"A STUDY ON CLINICAL SIGNIFICANCE OF HEMATOLOGICAL INDICES, ELECTRO CARDIOGRAPHIC CHANGES BY USING NATIONAL INSTITUTE OF HEALTH STROKE SCORE (NIHSS) IN ACUTE ISCHEMIC STROKE IN TERTIARY CARE CENTRE "

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CERTIFICATE

This is to certify that the dissertation titled "A STUDY ON CLINICAL SIGNIFICANCE OF HEMATOLOGICAL INDICES, ELECTRO CARDIOGRAPHIC CHANGES BY USING NATIONAL INSTITUTE OF HEALTH STROKE SCORE (NIHSS) IN ACUTE ISCHEMIC STROKE IN TERTIARY CARE CENTRE " submitted by Dr.S.Sakthivel to the faculty of General Medicine, The TamilNadu Dr. M.G.R Medical University, Chennai, Tamilnadu, in partial fulfilment of the requirement for the award of M.D DEGREE BRANCH - I (GENERAL MEDICINE) is a bonafide research work carried out by him under my direct supervision and guidance.

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I, Dr.S.SAKTHIVEL, solemnly declare that this dissertation entitled "A STUDY ON CLINICAL SIGNIFICANCE OF HEMATOLOGICAL INDICES, ELECTRO CARDIOGRAPHIC CHANGES BY USING NATIONAL INSTITUTE OF HEALTH STROKE SCORE (NIHSS) IN ACUTE ISCHEMIC STROKE IN TERTIARY CARE CENTRE " was done by me at Madras Medical College and Rajiv Gandhi Government General Hospital, Chennai during 2020-2023 under the guidance and supervision of my chief Prof. Dr. Nalini Kumaravelu,M.D. This dissertation is submitted to the Tamil Nadu Dr. M.G.R. Medical University towards the partial fulfilment of requirements for the award of M.D. Degree in General Medicine (Branch-I).

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ABBREVIATIONS

AIS : Acute Ischemic Stroke

TIA : Transient Ischemic Attack

WHO : World Health Organisation

GCS : Glasgow Coma Scale

NLR : Neutrophil-Lymphocyte Ratio

PLR : Platelet-lymphocyte Ratio

ECG : Electro Cardio Gram

SBP : Systolic Blood Pressure

DBP : Diastolic Blood Pressure

SAH : Subarachnoid Hemorrhage

ICH : Intra Cerebral Hemorrhage

MI : Myocardial Infarction

MCA : Middle Cerebral Artery

ACA : Anterior Cerebral Artery

PCA : Posterior Cerebral Artery

ICA : Internal Carotid Artery

SCA : Superior Cerebellar Artery

AICA : Anterior inferior Cerebellar Artery

PICA : Posterior inferior Cerebellar Artery

PcoA : Posterior Communicating Artery

ASA : Anterior Spinal Artery

LVO : Large Vessels Occlusion

NCCT : Non Constrast Computerised Tomography

CTA : Computed Tomography Angiography

MRI : Magnetic Resonance Imaging

MRA : Magnetic Resonance Arteriography

MRV : Magnetic Resonance Venography

ICH : Intracerebral Hemorrhage

FLAIR : Fluid Attenuated Inversion Recovery

DWI : Diffusion-weighted imaging

PWI : Perfusion-weighted imaging

SWI : Susceptibility Weighted Imaging

rtPA : Recombinant tissue plasminogen activator

EVT : Endovascular Thrombectomy

NIHSS : National Institutes of Health Stroke Scale

TOAST : Trail Of Acute Stroke Treatment

TH1/TH17 : T- helper cells 1/ T- helper cells 17

AHA/ASA : American Heart Association/ American Stroke Association

ASPECTS : Alberta Stroke Program Early Computed Tomography Score.

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ABSTRACT

Introduction

Acute ischemic stroke results from sudden loss of blood circulation to an area of brain leading to irreversible injury and neurological deficits persisting for more than 24 hours or until death. It accounts for 80-87% of all strokes. Platelets activation and aggregation are critical in the pathogenesis of acute ischemic stroke. Acute Ischemic stroke is an inflammatory event where the ischemic tissue releases chemokines and cytokines, and recruit peripheral circulating leucocytes. Lymphocytes also infiltrate the ischemic tissues and mediate inflammatory responses. Among the leucocytes, neutrophils were found to be an important mediator and early neutrophilia was found to be associated with larger stroke volumes and poor prognosis. In the recent years, the neutrophil to lymphocyte ratio (NLR) and platelet to lymphocyte ratio (PLR) have been emerged as the well accepted biomarkers for the assessment of overall inflammatory status. These are simple and cost effective biomarkers. Elevated levels of NLR and PLR have found to be related to oxidative stress and increased cytokine production in patients presenting with Acute ischemic stroke. The NLR has been used as an indicator to reflect the prevalence of intracranial atherosclerosis and is considered to be an independent risk factor for ischemic stroke and a poor prognosis. The PLR has been used to predict poor prognosis,the rate of insufficient recanalization and the size of infarcted area following stroke. Ecg changes in acute ischemic stroke,are new onset

QT prolongation and T wave inversion in precordial leads associated with poor Outcome. NIHSS grade severe is associated with significant poor outcome. If these parameters are taken together, predicting outcome will become easy and it will be helpful in taking appropriate interventions in initial hours by focusing on the correctable parameters in order to prevent mortality in acute stroke.

Aims and Objectives

To assess the correlation of Hematological indices, Electrocardiographics changes among the patients presenting with acute ischemic stroke by the application of NIHSS score.

Material and methods

The prospective observational study is proposed to be conducted after obtaining informed consent from the patients admitted in Medical emergency department and medical wards,IIM,MMC & RGGGH. In our study, 80 patients are selected as per inclusion and exclusion criteria, detailed history will be elicited.Clinical examination done after stabilization of Airway, Breathing, Circulation. Grading the Patients mild,moderate, moderate to severe & Severe stroke by using NIHSS scale. After initial clinical evaluation,Grading of the patient, Hematological indices including Neutophil-Lymphocyte ratio and Platelet-Lymphocyte ratio and Renal function test, Lipid profile ECG, Chest X ray and CT Brain done for all patients. Comparing the above parameters of the patients based upon the NIHSS Grading and find out the disease severity and outcomes.

Results

- ➢ There was statistically significant correlation was found between NLR and NIHSS score at the time of admission and value of NLR increased proportionately with the increasing NIHSS score.(P=<0.001)
- ➢ There was statistically significant correlation found between PLR and NIHSS score at the time of admission. The value of PLR increased proportionately with the increasing NIHSS score. (p=<0.001).
- ➢ In our study participants, who had severe NIHSS during admission, ST depression (57%), QT prolongation (57%), T wave inversion (42%).

conclusion

High levels of NLR and PLR were associated with the highest risk of unfavourable functional outcomes. Severe grade of National institute of health stroke scale is associated with statistically significant poor outcome. ST-T changes and QT prolongation in ECG had higher mortality and poor outcome.

These parameters are taken together, predicting outcome will become easy and it will be helpful in taking appropriate interventions in initial hours by focusing on the correctable parameters in order to prevent mortality in acute stroke.

INTRODUCTION

Acute ischemic stroke is not only a severe disabling cerebrovascular event but also has a great impact on patient's life and socioeconomic burden. Although it can be prevented by controlling relevant risk factors,its prevalence and incidence remain increasing with ageing and atherosclerotic process. It is the commenst cause of mortality after coronary artery disease and the commonest cause of chronic adult disability.

Stroke is the second leading cause of death and disability worldwide, and its prevalence is rising in emerging nations[1]. The majority of strokes are caused by ischaemic stroke caused by arterial occlusion. Management focuses on rapid reperfusion using intravenous thrombolysis and endovascular thrombectomy, both of which reduce disability but are time-consuming. Therefore, enhancing the healthcare system to shorten waiting times for treatments is essential to optimising the advantages of reperfusion therapy. Intravenous thrombolysis reduces disability when administered within 4.5 hours of the onset of a stroke. Thrombolysis is also beneficial in patients who have evidence of salvageable brain tissue from perfusion imaging for up to 9 hours and in patients who awake with stroke symptoms. Endovascular thrombectomy improves disability in a wide range of patients with large vessel occlusion when performed within 6 hours of stroke onset and in patients selected by perfusion imaging up to 24 hours later.

Many elements of secondary prevention of ischaemic stroke are shared with cardiovascular risk management in other fields, such as blood pressure control, cholesterol management, and antithrombotic medications. Other preventive interventions, such as anticoagulation for atrial fibrillation and carotid endarterectomy for severe symptomatic carotid artery stenosis, are tailored to the mechanism of stroke. Predicting the outcome of a stroke is still debatable. Many experimental and clinical studies and research have demonstrated the importance of the first few hours in an acute stroke[2].

The most critical time for intervention to be effective is in the early hours. Most studies have concentrated solely on conventional risk factors, such as GCS, blood pressure, and other clinical profiles at presentation, as well as radiological parameters. However, we considered the NLR ratio, PLR ratio, and ECG alterations in our investigation. There have been very few studies that have investigated the prognostic significance of these parameters using multivariate analysis. The importance of haematological markers in relation to NIHSS grading and the ability to forecast in-hospital mortality have both been examined in this study.

AIMS & OBJECTIVES

To study the clinical significance of Neutrophil-Lymphocyte ratio, Platelet-lymphocyte ratio and electrocardiographic changes in patient presenting with acute ischemic stroke by the application of National Institutes of health stroke scale.

REVIEW OF LITERATURE

Stroke is defined as a sudden neurological deficit of vascular aetiology (loss of blood circulation to a brain area) that lasts more than 24 hours. Acute ischemic stroke is increasingly recognised as a treatable neurological emergency.

Acute ischemic stroke caused by thrombotic or embolic occlusion of a cerebral artery deprives the brain of oxygen and nutrients beyond the combined severity and time thresholds, resulting in ischemic infarction. The clinical deficit in stroke patients is caused by a hypoperfused, hibernating, electrically non-functional part of the brain known as the ischaemic penumbra. This region gradually converts to irreversibly injured tissue (known as the ischaemic core) over time. This penumbral brain, however, can be saved and restored to normal function with rapid reperfusion[3].

EPIDEMIOLOGY

In 2019, the global prevalence of stroke was 101.5 million people, of which, 77.2 million people suffered from acute ischemic stroke. In the India, 87% of all strokes are ischemic, while intracranial hemorrhage (and subarachnoid hemorrhage represent 10% and 3%of all strokes, respectively. The lifetime risk of overt stroke is approximately one in four by the age of 80, while the risk of silent strokes approaches 100%. Before modern intervention, early mortality after stroke was almost 10%, while one-half of the patients developed moderate-to-severe disability and one-quarter were dependent on others.

Furthermore, 10% of all epilepsy, and 55% of newly diagnosed seizures in the elderly, can be attributed to stroke. Despite the increasing efficacy of secondary stroke prevention, the annual stroke recurrence risk is 2.5–4.0%.[4]

STROKE CLASSIFICATION

The Trial of Acute Stroke Treatment (TOAST) classification system[5] was developed in the early 1990s and is widely used with good interobserver agreement.
It classifies ischemic strokes into five subtypes based on the underlying pathophysiological mechanism, including

(1) large artery atherosclerosis

(2) cardioembolism

(3) small vessel occlusion

(4) stroke of other determined etiology

(5) stroke of undetermined etiology.

PATHOPHYSIOLOGICAL MECHANISMS

Acute ischemic stroke caused by a number of pathophysiological mechanisms. Thromboembolism, cardioembolism, small vessel disease, and cryptogenic thromboembolism are all common mechanisms. Other less common mechanisms include arterial dissection, vasospasm (for example, as a complication of SAH),

vasculitis, vasculopathy (for example, moyamoya disease), and haematological disorders such as hypercoagulable states and sickle cell disease[6].

Microatheroma with plaque rupture followed by microembolism, or lipohyalinosis and fibrinoid necrosis of the penetrating arteries, have been proposed as mechanisms.

Lacunar infarctions caused by small vessel disease account for up to 20% of all ischemic strokes and can result in distinctive lacunar syndromes, though the underlying mechanism for small vessel disease is unknown.

Cryptogenic strokes account for nearly one-quarter of all AIS patients, while "stroke of undetermined source (ESUS)" refers to patients who have imaging findings suggestive of embolism but no identifiable aetiology[7].

In atherosclerotic disease, acute Ischemic stroke may result from the rupture of an extracranial plaque, which causes thrombus formation, followed by thrombus distal embolization to intracranial vessels (e.g., artery-to-artery embolization).

Atherosclerotic plaque rupture within intracranial vessels can lead to in situ vessel occlusion.

Ischemia can be subdivided into thrombosis, embolism, decreased systemic perfusion.

THROMBOSIS

Thrombosis is any obstruction to blood flow caused by a localised occlusive disease in the blood vessels. Atherosclerosis is the most common vascular pathology, in which fibrous and muscular tissues proliferate within the subintima and fat forms plaques that can encroach on the lumen. The platelets will then adhere to the plaque, forming clumps that will act as a nidus for the deposition of fibrin, clot, and thrombus. The larger intracranial and extracranial arteries will be the most affected by atherosclerosis[8]..

A clot may form within the lumen of a vessel as a result of a haematological problem such as polycythemia, thrombocytosis, or a hypercoagulable state.

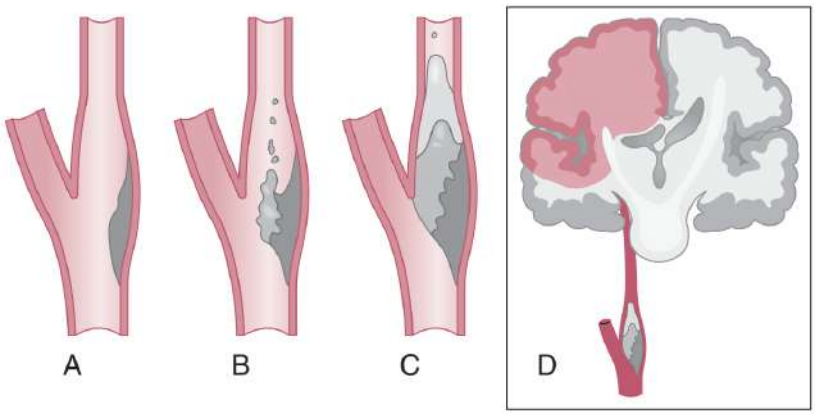


Figure 1: CAROTID ARTERY ATHEROSCLEROSIS

(A) plaque

(B) plaque with platelet fibrin emboli

(C) plaque with occlusive thrombus

(D) ischemic cerebral infarct due to embolization from ICA thrombus

EMBOLISM

Arterial occlusion can occur as a result of embolism from a cardiac source, such as atrial fibrillation, or as a result of in situ thrombus formation in the heart, which leads to thromboembolism[9].

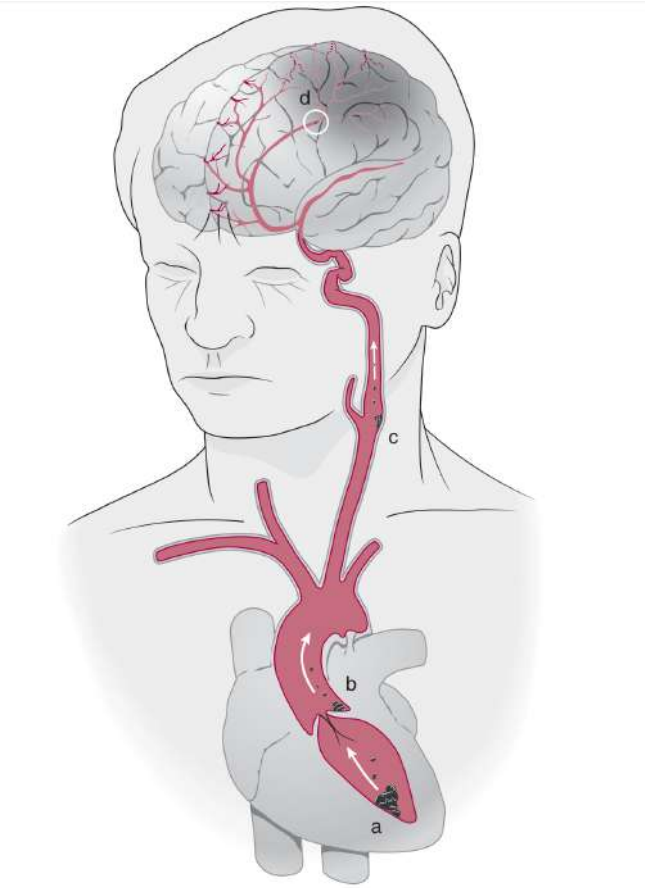


Figure 2: SOURCE OF EMBOLISM

a)Cardiac mural thrombus

b)Vegetation on heart valve

c)Emboli from carotid plaque

DECREASED SYSTEMIC PERFUSION

In decreased systemic perfusion, reduced blood flow to brain tissue is due to low systemic perfusion pressure. The common causes are cardiac failure and systemic hypotension (which is due to blood loss or hypovolemia). Poor perfusion is most prominent in border zone also known as watershed regions at the junction of the major vascular territories.

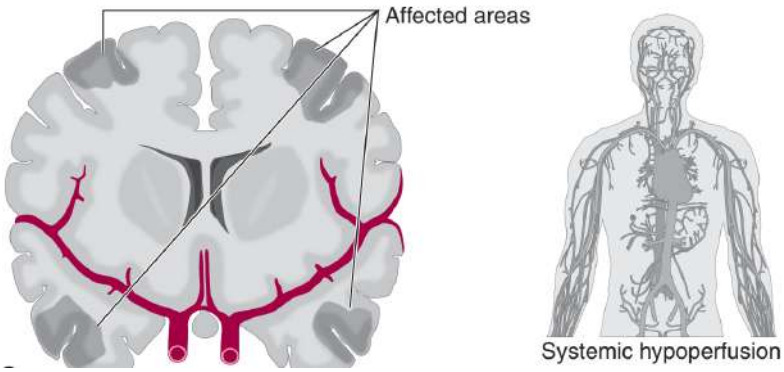


Figure 3: SYSTEMIC HYPOPERFUSION

CELLULAR EFFECTS OF ISCHAEMIA

Occlusion of a cerebral artery initiates a cascade of responses. Reduced cerebral blood flow (CBF) leads to reduced availability of glucose and oxygen and a mismatch in energy requirements and availability in neurons, glia and endothelial cells. Anoxic depolarization and reduced activity of glutamate reuptake leads to increased extracellular levels of glutamate. This leads to neuronal calcium influx (through the N-methyl-d-aspartate (NMDA) ion receptor (NMDAR)) and release of calcium from intracellular stores in neurons and glia (mediated via metabotropic glutamate receptors (mGluRs))[10-11].

Blood–brain barrier dysfunction and release of signalling molecules (for example, cytokines) from astrocytes, microglia and oligodendrocytes lead to an inflammatory response[12-13].

In neurons, the cumulative effect is cell death mediated through diverse pathways including necrosis and apoptosis.

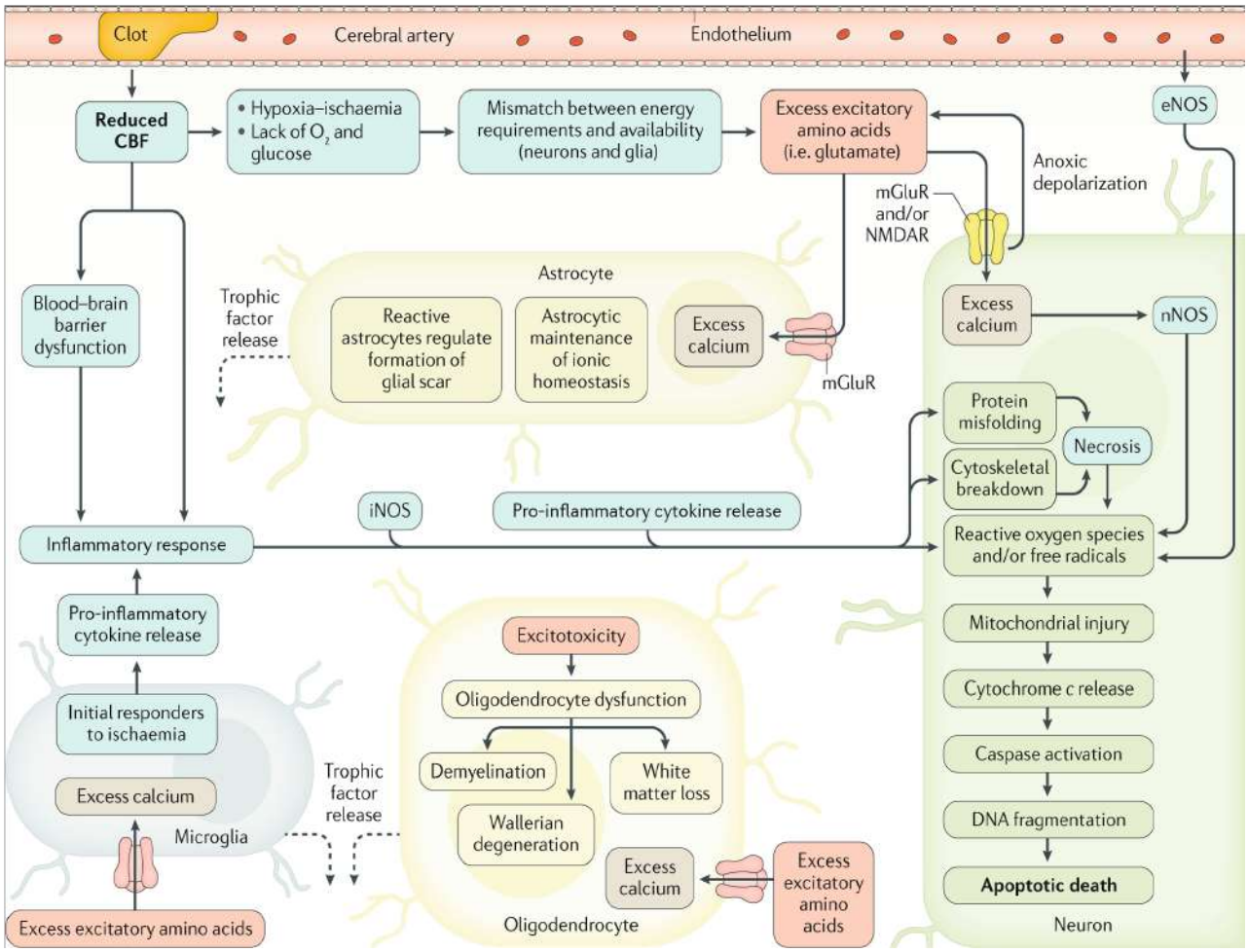


Figure 4: CELLULAR EFFECTS OF ISCHAEMIA

Ischemic stroke is complicated by post ischemic inflammation.

When cerebral ischemia occurred, proteins such as brain-derived antigens, danger-associated molecular patterns (DAMPs), cytokines, and chemokines were released into the systemic circulation from the injured brain regions. Many of these factors induced a cascade of systemic pro-inflammatory molecules and cellular events that aggravated brain damage in both acute and late clinical outcomes. The increasing level of white blood cell count during the early stages of AIS has been linked to a larger

stroke volume, more severe stroke deficits, and poor clinical outcomes. In the white blood cell family, neutrophils were the first to infiltrate the ischemic brain (permeation time ranged from 30 minutes to several hours in focal cerebral ischemia) and reached a peak early (1–3 days post stroke). Neutrophils deteriorated the injured brain by releasing proteolytic enzymes, which had a direct neurotoxic effect. Lymphocytes were proposed to be a pivotal subtype that determined the severity of neuroinflammation in acute brain injuries, with complicated and diverse influences. Several studies have confirmed the presence of other pro-inflammatory lymphocytes such as TH1, TH17, and T-cells. Oppositely, the potential of regulatory T-cells (Treg) and B-cells (Breg) neuroprotective properties.

In ischemic stroke, circulating platelets performed two major functions: First, it causes circulating arterial thrombosis and embolism; second, it acts as a prime motor of the activators stored in platelet granules (e.g., chemokines and cytokines) that mediated other peripheral blood cells.

ANATOMICAL LOCALISATION OF STROKE SYNDROMES

The common carotid arteries on each side ascend and bifurcate around the angle of the mandible, into external and internal carotid arteries .The external carotid artery supplies the structures in the anterior neck and majority of the head and face.

