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# Function of HDL in Stroke as Well as Comparison of its Levels in Ischemic and Hemorrhagic Stroke

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## ABSTARCT

**This research focuses on HDL cholesterol levels in ischemic and hemorrhagic strokes and their impacts,This study included 100 participants with ischemic and hemorrhagic strokes for comparative analysis,The results indicated distinct differences in HDL levels between the two types of strokes,Elevated HDL levels may correlate with reduced stroke risk, suggesting a protective role.**

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## ABBREVIATIONS

- DM - DIABETES MELLITUS
- FBG - FASTING BLOOD GLUCOSE
- TGL - TRIGLYCERIDES
- HDL - HIGH DENSITY LIPOPROTEIN
- LDL - LOW DENSITY LIPOPROTEIN
- VLDL - VERY LOW DENSITY LIPOPROTEIN
- CVA - CEREBROVASCULAR ACCIDENT
- CAD - CORONARY ARTERY DISEASE
- MCA - MIDDLE CEREBRAL ARTERY
- ACA - ANTERIOR CEREBRAL ARTERY
- ICA - INTERNAL CAROTID ARTERY
- VBA - VERTEBRO BASILAR ARTERY
- SBP - SYSTOLIC BLOOD PRESSURE
- DBP - DIASTOLIC BLOOD PRESSURE

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# CHAPTER ONE
# INTRODUCTION

Stroke is the third leading cause of death in developed countries, following cancer and ischemic heart disease. Additionally, it is the primary cause of physical impairment. Stroke is a prevalent medical crisis. The prevalence is increasing significantly in numerous developing nations due to the adoption of less healthful lifestyles. Treating this condition poses significant challenges, and current therapeutic methods have yet to demonstrate satisfactory effectiveness. Prevention is the optimal choice, but accurately predicting the occurrence of a stroke is difficult, necessitating a thorough examination of risk factors. A stroke, also known as a cerebral vascular accident, refers to the abrupt demise of brain cells caused by insufficient blood flow. The World Health Organization (WHO) provides a clinical definition of stroke as the sudden onset of certain signs and symptoms of localized neurological dysfunction that last for more than 24 hours or result in death, without any evident cause other than vascular origin.[1]

Stroke is the second most prevalent global cause of mortality, following coronary artery disease (CAD). The Global Burden of Disease study determined that the annual incidence of stroke in India was anticipated to be 89 per 100,000 people in 2005. This number is expected to rise to 91 per 100,000 in 2015 and 98 per 100,000 in 2030.2, It is one of India's biggest health issues. Both communicable and non-communicable diseases are a double burden for developing nations like India. The estimated adjusted prevalence rates of stroke in rural regions range from 84 to 262/100,000, while in urban areas they range from 334 to 424/100,0003. 119 to 145/100,000 is the incidence rate, according to recent population research4,5. It has been discovered that 9.2–30% of admissions to neurological wards and 0.9–4.5% of all medical admissions are related to stroke. According to six studies, between 10 and 15 percent of all strokes occur in India in people under the age of forty7. According to WHO forecasts, by 2054, low- and middle-income nations, primarily China and India, would account for 80% of global stroke cases.

Stroke is the abrupt demise of brain cells caused by insufficient blood circulation. It is a primary factor contributing to severe and enduring impairment. The consequences of a stroke are influenced by the severity and location of brain damage, however, the observable symptoms of a stroke do not reliably indicate its root cause or causes. Typical signs of a stroke include the sudden occurrence of one-sided paralysis, vision loss, speech difficulties, memory impairment, decreased cognitive function, coma, or death.[8].

The risk factors encompass Diabetes, Hypertension, Dyslipidemia, Atherosclerosis, advanced age, smoking, and other infrequent causes. There is compelling evidence that altering risk factors will decrease the likelihood of experiencing a stroke. Recent research indicates that a low level of HDL is a significant risk factor for the development of Atherosclerosis, which is the precursor to a cerebrovascular accident. The objective of my study is to investigate whether there is any disparity in the HDL levels between two stroke groups.

## *A. Aim of the Study*

To study serum HDL levels in patient with cerebrovascular accident and to compare the levels of serum HDL between two categories of stroke in patients admitted under General medicine, Katuri Medical College & Hospital.

## *B. Objectives of the Study*

- To study serum HDL level in patients with cerebrovascular accident.
- To compare the levels of serum HDL between two categories of stroke.
- Percentage of site involvement in Ischemic and Hemorrhagic stroke.
- To compare lipid profile between two types of stroke.
- To compare the ages between two types of stroke.
- To compare the Random Blood Sugar between two types of stroke.
- To compare the Blood Pressure between two types of stroke.

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# CHAPTER TWO
# REVIEW OF LITERATURE

*A. Burden of Stroke:*

Stroke is a global health issue. This medical condition is frequently encountered in both industrialized and developing countries, and it significantly contributes to illness, death, and disability. The prevalence increases significantly with age, and in numerous emerging nations & the prevalence is escalating due to the adoption of less healthful behaviors. Approximately 20% of individuals with an acute stroke will succumb to mortality within one month, while a minimum of 50% of those who survive will endure physical impairment. According to estimations from the Indian Council of Medical Research (ICMR), stroke is responsible for 41% of fatalities and 72% of disability adjusted life years (DALYS) among non-communicable diseases (NCDs).9 Cerebral thrombosis is typically the most common type of stroke observed in clinical research, with hemorrhage being the second most frequent form. However, the incidence of these strokes can vary significantly depending on the location. Subarachnoid hemorrhage and cerebral embolism are the subsequent leading causes of both mortality and morbidity.

*B. Classification of Stroke:*

➢ *Stroke is a Diverse Disease with Over 150 Identified Causes. Stroke can be Categorized into Distinct Subtypes:*

- Ischemic -Cerebral ischemia refers to the limited or disrupted blood flow to a specific region of the brain. Arise due to a blockage in a blood vessel that provides blood to the brain. Ischemic stroke can occur as a result of thrombosis, embolism10, systemic hypoperfusion11, or venous thrombosis12. Twelve Cryptogenic strokes, which are strokes of uncertain origin, account for approximately 30-40% of all ischemic strokes.13
- Hemorrhagic - Intracranial hemorrhage refers to the leakage of blood into a specific region of the brain as a result of the rupture of a blood artery or an aberrant vascular structure within the brain. Haemorrhagic strokes can be classified into two types: intracerebral strokes and subarachnoid strokes.

Out of all types of strokes, 88% are caused by a lack of blood flow (Ischemic) and 12% are caused by bleeding (Hemorrhagic).

Among the cases of hemorrhagic strokes, 9% are caused by an intracerebral hemorrhage, while 3% are caused by a subarachnoid hemorrhage.The differentiation between hemorrhagic and ischemic stroke is crucial for the management of stroke and making decisions on treatment.

