"SIRIRAJ AND ALLEN'S SCORE IN THE DIFFERENTIAL DIAGNOSIS OF ACUTE STROKE AND CORRELATION WITH COMPUTED TOMOGRAPHY SCAN OF BRAIN"

By

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Dissertation submitted to BLDE (Deemed to be University), Vijayapura

In partial fulfilment of the requirements for the award of the degree of

DOCTOR OF MEDICINE IN

GENERAL MEDICINE

Under the guidance of

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VIJAYAPURA, KARNATAKA.

2020

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ACKNOWLWDGEMENT

"Great things are not done by one person. They are done by a team of people"

-Steve Jobs

Foremost, I would like to express my sincere gratitude to my teacher and guide **DR.R.C.BIDRI**$_{\textbf{M.D}}$, Professor of medicine for his continuous support and guidance. His dynamism, vision, discipline, sincerity and motivation have deeply inspired me. It was a great privilege and honour to work and study under his guidance. I'm grateful for having him as my mentor. Also I'm forever indebted for the care, affection and help that I have constantly received from him and his family. His expert and vigilant supervision, immense knowledge and expertise have helped me to bring out this work in the present form.

My sincere thanks are due to **Dr. M S BIRADAR** $_{\textbf{M.D}}$ .Vice Chancellor, **Dr. Arvind Patil** $_{\textbf{M.S}}$ Principal, & **Dr. BADIGER SHARANABASAWAPPA** $_{\textbf{M.D}}$ Professor & HOD, Department of General Medicine, Shri B. M. Patil Medical College, Vijayapura, for permitting me to conduct this study. I wish to acknowledge my Professors and take this opportunity to express my deep sense of gratitude and sincere thanks to **Dr. S.N. BENTOOR, Dr. P.G.MANTUR, Dr. M S MULIMANI, Dr. L S PATIL, Dr. R.M. HONNUTAGI, Dr. S.S.DEVARMANI, Dr. ANAND P AMBALI, Dr. VIJAYKUMAR WARAD** and **Dr. S.M. BIRADAR** for their supervision and timely advice.

I am also thankful for the support extended by **Dr REHAAN INAMDAR, Dr. S.S. PATIL, Dr. RAVI KATTIMANI, Dr AFAQUE INAMDAR, Dr BASANGOUDA, Dr ANUJA.M.K, and Dr. GURU.**

My sincere thanks to the staff members of the Department of Medicine, **Mr GUMASTE, Mr GALGALI** and **Mr LAXMAN.**

I am forever grateful to my parents, **Mrs ASHA** and **Dr. RAJU** for their love, prayers and sacrifices for educating and preparing me for my future. I am also thankful to my sister **NEHA,** for always being there for me.

I would also extend my thanks to my friends **SURABHI, Dr.LEELA, Dr.ROOPA, Dr.JAYANTH, Dr.SAGAR, Dr.NAFASAT,** and my colleagues for their constant support and encouragement.

I am also thankful to **Dr. VIJAYA SORGANVI** for her help in making this thesis and statistical works.

I would be failing in my duty, if I would not acknowledge my thanks to all the patients who were kind enough to help for this study.

-Dr. NAYANA. R

LIST OF ABBREVATIONS USED

ABC's - AIRWAY, BREATHING, CARDIAC STATUS.

ACA - ANTERIOR CEREBRAL ARTERY

AoCA-ANTERIOR COMMUNICATING ARTERY

ADL -ACTIVITIES OF DAILY LIVING

AF - ATRIAL FIBRILLATION

BBB - BLOOD BRAIN BARRIER

BP - BLOOD PRESSURE

CCA- COMMON CAROTID ARTERY

CVA - CEREBRO VASCULAR ACCIDENT

DBP - DIASTOLIC PRESSURE

DM - DIABETES MELLITUS

DSY-DYSLIPEDEMIA

GCS - GLASGOW COMA SCALE

HDL - HIGH DENSITY LIPOPROTEINS

ICP - INTRA CRANIAL PRESSURE

IHD -ISCHEMIC HEART DISEASE

ICA -INTERNAL CAROTID ARTERY

ICH -INTRA CEREBRAL HEMORRHAGE

LDL -LOW DENSITY LIPOPROTEINS

MCA -MIDDLE CEREBRAL ARTERY

MI- MYOCARDIAL INFARCTION

NINDS -NATIONAL INSTITUTE OF NEUROLOGICAL DISORDERS AND STROKE

OCP -ORAL CONTRACEPTIVE PILLS

PACS -PARTIAL ANTERIOR CIRCULATION STROKE

PCA -POSTERIOR CEREBRAL ARTERY

PoCA-POSTERIOR COMMUNICATING ARTERY

POCS -POSTERIOR CIRCULATION STROKE

rtPA -RECOMBINANT TISSUE PLASMINOGEN ACTIVATOR

RHD -RHEUMATIC HEART DISEASE

RIND- REVERSIBLE ISCHEMIC NEUROLOGIC DEFICIT

SAH -SUB ARACHNOID HEMORRHAGE

SHT -SYSTEMIC HYPERTENSION

SBP -SYSTOLIC BLOOD PRESSURE

TACS -TOTAL ANTERIOR CIRCULATION STROKE

TG- TRIGLYCERIDES

TIA -TRANSIENT ISCHEMIC ATTACK

ABSTRACT

NEED FOR THE STUDY:

According to the World Health Organization (WHO) stroke is defined as, "rapidly developing clinical signs of focal (or global) disturbance of cerebral function, lasting for more than 24 hours or leading to death, with no apparent cause, other than that of vascular origin."

It is associated with increased long term mortality; stroke is the most common cause of death after cardiac disease and cancer. They cause around 200,000 deaths in United States and 334-424/ 100,100 deaths each year in India and a major cause of disability. About 15% to 25% of stroke survivors become disabled permanently, while 20% remain in institutional care for three months after their stroke. The incidence of stroke increases with age and the number of strokes is projected to increase as the elderly population grows, with a doubling in stroke deaths in the India by 2030.

Cerebrovascular disorders are increasing in prevalence and incidence in India due to rapid escalation of risk factors including Hypertension, Diabetes Mellitus, Smoking and obesity affecting considerable proportion of adult population.

Physicians who manage stroke would like to know with certainty, the type of lesion they are dealing with, that is if it is hemorrhagic or ischemic, as the modality of treatment approach differs. Traditional methods of diagnosis have been reportedly found to be inaccurate following the advent of Computed Tomography (CT) scan. The unavailability of CT scan at most centers in

developing countries highlights the need for a more reliable method of bedside diagnosis of differentiating between hemorrhagic and ischemic stroke. It is to meet this need particularly at the level of a primary care physician that, two diagnostic methods have been devised; the Allen's scoring system and the Siriraj stroke score.

METHODS:

It is an observational study. The study was conducted on 57 patients in Shri B M Patil Medical College hospital who were diagnosed and admitted with acute stroke on the basis of the History, Clinical examination and proved on CT/MRI scan. Patients were selected on the basis of the inclusion and exclusion criteria. Siriraj and Allen's score is noted on the day of admission, 24 hours after admission, the scores are calculated for each patient, simultaneously the CT scan findings are also noted and the results are statistically analyzed. This study was conducted from October 2018 to July 2020.

RESULTS:

CT scan revealed cerebral infarction in 73.7(%) of patients and cerebral hemorrhage in 26.3(%) of cases. Sensitivity of Siriraj Score is 96.45% for infarction and 95% for hemorrhage and its overall accuracy is 95.15%. The Allen's score had a sensitivity of 93.3% for infarction and 81.4% for hemorrhage. The overall predictive accuracy is 94.74%.

CONCLUSION:

Siriraj and Allen's score together has greater sensitivity for diagnosis of infarction and hemorrhage when considered together in the same patient.

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INTRODUCTION

Acute cerebrovascular disease has been known to physicians even before the time of Hippocrates.

Hippocrates, the father of medicine, first recognized stroke over 2400 years ago. At the time stroke was called apoplexy, which means "struck down by violence" in Greek. This was due to the fact that a person developed sudden paralysis and change in well-being.

Soranus of Ephesus (AD 98-138) wrote that "…. hemiplegic paralysis was common in old age, seldom occurred in youth and came often most in winter…", at times for no apparent reason, at others from clear causes such as indulgence, injury or association with other conditions [(1).]

The historical events that took place are as follows –

1. The term "Apoplexy" can be followed from Antiquity ,passing through the middle ages and renaissance .The definition was stated by Hippocrates who took the word "Apoplexy" where it meant **"**Astonished**,** Sudden loss of one's senses**"**
2. Jacob Werter(1620-95), first person who was Swiss physician to suggest that apoplexy was caused by disease of blood vessel in the brain.
3. Thomas Willis described the circle of wills in 1664.
4. Seddicot described spontaneous intracerebral haemorrhage in 1813.
5. Abberonbie, in 1828 described the obliterative arterial diseases of brain.
6. Johan Friedrichcrell, emphasized the pultaceous or atheromatous elements in some arterial lesions although he did not use the term atheroma.
7. Von Haller made similar observations and applying the term "atheroma" to the arterial lesions.

8. In 1860, Rudolf Virchow described imbibition theory that states there was deposition of blood constituents on the laminal surface of the arterial wall during the formation and growth of atheromatous plaques. He considered that the early lesions of atherosclerosis were based on a "loosening" of the connective tissue ground substance of the intima as a result of "imbibition" of constituents of the passing blood.
9. Vogel in 1847 and Chalatow in 1913 observed that atherosclerotic plaques contained relatively large amounts of cholesterol.
10. In 1825, Bonillord described localisation of lesion and aphasia.
11. The term cerebrovascular accident was introduced in 1927.

DEFINITION:

- "Acute stroke is defined as abrupt onset of focal neurological deficit that is attributable to a focal vascular cause."[(2)]
- Transient ischemic attack (TIA) - The neurological signs and symptoms last less than 24 hours without evidence of brain infarction on brain imaging."
- "Reversible ischemic neurologic deficit (RIND) – is a type of stroke due to occlusion of blood supply to brain leading to ischemia and neurological deficits which recover from 24 hours and up to few weeks".
- Stroke in evolution -is an evolving stroke is one in which neurological deficits appear to worsen over a given period after the initial stroke occurs, with or without the presence of appropriate medical intervention."

IMPACT OF STROKE:

The direct and indirect cost of acute stroke in United States alone was approximated to be $56.8 million in the year 2005. Every year in the US more than 7,00,000 people have stroke, one third in that are recurrent events .There was about 6.2million stroke death in the year 2015, making it the second leading cause of death worldwide.[2]

According to the recent study conducted in India in 2017, the crude stroke prevalence in different parts of India ranged from 44.29 to 559 cases per 1 lakh population. The cumulative incidence of stroke in India ranged from 105 to 152 cases per 1 lakh persons per year during the past two decades in different parts of the country. [2, 63]

Strokes are even more important because of prolonged disability they cause. Stroke is the most frequent cause of adult-onset disability. The history of the world has undoubtedly been altered by stroke. Many important leaders in science, medicine and politics have had their productivity cut permanently or prematurely short by stroke [3].

Among the stroke survivors around 15% and 30% become permanently disabled, while 20% of them remain in institutional care three months after the stroke. The economical and psychological costs of stroke are enormous. 35% of survivors with initial paralysis of the leg do not regain useful function, and 20-25% of all survivors are unable to walk without full physical assistance.

A rule of thumb in stroke recovery is that patients with mild deficits are more likely to make a good recovery than patients with initially more severe deficits. The ‘proportional recovery rule’

assumes that patients can on average improve around 70% (+/- 15%) of their lost function within 3-6 months after stroke

Incidence of stroke has increased considerably in India and other developing countries. On an average, in comparison with high income countries stroke occurs 15 year earlier in and causes more deaths in low and middle economic countries.

The increasing incidence of stroke may be a reflection of - increased longevity, adoption of high fat diet, sedentary life style, increasing prevalence of diabetes mellitus and hypertension and central obesity.

Physicians managing stroke would like to know with certainty, the type of lesion they are dealing with. Is the lesion hemorrhagic or is it an infarct? Why is the clinical examination important?

The 2 fundamental subtypes of stroke are haemorrhagic stroke and ischemic stroke. 87% of these vascular strokes are ischemic and 13% are hemorrhagic Clinicians must rapidly distinguish these 2 vascular subtypes because they have distinct aetiologies, prognoses, and treatments.

In third world countries, like ours, where diagnostic facilities are insufficient and also where available; due to economic burden to family - utilization of such facilities are delayed but in contrary it is well established that management and prognosis of patients with acute stroke syndrome vary depending mainly on stroke subtypes: therefore it is necessary for timely differentiation between them. If stroke patients are to derive benefit from thrombolytic therapy and antiplatelet drugs, cerebral infarction needs to be diagnosed quickly and correctly.

Classic descriptions of stroke suggest that some clinical features may differentiate these subtypes: headache, neck stiffness, vomiting, and coma are more common in hemorrhagic stroke whereas previous transient ischemic attacks, atrial fibrillation, and atherosclerosis risk factors are more common in ischemic stroke.

Traditional methods of diagnosis have been repeatedly found to be inaccurate following the advent of CT scan. Hence, it is very much crucial for timely differentiation between the strokes subtypes clinically, with fair amount of accuracy, which would be of great help for timely diagnosis of such cases.

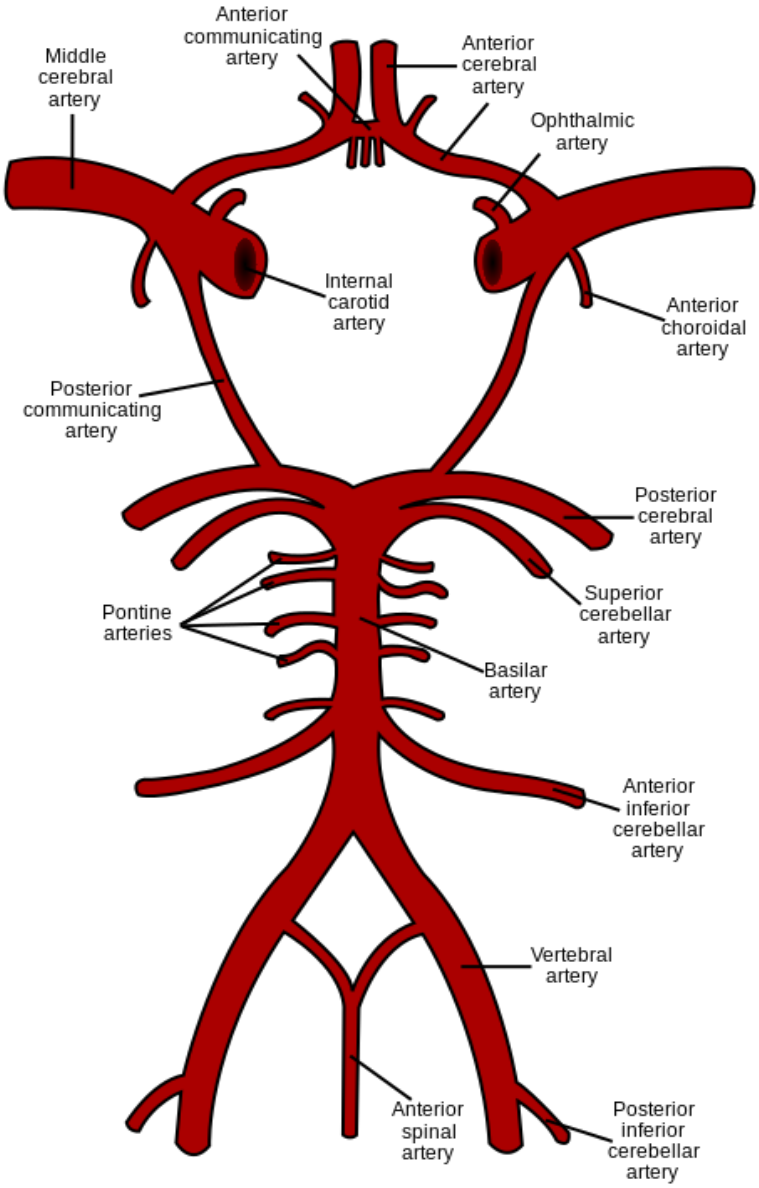
The unavailability of this facility at most centers in developing countries highlights the need for a more reliable method of bedside diagnosis. It is to meet this need particularly at the level of a primary care physician that two diagnostic methods have been devised; **the Allen's scoring system and the Siriraj stroke score.**

LITERARY RIVIEW OF ACUTE STROKE

ANATOMY OF CEREBRAL CIRCULATION

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

FIGURE 1: CIRCLE OF WILLIS



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

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

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

First branch of ICA is Ophthalmic artery, and it supplies the eye and other structures of orbit. Next artery from the the ICA is the Posterior communicating artery (PoCA). It joins posterior Cerebral Artery (PCA) at its first division. From last section of the ICA arises the anterior choroidal artery.

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

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

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

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

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

Collateral blood supply to the brain

In acute ischemic stroke, collateral circulation plays an important role in maintaining blood flow to the tissue that is at risk of progressing into ischemia, and in increasing the successful recanalization rate without hemorrhagic transformation.It has been reported that well-developed collateral circulation is associated with smaller infarct volume and better long-term neurological outcome, and it disappears promptly once the effective recanalization is achieved. Good collateral status leads to higher recanalization rate, smaller infarction volume, and better

neurological outcome.

Normally the anterior 2/3rd of the same side cerebral hemisphere is supplied by ICA. Little mixing of blood via the PoCA and so the posterior course is supplied by the PCA, basilar and vertebral artery. However there are various ways in which collateral blood supply to the brain can develop distal to occlusion of major arteries in the neck and head. The actual pattern of collateral blood flow depends on where the major blood vessels are occluded and on which collateral channels are anatomically available in a particular individual and which are free from disease.

Collateral blood flow development

The cerebral collateral circulation refers to the subsidiary network of vascular channels that stabilize cerebral blood flow when principal conduits fail.

Collateral blood vessels comprise extracranial routes including the facial, maxillary, middle meningeal, and occipital arteries, as well as intracranial routes. The intracranial collateral routes are divided into two pathways: the primary pathway includes the circle of Willis, whereas secondary pathways consist of pre-existing collateral routes that do not normally feed the territory but that develop in the setting of the impaired cerebral hemodynamics after acute stroke. These secondary pathways include the ophthalmic artery and leptomeningeal anastomoses with dural arterioles.

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

The revascularization after vascular occlusion involves several distinct processes.

'Angiogenesis,' the sprouting of endothelial cells to form capillary networks, is induced by

hypoxia: defective oxygenation of cells leads to the activation of hypoxia-inducible factor 1 and downstream transcription factors such as vascular endothelial growth factor, which bind to endothelial cells, thus signaling them to proliferate, migrate, and eventually form new vessels. In post-stroke angiogenesis, endothelial cells are activated, and in accordance with smooth muscle cells and pericytes, they work for functional and mature vascular formation.

Another process relating to revascularization in ischemic stroke is arteriogenesis, the induced development of new vessels triggered by fluid shear stress after the stenosis or occlusion of vessels. This increased shear stress results in the formation of large collateral arteries due to the proliferation of endothelial and smooth muscle cells. Once the hemodynamically relevant stenosis or occlusion occurs, pre-existing arterioles redistribute the blood flow by connecting high-perfusion and low-perfusion regions, thus increasing the shear stress in pre-existing arterioles and leading to the development of collateral vessels.

The robustness of the collateral circulation diminishes with age and other vascular comorbidities, such as hypertension and diabetes, hence more prevalent in patients with these comorbid conditions.

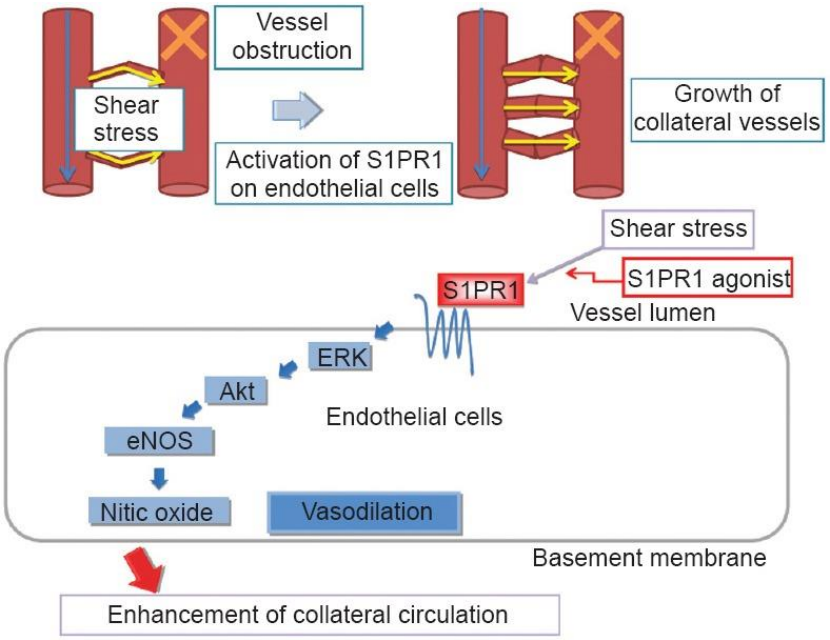


FIGURE 2: MECHANISM OF FORMATION OF COLLATERAL CIRCULATION

Vessel occlusion increases fluid shear stress, consequently up regulating and activating sphingosine-1-phosphate receptor 1 (S1PR1) and leading to the growth of collateral vessels presumably through the phosphorylation of extracellular signal-regulated kinases (ERK), protein kinase B (Akt), and endothelial nitric oxide synthase (eNOS) activation.[65]

Other areas of collateral blood flow are

- Around the orbit
- Leptomeningeal anastomoses
- Parenchymal anastomoses

Venous drainage

The venous compartment contains about 70%~80% of the circulatory volume inside the inflexible cranial cavity. The cerebral venous system can be divided into superficial and deep venous systems.

