*DISSERTATION ON*

# A STUDY ON THE COMPARISON OF NIHSS, APACHE II, AND SCANDINAVIAN STROKE SCALE IN THE PREDICTION OF EARLY MORTALITY (≤7 DAYS POST STROKE) OF ACUTE ISCHEMIC STROKE PATIENTS – PREMISE - IN A TERTIARY CARE HOSPITAL

***Submitted in partial fulfilment of requirements for***

*M.D. DEGREE BRANCH 1 GENERAL MEDICINE OF*

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

*CHENNAI.*


**INSTITUTE OF INTERNAL MEDICINE**

**MADRAS MEDICAL COLLEGE CHENNAI – 600 003**

**MAY – 2023**

## CERTIFICATE - I

This is to certify that the dissertation entitled **"A STUDY ON THE COMPARISON OF NIHSS, APACHE II, AND SCANDINAVIAN STROKE SCALE IN THE PREDICTION OF EARLY MORTALITY (≤7 DAYS POST STROKE) OF ACUTE ISCHEMIC STROKE PATIENTS – PREMISE - IN A TERTIARY CARE HOSPITAL"** is a bonafide work done by **Dr. POOJA L,** Registration Number **200120100522** at Madras Medical College, Chennai in partial fulfilment of the university rules and regulations for award of M.D., Degree in General Medicine (Branch-I) under our guidance and supervision during the academic year 2020-2023.

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## PLAGIARISM CERTIFICATE - II

This is to certify that this dissertation work titled '**A STUDY ON THE COMPARISON OF NIHSS, APACHE II, AND SCANDINAVIAN STROKE SCALE IN THE PREDICTION OF EARLY MORTALITY (≤7 DAYS POST STROKE) OF ACUTE ISCHEMIC STROKE PATIENTS – PREMISE - IN A TERTIARY CARE HOSPITAL'** of the candidate **Dr. POOJA L** with Registration Number **200120100522** for the award of **DOCTOR OF MEDICINE** 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 **14% percentage** of plagiarism in the dissertation.

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I, **Dr. POOJA L,** Registration number **200120100522** solemnly declare that this dissertation entitled **"A STUDY ON THE COMPARISON OF NIHSS, APACHE II, AND SCANDINAVIAN STROKE SCALE IN THE PREDICTION OF EARLY MORTALITY (≤7 DAYS POST STROKE) OF ACUTE ISCHEMIC STROKE PATIENTS – PREMISE - IN A TERTIARY CARE HOSPITAL"** was done by me at Madras Medical College and Rajiv Gandhi Government General Hospital, Chennai during 2020-2023 under the guidance and supervision of my chief **Prof. Dr. M. ANUSUYA, M.D.** This dissertation is submitted to the Tamil Nadu Dr.M.G.R. Medical University towards the partial fulfilment of requirements for the award of M.D. Degree in General Medicine (Branch-I)

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**- Dr POOJA L.**

## ABBREVIATIONS

| | | |
|---|---|---|
| MRS | - | Modified Rankin Score |
| NIHSS | - | National Institutes of Health Stroke Scale |
| PREMISE | - | Predicting Early Mortality of Ischemic Stroke |
| SSS | - | Scandinavian Stroke Scale |
| TIA | - | Transient Ischemic Attack |
| APACHE II | - | Acute Physiology and Chronic Health Evaluation II |
| NPCDC | - | National Programme for Prevention and Control of Cancer, Diabetes, Cardiovascular Diseases & Stroke |
| DALY | - | Disability-Adjusted Life Years |
| APS | - | Acute Physiology Score |
| ICMR | - | Indian Council of Medical Research |
| DM | - | Diabetes Mellitus |
| HTN | - | Systemic Hypertension |
| AF | - | Atrial Fibrillation |

| | | |
|---|---|---|
| MI | - | Myocardial Infarction |
| ICH | - | Intra Cerebral Hemorrhage |
| LDL | - | Low Density Lipoprotein |
| HDL | - | High Density Lipoprotein |
| Lp(a) | - | Lipoprotein A |
| AIS | - | Acute Ischemic Stroke |
| ACA | - | Anterior Cerebral Artery |
| PCA | - | Posterior Cerebral Artery |
| HIE | - | Hypoxic Ischemic Encephalopathy |
| NMDA | - | N-Methyl-D-Aspartate |
| AMPA | - | Alpha- Amino-3-Hydroxy-5-methyl-4-Isoxanole propionate |
| SWD | - | Spontaneous Waves of Depolarization |
| AHA/ASA | - | American Heart Association / American Stroke Association |
| PPV | - | Positive Predictive Value |
| NPV | - | Negative Predictive Value |

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# ABSTRACT

## ➢ BACKGROUND:

Stroke has been the second greatest cause of death worldwide and one of the major causes of mortality and morbidity in adults. The estimated 30-day case fatality rate following a first ischemic stroke varies widely across nations, although it is expected to be between 16 and 23% globally. Among these, much of the mortality occurs less than 7 days' post-stroke. It is therefore essential to have PREMISE Score that helps in predicting the early stroke mortality. One of the few eminent scales of neurological assessment useful in predicting early mortality of stroke patients are the NIHSS, APACHE II AND SCANDINAVIAN STROKE SCALE.

## ➢ OBJECTIVES:

To compare the efficiency of NIHSS, APACHE II AND SCANDINAVIAN STROKE SCALE in predicting the early mortality (≤7 days' post stroke) of acute ischemic stroke patients – Premise - admitted in a tertiary care hospital.

## ➢ METHODOLOGY:

This was a prospective observational study conducted in the Madras Medical College and Rajiv Gandhi Government General Hospital, Chennai for 6 months (May 2022 to October 2022). The study included 200 patients with Acute Ischemic Stroke, who met the inclusion and exclusion criteria. A detailed clinical history was elicited, neurological examination was recorded and baseline blood investigations were done. The APACHE II, NIHSS and

SCANDINAVIAN STROKE SCALE scores were assessed at admission within the first day (24 hours) of stroke. Outcome of the patient was assessed as discharge/death from the hospital.

- **RESULTS:**

ROC curve was generated to predict the cut-off value of these scores in patients who died. In NIHSS score, the cut-off value was 11.5, with an AUC of 0.737. In SSS Score, the cut-off value was 16.5, with an AUC of 0.852. In APACHE II score, the cut-off value was 12.5, with an AUC of 0.794. All these had a significant p-value of <0.0001. Even though the AUC for APACHE Ⅱ Score and SSS were slightly larger when compared with NIHSS, there were no statistically significant differences between these scales. The PPV - NPV of both SSS Scores and APACHE II were 74.49% - 75.49% and 73.68% - 73.33% respectively. The PPV and NPV of NIHSS Score were 65.74% - 70.65% respectively. The sensitivity and specificity of NIHSS were 72.45% and 63.73% respectively. The accuracy was 68%. The sensitivity and specificity of SSS were 74.49% and 75.49% respectively. The accuracy was 75%. The sensitivity and specificity of APACHE II were 71.43% and 75.49% respectively. The accuracy was 73.50%.

- **CONCLUSION:**

From the results obtained from this study, it was found that the scores SSS and APACHE II were equally as good as the NIHSS Score in the prediction of early mortality (≤7 days' post stroke) of acute ischemic stroke patients.

# INTRODUCTION

Stroke, a disabling cerebrovascular illness, has been the second greatest cause of death worldwide and one of the major causes of mortality and morbidity in adults. In high-income nations, the 30-day death rate of ischemic stroke has been calculated to be 15%. Most of the research concentrated on mid- and long-term mortality. A limited number of research have concentrated on in-hospital death rates pertaining to a median hospital stay of 1 to 2 weeks, and there had been little information available regarding the frequency and risk factors for early mortality in the first week.

However, early mortality predictors have received little attention to-date. This has been problematic because the initial few days following an acute event call for more accurate prognostication of bad outcomes, including death. Such information would have an impact on management choices, which may vary from recognising the need for increased monitoring to withdrawal from maximal therapy. Additionally, it would be very useful in informing partners and family members about the potential outcome of the stroke victim. [(1)]

Predicting Early Mortality of Ischemic Stroke (PREMISE), a straightforward scoring instrument, had been created as a clinically relevant tool to deliver these insights. [(2)]

In a multivariate analysis, characteristics that were independently linked with stroke unit mortality were used to create the PREMISE score. Age, admission NIHSS, pre-stroke MRS (Modified Rankin Score) >0, heart disease (defined as coronary artery disease, heart failure,

cardiomyopathy, or valve disease), diabetes mellitus, posterior circulation stroke syndrome (compared with anterior circulation stroke syndromes), and non-lacunar stroke – all these variables remained independently associated with a higher risk of early stroke unit mortality.

One of the factors in the PREMISE score - the National Institutes of Health Stroke Scale (NIHSS) has been widely used to assess the prognosis and degree of neurological deficit in stroke cases all over the world. Currently, the NIHSS scoring system had been frequently used to forecast mortality, assisting doctors in actively intervening to improve the prognosis of critically ill patients. Compared to other stroke scales, such as the Canadian neurological scale, the NIHSS had proved to be more accurate at predicting early mortality (mortality within 7 days or less). Additionally, a baseline NIHSS score of 6 had been linked to a high likelihood of a successful recovery.

Only 11 indicators of neurological function were scored by the NIHSS, and the approach ignored prognosticating variables including age and chronic health condition. Additionally, several NIHSS signs could not be tested always, particularly in patients presenting with reduced sensorium.

An alternative stroke measure that had been widely used in Scandinavian nations and had recently undergone validation was called the Scandinavian stroke scale (SSS). Neurological recovery, as assessed by the Scandinavian stroke scale in the first week following the beginning of the stroke, was found to be an independent predictor of early mortality and functional outcome in a multivariate logistic regression model. The SSS had the advantage of

being straightforward making it simple to carry out repeated measures immediately following an acute stroke phase.

The SSS was equally effective as the Gold standard score, NIHSS, in a 2016 prospective trial which included patients with acute stroke, in identifying individuals who had died or were dependent at 3-month follow-up. Inter-rater reliability was excellent for conscious level, orientation, and gait (kappa values of 0.84, 0.86, and 1.0, respectively) and moderate for facial palsy (kappa 0.59) [(3)]

The intensive care unit (ICU) had been using the Acute Physiology and Chronic Health Disease Classification System II (APACHE II) as a scientific, unbiased, and trustworthy score system to assess the severity of critically sick patients' illnesses and predict their prognosis. It had become the most widely used critical disease score system in the world since it was first used in clinical settings in 1985. The APACHE II considered physiological scores, health status, age, which could also influence the likelihood of death and the prognosis following a stroke. According to Chen et al., the APACHE II score had significant clinical importance and could be used for reasonable clinical assessment of illness and assessing the prognosis of acute ischemic stroke patients. [(4)]

This study intended to compare Acute Physiology and Chronic Health Evaluation (APACHE) II, National Institutes of Health Stroke Score (NIHSS), and SCANDINAVIAN STROKE SCALE in better prediction of stroke severity. The patients will be comprehensively assessed to compare the application values of these scoring systems in the prediction of early mortality of stroke, providing reference for the correct selection of stroke

scale. Hence this study will be conducted to compare the efficiency of NIHSS, APACHE II AND SCANDINAVIAN STROKE SCALE in the prediction of early mortality (≤7 days' post stroke) of acute ischemic stroke patients - Premise admitted in a tertiary care hospital.

## AIMS AND OBJECTIVES

To compare the efficiency of NIHSS, APACHE II AND SCANDINAVIAN STROKE SCALE in predicting the early mortality (≤7 days' post stroke) of acute ischemic stroke patients – Premise - admitted in a tertiary care hospital.

# REVIEW OF LITERATURE

It is becoming more widely recognized that developing countries are going through an epidemiologic transition akin to what happened in industrialized countries decades ago. While infectious diseases continue to be the leading cause of morbidity and mortality, chronic non-communicable diseases are on the rise, especially in the most developed developing nations, and are predicted to lead infectious diseases in the next decade or two. The majority of these illnesses, including coronary heart disease, stroke, and diabetes, are influenced by nutrition, smoking and drinking. [5]

The nervous system is the most complex system of our body executing multiple complex functions simultaneously through trillions of specialised cells and their processes called the neurons or the nerve cells. The cerebral cortex consists of many areas assigned to perform specific functions. One illness that has aided in our better understanding of the human brain is stroke. [6]

## ❖ DEFINITION:

Stroke was first described by the “the father of medicine” - Hippocrates in the 5th century BC. He called it “apoplexy” which in Greek meant “struck down by violence,” describing a person who suddenly falls and becomes unconscious. William Cole possibly used the term "stroke" for the first time in medicine when he published,” A Physico-Medical Essay Concerning the Late Frequencies of Apoplexies” in 1689. In 1970, the World Health Organization defined stroke as 'Rapidly developed 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 of vascular origin'.

The American Heart Association and American Stroke Association described the term "stroke" should be broadly used to include all of the following: Definition of CNS infarction: CNS infarction is brain, spinal cord, or retinal cell death attributable to ischemia, based on 1. pathological, imaging, or other objective evidence of cerebral, spinal cord, or retinal focal ischemic injury in a defined vascular distribution; or 2. clinical evidence of cerebral, spinal cord, or retinal focal ischemic injury based on symptoms persisting ≥24 hours or until death, and other aetiologies excluded. This includes both cerebral infarction or intracerebral and subarachnoid haemorrhage. A time window of 24 h distinguishes stroke from transient ischaemic attack (TIA), which is defined as a neurological deficit lasting less than 24 h. All vascular diseases affecting the brain, such as stroke, vascular dementia, and asymptomatic cerebrovascular disease, are together referred to as cerebrovascular disease. [7]

## ❖ EPIDEMIOLOGY:

According to National Programme for Prevention and Control of Cancer, Diabetes, Cardiovascular Diseases & Stroke (NPCDCS), stroke was the second leading cause of death worldwide in 2013 resulting in 6.5 million fatalities and 113 million DALYs. Over two thirds of these fatalities happened in developing nations. By 2050, over 80% of the anticipated worldwide burden of new strokes of 15 million will take place in developing and middle-income nations. Studies estimate that in India, the incidence of stroke in the general

population ranges from 116 to 163 per 100,000 people. According to ICMR, a report titled "India: Health of the Nation's States" has been released wherein stroke was the fifth-leading cause of disability-adjusted life Years (DALY) and the fourth-leading cause of death in 2016.

❖ **<u>TYPES OF STROKE</u>**

• ISCHEMIC -85%

• HEMORRHAGIC- 15%

- ISCHEMIC STROKE ETIOLOGY –
  - ❖ Thrombosis
    - ➢ Large vessel thrombosis
    - ➢ Lacunar (small vessel) stroke
    - ➢ Dehydration
  - ❖ Embolic occlusion
    - ➢ Artery to artery (Carotid bifurcation, Aortic arch, Arterial dissection).
    - ➢ Cardio-embolic (Atrial fibrillation, Mural thrombus, Myocardial infarction, Dilated cardiomyopathy, Valvular lesion like mitral stenosis; Mechanical valve; Infective endocarditis, Paradoxical embolus like Atrial septal defect; Patent foramen ovale, Atrial septal aneurysm, Spontaneous echo contrast).

- HEMORRHAGIC STROKE TYPES
    - ❖ Subdural / Epidural Haemorrhage
    - ❖ Subarachnoid haemorrhage
    - ❖ Intra-parenchymal haemorrhage

➢ UNCOMMON CAUSES:

- ❖ Hypercoagulable Disorders
- ❖ Protein C and S deficiency
- ❖ Anti-thrombin III deficiency
- ❖ Antiphospholipid antibody syndrome
- ❖ Factor V Leiden mutation
- ❖ Prothrombin G20210 mutation
- ❖ Systemic malignancy
- ❖ Sickle cell anaemia
- ❖ Beta thalassemia
- ❖ Polycythaemia Vera
- ❖ Homocysteinemia
- ❖ Disseminated intravascular coagulation
- ❖ Nephrotic syndrome
- ❖ Systemic lupus erythematosus
- ❖ Venous sinus thrombosis
- ❖ Fibromuscular dysplasia

- Vasculitis
- Drugs - Cocaine Amphetamine

## RISK FACTORS FOR STROKE

- NON MODIFIABLE RISK FACTORS:
  - ✓ Age
  - ✓ Sex
  - ✓ Ethnicity
  - ✓ Genetic causes
- MODIFIABLE RISK FACTORS:
  - ✓ Hypertension
  - ✓ Cardiac disease
  - ✓ Diabetes mellitus
  - ✓ Dyslipidaemia
  - ✓ Smoking
  - ✓ Alcoholism
  - ✓ Coagulation disorders
  - ✓ Oral contraceptive pills
  - ✓ Hyperhomocysteinemia
  - ✓ Migraine
  - ✓ Physical activity
  - ✓ Diet

## 1. AGE

The most significant risk factor for stroke is age. After the age of 55, the stroke rate doubles for both men and women every ten years. [8]

## 2. GENDER

The risk of stroke is higher for men. With the exception of the older ages, men's risk of stroke is 1.3 times greater than women's. At a high age of 80 to 85 years, the gender-based risk difference vanishes. When it comes to subarachnoid haemorrhage, women are at higher risk than men. [9]

## 3. ETHNICITY

People of African descent are at greater risk than Caucasians. This is due to ineffective management of modifiable risk variables. Chinese people have a higher rate of intracerebral haemorrhage than Caucasians do have. [10] East Asians and African Americans have higher rates of intracranial artery stenosis than Caucasians do in ischemic stroke.

## 4. GENETIC CAUSES

- CADASIL-notch-3 gene - Cerebral autosomal dominant arteriopathy with subcortical infarcts and leuko encephalopathy
- CARASIL-HTRA gene - cerebral autosomal recessive arteriopathy and leuko encephalopathy

- MELAS - mitochondrial myopathy encephalopathy, lactic acidosis, and stroke like episodes
- HANAC (hereditary angiopathy, nephropathy, aneurysm, and muscle cramps syndrome; COL4A1 mutation) [11]
- Homocystinuria, Fabry disease (alpha-galactosidase gene), Ehlers Danlos syndrome type IV, Marfan syndrome, pseudoxanthoma elasticum.
    - Stroke or a stroke-like episode is one of the characteristics of each of the aforementioned syndromes'.
- Childhood stroke incidence is increased by sickle cell anaemia.
- Children with sickle cell anaemia are more at risk due to higher middle cerebral artery transcranial ultrasonography velocities. [12]

## 5. HYPERTENSION

A significant modifiable risk factor for stroke is an increase in blood pressure. Both ischemic and haemorrhagic stroke are strongly and independently correlated with it.

As people get older, hypertension is more common. According to a meta-analysis including one million adults in 61 observational studies, there is a doubling of death from ischemic heart disease and stroke for every increase of 20 mm Hg systolic or 10 mm Hg diastolic blood pressure. [13]

## 6. DIABETES MELLITUS

Diabetes mellitus damages arterial blood vessels and increases the chance of having an ischemic stroke. DM raises the risk of Lacunar infarcts and recurrent stroke. [14]

Endothelial proliferation and thickening of the plasmatic membrane in small blood arteries (known as "lipo-hyalinosis"), which causes lacunar infarction, are thought to be caused by excessive glycation and oxidation, endothelial dysfunction, and enhanced platelet aggregation. According to the Framingham study, people who have glucose intolerance, have twice the risk of stroke when compared to non-diabetics. Diabetes significantly increases the risk of stroke due to hyperinsulinemia and enhanced insulin resistance. [15]

## 7. CARDIAC DISEASES

A significant risk factor for stroke is atrial fibrillation. Age raises both the prevalence and incidence of atrial fibrillation. [16]

According to The Framingham study, the attributable risk of AF for stroke increased from 1.5% in people between the ages of 50 and 59 to 23.5% in those between the ages of 80 and 89. Atrial fibrillation is proven to be the cause of over half of cardio embolic strokes. The risk of stroke is reduced by 68% by warfarin anticoagulation, according to a pooled review of AF studies.

Stroke risk is increased by cardiac valve defects including mitral valve prolapse, mitral stenosis and the calcification of the mitral annulus. Myocardial infarction and patent foramen ovale are additional risk factors for stroke. Following angioplasty and cardiac catheterization, there is a 0.2% to 0.3% stroke risk. Embolic problems can arise after radiofrequency ablation, pacing, electrophysiological treatments, and cardioversion.

Patients with uncomplicated MI are less likely to experience stroke or systemic embolism, but up to 12% of those with acute MI accompanied by a Left ventricular thrombus may do so. The rate can approach 20% in people with significant antero-apical infarcts and is higher in those with anterior than inferior infarcts. Cardiomyopathy brought on by hereditary or ischemia causes reduction in stroke volume resulting in a relative stasis inside the left ventricle that may activate coagulation processes and raise the risk of thromboembolic event while also impairing left ventricle systolic performance. In individuals with Valvular heart disease, antithrombotic treatment can lessen but not completely eliminate the risk of stroke and systemic embolism. [17]

## 8. DYSLIPIDEMIA

A known risk factor for cerebrovascular accident is abnormal lipid profiles. Atherosclerosis of the carotid arteries, which is linked to elevated cholesterol levels, raises the risk of brain infarcts brought on by large vessel disease. [18] On the other side, ICH risk is increased by low cholesterol levels. The risk of ICH is marginally increased in stroke patients who undergo severe cholesterol reduction.

High levels of fibrinogen are linked to high cholesterol, LDL, Lp(a), and raised triglyceride levels, which is consistent with higher thrombotic consequences in dyslipidaemias. The risk of stroke and total- and LDL- cholesterol are positively correlated, while HDL- cholesterol plays a preventive function in extra-cranial atherosclerosis. [19] In both men and women, the elderly, and among many racial and

ethnic groupings, higher levels of HDL cholesterol are linked to a lower risk of ischemic stroke. These findings confirm HDL cholesterol as a significant modifiable stroke risk factor.

## 9. SMOKING

In addition to raising the risk of ICH, smoking cigarettes raises the risk of ischemic stroke. [20] The risk is increased by both active and passive smoking. [21] Smokers can cut their risk of stroke by up to 50% by quitting.

## 10. ALCOHOLISM

Large amounts of alcohol consumption increase the risk of stroke. Alcohol abstainers may experience a somewhat reduced stroke risk. It's probable that short-term strong alcohol use raises the risk of a sudden stroke. [22] This could be caused by a rise in blood pressure, atrial fibrillation, or myocardial damage brought on by cardiomyopathy.

## 11. COAGULATION DISORDERS

Antiphospholipid antibody and lupus anticoagulant have been associated with ischaemic stroke. Coagulation disorder causing a venous thrombosis may give rise to paradoxical embolism through patent foramen ovale.

## 12. ORAL CONTRACEPTIVE PILLS

There is a high risk for stroke with oral contraceptives containing oestrogen content > 50micrograms.However low dose oestrogens disclosed no increased risk of stroke [23]

## 13. HOMOCYSTEINE

Homocysteine levels have been strongly linked to stroke in middle-aged men, according to a recent British Regional Heart Study. The biological plausibility that elevated homocysteine levels are atherogenic and pro-thrombotic is very strong. Although vitamins B6, B12, and folic acid lower homocysteine levels, there is insufficient evidence to support the claim that these measures will lower the frequency of stroke. [24]

## 14. MIGRAINE

A migraine attack may be complicated by a stroke. In young women, migraine itself seems to be a risk factor for stroke. [25] There is a clear correlation between migraine and stroke. The incidence of stroke rose from 10 per 1 lakh woman-years to 19 per 1 lakh woman-years in women if migraine was present. The risk of stroke related with migraines is negligible, though.

## 15. PHYSICAL ACTIVITY

Industrialization completely transformed human living style in the recent century, favouring sedentary lifestyles. There is strong evidence that exercise reduces a variety of cardiovascular disease risk factors, including those for stroke. Men and women who were moderately active had a 20% lower risk, while people who were very active had a 27% lower risk. A minimum of 30 minutes of daily exercise reduces the relative risk of stroke by 0.69 to 0.74. [26] By lowering weight, blood pressure, pulse rate, elevating HDL, enhancing insulin sensitivity, and decreasing platelet aggregability, it protects against atherosclerotic disease. High blood pressure, atherosclerosis and high glucose levels in

the serum are all important risk factors for the occurrence of stroke and are connected with obesity.

## 16. DIET

- Both ischemic and haemorrhagic stroke may be protected against by eating fruit and vegetables. A relative risk of stroke was 0.89 for individuals who consumed three to five servings of fruit and vegetables per day as opposed to those who consumed fewer portions. [27]
- Consuming more fish, milk, and green tea can reduce your risk of having a stroke. High-fat, high-cholesterol diets should be avoided.
- Consuming more salt is linked to higher blood pressure. A 100 mmol increase in salt intake is predicted to raise blood pressure by 10 mmHg, which corresponds to a 34% greater risk of stroke.
- A lower risk of stroke is linked to higher levels of vitamin C (high fruit and vegetable intake), which works through an antioxidant effect. Folate may help lower the risk of stroke by lowering homocysteine levels.

## 17. SERUM FIBRINOGEN

Increased plasma fibrinogen is strongly associated with stroke. Smoking rises fibrinogen levels, which in turn increases the risk of stroke. [28]

## ❖ BLOOD SUPPLY OF BRAIN

Ischemic brain stroke is due to sudden cessation of blood supply to the brain, which produces death of the corresponding neuronal cell and loss of neurological function. A thrombus or an embolus that completely occludes an artery in the brain causes an acute ischemic stroke (AIS). AIS occurs more frequently than haemorrhagic stroke. In order to enhance patient prognosis and to reduce morbidity and mortality, it is essential to diagnose acute brain stroke as soon as possible.

