"A STUDY ON PLATELET INDICES AS A PREDICTOR OF SEVERITY OF CAROTID ATHEROSCLEROSIS IN ACUTE ISCHEMIC STROKE AND ITS CORRELATION WITH NIHSS SCORE"- A CROSS SECTIONAL STUDY. (NIHSS-NATIONAL INSTITUTE OF HEALTH STROKE SCALE"

DR. MARAM SANJANA

DISSERTATION SUBMITTED TO SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION AND RESEARCH, TAMAKA, KOLAR, KARNATAKA

In partial fulfillment of the requirements for the degree of

DOCTOR OF MEDICINE

IN

GENERAL MEDICINE

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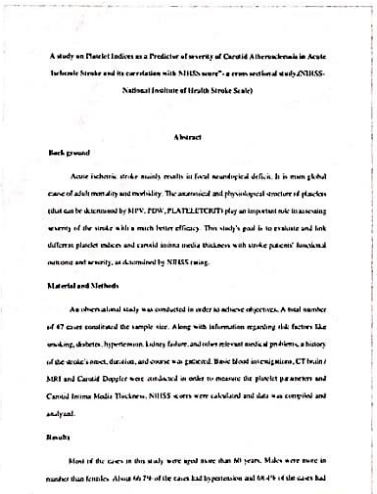

A study on Platelet Indices as a Predictor of severity of Carotid Atherosclerosis in Acute Ischemic Stroke and its correlation with NIHSS score"- a cross sectional study.(NIHSS- National Institute of Health Stroke Scale)

Abstract

Back ground

Acute ischemic stroke mainly results in focal neurological deficit. It is main global cause of adult mortality and morbidity. The anatomical and physiological structure of platelets (that can be determined by MPV, PDW, PLATELETCRIT) play an important role in assessing severity of the stroke with a much better efficacy. This study's goal is to evaluate and link different platelet indices and carotid intima media thickness with stroke patients' functional outcome and severity, as determined by NIHSS rating.

Material and Methods

An observational study was conducted in order to achieve objectives. A total number of 67 cases constituted the sample size. Along with information regarding risk factors like smoking, diabetes, hypertension, kidney failure, and other relevant medical problems, a history of the stroke's onset, duration, and course was gathered. Basic blood investigations, CT brain / MRI and Carotid Doppler were conducted in order to measure the platelet parameters and Carotid Intima Media Thickness. NIHSS scores were calculated and data was compiled and analysed.

Results

Most of the cases in this study were aged more than 60 years. Males were more in number than females. About 66.7% of the cases had hypertension and 68.4% of the cases had

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A study on Platelet Indices as a Predictor of severity of Carotid Atherosclerosis in Acute Ischemic Stroke and its correlation with NIHSS score"- a cross sectional study.(NIHSS- National Institute of Health Stroke Scale) Abstract Back ground Acute ischemic stroke mainly results in focal neurological deficit. It is main global cause of adult mortality and morbidity. The anatomical and physiological structure of platelets (that can be determined by MPV, PDW, PLATELETCRIT) play an important role in assessing severity of the stroke with a much better efficacy. This study's goal is to evaluate and link different platelet indices and carotid intima media thickness with stroke patients' functional outcome and severity, as determined by NIHSS rating. Material and Methods An observational study was conducted in order to achieve objectives. A total number of 47 cases constituted the sample size. Along with information regarding risk factors like smoking, diabetes, hypertension, kidney failure, and other relevant medical problems, a history of the stroke's onset, duration, and course was gathered. Basic blood investigations, CT brain / MRI and Carotid Doppler were conducted in order to measure the platelet parameters and Carotid Intima Media Thickness. NIHSS scores were calculated and data was compiled and analyzed. Results Most of the cases in this study were aged more than 60 years. Males were more in number than females. About 66.7% of the cases had hypertension and 68.4% of the cases had diabetes mellitus. The mean platelet volume in this study was 8.8, platelet distribution width was 12.4, plateletocrit was 2.8 in this study. The mean Carotid intima media thickness was 0.1 and mean NIHSS scores was 10.9 in this study. CIMT had negative but not significant correlation with MPV, PDW and PCT in this study. The pearson correlation coefficient was negative for MPW. The pearson correlation coefficient was positive but not significant for PDW, positive and significant for PCT with NIHSS. Conclusions This study had shown that, PCT had a positive and significant correlation with the NIHSS scores. CIMT had no correlation with any platelet indices. Key words: Acute ischemic stroke, platelet indices, NIHSS scores, Carotid Intima Media thickness. INTRODUCTION Definition of Stroke is rapid onset of focal neurological deficit due to vascular cause. Stroke is main global cause of adult mortality and morbidity, presenting challenges in the areas of medicine, finance, and rehabilitation. It accounts for 80% to 85% of all cerebrovascular disease.1 Stroke ranks third globally in terms of years lived with a handicap and is the second common cause of death around the world. As per reports, the prevalence of stroke in India is 1.27–2.20 per 1000 people.2 The current data indicates that the profile of risk factors for stroke is unfavourable and that it is ideally suited for prevention due to its high prevalence, significant sickness burden and expense, clearly identified modifiable risk factors, and efficacious preventative strategies.3 Ischemic stroke, also known as ischemic cerebrovascular disease, is a major global and Indian public health issue. According to the literature now in circulation, a stroke occurs in the United States once every 53 seconds. Seventy-five thousand Americans suffer from ischemic strokes, either acute or recurrent.4, 5 According to Indian studies, there is a significant range in the prevalence of stroke in India between 147 and 922/100,000 according to several community- based studies.6 A sizable portion of the expenditures are accounted for higher stroke incidence and the high cost for treatment. Therefore, stroke and its aftereffects are crucial concerns for insurance companies, government policy

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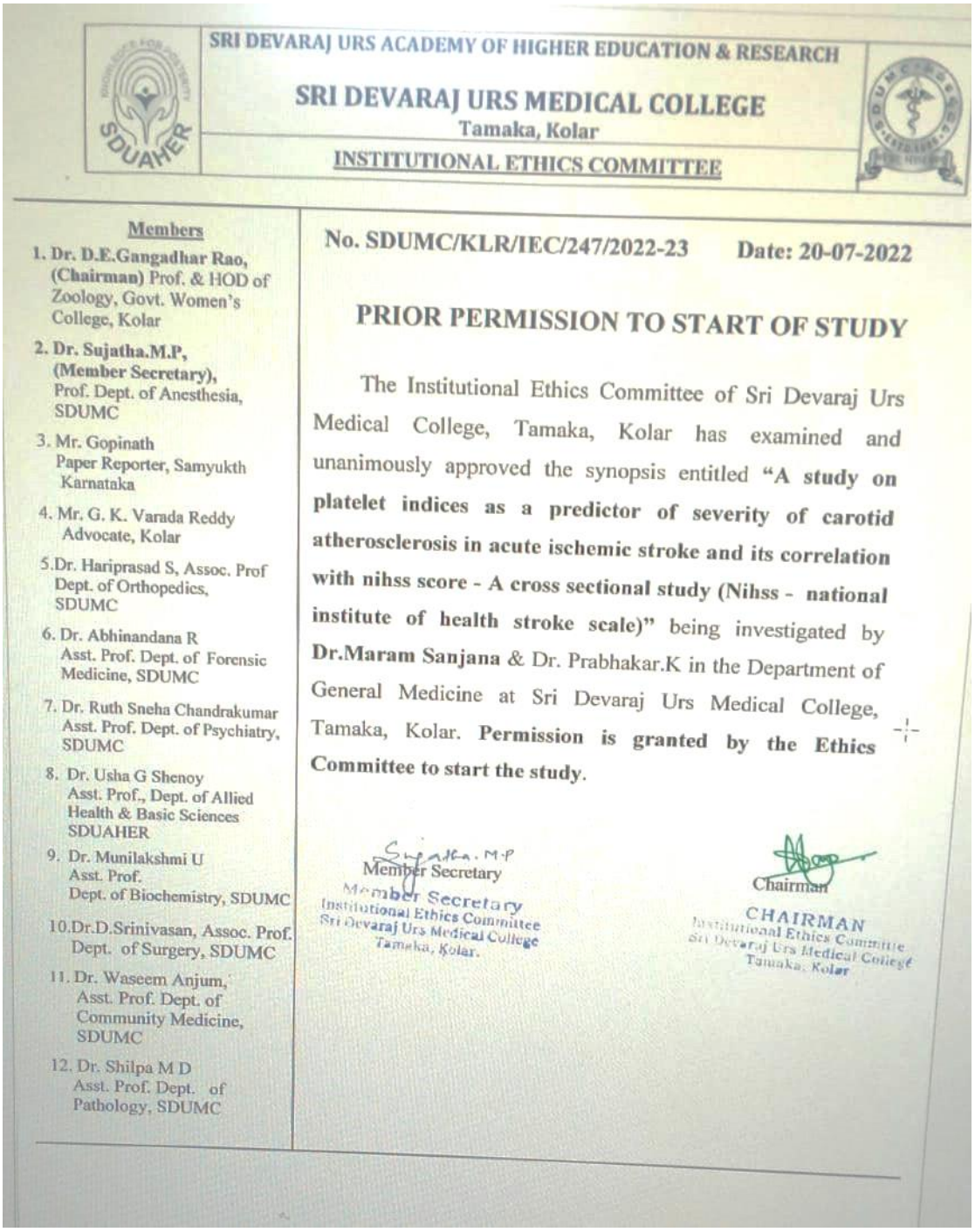

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PRIOR PERMISSION TO START OF STUDY

The Institutional Ethics Committee of Sri Devaraj Urs Medical College, Tamaka, Kolar has examined and unanimously approved the synopsis entitled **"A study on platelet indices as a predictor of severity of carotid atherosclerosis in acute ischemic stroke and its correlation with nihss score - A cross sectional study (Nihss - national institute of health stroke scale)"** being investigated by **Dr.Maram Sanjana** & Dr. Prabhakar.K in the Department of General Medicine at Sri Devaraj Urs Medical College, Tamaka, Kolar. **Permission is granted by the Ethics Committee to start the study.**

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A STUDY ON PLATELET INDICES AS A PREDICTOR OF SEVERITY OF CAROTID ATHEROSCLEROSIS IN ACUTE ISCHEMIC STROKE AND ITS CORRELATION WITH NIHSS SCORE"- A CROSS SECTIONAL STUDY. (NIHSS-NATIONAL INSTITUTE OF HEALTH STROKE SCALE

ABSTRACT

BACKGROUND: Acute ischemic stroke mainly results in focal neurological deficit. It is main global cause of adult mortality and morbidity. The anatomical and physiological structure of platelets (that can be determined by MPV, PDW, PLATELETCRIT) play an important role in assessing severity of the stroke with a much better efficacy. This study's goal is to evaluate and link different platelet indices and carotid intima media thickness with stroke patients' functional outcome and severity, as determined by NIHSS rating.

MATERIAL AND METHOD: An observational study was conducted in order to achieve objectives. A total number of 47 cases constituted the sample size. Along with information regarding risk factors like smoking, diabetes, hypertension, kidney failure, and other relevant medical problems, a history of the stroke's onset, duration, and course was gathered. Basic blood investigations, CT brain / MRI and Carotid Doppler were conducted in order to measure the platelet parameters and Carotid Intima Media Thickness. NIHSS scores were calculated and data was compiled and analyzed.

RESULTS: Most of the cases in this study were aged more than 60 years. Males were more in number than females. About 66.7% of the cases had hypertension and 68.4% of the cases had diabetes mellitus. The mean platelet volume in this study was 8.8, platelet distribution width was 12.4, plateletocrit was 2.8 in this study. The mean Carotid intima media thickness was 0.1 and mean NIHSS scores was 10.9 in this study. CIMT had negative but not significant correlation with MPV, PDW and PCT in this study. The pearson correlation coefficient was negative for MPW. The pearson correlation coefficient was positive but not significant for PDW, positive and significant for PCT with NIHSS.

CONCLUSION: This study had shown that, PCT had a positive and significant correlation with the NIHSS scores. CIMT had no correlation with any platelet indices.

KEY WORDS: Acute ischemic stroke, platelet indices, NIHSS scores, Carotid Intima Media thickness

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LIST OF ABBREVIATIONS USED
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AMPA	**Alpha amino 3 hydroxy 5 methyl 4 isoxanole propionate**
APTT	**Activated Partial Thromboplastin Time**
ACE inhibitor	**Angiotensin Converting enzyme inhibitor**
ANA	**Anti-nuclear Antibody**
ARBs	**Angiotensin Receptor Blockers**
CIMT	**Carotid Intima Media Thickness**
CT	**Computed tomography**
CVD	**Cardiovascular diseases**
CPR	**Crude Prevalence Rate**
DSA	**Digital Subtraction Angiography**
DNA	**Deoxy Nucleic Acid**
DASH	**Dietary Approach to stop hypertension**
ECHO	**Echocardiography**
ECG	**Electro Cardiogram**
HMG CoA	**β-Hydroxy β-methylglutaryl-CoA**
ICU	**Intensive Care Unit**
IH	**Intracranial Hemorrhage**
MPV	**Mean Platelet Volume**
MRI	**Magnetic Resonance Imaging**
NIHSS	**National Institute of Health Stroke Scale**
NMDA	**N methyl-D-aspartate**
PDW	**Platelet Distribution Width**
PCT	**Plateletocrit**
PT	**Prothrombin time**
SAH	**Sub Arachnoid Hemorrhage**
TIAs	**Transient Ischemic Attack**
WHO	**World Health Organization**

INTRODUCTION

INTRODUCTION

Definition of Stroke is rapid onset of focal neurological deficit due to vascular cause. Stroke is main global cause of adult mortality and morbidity, presenting challenges in the areas of medicine, finance, and rehabilitation. It accounts for 80% to 85% of all cerebrovascular disease.[1] Stroke ranks third globally in terms of years lived with a handicap and is the second common cause of death around the world. As per reports, the prevalence of stroke in India is 1.27–2.20 per 1000 people.[2]

The current data indicates that the profile of risk factors for stroke is unfavourable and that it is ideally suited for prevention due to its high prevalence, significant sickness burden and expense, clearly identified modifiable risk factors, and efficacious preventative strategies.[3] Ischemic stroke, also known as ischemic cerebrovascular disease, is a major global and Indian public health issue. According to the literature now in circulation, a stroke occurs in the United States once every 53 seconds. Seventy-five thousand Americans suffer from ischemic strokes, either acute or recurrent.[4, 5] According to Indian studies, there is a significant range in the prevalence of stroke in India between 147 and 922/100,000 according to several community-based studies.[6]

A sizable portion of the expenditures are accounted for higher stroke incidence and the high cost for treatment. Therefore, stroke and its aftereffects are crucial concerns for insurance companies, government policy makers, and healthcare providers nationwide. The financial ramifications of lost production are enormous, and preventing strokes is an economical way to avoid treatment costs.[7]