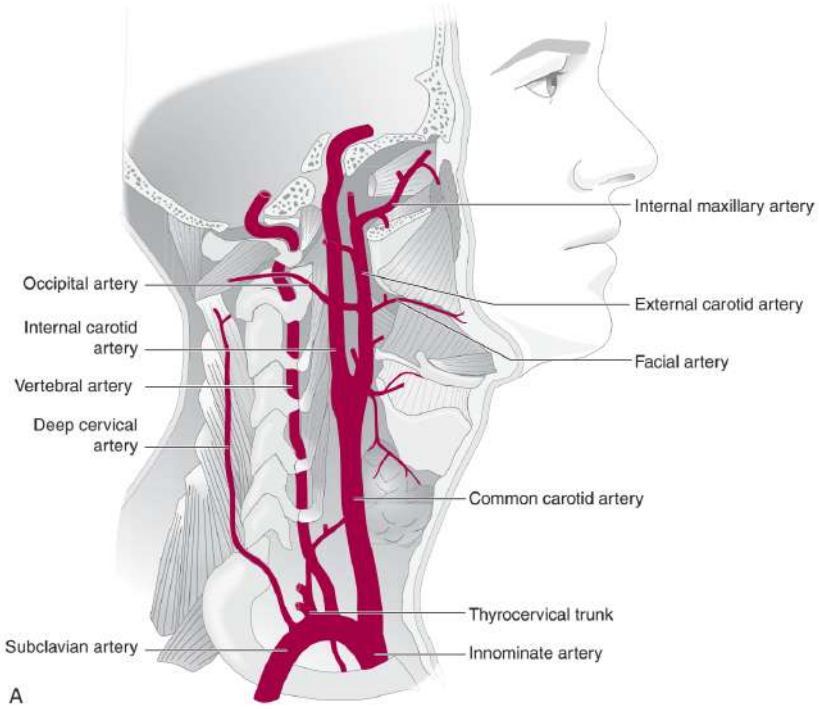


Figure 5: A) MAJOR NECK ARTERIES

The internal carotid ascend and enter into the skull, and by its intracranial branches, supply most of anterior 2/3rd of the brain which includes the entire parietal lobes and frontal lobes and most part of the temporal lobes. The internal carotid arteries and their branches supply the anterior circulation.

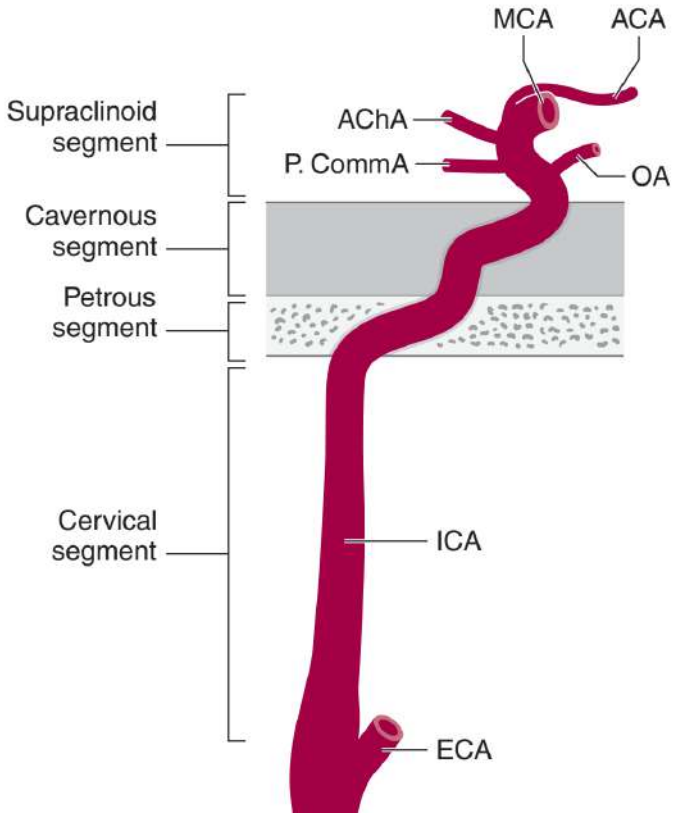


Figure 6: SEGMENTS OF INTERNAL CAROTID ARTERY

The main stem of the MCAs course laterally, giving off lenticulostriate artery branches to the basal ganglia and internal capsule. Although most often the lenticulostriate penetrating branches arise from the mainstem MCA, when the mainstem is short, the lenticulostriate branches may arise from the superior division branch. As they near the sylvian fissures, the MCAs trifurcate into small anterior temporal branches and large superior and inferior divisions.

The superior division supplies the lateral portions of the cerebral hemispheres above the sylvian fi ssures, and the inferior division supplies the temporal and inferior parietal lobes below the sylvian fissures[14].

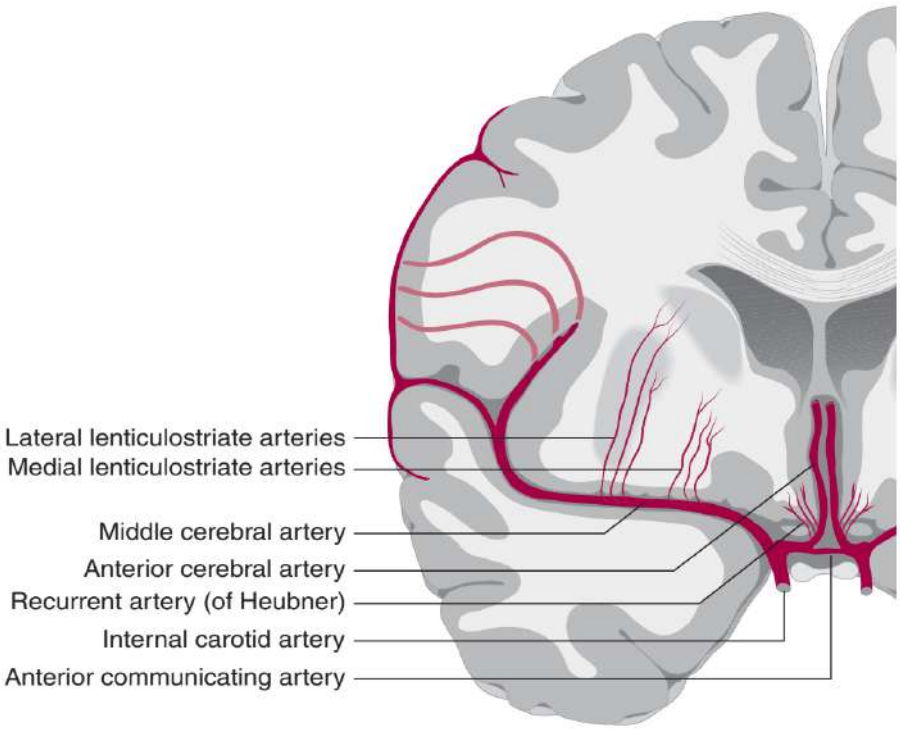


Figure 7: LENTICULOSTRIATE BRANCHES OF MCA

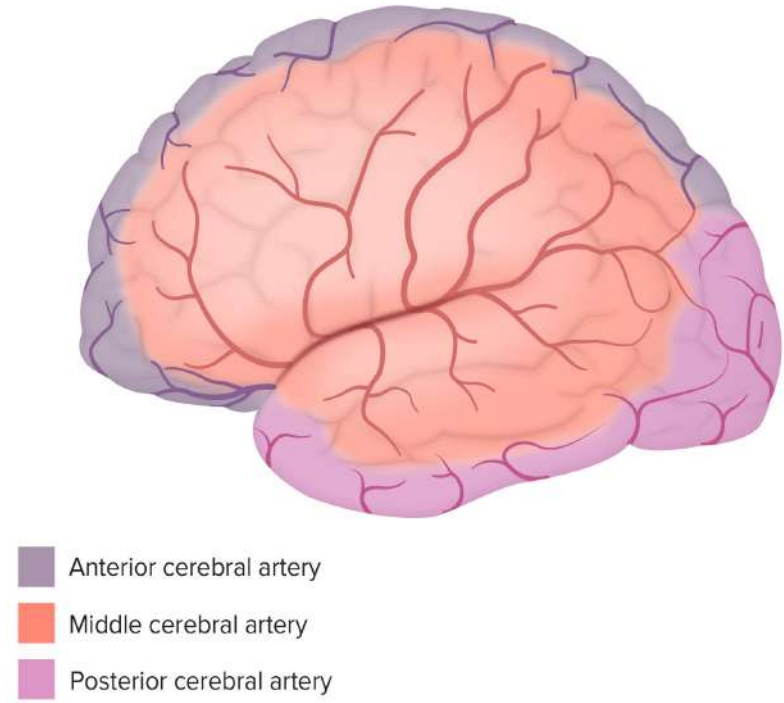


Figure 8: MCA SUPPLYING LATERAL SURFACE OF CEREBRAL HEMISPHERE

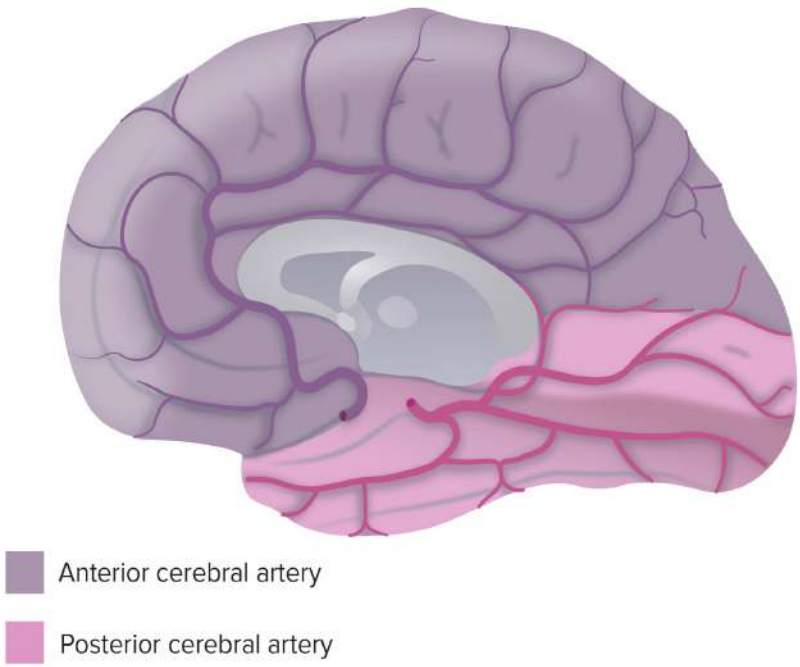


Figure 9: ACA & PCA SUPPLYING MEDIAL SURFACE OF CEREBRAL HEMISPHERE

The posterior circulation (vertebrobasilarsystem) is made up of two arteries, the left and right vertebral arteries, which emerge from the aorta via the subclavian artery.They enter the skull through the vertebral foramen and ascend the anterior surface of the medulla as they progress up towards the foramen magnum. At the level of the pons, the vertebral arteries merge to form the basilar artery. The basilar artery ascends and divides into the right and left posterior cerebral arteries at the midbrain level.

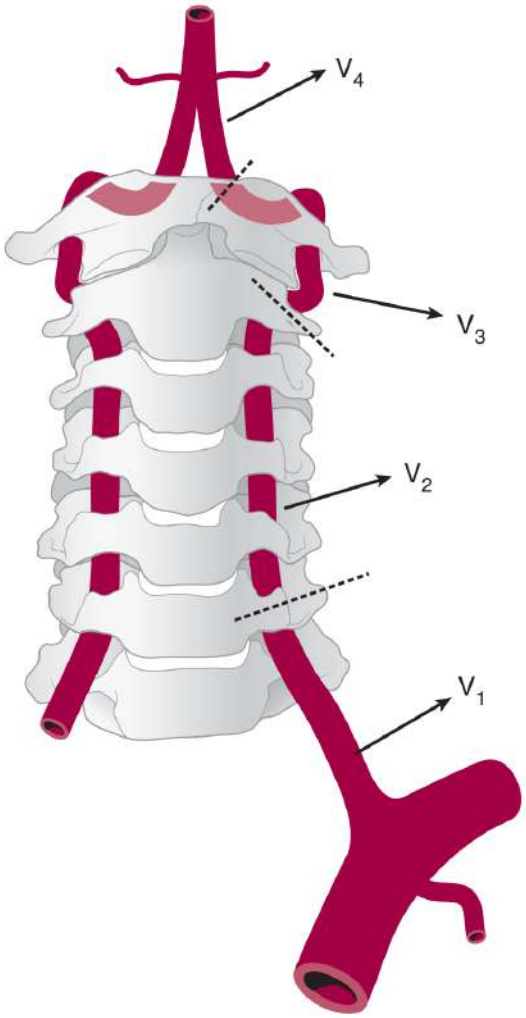


Figure 10: FOUR PARTS OF VERTEBRAL ARTERY

The brainstem, occipital lobe, thalamus, cerebellum, and a portion of the temporal lobe are all supplied by the vertebrobasilar system.

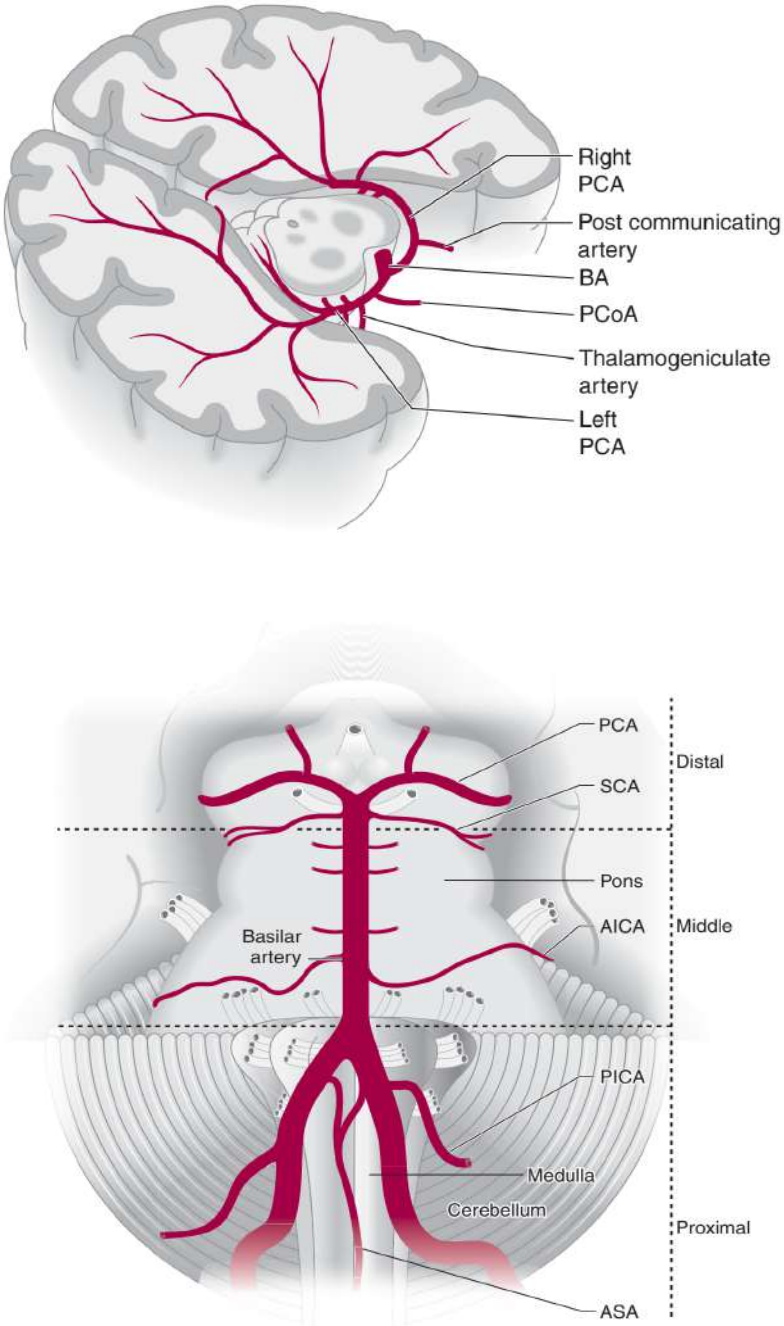


Figure 11: VERTEBROBASILAR SYSTEM

The Circle of Willis, located near the base of the skull, is made up of arteries that connect the left and right sides of the brain as well as the anterior and posterior systems. The anterior and posterior communicating arteries connect the posterior cerebral arteries with both the anterior and middle cerebral arteries of the anterior circulation.

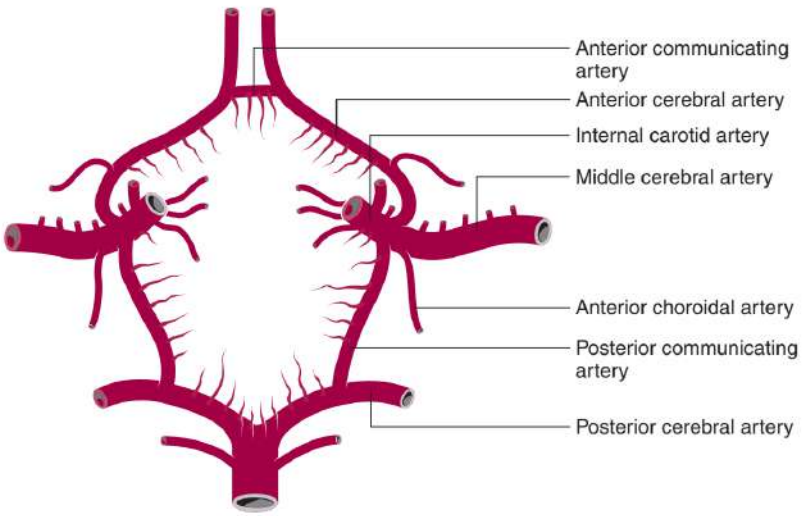


Figure 12: CIRCLE OF WILLIS

Ischemic stroke can be classified to acute infarction due to large vessels occlusion or blockage of "penetrating branches" of the vessels (lacunar stroke).

LARGE VESSEL OCCLUSION

LVO stroke ("cortical stroke") is invariably defined as severe stroke associated with blockages of the proximal intracranial anterior or posterior circulation.

It is the most severe form of acute ischemic stroke accounting for ~10% of acute ischemic strokes and is associated with poor outcome if not treated.

Inclusive definition of LVO [15]

1. Anterior circulation: Intracranial ICA, M1, A1
2. Posterior circulation: Basilar artery, P1

An NIHSS ≥6 has traditionally been used to predict a cortical stroke.

LARGE VESSEL OCCLUSION SYNDROMES

Internal Carotid Artery occlusion

Embolic blockage of the ICA, either distally or proximally, causes severe stroke with symptoms of all anterior circulation arteries. Occlusion caused by increasing atherosclerosis is usually milder, with a classic subacute appearance, and it can even be asymptomatic. Retinal ischemia caused by carotid emboli can be temporary (amaurosis fugax) or permanent (occlusion of the major or branch retinal arteries).

Anterior cerebral artery syndrome

Either side

- ➢ Contralateral leg weakness and sensory loss.
- ➢ Frontal lobe dysfunction (poor judgement, flat affect, apraxia, abulia = apathy and reduced speech, incontinence).
- ➢ Alien hand sign: one hand acts involuntarily.

Non-dominant hemisphere: Acute confusion and contralateral hemineglect.

Dominant hemisphere: Transcortical motor aphasia (due to involvement of the supplementary motor area). This is similar to Broca's aphasia, but with preservation of repetition.

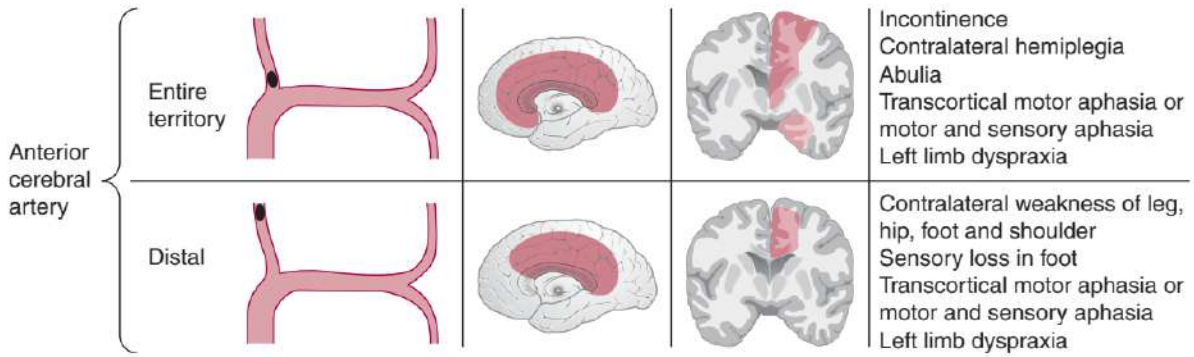


Figure 13: PATTERN OF ACA OCCLUSION AND THEIR ANATOMY CORRELATES

Proximal MCA syndromes (occlusion near the base of M1):

Either side:

- ➢ Contralateral hemiplegia involving the face and arm > leg.
- ➢ Contralateral hemisensory loss.
- ➢ Contralateral hemianopia.
- ➢ Ipsilateral conjugate eye deviation.

Nondominant hemisphere: Hemispatial neglect (generally of the left side) and anosognosia.

Dominant hemisphere: Global aphasia.

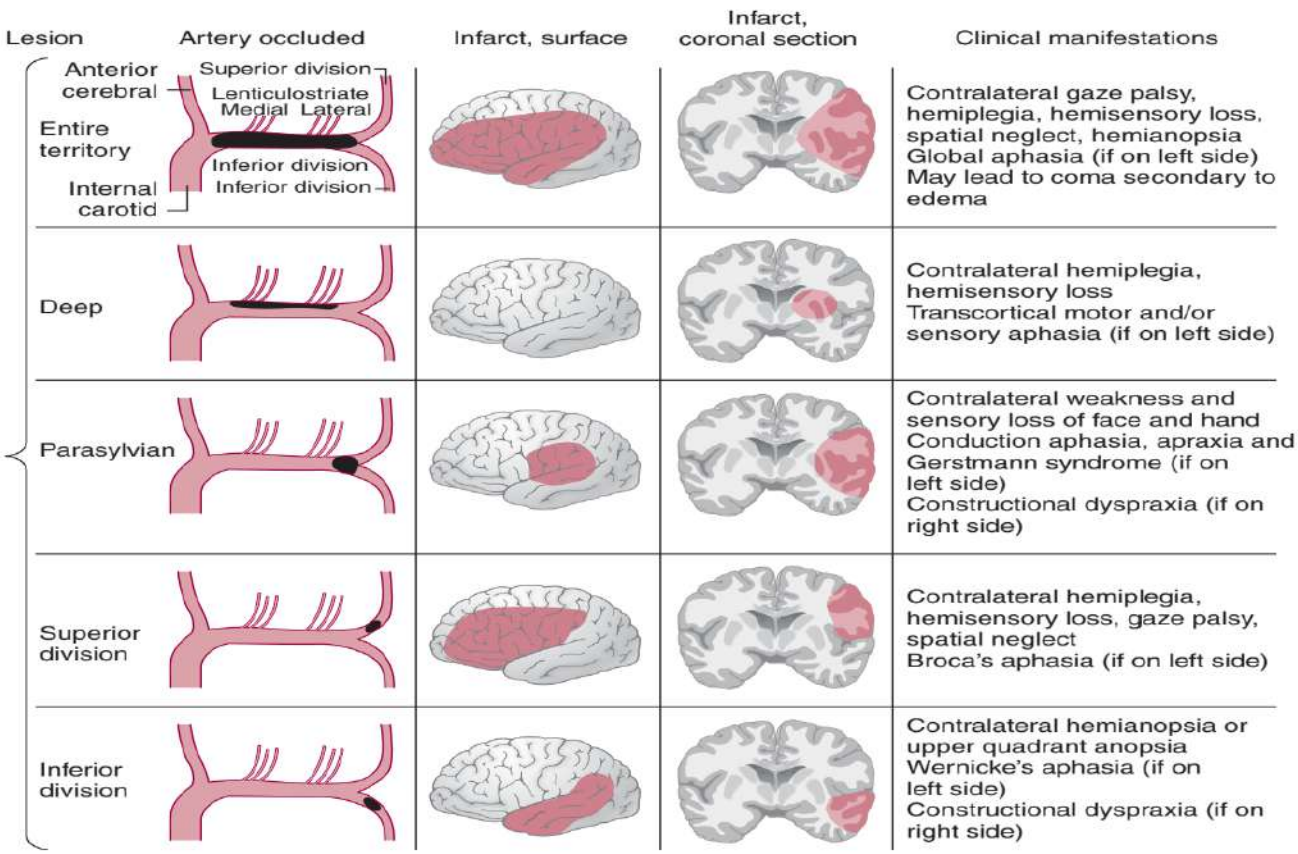


Figure 14: PATTERN OF MCA OCCLUSION AND THEIR ANATOMY CORRELATES

Posterior cerebral artery syndromes:

Either side:

- ➢ Hemianopia or superior quadrantanopia.
- ➢ Thalamic involvement may cause contralateral sensory loss (ventroposterolateral nuclei) or reduced arousal.
- ➢ Uncommonly, may cause contralateral hemiplegia (due to involvement of the internal capsule).

