# A PROSPECTIVE STUDY OF ROLE OF MEAN PLATELET VOLUME, PLATELET DISTRIBUTION WIDTH AS PROGNOSTIC MARKERS IN ACUTE ISCHEMIC STROKE PATIENTS

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(Reg. No: EC/NEW/INST/2020/1058)



## CERTIFICATE

No: 844/2021

**TITLE OF THE STUDY:**

**A prospective study of role of mean platelet volume, platelet distribution width as prognostic marker in acute ischemic stroke patients**

**PRINCIPAL INVESTIGATOR:**

**Dr. K. Ramkumar, Postgraduate in Department of General Medicine, Thanjavur Medical College.**

This to certify that the protocol submitted by the principal investigator of the above mentioned study has been reviewed as per standard ethical guidelines and the same has been **APPROVED** by the members of the Institutional ethical committee at its meeting held on **21.01.2021**.

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# LIST OF ABBREVATIONS:

CT - Computed Tomography

DM -Diabetes Mellitus

SHTN -Systemic hypertension

ED - Emergency Department

EMR - Electronic Medical Records

MRI -Magnetic Resonance Imaging

MPV-Mean Platelet Volume

PDW- Platelet Distribution Width

FBS – Fasting Blood Sugar

PPBS – Post prandial blood sugar

HbA1c – Glycosylated Hemoglobin

MRS - Modified Rankin Scale

BMI - Body Mass Index

# LIST of FIGURES

***Figure 1. Classification of Stroke:*** *.....33*

***Figure 2. Pathophysiology of Stroke****:* *.....36*

***Figure 3: Molecular mechanisms of stroke*****.....*** ***Error! Bookmark not defined.***

***Figure 4. Diagnosis of stroke ischaemic stroke to identify arterial and cardiac causes:*** *37*

***Figure 5. CT - Left middle cerebral artery (MCA) infarction:*** *.....38*

***Figure 6. AHA ACLS adult suspected stroke algorithm:*****.....** *39*

***Figure 7: Stroke therapy*** *.....41*

***Figure 8. Mechanism of Ischaemic stroke:*** *.....42*

***Figure 9. Cellular effects of ischemia:*** *.....43*

***Figure 10. Cerebral vasculature****:* *.....44*

***Figure 11. Pathophysiology of ischaemic Stroke:*****.....** *46*

***Figure 12: Histogram showing PDW*** *.....55*

***Figure 13.Age (years)*** *.....71*

***Figure 14.Age group*****.....** *72*

***Figure 15.Gender*****.....** *73*

***Figure 16.Smoker*** *.....74*

***Figure 17.Alcoholic*** *.....75*

***Figure 18. Anthropometry*****.....** *76*

***Figure 19.BMI (kg/m2)*** *.....77*

***Figure 20.BMI class*** *.....78*

*Figure 21.Hypertension*.....79

*Figure 22. Blood Pressure*.....**Error! Bookmark not defined.**

*Figure 23.Diabetes Mellitus*.....81

*Figure 24. Blood Sugar*.....82

*Figure 25.HbA1C*.....83

*Figure 26.Dyslipidemia*.....84

*Figure 27.Mean Platelet Volume (fl)*.....85

*Figure 28.Platelet Distribution Width*.....86

*Figure 29.MRS score day1*.....87

*Figure 30.MRS score day 7*.....88

*Figure 31. Mean Platelet Volume (fl) with MRS score day 1*.....89

*Figure 32.Mean Platelet Volume (fl) with MRS score day 7*.....90

*Figure 33.Platelet Distribution Width with MRS score day1*.....91

*Figure 34.Platelet Distribution Width with MRS score day 7*.....92

*Figure 35. Risk factors with MRS score day 1*.....94

*Figure 36. Risk factors with Mean Platelet Volume*.....97

*Figure 37. Risk factors with Platelet Distribution Width*.....99

# LIST OF TABLES:

*Table 1 Risk factors of Stroke:* ..... *34*

*Table 2. Types of ischaemic stroke based on the TOAST classification:* ..... *45*

*Table 3. Selection criteria for mechanical thrombectomy in acute ischemic stroke:* ..... *49*

*Table 4: Modified Rankin Scale ( MRS)*[104] ..... *66*

*Table 5.Age (years)* ..... *71*

*Table 6.Age group* ..... *72*

*Table 7.Gender* ..... *73*

*Table 8.Smoker* ..... *74*

*Table 9.Alcoholic* ..... *75*

*Table 10. Anthropometry* ..... *76*

*Table 11.BMI (kg/m2)* ..... *77*

*Table 12.BMI class* ..... *78*

*Table 13.Hypertension* ..... *79*

*Table 14. Blood Pressure* ..... *80*

*Table 15.Diabetes Mellitus* ..... *81*

*Table 16. Blood Sugar* ..... *82*

*Table 17.HbA1C* ..... *83*

*Table 18.Dyslipidemia* ..... *84*

*Table 19.Mean Platelet Volume (fl)* ..... *85*

*Table 20.Platelet Distribution Width* ..... *86*

***Table 21.MRS score day1*** ..... *87*

***Table 22.MRS score day 7*** ..... *88*

***Table 23. Mean Platelet Volume (fl) with MRS score day 1*** ..... *89*

***Table 24.Mean Platelet Volume (fl) with MRS score day 7*** ..... *90*

***Table 25.Platelet Distribution Width with MRS score day1*** ..... *91*

***Table 26.Platelet Distribution Width with MRS score day 7*** ..... *92*

***Table 27. Risk factors with MRS score day 1*** ..... *93*

***Table 28. Risk factors with Mean Platelet Volume*** ..... *95*

***Table 29. Risk factors with Platelet Distribution Width*** ..... *98*

# TABLE OF CONTENTS


**1 INTRODUCTION ..... 24**

**2 AIM AND OBJECTIVES ..... 26**

2.1 AIM: ..... 26

2.2 OBJECTIVES: ..... 26

**3 REVIEW OF LITERATURE ..... 27**

**4 RESEARCH QUESTION OR HYPOTHESIS ..... 61**

4.1 Research Question: ..... 61

4.2 Null Hypothesis: ..... 61

4.3 Alternate Hypothesis: ..... 61

**5 METHODOLOGY ..... 63**

5.1 Study Subjects: ..... 63

5.2 Study Design: ..... 63

5.3 Study setting: ..... 63

5.4 Sampling Procedure: ..... 63

5.5 Inclusion Criteria: ..... 64

5.6 Exclusion criteria: ..... 64

5.7 Sample Size: ..... 64

5.8 Study procedure: ..... 65

5.9 Ethical Consideration: ..... 67

5.10 Study Period: ..... 67

5.11 Statistical Methods: ..... 67

**6 RESULTS ..... 69**

**7 DISCUSSION ..... 100**

**8 LIMITATIONS ..... 105**

**9 RECOMMENDATIONS ..... 106**

**10 CONCLUSION ..... 107**

**11 SUMMARY OF RESULTS ..... 108**

**12 REFERENCES ..... 111**

**13 ANNEXURES ..... 120**

# 1 ABSTRACT


## Introduction:

The sudden onset of a neurological deficit that can be linked to a specific vascular cause is what defines a stroke. The burden of stroke is predicted to automatically rise in the approaching years due to the increase in the population's elderly people. Physiological parameters of haemostatic significance is mean platelet volume (MPV) and Platelet Distribution Width (PDW) , a measure of platelet function. Platelet count is not as crucial to haemostasis as changes in MPV and PDW.

## Objectives:

To establish the correlation between mean platelet volume and platelet distribution width with severity of acute ischemic stroke and to assess the usefulness of MPV and PDW in predicting prognosis in acute ischemic stroke.

## Methodology:

All Patients admitted with Acute Ischaemic Stroke in inpatient wards , Department of General Medicine, Thanjavur Medical College were consecutively selected till the sample size of 75 was reached. After obtaining Ethical committee clearance and obtaining informed written consent, the following investigations like complete hemogram, Diabetic profile, Blood pressure, Mean Platelet Volume, Platelet Distribution Width were to be done for the purpose of the study and results were to be done. MRS scale was used to

assess severity of stroke. All data were entered into Microsoft excel and analyse using SPSS version 16.

## Results:

The mean Age (years) among the subjects was 60.52 (± 7.33) ranging from 43 to 75 years. Among the subjects, 33 (44%) were in 61 - 70 years, 30 (40%) were in 51 - 60 years and 8 (10.67%) were in < 50 years. Among the subjects, 47 (62.67%) were Males and 28 (37.33%) were Females. Among the subjects, 29 (38.67%) were Smoker, 33 (44%) were Alcoholic. Among the subjects, 60 (80%) had Hypertension. Among the subjects, 32 (42.67%) had Diabetes Mellitus. The mean Mean Platelet Volume (fl) among the subjects was 10.28 (± 0.99) fl ranging from 8.7 to 12.4 fl. The mean Platelet Distribution Width among the subjects was 14.91 (± 1.23) ranging from 9 to 18.1%. Among the subjects, 40 (53.33%) had MRS Score 3 - 6 and 35 (46.67%) had MRS Score 0 – 2 on day 1. Among the subjects, 46 (61.33%) had MRS Score 3 - 6 and 29 (38.67%) had MRS Score 0 – 2 on day 7.

The mean Mean Platelet Volume (fl) with MRS score day 1showed that among Score 0 - 2 MPV was 9.83 (± 0.72) which is lower by 0.84 and statistically significant compared to MPV 10.67 (± 1.04) in Score 3 – 6. The mean Mean Platelet Volume (fl) with MRS score at day 7, among Score 0 - 2 MPV was 9.52 (± 0.47) which is lower by 1.23 and statistically significant compared to MPV 10.75 (± 0.94) in Score 3 – 6.

The mean Platelet Distribution Width with MRS score day 1, among Score 0 – 2 PDW was 14.81 (± 1.11) which is lower by 0.2 but not statistically significant compared to PDW 15 (± 1.34) in Score 3 – 6. The mean Platelet Distribution Width with MRS score day 7,

among Score 0 - 2 PDW was 14.47 (± 0.91) which is lower by 0.71 and statistically significant compared to PDW 15.19 (± 1.33) in Score 3 – 6.

92.3% of the obese subjects, and 83.3% of the subjects with dyslipidemia had MRS score 3 – 6 with higher severity of stroke and the differences were statistically significant. The Mean Platelet Volume among those with Hypertension, Diabetes and Dyslipidemia were significantly higher compared to those without the risk factors. Subjects with more than 60 years of age and obese subjects had significantly higher mean platelet volume and the differences were statistically significant. The Platelet Distribution Width among those with higher age group and Dyslipidemia were significantly higher compared to those without the risk factors.

## Conclusion:

Independent predictors of the risk of stroke, MPV and PDW are elevated in acute ischemic stroke. Higher MPV and PDW are linked to more severe stroke and have a worse prognosis. A quick and uncomplicated test called the mean platelet volume and platelet distribution width, which measures platelet reactivity, can be used to predict the severity and course of an acute ischemic stroke in patients.

## Keywords:

Acute Ischaemic Stroke, Mean Platelet Volume , Platelet Distribution Width

# 2 INTRODUCTION

Stroke is defined as having clinical symptoms of focal or generalised abnormalities of brain function that last for 24 hours or longer or result in death and have no other evident origin besides vascular. [1]The second-leading cause of death and the main contributor to disability worldwide is stroke. Because of the ageing population, there are more new stroke cases. Furthermore, stroke affects young people in low- and middle-income nations. Either an ischemic or hemorrhagic cause can result in a stroke. Stroke is consistently linked to high mortality and morbidity rates. The rise in stroke mortality is attributed to a variety of variables. When compared to hemorrhagic stroke, ischemic stroke is more prevalent and causes greater mortality and DALYs (disability-adjusted life-years lost).[2]

Stroke is a significant global health issue. It was the second-leading cause of mortality in the world in 2011, accounting for 6.2 million fatalities.[3]Additionally, cerebrovascular illness, which is the number one cause of adult disability, forces millions of stroke survivors to adjust to a lifestyle that includes limitations on daily activities. The mortality of stroke patients is impacted by a number of factors.

The severity of the stroke and the patient's age are the reliable indicators of outcome during the acute phase of the disease. The degree of neurological damage (e.g., altered mentation, language, behaviour, visual field deficit, motor deficit) and the size and position of the infarction on neuroimaging techniques like MRI or CT scans can be used to determine the severity of a stroke.Other important influences on stroke outcome include the ischemic stroke mechanism, epidemiologic variables, coexisting diseases, and stroke complications. [4]

The most crucial time for effective intervention is in the early hours following an acute stroke event. In order to address the hemodynamic parameters for improved outcomes, it is important to identify the prognostic factors as early as feasible.[5]For the clinician to provide a reasonable prognosis for each patient, to offer a practical management strategy, and to aid patients and their families in understanding the course of the disease, knowledge of prognostic indicators is essential.[6]

One of the most common causes of morbidity and mortality is cerebral vascular disease. When atherosclerosis problems affect a vessel's blood flow, an ischemic stroke can result, which eventually causes thrombus development. By forming intravascular thrombus following the erosion or rupture of atherosclerotic plaques, platelets play a crucial role in the pathogenesis of ischemic stroke. In order to effectively manage and treat stroke patients, it is essential to continuously monitor haemostatic parameters like platelet components due to the presence of blood clots in ischemic stroke. By forming intravascular thrombus following the erosion or rupture of atherosclerotic plaques, platelets play a crucial role in the pathogenesis of ischemic stroke. In order to effectively manage and treat stroke patients, it is essential to continuously monitor haemostatic parameters like platelet components due to the presence of blood clots in ischemic stroke.

# 3 AIM AND OBJECTIVES

## 3.1 **AIM:**

To assess the worth of Mean Platelet Volume (MPV) and Platelet Distribution Width (PDW) as a prognostic factor among patients with acute ischaemic stroke admitted in the inpatient wards , Department of General Medicine, Thanjavur Medical College

## 3.2 **OBJECTIVES:**

1.To determine the value of mean platelet volume and platelet distribution width

2. .To Investigate any possible effects of mean platelet volume (MPV) and platelet distribution width on short-term stroke prognosis

3. To establish the correlation between mean platelet volume and platelet distribution width with severity of acute ischemic stroke.

4.To assess the usefulness of MPV and PDW in predicting prognosis in acute ischemic stroke.

5. To correlate the value of mean platelet volume, platelet distribution width with other risk factors for acute ischemic stroke.

# 4 REVIEW OF LITERATURE

Review of Literature of this study is discussed under the following heads:

1. **Stroke**
    a. **Definition**
    b. **Epidemiology**
    c. **Types**
    d. **Pathophysiology**
    e. **Management**
2. **Ischemic Stroke**
    a. **Classification**
    b. **Pathophysiology**
    c. **Management**
3. **Platelet indices in stroke**
4. **Reviews of articles depicting the usefulness of Mean Platelet Volume (MPV) and Platelet Distribution Width (PDW) as a prognostic factor among patients with acute ischaemic stroke**

# STROKE

**Definition:**

"Rapidly developing symptoms and/or signs of localised and global loss of brain function lasting for at least 24 hours with no evident cause other than of vascular origin," is how stroke is defined.. [7]

**Burden:**

Globally, cerebrovascular accidents (stroke) are the second prominent cause of death and the third important cause of disability. [8]Stroke causes dementia and depression by causing the abrupt death of some brain cells owing to a lack of oxygen when the blood supply to the brain is cut off by a blocked or torn artery.[9]Around the world, low- and middle-income nations account for 70% of strokes and 87% of stroke-related fatalities and disability-adjusted life years. [10,11]The prevalence of ischemic infarctions, which account for about 87% of strokes, grew significantly between 1990 and 2016, which is attributed to lower mortality and better clinical interventions. The majority of strokes are caused by primary (first-time) haemorrhages, with secondary (second-time) haemorrhages accounting for between 10% and 25%.[12,13]

**Burden of Stroke in India**

Developing countries like India are experiencing a dual burden of communicable and non-communicable diseases as a result of this epidemiological change. In rural areas, the estimated prevalence rate of stroke is between 84 and 262/100,000, while in urban areas, it is between 334 and 424/100,000. According to recent population-based studies, the incidence (new cases) rate ranges from 119 to 145/100,000.[14]In India, there were 2.21

times as many brain infarcts as haemorrhages. In India, stroke deaths made up 1.2% of all fatalities. [15]

The term "stroke" was first used in 1599 to describe the abrupt onset of symptoms as a "stroke of God's hand." Physicians continued to use the term "apoplexy," a diagnosis that had been in use since the time of the Hippocratic literature, because it had not been incorporated into the medical lexicon of the day. Although the word "stroke" is related to the Greek word "apoplexia," which means to be struck by a fatal blow, it would be incorrect to make a direct comparison between our contemporary understanding of stroke and what has traditionally been known as apoplexy.[16]

**The World Health Organization's definition:**

A stroke is described by the World Health Organization as "rapidly established clinical symptoms of focal (or global) interruption of brain function, lasting more than 24 hours or resulting in death, with no clear cause other than of vascular origin" in 1970.[17]Despite its continued popularity, the World Health Organization definition places a strong emphasis on clinical symptoms. Due to significant developments in the "nature, chronology, clinical gratitude of stroke and its imitators, and imaging results that demand a new definition," it is currently regarded as out-of-date by the American Heart Association and American Stroke Association.[18]The American Heart Association and American Stroke Association revised their accepted definition of stroke in 2013 to include silent infarctions, including those of the cerebral, spinal, and retina. The American Heart Association/American Stroke Association continues to include "silent" pathology in their "conventional" clinical diagnosis of stroke, but this is a considerable buildup. To move toward a radiographic

demonstration (tissue-based definition) of infarction or haemorrhage was the justification for this change.

**Definition of Stroke:**

The term “stroke” should be generally 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 indication of cerebral, spinal cord, or retinal focal ischemic injury in a definite vascular distribution; or

2. Clinical indication of cerebral, spinal cord, or retinal focal ischemic injury based on symptoms continuing ≥24 hours or until death, and other aetiologies excepted. (Note: CNS infarction comprises haemorrhagic infarctions, types I and II; see “Haemorrhagic Infarction.”)

**Definition of ischemic stroke:**

“An episode of neurological dysfunction clinically defined by focal cerebral, spinal, or retinal infarction.” (Note: Evidence of CNS infarction is defined above.)

**Definition of silent CNS infarction:**

“Imaging or neuropathological indication of CNS infarction, without a history of acute neurological dysfunction attributable to the lesion.”

**Definition of intracerebral haemorrhage:**

“A focal collection of blood within the brain parenchyma or ventricular system. And, that is not caused by trauma.” (Note: Intracerebral haemorrhage comprises parenchymal haemorrhages after CNS infarction, types I and II—see “Hemorrhagic Infarction.”)

**Definition of stroke produced by intracerebral haemorrhage:**

“Rapidly developing clinical signs of neurological dysfunction attributable due to a focal collection of blood within the parenchyma of the brain or ventricular system. And that is not caused by trauma.”

**Definition of silent cerebral haemorrhage:**

“A focal collection of chronic blood products within the parenchyma of thebrain, subarachnoid space, or ventricular system on neuroimaging or neuropathological examination And that is not caused by trauma and without a history of acute neurological dysfunction attributable to the lesion.”

**Definition of subarachnoid haemorrhage:**

“Bleeding into the subarachnoid space (the space lying between the arachnoid membrane and the pia mater of the brain or spinal cord).”

**Definition of stroke caused by subarachnoid haemorrhage:**

“Rapidly emerging signs of neurological dysfunction and/or headache because of bleeding into the subarachnoid space (the space between the arachnoid membrane and the pia mater of the brain or spinal cord), which is not produced by trauma.”

**Definition of stroke caused by cerebral venous thrombosis:**

"Infarction or haemorrhage in the brain, spinal cord, or retina because of thrombosis of a cerebral venous structure. Symptoms or signs caused by reversible oedema without infarction or haemorrhage do not be eligible to be defined as stroke."

**Definition of stroke, not otherwise specified:**

An occurrence of acute neurological dysfunction supposed to be produced by ischemia or haemorrhage, continuing ≥24 hours or until death, but without adequate indication to be classified as one of the above.[19]

**Types:**

Generally, stroke is classified into two major types, such as,

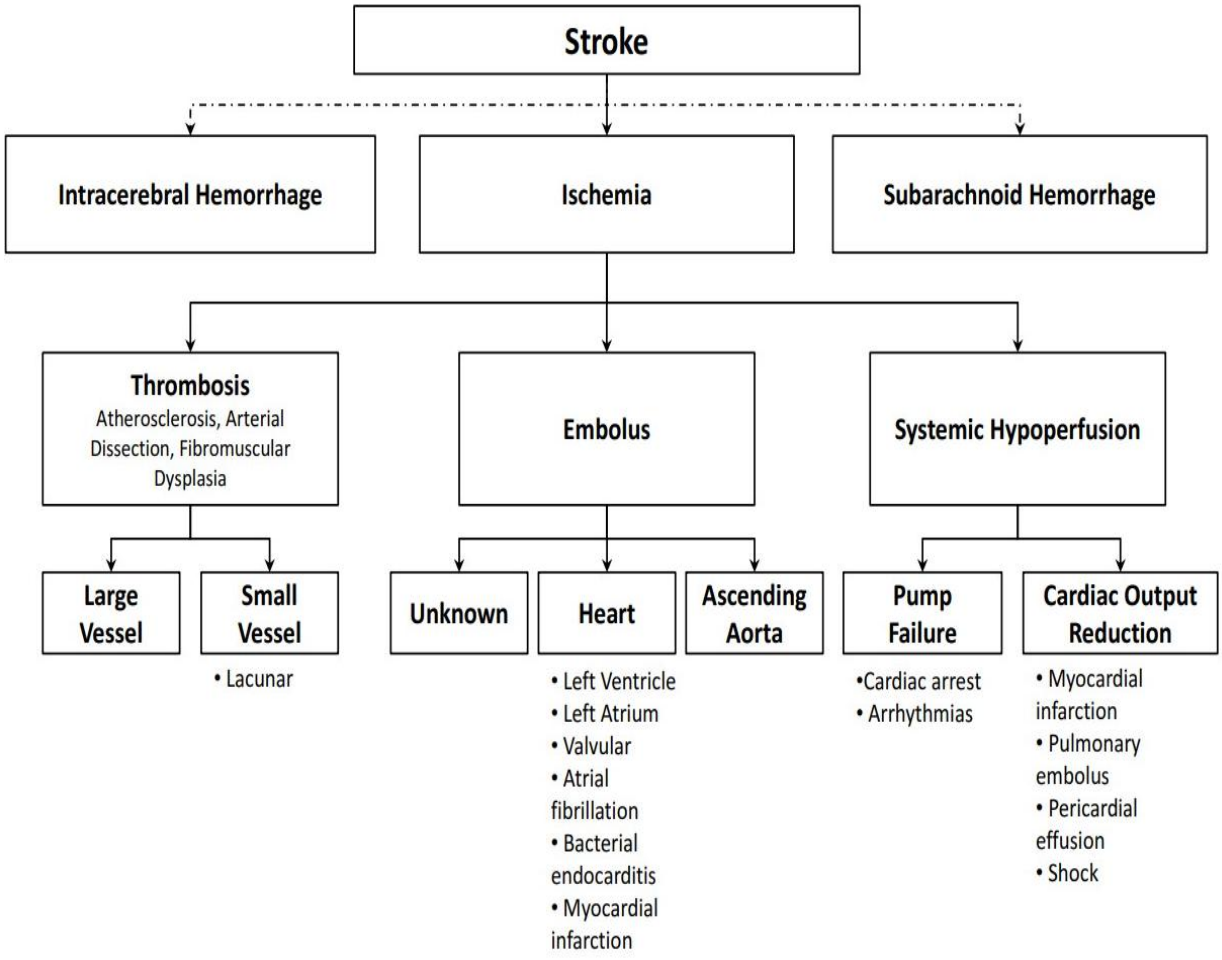
I. Ischaemic stroke (interruption of the blood supply to a part of the brain which results in sudden loss of function) (80% of stroke)
II. Haemorrhagic stroke(as a result of rupture of a blood vessel or an abnormal vascular structure) (20% of stroke). [20]
III. Cerebral Venous Thrombosis
IV. Spinal Cord Shock

80% of haemorrhagic strokes are caused by intracerebral and subarachnoid haemorrhages. Subarachnoid haemorrhage is primarily caused by saccular aneurysms, it is also linked to arteriovenous malformation, intracranial neoplasm, and some medications like anticoagulants. Intracerebral haemorrhage is caused by uncontrolled hypertension that

causes small vessels to burst.[21,22] The following image represents the classification of stroke, [23]

Stroke is a neurological disorder characterized by blockage of blood vessels. Clots form in the brain and interrupt blood flow, clogging arteries and causing blood vessels to break, leading to bleeding. Rupture of the arteries leading to the brain during stroke results in the sudden death of brain cells owing to a lack of oxygen. Stroke can also lead to depression and dementia.[24]

**Figure 1. Classification of Stroke:**



**RISK FACTORS :**

The following table represents the modifiable and non-modifiable risk factors of stroke, [26]

***Table 1 Risk factors of Stroke:***

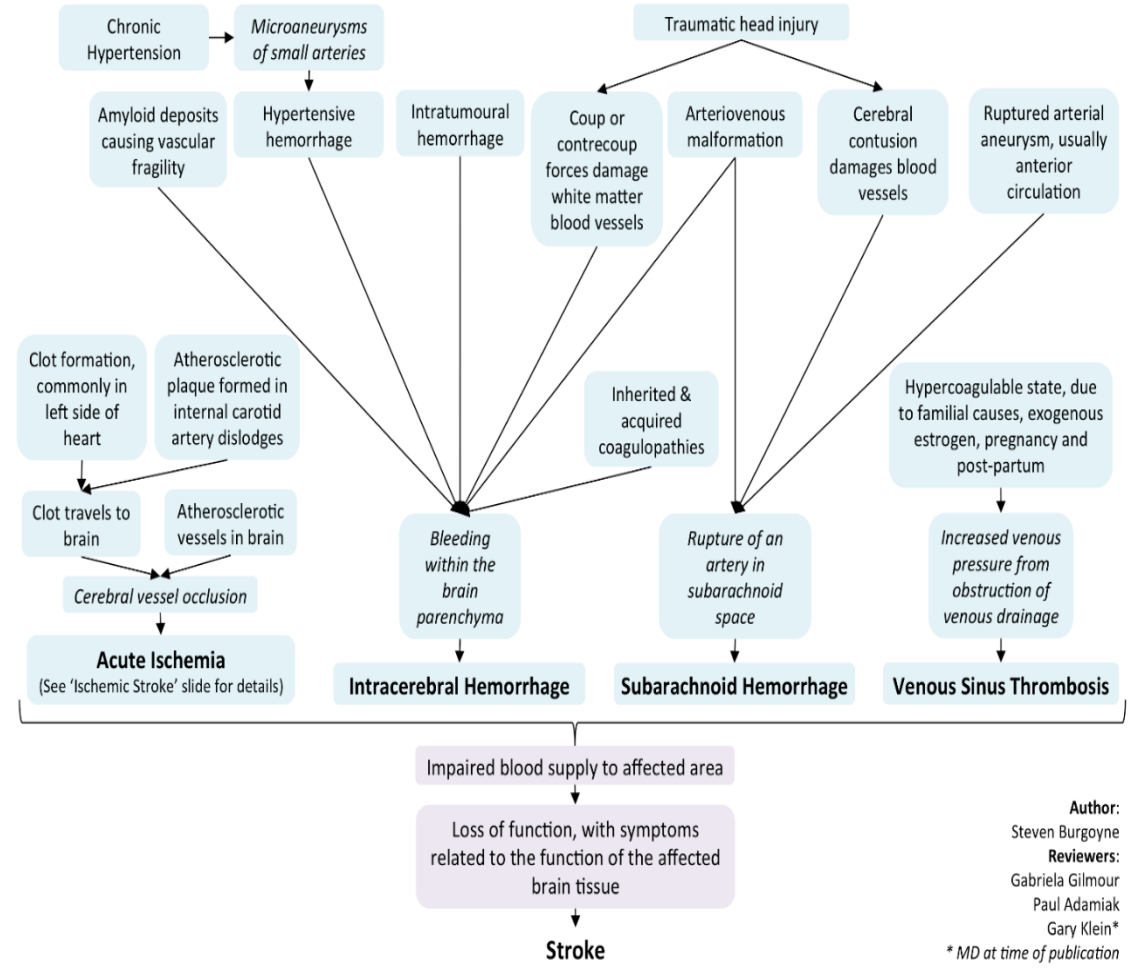
| Nonmodifiable risk factors | Modifiable risk factors |
|---|---|
| Age | Hypertension |
| Sex | Diabetes |
| Low birth weight | Dyslipidemia |
| Race/ethnicity | Obesity |
| Family history (including intracranial aneurysms) | Metabolic syndrome |
| | Diet and nutrition |
| Genetic predisposition (including Fabry disease, sickle cell disease, CADASIL, coagulopathies) | Cigarette smoking |
| | Alcohol consumption |
| | Physical inactivity |
| | Obstructive sleep apnea |
| | Large artery atherosclerosis: |
| | Extracranial carotid disease |
| | Extracranial vertebrobasilar disease |
| | Intracranial atherosclerosis |
| | Arterial fibrillation |
| | Aortic atherosclerosis |
| | Patent foramen ovale |
| | Prosthetic heart valves |
| | Valvular heart disease |
| | Cardiomyopathy |
| | Acute myocardial infarction |
| | Hypercoagulability |
| | Hyperhomocysteinemia |
| | Antiphospholipid antibody syndrome |

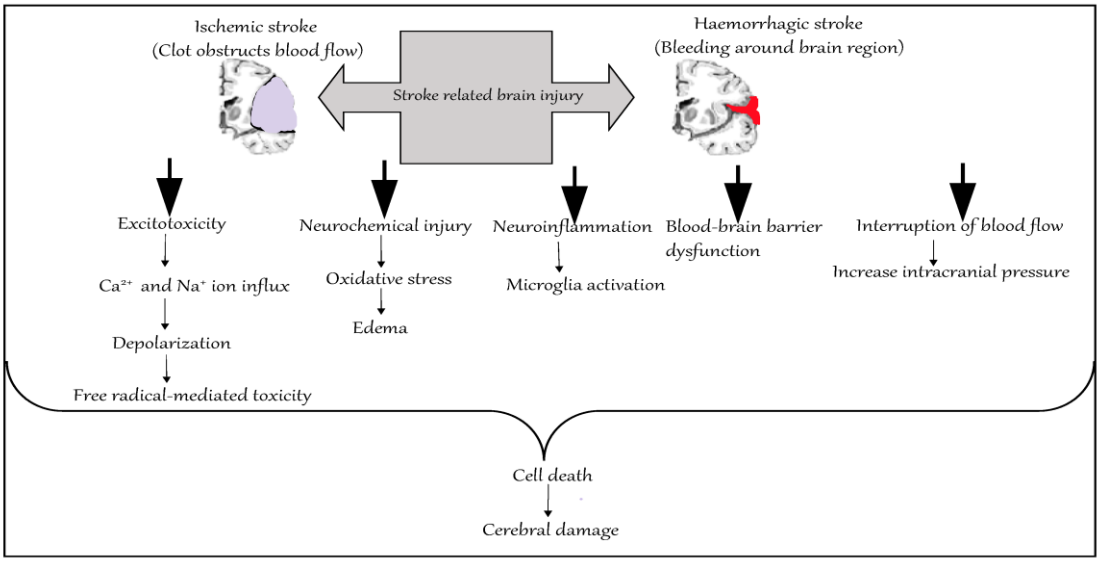
**Abbreviation:** CADASIL, cerebral autosomal dominant arteriopathy with subcortical infarcts and leukoencephalopathy.

