A Dissertation on

EVALUATION OF THE RISK FACTORS AND CLINICAL FEATURES OF STROKE IN CORRELATION WITH CT SCAN FINDINGS

Submitted to

THE TAMILNADU DR. M.G.R. MEDICAL UNIVERISTY

CHENNAI – 600 032

In partial fulfillment of the regulations

For the Award of the Degree of

M.D. (GENERAL MEDICINE)

BRANCH -1, PART - II

KILPAUK MEDICAL COLLEGE

CHENNAI – 600 010

MARCH - 2007

CERTIFICATE

This is to certify that **Dr. R. KUMARESAN,** Post graduate Student (April 2004 to March 2007) in the Department of Internal Medicine, Kilpauk Medical College, Chennai – 600 010, has done this dissertation on **"EVALUATION OF THE RISK FACTORS AND CLINICAL FEATURES OF STROKE IN CORRELATION WITH CT SCAN FINDINGS"** under my guidance and supervision in partial fulfillment of the regulation laid down by **THE TAMILNADU DR M.G.R MEDICAL UNIVERSITY** for the award of MD Degree in General Medicine.

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ACKNOWLEDGMENT

I greatfully acknowledge and sincerely thank **Prof. THIAGAVALLI KIRUBAKARAN, M.D.,** Dean, Govt. Kilpauk Medical College and **Prof. D.S. SOMASEKAR, M.D.,** Superintendent Govt. Royapettah Hospital Chennai, for permitting me to utilize the facilities available in the institution for my study.

I owe my sincere gratitude to **Prof. S.R. SAKUNTHALA, M.D.,** Professor and Head, Department of Internal Medicine, Kilpauk Medical College, for her constant encouragement and guidance in all stages of this study.

I whole heartedly express my sincere thanks to my respected chief **Prof. G. RAJENDRAN, M.D.,** Reader in Medicine, Chief IInd Medical Unit, Govt. Royapettah Hospital, Kilpauk Medical College, for his valuable guidance and support throughout my dissertation work.

I wish to thank **Dr. T. RAVINDRAN, M.D., Dr. A. NASREEN BEGUM, M.D.,** and **Dr. T. RAMESHKUMAR, M.D.,** Assistant Professors, Department of Internal Medicine, Kilpauk Medical College, for their valuable suggestions and help rendered throughout this work.

I also extend my thanks to **Dr. AMUDHAN,** Assistant Professor, Department of Radiology, Govt. Royapettah Hospital, for his kind and timely support.

I also thank my colleagues, house surgeons, staffs of our hospital for their contribution to the study.

Last but not least, with sincere gratitude, I thank all the patients who contributed so much to this study, without whom the study could not have been possible.

CONTENTS

Introduction

INTRODUCTION

Cerebrovascular accident or stroke is one among the three leading causes of death, surpassed only by ischemic heart disease and malignancy. Stroke is also a common cause of physical disability, which imposes a substantial burden to the community in the foreseeable future. It is estimated that the incidence of stroke is likely to increase by about 20% in the next 20 years. In a developing nation like India, Rheumatic heart disease forms a significant risk factor for stroke. The high incidence and serious consequences make it one of the most important challenges faced by medical profession today.

Atleast 50 percent of the neurological disorders in a general hospital are due to stroke. As remarked by a renowned neurologist C.M. Fisher, neurology is learnt “Stroke by Stroke”. The advent of imaging procedures such as Computerized Tomography, Magnetic Resonance Imaging, Carotid Doppler and Magnetic Resonance Angiography have led to better evaluation of stroke and its risk factors.

A risk factor is a characteristic for an individual or for a population, indicating that the individual or population has an increased risk of stroke compared with one without that characteristic. Such an association does not imply causality which is determined more by strength of the association, the consistency of the association in different studies and population and the presence of a dose response relationship.

The last decade has witnessed exciting advances in the field of stroke, both in terms of enhanced understanding and in the availability of a rich panoply of therapeutic options, most important being thrombolytic therapy. Several studies in the recent years, have proved beyond doubt, the role of thrombolytic therapy in acute cerebral infarction. The success of this modality of treatment rests in early recognition of stroke and institution of therapy, for which a good knowledge of risk factors and clinical presentation of stroke and investigative modalities available is crucial.

Inspite of revolutionary changes in the management of ischemic stroke, the ultimate goal should be its prevention in order to decrease the incidence, for which, a thorough understanding of the etiopathogenesis and risk factors seems essential.

Hence, the present study on stroke was undertaken.

Aim of the Study

AIM OF THE STUDY

1. To study the age and gender distribution.
2. To evaluate the risk factors in cerebrovascular disease.
3. To study the mode of presentation of different types of stroke.
4. To study the pathogenesis of stroke with the aid of CT Scan.
5. Correlative study of the above modalities.

Review of Literature

REVIEW OF LITERATURE

STROKE (Synonyms: Cerebrovascular Accident, Apoplexy)

DEFINITIONS

Stroke

WHO defined stroke as “rapidly developed clinical signs of focal or global disturbance of brain function; lasting more than 24 hrs or leading to death, with no apparent cause other than vascular origin”.

Completed Stroke

It is the term applied to the temporal profile of the stroke syndrome in which the deficit is prolonged and often permanent, causing demonstrable parenchymatous changes. Most completed strokes reach the maximum of neurological dysfunction within an hour of onset.

Stroke in Evolution

This describes the temporal profile in which the neurological deficit occurs in a stepwise or progressive fashion culminating in a major deficit in the absence of treatment. In the carotid arterial system, this progression may go upto 24 hrs. If the vertebrobasilar system is the site of ischemia, the deficit may progress for upto 72 hours.

Transient Ischemic Attack (TIA)

It is an acute loss of focal cerebral or monoocular function with symptoms lasting less than 24 hours, which after adequate investigation is presumed to be due to thrombotic or embolic vascular disease.

Reversible Ischemic Neurological Deficit (RIND)

It is the term applied to the temporal profile of the stroke syndrome in which the focal neurological ischemic deficit lasts longer than 24 hours but resolves within 3 weeks.

Stuttering Hemiplegia

It is characterized by repeated episodes of TIA or crescendo TIAs followed by fully evolved stroke. Internal carotid artery lesions produce this event.

Lacunar Stroke

It is secondary to lipohyalinosis or microatheroma of the small perforating branches within the brain substance producing small deep infarcts less that 1.5 cm in diameter.

Pathological types of stroke

1. Cerebral infarction – Cerebral thrombosis / embolism
2. Primary Intracerebral haemorrhage
3. Subarachnoid haemorrhage.

EPIDEMIOLOGY OF STROKE

Stroke epidemiology has lagged behind coronary artery disease, since strokes are not only less frequent, but occur later in life.[7] Stroke epidemiology is hampered by :

- Diagnosis still being largely a matter of clinical skill, without help of many confirmatory investigations.
- Being a disorder of late middle age and elderly, where other diseases frequently co-exist.
- A low post-mortem rate.
- Inaccuracy of death certificates.
- Being more pathologically diverse.

Incidence can only be reliably assessed in prospective community based studies using obsessional methodology to identify all possible patients. Hospital based studies are subject to referral bias since very mild and rapidly fatal strokes are least likely to be brought to the hospital. In any case, the incidence of stroke rises rapidly with age, about a quarter occur below the age of 65, and about a half below the age of 75.

Prevalence is difficult to measure because a large representative sample has to be identified as the denominator, a large proportion of that sample must be questioned and many past stroke episodes are forgotten by patients[8].

Prevalence depends on long-term survival which may be changing. In any event, stroke prevalence dose not even capture the burden of stroke since some patients die early after the onset and many of the survivors are not disabled at all.

The mortality of stroke is on the decline in the recent years. However, it is unlikely that treatment of hypertension is responsible since the decline had started earlier and treatment cannot explain more than 25% of the decline[9]. Treatment of TIAs is also unlikely, largely because only a small proportion of strokes are preceded by TIAs[10]. Based on retrospective analysis of subjects admitted in urban hospitals in India, strokes constitute 2% of all hospital cases and 20% of neurological admissions. Incidence in younger population (below 40 years) is 13-22%.

Seasonal and Diurnal Variation

Stroke incidence and mortality is higher in winter months[11], probably secondary to high blood pressure and complications like pneumonia being more fatal in winter[12].

Cerebral infarction occurs more frequently in an hour or two after waking in the morning[13]. Sub-arachnoid haemorrhage is unlikely to occur during sleep, whereas intracerebral haemorrhage is more likely to occur during strenuous activity.

RISK FACTORS

Stroke places a tremendous burden on health resources throughout the world. Vast majority of stroke patients have well recognized risk factors, and reducing their prevalence should make a considerable impact on stroke incidence. Nonmodifiable risk factors include age, sex, race, genetic factors and family history.

Age

Age is the strongest risk factor for stroke. Risk of stroke in people aged 75-84 yrs is 25 times the risk in people age 45-54 yrs[14].

Gender

There is a small male excess of strokes most prominent in middle age and disappearing in the very elderly and probably absent in the young[12]. However the difference is much less than that observed in myocardial infarction and peripheral vascular disease. It is more common in blacks than whites.

Genetic factors

Some genetically determined diseases are vascular anomalies like malformations, aneurysms, connective tissue disorders such as Ehler – Danlos Syndrome, Marfan's syndrome and hereditary causes of hypercholesterolemia. Several genetic determinants contribute to stroke risk. Of these, carotid intimal-medial wall thickness (IMT) is particularly relevant, because it is a surrogate measure of subclincial atherosclerosis and a strong predictor of future ischemic stroke. Studies of twins, siblings, and families have provided significant evidence for heritability, but the genes involved have not been identified. Some researchers have reported that IMT is high in people with functional variants of genes related to matrix deposition (*MMP3*), inflammation (interleukin 6), and lipid metabolism (hepatic lipase, *APOE, CETP*, and *PONI*)[15].

Family history

Sometimes stroke is clearly familial in some cases it is a simple mendelian pattern of inheritance (eg. Hemophilia). Family history of stroke is an independent risk factor for ischemic stroke with onset before age 70 years[16].

Modifiable / Preventable Risk Factors

Systemic Hypertension

Systemic hypertension is strongly associated with stroke risk, probably by increasing the extent and severity of atheroma, and the prevalence of microvascular disease in the small penetrating arteries. Stroke risk doubles with each 7.5 mmHg increase in diastolic blood pressure. A reduction of blood pressure 10-12 mm systolic, 5-10 mmHg diastolic is associated with 38% decrease in stroke[17]. Isolated systolic hypertension (ISH) and isolated diastolic hypertension (IDH) both are independent predictors of stroke. Patients with systolic and diastolic hypetension both are at the highest risk of stroke and should be treated more aggressively[18].

The strength of association of blood pressure and stroke is strong consistent, biologically plausible and treatment reduces stroke risk. Hence one can conclude that hypertension is a causal risk factor[19,20]. Hypertension and type 2 diabetes increase stroke risk independently, and their combination increases the risk drastically. A significant proportion of the risk of stroke assumed to be related to hypertension may be attributable to concomitant diabetes[21].

Diabetes Mellitus

Diabetes doubles the risk of stroke compared to non-diabetics.[20] In addition patients with diabetic retinopathy and autonomic neuropathy are at increased risk of ischemic stroke.[22] Raised Hb A1c is an independent risk factor

for stroke in people with and without diabetes[23]. Impaired glucose tolerance is an independent risk factor for future stroke in nondiabetic patients with TIA or minor ischemic stroke[24].

Hyperlipidemia

Increased levels of plasma total cholesterol, LDL cholesterol and decreasing levels of HDL cholesterol are strong risk factors for coronary artery disease[25]. Although their relationship to stroke is less clearcut compared to ischemic heart disease, there is almost certainly some association. This may be due to lipid levels being less associated with vascular events in the elderly (where more strokes occur) than in younger people (where coronary events are more common[26]). Cholesterol levels are also negatively associated with haemorrhage[27].

Smoking

Smoking is a strong risk factor for subarachnoid haemorrhage[28] (relative risk 3.0), and for cerebral infarction (relative risk 2.0) and there appears to be no association with parenchymal haemorrhage. Males and females are equally affected, but the association seems to be weaker in the elderly[29]. Stroke risk decreases 5 yrs after cessation of smoking. There is a positive association between history of smoking among spouses with the incidence of stroke[30].

Alcohol

Heavy consumption is an independent risk factor, while moderate consumption may be protective[31]. However, apoE genotype may modify this association, and even moderate alcohol intake may be associated with an increased risk of ischemic stroke among apoE4-positive older adults[32]. Drinking pattern and beverage type may also be important. Intake of more than 2 drinks per day may be associated with a higher risk for ischemic stroke[33]. Alcohol consumption also raises the blood pressure[34], affects blood lipids[35], increases the incidence of atrial fibrillation[36] and cardiomyopathy which predispose to stroke.

Heart Disease

Coronary artery disease is clearly associated with stroke. The risk for stroke is markedly increased after MI, particularly early after MI, compared with the expected risk in population without MI. Stroke is associated with a large increase in the risk for death after MI[37]. Cardiac failure, ECG changes and rapid heart rate further increase the risk. Left ventricular dysfunction even of mild degree, is independently associated with an increased risk of ischemic stroke[38]. The most frequent potential cardiac cause of embolism is atrial fibrillation, usually non-rheumatic in developed countries, rheumatic in developing nations[39].

Transient Ischemic Attacks

TIAs are ischemic strokes, recovering in 24 hrs. A TIA patient has an increased risk of stroke about 5–10 times greater than that of a non TIA patient of the same age. The risk of stroke after initial stroke / TIA is higher than the risk of cardiac events. The propensity after stroke / TIA to have the first recurrent ischemic event in the brain, rather than in the heart, has implications for prophylactic therapy selection[40].

Carotid Stenosis

Cervical carotid bruit is a risk factor for stroke, but not necessarily in the same arterial territory, because stenosis in one artery is likely to be associated with disease of other arteries[41]. Less than 75% occlusion has 1.3% annual incidence of stroke, while more than 75% occlusion has a risk of 10.5% per year. Ulcerated, echoluscent, heterogenous plaques with soft core are at higher risk. Aortic arch atheroma is an important independent risk factor for stroke, studies indicate a four times greater odds of stroke in patients with severe arch atheroma[42].

Haematocrit

Cerebral blood flow is strongly related to haematocrit. Increased haemotocrit in association with smoking, hypertension and plasma fibrinogen increase the risk of stroke.

Hormones

Although strokes are uncommon in females of the reproductive age group, attributing this to endogenous sex hormones is difficult[43], since high does estrogen given to elderly men with prostate cancer, increases risk of vascular event[44]. The use of oral contraceptive pills triples the risk of stroke in women especially smokers aged more than 30 years.

Hormone replacement therapy seems to have a protective effect[45]. It has been stated that natural menopause has no effect on stroke while surgical menopause without estrogen replacement doubles the risk.

Obesity

The relationship between obesity and stroke is seldom studied, and if present is probably secondary to its association with hypertension and diabetes. The risk of stroke is increased in men with metabolic syndrome, in the absence of past history of stroke, diabetes and cardiovascular disease at baseline[46].

