A Dissertation on

A COMPARATIVE AND VALIDITY STUDY OF ALLEN, SIRIRAJ AND GREEK SCORE IN DIFFERENTIATION OF ACUTE ISCHEMIC AND HEMORRHAGIC STROKE

Submitted to

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CHENNAI – 600032.

In partial fulfillment of the Regulations
for the Award of the Degree of

M.D. BRANCH - I

GENERAL MEDICINE

DEPARTMENT OF GENERAL MEDICINE
STANLEY MEDICAL COLLEGE
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CERTIFICATE

This is to certify that **Dr. M.ARIVUMANI,** Post -Graduate Student (MAY 2010 TO APRIL 2013) in the Department of General Medicine STANLEY MEDICAL COLLEGE, Chennai- 600 001, has done this dissertation on **"A COMPARATIVE AND VALIDITY STUDY OF ALLEN, SIRIRAJ AND GREEK SCORES IN DIFFERENTIATION OF ACUTE ISCHEMIC AND HEMORRHAGIC STROKE"** under my guidance and supervision in partial fulfillment of the regulations laid down by the Tamilnadu Dr.M.G.R. Medical University, Chennai, for M.D. (General Medicine), Degree Examination to be held in April 2013.

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DECLARATION

I **Dr.M.ARIVUMANI** declare that I carried out this work on "**A COMPARATIVE AND VALIDITY STUDY OF ALLEN, SIRIRAJ AND GREEK SCORE IN DIFFERENTIATION OF ACUTE ISCHEMIC AND HEMORRHAGIC STROKE**" at Department of Medicine, Government Stanley Medical college Hospital during the period of March 2012 to October 2012. I also declare that this bonafide work or a part of this work was not submitted by me or any other for any award, degree, and diploma to any other university, board either in India or abroad.

This is submitted to The Tamilnadu Dr.M.G.R. Medical University, Chennai in partial fulfillment of the rules and regulation for the M. D. Degree examination in General Medicine.

DR.M.ARIVUMANI

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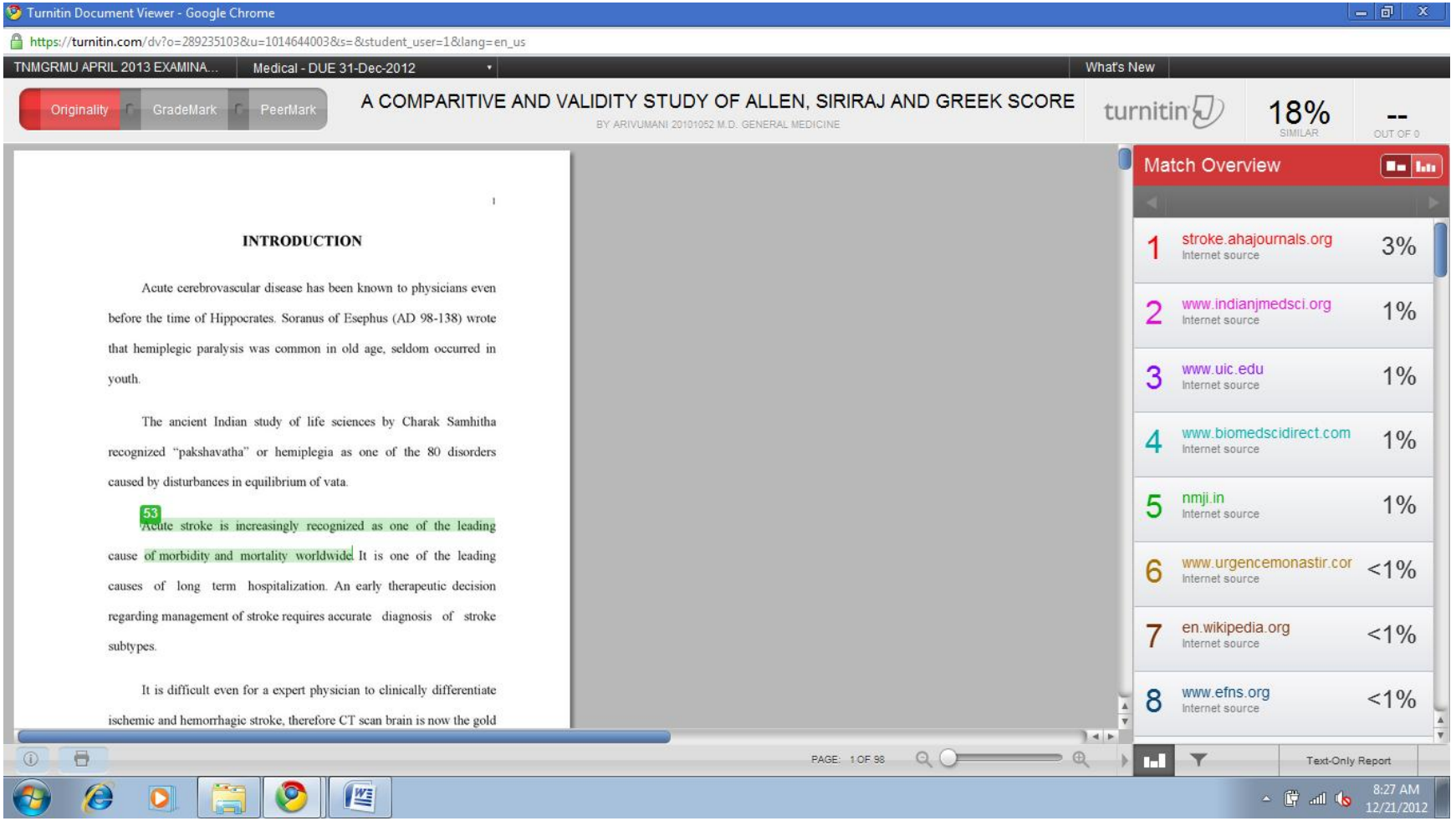
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53
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It is difficult even for a expert physician to clinically differentiate ischemic and hemorrhagic stroke, therefore CT scan brain is now the gold
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INTRODUCTION

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Acute stroke is increasingly recognized as one of the leading cause of morbidity and mortality worldwide. It is one of the leading causes of long term hospitalization. An early therapeutic decision regarding management of stroke requires accurate diagnosis of stroke subtypes.

It is difficult even for a expert physician to clinically differentiate ischemic and hemorrhagic stroke, therefore CT scan brain is now the gold standard tool for diagnosis of stroke subtypes. Time taken to obtain a CT scan brain delays treatment, therefore prehospital screening score systems can be used to differentiate ischemic and hemorrhagic stroke.

Many stroke scoring systems have been devised to clinically differentiate ischemic and hemorrhagic stroke in centers with limited brain imaging facilities.

The ALLEN, SIRIRAJ and GREEK stroke scores are the main existing model scores devised to differentiate clinically between hemorrhagic and ischemic stroke.

REVIEW OF LITERATURE

Stroke is defined as rapidly developing focal or global disturbance of cerebral function lasting for 24 hours or longer with no apparent cause other than vascular origin.[1] It is due to inadequate blood flow to brain either due to block of a artery by a thrombus or emboli, rupture of a blood vessel, altered permeability of a vessel wall and increased viscosity. This leads to inadequate supply of nutrients and oxygen to the brain.

Cerebrovascular vascular disease is a preventable and treatable condition. Stroke is the major cause of morbidity in the developing world.

ANATOMY OF CEREBRAL CIRCULATION

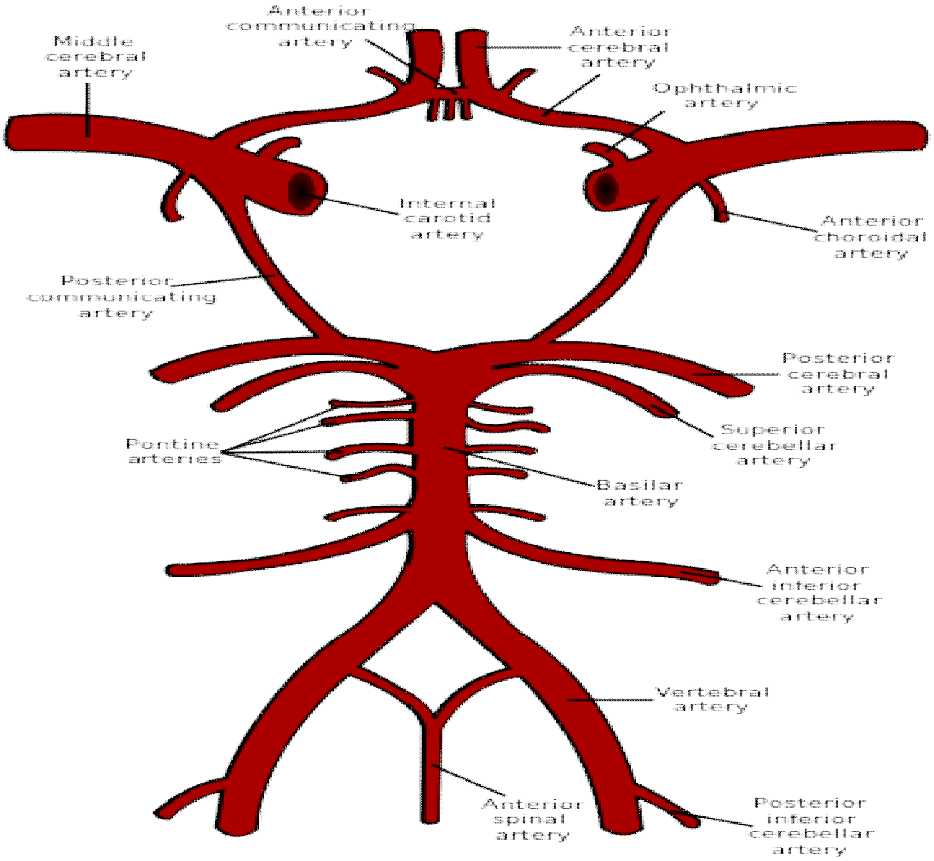
The blood supply of brain is by anterior and posterior circulation. Anterior circulation is by the branches of right and left internal carotid arteries.

The brain and the surrounding structures are supplied by a circle of arteries named circle of Willis named after Thomas Willis an English physician.[2] The circle of Willis is part of cerebral circulation and formed by following arteries on both sides[2] namely,

anterior cerebral artery, anterior communicating artery, posterior cerebral artery, posterior communicating arteries, internal carotid arteries.

The basilar artery divides into right and left posterior cerebral artery which is connected to internal carotid artery via posterior communicating artery. The internal carotid artery gives rise to right and left anterior cerebral artery which are interconnected by anterior communicating artery.

CIRCLE OF WILLIS



Middle cerebral artery and Basilar artery are also part of circle of Willis. [3]

Anterior cerebral artery

The anterior cerebral arteries arise from the internal carotid artery on each side and is part of circle of Willis. Anterior cerebral arteries supplies medial aspect of frontal lobe and superomedial aspect of parietal lobe , anterior four fifths of corpus callosum , anterior portion of internal capsule , basal ganglia and olfactory tract . Anterior cerebral artery is divided into five segments. [4]

- A1 segment – This segment extends from internal carotid artery to anterior communicating artery .The branches of this segment supplies caudate nucleus and anterior portion of internal capsule, anterior hypothalamus and anterior perforated substance.

- A2 segment – This segment extends from anterior communicating artery to the bifurcation forming callosomarginal arteries and pericallosal arteries. The distal medial striate artery also called as recurrent artery of

Heubner is a branch of this segment which irrigates internal capsule .

- A3 segment- This segment also called as pericallosal artery is the terminal branch of anterior cerebral artery .

- A4 and A5 segments are the smaller branches of callosal arteries. These segments called as callosal marginal arteries are found only in 60 % of individuals.[5] These vessels are seen angiographiclly only in 50% of individuals.[6]

Middle cerebral artery

Middle cerebral artery is the paired artery and it is the major branch of internal carotid artery supplying brain. Middle cerebral artery is divided into four segments. [7]

- M 1 segment- Also called as sphenoid segment give rise to lenticostriate branches which supplies posterior limb of internal capsule, caudate nucleus , corona radiata , outer globus pallidus .

- M 2 segment also called as sylvian segment is divided into superior and inferior divisions supplying inferior parietal and temporal lobes .

- M 3 segment or opercula segment is sometimes grouped under M2 segment.

- M 4 segment is the terminal segment supplies cortex.

Middle cerebral artery supplies major aspect of lateral aspect of brain except superior inch of frontal and parietal lobe.

Superior division supplies inferior frontal lobe (Broca's area) , inferior division supplies lateral temporal lobe (Wernicke's area) and deep branches supplies basal ganglia and internal capsule.

Posterior cerebral artery

Posterior cerebral artery is the terminal branch of basilar artery and it's a paired artery . It supplies occipital lobe, posteromedial portion of temporal lobe. Posterior cerebral artery is divided into four segments.

- P1 segment extends from its origin to posterior communicating artery.

- P2 segment is from the posterior communicating artery around midbrain which is divided into anterior and posterior sub segments.
- P3 segment is quadrigeminal segment and
- P 4 segment is cortical segment. [8]

Vertebrobasilar system

Vertebral artery arises from the first part of subclavian artery on either side and ascends up via transverse foramen of upper six cervical vertebras and foramen magnum. At lower border of Pons it meets the opposite side vertebral artery and forms basilar artery. The various branches of Vertebrobasilar system in cranium are

- Meningeal arteries
- Anterior and posterior spinal arteries
- Posterior inferior cerebellar arteries- It supplies a wedge shaped area in lateral aspect of medulla which contains descending tract of trigeminal nerve, lateral spinothalamic tract, lateral vestibular nucleus ,IX and X cranial nerve nuclei, descending cervical sympathetic tract and inferior cerebellar peduncle.

- Anterior inferior cerebellar artery- supplies Pons, lateral part of tegmentum of midbrain, anterior part of lower cerebellum.

- Superior cerebellar artery- gives branches to colliculi, cerebral peduncle and end in supplying vermis and lateral aspect of lobes of cerebellum.

- Pontine and paramedian branches, short and long circumferential arteries which supplies upper medulla and anterior part of Pons.

- Posterior cerebral arteries- arise from basilar artery winds round the cerebral peduncle and reaches calcarine sulcus of occipital lobe.

Venous system of brain

Venous drainage of brain is by numerous small veins which ultimately drain into venous sinuses which lays in between two layers of duramater. They open directly or indirectly into internal jugular vein.

The veins draining are divided into superficial and deep veins. The superficial veins are again divided into superior and inferior group. The superior group drains upper half of the brain into superior sagittal sinus and the inferior group drains into venous sinus at the base of the brain.

Great vein of Galen belongs to deep venous system and it drains basal ganglia and choroid plexus of third and fourth ventricle. The diploic veins in between the skull bones drain either into sinuses or superficial veins of scalp.

CLASSIFICATION OF STROKE

Classification of stroke is based on the pathogenesis and its shown below: [9]

1. Cerebral infarction
   - Athero embolic
   - Embolic
   - Lacunar
   - Undetermined etiology
2. Intracranial hemorrhage
3. Sub arachnoid hemorrhage
4. Others unspecified

FREQUENCY OF STROKE SUBTYPES

Harvard cooperative Stroke registry tool showed the following frequency of stroke subtypes among 649 patients admitted with stroke. [10]

Cerebral thrombosis……53%

Cerebral embolism……...31%

Cerebral hemorrhage……..10%

Sub arachnoid hemorrhage…...06%

Framingham study done by a neurologist by in hospital assessment showed the following stroke subtypes. [11]

Atherothrombotic infarction……44%

Transient ischemic attacks……21%

Cerebral embolism…….....21%

Intracranial hemorrhage……..…12%

Intracerebral hemorrhage……...5.1%

Subarachnoid hemorrhage….....6.6%

Others ……2%

Many Studies done in our continent showed that percentage of hemorrhagic stroke is greater in Asian population than other population. [12] But the overall incidence of Atherothrombotic stroke secondary to large vessel atherosclerosis and Lacunar infarction were the most common stroke subtypes.

Prompt and early diagnosis of cause of stroke is very essential, since the management of acute ischemic and acute hemorrhagic stroke is entirely different.[13]

EPIDEMIOLOGY OF STROKE IN INDIA

It is difficult to ascertain the exact incidence, prevalence, morbidity, mortality rates in a developing countries like ours with more than one billion population.

Most of the published data are from retrospective studies done by analysis of patients admitted to the urban/tertiary care hospital where as most of our population lives in villages and small towns.