The superficial vein system drains cortical surfaces of both cerebral hemispheres. Superficial

veins drain mainly into four groups, according to their territory: superior sagittal sinus, sphenoparietal sinus and cavernous sinus, inferior sagittal sinus and Galen vein along with tributaries of the sinuses. The deep system comprises deep cortical draining veins, the lateral sinus, straight sinus and sigmoid sinus, which are concerned with the drainage of the central structures of the hemispheres, basal ganglia, corpus callosum, pineal region limbic system and thalamus.

Both two systems afflux into the internal jugular veins.

The cerebral venous system contains important structural differences from the cerebral artery system. First, small veins and venules do not have encompassing smooth muscle cells. Thus, they cannot contract as strongly as arteries. The venule is a crucial contributor to cerebrovascular resistance. During pathological conditions, the veins may be easily compressed and even flattened by high ICP. Increases in central venous pressure or intracranial venous pressure can lead to an increase in the hydrostatic pressure of upstream cerebral veins and reduce the blood outflow. The blood flow of cerebral veins is often in the same direction as in neighboring arteries. [(4)]

Secondly, compared with arteries, cerebral veins have thin walls and possess no valves to prevent the back flow of venous blood. Because both higher blood pressure and more rapid blood flow occur in arteries than in veins, the risk of thrombosis is much higher in veins.

The harmony between the arterial blood inflow and venous output is crucial for brain homeostasis.

However, brain ischemic events, brain infarct, and venous hemorrhage occur approximately in 10% to 75% of patients with cerebral venous hypertension, all of which are endpoints of elevated cerebral venous pressure and cause detrimental effects on the homeostatic environment.

Once the reduction of venous flow is unmatched with an increased arterial flow, venous congestion may occur and catastrophic events, such as capillary occlusion, collapse, or hemorrhage follows.

EPIDEMIOLOGY OF STROKE

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

Stroke is the leading cause of disability in adults. Stroke poses major socioeconomic challenge in rehabilitation of survivors. "Out of thousands of stroke survivors each year, 30% require assistance with activities of daily living, 20% require assistance with ambulation and 16% require institutional care. The human and financial cost of stroke is immense and its estimated annual economic impact in our society, both directly in health care and indirectly in lost income is approximately $41billion."[(5)]

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

fear that it may begin to rise. In Sweden, there was a 38% increase in stroke among women from 1975- 78 and 1983- 85. [6]

Epidemiology of stroke in India

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

Analysis of data from major urban university hospitals suggested that nearly 2% of all hospital cases, 4.5% of medical and 20% of neurological admissions are from stroke.[7]

In early 1980's, in Asia the stroke prevalence was around 500-700 per 1,00,000 in the western countries and 900 per 1,00,000[8]

A recent study conducted in 2017 showed that the crude stroke prevalence in different parts of India ranged from 44.29 to 559/100,000 persons during the past two decades. The cumulative incidence of stroke in India ranged from 105 to 152/100,000 persons per year during the past two decades in different parts of the country.

The annual incidence rate of stroke ranged from 105-262/1,00,000 population with the incidence falling within the range reported in the west i.e,100-300/1,00,000 population. The subtypes of strokes were infarction in 68% and haemorrhagic in 32%.The cerebral infarct to haemorrhage ratio was 2.21. Haemorrhage cases were more compared to western countries. [9,10]

The stroke is the cause for 1.2% of total deaths in the country, including all ages with gender ratio M:F=1.24.[11] The stroke death increased with age proportionately and 2.4% of all deaths were in the age group of >70years.

Diabetes mellitus, hypertension, tobacco use and low haemoglobin were the most Important risk factors for ischemic stroke. [7]

RISK FACTORS FOR STROKE

1. **Non modifiable risk factors**

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

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

    Heredity- Increased incidence of stroke is noted in families.

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

2. **Modifiable risk factors**

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

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

BP causes 46% decline in stroke and treatment of isolated systolic hypertension reduces stroke risk by 36%.About 40% of strokes can be attributed to systolic blood pressure of >140mmhg[7,12,13]

b) Smoking – The Framingham study showed compared to non-smokers, smokers have three fold increase of ischemic strokes .After the study data shows that smoking is hazardous to all forms of stroke like it is to the coronary heart disease.[14,15]

c) Diabetes mellitus- It increases the risk of stroke by 1.8-3.5 times.
Most of the ischemic strokes in them are lacunar strokes. It was the sixth most important predictive factor for stroke according to Framingham study.[16]

d) Blood lipids- Dyslipidemias increase the risk of stroke by 1-2 times. There is a higher incidence in patients with low level of high density lipoproteins (HDL) and high levels of low density lipoproteins (LDL) and triglycerides (TG)[13].The 20-30% relative risk reduction in stroke that occurs within 1-2 years after institution of statins is due to its pleotropic effect which improves the endothelial function, Plaque stabilisation, anti-thrombotic properties, diminished inflammation and improved hemorrheologic environment.

e) Obesity- The Whitehall study showed that "body mass index (BMI) was predictive of stroke in both smokers and non-smokers. It was estimated that having a BMI above 25kg/m2 and smoking accounts for 60% strokes in up to 65years"[4]

f) Cardiovascular diseases- According to Framingham study, "ECG changes of LVH increases the risk of ischemic stroke by tenfold; non-specific ST and T changes by

four fold and congestive cardiac failure by nine fold."[14,17] Mitral valve prolapse, prosthetic valves, endocarditis, peripheral vascular disease, MI, cardiac arrhythmias are the risk factors for embolic stroke.[18]

g) Alcohol-The risk is variable. In low to moderate consumption lowers overall mortality, while heavy consumption increases the risk of haemorrhage. Embolic strokes result from cardiac arrhythmias and cardiac wall motion abnormalities, hypertension, enhanced platelet aggregation and activation of clotting cascade, which are common in alcoholics.[19]

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

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

j) Oral contraceptives- Young women consuming oral contraceptives have risk for stroke and much more in those whose oestrogen content is more than 50mcg.[20]

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

Based on the findings of many different studies, the risk factors for stroke in India [21] (WHO Task Force, 1989) has been given below in a tabular form.

TABLE 1: RISK FACTORS OF STROKES IN INDIA (WHO TASK FORCE, 1989)

Risk Factors	All Strokes	Ischemic Stroke	Hemorrhagic Stroke
Hypertension	+	0	0
Systolic	+	+	+
Diastolic	+	+	+
Diabetes mellitus	0	0	0
Heart disease	+	+	0
Transient ischemic attack	0	0	0
Obesity	-	0	0
Platelet hyper-aggregability	0	0	0
Alcoholism	0	0	0
Smoking	-	-	-
Elevated blood lipid levels	+	+	+
Cholesterol	-	+	-
Triglycerides	-	+	-
Low density lipoproteins	0	0	0
Hyperuricemia	+	+	0
Infections	+	+	0
Genetic or familial factors	0	+	-
Migraine	0	0	0

Cold temperature	0	0	NM
High oestrogen contraceptives	0	0	NM
Socioeconomic status	0	0	0
Hematocrit	0	+	0
Increased fibrinogen	0	NM	0
Proteinuria	0	+	0
Sodium intake	0	+	0

CLASSIFICATION OF STROKE

1) ACCORDING TO PATHOGENESIS:

A. ISCHEMIC STROKES:

i. ATHEROTHROMBOSIS CAUSING CEREBRAL INFARCTION

Stroke is preceded by minor signs or one or more transient attacks of focal neurologic dysfunction. The thrombotic stroke syndrome develops in one of several ways. There may be a single episode, but typically the whole stroke evolves over a few minutes or hours or less. Atheromatous plaques preferentially form at branching points and curves of the cerebral arteries. The characteristic feature is a "stuttering" or intermittent progression of neurological deficits. The deficits may be episodic and evolves gradually- slow stroke

The most frequent sites are

(1) in the internal carotid artery; at its origin from the common carotid;

(2) in the cervical part of the vertebral arteries and at their junction to form the basilar artery

(3) in the stem or at the main bifurcation of the middle cerebral arteries

(4) in the proximal posterior cerebral arteries as they wind around the midbrain

(5) in the proximal anterior cerebral arteries as they pass anteriorly and curve over the corpus callosum.

The occlusive plaque or a thrombus formed on a plaque occupies the lumen of a major intracerebral vessel,such as the MCA and stops flow to those areas supplied by the artery. Occlusion of a more proximal vessel such as the distal carotid artery by atherosclerosis leads to infarction in the territory between major branches of the internal carotid circulation, most

susceptible to reduced blood flow - watershed infarction

Atherothrombosis in a proximal vessel serving as the nidus for an embolus- artery to artery embolism

ii. CEREBRAL EMBOLISM:

Most common cause of ischemic strokes and of all the types of stroke, cerebral embolism develops most rapidly, "like a bolt out of the blue."

The full-blown picture evolves within seconds. Abruptness with which the stroke develops and the lack of prodromal symptoms point strongly to embolism

The embolic material consists of a fragment that has broken away from a thrombus within the heart -"cardio-embolic"

The source also may be intra-arterial from the distal end of a thrombus within the lumen of an occluded or severely stenotic carotid or vertebral artery, or a clot that originates in the systemic venous system and passes through an aperture in the heart walls, or the origin of an embolus may be from large atheromatous plaques in the aorta.

Thrombotic or infected material (endocarditis) that adheres to the aortic or mitral heart valves and breaks free and the clots originating on prosthetic heart valves are also the sources of embolism,

Embolism caused by fat, tumor cells (atrial myxoma), fibrocartilage, amniotic fluid, or air enters into the differential diagnosis of stroke only in special circumstances.

The embolus usually becomes arrested at a bifurcation or other site of natural narrowing of the lumen of an intracranial vessel.

The territories of the middle cerebral artery, particularly the superior division are most frequently involved.

Because of the rapidity with which embolic occlusion develops, useful collateral influx does not become established.

iii. CEREBRAL VENOUS THROMBOSIS:

Occlusion of cortical veins that are the tributaries of the dural sinuses takes the form of a venous infarctive stroke.

The factors which favour the occurence of venous thrombosis are taking of birth control pills or postpartum and postoperative states, which are often characterized by thrombocytosis and hyperfibrinogenemia.

Hypercoagulable conditions also occur in cancer (particularly of the pancreas and colon and other adenocarcinomas), cyanotic congenital heart disease, sickle cell disease, antiphospholipid antibody syndrome, protein S or C deficiency, primary or secondary polycythemia.

A stroke in a patient suffering from any one of these systemic conditions should suggest venous thrombosis

Cortical vein thrombosis should be suspected in the situation of multiple haemorrhagic infarctions in one hemisphere without a source of embolism or atherothrombosis

iv. ARTERITIS:

Immunologic studies show that in most of these processes there is an abnormal deposit of complement-fixing immune-complex on the endothelium, leading to inflammation, vascular occlusion or rupture with small haemorrhage.

v. HYPERCOAGULABLE STATES:

Non-bacterial thrombotic endocarditis, consist of fibrin and platelets and are loosely attached to the mitral and aortic valves and contiguous endocardium. They are a common source of cerebral embolism.

In DIC there is the occurrence of widespread fibrin thrombi in small vessels, resulting in numerous small infarctions in the brain. In some cases, cerebral haemorrhage is quite extensive similar to a primary hypertensive haemorrhage.

Other conditions include Antiphospholipid Antibody Syndrome, Thrombotic Thrombocytopenic Purpura, Polycythemia Vera, Thrombocytosis and Thrombocythemia,

In the Sickle Cell Disease large and small ischemic lesions of the brain are the most common neurologic complications

vi. CEREBRAL ANOXIA:

This type of infarction follows cardiac arrest and other forms of prolonged hypotension or hypoxia.

Widespread cortical infarction also affecting the deep nuclei is seen, i.e., the most metabolically active regions of the cerebral hemispheres.

In the case of reduced blood flow to the cerebral hemispheres, there is a tendency for regional infarctions to occur in the areas of lowest blood flow that lie between the major surface arteries, referred to metaphorically as a watershed infarction.

Pure hypoxia-anoxia without hypotension produces another type of damage in areas susceptible to reduced oxygen delivery, mainly affecting the hippocampi.

vii. DISSECTION OF THE CERVICAL AND INTRACRANIAL ARTERIES:

Internal Carotid Artery Dissection: The ischemic manifestations consist of transient attacks in the territory of the internal carotid, followed frequently by the signs of hemispheral stroke, which may be abrupt or evolve smoothly over a period of minutes to hours or over several days in a fluctuating or stepwise fashion.

Intracranial Arterial Dissection: patients have had sudden strokes that suggested embolic infarction, and a small number present with subarachnoid hemorrhage.

vii. ANGIOGRAPHIC COMPLICATIONS:

During cerebral arteriography, emboli may arise from the tip of the catheter, or manipulation of the catheter may dislodge atheromatous material from the aorta or carotid or vertebral arteries and account for some of the strokes during this procedure.

B. CEREBRAL ISCHEMIA:

TRANSIENT ISCHEMIC ATTACKS:

'A brief episode of neurological dysfunction caused by focal brain or retinal ischaemia, with clinical symptoms typically lasting less than 1 hour, and without evidence of acute infarction' 20 percent of infarcts that follow TIAs occur within a month after the first attack, and approximately 50 percent within a year (Whisnant et al).

Most cases are intimately related to vascular stenosis and, usually, to ulceration as a result of atherosclerosis and thrombus formation.

The occurrence of Carotid TIA is a predictor for both cerebral infarction and myocardial infarction

Clinical Features- Definite/Probable TIA:

Symptoms suggesting carotid TIA

– Amaurosis fugax

– Speech deficit (aphasia)

– Unilateral motor and/or sensory symptoms affecting the face and/or limbs; these symptoms usually indicate ischaemia in the carotid territory.

Symptoms suggesting vertebrobasilar TIA

– Motor and/or sensory symptoms affecting the face and/or limbs, bilateral or changing sides between attacks

– Loss of vision in the right and left field of vision (cortical blindness); homonymous hemianopsia may also be seen in carotid TIAs.

C. HAEMORRHAGIC STROKE:

i. INTRACEREBRAL HEMORRHAGE:

Defined as 'Irruption of blood in the cerebral parenchyma'

Occurs when small (50–700 μm) penetrating arteries rupture with subsequent leaking of arterial blood into the brain parenchyma.

The most common sites of a cerebral haemorrhage are

(1) The putamen and adjacent internal capsule

(2) The central white matter of the temporal, parietal, or frontal lobes

(3) The thalamus

(4) One or the other cerebellar hemisphere

(5) The pons.

Multiple forms of oedema are present after ICH, but its main component is probably vasogenic. There are three phases of oedema formation after ICH:

(i) The very early phase (first few hours) which involves hydrostatic pressure and clot retraction with movement of serum from the clot into the surrounding tissue;

(ii) The second phase (first few days) in which the coagulation cascade and thrombin production (which also induces inflammatory cell infiltration, mesenchymal cell proliferation, and scar formation) play major roles;

(iii) The third phase, related to erythrocyte lysis and haemoglobin toxicity, which has been shown to trigger neurotoxic and apoptotic mechanisms. Another potential pathogenic mechanism is activation of leucocytes at the injury site within the first few days, attributable to the widespread inflammation.

ICH is a dynamic process and haematoma enlargement occurs in about one-third of patients, causing midline shift and accelerates neurological deterioration.

Clinical Features: Headache, Vomiting, Decreased level of consciousness, Seizures.

<u>Commonest causes of apparently ‘spontaneous’ intracerebral haemorrhage:</u>

- Cerebral amyloid angiopathy (lobar ICH)
- Haemorrhagic transformation of cerebral infarction
- Vascular malformations- Cerebral arteriovenous malformation, Intracranial arterial aneurysm, Cerebral cavernous malformation, Dural arteriovenous fistula
- Venous disease-Cerebral venous thrombosis

- Haematological diseases

ii.SUBARACHNOID HAEMORRHAGE:

Saccular aneurysms also have been called "berry" aneurysms. They take the form of small, thin-walled blisters protruding from arteries of the circle of Willis or its major branches. Their rupture causes a flooding of the subarachnoid space with blood under high pressure.

The aneurysms are located at vessel bifurcations and branching.

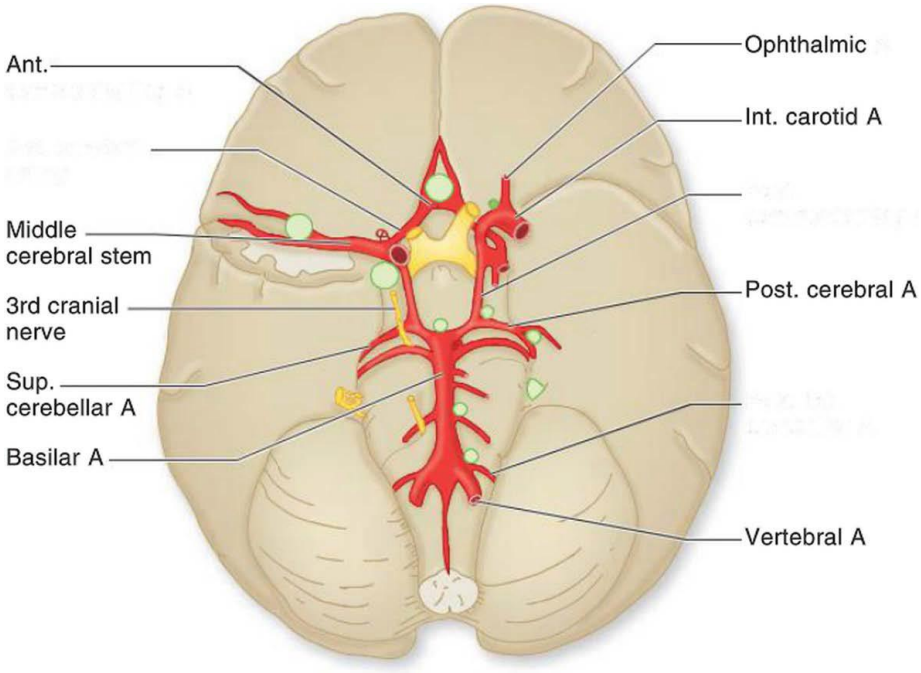
With rupture of the aneurysm, blood under high pressure is forced into the subarachnoid space and the resulting clinical events assume one of three patterns:

(1) The patient is stricken with an excruciating generalized headache and vomiting and falls unconscious almost immediately;

(2) Severe generalized headache develops in the same instantaneous manner but the patient remains relatively lucid with varying degrees of stiff neck-the most common syndrome

(3) Rarely, consciousness is lost so quickly that there is no preceding complaint.

FIGURE 3: PRINCIPAL SITES OF INTRACRANIAL ANEURYSMS.



Approximately 90 per cent of aneurysms arise from branches of the anterior half of the circle of Willis.

d) Stroke of undetermined origin:

i. Multi infarct dementia in lacunar syndrome
ii. Fibro muscular disease
iii. Winiwarter-Buerger disease
iv. Aortic arch syndrome

2) ETIOLOGICAL CLASSIFICATION:

i. Atherosclerotic thrombosis
ii. Transient ischemic attacks
iii. Embolism
iv. Ruptured or unruptured saccular aneurysm
v. Arteritis
vi. Cerebral thrombophlebitis
vii. Amyloid angiopathy
viii. Dissecting aortic aneurysm

3) CLINICAL CLASSIFICATION:

I. Arterial territories (Oxfordshire stroke subtype classification)

i. Anterior circulation syndrome

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

ii. Posterior circulation syndrome

- Vertebrobasilar artery syndrome

- Posterior cerebral artery syndrome

II. Clinical manifestations

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

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

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

iv. Complete stroke: sudden onset persisting neurological deficit not progressing beyond 96hrs. [7]

TABLE 2: DIFFERENCES BETWEEN VARIOUS TYPES OF STROKE

Type	**Thrombosis**	**Embolism**	**Hemorrhage**
Incidence		Most Common cause of ischemic stroke	Least common than thrombotic and embolic
Age	Usually Old age	Usually Young	Usually young
Causes	Thrombus superimposed on atherosclerotic plaques.	Cardio-embolic Cardiac mural thrombi, Myocardial dysfunction, valvular disease, atrial fibrillation, atheromatous plaques in	Spontaneous, Uncontrolled and chroinic htn, Ruptured aneurysms, vascular malformations

		the aorta, tumor cells in atrial myxoma	
Common sites	1. ICA at the origin of CCA 2.Cervical part of vertebral arteries and at their junction to form basilar artery 3.Stem/bifurcation of MCA. 4.In the proximal PCA and ACA.	The territory of MCA, a direct extension of the internal carotid artery Particularly the superior division of MCA.	Intraparenchymal hemorrhage d/t HTN-basal ganglia, thalamus, pons, cerebellum SAH-saccular/berry aneursyms.
Clinical presentation	Varied Stuttering/Intermittent progression. Step like fashion. stroke is preceded by minor signs or TIA Watershed infarcts.	Rapid progression “Like a bolt out of the blue” Lack of prodromal symptoms. No Collateral circulation is formed.	Apoplexy a/w vomiting, headaches, seizures, decreased level of consciousness.
Time of onset	Subacute, began during sleep or within1 hour of awakening suggested	Hyperacute rapid onset of symptoms and a lack of warning symptoms	Hyperacute Occurs at the height of emotion/excitement.

	thrombosis with atherosclerosis		
Weakness	Slow stroke Intermittent progression of neurological deficits over several hours or a day. Episodic deficits.	Full at onset, Rapid progression. Full blown picture evolves within seconds.	Progressing over hours with increasing size of the hematoma.
Headache	+/-	-	+++
Seizures	+/-	+	++
Progression	Can progress over 72hours	High at onset	Upto 6 hours

PATHOGENESIS OF STROKE

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

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

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

Haemorrhage may be epidural, subdural, subarachnoid, intra parenchymal or intraventricular in location. Haemorrhage could result from arterial hypertension, saccular aneurysm, arteriovenous malformations, blood dyscrasias, vasculitis, sympathomimetic drugs, cerebral amyloid angiopathy, trauma and neoplasms. [20,22]

Pathophysiology of cerebral infarction

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

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

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

Cerebral blood flow at rest in adult is approximately 50-55ml/100gm/min. The cerebral

microcirculation distributes blood to brain. Any decrease in blood supply to the microcirculation leads to cerebral ischemia. The magnitude of flow reduction is a function of collateral blood flow which depends on individual vascular anatomy as well as the site of occlusion.

