	33.3	78.2	11.48	90.6	6.7	201	36	1	132	6	193	238	218	9.2	153	111	30	101	HYPODENSITY IN LEFT CEREBELLAR HEMISPHERE
60	16.1	45	112	10.8	90.2	8.5	317	34	0.8	139	4.4	138	241	300	10.9	249	180	42	170	HYPODENSITY IN RIGHT OCCIPITA PARIETAL REGION
61	10.8	35.2	71.7	7.54	36.6	37.4	336	21	0.9	136	4.2	128	160	230	7.8	135	146	29	77	HYPODENSITY IN RIGHT CEREBELLAR REGION
62	12.1	34.8	80	17.51	91.3	5.4	236	19	0.7	136	3.8	124	90	118	5.8	157	97	39	98	HYPODENSITY IN LEFT CEREBELLAR HEMISPHERE
63	15.1	43.4	82.4	17.75	86.7	9.1	156	29	1	130	4.7	320	400	520	13.8	186	125	34	127	MCA DOT SIGN IN RIGHT MCA
64	11.9	34.3	90.5	13.56	64.4	28.5	254	43	0.8	131	4.2	113	90	130	5.6	236	139	30	106	HYPODENSITY IN LEFT ACA WATERSHED REGION
65	14.7	42.7	88.8	9.51	67.5	19.9	179	28	1.1	134	3.7	84	90	116	5.6	156	95	40	96	HYPODENSITY IN RIGHT CAPSULO GANGLIONIC AREA
66	14.7	45.5	89.9	16.44	63.2	2..1	124	60	5.3	135	3.9	243	136	165	5.9	176	123	45	74	HYPODENSITY IN BILATERAL FRONTO TEMPORO PARIETAL REGION
67	8.1	24.3	80.5	19.5	73.6	17.5	518	26	0.7	127	5.5	184	113	149	6.3	201	197	38	94	HYPODENSITY IN LEFT FRONTOTEMPOROPARIETOOCCIPITAL REGION
68	15	42.4	88.1	9.43	73.8	16.2	286	29	1	140	3.7	285	176	153	6.9	203	348	49	84	HYPODENSITY IN RIGHT FRONTOTEMPROPARIETAL REGION

SL NO	ARTERY INVOLVED								OXFORDSHIRE COMMUNITY STROKE PROJECT				CAROTID DOPPLER	ECG	2D ECHO	CXR
SL NO	ICA	ACA	MCA	PCA	BASILLAR	SICA	PICA	VERTEBRAL	TACS	PACS	LAC	POC				
2	0	1	0	0	0	0	0	0	0	1	0	0	DIFFUSE ATHEROSCLEROTIC CHANGES IN B/L CAROTID ARTERY < 50% STENOSIS , INCREASED INTIMAL THICKNESS VERTEBRAL ARTERY NORMAL FLOW ,DIRECTIOM	NO ST T CHANGES	NO RWMA, NORMAL CHAMBERS LVEF 60%	NO RADIOLOGICAL ABNORMALITY
3	0	0	1	0	0	0	0	0	0	1	0	0	BOTH COMMON CAROTID NORMAL FLOW , NORMAL IMTIMAL THICKNESS, NO STENOSIS , VERTEBRAL ARTERIES NORMAL	NO ST T CHANGES	NO RWMA, NORMAL CHAMBERS LVEF 60%	NO RADIOLOGICAL ABNORMALITY
4	0	1	1	0	0	0	0	0	1	0	0	0	DIFFUSE ATHEROSCLEROTIC CHANGES IN B/L CAROTID ARTERY 70% STENOSIS in left 44% stenosis in right , INCREASED INTIMAL THICKNESS VERTEBRAL ARTERY NORMAL FLOW ,DIRECTIOM	NO ST T CHANGES	NO RWMA, NORMAL CHAMBERS LVEF 60%	NO RADIOLOGICAL ABNORMALITY
5	0	1	1	0	0	0	0	0	1	0	0	0	-	NO ST T CHANGES	NO RWMA, NORMAL CHAMBERS LVEF 60%	NO RADIOLOGICAL ABNORMALITY