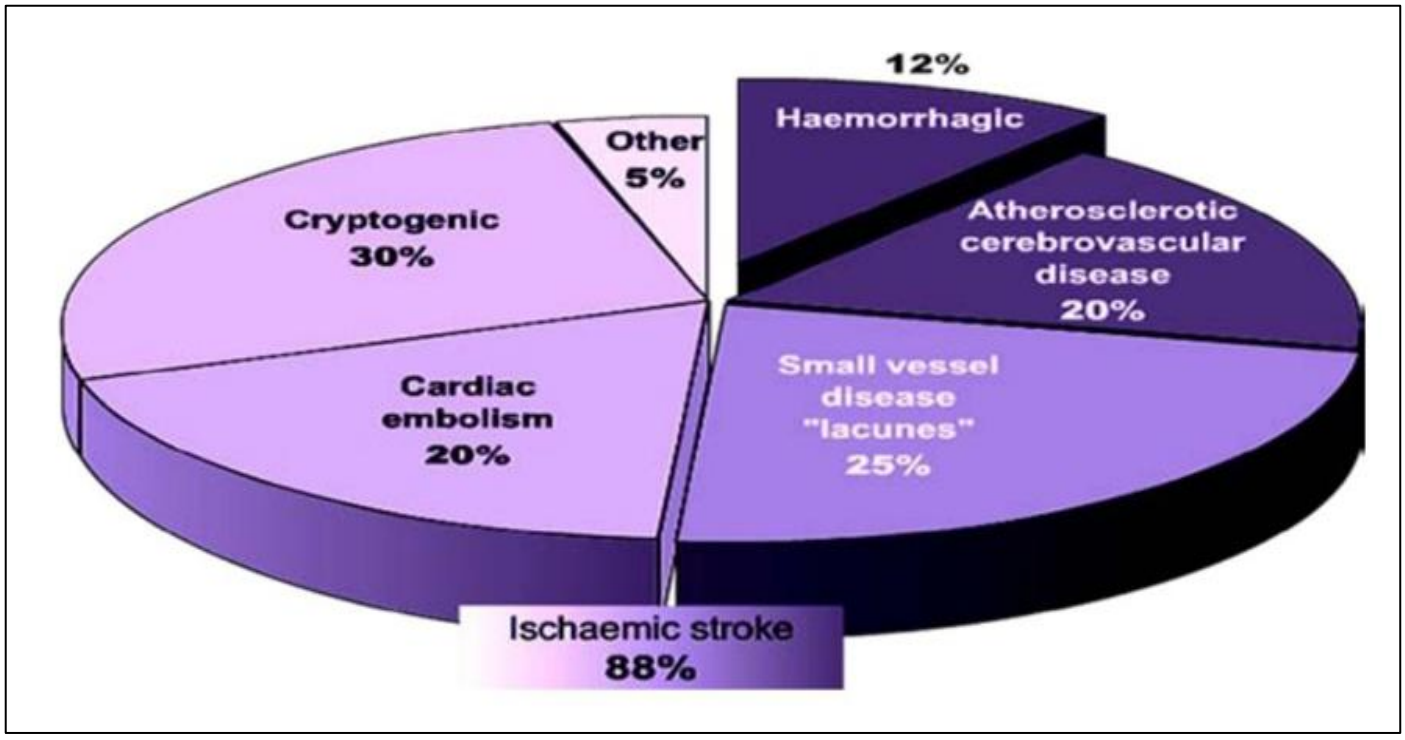


Fig 1: Schematic Representation of Classification of Stroke

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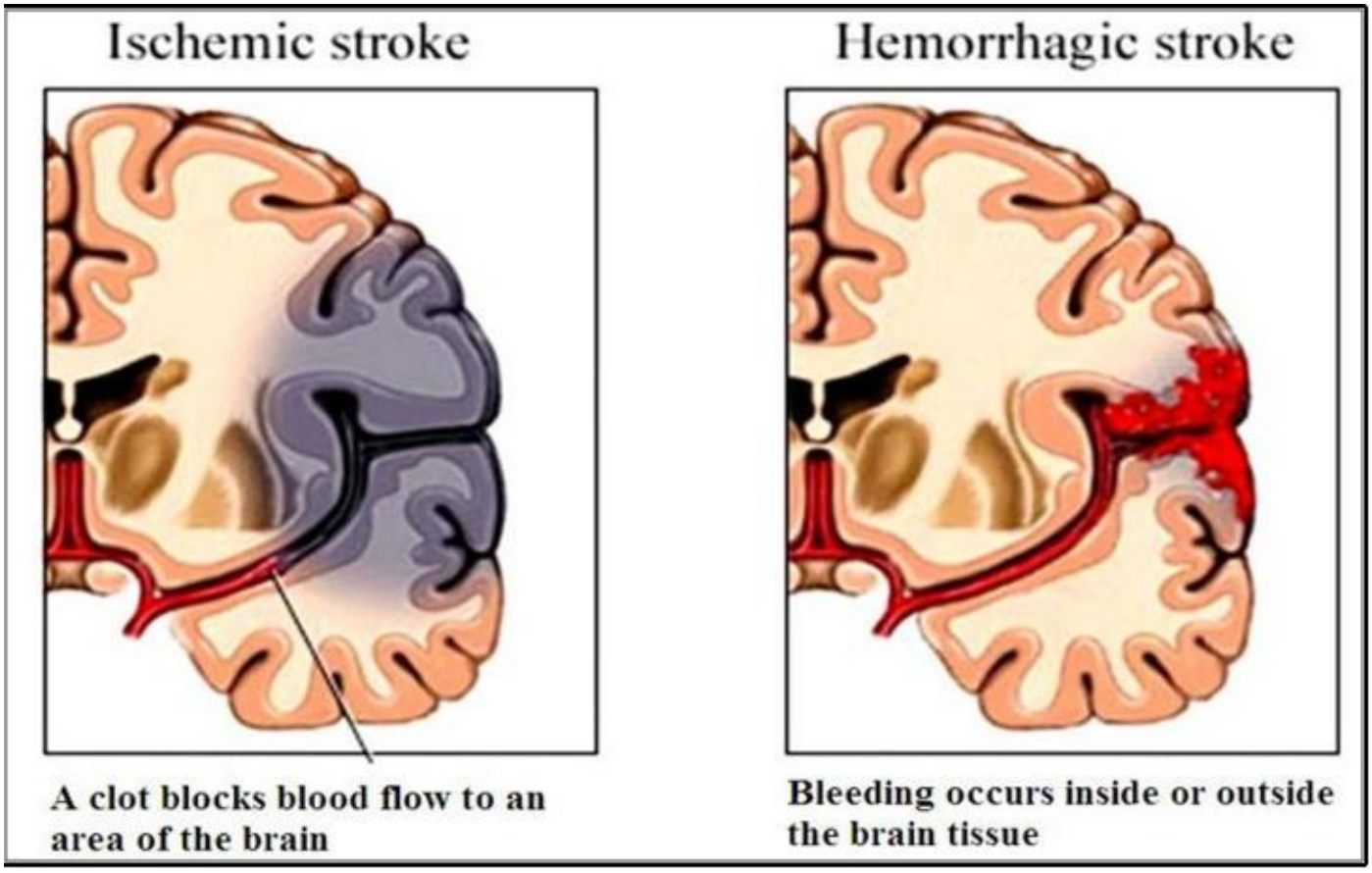


Fig 2: Classification of Stroke

*C. Ischemic Stroke Subtypes:*

Multiple categorization systems exist for categorizing the subtypes of Ischemic strokes, each possessing distinct advantages and disadvantages.

➢ *Stroke Data Bank Subtype (NINDS) Classification*

The National Institute of Neurological Disorders and Stroke (NINDS) classification is based on the Harvard Stroke Registry categorization. The Stroke Data Bank has been acknowledged.[14]

- Infarction of unknown cause
- Infarction with normal angiogram
- Infarction in association with arterial pathology
- Embolism from a cardiac source
- Infarction due to atherosclerosis
- Lacunar infarct
- Parenchymatous or intracerebral hemorrhage
- All other strokes

➢ *Oxford Classification*

The classification of a stroke episode is mostly dependent on the initial symptoms and is determined by the magnitude of these symptoms15.

- Total anterior circulation stroke (TAC)
- Partial anterior circulation stroke (PAC)
- Lacunar stroke (LAC)
- Posterior circulation stroke (POC)

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*D. Risk Factors:*

Epidemiological studies have identified risk variables that occur several years before the onset of a stroke.

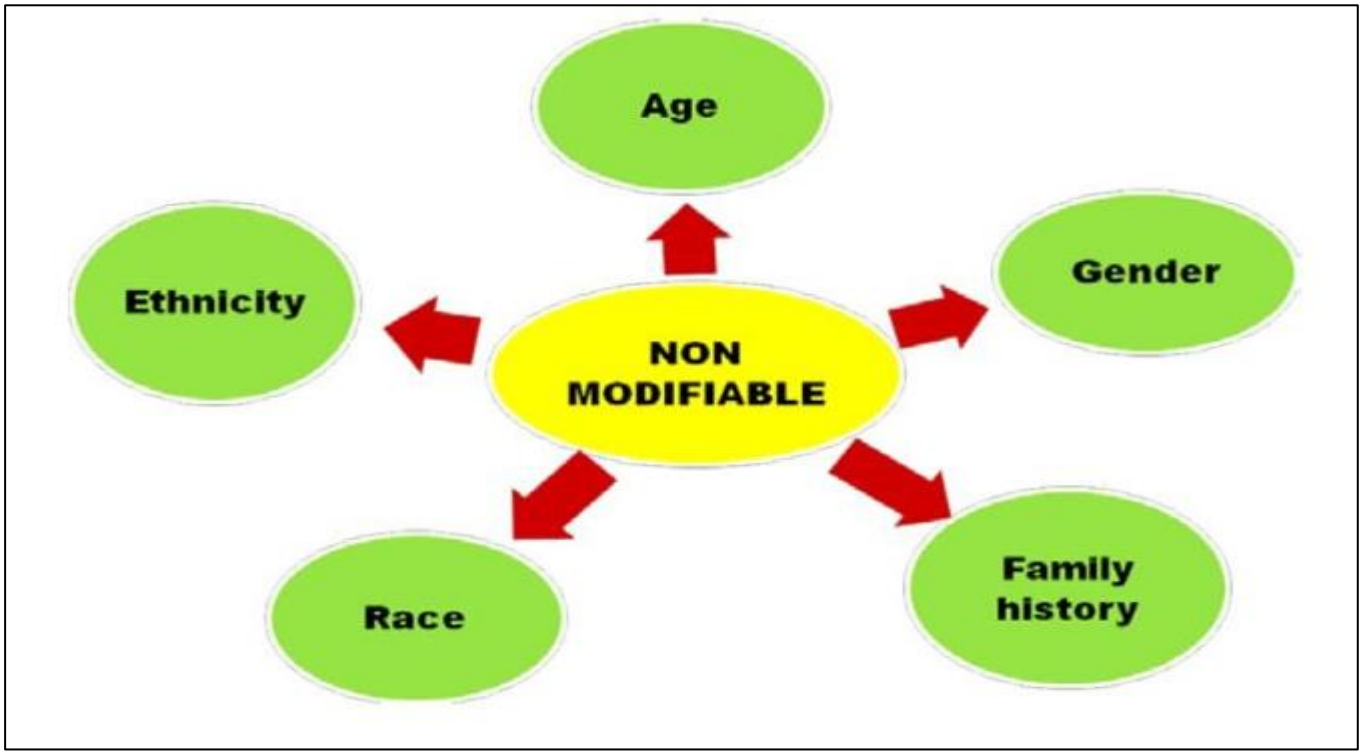


Fig 3: Non-Modifiable Risk Factors

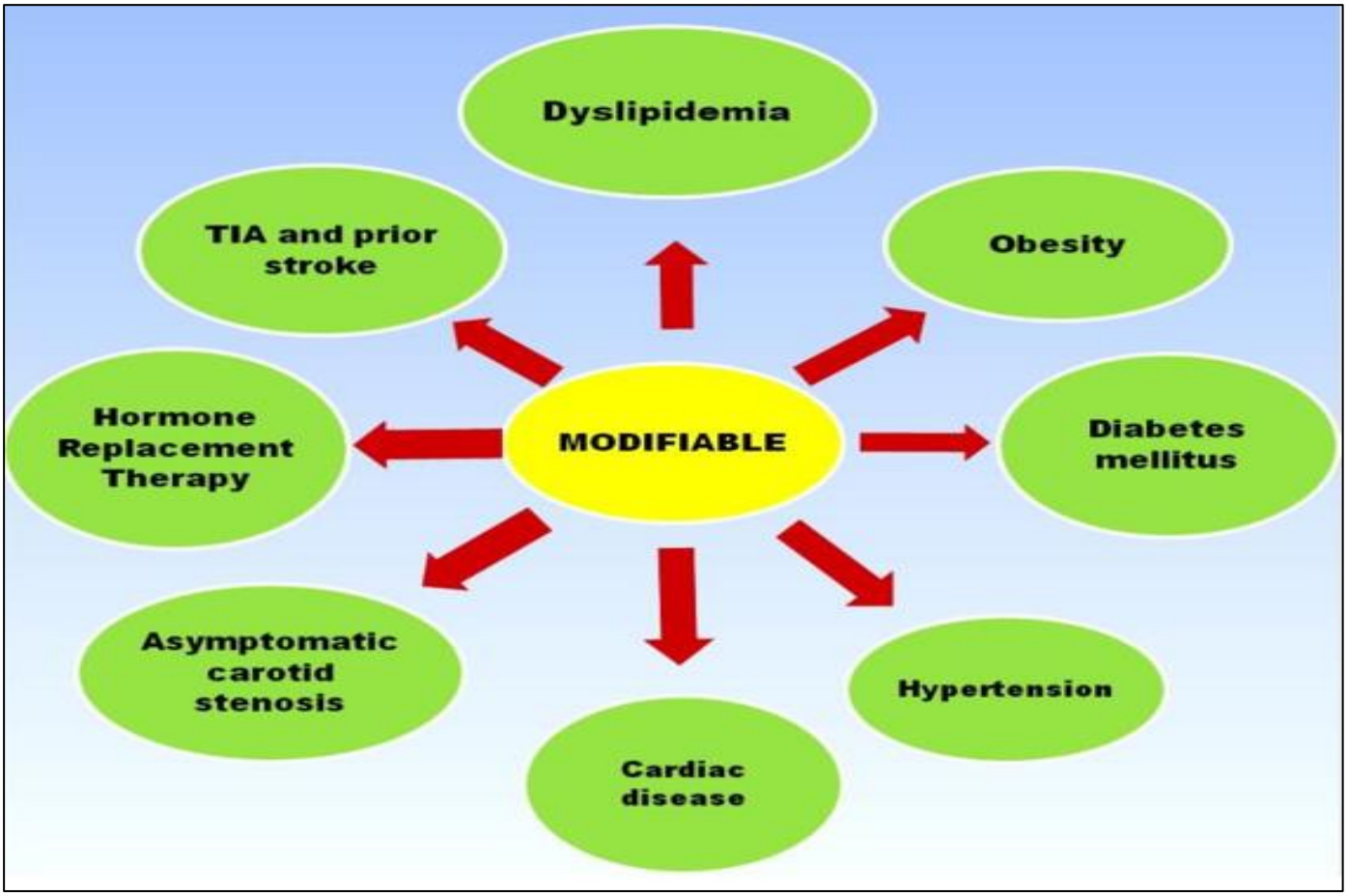


Fig 4: Modifiable Risk Factors

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- *Age:*

Stroke risk is primarily determined by age. The incidence of stroke rises as individuals grow older. The stroke rate in both men and women more than doubles for each consecutive 10-year period beyond the age of 55. The prevalence of atherosclerosis rises with advancing age, hence augmenting the susceptibility to ischemic stroke and myocardial infarction. In India, around 20% of all strokes occur in individuals under the age of 40. The prevalence of stroke in young individuals is increasing due to the significant consequences of early impairment. The incidence of stroke among those aged 80 and above is roughly 27%, while it is 13% for individuals aged 60 to 79.[17]

- *Sex:*

The incidence rates of stroke are 1.25 times higher in males. However, due to women's greater life expectancy, more women die from stroke each year compared to men. The prevalence of stroke among individuals aged 35 to 44 years is greatest among women.However, the heightened risk linked to pregnancy is most notable in the postpartum period.

