“IDENTIFICATION OF BEDSIDE CLINICAL SCORING SYSTEMS IN CLASSIFYING STROKE AND ITS SUBSEQUENT MANAGEMENT.”

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ENDORSEMENT

This is to certify that the dissertation entitled **"IDENTIFICATION OF BEDSIDE CLINICAL SCORING SYSTEMS IN CLASSIFYING STROKE AND ITS SUBSEQUENT MANAGEMENT"** is a bonafide research work done by **REG NO. BG0115003.**

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ABBREVIATIONS

ACA	-	anterior cerebral artery
AF	-	Atrial fibrillation
APLA	-	Anti phospholipid antibody syndrome
AUC	-	Area under Curve
BS	-	Besson score
CAA	-	Cerebral amyloid angiopathy
CAD	-	Coronary Artery Disease
CNS	-	Central nervous system
CT	-	computerized tomography
CTA	-	CT angiography
CVA	-	Cerebrovascular Accident.
DALY	-	Disability Adjusted Life-Year
ED	-	Emergency department
EIC	-	Early Ischemic Changes
GHSS	-	Guy's hospital score
GS	-	Greek score
HbA1c	-	Glycosylated hemoglobin
HDS	-	High density sign
ICH	-	intracranial hemorrhage
LAD	-	Left anterior descending artery
LDL	-	Low density lipoprotein
LVH	-	Left Ventricular Hypertrophy
MCA	-	Middle Cerebral Artery
MCV	-	Mean corpuscular volume

mg%	-	Milligram percentage
mg/dL	-	Milligrams per deciliter
MI	-	myocardial infarction
mL	-	Milliliter
mmHg	-	Millimeters of mercury
mmol/L	-	Millimole per litre
MRI	-	magnetic resonance imaging
mRS	-	Modified Rankin scale
PAN	-	Polyarteritis nodosa
PCA	-	Posterior cerebral artery
PE	-	Pulmonary embolism
PPBS	-	Post prandial blood sugar
RBS	-	Random blood sugar
ROC	-	Receiver operating characteristic
RtPA	-	Recombinant tissue plasmin activator
SAH	-	Subarachnoid hemorrhage
SBP	-	Systolic blood pressure
SS	-	Siriraj score
TIA	-	Transient Ischemic Attack
UK	-	United kingdom
WHO	-	World Health Organization

ABSTRACT

Background and objectives

Stroke is the third leading cause of premature death globally and is associated with up to 5.54 million deaths every year, two thirds of which occur in resource poor countries.[2] early identification of ischemic stroke is crucial as it leads to earlier treatment initiation with aspirin. Neuro-Imaging techniques have been valuable to distinguish between these Ischemic and hemorrhagic stroke. Quite unfortunately a large group of the population do not have access to these facilities, and even if accessible most of them find it unaffordable. To overcome these difficulties and to enhance clinical bedside diagnosis, clinical stroke scores have been developed. The most commonly used ones include the Besson score (BS), [13] the Greek stroke score (GSS) [14] and the Siriraj stroke score (SSS).[15]
The objectives of present study were to identify which of the scores have the best sensitivity and specificity to identify between ischemic and haemorrhagic stroke in our Indian population within our demographic setting.

Methods

The present cross sectional study was conducted on patients with Stroke admitted in KLES Dr Prabhakar Kore Hospital and Medical Research Centre, Belagavi from Jan 2016 to Dec 2016. Relevant data was collected by a detailed interview with patient or the attender, clinical examination, lab reports and neuro-imaging. The diagnosis of stroke was entertained after fulfilling WHO definition of stroke by the patient. They were then scored according to the scoring systems and validity tests of these scores were obtained by comparing it with neuroimaging. These findings were noted on a predesigned and pretested

proforma. Statistical test –Mann Whitney-U test and Chi-square tests also may be used for analysis.

Results

61 patients (42 patients (68.85%) ischemic stroke and 19 patients (31.15%) hemorrhagic stroke) were included in our study. For hemorrhagic stroke Greek score had the highest specificity (97.62%) while Siriraj score had the highest sensitivity (78.95%). For ischemic stroke Besson score had the highest specificity (94.74%) while Siriraj score had the highest sensitivity (80.95%). The Siriraj scoring system was better tool of scoring in identifying type of stroke in our study with the highest AUC (0.902) on the ROC curve.

Conclusion

In the present involving 61 patients with Stroke, on comparing different scoring systems (GS, SS & BS), we found SS was a better scoring system for both types of strokes. Although these scoring systems help in differentiating ischemic and hemorrhagic stroke on arrival of patients to the casualty, all have certain limitations. Hence we feel neuroimaging is the still the best in differentiating the type of stroke.

Keywords

Stroke; Siriraj; Besson; Greek; clinical score

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INTRODUCTION

A stroke, or cerebrovascular accident, is defined as abrupt onset of a neurologic deficit of vascular origin. World Health Organization defines the clinical syndrome of "stroke" as, rapidly developing clinical signs of focal (or global) disturbance of cerebral function with symptoms lasting 24 hours or longer or leading to death, with no apparent cause other than vascular origin.[1]

Stroke is the third leading cause of premature death globally and is associated with up to 5.54 million deaths every year, two thirds of which occur in resource poor countries.[2] According to the India stroke factsheet updated in 2012, the estimated age-adjusted prevalence rate for stroke ranges between 84/100,000 and 262/100,000 in rural and between 334/100,000 and 424/100,000 in urban areas.[3]

Thus, cerebrovascular disease is a huge public health problem imposing both as a large disease burden and a large economic burden on our country.[4]Incidence of stroke varies considerably from country to country. Based on the review of available information in India, the prevalence of stroke was estimated as 203 per 100,000 population above 20 years, amounting to a total of about 1 million cases.[5] The mortality rate of stroke in the acute phase is as high as 20% and it remains higher for several years after the acute event in stroke patients than in the general population.[6] Stroke is an illness of escalating socioeconomic importance, especially among the ageing population.

The poor are increasingly affected by stroke, because of both the changing population exposures to risk factors and not being able to bear the expense for stroke care. Majority of stroke survivors continue to live with disabilities, and the costs of

ongoing rehabilitation and long term-care are largely undertaken by family members, which impoverish their families.[7, 8]

Stroke is divided into two main subtype's ischemic or hemorrhagic stroke. Most strokes (87%) are ischemic strokes.[9]Atherosclerosis occurring in arteries supplying the brain; both large and small is the most common cause of ischemic stroke. Atherosclerosis occurring in proximal aorta is also a source of atherogenic brain emboli. Large artery atherosclerotic infarction occurs when there is an impediment to normal perfusion, usually caused by a severe arterial stenosis or occlusion due to atherosclerosis and coexisting thrombosis or artery to artery embolism. Microatheroma, lipohyalinosis, and other occlusive diseases of the small penetrating brain arteries are the most frequent causes of small, sub-cortical "lacunar" infarcts. About 20% of ischemic strokes are due to cardiogenic embolism, most commonly from atrial fibrillation. A variety of other occlusive disorders may be the primary cause or variably contribute to stroke pathogenesis.[10]Most cases of spontaneous intracranial hemorrhage (ICH) are attributed to hypertension or amyloid angiopathy. ICH occurs most frequently in the putamen (35 to 50%), followed by lobar (30%), thalamus (10 to 15%), pons (5 to 12%), caudate (7%), and the cerebellum (5%).[11]

Accordingly, stroke is also an important cause of morbidity and long term disability, up to 40% of survivors are not expected to recover their independence and self-care.[6]

Early identification of ischemic stroke is crucial as it leads to earlier treatment initiation with aspirin. Imaging techniques such as computerized tomography (CT) scan and magnetic resonance imaging (MRI) have been valuable in this regard to

distinguish between these subtypes. Quite unfortunately, in developing countries like ours, where a large group of the population is below poverty line and dwelling in rural areas, do not have access to these facilities, and even if accessible most of them find it unaffordable.

To overcome these difficulties and to enhance clinical bedside diagnosis, clinical stroke scores have been developed. The most commonly used ones include the Guy's hospital score (GHSS), [12] the Besson score (BS), [13] the Greek stroke score (GSS) [14] and the Siriraj stroke score (SSS).[15]

These scores can be potentially used identify the stroke subtypes. While these scores are not more accurate than neuro-imaging, they are simple, cheap and practical. However, their validity in the diagnosis of stroke in resource poor settings remains debatable.

This impelled us to identify clinical scoring systems in distinguishing between hemorrhagic and ischemic stroke.

OBJECTIVES

The objectives of present study were;

- To identify which of the scores have the best sensitivity and specificity to identify between ischemic and haemorrhagic stroke in our Indian population within our demographic setting.

REVIEW OF LITERATURE

STROKE

Historical perspectives

The term "Stroke" or "Cerebrovascular accidents" has come to signify the abrupt impairment of brain function due to a variety of pathological changes involving one (focal) or several (multifocal) intracranial or extracranial blood vessels.[16]

Edwin Smith Papyrus, an ancient Egyptian medical text, made the first historical reference to the nervous system back in 3500 B.C., when it described the brain and the fluids that covered the brain.

The concept of stroke was first noted by Hippocrates from 460 to 370 before the Common Era. During this period, the presentation of convulsions and paralysis were termed as apoplexy. Over the next several hundred years, scholars focused on physical symptoms and potential causes.

Hippocrates who took the word "Apoplexy" from common non-medical use where it meant **"**Astonished**,** Suddenly benefit of one's senses**"** and applied it descriptively to stroke.

- Jacob Werter, a Swiss physician was the first person to propose that apoplexy was caused by disease of the blood vessel in the brain.
- Thomas Willis illustrated the circle of wills in 1664 by cadaver experiments.
- Seddicot described spontaneous intracerebral hemorrhage in 1813.

- In 1828 Abberonbie, explained the obliterative arterial disease of cerebral arteries.
- Johan Friedrich crell, suggested the pultaceous or atheromatous elements in arterial lesions although he did not use the term atheroma.
- Von Haller made similar observations and applying the term “atheroma” to the arterial lesions.
- 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.
- 1877 Osler reported a case of Subarachnoid and intracerebral hemorrhage due to ruptured aneurysm.
- In 1914, Ramsay hunt was the first to describe comprehensive description of spontaneous carotid occlusion without crest disease of the intracranial vessel producing cerebral infarction.
- Dandy performed the first air ventriculogram.
- Denny brown introduced the concept of vascular insufficiency.
- Oldendorf developed the basis for computerized tomography (CT) in 1961 and the technique was applied to clinical diagnosis by an electrical engineer, Hounsfield, in 1973. This lead to a more precise categorization of ischemic and hemorrhagic CVA.
- Karim in 1978 used Platelet anti-aggregating drugs like aspirin for TIA’s.

- 1971 Raymond Damadian brought to notice that nuclear magnetic relaxation times of tumors and tissues differed encouraging scientists to use MRI to study disease.
- MRIs advancement significantly occurred in 2003, when the Nobel Prize was won by Paul C. Lauterbur and Peter Mansfield for their discoveries of using MRIs as a diagnostic tool.

  Within the last decade, the magnitude of research has grown exponentially.

The term apoplexy has waned, and the term stroke has become common place in the medical setting.[17]

Definition

WHO defines "stroke as rapid development of clinical signs of focal (or global) brain function disorders, with symptoms which last 24 hours or longer or lead to death, without other clear cause, except signs of blood vessel damage".[18]

Prevalence

Worldwide

It is projected that nearly 4 million people suffer from stroke yearly; of which about 570,000 cases occur in Europe and about 500,000 in United States of America. Global epidemiological trials show that rates grow exponentially with age, from 0.3‰ in the third and fourth decade of life, all the way to 30‰ in the eighth and ninth decade of life, which makes an average of 1–2%. Recent information demonstrates that the incidence of stroke in France is 114 cases for each 100,000 people per year, in Germany 350, in Italy 223, in Spain 141– 220, and in UK 161.[19,20]

Although rates of stroke mortality and burden vary considerably among countries, low-income countries are the most severely affected. There has been a 42% decrease in stroke incidence in high-income countries and >100% increase in low- to middle-income countries.[21, 22]

Morbidity and Mortality [23]

- 400-800 strokes per 100,000.
- 5.7 million Deaths.
- 16 million new acute strokes annually.
- 28,500,000 DALYs (disability adjusted life-year).
- 28-30 day case fatality ranges between 17%-35%.

Stroke in India

Estimates of the prevalence of stroke in India range from 44 to 843 per 100,000 population.[24,25]Based on the estimates by Dalal et al. in 2008, age adjusted annual incidence per 100,000 population is 152.[26]

Morbidity and Mortality [23]

- Prevalence 90-222 per 100,000.
- 102,620 million deaths.
- 1.44-1.64 million cases of new acute strokes every year.
- 6,398,000 DALYs.
- 12% of strokes occur in the population aged <40 years
- 28-30 day case fatality ranges from 18-41%.

Well-designed population based study was done in Vellore, South India that was conducted in two phases. In the first phase, a total urban and rural population of 258,576 was evaluated. After examination by neurologist, 147 cases of hemiplegia were detected. The prevalence rate of hemiplegia was calculated as 56.9 per 100,000 populations (68.5 in male and 44.8 in female). The prevalence rate was more in urban area than in rural area and increased with age. Subsequently during the second phase, the population was kept under surveillance for two years, prevalence and annual incidence rate of 84 and 13 respectively per 100,000 populations was reported.[27]

Cerebrovascular disease constituted 0.9 to 4.5% of total medical admissions. Ischemic stroke from thrombosis and embolism constituted between 57.3% and 82.7% of all strokes. The incidence of hemorrhagic stroke was between 13.6% and 37.9% of total stroke cases. Most of these studies were retrospective.[28]

The Kolkata study[29] demonstrated that the basal ganglia-thalamic region was the commonest site (75%) of hemorrhage. On the contrary, the sub cortical region was the commonest site of infarction (75.6%). This tendency for sub cortical infarct also is common in other Asian races. A study based on noninvasive tests to determine subtypes of ischemic stroke from a hospital-based registry of Southern India has attributed "41% of strokes to large artery atherosclerosis, 18% to lacunar causes, 10% to cardio embolic causes, and 4% to causes such as Takayasu syndrome, Moya Moya disease, carotid dissection, hyperhomocysteinemia, anticardiolipin antibody, and protein S deficiency".[30] The rest 27% of the cases of ischemic stroke were of undetermined origin. Among cardioembolic stroke, rheumatic heart disease (29%) and ischemic heart disease (27%) are predominant causes.[30]

In India, the pooled data incorporating all the studies reveal that ischemic stroke occurs in 68-80% and hemorrhagic stroke in 20-32%. Ischemic stroke comprise large vessel (41%), lacunar (18%), cardioembolic (10%), other determined (10%), and undetermined (20%) subtypes. The extracranial carotid disease is the etiological factor in 25-26% and intracranial carotid disease in 30% of ischemic stroke cases.[31]

Classification of stroke[32]

Broadly, strokes are classified as either hemorrhagic or ischemic. Acute ischemic stroke refers to stroke caused by thrombosis or embolism and is more common than hemorrhagic stroke.[33]

There are many classifications according to etiology vascular territory and by time course etc.

1. Classification by time course:

    a) Transient ischemic attack.
    b) Reversible ischemic neurological deficit.
    c) Stroke in evolution.
    d) Completed stroke.

2. by arterial territory:

    a) Internal carotid artery territory.
    b) Vertebrobasilar territory.
    c) Lenticulo-striate.

3. by underlying pathology:

   a) Atheromatous occlusion of vessels.
   b) Atheroembolism.
   c) Lipohylinoid necrosis.
   d) Charcot Bouchard aneurysm rupture.

4. According to cause: [30]

   a) Atherosclerosis.
   b) Embolism of cardiac origin.
   c) Vasculitis: Primary CNS, PAN, Collagen Vascular Disease, temporal arteritis, infectious vasculitis.
   d) Hematological Disorders: Hemoglobinopathies, hyperviscosity syndrome, hypercoagulability states, protein C and S deficiency, APLA syndrome.
   e) Drugs: Cocaine, alcohol, amphetamines, OC pills.
   f) Others: Moya Moya, migraine, fibromuscular dysplasia.
   g) Cerebral Venous Thrombosis.
   h) Intracerebral hemorrhage.