Diet

High salt intake[47], decreased intake of fruits, vegetables[48], deficiency of selenium and vitamin E are associated with increased risk, while high potassium diet by reducing blood pressure reduces risk of stroke[49].

Other Risk Factors

1. High serum uric acid levels are strong risk factor for stroke[50].

2. Severe obstructive sleep apnea syndrome increases the risk of ischemic stroke in elderly population and the increase is independent of other risk factors, including hypertension[51, 52].

3. Physical inactivity is associated with an increased risk of stroke. A high level of leisure time physical activity reduces the risk of all subtypes of stroke[53].

4. Chagas Disease is a risk factor for stroke, independent of systolic dysfunction or presence of cardiac arrhythmias[54].

5. Fabry Disease is also one of the cause of unexplained stroke in young patients, especially in those with the combination of infarction in the vertebrobasilar artery system and proteinuria[55].

6. Moyamoya Disease is a risk factor for stroke[56].

7. Higher serum ferritin concentrations in postmenopausal women are associated with an increased risk of ischemic stroke[57].

8. A high Lp(a) concentration is associated with a higher incidence of ischemic stroke in blacks and white women, but not in white men[58].

9. Peripheral arterial disease is a strong marker of multifocal atherosclerotic disease. Individuals with intermittent claudication and asymptomatic peripheral vascular disease (as defined by an abnormal low ankle-brachial systolic pressure index) are at excess risk of stroke[59].

10. Raised plasma factor VII coagulant activity, raised tissue plasminogen activator antigen, low blood fibrinolytic activity and raised von Willebrand factor are risk factors for coronary artery disease and may also be risk factors for stroke[60].

11. Hyperhomocyteinaemia is a risk factor for stroke[61].

12. Oral contraceptives increase the risk of ischemic stroke, and less so, haemorrhagic strokes[62].

13. Arterial dissection

14. Antiphospholipid syndrome

15. Protein C, protein S deficiencies

16. Other less documented risk factors include.
    a. Sickle cell disease
    b. Drug abuse
    c. Low socioeconomic factors
    d. Stress
    e. Alpha (1) antichymotrypsin polymorphism

f. Boiled and unfiltered coffee consumption

g. Pregnancy and puerperium

h. Infection – Chlamydia pneumoniae, periodontal disease, meningeo vascular syphilis, Helicobacter pylori.

i. Cholesterol embolisation syndrome

j. Trauma

k. Fibromuscular dysplasia

l. Irradiation

PATHOPHYSIOLOGIC CLASSFICATION OF STROKE

I. ISCHEMIC – 85%

Thrombotic		**Embolic**	
Lacunar	20-25%	Cardioembolic	20%
Large Vessel	1-5%	Artery – artery	15%
		Cryptogenic	30%
		Others	10%

II. HAEMORRHAGIC – 15%

Intraparenchymal	-	10%
Subarachnoid	-	1-2%
Subdural	-	<1%
Epidural	-	<1%

ISCHEMIC STROKE

Ischemic stroke is caused by a sudden occlusion of an artery supplying the brain or less often by low flow distal to an already occluded artery. The causes of ischemic stroke include:

a. Thrombosis

Atherosclerosis

Vasculitis

Collagen vascular disease: Temporal (Giant cell) arteritis, polyarteritis nodosa, Wegener’s granulomatosis, Takayasu’s arteritis, syphilis.

Meningitis: Tuberculous, fungi, syphilis, bacteria, herpes zoster

Arterial dissection: Carotid, vertebral, intracranial arteries.

Haematological disorders: Polycythemia, thrombocytosis, thrombotic thrombocytopenic purpura, DIC, dysproteinemias, haemoglobinopathies (sickle cell disease).

Miscellaneous: Cocaine, amphetamine, moyamoya disease, fibromuscular dysplasia, Binswanger's disease.

b. Embolism

Cardiac Source

Dysrhythmia – Atrial fibrillation, sick sinus syndrome

Coronary artery disease

Rheumatic heart disease

Cardiomyopathy

Prosthetic valve

Congenital heart disease - MVP, patent foramen ovale

Infective endocarditis

Atrial myxoma

Non-bacterial thrombotic endocarditis.

Atherothrombotic arterial disease

Bifurcation of common carotid artery, distal vertebral artery and aortic arch.

Unknown source

May be associated with a hypercoagulable state

c. Vasoconstriction

Following subarachnoid haemorrhage, migraine, eclampsia etc.

d. Venous occlusion

Dehydration, post-partum, systemic cancer etc.

Brain is an obligate aerobe and obtains energy from oxidative metabolism of glucose. Since the brain glucose stores are negligible, a decrease in cerebral blood flow causes ischemia. The normal cerebral blood flow is 50 ml/100 g/min. When this falls below a critical value of 20 ml/100 g/min, there is loss of neuronal electronic function which is a reversible stage. When it decreases to less than 10 ml/100 g/min, then aerobic mitochondrial metabolism fails and anaerobic metabolism leads to lactic acidosis[63]. As a sequel, sodium and water enters the cell, potassium leaks out, due to failure of energy dependent intracellular homeostasis, leading to irreversible cell death.

In man, it is not known exactly how long and how severe focal ischemia has to be before complete recovery of function is impossible. The ischemic and infarcted brain cannot autoregulate. Therefore modest increase in cerebral perfusion pressure could cause hyperemia, increased cerebral blood flow, edema and haemorrhagic infarction which in turn increases intracranial tension and decreases cerebral perfusion pressure. On the other hand, a decrease in cerebral perfusion pressure may exacerbate ischemia.

Ischemic penumbra is an area around an infarcted tissue, that is in the reversible state of electrical failure, where flow is decreased, function depressed, oxygen extraction fraction is high, but recovery is still possible. Thrombolytic agents are used in this window time to salvage the ischemic penumbra zone.

Ischemic cerebral edema is partly cytotoxic and partly vasogenic. Cytotoxic edema starts within minutes and affects grey matter. Vasogenic edema starts several hours later and affects more white matter, since the damaged blood brain barrier allows plasma to enter the extracellular space.

Differentiating signs of Thrombosis and Embolism

Thrombosis	Embolism
Preceding brief shot gun like TIA	Single or infrequent but longer – lasting TIA or strokes
TIAs all in same vascular territory	Infarcts in multiple vascular territories

Onset of stroke after sleep	Onset during activity or sudden strain, cough or sneeze.
Absence of distal embolus by angiography	Presence of distal intra arterial embolus by angiography or transcranial doppler
Infarct on CT or MRI near border zone of affected artery	Infarct on CT or MRI in heart of vascular territory wedge shaped and abutting on cortical surface
Presence of risk factors for atherosclerosis	Presence of known cardiac, arterial or venous source of embolus
Occlusion or severe stenosis of a large artery shown by USG or angiography	Haemorrhagic cerebral infarct on CT

HAEMORRHAGIC STROKE

Intracranial haemorrhage accounts for approximately 15% strokes. The overall mortality for this subtype of stroke is from 25% to 60%. In nearly 70% of patients hypertension is the commonest cause. Other causes include arteriovenous malformation, aneurysm, coagulopathy, drugs, amyloid angiopathy, metastatic tumours, cavernous angiomas, dural arterial-venous fistula, capillary telangiectasia –etc.

Location of hypertensive haemorrhage :

Putamen	-	60 – 65%
Thalamus	-	15 - 25%
Pons	-	5 - 10%
Cerebellum	-	1 – 5%
Subcortical white matter	-	1 – 2%

Clinical presentation of intracranial haemorrhage are mainly due to symptoms of raised intracranial tension and specific for the location of haematoma. Characteristically haemorrhage presents with progression of deficits over a period of hours. Seizures at the time of presentation occurs usually in cortical haemorrhage.

CT continues to be the gold standard for diagnosis of acute intracranial haemorrhage and evaluating its prognosis. MRI appears normal in the first 24 hrs, but is more specific than CT in determining the age of haemorrhage. Acute

haemorrhage appears as a hyperdense area having attenuation values of 50-100 Hounsfeld units. They become hypodense in the ensuring couple of weeks. The periphery of the clot is absorbed at a rate of 0.7 mm/day.

SUBARACHNOID HAEMORRHAGE [SAH]

The incidence of SAH increases with age being more common in women. Causes include rupture of a saccular aneurysm, bleeding from vascular anomaly, extension into subarachnoid space from primary intracerebral haemorrhage. Idiopathic SAHs are localized to perimesencephalic cisterns and are benign[64]. Chronic, recurrent bleed produces superficial hemosiderosis of central nervous system[65].

SAH usually presents as excruciating headache followed by loss of consciousness. Focal neurologic deficits such as hemiparesis, aphasia, abulia may occur. Delayed neurological deficits may be due to re-rupture, hydrocephalus, vasospasm and hyponatremia.

Hall mark of SAH is blood in CSF. High quality, non-contrast CT localizes blood in 95% of cases within 72 hrs. Lumbar puncture is indicated only if CT scan is not available. Four-vessel conventional X-ray angiography is performed to localize, define anatomic details for interventional therapy.

INTRACRANIAL VENOUS THROMBOSIS

Thrombosis in dural sinuses /cerebral veins is much less common than arterial thrombosis. However, it should be suspected in high risk groups mainly postpartum and malignancies. Venous thrombosis can result from diseases that alter clotting factor or cellular constituents of blood.

CAUSES

Local	**Systemic**
Head injury	Dehydration
Intracranial surgery	Septicaemia
Sepsis	Oral contraceptives
Subdural empyema	Hypercoagulable states
Meningitis	Drugs
Tumor invasion of sinuses	Non- metastatic effect of intracranial malignancy
Catheterization of jugular vein	

Symptoms depend on structure involved, extent and rapidity of thrombosis and collaterals. Headache, nausea, vomiting, convulsions, depressed alertness are frequent. Focal signs may be subtle or absent, but papilledema and meningeal signs are often found. Cavernous sinus thrombosis following infection of orbit, paranasal sinus produce orbital signs. Cerebral angiography is the definitive indication. Mortality is 15–30%, mainly results from cerebral

edema and secondary haemorrhagic infarction. Heparinization improves prognosis.

STROKE IN THE YOUNG

Ischemic stroke is often considered as a disease of middle aged and elderly persons but it does occur in patients aged 40 years or less. This may be due to improved recognition and increased prevalence of risk factors. Predominant causes are:

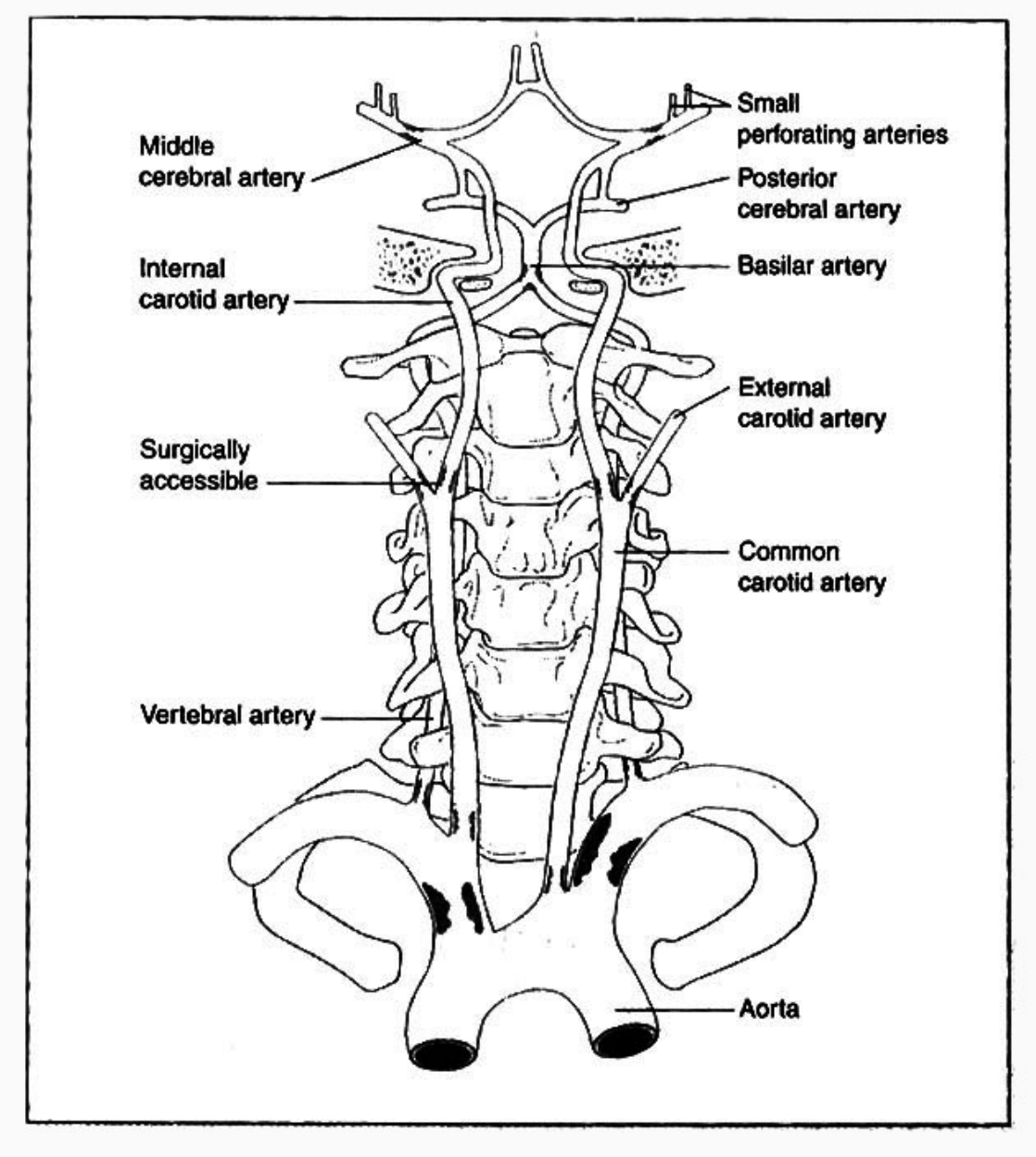
- **Cardiovascular:** Rheumatic heart disease, infective endocarditis, prosthetic valve, myxoma, mitral valve prolapse etc.
- **Non–atherosclerotic vasculopathies:** Tuberculosis, syphilis, collagen vascular disease, dissection, trauma, migraine etc.
- **Hypercoagulable states:** Ranging from disturbances in blood components, coagulation factors, platelet functions and deficiencies of clotting inhibitors or fibrinolytic system predispose to stroke.
- **Atherosclerosis:** May occur prematurely secondary to earlier onset of risk factors such as hypercholesterolemia, smoking, diabetes mellitus, hypertension.