Dominant hemisphere:

- ➢ Alexia without agraphia (patients are able to write but not read). This results from infarction of the splenium of the corpus callosum, thereby cutting off visual information from the language processing centers.
- ➢ Difficulty naming objects (transcortical sensory aphasia).
- ➢ Visual agnosia (inability to describe what an object is used for).
- ➢ Altered memory (anterograde amnesia), if the medial temporal lobes are involved.

Nondominant hemisphere:

- ➢ Prosopagnosia (inability to recognize faces).

Bilateral infarctions: Cortical blindness (Anton syndrome).

Proximal basilar artery syndrome (base of the basilar artery):

- ➢ Altered level of consciousness (ranging from somnolence to coma).
- ➢ Quadriplegia. May also have "crossed" paralysis (e.g. left face and right limbs).
- ➢ Oculomotor abnormalities, which may include horizontal gaze palsy (bilateral or unilateral), internuclear ophthalmoplegia (unilateral or bilateral), one and a half syndrome, skew deviation, gaze paretic nystagmus, or bilateral ptosis.
- ➢ Pinpoint pupils.

- ➢ Bulbar symptoms, which may include: Facial weakness, dysphagia, dysarthria, palatal myoclonus.
- ➢ Pseudobulbar affect.
- ➢ Sensory loss to light touch.

Mid basilar artery (Locked-in syndrome):

- ➢ Locked-in state
- ➢ Quadriplegia and facial paralysis
- ➢ Horizontal gaze palsy (vertical gaze remain intact).
- ➢ Dysphagia.
- ➢ Vertigo.
- ➢ Hearing loss can occur.

Top of the basilar syndrome:

This results in ischemia of the midbrain, thalamus, and occipital lobes (but not the pons). The cerebellum may also be involved via the superior cerebellar artery.

- ➢ Altered level of consciousness (ranging from somnolence to coma).
- ➢ Pupillary abnormalities (may involve afferents to Edinger-Westphal nucleus, CN3 nucleus, or descending sympathetic system). Pupils may be dilated, mid-position, or small.
- ➢ Vertical gaze impairment, internuclear ophthalmoplegia, or skew deviation.

- ➢ Hemianopsia or complete cortical blindness.
- ➢ Amnesia, agitation, and hallucinations (may involve colors and objects).
- ➢ Ataxia, tremor, dysarthria (if the cerebellum is involved).
- ➢ Homonymous hemianopsia.

LACUNAR STROKE

Lacunar infarcts are small (2 to 15 mm in diameter) noncortical infarcts caused by occlusion of a single penetrating branch of a large cerebral artery.

1)Pure motor hemiparesis, Dysarthria-clumsy hand syndrome, Ataxic hemiparesis

Symptoms:

- ➢ Pure motor: Isolated contralateral face/arm/leg weakness. No sensory signs. Dysarthria and dysphagia may be present.
- ➢ Dysarthria-clumsy hand: Dysarthria, facial weakness, slight weakness/clumsiness of the contralateral hand.
- ➢ Ataxic hemiparesis: Ipsilateral hemi-body weakness and limb ataxia (that is disproportionate to the weakness).

Localization:

- ➢ Corona radiata (small MCA branches).
- ➢ Posterior limb of internal capsule (lenticulostriate arteries, anterior choroidal artery, or perforators from the posterior cerebral artery).

- ➢ Cerebral peduncle (small proximal posterior cerebral artery branches).
- ➢ Anterior pons (basilar perforators).

2)Pure sensory stroke (thalamic lacunar stroke)

Symptoms: Unilateral sensory loss of all modalities in face, arm and leg without motor deficit.

Localization: Infarction of the ventral posterior lateral (VPL) and ventral medial nuclei (VPM), supplied by thalamo-perforators from the posterior cerebral artery.

3)Sensorimotor stroke (thalamocapsular lacunar stroke)

Symptoms: Combination of thalamic lacune plus pure motor hemiparesis.

Localization: Posterior limb of the internal capsule plus either thalamic VPL/VPM or thalamic somatosensory radiation. May result from infarction of the thalamoperforator branches of the posterior cerebral artery, or lenticulostriate arteries.

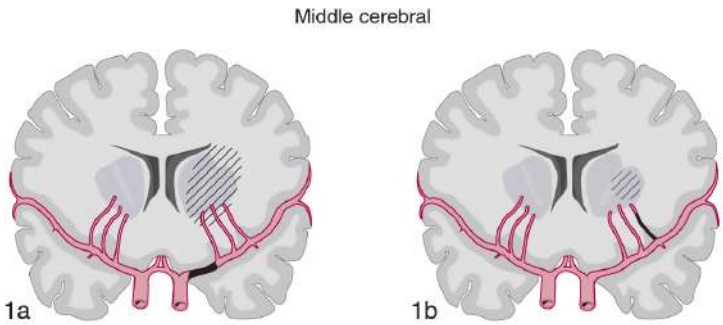


Figure 15: MCA Lacunar infarct

1a) Basal ganglionic capsular infarct due to MCA occlusion.

1b) Smaller deep capsular infarct due to lenticulostriate artery occlusion.

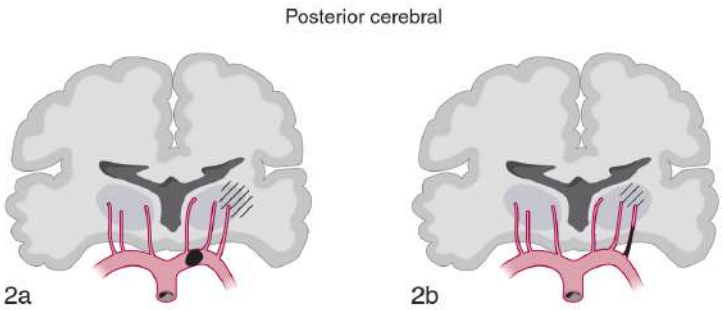


Figure 16: PCA Lacunar infarct

2a) Lateral thalamic infarct due to PCA occlusion.

2b) Lateral thalamic infarct due to thalamogeniculate artery occlusion.

RISK FACTORS

NON MODIFIABLE RISK FACTORS :

- ➢ Age
- ➢ Gender (male > female, except at extremes of age)

- ➢ Race (Afro- Caribbean > Asian > European)
- ➢ Previous vascular event: MI, Stroke, Peripheral vascular disease
- ➢ Heredity
- ➢ Sickle cell disease
- ➢ High Fibrinogen

MODIFIABLE RISK FACTORS[16] :

- ➢ Hypertension
- ➢ Cigarette smoking
- ➢ Hyperlipidemia
- ➢ Diabetes Mellitus
- ➢ Heart disease- Atrial fibrillation, Congestive cardiac failure, Infective endocarditis
- ➢ Excess alcohol usage
- ➢ Oestrogen containing drugs
- ➢ Polycythemia

The most well-documented and curable risk factor for stroke is high blood pressure. Stroke risk is significantly reduced by lowering BP, with a reduction of 31% for every 10mmHg in systolic BP. Guidelines advise lowering blood pressure to 140/85 mmHg or less.

Randomized controlled trials have not provided enough proof that lowering stroke risk by enhancing glucose control.

Stroke risk was weakly positively correlated with ischemic stroke serum cholesterol level. The incidence of ischemic stroke was found to be negatively correlated with high-density lipoprotein (HDL).

Whether persistent, paroxysmal, or permanent, atrial fibrillation (AF) is a significant independent risk factor for ischemic stroke (risk nearly 5-fold).

DIAGNOSIS

The current AHA/ASA stroke guidelines recommendation to enact “an organized protocol for the emergency evaluation of patients with suspected

stroke". A door-to-needle time of ≤60 minutes is the benchmark for achieving rapid treatment with intravenous alteplase[17].

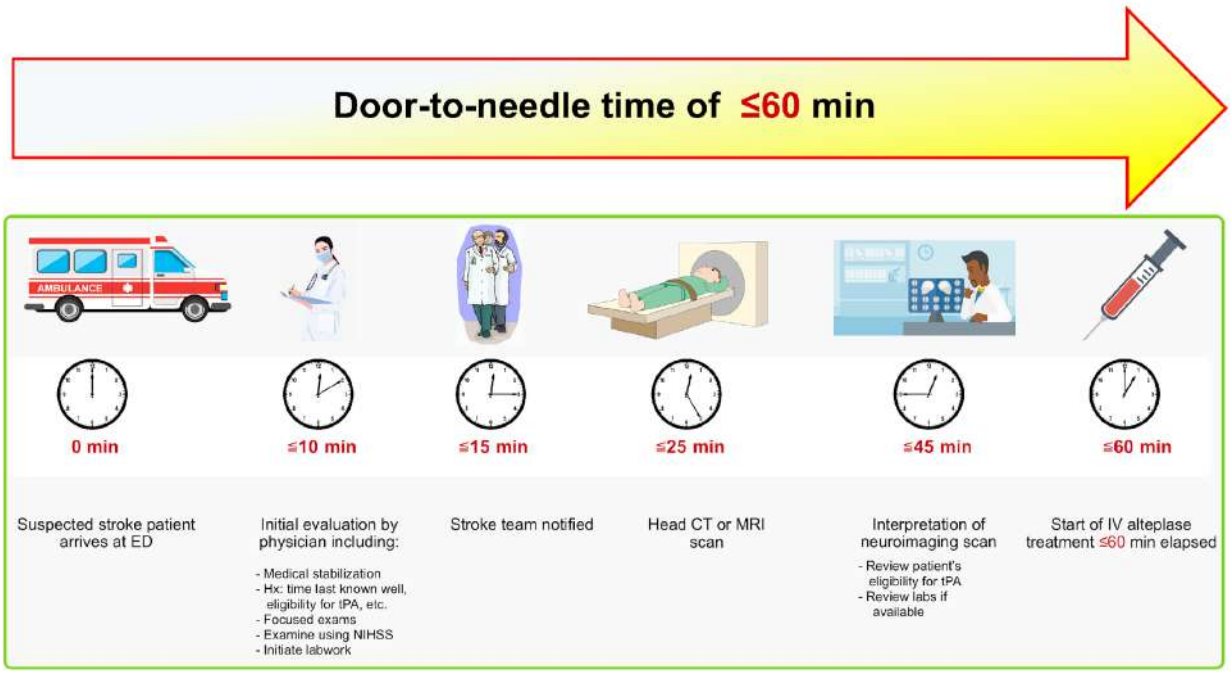


Figure 17: IN-HOSPITAL TIMELINE

In patients with large vessel AIS, 1.9 million neurons, 14 billion synapses, and 7.5 miles of myelinated fibres are destroyed for every minute of infarct evolution. For acute stroke recognition, various detection tools derived from the National Institutes of Health Stroke Scale (NIHSS) are available for use by emergency medical services in the prehospital setting[18-19].

The 42-point NIHSS is a systematic assessment tool that must be used within 10 minutes of the patient's arrival at the emergency room. It aids the healthcare provider in quickly determining the degree of neurological deficit brought on by a stroke. The

NIHSS sheds light on the stroke's underlying aetiology (e.g., cortical versus lacunar) as well as its location (e.g., anterior versus posterior circulation, left versus right hemisphere).The NIHSS scores are related to outcome and can identify patients who will benefit from reperfusion therapy or who are at risk of developing complications from reperfusion therapy or from the stroke itself. Notably, the NIHSS favours left hemispheric and anterior circulation strokes because the majority of the points are assigned to speech deficits and limb weakness, as opposed to extinction or limb ataxia, which favour right hemispheric or posterior circulation strokes, respectively.

Table. 1: NATIONAL INSTITUTE OF HEALTH STROKE SCALE

	Category	Description	Score
1A	Level of	Alert	0

	consciousness	Drowsy Stuporous Coma	1 2 3
1B	Level of consciousness-questions	Answers both correctly Answers one correctly Answers both incorrectly	0 1 2
1C	Level of consciousness-commands	Performs both tasks correctly Performs one task correctly Performs neither task correctly	0 1 2
2	Best gaze	Normal Partial gaze palsy: can be overcome Partial gaze palsy: corrects with oculocephalic reflex Forced gaze palsy: cannot be overcome	0 1 1 2
3	Visual fields	No visual loss Partial hemianopia Complete hemianopia Bilateral hemianopia	0 1 2 3
4	Facial Palsy	Normal symmetric movements Minor paralysis Partial paralysis Complete paralysis of one or both sides	0 1 2 3

5A	Left Arm Motor Drift	No drift Drift Some effort against gravity No effort against gravity No movement	0 1 2 3 4
5B	Right Arm Motor Drift	No drift Drift Some effort against gravity No effort against gravity No movement	0 1 2 3 4
6A	Left Leg Motor Drift	No drift Drift Some effort against gravity No effort against gravity No movement	0 1 2 3 4
6B	Right Leg Motor Drift	No drift Drift Some effort against gravity No effort against gravity No movement	0 1 2 3 4
7	Limb Ataxia	Absent Present in one limb Present in two limbs	0 1 2
8	Sensation	Normal; no sensory loss Mild-to-moderate sensory loss Severe-to-total sensory loss	0 1 2
9	Best Language	No aphasia; normal	0

		Mild-to-moderate aphasia Severe aphasia Mute; global aphasia	1 2 3
10	Dysarthria	Normal Mild-to-moderate dysarthria Severe dysarthria	0 1 2
11	Extinction/Inattention	No neglect Partial neglect Complete neglect	0 1 2

NLR and PLR are novel composite biomarkers that can be used to assess the severity of the systemic inflammatory burden in ischemic stroke. These biomarkers are easy to use and affordable. They kept their prognostic values in a single form and a mixed form with two distinguishing features: First, the combined index reflected pro-inflammatory or pro-coagulant status as well as immunosuppression, allowing for more precise selection of common biomarkers; second, the ratios were more stable than a single blood parameter, which could be influenced by several factors such as dehydration, over-hydration, and blood specimen handling.

In patients presenting with acute ischemic stroke, elevated levels of NLR and PLR have been linked to oxidative stress and increased cytokine production. The NLR is regarded as a separate risk factor for ischemic stroke and has a poor prognosis. It has been used as an indicator to reflect the prevalence of intracranial atherosclerosis. Following a stroke, the rate of insufficient recanalization, the size of the infarcted area, and poor prognosis have all been predicted using the PLR.

The NLR primarily represents inflammatory damage, whereas the PLR includes thrombosis in addition to inflammation. This may help us identify high-risk patients more effectively[20-21].

NEURO IMAGING

Brain and neurovascular imaging plays a crucial role in acute ischemic stroke by[22-23].

- ➢ Distinguishing ischemia from haemorrhage (Is there an ICH at NCCT that makes IV-tPA or EVT contraindicated, or is there a large well-established hypoattenuating infarct?)
- ➢ Excluding stroke mimics such as tumours,
- ➢ Evaluating the condition of the large cervical and intracranial arteries (Is there a proximal LVO seen on CTA that can be treated with EVT?)
- ➢ Assess the volume of irreversibly infarcted brain tissue (ie, infarction core)
- ➢ Assess the amount of potentially salvageable brain tissue at risk of infarction (ie, ischemic penumbra)

NONCONTRAST HEAD CT

- ➢ It is the most common imaging modality used for triage of acute ischemic stroke,
- ➢ It is widely available, rapid, and can easily detect intra cerebral hemorrhage.
- ➢ During the hyperacute stage of AIS, NCCT appears normal but can reveal subtle signs of early ischemia, such as gray-white matter differentiation loss, cortical swelling, hypodensity from cytotoxic edoema, hyperdense middle cerebral artery (MCA) sign, or sulcal effacement. When performed 6 hours after symptom onset, the prevalence of stroke findings on NCCT is 61%, which gradually increases 24 hours later[22]. It is worth noting that early evidence of

infarction on NCCT may indicate a poor prognosis with poor functional outcome.

- ➢ The Alberta Stroke Program Early Computed Tomography Score (ASPECTS) objectively quantifies ischemic changes in the anterior circulation of the head on NCCT and identifies patients who are unlikely to recover after reperfusion therapy.
- ➢ An ASPECTS of 10 indicates a normal NCCT, while a score of 0 indicates extensive ischemic changes.
- ➢ In one study, an ASPECTS ≤ 7 was associated with functional dependence and death at three months, and a predicted functional outcome with a sensitivity of 78% and specificity of 96%[24].

MRI BRAIN

Standard brain MRI protocols that include conventional T1-weighted, T2-weighted, FLAIR, DWI, and the apparent diffusion coefficient (ADC) map can reliably diagnose acute ischemic stroke[23].

Major drawbacks of MRI are that it is not readily available and its use may be limited by contraindications (e.g. metal implant or pacemakers) or patient intolerance (i.e. claustrophobia).

T1-weighted imaging (T1-WI) - The cerebrospinal fluid (CSF) will have a low signal intensity with relation to the brain tissue.

T2-weighted imaging (T2-WI) - The Cerebrospinal fluid will have a high signal intensity with relation to the brain tissue.

DWI is inheretently a T2 weight sequence.

- ➢ Structures with increased diffusion such as CSF will appear dark on DWI images.
- ➢ Lesions with decreased diffusion will appear bright (hyperintense signal).

ADC maps provide pure information on diffusion without any T2 weighting.

- ➢ Structures with increased diffusion such as CSF will appear bright on ADC maps.
- ➢ Lesions with decreased diffusion will appear dark (hypointense signal).

Brain MRI with DWI is superior to NCCT for the detection of acute infarction. It is the diagnostic gold standard in acute cerebrovascular syndrome to differentiate TIA from infarction and non-ischemic mimics[25].

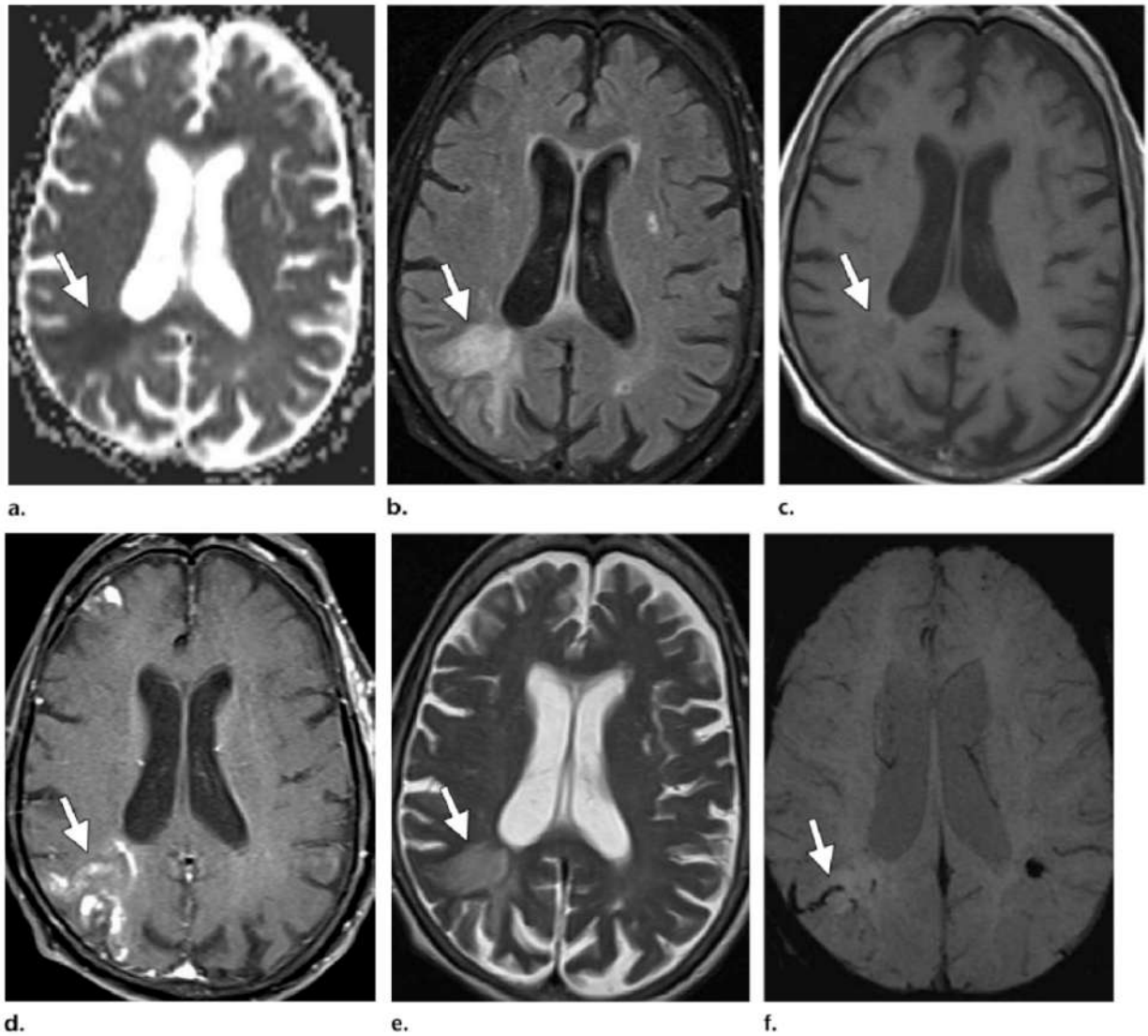


Figure 18: MRI sequences in acute ischemic stroke

(a) ADC map shows an area of low signal intensity in the right parietooccipital junction (arrow), a finding that usually persists for about 1 week.
(b) FLAIR MR image shows a corresponding area of high signal intensity (arrow).
(c) T1- weighted MR image shows a corresponding area of low signal intensity (arrow).
(d) Gadolinium-based contrast-enhanced T1-weighted MR image shows parenchymal enhancement in the affected area (arrow). Parenchymal enhancement is usually seen about 1 week after stroke.
(e) T2-weighted MR image shows a corresponding area of high signal intensity (arrow).
(f) SWI: show early blood products (arrow) indicative of hemorrhagic transformation.
These findings are indicative of an early subacute stroke, likely around 7–10 days old.

DWI- FLAIR MISMATCH

It refers to evidence of a hyperintense lesion on DWI consistent with acute infarction but no corresponding signal abnormality on FLAIR images. This mismatch indicates that the stroke was relatively recent (within 4.5 hours), as there was insufficient time for the development of a hyperintense signal on FLAIR, a sign of vasogenic edoema. This DWI-FLAIR mismatch has been used in some trials to select patients for IV-tPA treatment when the time of stroke onset is unknown or unwitnessed.

SUSCEPTIBILITY-WEIGHTED IMAGING (SWI)

SWI is particularly sensitive to compounds which distort the local magnetic field. therefore it's useful in detecting blood products, calcium, etc. SWI is the most sensitive sequences for depicting hemorrhagic transformation in patients with ischemic stroke. Hemorrhagic transformation demonstrates a spectrum of findings ranging from small petechial areas of micro bleeding to large parenchymal hematoma.

PERFUSION SCAN

Perfusion studies (Multimodal CT or MRI) can provide critical information about the volume of irreversibly damaged tissue (infarction core) and the volume of critically hypoperfused tissue that may be salvageable with reperfusion (ischemic penumbra). This will guide future treatment for patients who fall outside of the time ranges for thrombolysis or whose symptom onset is unknown[26].

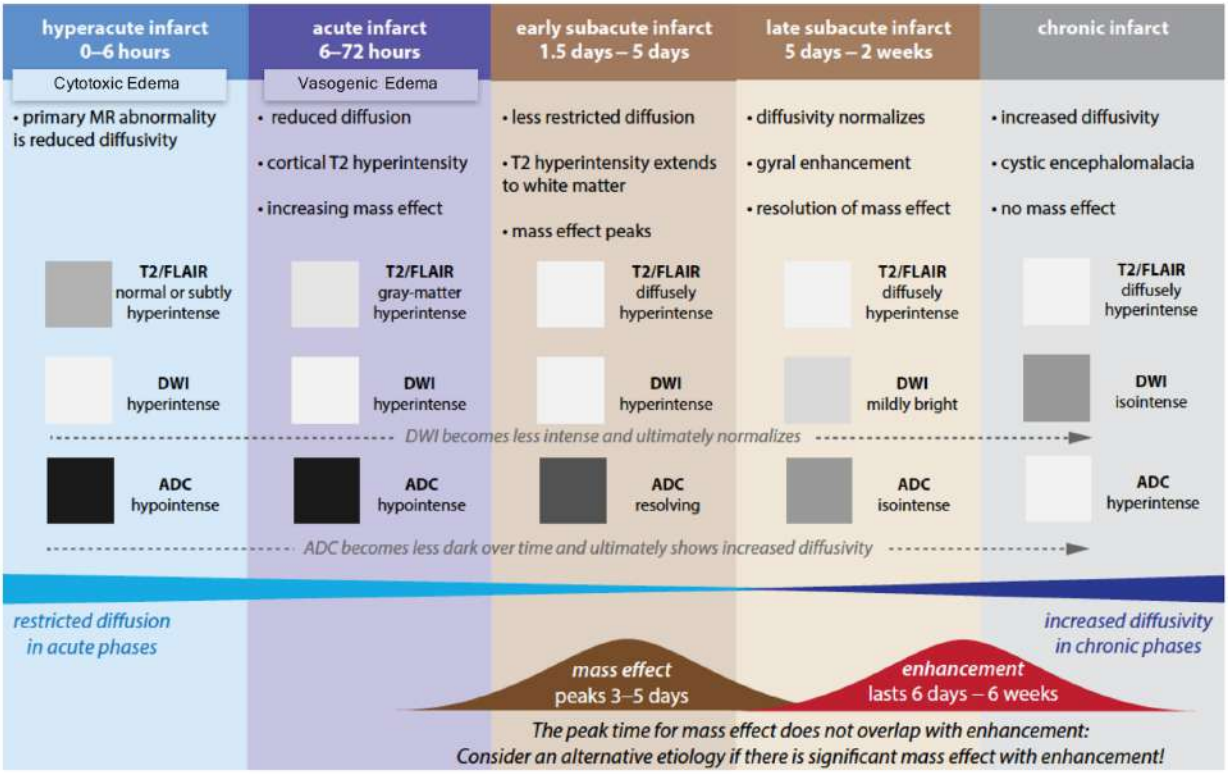


Figure 19: ISCHEMIC CHANGES ON MRI

VASCULAR IMAGING : CTA/ MRA

A head and neck CTA/MRA can detect extracranial and intracranial stenosis in the carotid and vertebral circulations, as well as an LVO. A CTA can also detect aortic dissection, which is a contraindication to IV TPA[27].

