COMPARITIVE STUDY ON CLINICAL PROFILE OF STROKE IN MALES AND FEMALES

Dissertation submitted to

THE TAMIL NADU DR. M.G.R MEDICAL UNIVERSITY

In partial fulfillment of the regulations
For the award of the degree of

M.D. DEGREE IN GENERAL MEDICINE
BRANCH – I

GOVT. STANLEY MEDICAL COLLEGE & HOSPITAL
THE TAMIL NADU DR. M.G.R. MEDICAL UNIVERSITY
CHENNAI, INDIA.

MARCH 2011

CERTIFICATE

This is to certify that this is a bonafide work titled **"COMPARITIVE STUDY ON CLINICAL PROFILE OF STROKE IN MALES AND FEMALES"**, is the original dissertation work done by **Dr .K .VIDHYA** in the Department of General Medicine, Government Stanley Hospital, Stanley Medical College, Chennai – 600 001, in partial fulfillment of the requirements for MD (General Medicine) Branch I Examination, of the Tamil Nadu Dr. M.G.R Medical University, Chennai to be held in March 2009**.**

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DECLARATION

I **Dr. K. VIDHYA** solemnly declare that the dissertation titled **"COMPARITIVE STUDY ON CLINICAL PROFILE OF STROKE IN MALES AND FEMALES"** was done by me during 2009-2010 under the guidance and supervision of my unit chief **PROF. G. SUNDARAMURTHY., M.D.,**

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ACKNOWLEDGEMENTS

I wish to express my gratitude to **Dr. C. VAMSADHARA M.D., Ph.D.,** Dean, Govt. Stanley medical college and hospital for allowing me to avail the facilities needed for the study.

With the deep sense of indebtedness and reverence, I thank **Prof. S. MAGESH KUMAR M.D.,** Professor and Head of the department of Internal Medicine for permitting me to do the study and for his encouragement.

I am extremely thankful to my Unit Chief **Prof. G. SUNDARAMURTHY M.D.,** for his valuable assistance and guidance.

I am also thankful to **Prof. S. RAMASAMY M.D.,** Professor of medicine for having allowed me to embark on this dissertation.

I would like to express my sincere gratitude to my Assistant Professors **Dr. S. ASHOK KUMAR M.D.,** and **Dr. M. MANIMARAN D.M**., and to Medical Registrar **Dr. G. VASUMATHI M.D**., for their generous help and guidance.

Last but not the least, my sincere thanks to all the patients who co-operated for this study without whom this study could not have been undertaken and to all my colleagues who helped me and shared their knowledge about the study.

CONTENTS

INTRODUCTION

INTRODUCTION

Stroke is one of the most important non- communicable disease of public health importance. Stroke is defined as an abrupt onset of a focal neurological deficit that is attributable to a focal vascular cause. WHO (World Health Organization) defines the syndrome of stroke as "rapidly developing clinical signs of focal or global disturbance of cerebral function with symptoms lasting or leading to death, with no apparent cause other than vascular origin".

It includes

1. Sub arachnoid hemorrhage

It excludes

1. Transient ischemic attack
2. Subdural hematoma
3. Subdural hemorrhage
4. Infarction caused by infection or tumor

History of stroke dates to 400 B.C, when Hippocrates first described the medical aspects of stroke. Anatomy of brain was first brought to spotlight by Andreas Vesalius in the 16^{th} century. Attention on pathology, cause of disease, disease presentation evolved over 17^{th} and 18^{th} century.

The major outbreak in the investigation of stroke cases came in the mid 1960's with the introduction of computed tomography by Hounsfield. It allowed clear

distinction between brain ischemia and hemorrhage and allowed description of size and location of the lesion. Further advance came in the form of MRI brain which helped anatomical and physiological correlation.

Medical treatment changed dramatically during later portions of the twentieth century after some of the risk factors has been well enumerated. Usage of aspirin after widespread therapeutic trials having been proven beneficial was in vogue since 1970's. Treatment of acute stroke patients was again refined after the approval of thrombolytic therapy in late 90's. Stroke which was once thought to be irremediable disease now has gone to the extent of viewing it as an emergency "Time lost is brain lost."

Stroke represents the third leading cause of death after Coronary artery disease and Cancer in the west with overall prevalence of 800 per lakh population. There is a lack of reliable information on epidemiology of stroke in our country. But community surveys from different regions of India showed a Crude Prevalence Rate of stroke presumed to be of vascular origin in the range of 203 per lakh population.

Stroke is a result of series of insult to cerebral and cardiovascular systems. The insult comes in the form of risk factors which appreciably differs for different communities. Stroke prevention strategies now revolve around the management of these risk factors which forms the backbone of the management of stroke in current era. Socioeconomic factors, dietary habits, lifestyle behaviors, different patterns of risk factors and environmental conditions have shown varying incidence of stroke observed in different parts of the world. Present statistics are in major part contributed by the studies from the developed countries. Since various ethnic groups and communities exists in our country, we need more studies from our nation involving large population to find out the

corresponding incidence, prevalence, morbidity, mortality, risk factors and outcome. This will enable us to devise our own strategy in the primary prevention of stroke for our population.

Stroke is a leading cause of disability in adults of stroke survivors. Of the 100 stroke survivors 30 % requires assistance with activities of daily living, 20 % requires assistance for ambulation and 16 % requires institutional care. Hence both directly and indirectly there is an appreciable loss of national economy.

As the projected mortality and morbidity by WHO shows an increasing trend more attention should be made to find out the high risk population and focus on prevention strategies.

As with the ethnic and racial variations found in a number of studies, by initiating this study an attempt is made to compare the

1) age predilection
2) clinical presentation
3) type of lesion
4) risk factor profile
5) outcome

in male and female population selected from urban Tamilnadu.

This study would throw light on the differences in the clinical profile of stroke between the representative samples of male and female population and thereby to formulate newer stroke prevention strategies.

REVIEW OF LITERATURE

REVIEW OF LITERATURE

WHO defines stroke as a syndrome of "rapidly developing clinical signs of focal or global disturbance of cerebral function with symptoms lasting or leading to death, with no apparent cause other than vascular origin"[38].

The initial definition of transient ischemic attack (TIA) required that all neurologic signs and symptoms resolve within 24 hours regardless of whether there is imaging evidence of new permanent brain injury and stroke is said to have occurred if the neurologic signs and symptoms last for more than 24 h. However, a newly proposed definition classifies those with new brain infarction as ischemic strokes regardless of whether symptoms persist.[6, 26]

Definition of stroke is clinical, and laboratory studies including brain imaging are used to support the diagnosis.

EPIDEMIOLOGY:

MORBIDITY[3]:

According to WHO neurological disorders constitute 6.3 % of the global burden of disease, out of which 55% (3.46% of total global burden of disease) is caused by Cerebro vascular accident. Neurological disorders contribute to 92 million DALY's (Disability adjusted life year) in year 2005 which has been projected to increase to nearly 103 million by the year 2030 .Of these cerebro vascular accident contribute to more than 55 % of the burden in DALY's. In future this may have an enormous impact in the health care providing system of the country.

DALY for individual neurological disorder as % of neurological disorder[41]

Cerebrovascular disease	55%
Alzheimer and other dementias	11.9%
Migraine	8.3%
Epilepsy	7.9%
Tetanus	7%
Meningitis	5.8%
Parkinson's disease	1.8%
Multiple sclerosis	1.6%
Japanese encephalitis	0.6%
Poliomyelitis	0.1%

Number of DALYs for cerebro vascular accident and as percentage of global DALYs projected for 2005, 2015 and 2030[41]

YEAR	No. of DALY's lost (in 1000's)	% of total DALY
2005	50,785	3.46 %
2015	53,815	3.63 %
2030	60,864	3.99 %

In the south East Asian countries DALY lost due to cerebro vascular accident has been 1.93 %[41]. The burden has been on the high in developing countries reflecting a double burden due to coexistence of communicable and non – communicable diseases.

Stroke is among the top five important causes of disability in both developing and developed countries. Increase in life expectancy can ultimately increase the burden in health care in forthcoming years in countries like ours. Number of DALY lost for our Indian scenario was about 5.8 millions in the year 1998 and has increased to the extent of 6.4 millions in the year 2004.

MORTALITY[41]:

Cerebro vascular accident is the second worldwide leading cause of mortality as per WHO statistics[6]. Neurological diseases constitute 12 % of total mortality worldwide, out of which cerebro vascular accident is responsible for 85 % (9.9 % of total mortality).

The mortality rate for Cerebro vascular accident in developing countries accounts to around 15 %.The mortality rate due to Cerebro vascular accident in India is 0.6/1000 population.(1.2 % of total deaths in India)

Deaths attributable to Cerebro vascular accident as percentage of total deaths 2005, 2015 and 2030[41]

YEAR	MORTALITY RATE
2005	9.9 %
2015	10.19 %
2030	10.63 %

Mortality for individual neurological disorder as % of total neurological disorder[41]

Cerebrovascular disease	85%
Alzheimer and other dementias	6.28%
Tetanus	2.83%
Meningitis	2.24%
Epilepsy	1.86%
Parkinson's disease	1.55%
Multiple sclerosis	0.24%

INCIDENCE AND PREVALENCE:

According to WHO the age-standardized incidence rates per 100,000 varied from 101 to 285 in men and from 47 to 198 in women[71]. Wide range of variability is due to the ethnic and racial variation influencing the stroke epidemiology. The age-standardized prevalence rates per 100,000 were between 250 – 350[18].

There is a significant variation among different communities and regional groups eg. there is a higher incidence of stroke among African Americans in U.S , as also the presence of stroke belt in southeastern U.S. In India, the prevalence of stroke has been estimated at 203 per 100,000 population older than 20 (Anand and others 2001)[42]. Stroke incidence is heterogenous in population and changes over time. In our country many studies are needed to be conducted in various regions to identify the stroke patterns.

The prevalence rates of stroke from various major epidemiological studies in India[18]

Zone	Place	Rural/ Urban	Year	Population	Crude Prevalence Rate per 100000	Age adjusted prevalence Rate per 100000
North	Haryana	Urban	1971-1974	79,046	44	
	Kashmir	Rural	1986	63,645	143	244
West	Mumbai	Urban	1985	14,010	842	424
	Mumbai	Urban	1997	145,456	220	
East	West Bengal	Rural	1989-1990	37,286	126	
	West Bengal	Rural	1992-1993	20,842	147	
	Kolkata	Urban	1989-1999	50,291	147	334
South	Vellore	Rural	1969-1971	258,576	57	84
	Karnataka	Rural	1982-1984	57,660	52	
	Bangalore	Rural	1993-1995	51,055	165	262
	Bangalore	Urban	1993-1995	51,502	136	

The Annual incidence rates of stroke from various epidemiological studies in India[18]

Place	Rural/ Urban	Year	Population	Crude Incidence Rate per 100000	Age adjusted incidence Rate per 100000
Vellore	Rural	1969-1971	258,576	13	
Kolkata	Urban	1998-1999	50291	36	105
West Bengal	Rural	1993-1998	20842	124	262

CLASSIFICATION:

ISCHEMIC STROKE:

Acute occlusion of intracranial vessel causing reduction in blood flow to the brain region it supplies. It accounts to 80 % of all strokes.

MAIN CAUSES[1, 4, 14] include

1. ATHEROTHROMBOSIS

   MOST COMMON SITE OF OCCURANCE[14, 4]: Major arterial branches (e.g., the carotid bifurcation in the neck or intracranial branch points) and at vessel origins (e.g., the origin of the vertebral artery from the subclavian artery).

2. EMBOLIC OCCLUSION

   POTENTIAL SOURCES OF CARDIOEMBOLISM[4]: Atrial fibrillation, Mural thrombus, Ventricular akinesis after myocardial infarction, Dilated cardiomyopathy, and Valvular disease.

3. HYPERCOAGULABLE STATES

   MOST COMMON CAUSES[1]: Antiphospholipid antibody syndrome, Protein C & S deficiency, Hyperhomocysteinemia and other coagulopathies.

4. VENOUS SINUS THROMBOSIS

   MOST COMMON CAUSES[44]: Infection is the most common cause in children but in adults, pregnancy, severe dehydration, sickle cell anemia, malignancy, and hypercoagulable states contribute majority of cases.

5. VASCULITIS

   MOST COMMON CAUSES[1]:

   a) Meningovascular syphilis, Arteritis secondary to pyogenic and tuberculous meningitis.
   b) Connective tissue diseases like polyarteritis nodosa, lupus erythematosus, etc.,

HEMORRHAGIC STROKE:

It accounts for 20 % of all strokes and is associated with a mortality rate of around 50 %. It is of two types

1. Intracerebral hemorrhage accounting for 15 %
2. Subarachnoid hemorrhage accounting for 5 %

MOST COMMON CAUSES include

1. HYPERTENSION

MOST COMMON SITES[4, 14]: Lateral ganglionic and capsular, Thalamus, Lobar white matter, Caudate, Pons and Cerebellum.

2. NON HYPERTENSIVE CAUSES

a. Vascular malformations (saccular or mycotic aneurysms, arteriovenous malformations, cavernous angiomas)

MOST COMMON SITES OF ANEURYSM[14]: Junctions of larger vessels of the circle of Willis commonly at internal carotid artery with posterior communicating artery junction, anterior cerebral artery with anterior communicating artery junction and middle cerebral artery trifurcation.

b. Intracranial tumors

c. Bleeding disorders

d. Anticoagulant and fibrinolytic treatment

e. Cerebral amyloid angiopathy

f. Hemorrhagic infarction

g. Trauma

PATHOPHYSIOLOGY[26, 44]:

Cerebral blood flow distributes blood to target organ by regulating blood flow and distributing oxygen and glucose to the brain while removing the byproducts of metabolism.

Reduced blood flow to brain causes cerebral ischemia and energy failure is rapid as neurons lack glycogen. Ischemia causes impairment of brain energy metabolism, loss of aerobic glycolysis, intracellular accumulation of sodium and calcium ions, release of excitotoxic neurotransmitters, elevation of lactate levels with local acidosis, free radical production, cell swelling, over activation of lipases and proteases, and cell death.

Normal cerebral blood flow at rest in the normal adult brain is approximately 50-55 ml /100 g per minute and the cerebral metabolic rate of oxygen is 165 mmol /100 g per minute.

When blood flow decreases to 18 ml /100 g per minute, the brain reaches a threshold for electrical failure at which state if blood flow recovers brain tissue also recovers.

When blood flow falls to less than 8 ml /100 g per minute, the neurons reaches the threshold of membrane failure and cell death result and thereby causing permanent neurological sequelae.

The ischemic penumbra, or area of misery perfusion, is the area of the ischemic brain between these two flow thresholds were some neurons are functionally silent but structurally intact and potentially salvageable.

Hemorrhage is caused by bleeding into or around the brain. It produces neurological symptoms by producing a mass effect on neural structures or by the direct toxic effect of blood itself or by increasing intracranial pressure.

CLINICAL CLASSIFICATION OF STROKE:

Oxfordshire / Bamford classification of stroke [8]

The Classification is based on clinical features of the presenting stroke. It allows us to reliably comment on stroke territory, allows easy identification of syndromes that do not fit classically, and also gives clinical prognostication. However they can be further refined by the investigatory modalities like CT or MRI brain.

Total Anterior Circulation Syndrome (TACS)

- Constitutes 20% of strokes.
- Diagnosis suspected by the presence of combination of three of the following manifestations
    - Motor weakness (with or without sensory deficit in the form of hemisensory loss) of at least 2 of 3 body areas (face/arm/leg).
    - Homonymous hemianopia.
    - Higher cerebral dysfunction (dysphasia in dominant hemisphere involvement: dyspraxia in non dominant hemisphere involvement).
- If drowsy with unilateral weakness, other two factors can be assumed to be present.

Partial Anterior Circulation Syndrome (PACS)

- Constitutes 35% of strokes.

- Diagnosis suspected with the presence of
    - Two of the three of the TACS criteria or
    - Restricted motor/sensory deficit eg. One limb, face and hand
- Usually not associated with drowsiness
- Recurrence is high, especially in the first three months.