Data analysis of the records collected from major hospitals suggest that 2 % of total admissions in hospital, 4.5% of admissions in neurology wards are due to cerebrovascular disease and incidence of stroke in young (below 40 years) is high 13.32% as compared to western data. [14]

The data from semi urban areas of Vellore shows prevalence rate of 56.9 per 100,000 population,[15] where as higher rate of 143 per 100,000 population has been reported by Razdan et al[16] from Anantnag district of South Kashmir.

RISK FACTORS FOR STROKE

Based on the findings from different studies the risk factors for cerebrovascular accident in India are as follows .

Ischemic stroke

Age, sex, race

Hypertension

Diabetes mellitus

Smoking

Alcoholism

Hyperlipidemia

Heart disease

Obesity

Oral contraceptives

Family history of stroke

Snoring

Hypercoagulable states

Hemorrhagic stroke

Hypertension

Aneurysm

Arterio venous malformation

Coagulopathy

Drugs- cocaine, amphetamine

Risk factors include modifiable and non modifiable risk factors.

Non modifiable risk factors

Non modifiable risk factors include age, race, ethnicity and heredity. Age is the most important risk factor, for every 10 years after 55 years stroke risk doubles in both men and women.[17]

Framingham study showed that paternal and maternal history of stroke caries high risk in off springs. [18]

Black races have more risk than white races. [19]

Modifiable risk factors for ischemic stroke

Hypertension is an one of the important modifiable risk factor for ischemic stroke. The prevalence of ischemic stroke in hypertensive patients (BP > 140/90mmHg as per JNC definition) is 45% at 50 years of age, 60% at 60 years of age, and 70 % at 70 years of age.[20]

Cardiac diseases are one of the important risk factor. Atrial fibrillation is one of the important treatable risk factor for ischemic

stroke. Above the age of 55 years, incidence of Atrial fibrillation doubles for every successive 10 years. [21]

In stroke prevention in Atrial fibrillation trials (SPAF) aspirin 325mg or warfarin is used in the prevention of Atrial fibrillation in stroke patients. Aspirin has been shown to decrease the risk of non cardio embolic stroke but does not prevent embolic stroke from cardiac source. [22, 23]

The current recommendation is to use Warfarin for high risk candidates and reserve aspirin for low risk candidates for stroke prevention in Atrial fibrillation. [24]

Valvular heart disease particularly mitral stenosis and myocardial disease are also important risk factors for stroke .

Diabetes mellitus is an important independent risk factor, those with diabetes has twice higher risk for thromboembolic stroke.[25] Diabetic patients are more prone to premature atherosclerosis and they are also more prone to get coexistent obesity, hypertension, dyslipidemia.

There is two times higher risk for ischemic stroke in smokers when compared to nonsmokers. [26]

Alcohol when consumed in moderate amounts reduce cerebrovascular disease , however increased alcohol consumption is associated with risk of Intracerebral hemorrhage. [27]

Life style factors like physical inactivity , diet , obesity is associated with increased incidence of stroke.

PATHOPHYSIOLOGY OF STROKE

The main mechanisms causing brain injury in stroke are ischemia and hemorrhage.

Pathophysiology of ischemic stroke

Glucose is the major substrate of energy for brain. But as the brain does not store it, the effects of ischemia are rapid. [28]

The anterior circulation ischemic stroke could be due to occlusion of a major intracranial artery or a small penetrating artery. The ischemic stroke can again be either due to

1. Emboli from atherosclerotic narrowing at the bifurcation of common carotid, cardiac source or from a atheroma of arch of aorta .

2. Combination of atherosclerotic stenosis and superimposed thrombosis. Lipohyalinosis of small penetrating arteries cause Lacunar stroke.

The extent of ischemic injury and progress depends on many factors[29-32.]

1. Duration of ischemia- brain tolerates short duration ischemia better
2. Collateral circulation
3. Systemic blood pressure – hypotension causes global ischemia
4. Hypercoagulable states increases progression of ischemia
5. Elevated body temperature associated with ischemic injury
6. Hypoglycemia and hyperglycemia adversely affects the ischemia.

Ischemic penumbra- after a ischemic insult the core of infracted tissue is surrounded by a oligemic zone called ischemic penumbra.[33] The critical time during which this oligemic zone can be prevented from infarction by reperfusion therapy is “window of opportunity” . The neuronal death occurs by two process called coagulative necrosis and apoptosis.

Pathophysiology of hemorrhagic stroke

Hypertension and atherosclerosis interact in many ways. Atherosclerosis reduces the resilience of the arterial wall and induces hypertension, hypertension in turn worsens atherosclerosis.

In hemorrhage blood usually leaks into brain parenchyma, ventricles or subarachnoid space. The exact nature of vascular lesion which causes rupture of vessel wall is not known. But it appears that hemorrhage occur due to effect of hypertension on arterial walls.

Fisher considered the primary pathological mechanism of hemorrhage was due to Lipohyalinosis and fibrinoid necrosis of vessel wall.[34, 35] Cerebral amyloid angiopathy has been recently postulated as mechanism of hemorrhage in normotensive elderly patients. [36]

Hypertension is the main causal factor for putamen and thalamic hemorrhage which are due to rupture small penetrating vessels of size 50-150microns in diameter. [37, 38] An association of 72% and 81% has been reported between hypertension and Intracerebral hemorrhage. [39] Local hematoma formations destroy parenchyma, displace adjacent structures and dissect them. Brain edema occurs due to mass effect which is the major cause for mortality.

Others causes of hemorrhage include

Rupture of secular aneurysm and arteriovenous malformations

Anticoagulation induced bleed

Tumor bleed

Blood dyscrasias

The common sites of hypertensive hemorrhage are as follows

Putamen and internal capsule……..35-50%

Lobar …….....30%

Thalamus ……..10-15%

Cerebellar……15%

Pons …….5-10%

Caudate nucleus……5%

CLINICAL FEATURES OF STROKE

Clinical features of Atherothrombotic stroke

Ischemic stroke can be either thrombotic or embolic. The main episode of stroke is preceded by episodes of transient ischemic attacks which indicate evolving nature of thrombus.

Transient ischemic attack is defined as the focal neurological defect lasting for less than 24 hours. Having developed a episode of TIA is a risk factor for developing stroke.[40] 20 % of ischemic stroke that

occurs after first episode of TIA occurs within first month and 50% develops stroke within one year.

Reversible ischemic neurological deficit (RIND) is defined as the signs of ischemia lasting for more than 24 hours but less than 72 hours.

Prolonged reversible ischemic neurological deficit (PRIND) is defined as the signs of ischemia lasting more than 24 hours but less than 7 days.

Stroke in evolution is the type of stroke that progresses or fluctuates while the patient is under observation. Completed stroke is the one in which no progression or deterioration occurs beyond 72 hours.

Thrombotic stroke usually have onset during sleep or while getting up from sleep. Embolic stroke causes fluctuating neurological deficits due to the embolic being migrating or lysing or disappearing. [41, 42]

Headache, vomiting and loss of consciousness are less frequent in ischemic stroke. There is no neck stiffness. The resulting neurological deficit in ischemic stroke usually corresponds to the vascular territory involved.

The most commonly involved artery is the middle cerebral artery producing complete or partial motor hemi deficit, hemianopia, hemianaesthesia. Speech is affected if dominant hemisphere is involved.

In most cases of embolic stroke the source of emboli is cardiac, the most common cause being atrial fibrillation due to atherosclerotic, rheumatic or ischemic heart disease. [43]

Clinical features of embolic stroke

Embolic stroke develops rapidly like a bolt out of blue. Full blown picture develops within minutes or seconds. Embolic stroke occurs during activity.

Clinical features of hemorrhagic stroke

The onset of hemorrhagic stroke is dramatic. It usually occurs while patient is awake and active and rarely during sleep. [44] It is associated with headache and vomiting at the onset. Patients usually have depressed level of consciousness or in coma.

NATURAL COURSE OF STROKE

In developed countries the average age of onset of first episode of stroke or transient ischemic attack is 73 years. The

probability of first stroke is 1.6 per 1000 population and that of TIA is 0.42 per 1000 population. [45]

In less developed countries stroke occurs in little earlier age due to different population age structure. The mortality in stroke patients is highest in the first week and 20% to 50% of mortality occurs in first 30 days. The mortality depends on the age, stroke subtype , severity , co morbid illness .

Those who survive may improve with varying levels of disability. Spontaneous recovery occurs up to six months. [46] The risk of recurrence of stroke after the first episode is 10 percent in the first year and 5 percent per year thereafter. [47]

CLINICAL DIAGNOSIS OF STROKE

History and thorough clinical assessment is essential for diagnosis of stroke and for deciding treatment plans. The reliabilities of individual neurological findings can be improved by standardized scoring systems. [48] Studies have shown that history and physical examination can be used to distinguish ischemic and hemorrhagic stroke. [49-51] Studies have also shown that chance of Intracerebral hemorrhage is

doubled if the patients has at least one of the following features on arrival. [50]

1. Coma on arrival
2. Severe headache
3. Vomiting
4. Systolic BP> 220 mmHg
5. Current warfarin therapy
6. Blood sugar > 160mg/dl in a non diabetic patient.

Diagnostic errors can still occur when decisions are taken solely based on clinical features. [52] Imaging is mandatory to distinguish ischemic or hemorrhagic stroke. [53]

The clinical accuracy of differentiating stroke from non-stroke has 95% sensitivity and 66 to 97% specificity.[54] However, the accuracy of clinical acumen decreases while sub typing the stroke which has 68% sensitivity and 67% specificity.

We have to be sure that there is no intracranial hemorrhage before initiating antiplatelet or anticoagulant therapy. Hemorrhagic stroke caries significant morbidity and mortality if not promptly diagnosed and treated.

Ischemic stroke should also be diagnosed early and treatment should be initiated as soon as possible to benefit from early thrombolytic and antiplatelet therapy. [55]

IMAGING IN STROKE

Triage of patients for appropriate treatment has improved with imaging techniques.[56, 57.] The imaging techniques addresses four important apects

1. Whether hemorrhage is present
2. Whether there is intra-arterial thrombus
3. Identifies the size of the core of irreversibly infarcted tissue
4. Size of hypoperfused tissue that is at risk of reinfarction.[58-60]

There are myriad of imaging techniques available now which includes CT scan brain, MRI brain.

IMAGING IN ISCHEMIC STROKE

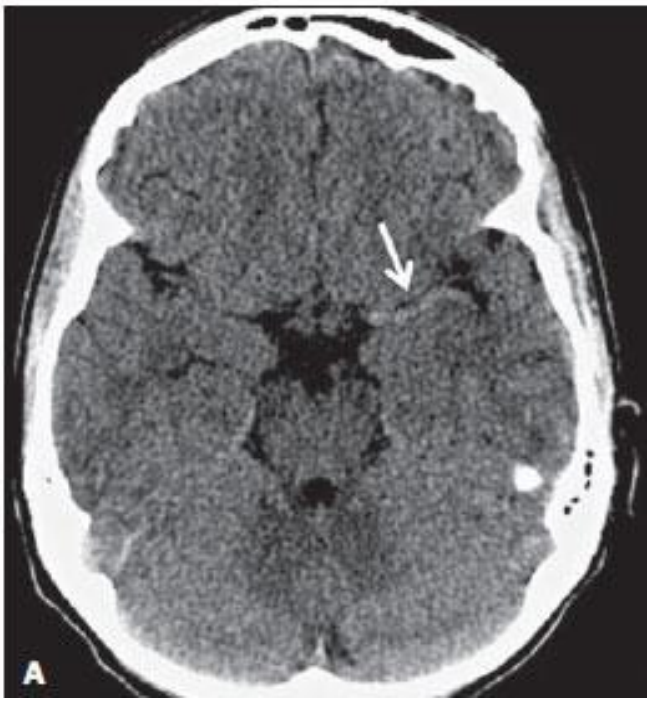
The imaging manifestations of ischemic stroke vary with time. More than 60% of CT scan obtained within few hours after cerebral infarctions are normal. There are few early signs in CT scan brain that can be identifies in less than 6 hours. They are

1. MCA dot sign

2. Insular ribbon sign

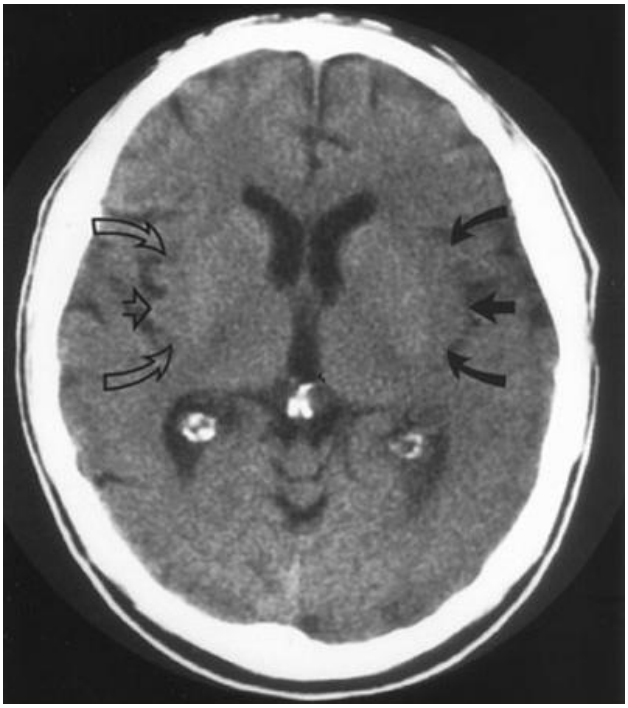
3. Obscuration of lentiform nucleus

MCA dot sign: MCA dot sign is one of the early sign of infarction in CT scan brain which is the punctate hyperdensity seen in sylvian fissure. It denotes thrombosis in M2 and M3 segment of middle cerebral artery. [61]



MCA DOT SIGN

Insular ribbon sign: At lateral margins of the insula, the white – gray matter interface is lost ("insular ribbon").



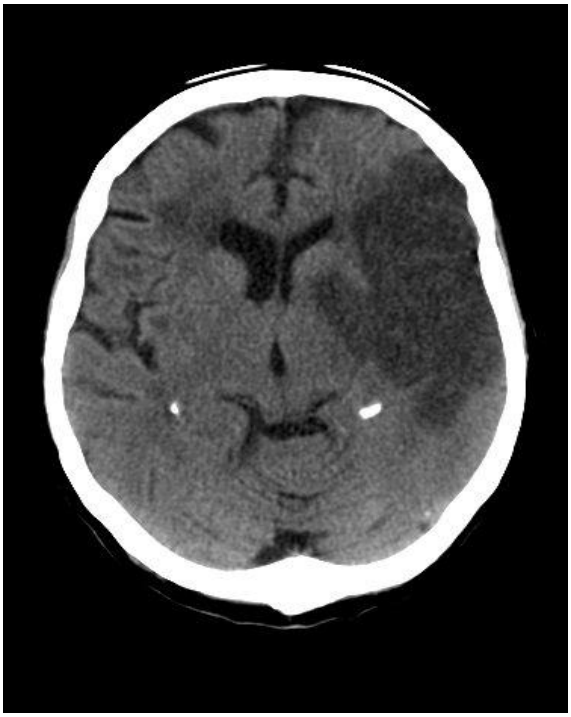
INSULAR RIBBON SIGN

Sub acute infarct

As ischemic stroke is a dynamic process, it evolves over time. After first 24-48 hours large vessel infarct appears on non enhanced CT scan brain. They appear as wedge shaped areas of decreased attenuation that involves both gray and white matter. Enhancement patterns are typically patchy or gyral which appears as early as 3-4 days and persists as long as 10 weeks.

Chronic infarct

Chronic infarct in a CT scan appears as well delineated encephalomalacic changes. Adjacent sulci become prominent with dilated ipsilateral ventricles. Rarely dystrophic calcification may be seen.



HYPODENSE LESION SHOWING INFARCT

MRI brain

MRI brain detects ischemic zones within a few hours of stroke before it becomes detectable in CT scan brain.MRI also reveals flow voids, hemosiderin deposition as well as alteration resulting from necrosis and gliosis.

The most sensitive and specific sequence in MRI brain for detecting infarct within minutes of its occurrence is diffusion weighted imaging (DWI).[62] MR perfusion imaging is done to differentiate viable and nonviable tissue.[63-65] Magnetic resonance angiography (MRA) is

useful in identifying the vascular occlusion. Gated recalled echo (GRE) sequence is to exclude haemorrhage.[66]

MR angiogram is used to delineate blood flow, vascular lesions including atheromatous plaque in carotid and Vertebrobasilar systems.