Although many risk factors may cause stroke, these factors can only account for half of stroke cases.[8] It is possible to modify risk factor of hypertension that contribute ischemic stroke including obesity, inflammation, high-fat diets, sedentary lifestyles, and smoking.[9]

Numerous clinical outcomes have been linked to ischemic cerebrovascular sickness, contingent type and intensity of symptoms of neurology, the affected region of brain, the resultant artery, and risk factor for stroke. This categorization can be used to determine the prognosis of the patient and to inform decisions about evaluation and therapy. When a patient has quickly escalating clinical presentation of focused or occasionally global, loss of function of brain with manifestations lasting more than 24 hours or leading to death, and there is no other clear reason other than vascular origin, it is called a stroke.[10]

Because it speeds up the process, platelet activation and aggregation are important factors in pathophysiology of acute ischemic stroke. The rupture of an atherosclerotic plaque is the primary cause of arterial thrombosis. Plaque accumulation in the vascular intima is the cause of atherosclerosis. This plaque is made up of fibrofatty streaks that contain lipids and foam cells. When this ruptures, it forms a thrombus around which a blood clot is formed by the following mechanism involving platelets.[11]

Initially, after a vascular injury to the vessel intima due to a plaque rupture, local factors induce vasoconstriction. Then, new platelets are formed and these bind to the site of injury via GpIb receptors to von willebrand factor and get activated, to release granules like ADP and thromboxane A2 which further induce platelet aggregation through the GpIIb-IIIa receptor that binds to fibrinogen, forming the "primary hemostatic plug". After this the clotting factors are activated and fibrin is formed resulting in the definitive secondary hemostatic plug.[12]

Granule-releasing new platelets are larger in size because they are more active metabolically and enzymatically throughout their creation. The "Mean platelet volume MPV" is a reliable indicator of platelet size and function. As a trustworthy gauge of platelet size and function is "Mean Platelet Volume (MPV)."[13]

So, the anatomical and physiological structure of platelets (that can be determined by MPV, PDW, PLATELETCRIT) play an important role in assessing severity of the stroke with a much better efficacy.[14]

A commonly used standardised method of assessing stroke symptoms and indicators is 15-item National Institute of Health and Stroke Scale (NIHSS). Higher score denote greater severity. The scoring range is shown to range from 0 to 42 points. This score has a sensitivity of 72% and specificity of 95% which is considered acceptable, and hence being correlated with platelet indices.[15]

Atherosclerosis can also be assessed using carotid intima media thickness as vessel wall is altered through a process called Vascular remodeling which is the high blood pressure response leading to the reduced diameter of the blood vessel due to vessel wall hypertrophy, which can be measured using carotid doppler, and hence is a good indicator in the assessment of stroke.[16]

The objective of this present study is to assess and correlate various platelet indices and carotid intima media thickness with severity and functional outcome of stroke patients which is measured with NIHSS scoring.

AIMS & OBJECTIVES

AIMS OF OBJECTIVES

OBJECTIVES OF THE STUDY

- To analyse and correlate platelet indices with NIHSS score to predict the severity.
- To assess the relationship between the carotid intima media thickness and the platelet indices in patients admitted with acute ischaemic stroke.

REVIEW OF LITERATURE

REVIEW OF LITERATURE

HISTORICAL PERSPECTIVE

Even before Hippocrates, people had been afflicted by cerebral vascular disorders for aeons. Long before cardiovascular disorders were ever diagnosed, phrases like "paralysis," "stroke," and "apoplexy" were frequently used interchangeably. Hippocrates provided first documented explanation of a stroke and created the word "apoplexy," which means "astonished."[17]

There was a 2000-year gap in the literature after Hippocrates about the progress and stroke comprehension. Werter noted for the first time that injury to cerebral blood vessels could cause apoplexy.

Fundamentals of circulation were explained by William Harvey for first time. Thomas Willis wrote a description of the Circle of Willis in 1664. Brunner determined the importance of a brain artery aneurysm in the subarachnoid hemorrhage's cause. Subsequent research was conducted by Morgagni and Charcot.

In 1813, Seddict reported on spontaneous intracranial haemorrhage and obliterative vascular disease of the cerebral arteries. Virchow gave a description of the development of vascular thrombosis in 1860. The location of lesions and aphasia were documented by Bonillord in 1825. Von Graafe utilised the Helmoltz ophthalmoscope for the first time in 1860 to diagnose neurological diseases. Heubner (1964) described the cerebral artery that bears its name as well as the several anastomoses that join the main branches.[18]

Jackson et al. presented the theory of nervous system levels in the diagnosis of brain sickness in 1862. Hamrich Quinn invented the lumbar puncture procedure in 1895.

When diabetic urine samples were heated to a point where they coagulated, proteinuria was initially identified.[19] Keen and Choluverkis created a radio-immuno test in 1963 to detect low urine albumin concentrations. Since the creation of more advanced methods for identifying minute levels of proteins in urine, the diagnosis of microalbuminuria has advanced significantly. It first appeared as a sign of diabetics' early renal failure and is currently recognised as a biochemical correlate of cerebrovascular illnesses that are not diabetic.[20, 21]

ISCHEMIC STROKE OF ACUTE ORIGIN

In wealthy nations, it ranks as the second most frequent cause of illness and mortality. A major public health concern in developing nations such as India is stroke.[22] Majority of stroke cases, which are more common as people age, are caused by ischemic stroke, according to a review of the stroke's morbidity pattern.[23]

EPIDEMIOLOGY

The World Health Organisation (WHO) estimates that when it comes to mortality rates adjusted for age, gender, and burden (measured in disability adjusted life year loss rates, or DALYs), national differences can reach up to ten times. In comparison to high income countries, they are located in North Asia, Eastern Europe, Central Africa, and the South Pacific.[24]

Based on figures from the World Health Organisation, stroke accounted for 6,19,000 of the 9.4 million deaths (India) in 1990; this translates to a stroke mortality rate of 73 per100,000. According to projections, stroke killed four times as many people as

rheumatic heart disease, almost as many as ischemic heart disease, 1.4 times as many as tuberculosis, and 22 times more people than malaria.[25]

According to the literature that is now accessible, India will have a significant socioeconomic burden covering the costs of "stroke victims'" rehabilitation since more people are living to reach the peak age range of 55 to 65, which is when strokes most commonly occur. A study indicated that 320 cases out of 145, 456 persons had hemiplegia that was suspected to be caused by cardiovascular disease (CVD). This means that the crude rate of prevalence (CPR) was 220 per lakh people overall.[26]

Case fatality rate has significantly decreased, according to studies that are presently available, and this has resulted in a high survival rate with residual disability. This has made stroke survivors' needs for occupational rehabilitation a serious social issue.[27] This suggests that there might be a "Stroke epidemic" in India already.

RISK FACTORS

These risk factors have been well researched, as stroke is a complicated disease. Patients of different ages have different risk factor profiles, which also vary. Among the most common modifiable proatherothrombotic factors that might cause stroke include dyslipidemia, smoking, drinking, diabetes mellitus, and hypertension. Additional risk factors include vasculitis, high homocysteine, migraines, insufficient physical activity, elevated homocysteine, fibrinogen and clotting factors, oral contraceptives, and hormonal supplements. The risk factors for ischemic stroke that cannot be changed include genetic predisposition, age, gender, ethnic and geographic background, and familial tendency. The frequency of cerebral infarction does not go down even when these risk factors are sufficiently addressed, which emphasises the need to look into new and unknown risk factors.[28]

Age

The available literature showed that an individual's age raises their risk of stroke. Because the embolism starting in the heart, individuals under 45 are high likely to have stroke; patients over 65 are likely to have major and small artery abnormalities, which frequently accompany embolism.[29] According to available data, over 75% of stroke incidents involve elderly persons over the age of 60.[30]

Gender

Cerebral infarction affects men more frequently than women. Men are more likely than women to experience atherosthrombotic infarction and transitory attacks, but women are somewhat more likely to get cerebral embolism. A study found that among patients over 40 who had an ischemic stroke found 5.5:1 as male to female ratio.[31]

High blood pressure

Hypertension is significant risk factor of thrombotic stroke and cerebral haemorrhage. Individuals with proven hypertension have a right fold increase in stroke risk, but those with borderline hypertension had a about threefold risk. For males and women, respectively, left ventricular hypertrophy raises the risk of ischemia by 3.3 and 3.1. It has been demonstrated that higher blood pressure readings, both systolic and diastolic, indicate a higher risk. Based on available data, men over 65 who suffer from isolated hypertension are twice as likely to experience a cerebral infarction.[32]

Diabetes mellitus

Diabetics are more likely to develop coronary, cerebral, and femoral atherosclerosis. One independent risk factor for thromboembolic stroke is an increased

degree of glucose tolerance. In men and women relative risk was 1.8 times of diabetes for stroke and 2.2 after controlling for other risk factors.

Heart disease

Whether overt or covert, deterioration in heart function increases risk of stroke considerably. Persistent atrial fibrillation, vulvar heart problems, intra-cardiac thrombus, left ventricular hypertrophy, and pre-existing coronary heart disease are among the cardiac ailments. Atrial fibrillation increases the risk of stroke in people with concurrent cardiovascular risk factors. The risk of stroke is increased by a factor of 17 when atrial fibrillation and mitral stenosis coexist, while it is lowest when atrial fibrillation occurs alone.[29]

Smoking

Smoking cigarettes is dangerous risk factor for stroke. Blood viscosity, arterial vasoconstriction, and platelet aggregation are all increased by smoking. Smoking causes sympathetic nervous system activation and endothelial damage. According to a meta-analysis, smoking increases the risk of stroke by fifty percent in comparison with non-smokers and was independent risk factor in the occurence of strokes in all age groups and both sexes.[33, 34]

Consumption of alcohol

Research has indicated a favourable correlation between alcohol use and the incidence of hemorrhagic stroke; however, the relationship with ischemic stroke is less certain. Consuming alcohol increases the occurrence of both subarachnoid haemorrhage

(SAH) and intracranial haemorrhage (IH), even after accounting for other pertinent risk factors. Actually, drinking alcohol helps prevent ischemic strokes.[35]

Obesity

Obesity in general has a lower stroke risk than obesity in abdomen. Literature has demonstrated high blood pressure, blood glucose, and atherogenic serum lipids are present in obese persons.[31]

Physical activity

Excessive drinking, smoking, age, socioeconomic class, systolic blood pressure and a history of ischemic heart disease or stroke all have strong negative correlation with the risk of stroke. After 30 years on average, sports activity was found to reduce the incidence of fatal stroke by 60%.[31]

Oral contraceptives

Oral contraceptives have been linked to a five-fold increase in stroke risk in adults over 35. It is more likely that thrombosis, not atherosclerosis, is the cause of arterial occlusion. It has been demonstrated that oral contraceptives increase platelet aggregation and change clotting factors in a way that promotes thrombogenesis.[31]

Serum Lipids

Framingham heart study and Honolulu heart study had shown no significant relationship between blood cholesterol and risk of stroke. These studies have shown a favourable correlation between blood pressure and stroke but not between cholesterol and

stroke. A case control research found no appreciable differences in serum cholesterol, triglyceride, or LDL cholesterol levels between ischemic stroke patients and matched controls. An association has been discovered between higher cholesterol levels and death from ischemic stroke, controlling for age, cigarette smoking, diastolic blood pressure and race/ ethnic group using a proportional hazards regression. [35]

PATHOPHYSIOLOGY

The two primary causes of brain damage are haemorrhage and ischemia. In ischemic stroke, accounting for 80% of all strokes, the neurons are deprived of necessary substrates due to decreased or absent circulation. As brain tissue cannot metabolise anaerobically, the deprivation of glucose resulting from ischemia causes damage to the tissue. Non-traumatic intracranial haemorrhage accounts for 10 to 15% of all strokes. Brain tissue damage causes connecting pathways disruption and localised injury from intracranial haemorrhage from deeply piercing vessels. A biochemical substance that is destructive is released from various sources plays a significant role in tissue destruction.[37, 38]

CLASSIFICATION OF STROKE BASED ON PATHOPHYSIOLOGY

Hemorrhage of primary origin

Hemorrhage of Subarachnoid region

Intracerebral haemorrhage

Stroke due to ischemia

Large vessel disease (Carotid vertebral stenosis)

Small vessel disease (lacunar stroke)

Cardioembolic stroke

In situ thrombosis

Other causes

Vasculitis

Carotid and vertebral dissection

Porthrombotic disorders

Infections

Other rare causes

Undetermined

Focal Injury due to ischemia

Ischemia results due to blockage of cerebral artery in affected vascular region. An embolus cannot be distinguished from a thrombus induced lesion. An embolism from an artery to another may result from a thrombosis of the arterial. Regardless of the underlying cause, hypoxia or anoxia define the molecular routes of neuronal injury. The extent and trajectory of ischemia damage are influenced by multiple factors.[39, 40]

Onset rate and duration

The brain can withstand an ischemia event better if it occurs slowly or over a shorter period of time.

Collateral circulation

The effect of ischemia injury depends on the collateral circulation condition in the afflicted brain region. When there is strong collateral circulation, the result is better.

Systemic circulation role

The constant cerebral perfusion pressure is determined by a sufficiently high systemic blood pressure. Global cerebral ischemia may be the root cause of systemic hypotension.

Hematological factors

In a hypercoagulable state, the arterial obstruction is made worse by the growth and size of small thrombi.

Temperature

Elevated body temperature influences higher cerebral ischemia injury.

Blood Flow of Cerebrum

Typical blood flow of cerebrum varies between 50 and 60 ml/100 g/min in various parts of the brain. Brain blood flow is compensated for by collateral opening, an auto-regulatory mechanism known as local vasodilatation, and enhanced blood oxygen and glucose extraction. In an effort to preserve energy storage, there is an electrical calm and a sharp decrease in synaptic activity when the CBF drops to 20 ml/100 g/min. Neural injury is irreversible when cerebral blood flow is less than 10 ml/100 mg/min.[40, 41]

Neuronal Injury mechanism

The formation of minute thrombi is a complex phenomenon that affects cerebral arterioles and capillaries microcirculation. Damage-causing vasoactive enzymes produced

by ischemia and secreted by endothelium, leucocytes, platelets, and other neural cells activate, causing microthrombus formation.

The two primary neurotransmitters implicated in molecular level hypoxic-ischemic neuronal injury are glutamate and aspartate response. When the cellular energy reserve is depleted, the process known as excitotoxicity begins. An energy-dependent mechanism is responsible for clearing the glutamate that is normally stored inside the synaptic terminals. A low energy state causes an increase in glutamate (and aspartate) concentration in the extracellular space, which causes calcium channels connected to alpha amino 3 hydroxy 5 methyl 4 isoxanole propionate (AMPA) and N methyl-D-aspartate (NMDA) receptors to open. Potassium ions export and calcium, sodium, and chloride ions enter when a persistent membrane depolarizes.