Lacunar Syndrome (LACS)

- Constitutes 20% of strokes.
- Presentation may include the occurrence of any one of the following
    - Pure motor (commonest)- Complete or incomplete weakness of 1 side
    - Pure sensory -Sensory symptoms and/or signs
    - Combination of motor and sensory
    - Dysarthria or dysphasia
    - Ataxic hemiparesis - Hemiparesis and ipsilateral cerebellar ataxia.
- Often CT- MRI will delineate the infarct. It is an intrinsic disease of single basal perforating artery (usually end arteries).
- Often silent and under diagnosed.

Posterior Circulation Syndrome (POCS)

- Constitutes 25% of strokes.
- Affecting brainstem, cerebellar or occipital lobes.
- Thrombosis is more pronounced than embolism.
- Presentation is variable and frequently complex. It includes

- Bilateral motor/sensory deficit
- Disordered conjugate eye movement
- Isolated homonymous hemianopia
- Ipsilateral cranial nerve palsy with contra lateral motor and or sensory deficit
- Coma, disordered breathing, and horner's syndrome can be associated.

BAMFORD CLASSIFICATION OF STROKES [8]

Syndromes	TACS	PACS	LACS	POCS
Site Of Lesion	Fronto -tempo-Parietal lobes Thalamus/internal capsule/occipital	Lobar	Small deep lesion in corona radiata, internal capsule, thalamus or ventral pons	Brain stem/ cerebellum/occipital lobe
Pathology Infarction	85%	85%	95-98%	85%
Hemorrhage	15%	15%	2-5%	15%
Etiology	Occlusion of Ipsilateral ICA or MCA and occasionally PCA; by embolism from heart, aortic arch or carotid or vertebrobasillar arteries, or in situ thrombosis.	Occlusion of branch of MCA or PCA; by embolism from heart, aortic arch.	Usually lipohyalinosis, microatheroma or 'complex disease' (fibrinoid necrosis) of small perforating artery. Rarely arteritis or embolism.	Occlusion of VBA or PCA , or branches ; by insitu thrombosis or embolism from heart, aortic arch or VBA.
Hemorrhage	Any of possible cause	Any of possible cause	Any of possible cause	Any of possible cause
Recurrence Rate	low	High in first 3 months	Low but steady over 12 months	High in first 2 months and steady over 12 months

(Adapted from Hankes and Lees, 2001)

RISK FACTORS OF STROKE:

Various epidemiological studies have indicated that stroke does not occur at random. It has been found that several factors precede the disease which contributes to the occurrence of stroke. These are called RISK FACTORS[46].

The consequences of this preventable epidemic will have an impact on individual mortality and morbidity, family suffering, and staggering economic costs. The intervention in these risk factors can be widely applied because of their low cost and minimal risk[46]. Hence evaluation of the clinical risk profile can help us to decide on the preventive treatment which is to be offered.

The significance of a risk factor is determined by the relative risk which represents the strength of the association between the risk factor and the chance of having a stroke. Population attributable risk is of more relevance in considering the importance of a risk factor in a particular population.

Identification of the clinical patterns and the risk factors and to control or modify the risk factors remain the most important means of reducing the stroke incidence in western nations[58] which has been brought about by several population based studies. Hence more number of studies need to be done in our country to find the risk factor profile of our nation.

The most important means of reducing stroke incidence and there by stroke disability is by modifying the risk factors to the extent possible. Hence complete knowledge about the risk factors in the prevailing area is absolutely essential.

Risk factors have been classified into non modifiable and modifiable.

NON MODIFIABLE RISK FACTORS[51]:

- Age
- Gender
- Race/Ethnicity
- Family history
- Genetics

As these factors are non modifiable they don't contribute much for the framing of prevention strategy measures. Hence more importance has been insisted on the prevention strategy based on the modifiable risk factors.

MODIFIABLE RISK FACTORS[51]:

- Arterial hypertension
- Asymptomatic carotid bruit
- Cardiac disease
- Diabetes mellitus
- Dyslipidemia
- Cigarette smoking
- Alcohol consumption
- Elevated homocysteine
- Low serum folate
- Elevated anticardiolipin antibodies
- Oral contraceptive use
- Obesity
- Prior stroke

- Aortic arch atheromatosis
- Increased fibrinogen levels

These risk factors are of prime importance as they could be used for the framing of prevention strategies.

NON MODIFIABLE RISK FACTORS:

Spotlights people at increased risk of stroke. These are the factors towards which the prevention strategies could not be targeted.

AGE:

Stroke incidence increases dramatically with increasing age. Increasing age is the most powerful risk factor for stroke[64]. The incidence of stroke doubles each decade past 55 years of age. Half of all strokes occur in people older than 70 to 75 years. Men develop ischemic strokes at higher rates than women up to the age of 75 years.

The mean age of stroke presentation is 57 to 71 yrs is relatively lesser in Asian population than in than west of 76 to 80 yrs[58, 34, 32]. Atherosclerosis and cardio embolism secondary to atrial fibrillation are the leading cause of ischemic stroke in elderly while cerebral amyloid angiopathy and chronic hypertension are the leading causes of intracerebral hemorrhage in elderly[54].

FAMILY HISTORY:

An increased risk is seen with a family history of stroke among first-degree relatives. There are a number of genetic causes of stroke. Some inherited diseases like hereditary dyslipoproteinemias which predispose to accelerated atherosclerosis,

coagulopathies, and tendencies for intracranial aneurysms and AV malformations increases the chance of getting a stroke.

Also presence of the apolipoprotein K2 allele in elderly individuals and the deletion of the gene for the angiotensin converting enzyme may increase the risk of stroke. But it is somewhat controversial.

Other Potential reasons for stroke to run in families are genetic determination of stroke risk factors, and a common familial exposure to environmental or lifestyle risks. Earlier studies suggested an increased risk for men whose mothers died of stroke and women who had a family history of stroke. In the Framingham Study offspring analyses revealed that both paternal and maternal histories[45] were associated with an increased risk of stroke[78].

GENDER:

Men develop ischemic strokes at higher rates than women up to the age of 75 years. The male-to-female ratio was one to seven[71]. Stroke incidence rates are 1.25 times greater in men, but because women tend to live longer than men, more women than men die of stroke each year. Mean age of females is more than that of males[58].

Stroke is uncommon among women of childbearing age. The relative risk of stroke is increased among users of high-dose estrogen oral contraceptives, particularly with coexistent arterial hypertension, cigarette smoking, increasing age, migraine, and saccular aneurysm[3].

Risk of thrombosis associated with pregnancy is high in the postpartum period. The risk of cerebral infarction is increased in the 6 weeks after delivery but not during pregnancy.

Compared with males, female patients were significantly showed higher level of handicap, as defined by prestroke Rankin Scale, and a higher frequency of hypertension, atrial fibrillation, and antihypertensive treatment. History of myocardial infarction, current or previous smoking, alcohol consumption, and antiplatelet therapy were significantly more frequent in male patients[5]

Two recent postmenopausal hormone replacement studies showed no benefit in reducing the incidence of stroke in a cohort of women with coronary heart disease (Hulley et al. 1998; Herrington et al. 2000)[28].

Women's Health Initiative, a prospective randomized trial of estrogen therapy in healthy postmenopausal women, was halted prematurely because the risks outweighed the benefits. Absolute excess risks per 10,000 person-years attributable to estrogen plus progestin were Eightfold for strokes[55] (Writing Group for the Women's Health Initiative Investigators. 2002)

RACE/ ETHNICITY:

Clinical profile and outcome has been studied in various populations and have shown significant variations among different races.

The rate of cerebral infarction is higher in blacks than in whites; this could be partially explained by the higher prevalence of diabetes and arterial hypertension experienced by blacks. Blacks also have higher rates of intracranial atherosclerotic occlusive disease, compared with whites[58].

Also most of the local southasian and far eastern studies have suggested that the proportion of intracerebral hemorrhage was significantly higher 20 – 45 % than in the west 10 – 20 % : while cerebral infarction varied between 55 – 70 % in the local studies and 60- 85 % in the western studies.[9, 58, 59]

The stroke incidence and case fatality rates are also markedly different among the major ethnic groups in Auckland and New Zealand. Maori and Pacific Islands people have a higher mortality within 28 days of stroke when compared with Europeans, especially men[10].

MODIFIABLE RISK FACTORS:

Risk factor which can be controlled or modified thereby decreasing the incidence of stroke is the prime focus of management in this era. From a practical viewpoint, these risk factors that have emerged from various studies are modifiable by lifestyle and/or pharmacotherapy.

The major risk factors are discussed.

HYPERTENSION:

Hypertension is the most important modifiable determinant of both first and recurrent stroke (Eastern Stroke and Coronary Heart Disease Collaborative Research Group 1998)[42]. Arterial hypertension predisposes to ischemic stroke by aggravating atherosclerosis and accelerating heart disease, increasing the relative risk of stroke three to fourfold.

Treatment of systolic hypertension slows the progression of atherosclerosis. Decreasing systolic blood pressure by approximately 10 mmHg reduces the relative risk of stroke by 35% to 40%.The PROGRESS Trial evaluated the effects of antihypertensive on the risk of stroke in patients with histories of stroke or transient ischemic attack (TIA). Regardless of blood pressure at entry, patients clearly benefited from treatment (PROGRESS Collaborative Group 2001)[52].

Lowering blood pressure to levels below those traditionally defining hypertension appears to reduce the risk of stroke even further. The risk is greater for patients with isolated systolic hypertension and elevated pulse pressure and hence treatment of isolated systolic hypertension in the elderly has proven to be effective in reducing stroke risk.

Blood pressure treatment, resulting in a reduction in systolic of 10-12 mm Hg and 5-6 mm Hg diastolic, is associated with a 38% reduction in stroke incidence. Lowering blood pressure in stroke survivors helps prevent recurrent stroke and is more important than the specific hypotensive agent used.

Data are particularly strong in support of thiazide diuretics, angiotensin-converting enzyme inhibitors, and angiotensin receptor blockers. However, the effects of blood pressure lowering agents may vary in the different age groups[53]

Hypertension is the most readily recognized factor in the genesis of primary intracerebral hemorrhage.

The cooperative studies of the Veterans Administration (Freis et al) and the report by Collins and associates (collating 14 randomized trials of antihypertensive drugs) have convincingly demonstrated that the long-term control of hypertension decreases the incidence of both atherothrombotic infarction and intracerebral hemorrhage.

DIABETES MELLITUS:

Diabetes mellitus hastens the atherosclerotic process in both large and small arteries. Weinberger and colleagues and Roehmholdt and coworkers have found diabetic patients to be twice as liable to stroke as age matched nondiabetic groups[76]. The diabetes is an independent risk factor among the associated risk factors as found in a number of large prospective studies, including Gothenburg cohort study, and the Framingham study.

The relative risk of stroke in diabetes is higher in women, and may be as high as 13 times the normal risk in the younger age groups. High insulin levels increase the risk for atherosclerosis and may represent a pathogenetic factor in cerebral small vessel disease.

There is a fourfold increase in the relative risk of cardiovascular event among patients with diabetes and hypertension than among those without the two conditions (HT and DM study…HDS 1993)[29]. There in more than an additive risk when both conditions co exist.

The frequency of diabetes mellitus is found to be higher in Indian population 18% -42.5% when compared to the western population 10%-26%, adding to the risk of disease burden[73]. The resulting impairment tends to be greater and survival poorer in diabetics than in nondiabetic stroke patients.

Diabetic persons with retinopathy and autonomic neuropathy appear to be a group at particularly high risk for ischemic stroke. However, presently no evidence exists that tighter diabetic control over time decrease the risk of stroke or stroke recurrence.

DYSLIPIDEMIA:

High Total Cholesterol and High Low-Density Lipoprotein concentration have well correlated with atherosclerosis of cerebral vasculature. Low total cholesterol has been associated with hemorrhagic stroke[81, 62]. Carotid atheroma is linked to increased LDL and may be inversely related to HDL .But HDL association is less clear in relation to CVA. Lipid-lowering agents may slow progression of atherosclerotic plaque growth and may possibly cause a regression in plaque formation[12].

In Scandinavian Simvastatin Survival Study, the post hoc analysis have showed a 28% reduction in fatal or nonfatal stroke and TIAs[60,39,47](Pedersen et al. 1998).

The Long-Term Intervention with Pravastatin in Ischemic Disease (LIPID)[50] reported 20% reduction in the risk of stroke.

Several trials have confirmed that statin drugs reduce the risk of stroke even in patients without elevated LDL or low HDL. The recently reported SPARCL (Stroke Prevention by Aggressive Reduction in Cholesterol Levels) trial showed benefit in secondary stroke reduction for patients with recent stroke or TIA who were prescribed atorvastatin, 80 mg/d. Although studies specifically targeting primary prevention of stroke are still underway, results for patients with cardiovascular risk factors or dyslipidemia have been compelling, with a 16–30%relative risk reduction for stroke.

CIGARETTE SMOKING AND TOBACCO CHEWING:

Cigarette smoking is a major risk factor for stroke[78, 70]. It is a very important preventable cause of stroke. Cigarette smoking is an independent risk factor[25] for ischemic stroke in men and women of all ages, and a leading risk factor of carotid atherosclerosis in men[30].

The risk of stroke in smokers is two to three times greater than in nonsmokers. Heavy smoking (more than one pack per day) carries eleven times the ischemic stroke risk and four times the subarachnoid hemorrhage risk of people who do not smoke. The mechanisms of enhanced atherogenesis promoted by cigarette smoking are incompletely understood, but include reduced capacity of the blood to deliver oxygen, cardiac arrhythmias, and triggering of arterial thrombosis and arterial spasm. Some of the studies have shown betel nut chewing with or without tobacco is associated with increased risk of stroke[7].

Smoking with oral contraceptives carries 22 times the risk of stroke without them in females. More than 5 years may be required before a reduction in stroke risk is observed after cessation of smoking[51, 75].

ALCOHOL CONSUMPTION:

A J-shaped association between alcohol consumption and ischemic stroke exist. Light to moderate use (up to two drinks a day) evenly distributed throughout the week offers a reduced risk and may elevate HDL concentration[58, 57, 72], whereas heavy drinking is associated with an increased risk of total stroke. . Heavy drinking has a different cut off for a male and a female. Heavy drinking is considered when a male take more than 35 – 45 gm per day and that when a females take more than 22 – 30 gm per day. This association is found predominantly in white population. In a study conducted in a cohort in population of Sweden it has been demonstrated that ischemic stroke was found in men (not females) who were intoxicated or had reported episode of binge drinking a few times a year

Alcohol may induce arrhythmias and cause Cardiomyopathy with global hypokinesis, thereby predisposing to formation of cardiac emboli. Alcohol has also been linked to hypertension, increased platelet aggregation, and decreased production of circulating clotting factors due to insult to liver and hence contributing to stroke.

The association between alcohol consumption and stroke risk appears much stronger for intracerebral and subarachnoid hemorrhage than for ischemic stroke[72]. In western studies it has shown the risk of hemorrhagic stroke more than doubled for light drinkers and nearly tripled for those considered to be heavy drinkers. Positive linear relationship has been demonstrated between moderate alcohol consumption and risk of intracerebral hemorrhage. A reduction in alcohol consumption was accompanied by a

reduction in subsequent hemorrhagic stroke suggesting a possible causal relation between the two.

VENOUS THROMBOSIS:

Cerebral vein thrombosis (CVT) is a rare but important cause of stroke that is often missed or discovered late in diagnosis[12]. Infection is the most common cause of CVT in children whereas in adults, most are associated with postpartum[42]. Infrequent etiologies include severe dehydration, sickle cell anemia, malignancy, hypercoagulable states and usage of oral contraceptive agents.

Severe headache, nausea, and vomiting are nonspecific but common symptoms. Papilledema may be the only abnormality on initial examination. Fluctuating focal neurologic deficits, such as unilateral weakness, numbness, or seizures, may appear.