- *Race:*

African-Americans in the age range of 45 to 55 face a significantly greater likelihood of death from stroke compared to Caucasians. This risk diminishes as individuals get older. During a study conducted in both hospital and community settings, all instances of stroke in Northern Manhattan were examined. The results showed that black patients had a stroke incidence rate that was 2.4 times higher than that of white patients, after adjusting for age.

- *Family History:*

A familial history of stroke, transient ischemic attack (TIA), or myocardial infarction is linked to a 1.4 to 3.3 times higher risk for stroke.[18] The Framingham Study found that the risk of stroke was higher among individuals with a family history of stroke from both their father's and mother's side.

Individuals afflicted with Cerebral autosomal dominant arteriopathy with subcortical infarcts and Leukoencephalopathy (CADASIL), a rare genetic ailment, have a 50% probability of inheriting the disease. CADASIL is caused by a mutation in a gene that results in the impairment of brain blood vessels, leading to the obstruction of blood flow.[19]

- *Diabetes:*

People with diabetes mellitus have a higher chance of experiencing a thromboembolic stroke, regardless of any other cardiovascular risk factors. Multiple epidemiological studies have demonstrated a two to six times higher risk association between diabetes and stroke.[38]. Approximately 40% of ischemic strokes are believed to be caused by diabetes, either on its own or in conjunction with hypertension[20]. This risk may arise from the expedited progression of atherosclerosis as a result of the heightened occurrence of other risk factors, such as central obesity, elevated cholesterol, and hypertension linked to diabetes[21].

- *Hypertension:*

Each increase of 20 mm Hg in systolic blood pressure results in a twofold increase in the mortality rates of stroke and heart disease.[22]. The heightened risk is linked to the expedited progression of atherosclerosis, resulting in a greater occurrence of atherothrombotic events[23]. Longitudinal studies have shown that those with high-normal blood pressure (130–139 mmHg systolic, 85–89 mm Hg diastolic, or both) are twice as likely to develop heart disease and stroke compared to those with blood pressure below 120/80mmHg[24]. Based on the current trial results, however limited, it is indicated that reducing blood pressure by 5-6 mmHg diastolic and 10-12 mmHg systolic can potentially decrease the annual risk of stroke from 7% to 4.8%.

- *Atrial Fibrillation:*

Atrial fibrillation (AF) is the primary cardiac factor contributing to stroke, and it is amenable to treatment. Atrial fibrillation (AF) carries a risk that is three to five times higher than normal25. The occurrence of AF doubles with each subsequent decade of life after the age of 55. The factors that contribute to stroke in atrial fibrillation (AF) include the presence of atherosclerotic plaque, enlargement and impaired function of the left atrium, malfunction of the atrial endothelium, and the development of fibrosis and a hypercoagulable state. Stroke survivors with atrial fibrillation may experience a higher incidence of recurrence and more severe functional impairments26. Individuals diagnosed with atrial fibrillation (AF) experience a 5% year increase in the likelihood of suffering a stroke. This risk is even higher for those with valvular atrial fibrillation[27].

- *Cardiac Disease:*

The main cause of stroke related to heart disease is the formation of thrombotic material on the atrial or ventricular wall or the left heart valves, leading to embolism. Cardio embolism accounts for 20% of all ischemic strokes. The risk of ischemic stroke is higher over the first 5 years following a myocardial infarction, with an estimated incidence of 8.1% over this period. The heightened risk is associated with the magnitude of left ventricular dysfunction, with an 18% elevated risk for every 5% reduction in ejection fraction[28].

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- *Alcohol:*

It has been suggested recently that moderate alcohol use may protect against stroke by increasing blood levels of an endogenous "thrombus dissolver." Blood pressure is known to be the main risk factor for stroke, and it can rise as a result of both binge drinking and excessive drinking. Drinking too much alcohol can also cause atrial fibrillation, which increases the risk of stroke even more. Alcohol consumption between 150 and more than 300 grams is substantially associated with an increased risk of both cardioembolic and cryptogenic stroke. Those who are more susceptible to other contributing variables may get a cardiogenic embolism if they consume more than 40g of alcohol 29.

- *Smoking:*

Smoking escalates the likelihood of stroke by approximately two-fold, exhibiting a distinct correlation with dosage. Former smokers still face a heightened risk of stroke even after quitting smoking.30 It causes a decrease in the ability of the endothelium to dilate blood vessels, which is directly related to the dosage. Additionally, it can also cause spasms in the coronary arteries. Smoking significantly contributes to the development of stroke by causing direct damage to blood vessels and by affecting the flow of blood in the body.

- *Stress:*

Stress alters both the neuroendocrine and autonomic nervous systems.Cortisol, a hormone linked to stress, is released when the sympathetic nervous system and the hypothalamic-pituitary-adrenal axis are activated. This hormonal response contributes to the development of insulin resistance, hypertension, abdominal obesity, and dyslipidemia, all of which raise the risk of cardiovascular disease (CVD).[31].

- *Dyslipidemia:*

The association between cholesterol levels and the risk of stroke lacks definitive data. According to the Asia Pacific Cohort Studies Collaboration, there is a 25% higher risk of ischemic stroke for each 1 mmol/L (38.7 mg/dl) increase in total cholesterol. Additional research also did not demonstrate a correlation between the level of cholesterol in the bloodstream and the risk of stroke[32,33].

- *Obesity:*

Obesity heightens the likelihood of experiencing a stroke and can contribute to elevated blood pressure and cholesterol levels, which in turn can lead to heart disease and diabetes. Following the correction for cardiovascular risk factors, women with a body mass index (BMI) exceeding 27 exhibited a notable rise in the likelihood of experiencing an ischemic stroke. Individuals with a body mass index (BMI) beyond 30 exhibited a relative risk of 1.95 for ischemic stroke. Furthermore, for each incremental unit rise in BMI, there was a corresponding 6% increase in the adjusted relative risk[34]. The Framingham research defines obesity as having a relative weight greater than 30% over average according to the Metropolitan Life chart. This study found that obesity was a significant factor in the occurrence of cerebral infarction in men aged 35 to 64 and women aged 65 to 94.

- *Physical Inactivity:*

It has been shown that middle-aged guys who engage in moderate to high levels of physical exercise are protected against stroke35. Regardless of age, sex, or ethnicity, the Northern Manhattan Stroke Study (NOMASS) discovered that increasing physical activity during leisure time decreased the risk of stroke. The degree of intensity and duration were linked to the decreased risk.

- *Transient Ischemic Attacks:*

The mean incidence of stroke in individuals with transient ischemic attacks (TIAs) is approximately 4%. After accounting for significant cardiovascular risk factors that make someone more likely to have a stroke, transient ischemic attack (TIA) continues to be a separate risk factor for both stroke and heart attack.

- *Infection and Inflammation:*

Several studies have demonstrated a correlation between acute bacterial or viral infections and a heightened susceptibility to ischemic stroke, particularly when these illnesses occur within one week prior to the onset of stroke36. A separate study demonstrated a correlation between persistent infections, particularly bronchitis and periodontal disease, and an elevated risk of ischemic stroke. The understanding of infectious causes for stroke is limited, however it is believed that these mechanisms involve the upregulation of cytokine production and the promotion of blood clot formation37.

- *Hypercoagulability:*

The involvement of hypercoagulability in stroke is a subject of debate. The hypercoagulable state may have a greater significance in younger patients who have experienced a stroke. Arterial hypercoagulability is a contributing factor to cerebrovascular disease. Congenital and acquired disorders, such as polymorphisms in fibrinogen, platelet glycoproteins, and factor XII gene, as well as antiphospholipid antibodies (including anticardiolipin and lupus anticoagulants), are linked to an elevated risk of stroke. Several coagulopathies, such as deficits in protein C and S, mutations in Factor V Leiden, and other defects in clotting

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factors, might result in a higher likelihood of developing venous thrombosis. Nevertheless, there is no significant correlation between various diseases and vascular events such as myocardial infarction (MI) and stroke.

- *Hormone Replacement Therapy:*

Multiple studies have demonstrated a correlation between hormone replacement therapy (HRT) and an elevated risk of stroke, particularly ischemic stroke, which tends to be severe in nature. The likelihood of adverse effects is reduced in younger women who are undergoing hormone replacement therapy (HRT) and are receiving the lowest dosage. However, when administered at a high dosage, the likelihood of adverse effects rises to 62%, primarily attributed to estrogen-induced coagulation problems. Previous hypothesis suggested that hormone replacement therapy could potentially decrease the incidence of stroke. However, current clinical trials have demonstrated no evidence of the benefits of postmenopausal hormone replacement therapy in lowering the occurrence or severity of strokes.39,40

- *Hyperhomocysteinemia:*

Hyperhomocysteinemia has garnered more attention in the last 10 years and is now recognized as a notable independent risk factor for cardiovascular disease, alongside smoking, dyslipidemia, hypertension, and obesity. In order for new risk factors to be significant in disease prevention within a population, they must either contribute significantly to the overall disease burden or have a noticeable effect on the prevalence of the disease in that population when modified.

An increased amount of homocysteine in the blood is a risk factor on its own for nonfatal stroke41. According to the homocysteine studies partnership, a decrease of 25% (about 3 mmol/L) in homocysteine levels is linked to a 19% decrease in the risk of stroke52.Elevated levels of homocysteine are associated with advancing age. Younger men tend to have higher levels of homocysteine than women42.

*E. Pathophysiology of Ischemic Stroke:*

Atherosclerosis of the carotid arteries is a primary contributor to the occurrence of stroke and transient ischemic attacks (TIAs).[43] The fibrous plaque is the most prevalent precursor of atherosclerosis that leads to ischemic lesions. Fibrous plaque is distinguished by the existence of smooth muscle cells and macrophages in the innermost layer of the blood vessel wall, which extends into the central cavity of the vessel. Oxidative alteration of LDL is a necessary condition for the uptake and storage of cholesterol by macrophages. When the quantities of LDL in the plasma are increased, the endothelial cells, monocytes, and macrophages in the artery wall may break down the native LDL particles. This process might result in the depletion of the body's normal antioxidant defenses.

Risk factors including diabetes, insulin resistance, and cigarette smoking might hasten the oxidative modification of small, compact LDL particles 44. In addition to increasing the release of fatty acids and the production of lipo-peroxides, it causes the breakdown of lipid components in the plasma membrane. Often referred to as "foam cells," macrophages are a feature of the arterial fatty streak that phagocytose oxidized LDL. Low-density lipoprotein (LDL) that has been oxidized can cause endothelial dysfunction and has cytotoxic qualities. The pathways activated by oxidized LDL accelerate the development of the fatty stripe into a more complex lesion[45].

- It improves the absorption by macrophages, resulting in an increase in cholesteryl ester content.
- It attracts circulating monocytes through chemotaxis.
- It hinders the movement of tissue macrophages.
- It exhibits cytotoxic properties.
- It has the ability to modify the way genes are expressed in nearby cells, such as by causing the production of MCP-1 and colony stimulating factors.
- This substance has the ability to stimulate an immune response and trigger the production of autoantibodies.
- It has the potential to negatively modify the processes involved in blood clotting.
- It can negatively modify the vasomotor characteristics of coronary arteries.

Cytokines released by mononuclear cells promote cell recruitment and make it easier for macrophages' scavenger receptors to further oxidize and absorb LDL. These scavenger receptors are not inhibited by cholesterol, in contrast to LDL receptors. High levels of cellular cholesterol are continually gathered by macrophages, which causes foam cells to develop. Mitogens, which are also secreted by mononuclear cells, cause smooth muscle cells to proliferate and change fatty streaks into intermediate atherosclerotic plaques.