A number of deleterious enzymes including lipases, proteases and endonucleases become active when intracellular calcium is present. Integrity of the cell is compromised by resulting cytokines and other mediator release.[41, 42]

Penumbra due to Ischemia (IP)

Within one hour of a hypoxic ischemic insult, an oligeamic zone encircling the infarction site emerges, rendering autoregulation useless. The "Window of Opportunity" is a crucial window of time when a certain amount of brain tissue is vulnerable. Restoring the entire window of opportunity within the critical two to four hours can be achieved via reperfusion of the ischemic but viable brain tissue.[43, 44]

Neuronal death

It is known that damaged neurons perish by two processes: apoptosis and coagulation necrosis. The neurons inflate, shrink, and go through pyknosis—a visible

nuclear chromatin condensation—when they have coagulation necrosis. Coagulation necrosis takes six to twelve hours to complete. Pan-necrosis, the result of extensive chromatolysis, happens within twenty four hours. Astrocyte grows, fragment and subsequently myelin sheath will degrade. Eight to twelve hours after artery occlusion, eosinophilic cytoplasm and shrinking nuclei show irreversible cellular damage. When cells undergo coagulation necrosis, they appear differently than when they undergo apoptosis.[38]

The neurons of are often preprogrammed for death in response to specific stimuli like ischemia, is called apoptosis.[45] While coagulation necrosis starts six hours after the artery is blocked, ischemia occurs one hour after apoptosis. Future studies should focus on the alteration of the DNA cleavage process that causes the apoptosis that leads to neuronal death.[39]

Stroke due to ischemia

Embolism, Thrombosis and global ischemic stroke are main causes of ischemic strokes; however, not all ischemic strokes follow this pattern; some may present in a different way, leading to atypical stroke mechanisms. Notable examples of less common causes of stroke include vasospasm, and various forms of arteritis.

Thrombosis

Atherosclerosis is primary cause for vascular blockage that leads to thrombotic stroke. The pathological changes that atherosclerotic plaque experiences include ulcerations, thrombosis, calcifications, and intraplaque haemorrhage. The plaque's composition, structure, and consistency all affect how easily it can rupture or become inflamed. When any of these pathological alterations occur, endothelium disruption takes

place through a complex process that activates a number of harmful vasoactive enzymes. Little nidi of fibrin and platelets form as a result of the platelets adhering and aggregating to the vascular wall. Within an hour, leucocytes arrive at the site and trigger an inflammatory reaction.[46, 47]

Thrombotic occlusion can be caused by dissection of a vascular wall, gaint cell and Takayasu arteritis, as well as atherosclerosis. Clot formation brought on by fibromuscular dysplasia and a hypercoagulable state.

The occlusion of 100 – 400 mm deep penetrating arteries results in Lacunar infarcts. The most often injured regions are putamen and pallidum, which are followed by the internal capsule, thalamus, pons and caudate nucleus.[48, 49]

Embolism

An embolic stroke can occur from an embolised artery in the central circulation for a number of causes. It is recognised that fibrin, fragments of atheromatous plaque, fat, air, cancer or metastases, bacterial clusters, and other elements can embolise into central circulation. The cerebral and cerebellar arteries' superficial branches are the arteries that are most commonly affected. Approximately 80% of the blood carried by the major neck arteries is carried by the middle cerebral arteries, the site of emboli damage that occurs most commonly.[48, 50]

The emboli commonly originate in left cardiac chambers and main arteries. Haemorrhagic infarction can happen because hemorrhagic stroke is a common stroke that follows an embolic stroke. The bleeding that takes place within the necrotizing brain tissue is called a hemorrhagic infarct (ischemic infarct).

In embolic stroke, Many risk factors are responsible in pathophysiological pathways of hemorrhagic infarct. When blood flow returns to the ischemic area after an embolus lyses spontaneously, ischemic tissue often reperfuses, resulting in hemorrhagic transformation. Upon lysis or restoration of the ischemic microcirculation by the occluding embolus, blood flow returns to the affected area of the brain. Hemorrhagic or red infarcts are areas devoid of blood. When areas with inadequate blood flow persist, pale or anaemic infarcts form. Since hemorrhagic transformation can result from persistent proximal artery occlusion, it is not invariably linked to the migration of embolic debris. When leptomeningeal artery reperfusion results in collateral circulation due to persistent arterial occlusion, hemorrhagic infarction around infarcts happens. In embolic stroke, hemorrhagic infarction and ischemic infarction frequently coexist.[50]

Global Ischemic or hypotensive Stroke

Any cause for a significant drop in systemic blood pressure can lead to a hypotensive stroke. The cerebral cortex's Purkunje cell layer and the hippocampus's pyramidal cell layer are two types of neurons that are particularly vulnerable to ischemia. Grey matter in the brain is also susceptible to ischemia. Owing to glutamate excess, these neurons are more vulnerable to global ischemia.

The areas between the major cerebral and cerebellar artery zones, also referred to as the "watershed area" or "boundary zone," are most severely damaged by global ischemia. The area most commonly affected is the parietal, temporal, and occipital triangle, which joins the anterior, middle, and posterior cerebral arteries.[50, 51]

CLINICAL MANIFESTATIONS

Stroke Athero thrombosis

Prodromal TIA events, which often last less than ten minutes and signify a developing thrombus in major arteries, occur before to stroke. Embolism is the cause of the repeated occurrences of distinct pattern TIAs that linger for several hours and may cause a more severe impairment. According to published research, 20% of strokes occur after TIAs within 30 days of the initial episode, and 50% occur within a year.[52]

Most TIAs last 2 to 15 minutes, however they can last anywhere from a few seconds to 12 to 24 hours. Between the assaults, no neurological abnormalities are visible. A transient ischemic attack (TIA) can last anywhere from a few seconds to several minutes and impact various bodily locations simultaneously. It is recognised that upright postures are more conducive to TIAs than supine positions.[53]

The neurological deficit associated with an evolving stroke is what advances or varies while the patient is being observed; on the other hand, a finished stroke indicates that no more deterioration will take place. There are varying degrees of neurological impairments caused by the spreading, moving, lysing, and eventually vanishing embolus.[54]

Headache, nausea, and loss of consciousness are the primary symptoms at the beginning of a stroke; these symptoms are uncommon in intracranial haemorrhage. In contrast to an intracerebral or subarachnoid haemorrhage, the headache is moderate, and there is no neck stiffness. Numerous vascular syndromes have been identified, and the participation of particular regions suggests the involvement of particular vascular territories.

Lacunar infarcts

Lacunar infarcts results due to infarction of deeper regions of cerebral hemisphere, affecting the thalamus, internal capsule, basal ganglia and paramedian region. These infarcts are caused by lipohyalinosis or micro atheromas. Ten to fifteen percent of cases had these infarcts. This kind of infarct is too little in thirty people. Only clinical symptoms, such as those of pseudo bulbar syndrome, ataxic hemi paresis, clumsy hand dysarthria syndrome, and pure motor or sensory stroke, can be used to make the diagnosis.

Infarction due to emboli

Fragments of the mural thrombus and aggregates of platelets constitutes emboli. This kind of thrombus mainly originates in the heart and is caused by valvular heart disorders, atrial fibrillation, rheumatic fever, cardiomyopathy, bacterial endocarditis, paradoxical emboli, atherosclerosis, and cardiac catheterization or surgery.

Any time of day could see an embolus attack. The brain damage quickly goes away due to embolus fragmentation. The nature of a persistent neurological deficiency is determined by the affected territory. By lodging in to branches of the middle cerebral artery, the small emboli can result in Wernicke's aphasia, monoplegia, Brocas aphasia, and other disorders. Awareness is usually retained following notable deficits.

INVESTIGATIONS

Usually, a comprehensive physical examination and a detailed history guide the proper investigation. The basic tests, such as peripheral smear, hematocrit, PT APTT, and full blood picture, can be used to identify the cause of stroke. Among the biochemical exams are the lipid profile, liver function tests, blood urea, electrolytes, and fasting blood

glucose. An ECG, a chest X-ray, an echocardiogram, and a urine test for protein, glucose, and cell casts are among the extra testing.

The Doppler Doppler DSA, ultrasound, CT, and MRI are the remaining particular examinations. Among the specific tests that could be recommended in some situations are hematoxylophoresis, anti DNA, bone marrow aspiration, ANA, antithrombin III, complement studies, lupus anticoagulant, serum viscosity and blood culture.

CT Scan

Imaging determines the cerebral ischemia signs and symptoms. Data that has been published indicates that 60% of scans are carried out in the initial hours after a brain infarction. Although some early signs of acute stroke, such as hyperattenuating artery, lentiform nucleus obscuration, loss of the gray-white interface along the lateral insula and effacement of the gray-white junction together with the cortex, may be present, these scans are typically normal.

Sub-acute infarcts

Non-enhanced CT scans after 24 to 48 hours show the wedge-shaped zones of reduced attenuation in significant vascular infarcts, which include both grey and white matter. The mass effect first increases and begins to decrease after seven to ten days. In fifteen to twenty percent of MCA occlusions, hemorrhagic change may be seen. The enhanced pattern looks patchy/ gyral in three to four days after ictus and can last for eight to ten weeks.

Chronic infarcts

The encephalomalacic regions are well defined by the chronic infarcts. There may be expansion of the ipsilateral ventricle and a prominent of the neighbouring sulci. The improvement vanishes after ten weeks. Moreover, dystrophic calcifications are visible.

MRI (Magnetic Resonance Imaging)

A few hours after the stroke, it may reveal the ischemia zones before a CT scan picks them up. In addition to the CT scan, an MRI can show flow voids, hemosiderin, and changes brought on by gliosis and ischemic necrosis. MRI angiography shows the carotid and vertebrobasilar systems' blood flow as well as vascular lesions, such as atheromatous plaques.

DSA

DSA allows for the visualisation of the cervical and basal intracranial arteries as well as their collaterals. Compared to standard arteriography, this approach carries a lesser risk. The main disadvantage of this method is that in people with pre-existing cardiac conditions, the injected material may result in cardiac decompensation.

Electro encephalo graphy

It is not very useful for identifying infarction or differentiating it from haemorrhage.

PROGNOSIS

According to a Minitoba study, men are more likely than women to survive, as are the young compared to the old, married individuals compared to single people, and victims of bleeding in rural as opposed to urban settings.[56]

Recurrent stroke has been associated with a number of risk factors including diabetes mellitus, obesity, heart disease, hyperlipidemias, atrial fibrillation, oestrogen medicine, physical inactivity, high alcohol intake, and smoking.[57]

About 85% of individuals with cerebral infarction survived for thirty days. When hemorrhagic stroke occurs during an acute stroke, the case fatality rate is higher, ranging from 30% to 80% for intracranial haemorrhage and 20% to 50% for subarchnoid haemorrhage.[58]

Large infarcts may cause the infracted tissue to enlarge, which may be followed by the central structures being displaced, a tentorial herniation, and the patient's death. When a middle cerebral artery stroke is complete, the prognosis is quite bad.[59]

Patients who exhibit bilateral pyramidal tract indications, generalised seizures, or aberrant breathing patterns are at a very high risk of dying. These neurological symptoms are indicative of brain stem malfunction.[60]

TREATMENT

Medical treatment of stroke

There are two stages to treating a stroke: preventive and immediate treatment.

Primary prevention

Those who have never had a stroke before can still use the fundamental preventive strategies. As a main preventive measure, HMG CoA inhibitors, often known as statins, and other platelet anti-agrregants may be used.

Platelet antiaggregants

Aspirin is the recommended drug for stroke prevention. Dosage remains same for both type of stroke. Young, healthy individuals without vascular risk factors are not recommended to take antiplatelet drugs, such as low-dose aspirin. It is recommended that those with cardiovascular risk factors take aspirin at a modest dosage. Platelet antiaggregants may necessitate a transfusion and raise the risk of gastrointestinal bleeding. For primary prevention, other drugs like dipyridamole, ticlopidine, and clopidogril have also been studied.[61]

HMG Co-A reductase inhibitors

A Scottish study found that while statins, such as pravastatin, helped against coronary heart disease, they had no discernible impact on the prevention of stroke in individuals with moderate hypercholesterolemia.[62]

Secondary prevention

It is advised that people who have experienced a stroke take the secondary preventive measures. The two main approaches are the application of anti-platelet drugs such as Statins and modification of lifestyle including exercise. Additional therapies include thrombin inhibitors and antihypertensives.

Platelet antiaggregants

Aspirin

According to research, taking aspirin can reduce relative risk by 20–25%. The recommended daily aspirin dosage is 325 milligrammes, yet this is still up for debate. 325 milligrammes of aspirin per day is advised by research conducted in the USA and

Britain.58, 59 In India, taking aspirin on a modest daily dose is the current practice. Since Indians have demonstrated intolerance to high aspirin dosages, 75–150 mg of aspirin should be taken daily. Aspirin side effects include hearing loss, tinnitus, and arthritis.

Ticlopidine

Patients with mild strokes experienced a 12% relative risk reduction, according to a research. Ticlopidine 250 mg twice a day is the recommended dosage. The individuals experienced diarrhoea, dermatitis, and reversible agranulocytosis as side effects.[65]

Clopidogrel

Studies contrasting aspirin and clopidogrel have demonstrated a 9% decrease in relative risk for MI, stroke, and mortality. Ticlopidine, on the other hand, doesn't need blood monitoring. A daily dose of 75 mg is advised. Side effects are same as aspirin.

Dipyridamole

A research noted that, dipyridamole, given twice daily in an extended release formulation is effective when compared to a placebo in preventing stroke. People cannot tolerate higher dosages due to the giddiness that is a side effect, and the medication's efficacy has not been shown to be greater than that of aspirin.[66]

HMG – CoA reductase inhibitors

Those with coronary disease, including those with normal serum cholesterol, had a 32% relative risk reduction in stroke risk when taking pravastatin medication. Simvastatin has a proven role in secondary prevention in coronary heart disease and high cholesterol.[66]

Antihypertensives

It is possible to utilise medications that lower blood pressure, such as ACE inhibitors, thiazide diuretics and angiotensin receptor blockers (ARBs).

Lifestyle modification

Giving up smoking and managing blood pressure The lifestyle intervention approaches to prevent stroke are diabetes control, regular exercise, weight loss, and low-fat diets (such as Dietary Approaches to Stop Hypertension, or DASH). Written prescriptions for exercise and specific drugs to help with quitting smoking can improve the chances of these interventions succeeding.

Acute treatment

Patients suffering from cerebral ischemia ought to be admitted to a hospital for additional assessment and care. Under close nursing and medical supervision, patient must get treatment in ICU or stroke unit.