## Pathophysiology:

The following image represents the pathophysiology of stroke, [27]

**Figure 2. Pathophysiology of Stroke**:



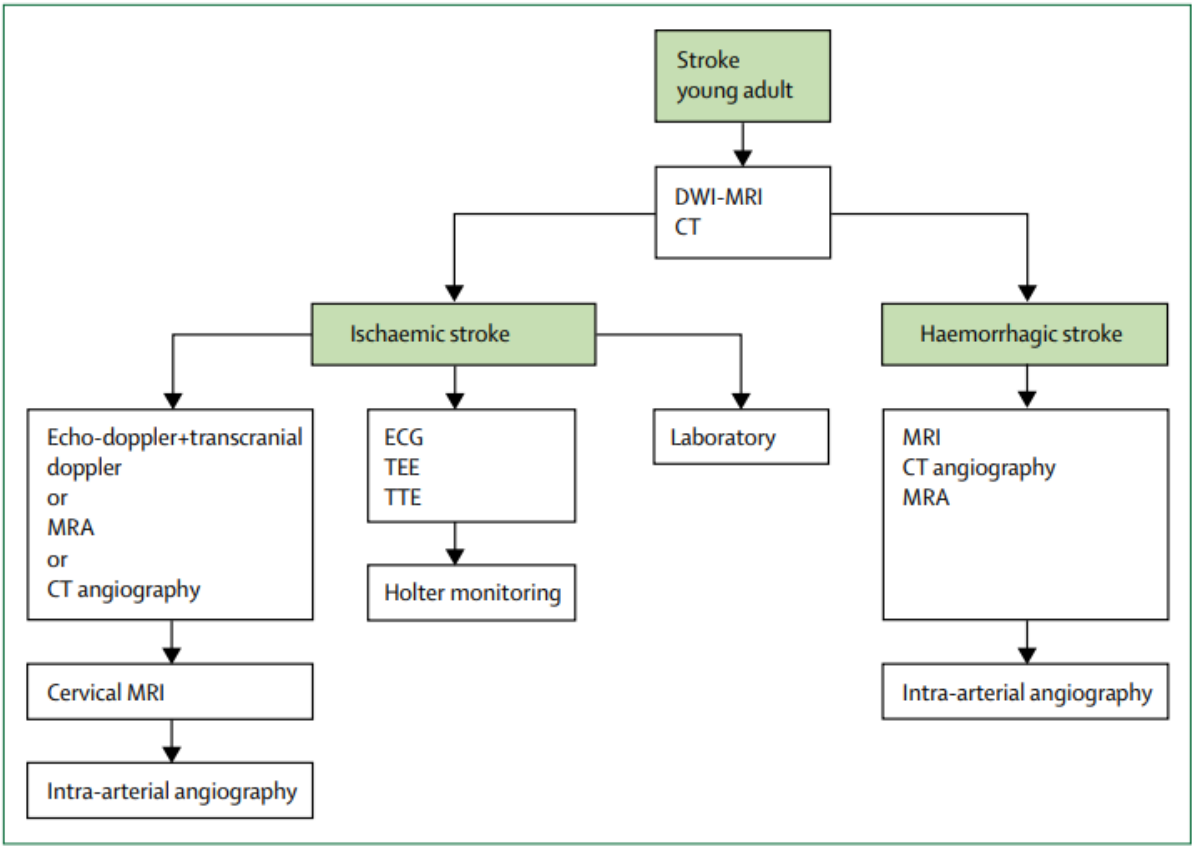

Two internal carotids located anteriorly and two vertebral arteries located posteriorly control the blood flow to the brain (the circle of Willis). Haemorrhagic stroke is brought

on by bleeding or blood vessel leaks, whereas ischemic stroke is brought on by insufficient blood and oxygen delivery to the brain.Around 85% of stroke patients lose their lives as a result of ischemic occlusions, with intracerebral haemorrhage accounting for the remaining 15%. In the brain, ischemic occlusion causes thrombotic and embolic situations.Vascular narrowing brought on vascular atherosclerosis affects blood flow in thrombosis. Plaque accumulation eventually causes the vascular chamber to narrow and clot, leading to thrombotic stroke. Reduced blood supply to the brain region in an embolic stroke results in an embolism; the reduced blood flow to the brain results in acute stress and premature cell death (necrosis). Following necrosis, the plasma membrane is disrupted, organelles enlarge and leak cellular contents into extracellular space, and neuronal function is lost. Inflammation, energy failure, loss of homeostasis, acidosis, elevated intracellular calcium levels, excitotoxicity, free radical-mediated toxicity, cytokine-mediated cytotoxicity, complement activation, deterioration of the blood-brain barrier, activation of glial cells, oxidative stress, and leukocyte infiltration are additional significant events that contribute to stroke pathology.[24,28–32]

**Diagnosis:**

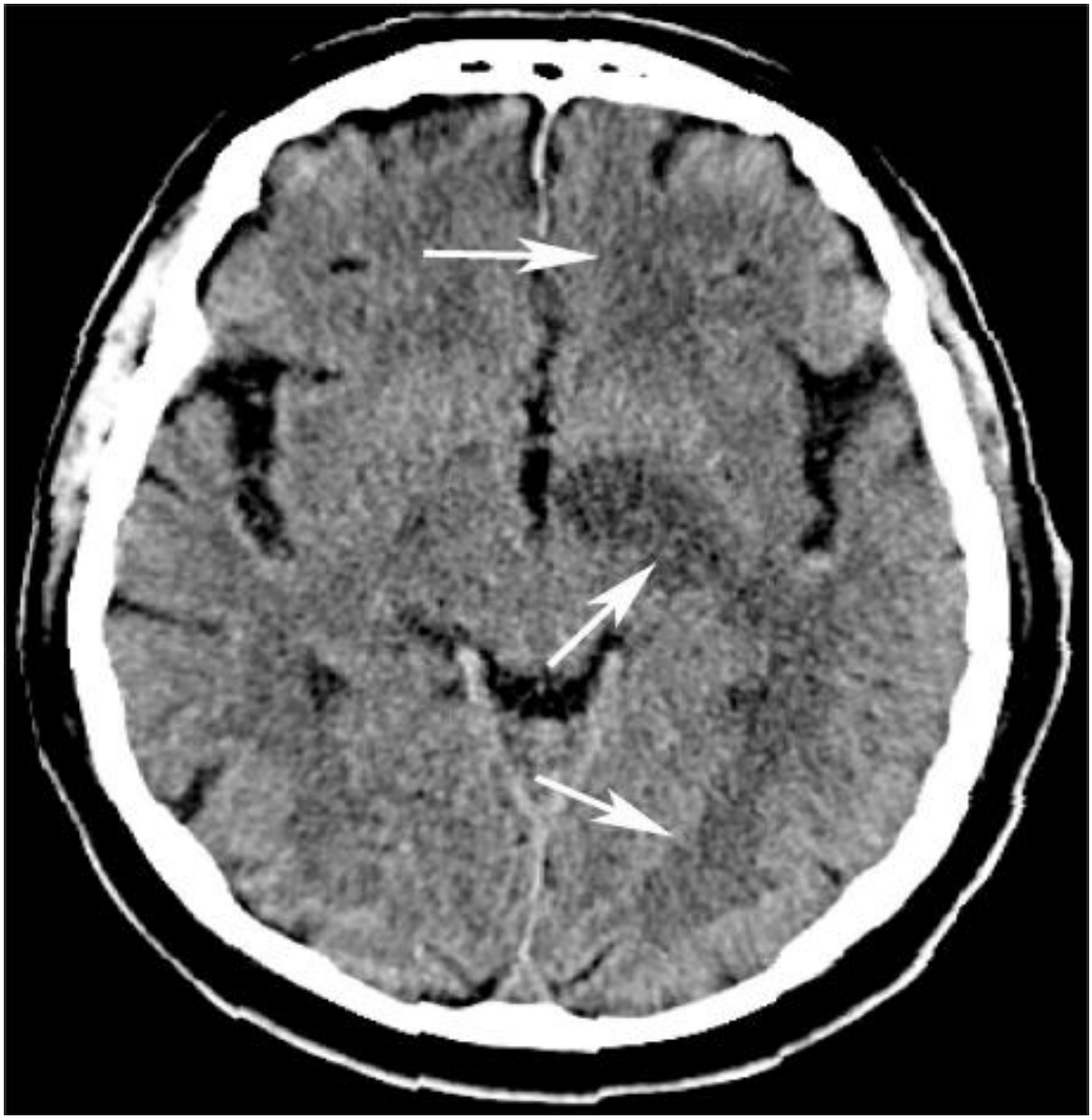
The following image represents the diagnosis of stroke ischaemic stroke to identify arterial and cardiac causes. [33]

**Figure 3. Diagnosis of stroke ischaemic stroke to identify arterial and cardiac causes:**




Both magnetic resonance imaging (MRI) and computerised tomography (CT) imaging are effective diagnostic methods. Although CT imaging is more often utilised, MRI provides information that is more precise and dependable. MRI imaging can distinguish between thrombus and haemorrhage.[34] The following image represents the computer tomography demonstrating Left middle cerebral artery (MCA) infarction indicated as Axial nonenhanced hypoattenuating foci throughout the left sided white matter (with arrows) and sulcal effacement in the left MCA territory, consistent with infarction.[35]

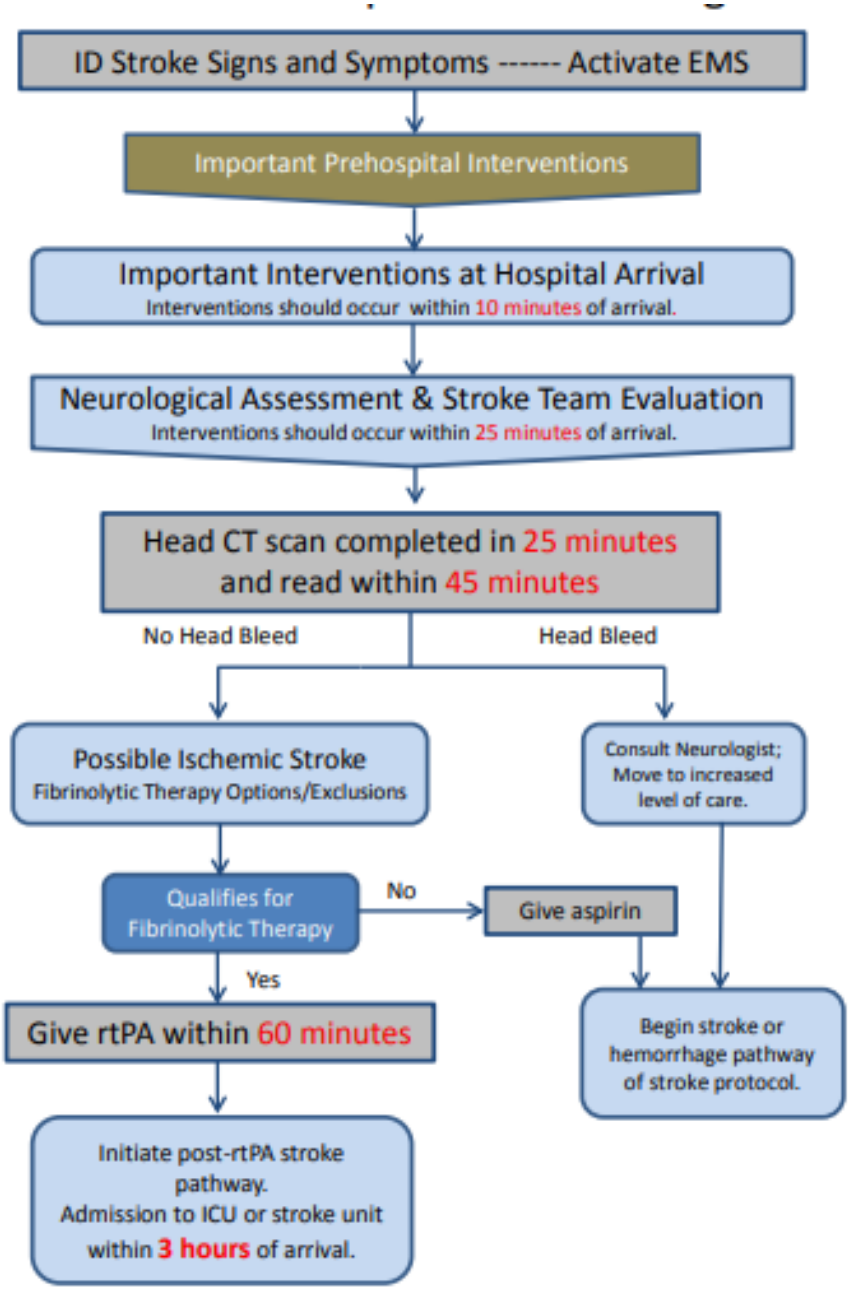
**Figure 4. CT - Left middle cerebral artery (MCA) infarction:**



## Management:

The following algorithm represents the AHA ACLS adult suspected stroke algorithm, [36]

**Figure 5. AHA ACLS adult suspected stroke algorithm:**


ID Stroke Signs and Symptoms ------ Activate EMS
Important Prehospital Interventions
Important Interventions at Hospital Arrival
Interventions should occur within 10 minutes of arrival.
Neurological Assessment & Stroke Team Evaluation
Interventions should occur within 25 minutes of arrival.
Head CT scan completed in 25 minutes and read within 45 minutes
No Head Bleed
Head Bleed
Possible Ischemic Stroke
Fibrinolytic Therapy Options/Exclusions
Consult Neurologist; Move to increased level of care.
Qualifies for Fibrinolytic Therapy
No
Give aspirin
Yes
Give rtPA within 60 minutes
Begin stroke or hemorrhage pathway of stroke protocol.
Initiate post-rtPA stroke pathway.
Admission to ICU or stroke unit within 3 hours of arrival.

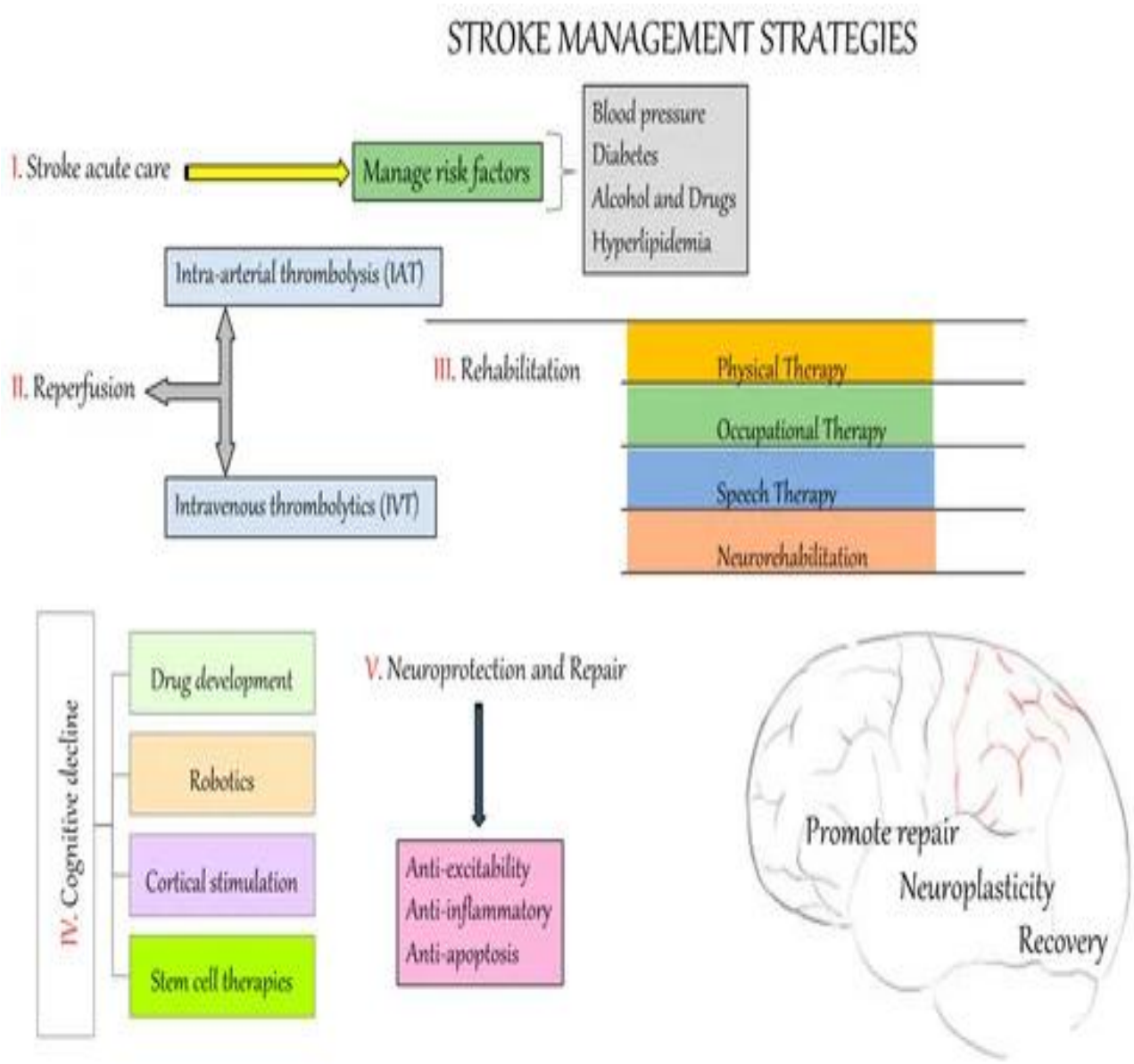



**Figure 6: Stroke therapy**

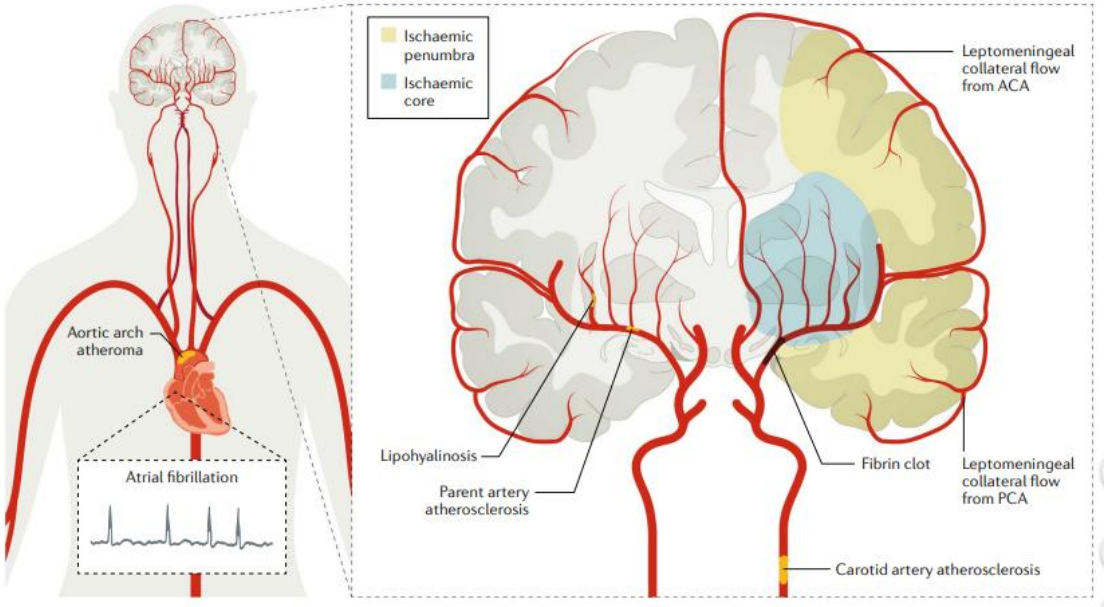
## Ischemic Stroke:

A thrombotic or embolic event that reduces blood flow to the brain is the aetiology of an ischemic stroke. When a thrombotic event occurs, the blood vessel that carries blood to the brain becomes dysfunctional, usually as a result of atherosclerotic disease, arterial dissection, fibromuscular dysplasia, or an inflammatory condition. An embolic event occurs when bodily debris obstructs blood flow through the afflicted channel.[37]

**Mechanism:**

The following image represents the mechanism of ischaemic stroke, [38]

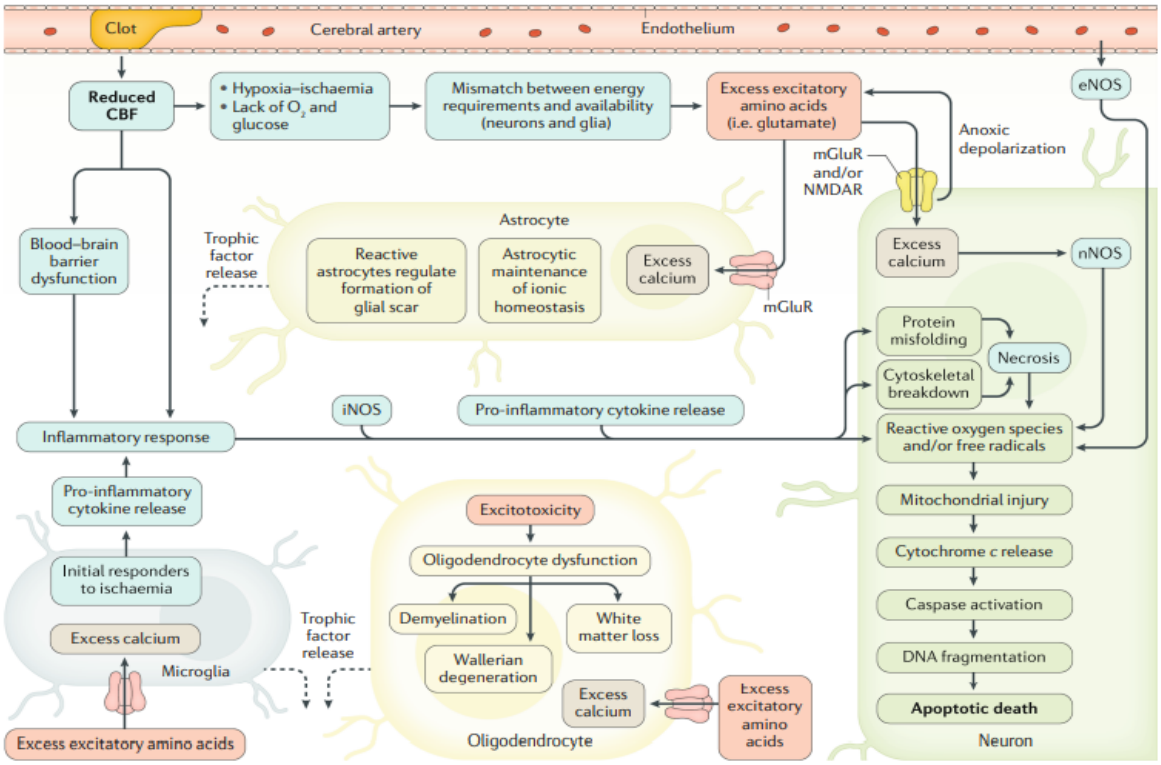
**Figure 7. Mechanism of Ischaemic stroke:**




A cascade of cellular events that result in necrosis and apoptosis are brought on by cerebral ischemia, which reduces the availability of glucose and oxygen to the brain.The

following image represents the cascade of the cellular events following cerebral ischemia,

[39]**Figure 8. Cellular effects of ischemia:**

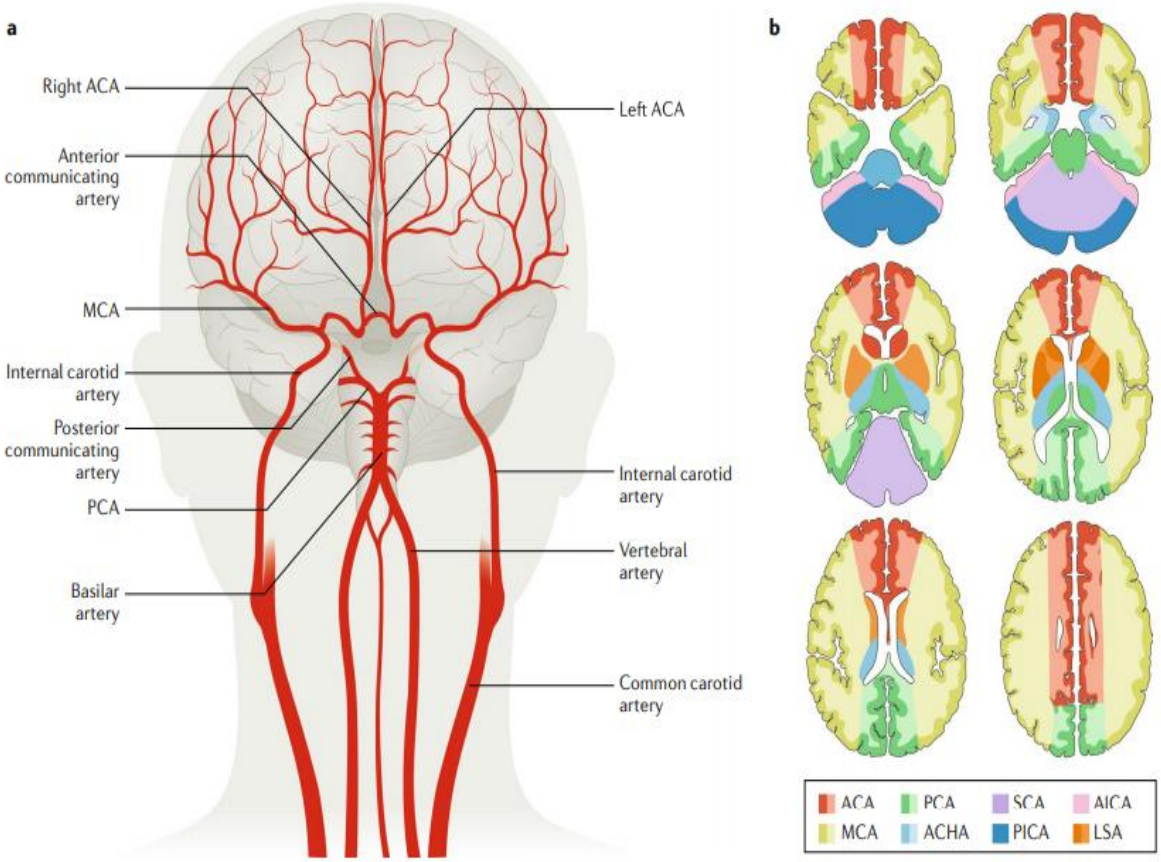



The major arteries of the brain include,

- ACA - anterior cerebral artery
- ACHA - anterior choroidal artery
- AICA - anterior inferior cerebellar artery
- LSA - lenticulostriate artery
- MCA - middle cerebral artery
- PCA - posterior cerebral artery
- PICA - posterior inferior cerebellar artery
- SCA – Superior Cerebellar Artery