Blood supply to the brain[95]

The blood supply to the brain is delivered by the two internal carotid and two vertebral arteries which anastomose at the base of the brain to form the circle of Willis. The carotid artery system supplies the anterior two-thirds of the

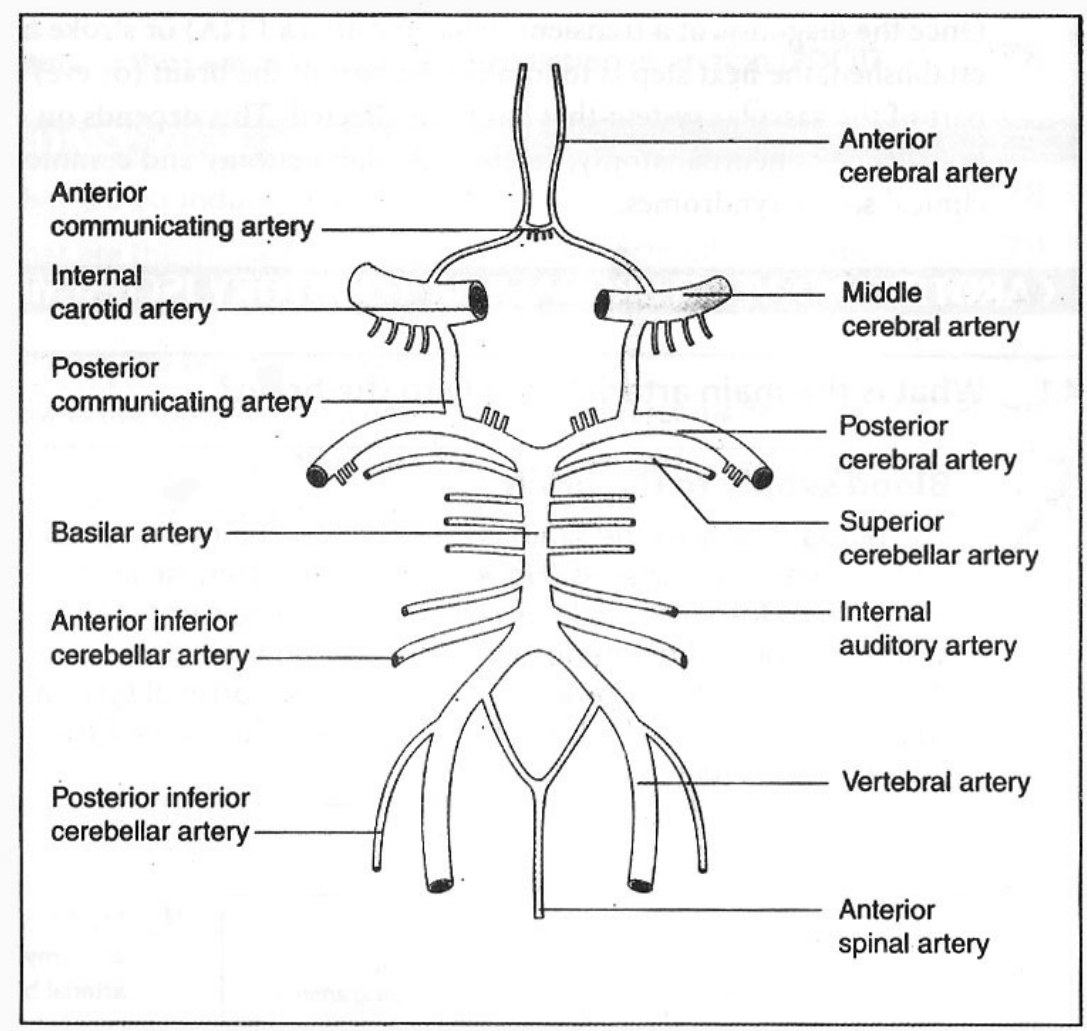
brain (hence it is called the *anterior circulation)* (Fig.1). The vertebrobasilar arterial system supplies the posterior third of the brain (hence it is called the *posterior circulation)* (Fig.2).

FIGURE - 1



The anatomy of the arterial blood supply to the brain. Sites that are most often affected by atherosclerosis are shown as dark indentations in the arterial lumen.

FIGURE – 2



Diagrammatic representation of the circle of Willis at the base of the brain, as seen from below in relation to the optic chiasma.

STROKE SYNDROMES [95]

Internal carotid artery territory (or) anterior circulation stroke[95]

Branches	Region supplied	Syndrome caused by ischemia
Ophthalmic artery	Retina and optic nerve	Monoocular blindness or altitudinal field detect
Anterior choroidal artery	Globus pallidus internal capsule choroid plexus	Contralateral hemiparesis, hemisensory loss and homonymous hemianopia
Middle cerebral artery	Frontal lobe	Contralateral facial weakness, hemiparesis and hemisensory loss (arm>leg), homonymous hemianopia, and global aphasia (dominant hemisphere) or visual-spatial – perceptual dysfunction (non-dominant hemisphere)
Medial lenticulostriate artery	Internal capsule	Contralateral pure motor hemiparesis
Lateral lenticulostriate artery	Putamen, globus pallidus, caudate nucleus, internal capsule, corona radiata	Contralateral hemiparesis, dysphasia (dominant hemispehre) or visual –spatial – perceptual dysfunction (non-dominant hemisphere)
Superior division of middle cerebral artery Prerolandic branch	Frontal and anterior parietal lobes	Contralateral central facial weakness, hemiparesis and hemisensory loss, ipsilateral deviation of head and eyes, and global or motor aphasia (dominant hemisphere) Contralateral face and

Branches	Region supplied	Syndrome caused by ischemia
Rolandic branch Anterior parietal branch		arm weakness, and motor aphasia (dominant hemisphere) Contralateral central facial weakness, hemiparesis and hemisensory loss, and dysarthria (resembling lacunar syndrome) conduction aphasia and bilateral ideomotor apraxia
Inferior division of middle cerebral artery Posterior parietal branch Angular branch Posterior temporal branch Anterior temporal branch Temporal polar branch	Inferior parietal and lateral temporal lobes	Homonymous hemianopia, Wernicke's aphasia or agitated confusional state (dominant hemisphere), left visual neglect (right-sided lesion)
Anterior cerebral artery	Anterior and superior medial frontal lobe	Contralateral foot and leg weakness or hemiparesis (leg>arm), abulia, incontinence, grasp reflexes

Vertebrobasilar territory (or) posterior circulation stroke[95]

Branches	Region supplied	Syndrome caused by ischemia
Vertebral and basilar artery	Brainstem and cerebellum	Various syndromes including: diplopia, ophthalmoplegia or gaze palsies; vertigo, nausea and nystagmus; dysarthria, dysphagia and bulbar weakness; ipsilateral facial sensory loss and weakness (nuclear or infranuclear); hiccups and respiratory failure contralateral hemiparesis or tetraparesis; contralateral or bilateral sensory loss; coma
Basilar artery Top of basilar artery	Rostral midbrain Part of thalamus Inferior temporal occipital lobes	Variable pupillary abnormalities Ptosis or lid retraction Supranuclear vertical gaze paresis Somnolence Hemiballismus Amnesia Cortical blindness
Superior cerebellar artery	Midbrain (dorsolateral) Superior cerebellar peduncle Superior cerebellum	Ipsilateral Horner's syndrome Ipsilateral limb ataxia and tremor Contralateral spinothalamic sensory loss Contralateral central facial weakness Sometimes contralateral IVth nerve palsy
Anterior inferior cerebellar artery	Base of pons Rostral medulla Inferior cerebellum Cochlea	Ipsilateral Horner's syndrome Ipsilateral facial sensory loss (pain, temperature) Ipsilateral nuclear facial and abducens palsy

Branches	Region supplied	Syndrome caused by ischemia
	Vestibule	abducens palsy Ipsilateral deafness and tinnitus Vertigo, nausea, vomiting and nystagmus Ipsilateral ataxia of limbs and dysarthria
Posterior inferior cerebellar artery	Lateral medulla Inferior cerebellum	Ipsilateral Horner's syndrome Ipsilateral facial sensory loss (pain, temp) Vertigo, nausea, vomiting and nystagmus Ipsilateral paralysis of palate (dysphagia) Ipsilateral paralysis of larynx (dysphonia) Ipsilateral ataxia of limbs Contralateral hemisensory loss below neck
Paramedian branches	Paramedian pons	Any of the lacunar syndromes: Pure motor hemiparesis Pure hemisensory loss Hemiparesis – hemisensory loss Ataxic hemiparesis Internuclear ophthalmoplegia Locked- in syndrome, if bilateral
Thalamic – subthalamic (Thalamoperforating)	Posteromedial thalamus inferiorly	Hemisensory loss, amnesia
Paramedian mesencephalic arteries	Rostral medial midbrain	Hemisensory-motor loss

Branches	Region supplied	Syndrome caused by ischemia
Posterior cerebral artery	Occipital lobe inferior temporal lobe	Contralateral homonymous hemianopia Cortical blindness if bilateral Amnesia (especially if bilateral)
Thalamogeniculate	Ventrolateral thalamus	Pure hemisensory loss
Posterior choroidal arteries	Anterior and posterior thalamus	Hemisensory loss, amnesia
Posterior communicating artery. Polar arteries (tuberothalamic)	Anterior lateral thalamus	Hemisensory loss, amnesia

Lacunar Stroke Syndromes

Infarct Location	Clinical Features
Posterior limb of internal capsule, crus cerebri midbrain or basis pontis	Pure motor hemiparesis face, arm, leg, foot and toes are almost always involved.
Ventrolateral thalamus	Pure sensory stroke
Base of the pons	Ataxic hemiparesis
Base of the pons or genu of the internal capsule	Dysarthria and a clumsy hand or arm
Genu and anterior limb of internal capsule and adjacent white matter of corona radiata	Pure motor hemiparesis with motor aphasia

COMPUTED TOMOGRAPHY

CT was invented by Godfrey N. Hounsfield.

The typical radiation dose for a plain head CT is 40 to 60 milligray.

Densities of various tissues in CT brain

Tissue	Density (Hounsfield units)	Gray Scale
Air	-1000	Black (↓↓↓)
Fat	-100	Black (↓↓)
CSF	0	Black (↓)
Brain	30	Gray (-)
Extravasated Blood	100	White (↑↑)
Contrast Medium Enhancement	100	White (↑↑)
Bone	1000	White (↑↑↑)

Note:

(↓↓↓) - marked hypoattenuation (↓↓)- moderate hypoattenuation

(↓) - mild hypoattenuation (-) Isoattenuation to brain

(↑↑) - moderate hyperattenuation

(↑↑↑) - marked hyperattenuation.

An early CT is advisable in all stroke patients due to two reasons.

a) To differentiate ischemic stroke from haemorrhage, as it could change the management.

b) To identify the presence of an underlying tumor, vascular malformation, haematoma that could mimic stroke.

Though acute infarcts are more frequently visible on MRI than on CT, the latter is preferred as it is widely available, can be done on an emergency basis and it fulfills the basic needs for immediate management. Also patient cooperation is less of a problem in CT. Contrast enhancement on CT helps to pick up additional cases of acute infarction by showing relative hypodensity of involved area as compared to normal enhancing brain or by showing evidence of luxury perfusion.

It is also essential in patients with stroke who may require treatment with tissue plasminogen activator.

CT is also helpful in identifying patients at higher risk for haemorrhage. If an infarction is clearly visible and is atleast one half the size of the MCA territory, then the age of the infarct is likely to be >3 hrs or severe damage has already occurred. In such patients the risk of haemorrhage is high and these patients should not be treated with tissue plasminogen activator.

CT scan exposes the patient to minimal radiation and can be repeated if there is clinical suspicion of new infarction or recurrent haemorrhage (or) haemorrhage into an infarct (especially in patient on anticoagulation therapy).

CT findings in cerebral infarction

State	Findings
Hyperacute (<12 hrs)	Normal (50-60%) Hyperdense artery (25-50%) Obscuration of lentiform nuclei
Acute (12-24 hrs)	Low density basal ganglia Loss of grey-white matter interface (insular ribbon sign) Sulcal effacement
1-3days	Mass effect Wedge shaped low density area involving grey and white matter. Haemorrhagic transformation, gyral enhancement
4-7 days	Gyral enhancement Mass effect, edema persists
1-7weeks	Contrast enhancement persists Mass effect resolves
Months to years	Encephalomalacic change, Volume loss Rarely calcification

Early signs of cerebral edema

a. Effacement of sulci and the sylvian fissure

b. Decrease in size of the ipsilateral ventricle.

Cerebral edema involves both the gray and white matter and is restricted to the zone of infarction. The edema pattern may be helpful in differentiation from brain tumor which rarely involves the gray matter.

The use of contrast agents may be necessary when the diagnosis is unclear or a neoplasm is suspected. Contrast agents must be used only in selected cases as they may adversely affect prognosis or increase edema.

CT brain in Intracerebral haemorrhage

Among laboratory methods for the diagnosis of intracerebral haemorrhage the CT scan occupies the foremost position. This procedure has proved totally reliable in the detection of haemorrhages that are 1.0 cm or more in diameter.

If the volume of haematoma from CT is less than 30 ml it favours a better outcome. If volume of haematoma calculated from the CT brain is ≥ 60 ml the prognosis is poor.

Lacunar infarcts in internal capsule, thalamus, brainstem and cerebellum are difficult to be detected by CT.

The major limitation of CT is in the posterior fossa where linear artifacts appear because bone selectively attenuates the X-ray beam. The resulting beam hardening creates dense or lucent streaks that project across the brainstem and may obscure underlying lesions.

Helical CT can be done in an uncooperative patient as time require to perform it is less. There is some loss of resolution with this technique as scanning time decrease such that in routine brain imaging for stroke patients, helical scanning is not employed.

MAGNETIC RESONANCE IMAGING (MRI)

MRI offers greater sensitivity and specificity in the detection of acute infarcts than CT. This is due to its substantially better soft tissue resolution,

grey-white matter differentiation and its ability to image in multiple planes. MRI is usually not indicated for emergency diagnosis, reasons being :

- Not easily available
- Monitoring of ill patients difficult within the MRI machine
- Time required to perform MRI is greater than CT
- Acute SAH can be easily missed on MRI.

MRI is presently used where available as a problem solving modality and is specifically useful in the diagnosis of ischemic stroke involving brainstem and cerebellum. MRI is preferred as the first modality in patients with suspected posterior fossa infarcts and in patients with TIAs.

MRI Findings in Cerebral Infarction

State	Findings
Immediate	Intravascular contrast enhancement Alteration of perfusion-diffusion co-efficient.
< 12 hrs	Anatomic changes of T_1 images (gyral thickening, sulcal effacement, loss of grey white interface).
12-24 hrs	Hyperintensity, mass effect, Leptomeningeal enhancement
1-3 days	Obvious abnormality on T_1, T_2 images (early parenchymal contrast enhancement, haemorrhagic transformation).
4-7 days	Parenchymal enhancement haemorrhage 25%
1-8 weeks	Mass effect resolves, Decreased signal on T_2 images enhancement persists Haemorrhagic signal evolves
Months to years	Encephalomalacic changes, volume loss, Haemosiderin staining

ELECTROCARDIOGRAPHIC CHANGES IN STROKE

Cardiovascular regulation is carried out by descending pathways from forebrain which converge in hypothalamus, hence cerebrovascular accident can produce changes in the ECG. These changes are often misdiagnosed as organic. Stroke can cause ST elevation or depression and 'Q' waves but plasma CPK levels do not rise. These changes normalize in 2 weeks time. Commonest changes observed are T wave inversion, sinus tachycardia. Other changes include ST depression, sinus QT prolongation, conduction disturbances and ectopic rhythms.

