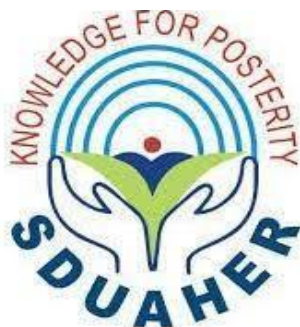


**AN OBSERVATIONAL STUDY OF TRIGLYCERIDE
GLUCOSE INDEX AS A PROGNOSTIC MARKER IN
ELDERLY HYPERTENSIVE PATIENTS WITH ACUTE
ISCHEMIC STROKE IN TERTIARY CARE CENTRE**

BY

DR. PERAM BALAKRISHNA



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

In partial fulfillment of the requirements for the degree of

DOCTOR OF MEDICINE

IN

GENERAL MEDICINE



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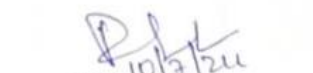
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ABSTRACT

Introduction: A primary cause of morbidity and death is ischemic cerebrovascular stroke. Hypertension, diabetes, and stroke are linked to atherosclerotic vessel. The triglyceride glucose index (T2DM) is a reliable marker for insulin resistance, linked to metabolic disorders like diabetes and cardiovascular diseases. However, its prognostic significance in elderly hypertensive patients with acute ischemic stroke needs further study.

Materials and Methods: The present prospective observational study took place in the tertiary care center of the tertiary care center. 81 elderly hypertensive and Research Center. After symptoms at 48 months from July 2021 to December 2022. Prior to the onset of the stroke, all of the patients had a confirmed diagnosis of hypertension.

Results and observations: The study included the prevalence of hypertension among elderly aged 71-80 years with a mean age of 74.5 years (SD 4.5). predominantly from urban areas (68%), predominantly Hindu (80%), Marathi (18%), and other religions (12%). The majority of subjects were of high socioeconomic status (65%), with a history of hypertension (70%), and no history of stroke (10%). The fasting triglyceride levels were 140-160 mg/dL, and the triglyceride glucose index was 1.5-1.8. The study found significant differences in blood pressure, stroke severity, poor functional outcome, neurological outcomes, and mortality rates among the groups based on the triglyceride glucose index. The incidence of stroke recurrence was high in group IV, poor functional outcome was high in group IV, neurological outcomes were high in group IV, and mortality was high in group IV.

Conclusion: The T2DM index is a risk factor for stroke mortality and recurrence in elderly hypertensive patients. However, further study is needed to confirm the association between stroke, death, and recurrence in hypertensive stroke patients. The

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PRIOR PERMISSION TO START OF STUDY

The Institutional Ethics Committee of Sri Devaraj Urs Medical College, Tamaka, Kolar has examined and unanimously approved the synopsis entitled "An observational study of triglyceride glucose index as a prognostic marker in elderly hypertensive patients with acute ischemic stroke, in a tertiary care centre" being investigated by Dr. Peram Bala krishna & Dr. Vidyasagar C.R in the Department of General Medicine at Sri Devaraj Urs Medical College, Tamaka, Kolar. **Permission is granted by the Ethics Committee to start the study.**

Sujatha.M.P.
Member Secretary

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ABBREVIATIONS

TyG	Triglyceride-glucose
DALY	Disability adjusted life years
APLA	Antiphospholipid Antibodies
CNS	Central Nervous System
SLE	Systemic Lupus Erythematosus
SHT	Subclinical Hypothyroidism
CADASIL	Cerebral autosomal dominant arteriopathy with subcortical infarcts and leucoencephalopathy
TIA	Transient ischemic attack
DNA	Deoxyribonucleic Acid
MRI	Magnetic Resonance Imaging
CT	Computed Tomography
ATP	Adenosine Triphosphate
NOS	Nitric Oxide Synthase
OCSP	The Oxford Community Stroke Project classification
TACI	Total anterior circulation infarct
PACI	Partial anterior circulation infarct

LACI	Lacunar infarct
POCI	Posterior circulation infarct
ACS	Anterior circulation stroke
ICA	Internal carotid artery
MCA	Middle cerebral artery
PCS	Posterior circulation stroke
TCD	Transcranial Doppler
SPECT	Single-photon emission computed tomography
CRP	C-reactive protein
IL-6	interleukin-6
TNF-alpha	Tumor necrosis factor-alpha
ROS	Reactive oxygen species
NO	Nitric oxide

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ABSTRACT

Introduction: A primary cause of morbidity and death is ischemic stroke, especially within elderly hypertensive patients. Insulin resistance, a risk factor, is linked to cerebrovascular events. The triglyceride-glucose index (TyG) is a reliable marker for insulin resistance, linked to metabolic disorders like diabetes and cardiovascular diseases. However, its prognostic significance in elderly hypertensive patients with acute ischemic stroke remains underexplored.

Materials and Methods: The present prospective observational study took place on 64 patients in the department of general medicine, RL Jalappa Hospital and Research Centre, Kolar, temporarily for 18 months from July 2022 to December 2023. Prior to the initiation of the study, Ethical and Research Committee clearance was obtained from Institutional Ethical Committee.

Results and observations: The study analyzed the prevalence of hypertensives among individuals aged 71-80 years, with a majority of subjects being males (54.69%), predominantly from urban areas (64.06%), predominantly Hindus (30.06%), Muslims (32.81%), Christians (21.87%), and other religions (6.25%). The majority of subjects were of high socioeconomic status (54.69%), with a history of hypertension (100%). The fasting triglyceride levels were 167.5 ± 15.1 mg/d, and the triglyceride glucose index was 8.9 ± 0.6 . The study found significant differences in blood pressure, stroke recurrence, poor functional outcome, neurological worsening, and mortality rates among the groups based on the triglyceride glucose index. The incidence of stroke recurrence was high in group IV, poor functional outcome was high in group III, neurological worsening was high in group IV, and mortality was high in group IV.

Conclusion: The TyG index, a risk factor for stroke mortality and recurrence, is linked to higher risk in hypertensive patients. However, neither TG index nor HOMA-IR can accurately predict stroke death or recurrence in nondiabetic acute ischemic stroke patients. The TyG index may be useful for assessing risk in the broader community. High TG index correlates with unfavorable consequences after a stroke, including increased mortality and stroke recurrence. The TG index's linearity suggests it could improve ischemic stroke risk classification for the general population.

Key words: Stroke, Triglyceride glucose index, stroke recurrence, insulin resistance, mortality, diabetes.

INTRODUCTION



INTRODUCTION

Ischemic stroke remains a significant source of morbidity and mortality, particularly in the elderly population with underlying hypertension. Among the various risk factors associated with ischemic stroke, insulin resistance has garnered increasing attention due to its potential role in the pathogenesis and prognosis of cerebrovascular events. The triglyceride-glucose Index (TyG index), inferred from Fasting Triglyceride and Glucose levels, has emerged as a reliable surrogate marker for Insulin Resistance. Elevated TyG index has been implicated in the development and progression of various metabolic disorders, including diabetes mellitus and cardiovascular diseases. However, its prognostic significance in elderly hypertensive patients with acute ischemic stroke remains underexplored⁴⁷.

Given the high prevalence of hypertension in the elderly and its association with adverse cardiovascular outcomes, including ischemic stroke, there is a critical need to identify reliable prognostic markers for risk stratification and management in this vulnerable population. Understanding the relationship between TyGLUCOSE index and Ischemic stroke outcomes could provide valuable insights into the underlying mechanisms linking insulin resistance to cerebrovascular events and inform personalized therapeutic strategies^{48,49}.

Against this backdrop, this observational study aims to investigate the prognostic value of the TyGlucose Index in elderly hypertensive Patients presenting with acute ischemic stroke. By examining the association between TyG index levels and clinical outcomes, including stroke severity, functional disability, and mortality rates, we seek to elucidate the Character of insulin resistance in shaping the trajectory of Ischemic stroke in this high-risk population. This study endeavors to contribute to

the growing body of evidence on the utility of TyG index as a prognostic marker in ischemic stroke.

AIMS & OBJECTIVES

AIMS AND OBJECTIVES

AIM:

The present Study have existence toinvestigate the prognostic value of the Triglyceride GLucoseindex in elderly hypertensive Patients presenting with Acute Ischemic stroke at a tertiary care center.

OBJECTIVES:

The following were the objectives of present study:

- To estimate fasting triglyceride glucose levels in elderly hypertensive patient with ischemic stroke.
- To estimate fasting glucose-index in elderly hypertensive patient with Acuteischemic stroke
- To calculate-triglyceride glucoseindex in elderly hypertensive patient with Acute ischemic stroke.

REVIEW OF LITERATURE



REVIEW OF LITERATURE

Among all neurological illnesses, stroke is most common in the world and the most frequent. Hippocrates, the Father of Medicine, defined stroke as the abrupt onset of paralysis and named the condition "apoplexy," which means "stricken down with violence" and is commonly used by doctors today.⁶ The name "apoplexy" does not specify a particular diagnosis because abrupt paralysis can be caused by a wide range of diseases

Johann Jacob Wepfer carried out the Study on the Pathological apoplexy appearances following Hippocrates. He was the first to find evidence of post-mortem bleeding in the brains of apoplexy patients. He learned from the autopsy that the brain receives blood from the vertebral and carotid arteries. He hypothesised that apoplexy might possibly result from blockage of the main arteries. Thus, when apoplexy was categorized based on blood vessel pathology in 1928, the terms Stroke and Cerebrovascular disease (where "cerebro" refers to a portion of the brain and "vascular" to blood vessels) were born.⁷ In humans, brain injury starts the moment a blood vessel is blocked and lasts for a few days. Consequently, the window of time for treating the majority of common

PREVALANCE OF STROKE

Stroke is the illness that plagues wealthy and industrialized countries. Men more likely than women to have a stroke. Whilst it can happen at any age, it is more common in the elderly. An estimated 700,000 occurrences of stroke, 600,000 ischemic lesions, 100,000 hemorrhage's, and 175,000 fatalities from these causes occur Every year.

RISK FACTORS

According to estimates, stroke will rank fourth among the main causes of disabilityadjusted lifeyears (DALY) by 2020.A stroke is characterized as an abrupt onset of a non-decisive, focused neuro logical impairment that feels as though God has struck the patient.Hemorrhagic or ischemia can cause a stroke. IschemicStroke in 87% of cases.⁸

It has been demonstrated that blood pressure control lowers a person's risk of stroke.Systemic hypertension rates as the most significant risk factor associated with stroke.

Supplementary risk variables include

- DIABETES
- HYPERLIPIDEMIA.

Smoking: a higher risk factor for carotid atherosclerosis

Atrial fibrillation: primarily caused by a heart condition

Embolic stroke is caused by both bacterial and non-bacterial infectious endocarditis.

RIGHT TO LEFT SHUNT

- Systemic – “Hypercoagulable conditions like APLA”
- “Usage of Oral Contraceptive Pills”.
- “Symptomatic Carotid Artery Stenosis-70 to 99% of patients developstroke.”

ISCHEMIC STROKE

The term "ischemic stroke" describes a condition in which “cerebral perfusion is reduced as a result of thrombosis or embolism.”

Etiology:

Common Causes:

Thrombosis, large vessel thrombosis, lacunar stroke (small vessel); Dehydration; embolic- blockage; artery to artery; Aortic arch, carotid bifurcation, arterial dissection, myocardial infarction, dilated cardiomyopathy, atrial fibrillation, mural thrombus, and valvular lesions bacterial endocarditis, mechanical valve, and mitral stenosis, Atrial septal defect; paradoxical embolus; Atrial septal aneurysm, spontaneous echo contrast, and patent foramen ovale

Rare Causes:

Protein C and S deficiencies, antithrombin III deficiency, antiphospholipid syndrome, hypercoagulable diseases Systemic malignancy; Prothrombin G20210 mutation; Factor V Leiden mutation; Thalassemia, Polycythemia vera, Homocysteinemia, Systemic lupus erythematosus, Sickle Cell Anemia, and Thrombotic thrombocytopenic purpura Dysproteinemias; disseminated intravascular coagulation; syndrome of nephrotic kidneys; Primary CNS vasculitis, systemic vasculitis, fibromuscular dysplasia/vasculitis, venous sinus thrombosis, and inflammatory bowel disease

These are a few uncommon reasons of ischemic stroke:

HYPERCOAGULABLE DISORDERS:

An elevated risk of thrombosis is linked to several illnesses. Arterial thrombosis is caused by a lack of protein C and S. Embolic stroke is caused by Libman-Sacks endocarditis in SLE. Children who have sickle cell anemia frequently experience strokes.

FIBROMUSCULAR DYSPLASIA:

Fibromuscular dysplasia mostly affects the vertebral arteries and is more common in women. Typically, there is a partial occlusion. It is typical for renal artery involvement to result in hypertension. usually asymptomatic, but stroke and audible bruit may be present.

TEMPORAL ARTERITIS:

Age related temporal arteritis is prevalent. Giant cells cause granulomatous inflammation in the temporal arteries.

TAKAYASU ARTERITIS:

Idiopathic giant cell arteries, known as Takayasu arteritis, cause thrombosis in large blood vessels such as the carotid, vertebral, or aortic arch.

MOYA MOYA DISEASE:

Moya Moya illness typically affects the major Arteries. The blockage happens in the anterior and middle cerebral artery stems. A puff of smoke appears when collateral circulation is present.

REVERSIBLE POSTERIOR LEUKOENCEPHALOPATHY:

Cerebral ischemia is caused by a significant segmental vasoconstriction of the brain. Although the pathogenesis is unknown, the ischemia is totally reversible

LEUKOARAIOSIS:

Sometimes referred to as periventricular white matter illness, leukoaraiosis is the cause of many tiny vessel infarcts inside subcortical white matter. This is the “Result of Lipohyalinosis in tiny arteries” that penetrate the brain. Long-term SHT is where it commonly occurs.

The most typical sign of “Cerebral Autosomal dominant artery disease with Subcortical Infarcts and Leukoencephalopathy,” or CADASIL, is small vascular stroke.

TRANSIENT ISCHEMIC ATTACK (TIA):

Transient Ischemic Attack is the name used if the neurological signs and symptoms go away in less than a day (24 hours), regardless of any new brain alterations seen on imaging¹⁰. A third of TIA cases are in hypertensive people. A stroke either comes before or after them. 20% of TIA stroke cases happen within a month, and 50% happen within a year.¹¹ The early warning indicator of a potential blood vessel blockage is a TIA. Extended transient Ischemic attacks (TIAs) and many, differently patterned episodes are most likely caused by embolism; recurring, brief TIAs with similar patterns are primarily caused by atherosclerosis and thrombosis. Increased blood viscosity, such as that seen in leukemia, polycythemia vera, and hypercoagulable conditions, can also cause TIA. Rather than the carotid system, the vertebrobasilar system is where it most frequently happens.¹³

Following a transient ischemic event, a myocardial infarction is highly likely to develop.

