A Dissertation on

STUDY ON RISK FACTORS, CLINICO-RADIOLOGICAL PROFILE AND THE OUTCOME IN PATIENTS WITH ACUTE CEREBROVASCULAR DISEASE

Submitted to

THE TAMILNADU DR. M.G.R. MEDICAL UNIVERSITY
CHENNAI – 600032

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

M.D. BRANCH - I

GENERAL MEDICINE

DEPARTMENT OF GENERAL MEDICINE
STANLEY MEDICAL COLLEGE
CHENNAI – 600 001
MAY 2018

CERTIFICATE BY INSTITUTION

This is to certify that **Dr. ABDUL LATHEEF ABDUL GAFOOR,** Post - Graduate Student (May 2015 TO April 2018) in the Department of General Medicine, STANLEY MEDICAL COLLEGE, Chennai- 600 001, has done this dissertation on "**Study On Risk Factors, Clinico-Radiological Profile And The Outcome In Patients With Acute Cerebrovascular Disease**" under my guidance and supervision in partial fulfillment of the regulations laid down by the Tamilnadu, Dr. M. G. R. Medical University, Chennai, for M.D. (General Medicine), Degree Examination to be held in May 2018.

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DECLARATION

I, **Dr. ABDUL LATHEEF ABDUL GAFOOR,** declare that I carried out this work on "**Study On Risk Factors, Clinico-Radiological Profile And The Outcome In Patients With Acute Cerebrovascular Disease**" at the Department and Medical wards of Government Stanley Hospital .

I also declare that this bonafide work or a part of this work was not submitted by me or any other for any award, degree, or diploma to any other university, board either in India or abroad.

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

Dr. ABDUL LATHEEF ABDUL GAFOOR

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Above all, I thank the **ALMIGHTY** for gracing me this opportunity and for giving me courage and good health so that I could carry on my academic course without much hindrance.

Dr. ABDUL LATHEEF ABDUL GAFOOR

CERTIFICATE

This is to certify that this dissertation work titled "**Study On Risk Factors, Clinico-Radiological Profile And The Outcome In Patients With Acute Cerebrovascular Disease**" of the candidate **Dr. ABDUL LATHEEF ABDUL GAFOOR** with registration number: **201511051** for the award of **M.D.** in the branch of **GENERAL MEDICINE**. I personally verified the *urkund.com* website for the purpose of plagiarism check. I found that the uploaded thesis file contains from introduction to conclusion pages and result shows **2 percentage of plagiarism** in the dissertation.

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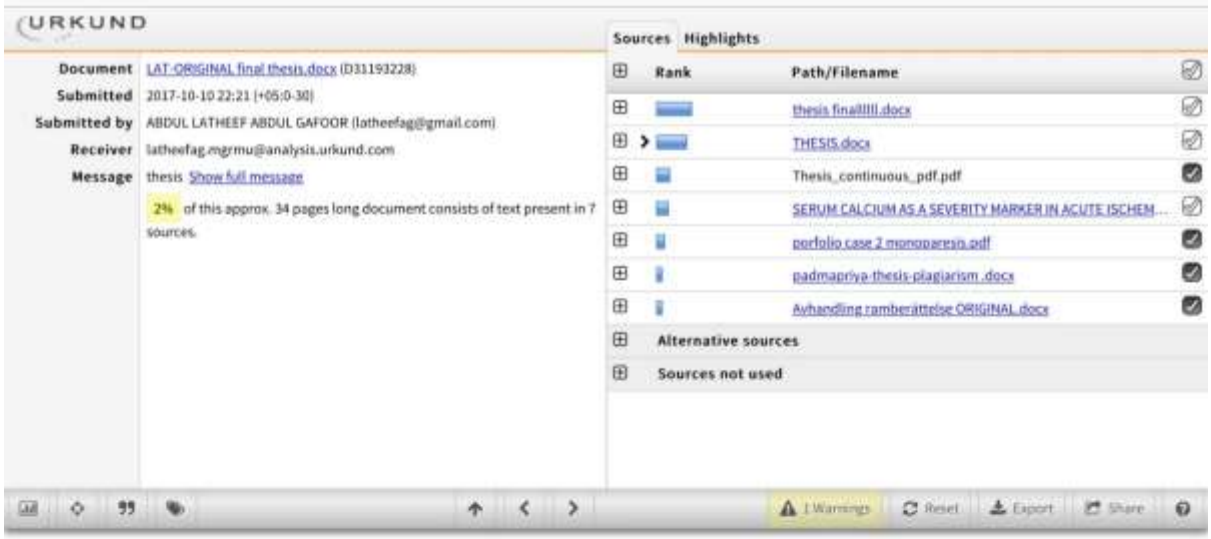

INTRODUCTION

In recent years, with more than 1.2 billion inhabitants, India is undergoing remarkable economic and demographic changes resulting in a transition from poverty related infectious and nutritional deficiency diseases toward lifestyle related cardiovascular and cerebrovascular diseases. In aging population stroke or cerebro-vascular disease is a non-communicable disease of increasing social economic importance. According to WHO stroke remains the second most common cause of worldwide mortality. In the future, demographic changes, urbanization & increased exposure to major risk factors will fuel the stroke burden in South Asian countries (1). Stroke is a devastating and disabling cerebrovascular disease with some amount of residual deficit leading onto economic loss. In 2010, the absolute numbers of people with first stroke (16.9 million), stroke survivors (33 million), stroke related deaths (5.9 million), and DALYs lost (102 million) were high and had significantly increased since 1990 (68%, 84%, 26%, and 12% increase, respectively), with most of the burden (68.6% incident strokes, 52.2% prevalent strokes, 70.9% stroke deaths, and 77.7% DALYs lost) happened in low income and middle income countries. In 2010, 5.2 million ,that is 31% of strokes were in children (aged >20 years old) and young and middle-aged adults (20–64 years), to which children, young and middle aged adults from low income and middle income countries contributed almost 74,000 (89%) and 4.0 million (78%), respectively, of the total burden. If at all, these trends in stroke incidence, mortality, and DALYs continue, by 2030 there will be almost 12 million stroke deaths, 70 million stroke survivors, and more than 200 million DALYs lost globally.(2) National Commission on Macroeconomics and Health, estimated 1.67 million stroke cases in India by the year 2015, suggesting that stroke will be a rising epidemic in India in the near future. This may be due to the high prevalence of hypertension, diabetes, dyslipidemia, the fast changing lifestyles and restructuring of the population.(3) Because of high incidence of stroke and the high costs expended for each strokevpatient, it accounts for a sizeable amount of the health care costs. Thus, stroke and its sequelae are important issues for health care planners in government, insurance companies and medical services everywhere. Because the costs of treatment and the economic consequences of lost productivity are so great, prevention of stroke will be a very cost effective strategy. Time and again it has been proved that stroke is the eventual result of a series of insults on the cerebral and cardiovascular systems. These risk factors not only determine when a stroke will occur, but also the type and severity of the cerebrovascular accident. The risk factors which lead on to stroke differs among different communities, changes with age and sex. Assessing these risk factors can help us devise preventive strategies in stroke. Since most of the studies are done in populations from developed nations, it is imperative that we need more studies from developing nations like India to find out corresponding statistics. Even among Indians there are

INSTITUTIONAL ETHICAL COMMITTEE,
STANLEY MEDICAL COLLEGE, CHENNAI-1

Title of the Work : Study on risk factors, clinic- Radiological profile and the outcome in patients with acute cerebrovascular disease.

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The request for an approval from the Institutional Ethical Committee (IEC) was considered on the IEC meeting held on 21.04.2017 at the Council Hall, Stanley Medical College, Chennai-1 at 2PM

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

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

1. You should inform the IEC in case of changes in study procedure, site investigator investigation or guide or any other changes.
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6. You should submit the summary of the work to the ethical committee on completion of the work.

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INTRODUCTION

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Because of high incidence of stroke and the high costs expended for each stroke patient, it accounts for a sizeable amount of the health care costs. Thus, stroke and its sequelae are important issues for health care planners in government, insurance companies and medical services everywhere. Because the costs of treatment and the economic consequences of lost productivity are so great, prevention of stroke will be a very cost effective strategy.

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series of insults on the cerebral and cardiovascular systems. These risk factors not only determine when a stroke will occur, but also the type and severity of the cerebrovascular accident. The risk factors which lead on to stroke differs among different communities, changes with age and sex. Assessing these risk factors can help us devise preventive strategies in stroke. Since most of the studies are done in populations from developed nations, it is imperative that we need more studies from developing nations like India to find out corresponding statistics. Even among Indians there are considerable life style differences among populations from various communities and regions. So to be more specific we need studies from south India itself if we have to affirmatively assess the stroke indices in our population.

Cerebrovascular disease is the most common cause of neurological morbidity and mortality in adults, with ischemic stroke accounting for the majority of cases (80%) globally. The etiology of stroke in majority of cases could be identified by a proper history taking, an adequate general examination and judicious use of investigations. A cardiovascular examination and imaging of the brain usually gives ample information regarding the etiology, type, site and severity of the stroke. Many studies conducted before have shown that ischemic

strokes were common than hemorrhagic and the etiology was different for both. The etiology and site of stroke varies with different communities too. Statistic data on the etiology and site of strokes with proper correlation with brain imagery is still scanty for south Indian population. By this study an attempt is made to find out these indices with respect to a population catered by a tertiary care hospital in urban Tamil Nadu.

The clinical features of stroke also vary with type of stroke. For example, a history of loss of consciousness and seizures is more in favor of a hemorrhagic lesion. In addition lesions in various sites give rise to different clinical pictures. An attempt is made to find out the clinical profile of stroke in this study population. The ultimate outcome of the study population also will be seen. These data will help us to assess how they differ from the western data.

AIMS & OBJECTIVES OF THE STUDY

1. To study the various risk factors of acute cerebrovascular accident in a tertiary care center.

2. To study the clinical profile of acute CVA.

3. To study the vascular etiology in acute CVA.

4. To assess the factors affecting outcome of stroke during hospital stay.

REVIEW OF LITERATURE

HISTORICAL REVIEW :

The word ‘stroke’ was introduced by William Cole in a Physico-medical Essay in 1689. Before Cole, the word ‘apoplexy’ was used to describe acute non traumatic brain injuries. During the 1950s, physicians felt the need to find out a terminology for temporary vascular related events of brain dysfunction that do not qualify as strokes and ‘transient ischemic attack’ came into use(4).

EPIDEMIOLOGY :

Worldwide cerebrovascular disease or stroke is the second leading cause of death and it occurs predominantly in the middle age and older adults. In 2005, WHO estimated 5.7 million deaths caused by stroke which was equivalent to 9.9% of total deaths. Out of those stroke deaths, over 85 percent where living in the low to middle income countries and 33% were aged less than 70 years. In major hospitals stroke accounts for 2%of hospital registrations 1.5% of medical registration and 9 to 30%of neurological admissions.(3)

Developing countries like India now faces a double burden of communicable and noncommunicable diseases. Stroke is one among the leading causes of death and disability in India. The estimated adjusted range of prevalence rate of stroke is 84 to 262 per 1 lakh in rural and 334 to 424 per 1 lakh population. The incidence rate is 119 to 145 per 1 lakh which is based on recent population based studies. There is a wide variation in case fatality rates among the states with the highest rate being 42 % in Kolkata.(6)

DEFINITION:

'Stroke' is classically characterized as a neurological deficit attributed to an acute focal injury of the central nervous system (CNS) by a vascular cause, including cerebral infarction, intracerebral hemorrhage (ICH), and subarachnoid hemorrhage (SAH). Despite its global impact, the term "stroke" is not consistently defined in clinical practice, in clinical research, or in assessments of the public health.(4).

The current World Health Organization definition of stroke (introduced in 1970 and still used) is "rapidly developing clinical signs of focal (or global) disturbance of cerebral function, lasting more than 24 hours or leading to death,

with no apparent cause other than that of vascular origin".(4)

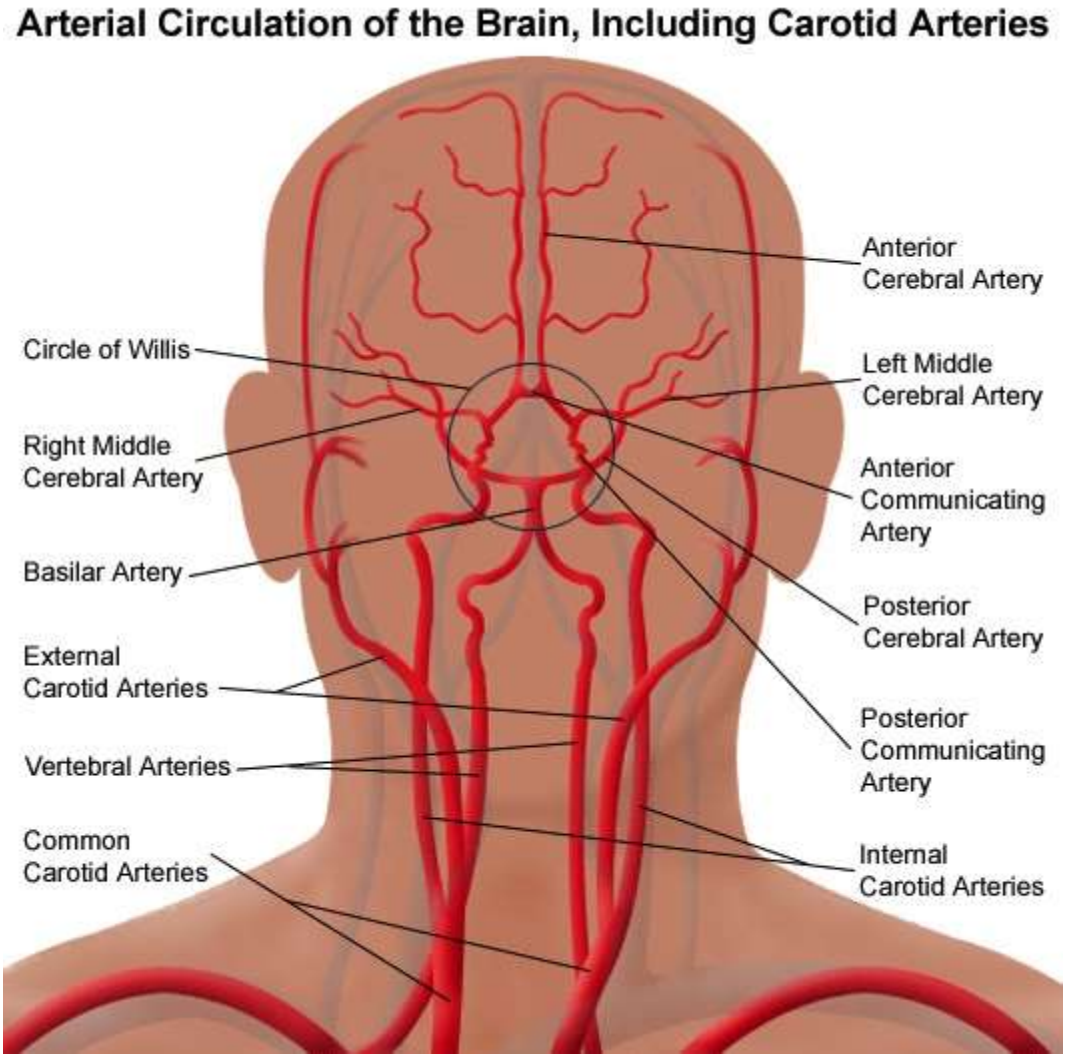
LACUNAR STROKE:

Defined as infarction following atherothrombotic or lipohyalinotic occlusion of a small artery in the brain. The term small vessel stroke denotes occlusion of a small penetrating artery. The size of the artery is 30 to 300 μm branches that penetrate the deep gray and white matter of the cerebrum or brainstem. The infarcts size is range from 3 mm to 2 cm in diameter. Hypertension and age are the principal risk factors. (7)

ANATOMY

The Brain is one of the largest and most complex organs in the human body. It is made up of more than 100 billion nerves that communicate in trillions of connections called synapses.(8) The central nervous system consists of the brain and the spinal cord. The peripheral nervous system consists of the extensions of neural structures beyond the central nervous system and includes somatic and autonomic divisions.(9) At rest the brain, which is only 2% of total body weight, receives 20% of the cardiac output and consumes about 20 %of the total inspired oxygen .(10)

The principal arterial inflow to the brain in humans is via four arteries: two internal carotids arteries and two vertebral arteries. In humans, the carotid arteries are quantitatively the most significant. The vertebral arteries unite to form the basilar artery and the basilar artery and the carotids form the circle of Willis below the hypothalamus. The circle of Willis is the origin of the six large vessels supplying the cerebral cortex.(11)



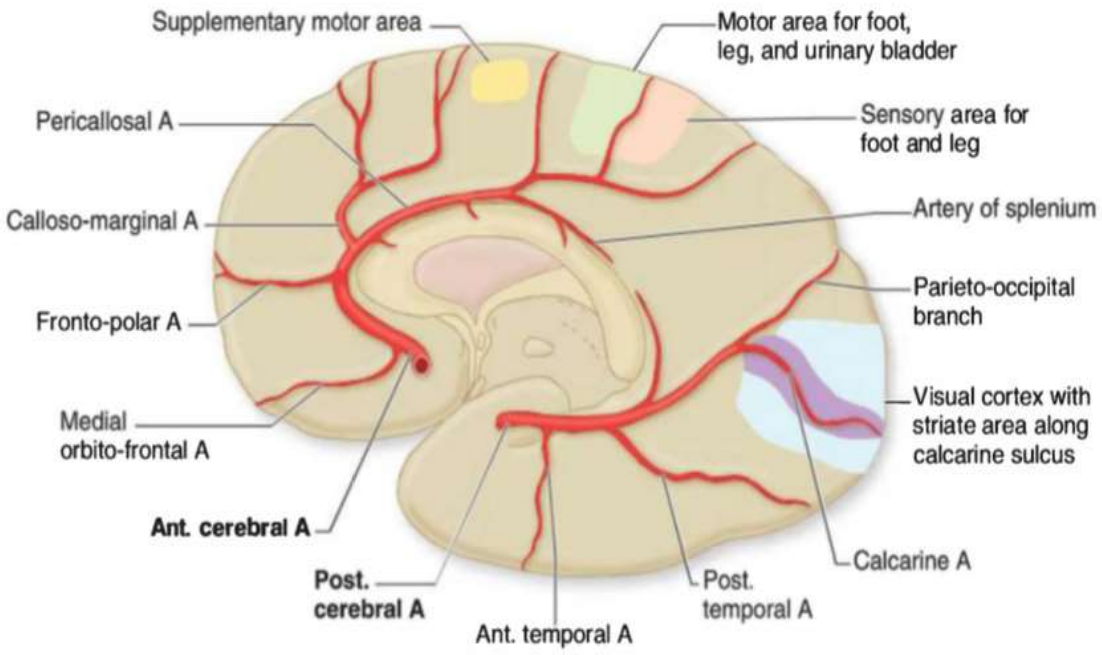
The internal carotid artery is a terminal branch of the common carotid artery. It arises most frequently between C3 and C5 vertebral level, where the common carotid bifurcates to form the internal carotid and the external carotid artery .(12). Internal carotid artery divides into seven segments which was described by Bouthillier et al in 1996. There are seven segments in the Bouthillier classification: 1)cervical segment, 2) petrous (horizontal) segment,3) lacerum segment, 4)cavernous segment, 5) clinoid segment, 6)ophthalmic (supraclinoid) segment,7)communicating (terminal) segment.(12)

The Posterior communicating artery (PCOM) originates from the posterior aspect of the C7 segment of the internal carotid artery and extends postero-medially to anastomose with the ipsilateral posterior cerebral artery and form part of the circle of Willis. It makes up posterior linkage in the circle of Willis. The PCOM gives off many fine, scarcely visible, perforating branches. The largest perforating branch is called the premamillary or anterior thalamoperforating artery. Tiny branches supply the adjacent optic chiasm, optic tract, hypothalamus, and midbrain.(14)

The anterior cerebral artery along with the middle cerebral artery forms at the termination of the internal carotid artery. It divides into two major branches;

pericallosal and callosomarginal arteries .It supplies the medial aspect of the cerebral hemispheres back to the parietal lobe.(16)

The MCA originates at the bifurcation of the ICA as the larger and more direct branch. It originates lateral to the optic chiasma. The M1 segment is the sphenoidal segment, M2 segment is referred to as the insular segment. M3 segment called the opercular segment and M4 as the cortical segment.(17)



The middle cerebral arteries supply the majority of the lateral surface of the hemisphere, except the superior portion of the parietal lobe where it supplied by ACA and the inferior portion of the temporal lobe and occipital lobe where it is