THROMBOLYTIC THERAPY IN STROKE

In the recent years, the use of thrombolytic therapy in cerebral infarction has been studied extensively[66]. The European co-operative acute stroke study (ECASS) tested intravenous recombinant tissue plasminogen activator (rtPA : 1.1 mg/kg to a max. 100 mg; 10% as bolus, the remainder over 60 min) within 6 hrs of onset of symptoms.

The National institute of neurological disorders and stroke (NINDS) study tested rtPA(0.9 mg/kg to a 90 mg max; 10% as bolus and the remainder over 60 min) within 3 hours of onset of symptoms. Improved clinical outcome and decreased bleeding hazard observed in NINDS study was probably due to lower dose of rtPA and earlier institution of therapy.

A recent trial of the fibrinolytic agent ancrod in ischemic stroke is being evaluated. Recent trials PROACT I and II (Prolyse in acute cerebral thromboembolism) using intra-arterial thrombolysis upto the sixth hour showed benefit. However intra-arterial thrombolysis is not approved by the FDA and remains experimental.

REHABILITATION

Proper rehabilitation of stroke patient includes, early physical, occupational and speech therapy. It is directed towards educating the patient and family about the neurological deficit, preventing complications of immobility, and providing encouragement and instruction in overcoming the deficit. The goal of rehabilitation is to return the patient to home early and to maximize recovery by providing a safe regimen suited to the individual patient.

Materials and Methods

MATERIALS AND METHODS

Patients with cerebrovascular accident satisfying the inclusion criteria, admitted in our hospital during the period from January 2005 to December 2005 were studied.

Inclusion Criteria

Patients with a clinical diagnosis of stroke and on whom CT has been done were included. The various types of stroke in this group were:

- Ischemic stroke
- Haemorrhagic stroke
    - Intraparenchynal
    - Subarachnoid haemorrhage
- Cortical vein thrombosis

Exclusion Criteria

- Neurological deficit lasting for less than 24 hrs.
- Cases presenting 48 hrs after onset.
- Space Occupying lesions
- Subdural haemorrhage
- Extradural haemorrhage
- Patients with history of head injury.
- Patients in whom CT Scan brain could not be performed.

A detailed history in each case regarding onset, predisposing factors and nature of stroke was recorded, followed by a thorough clinical examination.

The major risk factors included in the study were hypertension, diabetes mellitus, hyperlipidemia along with other factors such as age, sex, menopause, puerperium which were evaluated. History of TIA was noted. In young patients family history of stroke and history of exposure to sexually transmitted disease were recorded. Habituations to smoking, alcohol was noted. Special clinical evaluation of cardiovascular system giving importance to rhythm disturbances, cardiac failure, valvular heart disease, prosthetic valve was made.

Clinical neurological examination was done and the patients were grouped into those having cortical involvement, internal capsular involvement or brainstem involvement delineating the major blood vessels involved in the process. Ophthalmoscopic examination of the fundus was done. Patients with blood pressure readings of ≥160/90 mm Hg on two occasions was taken as hypertension.

Baseline investigations included complete blood count, blood sugar, urea, creatinine, electrolytes, cholesterol, urine analysis and chest x-ray. ECG was taken at admission, after 3 days and at discharge. CT Brain was done for all patients. Echocardiography and CSF analysis were done in relevant cases.

The results of all data are expressed in tabular forms for analysis. For the analysis of ECG, those patients giving a definite history or evidence of diabetes

mellitus, coronary artery disease, rheumatic heart diseases, electrolyte disturbances or those on anti-arrhythmatic drugs were avoided.

Observations and Results

OBSERVATIONS AND RESULTS

TABLE – 1

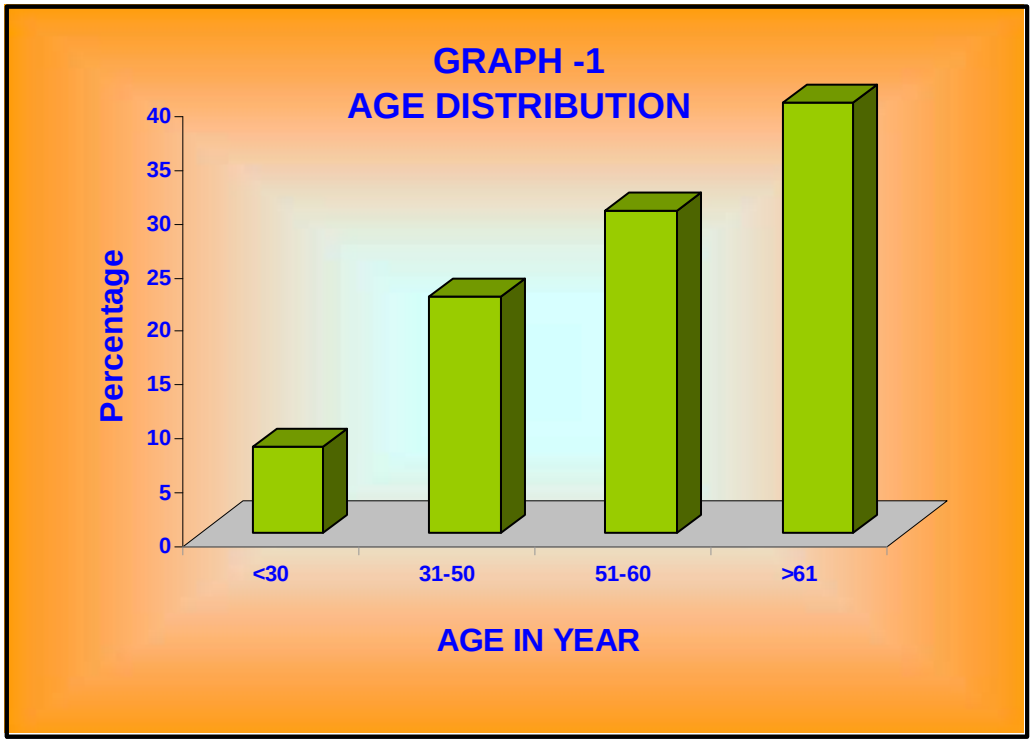
AGE DISTRIBUTION

Age in Year	Number (n=100)	Percentage
< 30	8	8
31 – 50	22	22
51 - 60	30	30
> 61	40	40

TABLE – 2

SEX DISTRIBUTION

Age in Year	Male	Female
Less than 50	15	15
More than 50	47	23
Total	**62**	**38**


GRAPH -1
AGE DISTRIBUTION
Percentage
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35
30
25
20
15
10
5
0
<30
31-50
51-60
>61
AGE IN YEAR

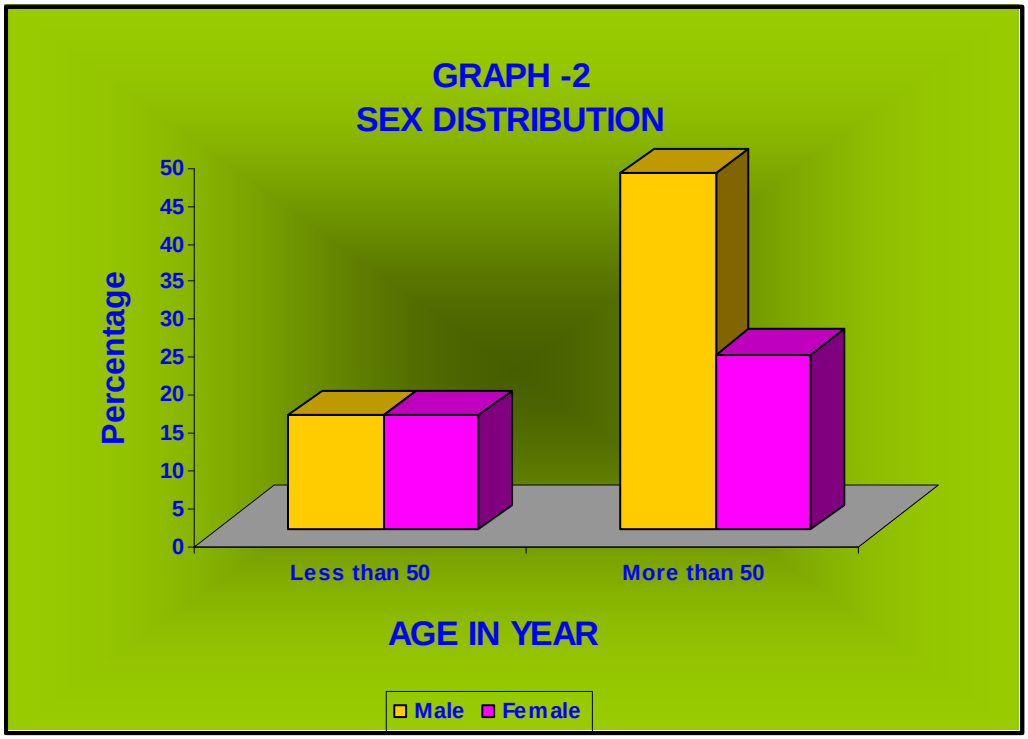

GRAPH -2
SEX DISTRIBUTION
Percentage
50
45
40
35
30
25
20
15
10
5
0
Less than 50
More than 50
AGE IN YEAR
Male
Female

TABLE – 3

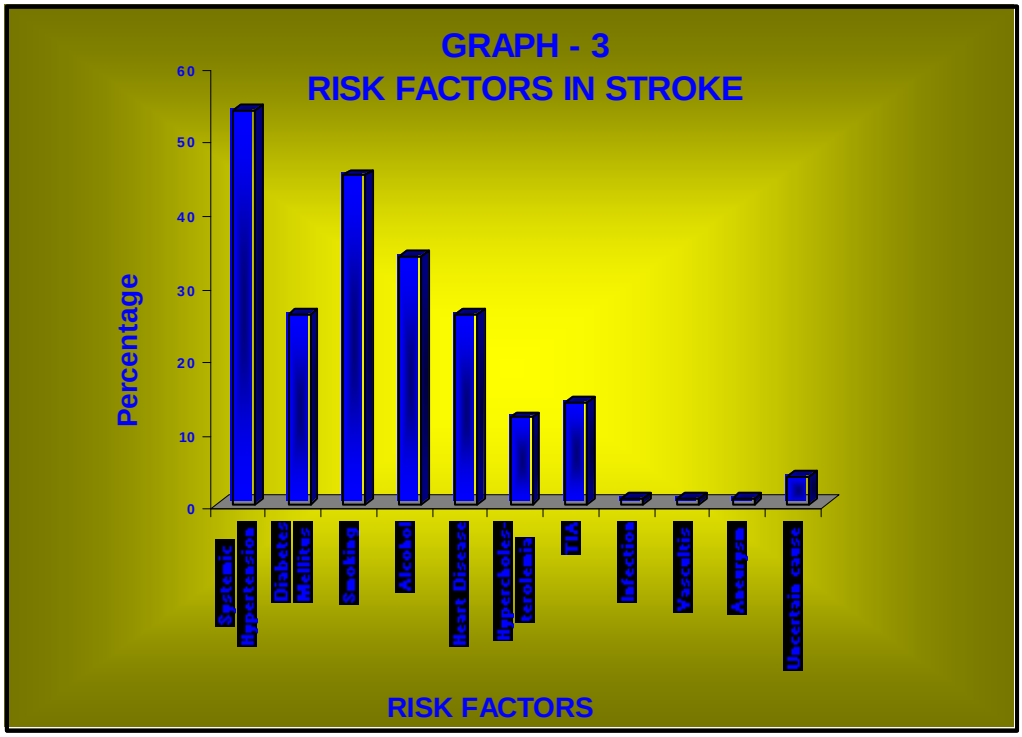
RISK FACTORS IN STROKE

S.No.	Risk Factors	Number	Percentage
1.	Systemic Hypertension	54	54
2.	Diabetes Mellitus	26	26
3.	Smoking	45	45
4.	Alcohol	34	34
5.	Heart Disease	26	26
6.	Hypercholesterolemia	12	12
7.	Transient Ischemic Attack	14	14
8.	Infection	1	1
9.	Vasculitis	1	1
10.	Aneurysm	1	1
11.	Uncertain cause	4	4

TABLE – 4

INCIDENCE OF CARDIAC DISEASES ASSOCIATED WITH STROKE

Disease	Number (n=26)	Percentage
Coronary Artery Disease	15	57.7
Rheumatic Heart Disease	7	26.9
Mitral Valve Prolapse	4	15.4


GRAPH - 3
RISK FACTORS IN STROKE
Percentage
60
50
40
30
20
10
0
Systemic Hypertension
Diabetes Mellitus
Smoking
Alcohol
Heart Disease
Hypercholes-terolemia
TIA
Infection
Vasculitis
Aneurysm
Uncertain cause
RISK FACTORS

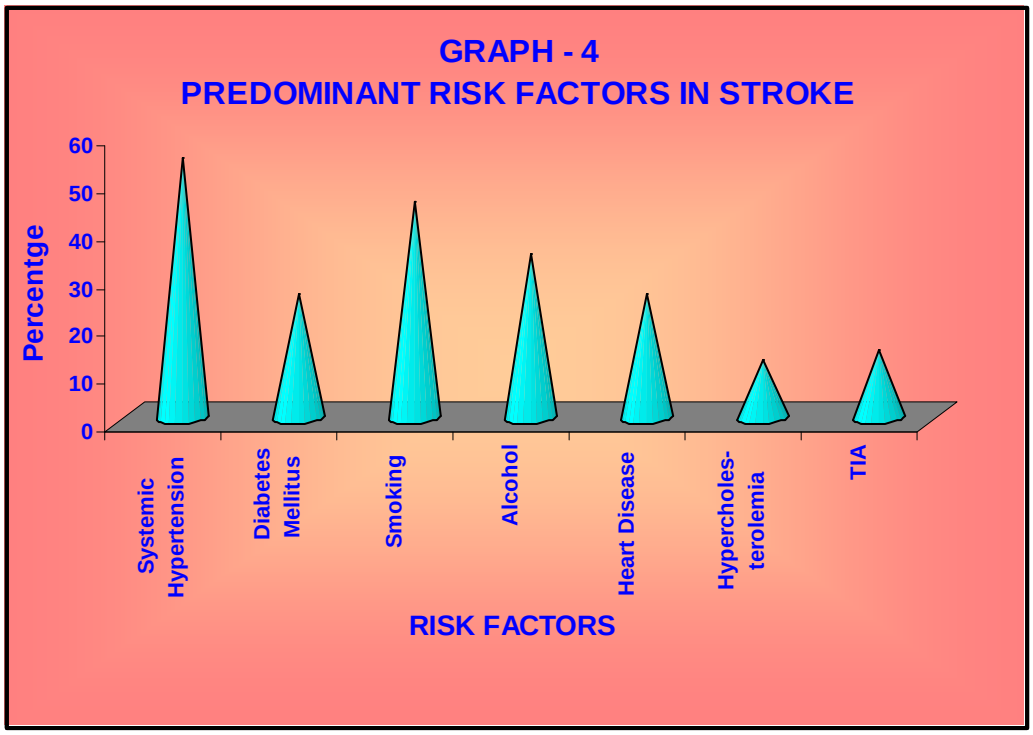

GRAPH - 4
PREDOMINANT RISK FACTORS IN STROKE
Percentge
60
50
40
30
20
10
0
Systemic Hypertension
Diabetes Mellitus
Smoking
Alcohol
Heart Disease
Hypercholes-terolemia
TIA
RISK FACTORS