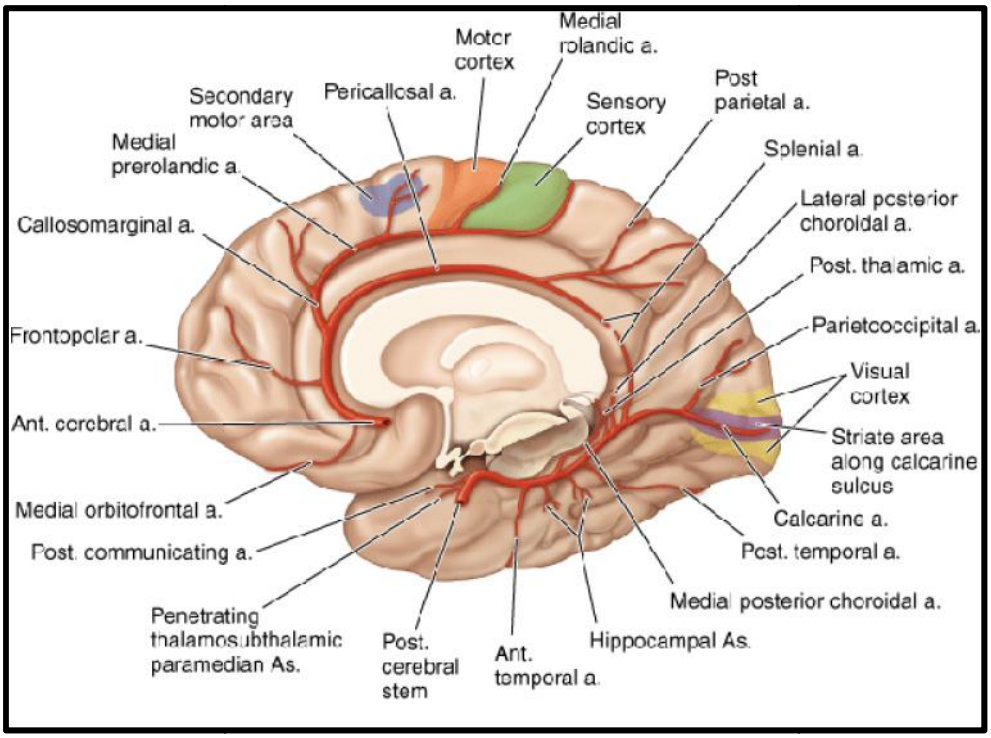


Figure 1. Cerebral hemisphere showing medial aspect - Branches of ACA[34]

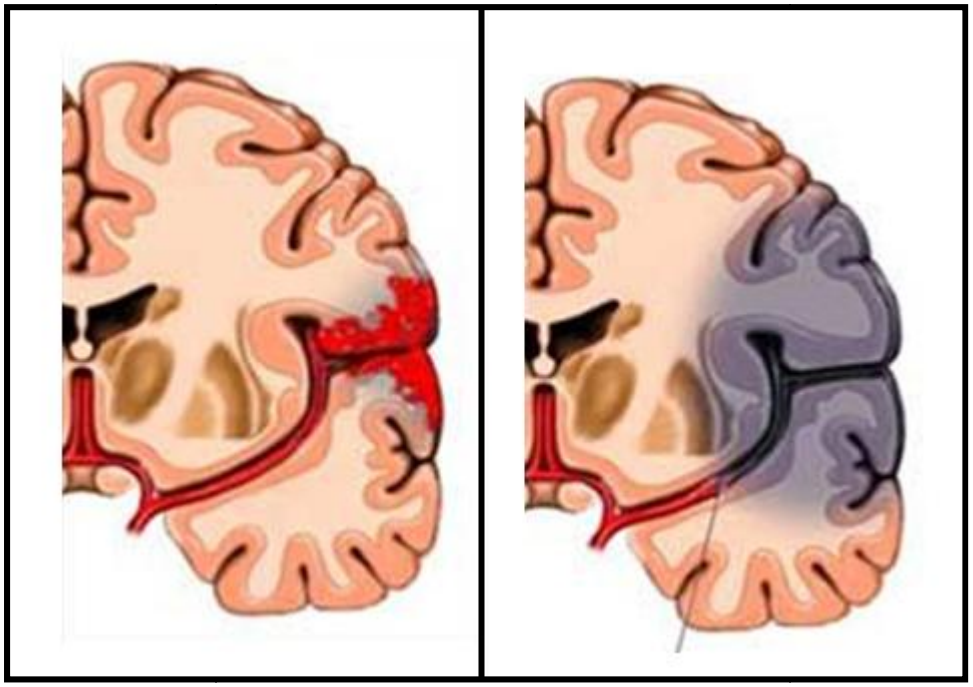


Figure 2. Pathology of hemorrhagic and ischemic stroke[35]

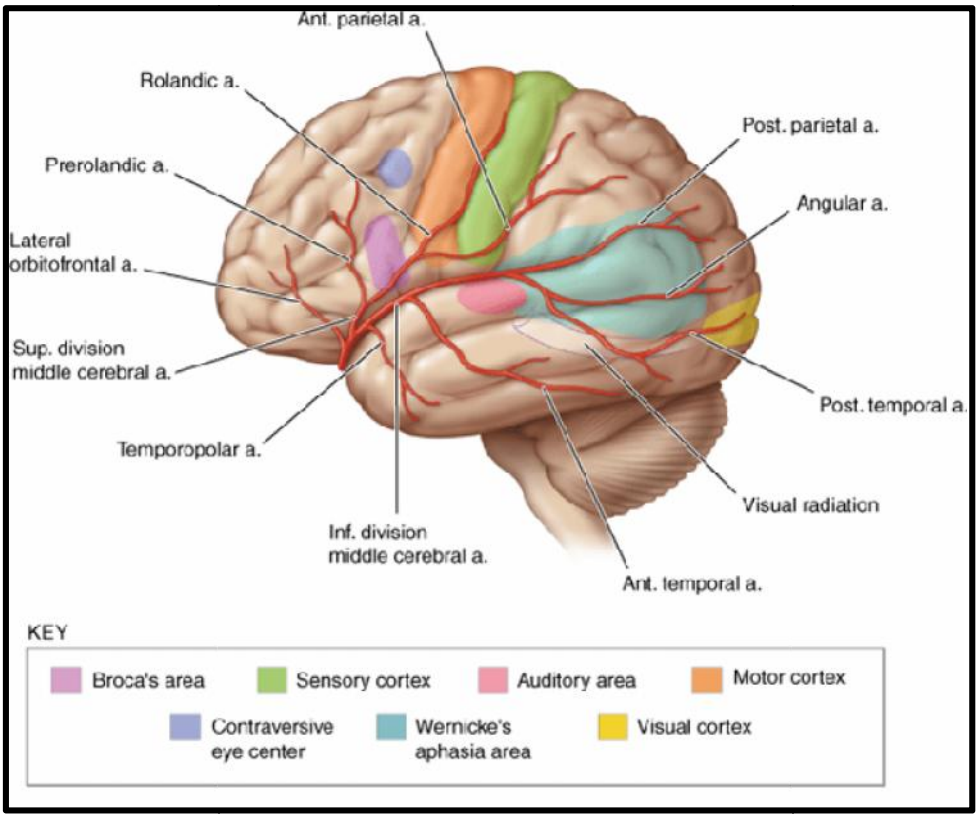


Figure 3. Cerebral hemisphere showing lateral aspect with branches of middle cerebral artery

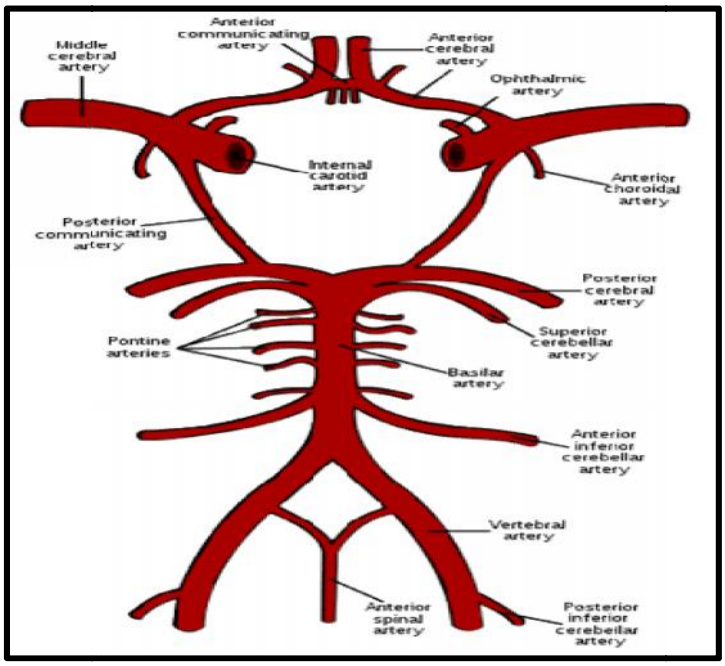


Figure 4. Schematic representation of the circle of Willis, arteries of the brain, and brainstem[35]

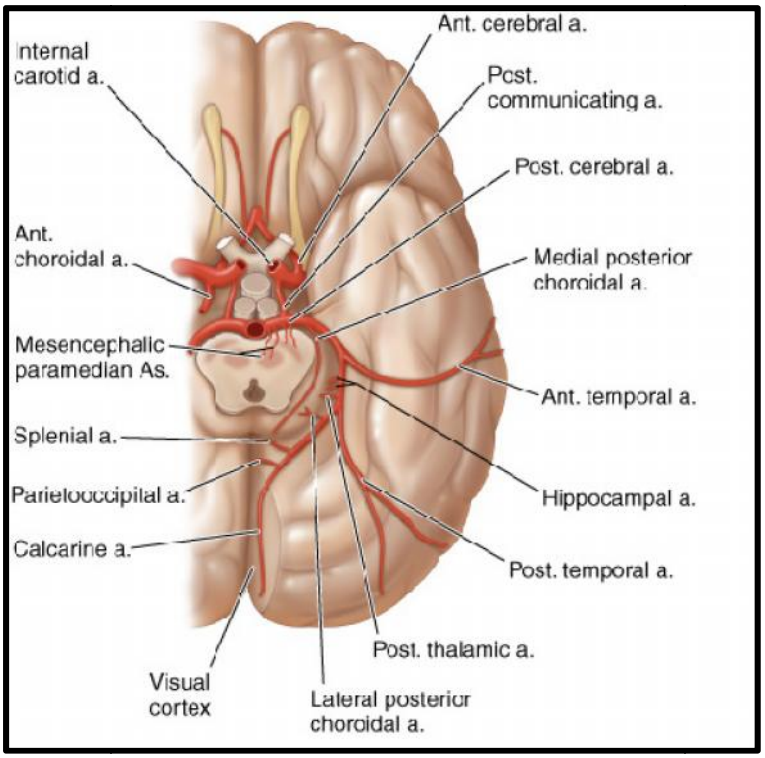


Figure 5. Inferior aspect of the brain with branches of posterior cerebral artery[34]

ISCHEMIC STROKE

Thrombotic cerebral infarction results from the atherosclerotic obstruction of large cervical and cerebral arteries, with ischemia in all or part of the territory of the occluded artery. This can be due to occlusion at the site of the main atherosclerotic lesion or to embolism from this site to more distal cerebral arteries.

Embolic cerebral infarction is due to embolism of a clot in the cerebral arteries coming from other parts of the arterial system, for example, from cardiac lesions, either at the site of the valves or of the heart cardiac cavities, or due to rhythm disturbances with stasis of the blood, which allows clotting within the heart as seen in atrial fibrillation.

Lacunar cerebral infarctions are small deep infarcts in the territory of small penetrating arteries, due to a local disease of these vessels, mainly related to chronic

hypertension. Several other causes of cerebral infarction exist and are of great practical importance for patient management. As they are relatively rare they can be ignored for most epidemiological purposes.

In India, frequency of ischemic stroke is between 60 to 80%.[26, 29, 31, 36]Further, lacunar, large vessel and cardioembolic types occur at 18%, 41%, 10%respectively[38] while other determined and undetermined types occur in 10% and 20 respectively.[36]

Etiology[37]

Ischemic strokes result from events that limit or stop blood flow, such as extracranial or intracranial thrombo-embolism, thrombosis in situ or relative hypoperfusion. As blood flow decreases, neurons cease functioning, and irreversible neuronal ischemia and injury begin at blood flow rates of less than 18 mL/100 g of tissue/min.

Pathophysiology

When an ischemic stroke occurs, the blood supply to the brain is interrupted, and brain cells are deprived of the glucose and oxygen they need to function. Ischemic stroke is a complex entity with multiple etiologies and variable clinical manifestations. Nearly45% of ischemic strokes are caused by small or large artery thrombus, 20% are embolic in origin, and others have an unknown cause.[38]

Thrombosis can form in the extracranial and intracranial arteries when the intima is roughened and plaque forms along the injured vessel. The endothelial injury (roughing) permits platelets to adhere and aggregate, then coagulation is activated and thrombus develops at site of plaque. Blood flow through the extracranial and intracranial systems decreases, and the collateral circulation maintains function. When

the compensatory mechanism of collateral circulation fails, perfusion is compromised, leading to decreased perfusion and cell death.[38]

During an embolic stroke, a clot travels from a distant source and lodges in cerebral vessels. Micro emboli can break away from a sclerosed plaque in the carotid artery or from cardiac sources such as atrial fibrillation, patent foramen ovale, or a hypokinetic left ventricle. Emboli in the form of blood, fat, or air can occur during surgical procedures, most commonly during cardiac surgery, but also after long bone surgeries.[38]

Less common causes of ischemic stroke include carotid dissection and the presence of coagulopathies, such as those resulting from antiphospholipid antibodies. Other causes include arteritis, infection, and drug abuse, such as the use of cocaine. While still not completely understood, the presence of periodontal disease and tooth loss is also an associated risk for ischemic stroke.[38]

As a thrombosis or emboli cause a decrease in blood supply to the brain tissue, events occur at the cellular level, referred to as the ischemic cascade. Neurons and support cells require a careful balance of variables such as temperature, pH, nutrition, and waste removal in their environment to function optimally. Intensive basic scientific research during the last two decades has given healthcare professionals an increased understanding of the ischemic cascade in the format of the precise environmental alterations involved in the pathophysiology of ischemic injury at the cellular level. Understanding the ischemic cascade has led to the concept of a therapeutic time window for treatment possibilities. Often, there is a core region of dead cells surrounded by an area of hypo perfused tissue. The hypo perfused area may be rescued; this area is referred to as the penumbra region.[38]

Neuroprotection is a broad term that refers to pharmacological and non pharmacological treatments used to halt the cellular events in the ischemic cascade, forming the theoretical basis for many of the acute stroke therapies under study as well as the rationale for intervening within a therapeutic time window following ischemic stroke.[37]

Risk factors of ischemic stroke[37]

A risk factor is a characteristic of an individual or population associated with increased risk of disease compared to those without it. Multiple risk factors are associated with cerebral infraction and have been studied in great detail. Risk factor profile for ischemic stroke differs and is variable in young and elderly patients. Various pro-atherothrombotic processes leading to macrovascular complications are well known. Diabetes mellitus, hypertension, smoking, alcoholism and dyslipidemia are some of the prominent modifiable risk factors for atherothrombotic ischemic stroke. Other risk factors of stroke are raised homocysteine, obesity, inadequate physical activity, migraine, oral contraceptives and hormonal supplements, fibrinogen and clotting factors, vasculitis, collagen vascular diseases and cardiac disorders to name a few. Age, gender, ethnic and geographical background, genetic inheritance and familial predisposition are some of the non-modifiable risk factors of ischemic stroke. In spite of the adequate control of these conventional risk factors, the incidence of cerebral infarction is not curbed, emphasizing a need to look into novel and unrecognized risk factors. Risk factors for ischemic stroke include modifiable and non-modifiable etiologies. Identification of risk factors in each patient can uncover clues to the cause of the stroke and the most appropriate treatment and secondary prevention plan. Non-modifiable risk factors include the following:

- Age
- Race
- Sex
- Ethnicity
- History of migraine headaches
- Sickle cell disease
- Fibromuscular dysplasia
- Heredity

Modifiable risk factors include the following:

- Hypertension
- Diabetes mellitus
- Cardiac disease - Atrial fibrillation, valvular disease, mitral stenosis, and structural anomalies allowing right to left shunting, such as a patent foramen ovale and atrial and ventricular enlargement
- Hypercholesterolemia
- Transient ischemic attacks (TIAs)
- Carotid artery stenosis
- Hyperhomocysteinemia
- Lifestyle issues - Excessive alcohol intake, tobacco use, illicit drug use, obesity, physical inactivity
- Oral contraceptive use

Among the types of cardiac disease that increase stroke risk are atrial fibrillation, valvular disease, mitral stenosis, and structural anomalies allowing right-

to-left shunting, such as a patent foramen ovale and atrial and ventricular enlargement.

Cerebral Hemorrhage

Intracranial bleed could be subarachnoid hemorrhage, intraparenchymal hemorrhage or intraventricular hemorrhage. Seventy to ninety percent of spontaneous ICHs are associated with hypertension.[40] Analysis of eleven separate series of hypertensive ICH revealed that 64% occur in basal ganglia, 13% in thalamus, 16% in hemispheric white matter, 10 to 12% in pons and 8 to 10% in cerebellum.[40]

Risk factors in cerebral hemorrhage

Age and race

Age is the greatest risk factor for ICH. Incidence rates increase dramatically among persons older than 60.

Hypertension

Hypertension is the most important and prevalent modifiable risk factor for ICH. In the biracial population of Greater Cincinnati during 1988, the presence of hypertension among patients with ICH was remarkably similar for whites (73%), African-Americans (71%), men (72%), and women (73%). Untreated hypertension is a greater risk factor than treated hypertension, and hypertensive patients who discontinue their medications have greater risk than those who continue them. Among modifiable risk factors for ICH, hypertension accounts for the greatest attributable risk for hemorrhage. The relative effect of hypertension as a risk factor for ICH is greater in younger patients than the elderly.[41]

Cerebral amyloid angiopathy

Once thought to be a rare cause of ICH, cerebral amyloid angiopathy (CAA) is now considered an important cause of lobar hemorrhage in the elderly. Its principal pathological feature is the deposition of amyloid protein in the media and adventitia of leptomeningeal arteries, arterioles, capillaries, and, less often, veins.[41]

Apolipoprotein E and CAA

Several studies have examined the relationship of Apolipoprotein E E2 and E4 with lobar ICH and CAA.