Ultrasound and Doppler flow studies

They are used to detect atheromatous plaque, stenosis of large vessels particularly carotid artery. [67]

IMAGING IN HEMORRHAGIC STROKE

CT scan brain

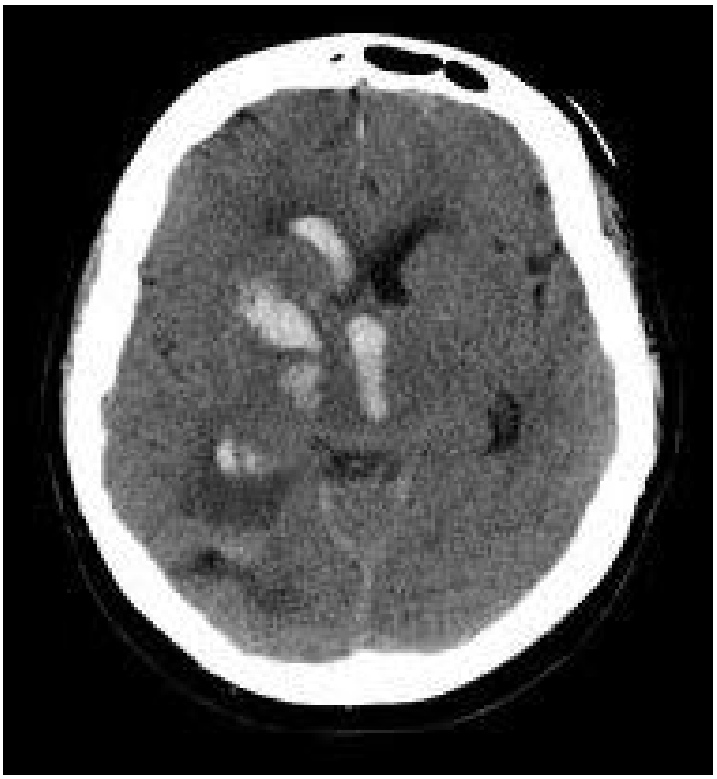
CT scan brain is the most sensitive test for diagnosing haemorrhage. It appears as high attenuation hypedense area with a hounsefield unit range of 40-60 HU. The increased density is due to increased hemoglobin protein ratio in relation to surrounding parenchyma. Therefore in severly anemic patients due to low hemoglobin haemorrhage may appear as isodense or hypodense. [68, 69]

Haemorrhage is visible in CT scan brain within three hours of onset as a hyperdense lesion surrounded by a peripheral rim of edema. The mass effect is proportional to the size of the edema. With time the

density decreases and the lesion becomes progressively hypodense due to breakdown of hemoglobin.

Contrast CT scan helps to delineate whether the bleed is tumor bleed or from arteriovenous malformation.

CT scan brain is sensitive in detecting haemorrhage of 1 to 1.5cm in cerebral cortex or cerebellum, however it may miss small haemorrhage in brainstem.



RIGHT CAPSULOGANGLIONIC HEMORRHAGE WITH INTRAVENTRICULAR EXTENSION

MRI brain

MRI brain is useful in detecting residual haemorrhage which remains visible for long time even though not detected by CT scan brain.[70] MR angioraphy is used to detect arteriovenous malformation or aneurysms.

PROGNOSIS

The prognosis of ischemic stroke depends on the size of the infarct. Large infarcts casuing cerebral edema with displacement of cerebral structures and tentorial herniation has the worst prognosis. Coma at the onset has worse prognosis. The mortality rate in small infarction is 3 to 6% where large basilar infarct associated with deep coma has mortality rate of 40%.[71]

Long term prognosis depends on age, comorbid illness, recurrence of event and severity of neurological deficit at onset.[72-74] Longer the recovery time poorer the prognosis. Recurrent stroke is common at same or another site particularly if the patient has diabetes milletus or hypertension.

Survival and recovery is better in embolic stroke when compared to thrombotic stroke.

In case of haemorrhagic stroke prognosis depends on size,site, spread of hematoma and cerebral edema. Hematoma larger than 50mm is associated with worse prognosis. Level of consciousness at onset is important indicator of prognosis. Intraventricular extension of haemorrhage per se has worst prognosis. Pontine , thalamic and putaminal haemorrhage has poor prognosis.

TREATMENT

Ischemic stroke

In first few days both cellular and vasogenic edema may threaten life which is managed with mannitol,dexamethasone and loop diuretics. Patient should be kept in horizontal position to maintain cerebral circulation.

Intravenous thrombolytic therapy is useful if the patient presents within three hours of onset of ischemic stroke satisfying strict neuroradiological exclusion criteria to prevent misdiagnosis.[75]

Heparin or heparinoids is useful when stroke is in progression. Antiplatelets drugs is useful in preventing thrombotic or embolic strokes.

Haemorrhagic stroke

The medical management of hemorrhagic stroke consists of adequate ventilation,maintenance of blood pressure, antiedema measures. Surgical removal of clot is life saving particularly in patients with lobar or cerebellar haematomas.

STROKE SCORES

Although the burden of stroke is increasing over years, the imaging facilities is limited in the rural areas and even in the available centers it is readily available round the clock in some public sectors. Several clinical scores are available to clinically differentiate acute ischemic and hemorrhagic stroke.

Three such scores are available to clinically differentiate ischemic and hemorrhagic stroke. They are

1. Allen or Guy's hospital score.
2. Siriraj score.
3. Greek score.

Several studies have been done to compare and validate the utility of these score in clinical practice. Each studies have shown different sensitivity and specificity patterns depending the population and races studied.

Allen and Siriraj stroke scoring systems were the previously existing scores derived from logistic regression technique.[76,77] The comparison Allen or Guy's hospital score and Siriraj scores have been done in many studies. These validation suggest that Siriraj stroke score is

better than the Allen or Guys hospital stroke score. They also concluded that sensitivity of the Siriraj stroke score is 80 - 90%.[78, 79]

However Wadhwani Jyoti et al in their study concluded that Siriraj score has 92.54% sensitivity in diagnosing ischemic stroke and sensitivity of 87% for diagnosis of hemorrhagic stroke. The overall accuracy of the Siriraj stroke score is 91.11%. The Allen score had a sensitivity of 93.42% and 66.66% for diagnosing ischemic and hemorrhagic stroke respectively. The overall accuracy of the Allen score is 87%.[80]

ALLEN OR GUY'S HOSPITAL SCORE

Allen score was devised by C.M.C.Allen at Guy's hospital London.[77] It was devised to assist the physicians without easy access to CT scan facilities for clinical diagnosis of stroke.

Allen score is based on clinical variables. The onset of symptoms and other associated symptoms like headache, vomiting, neck stiffness, conscious level at 24 hours, diastolic blood pressure 24 hours after admission and history of hypertension.

Atheroma markers like diabetes mellitus, angina and intermittent claudication are included. Previous history of TIA or stroke and heart disease is also included in computation of score.

Allen score (<or =4 infarct, 5-24 equivocal, >24 hemorrhagic)

Variable	Clinical feature	Score
Apoplectic onset(vomiting, neck stiffness, headache within 2 hours and loss of consciousness)	One >or= 2of these	0 21.9
Conscious level (24 hours after admission)	Alert Drowsy Unconscious	0 7.3 14.6
Babinski reflex	Both flexor or single extensor Bilateral extensor	0 7.1
Diastolic Blood pressure (24 hours after admission)	Times	0.17
Atheroma markers (Diabetes mellitus, angina, intermittent claudication)	Absent >or= 1	0 -3.7
H/O Hypertension	Absent Present	0 -4.1
Past H/O (Transient ischemic attack or stroke)	None Any number of events	0 -6.7
Heart disease • Aortic or mitral murmur • Cardiac failure • Cardiomyopathy • Atrial fibrillation • Cardiomegaly • Myocardial infarction within six months	Absent	0 -4.3 -4.3 -4.3 -4.3 -4.3 -4.3
Constant		-12
Total		

Many European validation studies on Allen stroke score have been done and they reported that this score reasonably accurate.

Disadvantages of Allen score

1. It needs detailed history and clinical examination.
2. Since this score has parameters like conscious level and diastolic blood pressure at 24 hours it cannot be used on admission. [76]

SIRIRAJ SCORE

Siriraj stroke score was devised at Siriraj hospital Medical School at University of Mahidol, Bangkok. Poungvarin et al in Siriraj Hospital developed the score by stepwise discriminant analysis of variables followed by linear discriminant equation. He reported the overall accuracy of 90.3 %, the sensitivity for hemorrhage being 89.3%, and sensitivity for infarction being 93.2%.[76]

The clinical variable included in the score are conscious level at admission, vomiting, headache within two hours of onset, diastolic blood pressure on admission, atheroma markers like diabetes mellitus, intermittent claudication and angina.

Siriraj score has been studied and validated in many countries with varying accuracy.

Nigerian study showed accuracy of 58% for ischemic stroke and 50% for hemorrhagic stroke with a accuracy of 54.2%. [81]

Ethiopian study showed accuracy of 61.5% for ischemic stroke and 77% for hemorrhagic stroke with a accuracy of 69.2%.[82]

Hung LY et al in their study showed sensitivity of Siriraj score for ischemic and hemorrhagic stroke were 90 % and 85 % respectively. [83]

Siriraj score (<-1 infarct, -1 t0 +1 equivocal, > 1haemorrhagic)

Variable	Score
Conscious level at admission	
Alert	0
Drowsy or stupor	2.5
Coma or semi coma	5
Vomiting	
Yes	0
No	2
Headache within 2 hours	
Yes	0
No	2
Diastolic blood pressure(mmHg)	
Times	0.1
Atheroma markers(angina, diabetes mellitus or intermittent claudication)	
None	0
One or more	- 3
Constant	-12

GREEK SCORE

Greek stroke score was devised by a team from Athens. Efstathiou SP and co-workers proposed a model i.e., Greek score which helps the physician to differentiate ischemic and hemorrhagic stroke easily. The parameters used in calculating Greek score are easily available to physician soon after admission.[84]

They showed that the sensitivity, specificity, positive predictive value and negative predictive value is 99%, 99%, 97% and 97% respectively. The above observation is much better when compared to Allen and Siriraj scores.[84]

Greek score (<or =3- infarct. 4-12- equivocal, >13 hemorrhage)

Signs/symptoms	Score
Neurological deterioration within 3 hours of admission	6
Vomiting	4
WBC count	4
Decreased level of consciousness	3

Comparison study done in India showed Greek score was better than Allen and Siriraj score in diagnosis of stroke.[85] However

the sensitivity, specificity, positive predictive value and negative predictive value of Indian study was lower than that of original Greek study.

Aamod Soman and co-workers from Grant Medical college Hospital, Mumbai reported that the specificity of Allen score and Greek score for diagnosing hemorrhage is similar. However the Allen's score can be computed only after 24 hours, so they concluded that Greek score is better than Allen's score. [85]

Advantages

1. The parameters used in calculating Greek score are simple and easy to remember at the bedside

2. All the parameters are available to the treating doctor within first 3 hours of admission.

3. No need of calculator

4. It has high specificity for in diagnosing intracerebral hemorrhage.

AIMS AND OJECTIVES

To compare and validate the ALLEN, SIRIRAJ and GREEK clinical stroke scores in differentiation of acute ischemic and hemorrhagic stroke.

The objective is to study the accuracy and validity of this scores in diagnosis of stroke subtype, so that these scores can be used in diagnosis of stroke subtypes in centers where neuroimaging facilities are not available or could not be done due to financial constraints.

MATERIALS AND METHODS

Study population:

100 patients admitted with clinical findings suggestive of stroke at Department of General Medicine were included in the study.

Study period:

March 2012 to October 2012

Study design: cross sectional study

Inclusion criteria:

1) All patients with acute anterior circulation stroke presenting within 24 hours.

2) Age 20 to 75 years

Exclusion criteria:

1) Age <20years, >75 years

2) Patients presenting after 24 hours

3) Patient with chronic severe Neurological illness

4) Patients already on antiplatelet therapy

Informed consent was obtained from all patients before enrolling in the study. The study was clearly explained to the patients and relatives in their own language.

Parameters assessed:

1) Detailed history of the patient
2) Examination with detailed neurological examination
3) Complete blood count
4) Screening for diabetes
5) Renal function test
6) Liver function tests
7) Bleeding time/clotting time
8) PT/PTT/INR
9) Chest X ray
10) ECG
11) CT scan brain
12) Calculation of ALLEN, SIRIRAJ AND GREEK score calculation.

Methods:

All patients between age 20 to 75years admitted in medical wards with history and clinical findings suggestive of anterior circulation stroke are included in the study.

Siriraj and Greek scores was calculated at admission and the Allen score was calculated after 24 hours.

CT scan brain was done for all patients at TNMSC CT scan centre, Stanley Medical Hospital. The results of scores and Radiologist report of CT scan brain were compared. They were statistically analyzed.

OBSERVATION AND RESULTS

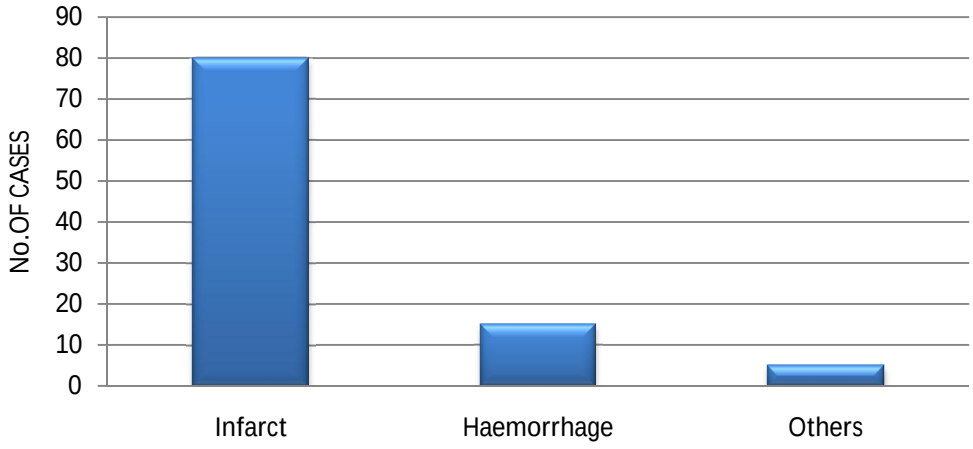
The sample size of our study is 100 patients. All hundred patients those who were clinically diagnosed to have stroke underwent CT scan brain within 24 hours which was considered gold standard .

Out of 100 cases 80 were proven to be infarct , 15 were proven to be hemorrhagic stroke by CT scan brain. Among rest 5 ,one had tuberculoma, one had CNS toxoplasmosis, one patient had cortical vein thrombosis, one had Intracerebral schwanoma and last one had glioma. The five were excluded from the study.

Table 1- showing distribution of cases according to CT scan

CT scan diagnosis	No. of patients
Infarct	80
Haemorrhage	15
Others	5
Total	100

Figure 1-Distribution of cases



Age distribution

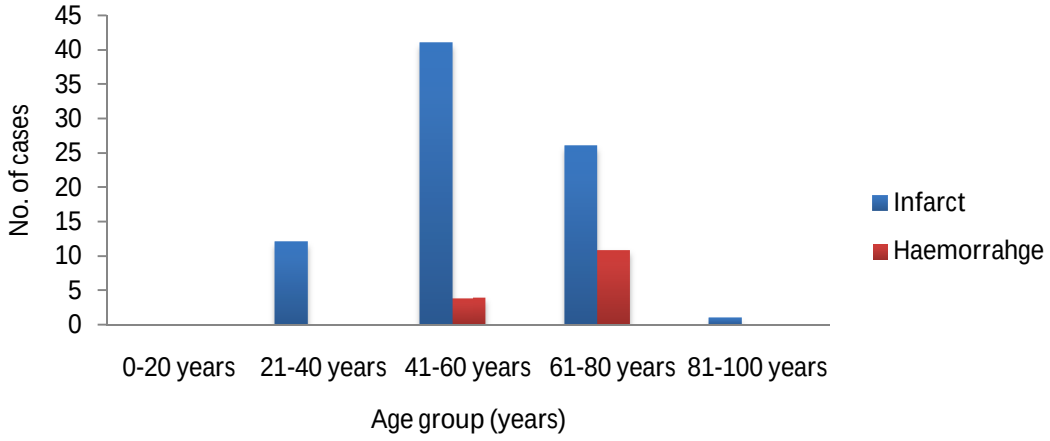
The age group of the patients in our study ranges from 28 to 89 years

Table 2- Age distribution of Stroke cases

Age groups(years)	Infarction (n=80)		Haemorrhage (n=15)	
	Number	Percent	Number	Percent
0-20	--	--	--	--
21-40	12	15.00%	--	--
41-60	41	51.25%	04	26.67%
61-80	26	32.50%	11	73.33%
81-100	01	1.25%	--	--
Total	80	100	15	100

Ischemic stroke was more common in age group of 41-60 years (51.25%) . Hemorrhagic stroke was common in age group of 61-80 years (73.33%). Hemorrhagic stroke was not seen in age group below 40 years in our study.