Supportive treatment

If necessary, airway protection should be maintained while administering oxygen as part of the supportive treatment. It is important to avoid aspiration and atelactasis and to take the necessary precautions against infection, deep vein thrombosis, and pulmonary embolism. It's crucial to have adequate blood pressure control. The hypovolemia that exacerbates myocardial infarction and results in hypotension allows for the management of the fluid and electrolyte balance. It's imperative to treat the fever appropriately.[10]

Medical Management

Anti edema treatment

The recommended treatment is mannitol 20%, which should be administered at a dose of 1 gm/kg body weight over 20 to 30 minutes. Afterwards, 50 g should be taken every 2 to 3 hours, depending on the patient's clinical reaction. First, cytogenic edoema and then vasogenic edoema are caused by an ischemic stroke.

Thrombolysis

According to a study, intravenous rtPA was clearly helpful for certain individuals with acute stroke. The NINDS study found that inyravenous rtPA (0.9 mg/kg to a 90 mg max; 10% as a bolus, then the reminder over 60 mins) outperformed a placebo in individuals with ischemic stroke within three hours after onset. Half of the patients underwent therapy in less than ninety minutes, according to the study.

Anti-platelet Drugs

Antiplatelet medications may stop atherothrombotic occurrences. Dipyridamole, aspirin, clopidogrel, and ticlopidine are medications used as antiplatelet agents.

Aspirin

The most extensively researched antiplatelet agent is aspirin. It causes platelets' cyclooxygenase enzyme to become acetylated. This medication prevents the production of thromboxane A2, a substance that constricts blood vessels and aggregates platelets. Although this medication is cheap and can be taken in small doses, stomach irritation or ulcers are the most frequent side effects. The medication works equally well when used at low (30–75 mg daily) and high (daily 650–1300 mg) dosages.

Ticlopidine

This drug prevents ADP from binding to platelets and triggering platelet aggregation by binding to the ADP receptors on the platelets. It is rarely used and induces neutropenia.

Dipyridamole

Platelet phosphodiesterase inhibition, which is in charge of breaking down cyclic AMP, is how dipyridamole works. Although it has been demonstrated to be more effective, patients cannot tolerate high dosages of it because of giddiness and other negative effects.

Clopidogrel

Clopidogrel functions in a manner akin to that of ticlopidine, yet it is both as safe and efficacious as aspirin.

Anticoagulation

Research has indicated a questionable involvement of anticoagulation in cerebral ischemia caused by atherothrombosis. There is no proof to suggest that low molecular weight heparin is preferable to aspirin.There was no further benefit from the aspirin, and there have been reports of increased bleeding rates.[10]

Neuro protection

The idea of neuro protection strengthens the brain's ability to withstand ischemia. Although it hasn't been thoroughly investigated, patients going into cardiac arrest may benefit from hypothermia as a therapeutic option. Drugs that block excitatory amino acid

pathways have been found to protect neurons and glia in animals, despite a wealth of clinical data to the reverse.[10]

Surgical therapy

It has been demonstrated that carotid endarterectomy in 70 – 80% high grade is beneficial for sympathetic lesions of high grade carotid stenosis. Incidence of ipsilateral hemisphere stroke is effectively decreased by the surgical treatment. In certain centres, percutaneous angioplasty, also known as balloon angioplasty, has been tested with positive results.[10]

Rehabilitation

A comprehensive physical, occupational, and speech treatment programme is necessary for the proper rehabilitation of stroke victims. Among other effects of immobility, pressure sores, muscle contractures, pneumonia, DVT, and pulmonary embolism can be avoided by telling the patient and their family about their neurologic impairment. Patients should receive support and direction in order to overcome the impairment. The two major goals of rehabilitation are to maximise recovery and get the patient back home. This is achieved by offering a gradual, safe regimen customised for every patient. Even years after a stroke, hemiparesis has been demonstrated to improve with restraint therapy, suggesting that physical therapy may be able to wake up dormant neural networks.[10]

PLATELET INDICES

PLATELET COUNT (PC)

Between one and half lakhs to four lakhs of platelets per cubic centimetre of blood is thought to be normal. Thrombocytosis denotes a larger platelet count, while thrombocytopenia denotes a lower level.

There are four primary ways that thrombocytopenia can occur.

1. A greater loss of platelets in the periphery
2. A reduction in platelet synthesis
3. Irreversible thrombocytopenia
4. Containment in an enlarged spleen.

Platelet counts below 1,50,000/cmm are associated with severe haemorrhage, while counts between 1,50,000 to 50,000/cmm are typically associated with mild spontaneous bleeding and bleeding after surgery or trauma. Platelets below 20,000/cmm are associated with severe haemorrhage.

There are main and secondary aetiologies for thrombocytosis. Idiopathic myelofibrosis, Polycythemia Vera, essential thrombocytopenia, and chronic myeloid leukaemia are a few of the main causes.

The following are examples of secondary or reactive aetiology: splenectomy, cancer, trauma, infections, haemorrhage, and chronic inflammatory disease.

The platelet count is somewhat elevated in reactive thrombocytosis, although it is not clinically significant.

MEAN PLATELET VOLUME (MPV)

The platelet count is somewhat elevated in reactive thrombocytosis, although it is not clinically significant. However, if hemolysis is not completely cured in chronic

myeloid leukaemia, prolonged thrombocytosis after splenectomy may increase the risk of thromboembolic events.[67]

A large increase in mean platelet volume is connected with enhanced adhesion, aggregation, exposure of the glycoprotein receptor on the platelet surface, and higher fibrinogen binding. These variables modify the metabolism of platelets and the interplatelet signalling pathway, which ultimately impairs a number of metabolic pathways, including the synthesis and release of thromboxane A2, increased metabolism of calcium, and the creation of ADP. Increased platelet sensitivity in diabetes mellitus may result in a platelet volume gradient, an increase in platelet turnover, or a decrease in platelet survival. The discharge of substances from platelet granules may also be connected to it.

There may be a connection between MPV and the frequency of cardiovascular problems in type 2 diabetes. A growing amount of evidence indicates that MPV poses a serious danger for the vascular complications linked to type 2 diabetes, and because of increased platelet activity, type 2 diabetes is believed to be a prothrombotic state. Therefore, increased MPV in diabetes mellitus may cause thrombotic vascular issues by acting as a procoagulant. Nevertheless, MPV can also be increased by a few coronary artery risk factors, including type 2 diabetes, smoking, hypertension, and hypercholesterolemia. [67]

PLATELET DISTRIBUTION WIDTH (PDW)

Platelet heterogenicity, which can be caused by heterogeneous megakaryocyte demarcation and platelet ageing, is measured by platelet distribution width. A high PDW suggests a significant anisokaryocytosis. Platelet activity is measured by platelet distribution width, in addition to enhancing the heterogeneity of the platelet volume

distribution. Numerous investigations have demonstrated that larger and more numerous platelets may have an impact on the width of the platelet distribution, which may contribute to the pathophysiology of vascular problems.[68]

PLATELETCRIT:

PCT is the volume of blood that platelets occupy as a percentage, determined using the equation

$$PCT = \text{platelet count} \times MPV / 10{,}000$$

REVIEW OF RELATED LITERATURE

In a study by OT et al, the MPV was associated with the traditional risk factors for ischemic stroke and its outcome using the modified Rankin scale [mRS]. A statistically significant relationship between MPV and carotid intima media thickness (CIMT), type 2 diabetes mellitus, and hypertension was found in the study. Furthermore, there was a positive association (correlation coefficient of 0.818) between the MPV at presentation and mRS, indicating that higher MPV was linked to more severe impairment. Predicting the degree of stroke and neurological recovery at the time of ischemic stroke presentation may be possible with the use of the MPV. To support its prognostic value in AIS, a larger investigation with a diverse sample is necessary.[69]

In a study by Shah et al, control group had a greater platelet count and a lower mean platelet volume. The study group had increased PDW and PLCR.By the end of the first week, 57% of stroke cases were self-sufficient. After first week, patients with a high mean platelet volume performed worse than the control group (p value=<0.05). Regardless of the kind of stroke, mean platelet volume and the immediate prognosis from an ischemic stroke are inversely correlated.[70]

In a research by Yao et al., multivariate logistic regression analysis revealed that the NIHSS, TOAST classification, hyperglycemia, and mean platelet volume (MPV) were significant predictors of clinical outcomes in AIS patients after three months. Higher MPV has been associated with a worse three-month prognosis, especially in stroke patients with large-artery atherosclerosis, according to research. Platelet count and MPV have a negative correlation. In patients who have a positive result, there is a strong linear association between MPV and the platelet-to-lymphocyte ratio (PLR), as well as between MPV and platelet distribution width (PDW). Overall, individuals with AIS who received thrombolytic therapy and had lower NIHSS and MPV levels at admission had better functional results. Further research is needed to see whether the correlations between MPV, PDW, and PLR can be used to assess stroke patients' prognoses.[71]

The LGI score and the baseline and day 7 NIHSS scores shown a positive connection, respectively, in a study by Zhou et al. Multivariate regression research revealed that LGI score as an independent predictor of both END and the severity of the stroke. Crude model indicated that the LGI score in the fourth quartile was associated with a higher likelihood of unfavourable outcomes on day 90 when compared to the LGI score in the first quartile. After adjusting for pertinent covariates, a poor outcome on day 90 was found to be independently correlated with an LGI score in the fourth percentile. Finally, on day 90 after the stroke started, the ROC analysis showed a poor outcome. The LGI score has a strong correlation with the severity of acute ischemic stroke and may be a better predictor of poor outcomes for individuals 90 days following an acute ischemic stroke.[72]

MATERIALS AND METHODS

MATERIAL AND METHODS

An observational study was conducted September 2022 and December 2023 on patients referred to Department of General Medicine at R.L. Jalappa Hospital and Research Center attached to SDUMC, Kolar. Before starting the approval from institution ethics committee was obtained. An informed consent was obtained from all the cases before including them in to the study. The sample size calculation was as follows,

Sample size:

The correlation coefficient (r) between PLR and Left CIMT, which is 0.398 (i.e., r = 0.398), was used to assess the sample size in the Yilmaz et al. study. With 80% power and 95% confidence level, these numbers were substituted into the formula below to provide a sample size of 42. A sample size of 42 + 4.2 = 47 participants was included in the study, accounting for a 10% non-response rate.

Total sample size = $N = [(Z\alpha+Z\beta)/C]^2 + 3$

$Z\alpha$ = Standard normal deviate for $\alpha = 1.960$

$Z\beta$ = Standard normal deviate for $\beta = 0.842$

r = Correlation coefficient = 0.398

$C = 0.5 * \ln[(1+r)/(1-r)]$

The inclusion and exclusion criteria were as follows,

Inclusion criteria

1. Patients aged more than 30 years admitted to RLJH with diagnosis of acute ischemic stroke-diagnosed with CT/MRI, and who give informed consent.

Exclusion criteria

1) Cardio embolic Stroke
2) Stroke due to hemorrhage
3) Large platelet hereditary disorders of known cases
4) Drugs that lower platelet count, such as antineoplastic drugs and hydroxyurea.
5) Individuals using antiplatelet drugs such as clopidogrel or aspirin.

METHOD OF DATA COLLECTION

DATA ACQUISITION

History about the beginning, length, and course of the stroke was gathered, together with information about risk factors such as smoking, diabetes, hypertension, kidney failure, and other pertinent medical conditions.

All vital signs, such as heart rate, blood pressure, and respiration rate, were recorded and continuously monitored upon admission.

1. Basic blood investigations

Platelet indices, or CBC, are gathered into EDTA tubes and processed in an hour. Samples were kept at room temperature in between collection and processing.

2. CT brain/MRI.

3. CAROTID DOPPLER

B-mode ultrasound will be used to assess the carotid intima-media thickness (CIMT) at a distance of one centimetre from the carotid bulb.

In supine position and three carotid artery (common artery) sites were examined via a neck-extended ultrasonography procedure.

4.2 D ECHO: Done to rule out cardio embolic stroke.

Statistical methods:

After entering the data into an Excel sheet, the data was analysed using SPSS 22 version. To represent the categorical variables, proportions and frequencies were utilised. The chi-square test or Fischer's exact test (for 2x2 tables only) was the significant test for qualitative data. Yates adjustment was applied when the chi-square rules were not satisfied (for 2x2 tables only). Continuous variables were represented using the mean and standard deviation. The independent t test or the Mann Whitney U test was used as a significant test to find the mean difference, respectively, between two qualitative and quantitative variables.

Data visualisation: A range of graphs, including scatter plots, pie charts, and bar charts, were made using Microsoft Word and Excel.

A P value (the probability that the result is true) of less than 0.05 was deemed statistically significant.

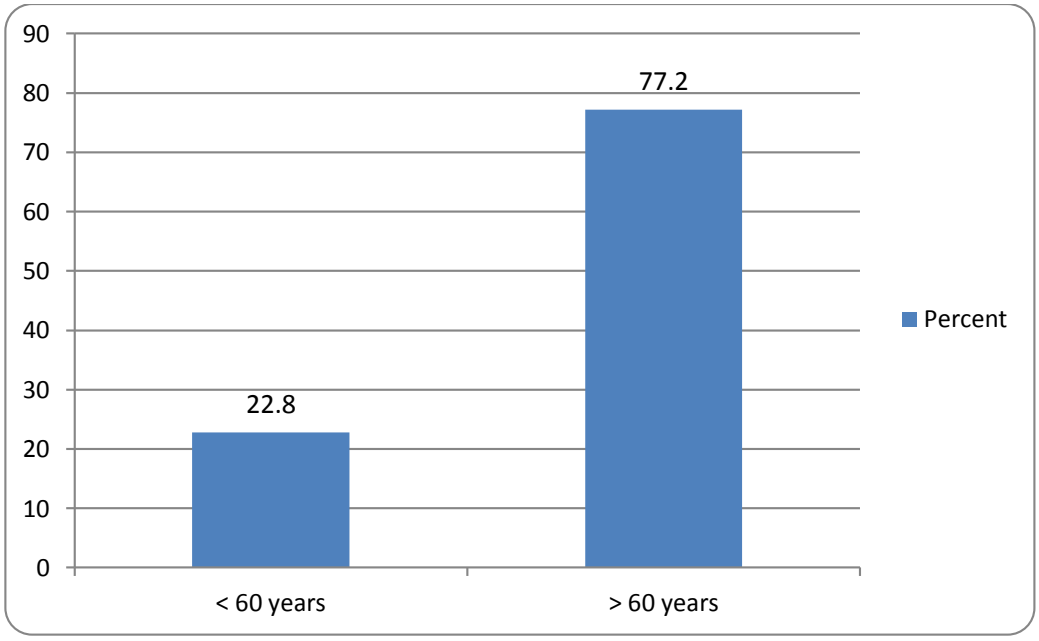
RESULTS

RESULTS

Table 1. Age of study group

Age group	Frequency	Percent
< 60 years	13	22.8
> 60 years	44	77.2
Total	57	100

Chart 1. Age of study group

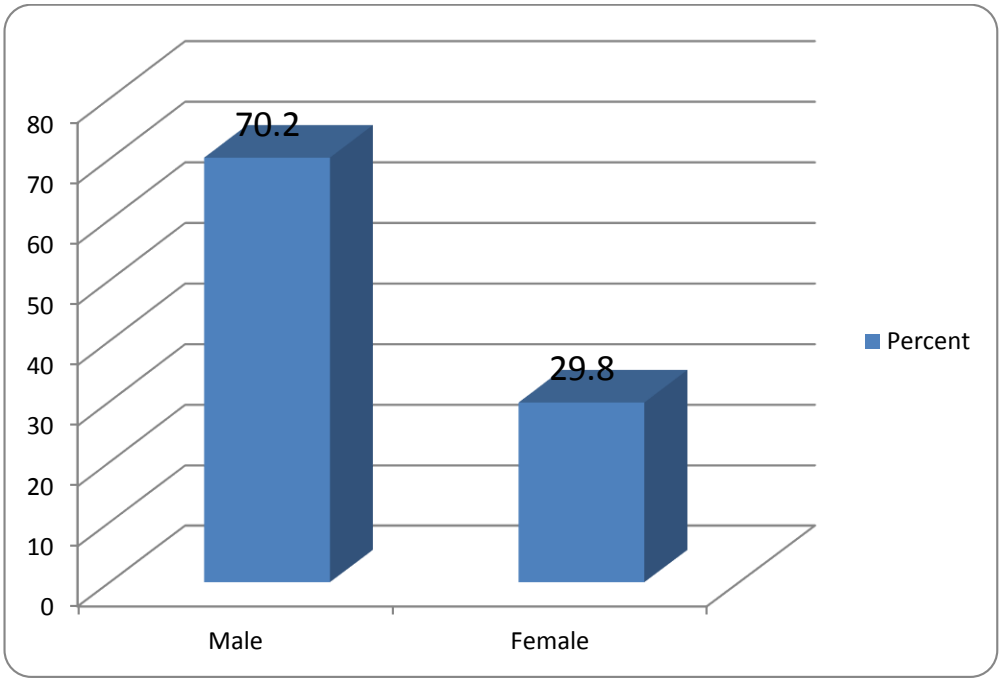


According to this study, 77.8% of the instances included people who were older than 60.