6	0	0	1	0	0	0	0	0	0	1	0	0	DIFFUSE ATHEROSCLEROTIC CHANGES IN B/L CAROTID ARTERY38 %STENOSIS in right 46% in left , INCREASED INTIMAL THICKNESS VERTEBRAL ARTERY NORMAL FLOW ,DIRECTIOM	NO ST T CHANGES	NO RWMA, NORMAL CHAMBERS LVEF 60%	NO RADIOLOGICAL ABNORMALITY
7	0	0	1	0	0	0	0	0	0	1	0	0		NO ST T CHANGES	NO RWMA, NORMAL CHAMBERS LVEF 60%	NO RADIOLOGICAL ABNORMALITY
8	0	0	1	0	0	0	0	0	0	1	0	0		NO ST T CHANGES	NO RWMA, NORMAL CHAMBERS LVEF 60%	NO RADIOLOGICAL ABNORMALITY
9	0	0	1	0	0	0	0	0	0	1	0	0		NO ST T CHANGES	NO RWMA, NORMAL CHAMBERS LVEF 60%	NO RADIOLOGICAL ABNORMALITY
10	0	0	1	0	0	0	0	0	0	1	0	0	BOTH COMMON CAROTID NORMAL FLOW , NORMAL IMTIMAL THICKNESS, NO STENOSIS , VERTEBRAL ARTERIES NORMAL	NO ST T CHANGES	NO RWMA, NORMAL CHAMBERS LVEF 60%	NO RADIOLOGICAL ABNORMALITY
11	0	0	1	0	0	0	0	0	0	1	0	0		NO ST T CHANGES	NO RWMA, NORMAL CHAMBERS LVEF 60%	NO RADIOLOGICAL ABNORMALITY
12	0	0	1	0	0	0	0	0	0	0	1	0		NO ST T CHANGES	NO RWMA, NORMAL CHAMBERS LVEF 60%	NO RADIOLOGICAL ABNORMALITY
13	0	0	1	0	0	0	0	0	0	0	1	0		NO ST T CHANGES	NO RWMA, NORMAL CHAMBERS LVEF 60%	NO RADIOLOGICAL ABNORMALITY
14	0	0	1	0	0	0	0	0	1	0	0	0		NO ST T CHANGES	NO RWMA, NORMAL CHAMBERS LVEF 60%	NO RADIOLOGICAL ABNORMALITY
15	0	0	1	0	0	0	0	0	0	0	1	0		NO ST T CHANGES	NO RWMA, NORMAL CHAMBERS LVEF 60%	NO RADIOLOGICAL ABNORMALITY
16	0	0	1	1	0	0	0	0	0	1	0	0		NO ST T CHANGES	NO RWMA, NORMAL CHAMBERS LVEF 60%	NO RADIOLOGICAL ABNORMALITY
17	0	0	1	0	0	0	0	0	0	1	0	0	BOTH COMMON CAROTID NORMAL FLOW , NORMAL IMTIMAL THICKNESS, NO STENOSIS , VERTEBRAL ARTERIES NORMAL	NO ST T CHANGES	NO RWMA, NORMAL CHAMBERS LVEF 60%	NO RADIOLOGICAL ABNORMALITY
18	0	0	0	1	0	0	0	0	0	0	0	1		NO ST T CHANGES	NO RWMA, NORMAL CHAMBERS LVEF 60%	NO RADIOLOGICAL ABNORMALITY
19	0	0	1	0	0	0	0	0	0	0	1	0		NO ST T CHANGES	NO RWMA, NORMAL CHAMBERS LVEF 60%	NO RADIOLOGICAL ABNORMALITY
20	0	0	0	0	0	0	1	0	1	0	0	0	-	NO ST T CHANGES	NO RWMA, NORMAL CHAMBERS LVEF 60%	NO RADIOLOGICAL ABNORMALITY
21	0	0	1	0	0	0	0	0	0	1	0	0		NO ST T CHANGES	NO RWMA, NORMAL CHAMBERS LVEF 60%	NO RADIOLOGICAL ABNORMALITY
22	0	1	1	0	0	0	0	0	1	0	0	0	-	NO ST T CHANGES	NO RWMA, NORMAL CHAMBERS LVEF 60%	NO RADIOLOGICAL ABNORMALITY
23	0	0	1	0	0	0	0	0	1	0	0	0	-	NO ST T CHANGES	NO RWMA, NORMAL CHAMBERS LVEF 60%	NO RADIOLOGICAL ABNORMALITY