PATHOPHYSIOLOGY OF ISCHEMIC STROKE:

The origin and risk factors of an ischemic stroke determine its pathophysiology. The disease could result from an embolism or thrombus, or it could be from diminished perfusion brought on by occlusion-causing stenosis in the artery system

- Alterations in the “permeability of the vessel wall”
- A variation in Viscosity, primarily an increase in blood viscosity
- rupture of a blood vessel
- atherosclerosis
- aneurysm dilatation
- hypertension
- atherosclerosis.
- arteritis, vasospasm
- developmental malfunctions

Differentiating between a thrombosis and an embolic lesion is challenging. When an embolic stroke happens, it happens abruptly and causes the greatest possible neurological loss. Stroke progression develops more slowly and gradually in thrombotic stroke. Artery to artery embolism can result to thrombosis of the vessels. The most common causes of stenosis are plaque deposition and atherosclerosis.

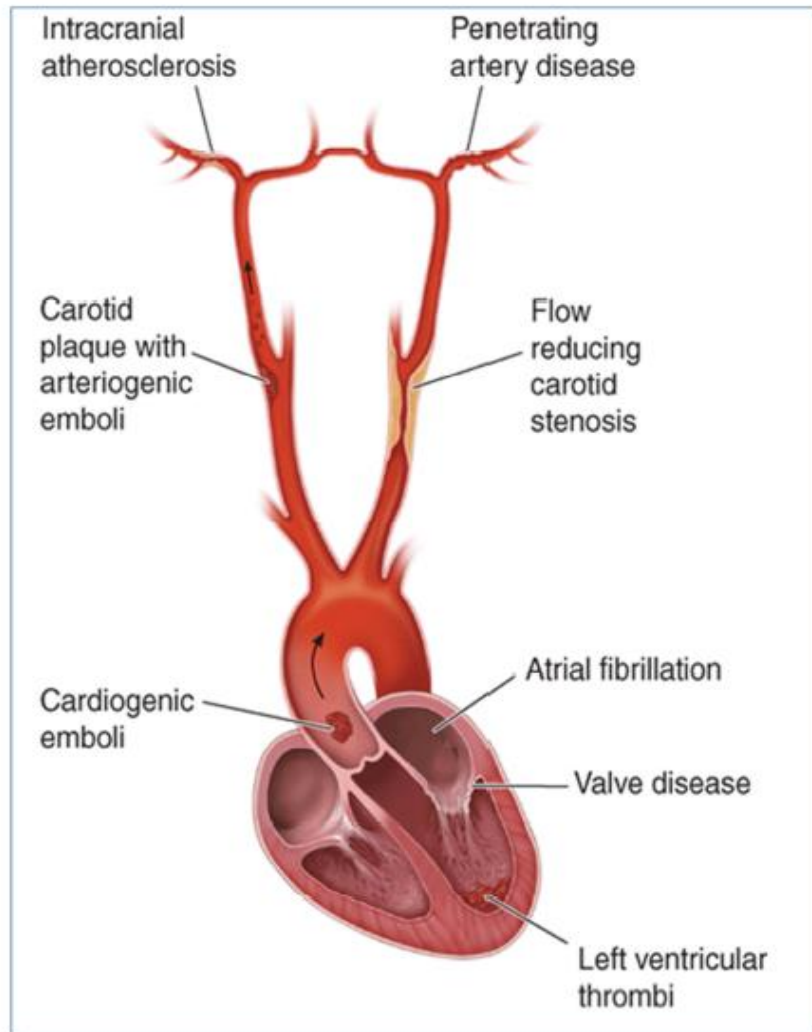


Figure 1: Stenosis and Embolism, Thrombosis.

The level of ischemia ,depends on the type and Duration of obstruction as well as the existence or the absence of other risk factors.

- Blood Pressure;

“Perfusion pressure in the Brain is determined by systemic blood pressure.”

Global cerebral ischemia,results from a Drop in the cerebral perfusion pressure caused by blood Pressure.

- “Hyperthermia”;

If the body temperature is higher, the severity of an ischemic injury increases.

- “Glucose Level”:

‘Hypoglycemia and Hyperglycemia are linked to less favorable results’

- Hypercoagulable state;

A concomitant hypercoagulable state exacerbates blood vessel blockage and increases the production of microthrombi.

NEURONAL DEATH:

Neurons can die in two different ways:

- Apoptosis:

The term "apoptosis" refers to the planned cell death that takes place in neurons under specific circumstances, such as ischemia. The “autolytic process that leads to cell death” is initiated by damage to the nucleus, which is then followed by the activation of suicide proteins in the nuclei. DNA cleavage acts as mediator in this process. The entire apoptotic process barely takes one hour..¹²

- Coagulation Necrosis:

A mechanism called coagulation necrosis causes cell death without causing inflammations. This is because both chemical and physical stimuli can harm the plasma membrane. The cell expands at first and then contracts. It takes 6 to 12 hours for this process to progress. Within a day, there is total necrosis¹³.

The appearance of apoptosis-related cell death differs from that of coagulation necrosis-related cell death.¹⁴

➤ Thrombosis:

The most frequent pathogenic reason for blood artery obstruction that results in thrombosis leads to atherosclerosis. The resulting plaque may become calcified, ulcerated, or broken. When the endothelium is damaged, thrombosis and occlusion-causing substances are released together with the activation of vasoactive enzymes.¹⁵

The production of microthrombi, or hypercoagulable states brought on by clot formation, in disease such as APLA and giant cell arteritis are the other pathological origins of thrombosis.

➤ Lacunar Infarcts:

A lacunar infarct is brought on by blockage of the small penetrating arteries that arise from the cerebral arteries. These could have a diameter of 100–400 μm. The infarct has a diameter roughly 20 mm. Lipohyalinosis is the cause of the pathology.¹⁶
¹⁷Instances range from 10 to 30 percent of all strokes. The lengthy, tortuous tiny arterioles in persons with persistent hypertension can develop microaneurysms, which increase the arteriole's susceptibility to blockage.

➤ Embolism:

There are several reasons why an artery may become embolized, but cardiac sources are the most frequent ones. Since the main cerebral artery carries 80% of blood flow, the majority of emboli obstruct it.¹⁸ In addition, less frequently, the superficial branches of the cerebral and cerebellar arteries are affected. Vasospasm can

also be brought on by embolic occlusion because it irritates the blood vessel. Younger patients experience vasospasm more frequently than older patients due to less atherosclerotic blood vessels in the former group.

The following are significant risk factors for paleor hemorrhagic infarcts:

A)The infarct's size

B) The collaterals' suitability

C) These start of anticoagulant and thrombolytic therapy

Hemorrhagic infarct is not solely associated with hypertension as a risk factor^{19,20}

➤ global Ischemia:

Another name for global ischemia is a hypotensive stroke. Any source of systemic hypotension has the potential to result in global ischemia. The purkinje cell layer of the cerebral cortex and the pyramidal cell layer of the hippocampal regions are the most often impacted cells. The brain's greymatter may potentially be impacted by this Ischemia. It most Frequently occurs in the Watershed region, which is situated where the “anterior,middle, and posteriorcerebral arteries” converge. Ten percent of infarcts are water shed infarcts. Carotid stenosis is the etiology in forty percent of instances.^{21,22}

A cerebral bloodflow of 50–60ml/100 gm/min is typical.²³Below this point, there is an increase in oxygen extraction from other cells, collaterals open, and cerebral vasodilatation occurs. Less than 20 milliliters per 100 grams per minute impairs the cerebral autoregulation process. A brain infarction occurs over the course of an hour if

the bloodflow is “16–18 ml/100 gm/min”. And lastly, if blood flow is less than 10 ml/100 gm/min, irreversible neuronal damage happens.²⁴

When an infarction occurs, more water accumulates both intracellularly and in the spaces between cells, which causes swelling of the infarcted area. In addition, leucocytes are recruited and inflammatory mediators are released, which lead to vasodilation and platelet aggregation.

Immune-regulated “platelets, erythrocytes, and leukocytes” attaching to the artery wall cause “occlusion and ischemia.”.The term global ischemia refers to the entire lack of blood flow to brain tissue, which results in irreversible neuronal death. Focal ischemia occurs when the ‘collaterals are able to keep the blood and oxygen flowing’.Ischemic is the core area that surrounds the infarcted area. This region, referred to as the ischemic penumbra, has minimal perfusion and living neurons.²⁵The key factor in ischemic stroke is maintaining blood flow to this ischemic penumbra because, once the flow is restored, the injured area of ischemia recovers; otherwise, it progresses to infarction, which worsens the clinical condition and result. Ischemic penumbra is observed within diffusion- and perfusion imaging on MRI and CT. Preserving the ischemic penumbra is the aim of newly discovered treatment procedures such as revascularization therapies, which are intended to prevent further infarction.

An infarct might be of two types.

- pale Infarct
- hemorrhagic Infarct

a) pale infarct:

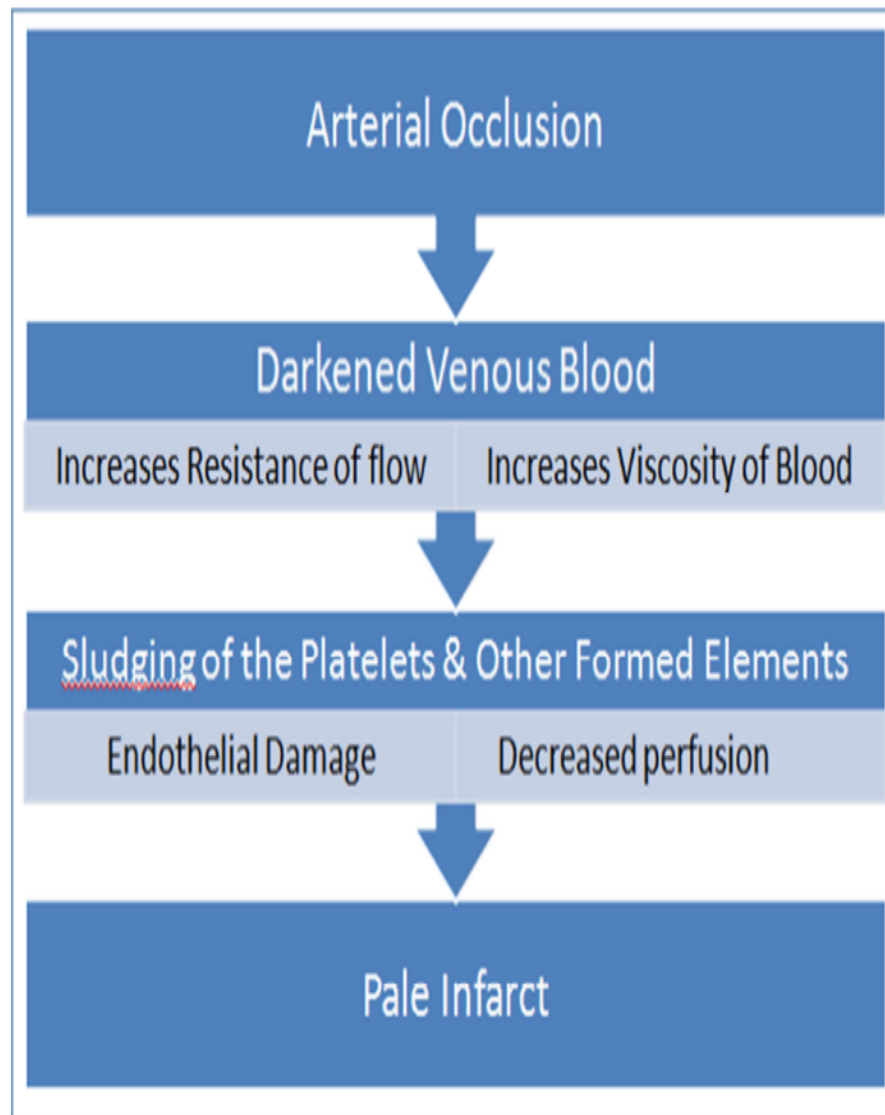


Figure 2: Flow chart of pale infarct pathogenesis.

b) Hemorrhagic Infarct:

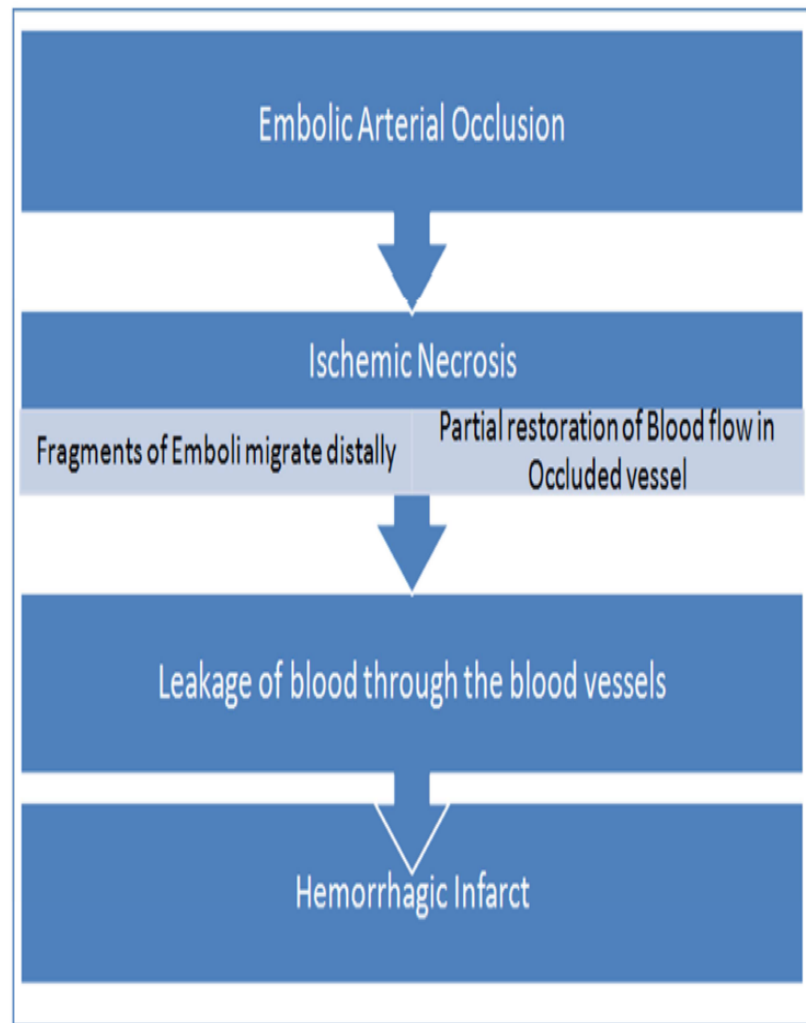


Figure 3: Flow chart of hemorrhagic infarct pathogenesis.

BIOCHEMICAL CHANGES:

When these depolarized cells are damaged, ATP and creatine kinase are depleted and potassium is effluxed. According to Groen et al., the risk of stroke is increased by low serum potassium²⁶. The destruction of phospholipids in neuronal cell membranes caused by the accumulation of free fatty acids modifies the homeostasis of

calcium. These alterations result in necrosis's histological characteristics. Cellular acidosis is brought by elevated intracellular calcium and extracellular potassium.