The following image represents the cerebral vasculature,[40]

**Figure 9. Cerebral vasculature***:*




## CLASSIFICATION:

The TOAST classification, which uses Trial of ORG 10172 in Acute Stroke Treatment, is the one most frequently used for ischemic stroke.The following table represents the pathological or etiological types of ischaemic strokebased on the TOAST classification and its causes and prevalence, [41]

The most prevalent subtype of stroke (37.3%) was large artery atherosclerosis, and the most often affected region (49.6%) was the middle cerebral artery. The most frequent subtype of infarctions in the anterior, middle, vertebral, and anterior and posterior inferior cerebellar artery territories was large-artery atherosclerosis. In the areas of the basilar and posterior cerebral arteries, small vessel blockage was the predominant subtype. In the area of the superior cerebellar artery, cardio-embolism was the main factor.[42]

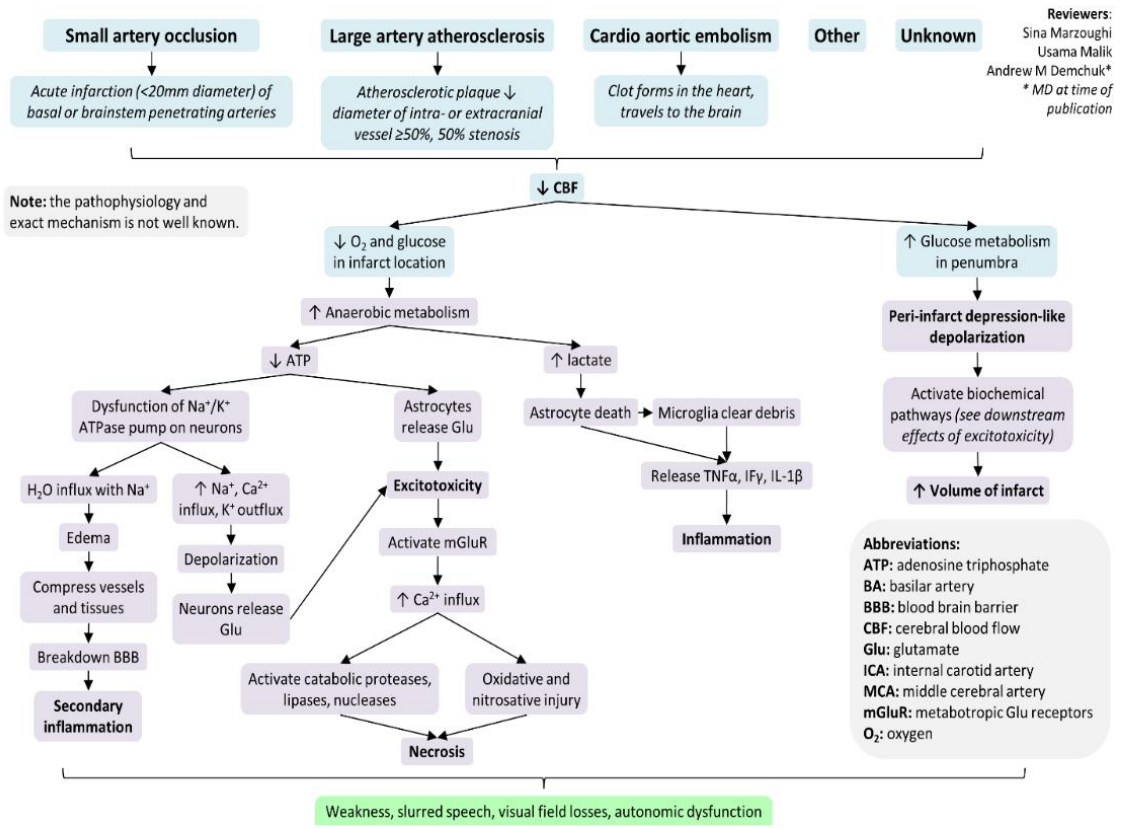
**Table 2. Types of ischaemic stroke based on the TOAST classification:**

| Stroke type | Causes | Percentage |
|---|---|---|
| Large artery thrombotic strokes | Atherosclerotic plaques in the large blood vessels of the brain lead to ischemia and infarction | 20% |
| Small penetrating artery thrombotic stroke (Lacunar stroke) | One or more vessels in the brain are affected (microatheromatosis) | 25% |
| Cardiogenic embolic stroke | Associated with cardiac dysrhythmias, valvular heart disease, and thrombi in the left ventricles | 15% |
| Cryptogenic strokes | Cause is unknown | 5-10% |
| Strokes associated with other causes | Such as illicit drug use | 20-25% |

## PATHOPHYSIOLOGY:

The following image represents the pathophysiology of ischaemic stroke, [27]

**Figure 10. Pathophysiology of ischaemic Stroke:**




## CLINICAL PRESENTATION[43–47]

Clinical presentation of stroke depends on the area of the brain affected by occlusion of the arteries. AHA/ASA (American heart association/American stroke association) have popularised the FAST algorithm to diagnose stroke in the prehospital setting. FAST acronym stands for facial droop, arm weakness, slurred speech and time of onset. Another easy way to remember to signs of stroke is the 6S method or the BEFAST method.

Stroke: Remember the 6S Method to Diagnose Stroke

- **S**udden (symptoms usually start suddenly)
- **S**lurred Speech (speech is not clear, as if drunk)
- **S**ide Weak (face, arm or leg or all three can get weak)
- **S**pinning (vertigo)
- **S**evere headache
- **S**econds (note the time when the symptoms start and rush to the hospital)

**Befast**: **B**alance (loss of balance/dizziness), **E**yes (disturbance of vision in one or both eyes), **F**ace (facial droop), **A**rm (weakness)and **S**peech (slurred) test.

Rapid Arterial Occlusion Evaluation Scale[48]

| |
|---|
| Facial palsy: Absent (0), mild (1), and moderate (2) |
| Arm motor impairment: Normal to mild (0), moderate (1), and severe (2) |
| Leg motor impairment: Normal to mild (0), moderate (1), and severe (2) |
| Head/gaze deviation: Absent (0) and present (1) |
| Aphasia: Performs tasks correctly (0), performs one task correctly (1), and performs neither task (2) |
| Agnosia: Recognizes his/her arm and deficit (0), does recognize his/her arm but not or deficit (1), and does not recognize his/her arm or deficit (2) |

A score of ≥ 5, indicates higher likelihood of large vessel occlusion with 85% sensitivity and 68% specificity

## NIH Stroke Scale

| *Tested item* | *Title* | *Responses and Scores* |
|---|---|---|
| 1A | Level of consciousness | 0-Nert |
| | | 1-Drowsy |
| | | 2-Obtunded |
| | | 3-Coma/unresponsive |
| 1B | Orientation questions (2) | 0-Answers both correctly |
| | | 1-Answers 1 correctly |
| | | 2-Answers neither correctly |
| 1C | Response to commands (2) | 0-Performs both tasks correctly |
| | | 1-Peforms 1 task correctly |
| | | 2-Performs neither |
| 2 | Gaze | 0-Normal horizontal movements |
| | | 1-Partial gaze palsy |
| | | 2-Complete gaze palsy |
| 3 | Visual fields | 0-No visual field defect |
| | | 1-Partial hemianopia |
| | | 2-Complete hemianopia |
| | | 3-Blateral hemianopia |
| 4 | Facial movement | 0-Normal |
| | | 1-Minor facial weakness |
| | | 2-Partial facial weakness |
| | | 3-Complete unilateral palsy |
| 5 | Motor function (arm) | 0-No drift |
| | a. Left | 1-Drift before 10 s |
| | b. Right | 2-Falls before 10 s |
| | | 3-No effort against gravity |
| | | 4-No movement |
| 6 | Motor function (leg) | |
| | a. Left | 1-Drift before 5 s |
| | b. Right | 2-Falls before 5 s |
| | | 3- No effort against gravity |
| | | 4-No movement |
| 7 | Limb Bladia | 0-No bladia |
| | | 1-Ataxia in 1 limb |
| | | 2-Ataxia in 2 limbs |
| 8 | Sensory | 0-No sensory loss |
| | | 1-Mild sensory loss |
| | | 2-Severe sensory loss |
| 9 | Language | 0-Normal |
| | | 1-Mild aphasia |
| | | 2-Severe aphasia |
| | | 3-Mute or global aphasia |
| 10 | Articulation | 0-Normal |
| | | 1-Mild dysarthria |
| | | 2-Severe dysarthria |
| 11 | Extinction or inattention | 0-Absent |
| | | 1-Mild loss (1 sensory modality lost) |
| | | 2-Severe loss (2 modalities lost) |

**MANAGEMENT:**[43–46]

There are two modalities of treatment available for treatment of acute ischemic stroke. Intravenous thrombolysis and mechanical thrombectomy.

Management options include majorly thrombolysis using tPA or mechanical removal of clot (Thrombectomy), anti-platelet drugs and Ancrod. [49] The following table represents the American Heart Association/American Stroke Association recommended selection criteria for mechanical thrombectomy in acute ischemic stroke. [50]

**Table 3. Selection criteria for mechanical thrombectomy in acute ischemic stroke:**

| |
|---|
| Functionally independent pre-stroke (mRS score of 0 to 1) |
| Acute ischemic stroke receiving intravenous r-tPA within 4.5 hours of onset |
| Stroke caused by occlusion of the internal carotid artery or proximal (M1) middle cerebral artery on imaging (CT angiography) |
| Age ≥18 years old |
| NIHSS score of ≥6 |
| CT brain without evidence of large infarct (ASPECTS score ≥6) |
| Treatment is able to be initiated (groin puncture) within 6 hours of symptom onset |

**Anti-platelet therapy:**

Aspirin administered within 48 hours decreases the risk of recurrent stroke and resulting improvement in outcome. [51,52]Aspirin offers less benefit than reperfusion treatments, although it is inexpensive and widely available. Alternative options include

clopidogrel or aspirin-dipyridamole combinations, which are more expensive than aspirin but slightly more effective than aspirin in the early detection of stroke.[53]In individuals who are at high risk, the combination of aspirin and clopidogrel, when given continuously for around 3 weeks after the commencement of a small stroke or Transient Ischaemic Attack, reduced the incidence of subsequent strokes.[54,55,56]

**ALTEPLASE**[57,58]

For patients who meet the inclusion criteria and have symptom onset or last known baseline within 3 hours, the AHA/ASA advises intravenous (IV) alteplase. A maximum dose of 90 mg of IV alteplase is administered at 0.9 mg/kg. The first 10% of the dose is administered as a bolus during the course of the first minute, and the remaining 90% is administered over the following 60 minutes. For those chosen candidates, the time has been increased to 4.5 hours.

The diagnosis of an ischemic stroke with "measurable neurological damage," the onset of symptoms 3 hours or less prior to treatment, and the age of 18 or older are inclusion criteria.

**Role of statins:**

Statin (3-hydroxy-3-methyl-glutaryl-coenzyme A reductase) is a lipid-lowering agent that also has multiple cholesterol-independent pleiotropic effects, comprising of,

a. Improvement of endothelial function,
b. Inhibition of inflammation & thrombosis,
c. Promotion of angiogenesis & neuroprotection,
d. Stabilization of atherosclerotic plaques.[59]

One can utilise statins like fluvastatin, simvastatin, rosuvastatin, atorvastatin, pravastatin, and rosuvastatin. Through a comprehensive review and meta-analysis, Irene Tramacere et al. examined the effect of statins for secondary prevention in ischemic stroke patients or transient ischemic attack patients. They came to the conclusion that statins are associated with a reduction in the absolute risk of cardiovascular events and ischemic strokes.[60]

**Role of Physiotherapy:**

The primary goals of the physiotherapy and rehabilitation is to prevent complications, minimise impairments and maximise their function. The interventions include positioning, early mobilisation, Modified Constraint-Induced Movement Therapy, strength trainingand that improves the balance, gait and mobility. [61]

## Blood pressure[43,45]

The recommended blood pressure management is less than 180/105 mm Hg for the first 24 hours following IV alteplase, according to the guidelines. A new advice is for patients with concomitant illnesses like acute heart failure or aortic dissection to initially decrease their blood pressure by 15%. In patients with BP less than 220/120 mm Hg, who did not get IV alteplase, and who do not have any concomitant disorders requiring blood pressure lowering, there is no advantage of antihypertensive therapy to prevent death or dependency. The first 48 to 72 hours following an acute ischemic stroke are affected by this.

The recommendation in the guideline says it could be reasonable to lower blood pressure by 15% in the first 24 hours for patients with greater than or equal to 220/120 mm Hg who did not get IV alteplase, while the benefit is not guaranteed.

10–20 mg of IV labetalol and 5 mg of IV nicardipine every hour. Every five to fifteen minutes, increase the dosage by 2.5 mg. 15 milligrammes per hour is the maximum dose.

1 to 2 mg of cilnidipine IV every hour. Every 15 minutes, double the dosage. 21 mg maximum per hour

Enalapril and hydralazine could be options.

Because cerebral perfusion pressure depends on maintaining an elevated MAP when ICP rises as a result of an ischemic event, hypotension and hypovolemia should be avoided.

**Platelet Indices in Stroke**

By sticking to the vascular endothelium, aggregating with other platelets, and starting the coagulation cascade, platelets maintain haemostasis by preventing considerable blood loss by producing a fibrin mesh. Inflammation, tissue development, and immunological response all depend on platelets. A series of derived platelet characteristics known as platelet indices (PI) — including plateletcrit, mean platelet volume (MPV), and platelet distribution width (PDW) — are collected as part of the automatic complete blood count.

In people with hypertension and diabetes mellitus, two diseases that lead to the development of vascular disease, platelet size is also observed to be increased. Since stroke is a significant public health issue, it is necessary to identify additional risk factors and potential preventive interventions.Platelets undergo a series of events (adhesion, release of granule contents, shape change, and aggregation) in response to vascular injury or trauma, which quickly lead to the creation of a platelet-fibrin plug. Thus, there is proof that acute ischemic stroke magnifies platelet function.[62]

Mean platelet volume, as well as platelet count, are an index of haemostasis and its dysfunction i.e. thrombosis. Changes in MPV play a more important role in haemostasis than platelet count. The mean lifespan of light platelets is shorter than that of heavy platelets Perturbed megakaryocyte platelet haemostatic axis (MPHA), results in the formation of hyper-functional platelets, which may contribute to the development of vascular disease or an acute thrombotic event such as ischemic stroke or myocardial infarction. Increase in platelet volume has been reported as a risk factor for acute myocardial infarction, acute cerebral ischemia, transient ischemic attacks, and for death or recurrent vascular events after myocardial infarction . Moreover, increased platelet size has been reported in patients with vascular risk factors such as diabetes , hypercholesterolemia, smoking, metabolic syndrome and in patients with renal artery stenosis.[63,64]

Mean platelet volume (MPV) and Platelet Distribution Width (PDW) is a commonly used biomarker of platelet function and activation

**Platelet Distribution Width (PDW) and Mean Platelet Volume**

Platelet (PLT) is an important blood cell which plays a major role in physiological and pathological processes such as coagulation, thrombosis, inflammation, and maintenance the integrity of vascular endothelium. [65]PLT indices are a set of variables that are used to assess the quantity, morphology, and proliferation of PLTs. Platelet count, mean platelet volume, platelet volume distribution width, platelet crit, and platelet large cell ratio are all platelet indices (PLCR). The MPV is the PCT/PLT count ratio. PDW, which is used to define the different types of PLT volume, is the coefficient of PLT volume change. These indices have a long history of use in the diagnosis of haematological disorders, but more recently it was shown that they can affect a patient's prognosis in infectious infections. A measure of platelet function and activity, platelet distribution width (PDW) displays

variation in platelet size. A measure of platelet function and activity, platelet distribution width (PDW) displays variation in platelet size. Patients with diabetes mellitus, cancer, cardio-cerebrovascular diseases, and respiratory illnesses have been shown to have higher PDW readings.[66,67]Ninety-five percent of the individuals had a MPV between 7.2 and 11.7 fL.[63,64,68–74]Furthermore, it has recently been discovered that individuals with critical illnesses, coronary artery disease, cancer, pulmonary embolism, and chronic obstructive pulmonary disease have higher morbidity and mortality rates when their PDW levels are higher. PDW reveals how uniformly sized the platelets are. A high PDW implies that platelet size varies significantly, whereas a normal PDW suggests that platelets are generally the same size. This difference is a sign of platelet activation and has been linked to vascular disorders and several malignancies. If there is any blood vessel damage, platelet activation mostly happens during the haemostasis process. Additionally, both acute and chronic inflammatory response processes involve platelet activation.[75–96]

The formula to calculate PDW is: SD/MPV (in fL) and [SD/MPV]x100 (in %). Normal values of PDW are between 10 % and 17.9 %.[97,98]

Platelet size (MPV), a physiological characteristic of haemostatic importance, is a sign (and possibly a factor of) platelet function. Discocytes, which make up up to 65% of platelets, are smooth, disc-shaped cells; the remaining 10% to 35% of platelets are less clearly defined cells (spherical platelets). The interpretation of platelet functional manifestations and experimental measurements of platelet size are both significantly impacted by morphological considerations of platelets. Platelets can be divided into fractions by variances in platelet volume using counter flow centrifugation. The relevance of the mean platelet volume (MPV) as a measure of platelet functional capability is supported by the correlations between these variations in platelet volume and variations in

density, dense body content, platelet aggregation to ADP (adenosine diphospate), and serotonin uptake and release.[64,71,99]Large platelets produce more prothrombotic factors, are physiologically more reactive, and aggregate more readily. Additionally, compared to small platelets, they have more dense granules and release more beta thromboglobulin and serotonin. Platelet count and mean platelet volume both serve as indicators of haemostasis and its dysfunction, or thrombosis. Platelet count is not as crucial to haemostasis as changes in MPV. A variety of intrinsic and extrinsic factors control platelet volume. Compared to heavy platelets, light platelets have a lower average lifespan.[100–103]

The normal mean platelet volume ranges between 7.5fl to 10.5 fl.

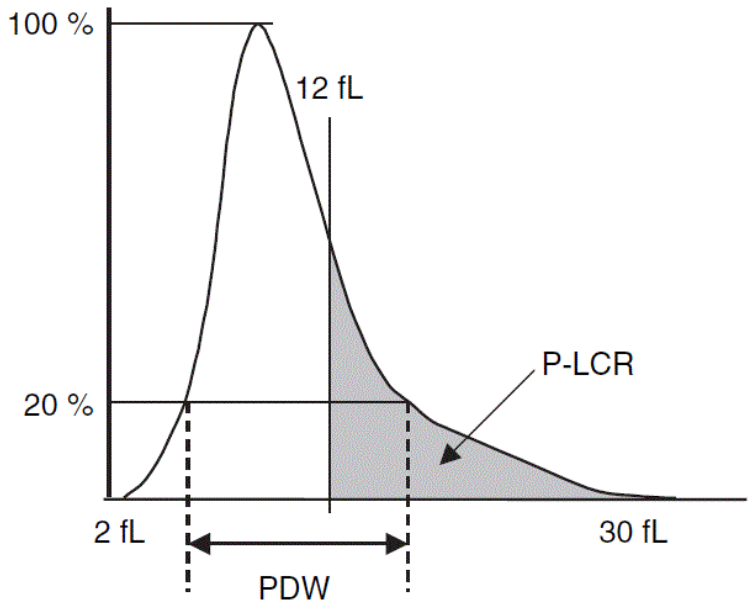



Fig 1. Histogram of platelet size distribution and the definition of platelet size deviation width (PDW), and platelet-large cell ratio (P-LCR). The distribution width at the level of 20% was defined as PDW, and the percentage of the platelets with a size of more than 12 fL was defined as P-LCR.

**Figure 11: Histogram showing PDW**

**Reviews of articles depicting the usefulness of Mean Platelet Volume (MPV) and Platelet Distribution Width (PDW) as a prognostic factor among patients with acute ischaemic stroke**

1. **Zheng et al (2022)**[73]did a systematic review and meta-analysis to explore the effect of MPV, PDW on clinical outcomes in Acute Ischaemic Stroke(AIS) and suggested that elevated MPV may be a predictive marker of adverse clinical outcome of AIS, especially in non-thrombolytic patients, while PDW has insufficient value in predicting clinical outcome of AIS.
2. **Yao et al (2022)**[104]did a study to investigate the baseline characters that influence 3-month clinical outcomes in patients with acute ischemic stroke (AIS) after thrombolytic therapy among 241 AIS patients who are treated with thrombolytic therapy with recombinant tissue plasminogen activator and found a correlation between elevated MPV and worse outcome at 3 months, particularly in large-artery atherosclerosis stroke patients. MPV and platelet count are negative correlated ($r = -0.375$, $p = 0.000$). MPV and platelet-to-lymphocyte ratio (PLR) ($r = 0.83$, $p = 0.000$), MPV and platelet distribution width (PDW) ($r = 0.820$, $p = 0.000$) both have highly positive linear correlations in patients with good outcome.
3. **Sotero et al (2021)**[63]analysed the association of platelet indices and prognosis of stroke in a cohort of 267 patients treated with rtPA.134 (50.2%) females, with a median (IQR) age of 74 years (64–82). MPV values were higher in patients with mRS 3–6 (median 8.2fL versus 7.8fL, $p = 0.013$). This association remained significant (OR = 1.36, 95% CI 1.003-1.832, $p = 0.048$) after adjustment for

variables associated with prognosis. There was a trend to unfavourable prognosis at 90 days in patients with higher MPV. Regarding the association between platelet parameters and haemorrhagic transformation, higher PDW was associated with more severe haemorrhagic transformation.

4. **Kamat et al (2020)**[105]conducted a study on 60 patients with ischemic stroke and found that 40% of the study patients were aged between 60-80 years and 60% of study population were male and 40% female. The co-morbid conditions present were hypertension in 50%, diabetes mellitus in 41% .The MPV was raised (>12.5fl) in 64 %of the patients. The clinical severity of stroke at presentation as determined by the Modified Rankin's scale (MRS) were moderate (20%), moderately severe (35%) and severe disability in 36.7% of cases. In our study MPV showed statistically significant correlation with Ischemic stroke severity. Clinical outcome as assessed by comparing MRS at admission and discharge with presentation MPV showed 25% moderately, 30% moderately severe and 23% were severely disabled. However, no statistical significance was seen.
5. **Shubham Sharma and Somya Goyal (2020)**[106] **did a study to** study the Association between platelet indices and severity of acute ischemic stroke in 100 Diabetes patients. Diabetic patients with ischemic stroke had higher PDW , MPV & PCT. No significant association was found between platelet indices and severity of stroke. Higher value of MPV, PDW & PCT (Platelet count) were associated with poor outcome.
6. **Govind D et al (2020)**[107]aimed to find the association between various hematological indices such as Mean platelet Volume (MPV), Platelet Distribution Width (PDW), Platelet to Lymphocyte ratio (PLR), and National Institute of Health

Stroke Scale (NIHSS). There is a positive correlation between PLR and NIHSS scores.

7. **Punekar and Shukla (2019)**[108]showed that there was direct relationship of MPV, PC and PLCR with severity of ischemic stroke and inverse relationship of PDW and PCT with severity of ischemic stroke. PC and PLCR were higher in patient with ischemic stroke but PC showed not significant change whereas PLCR showed significant abnormal changes in ischemic stroke patient.
8. **Mohammed et al (2019)**[69]did a study to determine whether an association exists between MPV and plateletcrit (PCT) and outcome of 157 patients with acute ischemic stroke. About 50% of the participants have favourable outcome. MPV was significantly higher in the unfavourable group (10.4 ± 2.3 fL) than in the favourable one (8.7 ± 1.3 fL) (P < 0. 001). The mean PCT was significantly higher in the unfavourable group (0.28 ± 0.1%) than in the favourable one (0.25 ± 0.1%) (P = 0. 04) but not considered as an independent predictor of poor short-term outcome of acute stroke.
9. **Gao et al (2018)**[109]did a study to determine the associations between PDW and clinical outcomes after intravenous thrombolysis in stroke patients. PDW was significantly higher for a good outcome than a poor outcome (p=0.005), with median (interquartile range) values of 16.2 (13.2–17.2) and 13.6 (12.5–15.9), respectively. PDW was also higher in patients with early neurological improvement than in patients without improvement (p=0.020) and did not differ between haemorrhage and non haemorrhage patients. The association between PDW ,16.05% and poor outcome remained in a multivariable logistic regression analysis, with an OR of 6.68 and a 95% CI of 1.69–26.49 (p=0.007).

10. **Vyawahare and Donge (2018)**[110]did a study to assess an association of platelet indices in acute ischemic stroke among 100 consecutive cases of acute ischemic stroke and equal number of age and gender match healthy controls were enrolled in study. Cases and controls ranged from 55 to 65 years of age with males (55%) outnumbering females (45%) in both groups. Incidence of hypertension, diabetes, alcohol, obesity, dyslipidaemias well as mean MPV, PDW, P-lcr and platelet count were more in cases than controls. Platelet indices increased in severe stroke, amongst MPV and platelet count increase was statistically significant. 25% mortality observed in studied cases. MPV and platelet count were increased in death and dependent cases compared to discharge and independent cases while PDW and P-lcr had positive correlation. Acute ischemic stroke associate hyponatremia had higher mortality.
11. **Sarkar et al (2018)**[101]studied 70 non diabetic non hypertensive ischemic stroke patients without previous thrombotic events & not on anti platelet medications within 24 hour of onset of symptoms & compared with equal number of age and sex matched controls. Mean age of patients was 55 ± 7.11 and of controls was 52 ± 5.37. According to CNS patients were divided in two groups; with comprehension deficit (1st group, 32 patients) & without comprehension deficit (2nd group, 38 patients).Mean value for PDW & MPV in 1st group was 18.675 ± 3.494 & 12.894 ± 1.270 respectively and in 2nd group was 18.62 ± 3.387 & 12.42 ± 0.984 respectively and was significantly higher than mean value of 15.694 ± 3.127 & 10.46 ± 1.273 of PDW & MPV respectively in controls. In both study groups PDW & MPV was found to be significantly associated with severity of motor deficit.
12. **Meena et al (2017)**[111]did a study to establish the correlation between MPV and stroke among fifty patients diagnosed with an acute ischemic stroke and showed

significant in reduction in Mean RBC, increase in Mean HB , increase in mean MPV and reduction in mean platelet count among the two groups.

13. **Ugur Lok et al (2017)**[112]did a study to investigate any possible effects of mean platelet volume (MPV) on short-term stroke prognosis and functional outcome in 250 patients with first-ever acute ischemic stroke (FEAIS) and showed that in subgroups based on MRS scores, there were no statistically significant differences according to median latency (p=0.087), median hospitalization (p=0.394), TIV (p=0.201), and MPV levels (p=0.847). Furthermore, there were no differences in MPV levels between the MRS based groups (p=0.527).
14. **Al Tameemi et al (2012)**[113]did a study assessing the relation of acute IS with different platelet indices among Fifty patients. 25 of them had first acute Ischaemic Stroke (IS) (mean age 64 years, 12 (48%) were males) [group 1], while the rest 25 patients were those with more than one IS (mean age 68 years, 16 (64%) were males) [group 2] in comparison with the control group (20) subjects (mean age 57 years, 10 (50%) males). The mean platelet count (MPC) was found to show significant difference between group 2 versus group 1 and control (P=0.012, P=0.023 respectively), while no statistically significant differences were reported with the other indices (MPV, PDW or P-LCR). Linear negative correlation was demonstrated between MPC and MPV, PDW and P-LCR in group 1, such correlation wasn't found in group 2.
15. **O' Malley et al (1995)**[100]studied 58 stroke patients consecutively admitted to a geriatric medical unit. Mean platelet volume was higher in acute stroke (11.3 compared with 10.1 fL in control subjects; P<.001, Student's t test). In addition, platelet count was reduced in stroke patients (255×109/L) compared with control subjects (299×109/L; P<.01).

# 5 RESEARCH QUESTION OR HYPOTHESIS

## 5.1 **RESEARCH QUESTION:**

Whether Mean Platelet Volume (MPV) and Platelet Distribution Width (PDW) can be used as a prognostic factor among patients with acute ischaemic stroke?

## 5.2 **NULL HYPOTHESIS:**

There is no association between Mean Platelet Volume (MPV) and Platelet Distribution Width (PDW) and prognosis of acute ischaemic stroke

## 5.3 **ALTERNATE HYPOTHESIS:**

There is association between Mean Platelet Volume (MPV) and Platelet Distribution Width (PDW) and prognosis of acute ischaemic stroke

# 6 METHODOLOGY

## 6.1 **STUDY SUBJECTS:**

Patients admitted with Acute Ischaemic Stroke in inpatient wards , Department of General Medicine, Thanjavur Medical College.