Figure 2-Age distribution of stroke cases

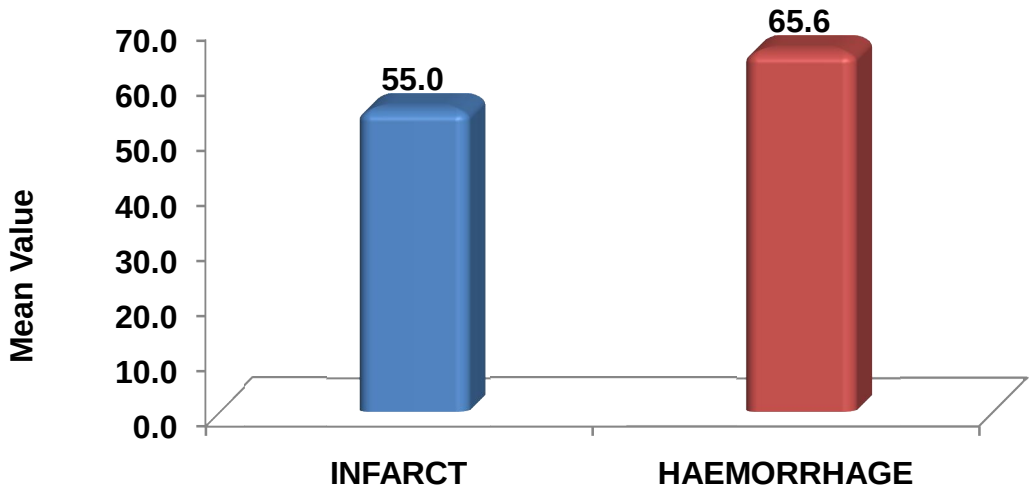


The mean age in infarct group is 55 years where as in hemorrhagic group it is 65.60 years

Table 3 showing Independent samples T-test to compare mean age

CT scan result	N	Mean (years)	Std. Deviation	t-Value	P-Value
Infarct	80	55.01	13.001	3.027	0.003
Hemorrhage	15	65.60	8.542		

Figure 3 Showing Mean Age years



Sex distribution

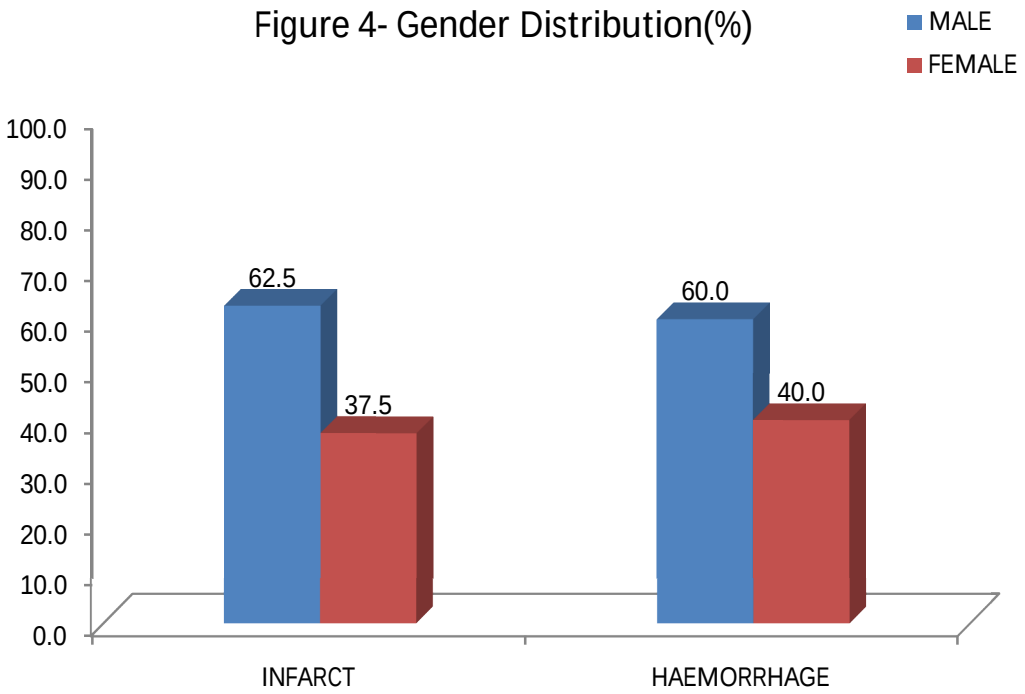
Out of 95 patients in ischemic group 50 were males(62.5%) and 30 were females (37.5%). In hemorrhagic group

10 were males (62.1%) and 5 were females (37.9%) . Therefore males outnumbered the females on the whole.

Table 4 – Showing gender distribution among two groups

Gender	Infarct		Hemorrhage		Total	
	N	%	N	%	N	%
MALE	50	62.5	9	60.0	59	62.1
FEMALE	30	37.5	6	40.0	36	37.9
Total	**80**	**100.0**	**15**	**100.0**	**95**	**100.0**

Figure 4- Gender Distribution(%)



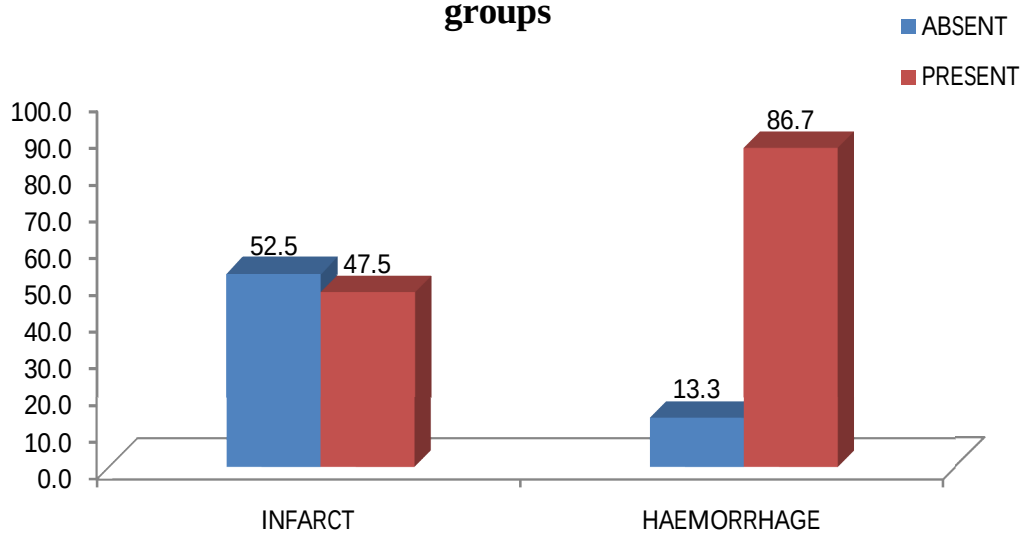
Hypertension

Table 5 – showing percentage of hypertension in two groups

Hypertension	Infarct		Hemorrhage		Total		P value
	N	%	N	%	N	%	
Absent	42	52.5	2	13.3	44	46.3	0.005
Present	38	47.5	13	86.7	51	53.7	

Hypertension is a major risk factor for hemorrhagic stroke when compared to ischemic stroke. 47.5% of patients were hypertensive in ischemic group where as 86.7% of patients were hypertensive hemorrhagic group. The P value is 0.005 which is significant.

Figure 5- percentage of Hypertension patients in two groups



Atheroma markers

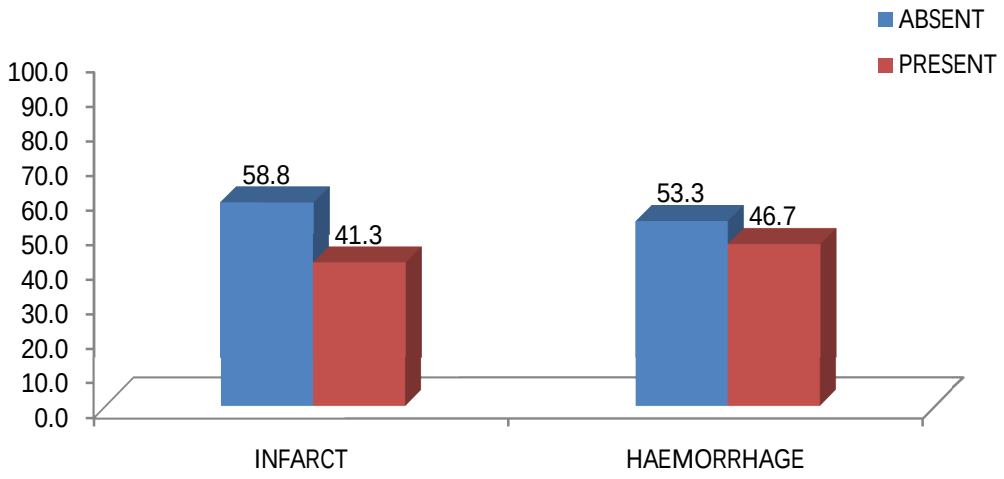
Atheroma markers like diabetes mellitus, angina, and intermittent claudication were present in 41.3 % of infarct group and 46.7 % of hemorrhagic group.

Table 6 – Showing percentage of atheroma markers in two groups

Atheroma Markers	Infarct		Haemorrhage		Total		P value
	N	%	N	%	N	%	
Absent	47	58.8	8	53.3	55	57.9	0.697
Present	33	41.3	7	46.7	40	42.1	
Total	**80**	**100.0**	**15**	**100.0**	**95**	**100.0**	

Diabetes mellitus was the commonest atheroma markers in both our study group, followed by angina.

Figure 6 Showing percentage of Atheroma Markers in two groups.



Heart disease

Heart disease were present in 14 (17.5%) out of 80 patients in infarct group. Out of 14 patients

3 had rheumatic heart disease,

10 had coronary artery disease and

1 had hypertrophic non obstructive Cardiomyopathy.

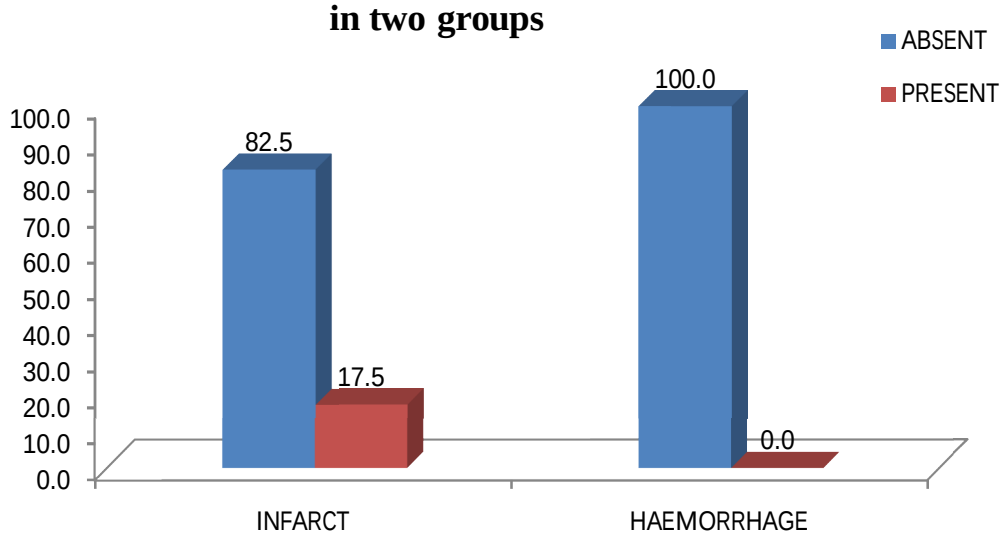
Atrial fibrillation was present in 3 of these patients.

Table 7 – shows percentage of heart disease in two groups

Heart Disease	**Infarct**		**Haemorrhage**		**Total**		**P value**
	N	**%**	**N**	**%**	**N**	**%**	
Absent	66	82.5	15	100.0	81	85.3	0.116*
Present	14	17.5	0	0.0	14	14.7	
Total	**80**	**100.0**	**15**	**100.0**	**95**	**100.0**	

In hemorrhagic group none of them had heart disease.

Figure 7 - Showing percentage of Heart Disease in two groups



Past history of TIA or Stroke

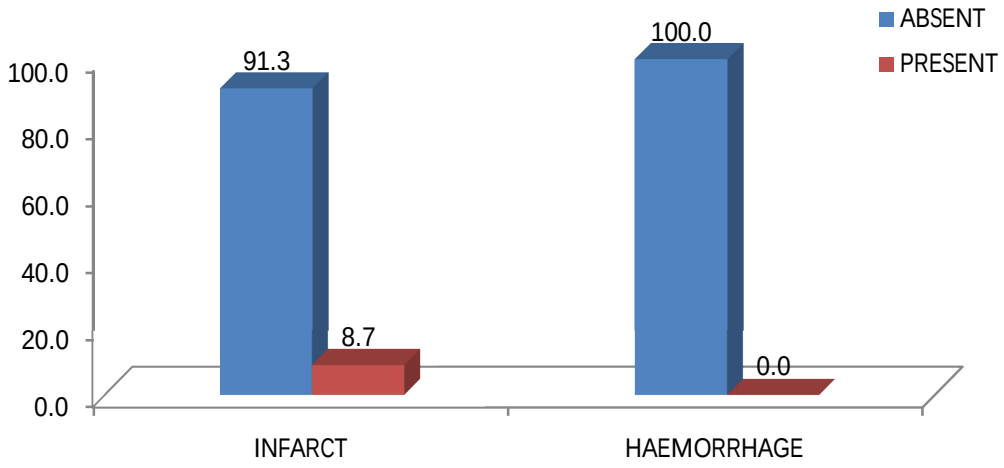
Past history of transient ischemic attack or stroke was present in 8.7 percent of our patients in infarct group.

Table 8 – Showing percentage past history of TIA/Stroke in two groups

Past History of TIA/Stroke	Infarct		Haemorrhage		Total		P value
	N	%	N	%	N	%	
Absent	73	91.3	15	100.0	88	92.6	0.592*
Present	7	8.8	0	0.0	7	7.4	
Total	**80**	**100.0**	**15**	**100.0**	**95**	**100.0**	

In hemorrhagic group none had past history of TIA or Stroke.

Figure 8 - Showing percentage of past History of TIA/Stroke in two groups



Headache

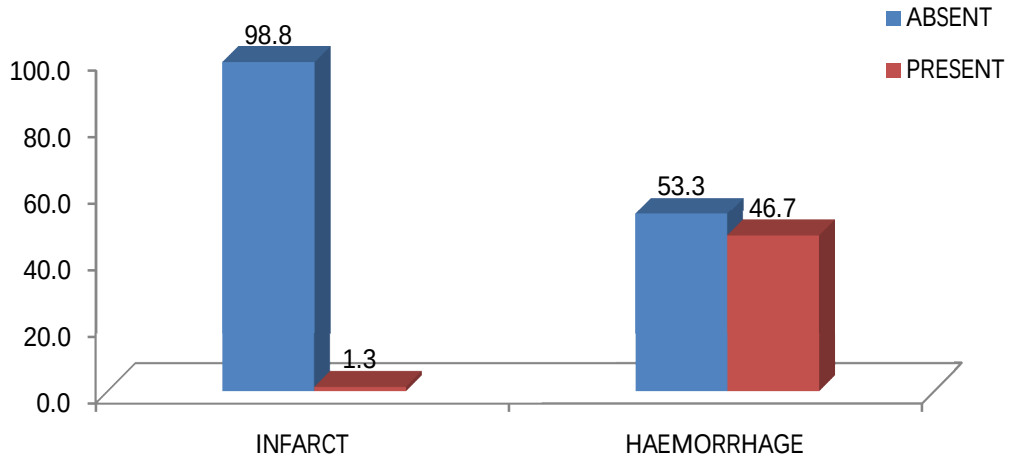
Headache at onset was present in majority of the patients who had hemorrhagic stroke.

Table 9 shows percentage of patients who had headache in each group

Headache	Infarct		Haemorrhage		Total		p value
	N	%	N	%	N	%	
Absent	79	98.8	8	53.3	87	91.6	
Present	1	1.3	7	46.7	8	8.4	**<0.001**
Total	**80**	**100.0**	**15**	**100.0**	**95**	**100.0**	

1 Patient (1.3%) in infarct group had headache where is in hemorrhagic group 7 (46.7%) had headache which is statiscally significant (P <0.001).