MANAGEMENT

STROKE UNITS

Key features are beneficial in stroke units[28-29].

1. A specialised stroke unit for only acute stroke patients.
2. A stroke-specific multidisciplinary care (physiotherapy, speech and language therapy, occupational therapy) and high nursing ratios.
3. Prevention of secondary brain insults by maintaining physiological homeostasis and monitoring of neurological status.
4. Bedside cardiac telemetry if atrial fibrillation has not been confirmed.
5. Thrombolysis for selected patients.

Two steps in management

1. Treatment of acute ischemic stroke
2. Secondary prevention

Treatment of acute ischemic stroke

Airway should be assessed immediately upon arrival for airway compromise. Patients who are unable to clear oral secretions or maintain airway stability should be immediately intubated (neurocritical care intubation).

Breathing: Provide supplemental O2 if oxygen saturation is <94%. Supplemental O2 is not recommended in non-hypoxic patients with ‘AIS’.

Circulation should be assessed immediately upon arrival for hemodynamic stability.Hypotension with evidence of poor perfusion (shock state) can mimic stroke.

Cerebrovascular accidents frequently have high blood pressure. The approach to blood pressure management in 'AIS' is inherently different from the approach in acute hemorrhagic stroke. Obtain peripheral intravenous (IV) access & Initiate labwork.

DISABILITY: Perform a focused neurological exam and obtain a point-of-care glucose.

ECG: Baseline ECG assessment is recommended.

Table. 2: TARGETS FOR MAINTAINING HOMEOSTASIS IN ACUTE ISCHAEMIC STROKE PATIENTS[30]

Variable	Target

Oxygen saturation	Oxygen supplementation if saturation <95%
Hydration	Assessed within 4 hours using multiple tools
Swallowing	Screen for dysphagia with validated tool within 4 hours and before any oral intake (including medication)
Plasma glucose	5–15 mmol/L
Blood pressure	No target. Indication for treatment: ➢ ≥185 or ≥110 mmHg ➢ Hypertensive encephalopathy, nephropathy, cardiac failure or myocardial infarction ➢ Aortic dissection ➢ Pre-eclampsia/eclampsia

The goal of acute ischaemic stroke treatment is to recanalize occluded cerebral arteries and reperfuse the ischaemic penumbra in order to salvage ischaemic viable brain tissue.

Table. 3: INDICATIONS AND CONTRA-INDICATIONS OF THROMBOLYSIS[31-33]

INDICATION	CONTRA-INDICATION
Clinical diagnosis of stroke	Systolic blood pressure >185 mmHg or diastolic blood pressure >110 mmHg

Stroke onset within ≤3 hours	International normalised ratio (INR) >1.7
CT scan showing no haemorrhage or edema >1/3rd of the MCA territory.	Direct oral anticoagulant (DOAC) use within 48 hours
Age 18-80 years	Platelets <100 000/mm3
NIHSS >3 and <25	Active internal bleeding
	Intracranial or intraspinal surgery Severe head trauma within 3 months
	Intracerebral vascular malformations, Intracranial malignancy, Previous intracerebral haemorrhage
	Ischaemic stroke within 3 months
	Infective endocarditis

Intravenous rtPA (alteplase) is proven and licenced to improve functional outcome in acute ischaemic stroke up to 4.5 hours after symptom onset. Which cleaves plasminogen on the surface of thrombi to form plasmin,a powerful endogenous fibrinolytic enzyme[32]. Injection Alteplase 0.9 mg/kg iv (maximum dose of 90 mg administered as initial bolus and remaining 90% over 1 hour).

ENDOVASCULAR PROCEDURES

ALGORITHM FOR INDICATION OF THROMBECTOMY IN ACUTE ISCHEMIC STROKE[34-35]

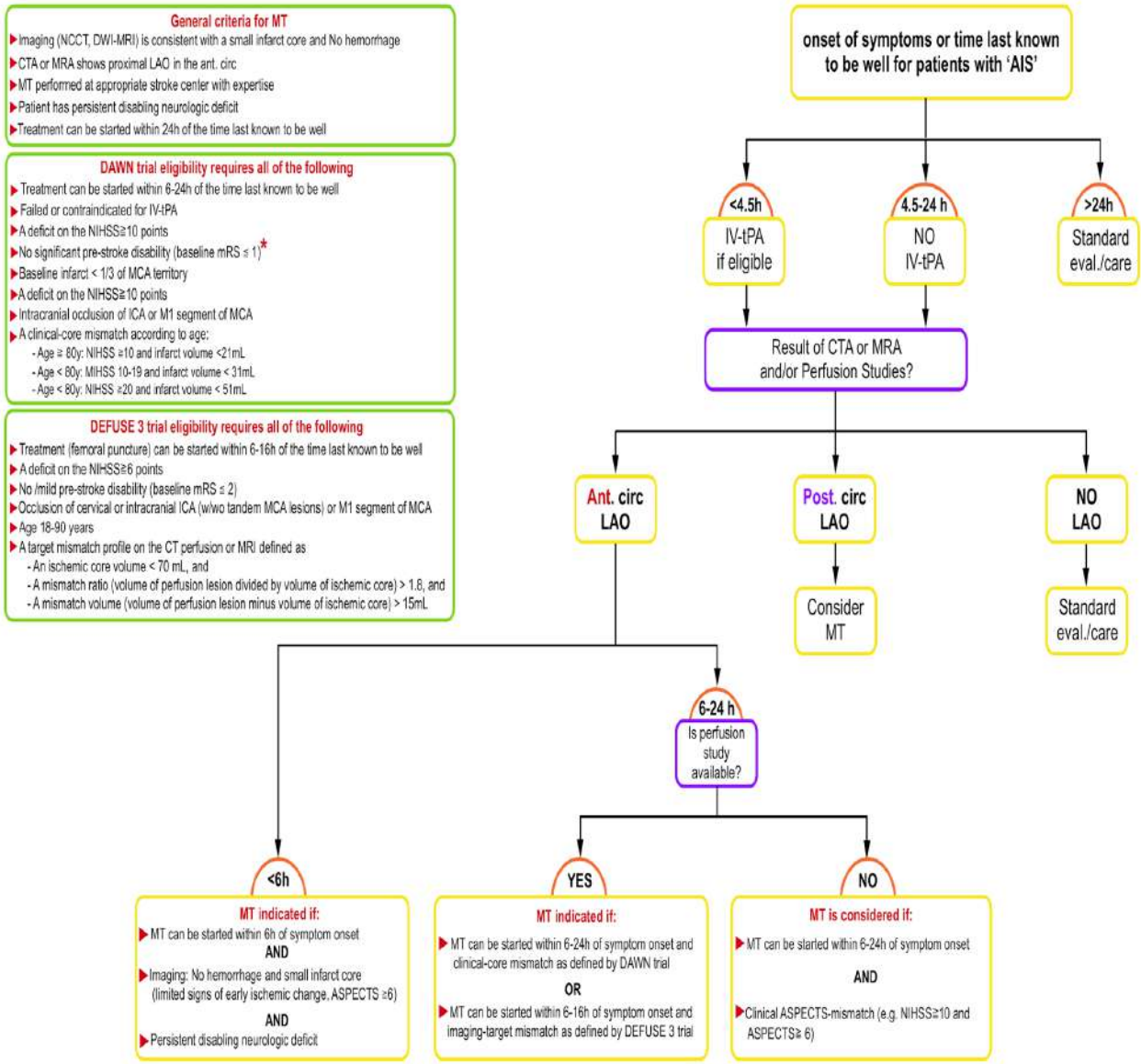


Figure 20: ALGORITHM FOR INDICATION OF THROMBECTOMY

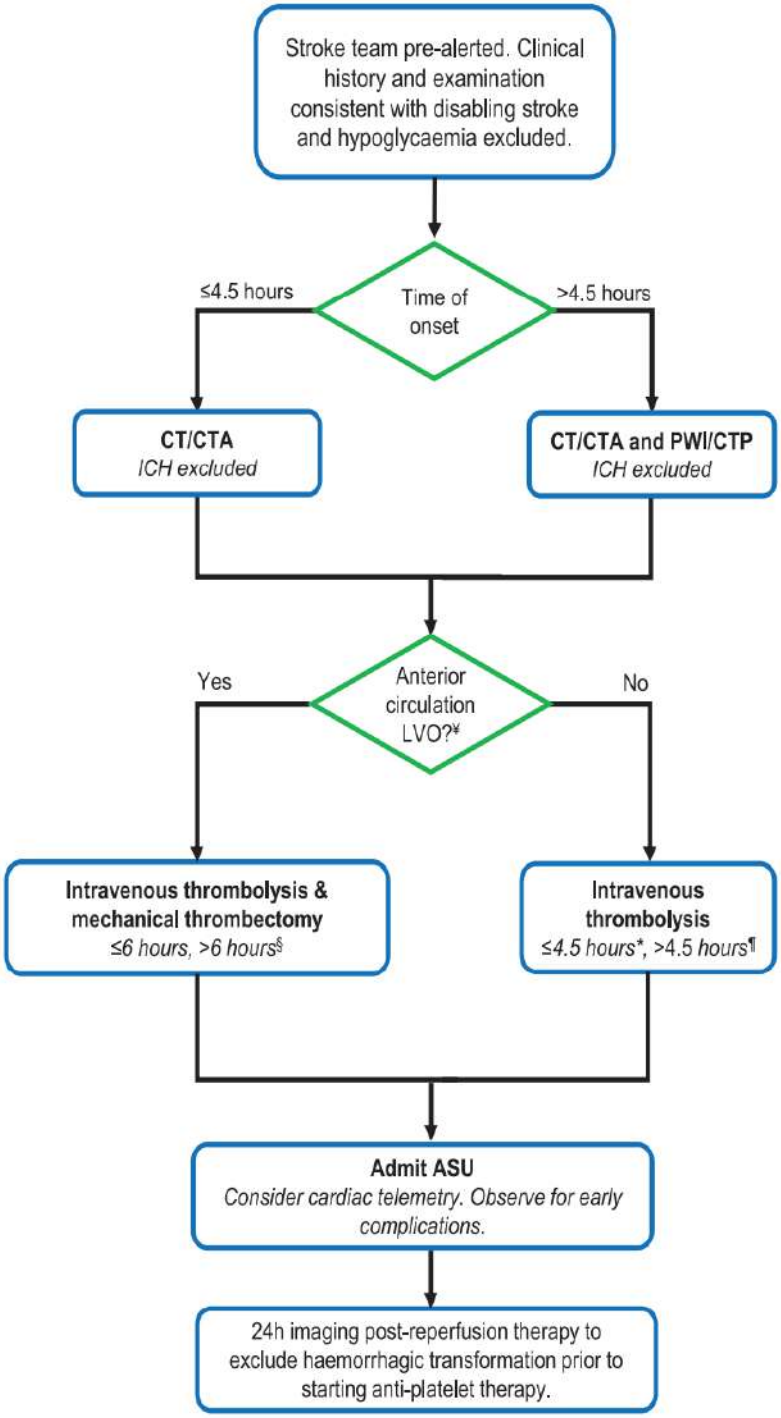


Figure 21: OVER VIEW OF REPERFUSION THERAPY

SECONDARY PREVENTION

ALGORITHM FOR SECONDARY PREVENTION IN ACUTE ISCHEMIC STROKE[36-37]

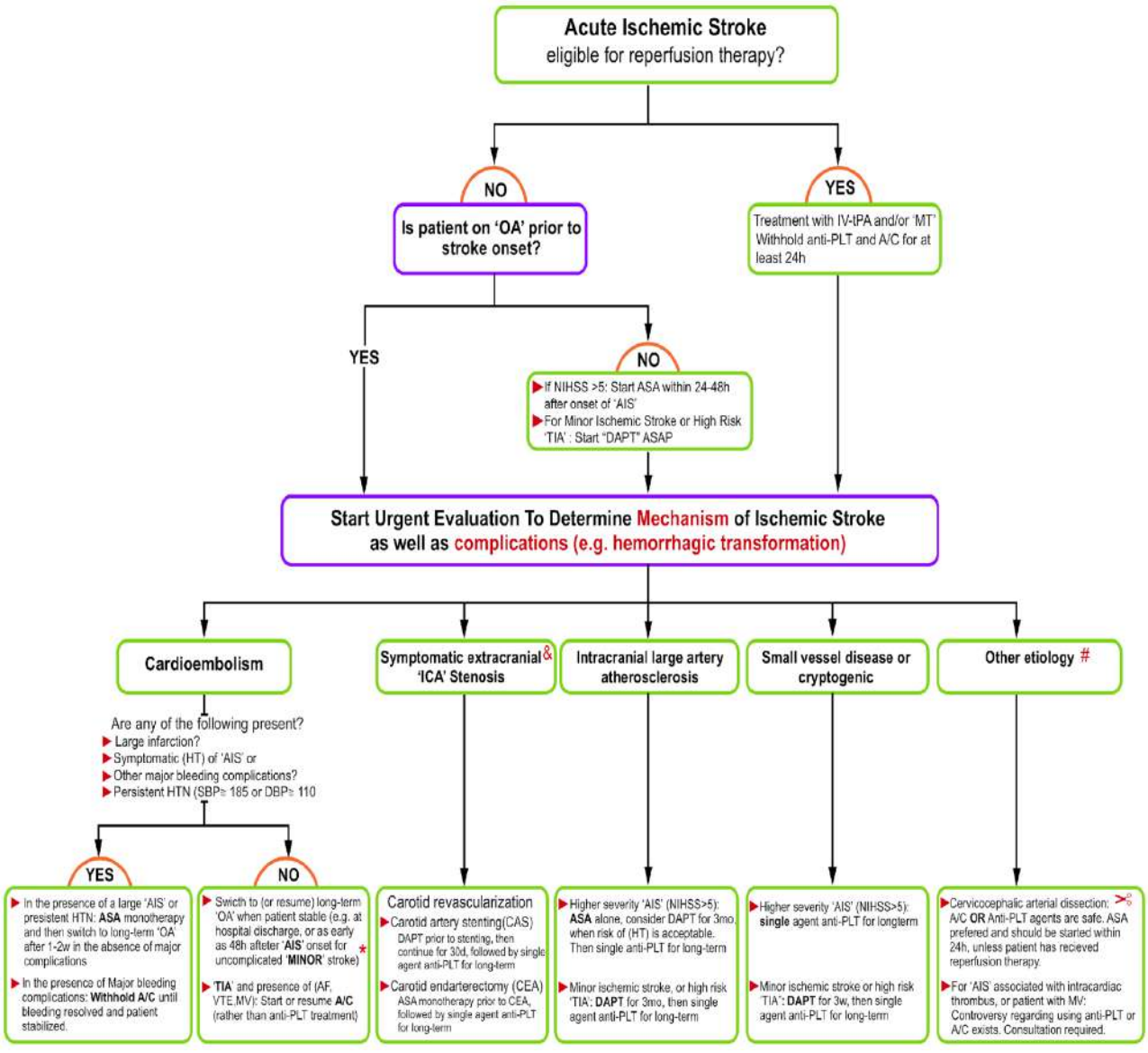


Figure 22: ALGORITHM FOR SECONDARY PREVENTION

STUDY SETTING

Patient who are admitted in the Medical wards in the Department of Internal Medicine , Rajiv gandhi government general Hospital and Madras medical College, Chennai-03

ETHICAL APPROVAL

Approval was obtained from the Institute ethical committee in order to perform the study.

STUDY GROUP

Patients of age > 18 years who presents with acute ischemic stroke within 2 days of onset, who will be admitted in Internal Medicine department and whom will undergo Computerised Tomography of Brain at Rajiv gandhi government general Hospital and Madras medical college.

STUDY DESIGN

Prospective observational study.

POPULATION STUDIED : 80

DURATION OF THE STUDY

6 months (June 2022 to November 2022)

CONSENT

Written informed consent was obtained from all the patients included in the study. For patients who could not give consent, it was obtained from the close attenders of the patient.

INCLUSION CRITERIA

- ➢ Stroke within 2 days of onset, confirmed by history, neurological examination and imaging modalities of both anferior & posterior circulation as per WHO Criteria.
- ➢ Patients who are willing to give written informed.

EXCLUSION CRITERIA

- ➢ Patients with hemorrhagic stroke and previous attack of ischemic stroke.
- ➢ Patients with comorbid medical illness likely to interfere with platelet function or morphology like Cardiac bypass surgary, chronic liver disease, leukemia, active infections and autoimmune diseases.
- ➢ Patients with Ischemic Heart disease, Congenital Heart disease, Pulmonary disease and Aspiration pneumonitis.
- ➢ Patients receiving medication likely to interfere with platelet morphology or function(NSAIDs) and QT prolonging drugs (Anti-arrhythmic/TCA/Macrolides).

- ➢ Patients presenting 48hrs after the onset of neurological symptoms.
- ➢ Patients < 18 years of Age.

MATERIALS USED

- ➢ Sphygmomanometer
- ➢ Computerised Tomography film – Brain
- ➢ Electrocardiogram (QT interval calculated by Bazett formula QTc = QT interval/ √(RR interval)
- ➢ Blood samples for complete blood count.

METHOD OF DATA COLLECTION

Sample size : 80 cases will be studied.

PROTOCOL OF THE STUDY

- ➢ Population of the study was defined as per inclusion and exclusion criteria.
- ➢ All patients of age > 18 years who got admitted with acute ischemic stroke in General Medicine wards were selected.
- ➢ All patients who are included in the study had a clinical diagnosis of stroke and supported by immediate neuroimaging.
- ➢ Computed Tomography of Brain was done immediately after admission.

- ➢ For every patients, the following details were collected in a semi structured proforma.
- ➢ Complete clinical history, address and contact number, risk factors like moking, alcohol, hypertension, diabetes mellitus , and previous admission history (if any) and treatment history, GCS, Systolic and Diastolic Blood pressure were obtained.
- ➢ Patients were also assigned a grade based on NIHSS (National Institute of Health Stroke Severity) scale.

Table. 4: NIHSS SCALE

NIHSS SCORE	Grading
1-4	Mild
5-15	Moderate
16-20	Moderate-severe
21-42	Severe

- ➢ Blood samples were obtained by venipuncture with in 24 hours of admission and Routine investigations done.

- ➢ Comparing Neutrophil lymphocyte ration, Platelet lymphocyte ratio and ECG findings in all the patients during the course of hospital stay based upon the NIHSS grading.

FOLLOWING PARAMETERS WERE TAKEN UP FOR THE STUDY

- ➢ Neutrophil lymphocyte ratio
- ➢ Platelet lymphocyte ratio
- ➢ Electrocardiographic findings

CONFLICT OF INTEREST

- ➢ None

STATISTICAL ANALYSIS

The results of the study titled "A STUDY ON CLINICAL SIGNIFICANCE OF HEMATOLOGICAL INDICES, ELECTRO CARDIOGRAPHIC CHANGES BY USING NATIONAL INSTITUTE OF HEALTH STROKE SCORE (NIHSS) IN ACUTE ISCHEMIC STROKE IN TERTIARY CARE CENTRE" conducted among 80 patients are as follows:

Age of the study participants:

Table 5: Age of the study participants (n=80)

Age group (In years)	NIHSS grading on admission				Fischer exact test value	P value
	Mild	Moderate	Severe	Total		
<40	1 4.2%	2 4.1%	2 28.6%	5 6.3%	8.113	0.366
41-50	5 20.8%	8 16.3%	1 14.3%	14 17.5%		
51-60	11 45.8%	19 38.8%	1 14.3%	31 38.8%		
61-70	4 16.7%	13 26.5%	3 42.9%	20 25%		
71-80	3 12.5%	7 14.3%	0	10 12.5%		
Total	24 100%	49 100%	7 100%	80 100%		

Among the study participants, 6.3% were less than 40 years of age, 17.5% were from 41 to 50 years, 38.8% were from 51 to 60 years, 25% were from 61 to 70 years and 12.5% were from 71 to 80 years of age.

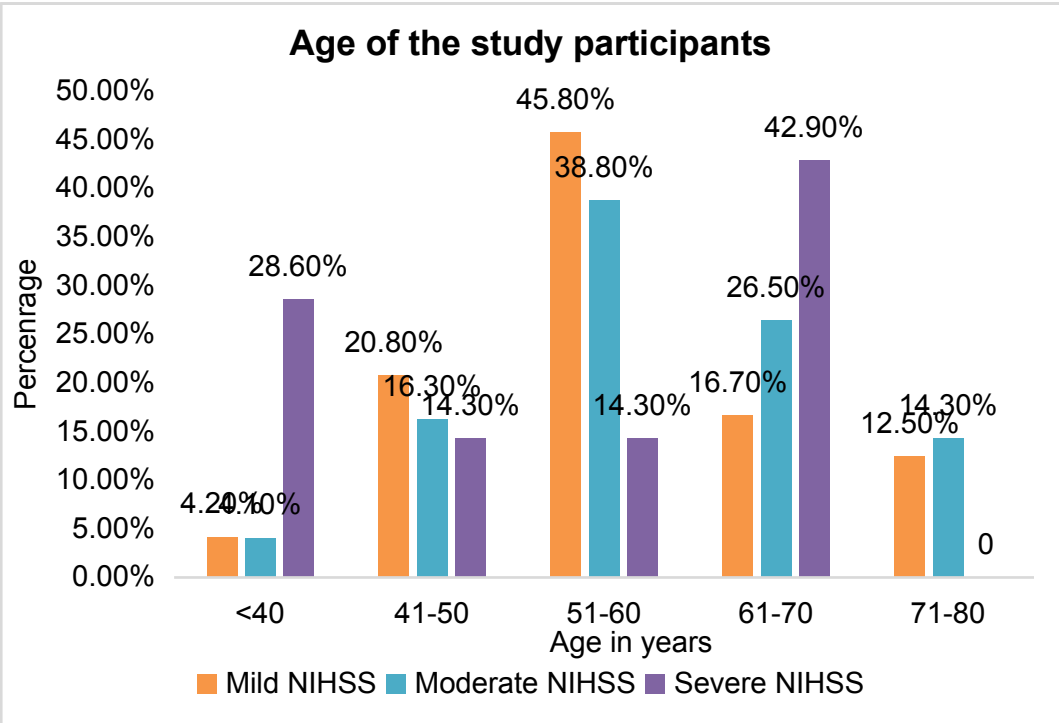
In participants who had severe NIHSS (National Institute of Health Stroke Score) during admission, 28.6% were less than 40 years of age, 14.3% were from 41 to 50 years, 14.3% were from 51 to 60 years and 42.9% were from 61 to 70 years.

In participants who had moderate NIHSS during admission, 4.1% were less than 40 years of age, 16.3% were from 41 to 50 years, 38.8% were from 51 to 60 years, 26.5% were from 61 to 70 years and 14.3% were from 71 to 80 years of age.

In participants who had mild NIHSS during admission, 4.2% were less than 40 years of age, 20.8% were from 41 to 50 years, 45.8% were from 51 to 60 years, 16.7% were from 61 to 70 years and 12.5% were from 71 to 80 years of age.

The difference was not statistically significant by Fischer exact test.

