DISSERTATION

ON

CLINICAL PROFILE, NEUROIMAGING EVALUATION and TREATMENT OUTCOME OF ACUTE ISCHEMIC STROKE PATIENTS

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CERTIFICATE

This is to certify that this dissertation entitled **"CLINICAL PROFILE, NEUROIMAGING EVALUATION and TREATMENT OUTCOME OF ACUTE ISCHEMIC STROKE PATIENTS"** submitted by **Dr. G. MUGUNTHAN** appearing for **D.M. Neurology** Degree (Branch - I) examination in **August 2009** is a bonafide record of work done by him under my direct guidance and supervision in partial fulfillment of regulations of the Tamil Nadu Dr. M.G.R. Medical University, Chennai. I forward this to the Tamil Nadu Dr.M.G.R. Medical University, Chennai, Tamil Nadu, India.

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DECLARATION

I solemnly declare that the dissertation titled "**CLINICAL PROFILE, NEUROIMAGING EVALUATION and TREATMENT OUTCOME OF ACUTE ISCHEMIC STROKE PATIENTS"** is done by me at Institute of Neurology, Madras Medical College & Govt. General Hospital, Chennai, during 2007-2009 under the guidance and supervision of **Prof. V. NATARAJAN, M.D., D.M.,**

The dissertation is submitted to The Tamil Nadu Dr. M.G.R. Medical University towards the partial fulfillment of requirements for the award of **D.M., degree in Neurology.**

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CONTENTS

INTRODUCTION

The World Health Organisation defines the Stroke in an adult as **"**rapidly developing clinical symptoms and signs of focal (at times global) disturbance of cerebral function, lasting for more than 24 hours or leading to death with no apparent cause other than that of vascular origin" [2]

Recent data has shown that about 87% of strokes are ischemic and 13% is due to hemorrhage (intracerebral or subarachnoid) [1]

Stroke is a huge public health concern because of its high morbidity and disability. It is the second commonest cause of mortality and the most common cause of morbidity.

The prevalence rate of stroke in India is 545 per 100,000 [3]. Recent studies showed that the age-adjusted annual incidence rate in India is 105/100,000 in the urban community and 262/100,000 in a rural community [4] [5].

Incidence of stroke in United States is 200 / 100000 population. It is age related, relatively uncommon before the age of 50 but doubling every decade after the age of 55. It is more common in men. Most acute stroke fatalities occur in the first 30 days after onset. 8 to 12% of ischemic strokes result in death within 30 days. [6]

The Clinical features of the ischemic stroke depend upon the extent and severity of involvement of specific arteries. Time and Mode of onset of the stroke might help us to determine the aetiology. A large number of risk factors for stroke have been described, a reflection of the heterogeneity of the disease.

Only 2% of ischemic strokes enter into the medical attention with in the golden time window i.e. three hours of onset of stroke.

Neuroimaging, especially CT Scan Brain and MRI Brain helps to confirm the ischemic strokes, rule out the stroke mimics, find out the involved arterial territory and the extent & pattern of involvement.

Another novelty is that stroke is no more considered as unavoidable and untreatable. There is now a clear consensus that stroke is an emergency and that specialized units and teams will improve the outcome and may lower the costs.

> The role of heparin in the management of acute ischemic stroke is still controversial. Many studies continue to show no proven benefits.

In this study, the clinical profile, risk factors, neuroimaging assessment of infarcts and treatment outcome of the patients with acute ischemic strokes were analyzed.

Aims of the study

1. To analyze the risk factors and clinical profile of acute ischemic stroke.

2. To analyze the pattern of infarcts and their regional distribution based on neuroimaging.

3. To assess the treatment outcome of the acute ischemic stroke patients.

REVIEW OF LITERATURE

Stroke is the rapid development of a focal neurologic deficit caused by a disruption of blood supply to the corresponding area of brain. The brain, in contrast with other organs, localizes specific functions to particular regions. Hence occlusion of an artery supplying a small area of brain has a profound and specific effect.

EPIDEMIOLOGY:

Annually, 15 million people worldwide suffer a stroke. Of these, 5 million die and another 5 million are left permanently disabled, placing immense burdens on family and community. The World Health Organization (WHO) estimates that a stroke occurs every 5 seconds [7]. In 2005, it accounted for approximately 10% of all deaths worldwide. Globally, stroke is the second leading cause of death [8]. More than 80% of strokes occur in the developing countries.

Risk factors for ischemic stroke:

Generally, risk factors for stroke can be classified as modifiable and non modifiable (Sacco et al., 1977) [9]. Non modifiable risk factors for stroke are important to detect, even if no measure can be taken to eliminate them, because their presence helps to identify individuals at higher risk and thus justifies the implementation of vigorous treatments to reduce the modifiable risk factors.

Non- modifiable risk factors:

a) Age
b) Gender
c) Family history
d) Race/Ethnicity
e) Genetics

Modifiable risk factors:

a) Hypertension,
b) TIA and Prior stroke
c) Cardiac disease
d) Diabetes Mellitus
e) Cigarette smoking
f) Dyslipidemia
g) Alcohol consumption
h) Asymptomatic carotid artery disease
i) Aortic arch atheromatosis
j) Elevated Homocysteine
k) Elevated anticardiolipin antibodies
l) Oral contraceptives use
m) Obesity

Non- modifiable risk Factors:

AGE:

Increasing age is the most powerful and important risk factor for stroke. The incidence of stroke doubles each decade past 55 years of age for both men and women (Brown et al., 1966: Wolf et al., 1992) [10], [11]. Half of all strokes occur in people older than 70 to 75 years in the western world.

SEX:

The incidence of ischemic strokes is more common in males in all age groups, whereas ischemic infarcts are more common in females beyond 6o years of age. It is probably due to protective benefits of reproductive age group and increased prevalence of vascular risk factors in males (Bogousslavsky et al 1988)[12], (Brown et al) [13].

FAMILY HISTORY:

It plays a minor role in the pathogenesis of cerebral infarct. However, increased risk is seen with a family history of stroke among first degree relatives. In the Framingham study, parental history of stroke or coronary artery disease constituted a risk factor for stroke [Kiely et al., 1993][14].

RACE & ETHNICITY:

Generally, the incidence of stroke appears to be higher in non-Caucasians than in Caucasians [Sacco ct al., 1997] [9].

Modifiable risk Factors:

HYPERTENSION:

In middle and late adult life, hypertension is undoubtedly the strongest modifiable risk factor for ischemic stroke. Hypertension is present in approximately 60 - 70% of ischemic stroke cases. The risk of stroke rises in proportion to blood pressure, for males as well as for females, and almost doubles for every 7.5 mm Hg increment in diastolic blood pressure (DBP) (Collins and McMahon, 1994) [15].

In a meta-analysis (Prospective Studies Collaboration, 1995) [16], the relative risk for developing stroke between the highest and the lowest quintiles in DBP was tenfold, fivefold and twofold for individuals aged at the time of screening <45, 45-64 and > 65 years, respectively. The relationship between systolic blood pressure, including ‘isolated systolic hypertension’, may be even stronger than for DBP (Shaper et al., 1991; Keli et al., 1992) [17].

DIABETES MELLITUS:

Diabetes is associated with stroke, independently of the various cardiovascular risk factors which usually accompany this disease (hypertension, dyslipidemia and obesity). It increases the risk of ischemic cerebrovascular disease two to four fold (Weinberger et al and).

Stroke is due to atherosclerosis, cardiac embolism and rheological abnormalities.

SMOKING :

Cigarette smoking is an independent risk factor for ischemic stroke in men and women of all ages. In their meta-analysis, Shinton and Beevers (1989) [18] estimated that the relative risk of stroke for smokers and former smokers, as compared to non smokers, was 1.5 and 1.17, respectively. The risk for stroke is two to three times greater than in non-smokers.

The risk of stroke increased in proportion to the number of cigarettes smoked per day and was higher for women as compared to men. It is due to enhanced atherogenesis, reduced capacity of the blood to deliver oxygen, cardiac arrhythmias, and arterial spasm. Homer and Ingall have documented the importance of long duration cigarette smoking in the development of carotid atherosclerosis.

ALCOHOLISM:

There is J shaped association between alcohol consumption and ischemic stroke. Light use up to one or two drinks per day evenly distributed throughout the week offers a reduced risk, whereas moderate to heavy drinking is associated with an increased risk for stroke.

DYSLIPIDEMIA:

High total cholesterol and high levels of LDL lead to atherosclerosis. But the direct association of total cholesterol levels with cerebrovascular disease is less clear. In a large meta-analysis of 45 prospective cohorts including 13,000 strokes (Prospective Studies Collaboration. 1995) [16], plasma total cholesterol levels were highly significantly associated with the risk of developing stroke, but only in the subset of individuals aged <45 at the time of screening. In contrast, no association was observed for older groups.

Recent Meta analyses however have suggested that ischemic stroke risk increases with increasing serum cholesterol and the reduction in stroke risk is associated with statin therapy. (Amarenco et al 2004, Tirschwell et all 2004)[20]. Prospective studies and interventional studies (Crouse et al., 1997) [21] using highly effective lipid-lowering agents show that reducing cholesterol levels in plasma significantly decreases the risk of stroke.

ATRIAL FIBRILLATION:

Chronic non-valvular atrial fibrillation is associated with an over all risk for stroke of approximately five to six fold and above 18 fold if there is associated rheumatic heart disease. The prevalence of AF increases with advancing age and is 0.5% for patients aged 50 -59 years

and 8.8% for those aged 80 – 89 years. 70% of individuals with AF are between 65 and 85 years of age. It accounts for two thirds of cardiac emboli.

Blood supply of the brain:

Brain is supplied by two large arteries i.e. Internal carotid artery and Vertebrobasilar artery.

MIDDLE CEREBRAL ARTERY:

It originates from internal carotid artery. It has superficial and deep hemispherical branches. The superficial cortical branches supply lateral surface of cerebral hemisphere (Frontal, Parietal and temporal lobes). Deep hemispherical branches are of two types, (a) deep penetrating vessels from the cortical arteries, (b) Lenticulo striate branches from MCA stem. Deep penetrating vessels supply fronto parieto temporal sub cortical white matter. Lenticulo striate branches supply the putamen, head & body of caudate nucleus, globus pallidus, posterior limb of internal capsule and corona radiata.