## 6.2 **STUDY DESIGN:**

Cross sectional Observational study

## **6.3 STUDY SETTING:**

Department of General Medicine, Thanjavur Medical College., Tamil Nadu

## 6.4 **SAMPLING PROCEDURE:**

All Patients admitted with Acute Ischaemic Stroke in inpatient wards , Department of General Medicine, Thanjavur Medical College were consecutively selected till the sample size was reached.

## 6.5 **INCLUSION CRITERIA:**

1. Age more than 18 years

2. Confirmed cases of stroke by history, neurologic examination and imaging modalities.

3. Patients who are willing to give written, informed consent

## 6.6 **EXCLUSION CRITERIA:**

1. Patients with haemorrhagic stroke and previous attack of ischemic stroke.

2. Patients with comorbid medical illnesses likely to interfere with platelet function or morphology like chronic kidney disease, heart bypass surgery, chronic liver diseases and leukaemia.

3. Patients receiving medication likely to interfere with platelet morphology or function like aspirin and other NSAIDs, antihistamines and some antibiotics.

4. Patients presenting 48hrs after onset of neurological symptoms

## 6.7 **SAMPLE SIZE:**

According to Sarkar et al[101] study, considering the standard deviation of Platelet Distribution Width among patients with Acute Ischaemic stroke with comprehension deficit as 18.68 with a precision of 5 and 95% confidence interval, the sample size is calculated as

$N = Z21-\alpha/2 * \sigma2/ d2$

$Z1-\alpha/2$ - two tailed proabability for 95% confidence interval = 1.96

$\sigma$ - standard deviation of Platelet Distribution Width among patients with Acute Ischaemic stroke with comprehension deficit = 18.68

d - precision or allowable error for Platelet Distribution Width among patients with Acute Ischaemic stroke with comprehension deficit = 5

N = 1.96^2 * 18.68^2 / 0.05^2

N = 53.62

Thus the total sample size required for the study is 75 after addition of 20% non-responsive rate

## 6.8 **STUDY PROCEDURE:**

All Patients admitted with Acute Ischaemic Stroke in inpatient wards , Department of General Medicine, Thanjavur Medical College were consecutively selected till the sample size of 75 was reached. After obtaining Ethical committee clearance and obtaining informed written consent, the following investigations were to be done for the purpose of the study and results were to be done.

PARAMETER ANALYSED:

Complete hemogram

Fasting and post prandial blood sugars, HbA1c

Fasting lipid profile

Renal function tests and serum electrolytes

Mean platelet volume and platelet distribution width

Electrocardiogram

Echocardiography

Imaging studies for patients alone (CT , MRI)

Modified Rankin Scale ( MRS)

**Table 4: Modified Rankin Scale ( MRS)**[114]

| Modified Rankin Scale | |
|---|---|
| 0 | No symptoms |
| 1 | No significant disability. Able to carry out all usual activities, despite some symptoms. |
| 2 | Slight disability. Able to look after own affairs without assistance, but unable to carry out all previous activities. |
| 3 | Moderate disability. Requires some help, but able to walk unassisted. |
| 4 | Moderate severe disability. Unable to attend to own bodily needs without assistance, and unable to walk unassisted. |
| 5 | Severe disability. Requires constant nursing care and attention, bedridden, incontinent. |
| 6 | Dead |

## 6.9 **ETHICAL CONSIDERATION:**

Institutional Ethical Committee approval was obtained before the start of the study.

Informed written consent was obtained from each participant.

Source of Funding: NIL

Conflict of Interest: NIL

## 6.10 STUDY PERIOD:

2 years

## 6.11 STATISTICAL METHODS:

**Descriptive Statistics:**

1. Numerical variables like age are represented in mean, median, mode and standard deviation.
2. Categorical variables like gender are represented in frequencies and percentages. Pie-charts and bar diagrams are used as appropriate.

**Inferential Statistics:**

3. When a Numerical variable is associated with the Numerical variables such as Pearson's correlation test is used after checking for normality. The Pearson product-moment correlation coefficient (Pearson's correlation, for short) is a measure of the strength and direction of association that exists between two variables measured on at least an interval scale.

4. When a Categorical Variable is associated with a categorical variable, the variables are represented in both by tables and bar diagrams. For test of significance, chi-square test is used. The chi-square test for independence, also called Pearson's chi-square test or the chi-square test of association, is used to discover if there is a relationship between two categorical variables. Fisher's exact test is used when more than 20% of the cell values have expected cell value less than 5.
5. The independent-samples t-test (or independent t-test, for short) compares the means between two unrelated groups on the same continuous, dependent variable.
6. The one-way analysis of variance (ANOVA) is used to determine whether there are any statistically significant differences between the means of three or more independent (unrelated) groups
7. P-values less than 0.05 were considered statistically significant.
8. Data was entered in MS excel sheet and analysed using SPSS software version 16.

# 7 RESULTS

Results of the study is discussed under the following headings:

**I.Age (years)**

**II.Age group**

**III.Gender**

**IV.Smoker**

**V.Alcoholic**

**VI. Anthropometry**

**VII.BMI (kg/m2)**

**VIII.BMI class**

**IX.Hypertension**

**X. Blood Pressure**

**XI.Diabetes Mellitus**

**XII. Blood Sugar**

**XIII.HbA1C**

**XIV.Dyslipidemia**

**XV.Mean Platelet Volume (fl)**

**XVI.Platelet Distribution Width**

**XVII.MRS score day1**

**XVIII.MRS score day 7**

**XIX.Mean Platelet Volume (fl) with MRS score day 1**

**XX.Mean Platelet Volume (fl) with MRS score day 7**

**XXI.Platelet Distribution Width with MRS score day1**

**XXII.Platelet Distribution Width with MRS score day 7**

**XXIII. Risk factors with MRS score day 1**

**XXIV. Risk factors with Mean Platelet Volume**

**XXV. Risk factors with Platelet Distribution Width**

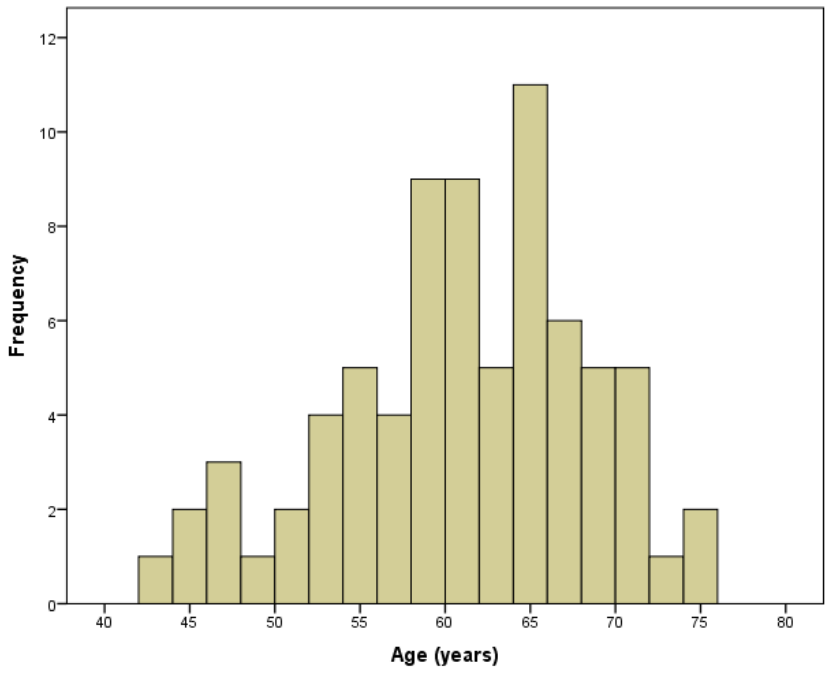
### *I.*AGE *(years)*

The mean Age (years) among the subjects was 60.52 (± 7.33) ranging from 43 to 75 years.

***Table 5.Age (years)***

| Age (years) | |
|---|---|
| **Mean** | 60.52 |
| **Median** | 60 |
| **Std. Deviation** | 7.33 |
| **Range** | 32 |
| **Minimum** | 43 |
| **Maximum** | 75 |

***Figure 12.Age (years)***



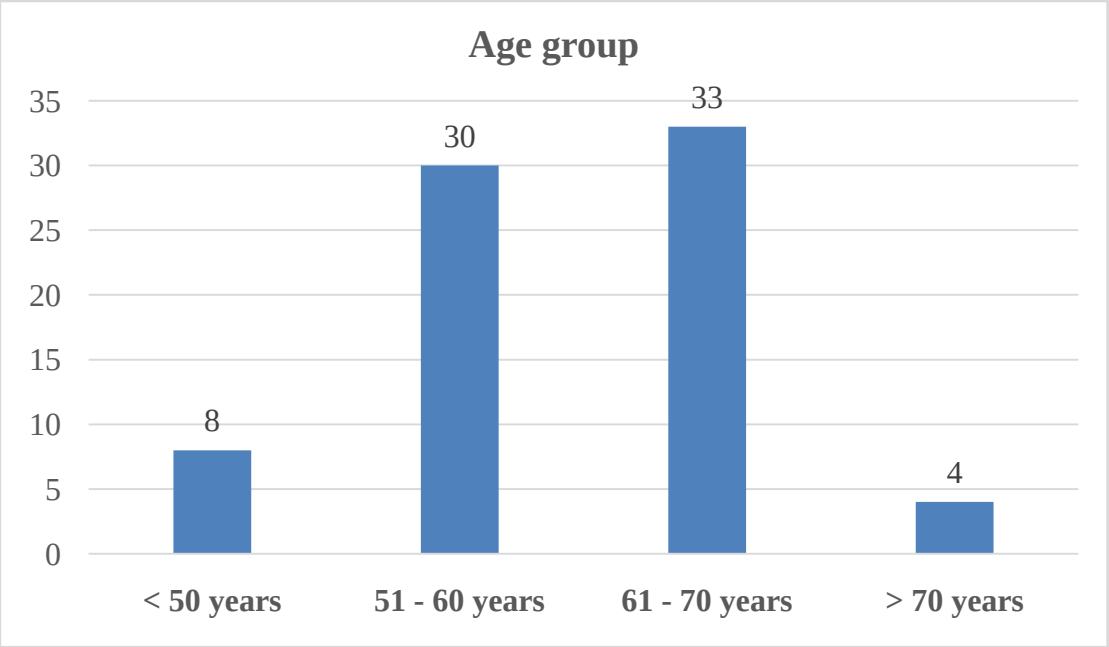
### *II.AGE GROUP*

Among the subjects, 33 (44%) were in 61 - 70 years, 30 (40%) were in 51 - 60 years and 8 (10.67%) were in < 50 years

***Table 6.Age group***

| Age group | Frequency | Percent |
|---|---|---|
| **< 50 years** | 8 | 10.67 |
| **51 - 60 years** | 30 | 40.00 |
| **61 - 70 years** | 33 | 44.00 |
| **> 70 years** | 4 | 5.33 |
| **Total** | 75 | 100.00 |

***Figure 13.Age group***



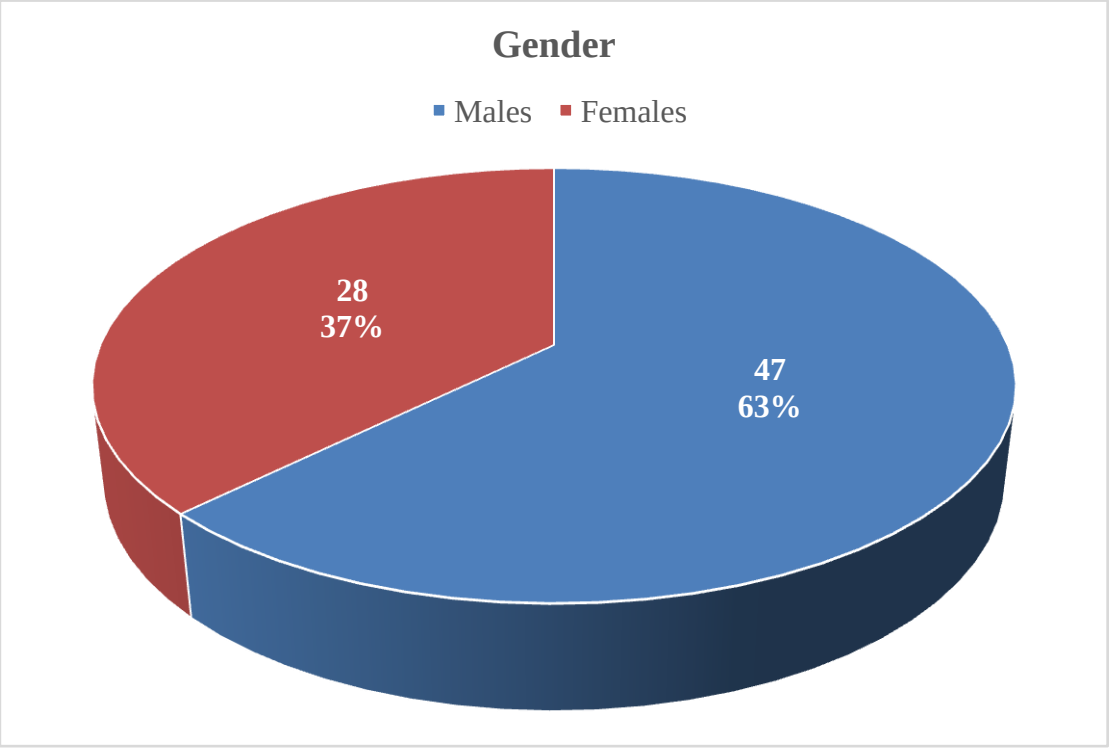
### *III.GENDER*

Among the subjects, 47 (62.67%) were Males and 28 (37.33%) were Females

***Table 7.Gender***

| Gender | Frequency | Percent |
|---|---|---|
| **Males** | 47 | 62.67 |
| **Females** | 28 | 37.33 |
| **Total** | 75 | 100.00 |

**Figure 14.Gender**



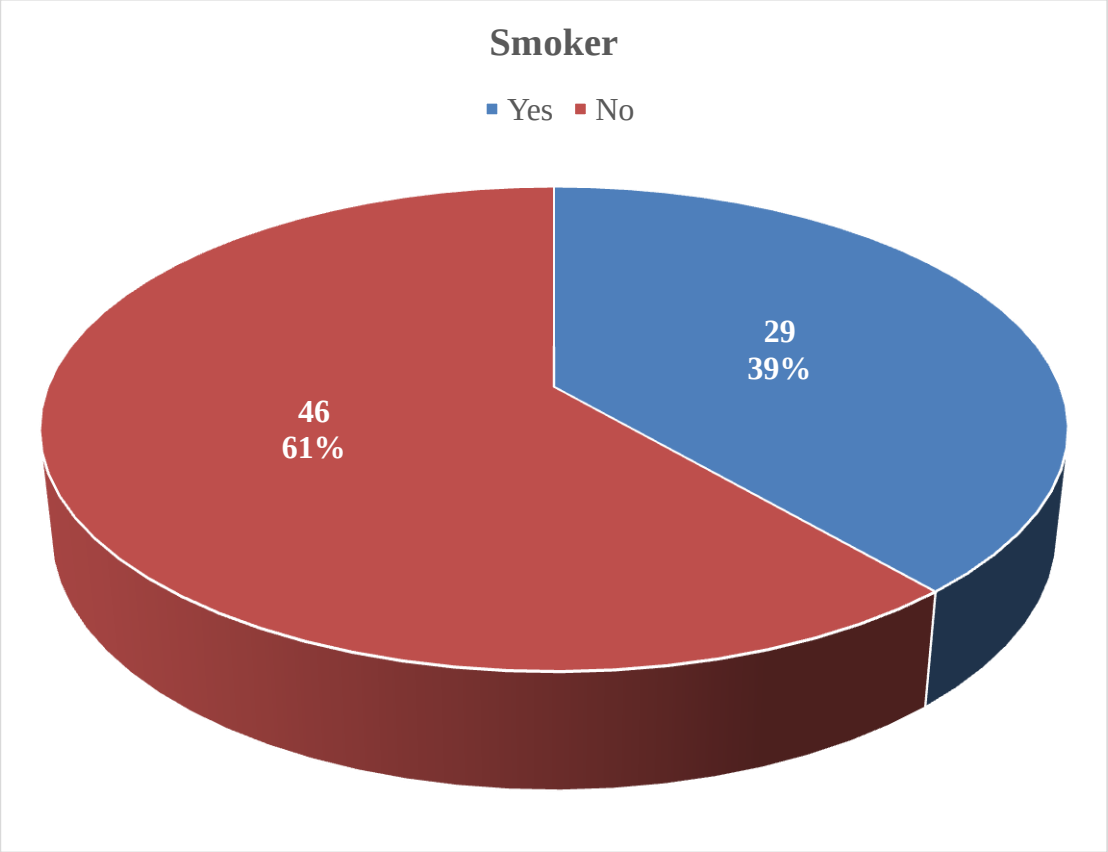
### *IV.SMOKER*

Among the subjects, 29 (38.67%) were Smoker

***Table 8.Smoker***

| Smoker | Frequency | Percent |
|---|---|---|
| **Yes** | 29 | 38.67 |
| **No** | 46 | 61.33 |
| **Total** | 75 | 100.00 |

***Figure 15.Smoker***



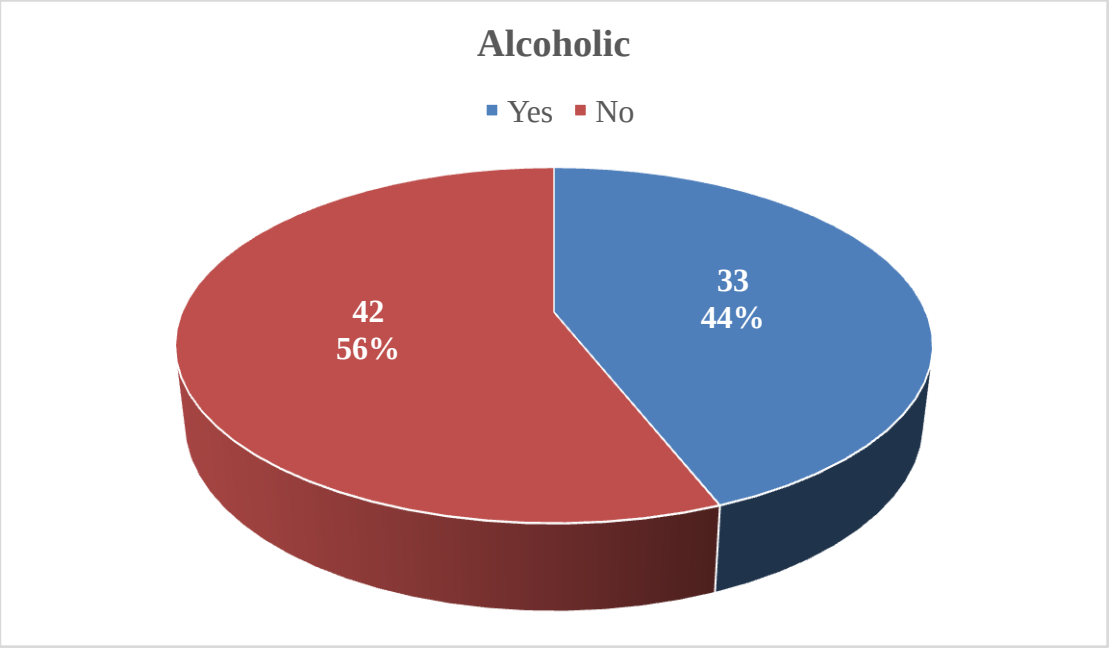
### *V.ALCOHOLIC*

Among the subjects, 33 (44%) were Alcoholic

***Table 9.Alcoholic***

| Alcoholic | Frequency | Percent |
|---|---|---|
| **Yes** | 33 | 44.00 |
| **No** | 42 | 56.00 |
| **Total** | 75 | 100.00 |

***Figure 16.Alcoholic***



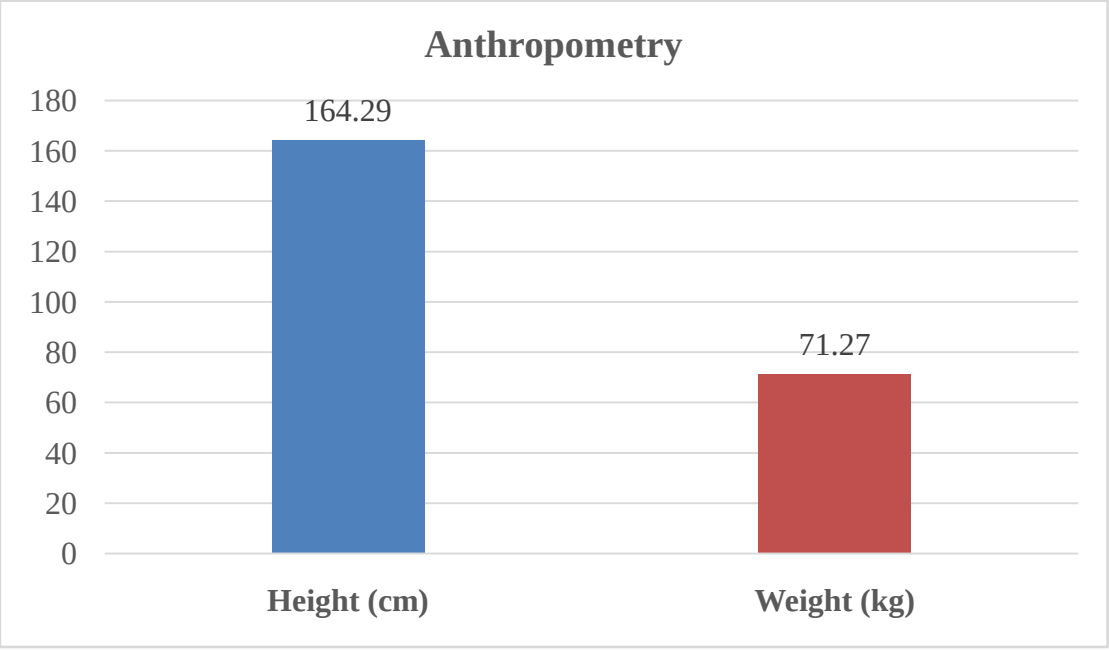
## *VI. ANTHROPOMETRY*

The mean Height (cm) among the subjects was 164.29 (± 8.08) cm ranging from 150 to 185 cm. The mean Weight (kg) among the subjects was 71.27 (± 7.81) kg ranging from 55 to 87.5 kg.

***Table 10. Anthropometry***

| | N | Mean | Std. Deviation | Minimum | Maximum |
|---|---|---|---|---|---|
| **Height (cm)** | 75 | 164.29 | 8.08 | 150.0 | 185.0 |
| **Weight (kg)** | 75 | 71.27 | 7.81 | 55.0 | 87.5 |

***Figure 17. Anthropometry***



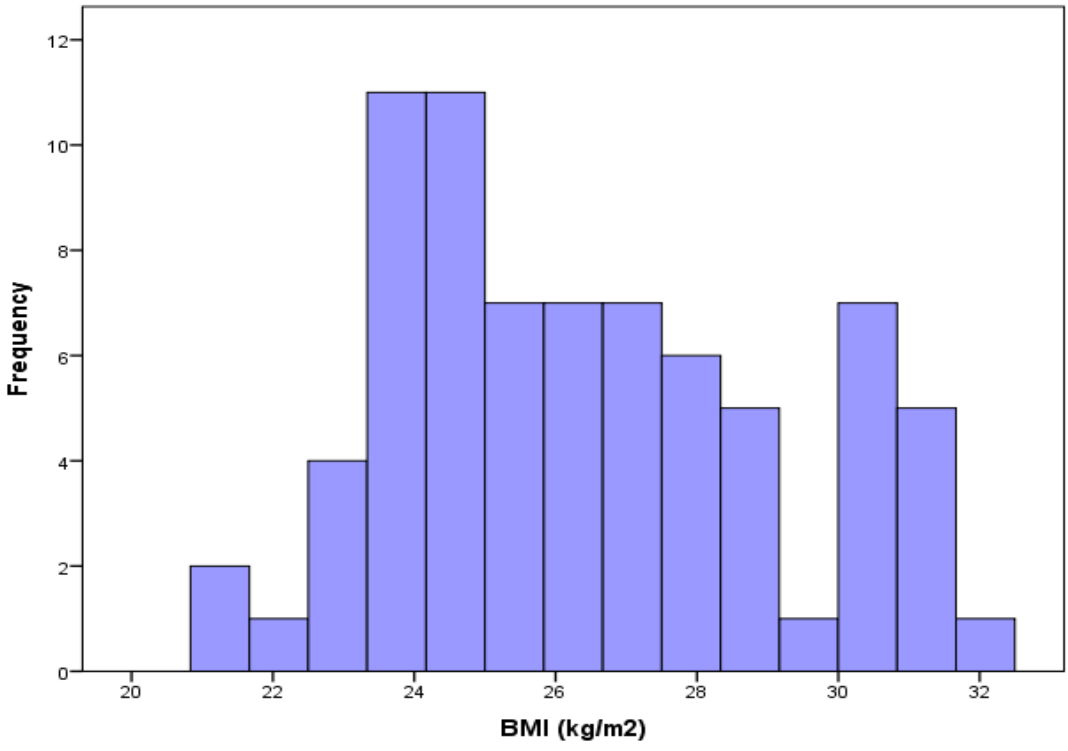
### *VII.BMI (kg/m2)*

The mean BMI (kg/m2) among the subjects was 26.4 (± 2.7) kg/m2 ranging from 21.5 to 32.2 kg/$m^2$.

***Table 11.BMI (kg/m2)***

| BMI (kg/m2) | |
|---|---|
| **Mean** | 26.40 |
| **Median** | 26.1 |
| **Std. Deviation** | 2.70 |
| **Range** | 10.7 |
| **Minimum** | 21.5 |
| **Maximum** | 32.2 |

***Figure 18.BMI (kg/m2)***



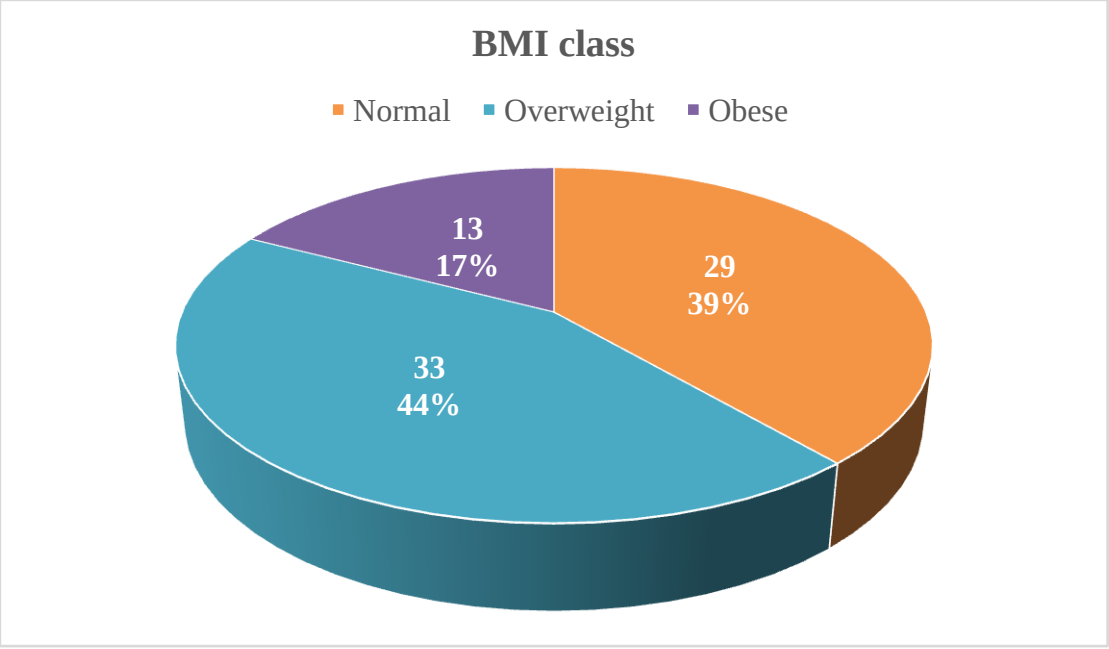
### *VIII.BMI class*

Among the subjects, 33 (44%) were Overweight, 29 (38.67%) were Normal and 13 (17.33%) were Obese

***Table 12.BMI class***

| BMI class | Frequency | Percent |
|---|---|---|
| **Normal** | 29 | 38.67 |
| **Overweight** | 33 | 44.00 |
| **Obese** | 13 | 17.33 |
| **Total** | 75 | 100.00 |

***Figure 19.BMI class***



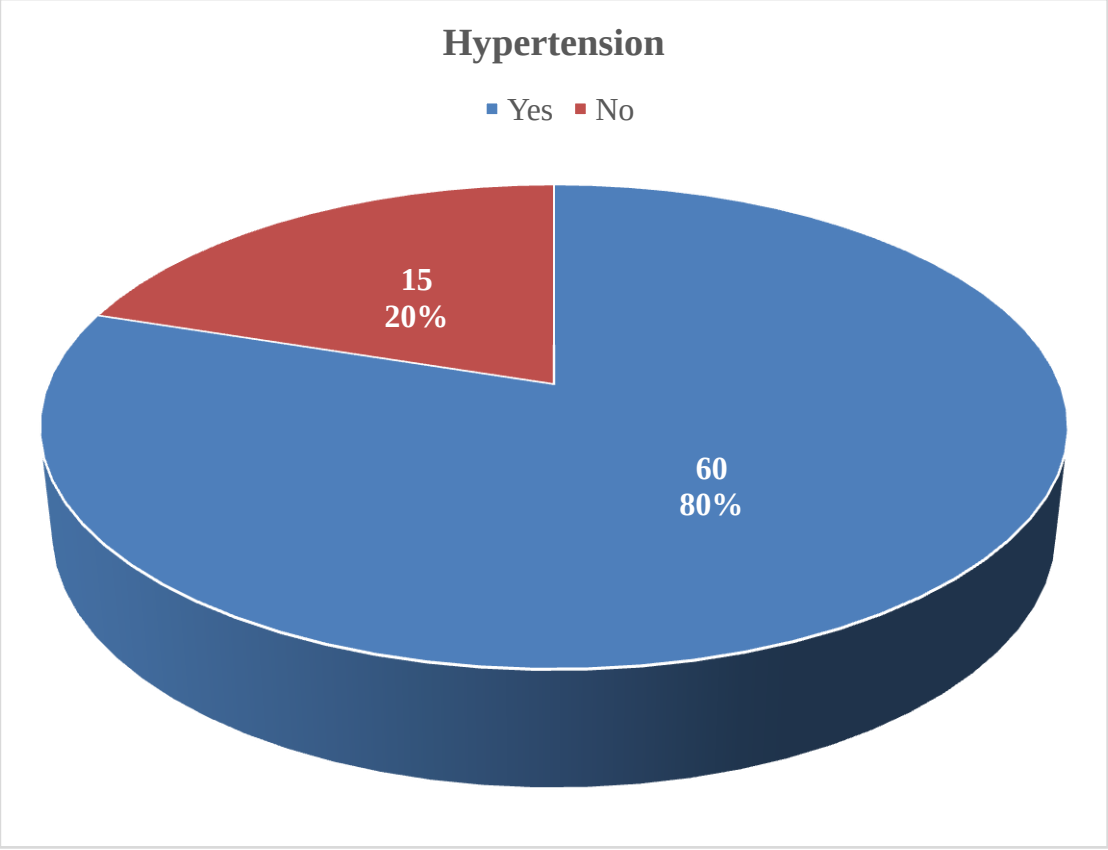
### *IX.HYPERTENSION*

Among the subjects, 60 (80%) had Hypertension

***Table 13.Hypertension***

| Hypertension | Frequency | Percent |
|---|---|---|
| **Yes** | 60 | 80.00 |
| **No** | 15 | 20.00 |
| **Total** | 75 | 100.00 |

***Figure 20.Hypertension***



### *X. Blood Pressure*

The mean Systolic Blood Pressure among the subjects was 154.85 (± 12.82) mm Hg ranging from 128 to 184 mm Hg. The mean Diastolic Blood Pressure among the subjects was 85.63 (± 6.99) mm Hg ranging from 70 to 106 mm Hg.