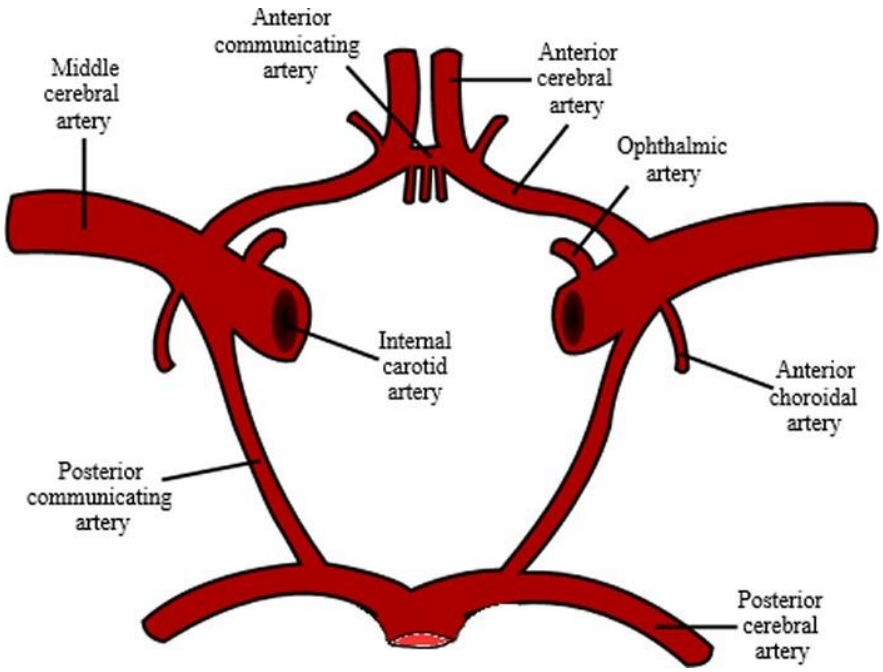
The arterial supply of the brain is from 2 pairs of blood vessels namely the vertebral arteries form the posterior circulation and the internal carotid arteries form the anterior circulation. Both anterior and posterior circulations interconnect in the base of the cranial cavity and produce the Circle of Willis.

ANTERIOR CEREBRAL CIRCULATION - The Internal Carotid Artery from the common carotid artery proceeds superiorly to enter into the cranial cavity through the carotid canal. Each internal carotid artery releases the anterior cerebral artery, middle cerebral artery, posterior connecting artery, and ophthalmic artery upon entering the cranial cavity. The medial half of the orbital surface with the exception of the occipital lobe and the entire medial surface of the cerebral hemisphere, are supplied by the anterior cerebral artery.

A major branch of the ACA called the Recurrent Artery of Heubner produces facio-brachial monoplegia when it is affected. The entire lateral surface of cerebrum including lateral half of orbital surface, the medial half of orbital surface, lower temporal and occipital pole is supplied

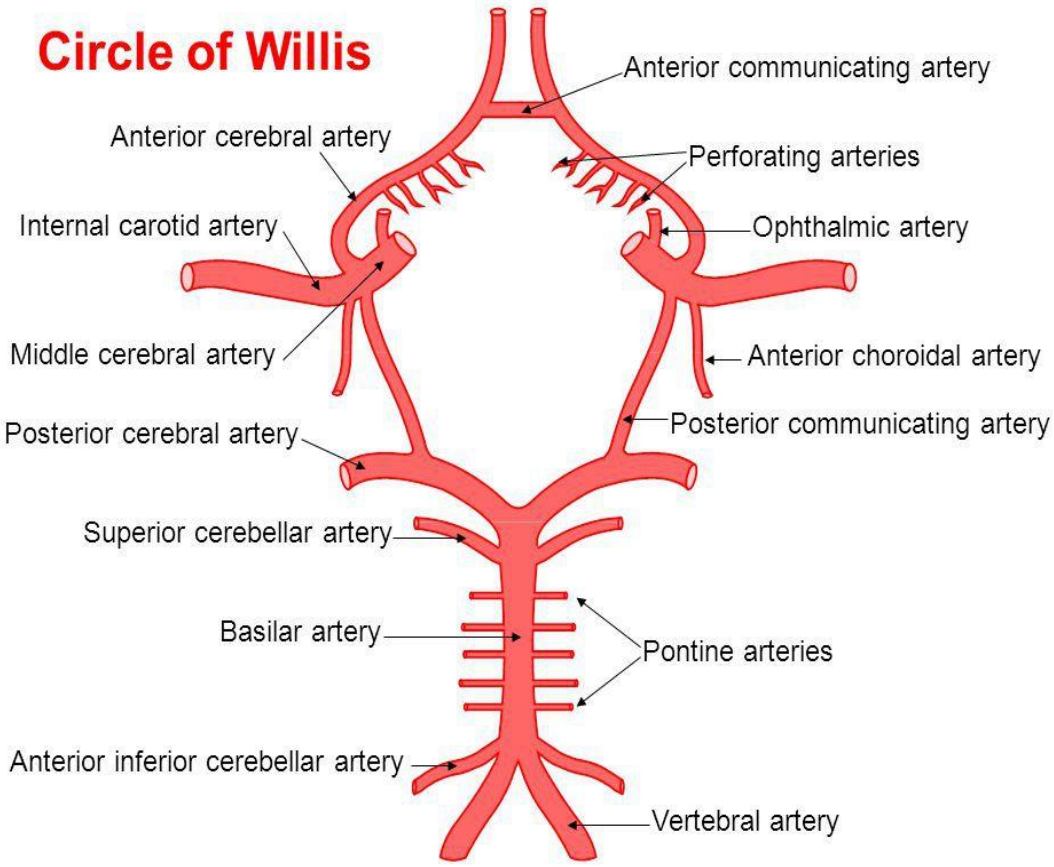
by the middle cerebral artery. The Posterior cerebral artery supplies the medial surface of temporal and occipital lobes and their tentorial surface and also supplying cerebellum, brainstem, midbrain and thalamus.

## INTERNAL CAROTID ARTERY AND ITS BRANCHES




POSTERIOR CEREBRAL CIRCULATION – Each Vertebral artery, arising from the subclavian artery, through the foramen magnum enters the cranial cavity. Each vertebral artery gives a small meningeal branch and 3 additional branches namely the Anterior spinal artery, Posterior spinal artery and Posterior inferior cerebellar artery and eventually joins on either side to form the basilar artery. The Basilar artery further bifurcates into 2 posterior cerebral arteries.

CIRCLE OF WILLIS - Circle of Willis is formed by the branches from vertebra-basilar arteries and internal carotid systems of arteries at the base of the brain. These inter-connections are formed by an anterior communicating artery which interconnects 2 anterior cerebral arteries, 2 posterior communicating arteries one on each side connecting the posterior cerebral artery with the internal carotid artery.




The circle of Willis also called circulus arteriosus cerebri is an anastomotic system of arteries located at the base of the brain. The “circle” was named after Thomas Willis a pupil of Richard Lowe. The forebrain and hindbrain's blood supply are from the circle of Willis, which surrounds the stalk of the pituitary gland. The circle of Willis is formed when the internal

carotid artery divides into the anterior cerebral artery and middle cerebral artery. The anterior cerebral arteries are then connected by an anterior communicating artery. They form the anterior half (anterior circulation) of the circle of Willis. Posteriorly, the basilar artery, formed by the left and right vertebral arteries, further 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. [29]

## ❖ PATHOPHYSIOLOGY OF ISCHEMIC STROKE

### CEREBRAL BLOOD FLOW

The average cerebral perfusion is 50–60 ml per 100 g per minute. The effects of brain ischemia are seen quickly because neurons cannot generate energy anaerobically and there is no glucose stored in the brain. Vascular auto-regulatory mechanisms respond to ischemia by increasing local vasodilatation, opening collaterals, and utilising more oxygen and glucose from the blood. [30]

- Within 4 to 10 minutes after cerebral perfusion reaches nil, brain tissue will die.
- Irreversible neuronal injury occurs when brain tissue perfusion is reduced to less than 10ml/100gm/min.
- Infarction occurs in less than an hour if the rate drops to less than 16 to 18ml/ 100 g/min.
- Electrical silence and a reduction in synaptic activity occur when blood flow falls to less than 20ml/100g/min in order to protect energy reserves. If this persists over several hours or days, it causes ischemia without infarction. [31]

It is known as a Transient Ischemic Attack (TIA) if reperfusion occurs before considerable amount of cell death, which could result in only temporary symptoms.

In order to meet the criteria for a TIA, all neurological symptoms must go away within 24 hours, regardless of whether imaging tests reveal a fresh permanent brain injury.

Systemic hypotension causes a generalised decrease in cerebral blood flow, which leads to syncope. Infarction forms at the border zone of major artery territories if the reduced blood flow lasts for an extended period of time. The condition known as hypoxic ischemic encephalopathy (HIE) is caused by severe global hypoxia ischemia. [30]

## FOCAL ISCHEMIC INJURY

Ischemia of the afflicted vascular region might result from a thrombus or embolus-induced vascular obstruction. At the level of the gross tissue, vascular impairment causes acute ischemia or infarction, which is a dynamic process that develops over time. Neuronal damage is caused by hypoxia at the cellular level.

## NEURONAL INJURY

Ischemia causes the endothelium, leucocytes, platelets, and other neuronal cells to generate harmful vasoactive enzymes that encourage the formation of micro thrombus, which obstructs the cerebral microcirculation. [32]

At the molecular level, glutamate and aspartate over activity are the primary causes of hypoxic ischemic neuronal damage and is called excito-toxicity. It starts when cellular energy reserves are depleted. Normally, glutamate is stored and cleared in synaptic terminals and

extracellular space respectively. This causes the opening of calcium channels associated with N-Methyl-D-Aspartate (NMDA) and Alpha- Amino-3-Hydroxy-5-methyl-4-Isoxanole propionate (AMPA) receptors. The influx of calcium, sodium, and chloride ions and the efflux of potassium ions are caused by persistent membrane depolarization. [33] Proteases, lipases, and endonucleases are among the destructive enzymes that are activated when intracellular calcium levels rise. These result in the production of cytokines and other mediators, which degrade the integrity of the cell. [34]

Several mechanisms mediate the inflammatory response to tissue injury. Tumour Necrosis Factor is the main component of inflammatory mediators. Within 30 minutes of the ischemic areas being cleared, leucocyte recruitment takes place. Vasoactive molecules such as oxygen free radicals, metabolites of arachidonic acid (cytokines) and nitric acid are activated by leucocytes. These result in increased vasodilatation and increased leucocyte adhesion, increased permeability, and increased platelet aggregation to the endothelium wall.

Nitric acid and other smooth muscle relaxants, as well as vasoactive substances including endothelin, peptides, and eicosanoids, function as vasodilators. Endothelium adhesion molecules are activated to cause leucocyte adhesion to the endothelial wall, which is a crucial stage in the beginning of the inflammatory process.

## ISCHEMIC PENUMBRA

An oligemic area called the ISCHEMIC PENUMBRA appears within an hour of the hypoxic ischemic insult and it forms around the infarct's inner core, where the auto-regulation does not

work but the cellular integrity is maintained. An increase in ischemia in this area causes worsening of neurological deficits which can be prevented, partially or completely, by re-perfusion within a critical time period (2-4hrs) called THE WINDOW OF OPPORTUNITY. (35)

The production of Spontaneous Waves of Depolarization (SWD) is directly tied to the patho-physiology of the ischemic penumbra. These have a multifocal origin, with some coming from the infarct's centre and others from its ischemic penumbra. They are linked to a sustained rise in extracellular potassium and synaptic glutamate. Prior to irreversible neuronal death, the neuron experiences hypoxic/rapid depolarisations.

## NEURONAL DEATH

Neuronal death occurs by 2 processes

- Coagulation necrosis
- Apoptosis
- ✓ Apoptotic mechanisms start within an hour of ischemic injury whereas necrosis sets in about 6 hours after arterial occlusion.

## CEREBRAL EDEMA

Cerebral oedema is a major cause of death and disability, thus it is very concerning. The cause of this cerebral oedema is neurogenic inflammation, which is mediated by neuropeptides like substance P.

Klatzo divided oedema into the following categories:

- ✓ CYTOTOXIC OEDEMA - which develops within hours and is reversible. When ATP-dependent ion transport fails and oxygen-derived free radicals are released, the cellular components of the brain, such as neurons, glia, and endothelial cells, enlarge.
- ✓ VASOGENIC EDEMA - develops over the course of several hours and is irreversible. It results in enhanced permeability to serum proteins. This causes an increase in the volume of extracellular fluid and intracranial pressure (ICP). As a result, there occurs a compartment shift within the brain, compressing neuronal structures and cerebral arteries. A prolonged increase in intracranial pressure causes persistent ischemia and permanent brain cell damage. Severe cases might result in brain herniation and even death. [36] [37]

## DIAGNOSIS

Diagnosis of stroke is through clinical history and physical examination with appropriate investigations. The physical examination can help identify the affected artery region and advocate for appropriate tests like MRI for posterior circulation stroke. Posterior circulation stroke is better understood in MRI than CT.

Baseline investigations include

- Complete blood count
- Blood glucose level
- Renal function test

- Serum electrolytes
- Fasting lipid profile
- ECG and echocardiography
- Carotid Doppler

Specific investigations include

- NON – ENHANCED CT BRAIN –

When a stroke is suspected, CT is the first test of choice. Differentiating between ischemic and haemorrhagic stroke is the main goal. The primary use of non-enhanced CT is the detection of bleeding or other potential stroke mimics (such as tumours, extradural hematomas, abscesses, and arteriovenous malformation) that may be the origin of the neurologic deficit. CT is particularly sensitive for the visualization of haemorrhagic lesions.

The subset of ischemic stroke can be divided based on timing from the onset of stroke symptoms into

- ✓ hyper-acute - first 6 hours
- ✓ acute - 6 to 48 hours
- ✓ subacute - 48hr to weeks
- ✓ chronic stroke - weeks to months

Hyper acute Stage - during the first 3 to 6 hours produce loss of grey-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 hypo density; and hyper density in a circle of Willis vessel. [38]

The earliest visible CT sign is a hyper dense section of a vessel that represents immediate visualisation of the intravascular thrombus/embolus and is known as the HYPER DENSE MIDDLE CEREBRAL ARTERY SIGN because it is most frequently seen in the middle cerebral artery. [39]

In the subacute period, a phenomenon known as the "CT FOGGING EFFECT" occurs where hypodensed infarcted areas vanish and become isodense. This is possibly because the oedema in the infarcted area has resolved. This often happens 2 to 6 weeks following the onset of stroke.

Additionally, in 15 to 20% of the patients during this time, there is a danger of haemorrhagic transformation. In majority of the cases, this happens 4 to 6 days after the onset of stroke. Once it has occurred, the hyper density CT image could last for eight to ten weeks.

After weeks to months, the damaged necrotic tissue becomes reabsorbed. This leads to the development of encephalomalacia and gliosis of surrounding brain tissue. [40] [41]

- MAGNETIC RESONANCE IMAGING -

Diffusion weighted MRI imaging is more sensitive for the diagnosis of hyper acute ischemia and magnetic resonance imaging (MRI) has become an indispensable tool for the treatment of stroke patients. MRI may be especially helpful for TIA patients because it is more likely to detect new infarction, which is a reliable indicator of subsequent stroke.

Both intracranial stenosis of major vessels and extra-cranial internal carotid plaque are detectable with great sensitivity using magnetic resonance angiography. In almost every stroke case, MRI demonstrates greater information over CT.

## TREATMENT

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

### 1) GENERAL SUPPORTIVE CARE

#### ● CIRCULATION, AIRWAY AND BREATHING

The main cause of a stroke is an inadequate oxygenation and energy delivery to the target tissue. To prevent further cellular damage, systemic hypoxemia and hypotension should be prevented and, if present, should be addressed.

#### ● TEMPERATURE

Experimental and localised hypoxic brain damage models have demonstrated that hypothermia is neuroprotective. Hypothermia may slow down the use of stored energy, lower intracellular acidosis, slow calcium entry into ischemic cells, suppress oxygen free radical formation, change apoptotic signals, reduce cytokine and inflammatory production, and lessen the effect of excitatory amino acids. Since mild to moderate hypothermia is linked to better neurological outcomes for cardiac arrest patients, the AHA first suggested hypothermia as a neuroprotective measure for comatose cardiac arrest patients. [(42)] Patients who are hypoxic should receive supplemental oxygen to maintain oxygen saturation >94 percent.

- The Paracetamol (Acetaminophen) In Stroke (PAIS) trial evaluated 1400 adults no later than 12 hours after symptom onset of acute ischemic stroke or intracerebral hemorrhage. Included patients had a body temperature of 36 to 39ºC. Compared with placebo, paracetamol (acetaminophen) 1 g six times daily for three days did not improve outcome. However, a post-hoc subgroup analysis of 661 patients with a baseline body temperature of 37 to 39ºC suggested benefit for paracetamol. [43]

- In a systematic review and meta-analysis of five small randomized controlled trials with a total of 293 patients, there was no benefit for pharmacologic temperature reduction for acute stroke. All the trials enrolled patients within 24 hours of stroke onset, and the duration of treatment ranged from 24 hours to five days. With addition of results from the PAIS trial, the updated meta-analysis found no difference between active treatment and control for a favourable outcome (odds ratio [OR] 1.1, 95% CI 0.9-1.3)

In the first few hours following stroke onset, about one-third of patients admitted with stroke will be hyperthermic (temperature >37.6°C). Hyperthermia is linked to a poor neurological function in the context of acute ischemic stroke, probably as a result of higher metabolic demands, augmented neurotransmitter release, and increased free radical generation. Antipyretic drugs should be given to hyperthermic stroke patients in order to lower their temperature. Sources of hyperthermia (temperature >38°C) should be located and treated. [42]

- **BLOOD PRESSURE MANAGEMENT:**

During an acute ischemic stroke, elevated blood pressure is most commonly present. Extreme arterial hypertension certainly has negative effects because it can cause heart problems, renal insufficiency, and encephalopathy. It is fair to aim to reduce blood pressure by 15% within the first 24 hours following the beginning of stroke in individuals with noticeably increased blood pressure who do not get fibrinolysis. Although the blood pressure that would require such therapy is unknown, it is generally agreed that drugs should not be started until either the systolic or the diastolic blood pressure is >220 mmHg or >120 mm Hg respectively.

- **BLOOD GLUCOSE:**

Cases with hypoglycemia during acute ischemic stroke are probably caused by antidiabetic drugs. Permanent brain damage may be the outcome of severe or protracted hypoglycemia. Patients with an acute ischemic stroke should be addressed if they have hypoglycemia (blood glucose < 60 mg/dl). 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 because persistent in-hospital hyperglycemia during the first 24 hours after stroke is associated with worse outcomes than normo-glycemia. [42]

## 2) SPECIFIC MANAGEMENT

Reperfusion is the best treatment for an acute ischemic stroke. It must be accomplished before the ischemic area of the brain is entirely infarcted for it to be effective.

- **THROMBOLYSIS:**

In 1996, the US FDA gave its approval for intravenous rtPA usage. A wide range of patients had better outcomes when treated within three hours of the onset of symptoms and a slightly more selective range of patients when treated between three and four hours after the onset of stroke. Most significantly, earlier therapy increases the likelihood of a successful outcome. Major stroke patients within 3 hours of onset (NIHSS score >22) have a very dismal prognosis, however some benefit from intravenous rtPA therapy is still there. Increased rates of intracranial hemorrhage, which can be fatal, are linked to intravenous rtPA therapy. Before thrombolysis, it's crucial to choose the right patient. [44]

- **THROMBOLYTIC THERAPY:**

**INDICATIONS**:

i) Diagnosis of ischemic stroke causing measurable neurological deficit

ii) Onset of symptoms < 4.5 hours before beginning treatment

iii) Age ≥ 18years

iv) Consent by patient or relatives

**CONTRAINDICATIONS:**

i) Cerebral imaging showing intracerebral hemorrhage

ii) Cerebral imaging demonstrating large infarction, >1/3 territory of a cerebral hemisphere (CT hypo density or diffusion restriction on MRI)

iii) Head trauma within 3 months prior to stroke

iv) History of intracranial hemorrhage

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

vi) Active bleeding or arterial puncture at non-compressible site.

vii) 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

viii) Blood glucose

**RELATIVE CONTRAINDICATIONS:**

i) Minor or resolving stroke

ii) Seizure at onset

iii) Major surgery or trauma within 14 days

iv) Gastrointestinal or urinary bleeding within 21 days

v) Myocardial infarction within 3 months

**DOSE ADMINISTRATION AND PRECAUTIONS:**

- ✓ Infusion of rtPA @ 0.9 mg/kg (maximum dose 90 mg) over 60 minutes, with 10% of the dose given as a bolus over 1 minute.
- ✓ Measurement of blood pressure and 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.
- ✓ 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, discontinuing the infusion (if IV rtPA is being administered) and doing an emergency CT scan. [45]

- **ANTITHROMBOTIC MANAGEMENT:**

- Anti-platelet agents form the mainstay for secondary prevention of non-cardio embolic stroke.

- Aspirin is the only anti-platelet agent that has proven effectiveness against Acute Ischemic stroke.

- Clopidogrel, a P2Y12-receptor antagonist, inhibits platelet aggregation synergistically with aspirin.

- According to the Clopidogrel in High Risk Patients with Acute Non-Disabling Cerebrovascular Events (CHANCE) study, taking aspirin with Clopidogrel lowers the risk of another stroke without raising the risk of bleeding events in patients who have already experienced a minor stroke or transient ischaemic attack (TIA). [46]

- **ANTI EDEMA MEASURES AFTER ISCHEMIC STROKE:**

A delayed decrease in the level of consciousness is frequently experienced due to the edema of the infarcted tissue after an acute cerebral infarction. Despite the fact that cytotoxic edema often reaches its peak 3 to 4 days after injury, early reperfusion of a sizable amount of necrotic tissue can cause the edema to intensify within the first 24 hours, a condition known as malignant edema.

Careful supervision is necessary in individuals with severe stroke or posterior fossa infarctions for early intervention to treat the potentially fatal edema.

Increased intracranial pressure is reduced via hyperventilation, hypertonic saline, osmotic diuretics, intraventricular drainage of cerebrospinal fluid, and decompressive surgery.

- **GENERAL SUPPORTIVE TREATMENT:**

- Adequate nutrition is essential because dehydration and malnutrition may slow the recovery.

- Dehydration has been a potential cause of DVT after stroke.

- Swallowing difficulties can lead to a potential high risk of pneumonia. The patient may be given a tight "nothing by mouth" order until a swallowing capacity evaluation is finished. The majority of patients begin their treatment with intravenous fluids. It is possible to place a nasogastric tube to administer drugs and offer feedings.

- The second leading cause of death after a stroke is pneumonia, which is more common in seriously ill, immobile patients and those who are unable to cough. Pneumonia brought on by a stroke doubles hospital expenses, mortality, and length of stay.

Atelectasis and immobility both contribute to the development of pneumonia. Pneumonia can be avoided with early mobilization and proper pulmonary care. The development of atelectasis may be slowed down by exercise and encouragement to take deep breaths. Antibiotic usage as a preventative measure is not advocated for stroke patients at risk for pneumonia. [42]

- Ancillary care also includes bowel control to prevent constipation and faecal impaction or diarrhoea. In patients with moderate stroke severity, constipation was linked to poor outcomes at 12 weeks and occurs in 30–60% of patients 4 weeks after stroke.

- **SURGICAL MANAGEMENT OF ISCHEMIC STROKE:**

✓ DECOMPRESSIVE SURGERY:

Decompressive surgeries are mostly indicated for brainstem posterior circulation stroke where delayed swelling and significant infarction of the cerebellum frequently occurs. Edema can cause brain stem compression and can advance very quickly to a loss of brain stem function, even if the early symptoms may be restricted to reduced cerebellar function. The

clinical outcome has a respectable quality of life and frequently results in lifesaving emergency posterior fossa decompression with partial excision of the infarcted tissue. [42]

## PROGNOSIS:

The patient's family, other healthcare professionals, insurance companies, and patients frequently urge clinicians to forecast outcomes following a stroke. Age, stroke severity, stroke mechanism, infarct site, concomitant diseases, clinical findings, and associated sequelae are only a few of the many variables that affect a stroke patient's prognosis. Additionally, thrombolysis, mechanical thrombectomy, care in a stroke unit, and rehabilitation can all have a significant impact on how an ischemic stroke turns out. For a clinician to give an accurate prognosis for a specific patient, to offer a logical approach to patient management, and to assist the patient and family in understanding the course of the disease, they must be aware of the key variables that determine prognosis.

The estimated 30-day case fatality rate following a first ischemic stroke varies widely across nations, although it is expected to be between 16 and 23% globally. According to a cohort study of persons 18 to 49 years' old who had their first stroke during the previous 30 days, the mortality risk remained higher than it was in the general population up to 15 years after the stroke. The cumulative mortality rate was 32 percent in a 10-year follow-up study of 322 patients who had mild ischemic strokes, which is nearly double the rate for the general population.

## PREDICTORS OF MORTALITY:

Stroke severity and patient age are the best indicators of outcome during the acute phase of a stroke. The degree of neurologic impairment (e.g., altered mentation, language, behaviour, visual field loss, motor deficit) and the size and location of the infarction on neuroimaging with magnetic resonance imaging (MRI) or computed tomography can be used to clinically assess the severity of a stroke (CT).