TABLE – 5

CLINICAL PRESENTATION

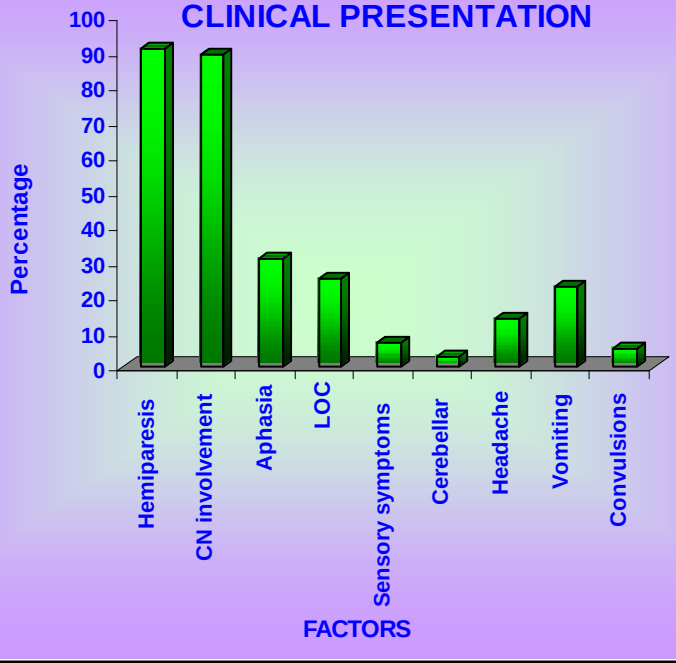
S.No.	Factors	Number	Percentage
1.	Hemiparesis	91	91
2.	Cranial nerve Involvement	89	89
3.	Aphasia	31	31
4.	Loss of consciousness	26	26
5.	Sensory symptoms	7	7
6.	Cerebellar	3	3
7.	Headache	14	14
8.	Vomiting	23	23
9.	Convulsions	5	5

TABLE – 6

APHASIAS

Factors	Number (n=31)	Percentage
Global	22	71
Motor	7	22.6
Sensory	2	6.4

GRAPH - 5
CLINICAL PRESENTATION



PIE CHART - 1
APHASIAS

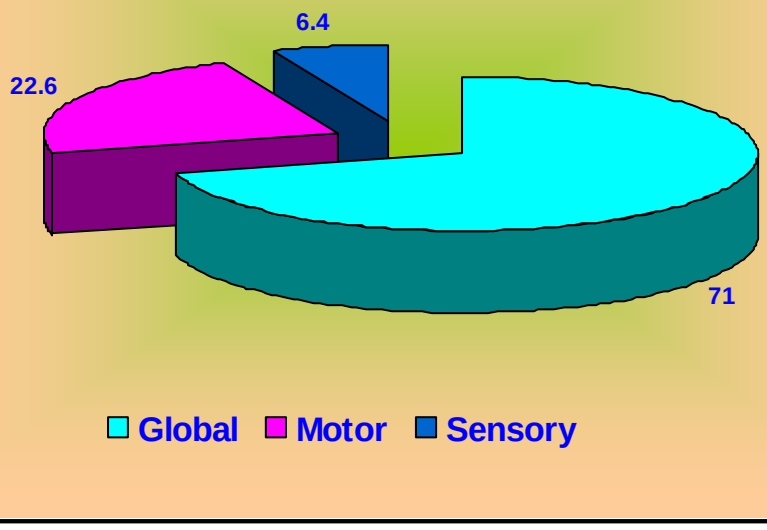


TABLE 7

TIME OF OCCURRENCE OF STROKE

Time	Thrombotic (n=70)		Embolic (n=12)		Haemorrhagic (n=18)	
	No	%	No	%	No	%
4 am – 8 am	31	44.3	5	41.7	8	44.4
8 am – 12 pm	10	14.3	4	33.3	4	22.2
12 pm - 4 am	29	41.4	3	25.0	6	33.3

TABLE 8

SIGNIFICANCE OF ACTIVITY

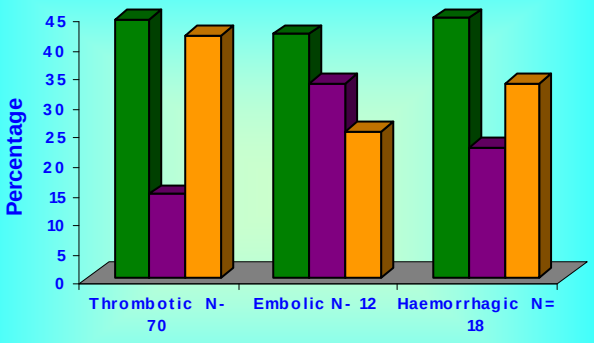
Factor	Activity		Sleep	
	No	%	No	%
Thrombotic (n=70)	30	42.9	40	57.1
Embolic (n=12)	9	75	3	25.0
Haemorrhagic (n=18)	14	77.8	4	22.2

TABLE 9

NATURE OF STROKE

S.No.	Stroke	No	Percentage
1.	Ischemic	82	82
2.	Haemorrhagic	18	18

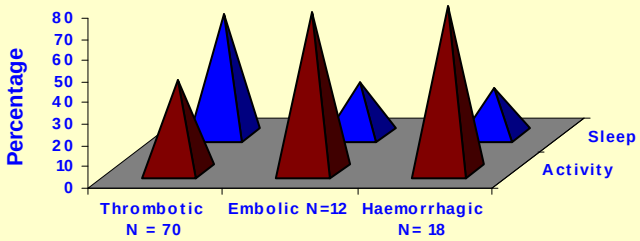
GRAPH -6
TIME OF OCCURRENCE OF STROKE



TIME

4 AM - 8 AM 8 AM - 12 PM 12PM- 4 AM

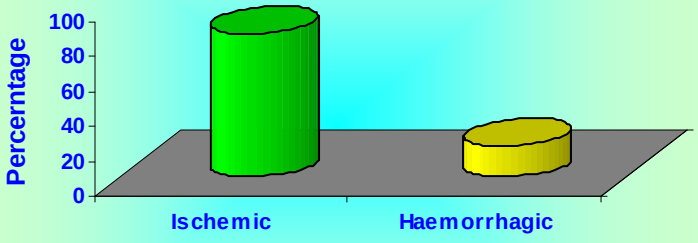
GRAPH - 7
SIGNIFICANCE OF ACTIVITY



FACTOR

Activity Sleep

GRAPH - 8
NATURE OF STROKE



STROKE

TABLE 10

AREA OF THE BRAIN INVOLVED IN ISCHEMIC STROKE

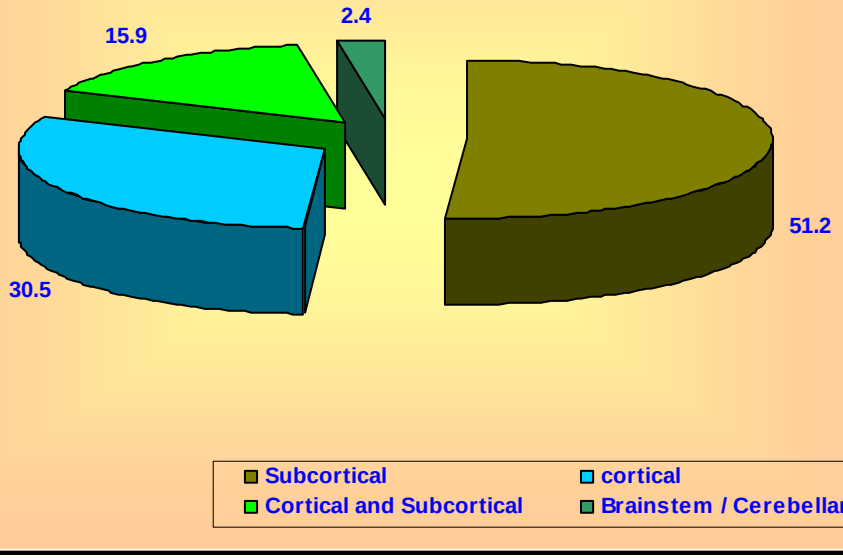
S.No.	Factor	No	Percentage
1.	Subcortical	42	51.2
2.	Cortical	25	30.5
3.	Cortical and Subcortical	13	15.9
4.	Brainstem /Cerebellar	2	2.4

TABLE 11

DISTRIBUTION OF ARTERIAL TERRITORY IN ISCHEMC STROKE

S.No.	Artery Involved	No (n=82)	Percentage
1.	MCA	73	89
2.	PCA	4	4.9
3.	Multiple	2	2.4
4.	MCA, PCA	1	1.2
5.	MCA, ACA	1	1.2
6.	ACA	1	1.2

PIE CHART - 2
AREA OF THE BRAIN INVOLVED IN ISCHEMIC STROKE



PIE CHART - 3
DISTRIBUTION OF ARTERIAL TERRITORY IN ISCHEMIC STROKE

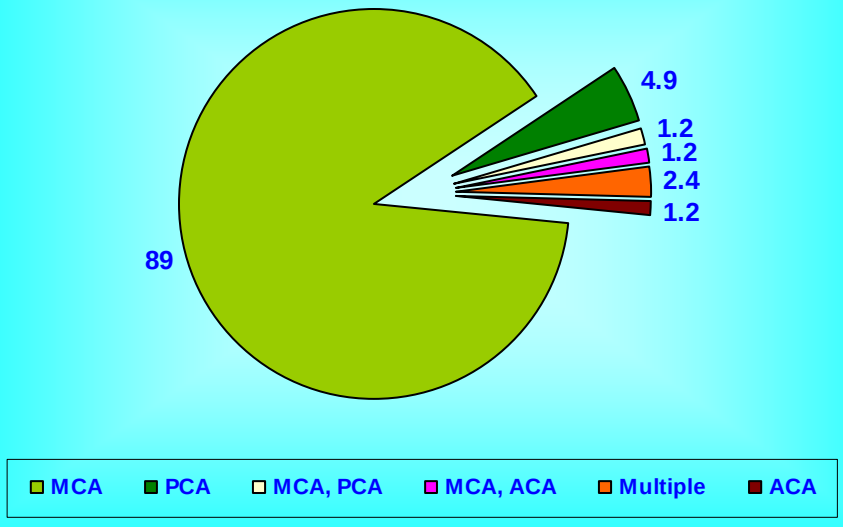


TABLE 12

LOCATION OF HAEMORRHAGE

S.No.	Factor	No (n=18)	Percentage
1.	Putamen	7	38.9
2.	Cortical	4	22.2
3.	Putamen, Thalamus	3	16.7
4.	Pons	2	11.1
5.	Cerebellum	1	5.5
6.	Subarachnoid Haemorrhage	1	5.5

TABLE 13

ECG CHANGES IN STROKE

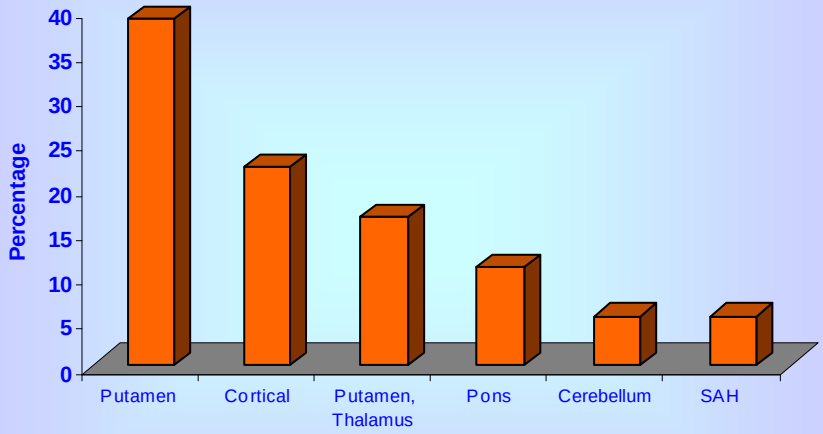
S.No.	ECG	No (n=25)	Percentage
1.	T wave Inversion	10	40
2.	Sinus Tachycardia	8	32
3.	Sinus Bradycardia	4	16
4.	ST Depression	3	12
5.	Conduction Disturbance	2	8
6.	VPC	1	4
7.	Normal	2	8

TABLE 14

LEVEL OF CONSCIOUSNESS AND PROGNOSIS

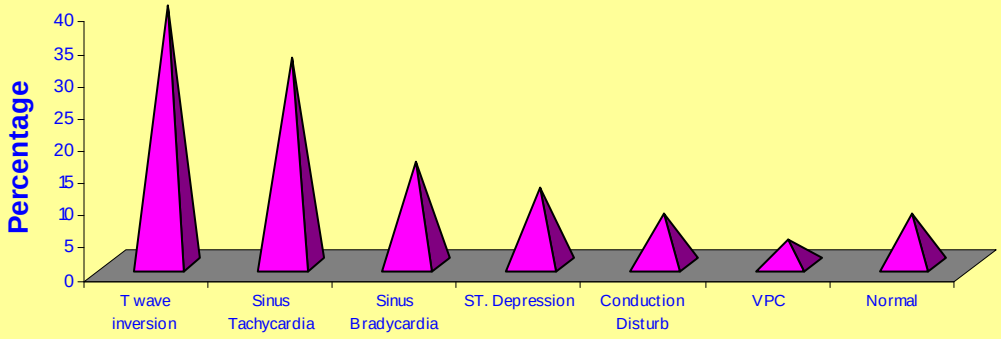
Factor	LOC (n=26)		Death	
	No	%	No	%
Ischemic Stroke	11	42.3	5	45.5
Haemorrhagic Stroke	15	57.7	13	86.7