A necrotic core with a large concentration of lipids outside of cells is a characteristic of the mature lesion.A fibrous crown composed of smooth muscle cells and connective tissue envelops this core. Because the lesion blocks the lumen, blood flow is impeded. The loss of endothelial cells, the presence of a necrotic core with many cholesterol crystals outside the cells, and the substantial calcification are characteristics of the complex lesion. Plaque rupture, which causes a blood clot to develop, is what turns

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this chronic inflammatory disease that affects blood vessel walls into an abrupt and serious clinical event. When an intracranial artery is acutely blocked, blood flow to the particular area of the brain it supplies is reduced.

*F. Neuronal Injury Mechanism:*

➢ *There are Two Distinct Processes by Which Focal Cerebral Infarction Occurs:*

- **Necrotic pathway:** The necrotic route involves the fast breakdown of cellular cytoskeleton, primarily caused by cellular energy depletion.
- **Apoptotic pathways:** An apoptotic pathway characterized by the activation of cellular mechanisms that induce programmed cell death. Lower levels of ischemia, observed in the ischemic penumbra, promote apoptotic cell death, leading to the demise of cells many days to weeks later.

The creation of tiny blood clots, known as microscopic thrombi, is a complex process that hinders the flow of blood in the small blood vessels of the brain, specifically the arterioles and capillaries. The creation of microthrombus is initiated by the activation of damaging vasoactive enzymes, which are secreted by the endothelium, leucocytes, platelets, and other neuronal cells in response to ischemia. One of the first cell types to respond to hypoxia is endothelial cells. Numerous physiological and pharmacological consequences are the outcome of this reaction, which shows itself at the morphological, biochemical, and immunological levels. Endothelial cells change morphologically, becoming larger and acquiring tiny projections on the inside called "microvilli" that resemble fingers. As a result, the capillary channel becomes less open. White blood cells, red blood cells, platelets, and fibrin all contribute to mechanical blockage.[46]

Endothelial cells have a role in regulating the vascular tone of the microcirculation by mediating the effects of vasoactive substances such as endothelin peptides, eicosanoids, and smooth muscle relaxants, including nitric oxide. Activation of endothelial adhesion molecules facilitates the attachment of leukocytes to the endothelium wall, which is a crucial step in the commencement of the inflammatory process.[47]

The excessive activity of some neurotransmitters, especially glutamate and aspartate, has a major impact on the molecular level incidence of hypoxic-ischemic neuronal injury. The exhaustion of cellular energy stores triggers the "excitotoxicity" phenomena. By denying neurons oxygen and glucose, ischemia causes necrosis by impairing mitochondria's capacity to produce ATP. When there is no ATP present, membrane ion pumps stop working and neurons depolarize, which raises intracellular calcium levels.

An energy-dependent process removes glutamate from the extracellular environment, where it is normally stored at the synaptic terminals. Glutamate is released from synaptic terminals in response to cellular depolarization. Glutamate and aspartate levels in the extracellular area significantly increase when energy is being depleted. This results in the activation of calcium channels connected to alpha-amino-3-hydroxy-5-methyl-4-isooxazole propionate (AMPA) and N-methyl-D-aspartate (NMDA) receptors. As a result of the membrane's ongoing depolarization, potassium ions leave and calcium, sodium, and chloride ions enter[48]

Proteases, lipases, and endonucleases are among the damaging enzymes that are activated by intracellular calcium.This activation facilitates the release of cytokines and other mediators, ultimately leading to the disruption of cellular integrity. The inflammatory response to tissue injury is triggered by the fast synthesis of numerous diverse inflammatory mediators, with tumor necrosis factor being a pivotal component. Within 30 minutes of the event and the subsequent restoration of blood flow, leukocytes migrate to the locations that are not receiving blood flow. Leukocytes not only mechanically block small blood vessels but also stimulate the synthesis of vasoactive chemicals such nitric oxide, cytokines, metabolites of arachidonic acid, and oxygen free radicals. The physiologic consequences of these mediators include leukocyte adherence to the endothelium wall, platelet aggregation, enhanced permeability, vasodilation, vasoconstriction, and immunoregulation.

Free radicals are generated through the breakdown of membrane lipids and the malfunctioning of mitochondria, resulting in the destructive catalysis of membranes and potential harm to other essential cellular functions.

*G. Pathophysiology of Hemorrhagic Stroke:*

Bleeding directly into the brain parenchyma is a symptom of haemorrhagic stroke. The underlying mechanism is generally thought to be leakage from tiny intracerebral arteries that are impaired by chronic hypertension.[49]

Stroke can result from hypertension in a variety of ways. The endothelium and smooth muscle activity inside intracerebral arteries will be significantly altered by elevated intraluminal pressure. Increased blood-brain barrier permeability may result from increased endothelial stress, resulting in the occurrence of localized or multifocal brain edema. Smooth muscle cell and endothelial degeneration increase the likelihood of intracerebral hemorrhages.[50]

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Intracerebral hemorrhage (ICH) is characterized by three separate phases: (1) the initial bleeding, (2) the enlargement of the blood clot, and (3) the swelling around the clot. The primary bleeding occurs due to the rupture of cerebral arteries, which is influenced by the risk factors stated earlier. The illness outcome is mostly determined by the last two phases of development. Hematoma enlargement, which occurs many hours after the initial appearance of symptoms, leads to an elevation in intracranial pressure (ICP) that damages the local tissue and the blood-brain barrier. In addition, when venous outflow is blocked, it triggers the release of tissue thromboplastin, leading to a localized coagulation disorder. Hematoma growth is linked to hyperglycemia, hypertension, and anticoagulation in more than one-third of patients. The quantity of the bleeding at the beginning and the speed at which the blood clot grows are crucial factors in forecasting the worsening of neurological condition. Hematoma size larger than 30cc is strongly correlated with significantly higher fatality rates. After the hematoma expands, cerebral edema develops due to inflammation and damage to the blood-brain barrier. The main cause of neurological impairment is the edema that occurs around the hematoma, which grows gradually over several days after the original injury.[51,52]

Up to 40% of cases of Intracerebral Hemorrhage (ICH) involve the bleeding spreading into the cerebral ventricles, resulting in intraventricular hemorrhage (IVH). This is linked to a condition called acute obstructive hydrocephalus, which significantly worsens the prognosis. Intracranial hypertension and the associated swelling can cause disruption or compression of nearby brain tissue, resulting in neurological impairment. Significant movement of brain tissue can lead to an increase in pressure inside the skull, known as intracranial pressure (ICP), which can result in life-threatening herniation syndromes.[53,54]

Intracerebral hemorrhage (ICH) occurs when blood rapidly accumulates within the brain tissue, causing a disturbance in the normal structure and a rise in local pressure. The major damage caused by hematoma expansion is primarily due to mechanical damage resulting from the mass effect. This damage happens from minutes to hours after the bleeding starts, depending on the dynamic of expansion. Secondary damage is mostly caused by the presence of blood within the brain tissue and may be influenced by factors such as the initial size of the blood clot, the age of the patient, or the volume of the brain's ventricles54,55,56. It can happen through various pathogenic pathways that run parallel to each other, including: (1) the toxicity of blood cells; (2) excessive metabolism; (3) overstimulation of nerve cells; (4) the spread of depression-like activity in the brain; and (5) the presence of oxidative stress and inflammation57,58,59. In the end, this process of pathogenesis results in permanent damage to the various components of the neurovascular unit, including gray and white matter. This is then followed by the breakdown of the blood-brain barrier and the development of severe brain swelling, leading to extensive death of brain cells. While local inflammatory mediators produced in response to brain death or injury can worsen the damage caused by intracerebral hemorrhage (secondary injury), the presence of inflammatory cells, such as microglia/macrophages, is crucial for the elimination and clearance of cellular debris from the hematoma, which is the ongoing source of inflammation. Swiftly eliminating impaired tissue is crucial for minimizing the duration of harmful disease progression, therefore enabling expedited and more effective recuperation.60,61.