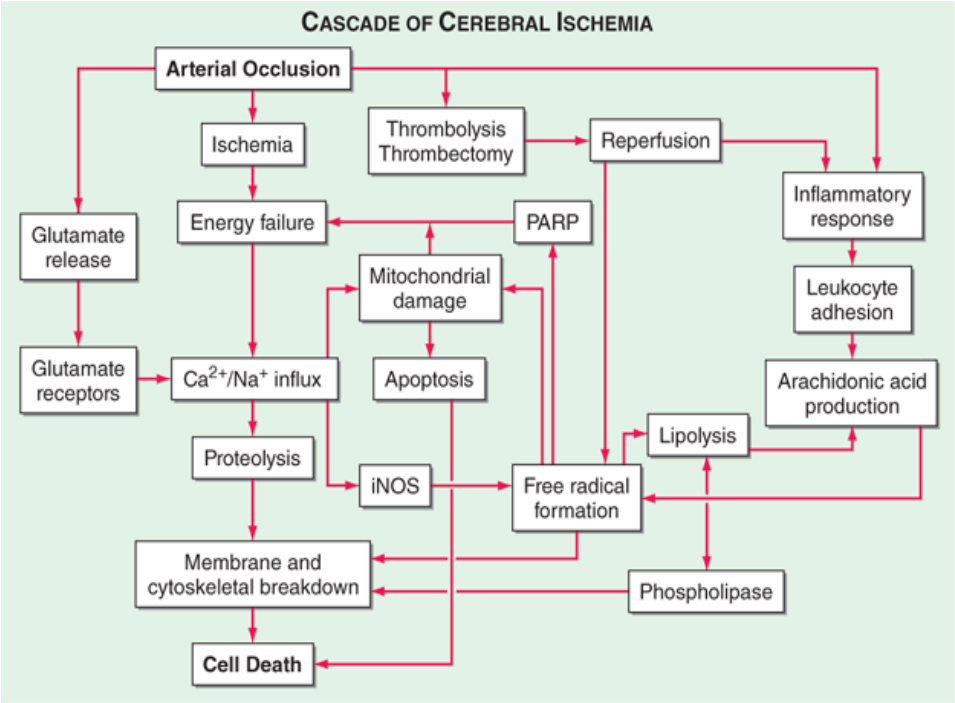
Complete interruption of cerebral blood flow suppresses electrical activity within 12-15 seconds, inhibits synaptic excitability of cortical neurons after 2-4 minutes and inhibits electrical excitability after 4-6 minutes. As the blood flow reduces to 18ml/100gm per minute, the brain reaches a threshold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

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

Cellular death via two distinct mechanisms:

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

FIGURE 4: CASCADE OF CEREBRAL ISCHEMIA



CARDIOEMBOLIC STROKE

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

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

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

within vascular territory.

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

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

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

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

Internal carotid dissection or vertebral arteries dissection or even dissection of vessels beyond the circle of willis is a common source of embolic stroke in young (age 45years) patients. Usually it is painful and precedes the stroke by several hours or days.[22,23]

CLINICAL FEATURES OF ISCHEMIC STROKES

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

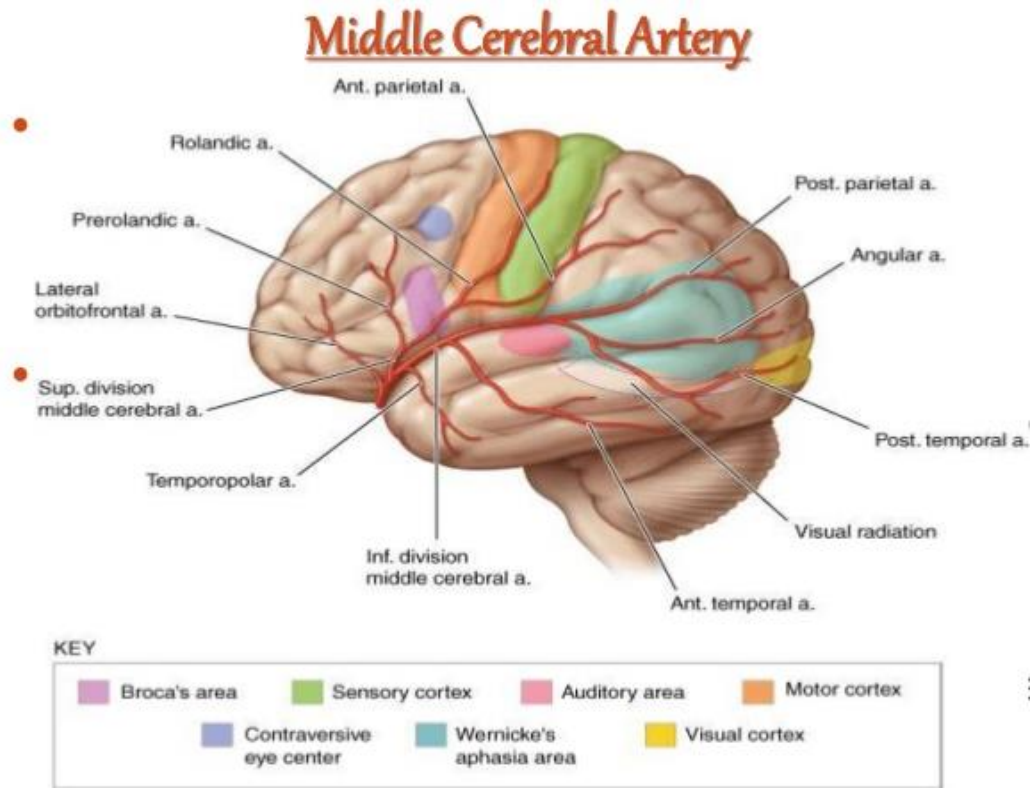
A. Middle Cerebral Artery territory involvement:

The MCA territory is the arterial territory most frequently affected by ischaemic stroke. MCA infarcts can be divided into superficial (involving the cortex and underlying white matter), deep (involving the basal ganglia, the internal capsule and the deep white matter),and combined MCA

infarcts are mainly caused by cardio embolism, internal carotid artery (ICA) thrombosis, dissection, or embolism.

Clinical presentation differs depending on whether the left (dominant for language) or the right (Non-dominant) hemisphere is involved. In left hemispheric stroke oral and written language disturbances dominate, in right-sided strokes neglect is almost always present.

FIGURE 5: DIAGRAM SHOWING LATERAL ASPECT OF CEREBRAL HEMISPHERE SHOWING DISTRIBUTION AND BRANCHES OF MIDDLE CEREBRAL ARTERY



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

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

Signs and symptoms of MCA territory involvement:

FUNCTIONAL DEFICITS:

Large middle cerebral artery infarcts: Large infarcts cover at least two sub territories of the MCA (deep, superficial, anterior or superior, and posterior or inferior)

They are usually caused by cardio embolism, ICA occlusion or dissection

They have an unfavourable prognosis and produce a severe neurological deficit :

- gaze deviation
- contralateral hemiplegia,
- global aphasia
- neglect with anosognosia
- hemianopia
- Reduced consciousness.

Middle cerebral artery anterior or superior division infarcts:

- contralateral hemiparesis with predominant faciobrachial deficit,
- hemisensory loss,
- gaze deviation towards the lesion

- Decreased visual exploration toward the opposite side.
- Left -sided infarcts also produce a non-fluent aphasia, ranging from mutism to typical Broca's aphasia and to articulatory syntactical and naming difficulties.
- Bucco-facial apraxia

Middle cerebral artery posterior or inferior division infarcts

- Hemisensory loss is common and visual field defects (homonymous hemianopia or upper quadrantanopia) are usually present.
- Motor deficits are absent or mild.
- In left -sided strokes, fluent aphasia predominates.
- The most severe form is Wernicke's aphasia with anosognosia and behavioural disturbances including persecutory delusions. In right hemispheric stroke, neglect, anosognosia, and
- An agitated confusional state usually occurs.
- Constructional apraxia can be present.

Anterior choroidal artery infarcts

- Pure motor deficit followed by motor and sensory dysfunction.
- The classical '3H' syndrome composed by hemiparesis, hemihypesthesia, hemianopia
- Isolated hemianopia
- pseudobulbar palsy

NEUROLOGICAL DEFICITS:

1. Somatic motor area for face and arm and the fibres for leg descend and enter the corona radiata and corresponding somatic sensory system.

- Opposite side face, arm and leg palsy.
- Sensory defect in the same area (fine touch , pinprick, vibration, joint position, and all cortical sensations.)

2. Dominant hemisphere motor speech area:
   - Motor aphasia
3. Cental, suprasylvian speech area and parietooccipital cortex of the dominant hemisphere -Gerstmann syndrome comprising right left confusion, finger agnosia, alexia, acalculia , anomia, jargon speech, sensory agraphia, Cental aphasia, word deafness.
4. Central speech area (parietal operculum): conduction aphasia
5. Non dominant parietal lobe:
   - Apractognosia of the non-dominant hemisphere, anosognosia, hemiasomatognosia, unilateral neglect, agnosia for the left half of external space, dressing “apraxia”, constructional apraxia, distortion of visual coordinates, impaired reading, visual illusions.
6. Optic radiation deep to second temporal convolution: Homonymous hemianopia.
7. Frontal contraversive eye field or projecting fibres: conjugate gaze paralysis to the opposite side.

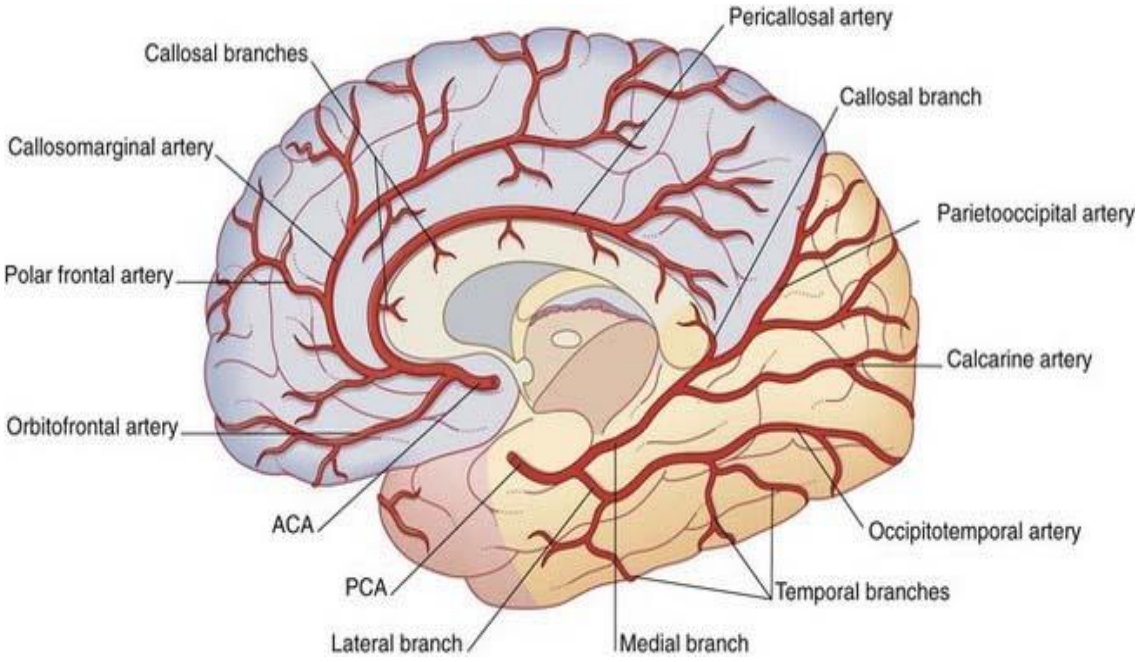
B. <u>Anterior Cerebral Artery (ACA) territory involvement:</u>

Strokes in the anterior cerebral artery (ACA) territory are uncommon(<2% in stroke registries).

The ACA it categorised into two parts:

- Pre-communal (A1) segment; connecting the internal carotid artery to the anterior communicating artery.
- Post-communal (A2) segment; distal to the anterior communicating artery.

FIGURE 6: DIAGRAM SHOWING MEDIAL ASPECT OF CEREBRAL HEMISPHERE SHOWING DISTRIBUTION AND BRANCHES OF ANTERIOR CEREBRAL ARTERY



Signs and symptoms of ACA territory involvement

FUNCTIONAL DEFICITS:

- Left -sided infarcts cause mutism,

- Transcortical motor aphasia,
- Hemiparesis
- Occasionally left –arm apraxia.
- Right-sided infarcts cause acute confusional state, hemiparesis, and motor neglect
- Hemiparesis predominates in the lower limb when the precentral gyrus is involved
- Proportional hemiparesis occurs when there is occlusion of the recurrent artery of Heubner that supplies the internal capsule.
- Bilateral infarcts may produce akinetic mutism
- Gait apraxia
- Paraparesis
- Sphincter dysfunction.
- Basal ganglia symptoms, including parkinsonian gait, tremor, and facial dystonia, can be observed in bilateral ACA infarcts

NEUROLOGICAL DEFICITS:

1. Motor area for leg: Paralysis of contralateral side foot and leg.
2. Area of arm in the cortex or descending fibres to corona radiata: paresis will be of lesser severity of opposite side.
3. Sensory area for lower limb: Cortical sensory loss over toes, foot and leg.
4. Paracentral lobule sensorimotor area: urinary incontinence
5. Posterior frontal lobe medial surface : contralateral grasp reflex, sucking reflex
6. Leg motor area: Impairment of gait and stance

7. Corpus callosum: Dyspraxia of left limbs, tactile aphasia in left limbs anterior choroidal artery territory involvement- contralateral hemiplegia, homonymous hemianopia and hemianesthesia.

Internal Carotid Artery Territory Involvement:

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

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

Stroke within The Posterior Circulation

PCA infarcts are due to cardioembolism in one-third of cases. Significant vertebrobasilar atheroma with occlusion or artery-to-artery embolism accounts for another one-quarter.

Local PCA stenosis or occlusions are much less frequent.

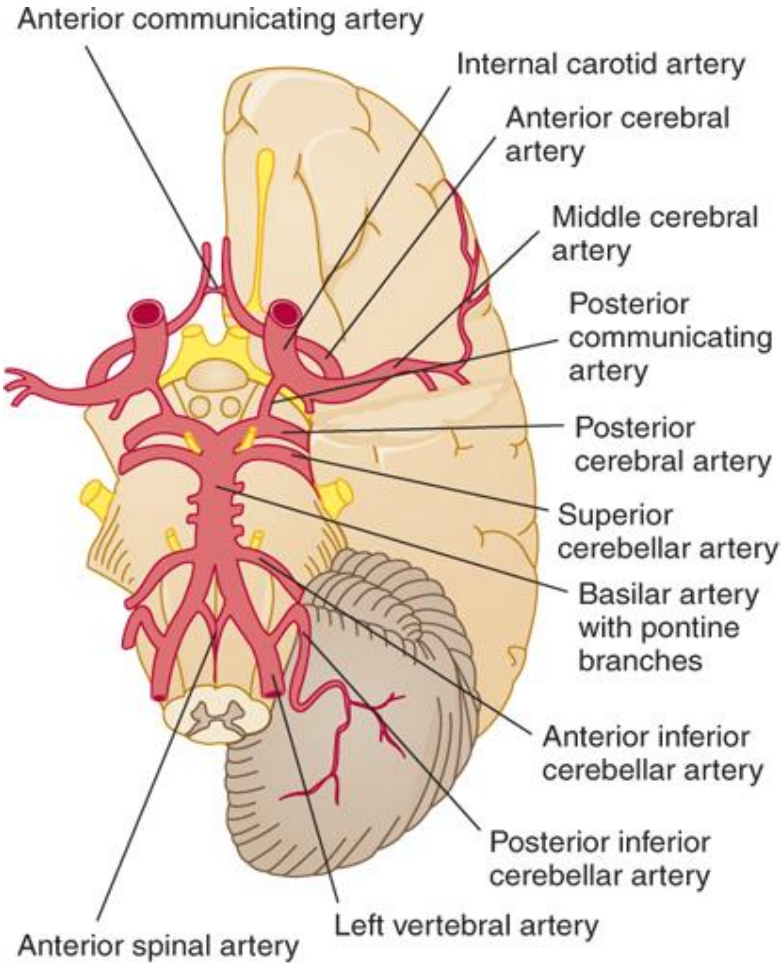
Infarcts associated with migraine account for a few cases.

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

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

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

FIGURE 7: DIAGRAM SHOWING INFERIOR ASPECT OF THE BRAIN WITH THE DISTRIBUTION AND BRANCHES OF POSTERIOR CEREBRAL ARTERY



Signs and symptoms of PCA territory involvement

FUNCTIONAL DEFICITS:

- Visual field defects
- Hemianopia
- Severe unilateral headache
- ataxia or involuntary movements.

- Transient paresis.
- Hemiparesis in PCA infarcts can be due to infarction of the cerebral peduncle or less frequently to infarction of the anterior segment of the posterior limb of the internal capsule
- Superior quadrantanopia is more common than inferior, because the inferior striate cortex is more susceptible to ischaemia due to poor collateral circulation.
- Neuropsychological manifestations: transcortical sensory aphasia or anomic aphasia, alexia with or without agraphia, visual or colour agnosia

NEUROLOGICAL DEFICITS:

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

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

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

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

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

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

(7) Non dominant hemisphere: Complex hallucinations

Central Territory:

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

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

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

4. Cerebral peduncle: Contralateral hemiplegia

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

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

Vertebral and Posterior Inferior cerebellar arteries

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

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

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

4. Fourth part of vertebral artery (V4) travels upwards and unites to further vertebral artery. This

part gives branches that supply brainstem and cerebellum.

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

The first vertebral division gets affected by atherothrombotic lesions at the site of its vessel origin and leads to posterior circulation emboli, usually lesions affect V1 to V4 parts. Stenosis near to the PICA origin can damage the lateral medulla and posterior inferior part of the brain. There is retrospective blood flow in ipsilateral vertebral artery if obstruction in subclavian artery is near the point of origin of vertebral artery. Ipsilateral arm exercise may increase demand on vertebral flow, producing TIAs, or subclavian steal of posterior circulation.

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

Medial medullary syndrome rarely occurs, which causes infarction of the pyramid leading to contralateral hemiparesis sparing the face. Joint position sense of contralateral side and ipsilateral tongue weakness occurs when the medial lemniscus and hypoglossal nerve emerging fibres are involved.

Small vessel Stroke/Lacunar stroke:

Small penetrating branches of the cerebral arteries may become occluded, and the resulting

infarcts may be so small or so situated as to cause no symptoms.

As the softened tissue is removed by macrophages, a small cavity, or lacune, remains. The lesions were first described by Durant-Fardel in 1843.

Lacunes are usually caused by occlusion of small arteries, 50 to 200 microns in diameter, and the cribriform state, to mere thickening of vessels and fraying of the surrounding tissue. The term lacunar infarction refers to infarction due to occlusion of a small artery (30-300μm) in the brain, now termed as small vessel stroke.

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

Three mechanisms for lacunar infarction:

1) Local type of fibrohyalinoid arteriolar sclerosis that involves the orifice or proximal part of a small penetrating blood vessel (lipohyalinosis).

2) Atherosclerosis of a large trunk vessel that occludes the origin of these same small vessels. This is prone to involve several adjacent vessels and cause, at times, larger lacunes or the atherosclerosis extends from a trunk vessel into a smaller one.

3) Entry of small embolic material into one of the vessels.

Lacunes are situated, in descending order of frequency,in the putamen and caudate nuclei, thalamus, basis pontis, internal capsule, and deep in the central hemispheral white matter. The cavities range from 3 to 15 mm in diameter.

The most common lacunar syndromes:

(1) Pure motor hemiparesis.

(2) Pure sensory stroke.

(3) Ataxic hemiparesis.

(4) Dysarthria and a clumsy hand or arm.

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

Small-vessel infarct may manifest as TIA.

INITIAL ASSESMENT OF A STROKE PATIENT:

(A) Immediate evaluation

1. Stabilisation of airway, breathing and circulation.

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

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

4. Other conditions requiring immediate intervention to be excluded.

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

(B) History taking

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

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

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

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

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

6. Determine eligibility for therapeutic intervention like revascularisation.

(C) Systemic examination

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

body temperature.

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

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

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

(D) <u>Investigations</u>

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

-Complete blood count

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

-Plasma glucose level.(will be usually on the higher side due to stress induced hyperglycemia)

-Blood urea, nitrogen and serum creatinine.

-Lipid profile.

-Urine analysis.