Cytotoxic edema is caused by the buildup of cytokines and other inflammatory mediators, such as prostaglandins, which causes the cell to enlarge..²⁷

CHANGES MAY BE DUE TO ALTERED METABOLIC FACTORS:

Glutamate:

Glutamate transporters remove glutamate, an excitatory neurotransmitter, from extracellular environment. The glutamate transporters release glutamate after a stroke. Because glutamate is excitotoxic, when it is released after a stroke, brain damage results. Moreover, glutamate raises calcium influx, which causes prolonged depolarization, enzyme activation, cytokine release, and cellular integrity degradation..²².

Mitochondrial dysfunctions:

Brain ischemia is caused by the activation of neuronal NOS and inducible nitric oxide in glial cells, which results in mitochondrial damage²³

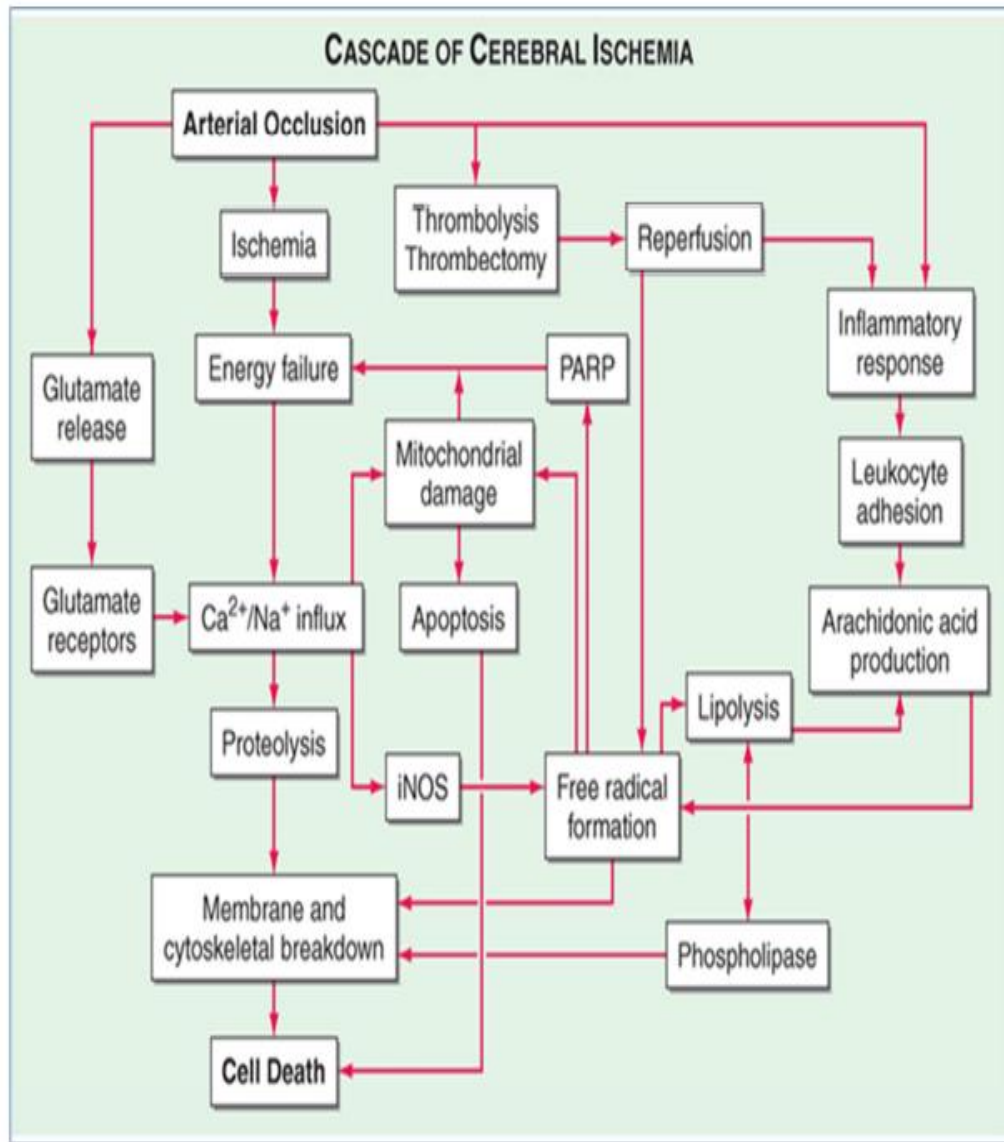


Figure 4: Cascade of cerebral ischemia.

TYPES OF ISCHEMIC STROKE:

Different classification systems are used to describe acute ischemic stroke. The early symptoms are the primary factor used in the Oxford Community Stroke Project (OCSP) classification, commonly referred to the Birmingham or Oxford classification. Depending on the severity of the symptoms, stroke event is classed as follows:

- a) total anterior circulation infarct (TACI).
- b) partial anterior circulation infarct (PACI).
- c) lacunar infarct (LACI).
- d) posterior circulation infarct (POCI).

above four factors foretell the prognosis, the location of afflicted brain region, the underlying etiology, and the severity of the stroke

According to 'TOAST (Trial of Organization 10172 in Acute Stroke Treatment) classification', a stroke is categorized as being caused by clinical symptoms as well as the findings of additional investigation.

- a) Atherosclerosis-related thrombosis or embolism in a major artery
- b) A cardiac origin embolism
- c) A little blood vessel blockage.
- d) Additional identified causes.
- e) Undetermined cause: two plausible explanations, no causation found, or insufficient research.

a)

(ACS):Anterior CirCulatory STROKE

The major blood vessels involved in ACS are

- principally the internal carotid artery (ICA) in the carotid artery
- middle cerebral artery (MCA)
- Anterior cerebral artery

- Anterior choroidal artery

Angios fugax, hemiplegia, hemianisocoria, aphasia, and anosognosia can result when the anterior cerebral artery and middle cerebral artery are blocked at the carotid artery's top

Middle Cerebral artery (MCA):

Wernicke's aphasia, hemianesthesia, homonymous hemianopia, gaze preference to same side, and contralateral hemiplegia are all indications of MCA involvement.²⁵

Anterior cerebral artery (ACA):

Urinary incontinence, significant abulia, paraparesis/quadriparesis, and bilateral pyramidal symptoms are all brought on by anterior cerebral artery involvement.

Anterior choroidal artery:

Contralateral hemiplegia, homonymous hemianopia, and hemianesthesia are caused by involvement of the anterior choroidal artery.

Common carotid artery:

Jaw claudication is caused by involvement of the common carotid artery.

POSTERIOR CIRCULATION STROKE (PCS):

The posterior cerebral artery, posterior inferior cerebellar artery, vertebral artery, and basilar artery are the arteries that are affected. The development of Emboli/Atheroma at the apex of the basilar artery is a Major cause of posterior circulation disorders. Occlusion of the posterior cerebral artery causes P1 and P2 syndromes. The characteristics are

- homonymous hemianopia on the contralateral side with macular sparing.
- Distal posterior CA infarctions bilaterally
- Cortical blindness
- Anton's Syndrome
- Balint's syndrome: This condition is brought on by an infarction in the watershed region between PCA and MCA
- Embolic blockage of the basilar artery leads to ptosis, Pupillary asymmetries, lack of light reaction, and somnolence in both eyes
- vertebral And posterior ICA - Lateral medullary Syndrome, Medial medullary Syndrome
- Quadriplegia may arise from anterior spinal artery occlusion, but hemiparesis is not characteristic vertebral artery occlusions.

INVESTIGATION:

CT brain;

The Infarct might not show up consistently for a day or two. and brain Ct scan performed in initial hours following an infarction typically reveal no abnormalities.

MRI;

The location and degree of “Infarction in all regions of brain”, including cortical surface Along with posteriorfossa, can be accurately determined by MRI

Conventional Xray cerebral angiography:

The Goldstandard for diagnosing and quantifying cerebral artery atherosclerotic stenosis is conventional x-ray cerebral angiography.

Ultrasound-Technique:

By using a combination of a Dopplerultrasound assessment flow velocity and a B-mode ultrasound image, often known as "duplex" ultrasound, it is possible to accurately identify and quantify stenosis at internal carotid Artery origin. Additionally helpful is the measurement of vertebro basilar flow and THE ACa,MCa and PCa flow using transcranial Doppler. Because stnotic lesions in big cerebral arteries increases Systolic flow velocity, this latter approach can identify them. TCd can enhance majorartery recanalization and aid in thrombolysis after recombinant tissue plasminogen activator delivery.

PERFUSION-Techniques:

“PEt and Xenon methods (mostly xenon-CT) “may both measur Cerebral bloodflow. Although these instruments are typically employed in research, they can be helpful in assessing the importance of artery stenosis and in the planning stages of revascularization procedures. Relativ cerebral Blood flow is reported using “MRperfusion methods and single-photon emission computed tomography (SPECT)”.

Disabilityadjusted Lifeyear (DALY):

The global burden of disease is measured using disability adjusted life year. It is the amount of healthy time that the patient loses as a result of early death and living with illness. In India, the DALY is frequently assessed using the Modified Rankin's scale.²⁸

modified Rankin's scale²⁹:

It is among the most widely utilized clinical outcome metrics in stroke patients.

0 → “no symptoms”

1 → “no significant Disability,” capable of performing all routine tasks despite certain indications

2 → Slight disability, capable of handling personal matters on their own without help, but unable to complete all prior tasks

3 → Moderate disability, needs assistance, yet is able to walk without it

4 → Moderately severe disability. unable to walk without assistance and unable to manage to own physical demands without help.

5 → severe Disability, needs continuous nursing care and supervision; they are bedridden and incontinent.

6 → Dead.

TRIGLYCERIDE GLUCOSE INDEX (TyG Index)

The Triglyceride Glucose Index (TyG Index) is a mathematical formula used to assess insulin resistance.

Calculation:

TyGlucose Index is calculated by using formula: $TyG = \ln [\text{fasting Triglycerides (mg/dL)} \times \text{Fasting Glucose (mg/dL)} / 2]$, where \ln represents the natural logarithm.

- **Explanation of Components:**

- Fasting Triglycerides: Measure of triglyceride levels in the bloodstream after an overnight fast.

- Fasting Glucose: Measure of glucose levels in the bloodstream after an overnight fast³⁰.

Significance of TyG Index:

- **Insulin Resistance Assessment:** TyGlucose Index benefit as a Surrogate Marker for insulin resistance, reflecting the body's reduced sensitivity to insulin, a hormone crucial for glucose metabolism.
- **Metabolic Health Indicator;** Increased TyG Index levels are indicative of dysregulation in the metabolism of fat and glucose, which are key components of metabolic diseases such as dyslipidemia and diabetes.
- **Cardiovascular Risk Prediction:** Some Studies have demonstrated this particular higher TyG Index values are linked to a higher incidence of cardiovascular illnesses, such as coronary artery disease, stroke, and peripheral Arterial disease.

- Inflammatory and Oxidative Stress Marker: Insulin resistance, as indicated by elevated TyG Index, is often accompanied by chronic low-grade inflammation and increased oxidative stress, both of which contribute to the development and progression of metabolic and cardiovascular diseases³¹.

Interpretation of TyG Index:

- Interpretation Guidelines: While there is no universally accepted cutoff for defining elevated TyG Index, values above certain thresholds (e.g., 8.5 or 9.0) have been proposed as indicators of increased insulin resistance and metabolic risk.
- Population-specific Considerations: Interpretation of TyG Index should consider age, sex, ethnicity, and other demographic factors, as reference ranges may vary among different populations.
- Clinical Context: Interpretation of TyG Index values should be integrated with clinical context, including other risk factors, comorbidities, and patient history, to inform decision-making regarding risk assessment and management strategies.

Triglyceride Glucose Index (TyG Index) like a prognostic Marker for Cardiovascular risk:

Association with Cardiovascular Risk³²:

- Previous Studies: Numerous epidemiological studies have demonstrated a marked Association between elevated TyGLUCOSE Index levels and an increased risk of cardiovascular events. These events include myocardial

infarction, stroke, coronary artery disease, peripheral arterial disease, and cardiovascular mortality.

- **Meta-Analyses:** Meta-analyses of Observational studies have consistently shown that Higher TyGLUCOSE Index values are independently related with a greater risk of cardiovascular events, even after adjusting for traditional risk factors such as age, sex, smoking, hypertension, and dyslipidemia.
- **Dose-Response Relationship:** Some studies have suggested a dose-response relationship, where higher TyG Index levels are associated with a progressively higher risk of cardiovascular events. This strengthens the evidence supporting the role of TyGLUCOSE Index as a predictor of cardiovascular risk.

Predictive Value³³:

- **Prognostic Utility:** TyG Index has emerged as a promising prognostic marker for predicting outcomes in various cardiovascular conditions, including ischemic stroke. Studies have shown that elevated TyG Index levels at baseline are associated with an increased risk of adverse outcomes, such as recurrent strokes, functional disability, and mortality, in patients with ischemic stroke.
- **Longitudinal Studies:** Longitudinal cohort studies have demonstrated that individuals with higher TyG Index values are more likely to experience recurrent cardiovascular events and have worse long-term outcomes compared to those with lower TyG Index levels. This suggests that TyGlucose Index could be used to know high-risk patients who may benefit from more aggressive management strategies.

Mechanisms:**INSULIN RESISTANCE:**

Insulin resistance plays a major role in the pathogenesis of metabolic disorders. Including dyslipidemia and Type 2 diabetes. Increased TyG Index levels are indicative of underlying insulin resistance, which raises the risk of cardiovascular events by interfering with endothelial dysfunction, proinflammatory states, and dysregulated glucose and lipid METABOLISM.

INFLAMMATION:

Insulin resistance and cardiovascular disease are closely associated with chronic low-grade inflammation, which is defined by elevated levels of proinflammatory cytokines and acute-phase reactants. Higher levels of inflammatory markers, which encourage atherosclerosis and plaque instability, such as C-reactive protein (CRP), interleukin-6 (IL-6), and tumor necrosis factor-alpha (TNF-alpha), have been linked to elevated TyG indexes.

OXIDATIVE STRESS:

Increased oxidative stress, which arises from an imbalance between the generation of reactive oxygen molecules and antioxidant defenses, is also linked to insulin resistance. Cardiovascular disease is facilitated by oxidative stress, which also increases vascular inflammation, lipid peroxidation, and endothelial dysfunction..

ENDOTHELIAL DYSFUNCTION:

Atherosclerosis and thrombosis are made more likely by endothelial dysfunction, which is characterized by decreased nitric oxide (NO) bioavailability and

elevated adhesion molecule expression. Endothelial function is disrupted by insulin resistance and related metabolic abnormalities, which increases the risk of cardiovascular disease.³⁴.