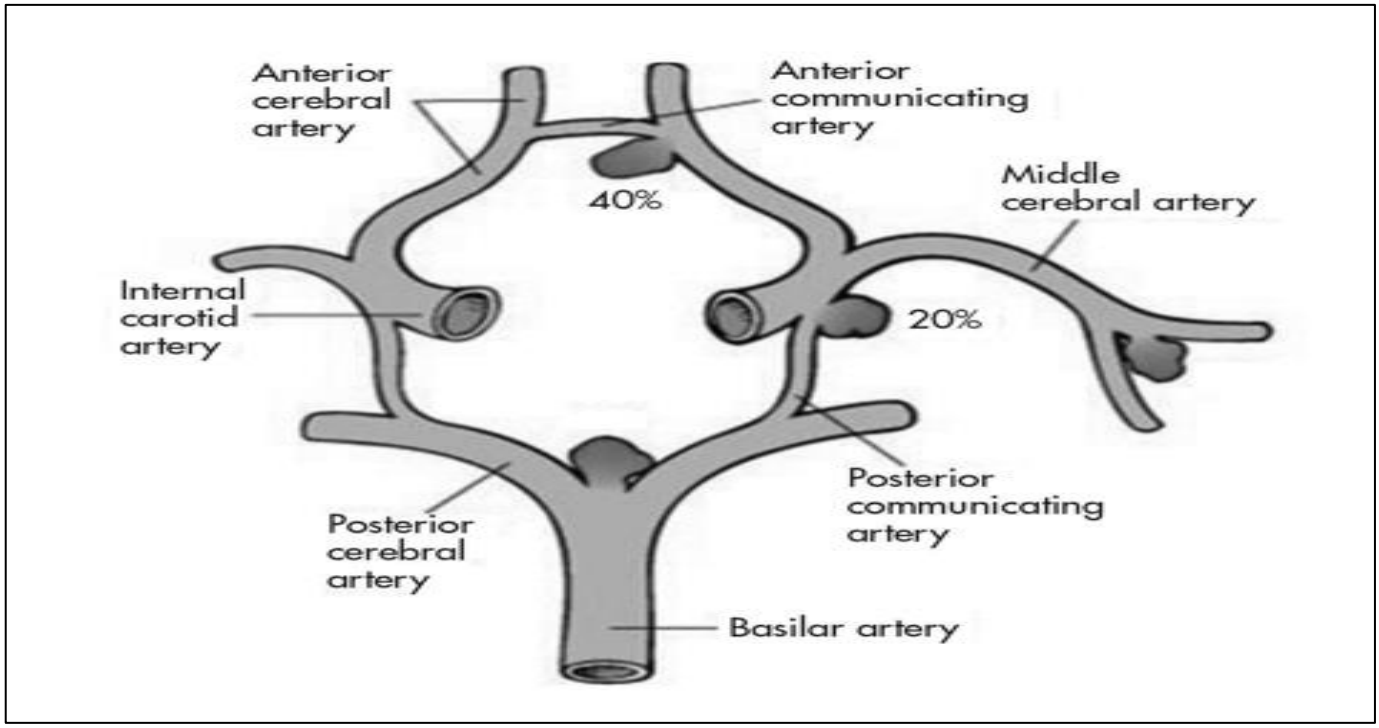


Fig 5: Site of Hemorrhage in Stroke

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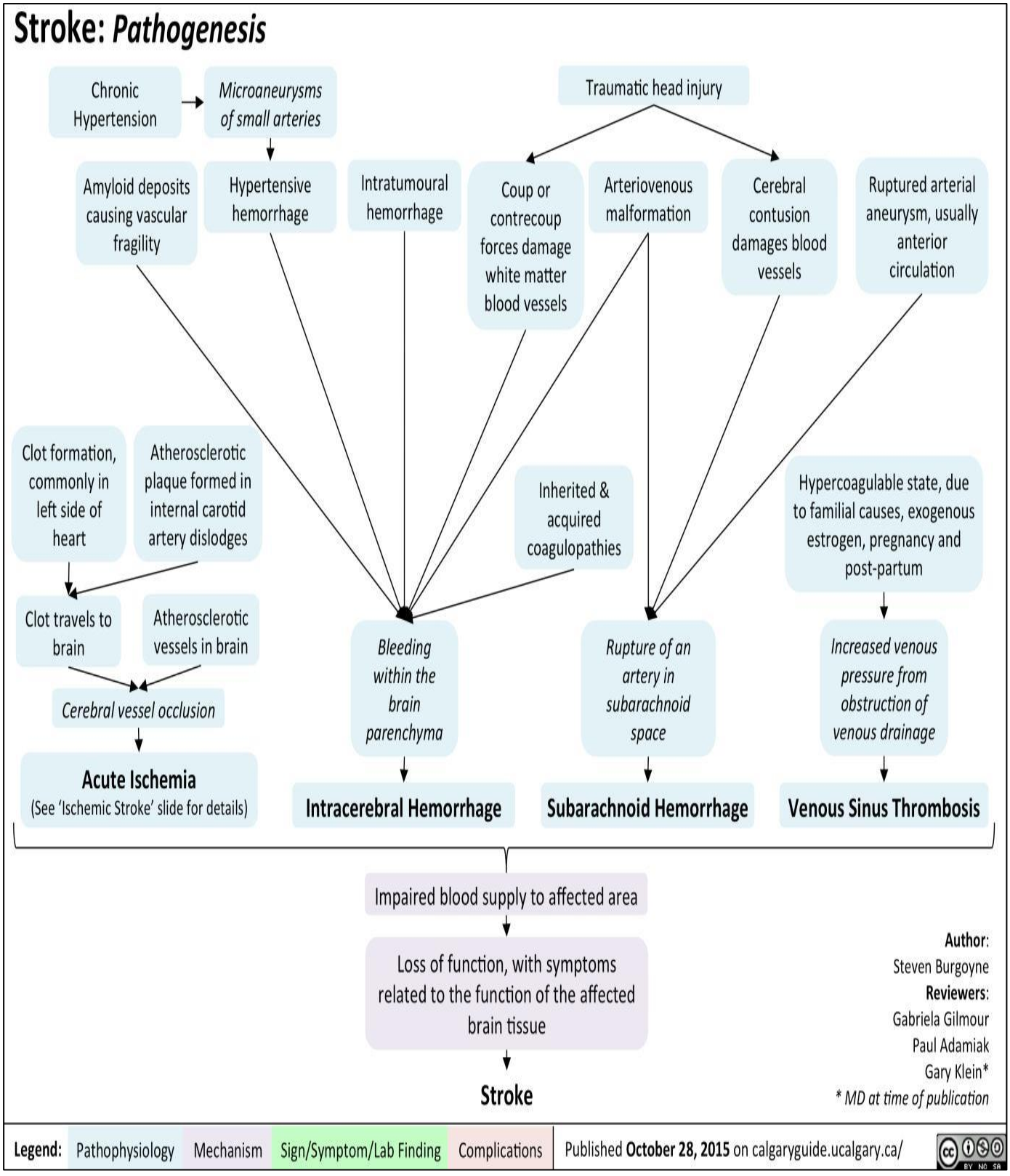


Fig 6: Stroke Pathogenesis

*H. Symptoms and Signs:*

The clinical symptoms of stroke exhibit significant variability due to the intricate structure of the brain and its blood vessels. The impact of stroke varies among individuals. The clinical manifestations vary according on the kind, location, and extent of the brain tissue injury. Neurological symptoms will appear quickly when neurons do not have glycogen, resulting in rapid energy failure[62]. There are five prominent indicators of stroke, with the majority of patients experiencing two or more of these characteristics:

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- Hemiparesis is a condition characterized by the partial loss of sensory and/or motor function on one side of the body, affecting areas such as the arm, face, and leg. It is observed in approximately 85% of ischemic stroke patients.
- Experience challenges in verbal communication or comprehending spoken language.
- There is a sudden disruption in the ability to see clearly in one or both eyes.
- Experiencing abrupt challenges in walking, dizziness, and a lack of balance or coordination.
- Severe headache of unknown origin.

➢ *D.D's of Stroke:*

- Metabolic brain injury
- Cervical and craniocerebral trauma
- Encephalitis and meningitis
- The intracranial mass
- Subdural hemorrhage
- Seizures with enduring neurological symptoms
- Persistent neurological symptoms associated with migraine
- Hyperglycemia (hyperoosmolar coma that is not ketotic)
- Hypoglycemia
- Ischemia after cardiac arrest
- Narcotic or drug overdose

*I. Diagnosis:*

The assessment of clinical symptoms and the application of neuroimaging methods are necessary for the diagnosis of stroke. Using time-sensitive treatments, such thrombolysis, requires prompt evaluation. Chronic ailments such as diabetes mellitus, hypertension, cardiovascular diseases, and other neurological problems are among the many medical histories and current occurrences that are meticulously documented. Following the process of obtaining the patient's medical history, the subsequent diagnostic measures involve measuring the blood pressure in both arms, assessing the pulse, doing a neurological examination, and evaluating the state of consciousness. Various further tests are employed to validate the diagnosis.

After identifying a stroke, it is essential to do a brain imaging scan to ascertain whether the stroke was caused by ischemia or hemorrhage. The primary diagnostic tool for identifying stroke is Computerized Tomography (CT scan). It can provide a realistic representation of both hemorrhagic and ischemic strokes, regardless of the severity of the attack or symptom.

Transient ischemic attacks (TIAs) and strokes are commonly diagnosed by magnetic resonance imaging (MRI), a more thorough diagnostic method than computed tomography (CT) scans.Additional tests might include a transcranial or carotid Doppler ultrasonography, an electrocardiogram (ECG), a random blood sugar test, a serum cholesterol test, and more.

*J. Evaluating the Severity of Stroke:*

The NIHSS score and the modified Rankin scale were used to evaluate the severity of the stroke.

➢ *NIHSS Score:*

A popular and reliable tool for determining the clinical severity of a stroke is the National Institutes of Health Stroke Scale (NIHSS)63. Early on in a stroke, the NIHSS score is a very good predictor of the final clinical outcome and has been used to assess a person's eligibility for acute stroke therapy trials, including thrombolysis.64. The utilization of the NIHSS score in clinical trials or clinical decision-making methods may induce bias against patients with right-hemisphere stroke.[65]

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Providence | Health System

# NIH STROKE SCALE

PSVMC - Providence St. Vincent Medical Center
PMH - Providence Milwaukie Hospital
PPMC - Providence Portland Medical Center

PATIENT IMPRINT

| Category | Score/Description | | Date/Time Initials | Date/Time Initials | Date/Time Initials | Date/Time Initials | Date/Time Initials |
|---|---|---|---|---|---|---|---|
| 1a. **Level of Consciousness** (Alert, drowsy, etc.) | 0 = Alert<br>1 = Drowsy<br>2 = Stuporous<br>3 = Coma | | | | | | |
| 1b. **LOC Questions** (Month, age) | 0 = Answers both correctly<br>1 = Answers one correctly<br>2 = Incorrect | | | | | | |
| 1c. **LOC Commands** (Open/close eyes, make fist/let go) | 0 = Obeys both correctly<br>1 = Obeys one correctly<br>2 = Incorrect | | | | | | |
| 2. **Best Gaze** (Eyes open - patient follows examiner's finger or face) | 0 = Normal<br>1 = Partial gaze palsy<br>2 = Forced deviation | | | | | | |
| 3. **Visual Fields** (Introduce visual stimulus/threat to pt's visual field quadrants) | 0 = No visual loss<br>1 = Partial Hemianopia<br>2 = Complete Hemianopia<br>3 = Bilateral Hemianopia (Blind) | | | | | | |
| 4. **Facial Paresis** (Show teeth, raise eyebrows and squeeze eyes shut) | 0 = Normal<br>1 = Minor<br>2 = Partial<br>3 = Complete | | | | | | |
| 5a. **Motor Arm - Left**<br>5b. **Motor Arm - Right** (Elevate arm to 90° if patient is sitting, 45° if supine) | 0 = No drift<br>1 = Drift<br>2 = Can't resist gravity<br>3 = No effort against gravity<br>4 = No movement<br>X = Untestable (Joint fusion or limb amp) | Left | | | | | |
| | | Right | | | | | |
| 6a. **Motor Leg - Left**<br>6b. **Motor Leg - Right** (Elevate leg 30° with patient supine) | 0 = No drift<br>1 = Drift<br>2 = Can't resist gravity<br>3 = No effort against gravity<br>4 = No movement<br>X = Untestable (Joint fusion or limb amp) | Left | | | | | |
| | | Right | | | | | |
| 7. **Limb Ataxia** (Finger-nose, heel down shin) | 0 = No ataxia<br>1 = Present in one limb<br>2 = Present in two limbs | | | | | | |
| 8. **Sensory** (Pin prick to face, arm, trunk, and leg - compare side to side) | 0 = Normal<br>1 = Partial loss<br>2 = Severe loss | | | | | | |
| 9. **Best Language** (Name item, describe a picture and read sentences) | 0 = No aphasia<br>1 = Mild to moderate aphasia<br>2 = Severe aphasia<br>3 = Mute | | | | | | |
| 10. **Dysarthria** (Evaluate speech clarity by patient repeating listed words) | 0 = Normal articulation<br>1 = Mild to moderate slurring of words<br>2 = Near to unintelligable or worse<br>X = Intubated or other physical barrier | | | | | | |
| 11. **Extinction and Inattention** (Use information from prior testing to identify neglect or double simultaneous stimuli testing) | 0 = No neglect<br>1 = Partial neglect<br>2 = Complete neglect | | | | | | |
| | TOTAL SCORE | | | | | | |

| INITIAL | SIGNATURE | INITIAL | SIGNATURE | INITIAL | SIGNATURE |
|---|---|---|---|---|---|
| | | | | | |
| | | | | | |

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Fig 7: NIH Stroke Scale

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➢ *Modified Rankin Scale:*

The Modified Rankin scale is utilized to assess the clinical outcome following a stroke. The scale ranges from 0 to 6, representing the spectrum of health status from optimal well-being without any symptoms to the state of mortality.

| Level | Description |
|---|---|
| 0 | No symptoms |
| 1 | No significant disability, despite symptoms; able to perform all usual duties and activities |
| 2 | Slight disability; unable to perform all previous activities but able to look after own affairs without assistance |
| 3 | Moderate disability; requires some help, but able to walk without assistance |
| 4 | Moderately severe disability; unable to walk without assistance and unable to attend to own bodily needs without assistance |
| 5 | Severe disability; bedridden, incontinent and requires nursing care and attention |

Fig 8: Modified Rankin Scale

➢ *Lipoproteins*

Lipoproteins are categorized into distinct groups:

- Chylomicrons
- VLDL (Pre Beta - lipoprotein) IDL (Broad beta)
- LDL (Beta lipoprotein) HDL (Alpha lipoprotein)

✓ *HDL:*

Elevated levels of HDL particles are closely linked to reduced buildup of atherosclerosis in arterial walls. This is significant because atherosclerosis ultimately leads to abrupt plaque ruptures, cardiovascular disease, stroke, and other vascular disorders. HDL particles are commonly known as "good cholesterol" due to their ability to remove fat molecules from arterial walls, decrease macrophage buildup, and thereby aid in the prevention or reversal of atherosclerosis.