-Chest X-Ray

-Electrocardiogram.

-Carotid Doppler

2. Non enhanced cranial CT (computed tomography)

-Primary modality is the brain imaging study for evaluation of stroke.[24,25]

(a) Hyperacute infarcts (<12 hours)

Early CT signs of ischemic stroke in MCA territory: Sulcal effacement, effacement of the sylvian fissure, obscuration of the lentiform nucleus, loss of grey-white matter differentiation and

Horizontal part of MCA may be hyperdense (dense MCA sign) before infarction becomes visible.

(b) Subacute infarcts

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

(c) Chronic infarcts

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

"Scan negative infarcts usually occur with lower brainstem infarcts, lacunar infarcts, early scans(60% cases within 12 hours of ictus may not pick up the lesion), scans done after 2-3 week may not show or may underestimate the size of the infarct." [(26)]

(d) Acute haemorrhage

They are hyperdense on non-contrast CT brain.

The location of the haematoma can often indicate the likely aetiology. In a hypertensive patient, a deep haematoma in the basal ganglia or external capsule is most likely due to chronic hypertension. Superficial cortical 'lobar' haematomas in elderly patients are most likely due to amyloid angiopathy. Any haematoma in the region of the Sylvian fissure or anterior, inferior frontal lobe should prompt consideration of a ruptured aneurysm or vascular malformation. The volume of the haematoma is directly related to prognosis. Haematoma volume greater than 60 mL generally indicates poor prognosis. Brain CT detects symptomatic ICH within minutes of symptom onset but may lack sensitivity if delayed for >1 week after ICH onset as haemorrhage is isodense to brain tissue.

FIGURE 8: CT SHOWING HYPER DENSE MCA SIGN- HIGH ATTENUATION WITHIN THE EXPECTED LOCATION OF THE RIGHT MCA (ARROW) CONSISTENT WITH AN ACUTE THROMBUS

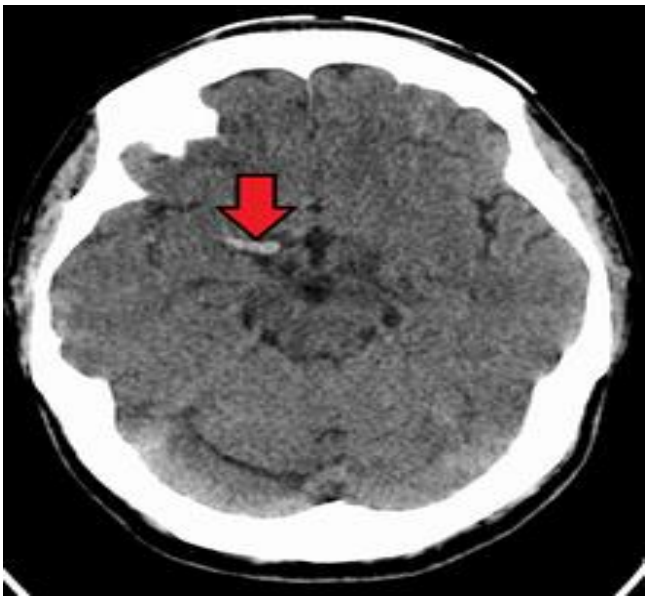


FIGURE 9: CT SHOWING LEFT BASAL GANGLIA INFRACTION

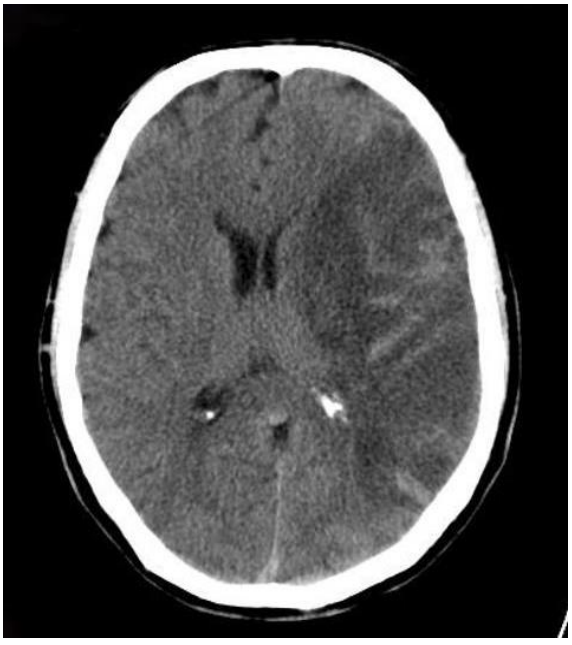


FIGURE 10: CT SHOWING LEFT MCA TERRITORY INFARCTION

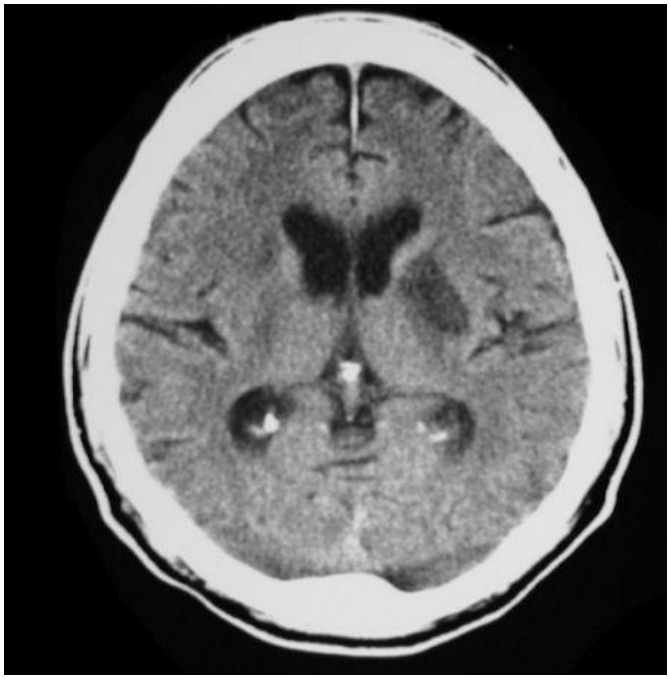
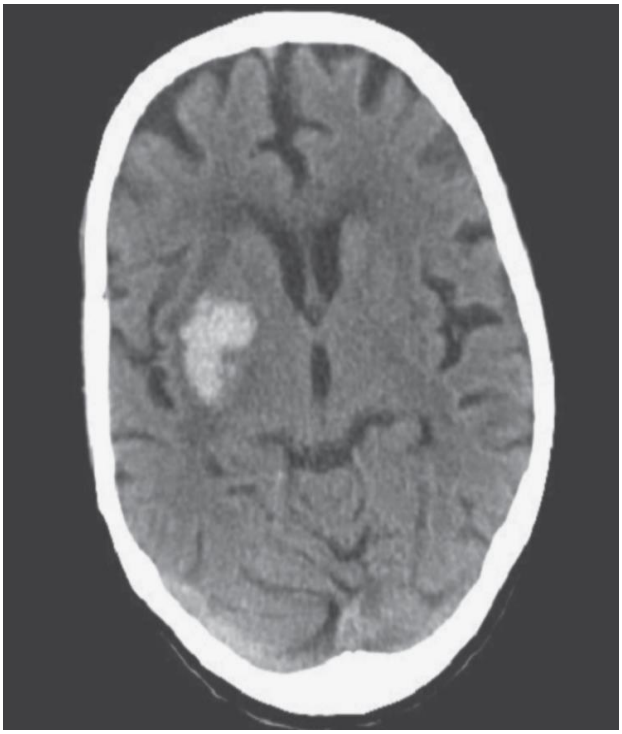


FIGURE 11: CT SHOWING INTRACEREBRAL HEMORRHAGE IN THE RIGHT BASAL GANGLIA



3. Multimodal CT

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

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

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

4. Magnetic Resonance Imaging (MRI)

-Superior to CT for detecting cerebral ischemia.

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

5. Multimodal MRI

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

ii. Perfusion weighed MRI (PWI) – gives relative measurement of brain hemodynamics.[27]

6. MRI angiography

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

7. Other brain imaging methods

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

ii. Single photon-emission computed tomography (SPECT) identifies "thresholds for reversible ischemia and could be helpful in predicting outcomes or monitoring responses to treatment." [28]

iii. Duplex Doppler Ultrasonography

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

iv. Transcranial Doppler sonography evaluates blood flow velocity and patency of the main intracranial arteries and in identification of high intensity transient micro embolic signals. [29]

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

Treatment of Acute Ischemic Stroke:

(A) General supportive care

a. OXYGENATION: The brain consumes 20% of the oxygen in the circulation. In the absence of oxygen, neuronal death occurs in 1–3 minutes, hence maintaining adequate tissue oxygenation is important to prevent hypoxia and potential worsening of the neurological injury.

Hypoxia is more likely to develop in patients with large stroke, advanced age, dysphagia, and pre-existing cardiac and pulmonary disease. There are several central (neurogenic) and peripheral

(non-neurogenic) causes of hypoxia in stroke. Neurogenic causes include alveolar hypoventilation due to extensive brainstem or hemispheric stroke, central obstructive sleep apnoea, and paralysis of the respiratory muscles.

Peripheral causes of hypoxia include airway obstruction, aspiration pneumonia.

Monitoring with pulse oximetry should be done to the Patients with acute stroke.

- The goal is to maintain oxygen saturation ≥ 92%
- 100% oxygen through a nasal cannula at a rate of 1–3 L/min if oxygen saturation <92%
- Identify and treat the cause of hypoxia

b. AIRWAY:

- Secure airway
- Oropharyngeal suctioning and semi-upright position in patients at risk of aspiration
- Elective intubation in patients with Glasgow Coma Scale score <9 and signs of increased intracranial pressure

C. BREATHING:

- Maintain PaO2 >80 mmHg, PaCO2 <45 mmHg
- Monitor breathing patterns particularly during sleep
- Ventilatory support in patients with respiratory distress or inability to protect airway due to impaired consciousness or oropharyngeal weakness.

D.CIRCULATION:

- Maintain systolic blood pressure (BP) >140 mmHg and central venous pressure >5 mmHg in patients with ischaemic stroke or raised ICP.
- 0.9% NaCI infusion at 1–3 mL/kg/h or vasopressor agents when systolic BP <140 mmHg or central venous pressure <5 mmHg.
- Avoid Hypotension in patients at risk of infarct growth or in those with raised ICP.
- In most circumstances, the blood pressure should generally not be lowered, except in acute renal failure, acute pulmonary oedema, hypertensive encephalopathy, acute myocardial infarction or aortic dissection. Antihypertensive agents should be withheld

unless the unless the systolic blood pressure is >220 mmHg or diastolic blood pressure is >120 mmHg.[31]

E.HEAD POSITION:

Keep head elevated at 30° in straight position (not turned to sides) in order to maximize venous outflow and minimize the risk of aspiration.

F. MAINTENANCE OF OPTIMAL BODY TEMPERATURE:

Hyperthermia (>37.5°C) is not desirable in acute stroke because it increases cerebral metabolism, depletes energy reserves, and accelerates neuronal death.

Each 1°C elevation in body temperature within 6 hours of symptom onset increases the relative risk of death or disability by 2.2 per cent.

Increased body temperature in acute stroke is associated with poor neurological outcome, due to increased neurotransmitters release, increased metabolic demands and increased free radical production. [30]

Hypothermia, on the other hand, is neuroprotective after brain injury. For each 1°C decrease in temperature, the cerebral metabolic rate decreases by 6–7%

Since therapeutic hypothermia is associated with serious side effects such as infection, bleeding diathesis, hypotension, and cardiac arrhythmias it is suggested that maintenance of normothermia (<37°C core body temperature) is a reasonable goal in acute stroke.

G.MAINTENANCE OF GLYCAEMIA CONTROL:

Glucose is the main substrate for energy production in the brain.

Glycogen stores within the brain are very limited (barely sufficient to maintain neural function

for 1–3 minutes in an event of ischaemia.

When oxygen supply is insufficient, lactate accumulates in the brain; the resulting intracellular acidosis reduces tissue pH to approximately 6.5. Tissue acidosis in the presence of ischaemia accelerates cell death.

Also, stress induced hyperglycaemia accelerates the conversion of ischaemic tissue into infarction, resulting in rapid formation of larger infarcts than those seen in normoglycaemia, Hence elevated blood glucose is also associated with increased 30-day mortality and poor functional outcome after stroke.

H. Anticonvulsant therapy in patients with recurrent clinical or electrographic seizures.

I. Prevention and treatment of Deep Vein Thrombosis.

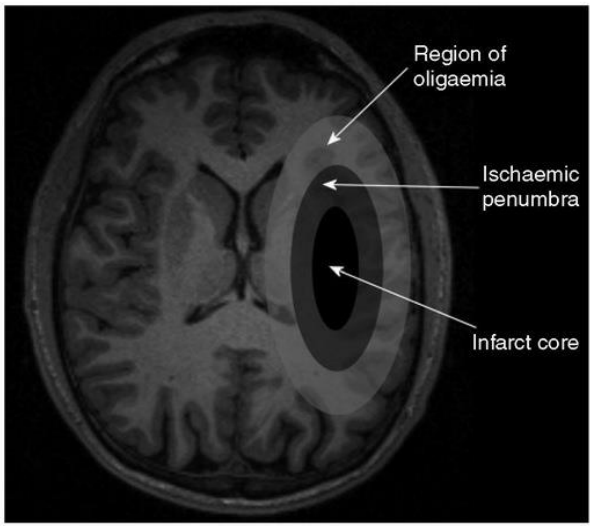
(B) Measures to Restore or Improve Perfusion

(i) Thrombolytic therapy:

The goal of the thrombolytic treatment is to remove the occlusion and to restore blood flow to the hypoperfused brain tissue. Typically, the hypoperfused area consists of irreversibly damaged brain tissue (infarct core) that is surrounded by viable and potentially salvageable brain parenchyma (ischaemic penumbra). The ischaemic penumbra can be salvaged by a timely vessel recanalization.

"The NINDS study used IV rtPA (0.9 mg/kg to a 90-mg max; 10% as a bolus, then the remainder over 60 min) within 3 hour of onset resulted in good functional outcome though incidence of hemorrhage increased after thrombolysis. In ECASS-II, intravenous rtPA was not more effective than placebo in improving neurological outcomes at 3 months after stroke." [(32)]

FIGURE 12: REGION OF PENUMBRA



In the multicenter acute stroke trial Europe study group (MAST-E), streptokinase in the dose of 1.5 million units over one hour was associated with hemorrhagic transformation of ischemic infarct and hence not recommended.[33]

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

INDICATIONS AND CONTRAINDICATIONS FOR IV RTPA IN ACUTE ISCHAEMIC STROKE:

Indications:

- Age >18 years
- Time window <4.5 hours
- National Institutes of Health Stroke Study (NIHSS) score: 4–25 points
- Symptoms persisting for ≥30 minutes without significant improvement

Contraindications

- Evidence of intracranial haemorrhage on pretreatment CT or MRI
- Recent (within 3 months) intracranial or intraspinal surgery, serious head trauma, or previous stroke
- History of intracranial haemorrhage
- Uncontrolled hypertension at time of treatment (>185 mmHg systolic or >110 mmHg DBP)
- Seizure at the onset of stroke
- Active internal bleeding
- Intracranial neoplasm, arteriovenous malformation, or aneurysm
- Known bleeding diathesis

(ii) Anticoagulants

According to the Joint Guideline Statement from the AHA and AAN, urgent routine anticoagulation with the goal of improving neurological outcomes or preventing early recurrent stroke is not recommended as it is associated with increased risk of bleeding complications. [34]

(iii) Antiplatelet Aggregation Agents

a) Aspirin

Aspirin has proved t o b e the most consistently useful drug in the prevention of thrombotic and possibly, embolic strokes.

The acetyl moiety of aspirin combines with the platelet membrane and inhibits platelet cyclooxygenase, thus preventing the production of thromboxane A2, a vasoconstricting

prostaglandin, and also prostacyclin, a vasodilating prostaglandin.

The International Stroke Trial (IST) demonstrated a significant lowering in ischemic events recurrence by using aspirin within first 2 weeks, but acute mortality was not reduced. At 6 months, patients' assigned aspirin had a significantly lower incidence of dependency and death. (35)

The Chinese Acute Stroke Trial (CAST) showed that mortality was significantly reduced with aspirin, but the rates of long term complete recovery or death and disability were not significantly improved.(36)

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

b) Ticlopidine

In the ticlopidine aspirin stroke study, the risk of non-fatal stroke or death from any cause at 3 years was lower in ticlopidine group as compared to aspirin group (17% vs 19%). The 3 year risk of fatal or non-fatal risk was also lower (10% vs 13%). Thus it was concluded that ticlopidine was more effective than aspirin. The Canadian American ticlopidine study concluded that an exclusive benefit cannot be claimed for ticlopidine over aspirin in treating patients with stroke.(37)

c) Clopidogrel

It is a theinopyridine derivative which is a potent inhibitor of platelet aggregation caused by ADP. In a trial recent stroke/TIA patients were randomized to receive clopidogrel 75 mg/day with low dose aspirin 75 mg/day, showed no statistically significant difference in outcome

between the two treatment groups. Clopidogrel can be given to patients allergic to aspirin. [38]

The combined use of these antiplatelet drugs with aspirin has generally been slightly superior to aspirin alone in secondary stroke prevention, but with an increased risk of cerebral hemorrhage in some studies.

(C) Neuroprotective agents

Hypothermia is probably the most powerful neuroprotectant. Busto et al. (1987) first discovered that moderate decrease of brain temperature provides neuroprotection against experimental brain ischemia. Hypothermia inhibits glutamate-induced excitotoxicity while reducing the production of superoxide, peroxynitrite, hydrogen peroxide, and hydroxyl radicals to relieve oxidative stress after cerebral ischemia. Hypothermia has also been reported to reduce apoptosis, autophagy, and inflammation, as well as blood-brain barrier leakage and brain metabolism after cerebral ischemia, which suggests that the use of hypothermia during/after cerebral ischemia at a temperature between 33°C and 30°C provides high therapeutic potential in the treatment of patients with stroke.

Six variant drugs have been tried like Calcium channel antagonists (nicardipine, nimodipine), NMDA receptor agonist (selfolate, eliprodil), ICAM-1 antibodies (Enlimomab), GABAergic antagonists (diazepam, Clomethiozole), glutamate antagonists (leleluzole), free radical scavengers (tirilazed, dihydrolipoate), lipid peroxidation inhibitors, required before routine use. [39]

(3) Surgical Interventions

The MERCI (Mechanical Embolus Removal in Cerebral Ischemia) single-arm trial investigated endovascular thrombectomy to restore patency of occluded intracranial vessels within 8 hours of ischemic stroke symptoms. Recanalization of the target vessel occurred in 48% of treated patients and in 60% following use of adjuvant endovascular methods, and successful recanalization at 90 days correlated well with favourable outcome. The North American Symptomatic Carotid Surgery Trial (ECST) evaluated patients with symptomatic stenosis of carotid arteries found a substantial benefits in patients with a stenosis of >70%.[37]

(4) Treatment of Acute Neurological Complications

(a) Brain oedema and increased intracranial pressure-

Brain oedema is the major cause of stroke-related mortality during the acute stroke phase. Within minutes of cerebral ischaemia, cytotoxic oedema develops due to the mobilization of water molecules from the interstitial space to the intracellular space.

As ischaemia persists, the blood–brain barrier breaks down and plasma proteins start to leak into the brain parenchyma causing vasogenic oedema causing significant expansion of the intracranial volume and traction or compression of the intracranial structures.

The goals of management to reduce brain oedema are:

- To maintain ICP <20 mmHg and cerebral perfusion pressure >60 mmHg
- Head elevation to 30° to increase venous outfl ow, intubation, sedation, hyperventilation to maintain PaCO2 25–30 mmHg, and control of hyperthermia, hyperglycaemia, and seizures

- Consult with neurosurgery to assess for hemicraniectomy, decompression, external ventricular drain placement, or haematoma evacuation
- ICP monitoring for patients with Glasgow Coma Scale score <9, intraventricular haemorrhage and hydrocephalus, and signs of herniation
- 20% mannitol 0.25–1 g/kg IV bolus, every 4–6 hours as needed
- 23.4% NaCl 0.5–1 mL/kg IV bolus, every 4–6 hours as needed when there is high osmolar gap, renal insufficiency, high ICP despite mannitol therapy, diabetes, and hypotension or haemodynamic failure
- Metabolic suppression therapy with barbiturates or hypothermia to reduce ICP refractory to osmotherapy
- Suboccipital decompressive craniectomy or cerebellar resection in space occupying cerebellar oedema

Osmotherapy and hyperventilation are helpful for patients who have raised intracranial pressure features. Corticosteroids are not recommended Surgical decompression and evacuation is recommended in large cerebellar infarctions that are leading to hydrocephalus and brain stem compression . It is lifesaving procedure, but survivors have neurological impairments. [40]

(b) Seizures

seizures are quite infrequent as the initial manifestation of an ischemic stroke, and when they do occur in this fashion, an embolus is usually the causative mechanism.

The rate of seizures is higher after hemorrhagic than ischemic strokes and for the latter category, larger cortical strokes were more likely to result in a seizure disorder.

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

It is adviced to start one of the main epilepsy medications only if there has been a seizure, and continue it for about 12 months.

(c) Haemorrhagic Transformation

Antithrombotics agents, especially anticoagulants and thrombolytic agents, increases the likelihood of serious haemorrhagic transformation. The early use of aspirin also is associated with a small increase in the risk of clinically detectable haemorrhage. [35]

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

Motor recovery from stroke

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

The prognosis of arm motor recovery in hemiplegic stroke patients is poor with good functional outcome in only 5–20%.