Noncontrast CT will determine whether acute hemorrhage or mass effect is present but Contrast CT may demonstrate the empty delta sign in the sagittal sinus. MRI and MR venogram have better sensitivity for detection of CVT. Gold standard diagnostic tool is conventional cerebral angiography. A small randomized trial demonstrated safety and efficacy of intravenous heparin in CVT, even in patients with preexisting hemorrhage. Consequently, acute anticoagulation is recommended for most patients with CVT. After the acute period, oral anticoagulation is typically used for several months until MRI or MRV demonstrates sinus patency.

Mortality from CVT is estimated to be 5% to 30%, but survivors have a good prognosis with little residual deficit[44].

PHYSICAL ACTIVITY:

Regular exercise lowers blood pressure, decreases insulin resistance, increases HDL Cholesterol, and is associated with lower cardiovascular morbidity and mortality. Medium to high levels of physical exercise has shown to decrease the incidence of stroke in both men[49] and women[48]. Habitual snoring increases the risk of stroke. It adversely affects outcome of patients admitted to the hospital with stroke.

OBESITY:

The prevalence of obesity has increased particularly in the western countries. More than 61% of adult Americans are overweight, and 27% are obese.

Obesity, particularly abdominal or truncal is an important risk factor for cardiovascular disease in men and women of all ages.

ATRIAL FIBRILLATION:

Cardio embolism is associated with 20 % of all ischemic strokes[79]. The most significant causes of cardio embolic stroke in most of the world are nonrheumatic (often called nonvalvular) atrial fibrillation, acute myocardial infarction, prosthetic valves, rheumatic heart disease, and ischemic Cardiomyopathy. Atrial fibrillation is associated with an overall risk of stroke of approximately five to six folds, and a mortality of approximately twice that of age and sex matched individuals without atrial fibrillation.

Nonrheumatic atrial fibrillation is the most common cause of cerebral embolism overall. Prevalence of non valvular atrial fibrillation (NVAF) increases with advancing age. The number of patients with atrial fibrillation is likely to increase 2.5-fold during the next 50 years in the west, reflecting the growing proportion of elderly individuals. Coordinated efforts are needed to face the increasing challenge of optimal stroke

prevention and rhythm management in patients with atrial fibrillation. *(*JAMA. 2001; 285: 2370-2375).

Heart failure, arterial hypertension, diabetes, prior stroke or TIA, prior cardio embolism, poor left ventricular function, mitral stenosis, prosthetic heart valve, and increasing age increase the risk of embolism in patients with atrial fibrillation. High-risk patients have a 5-7% yearly risk of thromboembolism.

CAROTID ARTERY DISEASE:

Asymptomatic carotid disease carries a greater risk of vascular death from coronary artery disease than from stroke. Persons with an asymptomatic carotid bruit have an estimated annual risk of stroke of 1.5% at 1 year and 7.5% at 5 years. Asymptomatic carotid artery stenosis less than 75% carries a stroke risk of 1.3% annually; with stenosis greater than 75%, the combined TIA and stroke rate is 10.5% per year, with most events occurring ipsilateral to the stenosed carotid artery[19]. Symptomatic carotid artery stenosis greater than 70% carries an annual risk of stroke of approximately 15%.

Plaque structure rather than degree of carotid artery stenosis may be a critical factor in determining stroke risk. Ultrasonographic carotid artery plaque morphology may identify the subgroup of patients at high risk of stroke.

PRIOR STROKE:

Patients with a first stroke are at greater risk of recurrent stroke, especially, early after the first stroke. Those who suffer a recurrent stroke have a higher mortality than patients with first stroke. If the recurrence is contralateral to the first stroke, prognosis for functional recovery is poor.

ELEVATED FIBRINOGEN LEVELS:

Elevation of plasma fibrinogen is an independent risk factor for the development of stroke. Also fibrinogen levels are closely correlated with other stroke risk factors such as cigarette smoking, arterial hypertension, diabetes, obesity, hematocrit levels, and spontaneous echocardiographic contrast.

HOMOCYSTEINE ELEVATION:

Fasting total homocysteine (normal 5-15 mmol/L), a sulfhydryl-containing amino acid, have been associated with an increased risk of stroke[58] and thrombotic events in case-controlled studies. It predisposes young individuals to vascular events due to its procoagulant state and is an independent risk factor for stroke. Hyperhomocysteinemia can be caused by an error in metabolism of sulphur containing amino acids. Other causes of homocysteine elevation are nutritional deficiencies of vitamin B6, B12, folic acid and genetic defect in the methylene tetrahydro folate reductase gene. Serum folate concentrations less than or equal to 9.2 mmol/liter have been associated with elevated plasma levels of homocysteine, and a decreased folate concentration alone may be a risk factor for ischemic stroke, particularly among blacks (Giles et al. 1995).

Despite of clear evidence from Framingham study and Rotterdam study that the elevated plasma levels of homocysteine are associated with a higher risk of vascular events independently, many prospective treatment trials provide no evidence that folic acid and vitamin B therapy can reduce the subsequent vascular events in patients with elevated homocysteine even though they can decrease the plasma levels of homocysteine further more the combination of folic acid and vitamin B therapy may actually be harmful in some patients group.

ANTI PHOSPHOLIPID ANTIBODY SYNDROME:

The presence of anticardiolipin antibodies in association with thrombotic events without evidence of connective tissue disease is referred to as primary antiphopholipid syndrome. Lupus anticoagulant and anticardiolipin antibodies are recognized markers for increased risk of thrombosis, abortion, cerebral ischemia and multiinfarct state.

Various studies showed that among patients with antiphospholipid antibody[23], around 30% had neurological manifestations consistent with cerebral infarct, better correlating with IgG than IgM antibody. Patients with higher levels of antibodies are likely to have multiple cerebral infarcts than those with lower levels.

PREVENTION OF STROKE[46]

The accumulated evidence from various studies lead to broad consensus of expert opinion that stroke is preventable. This is best expressed in a report of WHO expert committee as following.

- PRIMARY PREVENTION
    1. Population strategy
        A. Prevention in whole population
        B. Primordial prevention in whole population
    2. High risk strategy
- SECONDARY PREVENTION

PRIMARY PREVENTION[51]:

It is the action taken prior to the onset of disease, which removes the possibility that a disease will occur. It can be implemented by means of targeting at the whole population irrespective of the individual risk level or by targeting the high risk populations[58]. Attempting to provide stroke prevention therapy to general population is not cost effective whereas identifying and giving therapy to people with risk factors for stroke i.e., high risk population is cost effective and lead to significant reduction in morbidity and mortality from stroke.

The population strategies centre around good control of hypertension, diabetes, and dyslipidemia by pharmacotherapy and behavioral modifications like abstaining from smoking and alcoholism. Emphasis should also be made on dietary modifications and encouraging physical activities.

This prevention strategy involves the use of mass media and public education systems[58]. So by targeting high risk population a heavy economic benefit is obtained by reducing the DALY which would have been lost otherwise.

The primordial prevention is a novel approach to primary prevention of stroke. It involves the prevention of emergence and spread of risk factors and life styles that have not been observed in population.

SECONDARY PREVENTION:

The aim of secondary prevention is to prevent the recurrence and progression of the stroke. It is a continuation of primary prevention in a post stroke patient. The principles governing secondary prevention are the same as those of primary prevention. Early diagnosis and management of stroke improves the outcome in the diseased individual.

Complementary strategies[46] can be used to lessen the impact of the disease.

- Risk factors can be lowered through population-wide public health measures, such as national campaigns.
- Identification of higher-risk subgroups of the population who stand to benefit the most from specific, low-cost prevention interventions.
- Resources should be allocated to acute as well as secondary prevention interventions. For countries with limited resources, a critical first step in developing a comprehensive plan is better assessment of cause-specific mortality and morbidity, as well as the prevalence of the major preventable risk factors.

PREVENTING STROKE RECURRENCE BY MEDICAL THERAPY

Evidence from several clinical studies favors the use of platelet antiaggregants as the first line of therapy in patients at high risk for stroke (Antithrombotic Trialists' Collaboration 2002). These agents are indicated for secondary prevention of stroke. Meta-analyses have shown that aspirin reduces the combined risk of stroke, myocardial infarction, and vascular death by approximately 25%[59]. The optimal dose of aspirin remains a source of controversy. Two large trials, the International Stroke Trial (IST) and the Chinese Acute Stroke Trial (CAST), found that the use of aspirin within 48 h of stroke onset reduced both stroke recurrence risk and mortality minimally[15].

Ticlopidine reduces the relative risk of death or nonfatal stroke by 12% in comparison with aspirin but has the disadvantage of causing diarrhea, skin rash, neutropenia and thrombotic thrombocytopenic purpura. The Clopidogrel versus Aspirin

in Patients at Risk of Ischemic Events study (CAPRIE) assessed the relative efficacy of clopidogrel (75 mg daily) and aspirin (325 mg daily) in reducing the incidence of ischemic stroke, myocardial infarction, or symptomatic atherosclerotic peripheral arterial disease[60]. The results of this study showed that clopidogrel was modestly more effective (8.7% relative risk reduction) than aspirin in reducing the combined risk of ischemic stroke, myocardial infarction, and vascular death in patients with atherosclerotic disease. But aspirin is inexpensive, can be given in low doses, and could be recommended for all adults to prevent both stroke and MI. Management of Atherothrombosis with Clopidogrel in High-Risk Patients (MATCH) trial compared clopidogrel in combination with aspirin to clopidogrel alone in the secondary prevention of TIA or stroke. The MATCH trial found no difference in TIA or stroke prevention with this combination, but did show a small but significant increase in major bleeding complications (3% vs. 1%) [17].

To date there is no evidence to support the use of aspirin in primary prevention of stroke among low-risk, middle-aged people.

Oral anticoagulation with warfarin is indicated for primary and secondary prevention of stroke in patients with NVAF [20, 37, 42]. Advancing age increases the risk of major hemorrhage in patients given warfarin for stroke prevention. NVAF Patients at high risk of stroke should be treated with dose adjusted Warfarin[11] (INR 2.0-3.0).

Hypertension is the leading cause of primary intracerebral hemorrhage. Prevention is aimed at reducing hypertension, excessive alcohol use, and use of illicit drugs such as cocaine and amphetamines.

INVESTIGATIONS

CT scan forms the modality of choice for diagnosis of hemorrhage. It localizes small hemorrhage, hemorrhagic infarcts and subarachnoid hemorrhage. Infarcts could be localized by it only after 24 to 48 hrs. CT angiogram detects carotid disease and intravascular occlusions.

Diffusion weighed MRI is the earliest to detect acute infarct. It appreciates infarct even within few minutes. It detects small lacunar lesions deep in the hemisphere and also shows abnormalities in the brainstem. MR angiogram effectively detects stenosis of extracranial ICA and large intracranial vessels. MRI with fat saturation will visualize extracranial or intracranial arterial dissection.

B mode ultrasound with Doppler detects and quantifies stenosis at the origin of ICA. Transcranial Doppler assesses MCA, ACA, PCA flows.

Digital arteriography shows occlusion of large vessels, aneurysm, vascular malformation and arteritis. Reserved for situations where less invasive measures are adequate.

Lumbar puncture with CSF being hemorrhagic implies bleed into intraventricular space. Very minimal bleed can be detected by positivity for CSF xanthochromia.

In stroke of unknown origin ECG and echocardiogram are mandatory. In suspected embolic stroke transesophageal echo should be advised. Tests regarding association of risk factors like ECG, blood sugar levels and serum cholesterol levels should be done. On grounds of suspicion of hypercoagulable state like hyperhomocysteinemia, antiphospholipid antibody syndrome, protein S and C deficiency, investigations should be directed towards it.

TREATMENT

Acute Ischemic Stroke treatments designed to reverse or lessen the amount of tissue infarction and improve clinical outcome are as follows.

(1) Medical Support: Aims to make the undamaged cells to be protected (ischemic penumbra) by optimizing cerebral perfusion in that area. Collateral blood flow in the ischemic brain is blood pressure dependent and hence it should not be reduced drastically, exceptions being malignant hyperthermia, association of myocardial infarction, and when BP is < 185/110 mm hg and thrombolytic therapy is anticipated. Blood glucose levels should be maintained to <110 mg/dl. 5 to 10 % will develop cerebral edema which peaks on the 2nd or 3rd day. IV mannitol is the drug of choice and steroids have no role. In cerebellar infarcts in view of impending brain stem compression surgical decompression is undertaken almost as soon as the edema becomes clinically apparent.

(2) Intravenous thrombolysis (National Institute of Neurological Disorders and Stroke rtPA Stroke Study Group 1995)[42]: Effective if treatment is done within 3 hrs of onset[2]. rtPA which converts plasminogen to plasmin which by itself is a proteolytic enzyme having the capacity of degrading fibrinogen to fibrin is the drug used at the dose of 0.9 mg/kg iv to a maximum of 90 mg. Out of this 10 % is administered as bolus and the rest as infusion over 60 minutes. Has 6 % risk of intracerebral bleed.

(3) Endovascular Techniques: Large clot volume usually fails to open up with iv rtPA alone. Intra arterial route increases the concentration of the drug at the site of clot and minimizes systemic complications to some extent.

In patients who failed to recanulate with rtPA or if patient is ineligible or having contraindication for thrombolysis, endovascular thrombectomy can be attempted. Patients showed improvement if done within 8 hrs of onset of symptoms.

(4) Antithrombotic Treatment: Use of aspirin within 48 hrs of onset reduced both stroke recurrence risk and mortality (Chinese Acute Stroke Trial Collaborative Group 1997; International Stroke Trial Collaborative Group 1997)[42]. Due to cost effectiveness, aspirin is preferred over clopidogrel. Trials do not support the use of anticoagulants for patient with thrombotic stroke. The U.S. Trial of Organon 10172 in Acute Stroke Treatment (TOAST), an investigational low-molecular-weight heparin, failed to show any benefit over aspirin. Use of SC unfractionated heparin versus aspirin was tested in international stroke trial (IST). Heparin given SC afforded no additional benefit over aspirin and increased bleeding rates. But there is a role for anticoagulants in ischemic strokes.

(5) Surgical measures: Carotid atherosclerosis can be managed by endarterectomy and endovascular stenting with or without balloon angioplasty.

(6) Neuroprotectives: Drugs that block excitatory amino acids (glutamic acid) and hypothermia are said to benefit but yet to be proven in humans.

(7) Stroke Centers and Rehabilitation: It includes physical therapy, occupational therapy and speech therapy. Measures targeted against prevention of pneumonia, UTI, DVT, and pulmonary embolism.

PROGNOSIS AND OUTCOME

Prognosis of the stroke depends on several factors such as age, etiology, size of the lesion and the time that lapse before initiation of treatment. The prognosis for survival after cerebral infarction is better than after cerebral or subarachnoid hemorrhage. The only proved effective therapy for acute stroke requires initiation within 3 hours after stroke onset, and the prognosis therefore depends on the time that elapses before arrival at the hospital.

Initial stroke severity is one of the strongest predictors of outcome, and early evidence of improvement is a good prognostic sign. Loss of consciousness after a cerebral infarct implies a poorer prognosis than otherwise. Recovery also depends on the size and location of the infarction or hemorrhage. Small infarctions, particularly subcortical lacunar strokes, may result in little chronic deficit, whereas large cortical infarctions may cause severe, permanent disability. The extent of the infarct governs the potential for rehabilitation.

Patients with massive strokes from which meaningful recovery is unlikely should receive palliative care. Patients who have had a cerebral infarct are at risk for additional strokes and for myocardial infarcts. Statin therapy and antiplatelet therapy reduces the recurrence rate. Occurrence of stroke in younger patients has a better prognosis than older patients. Despite these predictors, the marked variability among patients makes early prognostication difficult.

AIMS OF THE STUDY

AIMS OF THE STUDY

To make a comparative study on clinical profile of stroke in males and females with regard to variability in

- Age Predilection
- Clinical presentation
- Type of stroke
- Frequency of risk factor in each group
- Outcome difference during the hospital stay

MATERIALS & METHODS

MATERIALS AND METHODS

STUDY PLACE: Dept. Of Medicine, Govt. Stanley Medical College, Chennai.