ANTERIOR CEREBRAL ARTERY:

It also arises from internal carotid artery and supplies the anterior three quarters of the medial surface of cerebral hemisphere, orbito frontal cortex, a strip of the lateral surface of the cerebral hemisphere along the superior border. Deep branches supply anterior limb of internal capsule and head of caudate.

POSTERIOR CEREBRAL ARTERY:

It arises from the rostral end of the basilar artery. P1 segment gives rise to thalamo perforating arteries to midbrain and thalamus. P2 segment gives rise to thalamo geniculate artery supplying ventroposteromedial and lateral nucleus of thalamus, pial branches to medial temporal cortex and calcarine arteries to medial occipital cortex.

Vertebral artery:

They supply the lower three fourths of the pyramid, the medical lemniscus in medulla, restiform body and postero inferior part of cerebellar hemisphere.

Basilar artery:

It gives rise to paramedian perforators, short and long circumferential arteries. They supply pons and mid brain either directly or indirectly through AICA & SCA, which also originate from basilar artery.

POSTERIOR INFERIOR CEREBELLAR ARTERY:

It usually arises from vertebral artery and supplies the inferior cerebellar peduncle, dorsolateral tegmentum of medulla, inferior surface of vermis and adjacent cerebellar hemispheres.

ANTERIOR INFERIOR CEREBELLAR ARTERY:

It originates from basilar artery and supplies the flocculus, inferior surface of cerebellar

hemispheres, middle cerebellar peduncle and lateral pontine tegmentum.

SUPERIOR CEREBELLAR ARTERY:

It starts from basilar artery and supplies lateral tegmentum of midbrain, superior cerebellar peduncle, superior surface of cerebellum and cerebellar nuclei.

Clinical features of ischemic stroke:

CAROTID TERRITORY INFARCTS:

They include combination of MCA and ACA syndrome. In addition amaurosis fugax is the sole feature that distinguishes the carotid artery syndrome from MCA syndrome.

MIDDLE CEREBRAL ARETRY SYNDROME:

It is one of the most common manifestations of cerebrovascular disease. MCA territory was involved in more than two-thirds of all infarcts (Lausanne stroke registry, 1988) [12]. The clinical features are varied and depend on whether the site of occlusion is in the stem, superior division, inferior division or lenticulo striate branches and whether there is good collateral flow.

Stem occlusion of MCA:

There is a large hemispheric infarction with contra lateral hemiplegia, conjugate eye deviation towards the side of the infarct, hemianesthesia and homonymous hemianopia. Associated global aphasia occurs if dominant hemisphere is involved and hemi neglect with non-dominant hemispheric lesions.

SUPERIOR DIVISION OF MCA INFARCT:

It produces more weakness of face and arm, compared to leg. Broca's aphasia is common with superior division infarct in dominant hemisphere.

INFERIOr division of MCA INFARCT:

Wernicke's aphasia is seen with dominant hemisphere infarction and behavioural disturbances are seen with non dominant infarcts.

Gerstmann's syndrome consisting of acalculia, agraphia, finger agnosia, right – left disorientation is seen with dominant parietal hemisphere lesion.

Anosognosia or denial of hemiparesis is commonly seen in right parietal hemisphere strokes.

Lenticulo striate branch occlusion

They produce lacunar syndromes or striatocapsular infarcts depending upon the number of vessels involved.

Lacunar Syndromes:

Ischemic strokes resulting from small vessel or penetrating artery disease have unique clinical, radiological and pathological features. Lacunar infarcts are small ischemic infarctions in the deep regions of brain or brain stem that range in diameter from 0.5 to 15 mm.

These infarctions result from occlusion of penetrating arteries, chiefly middle cerebral, posterior cerebral and basilar arteries. They are mostly due to atherothrombosis and lipohyalinosis and less commonly due to embolism. At least 20 lacunar syndromes have been described (Miller Fisher et al) [22].

Five best recognised syndromes are (1) Pure motor Hemiparesis, (2) Pure sensory stroke, (3) Sensory motor stroke, (4) Ataxic Hemiparesis, (5) Dysarthria – clumsy hand syndrome.

Pure motor Hemiparesis is often caused by lacune in internal capsule, corona radiata and basis pontis. Contra lateral Hemiparesis accompanied by dysarthria at onset occurs. There should be no aphasia, apraxia, agnosia or higher cortical disturbances. [23] [24]

Pure sensory stroke is caused by a lacune involving the ventroposterolateral nucleus of the thalamus and post central gyrus. [25]

Sensory motor stroke is caused by a lacune involving internal capsule and thalamus or posterior limb of internal capsule. [24]

Ataxic Hemiparesis is often caused by a lacune in posterior limb of internal capsule (23-39%) and pons (19-31%) followed by corona radiata (13-31%) and thalamus (11-13%). [23] [25]

Dysarthria - Clumsy hand syndrome is often caused by a lacuna involving the deep areas of the basis pontis.

ANTERIOR CEREBRAL ARTERY syndrome:

ACA infarctions are uncommon, constituting 0.6% to 3% of total infarcts. The clinical features of ACA infarct vary according to the site of involvement and the extent of collateral blood flow. Contra lateral weakness involving primarily the lower extremity and sphincter incontinence are characteristic. Other features include abulia, akinetic mutism, impaired memory, emotional disturbance and paratonia.

Vertebrobasilar TERRITORY syndromes:

POSTERIOR CEREBRAL ARTERY SYNDROME:

The manifestations of PCA infant are variable, depending on the site of the occlusion and the availability of collateral blood flow. Occlusion of pre communal P1 segment causes mid brain, thalamic and medial occipital hemispheric infarction. Post communal occlusion causes thalamic and hemispheric infarction or isolated hemispheric infarction.

Occlusion of the calcarine artery results in medial occipital and medial temporal lobe infarction. It causes contra lateral homonymous hemianopia.

Thalamic infarcts produce varied manifestations depending on the arterial territories involved. Postero lateral thalamic infarction due to P2 segment PCA occlusion result in sensory stroke, sensorimotor stroke and thalamic syndrome of Dejerine - Roussy. Anterior thalamic infarctions due to occlusion of polar artery lead to neuropsychological disturbance, emotional facial paresis and visual field deficit.

Paramedian infarctions due to involvement of thalamo perforators from P1 PCA result in altered sensorium, memory loss and vertical gaze abnormalities.

Lesions involving the thalamus and posterior cerebral hemisphere accounted for 37% of cases, whereas 63% of patients had infarcts that were entirely hemispheric (Goto eta al 1979) [26].

PICA syndrome:

It affects inferior part of vermis, vestibulocerebellum and major part of lateral cerebellar hemispheres. It results in prominent vertigo, ataxia, nystagmus and limb dysmetria. It can also cause Wallenberg's (lateral medullary) syndrome.

AICA SYNDROME:

It causes ventral cerebellar infarction. The clinical features include vertigo, nausea and nystagmus. They also produce ipsilateral facial hypalgesia, facial paralysis and deafness due to involvement of pontine tegmentum.

SCA Syndrome:

It results in dorsal cerebellar syndrome. Ipsilateral ataxia, limb incoordination, severe intention tremor are more common than vertigo.

Top of basilar syndrome:

It is caused by occlusive vascular disease, often embolic in nature of rostral basilar artery. It result in behavioural abnormalities, somnolence, peduncular hallucinosis, memory disturbances or agitated delirium, ocular abnormalities.

BORDER ZONE (WATERSHED) ischemic syndromes:

Low flow infarction also called border zone infarctions are the result of critically reduced cerebral perfusion pressure in far downstream brain arteries that leads to a critically reduced cerebral blood flow and oxygen supply in certain vulnerable brain areas.

They occur in the border zone between adjacent arterial perfusion beds. Cardiac arrest, sustained and severe arterial hypotension, prolonged hypoxemia and bilateral severe carotid artery disease or occasionally vertebrobasilar artery disease leads to ischemia in the watershed areas between the major circulations. They are occasionally caused by microembolism or hyperviscosity states. Border zone infarctions account for 5 to 10% of all ischemic infarcts

(Bernd Ringelstein) [27]. Bogousslavsky and regli et al [12] reported than 5.2% of all strokes are sub cortical border zone infarcts and 6% in Bladen et al series [28].

They are of two types; A) Cortical or External border zone infarcts, B) Deep or Internal border zone infarcts.

Cortical or External watershed Infarcts:

Zone of ischemia lies between the pial vessels of two vascular territories like MCA & ACA, MCA & PCA. Ischemia between ACA and MCA territories causes infarction in the high parietal and frontal cortex. The weakness of limbs is more in the shoulder and hip joint than the hand and faces. Ischemia in the border zone of MCA and PCA leads to parieto temporo occipital junction infarcts.

Internal or deep border zone infarcts:

Zone of ischemia lies between the territories of the lenticulo striate branches and the medullary penetrating vessels from the convexity. It affects mainly the sub cortical frontoparietal white matter especially centrum semiovale. It can't be called as watershed because these two arteries are terminal and don't anastomose with each other.

Diagnostic evaluation

Neuro imaging:

CT BRAIN:

Non enhanced cranial CT scan is done in all patients because it may detect haemorrhage or mass lesions that can present as ischemic stroke. Early CT signs of ischemic stroke in the MCA territory, such as loss of grey - white matter differentiation, insular ribbon sign, sulcal effacement, effacement of the sylvian fissure and obscuration of lentiform nucleus are very important. Dense MCA sign (Hyper density in the horizontal part of the MCA in NECT) can be seen in few patients before the infarction becomes visible. It indicates thrombotic or embolic occlusion of MCA stem predicting a large cortical infarct.

On admission, CT brain is negative in approximately one third of patients in whom ischemic stroke has been diagnosed clinically. This could be due to taking CT scan early before obvious tissue damage and changes in density of lesion occurs.

CT Brain may not detect relatively small infarcts in Vertebrobasilar system, infarcts near base, infarct <5mm in diameter and infarcts with mild oedema.

MRI Brain:

It produces images that are more detailed and clear than those of CT brain and it provides more information about tissue characteristics. It can detect infarct in early stages, especially with DWI imaging, infarcts in posterior fossa structures and small infarcts.

Blood glucose:

Fasting blood glucose, glycosylated haemoglobin are good indicators of diabetes mellitus control.