Clinical Utility of TyG Index^{35,36},

Risk Stratification:

- **Identifying High-Risk Individuals:** Depending on an individual's insulin resistance status, the TyG Index can be a helpful tool for classifying them into various risk groups for cardiovascular events. Increased sensitivity to insulin and metabolic disorders are linked to higher TyG Index readings and are recognized as indicators for cardiovascular illnesses.
- **Helping to Make Treatment Decisions:** Clinicians can adjust treatment plans for patients who are identified as having a greater chance of cardiovascular events. This could involve lifestyle changes targeted at enhancing sensitivity to insulin and metabolic health, as well as more vigorous control of modifiable hazards like obesity, dyslipidemia, and hypertension

Monitoring:

- **Evaluation of Treatment Response:** Keeping a close eye on TyG Index values can give important information about how well therapies to enhance sensitivity to insulin and metabolic function are working. For instance, over time, pharmaceutical interventions like insulin-sensitizing drugs and lifestyle changes like eating adjustments and greater physical activity may result in lower TyG Index results.

- **Early Detection of Metabolic Dysfunction:** Changes in TyG Index levels over time can also serve as an early indicator of metabolic dysfunction and insulin resistance progression. Monitoring TyG Index longitudinally allows clinicians to intervene promptly and implement targeted interventions to prevent or delay the onset of cardiovascular complications.

Prognostic Value in Elderly Hypertensive Patients with Acute Ischemic Stroke^{37,38,39}:

- **Prediction of Stroke Severity:** Elevated TyG Index levels at baseline have been associated with increased stroke severity in elderly hypertensive patients with acute ischemic stroke. Higher TyG Index values may reflect underlying metabolic abnormalities and systemic inflammation, which contribute to Pathogenesis of ischemicstroke and exacerbate its severity.
- **Functional Outcomes:** Studies have suggestedthat TyG Index may also Predict functional outcomes following acute ischemic stroke. higher TyG index levels have beenassociated with greater functional disability and poorer recovery rates, potentially due to the adverse effects of insulin resistance on neurovascular function and brain tissue repair mechanisms.
- **Mortality Prediction**Higher fatality rates in elderly hypertension patients experiencing acute ischemic stroke have been associated with elevated TyG Index levels. larger TyG Index readings and lower survival rates in this population are linked to greater neurological damage, a larger load of comorbidities, and a more susceptibility to consequences.

PUBLISHED PAPAER:

Study conducted by **Li et al.** investigated the correlation between the TriglycerideGlucose index (TyG index) and the consequences of ischemic stroke, focusing on the role of inflammation. Their research provided significant insight into the TyG index's capacity to forecast worse outcomes for ischemic stroke patients, and it was published in the journal *Atherosclerosis* in 2021. They found a robust association between higher TyGlucoseIndex levels and worse outcomes for stroke survivors after conducting a thorough analysis. It also provides insight into the pathophysiology linking metabolic dysfunction and these could impact stroke severity-inflammation mediated pathway in this relationship.⁴⁰.

Zhang et al. *Diabetes Research and Clinical Practice* did an expansive research in a similar experiment to prove these results. analysed and reported on the prognostic capability of TyGLUCOSE in 2020 to risk-stratify clinical outcomes in acute ischemic stroke patients. Using data from In the setting of the CATIS project, these authors aimed to assess what benefit this assessment represented by using TyGindex. Stroke care in acute event. Their results showed that the TyGlucose Index was able to capture this value as a pragmatic clinical outcome in acute ischemic stroke, medical care. a valuable tool for providers to assess risk and optimize treatment⁴¹.

Another study **Jin et al.** aimed to assess the predictive value of TyG index in acute ischemic stroke. Published in the *Journal of Atherosclerosis and Thrombosis* in 2021. "With careful examination, they showed that the two factor Conclusions: The TyGLUC index was an accurate predictor of poor outcomes in patients with acute ischemic stroke. This was illustrated in their own study that demonstrated possible clinical applications of the TyG index - which led to a discussion about how ischemic

helping stroke patients to decide for themselves what course of treatment is right and the risks involved⁴².

The predictive value of TyGLUCOSE INDEX goes beyond acute stroke events as, It was evidenced by **Guo et al.** who carried out a prospective cohort study analysis⁴³.

Ren et al. added to the increasing body of evidence demonstrating the prognostic value of the TyGLUCOSE Index by conducting a cohort research on the Prospect of ischemic stroke⁴⁴.

Ying Liao et al. conducted a retrospective observational study to investigate the correlation between the Triglyceride-Glucose index (TyG) and Death in Intensive care unit (ICU) Patients. Utilizing data from the Medical information Mart for Intensive Care III (MIMIC-III) database, their Study Included in 3026 Patients had an initial Triglyceride and Glucose Data on the First day of ICU admission. These patients were categorized into quartiles (Q1–Q4) based on TyG index levels. Over a follow-up period of 10.46 years, 1148 patients (37.9%) experienced all-cause mortality, with a notable proportion occurring during hospital (11.6%) and ICU (8.5%) stays. Kaplan–Meier analysis demonstrated a significant Prognostication between higher TyGlucose index levels and increased risk of all-cause mortality (log-rank $P = 0.021$). Furthermore, multivariable cox proportional hazards analyses revealed that elevated TyGLUCOSE Index was independently associated with higher risks of Intensive care unit death (HR: 1.72, 95% CI 1.18–2.52, $P = 0.005$) and hospital death (HR: 2.19, 95% CI 1.59–3.03, $P < 0.001$). Specifically, each 1-unit increase in TyG index was associated with a 1.19-fold higher in the risk of death during hospitalization. These findings suggest a strong correlation between TyG index and all-cause Death in critically ill patients, indicating

its potential utility as a prognostic marker for identifying individuals at heightened risk of ICU and hospital mortality⁴⁵.

Weiyan Guo et al. embarked on a comprehensive investigation to explore the intricate relation between the triglyceride-glucose (TyG) index and the risk of recurrent stroke within 1 year following acute ischemic stroke (AIS). Their study encompassed a sizable cohort of 2,288 AIS patients, where meticulous baseline assessments, including TyG index calculations, were conducted. Subsequently, recurrent stroke occurrences were meticulously tracked over the ensuing 12 months. Leveraging sophisticated statistical methodologies such as multivariable Cox regression and restricted cubic spline analyses, the researchers uncovered a nuanced, nonlinear correlation Between the TyGLUCOSE Index and the risk of recurrent stroke, particularly noteworthy implications observed in female patients. Notably, individuals situated in the highest Quartile of TyGindex manifestation exhibited a substantially heightened risk of recurrent stroke, underscoring the potential of the TyG index as a pivotal prognostic indicator for post-AIS recurrent stroke events, particularly among female AIS patients. By including Weiyan Guo as an author, the study duly acknowledges their invaluable contributions to the research endeavor⁴⁶.

MATERIALS AND METHODS

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MATERIALS AND METHODS

PLACE OF STUDY:

The present study was carried out in the department of general medicine, RL Jalappa Hospital and Research Centre, Kolar

TYPE OF STUDY:

The present study was prospective observational study.

DURATION OF STUDY:

The study was carried out for a session of 18months, ,from July 2022 to December 2023.

SAMPLE SIZE:

The study keeps conducted on 64patients.

The sample size was calculated by using the formula:

$$n = \frac{z_{1-\alpha/2}^2 \sigma^2}{d^2}$$

where,

s is the Standard deviation

d is the Precision

1- $\alpha/2$ is the Desired Confidence level.

Yimo Zhou et al. reported the median TyG index to be 8.73 (IQR, 8.33–9.21). We computed SD from IQR assuming normal distribution of the values using the formula $SD = IQR/1.35$.

$SD = 0.65$

Assuming the expected population standard deviation to be **0.65** for Triglyceride-Glucose Index, and employing t-distribution to estimate sample size, the study required a sample size of **64 subjects** to estimate a mean with **95% confidence** and a **precision of 0.16**.

INCLUSION CRITERIA:

Patients meeting the following criteria were enrolled into the study.

- Patients aging more than 60 years.
- Patients who were known cases of hypertension and on treatment.
- Patients newly diagnosed with ischemic stroke
- Patients willing to give consent.
- Patients willing to participate.

EXCLUSION CRITERIA:

Patients meeting the following criteria were excluded from the study.

- Patients who were known cases of type 2 diabetes mellitus or on glucose lowering drugs.
- Patients on lipoprotein lowering drugs (statins).
- Patients with haemorrhagic stroke.

- Patients with embolic stroke.
- Patients who were not willing to give consent.
- Patients not willing to participate.

INFORMED CONSENT

All the patients fulfilling selection criteria were explained about the details of the disease process, options of treatment, ultimate outcome, possible effects, complications and chances of recurrence in both procedure and a written informed consent was obtained before enrolment. They were informed of their right to withdraw from the study at any stage.

DATA COLLECTION

- A detailed clinical history and physical examination was carried out on patients followed by a thorough review of their hospital records.
- All the patients meeting inclusion criteria were included in the study.
- Data on Blood pressure, Triglyceride levels, FBS levels, NCCT/MRI BRAIN and ECG was collected.
- The Triglyceride Glucose Index, derived as $\text{Ln}[\text{fasting triglyceride (mg/dL)} \times \text{fasting glucose (mg/dL)} / 2]$, is the logarithmized product of Triglycerides and Glucose.
- These values are recorded and noted down in the master charts.
- All the data was documented and analyzed by subjecting to statistical analysis.

STATISTICAL ANALYSIS

The collected data was entered into Microsoft Excel Worksheet-2010 and data was taken into IBM SPSS Statistic for windows, version 24 (IBM Corp., Armonk, N.Y., USA) software for calculation of frequency, percentage, mean, standard deviation and probability value.

Qualitative data was represented in the form of frequency and percentage.

- Association between qualitative variables was assessed by Chi Square test with continuity correction for 2 x 2 tables and
- Fisher's exact test for all 2 x 2 tables, where P value of chi square test was not valid due to small counts.

Quantitative data was represented using mean and standard deviation.

- Analysis of quantitative data within the groups was done using paired t test if data passes 'Normality test'.
- Analysis Of Variance (ANOVA) was used to compare more than two groups.

* A '**P**' value of <0.05 was considered statistically significant.

RESULTS



RESULTS

The present prospective observational study was conducted on 64 patients in the department of general medicine, RL Jalappa Hospital and Research Centre, Kolar, for a period of 18 months from July 2022 to December 2023.

The following were the study results:

Table 1: AGE WISE DISTRIBUTION OF SUBJECTS.

AGE GROUP (YEARS)	NUMBER OF SUBJECTS (N)	PERCENTAGE (%)
61 to 70	20	31.25%
71 to 80	28	43.75%
81 to 90	14	21.88
> 90	2	3.12
Total	64	100

In the present study, the subjects were categorized into four age groups. The above table gives data on distribution of study subjects based on their age.

Majority of subjects were in age group of 71 to 80 years i.e. 28 (43.75%) subjects followed by 20 (31.25%) subjects in age group of 61 to 70 years, 14 (21.88%) Subjects in age group of 81 to 90years and finally 2 (3.12%) subjects of age >90 years.

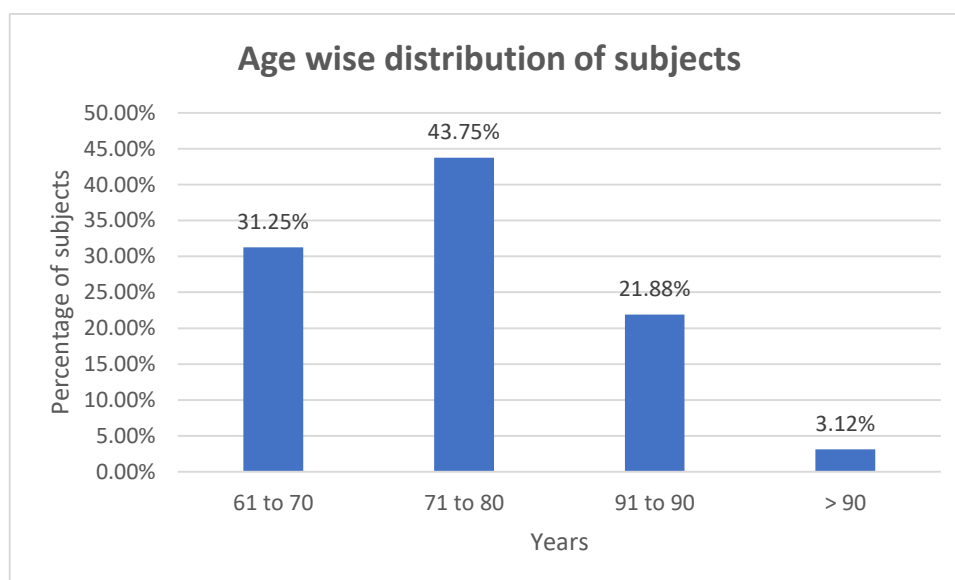


Figure 5: Age wise distribution of subjects

Table 2: Distribution of subjects basing on their gender.

Gender	Number of subjects (N)	Percentage (%)
Male	35	54.69%
Female	29	45.31%
Total	64	100

The above table gives data on distribution of subjects according to gender.

Majority of subjects were males i.e., 35 (54.69 %) followed by 29 (45.31 %) females.

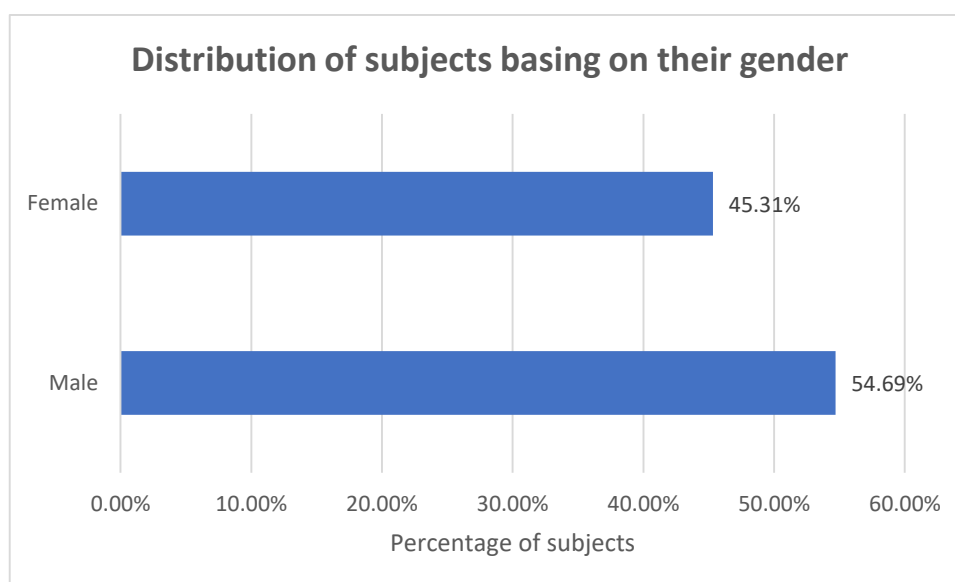


Figure 6: Distribution of subjects basing on their gender.

Table 3: Distribution of subjects basing on their area of residence.