STUDY DESIGN: Prospective Cross Sectional Observational Study.

STUDY POPULATION: 50 Males and 50 Females.

INCLUSION CRITERIA:

All patients admitted with sudden onset of focal neurologic deficit attributable to a focal vascular cause.

EXCLUSION CRITERIA:

Patients not satisfying the above said criteria.

Patients with an already existing focal neurological deficit.

Patients with subdural hemorrhage or extradural hemorrhage.

A detailed history is taken and complete physical examination of all the systems focusing mainly on neurological and cardiovascular system are performed after taking verbal consent from the patient or relatives. All details are recorded on a self designed questionnaire which included patient's name, age, presenting history, past history of cerebro vascular accident or a cardiac disease, hypertension, diabetes mellitus, smoking and alcoholism. Findings of the examination are also recorded in the form which includes general condition of the patient, peripheral markers of hypercholesterolemia, carotid bruit, pulse rate, rhythm, vessel wall thickening, renal bruit, blood pressure, cardiac and neurological examination.

The patients are then subjected to blood investigations like complete blood counts, renal function test, blood sugar levels, total cholesterol, ECG, echocardiogram, brain imaging like CT. In suspected cases of cortical venous thrombosis, MRI with MR venogram was taken. Coagulation profile was done only in suspected cases.

Type of stroke, site and extent of lesion noted. Patients were treated with all the required measures and neurological consultation was sought and their advice was also incorporated into the management protocol. These patients were followed up till discharge. Daily assessment of the neurological status was done and the prognosis assessed. Data analyzed and sorted to see the differences between the two groups. Clinical manifestations categorized and risk factors plotted from history as well as from the examinations.

The study conducted is a Prospective Cross Sectional Observational Study on two groups of patients. This study has been intended to throw light on the differences in the clinical profile of stroke between the representative samples of male and female population and thereby to formulate newer prevention strategies.

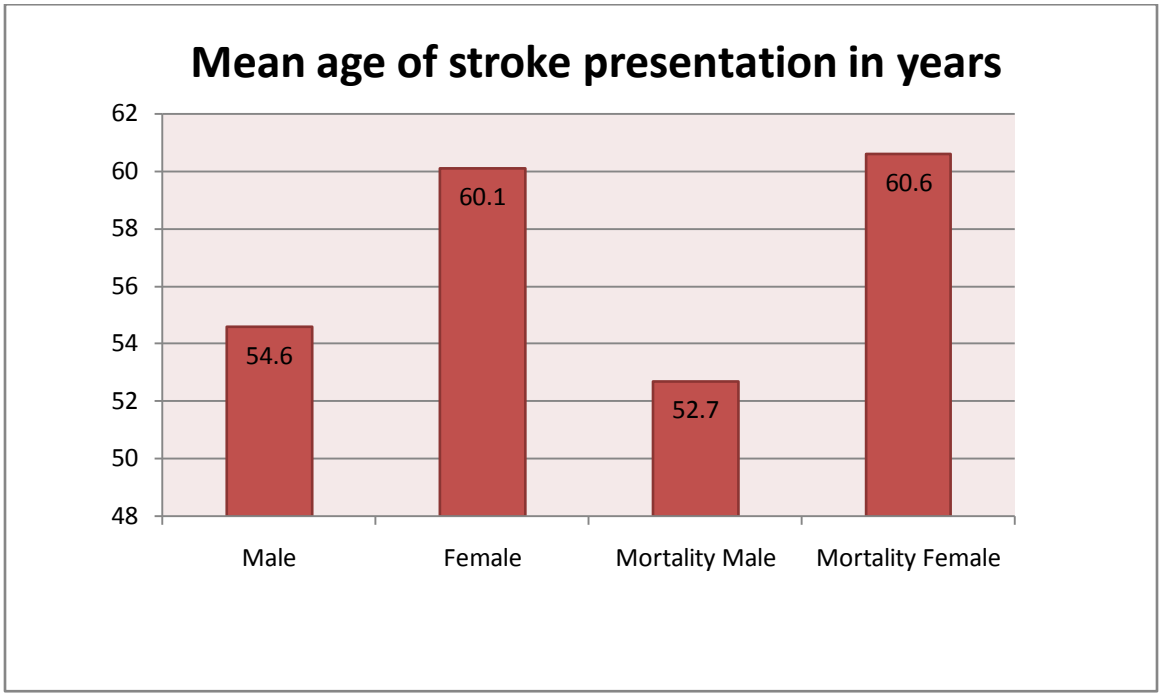
OBSERVATIONS

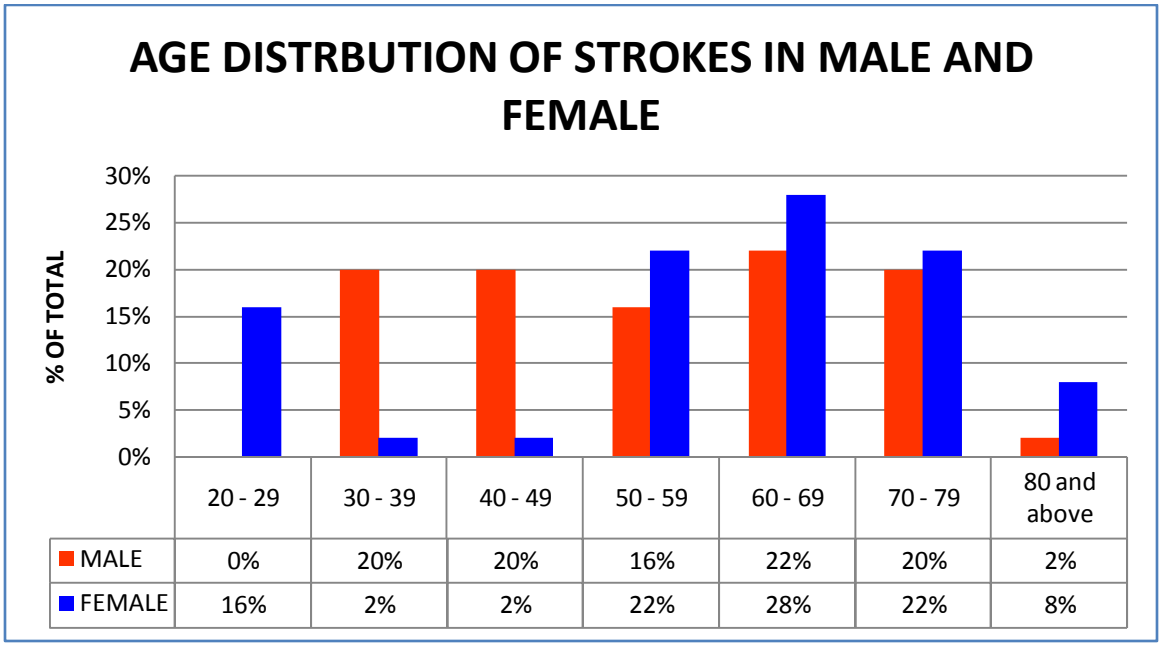
Table 1 Mean age of stroke presentation in years

	Number(total)	Minimum age	Maximum age	Mean
Male	50	33	83	54.6
Female	50	25	88	60.1
Infarct	80	25	88	58.7
Hemorrhage	20	31	76	52.2
Mortality Male	12	35	75	52.7
Mortality Female	6	31	8	60.6

Table 2 Age distribution of stroke in males and females

Age		Gender		Total
		Male	Female	
20 – 29	Total number	0	8	8
	% of Total	0%	16%	8%
30 – 39	Total number	10	1	11
	% of Total	20%	2%	11%
40 – 49	Total number	10	1	11
	% of Total	20%	2%	11%
50 – 59	Total number	8	11	19
	% of Total	16%	22%	19%
60 – 69	Total number	11	14	25
	% of Total	22%	28%	25%
70 – 79	Total number	10	11	21
	% of Total	20%	22%	21%
80 and above	Total number	1	4	5
	% of Total	2%	8%	5%
Total	Total number	50	50	100
	% of Total	100%	100%	100%





	20 - 29	30 - 39	40 - 49	50 - 59	60 - 69	70 - 79	80 and above
MALE	0%	20%	20%	16%	22%	20%	2%
FEMALE	16%	2%	2%	22%	28%	22%	8%

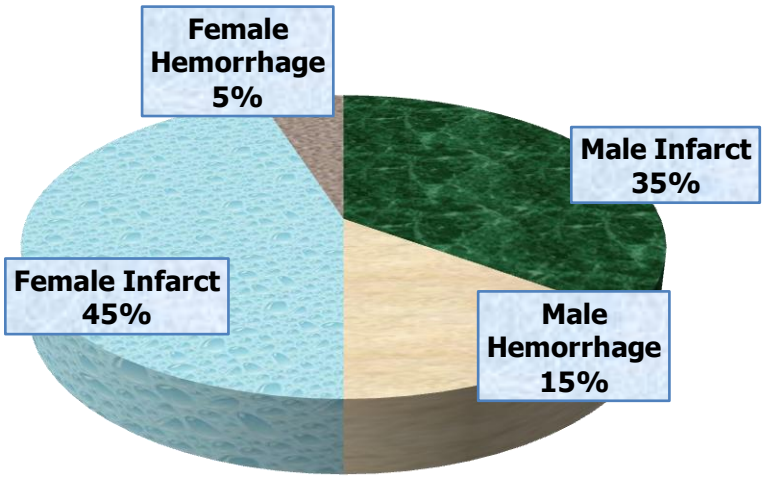
Table 3 Distribution of lesion in males and females

	Male		Female	
Total no. of cases	50		50	
Lesion	Infarct	Hemorrhage	Infarct	Hemorrhage
No. of cases	35	15	45	5
Percentage	70%	30%	90%	10%

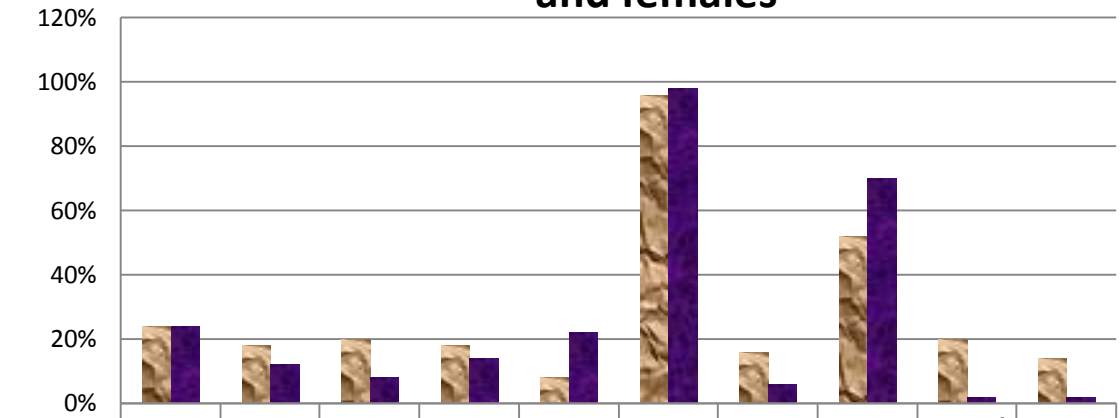
Table 4 Distribution of clinical features in males and females

Clinical features	Male		Female		Total	
Altered sensorium	12	24%	12	24%	24	24%
Headache	9	18%	6	12%	15	15%
Vomiting	10	20%	4	8%	14	14%
Seizures	9	18%	7	14%	16	16%
Aphasia	4	8%	11	22%	15	15%
Motor deficit	48	96%	49	98%	97	97%
Sensory deficit	8	16%	3	6%	11	11%
Facial nerve palsy	26	52%	35	70%	61	61%
Rest of cranial nerves lesions	10	20%	1	2%	11	11%
Ataxia	7	14%	1	2%	8	8%

Distribution of lesion in males and females



Distribution of clinical features in males and females



	Altered sensorium	Headache	Vomiting	Seizures	Aphasia	Motor deficit	Sensory deficit	Facial nerve palsy	Rest of cranial nerves lesions	Ataxia
Male	24%	18%	20%	18%	8%	96%	16%	52%	20%	14%
Female	24%	12%	8%	14%	22%	98%	6%	70%	2%	2%

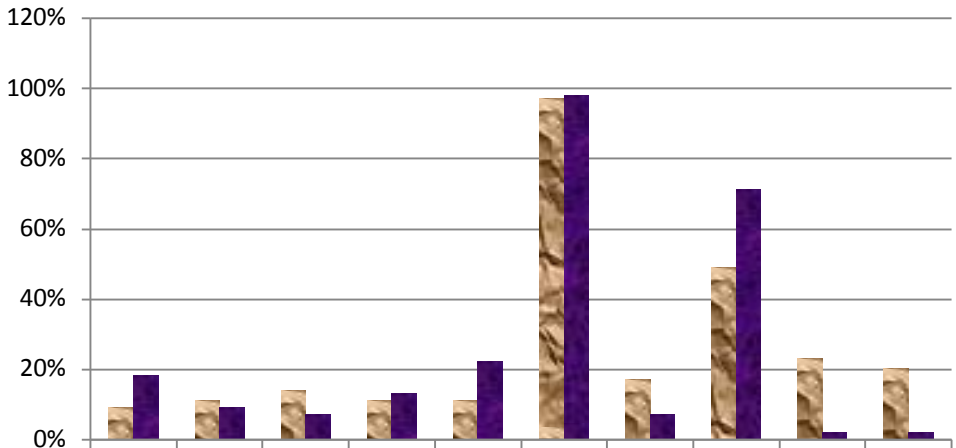
Table 5 Lesion wise distribution of clinical features in males and females

Lesion / Clinical Features	Infarct			Hemorrhage		
	Male	Female	Total	Male	Female	Total
Altered sensorium	9%	18%	14%	60%	80%	65%
Headache	11%	9%	10%	33%	40%	35%
Vomiting	14%	7%	10%	33%	20%	30%
Seizures	11%	13%	13%	33%	20%	30%
Aphasia	11%	22%	18%	0%	20%	5%
Motor deficit	97%	98%	98%	100%	100%	100%
Sensory deficit	17%	7%	11%	13%	0%	10%
Facial nerve palsy	49%	71%	61%	60%	60%	60%
Rest of cranial nerves lesions	23%	2%	11%	13%	0%	10%
Ataxia	20%	2%	10%	0%	0%	0%

Table 6 Distribution of risk factors in males and females

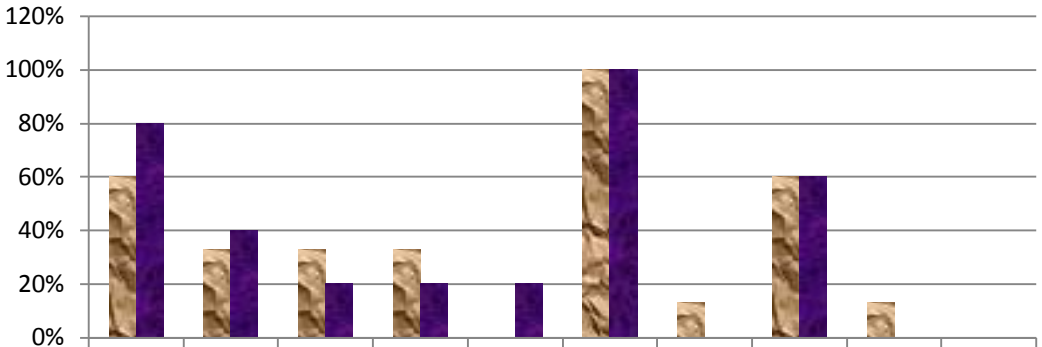
Risk Factors	Male		Female		Total	
Systemic Hypertension	22	44%	33	66%	55	55%
Diabetes Mellitus	11	22%	19	38%	30	30%
Hypercholesterolemia	20	40%	24	48%	44	44%
Smoking	41	82%	4	8%	45	45%
Alcoholism	40	80%	1	2%	41	41%
Coronary Artery Disease	11	22%	6	12%	17	17%
Atrial Fibrillation	1	2%	5	10%	6	6%
Left ventricular hypertrophy	3	6%	7	14%	10	10%
Vascular Malformation	1	2%	0	0%	1	1%
Carotid Atheroma	3	6%	1	2%	4	4%
Post Partum	0	0%	4	8%	4	4%
Hyperhomocysteinemia	3	6%	0	0%	3	3%
Anti phospholipid antibody syndrome	0	0%	1	2%	1	1%