Figure 23: Age of the study participants (n=80)



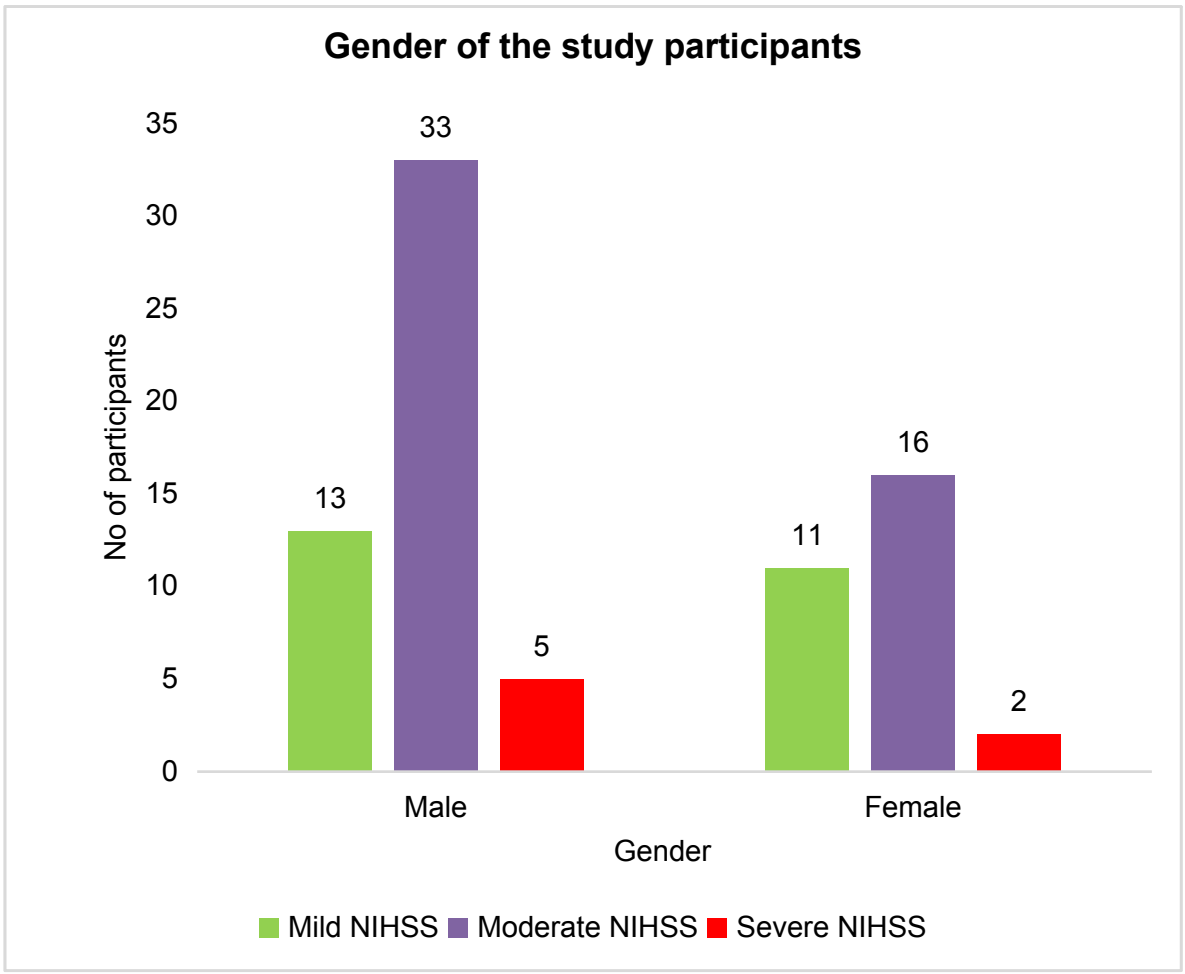
Gender of the study participants:

Table 6: Gender of the study participants (n=80)

Gender	NIHSS grading on admission				Fischer exact test value	P value
	Mild	Moderate	Severe	Total		
Male	13 54.2%	33 67.3%	5 71.4%	51 63.7%	1.405	0.591
Female	11 45.8%	16 32.7%	2 28.6%	29 36.3%		
Total	24 100%	49 100%	7 100%	80 100%		

Among the study participants, 51 were males and 29 were females. In participants who had severe NIHSS during admission, 5 were males and 2 were females. In participants who had moderate NIHSS during admission, 33 were males and 16 were females. In participants who had mild NIHSS during admission, 13 were males and 11 were females. This difference was not statistically significant by Fischer exact test.

Figure 24: Gender of the study participants (n=80)



Mean systolic BP of the study participants:

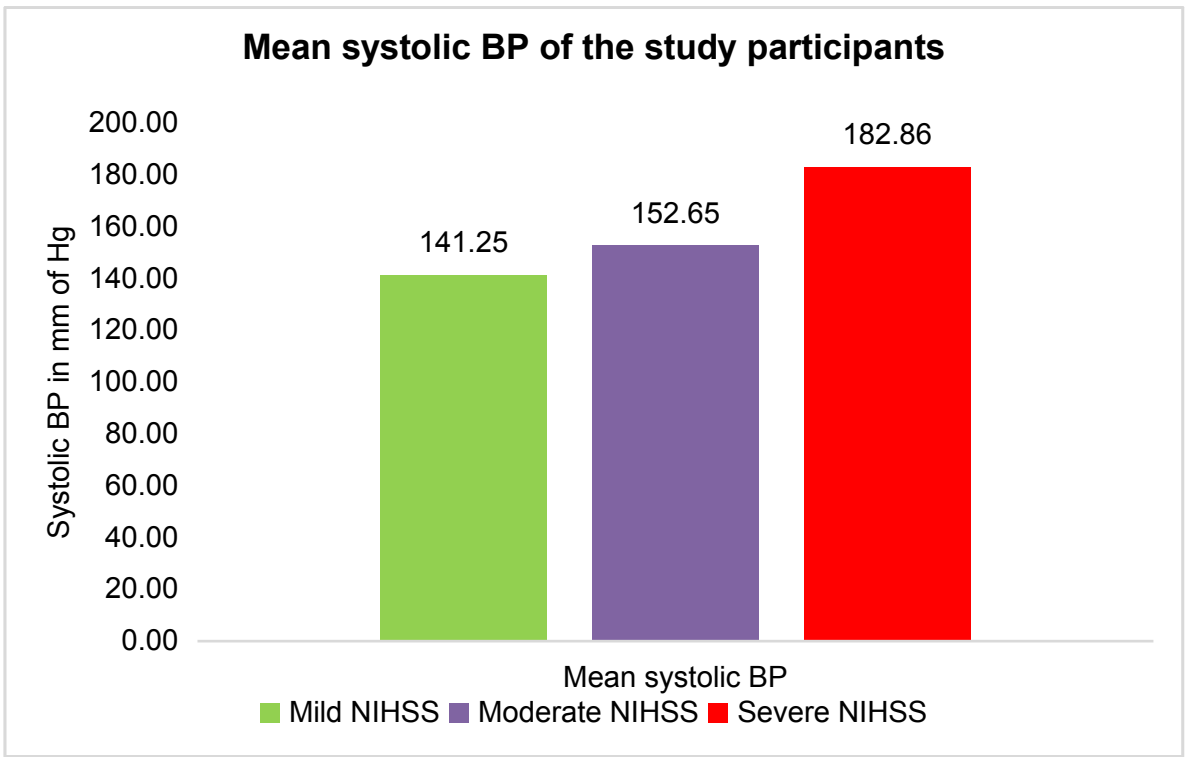
Table 7: Mean systolic BP of the study participants (n=80)

Mean systolic BP (in mm Hg)	NIHSS grading on admission			F value	P value
	Mild	Moderate	Severe		
Mean ± SD	141.25 ± 18.01	152.65 ± 18.57	182.86 ± 27.51	12.750	**<0.001***

*- statistically significant by ANOVA test

The mean systolic blood pressure of participants who had severe NIHSS on admission was 182.86 ± 27.51, who had moderate NIHSS on admission was 152.65 ± 18.57 and who had mild NIHSS on admission was 141.25 ± 18.01 (mm of Hg). This difference was statistically significant by ANOVA test.

Figure 25: Mean systolic BP of the study participants (n=80)



Mean diastolic BP of the study participants:

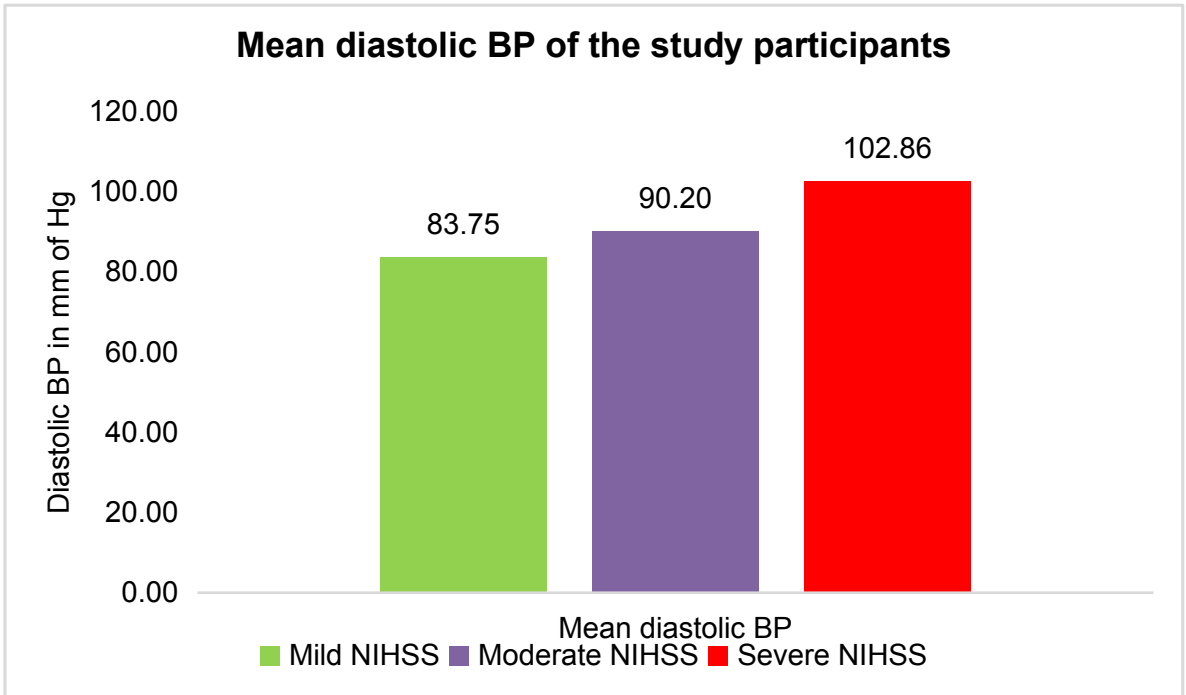
Table 8: Mean diastolic BP of the study participants (n=80)

Mean diastolic BP (in mm Hg)	NIHSS grading on admission			F value	P value
	Mild	Moderate	Severe		
Mean ± SD	83.75 ± 10.13	90.20 ± 10.89	102.86 ± 7.55	9.463	**<0.001***

*- statistically significant by ANOVA test

The mean diastolic blood pressure of participants who had severe NIHSS on admission was 102.86 ± 7.55, who had moderate NIHSS on admission was 90.20 ± 10.89 and who had mild NIHSS on admission was 83.75 ± 10.13 (mm of Hg). This difference was statistically significant by ANOVA test.

Figure 26: Mean diastolic BP of the study participants (n=80)



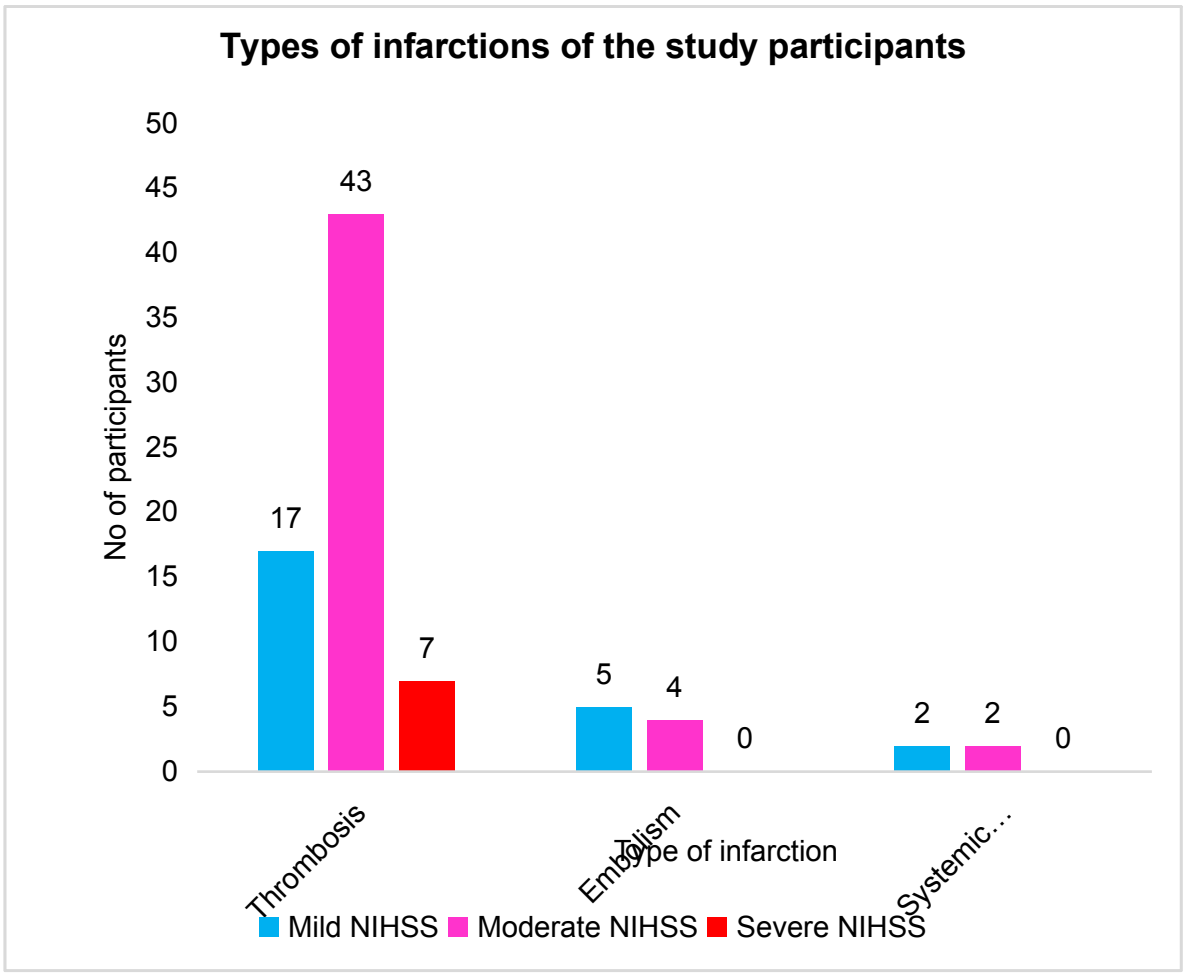
Types of infarctions of the study participants:

Table 9: Types of infarctions of the study participants (n=80)

Types of infarctions	NIHSS grading on admission				Fischer exact test value	P value
	Mild	Moderate	Severe	Total		
Thrombosis	17 70.8%	43 87.8%	7 100%	67 83.8%	4.088	0.304
Embolism	5 20.8%	4 8.2%	0	9 11.3%		
Systemic hypoperfusion	2 8.3%	2 4.1%	0	4 5%		
Total	24 100%	49 100%	7 100%	80 100%		

Thrombosis was the major type of infarction found in 67 participants, followed by embolism in 9 participants and systemic hypoperfusion in 4 participants. In participants who had severe NIHSS during admission, all participants (7 participants) had thrombosis. In participants who had moderate NIHSS during admission, 43 had thrombosis, 4 had embolism and 2 had systemic hypoperfusion. In participants who had mild NIHSS during admission, 17 had thrombosis, 5 had embolism and 2 had systemic hypoperfusion. This difference was not statistically significant by Fischer exact test.

Figure 27: Types of infarctions of the study participants (n=80)



Duration of hospitalization among the study participants:

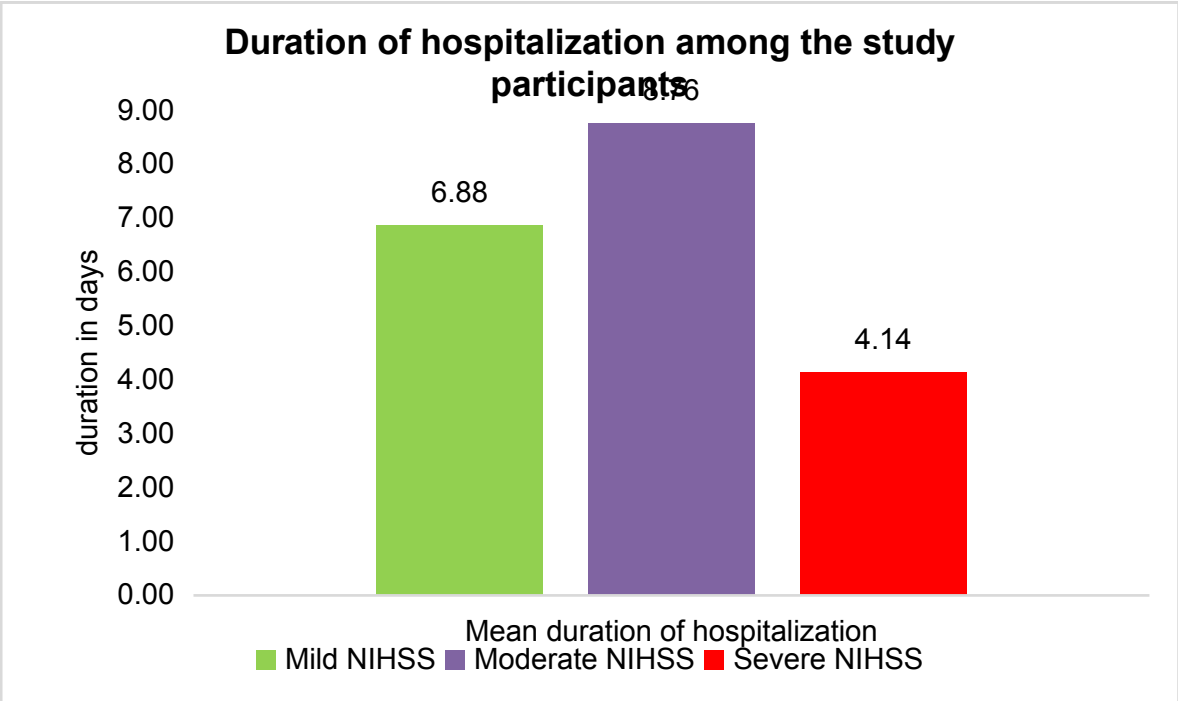
Table 10: Duration of hospitalization among the study participants (n=80)

Duration of hospitalization (in days)	NIHSS grading on admission			F value	P value
	Mild	Moderate	Severe		
Mean ± SD	6.88 ± 1.91	8.76 ± 2.98	4.14 ± 3.62	10.356	**<0.001***

*- statistically significant by ANOVA test

The mean duration of hospitalization of participants who had severe NIHSS on admission was 4.14 ± 3.62, who had moderate NIHSS on admission was 8.76 ± 2.98and who had mild NIHSS on admission was 6.88 ± 1.91 (in days). This difference was statistically significant by ANOVA test.

Figure 28: Duration of hospitalization among the study participants (n=80)



Mean NLR among the study participants:

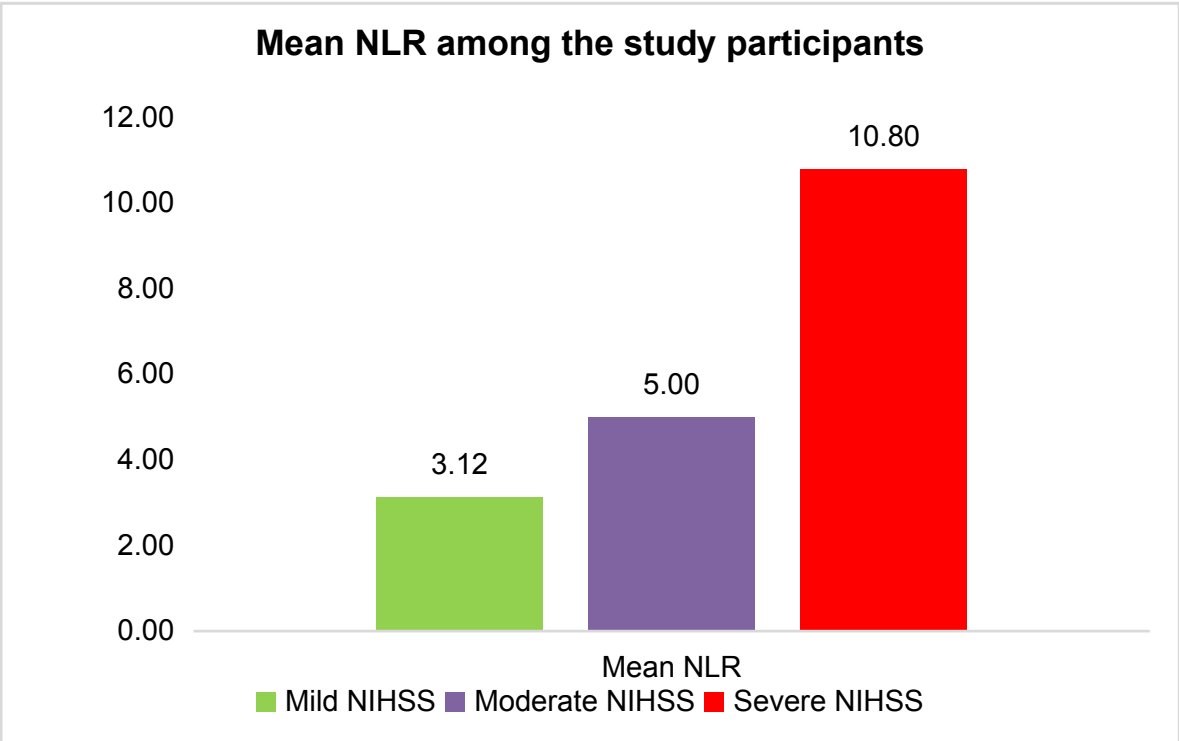
Table 11: Mean NLR among the study participants (n=80)

Mean NLR	NIHSS grading on admission			F value	P value
	Mild	Moderate	Severe		
Mean ± SD	3.12 ± 0.95	5.00 ± 1.99	10.80 ± 3.05	33.69	**<0.001***

*- statistically significant by ANOVA test

The mean Neutrophil-Lymphocyte Ratio of participants who had severe NIHSS on admission was 10.80 ± 3.05, who had moderate NIHSS on admission was 5.00 ± 1.99 and who had mild NIHSS on admission was 3.12 ± 0.95. This difference was statistically significant by ANOVA test.

Figure 29: Mean NLR among the study participants (n=80)



Mean PLR among the study participants:

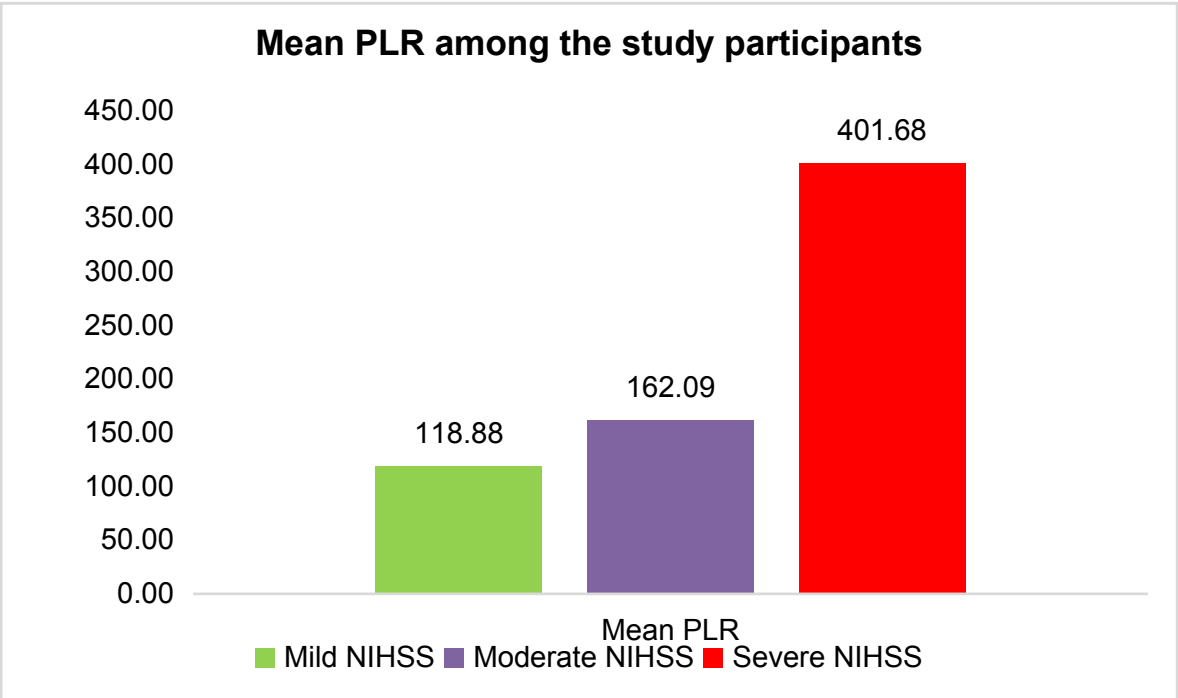
Table 12: Mean PLR among the study participants (n=80)

Mean PLR	NIHSS grading on admission			F value	P value
	Mild	Moderate	Severe		
Mean ± SD	118.88 ± 23.25	162.09 ± 58.61	401.68 ± 168.58	27.856	**<0.001***

*- statistically significant by ANOVA test

The mean Platelet lymphocyte ratio of participants who had severe NIHSS on admission was 401.68 ± 168.58, who had moderate NIHSS on admission was 162.09 ± 58.61 and who had mild NIHSS on admission was 118.88 ± 23.25. This difference was statistically significant by ANOVA test.