Lipid profile:

Since LDL-cholesterol levels <100 mg/dl throughout life are associated with a very low risk for CHD in populations, they can be called *optimal.* Even when LDL-cholesterol concentrations are *near optimal* (100–129 mg/dl), atherogenesis occurs; hence, such levels must also be called *above optimal.* At levels that are *borderline high* (130–159 mg/dl), atherogenesis proceeds at a significant rate, whereas at levels that are *high* (160–189 mg/dl) and very high (≥190 mg/dl) it is markedly accelerated. Similarly, triglycerides are classified in to three categories. Normal: < 150 mg/dl, Borderline high: 150 – 199 mg/dl, High: 200 - 399 mg/dl. HDL cholesterol values are classified in to two categories, Low < 40 mg/dl and Normal > 40 mg/dl. (National Cholesterol Education Program Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults - Adult Treatment Panel III, 2002) [35].

ELECTROCARDIOGRAM:

The heart should be assessed in all patients with stroke. ECG may reveal evidence of myocardial ischemia or infarction, arrhythmias especially atrial fibrillation, Left Ventricular Hypertrophy.

EchoCARDIOGRAM:

They are relative safe methods of evaluating cardiac anatomy and structural problems. It allows the detection of potential cardiogenic sources of cerebral embolism such as left atrial thrombi, atrial septal aneurysm, patent foramen ovale with right to left shunt, valvular heart disease, cardiac failure and cardiomyopathy. It can also detect mitral valve prolapse, hypokinetic left ventricular segment and left ventricular dysfunction and hypertrophy.

Individuals with mitral annular calcifications have two fold risk for stroke compared to those without. MVP stroke risk is more among men older than 50 years with thick mitral leaflet on echocardiography and with mitral regurgitation.

Transoesophageal Echocardiography is more accurate and detects structural problems better compared to Trans thoracic Echocardiography.

ROLE OF HEPARIN IN THE TREATMENT OF ACUTE ISCHEMIC STORKE:

Heparin is heterogenous mixture of sulfate and mucopolysaccharides. It activates antithrombin III and inhibits regulation factors II, IX, X, XI & XII and also blocks the conversion of fibrinogen to fibrin. It accelerates fibrinolysis and inactivates thrombin. The anticoagulant effect is quantified based on activated partial thromboplastin time. The therapeutic range is 1.5 to 2.0 times the normal control value. LMWH exerts its anticoagulation effect in a more selective pattern, affecting almost exclusively the intrinsic clotting pathway. Conventional heparin can be given subcutaneously or continuous intravenous infusion. SC heparin dose ranges from 10000 – 25000 IU / day.

In a randomised double blind controlled trial of LMWH (Fraxiparin), 312 patients were randomized within 48 hours of stroke to receive Fraxiparin 4100 IU subcutaneously either once or twice daily or placebo. It was continued for 10 days. There was no difference between the groups at 3 months. However after 6 months, there was a significant dose dependant reduction in the rate of poor outcome among the three study groups in favour of patients treated with Fraxiparin twice daily(Kay et al 1995) [36]. Second, randomised double blind study involved 750 patients in 120 centres failed to confirm these observations.

International stroke trial (IST) studied approximately 20,000 patients who were randomized within 48 hours of ischemic stroke onset to receive a fixed dose of 10,000 or 25,000 units of unfractionated heparin subcutaneously daily and compared with no heparin. Treatment was continued for 14 days or until hospital discharge if shorter. There was no significant difference in the rate of death or disability. Patients receiving heparin had significantly fewer recurrent ischemic strokes at 2 weeks, but this was negated by a similar increase in hemorrhagic stroke (International stroke trial collaborative Group 1997). [37]

Definite data regarding the safety and efficacy of intravenous heparin for acute ischemic stroke or cardio embolic stroke are lacking, but intravenous heparin is sometimes given to some patients with small cardio embolic infarcts associated with intra cardiac thrombi diagnosed by ECHO to prevent recurrence.

Conventional heparin appears to be ineffective in patients with acute partial stable stroke. A large randomized study evaluated the use of heparin in 225 patients with non cardio embolic stroke in which there was no significant difference in stroke progression or death at 7 days.

Other Low molecular weight heparin and heparinoids also remain unproven in acute ischemic stroke. The overall results of the TOAST study utilizing danaparoid for patients with ischemic stroke treated within 24 hours of symptom onset showed no benefit for anticoagulation (TOAST, 1998) [38].

Although convincing statistical proof is still lacking, anecdotal evidence supports early initiation of intravenous or subcutaneous conventional heparin to prevent the stroke recurrence in many situations. They include cardiac emboli, cerebral infarct with echo evidence of intramural thrombus, stroke in evolution, progressive or recent ischemic symptoms, cresendo TIA, fluctuating basilar artery thrombosis, intra luminal arterial thrombus, extracranial cervicocephalic arterial dissection or hyper coagulable states.

The role of heparin in preventing stroke in patients with TIA or recent stroke and preventing the progression of an acute stroke has never been answered satisfactorily. The most recent American stroke Association Guidelines for the early treatment of patients with ischemic stroke recommends the subcutaneous administration of heparin to prevent deep venous thrombosis.

Hemorrhagic complications are the most frequent side effects of heparin therapy. The frequency of ICH is between 1-7% and is higher in patients with large ischemic stroke. Heparin is unsafe and contraindicated in patients with large cerebral infarcts, infarcts with hemorrhagic transformation, bleeding peptic ulcer, uremia, hepatic failure, elevated BP >220/120mmHg or sepsis.

TREATMENT OUTCOME & MORTALITY RATE:

The prognosis for functional recovery after an ischemic stroke is influenced by various clinical and medical factors. The main predictors are age, co morbid illnesses, sex, severity of the initial deficit, severity and size of the infarct, aetiology and location of stroke, time interval from the onset to reach the hospital and the type of medical care, including stroke unit, stroke team, and stroke pathway.

Patients with acute ischemic stroke are at high risk of neurologic and medical complications like space occupying oedema formation, hemorrhagic transformation of infarct, epileptic seizures, infections, aspiration and venous thromboembolism. Most complications occurred during the first 4 days after admission. They should be monitored closely for early detection of these complications.

In population based studies, case fatality rates in the first month of ischemic stroke range between 8% and 15%. The commonest causes of death in the first few days are cerebral oedema leading to herniation, infections, myocardial infarction, arrhythmias and respiratory failure.

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SUBJECTS AND METHODS

This study was conducted during the period of February 2007 to January 2009. It was done among the patients admitted to Government General Hospital, Chennai.

All the patients above the age of 15 years with clinical features suggestive of stroke were taken. All were subjected to CT scan of brain and patients with evidence of acute ischemic infarct were included in this study.

Inclusion criteria:

1. Patients aged over 15 years who were admitted to Government General Hospital with acute ischemic stroke.

2. Neuro imaging showing ischemic infarct.

exclusion criteria:

1. Patients with hemorrhagic stroke.

2. Imaging showing venous infarct.

study period:

February 2007 to January 2009 (24 months).

Study Design:

Observational and Prospective study.

ETHICAL COMMITTEE APPROVAL: Obtained

Number of Patients included:

490 patients were included in this study after satisfying the inclusion and exclusion criteria.

Methodology:

After getting an informed and written consent from the patients or their relatives, they were included in this study. Patients' details regarding the age, sex, and risk factors like hypertension, diabetes mellitus, past history of TIA, hypercholesterolemia, valvular heart disease, atrial fibrillation, smoking and alcoholism were recorded.

History of the stroke including the time of onset of the stroke, its relationship with activity and the mode of the onset were collected.

Each patient was examined thoroughly. The side of the Hemiparesis, associated facial weakness, presence of aphasia or dysarthria, sensory deficit, visual deficit, lower cranial nerve involvement, cerebellar involvement and the severity of the stroke based on the NIHSS were noted.

All patients were investigated with CT brain plain. MRI Brain was done if necessary. Most of the patients underwent serum lipid profile and cardiac evaluation with ECG & ECHO.

These were utilized to analyze the clinical profile, risk factors, infarct pattern and regional distribution.

Then a proportion of patients who did not have any medical or neurological contraindications for heparin were given a course of heparin.

The treatment outcomes of all patients were assessed on the 10^{th} day of onset of stroke based on modified Ranikin Score. This score assesses functional independence and impact on activities in daily living and grades patients from 0 (no symptoms) to 6 (death). Mortality rate was also assessed in all patients on or before tenth day of stroke onset.

Observation and results

A total of 490 patients aged above 15 years who were admitted to Government General Hospital, Chennai between February 2007 to January 2009 with clinical features and imaging suggestive of acute ischemic stroke were included in this study.

AGE DISTRIBUTION:

The maximum number of patients were in the age group between 51 and 60 years followed by the age group between 41 and 50, 61 and 70 years. Table 1 shows the age distribution in this study.

TABLE - 1: AGE DISTRIBUTION

Age group in years	*No. of Patients*	*% of Total Patients (490)*
15 – 40	46	9.38
41 – 50	120	24.48
51 – 60	181	36.94
61 – 70	119	24.29
> 70	24	4.9
Total	***490***	***100***

SEX Distribution:

There were 274 males (55.9%) and 216 females (44.1%) among the 490 patients in this study.

TABLE 2: SEX DISTRIBUTION IN THIS STUDY

Sex	*No. of Patients*	*% of Total Patients (490)*
Males	274	55.9
Females	216	44.1
Total	***490***	***100***

Age and Sex distribution:

Males predominated in the age group between 51 and 60 years followed by 41 to 50 years. Females predominated in the age group between 51 and 60 years followed by 61 to 70 years. Around two thirds of males (64.7%) were in the age group between 41 and 60 years and two-third of females (65.7%) were in the age group between 51 and 70 years. The Table 3 shows age distribution based on sex.

TABLE 3: AGE DISTRIBUTION BASED ON SEX

Age Group in Years	*Males (274)*	*Females (216)*
15 – 40	28 (10.2%)	18 (8.3%)
41 – 50	78 (28.5%)	42 (19.4%)
51 – 60	99 (36.2%)	82 (38.0%)
61 – 70	55 (20.1%)	64 (29.7%)
> 70	14 (5.0%)	10 (4.6%)
Total	***274 (100%)***	***216 (100%)***

Risk factors

HYPERTENSION:

Among 490 patients, 322 patients had hypertension (65.71%)

TABLE -4: PREVALENCE OF HYPERTENSION IN THIS STUDY

Duration in years	*No. of patients*	*% of total Patients (490)*
Detected now	48	9.79
0 - 1 year	54	11.03
1 - 5 years	98	20.00
> 5 years	122	24.89
Total	***322***	***65.71***

DIABETES:

Out of 490 patients, 216 patients were diabetic (44.08%)

TABLE -5: PREVALENCE OF DIABETES MELLITUS IN THIS STUDY

Duration	*No. of patients*	*% of Total Patients (490)*
Detected now	24	4.89
0 - 1 years	34	6.94
1 - 5 years	72	14.69
> 5 years	86	17.56
Total	***216***	***44.08***

SMOKING:

217 patients were smokers (44.28%), (212 males and 5 females).