GRAPH - 9
LOCATION OF HAEMORRHAGE



SITE OF HAEMORRHAGE

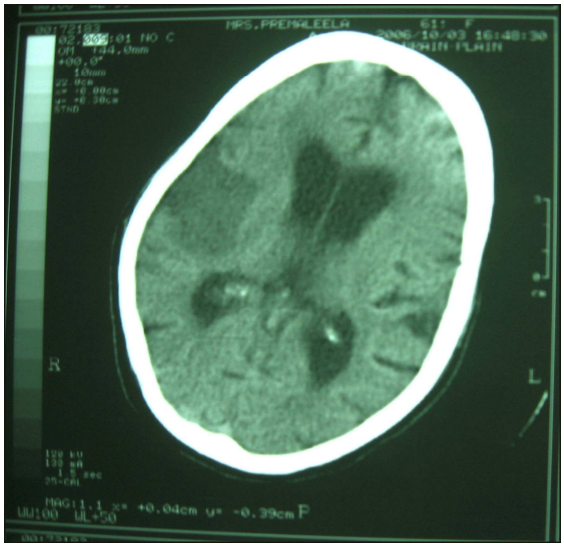
GRAPH - 10
ECG CHANGES IN STROKE



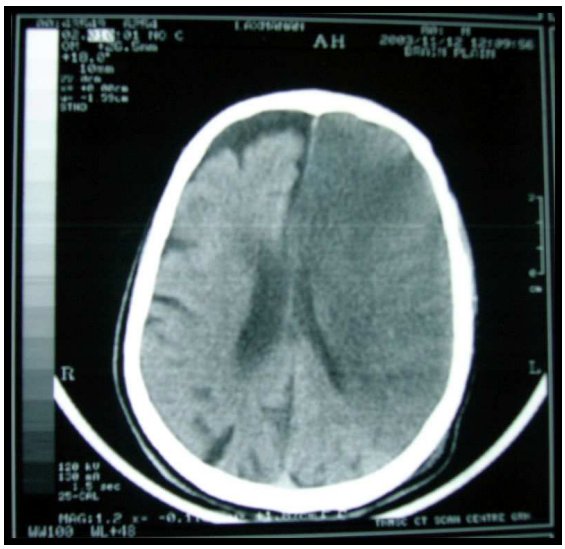
ECG CHANGES

CT SCAN PICTURES

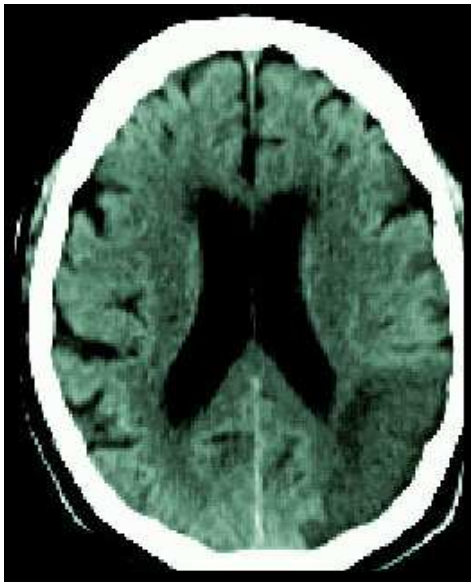
Right MCA Infarct



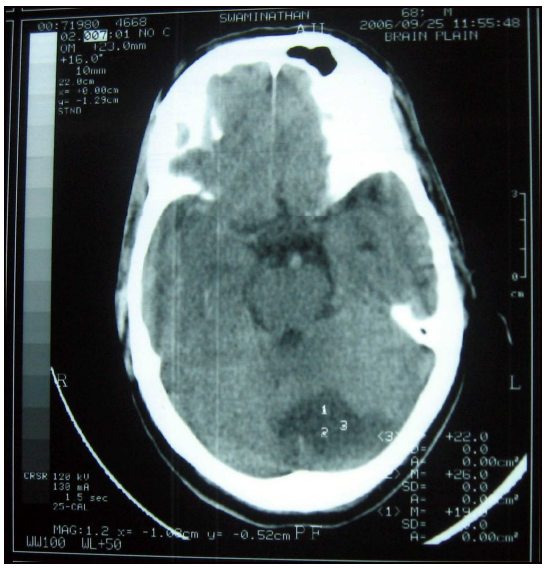
Left ACA and MCA Infarct



Left PCA Infarct



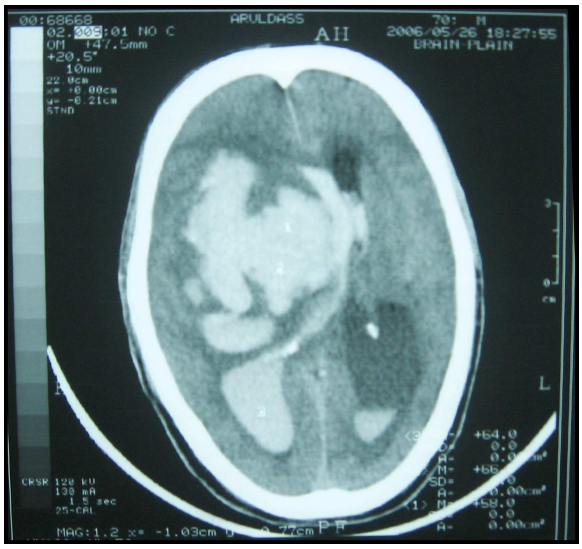
Left Cerebellar Infarct

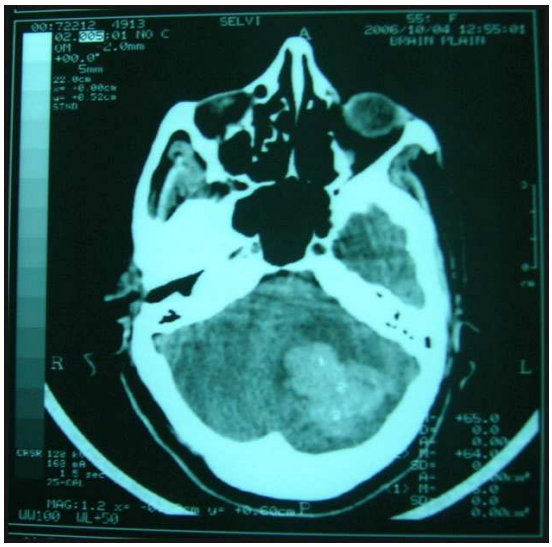


CT SCAN PICTURES

Large Right Intra Cerebral Bleed With Intra Ventricular Extension

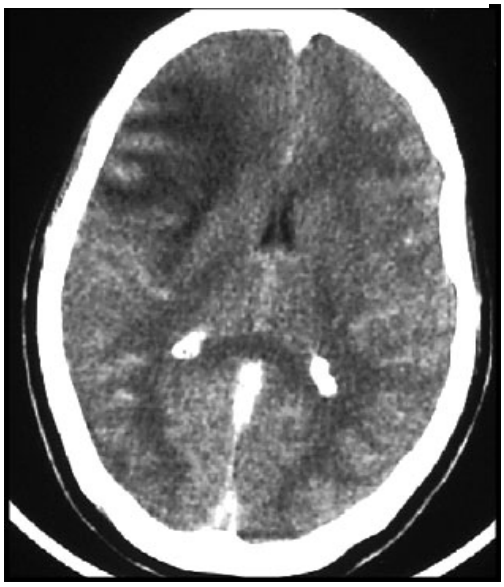
Left Cerebellar Haemorrhage With Fourth Ventricular Extension

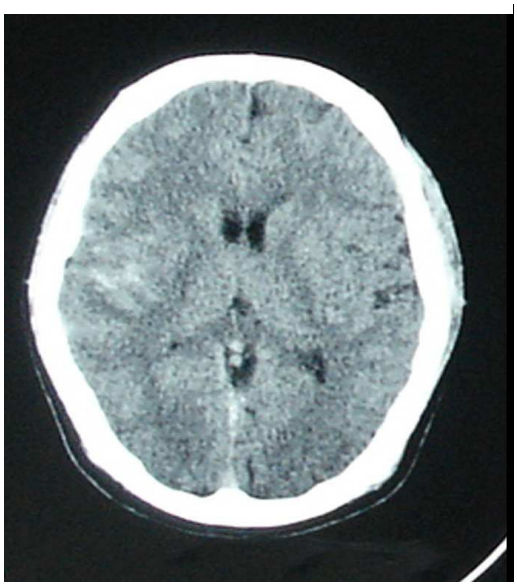




Cortical Venous Thrombosis

Subarachnoid Haemorrhage





Analysis of Data and Discussion

ANALYSIS OF DATA AND DISCUSSION

Age

The maximum incidence of stroke in this study was observed in the above 60 yrs age group (40%). According to Banford and Sandercock et al., 1987, maximum incidence of stroke was in the above 70 years age group. The Framingham study (1979) showed that 80% of patients, were above 65 years of age. Compared to Framingham study, a lower incidence of stroke in this study could be explained by:

- Many cases of old stroke not being brought to the hospital.
- A high mortality rate in cases of old stroke.
- Higher prevalence of rheumatic heart disease in India compared to western population.
- Majority of young strokes being hospitalized.

In this study incidence of stroke in below 50 years age group was 30%, and above 50 yrs age group was 70%. This corelates well with the Vellore study by Abraham J. Inbaraj et al., where the incidence below 40 years was 27% and above 40 years was 73%, and the Rohtak rural study, where the prevalence of stroke below 40 yrs, was 28.8%. However in the Kashmir rural study, the prevalence below 40 years was 41.1% (Koul RL et al.).

Sex

With regard to sex distribution, the male to female ratio in this study was 1.6:1. In the below 50 years age, ratio was equal and in the above 50 years age group ratio was 2:1. Higher female preponderance in the younger age group could be attributed to higher prevalence of rheumatic heart disease.

In a study by Das Gupta et al., in 1984 male to female ratio was 1.3 : 1, which is almost similar to this study[67]. In the Madurai study by Aleem MA et al., 1996, the male to female ratio in young strokes was 1.1:1, which corelates well with this study[68]. Study done by Jotideb Mukhopadhyay et al., 1998, concluded an equal incidence in males and females[69].

An epidemiological study by K.R. Dhamija et al., 1998, recorded a male to female ratio of 1.7 : 1, which is in accordance with this study[70]. In a study published in Kashmir (1989), males constituted 64% of stroke patients. In the Saudi study by Qari FA et al., 2000, the male to female ratio was 3.4:1.

RISK FACTORS

(i) Systemic Hypertension

Hypertension was the commonest predisposing factor in this study, present in 54% of the patients. Among the patients with haemorrhagic stroke, 77.8% were hypertensives. The Framingham 18 years follow up study shows that hypertension was the most powerful precursor of stroke in both infarction and haemorrhage, which tallies with this study. In a population study of stroke patients in Richmond by Williams CA et al., 2003, 58.3% of patients were hypertensives, which corelates well with this study[71]. In Oxford Shire community stroke project, hypertension was present in 52% patients and Review of stroke studies by Dalal and Dalal gives a figure of 51%, which is similar to this study.

In a study by Rahman KMN et al., 2002, 78.82% of stroke patients were hypertensives[72], and a study by Aszal C et al., 2002, documented hypertension in 75% of patients[73]. Risk factors analysis by Jotideb et al., recorded 76% as hypertensives[69], while an epidemiological study by K.R. Dhamija et al., showed 34.7% as hypertensives[70]. Studies by Nielson W.B. et al., and Hart CL et al., 2002 state that elevation of both systolic and diastolic blood pressure is associated with increased risk of stroke[74].

ii) Diabetes Mellitus

Diabetes mellitus was present in 26% of stroke patients in this study. In the Oxford Shire community stroke project, the incidence of diabetes mellitus in stroke was 28% which corelates well with this study. According to a study Manorama Devi in. “The incidence of stroke among Indian Nationals in UAE” diabetes was present in 20% of patients.

The incidence and severity of stroke are increased by the presence of diabetes and the outcome from stroke are poorer, according to a study by Baird TA et al., 2002[75]. Study by Wannamathe et al., 1999 confirms diabetes mellitus as an independent risk factor for stroke[76]. Similar conclusions were drawn from the study of Hark CL et al., in 2000[9].

iii. Smoking

Smoking was a risk factor in 45% of patients in this study. None of the females were smokers. According to a study by Williams CA et al., 2003, 33.3% of ischemic stroke patients and 26.3% of haemorrhagic stroke patients were smokers[71]. In a study by Ansari K et al., 2001, 29% of elderly stroke patients were smokers. Study on multiple cerebral infarcts by B Reddy et al., recorded a figure of 62%[77].

Christensen et al., 2001, concluded that stroke patients who smoke are on an average nine years younger than non-smokers[78]. Study done by Bonita R et al., 1999 demonstrated that active as well as passive smoking increases the risk of stroke[79].

iv. Alcohol consumption

Alcohol consumption was present in 34% of patients in this study. Study done by William CA et al., 2003 concluded that 41.6% of ischemic stroke patients and 21.1% of haemorrhagic stroke patients were alcoholics[71]. A study on multiple cerebral infarcts done by B Reddy et al., showed 30% of stroke patients were alcoholics[77].

Studies by Reynolds K et al., 2003[80] and Wannamathe SG et al., 1996 state that light or moderate alcohol consumption may be protective, while heavy alcohol consumption increases risk of stroke largely mediated through blood pressure[76]. Relationship of alcohol and stroke can be due to non-valvular atrial fibrillation or cardiomyopathy (Lancet – 1995).

v. Heart disease

26% of patients in this study had cardiac disease associated with stroke. Among the cardiac diseases, coronary artery disease was present in 15 patients (57.7%), rheumatic heart disease in 7 patients (26.9%) and mitral valve prolapse in 4 patients (15.4%). Study by Ansari K et al., 2001, showed that 23% of stroke patients had cardiac disease. Studies by B Reddy et al.,[77] Kundu TN et al.,[81] Aleem M.A. et al.,[68] documented an incidence of 36%, 35% and 30.8% respectively.

The incidence of cardiac causes of embolic stroke in this study was 11%, of which 7% contributed by rheumatic heart disease with atrial fibrillation and

4% mitral valve prolapse with infective endocarditis. This shows that rheumatic heart disease with atrial fibrillation is the predominant cause of cardioembolic stroke. This is in accordance with studies by V.K. Gupta in Kashmir and A. Panagriya, Jaipur et al.,

The incidence of coronary artery disease in stroke patients in this study was 15%. Among them, one patient developed stroke 4 days after the onset of acute myocardial infarction. Studies by Mooe T et al., 1999[82] and Errikcson P et al.,[83] 1997 state that the risk for stroke is highest in the first 5 days after myocardial infarction, predictors being atrial fibrillation, ST elevation and previous stroke. A study by Loh E et al. 1997, state that decreased ejection fraction and older age are predictors for risk, while anti-coagulation appears to have a protective effect against stroke after acute myocardial infarction[84].

vi. Hypercholesterolemia

In this study 12% of patients had elevated total cholesterol levels, indicating a causal relationship between stroke and lipid profile. Similar observations were also made by Shridharan, Apollo Hospitals, Chennai, H. Jacobs, D.R. Wentworth (1999), and Prospective studies collaboration the Lancet 1995[85]. The literature does not give a clear relation between serum lipids and stroke. Studies by NA Rajwade et al., (1996)[86] Sarti C et al., (2000)[87] state that the lipid levels in stroke patients were observed to be higher but not

significant and their associations are relatively weak. A study by Wannamathe SG et al., (2000) concludes that elevated total cholesterol showed a weak positive association, while elevated HDL cholesterol was associated with a significant decrease in the risk of stroke[88]. However studies by Nair M. Radhakrishnan et al., at Kerala and Jotideb et al., West Bengal, suggested that hyperlipidemia is an important risk factor for stroke[69]. It may be concluded that hypercholesterolemia is a risk factor for stroke, although the risk imparted is lower than that for myocardial infarction.

vii. Transient Ischemic Attack and Other Factors

In this study, past history of stroke or Transient ischemic attacks were present in 14% of patients. According to a study by William CA et al. (2003) history of prior neurovascular disease was present in 36.2%[71]. MM Singh et al., (1996) in his study stated that transient ischemic attack is associated with mild to moderate stenosis of internal carotid artery and is an important predictor of ischemic stroke. Other etiological factors in this study include infective endocarditis, tuberculous meningitis and subarachnoid haemorrhage secondary to aneurysms.