Figure 9 - Showing perentage of patients with Headache in both groups



Vomiting

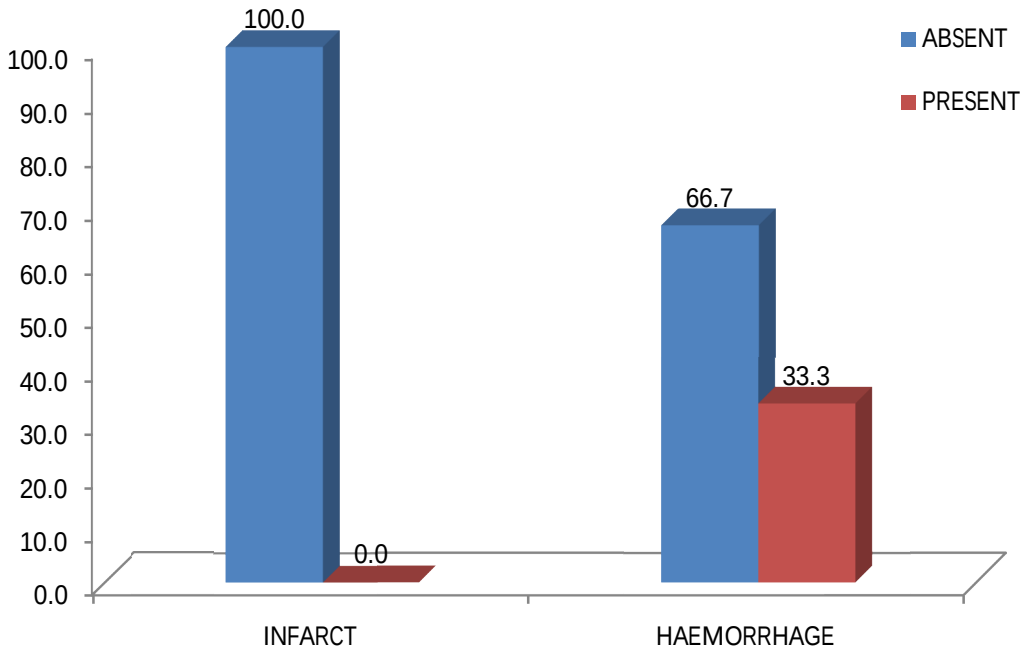
Vomiting was present in 5 patients (33.3%) in who had hemorrhagic stroke.

Table 10 – showing percentage of patients who had vomiting

Vomiting	Infarct		Haemorrhage		Total		P value
	N	%	N	%	N	%	
Absent	80	100.0	10	66.7	90	94.7	
Present	0	0.0	5	33.3	5	5.3	**<0.001**
Total	**80**	**100.0**	**15**	**100.0**	**95**	**100.0**	

In patients with infarct none had vomiting. The above values are statiscally significant (P value < 0.001).

Figure -10 - Showing percentage of patients with Vomiting



Apoplectic onset

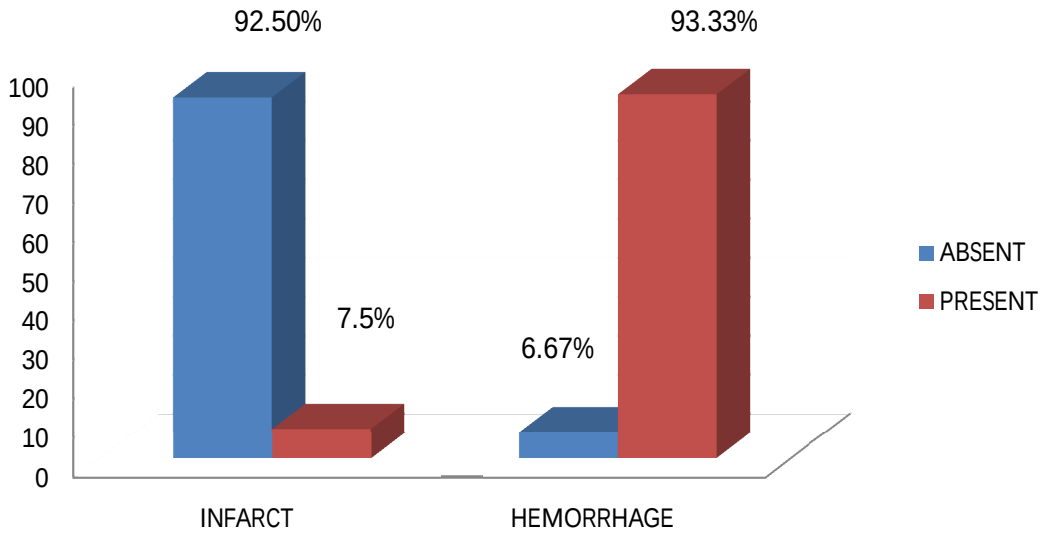
Apoplectic onset is taken as present if two out of the three is present (loss of consciousness, headache within two hours of onset, vomiting and neck stiffness). It was present in 6 patients who had infarct (7.5%).

Table 11 – showing percentage of patients who had apoplectic onset

Apoplectic Onset	Infarct		Haemorrhage				P – Value
	N	%	N	%	N	%	
Absent	74	92.5	1	6.67	76	80.0	**<0.001**
Present	6	7.5	14	93.33	19	20.0	
Total	**80**	**100.0**	**15**	**100.0**	**95**	**100.0**	

In patients who had hemorrhagic stroke 14 out 15 patients (93.33%) had apoplectic onset. Only one patient in hemorrhagic group did not have apoplectic onset who had small bleed and he recovered well. The statistical analysis shows that the values are statiscally significant (P value< 0.001).

Figure 11 – Showing percentage of apoplectic onset in two groups



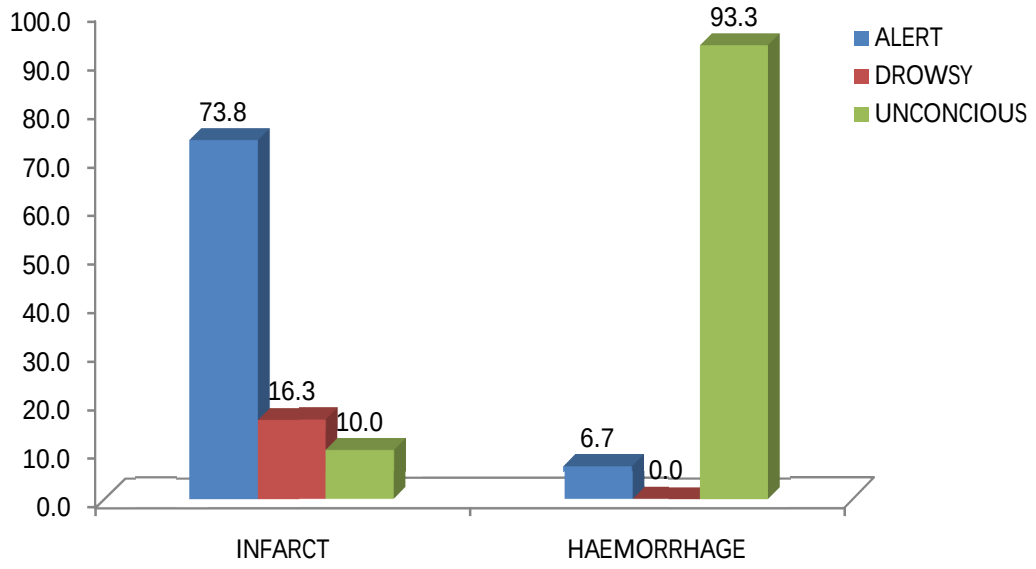
Conscious level at admission

In infarct group 59 patients (73.8%) were alert on admission, 13 patients (16.3%) were drowsy and 8 patients (10%) were unconscious at admission.

Table 12 – showing level of consciousness of patients at admission

Conscious Level at admission	Infarct		Haemorrhage				P Value
	N	%	N	%	N	%	
Alert	59	73.8	1	6.7	60	63.2	
Drowsy	13	16.3	0	0.0	13	13.7	**<0.001**
Unconscious	8	10.0	14	93.3	22	23.2	

Figure 12 - Level of consciousness at Admission (%)



In patients with hemorrhagic stroke 1 patient (6.7%) was alert and rest 14 (93.3%) of them were unconscious at admission. The above values are statistically significant (P value < 0.001).

Conscious level at 24 hours

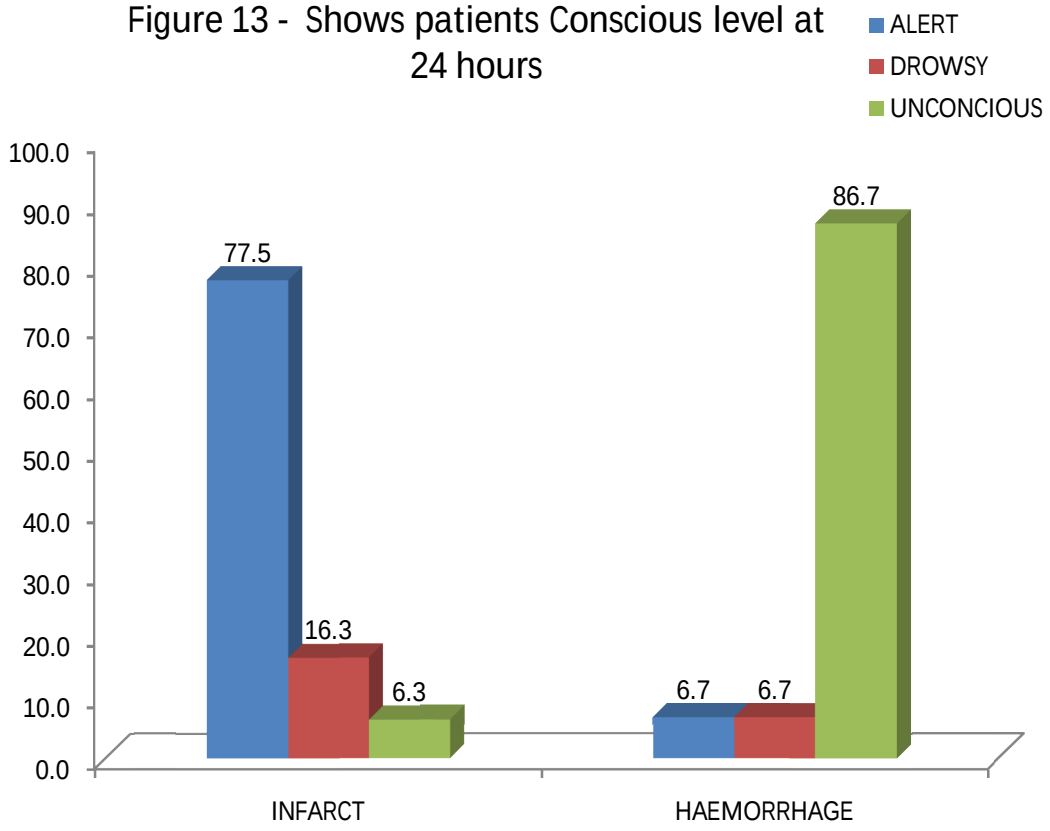
Table 13- shows conscious level of patients after 24 hours

Conscious Level at 24 Hours	Infarct		Haemorrhage		Total		P value
	N	%	N	%	N	%	
Alert	62	77.5	1	6.7	63	66.3	
Drowsy	13	16.3	1	6.7	14	14.7	**<0.001**
Unconscious	5	6.3	13	86.7	18	18.9	
Total	**80**	**100.0**	**15**	**100.0**	**95**	**100.0**	

At 24 hours 62 patients (77.5%) were alert, 13 patients (16.3%) were drowsy and 5 patients (6.3) were unconscious infarct group.

In patients with hemorrhagic stroke 1patient was alert (6.3%), 1 patient was drowsy and 13 patients (86.7%) were unconscious at 24 hours which is statiscally significant (P< 0.001).

Figure 13 - Shows patients Conscious level at 24 hours



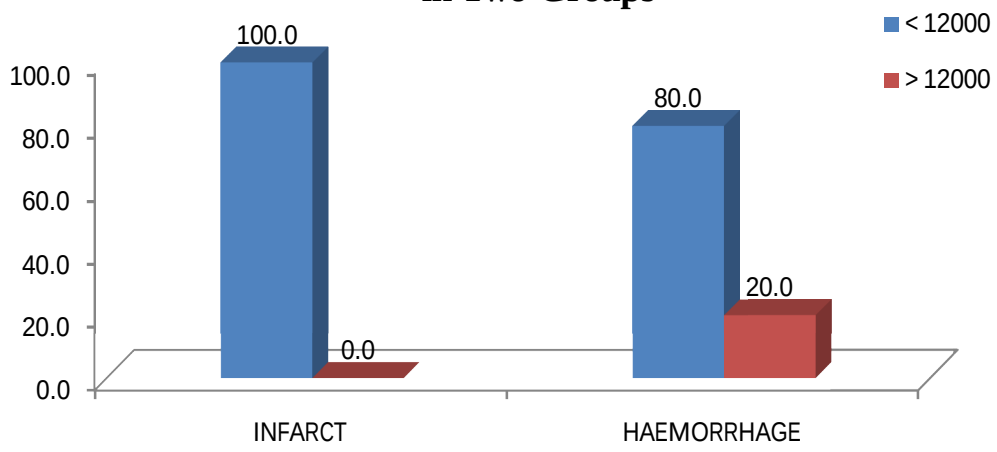
WBC count

Table 14 – WBC count in two groups

WBC count	Infarct		Haemorrhage		Total		P value
	N	%	N	%	N	%	
<12000	80	100.0	12	80	92	96.8	**0.003**
>12000	0	0.0	3	20	3	3.2	
Total	**80**	**100.0**	**15**	**100.0**	**95**	**100.0**	

WBC count is increased <12000 in 3 patients (20%) with hemorrhagic stroke. In infarct group none had elevated WBC count.

Figure 14. Showing percentage of WBC count >12000 in Two Groups



Analysis of Allen's stroke score

In our study 80 patients had infarct which was proven by CT scan brain. Out of 80 Allen stroke score diagnosed 66 patients correctly as having infarct, however it also misdiagnosed 6 patients as having hemorrhage who actually had infarct in CT scan brain. Allen score was equivocal in 8 patients.

Table 15 shows comparison of Allen score results with CT scan brain

Allen score	**CT scan result**		**Total**
	Infarct	**Hemorrhage**	
Infarct	66	1	67
Equivocal	8	0	8
Hemorrhage	6	14	22
Total	**80**	**15**	**95**

15 patients had hemorrhage which was proven by CT scan brain. Out of 15, Allen stroke score diagnosed 14 patients correctly as having hemorrhage, however it wrongly diagnosed 1 patient as having infarct who actually had hemorrhage in CT scan brain.

Table 16- Evaluation of Allen score with statistical parameters for infarct

Parameter	Estimate	Lower - Upper 95% CIs
Sensitivity	82.50%	(72.74, 89.28)
Specificity	93.33%	(70.18, 98.81)
Positive Predictive Value	98.51%	(92.02, 99.74)
Negative Predictive Value	50.00%	(32.63, 67.37)
Diagnostic Accuracy	84.21%	(75.57, 90.19)

Allen stroke score has 82.50 % sensitivity and 93.33 % specificity for infarction. The positive predictive value is 98.50 % and the negative predictive is 50.00 % for infarction. The diagnostic accuracy of Allen's score for diagnosing infarct is 84.21%.

Table 16 - Evaluation of Allen score with statistical parameters for hemorrhage

Parameter	Estimate	Lower - Upper 95% CIs
Sensitivity	93.33%	(70.18, 98.81)
Specificity	92.50%	(84.59, 96.52)
Positive Predictive Value	70.00%	(48.10, 85.45)
Negative Predictive Value	98.67%	(92.83, 99.76)
Diagnostic Accuracy	92.63%	(85.56, 96.39)

Allen stroke score has a sensitivity of 93.33 % and 92.50 % for hemorrhage. It also has the positive predictive value of 70 % and negative predictive of 98.67% for hemorrhage. The diagnostic accuracy is 92.63%.

Analysis of Siriraj stroke score

Table 17- Correlation of Siriraj stroke score with CT scan brain

Siriraj stroke score	CT scan brain		Total
	Infarct	Haemorrhage	
Infarct	70	1	71
Equivocal	2	0	2
Haemorrhage	8	14	22
Total	**80**	**15**	**95**

Out of 80 patients who had infarct in CT scan brain Siriraj stroke score correctly diagnosed 70 patients as having infarct. However it showed 8 patients as having hemorrhage who actually had infarct in CT scan. Siriraj score was equivocal in 2 patients. Out of 15 patients who had hemorrhage in imaging, Siriraj stroke score diagnosed 14 patients correctly.