TABLE -6: PROPORTION OF SMOKING IN THIS STUDY

Duration	*No. of patients*	*% of Total Patients (490)*
0 - 5 years	18	3.67
5 - 10 years	43	8.78
> 10 years	156	31.83
Total	***217***	***44.28***

ALCOHOLISM:

198 patients were alcoholic (40.4%) (186 were males, 12 were females).

TABLE -7: PREVALENCE OF ALCOHOLISM IN THIS STUDY

Duration	*No. of patients*	*% of Total Patients (490)*
0 - 5 years	20	4.08
5 - 10 years	46	9.39
> 10 years	132	26.93
Total	***198***	***40.40***

Clinical features:

The clinical features of all the patients were studied in detail. The commonest presentation was the weakness of the extremities with or without speech, language, sensory and other cranial nerve disturbances.

hemiparesis:

It was the commonest clinical presentation and seen in 422 patients among the 490 patients (86.12%) in this study. Among the 422 patients, Right hemiparesis was found in 188 patients (44.54%) and Left hemiparesis was seen in 234 patients (55.46%).

TABLE - 8: DISTRIBUTION OF HEMIPARESIS

Side of hemiparesis	*No. of Patients*	*% of total Patients (490)*
Right	188	38.36
Left	234	47.76
Total	***422***	***86.12***

In addition, facio brachial monoparesis was seen in 14 patients, of which 8 were right sided and 6 were left sided.

UMN Facial parEsis:

It was seen in 402 patients among the 422 patients who had Hemiparesis. Right UMN facial paresis was found in 179 cases (44.5%) and Left UMN facial paresis was documented in 223 cases (55.5%).

TABLE - 9: DISTRIBUTION OF UMN FACIAL PARESIS

Side of UMN facial paresis	*No. of Patients*	*% of total Patients (490)*
Right	179	36.53
Left	223	45.51
Total	***402***	***82.04***

95.2% of patients with Hemiparesis were found to have UMN facial paresis. None of the patients had isolated UMN facial paresis. 20 patients had isolated Hemiparesis.

Severity of weakness in UPPER AND LOWER LIMBS:

The table 10 shows the severity of weakness between UL & LL.

TABLE - 10: SEVERITY OF UL & LL WEAKNESS

	No. of Patients	*% of total Patients (490)*
UL = LL	334	68.16
UL weaker than LL	53	10.82
LL Weaker than UL	35	7.14
Total	***422***	***86.12***

Hemparesis in relation to arterial territory:

The table 11 shows the proportion of Hemiparesis in relation to arterial territory. Among the 422 patients with Hemiparesis, 386 cases were due to anterior circulation infarct (91.47%) and 36 patients were due to posterior circulation infarct (8.53%).

TABLE - 11: HEMIPARESIS IN RELATION TO ARTERIAL TERRITORY

	No. of Patients	*% of total Patients (490)*
Anterior Circulation	386	78.77
Posterior Circulation	36	7.35
Total	***422***	***86.12***

aphasia:

Aphasia was seen in 146 patients among the 490 patients (29.8%). Of these 146 patients, 133 patients (91.1%) had right hemiparesis and 7 patients (4.8%) had left hemiparesis and 6 patients had aphasia as the only clinical manifestation. Broca's aphasia was the commonest type of aphasia followed by global aphasia.

TABLE -12: DISTRIBUTION OF APHASIA

Type of Aphasia	*No. of Patients*	*% of total Patients (490)*
Global	38 (26.02%)	7.78
Broca's	44 (30.14%)	8.98
Wernicke's	29 (19.86%)	5.92
Transcortical motor	24 (16.44%)	4.89
Transcortical sensory	11 (7.54%)	2.25
Total	***146***	***29.8***

dysarthria:

Among the 490 patients in this study, 195 patients had dysarthria.

TABLE -13: DISTRIBUTION OF DYSARTHRIA

	No. of Patients	*% of total Patients (490)*
Anterior Circulation a. Left Hemiparesis	151	30.81
b. Right Hemiparesis	09	1.82
Post Circulation	35	7.14
Total	***195***	***39.77***

Sensory involvement:

It was seen in 234 patients out of the 490 patients (47.75%). Unilateral hemi sensory loss was present in 228 patients and crossed hemisensory loss in 6 patients. The table 14 shows the distribution of sensory deficit in this study.

TABLE -14: DISTRIBUTION OF SENSORY DEFICIT

Side of Hemi sensory loss	*No. of Patients*	*% of total Patients (490)*
Right side	103	21.02
Left side	125	25.51
Crossed	6	1.22
Total	***234***	***47.75***

Other clinical features:

TABLE -15: DISTRIBUTION OF OTHER CLINICAL FEATURES

S.No	*Clinical features*	*No. of Patients*	*% of total Patients (490)*
1.	LMN facial paresis	10	2.04
2.	Diplopia	12	2.44
3.	Dysphagia and Regurgitation	73	14.89
4.	Homonymous hemianopia	18	3.67
5.	Ataxia	39	7.96
6.	Wallenburg's syndrome	11	2.24
7.	Seizures	31	6.32

8.	TIA in the past	96	19.59

Time of onset of stroke:

TABLE -16: TIME OF ONSET OF STROKE

Time of onset	*No. of patients*	*% of total Patients (490)*
Noted on awakening	277	56.53
During activities	105	21.43
Rest	108	22.04
Total	***490***	***100***

In more than 50% of patients, stroke was noticed immediately on awakening. 22% of patients had stroke at rest, followed by 21% of patients having stroke during activities.

MODE of onset of stroke:

In this study, it was assessed only in those who had stroke while they were awake. Of the 213 patients, stroke onset was sudden and acute in 163 patients (76.53%), stepwise and gradual progression in 41 patients (19.25%) followed by fluctuating course in 9 patients (4.22%). It could not be defined clearly in the remaining 277 patients who noticed stroke immediately on awakening.

TABLE -17: MODE OF ONSET OF STROKE

Time of onset	*No. of patients*	*% of total Patients*
Sudden & Acute	163	76.53
Gradual progression	41	19.25
Fluctuating	9	4.22
Total	***213***	***100***

Time interval from STROKE ONSET to hospitalization:

This table shows the time interval from the onset of stroke to hospitalization.

TABLE -18: TIME INTERVAL FROM STROKE ONSET TO HOSPITALIZATION

Time interval (Hours)	*No. of patients*	*% of total Patients (490)*
0 - 3	22	4.49
3 – 6	62	12.65
6 – 12	83	16.94
12 - 24	266	54.29
> 24	57	11.63
Total	***490***	***100***

Only 4.49% of patients reached the hospital within three hours of onset of stroke.

Time interval from STROKE ONSET to CT brain: (Table 19)

Time interval (Hours)	*No. of patients*	*% of total Patients (490)*
0 - 3	12	2.45
3 - 6	47	9.59
6 – 12	97	19.79
12 - 24	186	37.97
> 24	148	30.20
Total	***490***	***100***

Only 2.45% of stroke patients underwent CT scan brain within three hours of onset of stroke. More two-third of stroke patients underwent CT brain only after 12 hours of stroke onset.

Severity of stroke based on NIHSS on admission:

National Institute of Health Stroke Scale was applied in all patients to assess the severity of stroke at the time of admission.

TABLE -20: SEVERITY OF STROKE BASED ON NIHSS

NIHSS	*No*	*% of total Patients (490)*
Mild Stroke (≤ 6)	137	27.96
Moderate (7-10)	178	36.33
Moderately severe (11-15)	133	27.14
Severe (16-22)	25	5.10
Very severe (≥ 23)	17	3.47

Total	***490***	***100***

Among the total 490 patients, 64.29 % had mild to moderate stroke, followed by 27.14% of moderately severe stroke. Severe and very severe stroke were seen in 8.57% of patients.

Neuro imaging:

CT brain plain (non enhanced CT scan) was done in all patients.

PROPORTION of arterial territory involvement:

Internal carotid territory was involved in 410 patients, Vertebrobasilar territory was involved in 74 patients and both were affected in 6 patients.

TABLE -21: PROPORTION OF ARTERIAL TERRITORY INVOLVEMENT

Arterial Territory	*No. of patients*	*% of total Patients (490)*
Internal Carotid territory	410	83.67
Vertebrobasilar territory	74	15.10
Both	6	1.23
Total	***490***	***100***

Distribution of INDIVIDUAL artery involvement:

This table shows the distribution of involvement of individual artery in this study group.

TABLE -22: DISTRIBUTION OF INDIVIDUAL ARTERY INVOLVEMENT

Arterial territory	*No. of patients*	*% of total Patients (490)*
MCA	380	77.55
ACA	24	4.90
MCA & ACA	6	1.22
PCA	42	8.57
PICA	17	3.48

Basilar artery	9	1.84
SCA	4	0.81
AICA	2	0.41
Multiple	6	1.22
Total	***490***	***100***

Pattern of MCA territory infarcts:

MCA territory was divided into three regions - Cortex, Sub cortical white matter (Corona radiata & Centrum semiovale) and Gangliocapsular region. This table shows the distribution of MCA territory involvement based on these regions.

TABLE -23: PATTERN OF MCA INFARCTS BASED ON REGIONS

Pattern of MCA territory	**No. of patients**	***% of total MCA Infarcts***
Cortex	143	37.63
Sub cortical white matter	110	28.95
Gangliocapsular region	127	33.42
Total	***380***	***100***

Out of the 127 patients with gangliocapsular infarcts, 96 patients had internal capsule infarcts and 31 patients had basal ganglia infarcts.

TABLE -24: PATTERN OF MCA INFARCTS BASED ON BRANCHES

MCA	***No. Of patients***	***% of total MCA Cortical infarcts (143)***
Stem Occlusion	30	20.98
Superior Division	71	49.65
Inferior Division	42	29.37
Total	***143***	***100***

LACUNAR INFARCTS:

The following table shows the proportion of lacunar infarcts in this study. Among the total 490 patients, 105 patients showed lacunar infarcts (21.43% of total).