***Table 14. Blood Pressure***

| | **N** | **Mean** | **Std. Deviation** | **Minimum** | **Maximum** |
|---|---|---|---|---|---|
| **Systolic Blood Pressure (mm Hg)** | 75 | 154.85 | 12.82 | 128.0 | 184.0 |
| **Diastolic Blood Pressure (mm Hg)** | 75 | 85.63 | 6.99 | 70.0 | 106.0 |

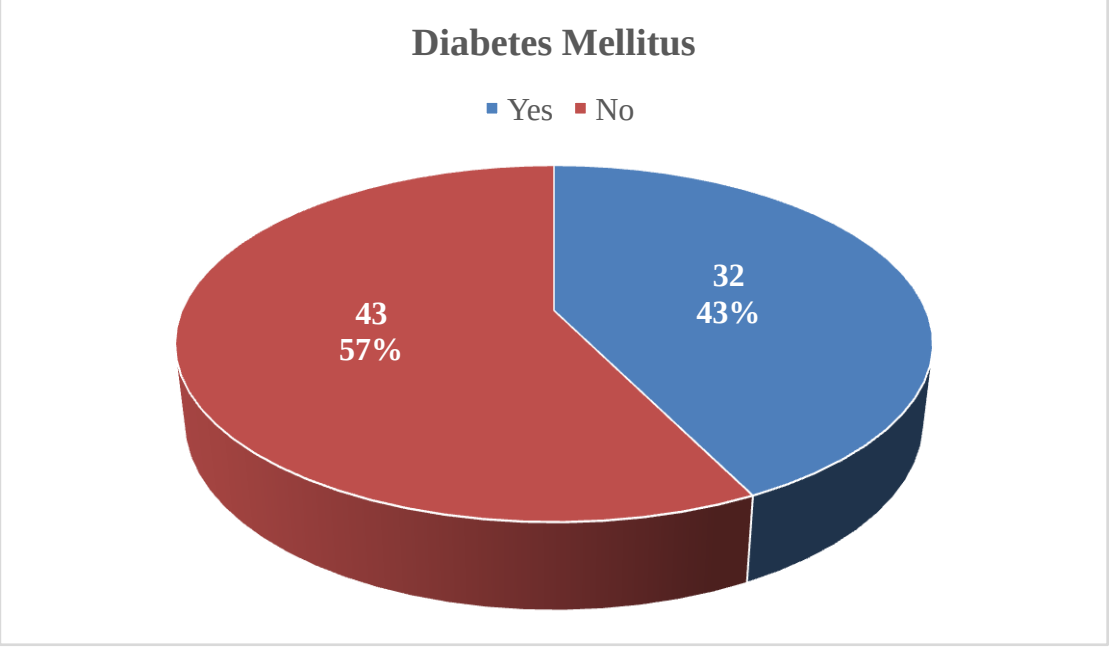
### *XI.Diabetes Mellitus*

Among the subjects, 32 (42.67%) had Diabetes Mellitus

***Table 15.Diabetes Mellitus***

| Diabetes Mellitus | Frequency | Percent |
|---|---|---|
| **Yes** | 32 | 42.67 |
| **No** | 43 | 57.33 |
| **Total** | 75 | 100.00 |

***Figure 21.Diabetes Mellitus***



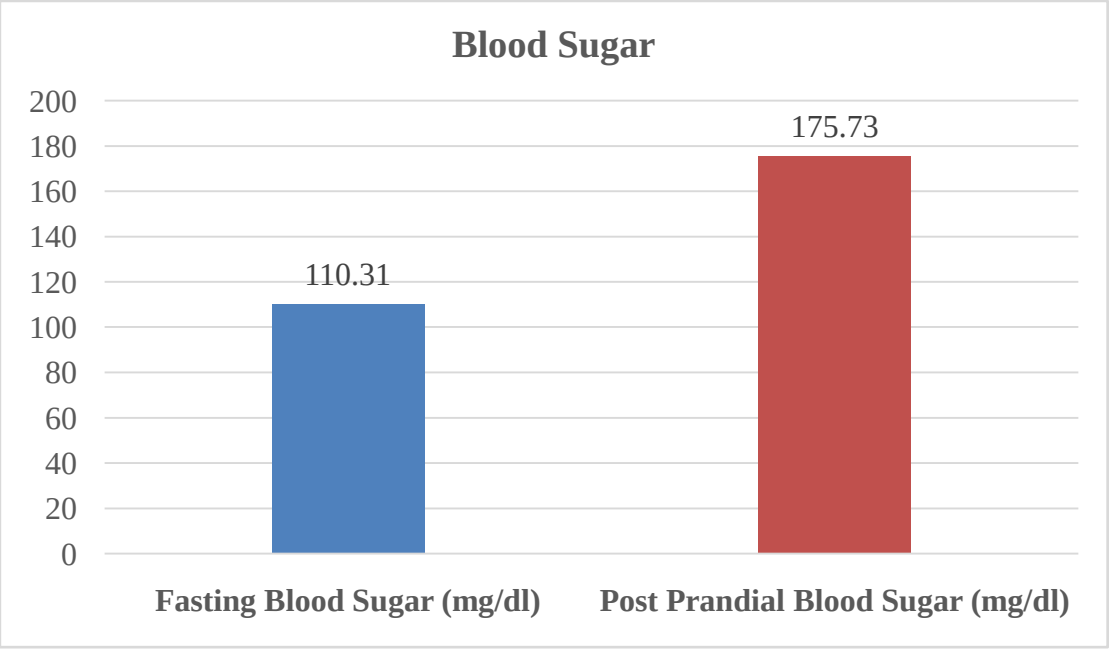
## *XII.BLOOD SUGAR*

The mean Fasting Blood Sugar (mg/dl) among the subjects was 110.31 (± 21) mg/dl ranging from 86 to 151 mg/dl. The mean Post Prandial Blood Sugar (mg/dl) among the subjects was 175.73 (± 45.75) mg/dl ranging from 117 to 287 mg/dl.

***Table 16. Blood Sugar***

| | N | Mean | Std. Deviation | Minimum | Maximum |
|---|---|---|---|---|---|
| **Fasting Blood Sugar (mg/dl)** | 75 | 110.31 | 21.00 | 86.0 | 151.0 |
| **Post Prandial Blood Sugar (mg/dl)** | 75 | 175.73 | 45.75 | 117.0 | 287.0 |

***Figure 22. Blood Sugar***



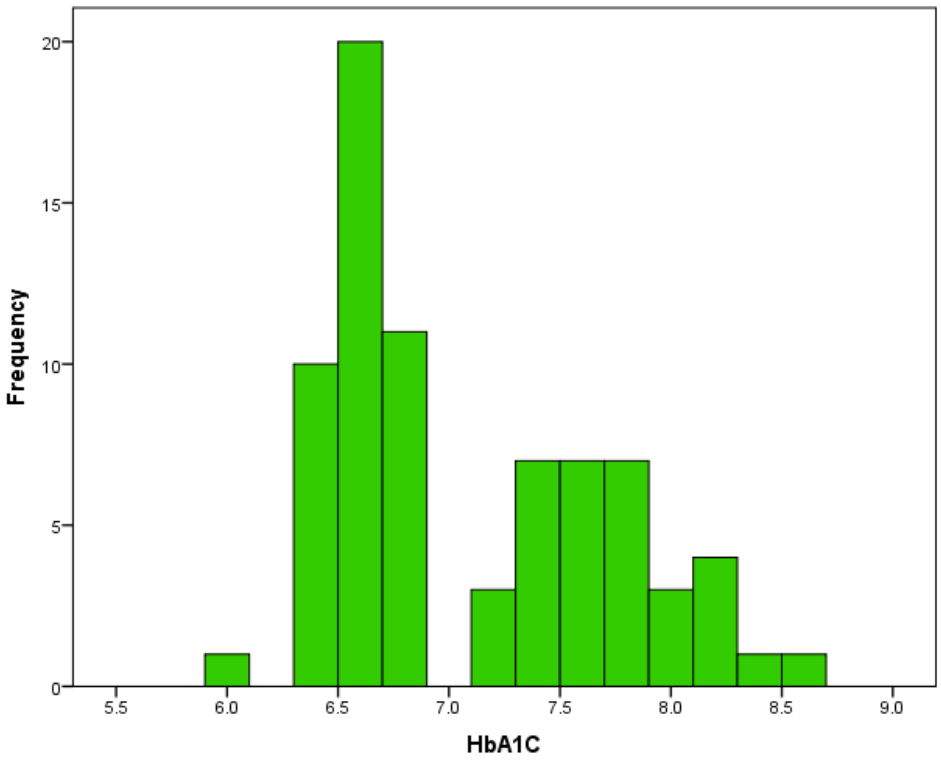
### *XIII.HbA1C*

The mean HbA1C among the subjects was 7.04 (± 0.62) ranging from 6 to 8.5%.

***Table 17.HbA1C***

| HbA1C | |
|---|---|
| **Mean** | 7.04 |
| **Median** | 6.8 |
| **Std. Deviation** | 0.62 |
| **Range** | 2.5 |
| **Minimum** | 6 |
| **Maximum** | 8.5 |

***Figure 23.HbA1C***



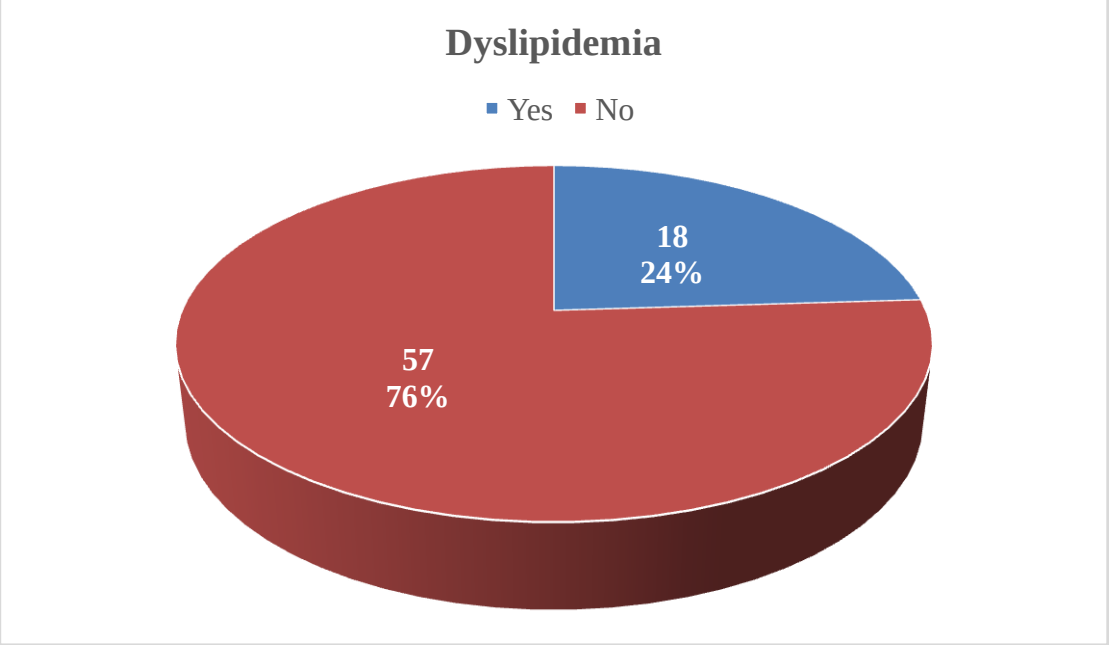
## *XIV.DYSLIPIDEMIA*

Among the subjects, 18 (24%) had Dyslipidemia

***Table 18.Dyslipidemia***

| Dyslipidemia | Frequency | Percent |
|---|---|---|
| **Yes** | 18 | 24.00 |
| **No** | 57 | 76.00 |
| **Total** | 75 | 100.00 |

***Figure 24.Dyslipidemia***



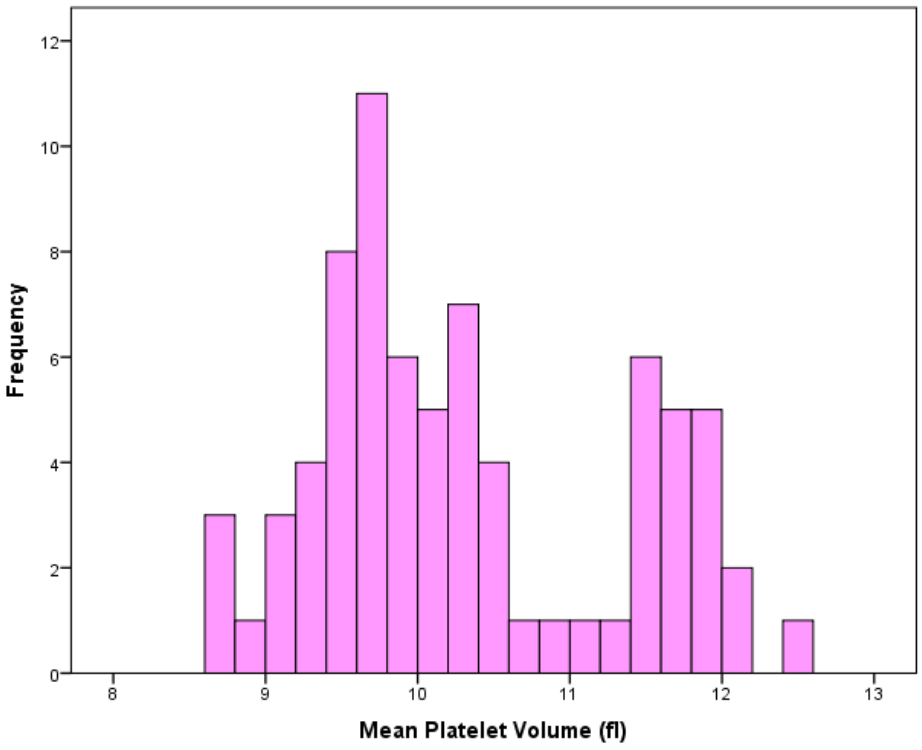
### *XV.MEAN PLATELET VOLUME (fl)*

The mean Mean Platelet Volume (fl) among the subjects was 10.28 (± 0.99) fl ranging from 8.7 to 12.4 fl

***Table 19.Mean Platelet Volume (fl)***

| Mean Platelet Volume (fl) | |
|---|---|
| **Mean** | 10.28 |
| **Median** | 10.1 |
| **Std. Deviation** | 0.99 |
| **Range** | 3.7 |
| **Minimum** | 8.7 |
| **Maximum** | 12.4 |

***Figure 25.Mean Platelet Volume (fl)***



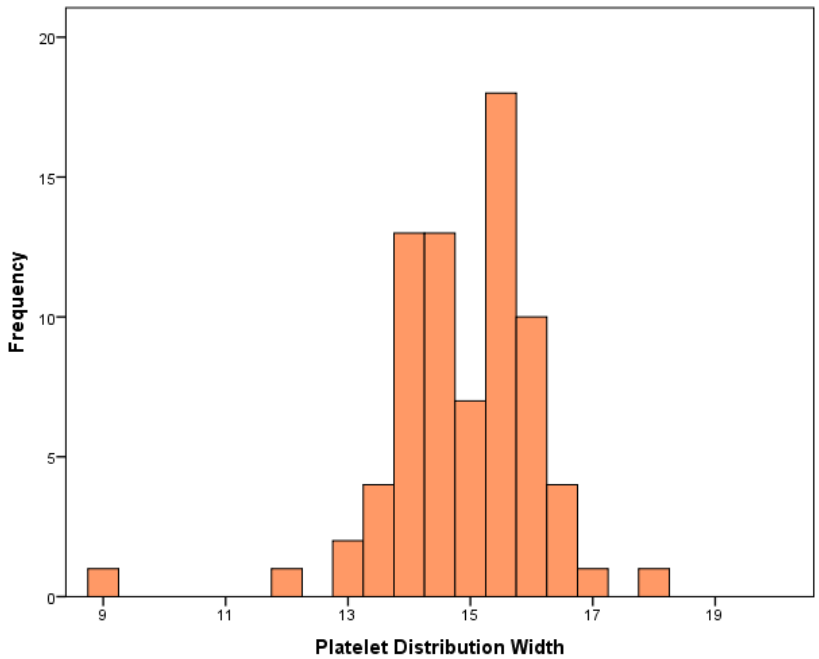
## *XVI.PLATELET DISTRIBUTION WIDTH*

The mean Platelet Distribution Width among the subjects was 14.91 (± 1.23) ranging from 9 to 18.1%.

***Table 20.Platelet Distribution Width***

| **Platelet Distribution Width (%)** | |
|---|---|
| **Mean** | 14.91 |
| **Median** | 15.1 |
| **Std. Deviation** | 1.23 |
| **Range** | 9.1 |
| **Minimum** | 9 |
| **Maximum** | 18.1 |

***Figure 26.Platelet Distribution Width***



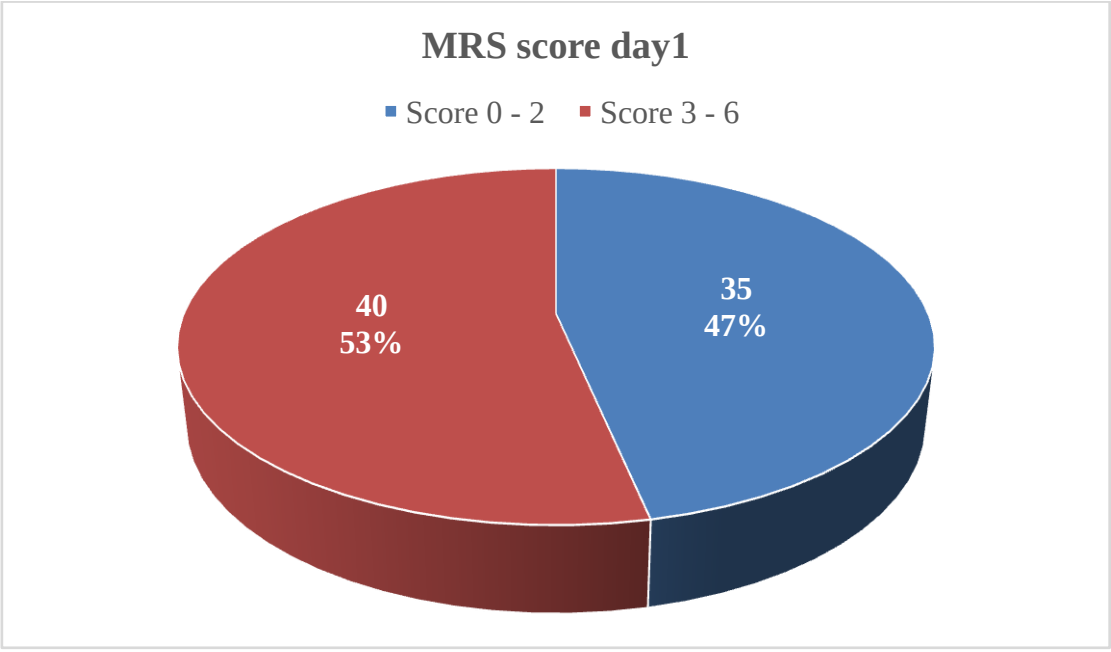
### *XVII.MRS score day1*

Among the subjects, 40 (53.33%) had MRS Score 3 - 6 and 35 (46.67%) had MRS Score 0 – 2 on day 1.

***Table 21.MRS score day1***

| MRS score day1 | Frequency | Percent |
|---|---|---|
| **Score 0 – 2** | 35 | 46.67 |
| **Score 3 – 6** | 40 | 53.33 |
| **Total** | 75 | 100.00 |

***Figure 27.MRS score day1***



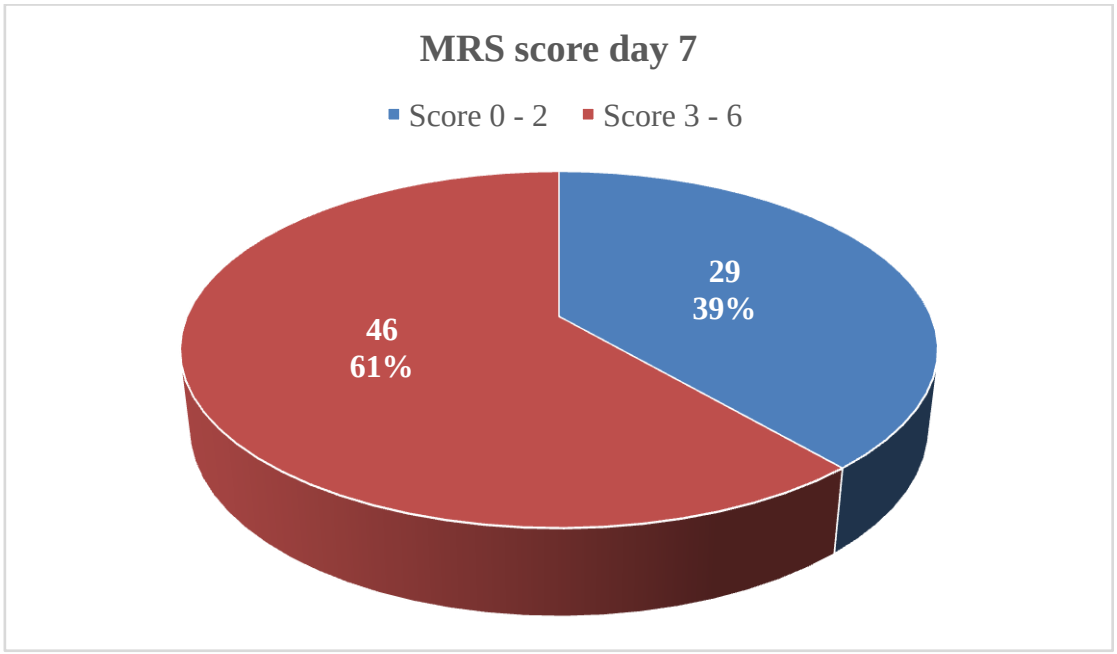
### *XVIII.MRS SCORE DAY 7*

Among the subjects, 46 (61.33%) had MRS Score 3 - 6 and 29 (38.67%) had MRS Score 0 – 2 on day 7.