Table 18- Evaluation of Siriraj score with statistical parameters for infarct

Parameter	Estimate	Lower - Upper 95% CIs
Sensitivity	87.50%	(77.70, 93.50)
Specificity	93.33%	(66.03, 99.60)
Positive Predictive Value	98.50%	(91.34, 99.92)
Negative Predictive Value	58.33%	(36.94, 77.20)
Diagnostic Accuracy	89.47%	(81.70, 94.18)

Siriraj stroke score has 87.50 % sensitivity and 93.33% specificity for infarction. The positive predictive value is 98.50 % and the negative predictive is 58.33 % for infarction. The diagnostic accuracy of Siriraj score for diagnosing infarct is 89.47 %.

Table 19- Evaluation of Siriraj score with statistical parameters for hemorrhage

Parameter	Estimate	Lower - Upper 95% CIs
Sensitivity	93.33%	(66.03, 99.60)
Specificity	90.00%	(80.70, 95.00)
Positive Predictive Value	63.64%	(40.80, 81.90)
Negative Predictive Value	98.63%	(91.57, 99.92)
Diagnostic Accuracy	90.53%	(82.97, 94.94)

Siriraj stroke score has 93.33 % sensitivity and 90.00% specificity for hemorrhage. The positive predictive value is 63.64 % and the negative predictive is 98.63 % for hemorrhage. The diagnostic accuracy of Siriraj score for diagnosing hemorrhage is 90.53 %.

Analysis of Greek stroke score

Table 20- Correlation of Greek stroke score with CT scan brain

Greek stroke score	CT scan brain		Total
	Infarct	Haemorrhage	
Infarct	73	1	74
Equivocal	5	0	5
Haemorrhage	2	14	16
Total	**80**	**15**	**95**

Out of 80 patients who had infarct in CT scan brain Greek stroke score correctly diagnosed 73 patients as having infarct. However it showed 2 patients as having hemorrhage who actually had infarct in CT scan. Siriraj score was equivocal in 5 patients. Out of 15 patients who had hemorrhage in imaging, Greek stroke score diagnosed 14 patients correctly.

Table 21- Evaluation of Greek score with statistical parameters for infarct

Parameter	Estimate	Lower - Upper 95% CIs
Sensitivity	91.25%	(82.25, 96.11)
Specificity	93.33%	(66.63, 99.65)
Positive Predictive Value	98.64%	(91.60, 99.92)
Negative Predictive Value	66.66%	(43.10, 84.51)
Diagnostic Accuracy	97.75%	(92.17, 99.38)

Greek stroke score has a sensitivity of 91.25 % % and specificity of 93.33 % for infarction. The positive predictive value is 98.64 % % and the negative predictive is 66.66 %% for infarction. The diagnostic accuracy of Greek score for diagnosing infarct is 97.75 %.

Table 22- Evaluation of Greek score with statistical parameters for hemorrhage

Parameter	Estimate	Lower - Upper 95% CIs
Sensitivity	93.33%	(70.18, 98.81)
Specificity	97.50%	(91.34, 99.31)
Positive Predictive Value	87.50%	(63.98, 96.50)
Negative Predictive Value	98.73%	(93.17, 99.78)
Diagnostic Accuracy	96.84%	(91.12, 98.92)

Greek stroke score has 93.33% sensitivity and 97.50 % specificity for hemorrhage. The positive predictive value is 87.50 % and the negative predictive is 98.73 % for hemorrhage. The diagnostic accuracy of Greek score for diagnosing hemorrhage is 96.84%.

DISCUSSION

Management of cerebrovascular accident is mainly based on the differentiation of ischemic stroke from hemorrhagic stroke and so is the prognosis. In remote areas where Computed tomography is not easily available, Clinical stroke scores can be used to differentiate ischemic stroke from hemorrhagic stroke. These scores are simple, can be used for screening at bedside and don't consume time.

Computed tomography is safe, non invasive, accurate method for differentiating acute ischemic and hemorrhagic stroke. However, this facility is not freely available in India and other developing countries and even if available, many cannot afford it. The treating physicians in charge of stroke unit should therefore assess the patient's history and clinical features in detail to subtype the stroke. In such setting stroke scores can be used to subtype the stroke.

The present study was carried out in 100 consecutive new stroke patients admitted to Stanley Medical College Hospital, Chennai. Those patients who were clinically diagnosed to have stroke were included in the study. The Allen score was calculated for all of them after 24 hours. Siriraj and Greek stroke scores were calculated for all of them at admission.

CT scan brain was taken for all of them. The stroke scores were compared with imaging reports and analyzed, with an aim to assess the validity of Allen, Siriraj and Greek stroke score to differentiate ischemic stroke from hemorrhagic stroke.

Sensitivity of Allen score for infarct

The sensitivity of Allen score in diagnosing infarction in the present study is 82.50 %. In study by Sandercock et al, sensitivity of Allen's score for infarction is 78%.[86] Hawkins et al study from Auckland shows the sensitivity of Allen score in diagnosing infarct is 78%.[87]

Kochar et al (Bikaner) reported that the Allen score has sensitivity of 91% in diagnosing infarct. [88]

The sensitivity of Allen score for diagnosing infarct is 93% and 52% in studies conducted by Wadhwani et al (Indore) [80] and Badam et al (Sevagram) [89] respectively. The comparison of Allen's score in the diagnosis of infarction with other validation studies is given below.

Our study result of 82.50% sensitivity is similar to other validated studies

Table 23- comparison of sensitivity of present study other validation studies on Allen stroke score for infarct

Study	Place	Sensitivity (%)
Sandercock et al	Oxford	78.00
Hawkins et al	Auckland	78.00
Kochar et al	Bikaner	91
Wadhwani et al	Indore	93
Badam et al	Sevagram	52
Present study	Chennai	82.50

Specificity of Allen score for infarct

The specificity of Allen score in diagnosing infarction in the present study is 93.33 %. Hawkins et al study from Auckland shows the specificity of Allen score in diagnosing infarct is 70%.[87]

Kochar et al (Bikaner) reported that the Allen score has specificity of 60% in diagnosing infarct. [88]

The specificity of Allen score for diagnosing infarct is 86% in studies conducted Badam et al (Sevagram). [89] The specificity of 93.33% in our study is in contrary to the other studies.

Table 24-comparison of specificity of present study to other validation studies on Allen stroke score for infarct

Study	Place	Specificity (%)
Hawkins et al	Auckland	70.00
Kochar et al	Bikaner	60.00
Glen CH et al[90]	New Zealand	70.00
Badam et al	Sevagram	86.00
Present study	Chennai	93.33

Positive predictive value of Allen score for infarction

The predictive value of a positive test in the present study for infarction by Allen score is 98.51 %. In the study by Sandercock et al, the positive predictive value in the diagnosis of infarction by Allen's score is 78%.[86] In the study conducted by Celani et al, the positive predictive value in the diagnosis of infarction by Allen score was 93%.[79]

In a study conducted by Huang JA et al the positive predictive value is 100.00% which is comparable to our study.[91]

Table 25-Comparison of positive predictive value of present study in the diagnosis of infarction with other validation studies on Allen stroke score

Study	Infarction (%)
Kochar et al	77.00
Sandercock et al	78.00
Celani et al	93.00
Huang JA, et al	100.00
Present study	98.51

Sensitivity of Allen score for hemorrhage

The sensitivity of Allen score in diagnosing hemorrhage in the present study is 93.33%. In study by Sandercock et al, sensitivity of Allen score for hemorrhage is 88%.[86] Hawkins et al study from Auckland shows the sensitivity of Allen score in diagnosing infarct is 31 %. [87]

Kochar et al (Bikaner) reported that the Allen score has sensitivity of 60% in diagnosing hemorrhage. [88]

The sensitivity of Allen score for diagnosing hemorrhage is 35% in a study Badam et al (Sevagram). [89]

Our study result of 93.33% sensitivity is contrary to other validated studies.

Table 26-comparison of sensitivity of present study to other validation studies on Allen stroke score for hemorrhage

Study	Place	Sensitivity (%)
Sandercock et al	London	88.00
Hawkins et al	Auckland	31.00
Kochar et al	Bikaner	60.00
Badam et al	Sevagram	35.00
Present study	Chennai	93.33

Specificity of Allen score for hemorrhage

The specificity of Allen score in diagnosing hemorrhage is 92.50%. Hawkins et al study from Auckland shows the specificity of Allen score in diagnosing hemorrhage is 95%. [87]

Kochar et al (Bikaner) reported that the Allen score has specificity of 91% in diagnosing hemorrhage. [88]

The specificity of Allen score for diagnosing hemorrhage is 88% in studies conducted Badam et al (Sevagram). [89]

The specificity of 92.50% in our study is similar to the other studies.

Table 27- comparison of specificity of present study compared to other validation studies on Allen stroke score for hemorrhage

Study	Place	Specificity (%)
Hawkins et al	Auckland	95.00
Kochar et al	Bikaner	91.00
Glen CH et al	New Zealand	95.00
Badam et al	Sevagram	88.00
Present study	Chennai	92.50

Sensitivity of Siriraj score for infarct

The sensitivity of Siriraj score in diagnosing infarction in the present study is 87.50% and. In study by Poungvarin et al, sensitivity of Siriraj score for infarction is 93%.[76] Hui et al from Hong Kong and Hawkins et al study from Auckland shows the sensitivity of Siriraj score in diagnosing infarct is 78% and 61% respectively. [92]

Kochar et al (Bikaner) reported that Siriraj score has sensitivity 73% in diagnosing infarct. [88]

The sensitivity of Siriraj score for diagnosing infarct is 93% and 52 % in studies conducted by Wadhwani et al (Indore) [80] and Badam et al (Sevagram) [89] respectively.

Our study result of 87.50% sensitivity is similar to other validated studies like Poungvarin et al, Wadhwani et al.

Table 28 - comparison of sensitivity of present study other validation studies on Siriraj stroke score for infarct

Study	Place	Sensitivity (%)
Poungvarin et al	Bangkok	93.00
Hui et al	Hong Kong	78.00
Hawkins et al	Auckland	61.00
Kochar et al	Bikaner	73.00
Wadhwani et al	Indore	93.00
Badam et al	Sevagram	52.00
Present study	Chennai	87.50

Specificity of Siriraj score for infarct

The specificity of Siriraj score in diagnosing infarction in the present study is 93.33%. Hawkins et al study from Auckland shows the specificity of Siriraj score in diagnosing infarct is 74%.[87]

Kochar et al (Bikaner) reported that Siriraj score has specificity of 80% in diagnosing infarct. [88]

The specificity of Siriraj score for diagnosing infarct is 82% in study conducted Badam et al (Sevagram). [89] The specificity of 93.33% in our study is near to Kochar et al study.

Table29 - comparison of specificity of present study to other validation studies on Siriraj stroke score for infarct

Study	Place	Specificity (%)
Hawkins et al	Auckland	74.00
Kochar et al	Bikaner	85.00
Glen CH et al	New Zealand	70.00
Badam et al	Sevagram	82.00
Present study	Chennai	93.33

Sensitivity of Siriraj score for hemorrhage

The sensitivity of Siriraj score in diagnosing hemorrhage in the present study is 93.33%. In study by Poungvarin et al, sensitivity of Siriraj score for hemorrhage is 89%.[76] Hui et al[90] and Hawkins et al[87] in their study showed that the sensitivity of Siriraj score in diagnosing hemorrhage is 91% and 48% respectively.

Kochar et al (Bikaner) reported that Siriraj score has sensitivity 85% in diagnosing hemorrhage. [88]

The sensitivity of Siriraj score for diagnosing hemorrhage is 44% in a study Badam et al (Sevagram). [89] Our study result of 93.33% is similar to the Hui et al and Poungvarin et al study reports.

Table 30- comparison of sensitivity of present study to other validation studies on Siriraj stroke score for hemorrhage

Study	Place	Sensitivity (%)
Poungvarin et al	Bangkok	89.00
Hui et al	Hong Kong	91.00
Hawkins et al	Auckland	48.00
Kochar et al	Bikaner	85.00
Badam et al	Sevagram	44.00
Present study	Chennai	93.33

Specificity of Allen score for hemorrhage

The specificity of Siriraj score in diagnosing hemorrhage is 90%. Celani et al[79] and Hawkins et al[87] in their showed the specificity of Siriraj score in diagnosing hemorrhage is 94% and 85% respectively. Hui et al showed that the specificity of Siriraj score for hemorrhage is 90%.[90]

Kochar et al (Bikaner) reported that Siriraj score has specificity of 73% in diagnosing hemorrhage. [88]

The specificity of Siriraj score for diagnosing hemorrhage is 85% in studies conducted Badam et al (Sevagram). [89]

The specificity of 90% in our study is similar to the other studies.

Table 31 - comparison of specificity of present study compared to other validation studies on Siriraj stroke score for hemorrhage

Study	Place	Specificity (%)
Celani et al	Italy	94.00
Hawkins et al	Auckland	85.00
Hui et al	Hong Kong	91.00
Kochar et al	Bikaner	90.00
Badam et al	Sevagram	88.00
Present study	Chennai	90.00

Greek score

The sensitivity, specificity, positive predictive value and negative predictive value of Greek score for diagnosing infarct in our study is 91.25%, 93.33%, 98.64 and 66.66% respectively.

The sensitivity, specificity, positive predictive value and negative predictive value of Greek score for diagnosing hemorrhage in our study is 93.33%, 97.50% , 87.50% and 98.75% respectively.

The original Greek study by a team from Athens showed sensitivity of 99%, specificity of 99%, positive predictive value of 97%, negative predictive value of 97%.[84] Our study also shows similar results.

Berhe et al showed that the sensitivity, specificity, positive predictive value and negative predictive value of Greek score for diagnosing hemorrhage is 77.80%, 89.30%, 87.50% and 80.60% respectively. [93]

All the three scores are mainly based on clinical symptoms. In our study patients diagnosed to have massive infarct by CT Scan brain were wrongly predicted to have hemorrhage by the stroke scores. This fallacy

is may be due to the weightage given to apoplectic onset which favors hemorrhage according to the scores.

On the other way, one patient with a small hemorrhage as detected by CT scan had clinical score favoring infarction because of their minimal symptoms and therefore incorrectly diagnosed as infarction by clinical scores.

Another fallacy is that all scores lack formal definitions for some Variables. In all three scoring systems level of consciousness was given more weightage. Even in large infarcts cerebral edema leads to midline shift, brainstem compression, alteration in the level of consciousness and gives false interpretation of hemorrhage.

Hypertension is one of the most important risk factor for both infarcts as well as hemorrhage, but in Allen and Siriraj scores high blood pressure favors more the hemorrhage than the infarction and hence gives false results.

In general the existing scoring protocols classify those with severe stroke as hemorrhagic strokes and those with less severe strokes of as ischemic regardless of their etiology, therefore scoring methods needs modifications in their variables.

When computerized tomography is not immediately available and the main concern for the treating physician is to rule out hemorrhage to start antithrombotic therapy.

Therefore a test with highest specificity and negative predictive value for hemorrhage is essential. In present study Greek score has the highest specificity for hemorrhage followed by Allen and Siriraj stroke score. Greek score has the highest negative predictive value for hemorrhage followed by Siriraj and Allen score stroke score.

But Allen's score needs a very detailed clinical evaluation and history, at least twenty-hours of observation and is much more difficult to calculate.

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

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

CONCLUSION

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

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

- When both the Siriraj and Greek stroke scores considered together in a same patient, sensitivity and specificity for diagnosis of infarction and hemorrhage can be increased.

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

- Nevertheless CT scan is the gold standard for differentiating ischemic and hemorrhagic stroke when the facility is available round the clock.

APPENDIX I -BIBLIOGRAPHY

1. Thomas Truelsen, Stephen Begg, Colin Mathers. The global burden of cerebrovascular disease.

2. Uston, Cagatay (February 20, 2004). "Dr. Thomas Willis' Famous Eponym: The Circle of Willis". Turkish Journal of Medical Sciences. 34: 271–274.

3. Purves, Dale; George J. Augustine, David Fitzpatrick, William C. Hall et al. Neuroscience, 4th Ed. Sinauer Associates. pp. 834–5.

4. Moore KL, Dalley AR. Clinically Oriented Anatomy, 5th Ed., Lippincott Williams & Wilkins, Toronto. Copyright 2007.

5. Hugo A. Krayenbühl, Mahmut Gazi Yasargil, Peter Huber, George Bosse. Cerebral Angiography, Thieme, pp. 79–91.

6. Schneider, Marco A. Stefani; Marrone, Antonio C.H; Severino et al. "Anatomic Variations of Anterior Cerebral Artery Cortical Branches", Clinical Anatomy (13): 321–236.