Table 2. Sex of study group

Sex	Frequency	Percent
Male	40	70.2
Female	17	29.8
Total	57	100

Chart 2. Sex of study group

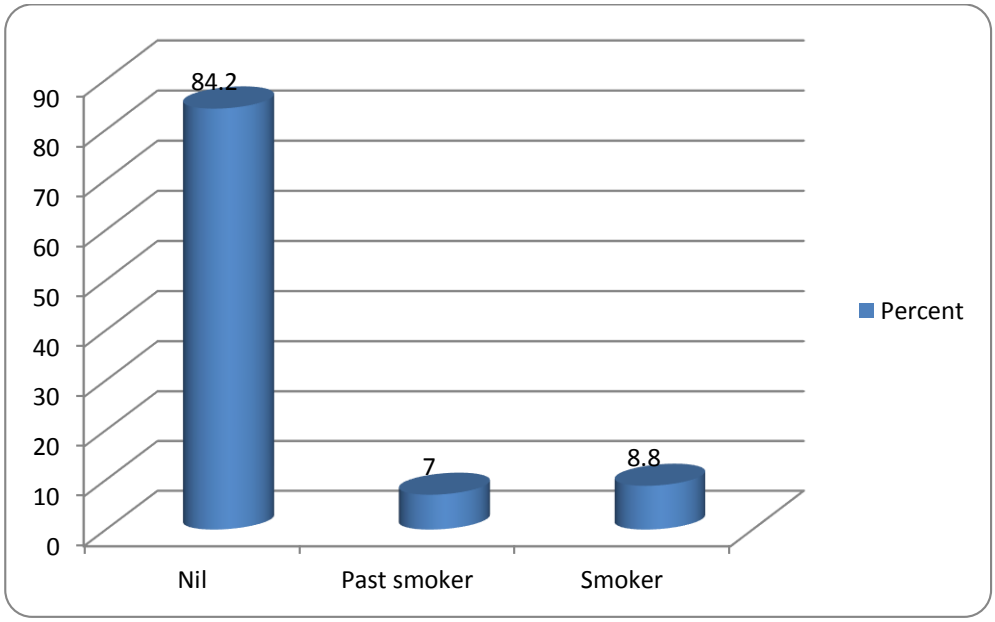


About 70.2% of the cases in this study were males.

Table 3. Smoking status of the study group

Smoking status	Frequency	Percent
Nil	48	84.2
Past smoker	4	7.0
Smoker	5	8.8
Total	57	100

Chart 3. Smoking status of the study group

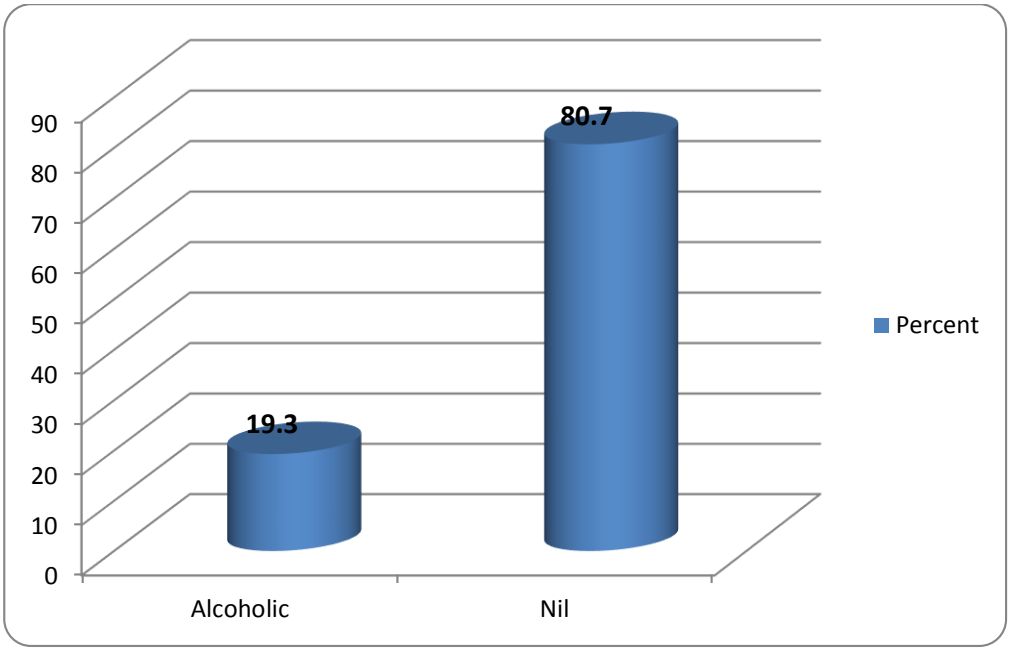


About 7.0% of the cases were past smokers and 8.8% of the cases were smokers.

Table 4. Alcohol status of the study group

Alcohol	Frequency	Percent
Alcoholic	11	19.3
Nil	46	80.7
Total	57	100

Chart 4. Alcohol status of the study group

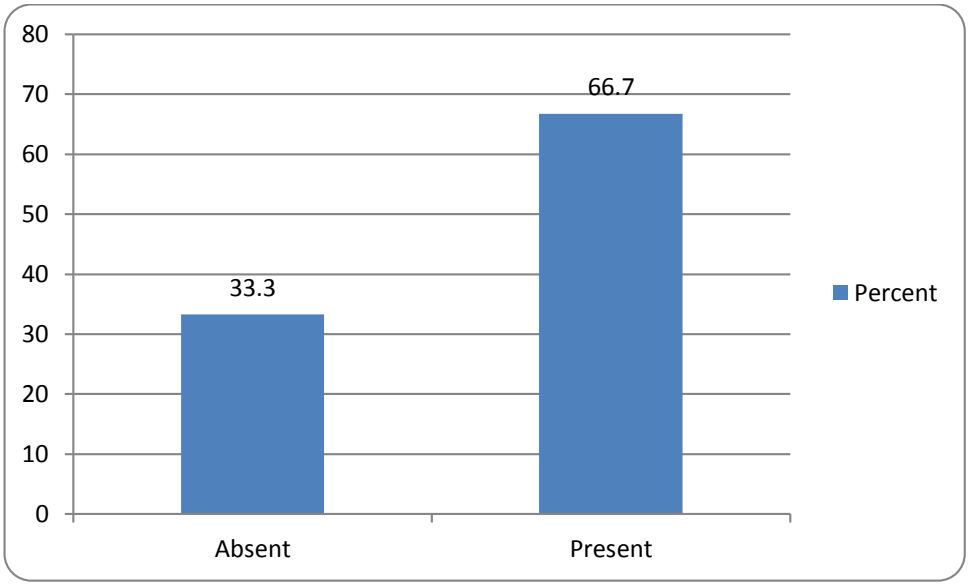


Alcoholics made up about 19.3% of the cases in our investigation.

Table 5. Hypertension of study group

Hypertension	Frequency	Percent
Absent	19	33.3
Present	38	66.7
Total	57	100

Chart 5. Hypertension of study group

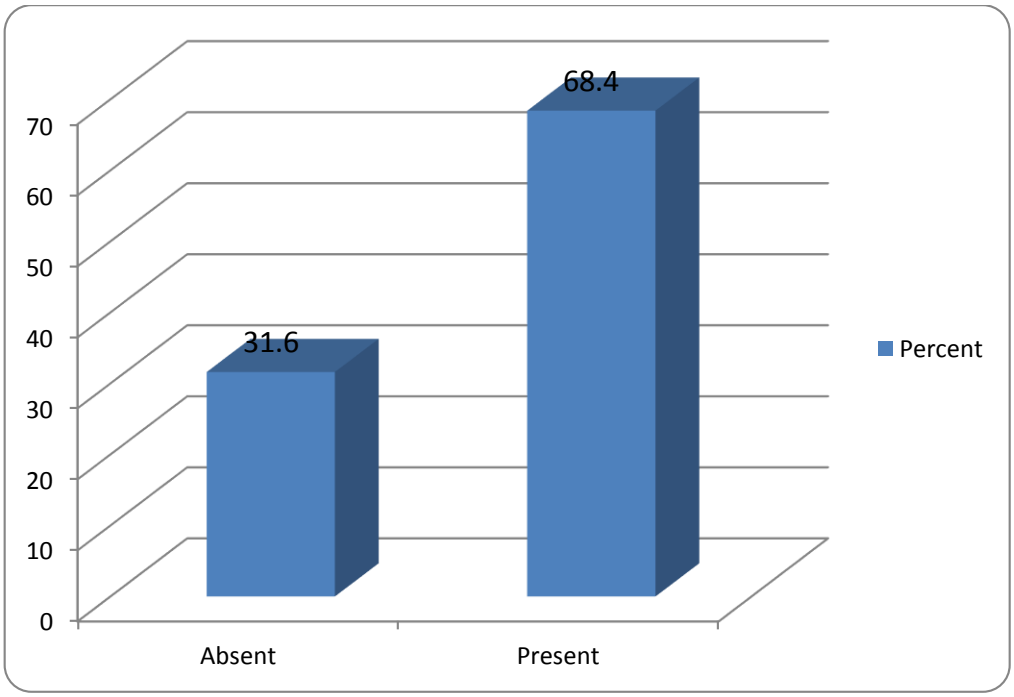


In this study, 66.7% of the cases had hypertension.

Table 6. Diabetes of study group

Diabetes	Frequency	Percent
Absent	18	31.6
Present	39	68.4
Total	57	100

Chart 6. Diabetes of study group

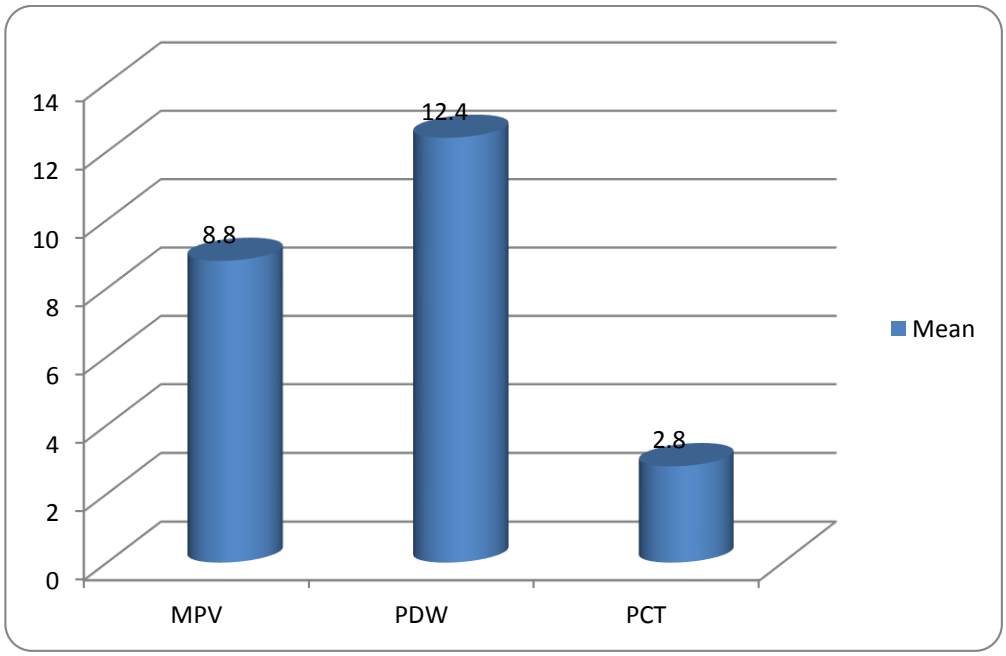


About 68.4% of the cases in this study had diabetes mellitus.