Lesion wise distribution of clinical features in males and females -INFARCT



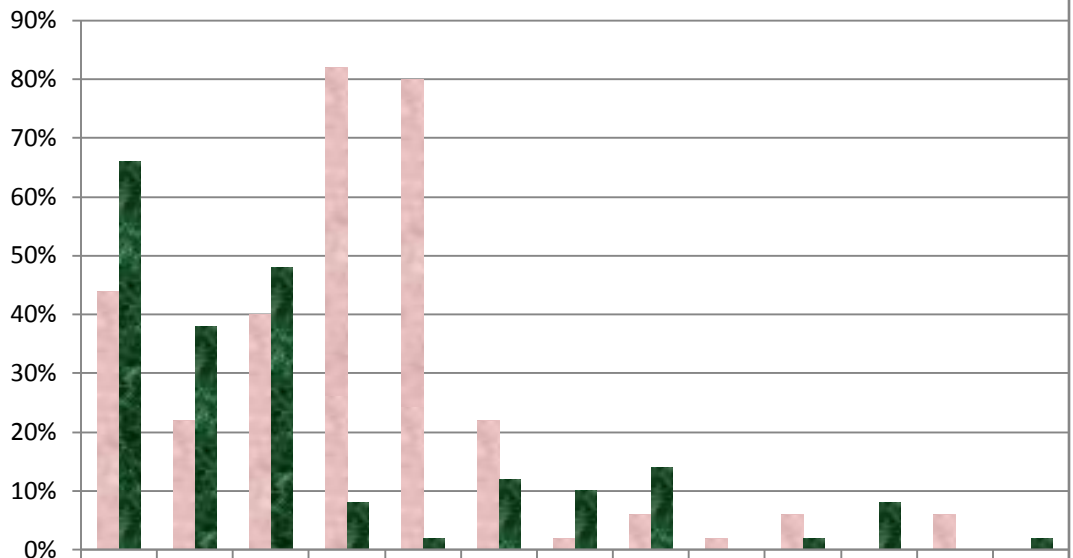
	Altered sensorium	Headache	Vomiting	Seizures	Aphasia	Motor deficit	Sensory deficit	Facial nerve palsy	Rest of cranial nerves lesions	Ataxia
Male Infarct	9%	11%	14%	11%	11%	97%	17%	49%	23%	20%
Female Infarct	18%	9%	7%	13%	22%	98%	7%	71%	2%	2%

Lesion wise distribution of clinical features in males and females-HEMORRHAGE



	Altered sensorium	Headache	Vomiting	Seizures	Aphasia	Motor deficit	Sensory deficit	Facial nerve palsy	Rest of cranial nerves lesions	Ataxia
Male Hemorrhage	60%	33%	33%	33%	0%	100%	13%	60%	13%	0%
Female Hemorrhage	80%	40%	20%	20%	20%	100%	0%	60%	0%	0%

Distribution of risk factors in males and females



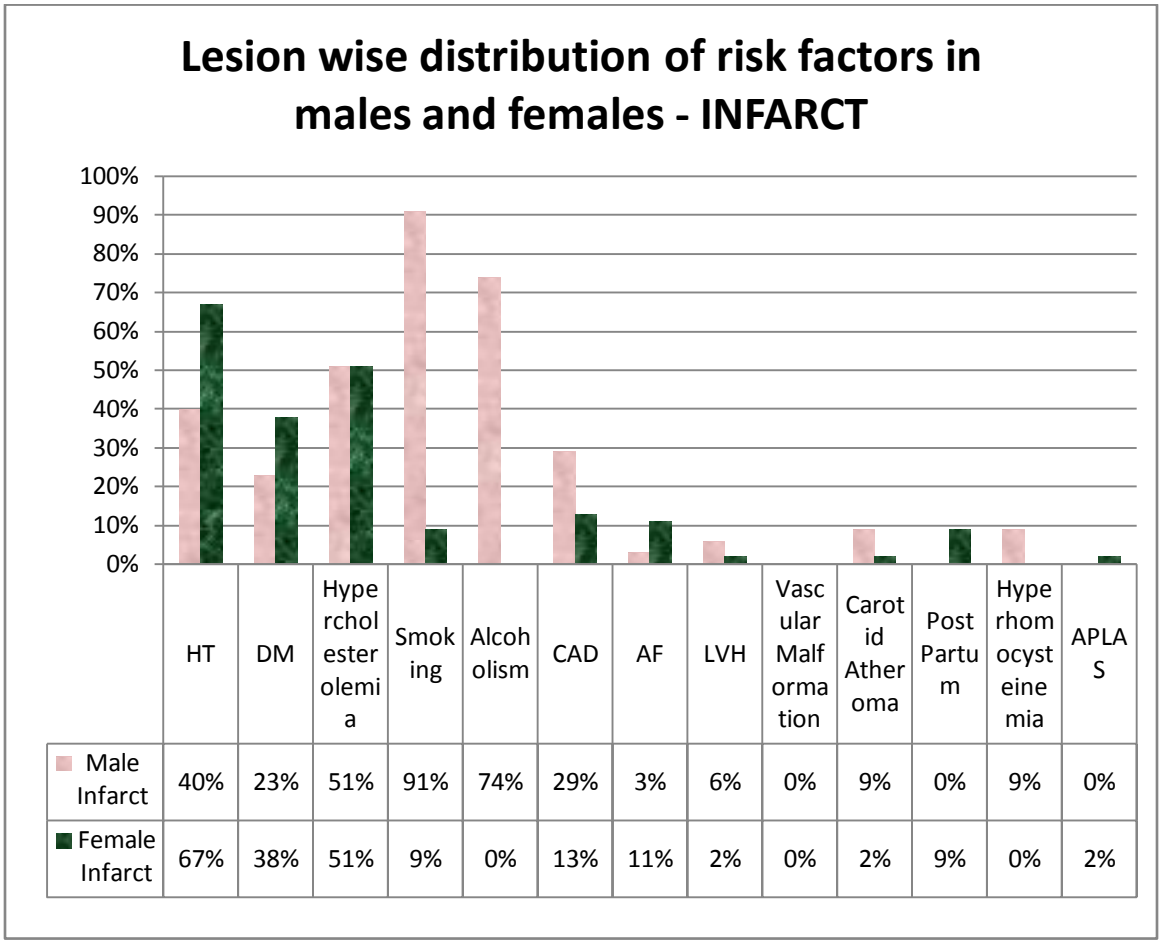
	HT	DM	Hypercholesterolemia	Smoking	Alcoholism	CAD	AF	LVH	Vascular Malformation	Carotid Atheroma	Post Partum	Hyperhomocysteinemia	APLAS
Male	44%	22%	40%	82%	80%	22%	2%	6%	2%	6%	0%	6%	0%
Female	66%	38%	48%	8%	2%	12%	10%	14%	0%	2%	8%	0%	2%

Table 7 Lesion wise distribution of risk factors in males and females

Risk Factors / Lesion	Infarct			Hemorrhage		
	Male	Female	Total	Male	Female	Total
Systemic Hypertension	40%	67%	55%	53%	60%	55%
Diabetes Mellitus	23%	38%	31%	20%	40%	25%
Hypercholesterolemia	51%	51%	51%	13%	20%	15%
Smoking	91%	9%	45%	60%	0%	45%
Alcoholism	74%	0%	33%	93%	20%	75%
Coronary Artery Disease	29%	13%	20%	7%	0%	5%
Atrial Fibrillation	3%	11%	8%	0%	0%	0%
Left ventricular hypertrophy	6%	2%	4%	40%	20%	35%
Vascular Malformation	0%	0%	0%	7%	0%	5%
Carotid Atheroma	9%	2%	5%	0%	0%	0%
Post Partum	0%	9%	5%	0%	0%	0%
Hyperhomocysteinemia	9%	0%	4%	0%	0%	0%
Anti phospholipid antibody syndrome	0%	2%	1%	0%	0%	0%

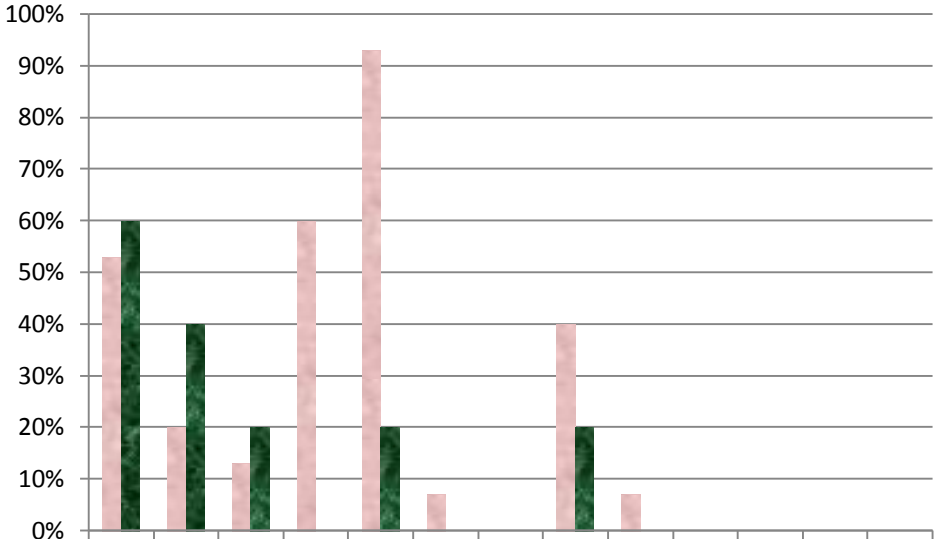
Table 8 Distribution of stroke syndromes in males and females

Syndrome		Male	Female	Total
Total anterior circulation (TACS)	Total number	11	14	25
	% of Total	22%	28%	25%
Partial anterior circulation (PACS)	Total number	5	11	16
	% of Total	10%	22%	16%
Lacunar (LACS)	Total number	26	24	50
	% of Total	52%	48%	50%
Posterior circulation (POCS)	Total number	8	1	9
	% of Total	16%	2%	9%
Total	Total number	50	50	100



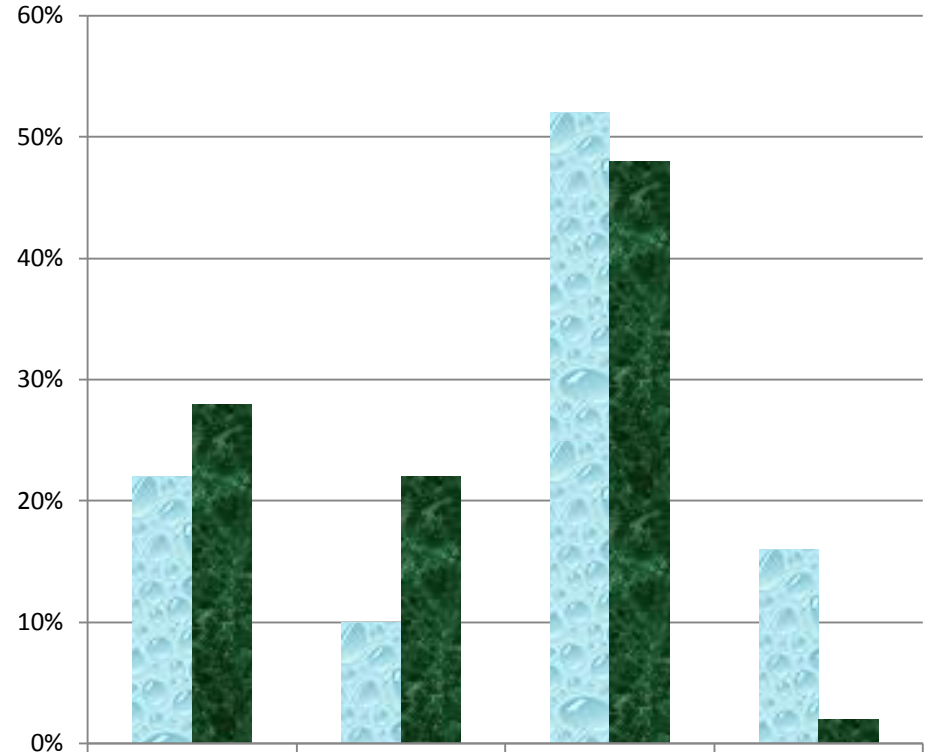
	HT	DM	Hypercholesterolemia	Smoking	Alcoholism	CAD	AF	LVH	Vascular Malformation	Carotid Atheroma	Post Partum	Hyperhomocysteinemia	APLAS
Male Infarct	40%	23%	51%	91%	74%	29%	3%	6%	0%	9%	0%	9%	0%
Female Infarct	67%	38%	51%	9%	0%	13%	11%	2%	0%	2%	9%	0%	2%

Lesion wise distribution of risk factors in males and females - HEMORRHAGE



	HT	DM	Hypercholesterolemia	Smoking	Alcoholism	CAD	AF	LVH	Vascular Malformation	Carotid Atheroma	Post Partum	Hyperhomocysteinemia	APLAS
Male Hemorrhage	53%	20%	13%	60%	93%	7%	0%	40%	7%	0%	0%	0%	0%
Female Hemorrhage	60%	40%	20%	0%	20%	0%	0%	20%	0%	0%	0%	0%	0%

Distribution of stroke syndromes in males and females



	(TACS)	(PACS)	(LACS)	(POCS)
GENDER Male	22%	10%	52%	16%
GENDER Female	28%	22%	48%	2%

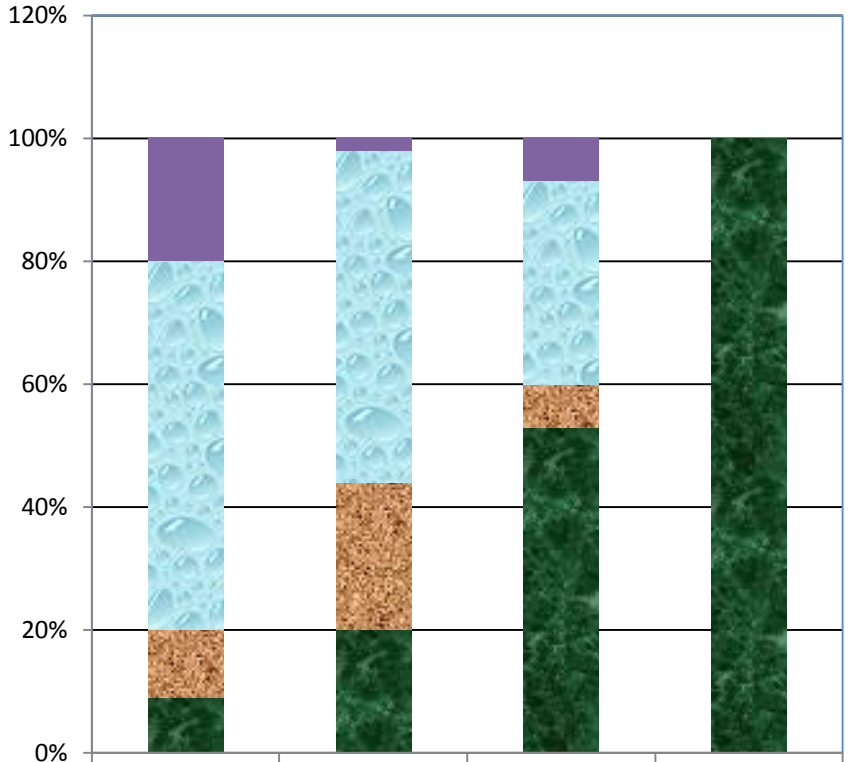
Table 9 Lesion wise distribution of stroke syndrome in males and females