Aneurysms and vascular malformations

Although ruptured berry aneurysms typically cause SAH, on occasion bleeding is directed into the brain parenchyma without significant subarachnoid extension.[41]

Anticoagulant-and thrombolytic associated ICH

The use of warfarin for prevention of ischemic stroke among patients with atrial fibrillation increased significantly during the late 1980s and 1990s following publication of the Stroke Prevention in Atrial Fibrillation (SPAF) trials, European Atrial Fibrillation Trial, and other important studies on this topic. Several trials have tested warfarin for secondary stroke prevention in patients with cerebral ischemia of non-cardiac origin. The Warfarin-Aspirin Recurrent Stroke Study (WARSS) compared aspirin to warfarin (goal INR 1.4–2.8), and found no difference between groups in effectiveness or risk of major hemorrhage (including ICH).[41]

Antiplatelet drugs

Antiplatelet drugs probably increase the risk of ICH by a small amount. The absolute risk of intracranial hemorrhage among elderly persons taking aspirin has been estimated at 0.2–0.3% annually (vs. 0.15% in similar persons not taking anti platelet or anticoagulants).[41]

Cerebral microbleeds

The use of gradient echo MRI to detect small, asymptomatic hemorrhages in the brain parenchyma ("microbleeds") has received considerable recent attention. Gradient echo MRI accentuates signal dropout from chronic blood products and is more sensitive at detecting small hemorrhages than standard T2 sequences.[41]

Prior cerebral infarction

Prior cerebral infarction is associated with a 5- to 22-fold increased risk of ICH. The strong relationship between ICH and cerebral infarction is not surprising since hemorrhage and infarction share similar risk factors, such as hypertension. In the GERFHS case-control study in Greater Cincinnati 15% of ICH patients had a history of previous ischemic stroke; the multivariate odds ratio for ICH in patients with prior stroke compared to controls was 7.0.[41]

Heavy alcohol use

Numerous studies have identified a relationship between alcohol use and the risk of hemorrhagic stroke.[41]

Tobacco use

There may be a weak association between tobacco use and ICH but data have been conflicting. It is suggested that current smoking (as opposed to past smoking or never smoking) increases the risk of ICH in a dose-dependent manner.[41]

Diabetes mellitus

Diabetes is associated with greater risk of ICH in some case-control studies. A review of available data produced an overall risk ratio of 1.3 with borderline statistical significance. The association of diabetes and ICH may vary by age group and location of hemorrhage.[41]

Heritability

There is a genetic component to ICH risk but its absolute value is small. Among probands in the GERFHS case-control study, [35] 6% of patients had an affected first-degree relative and 6% an affected second-degree relative. Among cases the odds ratio for an affected first-degree relative was high (6.3) but the population attributable risk was low (0.05).[41]

Serum cholesterol

In prospective studies, hemorrhagic stroke has been found to occur at higher rates in persons with low levels of blood total cholesterol than in persons with higher levels.[36, 37] This finding was first noted following World War II among rural Japanese, who had very low serum cholesterol levels by Western standards (less than 160 mg/dL) and also had a marked increase in incidence of ICH.[42-43]

Drugs

Intracranial hemorrhage secondary to cocaine[42] and amphetamine[45, 46] addiction is reported in literature. Drug abuse is an important consideration in non-traumatic parenchymal ICH, representing approximately 0.5% of overall ICH, but a higher percentage of ICH is seen in adolescents and young adults.[47, 48]

History and clinical presentation

Assessment of the patient with a stroke begins with recognition of the event as a stroke in the prehospital phase of care and continues throughout care. Emergency medical technicians and ambulance staff members need training in the recognition of signs and symptoms of stroke. Tools such as the Face Arm Speech Test[42] and the shortened National Institutes of Health Stroke Scale[43] have been tested and found to be effective in increasing the diagnostic accuracy of ambulance staff. The National Association of EMS Physicians has published standards for acute stroke prehospital care[44] which the AHA did not seek to duplicate but continued to emphasize the need for immediate diagnosis and evaluation.[49]

In the emergency department (ED), as the patient arrives, preferably by ambulance, a suspected stroke is treated as an acute event until diagnostic evidence suggests otherwise. Neurological assessment is based on both subjective and objective data, and a careful medical history is crucial to establish the exact time of onset of stroke signs and symptoms. Essential data to include are a quick history of timing of the event, pertinent past medical history, and risk factors. The full NIHSS can be used to guide the neurologic assessment.[49]

NIH Stroke Scale

	Category	**Description**	**Score**
1a	level of consciousness (LOC)	Alert	0
		Drowsy	1
		Stuporous	2
		Coma	3
1b	LOC questions (month, age)	Answers both correctly	0
		Answers 1 correctly	1
		Incorrect on both	2
1c	Answers both correctly Answers 1 correctly Incorrect on both	Obeys both correctly	0
		Obeys 1 correctly	1
		Incorrect on both	2
2	Best gaze (follow finger)	Normal	0
		Partial gaze palsy	1
		Forced deviation	2
3	Best visual (visual fields)	No visual loss	0
		Partial hemianopia	1
		Complete hemianopia	2
		Bilateral hemianopia	3
4	Facial palsy (show teeth, raise brows, squeeze eyes shut)	Normal Minor	0
		Partial Complete	1
5	Motor arm left* (raise 90°, hold 10 seconds)	No drift	0
		Drift	1
		Cannot resist gravity	2
		No effort against gravity	3
		No movement	4

6	Motor arm right* (raise 90°, hold 10 seconds)	No drift	0
		Drift	1
		Cannot resist gravity	2
		No effort against gravity	3
		No movement	4
7	Motor leg left* (raise 30°, hold 5 seconds)	No drift	0
		Drift	1
		Cannot resist gravity	2
		No effort against gravity	3
		No movement	4
8	Motor leg right* (raise 30°, hold 5 seconds)	No drift	0
		Drift	1
		Cannot resist gravity	2
		No effort against gravity	3
		No movement	4
9	Limb ataxia (finger-nose, heel-shin)	Absent	0
		Present in 1 limb	1
		Present in 2 limbs	2
10	Sensory (pinprick to face, arm, leg)	Normal	0
		Partial loss	1
		Severe loss	2
11	Extinction/neglect (double simultaneous testing)	No neglect	0
		Partial neglect	1
		Complete neglect	2
12	Dysarthria (speech clarity to "mama, baseball, huckleberry, tip-top, fifty-fifty")	Normal articulation	0
		Mild to moderate dysarthria	1

		Near to unintelligible or worse	2
13	Best language** (name items, describe pictures)	No aphasia	0
		Mild to moderate aphasia	1
		Severe aphasia	2
		Mute	3
	Total		0-42

* For limbs with amputation, joint fusion, etc., score 9 and explain. ** For intubation or other physical barriers to speech, score 9 and explain. Do not add 9 to the total score.

Symptoms of Ischemic Stroke According to Cerebral Circulation[50]

Brainstem

- Hemiparesis or quadriparesis
- Motor or sensory loss in all four limbs
- Eye movement abnormalities, such as diplopia and dysconjugate gaze
- Oropharyngeal weakness
- Vertigo, tinnitus
- Nausea, vomiting
- Dysmetria

Cerebellum

- Ipsilateral limb ataxia
- Gait ataxia

Vertebrobasilar circulation

- Symptoms correlate with brainstem and cerebellar functions as above
- Cranial nerve deficits in cranial nerves III – XII

Anterior Circulation Symptoms

- Carotid artery
- Contralateral motor and sensory loss
- Amaurosis fugax or Transient monocular blindness(caused by emboli to retinal artery)

Anterior Cerebral Artery

- Confusion
- Personality change
- Incontinence
- Contralateral motor or sensory loss in leg greater than arm

Middle Cerebral Artery

- Contralateral motor or sensory loss (arm greater than leg)
- Contralateral motor loss in lower face
- Contralateral visual field loss
- Language deficit (dominant hemisphere)
- Spatial-perceptual deficit (non dominant hemisphere)

Posterior Cerebral Artery

- Contralateral sensory loss
- Ipsilateral visual field deficit
- Cortical blindness

In addition to these symptoms, determining dominance is important as the dominant hemisphere is primarily responsible for language function. Handedness determines dominance for most people. Right-handed people are left-hemisphere dominant; left-handed people are also left-hemisphere dominant about 60% of time. The clinical features that are more common with a dominant left cerebral hemisphere lesion include aphasia, agraphia, acalculia, apraxia, a left gaze preference, a right visual field deficit along with right-sided hemiparesis, and a right-sided hemisensory loss. Common features of a non dominant right cerebral hemisphere include neglect (left-sided hemi inattention), right gaze preference, left visual field deficit, dysarthria, flat affect, left-sided hemiparesis, and left-sided hemisensory loss.[50]

The presence of a transient ischemic attack or other conditions needs to be ruled out to ensure that patients receive the appropriate treatment for their condition. A wide range of abnormalities can mimic a stroke, including hypoglycemia, migraine, seizure, and trauma.[50]

Clinical features of hemorrhagic stroke

The clinical features used to define ICH were presentation with a gradual progression (over minutes or days) or sudden onset of focal neurological deficit, usually accompanied by signs of increased intracranial pressure such as vomiting or diminished consciousness. As many as 91% of patients were hypertensive (blood pressure 160/100mmHg or higher) at the onset of their stroke.[21]

Vomiting was far more common in ICH and SAH (51% and 47% respectively) than for ischemic stroke (4–10% of cases). While SAH presented with headache at onset in 78% of cases, 33% of cases of ICH also had a headache at onset compared to

3–12% of ischemic stroke subtypes. Finally, SAH and ICH both presented with coma in 24% of cases compared to 0–4% of ischemic stroke subtypes.

A particular characteristic of ICH was the smooth or gradual progression of stroke in 63% of cases, with sudden onset in 34% of cases. A smooth or gradual onset of stroke was seen in only 5–20% of ischemic stroke subtypes and 14%ofSAH.Thus, ICH is the stroke subtype most likely to worsen significantly in the first 24 hours.[41]

Differentiation of thrombosis, embolism and hemorrhage

It is difficult to frame definite rules to differentiate cerebral hemorrhage, ischemic and embolic infarction. However with history and detailed clinical examination majority of the cases can be diagnosed at the bed side.

Clinical presentation of symptoms by subtype[41]

	Thrombosis	**Lacunar**	**Embolus**	**ICH**	**SAH**
Maximal at onset	**40%**	**38%**	**79%**	**34%**	**80%**
Stepwise	**34%**	**32%**	**11%**	**3%**	**3%**
Gradual	**13%**	**20%**	**5%**	**63%**	**14%**
Fluctuating	**13%**	**10%**	**5%**	**0%**	**3%**

a. **CLINICAL**

b.

Cerebral thrombosis

The important diagnostic criteria of atherothrombotic infarction are:

- History of prodromal Transient ischemic attacks (TIAs).
- Intermittent stepwise evolution of neurologic deficit with recovery and improvement between worsening rather than steady progression.
- Relative preservation of consciousness unless upper part of basilar territory is involved.
- Onset during sleep.
- On arising or during a period of hypotension, evidence of atherosclerosis.
- Normal Cerebrospinal fluid (CSF).

However, TIAs are seen in only 30% of cases, consciousness may be impaired in massive cerebral infarction, CSF will be blood stained in hemorrhagic infarction.

Cerebral embolism

The clinical syndrome is characterized by abrupt development of completed stroke within few seconds. Transient ischemic attacks are uncommon. Presence of atrial fibrillation, myocardial infarction, and endocarditis favor the diagnosis of embolism. Evidence of recent embolism in other organs, rapid improvement from stroke, relative preservation of consciousness favors the diagnosis.

Cerebral hemorrhage

Presence of hypertension (70 to 80% of patients), frequent occurrence of headache, absence of prodromal phenomena, rapid development of neurologic deficit over a period of minutes to hours, onset during waking hours, deepening stupor or coma, nuchal rigidity except when in deep coma, suggest the diagnosis of ICH.

In a series of 244 cases of proven ICH it was possible to identify four major presenting groups.[73]

1. Sudden onset without loss of consciousness (89 cases)
2. Sudden onset with loss of consciousness (117 cases)
3. Gradual onset without loss of consciousness (23 cases)
4. Gradual onset with later loss of consciousness (4 cases)

The exact onset of remaining 12 cases was unknown. Thus, in about 50% cases patients did not lose consciousness at or within 24 hours of ictus. Severe headache was a feature of only 50% cases and vomiting was always universal.[74]

Subarachnoid hemorrhage

Typical presentation of subarachnoid hemorrhage is sudden severe headache, widespread or predominant on one side or the posterior part of the head and stiff neck. There are no cerebral symptoms or signs in pure SAH. However, localizing findings occur in ICH or cerebral infarction secondary to SAH.

Conditions that Mimic Ischemic Stroke[50]

- Unrecognized seizures
- Confusional states
- Syncope
- Toxic or metabolic disorders including, but not limited to the following
    - Hypoglycemia
    - Drug overdose
    - Hyponatremia
    - Migraine
    - Concussion with head injury
    - Encephalopathies or encephalitis
    - Eclampsia
    - Brain tumors
    - Subdural hematoma

Diagnosis

Diagnostic studies help to confirm stroke, detect early potentially life-threatening complications, and direct specific care given; those recommended in the AHA guidelines are shown in Figure. These diagnostic tests are available in most EDs 24 hours a day. Blood glucose can be checked in the ambulance with a finger stick or upon ED arrival and is helpful in ruling out hypoglycemia as a cause for the event or hyperglycemia as a compounding factor.[40] A computed tomography (CT) scan without contrast is recommended to rule out the presence of a hemorrhagic stroke that would preclude the use of thrombolysis.[47] Adjunct studies may include a CT angiogram, magnetic resonance imaging (MRI), and cerebral angiography. A CT

angiogram can be used to identify large vessel stenosis or occlusion. MRI allows for better visualization of possible infarcted areas and angiography is used when intra-arterial (IA) thrombolysis is indicated or when surgical interventions are being considered.[50]

Recommended Tests in Evaluation of Acute Stroke[50]

All Patients

- CT of the brain without contrast
- MRI can be considered at qualified centers
- Electrocardiogram
- Complete blood count with platelet count
- Serum electrolytes
- Blood glucose
- Prothrombin time, activated partial thromboplastin time, and international normalized ratio
- Renal function tests
- Oxygen saturation

Selected Patients

- Chest X ray
- Hepatic function tests
- Arterial blood gas levels (if hypoxia suspected)
- Markers of cardiac ischemia
- Lumbar puncture (if subarachnoid hemorrhage is suspected and CT is negative)

- Erythrocyte sedimentation rate (ESR), syphilis serology
- Lipid profile
- Toxicology screen
- Blood alcohol level
- 2D echocardiography
- Pregnancy test
- Electroencephalogram (when seizures suspected)

Acute stroke care organization[51]

Experience with the revascularization procedures emphasizes time is a primary factor for the prediction of clinical outcome. Organizing prehospitalization care is essential to minimize the delay of therapy initiation. Transport to the closest primary stroke center or comprehensive stroke center should be immediate and rapid. It may even involve air medical transport. Implementation of the guidelines for acute care organization shortens the course of procedures in the acute phase of stroke and may improve the patient's outcome.[52] In patients with moderate or severe clinical deficit (NIHSS > 8), performing of vascular intracerebral imaging is advocated to select the subjects with large-vessel occlusion. In such instances, it is recommended to transfer the patient to a center where it is feasible to carry out endovascular treatment. The secondary transport should not postpone the administration of fibrinolysis.

Principles for improving door-to-needle times.

Hospital notification by emergency medical system in advance[52]
Rapid triage protocol and activation of stroke team
Availability of stroke protocols
Rapid imaging and laboratory tests
Mixing rtPA when the patient is eligible and rapid access to intravenous administration
Data feedback and periodic data review by the whole stroke team

Initial acute clinical examination by a trained neurologist evaluates the level of consciousness and looks for focal neurologic signs. Assessment of the neurological-deficit degree using the National Institute of Health Stroke Scale (NIHSS) scale correlates with the site of arterial obliteration. Patients with occlusion of main intracerebral artery only very rarely present a deficit with NIHSS lower than 10 points. Reliability of the clinical diagnosis of vascular territory (carotid versus vertebrobasilar) is only moderate. Oculomotor and visual symptoms are the most sensitive indicators of posterior circulation stroke. Clinical examination is insensitive to distinguish between ischemic and hemorrhagic stroke, though the latter initially presents more frequently headache, signs of intracranial hypertension, and sudden impairment of consciousness.

Neuroimaging[51]

CT scan is, since the institution of thrombolytic reperfusion therapy, an integral component of acute stroke diagnostics. It is widely available, fast, and has excellent sensitivity for the detection of acute intracranial hemorrhage. Brain CT is also useful for the detection of early ischemic changes (EIC). These abnormities are displayed as discrete parenchymal hypo attenuation. The Alberta stroke program early CT score (ASPECTS) is a simple semi quantitative assessment of EIC.[53] Changes are screened in 10 regions of the middle cerebral artery territory (4 subcortical and 6 cortical). Extension of EIC in 4 and more regions represents an increased risk of hemorrhagic transformation and it is a contraindication for fibrinolytic therapy. Presence of the high-density sign (HDS) in the intracranial artery on the native CT scan has high predictive value for a thrombotic occlusion of the vessel. Its length inversely correlates with the efficacy of fibrinolytic recanalization. CT angiography (CTA) provides visualization of the cervical and cerebral arteries with high resolution. In patients with an acute ischemic stroke, CTA finds a thrombotic occlusion of intracerebral vessels in more than 60% of cases. CTA was used as a selection criterion in all recent studies that proved the benefit of mechanical thrombectomy in patients with acute occlusion of the main intracerebral artery.[54-57] CTA may also display collateral supply at the level of Willis′ circle arteries and the retrograde filling of the cortical arteries by leptomeningeal junctions. Capacity of leptomeningeal collaterals in the ischemic hemisphere can be evaluated in comparison with the cortical arteries in the opposite unaffected side. The development of arterial collaterals lowers the speed of infarction growth and increases the chance of a good clinical outcome with timely recanalization. More sophisticated methods for penumbra imaging include perfusion computed tomography or magnetic resonance diffusion- and perfusion-

weighted imaging. Yet, they have actually not become a routine part of acute stroke protocol in most centers because of time consumption, patient tolerance, and temporal accessibility. These techniques may be reserved for patients presenting later in the course of ischemic stroke.