If some arm grip is present within the fi rst weeks, outcome is not as bad, whereas the absence of a hand grip within a month is indicative of very poor motor outcome.

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

absence of return of motor function in the leg within 2 weeks is associated with poor functional arm outcome.

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

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

Neurophysiologic mechanism for recovery from stroke

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

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

Certain clinical observations shows the bilateral representaion of the brain.

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

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

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

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

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

SIRIRAJ STROKE SCORE

Siriraj stroke score was developed in Bangkok, Thailand and subsequently validated in the same population. Siriraj Stroke Score is based on 5 variables that can be assessed bedside. These variables are the level of consciousness of the patient at presentation, history of vomiting after the onset of symptoms, history of headache within 2 months of onset of symptoms, diastolic blood pressure measured at the time of admission and atheroma markers in the patient's like history of angina, diabetes and intermittent claudication.

These above variables are given score based upon the clinical features in the patient at presentation.

In considering the variable, level of consciousness scores of 0, 1, and 2 are given based on presentation and these scores are multiplied by a constant.

All the variables are given scores and a constant of 12 is subtracted to give the final score.

If the score is <1, it is diagnosed to be infarct by Siriraj Stroke Score. If the score is between -1 and +1, equivocal and if the score is >1, it is considered to be hemorrhage by the Siriraj Stroke Score.

TABLE 3: SIRIRAJ STROKE SCORE BASED ON FIVE VARIABLES WHICH CAN BE ASSESSED BEDSIDE.

SL NO.	VARIABLE	CLINICAL FEATURES	SCORE
1	Level of consciousness	Alert	0
		Drowsy/stuporous	1
		Semi-comatose/ comatose	2
2	History of vomiting after onset	Absent	0
		Present	1
3	History of headache within 2 hours of onset	Present	0
		Absent	1
4	Diastolic Blood Pressure	In mm of hg	+
5	Atheroma markers(angina, claudication, diabetes)	None	0
		One or more	1
	Constant		12

Calculation:

Siriraj Stroke Score (SSS) = (2.5* level of consciousness) + (2 * vomiting) + (2 *headache) + (0.1 *DBP) – (3* atheroma markers) -12

If symptoms are not clear, it is recorded as zero/absent.

- **Score of >+1** **Haemorrhage**
- **Score of <-1** **Infarction**
- **Score of +1 to -1** **Equivocal**

GUYS HOSPITAL/ ALLENS SCORE

Guy's hospital score was conducted by Allen et al in the Oxfordshire Community Stroke Project.

Allen's score is also based on clinical variables. The onset of symptoms and other associated symptoms like headache, vomiting and neck stiffness, the level of consciousness 24 hours after admission, the diastolic blood pressure, history of hypertension, history of atheroma markers like angina, diabetes and intermittent claudication, history of previous events like transient ischemic attack or stroke and history of heart disease.

On examination, aortic or mitral murmur, history of cardiac failure, cardiomyopathy, atrial fibrillation, cardiomegaly or myocardial infarction within 6 months.

Each clinical variable is given a score based on presentation and history and the total score is subtracted by a constant of 12.6 and the final score is obtained.

If the score is <14, it is diagnosed to be cerebral infarction by Allen's score

If the score is $\geq$ 14, it is considered to be cerebral hemorrhage by the score.

TABLE 4: GUY'S HOSPITAL/ ALLEN'S SCORE

VARIABLE	CLINICAL FEATURES	SCORE
Apoplectic onset Loss of consciousness Headache within 2 hours, vomiting, neck stiffness	None or one	0
	Two or more	21
Level of consciousness(24 hours after admission)	Alert	0
	Drowsy	7.3
	Unconscious	14.6
Plantar responses	Both flexor or single extensor	0
	Both extensor	7.1
Diastolic blood pressure(24 hours after admission) *(0.17)		
Atheroma Markers(Diabetes, angina, intermittent claudication)	None	0
	One or more	-3.7
History of hypertension	Not Present	0
	Present	-4.1
Previous event(transient ischemic attack)	None	0
	Any number of previous events	-6.7

Heart disease	None	0
	Aortic or mitral murmur	-4.3
	Cardiac failure	-4.3
	Cardiomyopathy	-4.3
	Atrial fibrillation	-4.3
	Cardiomegaly	-4.3
	Myocardial infarction within 6 months	-4.3
Constant		-12.6

Interpretation: ≥ 14 = Cerebral hemorrhage **< 14 =Cerebral Infarction**

When computed tomography is not immediately available and the clinician wishes to start antithrombotic treatment, the Siriraj Score (and possibly the Allen Score) can be useful to identify patients at low risk of intracerebral hemorrhage. In their study Celani MG et al[41] showed that the accuracy of both scores when compared to computed tomography was 89%. The Siriraj score is simpler, can be used immediately after stroke and has a 93% positive predictive value for ischemia.

The Siriraj stroke score was developed in Bangkok. Thailand and subsequently validated in the same population. Poungvarin N et al[42] who had developed this score reported an overall accuracy of 90.3%, the sensitivity for hemorrhage being 89.3% and that for infarction being 93.2%. Validation study of the Guy's hospital score was conducted by Allen et al in the Oxfordshire community stroke project. Both the scores are based mainly on clinical symptomatology.

The Siriraj stroke score can be used as reliable bedside method for diagnosing acute stroke and for deciding which patients should have priority for computed tomography; it is also a valuable tool for epidemiology studies of stroke incidence and outcome. Hung LY et al[43] in their study showed that the diagnostic sensitivities of the Siriraj stroke score for intracranial hemorrhage and infarction were 85% and 90% respectively with an overall predictive accuracy of 88.5%

It is likely that CT scan remains essential for the early detection of hemorrhage and for the rationale use of antihemostatic drugs. However, when resources are limited and CT scan facilities are not available, Jyothi Wadhwani et al[44] have suggested Siriraj stroke score as a simple and practical method of screening patients for intracerebral hemorrhage. According to their study, they found that the sensitivity of Siriraj stroke score was 92.54% for infarction and 87% for hemorrhage and its overall accuracy was91.11%. The Allen's score had a

sensitivity of 93.42% for infarction and 66.66% for hemorrhage and overall accuracy was 87%

AIMS AND OBJECTIVE OF THE STUDY:

1. To differentiate between cerebral infarct and intracerebral hemorrhage on the basis of clinical stroke score (Siriraj stroke score and Allen's Hospital score)

2. To find out the validity of these scoring systems by comparing those with CT scan findings.

MATERIALS AND METHODS

SOURCE OF DATA

Patients admitted in the medicine ICU/WARDS OF BLDEUS Shri BM Patil medical college and research Centre, Vijayapura and who fulfill the inclusion criteria.

Patients attending the medicine OPD/ executive health check-up schemes that fulfill the inclusion criteria

Period of study:

The study will be conducted during the period of October 2018 to June 2020

METHOD OF COLLECTION OF DATA:

The data is collected according to proforma in terms of details history, clinical examination and necessary investigations of the patients who fulfill the inclusion criteria

Types of study: Prospective cross sectional study

Sample size calculation

By using formula

$$n = \frac{Z^2 * P(1-P)}{d^2}$$

n will be (a+c) if we use sensitivity as P, and n will (b+d) if we use Specificity as P in formula above.

where,

Z = z statistic at 5% level of significance

'd' is margin of error

'p' is expected prevalence rate

Sample size- with the anticipated sensitivity and specificity of Allen's score with CT scan 95% and 89 respectively, and at 95% confidence level, the sample size calculated is 55, using the above statistical formula.

Statistical analysis

Data will be presented using Diagrams, percentages Mean and Standard Deviation.

Score will be compared with CT SCAN report using sensitivity, specificity, positive predictive and negative predictive values.

Mc Nemer's chi square test will be applied to find the significant difference.

INCLUSION CRITERIA

Patients who presented with a focal or global disturbance of cerebral function lasting greater than 24hours were considered.

- Age > 18 years

- Of both sex

EXCLUSION CRITERIA

- Transient Ischemic Attacks
- Patients with stroke due to other causes like tuberculoma, tumor, trauma, epilepsy
- Patients with Sub-Arachnoid hemorrhage
- Patients whose mean time delay is more than 72 hours from the onset of symptoms to the time of admission.

Investigations or interventions required in this study are routine standard procedures. There is no animal experiment involved in this study. Investigations include:

1. Complete Blood Count
2. Fasting Blood Sugar Level
3. Lipid Profile
4. Electrocardiogram
5. Two dimensional Echocardiography
6. CT Brain Plain

The result of clinical score and CT scan were compared to determine the accuracy of these scores.

RESULTS AND OBSERVATION

- Out of 57 cases admitted into the study 42 cases were proven to be having infarction and 15 cases were proven to have haemorrhage by CT scan, which was considered gold standard.
- Out of 57 cases, 36 were males and 21 were females
- 21 out of 57 cases were found to be hypertensive.
- 5 out of 15 CT scan proven haemorrhages was found to be hypertensive.

TABLE 5: DISTRIBUTION OF PATIENTS ACCORDING TO AGE (YEARS)

Age(years)	No. of patients	Percentage
< 30.00	2	3.5
30.00 - 39.00	3	5.3
40.00 - 49.00	6	10.5
50.00 - 59.00	12	21.1
60.00 - 69.00	16	28.1
70+	18	31.6
Total	57	100.0

- In our study, incidence of stroke was maximum in the age above 60 years, with maximum incidence in the age group of 60-80 years with 28 cases out of the total cases.

FIGURE 13: DISTRIBUTION OF PATIENTS ACCORDING TO AGE (YEARS)

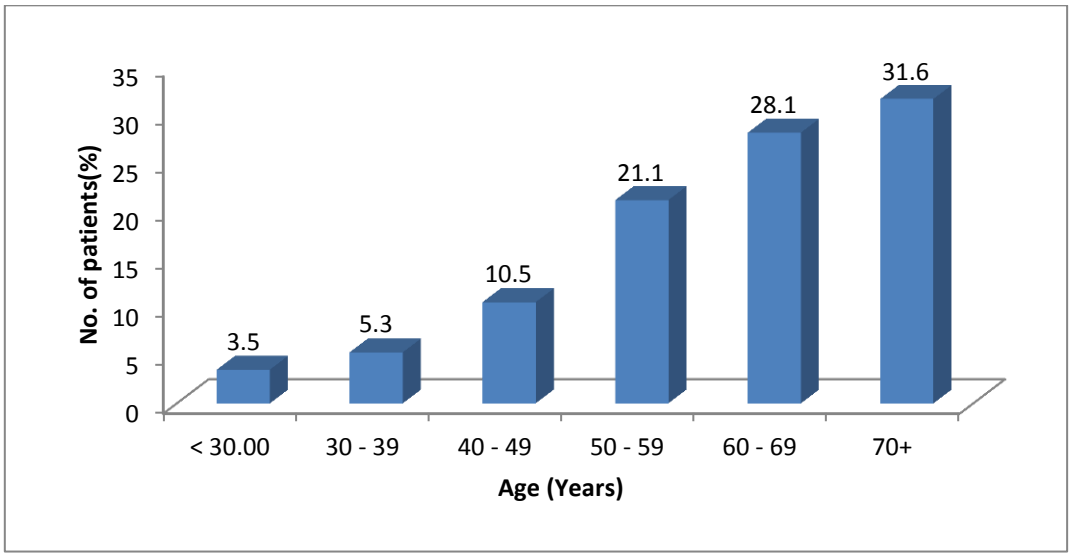


TABLE 6: DISTRIBUTION OF AGE GROUP IN CASES OF HAEMORRHAGE:

Age (Years)	No. of patients	Percentage
< 30	1	6.7
30-40	0	0
40 - 49	2	13.3
50 - 59	5	33.3
60 - 69	3	20.0
70 - 79	3	20.0
80+	1	6.7
Total	15	100.0

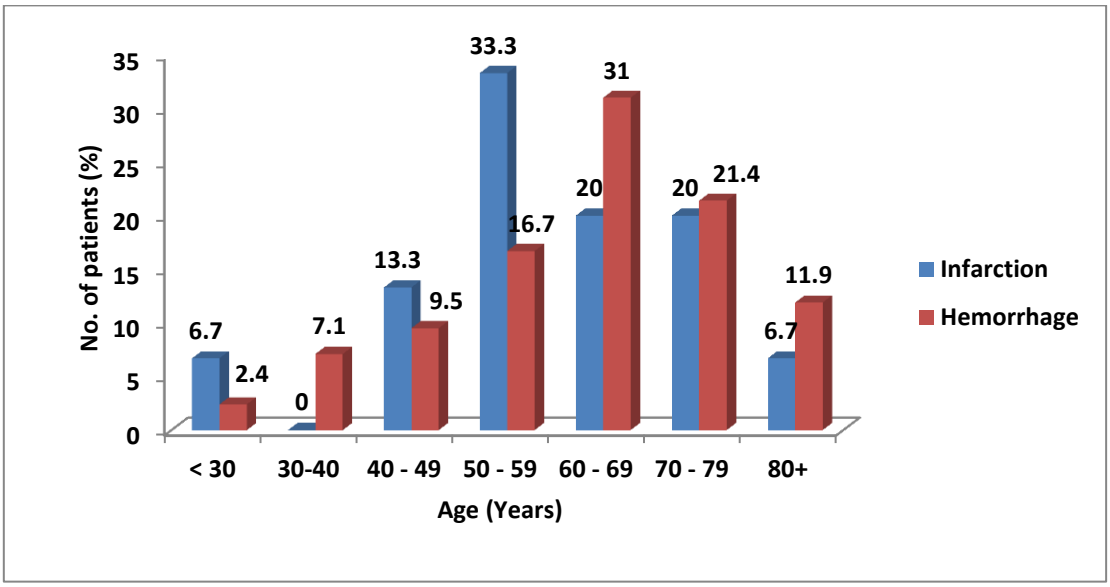
- Cases of hemorrhage were more in the age group of 60-79 (40%)
- With minimum age of haemorrhage being 28 years and the maximum age being 86 years.
- The mean age of haemorrhage in this study is found to be 59.73 years.

TABLE 7: DISTRIBUTION OF AGE GROUP IN CASES OF INFARCTION:

Age (Years)	No. of patients	Percentage
< 30.00	1	2.4
30.00 - 39.00	3	7.1
40.00 - 49.00	4	9.5
50.00 - 59.00	7	16.7
60.00 - 69.00	13	31.0
70.00 - 79.00	9	21.4
80.00+	5	11.9
Total	42	100.0

- Cases of infarction were more in the age group of 60-69 (31%)
- With minimum age of infarction being 27 years and the maximum age being 85 years.
- The mean age of haemorrhage in this study is found to be 60.9 years.

FIGURE 14: AGE DISTRIBUTION OF STROKE CASES:

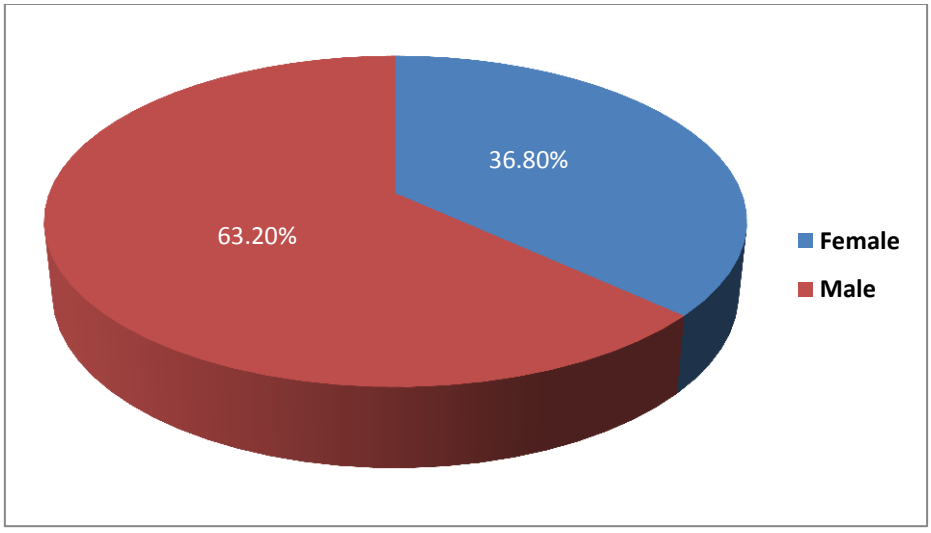


- The age of cerebral infarction being maximum between 50-59 years.
- The age of hemorrhage being maximum between 60-69 years.

TABLE 8: DISTRIBUTION OF PATIENTS ACCORDING TO GENDER

Gender	No. of patients	Percentage
Female	21	36.8
Male	36	63.2
Total	57	100.0

FIGURE 15: SEX DISTRIBUTION OF THE STROKE CASES



- In our study, out of 57 patients, 21 were females and 36 were males

TABLE 9: TYPE OF CEREBROVASCULAR DISEASE IN PATIENTS OF STROKE CONFIRMED BY CT SCAN

Type of Cerebro-vascular disease	No. of patients	Percentage
Hemorrhage	15	26.3
Infarction	42	73.7
Total	57	100.0

- The table shows that the incidence of stroke due to cerebral infarction, 73.7% was higher than cerebral haemorrhage, 26.3%.
- In the present study amongst the patients of cerebral infarction, most common site of vascular territory involvement was left MCA followed by right MCA.
- Similarly, amongst the patients of haemorrhage left basal ganglia was most common site of involvement.

FIGURE 16: TYPE OF CEREBROVASCULAR DISEASE IN PATIENTS OF STROKE CONFIRMED BY CT SCAN

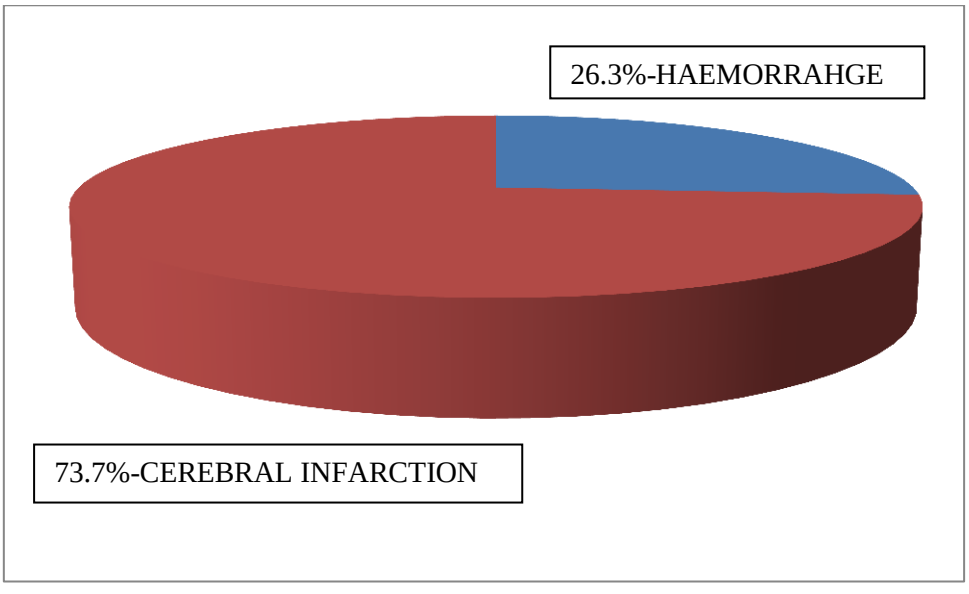


TABLE 10: DISTRIBUTION OF PATIENTS ACCORDING TO HYPERTENSION

Hypertension	No. of patients	Percentage
NO	36	63.2
YES	21	36.8
Total	57	100.0

- Out of the 57 patients, 21 patients (36.8%) were hypertensive.

TABLE 11 : INCIDENCE OF HYPERTENSION IN PATIENTS WITH STROKE

Incidence of Hypertension	Infraction		Hemorrhage	
	Number	Percentage	Number	Percentage
Hypertensive	15	35.71	7	46.67
Non Hypertensive	27	64.29	8	53.33
Total	42	100	15	100

- In our study 35.71% of patients who had infarction were hypertensive and 46.67% of patients who had cerebral hemorrhage were hypertensive.