TABLE -25: LACUNAR INFARCTS

Region involved	***No. Of patients***	***% of total lacunar infarcts***

		(105)
Gangliocapsular region	74	70.48
Sub cortical white matter	23	21.90
Thalamus	6	5.71
Pons	2	1.91
Total	***1o5***	***100***

Gangliocapsular region was commonly involved in 74 patients, followed by sub cortical white matter in 23 patients. Among the gangliocapsular infarcts, 65 patients had internal capsule infarcts and 9 patients had basal ganglia infarcts.

Border zone infarctS:

In this study, 43 patients had border zone infarcts. Of which, 28 cases were external border zone infarcts and 15 cases were internal border zone infarcts. Among the external border zone infarcts, 9 cases involved between MCA & ACA and the remaining 19 cases involved between MCA & PCA.

TABLE -26: PATTERN OF BORDER ZONE INFARCTS

Border zone infarcts	***No***	***% of total Patients (490)***
External border zone	28	5.71
Internal border zone	15	3.06
Total	***43***	***8.77***

Regional distribution of infarcts:

This table shows the regional distribution of infarcts in this study.

TABLE -27: REGIONAL DISTRIBUTION OF INFARCTS

Regions	***No. of patients***	***% of total Patients (490)***
Cortex (Frontal, Parietal and lateral temporal)	143	29.18
Occipital cortex	30	6.12
Medial temporal cortex	8	1.63
Sub cortical white matter	110	22.45
Gangliocapsular	127	25.92
Thalamus	25	5.10

Midbrain	14	2.86
Pons	10	2.04
Medulla	11	2.24
Cerebellum	26	5.31

In the anterior circulation, cortex was most commonly involved followed by gangliocapsular region and sub cortical white matter. Both cortex and sub cortical white matter were involved in 48 patients out of the total 253 patients. Both sub cortical white matter and deep gray matter (basal ganglia) were affected in 37 patients out of the total 227 patients.

In the Vertebrobasilar territory, medial occipital cortex was the commonest region affected followed by cerebellum and thalamus with equal frequency.

Serum lipid profile:

It was done in 426 patients (86.94 % of the total).

LDL CHOLESTEROL:

TABLE -28: RANGE OF LDL CHOLESTEROL VALUES IN THIS STUDY

LDL range (mg/dl)	*No. of Patients*	*% of total Patients (426)*
< 100	22	5.16
100 – 129	38	8.92
130 – 159	101	23.71
160 – 189	187	43.90
> 190	78	18.31
Total	***426***	***100***

TRIGLYCERIDES:

TABLE -29: RANGE OF TRIGLYCERIDES VALUES IN THIS STUDY

TGL range	*No. of Patients*	*% of total Patients (426)*
< 150	62	14.56
150 – 199	149	34.97

200 – 399	215	50.47
> 400	Nil	
Total	***426***	***100***

HDL CHOLESTEROL:

TABLE -30: RANGE OF HDL CHOLESTEROL VALUES IN THIS STUDY

HDL range (mg/dl)	*No. of Patients*	*% of total Patients (426)*
> 40	172	40.38
< 40	254	59.62
Total	***426***	***100***

Echo cardiography:

It was done in 315 patients (64.29 % of the total). **(Table: 31)**

Echo	*No*	*% of total Patients (315)*
LVH with Normal EF	140	44.44
Normal study	72	22.86
LV systolic dysfunction	48	15.25
LV Global dysfunction	5	1.59
Rheumatic Heart disease	19	6.03
MVPS with MR	3	0.95
MVPS without MR	16	5.08
LA clot	4	1.27
Aortic valve sclerosis	3	0.95
Mitral annular calcification	2	0.63
Prosthetic valve	3	0.95
Total	***315***	***100***

Treatment outcome on 10th day:

151 patients were randomly selected for heparin treatment who otherwise were not having any neurological and medical contra indications to heparin. Injection heparin 5000 IU intravenously or subcutaneously twice daily was given for one week to 10 days. Remaining 339 patients were treated with antiplatelets. In the heparin subgroup, 12 patients worsened after heparin because of hemorrhagic transformation, leading to withdrawl of heparin.

Modified ranikin score (mRS) on 10th day:

mRS was applied in all patients on 10th day or at the time of discharge or death, whichever was earlier to assess the treatment outcome.

TABLE -32: MODIFIED RANIKIN SCORE ON 10TH DAY (entire group)

mRS grading	*No. Of Patients*	*% of total Patients (490)*
0	6	1.22
1	92	18.78
2	142	28.98
3	167	34.08
4	24	4.90
5	25	5.10
6	34	6.94
Total	***490***	***100***

mRS of hepaRIn subgroup patients and no heparin SUBgroup:

Modified Ranikin score was also applied to assess the difference in the treatment outcome between the patients in heparin subgroup and those in no heparin subgroup. Test of Proportion (Z test) was applied to look for any statistical significance between these two groups.

TABLE -33: mRS of HEPARIN AND NO HEPARIN SUBGROUPS:

mRS grading	Heparin *subgroup*	*No Heparin sub group*	*p value*
0	4 (2.64%)	2 (o.59%)	>0.05
1	35 (23.18%)	57 (16.81%)	>0.05
2	44 (29.14%)	98 (28.91%)	>0.05
3	48 (31.79%)	119 (35.11%)	>0.05

4	7 (4.64%)	17 (5.01%)	>0.05
5	8 (5.30%)	17 (5.01%)	>0.05
6	5 (3.31%)	29 (8.55%)	**<0.05**
Total	***151 (100)***	***339 (100)***	

In our study, heparin subgroup showed statistically significant benefits in preventing the deaths (mRS - 6), compared to those who were not given heparin.

mRS OF HEPAIN SUBGROUP PATIENTS BASED ON THE TIME OF STARTING THE HEPARIN: (Table 34)

Patients in the heparin subgroup were subdivided in to five groups based on the time of starting the heparin from the stroke onset. They were those who received heparin with in 6 hours of stroke onset, between 6 and 12 hours, 12 – 24 hours, 24 – 48 hours and more than 48 hours from stroke onset.

mRS grading	***Heparin started within 6 hours of stroke onset (Total:10)***	***6 – 12 hours of stroke onset (Total:30)***	***12 – 24 hours of stroke onset (Total:67)***	***24 – 48 hours of stroke onset (Total: 33)***	***>48 hours of stroke onset (Total: 11)***	***p value***
0	2	1	1	0	0	**<o.o5**
1	1	9	13	8	4	>o.o5
2	3	5	23	10	3	>o.o5
3	2	9	24	11	2	>o.o5
4	1	2	2	1	1	>o.o5
5	0	3	2	2	1	>o.o5
6	1	1	2	1	0	>o.o5

Treatment outcome based on Modified Ranikin Score was assessed in all the five groups. Test of Proportion was applied to look for any statistical significance between these subgroups. For statistical purposes, the above five groups are clubbed in to two groups, those who received heparin with in 12 hours and after 12 hours. In this study, those who received heparin with in twelve hours showed significant benefits in mRS Zero group.

Mortality on 10th day: (TABLE 35)

34 patients were dead on or before 10th day (6.93%).

Cause of Death	*No .of Patients*	*% of total Patients (34)*
Infection	12	35.29
Cerebral oedema	8	23.53
Myocardial infarction	7	20.59
Respiratory failure	4	11.77
Metabolic encephalopathy	3	8.82
Total	***34***	***100***

One-third of patients died because of infection. Other common causes were cerebral oedema and myocardial infarction.

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discussion

SEX DISTRIBUTION:

In Our study, sex distribution showed general male preponderance in stroke. Males constituted 55.9% of the total and 44.1% were females.

According to the Barcelona stroke registry of 3577 patients collected over 17 years from 1977 onwards, 57.3% were males and 42.7% were females. [39]

Bogousslavsky et al in 1988 found that 61.5% of stroke patients were males and 38.5% were females (Lausanne stroke registry of 1000 Patients). [12]

Both the studies showed male predominance in ischemic stroke. Our study showed almost similar results to Barcelona stroke registry.

AGE DISTRIBUTION:

Age distribution in our study showed that maximum number of patients was in the age group

between 51 and 60 years (36.94%) followed by the age group 41 and 50 years (24.48%) and the age group between 61 and 70 years (24.29%). The mean age was 54.6 ± 11.4 years for the entire group in this study.

According to Lausanne stroke registry in 1988, maximum number of patients was in the age group between 60 and 80 years. The same stroke registry documented that mean age was 60.7 ± 13.9 years for the entire group. [12]

Marti-Vilalta et al in 1998 reported [39] in a series of stroke patients that mean age was 66.4 ± 13 years for the entire group. 55% of the total patients were in the age group between 65 and 84 years followed by 33% in the age group between 45 and 64 years.

The mean age in our study was one decade earlier compared to the above two western studies. This could be due to the fact that Indian population develop stroke one to two decades earlier than that of the Western population, because of early atherosclerosis and increasing number of young hypertension and diabetes patients.

AGE DISTRIBUTION BASED ON SEX:

Age distribution based on the sex in our study showed that among the males, 64.7% of cases were in the age group between 41 and 60 years. But 67.7% females were in the age group between 51 and 70 years. The mean age in our study was 52.4 ± 12.2 for men, 56.8 ± 10.6 for women.

This was concordant with Bogousslavsky et al's Lausanne stroke registry [12] which confirmed the general male predominance in stroke with female preponderance in extreme age groups (<30 and ≥ 70-80 years).

The mean age was 64.80 ± 11.97 for men and 68.54 ± 13.92 for women in the Barcelona Stroke Registry [39].

Women were more affected due to stroke beyond 50 to 60 years, one decade later than that of men. It may be related to protective benefits of reproductive age group before 50 years of age, longer life expectancy in females and earlier death in men with vascular risk factors.

RISK fACTORS:

HYPERTENSION:

Arterial hypertension is defined as systolic BP greater than 140 mm. Hg. or diastolic BP greater than 90 mm Hg. It is one of the most important and effective modifiable risk factors. But unfortunately it remains untreated or under treated (Hajjar and Kotchen, 2003) [40]. It predisposes to ischemic stroke by aggravating atherosclerosis and increases the relative risk for stroke three to four fold. Blood pressure treatment, resulting in modest reduction in SBP of 10

to 12 mm Hg. and 5 to 6 mm Hg. Diastolic BP is associated with a 38% reduction in stroke incidence. (MacMohan and Rodgers, 1996) [41].