Lesion / Syndrome		Infarct			Hemorrhage		
		Male	Female	Total	Male	Female	Total
Total anterior circulation	Total number	3	9	12	8	5	13
	% of Total	9%	20%	15%	53%	100%	65%
Partial anterior circulation	Total number	4	11	15	1	0	1
	% of Total	11%	24%	19%	7%	0%	5%
Lacunar	Total number	21	24	45	5	0	5
	% of Total	60%	53%	56%	33%	0%	25%
Posterior circulation	Total number	7	1	8	1	0	1
	% of Total	20%	2%	10%	7%	0%	5%
Total	Total number	35	45	80	15	5	20
	% of Total	100%	100%	100%	100%	100%	100%

Table 10 Outcome in males and females

Outcome		Male	Female	Total
Improved	Total number	16	20	36
	% of Total	32%	40%	36%
Static	Total number	22	21	43
	% of Total	44%	42%	43%
Mortality	Total number	12	9	21
	% of Total	24%	18%	21%
Total	Total number	50	50	100
	% of Total	100%	100%	100%

Lesion wise distribution of stroke syndrome



	Male Infarct	Female Infarct	Male Hemorrhage	Female Hemorrhage
Posterior circulation	20%	2%	7%	0%
Lacunar	60%	54%	33%	0%
Partial anterior circulation	11%	24%	7%	0%
Total anterior circulation	9%	20%	53%	100%

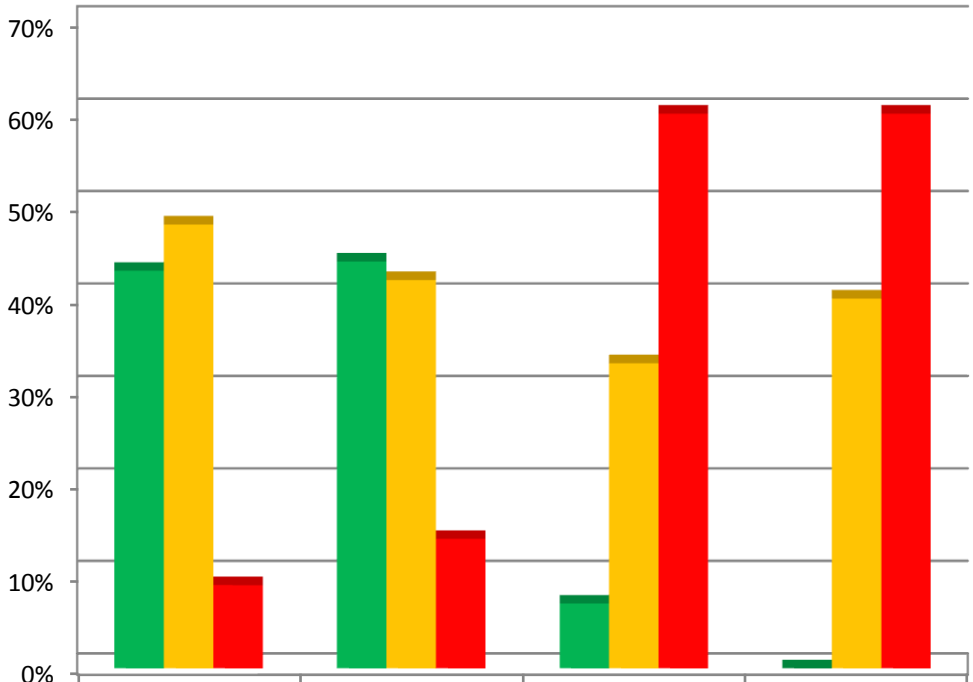
Table 11 Lesion wise outcome in males and females

Lesion / Syndrome		Infarct			Hemorrhage		
		Male	Female	Total	Male	Female	Total
Improved	Total number	15	20	35	1	0	1
	% of Total	43%	44%	44%	7%	0%	5%
Static	Total number	17	19	36	5	2	7
	% of Total	49%	42%	45%	33%	40%	35%
Mortality	Total number	3	6	9	9	3	12
	% of Total	9%	13%	11%	60%	60%	60%
Total	Total number	35	45	80	15	5	20
	% of Total	100%	100%	100%	100%	100%	100%

Table 12 Syndrome wise outcome

Syndrome \ Outcome		Improved	Static	Mortality
Total anterior circulation	Total number	1	8	16
	% of Total	3%	19%	76%
Partial anterior circulation	Total number	7	8	1
	% of Total	19%	19%	5%
Lacunar	Total number	27	21	2
	% of Total	75%	49%	10%
Posterior circulation	Total number	1	6	2
	% of Total	3%	14%	10%
Total	Total number	36	43	21
	% of Total	100%	100%	100%

Lesion wise outcome in males and females

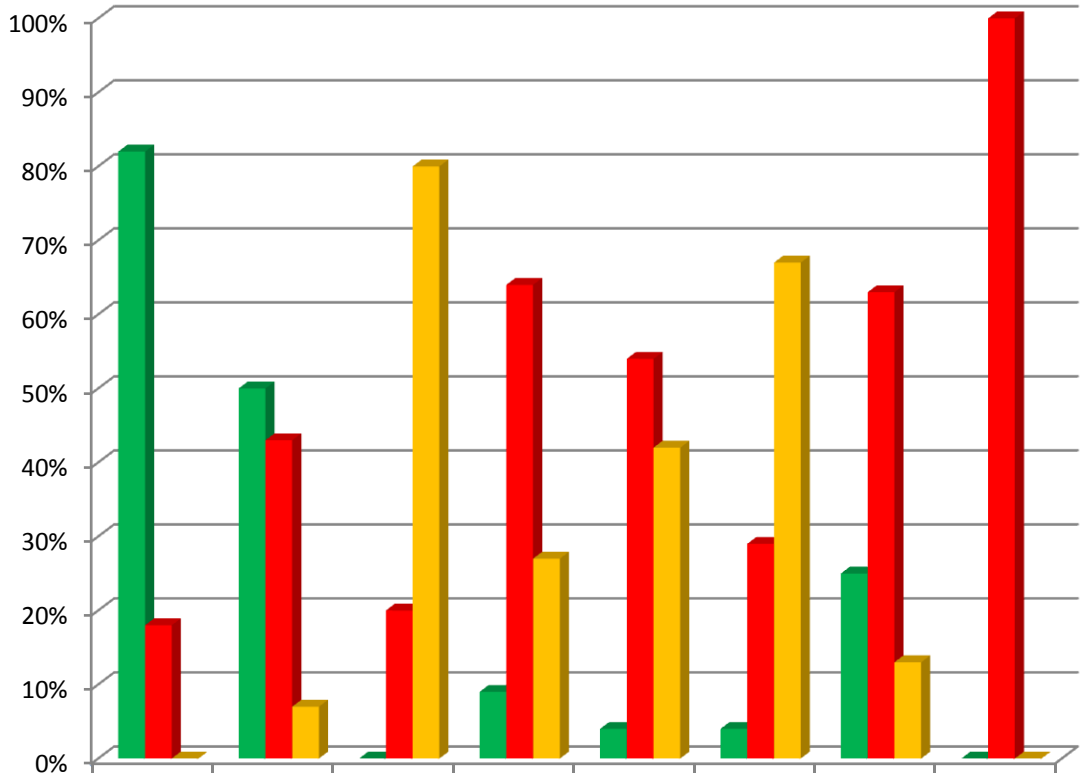


	Male Infarct	Female Infarct	Male Hemorrhage	Female Hemorrhage
Improved	43%	44%	7%	0%
Static	48%	42%	33%	40%
Mortality	9%	14%	60%	60%

Table 13 Syndrome wise outcome in males and females

Syndrome \ Outcome		Improved		Static		Mortality		Total	
		Total No.	% of total	Total No.	% of total	Total No.	% of total	Total No.	% of total
TACS		0	0%	8	33%	16	67%	24	100%
	Male	0	0%	2	18%	9	82%	11	100%
	Female	1	7%	6	43%	7	50%	14	100%
PACS		7	44%	8	50%	1	6%	16	100%
	Male	4	80%	1	20%	0	0%	5	100%
	Female	3	27%	7	64%	1	9%	11	100%
LACS		27	54%	21	42%	2	4%	50	100%
	Male	11	42%	14	54%	1	4%	26	100%
	Female	16	67%	7	29%	1	4%	24	100%
POCS		1	11%	6	67%	2	22%	9	100%
	Male	1	13%	5	63%	2	25%	8	100%
	Female	0	0%	1	100%	0	0%	1	100%

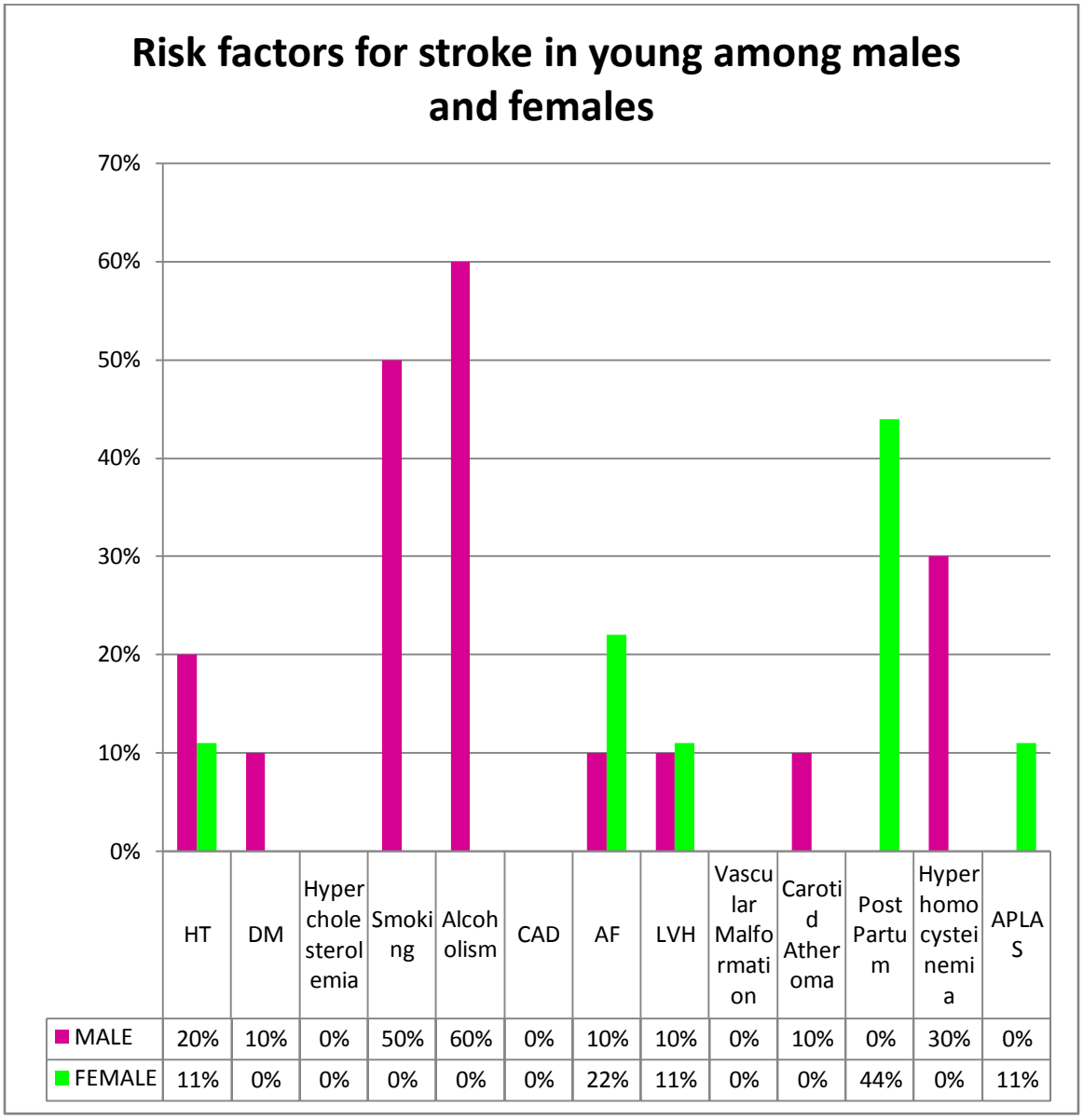
Syndrome wise outcome in males and females



	TACS		PACS		LACS		POCS	
	Male	Female	Male	Female	Male	Female	Male	Female
MORTALITY	82%	50%	0%	9%	4%	4%	25%	0%
STATIC	18%	43%	20%	64%	54%	29%	63%	100%
IMPROVED	0%	7%	80%	27%	42%	67%	13%	0%

Table 14 Risk factors for stroke in young among males and females

Risk factors	Male (Total 10 pts)		Female (Total 9 pts)		Total (19 pts)	
	Total No.	% of total	Total No.	% of total	Total No.	% of total
Systemic Hypertension	2	20%	1	11%	3	16%
Diabetes Mellitus	1	10%	0	0%	1	5%
Hypercholesterolemia	0	0%	0	0%	0	0%
Smoking	5	50%	0	0%	5	26%
Alcoholism	6	60%	0	0%	6	32%
Coronary artery disease	0	0%	0	0%	0	0%
Atrial Fibrillation	1	10%	2	22%	3	16%
Left ventricular hypertrophy	1	10%	1	11%	2	11%
Vascular Malformation	0	0%	0	0%	0	0%
Carotid Atheroma	1	10%	0	0%	1	5%
Post Partum	0	0%	4	44%	4	21%
Hyperhomocysteinemia	3	30%	0	0%	3	16%
Antiphospholipid antibody syndrome	0	0%	1	11%	1	5%



	HT	DM	Hypercholesterolemia	Smoking	Alcoholism	CAD	AF	LVH	Vascular Malformation	Carotid Atheroma	Post Partum	Hyperhomocysteinemia	APLAS
MALE	20%	10%	0%	50%	60%	0%	10%	10%	0%	10%	0%	30%	0%
FEMALE	11%	0%	0%	0%	0%	0%	22%	11%	0%	0%	44%	0%	11%