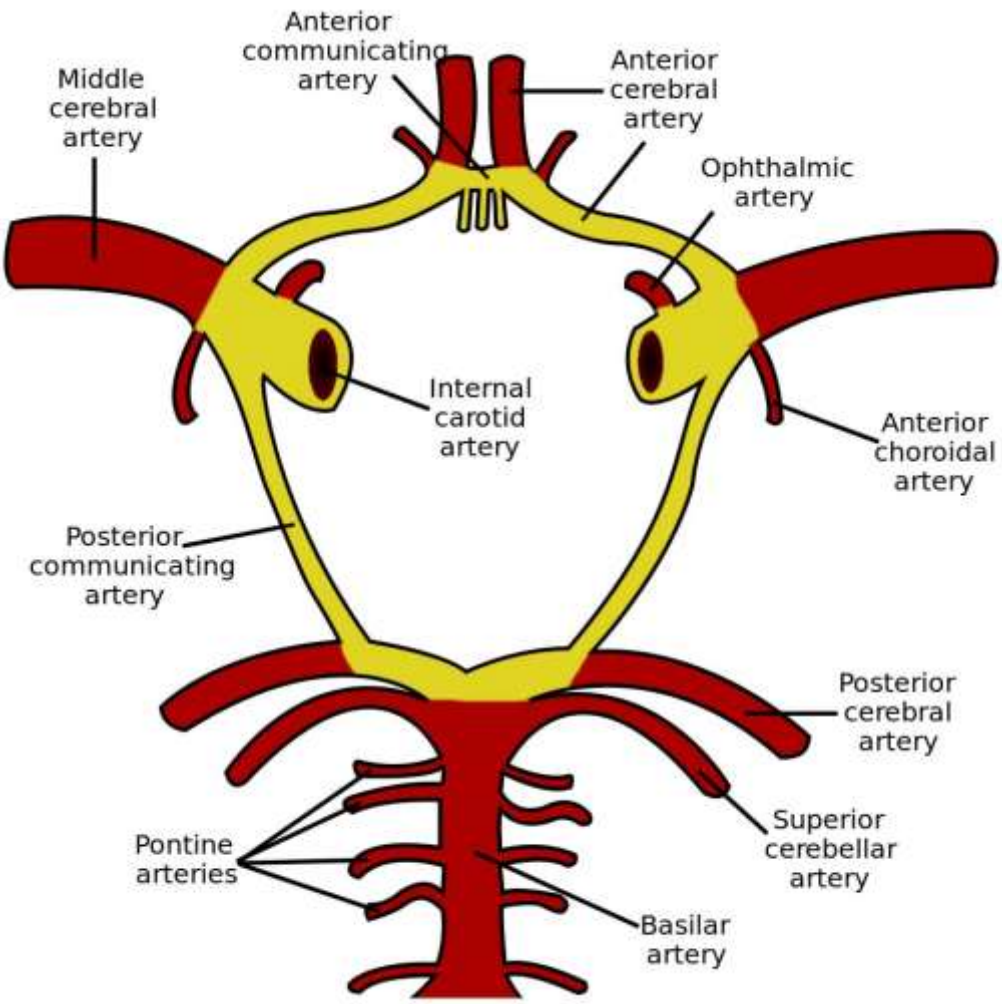
supplied by PCA. In addition, they supply part of the internal capsule and basal ganglia.(18)

The vertebral artery arises from the proximal subclavian artery ascends to pass through the transverse foramina of the sixth to second cervical vertebrae, giving off small muscular branches on the way. It enters the skull through the foramen magnum. It unites with the opposite vertebral artery on the ventral surface of the brainstem at the pontomedullary junction to form the basilar artery.(19)

The basilar artery is part of the posterior cerebral circulation. It arises from the confluence of the left and right vertebral arteries at the base of the pons. The basilar artery runs cranially in the central groove of the pons called basilar groove towards the midbrain within the pontine cistern. It terminates by dividing into two posterior cerebral arteries at the upper pons. Along the course of the basilar artery it gives off paired branches that are anterior inferior cerebellar artery, pontine artery, superior cerebellar artery and sometimes labyrinthine artery.(20)

The posterior cerebral arteries (PCA) are the terminal branches of the basilar artery and supply the occipital lobes and posteromedial temporal lobes. Posterior communicating artery,medial and lateral posterior choroidal arteries, lateral posterior choroidal arteries, calcarine artery these are main branches of posterior cerebral artery.(21)

CIRCLE OF WILLIS



The circle of Willis also called circulus arteriosus cerebri is an anastomotic system of arteries that sits at the base of the brain. The “circle” was named after Thomas Willis by his student Richard Lowe. The circle of Willis encircles the stalk of the pituitary gland and provides important communications between the blood supply of the forebrain and hindbrain .The circle of Willis is formed when the internal carotid artery enters the cranial cavity bilaterally and divides into the

anterior cerebral artery and middle cerebral artery . The anterior cerebral arteries are then united by an anterior communicating artery. These connections form the anterior half (anterior circulation) of the circle of Willis. Posteriorly, the basilar artery, formed by the left and right vertebral arteries, branches into a left and right posterior cerebral artery , forming the posterior circulation. The PCAs complete the circle of Willis by joining the internal carotid system anteriorly via the posterior communicating arteries.(22)

The arrangement of the brain's arteries into the circle of Willis creates collaterals in the cerebral circulation. If one part of the circle becomes blocked or one of the arteries supplying the circle is blocked or narrowed, blood flow from the other blood vessels can preserve the cerebral perfusion. The anterior communicating artery and posterior communicating arteries of the circle of Willis provide the main route for collateral blood flow in cases of carotid artery obstruction.(23)

RISK FACTORS FOR STROKE

Risk factors: Stroke risk factors have been classified as modifiable, non-modifiable, traditional and novel. Stroke risk varies according to differences in these factors.

Modifiable risk factors:

1. Hypertension: Hypertension is the most prevalent modifiable risk factor for stroke .The prevalence of hypertension increases with age. The Framingham Heart Study investigators reported the lifetime risk of hypertension to be approximately 90% for men and women who were non-hypertensive at age 55 or 65 years and survived to ages 80–85 years old (Vasan, Beiser et al. 2002). A meta-analysis of one million adults enrolled in 61 observational studies concluded that death from ischemic heart disease and stroke increases progressively and linearly with systolic blood pressure levels as low as 115 mm Hg and diastolic 75 mm Hg upward. This study also found that for every 20 mm Hg systolic or 10 mm Hg diastolic increase in blood pressure there is a doubling of mortality from both ischemic heart disease and stroke.

2. Diabetes mellitus: Persons with either type I or type II diabetes mellitus have an increased susceptibility for large artery atherosclerosis and small artery occlusive disease. Diabetes mellitus also leads to renal or cardiac disease, which indirectly promote arterial hypertension and stroke. Incidence of cerebrovascular disease in diabetic men was reported to be twice as that of non-diabetics and almost three times greater in diabetic women in the Framingham Study. It is postulated that excessive glycation and oxidation, endothelial dysfunction and increased platelet aggregation may be responsible for endothelial proliferation and thickening of

plasmatic membrane in small blood vessels ('lipohyalinosis') leading to lacunar infarction. (25)

Diabetes also increases the levels of fibrinogen and clotting factors, increase platelet aggregation, which in turn promotes arterial thrombosis. When compared to non-diabetic persons, the risk of stroke increases in proportion to increases in levels of blood glucose especially in young adults. A history of diabetes is associated with an approximately 11 times increase in the risk of stroke in persons aged 15 to 55 years. Diabetic patients with stroke are typically younger than patients with stroke who do not have diabetes mellitus.(26)

3. Dyslipidemia:

Abnormal lipid profile is an established risk factor for cerebrovascular accident. Most epidemiologic studies find no consistent relationship between cholesterol levels and overall stroke risk. Some studies, however, have found a positive relationship between total and low density lipoprotein cholesterol levels and the risk of ischemic stroke. Increased high density lipoprotein cholesterol levels are associated with reduced risk of ischemic stroke in men and women, in the elderly, and among different racial and ethnic groups. These data add to the evidence relating lipids to stroke and support HDL cholesterol as an important modifiable stroke-risk factor. Elevated levels of triglycerides are one of the

important components of the metabolic syndrome, a modifiable risk factor for stroke. The etiologic fraction estimates suggest that elimination of the metabolic syndrome would result in a 19% reduction in overall stroke, a 30% reduction of stroke in women, and a 35% reduction of stroke among Hispanics.(24)

4. Obesity:

Obesity has been described as "the great humbler". With the exception of bariatric surgery, no major treatment breakthroughs have emerged despite decades of scientific inquiry. Obesity is known to affect the overall health of a population. According to the World Health Organization, overweight and obesity are the fifth leading risk for global deaths. Four important themes emerge from research on the association between obesity and stroke risk. First, compared with BMI, measures of central obesity are better predictors of stroke in most but not every study. Second, the relative risk for stroke associated with obesity seems to be higher for middle-aged compared with older individuals. Third, the association is true for both ischemic and intraparenchymal hemorrhage, but it is more consistently demonstrated for the former. Finally the association between obesity and increased risk for stroke is substantially explained by hypercholesterolemia, hypertension, and diabetes mellitus.(24)(27)

5. Smoking:

Cigarette smoking is a well recognized and modifiable risk factor for ischemic and hemorrhagic stroke. In the U.S., nearly 25% of adults are affected by cigarette smoking. Smoking is an independent stroke risk factor, increasing the risk of stroke by about 50%.Passive smoking also increases the risk of ischemic stroke.(24). A meta analysis of 32 studies estimated a twofold increased risk of ischemic stroke for smokers versus nonsmokers and a threefold increased risk for subarachnoid hemorrhage.(28)

Cigarette smoking is a potent risk factor for advanced atherosclerosis, myocardial infarction and ischemic stroke. Even environmental tobacco smoke may convey some risk. It also increases hemoglobin, serum fibrinogen, platelet aggregation, LDL cholesterol, and the hematocrit as it decreases HDL cholesterol levels. Smoking promotes the progression of atherosclerotic plaques of the carotid artery.

6. Alcohol:

Excessive alcohol consumption is an important, unrecognized independent risk factor for cerebrovascular disease in men. Many cohort and case-control studies have found an increased risk for hemorrhagic and ischemic strokes to be related to heavy alcohol consumption. The association of alcohol consumption with hemorrhagic stroke is more linear than its association with ischemic stroke.

Alcohol consumption increases the risk of both intracerebral (ICH) and subarachnoid (SAH) hemorrhages. Increased risk for ICH due to alcohol consumption has been attributed to alcohol-induced hypertension and impaired hemostasis. A transient increase in blood pressure during heavy alcohol intake and withdrawal may contribute to the rupture of small cerebral arteries or to an aneurysm, suggesting that episodic heavy drinking is a particular risk factor for SAH. Excessive chronic alcohol consumption is associated with cardiomyopathy, which is known to predispose to cardiac arrhythmias and heart failure, both of which increase the risk for embolic stroke.(29)

7. Atrial fibrillation:

Atrial fibrillation (AF) occurs in epidemic proportion and is now recognized to occur in about 2 % of the general population. Its prevalence is age related about 10 % of 80 year old have this arrhythmia with hypertension, valvular disease and heart failure being the most frequent underlying conditions. Up to 10 % of cases of AF may be idiopathic, although genetic, autonomic, inflammatory, infective and toxic causes may account for many of these. Thromboembolic stroke occurs in about 5 % of AF patients each year. AF related to thromboembolic stroke accounts for 15–20 % of all strokes.(28)(30)

8. Other heart disease:

Stroke or systemic embolism is less common among uncomplicated MI patients but can occur in up to 12% of patients with acute MI complicated by a Left ventricle thrombus. The rate is higher in those with anterior than inferior infarcts and may reach 20% of those with the large anteroapical infarcts. Cardiomyopathy due to ischemic or genetic causes leads Left ventricle systolic function is impaired, the reduced stroke volume creates a condition of relative stasis within the left ventricle that may activate coagulation processes and increase the risk of thromboembolic event. Antithrombotic therapy can reduce, but not eliminate, the likelihood of stroke and systemic embolism in patients with valvular heart disease.(31)

9. Physical activity:

In recent era because of industrialization human life style entirely changed towards sedentary life. Substantial evidence exists that physical activity exerts a beneficial effect on multiple cardiovascular disease risk factors, including those for stroke. Moderately active men and women had a 20% lower risk, and those who were highly active had a 27% lower risk.(31)

10. Carotid atherosclerosis:

Carotid atherosclerosis produces an estimated 10% of ischemic stroke. Carotid disease can be classified by whether stenosis is symptomatic or asymptomatic and by the degree of stenosis. Greater degrees of arterial narrowing are generally associated with a greater risk of stroke, except that those with near occlusions or at lower risk of stroke. Dissection of internal carotid or vertebral arteries or even beyond the circle of Willis is a common source of embolic stroke in young patients.

11. Drug abuse:

Drug abuse is a significant cause of stroke, especially in young adults and adolescent. Heroin, Amphetamine, Methylphenidate, Cocaine, Phencyclidine, lysergic acid diethylamide these are commonly abusing drugs cause stroke either ischemic or hemorrghic stroke. Possible mechanisms incorporate in the pathogenesis of stroke includes endocarditis with secondary central nervous system vessel changes, direct toxic injury to vessels, embolization of foreign material, pharmacological alteration of vascular function, and immunogenic vascular injury that is vasculitis.(32)

12. Post menopausal use of estrogens:

Strokes are an important cause of disability and death among older women. Based on observational studies, the effect of postmenopausal hormone therapy on the risk of stroke is uncertain. Recent case control studies and cohort studies have reported that postmenopausal hormone therapy increases, decreases, or has no significant effect on stroke risk.(33)

13. Stroke in pregnancy and postpartum period:

There is an increased incidence of cerebrovascular events during pregnancy and the postpartum period. The risk of both cerebral infarction and intracerebral hemorrhage appears to be mainly in the 6 week period after delivery rather than during the pregnancy itself. Carotid artery dissection may also be encountered late in pregnancy or soon after delivery. The occurrence of paradoxical embolus is always a consideration in pregnancy because of a tendency to form clots in the pelvic and leg veins, coupled with increased right heart pressures. Amniotic fluid embolus may rarely cause stroke in this manner and should be suspected in multiparous women who have had uterine tears; there are almost invariably signs of acute pulmonary disease from simultaneous occlusion of lung vessels. A rare peripartum cardiomyopathy is yet another source of embolic stroke.(34)

14. Stroke after cardiac surgery:

Neurologic complications are second only to heart failure as a cause of morbidity and mortality following cardiac surgery, and the presence of neurologic sequelae significantly increases the likelihood of requiring long term care. Women are at higher risk to suffer early and delayed perioperative strokes. The frequency of this complication is reported to be as high as 5% in patients undergoing CABG surgery, almost 9% in CABG patients' ≥75 years of age, and nearly 16% in patients undergoing valve surgery or those with preexisting cerebrovascular disease.

II. Non modifiable risk factors:

Age: It is assumed that the average age of patients with stroke in developing countries is usually 15 years younger than those in developed countries. In India, nearly one-fifth of patients with first-ever stroke admitted to hospitals has been estimated to be aged 40 years or less. But the Mumbai.(37) and Trivandrum.(38) registries showed that the mean age of patients with stroke was 66 and 67 years respectively. In contrast, in the Bangalore study the mean age was 54.5 years.(39)

Gender: There is growing recognition of the clinical and public health importance of stroke in women. Although age-specific stroke incidence and mortality rates are higher in men than in women, stroke affects a greater of women because of their increased longevity and the fact that stroke event rates increase substantially in the oldest age group. Moreover, stroke related outcomes, including disability and quality of life, are consistently poorer in women than in men. Migraine is an independent risk factor for stroke. The risk of stroke associated with migraine was even higher in women under the age of 45 years and in women who used oral contraceptives.(40)

Race/Ethnicity: Stroke affects all ethnic groups and races in the world. No geographic region of the world is protected from ischemic stroke. Blacks and some Hispanic Americans have high stroke incidence and mortality rates compared with whites. Possible reasons for the high incidence and mortality rate of strokes in blacks include a higher prevalence of hypertension, obesity, and diabetes mellitus within the black population. Epidemiological studies have also shown an increase in stroke incidence among self-identified Hispanic populations. Chinese and Japanese populations generally have high stroke incidence rates as well.(36)

Family History: Both paternal and maternal history of stroke may be associated with increased risk of stroke. This increased risk could be mediated through a variety of mechanisms, including genetic heritability of stroke risk factors, the inheritance of susceptibility to the effects of such risk factors, familial sharing of cultural, environmental and lifestyle factors, and the interaction between genetic and environmental factors. Studies with twins provide strong data suggesting familial inheritance of stroke.(36)

Other less common causes: Hypercoagulable disorders, temporal arteritis, necrotizing arteritis, drugs like amphetamines and cocaine, leukaoariosis, cerebral autosomal dominant arteriopathy with subcortical infarcts and leukoencephalopathy etc.

PATHOPHYSIOLOGY

The brain requires a constant supply of substrate to maintain normal function, oxygen and glucose are the two most important substrates utilized in normal cerebral metabolism. At a given time brain contains about 7 ml of oxygen which at normal rates of utilization can last up to 10 seconds. Since there are virtually no reserves of oxygen in the brain, a total lack of oxygen leads to

reversible cerebral impairment in the initial stages but irreversible lesions follow within 6 to 8 minutes unless the oxygen supply is promptly restored.(11)

Acute occlusion an intracranial vessel causes reduction in blood flow to the region it supplies. Cerebral infarction basically comprises two pathophysiologic processes: 1) Loss of the supply of oxygen and glucose secondary to vascular occlusion, 2) Sequence of changes in cellular metabolism consequent to the collapse of energy-producing processes, ultimately with disintegration of cell structures and their membranes, a process called necrosis.(41)

At the center of an ischemic stroke is a zone of infarction. The necrotic tissue swells rapidly, mainly because of excessive intracellular water content called cytotoxic edema. Anoxia also causes necrosis and swelling of cerebral tissue.Baesd on this oxygen lack must be a factor common to both infarction and anoxic encephalopathy. The effects of ischemia, whether functional and reversible or structural and irreversible, depend on its degree and duration. The margins of the infarct are hyperemic, being supplied by meningeal collaterals, and here there is only minimal or no parenchymal damage.(41)

Tissue surrounding the core region of infarction is ischemic but reversibly dysfunctional and referred to as the ischemic penumbra. Penumbra zone that is marginally perfused and contains at risk but viable neurons. The neurons in the penumbra are considered to be physiologically "stunned" by moderate ischemia and subject to salvage if blood flow is restored in a certain period of time.(41)

Critical threshold of cerebral blood flow, below this level functional impairment occurs. In several animal species, including macaque monkeys and gerbils, the critical level was 23ml/100g/min (normal is 55); if, after a short period of time, CBF is restored to higher levels, the impairment of function can be reversed. Reduction of CBF below 10 to12ml/100g/min causes infarction, almost regardless of its duration. The critical level of hypoperfusion that abolishes function and leads to tissue damage is therefore a CBF between 12 and 23ml/100g/min. At these levels of blood flow the electroencephalogram is slowed, and below this level it becomes isoelectric.(41)

In the region of marginal perfusion, efflux potassium from the injured cell leads potassium rises. Adenosine triphosphate and creatine phosphate are depleted. These biochemical abnormalities are reversible if the circulation is quickly restored to normal. Cerebral blood flow of 6 to 8ml/100gm/min causes marked ATP

depletion, increase in extracellular K, increase in intracellular Ca, and cellular acidosis, invariably leading to histologic signs of necrosis. Free fatty acids are activated and destroy the phospholipids of neuronal membranes. Prostaglandins, leukotrienes, and free radicals accumulate, and intracellular proteins and enzymes are denatured. Cells then swell, a process called cellular or cytotoxic edema.(41)

Body temperature is yet another important factor in determining the extent of infarction. A reduction of even 2 to 3°C reduces the metabolic requirements of neurons and increases their tolerance to hypoxia by 25 to 30%.Role of excitatory neurotransmitters in stroke, particularly glutamate and aspartate, which are formed from glycolytic intermediates of the Krebs cycle. These neurotransmitters, released by ischemic cells, excite neurons and produce an intracellular influx of Na and Ca. These changes are in part responsible for irreversible cell injury.(41)

The magnitude of flow reduction is a function of collateral blood flow and this depends on individual vascular anatomy, the site of occlusion, and likely systemic blood pressure. A decrease in cerebral blood flow to zero causes death of brain tissue within 4 to 10 minutes. If blood flow restored prior to a significant amount of cell death, the patient may experience only transient symptoms, and clinical syndrome called TIA.(42)

CLINICAL FEATURES:

A typical ischemic stroke presents with the abrupt onset of a focal neurological deficit and is characterized clinically by mode of onset and subsequent course. The symptoms and signs of an ischemic stroke vary depending on the location of the occlusion and the extent of collateral flow. Virtually any symptom of brain dysfunction may occur. However, the abrupt onset of a hemiparesis in an individual in atherosclerotic age groups is the hallmark. Symptoms and signs of carotid system disease most commonly affect the distribution of the middle cerebral artery and the patient may exhibit a contralateral hemiparesis, hemi sensory deficit and hemianopia. If the dominant hemisphere is involved, there is usually some degree of aphasia. Either anterior (carotid) or posterior (vertebro basilar) circulation may be involved.

The more extensive the area of brain affected, the more functions that are likely to be lost. These are pure motor hemiparesis of limbs and face, pure sensory, reduction in sensory or vibratory sensation, aphasia, confusion, memory disturbance, visual disturbances in one or both eyes , dizziness or loss of balance and coordination, and headache, nausea, vomiting, altered smell, taste, hearing, or vision (total or partial), in cranial nerve involvement vision, smell, ocular movements , hearing etc… In cerebellar involvement features like ataxia,

nystagmus, incoordination, gait abnormality, intention tremor in some cases cerebellar infarction with edema can lead to sudden respiratory arrest due to raised intra cranial pressure in the posterior fossa. Hemorrhagic stroke present as sudden loss of consciousness, seizures.

MCA Stroke syndromes:

MCA Stem (M1) Occlusion Syndrome:

The clinical picture of total occlusion of the stem is one of contralateral hemiplegia (involving the face, arm, and leg as a result of infarction of the posterior limb of the internal capsule), hemi-anaesthesia, and homonymous hemianopia (because of infarction of the lateral geniculate body), with deviation of the head and eyes toward the side of the lesion. There is a variable but usually global aphasia with left hemispheric lesions and anosognosia and amorphosynthesis with right sided lesions.(43)

MCA branch syndrome:

Major infarction in the territory of the superior division causes a dense sensorimotor deficit in the contralateral face, arm, but, to a lesser extent the leg, as well as ipsilateral deviation of the head and eyes. With left-sided lesions there is initially a global aphasia, which changes to a predominantly nonfluent (Broca's)

aphasia, with the emergence of an effortful, hesitant, grammatically simplified, and dysmelodic speech.(43)

Anterior Cerebral Artery Stroke Syndromes:

Occlusion of the stem of the anterior cerebral artery, proximal to its connection with the anterior communicating artery is usually well tolerated, because adequate collateral flow is provided by the anterior or cerebral artery of the opposite side. Complete infarction is a result of occlusion of one anterior cerebral artery distal to the anterior communicating artery results in a sensorimotor deficit of the opposite foot and leg and, to a lesser degree, of the shoulder and arm, with sparing of the hand and face. With occlusion of penetrating branches of the ACA, the anterior limb of the internal capsule and caudate is usually involved. With occlusion of penetrating branches of the ACA, the anterior limb of the internal capsule and caudate is usually involved.(43)

Posterior Cerebral Artery Stroke Syndromes:

Occlusion of the posterior cerebral artery produces a greater variety of clinical effects than occlusion of any other artery because both the upper brainstem, which is replete with important structures and the inferomedial parts of the temporal and occipital lobes lie within its supply.