✓ *HDL Metabolism:*

- HDL particles are synthesized in the bloodstream through the incorporation of lipids into ApoA-1.
- ApoA-1 is produced in both the liver and gut and makes up approximately 70% of the apoproteins found in HDL.
- **The process by which high-density lipoproteins (HDL) remove unesterified cholesterol from the bloodstream:** The nascent HDL is discoid in shape, rich in PLs (largely phosphatidyl choline) ,Comprises apoproteins A, C, and E, and undergoes rapid conversion into spherical particles after cholesterol accumulation.
- **Esterification of Cholesterol:** HDL takes up cholesterol, which is then bound to the HDL disc by LCAT. Apo A-1 activates LCAT.LCAT catalyzes the transfer of fatty acids from carbon 2 of phosphatidyl choline to cholesterol, resulting in the formation of cholesteryl ester (which is stored in the core of HDL) and lyso-phosphatidyl choline, which binds to albumin. The progressive buildup of cholesterol ester (CE) causes the transformation of newly formed disc-shaped high-density lipoprotein (HDL) into spherical particles. HDL transports cholesterol ester (CE) to VLDL/LDL in return for triglycerides (TAG). The process is facilitated through CETP, and the liver ultimately absorbs the CEs.

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*K. Hdl's Anti-Atherogenic Role:*

Historically, the involvement of HDL in reverse cholesterol transfer has been attributed to its anti-atherosclerotic effect. Pre β HDL, which are small HDL precursors, extract free cholesterol from cell membranes in the tissue fluid. Upon encountering the pre β particle, the unbound cholesterol undergoes esterification facilitated by LCAT (Lecithin cholesterol acyl transferase), resulting in increased hydrophobicity. As the cholesterol ester moves into the particle's center, a gradient is created that makes it easier for free cholesterol to move from the cell membrane to the pre-β HDL. As a result of continuous cholesterol absorption, larger α-migrating HDL3 particles accumulate and migrate from the tissue fluid into the plasma66Hepatic and lipoprotein lipase, as well as CETP (cholesterol ester transfer protein) and PLTP (phospholipid transfer protein), aid in the remodeling of HDL in the circulation, as well as the transfer of apolipoproteins from other lipoproteins. This process leads to the formation of two main categories of HDL, namely HDL2 and HDL3.

- Those that possess apoA1 but lack apoA2
- Those that include both.

The destiny of cholesterol in the HDL pool involves either its uptake by the liver for recycling or its transfer back to VLDL or LDL by CETP.

*L. Hdl's Anti-Oxidative Action: Two Theories*

There are two hypothesis that overlap: 1) Direct Metabolism and 2) Transfer theories.

The Direct Metabolism theory states that HDL interacts with LDL in the subintimal space and, with PON-1's help, works to prevent LDL oxidation by dissolving lipid hydroperoxides.There is proof that HDL cannot remove the physiologically active lipids in oxidized LDL without PON-1.[67]

According to Transfer theory, HDL served as a safeguard for LDL by preventing lipid peroxidation. It accomplished this by functioning as a storage system for lipid peroxides that were produced on LDL.

According to Transfer hypothesis, HDL served as a safeguard for LDL by preventing lipid peroxidation. It accomplished this by storing lipid peroxides produced on LDL and interrupting the process of lipid peroxide spread. When phospholipid hydroperoxides (PLHP) are connected to HDL, they can be broken down by HDL-associated enzymes such as PON-1, PAF-AH, and LCAT. This can result in the production of non- atherogenic substances. Alternatively, the PLHP can be transferred to cholesterol by LCAT, leading to the formation of oxidized cholesteryl esters within HDL. The oxidized cholesterol esters are then transported by HDL to the liver for excretion63,64,65. Therefore, the amount of HDL particles that carry the preventive enzymes PON-1 and PAF-AH may have a greater positive effect than the precise levels of HDL cholesterol in the blood.[68,69]

*M. Hdl's Anti-Thrombotic and Endothelial Activities*

High-density lipoprotein (HDL) cholesterol is a material that aids in the removal of cholesterol from cells and helps stop fatty deposits from forming in the arteries. A number of other endothelial and antithrombotic properties of HDL help to protect the cardiovascular system. By boosting the expression of endothelial NO synthase (eNOS), preserving the lipid composition in caveolae where eNOS is found next to signaling molecules, and activating eNOS via a kinase cascade initiated by the high-affinity HDL receptor scavenger receptor class B type 1 (SR-B1), HDL promotes the synthesis of nitric oxide (NO). Through signaling that is initiated by SR-B1, HDL also protects endothelial cells from programmed cell death and promotes their proliferation and motility. Multiple ways by which HDL protects against arterial and venous thrombosis and shows antithrombotic characteristics have been shown. Activating the synthesis of prostacyclin is one of these steps. HDL's capacity to lower tissue and factor selectin expression, prevent thrombin production via the protein C pathway, and either directly or indirectly lower platelet activation may further contribute to its antithrombotic actions.[73,7]

*N. The Role of Hdl In Reverse Cholestrol Transport:-*

The transport protein ABCA1 mediates the efflux of cholesterol from peripheral cells. The liver's absorption of CE is facilitated via a cell surface receptor called SR-B1 (scavenger receptor class B type 1), which specifically binds to HDL.

- Hepatic lipase catalyzes the hydrolysis of cholesteryl esters (CE) in a manner similar to triglycerides (TAG) and phospholipids (PLs). Additionally, it turns high-density lipoprotein 2 (HDL2) into high-density lipoprotein 3 (HDL3).
- Therefore, cholesterol is transported from the tissues to the liver and subsequently eliminated through bile in the form of bile salts. This refers to the process of transporting cholesterol from peripheral tissues back to the liver for elimination.

➢ *Subfractions of HDL:*

There have been five distinct subdivisions of HDL that have been recognized. The types, ranked in order of effectiveness in cholesterol removal from largest to smallest, are 2a, 2b, 3a, 3b, and 3c.

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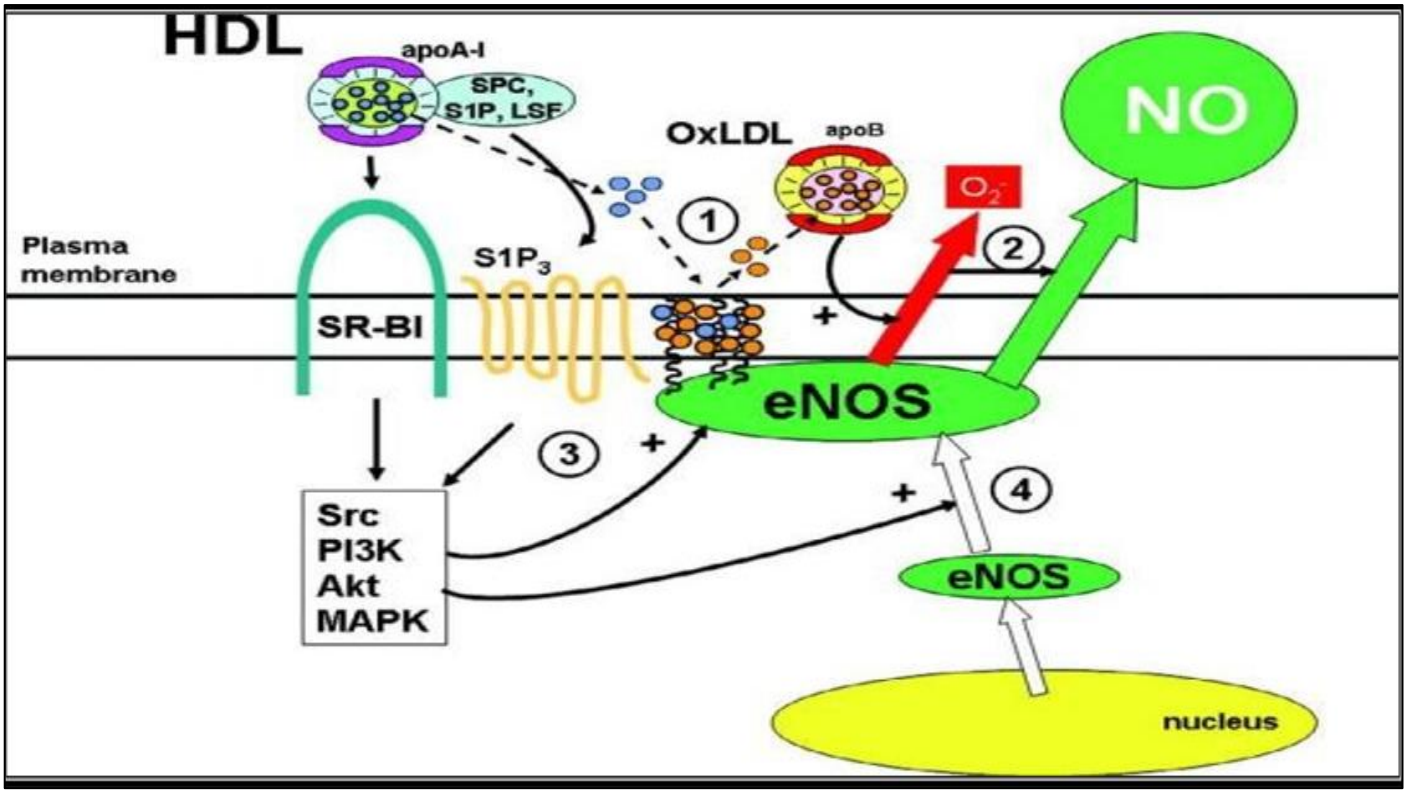