In our study, hypertension was present in 65.71% of patients and around 37% of these patients had hypertension of more than five years duration.

Marti-Vilalta et al in 1998 reported that 53% of patients with ischemic stroke had hypertension [39]. According to Lausanne stroke registry, hypertension was seen in 45.5% of patients [12]. The increased incidence of hypertension in our study could be due to general increase in HT population in Southeast Asia.

According to Trivandrum Stroke Registry, hypertension was seen in 80% of stroke patients, which was slightly higher than our study [42]

According to Emre kumral and Gamze in 2002, HT was reported in 62% of patients with ischemic stroke, which was almost similar to our study. [43]

DIABETES MELLITUS:

DM is associated with stoke, independently of the various cardiovascular risk factors which usually accompany this disease. Indeed, the relative risk of stroke of all ages was 1.8 for diabetic men and 3.0 for diabetic women (Shinton and Beever, 1989, Burchfield et al 1994) [18] [19].

In our study, diabetes mellitus was found in 44.08% of patients. 39% of these patients had diabetes of more than five years duration. 11% of these patients were diagnosed to have DM during this present admission, after having ruled out the possibility of stress induced hyperglycemia with the help of HbA1C and follow up blood glucose testing after one week to ten days.

According to Guy Van Melle et al, diabetes mellitus was seen in 12.6% of patients. [12]

Barcelona stroke registry documented 21% of patients with diabetes among the ischemic strokes. [39]

Emre Kumral et al reported 26% of diabetes among ischemic stroke patient. [43]

The increase in diabetic population in our study reflected the general increase in the incidence of diabetes in the Southeast Asia.

Smoking:

It raises the blood fibrinogen, enhances platelet aggregation and increases the hematocrit level and blood viscosity. Smoking cessation substantially decreases the risk for subsequent stroke. The incidence of stroke is 50% higher in smokers.

Our study showed that 217 patients out of total 490 patients (44.6%) were found to be smokers currently. 71.8% of these patients were smokers of more than 10 years duration. Among these 217 patients, 212 were males and 5 were females.

According to Bogousslavsky et al, cigarette smoking was found in 45.6% of patients in his stroke series [12]. Hamburg stoke data bank reported 44% of smoking among ischemic stroke patients [44]. Our study showed similar results to these studies.

36.2% of ischemic stroke patients were smokers in Barcelona stroke registry [3]. Emre Kumral reported that smoking was present in 28% of patients. [43]

AlcoholiSm:

Chronic heavy alcohol consumption and binge drinking may exert their harmful effects through changes in Blood Pressure, platelet aggregability, blood coagulation and the level of triglycerides.

In this study, 198 patients were alcoholic (40.4%) among the total 490 patients. Of these 198 patients, 186 cases were males and 12 cases were females. Around 70% of these patients were alcoholic for more than 10 years duration.

CLINCAL FEATURES:

HEMIPARESIS:

In our study, motor deficit in the form of Hemiparesis was the commonest presentation and seen in 422 patients, constituting 86.12% of the total. Left Hemiparesis was more common (55.5%) that Right Hemiparesis (44.5%).

Our study showed similar results to Lausanne stroke registry [12], in which 83.2% of patients had a motor deficit. But right hemiparesis was more common (56%) than left hemiparesis in that study (44%). But our study showed the reverse results that left hemiparesis was more common than the right hemiparesis for unknown reasons.

According to Barcelona stroke series published in 1998, 78.1% of ischemic stroke patients had motor deficit in the form of Hemiparesis, which was slightly lesser in number than our study. [39]

Caplan and Mohr et al reported 58% of right Hemiparesis and 42% of Left Hemiparesis. [45]

In our study, 91.5% of Hemiparesis were due to anterior circulation stroke and 8.5% were due to posterior circulation stroke, which in addition to hemiparesis had other manifestations.

In our study, Upper and lower limbs were equally involved in 68.2% of patients, Upper limb was more involved in 10.82% and Lower limb was more involved in 7.14%. All the 30 anterior

cerebral artery infarcts showed more LL involvement and 5 patients with MCA infarcts also manifested with predominant LL involvement. Facio brachial monoparesis was seen in 14 patients (2.5%) (right side 8 patients, left side 6 patients).

According to Bogousslavsky et al, Upper and Lower limbs were equally involved in 44.4% of patients, Upper limb was more involved in 13.4 % of patients and Lower limb was more involved in 2% of patients. Facio brachial monoparesis was seen in 32% of patients. [12]

Herman et al 1982 reported that hemiparesis with uniform weakness of upper and lower limbs was seen in two-third of patients with motor deficit, almost similar to our study. [29]

Facio brachial monoparesis was seen in 20-29% (Rascol et al 1982, Melo et al 1992) [30] [31] which was several times more than in our study.

UMN facial paresis ipsilateral to the side of Hemiparesis was seen in 402 patients out of 422 patients. Remaining 20 patients had only Hemiparesis, who were found to have sub cortical infarcts.

APHASIA & DYSARTHRIA:

In our study, aphasia was found in 146 patients (29.8%) out of 490 patients. Broca's aphasia was the most common type seen in 8.98% followed by Global aphasia in 7.78% and Wernicke's aphasia in 5.92%. In General, motor aphasia was seen in 13.87%, Sensory aphasia in 8.17% and Global aphasia in 7.78% of patients. Among these 146 patients, 7 patients had left Hemiparesis. They were left handed and language area was probably in the right hemisphere, which was not proven in this study.

In this study, Dysarthria was documented in 39.77% of patients of which 30.8% had left Hemiparesis and 9.14% had posterior circulation (Vertebrobasilar artery) stroke. 1.82 % of right Hemiparesis patients also had dysarthria.

According to Julien Bogousslavsky et al, aphasia was seen in 33.6 % of patients. The commonest type was motor aphasia in 12.7%, followed by global aphasia in 11.2% and sensory aphasia in 9.7%. Dysarthria was documented in 12.4% of patients. Our study showed almost similar results with lesser number in global aphasia patients and increased number of dysarthria patients [12].

Arboix et al in his stroke series reported that aphasia or dysarthia was seen in 50% of patients. Our study showed that 69.5% of patients had aphasia or dysarthria. [39]

Laske et al 1981 documented that approximately 25% of stroke patients had significant aphasia. [46]

Sensory dEFICIT:

In our study, sensory disturbances were found in 234 patients (47.75%) of patients. Left side was involved in 25.5%, right side in 21.0% and crossed sensory deficit was seen in 1.22%.

According to Lausanne stroke registry, 46.4% had sensory deficit [12]. Marti Vilalta et al reported 47% of sensory disturbances. Our study showed almost similar results. [39]

Hemianopia:

Homonymous hemianopia was seen in 3.67% of patients in our study. According to Barcelona stroke registry, it was seen in 23% of patients [39]. Julien Bogousslavsky reported homonymous hemianopia in 13.6% of patients [12]. Low proportion of homonymous hemianopia in our study could be due to more anterior circulation infarcts in our study.

DYSPHAGIA:

It is one of the significant complications of several stroke syndromes. Gordon et al 1987 [47] found that 45% of stroke patients had dysphagia on admission. Wade et al 1987 [48] reported dysphagia in 43% of stroke patients. Baner et al 1989 [49] described in 30% of stroke patients. Smitherd et al 1997 [50] found that 51% of patients had dysphagia on admission.

In our study, dysphagia was seen in 14.89% of the patients. It was significantly lower than that in the above series. This could be due to more anterior circulation strokes in our study.

TIME OF ONSET OF STROKE:

In our study, 56.53% of the total patients were noted to have stroke on awakening, 21.43% had stroke during activities and 22.04% had the deficit during rest. Circadian changes in catecholamine levels, blood pressure, platelet aggregability, blood coagulability and fibrinolytic activity could explain the increased strokes noted on awakening.

MODE OF ONSET OF STROKE:

In this study, it was assessed only in those who had stroke while they were awake. Of the 213 patients, stroke onset was sudden and acute in 163 patients (76.53%), stepwise and gradual progression in 41 patients (19.25%) followed by fluctuating course in 9 patients (4.22%). It could not be defined clearly in the remaining 277 patients who noticed stroke immediately on awakening.

In Lausanne stroke registry, neurologic deficit was complete immediately or within few minutes after stroke onset in 62.7% of cases, progressed smoothly in 30.7% and fluctuated in 6.6%. [12]

Arboix et al in 1998 reported that stroke onset was sudden in 46.6% of cases, while it was gradual or stepwise over hours in 37.1% and fluctuating in 11.7%. [39]

According to Mohr and Louis Caplan in Harvard Co-operative stroke registry, deficit was sudden at onset in 53%, stepwise or gradual in 38% and fluctuating in 9% of patients. [45]

TIME INTERVAL FROM STROKE ONSET TO HOSPITALIZATON:

In this study, Only 22 patients (4.4% of total) had reached the hospital with in the golden period of stroke (i.e. 3 hrs), which necessitated the importance of public awareness about stroke. Around 65% of patients reached the hospital only after 12 hours of stroke onset.

In Kay et al series, among the 773 patients with stroke admitted in one year, 63% arrived at hospital within 12 hours, 76% within 24 hours, and 85% within 48 hours of ictus [51].

In a study of 457 patients in North Carolina, USA, more than half of all patients with stroke did not present within 24 hours of onset, and one third did not present within 48 hours [52]. In the United States Stroke Data Bank, conversely, about half of the patients were admitted by 12 hours and two thirds by 24 hours after onset. [53]

TIME INTERVAL FROM STROKE ONSET TO FIRST CT SCAN BRAIN:

In this study, CT scan was done only in 2.45% of total patients with in three hours of the stroke onset. This constituted only 50% of those patients who reached hospital within 3 hrs. This necessitated the importance of stroke awareness among medical personnel. More than two thirds of the patients underwent CT scan after twelve hours of the onset.

SEVERITY OF STROKE BASED ON NIHSS:

In this study, moderate stroke was most common and seen in 36.3% followed by mild stroke in 28.0% and moderately severe in 27.1%. Severe and very severe stroke constituted 8.6%. In General, two-thirds of patients (64.2%) had mild to moderate stroke. This might be due to selection bias and improvements in risk factors management.