The thalamic syndrome of Dejerine and Roussy follows infarction of the sensory relay nuclei in the thalamus, the result of occlusion of thalamogeniculate branches. There is both a deep and cutaneous sensory loss, usually severe in degree, of the opposite side of the body, including the trunk and face, sometimes accompanied by a transitory hemiparesis. A homonymous hemianopia may be conjoined.(43)

Central midbrain and subthalamic syndromes are a result of occlusion of the interpeduncular branches of the posterior cerebral artery. The clinical syndromes include palsies of vertical gaze, stupor, or coma. Syndromes of the paramedian arteries as follows.(43)

a) Weber syndrome: Third-nerve palsy combined with contralateral hemiplegia

b) Claude syndrome: Third nerve palsy with contralateral ataxic tremor

c) Benedikt syndrome: Oculomotor palsy with contralateral cerebellar ataxia, tremor, and corti-cospinal signs may have choreoathetosis

Vertebral Artery Stroke Syndromes :

Medial medullary syndrome: Occlusion of the vertebral artery or one of its medial branches produces an infarct that involves the medullary pyramid, the medial lemniscus, and the emergent hypoglossal fibers leads to Contralateral paralysis of

arm and leg with sparing of the face, contralateral loss of position and vibration sense, and ipsilateral paralysis and later atrophy of the tongue.

Lateral Medullary Syndrome: Known as Wallenberg syndrome. It is produced by infarction of a wedge of lateral medulla lying posterior to the inferior olivary nucleus. It is due to occlusion of the vertebral artery or posterior inferior cerebellar artery. Clinical features follows as

a) symptoms derived from the vestibular nuclei-vertigo, nystagmus, oscillopsia, vomiting.

b) spinothalamic tract-contralateral or, less often, ipsilateral impairment of pain and thermal sense over half the body.

c) descending sympathetic tract-ipsilateral Horner syndrome-miosis, ptosis, decreased sweating.

d)fibers of the ninth and tenth nerves-hoarseness, dysphagia, hiccough, ipsilateral paralysis of the palate and vocal cord, diminished gag reflex.

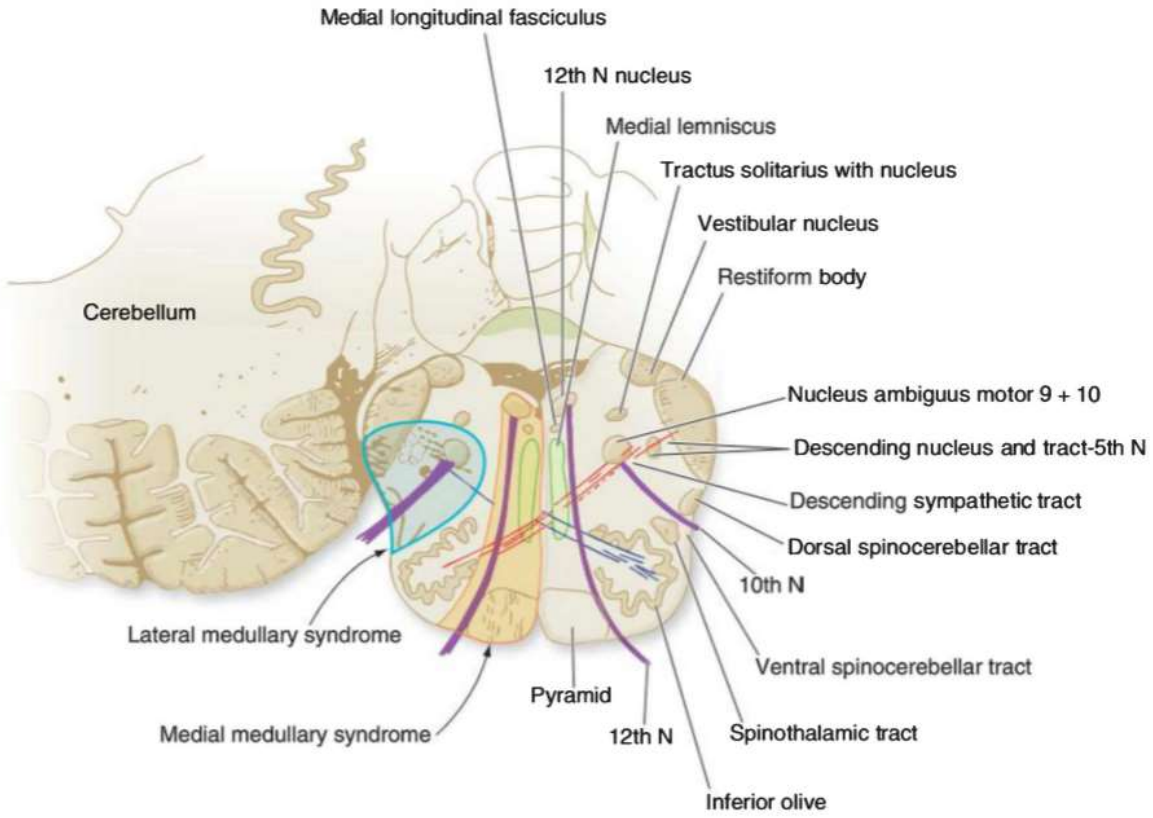
e) utricular nucleus-vertical diplopia and illusion of tilting of vision and rotation of the vertical meridian, rarely so severe as to produce upside down vision.

f) Olivocerebellar, spinocerebellar fibers, restiform body and inferior cerebellum-ipsilateral ataxia of limbs, falling or toppling to the ipsilateral side, and the sensation of lateropulsion.

f) descending tract and nucleus of the fifth nerve-pain, burning, and impaired sensation over ipsilateral half of the face.

g) nucleus and tractus solitaries-loss of taste..

h) cuneate and gracile nuclei-numbness of ipsilateral limbs.



DIAGNOSIS:

Diagnosis of stroke is based on clinical history and physical examination with relevant investigations.

Clinical History and Physical Examination

This involves history of presenting illness, as well as past illness and the family history. The intake of recreational drugs (alcohol, tobacco, others), and history of usage of oral contraceptives is also important. The physical examination can localize the arterial territory involved and help in advocating relevant investigations like MRI for posterior circulation ischemic stroke because posterior circulation better appreciated in MRI than CT.

Investigations mainly include base line investigations and specific investigations.

Base line investigations:

Investigation	Condition
Complete blood count	Anaemia,polycythemia,lukemias,thrombocytopenia Infections
Blood glucose level	DM, stroke mimic like hyperglycemia or hypoglycemia
Lipid profile	Dyslipidemia
Renal function test	CKD
Electrolytes	Hyponatremia,hypernatreimia
ESR	Connective tissue disorders like vasculitis
ECG	Myocardial infarction,atrial fibrillation,arrythemias
Echo	LV function, LV thrombus

Specific investigations:

Non-enhanced CT:

CT is initial investigation of choice in suspected stroke patients. Primary aim is to diffentiate between ischemic and hemorrhagic stroke. CT is highly sensitive for the depiction of hemorrhagic lesions, and the key role of non-enhanced CT is the detection of hemorrhage or other possible mimics of stroke (like tumors, extradural hematomas, abscesses, arteriovenous malformation) that could be the cause of the neurologic deficit.

The subset of ischemic stroke can be divided into hyperacute, acute, subacute and chronic stroke based on timing from the onset of stroke symptoms, which is the first 6 hours, 6 to 48 hours, 48hr to weeks, and weeks to months respectively. The CT findings during the first 3 to 6 hour (hyperacute) include loss of gray-white matter differentiation of cortical gyrus, basal ganglia or insula; loss of cortical sulci or narrowing of the Sylvian fissure; compression of ventricular system and basal cisterns; area of hypodensity; and hyperdensity in a circle of Willis vessel.(44)

The earliest CT sign visible is a hyperdense segment of a vessel,

representing direct visualisation of the intravascular thrombus/embolus and as such is visible immediately , it is most often observed in the middle cerebral artery called hyperdense middle cerebral artery sign.(45)

In subacute stage 48hrs to weeks phenomena called CT fogging effect' where hypodensed infarcted area disappear, becoming isodense. This is probably dues to resolution of edema in the infarcted area. This usually occurs between 2 to 6 weeks after the onset of stroke. In addition to that, there is also a risk of hemorrhagic transformation in 15 to 20% of the cases during this period of time. Most of the time, this occurred within 4to 6 days after onset of stroke. Once happened, the hyperdensity CT image may persist up to 8 to 10weeks.In chronic stage weeks to months the damaged necrotic tissue is resorbed. This results in formation of encephalomalacia accompanied by gliosis of adjacent brain tissue.(44,46)

Magnetic Resonance Imaging:

Magnetic Resonance Imaging (MRI) has emerged as an invaluable tool for the care of stroke patients, whereas, diffusion weighted MRI imaging is more sensitive for the detection of hyper acute ischemia. Conventional brain MRI studies can take up to one hour to complete. The study is not very good at detecting

cytotoxic or intracellular edema that is seen in the acute or less than 24 hour phase of stroke MRI may have particular utility in patients with TIA, because it is also more likely to identify new infarction, which is a strong predictor of subsequent stroke.(46)

TREATMENT

The goal for the acute management of patients with stroke is to stabilize the patient and to complete initial clinical evaluation and assessment, including imaging and laboratory studies.

GENERAL SUPPORTIVE CARE

- Airway, Ventilatory Support, and Supplemental Oxygen

Stroke is a primary failure of focal tissue oxygenation and energy supply. Systemic hypoxemia and hypotension should be avoided and, if present, corrected to limit further cellular damage.

- Hyperthermia:

Approximately one third of patients admitted with stroke will be hyperthermic (temperature >37.6°C) within the first hours after stroke onset. In the setting of acute ischemic stroke, hyperthermia is associated with poor neurological outcome, possibly secondary to increased metabolic demands,

enhanced release of neurotransmitters, and increased free radical production. Sources of hyperthermia (temperature >38°C) should be identified and treated, and antipyretic medications should be administered to lower temperature in hyperthermic patients with stroke.(48)

- Hypothermia:

Hypothermia has been shown to be neuroprotective in experimental and focal hypoxic brain injury models. Hypothermia may delay depletion of energy reserves, lessen intracellular acidosis, slow influx of calcium into ischemic cells, suppress production of oxygen free radicals, alter apoptotic signals, inhibit inflammation and cytokine production, and lessen the impact of excitatory amino acids. Mild to moderate hypothermia is associated with improved neurological outcomes among patients with cardiac arrest, which led to hypothermia becoming the first neuroprotective strategy to be recommended by the AHA in comatose patients after cardiac arrest.(48)

- Blood pressure management:

Elevated blood pressure is common during acute ischemic stroke. Extreme arterial hypertension is clearly detrimental, because it leads to

encephalopathy, cardiac complications, and renal insufficiency. In patients with markedly elevated blood pressure who do not receive fibrinolysis, a reasonable goal is to lower blood pressure by 15% during the first 24 hours after onset of stroke. The level of blood pressure that would mandate such treatment is not known, but consensus exists that medications should be withheld unless the systolic blood pressure is >220 mm Hg or the diastolic blood pressure is >120mmHg.(48)

- Blood Glucose:

Hypoglycemia during acute ischemic stroke is rare and likely related to antidiabetic medications. Severe or prolonged hypoglycemia can result in permanent brain damage. Hypoglycemia (blood glucose <60 mg/dL) should be treated in patients with acute ischemic stroke.

Persistent in hospital hyperglycemia during the first 24 hours after stroke is associated with worse outcomes than normoglycemia, and thus, it is reasonable to treat hyperglycemia to achieve blood glucose levels in a range of 140 to 180 mg/dL and to closely monitor to prevent hypoglycemia in patients with acute ischemic stroke.(48)

SPECIFIC MANAGEMENT:

Revascularization measures the optimal treatment for acute ischemic stroke is

reperfusion. To be successful, it must be achieved before ischemic area in brain is completely infarcted.

Thrombolysis:

The US FDA approved the use of intravenous rtPA in 1996. Its use is associated with improved outcomes for a broad spectrum of patients who can be treated within 3 hours of the last known well time before symptom onset and a mildly more selective spectrum of patients who can be treated between 3 and 4.5 hours of the last known well time. Most importantly, earlier treatment is more likely to result in a favorable outcome. Patients within 3 hours of onset with major strokes (NIHSS score >22) have a very poor prognosis, but some positive treatment effect with intravenous rtPA remains. Treatment with intravenous rtPA is associated with increased rates of intracranial hemorrhage, which may be fatal. Patient selection is important before thrombolysis.(48)(50)

THROMBOLYTIC THERAPY:

Indications:

i) Diagnosis of ischemic stroke causing measurable neurological deficit

ii) Onset of symptoms < 4.5 hours before beginning treatment

iii) Age $\geq$ 18years

iv) Consent by patient or relatives

Contraindications:

a) Cerebral imaging showing intracerebral hemorrhage

b) Cerebral imaging demonstrating large infarction, >1/3 territory of a cerebral hemisphere (CT hypodensity or djffusion restriction on MRI)

c) Head trauma within 3 months prior to stroke

d) History of intracranial hemorrhage

e) Elevated blood pressure; systolic > 185 mm Hg or diastolic >110 mm Hg that has not responded to medications

f) Active bleeding or arterial puncture at noncompressible site

g) Hematologic alterations :

Platelet count < 100,000/mm3

Heparin administered within 48 h resulting in a PTT above normal range

Current use of anticoagulation with INR > 1.7 or Prothrombin time >15 s

h) Blood glucose <50 mg/dl

Relative contraindications:

Minor or resolving stroke

Seizure at onset

Major surgery or trauma within 14 days

Gastrointestinal or urinary bleeding within 21 days

Myocardial infarction within 3 months

Dose administration and precautions:

Infuse rtPA @ 0.9 mg/kg (maximum dose 90 mg) over 60 minutes, with 10% of the dose given as a bolus over 1 minute. Measure blood pressure and perform neurological assessments every 15 minutes during and after IV rtPA infusion for 2 hours, then every 30 minutes for 6 hours, then hourly until 24 hours after IV rtPA treatment.

Obtain a follow-up CT or MRI scan at 24 hours after IV rtPA before starting anticoagulants or antiplatelet agents.

If the patient develops severe headache, acute hypertension, nausea, or vomiting or has a worsening neurological examination, discontinue the infusion (if IV rtPA is being administered) and obtain emergent CT scan.(48,51)

Antithrombotic management:

Aspirin is the only antiplatelet agent that has been proven effective for the acute treatment of Ischemic stroke. Antiplatelet agents are the mainstay for secondary prevention of non-cardioembolic stroke. Clopidogrel, a P2Y12-receptor antagonist, inhibits platelet aggregation synergistically with aspirin. The Clopidogrel in High-Risk Patients with Acute Nondisabling Cerebrovascular Events (CHANCE) study showed that aspirin plus clopidogrel is more effective than aspirin alone in

reducing the risk of subsequent stroke without increasing the risk of bleeding events in patients already having an episode of minor stroke or TIA.(52)

Hypothermia:

Hypothermia has been shown to improve neurological outcomes after global ischemia–hypoxia in comatose patients who have had cardiac arrest, and is one of the most extensively studied and powerful therapeutic strategies in acute ischemic stroke. The protective mechanisms of therapeutic hypothermia affect the ischemic cascade across several parallel pathways and, when coupled with reperfusion strategies, might yield synergistic benefits for patients who have had a stroke.(53)

Anti edema measures after ischemic stroke:

Acute cerebral infarction is often followed by a delayed deterioration caused by edema of the infarcted tissue. Although the cytotoxic edema normally peaks 3 to 4 days after injury, early reperfusion of a large volume of necrotic tissue can accelerate the edema to a potentially critical level within the first 24 hours, a circumstance termed malignant edema. In patients with severe stroke or posterior fossa infarctions, careful observation is required for early intervention to address potentially life-threatening edema. Hyperventilation, hypertonic saline, osmotic diuretics, intraventricular drainage of cerebrospinal fluid, and

decompressive surgery all these decrease raised intracranial pressure.(48)

General Supportive treatment:

Sustaining nutrition is important because dehydration or malnutrition may slow recovery. Dehydration is a potential cause of DVT after stroke. Impairments of swallowing are associated with a high risk of pneumonia. The patient may be placed on a strict nothing-by-mouth order until an assessment of the ability to swallow is completed. Most patients are treated initially with intravenous fluids. Nasogastric tube may be inserted to provide feedings and to facilitate administration of medications. Bowel management to avoid constipation and fecal impaction or diarrhea is also a component of ancillary care. Constipation occurs in 30 -60% of patients 4 weeks after stroke, and in patients with moderate stroke severity, constipation was associated with poor outcomes at 12 weeks.

Pneumonia, which is most likely to occur in seriously affected, immobile patients and those who are unable to cough, is an important cause of death after stroke. Stroke-associated pneumonia increases length of stay, mortality, and hospital costs. Immobility and atelectasis can lead to development of pneumonia. Early mobility and good pulmonary care can help prevent pneumonia.. Exercise

and encouragement to take deep breaths may help to lessen the development of atelectasis. Prophylactic use of antibiotics is not recommended to prevent pneumonia in stroke patients.(48)

SURGICAL MANAGEMENT OF ISCHEMIC STROKE:

Decompressive surgery :

Indicated mainly for posterior infarcts involving brainstem. Large infarction of the cerebellum occurs, delayed swelling commonly follows. Although the early symptoms may be limited to impaired function of the cerebellum, edema can cause brain stem compression and can progress very rapidly to a loss of brain stem function. Emergent posterior fossa decompression with partial removal of the infarcted tissue is often lifesaving and produces a clinical outcome with a reasonable quality of life.(48)

Treatment of Intracerebral hemorrhage (ICH):

Although about 40% of patients with a hypertensive ICH die, others have a good to complete recovery if they survive the initial hemorrhage. The ICH Score is a validated clinical grading scale that is useful for stratification of mortality risk and clinical outcome.(56)

Evacuation of supratentorial hematomas does not appear to improve

outcome for most patients. The International Surgical Trial in Intracerebral Haemorrhage (STICH) randomized patients with supratentorial ICH to either early surgical evacuation or initial medical management. No benefit was found in the early surgery arm, although analysis was complicated by the fact that 26% of patients in the initial medical management group ultimately had surgery for neurologic deterioration.(56)

MATERIALS AND METHODS

STUDY AREA: Stanley Medical College, Department Of General Medicine

STUDY POPULATION:

All the patients presenting with acute stroke during the study period, who met the inclusion criteria and also who were willing to participate in this study.

STUDY DESIGN: A Prospective, Descriptive Observational Study

SAMPLE SIZE: **200**

STUDY DURATION: April 2017 to September 2017

INCLUSION CRITERIA:

All patients presenting with signs and symptoms suggestive of new onset stroke above the age of 20 years, which was also confirmed by radiological studies.

EXCLUSION CRITERIA:

a) Patient below age of 20 yrs
b) Todd's palsy
c) Head injury
d) Infective etiology
e) Metastatic etiology
f) Recurrent stroke
g) Pre-existing severe physical or cognitive disability

METHODOLOGY:

The patients enrolled in the study were subjected to a detailed clinical history and physical examination. Clinical history was obtained from the relatives when the patient is having speech disturbances, altered sensorium and loss of consciousness. An analytical approach was adopted to assess the risk factors, clinico-radiological profile and the outcome in patients with acute cerebrovascular disease. Data collected from 200 selected subjects was internally compared and statistically analysed by using descriptive and inferential statistics based on the formulated objectives of the study.

The following investigations were carried out as routine :

- Complete blood count
- Renal function test
- Fasting and postprandial sugars
- Fasting lipid profile
- Urine examination
- Electrocardiogram
- 2D ECHO
- Computed tomography(CT brain)
- MRI brain (if required)

Hypertension: Patients who are already on antihypertensive medications were taken as hypertensive. Patients defined as hypertensive when SBP >140 mm of hg and DBP >90mm of hg.

Diabetes Mellitus: Patients who are already on oral hypoglycemic agents and insulin were consider as diabetics. Whose HBA1c ≥6.5% and fasting blood glucose ≥126mg/dl were defined as diabetes.

Dyslipidemia: Dyslipidemia was taken as serum cholesterol >200mg/dl, LDL cholesterol >130mg/dl and HDL cholesterol <35mg/dl in females and <40mg/dl in males

Obesity: Patients were considered obese if BMI ≥23kg/m^2 overweight and >25kg/m^2 as per WHO Asian /pacific guidelines.(59)

Heart disease: Patients who had h/o pre-existing coronary artery disease, congestive heart failure, atrial fibrillation or ECG , Echocardiography suggestive of CAD.

OUTCOME :

The outcome will be studied during the hospital stay or at the time of discharge. Outcome assessed by patient general condition and CNS examination as

1. Complete recovery (with no sequelae).
2. Partial recovery (moderate sequelae).
3. Death.

DATA COLLECTION :

Detailed history was obtained from the patients or the patient's attendants after getting an informed written consent. Detailed clinical examination and necessary investigations was done and entered in a pre-structured questionnaire. Patients were followed up till the time of discharge from the hospital and at the end of the study, the outcome of interest was measured from the data obtained using appropriate statistical methods.

STATISTICAL ANALYSIS:

Descriptive statistics was done for all data and suitable statistical tests of comparison were done. Continuous variables were analysed with the Unpaired t test / single factor ANOVA and categorical variables were analysed with chi squared test / Fisher Exact Test. Regression analysis done and odds ratio with confidence interval calculated. Statistical significance was taken as $P < 0.05$. The data was analysed using SPSS Version 16. Microsoft Excel 2010 was used to generate charts.