CLINICAL PRESENTATION

- Hemiparesis was the commonest presentation in this study, present in 91% of patients. This is in accordance with the study by Rahman KM et al., (2002) where hemiplegia was present in 88.24%[72].

- 26% of patients presented with loss of consciousness. 83.3% of haemorrhagic stroke patients and 22% of ischemic stroke patients had altered consciousness level. Study by Rahman KM et al., suggested that 54.84% of haemorrhagic stroke patients present with altered consciousness[72].
- Headache and vomiting were present in 14 and 23 patients respectively. Incidence of headache and vomiting were more in the haemorrhagic stroke patients and among the ischemic stroke patients, it was more common in posterior circulation stroke.
- 5 patients presented with convulsions, out of which 2 had embolic stroke, 1 patient had thrombotic stroke in the cortical territory and 2 had haemorrhagic stroke.
- Language disturbances were observed in 31 patients. 22 had global aphasia, 7 had motor aphasia and 2 patients had sensory aphasia. These presentations correlated well with involvement of cortical territory.
- 7 patients had sensory disturbances, out of which 3 had thalamic infarct, one patient had thalamic haemorrhage, one had lateral medullary syndrome, one had brainstem stroke and one cortical infarct.
- Cerebellar involvement was observed in 3 patients. One had lateral medullary syndrome, one had cerebellar haemorrhage and the last one had brainstem stroke with cerebellar involvement.

Time of occurrence

In this study, 44.3% of thrombotic stokes, 41.7% of embolic strokes and 44.4% of haemorrhagic strokes occurred in the early morning hours between 4 am and 8 am. A study by Lago A et al., (1998) observed a higher frequency of strokes during the day and lower frequency in the last hours of the evening in all types of stroke, which is in accordance with this study[89]. Similar observations, were made by Chaturvedi S. et al., (1999)[90] and Casetta I et al., (2002) in ischemic stroke[91]. They suggest the circadian variability in blood pressure and a concurrent morning hypercoagulability as possible determinants of this pattern.

57.1% of thrombotic strokes occur during sleep (40% in Michael Reel Stroke Registry)[92]. 75% of embolic strokes and 77.8% of haemorrhagic strokes occurred during activity in this study (68% and 64% respectively in Michael Reel stroke registry).

CT SCAN CORRELATION

- CT scan is the single most important non invasive investigation to distinguish infarction from cerebral haemorrhage.
- After 6 hrs, CT brain may show lesion in <1/3 of patients with cerebral infarct and at 12 hrs 50% show evidence of infarction. Over 90% supratentorial infarctions (moderate to large) can be detected within 24 hrs.

- In this study, out of the 100 patients studied, CT Scan showed ischemic stroke in 82% and haemorrhage in 18%. Studies by K.R.Dhamija et al.,

(1998)[70] and Quari FA et al., (2000) observed cerebral infarction in 82.6% and 77% respectively, which tallies with this study.

- Regarding the distribution of arterial territory in ischemic stroke, MCA was involved in 89%, PCA in 4.9%, ACA in 1.2%. Involvement of more than one arterial territory was observed in 4.8%. Study by K. Srinivasa Rao et al., documented 54% in the MCA, 4.5% in the PCA, 4.5% in the ACA, and 27% at multiple sites.
- With respect to the area of the brain involved in ischemic stroke, 51.2% had subcortical and 30.5% cortical infarcts, combination of both was observed in 15.9%. Brainstem and cerebellar lesions found in 2.4%. In a study by Sandya Purohit et al., (1999) cortical infarcts were found in 18%.
- Among the 18 patients with haemorrhagic stroke, 7 patients had putamen haemorrhage (38.9%), cortical haemorrhage in 4 (22.2%), thalamic haemorrhage in 3 (16.7%). 2 patients had pontine haemorrhage and both of them expired. Cerebellar and subarachnoid haemorrhage were documented in one patient each. According to a study by K. Srinivasa Rao et al., (1998), 40% haemorrhages occurred in basal ganglia, and 27% in the thalamic region.
- Among laboratory methods for diagnosis of intracerebral haemorrhage the CT scan occupies the foremost position. This procedure has proved totally reliable in the detection of haemorrhages that are 1.0 cm or more in diameter. If the volume of haematoma from CT is less than 30ml it

favours a better prognosis. If volume of haematoma calculated from the CT brain is ≥ 60 ml the prognosis is poor.

- The distinct appearance of the fresh haemorrhage changes with time, disappearing slowly in the subsequent days to weeks, depending on the size of the haemorrhage. Thereafter the haemorrhage appears as a region of low densities on CT scan, which can be mistaken for an old infarct. So if CT imaging is delayed for more than a few weeks after stroke, it may not possible to reliably distinguish between ischemic and haemorrhagic stroke
- Hypertension was the commonest predisposing factor in this study, present in 54% of patients. Among patients with haemorrhagic stroke as evidenced by CT scan 86.6% of them were hypertensives.
- Diabetes mellitus was present in 26% of stroke patients in this study. Among the patients with diabetes mellitus CT scan showed increased incidence of infarct than haemorrhage.
- Other important risk factors such as alcohol consumption, hypercholesterolemia, TIA were present in 34%, 12%, 14% of the patients respectively: with CT scan showing increased preponderance to infarct pattern.
- Cardiac causes namely RHD, MVPS increases the incidence of ischemic embolic stroke which in this study was 11% as shown by the infarcts in CT scan.

- Haemorrhagic stroke as evidenced from CT scan showed increased incidence of headache and vomiting. However among the infarct patients it was more associated with posterior circulation strokes.
- Convulsions occurred in 11% of patients with haemorrhagic stroke and 0.4% of ischemic stroke. Among the patients with ischemic strokes convulsions was more common in cortical infarcts.
- Among the haemorrhagic stroke revealed by CT scan, 83.3% presented with loss of consciousness where as it was 22% in ischemic stroke.
- Sensory disturbances were more common in patients with thalamic and cortical infarcts as revealed by CT scan.
- Patients with language disturbances had more incidence of cortical territory infarct in CT scan.
- In this study, CT scan showed cerebellar involvement in 3 patients, one had lateral medullary syndrome, one had cerebellar haemorrhage and other one had brainstem stroke with cerebellar involvement.

ECG CHANGES IN STROKE

For the analysis of ECG, patients with coronary artery disease, rheumatic heart disease, those with definite history of diabetes and hypertension, renal failure patients, those on anti-arrythmic drugs were excluded. Out of the 100 patients, 25 satisfying the criteria were selected and their ECGs were analysed.

The commonest change observed was T wave inversion and sinus tachycardia present in 40% and 32% respectively. Sinus bradycardia in 16% and

ST depression in 12%. 8% had conduction disturbance, Ventricular premature contractions observed in 4% and 8% had normal ECG.

PROGNOSIS

Loss of consciousness at the time of admission is considered to be an adverse prognostic factor. In this study, 5 out of the 11 ischemic stroke patients and 13 out of the 15 haemorrhagic stroke patients, presenting with loss of consciousness expired. On an average, 69.2% of patients presenting with loss of consciousness died. Studies of M Das et al., (1999) and IS Gambhir et al., (1998) state that level of consciousness at admission by Glasgow coma scale is a very important prognostic factor and 82.14% of patients with a score less than 8 expired.

Similar observations were made by Gambir IS et al.,[93] (1998) and Joshi PP et al ., (1999)[94]. Study by Masquardsen et al., states that nearly all patients who were deeply comatosed at the time of admission died within 24 hrs. Semicomatosed patients also fared badly. The above results were comparable to this study.

Conclusion

CONCLUSIONS

- The risk factors were present in more than 90% of cases.
- The incidence of stroke was more common in the above 60 years age group.
- The incidence in males was one and a half times more than in the females, with the difference being negligible or absent in the less than 50. This is attributed to increased prevalence of rheumatic heart disease in young females.
- Hypertension was the commonest risk factor in both ischemic and haemorrhagic strokes, present in more than half of the patients, followed by smoking, and diabetes mellitus.
- There is a weak positive association between hypercholesterolemia and stroke.
- Heart disease was the predominant risk factor in the young, of which rheumatic mitral stenosis with atrial fibrillation was found to be the most common, followed by mitral valve prolapse with infective endocarditis.
- The presence of atherosclerosis increases the risk of stroke.
- Risk for ischemic stroke doubles for smokers. In smokers, stroke occurs after an average of 10-20 years of smoking.
- Only about 12% of patients experienced transient ischemic attack.
- Other important causes of stroke in the young were vasculitis and aneurysmal rupture.

- Hemiparesis was the commonest presenting feature in both ischemic and haemorrhagic stroke followed by cranial nerve involvement, aphasia and loss of consciousness.
- Headache and vomiting were more common in haemorrhagic stroke. In ischemic stroke, it was more common in the posterior circulation stroke.
- Convulsions were more common in embolic stroke involving cortical territory.
- Peak incidence of stroke occurred between 4 am to 8 am irrespective of the type of stroke.
- More than half of thrombotic strokes occur during sleep. Nearly three fourths of embolic and haemorrhagic strokes occur during activity.
- Majority of strokes were found to be infarcts, commonest being the middle cerebral artery territory followed by posterior cerebral artery.
- Among ischemic strokes, involvement of sub-cortical structures i.e. Internal capsule, putamen, basal ganglia were more common compared to cortical structure.
- Among haemorrhagic strokes, putamen involvement was the commonest followed by cortical haemorrhage.
- Commonest ECG change observed was T wave inversion and sinus tachycardia.
- Loss of consciousness is an adverse prognostic factor with a mortality rate of 69%.

- Computed Tomography is likely to remain the principal imaging technique for stroke patients in foreseeable future, not only because it excludes several non-stroke pathologies and reliably distinguishes haemorrhage from infarction, but because it is far more widely available, relatively less expensive, easier and safer to use in acutely ill stroke patients.
- CT scan is also essential in patients with stroke who require thrombolytic treatment with tissue plasminogen activator.

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Proforma

PROFORMA

Name	:	Study No.	:
Age/Sex	:	IP No.	:
Occupation	:	DOA	:
Place	:	DOD	:

HISTORY

PRESENT HISTORY

- Weakness of limbs
- Sensory disturbance
- Cranial Nerve palsies
- Language disturbance Global / Motor / Sensory aphasia
- Memory disturbance
- Cerebellar symptoms
- Headache, Vomiting, Convulsions
- Loss of Consciousness
- Autonomic nervous system symptoms
- Activity/ Sleep, Time of onset
- Trauma, Fever

PAST HISTORY

- Transient ischemic attack
- Stroke
- Diabetes mellitus
- Systemic hypertension
- CAD
- Rheumatic heart disease
- Intermittent claudication
- H/O oral contraceptive intake

FAMILY HISTORY

Stroke / Diabetes mellitus / Systemic hypertension / CAD

PERSONAL HISTORY

- Smoker - / Day Years
- Alcoholic - / Day Years

MENSTRUAL HISTORY

- Pre – Menopausal
- Post – Menopausal

GENERAL EXAMINATION

Febrile / Afebrile Anemia / Cyanosis / Clubbing / Pedal edema.

Pulse	:		Peripheral pulses	:
BP	:		Respiration	:
CVS	:	S1 S2, murmurs		
RS	:	NVBS , added sounds		
P/A	:	Bowel sounds, organomegaly		

CNS

- Handedness
- Conscious/Semi–conscious / Unconsciousness
- Orientation
- Speech-Normal/Aphasia-Global/Motor/Sensory
- Cranial nerve palsies
- Motor weakness
- Sensory abnormalities
- Cerebellum
- Carotids
- Fundus
- Gait
- Involuntary movements
- Signs of meningeal irritation

CLINICAL DIAGNOSIS:

INVESTIGATIONS

TC	DC	Hb%
PCV	ESR	Platelet count
Blood -	Sugar	
	Urea	Urine Complete
	Creatinine	
	Electrolytes	CSF analysis
Serum Cholesterol		
Chest X-ray	ECG	
CT Brain	MRI	
Echocardiography	Carotid Doppler	

FINAL DIAGNOSIS:

Abbreviations

ABBREVIATIONS

ACA	-	Anterior Cerebral Artery
AF	-	Atrial Fibrillation
ASMI	-	Anteroseptal Myocardial Infarction
BG	-	Basal Ganglia
BS	-	Brainstem
C	-	Cortical
CAD	-	Coronary Artery Disease
CHO	-	Serum Cholesterol
CN	-	Cranial Nerve
CT	-	Computed Tomography
DIC	-	Disseminated Intravascular Coagulation
DM	-	Diabetes Mellitus
E	-	Expired
ECG	-	Electrocardiogram
ECHO-	Echocardiogram	
EM	-	Embolic
G	-	Global Aphasia
H	-	Haemorrhage
I	-	Infarction
IE	-	Infective Endocarditis
IVCD	-	Intra Ventricular Conduction Defect
IWMI	-	Inferior Wall Myocardial Infarction
LH	-	Left Hemiparesis
LMS	-	Lateral Medullary Syndrome
LOC	-	Loss of Consciousness
M	-	Motor Aphasia
MCA	-	Middle Cerebral Artery
MI	-	Myocardial Infarction
MR	-	Mitral Regurgitation

MS	-	Mitral Stenosis
MVP	-	Mitral Valve Prolapse
N	-	Normal
P	-	Putamen
PCA	-	Posterior Cerebral Artery
PH	-	Past History of Stroke
PHT	-	Pulmonary Hypertension
R	-	Recovered
RBBB	-	Right Bundle Branch Block
RH	-	Right Hemiparesis
RHD	-	Rheumatic Heart Disease
S	-	Sensory Aphasia
SAH	-	Sub Arachnoid Haemorrhage
SB	-	Sinus Bradycardia
SC	-	Subcortical
SHT	-	Systemic Hypertension
ST	-	Sinus Tachycardia
ST↓	-	ST Depression
T	-	Thalamus
T↓	-	T wave Inversion
TBM	-	Tuberculous Meningitis
Th	-	Thrombotic
TIA	-	Transient Ischemic Attack
VEG	-	Vegetation