Area of residence	Number of subjects (N)	Percentage (%)
Urban	41	64.06%
Rural	23	35.94%
Total	64	100

The above table gives data on distribution of subjects according to their area of residence.

Majority of subjects reside in urban areas i.e. 41 (64.06%) subjects followed by 23 (35.94%) subjects residing in rural area.

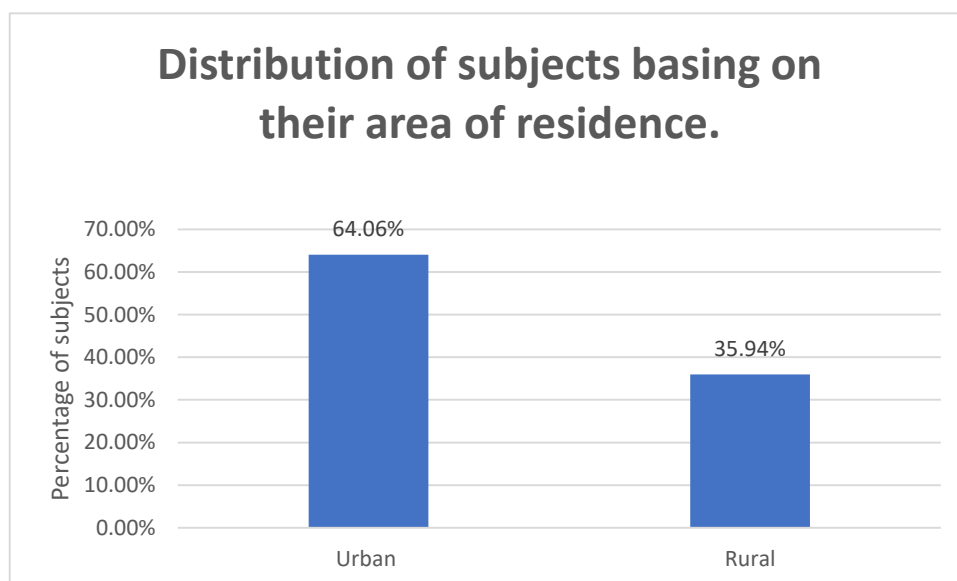


Figure 7: DISTRIBUTION OF SUBJECTS BASING ON THEIR AREA OF RESIDENCE.

Table 4: DIstribution of Subjects basing on their religion.

Religion	Number of subjects (N)	Percentage (%)
Hindu	25	39.06%
Muslim	21	32.81%
Christian	14	21.87%
Others	4	6.25%
Total	64	100

The above table gives data on distribution of subjects according to their religion.

Majority of subjects were Hindus i.e. 25 subjects (39.06%) followed by 21 subjects (32.81 %) were Muslims, 14 subjects (21.87%) were Christian and 4 subject (6.25%) belonged to other religions.

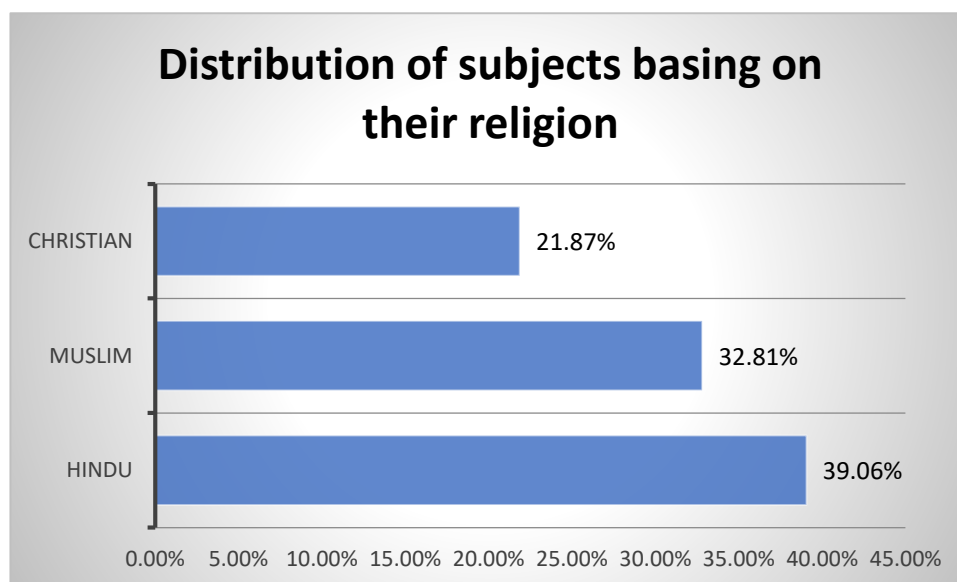


Figure 8: Distribution of subjects basing on their religion.

Table 5: Distribution of subjects basing on their socioeconomic status.

Socioeconomic status	Number of subjects (N)	Percentage (%)
High	35	54.69%
Middle	10	15.62%
Low	19	29.69%
Total	64	100

The above table gives data on distribution of subjects according to their socioeconomic status.

Majority of subjects were with high socioeconomic status i.e. 35 subjects (54.69%) followed by 19 subjects (29.69%) with low socioeconomic status and 10 subjects (15.62%) followed by middle socioeconomic status.

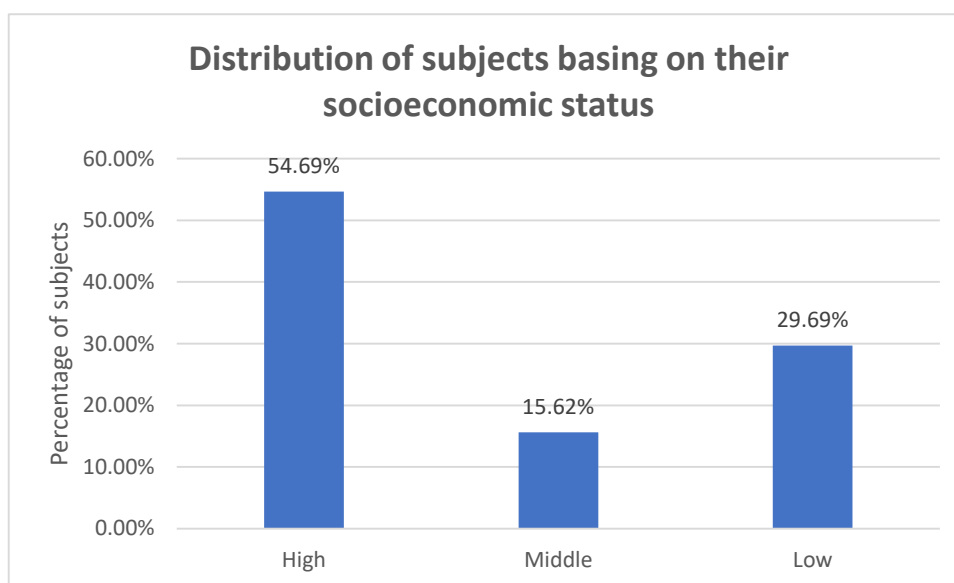


Figure 9: Distribution of subjects basing on their socioeconomic status.

Table 6: Distribution of subjects basing on their past medical history.

Past medical history	Number of subjects (N)	Percentage (%)
Hypertension	64	100

The above table gives data on distribution of subjects according to their past medical history.

Majority of subjects had history of hypertension i.e. 64 subjects (100 %)

Table 7: Mean biochemical parameters of study subjects.

Biochemical parameters	Mean	SD
FASTING TRIGLYCERIDE(mg/dl)	167.5	15.1
FASTING GLUCOSE(mg/dl)	91.8	10.5
triglyceride glucose index	8.9	0.6

The above table gives data on distribution of subjects according to their mean biochemical parameters.

The fasting triglyceride levels were 167.5 ± 15.1 mg/dL, fasting glucose levels were 91.8 ± 10.5 mg/dL and triglyceride glucose index were 8.9 ± 0.6 .

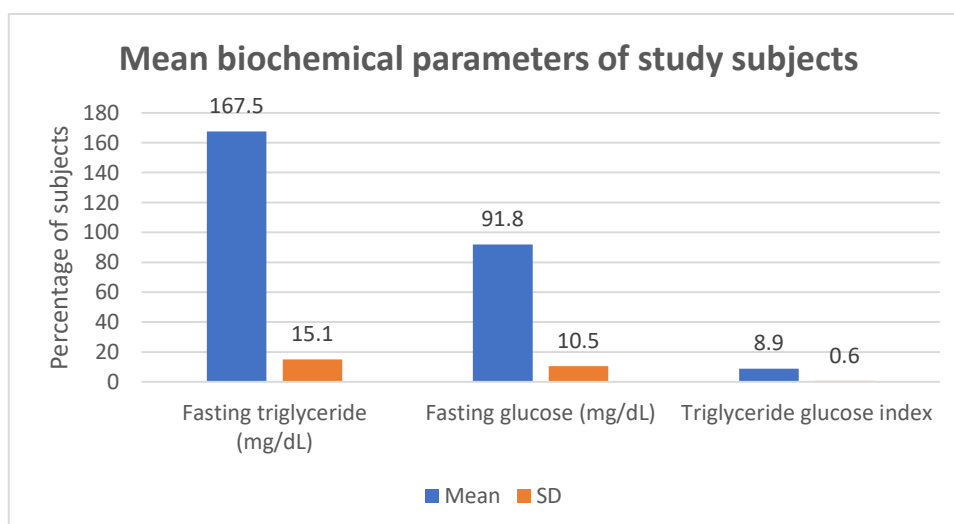


Figure 11: Mean biochemical parameters of study subjects

Table 8: Distribution of subjects basing on their triglyceride glucose index

triglyceride glucose index	Group Name	Number of subjects (N)	Percentage (%)
≤ 8.33	I	16	25
8.34 to 8.73	II	16	25
8.74 to 9.20	III	16	25
≥ 9.21	IV	16	25
Total	-----	64	100

The above table gives data on distribution of subjects according to their triglyceride glucose index

In the present study, the Subjects are categorized Mainly to Four Groups based on their Triglyceride glucose index.

Based on triglyceride glucose index, there were 16 (25%) subjects belonging to Group I, 16 (25%) subjects belonging to Group II, 16 (25%) subjects belonging to Group III, 16 (25%) subjects belonging to Group IV.

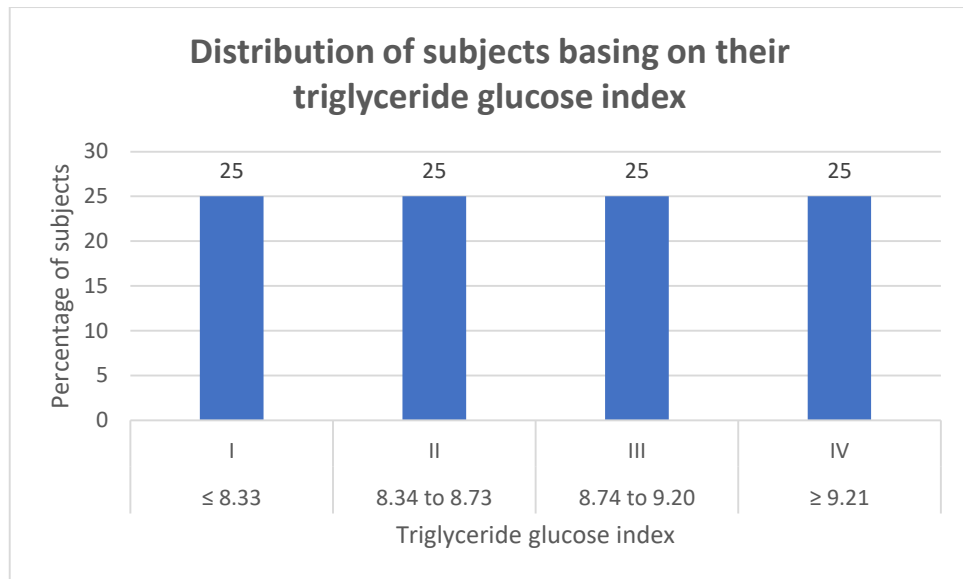


Figure 12: Distribution of subjects basing on theirtriglyceride glucose index

Table 9: Mean blood pressure of subjects Vs triglyceride glucose index group.

Blood pressure (mmHg)	triglyceride glucose index group				p-value
	I (Mean \pm SD)	II (Mean \pm SD)	III (Mean \pm SD)	IV (Mean \pm SD)	
Systolic	148.3 \pm 23.2	148.9 \pm 23.5	149.1 \pm 22.5	151.3 \pm 23.8	0.0001
Diastolic	85.7 \pm 13.1	87.5 \pm 13.8	88.1 \pm 13.3	88.9 \pm 13.5	0.0001

The above table gives data on distribution of subjects according to the mean blood pressure of subjects Vs triglyceride glucose index group.

The mean systolic blood pressure in group **I** subjects was 148.3 \pm 23.2 mmHg, in group **II** was 148.9 \pm 23.5 mmHg, in group **III** was 149.1 \pm 22.5 mmHg and that in group **IV** subjects was 151.3 \pm 23.8 mmHg. The p-value calculated was **0.0001** indicating a very high statistical difference between groups in terms of systolic blood pressure of subjects and TRIGYCERIDE GLUCOSE INDEX group. Systolic pressure was high in Group **IV**.

The mean diastolic blood pressure in group **I** subjects was 85.7 \pm 13.1 mmHg, in group **II** was 87.5 \pm 13.8 mmHg, in group **III** was 88.1 \pm 13.3 mmHg and that in group **IV** subjects was 88.9 \pm 13.5 mmHg. The p-value calculated was **0.0001** indicating a very high statistical difference between groups in terms of diastolic blood

pressure of subjects and TRIGLYCERIDE GLUCOSEINDEX group. Diastolic pressure was high in Group IV.

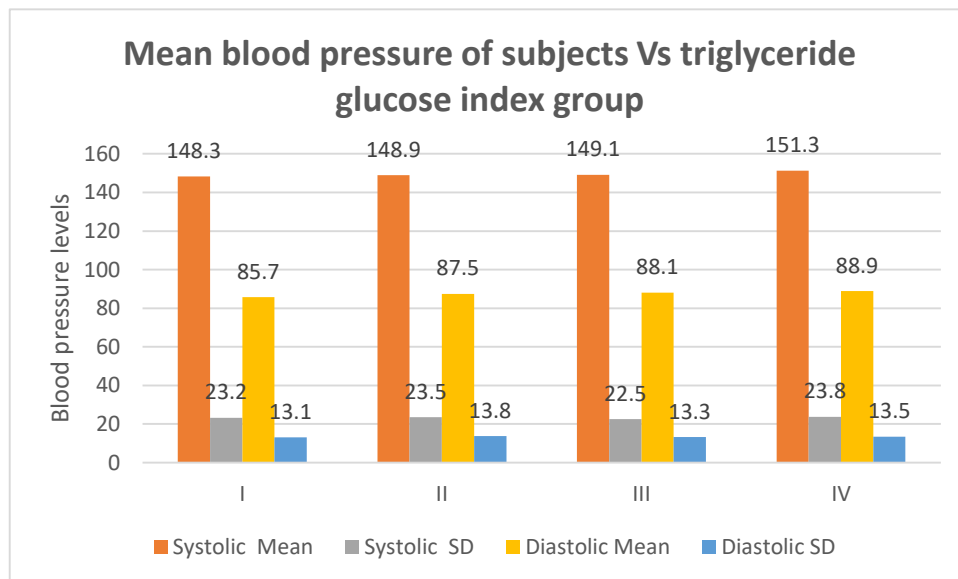


Figure 13: Mean blood pressure of subjects Vs Triglyceride glucose index group

Table 10: Distribution of subjects basing on triglyceride glucose index group Vs events.