***Table 22.MRS score day 7***

| MRS score day 7 | Frequency | Percent |
|---|---|---|
| **Score 0 – 2** | 29 | 38.67 |
| **Score 3 – 6** | 46 | 61.33 |
| **Total** | 75 | 100.00 |

***Figure 28.MRS score day 7***



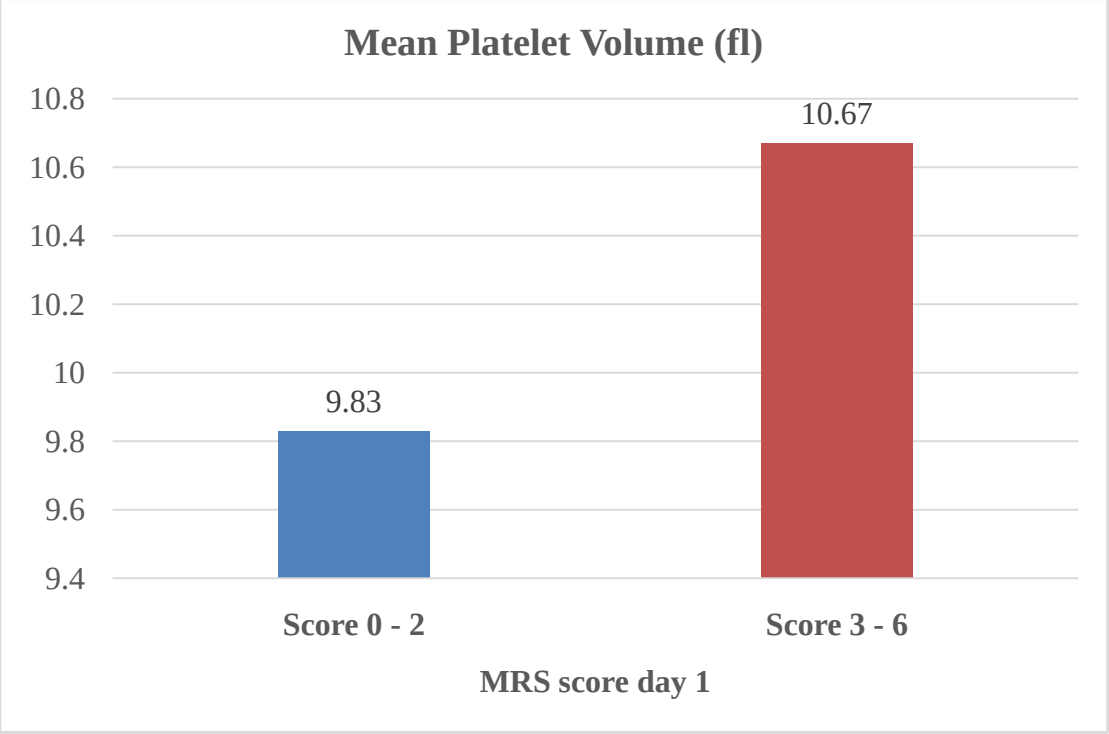
### *XIX.MEAN PLATELET VOLUME (FL) WITH MRS SCORE DAY 1*

The mean Mean Platelet Volume (fl) with MRS score day 1, among Score 0 - 2 MPV was 9.83 (± 0.72) which is lower by 0.84 and statistically significant compared to MPV 10.67 (± 1.04) in Score 3 - 6

***Table 23. Mean Platelet Volume (fl) with MRS score day 1***

| | MRS score day1 | N | Mean | Std. dev. | Mean diff. | p value by 't' test |
|---|---|---|---|---|---|---|
| **Mean Platelet Volume (fl)** | **Score 0 - 2** | 35 | 9.83 | 0.72 | 0.839 | 0.001 |
| | **Score 3 - 6** | 40 | 10.67 | 1.04 | | |

***Figure 29. Mean Platelet Volume (fl) with MRS score day 1***



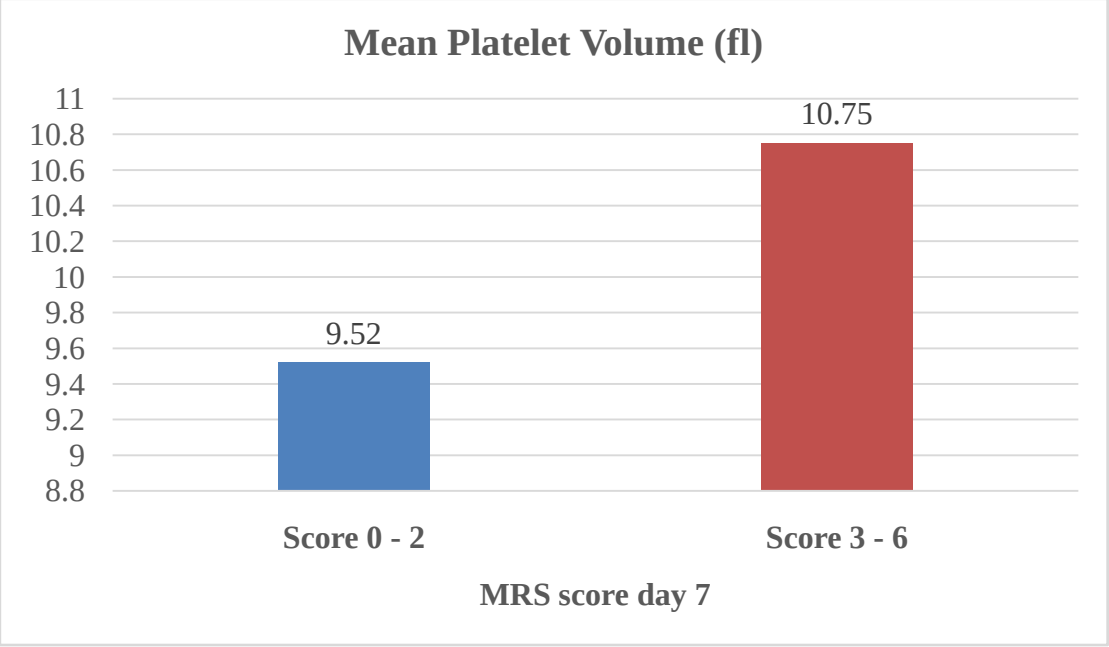
### *XX.MEAN PLATELET VOLUME (FL) WITH MRS SCORE DAY 7*

The mean Mean Platelet Volume (fl) with MRS score at day 7 among Score 0 - 2 MPV was 9.52 (± 0.47) which is lower by 1.23 and statistically significant compared to MPV 10.75 (± 0.94) in Score 3 - 6

***Table 24.Mean Platelet Volume (fl) with MRS score day 7***

| | MRS score day 7 | N | Mean | Std. dev. | Mean diff. | p value by 't' test |
|---|---|---|---|---|---|---|
| **Mean Platelet Volume (fl)** | **Score 0 - 2** | 29 | 9.52 | 0.47 | 1.231 | 0.001 |
| | **Score 3 - 6** | 46 | 10.75 | 0.94 | | |

***Figure 30.Mean Platelet Volume (fl) with MRS score day 7***



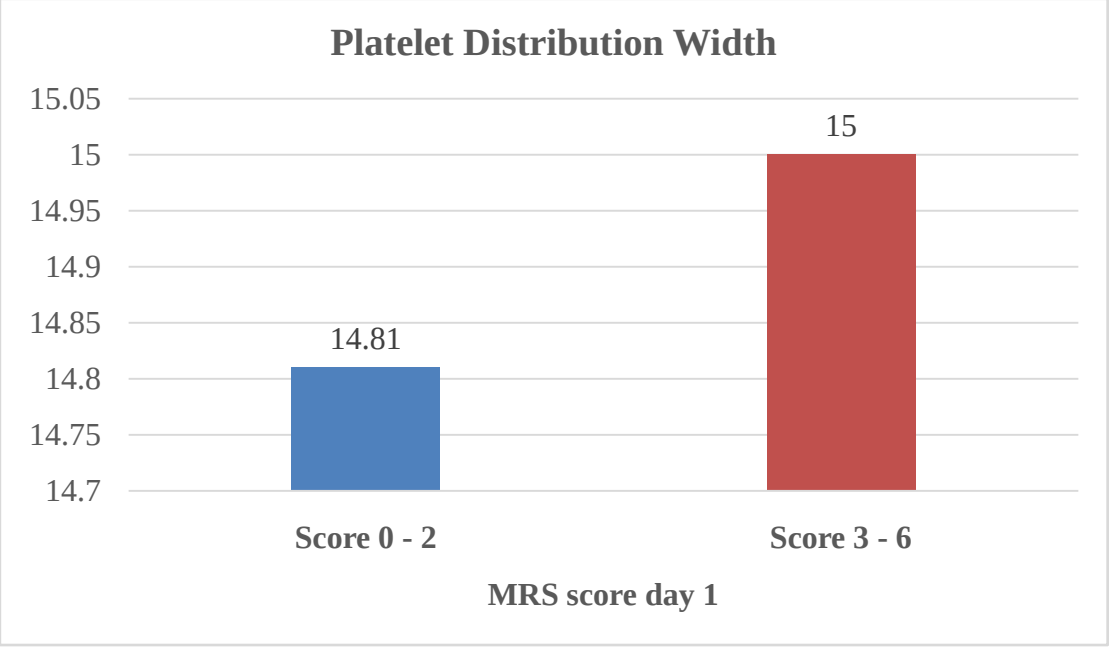
### *XXI.PLATELET DISTRIBUTION WIDTH WITH MRS SCORE DAY1*

The mean Platelet Distribution Width with MRS score day 1 among Score 0 - 2 PDW was 14.81 (± 1.11) which is lower by 0.2 but not statistically significant compared to PDW 15 (± 1.34) in Score 3 - 6

***Table 25.Platelet Distribution Width with MRS score day1***

| | MRS score day1 | N | Mean | Std. dev. | Mean diff. | p value by 't' test |
|---|---|---|---|---|---|---|
| **Platelet Distribution Width** | **Score 0 - 2** | 35 | 14.81 | 1.11 | 0.197 | 0.494 |
| | **Score 3 - 6** | 40 | 15.00 | 1.34 | | |

***Figure 31.Platelet Distribution Width with MRS score day1***



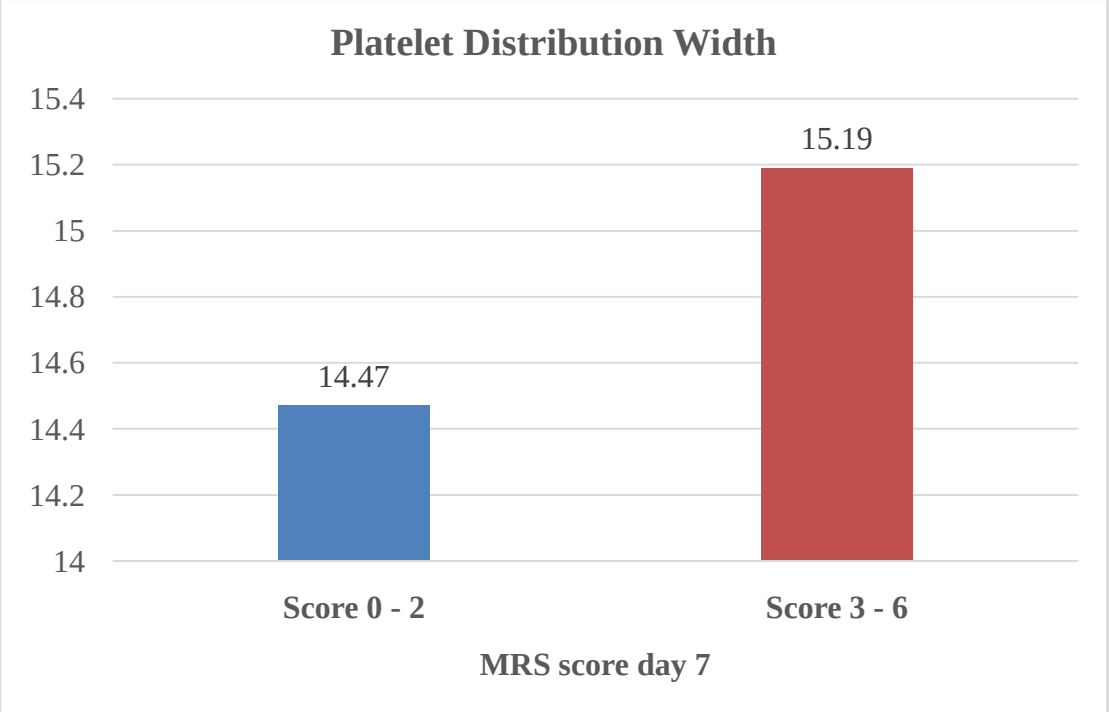
### *XXII.PLATELET DISTRIBUTION WIDTH WITH MRS SCORE DAY 7*

The mean Platelet Distribution Width with MRS score day 7, among Score 0 - 2 PDW was 14.47 (± 0.91) which is lower by 0.71 and statistically significant compared to PDW 15.19 (± 1.33) in Score 3 - 6

***Table 26.Platelet Distribution Width with MRS score day 7***

| | MRS score day 7 | N | Mean | Std. dev. | Mean diff. | p value by 't' test |
|---|---|---|---|---|---|---|
| **Platelet Distribution Width** | **Score 0 - 2** | 29 | 14.47 | 0.91 | 0.715 | 0.013 |
| | **Score 3 - 6** | 46 | 15.19 | 1.33 | | |

***Figure 32.Platelet Distribution Width with MRS score day 7***



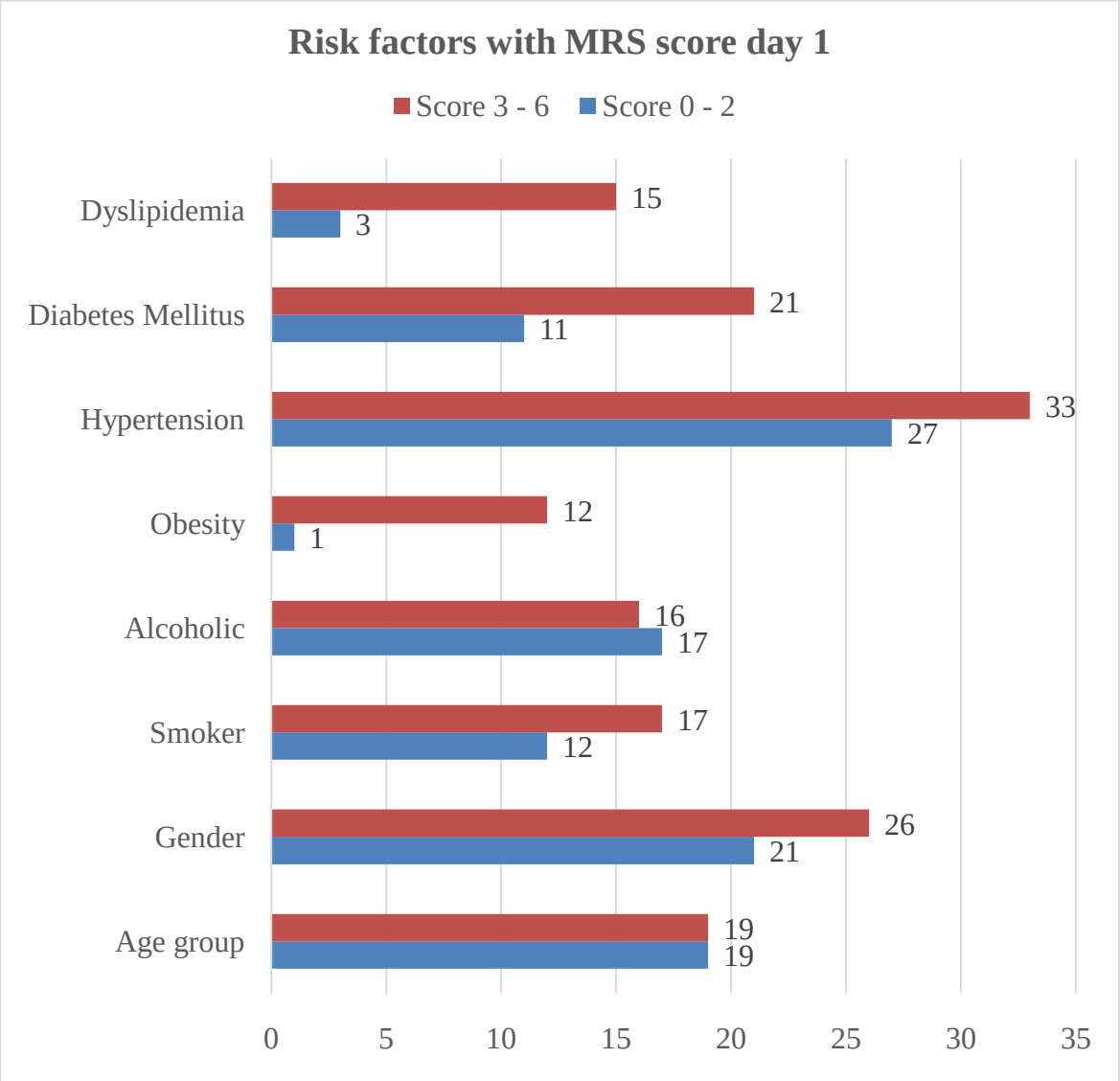
### *XXIII. RISK FACTORS WITH MRS SCORE DAY 1*

92.3% of the obese subjects, and 83.3% of the subjects with dyslipidemia had MRS score 3 – 6 with higher severity of stroke and the differences were statistically significant. The other risk factors like age, gender, smoking, alcoholic, hypertension and diabetes mellitus was not statistically significant with the MRS score distribution.

***Table 27. Risk factors with MRS score day 1***

| **Risk factors** | **MRS score day1** | | **Chi sq. p value** |
|---|---|---|---|
| | **Score 0 - 2** | **Score 3 - 6** | |
| **Age group** | 19 (50%) | 19 (50%) | 0.558 |
| **Gender** | 21 (44.7%) | 26 (55.3%) | 0.655 |
| **Smoker** | 12 (41.4%) | 17 (58.6%) | 0.466 |
| **Alcoholic** | 17 (51.5%) | 16 (48.5%) | 0.456 |
| **Obesity** | 1 (7.7%) | 12 (92.3%) | **0.002** |
| **Hypertension** | 27 (45%) | 33 (55%) | 0.583 |
| **Diabetes Mellitus** | 11 (34.4%) | 21 (65.6%) | 0.066 |
| **Dyslipidemia** | 3 (16.7%) | 15 (83.3%) | **0.003** |

***Figure 33. Risk factors with MRS score day 1***



### *XXIV. RISK FACTORS WITH MEAN PLATELET VOLUME*

The Mean Platelet Volume among those with Hypertension, Diabetes and Dyslipidemia were significantly higher compared to those without the risk factors. Subjects with more than 60 years of age and obese subjects had significantly higher mean platelet volume and the differences were statistically significant.

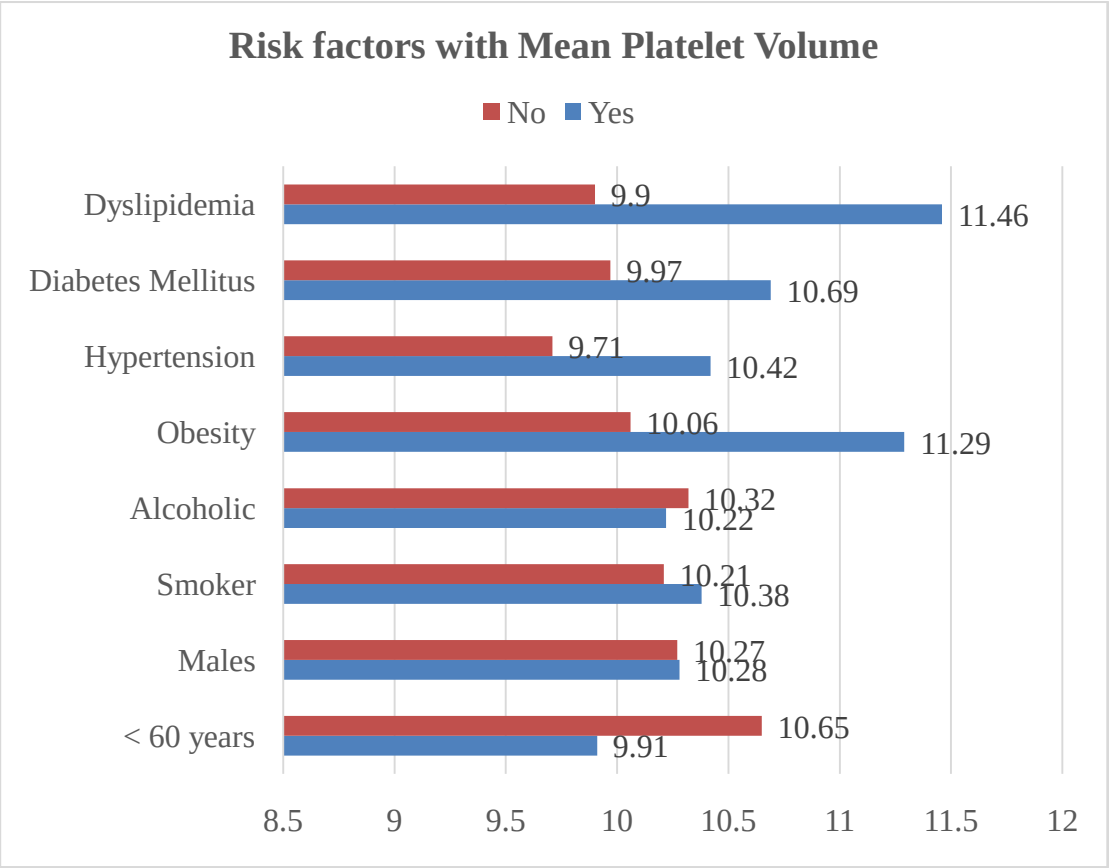
The other risk factors like gender, smoking and alcoholic were not statistically significant with the mean platelet volume distribution.

***Table 28. Risk factors with Mean Platelet Volume***

| | | Mean Platelet Volume (fl) | | | | |
|---|---|---|---|---|---|---|
| | | **N** | **Mean** | **Standard Deviation** | **Mean diff** | **t' test p value** |
| **Age group** | **< 60 years** | 38 | 9.91 | 0.81 | 0.74 | **0.001** |
| | **> 60 years** | 37 | 10.65 | 1.03 | | |
| **Gender** | **Males** | 47 | 10.28 | 0.99 | 0.01 | 0.956 |
| | **Females** | 28 | 10.27 | 1.00 | | |
| **Smoker** | **Yes** | 29 | 10.38 | 0.96 | 0.16 | 0.486 |
| | **No** | 46 | 10.21 | 1.02 | | |

| | | | | | | |
|---|---|---|---|---|---|---|
| **Alcoholic** | **Yes** | 33 | 10.22 | 0.96 | 0.10 | 0.654 |
| | **No** | 42 | 10.32 | 1.03 | | |
| **Obesity** | **Yes** | 13 | 11.29 | 0.91 | 1.23 | **0.001** |
| | **No** | 62 | 10.06 | 0.87 | | |
| **Hypertension** | **Yes** | 60 | 10.42 | 1.03 | 0.70 | **0.001** |
| | **No** | 15 | 9.71 | 0.51 | | |
| **Diabetes Mellitus** | **Yes** | 32 | 10.69 | 1.08 | 0.73 | **0.002** |
| | **No** | 43 | 9.97 | 0.80 | | |
| **Dyslipidemia** | **Yes** | 18 | 11.46 | 0.70 | 1.56 | **0.001** |
| | **No** | 57 | 9.90 | 0.74 | | |

***Figure 34. Risk factors with Mean Platelet Volume***




## *XXV. RISK FACTORS WITH PLATELET DISTRIBUTION WIDTH*

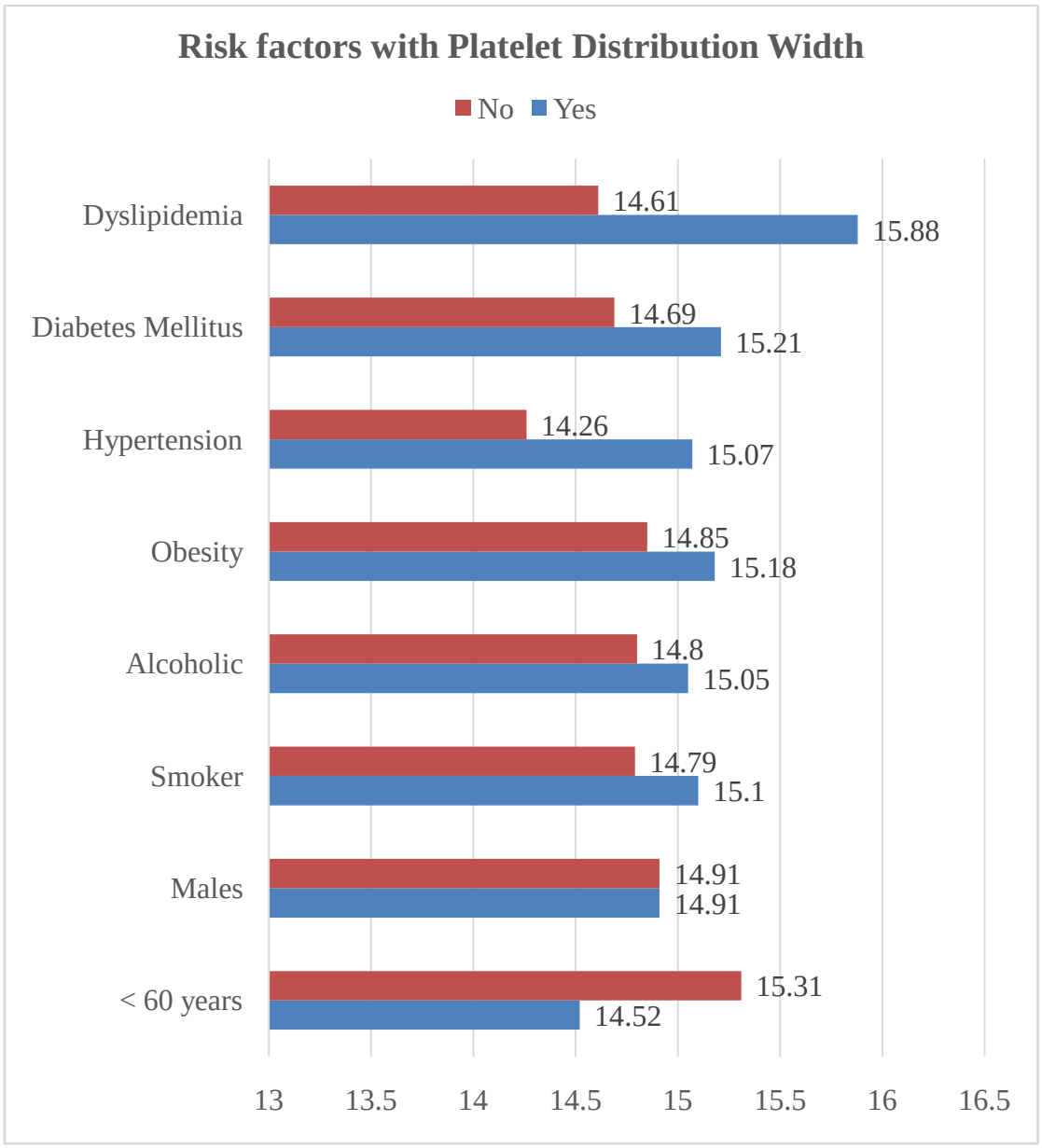
The Platelet Distribution Width among those with higher age group and Dyslipidemia were significantly higher compared to those without the risk factors.

The other risk factors like gender, smoking, alcoholic, diabetes and hypertension were not statistically significant with the Platelet Distribution Width distribution.

*Table 29. Risk factors with Platelet Distribution Width*

| | | Platelet Distribution Width | | | | |
|---|---|---|---|---|---|---|
| | | **N** | **Mean** | **Standard Deviation** | **Mean diff** | **t' test p value** |
| **Age group** | **< 60 years** | 38 | 14.52 | 1.31 | 0.78 | **0.004** |
| | **> 60 years** | 37 | 15.31 | 1.02 | | |
| **Gender** | **Males** | 47 | 14.91 | 1.42 | 0.01 | 0.982 |
| | **Females** | 28 | 14.91 | 0.86 | | |
| **Smoker** | **Yes** | 29 | 15.10 | 1.16 | 0.31 | 0.274 |
| | **No** | 46 | 14.79 | 1.28 | | |
| **Alcoholic** | **Yes** | 33 | 15.05 | 1.12 | 0.24 | 0.395 |
| | **No** | 42 | 14.80 | 1.31 | | |
| **Obesity** | **Yes** | 13 | 15.18 | 1.93 | 0.33 | 0.549 |
| | **No** | 62 | 14.85 | 1.05 | | |
| **Hypertension** | **Yes** | 60 | 15.07 | 1.03 | 0.81 | 0.086 |
| | **No** | 15 | 14.26 | 1.74 | | |
| **Diabetes Mellitus** | **Yes** | 32 | 15.21 | 1.03 | 0.53 | 0.057 |
| | **No** | 43 | 14.69 | 1.33 | | |
| **Dyslipidemia** | **Yes** | 18 | 15.88 | 0.74 | 1.27 | **0.001** |
| | **No** | 57 | 14.61 | 1.20 | | |

***Figure 35. Risk factors with Platelet Distribution Width***



# 8 DISCUSSION

The main objective of the study is to assess the correlation of Mean Platelet Volume (MPV) and Platelet Distribution Width (PDW) and stroke severity and also to find their use as a prognostic factor among patients with acute ischaemic stroke.

The mean Age (years) among the subjects was 60.52 (± 7.33) ranging from 43 to 75 years.Among the subjects, 33 (44%) were in 61 - 70 years, 30 (40%) were in 51 - 60 years and 8 (10.67%) were in < 50 years.Among the subjects, 47 (62.67%) were Males and 28 (37.33%) were Females.Among the subjects, 29 (38.67%) were Smoker, 33 (44%) were Alcoholic.Among the subjects, 60 (80%) had Hypertension.Among the subjects, 32 (42.67%) had Diabetes Mellitus. Kamat et al [105]conducted a study on 60 patients with ischemic stroke and found that 40% of the study patients were aged between 60-80 years and 60% of study population were male and 40% female. The co-morbid conditions present were hypertension in 50%, diabetes mellitus in 41%

The mean Mean Platelet Volume (fl) among the subjects was 10.28 (± 0.99) fl ranging from 8.7 to 12.4 fl.The mean Platelet Distribution Width among the subjects was 14.91 (± 1.23) ranging from 9 to 18.1%.Among the subjects, 40 (53.33%) had MRS Score 3 - 6 and 35 (46.67%) had MRS Score 0 – 2 on day 1.Among the subjects, 46 (61.33%) had MRS Score 3 - 6 and 29 (38.67%) had MRS Score 0 – 2 on day 7.

The mean Mean Platelet Volume (fl) with MRS score day 1showed that among Score 0 - 2 MPV was 9.83 (± 0.72) which is lower by 0.84 and statistically significant compared to MPV 10.67 (± 1.04) in Score 3 – 6.The mean Mean Platelet Volume (fl) with MRS score

at day 7, among Score 0 - 2 MPV was 9.52 (± 0.47) which is lower by 1.23 and statistically significant compared to MPV 10.75 (± 0.94) in Score 3 – 6.

The mean Platelet Distribution Width with MRS score day 1, among Score 0 – 2 PDW was 14.81 (± 1.11) which is lower by 0.2 but not statistically significant compared to PDW 15 (± 1.34) in Score 3 – 6.The mean Platelet Distribution Width with MRS score day 7, among Score 0 - 2 PDW was 14.47 (± 0.91) which is lower by 0.71 and statistically significant compared to PDW 15.19 (± 1.33) in Score 3 – 6.

92.3% of the obese subjects, and 83.3% of the subjects with dyslipidemia had MRS score 3 – 6 with higher severity of stroke and the differences were statistically significant. The Mean Platelet Volume among those with Hypertension, Diabetes and Dyslipidemia were significantly higher compared to those without the risk factors. Subjects with more than 60 years of age and obese subjects had significantly higher mean platelet volume and the differences were statistically significant.The Platelet Distribution Width among those with higher age group and Dyslipidemia were significantly higher compared to those without the risk factors.