Table 7. Platelet parameters of the study group

Platelet parameters	Mean	SD
MPV	8.8	1.2
PDW	12.4	1.8
PCT	2.8	0.6

Chart 7. Platelet parameters of the study group

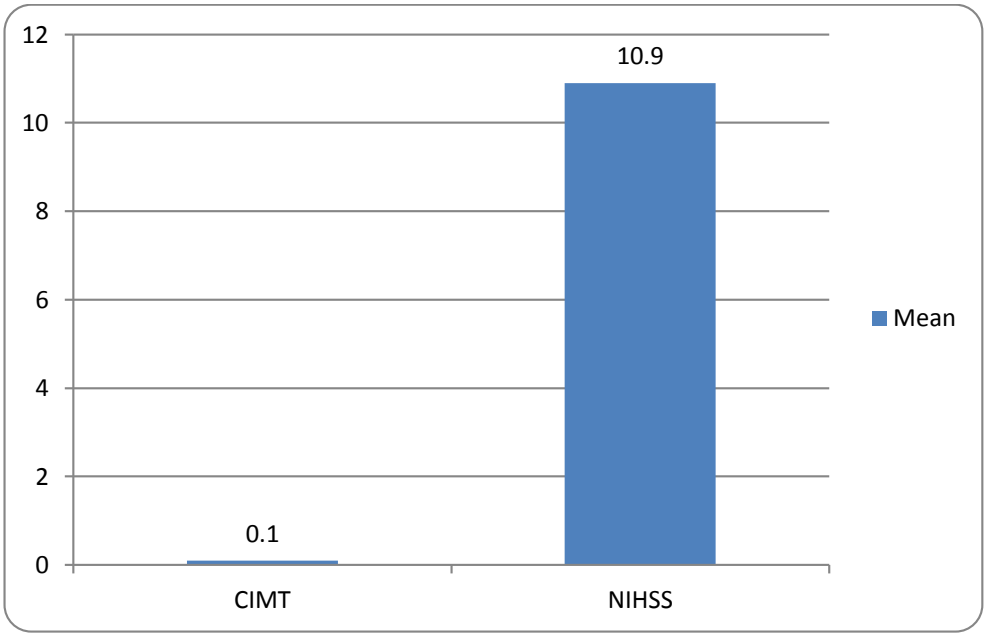


The mean platelet volume in this study was 8.8, platelet distribution width was 12.4, plateletocrit was 2.8 in this study.

Table 8. CIMT and NIHSS scores of study group

CIMT and NIHSS	Mean	SD
CIMT	0.1	0.1
NIHSS	10.9	6.9

Chart 8. CIMT and NIHSS scores of study group

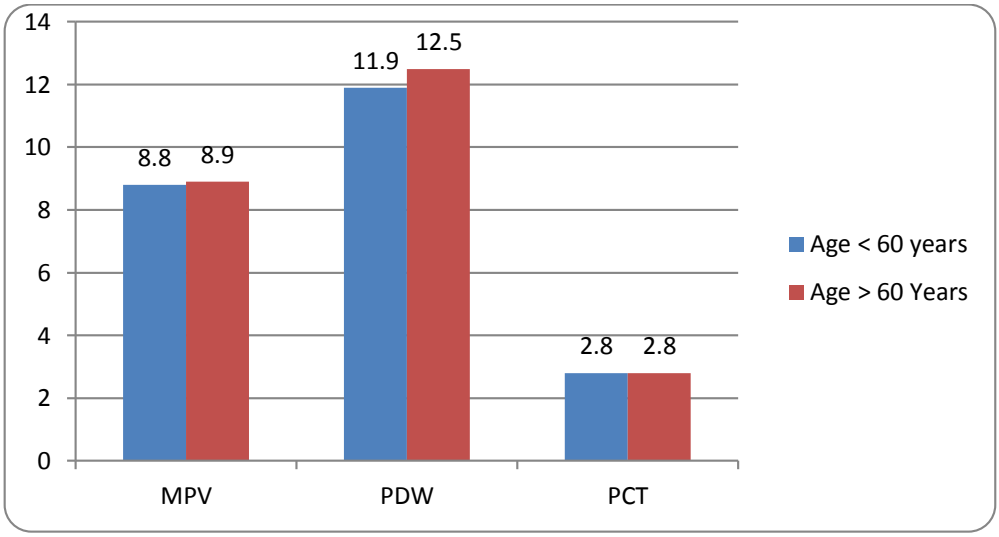


The mean Carotid intima media thickness was 0.1 and mean NIHSS scores was 10.9 in this study.

Table 9. Age, sex and platelet indices of study group

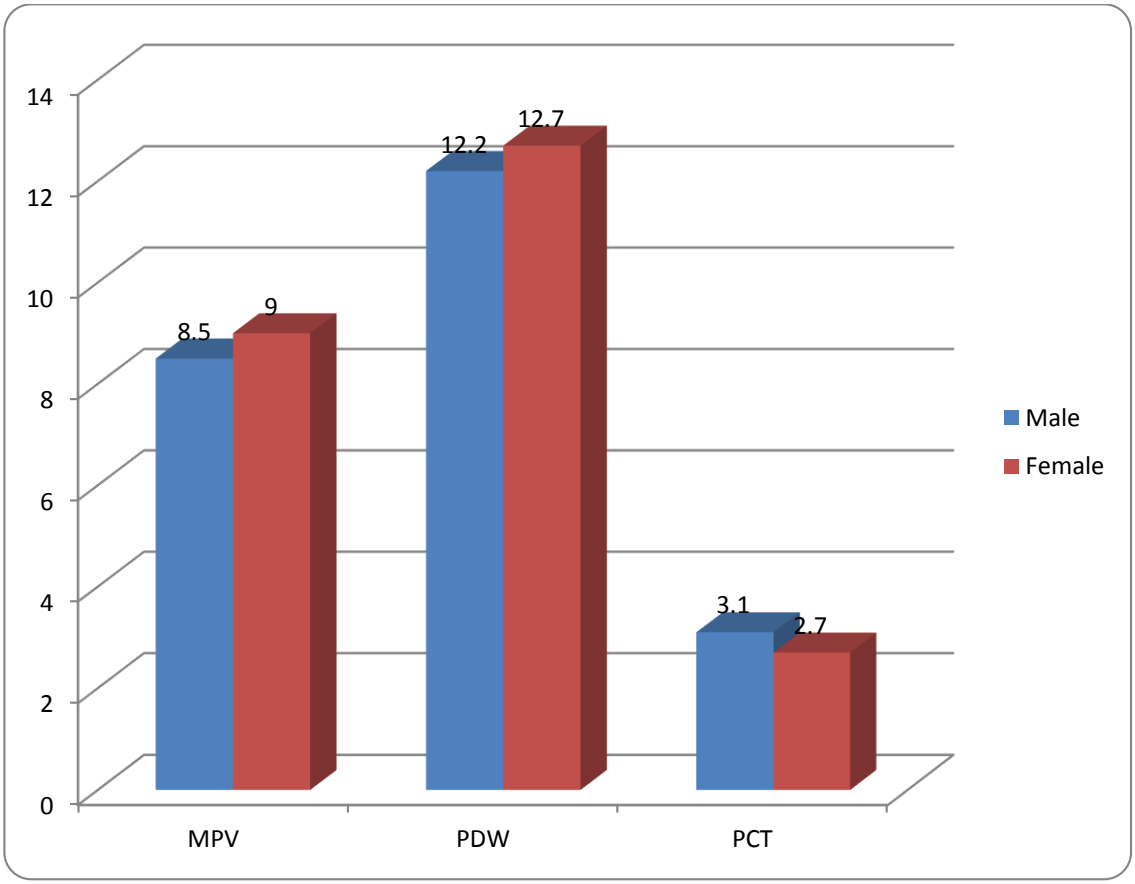
		MPW	PDW	PCT
Age	**< 60 years**	8.8	11.9	2.8
	> 60 years	8.9	12.5	2.8
Sex	**Male**	8.5	12.2	3.1
	Female	9.0	12.7	2.7

Chart 9. Age, sex and platelet indices of study group



Mean platelet volume was 8.8 in cases aged less than 60 years and 8.9 in cases aged more than 60 years. The platelet distribution width was 11.9 in less than 60 years and 12.5 in those who were aged more than 60 years, The mean plateletocrit was 2.8 in cases aged more and less than 60 years.

Chart 10. Sex and platelet indices of study group

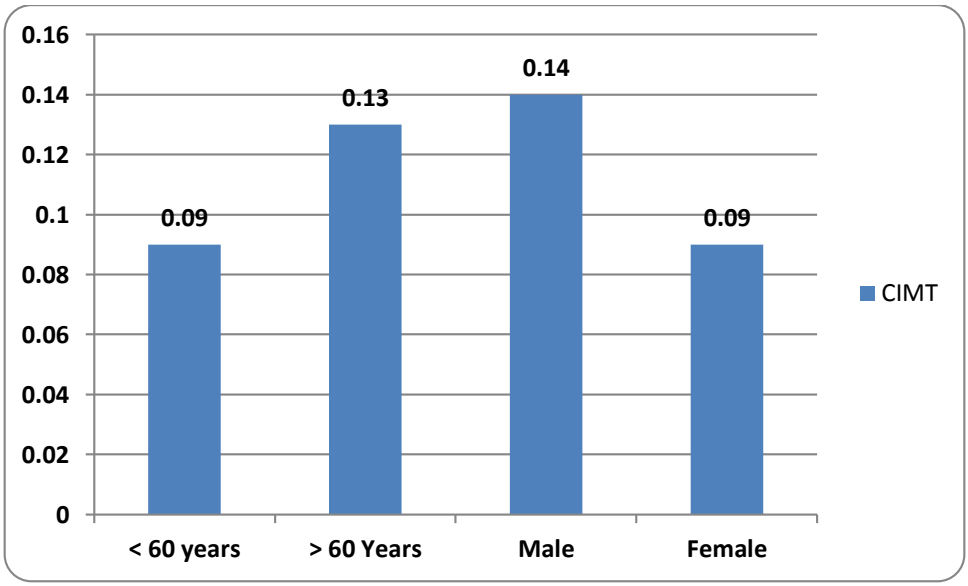


The mean platelet volume in males was 8.5 and females was 9, platelet distribution width was 12.2 in males and 17.7 in females and plateletocrit was 3.1 in males and 2.7 in females.

Table 10. Age, sex and CIMT & NIHSS of study group

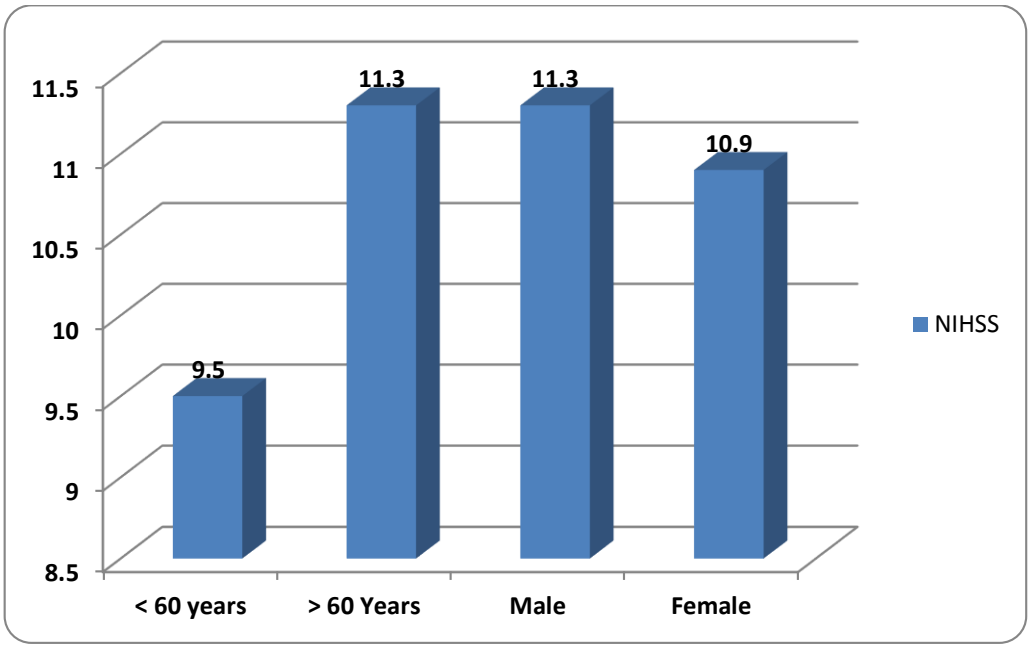
		CIMT	NIHSS
Age	< 60 years	0.09	9.5
	> 60 years	0.13	11.3
Sex	Male	0.14	11.3
	Female	0.09	10.9

Chart 11. Age, sex and CIMT & NIHSS of study group



The mean CIMT was 0.09 in cases aged less than 60 years and 0.13 in cases aged more than 60 years. The mean CIMT was 0.14 in males and 0.09 in females.

Chart 12. Age, sex and NIHSS of study group



Mean NIHSS was 9.5 in cases aged less than 60 years and 11.3 in cases aged more than 60 years. The mean NIHSS was 11.3 in males and 10.9 in females.

Table 11. Correlation of platelet indices and CIMT

		CIMT
MPV	r	-.012
	P value	.930
	N	57
PDW	r	-.200
	P value	.136
	N	57
PCT	r	-.090
	P value	.507
	N	57

CIMT had negative but not significant correlation with MPV, PDW and PCT in this study.

Table 12. Correlation of platelet indices and NIHSS

		NIHSS
MPV	r	-.059
	P value	.661
	N	57
PDW	r	.197
	P value	.143
	N	57
PCT	r	.521**
	P value	.000
	N	57

The Pearson correlation coefficient was negative for MPW. The Pearson correlation coefficient was positive but not significant for PDW, positive and significant for PCT with NIHSS.

DISCUSSION

DISCUSSION

A stroke is characterised by the sudden onset of a localised neurological impairment that has a vascular origin. Stroke ranks third globally in terms of years lived with a handicap and is the second most prevalent reason for death worldwide. According to reports, the prevalence of stroke in India is 1.27–2.20 per 1000 people.[1, 2]

Even though a lot of risk factors have been connected to stroke, they can only explain 50% of stroke cases.[8] The risk factors for hypertension, which include obesity, inflammation, high-fat diets, sedentary lifestyles, and smoking, can be changed in order to reduce the chance of an ischemic stroke.[9]

Because it speeds up the process, platelet activation and aggregation are important factors in the pathophysiology of acute ischemic stroke. Rupture of an atherosclerotic plaque is the main cause of arterial thrombosis. Plaque accumulation in the intima of the vessel is the cause of atherosclerosis. Fibrofatty streaks containing lipids and foam cells make up this plaque. When this bursts, a thrombus forms around it, and platelets work in a subsequent mechanism to form a blood clot.[11]

Vasoconstriction is first induced by local causes following vascular damage to the vessel intima as a result of a plaque rupture. New platelets are then produced, and these attach to the injury site. Once activated, they further promote platelet aggregation via the GpIIb-IIIa receptor, which attaches to fibrinogen to form the "primary hemostatic plug". Following this, fibrin is produced and the clotting factors are activated, producing the final secondary hemostatic plug.[12]

Thus, much more effectively, the morphological and physiological structure of platelets—which may be ascertained by MPV, PDW, and PLATELETCRIT plays a significant role in determining the severity of the stroke.[14]

A commonly used standardised method of assessing stroke symptoms and indicators is the 15-item NIHSS scale. Higher score denote greater severity. This score is connected with platelet indices because it has an acceptable sensitivity of 72% and specificity of 95%.[15]

This study's goal is to evaluate and link different platelet indices and carotid intima media thickness with stroke patients' functional outcome and severity, as determined by NIHSS rating.

In September 2022 and December 2023, a study of observation was carried out on patients who were referred to the Department of General Medicine. The study had a sample size of 47 participants.