## PREMISE SCORE:

Although many studies on stroke mortality predictors have already been published, they have examined heterogeneous stroke cohorts (ischemic and haemorrhagic strokes), were treated in different systems of care (general ward versus stroke unit), and took into account various time points of case fatality (in-hospital to 1-year mortality) because they were conducted when specialised stroke unit management and specific stroke treatments were not yet widely available. These studies mostly concentrated on short- and long-term mortality. A limited number of research have concentrated on in-hospital death rates pertaining to a median hospital stay of 1 to 2 weeks, and there is little information available regarding the frequency and risk factors for early mortality in the first week.

The Nationwide Austrian Stroke Unit Registry found risk variables for acute ischemic stroke-related early stroke unit death. The proposed PREMISE (Predicting Early Mortality of Ischemic Stroke) risk model uses only variables that are easily accessible shortly after the onset of ischemic stroke when a patient is admitted to the stroke unit, and it is a straightforward,

quickly calculable score that explains >85% of early stroke deaths. This score may assist stroke clinicians in performing early bedside estimation of mortality risk in ischemic stroke patients due to its good diagnostic accuracy and practicability. Early death was statistically impossible with score points 0 to 3 (1%), but it grew to 35% in the category with the highest score, 10 points.

Age, pre-stroke functional status (MRS score >0), stroke severity (NIHSS), diabetes mellitus, prior heart disease, posterior circulation stroke syndrome (compared to anterior circulation stroke syndromes), and non-lacunar stroke cause were found to be independent predictors for stroke unit mortality. Hyperlipidemia was linked to a decreased risk of post-stroke death, in line with earlier investigations (lipid paradox).

It should be noted that following multivariate analysis, atrial fibrillation did not continue to be an independent predictor of stroke death – although it is obviously related with mortality in univariate analysis. Patients with posterior circulation stroke syndromes had a higher short-term mortality rate than patients with anterior circulation stroke syndromes, which is most likely due to brain stem damage or an infra-tentorial mass effect, both of which frequently result in early, severe, and potentially fatal complications. It is well recognised that the NIHSS does not accurately reflect the range of neurological abnormalities connected to the posterior circulation, which may further lead to an independent influence of posterior circulation stroke syndrome on mortality. [47]

**Table 3. Risk Prediction Score for Early Ischemic Stroke Mortality (PREMISE)**

| Risk Factors for Stroke Unit Mortality | Points |
|---|---|
| Age | |
| 60–69, y | +1 |
| ≥70, y | +2 |
| Preexisting disability | |
| Modified Rankin Scale scores 1–5 | +1 |
| Stroke severity | |
| NIHSS 5–11 | +2 |
| NIHSS 12–23 | +4 |
| NIHSS ≥24 | +5 |
| Vascular diseases | |
| Diabetes mellitus | +1 |
| Heart disease* | +1 |
| Clinical stroke syndrome | |
| Posterior circulation stroke syndrome | +1 |
| Stroke cause | |
| Nonlacunar | +1 |
| Maximal score points | =12 |

NIHSS indicates National Institutes of Health Stroke Scale.

*Defined as coronary artery disease, heart failure, cardiomyopathy, or valve disease.

**NEUROLOGIC SEVERITY** — The severity of stroke on neurologic examination is probably the most important factor affecting short- and long-term outcome. Generally, large strokes with severe initial clinical deficits have poor outcomes compared with smaller strokes.

- ✓ **NIHSS SCORE:**

The National Institutes of Health Stroke Scale (NIHSS), a 15-item scale, is used to quantitatively assess neurologic damage in numerous research studies and more frequently used in clinical practise.

When patients first report with symptoms of a stroke, the NIHSS score is most frequently employed. The NIHSS has been shown in numerous studies to be a reliable indicator of stroke outcome. In one study, nearly 1200 patients enrolled in a clinical trial's NIHSS ratings collected within 24 hours after the beginning of acute ischemic stroke symptoms were analysed.

- Each additional point on the NIHSS decreased the odds of an excellent outcome at three months by 17 percent.

- An NIHSS score below six indicated a successful recovery (ability to live independently, regardless of capacity to return to work or education), while a score above sixteen indicated a significant likelihood of severe disability or death.

| | |
|---|---|
| 1a—Level of consciousness | 0 = Alert; keenly responsive<br>1 = Not alert, but arousable by minor stimulation<br>2 = Not alert; requires repeated stimulation<br>3 = Unresponsive or responds only with reflex |
| 1b—Level of consciousness questions:<br>What is your age?<br>What is the month? | 0 = Answers two questions correctly<br>1 = Answers one question correctly<br>2 = Answers neither questions correctly |
| 1c—Level of consciousness commands:<br>Open and close your eyes<br>Grip and release your hand | 0 = Performs both tasks correctly<br>1 = Performs one task correctly<br>2 = Performs neither task correctly |
| 2—Best gaze | 0 = Normal<br>1 = Partial gaze palsy<br>2 = Forced deviation |
| 3—Visual | 0 = No visual lost<br>1 = Partial hemianopia<br>2 = Complete hemianopia<br>3 = Bilateral hemianopia |
| 4—Facial palsy | 0 = Normal symmetric movements<br>1 = Minor paralysis<br>2 = Partial paralysis<br>3 = Complete paralysis of one or both sides |
| 5—Motor arm<br>Left arm<br>Right arm | 0 = No drift<br>1 = Drift<br>2 = Some effort against gravity<br>3 = No effort against gravity<br>4 = No movement |
| 6—Motor leg<br>Left leg<br>Right leg | 0 = No drift<br>1 = Drift<br>2 = Some effort against gravity<br>3 = No effort against gravity<br>4 = No movement |
| 7—Limb ataxia | 0 = Absent<br>1 = Present in one limb<br>2 = Present in two limbs |
| 8—Sensory | 0 = Normal; no sensory loss<br>1 = Mild-to-moderate sensory loss<br>2 = Severe-to-total sensory loss |
| 9—Best language | 0 = No aphasia; normal<br>1 = Mild-to-moderate aphasia<br>2 = Severe aphasia<br>3 = Mute; global aphasia |
| 10—Dysarthria | 0 = Normal<br>1 = Mild-to-moderate dysarthria<br>2 = Severe dysarthria |
| 11—Extinction and inattention | 0 = No abnormality<br>1 = Visual, tactile, auditory, spatial, or personal inattention<br>2 = Profound hemi-inattention or extinction |
| Score = 0–42 | |

Figure 1. The National Institutes of Health Stroke Scale (NIHSS).
Note: NIHSS is a systematic assessment tool that provides a quantitative measure of stroke-related neurological impairments.

The association between NIHSS score and ultimate prognosis varies depending on how long after the stroke started, in part because early deficits caused by strokes are frequently unstable and because many people tend to have gradually recovery. As a result, the NIHSS score linked to a specific impairment outcome decreases with time. An NIHSS score of >22 was found to be the strongest predictor of a bad prognosis after 24 hours, and an NIHSS score of >16 was found to be the best predictor at 7 to 10 days. [48] Additionally, over time, there is a growing link between NIHSS results and actual disability outcomes.

## SCANDINAVIAN STROKE SCALE:

The Scandinavian Stroke Scale was created with non-neurologists in mind. It performs similarly to the NIHSS in terms of predicting stroke-related dependency or death.

It has been demonstrated that the severity of the ASPECTS scores in acute stroke correlates with the level of neurological impairment as determined by the Scandinavian Stroke Scale. The majority of the scale's components yielded positive results, with high rates of agreement across raters.

# SCANDINAVIAN STROKE SCALE

| | **Consciousness** *(circle score)* |
|---|---|
| 6 | Fully conscious |
| 4 | Somnolent but can be awakened fully |
| 2 | Reacts to verbal command |
| 0 | Comatose |
| | |
| | **Eye Movement** *(circle score)* |
| 4 | No gaze palsy |
| 2 | Gaze palsy present (difficulty in moving eyes) |
| 0 | Conjugate eye deviation (eyes deviate to same side) |
| | |
| | **Arm, Motor Power** (affected side) *(circle score)* |
| 6 | Raises arm with normal strength |
| 5 | Raises arm with reduced strength |
| 4 | Raises arm with flexion in elbow |
| 2 | Can move but not against gravity |
| 0 | Paralysis |
| | |
| | **Hand, Motor power** (affected side) *(circle score)* |
| 6 | Normal strength |
| 4 | Reduced strength in full range |
| 2 | Some movement, fingertips do not reach palm |
| 0 | Paralysis |
| | |
| | **Leg, Motor Power** (affected side) *(circle score)* |
| 6 | Normal strength |
| 5 | Raises straight leg with reduced strength |
| 4 | Raises leg with flexion in knee |
| 2 | Can move but not against gravity |
| 0 | Paralysis |

| | **Orientation** *(circle score)* |
|---|---|
| 6 | Correct time, place, person |
| 4 | Two of these |
| 2 | One of these |
| 0 | Completely disorientated |
| | |
| | **Speech** *(circle score)* |
| 10 | No aphasia |
| 6 | Incoherent speech |
| 3 | More than yes/no but no longer sentences |
| 0 | Only yes/no or less |
| | |
| | **Facial Palsy** *(circle score)* |
| 2 | None/dubious |
| 0 | Present |
| | |
| | **Gait** *(circle score)* |
| 12 | Walks 5m without aids |
| 9 | Walks with aids |
| 6 | Walks with the help of another person |
| 3 | Sits without support |
| 0 | Bedridden/wheelchair |
| | |
| | ☐ **TOTAL SCORE** Out of 58 |
| | **High Risk SSS < 10** |
| | **Intermediate Risk SSS 10 – 40** |
| | **Low Risk SSS > 40** |

Because they are objective items and the scale has a strong correlation during the acute stroke period, when such elements are more obvious and traceable in neurological evaluation, consciousness and orientation showed good concordance rates. The palsy severity items had a fair level of concordance, demonstrating the instrument's ability to forecast affected people's motor recovery.

Due to the speech item's contribution of 5.8% to the overall scale score, which allows for potential underestimating of lesions in the right hemisphere, the SSS gives preference to designing its questions to evaluate the dominant hemisphere, which is also indicated in the NIHSS. The SSS and NIHSS have different scoring systems, so it is important to carefully examine the correlations between the scales' scores. [49]

- ✓ **APACHE II SCORE**

Acute physiology score (APS), age, and CPS are the three components that make up the APACHE II score system. It makes predictions about the severity of the sickness based on the APACHE II score, where the total score runs from 0 to 71 and the severity and value are positively associated. The APACHE II score also takes into account a patient's prior physical problems, test results, and signs from across their entire body. It has grown to be the most widely used critical disease prediction evaluation method. [50]

Table 5: The APACHE II Severity of Disease Classification System

| Physiologic Variable | | +4 | +3 | +2 | +1 | 0 | +1 | +2 | +3 | +4 |
|---|---|---|---|---|---|---|---|---|---|---|
| **Temperature - rectal** (°C) | | ≥41 | 39-40.9 | | 38.5-38.9 | 36-38.4 | 34-35.9 | 32-33.9 | 30-31.9 | ≤29.9 |
| **Mean Arterial Pressure** (mm Hg) | | ≥160 | 130-159 | 110-129 | | 70-109 | | 50-69 | | ≤49 |
| **Heart Rate** | | ≥180 | 140-179 | 110-139 | | 70-109 | | 55-69 | 40-54 | ≤39 |
| **Respiratory Rate** (nonventilated or ventilated) | | ≥50 | 35-49 | | 25-34 | 12-24 | 10-11 | 6-9 | | ≤5 |
| **Oxygenation** (mmHg) a. $FiO_2 > 0,5$ use A-a$DO_2$ b. $FiO_2 < 0,5$ use $PaO_2$ | a | ≥500 | 350-499 | 200-349 | | <200 | | | | |
| | b | | | | | > 70 | 61-70 | | 55-60 | <55 |
| **Arterial pH** | | ≥7.7 | 7.6-7.69 | | 7.5-7.59 | 7.33-7.49 | | 7.25-7.32 | 7.15-7.24 | <7.15 |
| **Serum Sodium** (mmol/l) | | ≥180 | 160-179 | 155-159 | 150-154 | 130-149 | | 120-129 | 111-119 | ≤110 |
| **Serum Potassium** (mmol/l) | | ≥7 | 6-6.9 | | 5.5-5.9 | 3.5-5.4 | 3-3.4 | 2.5-2.9 | | <2.5 |
| **Serum Creatinine** (mg/dl, Double point score for acute renal failure) | | ≥3.5 | 2-3.4 | 1.5-1.9 | | 0.6-1.4 | | <0.6 | | |
| **Hematocrit** (%) | | ≥60 | | 50-59.9 | 46-49.9 | 30-45.9 | | 20-29.9 | | <20 |
| **White Blood Count** (in 1000/mm³) | | ≥40 | | 20-39.9 | 15-19.9 | 3-14.9 | | 1-2.9 | | <1 |
| **Glasgow-Coma-Scale** (GCS) | | Score = 15 minus actual GCS | | | | | | | | |
| **Serum $HCO_3$** (venous, mmol/l, use if no ABGs) | | ≥52 | 41-51.9 | | 32-40.9 | 22-31.9 | | 18-21.9 | 15-17.9 | <15 |
| **A = Total Acute Physiology Score APS** | | Sum of the 12 individual variable points | | | | | | | | |

**B = Age Points**

| | |
|---|---|
| ≤44 years | 0 points |
| 45-54 years | 2 points |
| 55-64 years | 3 points |
| 65-74 years | 5 points |
| ≥75 years | 6 points |

**C = Chronic Health Points**

If the patient has a history of severe organ system insufficiency or is immunocompromised assign points as follows:

a. For nonoperative or emergency postoperative patients – 5 points
b. For elective postoperative patients – 2 points

**APACHE II Score = Sum of A (APS points) + B (Age points) + C (Chronic Health points)**

(From: Knaus WA, Draper EA, Wagner DP, Zimmerman JE. APACHE II: a severity of disease classification system. Crit Care Med 1985;13(10):818-29)

The prognosis and death risk of a stroke patient can both be determined using the APACHE II score. According to the study, APACHE II and NIHSS scores closely correlate with how sick acute ischemic stroke patients are. Additionally, there was a clear difference in the APACHE II and NIHSS scores for patients in the death group and those in the survival group, and there was a significant correlation between severity and these scores. A possible explanation for why the APACHE II value for the death group was significantly higher than that for the survival group is that some APACHE II parameters, such as temperature and white blood cell count, are related to inflammation, and inflammation is involved in the pathological process of the Acute Ischemic Stroke patients.

This study intended to compare Acute Physiology and Chronic Health Evaluation (APACHE) II, National Institutes of Health Stroke Score (NIHSS), and SCANDINAVIAN STROKE SCALE in better prediction of stroke severity. The patients will be comprehensively assessed to compare the application values of these scoring systems in the prediction of early mortality of stroke, providing reference for the correct selection of stroke scale. Hence this study will be conducted to compare the efficiency of NIHSS, APACHE II AND SCANDINAVIAN STROKE SCALE in the prediction of early mortality (≤7 days' post stroke) of acute ischemic stroke patients - Premise admitted in a tertiary care hospital.

# MATERIALS AND METHODS

**STUDY TITLE:** A STUDY ON THE COMPARISON OF NIHSS, APACHE II, AND SCANDINAVIAN STROKE SCALE IN THE PREDICTION OF EARLY MORTALITY (≤7 DAYS POST STROKE) OF ACUTE ISCHEMIC STROKE PATIENTS – PREMISE - IN A TERTIARY CARE HOSPITAL

**STUDY DESIGN:** Prospective observational study

**STUDY PERIOD**: 6 months (May 2022 to October 2022)

**STUDY CENTRE:** Madras Medical College and Rajiv Gandhi Government General Hospital, Chennai

**STUDY POPULATION**:

- This Prospective Observational study was conducted on 200 patients admitted with Acute Ischemic Stroke in Government Rajiv Gandhi General Hospital & Madras Medical College during the study period from May 2022 to October 2022.
    - Sample size (n)= $Z^2pq/d^2$
    - z= Confidence interval at 95% (1.96)
    - Base line studies showed early mortality rate (≤7 days' post stroke) is 13% in patients admitted with acute ischemic stroke [51]

- p= 13
- q= 1-p (87)
- d=Absolute precision (5)
- SAMPLE SIZE (n) = 173.79 + additional 10% for non-response / attrition.

**Sample size - 200 cases.**

**INCLUSION CRITERIA:**

All patients above 45 years of age whose diagnosis are in accordance with the definition of Acute Ischemic Stroke as given by AHA/ASA, and confirmed by head computed tomography (CT) or magnetic resonance imaging (MRI) [47]

**EXCLUSION CRITERIA:**

- Patients with Young Stroke.
- Patients with Transient Ischemic Attacks.
- Patients with mild conditions who had only sensory symptoms or a muscle strength of ≥ grade IV.
- Patients who develop stroke during the course of hospital admission for other illness.
- Any patient who is already intubated/ventilated while admitting in our hospital.
- Patients who are not willing to participate in the study.

**DATA COLLECTION AND METHODOLOGY:**

- 200 patients with Acute Ischemic Stroke, who meet the inclusion and exclusion criteria, will be selected after obtaining informed consent.
- A detailed clinical history will be elicited and the details including vitals and neurological examination will be recorded.
- Baseline blood investigations like complete blood count, renal and liver function tests and ABG will be done.
- The APACHE II, NIHSS and SCANDINAVIAN STROKE SCALE scores will be assessed at admission within the first day (24 hours) of stroke.
- Outcome of the patient will be assessed as discharge/death from the hospital.

**ANALYSIS: STATISTICAL METHODS**

The data collected during the study was formulated into a master chart in Microsoft office excel and statistical analysis was done with help of computer using statistical software package SPSS V.17 for windows. Using this software, frequencies, range, mean, standard deviation and 'p' were calculated through ROC, Pearson correlation and chi square test.

P value of $< 0.05$ was taken as significant.

**ETHICAL CLEARANCE**: Approved

**CONSENT:** Individual written and informed

**CONFLICT OF INTEREST:** NIL

**FINANCIAL SUPPORT:** NIL

**PARTICIPANTS:** Patients of age >45 years, admitted as in-patients at Government Rajiv Gandhi General Hospital, Chennai who were diagnosed as Acute Ischemic Stroke.

# RESULTS

## TABLE 01. DISTRIBUTION OF AGE GROUP AMONG STUDY POPULATION

| | | FREQUENCY | PERCENTAGE % |
|---|---|---|---|
| AGE GROUP | 46-59 | 116 | 58.0% |
| | 60-69 | 35 | 17.5% |
| | >=70 | 49 | 24.5% |

In the present study - age group among patients, most of the patients were aged 46-59 years - 116 (58%), followed by 60-69 years - 35 (17.5%), and >70 years - 49 (24.5%) patients.

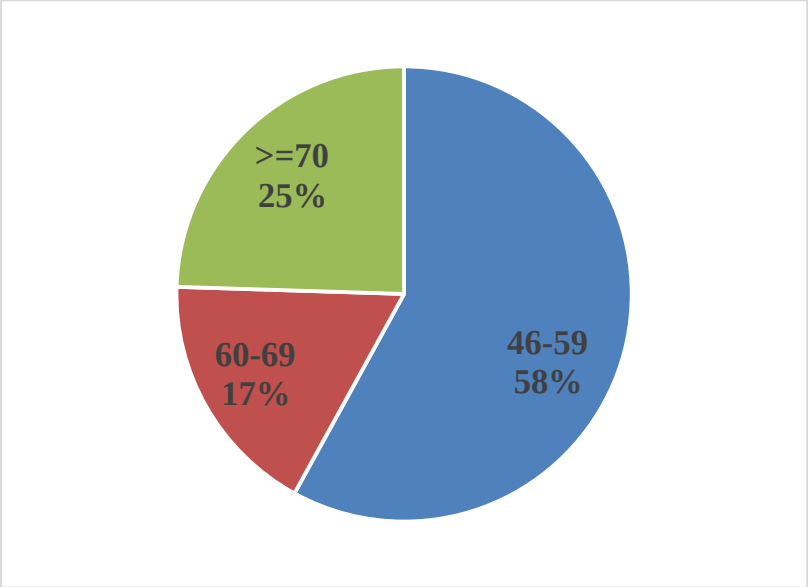



**FIGURE 01. DISTRIBUTION OF AGE GROUP AMONG STUDY POPULATION**

**TABLE 02. SEX-WISE DISTRIBUTION AMONG STUDY POPULATION**

| | | FREQUENCY | PERCENTAGE % |
|---|---|---|---|
| SEX | F | 66 | 33.0% |
| | M | 134 | 67.0% |

In the present study - gender among patients, most of patients were males - 134 (67%), followed by females - 66 (33%) patients.

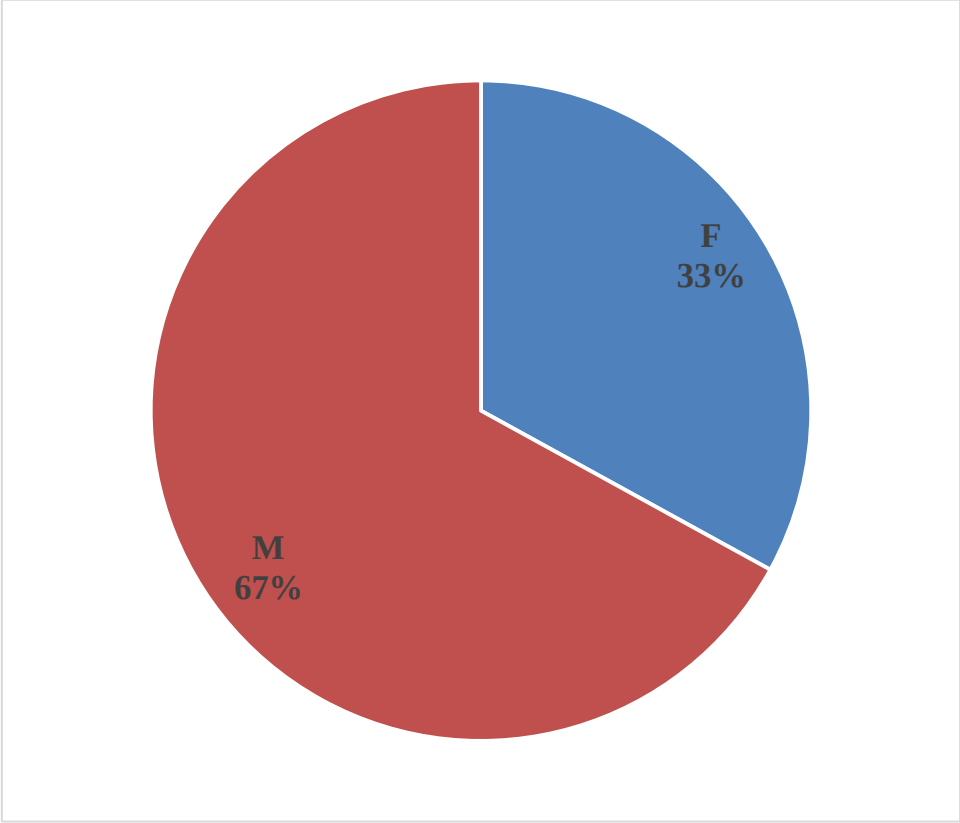



**FIGURE 02. SEX-WISE DISTRIBUTION AMONG STUDY POPULATION**

**TABLE 03. DISTRIBUTION OF TIME INTERVAL BETWEEN THE ONSET OF WEAKNESS AND HOSPITAL ADMISSION**

| | | FREQUENCY | PERCENTAGE % |
|---|---|---|---|
| **TIME INTERVAL BETWEEN ONSET OF WEAKNESS AND HOSPITAL ADMISSION** | **12-24 H** | 82 | 41.0% |
| | **6-12 H** | 110 | 55.0% |
| | **< 6 H** | 8 | 4.0% |

In the present study, comparing the time interval between the onset of weakness and hospital admission among study, most of the patients were admitted between 6-12h - 110 (55%) patients, followed by 82 (41%) patients between 12-24h, and 8 patients (4%) were admitted less than six hours.

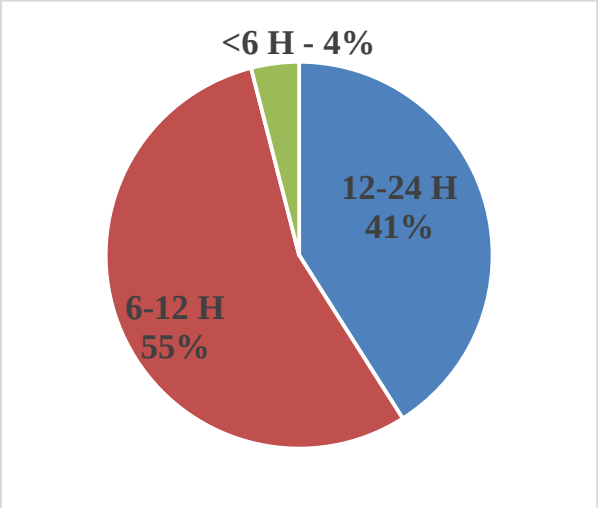



**FIGURE 03. DISTRIBUTION OF TIME INTERVAL BETWEEN THE ONSET OF WEAKNESS AND HOSPITAL ADMISSION**

**TABLE 04. DISTRIBUTION OF DIABETIC AMONG STUDY POPULATION**

| | | FREQUENCY | PERCENTAGE % |
|---|---|---|---|
| **DIABETIC** | **NO** | 88 | 44.0% |
| | **YES** | 112 | 56.0% |

In the present study - diabetes among patients, most of the patients had diabetes - 112 (56%) patients, and 88 (44%) patients had no diabetes.