Figure 30: Mean PLR among the study participants (n=80)



Hospital mortality of the study participants:

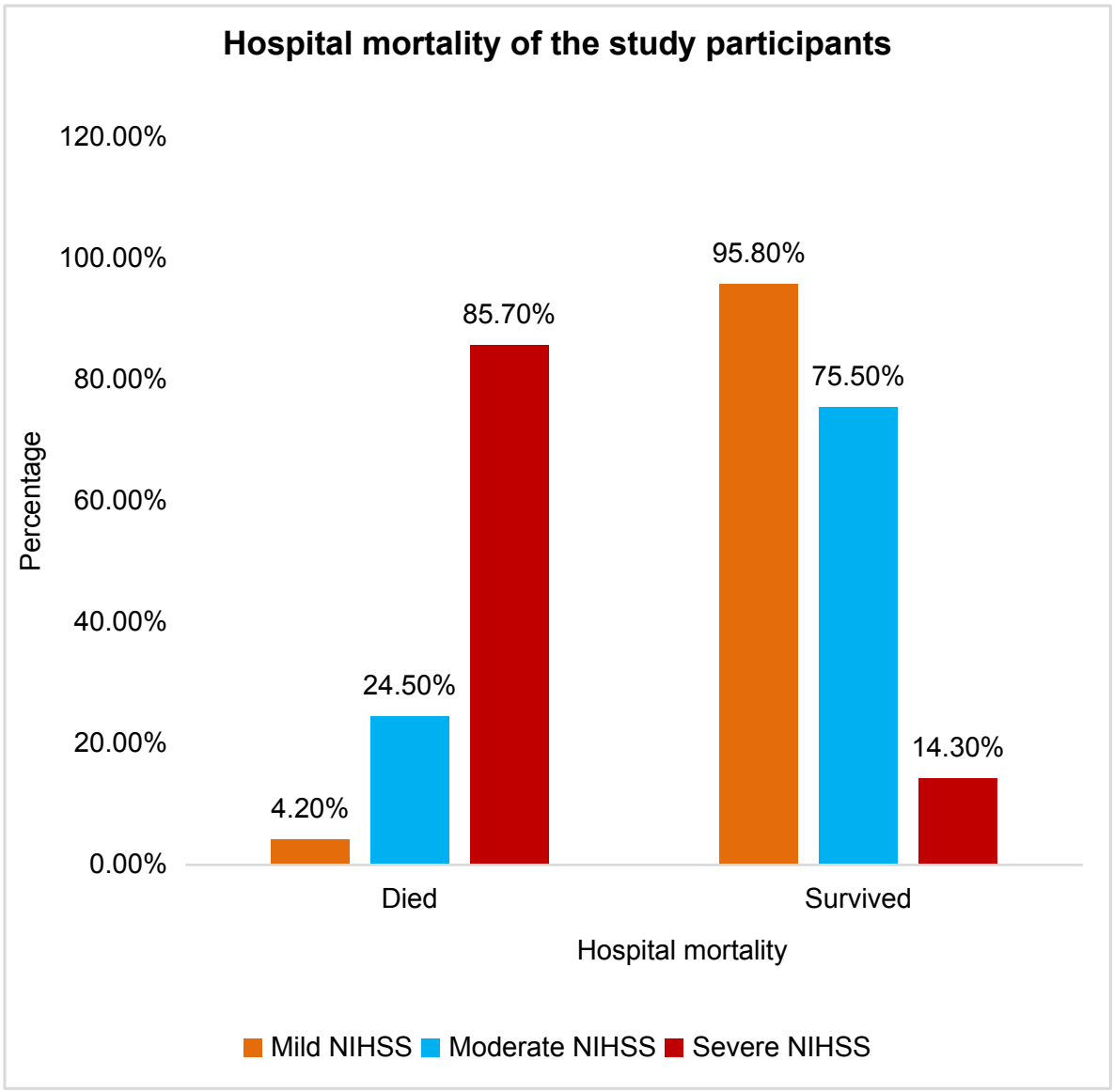
Table 13: Hospital mortality of the study participants (n=80)

Hospital mortality	NIHSS grading on admission				Fischer exact test value	P value
	Mild	Moderate	Severe	Total		
Died	1 4.2%	12 24.5%	6 85.7%	19 23.8%	17.553	**<0.001***
Survived	23 95.8%	37 75.5%	1 14.3%	61 76.3%		
Total	24 100%	49 100%	7 100%	80 100%		

*- statistically significant by Fischer exact test

Among the study participants, 23.8% died and 76.3% survived. In participants who had severe NIHSS during admission, 85.7% participants died and 14.3% survived. In participants who had moderate NIHSS during admission, 24.5% participants died and 75.5% survived. In participants who had mild NIHSS during admission, 4.2% of participants died and 95.8% survived. This difference was statistically significant by Fischer exact test.

Figure 31: Hospital mortality of the study participants (n=80)



NIHSS grading on admission and 2 days after admission:

Table 14: NIHSS grading on admission and 2 days after admission (n=80)

Mean ± SD	NIHSS grading on admission		
	Mild	**Moderate**	**Severe**
NIHSS on admission	3.71 ± 0.46	8.14 ± 2.69	18.29 ± 2.92
NIHSS on day 2	4.08 ± 1.81	10.10 ± 6.59	24.43 ± 3.55
Paired T test value	0.952	2.883	6.557
P value	0.351	**0.006***	**0.001***

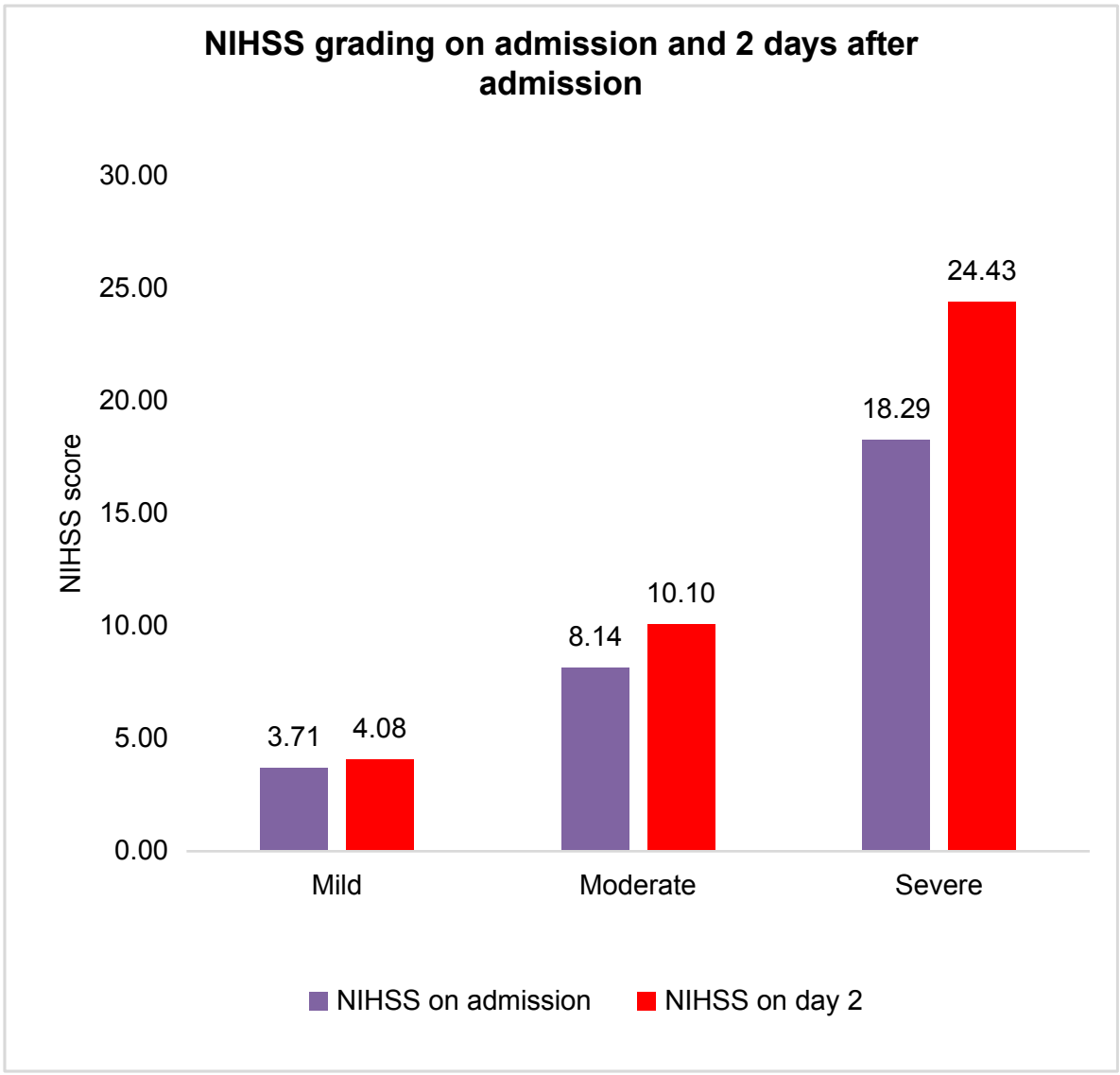
*- statistically significant by paired t test

In participants who had severe NIHSS grade on admission, the mean NIHSS score during admission was 18.29 ± 2.92 and mean NIHSS score on day 2 was 24.43 ± 3.55. This increase was statistically significant by paired t test.

In participants who had moderate NIHSS grade on admission, the mean NIHSS score during admission was 8.14 ± 2.69 and mean NIHSS score on day 2 was 10.10 ± 6.59. This increase was statistically significant by paired t test.

In participants who had mild NIHSS grade on admission, the mean NIHSS score during admission was 3.71 ± 0.46 and mean NIHSS score on day 2 was 4.08 ± 1.81. This increase was not statistically significant by paired t test.

Figure 32: NIHSS grading on admission and 2 days after admission (n=80)



ECG findings among participants with mild NIHSS on admission:

Table 15: ECG findings among participants with mild NIHSS on admission (n= 24)

ECG finding	Frequency	Percentage
ST depression	8	33.3
Normal sinus rhythm	6	25
T wave inversion in lead V5, V6, I, aVL	6	25
Atrial fibrillation	5	20.8
Left axis deviation	2	8.3
aVR ST elevation with diffuse ST depression	2	8.3

Among participants with mild NIHSS on admission, ST depression was found in 8 participants, Normal sinus rhythm was in 6 participants, T wave inversion in lead V5, V6, I, aVL was in 6 participants, Atrial fibrillation was in 5 participants, Left axis deviation was in 2 participants and aVR ST elevation with diffuse ST depression was in 2 participants.

ECG findings among participants with moderate NIHSS on admission:

Table 16: ECG findings among participants with moderate NIHSS on admission (n= 49)

ECG finding	Frequency	Percentage
ST depression in V5, V6, I, aVL	12	24.5
T wave inversion in lead V5, V6, I, aVL	10	20.4
T wave inversion in lead V1-V6	9	18.4
Left axis deviation	6	12.2
LBBB	4	8.2
aVR ST elevation with diffuse ST depression	3	6.1
Normal sinus rhythm	3	6.1
Left axis deviation	2	4.1
Sinus tachycardia	2	4.1
RBBB	1	2.0
QT prolongation	1	2.0
Sinus bradycardia	1	2.0
Atrial fibrillation	1	2.0

Among participants with moderate NIHSS on admission, 12 participants had ST depression in V5, V6, I, aVL, 10 had T wave inversion in lead V5, V6, I, aVL, 9 had T wave inversion in lead V1-V6, 6 had Left axis deviation, 4 had LBBB, 3 had aVR ST elevation with diffuse ST depression, 3 had Normal sinus rhythm, 2 had Left axis

deviation, 2 had Sinus tachycardia, 1 had RBBB, 1 had QT prolongation, 1 had Sinus bradycardia and 1 had Atrial fibrillation

ECG findings among participants with severe NIHSS on admission:

Table 17: ECG findings among participants with severe NIHSS on admission (n= 7)

ECG finding	Frequency	Percentage
ST depression	4	57.1
QT prolongation	4	57.1
T wave inversion in lead V1-V6	3	42.9
T wave inversion in lead V5, V6	2	28.6
Left axis deviation	2	28.6
T wave inversion in lead V5, V6, I, aVL	2	28.6
LBBB	1	14.3

Among participants with severe NIHSS on admission, 4 had ST depression, 4 had QT prolongation, 3 had T wave inversion in lead V1-V6, 2 had T wave inversion in lead V5, V6, 2 had Left axis deviation, 2 had T wave inversion in lead V5, V6, I, aVL and 1 had LBBB.

Comparison of survival among severe NIHSS participants:

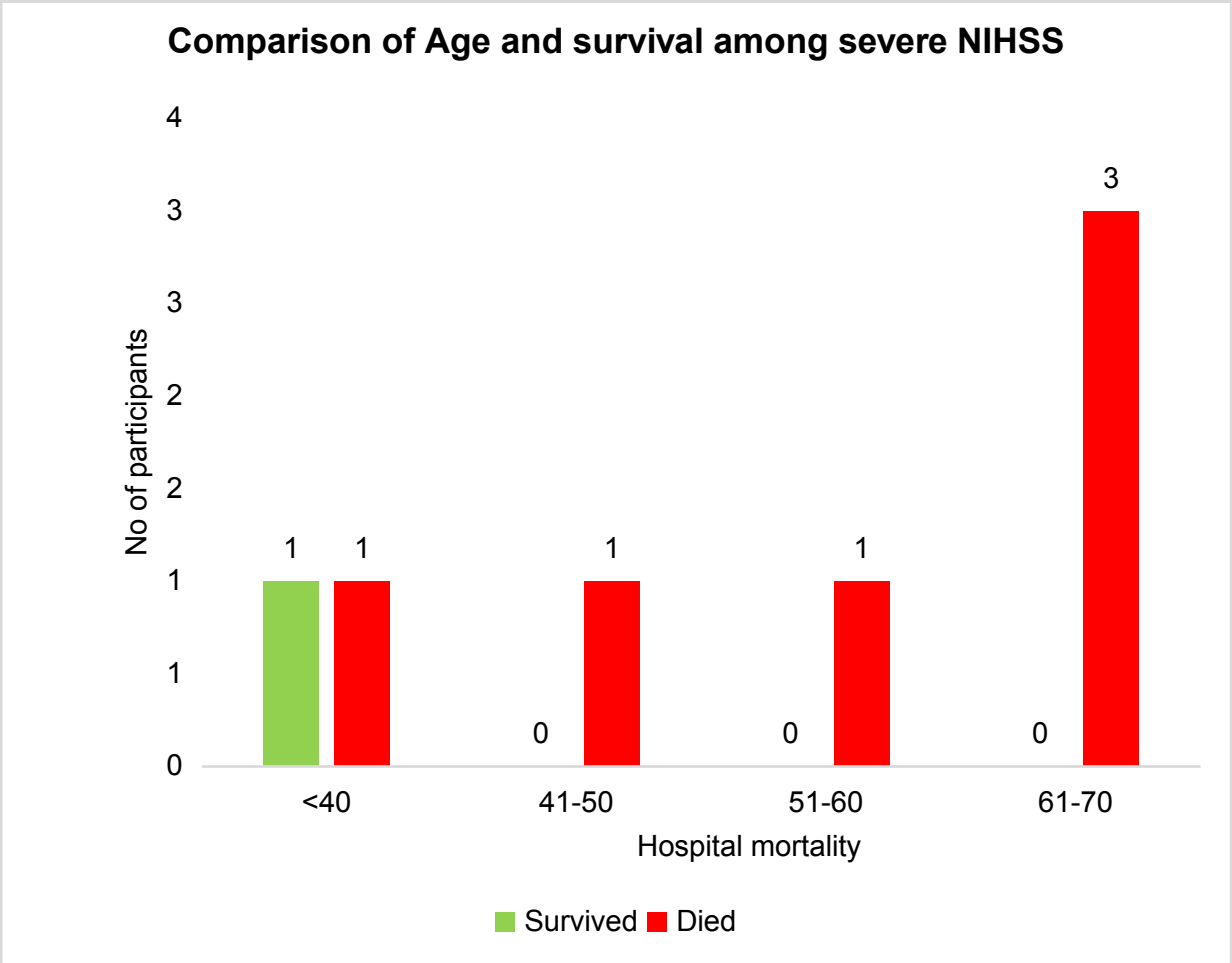
Comparison of Age and survival among severe NIHSS participants:

Table 18: Comparison of Age and survival among severe NIHSS participants (n=7)

Age group (In years)	Survived	Death	Total	Fischer exact test value	P value
<40	1 100%	1 16.7%	2 28.6%	3.446	0.571
41-50	0	1 16.7%	1 14.3%		
51-60	0	1 16.7%	1 14.3%		
61-70	0	3 50%	3 42.9%		
Total	1 100%	6 100%	7 100%		

Among participants with severe NIHSS on admission, 1 survived in less than 40 years and 1 died in less than 40 years of age. 1 died in 41 to 50 years, 1 died in 51 to 60 years and 3 died in 61 to 70 years of age. This difference was not statistically significant by Fischer exact test.

Figure 33: Comparison of Age and survival among severe NIHSS participants (n=7)



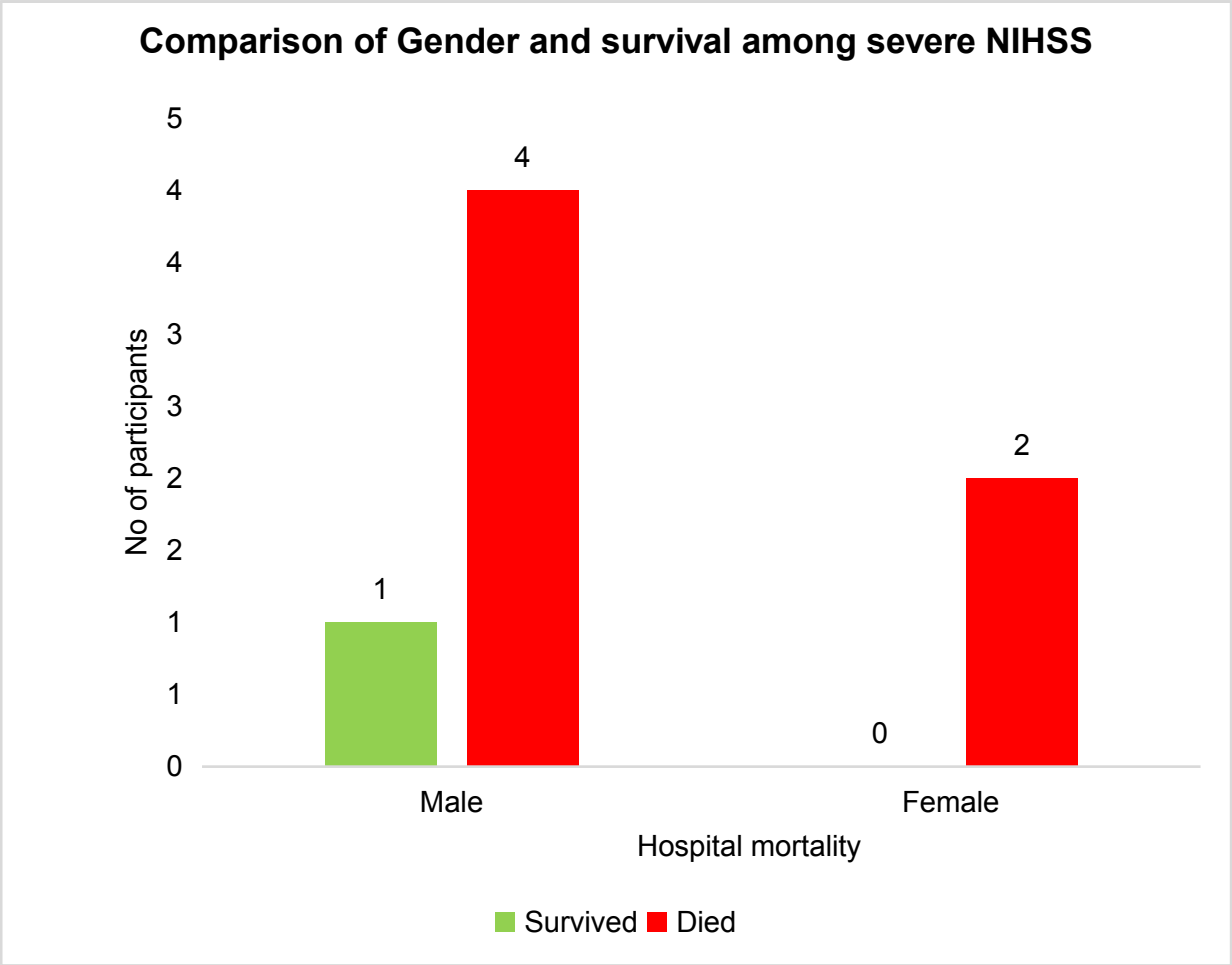
Comparison of Gender and survival among severe NIHSS participants:

Table 19: Comparison of Gender and survival among severe NIHSS participants (n=7)

Gender	Survived	Death	Total	Fischer exact test value	P value
Male	1 100%	4 66.7%	5 71.4%	0.738	0.714
Female	0	2 33.3%	2 28.6%		
Total	1 100%	6 100%	7 100%		

Among participants with severe NIHSS on admission, 1 survived in male and n one survived in female. 4 died in males and 2 survived in females This difference was not statistically significant by Fischer exact test.

Figure 34: Comparison of Gender and survival among severe NIHSS participants (n=7)



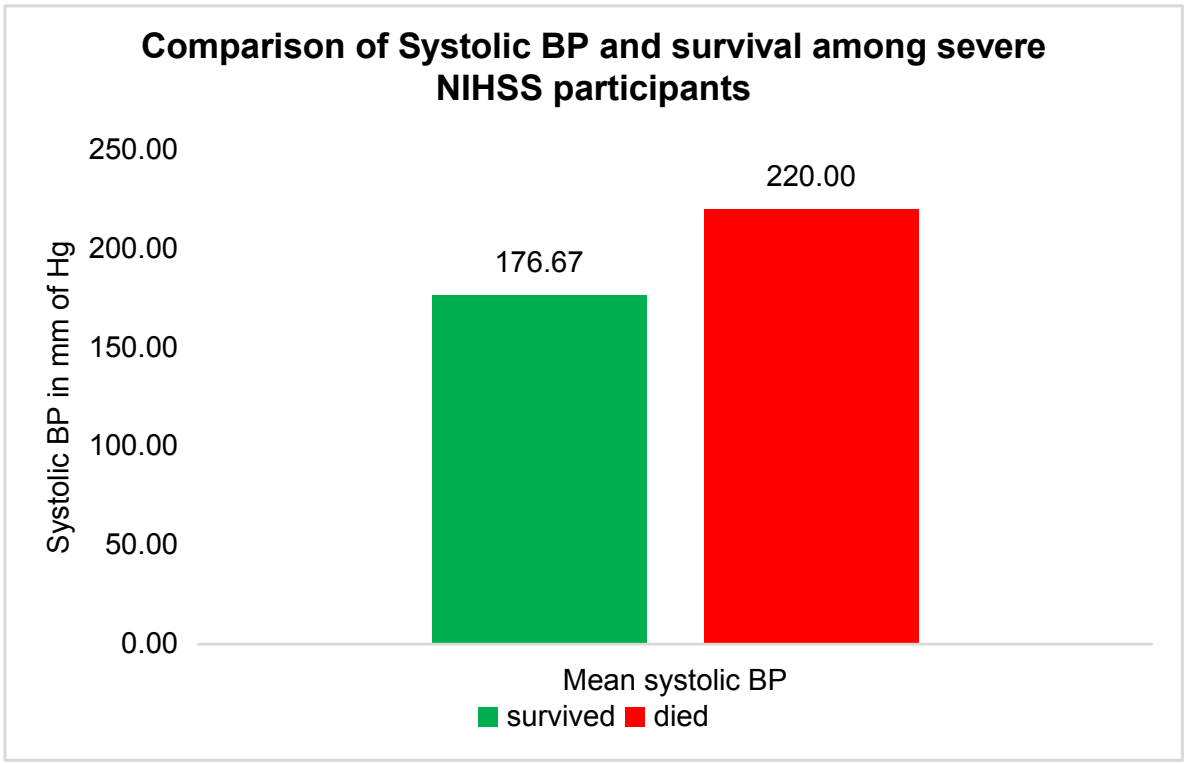
Comparison of Systolic BP and survival among severe NIHSS participants:

Table 20: Comparison of mean Systolic BP and survival among severe NIHSS participants (n=7)

Systolic BP (in mm of Hg)	Survived	Died	T test value	P value
Mean ± SD	176.67 ± 24.22	220 ± 25.40	1.656	0.159

Among the participants with severe NIHSS on admission, mean systolic BP of those who survived was 176.67 ± 24.22 and mean systolic BP of those who died was 220 ± 25.40 mm of Hg. This difference was not statistically significant by independent t test.