Fig 9: Anti-Thrombotic Function of HDL

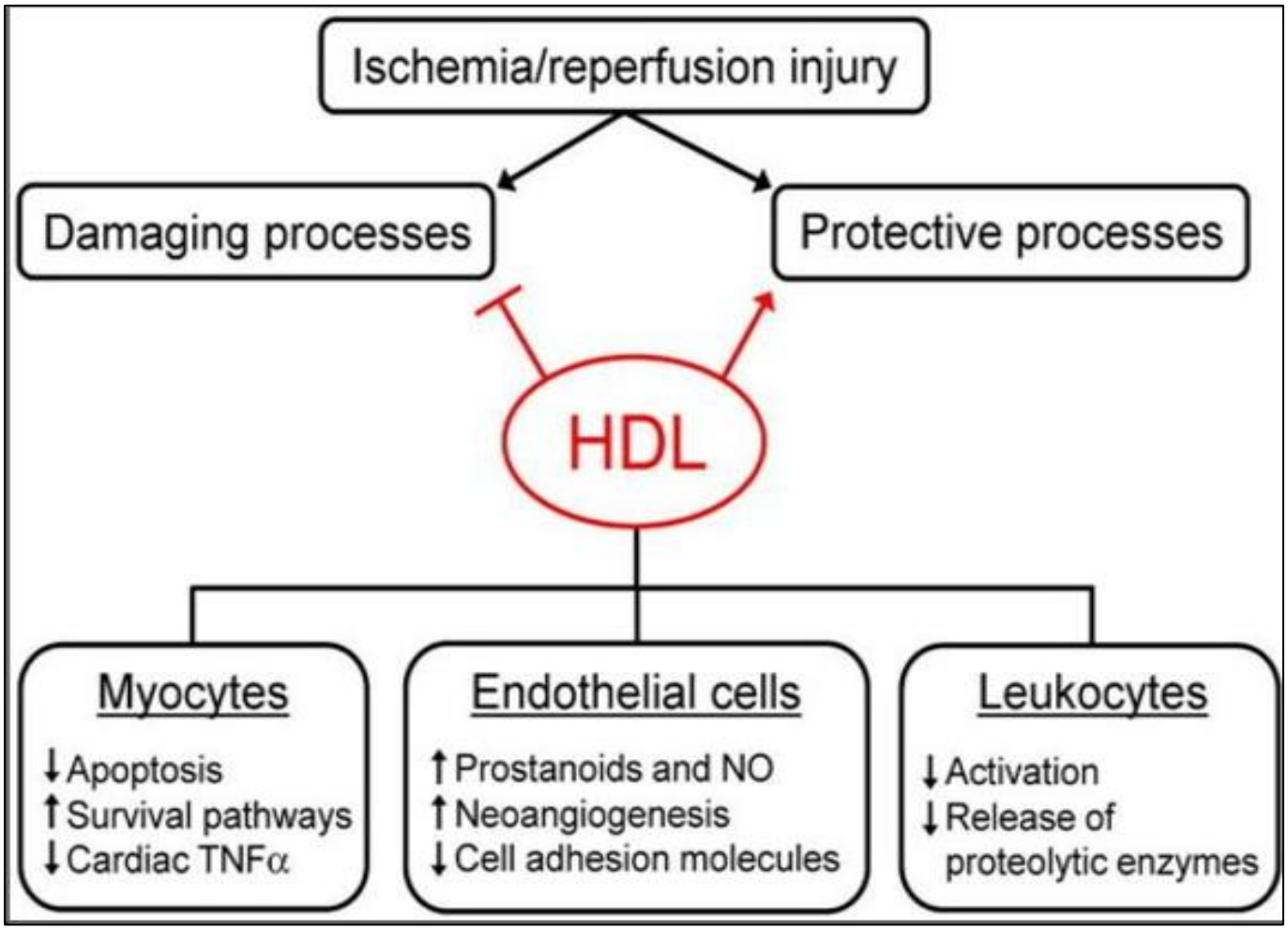


Fig 10: Schematic Representation of Functions FHDL

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*O. HDL and Stroke*

Significantly lower rates of nonfatal stroke were associated with elevated HDL cholesterol levels.75 Both smokers and non-smokers have a negative correlation between their HDL cholesterol levels and their risk of ischemic stroke, a nonfatal stroke. Men who already have coronary heart disease (CHD) are more likely to have this relationship, which is especially important for hypertensive men. HDL levels below 40 mg/dl are more likely to be associated with thromboembolic stroke than levels over 60 mg/dl. [75,76]

An increase in high-density lipoprotein cholesterol (HDL-C) was associated with a 50% lower risk of nonfatal stroke in people with high blood pressure. High density lipoprotein-cholesterol (HDL-C) levels have been linked negatively to stroke in a number of studies. For every 10 mg/dl rise in HDL-C, the risk of ischemic stroke decreased by 11% to 15%. There was an inverse relationship between plaque area and HDL3. People with tiny and medium-sized HDL particles had a lower incidence of ischemic stroke, including lacunar infarcts and ICH. HDL is made up of two main fractions: HDL3, which is dense, and HDL2, which is larger and less dense.Through its effect on the artery endothelium, HDL3, in excess of HDL2, seems to prevent LDL oxidation and protect against atherosclerosis. Total cholesterol levels and the risk of hemorrhagic stroke are inversely correlated, according to several studies. Hemorrhagic stroke incidence seems to be negatively correlated with elevated LDL cholesterol levels. There seems to be a favorable correlation between HDL cholesterol levels and the risk of intracerebral hemorrhage[77]. Several studies indicate that there is no substantial correlation between overall cholesterol or HDL-C levels and the risk of hemorrhagic stroke. Women who had LDL-C levels below 70 mg/dL and low triglyceride levels had a higher chance of experiencing a hemorrhagic stroke.[78]

➢ *Functions of HDL:*

- Cholesterol Transport Reversal.
- Activity that reduces inflammation.
- Antioxidant activity.
- Activity that prevents or inhibits apoptosis.
- Restoration of the endothelium.
- Thrombolytic activity inhibition.
- Activity against infectious agents.

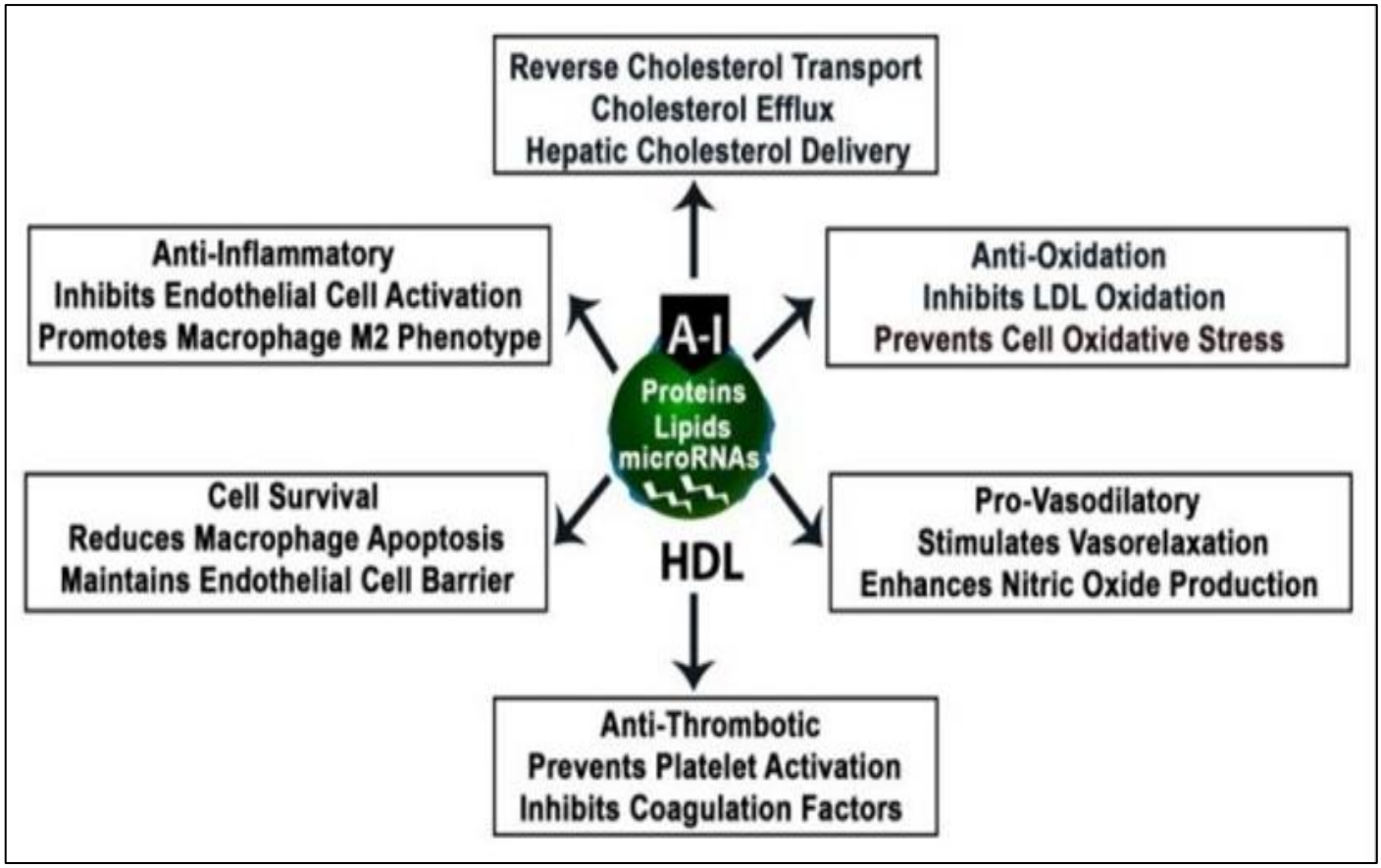


Fig 11: Functions of HDL

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*P. Exercise and Diet:*

Specific modifications in dietary and physical activity patterns can potentially enhance the elevation of HDL levels:

- Reduced consumption of carbs.
- Aerobic exercise.
- Weight reduction.
- Magnesium supplements elevate high-density lipoprotein cholesterol (HDL-C).
- Incorporating soluble fiber into one's diet.
- The intake of omega-3 fatty acids, such as fish oil or flax oil.
- The act of consuming pistachio nuts.
- Higher consumption of unsaturated fats.
- The ingestion of medium-chain triglycerides (MCTs) such as caproic acid, caprylic acid, capric acid, and lauric acid.
- Eliminating trans fatty acids from one's diet.

Saturated fats have a varied impact on HDL cholesterol, increasing its levels. However, they also boost the levels of total cholesterol and LDL cholesterol. A high-fat, adequate- protein, low-carbohydrate ketogenic diet may produce a comparable effect similar to niacin (vitamin B3) intake, resulting in decreased LDL cholesterol and increased HDL cholesterol. This effect is achieved through the coupling of Beta-hydroxybutyrate with the Niacin receptor 1. HDL levels can be elevated through smoking cessation or moderate alcohol use.

*Q. Niacin & Drugs*

Fibrate and niacin are two pharmacological therapeutic options for increasing HDL cholesterol levels. By selectively inhibiting hepatic diacylglycerol acyltransferase 2, niacin, often referred to as vitamin B3, increases HDL levels while decreasing triglyceride synthesis and VLDL secretion. Receptors HM74 (also called Niacin receptor 2) and HM74A/GPR109A (also called Niacin receptor 1) are activated to do this.

Pharmacologic administration of niacin at a dosage of 1 to 3 grams per day can raise HDL levels by 10-30%, making it the most potent drug for increasing HDL-cholesterol. A controlled experiment showed that niacin medication can effectively decrease the progression of atherosclerosis and the occurrence of cardiovascular events 79. Niacin products marketed as "no-flush" do not contain free nicotinic acid and are therefore ineffective at increasing HDL levels. On the other hand, products labeled as "sustained-release" may contain free nicotinic acid, but some brands have the potential to cause liver damage. Consequently, the most recommended form of niacin for raising HDL levels is the least expensive immediate-release preparation. Both fibrates and niacin elevate the levels of artery toxic homocysteine, which can be mitigated by consuming a multivitamin with substantial quantities of B-vitamins. However, several European trials investigating the efficacy of widely used B-vitamin combinations have demonstrated an average reduction of 30% in homocysteine levels, but have not shown any significant benefits in reducing cardiovascular event rates.

A study conducted in 2011 on the effects of niacin supplementation in patients undergoing statin medication was prematurely terminated. The study found that the addition of niacin did not result in any improvement in heart health. However, it did reveal an elevated risk of stroke among the participants.

Contrarily, whereas statins are efficacious in reducing elevated levels of LDL cholesterol, they generally exhibit minimal or negligible impact in increasing HDL cholesterol. However, studies have shown that both rosuvastatin and pitavastatin effectively increase HDL levels.

Lovaza has been demonstrated to elevate HDL-C levels. Nevertheless, the most compelling evidence available thus far indicates that it does not provide any advantages in terms of preventing cardiovascular disease, whether it be for primary or secondary prevention.

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# CHAPTER THREE
# MATERIALS & METHODS

- **STUDY DESIGN:** OBSERVATIONAL STUDY
- **DURATION OF STUDY:** 18 MONTHS (August 2022 - February 2024)
- **PLACE OF STUDY :** The study was conducted on 100 patients of **STROKE** admitted in Katuri Medical College, Guntur, General Medicine department, in both Male and Female OPD, Ward & Intensive medical care.
- **STUDY POPULATION:** Patients meeting the criteria at the general medicine department of Katuri Medical College were included. Informed written consent will be obtained.
- **SAMPLE SIZE:**100 patients

*A. Inclusion Criteria:*

- Age:35-70 years.
- Both Male and Female.
- Patient admitted with Stroke with neurological weakness(ON NCCT/MRI BRAIN PROVEN).

*B. Exclusion Criteria:*

- Patient refusal.
- Pre-existing cardiac diseases.
- Presumptive diagnosis of stroke with no evidence on NCCT/MRI BRAIN.
- Stroke due to tumor.
- Stroke due to trauma.
- Liver disease.