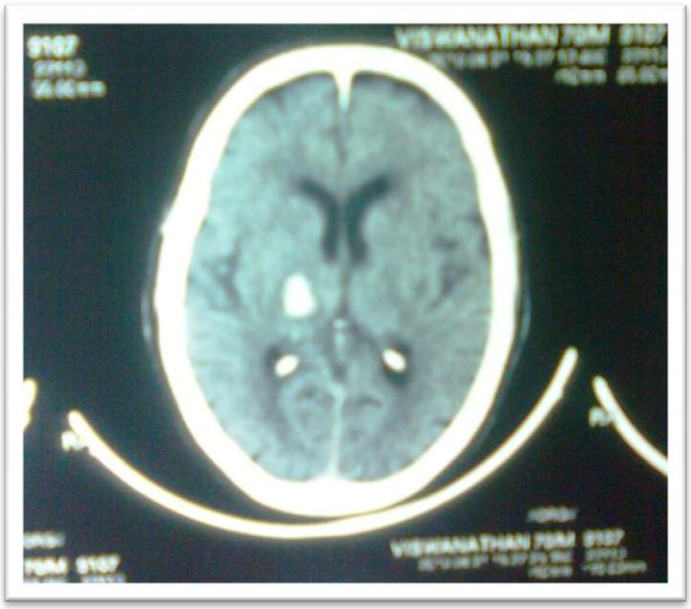


Figure.1 RIGHT THALAMUS, PUTAMEN, CORONA RADIATA HEMORRHAGE

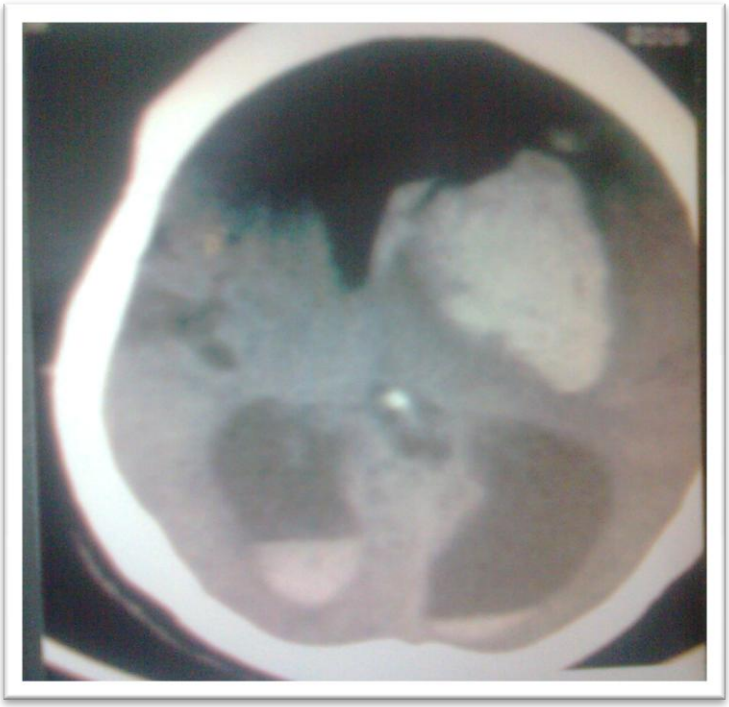


Figure.2-LEFT CAPSULOGANGLIONIC HEMORRHAGE WITH INTRAVENTRICULAR EXTENSION WITH MIDLINE SHIFT

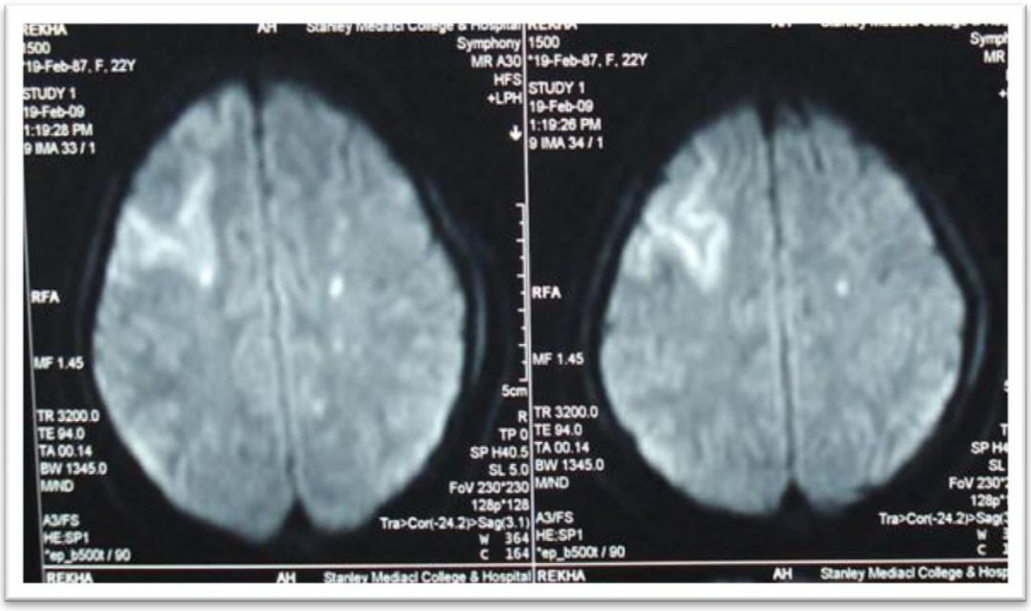


Figure.3- INFARCT RIGHT FRONTAL & PARIETAL LOBE

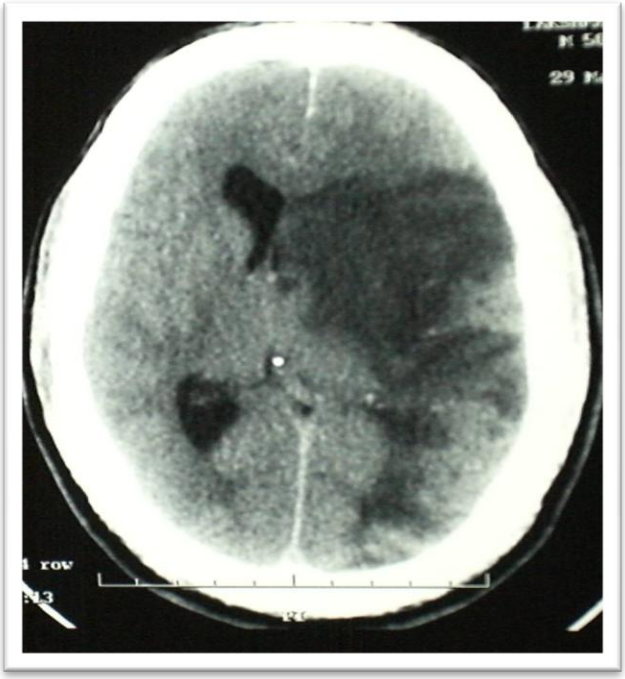


Figure.4- LEFT MASSIVE MCA INFARCT WITH MIDLINE SHIFT

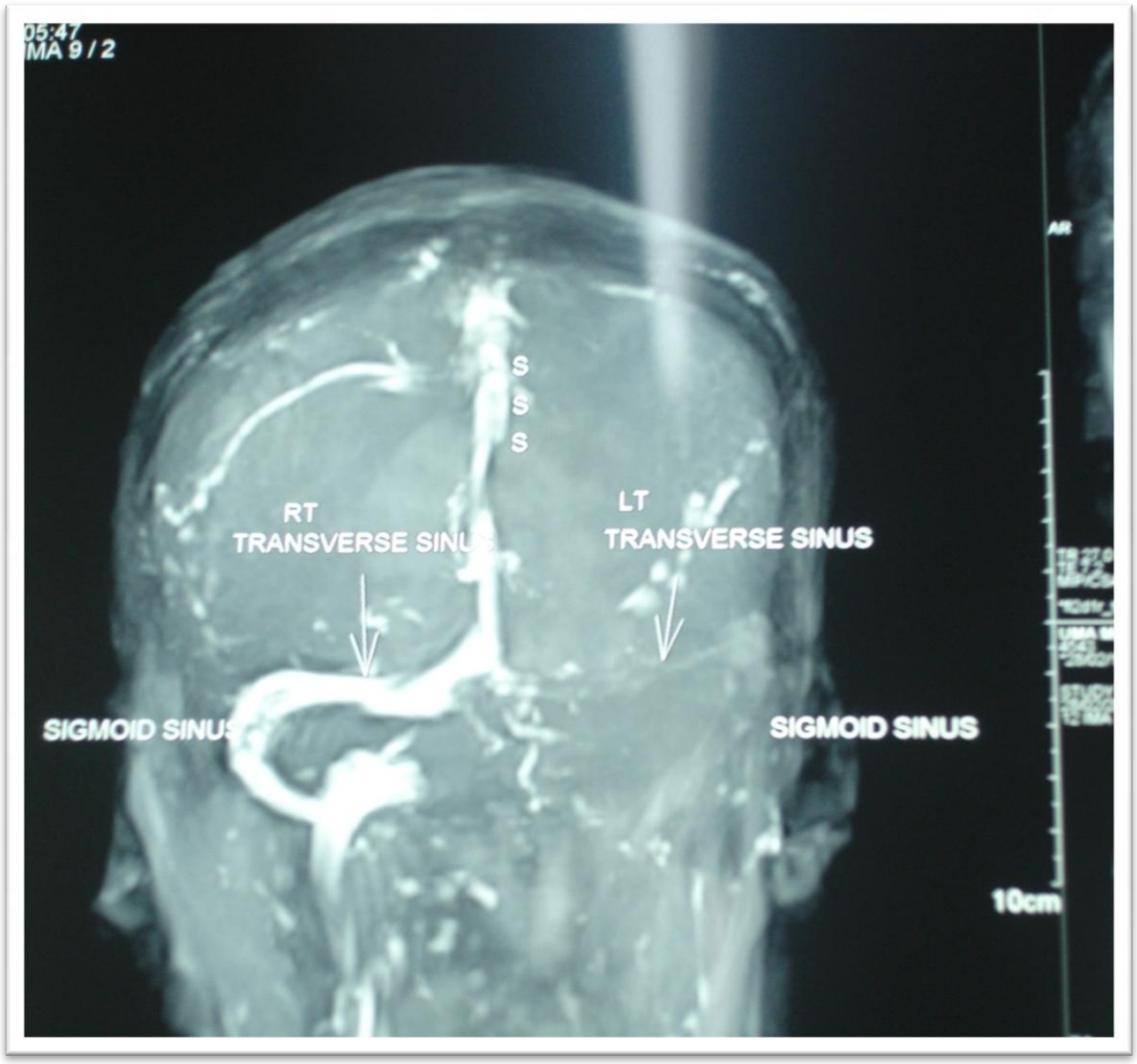


Figure.5- MRV SHOWING SIGMOID AND TRANSVERSE SINUS THROMBOSIS ON LEFT

RESULTS

RESULTS

In the study titled “Comparative study of stroke in males and females”, 50 males and 50 females were enrolled based on the framed inclusion and exclusion criteria. They were analyzed according to their age, clinical features, risk factors, type of stroke and outcome.

In both the sexes, ischemic stroke was the commonest type of stroke accounting for 80 % of the total. Out of the 50 males, 15 males had hemorrhagic stroke (30 %) and 35 males had ischemic stroke (70 %). Among the females studied, 5 females had a hemorrhagic stroke (10 %) and 45 females (90 %) had ischemic stroke depicting the higher incidence of hemorrhagic stroke in males. Of the hemorrhagic strokes, 87% was caused by cerebral bleed and rest 13 % by the subarachnoid hemorrhage.

The mean age of presentation in males in our study was 54.6 yrs and that of females was 60.1yrs. Maximum number of cases in total population was found in the age group of 60 – 69 yrs. Mean age for infarct in males and females were 56.3 and 60.5 yrs respectively, while that of mean age for hemorrhage in males and females were 50.7 and 56.4yrs respectively.

In the two groups, stroke in young had a total of 19 cases, out of which 10 were males and 9 were females. Though the difference is not significant, the age group which females presented maximally was between 20 – 29 yrs (88%) and that males predominated in the 30 – 39 yrs (100%) group.

The most common presentation in both the groups was motor deficit accounting for 96% in males and 98% in females. Next common presentation was seventh cranial nerve palsy accounting for 52% in males and 70% in females. Third common feature was altered sensorium in both males and females contributing to 24% each. The presentation

of altered sensorium (65%), headache (35%), vomiting (30%) and seizures (30%) were more frequent in hemorrhagic stroke than ischemic stroke.

In both the sexes altered sensorium was the most common presentation of hemorrhagic stroke with females outnumbering males (80% against 60%) but the frequency of seizures, headache and vomiting were all equal in males (33%) whereas in females headache predominated over the other two features (40%).

Incidence of altered sensorium in ischemic stroke was 18% in females and 9% in males. Hence altered sensorium is frequent in females than males irrespective of the stroke type, a possible contributing factor for the observed poor outcome in females. Ataxia and rest of cranial nerve palsies were observed more frequently in males due to the higher incidence of posterior circulation stroke in males.

According to the Bamford classification of stroke, the cases were observed and analyzed. The most common type of stroke observed was LACS accounting to 52% and 48% in male and females, followed by TACS 28% in female against 22% in male. The third most common stroke in male was POCS 16% while in female the POCS was only 2%. But PACS was more frequent in a female (22%) than in a male (10%). In cases of male infarct the most common type of stroke syndrome was LACS followed by POCS, PACS and TACS. Female infarcts also presented commonly with LACS but the second common presentation was PACS followed by TACS and POCS. Of the males with hemorrhagic stroke the frequently associated stroke syndrome was TACS followed by LACS, but all the female hemorrhagic strokes presented with TACS.

On analyzing the risk factor profile of the two samples, the most commonly associated risk factors in males in the order of association as from the observations made was smoking(82%), alcoholism(80%), hypertension (44%), hypercholesterolemia (40%),

diabetes mellitus and coronary artery disease (CAD) 22% each. In the females, the most commonly associated risk factors in the order of occurrence was hypertension (66%), hypercholesterolemia (48%), diabetes mellitus (38%), LVH(14%), CAD (12%), and AF contributing to 10%.

Of the total stroke patients analyzed 55% of patients had hypertension hence being the most common risk factor for stroke. Mean arterial blood pressure was higher in patients with hemorrhagic strokes (133mmHg in males & 130 in mmHg females) than ischemic strokes (115 mmHg in males 117 mmHg in females) and showed no significant difference between the groups. In the study 44% of males had hypertension and 66% of females had hypertension. Hypertension is the leading risk factor for stroke in females but in males the major association is with smoking and alcoholism. Hypertension as a risk factor for hemorrhagic stroke was 53% and 60% in the male and the female groups, whereas in cases of ischemic stroke females showed a higher association to the extent of 67% when compared to 40 % in males.

Hypercholesterolemia was present in 44% of the population studied, with females outnumbering males (48% against 40%). Out of these 93% presented with ischemic stroke and the remaining 7% presented with a hemorrhagic stroke. Also of the 80 cases of infarct, 51% were hypercholesterolemics and of the 20 cases of hemorrhagic stroke, 15% were hypercholesterolemics. Hence hypercholesterolemia is closely associated with ischemic stroke rather than hemorrhagic stroke. Hypercholesterolemia contributed as a risk factor for stroke in females more than that in males possibly due to postmenopausal hormone changes affecting the lipid profile.

Smoking was observed in 45% of the total patients. Among these 80% had ischemic stroke and 20% had hemorrhagic stroke. Smoking was associated with 82% of

the male strokes whereas females had an association to about 8%. Only 4 female smokers were present in the study and all sustained ischemic stroke. Male smokers present in our study were 41 in numbers, out of them 32 (78%) sustained an ischemic stroke and rest 9 (22%) had a hemorrhagic stroke. Smoking as a risk factor for ischemic stroke in males came up to 91% and formed a leading causal factor among male infarcts, whereas it accounted for as a risk factor in 9% of female infarcts. Among the hemorrhagic stroke in males, smoking was associated as a risk factor in 60%.

Alcoholism was observed as the next commonly associated risk factor. Of the 100 patients, 41 were alcoholic (41%). Among these, 26 sustained ischemic strokes accounting to 63% and 15 patients sustained a hemorrhagic stroke accounting to 37%. But of the 20 cases of hemorrhagic strokes, alcoholism was associated with 75 % of the cases and of the 80 ischemic strokes, 33% were alcoholic. This clearly depicts the closer relationship of alcoholism with hemorrhagic stroke more than the ischemic stroke.

The frequency of male strokes with alcoholism was 80% whereas female strokes had a frequency of only 2%. Only one of the female in the study was an alcoholic and she presented with a hemorrhagic stroke. In males, 74% of those who had an ischemic stroke were alcoholics. The association of alcoholism as a risk factor was stronger for hemorrhagic stroke coming up to 93%.

Since the habits of alcoholism and smoking were high among our males, they contribute as the major risk factor for stroke in this group. This relationship might get altered significantly if the female population adapts to these habits.

In our study, the next associated risk factor was diabetes mellitus which was present in 30 patients (30%). Of these 83% had an ischemic stroke and 17% had a hemorrhagic stroke. Of the 80 infarcts, 31% were diabetic and that out of the 20 cases of

hemorrhagic strokes, 25% were diabetics. This showed a better correlation of diabetes with ischemic stroke than hemorrhagic stroke. Male infarcts had an association with diabetes to the extent of 23% whereas females showed an association of 38%. In the hemorrhagic strokes also the association of diabetes was 20% in males and 40% in females. Thereby in the study conducted, female strokes showed a greater relationship with regard to diabetes as a risk factor than males.