Intensive care units ("stroke units") [51]

Intensive care in a stroke unit is supported as well as reperfusion therapy by class I evidence. The aggregate of measures helps to prevent complications and improve the functional outcome of the ischemic stroke patients.[57] Systematic continuous monitoring of oxygenation with a pulse oximetry is essential for acute stroke patients. Current recommendations support oxygen supplementation if SpO_2 falls below 94%. Intubation and mechanical ventilation may be demanded if the level of consciousness decreases (GCS<8) and in the case of respiratory insufficiency due to cerebral (brainstem ischemia) or extra cerebral causes (pneumonia, cardiac failure, etc.). Dysphagia, which represents an increased risk of aspiration, should be systematically checked for using the water swallow test or more accurate gugging swallowing screen (GUSS). Blood pressure should be monitored regularly. Extreme hypertension (BP>220/120) and hypotension are advised to be avoided, but the effect of acute BP lowering is not clear. Euvolemia should be maintained by the iso-osmotic infusions. Hyperglycemia is associated with a higher stroke volume and increased rate of the infectious complications. Although tight glycemic controls and maintaining the glucose level did not demonstrate a decrease of adverse events, it is preferred to maintain the serum glucose within a range of 8.0–10.0 mmol/l with an insulin regimen. Avoiding hyperpyrexia preferentially with paracetamol and treatment of infections protects the brain tissue from amplification of the ischemic cascade. For

instrumental neuromonitoring (measurement of intracranial pressure, near-infrared spectroscopy, micro dialysis, and transcranial ultrasound), there is an insufficient evidence in the acute stroke care. Patients with unexplained altered consciousness should undergo EEG examination. Anticonvulsive therapy (valproate or levetiracetam) is advocated in patients with acute onset of symptomatic epileptic seizures within the acute stroke period. Osmotic therapy (hypertonic saline solutions are more effective than mannitol) reduces edema in the ischemic zone, but its effect on the clinical outcome is less clear.

Intravenous thrombolysis[51]

Intravenous thrombolysis (IVT) by recombinant tissue plasmin activator (rtPA) has been recently approved for recanalization therapy in patients with acute ischemic stroke. In 1995, the NINDS study group reported that the patients receiving rtPA in a total dose of 0.9 mg/kg within 3 h after the onset of clinical symptoms were significantly less disabled at 3 months than those who received only a placebo.[58] Several subsequent trials failed to show efficacy of the thrombolytic therapy when administered within a longer therapeutic window of up to 6 h. The ECCAS 3 trial has been finally proved in 2008 and showed that the intravenous thrombolysis significantly improved clinical outcome in those patients who received this therapy between 3 and 4.5 h after onset of the clinical symptoms.[59] Inclusion and exclusion criteria have been revised recently. This important modification enables indicating this therapy in patients who are manifested by an inaugural epileptic seizure and in those who take a chronic anti-vitamin K therapy and have an insufficient anticoagulation level (INR<1.7). Yet, there are discrepancies in the European and American recommendations for thrombolysis given between 3 and 4.5 h. AHA/ASA

does not advise administering the therapy after 3 h in patients who fulfil one of these following criteria: (1) age > 80 years, (2) previous stroke, (3) anti-vitamin K therapy in course not respecting INR, and (4) diabetic retinopathy. During thrombolysis, the vital functions, especially blood pressure, must be monitored regularly and its value should be kept below 185/110. Functional status of the patients is normally evaluated at 90 days after the stroke onset. In spite of its beneficial effect a sudden reperfusion may be deleterious, leading to the disruption of the blood–brain barrier and hemorrhagic transformation of the ischemic tissue. Symptomatic intracerebral hemorrhage (sICH) occurs in 3–8% of patients who received the intravenous thrombolysis.[60]This is one of the reasons why thrombolytic therapy given for acute ischemic stroke did not significantly reduce mortality of patients. Risk factors for sICH are as follows: patients′ age, blood glucose level, degree of the initial neurological deficit, and violation of the procedure. Agents blocking the brain–brain barrier degradation and potentially reducing the hemorrhagic complications of the fibrinolysis are now under investigation. Extra cerebral hemorrhagic complications and allergic reaction are the other most frequent adverse events of the IVT. There is still insufficient evidence regarding the other fibrinolytic agents that were tested in the acute ischemic stroke patients. Promising results have been shown in a non-randomized trial with tenecteplase, which has a longer half-time, greater fibrin specificity, and a higher resistance to the inhibition by plasminogen activator inhibitor.

Inclusion and exclusion criteria of fibrinolytic therapy for acute ischemic stroke.[51]

Inclusion criteria
Clinical diagnosis of ischemic stroke with the sudden onset of neurological functional deficit (NIHSS ≥ 4 points or functionally significant deficit that could cause invalidity)
Onset of the symptoms less than 4.5 h, with the exception of an acute occlusion of the basilar artery (in this case, the longer therapeutic window is accepted)
Age > 18 years
EXCLUSION CRITERIA
Severe head trauma within the last 3 months
Suspicion of subarachnoid hemorrhage
Arterial puncture in non-compressible locations in the last 7 days
Intracranial hemorrhage in the last 6 months
Arterio-venous malformation or aneurysm of the cerebral arteries
Cerebral or spinal operation within the last 4 weeks
Uncontrolled arterial hypertension (systolic BP > 185 and/or diastolic BP > 110 mmHg)
Serious bleeding event within the last 3 weeks or hemorrhagic diathesis
Childbirth in the last 10 days or the third trimester of the pregnancy
Thrombocytopenia < 100,000

Administration of heparin in the last 48 h and APTT over the normal range
Therapy by anti-vitamin K and INR > 1.7
Therapy by thrombin direct inhibitors or activated factor X inhibitors and a significant alteration of the sensitive laboratory tests (APTT, INR, thrombocytopenia, and relevant tests of activated factor X activity)
Hypoglycemia < 2.7 mmol/l
Early ischemic changes in more than one-third of MCA territory on the CT scan
RELATIVE CONTRAINDICATIONS
Minor neurological deficit (NIHSS < 4 points) or quick spontaneous deficit resolution
Premorbid severe neurological deficit (mRS ≥ 4 points)
Pregnancy
Epileptic seizure with the postictal neurological deficit
Serious injury or operation in the last 2 weeks
Hyperglycemia > 22.2 mmol/l
Acute myocardial infarction in the last 3 months
Intracranial tumor

Note: APTT—activated partial prothrombin time; INR—international normalized ratio; MCA—middle cerebral artery; NIHSS—National Institute of Health Stroke Scale.

Endovascular therapy[51]

Application of IVT in acute stroke patients with occlusion of the major intracerebral artery proved only the modest effect on recanalization of the vessel. The recanalization rate is estimated as 30% in occlusion of the middle cerebral artery or the basilar artery and as low as 10% in distal occlusion of the internal carotid artery (so called T-occlusion).[62]Trials using old technology (intra-arterial thrombolysis, Merci catheter, etc.) found no benefit over IVT alone.[63-65] Current techniques of endovascular treatment (thrombectomy with modern stent retrievers) now constitute class IA indication for treatment of all patients with acute ischemic stroke, presenting within <6 h of symptom onset (or last seen well) irrespective of the presence or absence of contraindications for thrombolysis.[51]

Management of Intracerebral hemorrhage[51]

The principal element in the acute ICH therapy is to prevent progression of hematoma. Elevated blood pressure should be decreased aggressively to reach systolic blood pressure below 160 mmHg and mean arterial pressure below 110 mmHg.[66] Pharmacological options comprise the intravenous administration of labetalol, nicardipine, enalapril, hydralazine, urapidil, or nitrates. If a coagulopathy is presented, the reversal of its action is of the essence. Anticoagulant effect of warfarin should be counteracted by the administration of vitamin K together with prothrombin complex concentrates along with fresh frozen plasma (FFP). Direct oral anticoagulant dabigatran disposes of specific antidote idarucizumab that was recently approved for its activity reversal. Antidote of factor Xa inhibitors, andexanet alfa is currently studied in clinical phase. Osmotic therapy may relieve the symptoms of intracranial hypertension. Preventive management of thromboembolism and aspiration pneumonia

has similar rules as in the ischemic stroke patients. In spite of several trials, the indication for surgical treatment of the spontaneous ICH is still not resolved. Evacuation of ICH due to aneurysm or AVM and infratentorial hematoma larger than 3 cm in diameter is recommended. Surgical removal of the more superficially located hemorrhages is also generally accepted; especially when the consciousness deteriorates subsequently.[67] Ventricular drainage should be performed in the case of obstruction hydrocephalus due to the hemocephalus.

Clinical scoring systems in classifying stroke

Early bedside identification of subtype of stroke can be crucial and lifesaving. But the similarities and overlap of clinical features make this distinction difficult. This is especially useful in ischemic stroke as it can lead to early initiation of antiplatelet therapy thrombolysis.[51] In rural areas were where CT scan has not gained its access, physically and financially, clinical determinism becomes the most important factor. The earliest stroke classifying strokes were the Siriraj score (SS)[68] and Guy's hospital score (GHS) or the Allen score.[69] Recently the Greek score (GS) was introduced from Athens with claims of better sensitivity, specificity, positive and negative predictive value than the previous scores.[70] Besson et al also proposed a scoring system (Besson score or BS) that could be used to identify a fairly good proportion of ischemic stroke patients at the bedside, with good safety margin for appropriate therapy.[71]

Guy's hospital score (GHS)

The first scoring system developed in 1984 as a clinical diagnostic tool for intracranial hemorrhage (ICH). The score for each patient is obtained by calculating several clinical variables with a constant of 12.6 subtracted namely, level of

consciousness 24 hours after admission+7.3 (drowsy) or + 14.6 (unarousable); bilateral extensor plantar responses + 7.1;apoplectic onset(defined by the presence of any two of loss of consciousness at onset, headache within two hours, or neck stiffness) +21.9; diastolic blood pressure after24 hours +(blood pressurexO.17); aortic and mitral valve disease -4 3; cardiac failure -4.3;cardiomyopathy-4 3; atrial fibrillation -4 3; cardiothoracic ratio over 0.5 on chest radiography -4-3; myocardial infarction within six months -4 3; angina, claudication, or diabetes -3 7; previous transient ischemic attack or stroke -6.7; and history of hypertension-4.1.[68,69]

Siriraj score[68]

Calculating the Guy's Hospital score at the bedside is not simple due to the large no of variables, calculations involved and the need to wait for 24 hrs to complete the score. The Siriraj stroke score was developed in Thailand so that only simple computation would be needed, making it more pertinent at the bedside. Calculation of SS is shown in the Fig.6.

Fig 6. Siriraj score (SS) [72]

Scoring system	*Variable*	*Clinical feature*	*Score*	*Diagnosis*
Siriraj scoring system	Consciousness (×2.5)	Alert	0	• <-1=Ischemic stroke • >1=Hemorrhagic stroke
		Drowsy, stupor	1	
		Semicoma, coma	2	
	Vomiting (×2)	No	0	
		Yes	1	
	Headache within 2 h (×2)	No	0	
		Yes	1	
	Diastolic blood pressure (×0.1)			
	Atheroma markers (×3)	None	0	
	Diabetes, angina, intermittent daudication	One or more	1	
	Constant		−12	

A score above 1 was classified as cerebral haemorrhage whereas a score below -1 was classified as cerebral infarction.

Greek Score[14]

A relatively new score proposed by a team from Athens. It was used to differentiate between ischemic and hemorrhagic stroke. It claimed to have better sensitivity and specificity than the previous scoring systems. The diagnostic model is easily memorized and can be applied at the bedside with no need of a calculator. The authors of the scoring system observed a sensitivity of 95%, a specificity of99%, a positive predictive value of 97% and a negative predictive value of 99%.[14]

Besson scoring system[72]

Besson et al also suggested a scoring system which could be used to identify a good proportion of ischemic stroke patients at bedside. It had comparatively larger no of variables when compared to Greek score. Only one study has tested Besson score in India till date as per the best of our knowledge.[72]

METHODOLOGY

The present study was conducted in the Department of Medicine, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belagavi.

Study design and duration

The study design was a hospital based cross-sectional study.

Study period

This study was done for the period of one year from January 2016 to December 2016.

Place

The present study was carried out at KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belagavi a tertiary care teaching hospital attached to Jawaharlal Nehru Medical College, Belagavi.

Source of Data

Patients presenting with stroke above 18yrs of age to KLES Dr Prabhakar Kore Hospital and MRC, Belagavi

Sample size

A total of 61 patients with stroke (ischemic and hemorrhagic) were studied.

Sampling procedure

The following formula was used:

$$(z\alpha^2 pq)/d^2$$

Where, $z\alpha$= 1.96 (at 95% confidence interval)

p=sensitivity (as obtained from previous studies)

q= (100-p),

d= absolute error

Selection criteria

Inclusion Criteria:

- All patients of stroke >18yrs of age admitted in the department of medicine with the diagnosis of stroke *(according to WHO criteria as "Rapidly developing signs of focal (or global) disturbance of cerebral function, leading to death or lasting longer than 24 hours , with no apparent cause other than vascular")*
- Neuroimaging showing intracerebral hemorrhage or cerebral infarction

Exclusion Criteria:

- Patients with stroke due to other causes such as space occupying lesions, trauma
- Patients receiving anticoagulant therapy
- Patients with SAH(traumatic)

Ethical clearance

Prior to the commencement, the study was approved by the Institutional Ethics Committee, Jawaharlal Nehru Medical College, Belagavi.

Informed consent

The patients who fulfilled the selection criteria were informed about the nature of study and a written informed consent was obtained. In case of comatose patients, the relatives / caretakers were informed about the study. The patients/caregivers expressing their willingness to participate in the study were enrolled after obtaining a written informed consent. (Annexure–I).

Data collection

The selected patients' demographic data such as age and sex were recorded. History of other co-morbid conditions such as, hypertension, diabetes mellitus, previous stroke, personal history such as habits of alcohol consumption, smoking, were noted. A thorough physical examination was conducted for vitals (pulse rate, blood pressure and respiratory rate) followed by systemic examination. The diagnosis of stroke was entertained after fulfilling WHO definition of stroke by the patient. Clinical stroke scores were calculated. These findings were noted on a predesigned and pretested proforma (Annexure-II).

Investigations

Venous blood samples (10 mL) were collected immediately on admission to intensive care unit from the selected patients and were subjected following investigations.

- Hemogram (CBC)
- X-ray chest
- 12 lead ECG
- CT/MRI

Calculation of various stroke scores

The stroke scores were calculated from this data. The scoring systems are adapted from existing literature and are detailed below. The inferences of the scoring systems were "ischemic stroke," "hemorrhagic stroke," or uncertain, except in the BS where the inferences were "ischemic stroke" and "non ischemic stroke."

- **Greek score**

Parameter	score
Neurological deterioration within three hours of admission	6
Vomiting	4
Total leukocyte >12000	4
Decreased level of consciousness at admission	3
TOTAL	17

  - ≤3 ischemic stroke
  - >3 →<11 equivocal/uncertain
  - ≥11 hemorrhagic stroke

- **Siriraj score :**
    - <-1= ischemic stroke
    - >1= hemorrhagic stroke
    - -1 → 1 = equivocal/ uncertain

Parameter	Score	
level of consciousness (x2)	alert	0
	drowsy/stupor	1
	coma	2
vomiting (x2.5)	no	0
	yes	1
headache (x2.5)	no	0
	yes	1
atheroma markers (diabetes mellitus, angina, intermittent claudication) (x-3)	none	0
	one or more	1
diastolic BP (x0.1)	mmhg	
constant		-12

- **Bessons score:**<1 = ischemic stroke

PARAMETER	SCORE	
Alcohol consumption	Absent	0
	Present	2
Plantar response (x1.5)	Bilateral flexor	0
	Extensor ipsilateral to deficit	1
	Extensor contralateral to deficit	2
	Both extensors	3
Headache	Absent	0
	Present	3
History of transient neurological deficit	Absent	0
	Present	-5
Hyperlipidemia	Absent	0
	Present	-1.5
Atrial fibrillation at admission	Absent	0
	present	-2.5

Statistical methods

The data obtained was coded and entered into Microsoft excel spreadsheet and data was analyzed using SPSS version 23 and MedCalc software. The categorical data was expressed in terms of rates, ratios and percentages and the continuous data was expressed in terms of mean ± standard deviation. The comparison of categorical data was done using Chi-square test or Fisher's exact test. Continuous data was compared using independent sample 't' test. At 95% confidence interval, a probability (p) value of ≤ 0.050 was considered as statistically significant.

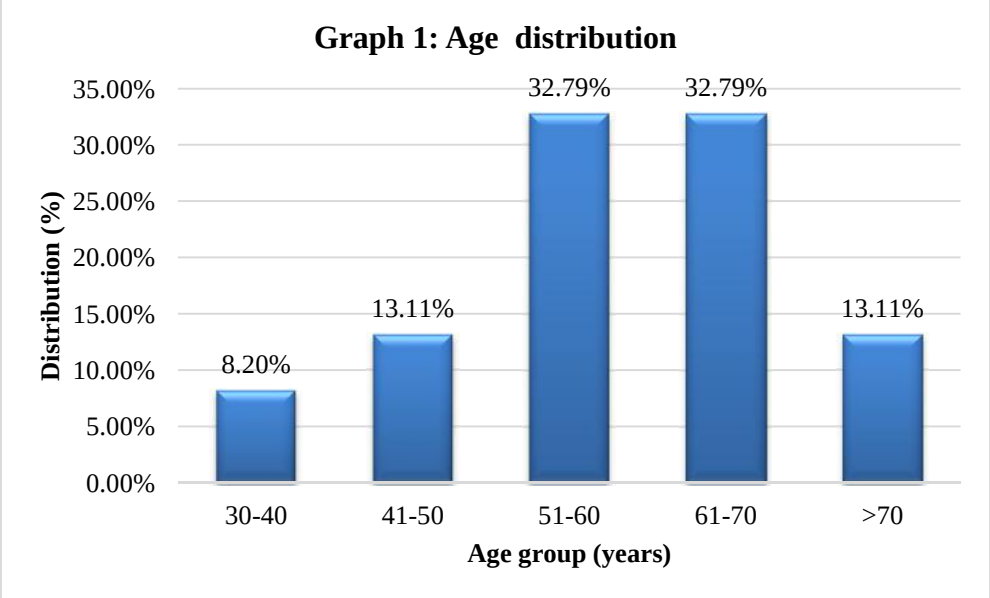
RESULTS

The present study was conducted on 61 patients presenting with acute stroke, in KLES Dr Prabhakar Kore hospital and MRC Belagavi during the period Jan 2016 to Dec 2016.

The data obtained was tabulated as below.