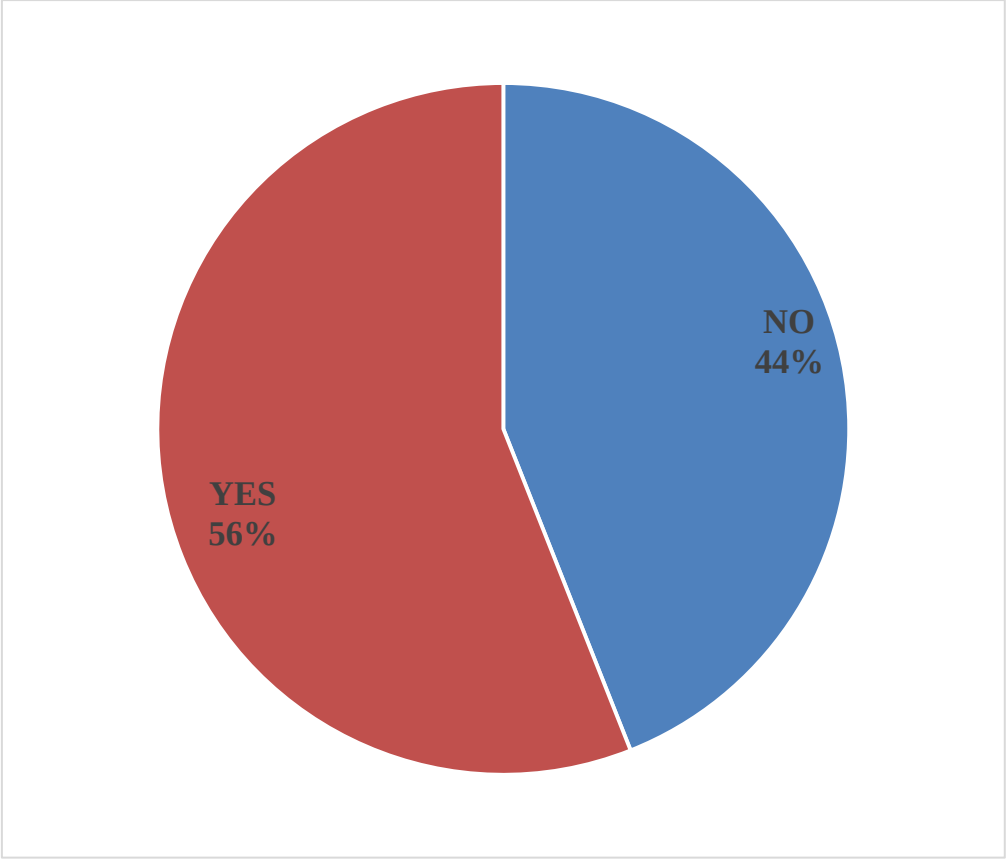



**FIGURE 04. DISTRIBUTION OF DIABETIC AMONG STUDY POPULATION**

**TABLE 05. DISTRIBUTION OF HEART DISEASE AMONG STUDY POPULATION**

| | | FREQUENCY | PERCENTAGE % |
|---|---|---|---|
| **HEART DISEASES** | **NO** | 175 | 87.5% |
| | **YES** | 25 | 12.5% |

In the present study - heart disease among patients, most of the patients had no heart disease - 175 (87.5%) patients and 25 (12.5%) patients had heart disease.

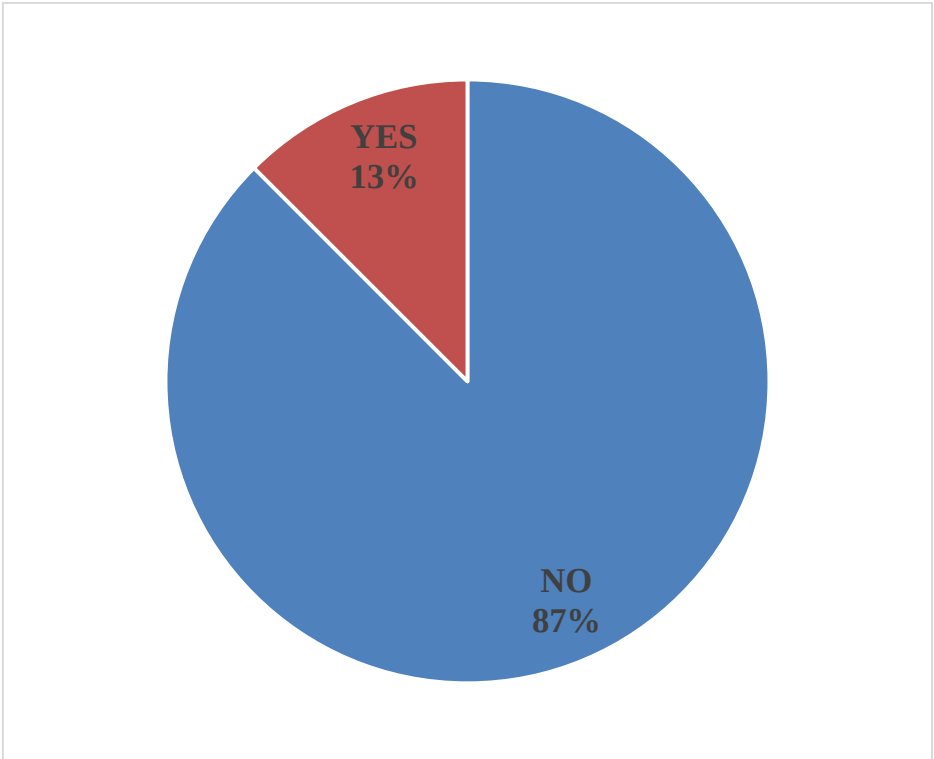



**FIGURE 05. DISTRIBUTION OF HEART DISEASE AMONG STUDY POPULATION**

**TABLE 06. DISTRIBUTION OF SYSTEMIC HYPERTENSION AMONG STUDY POPULATION**

| | | FREQUENCY | PERCENTAGE % |
|---|---|---|---|
| SYSTEMIC HYPERTENSION | NO | 26 | 13.0% |
| | YES | 174 | 87.0% |

In the present study - systemic hypertension among patients, most of patients had systemic hypertension - 174 (87%) and 26 (13%) patients had no systemic hypertension.

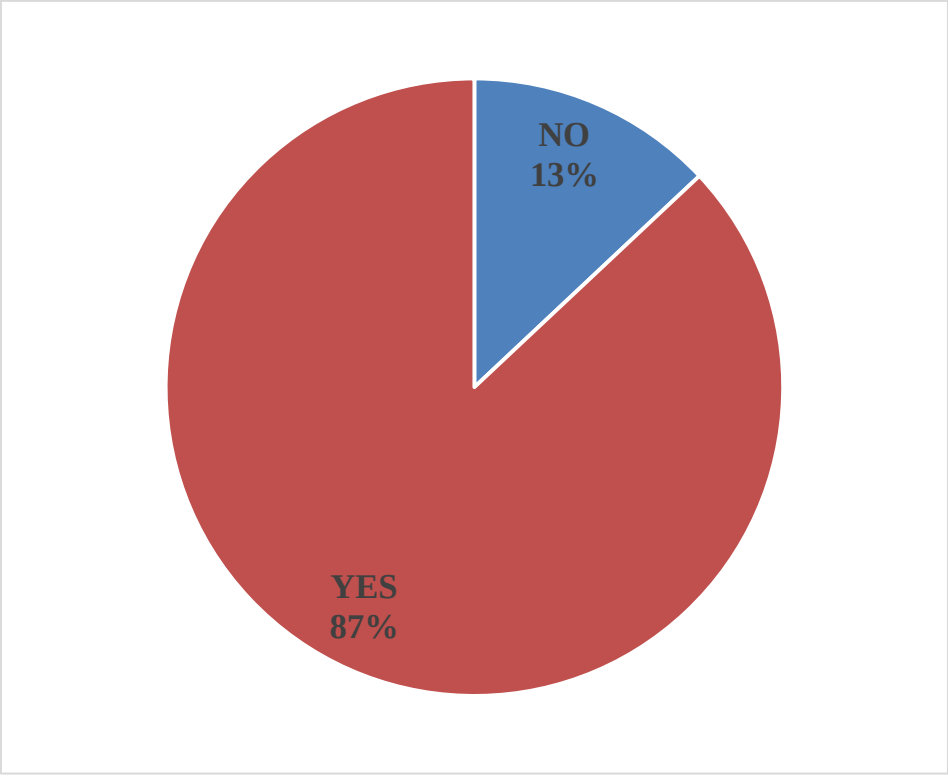



**FIGURE 06. DISTRIBUTION OF SYSTEMIC HYPERTENSION AMONG STUDY POPULATION**

**TABLE 07. DISTRIBUTION OF OTHER COMORBIDITIES AMONG STUDY POPULATION**

| | | FREQUENCY | PERCENTAGE % |
|---|---|---|---|
| **OTHER COMORBIDITY** | **AKI** | 8 | 4.0% |
| | **CA** | 1 | 0.5% |
| | **CKD** | 12 | 6.0% |
| | **H** | 3 | 1.5% |
| | **L** | 12 | 6 |
| | **N** | 164 | 82% |

*AKI- acute kidney injury; CA- carcinoma breast; CKD- chronic kidney disease;*

*H- hypothyroidism; L- dyslipidaemia; N- no other comorbidity*

In the present study, distribution of other comorbidities among study, dyslipidaemia 12 (6%) was a common comorbidity among patients. In addition, 12 (6%) had chronic kidney disease, 8 (4%) had acute kidney injury, 3 (1.5%) had hypothyroidism, one had cancer breast, and 164 had no other comorbidities.

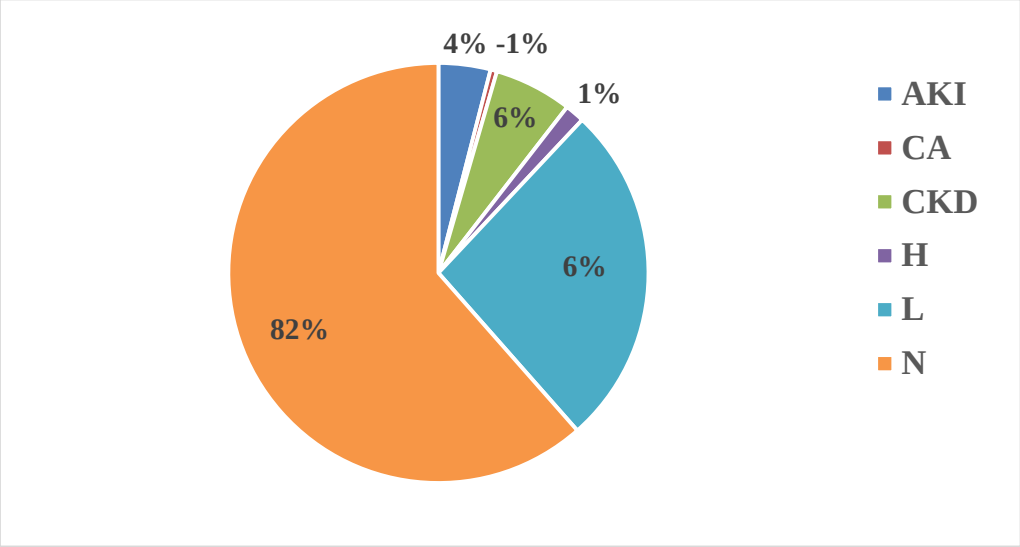



**FIGURE 07. DISTRIBUTION OF OTHER COMORBIDITIES AMONG STUDY POPULATION**

**TABLE 08. DISTRIBUTION OF ADDICTION AMONG STUDY POPULATION**

| | | FREQUENCY | PERCENTAGE % |
|---|---|---|---|
| ADDICTION | AL | 24 | 12.0% |
| | N | 147 | 73.5% |
| | S | 45 | 22.5% |

*AL- alcoholic; S- smoking; N- no addiction*

In the present study, addiction among study, 147 (73.5%) patients had no addiction, 45 (22.5%) patients were smokers, and 24 (12%) patients were alcohol consumers.

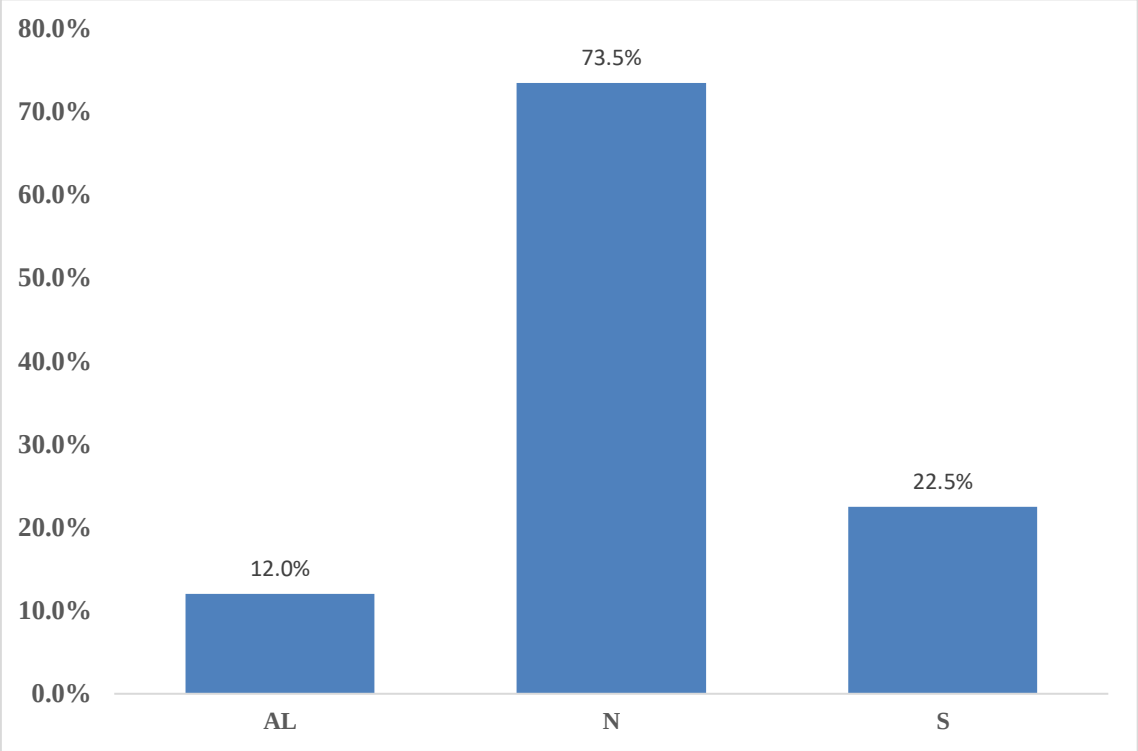



**FIGURE 08. DISTRIBUTION OF ADDICTION AMONG STUDY POPULATION**

**TABLE 09. DISTRIBUTION OF TYPES OF ACUTE ISCHEMIC STROKE AMONG STUDY POPULATION**

| | | FREQUENCY | PERCENTAGE % |
|---|---|---|---|
| ACUTE ISCHEMIC STROKE - PRIMARY/ RECURRENT | P | 185 | 92.5% |
| | R | 15 | 7.5% |

*P- primary; R- recurrent*

In the present study, acute ischemic stroke among study, 185 (92.5%) patients had a primary acute ischemic stroke, and 15 (7.5%) patients had a recurrent ischemic stroke.

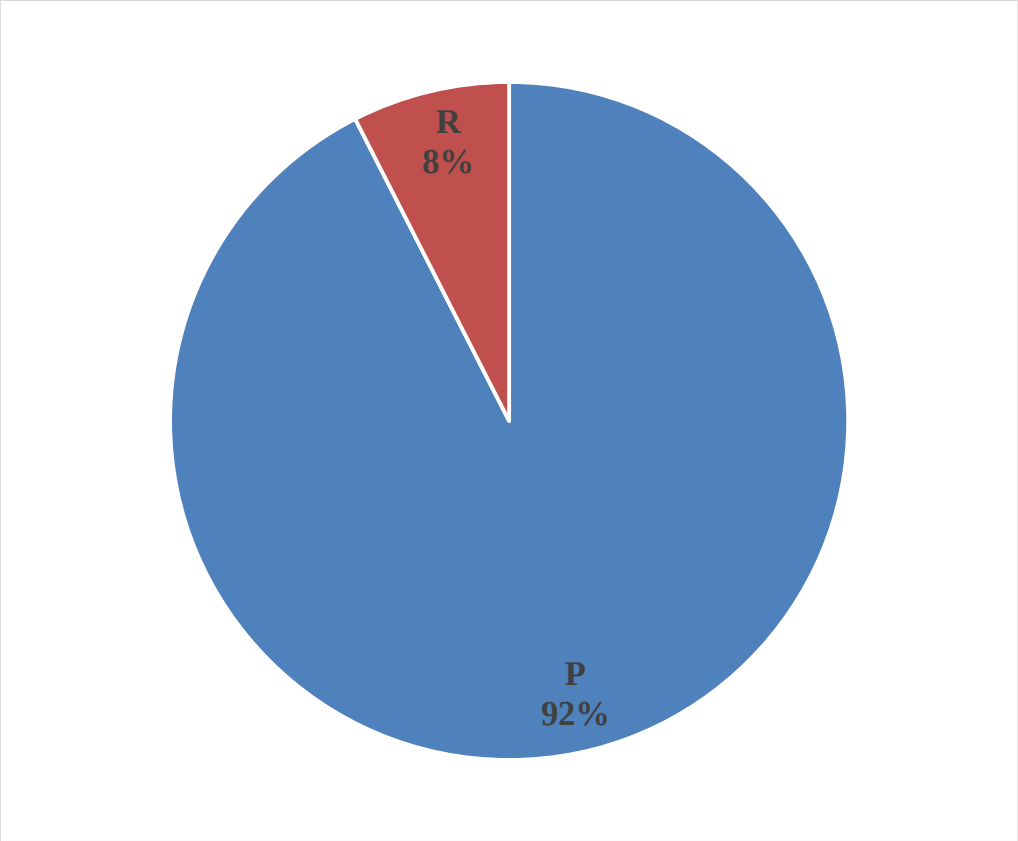



**FIGURE 09. DISTRIBUTION OF TYPES OF ACUTE ISCHEMIC STROKE AMONG STUDY POPULATION**

**TABLE 10. DISTRIBUTION OF POSTERIOR CIRCULATION STROKE AMONG STUDY POPULATION**

| | | FREQUENCY | PERCENTAGE % |
|---|---|---|---|
| POSTERIOR CIRCULATION STROKE | NO | 161 | 80.5% |
| | YES | 39 | 19.5% |

In the present study, posterior circulation stroke among study, 39 (19.5%) patients had a posterior circulation stroke, and 161 (80.5%) patients had no posterior circulation stroke.

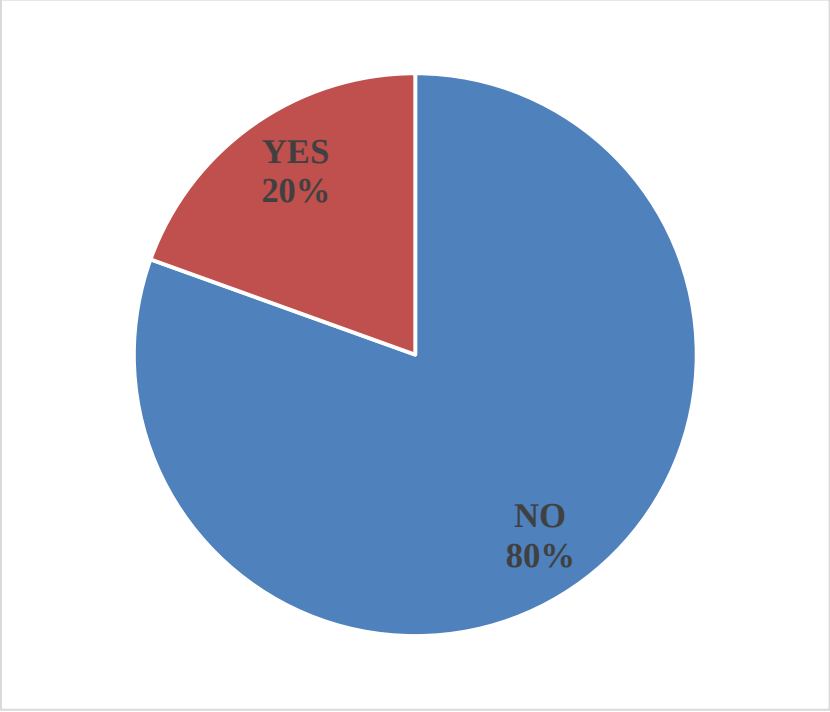



**FIGURE 10. DISTRIBUTION OF POSTERIOR CIRCULATION STROKE AMONG STUDY POPULATION**

**TABLE 11. DISTRIBUTION OF GLASGOW COMA SCALE AMONG STUDY POPULATION**

| | | FREQUENCY | PERCENTAGE % |
|---|---|---|---|
| GCS | VERY MILD/NIL (15) | 32 | 16.0% |
| | MILD (13-14) | 57 | 28.5% |
| | MODERATE (9-12) | 28 | 14.0% |
| | SEVERE (3-8) | 83 | 41.5% |

The present study classified the Glasgow Coma Scale among patients. Most patients had GCS – (3-8) severe 83 (41.5%)

Very mild (15 GCS): 32 (16%), mild (13-14 GCS): 57 (28.5%), and moderate (9-12 GCS): 28 (14%) patients.

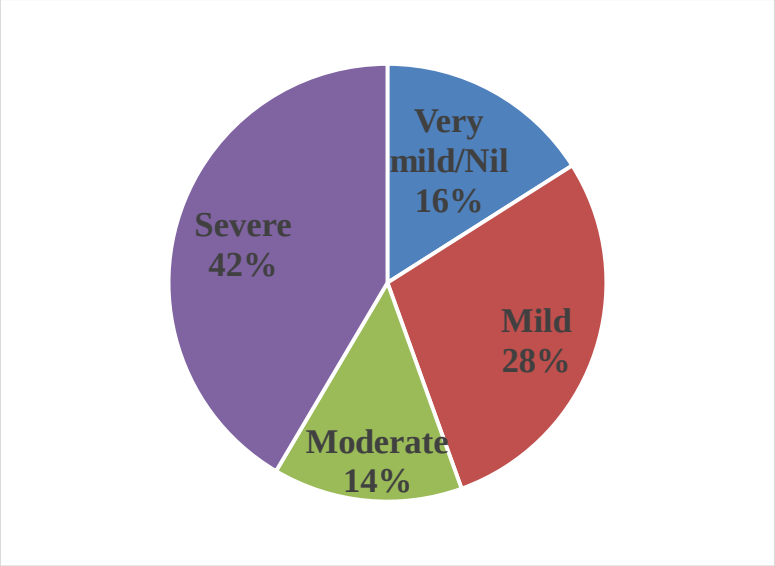



**FIGURE 11. DISTRIBUTION OF GLASGOW COMA SCALE AMONG STUDY POPULATION**

**TABLE 12. DISTRIBUTION OF NIHSS SCORE AMONG STUDY POPULATION**

| | | FREQUENCY | PERCENTAGE % |
|---|---|---|---|
| NIHSS SCORE | MILD (5-11) | 80 | 40.0% |
| | MODERATE (12-23) | 120 | 60.0% |
| | SEVERE (>23) | 0 | 0 |

The present study classified the NIHSS score among patients. Most patients- 120 (60%) had NIHSS – moderate (12-23); followed by 80 (40%) had NIHSS - mild. None had NIHSS – severe score.

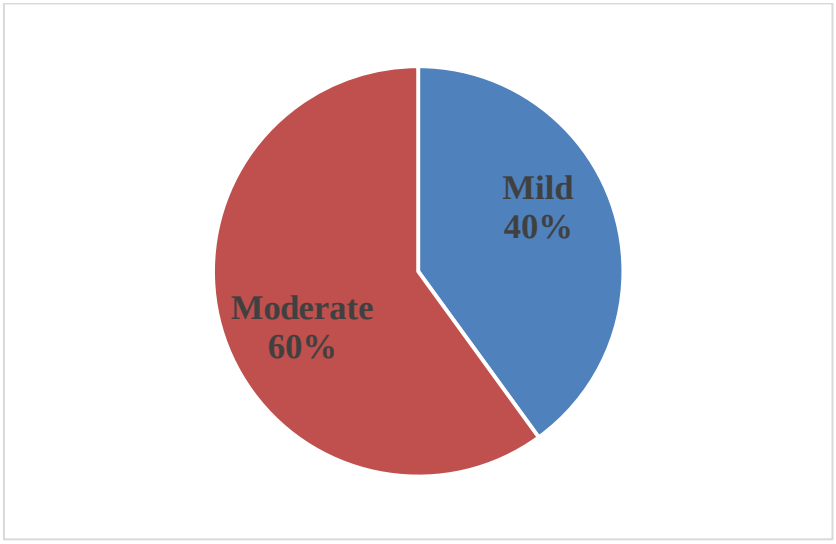



**FIGURE 12. DISTRIBUTION OF NIHSS SCORE AMONG STUDY POPULATION**

**TABLE 13. DISTRIBUTION OF SCANDINAVIAN STROKE SCALE (SSS) AMONG STUDY POPULATION**

| | | FREQUENCY | PERCENTAGE % |
|---|---|---|---|
| SCANDINAVIAN STROKE SCALE | VERY MILD (>40) | 8 | 4.0% |
| | MILD (26-40) | 66 | 33.0% |
| | MODERATE (10-25) | 53 | 26.5% |
| | SEVERE (<10) | 73 | 36.5% |

The present study classified the Scandinavian stroke scale among patients. Most of the patients had severe SSS- 73 (36.5%), followed by mild SSS-66 (33%), moderate SSS-53 (26.5%), and very mild SSS- 8 (4%) patients.