DATA COLLECTION FORM : Enclosed

CONFLICT OF INTEREST IF ANY - Nil

PRIVACY/CONFIDENTIALITY OF STUDY SUBJECTS - Maintained

DATA ANALYSIS

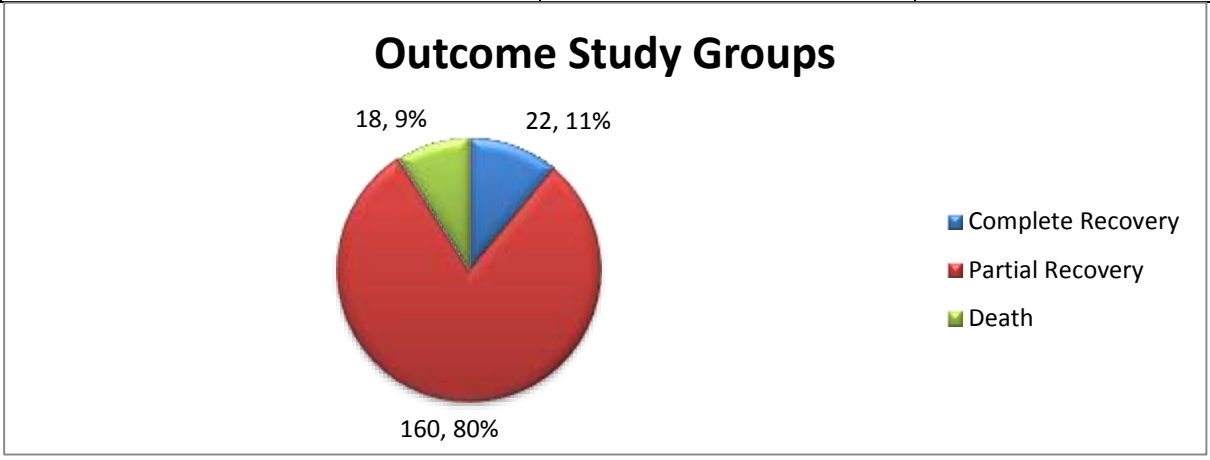
Present study was done in Stanley medical college hospital, Chennai-01 among the patients who were admitted in ICU and Medical wards during the period of April 2017 to September 2017, with stroke of various etiologies.

In this study, an analytical approach was adopted to assess the risk factors, clinico-radiological profile and the outcome & factors influencing outcome in patients with acute cerebrovascular disease.

Datas collected from the selected subjects were internally compared and statistically analysed by using descriptive and inferential statistics based on the formulated objectives of the study. A total of 200 patients were studied and the results were as given below.

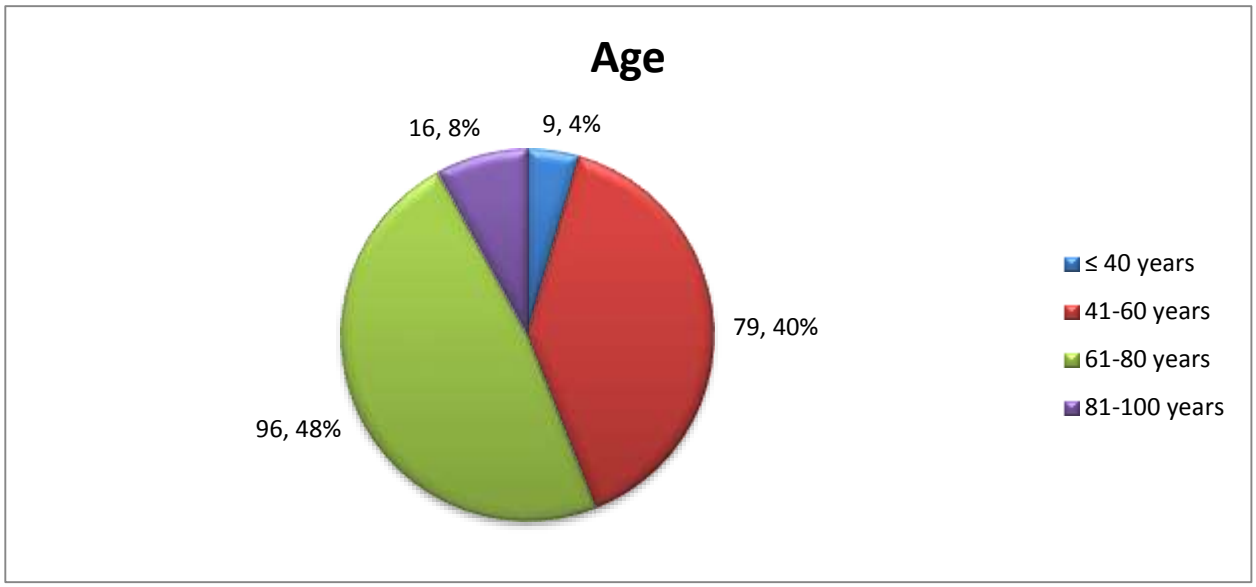
Outcome Study Groups

Outcome Study Groups	Number	(%)
Complete Recovery	**22**	**11.00**
Partial Recovery	**160**	**80.00**
Death	**18**	**9.00**
Total	**200**	**100.00**



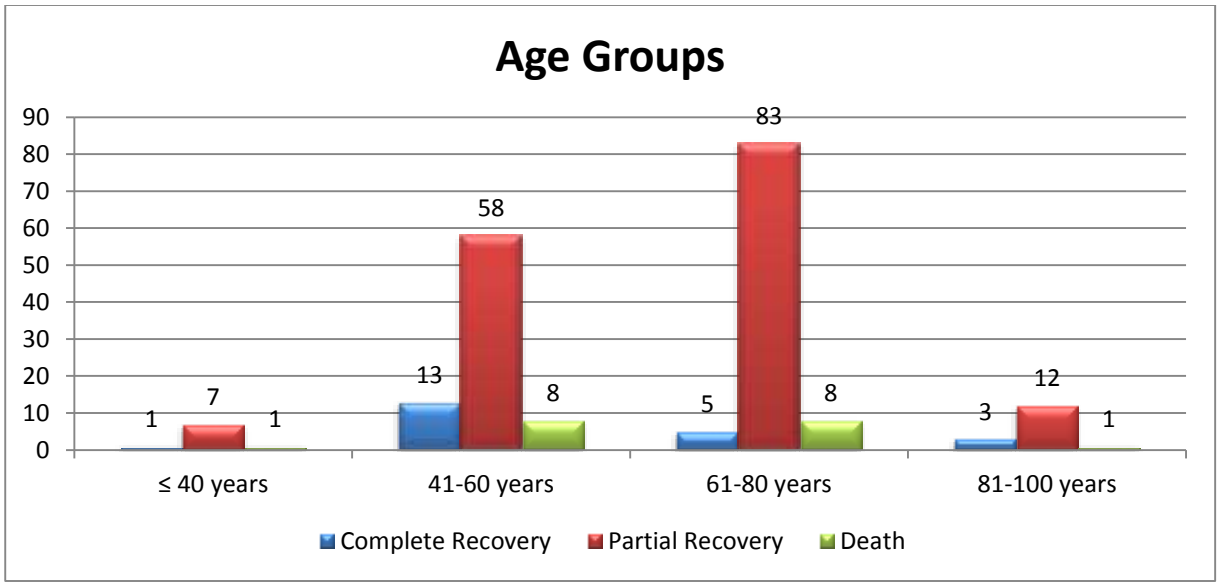
In the present study, 80 % recovered partially, 11 % recovered completely during the hospital stay and 9 % patients died in hospital.

Age



Age	Number	%
≤ 40 years	9	4.50
41-60 years	79	39.50
61-80 years	96	48.00
81-100 years	16	8.00
Total	200	100.00

In my study, stroke was more common in the age group of 61 to 80 years. Stroke in young (<45yrs) was found only in 7% of patients.



Age Groups	Complete Recovery	Partial Recovery	Death	Complete Recovery (%)	Partial Recovery (%)	Death (%)
≤ 40 years	1	7	1	4.55	4.38	5.56
41-60 years	13	58	8	59.09	36.25	44.44
61-80 years	5	83	8	22.73	51.88	44.44
81-100 years	3	12	1	13.64	7.50	5.56
Total	22	160	18	100.00	100.00	100.00

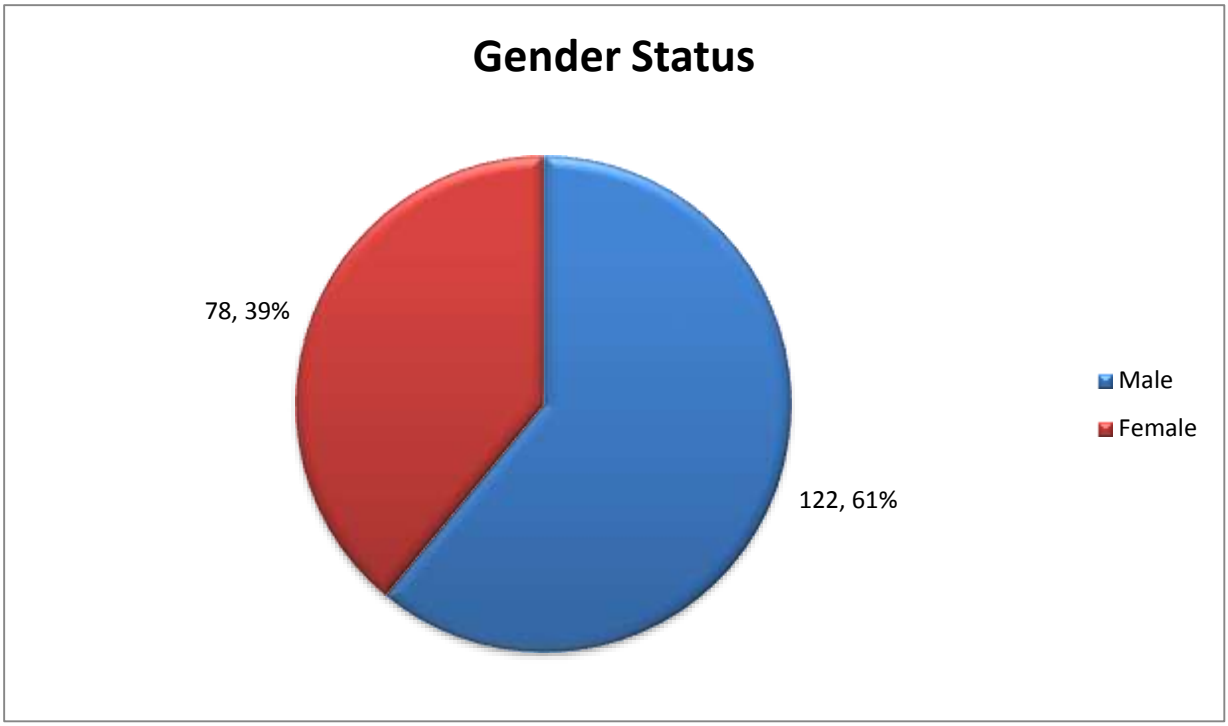
Age Distribution	Complete Recovery	Partial Recovery	Death
Mean	60.00	63.31	63.17
SD	13.38	11.41	11.95
P value Single Factor ANOVA			0.4592

Age distribution table depicts the classification of subjects by age based on outcome groups. It is evident from the results that majority in complete recovery group were in 41-60 years age group (59.09%) with a mean age of 60.00 years. Similarly in partial recovery

group majority were in 61-80 years age group (51.88%) with a mean age of 63.31 years and in death group majority were in 41-60 and 61-80 years age group (44.44%) with a mean age of 63.17 years. (p=0.4592).

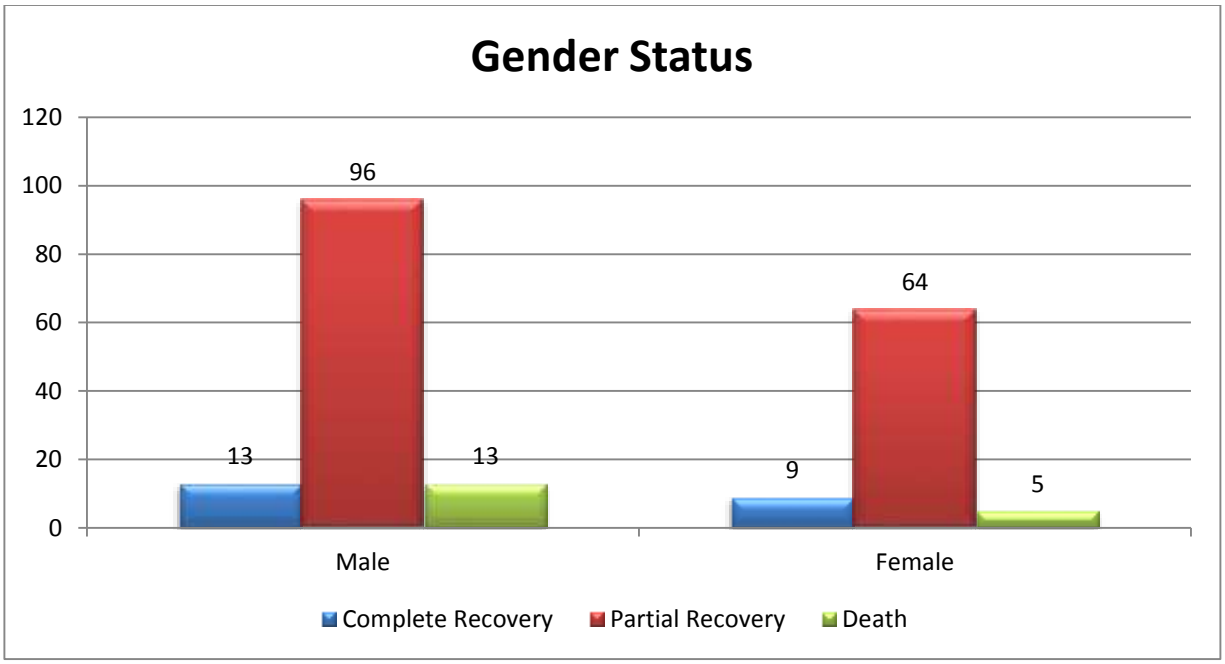
The data subjected to statistical single factor ANOVA test reveals the existence of statistically insignificant association between age distribution and outcome in patients with acute cerebrovascular disease ($p > 0.05$).

Gender



Gender Status	Number	%
Male	122	61.00
Female	78	39.00
Total	200	100.00

In this study stroke was more common in males than females, 61% of patients were males and 39% of patients were female.



Gender Status	Complete Recovery	Partial Recovery	Death	Complete Recovery (%)	Partial Recovery (%)	Death (%)
Male	13	96	13	59.09	60.00	72.22
Female	9	64	5	40.91	40.00	27.78
Total	22	160	18	100.00	100.00	100.00
P value Chi Squared Test				0.5909		

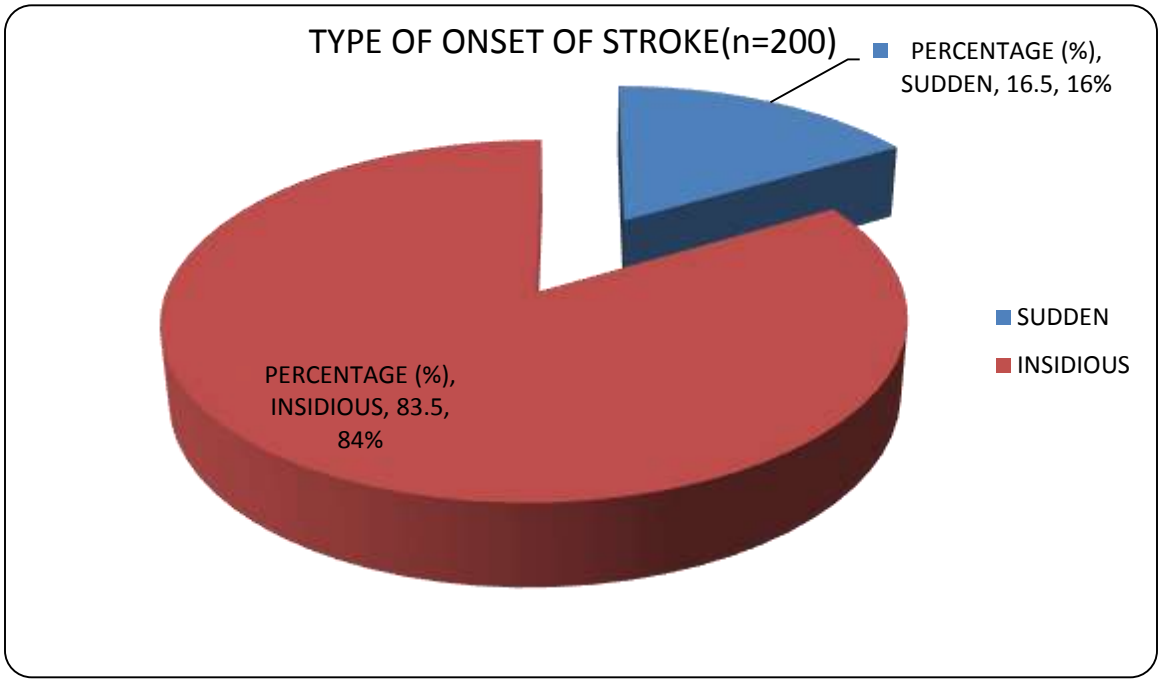
Majority of the subjects were males in complete recovery group (59.09%), in partial recovery group (60.00%) and in death group (72.22%)

(p= 0.5909).

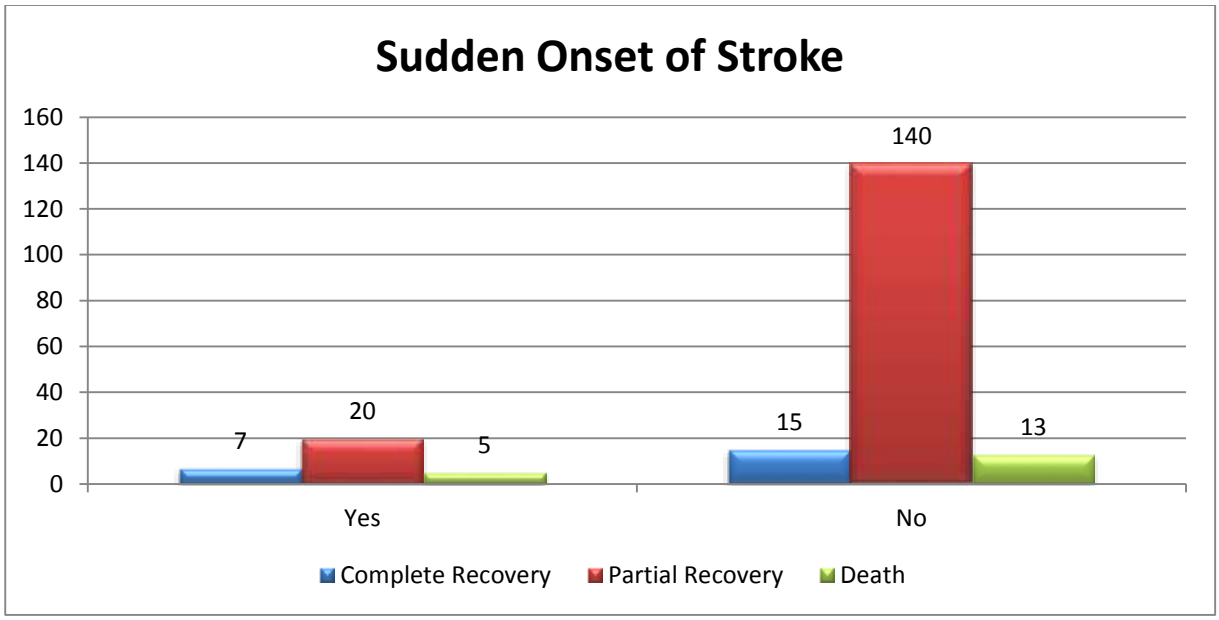
The data subjected to statistical chi squared test reveals the existence of statistically insignificant association between gender status and outcome in patients with acute cerebrovascular disease ($p > 0.05$).

Onset of Stroke

ONSET	NO OFPATIENTS	PERCENTAGE (%)
SUDDEN	33	16.5
INSIDIOUS	167	83.5
TOTAL	200	100.0



In the present study, insidious onset was more common (83.5%) than sudden onset of stroke.



Sudden Onset of Stroke	Complete Recovery	Partial Recovery	Death	Complete Recovery (%)	Partial Recovery (%)	Death (%)
Yes	7	45	5	31.82	28.13	27.78
No	15	115	13	68.18	71.88	72.22
Total	22	160	18	100.00	100.00	100.00
P value Chi Squared Test				0.0251		

Sudden onset of stroke table depicts the classification of subjects by suddenness of stroke and symptoms based on outcome groups. It is evident from the results that majority of the subjects didn't have sudden onset of stroke in complete recovery group (68.18%) and partial recovery group (71.88%) and vomiting/dizziness in death group (72.22%)-(p= 0.0251).