FIGURE 17: INCIDENCE OF HYPERTENSION IN PATIENTS WITH STROKE

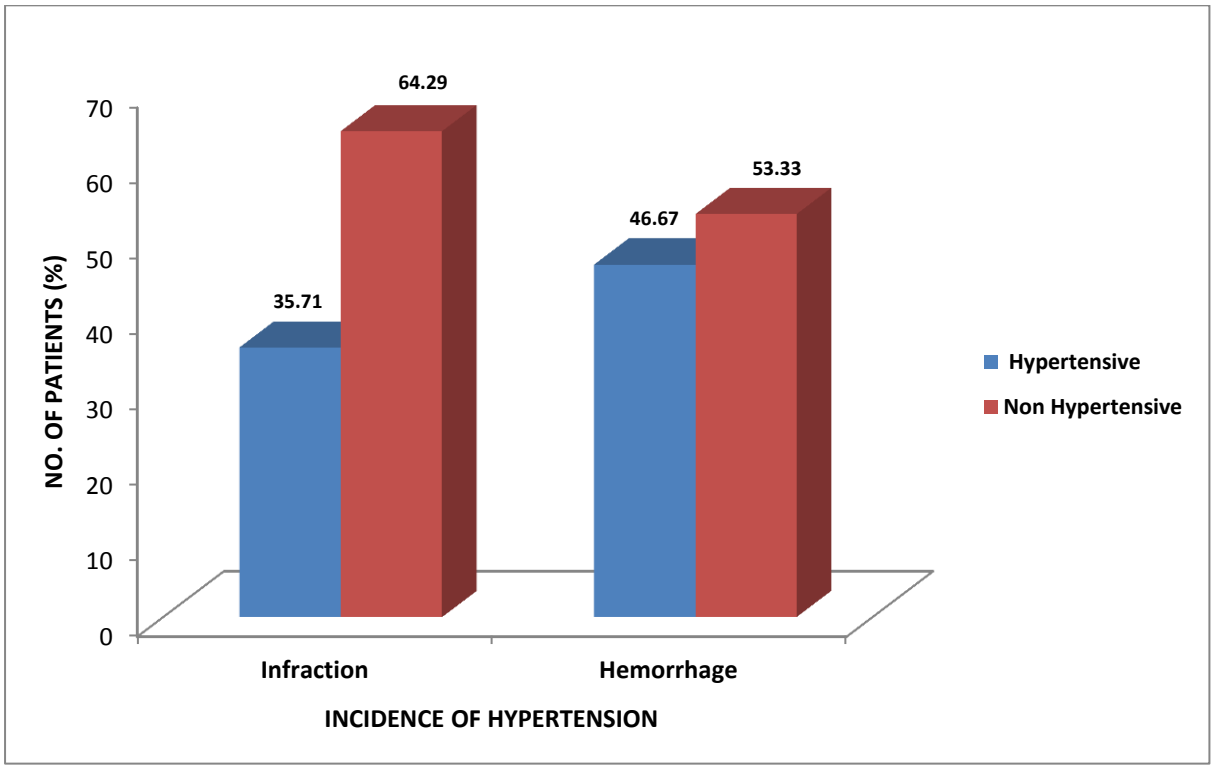


TABLE 12: DISTRIBUTION OF PATIENTS ACCORDING TO CONSCIOUSNESS

CONSCIOUSNESS	NO. OF PATIENTS	PERCENTAGE
ALERT	27	47.4
DROWSY	18	31.6
UNCONS.	12	21.1
Total	57	100.0

Out of the 57 patients who presented with stroke, 27(47.4%) patients were alert, 18(31.6%) patients were drowsy and 12 patients (21.1%) were unconscious at presentation.

FIGURE 18: DISTRIBUTION OF PATIENTS ACCORDING TO CONSCIOUSNESS

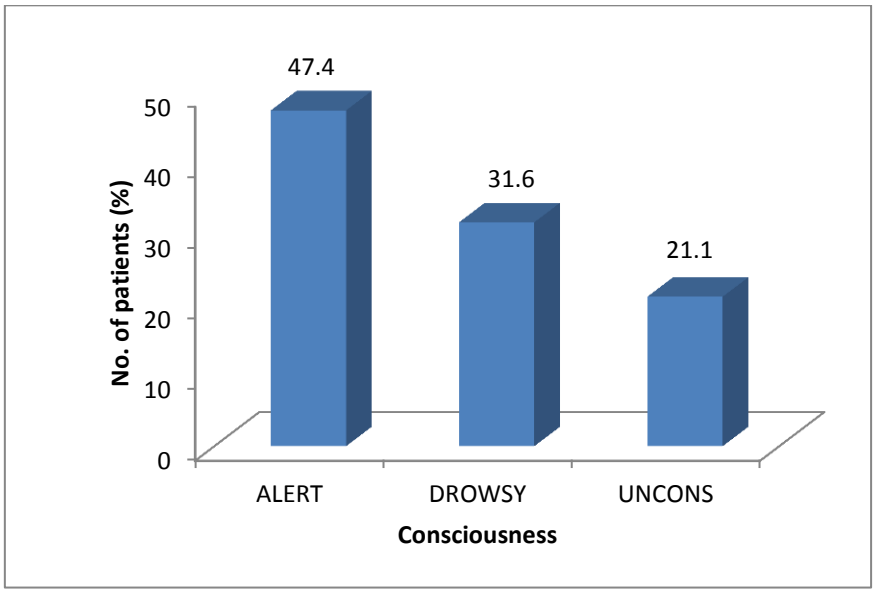


TABLE 13: SYMPTOMS IN PATIENTS WITH STROKE

Symptoms	Infraction(n=42)		Hemorrhage(n=15)	
	Number	Percentage	Number	Percentage
Headache	3	7.14	3	20
Vomiting	35	83.33	15	99.99
Unconscious	17	40.48	14	93.33

In cases of infarction only 3 patients, (7.14%) presented with headache, while 20% presented with headache in the hemorrhage group. History of vomiting was present in 35 patients of infarction (83.33%) and in 15 patients of hemorrhage (99.99%).

In cases of infarction, 17 patients (40.48%) were unconscious on presentation and in cases of hemorrhage 14 patients (93.33%) were unconscious on presentation.

FIGURE 19: SYMPTOMS OF THE PATIENTS IN THE STUDY

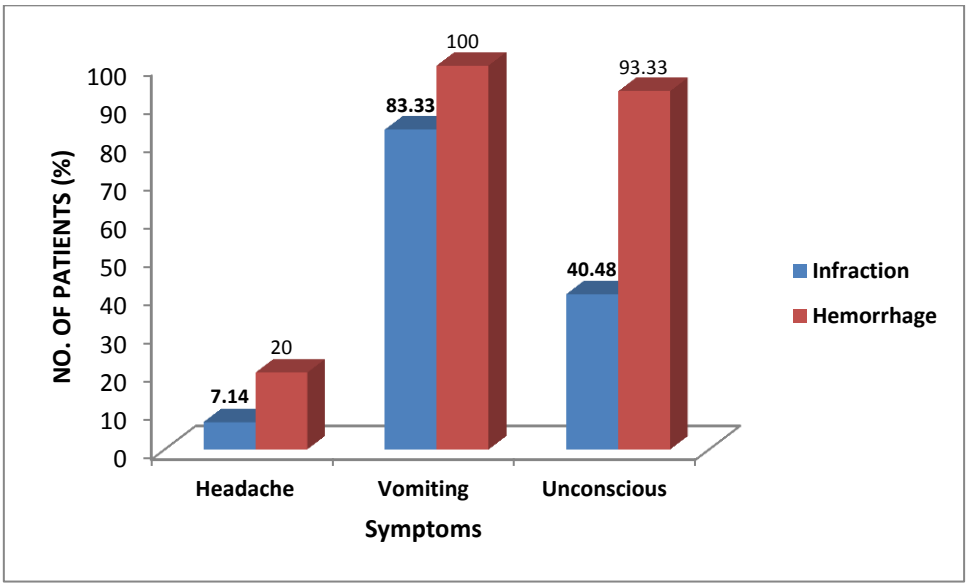


TABLE 14: CORRELATION OF SIRIRAJ STROKE SCORE DIAGNOSIS WITH CT SCAN

Siriraj stroke score diagnosis	Infraction(n=42)		Hemorrhage(n=15)	
	Number	Percentage	Number	Percentage
Correct correction	39	92.8	15	100
Incorrect correction	0	00	0	0
Equivocal	3	7.2	0	0
Total	42	100	15	100

39 (92.8%) patients of infarction and 15(100%) of cerebral haemorrhage were correctly diagnosed by Siriraj score when correlated with CT scan of brain.

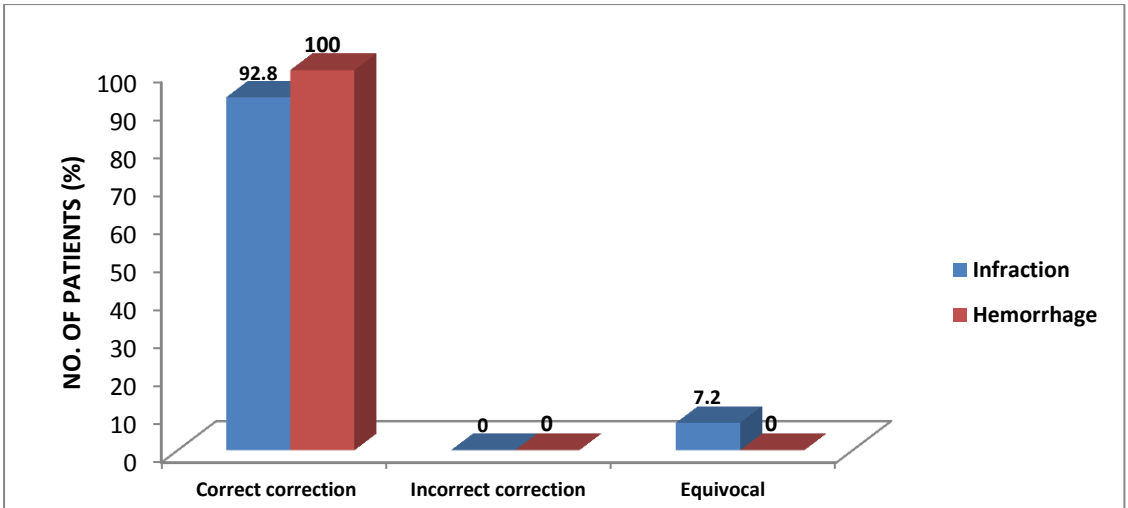


FIGURE 20: SIRIRAJ STROKE SCORE DIAGNOSIS WITH CT SCAN

TABLE 15: RESULT OF SIRIRAJ STROKE SCORE IN COMPARISON WITH CT SCAN DIAGNOSIS

Symptoms	Test Diagnosis		CT Scan	
	Infract	Hemorrhage	Infraction	Hemorrhage
<1	39	0	39	0
-1 to 1	0	0	3	0
>1	0	15	0	15

TABLE 16: RESULT OF SIRIRAJ STROKE SCORE IN COMPARISON WITH CT SCAN

Siriraj score / CT Scan	Hemorrhage(>1)		Non Hemorrhage(<1)		Total
	N	%	N	%	
Hemorrhage	15	100	0	0	15
Non Hemorrhage	3		39		42
Total	18		39		

- Out of 39 patients diagnosed to be having infarction by Siriraj stroke score all 39 were found to be having an infarct by CT scan.
- All the patients diagnosed to have haemorrhage by Siriraj Stroke Score had it proven by CT scan.
- 3 patients whose Siriraj Stroke Score were equivocal, were found to have infarction.

TABLE 17: EVALUATION OF SIRIRAJ STROKE SCORE WITH STATISTICAL PARAMETERS

PARAMETER	INFARCTION	HEMORRHAGE
Sensitivity	96.45%	95%
Specificity	92.57%	95.2%
Positive predictive value	97%	97.88%
Negative predictive value	90.86%	94.76%
Accuracy	94.74%	95.15%

The sensitivity for infarction by Siriraj stroke score is 96.45% while for hemorrhage is 95%. Specificity for infarction by Siriraj stroke score is 92.57% and for hemorrhage is 97.88%. The positive and negative predictive values for infarction are 97% and 90.86% and for hemorrhage are 97.88% and 94.76% respectively.

TABLE 18: CORRELATION OF ALLEN'S STROKE SCORE DIAGNOSIS WITH CT SCAN

Allen's stroke score diagnosis with CT scan	Infraction(n=42)		Hemorrhage(n=15)	
	Number	Percentage	Number	Percentage
Correct correction	42	100	15	100
Incorrect correction	0	0	0	0

- 42 patients of infarction and 15 patients of cerebral haemorrhage were correctly diagnosed by Allen's score when correlated with CT scan of brain.

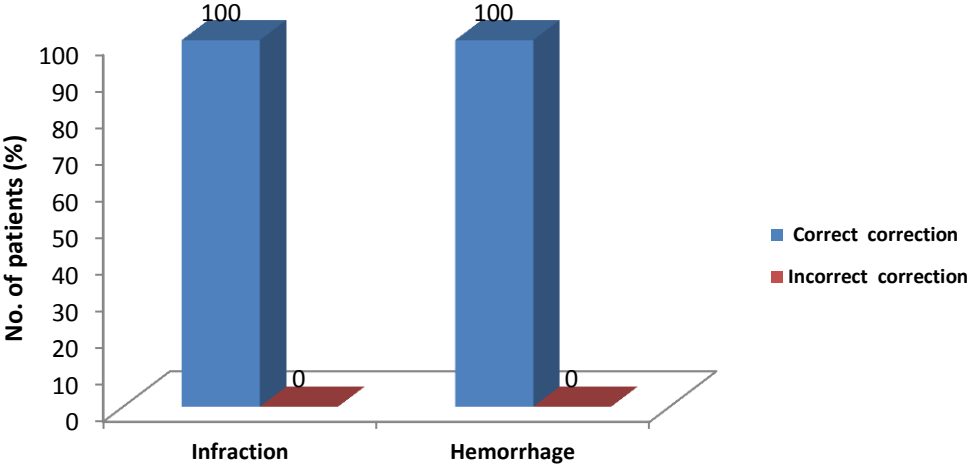


FIGURE 21: ALLEN'S STROKE SCORE DIAGNOSIS WITH CT SCAN

TABLE 19: RESULT OF ALLEN'S STROKE SCORE IN COMPARISON WITH CT SCAN

Allen's score / CT Scan	Hemorrhage(≥14)		Non Hemorrhage(<14)		Total
	N	%	N	%	
Hemorrhage	15	100	0	0	15
Non Hemorrhage	3	7.2	39	92.8	42

Out of 15 patients diagnosed to be having an hemorrhage by Allen's score, 15 were found to be having hemorrhage on CT scan and similarly all 39 patients found to be having infarction by Allen's score were found to have an infarct on the CT scan.

TABLE 20: EVALUATION OF ALLEN'S STROKE SCORE WITH STATISTICAL PARAMETERS:

PARAMETER	INFARCTION	HEMORRHAGE
Sensitivity	93.33%	81.4%
Specificity	92%	95.2%
Positive predictive value	97%	98.00%
Negative predictive value	90.86%	92.76%
Accuracy	94.74%	95.15%

Sensitivity for Infarction by Allen's score is 93.33% while for hemorrhage is 81.4%. Specificity for infarction by Allen's score is 92% and for hemorrhage is 95.2%. The positive and negative predictive values for infarction are 97% and 90.86% and for hemorrhage are 98% and 92.76% respectively.

TABLE 21: DISTRIBUTION OF PATIENTS ACCORDING TO SBP

SBP	No. of patients	Percentage
< 120	6	10.5
120- 139	11	19.3
140- 159	12	21.1
160+	28	49.1
Total	57	100.0

Most of the patients presenting with a stroke had a systolic blood pressure of more than 160mm of hg (49.1%).

FIGURE 22: DISTRIBUTION OF PATIENTS ACCORDING TO SBP

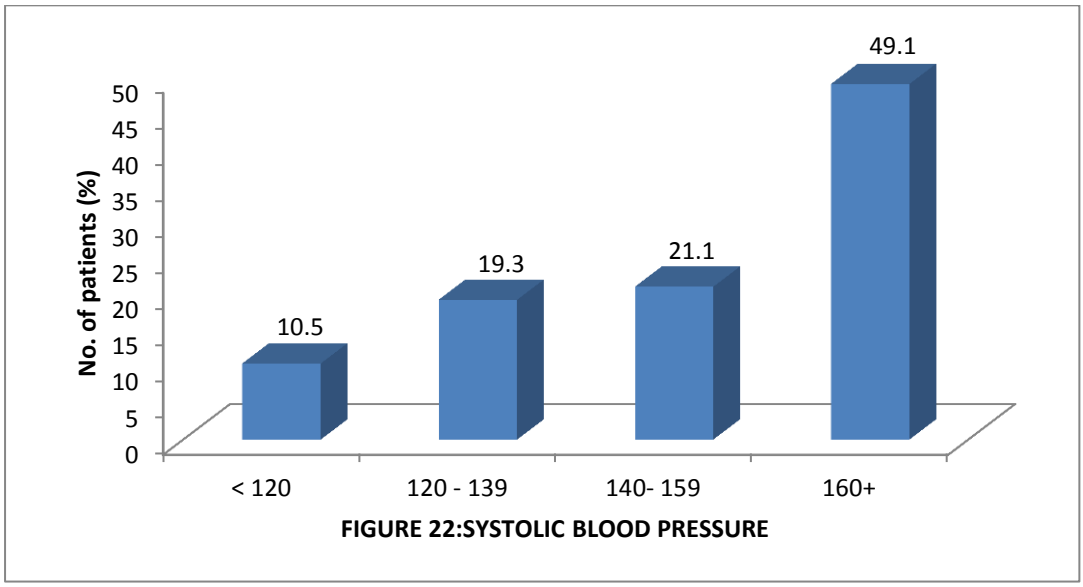


TABLE 22: DISTRIBUTION OF PATIENTS ACCORDING TO DBP

DBP	No. of patients	Percentage
< 80	7	12.3
80- 89	12	21.1
90- 99	17	29.8
100+	21	36.8
Total	57	100.0

Most of the patients presenting with a stroke had a diastolic blood pressure of more than 100mm of hg (36.8%).

FIGURE 23: DISTRIBUTION OF PATIENTS ACCORDING TO DBP

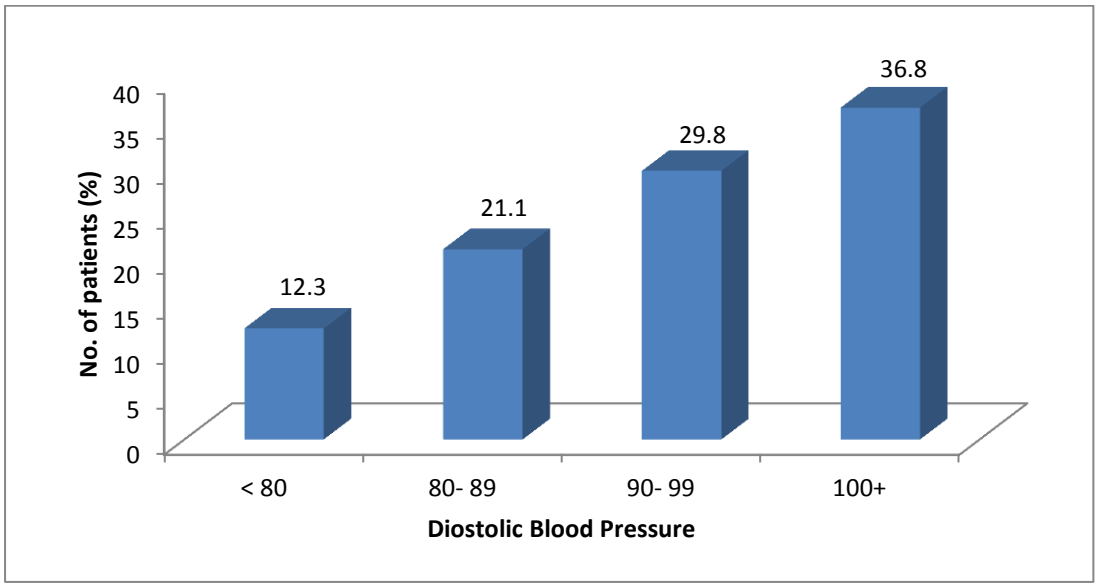


TABLE 23: DISTRIBUTION OF PATIENTS ACCORDING TO ATHEROMA MARKERS

ATHEROMA MARKERS	No. of patients	Percentage
NO	33	58
YES	24	42
Total	57	100.0

Out of the 57 patients, 24 patients (42%) had atheroma markers which included Diabetes Mellitus, Previous history of angina and history of intermittent claudication.

FIGURE 24: DISTRIBUTION OF PATIENTS ACCORDING TO ATHEROMA MARKERS

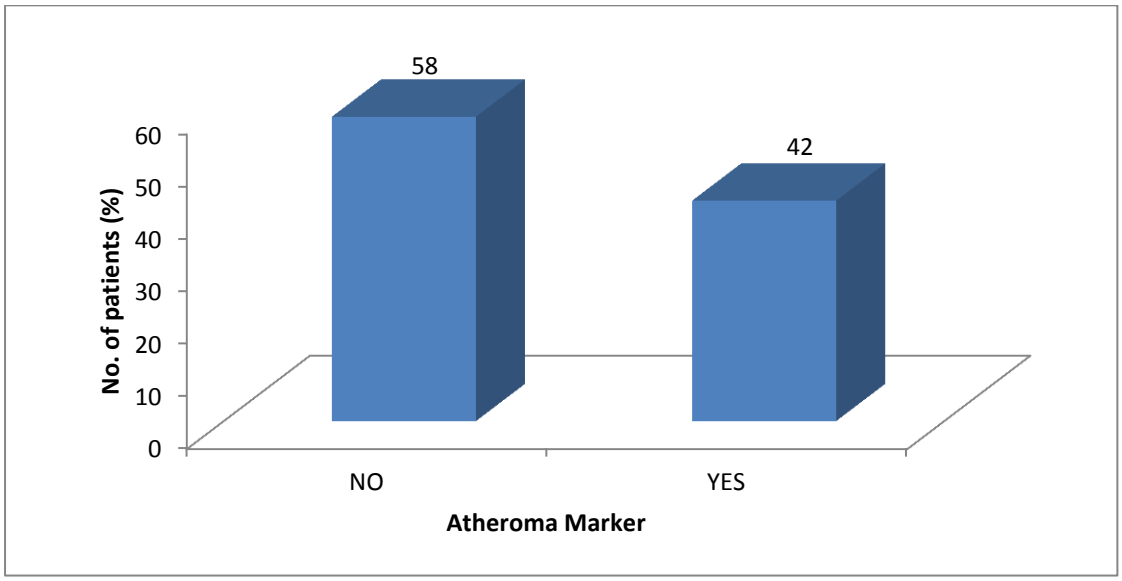


TABLE 24: DISTRIBUTION OF PATIENTS ACCORDING TO TOTAL CHOLESTEROL

TOTAL CHOLESTEROL	No. of patients	Percentage
<200	44	77.2
200.00+	13	22.8
Total	57	100.0

About 12 patients (22.8%) of the total number of stroke cases had deranged levels of high density lipoproteins and 44 patients(77.2%) had normal levels.