Zheng et al [73] did a systematic review and meta-analysis to explore the effect of MPV, PDW on clinical outcomes in Acute Ischaemic Stroke (AIS) and suggested that elevated MPV may be a predictive marker of adverse clinical outcome of AIS, especially in non-thrombolytic patients, while PDW has insufficient value in predicting clinical outcome of AIS.Yao et al[104] found a correlation between elevated MPV and worse outcome at 3 months, particularly in large-artery atherosclerosis stroke patients. MPV and platelet count are negative correlated ($r = -0.375$, $p = 0.000$). MPV and platelet-to-lymphocyte ratio (PLR) ($r = 0.83$, $p = 0.000$), MPV and platelet distribution width (PDW) ($r = 0.820$, $p = 0.000$) both have highly positive linear correlations in patients with good outcome. Sotero

et al[63]found that there was a trend to unfavourable prognosis at 90 days in patients with higher MPV. And higher PDW was associated with more severe haemorrhagic transformation.Kamat et al [105] showed that MPV showed statistically significant correlation with Ischemic stroke severity. Shubham Sharma and Somya Goyal[106]found that Diabetic patients with ischemic stroke had higher PDW , MPV & PCT and were associated with poor outcome.Govind D et al [107]found a positive correlation with Platelet to Lymphocyte ratio (PLR), and National Institute of Health Stroke Scale (NIHSS). Punekar and Shukla [108] showed that there was direct relationship of MPV, PC and PLCR with severity of ischemic stroke and inverse relationship of PDW and PCT with severity of ischemic stroke. PC and PLCR were higher in patient with ischemic stroke but PC showed not significant change whereas PLCR showed significant abnormal changes in ischemic stroke patient.Mohammed et al [69]found that MPV was significantly higher in the unfavourable group (10.4 ± 2.3 fL) than in the favourable one (8.7 ± 1.3 fL) (P < 0. 001). The mean PCT was significantly higher in the unfavourable group (0.28 ± 0.1%) than in the favourable one (0.25 ± 0.1%) (P = 0. 04) but not considered as an independent predictor of poor short-term outcome of acute stroke.Gao et al[109]found that PDW was significantly higher for a good outcome than a poor outcome (p=0.005), with median (interquartile range) values of 16.2 (13.2–17.2) and 13.6 (12.5–15.9), respectively. PDW was also higher in patients with early neurological improvement than in patients without improvement (p=0.020) and did not differ between haemorrhage and non haemorrhage patients. The association between PDW ,16.05% and poor outcome remained in a multivariable logistic regression analysis, with an OR of 6.68 and a 95% CI of 1.69–26.49 (p=0.007). Vyawahare and Donge [110]found that incidence of hypertension, diabetes, alcohol, obesity, dyslipidaemias well as mean MPV, PDW, P-lcr and platelet count were more in ischaemic stroke cases than controls. Platelet indices increased in severe stroke,

amongst MPV and platelet count increase was statistically significant. 25% mortality observed in studied cases. Sarkar et al[101]found that mean value for PDW & MPV in 1st group(with comprehensive deficit) was 18.675 ± 3.494 & 12.894 ± 1.270 respectively and in 2nd group (without comprehensive deficit) was 18.62 ± 3.387 & 12.42 ± 0.984 respectively and was significantly higher than mean value of 15.694 ± 3.127 & 10.46 ± 1.273 of PDW & MPV respectively in controls. Meena et al [111] showed significant in reduction in Mean RBC, increase in Mean HB , increase in mean MPV and reduction in mean platelet count among the ischaemic cases and controls. Ugur Lok et al[112]found no differences in MPV levels between the MRS based groups (p=0.527).Al Tameemi et al [113]found that the mean platelet count (MPC) was found to show significant difference between group 2 (more than one IS) versus group 1 (First IS) and control (P=0.012, P=0.023 respectively). Linear negative correlation was demonstrated between MPC and MPV, PDW and P-LCR in group having first Ischaemic Stroke. O' Malley et al (1995)[100]found that mean platelet volume was higher in acute stroke (11.3 compared with 10.1 fL in control subjects; P<.001). In addition, platelet count was reduced in stroke patients (255×109/L) compared with control subjects (299×109/L; P<.01).

According to a study by Greisenegger et al., patients in the highest quintile of MPV were significantly more likely than those in the lowest quintile to experience a severe stroke, which is defined as a modified Rankin Scale score of 3 to 6.[115]Sadeghi et alsystematic .'s review and meta-analysis of 27 research demonstrated that patients with stroke had higher MPV than controls in 17 of the trials. In contrast to two research that showed a larger MPV in the control group, ten investigations found no difference in MPV between patients and controls.[116]Shah et al showed that there was significant correlation between MPV and the degree of glycaemic control only in diabetic patients.[117]

# 9 LIMITATIONS

Confounding factors like RBC count, Platelet Count, Neutrophil-Lymphocyte Ratio were not studied

Smaller sample size decreases the chance of generalisability of study

Hospital based study in a tertiary care setting may cause bias as multi-morbidity patients with stroke present mainly in tertiary care hospital skewing the outcome and prognosis.

We did not follow up the case after discharge for total outcome of the patient in terms of morbidity

# 10 RECOMMENDATIONS

Further studies with increased sample size matched for confounding factors done also in other settings such as primary and secondary care will represent the true nature of findings. MPV and PDW should be encouraged as a predictive factor and prognostic tool in the severity of ischemic stroke or mortality.

# 11 CONCLUSION

Based on MRS (3-6), higher MPVs and PDW are linked to more severe strokes and have worse outcomes in terms of morbidity and mortality. Diabetes patients had considerably higher MPV levels, and because DM is a procoagulant condition, individuals are more likely to experience thrombotic events. Mean platelet volume and Platelet Distribution Width, a measure of platelet reactivity, is a quick and uncomplicated test that is included in a panel of hemograms and can be a useful indicator of the severity and prognosis in patients with acute ischemic stroke.

# 12 SUMMARY OF RESULTS

- The mean Age (years) among the subjects was 60.52 (± 7.33) ranging from 43 to 75 years.
- Among the subjects, 33 (44%) were in 61 - 70 years, 30 (40%) were in 51 - 60 years and 8 (10.67%) were in < 50 years.
- Among the subjects, 47 (62.67%) were Males and 28 (37.33%) were Females.
- Among the subjects, 29 (38.67%) were Smoker, 33 (44%) were Alcoholic.
- The mean Height (cm) among the subjects was 164.29 (± 8.08) cm ranging from 150 to 185 cm. The mean Weight (kg) among the subjects was 71.27 (± 7.81) kg ranging from 55 to 87.5 kg.
- The mean BMI (kg/m2) among the subjects was 26.4 (± 2.7) kg/m2 ranging from 21.5 to 32.2 $kg/m^2$.
- Among the subjects, 60 (80%) had Hypertension. The mean Systolic Blood Pressure among the subjects was 154.85 (± 12.82) mm Hg ranging from 128 to 184 mm Hg. The mean Diastolic Blood Pressure among the subjects was 85.63 (± 6.99) mm Hg ranging from 70 to 106 mm Hg.
- Among the subjects, 32 (42.67%) had Diabetes Mellitus
- The mean Fasting Blood Sugar (mg/dl) among the subjects was 110.31 (± 21) mg/dl ranging from 86 to 151 mg/dl. The mean Post Prandial Blood Sugar (mg/dl) among the subjects was 175.73 (± 45.75) mg/dl ranging from 117 to 287 mg/dl.
- The mean HbA1C among the subjects was 7.04 (± 0.62) ranging from 6 to 8.5%.
- The mean Mean Platelet Volume (fl) among the subjects was 10.28 (± 0.99) fl ranging from 8.7 to 12.4 fl.

- The mean Platelet Distribution Width among the subjects was 14.91 (± 1.23) ranging from 9 to 18.1%.
- Among the subjects, 40 (53.33%) had MRS Score 3 - 6 and 35 (46.67%) had MRS Score 0 – 2 on day 1. Among the subjects, 46 (61.33%) had MRS Score 3 - 6 and 29 (38.67%) had MRS Score 0 – 2 on day 7.
- The mean Mean Platelet Volume (fl) with MRS score day 1showed that among Score 0 - 2 MPV was 9.83 (± 0.72) which is lower by 0.84 and statistically significant compared to MPV 10.67 (± 1.04) in Score 3 – 6.
- The mean Mean Platelet Volume (fl) with MRS score at day 7, among Score 0 - 2 MPV was 9.52 (± 0.47) which is lower by 1.23 and statistically significant compared to MPV 10.75 (± 0.94) in Score 3 – 6.
- The mean Platelet Distribution Width with MRS score day 1, among Score 0 – 2 PDW was 14.81 (± 1.11) which is lower by 0.2 but not statistically significant compared to PDW 15 (± 1.34) in Score 3 – 6.The mean Platelet Distribution Width with MRS score day 7, among Score 0 - 2 PDW was 14.47 (± 0.91) which is lower by 0.71 and statistically significant compared to PDW 15.19 (± 1.33) in Score 3 – 6.
- 92.3% of the obese subjects, and 83.3% of the subjects with dyslipidemia had MRS score 3 – 6 with higher severity of stroke and the differences were statistically significant. The other risk factors like age, gender, smoking, alcoholic, hypertension and diabetes mellitus was not statistically significant with the MRS score distribution.
- The Mean Platelet Volume among those with Hypertension, Diabetes and Dyslipidemia were significantly higher compared to those without the risk factors. Subjects with more than 60 years of age and obese subjects had significantly higher

mean platelet volume and the differences were statistically significant. The other risk factors like gender, smoking and alcoholic were not statistically significant with the mean platelet volume distribution.

- The Platelet Distribution Width among those with higher age group and Dyslipidemia were significantly higher compared to those without the risk factors. The other risk factors like gender, smoking, alcoholic, diabetes and hypertension were not statistically significant with the Platelet Distribution Width distribution.

# 13 REFERENCES


1. World Health Organization. Global status report on noncommunicable diseases 2014. 280 p.

2. Katan M, Luft A. Global Burden of Stroke. Seminars in Neurology. 2018 Apr 23;38(02):208–11.

3. Kasper DL, Fauci AS, Hauser SL, Longo DL (Dan L, Jameson JL, Loscalzo J. Harrison's principles of internal medicine.

4. Edwardson MA, Dromerick AW. Ischemic stroke prognosis in adults - UpToDate. In: UpToDate. 2018.

5. Bhatia RS, Garg RK, Gaur SPS, Kar AM, Shukla R, Agarwal A, et al. Predictive value of routine hematological and biochemical parameters on 30-day fatality in acute stroke. Neurology India. 2004 Jun;52(2):220–3.

6. Kannan R, Alex AG, Sudeep K. A clinical study of factors predicting prognosis in acute ischemic stroke in a tertiary care center in north Kerala. International Journal of Research in Medical Sciences Kannan R et al Int J Res Med Sci. 2017;5(10):4344–7.

7. Young AR, Ali C, Duretête A, Vivien D. Neuroprotection and stroke: Time for a compromise. Vol. 103, Journal of Neurochemistry. J Neurochem; 2007. p. 1302–9.

8. WHO | Global health agenda. WHO. 2016;

9. Owolabi M, Arnett D, Akinyemi R, Arulogun O, Gebregziabher M, Jenkins C, et al. The burden of stroke in Africa: a glance at the present and a glimpse into the future: review article. Cardiovascular Journal Of Africa. 2015;26(2):S27–38.

10. Feigin VL, Forouzanfar MH, Krishnamurthi R, Mensah GA, Connor M, Bennett DA, et al. Global and regional burden of stroke during 1990-2010: Findings from the Global Burden of Disease Study 2010. The Lancet. 2014 Jan 18;383(9913):245–55.

11. Strong K, Mathers C, Bonita R. Preventing stroke: saving lives around the world. Vol. 6, Lancet Neurology. Elsevier; 2007. p. 182–7.

12. Roger VL, Go AS, Lloyd-Jones DM, Adams RJ, Berry JD, Brown TM, et al. Heart Disease and Stroke Statistics—2011 Update. Circulation. 2011 Feb 1;123(4):e18–209.

13. Global, regional, and national burden of stroke, 1990–2016: a systematic analysis for the Global Burden of Disease Study 2016. Lancet Neurol. 2019 May;18(5):439–58.

14. Pandian JD, Sudhan P. Stroke Epidemiology and Stroke Care Services in India. Journal of Stroke. 2013 Sep;15(3):128.

15. Tapas Kumar, BANERJEE, Shyamal Kumar D. Epidemiology of stroke in Chile. Neurology Asia. 2006;11(4):1–4.

16. Coupland AP, Thapar A, Qureshi MI, Jenkins H, Davies AH. The definition of stroke. Journal of the Royal Society of Medicine. 2017 Jan 13;110(1):9–12.

17. Aho K, Harmsen P, Hatano S, Marquardsen J, Smirnov VE, Strasser T. Cerebrovascular disease in the community: results of a WHO collaborative study. Bulletin of the World Health Organization. 1980;58(1):113–30.

18. Sacco RL, Kasner SE, Broderick JP, Caplan LR, Connors JJ, Culebras A, et al. An Updated Definition of Stroke for the 21st Century: A Statement for Healthcare Professionals From the American Heart Association/American Stroke Association. Stroke. 2013 Jul 1;44(7):2064–89.

19. Sacco RL, Kasner SE, Broderick JP, Caplan LR, Connors JJB, Culebras A, et al. An updated definition of stroke for the 21st century: a statement for healthcare professionals from the American Heart Association/American Stroke Association. Stroke. 2013 Jul;44(7):2064–89.

20. Bamford J, Sandercock P, Dennis M, Burn J, Warlow C. Classification and natural history of clinically identifiable subtypes of cerebral infarction. Lancet (London, England). 1991 Jun;337(8756):1521–6.

21. Caplan's Stroke: A Clinical Approach. 5th ed. Cambridge: Cambridge University Press; 2016.

22. Ingall TJ. Neurology in Clinical Practice: Principles of Diagnosis and Management. Mayo Clinic Proceedings. 1997;72(3):289–90.

23. Causes of Ischemic Stroke - Differential Diagnosis Algorithm Thrombosis - Atherosclerosis, Arterial Dissection, [Internet]. [cited 2020 Sep 12]. Available from: https://www.grepmed.com/images/8522/differential-diagnosis-neurology-algorithm-ischemic-causes-stroke

24. Kuriakose D, Xiao Z. Pathophysiology and Treatment of Stroke: Present Status and Future Perspectives. Int J Mol Sci. 2020 Oct 15;21(20):7609.

25. Elkind MS, Sacco RL. Stroke risk factors and stroke prevention. Semin Neurol. 1998;18(4):429–40.

26. Coco D Lo, Lopez G, Corrao S. Cognitive impairment and stroke in elderly patients. Vol. 12, Vascular Health and Risk Management. Dove Medical Press Ltd.; 2016. p. 105–16.

27. Stroke: Pathogenesis | Calgary Guide [Internet]. [cited 2020 Sep 12]. Available from: https://calgaryguide.ucalgary.ca/ischemic-stroke-pathogenesis/

28. Wang J, Fields J, Zhao C, Langer J, Thimmulappa RK, Kensler TW, et al. Role of Nrf2 in Protection against Intracerebral Hemorrhage Injury in Mice. Free Radic Biol Med. 2007 Aug 1;43(3):408–14.

29. Broughton BRS, Reutens DC, Sobey CG. Apoptotic mechanisms after cerebral ischemia. Stroke. 2009 May;40(5):e331-339.

30. Musuka TD, Wilton SB, Traboulsi M, Hill MD. Diagnosis and management of acute ischemic stroke: speed is critical. CMAJ. 2015 Sep 8;187(12):887–93.

31. Qureshi AI, Ali Z, Suri MFK, Shuaib A, Baker G, Todd K, et al. Extracellular glutamate and other amino acids in experimental intracerebral hemorrhage: an in vivo microdialysis study. Crit Care Med. 2003 May;31(5):1482–9.

32. Gelderblom M, Leypoldt F, Steinbach K, Behrens D, Choe CU, Siler DA, et al. Temporal and spatial dynamics of cerebral immune cell accumulation in stroke. Stroke. 2009 May;40(5):1849–57.

33. Ferro JM, Massaro AR, Mas JL. Aetiological diagnosis of ischaemic stroke in young adults. The Lancet Neurology. 2010;9(11):1085–96.

34. Merino JG, Warach S. Imaging of acute stroke. Nature reviews Neurology. 2010 Oct;6(10):560–71.

35. Birenbaum D, Bancroft LW, Felsberg GJ. Imaging in acute stroke. The western journal of emergency medicine. 2011 Feb;12(1):67–76.

36. ACLS Stroke Protocol | Learn & Master ACLS/PALS [Internet]. [cited 2020 Sep 12]. Available from: https://acls-algorithms.com/adult-stroke-algorithm/step-3-acls-stroke-protocol

37. Hui C, Tadi P, Patti L. Ischemic Stroke. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2022 [cited 2022 Dec 11]. Available from: http://www.ncbi.nlm.nih.gov/books/NBK499997/

38. Campbell BC V, De Silva DA, Macleod MR, Coutts SB, Schwamm LH, Davis SM, et al. Ischaemic stroke. Nature Reviews Disease Primers. 2019;5(1):70.

39. George PM, Steinberg GK. Novel Stroke Therapeutics: Unraveling Stroke Pathophysiology and Its Impact on Clinical Treatments. Neuron. 2015 Jul;87(2):297–309.

40. Savoiardo M. The vascular territories of the carotid and vertebrobasilar systems. Diagrams based on CT studies of infarcts. Italian journal of neurological sciences. 1986 Aug;7(4):405–9.

41. Adams HPJ, Bendixen BH, Kappelle LJ, Biller J, Love BB, Gordon DL, et al. Classification of subtype of acute ischemic stroke. Definitions for use in a multicenter clinical trial. TOAST. Trial of Org 10172 in Acute Stroke Treatment. Stroke. 1993 Jan;24(1):35–41.

42. Chung J, Park SH, Kim N, Kim W, Park JH, Ko Y, et al. Trial of ORG 10172 in Acute Stroke Treatment (TOAST) Classification and Vascular Territory of Ischemic Stroke Lesions Diagnosed by Diffusion-Weighted Imaging. Journal of the American Heart Association. 3(4):e001119.

43. Prasad K, Kaul S, Padma MV, Gorthi SP, Khurana D, Bakshi A. Stroke management. Ann Indian Acad Neurol. 2011 Jul;14(Suppl1):S82–96.

44. Herpich F, Rincon F. Management of Acute Ischemic Stroke. Crit Care Med. 2020 Nov;48(11):1654–63.

45. Phipps MS, Cronin CA. Management of acute ischemic stroke. BMJ. 2020 Feb 13;368:l6983.

46. Hollist M, Morgan L, Cabatbat R, Au K, Kirmani MF, Kirmani BF. Acute Stroke Management: Overview and Recent Updates. Aging Dis. 2021 Jul;12(4):1000–9.

47. Chugh C. Acute Ischemic Stroke: Management Approach. Indian J Crit Care Med. 2019 Jun;23(Suppl 2):S140–6.

48. Pérez de la Ossa N, Carrera D, Gorchs M, Querol M, Millán M, Gomis M, et al. Design and validation of a prehospital stroke scale to predict large arterial occlusion: the rapid arterial occlusion evaluation scale. Stroke. 2014 Jan;45(1):87–91.

49. Muir KW. Medical management of stroke. Journal of Neurology Neurosurgery and Psychiatry. 2001 Apr 1;70(SUPPL. 1):112–6.

50. Prabhakaran S, Chang P. Recent advances in the management of acute ischemic stroke. Vol. 6, F1000Research. Faculty of 1000 Ltd; 2017.

51. CAST: randomised placebo-controlled trial of early aspirin use in 20,000 patients with acute ischaemic stroke. CAST (Chinese Acute Stroke Trial) Collaborative Group. Lancet (London, England). 1997 Jun;349(9066):1641–9.

52. The International Stroke Trial (IST): a randomised trial of aspirin, subcutaneous heparin, both, or neither among 19435 patients with acute ischaemic stroke. International Stroke Trial Collaborative Group. Lancet (London, England). 1997 May;349(9065):1569–81.

53. Sacco RL, Diener HC, Yusuf S, Cotton D, Ôunpuu S, Lawton WA, et al. Aspirin and Extended-Release Dipyridamole versus Clopidogrel for Recurrent Stroke. New England Journal of Medicine. 2008 Sep 18;359(12):1238–51.

54. Johnston SC, Easton JD, Farrant M, Barsan W, Conwit RA, Elm JJ, et al. Clopidogrel and Aspirin in Acute Ischemic Stroke and High-Risk TIA. The New England journal of medicine. 2018/05/16 ed. 2018 Jul 19;379(3):215–25.

55. Johnston SC, Elm JJ, Easton JD, Farrant M, Barsan WG, Kim AS, et al. Time Course for Benefit and Risk of Clopidogrel and Aspirin After Acute Transient Ischemic Attack and Minor Ischemic Stroke. Circulation. 2019 Aug;140(8):658–64.

56. Junling D, Fajun W, Sophia S. Use of Dual Antiplatelet Therapy Following Ischemic Stroke. Stroke. 2020 May 1;51(5):e78–80.

57. Demaerschalk BM, Kleindorfer DO, Adeoye OM, Demchuk AM, Fugate JE, Grotta JC, et al. Scientific Rationale for the Inclusion and Exclusion Criteria for Intravenous Alteplase in Acute Ischemic Stroke: A Statement for Healthcare Professionals From the American Heart Association/American Stroke Association. Stroke. 2016 Feb;47(2):581–641.

58. Powers WJ, Rabinstein AA, Ackerson T, Adeoye OM, Bambakidis NC, Becker K, et al. 2018 Guidelines for the Early Management of Patients With Acute Ischemic Stroke: A Guideline for Healthcare Professionals From the American Heart Association/American Stroke Association. Stroke. 2018 Mar;49(3):e46–110.

59. Davignon J. Beneficial cardiovascular pleiotropic effects of statins. Circulation. 2004;109(23_suppl_1):III–39.

60. Tramacere I, Boncoraglio GB, Banzi R, Del Giovane C, Kwag KH, Squizzato A, et al. Comparison of statins for secondary prevention in patients with ischemic stroke or transient ischemic attack: a systematic review and network meta-analysis. BMC Medicine. 2019;17(1):67.

61. Stroke: Physiotherapy Treatment Approaches - Physiopedia [Internet]. [cited 2020 Oct 3]. Available from: https://www.physio-pedia.com/Stroke:_Physiotherapy_Treatment_Approaches

62. Vagdatli E, Gounari E, Lazaridou E, Katsibourlia E, Tsikopoulou F, Labrianou I. Platelet distribution width: a simple, practical and specific marker of activation of coagulation. Hippokratia. 2010;14(1):28–32.

63. Dourado Sotero F, Calçada A, Aguiar de Sousa D, Dias M, Fonseca AC, Pinho e Melo T, et al. Mean Platelet Volume is a Prognostic Marker in Acute Ischemic Stroke Patients Treated with Intravenous Thrombolysis. Journal of Stroke and Cerebrovascular Diseases. 2021 Jun 1;30(6):105718.

64. Korniluk A, Koper-Lenkiewicz OM, Kamińska J, Kemona H, Dymicka-Piekarska V. Mean Platelet Volume (MPV): New Perspectives for an Old Marker in the Course and Prognosis of Inflammatory Conditions. Mediators of Inflammation [Internet]. 2019 [cited 2022 Dec 11];2019. Available from: https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6501263/

65. Golebiewska EM, Poole AW. Platelet secretion: From haemostasis to wound healing and beyond. Blood Rev. 2015 May;29(3):153–62.

66. Zhang S, Cui YL, Diao MY, Chen DC, Lin ZF. Use of Platelet Indices for Determining Illness Severity and Predicting Prognosis in Critically Ill Patients. Chin Med J (Engl). 2015 Aug 5;128(15):2012–8.

67. Budak YU, Polat M, Huysal K. The use of platelet indices, plateletcrit, mean platelet volume and platelet distribution width in emergency non-traumatic abdominal surgery: a systematic review. Biochem Med (Zagreb). 2016 Jun 10;26(2):178–93.

68. Bessman JD, Gilmer PR, Gardner FH. Use of mean platelet volume improves detection of platelet disorders. Blood Cells. 1985;11(1):127–35.

69. Mohamed AAB, Elnady HM, Alhewaig HK, Moslem Hefny H, Khodery A. The mean platelet volume and plateletcrit as predictors of short-term outcome of acute ischemic stroke. Egypt J Neurol Psychiatr Neurosurg. 2019;55(1):4.

70. Bessman JD, Williams LJ, Gilmer PR. Mean platelet volume. The inverse relation of platelet size and count in normal subjects, and an artifact of other particles. Am J Clin Pathol. 1981 Sep;76(3):289–93.

71. Park Y, Schoene N, Harris W. Mean platelet volume as an indicator of platelet activation: methodological issues. Platelets. 2002;13(5–6):301–6.

72. Korniluk A, Koper-Lenkiewicz OM, Kamińska J, Kemona H, Dymicka-Piekarska V. Mean Platelet Volume (MPV): New Perspectives for an Old Marker in the Course and Prognosis of Inflammatory Conditions. Mediators Inflamm. 2019;2019:9213074.

73. Zheng YY, Wang L, Shi Q. Mean platelet volume (MPV) and platelet distribution width (PDW) predict clinical outcome of acute ischemic stroke: A systematic review and meta-analysis. J Clin Neurosci. 2022 Jul;101:221–7.

74. Demirin H, Ozhan H, Ucgun T, Celer A, Bulur S, Cil H, et al. Normal range of mean platelet volume in healthy subjects: Insight from a large epidemiologic study. Thromb Res. 2011 Oct;128(4):358–60.

75. Bae MH, Lee JH, Yang DH, Park HS, Cho Y, Chae SC. White blood cell, hemoglobin and platelet distribution width as short-term prognostic markers in patients with acute myocardial infarction. Journal of Korean Medical Science. 2014;29(4):519–26.

76. Sevuk U, Bahadir MV, Altindag R, Baysal E, Yaylak B, Ay N, et al. Value of serial platelet indices measurements for the prediction of pulmonary embolism in patients with deep venous thrombosis. Therapeutics and Clinical Risk Management. 2015;11:1243.

77. Zhang S, Cui YL, Diao MY, Chen DC, Lin ZF. Use of platelet indices for determining illness severity and predicting prognosis in critically ill patients. Chinese medical journal. 2015;128(15):2012–8.

78. Khandekar M, Khurana A, Deshmukh S, Kakrani A, Katdare A, Inamdar A. Platelet volume indices in patients with coronary artery disease and acute myocardial infarction: an Indian scenario. Journal of clinical pathology. 2006;59(2):146–9.

79. Bülbül Y, Aydin Özgür E, Örem A. Platelet indices in obstructive sleep apnea: the role of mean platelet volume, platelet distribution widht and plateletcrit. Tuberk Toraks. 2016;64(3):206–10.

80. Fu S, Liu L, Zhang X, Liu ZP, Wang RT. Platelet indices in laryngeal cancer. Cancer Biomarkers. 2018;21(3):675–80.

81. Vagdatli E, Gounari E, Lazaridou E, Katsibourlia E, Tsikopoulou F, Labrianou I. Platelet distribution width: a simple, practical and specific marker of activation of coagulation. Hippokratia. 2010;14(1):28.

82. Tzur I, Barchel D, Izhakian S, Swarka M, Garach-Jehoshua O, Krutkina E, et al. Platelet distribution width: a novel prognostic marker in an internal medicine ward. Journal of Community Hospital Internal Medicine Perspectives. 2019 Nov 2;9(6):464–70.

83. Wang F, Meng Z, Li S, Zhang Y, Wu H. Platelet Distribution Width Levels Can Be a Predictor in the Diagnosis of Persistent Organ Failure in Acute Pancreatitis. Gastroenterology Research and Practice. 2017 Dec 27;2017:e8374215.

84. Kern A, Gil RJ, Bojko K, Sienkiewicz E, Januszko-Giergielewicz B, Górny J, et al. Platelet distribution width as the prognostic marker in coronary bifurcation treatment. European Journal of Clinical Investigation. 2017;47(7):524–30.

85. Białas AJ, Pedone C, Piotrowski WJ, Incalzi RA. Platelet distribution width as a prognostic factor in patients with COPD–pilot study. International Journal of Chronic Obstructive Pulmonary Disease. 2017;12:2261.

86. Artunc Ulkumen B, Pala HG, Calik E, Oruc Koltan S. Platelet distribution width (PDW): A putative marker for threatened preterm labour. Pak J Med Sci. 2014;30(4):745–8.

87. Sezgi C, Taylan M, Kaya H, Selimoglu Sen H, Abakay O, Demir M, et al. Alterations in platelet count and mean platelet volume as predictors of patient outcome in the respiratory intensive care unit. The clinical respiratory journal. 2015;9(4):403–8.

88. Yu Y, Li N, Yun Z, Niu Y, Xu J, Liu Z, et al. Preoperative mean platelet volume and platelet distribution associated with thyroid cancer. Neoplasma. 2017;64(4):594–8.

89. Rechciński T, Jasińska A, Foryś J, Krzemińska-Pakuła M, Wierzbowska-Drabik K, Plewka M, et al. Prognostic value of platelet indices after acute myocardial infarction treated with primary percutaneous coronary intervention. Cardiology Journal. 2013;20(5):491–8.

90. Takeuchi H, Abe M, Takumi Y, Hashimoto T, Kobayashi R, Osoegawa A, et al. The prognostic impact of the platelet distribution width-to-platelet count ratio in patients with breast cancer. PloS one. 2017;12(12):e0189166.

91. Song X, Zhu H, Pei Q, Tan F, Li C, Zhou Z, et al. Significance of inflammation-based indices in the prognosis of patients with non-metastatic colorectal cancer. Oncotarget. 2017;8(28):45178.