Age group

According to this study, 77.8% of the instances included people who were older than 60. The findings of this study are consistent with Lok et al where the mean of age was 72.3 years.[73] According to a research by Govind et al, 45–60 age range accounted for almost 62.5% of the cases.[74] The average age of the cases in a research by Cao et al. was 66.81 years.[75]

Sex

Males made up about 70.2% of the cases in this investigation. Males made up the bulk of the cases in a research by Lok et al.[73] Govind et al had noted that, majority of the cases were males.[74] Cao et al had noted that, about 68.0% were males.[75]

Smoking status

About 7.0% of the cases were past smokers and 8.8% of the cases were smokers. Lok et al had reported that, 29.2% of the cases had habit of smoking.[73] Govind et al had shown that, about 90.6% of the males and 18.7% of the females were smokers.[74]

Alcohol status

Alcoholics made up about 19.3% of the cases in our investigation. According to a study by Govind et al., there were no female alcoholics and around 87.5% of the male participants.[74]

Hypertension

Hypertension was present in 66.7% of the cases in this study. Lok et al reported 74.0% of the cases had hypertension.[73] Govind et al noted 87.5% of the males and 75.0% of the females had hypertension.[74] Cao et al noted, about 77.7% of the cases were hypertensives.[75]

Diabetes mellitus

About 68.4% of the cases in this study had diabetes mellitus. Lok et al had noted that, about 29.6% of the cases had diabetes mellitus.[73] In a study by Govind et al, 81.2% of the males and 62% of the females had diabetes mellitus.[74] Cao et al had reported that, about 39.5% had diabetes mellitus.[75]

Platelet indices

The mean platelet volume in this study was 8.8, platelet distribution width was 12.4, plateletocrit was 2.8 in this study. In a study by Lok et al, the mean platelet volume was 8.7.[73]

Carotid intima media and NIHSS scores

The mean Carotid intima media thickness was 0.1 and mean NIHSS scores was 10.9 in this study. Lok et al had shown that, the NIHSS score was 6 in their study.[73]

Age with Platelet indices

Mean platelet volume was 8.8 in cases aged less than 60 years and 8.9 in cases aged more than 60 years. The platelet distribution width was 11.9 in less than 60 years and 12.5 in those who were aged more than 60 years, the mean plateletocrit was 2.8 in cases aged more and less than 60 years.

Sex with Platelet indices

The mean platelet volume in males was 8.5 and females was 9, platelet distribution width was 12.2 in males and 17.7 in females and plateletocrit was 3.1 in males and 2.7 in females.

Age with CIMT

The mean CIMT was 0.09 in cases aged less than 60 years and 0.13 in cases aged more than 60 years. The mean CIMT was 0.14 in males and 0.09 in females.

Age with NIHSS scoring

Mean NIHSS was 9.5 in cases aged less than 60 years and 11.3 in cases aged more than 60 years. The mean NIHSS was 11.3 in males and 10.9 in females.

Correlation of CIMT with Platelet indices

CIMT had negative but not significant correlation with MPV, PDW and PCT in this study.

Correlation of NIHSS with Platelet indices

The pearson correlation coefficient was negative for MPW. The pearson correlation coefficient was positive but not significant for PDW, positive and significant for PCT with NIHSS. In a study by Govind et al, the mean platelet volume was 12.48 in cases with NIHSS scores of less than 6 and 12.87 in cases with NIHSS score of more than 6.[74] Platelet distribution width was 18.42 in cases with NIHSS score of less than 6 and 18.48 in cases in NIHSS of more than 6.[74]

LIMITATIONS

LIMITATIONS

The limitations of our study include a small sample size, done in a single hospital setting, correlation of whole blood clotting with the severity of the envenomation was not done, species of the snake was not included and a non-randomized study. Also, time from the snake bite to the index test is not included which can be another probable confounding variable while evaluating the efficacy of WBCT. A larger study with a higher sample size is required to find the validation of Whole blood clotting time and PT/INR following administration of ASV.

CONCLUSION

CONCLUSION

This study was undertaken to find out platelet indices as predictor for ischemic stroke and to find out its correlation with NIHSS scores. This study had shown that, PCT had a positive and significant correlation with the NIHSS scores. CIMT had no correlation with any platelet indices. But this study is not without limitations. The sample size was small to generalize the study findings and it was an observational study. A longitudinal study with larger sample size can bring out more facts about the use of platelet indices in prediction of acute ischemic stroke.

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ANNEXURES

PROFORMA

Particulars of the patients

NAME:

AGE: ____ YEARS

SEX: MALE/FEMALE

OCCUPATION:

LOCATION:

HOSPITAL NUMBER:

DATE AND TIME OF ADMISSION : __/__/20__ AT __:__ AM/PM

DATE OF DISCHARGE:__/__/20__

ADMISSION DIAGNOSIS:

BRIEF HISTORY:

SYMPTOMS ON PRESENTATION:

□ Icterus	□ Dizziness	□ gastritis
□ Nausea	□ Weakness	□ amenorrhea
□ Vomiting	□ Abdominal Pain	□ Peripheral edema/swelling
□ Palmar erythema	□ Reduced appetite	
□ Hematemesis	□ dyspepsia	□ Restlessness
□ Headache	□ Hematemesis	

PRIOR TREATMENT:

□ **YES** □ **NO**

PROVIDER : SUPPORTIVE : TREATMENT :

PAST HISTORY:

□ DIABETES MELLITUS	
□ HYPERTENSION	□ RENAL DISORDER
□ LIVER DISORDER	□ TUBERCULOSIS
□ CARDIOVASCULAR DISEASE	□ BRONCHIAL ASTHMA

PERSONAL HISTORY:

- DIET:
- APPETITE:
- SLEEP:
- BOWEL AND BLADDER:
- HABITS:
- SOCIOECONOMIC STATUS

GENERAL PHYSICAL EXAMINATION: Height: ____Cms , Weight: ____ kgs , BMI: ____kg / m^2

Pallor/Cyanosis/Icterus/Clubbing/edema/Generalized lymphadenopathy/Gynaecomastia/ Testicular atrophy/ Spider navi/ Pupura/ Petechiae/ Caput medusae

VITAL DATA

- Pulse: ___ bpm

a. Temperature:____^oF
b. BP: ____ mmHg
c. Respiration rate: ____ cpm
d. SpO2: ____% @ RA

Systemic examination :

- Per abdomen:
- Respiratory system:
- Cardio vascular system:
- Central nervous system:

INVESTIGATIONS:

Routine: CBC

- Platelets :…….. Thousands/mm^3
- MPV :
- Plateletcrit:
- PDW:
- 2 D ECHO:
- CT:
- MRI:

NIHSS SCORE :

INFORMED CONSENT FORM

Title: - Platelet indices as a predictor of carotid atherosclerosis in acute ischemic stroke correlation with NIHSS scoring.

Principal investigator: Dr.Maram Sanjana

I, Mr/Ms/Mrs. …….. Have been explained in my own understandable language, that I will be included in a study which is

I have been explained that my clinical findings, investigations, findings will be assessed and documented for study purpose.

I have been explained my participation in this study is entirely voluntary and I can withdraw from the study any time and this will not affect my relation with my doctor or treatment for my ailment.

I have been explained about the risk/ benefit of the study.

I understand that the medical information produced by this study will become part of institutional records and will be kept confidential by my said institute.

I agree not to restrict the use of any data or result that arise from this study provided such a use is only for scientific purpose(s).

I have principal investigator mobile number for enquiries.

I have been informed that standard of care will be maintained throughout the treatment period.

I, in my sound mind, give full consent to be added in the part of this study.

Investigator:

Dr. Maram Sanjana

Phone number- 80085 15588

Participant's signature/ thumb impression

Name:

Signature/thumb impression of the witness:

Date:

Name:

Relation to patient

ಮಾಹಿತಿ ನೀಡಿದ ಒಪ್ಪಿಗೆ

ಶೀರ್ಷಿಕೆ: - ತೀವ್ರವಾದ ರಕ್ತಕೊರತೆಯ ಪಾರ್ಶ್ವವಾಯು ಮತ್ತು NIHSS ಸ್ಕೋರಿಂಗ್‌ನೊಂದಿಗಿನ ಪರಸ್ಪರ ಸಂಬಂಧದಲ್ಲಿ ಶೀರ್ಷಧಮನಿ ಅಪಧಮನಿಕಾಠಿಣ್ಯದ ಮುನ್ಸೂಚಕವಾಗಿ ಪ್ಲೇಟ್‌ಲೆಟ್ ಸೂಚ್ಯಂಕಗಳು.

ಪ್ರಧಾನ ತನಿಖಾಧಿಕಾರಿ: ಡಾ.ಮರಮ್ ಸಂಜನಾ

ನಾನು, ಶ್ರೀ/ಮತಿ/ಶ್ರೀಮತಿ. ..... ನನ್ನ ಸಂಪೂರ್ಣ ಅರ್ಥವಾಗುವ ಭಾಷೆಯಲ್ಲಿ ವಿವರಿಸಲಾಗಿದೆ, ಅದು ಒಂದು ಅಧ್ಯಯನದಲ್ಲಿ ನನ್ನನ್ನು ಸೇರಿಸಿಕೊಳ್ಳಲಾಗುವುದು

ನನ್ನ ಕ್ಲಿನಿಕಲ್ ಸಂಶೋಧನೆಗಳು, ತನಿಖೆಗಳು, ಸಂಶೋಧನೆಗಳನ್ನು ಮೌಲ್ಯಮಾಪನ ಮಾಡಲಾಗುತ್ತದೆ ಮತ್ತು ಅಧ್ಯಯನ ಉದ್ದೇಶಕ್ಕಾಗಿ ದಾಖಲಿಸಲಾಗುತ್ತದೆ ಎಂದು ನನಗೆ ವಿವರಿಸಲಾಗಿದೆ.

ಈ ಅಧ್ಯಯನದಲ್ಲಿ ನನ್ನ ಭಾಗವಹಿಸುವಿಕೆಯು ಸಂಪೂರ್ಣವಾಗಿ ಸ್ವಯಂಪ್ರೇರಿತವಾಗಿದೆ ಮತ್ತು ನಾನು ಯಾವುದೇ ಸಮಯದಲ್ಲಿ ಅಧ್ಯಯನದಿಂದ ಹಿಂದೆ ಸರಿಯಬಹುದು ಮತ್ತು ಇದು ನನ್ನ ವೈದ್ಯರೊಂದಿಗಿನ ನನ್ನ ಸಂಬಂಧ ಅಥವಾ ನನ್ನ ಕಾಯಿಲೆಯ ಚಿಕಿತ್ಸೆಯ ಮೇಲೆ ಪರಿಣಾಮ ಬೀರುವುದಿಲ್ಲ ಎಂದು ನನಗೆ ವಿವರಿಸಲಾಗಿದೆ.

ಅಧ್ಯಯನದ ಅಪಾಯ/ಪ್ರಯೋಜನದ ಬಗ್ಗೆ ನನಗೆ ವಿವರಿಸಲಾಗಿದೆ.

ಈ ಅಧ್ಯಯನದಿಂದ ಉತ್ಪತ್ತಿಯಾಗುವ ವೈದ್ಯಕೀಯ ಮಾಹಿತಿಯು ಸಾಂಸ್ಥಿಕ ದಾಖಲೆಗಳ ಭಾಗವಾಗುತ್ತದೆ ಮತ್ತು ನಾನು ಹೇಳಿದ ಸಂಸ್ಥೆಯು ಗೌಪ್ಯವಾಗಿಡುತ್ತದೆ ಎಂದು ನಾನು ಅರ್ಥಮಾಡಿಕೊಂಡಿದ್ದೇನೆ.

ಈ ಅಧ್ಯಯನದಿಂದ ಉಂಟಾಗುವ ಯಾವುದೇ ಡೇಟಾ ಅಥವಾ ಫಲಿತಾಂಶದ ಬಳಕೆಯನ್ನು ನಿರ್ಬಂಧಿಸದಿರಲು ನಾನು ಸಮ್ಮತಿಸುತ್ತೇನೆ, ಅಂತಹ ಬಳಕೆಯನ್ನು ಕೇವಲ ವೈಜ್ಞಾನಿಕ ಉದ್ದೇಶ(ಗಳಿಗೆ)

ವಿಚಾರಣೆಗಾಗಿ ನಾನು ಪ್ರಧಾನ ತನಿಖಾಧಿಕಾರಿಯ ಮೊಬೈಲ್ ಸಂಖ್ಯೆಯನ್ನು ಹೊಂದಿದ್ದೇನೆ.

ಚಿಕಿತ್ಸೆಯ ಅವಧಿಯುದ್ದಕ್ಕೂ ಆರೈಕೆಯ ಗುಣಮಟ್ಟವನ್ನು ನಿರ್ವಹಿಸಲಾಗುವುದು ಎಂದು ನನಗೆ ತಿಳಿಸಲಾಗಿದೆ.

ನಾನು, ನನ್ನ ಉತ್ತಮ ಮನಸ್ಸಿನಲ್ಲಿ, ಈ ಅಧ್ಯಯನದ ಭಾಗದಲ್ಲಿ ಸೇರಿಸಲು ಸಂಪೂರ್ಣ ಒಪ್ಪಿಗೆಯನ್ನು ನೀಡುತ್ತೇನೆ.

ತನಿಖಾಧಿಕಾರಿ: ಡಾ.ಮರಮ್ ಸಂಜನಾ

ದೂರವಾಣಿ ಸಂಖ್ಯೆ- 8008515588

ಭಾಗವಹಿಸುವವರ ಸಹಿ/ಹೆಬ್ಬೆರಳಿನ ಗುರುತು

ಹೆಸರು:

ಸಾಕ್ಷಿಯ ಸಹಿ/ಹೆಬ್ಬೆರಳಿನ ಗುರುತು: ದಿನಾಂಕ:

ಹೆಸರು:

ರೋಗಿಗೆ ಸಂಬಂಧ

PATIENT INFORMATION SHEET

STUDY TITLE: "Platelet indices as a predictor of carotid atherosclerosis in acute ischemic stroke and correlation with NIHSS scoring."

STUDY SITE: R.L Jalappa Hospital and Research Centre, Tamaka, Kolar.

This is to inform you that, you require Basic blood investigations (CBC), CT brain/ MRI Brain, Carotid Doppler for making treatment plan for you condition that is acute ischemic stroke. The above mentioned investigations are required for the making the diagnosis of the disease extent of the disease and for planning of the treatment. The patient with history of weakness or ischemic stroke referred to department of General Medicine at R.L Jalappa hospital and research Centre, Tamaka, Kolar to undergo above mentioned investigations and of those patients who meet the inclusion criteria will be taken for the study.

We are conducting this study to predict the onset and severity of this condition.

If you are willing you will be enrolled in this study and we will do above mentioned investigations and other relevant investigations.