The data subjected to statistical chi squared test reveals the existence of statistically

significant association between sudden onset of stroke and outcome in patients with acute cerebrovascular disease ($p < 0.05$)

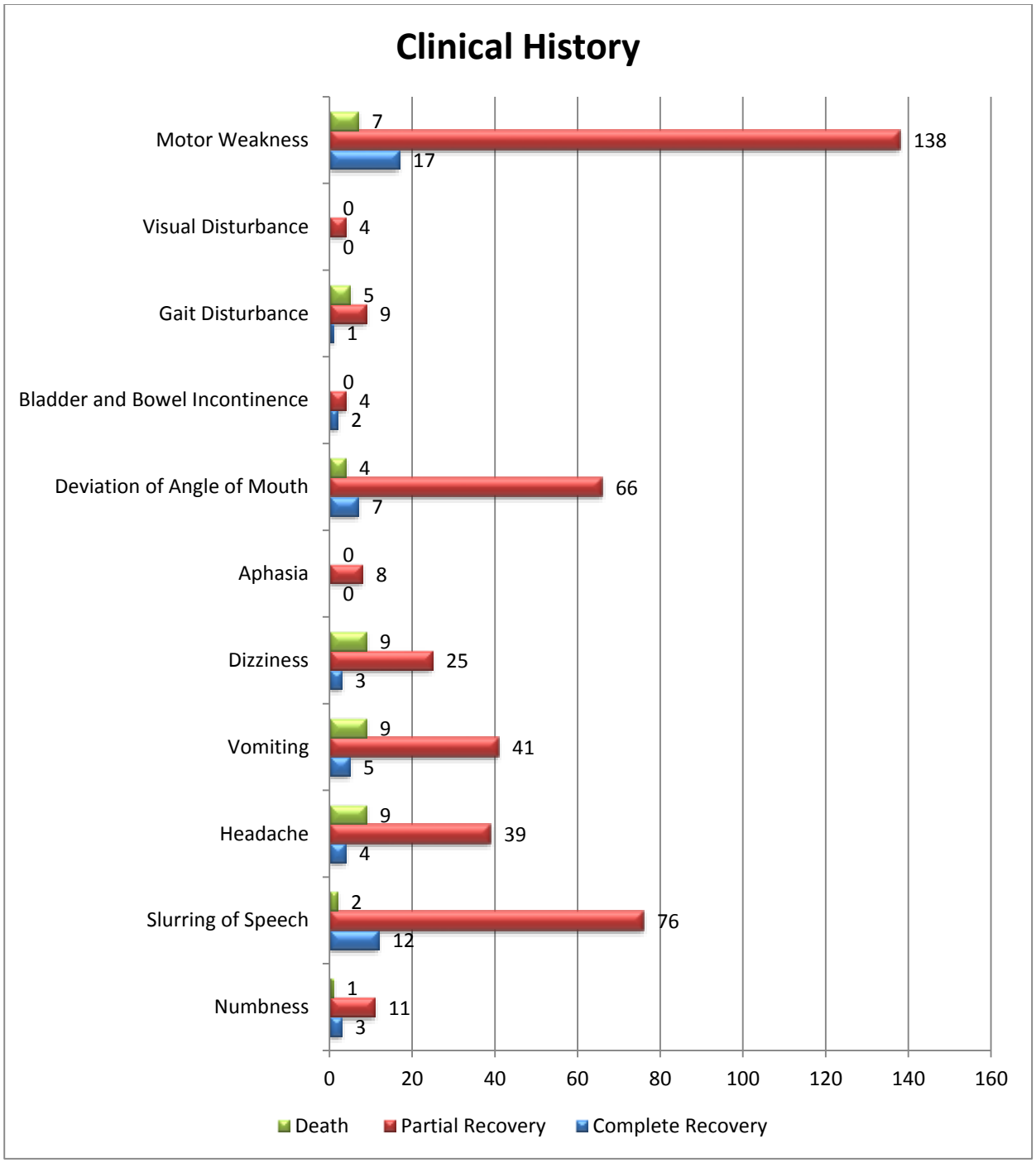
Discussion:

The increased incidence of patients with sudden onset of stroke in complete recovery group compared to partial recovery (percentage difference of 3.69 points, 12% higher) and in complete recovery group compared to death group (percentage difference of 4.04 points, 13% higher).

Clinical History

PRESENTING SYMPTOMS	NO OF PATIENTS (n=200)	PERCENTAGE(%)
MOTOR WEAKNESS	152	76
SENSORY SYMPTOMS	9	4.5
LOC/SEIZURE	20	10
SPEECH DISTURBANCE	90 + 8	49
HEADACH/DIZZINESS/VOMITING	74	37
VISUAL DISTURBANCE	4	2
FACIAL PALSY	77	38.5

Motor weakness was the commonest presenting symptom (76%) in this study, followed by speech disturbance in 49%.


Clinical History
Motor Weakness
7
138
17
Visual Disturbance
0
4
0
Gait Disturbance
5
9
1
Bladder and Bowel Incontinence
0
4
2
Deviation of Angle of Mouth
4
66
7
Aphasia
0
8
0
Dizziness
9
25
3
Vomiting
9
41
5
Headache
9
39
4
Slurring of Speech
2
76
12
Numbness
1
11
3
0
20
40
60
80
100
120
140
160
Death
Partial Recovery
Complete Recovery

Clinical History	Complete Recovery	Partial Recovery	Death	Complete Recovery (%)	Partial Recovery (%)	Death (%)	P value Chi Squared Test
Numbness	3	11	1	13.64	6.88	5.56	0.0411
Slurring of Speech	12	76	2	54.55	47.50	11.11	0.0029
Headache	4	39	9	18.18	24.38	50.00	<0.0001
Vomiting	5	41	9	22.73	25.63	50.00	0.0317
Dizziness	3	25	9	13.64	15.63	50.00	0.0001
Aphasia	0	8	0	0.00	5.00	0.00	0.3513
Deviation of Angle of Mouth	7	66	4	31.82	41.25	22.22	0.2305
Bladder and Bowel Incontinence	2	4	0	9.09	2.50	0.00	0.1744
Gait Disturbance	1	9	5	4.55	5.63	27.78	0.0033
Visual Disturbance	0	4	0	0.00	2.50	0.00	0.6007
Motor Weakness	17	138	7	77.27	86.25	38.89	<0.0001

Clinical history table depicts the classification of subjects by clinical signs and symptoms based on outcome groups. It is evident from the results that majority of the subjects had motor weakness in complete recovery group (77.27%) and partial recovery group (86.25%) and vomiting/dizziness in death group (38.89%).(p= <0.0001)

The data subjected to statistical chi squared test reveals the existence of statistically

significant association between clinical history (numbness, slurring of speech, headache, vomiting, dizziness, gait disturbance and motor weakness) and outcome in patients with acute cerebrovascular disease ($p < 0.05$)

Discussion:

The decreased incidence of patients with motor weakness in complete recovery group compared to partial recovery (percentage difference of 8.98 points, 10% lower) and increased incidence of patients with motor weakness in complete recovery group compared to death group (percentage difference of 38.38 points, 50% higher).
The increased incidence of patients with numbness in complete recovery group compared to partial recovery (percentage difference of 6.76 points, 50% higher) and in complete recovery group compared to death group (percentage difference of 8.08 points, 59% higher).

The increased incidence of patients with slurring of speech in complete recovery group compared to partial recovery (percentage difference of 7.05 points, 13% higher) and in complete recovery group compared to death group (percentage difference of 43.43 points, 80% higher).

The decreased incidence of patients with headache in complete recovery group compared to partial recovery (percentage difference of 6.19 points, 25% lower) and in complete recovery group compared to death group (percentage difference of 31.82 points, 74%

lower).

The decreased incidence of patients with vomiting noted in complete recovery group compared to partial recovery (percentage difference of 2.90 points, 11% lower) and in complete recovery group compared to death group (percentage difference of 27.27 points, 55% lower).

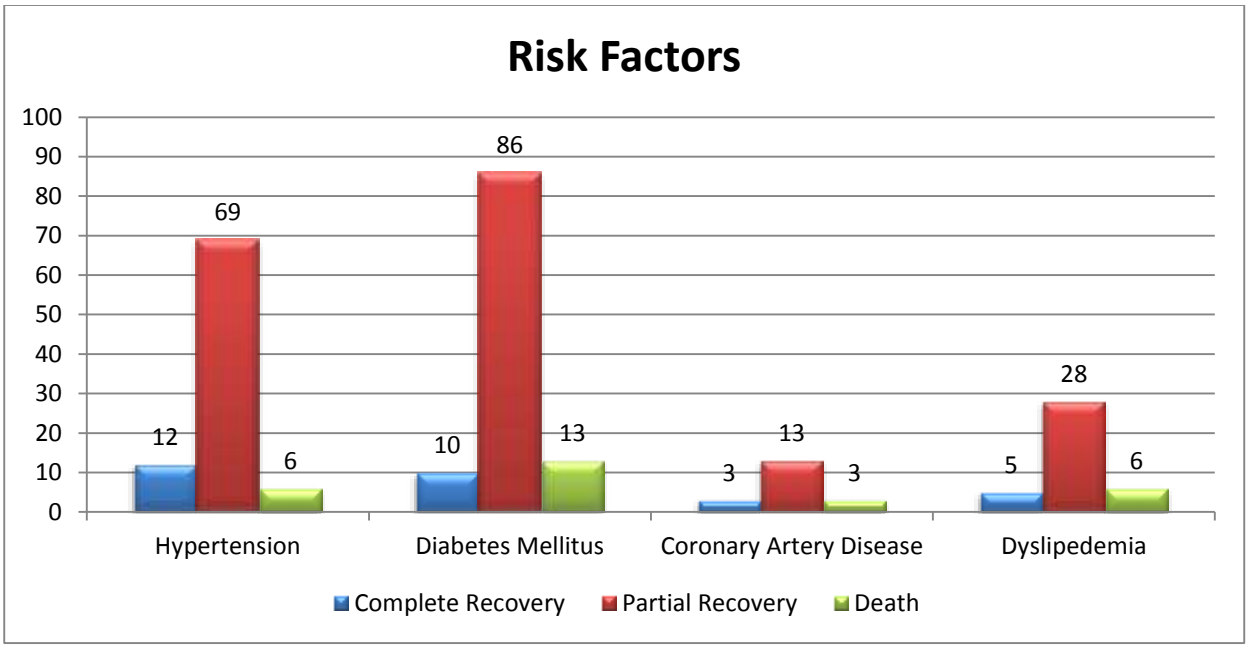
The decreased incidence of patients with dizziness in complete recovery group compared to partial recovery (percentage difference of 1.99 points, 13% lower) and in complete recovery group compared to death group (percentage difference of 36.36 points, 73% lower).

The decreased incidence of patients with gait disturbance in complete recovery group compared to partial recovery (percentage difference of 1.08 points, 19% lower) and in complete recovery group compared to death group (percentage difference of 23.23 points, 84% lower).

Comorbidities & Risk factors

RISK FACTOR	NO OF PATIENTS	PERCENTAGE (%)
HYPERTENSION	139	69.5
DIABETES MELLITUS	109	54.5
DYSLIPIDEMIA	50	25
CORONARY ARTERY DISEASE	21	10.5
ATRIAL FIBRILLATION	14	7
OBESITY	68	34
TOTAL	200	100.0

In this study, 69.5% of study population had hypertension, which is the commonest risk factor for stroke in adults. 54.5% of patients had Diabetes Mellitus, 10.5% had Coronary heart disease, 7% had Atrial Fibrillation, 25% had Dyslipidemia and 34% had obesity as risk factor for stroke.



Comorbidities	Complete Recovery	Partial Recovery	Death	Complete Recovery (%)	Partial Recovery (%)	Death (%)	P value Chi Squared Test
Hypertension	12	69	6	54.55	43.13	33.33	0.3953
Diabetes Mellitus	10	86	13	45.45	53.75	72.22	0.2188
Coronary Artery Disease	3	13	3	13.64	8.13	16.67	0.3942
Dyslipedemia	5	28	6	22.73	17.50	33.33	0.2533
Atrial Fibrillation	10	3	1	45.45	1.88	5.56	<0.0001

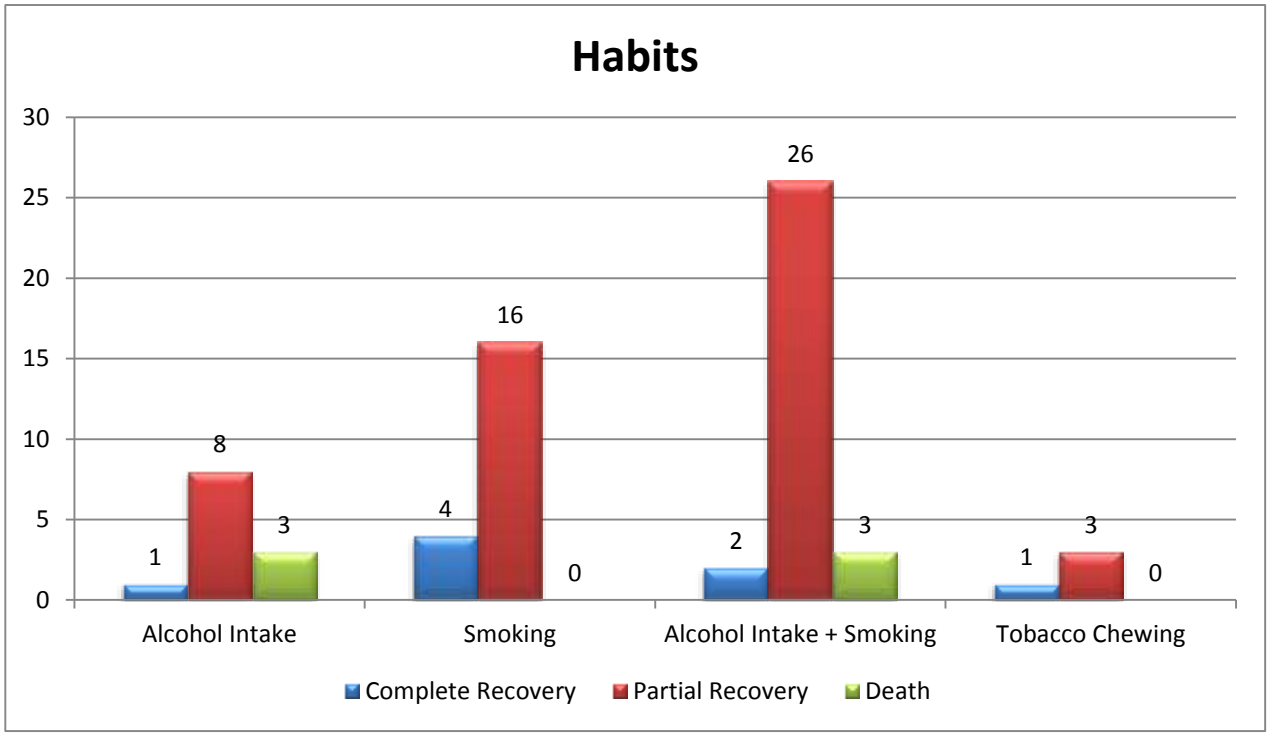
Comorbidities table depicts the classification of subjects by comorbid conditions and symptoms based on outcome groups. It is evident from the results that majority of the subjects did have hypertension in complete recovery group (54.55%) and diabetes mellitus in partial recovery group (53.75%) and diabetes mellitus in death group (72.22%) as their major comorbid condition.(p= 0.0251)

The data subjected to statistical chi squared test reveals the existence of statistically significant association between atrial fibrillation and outcome in patients with acute cerebrovascular disease ($p < 0.05$)

Discussion

The increased incidence of atrial fibrillation in complete recovery group compared to partial recovery (percentage difference of 43.58 points, 96% higher) and in complete recovery group compared to death group (percentage difference of 39.90 points, 88% higher).

Habits



Habits	Complete Recovery	Partial Recovery	Death	Complete Recovery (%)	Partial Recovery (%)	Death (%)	P value Chi Squared Test
Alcohol Intake	1	8	3	4.55	5.00	16.67	0.1457
Smoking	4	16	0	18.18	10.00	0.00	0.1622
Alcohol Intake + Smoking	2	26	3	13.64	24.38	33.33	0.0237
Tobacco Chewing	1	3	0	4.55	1.88	0.00	0.5753

Habits table depicts the classification of subjects by habitual conditions and symptoms based on outcome groups. It is evident from the results that majority of the subjects did smoke in complete recovery group (18.18%) and smoked + consumed alcohol in partial

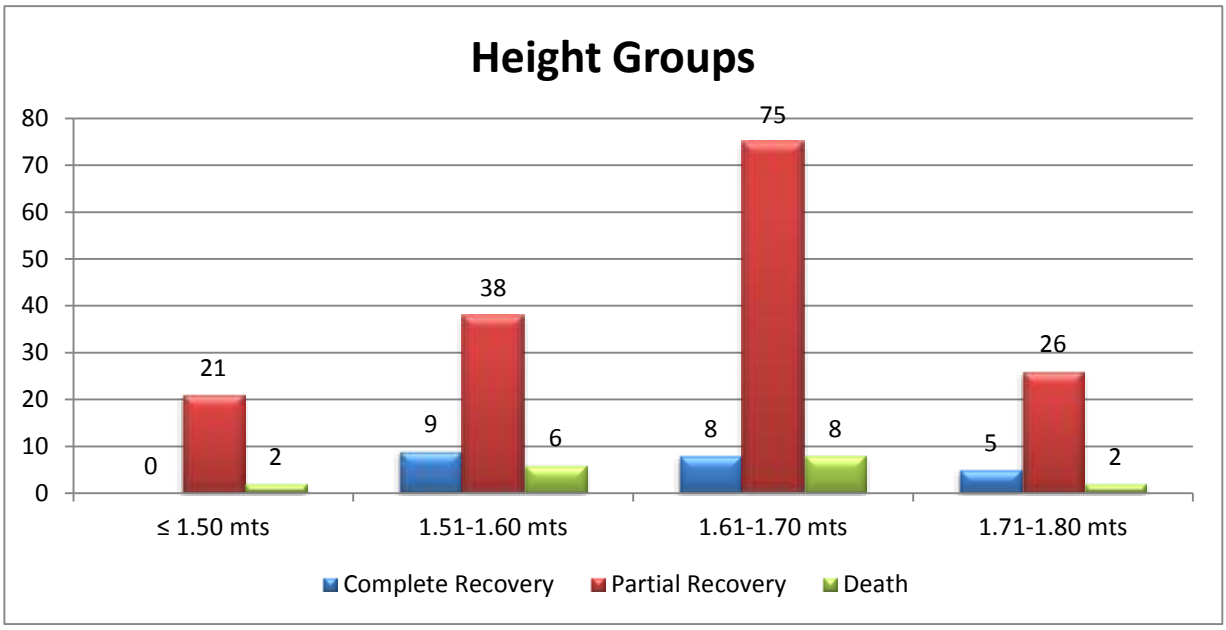
recovery group (24.38%) and smoked + consumed alcohol in death group (33.33%).(p= 0.0251)

The data subjected to statistical chi squared test reveals the existence of statistically significant association between additive effect of smoking + consuming alcohol and outcome in patients with acute cerebrovascular disease ($p < 0.05$)

Discussion

The decreased incidence of smoking + consuming alcohol in complete recovery group compared to partial recovery (percentage difference of 10.74 points, 44% lower) and in complete recovery group compared to death group (percentage difference of 19.70 points, 59% lower).

Height



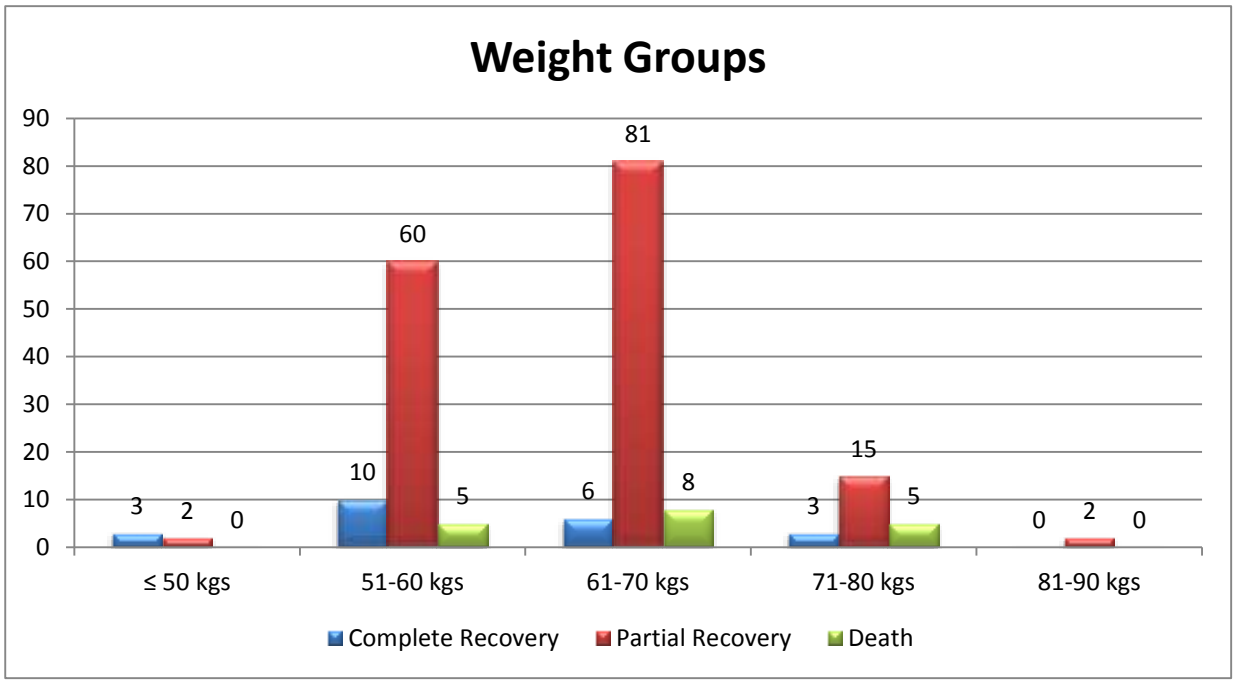
Height Groups	Complete Recovery	Partial Recovery	Death	Complete Recovery (%)	Partial Recovery (%)	Death (%)
≤ 1.50 mts	0	21	2	0.00	13.13	11.11
1.51-1.60 mts	9	38	6	40.91	23.75	33.33
1.61-1.70 mts	8	75	8	36.36	46.88	44.44
1.71-1.80 mts	5	26	2	22.73	16.25	11.11
Total	22	160	18	100.00	100.00	100.00

Height Distribution	Complete Recovery	Partial Recovery	Death
Mean	1.63	1.62	1.62
SD	0.07	0.09	0.08
P value Single Factor ANOVA			0.9231

Height distribution table depicts the classification of subjects by height on outcome groups. It is evident from the results that majority in complete recovery group were in 1.51-1.60 mts height group (40.91%) with a mean height of 1.63 mts. Similarly in partial recovery group majority were in 1.61-1.70 mts height group (46.88%) with a mean height of 1.62 mts and in death group majority were in 1.61-1.70 mts height group (44.44%) with a mean height of 1.62 mts. (p=0.9231)

The data subjected to statistical single factor ANOVA test reveals the existence of statistically insignificant association between height distribution and outcome in patients with acute cerebrovascular disease ($p > 0.05$)

Weight



Weight Groups	Complete Recovery	Partial Recovery	Death	Complete Recovery (%)	Partial Recovery (%)	Death (%)
≤ 50 kgs	3	2	0	13.64	1.25	0.00
51-60 kgs	10	60	5	45.45	37.50	27.78
61-70 kgs	6	81	8	27.27	50.63	44.44
71-80 kgs	3	15	5	13.64	9.38	27.78
81-90 kgs	0	2	0	0.00	1.25	0.00
Total	22	160	18	100.00	100.00	100.00

Weight Distribution	Complete Recovery	Partial Recovery	Death
Mean	60.27	63.24	64.94
SD	8.99	7.10	6.75
P value Single Factor ANOVA			0.1075