24	0	0	1	0	0	0	0	0	0	1	0	0		NO ST T CHANGES	NO RWMA, NORMAL CHAMBERS LVEF 60%	NO RADIOLOGICAL ABNORMALITY
25	0	0	1	0	0	0	0	0	0	1	0	0		NO ST T CHANGES	NO RWMA, NORMAL CHAMBERS LVEF 60%	NO RADIOLOGICAL ABNORMALITY
26	0	0	0	1	0	0	0	0	0	0	0	1	BOTH COMMON CAROTID NORMAL FLOW , NORMAL IMTIMAL THICKNESS, NO STENOSIS , VERTEBRAL ARTERIES NORMAL	NO ST T CHANGES	NO RWMA, NORMAL CHAMBERS LVEF 60%	NO RADIOLOGICAL ABNORMALITY
27	0	0	1	0	0	0	0	0	0	1	0	0		NO ST T CHANGES	NO RWMA, NORMAL CHAMBERS LVEF 60%	NO RADIOLOGICAL ABNORMALITY
28	0	0	0	1	0	0	0	0	0	0	1	0		NO ST T CHANGES	NO RWMA, NORMAL CHAMBERS LVEF 60%	NO RADIOLOGICAL ABNORMALITY
29	0	1	1	0	0	0	0	0	1	0	0	0	-	NO ST T CHANGES	NO RWMA, NORMAL CHAMBERS LVEF 60%	NO RADIOLOGICAL ABNORMALITY
30	0	0	1	0	0	0	0	0	0	1	0	0	DIFFUSE ATHEROSCLEROTIC CHANGES IN B/L CAROTID ARTERY < 50% STENOSIS , INCREASED INTIMAL THICKNESS VERTEBRAL ARTERY NORMAL FLOW ,DIRECTION	NO ST T CHANGES	NO RWMA, NORMAL CHAMBERS LVEF 60%	NO RADIOLOGICAL ABNORMALITY
31	0	0	1	0	0	0	0	0	0	1	0	0		NO ST T CHANGES	NO RWMA, NORMAL CHAMBERS LVEF 60%	NO RADIOLOGICAL ABNORMALITY
32	0	0	1	0	0	0	0	0	1	0	0	0	-	NO ST T CHANGES	NO RWMA, NORMAL CHAMBERS LVEF 60%	NO RADIOLOGICAL ABNORMALITY
33	0	0	1	0	0	0	0	0	1	0	0	0		NO ST T CHANGES	NO RWMA, NORMAL CHAMBERS LVEF 60%	NO RADIOLOGICAL ABNORMALITY
34	0	0	1	0	0	0	0	0	0	1	0	0		NO ST T CHANGES	NO RWMA, NORMAL CHAMBERS LVEF 60%	NO RADIOLOGICAL ABNORMALITY
35	0	0	1	1	0	0	0	0	0	1	0	0	BOTH COMMON CAROTID NORMAL FLOW , NORMAL IMTIMAL THICKNESS, NO STENOSIS , VERTEBRAL ARTERIES NORMAL	NO ST T CHANGES	NO RWMA, NORMAL CHAMBERS LVEF 60%	NO RADIOLOGICAL ABNORMALITY
36	0	0	1	0	0	0	0	0	0	1	0	0		NO ST T CHANGES	NO RWMA, NORMAL CHAMBERS LVEF 60%	NO RADIOLOGICAL ABNORMALITY
37	0	0	0	1	0	0	0	0	0	0	0	1		NO ST T CHANGES	NO RWMA, NORMAL CHAMBERS LVEF 60%	NO RADIOLOGICAL ABNORMALITY
38	0	1	1	0	0	0	0	0	1	0	0	0		NO ST T CHANGES	NO RWMA, NORMAL CHAMBERS LVEF 60%	NO RADIOLOGICAL ABNORMALITY
39	0	1	1	0	0	0	0	0	1	0	0	0	-	NO ST T CHANGES	NO RWMA, NORMAL CHAMBERS LVEF 60%	NO RADIOLOGICAL ABNORMALITY
40	0	0	1	0	0	0	0	0	1	0	0	0		NO ST T CHANGES	NO RWMA, NORMAL CHAMBERS LVEF 60%	NO RADIOLOGICAL ABNORMALITY