FIGURE 25: DISTRIBUTION OF PATIENTS ACCORDING TO TOTAL CHOLESTEROL

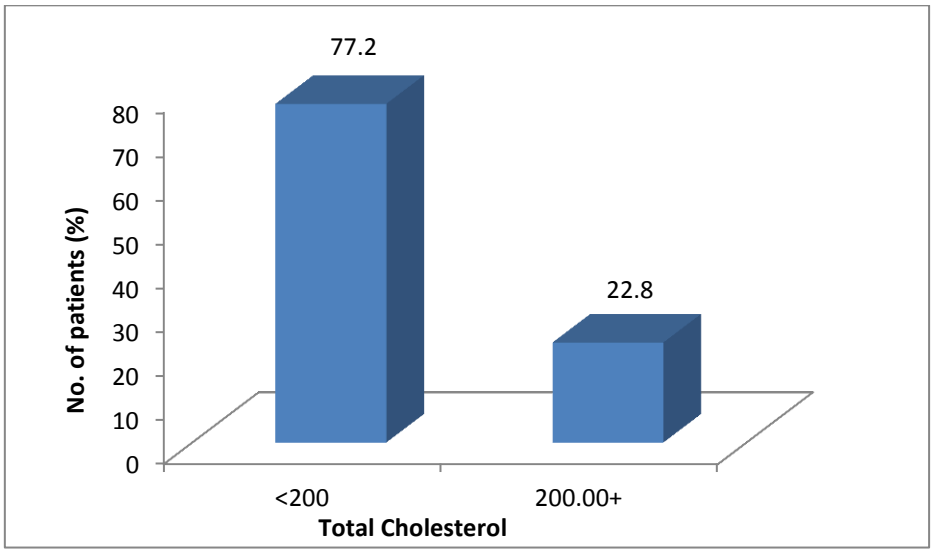
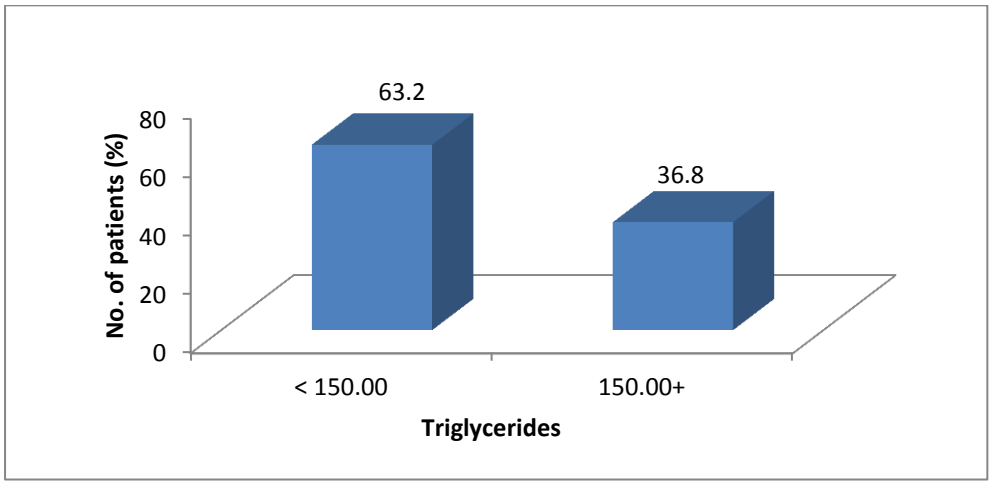


TABLE 25: DISTRIBUTION OF PATIENTS ACCORDING TO TRIGLYCERIDES

TRIGLYCERIDES	No. of patients	Percentage
< 150.00	36	63.2
150.00+	21	36.8
Total	57	100.0

About 36.8% of the total patients with stroke, 21 patients had hypertriglyceridemia and the remaining 63.2% of the patients had normal triglyceride levels.

FIGURE 26: DISTRIBUTION OF PATIENTS ACCORDING TO TRIGLYCERIDES



DISCUSSION

Cerebral haemorrhage is found in 5-10% of patients with stroke in Europe and America[45]. But in the developing countries, the incidence is far more common, partly because of poorly controlled hypertension and irregular treatment. It is well established that the management and prognosis of the acute stroke syndrome depends on the diagnosis of haemorrhage or infarction. Hence, it is crucial to establish the correct diagnosis as soon as possible.

Computerised tomography is an accurate, safe and non-invasive procedure for differentiating between cerebral haemorrhage and infarction. However, this facility is not freely available in India and other developing countries and even if available, many cannot afford it. Primary care physicians have to resort to the usual routine procedures in conjunction with the clinical features, which have been shown unreliable.

In 1984, Allen's score was developed as a clinical diagnostic tool for differentiating between the two types of stroke and it was later validated by other investigators[46]. The score for each patient is obtained by thirteen clinical variables with a constant. Calculating Allen's score at the bedside is not easy. The Siriraj Stroke score was developed so that only simple calculations would be needed, making it more readily applicable at the bedside[47].

A cerebral haemorrhage usually resulted in a very high score which was clearly distinguished from the score in infarction. Both the scores however, had a tilt towards infarction in the method of calculation, which may result in the under diagnosis of haemorrhage. As the prevalence of haemorrhagic stroke differs between various populations and the predictive value of any diagnostic score depends greatly on the prevalence of the disease being considered; the score systems may not be applicable transculturally. Hence, they are needed

to be validated before they can be accepted as a screening diagnostic tool.

The Siriraj Stroke Score and the Allen's stroke score considers the onset of symptoms, the level of consciousness, history of vomiting and history of headache and was given different scores.

Both scores consider the diastolic blood pressure of the patients in their scoring systems. Both scores consider the atheroma markers like diabetes, angina and intermittent claudication in their scores showing their importance in differential diagnosis of acute stroke.
Allen's stroke score also considers the history of hypertension, previous history of transient ischemic attack or stroke and history of heart disease.

In the present study, sensitivity of Siriraj stroke score in accurately diagnosing infarction is 96.45% and haemorrhage is 95% and specificity for infarction is 88.57% and for hemorrhage is 95.2%.
In comparison to sensitivity of previous validation studies, sensitivity of infarction in study by Celani et al by Siriraj Score was 93.3% and for haemorrhage was 61%

Sensitivity for infarction by Siriraj Stroke Score validation study was 93.2% and for haemorrhage was 89.3%.

Sensitivity for infarction by Hung LY et al for infarction was 90% and for haemorrhage was 85% by Siriraj Stroke Score.

In one study conducted in India by Jyothi Wadhwani et al, sensitivity for infarction was

92.54% and for haemorrhage was 87% by Siriraj stroke Score.

In another study conducted in India by Dr SSS Rao Kudaravalli, sensitivity of Siriraj stroke score in diagnosing infarction was 88.33% and that in diagnosing haemorrhage is 85%

Comparison of Sensitivity of the present study with other validation studies on Siriraj Stroke score:

TABLE 26: COMPARISION OF SENSITIVITY

STUDY	INFARCTION (%)	HAEMORRHAGE (%)
Celani et al[41]	93.30	61.00
Siriraj Stroke Validation Study[42]	93.20	89.30
Hung LY et al[43]	90.00	85.00
Jyothi Wadhwani et al[55]	92.54	87.00
SSS Rao Kudaravalli	88.33	85.00
Present Study	96.45	95.00

The positive predictive value of Siriraj Stroke Score in diagnosing infarction is 97% and haemorrhage is 97.88%.

In study by Celani et al, positive predictive value of Siriraj Stroke Score in diagnosing infarction is 93% and haemorrhage is 63%.

In Siriraj Stroke Score Validation study, positive predictive value for infarction is 73.92% and for haemorrhage is 91.27%.

In SSS Rao Kudaravlli's study, the positive predictive value in diagnosing infarction is 94.64% and for haemorrhage is 94.44%

Compared to previous studies, positive predictive value for both infarction and haemorrhage by Siriraj Stroke Score is high in the current study.

TABLE 27: COMPARISION OF PREDICTIVE VALUE OF POSITIVE TEST WITH OTHER VALIDATION STUDIES ON SIRIRAJ STROKE SCORE

STUDY	INFARCTION (%)	HAEMORRHAGE (%)
Celani et al[53]	93.00	63.00
Siriraj Stroke Validation Study[54]	73.92	91.27
SSS Rao Kudaravalli	94.64	91.27
Present Study	97	97.88

The sensitivity of Allen's score in diagnosing infarction is 93.3% and haemorrhage in the present study is 81.4%.

In the study by Celani et al, sensitivity of Allen's score for infarction is 91%.

In study by PAG Sandercock et al, sensitivity of Allen's score for infarction is 78%.

In another study conducted in India by Jyothi Wadhwani et al, sensitivity of Allen's score for infarction was 93.42% and for infarction was 66.66%.

The comparison of Allen's score in the diagnosis of infarction with other validation studies is given below.

TABLE 28: COMPARISON OF SENSITIVITY OF THE PRESENT STUDY WITH OTHERVALIDATION STUDIES ON ALLEN'S STROKE SCORE

STUDY	INFARCTION (%)	HAEMORRHAGE (%)
Celani et al[53]	91.00	-
PAG Sanducock et al	78.00	-
Jyothi Wadhwani et al	93.42	66.6
SSS Rao Kudaravalli	95.00	66.6
Present Study	93.30	81.4

The predictive value of a positive test in the present study in the present study for infarction is 97% and that for haemorrhage by Allen's score is 98%

In the study by PAG Sandercock [(48)] et al, the positive predictive value in the diagnosis of infarction by Allen's score is 78%

In the study conducted by Celani et al, the positive predictive value in the diagnosis of infarction by Allen's score was 93%

TABLE 29: COMPARISION OF PREDECTIVE VALUE OF A POSITIVE TEST OF PRESENT STUDY IN THE DIAGNOSIS OF INFARCTION WITH OTHER VALIDATION STUDIES ON ALLEN'S STROKE SCORE

STUDY	INFARCTION (%)
PAG Sanducock et al	78.00
Celani et al	93.00
SSS Rao Kudaravalli	83.82
Present Study	97.00

The overall predictive accuracy of Siriraj Stroke score by present study is 95.15%. In the study conducted by Hung L Y et al, the overall predictive accuracy by Siriraj Stroke Score was 88.5%

In Siriraj Stroke Score Validation study, the overall predictive accuracy of Siriraj Stroke Score was 90.3%.In the study by Dr SSS Rao Kudaravalli, the predictive accuracy was 87%. In the study by Jyothi Wadhwani et al, the overall predictive accuracy of Siriraj Stroke Score was 91.11%

TABLE 30: COMPARISION OF OVERALL PREDICTIVE ACCURACY OF SIRIRAJ STROKE SCORE OF PRESENT STUDY WITH OVER VALIDATION STUDIES

STUDY	OVERALL PREDICTIVE VALUE (%)
Siriraj Stroke Score Validation study	90.30
Hung LY et al	88.50
Jyothi Wadhwani et al	91.11
Dr SSS Rao kudaravalli	87.00
Present Study	95.15

The overall predictive accuracy of Allen's score by present study is 94.74%. In the study by Jyothi Wadhwani et al, the overall predictive accuracy of Allen's score was 87%. And in the study by Dr SSS Rao Kudaravalli, the predictive accuracy was 86%.

TABLE 31: COMPARISON OF OVERALL PREDICTIVE ACCURACY OF ALLEN'S STROKE SCORE OF PRESENT STUDY WITH OTHER VALIDATION STUDIES

STUDY	OVERALL PREDICTIVE VALUE (%)
Jyothi Wadhwani et al	87.00
Dr SSS Rao kudaravalli	86.00
Present Study	94.74

Both the scores are based mainly on clinical symptomatology. In this study all patients shown to have a large infarct (ischemic stroke) by CT scan, but who were predicted by clinical scores to have haemorrhage due to their severe symptoms and therefore high apoplectic onset scores. On the other way, patients with a small haemorrhage as detected by CT scan had clinical score favouring infarction because of their minimal symptoms and therefore incorrectly diagnosed as infarction by clinical scores.

Another fallacy is that both scores lack formal definitions for some variables. The main problem relates to the level of consciousness because it is an important weighing factors in both scoring protocols. In large infarcts cerebral oedema leads to midline shift, brainstem compression, alteration in the level of consciousness and gives false interpretation of haemorrhage.

Hypertension is one of the most important risk factor for both infarcts as well as haemorrhage, but in both scores high blood pressure favours more haemorrhage than the

infarction and hence gives false results.

In general both scoring methods tend to classify severe strokes as haemorrhagic strokes and strokes of less severity as ischemic regardless of their aetiology, therefore both scoring methods needs modifications in their variables.

When computerised tomography is not immediately available and the doctors wish to start antithrombotic therapy, Siriraj stroke score can be applies for the differential diagnosis of haemorrhage and infarction as the sensitivity and predictive value of a positive test for haemorrhage were higher when compared to that of Allen's score. Secondly Allen's score needs a very detailed evaluation and history, at least seventy-hours of observation and is much more difficult to calculate.

Since, the Siriraj stroke score was introduced as a diagnostic tool for the acute stroke syndrome in Thailand, at least half of all the patients with stroke have been spared CT scan. An estimated seven million pounds a year is saved. A similar massive benefit can be expected in our country as well.

Further validation studies requiring large population are required in India before the widespread use of Siriraj stroke score as a routine screening method to differentiate cerebral haemorrhage from infarction can be accepted.

CONCLUSION

- In the present study, the predictive accuracy of Siriraj stroke score for infarction is 97% and haemorrhage is 95.15%. Sensitivity is 96.45% for infarction and 95% for haemorrhage.

- In the present study, the predictive accuracy of Allen's score for infarction is 94.74% and haemorrhage is 95.15%. Sensitivity was 93.3% for infarction and 81.4% for haemorrhage.

- The sensitivity for infarction was higher with Siriraj score (96.45% versus 93.3%), and also Siriraj score had a higher sensitivity for haemorrhage (95% versus 81.4%).

- The Siriraj stroke score is much simpler, easier to calculate and the time needed to diagnose is much less than Allen's score, which is difficult to calculate, needs detailed clinical evaluation and at least monitoring for 24 hours.

- When clinicians wish to start antithrombotic treatment while waiting for the scan results, they can rely on Siriraj stroke score, as the sensitivity to detect haemorrhage is much higher than Allen's score.

- Further validation studies requiring a large number of participants are needed before wide acceptance of Siriraj stroke as a screening tool in the diagnosis of stroke.

- When both the scores are considered together in a same patient, sensitivity for diagnosis of infarction and haemorrhage is increased.

SUMMARY

In our study, a total of 57 cases of stroke were considered out of which --- were proven to be of cerebral infarction and --- were proved to be of cerebral hemorrhage

The Siriraj stroke score has identified 42 cases of 57 as cerebral infarction with a sensitivity of 96.45% and specificity of 88.57%.

The Siriraj stroke score has identified 15 cases of 57 as cerebral haemorrhage with a sensitivity of 95% and specificity of 95.2%

The overall predictive accuracy of Siriraj Stroke Score in differential diagnosis of acute stroke into infarction is 94.74% and for haemorrhage is 95.15%

The Allen's score has identified 42 cases as cerebral infarction with a sensitivity of 93.3% and specificity of 92%

The Allen's score has identified 15 cases as cerebral haemorrhage correctly with a sensitivity of 81.4% and specificity of 95.2%

The overall predictive accuracy of Allen's or Guy's hospital score in the differential diagnosis of acute stroke into infarction is 94.74% and for haemorrhage is 95.15%

In developing countries like India, where CT scan facilities are not readily available and when the clinicians wishes to start antithrombotic therapy, they can rely upon the Siriraj and Allen's Stroke scores by carefully evaluating all the variables in the scoring systems. With minor modifications in the variables of both the scoring systems, results would be more appropriate.

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ANNEXURE I

B.L.D.E (Deemed to be University)
SHRI.B.M.PATIL MEDICAL COLLEGE HOSPITAL & RESEARCH CENTRE
VIJAYAPUR – 586103

IEC/NO-286/18
17/11/2018

INSTITUTIONAL ETHICAL COMMITTEE

INSTITUTIONAL ETHICAL CLEARANCE CERTIFICATE

The Ethical Committee of this college met on 13-11-2018 at 03-15 PM scrutinize the Synopsis of Postgraduate Students of this college from Ethical Clearance point of view. After scrutiny the following original/corrected and revised version synopsis of the Thesis has accorded Ethical Clearance.

Title : Siriraj and allens score in the differential diagnosis of acute stroke and correlation with computed tomography scan of brain.

Name of P.G. Student : Dr Nayana.R
Department of General Medicine.

Name of Guide/Co-investigator: Dr. R.C.Bidri, Professor of General Medicine.

DR RAGHAVENDRA KULKARNI
CHAIRMAN
Institutional Ethical Committee

Following documents were placed before E.C. for Scrutinization:

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

ANNEXURE II

SHRI B.M. PATIL MEDICAL COLLEGE HOSPITAL AND RESEARCH CENTRE,VIJAYAPURA-586 103

RESEARCH INFORMED CONSENT FORM

TITLE OF THE PROJECT : SIRIRAJ AND ALLENS SCORE IN THE DIFFERENTIAL DIAGNOSIS OF ACUTE STROKE AND CORRELATION WITH COMPUTED TOMOGRAPHY SCAN OF BRAIN

PG GUIDE : DR. R. C. BIDRI

PG STUDENT : DR. NAYANA R

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

BENEFITS:-

I understand that my participation in this study will help the investigator to diagnose the disease better and will help in the management of the disease.

PROCEDURE:-

I understand that relevant history will be taken and I will undergo detailed clinical examination after which necessary investigations will be done and accordingly treatment will be given.

RISK AND DISCOMFORTS:-

I understand there is no risk involved and I will experience no pain during the procedures performed.

CONFIDENTIALITY:-

I understand that medical information produced by this study will become a part of

my hospital records and will be subjected to the confidentiality and privacy regulation of the said hospital. Information of a sensitive personal nature will not be a part of the medical records, but will be stored in the investigator's research file.

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

REQUEST FOR MORE INFORMATION:-

I understand that I may ask more questions about the study at any time Concerned. The researcher is available to answer my questions or concerns. I understand that I will be informed of any significant new findings discovered during the course of this study, which may influence my continued participation.

REFUSAL OR WITHDRAWAL OF PARTICIPATION:-

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

INJURY STATEMENT:-

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

I have explained to (patient's / relevant guardian's name) the purpose of the research, the procedures required, and the possible risks and benefits to the best of my ability in patient's own language.

_____ _____

Investigator / P. G. Guide Date

I confirm that …….(Name of the PG guide / chief researcher) has explained to me the research, the study procedures that I undergo and the possible risks and discomforts as well as benefits that I may experience. I have read and I understand this consent form. Therefore, I agree to give my consent for my participation as a subject in this research project.

_____ _____

Participant / guardian Date

_____ _____

Witness to signature Date

APPENDIX – VII

SCHEME OF CASE TAKING

B.L.D.E.U'S SHRI B. M. PATIL MEDICAL COLLEGE HOSPITAL AND RESEARCH CENTRE, VIJAYAPURA.