Event	TRIGLYCERIDE INDEX GROUP				Total	p-value
	I (N %)	II (N %)	III (N %)	IV (N %)		
Stroke recurrence	1 (3.70 %)	1 (3.70 %)	9 (33.33 %)	10 (37.03 %)	27	0.0001
Poor functional outcome	5 (14.70 %)	6 (17.64 %)	11 (32.35 %)	12 (35.29 %)	34	0.0001
Neurologic worsening	6 (21.42 %)	6 (21.42 %)	8 (28.57 %)	8 (28.57 %)	28	0.001
Mortality	1 (10 %)	1 (10 %)	3 (30 %)	5 (50 %)	10	0.02

The above table gives data on distribution of subjects basing on TRIGLYCERIDE GLUCOSEINDEX group Vs events.

Out of **27** subjects with stroke recurrence, majority were in group IV, i.e., 10 subjects (37.03 %); followed by 9 subjects (33.33 %) group III; 1 subject (3.70 %) group I and II respectively. The p-value calculated was **0.0001** indicating a highly significant statistical difference among the TRIGLYCERIDE GLUCOSEINDEX

groups in terms of Stroke recurrence. The incidence was high in group IV (i.e., TRIGLYCERIDE GLUCOSE INDEX ≥ 9.21).

Out of **34** subjects with poor functional outcome, majority were in group IV, i.e., 12 (35.29 %) subjects; followed by 11 (32.35 %) subjects in group III; 6 (17.64 %) subjects in group II and 5 (14.70 %) subjects in group I. The p-value calculated was **0.0001** indicating a highly significant statistical difference between the Triglyceride Glucose Index groups in terms of poor functional outcome. The incidence was high in group IV (i.e., Triglyceride Glucose Index ≥ 9.21).

Out of **28** subjects with neurological worsening, majority were in group III and IV, i.e., 8 (28.57 %) subjects respectively; followed by 6 (21.42 %) subjects in group I and II respectively. The p-value calculated was **0.0001** indicating a highly significant statistical difference between the Triglyceride Glucose Index groups in terms of neurological worsening. The incidence was high in group IV (i.e., Triglyceride Glucose Index of Group III: 8.74 to 9.20 Triglyceride Glucose Index of Group IV ≥ 9.21).

Out of **10** subjects with mortality, majority were in group IV, i.e., 5 (50 %) subjects; followed by 3 (30 %) subjects group III; 1 (10 %) subject group I and II respectively. The p-value calculated was **0.0001** indicating a highly significant statistical difference between the Triglyceride Glucose Index groups in terms of mortality. The incidence was high in group IV (i.e., Triglyceride Glucose Index ≥ 9.21).

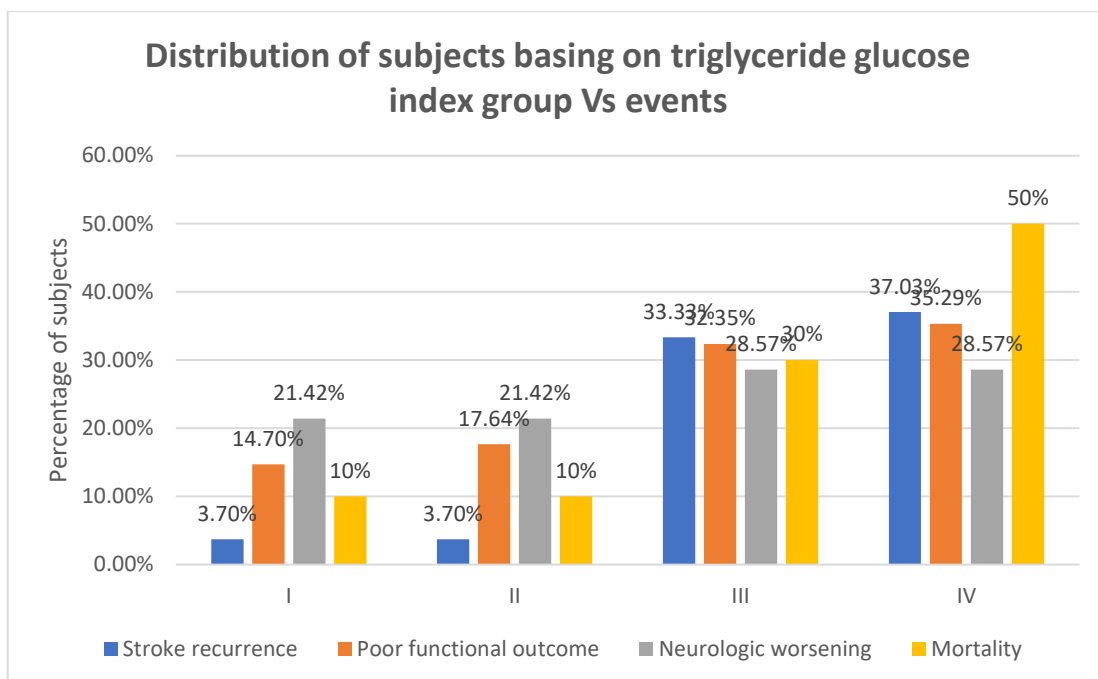


Figure 14: Distribution of subjects basing on Triglyceride Glucose Index Group Vs events.

DISCUSSION



DISCUSSION

Especially in low- and middle-income nations, stroke is a leading cause of mortality and disability. Improving post-stroke outcomes is crucial, and identifying high-risk patients and implementing effective secondary preventions is essential.⁵⁰ Insulin resistance (IR)⁵¹⁻⁵³ is prevalent in stroke patients, promoting atherosclerosis, hemodynamic disturbances, and platelet adhesion, potentially triggering stroke recurrence in IS patients.^{54,55}

The TyGIndex is a marker for insulin resistance, indicating a reduced sensitivity to insulin, a hormone essential for glucose metabolism. It can help stratify individuals into risk categories for cardiovascular events based on their insulin resistance status.⁵² Higher TyG Index values indicate greater insulin resistance and metabolic dysfunction, known risk factors for cardiovascular diseases. Clinicians can tailor treatment strategies by identifying individuals at higher risk, such as aggressive management of modifiable risk factors like hypertension, dyslipidemia, and obesity, and lifestyle interventions aimed at improving insulin sensitivity and metabolic health.⁵³

Regular monitoring of TyG Index levels can provide insights into the effectiveness of interventions, such as lifestyle modifications and pharmacological interventions. Changes in TyG Index levels over time can also indicate metabolic dysfunction and insulin resistance progression, allowing clinicians to intervene promptly and implement targeted interventions to prevent or delay cardiovascular complications.⁵⁵

Recently, it remains to be shown that the triglyceride Glucose index (TyG), a novel calculator for metabolic abnormalities, is linked to the Risk of cardiovascular disease (CVD) in those who appear to be in good condition.

AGE:

Majority of Subjects Were in Age Goup of 71 to 80 years i.e. 43.75% subjects followed by 31.25% subjects in age group of 61 to 70 years, 21.88% Subjects in Age group of 81 to 90years and finally 3.12% subjects of age >90 years.

Our study was in correlation with **Zhou Y et al⁵⁵**, **Yang X et al⁵⁶**

Study by	Majority age (in years)
Zhou Y et al⁵⁵	65-75
Yang X et al⁵⁶	65-75
Present study	71 to 80 (43.75%)

GENDER:

Majority of subjects were males i.e., 54.69 % subjects followed by 45.31 % female subjects.

Our study was in correlation with **Zhou Y et al⁵⁵**, **Yang X et al⁵⁶**

Study by	Majority (Percentage)
Zhou Y et al⁵⁵	Males (63.5%)
Yang X et al⁵⁶	Males (64.8%)
Present study	Males (54.69%)

AREA OF RESIDENCE:

Majority of subjects reside in urban areas i.e. 64.06% subjects followed by 35.94% subjects residing in rural area.

Our study was in correlation with **Cadilhac DA et al⁵⁷**, **Liu R et al⁵⁸**, **Tajiri N et al⁵⁹**

Study by	Majority (Percentage)
Cadilhac DA et al⁵⁷	Urban (56.3%)
Liu R et al⁵⁸	Urban (61.6%)
Tajiri N et al⁵⁹	Urban (54.9%)
Present study	Urban (64.06%)

RELIGION:

Majority of subjects were Hindus i.e. 39.06% subjects followed by 32.81 % subjects were Muslims, 21.87% subjects were Christian and 6.25% subjects belonged to other religions.

Our study was in correlation with **Grundy, Scott M., et al⁴⁷**, **Guo Y et al⁶⁰**

Study by	Majority (Percentage)
Grundy, Scott M., et al⁴⁷	Hindus (53.2%)
Guo Y et al⁶⁰	Hindus (42.9%)
Present study	Hindus (39.06%)

SOCIOECONOMIC STATUS:

Majority of subjects were with high socioeconomic status i.e. 54.69% subjects followed by 19 29.69% subjects with low socioeconomic status and 15.62% subjects followed by middle socioeconomic status.

Our study was in correlation with **Lavallée PC et al⁶⁰**, **Marshall IJ et al⁶¹**, **Addo J et al⁶²**

Study by	Majority (Percentage)
Lavallée PC et al⁶⁰	High (61.2%)
Marshall IJ et al⁶¹	High (48.5%)
Addo J et al⁶²	High (49.8%)
Present study	High (54.69%)

MEAN BIOCHEMICAL PARAMETERS:

The fasting triglyceride levels were 167.5 ± 15.1 mg/d, fasting glucose levels were 91.8 ± 10.5 mg/dL and triglyceride glucose index were 8.9 ± 0.6 .

Our study was in correlation with **Miao M et al⁶⁷**

Study by	Mean		
	Fasting triglyceride levels	Fasting glucose levels	Triglyceride glucose index
Miao M et al⁶⁷	152.2 ± 22.1	83.5 ± 13.0	10.0 ± 6.1
Present study	167.5 ± 15.1	91.8 ± 10.5	8.9 ± 0.6

TRIGLYCERIDE GLUCOSE INDEX:

Based on triglyceride glucose index, there were 25% subjects belonging to Group I, 25% subjects belonging to Group II, 25% subjects belonging to Group III, 25% subjects belonging to Group IV.

Our study was in correlation with **Hu L et al⁶⁸, Liu D et al⁶⁹, Hoshino T et al⁷⁰**

MEAN BLOOD PRESSURE:

The mean systolic blood pressure in group **I** subjects was 148.3 ± 23.2 mmHg, in group **II** was 148.9 ± 23.5 mmHg, in group **III** was 149.1 ± 22.5 mmHg and that in group **IV** subjects was 151.3 ± 23.8 mmHg. The p-value calculated was **0.0001** indicating a very high statistical difference between groups in terms of systolic blood pressure of subjects and Triglyceride Glucose Index group. Systolic pressure was high in Group **IV**.

The mean diastolic blood pressure in group **I** subjects was 85.7 ± 13.1 mmHg, in group **II** was 87.5 ± 13.8 mmHg, in group **III** was 88.1 ± 13.3 mmHg and that in group **IV** subjects was 88.9 ± 13.5 mmHg. The p-value calculated was **0.0001** indicating a very high statistical difference between groups in terms of diastolic blood pressure of subjects and Triglyceride Glucose Index group. Diastolic pressure was high in Group **IV**.

Our study was in correlation with **Zhou Y et al⁵⁵, Miao M et al⁶⁷, Carcel C et al⁷¹, Song L et al⁷²**

TRIGLYCERIDE GLUCOSE INDEX VS EVENTS:

Out of **27** subjects with stroke recurrence, majority were in group IV, i.e. 37.03 % subjects; followed by 33.33 % subjects group III; 3.70 % subjects group I and II respectively. The p-value calculated was **0.0001** indicating a highly significant statistical difference between the Triglyceride Glucose Index groups in terms of Stroke recurrence. The incidence was high in group IV (i.e., Triglyceride Glucose Index \geq **9.21**).

Out of **34** subjects with poor functional outcome, majority were in group IV, i.e., 35.29 % subjects; followed by 32.35 % subjects in group III; “17.64 % Subjects in Group II and 14.70 % subjects in group II. The p-value calculated was **0.0001** indicating a highly significant statistical difference between the Triglyceride Glucose Index groups in terms of poor functional outcome. The incidence was high in group IV (i.e., Triglyceride Glucose Index \geq **9.21**).

Out of **28** subjects with neurological worsening, majority were in group III and IV, i.e., 28.57 % subjects respectively; followed by 21.42 % Subjects in Group I and II respectively. The p-value calculated was **0.0001** indicating a highly significant statistical difference between the Triglyceride Glucose Index groups in terms of neurological worsening. The incidence was high in group IV (i.e. Triglyceride Glucose Index of Group **III: 8.74 to 9.20 Triglyceride Glucose Index of Group IV \geq 9.21**).

Out of **10** subjects with mortality, majority were in group IV, i.e., 50 % subjects; followed by 30 % subjects group III; 10 % subject group I and II respectively. The p-value calculated was **0.0001** indicating a highly significant statistical difference between the Triglyceride Glucose Index groups in terms of mortality. The incidence was high in group IV (i.e., Triglyceride Glucose Index \geq **9.21**).

Our study was in correlation with **Addo J et al⁶³, Gürsoy GT et al⁷³, Zhou Z⁷⁴**

CONCLUSION



CONCLUSION

An elevated TG index has been linked to a Greater the risk of stroke death and recurrence in hypertensive patient. However, in nondiabetic acute ischemic stroke patients, neither TG index nor HOMA-IR can be a reliable indicator of stroke death or recurrence. The TyGLUCOSE Index may be useful in maximizing risk assessment Within the broader community. Moreover, a strong correlation has been shown between a high TG index and numerous unfavourable consequences following a stroke, including increased mortality and stroke recurrence.

TG index's linearity as an ischemic stroke indicator is suggested by the proportionate correlation between its increase and the prevention of ischemic stroke. According to our research, TG index may be useful in improving ischemic stroke risk classification for the general population.