Master Chart

MASTER CHART

S. No.	Age	Sex	Time of occurrence	Activity/ Sleep	Weakness	Cranial Nerve	Aphasia	Sensory	Headache /Vomiting	LOC/Fits	SHT/DM	Smoking /Alcohol	TIA/PH	CAD /RHD	CHO	ECG	ECHO	Clinical Diagnosis	CT			Outcome
																			I/H	Artery	Cortical/ Subcortical	
1	55	M	7.00PM	S	RH	7	-	-	-	-	DM	S/A	-	-	120	T ↓	-	I-TH	I	MCA	SC	R
2	65	M	11.00PM	S	RH	7	G	-	-	-	SHT	S/A	-	-	176	ST	DD	I-TH	I	MCA	C,SC	R
3	50	M	3.00PM	S	LH	7	-	-	-	-	SHT	S/A	TIA	-	133	ST	-	I-TH	I	MCA	SC	R
4	58	M	4.00PM	A	LH	7	-	-	-	-	-	S/A	-	-	225	VPC	-	I-TH	I	MCA	C	R
5	85	F	7.00AM	S	LH	7	M	-	-	-	-	-	PH	-	234	T↓	-	I-TH	I	MCA	SC	R
6	65	M	2.00PM	A	RH	7	G	-	-	-	-	S	-	-	177	T↓	-	I-TH	I	MCA	C	R
7	45	F	2.00AM	S	RH	7	G	-	-	LOC/F	-	-	TIA/PH	RHD	176	RAD, LAE, AF	MS. MR, PHT	RHD, I-EM	I	MCA	C	E
8	62	M	11.00PM	S	LH	-	-	-	-	-	SHT	-	PH	-	142	ST	-	I-TH	I	MCA	SC	R
9	68	F	9.00PM	A	LH	-	-	-	-	-	SHT/DM	-	-	-	205	LVH	-	I-TH	I	MCA	SC	R
10	48	M	3.00PM	A	RH	7	-	-	-	-	SHT	-	-	-	136	N	-	I-TH	I	MCA	SC	R
11	60	M	11.00AM	A	LH	7	-	-	H/V	LOC	SHT/DM	S	-	-	148	IVCD, LVH	-	H	H	-	P,T,IC	E
12	70	M	12.00AM	S	RH	7	-	-	-	-	SHT/DM	-	-	-	153	N	-	I-TH	H	-	P,BG,T,IC	R
13	36	F	12.00PM	A	RH	7	G	-	H	-	-	-	-	-	172	LVH	MVP, MR, VEG+	I-EM	I	MCA	C,SC	E
14	70	F	11.00AM	S	RH	7	G	-	-	F	SHT/DM	-	-	-	156	N	-	I-TH	I	MCA	C	R
15	72	M	3.00PM	A	RH	7	-	-	V	LOC	SHT	S/A	-	-	175	ST	-	H	H	-	C	E
16	49	M	6.00AM	A	LH	7	-	-	V	LOC	SHT	S/A	-	-	165	N	-	I-TH	I	MCA	C	R
17	75	F	5.00AM	A	RH	7	-	+	-	LOC	SHT	-	-	-	172	N	DD, LVH	I-TH	H	-	P,BG,T,IC	E
18	60	M	6.00AM,	S	LH	7	-	-	-	-	SHT	S	-	-	185	ST	-	I-TH	I	MCA	SC	R
19	60	M	7.00AM	A	RH	7	G	-	-	-	SHT	S/A	-	-	192	IVCD	-	I-TH	I	MCA	SC	R
20	58	M	2.00PM	S	RH	7	G	-	-	-	-	S	-	-	210	ST	-	I-TH	I	MCA	C	R
21	54	M	9.00PM	A	RH	7	-	-	-	-	-	S/A	-	-	175	SB	-	I-TH	I	MCA	SC	R
22	81	M	9.00PM	A	LH	7	-	-	-	LOC	DM	S/A	-	-	180	ST, ST ↓	-	I-TH	I	MCA	SC	R

23	48	M	10.00AM	A	RH	7	-	-	-	-	-	S	-	-	186	SB, LVH	MVP, MR	I-EM	I	MCA	SC	R
24	70	F	7.00AM	S	LH	7	-	-	-	-	SHT	-	-	-	157	N	DD, LVH	I-TH	I	MCA	SC	R
25	78	F	2.00PM	S	RH	7	G	-	V	-	-	-	-	-	142	T ↓	-	I-TH	I	MCA	C,SC	R
26	18	F	8.00AM	S	LH	7	G	-	H	-	-	-	-	-	150	ST	-	I-TH-EBM	I	MCA	C	E
27	65	F	7.00AM	A	LH	7	-	-	H	LOC/F	SHT	-	-	-	126	N	-	H	H	-	P,BG	R
28	52	M	8.00PM	A	Cerebellar	9,10,5	-	+	H	-	SHT/DM	S/A	-	-	134	N	-	I-TH	I	PCA	LMS	E
29	50	M	6.00A	A	LH	7	-	-	V	LOC	SHT	A	-	CAD	136	OLD IWMI	-	H	H	-	P.BG	E
30	73	M	11.00PM	A	-	-	-	+	-	LOC	DM	S/A	-	CAD	164	ST T	-	H	I	PCA	C	E
31	75	M	10.00AM	S	RH	7	G	-	-	-	DM	-	-	-	200	T ↓	-	I-TH	I	MCA	C	R
32	85	F	7.00AM	S	LH	7	-	-	-	-	SHT	-	-	-	140	LVH, ST, ST↓	DD, LVH	I-TH	I	MCA	SC	R
33	48	M	7.00AM	S	RH	7,12	-	-	-	-	SHT/DM	S	-	-	188	N	-	I-TH	I	MCA	SC	R
34	60	F	4.00PM	A	LH	7	-	-	V	-	SHT/DM	-	TIA	-	136	ST	-	I-TH	I	ACA,MCA	C	R
35	40	F	4.00PM	A	LH	7	-	-	-	-	-	-	-	RHD	148	LAD, AF, RBBBT	MS, PHT	I-EMB, RHD	I	MCA	SC	R
36	80	F	6.00AM	S	LH	7	-	-	-	-	SHT	-	-	CAD	150	LBBB LAD	-	I-TH	I	MCA	SC	R
37	36	M	11.00AM	S	LH	7	-	-	-	-	-	S/A	-	-	140	ST, T ↓	-	I-TH	I	MCA	SC	R
38	75	M	6.00 AM	S	RH	7	G	-	-	-	SHT/DM	S/A	-	-	158	ST	DD	I-TH	I	MCA	C,SC	R
39	60	F	6.00AM	S	-	7,8,9,10	-	+	-	-	SHT/DM	-	-	-	174	T LVH	-	I-TH	I	PCA	C,SC	R
40	60	F	6.00PM	A	Cerebellar	-	-	-	-	-	SHT/DM	-	-	-	144	LVH	-	I-TH	H	-	Cerebellar	R
41	80	F	8.00PM	A	LH	7	-	-	H	LOC	SHT	-	-	-	196	T ST↓	-	H	H	-	SC-P, BG	E
42	60	F	4.00AM	S	LH	7	-	-	-	-	SHT	-	-	-	198	N	-	I-TH	I	MCA	SC	R
43	65	M	8.00PM	A	-	-	-	-	V	LOC	SHT	S/A	-	-	174	LVH	-	H	H	-	C	E
44	55	F	6.00AM	S	RH	7	S	-	-	LOC	SHT	-	-	-	124	LVH	-	I-TH	H	-	C	E
45	70	F	6.00AM	S	RH	7	-	-	-	-	SHT/DM	-	-	-	176	ST	DD	I-TH	I	MCA	SC	R
46	73	M	3.00AM	S	RH	7	-	-	-	-	SHT	S	-	-	186	T↓	DD, LVH	I-TH	I	MCA	SC	R
47	52	M	10.00AM	A	RH	7	G	-	-	-	SHT/DM	S	-	-	146	ST	-	I-TH	I	MCA	C,SC	R

48	58	M	3.00PM	A	LH	7	-	-	-	-	SHT	S	-	-	152	ST	-	I-TH	I	MCA	SC	R
49	55	F	10.00AM	A	LH	7	-	-	-	-	-	-	-	-	164	T↓, ST ↓	-	I-TH	I	MCA	SC	R
50	58	F	7.00AM	S	-	-	-	-	H/V	LOC	SHT/DM	-	PH	-	178	ST T ST ↓	DD	H	I	MCA, PCA	C,SC	E
51	48	F	2.00PM	A	-	-	-	-	-	LOC/F	SHT	-	PH	CAD	182	ST T	-	H	H	-	PONTINE	E
52	45	M	8.00AM	A	RH	7	M	-	-	-	-	S/A	-	-	234	IVCD ST ↓ RBBB	-	I-TH	I	MCA	SC	R
53	55	M	6.00AM	S	LH	7	-	-	-	-	-	S/A	-	-	114	SB	-	I-TH	I	MCA	SC	R
54	64	M	2.00PM	S	RH	7	-	-	-	-	SHT	-	-	-	163	T↓	DD	I-TH	I	MCA	SC	R
55	50	M	4.00PM	S	RH	7	G	+	-	-	-	S/A	-	CAD	162	T↓	-	I-TH	I	MCA	C,SC	R
56	36	F	5.00AM	A	-	-	-	-	-	LOC	-	-	PH	-	124	ST	MVP, VEG+ AML	I-EM, MVP IE	I	MCA	C	E
57	53	M	5.00AM	S	RH	7	G	-	-	LOC	SHT	S/A	-	CAD	134	ST, T↓	-	I-TH	I	MCA	C	R
58	65	M	8.00PM	S	LH	7	-	-	-	-	SHT	S/A	-	-	208	N	-	I-TH	I	MCA	SC	R
59	60	M	7.00PM	S	RH	-	-	-	-	LOC	DM	S/A	-	-	222	RBBB	-	H	H	-	PONTINE	E
60	20	F	6.00AM	S	RH	7	-	-	H/V	-	-	-	-	RHD	154	RAD, LAE, AF	MS, PHT	RHD, I-EM	I	MCA	C	E
61	70	M	4.00AM	S	RH	7	G	-	-	-	SHT	S/A	-	-	170	T↓	-	I-TH	I	MCA	C	R
62	75	M	4.00PM	S	RH	7	M	-	-	-	SHT	S/A	-	-	128	T↓	DD, LVH	I-TH	I	MCA	C	R
63	55	M	6.00AM	S	LH	7	-	-	H	-	-	S/A	-	-	182	ST, T↓	-	I-TH	I	MCA	SC	R
64	55	F	8.00AM	A	RH	7	S	-	-	-	SHT/DM	-	-	-	194	IVCD	-	I-TH	I	MCA	C	R
65	65	M	5.00AM	S	LH	7	-	-	-	-	DM	-	-	-	136	ST, IVCD	-	I-TH	I	MCA	SC	R
66	38	M	8.00AM	A	RH	7	M	-	-	LOC	SHT	A	-	CAD	196	T↓	-	I-TH	I	MCA	C,SC	R
67	65	M	2.00PM	A	RH	7	-	-	-	-	SHT	-	-	-	142	N	-	I-TH	I	MCA	SC	R
68	40	F	4.00AM	S	LH	7	-	-	-	-	-	-	-	-	210	N	MVP, VEG+	I-EM, MVP IE	I	MCA	SC	E
69	29	F	1.00PM	A	RH	7	-	-	V	F	-	-	PH	RHD	156	LAE, RAD, ST	MS, PHT	I-EM, RHD	I	MCA	SC	R
70	30	F	2.00PM	A	LH	7	-	-	V	-	-	-	-	RHD	132	LAE, RAD, AF	MS, MR	I-EM, RHD	I	MCA	SC	R
71	72	M	7.00AM	S	RH	7	G	-	-	-	SHT	-	-	-	194	N	-	I-TH	I	MCA	C	R

72	80	M	8.00AM	S	LH	7	-	-	-	-	DM	-	-	CAD	126	ST	-	I-TH	I	MCA	SC	R
73	68	M	2.00PM	A	LH	7	-	-	V	-	SHT	-	-	-	187	N	DD	I-TH	I	MCA	SC	R
74	28	F	4.00PM	A	RH	7	G	-	V	-	-	-	-	RHD	146	LAE, RAD, AF	MS	RHD, I-EM	I	MCA	C	E
75	70	M	9.00PM	A	LH	7	-	-	-	-	DM	-	-	CAD	134	T↓	-	I-TH	I	MCA	SC	R
76	74	M	8.00AM	S	RH	7	-	-	-	-	SHT	-	-	-	158	DD	-	I-TH	I	MCA	SC	R
77	30	F	6.00PM	A	LH	7	-	-	V	-	-	-	PH	RHD	128	RAD, LAE, AF	MS,MR ,PHT	RHD, I-EM	I	MCA	SC	R
78	40	M	8.00AM	A	-	-	-	-	H/V	LOC	SHT	S/A	-	-	148	N	-	H	SAH	-	-	E
79	39	M	8.00AM	S	RH+Cer ebellar	7,8,9,10	-	+	-	-	-	S/A	-	CAD	136	ST	-	I	I	PCA	BS	E
80	40	M	10.00AM	A	RH	7	M	-	-	-	DM	S/A	-	CAD	160	T↓	-	I-TH	I	MCA	C	R
81	52	M	2.00AM	A	RH	7	M	-	-	-	DM	S/A	TIA	CAD	145	ST, T↓	-	I-TH	I	MCA	C	R
82	73	M	1.00A	S	LH	7	-	-	H/V	LOC	SHT	S	PH	-	134	ST, T↓	DD, LVH	I-TH	I	MULTI PLE	C,SC	E
83	60	M	8.00PM	A	RH	7	G	-	-	LOC	SHT	A	-	-	146	ST	-	I-TH	I	MCA	C	R
84	42	M	4.00PM	A	LH	7	-	-	H/V	LOC	-	-	-	-	154	ST↓	-	I-TH	H	-	C	E
85	60	M	5.00PM	A	RH	7	G	-	-	-	-	S	-	-	183	T↓	-	I-TH	I	MCA	C	R
86	50	F	6.00AM	A	RH	7	-	-	V	LOC	SHT	-	-	-	138	N	-	I-TH	I	MCA	SC,C	R
87	62	F	8.00AM	S	RH	7	M	-	V	-	SHT	-	-	-	152	T↓	-	H	I	MCA	C	R
88	50	M	12.00AM	S	RH	7	G	+	H/V	-	SHT	S	-	-	170	LVH	-	I-TH	I	MULTI PLE	C,SC	E
89	58	M	8.00AM	A	LH	7	-	-	-	-	-	S	-	-	139	T↓	-	I-EM	I	MCA	SC	R
90	82	F	4.00AM	S	RH	7	-	-	V	-	SHT	-	-	-	217	LVH	DD, LVH	I-TH	H	-	P,BG	R
91	38	M	10.00PM	A	LH	7	-	-	-	-	-	S	-	-	182	ST	-	I-TH	I	MCA	SC	R
92	73	M	6.00AM	A	LH	7,6	-	-	-	-	SHT	S	-	-	210	ST	DD, LVH	I-TH	I	MCA	SC	R
93	60	M	6.00AM	A	LH	7	-	-	-	-	-	S	-	-	163	T↓	-	H	I	MCA	SC	R
94	73	M	8.00AM	A	RH	7	-	-	V	LOC	DM	S/A	-	CAD	161	ASMI	-	I-TH	H	-	BG	E
95	68	M	6.00AM	S	RH	7	G	-	H/V	-	DM	A	PH	CAD	145	ST	-	I-TH	I	MCA	C,SC	R
96	60	M	11.00PM	A	RH	-	-	-	-	-	-	A	-	CAD	174	T↓	-	I-TH	I	ACA	SC	R
97	30	F	10.00AM	A	RH	7	G	-	-	LOC	-	-	-	-	132	T↓	-	H	H	-	P	E

98	68	F	7.00AM	A	LH	7	-	-	-	LOC	SHT	-	-	-	118	N	-	H	H	-	P,BG	R
99	60	F	7.00PM	S	RH	7	-	-	-	-	-	-	-	-	230	SB	-	I-TH	I	MCA	SC	R
100	69	M	2.00PM	A	LH	7	-	-	-	-	-	S/A	TIA	-	150	ST	-	I-TH	I	MCA	C,SC	R