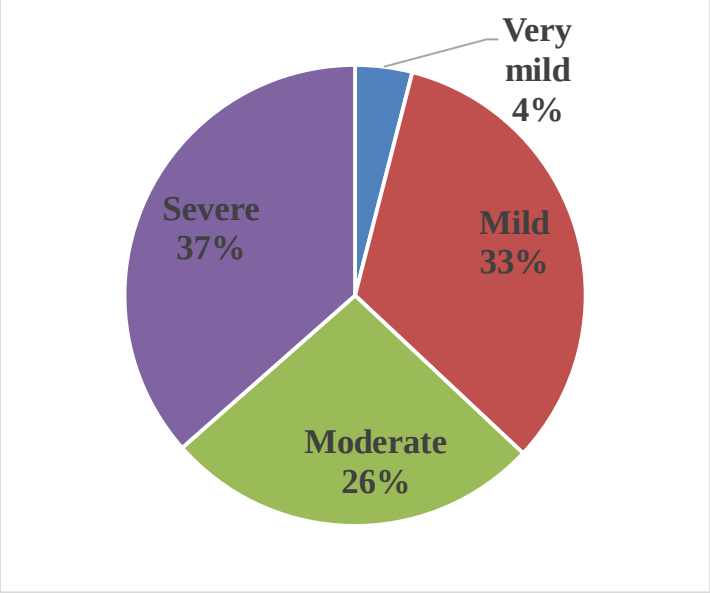



**FIGURE 13. DISTRIBUTION OF SCANDINAVIAN STROKE SCALE AMONG STUDY POPULATION**

**TABLE 14. DISTRIBUTION OF APACHE II SCORE AMONG STUDY POPULATION**

| | | FREQUENCY | PERCENTAGE % |
|---|---|---|---|
| APACHE II SCORE | VERY MILD (0-4) | 33 | 16.5% |
| | MILD (5-14) | 85 | 42.5% |
| | MODERATE (15-24) | 71 | 35.5% |
| | SEVERE ( 25 & MORE) | 11 | 5.5% |

The present study classified the APACHE II score among patients. Most patients had mild APACHE II - 85 (42.5%), followed by moderate APACHE II - 71 (35.5%), very mild APACHE II - 33 (16.5%), and severe APACHE II - 11 (5.5%) patients.

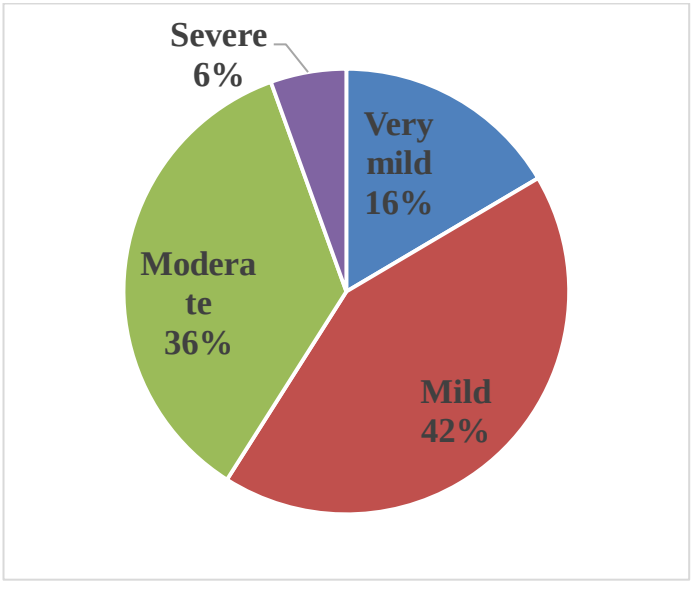



**FIGURE 14. DISTRIBUTION OF APACHE II SCORE AMONG STUDY POPULATION**

**TABLE 15. CROSS-TABULATION OF AGE BETWEEN MORTALITY AMONG THE STUDY POPULATION**

| | | | MORTALITY | | TOTAL | P VALUE |
|---|---|---|---|---|---|---|
| | | | NO | YES | | |
| AGE GROUP | 46-59 | FREQUENCY | 79 | 37 | 116 | <0.0001 |
| | | % | 77.5% | 37.8% | 58.0% | |
| | 60-69 | FREQUENCY | 17 | 18 | 35 | |
| | | % | 16.7% | 18.4% | 17.5% | |
| | >=70 | FREQUENCY | 6 | 43 | 49 | |
| | | % | 5.9% | 43.9% | 24.5% | |
| TOTAL | | FREQUENCY | 102 | 98 | 200 | |
| | | % | 100.0% | 100.0% | 100.0% | |

Among all, patients in age group more than 70 years are having higher mortality rate that other age group. There is a significant difference in age group between mortality ($p<0.0001$)

In this study, Age of the patient at admission was independently associated with a higher risk of early stroke unit mortality.

**TABLE 16. CROSS-TABULATION OF GENDER BETWEEN MORTALITY AMONG THE STUDY POPULATION**

| | | MORTALITY | | | | P VALUE |
|---|---|---|---|---|---|---|
| | | NO | | YES | | |
| | | FREQUENCY | % | FREQUENCY | % | |
| SEX | F | 34 | 51.5% | 32 | 48.5% | 0.919 |
| | M | 68 | 50.7% | 66 | 49.3% | |

In the present study, out of 200 patients, 32 (48.5%) female patients died, and 24 (51.5%) female patients survived. In male patients, 66 (49.3%) died, and 68 (50.7%) survived. There was no significant difference in gender between mortality (p=0.919)

In this study, Gender of the patient was not independently associated with a higher risk of early stroke unit mortality.

**TABLE 17. CROSS-TABULATION OF THE TIME INTERVAL BETWEEN THE ONSET OF WEAKNESS AND HOSPITAL ADMISSION BETWEEN MORTALITY AMONG THE STUDY POPULATION**

| | | MORTALITY | | | | P VALUE |
|---|---|---|---|---|---|---|
| | | NO | | YES | | |
| | | FREQUENCY | % | FREQUENCY | % | |
| TIME INTERVAL BETWEEN ONSET OF WEAKNESS AND HOSPITAL ADMISSION | 12-24 H | 36 | 43.9% | 46 | 56.1% | 0.009 |
| | 6-12 H | 58 | 52.7% | 52 | 47.3% | |
| | < 6 H | 8 | 100.0% | 0 | 0.0% | |

In the present study, comparing the time interval between the onset of weakness and hospital admission between mortality, among the patients who were admitted between 12-24h, 46 (56.1%) died and 36 (43.9%) patients survived. Among the patients who were admitted between 6-12h, 52 (47.3%) patients died and 36 (43.9%) patients survived, and none died at admission less than six hours. There was a significant difference in the time interval between the onset of weakness and hospital admission between mortality (p=0.009).

Among all, patients admitted within 6 hours had the least (practically zero) mortality.

In this study, time interval between the onset of weakness and hospital admission was independently associated with a higher risk of early stroke unit mortality.

**TABLE 18. CROSS-TABULATION OF DIABETIC MORTALITY**

| | | MORTALITY | | | | P VALUE |
|---|---|---|---|---|---|---|
| | | NO | | YES | | |
| | | FREQUENCY | % | FREQUENCY | % | |
| DIABETIC | NO | 63 | 71.6% | 25 | 28.4% | <0.0001 |
| | YES | 39 | 34.8% | 73 | 65.2% | |

In the present study, comparing the mortality among the diabetic, 73 (65.2%) diabetic patients died, and 39 (34.8%) patients survived. On the other hand, of patients without diabetes, 25 (28.4%) died, and 63 (71.6%) survived. There was a significant difference

in people with diabetes between mortality (p=<0.0001).

In this study, diabetes was independently associated with a higher risk of early stroke unit mortality.

**TABLE 19. CROSS-TABULATION OF HEART DISEASE BETWEEN MORTALITY**

| | | MORTALITY | | | | P VALUE |
|---|---|---|---|---|---|---|
| | | NO | | YES | | |
| | | FREQUENCY | % | FREQUENCY | % | |
| HEART DISEASES | NO | 98 | 56.0% | 77 | 44.0% | <0.0001 |
| | YES | 4 | 16.0% | 21 | 84.0% | |

In the present study, comparing the mortality among patients with heart disease, 21 (84%) heart disease patients died, and 4 (16%) patients survived. On the other hand, 77 (44%) died of patients without heart disease, and 98 (56%) survived. There was a significant difference in heart disease between mortality (p=<0.0001).

In this study, heart disease was independently associated with a higher risk of early stroke unit mortality.

**TABLE 20. CROSS-TABULATION OF HYPERTENSION BETWEEN MORTALITY**

| | | MORTALITY | | | | P VALUE |
|---|---|---|---|---|---|---|
| | | NO | | YES | | |
| | | FREQUENCY | % | FREQUENCY | % | |
| SYSTEMIC HYPERTENSION | NO | 22 | 84.6% | 4 | 15.4% | <0.0001 |
| | YES | 80 | 46.0% | 94 | 54.0% | |

In the present study, comparing the mortality among patients with systemic hypertension, 94 (54%) systemic hypertension patients died, and 80 (46%) patients survived. On the other hand, 4 (15.4%) died among patients without systemic hypertension, and 22 (84.6%) survived. There was a significant difference in systemic hypertension between mortality (p=<0.0001).

In this study, systemic hypertension was independently associated with a higher risk of early stroke unit mortality.

**TABLE 21. CROSS-TABULATION OF ADDICTION BETWEEN MORTALITY**

| | | MORTALITY | | | | P VALUE |
|---|---|---|---|---|---|---|
| | | NO | | YES | | |
| | | FREQUENCY | % | FREQUENCY | % | |
| ADDICTION | AL | 5 | 62.5% | 3 | 37.5% | 0.804 |
| | N | 74 | 50.3% | 73 | 49.7% | |
| | S | 16 | 55.2% | 13 | 44.8% | |
| | S, AL | 7 | 43.8% | 9 | 56.3% | |

*AL- alcoholic; S- smoking; N- no addiction*

The present study compared addiction between mortality; 13 (44.8%) smokers died, and 16 (55.2%) patients survived. 9 (56.3%) patients who were both alcohol consumers and smokers died, and 7 (43.8%) patients survived. 3 (37.5%) patients who were alcohol consumers died, and 5 (62.5%) patients survived. There was no significant difference in addiction between mortality (p=0.804).

In this study, addiction was not independently associated with a higher risk of early stroke unit mortality.

**TABLE 22. CROSS-TABULATION OF TYPES OF ACUTE ISCHEMIC STROKE BETWEEN MORTALITY**

| | | MORTALITY | | | | P VALUE |
|---|---|---|---|---|---|---|
| | | NO | | YES | | |
| | | FREQUENCY | % | FREQUENCY | % | |
| ACUTE ISCHEMIC STROKE - PRIMARY/ RECURRENT | P | 95 | 51.4% | 90 | 48.6% | 0.727 |
| | R | 7 | 46.7% | 8 | 53.3% | |

*P- primary; R- recurrent*

The present study compared mortality between patients with types of acute ischemic stroke; 90 (48.6%) patients with primary acute ischemic stroke died, and 95 (51.4%) patients survived. On the other hand, 8 (53.3%) patients with recurrent acute ischemic stroke died, and 7 (46.7%) patients survived. There was no significant difference in types of acute ischemic stroke between mortality (p=0.727). There was no mortality difference between primary or recurrent ischemic stroke in this study and thus, was not independently associated with a higher risk of early stroke unit mortality.

**TABLE 23. CROSS-TABULATION OF POSTERIOR CIRCULATION STROKE BETWEEN MORTALITY**

| | | MORTALITY | | | | P VALUE |
|---|---|---|---|---|---|---|
| | | NO | | YES | | |
| | | FREQUENCY | % | FREQUENCY | % | |
| POSTERIOR CIRCULATION STROKE | NO | 92 | 57.1% | 69 | 42.9% | <0.0001 |
| | YES | 10 | 25.6% | 29 | 74.4% | |

The present study compared posterior circulation stroke between mortality; 29 (74.4%) patients with posterior circulation stroke died, and 10 (25.6%) patients survived. Conversely, 69 (42.9%) patients without posterior circulation stroke died, and 92 (57.1%) patients survived. There was a significant difference in posterior circulation stroke between mortality (p=<0.0001).

In this study, posterior circulation stroke was independently associated with a higher risk of early stroke unit mortality.

**TABLE 24. CROSS-TABULATION OF GLASGOW COMA SCALE BETWEEN MORTALITY AMONG THE STUDY POPULATION**

| | | | MORTALITY | | TOTAL | P VALUE |
|---|---|---|---|---|---|---|
| | | | NO | YES | | |
| GCS | VERY MILD/NIL | FREQUENCY | 31 | 1 | 32 | <0.0001 |
| | | % within GCS | 96.9% | 3.1% | 100.0% | |
| | MILD | FREQUENCY | 38 | 19 | 57 | |
| | | % within GCS | 66.7% | 33.3% | 100.0% | |
| | MODERATE | FREQUENCY | 15 | 13 | 28 | |
| | | % within GCS | 53.6% | 46.4% | 100.0% | |
| | SEVERE | FREQUENCY | 18 | 65 | 83 | |
| | | % within GCS | 21.7% | 78.3% | 100.0% | |
| TOTAL | | FREQUENCY | 102 | 98 | 200 | |
| | | % within GCS | 51.0% | 49.0% | 100.0% | |

The present study compared the Glasgow Coma Scale between mortality; 65 (78.3%) patients with a severe GCS score died, and 18 (21.7%) patients survived. 19 (33.3%) patients with a mild GCS score died, and 38 (66.7%) patients survived. 13 (46.4%) patients with a moderate GCS score died, and 15 (53.6%) patients survived. Only one (3.1%) patient with a very mild GCS score died, and 31 (96.9%) patients survived. **There was a significant difference in the Glasgow coma scale between mortality (p=<0.0001).** Mortality was seen in more numbers in patients with severe GCS at admission. More severe the GCS at admission, greater the risk of mortality.

In this study, GCS was independently associated with a higher risk of early stroke unit mortality.

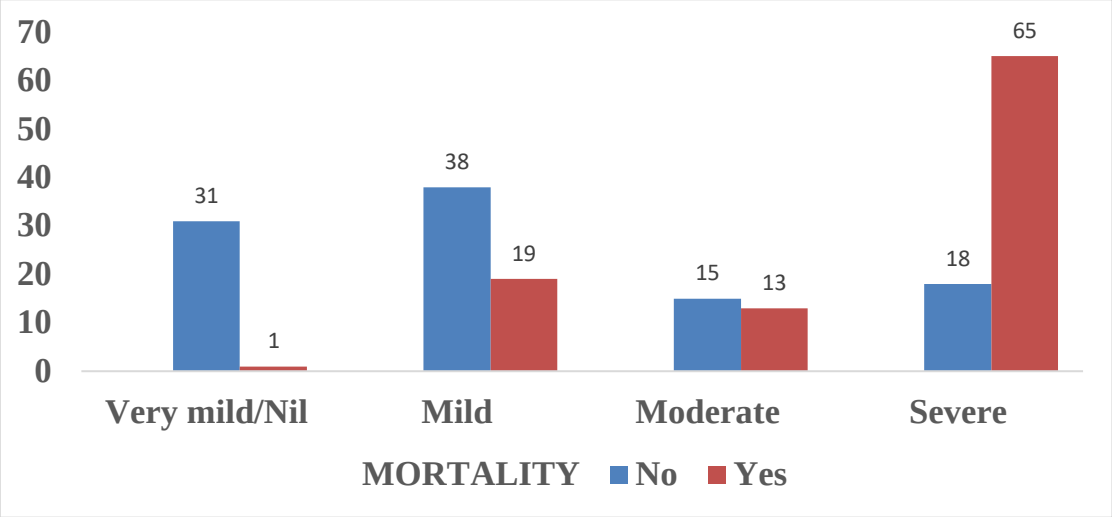



**FIGURE 15. DISTRIBUTION OF GLASGOW COMA SCALE BETWEEN MORTALITY**

**TABLE 25. CROSS-TABULATION OF NIHSS SCORES BETWEEN MORTALITY**

| | | | MORTALITY | | TOTAL | P VALUE |
|---|---|---|---|---|---|---|
| | | | NO | YES | | |
| NIHSS SCORE | MILD | FREQUENCY | 60 | 20 | 80 | <0.0001 |
| | | % within NIHSS SCORE | 75.0% | 25.0% | 100.0% | |
| | MODERATE | FREQUENCY | 42 | 78 | 120 | |
| | | % within NIHSS SCORE | 35.0% | 65.0% | 100.0% | |
| TOTAL | | FREQUENCY | 102 | 98 | 200 | |
| | | % within NIHSS SCORE | 51.0% | 49.0% | 100.0% | |

The present study compared the NIHSS scores between mortality; 78 (65%) patients with moderate NIHSS scores died, and 42 (35%) survived. Twenty (25%) patients with

mild NIHSS scores died, and 60 (75%) survived. None had severe NIHSS score. **There was a significant difference in the NIHSS scores between mortality (p=<0.0001).** More severe the NIHSS score at admission, greater the risk of mortality.

In this study, NIHSS Score was independently associated with a higher risk of early stroke unit mortality.

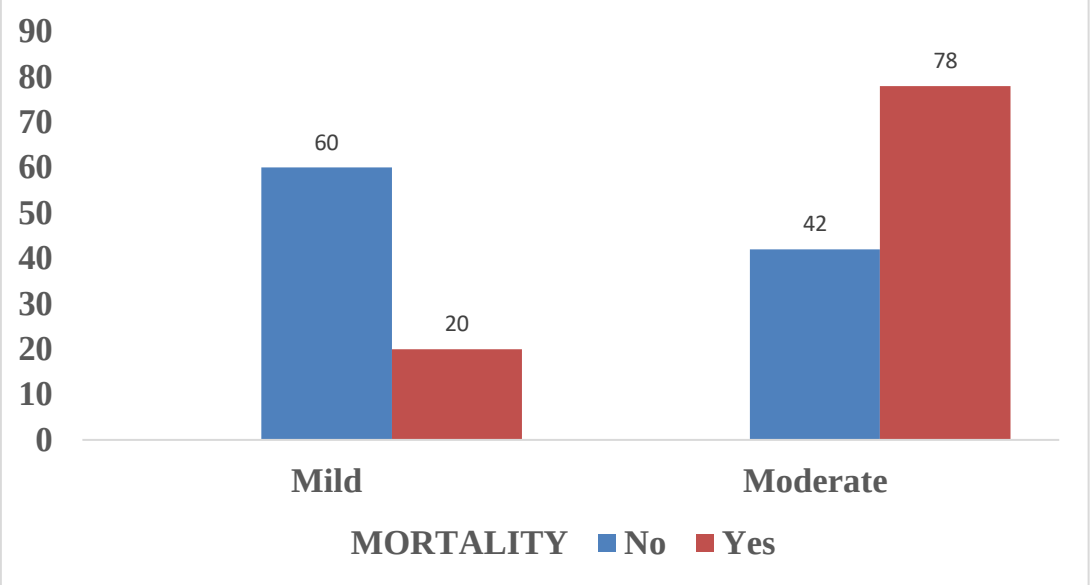



**FIGURE 16. DISTRIBUTION OF NIHSS SCORES BETWEEN MORTALITY**

**TABLE 26. CROSS-TABULATION OF SCANDINAVIAN STROKE SCALE (SSS) BETWEEN MORTALITY**

| | | | MORTALITY | | TOTAL | P VALUE |
|---|---|---|---|---|---|---|
| | | | NO | YES | | |
| SCANDINAVIAN STROKE SCALE | VERY MILD | FREQUENCY | 7 | 1 | 8 | <0.0001 |
| | | % within SSS | 87.5% | 12.5% | 100.0 % | |
| | MILD | FREQUENCY | 62 | 4 | 66 | |
| | | % within SSS | 93.9% | 6.1% | 100.0 % | |
| | MODERATE | FREQUENCY | 19 | 34 | 53 | |
| | | % within SSS | 35.8% | 64.2% | 100.0 % | |
| | SEVERE | FREQUENCY | 14 | 59 | 73 | |
| | | % within SSS | 19.2% | 80.8% | 100.0 % | |
| TOTAL | | FREQUENCY | 102 | 98 | 200 | |
| | | % within SSS | 51.0% | 49.0% | 100.0 % | |

The present study compared the Scandinavian Stroke Scale between mortality; 59 (80.8%) patients with a severe SSS score died, and 14 (19.2%) patients survived. 34 (64.2%) patients with a moderate SSS score died, and 19 (35.8%) patients survived. 4 (6.1%) patients with a mild SSS score died, and 62 (93.9%) patients survived. Only one (12.4%) patient with a very mild SSS score died, and 7 (87.5%) patients survived. **There was a significant difference in the Scandinavian Stroke Scale between mortality (p=<0.0001).**

Mortality was the highest in patients who had severe- SSS Score at admission followed by moderate- SSS Score.

In this study, SSS Score was independently associated with a higher risk of early stroke unit mortality.

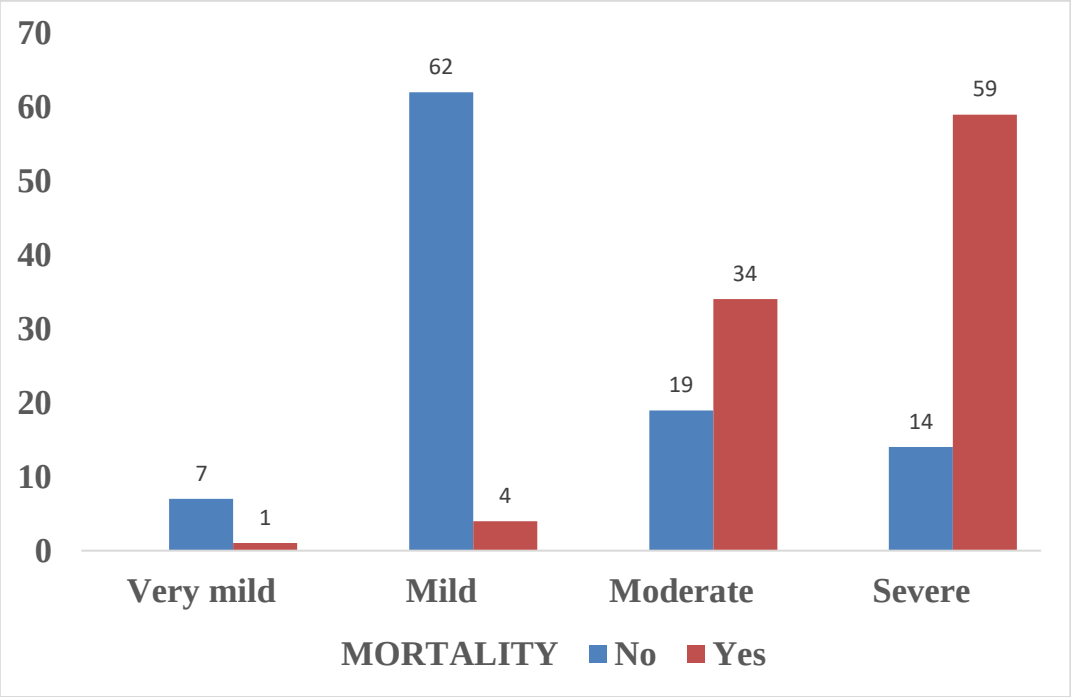



**FIGURE 17. DISTRIBUTION OF SCANDINAVIAN STROKE SCALE BETWEEN MORTALITY**

**TABLE 27. CROSS-TABULATION OF APACHE II SCORE BETWEEN MORTALITY**

| | | | MORTALITY | | TOTAL | P VALUE |
|---|---|---|---|---|---|---|
| | | | NO | YES | | |
| APACHE II SCORE | VERY MILD | FREQUENCY | 29 | 4 | 33 | <0.0001 |
| | | % within APACHE II SCORE | 87.9% | 12.1% | 100.0% | |
| | MILD | FREQUENCY | 54 | 31 | 85 | |
| | | % within APACHE II SCORE | 63.5% | 36.5% | 100.0% | |
| | MODERATE | FREQUENCY | 19 | 52 | 71 | |
| | | % within APACHE II SCORE | 26.8% | 73.2% | 100.0% | |
| | SEVERE | FREQUENCY | 0 | 11 | 11 | |
| | | % within APACHE II SCORE | 0.0% | 100.0 % | 100.0% | |
| TOTAL | | FREQUENCY | 102 | 98 | 200 | |
| | | % within APACHE II SCORE | 51.0% | 49.0% | 100.0% | |

The present study compared the APACHE II score between mortality; 52 (73.2%) patients with a moderate APACHE II score died, and 19 (26.8%) patients survived. 31 (36.5%) patients with a mild APACHE II score died, and 54 (63.5%) patients survived. 11 (100%) patients with a severe APACHE II score died, and no one survived. 4 (12.1%) patients with a very mild APACHE II score died, and 29 (87.9%) patients

survived. **There was a significant difference in the APACHE II score between mortality (p=<0.0001).**

Mortality was the highest in patients who had severe- APACHE II Score at admission followed by moderate- APACHE II Score.