SUMMARY



SUMMARY

- Majority of “Subjects were in Age Group of 71 to 80 years i”.e. 43.75% subjects followed by 31.25% subjects in age group of 61 to 70 years, 21.88% Subjects in Age group of 81 to 90years and finally 3.12% subjects of age >90 years.
- Majority of subjects were males i.e., 54.69 % subjects followed by 45.31 % female subjects.
- Majority of subjects reside in urban areas i.e. 64.06% subjects followed by 35.94% subjects residing in rural area.
- Majority of subjects were Hindus i.e. 39.06% subjects followed by 32.81 % subjects were Muslims, 21.87% subjects were Christian and 6.25% subjects belonged to other religions.
- Majority of subjects were with high socioeconomic status i.e. 54.69% subjects followed by 19 29.69% subjects with low socioeconomic status and 15.62% subjects followed by middle socioeconomic status.
- Majority of subjects had history of hypertension, i.e. 100 % subjects.
- The fasting triglyceride levels were 167.5 ± 15.1 mg/d, fasting glucose levels were 91.8 ± 10.5 mg/dL and triglyceride glucose index were 8.9 ± 0.6 .
- Based on triglyceride glucose index, there were 25% subjects belonging to Group I, 25% subjects belonging to Group II, 25% subjects belonging to Group III, 25% subjects belonging to Group IV.
- it has a very high statistical Difference between groups in terms of blood pressure of subjects and Triglyceride Glucose Index group. Both systolic and diastolic pressure was high in Group **IV**.

- There was a highly Significant Statistical difference among the Triglyceride Glucose Index groups in terms of stroke recurrence. The incidence was high in group IV (i.e., Triglyceride Glucose Index ≥ 9.21).
- Regarding poor functional result, there was a statistically significant variance between the groups based on the triglyceride glucose index. The incidence was high in group IV (i.e., Triglyceride Glucose Index ≥ 9.21).
- Considering neurological deterioration, there was a statistically significant distinction between the Triglyceride Glucose Index groups.. The incidence was high in group IV (i.e., Triglyceride Glucose Index of Group **III: 8.74 to 9.20 Triglyceride Glucose Index** of Group **IV ≥ 9.21**).
- In terms of mortality, there was a profound statistical disparity between the groups based on the triglyceride Glucose index.. The incidence was high in group IV (i.e., Triglyceride Glucose Index ≥ 9.21).

BIBLIOGRAPHY

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ANNEXURES



ANNEXURES

INFORMED CONSENT FORM

Name of the study - **"AN OBSERVATIONAL STUDY OF TRIGLYCERIDE GLUCOSE INDEX AS A PROGNOSTIC MARKER IN ELDERLY HYPERTENSIVE PATIENTS WITH ACUTE ISCHEMIC STROKE IN A TERTIARY CARE CENTRE "**

I have read the foregoing information, or it has been read to me. I have had the opportunity to ask questions about it and any questions that I have asked have been answered to my satisfaction. I consent voluntarily to participate as a participant in this research.

Print Name of Participant _____

Signature of Participant _____ Date _____

For illiterate -

I have witnessed the accurate reading of the consent form to the potential participant, and the individual has had the opportunity to ask questions. I confirm that the individual has given consent freely.

Print name of witness _____ AND Thumb print of participant _____

Signature of witness _____ Date _____

Statement by the researcher/person taking consent

I have accurately read out the information sheet to the potential participant with the best of my ability. I confirm that the participant was given an opportunity to ask questions about the study, and all the questions asked by the participant have been answered correctly and to the best of my ability. I confirm that the individual has not been coerced into giving consent, and the consent has been given freely and voluntarily.

A copy of this ICF has been provided to the participant.

Print Name of Researcher taking the consent _____

Signature of Researcher taking the consent _____ Date _____

ಮಾಹಿತಿ ನೀಡಿದ ಒಪ್ಪಿಗೆ ನಮೂನೆ

ಅಧ್ಯಯನದ ಹೆಸರು - “ಟ್ರೈಗ್ಲಿಸರೈಡ್ ಗ್ಲೂಕೋಸ್ ಇಂಡೆಕ್ಸ್‌ನ ಒಂದು ಅವಲೋಕನಾತ್ಮಕ ಅಧ್ಯಯನವು ಒಂದು ಪ್ರಾಗ್ನೋಸ್ಟಿಕ್ ಮಾರ್ಕರ್ ಆಗಿ ಹಿರಿಯ ಹೈಪರ್‌ಟೆನ್ಸಿವ್ ರೋಗಿಗಳಲ್ಲಿ ತೀವ್ರತರವಾದ ರಕ್ತಚೂರತೆಯ ಸ್ಕ್ರೀನ್ ಹೊಂದಿರುವ ರೋಗಿಗಳಲ್ಲಿ ನಾನು ಮೇಲಿನ ಮಾಹಿತಿಯನ್ನು ಓದಿದ್ದೇನೆ ಅಥವಾ ಅದನ್ನು ನನಗೆ ಓದಿದ್ದೇನೆ. ಅದರ ಬಗ್ಗೆ ಪ್ರಶ್ನೆಗಳನ್ನು ಕೇಳಲು ನನಗೆ ಅವಕಾಶವಿದೆ ಮತ್ತು ನಾನು ಕೇಳಿದ ಯಾವುದೇ ಪ್ರಶ್ನೆಗಳಿಗೆ ನನ್ನ ತೃಪ್ತಿಗೆ ಉತ್ತರಿಸಲಾಗಿದೆ. ಈ ಸಂಶೋಧನೆಯಲ್ಲಿ ಪಾಲ್ಗೊಳ್ಳುವವನಾಗಿ ಭಾಗವಹಿಸಲು ನಾನು ಸ್ವಯಂಪ್ರೇರಣೆಯಿಂದ ಸಮ್ಮತಿಸುತ್ತೇನೆ.

ಭಾಗವಹಿಸುವವರ ಹೆಸರನ್ನು ಮುದ್ರಿಸಿ _____

ಭಾಗವಹಿಸುವವರ ಸಹಿ _____ ದಿನಾಂಕ _____

ಅನಕ್ಷರಸ್ಥರಿಗೆ -

ಸಂಭಾವ್ಯ ಪಾಲ್ಗೊಳ್ಳುವವರಿಗೆ ಒಪ್ಪಿಗೆಯ ನಮೂನೆಯ ನಿಖರವಾದ ಓದುವಿಕೆಯನ್ನು ನಾನು ನೋಡಿದ್ದೇನೆ ಮತ್ತು ವ್ಯಕ್ತಿಯು ಪ್ರಶ್ನೆಗಳನ್ನು ಕೇಳಲು ಅವಕಾಶವನ್ನು ಹೊಂದಿದ್ದೇನೆ. ವ್ಯಕ್ತಿಯು ಮುಕ್ತವಾಗಿ ಒಪ್ಪಿಗೆ ನೀಡಿದ್ದಾರೆ ಎಂದು ನಾನು ದೃಢೀಕರಿಸುತ್ತೇನೆ.

ಸಾಕ್ಷಿಯ ಹೆಸರನ್ನು ಮುದ್ರಿಸಿ _____ ಮತ್ತು ಭಾಗವಹಿಸುವವರ ಹೆಬ್ಬರಳು ಮುದ್ರೆ

ಸಾಕ್ಷಿಯ ಸಹಿ _____ ದಿನಾಂಕ _____

ಒಪ್ಪಿಗೆಯನ್ನು ತೆಗೆದುಕೊಳ್ಳುವ ಸಂಶೋಧಕ/ವ್ಯಕ್ತಿಯ ಹೇಳಿಕೆ

ಸಂಭಾವ್ಯ ಭಾಗವಹಿಸುವವರಿಗೆ ನನ್ನ ಸಾಮರ್ಥ್ಯಕ್ಕೆ ತಕ್ಕಂತೆ ನಾನು ಮಾಹಿತಿ ಹಾಳೆಯನ್ನು ನಿಖರವಾಗಿ ಓದಿದ್ದೇನೆ.

ಭಾಗವಹಿಸುವವರಿಗೆ ಅಧ್ಯಯನದ ಕುರಿತು ಪ್ರಶ್ನೆಗಳನ್ನು ಕೇಳಲು ಅವಕಾಶವನ್ನು ನೀಡಲಾಗಿದೆ ಎಂದು ನಾನು ಖಚಿತಪಡಿಸುತ್ತೇನೆ ಮತ್ತು ಭಾಗವಹಿಸುವವರು ಕೇಳಿದ ಎಲ್ಲಾ ಪ್ರಶ್ನೆಗಳಿಗೆ ಸರಿಯಾಗಿ ಉತ್ತರಿಸಲಾಗಿದೆ ಮತ್ತು ನನ್ನ ಸಾಮರ್ಥ್ಯದ ಅತ್ಯುತ್ತಮ. ಸಮ್ಮತಿಯನ್ನು ನೀಡುವಂತೆ ವ್ಯಕ್ತಿಯನ್ನು ಒತ್ತಾಯಿಸಲಾಗಿಲ್ಲ ಮತ್ತು ಒಪ್ಪಿಗೆಯನ್ನು ಮುಕ್ತವಾಗಿ ಮತ್ತು ಸ್ವಯಂಪ್ರೇರಣೆಯಿಂದ ನೀಡಲಾಗಿದೆ ಎಂದು ನಾನು ದೃಢೀಕರಿಸುತ್ತೇನೆ.

ಈ ICF ನ ಪ್ರತಿಯನ್ನು ಭಾಗವಹಿಸುವವರಿಗೆ ಒದಗಿಸಲಾಗಿದೆ.

ಸಮ್ಮತಿಯನ್ನು ತೆಗೆದುಕೊಳ್ಳುವ ಸಂಶೋಧಕರ ಹೆಸರನ್ನು ಮುದ್ರಿಸಿ _____

ಸಮ್ಮತಿಯನ್ನು ತೆಗೆದುಕೊಳ್ಳುವ ಸಂಶೋಧಕರ ಸಹಿ _____ ದಿನಾಂಕ _____

Patient information sheet

**Name of the study - " AN OBSERVATIONAL STUDY OF TRIGLYCERIDE GLUCOSE INDEX
AS A PROGNOSTIC MARKER IN ELDERLY HYPERTENSIVE PATIENTS WITH ACUTE
ISCHEMIC STROKE IN A TERTIARY CARE CENTRE "**

Stroke is a leading cause of morbidity and mortality world wide . Many studies have shown that high blood pressure is the leading risk factor for the global disease burden, in particular, the risk of stroke. They are associated with increased morbidity and mortality, decreased quality of life and increased healthcare expenditures, at Sri Devaraj Urs Academy of Higher Education & Research has decided to undertake a study on this regard.

We are inviting the patients with ischemic stroke to take part in this study, however based on criteria list, eligible participants will be chosen among the interested ones.

Your participation in this research is entirely voluntary. It is your choice whether to participate or not. We will collect blood samples to test Fasting blood glucose, fasting triglyceride, CT brain plain/ MRI brain plain. You can take your regular antihypertensive medications during the exercise sessions.

By participating in this research you will benefit by improved strength, balance required to do your daily activities effectively. Your participation will also help us to use the outcomes of this study for future subjects. Your participation in this study will not put you at any risk.

All information collected from you will be strictly confidential & will not be disclosed to any outsider. This information collected will be used for research purpose. This information will not reveal your identity & this study have been reviewed by central ethical committee.

There is no compulsion to participate in this study, further you are at the liberty to withdraw from the study at any time if you wish to do so. Your treatment aspect will not be affected if you not wish to participate. You are required to sign only if you voluntarily agree to participate in proposed study. A copy of this document will be given to you for your information.

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ರೋಗಿಯ ಮಾಹಿತಿ ಹಾಳೆ

ಅಧ್ಯಯನದ ಹೆಸರು -“ ಟ್ರೈಗ್ಲಿಸರೈಡ್ ಗ್ಲೂಕೋಸ್ ಇಂಡೆಕ್ಸ್‌ನ ಒಂದು ಅವಲೋಕನಾತ್ಮಕ ಅಧ್ಯಯನವು ಒಂದು ಪ್ರಾಗ್ನೋಸ್ಟಿಕ್ ಮಾರ್ಕರ್ ಆಗಿ ವಯಸ್ಸಾದ ಅಧಿಕ ರಕ್ತದೊತ್ತಡ ರೋಗಿಗಳಲ್ಲಿ ತೀವ್ರ ರಕ್ತಕೊರತೆಯ ಸ್ಕ್ರೀನ್‌ಗೆ ಒಳಗಾಗುತ್ತದೆ.

ಸ್ಟ್ರೋಕ್ ಪ್ರಪಂಚದಾದ್ಯಂತ ರೋಗಿ ಮತ್ತು ಮರಣಕ್ಕೆ ಪ್ರಮುಖ ಕಾರಣವಾಗಿದೆ. ಜಾಗತಿಕ ಕಾಯಿಲೆಯ ಹೊರಗೆ, ನಿರ್ದಿಷ್ಟವಾಗಿ, ಪಾರ್ಶ್ವವಾಯು ಅಪಾಯಕ್ಕೆ ಅಧಿಕ ರಕ್ತದೊತ್ತಡವು ಪ್ರಮುಖ ಅಪಾಯಕಾರಿ ಅಂಶವಾಗಿದೆ ಎಂದು ಅನೇಕ ಅಧ್ಯಯನಗಳು ತೋರಿಸಿವೆ. ಅವರು ಹೆಚ್ಚಿದ ಕಾಯಿಲೆ ಮತ್ತು ಮರಣ, ಕಡಿಮೆ ಗುಣಮಟ್ಟದ ಜೀವನ ಮತ್ತು ಹೆಚ್ಚಿದ ಆರೋಗ್ಯ ವೆಚ್ಚಗಳೊಂದಿಗೆ ಸಂಬಂಧ ಹೊಂದಿದ್ದಾರೆ, ಶ್ರೀ ದೇವರಾಜ್ ಅರ್ಸ್ ಅಕಾಡೆಮಿ ಆಫ್ ಹೈಯರ್ ಎಜುಕೇಶನ್ ಮತ್ತು ರಿಸರ್ಚ್ ಈ ನಿಟ್ಟಿನಲ್ಲಿ ಅಧ್ಯಯನವನ್ನು ಕೈಗೊಳ್ಳಲು ನಿರ್ಧರಿಸಿದೆ.

ಈ ಅಧ್ಯಯನದಲ್ಲಿ ಪಾಲ್ಗೊಳ್ಳಲು ರಕ್ತಕೊರತೆಯ ಪಾರ್ಶ್ವವಾಯು ಹೊಂದಿರುವ ರೋಗಿಗಳನ್ನು ನಾವು ಆಹ್ವಾನಿಸುತ್ತಿದ್ದೇವೆ, ಆದಾಗ್ಯೂ ಮಾನದಂಡಗಳ ಪಟ್ಟಿಯನ್ನು ಆಧರಿಸಿ, ಆಸಕ್ತರಲ್ಲಿ ಅರ್ಹ ಭಾಗವಹಿಸುವವರನ್ನು ಆಯ್ಕೆ ಮಾಡಲಾಗುತ್ತದೆ.