41	0	0	1	0	0	0	0	0	0	1	0	0		NO ST T CHANGES	NO RWMA, NORMAL CHAMBERS LVEF 60%	NO RADIOLOGICAL ABNORMALITY
42	0	0	1	0	0	0	0	0	0	1	0	0		NO ST T CHANGES	NO RWMA, NORMAL CHAMBERS LVEF 60%	NO RADIOLOGICAL ABNORMALITY
43	0	0	0	0	0	0	1	0	0	0	0	1		NO ST T CHANGES	NO RWMA, NORMAL CHAMBERS LVEF 60%	NO RADIOLOGICAL ABNORMALITY
44	0	0	1	0	0	0	0	0	1	0	0	0	-	NO ST T CHANGES	NO RWMA, NORMAL CHAMBERS LVEF 60%	NO RADIOLOGICAL ABNORMALITY
45	0	0	1	0	0	0	0	0	1	0	0	0	BOTH COMMON CAROTID NORMAL FLOW , NORMAL IMTIMAL THICKNESS, NO STENOSIS , VERTEBRAL ARTERIES NORMAL	NO ST T CHANGES	NO RWMA, NORMAL CHAMBERS LVEF 60%	NO RADIOLOGICAL ABNORMALITY
46	0	0	0	0	0	0	1	0	0	0	0	1		NO ST T CHANGES	NO RWMA, NORMAL CHAMBERS LVEF 60%	NO RADIOLOGICAL ABNORMALITY
47	0	0	1	0	0	0	0	0	0	1	0	0		NO ST T CHANGES	NO RWMA, NORMAL CHAMBERS LVEF 60%	NO RADIOLOGICAL ABNORMALITY
48	0	0	1	0	0	0	0	0	0	1	0	0		NO ST T CHANGES	NO RWMA, NORMAL CHAMBERS LVEF 60%	NO RADIOLOGICAL ABNORMALITY
49	0	0	0	1	0	0	0	0	0	0	0	1	BOTH COMMON CAROTID NORMAL FLOW , NORMAL IMTIMAL THICKNESS, NO STENOSIS , VERTEBRAL ARTERIES NORMAL	NO ST T CHANGES	NO RWMA, NORMAL CHAMBERS LVEF 60%	NO RADIOLOGICAL ABNORMALITY
50	0	0	0	1	0	0	0	0	0	0	0	1		NO ST T CHANGES	NO RWMA, NORMAL CHAMBERS LVEF 60%	NO RADIOLOGICAL ABNORMALITY
51	0	0	0	1	0	0	0	0	0	0	0	1		NO ST T CHANGES	NO RWMA, NORMAL CHAMBERS LVEF 60%	NO RADIOLOGICAL ABNORMALITY
52	0	0	1	0	0	0	0	0	0	0	1	0		NO ST T CHANGES	NO RWMA, NORMAL CHAMBERS LVEF 60%	NO RADIOLOGICAL ABNORMALITY
53	0	1	0	0	0	0	0	0	1	0	0	0		NO ST T CHANGES	NO RWMA, NORMAL CHAMBERS LVEF 60%	NO RADIOLOGICAL ABNORMALITY
54	0	1	1	0	0	0	0	0	0	0	1	0	DIFFUSE ATHEROSCLEROTIC CHANGES WITH INCREASED INTIMOMEDIAL THICKNESS LEFT ICA 60% STENOSIS	NO ST T CHANGES	NO RWMA, NORMAL CHAMBERS LVEF 60%	NO RADIOLOGICAL ABNORMALITY
55	0	0	0	1	0	1	0	0	1	0	0	0	-	NO ST T CHANGES	NO RWMA, NORMAL CHAMBERS LVEF 60%	NO RADIOLOGICAL ABNORMALITY
56	0	0	1	0	0	0	0	0	1	0	0	0		NO ST T CHANGES	NO RWMA, NORMAL CHAMBERS LVEF 60%	NO RADIOLOGICAL ABNORMALITY
57	0	0	1	0	0	0	0	0	0	1	0	0		NO ST T CHANGES	NO RWMA, NORMAL CHAMBERS LVEF 60%	NO RADIOLOGICAL ABNORMALITY
58	0	1	1	0	0	0	0	0	1	0	0	0		NO ST T CHANGES	NO RWMA, NORMAL CHAMBERS LVEF 60%	NO RADIOLOGICAL ABNORMALITY