This will facilitate identifying use of platelet indices as a predictor of carotid atherosclerosis in acute ischemic stroke and its complications (if any)in an early stage and treating it. It will also benefit other patients with similar condition in future. You are free to opt-out of the study at any time if you are not satisfied or apprehensive to be a part of the study. Your treatment and care will not be compromised if you refuse to be a part of the study. The study will not add any risk or financial burden to you if you are part of the study. In case of any complication during procedures patient will be treated accordingly.
Your identity and clinical details will be confidential. You will not receive any financial benefit for being part of the study. Principal investigator will bear the cost of all investigations. You are free to contact Dr. Maram Sanjana or any other member of the above research team for any doubt or clarification you have.

Dr. MARAM SANJANA
Mobile no: 8008515588

ರೋಗಿಯ ವಿವರ ಪತ್ರ

ಅಧ್ಯಯನದ ಶೀರ್ಷಿಕೆ: "ತೀವ್ರವಾದ ರಕ್ತಕೊರತೆಯ ಪಾರ್ಶ್ವವಾಯು ಮತ್ತು NIHSS ಸ್ಕೋರಿಂಗ್‌ನೊಂದಿಗೆ ಪರಸ್ಪರ ಸಂಬಂಧದಲ್ಲಿ ಶೀರ್ಷಧಮನಿ ಅಪಧಮನಿಕಾಠಿಣ್ಯದ ಮುನ್ಸೂಚಕವಾಗಿ ಪ್ಲೇಟ್‌ಲೆಟ್ ಸೂಚ್ಯಂಕಗಳು."

ಸ್ಟಡಿ ಸೈಟ್: ಆರ್.ಎಲ್ ಜಾಲಪ್ಪ ಆಸ್ಪತ್ರೆ ಮತ್ತು ಸಂಶೋಧನಾ ಕೇಂದ್ರ, ಟಮಕ, ಕೋಲಾರ.

ತೀವ್ರವಾದ ರಕ್ತಕೊರತೆಯ ಪಾರ್ಶ್ವವಾಯುವಿಗೆ ಚಿಕಿತ್ಸೆ ನೀಡಲು ನಿಮಗೆ ಮೂಲಭೂತ ರಕ್ತ ಪರೀಕ್ಷೆಗಳು (CBC), CT ಮೆದುಳು / MRI ಮೆದುಳು, ಶೀರ್ಷಧಮನಿ ಡಾಪ್ಲರ್ ಅಗತ್ಯವಿದೆ ಎಂದು ಇದು ನಿಮಗೆ ತಿಳಿಸುತ್ತದೆ. ರೋಗದ ವ್ಯಾಪ್ತಿಯನ್ನು ಪತ್ತೆಹಚ್ಚಲು ಮತ್ತು ಚಿಕಿತ್ಸೆಯನ್ನು ಯೋಜಿಸಲು ಮೇಲೆ ತಿಳಿಸಿದ ತನಿಖೆಗಳು ಅಗತ್ಯವಿದೆ. ದೌರ್ಬಲ್ಯ ಅಥವಾ ರಕ್ತಕೊರತೆಯ ಪಾರ್ಶ್ವವಾಯು ಇತಿಹಾಸ ಹೊಂದಿರುವ ರೋಗಿಯನ್ನು ಆರ್.ಎಲ್ ಜಾಲಪ್ಪ ಆಸ್ಪತ್ರೆ ಮತ್ತು ಸಂಶೋಧನಾ ಕೇಂದ್ರ, ಟಮಕ, ಕೋಲಾರದಲ್ಲಿ ಜನರಲ್ ಮೆಡಿಸಿನ್ ವಿಭಾಗಕ್ಕೆ ಉಲ್ಲೇಖಿಸಲಾಗಿದೆ ಮತ್ತು ಈ ಮೇಲಿನ ತನಿಖೆಗಳಿಗೆ ಒಳಗಾಗಲು ಮತ್ತು ಸೇರ್ಪಡೆ ಮಾನದಂಡಗಳನ್ನು ಪೂರೈಸುವ ರೋಗಿಗಳನ್ನು ಅಧ್ಯಯನಕ್ಕೆ ತೆಗೆದುಕೊಳ್ಳಲಾಗುತ್ತದೆ.

ಈ ಸ್ಥಿತಿಯ ಆಕ್ರಮಣ ಮತ್ತು ತೀವ್ರತೆಯನ್ನು ಊಹಿಸಲು ನಾವು ಈ ಅಧ್ಯಯನವನ್ನು ನಡೆಸುತ್ತಿದ್ದೇವೆ.

ನೀವು ಸಿದ್ಧರಿದ್ದರೆ ನೀವು ಈ ಅಧ್ಯಯನಕ್ಕೆ ದಾಖಲಾಗುತ್ತೀರಿ ಮತ್ತು ನಾವು ಮೇಲೆ ತಿಳಿಸಿದ ತನಿಖೆಗಳು ಮತ್ತು ಶಸ್ತ್ರಚಿಕಿತ್ಸಾ ವಿಧಾನಗಳಿಗೆ ಅಗತ್ಯವಿರುವ ಇತರ ಸಂಬಂಧಿತ ತನಿಖೆಗಳನ್ನು ಮಾಡುತ್ತೇವೆ.

ತೀವ್ರವಾದ ರಕ್ತಕೊರತೆಯ ಪಾರ್ಶ್ವವಾಯು ಮತ್ತು ಅದರ ತೊಡಕುಗಳು (ಯಾವುದಾದರೂ ಇದ್ದರೆ) ಆರಂಭಿಕ ಹಂತದಲ್ಲಿ ಶೀರ್ಷಧಮನಿ ಅಪಧಮನಿಕಾಠಿಣ್ಯದ ಮುನ್ಸೂಚಕವಾಗಿ ಪ್ಲೇಟ್‌ಲೆಟ್ ಸೂಚ್ಯಂಕಗಳ ಬಳಕೆಯನ್ನು ಗುರುತಿಸಲು ಮತ್ತು ಅದಕ್ಕೆ ಚಿಕಿತ್ಸೆ ನೀಡಲು ಇದು ಸಹಾಯ ಮಾಡುತ್ತದೆ. ಭವಿಷ್ಯದಲ್ಲಿ ಇದೇ ರೀತಿಯ ಸ್ಥಿತಿಯನ್ನು ಹೊಂದಿರುವ ಇತರ ರೋಗಿಗಳಿಗೆ ಇದು ಪ್ರಯೋಜನವನ್ನು ನೀಡುತ್ತದೆ. ಅಧ್ಯಯನದ ಭಾಗವಾಗಲು ನೀವು ತೃಪ್ತರಾಗದಿದ್ದರೆ ಅಥವಾ ಭಯಪಡದಿದ್ದರೆ ಯಾವುದೇ ಸಮಯದಲ್ಲಿ ಅಧ್ಯಯನದಿಂದ ಹೊರಗುಳಿಯಲು ನೀವು ಸ್ವತಂತ್ರರಾಗಿದ್ದೀರಿ. ನೀವು ಅಧ್ಯಯನದ ಭಾಗವಾಗಲು ನಿರಾಕರಿಸಿದರೆ ನಿಮ್ಮ ಚಿಕಿತ್ಸೆ ಮತ್ತು ಕಾಳಜಿಗೆ ಧಕ್ಕೆಯಾಗುವುದಿಲ್ಲ. ನೀವು ಅಧ್ಯಯನದ ಭಾಗವಾಗಿದ್ದರೆ ಅಧ್ಯಯನವು ನಿಮಗೆ ಯಾವುದೇ ಅಪಾಯ ಅಥವಾ ಆರ್ಥಿಕ ಹೊರೆಯನ್ನು ಸೇರಿಸುವುದಿಲ್ಲ. ಕಾರ್ಯವಿಧಾನದ ಸಮಯದಲ್ಲಿ ಯಾವುದೇ ತೊಡಕುಗಳ ಸಂದರ್ಭದಲ್ಲಿ ರೋಗಿಗೆ ಅನುಗುಣವಾಗಿ ಚಿಕಿತ್ಸೆ ನೀಡಲಾಗುತ್ತದೆ.

ನಿಮ್ಮ ಗುರುತು ಮತ್ತು ಕ್ಲಿನಿಕಲ್ ವಿವರಗಳು ಗೌಪ್ಯವಾಗಿರುತ್ತವೆ. ಅಧ್ಯಯನದ ಭಾಗವಾಗಿರುವುದರಿಂದ ನೀವು ಯಾವುದೇ ಹಣಕಾಸಿನ ಪ್ರಯೋಜನವನ್ನು ಪಡೆಯುವುದಿಲ್ಲ. ನೀವು ಹೊಂದಿರುವ ಯಾವುದೇ ಸಂದೇಹ ಅಥವಾ ಸ್ಪಷ್ಟೀಕರಣಕ್ಕಾಗಿ ಡಾ. ಮರಮ್ ಸಂಜನಾ ಅಥವಾ ಮೇಲಿನ ಸಂಶೋಧನಾ ತಂಡದ ಇತರ ಸದಸ್ಯರನ್ನು ಸಂಪರ್ಕಿಸಲು ನೀವು ಮುಕ್ತರಾಗಿದ್ದೀರಿ.

ಡಾ. ಮರಮ್ ಸಂಜನಾ

ಮೊಬೈಲ್ ಸಂಖ್ಯೆ: 800851558

Slno	Age	Sex	Smoking status	Alcohol	Hypertension	Diabetes	MPV	PDW	PCT	CIMT	NIHSS
1	45	M	Nil	Nil	Absent	Absent	9.6	14.5	1.5	0.09	6
2	58	F	Nil	Nil	Absent	Present	8.5	10.3	2.6	0.12	7
3	62	M	Nil	Alcoholic	Present	Absent	10.1	11.4	3.5	0.14	26
4	45	F	Nil	Nil	Absent	Present	9.1	12.9	3.4	0.06	14
5	48	M	Past smoker	Nil	Present	Present	10.3	12.5	2.5	0.06	5
6	56	M	Nil	Nil	Absent	Present	8.9	10.1	3.6	0.09	5
7	52	F	Nil	Nil	Present	Absent	8.5	9.8	3.8	0.13	10
8	64	M	Nil	Nil	Present	Present	9.2	10	2.4	1.17	10
9	61	M	Smoker	Alcoholic	Present	Absent	10.6	13.4	3.5	0.14	22
10	62	F	Nil	Nil	Absent	Present	9.6	12	3.6	0.07	3
11	52	M	Nil	Nil	Present	Present	8.5	11.5	3.4	0.17	16
12	63	F	Nil	Nil	Present	Absent	8.7	13.4	3.5	0.08	11
13	68	M	Nil	Nil	Absent	Present	7.6	14.6	3.4	0.12	18
14	69	M	Nil	Alcoholic	Present	Absent	8.6	11.4	3.1	0.05	8
15	71	M	Nil	Nil	Present	Present	10.2	14.5	2.1	0.08	5
16	65	M	Nil	Nil	Present	Present	10.6	13.4	2.6	0.09	13
17	76	F	Nil	Nil	Absent	Present	9.5	16.8	3.4	0.12	28
18	56	M	Nil	Nil	Present	Absent	8.5	13.5	2.6	0.12	12
19	58	M	Past smoker	Nil	Present	Present	7.2	10.3	2.3	0.07	9
20	69	M	Nil	Nil	Absent	Absent	8.5	11.2	3.5	0.08	10
21	76	F	Nil	Nil	Present	Present	8.6	12.4	2.6	0.14	4
22	74	M	Nil	Nil	Absent	Present	7.5	11	2.4	0.1	12
23	85	F	Nil	Nil	Present	Present	7.4	12.5	3.1	0.08	9
24	65	M	Nil	Alcoholic	Absent	Absent	6.5	9.6	2.4	0.14	10
25	69	M	Smoker	Nil	Present	Absent	9.1	8.5	3.1	0.09	4
26	75	M	Nil	Nil	Present	Present	10.5	10.2	3.2	0.07	12
27	62	F	Nil	Nil	Absent	Absent	8.6	13.2	1.8	0.09	8
28	63	M	Nil	Nil	Present	Present	7.5	11.2	2.4	0.17	8
29	64	F	Nil	Nil	Absent	Present	6.5	12.4	3.4	0.08	26
30	71	M	Smoker	Alcoholic	Present	Present	8.5	13.4	2.6	0.12	5
31	65	M	Nil	Alcoholic	Present	Absent	8.6	11.4	3.1	0.05	8
32	68	M	Nil	Nil	Present	Present	10.2	14.5	2.1	0.08	5
33	62	M	Nil	Alcoholic	Present	Absent	10.1	11.4	3.5	0.14	26
34	55	M	Nil	Nil	Absent	Present	9.1	12.9	3.4	0.06	14
35	58	F	Past smoker	Nil	Present	Present	10.3	12.5	2.5	0.06	5
36	65	M	Smoker	Alcoholic	Present	Absent	10.6	13.4	3.5	0.14	22
37	70	F	Nil	Nil	Absent	Present	9.6	12	3.6	0.07	3
38	62	M	Nil	Nil	Present	Present	8.5	11.5	3.4	0.17	16
39	73	F	Nil	Nil	Present	Present	7.5	11.2	2.4	0.17	8
40	81	M	Nil	Nil	Present	Present	10.2	14.5	2.1	0.08	5

41	71	M	Nil	Nil	Present	Present	10.2	14.5	2.1	0.08	5
42	65	M	Nil	Nil	Present	Present	10.6	13.4	2.6	0.09	13
43	76	F	Nil	Nil	Absent	Present	9.5	16.8	3.4	0.12	28
44	56	M	Nil	Nil	Present	Absent	8.5	13.5	2.6	0.12	12
45	58	M	Past smoker	Nil	Present	Present	7.2	10.3	2.3	0.07	9
46	69	M	Nil	Nil	Absent	Absent	8.5	11.2	3.5	0.08	10
47	76	F	Nil	Nil	Present	Present	8.6	12.4	2.6	0.14	4
48	74	M	Nil	Nil	Absent	Present	7.5	11	2.4	0.1	12
49	85	F	Nil	Nil	Present	Present	7.4	12.5	3.1	0.08	9
50	65	M	Nil	Alcoholic	Absent	Absent	6.5	9.6	2.4	0.14	10
51	63	M	Nil	Nil	Present	Present	7.5	11.2	2.4	0.17	8
52	64	F	Nil	Nil	Absent	Present	6.5	12.4	3.4	0.08	26
53	71	M	Smoker	Alcoholic	Present	Present	8.5	13.4	2.6	0.12	5
54	65	M	Nil	Alcoholic	Present	Absent	8.6	11.4	3.1	0.05	8
55	68	M	Nil	Nil	Present	Present	10.2	14.5	2.1	0.08	5
56	81	M	Nil	Nil	Present	Present	10.2	14.5	2.1	0.08	5
57	71	M	Nil	Nil	Present	Present	10.2	14.5	2.1	0.08	5