ಈ ಸಂಶೋಧನೆಯಲ್ಲಿ ನಿಮ್ಮ ಭಾಗವಹಿಸುವಿಕೆಯು ಸಂಪೂರ್ಣವಾಗಿ ಸ್ವಯಂಪ್ರೇರಿತವಾಗಿದೆ. ಭಾಗವಹಿಸಬೇಕೋ ಬೇಡವೋ ಎಂಬುದು ನಿಮ್ಮ ಆಯ್ಕೆ. ಉಪವಾಸದ ರಕ್ತದಲ್ಲಿನ ಗ್ಲೂಕೋಸ್, ಉಪವಾಸ ಟ್ರೈಗ್ಲಿಸರೈಡ್, CT ಬ್ಲೈನ್ ಫ್ಲೇನ್ / MRI ಬ್ಲೈನ್ ಫ್ಲೇನ್ ಪರೀಕ್ಷಿಸಲು ನಾವು ರಕ್ತದ ಮಾದರಿಗಳನ್ನು ಸಂಗ್ರಹಿಸುತ್ತೇವೆ. ವ್ಯಾಯಾಮದ ಅವಧಿಯಲ್ಲಿ ನಿಮ್ಮ ನಿಯಮಿತ ಆಂಟಿಹೈಪರ್ಟೆನ್ಸಿವ್ ಔಷಧಿಗಳನ್ನು ನೀವು ತೆಗೆದುಕೊಳ್ಳಬಹುದು.

ಈ ಸಂಶೋಧನೆಯಲ್ಲಿ ಭಾಗವಹಿಸುವ ಮೂಲಕ ನಿಮ್ಮ ದೈನಂದಿನ ಚಟುವಟಿಕೆಗಳನ್ನು ಪರಿಣಾಮಕಾರಿಯಾಗಿ ಮಾಡಲು ಅಗತ್ಯವಿರುವ ಸುಧಾರಿತ ಶಕ್ತಿ, ಸಮತೋಲನದಿಂದ ನೀವು ಪ್ರಯೋಜನ ಪಡೆಯುತ್ತೀರಿ. ನಿಮ್ಮ ಭಾಗವಹಿಸುವಿಕೆಯು ಈ ಅಧ್ಯಯನದ ಫಲಿತಾಂಶಗಳನ್ನು ಭವಿಷ್ಯದ ವಿಷಯಗಳಿಗೆ ಬಳಸಲು ನಮಗೆ ಸಹಾಯ ಮಾಡುತ್ತದೆ. ಈ ಅಧ್ಯಯನದಲ್ಲಿ ನಿಮ್ಮ ಭಾಗವಹಿಸುವಿಕೆಯು ನಿಮಗೆ ಯಾವುದೇ ಅಪಾಯವನ್ನುಂಟು ಮಾಡುವುದಿಲ್ಲ.

ನಿಮ್ಮಿಂದ ಸಂಗ್ರಹಿಸಲಾದ ಎಲ್ಲಾ ಮಾಹಿತಿಯು ಕಟ್ಟುನಿಟ್ಟಾಗಿ ಗೌಪ್ಯವಾಗಿರುತ್ತದೆ ಮತ್ತು ಯಾವುದೇ ಹೊರಗಿನವರಿಗೆ ಬಹಿರಂಗಪಡಿಸಲಾಗುವುದಿಲ್ಲ. ಸಂಗ್ರಹಿಸಿದ ಈ ಮಾಹಿತಿಯನ್ನು ಸಂಶೋಧನಾ ಉದ್ದೇಶಕ್ಕಾಗಿ ಬಳಸಲಾಗುತ್ತದೆ. ಈ ಮಾಹಿತಿಯು ನಿಮ್ಮ ಗುರುತನ್ನು ಬಹಿರಂಗಪಡಿಸುವುದಿಲ್ಲ ಮತ್ತು ಈ ಅಧ್ಯಯನವನ್ನು ಕೇಂದ್ರ ನೈತಿಕ ಸಮಿತಿಯು ಪರಿಶೀಲಿಸಿದೆ. ಈ ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸಲು ಯಾವುದೇ ಬಲವಂತವಿಲ್ಲ, ಮುಂದೆ ನೀವು ಹಾಗೆ ಮಾಡಲು ಬಯಸಿದರೆ ಯಾವುದೇ ಸಮಯದಲ್ಲಿ ಅಧ್ಯಯನದಿಂದ ಹಿಂದೆ ಸರಿಯಲು ನಿಮಗೆ ಸ್ವಾತಂತ್ರ್ಯವಿದೆ. ನೀವು ಭಾಗವಹಿಸಲು ಬಯಸದಿದ್ದರೆ ನಿಮ್ಮ ಚಿಕಿತ್ಸೆಯ ಅಂಶವು ಪರಿಣಾಮ ಬೀರುವುದಿಲ್ಲ. ಉದ್ದೇಶಿತ ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸಲು ನೀವು ಸ್ವಯಂಪ್ರೇರಣೆಯಿಂದ ಒಪ್ಪಿಕೊಂಡರೆ ಮಾತ್ರ ನೀವು ಸಹಿ ಮಾಡಬೇಕಾಗುತ್ತದೆ. ನಿಮ್ಮ ಮಾಹಿತಿಗಾಗಿ ಈ ಡಾಕ್ಯುಮೆಂಟ್‌ನ ನಕಲನ್ನು ನಿಮಗೆ ನೀಡಲಾಗುತ್ತದೆ.

ಪಿ ಬಾಲ ಕೃಷ್ಣ

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PROFORMA

PARTICULARS OF PATIENTS

NAME:

AGE:

SEX:

OCCUPATION:

UHID NUMBER:

PHONE NUMBER:

ADDRESS:

COMPLAINTS WITH DURATION:

PREVIOUS HISTORY:

FAMILY HISTORY:

PAST HISTORY:

GENERAL PHYSICAL EXAMINATION

BUILT AND NOURISHMENT:

PALLOR/ICTERUS/CYANOSIS/CLUBBING/EDEMA/LYMPHADENOPATHY

VITAL DATA:

PULSE:

TEMPERATURE:

BP

RESPIRATORY RATE:

SYSTEMIC EXAMINATION

PER ABDOMEN:

RESPIRATORY SYSTEM:

CARDIO VASCULAR SYSTEM:

CENTRAL NERVOUS SYSTEM:

INVESTIGATIONS:

FASTING BLOOD GLUCOSE:

FASTING TRIGLYCERIDES:

CT BRAIN/MRI BRAIN PLAIN:

FINAL OUTCOME:

MASTER CHART

S. No	Age	Gender	Area of residence	Religion	Socioeconomic status	Past medical history	Fasting triglyceride	Fasting glucose	Triglyceride glucose index	Systolic Blood pressure	Diastolic Blood pressure	Stroke recurrence	Poor functional outcome	Neurologic worsening	Mortality
	(years)						(mg/dL)	(mg/dL)		(mm Hg)	(mm Hg)				
1	72	Female	Rural	Hindu	Low	Hypertension	163	98	8.8	169	89	Present	Absent	Present	Absent
2	80	Male	Urban	Hindu	High	Hypertension	167	93	9.1	156	91	Present	Present	Present	Absent
3	84	Male	Urban	Muslim	Middle	Hypertension	175	102	8.3	165	88	Absent	Absent	Absent	Absent
4	62	Female	Rural	Hindu	High	Hypertension	161	98	8.5	172	83	Present	Present	Present	Absent
5	77	Male	Urban	Muslim	Low	Hypertension	177	87	9.1	141	89	Present	Present	Present	Absent

6	81	Female	Rural	Muslim	High	Hyperte nsion	171	96	8.7	172	94	Present	Absent	Present	Absent
7	79	Male	Urban	Muslim	Middle	Hyperte nsion	162	99	9.4	153	79	Absent	Absent	Absent	Present
8	92	Female	Rural	Hindu	Low	Hyperte nsion	176	82	9	125	86	Present	Present	Present	Absent
9	66	Male	Urban	Hindu	High	Hyperte nsion	160	105	9.2	162	101	Absent	Present	Absent	Absent
10	75	Female	Urban	Muslim	High	Hyperte nsion	176	109	8.4	137	78	Present	Absent	Present	Absent
11	77	Male	Rural	Muslim	Low	Hyperte nsion,	182	83	9.3	154	96	Absent	Absent	Absent	Absent
12	65	Female	Rural	Hindu	High	Hyperte nsion	170	88	9	129	82	Present	Absent	Present	Present
13	88	Male	Rural	Muslim	Low	Hyperte nsion,	166	105	9.1	171	85	Absent	Absent	Absent	Absent
14	74	Female	Urban	Hindu	Middle	Hyperte nsion	184	106	8.8	169	91	Present	Absent	Present	Absent

15	78	Male	Rural	Muslim	High	Hyperte nsion	155	94	8.7	153	85	Absent	Present	Absent	Absent
16	83	Male	Urban	Christia n	High	Hyperte nsion,	170	92	9.5	150	94	Absent	Present	Absent	Present
17	71	Male	Urban	Hindu	Low	Hyperte nsion	180	90	8.6	156	81	Present	Absent	Present	Absent
18	72	Female	Rural	Muslim	High	Hyperte nsion	173	97	8.8	125	92	Absent	Present	Absent	Absent
19	69	Female	Urban	Christia n	Low	Hyperte nsion	168	92	9.4	167	92	Present	Absent	Present	Absent
20	95	Male	Rural	Hindu	High	Hyperte nsion	171	87	9.5	142	90	Absent	Absent	Absent	Absent
21	61	Female	Urban	Christia n	Middle	Hyperte nsion	164	100	9.2	135	78	Present	Present	Present	Present
22	79	Female	Urban	Muslim	Low	Hyperte nsion	184	94	8.8	167	96	Absent	Present	Absent	Absent
23	80	Female	Rural	Hindu	High	Hyperte nsion	176	99	8.3	144	83	Present	Absent	Present	Absent

24	84	Male	Urban	Hindu	High	Hyperte nsion	162	94	8.7	147	100	Absent	Absent	Absent	Absent
25	73	Male	Urban	Muslim	Low	Hyperte nsion	172	108	9.3	160	86	Present	Present	Present	Absent
26	79	Female	Rural	Hindu	High	Hyperte nsion	168	89	9	139	92	Absent	Absent	Absent	Absent
27	68	Male	Urban	Muslim	Middle	Hyperte nsion	182	84	9.1	171	86	Absent	Absent	Absent	Present
28	81	Female	Urban	Christia n	Low	Hyperte nsion	158	91	9.4	163	83	Present	Present	Present	Absent
29	77	Male	Rural	Christia n	High	Hyperte nsion	180	108	9	125	79	Present	Absent	Present	Absent
30	84	Female	Rural	Hindu	High	Hyperte nsion	166	103	8.9	158	95	Absent	Present	Absent	Absent
31	76	Female	Urban	Christia n	High	Hyperte nsion	174	92	9.2	146	85	Present	Absent	Present	Absent
32	70	Male	Urban	Hindu	High	Hyperte nsion	166	95	9.6	132	98	Absent	Present	Absent	Absent

33	72	Female	Urban	Hindu	High	Hyperte nsion	180	101	9.3	165	82	Absent	Present	Absent	Present
34	61	Male	Rural	Muslim	Low	Hyperte nsion	169	83	8.3	157	94	Present	Absent	Present	Absent
35	67	Male	Rural	Christia n	Middle	Hyperte nsion	174	106	8.6	150	84	Absent	Present	Absent	Absent
36	74	Female	Urban	Other	High	Hyperte nsion,	168	95	9.4	142	89	Absent	Present	Absent	Absent
37	85	Male	Urban	Muslim	High	Hyperte nsion	181	86	9.3	162	91	Absent	Present	Absent	Absent
38	62	Female	Urban	Hindu	Low	Hyperte nsion	177	102	8.9	139	95	Present	Present	Present	Absent
39	74	Female	Urban	Muslim	High	Hyperte nsion	160	96	8.5	153	97	Present	Absent	Present	Present
40	76	Male	Urban	Christia n	Low	Hyperte nsion	182	97	9.5	162	81	Present	Present	Present	Absent
41	66	Female	Urban	Hindu	High	Hyperte nsion	156	110	8.4	159	90	Absent	Absent	Absent	Absent

42	83	Male	Rural	Hindu	High	Hyperte nsion	178	101	9.4	120	82	Present	Absent	Present	Absent
43	71	Male	Rural	Muslim	High	Hyperte nsion	167	106	8.7	152	100	Absent	Present	Absent	Absent
44	63	Female	Urban	Hindu	Middle	Hyperte nsion	158	107	9.1	166	88	Absent	Absent	Absent	Absent
45	80	Male	Rural	Other	High	Hyperte nsion	183	106	8.6	157	96	Present	Absent	Present	Absent
46	73	Male	Urban	Christia n	Low	Hyperte nsion	172	98	9.4	145	88	Absent	Present	Absent	Present
47	85	Female	Urban	Muslim	High	Hyperte nsion	158	97	9.1	169	90	Absent	Absent	Absent	Absent
48	72	Male	Urban	Christia n	Middle	Hyperte nsion	180	104	8.5	148	84	Absent	Present	Absent	Absent
49	67	Male	Urban	Hindu	High	Hyperte nsion	162	106	9.3	138	86	Present	Absent	Present	Absent
50	64	Female	Rural	Muslim	High	Hyperte nsion	178	92	8.8	171	99	Absent	Present	Absent	Absent

51	75	Male	Urban	Hindu	Middle	Hyperte nsion	167	97	8.8	148	94	Absent	Present	Absent	Absent
52	80	Male	Urban	Christia n	Low	Hyperte nsion	163	100	9.5	167	93	Absent	Present	Absent	Absent
53	66	Female	Urban	Muslim	High	Hyperte nsion	186	90	8.9	124	85	Absent	Absent	Absent	Present
54	89	Male	Urban	Other	High	Hyperte nsion	170	100	8.7	148	97	Absent	Present	Absent	Absent
55	72	Female	Urban	Christia n	High	Hyperte nsion	156	106	8.9	151	94	Present	Present	Present	Absent
56	62	Male	Rural	Muslim	High	Hyperte nsion	178	93	9.2	169	87	Absent	Present	Absent	Absent
57	86	Female	Urban	Hindu	Low	Hyperte nsion	164	96	8.4	149	92	Absent	Present	Present	Absent
58	70	Male	Urban	Hindu	High	Hyperte nsion	170	92	8.9	162	82	Absent	Absent	Absent	Absent
59	72	Female	Rural	Muslim	Middle	Hyperte nsion	182	104	8.5	143	99	Absent	Present	Absent	Absent

60	88	Male	Urban	Hindu	Low	Hyperte nsion	158	108	9.1	148	83	Present	Absent	Present	Absent
61	61	Male	Urban	Christia n	High	Hyperte nsion	177	102	8.7	160	90	Absent	Present	Absent	Absent
62	75	Female	Urban	Hindu	Low	Hyperte nsion	165	89	9.3	155	96	Absent	Absent	Absent	Present
63	61	Male	Rural	Other	Low	Hyperte nsion	160	101	9.6	170	87	Present	Present	Present	Absent
64	90	Male	Urban	Christia n	High	Hyperte nsion	184	90	9.2	164	89	Absent	present	Absent	Absent