7. Osborn, Anne G.; Jacobs, John M. (1999), Diagnostic Cerebral Angiography, Lippincott Williams & Wilkins, pp. 143–144.

8. Eugen Braunwald, Anthony S Fauci, Dennis L Kasper et al. Harrison's principles of internal medicine 15th edition, McGraw Hill.2001.

9. "Brain Basics: Preventing Stroke". National Institute of Neurological Disorders and Stroke. Retrieved 2009-10-24.

10. Mohr JP, Capul LR, Melski JW et al. The Harvard cooperative stroke registry: A prospective study of cases hospitalized with stroke. Neurology, 1978; 28: 754.

11. Diabetes and cerebrovascular disease: The Framingham study, JAMA1979; 241:2085.

12. Asian Acute Stroke Advisory Panel. Stroke epidemiological data of nine Asian Countries. J Med Assoc Thai, 2000: 83:1-7.

13. African journal of neurological sciences.ISSN1015-8618, volume 20, num2.

14. Dalal PM, Dalal KP, Saraf D. Stroke in west central India. In: Oda T et al Eds. Internal medicine, Elsevier Sci publication, British Amsterdam: 37-44.

15. Abraham J, Rao PSS, Inderaj SG et al. Epidemiological study of hemiplegia due to stroke in South India. Stroke 1970; 1:477.

16. Razdan S, Kaul RL, Motta A, Kaul S. Cerebrovascular disease in rural Kashmir, India. Stroke 1989; 20:169-93.

17. Wolf PA, D'Agostino RB, O'Neal MA, Sytkowski P et al. Secular trends in stroke incidence and mortality. The Framingham Study. Stroke. 1992; 23:1551-1555.

18. Kiely DK, Wolf PA, Cupples LA, Beiser AS et al. Familial aggregation of stroke. The Framingham Study. Stroke. 1993; 24:1366-1371.

19. Howard G, Anderson R, Sorlie P, Andrews V et al. Ethnic differences in stroke mortality between non-Hispanic whites, Hispanic whites, and blacks: the National Longitudinal Mortality Study. Stroke. 1994; 25:2120-2125.

20. SHEP Cooperative Research Group. Prevention of stroke by antihypertensive drug treatment in older persons with isolated systolic hypertension: final results of the Systolic Hypertension in the Elderly Program (SHEP). JAMA. 1991; 265:3255-3264.

21. Benjamin EJ, Levy D, Vaziri SM, D Agostino RB et al. Independent risk factors for atrial fibrillation in a population-based cohort: the Framingham Heart Study. JAMA. 1994; 271:840-844

22. Warfarin versus aspirin for prevention of thromboembolism in atrial fibrillation: Stroke Prevention in Atrial Fibrillation II Study. Lancet. 1994; 343:687-691.

23. Miller VT, Pearce LA, Feinberg WM et al. Differential effect of aspirin versus warfarin on clinical stroke types in patients with

atrial fibrillation: Stroke Prevention in Atrial Fibrillation Investigators. Neurology. 1996; 46:238-240.

24. American College of Physicians. Guidelines for medical treatment for stroke prevention. Ann Intern Med 1994; 121:54-5.

25. Burchfiel CM, Curb JD, Rodriguez BL, Abbott RD et al. Glucose intolerance and 22-year stroke incidence: the Honolulu Heart Program. Stroke. 1994; 25:951-957.

26. Shinton R, Beevers G. Meta-analysis of relation between cigarette smoking and stroke. BMJ. 1989; 298:789-794.

27. Gorelick PB. Does alcohol prevent or cause stroke? Cerebrovascular Diseases. 1995; 5:379.

28. Jones TH, Morawetz RB, Crowell RM et al. Thresholds of focal ischemia in awake monkeys. J Neurosurg. 1981; 54:773-782.

29. Wass CT, Lanier WL. Glucose modulation of ischemic brain injury: review and clinical recommendations. Mayo Clin Proc.1996; 71: 801-812.

30. Bruno A, Biller J, Adams HP Jr et al. Acute blood glucose level and outcome from ischemic stroke. Neurology. 1999; 52: 280-284.

31. Reith J, Jorgensen HS, Pedersen PM, et al. Body temperature in acute stroke: relation to stroke severity, infarct size, mortality, and outcome. Lancet. 1996; 347: 422-425.

32. Schwab S, Spranger M, Aschoff A et al. Brain temperature monitoring and modulation in patients with severe MCA infarction. Neurology. 1997; 48: 762-767.

33. Pulsinelli WA. The ischemic penumbra in stroke. Sci Med. 1995; 1:16-25.

34. Fisher CM. The arterial lesions underlying lacunes. Acta Hemopathol, 1969; 12:1.

35. Fisher CM. Lacunar strokes and infarcts: A review. Neurology 1982; 321:871-884

36. Tyler KL, Poletti CE, Heros RC. Cerebral amyloid angiopathy with multiple intracerebral hemorrhages: Case report. J Neurosurg 1982; 57:286-290.

37. Heir DB, Davis KR, Richardson ER et al. Hypertensive putaminal hemorrhage. Annals of neurol 1997; 1:152-59.

38. Kare CS, Mohr JP, Stein BM et al. Stroke: pathophysiology, diagnosis and management, Churchill Livingstone 1986.

39. Furlan AJ, Whisnat JP, Elehack GR. The decreasing incidence of primary Intracerebral hemorrhage: A population study. Annals of neurology 1979; 5:367.

40. Easton JD.et.al. Definition and evaluation of transient ischemic attack. Stroke. 2009 Jun; 40(6):2276–93.

41. Wiggins WS, Moody DM, Toole JF et al. Clinical and computerized tomographic study of hypertensive Intracerebral hemorrhage. Archives of neurology 1978; 5:832.

42. Ken ML et al. Natural history of ischemic stroke. Eds. Cerebrovascular disease, Logon Butterworth's, 1983.

43. Minna, Pirnjo, Jaako. Atrial fibrillation and stroke. Stroke August 1996.

44. Dalsgaard Midson T. Survey of 1000 cases of apoplexy cerebri. Acta Psychiatric neurological Scandinavia 1956; 30:169.

45. Bamford J, Sandercock P, Dennis M et al. A prospective study of acute cerebrovascular disease in the community. J Neurol.Neurosurg.Psychiatry 1988; 51:1373-80

46. Recovery of motor function after stroke 1988; 19:1497-500.Bonita R, Beaglehole.

47. Burn J, Dennis M, Bamford J, Sandercock P et al. Long-term risk of recurrent stroke after a first-ever stroke. Stroke 25, 333-337. 1994.

48. Goldstein LB, Simel DL. Is this patient having a stroke? J Am Med Assoc. 2005; 293: 2391–2402.

49. Von Arbin M, Britton M, de Faire U et al. Accuracy of bedside diagnosis in stroke. Stroke. 1981; 12*:* 288–*293.*

50. Panzer RJ, Feibel JH, Barker WH, Griner PF. Predicting the likelihood of hemorrhage in patients with stroke. Arch Intern Med. 1985; 145*:* 1800–*1803.*

51. Gross CR, Shinar D, Mohr JP, et al. Interobserver agreement in the diagnosis of stroke type. Arch Neurol. 1986; 43*:* 893–*898.*

52. Muir KW, Weir CJ, Murray GD et al. Comparison of neurological scales and scoring systems for acute stroke prognosis. Stroke. 1996; 27*: 1817*–1820.

53. Britton M, Hindmarsh T, Murray V, Tyden SA. Diagnostic errors discovered by CT in patients with suspected stroke. Neurology. 1984; 34*:* 1504–1507.

54. Bamford J. Clinical examination in diagnosis and sub-classification of stroke. Lancet 1992; 339:400 - 402.

55. Hacke W, Kaste M, Fieschi C, Toni D, Lesaffre E, von Kummer R et al. Intravenous thrombolysis with recombinant tissue plasminogen activator for acute hemispheric stroke. The European Cooperative Acute Stroke Study. JAMA. Oct 4 1995; 274(13):1017-25.

56. Caplan LR. Treatment of acute stroke: still struggling. JAMA. 2004*;* 292*:* 1883–1885.

57. Caplan LR. Stroke thrombolysis: slow progress. Circulation. 2006*;* 114*:* 187–190.

58. Rowley HA. Extending the time window for thrombolysis: evidence from acute stroke trials. Neuroimaging Clin N Am. 2005*;* 15*:* 575–587.

59. Sims J, Schwamm LH. The evolving role of acute stroke imaging in intravenous thrombolytic therapy: patient selection and outcomes assessment. Neuroimaging Clin N Am. 2005*;* 15*:* 421–440.

60. Schellinger PD. The evolving role of advanced MR imaging as a management tool for adult ischemic stroke: a Western-European perspective. Neuroimaging Clin N Am. 2005*;* 15*:* 245–258.

61. Barber PA, Demchuk AM, Hudon ME et al. Hyper dense sylvian fissure MCA "dot" sign: a CT marker of acute ischemia. Stroke. 2001; 32:84-8.

62. Fiebach JB, Schellinger PD, Jansen O et al. CT and diffusion-weighted MR imaging in randomized order: diffusion-weighted imaging results in higher accuracy and lower interrater variability in the diagnosis of hyper acute ischemic stroke. Stroke. 2002*;* 33*:* 2206–2210.

63. Schaefer PW, Ozsunar Y, He J et al. Assessing tissue viability with MR diffusion and perfusion imaging. AJNR Am J Neuroradiol. 2003*;* 24*:* 436–443.

64. Hjort N, Butcher K, Davis SM et al. Magnetic resonance imaging criteria for thrombolysis in acute cerebral infarct. Stroke. 2005*;* 36*:* 388–397.

65. Butcher K, Parsons M, Baird T et al. Perfusion thresholds in acute stroke thrombolysis. Stroke. 2003*;* 34*:* 2159–2164.

66. Kidwell CS, Chalela JA, Saver JL, et al. Comparison of MRI and CT for detection of acute Intracerebral hemorrhage. JAMA. 2004*;* 292*:* 1823–1830.

67. Call G. Correlation of colour wave Doppler spectral flow analysis with pathology in carotid stenosis 1988; 19:584.

68. Dainer HM, Smirniotopoulos JG. Neuroimaging of hemorrhage and vascular malformations. Semin Neurol. Sep 2008; 28(4):533-47.

69. Aronovich BD, Reider-Groswasser II, Segev Y. Early CT changes and outcome of ischemic stroke. Eur J Neurol. Jan 2004; 11(1):63-5.

70. Ian M Taveras. Neuroradiology, 3rd ed., Williams and Wilkins, 1990.

71. Silver PL, Nom JW, Levis AS, hachunski VC. Early mortality following Stroke. A prospective review. Stroke 1984; 15: 492.

72. Coronna JJ, Levy DE. Clinical prediction of outcome in ischemic stroke. Neurology Clinics 1983; 1: 103.

73. Wolf PA. Atrial fibrillation. A major contributor to stroke in the elderly: The Framingham Study. Archives of Internal Med 1997; 147: 1561.

74. Toole JF, Xuson CP, Janeway R. Transient ischemic attacks: A study of 225 patients. Neurology 1978; 28: 746.

75. Dalal PM. Japanese Circulation J 48: 621-624. The First Asian Pacific Symposium on Stoke.

76. Poungvarin N, Viriyavejakul A, Komontri C. Siriraj stroke score and validation study to distinguish supratentorial intracerebral hemorrhage from infarction. BMJ. 1991; 302:1565-67

77. Allen CMC. Clinical diagnosis of acute stroke syndrome. Quarterly Journal of Medicine 1983; 52: 515.

78. Daga MK, Sarin K, Negi VS: Comparison of Siriraj Stroke score and Guy's Hospital Score to differentiate supratentorial ischemic and hemorrhagic stroke in the Indian population; JAPI 1994; 42:302-3.

79. Celani MG, Righetti E, Migliacci R, Zampolini M et al. Comparibility and validity of two clinical scores in the early differential diagnosis of acute stroke. Clijnica Neurologica, BMJ 1994;308:1674-6.

80. Jyoti Wadhwani, Riaz Hussain, and PG Raman: Nature of Lesion in Cerebrovascular Stroke patients: Clinical Stroke Score and Computed Tomography Scan Brain Correlation. J Assoc Physicians India 2002;50;777-81.

81. Abayomi S Ogun, Stephen OA Oluwole, Oluremi Aogunseyinde, Abiodun O Fatade et al. Accuracy of Siriraj stroke score in differentiating cerebral haemorrhage and infarction in African Nigerians. African Journal of Neurological Sciences 2001; 20:21-26.

82. Zenebe G, Asmera J, Alemayehu M. How accurate is Siriraj stroke score among Ethiopians? A brief communication. Ethiop Med J 2005; 43:35-38.

83. Hung LY, Wang PY, Wang Y, Chia LG et al. Clinical distinction between acute hemorrhage and acute ischemic stroke by Siriraj Stroke Score, Zhonghua y Zue Za Zhi (Taipei) 1995; 55: 248-52.

84. Efstathiou SP, Tsioulos DI, Zacharos ID, Tsiakou AG, Mitromaras AG, Mastorantonakis SE, et al. A new classification tool for

clinical differentiation between hemorrhagic and ischemic stroke. Journal of internal medicine. 2002; 252(2):121-9.

85. Soman A, Joshi Shashank R, Travade S et al. Greek stroke score, Siriraj score and Allen score in clinical diagnosis of intracerebral hemorrhage and infarct: validation and comparison study. Indian J Med Sci. 2004; 58:417-422

86. Sandercock PA, Allen CM, Corston RN, Harrison MH, Warlow CP. Clinical diagnosis of intracranial haemorrhage using Guy's hospital score. BMJ 1985; 291:1675–7.

87. Hawkins GC, Bonita R, Broad JB, Anderson NE. Inadequacy of clinical scoring systems to differentiate stroke subtypes in population-based studies. Stroke 1995; 26:1338–42.

88. Kochar DK, Joshi A, Agarwal N, Aseri S, Sharma BV, Agarwal TD. Poor diagnostic accuracy and applicability of Siriraj stroke score, Allen score and their combination in differentiating acute hemorrhagic and thrombotic stroke. J Assoc Physicians India. 2000; 48:584–8.

89. Badam P, Solao V, Pai M, Kalantri SP. Poor accuracy of the Siriraj and Guy's hospital stroke scores in distinguishing haemorrhagic from ischemic stroke in a rural, tertiary care hospital. Natl Med J India 2003; 16:8-12.

90. Hui AC, Wu B, Tang AS, Kay R. Lack of clinical utility of the Siriraj stroke score. Intern Med J 2002; 32:311–14.

91. Glen CH, Ruth B, Joanna BB, Neil EA. Inadequacy of Clinical Scoring Systems to Differentiate Stroke Subtypes in Population Based Studies. Stroke. 1995; 26:1338-1342.

92. Huang JA, Wang PY, Chang MC, Chia LG, Yang DY, Wu TC. Allen score in clinical diagnosis of intracranial hemorrhage. Zhonghua Yi Xue Za Zhi. 1994; 54(6):407-11.

93. Berhe T, Zenebe G, Melkamu Y (2009). Application of Greek Stroke Score in Ethiopia. A validation study. Intern. J. Neurol. 1

APPENDIX II

A COMPARATIVE AND VALIDITY STUDY OF ALLEN, SIRIRAJ AND GREEK SCORE IN DIFFERENTIATION OF ACUTE ISCHEMIC AND HEMORRHAGIC STROKE

PROFORMA

NAME: **AGE/SEX:**

I.P.NO: **UNIT:**

OCCUPATION: **ADDRESS:**

COMPLAINTS:

- PRESENTING SYMPTOMS:
- DURATION
- MODE OF ONSET:
- TIME OF ONSET
- APOPLECTICSYMPTOMS:HEADACHE/VOMITING/LOC
- HISTORY SUGGESTIVE OF CRANIAL NERVE INVOLVEMENT
- HISTORY OF SENSORY DISTURBANCES
- H/O BOWEL/BLADDER DISTURBANCES
- H/O FEVER, BLURRING OF VISION, SEIZURES
- H/O CHEST PAIN, PALPITATION, LIMB PAIN

PAST HISTORY

- HT
- DM
- CAD/RHD/ ANY OTHER HEART DISEASE
- TIA/CVA

PERSONAL HISTORY:

- DIET:
- SMOKING:
- ALCHOCOL:

FAMILY HISTORY:

TREATMENT HISTORY:

- H/O INTAKE OF ANTIPLATELET DRUGS: YES/NO
- IF YES INDICATION:

EXAMINATION

GENERAL EXAMINATION

- CONCIOUS LEVEL

AT ADMISSION	24 HOURS AFTER
ALERT/DROWSY/UNCONSCIOUS	ADMISSION

- ORIENTATION
- PALLOR/ICTERUS
- CYANOSIS/CLUBBING
- PEDAL EDEMA
- NEUROCUTANEOUS MARKERS

VITAL SIGNS

- PULSE:
- BP

  AT ADMISSION:

  24 HOURS AFTER ADMISSION:
- RESPIRATORY RATE:
- TEMPERATURE:

SYSTEMIC EXAMINATION:

CNS:

- HIGHER MENTAL FUNCTIONS:
- CRANIAL NERVES EXAMINATION
- MOTOR SYSTEM:
- SENSORY SYSTEM:
- AUTONOMIC NERVOUS SYSTEM:
- CEREBELLAR FUNCTIONS:
- SPINE AND CRANIUM:
- MENINGEAL SIGNS:

CARDIOVASCULAR SYSTEM:

RESPIRATORY SYSTEM:

ABDOMEN:

INVESTIGATIONS

- COMPLETE BLOOD COUNT
- RENAL FUNCTION TEST
- BLOOD SUGAR
- LIVER FUNCTION TEST
- FASTING LIPID PROFILE
- URINE ROUTINE
- BT/CT
- PT/PTT/INR
- CHEST X RAY
- ECG
- ECHOCARDIOGRAM
- CT SCAN BRAIN
- CALCULATION OF ALLEN, SIRIRAJ AND GREEK SCORE.