In this study, APACHE II Score was independently associated with a higher risk of early stroke unit mortality

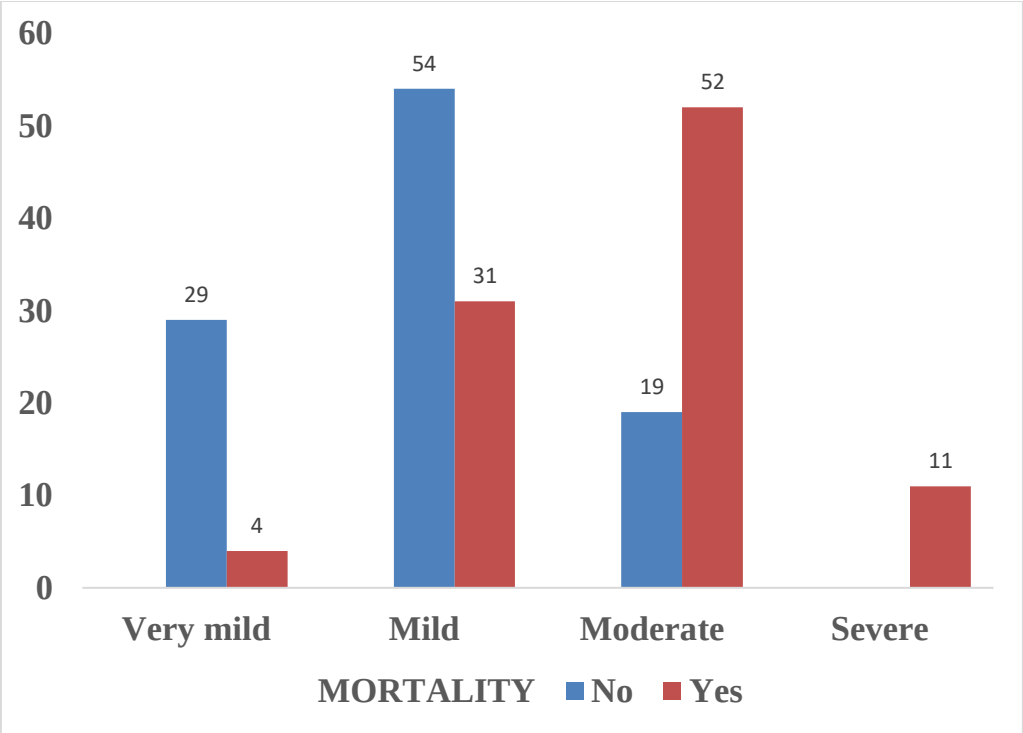



**FIGURE 18. DISTRIBUTION OF APACHE II SCORE BETWEEN MORTALITY**

**TABLE 28. ACCURACY OF NIHSS SCORE AS A MORTALITY PREDICTOR AMONG THE STUDY POPULATION**

| | | MORTALITY | | TOTAL |
|---|---|---|---|---|
| | | YES | NO | |
| NIHSS SCORE | >11.5 | 71 | 37 | 108 |
| | <11.5 | 27 | 65 | 92 |
| TOTAL | | 98 | 102 | 200 |

The present study compared the NIHSS scores between mortality; 71 patients with >11.5 scores died, and 37 survived. Twenty-seven patients with <11.5 scores died, and 65 survived.

**TABLE 29. ROC CURVE FOR NIHSS SCORE**

| | |
|---|---|
| **CUT OFF** | 11.5 |
| **AUC** | 0.737 |
| **P VALUE** | **<0.0001** |
| **SENSITIVITY** | 72.45% |
| **SPECIFICITY** | 63.73% |
| **PPV** | 65.74% |
| **NPV** | 70.65% |
| **ACCURACY** | 68.00% |

ROC curve was generated to predict the cut-off value of the NIHSS score in mortality patients. The cut-off value was 11.5, with an AUC of 0.737, with a significant p-value of <0.0001. With the cut-off value, the sensitivity of 72.45%, specificity of 63.73%, PPV of 65.74%, NPV of 70.65%, and accuracy of 68.00% were calculated.

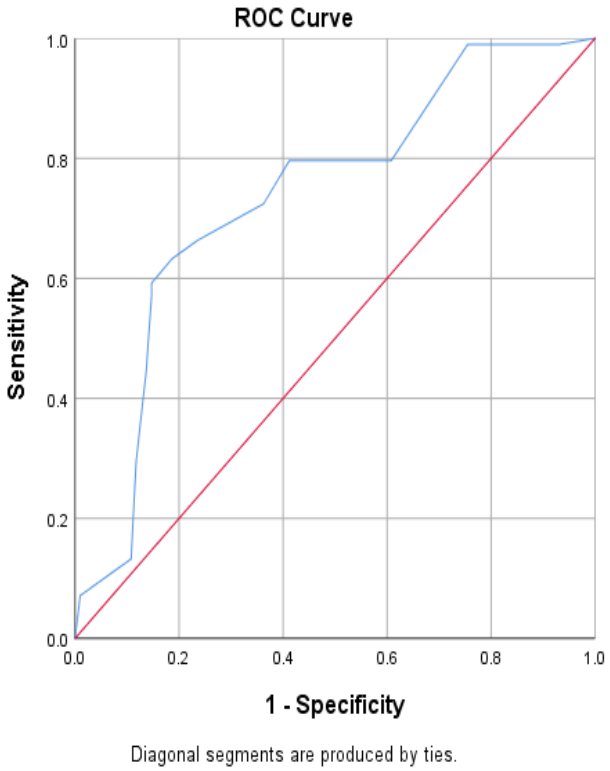



**FIGURE 19. ROC CURVE FOR NIHSS SCORES**

**TABLE 30. ACCURACY OF SCANDINAVIAN STROKE SCALE (SSS) SCORE AS A MORTALITY PREDICTOR AMONG THE STUDY POPULATION**

| | | MORTALITY | | TOTAL |
|---|---|---|---|---|
| | | YES | NO | |
| SCANDINAVIAN STROKE SCALE | <16.5 | 73 | 25 | 98 |
| | >16.5 | 25 | 77 | 102 |
| TOTAL | | 98 | 102 | 200 |

The present study compared the Scandinavian Stroke Scale between mortality; 73 patients with <16.5 scores died, and 25 survived. Twenty-five patients with >16.5 scores died, and 77 survived.

**TABLE 31. ROC CURVE FOR SCANDINAVIAN STROKE SCALE**

| | |
|---|---|
| **CUT OFF** | 16.5 |
| **AUC** | 0.852 |
| **P VALUE** | **<0.0001** |
| **SENSITIVITY** | 74.49% |
| **SPECIFICITY** | 75.49% |
| **PPV** | 74.49% |
| **NPV** | 75.49% |
| **ACCURACY** | 75.00% |

ROC curve was generated to predict the cut-off value of the Scandinavian stroke scale in mortality patients. The cut-off value was 16.5, with an AUC of 0.852, with a significant p-value of <0.0001. With the cut-off value, the sensitivity of 74.49%, specificity of 75.49%, PPV of 74.49%, NPV of 75.49%, and accuracy of 75.00% were calculated.

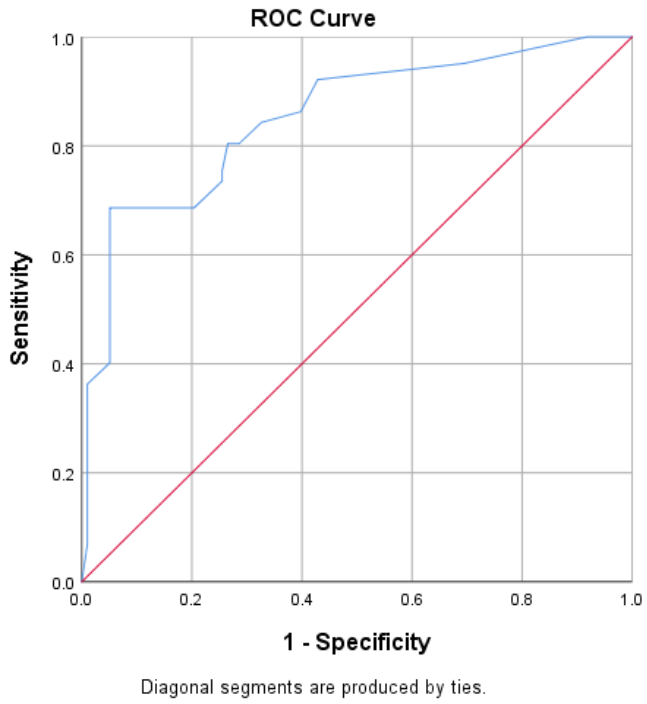



**FIGURE 20. ROC CURVE FOR SCANDINAVIAN STROKE SCALE**

**TABLE 32. ACCURACY OF APACHE II SCORE AS A MORTALITY PREDICTOR AMONG THE STUDY POPULATION**

| | | MORTALITY | | TOTAL |
|---|---|---|---|---|
| | | YES | NO | |
| APACHE II SCORE | >12.5 | 70 | 25 | 95 |
| | <12.5 | 28 | 77 | 105 |
| TOTAL | | 98 | 102 | 200 |

The present study compared the APACHE II score between mortality; 70 patients with >12.5 scores died, and 25 survived. Twenty-eight patients with <12.5 scores died, and 77 survived.

**TABLE 33. ROC CURVE FOR APACHE II SCORE**

| | |
|---|---|
| **CUT OFF** | 12.5 |
| **AUC** | 0.794 |
| **P VALUE** | **<0.0001** |
| **SENSITIVITY** | 71.43% |
| **SPECIFICITY** | 75.49% |
| **PPV** | 73.68% |
| **NPV** | 73.33% |
| **ACCURACY** | 73.50% |

ROC curve was generated to predict the cut-off value of the APACHE II score in mortality patients. The cut-off value was 12.5, with an AUC of 0.794, with a significant p-value of <0.0001. With the cut-off value, the sensitivity of 71.43%, specificity of 75.49%, PPV of 73.68%, NPV of 73.33%, and accuracy of 73.50% were calculated.

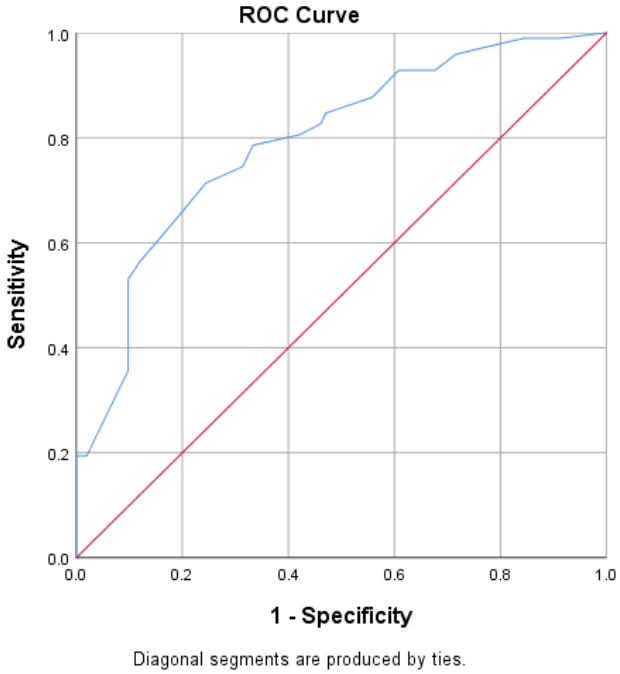



**FIGURE 21. ROC CURVE FOR APACHE II SCORE**

## DISCUSSION

Because of its outstanding diagnostic accuracy and practicability, the PREMISE score might help clinicians in the early bedside assessment of early mortality (≤7 days' post stroke) risk in Acute Ischemic Stroke patients.

One of the factors in the PREMISE score - the NIHSS had been widely used to assess the prognosis and degree of neurological deficit in stroke cases. Compared to other stroke scales, such as the Canadian Neurological Scale, the NIHSS had proved to be more accurate at predicting early mortality (mortality within 7 days or less). Although the scale's reliability had been evident, some parameters had consistently displayed moderate to low inter-rater reliability (kappa score less than 0.75). According to T. Askim et al, an alternative scale - the Scandinavian Stroke Scale was equally effective as the NIHSS Score in neurological assessment and also had excellent inter-rater reliability. [(3)] According to Chen et al., the APACHE II score had significant clinical importance and could be used for reasonable clinical assessment of illness and assessing the prognosis of acute ischemic stroke patients. [(4)]

Thus this study aimed to compare the efficiency of NIHSS, APACHE II AND SCANDINAVIAN STROKE SCALE in predicting the early mortality (≤7 days' post stroke) of acute ischemic stroke patients.

In-order to determine the risk variables for early stroke unit mortality following an acute ischemic stroke, data from this study was analysed and compared with the data from the studies by Gattringer et al., from the National Austrian Stroke Unit Registry [(1)];

Viderman D et al., from Kazakhstan [52]; Khurana S et al., from India [53]; and Dabilgou et al., from Africa. [54]

Over a period of 6 months, 200 patients were included in the study with a 7-day mortality rate of 49%. The early mortality (≤7 days' post stroke) according to Gattringer et al., was 21.6% in a secondary sensitivity analysis done in Austria, 36% for ischemic strokes by Viderman D et al., in Kazakhstan and 42.06% for ischemic strokes in urban population in India by Khurana S et al.,

In this study, the mean age of distribution was 60.45 years which is almost comparable to the study by Viderman D et al., (63 years) and less compared to the Austrian Stroke Unit Registry (74 years).

The following factors were the independent predictors associated with a higher risk of early stroke unit mortality in this study: Age, Time interval between the onset of weakness and hospital admission, Diabetes, Heart diseases, Systemic hypertension, Posterior Circulation Stroke, GCS score at admission, NIHSS score at admission, SSS score at admission, APACHE II score at admission. The following factors were not independently associated with a higher risk of early stroke unit mortality: Gender, Addiction, Recurrent ischemic stroke.

In this study, patients in age group more than 70 years were having higher mortality rate (43.9%) that other age group. Age of the patient at admission was independently associated with a higher risk of early stroke unit mortality ($p<0.0001$) similar to the Austrian Stroke Unit Registry.

In multivariate regularized logistic regression analysis, Female were 47% and Gender

of the patient was not an independent variable associated with early stroke unit mortality according to the Austrian Stroke Unit Registry. In this study also, females were 33% and Gender of the patient was not independently associated with a higher risk of early stroke unit mortality. (p=0.919)

In this study, among the time interval between the onset of weakness and hospital admission, the patients who were admitted between 12-24h had the highest mortality (56.1%). This factor was not individually considered as an independent variable associated with early stroke unit mortality in the Austrian Stroke Unit Registry which included all the patients admitted within 24 hours' post-stroke. In this study, there was a significant difference in the time interval between the onset of weakness and hospital admission between mortality (p=0.009) probably due to delay in seeking medical care and commencement of treatment.

In a multivariate logistic regression analysis, heart disease, diabetes mellitus, and posterior circulation stroke syndrome (compared with anterior circulation stroke syndromes) remained independently associated with a higher risk of early stroke unit mortality. ($p<0.001$) In this study, mortality among patients with diabetes, heart diseases, and posterior circulation stroke were 65.2%, 84% and 74.4% respectively. Thus, diabetes, heart diseases, and posterior circulation stroke were independently associated with a higher risk of early stroke unit mortality. ($p<0.0001$)

In this study, mortality among patients with Systemic hypertension was 54% and Systemic hypertension was independently associated with a higher risk of early stroke unit mortality ($p<0.0001$). This result is similar to the study by Dabilgou et al., where systemic hypertension was independently associated with higher early mortality.

Similar to the study by Gattringer et al., Addiction; and Recurrent ischemic stroke were not independently associated with a higher risk of early stroke unit mortality in this study. Their p-value were 0.804 and 0.727 respectively. The prevalence of Dyslipidaemia in this study was 6% similar to the study by Dhamija RK et al., in India where prevalence was 6.7% in 2016-17. [55]

In this study GCS, NIHSS, SSS and APACHE II scores were independently associated with a higher risk of early stroke unit mortality. There was a significant difference in all these scores between mortality (p=<0.0001). Mortality was the highest in patients who presented with severe GCS (3-8) at admission: 65 (78.3%) patients; moderate NIHSS (12-23) scores at admission- 78 (65%) patients; severe SSS Scores (<10) at admission: 59 (80.8%) patients and severe APACHE II Scores at admission: 11 (100%) patients. More severe the scores at admission, greater the risk of mortality. These results were similar to the studies by Viderman D et al., and by T. Askim et al.,

ROC curve was generated to predict the cut-off value of these scores in patients who died. In NIHSS score, the cut-off value was 11.5, with an AUC of 0.737, with a significant p-value of <0.0001. In SSS Score, the cut-off value was 16.5, with an AUC of 0.852, with a significant p-value of <0.0001. In APACHE II score, the cut-off value was 12.5, with an AUC of 0.794, with a significant p-value of <0.0001.

The AUC of all these scores were regarded as adequate. Even though the AUC for APACHE II Score and SSS were slightly larger when compared with NIHSS, there were no statistically significant differences between these scales.

| SCORES | NIHSS | SSS | APACHE II |
|---|---|---|---|
| **CUT OFF** | 11.5 | 16.5 | 12.5 |
| **AUC** | 0.737 | 0.852 | 0.794 |
| **P VALUE** | **<0.0001** | **<0.0001** | **<0.0001** |
| **SENSITIVITY** | 72.45% | 74.49% | 71.43% |
| **SPECIFICITY** | 63.73% | 75.49% | 75.49% |
| **PPV** | 65.74% | 74.49% | 73.68% |
| **NPV** | 70.65% | 75.49% | 73.33% |
| **ACCURACY** | 68.00% | 75.00% | 73.50% |

From a clinical perspective and patients' perspective of view, PPV and NPV were important. The PPV - NPV of both SSS Scores and APACHE II were 74.49% - 75.49% and 73.68% - 73.33% respectively. The PPV and NPV of NIHSS Score were 65.74% - 70.65% respectively. These points suggested that both SSS and APACHE II were equal in-fact slightly superior to NIHSS Score. But the sensitivity of APACHE II was slightly lower than that of NIHSS and SSS Scores.

The sensitivity and specificity of NIHSS were 72.45% and 63.73% respectively. The accuracy was 68%. The sensitivity and specificity of SSS were 74.49% and 75.49% respectively. The accuracy was 75%. The sensitivity and specificity of APACHE II were 71.43% and 75.49% respectively. The accuracy was 73.50%.

This study showed that the scores SSS and APACHE II were equally as good as the NIHSS Score in the prediction of early mortality (≤7 days' post stroke) of acute

ischemic stroke patients similar to the studies by Viderman D et al., and by T. Askim et al.,

Additionally, there was a clear difference in the APACHE II and NIHSS scores for patients in the death group and those in the survival group, and there was a significant correlation between severity and these scores. A possible explanation for why the APACHE II value for the death group was significantly higher than that for the survival group was that some APACHE II parameters, such as temperature and white blood cell count, were related to inflammation, and inflammation was involved in the pathological process of the Acute Ischemic Stroke patients.

Although the NIHSS, SSS and APACHE II Scores had a comparable measurement characteristic in terms of their capacity to detect outcomes, the SSS Score had a slight advantage due to its simplicity and practicality for usage in clinical settings.

# CONCLUSION

In this study,

- Most of the patients belonged to age group 46-59 years - (58%), followed by >70 years - (24.5%).
- Most of the patients were males - (67%) followed by females - (33%).
- The Time interval between the onset of weakness and hospital admission was 12-24h in most of the patients (56.1%).
- Systemic Hypertension (87%) was the most common co-morbidity followed by Diabetes (56%) and Heart Diseases (12.5%). Dyslipidaemia was noted in 6% patients.
- 19.5% patients had a Posterior Circulation Stroke.
- Most of the patients - (41.5%) had a GCS score of (3-8) – severe category at the time of admission.
- NIHSS – moderate category (12-23) was the neurological assessment score in most of the patients (60%).
- Most of the patients - (36.5%) had a severe SSS score (< 10) at the time of admission.
- APACHE II – mild category (5-14) was the neurological assessment score in most of the patients (42.5%).
- 49% was the mortality rate in this study.
- The following factors were **independently associated** with a higher risk of early stroke unit mortality: AGE, Time interval between the onset of weakness and hospital admission, Diabetes, Heart diseases, Systemic hypertension, Posterior

Circulation Stroke, GCS score at admission, NIHSS score at admission, SSS score at admission, APACHE II score at admission.

- The following factors were **not independently associated** with a higher risk of early stroke unit mortality: Gender, Addiction, Recurrent ischemic stroke.
- The PPV - NPV of both SSS Scores and APACHE II were 74.49% - 75.49% and 73.68% - 73.33% respectively. The PPV and NPV of NIHSS Score were 65.74% - 70.65% respectively. These points suggested that both SSS and APACHE II were equal in-fact slightly superior to NIHSS Score. But the sensitivity of APACHE II was slightly lower than that of NIHSS and SSS Scores.
- The sensitivity and specificity of NIHSS were 72.45% and 63.73% respectively. The accuracy was 68%. The sensitivity and specificity of SSS were 74.49% and 75.49% respectively. The accuracy was 75%. The sensitivity and specificity of APACHE II were 71.43% and 75.49% respectively. The accuracy was 73.50%.
- This study showed that The Scandinavian Stroke Scale (SSS) and the APACHE II score were as good as the NIHSS Score in the neurological assessment for the Prediction of Early Mortality (≤7 days' post stroke) of ACUTE ISCHEMIC STROKE Patients (PREMISE).
- Although the NIHSS, SSS and APACHE II Scores had a comparable measurement characteristic in terms of their capacity to detect outcomes, the SSS Score had a slight advantage due to its simplicity and practicality for usage in clinical settings.

## LIMITATIONS OF THE STUDY

- The time interval between the onset of weakness and hospital admission was >4.5 Hours in most of the patients and so specific treatment like Thrombolysis could not be started in most of them which could have improved the prognosis of the patients. This was due to delay in approaching the health care by the patients due to lack of awareness and resorting to alternative herbal treatment in most of the cases, and due to late referrals in few cases. Early hospitalization, appropriate management and timely referral for advanced management might reduce the morbidity and mortality of Stroke.
- This study was limited to assess the credibility of NIHSS, SSS and APACHE II Scores in the prediction of early mortality (≤7 days' post stroke) in Acute Ischemic Stroke patients and was not extended to assess the neurological outcome of these patients at 3 months follow up.
- The sample size of this study is relatively small and large population based studies in extensive Stroke Unit and multi-centre based studies are needed for further establishment of the outcome.

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**THE COMPARISON OF NIHSS, APACHE Ⅱ, AND SCANDINAVIAN STROKE SCALE IN THE PREDICTION OF EARLY MORTALITY (≤7 DAYS' POST STROKE) OF ACUTE ISCHEMIC STROKE PATIENTS – PREMISE - IN A TERTIARY CARE HOSPITAL.**

**PROFORMA**

Proforma No:

Name:

Age/Sex:

In Patient No.

Occupation:

Place:

Date of admission:

Time interval between onset of weakness and hospital admission:

Outcome at 7 days:

Date of discharge/death:

- **HISTORY TAKING:**

  - **PRESENTING COMPLAINTS:**

| QUESTIONS | YES/NO |
|---|---|
| H/o inability to move right/left upper and/or lower limb | |
| H/o deviation of angle of mouth | |
| H/o slurring of speech | |
| History relating to aetiology: headache/vomiting/epilepsy/blurring of vision/vomiting/fever/chest pain/palpitation | |
| H/o sensory disturbances | |
| H/o cranial nerve involvement | |
| H/o autonomic involvement | |

- **OTHER HISTORY:**

| **QUESTIONS** | **YES/NO** |
|---|---|
| Previous similar episodes/TIA OR RIND | |
| H/O Diabetes/Hypertension/ /Heart diseases/Other co-morbidity/ Recent vaccination/surgery/trauma | |
| H/O Smoking | |
| H/O Alcoholism | |
| H/O Treatment/drug abuse | |

- **EXAMINATION:**

| | **AT ADMISSION** |
|---|---|
| **VITALS** | |
| Heart rate | |
| Blood pressure | |
| Mean arterial pressure | |
| Respiratory rate | |
| Temperature | |
| Spo2 | |
| **HIGHER MOTOR FUNCTION** | |
| Consciousness | |
| Orientation to time, place, person | |
| Memory | |
| Intelligence | |
| Speech | |
| GCS | |
| **CRANIAL NERVE EXAMINATION** | |

| | |
|---|---|
| **MOTOR SYSTEM** | |
| Bulk | |
| Tone | |
| Power: MRC UL: Prox | |
| Distal | |
| LL: Prox | |
| Distal | |
| Reflexes: Superficial: | |
| Deep tendon reflexes: | |
| Plantar: | |
| Sensory involvement | |
| Cerebellar involvement | |
| Autonomic involvement | |
| Spine and cranium | |
| Gait | |
| Signs of meningeal irritation | |
| **OTHER SYSTEMS** | |

- **INVESTIGATIONS:**

| | **AT ADMISSION** |
|---|---|
| **Complete blood count:** | |
| Total count | |
| Haemoglobin | |
| haematocrit | |
| Platelet Count | |
| **Renal function tests:** | |
| Blood Sugar | |

| | |
|---|---|
| Blood Urea | |
| Serum Creatinine | |
| Serum Sodium | |
| Serum Potassium | |
| **Liver function tests:** | |
| Total Bilirubin | |
| SGOT | |
| SGPT | |
| SAP | |
| Total Proteins | |
| Serum Albumin | |
| ABG ARTERIAL pH<br>SERUM HCO3 | |
| **X RAY chest:** | |
| **ECG** | |
| **CT/MRI BRAIN:** | |
| **MODIFIED RANKIN SCALE:** | |
| **NIHSS SCORE:** | |
| **SCANDINAVIAN STROKE SCORE:** | |
| **APACHE II SCORE:** | |
| DURATION OF STAY IN HOSPITAL<br>OUTCOME: | |

## INFORMATION SHEET

We are conducting**, 'A STUDY ON THE COMPARISON OF NIHSS, APACHE II, AND SCANDINAVIAN STROKE SCALE IN THE PREDICTION OF EARLY MORTALITY (≤7 DAYS POST STROKE) OF ACUTE ISCHEMIC STROKE PATIENTS – PREMISE - IN A TERTIARY CARE HOSPITAL'** among patients attending Rajiv Gandhi Government General Hospital, Chennai and for that your specimen may be valuable to us.