Table 1. Age distribution

Age group (Years)	Distribution (n=61)	
	Number	**Percentage**
30-40	5	8.20%
41-50	8	13.11%
51-60	20	32.79%
61-70	20	32.79%
>70	8	13.11%
total	**61**	**100.00%**

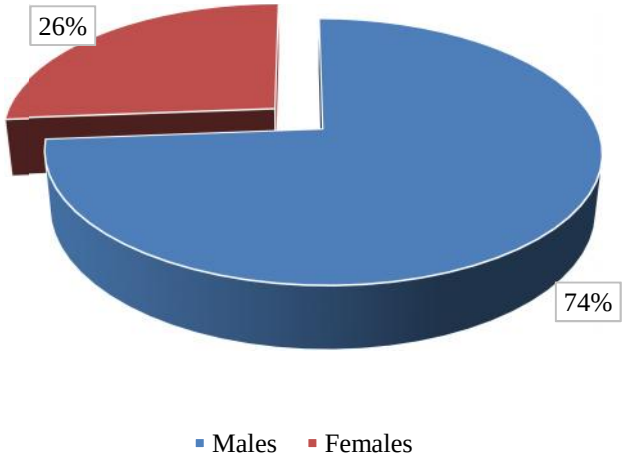


In the present study maximum number of patients were in the age group 51-60 and 61-70 years that is 20 (32.79%) in each group. The youngest patient was 33 years old and the oldest was 82 years. The mean age of stroke was 59.15.

Table 2. Sex distribution

Sex	Distribution (n=61)	
	Number	**Percentage**
Male	45	74
Female	16	26
Total	**61**	**100.00**

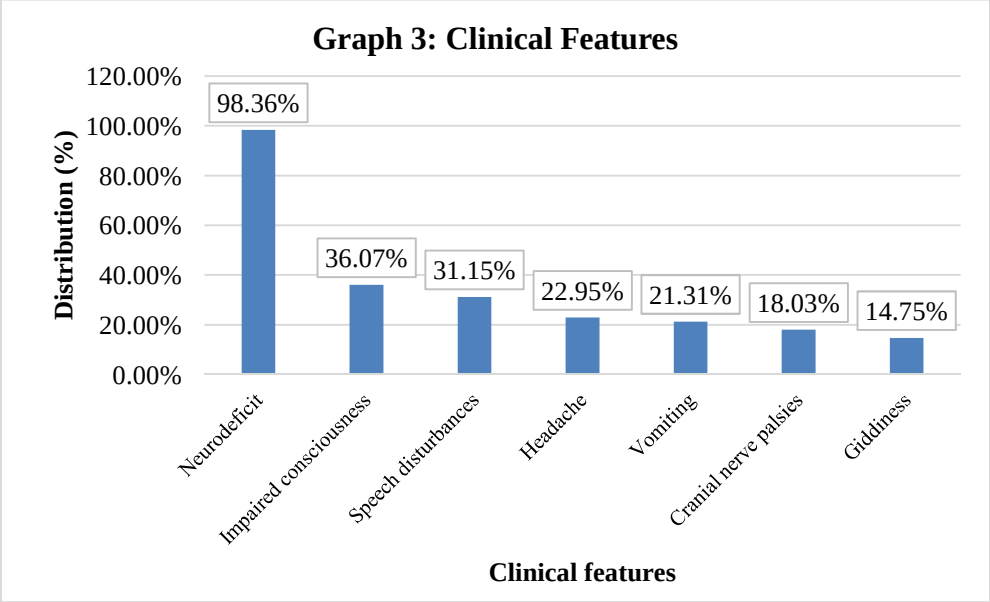
Graph 2: Sex distribution of stroke



In the present study out of 61 cases 45 (74%) were males and 16 (26%) were females. Male preponderance was seen with Male to Female ratio of 2.81:1.00.

Table 3. Clinical features

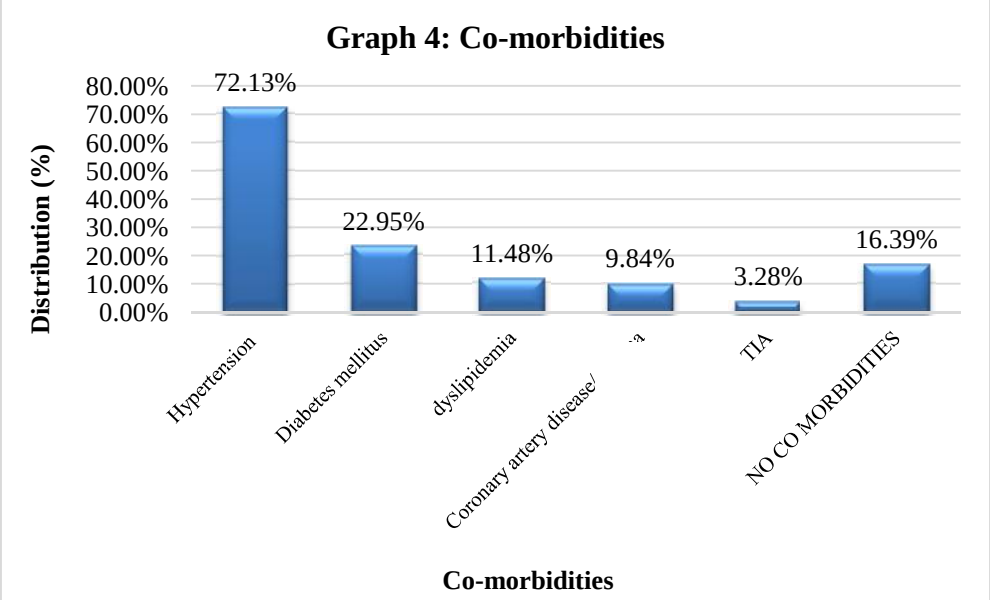
Clinical features	Distribution (n=61)	
	Number	Percentage
Neuro-deficits	60	98.36%
Impaired consciousness	22	36.07%
Speech disturbance	19	31.15%
Headache	14	22.95%
Vomiting	13	21.31%
Cranial nerve palsies	11	18.03%
Giddiness	9	14.75%



In our study the most of the patients presented with neuro-deficits 60 (98.36%), followed by impaired consciousness 22(36.07%), Speech disturbance 19 (31.15%), headache 14 (22.95%), vomiting 13 (21.31%), cranial nerve palsies 18(18.03%) and only 9(14.75%) with giddiness.

Table 4. Co morbid conditions

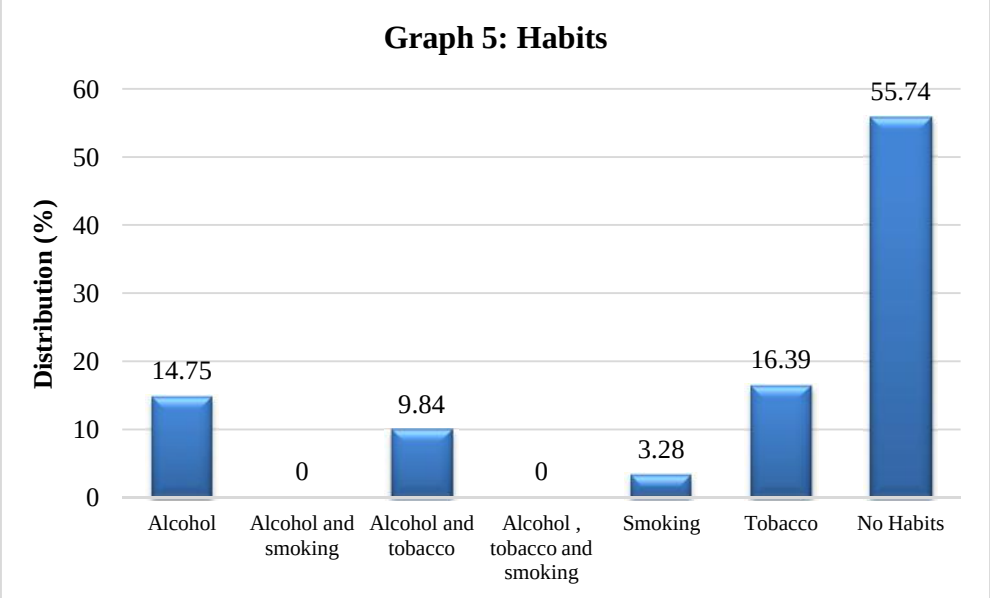
History	Distribution (n=61)	
	Number	Percentage
Hypertension	44	72.13%
Diabetes mellitus	14	22.95%
Dyslipidemia	7	11.48%
Coronary artery disease	6	9.84%
TIA	2	3.28%
NO CO MORBIDITIES	10	16.39%



In the present study most of the patients with stroke had hypertension 44 (72.13%).The other co morbid conditions are shown as above.

Table 5. Habits

Habits	Distribution (n=61)	
	Number	Percentage
Alcohol	9	14.75
Alcohol and smoking	0	o
Alcohol and tobacco	6	9.84
Alcohol , tobacco and smoking	0	0
Smoking	2	3.28
Tobacco	10	16.39
No Habits	34	55.74



In the present study tobacco chewing 10 (16.39%) was the single most common habit found among the patients with stroke. However 34 patients (55.74%) didn't have any habits.

Table 6. ECG Findings

ECG Findings	Distribution (n=61)	
	Number	**Percentage**
LVH	7	11.48%
AF	5	8.20%
old MI	4	6.56%
RBBB	1	1.64%
Normal	44	72.13%
Total	**61**	**100%**

Graph 6: ECG findings

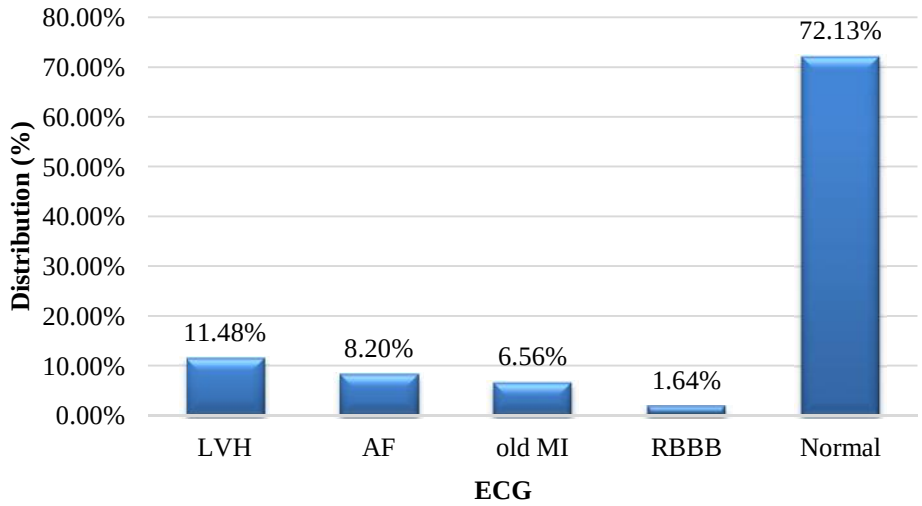


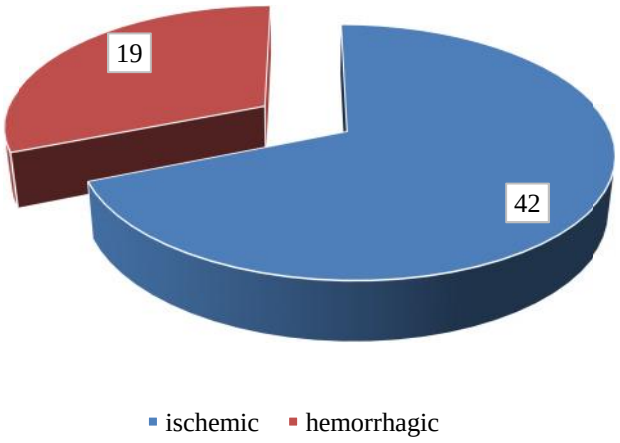
Table 8: ECG findings and stroke

In our study all patients were subjected to ECG tracing and the findings are depicted in the above table.

Table 7. Type of stroke on neuro-imaging

Neuro-imaging (CT/MRI)		
Ischemic	Hemorrhagic	total
42 (68.85%)	19 (31.15%)	61

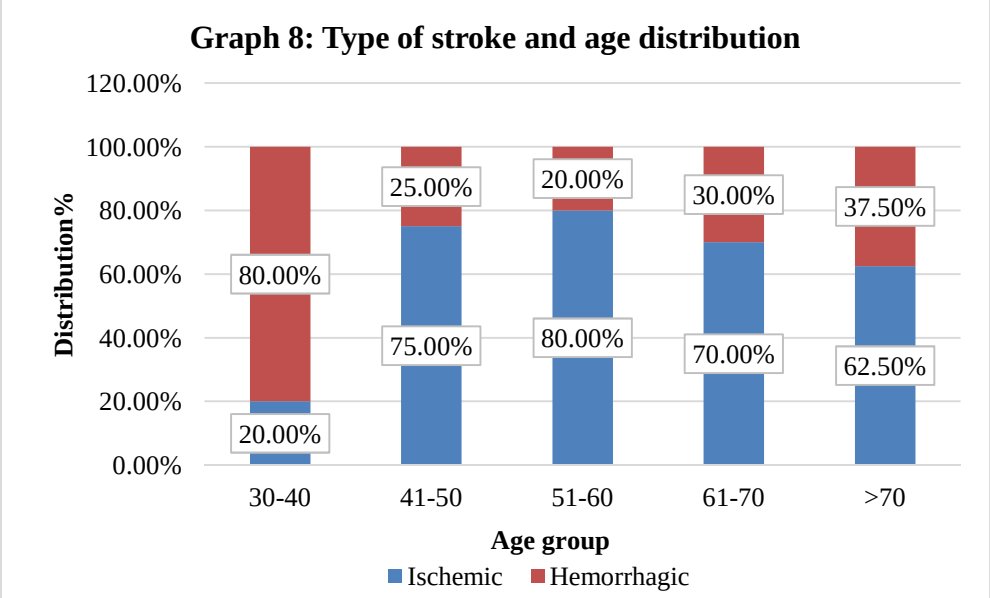
Graph 7: Stroke- Ischemic vs Hemorrhagic



All patients in our study were subjected to neuro-imaging (CT/MRI) of which 42 (68.85) were ischemic and 19 (31.15%) were hemorrhagic stroke.

Table 8. Age distribution and type of stroke

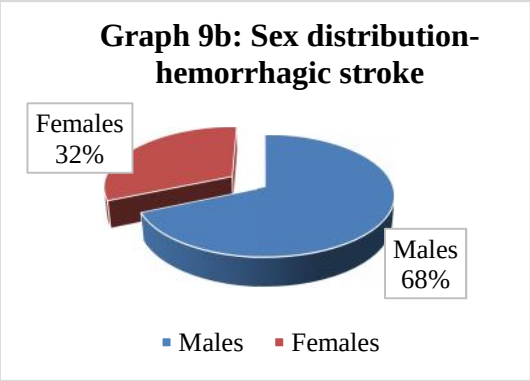
Age	Ischemic	Hemorrhagic	total
30-40	1 (20%)	4 (80%)	5
41-50	6 (75%)	2 (25%)	8
51-60	16 (80%)	4 (20%)	20
61-70	14 (70%)	6 (30%)	20
>70	5 (62.5%)	3 (37.5%)	8



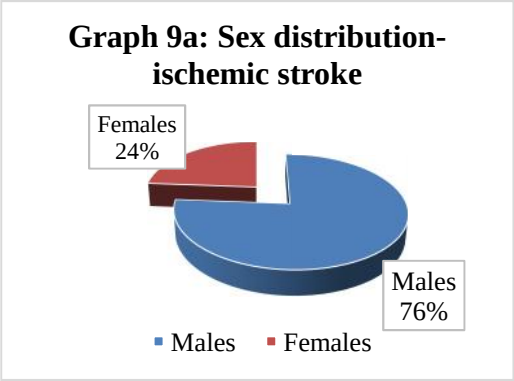
In our present study, type of stroke with varying age is shown the above table.

Table 9. Sex distribution and type of stroke

Sex	Distribution (n=61)		
	Ischemic stroke	**Hemorrhagic stroke**	**Total**
Male	32	13	45 (73.77%)
Female	10	6	16 (26.23%)
Total	**42**	**19**	**61**



Graph 9b: Sex distribution- hemorrhagic stroke

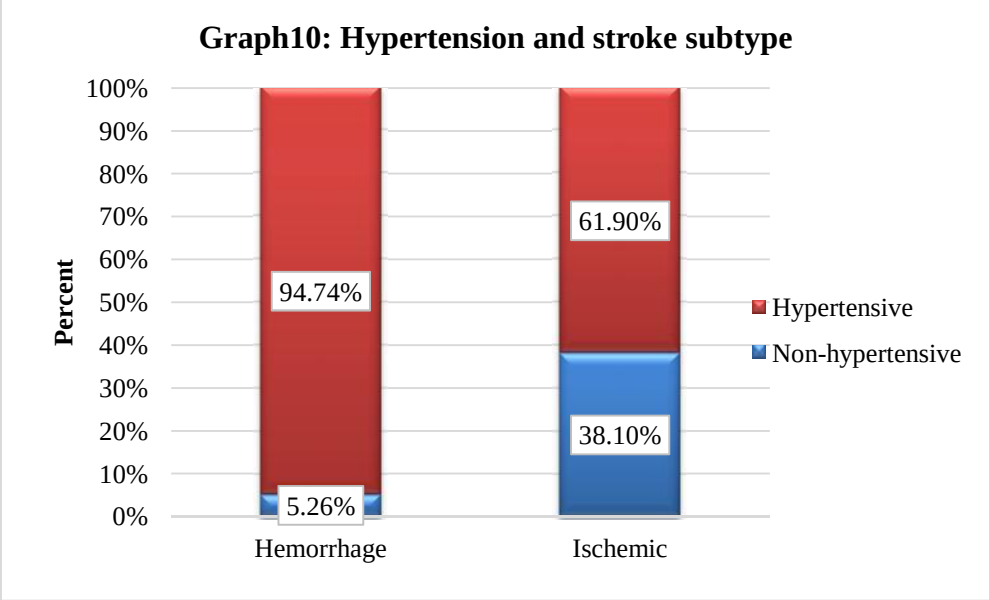


Graph 9a: Sex distribution- ischemic stroke

Among 42 patients of ischemic stroke 76% were males and 24% females, while among 19 patients of hemorrhagic stroke 68% were males and 32 % females.