ALLEN SCORE	SIRIRAJ SCORE	GREEK SCORE	CT SCAN BRAIN REPORT

INSTITUTIONAL ETHICAL COMMITTEE,
STANLEY MEDICAL COLLEGE, CHENNAI-1

Title of the Work	:	Comparative and validity study of GREEK, ALLEN and SIRIRAJ Clinical stroke scores in differentiation of Acute Ischemic and Haemorrhagic Stroke
Principal Investigator	:	Dr. M.Arivumani
Designation	:	PG in MD (Gen. Med.)
Department	:	Department of General Medicine Government Stanley Medical College, Chennai-1

The request for an approval from the Institutional Ethical Committee (IEC) was considered on the IEC meeting held on 06.03.2012 at the Council Hall, Stanley Medical College, Chennai-1 at 2PM

The members of the Committee, the secretary and the Chairman are pleased to approve the proposed work mentioned above, submitted by the principal investigator.

The Principal investigator and their team are directed to adhere to the guidelines given below:

1. You should inform the IEC in case of changes in study procedure, site investigator investigation or guide or any other changes.
2. You should not deviate from the area of the work for which you applied for ethical clearance.
3. You should inform the IEC immediately, in case of any adverse events or serious adverse reaction.
4. You should abide to the rules and regulation of the institution(s).
5. You should complete the work within the specified period and if any extension of time is required, you should apply for permission again and do the work.
6. You should submit the summary of the work to the ethical committee on completion of the work.

05/10/12

MEMBER SECRETARY,
IEC, SMC, CHENNAI

MASTER CHART

S.NO	A	S	COMPLAINTS	HT	AM	HD	T/S	VOM	H.ACHE	APO.ON	C.ADM	C.24	DBP.ADM	DBP.24	WBC	PL	A.S	S.S	G.S	CT
1	70	2	4	1	1	0	0	0	0	0	0	1	80	80	0	1	-12.4	-7	0	1
2	72	1	3	0	0	0	0	0	0	0	1	1	90	90	0	1	-1.4	-3	1	1
3	60	2	1	1	1	0	0	1	1	1	3	3	140	120	0	2	43.6	4	13	2
4	55	F	4	1	0	0	0	0	0	0	2	2	110	100	0	1	4.6	1.5	0	GLIOMA
5	48	1	1	1	1	0	0	0	0	0	1	1	100	90	0	1	-5.1	-5	0	1
6	38	1	2	0	0	1	0	0	0	0	1	1	80	90	0	1	-1.6	-4	0	1
7	68	2	4	1	0	0	0	1	1	1	3	3	110	100	0	1	36.8	6	17	2
8	54	2	3	1	0	1	0	0	0	0	1	1	100	90	0	1	-5.7	-2	0	1
9	45	1	1	0	1	0	0	0	0	1	3	2	140	110	0	1	26.9	6	9	1
10	50	2	3	0	1	0	0	0	0	0	2	1	110	100	0	1	0.7	-1.5	0	1
11	61	1	1	1	0	0	0	0	0	0	1	1	90	100	0	1	0.3	-3	0	1
12	54	2	3	1	0	1	0	0	0	0	1	1	100	90	0	1	-5.7	-5	0	1
13	38	1	1	1	0	0	0	0	0	0	1	1	100	90	0	1	-1.4	-2	3	1
14	40	1	1	0	0	1	0	0	0	0	1	1	70	70	0	1	-5.7	-5	0	1
15	70	2	4	1	0	0	0	0	1	1	3	3	120	110	0	1	38.5	9	13	2
16	62	1	4	0	0	0	0	0	0	0	1	1	90	80	0	1	-4.6	-3	0	1
17	49	1	4	1	1	0	0	0	1	1	3	3	140	130	0	2	45.3	8	13	2
18	42	1	1	1	1	0	0	0	0	0	1	1	110	90	0	1	-5.1	-4	0	1
19	72	2	4	1	1	0	0	0	0	0	2	2	100	90	0	1	2.2	-2.5	0	1
20	62	1	1	0	0	0	0	0	0	0	2	2	90	90	0	1	2.7	-2.5	3	CNS TOX
21	70	1	4	0	0	0	0	0	0	0	2	2	80	80	0	1	8.3	-1.5	0	1

22	62	2	1	0	1	0	0	0	0	0	1	1	100	100	0	1	0.7	-5	0	1
23	65	2	1	1	1	0	0	0	0	0	1	1	100	100	0	1	-1.7	-5	0	1
24	85	2	3	1	1	0	0	0	1	1	3	3	120	110	0	1	34.8	6	9	1
25	56	2	3	0	0	1	0	0	0	0	2	2	80	70	0	1	2.3	-4.5	3	1
26	59	1	4	1	0	0	0	0	0	0	2	2	110	100	0	1	7.6	1.5	3	1
27	72	2	1	1	1	0	0	0	0	0	1	1	100	90	0	1	-5.1	-5	0	1
28	74	2	4	0	1	0	0	0	0	0	1	1	110	100	0	1	0.7	-4	0	1
29	52	1	3	1	1	0	0	0	0	0	1	1	90	90	0	1	-5.1	-6	0	1
30	68	2	2	1	0	0	0	0	0	1	3	3	140	120	0	2	47.3	8	13	2
31	55	1	1	0	0	1	0	0	0	0	1	1	80	90	0	1	-1.6	-7	0	1
32	45	1	4	0	0	1	0	0	0	0	1	1	100	100	0	1	0.09	-2	0	1
33	75	1	4	0	1	0	0	1	0	1	3	3	100	100	1	2	37.2	2	13	2
34	65	1	4	1	1	0	0	0	0	0	1	1	90	100	0	1	-3.4	-6	0	1
35	50	2	3	1	0	0	1	0	0	0	1	1	80	80	0	1	-9.5	-4	0	1
36	47	2	2	0	0	0	0	0	0	0	2	2	80	80	0	1	8.3	-2.5	3	1
37	60	1	2	1	1	0	1	0	0	1	3	3	100	90	0	1	24.7	4	13	1
38	62	1	2	0	0	0	0	0	0	0	1	1	80	80	0	1	1	-1.5	0	CVT
39	45	1	3	1	0	0	1	0	0	0	1	1	110	110	0	1	-4.7	-4	0	1
40	44	1	4	0	0	0	0	0	0	0	1	1	80	80	0	1	1	-4	0	1
41	58	1	2	1	1	0	0	0	0	0	1	1	100	90	0	1	-5.1	-5	0	1
42	68	2	1	1	0	0	1	0	0	1	3	3	110	100	0	1	30.1	8	9	1
43	32	1	1	0	0	0	0	0	0	0	1	1	90	80	0	1	1	-3	0	1
44	57	1	2	1	0	0	0	0	0	1	3	3	140	120	0	2	47.3	11	13	2
45	48	1	4	0	1	0	0	0	0	0	1	1	100	90	0	1	-0.9	-5	0	1

46	62	2	1	1	1	0	0	0	0	0	1	1	120	100	0	1	-3.4	-3	0	1
47	70	2	4	1	0	0	0	0	0	0	1	1	110	110	0	1	2	-1	0	1
48	49	1	4	0	0	1	0	0	0	0	1	1	90	80	0	1	-3.3	-6	0	1
49	66	1	3	0	1	0	0	0	0	0	1	1	100	90	0	1	-0.9	-5	0	1
50	72	2	4	1	1	0	0	0	0	1	3	3	130	110	0	2	41.9	5	13	2
51	50	1	2	0	0	0	0	0	0	0	1	1	90	90	0	1	2.7	-3	0	1
52	62	1	4	1	0	0	0	0	0	1	3	3	110	110	1	2	45.6	6	9	2
53	58	1	3	0	1	0	0	0	0	0	1	1	100	100	0	1	0.7	-5	0	1
54	54	1	2	0	0	0	0	0	0	0	3	3	100	100	0	1	19	3	9	SCHW
55	70	1	3	1	0	0	0	0	0	0	1	1	100	110	0	1	2	-2	0	1
56	68	1	3	0	0	1	0	0	0	0	1	1	100	90	0	1	-1.6	-2	0	1
57	39	1	1	0	1	0	0	0	0	0	2	2	90	90	0	1	6.3	-6	3	1
58	37	1	1	0	0	0	0	0	0	0	1	1	100	90	0	1	2.7	-2	0	1
59	57	1	1	0	0	0	0	0	0	0	1	1	80	80	0	1	1	-1.5	3	T.CLOMA
60	60	1	4	1	1	0	0	0	0	1	3	2	110	100	0	1	25.8	4	9	1
61	56	2	3	1	1	0	0	0	0	0	1	1	90	80	0	1	-3.1	-3	0	1
62	50	2	1	0	0	1	0	0	0	0	1	1	80	80	0	1	-3.3	-7	0	1
63	70	1	2	1	1	0	0	1	1	1	3	3	120	110	0	1	34.8	6	13	2
64	55	2	2	1	1	0	0	0	0	0	2	2	80	90	0	1	2.2	-4.5	3	1
65	62	1	4	1	1	0	1	0	0	0	3	3	90	90	0	1	9.7	-2	3	1
66	46	1	3	0	0	0	0	0	0	0	1	1	100	90	0	1	2.7	-2	0	1
67	28	1	1	0	0	0	0	0	0	0	3	3	100	100	0	1	19	3	9	1
68	40	1	2	0	0	0	0	0	0	0	1	1	100	100	0	1	4.4	-2	0	1
69	33	1	3	0	0	1	0	0	0	0	1	1	100	90	0	1	-1.6	-2	0	1
70	41	1	1	1	1	0	0	0	0	0	1	1	100	100	0	1	0.3	-2	0	1

71	69	1	4	1	1	0	0	0	0	0	1	1	90	80	0	1	-6.8	-6	0	1
72	63	1	3	0	0	0	0	0	0	0	1	1	80	90	0	1	-0.9	-7	0	1
73	89	1	2	1	1	0	1	0	0	0	1	1	90	90	0	1	-11.8	-7	0	1
74	48	1	3	0	0	0	0	0	0	0	1	1	90	90	0	1	2.7	-3	0	1
75	34	1	3	0	0	0	0	0	0	0	1	1	80	80	0	1	1	-4	0	1
76	52	1	4	0	0	0	0	0	0	1	3	2	100	80	0	1	30.2	7	13	2
77	46	2	1	1	1	0	0	0	0	0	2	2	100	90	0	1	2.2	-2.5	3	1
78	60	1	2	0	0	0	0	0	0	0	1	1	90	90	0	1	2.7	-3	0	1
79	65	2	3	1	0	0	0	0	0	0	1	1	100	100	0	1	0.3	-2	0	1
80	55	2	1	0	0	0	0	0	0	0	1	1	90	80	0	1	1	-3	0	1
81	70	2	4	0	0	0	0	0	0	0	2	2	100	100	0	1	11.7	0.5	0	1
82	65	2	3	1	0	0	0	0	0	0	1	1	80	80	0	1	-3.1	-4	0	1
83	38	1	3	0	0	0	0	0	0	0	1	1	100	90	0	1	2.7	-2	0	1
84	80	1	2	1	0	0	0	0	0	1	3	3	110	110	0	1	38.5	8	13	2
85	65	2	3	1	0	0	0	0	0	0	1	1	100	100	0	1	0.3	-2	0	1
86	80	1	2	1	0	1	0	0	0	0	1	1	110	100	0	1	-4	-4	0	1
87	41	2	1	0	0	1	0	0	0	0	1	1	100	90	0	1	-1.6	-2	0	1
88	54	1	3	1	1	0	0	0	0	0	2	2	100	100	0	1	3.9	-2.5	0	1
89	63	1	4	1	1	0	0	0	0	1	3	2	120	110	0	1	27.5	6	13	1
90	49	2	2	0	1	0	0	0	0	0	1	1	100	90	0	1	-4.3	-5	0	1
91	62	1	1	1	0	0	0	0	0	0	1	1	110	110	0	1	2	0	3	2
92	58	2	2	0	1	1	0	0	0	0	2	1	110	100	0	1	-3.6	-1.5	3	1
93	72	1	4	1	1	0	0	0	1	1	3	3	140	120	1	1	43.6	8	13	2

94	44	1	3	0	0	0	0	0	0	0	1	1	100	90	0	1	2.7	-2	0	1
95	68	2	1	1	0	0	1	0	0	0	2	1	100	90	0	1	-8.1	-2	3	1
96	45	1	4	0	0	0	0	0	0	0	1	1	90	90	0	1	2.7	-3	0	1
97	30	2	3	0	0	0	0	0	0	0	1	1	90	80	0	2	1	-3	0	1
98	48	1	4	0	1	0	0	0	0	0	1	1	90	90	0	1	-0.9	-6	0	1
99	67	2	2	1	1	0	0	1	1	1	3	3	130	110	0	1	34.8	3	13	2
100	56	1	3	1	0	0	0	0	0	0	1	1	100	90	0	1	-1.4	-2	0	1

KEY TO MASTER CHART

1	S.NO-SERIAL NUMBER	
2.	AGE	
3.	SEX	1-MALE 2-FEMALE
4.	COMPLAINTS	1.RIGHT HEMIPARESIS 2.RIGHT HEMIPLEGIA 3.LEFT HEMIPARESIS 4.LEFT HEMIPLEGIA
5.	HT-HYPERTENSION	0 – ABSENT 1 – PRESENT
6.	AM- ATHEROMA MARKERS	0 – ABSENT 1 – PRESENT
7.	HD- HEART DISEASE	0 – ABSENT 1 – PRESENT
8.	T/S- PAST HISTORY OF TIA/STROKE	0 – ABSENT 1 – PRESENT
9.	VOM-VOMITING	0 – ABSENT 1 – PRESENT
10.	HEADACHE	0 – ABSENT 1 – PRESENT
11.	APO.ON- APOPLECTIC ONSET	0 – ABSENT 1 – PRESENT
12.	C.ADM- CONSCIOUS LEVEL AT ADMISSION	1 – ALERT 2 – DROWSY 3 – UNCONCIOUS
13.	C.24- CONSCIOUS LEVEL AT 24 HOURS	1 – ALERT 2 – DROWSY 3 – UNCONCIOUS
14.	D.BP ADM- DIASTOLIC BLOOD PRESSURE AT ADMISSION	
15.	DBP.24-DIASTOLIC BLOOD PRESSURE AT 24 HOURS	
16.	WBC- WBC COUNT	0 - < 12000 1 - > 12000
17.	PL-PLANTAR	1 – UNILATERAL EXTENSOR 2 – BILATERAL EXTENSOR
18.	A.S- ALLEN SCORE	<4 – INFARCT 5-24 – EQUIVOCAL >24 – HEMORRHAGE
19.	S.S-SIRIRAJ SCORE	< -1 – INFARCT -1 TO +1 – EQUIVOCAL > 1– HEMORRHAGE
20.	G.S- GREEK SCORE	< 3– INFARCT 3-12– EQUIVOCAL >OR = 13 – HEMORRHAGE
21.	CT- CT SCAN BRAIN RESULT	1 – INFARCT 2 – HEMORRHAGE GLIOMA CNS TOX – CNS TOXOPLASMOSIS SCHW – SCHWANOMA CVT - CORTICAL VEIN THROMBOSIS T.CLOMA - TUBERCULOMA