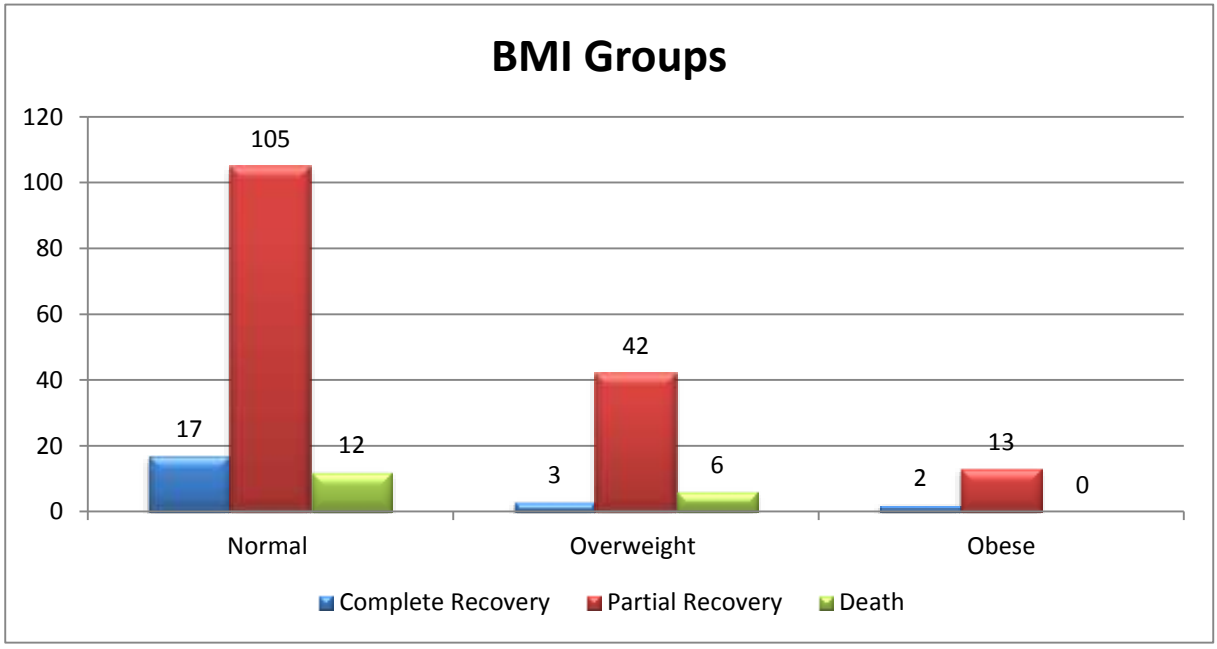
Weight distribution table depicts the classification of subjects by weight on outcome groups. It is evident from the results that majority in complete recovery group were in 51-60 kgs weight group (45.45%) with a mean weight of 60.27 kgs. Similarly in partial recovery group majority were in 51-60 kgs weight group (37.50%) with a mean weight of 62.34 kgs. and in death group majority were in 61-70 kgs weight group (44.44%) with a mean weight of 64.94 kgs.. (p=0.1075)

The data subjected to statistical single factor ANOVA test reveals the existence of statistically insignificant association between weight distribution and outcome in patients with acute cerebrovascular disease. ($p > 0.05$)

BMI

BMI	NO OF PATIENTS	PERCENTAGE (%)
OBESITY	65	32.5
OVER WEIGHT	59	29.5
NORMAL	67	33.5
UNDERWEIGHT	9	4.5
TOTAL	200	100.0

In my study only 1/3rd (33.5%) of the patients had normal BMI. The remaining patients were either obese or overweight.



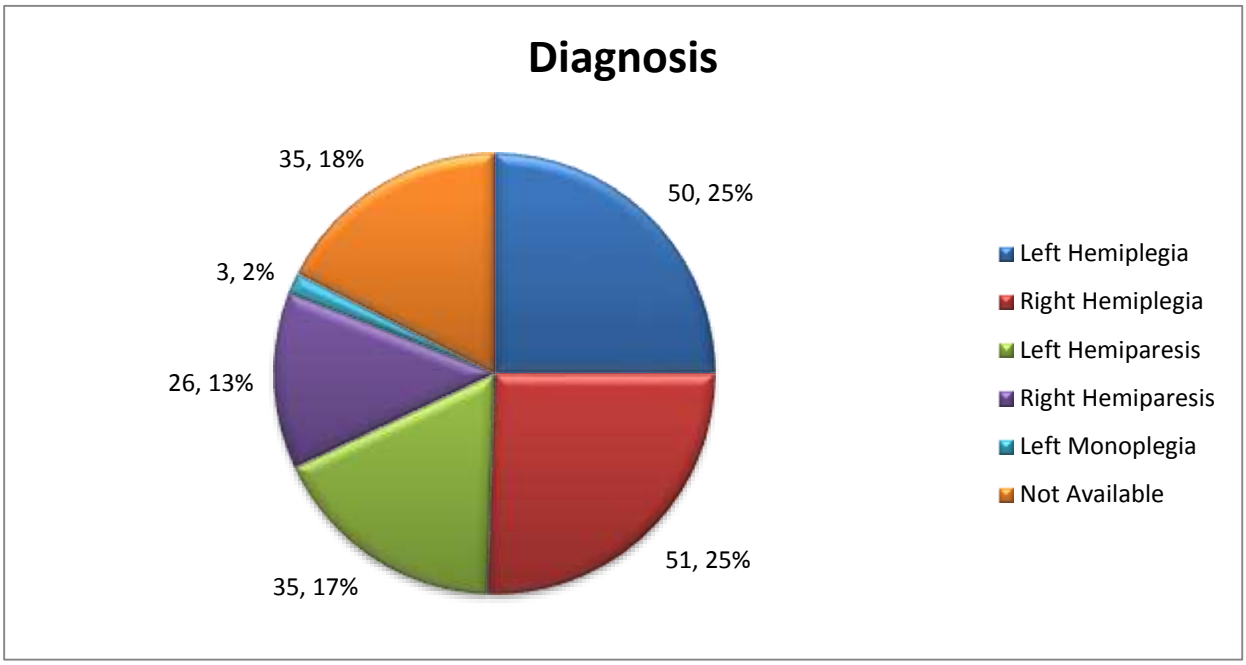
BMI Groups	Complete Recovery	Partial Recovery	Death	Complete Recovery (%)	Partial Recovery (%)	Death (%)
Normal	17	105	12	77.27	65.63	66.67
Overweight	3	42	6	13.64	26.25	33.33
Obese	2	13	0	9.09	8.13	0.00
Total	22	160	18	100.00	100.00	100.00

BMI Distribution	Complete Recovery	Partial Recovery	Death
Mean	22.82	24.22	24.81
SD	3.88	3.54	2.49
P value Single Factor ANOVA			0.1472

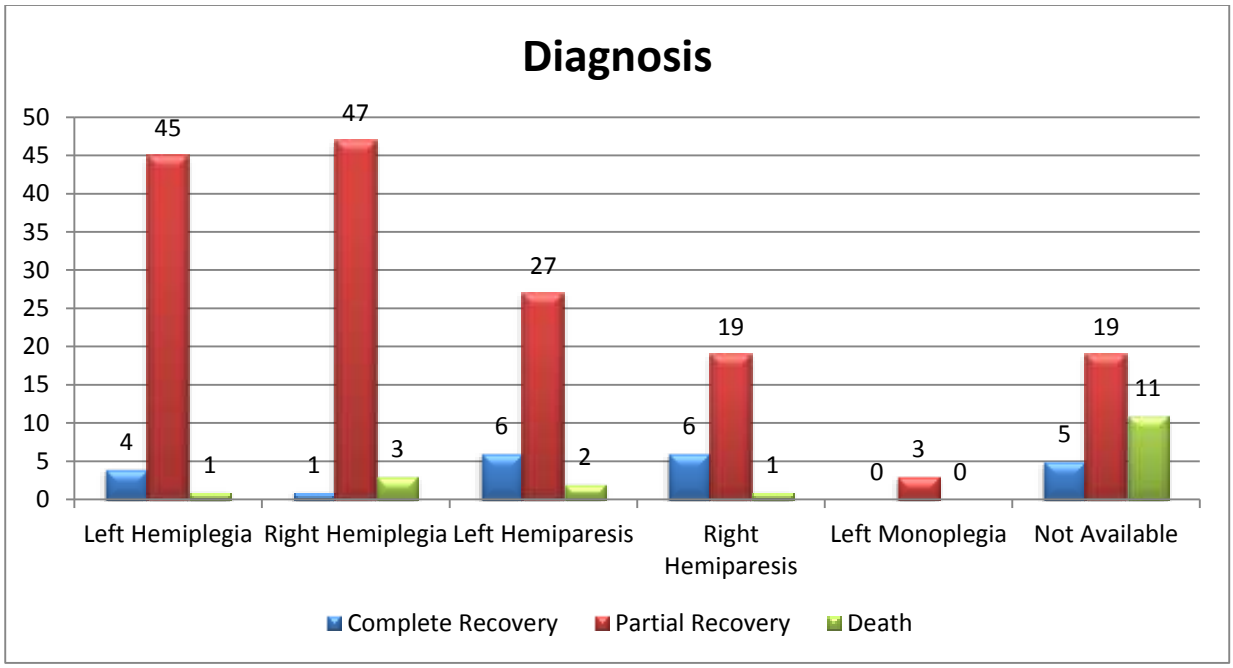
BMI distribution table depicts the classification of subjects by BMI on outcome groups. It is evident from the results that majority in complete recovery group were in normal BMI group (77.27%) with a mean weight of 22.82. Similarly in partial recovery group majority were in normal BMI group (65.63%) with a mean weight of 24.22.and in death group majority were in normal BMI group (66.67%) with a mean weight of 24.81 (p=0.1472)

The data subjected to statistical single factor ANOVA test reveals the existence of statistically insignificant association between BMI distribution and outcome in patients with acute cerebrovascular disease. ($p > 0.05$)

Diagnosis



Diagnosis	Number	%
Left Hemiplegia	50	25.00
Right Hemiplegia	51	25.50
Left Hemiparesis	35	17.50
Right Hemiparesis	26	13.00
Left Monoplegia	3	1.50
Other symptoms	35	17.50
Total	200	100.00



Diagnosis	Complete Recovery	Partial Recovery	Death	Complete Recovery (%)	Partial Recovery (%)	Death (%)
Left Hemiplegia	4	45	1	18.18	28.13	5.56
Right Hemiplegia	1	47	3	4.55	29.38	16.67
Left Hemiparesis	6	27	2	27.27	16.88	11.11
Right Hemiparesis	6	19	1	27.27	11.88	5.56
Left Monoplegia	0	3	0	0.00	1.88	0.00
Other symptoms	5	19	11	22.73	11.88	61.11
Total	22	160	18	100.00	100.00	100.00
P value Chi Squared Test				0.0001		

Diagnosis table depicts the classification of subjects by diagnosis based on outcome groups. It is evident from the results that majority of the subjects were diagnosed hemiparesis (both right and left side) in complete recovery group (27.27%), hemiplegia

(mainly right side) in partial recovery group (29.38%) and hemiplegia (mainly right side) in death group (16.67%).(p= 0.0001)

The data subjected to statistical chi squared test reveals the existence of statistically significant association between diagnosis made and outcome in patients with acute cerebrovascular disease ($p < 0.05$)

Discussion:

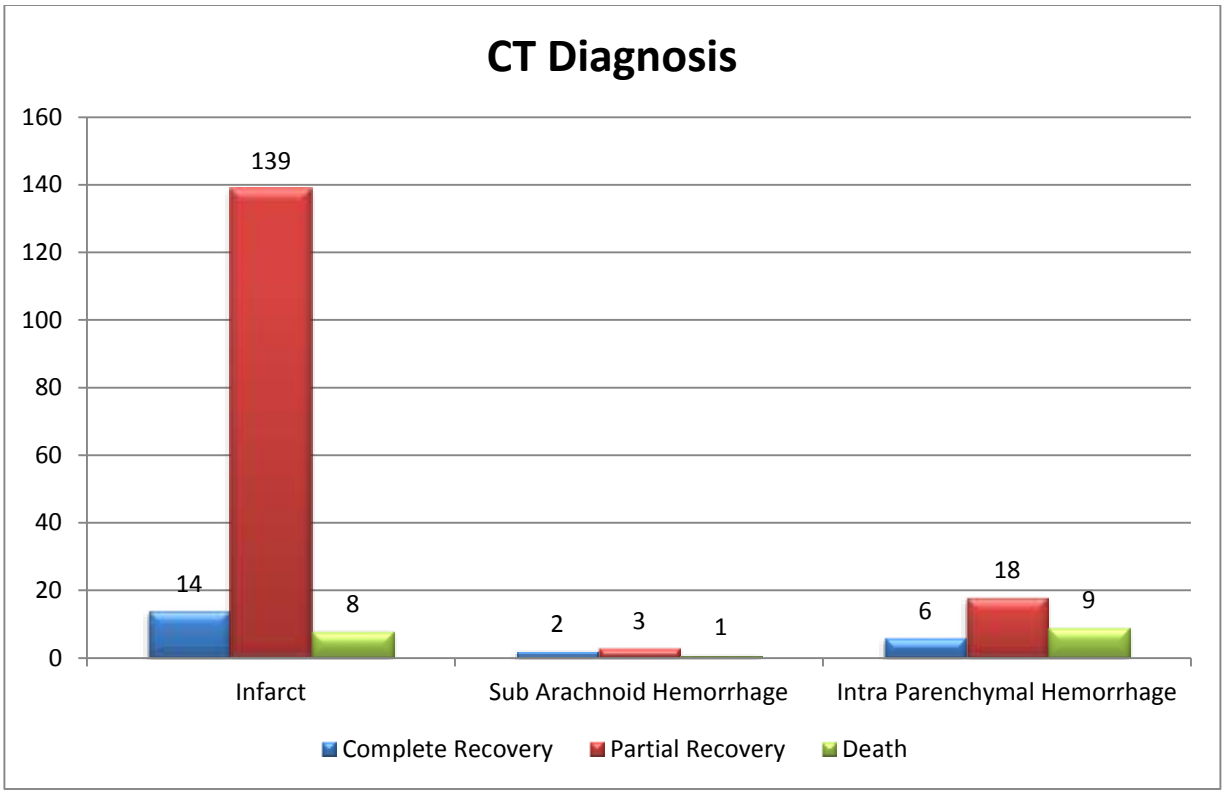
In our study the diagnosis status between outcome groups was meaningfully statistically significant. This is exhibited by the increased percentage of patients diagnosed with hemiparesis in complete recovery group (54.54%) compared to partial recovery (28.76%) and death groups (16.67%). The incidence of hemiparesis was 47% higher (percentage difference of 25.80 points) in complete recovery group compared to partial recovery and 69% higher (percentage difference of 37.88 points) in complete recovery group compared to death group.

CT Diagnosis

CT IMAGING	NO OF PATIENTS	(%)
INFARCT	161	80.5 %
INTRA PARENCHYMAL BLEED	33	16.5 %
SAH	6	3 %
TOTAL	200	100 %

Cerebral infarct was the commonest finding in the CT imaging about 80.5% followed by Intra-parenchymal bleed (16.5%) and Subarachnoid hemorrhage (3%).

CT Diagnosis	Complete Recovery	Partial Recovery	Death	Complete Recovery (%)	Partial Recovery (%)	Death (%)
Infarct	14	139	8	63.64	86.88	44.44
Sub Arachnoid Hemorrhage	2	3	1	9.09	1.88	5.56
Intra Parenchymal Hemorrhage	6	18	9	27.27	11.25	50.00
Total	22	160	18	100.00	100.00	100.00
P value Chi Squared Test				<0.0001		



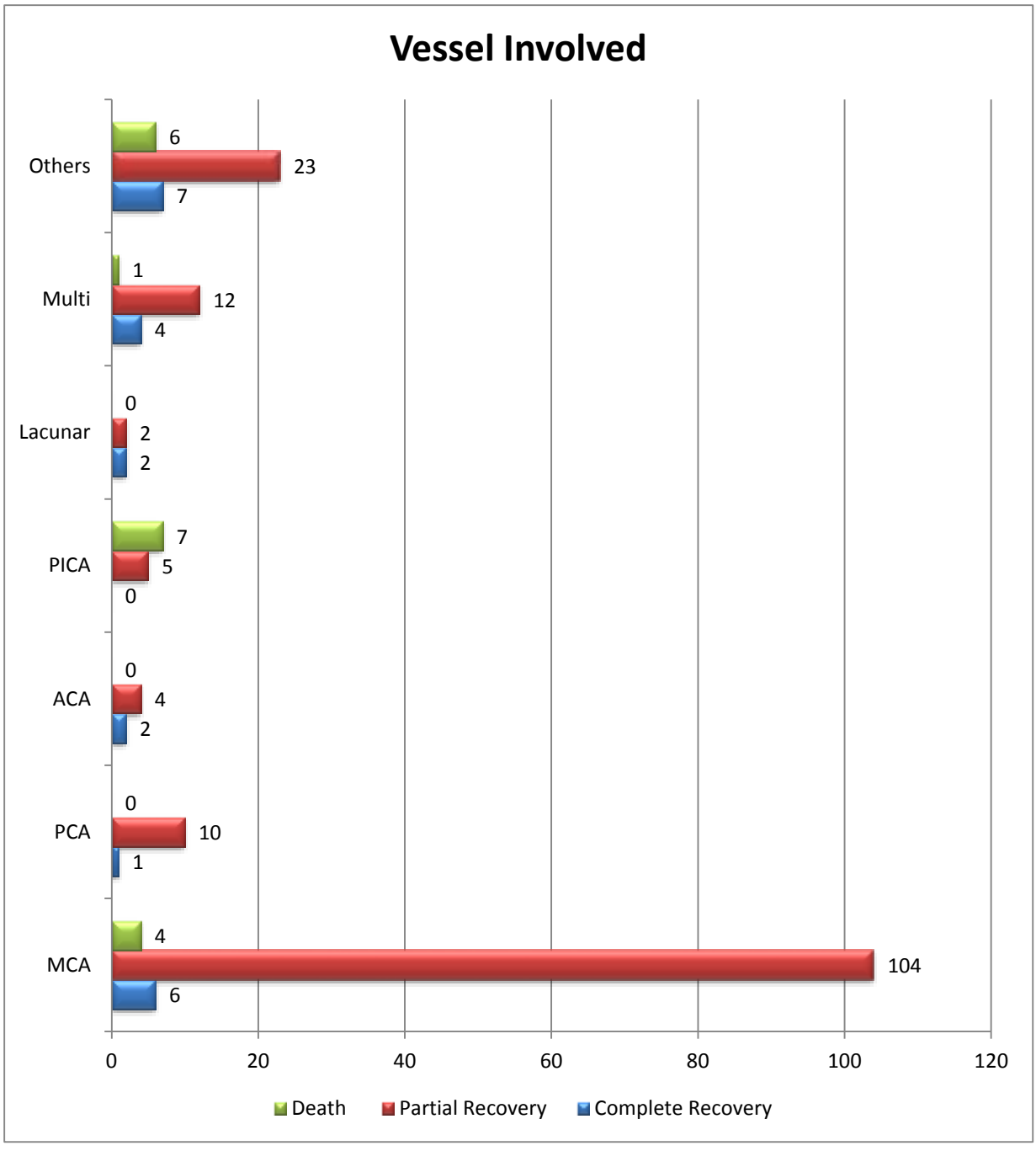
CT diagnosis table depicts the classification of subjects by diagnosis on CT evaluation and symptoms based on outcome groups. It is evident from the results that majority of the subjects had infarct in complete recovery group (63.64%) and also in partial recovery group (86.88%) followed by hemorrhage (especially intra parenchymal type) in death group (55.56%).(p= <0.0001)

The data subjected to statistical chi squared test reveals the existence of statistically significant association between CT diagnosis and outcome in patients with acute cerebrovascular disease. ($p < 0.05$)

Discussion

The decreased incidence of infarct type of stroke in complete recovery group compared to partial recovery (percentage difference of 23.24 points, 13% lower) and increased incidence of infarct type of stroke in complete recovery group compared to death group (percentage difference of 19.19 points, 30% higher).

Vessel Involved



In my study, MCA territory involvement was more common (57%); followed by PICA (6%), PCA (5.5%) and ACA (3%) and 8.5% of patients had multiple infarcts in CT.

Vessel Involved	Complete Recovery	Partial Recovery	Death	Completel Recovery (%)	Partial Recovery (%)	Death (%)
MCA	6	104	4	18.18	65.00	22.22
PCA	1	10	0	4.55	6.25	0.00
ACA	2	4	0	9.09	2.50	0.00
PICA	0	5	7	0.00	3.13	38.89
Lacunar	2	2	0	9.09	1.25	0.00
Multi	4	12	1	18.18	7.50	5.56
Others	9	23	6	40.91	14.38	33.33
Total	22	160	18	100.00	100.00	100.00
P value Chi Squared Test				<0.0001		

Vessel involved table depicts the classification of subjects by type of vessel affected and symptoms based on outcome groups. It is evident from the results that majority of the subjects had involvement of MCA vessel in complete recovery group (18.18%) and also same in partial recovery group (65.00%) followed by PICA involvement death group (38.89%).(p= <0.0001)

The data subjected to statistical chi squared test reveals the existence of statistically significant association between vessel involved and outcome in patients with acute cerebrovascular disease. ($p < 0.05$)

Discussion:

The increased incidence of MCA involvement in recovery groups (complete and partial) compared to death group (percentage difference of 70.05 points, 76% higher) and increased incidence of PICA involvement in death group (complete and partial) compared to recovery groups (percentage difference of 35.76 points, 92% higher).

Logistic Regression Analysis

Logistic regression model for statistically Significant Independent Predictors of death outcome in patients with acute cerebrovascular disease :

Predictor Variable	Risk Ratio	Confidence Interval	P value
Age > 60 Years	1.64	0.19–14.46	0.951
Gender-male	1.93	0.34-10.8	0.807
Diagnosis - Hemiplegia	3.04	1.33-6.95	0.014*
Sudden Onset	0.45	0.18-0.81	0.045*
Smoker + alcoholic	4.29	1.12-16.52	0.039*
Hypertension	1.10	0.86-1.41	0.062
Diabetes	1.37	0.71-2.63	0.107
CAD	0.92	0.67-1.22	0.735
Dyslipedaemia	0.23	0.09-2.11	0.368
Atrial fibrillation	0.87	0.10-7.73	1.000
BMI - Obese	4.22	1.14-12.66	0.245
Type of Stroke – Hemorrhage	4.59	1.38-12.34	0.001*
Vessel Involved – PICA	1.62	0.56-3.84	0.007*

Multivariate analysis demonstrated that after adjusting for LAVI.