Table 10.Associationofhypertension with stroke

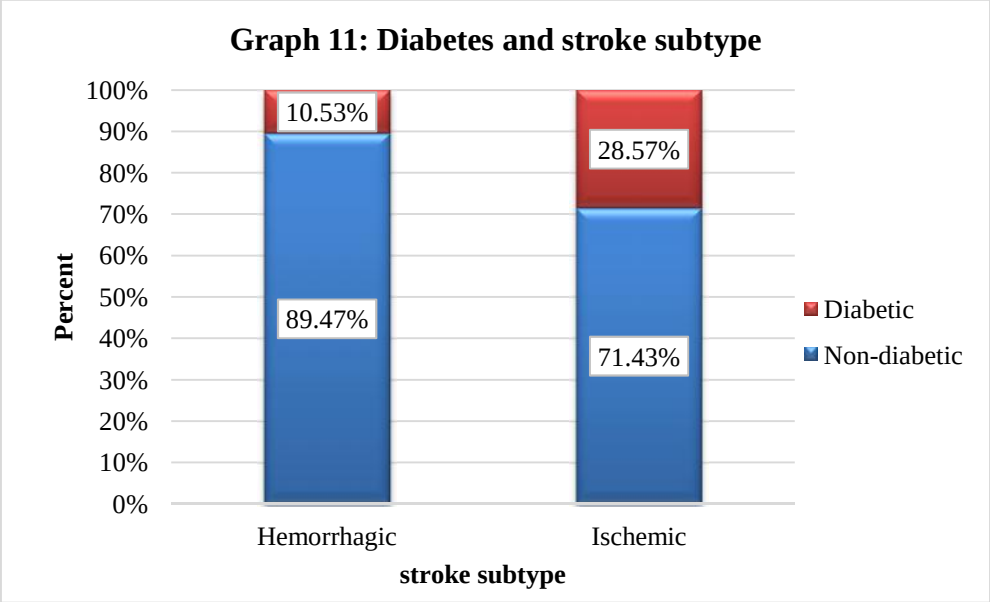
Co-morbidity	Distribution (n=61)		
	Hemorrhagic stroke	Ischemic stroke	TOTAL
Non-Hypertensive	1 (5.26%)	16 (38.10%)	17 (27.87%)
Hypertensive	18 (94.74 %)	26(61.90%)	44 (72.13%)
TOTAL	19	42	61
p value	0.0193		



In our study 94.74% of patients with hemorrhagic stroke had hypertension whereas 61.90% of ischemic stroke patients had hypertension.

Table 11.Associationof Diabetes with stroke

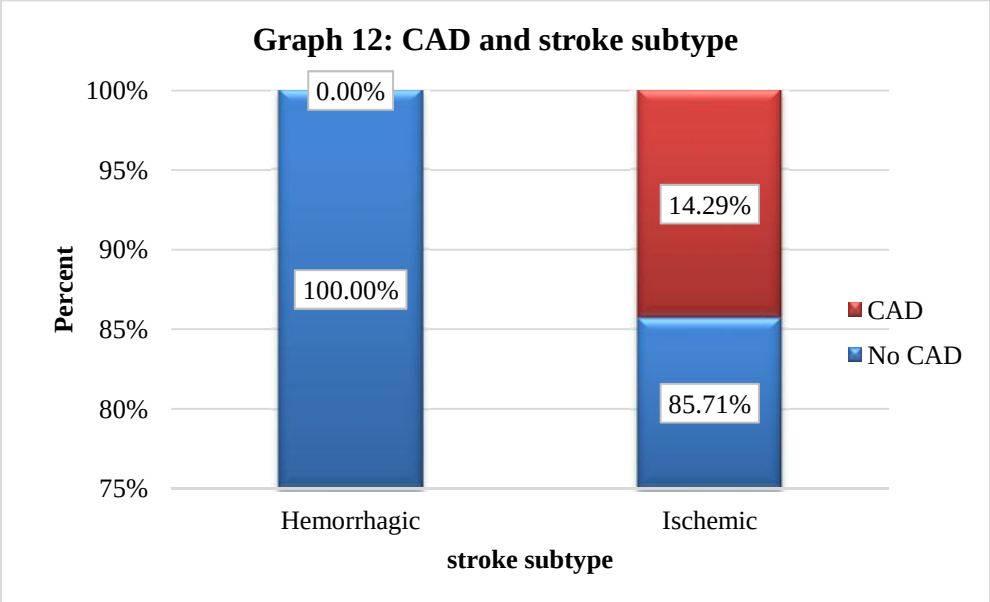
Co-morbidity	Distribution (n=61)		
	Hemorrhagic	Ischemic	**TOTAL**
Non-diabetic	17 (89.47%)	30 (71.43%)	47 (77.05%)
Diabetic	2 (10.53%)	12 (28.57%)	14 (22.95%)
TOTAL	19	42	61
p value	0.2212		



Different type of stroke in patients of diabetes and non-diabetes is shown in the above table.

Table 12.AssociationofCoronary artery disease (CAD) with stroke

Co-morbidity	Distribution (n=61)		
	Hemorrhagic	Ischemic	TOTAL
No CAD	19 (100%)	36 (85.71%)	55 (90.16%)
CAD	0	6 (14.29%)	6 (9.84%)
TOTAL	19	42	61
p value	0.2038		

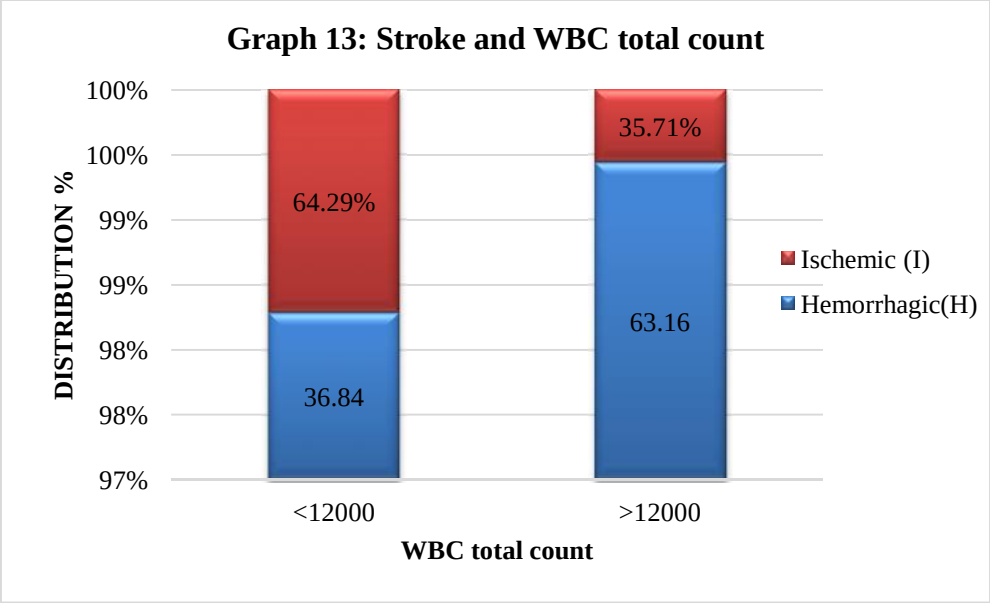


In our study; patients with hemorrhagic stroke, none had CAD whereas in patients with ischemic stroke 6 had associated CAD.

Table 13.Association of Total WBC count with stroke

WBC total count	Distribution (n=61)	
	Hemorrhagic stroke	Ischemic stroke
≤12000	7 (36.84%)	27 (64.29%)
>12000	12 (63.16%)	15 (35.71%)
Total	19 (100%)	42 (100%)

	Hemorrhagic stroke		Ischemic stroke	
WBC TOTAL COUNT	MEAN	S.D.	MEAN	S.D.
	13152.63	3592.95	11080.95	3683.07
p VALUE	0.0449			

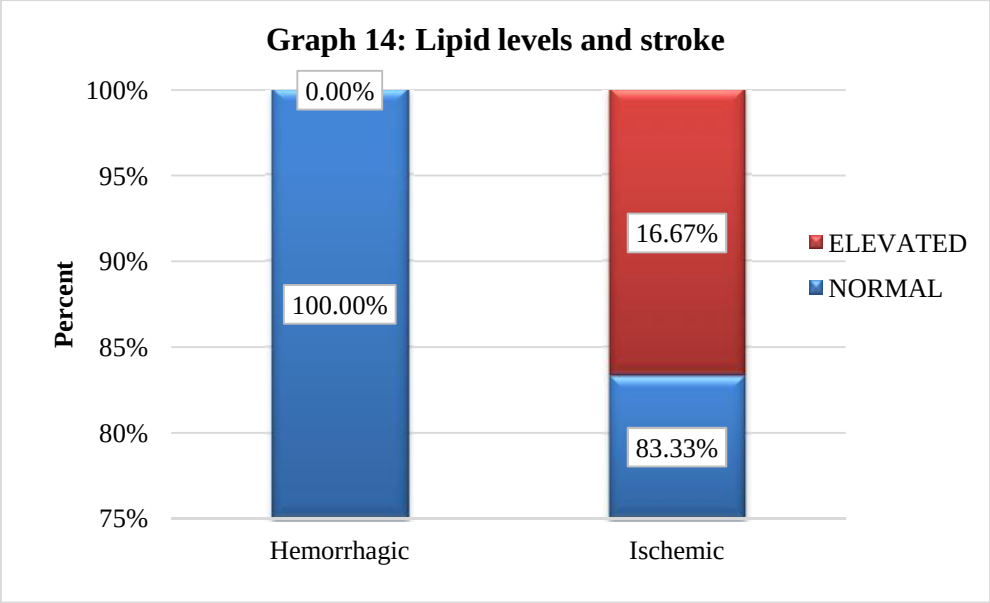


In the present study, we observed in patients with total WBC count ≤12000, 7 had hemorrhagic stroke and 27 had ischemic stroke. In patients with total WBC count >12000 12 had hemorrhagic stroke and 15 had ischemic stroke.

Table 14.AssociationofDyslipidemia with stroke

LIPIDS	Distribution (n=61)		
	Hemorrhagic	**Ischemic**	**Total**
Normal	19 (100%)	35 (83.33%)	54 (88.52%)
Elevated	0 (0)	7 (16.67%)	7 (11.48%)
TOTAL	19	42	61

(P value-0.1449)



In the present study; lipid abnormalities with stroke, we found 54 patients with normal lipids (19 hemorrhagic and 35 ischemic stroke) and in patients with elevated lipid 7 were in the ischemic stroke group. The same is shown in the above table.

Table 15a: Comparison of Greek Score (GS) with neuro-imaging for the diagnosis of hemorrhagic stroke

GREEK SCORE	IMAGING REPORT	
	Hemorrhagic stroke	Not hemorrhagic stroke
Hemorrhagic stroke	10	1
Non hemorrhagic stroke	9	41

Table 15b: Validity of Greek scoring system by different tests

SENSITIVITY	SPECIFICITY	POSITIVE PREDICTIVE VALUE (PPV)	NEGATIVE PREDICTIVE VALUE (NPV)
52.63%	97.62%	90.91%	82.00%

Diagnosis of hemorrhagic vs non hemorrhagic stroke by GS and comparing with neuro-imaging is shown above. Sensitivity and specificity of GS is 52.63 and 97.62 respectively with positive predictive value (PPV) of 90.91% and negative predictive value (NPV) of 82%.

Table 16a. Comparison of Greek Score with neuro-imaging for the diagnosis of Ischemic stroke

GREEK SCORE	IMAGING REPORT	
	Ischemic stroke	Non ischemic stroke
Ischemic stroke	26	3
Non ischemic stroke	16	16

Table 16b: Validity of Greek scoring system by different tests

SENSITIVITY	SPECIFICITY	POSITIVE PREDICTIVE VALUE (PPV)	NEGATIVE PREDICTIVE VALUE (NPV)
61.90%	84.21%	89.66%	50.00%

GS and comparison with neuro-imaging for Ischemic stroke revealed above facts as shown in table 16a. The validity of the same by different tests showed sensitivity 61.9%, specificity of 84.21%, PPV of 89.66% and NPV of 50%

Table 17a. Comparison of Siriraj Score (SS) with neuro-imaging for the diagnosis of hemorrhagic stroke

SIRIRAJ SCORE	IMAGING REPORT	
	Hemorrhagic stroke	Not hemorrhagic stroke
Hemorrhagic stroke	15	2
Non hemorrhagic stroke	4	40

Table 17b: Validity of Siriraj scoring (SS) system by different tests

SENSITIVITY	SPECIFICITY	POSITIVE PREDICTIVE VALUE (PPV)	NEGATIVE PREDICTIVE VALUE (NPV)
78.95%	95.24%	88.24%	90.91%

In our present study applying SS for diagnosing ischemic stroke, the above table 17a shows the split up. When validity tests were applied sensitivity was 78.95% and specificity was 95.24% with PPV of 88.24% and NPV of 90.91%

Table 18a. Comparison of Siriraj Score with neuro-imaging for the diagnosis of Ischemic stroke

SIRIRAJ SCORE	IMAGING REPORT	
	Ischemic stroke	Non ischemic stroke
Ischemic stroke	34	2
Non ischemic stroke	8	17

Table 18b: Validity of Siriraj scoring system by different tests

SENSITIVITY	SPECIFICITY	POSITIVE PREDICTIVE VALUE	NEGATIVE PREDICTIVE VALUE
80.95%	89.47%	94.44%	68.00%

In our study, Siriraj score had a sensitivity of 80.95% and a specificity of 89.47% in identifying ischemic stroke. It had a positive predictive value of 94.44% and a negative predictive value of 68%.

Table 19a. Comparison of Besson Score with neuro-imaging for the diagnosis of Ischemic stroke

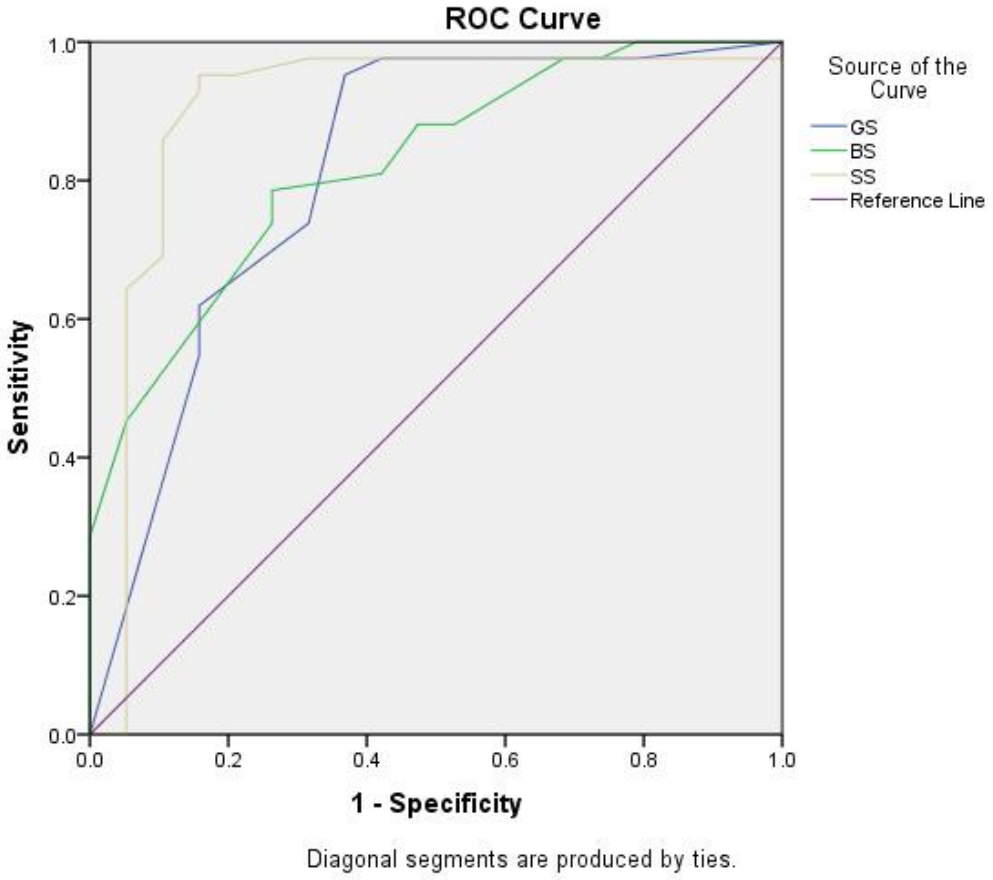
BESSON SCORE	IMAGING REPORT	
	Ischemic stroke	Non ischemic stroke
Ischemic stroke	19	1
Non ischemic stroke	23	18

Table 19b: Validity of Besson scoring system by different tests

SENSITIVITY	SPECIFICITY	POSITIVE PREDICTIVE VALUE	NEGATIVE PREDICTIVE VALUE
45.24%	94.74%	95.00%	43.90%

In our present study, Besson score was applied for the diagnosis of ischemic stroke and found to have the above result as shown in table 19a. Validity of scoring system showed sensitivity of 45.24%, specificity of 94.74%, and positive predictive value of 95% and a negative predictive value of 43.90%.

Table 20: Receiver operating characteristic (ROC) curve of Greek score (GS), Siriraj score (SS) and Besson score (BS)



Area under the Curve

Test Result Variable(s)	Area
GS	.813
BS	.822
SS	.902

In our study on plotting the ROC curve for various scoring systems, observation for Area under curve (AUC) are SS (0.902), BS (0.822) and GS (0.813).