DEPARTMENT OF MEDICINE

PROFORMA

"SIRIRAJ AND ALLENS SCORE IN THE DIFFERENTIAL DIAGNOSIS OF ACUTE STROKE AND CORRELATION WITH COMPUTED TOMOGRAPHY SCAN OF BRAIN"

Name: CASE NO:

Age: OP/IP NO:

Sex: DOA:

Religion: DOD:

Occupation:

Address:

Presenting Complaints:

History of presenting complaints:

Past History:

Family history:

Personal History:

Diet –

Appetite –

Sleep –

Bowel –

Bladder –

Habits –

Obstetric and menstrual history (in females):

Physical Examination:

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

Vitals:

Pulse rate: Respiratory rate:

Blood pressure: Temperature:

Systematic Examination:

1.Nervous system examination:

Appearance and Behaviour:

Consciousness:

- (If conscious)
  - Oriented
  - Confused
  - Drowsy
  - Stupor
  - Coma
- If consciousness is diminished/ in coma

GCS SCORING:

Eye opening: SCORE:

- Open spontaneously 4
- Open only to verbal stimuli 3
- Open only to pain 2
- Never open 1

Best verbal response: SCORE:

- Oriented and converses 5
- Converses, but disoriented, confused 4
- Uses inappropriate words 3
- Makes incomprehensible sounds 2
- No verbal response 1

Best motor response: SCORE:

- Obeys commands 6
- Localizes pain 5
- Exhibits flexion withdrawal 4
- Decorticate rigidity 3
- Decerebrate rigidity 2
- No motor response 1

TOTAL GCS SCORE:

Handed ness:

Mental status examination

i.Memory :

a) Immediate-

b) Recent-

c) Remote-

iiOrientation :

iii.Speech and language :

iv intelligence:

c.Cranial nerves examination:

I. Olfactory Nerve

II. Optic Nerve

III. Oculomotor Nerve

IV. Trochlear Nerve

V. Trigeminal Nerve

VI. Abducens Nerve

VII. Facial Nerve

VIII. Vestibulocochlear Nerve

IX. Glossopharyngeal Nerve

X. Vagus Nerve

XI. Accesory Nerve

XII. Hypoglossal Nerve

Motor system examination:

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

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

d.Sensory system examination:

	Right	Left

Touch

Pain

Temperature

Vibration

Joint sense

Cortical sensation

e.Cerebrallar system examination:

	Right	Left
Finger Nose test		
Knee Heel test		
Dysdiadokinesia		

g.GAIT:

h.Signs of Autonomic Disturbances – Present/Absent

i.Signs of Meningeal iriitation

Neck Rigidity/Kernings sign/Brudzenski sign

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

2. Cardiovascular System:

Examination of the arterial System

- Pulse Rate
- Rhythm
- Volume
- Character
- Condition of the vessel wall
- Equality on both the sides
- Radio-radial delay
- Radio-femoral delay
- Other Peripheral pulsations R L

Dorsalis Pedis

Posterior Tibial

Popliteal

Femoral

Brachial

Carotid

Blood Pressure:

Signs Of Infective Endocarditis:

Signs of congestive cardiac Failure:

Precordial Examination:

Inspection:

Apical impulse location, character

Shape of precordium

Other pulsations

Scars and sinuses

Palpation:

Apical impulse: Site

Character

Thrills:

Epigastric Pulsation

Parasternal Heave

Auscultation:

Mitral area:

Tricuspid area:

Pulmonary area:

Aortic area:

3. Respiratory System:

4. Per Abdomen Examination:

Final Clinical diagnosis:

SIRIRAJ STROKE SCORE:

SL NO	VARIABLE	CLINICAL FEATURES	SCORE	SCORE
1	Level of consciousness	• Alert • Drowsy • Semicomatose/ Comatose	0 1 2	
2	History of vomiting after onset	• Absent • Present	0 1	
3	History of Headache within 2 hours of onset	• Absent • Present	0 1	
4	Diastolic Blood pressure	In mm of hg		
5	Atheroma Markers	• None • One or more	0 1	
			TOTAL	

SIRIRAJ SCORE:

SSS = (2.5x (1)) + (2x (2)) + (2x (3)) + (0.1x DBP) – (3x (5)) -12

STROKE PROTOCOL:

ALLEN'S SCORE:

VARIABLE	CLINICAL FEATURES	SCORE	PT SCORE
Apoplectic onset Loss of consciousness	None or more	0	
Headache within two hours, vomiting, neck stiffness	Two or more	21.9	
Level of consciousness (24 hours after admission)	• Alert • Drowsy • Unconscious	0 7.3 14.6	
Plantar responses	• Both flexor or single extensor • Both extensor	0 7.1	
Diastolic blood pressure(24 hours after admission) (*0.17)			
Atheroma markers (diabetes ,angina ,intermittent claudication)	None One or more	0	
History of Hypertension	Not present Present	0 -4.1	
Previous event(Transient Ischemic Attack)	None Any number of previous events	0 -6.7	
Heart Disease	• None • Aortic or mitral murmur • Cardiac failure • Cardiomyopathy • Atrial fibrillation • Cardiomegaly • MI within 6 months	0 -4.3 -4.3 -4.3 -4.3 -4.3 -4.3	
Constant		-12.6	
		TOTAL	

ELECTROCARDIOGRAM:

Standardisation:

Rate, Rhythm:

P Wave:

PR Interval:

QRS Complex:

ST Segment:

T Wave:

Axis:

FINAL INTERPRETATION:

2-DIMENSIONAL ECHOCARDIOGRAPHY:

COMPLETE BLOOD COUNT:

Hemoglobin	gm. %
Total WBC counts	Cells/mm^3
Differential counts -	
Neutrophils	%
Lymphocytes	%
Eosinophils	%
Monocytes	%
Basophils	%
ESR	mm after 1 hour

RANDOM BLOOD SUGAR LEVEL:

FASTING BLOOD SUGAR LEVEL:

POST-PRANDIAL SUGAR LEVEL:

LIDPID PROFILE:

PARAMETER	VALUES (mg/dL)
Serum Triglyceride	
Serum Cholestrol(total)	
Serum Cholestrol(HDL)	

Serum Cholestrol(LDL)	
Serum Cholestrol(VLDL)	

Patient condition on discharge : Improved/Worsened/same/Expired

CONCLUSION:

DATE:

SIGNATURE:

ALLEN'S SCORE												SIRIRAJ'S SCORE									CT DIAGNOSIS	AGE	GENDER	SBP	DBP	HTN	ATHEROMA M	CONS.	LIPID PROFILE					BLD SUGAR	ECHO
HOSP NO.	ONSET	CONSC.	PLANTARS	DBP	ATHEROMA	HTN	PRE-EVENTS	HEART DISEASE	CONSTANT	SCORE	DIAGNOSIS	HOSP NO	CONS.	VOMITTING	HEADACHE	DBP	ATHEROMA	CONSTANT	SCORE	DIAGNOSIS									TG	TC	HDL	LDL	VLDL		
15933	0	0	0	15.3	0	-4.1	0	0	-12.6	-1.4	INFARCTION	15933	0	0	0	9	-3	-12	-6	INFARCTION	INFARCTION	66	M	160	90	YES	YES	ALERT	118	200	42	144	25	198	LVH
13700	21.9	14.6	0	18.7	0	0	0	0	-12.6	42.6	HEMORRHAGE	13700	5	2	2	13	0	-12	10	HEMORRHAGE	HEMORRHAGE	67	F	216	130	NO	NO	UNCONS	114	211	45	144	23	166	LVH
13329	0	7.3	0	13.6	0	0	0	0	-12.6	8.3	INFARCTION	13329	2.5	2	0	8	0	-12	0.5	EQUIVOCAL	INFARCTION	80	F	150	70	NO	NO	DROWSY	130	158	33	99	26	73	N
18588	0	0	0	15.3	0	-4.1	0	0	-12.6	-1.4	INFARCTION	18588	0	2	0	9	0	-12	-1	INFARCTION	INFARCTION	60	M	140	90	YES	NO	ALERT	114	198	44	126	26	110	N
2591	21.9	7.3	0	17	-3.7	-4.1	-6.7	0	-12.6	19.1	HEMORRHAGE	2591	2.5	2	2	10	-3	-12	1.5	HEMORRHAGE	HEMORRHAGE	64	M	190	100	YES	YES	DROWSY	110	202	46	128	28	128	LVH
5752	21.9	7.3	7.1	13.6	0	-4.1	0	0	-12.6	33.2	HEMORRHAGE	5752	2.5	2	2	8	0	-12	2.5	HEMORRHAGE	HEMORRHAGE	75	M	150	89	NO	NO	DROWSY	118	212	64	128	26	109	N
5793	21.9	14.6	7.1	11.9	0	0	0	0	-12.6	42.9	HEMORRHAGE	5793	2.5	2	2	8	0	-12	2.5	HEMORRHAGE	HEMORRHAGE	75	M	130	80	NO	NO	DROWSY	110	200	56	130	24	106	LVH
5405	0	14.6	0	11.9	-3.7	0	0	-8.6	-12.6	1.6	INFARCTION	5405	5	0	0	7	-3	-12	-3	INFARCTION	INFARCTION	70	F	130	70	NO	YES	UNCONS	118	200	56	134	28	92	RHD,IHD
5675	0	7.3	0	13.6	-3.7	-4.1	-6.7	0	-12.6	-6.2	INFARCTION	5675	0	0	0	10	-3	-12	-5	INFARCTION	INFARCTION	56	M	164	100	YES	YES	ALERT	185	165	43	85	37	122	N
6185	0	0	0	11.9	0	0	0	0	-12.6	-0.7	INFARCTION	6185	0	0	0	9	0	-12	-3	INFARCTION	INFARCTION	76	F	170	90	NO	NO	ALERT	188	175	24	114	38	145	N
28763	0	0	0	13.6	0	0	0	0	-12.6	1	INFARCTION	28763	0	0	0	8	0	-12	-4	INFARCTION	INFARCTION	62	M	140	100	NO	NO	ALERT	163	194	39	123	33	80	N
8508	21.9	14.6	0	13.6	0	-4.1	0	0	-12.6	33.4	HEMORRHAGE	8508	5	2	0	8	-3	-12	0	EQUIVOCAL	INFARCTION	35	M	130	90	YES	NO	UNCONS	200	180	28	116	40	192	N
15909	0	0	0	15.3	0	0	0	0	-12.6	2.7	INFARCTION	15909	0	0	0	9	0	-12	-3	INFARCTION	INFARCTION	65	M	168	90	NO	NO	ALERT	302	197	26	110	60	180	N
8902	0	0	0	17	0	-4.1	-6.7	0	-12.6	-6.4	INFARCTION	8902	0	0	0	10	0	-12	-2	INFARCTION	INFARCTION	40	F	160	100	YES	YES	ALERT	134	189	46	117	27	88	IHD
16866	0	0	0	15.3	0	-4.1	0	0	-12.6	-1.4	INFARCTION	16866	0	0	0	11	-3	-12	-4	INFARCTION	INFARCTION	62	F	180	110	YES	NO	ALERT	295	257	23	173	59	91	LVH
17178	21.9	14.6	0	11.9	-3.7	0	0	0	-12.6	32.1	HEMORRHAGE	17178	5	0	0	7	-3	-12	-3	INFARCTION	INFARCTION	43	M	120	70	NO	YES	UNCONS	140	160	48	130	28	110	N
8461	0	0	0	15.3	-3.7	-4.1	0	0	-12.6	-5.1	INFARCTION	8461	0	0	0	9	-3	-12	-6	INFARCTION	INFARCTION	62	M	140	80	YES	YES	ALERT	150	180	62	130	28	162	LVH
17304	0	14.6	0	11.9	-3.7	-4.1	-6.7	0	-12.6	-0.6	INFARCTION	17304	5	0	0	9	-3	-12	-1	INFARCTION	INFARCTION	65	F	130	90	YES	YES	UNCONS	99	180	44	140	20	300	IHD
7114	21.9	7.3	0	17	0	0	0	0	-12.6	33.6	HEMORRHAGE	7114	2.5	0	0	10	0	-12	0.5	EQUIVOCAL	INFARCTION	55	M	180	90	YES	NO	DROWSY	156	180	62	130	28	162	IHD

ALLEN'S SCORE												SIRIRAJ'S SCORE									CT DIAGNOSIS	AGE	GENDER	SBP	DBP	HTN	ATHEROMA M	CONS.	LIPID PROFILE					BLD SUGAR	ECHO
HOSP NO.	ONSET	CONSC.	PLANTARS	DBP	ATHEROMA	HTN	PRE-EVENTS	HEART DISEASE	CONSTANT	SCORE	DIAGNOSIS	HOSP NO	CONS.	VOMITTING	HEADACHE	DBP	ATHEROMA	CONSTANT	SCORE	DIAGNOSIS								TG	TC	HDL	LDL	VLDL		
7107	0	7.3	0	15.3	0	0	0	-4.3	-12.6	5.7	INFARCTION	7107	2.5	0	0	9	-3	-12	-3.5	INFARCTION	INFARCTION	75	F	140	90	NO	YES	DROWSY	110	180	58	130	28	160	IHD
7564	21.9	0	7.1	18.02	0	-4.1	0	0	-12.6	30.32	HEMORRHAGE	7564	0	2	2	11	0	-12	3	HEMORRHAGE	HEMORRHAGE	56	F	210	110	YES	NO	ALERT	112	209	58	132	23	104	LVH
7060	21.9	14.6	7.1	15.3	0	-4.1	0	0	-12.6	42.2	HEMORRHAGE	7060	5	2	2	9	-3	-12	3	HEMORRHAGE	HEMORRHAGE	72	M	160	90	NO	NO	UNCONS	116	209	54	132	23	88	LVH
43677	21.9	14.6	7.1	17	-3.7	-4.1	0	0	-12.6	40.2	HEMORRHAGE	43677	5	2	2	10	-3	-12	4	HEMORRHAGE	HEMORRHAGE	59	F	180	100	YES	YES	UNCONS	134	165	48	88	32	187	N
43676	0	0	0	17	0	0	0	0	-12.6	4.4	INFARCTION	43676	0	2	0	7	0	-12	-3	INFARCTION	INFARCTION	80	M	110	70	NO	NO	ALERT	145	158	46	84	29	113	N
43052	0	0	0	22.1	0	0	0	0	-12.6	9.5	INFARCTION	43052	0	0	0	10	0	-12	-2	INFARCTION	INFARCTION	68	M	220	130	NO	NO	ALERT	123	165	76	64	25	81	N
43709	0	0	0	11.9	0	-4.1	-6.7	0	-12.6	-11.5	INFARCTION	43709	0	0	0	9	-3	-12	-6	INFARCTION	INFARCTION	65	F	180	90	YES	NO	ALERT	144	150	45	80	30	159	N
1796	21.9	14.6	7.1	17	-3.7	-4.1	-6.7	0	-12.6	33.5	HEMORRHAGE	1795	5	2	2	10	-3	-12	4	HEMORRHAGE	HEMORRHAGE	45	F	150	90	YES	YES	UNCONS	120	130	40	80	30	136	LVH
3360	21.9	14.6	7.1	15.3	0	0	0	0	-12.6	46.3	HEMORRHAGE	3360	5	2	2	9	0	-12	6	HEMORRHAGE	HEMORRHAGE	28	F	130	90	NO	NO	UNCONS	138	123	38	66	30	180	N
2497	21.9	0	7.1	15.3	0	0	0	0	-12.6	31.7	HEMORRHAGE	2497	2.5	2	2	9	0	-12	3.5	HEMORRHAGE	HEMORRHAGE	65	M	170	90	NO	NO	DROWSY	116	128	43	69	32	97	N
2491	0	7.3	0	15.3	-3.7	-4.1	0	0	-12.6	2.2	INFARCTION	2491	2.5	0	0	9	-3	-12	-3.5	INFARCTION	INFARCTION	50	F	136	90	YES	YES	DROWSY	136	124	32	65	27	158	LVH
3330	21.9	7.3	7.1	18.7	0	0	0	0	-12.6	42.4	HEMORRHAGE	3330	5	2	2	11	0	-12	8	HEMORRHAGE	HEMORRHAGE	45	M	230	110	NO	NO	UNCONS	150	118	46	89	44	204	N
364	21.9	14.6	7.1	17	0	0	0	0	-12.6	48	HEMORRHAGE	364	2.5	2	2	9	0	-12	3.5	HEMORRHAGE	HEMORRHAGE	50	M	160	100	NO	NO	DROWSY	110	132	42	98	45	201	N
14895	0	0	0	13.6	-3.7	0	0	-4.3	-12.6	-7	INFARCTION	14895	0	0	2	8	-3	-12	-5	INFARCTION	INFARCTION	82	F	140	80	NO	YES	ALERT	158	184	44	82	32	123	IHD
15764	0	0	0	15.3	0	0	0	0	-12.6	2.7	INFARCTION	15764	2.5	0	0	8	0	-12	-1.5	INFARCTION	INFARCTION	72	F	140	80	NO	NO	DROWSY	108	157	45	108	28	217	N
8983	0	0	0	18.7	0	-4.1	0	0	-12.6	2	INFARCTION	8983	0	0	0	10	-3	-12	-5	INFARCTION	INFARCTION	70	F	160	100	YES	NO	ALERT	98	203	43	140	20	155	LVH
15041	0	0	0	15.3	0	0	0	0	-12.6	2.7	INFARCTION	15041	0	2	0	8	0	-12	-2	INFARCTION	INFARCTION	35	M	140	80	NO	NO	ALERT	148	162	30	102	30	115	N
15032	0	7.3	0	15.3	0	0	0	0	-12.6	10	INFARCTION	15032	2.5	0	0	8	0	-12	-1.5	INFARCTION	INFARCTION	57	F	170	100	NO	YES	DROWSY	130	180	45	98	28	154	N
18971	0	0	0	11.9	0	0	0	0	-12.6	-0.7	INFARCTION	18971	0	0	0	7	0	-12	-5	INFARCTION	INFARCTION	40	M	110	70	NO	NO	ALERT	525	132	47	105	46	145	N

ALLEN'S SCORE												SIRIRAJ'S SCORE									CT DIAGNOSIS	AGE	GENDER	SBP	DBP	HTN	ATHEROMA M	CONS.	LIPID PROFILE					BLD SUGAR	ECHO
HOSP NO.	ONSET	CONSC.	PLANTARS	DBP	ATHEROMA	HTN	PRE-EVENTS	HEART DISEASE	CONSTANT	SCORE	DIAGNOSIS	HOSP NO	CONS.	VOMITTING	HEADACHE	DBP	ATHEROMA	CONSTANT	SCORE	DIAGNOSIS								TG	TC	HDL	LDL	VLDL		
30320	0	0	0	15.3	-3.7	-4.1	0	-4.3	-12.6	-9.4	INFARCTION	30320	2.5	0	0	9	-3	-12	-3.5	INFARCTION	INFARCTION	55	M	130	80	YES	YES	DROWSY	131	110	25	50	26	181	IHD
32427	0	0	0	13.6	0	0	-6.7	0	-12.6	-5.7	INFARCTION	32427	0	0	0	9	0	-12	-3	INFARCTION	INFARCTION	70	M	160	80	NO	YES	ALERT	150	165	38	110	43	154	N
30712	0	7.3	0	15.3	0	0	-6.7	0	-12.6	3.3	INFARCTION	30712	2.5	0	0	8	0	-12	-1.5	INFARCTION	INFARCTION	42	M	180	100	NO	YES	DROWSY	221	219	36	139	44	147	LVH
30577	0	0	0	17	0	0	0	0	-12.6	4.4	INFARCTION	30577	0	2	2	10	-3	-12	-1	INFARCTION	INFARCTION	60	M	170	100	NO	NO	ALERT	483	207	37	74	97	194	IHD
29895	21.9	14.6	7.1	17	0	0	0	0	-12.6	48	HEMORRHAGE	29895	5	2	0	10	0	-12	5	HEMORRHAGE	HEMORRHAGE	54	M	160	100	NO	NO	UNCONS	140	139	40	108	56	100	LVH
29227	0	0	0	15.3	0	0	0	0	-12.6	2.7	INFARCTION	29227	0	0	0	8	0	-12	-4	INFARCTION	INFARCTION	52	F	240	140	NO	NO	ALERT	182	179	35	108	36	101	N
17450	0	0	0	11.9	0	0	0	0	-12.6	-0.7	INFARCTION	17450	0	0	0	7	0	-12	-5	INFARCTION	INFARCTION	70	M	110	70	NO	NO	ALERT	109	140	22	96	22	139	N
32138	0	0	7.1	15.3	0	0	0	0	-12.6	9.8	INFARCTION	32138	0	0	2	7	0	-12	-3	INFARCTION	INFARCTION	80	F	110	80	NO	NO	ALERT	200	187	45	110	30	180	N
32038	0	7.3	0	13.6	0	0	0	0	-12.6	8.3	INFARCTION	32038	2.5	0	0	8	0	-12	-1.5	INFARCTION	INFARCTION	30	M	130	80	NO	NO	DROWSY	209	188	47	120	35	179	N
30332	0	7.3	0	11.9	0	0	0	0	-12.6	6.6	INFARCTION	30332	2.5	0	0	7	0	-12	-2.5	INFARCTION	INFARCTION	58	M	110	80	NO	NO	DROWSY	202	199	39	119	40	95	N
15787	21.9	7.3	7.1	17	-3.7	0	-6.7	0	-12.6	30.3	HEMORRHAGE	15787	2	2	0	13	0	-12	5	HEMORRHAGE	HEMORRHAGE	55	M	180	130	YES	YES	DROWSY	150	160	40	130	26	139	N
31768	0	0	0	15.3	-3.7	0	0	0	-12.6	-1	INFARCTION	31768	0	0	0	9	0	-12	-3	INFARCTION	INFARCTION	76	M	130	90	NO	YES	ALERT	112	169	43	110	23	198	LVH
14060	0	7.3	0	13.6	0	0	0	-4.3	-12.6	4	INFARCTION	14060	2.5	0	0	7	-3	-12	-5.5	INFARCTION	INFARCTION	27	F	110	70	NO	YES	DROWSY	110	190	46	114	26	110	RHD
13978	0	7.3	0	11.9	0	-4.1	-6.7	0	-12.6	-4.2	INFARCTION	13978	2.5	0	0	8	0	-12	-1.5	INFARCTION	INFARCTION	85	M	210	100	YES	YES	ALERT	116	209	54	132	23	74	N
13996	0	0	0	13.6	-3.7	-4.1	0	0	-12.6	-6.8	INFARCTION	13996	0	0	0	10	-3	-12	-5	INFARCTION	INFARCTION	61	M	160	100	YES	YES	ALERT	96	169	39	112	19	125	N
13183	0	7.3	0	13.6	-3.7	0	0	0	-12.6	4.6	INFARCTION	13183	2.5	0	0	8	-3	-12	-4.5	INFARCTION	INFARCTION	65	M	120	80	NO	YES	DROWSY	156	100	17	52	31	160	N
625	0	0	0	15.3	0	0	0	0	-12.6	2.7	INFARCTION	625	0	2	0	9	0	-12	-1	INFARCTION	INFARCTION	76	M	140	90	NO	NO	ALERT	156	123	28	51	34	148	N
7966	21.9	14.6	0	17	-3.7	-4.1	0	-4.3	-12.6	28.8	HEMORRHAGE	7966	5	2	0	11	-3	-12	3	HEMORRHAGE	HEMORRHAGE	86	M	210	110	NO	NO	UNCONS	74	184	41	128	15	344	MR
43968	0	0	0	17	0	-4.1	-6.7	0	-12.6	-6.4	INFARCTION	15499	0	0	0	9	0	-12	-3	INFARCTION	INFARCTION	60	M	140	90	YES	YES	ALERT	123	190	39	126	25	159	IHD