- The risk of death in patients with acute cerebrovascular disease diagnosed with hemiplegia is 3.04 times significantly more than in patients diagnosed with hemiparesis. It is statistically significant with a p-value of 0.014

- The risk of death in patients with acute cerebrovascular disease presenting with sudden onset is 0.45 times (55% lower) significantly less than in patients presenting with gradual onset. It is statistically significant with a p-value of 0.045
- The risk of death in patients with acute cerebrovascular disease having addictive effect of smoking + alcohol is 4.29 times significantly more than in patients having no additive habit. It is statistically significant with a p-value of 0.039
- The risk of death in patients with acute cerebrovascular disease diagnosed with CT diagnosis hemorrhage is 4.59 times significantly more than in patients diagnosed with Infarct. It is statistically significant with a p-value of 0.001
- The risk of death in patients with acute cerebrovascular disease diagnosed with PICA/PCA vessel involved is 1.62 times significantly more than in patients with MCA/ACA vessel involved. It is statistically significant with a p-value of 0.007

DISCUSSION AND SUMMARY

Stroke was more common above the age of 61 years (74%); 61 to 80 years constituted 48%. Stroke in young was found only in 7% of patients. This is similar to a study done by Vaidya CV et al in which most common age group was 61-70 years.(60)

The studies done by Vaidya CV et al and Renjen et al revealed male preponderance (59.7% and 67.6% respectively) (60) (61) which is in accordance to my study which showed 61% male preponderance.

Mortality was more in males (6.5%) but there is no significant statistical difference between both gender and the outcome of Stroke. This was dissimilar to a study done by Turtzo and McCullough in which they found that females had poorer functional outcome after acute ischemic stroke than males.(65)

In the present study, insidious onset was more common (83.5%) than sudden onset of stroke. The increased incidence of patients with sudden onset of stroke noted in complete recovery group compared to partial recovery and in complete recovery group compared to death group. (60) (61)

Motor weakness was the commonest presenting symptom (76%) in my study. It is similar to the study done by Nagaraja et al which postulated that motor weakness was the commonest clinical feature in about 82% of patients. . Study revealed reduced incidence of patients with motor weakness in complete recovery group compared to partial recovery and increased incidence of patients with motor weakness in complete recovery group compared to death group . Another study done by Vaidya CV et al also had similar finding in 72.6% of patients.(60) Patients presenting with clinical history of headache, vomiting, dizziness and gait disturbance have greater risk and incidence of death compared numbness, slurring of speech.

Comparison of risk factors with various studies:

	Renjen et al(61)	Sridharan et al(26)	Nagaraja et al(39)	**Present study**
HTN	56.9%	81.6%	48%	69.5%
DM	34.8%	48.7%	23.1%	**54.5%**
CAD	18%	-	-	10.5%
AF	5.3%	8.7%	9.7%	7%
Dyslipidemia	23.7%	25.3%	-	25%

Among risk factors, increased incidence of atrial fibrillation noted in complete recovery group compared to partial recovery and death group.

In the current study 33.5% of the patients had normal BMI while the study done by Kuriakose et al revealed 20.3% had normal BMI. Obesity was observed in 32.5% of patients in the present study while it was higher in the study done by Kuriakose et al(41.5%).(62) But the results revealed insignificant association between BMI distribution and outcome in patients.

Tobacco use was noted in 41.8% of males in the present study. This was almost comparable to the study done by Renjen et al (61)in which 38.9% had tobacco use, but it was more in the studies done by Sridharan et al and Nagaraja et al.(26) (39) Alcohol use was observed in 27.5% of males in my study, and this was comparable with that of Nagaraja et al(25.1%).(39) Analysis revealed statistically significant association between additive effect of smoking + consuming alcohol and outcome in patients with acute cerebrovascular disease. ($p < 0.05$)

Majority of the patients in this study (80.5%) had cerebral infarct. Hemorrhagic stroke was observed in 19.5% of patients with Intraparenchymal bleed (16.5%) and subarachnoid hemorrhage (3%);. In the study done by Pandian et al 68% had infarct and remaining 32% had hemorrhagic stroke.(6)

Patients presenting with CT diagnosis as hemorrhage have greater risk and incidence of death compared to infarct. The findings were very similar to study done by Vaidya CV et al with 74.6% showing ischemic stroke and 22.9% having hemorrhagic stroke.(60) This is also similar, to the study by Nagaraja et al with 73.8% having ischemic stroke,13.7% with intra-cerebral hemorrhage and 1.4% with subarachnoid hemorrhage.(39)

In present study, MCA territory involvement was more common (57%) which was comparable to study done by Ng et al in which majority had MCA territory involvement (50.8%). (63) Data analysis revealed increased incidence of MCA involvement in recovery groups (complete and partial) compared to death group and increased incidence of PICA/PCA involvement in death group compared to recovery groups.

CONCLUSION

Age, gender, hypertension, diabetes mellitus, CAD, dyslipedemia, height, weight and BMI had no statistically significant role to play on deciding the outcome in patients with acute cerebrovascular disease.

When internal comparison among complete recovery, partial recovery and death groups surgery were matched, the following conclusions were observed

- Patients diagnosed with hemiplegia have greater risk and incidence of death compared to hemiparesis (which favours recovery)
- Patients presenting with clinical history of headache, vomiting, dizziness and gait disturbance have greater risk and incidence of death compared numbness, slurring of speech (which favours recovery)
- Patients presenting with atrial fibrillation as comorbidity have a higher incidence of complete recovery {cardio-embolic stroke}
- Patients presenting with additive effect of smoking + alcoholism have greater risk and incidence of death compared to either habits separately or no habits
- Patients presenting with CT diagnosis as hemorrhage have greater risk and incidence of death compared to infarct
- Patients presenting with posterior circulation involvement have greater risk

and incidence of death compared to MCA/ACA vessel involvement

The ideal individual with high risk of death among patients with acute cerebrovascular disease

- Hemorrhagic stroke
- Presenting with delayed onset hemiplegia
- Posterior circulation involvement
- History of smoking plus alcoholism

This study is a hypothesis proving study.

Hence results have high clinical significance.

LIMITATIONS

- As it is a hospital based study, these results cannot be extrapolated to the general population.

RECOMMENDATIONS

- Large population based studies are needed for further establishment of outcome of Stroke.
- Steps should be taken to increase the awareness of Stroke in high risk population.
- Early identification and management of risk factors will reduce the incidence and outcome of Stroke.
- Early hospitalization, appropriate management and timely referral for advanced management will reduce the morbidity and mortality of Stroke.
- Psycho-social and occupational rehabilitation will help to prevent further progression of the morbidity.

BIBLIOGRAPHY

1. Prasad K, Vibha D, Meenakshi. Cerebrovascular disease in South Asia – Part I: A burning problem. JRSM Cardiovasc Dis [Internet]. 2012 Oct 31 [cited 2016 Oct 25];1(7). Available from: http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3738368/

2. Feigin VL, Forouzanfar MH, Krishnamurthi R, Mensah GA, Connor M, Bennett DA, et al. Global and regional burden of stroke during 1990-2010: findings from the Global Burden of Disease Study 2010. Lancet Lond Engl. 2014 Jan 18;383(9913):245–54.

3. Kulshrestha M, Vidyanand. An Analysis of the Risk Factors and the Outcomes of Cerebrovascular Diseases in Northern India. J Clin Diagn Res JCDR. 2013 Jan;7(1):127–31.

4. Sacco RL, Kasner SE, Broderick JP, Caplan LR, Connors JJ (Buddy), Culebras A, et al. An Updated Definition of Stroke for the 21st Century. Stroke. 2013 Jul 1;44(7):2064–89.

5. Stroke Facts | cdc.gov [Internet]. [cited 2016 Nov 2]. Available from: http://www.cdc.gov/stroke/facts.htm

6. Pandian JD, Sudhan P. Stroke Epidemiology and Stroke Care Services in India. J Stroke. 2013 Sep;15(3):128–34.

7. L. Kasper dennis, L. Hauser stephen, Jameson JL, s. fauci A, L. Longo dan, Loscalzo J. Harrison's Principles of internal Medicine. 19th edition. Vol. second. United ststes of america: McGraw-Hill education; 2015. 2565 p.

8. Hoffman M, MD. Brain (Human Anatomy): Picture, Function, Parts, Conditions, and More [Internet]. WebMD. [cited 2016 Nov 3]. Available from: http://www.webmd.com/brain/picture-of-the-brain

9. Brain Anatomy: Overview, Gross Anatomy: Cerebrum, Gross Anatomy: Cortex. 2016 Oct 28 [cited 2016 Nov 3]; Available from: http://emedicine.medscape.com/article/1898830-overview?pa=E0i3DFDTRC4a%2FexTvUUs3qgwx2egSNC15HDJt%2F17x49NuoXA01BpY6pxid1Jxk1c5sVYDIsCRzFeu3DDBP7823g%2B8AknVpbx2ndXJeAvD4w%3D

10. Cerebrovascular Diseases | Harrison's Principles of Internal Medicine, 19e | AccessMedicine | McGraw-Hill Medical [Internet]. [cited 2016 Nov 5]. Available from: http://accessmedicine.mhmedical.com/content.aspx?bookid=1130&sectionid=79755261

11. E.Barrett K, M. Barman S, L. Brooks H, Biotano S. Circulation Through Special Regions; Ganong's Review of Medical Physiology. Twenty-Third Edition. United ststes of america: The McGraw-Hil; 2010. 569 to 577.

12. Jones J. Internal carotid artery | Radiology Reference Article | Radiopaedia.org [Internet]. Radiopaedia. [cited 2016 Nov 6]. Available from: https://radiopaedia.org/articles/internal-carotid-artery-1

13. Gaillard F. Ophthalmic artery | Radiology Reference Article | Radiopaedia.org [Internet].

Radiopaedia. [cited 2016 Nov 6]. Available from: https://radiopaedia.org/articles/ophthalmic-artery

14. Jones J. Posterior communicating artery | Radiology Reference Article | Radiopaedia.org [Internet]. Radiopaedia. [cited 2016 Nov 6]. Available from: https://radiopaedia.org/articles/posterior-communicating-artery

15. Mudgal P. Anterior choroidal artery | Radiology Reference Article | Radiopaedia.org [Internet]. Radiopaedia. [cited 2016 Nov 7]. Available from: https://radiopaedia.org/articles/anterior-choroidal-artery

16. Gaillard F. Anterior cerebral artery | Radiology Reference Article | Radiopaedia.org [Internet]. Radiopaedia. [cited 2016 Nov 6]. Available from: https://radiopaedia.org/articles/anterior-cerebral-artery

17. Pai SB, Varma RG, Kulkarni RN. Microsurgical anatomy of the middle cerebral artery. Neurol India. 2005 Apr 1;53(2):186.

18. Jones J. Middle cerebral artery | Radiology Reference Article | Radiopaedia.org [Internet]. Radiopaedia. [cited 2016 Nov 6]. Available from: https://radiopaedia.org/articles/middle-cerebral-artery

19. Gaillard F. Vertebral artery | Radiology Reference Article | Radiopaedia.org [Internet]. Radiopaedia. [cited 2016 Nov 8]. Available from: https://radiopaedia.org/articles/vertebral-artery

20. Jones J. Basilar artery | Radiology Reference Article | Radiopaedia.org [Internet]. Radiopaedia. [cited 2016 Nov 9]. Available from: https://radiopaedia.org/articles/basilar-artery

21. Gaillard F. Posterior cerebral artery | Radiology Reference Article | Radiopaedia.org [Internet]. Radiopaedia. [cited 2016 Dec 16]. Available from: https://radiopaedia.org/articles/posterior-cerebral-artery

22. Circle of Willis Anatomy: Overview, Gross Anatomy, Natural Variants. 2016 Apr 7 [cited 2016 Nov 9]; Available from: http://emedicine.medscape.com/article/1877617-overview

23. Anvekar DB. Circle of Willis and its clinical significance [Internet]. [cited 2016 Nov 9]. Available from: http://www.neuroradiologycases.com/2012/07/circle-of-willis-and-its-clinical.html

24. Romero JR, Morris J, Pikula A. Stroke prevention: modifying risk factors. Ther Adv Cardiovasc Dis. 2008 Aug;2(4):287–303.

25. Dalal PM, Parab PV. Cerebrovascular disease in type 2 diabetes mellitus. Neurol India. 2002 Dec;50(4):380–5.

26. Sridharan R. Risk Factors for Ischemic Stroke: A Case Control Analysis. Neuroepidemiology. 1992 Apr 14;11(1):24–30.

27. Kernan WN, Inzucchi SE, Sawan C, Macko RF, Furie KL. Obesity. Stroke. 2013 Jan 1;44(1):278–86.

28. Rundek T, Sacco RL. Risk Factor Management to Prevent First Stroke. Neurol Clin. 2008 Nov;26(4):1007–ix.

29. Sundell L, Salomaa V, Vartiainen E, Poikolainen K, Laatikainen T. Increased Stroke Risk Is Related to a Binge Drinking Habit. Stroke. 2008 Dec 1;39(12):3179–84.

30. Stroke Prevention in Atrial Fibrillation - The Unmet Need and Morbidity Burden [Internet]. [cited 2016 Nov 11]. Available from: https://www.radcliffecardiology.com/articles/stroke-prevention-atrial-fibrillation-unmet-need-and-morbidity-burden

31. Sacco RL, Adams R, Albers G, Alberts MJ, Benavente O, Furie K, et al. Guidelines for Prevention of Stroke in Patients With Ischemic Stroke or Transient Ischemic Attack. Circulation. 2006 Mar 14;113(10):e409–49.

32. 869.full.pdf [Internet]. [cited 2016 Nov 13]. Available from: http://stroke.ahajournals.org/content/strokeaha/13/6/869.full.pdf

33. Simon JA, Hsia J, Cauley JA, Richards C, Harris F, Fong J, et al. Postmenopausal Hormone Therapy and Risk of Stroke. Circulation. 2001 Feb 6;103(5):638–42.

34. H.Ropper A, A.Samuels M, P.Klein J. Adams and Victor's Principles of Neurology. Tenth. United ststes of america: McGraw-Hill education; 2014. 836 p.

35. Hogue CW, Murphy SF, Schechtman KB, Dávila-Román VG. Risk Factors for Early or Delayed Stroke After Cardiac Surgery. Circulation. 1999 Aug 10;100(6):642–7.

36. Goldstein LB, Adams R, Becker K, Furberg CD, Gorelick PB, Hademenos G, et al. Primary Prevention of Ischemic Stroke. Stroke. 2001 Jan 1;32(1):280–99.

37. Dalal PM, Malik S, Bhattacharjee M, Trivedi ND, Vairale J, Bhat P, et al. Population-based stroke survey in Mumbai, India: incidence and 28-day case fatality. Neuroepidemiology. 2008;31(4):254–61.

38. Sridharan SE, Unnikrishnan JP, Sukumaran S, Sylaja PN, Nayak SD, Sarma PS, et al. Incidence, Types, Risk Factors, and Outcome of Stroke in a Developing Country. Stroke. 2009 Apr 1;40(4):1212–8.

39. Nagaraja D, Gururaj G, Girish N, Panda S, Roy AK, Sarma GRK, et al. Feasibility study of stroke surveillance: data from Bangalore, India. Indian J Med Res. 2009 Oct;130(4):396–403.

40. Reeves MJ, Bushnell CD, Howard G, Gargano JW, Duncan PW, Lynch G, et al. Sex differences in stroke: epidemiology, clinical presentation, medical care, and outcomes. Lancet Neurol. 2008 Oct;7(10):915–26.

41. H.Ropper A, A.Samuels M, P.Klein J. Adams and Victor's Principles of Neurology. Tenth. United ststes of america: McGraw-Hill education; 2014. 788 p.

42. L. Kasper dennis, L. Hauser stephen, Jameson JL, s. fauci A, L. Longo dan, Loscalzo J. Harrison's

Principles of internal Medicine. 19th edition. Vol. second. United ststes of america: McGraw-Hill education; 2015. 2560 p.

43. H.Ropper A, A.Samuels M, P.Klein J. Adams and Victor's Principles of Neurology. tenth. United ststes of america: McGraw-Hill education; 2014. 794 p.

44. Poh T. Time course of ischemic stroke on non-enhanced CT. [Internet]. Brain Stories. 2013 [cited 2016 Nov 19]. Available from: https://teddybrain.wordpress.com/2013/02/11/time-course-of-ischemic-stroke-on-non-enhanced-ct/

45. Gaillard F. Ischaemic stroke | Radiology Reference Article | Radiopaedia.org [Internet]. Radiopaedia. [cited 2016 Nov 19]. Available from: https://radiopaedia.org/articles/ischaemic-stroke

46. Birenbaum D, Bancroft LW, Felsberg GJ. Imaging in Acute Stroke. West J Emerg Med. 2011 Feb;12(1):67–76.

47. Sarkar S, Ghosh S, Ghosh SK, Collier A. Role of transcranial Doppler ultrasonography in stroke. Postgrad Med J. 2007 Nov;83(985):683–9.

48. Jauch EC, Saver JL, Adams HP, Bruno A, Connors JJ (Buddy), Demaerschalk BM, et al. Guidelines for the Early Management of Patients With Acute Ischemic Stroke. Stroke. 2013 Mar 1;44(3):870–947.

49. 40_advances_in_acute.pdf [Internet]. [cited 2016 Nov 19]. Available from: http://www.apiindia.org/pdf/medicine_update_2011/40_advances_in_acute.pdf

50. Murthy JMK. Thrombolysis for stroke in India: Miles to go….. Neurol India. 2007 Jan 1;55(1):3.

51. Ciccone A, Valvassori L, Nichelatti M, Sgoifo A, Ponzio M, Sterzi R, et al. Endovascular treatment for acute ischemic stroke. N Engl J Med. 2013 Mar 7;368(10):904–13.

52. Zhang Q, Wang C, Zheng M, Li Y, Li J, Zhang L, et al. Aspirin plus Clopidogrel as Secondary Prevention after Stroke or Transient Ischemic Attack: A Systematic Review and Meta-Analysis. Cerebrovasc Dis. 2014 Dec 24;39(1):13–22.

53. Wu T-C, Grotta JC. Hypothermia for acute ischaemic stroke. Lancet Neurol. 2013 Mar;12(3):275–84.

54. Savitz SI, Chopp M, Deans R, Carmichael T, Phinney D, Wechsler L, et al. Stem Cell Therapy as an Emerging Paradigm for Stroke (STEPS) II. Stroke J Cereb Circ. 2011 Mar;42(3):825–9.

55. L. Kasper dennis, L. Hauser stephen, Jameson JL, s. fauci A, L. Longo dan, Loscalzo J. Harrison's Principles of internal Medicine. 19th edition. Vol. second. United ststes of america: McGraw-Hill education; 2015. 2571 p.

56. L. Kasper dennis, L. Hauser stephen, Jameson JL, s. fauci A, L. Longo dan, Loscalzo J. Harrison's Principles of internal Medicine. 19th edition. Vol. second. United ststes of america: McGraw-Hill education; 2015. 2584,2585.

57. L. Kasper dennis, L. Hauser stephen, Jameson JL, L. Longo dan, Loscalzo J. Harrison's Principles of internal Medicine. 19th edition. Vol. second. United ststes of america: McGraw-Hill education; 2015. 2563 p.

58. 02_oa_a_study_on.pdf [Internet]. [cited 2016 Dec 25]. Available from: http://www.japi.org/october_2013/02_oa_a_study_on.pdf

59. NICE Guidelines need specific BMI cut–off limits for Asians. The BMJ [Internet]. 2016 Dec 15 [cited 2016 Dec 18]; Available from: http://www.bmj.com/content/349/bmj.g6608/rr/805398

60. Vaidya CV, Majmudar DK. A retrospective study of clinical profile of stroke patients from GMERS Medical College and Hospital, Gandhinagar, Gujarat. Int J Clin Trials. 2014 Aug 1;1(2):62–6.

61. Renjen PN, Beg MA, Ahmad K. Epidemiological study of incidence and risk factors of Ischemic stroke subtypes according to Trial of ORG 10172 in acute stroke treatment criteria: A 3 years, hospital-based study. Int J Med Public Health [Internet]. 2015 [cited 2016 Dec 15];5(1). Available from: /article/326

62. A prospective study of clinical profile of stroke in a tertiary care hospital.

63. Ng YS, Stein J, Ning M, Black-Schaffer RM. Comparison of Clinical Characteristics and Functional Outcomes of Ischemic Stroke in Different Vascular Territories. Stroke. 2007 Aug 1;38(8):2309–14.

64. Sidhartha JM, Purma AR, Reddy LVPK, Sagar NK, Teja MP, Subbaiah MV, et al. Risk factors for medical complications of acute hemorrhagic stroke. J Acute Dis. 2015 Aug;4(3):222–5.