Figure 35: Comparison of mean Systolic BP and survival among severe NIHSS participants (n=7)



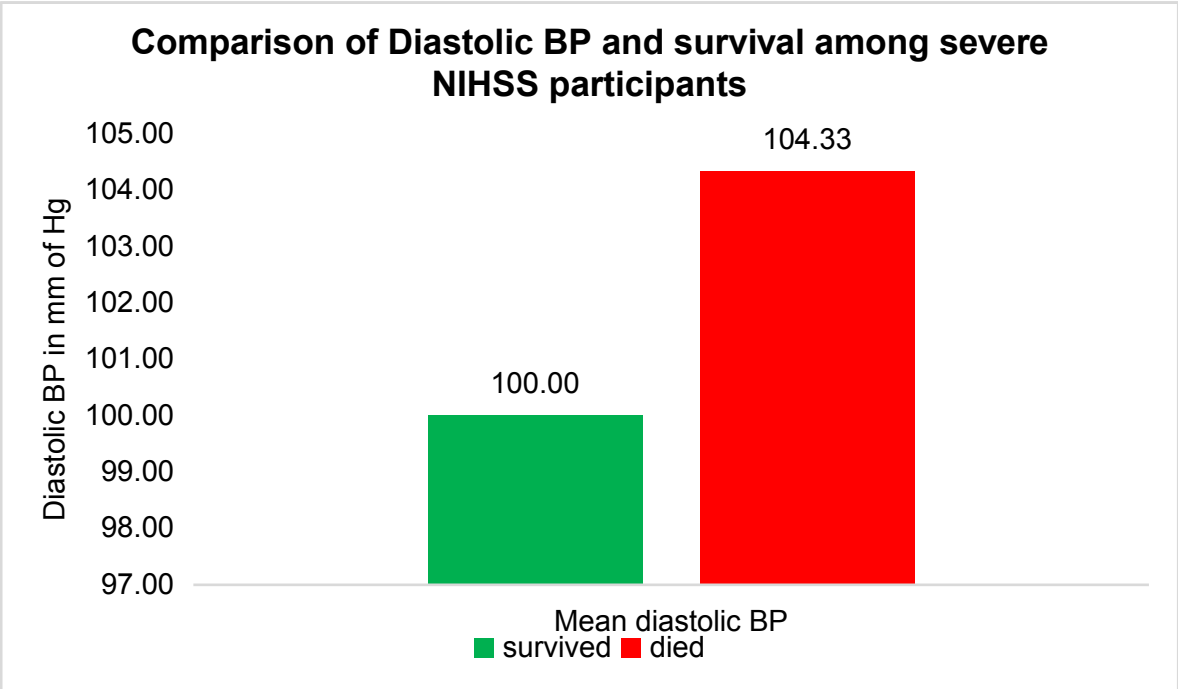
Comparison of Diastolic BP and survival among severe NIHSS participants:

Table 21: Comparison of mean Diastolic BP and survival among severe NIHSS participants (n=7)

Diastolic BP (in mm of Hg)	Survived	Died	T test value	P value
Mean ± SD	100.00 ± 9.25	104.33 ± 8.16	0.378	0.721

Among the participants with severe NIHSS on admission, mean diastolic BP of those who survived was 100.00 ± 9.25 and mean diastolic BP of those who died was 104.33 ± 8.16 mm of Hg. This difference was not statistically significant by independent t test.

Figure 36: Comparison of mean Diastolic BP and survival among severe NIHSS participants (n=7)



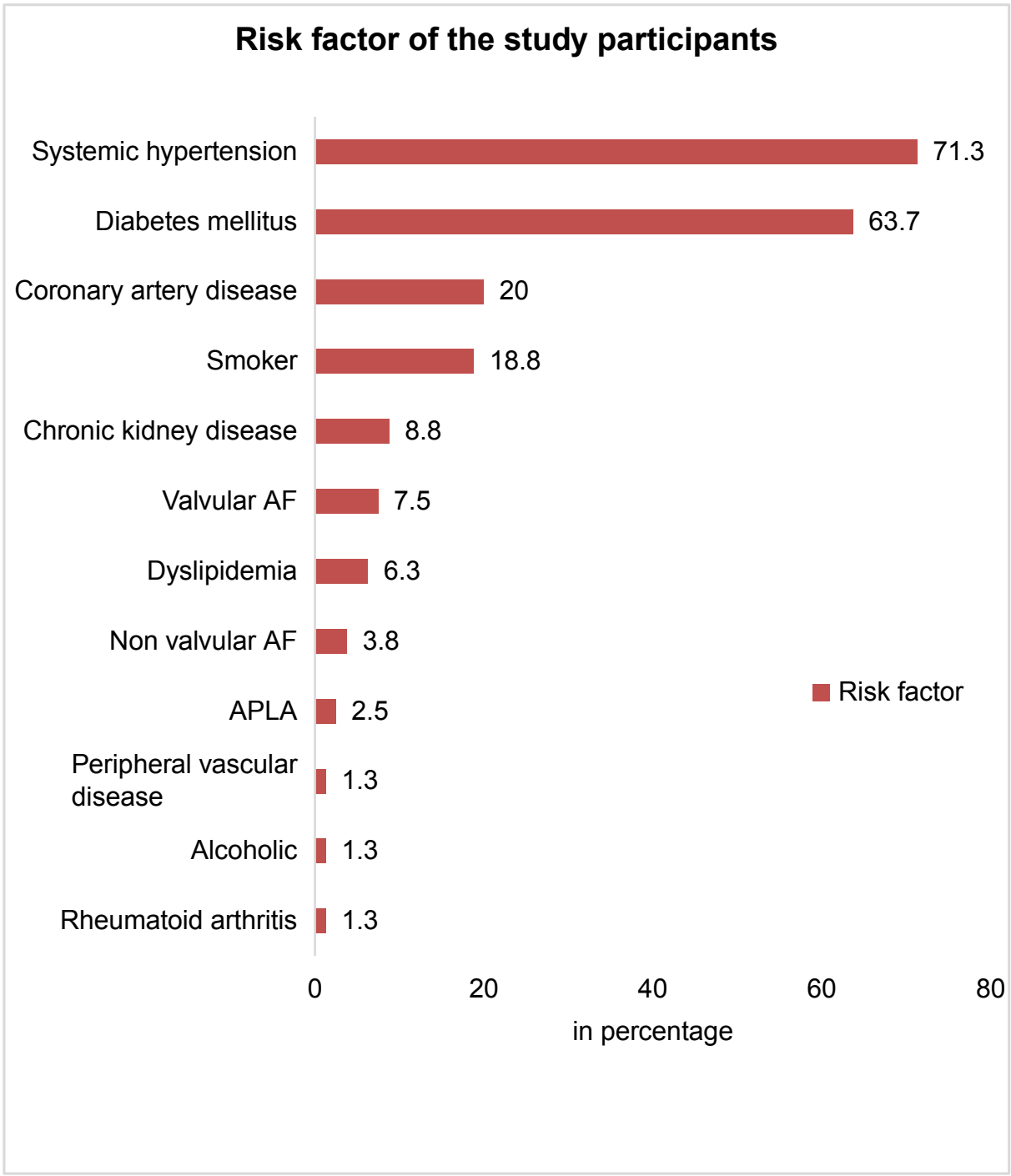
Risk factor of the study participants:

Table 22: Risk factor of the study participants (n=80)

Risk factor	Frequency	Percentage
Systemic hypertension	57	71.3
Diabetes mellitus	51	63.7
Coronary artery disease	16	20
Smoker	15	18.8
Chronic kidney disease	7	8.8
Valvular AF	6	7.5
Dyslipidemia	5	6.3
Non valvular AF	3	3.8
APLA	2	2.5
Rheumatoid arthritis	1	1.3
Alcoholic	1	1.3
Peripheral vascular disease	1	1.3

Among the study participants, 71.3% had Systemic hypertension, 63.7% had Diabetes mellitus, 20% had coronary artery disease, 18.8% were Smokers, 8.8% had chronic kidney disease, 7.5% had Valvular AF, 6.3% had Dyslipidemia, 3.8% had Non valvular AF, 2.5% had APLA, 1.3% had Rheumatoid arthritis, 1.3% were Alcoholic and 1.3% had Peripheral vascular disease

Figure 37: Risk factor of the study participants (n=80)



RESULTS

- This study was conducted among 80 participants.
- Among the study participants, 6.3% were less than 40 years of age, 17.5% were from 41 to 50 years, 38.8% were from 51 to 60 years, 25% were from 61 to 70 years and 12.5% were from 71 to 80 years of age.
- Among the study participants, 51 were males and 29 were females.
- The mean systolic blood pressure of participants who had severe NIHSS on admission was 182.86 ± 27.51, who had moderate NIHSS on admission was 152.65 ± 18.57 and who had mild NIHSS on admission was 141.25 ± 18.01 (mm of Hg). This difference was statistically significant by ANOVA test.
- The mean diastolic blood pressure of participants who had severe NIHSS on admission was 102.86 ± 7.55, who had moderate NIHSS on admission was 90.20 ± 10.89 and who had mild NIHSS on admission was 83.75 ± 10.13 (mm of Hg). This difference was statistically significant by ANOVA test.
- Thrombosis was the major type of infarction found in 67 participants, followed by embolism in 9 participants and systemic hypoperfusion in 4 participants.
- The mean duration of hospitalization of participants who had severe NIHSS on admission was 4.14 ± 3.62, who had moderate NIHSS on admission was 8.76 ± 2.98and who had mild NIHSS on admission was 6.88 ± 1.91 (in days). This difference was statistically significant by ANOVA test.

- The mean Neutrophil-Lymphocyte Ratio of participants who had severe NIHSS on admission was 10.80 ± 3.05, who had moderate NIHSS on admission was 5.00 ± 1.99 and who had mild NIHSS on admission was 3.12 ± 0.95. This difference was statistically significant by ANOVA test.
- The mean Platelet lymphocyte ratio of participants who had severe NIHSS on admission was 401.68 ± 168.58, who had moderate NIHSS on admission was 162.09 ± 58.61 and who had mild NIHSS on admission was 118.88 ± 23.25. This difference was statistically significant by ANOVA test.
- Among the study participants, 23.8% died and 76.3% survived. In participants who had severe NIHSS during admission, 85.7% participants died and 14.3% survived. This difference was statistically significant by Fischer exact test.
- In participants who had severe NIHSS grade on admission, the mean NIHSS score during admission was 18.29 ± 2.92 and mean NIHSS score on day 2 was 24.43 ± 3.55. This increase was statistically significant by paired t test.
- Among the study participants, 71.3% had Systemic hypertension, 63.7% had Diabetes mellitus, 20% had coronary artery disease, 18.8% were Smokers, 8.8% had chronic kidney disease, 7.5% had Valvular AF, 6.3% had Dyslipidemia, 3.8% had Non valvular AF, 2.5% had APLA, 1.3% had Rheumatoid arthritis, 1.3% were Alcoholic and 1.3% had Peripheral vascular disease
- In participants who had severe NIHSS grade on admission, 85.7% had systemic hypertension and 14.3% did not have hypertension.
- In participants who had severe NIHSS grade on admission, 70.8% had diabetes mellitus and 29.2% did not have diabetes.

- In participants who had severe NIHSS grade on admission, 100% did not have coronary artery disease,chronic kidney disease,rheumatoid arthritis, valvular AF, nonvalvular AF, dyslipidemia, APLA, peripheral vascular disease.
- In participants who had severe NIHSS grade on admission, 14.3% were smokers and 85.7% were nonsmokers.
- In participants who had severe NIHSS grade on admission, 14.3%% were alcoholic and 85.7% were nonalcoholic.
- Among participants with mild NIHSS on admission, ST depression was found in 8 participants, Normal sinus rhythm was in 6 participants, T wave inversion in lead V5, V6, I, aVL was in 6 participants, Atrial fibrillation was in 5 participants, Left axis deviation was in 2 participants and aVR ST elevation with diffuse ST depression was in 2 participants.
- Among participants with moderate NIHSS on admission, 12 participants had ST depression in V5, V6, I, aVL, 10 had T wave inversion in lead V5, V6, I, aVL, 9 had T wave inversion in lead V1-V6, 6 had Left axis deviation, 4 had LBBB, 3 had aVR ST elevation with diffuse ST depression, 3 had Normal sinus rhythm, 2 had Left axis deviation, 2 had Sinus tachycardia, 1 had RBBB, 1 had QT prolongation, 1 had Sinus bradycardia and 1 had Atrial fibrillation
- Among participants with severe NIHSS on admission, 4 had ST depression, 4 had QT prolongation, 3 had T wave inversion in lead V1-V6, 2 had T wave inversion in lead V5, V6, 2 had Left axis deviation, 2 had T wave inversion in lead V5, V6, I, aVL and 1 had LBBB.

- Among participants with severe NIHSS on admission, 1 survived in less than 40 years and 1 died in less than 40 years of age. 1 died in 41 to 50 years, 1 died in 51 to 60 years and 3 died in 61 to 70 years of age. This difference was not statistically significant by Fischer exact test.
- Among the participants with severe NIHSS on admission, mean systolic BP of those who survived was 176.67 ± 24.22 and mean systolic BP of those who died was 220 ± 25.40 mm of Hg. This difference was not statistically significant by independent t test.
- Among the participants with severe NIHSS on admission, mean diastolic BP of those who survived was 100.00 ± 9.25 and mean diastolic BP of those who died was 104.33 ± 8.16 mm of Hg. This difference was not statistically significant by independent t test.

DISCUSSION

This study was done with the purpose of predicting the mortality in patients who presents with acute ischemic stroke for the first time, by using the routine hematological indices and ECG.

The mean age of our study group was 57.39±13.86 years with the maximum cases in the age range of 61 to 70 years. This is in accordance with various other studies done by Grau et al, Aiyar et al and Naik M, Rauniyar, Sharma U.K et al[38].

Our study had male preponderance with male to female ratio of 2.1 :1 which was similar to all other studies done by Aiyar et al, Kay Sin Tan et al and R P Eapen et al[39-40].

In our study hypertension was the most common risk factor detected in 71% of the patients, which is similar to study conducted by Lok U et al with prevalence percentage of hypertension as 74%. Diabetes (63%) is the second prevalent cause which were in lines with studies conducted by Lok U et al, George J et al and Maydadomac F et al[41-42].followed by coronary artery disease (20%), smoking (18%), chronic kidney disease (8.8%), hyperlipidemia (6%), and alcoholism (1.3%).

Stroke stimulate the generation of neutrophils in the bone marrow and can lead to lymphopenia in a variety of ways. So, relative lymphopenia and neutrophilia can develop, resulting in an increased NLR. In our study the mean Neutrophil-Lymphocyte Ratio of participants who had severe NIHSS on admission was 10.80 ± 3.05, who had

moderate NIHSS on admission was 5.00 ± 1.99 and who had mild NIHSS on admission was 3.12 ± 0.95. There was statistically significant correlation was found between NLR and NIHSS score at the time of admission and value of NLR increased proportionately with the increasing NIHSS score.(P=<0.001)

Christensen et al and Balestrino et al[43-44] found that higher leucocyte and neutrophil lymphocyte ratio are observed during the acute period of stroke and these values are correlated with poor prognosis. Also previous study suggested that neutrophil lymphocyte ratio increases within 12 hour after onset of stroke and reaches peak value on the second day.

In our study the mean Platelet lymphocyte ratio of participants who had severe NIHSS on admission was 401.68 ± 168.58, who had moderate NIHSS on admission was 162.09 ± 58.61 and who had mild NIHSS on admission was 118.88 ± 23.25. There was statistically significant correlation found between PLR and NIHSS score at the time of admission. The value of PLR increased proportionately with the increasing NIHSS score .(p=<0.001). Similar to the studies done by Andres Perez et al,15 Pei-Hsun Sung et al16 and Xu J-H et al[20]. in which there was a positive correlation between PLR and NIHSS score at the time of admission also higher PLR had poor outcome as compared to patients with lower PLR values in acute ischemic stroke.

In our study participants who had severe NIHSS grade on admission, the mean NIHSS score during admission was 18.29 ± 2.92 and mean NIHSS score on discharge was 24.43 ± 3.55. This increase was statistically significant by paired t test. p=(<0.001)

Also in participants who had moderate NIHSS grade on admission, the mean NIHSS score during admission was 8.14 ± 2.69 and mean NIHSS score on day 2 was 10.10 ± 6.59. This increase was statistically significant by paired t test. P=(<0.006).

In our study participants, 23.8% died and 76.3% survived. In participants who had severe NIHSS during admission, 85.7% participants died and 14.3% survived. In participants who had moderate NIHSS during admission, 24.5% participants died and 75.5% survived. In participants who had mild NIHSS during admission, 4.2% of participants died and 95.8% survived. This difference was statistically significant by Fischer exact test. This study similar to Jeng et al done in acute ischemic stroke patients higher NIHSS score is correlated to increased hospital mortality.

In our study participants, who had severe NIHSS during admission, ST depression (57%), QT prolongation (57%), T wave inversion (42%). A large scale study done by Goldstein where it was seen in 32% of cases. T-wave inversion (15%), ST-segment depression (13%), and U-wave (28%). A similar study was done by Familoni et al., in 2006 where QTc prolongation was seen in 28% of the cases, T wave inversion in 21.8%, ST segment depression in 29.7%, U wave in 9.3%, and arrhythmia in 34.4% of the cases in study group. This conforms to the previous studies of Goldstein and Bozluolcay et al[45-46]., where ECG changes were demonstrated in 92 and 62.1% of patients, respectively.

Our findings that NLR and PLR correlated with clinical outcomes in AIS patients were consistent with previous studies, supporting the importance of NLR and PLR in predicting the pathological course of AIS.The different predictability of NLR and PLR

for AIS in our study broadened the options for manageable biomarkers to quench risk factors at the early stage of stroke.

CONCLUSION

From this study we conclude that;

1. High levels of NLR and PLR were associated with the highest risk of unfavourable functional outcomes.
2. Severe grade of National institute of health stroke scale is associated with statistically significant poor outcome.
3. High SBP and DBP are associated with increased risk of In-hospital mortality.
4. ST-T changes and QT prolongation in ECG had higher mortality and poor outcome.

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PROFORMA

A STUDY ON CLINICAL SIGNIFICANCE OF HEMATOLOGICAL INDICES, ELECTROCARDIOGRAPHIC CHANGES BY USING NATIONAL INSTITUTE OF HEALTH STROKE SCORE (NIHSS) IN ACUTE ISCHEMIC STROKE IN TERTIARY CARE CENTRE

I) DEMOGRAPHICS DATE: / /

SERIAL NO : IP. NO.

NAME : AGE: SEX:

ADDRESS :

OCCUPATION : INCOME :

II) STROKE PATHOLOGY

DATE AND TIME OF STROKE :

DATE OF ADMISSION :

REASON FOR DELAY :

ACTIVITY AT THE TIME OF STROKE ONSET :

MODE OF ONSET AND STROKE BEHAVIOUR:

1)SUDDEN WITH MAXIMAL DEFICIT

2)PROGRESSIVE

3)FLUCTUATING

4) REMISSION

LOCATION : 1) RT. HEMISPHERE

2) LT.HEMISPHERE

3) BILATERAL

4) BRAIN STEM

5) CEREBELLAR

6) OTHERS.

METHOD OF STROKE }

DIAGNOSIS } CLINICAL CRITERIA/CT/MRI

CT SCAN FINDING:

INFARCT : Y/N

LOCATION : AC/MC/PC

WATER SHED/GLOBAL/LACUNAR/OTHERS

AGE OF INFARCT : ACUTE/SUB ACUTE/OLD

TIME BETWEEN STROKE ON SET AND CT BRAIN :

PAST HISTORY OF STROKE : Y/N ANY

PERSISTING NEUROLOGICAL ILLNESS : Y/N

NIHSS SCORE ON ADMISSION AND ON 7TH DAY:

III) STROKE RISK FACTORS

SHT Y/N

DM Y/N

SMOKING Y/N

ALCOHOLISM Y/N

HIGH CHOLESSTEROL Y/N

IHD Y/N

RHD Y/N

AF Y/N

CAROTID STENOSIS Y/N

DISCHARGE STATUS : DHI /DHA /AH /EXP

DISCHARGE DATE

DURATION OF HOSPITALISATION

IV) ACUTE STROKE SYMPTOMS AND SIGNS

SYMPTOMS : HEADACHE

VERTIGO

VOMITING

GAIT DISTURBANCE

CONVULSIONS

SPEECH DEFICIT

SIGNS: HEMIANOPIA

DIPLOPIA

MOTOR SYSTEM : PARESIS AT ANY SITE PARESIS OF ARMS : Y/N R/L/B

PARESI OF LEGS : Y/N R/L/B PARESIS OF FACE : Y/N R/L/B

FIRST INVOLVED : FACE / ARMS / LEGS

NO SUCH ORDER : Y/N

SENSORY DEFICIT : Y/N

CEREBELLAR SIGNS : Y/N

VITALS :

PULSE RATE :

BLOOD PRESSURE :

RESPIRATORY RATE :

SPO2 :

TEMPERATURE :

INVESTIGATION:

HEMATOLOGICAL ON DAY 1 ON DAY 7

NLR

PLR

BIOCHEMICAL

SERUM ALBUMIN

BUN/CREATININE RATIO

LIPID PROFILE

ECG CHANGES

CHEST X RAY FINDINGS :

DATE OF ADMISSION NIHSS SCORE :

DATE OF DISCHARGE NIHSS SCORE :

ETHICAL COMMITTEE

INSTITUTIONAL ETHICS COMMITTEE
MADRAS MEDICAL COLLEGE, CHENNAI 600 003

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CERTIFICATE OF APPROVAL

To
Dr.SAKTHIVEL S,
MD Internal Medicine Post Graduate student,
Institute of Internal Medicine,
Madras Medical College,
Chennai-600 003.

Dear Dr. SAKTHIVEL S,

The Institutional Ethics Committee has considered your request and approved your study titled **"A STUDY ON CLINICAL SIGNIFICANCE OF HEMATOLOGICAL INDICES, ELECTRO CARDIOGRAPHIC CHANGES BY USING NATIONAL INSTITUTE OF HEALTH STROKE SCORE (NIHSS) IN ACUTE ISCHEMIC STROKE IN TERTIARY CARE CENTRE"- NO.10062022.** The following members of Ethics Committee were present in the meeting held on **15.06.2022** conducted at Madras Medical College, Chennai 3.

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

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

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

Member Secretary – Ethics Committee

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

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ANTI PLAGIARISM CERTIFICATE

This is to certify that the dissertation work titled "A STUDY ON CLINICAL SIGNIFICANCE OF HEMATOLOGICAL INDICES, ELECTRO CARDIOGRAPHIC CHANGES BY USING NATIONAL INSTITUTE OF HEALTH STROKE SCORE (NIHSS) IN ACUTE ISCHEMIC STROKE IN TERTIARY CARE CENTRE " of the candidate Dr. SAKTHIVEL. S with Registration Number 200120100527 for the award of M.D. degree in the branch of GENERAL MEDICINE is original. I personally verified the urkund.com website for plagiarism Check. I found that the uploaded thesis file contained Introduction to Conclusion pages and result showed 1.8 percentage of plagiarism.