In the study conducted, males were associated with coronary artery disease more commonly than females. There was association of coronary artery disease in 22% of males and 12% of females.

Left ventricular hypertrophy was seen in 10% of cases. This also was observed more in females than males (14% in females against 6% in males), probably due to the higher proportion of hypertensive cases in females compared to males. All of these cases sustained ischemic stroke possibly due to the concurrence of other atherosclerotic risk factors in these individuals.

Atrial fibrillation formed a predominant risk factor among females. In our study, 6 patients had atrial fibrillation, out of which 5 were females and 1 was a male. Among these 6 patients, 5 were rheumatic heart disease and one female was a postpartum Cardiomyopathy with left ventricular clot. AF contributed as a risk factor in females to the extent of 11% while that in males it was 3%. This disparity is possibly due to the higher incidence of rheumatic heart disease in females in our country.

In our study, 3 males and 1 female had carotid atheroma. All of these patients presented with infarcts. Post partum stroke observed in our study was 4 in number, 3 of them presented with venous infarct and one of them presented with post partum cardiomyopathy with left ventricular clot. Hyperhomocysteinemia was associated with

3 cases of stroke, all were males and presented with infarct as stroke in young. Only one case of vascular malformation was observed in the study population. It was a male who presented with posterior communicating artery aneurysm with subarachnoid hemorrhage. One case of antiphospholipid syndrome was observed. It was a female who presented as stroke in young and had deep vein thrombosis of left lower limb at the time of presentation.

The risk factors with regard to the stroke in young varied considerably between the groups. Females predominated with association of post partum CVT and AF whereas males predominated with association of alcoholism, smoking, and hyperhomocysteinemia.

On analyzing the outcome in the study, the mean age of mortality in males was 52.7 yrs and that in females was 60.6 yrs. Males had a poorer outcome than females (case fatality rate of 24% in males and that in females was 18%).

Among the infarcts, females showed a poorer outcome than males (13% against 9%) possibly due to the later age of presentation and other potential risk factors that are related to age. Among the hemorrhagic strokes, females and males had a similar outcome. In total, outcome as observed in our study was poor in males than females, probably due to the increased occurrence of hemorrhagic stroke in males.

The patients who presented with TACS had the worst outcome, of the died patients, 76% (57% in males & 43% in females) was contributed by TACS, might be due to the fact that it involves a larger area in the brain The best outcome group was the ones who presented with LACS (54% improved at the time of their discharge) with females predominating males (67% against 42%). On considering the clinical features, those who presented with altered sensorium at the time of admission had the worst outcome. Males

presenting with altered sensorium had a more chance of dying than females in our study (83% in males against 67% in females). Mean arterial blood pressure was high in the poor outcome people. Most commonly associated risk factors in the poor outcome group varied between male and female. Males predominated with alcoholism while female predominated with hypertension.

DISCUSSION

DISCUSSION

In this study conducted, the age, type of stroke, risk factor profile, clinical features and outcome of the patient during the hospital study were all observed. These findings are herewith described relating to the conclusions of some other similar studies. These differences so brought to light will allow us to make conclusions regarding the community in which the study is done.

We observed that the ischemic strokes (85%) were more common than the hemorrhagic stroke (15%). This was the conclusion also cited in American stroke association which says 87% of stroke is due to ischemic and 13% is due to hemorrhagic stroke.

In the study it is observed that there was higher incidence of stroke in the older age [31.]The mean age of male strokes was 54.6 yrs and that of female strokes was 60.1 yrs. According to study published in American stroke association, the mean age for men were 68.6 years, and 72.9 years for women, which means that women get their first strokes on an average 4.3 years later than men. This was done in the western population. In our population it is seen that the mean age is lesser than in the west possibly related to our risk factor profile and genetic make[27]. The incidence of stroke in our study in patients more than 60 yrs of age in males was 23 in number accounting to 36% and that in females was 30 in number accounting to 46 %, depicting a later peaking of stroke incidence in females. Studies say stroke is more common among men than women and the difference tends to decrease with age with women getting their first stroke about four and a half years later than men[67].

In the male population it is seen that the hemorrhagic stroke was common than that in the females. In the study done in the west, there is male predomination of hemorrhagic stroke[67]. In a study from the WHO MONICA project, the populations from Eastern Europe and Finland had higher incidence rates of SAH in men than in women similar to our study which showed 2 cases of SAH, all were males.

The most common presenting feature in both the groups was presence of motor deficit (97%). This goes in correlation with our Indian studies conducted, like the one by Man Mohan Mehndiratta et al. which found that 80.3% had hemiplegia. Altered sensorium and vomiting were all common with the hemorrhagic stroke than ischemic stroke.

The stroke incidence was observed to be high in the post menopausal women. Lesser incidence of women in the reproductive age group is probably related to genetic factors, positive effects of estrogen on the cerebral circulation[35]. A lifetime exposure to ovarian estrogens may protect against ischemic stroke, at least of the noncardioembolic type[16], an effect that seems to cease with menopause [40].

Hypertension was the commonest risk factor for both hemorrhagic and ischemic stroke. Hypertension is the single most important modifiable risk factor for ischemic stroke[69]. Hypertension is the most powerful modifiable risk factor for ICH[36]. Studies prove that hypertensive's have a fourfold rise of stroke at systolic blood pressure values more than 160 mmhg[58].

High blood cholesterol is a major risk factor for heart disease and also increases the risk of stroke. Western data's suggest that the increased levels of total cholesterol especially LDL was associated with higher incidence of ischemic stroke. Hypercholesterolemia was found more in females than males in our study. Majority of

the cases resulted in ischemic stroke. Studies show that women's total cholesterol is higher than men's from age 55 onwards, hence a postmenopausal surge of hypercholesterolemics have been noticed in our study. Research suggests that having high triglycerides may increase the risk for women more than for men.

Diabetes mellitus contributed as a significant risk factor for stroke. According to some studies, the frequency of diabetes mellitus was higher in Indian population 18 - 42% than in the western population 10 - 26%[73].

Cigarette smoking increases risk of ischemic stroke nearly two times[63.] In our study also 80% of the smokers sustained ischemic stroke and rest 20% sustained a hemorrhagic stroke. Honolulu heart program links cigarette smoking with both ischemic and hemorrhagic stroke.

Majority of the males in the study were alcoholic and of the hemorrhagic strokes it was associated with 75%, supported by studies saying increasing alcohol consumption increases risk for brain hemorrhage[24] .

Atrial fibrillation is common in female population than male population in our study which has been concluded in many other studies as, women are generally at higher risk than men for atrial fibrillation related cardio embolic stroke [21, 22, 66, 68, 74].

Incidence of CVT as from many studies showed a mean age of 38 years. Hypercoagulable state was the most common predisposing factor followed by pregnancy, malignancy, and hyperhomocysteinemia. Our study also showed 4 CVT patients, all presented as stroke in young, of which 3 were post partum females and one was a hyperhomocysteinemic male[33].

In considering the outcome in the two groups, total outcome was poor in the males than females possibly due to the higher incidence of hemorrhagic stroke which by

in itself has a poor outcome. On considering the infarct cases female had a poorer outcome than males, possibly due to later age at presentation and associated risk factors attributable to age. In a meta analysis, case fatality was higher among women than among men in 26 of 31 studies, and higher in male patients in only 3 studies. Case fatality was 1.25 times higher among women[67].

CONCLUSIONS

CONCLUSIONS

- Ischemic stroke was the most common type of stroke in both sexes. Hemorrhagic stroke was more common in males than females. Of the hemorrhagic strokes, intracerebral bleed was more common than subarachnoid hemorrhage.
- Mean arterial blood pressure was higher in patients with hemorrhagic strokes than ischemic strokes and showed no difference between the groups.
- The mean age of presentation of stroke in males was 54.6 and that in females was 60.1. Females had later peaking of incidence when compared to males.
- Among the stroke in young, the case clustering in females was in the third decade, whereas in males was in the fourth decade.
- The most common clinical presentation of stroke was motor deficit followed by seventh cranial nerve paralysis and altered sensorium. There was no disparity between males and females in this regard.
- The presentation of altered sensorium, headache, vomiting, and seizures were more common in hemorrhagic stroke than ischemic stroke. Among these females predominated over males with altered sensorium and headache whereas males predominated over females with vomiting and seizures.
- Ataxia and rest of cranial nerve palsy was more common in males than females.

  The most common type of stroke syndrome in both the sexes were LACS, next being TACS. PACS presented more in females and POCS presented more in males.

- Among the hemorrhagic strokes, the most common clinical syndrome was TACS. All the female cases belonged to this type. Even though the commonest clinical presentation in males was also TACS, some presented with LACS and PACS.
- Systemic hypertension is the commonest risk factor attributable to stroke in females, whereas smoking formed the predominant risk factor in males.
- Systemic hypertension was the most commonly associated risk factor for stroke in the total population. There was a higher frequency of hypertension in females than males. It formed the leading risk factor for stroke in females.
- Hypercholesterolemia has been associated in females more than that of males reflecting the post menopausal effect on the lipid profile. It was predominantly associated with ischemic stroke.
- Smoking was more commonly associated in males than females reflecting the altered behavioral pattern in males. Smoking as a risk factor in males is more significantly associated with infarcts than hemorrhagic stroke. Smoking therefore formed a leading causal factor among male infarcts.
- Major proportions of the male population in our study were alcoholics. Alcoholism as a contribution for causation of stroke was significantly high for hemorrhagic stroke than that for infarct. One female alcoholic was present in our study, who sustained a hemorrhagic stroke.
- Diabetes affected more females than males and contributed more towards infarct than hemorrhage in both the sexes.
- Coronary artery disease was more commonly associated with male than females.

- Left ventricular hypertrophy is more commonly associated with female than male possibly due to the higher incidence of hypertension in females in our study.
- Atrial fibrillation has been associated more in the females than the males possibly due to the higher incidence of rheumatic heart disease in females in our country.
- Young stroke in females was predominantly observed with the post partum females, major proportion due to cortical venous thrombosis.
- Among the infarcts, mortality rates were higher in females than males possibly due to the later age of presentation and other potential risk factors that are related to age, but in total, males had a poorer outcome than females probably due to the increased occurrence of hemorrhagic stroke in males.
- Patients who presented with TACS had a poorer outcome and that who presented with LACS had good outcome when compared to others. There was no disparity between the groups in this respect.
- Patients who presented with altered sensorium had a poor outcome, with males showing greater association than females.
- The most commonly associated risk factor in the poor outcome group in female population was hypertension and that in male population was alcoholism.

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ANNEXURES

PROFORMA

PROFORMA

1. Name:

2. Age:

3. Sex:

4. Marital status:

5. No of Children:

6. Educational status:

7. Occupation:

8. Family Income/yr :

9. Address:

10. Clinical presentation on admission:

11. Time of Onset of symptoms:

12. H/O Head trauma:

13. H/O Previous episodes of focal neurological deficit:

14. Hypertension: Y / N

    How many yrs:

    Treatment:

15. Diabetes: Y / N

    How many yrs:

    Type:

    Treatment:

16. Hyperlipidemia: Y/N

    Treatment:

17. Extramarital / Premarital exposure:

18. Alcohol: Y /N

19. Smoking: Y / N

20. Tobacco chewing: Y / N

21. Sedentary habits:

22. Menstrual H/O:

Premenopausal / Postmenopausal

Menopause: Yrs since menopause

23. Diet H /O: Veg /Non Veg

Duration

Type of cooking oil

Salt intake

Coffee/Tea

24. Personality: type A / type B

25. **General examination**

Conscious :

Orientation :

Anemia :

Clubbing :

Icterus :

Pedal edema :

JVP :

Markers of Hypercholesterolemia :

Optic Fundus :

PR : Rate /min : Regular/irregular : Vessel wall thickening

Carotid bruit + / -

BP : mmHg

RR : / min

Specific breathing pattern if present –

26. **CVS:**

Apical Impulse:

Heart Sounds:

Murmurs:

27. **RS:**

Air entry:

Adventitious sounds:

28. **P /A :**

29. **CNS :**

Higher functions:

Cranial nerves:

Motor system:

Sensory system:

Cerebellum:

Extra pyramidal:

INVESTIGATIONS:

1.Urine R /E :

2.RFT :

Urea

Creatinine

Electrolytes

3.CBC:

TC	DC	ESR
Hb	PCV	Platelets

4. FBS

PPBS

5. Total cholesterol:

6. CXR PA View:

7. ECG:

8. CT Brain:

9. MRI Brain: (if required)

CONSENT FORM

1. I agree to participate in study titled **"**Comparative study on clinical profile of stroke in males and females "

2. I confirm that I have been told about this study in my mother tongue and have had the opportunity to ask questions.

3. I understand that my participation is voluntary and I may refuse to participate at any time without giving any reason and without giving any reason and without affecting my benefits.

4. I agree not to restrict the use of any data / results that arise from this study.

Name of the participant:

Signature / Thumb print:

Witness: Investigator:

CERTIFICATE FOR APPROVAL OF ETHICAL COMMITTEE

To

Dr.K.Vidhya, PG in MD(GM)

Dear **Dr.K.Vidhya, PG in MD(GM)**

The Institutional Ethics Committee reviewed and discussed your application for approval of the project entitled

"Comparative study on clinical profile of stroke in males and females "

The following members of the ethics committee were present at the meeting held on 28.01.2008 at the Council Hall, Stanley Medical College, Chennai-1 at 10.00AM

Dr.C.B.Tharani, Director of Pharmacology,
Madras Medical College, Chennai-3 - Chairman of the Ethics Committee
Dr.S. Chitra, Vice-Principal,
Stanley Medical College, Chennai – 1- Member Secretary of the Ethics Committee

MEMBERS

Dr.Jayanthi
Prof.of Medical Gastroenterology
Dr.Madhavan
Prof.of Pharmacology
Dr.E.Dhandapani
Prof.of Medicine
Dr.Sujatha Sridharan
Prof.of Paediatrics
Thiru.Pachaiappan,
Junior Administrative Officer,
Thiru.A. Senthil Manoharan,
Advocate

We approve the project to be conducted in its presented form.

The Institutional Ethics Committee/Independent Ethics Committee expects to be informed about the progress of the study, any SAE occurring in the course of the study, any changes in the protocol and patient information/informed consent and asks to be provided a copy of the final report.

Yours sincerely,

Member Secretary,
Ethics Committee

MEMBER SECRETARY
ETHICAL COMMITTEE,
STANLEY MEDICAL COLLEGE
CHENNAI-600 001.

ABBREVATIONS

ABBREVIATIONS

ACA – Anterior Cerebral Artery

AF – Atrial Fibrillation

CAD- Coronary Artery Disease

CSF – Cerebro Spinal fluid

CT – Computed Tomogram

CVA – Cerebro Vascular Accident

CVT – Cerebral Venous Thrombosis

DALY – Disability Adjusted Life Year

DM – Diabetes Mellitus

DVT – Deep Vein Thrombosis

ECG – Electrocardiogram

HDL – High Density Lipoprotein

HT – Hypertension

ICA – Internal Carotid Artery

ICH – Intra Cerebral Hemorrhage

INR – International Normalized Ratio

IST – International Stroke Trial

LACS – Lacunar syndrome

LDL – Low Density Lipoprotein

LVH – Left Ventricular Hypertrophy

MCA – Middle Cerebral Artery

MRI – Magnetic Resonance Imaging

NVAF – Non Valvular Atrial Fibrillation

PACS – Partial Anterior Circulation Syndrome

PCA – Posterior Cerebral Artery

PCOM – Posterior Communicating Artery

PICA – Posterior Inferior Cerebellar Artery

POCS – Posterior Circulation Syndrome

RHD – Rheumatic Heart Disease

rtPA – Recombinant tissue plasminogen activator

SAH – Sub Arachnoid Hemorrhage

TACS – Total Anterior Circulation Syndrome

TC – Total Cholesterol

TIA – Transient Ischemic Attack

UTI – Urinary Tract Infection

VBA – Vertebro Basilar Artery

WHO – World Health Organization