65. Turtzo LC, McCullough LD. Sex Differences in Stroke. Cerebrovasc Dis Basel Switz. 2008 Nov;26(5):462–74.

ABBREVIATIONS

AHA	American Heart Associaton
BMI	Body mass index
CT	Computed Tomography
CNS	Central Nervous System
CABG	Coronary Artery Disease
CBF	Cerebral Blood Flow
CKD	Chronic Kidney Disease
DVT	Deep Vein Thrombosis
DALY	Disability Adjusted Life Years
HDL	High Density Lipoprotein
ICH	Intra Cerebral Haemorrhage
ICA	Internal Carotid Artery
ICP	Intra Cranial pressure
MCA	Middle Cerebral Artery
MRI	Magnetic Resonance Imaging
PCA	Posterior Cerebral Artery
PCOM	Posterior Communicating Artery
rtPA	Recombinant Tissue Plasminogen Activator
SAH	Sub Arachanoid Haemorrhage
TIA	Transient Ischemic Attack

STUDY PROFORMA

SL. NO:

- **NAME :** **AGE /SEX:** **IP NO:**
- **ADDRESS**
- **CONTACT NUMBER:**
- **DATE OF ADMISSION:** **DATE OF DISCHARGE/ DEATH:**
- **HISTORY OF PRESENTING ILLNESS :**

  - Altered sensorium, loss of consciousness
  - Convulsion, difficulty in concentration, memory, calculation, judgment
  - Depression/emotional disturbance
  - Speech disturbances
  - Weakness of upper and lower limb:
  - Hemiplegia/hemiparesis
  - Monoplegia/monoparesis
  - Crossed hemiplegia/hemiparesis
  - Stiffness of limbs
  - Involuntary movements
  - Gait disturbances
  - Sensory symptoms:
    - Touch
    - Numbness/ Hemianaesthesia/ Paresthesia
    - Cold/warmth
  - Abnormal sweating, bladder/bowel disturbance
  - Head ache, Vomiting
  - Dizziness, tinnitus
  - Cranial nerve symptoms:
    - Altered sensation of smell
    - Vision disturbance
    - Diplopia, squint, drooping of eyelids
    - Facial asymmetry/loss of taste sensation
    - Vertigo/tinnitus/deafness
    - Dysphagia
    - Hoarseness of voice
    - Dysarthria
  - **Onset**:
    - Sudden or insidious
    - Timing of onset: early morning or other time
    - Activity at the time of symptoms onset

PAST & PERSONAL HISTORY

Risk factors	Present / Duration	Absent
Systemic hypertension		
Diabetes mellitus		
CAD		
Dyslipidemia		
Atrial fibrillation		
h/o TIA or previous stroke		
Peripheral vascular disease		
Obesity		
Cigarette smoking/tobacco use		
Alcohol		
Illicit drug use		
OCP		
Physical inactivity		
Vascular malformation		
Congenital anomalies		
Polycythemia		

MENSTRUAL HISTORY : Age of menarche / parity /menopause: obtained / not yet obtained

FAMILY HISTORY: Parents / siblings with history of CVA / Hypertension / CAD / Diabetes mellitus

GENERAL EXAMINATION:-

Height:
Weight:
Body Mass index:
Pallor Icterus Clubbing Cyanosis
Pedal edema Lymphadenopathy
Neuro-cutaneus markers
Fundus examination:

VITAL SIGNS:-

Pulse
Blood pressure
Temperature

CNS EXAMINATION:

Higher mental function:

Handedness/Consciousness/0rientation/Emotional state
Speech/Memory/Intelligence

Cranial nerve examination:

Cranial Nerve	Right	Left
Olfactory		
Optic		
Oculomotor, Trochlear & Abducent		
Trigeminal		
Facial		
Vestibulocochlear		
Glossopharyngeal		
Vagus		
Spinal Accessory		
Hypoglossal		

Motor system:

Bulk/ Tone / Power of the muscle

Reflexes

Gait:

Sensory System:

Touch/ Pain & Temperature/Proprioception/Vibration sense/ Cortical sensation

Cerebellar signs:

Ataxia/ Intention tremor/ Dysdiadochokinesia/ Nystagmus

Meningeal signs:

PNS: any thickened nerves

Spine and Cranium:

Other systems examination:

Cardiovascular system:

Respiratory system:

Gastro intestinal system:

Musculoskeletal system:

INVESTIGATIONS:

Complete blood count	
Random blood sugar / FBS /PPBS	
HbA1C	
Fasting lipid profile	
Liver function test	
Renal function test	
Chest X ray	
ECG	
ECHO cardiogram	
4 vessel Doppler	
CT or MRI Brain	

FINAL DIAGNOSIS:

TYPE OF LESION:

Infarct (or)

Hemorrhage : SAH or intra parenchymal

LOCATION OF LESION:

Anterior cerebral artery territory

Middle cerebral artery territory

Posterior cerebral artery territory

TREATMENT GIVEN:

Medical

Surgical

CONDITION DURING HOSPITAL STAY & ON DISCHARGE :

Completely Recovered

Partially Recovered

Died

GOVT. STANLEY MEDICAL COLLEGE, CHENNAI- 600 001

INFORMED CONSENT

DISSERTATION TOPIC: "STUDY ON RISK FACTORS, CLINICO-RADIOLOGICAL PROFILE AND THE OUTCOME IN PATIENTS WITH ACUTE CEREBROVASCULAR DISEASE"

PLACE OF STUDY: GOVT. STANLEY MEDICAL COLLEGE, CHENNAI

NAME AND ADDRESS OF PATIENT:

I, _____ have been informed about the details of the study in my own language.

I have completely understood the details of the study.

I am aware of the possible risks and benefits, while taking part in the study.

I understand that I can withdraw from the study at any point of time and even then, I will continue to receive the medical treatment as usual.
I understand that I will not get any payment for taking part in this study.

I will not object if the results of this study are getting published in any medical journal, provided my personal identity is not revealed.

I know what I am supposed to do by taking part in this study and I assure that I would extend my full co-operation for this study.

Volunteer :	Witness :
Name and Address :	Name & Address :
Signature/Thumb impression :	Signature/thumb impression
Date:	Date :

Name and Signature of investigator:

GOVT. STANLEY MEDICAL COLLEGE , CHENNAI- 600 001

INFORMED CONSENT

DISSERTATION TOPIC: " STUDY ON RISK FACTORS,CLINICO-RADIOLOGICAL PROFILE AND THE OUTCOME IN PATIENTS WITH ACUTE CEREBROVASCULAR DISEASE"

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MASTER CHART

				CLINICAL HISTROY										CO-MORBID ILLNES		
	PERSONAL DETAILS			HEMIPLEGIA **/** HEMIPARESIS **/** MONOPLEGIA **/** MONOPARESIS	NUMBNESS	SLURRING /APHASIA(A)	HEADACHE **/** VOMITING **/** DIZZINESS	DEVIATION **OF** ANGLE	ONSET	LOC **/** SEIZURE	BLADDER **&** BOWEL I	GAIT DISTURBANCE	VISUAL DISTURBANCE	SHTN	DM	CAD
	NAME	AGE	SEX		Y/N		H/V/D	TO **R/L**	SUDDEN OR NOT	**L /+ S**	Y/N			Y/N & YEARS	Y/N & YEARS	Y/N & YEARS

1	Govindan	60	M	LEFT HEMIPLEGIA		Y		R								Y
2	Subramani	65	M	LEFT HEMIPLEGIA		Y		R		S			Y	16		
3	Shanmugam	75	M	RIGHT HEMIPLEGIA		A								20	20	
4	Pappammal	62	F	LEFT HEMIPARESIS										8	8	
5	Shanthi	62	F	RIGHT HEMIPARESIS		Y								12		
6	Kandhan	62	M	RIGHT HEMIPARESIS		Y		L						4	4	
7	Rajasekaran	55	M	LEFT HEMIPARESIS			D							6		
8	Balaraman	70	M	RIGHT HEMIPARESIS		Y			S							Y
9	Mohan	55	M	RIGHT HEMIPLEGIA	Y	Y	V+D							4		
10	Durairaj	56	M	RIGHT HEMIPLEGIA		Y								3	3	
11	Kannan	44	M	RIGHT HEMIPLEGIA				L						3		
12	Balan	59	M	LEFT HEMIPLEGIA		Y	V	R							4	
13	Vijayan	58	M						S	L+S	Y			8		
14	Jayakumari	56	F	LEFT HEMIPARESIS			H+V		S			Y		6	6	Y
15	Mohammed Ayub	57	M	LEFT HEMPLEGIA		Y	V							3		
16	Saroja bai	80	F	LEFT MONOPARESIS		Y		R						20	20	
17	Chandran	65	M	LEFT HEMIPARESIS		Y		R						5		
18	Mahendran	65	M	LEFT HEMIPLEGIA		Y		R		S				10		

19	Ramakrishnan	52	M				V		S			Y		7		
20	Nalini	50	F			Y		R							10	
21	Jayakanthi	67	F	RIGHT HEMIPLEGIA										9		
22	Poongavanam	65	F	LEFT HEMIPARESIS										8	10	
23	Janardhanan	66	M	RIGHT HEMIPARESIS			D		S					8	10	
24	Janaki	66	F			Y			S					1	3	
25	Dhanalakshmi	68	F	LEFT MONOPARESIS		Y		R						3		
26	Karpaga	72	F	RIGHT HEMIPLEGIA		A	D	L						4		
27	Parvathy	63	F	LEFT HEMIPARESIS				R						8	6	
28	Samykannu	83	M	RIGHT HEMIPAESIS				L								
29	Vasantha	61	F	LEFT HEMIPLEGIA												
30	Radhakrishnan	77	M	LEFT HEMIPARESIS			D							30	13	
31	Vimala	70	F	LEFT HEMIPARESIS		Y		R							15	
32	Abdul khadar	46	M	LEFT HEMIPLEGIA		Y		R							4	Y
33	Rajeswari	75	F				D					Y				
34	Subramani	55	M	RIGHT HEMIPLEGIA		Y		L								
35	Narayanamma	70	F	RIGHT HEMIPARESIS										15	10	Y
36	Devasigamani	59	F	RIGHT HEMIPLEGIA		Y	V+D	L						8	6	
37	Nasrullah	70	M	LEFT HEMIPLEGIA								Y				

38	Chellamma	65	F	LEFT HEMIPLEGIA			V+D	R	S		Y					
39	Shaheera	34	F	RIGHT HEMIPLEGIA						L+S						
40	Sangeetha	39	F	RIGHT HEMIPLEGIA		Y		L								
41	Khadija begum	73	F	LEFT HEMIPARESIS											20	
42	Krishnan	43	M	RIGHT HEMIPLEGIA		Y		L						5		
43	Kamala	66	F				H+V+D					Y		10	12	
44	Rani	55	F	LEFT HEMIPLEGIA			H							6		
45	Mohamood ibrahim	70	M	LEFT HEMIPLEGIA		Y	H							25	20	
46	Gomathi	60	F	RIGHT HEMIPARESIS		Y		L							10	
47	Elumalai	85	M	LEFT HEMIPLEGIA		Y		R	S							
48	Saroja	70	F			Y	H+V+D		S					21		
49	Sivagami	50	F	RIGHT HEMIPARESIS										5		
50	Vasantha	85	F	LEFT HEMIPLEGIA			V		S					30		
51	Muneera begum	75	F	RIGHT HEMIPLEGIA		Y										
52	Loganathan	61	M	LEFT HEMIPLEGIA										8	5	
53	Alphonsa	62	F	LEFT HEMIPLEGIA			V+D									
54	Abbas ali	58	M	RIGHT HEMIPARESIS			V+D							4	4	
55	Venkatesh	38	M	RIGHT HEMIPLEGIA		Y		L								
56	Lakshmi	67	F			Y	H+V+D								10	

57	Senthilraj	39	M						S	S						
58	Vasantha kumar	66	M	RIGHT HEMIPLEGIA		A		L						8	6	
59	Velakani	50	F	LEFT HEMIPLEGIA		Y		R						4		
60	Puspha	58	F	RIGHT HEMIPARESIS			H+V+D		S					5	1	
61	Ganesh	52	M	LEFT HEMIPLEGIA		Y		R							4	
62	Babu	52	M	RIGHT HEMIPLEGIA		Y	D							4	2	
63	Manjula	51	F	LEFT HEMIPLEGIA						L+S	Y			1	4	
64	Raman	51	M	LEFT HEMIPARESIS				R						3		
65	Devarajan	79	M			Y	V+D					Y		10	12	
66	Pethusamy	62	M	RIGHT HEMIPLEGIA		A		L						4	4	Y
67	Manoharan	60	M	RIGHT HEMIPLEGIA				L								
68	Pakkirasamy	83	M	RIGHT HEMIPLEGIA			H+V+D		S					4		Y
69	Anantharaj	59	M	RIGHT HEMIPARESIS		Y		L							4	
70	Ramakrishnan	52	M				V+D		S					8		
71	Soundara pandian	59	M		Y		H+V+D					Y		3		Y
72	Bhagyalakshmi	50	F	LEFT HEMIPARESIS		Y		R							5	
73	Loganathan	42	M	LEFT HEMIPARESIS		Y		L	S					4		
74	Devi	75	F	LEFT		Y	V									

				HEMIPLEGIA												
75	Ganeshan	70	M				H+V+D		S			Y		9	8	
76	Kasthuri	60	F				H +V			S					5	
77	Viswanathan	79	M	LEFT HEMIPARESIS			D							20	20	
78	Shanthi	65	F	RIGHT HEMIPARESIS		Y			S					8		
79	Ramalingam	61	M	LEFT HEMIPARESIS		Y										
80	Poongavanam	65	F	LEFT HEMIPLEGIA		Y								10	10	
81	Ambalagan	87	M				H+V+D		S	S				10		
82	Jayagandhi	67	F	RIGHT HEMIPLEGIA		Y		L								
83	Arokiya swamy	64	M	RIGHT HEMIPLEGIA											5	Y
84	Nanda kumar	55	M	RIGHT HEMIPLEGIA			V+D							8	8	Y
85	Mohan	53	M	LEFT HEMIPLEGIA			V							4		
86	Shakul hameed	65	M	LEFT HEMIPARESIS	Y				S							
87	Kanniyan	58	M	LEFT HEMIPARESIS		Y		R						4	3	Y
88	John	58	M			Y		R								
89	Mary	55	F	LEFT HEMIPLEGIA			D							5	5	
90	Dhanalakshmi	60	F	LEFT HEMIPLEGIA		Y	H+V+D						Y	10	10	5
91	Sundari	34	F	RIGHT HEMIPLEGIA		Y		L		S						

92	Sulochana	66	F	LEFT HEMIPLEGIA									6		
93	Yanathamma	58	F	LEFT HEMIPARESIS			H	R							
94	Sumaya begum	48	F				H+V+D			L	Y		4		
95	Jameela bai	65	F	RIGHT HEMIPLEGIA		Y							10	10	
96	Chandra	70	M	LEFT HEMIPARESIS		Y		R					20	15	#
97	Zaithum bee	75	F	RIGHT HEMIPLEGIA									10		
98	Victor john	53	M			Y		L						5	
99	Sasikala	59	F	RIGHT HEMIPLEGIA		Y		L					8	8	
100	Rama	53	F	LEFT HEMIPLEGIA	Y								4		
101	Chitty babu	62	M	RIGHT HEMIPLEGIA		Y							4	5	
102	Shanthi	40	F	LEFT HEMIPLEGIA		Y							2		
103	Rebecca	65	F	RIGHT HEMIPARESIS			D						8	8	
104	Muthuswamy	88	M	RIGHT HEMIPARESIS	Y										
105	Balaraman	60	M				V		S	S		Y	7		
106	Gunaseelan	55	M				D					Y	10	8	
107	Mariyam bee	57	F	LEFT HEMIPARESIS			D						8	10	
108	Deenadayalan	60	M	LEFT HEMIPLEGIA		Y				S			Y	16	13	
109	Veerammal	60	F	RIGHT HEMIPARESIS		Y		L						4	

110	Kuppammal	63	F	LEFT HEMIPLEGIA		Y		R							10	
111	Dasthagiri	70	M	LEFT HEMIPARESIS			D							6		
112	Surendran	68	M	RIGHTHEMIPARESIS		Y										Y
113	Narayanan	82	M	RIGHT HEMIPLEGIA	Y	Y	V+D		S					4		
114	Balasubramaniyan	63	M	RIGHT HEMIPLEGIA		Y								3		
115	Rani	70	F	LEFT HEMIPARESIS		Y		R								
116	Alaganathan	65	M	LEFT HEMIPLEGIA		Y		R								
117	Rama	53	F	RIGHT HEMIPLEGIA		Y		L							5	
118	Chinnaya	60	M	LEFT HEMIPARESIS										6		
119	Gnanasasikala	59	F	RIGHT HEMIPARESIS												
120	Devadoss	62	M	RIGHT HEMIPLEGIA		Y	V+D	L								
121	Duraikannu	72	M	LEFT HEMIPLEGIA								Y				
122	Krishna moorthy	61	M	LEFT HEMIPLEGIA			V+D	R	S		Y					
123	Balanarayan	56	M	RIGHT HEMIPLEGIA						L+S	Y					
124	Shanthi	40	F	RIGHT HEMIPLEGIA		Y		L								
125	Suseela	70	F	RIGHT HEMIPLEGIA		Y										

126	Ethirajan	63	M	LEFT HEMIPLEGIA										8	5	
127	Powly jose	64	F	LEFT HEMIPLEGIA			V+D									
128	Ramani	62	F	RIGHT HEMIPARESIS			V+D							4	4	
129	Sundaram	96	M	RIGHT HEMIPLEGIA		Y		L							4	
130	Bhagyalakshmi	48	F			Y	H+V+D							10	10	
131	Jayakumari	58	F	RIGHT HEMIPARESIS			H									
132	Rajeswari	67	F	RIGHT HEMIPLEGIA		A		L						8	6	
133	Abdul jilani	45	M	LEFT HEMIPLEGIA		Y		R						4		
134	Senthil kumar	45	M	RIGHT HEMIPARESIS			H+V+D		S						1	Y
135	Navaneetham	50	F	LEFT HEMIPARESIS										5	5	
136	Saraswathy	82	F	RIGHT HEMIPLEGIA		Y		L						20	20	
137	Prakash	40	M				H+V+D					Y				
138	Raman	63	M	LEFT HEMIPLEGIA			H							10	12	
139	Shaik masthan	50	M	LEFT HEMIPLEGIA		Y	H							6		
140	Kanakaraj	64	M	RIGHT HEMIPARESIS		Y		L							10	
141	Ananthan	76	M	LEFT HEMIPLEGIA		Y		R						25		

142	Ragupahty	70	M			Y	H+V+D		S						10	
143	Rajagopal	80	M	RIGHT HEMIPLEGIA		A									15	
144	Dhaani dharan	55	M	LEFT HEMIPLEGIA			V		S					8	8	
145	Chinnadurai	58	M				H+V+D			S						
146	Rathinasami	72	M	RIGHT HEMIPLEGIA		Y		L	S							
147	Shanmugam	75	M	RIGHT HEMIPLEGIA			V+D							10	15	
148	Govindan	55	M	RIGHT HEMIPLEGIA			V							5		Y
149	Ramachandran	73	M	LEFT HEMIPLEGIA											20	Y
150	Muruga malai	90	M	LEFT HEMIPARESIS											20	
151	Ravi	55	M	LEFT HEMIPARESIS		Y		R						6		
152	Mani	58	M			Y		R								Y
153	Rasul	84	M	LEFT HEMIPLEGIA			D								15	
154	Gajalakshmi	76	M	LEFT HEMIPLEGIA		Y	H+V+D						Y	10	10	
155	Usha rani	59	F	RIGHT HEMIPLEGIA		Y		L	S	S						
156	Sasikala	60	F	RIGHT HEMIPLEGIA										8	8	
157	Sekaran	51	M	LEFT HEMIPARESIS												
15	Munna	65	M	RIGHT		Y									10	

8				HEMIPARESIS												
159	Veeraiyan	75	M			Y								20		
160	Dhanasekaran	68	M	LEFT MONOPARESIS		Y		R						8	13	
161	Dhanaraj	62	M	RIGHT HEMIPLEGIA		A		L								
162	Ravi	60	M	LEFT HEMIPARESIS				R						15	5	
163	Michael	82	M	RIGHT HEMIPARESIS				L	S							
164	Vasantha	85	F	LEFT HEMIPLEGIA											15	
165	Govindammal	68	F	RIGHT HEMIPLEGIA										15	8	
166	Mani	67	F		Y		H					Y				Y
167	Safiya banu	83	F	LEFTHEMIPARESIS		Y		R						12	16	
168	Alamelu	58	F	LEFT HEMIPARESIS		Y		L						4		
169	Ramamoorthy	66	M	LEFT HEMIPLEGIA		Y	V									
170	Abdul Khader	70	M				H+V+D					Y		9	8	
171	Narasimaraj	63	M				H+V			S						
172	Ravindran	50	M	LEFT HEMIPARESIS			D							5		
173	Devansu	80	M	RIGHT HEMIPARESIS		Y									6	

174	Velumuruga n	69	M	LEFT HEMIPARESIS		Y									10	
175	Ganesh raju	55	M	LEFT HEMIPLEGIA		Y								5		
176	Thirunavakar asu	85	M	LEFT HEMIPLEGIA		Y		R								
177	Pachai	76	M	RIGHT HEMIPLEGIA		Y	D							10	10	
178	Devi	75	F	LEFT HEMIPLEGIA						L+S						
179	Nagarajan	79	M	LEFT HEMIPARESIS				R							8	
180	Jayaraj	80	M	RIGHT HEMIPLEGIA		Y	V									
181	Periyathambi	84	M	RIGHT HEMIPLEGIA		A		L	S					20		Y
182	Sankara kumar	80	M	RIGHT HEMIPLEGIA				L								
183	Hajarthaiah	62	M	RIGHT HEMIPLEGIA			H+V+D		S					8		Y
184	Ayub	51	M	RIGHT HEMIPARESIS		Y		L						4	4	
185	Bothammal	75	F					L	S					8		
186	Janaki	66	F	LEFT HEMIPLEGIA		Y	V	R							6	
187	Renuka	55	F							L+S				5		
188	Jayakumar	50	M		Y		V		S			Y				
189	Kuppu	60	M	LEFT HEMIPLEGIA		Y	V								15	

190	Giri	62	M			Y		R						10		
191	Perumal	60	M	LEFT HEMIPARESIS		Y		R								
192	Sundarajan	59	M	LEFT HEMIPLEGIA		Y		R						8	8	
193	Abul rafi	32	M			Y		R							4	
194	Muthu	51	F	RIGHT HEMIPARESIS			D							4		
195	Doraikannu	73	M	LEFT HEMIPLEGIA										15		
196	Koban raj	63	M	LEFT HEMIPARESIS			H	R								
197	Arumugam	55	M				H+V+D			L				5		
198	Rangarajan	66	M	RIGHT HEMIPLEGIA		Y									8	
199	Alagamuthu	65	M	LEFT HEMIPARESIS		Y		R						10	10	
200	Meenakshi	52	F	RIGHT HEMIPLEGIA		Y								4		