NEUROIMAGING:

Distribution of ARTERIAL TERRITORY INVOLVEMENT:

In our study, Carotid territory (Anterior circulation) was affected in 83.67% of patients (410/490), Vertebrobasilar territory (Posterior Circulation) was involved in 15.1% of patients (74/490) and both were affected in 1.2% of patients (6/490).

Barcelona Stroke Registry in 1998 reported that anterior circulation was affected in 70% of patients, posterior circulation was affected in 12% of patients, 4.5% of infarcts were multiple and infarct location could not be determined in the remaining 13.5%. [39]

According to Bogousslavsky et al, carotid territory was involved in 68%, vertebro basilar

territory in 26% and multiple territories in 3% of patients. [12]

In Harvard Stroke series, anterior circulation infarcts were seen in 63.5% and posterior circulation infarcts were seen in 26.5% of patients. [45]

Our study showed more involvement of anterior circulation which could be due to intracranial stenosis of anterior circulation being more common in Southeast Asian population.

Distribution of INDIVDUAL ARTERY INVOLVEMENT

In this study, MCA was affected in 77.55% of total patients, followed by PCA in 8.57% of total, ACA in 4.90% and PICA in 3.48% of patients. Basilar artery was involved in 1.84%, SCA in 0.81% and AICA in 0.41% of the total patients. Two patients had bilateral ACA infarcts and six patients had both MCA and ACA infarcts. Two patients had both MCA and PCA infarcts.

According to Barcelona stroke registry, MCA was the most common vascular territory affected by infarction in 66.5% followed by PCA in 6.6% and ACA in 2.8% of patients. [39]

Louis Caplan and Mohr et al documented that MCA was involved in 75% followed by PCA in 11%, ACA in 3% and basilar artery in 5% of cases [45]. Our study shows almost similar results to this series.

In our study, 92% of carotid territory infarcts involved MCA territory and 7% involved ACA territory. Among the posterior circulation strokes, 58% of infarcts involved PCA followed by PICA in 23.6% of patients.

In Bogousslasky et al series of stroke patients, 96% of Internal carotid territory infarcts involved MCA followed by ACA in 3% of patients. 48% of posterior circulation infarcts involved basilar and PICA where as PCA was affected in 36% of patients. [12]

PATTERN OF MCA TERRITORY INFARCTS:

In our study, among the MCA territory infarcts, 37.6% of patients involved cortex followed by the gangliocapsular area in 33.4% and sub cortical white matter in 29%. Of the gangliocapsular infarcts, 25.26% had internal capsule infarct and 8.16% had basal ganglia infarct.

In the Lausanne stroke registry, cortex involvement was seen in 51% of patients and involvement of deeper structures including sub cortical white matter and gangliocapsular areas was seen in 32% of patients. [12]

According to Marti-Vilalta et al, cortex was commonly involved in 52% of patients and deeper structures including sub cortical white matter and gangliocapsular areas was seen in 25.5% of patients. [39]

But our study showed increased proportion of involvement of deeper structures including both sub cortical white matter and basal ganglia to the extent of 62.4%. This was nearly twice that of in the western studies.

This could be due to increased incidence of hypertension and intracranial arterial stenosis commonly occurring in Asian population.

MCA SUbcortical INFARCTS:

It comprises central white matter of the cerebral hemispheres, supplied by deep medullary branches from superficial MCA branches and to some extent by the lenticulostriate arteries.

According to Emre Kumral et al in 2002, only 4.9% of patients with MCA territory infarcts involved corona radiate. This constituted 1.4% of total ischemic stroke patients. [43]

Read and Pettigrew reported in 1998 that the incidence of sub cortical infarcts was seen in 1.2% of total ischemic strokes. [54]

Bogousslavsky 1992 published that it accounted for only 1.2% to 2% of all strokes. [55]

But according to recent reports, the incidence of centrum semiovale infarcts is more than 7% in one series (Boiten et al 1997) [56] and 22% in another series (Leys et al 1994) [57].

But in our study, sub cortical infarcts constituted 29% of MCA infarcts and 22.45 % of total infarcts. This was several times more frequent compared to above studies. This exemplifies the fact that strokes in Indian subcontinent are different from Western hemisphere in the aetiology and pathophysiology.

MCA cortical INFARCTS:

MCA superior division was most commonly involved among the MCA cortical infarcts and seen in 49.65% followed by inferior division in 29.37% and stem occlusion in 20.98% in our study.

According to Lausanne stroke registry, MCA stem occlusion was seen in 13%, superior division was involved in 31% and inferior division was affected in 20%. [12]

Mohr and Louis Caplan et al reported that MCA superior division was occluded in 45.59% followed by stem occlusion in 41.17% and inferior division in 13.24%. [45]

LACUNAR infarcts:

In our study, 21.43% of total 490 patients (105/490) were found to have lacunar infarcts in Neuroimaging. Of these 105 patients, gangliocapsular area was affected in 74 patients (15.1% of total), followed by sub cortical white matter in 23 patients (4.7%). Thalamus was involved in

six patients and pons in two patients. Among the gangliocapsular lacunar infarcts, internal capsule was affected in 65 patients and basal ganglia in 9 patients.

According to Barcelona Stroke Registry, lacunar infarcts were seen in 10% of patients [39], where as it was seen in 13% of patients in Gross et al series [58], 19% in Harvard Stroke Registry [45] and in 21% in Bamford et al series [59]. Our study showed similar result to Bamford series.

Border zone infarcts

In our study, Border zone infarcts were seen in 43 patients out of the total 490 patients, constituting 8.77%. Among the 380 MCA infarcts, border zone infarcts were responsible for 11.31%. Of these, 15 cases involved internal border zone and the remaining 28 cases involved external border zone. Among the external border zone infarcts, 9 cases involved between MCA & ACA and the remaining 19 cases involved between MCA & PCA.

Border zone infarctions account for 5 to 10% of all ischemic infarcts (Bernd Ringelstein) [27] and 6% in Bladen et al series [28], almost similar to our study results.

According to Bogousslavsky, only 3% of patients had border zone infarcts [12]. According to Jorgensen et al in 1969, border zone infarcts were seen in 10% of all brain infarcts in an autopsy series. [60]

Regional distrubution

In this study, 29.18% of total infarcts involved frontal, parietal and lateral temporal cortex in various combinations. It was followed by gangliocapsular region involvement in 25.92% (Internal capsule: 19.6%, Basal ganglia: 6.32%) and sub cortical white matter in 22.45%, which involved frontal, parietal and temporal white matter.

Hence 51.63 % of the total infarcts involved frontal, parietal and temporal lobes in this study.

Among Vertebrobasilar artery infarcts, medical occipital cortex was most commonly involved in 30 patients (6.12% of total 490 patients), followed by cerebellum in 26 patients (5.3% of total) and thalamus in 25 patients (5.1% of total). Midbrain (2.8%), medulla (2.24%) and pons (2.04%) were the other regions involved.

According to Barcelona Stroke Registry, the main locations, either isolated or in combination, of cerebral infarcts were parietal (33.6%), temporal (29%), frontal (29%), internal capsule (18.5%), occipital cortex (9%), basal ganglia (7%), thalamus (5.5%), pons(9%), medulla (4.5%) and the midbrain(3.5%). [39]

Bogousslavsky et al in 1988 reported that pons was involved in 7% of total infarcts, medulla in 3.5% followed by midbrain in 1.9% and cerebellum in 1.9%. [12]

Hyper lipidemia

62.21% patients in our study had LDL value of more than 160 mg/dl, 50.47% of patients had TGL level of more than 200 mg/dl and 59.62% of patients had low HDL volume of less than 40 mg/dl.

Tribolet et al reported in his series that 70% of patients had hyperlipidemia. [61], similar to our study.

Hypercholesterolemia was found in 14.5% of patients with ischemic stroke patients in Lausanne stroke registry. [12]

The more number of patients with hyperlipidemia in our study could be due to increase in the prevalence of metabolic syndrome and the changing life style among the Indian population.

ECHOCARDIOGRAPHY:

Echocardiography showed LVH with normal EF in more than 40% of patients. LV systolic dysfunction was seen in 15.2% of patients. MVPS and rheumatic heart disease were found in 6% of patients each. ECHO was normal in 22.8% of patients. LA clot was seen in 1.27% of patients.

According to Lausanne stroke registry, LVH was found in 26.3%, left ventricular dysfunction was seen in 17.08%, MVPS was demonstrated in 20.2% of patients and ECHO was normal in 41%. [12]

Tribolet et al reported that out of the 853 patients admitted with ischemic stroke, dilated cardiomyopathy was seen in 19.1%, previous anterior wall myocardial infarction in 6.2%, left ventricular systolic dysfunction in 3.7%, mitral valve stenosis with enlarged left atria in 1.6%, intracardiac masses in 0.5% and valvular prosthesis in 0.2%. [61]

Treatment outcome BASED ON mRS:

Modified Ranikin score was applied to assess the treatment outcome on 10^{th} day or at the time of discharge which ever was earlier.

Among the 490 patients in our study, 1.22% of patients had no symptoms (mRS - Zero) at all on 10th day of stroke, 18.78% of patients were able to carry out their usual daily activities and 28.98% of patients had mild disability in doing their daily chores. A significant proportion of patients (34.08%) had moderate disability and 10% had moderately severe and severe disability. 6.94% of patients were dead on or before 10^{th} day.

According to Marti Vilalta et al, the functional limitation at the time of discharge was absent in 28% of patients, followed by mild disability in 22%, moderate disability in 12% and the severe disability was seen in 8%. The mean duration of hospital stay in this study was 23 days [39].

Compared to this study, our study showed similar number in mild and severe disability, whereas the percentage of moderate disability was more in our study. This could be due to assessing the treatment outcome on 10^{th} day in our study, instead of the time of discharge which was around 23 days in that study.

According to Mohr et al, up to 60% of patients require some assistance in daily activity two weeks after ischemic stroke. [34]

Treatment outcome BETWEEN HEPARIN & NO HEPARIN GROUPS:

Several randomized studies of unfractionated heparin or LMWH for the treatment of acute ischemic stroke continue to show no proven benefits in the reduction of stroke related mortality and morbidity, early stroke recurrence or stroke prognosis. The time window from stroke onset varied from 6 to 48 hours in these studies.

In our study, 151 patients received heparin out of the total 490 patients (30.82%) and the remaining 339 patients received antiplatelets (69.18%). Among the heparin group, 12 patients (7.95 %) worsened because of hemorrhagic transformation.