DISCUSSION

In this cross sectional study, 61 patients with hemorrhagic stroke and ischemic stroke were studied and evaluated for comparison to clinical scoring systems namely Greek score, Besson score and Siriraj score and same were compared to factors such as demographic, co-morbidities and neuro-imaging (CT/MRI).

In the present study, it was observed that maximum number of patients were in the age group 51-60 and 61-70 years [20 (32.79%) in each group]. The youngest patient with stroke was 33 years old and the oldest was 82 years. The mean age of stroke was 59.15. This is almost same to the study done by Sarkar et al and Saini et al.[75, 76]

In our study out of 61 cases 45 (74%) were males and 16 (26%) were females. Male preponderance was seen with Male to Female ratio of 2.81:1. Goswami et al in their study did not find any gender difference.[72]

The clinical presentation of patients were as follows: 60 patients (98.36%) presented with neuro-deficits, followed by impaired consciousness 22 (36.07%), Speech disturbance 19 (31.15%), headache 14 (22.95%), vomiting 13 (21.31%), cranial nerve palsies 18 (18.03%) and only 9 (14.75%) with giddiness. Some of these patients had an overlap of these symptoms. Study by Carlo et al found almost same clinical presentation in their study group.[77]

We studied for associated co-morbid conditions in our 61 patients and found 44 (72%) had hypertension, 14 (22.95%) had diabetes, 7 (11.48%) had dyslipidemia, 6 (9.84%) had coronary artery disease (CAD) and 2 patients (3.28%) had past history

of Transient Ischemic Attack (TIA). 10 patients (16.39%) did not have any co-morbid conditions.Most hospital-based and community-based studies have found hypertension as the most common risk factor. Even the study by Dalal et al and Banerjee et al have drawn the same conclusion.[78, 79]

In our 61 patients, 27 patients had one or the other habits like tobacco chewing, alcohol consumption, or smoking. 34 patients (55.74%) did not have any habits. A study by Banerjee found 40% of their stroke patients had the habit of chewing tobacco.[80]

ECG tracing of the patients revealed 44 patients (72.13%) had a normal ECG. 7 patients that is 11.48% had Left Ventricular Hypertrophy (LVH) (who were also hypertensives). 5 patients (8.20%) had Atrial fibrillation (AF). 4 patients (6.56%) had evidence of old myocardial infarction (MI). This is in sharp contrast to a study by Sarkar et al who found LVH in 41.46% of their patients.[75]

In our study, we attempted to categorize the type of stroke on neuro-imaging and found 42 patients (68.85%) had ischemic stroke and 19 patients (31.15%) had hemorrhagic stroke. Our study is almost similar to an Indian study by Banerjee et al.[6] and a south Asian study by Wasay et al.[25]

Similarly, type of stroke according to age was attempted in our study. To our observation, most patients with ischemic stroke were in the age group of 51-60 yrs i.e. 16 patients (80%) and 14 pts (70%) in 61-70 yrs age group. In the same age groups hemorrhagic stroke were 4 (20%) and 6 (30%) respectively. Similar observation were made by Wasay et al.[25]

When gender and type of stroke were analyzed 45 male pts had stroke (32 ischemic and 13 hemorrhagic stroke) and 16 female patients were found to have stroke (10 ischemic and 6 hemorrhagic). This is in sharp contrast to the study by Dalal et al.[78]

Comparison with co-morbid conditions like hypertension showed 44 patients (72.13%) having stroke (18 hemorrhagic and 26 ischemic) had hypertension, whereas in normotensive patients 17 (27.87%) (1 hemorrhagic and 16 ischemic) had stroke. Sarkar et al found similar association.[75]

Association of diabetes with stroke, majority of patients without diabetes (non-diabetic) had stroke that is 47 patients (77.05%) (17 hemorrhagic and 30 ischemic), whereas patients with diabetes 14 (22.95%) had stroke (2 hemorrhagic and 12 ischemic stroke). This is in sharp contrast to a study by Sarkar et al.[75]

Similarly comparison with CAD was done, only 6 patients (9.84%) with CAD had ischemic stroke, none had hemorrhagic stroke in this group. Most of the studies have not associated stroke with CAD.

Lab parameters like Total WBC count with stroke was compared and found to have 34 patients (55.74%) with total WBC count <12000 (7 hemorrhagic and 27 ischemic). 27 patients had total WBC counts >12000 (12 hemorrhagic and 15 ischemic). In our study though the difference of mean value for hemorrhagic stroke (13152.6) and ischemic stroke (11080.95) was small, it was statistically significant. A study by Efstathiou et al, the difference between the mean value for both hemorrhagic and ischemic stroke was more and it was very significant statistically.[14] Maybe this

difference in their study was due to large sample size as compared to our study of small sample size.

Similarly an attempt was made to compare stroke with lipids, majority of patients had normal lipids [54 (88.52%)]. All hemorrhagic stroke patients had normal lipids while 7 pts (11.48%) had lipid abnormality and all were ischemic stroke. This is in sharp contrast to the study by Saini et al.[76]

We attempted comparing the stroke patients in our study to various scoring systems like Greek score (GS), Siriraj score (SS) and Besson score (BS) and found to have the following observations.

Greek scoring system in our study showed highest specificity (97.62%) to diagnose hemorrhagic stroke whereas sensitivity was only 52.63%. similarly when Siriraj score (SS) was considered there was a high specificity of 95.24% and sensitivity of 78.95% for hemorrhagic stroke. In SS the sensitivity was more when compared to GS. SS was also helpful in identifying ischemic stroke in our study. When BS was used for identifying ischemic stroke, sensitivity and specificity revealed sensitivity of only 45.24% and specificity of 94.74% for identifying ischemic strokes alone

From the above observations we conclude SS is better for identifying both ischemic as well as hemorrhagic stroke. However in our observation we found GS is good for hemorrhagic and BS for ischemic stroke.

Finally we attempted to compare all three scores (GS, SS, and BS) by plotting Receiver operating characteristic (ROC) curve and comparing it with the area under curve (AUC) and found the following AUC's: GS- 0.813, SS- 0.902 and BS- 0.822.

From the above plotting chart of ROC curve with AUC revealed SS is good for diagnosing both the strokes. Similarly BS was the second alternative scoring system for diagnosing stroke and least was with GS. A study by Goswami et al compared in their study group all the three scoring systems (GS, BS, SS) and they found GS was better in diagnosing strokes compared to other 2 scoring systems.[72] They also found GS was better in identifying strokes followed by SS and last BS. This difference could be because of more sample size in their study group as compared to our small study.

CONCLUSION

In the present study of 61 patients with ischemic and hemorrhagic stroke, we observed significant correlation with various factors. Based on findings of the study prominent features are mentioned below.

- Among the patients presented with stroke maximum cases were in the 51-70yrs age group.
- Though males were more in number as compared to females whether gender influences the stroke or type of stroke is difficult to state because of small sample size.
- The common clinical presentations were neuro-deficits followed by impaired consciousness, speech disturbances, headache, vomiting and cranial nerve palsies.
- Hypertension was a significant co-morbid condition for stroke, more so for hemorrhagic stroke.
- Habits didn't have any bearing on strokes.
- ECG findings in majority of the patients were normal. 7 patients revealed LVH all were hypertensive.
- On comparing different scoring systems (GS,SS & BS), we found SS was a better scoring system for both types of strokes
- Although these scoring systems help in differentiating ischemic and hemorrhagic stroke on arrival of patients to the casualty, all have certain limitations. Hence we feel neuro-imaging is the best in differentiating the type of stroke.

To overcome biases like sex, habits, diabetes, and lipid abnormalities we feel it is worth to consider these factors with large sample size to see whether there are any co-relations.

SUMMARY

The present study was conducted to know various demographic factors, clinical presentation, clinical and CT/MRI findings and comparing with various factors like age, sex co-morbid condition and different scoring systems. The present study was done on 61 patients who presented with stroke to KLES Dr. Prabhakar Kore hospital and MRC, Belagavi during the period Jan 2016 to Dec 2016.

- Strokes were common in the age group 51-70 years.
- Ischemic strokes were more as compared to hemorrhagic strokes
- Male patients were more with stroke (ischemic stroke- 32, hemorrhagic stroke-13) as compared to females (10 ischemic stroke and 6 hemorrhagic stroke)
- Hypertension was observed in 44 pts, remaining 17 were normotensives
- 47 patients were non diabetic and 14 were diabetic
- Hypertension had significant influence on stroke but diabetes did not reveal any significant influence.
- Majority of hypertensive patients had hemorrhagic stroke (18 out of 19). Only one normotensive had hemorrhagic stroke.
- The Siriraj scoring system was better tool of scoring in identifying type of stroke in our study.

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ANNEXURE I

CONSENT FOR PARTICIPATION IN RESEARCH

Mr. /Mrs._____ we are requesting you to enroll yourself in study titled "**IDENTIFICATION OF BEDSIDE CLINICAL SCORING SYSTEMS IN CLASSIFYING STROKE AND ITS SUBSEQUENT MANAGEMENT.**-A Study conducted J. N. Medical College, Belagavi.

You have been requested to participate in research because your profile matches with the study group. All the patients admitted with STROKE can become participants of study. During the study you will be asked some questions and you are supposed to answer to the best of your knowledge.

Your participation in the research is absolutely voluntary. Your decision to participate in the study or otherwise will not affect your relationship with J.N.M.C. If you decide not participate you are free to withdraw at any time.

The purpose of research is to identify which of the scores have the best sensitivity and specificity to identify between ischemic and haemorrhagic stroke in our Indian population within our demographic setting.

Procedure involved

A detailed history, clinical examination, blood investigations and imaging investigations like CT/MRI which are not invasive procedures.

Risks and benefits

There are no risks involved and benefits are many. The study helps to identify various clinical features of the disease and identify different causative factors,

avoidance of which prevents future recurrence stroke. The results deduced at the end of study will help all similar patients admitted in the hospital to be diagnosed clinically.

Alternatives

Even if you decline to participate, there will not be any change in the line of your management or the relationship with your doctor. You will be told about all the new information that may affect your decision to participate in the study.

Withdrawing/removal from study

You can withdraw any time from the study as you wish. You will not be penalized for such a decision.

Privacy and confidentiality

The only people to know that you are a research subject are the members of research team. No information about you or provided by you during the research will be disclosed to others without your written permission except:

In case of emergency to protect your rights and welfare if required by law.

Financial incentives for participation

You will not be paid any monetary benefits or free gifts for participation in the research. You will not be reimbursed for expenses.

Authorization to publish results

When the results of the research are published or discussed in a conference, no information will be displayed that would disclose your identity. Any information that is obtained in connection with this study and that can be identified with you will remain confidential.

CONSENT STATEMENT

I, the undersigned, have been explained in my own vernacular language about the study and my participation in the study is voluntary. If I want I can withdraw at any time. Also I have been given enough time to clear my doubts about the study and my rights as a study participant.

In case you have any questions about your rights as a study participant you can contact Dr. Ganga Pilli (0831-2473777).

Signature or the left thumb impression of the participant or legally authorized representative.

Participant's name: _____Signature: _____

Witness name: _____ Signature: _____

Experimenter's name: _____ Signature: _____

Guardian's name: _____ Signature: _____

Place: _____

Date: _____

ANNEXURE-II

PROFORMA

Case No:

NAME:

AGE/SEX:

IP No.

ADDRESS:

OCCUPATION:

COMPLAINTS AT PRESENTATION:

RISK FACTORS

EXISTING DIAGNOSIS:

ADMISSION DIAGNOSIS:

1)

2)

3)

PHYSICAL EXAMINATION:

GENERAL CONDITION:

Pallor: Yes/No

Icterus: Yes/No

Lymphadenopathy: Yes/No

Cyanosis: Yes/No

Clubbing: Yes/No

Edema: Yes/No

VITALS:

Temperature:

Pulse:

Respiratory rate:

Blood pressure:

GCS: at admission:
after 3hrs:

SYSTEMIC EXAMINATION:

R. S.:

C.V.S.:

P.A.:

C.N.S.:

INVESTIGATIONS

- Total leukocyte count:
- ECG
- MRI/CT

Greek score

	score	**Pt score**
Neurological deterioration within three hours of admission	6	
Vomiting	4	
Total leukocyte >12000	4	
Decreased level of consciousness at admission	3	
TOTAL	17	

≤3 ischemic stroke

≥11 hemorrhagic stroke

BESSONS SCORE : <=1 Ischemic stroke >1 haemorrhagic stroke

			PT SCORE
Alcohol consumption	Absent	0	
	Present	2	
Plantar response (x1.5)	Bilateral flexor	0	
	Extensor ipsilateral to deficit	1	
	Extensor contralateral to deficit	2	
	Both extensors	3	
Headache	Absent	0	
	Present	3	
History of transient neurological deficit	Absent Present	0 -5	
Hyperlipidemia	Absent	0	
	Present	-1.5	
Atrial fibrillation at admission	Absent present	0 -2.5	
TOTAL			

SIRIRAJ SCORE

PARAMETER	SCORE		PT SCORE
LEVEL OF CONSCIOUSNESS (x2)	ALERT	0	
	DROWSY/STUPOR	1	
	COMA	2	
VOMITING (x2.5)	NO	0	
	YES	1	
HEADACHE (x2.5)	NO	0	
	YES	1	
ATHEROMA MARKERS (DIABETES MELLITUS, ANGINA, INTERMITTANT CLAUDICATION) (x-3)	NONE	0	
	ONE OR MORE	1	
DIASTOLIC BP (x0.1)	mmHg		
CONSTANT		-12	
TOTAL			