Guide & Supervisor sign with Seal

PROFESSOR
INSTITUTE OF INTERNAL MEDICINE
MMC & RGGGH, CHENNAI-600 003

PATIENT CONSENT FORM

Study Detail : A STUDY ON CLINICAL SIGNIFICANCE OF HEMATOLOGICAL INDICES, ELECTRO CARDIOGRAPHIC CHANGES BY USING NATIONAL INSTITUTE OF HEALTH STROKE SCORE (NIHSS) IN ACUTE ISCHEMIC STROKE IN TERTIARY CARE CENTRE.

Study Centre : Rajiv Gandhi Government General Hospital, Chennai.

Patient's Name :

Patient's Age :

Identification Number :

Patient may check (√) these boxes

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

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

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

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

o I hereby consent to participate in this study.

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

Signature of investigator Signature/Thumb impression of participant

Patient name and address

நோயாளி ஒப்புதல் படிவம்

ஆய்வு விவரம் Ć Ď ĘĚFEĜ ĘĖ ĎĚĘĖĘĎĎĚ ĘĘĖĖĘĚĘĎĎĖĎĖÉ ĘĚ ĚĚËĎĚĘĚĘĖĘĘĎĎĚ ĘĖĖĘĎÉĘÅ ÉĚĚÉĎĚĚĘ ĎĎĚĖĖĘĖĖĚĎÈĚĘĎ ĎĚĎĖĖĚĘ ÐĜ FĘĘĖÊ ĖĎĚĚĘĖĖĎĚ ĘĖĖĘĚĚĚFĚĚÉ ĘĚ ĚÉĎĚĚĚÉ ĘĚĖĘĚÉ ĘĎĘĖĚÉ ÂĖĘĚĚĘĘÁ ĘĖĖ ĎĎFĚÉ ĘĘĎĚÉĚËĘĎ ĘĚĖĘĚÉ ĘĖ ĚÉĖĚĘĚĎĖĜ ĎĎĖĚÉ ĎÉĖĚĖÉÄ

ஆய்வு மையம்Ć ராஜீவ் காந்தி அரசு பொது மருத்துவமனைÅ சென்னைÄ

நோயாளியின் பெயர்Ć

நோயாளியின் வயதுĆ

அடையாள எண் :

நோயாளி இந்த படிவத்தை ÂYÁ செய்யலாம்ĆÃ

o மேற்கண்ட ஆய்விற்கான நடைமுறையின் நோக்கத்தை நான் புரிந்து கொண்டேன் என்பதை உறுதிப்படுத்துகிறேனÄ கேள்வி கேட்க எனக்கு வாய்ப்பு உள்ளதுÅ எனது முழு திருப்திக்கும் எனது எல்லா கேள்விகளுக்கும் சந்தேகங்களுக்கும் பதில் அளிக்கப்பட்டுள்ளதுÄ

o ஆய்வில் நான் பங்கேற்பது தன்னார்வமானது என்பதையும்Å எனது சட்ட உரிமைகள் பாதிக்கப்படாமல்Å காரணமின்றி எந்த நேரத்திலும் திரும்பப் பெற எனக்கு சுதந்திரம் உள்ளது என்பதையும் நான் புரிந்துகொள்கிறேனÄ

o மருத்துவ ஆய்வின் ஸ்பான்சர்Å ஸ்பான்சர் சார்பாக பணிபுரியும் மற்றவர்கள்Å நெறிமுறைக் குழு மற்றும் ஒழுங்குமுறை அதிகாரிகள் எனது சுகாதார பதிவுகளைப் பார்க்க எனது அனுமதி தேவையில்லை என்பதை நான் புரிந்துகொள்கிறேன்Å தற்போதைய ஆய்வு மற்றும் மேற்கொண்டுள்ள எந்தவொரு ஆராய்ச்சியையும் பொறுத்தவரை இது தொடர்பாகÅ நான் ஆய்வில் இருந்து விலகினாலும் இந்த அணுகலை ஒப்புக்கொள்கிறேனÄ எவ்வாறாயினும்Å சட்டத்தின் கீழ் தேவைப்படாவிட்டால்Å மூன்றாம் தரப்பினருக்கு வெளியிடப்பட்ட அல்லது

வளியிடப்பட்ட எந்தவொரு தகவலிலும் எனது அடையாளம் வளிப்படுத்தப்படாது என்பதை நான் புரிந்துகொள்கிறேன்Ä இந்த ஆய்வில் எழும் எந்தவொரு தரவு அல்லது முடிவுகளின் பயன்பாட்டை கட்டுப்படுத்த வண்டாம் என்று நான் ஒப்புக்கொள்கிறேன்Ä

o மேற்கண்ட ஆய்வில் பங்கேற்கவும்Å ஆய்வின் போது கொடுக்கப்பட்ட அறிவுறுத்தல்களுக்கு இணங்கவும்Å ஆய்வுக் குழுவுடன் உண்மையுடன் ஒத்துழைக்கவும்Å எனது உடல்நலம் அல்லது நல்வாழ்வில் ஏதனும் சரிவு ஏற்பட்டால் அல்லது எதிர்பாராத அல்லது ஏதனும் ஏற்பட்டால் உடனடியாக ஆய்வு ஊழியர்களுக்கு அறிவிக்கவும் ஒப்புக்கொள்கிறேன்Ä அசாதாரண அறிகுறிகள்Ä

o இந்த ஆய்வில் பங்கேற்க நான் இதன்மூலம் ஒப்புக்கொள்கிறேன்Ä

o தவைக்கேற்ப விரிவான மருத்துவ பரிசோதனை மற்றும் இரத்த விசாரணைகளை மேற்கொள்ள நான் இதன்மூலம் அனுமதி அளிக்கிறேன்Ä

புலனாய்வாளரின் கையொப்பம் பங்கேற்பாளரின் கையொப்பம் A

கட்டவிரல் எண்ணம்

நோயாளியின் பெயர் மற்றும் முகவரி

MASTER

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S.NO	1. Age	2. Gender	3. SBP	4. DBP	5. Risk factor	6. Types of infarction	7. CT BRAIN FINDINGS	8. NIHSS at DOA	9. NIHSS garding on Admission	10. NIHSS at day 7	11. Duration of hospital stay(days)	12. NLR	13. PLR	14. ECG FINDINGS	15. In hospital mortality
1	70	Female	140	90	NIL	Thrombosis	Left parietal infarct	4	Mild	4	8	1.51	53.3	ST depression in V5, V6, I, aVL	No
2	54	Male	180	110	Systemic hypertension	Thrombosis	Right parieto temporal infarction	6	Moderate	6	8	1.51	110.8	ST depression in V5, V6, I, aVL	No
3	59	Male	160	90	Systemic hypertension	Thrombosis	Bilateral occipital infarction	6	Moderate	8	10	3.3	123.7	Normal sinus rhythm	No
4	64	Male	150	100	Systemic hypertension | Coronary artery disease | Diabetes mellitus	Thrombosis	Left FTP infarction	15	Moderate	22	4	8.9	314.5	aVR ST elevation with diffuse ST depression | T wave inverion in lead V1-V6 | QT prolongation	Yes
5	65	Female	160	110	Diabetes mellitus	Thrombosis	Left FTP infarction	16	Moderate - severe	22	3	9.5	352.5	Left axis deviation | ST depression & T wave inversion in lead V5, V6, I, aVL | QT prolongation	Yes
6	54	Male	140	90	Systemic hypertension | Diabetes mellitus	Thrombosis	Right midbrain infarction	4	Mild	4	8	2.2	147.7	ST depression & T wave inversion in lead V5, V6	No
7	32	Female	110	70	Valvular AF	Embolism	Right frontal infarction	4	Mild	4	6	4.1	121.2	Atrial fibrillation	No
8	64	Male	160	90	Systemic hypertension | Diabetes mellitus	Thrombosis	Left pontine infarction	7	Moderate	8	8	2.5	94.3	ST depression & T wave inversion in lead V5, V6, I, aVL	No
9	62	Male	180	100	Systemic hypertension | Diabetes mellitus	Thrombosis	Left cerebellar infarction	16	Moderate- severe	24	2	22.2	980	ST depression & T wave inversion in lead V5, V6, I, aVL | T wave inverion in lead V1-V6 | QT prolongation	Yes
10	52	Male	130	80	Peripheral vascular disease | Smoker	Thrombosis	Right parietal & centrum semiovale infarction	7	Moderate	6	6	6.7	152.0	Normal sinus rhythm	No
11	51	Male	160	90	Systemic hypertension | Smoker | Diabetes mellitus	Thrombosis	Left corona radiata and occipital infarction	8	Moderate	7	8	3.1	117.5	Normal sinus rhythm	No
12	56	Male	180	110	Systemic hypertension	Thrombosis	Right lateral medulla infarction	18	Moderate to severe	24	5	8.5	242.5	T wave inverion in lead V1-V6 | QT prolongation	Yes
13	73	Male	150	90	Systemic hypertension | Diabetes mellitus	Thrombosis	Left ganglio capsular infarction	4	Mild	4	5	4.6	140	ST depression & T wave inversion in lead V5, V6, I, aVL	No
14	45	Male	190	110	Systemic hypertension | Smoker | Diabetes mellitus	Thrombosis	Right FTP infarction	20	Moderate- severe	26	2	9.2	221.9	T wave inverion in lead V1-V6 | QT prolongation	Yes
15	74	Male	140	90	Diabetes mellitus	Thrombosis	Bilateral parieto occipital infarction	5	Moderate	5	6	3.27	178	Normal sinus rhythm	No
16	29	Male	220	100	Systemic hypertension	Thrombosis	Left parieto occipital infarction	16	Moderate - severe	18	12	8.1	229	ST depression & T wave inversion in lead V5, V6	No
17	41	Male	180	100	Systemic hypertension	Thrombosis	Right GC infarction	4	Mild	4	6	3.2	128.2	ST depression & T wave inversion in lead V5, V6, I, aVL	No
18	58	Female	160	90	Systemic hypertension | Diabetes mellitus	Thrombosis	Left pontine infarction	8	Moderate	14	5	8.6	245	T wave inverion in lead V1-V6 | Sinus bradycardia	Yes
19	52	Female	140	80	Valvular AF	Embolism	Left GC infarction	4	Mild	4	6	2.5	112.4	Atrial fibrillation	No
20	70	Male	150	90	Systemic hypertension | Dyslipidemia | Diabetes mellitus	Thrombosis	Left occipital infarction	6	Moderate	6	8	3.4	134.5	ST depression & T wave inversion in lead V5, V6, I, aVL	No
21	48	Male	150	90	Systemic hypertension | Diabetes mellitus	Thrombosis	Left cerebellar infarction	6	Moderate	6	8	4.5	147.6	ST depression & T wave inversion in lead V5, V6	No
22	64	Male	160	100	Systemic hypertension | Diabetes mellitus	Thrombosis	Right FTP infarction	8	Moderate	12	10	6.8	175.2	LBBB	No

S.NO	1. Age	2. Gender	3. SBP	4. DBP	5. Risk factor	6. Types of infarction	7. CT BRAIN FINDINGS	8. NIHSS at DOA	9. NIHSS garding on Admission	10. NIHSS at day 7	11. Duration of hospital stay(days)	12. NLR	13. PLR	14. ECG FINDINGS	15. In hospital mortality
23	80	Male	120	70	Systemic hypertension | Coronary artery disease | Non valvular AF | Diabetes mellitus	Embolism	Right GC infarction	4	Mild	4	6	3.1	117.4	Atrial fibrillation	No
24	53	Male	150	100	Systemic hypertension | Smoker	Thrombosis	Left FTP infarction	8	Moderate	7	10	6	152.4	ST depression & T wave inversion in lead V5, V6, I, aVL	No
25	52	Male	140	80	Coronary artery disease | Diabetes mellitus	Thrombosis	Right GC infarction	4	Mild	4	6	3.6	118.4	Normal sinus rhythm	No
26	64	Male	140	70	Diabetes mellitus	Thrombosis	Right occipital infarction	4	Mild	3	6	4	136.4	ST depression & T wave inversion in lead V5, V6, I, aVL	No
27	58	Male	170	110	Systemic hypertension	Thrombosis	Right GC infarction	4	Mild	4	6	2.6	117.4	ST depression & T wave inversion in lead V5, V6	No
28	62	Female	140	90	Systemic hypertension | Diabetes mellitus	Thrombosis	Right FTP infarction	18	Moderate-severe	26	3	8.7	328.4	Left axis deviation | LBBB	Yes
29	72	Male	140	80	Systemic hypertension | Diabetes mellitus	Thrombosis	Multiple watershed infarction	12	Moderate	8	8	3.2	150.2	aVR ST elevation with diffuse ST depression	No
30	42	Female	140	80	Systemic hypertension | Valvular AF	Embolism	Right centrum semiovale infarction	3	Mild	3	6	2.6	104.8	Atrial fibrillation	No
31	56	Female	140	90	Systemic hypertension | Diabetes mellitus	Thrombosis	Left midbrain infarction	6	Moderate	6	8	4.6	142.8	ST depression in V5, V6, I, aVL | T wave inversion in lead V5, V6, I, aVL,	No
32	65	Female	140	80	Diabetes mellitus	Thrombosis	Right GC infarction	3	Mild	3	5	3.6	112.4	Normal sinus rhythm	No
33	56	Female	150	80	Systemic hypertension | Chronic kidney disease	Thrombosis	Left FTP infarction	14	Moderate	22	10	7.6	182	Left axis deviation | ST depression & T wave inversion in lead V5, V6, I, aVL	Yes
34	70	Male	150	90	Systemic hypertension | Diabetes mellitus	Thrombosis	Right FTP infarction	7	Moderate	5	12	6.2	148	ST depression & T wave inversion in lead V5, V6, I, aVL	No
35	58	Male	130	80	Coronary artery disease | Diabetes mellitus	Thrombosis	Right GC infarction	4	Mild	4	7	2.6	108.2	Left axis deviation | aVR ST elevation with diffuse ST depression	No
36	62	Female	130	80	Diabetes mellitus	Thrombosis	Left occipital infarction	6	Moderate	6	8	4.8	127.2	Normal sinus rhythm	No
37	65	Female	150	80	Systemic hypertension | Diabetes mellitus	Thrombosis	Right GC infarction	4	Mild	No Answer	6	2.8	106.2	Normal sinus rhythm	No
38	76	Male	140	90	Systemic hypertension | Diabetes mellitus	Thrombosis	Bilateral occipital infarction	12	Moderate	No Answer	4	9.6	382.4	Left axis deviation | ST depression in V5, V6, I, aVL | T wave inversion in lead V5, V6, I, aVL, | T wave inverion in lead V1-V6	Yes
39	80	Male	140	70	Systemic hypertension | Diabetes mellitus	Thrombosis	Left parieto occipital infarction	8	Moderate	10	12	6.8	217.3	LBBB	No
40	68	Male	130	80	Diabetes mellitus	Thrombosis	Left GC infarction	6	Moderate	6	7	4.3	126.5	Normal sinus rhythm	No
41	52	Male	170	100	Systemic hypertension	Thrombosis	Right FTP infarction	10	Moderate	No Answer	4	6.6	152.4	ST depression & T wave inversion in lead V5, V6, I, aVL	Yes
42	55	Female	130	80	Valvular AF | Diabetes mellitus	Embolism	Left frontal infarction	7	Moderate	7	8	2.7	102.8	Atrial fibrillation	No
43	70	Male	130	80	Coronary artery disease | Diabetes mellitus	Thrombosis	Left FTP infarction	14	Moderate	18	16	7.8	168.4	aVR ST elevation with diffuse ST depression | T wave inverion in lead V1-V6	Yes

S NO	1. Age	2. Gender	3. SBP	4. DBP	5. Risk factor	6. Types of infarction	7. CT BRAIN FINDINGS	8. NIHSS at DOA	9. NIHSS garding on Admission	10. NIHSS at day 7	11. Duration of hospital stay(days)	12. NLR	13. PLR	14. ECG FINDINGS	15. In hospital mortality
44	76	Female	190	90	Systemic hypertension | Coronary artery disease | Chronic kidney disease | Diabetes mellitus	Thrombosis	Right FTP infarction	13	Moderate	No Answer	5	6.8	192	Left axis deviation | qRBBB | T wave inverion in lead V1-V6	Yes
45	67	Male	160	90	Systemic hypertension | Diabetes mellitus	Thrombosis	Left GC infarction	5	Moderate	4	7	4.8	115.6	ST depression & T wave inversion in lead V5, V6	No
46	72	Female	140	70	Coronary artery disease | Diabetes mellitus	Thrombosis	Right parieto occipital infarction	9	Moderate	12	10	6	182.4	LBBB | T wave inverion in lead V1-V6	Yes
47	44	Male	190	110	Systemic hypertension | Smoker	Thrombosis	Right FTP infarction	10	Moderate	8	12	5.6	156.6	ST depression & T wave inversion in lead V5, V6, I, aVL	No
48	51	Female	140	80	Diabetes mellitus	Thrombosis	Right GC infarction	4	Mild	4	7	3.6	136.2	Normal sinus rhythm	No
49	65	Male	160	100	Systemic hypertension | Smoker	Thrombosis	Right FTP infarction	9	Moderate	8	12	4.6	152.2	Left axis deviation | ST depression & T wave inversion in lead V5, V6, I, aVL	No
50	56	Male	130	80	Coronary artery disease | Non valvular AF	Embolism	Left FTP infarction	13	Moderate	12	14	4.8	134.6	Left axis deviation | LBBB | T wave inverion in lead V1-V6	No
51	48	Female	140	80	Diabetes mellitus	Thrombosis	Right fronto parietal infarction	6	Moderate	4	8	4.6	142.4	Normal sinus rhythm	No
52	53	Male	140	100	Systemic hypertension | Coronary artery disease | Diabetes mellitus	Thrombosis	Left pontine infarction	8	Moderate	9	10	3.2	136.2	Left axis deviation | ST depression & T wave inversion in lead V5, V6, I, aVL	No
53	56	Female	140	90	Systemic hypertension | Coronary artery disease | Diabetes mellitus	Thrombosis	Left FTP infarction	7	Moderate	6	10	3.2	128.4	ST depression & T wave inversion in lead V5, V6, I, aVL	No
54	51	Female	130	80	Systemic hypertension | Diabetes mellitus	Thrombosis	Left GC infarction	5	Moderate	4	8	2.9	113.4	Normal sinus rhythm	No
55	72	Male	160	100	Systemic hypertension | Chronic kidney disease | Diabetes mellitus	Thrombosis	Right FTP infarction	12	Moderate	No Answer	4	9.8	348.2	Sinus tachycardia | Left axis deviation | ST depression & T wave inversion in lead V5, V6, I, aVL	Yes
56	49	Male	190	110	Systemic hypertension | Chronic kidney disease	Thrombosis	Right FTP infarction	10	Moderate	12	15	5.2	168	Sinus tachycardia | ST depression & T wave inversion in lead V5, V6, I, aVL	No
57	67	Female	130	70	Non valvular AF	Embolism	Left FTP infarction	7	Moderate	5	8	3.4	122.4	Atrial fibrillation	No
58	56	Female	140	90	Systemic hypertension | Coronary artery disease | Diabetes mellitus	Thrombosis	Right lacunar infarct	3	Mild	3	7	2.6	98.4	ST depression & T wave inversion in lead V5, V6	No
59	38	Male	160	100	Systemic hypertension | Coronary artery disease | Chronic kidney disease	Thrombosis	Left FTP infarction	8	Moderate	7	14	6.2	150.4	LBBB | T wave inverion in lead V1-V6	No
60	42	Female	140	90	Systemic hypertension | Diabetes mellitus	Thrombosis	Right GC infarction	4	Mild	4	7	3.2	116.4	ST depression & T wave inversion in lead V5, V6	No

S NO	1 Age	2 Gender	3. SBP	4 DBP	5. Risk factor	6. Types of infarction	7. CT BRAIN FINDINGS	8. NIHSS at DOA	9. NIHSS garding on Admission	10. NIHSS at day 7	11. Duration of hospital stay(days)	12. NLR	13. PLR	14. ECG FINDINGS	15. In hospital mortality
61	58	Male	150	90	Systemic hypertension | Smoker | Diabetes mellitus	Thrombosis	Right occipital infarction	6	Moderate	5	8	3.4	128.8	Left axis deviation | T wave inversion in lead V5, V6, I, aVL,	No
62	32	Male	210	100	NIL	Thrombosis	Right FTP infarction	24	Severe	No Answer	2	9.4	457.5	ST depression & T wave inversion in lead V5, V6	Yes
63	54	Male	160	90	Smoker	Thrombosis	Left FTP infarction	7	Moderate	7	8	3.7	129.2	ST depression & T wave inversion in lead V5, V6	No
64	48	Male	170	100	Systemic hypertension | Smoker	Thrombosis	Left leteral medullary and pontine infarction	8	Moderate	No Answer	3	7.4	248.4	ST depression & T wave inversion in lead V5, V6, I, aVL	Yes
65	74	Male	140	80	Systemic hypertension | Dyslipidemia | Coronary artery disease | Smoker | Diabetes mellitus	Thrombosis	Multiple Lacunar infarction	4	Mild	4	8	2.4	104.6	Normal sinus rhythm | ST depression in V5, V6, I, aVL	No
66	47	Female	130	80	Systemic hypertension | Valvular AF	Embolism	Right GC infarction	5	Moderate	4	7	2.9	119.7	Atrial fibrillation	No
67	43	Female	150	100	Systemic hypertension | Chronic kidney disease	Thrombosis	Left FTP infarction	7	Moderate	7	9	3.0	125.4	ST depression & T wave inversion in lead V5, V6	No
68	60	Female	180	100	Systemic hypertension | Diabetes mellitus	Thrombosis	Right FTP infarction	12	Moderate	18	9	5.6	192	Normal sinus rhythm | Left axis deviation | LBBB | T wave inverion in lead V1-V6	Yes
69	50	Male	120	80	Systemic hypertension | Coronary artery disease | Diabetes mellitus	Systemic hypoperfusion	Left lacunar infarction	3	Mild	4	7	2.9	128.4	aVR ST elevation with diffuse ST depression	No
70	44	Female	140	80	NIL	Thrombosis	Right frontal infarction	3	Moderate	No Answer	6	2.4	106.8	Normal sinus rhythm	No
71	54	Male	160	90	Systemic hypertension | Smoker | Diabetes mellitus	Thrombosis	Left cerebellar infarction	3	Mild	12	7	6.2	178	ST depression & T wave inversion in lead V5, V6, I, aVL	Yes
72	58	Male	150	90	Dyslipidemia | Smoker | Diabetes mellitus	Thrombosis	Right parieto occipital infarction	7	Moderate	6	10	4.6	152.6	Normal sinus rhythm | ST depression & T wave inversion in lead V5, V6	No
73	63	Female	160	80	Systemic hypertension | Dyslipidemia | Diabetes mellitus	Systemic hypoperfusion	Left lacunar infarction	6	Moderate	5	8	3.4	142.7	ST depression & T wave inversion in lead V5, V6, I, aVL	No
74	57	Female	180	100	Systemic hypertension | Diabetes mellitus	Thrombosis	Right medullary infarction	4	Mild	6	15	3.5	148.6	T wave inversion in lead V5, V6, I, aVL,	No
75	38	Male	210	120	Systemic hypertension | Chronic kidney disease	Thrombosis	Left FTP infarction	9	Moderate	18	12	6.4	204.3	Left axis deviation | ST depression & T wave inversion in lead V5, V6, I, aVL	Yes
76	45	Male	160	80	Systemic hypertension | Smoker	Thrombosis	Left Thalamic infarct	5	Moderate	4	7	2.8	116.7	Normal sinus rhythm	No
77	55	Male	130	80	Valvular AF	Embolism	Right IC infarction	4	Mild	4	7	2.4	109.5	Atrial fibrillation	No
78	58	Male	110	70	Systemic hypertension | Coronary artery disease | Smoker | Diabetes mellitus	Systemic hypoperfusion	Left lacunar infarction	3	Mild	3	7	2.7	102.4	Left axis deviation | ST depression & T wave inversion in lead V5, V6	No
79	54	Female	140	90	Systemic hypertension | Dyslipidemia | Diabetes mellitus	Thrombosis	Right pontine infarction	6	Moderate	8	14	4.2	148.6	aVR ST elevation with diffuse ST depression | T wave inverion in lead V1-V6	No

S.NO	1. Age	2. Gender	3. SBP	4. DBP	5. Risk factor	6. Types of infarction	7. CT BRAIN FINDINGS	8. NIHSS at DOA	9. NIHSS garding on Admission	10. NIHSS at day 7	11. Duration of hospital stay(days)	12. NLR	13. PLR	14. ECG FINDINGS	15. In hospital mortality
80	70	Male	160	90	Systemic hypertension | Coronary artery disease | Smoker | Diabetes mellitus	Systemic hypoperfusion	Multiple watershed infarction	7	Moderate	5	9	3.9	145.8	Left axis deviation | ST depression & T wave inversion in lead V5, V6, I, aVL | T wave inverion in lead V1-V6	No