59	0	0	0	1	0	0	0	0	0	0	0	1	BOTH COMMON CAROTID NORMAL FLOW , NORMAL IMTIMAL THICKNESS, NO STENOSIS , VERTEBRAL ARTERIES NORMAL	NO ST T CHANGES	NO RWMA, NORMAL CHAMBERS LVEF 60%	NO RADIOLOGICAL ABNORMALITY
60	0	0	1	1	0	0	0	0	1	0	0	0	-	NO ST T CHANGES	NO RWMA, NORMAL CHAMBERS LVEF 60%	NO RADIOLOGICAL ABNORMALITY
61	0	0	0	1	0	0	0	0	0	0	0	1	DIFFUSE ATHEROSCLEROTIC CHANGES IN VERTEBRAL ARTERY , HYPERECHOIC PLAQUE IN BOTH CAROTID 32% IN RIGHT AND 21% LEFT STENOSIS	NO ST T CHANGES	NO RWMA, NORMAL CHAMBERS LVEF 60%	NO RADIOLOGICAL ABNORMALITY
62	0	0	0	1	0	0	0	0	0	0	0	1	BOTH COMMON CAROTID NORMAL FLOW , NORMAL IMTIMAL THICKNESS, NO STENOSIS , VERTEBRAL ARTERIES NORMAL	NO ST T CHANGES	NO RWMA, NORMAL CHAMBERS LVEF 60%	NO RADIOLOGICAL ABNORMALITY
63	0	0	1	0	0	0	0	0	0	1	0	0		NO ST T CHANGES	NO RWMA, NORMAL CHAMBERS LVEF 60%	NO RADIOLOGICAL ABNORMALITY
64	0	1	0	0	0	0	0	0	1	0	0	0	-	NO ST T CHANGES	NO RWMA, NORMAL CHAMBERS LVEF 60%	NO RADIOLOGICAL ABNORMALITY
65	0	0	1	0	0	0	0	0	0	1	0	0		NO ST T CHANGES	NO RWMA, NORMAL CHAMBERS LVEF 60%	NO RADIOLOGICAL ABNORMALITY
66	1	1	1	0	0	0	0	0	1	0	1	0		NO ST T CHANGES	NO RWMA, NORMAL CHAMBERS LVEF 60%	NO RADIOLOGICAL ABNORMALITY
67	1	1	1	0	0	0	0	0	1	1	0	0		NO ST T CHANGES	NO RWMA, NORMAL CHAMBERS LVEF 60%	NO RADIOLOGICAL ABNORMALITY
68	0	0	1	0	0	0	0	0	0	0	0	0		NO ST T CHANGES	NO RWMA, NORMAL CHAMBERS LVEF 60%	NO RADIOLOGICAL ABNORMALITY

SL NO	NIHSS					BARTHEL INDEX					PROCALCITONIN	MODIFIED RANKIN SCORE				
SL NO	D0	D4	DISCHARGE	1 MONTH	3 MONTH	D0	D4	DISCHARGE	1 MONTH	3 MONTH	D0	D0	D4	DISCHARGE	1 MONTH	3 MONTHS
2	16	16	16	12	12	0	10	10	15	40	4	5	5	5	5	4
3	14	12	12	12	12	0	10	10	15	40	2.05	5	5	5	5	4
4	17	17	14	12	12	0	10	10	15	45	6.07	5	5	5	5	4
5	18	18	18	DEATH		0	0	0	0	0	7.35	5	5	5	6	6
6	14	14	12	12	12	0	10	10	15	45	0.21	5	5	5	5	4
7	14	14	12	12	12	0	10	15	15	45	0.37	5	5	5	5	4
8	14	14	14	12	10	0	10	15	15	50	0.24	5	5	5	5	4
9	15	13	13	13	10	0	15	15	30	60	0.69	5	5	5	5	4
10	5	3	3	2	1	90	90	90	95	100	0.09	4	4	3	3	1
11	14	14	12	12	12	0	10	10	20	35	7.68	5	5	5	5	4
12	5	4	4	3	3	70	75	75	90	90	1.12	3	3	3	2	2
13	6	4	4	4	2	30	45	45	60	90	1.51	3	3	3	2	2
14	14	14	12	12	12	0	10	15	30	60	8.34	5	5	5	4	3
15	12	12	12	10	10	0	15	15	30	45	2.1	5	5	5	4	4
16	15	15	15	15	DEATH	0	5	5	5	0	7.36	5	5	5	5	6
17	9	9	9	8	5	0	10	10	10	40	0.25	5	5	5	4	4
18	3	1	1	0	0	90	95	95	100	100	0.21	4	3	3	1	0