We are selecting certain cases and if you are found eligible, we may be collecting your demographic details and your comorbidities. During the time of hospitalization, a detailed clinical history & neurological examination will be recorded and various scores will be calculated. On the subsequent days, patient will be reassessed and scores will be calculated. With the above collected data, these scores will be compared for their efficiency in predicting the in-hospital mortality.

The privacy of the patients in the research will be maintained throughout the study. In the event of any publication or presentation resulting from the research, no personally identifiable information will be shared.

Taking part in this study is voluntary. You are free to decide whether to participate in this study or to withdraw at any time; your decision will not result in any loss of benefits to which you are otherwise entitled.

Signature of Investigator Signature of Participant

## தகவல் தாள்

சென்னை ராஜீவ் காந்தி அரசு பொது மருத்துவமனையில் அனுமதிக்கப்படும் நோயாளிகளிடையே: 'A STUDY ON THE COMPARISON OF NIHSS, APACHE II, AND SCANDINAVIAN STROKE SCALE IN THE PREDICTION OF EARLY MORTALITY (≤7 DAYS POST STROKE) OF ACUTE ISCHEMIC STROKE PATIENTS – PREMISE - IN A TERTIARY CARE HOSPITAL' குறித்து ஒரு ஆய்வை நாங்கள் மேற்கொண்டு வருகிறோம், அதற்காக உங்கள் மாதிரி எங்களுக்கு மதிப்புமிக்கதாக இருக்கும்.

நாங்கள் சில நோயாளிகள் தேர்வு செய்கிறோம், நீங்கள் தகுதியுள்ளவராகக் கண்டறியப்பட்டால், சில சோதனைகளைச் செய்ய உங்கள் இரத்த மாதிரிகளை மற்றும் ஊடுகதிர் பயன்படுத்துகிறோம்.

ஆராய்ச்சியில் நோயாளிகளின் தனியுரிமை ஆய்வு முழுவதும் பராமரிக்கப்படும். ஆராய்ச்சியின் விளைவாக ஏதேனும் வெளியீடு அல்லது விளக்கக்காட்சி ஏற்பட்டால், தனிப்பட்ட முறையில் அடையாளம் காணக்கூடிய தகவல்கள் எதுவும் பகிரப்படாது.

**இந்த ஆய்வில் பங்கேற்பது தன்னார்வமானது. இந்த ஆய்வில் பங்கேற்க வேண்டுமா அல்லது எந்த நேரத்திலும் திரும்பப் பெறலாமா என்பதை நீங்கள் தீர்மானிக்க சுதந்திரமாக இருக்கிறீர்கள்; உங்கள் முடிவால் உங்களுக்கு உரிமையுள்ள எந்தவொரு நன்மைகளிலும் இடைவெளி ஏற்படாது.**

**புலனாய்வாளரின்**

**கையொப்பம்**

**பங்கேற்பாளரின்**

**கையொப்பம்**

## PATIENT CONSENT FORM

Study Detail : **'A STUDY ON THE COMPARISON OF NIHSS, APACHE II, AND SCANDINAVIAN STROKE SCALE IN THE PREDICTION OF EARLY MORTALITY (≤7 DAYS POST STROKE) OF ACUTE ISCHEMIC STROKE PATIENTS – PREMISE - IN A TERTIARY CARE HOSPITAL'**

Study Centre : Rajiv Gandhi Government General Hospital, Chennai.

Patient's Name :

Patient's Age :

Identification Number :

Patient may check (√) these boxes

I confirm that I have understood the purpose of procedure for the above stud have the opportunity to ask question and all my questions and doubts have b answered to my complete satisfaction.

I understand that my participation in the study is voluntary and that I am free withdraw at any time without giving reason, without my legal rights being affected.

I understand that my participation in the study is voluntary and that I am free withdraw at any time without giving reason, without my legal rights being affected.

I understand that sponsor of the clinical study, others working on the sponso behalf, the ethical committee and the regulatory authorities will not need my permission to look at my health records, both in respect of current study and any further research that may be conducted in relation to it, even if I withdra from the study I agree to this access. However, I understand that my identity will not be revealed in any information released to third parties or published unless as required under the law. I agree not to restrict the use of any data or results that arise from this study.

I agree to take part in the above study and to comply with the instructions gi during the study and faithfully cooperate with the study team and to immediately inform the study staff if I suffer from any deterioration in my health or wellbeing or any unexpected or unusual symptoms.

I hereby consent to participate in this study.

I hereby give permission to undergo detailed clinical examination and blood investigations as required.

Signature of investigator Signature/Thumb impression of participant

Patient Name and address :

நோயாளி ஒப்புதல் படிவம்

ஆய்வு விவரம் **: 'A STUDY ON THE COMPARISON OF NIHSS, APACHE II, AND SCANDINAVIAN STROKE SCALE IN THE PREDICTION OF EARLY MORTALITY (≤7 DAYS POST STROKE) OF ACUTE ISCHEMIC STROKE PATIENTS – PREMISE - IN A TERTIARY CARE HOSPITAL'**

ஆய்வு மையம்: ராஜீவ் காந்தி அரசு பொது மருத்துவமனை, சென்னை.

நோயாளியின் பெயர்:

நோயாளியின் வயது:

அடையாள எண் :

நோயாளி இந்த பெட்டிகளை (√)செய்யலாம்**:-**

1. மேற்கண்ட ஆய்விற்கான நடைமுறையின் நோக்கத்தை நான் புரிந்து கொண்டேன் என்பதை உறுதிப்படுத்துகிறேன். கேள்வி கேட்க எனக்கு வாய்ப்பு உள்ளது, எனது முழுதிருப்திக்கும் எனது எல்லா கேள்விகளுக்கும் சந்தேகங்களுக்கும் பதில் அளிக்கப்பட்டுள்ளது.
2. ஆய்வில் நான் பங்கேற்பது தன்னார்வமானது என்பதையும், எனது சட்டஉரிமைகள் பாதிக்கப்படாமல், காரணமின்றி

எந்த நேரத்திலும் திரும்பப்பெற எனக்கு சுதந்திரம் உள்ளது என்பதையும் நான் புரிந்து கொள்கிறேன்.

**3.** மருத்துவ ஆய்வின் ஸ்பான்சர், ஸ்பான்சர்சார்பாக பணிபுரியும் மற்றவர்கள், நெறிமுறைக்குழு மற்றும் ஒழுங்குமுறை அதிகாரிகள் எனது சுகாதாரபதிவுகளைப் பார்க்க எனது அனுமதி தேவையில்லை என்பதை நான் புரிந்து கொள்கிறேன், தற்போதைய ஆய்வு மற்றும் மேற்கொண்டுள்ள எந்தவொரு ஆராய்ச்சியையும் பொறுத்தவரை இது தொடர்பாக, நான் ஆய்வில் இருந்து விலகினாலும் இந்த அணுகலை ஒப்புக்கொள்கிறேன். எவ்வாறாயினும் , சட்டத்தின்கீழ் தேவைப்படாவிட்டால், மூன்றாம் தரப்பினருக்கு வெளியிடப்பட்ட அல்லது வெளியிடப்பட்ட எந்தவொரு தகவலிலும் எனது அடையாளம் வெளிப்படுத்தப்படாது என்பதை நான் புரிந்து கொள்கிறேன். இந்த ஆய்வில் எழும் எந்தவொரு தரவு அல்லது முடிவுகளின் பயன்பாட்டை கட்டுப்படுத்த வேண்டாம் என்று நான் ஒப்புக்கொள்கிறேன்.

**4.** மேற்கண்ட ஆய்வில் பங்கேற்கவும், ஆய்வின் போது கொடுக்கப்பட்ட அறிவுறுத்தல்களுக்கு இணங்கவும், ஆய்வுக்குழுவுடன் உண்மையுடன் ஒத்துழைக்கவும், எனது

உடல்நலம் அல்லது நல்வாழ்வில் ஏதேனும் சரிவுஏற்பட்டால் அல்லது எதிர்பாராத அல்லது ஏதேனும் ஏற்பட்டால் உடனடியாக ஆய்வு ஊழியர்களுக்கு அறிவிக்கவும் ஒப்புக்கொள்கிறேன். அசாதாரண அறிகுறிகள்.

5. இந்த ஆய்வில் பங்கேற்கநான் இதன் மூலம் ஒப்புக்கொள்கிறேன்.

6. விசாரணைகளை மேற்கொள்ள நான் இதன் மூலம் அனுமதி அளிக்கிறேன்.

புலனாய்வாளரின் கையொப்பம்

பங்கேற்பாளரின் கையொப்பம் /

கட்டைவிரல் எண்ணம்

நோயாளியின் பெயர் மற்றும் முகவரி

# ETHICS COMMITTEE APPROVAL LETTER

## INSTITUTIONAL ETHICS COMMITTEE
## MADRAS MEDICAL COLLEGE, CHENNAI 600 003

EC Reg. No(CDSCO).ECR/270/Inst./TN/2013/RR-20
EC Reg. No(DHR).EC/NEW/INST/2021/1618
Telephone No.044 25305301
Fax: 011 25363970

**CERTIFICATE OF APPROVAL**

To
**Dr. POOJA L,**
Post Graduate student,
MD General Medicine,
Institute of Internal Medicine,
Madras Medical College,
Chennai-600 003.

Dear Dr. POOJA L,

The Institutional Ethics Committee has considered your request and approved your study titled **"A STUDY ON THE COMPARISON OF NIHSS, APACHE II, AND SCANDINAVIAN STROKE SCALE IN THE PREDICTION OF EARLY MORTALITY (≤7 DAYS POST STROKE) OF ACUTE ISCHEMIC STROKE PATIENTS – PREMISE – IN A TERTIARY CARE HOSPITAL"- NO.28052022.** The following members of Ethics Committee were present in the meeting held on **18.05.2022** conducted at Madras Medical College, Chennai 3.

1. Prof.P.V.Jayashankar,MS Orth.,D.Orth.,M.Ch Orth (Liverpool) :Chairperson
2. Prof.N.Gopalakrishnan,MD.,DM., FRCP, Director, Inst.of Nephrology,MMC,Ch. : Member Secretary
3. Prof. K.M.Sudha, Prof. Inst. of Pharmacology,MMC,Ch-3 : Member
4. Prof. Alagarsamy Jamila ,MD, Vice Principal, Stanley Medical College, Chennai : Member
5. Prof.Meena Suresh, MD.,DGO.,Prof.of Obst & Gynaec, IOG,Chennai : Member
6. Prof.S.Lakshmi, Prof. of Paediatrics ICH Chennai :Member
7. Tmt.Arnold Saulina, MA.,MSW., :Social Scientist
8. Thiru S.Govindasamy, BA.,BL,High Court,Chennai : Lawyer
9. Thiru K.Ranjith, Ch- 91 : Lay Person

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

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

Member Secretary – Ethics Committee

MEMBER SECRETARY
INSTITUTIONAL ETHICS COMMITTEE
MADRAS MEDICAL COLLEGE
CHENNAI-600 003.

# PLAGIARISM DIGITAL RECEIPT

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| **Analysis address** | poojaloganathan.tnmg@analysis.urkund.com |

## Sources included in the report

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# MASTER CHART

| S.NO | NAME | AGE | SEX | D.O.A | TIME INTERVAL BETWEEN ONSET OF WEAKNESS AND HOSPITAL ADMISSION | DIABETIC | HEART DISEASES | SYSTEMIC HYPERTENSION | OTHER COMORBIDITY | ADDICTION | ACUTE ISCHEMIC STROKE - PRIMARY / RECURRENT | POSITIVE FOR CIRCULATION STROKE | GCS | MOTOR GRADING SCALE | NIHSS SCORE | SCANDINAVIAN STROKE SCALE | MRS SCORE | OUTCOME AFTER 7 DAYS | DURATION OF STAY IN HOSPITAL IN DAYS |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1 | RADHAKRISHNAN | 64 | M | 2-6-22 | 6-12 H | N | N | N | N | S | P | N | 15 | 4 | 13 | 29 | 4 | A | 5 |
| 2 | GOPI | 46 | M | 2-6-22 | 12-24 H | N | N | Y | CKD | S | P | N | 9 | 5 | 14 | 17 | 20 | A | 11 |
| 3 | KOKIAMMAL | 75 | F | 2-6-22 | 6-12 H | N | N | Y | N | N | P | N | 8 | 5 | 16 | 13 | 18 | D | 6 |
| 4 | MYTHILI | 48 | F | 2-6-22 | LESS 6 H | N | N | N | N | N | P | N | 15 | 3 | 8 | 39 | 2 | A | 5 |
| 5 | MUTHU | 76 | M | 2-6-22 | 12-24 H | N | N | Y | AKI | N | P | N | 10 | 5 | 14 | 4 | 32 | D | 4 |
| 6 | BHAVANI | 47 | F | 3-6-22 | 6-12 H | Y | N | Y | N | N | P | N | 14 | 3 | 10 | 35 | 10 | A | 9 |
| 7 | SRINIVASAN | 54 | M | 3-6-22 | 6-12 H | Y | N | Y | N | S | R | N | 8 | 5 | 23 | 4 | 15 | D | 6 |
| 8 | KRISHNAN | 54 | M | 3-6-22 | 12-24 H | Y | N | Y | L | S,AL | P | N | 8 | 5 | 19 | 4 | 20 | D | 6 |
| 9 | ABDUL RAZID | 72 | M | 5-6-22 | 6-12 H | Y | Y | Y | L | N | P | Y | 13 | 4 | 9 | 23 | 7 | D | 5 |
| 10 | KALAVATHY | 55 | F | 5-6-22 | 6-12 H | Y | N | Y | N | N | P | N | 15 | 4 | 9 | 35 | 6 | A | 5 |
| 11 | CHINNAKUZHANDHAI | 57 | F | 5-6-22 | 12-24 H | Y | N | Y | N | N | P | N | 8 | 5 | 21 | 4 | 15 | A | 10 |
| 12 | JOTHIYAMMAL | 55 | F | 5-6-22 | 12-24 H | Y | N | Y | CKD | N | P | N | 14 | 5 | 10 | 29 | 22 | A | 9 |
| 13 | IBRAHIM | 76 | M | 5-6-22 | 12-24 H | Y | N | Y | AKI | N | P | N | 7 | 5 | 18 | 6 | 39 | D | 2 |
| 14 | VENKATESHWARALU | 48 | M | 8-6-22 | 12-24 H | N | N | Y | L | N | P | N | 8 | 5 | 18 | 6 | 20 | D | 4 |
| 15 | VANITHA | 58 | F | 8-6-22 | 6-12 H | N | N | N | N | N | P | N | 15 | 3 | 5 | 48 | 3 | A | 5 |
| 16 | MANORMANI | 76 | F | 10-6-22 | 6-12 H | N | N | Y | N | N | P | N | 13 | 5 | 13 | 22 | 14 | D | 6 |
| 17 | SALIMA | 60 | F | 10-6-22 | 6-12 H | Y | N | Y | N | N | P | N | 8 | 5 | 20 | 6 | 13 | D | 6 |
| 18 | ELUMALAI | 74 | M | 10-6-22 | 6-12 H | N | N | Y | N | N | P | N | 13 | 4 | 8 | 35 | 10 | A | 5 |
| 19 | KANAGAVALLI | 74 | F | 10-6-22 | 6-12 H | Y | N | Y | N | N | P | Y | 3 | 5 | 20 | 2 | 17 | D | 1 |
| 20 | MOHAN | 58 | M | 14-6-22 | 12-24 H | Y | N | Y | N | N | P | N | 7 | 5 | 12 | 6 | 21 | D | 6 |
| 21 | SHANKARAN | 70 | M | 14-6-22 | 12-24 H | Y | Y | Y | L | S,AL | P | N | 10 | 4 | 9 | 31 | 16 | D | 5 |
| 22 | RAVISHANKARAN | 64 | M | 17-6-22 | 6-12 H | Y | Y | Y | N | N | P | Y | 13 | 4 | 9 | 23 | 5 | D | 6 |
| 23 | ELUMALAI | 74 | M | 17-6-22 | 6-12 H | N | N | Y | L | N | P | Y | 13 | 4 | 11 | 29 | 7 | A | 15 |
| 24 | AMEENA KHATHOON | 83 | F | 17-6-22 | 12-24 H | N | N | Y | L | N | R | N | 8 | 5 | 21 | 8 | 17 | D | 4 |
| 25 | SUBBULAXMI | 55 | F | 17-6-22 | 6-12 H | Y | N | Y | N | N | P | Y | 11 | 5 | 19 | 12 | 8 | A | 8 |
| 26 | GOVINDAMMAL | 83 | F | 17-6-22 | 6-12 H | Y | N | Y | L | N | R | N | 8 | 5 | 23 | 4 | 19 | D | 6 |
| 27 | AZHAGESHWARI | 65 | F | 17-6-22 | 12-24 H | Y | N | Y | N | N | P | N | 7 | 5 | 12 | 6 | 23 | D | 6 |
| 28 | SARALA | 70 | F | 21-6-22 | 12-24 H | Y | N | Y | AKI | N | P | N | 3 | 5 | 18 | 6 | 38 | D | 3 |
| 29 | SUJATHA | 50 | F | 21-6-22 | 6-12 H | N | N | Y | CKD | N | P | N | 13 | 4 | 8 | 35 | 7 | A | 5 |
| 30 | SARASWATHI | 75 | F | 21-6-22 | 12-24 H | N | N | Y | N | N | P | N | 4 | 5 | 18 | 6 | 26 | D | 4 |
| 31 | MOORTHY | 51 | M | 21-6-22 | 6-12 H | N | N | N | N | N | P | N | 15 | 3 | 5 | 48 | 2 | A | 5 |
| 32 | LOGANATHAN | 47 | M | 21-6-22 | 6-12 H | N | N | Y | L | N | P | Y | 13 | 4 | 11 | 29 | 4 | A | 9 |
| 33 | CHINNA PAYAN | 51 | M | 22-6-22 | LESS 6 H | N | N | N | N | N | P | N | 15 | 5 | 8 | 28 | 2 | A | 5 |
| 34 | NABI | 50 | M | 23-6-22 | 6-12 H | Y | Y | Y | N | N | P | Y | 13 | 4 | 9 | 23 | 4 | D | 6 |
| 35 | NARAYANAN | 49 | M | 25-6-22 | 12-24 H | N | N | Y | L | AL | R | N | 8 | 5 | 21 | 8 | 13 | A | 18 |
| 36 | SHANTHI | 59 | F | 25-6-22 | 6-12 H | N | N | N | N | N | P | N | 13 | 4 | 10 | 34 | 8 | A | 5 |
| 37 | GOVINDHAN | 58 | M | 25-6-22 | 6-12 H | Y | N | Y | N | S | P | N | 8 | 5 | 20 | 6 | 13 | D | 6 |
| 38 | MOHAMMED MOIDEEN | 70 | M | 29-6-22 | 12-24 H | Y | Y | Y | L | AL | P | N | 10 | 4 | 9 | 31 | 18 | D | 6 |
| 39 | SIVAPRAKASH | 67 | M | 29-6-22 | 12-24 H | Y | N | Y | L | S,AL | P | N | 8 | 5 | 19 | 4 | 23 | D | 5 |
| 40 | ANTHONY RAJ | 55 | M | 29-6-22 | 6-12 H | Y | N | Y | L | N | P | Y | 3 | 5 | 20 | 2 | 15 | D | 6 |
| 41 | NAGARAJAN | 56 | M | 29-6-22 | 6-12 H | Y | N | Y | N | N | P | N | 14 | 3 | 10 | 35 | 11 | A | 5 |
| 42 | RAGHU | 59 | M | 29-6-22 | 6-12 H | Y | N | Y | N | S | P | Y | 11 | 5 | 19 | 12 | 8 | A | 9 |
| 43 | DURAI SINGH | 55 | M | 29-6-22 | 6-12 H | Y | N | Y | N | N | P | N | 15 | 4 | 9 | 35 | 6 | A | 5 |
| 44 | GOPINATH | 58 | M | 30-6-22 | 12-24 H | Y | N | Y | N | N | P | N | 8 | 5 | 21 | 4 | 15 | A | 9 |
| 45 | KAVIYARASU | 53 | M | 30-6-22 | 12-24 H | N | N | Y | L | N | P | N | 9 | 4 | 12 | 18 | 13 | A | 9 |
| 46 | SARAVANAN | 77 | M | 30-6-22 | 6-12 H | Y | Y | Y | L | N | P | Y | 13 | 4 | 9 | 23 | 8 | D | 4 |
| 47 | RAMACHANDRAN | 75 | M | 30-6-22 | 6-12 H | N | N | Y | N | N | P | N | 13 | 5 | 13 | 22 | 14 | D | 6 |
| 48 | RAJA | 47 | M | 30-6-22 | 12-24 H | N | N | Y | L | N | P | N | 8 | 5 | 18 | 6 | 20 | D | 5 |
| 49 | NAGAMMAL | 59 | F | 1-7-22 | 6-12 H | N | N | Y | N | N | P | N | 8 | 5 | 16 | 13 | 15 | A | 18 |
| 50 | AMSAVALLI | 56 | F | 1-7-22 | 12-24 H | Y | N | Y | N | N | P | N | 15 | 4 | 9 | 35 | 6 | A | 5 |
| 51 | CHANDRAN | 49 | M | 1-7-22 | 6-12 H | Y | N | N | L | N | P | Y | 3 | 5 | 20 | 2 | 17 | D | 5 |
| 52 | SASI KUMAR | 78 | M | 3-7-22 | 6-12 H | N | N | Y | N | N | P | N | 13 | 5 | 13 | 22 | 14 | D | 5 |
| 53 | MURUGAN | 51 | M | 3-7-22 | 6-12 H | Y | N | Y | N | S | P | Y | 11 | 5 | 19 | 12 | 7 | D | 6 |
| 54 | JAYABAL | 68 | F | 3-7-22 | LESS 6 H | N | N | N | N | N | P | N | 14 | 5 | 9 | 26 | 6 | A | 5 |
| 55 | PUSHPAVATHY | 66 | F | 3-7-22 | 6-12 H | Y | Y | Y | N | N | P | Y | 8 | 4 | 13 | 16 | 12 | D | 4 |
| 56 | ANANDHARAJ | 61 | M | 3-7-22 | 12-24 H | N | N | Y | L | N | P | N | 8 | 5 | 18 | 6 | 21 | D | 4 |
| 57 | CHELLAIYA | 75 | M | 3-7-22 | 12-24 H | Y | Y | Y | L | S | P | N | 10 | 4 | 9 | 31 | 20 | D | 5 |
| 58 | GHOUSE | 47 | M | 3-7-22 | 6-12 H | Y | N | Y | N | AL | P | N | 8 | 5 | 23 | 4 | 15 | D | 6 |
| 59 | SELVAKUMAR | 54 | M | 4-7-22 | 6-12 H | N | N | Y | L | S,AL | P | Y | 13 | 4 | 11 | 29 | 4 | A | 5 |
| 60 | VIJAYA | 54 | F | 4-7-22 | 12-24 H | Y | N | Y | L | N | P | N | 8 | 5 | 19 | 4 | 20 | D | 6 |
| 61 | KAVI ARASAN | 58 | M | 4-7-22 | 12-24 H | Y | N | Y | CKD | N | P | N | 14 | 5 | 10 | 29 | 22 | A | 9 |
| 62 | MURALI | 54 | M | 4-7-22 | 12-24 H | N | N | Y | N | N | P | N | 15 | 5 | 12 | 22 | 3 | A | 9 |
| 63 | VASEEKARAN | 54 | M | 9-7-22 | 6-12 H | Y | Y | Y | N | N | P | Y | 13 | 4 | 9 | 23 | 4 | D | 5 |
| 64 | RAJARAJA SOZHAN | 56 | M | 9-7-22 | 6-12 H | Y | N | Y | N | N | P | N | 14 | 3 | 10 | 35 | 11 | A | 5 |
| 65 | SARAVANAN | 61 | M | 13-7-22 | 6-12 H | Y | Y | N | L | N | P | Y | 13 | 4 | 9 | 23 | 5 | D | 5 |
| 66 | MUTHULAKSHMI | 72 | F | 13-7-22 | 12-24 H | Y | N | Y | N | N | P | N | 7 | 5 | 12 | 6 | 23 | D | 3 |
| 67 | VELU | 80 | M | 13-7-22 | 6-12 H | N | N | N | N | S | P | N | 13 | 4 | 13 | 28 | 13 | A | 9 |
| 68 | JOHNSON | 49 | M | 13-7-22 | 6-12 H | N | N | Y | N | N | P | N | 13 | 4 | 8 | 35 | 7 | A | 5 |
| 69 | PARAMESHWARI | 53 | F | 16-7-22 | 6-12 H | Y | N | Y | N | N | P | N | 8 | 5 | 20 | 6 | 12 | D | 6 |
| 70 | SULOCHANA | 78 | F | 16-7-22 | 6-12 H | N | N | Y | N | N | P | N | 8 | 5 | 16 | 13 | 18 | D | 6 |
| 71 | SANU | 46 | F | 16-7-22 | 6-12 H | N | N | N | N | N | P | N | 15 | 3 | 5 | 48 | 2 | A | 5 |
| 72 | RAMESH | 48 | M | 16-7-22 | 12-24 H | Y | N | Y | N | N | P | N | 15 | 4 | 13 | 29 | 4 | A | 9 |
| 73 | THIRUNAVUKARASU | 65 | M | 16-7-22 | 6-12 H | Y | N | Y | N | N | P | N | 15 | 4 | 9 | 35 | 8 | A | 9 |
| 74 | SUBRAMANI | 64 | M | 17-7-22 | 12-24 H | Y | N | Y | N | N | P | N | 8 | 5 | 21 | 4 | 15 | D | 6 |