92. Zhang H, Liu L, Fu S, Liu YS, Wang C, Liu T, et al. Higher platelet distribution width predicts poor prognosis in laryngeal cancer. Oncotarget. 2017;8(29):48138.

93. Araz O, Albez FS, Ucar EY, Kerget B, Yılmaz N, Akgun M. Predictive value of mean platelet volume for pulmonary embolism recurrence. Lung. 2017;195(4):497–502.

94. Xie X, Zeng X, Cao S, Hu X, Shi Q, Li D, et al. Elevated pretreatment platelet distribution width and platelet count predict poor prognosis in nasopharyngeal carcinoma. Oncotarget. 2017;8(62):106089.

95. Khaled SA, NasrEldin E, Makarem YS, Mahmoud HF. Value of Platelet Distribution Width and Mean Platelet Volume in Disease Activity Score of Rheumatoid Arthritis . JIR. 2020 Sep 25;13:595–606.

96. Kaito K, Otsubo H, Usui N, Yoshida M, Tanno J, Kurihara E, et al. Platelet size deviation width, platelet large cell ratio, and mean platelet volume have sufficient sensitivity and specificity in the diagnosis of immune thrombocytopenia. British Journal of Haematology. 2005;128(5):698–702.

97. Farias MG, Schunck EG, Dal Bó S, de Castro SM. Definition of reference ranges for the platelet distribution width (PDW): a local need. Clin Chem Lab Med. 2010 Feb;48(2):255–7.

98. Guclu E, Durmaz Y, Karabay O. Effect of severe sepsis on platelet count and their indices. Afr Health Sci. 2013 Jun;13(2):333–8.

99. Budak YU, Polat M, Huysal K. The use of platelet indices, plateletcrit, mean platelet volume and platelet distribution width in emergency non-traumatic abdominal surgery: a systematic review. Biochem Med (Zagreb). 2016 Jun 10;26(2):178–93.

100. O'Malley T, Langhorne P, Elton R a., Stewart C. Platelet Size in Stroke Patients. Stroke. 1995 Jun;26(6):995–9.

101. Sarkar RN, Das CK, Bhattacharjee U, Banerjee M. Platelet Indices as a Marker of Severity in Non-diabetic NonHypertensive Acute Ischemic Stroke Patients. J Assoc Physicians India. 2018 Jul;66(7):40–2.

102. Sharda A, Flaumenhaft R. The life cycle of platelet granules. F1000Research [Internet]. 2018 [cited 2022 Dec 11];7. Available from: https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5832915/

103. Gunay-Aygun M, Huizing M, Gahl WA. Molecular Defects that Affect Platelet Dense Granules. Seminars in thrombosis and hemostasis. 2004 Oct;30(5):537.

104. Yao Y, Cao X, Zou R, Wen H, Zhang S, Xu H, et al. Study on the Baseline Factors and Platelet Indices That Predict Outcome of Acute Ischemic Stroke Patients after Thrombolytic Therapy. CED. 2022;51(3):357–64.

105. Kamat V, Kinagi S, Patil A. A study of mean platelet volume in acute ischemic stroke. International Journal of Advances in Medicine. 2021 Jan 27;8(2):214–9.

106. . Shubham Sharma, Somya Goyal. STUDY OF ASSOCIATION OF PLATELET INDICES WITH ACUTE ISCHEMIC STROKE IN PATIENTS WITH TYPE 2 DIABETES MELLITUS – A HOSPITAL BASED STUDY. PARIPEX - INDIAN JOURNAL OF RESEARCH:March 2020: 9(3):22-24 [Internet]. [cited 2022 Dec 11]. Available from: https://www.worldwidejournals.com/paripex/recent_issues_pdf/2020/March/study-of-association-of-platelet-indices-with-acute-ischemic-stroke-in-patients-with-type-2-diabetes-mellitus-a-hospital-based-study_March_2020_1583410918_8500823.pdf

107. Govind DD, Rao DMM, Sudheer DK. A Study on the correlation between platelet indices and severity score in acute ischemic stroke. International Journal of Medical Research and Review. 2020 Oct 31;8(5):363–8.

108. Department of Medicine, Netaji Subhash Chandra Bose Medical College, Jabalpur, (M.P), Punekar DJ. Study of Platelet Indices in Ischemic Stroke Patients. jmscr

[Internet]. 2019 Nov 9 [cited 2022 Dec 11];7(11). Available from: http://jmscr.igmpublication.org/v7-i11/42%20jmscr.pdf

109. Gao F, Chen C, Lyu J, Zheng J, Ma XC, Yuan XY, et al. Association between platelet distribution width and poor outcome of acute ischemic stroke after intravenous thrombolysis. Neuropsychiatr Dis Treat. 2018 Sep 3;14:2233–9.

110. Vyawahare M, Dhonge O. Correlation of platelet indices with severity and clinical outcome in patients with acute ischemic stroke. International Journal of Biomedical and Advance Research. 2018 Oct 24;9(10):338–44.

111. Meena P, Khare M, Airun A, Manohar, Saxena A. A Study of Platelet Indices in Patients of Acute Ischemic Stroke :: A Prospective Study. IOSR Journal of Dental and Medical Sciences. 2017 Aug 1;16:26–9.

112. Lok U, Gulacti U, Ekmekci B, Bulut T, Celik M. Predictive and prognostic role of mean platelet volume in patients with first-ever acute ischemic stroke. Neurosciences (Riyadh). 2017 Apr;22(2):119–26.

113. Al Tameemi W, Cabms D, Fibms F, Ameen A, Mbchb. Significance of Platelet Indices in Patients with Acute Ischemic Stroke. The Iraqi Journal of Agricultural Sciences. 2012 Sep 1;10.

114. Broderick JP, Adeoye O, Elm J. The Evolution of the Modified Rankin Scale and Its Use in Future Stroke Trials. Stroke. 2017 Jul;48(7):2007–12.

115. Greisenegger S, Endler G, Hsieh K, Tentschert S, Mannhalter C, Lalouschek W. Is elevated mean platelet volume associated with a worse outcome in patients with acute ischemic cerebrovascular events? Stroke. 2004 Jul;35(7):1688–91.

116. Sadeghi F, Kovács S, Zsóri KS, Csiki Z, Bereczky Z, Shemirani AH. Platelet count and mean volume in acute stroke: a systematic review and meta-analysis. Platelets. 2020 Aug 17;31(6):731–9.

117. Shah AB, Beamer N, Coull BM. Enhanced in vivo platelet activation in subtypes of ischemic stroke. Stroke. 1985;16(4):643–7.

# 14 ANNEXURES

## I – QUESTIONNAIRE

Name

Age/sex

Occupation

Education

Address

Date of admission

Date of discharge

Complaints

History of presenting illness

Past history

Treatment history

General examination

Vitals

Anthropometry

Systemic examination

Lab investigations

Imaging studies

## II- Consent Form

Participants name :

Address :

Title of the study:

A PROSPECTIVE STUDY OF ROLE OF MEAN PLATELET VOLUME, PLATELET DISTRIBUTION WIDTH AS PROGNOSTIC MARKERS IN ACUTE ISCHEMIC STROKE PATIENTS.

The details of the study have been provided to me in writing and explained to me in my own language. I confirm that I have understood the above study and had the opportunity to ask questions. I understand that my participation in the study is voluntary and that I am free to withdraw at any time without giving any reason, without the medical care that will normally be provided by the hospital being affected. I agree not to restrict the use of any data or results that arise from this study provided such a use is only for scientific purposes.i have been given an information sheet giving details of the study. I fully consent to participate in the abovestudy

Signature of theparticipant: Date:

Signature of thewitness: Date:

Signature of theinvestigator : Date:

நோயாளியின்ஒப்புதல்படிவம்

நோயாளியின்பெயர் :

முகவரி :

1 .மேற்கூறியஆய்வினைபற்றிஎழுத்துமூலமாகவும் ,
என்சொந்தமொழியிலும்முழுவிவரம்அறிந்துகொண்டேன்.

2.இந்தஆய்வில்நான்பங்கேற்பதற்கானதேவையைபற்றியும்,
எந்தசமயத்திலும்ஆய்வில்இருந்துவெளியேறமுடியும்என்றும்தெரிந்து
கொண்டேன்.

3.ஆய்வின்போதுபெறப்படும்எனதுதகவல்கள்மற்றும்மருத்துவக்குறிப்
புகள்அனைத்தையும்எந்தவிததடையும்இன்றிஆராய்ச்சிக்குபயன்படு
த்திகொள்ளலாம்.

4. மேற்கூறியஆய்விற்குஎனதுமுழுசம்மந்தம்தெரிவிக்கிறேன்.

நோயாளியின்கையொப்பம்: தேதி :

சாட்சியின்கையொப்பம் : தேதி:

ஆராய்ச்சியாளரின்கையொப்பம் : தேதி:

## III- PATIENT INFORMATION SHEET

I, Dr. RAM KUMAR K (Ph:9500952726) am conducting a ***"A PROSPECTIVE STUDY OF MEAN PLATELET VOLUME, PLATELET DISTRIBUTION WIDTH AS PROGNOSTIC MARKERS IN ACUTE ISCHEMIC STROKE PATIENTS "***

You are being invited to take part in a research study. Before you decide whether to take part, it is important for you to understand why the research is being done. Feel free to discuss the study with others if you wish. Please take time to decide whether or not you wish to take part.The study will follow patients admitted in diagnosis of ACUTE ISCHEMIC STROKE ,THANJAVUR MEDICAL COLLEGE Hospital over a period of time. The type of treatment patients receive will not be altered by taking part in this study.

If you agree to take part you will be observed from the time you agree to take part until you leave the hospital. No additional tests will be undertaken as part of the study, but you will be asked to give permission for your medical records to be examined in detail, in order to collect information about your health status and any treatments that you have throughout your hospital stay.Your treatment options will not be altered in any way by taking part in this study. Your doctor will decide on the best treatment for you. Participation in the study does not restrict your ability to change from one treatment option to another.There will be noadditional visits as part of the study.There are no anticipated disadvantages or risks involved in this study as it is an observational study.All information that is collected about you during the course of this study will be kept strictly confidential.This study was reviewed and approved by the Institutional Review Board and Ethics'

DR K RAM KUMAR
DEPARTMENT OF MEDICINE ,
THANJAVUR MEDICAL COLLEGE.
PHONE NO:9500952726

நோயாளியின்தகவல்படிவம்

மரு. கு. இராம்குமார் ஆகியநான்பக்கவாத நோயாளிகளினுடையஇரத்ததட்டன்னுக்களின் வடிவம்மற்றும்செயல்களில்ஏற்படும்மாற்றங்களை பற்றிஒருஆய்வறிக்கைமேற்கொள்கிறேன் .
இதனால்பக்கவாதபாதிப்பு உள்ளவர்களுக்கு நோயின் தீவிரதன்மைபற்றி முன்கணிப்பு செய்ய இயலும். இந்தஆய்வில் தேவையான இரத்த மாதிரிகளும் ,
மருத்துவபரிசோதனைகளும்உங்களின்முழுமையான சம்மந்தம்பெற்றபின்பே மேற்கொள்ளப்படும். நீங்கள் இந்ததஞ்சாவூர்மருத்துவக்கல்லூரியில் உள்நோயாளியாக சேர்ந்தநாள்முதல்குணமடைந்து வீட்டுக்கு செல்லும் வரை இந்த ஆய்வுமேற்கொள்ளப்படும்.
எந்தஒருதருணத்திலும்உங்களின்ரகசியத்தன்மைஉறுதிசெய்யப்படும்.
இந்தஆய்வில்பங்குபெறும்நோயாளிகளுக்குநேரடியானபிரச்சனைகள் உடல், மனம், சட்ட ,
சமூகமற்றும்பொருளாதாரரீதியில்ஏற்பட்டால்அதனைஉடனடியாககண்டறிந்துஅவற்றைநிவர்த்திசெய்யப்படும்.
இந்தஆய்வில்இருத்துவெளியேறவோஅல்லதுமருத்துவபரிசோதனைகளுக்குமறுப்புதெரிவிக்கவோமுழுசுதந்திரம்உண்டு.இந்தஆய்வில்சேகரிக்கப்படும்தகவல்கள்மற்றும்முடிவுகள்பின்னாளில்பக்கவாதபாதிப்புஉள்ளவர்களுக்குநோயின்தீவிரம்பற்றியமுன்கணிப்புஆராய்ச்சிகளுக்குபயன்படுத்தப்படும்.

மரு. கு. இராம்குமார்

தஞ்சாவூர்மருத்துவக்கல்லூரிமருத்துவமனை.

| L NO | NAME | AGE | GENDER | SMOKER | ALCOHOLISM | HEIGHT | WEIGHT | BMI | HTN | | | DM | | | | DYSLIPEDIMIA | MPV | PDW | MRS D1 | MRS D7 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | | | | | Y/N | SBP | DBP | Y/N | FBS | PPBS | HBA1C | | | | | |
| 1 | THANGARAJ | 69 | F | N | N | 167.5 | 68 | 24.1 | Y | 160 | 76 | Y | 128 | 215 | 8.1 | N | 9.2 | 14.2 | 1 | 1 |
| 2 | THANGARAJ | 60 | M | N | Y | 185 | 87.5 | 25.6 | Y | 136 | 84 | Y | 130 | 205 | 7.9 | Y | 11.7 | 15.3 | 2 | 2 |
| 3 | GOVINTHAMMAL | 59 | F | N | N | 160 | 65 | 25.4 | Y | 164 | 84 | Y | 134 | 234 | 8.2 | N | 10.2 | 15.1 | 1 | 2 |
| 4 | SAMIYAMMAL | 65 | F | N | N | 155 | 73 | 30.2 | Y | 140 | 86 | Y | 99 | 156 | 8.5 | Y | 11.9 | 16 | 2 | 2 |
| 5 | RAMASAMY | 58 | M | Y | Y | 160 | 60 | 23.4 | Y | 160 | 90 | Y | 142 | 231 | 7.3 | N | 9.2 | 14.1 | 1 | 1 |
| 6 | NALLAMAL | 63 | F | N | N | 165 | 70 | 25.7 | Y | 176 | 104 | N | 90 | 135 | 6.8 | N | 11.1 | 15.6 | 1 | 2 |
| 7 | SARASWATHI | 64 | F | N | N | 162.5 | 80 | 30.3 | Y | 136 | 84 | Y | 131 | 187 | 7.5 | Y | 11.9 | 16.1 | 2 | 2 |
| 8 | NALLA THAMBI | 70 | M | Y | Y | 152.5 | 70 | 30.1 | Y | 154 | 84 | Y | 135 | 186 | 6.4 | Y | 10.2 | 15.2 | 1 | 2 |
| 9 | SAVITHRI | 60 | F | N | N | 157.5 | 65 | 26.2 | Y | 174 | 90 | N | 94 | 126 | 6.8 | N | 9.7 | 13.8 | 1 | 1 |
| 10 | MURUGAVEL | 51 | M | Y | N | 182.5 | 82.5 | 24.8 | N | 146 | 82 | N | 93 | 167 | 6.5 | N | 10.3 | 15.8 | 1 | 2 |
| 11 | KRISHNAN | 65 | M | Y | Y | 180 | 75 | 23.1 | Y | 160 | 86 | Y | 151 | 245 | 7.8 | N | 9.5 | 13 | 1 | 1 |
| 12 | MOORTHY | 65 | M | Y | Y | 170 | 63 | 21.6 | N | 134 | 82 | N | 88 | 134 | 6.8 | N | 10.4 | 16.4 | 1 | 1 |
| 13 | POTHUM PONNU | 59 | F | N | N | 152.5 | 60 | 25.8 | Y | 154 | 86 | Y | 131 | 287 | 8 | N | 10.3 | 15.5 | 2 | 2 |
| 14 | VELLAISAMY | 55 | M | Y | N | 177.5 | 80 | 25.4 | Y | 154 | 90 | Y | 135 | 203 | 8 | N | 9.8 | 13.7 | 2 | 2 |
| 15 | GOVINDARAJ | 45 | M | N | Y | 172.5 | 68 | 22.7 | N | 150 | 74 | N | 91 | 129 | 6.6 | N | 10.5 | 15.7 | 1 | 2 |
| 16 | VEERAPANDI | 50 | M | N | N | 175 | 80 | 26.1 | N | 174 | 94 | N | 121 | 209 | 6.8 | Y | 9.5 | 14.7 | 2 | 2 |
| 17 | SIVAKOLUNTHU | 64 | M | Y | Y | 162.5 | 75 | 28.4 | Y | 146 | 84 | Y | 129 | 189 | 7.8 | N | 9.6 | 14.6 | 1 | 1 |
| 18 | SARATHAMBAL | 52 | F | N | N | 150 | 63 | 27.8 | N | 156 | 76 | N | 86 | 131 | 6.8 | N | 9.7 | 14.1 | 1 | 1 |
| 19 | JOSEPH | 59 | M | Y | Y | 175 | 75 | 24.5 | Y | 144 | 84 | Y | 134 | 167 | 7.5 | N | 9.6 | 14 | 2 | 2 |
| 20 | SIVANANTHAM | 49 | M | N | N | 155 | 75 | 31.2 | N | 138 | 78 | N | 98 | 139 | 6.5 | N | 9.9 | 9 | 2 | 2 |
| 21 | PETCHIYAMMAL | 70 | F | N | N | 150 | 70 | 31.1 | Y | 140 | 84 | Y | 137 | 251 | 7.4 | Y | 11.8 | 16.5 | 2 | 2 |
| 22 | GOMATHI | 63 | F | N | N | 157.5 | 60 | 24.2 | Y | 154 | 90 | Y | 128 | 217 | 7.3 | N | 9.3 | 14.5 | 1 | 1 |

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|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 23 | AKBAR ALI | 60 | M | Y | N | 180 | 85 | 26.2 | Y | 154 | 94 | Y | 132 | 189 | 7.8 | Y | 11.5 | 15.7 | 2 | 2 |
| 24 | AKILANDESWARI | 68 | F | N | N | 155 | 68 | 28.1 | Y | 170 | 106 | N | 87 | 128 | 6.7 | N | 10.2 | 15.4 | 1 | 1 |
| 25 | SUGUNANTHAN | 63 | M | Y | Y | 177.5 | 75 | 23.8 | Y | 144 | 82 | N | 96 | 178 | 6.5 | N | 11.7 | 17 | 2 | 2 |
| 26 | RAJAVEL | 53 | M | N | Y | 172.5 | 80 | 26.9 | N | 166 | 86 | N | 95 | 118 | 6.5 | N | 8.7 | 14.1 | 2 | 1 |
| 27 | KAMARAJ | 65 | M | Y | N | 162.5 | 65 | 24.6 | Y | 152 | 90 | N | 87 | 132 | 6.8 | N | 10.4 | 15.1 | 2 | 2 |
| 28 | NELSON | 66 | M | N | Y | 160 | 70 | 27.3 | Y | 150 | 82 | Y | 141 | 217 | 8.1 | N | 11.8 | 16.1 | 2 | 2 |
| 29 | CHINNA PONNU | 43 | F | N | N | 155 | 68 | 28.1 | N | 180 | 78 | N | 97 | 128 | 6.5 | N | 8.9 | 13.2 | 2 | 2 |
| 30 | MOHAMED FAZIL | 60 | M | Y | Y | 167.5 | 73 | 25.8 | Y | 144 | 84 | N | 120 | 178 | 6.6 | Y | 11.4 | 16.5 | 2 | 2 |
| 31 | BALAJI | 55 | M | N | Y | 177.5 | 78 | 24.6 | N | 160 | 86 | N | 93 | 137 | 6.5 | N | 9.6 | 13.9 | 1 | 1 |
| 32 | MOOKAN | 57 | M | Y | Y | 160 | 70 | 27.3 | Y | 150 | 82 | Y | 138 | 209 | 7.3 | N | 10.3 | 15.7 | 2 | 2 |
| 33 | BAGYA | 68 | F | N | N | 155 | 73 | 30.2 | Y | 156 | 86 | Y | 143 | 211 | 7.8 | Y | 11.4 | 15.3 | 2 | 2 |
| 34 | KARUPPAMAL | 59 | F | N | N | 155 | 58 | 23.9 | Y | 164 | 90 | N | 89 | 128 | 7.5 | N | 9.4 | 14.3 | 1 | 1 |
| 35 | MUTHUKRISHNAN | 75 | M | Y | N | 160 | 80 | 31.3 | Y | 174 | 104 | Y | 130 | 238 | 8.2 | Y | 12.1 | 16.5 | 2 | 2 |
| 36 | CHIDAMBARAM | 61 | M | Y | N | 175 | 75 | 24.5 | Y | 140 | 82 | N | 91 | 131 | 6.6 | N | 11.7 | 16.2 | 2 | 2 |
| 37 | CHINNAPILLAI | 59 | F | N | N | 155 | 75 | 31.2 | Y | 146 | 84 | Y | 134 | 247 | 7.7 | Y | 10.8 | 15.9 | 2 | 2 |
| 38 | ZARINA BEGUM | 69 | F | N | N | 160 | 73 | 28.3 | Y | 144 | 82 | N | 87 | 138 | 6.6 | N | 9.6 | 14.2 | 2 | 2 |
| 39 | KARTHIKEYAN | 45 | M | Y | Y | 165 | 65 | 23.9 | Y | 176 | 90 | N | 91 | 164 | 6.5 | N | 10.1 | 15.7 | 1 | 1 |
| 40 | MALAISAMY | 54 | M | N | Y | 160 | 58 | 22.5 | N | 164 | 76 | N | 89 | 133 | 7.6 | N | 9.8 | 13.9 | 2 | 2 |
| 41 | MUTHU | 60 | M | N | Y | 165 | 78 | 28.5 | Y | 128 | 82 | N | 88 | 127 | 6 | N | 9.7 | 14.1 | 2 | 2 |
| 42 | SELVI | 68 | F | N | N | 170 | 78 | 26.8 | Y | 166 | 90 | N | 93 | 135 | 6.5 | N | 9.1 | 14.8 | 1 | 1 |
| 43 | PALANIVEL | 61 | M | Y | Y | 172.5 | 85 | 28.6 | Y | 154 | 86 | N | 95 | 133 | 6.6 | N | 9.2 | 13.8 | 1 | 1 |
| 44 | SANTHANA BHARATHI | 65 | M | Y | Y | 167.5 | 78 | 27.6 | Y | 146 | 82 | N | 98 | 216 | 6.4 | Y | 11.5 | 15.7 | 2 | 2 |
| 45 | KARUPAIYA | 58 | M | Y | Y | 158 | 60 | 24.2 | N | 150 | 90 | N | 91 | 126 | 6.7 | N | 10.2 | 16 | 1 | 1 |
| 46 | ANANTHI | 66 | F | N | N | 167 | 68 | 24.1 | Y | 156 | 86 | N | 89 | 137 | 6.4 | N | 9.8 | 14.2 | 2 | 2 |

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|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 47 | SARAVANA VEL | 66 | M | Y | N | 168 | 63 | 22.3 | Y | 160 | 84 | Y | 147 | 265 | 7.1 | N | 10.5 | 15.7 | 1 | 2 |
| 48 | PRASANNA KUMAR | 47 | M | N | Y | 170 | 78 | 26.8 | Y | 180 | 94 | N | 95 | 173 | 6.5 | N | 11.5 | 15.2 | 2 | 2 |
| 49 | MANIKAM | 58 | M | Y | N | 160 | 55 | 21.5 | Y | 144 | 84 | N | 89 | 137 | 6.4 | N | 8.7 | 12.1 | 1 | 1 |
| 50 | RAMALINGAM | 64 | M | N | Y | 165 | 78 | 28.5 | Y | 154 | 86 | N | 118 | 209 | 6.5 | Y | 11.2 | 16.1 | 1 | 2 |
| 51 | POORNA CHANDRAN | 59 | M | Y | Y | 166 | 75 | 27.6 | N | 144 | 70 | N | 97 | 134 | 6.7 | N | 9.7 | 13.7 | 2 | 2 |
| 52 | MARIMUTHU | 56 | M | N | Y | 155 | 58 | 23.9 | Y | 170 | 106 | Y | 138 | 235 | 7.3 | N | 9.1 | 14.5 | 1 | 1 |
| 53 | HYDER ALI | 60 | M | N | Y | 164 | 68 | 24.8 | Y | 150 | 82 | N | 94 | 128 | 6.4 | N | 9 | 14.6 | 1 | 1 |
| 54 | KAVITHA | 66 | F | N | N | 160 | 60 | 23.4 | Y | 146 | 84 | Y | 99 | 215 | 7.4 | N | 9.5 | 13.5 | 1 | 1 |
| 55 | VENNILA | 70 | F | N | N | 163 | 70 | 26.5 | Y | 168 | 86 | Y | 134 | 196 | 7.5 | N | 10.7 | 15.4 | 2 | 2 |
| 56 | ZAKINA | 65 | F | N | N | 162 | 63 | 23.7 | Y | 156 | 74 | N | 91 | 125 | 6.7 | N | 10.1 | 15.3 | 1 | 1 |
| 57 | MOOKAIYE | 70 | F | N | N | 160 | 82 | 32.2 | Y | 164 | 90 | Y | 97 | 189 | 8.3 | Y | 11.4 | 15.6 | 2 | 2 |
| 58 | RAMANA SARASWATHI | 66 | F | N | N | 153 | 60 | 25.8 | Y | 170 | 86 | N | 95 | 131 | 6.5 | N | 10.1 | 15.3 | 1 | 1 |
| 59 | GANDHI | 65 | M | Y | Y | 165 | 68 | 24.8 | Y | 156 | 90 | Y | 129 | 194 | 7.1 | N | 9.6 | 14.3 | 2 | 2 |
| 60 | KANMANI | 62 | F | N | N | 157 | 60 | 24.2 | Y | 136 | 84 | Y | 127 | 256 | 7.3 | N | 9.7 | 14.3 | 2 | 2 |
| 61 | VAIDEKI | 57 | F | N | N | 158 | 65 | 26.1 | Y | 154 | 86 | N | 129 | 189 | 6.5 | N | 8.7 | 13.7 | 1 | 1 |
| 62 | RAMAMOORTHI | 54 | M | N | N | 175 | 70 | 22.9 | Y | 184 | 96 | N | 87 | 138 | 6.4 | N | 9.4 | 14.3 | 1 | 1 |
| 63 | KASTHOORI | 55 | F | N | N | 160 | 78 | 30.3 | Y | 146 | 82 | Y | 134 | 196 | 7.5 | Y | 11.7 | 15.6 | 2 | 2 |
| 64 | DHANA VEL | 67 | M | Y | Y | 161 | 70 | 27.3 | Y | 166 | 90 | N | 98 | 117 | 6.4 | N | 9.8 | 14.5 | 2 | 2 |
| 65 | KANIMOZHI | 46 | F | N | N | 160 | 68 | 26.4 | N | 154 | 76 | N | 91 | 121 | 6.5 | N | 9.5 | 14.8 | 1 | 1 |
| 66 | ESWARI | 75 | F | N | N | 159 | 78 | 31 | Y | 156 | 94 | Y | 123 | 219 | 7.8 | Y | 11.8 | 15.4 | 2 | 2 |
| 67 | VINOTHAN | 52 | M | N | Y | 164 | 65 | 24 | N | 146 | 82 | N | 86 | 132 | 6.6 | N | 9.5 | 13.9 | 1 | 1 |
| 68 | UPPILIYAPPAN | 71 | M | Y | N | 163 | 80 | 30.5 | Y | 130 | 82 | Y | 131 | 280 | 7.5 | Y | 12.4 | 15.6 | 2 | 2 |
| 69 | SAHUL HAMEED | 56 | M | Y | Y | 173 | 70 | 23.5 | Y | 148 | 70 | N | 93 | 128 | 6.4 | N | 10.1 | 15.8 | 1 | 1 |
| 70 | GANESAN | 47 | M | Y | N | 172 | 78 | 26.1 | Y | 164 | 84 | N | 96 | 136 | 6.3 | N | 9.7 | 14.7 | 2 | 2 |

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|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 71 | RANGARAJAN | 53 | M | N | Y | 160 | 70 | 27.3 | Y | 160 | 86 | N | 88 | 128 | 6.4 | N | 9.8 | 14.5 | 1 | 1 |
| 72 | NAVANEETHAN | 62 | M | Y | Y | 171 | 69 | 24.2 | Y | 146 | 82 | Y | 133 | 219 | 7.7 | N | 11.6 | 15.8 | 2 | 2 |
| 73 | SUBBURAJ | 72 | M | N | Y | 165 | 78 | 28.5 | Y | 176 | 86 | Y | 141 | 205 | 7.1 | Y | 12.1 | 18.1 | 1 | 2 |
| 74 | BALAMURUGAN | 65 | M | Y | N | 170 | 85 | 29.4 | Y | 136 | 90 | N | 96 | 178 | 6.8 | N | 10.1 | 15.1 | 2 | 2 |
| 75 | IYYAPAN | 60 | M | N | Y | 165 | 82 | 30.4 | N | 166 | 84 | N | 98 | 186 | 6.5 | N | 9.5 | 14.7 | 2 | 1 |