19	8	8	8	7	4	10	30	30	50	70	0.33	5	5	5	4	3
20	27	DEATH	DEATH			0	0	0	0	0	9.9	5	6	6	6	6
21	8	8	8	6	4	10	30	30	45	60	0.62	5	4	4	4	3
22	18	18	18	DEATH		0	0	0	0	0	9.07	5	5	5	6	6
23	16	DEATH	DEATH			0	0	0	0	0	4.78	5	6	6	6	6
24	14	6	6	4	4	0	25	30	70	70	2.63	5	4	4	3	3
25	14	14	14	10	8	0	0	10	35	45	4.3	5	5	5	4	4
26	5	5	5	4	2	90	90	90	95	95	0.49	4	4	3	3	1
27	17	17	15	15	15	0	0	0	10	30	5.84	5	5	5	5	4
28	5	4	4	3	3	65	65	65	75	90	2.84	4	4	4	3	3
29	18	DEATH	DEATH			0	0	0	0	0	8.1	5	6	6	6	6
30	8	8	8	6	5	10	10	20	60	60	1.88	5	5	5	4	3
31	12	12	10	10	10	0	20	20	40	40	0.34	5	5	5	4	4
32	23	23	20	20	DEATH	0	0	10	10	0	2.02	5	5	5	5	6
33	14	12	12	12	10	0	15	15	15	20	0.36	5	5	5	5	5
34	8	8	8	5	4	10	30	30	60	65	<0.05	5	5	5	4	4
35	8	8	8	6	5	10	10	30	60	80	0.36	5	5	5	4	3
36	10	10	8	8	6	10	60	70	70	80	2.83	5	4	4	4	3
37	6	18	18	18	DEATH	0	0	5	5	0	12.38	5	5	5	5	6
38	18	18	18	18	18	0	0	5	5	10	11.33	5	5	5	5	5
39	28	DEATH	DEATH			0	0	0	0	0	0.62	5	6	6	6	6
40	14	14	14	14	14	0	0	10	10	10	5.32	5	5	5	5	5
41	16	16	16	DEATH		0	0	10	0	0	10.19	5	5	5	6	6
42	14	14	14	12	10	0	5	5	20	30	0.55	5	5	5	5	5
43	3	3	3	2	2	60	60	60	75	75	0.29	5	4	4	4	3
44	21	DEATH	DEATH			0	0	0	0	0	14.45	5	6	6	6	6
45	14	14	14	14	12	0	5	5	5	10	7.35	5	5	5	5	5
46	2	2	2	0	0	95	100	100	100	100	0.29	4	0	0	0	0
47	19	19	19	DEATH		0	0	0	0	0	13.02	5	5	5	5	6
48	18	18	18	18	DEATH	5	5	5	5	0	5.05	5	5	5	5	6
49	2	2	2	2	2	95	95	95	95	95	0.91	3	2	2	2	2
50	7	7	7	7	4	15	15	50	50	60	<0.05	5	5	4	3	3
51	3	2	2	2	1	85	85	85	95	95	<0.05	3	3	3	2	2
52	2	2	2	0	0	90	95	95	100	100	0.25	3	2	2	0	0
53	23	23	23	DEATH		0	0	0	0	0	19.25	5	5	5	6	6
54	8	8	6	6	4	55	55	55	60	65	0.13	4	3	3	3	2
55	22	DEATH	DEATH			0	0	0	0	0	37.87	5	6	6	6	6
56	19	19	19	17	17	0	0	0	10	10	10.19	5	5	5	5	5
57	11	11	10	10	10	0	40	40	60	65	6.29	5	4	4	3	3
58	18	18	18	14	14	0	0	0	5	5	0.68	5	5	5	5	5
59	2	2	2	2	2	85	95	95	95	95	0.1	4	3	3	2	2
60	27	DEATH	DEATH			0	0	0	0	0	27.02	5	6	6	6	6
61	3	3	3	2	2	60	65	65	80	90	0.15	5	4	4	3	2
62	4	4	4	2	0	80	80	80	90	100	0.13	3	3	3	1	0
63	14	14	12	12	12	5	10	10	35	35	<0.05	5	5	5	5	4
64	30	DEATH	DEATH			0	0	0	0	0	54.3	5	6	6	6	6
65	14	14	12	12	10	5	10	10	30	65	0.12	5	5	5	4	4
66	27	27	DEATH			0	0	0	0	0	9.42	5	5	6	6	6
67	24	21				0	0	.	.	.	4.65	5	5			
68	26	25				0	0	.	.	.	7.21	5	5			