| 75 | MARIYA PUSHPAM | 48 | F | 17-7-22 | 12-24 H | N | N | Y | CKD | N | P | N | 9 | 5 | 14 | 17 | 20 | A | 9 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 76 | CHANDRAN | 58 | M | 17-7-22 | 12-24 H | N | N | Y | CKD | S | P | N | 9 | 5 | 14 | 17 | 21 | A | 7 |
| 77 | ANBU | 54 | M | 20-7-22 | 6-12 H | Y | N | Y | N | N | P | N | 8 | 5 | 20 | 6 | 12 | A | 11 |
| 78 | UDAYA KUMAR | 51 | M | 20-7-22 | 12-24 H | Y | N | Y | N | N | P | N | 15 | 4 | 13 | 29 | 4 | A | 9 |
| 79 | SELVI | 54 | F | 23-7-22 | 6-12 H | N | N | Y | N | N | P | N | 13 | 4 | 8 | 35 | 7 | A | 5 |
| 80 | UNNAMALAI | 70 | F | 25-7-22 | 6-12 H | Y | N | Y | N | N | P | Y | 3 | 5 | 20 | 2 | 17 | D | 2 |
| 81 | BHAGAVATHI | 59 | F | 25-7-22 | 6-12 H | N | N | Y | N | N | P | N | 8 | 5 | 16 | 13 | 15 | A | 13 |
| 82 | PANDIYAN | 59 | M | 25-7-22 | 12-24 H | Y | N | Y | N | N | P | N | 8 | 5 | 21 | 4 | 15 | A | 9 |
| 83 | RAJESHWARI | 65 | F | 25-7-22 | 6-12 H | N | N | Y | N | N | P | N | 13 | 5 | 13 | 22 | 13 | A | 12 |
| 84 | RAVI | 52 | M | 30-7-22 | 12-24 H | Y | N | Y | L | N | P | N | 7 | 5 | 12 | 6 | 20 | D | 6 |
| 85 | DEVI | 54 | F | 30-7-22 | 6-12 H | N | N | N | H | N | P | N | 15 | 4 | 13 | 29 | 3 | A | 5 |
| 86 | SENTHILKUMAR | 47 | M | 30-7-22 | 6-12 H | N | N | N | N | S | P | N | 15 | 3 | 5 | 48 | 2 | A | 5 |
| 87 | SRINIVASAN | 48 | M | 30-7-22 | LESS 6 H | N | N | N | N | N | P | N | 15 | 3 | 8 | 39 | 2 | A | 5 |
| 88 | ESWAR | 57 | M | 30-7-22 | 12-24 H | N | N | Y | L | N | P | N | 8 | 5 | 18 | 6 | 21 | A | 11 |
| 89 | SELVAMUTHU KUMAR | 53 | M | 30-7-22 | 6-12 H | N | N | Y | L | N | P | Y | 13 | 4 | 11 | 29 | 4 | A | 7 |
| 90 | RAJU | 70 | M | 30-7-22 | 6-12 H | Y | N | Y | N | S | P | Y | 11 | 5 | 19 | 12 | 10 | D | 5 |
| 91 | VISWANATHAN | 61 | M | 3-8-22 | 6-12 H | Y | N | Y | N | N | P | N | 14 | 3 | 10 | 35 | 11 | A | 9 |
| 92 | RANJITH | 70 | M | 3-8-22 | 6-12 H | Y | N | Y | L | S | R | N | 8 | 5 | 23 | 4 | 18 | D | 6 |
| 93 | BABU | 60 | M | 3-8-22 | 12-24 H | N | N | Y | N | S | P | N | 14 | 5 | 8 | 28 | 5 | A | 9 |
| 94 | VIJAYARANGAN | 55 | M | 3-8-22 | 12-24 H | Y | Y | Y | L | AL | P | N | 10 | 4 | 9 | 31 | 16 | A | 14 |
| 95 | BASKARAN | 59 | M | 3-8-22 | 6-12 H | Y | N | Y | N | N | P | N | 15 | 4 | 9 | 35 | 6 | A | 5 |
| 96 | VASANTHAKUMAR | 46 | M | 3-8-22 | 12-24 H | N | N | Y | N | S,AL | R | N | 8 | 5 | 21 | 8 | 13 | A | 14 |
| 97 | DHATCHAYANI | 65 | F | 4-8-22 | 12-24 H | N | Y | Y | L | N | P | N | 8 | 4 | 13 | 16 | 12 | D | 4 |
| 98 | KAMAL DASS | 68 | M | 5-8-22 | 6-12 H | Y | Y | Y | N | N | P | Y | 13 | 4 | 9 | 23 | 7 | D | 5 |
| 99 | VISWANATHAN | 49 | M | 7-8-22 | 12-24 H | Y | N | Y | CKD | N | P | N | 14 | 5 | 10 | 29 | 21 | A | 8 |
| 100 | TAMARAI | 58 | F | 8-8-22 | 12-24 H | Y | N | Y | L | N | P | N | 8 | 5 | 19 | 4 | 21 | D | 6 |
| 101 | GAJAPATHY | 46 | M | 8-8-22 | 12-24 H | N | N | Y | L | N | P | N | 8 | 5 | 18 | 6 | 20 | D | 5 |
| 102 | SUNDARAM | 69 | M | 8-8-22 | 6-12 H | N | N | Y | L | N | P | Y | 13 | 4 | 11 | 29 | 6 | A | 8 |
| 103 | MADHAN KUMAR | 78 | M | 8-8-22 | 12-24 H | Y | Y | Y | L | S,AL | P | N | 10 | 4 | 9 | 31 | 19 | D | 6 |
| 104 | KALYANA SUNDARAM | 67 | M | 8-8-22 | 6-12 H | Y | Y | Y | N | N | P | Y | 13 | 4 | 9 | 23 | 9 | D | 4 |
| 105 | MOORTHY | 76 | M | 8-8-22 | 6-12 H | N | N | Y | N | N | P | N | 13 | 5 | 13 | 22 | 14 | D | 5 |
| 106 | DEVAPRAKASH | 74 | M | 8-8-22 | 12-24 H | N | N | Y | N | S | R | N | 8 | 5 | 21 | 8 | 16 | D | 5 |
| 107 | SOMASUNDARAM | 47 | M | 12-8-22 | 12-24 H | Y | N | Y | L | N | P | N | 7 | 5 | 12 | 6 | 20 | D | 6 |
| 108 | BALAMURUGAN | 76 | M | 12-8-22 | 6-12 H | Y | N | Y | N | S | P | Y | 11 | 5 | 19 | 12 | 11 | D | 4 |
| 109 | PANNER SELVAM | 58 | M | 12-8-22 | 12-24 H | N | N | Y | N | S | P | N | 15 | 4 | 13 | 29 | 4 | A | 7 |
| 110 | NAGALAKSHMI | 57 | F | 12-8-22 | 12-24 H | Y | N | Y | H | N | P | N | 8 | 5 | 19 | 4 | 21 | D | 6 |
| 111 | SAMSON | 65 | M | 12-8-22 | 6-12 H | Y | N | Y | N | N | P | N | 15 | 4 | 9 | 35 | 8 | A | 5 |
| 112 | BIRJU | 55 | M | 14-8-22 | 6-12 H | Y | N | Y | N | N | P | Y | 3 | 5 | 20 | 2 | 15 | D | 5 |
| 113 | VAITHAIYA LINGAM | 49 | M | 15-8-22 | 6-12 H | Y | N | Y | N | N | P | N | 14 | 3 | 10 | 35 | 10 | A | 5 |
| 114 | BOPITHIYAMMAL | 79 | F | 15-8-22 | 6-12 H | N | N | Y | N | N | P | N | 13 | 4 | 8 | 35 | 11 | A | 9 |
| 115 | BASKAR | 64 | M | 15-8-22 | 6-12 H | Y | N | Y | N | S | R | N | 8 | 5 | 23 | 4 | 16 | D | 5 |
| 116 | RAMESH | 49 | M | 15-8-22 | 12-24 H | Y | N | Y | CKD | N | P | N | 9 | 5 | 14 | 17 | 20 | A | 13 |
| 117 | LAKSHMI | 48 | F | 15-8-22 | LESS 6 H | Y | N | N | N | N | P | N | 15 | 3 | 8 | 39 | 2 | A | 5 |
| 118 | ARUMUGAN | 82 | M | 18-8-22 | 12-24 H | Y | N | Y | AKI | N | P | N | 8 | 5 | 20 | 6 | 37 | D | 2 |
| 119 | DHAN SINGH | 75 | M | 18-8-22 | 12-24 H | Y | N | Y | AKI | N | P | N | 9 | 5 | 14 | 17 | 24 | D | 4 |
| 120 | MURUGANANDHAM | 51 | M | 18-8-22 | 6-12 H | N | N | Y | N | N | P | N | 8 | 5 | 16 | 13 | 14 | A | 12 |
| 121 | BOOPATHI | 75 | M | 18-8-22 | 6-12 H | N | Y | Y | L | N | P | Y | 13 | 4 | 9 | 23 | 8 | D | 4 |
| 122 | SIVALINGAM | 61 | M | 22-8-22 | 12-24 H | Y | N | Y | N | N | P | N | 8 | 5 | 21 | 4 | 15 | D | 5 |
| 123 | THIRUMAI MURUGAN | 79 | M | 23-8-22 | 12-24 H | Y | N | Y | AKI | AL | P | N | 7 | 5 | 17 | 4 | 23 | D | 3 |
| 124 | SANDU RAVINDHRA | 58 | M | 23-8-22 | 6-12 H | N | N | N | N | S | P | N | 15 | 4 | 8 | 37 | 3 | A | 5 |
| 125 | SIVA SETHURAMAN | 82 | M | 23-8-22 | 6-12 H | N | N | N | N | N | P | N | 11 | 5 | 19 | 12 | 11 | D | 5 |
| 126 | RAMASAMY | 83 | M | 23-8-22 | 6-12 H | Y | N | Y | N | S | P | Y | 3 | 5 | 20 | 2 | 18 | D | 4 |
| 127 | ANTHONY RAJ | 54 | M | 23-8-22 | 6-12 H | Y | N | Y | N | N | P | N | 8 | 5 | 20 | 6 | 12 | D | 6 |
| 128 | PONRAJ | 71 | M | 23-8-22 | 12-24 H | Y | N | Y | N | N | P | N | 8 | 5 | 21 | 4 | 17 | D | 4 |
| 129 | BALA KRISHNAN | 53 | M | 25-8-22 | 12-24 H | N | N | Y | N | S,AL | R | N | 8 | 5 | 21 | 8 | 13 | A | 10 |
| 130 | MARIMUTHU | 54 | M | 26-8-22 | 12-24 H | Y | N | Y | N | N | P | N | 15 | 4 | 13 | 29 | 3 | A | 5 |
| 131 | TAMIL VANNAN | 70 | M | 26-8-22 | 12-24 H | Y | N | Y | N | S,AL | P | N | 7 | 5 | 12 | 6 | 23 | D | 4 |
| 132 | NASIR | 49 | M | 26-8-22 | 6-12 H | Y | N | Y | N | S | P | Y | 11 | 5 | 19 | 12 | 7 | D | 6 |
| 133 | MASTHAN BASHA | 55 | M | 29-8-22 | 6-12 H | N | N | Y | N | N | P | N | 13 | 4 | 8 | 35 | 8 | A | 5 |
| 134 | ABDUL GONGI | 54 | M | 29-8-22 | 12-24 H | Y | Y | Y | L | AL | P | N | 10 | 4 | 9 | 31 | 15 | A | 14 |
| 135 | ASWIN KUMAR | 48 | M | 29-8-22 | 12-24 H | N | N | Y | N | S,AL | R | N | 8 | 5 | 21 | 8 | 9 | A | 14 |
| 136 | SHANTHY | 61 | F | 29-8-22 | 6-12 H | Y | N | Y | N | N | P | N | 14 | 3 | 10 | 35 | 11 | A | 7 |
| 137 | KAMALA | 55 | F | 29-8-22 | 6-12 H | N | N | N | N | N | P | N | 15 | 3 | 5 | 48 | 3 | A | 5 |
| 138 | LEELA | 51 | F | 29-8-22 | 6-12 H | N | N | Y | L | N | P | Y | 13 | 4 | 11 | 29 | 4 | A | 5 |
| 139 | JAYARATHINAM | 67 | M | 30-8-22 | 6-12 H | N | N | N | N | S | P | N | 15 | 5 | 12 | 22 | 6 | A | 9 |
| 140 | ROVAMPAL | 59 | F | 30-8-22 | 12-24 H | N | N | Y | L | S,AL | P | N | 8 | 4 | 14 | 22 | 20 | D | 6 |
| 141 | RANI | 78 | F | 30-8-22 | 6-12 H | Y | Y | N | L | N | P | Y | 13 | 4 | 9 | 23 | 8 | D | 3 |
| 142 | GANESH | 74 | M | 30-8-22 | 6-12 H | N | N | Y | N | N | P | N | 8 | 5 | 16 | 13 | 17 | D | 6 |
| 143 | NIRMALA | 56 | F | 30-8-22 | 12-24 H | Y | N | Y | CKD | N | P | N | 7 | 5 | 17 | 8 | 25 | D | 6 |
| 144 | LATHA | 52 | F | 30-8-22 | 12-24 H | N | N | Y | N | N | P | N | 15 | 4 | 13 | 29 | 4 | A | 9 |
| 145 | CHINNADURAI | 55 | M | 1-9-22 | 6-12 H | Y | N | Y | N | S,AL | R | N | 8 | 5 | 23 | 4 | 16 | A | 8 |
| 146 | VIGNESH | 76 | M | 1-9-22 | 12-24 H | Y | N | Y | L | S,AL | P | N | 8 | 5 | 19 | 4 | 24 | D | 3 |
| 147 | MANIKKAM | 78 | M | 1-9-22 | 6-12 H | Y | N | Y | N | N | P | N | 9 | 3 | 13 | 30 | 15 | A | 13 |
| 148 | PARTHASARTHY | 48 | M | 1-9-22 | 6-12 H | N | N | Y | N | N | P | N | 15 | 3 | 5 | 48 | 2 | D | 5 |
| 149 | SETHUMADHAVAN | 51 | M | 1-9-22 | LESS 6 H | N | N | N | L | N | P | N | 15 | 4 | 9 | 35 | 2 | A | 5 |
| 150 | AHAMEDULLAH | 57 | M | 2-9-22 | 12-24 H | N | N | Y | L | N | P | N | 3 | 5 | 20 | 4 | 25 | D | 2 |
| 151 | DHINAKKARAN | 49 | M | 5-9-22 | 6-12 H | N | N | Y | N | N | P | N | 8 | 5 | 16 | 13 | 14 | A | 16 |
| 152 | ARUN KUMAR | 74 | M | 5-9-22 | 12-24 H | N | N | Y | L | N | P | N | 8 | 5 | 18 | 6 | 23 | D | 4 |
| 153 | KALIYAN | 56 | M | 5-9-22 | 12-24 H | Y | Y | Y | L | S | P | N | 10 | 4 | 9 | 31 | 14 | A | 9 |
| 154 | SURESH | 67 | M | 5-9-22 | 6-12 H | Y | N | Y | N | N | P | N | 15 | 4 | 9 | 35 | 8 | A | 5 |
| 155 | DHANASEKAR | 61 | M | 5-9-22 | 6-12 H | N | N | Y | N | N | P | N | 13 | 5 | 13 | 22 | 11 | A | 12 |
| 156 | BALAMURUGAN | 67 | M | 10-9-22 | 12-24 H | N | N | Y | N | S | R | N | 8 | 5 | 21 | 8 | 12 | A | 13 |
| 157 | MURALI | 77 | M | 12-9-22 | LESS 6 H | Y | N | N | L | S,AL | P | N | 14 | 5 | 9 | 24 | 10 | A | 14 |

| | | | | | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 158 | VAITHAIYALINGAM | 48 | M | 13-9-22 | 6-12 H | N | N | Y | L | N | P | Y | 13 | 4 | 11 | 29 | 2 | A | 7 |
| 159 | MANOHAR | 57 | M | 14-9-22 | 6-12 H | Y | N | Y | N | N | P | N | 14 | 3 | 10 | 35 | 11 | A | 5 |
| 160 | RENUKA | 52 | F | 15-9-22 | 12-24 H | N | N | Y | CKD | N | P | N | 9 | 5 | 14 | 17 | 20 | A | 15 |
| 161 | NAGALAKSHMI | 64 | F | 16-9-22 | 12-24 H | Y | N | Y | CA | N | P | N | 7 | 5 | 12 | 6 | 21 | D | 6 |
| 162 | THULASI | 52 | F | 21-9-22 | 6-12 H | Y | N | Y | N | N | R | N | 8 | 5 | 23 | 4 | 15 | D | 6 |
| 163 | GOPALAKRISHNAN | 54 | M | 21-9-22 | 6-12 H | Y | N | Y | N | S | P | Y | 11 | 5 | 19 | 12 | 9 | D | 6 |
| 164 | PRASHANTH | 84 | M | 21-9-22 | 6-12 H | Y | Y | Y | AKI | N | P | Y | 13 | 4 | 9 | 23 | 42 | D | 2 |
| 165 | PRABHUKUMAR | 51 | M | 21-9-22 | 12-24 H | Y | N | Y | N | N | P | N | 8 | 5 | 21 | 4 | 14 | A | 11 |
| 166 | ANTHONY SAMY | 56 | M | 22-9-22 | 12-24 H | N | N | Y | N | S | P | N | 9 | 4 | 12 | 18 | 14 | A | 13 |
| 167 | BALAKRISHNAN | 82 | M | 23-9-22 | 6-12 H | Y | N | Y | N | N | P | Y | 3 | 5 | 20 | 2 | 18 | D | 2 |
| 168 | SHANMUGAPRIYA | 47 | F | 23-9-22 | 12-24 H | Y | N | Y | L | N | P | N | 8 | 5 | 19 | 4 | 20 | D | 6 |
| 169 | AMARAVATHY | 58 | F | 23-9-22 | 6-12 H | Y | Y | Y | L | N | P | Y | 13 | 4 | 9 | 23 | 5 | D | 5 |
| 170 | SAMUNDESHWARI | 72 | F | 23-9-22 | 12-24 H | Y | N | Y | N | N | P | N | 7 | 5 | 18 | 6 | 42 | D | 4 |
| 171 | MARY RANI | 49 | F | 23-9-22 | 12-24 H | Y | N | Y | N | N | P | N | 7 | 5 | 18 | 6 | 35 | D | 5 |
| 172 | SAMPATH | 51 | M | 25-9-22 | 6-12 H | N | N | N | N | S | P | N | 14 | 5 | 8 | 28 | 4 | A | 5 |
| 173 | SUJAN | 64 | M | 29-9-22 | 6-12 H | Y | N | Y | N | N | P | N | 8 | 5 | 20 | 6 | 13 | D | 6 |
| 174 | MYNA | 56 | F | 29-9-22 | 6-12 H | N | N | N | N | N | P | N | 15 | 3 | 5 | 48 | 3 | A | 5 |
| 175 | RAJAKUMARI | 56 | F | 29-9-22 | 6-12 H | N | N | Y | N | N | P | N | 13 | 4 | 8 | 35 | 8 | A | 5 |
| 176 | MANILA | 51 | F | 29-9-22 | 12-24 H | Y | N | Y | N | N | P | N | 8 | 5 | 21 | 4 | 14 | D | 4 |
| 177 | KOTAIYAMMAL | 50 | F | 30-9-22 | 12-24 H | Y | N | Y | N | AL | P | N | 7 | 5 | 12 | 6 | 20 | A | 10 |
| 178 | SUGANTHI | 56 | F | 30-9-22 | 12-24 H | Y | N | Y | AKI | N | P | N | 7 | 5 | 18 | 6 | 36 | D | 5 |
| 179 | KAMATCHI | 61 | F | 30-9-22 | 6-12 H | N | N | Y | H | N | P | N | 13 | 4 | 8 | 35 | 8 | A | 7 |
| 180 | SELVAM | 72 | M | 1-10-22 | 6-12 H | Y | N | Y | N | N | P | N | 8 | 5 | 20 | 6 | 15 | D | 4 |
| 181 | VENUGOPAL | 64 | M | 1-10-22 | 12-24 H | N | N | Y | N | N | P | N | 14 | 5 | 8 | 28 | 5 | A | 9 |
| 182 | DASARADHAN | 54 | M | 1-10-22 | 6-12 H | Y | Y | Y | L | S,AL | P | Y | 13 | 4 | 9 | 23 | 4 | D | 6 |
| 183 | RUDHRA KUMAR | 68 | M | 1-10-22 | 6-12 H | N | N | N | N | AL | P | N | 15 | 3 | 5 | 48 | 5 | A | 5 |
| 184 | MUNIYAMMAL | 55 | F | 1-10-22 | 12-24 H | Y | Y | Y | L | N | P | N | 10 | 4 | 9 | 31 | 14 | A | 7 |
| 185 | SAROJA | 58 | F | 1-10-22 | 6-12 H | Y | N | Y | N | N | P | N | 12 | 3 | 9 | 37 | 7 | A | 5 |
| 186 | RANI | 49 | F | 2-10-22 | 12-24 H | N | N | Y | N | N | R | N | 8 | 5 | 21 | 8 | 13 | A | 7 |
| 187 | JAYALAKSMI | 47 | F | 2-10-22 | 6-12 H | Y | N | Y | L | N | P | Y | 3 | 5 | 20 | 2 | 14 | D | 5 |
| 188 | CHANDRA | 59 | F | 2-10-22 | 6-12 H | Y | N | Y | N | N | P | N | 14 | 3 | 10 | 35 | 11 | A | 5 |
| 189 | AMBIKA | 55 | F | 4-10-22 | 6-12 H | Y | Y | Y | L | N | P | Y | 13 | 4 | 9 | 23 | 7 | D | 6 |
| 190 | MOHAN | 72 | M | 4-10-22 | 6-12 H | N | N | Y | N | N | P | N | 8 | 5 | 16 | 13 | 17 | D | 5 |
| 191 | MAHALAKSHMI | 65 | F | 4-10-22 | 6-12 H | N | Y | Y | N | N | P | Y | 11 | 5 | 19 | 12 | 10 | D | 5 |
| 192 | RAJESH | 52 | M | 4-10-22 | 12-24 H | N | N | Y | CKD | S | P | N | 15 | 4 | 13 | 29 | 4 | A | 5 |
| 193 | EGAMBARAM | 65 | M | 4-10-22 | 6-12 H | N | N | Y | L | S,AL | P | Y | 13 | 4 | 11 | 29 | 4 | A | 7 |
| 194 | KANTHAMMAL | 57 | F | 6-10-22 | LESS 6 H | N | N | Y | N | N | P | N | 15 | 5 | 8 | 28 | 5 | A | 5 |
| 195 | MUTHAIYA | 56 | M | 7-10-22 | 6-12 H | N | N | Y | N | N | P | N | 13 | 5 | 13 | 22 | 11 | A | 9 |
| 196 | MUTHU | 49 | M | 8-10-22 | 12-24 H | Y | N | Y | L | S,AL | P | N | 8 | 5 | 19 | 4 | 19 | D | 6 |
| 197 | KAMALANATHAN | 60 | M | 9-10-22 | 12-24 H | Y | N | Y | CKD | N | P | N | 13 | 4 | 9 | 23 | 14 | D | 6 |
| 198 | VIJAYAN | 63 | M | 10-10-22 | 12-24 H | N | N | Y | L | N | P | N | 8 | 5 | 18 | 6 | 21 | D | 6 |
| 199 | ELANGO | 48 | M | 10-10-22 | 6-12 H | N | N | N | N | S | P | N | 14 | 5 | 8 | 28 | 4 | A | 5 |
| 200 | PALANI | 71 | M | 10-10-22 | 6-12 H | Y | N | Y | N | S | R | N | 8 | 5 | 23 | 4 | 18 | D | 5 |

## KEY TO MASTER CHART

M - MALE
F - FEMALE
Y - YES
N - NO
L - DYSLIPIDEMIA
AKI - ACUTE KIDNEY INJURY
CKD - CHRONIC KIDNEY DISEASE
CA - CARCINOMA BREAST
H - HYPOTHYROIDISM
S - SMOKING
AL - ALCOHOLISM
P - PRIMARY
R - RECURRENT
A - ALIVE
D - DEATH