Modified Ranikin Score was applied to assess any difference in the outcome between the two groups. Heparin subgroup showed statistically significant benefits ($p < 0.05$) in preventing the deaths (mRS - 6), compared to those who were not given heparin. But there was no statistically significant benefit in improving the disability on tenth day between the two groups. Overall heparin might have helped in reducing the mortality in this study without influencing the disability.

mRS OF HEPAIN SUBGROUP PATIENTS BASED ON THE TIME OF STARTING THE HEPARIN:

Among the heparin subgroup, treatment outcome was assessed between those who received heparin at various intervals from stroke onset. In this study, those who received heparin with in twelve hours showed significant benefits in mRS Zero group. These patients improved to no disability group on tenth day. Apart from this, there was no significant difference in other disability mRS groups or death rate between those who were treated with heparin before 12 hours and after 12 hours of stroke onset.

A recent Italian study of acute stroke patients suggested that heparin may be of benefit, if given within 3 hours of symptom onset for non lacunar stroke. (Camerlingo M, 2005) [62]

But there were several limitations in our study as far as heparin treatment was concerned. Our study was not a randomized controlled trial so that the results of this study can't be extrapolated to general population. Those patients who had moderate to large size infarcts were automatically excluded from the heparin group, who otherwise would have had a poor prognosis. Our study assessed the treatment outcome on tenth day. But most of the studies in the literature assessed the disability status at 3 and 6 months.

MORTALITY RATE:

In our study, 34 patients were dead on or before tenth day and the mortality rate was 6.93%. The commonest cause of death was infection (Pneumonia, Urinary tract infections and Septicemia), which was responsible for 35% of total deaths followed by cerebral oedema in 23.5% and myocardial infarction in 20.5%. Respiratory failure was responsible in 11.77% and metabolic encephalopathy in 8.82% of deaths. Cerebral oedema as a cause of death was proved by the follow up CT scans which showed transtentorial herniation.

According to the Rochester Epidemiologic Project, the risk for death after first ischemic stroke was 7% at 7 days, 14% at 30 days, 27% at 1 year, and 53% at 5 years [63], almost similar to our study.

According to Barcelona stroke registry, 30 day mortality rate for ischemic stroke was 12% and the most frequent complication was infection seen in 14.5% of patients [39]. 30 day mortality rate was 14% in Pilot Stroke data Bank [64], 10.3% in Hamburg Stoke data Bank [44] and Besancon Stroke registry reported 13.6% stroke mortality rate.[65].

Trivandrum Stroke Registry reported that the 28th day case fatality rate in both ischemic and hemorrhagic strokes was 24.5% [42]. Julien Bogousslavsky et al reported in his stroke series that the overall mortality rare was 5.9% for ischemic strokes of which 86.4% of total deaths were directly or indirectly due to stroke. [12]

The reduced mortality rate in our study could be due to two reasons. A) Patients were followed up only for 10 days. If they were followed up for 30 days, then mortality rates may be comparable to the above studies. B) More number of patients in this study had mild to moderate stroke based on NIHSS on the day of admission.

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SUMMARY:

Salient Features in this study were:

- The maximum number of patients was in the age group between 51 and 60 years. (36.94%). Our population developed ischemic stroke one to two decades earlier than the western population.

- Males (55.9%) were more affected than Females (44.1%). Males predominated in the age group between 41 and 60 years (64.7%), while females predominated in the age group between 51 and 70 years (67.7%).

- Common risk factors were hypertension (65.71%), hyperlipidemia (62.21%), diabetes mellitus (44.08%), smoking (44.28%) and alcoholism (40.04%). Modification of lifestyle and proper management of these modifiable risk factors might play a major role in the primary and secondary prevention of ischemic stroke.

- Left hemiparesis (55.46%) was more common, Aphasia was seen in 29.8% and Dysarthria in 39.7% of patients.

- More number of patients (56.53%) had noted the stroke on awakening.

- Only 4.4% of patients reached the hospital with in three hours. CT scan Brain was done only in 2.45% of patients with in three hours. This exemplifies the need of Stroke awareness among the public so that the patients can reach the hospital with in the therapeutic window.

- 64.29% of patients had mild to moderate stroke.

- Anterior circulation (83.67%) was significantly more affected than the Posterior circulation (15.10%)

- MCA was involved in 77.55% of total infarcts, followed by PCA in 8.57% and ACA in 4.9% of total infarcts.

- Among the MCA infarcts, cortex was commonly involved (37.63%), followed by gangliocapsular region (33.42%) and sub cortical white matter (28.95%).

- Among the Vertebrobasilar territory, medial occipital cortex was commonly involved (6.12% of total infarcts), followed by cerebellum (5.31%) and thalamus (5.1%).

- Border zone infarcts were noted in 8.77%.

- 48.98% of patients had no to slight disability (MRS 0 - 2) on tenth day of stroke onset.

- 7.94% of patients developed hemorrhagic transformation following heparin treatment.

- Mortality rate was 6.93% on tenth day of stroke onset. Commonest cause was infection (35.29%), followed by cerebral oedema (23.53%).

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CLINICAL PROFILE, NEUROIMAGING EVALUATION and TREATMENT OUTCOME OF ACUTE ISCHEMIC STROKE PATIENTS.

-- MIN, GGH, CHENNAI.

Name: DOA: DOD: Unit: Ward:
Age/Sex: I.P / O.P No: MIN No:
Address: **Diagnosis:**

Condition at Discharge: Alive/Dead

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HISTORY:

Inability / weakness of Rt / Lt, UL / LL -- Date and Time of stroke, Nature of onset, progression if any and duration of stroke prior to hospitalization

Deviation of angle of mouth to Rt / Lt

Speech Disturbances / Aphasia

Visual Disturbances / Dysphagia , Regurgitation / Ataxia

Activity when symptoms noticed first: On Awakening / Rest / Activity

Time interval from onset of illness to hospitalization: GGH: Others:

Past History:
Hypertension –
Diabetes –
TIA ---

Personal History:

Alcoholic – Content: Amount: Frequency: Duration:

Smoking – Beedi / Cigarette ; Number/Day: Duration:

EXAMINATION:

HR: PR: BP: / , RR: Peripheral Pulses:
Consciousness: Speech: Aphasia:
Facial palsy – Rt / Lt UMN / LMN
Other cranial Nerves:

Motor System: Rt Lt
Tone UL
LL
Power UL Pyramidal Distribution
LL
DTR UL
LL
Abdominal
Plantar
Sensory System: Involuntary movts: Cerebellum:
Gait: Carotids: Bladder/Bowel:
CVS: RS: ABDOMEN:

NIHSS:

INVESTIGATION:

1. CT Scan (Plain): Regions Involved—

Artery & branch –

CT Scan (Contrast):

Time Interval Between Ictus and CT Scan –

Time Interval Between Hospitalization and CT Scan –

Repeat scan(If any) Findings –

2. MRI Brain, MRA and MRV:

3. Blood: TC- DC- P L E M ,Hb- g%, ESR- ,Platelet-

Sugar(R)- ,(F)- (PP)- ,Urea, Creatinine-

Na+ , K+ ,Cl- ,HCO3-

4.Serum Lipid profile (Fasting):

Cholesterol- , LDL- ,HDL- , Triglycerides-

4. Chest X-ray-

5. ECG-

6. ECHO-

TREATMENT:

Heparin: Time interval from onset of illness-

LMWH / Unfractionated, Duration of treatment:

Antiplatelets –

Antiedema measures-

OUTCOME on 10th Day:

Modified Rankin Scale:

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National Institute of Health Stroke Scale (NIHSS):

_____

1a) Level of conciousness :_____

0 Alert, 1 Drowsy, 2 stuporous, 3 comatose.

1b) Level of consciousness Questions :Month, Age

0 Both correct, 1 one correct, 2 Incorrect.

1c) Level of consciousness Commands: open and close eyes, make fist

0 obeys both correctly, 1 obeys one correctly, 2 Incorrect

2) Best gaze :_____

0 Normal, 1 partial gaze palsy, 2 Forced deviation

3) Best Visual:_____
(introduce visual stimulus/threat to patient's visual quadrants)

0 No loss, 1 partial hemianopia, 2 complete hemianopia
3 Bilateral hemianopia

4) Facial palsy : _____
(show teeth, raise eyebrows and squeeze eyes shut)

0 Normal, 1 minor asymmetry, 2 partial(lower face paralysis), 3 complete

5) Motor Arm-Left :_____
(Elevate extremity 90deg and score drift/movement)

0 drift, 1 drift, 2 Can't resist against gravity, 3 No effort against Gravity, 4 no movement,

6) Motor Arm-right :_____

(Elevate extremity 90deg and score drift/movement)

0 drift, 1 drift, 2 Can't resist against gravity, 3 No effort against Gravity, 4 no movement,

7) Motor leg-left :_____
(Elevate extremity 90deg and score drift/movement)

0 drift, 1 drift, 2 Can't resist against gravity, 3 No effort against Gravity, 4 no movement,

8) Motor leg-right :_____
(Elevate extremity 90deg and score drift /movement)

0 drift, 1 drift, 2 Can't resist against gravity, 3 No effort against Gravity, 4 no movement,

9) Limb ataxia :_____

0 Absent, 1 present in upper or lower, 2 present in both

10) Sensory :_____
(pin prick to face,arm trunk and leg compare side to side)

0 Normal, 1 partial loss, 2 Dense loss

11) Best language :_____
(Name items,describe a picture and read sentences)

0 No aphasia, 1 Mild-moderate aphasia, 2 severe aphasia, 3 Mute

12) Dysarthria:_____
(Evaluate speech clarity by patient repeating listed words)

0 Normal, 1 Mild-moderate slurring, 2 severe, nearly intelligible

13) Extinction and Inattention :_____

0 No neglect, 1 partial neglect, 2 profound neglect

MODIFIED RANKIN SCALE (mRS):

0 = No symptoms at all

1 = No significant disability despite symptoms; able to carry out all usual duties and activities

2 = Slight disability; unable to carry out previous activities, but able to look after own affairs without assistance

3 = Moderate disability; requiring some help, but able to walk without assistance

4 = Moderately severe disability; unable to walk without assistance and unable to attend to own bodily needs without assistance

5 = Severe disability; bedridden, incontinent and requiring constant nursing care and attention

6 = Dead