ANNEXURE III - MASTER CHART

Serial Number	In patient number	Age (Years)	Sex	Presenting complaints																Past History										Examination																	TYPE OF STROKE			
																														BP		Systemic															IMAGING REPORT	GREEK SCORE REPORT	BESSON SCORE REPORT	SIRIRAJ SCORE REPORT
				Dysarthria	dysphagia	visual loss	Weakness	Tingling	Global aphasia	Giddiness	Slumping	Motor aphasia	Deviation angle of mouth	Altered sensorium	Drowsiness	Unconscious	headache	vomiting	Diabetes mellitus	Hypertension	Coronary artery disease/Angina	dyslipidemia	intermittant claudication	TIA	tobbaco chewer	Smoking	Alcohol consumption	Systolic (mm Hg)	Diastolic (mm Hg)	Cardiovascular system	Respiratory system	Per abdomen	GCS	Worsening of consciousness after 3 hrs	Level of consciousness	plantars	total leukocyte count	ecg	MRI	greek score	besson score	siriraj score								
1	713851	53	M	-	-	-	+	-	-	+	-	-	-	-	-	+	+	+	-	+	-	-	-	-	+	-	-	170	110	S1S2+	nvbs	nad	14	+	alert	↑↑	15500	LVH	right putaminal bleed with bi-basal ganglionic calcification	14	6	3	H	H	H	H
2	716131	63	F	-	-	-	+	-	-	+	-	-	+	-	-	-	-	-	-	-	-	+	-	-	-	-	-	110	70	S1S2+	nvbs	nad	15	-	alert	↓↓	9300	WNL	Acute left MCA territory infarct	0	-1.5	-5	I	I	I	I
3	712552	57	M	-	-	-	+	-	-	-	-	-	-	+	-	-	-	-	-	+	-	-	-	-	+	-	+	130	90	S1S2+	nvbs	nad	7	-	drowsy	↓↑	19600	WNL	acute rt MCA territory infarct with hypertensive ischaemic changes	7	3.5	-0.5	I	E	H	E
4	715538	50	M	+	-	-	+	-	-	-	+	-	+	-	+	-	-	-	-	-	+	-	-	-	-	-	+	140	90	AF	nvbs	nad	9	-	drowsy	↓↑	16900	AF	acute rt MCA territory infarct	7	-1	-0.5	I	E	I	E
5	716301	66	M	-	-	-	+	-	-	+	-	-	+	-	-	-	-	-	-	+	+	-	-	-	-	-	-	150	90	S1S2+	nvbs	nad	15	-	alert	↓↑	10800	WNL	Subacute infarct rt thalamus and left high frontal region	0	1.5	0	I	I	H	E
6	718067	78	M	-	-	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	-	-	+	-	110	70	S1S2+	nvbs	nad	15	-	alert	↓↓	7200	WNL	s/a infarct in post. Limb of int. capsule, corona radiata, putamen. Htn changes	0	0	-8	I	I	I	I
7	719271	60	F	-	-	-	+	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	150	80	S1S2+	nvbs	nad	13	-	alert	↑↓	15100	WNL	a/c left frontoparietal and basl ganglia infarct with hyperdense left MCA	7	1.5	-1.5	I	E	H	I
8	718668	68	M	+	-	-	+	-	-	+	-	-	-	-	-	-	-	-	-	+	+	-	-	-	-	-	-	150	100	S1S2+	nvbs	nad	15	-	alert	↑↓	13500	WNL	Htn bleed in left thalamus. Htn changes. Age related cerebral atrophy	4	1.5	-14	H	E	H	I
9	716284	82	M	-	-	-	+	-	-	-	+	-	-	+	-	-	+	-	-	+	+	-	-	-	-	-	-	180	90	S1S2+	nvbs	nad	11	-	drowsy	↓↑	8600	RBBB	s/a infarct in rt MCA territory	3	4.5	-1.5	I	I	H	I
10	706471	65	M	-	-	-	+	-	-	+	-	-	-	-	-	-	-	-	-	-	+	-	-	+	-	-	-	160	90	S1S2+	nvbs	nad	11	-	drowsy	↓↑	13200	WNL	s/a infarct in rt MCA territory , gliosis in the left MCA territory	7	-3.5	-0.5	I	E	I	E
11	749449	52	M	+	-	-	+	-	-	-	-	-	-	-	-	-	+	-	-	+	-	-	-	-	-	+	-	180	100	S1S2+	nvbs	nad	15	-	alert	↓↑	10600	WNL	s/a infarct in rt MCA territory	0	4.5	-3	I	I	H	I
12	715557	48	M	-	-	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	110	70	S1S2+	nvbs	nad	15	-	alert	↓↓	5600	WNL	s/a infarct in lt MCA territory	0	0	-5	I	I	I	I
13	715285	70	M	-	-	-	+	-	-	-	-	-	-	+	-	-	-	-	-	+	+	-	-	-	-	-	+	110	90	S1S2+	nvbs	nad	15	-	alert	↓↓	8600	WNL	acute infarct in left temporal and parietal region	0	0	-6	I	I	I	I
14	711366	46	F	-	-	-	+	-	-	-	-	-	-	+	-	-	+	+	-	-	+	-	-	-	-	-	-	190	100	S1S2+	nvbs	nad	13	+	drowsy	↓↑	12600	WNL	intraparenchymal Hg in the rt lentiform nucleus	17	4.5	4.5	H	H	H	H
15	718920	54	M	+	-	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	-	-	+	-	150	90	S1S2+	nvbs	nad	15	-	alert	↓↑	11300	WNL	acute infarct in the post limb of the int capsule	0	1.5	-3	I	I	H	I
16	715628	67	M	+	-	-	+	-	-	-	-	-	-	-	-	+	-	+	-	+	+	-	-	-	+	-	-	150	70	S1S2+	nvbs	nad	4	+	coma	↑↑	13600	WNL	s/a infarct in right int capsule and corona radiata. Focal gliosis in lt frontal area	17	4.5	-1	I	H	H	E
17	720055	77	F	-	-	-	+	-	-	-	+	-	-	-	-	+	-	+	-	-	+	-	-	-	-	-	-	170	90	S1S2+	nvbs	nad	7	+	stupor	↓↑	13700	WNL	rt htn thalamic bleed with perilesional edema & mass effect with midline shift	17	1.5	1.5	H	H	H	H
18	716376	52	M	+	-	-	+	-	-	-	-	-	+	-	-	-	-	-	-	+	+	-	-	-	+	-	-	180	110	S1S2+	nvbs	nad	12	-	drowsy	↓↓	7200	LVH	acute rt MCA territory infarct with hypertensive ischaemic changes	3	0	-1.5	I	I	I	I
19	764259	55	M	+	-	-	+	-	-	-	-	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	+	110	60	S1S2+	nvbs	nad	15	-	alert	↑↓	11400	WNL	s/a infarct of lt frontotempero parietal region (lt mca)	0	1.5	-6	I	I	H	I
20	765185	48	M	+	+	-	+	-	-	-	-	-	-	-	-	-	-	-	-	+	-	+	-	-	+	-	-	100	76	S1S2+	nvbs	nad	15	-	alert	↓↑	16800	old ihd	s/a infarct in rt MCA territory	4	0	-7.4	I	E	I	I
21	779524	72	F	-	-	-	+	-	-	-	-	-	-	+	-	-	-	-	-	+	-	-	-	-	-	-	-	160	90	S1S2+	nvbs	nad	7	-	drowsy	↓↑	8600	WNL	rt ICA territory infarct	3	1.5	-2	I	I	H	I
22	788233	58	M	-	-	-	-	-	-	-	-	-	-	-	+	+	-	-	+	-	-	-	-	-	+	-	-	170	100	S1S2+	nvbs	nad	5	-	coma	↑↑	9100	WNL	rt intra parenchymal Hg with perilesional edema in pons & midbrain. HTN related changes	9	7.5	2.5	H	E	H	H
23	718253	35	M	-	-	-	+	+	-	-	-	-	-	-	-	-	+	+	-	+	-	-	-	-	-	-	+	220	110	S1S2+	nvbs	nad	15	+	alert	↓↑	16900	WNL	intraparenchymal Hg in the rt lentiform nucleus, thalamus, corona radiata and post limb of int capsule	14	6.5	3	H	H	H	H
24	721419	36	M	+	-	-	+	-	-	-	-	-	-	-	+	-	-	-	-	-	-	-	-	-	+	-	+	180	110	S1S2+	nvbs	nad	8	-	stupor	↑↑	13500	WNL	s/a infarct in b/l MCA territory	7	6.5	1.5	I	E	H	H
25	768968	68	F	-	-	-	+	-	+	-	-	-	-	-	+	-	-	-	-	+	+	-	-	-	-	-	-	220	120	S1S2+	nvbs	nad	10	-	drowsy	↓↑	13300	WNL	intraparenchymal Hg with perilesional edema in Lt frontal, temporal region & corona radiata, htn changes	7	3	-0.5	H	E	H	E
26	778607	57	M	-	-	-	+	-	-	-	-	-	-	-	-	-	+	-	-	-	-	+	-	-	-	-	-	140	90	S1S2+	nvbs	nad	15	-	alert	↓↓	10700	WNL	acute infarct in lt MCA territory	0	1.5	-6	I	I	H	I
27	777804	70	F	+	-	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	-	-	-	-	184	100	S1S2+	nvbs	nad	13	-	alert	↓↑	8800	WNL	acute intraparenchymal bleed in Rt thalamus and gangliocapsular region	0	4	2	H	I	H	H
28	714680	52	M	+	-	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	+	-	-	-	-	140	90	S1S2+	nvbs	nad	15	-	alert	↓↓	18000	WNL	acute left corona radiata and putamen infarct	4	-1.5	-3	I	E	I	I
29	771832	45	M	+	-	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	+	140	90	S1S2+	nvbs	nad	15	-	alert	↑↓	10800	WNL	s/a infarct in left MCA + PCA territory	0	3.5	-3	I	I	H	I
30	775026	60	F	-	-	-	+	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	-	-	-	-	-	170	90	S1S2+	nvbs	nad	15	-	alert	↑↓	10800	WNL	s/a infarct in the left ACA territory	0	1.5	-6	I	I	H	I
31	710776	58	M	-	-	-	+	-	-	-	-	-	+	-	-	-	-	-	-	+	+	+	-	-	-	-	+	160	90	S1S2+	nvbs	nad	11	-	drowsy	↓↑	13500	old ihd	rt malignant MCA territory infarct with haemorrhagic transformation	7	1.5	-3.5	I	E	H	I
32	719080	56	M	+	-	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	+	+	-	-	-	-	-	100	70	S1S2+	nvbs	nad	15	-	alert	↓↑	6400	old ihd	acute infarct in the right MCA territory	0	1.5	-8	I	I	H	I
33	718927	68	M	-	-	-	+	-	-	-	+	-	-	-	-	+	+	+	-	-	+	-	-	-	-	-	-	160	100	S1S2+	nvbs	nad	13	+	drowsy	↑↓	9600	LVH	lt intraparenchymal Hg with intraventricular extension with midline shift	13	4.5	4.5	H	H	H	H
34	710851	55	M	+	-	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	110	70	S1S2+	nvbs	nad	15	-	alert	↓↓	10000	WNL	lacunar infarct in left posterior putamen	0	0	-3	I	I	I	I
35	726506	62	F	+	-	-	+	-	-	-	-	-	+	-	-	-	-	-	-	-	+	-	-	-	-	-	-	140	80	AF	nvbs	nad	15	-	alert	↓↓	6700	AF	s/a infarct in rt MCA territory. HTN ischaemic changes	0	-2.5	-4	I	I	I	I
36	730439	45	M	-	-	-	+	-	-	-	-	-	-	-	-	+	+	+	-	-	-	-	-	-	+	-	-	136	86	S1S2+	nvbs	nad	8	+	drowsy	↑↓	9800	WNL	intra-parenchymal Hg with perilesional edema in lentiform nucleues, post limb of int capsule with midline shift	13	4.5	6.5	H	H	H	H
37	747233	45	M	-	-	-	+	-	-	-	-	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	+	120	70	S1S2+	nvbs	nad	12	-	alert	↓↓	10500	WNL	s/a infarct in lt parietal region	0	2	-5	I	I	H	I
38	745068	65	M	+	-	-	+	-	-	-	-	-	-	-	-	-	-	-	-	+	+	-	-	-	+	-	-	160	100	S1S2+	nvbs	nad	15	-	alert	↑↓	6600	LVH	s/a infarct in rt MCA and PCA territory. Hypertension ischaemic changes	0	0	-5	I	I	I	I
39	749720	65	M	-	-	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	+	150	80	S1S2+	nvbs	nad	15	-	alert	↑↓	14200	LVH	infarct in the left MCA, PCA territory. Lt Carotid atheroma	4	3.5	-7	I	E	H	I
40	735103	72	M	-	-	+	+	-	-	-	-	-	-	+	-	-	-	-	-	+	+	-	+	-	-	-	-	150	90	S1S2+	nvbs	nad	15	-	alert	↓↓	10800	WNL	acute infarct in the Lt PCA territory with HTN changes	0	-1.5	-6	I	I	I	I
41	747471	55	M	-	-	-	+	-	-	-	-	-	+	-	-	-	-	-	-	-	+	-	-	-	-	-	-	140	90	AF	nvbs	nad	15	-	alert	↑↓	10000	AF	s/a infarct in lt PCA & MCA territory	0	-1	-3	I	I	I	I
42	746438	62	M	-	-	-	+	+	-	-	-	-	-	-	-	-	-	-	-	-	+	+	-	-	-	-	+	200	100	S1S2+	nvbs	nad	15	-	alert	↓↓	8500	WNL	infarct in the left post limb of internal capsule	0	2	-2	I	I	H	I
43	738398	55	F	-	-	-	+	-	+	-	-	-	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	100	70	AF	nvbs	nad	10	-	drowsy	↑↓	17400	AF	s/a infarct in lt PCA & MCA territory with midline shift	7	-1	-2.5	I	E	I	I
44	739794	80	M	-	-	-	+	-	-	-	-	-	-	-	+	-	-	-	-	-	+	-	-	-	-	-	-	220	110	S1S2+	nvbs	nad	11	+	alert	↑↑	15200	WNL	intraparenchymal Hg with perilesional edema in left temporal region	17	7.5	5.5	H	H	H	H
45	742983	50	F	-	-	-	+	-	+	+	-	+	-	-	-	-	-	-	-	-	+	-	-	-	-	-	-	130	80	S1S2+	nvbs	nad	13	-	alert	↑↓	9700	WNL	s/a infarct in the left putamen, caudate nucleus, corona radiata and centrum semiovale	0	1.5	-4	I	I	H	I
46	713078	33	M	+	-	-	+	-	-	-	-	-	+	-	-	-	+	+	-	+	-	-	-	-	+	-	+	180	110	S1S2+	nvbs	nad	10	-	drowsy	↑↑	10200	WNL	Lt intraparenchymal Hg with midline shift	4	9.5	3	H	E	H	H

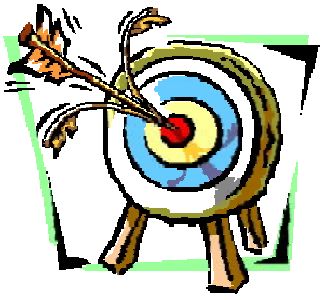
ANNEXURE III - MASTER CHART

Serial Number	In patient number	Age (Years)	Sex	Presenting complaints																Past History										Examination																	TYPE OF STROKE			
																														BP		Systemic														IMAGING REPORT	GREEK SCORE REPORT	BESSON SCORE REPORT	SIRIRAJ SCORE REPORT
				Dysarthria	dysphagia	visual loss	Weakness	Tingling	Global aphasia	Giddiness	Slumping	Motor aphasia	Deviation angle of mouth	Altered sensorium	Drowsiness	Unconscious	headache	vomiting	Diabetes mellitus	Hypertension	Coronary artery disease/Angina	dyslipidemia	intermittant claudication	TIA	tobbaco chewer	Smoking	Alcohol consumption	Systolic (mm Hg)	Diastolic (mm Hg)	Cardiovascular system	Respiratory system	Per abdomen	GCS	Worsening of consciousness after 3 hrs	Level of consciousness	plantars	total leukocyte count	ecg	MRI	greek score	besson score	siriraj score				
47	711759	75	M	-	-	-	+	-	-	+	-	-	-	-	-	-	+	+	+	-	-	-	-	-	-	-	180	100	S1S2+	nvbs	nad	15	-	alert	↓↓	15800	WNL	acute intraparenchymal Hg in left cerebellar lobe	8	3	2	**H**	E	H	H
48	716125	67	M	-	-	-	+	-	-	+	-	-	+	-	-	-	+	-	-	+	-	-	-	-	-	+	140	90	S1S2+	nvbs	nad	15	-	alert	↓↓	14300	WNL	acute left pontine infarct	8	3	-1	**I**	E	H	E
49	733034	61	F	+	-	-	+	-	-	+	-	-	-	-	-	-	-	-	+	-	-	-	-	-	-	-	170	100	S1S2+	nvbs	nad	14	-	alert	↓↓	8600	WNL	hyperacute inraparenchymal Hg in the rt lentiform nucleus and corona radiate	0	0	-2	**H**	I	I	I
50	753929	62	M	+	-	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	-	-	130	80	S1S2+	nvbs	nad	11	-	alert	↓↓	7200	WNL	s/a infarct in the left MCA territory	0	-1.5	-12	**I**	I	I	I
51	764487	62	M	-	-	-	+	-	-	+	-	-	-	-	-	-	+	-	-	+	-	-	-	-	-	-	190	120	S1S2+	nvbs	nad	15	-	alert	↓↓	10600	WNL	hyperacute bleed in left high fronto parietal region with perilesional edema	0	3	2	**H**	I	H	H
52	764777	37	M	-	-	-	+	-	-	-	+	-	-	-	-	-	-	-	-	+	-	-	-	+	-	+	200	110	S1S2+	nvbs	nad	7	+	stupor	↓↑	15600	LVH	intraparenchymal Hg in rt parietotemporal and thalamoganglionic region with midline shift	13	3.5	1.5	**H**	H	H	H
53	709804	55	F	-	-	-	+	-	+	-	-	-	-	-	-	-	-	+	-	+	-	-	-	-	-	-	170	120	S1S2+	nvbs	nad	7	-	drowsy	↑↓	12100	WNL	intraparenchymal Hg in lt thalamus & int capsule (genu) with intraventricular extension	11	1.5	4.5	**H**	H	H	H
54	713418	60	M	-	-	-	+	-	-	-	-	-	-	-	-	-	-	+	-	+	-	-	-	-	+	-	190	110	S1S2+	nvbs	nad	14	-	alert	↓↑	22500	WNL	acute hematoma in the rt thalamocasule region with intra ventricular extension and midline shift	4	1.5	1	**H**	E	H	E
55	774493	65	F	-	-	-	+	-	-	-	-	-	-	-	-	-	-	+	+	+	-	-	-	-	-	-	140	90	S1S2+	nvbs	nad	15	-	alert	↓↑	6200	WNL	acute infarct in the right corona radiata and post limb of internal capsule	0	1.5	9	**I**	I	H	H
56	721577	70	M	-	-	-	+	-	-	-	-	-	-	-	+	-	-	-	-	-	+	-	-	-	-	-	160	90	AF	nvbs	nad	10	-	drowsy	↓↑	14900	AF	rt MCA territory infarct with old left PCA territory infarct	4	-1	-3	**I**	E	I	I
57	716594	79	M	-	-	-	+	-	+	-	-	-	-	-	-	-	-	-	+	-	-	-	+	-	-	-	180	90	S1S2+	nvbs	nad	15	-	alert	↓↓	4700	WNL	embolic infarct in Lt MCA territory	0	-5	-3	**I**	I	I	I
58	716905	65	F	-	-	-	+	-	+	-	-	-	-	-	-	-	-	-	+	+	+	-	-	-	-	-	150	90	S1S2+	nvbs	nad	10	-	alert	↑↑	14000	WNL	acute left MCA territory infarct	4	4.5	-3	**I**	E	H	I
59	734184	40	M	-	-	-	+	-	-	-	+	-	-	-	-	+	+	+	-	+	-	-	-	-	-	+	210	120	S1S2+	nvbs	nad	13	+	drowsy	↑↑	16500	LVH	intraparenchymal Hg in fronto parietotemperal region, corona radiata, int capsule and lentoform nucleus	17	7.5	4	**H**	H	H	H
60	739193	60	F	-	-	-	+	-	-	-	-	+	-	-	-	+	-	-	-	+	-	+	-	-	-	-	140	90	S1S2+	nvbs	nad	8	-	stupor	↓↓	13300	WNL	left MCA territory infarct, gliosis in the left PCA territory	7	-1.5	-1.5	**I**	E	I	I
61	748461	60	M	-	-	-	+	-	+	-	-	-	-	-	+	-	+	-	-	-	+	-	-	-	-	-	140	90	S1S2+	nvbs	nad	8	-	drowsy	↑↓	8300	old ihd	S/A Infarct in the left frontoparietal region, insular cortex & internal capsule	7	1.5	-1.5	**I**	E	H	I

ANNEXURE IV – KEY TO MASTER CHART

A	-	Absent
ACA	-	Anterior cerebral artery
AF	-	Atrial fibrillation
E	-	Equivocal
ECG	-	Electrocardiogram
F	-	Female
GCS	-	Glasgow Coma scale
H	-	Hemorrhage
Hg	-	Hemorrhage
HTN	-	Hypertension
I	-	Ischemic
IHD	-	ischemic heart disease
Lt	-	Left
M	-	Male
MCA	-	Middle cerebral artery
NAD	-	No abnormality detected
NVBS	-	normal vesicular breath sound
PCA	-	Posterior cerebral artery
RBBB	-	right bundle branch block
Rt	-	Right
S/A	-	sub acute
WNL	-	Within normal Limits

Introduction



Objectives

Review of Literature

Methodology

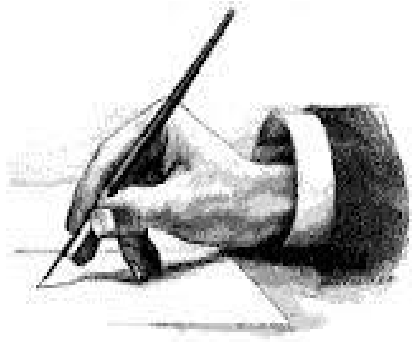
Results

Discussion

Conclusion

Summary

Bibliography



Annexure-I

Annexure-II

Annexure-III

Annexure-IV