*C. Methods of Data Collection:*

- Sample size: 100 patients
- An Observational study was conducted, involving 50 patients diagnosed with Ischemic stroke and 50 patients diagnosed with Hemorrhagic stroke. None of the patients in either group exhibited any heart conditions.
- Patients were questioned to gather demographic information, including age and sex. Additionally, their medical history of other concomitant diseases was recorded, along with their presenting concerns. In addition, these patients underwent a physical examination to conduct a thorough neurological clinical assessment, accompanied by relevant investigations. The recorded findings were documented on a pre-designed and pre-tested proforma.

*D. Sample Collection:*

Using aseptic measures, a 6ml fasting venous blood sample was taken from each individual.2 milliliters of blood were transferred to plain tubes, and another 2 milliliters of blood were placed to vacutainers containing EDTA. In order to distinctly separate the serum, the vacutainers containing the blood samples were centrifuged for 15 minutes at a force of 2000 g after being left at room temperature for 30 minutes. As soon as the serum was separated, the following parameters were calculated.

*E. Measuring Hdl Cholestrol:*

- METHOD: Phospho-tungstic acid method, endpoint.
- PRINCIPLE: Chylomicrons (CM), LDL, and VLDL are separated from serum or plasma by causing them to form a solid precipitate using phosphotungstate in the presence of divalent cations like Magnesium. The HDL cholesterol in the remainder of the solution remains unchanged and is measured using a cholesterol reagent.

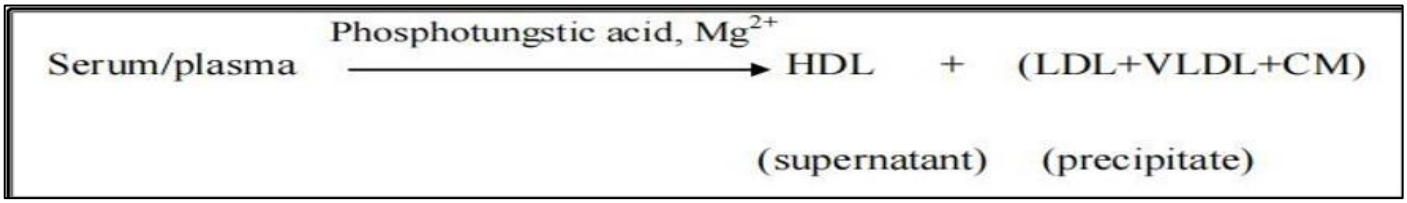


Fig 12: Showing The Biochemical Reaction

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*F. Reagent Composition:*

- **Reagent1:** Precipitating reagent

| | |
|---|---|
| Phosphotungstic acid | 2.4mmol/l |
| Magnesium chloride | 40mmol/l |

Fig 13: Composition of the Regnts

- HDL cholesterol standard – 25mg/dl
- SAMPLE: Un-hemolyzed serum used

- PRECIPITATION: Precipitation of LDL,VLDL and Chylomicrons done as follows:

| Pipette | Volume |
|---|---|
| Sample | 250µl |
| Precipitating reagent | 500µl |

Fig 14: Volume of Sample & Reagent Required

The mixture was thoroughly combined and then left undisturbed for 10 minutes at room temperature. Afterwards, it was subjected to centrifugation at a speed of 4000 revolutions per minute for 10 minutes, resulting in the collection of a transparent liquid layer above the sediment. The liquid portion, known as the supernatant, was utilized to ascertain the concentration of HDL cholesterol in the given sample.

| Pipette into tubes marked | Blank | Standard | Test |
|---|---|---|---|
| Cholesterol working reagent | 1000µl | 1000µl | 1000µl |
| Distilled water | 50µl | - | - |
| HDL standard | - | 50µl | - |
| Supernatant | - | - | 50µl |

Fig 15: Volume of Sample & Reagent Required for Cholestrol & HDL Estimation

➢ *Assay Procedure:*

Thoroughly combined and left to rest (incubated) for 10 minutes at ambient temperature. The standard and test samples have been evaluated for absorbance at 505 nm relative to the reagent blank.

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$$\text{HDL cholesterol (mg/dl)} = \frac{\text{Absorbance of test}}{\text{Absorbance of standard}} \times \text{conc.of standard} \times \text{dilutionfactor}$$

$$= \frac{\text{Absorbance of the test}}{\text{Absorbance of the standard}} \times 25 \times 3$$

$$= \frac{\text{Absorbance of the test}}{\text{Absorbance of the standard}} \times 75$$

Fig 16: Calculation of HDL Cholestrol
Linearity-upto 125mg/dl

*G. Estimation of Total Cholesterol:*

- **METHOD:** Cholesterol oxidase-Peroxidase Enzymatic, endpoint method.
- **PRINCIPLE:** Cholesterol esterase releases free cholesterol from cholesterol esters, which is then oxidized by cholesterol oxidase to form cholestenone. This process also generates hydrogen peroxide. The hydrogen peroxide undergoes a chemical reaction with 4 amino antipyrine and a phenolic molecule in the presence of peroxidase, resulting in the formation of a complex that exhibits a red color.

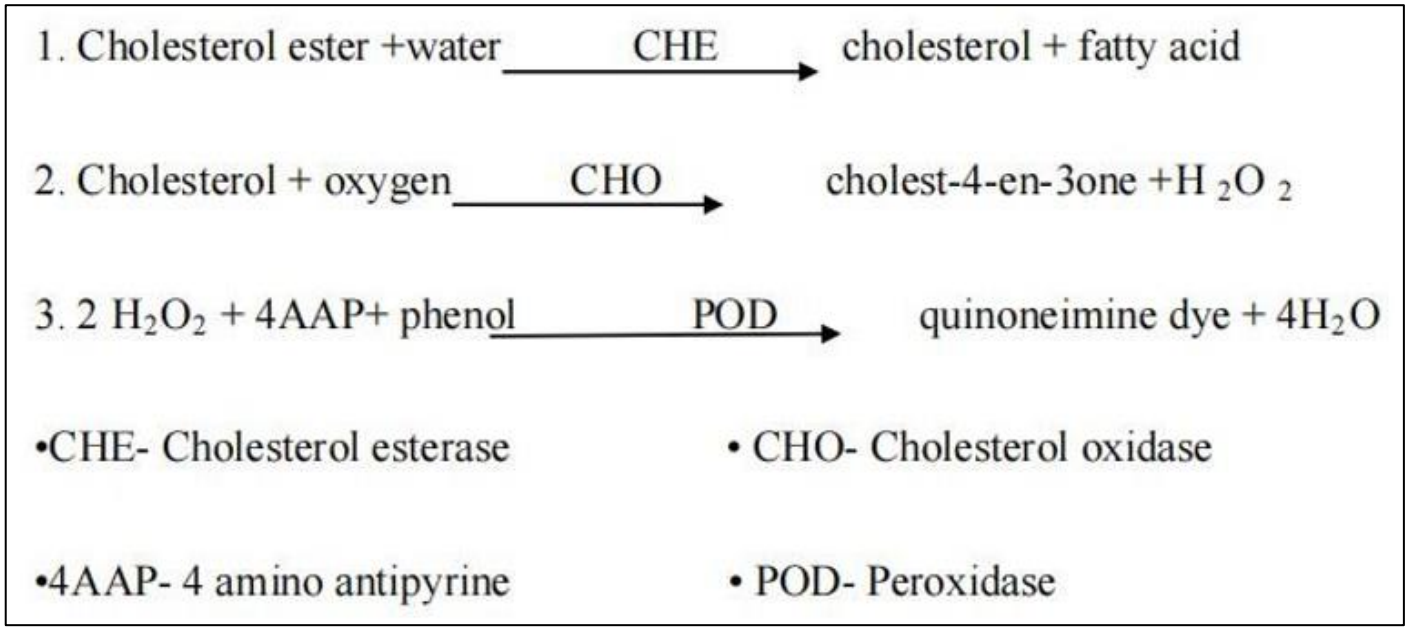


Fig 17: Chemical Reactions During Measurment of Cholestrol

The absorbance of the quinoneimine produced is directly related to the content of cholesterol.

➢ *Reagent:*

- Reagent-1(Enzyme/chromogen)
- Reagent-1A(BUFFER) Cholesterol standard-200mg/dl

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➢ *Reconstituted Reagent:*
Combine the contents of one bottle of reagent-1 with one bottle of reagent-1A.

| | Blank | Standard | Test |
|---|---|---|---|
| Working reagent | 1000µl | 1000µl | 1000µl |
| Distilled water | 10µl | - | - |
| Standard | - | 10µl | - |
| Sample | - | - | 10µl |

Fig 18: Assay Procude with Volume of Sample for Analysis

Thoroughly combined and left to sit for 10 minutes at ambient temperature. The test and standard's absorbance were measured against the reagent blank at a wavelength of 505 nm.

$$\text{Cholesterol(mg/dl)} = \frac{\text{Absorbance of test} \times \text{concentration of standard(mg/dl)}}{\text{Absorbance of standard}}$$

Fig 19: Calculation for Cholestrol Measurment

➢ *Reference Range:*

- 150-200 mg/dl
- Linearity - up to 750 mg/dl
- Sensitivity-1mg/dl

➢ *Interference:*
Hemoglobin levels of up to 200mg/dl, ascorbate levels of up to 12mg/dl, bilirubin levels of up to 10mg/dl, and triglyceride levels of up to 700mg/dl do not have any impact on the test.

*H. Triglycerides Estimation:*

- **PROCEDURE:** GPO-PAP technique, using an endpoint measurement
- **METHODOLOGY:** The methodology used is a colorimetric, enzymatic approach that involves the use of glycerol phosphate oxidase.

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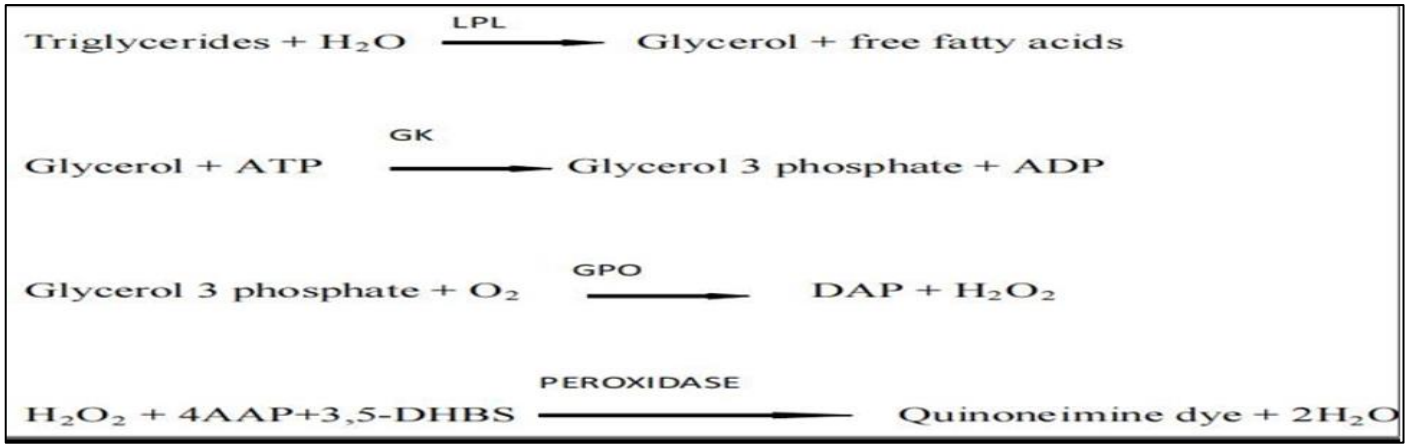


Fig 20: Principle in Triglycerides Estimation

- LPL stands for Lipoprotein lipase,
- whereas GK stands for Glycerol kinase.
- GPO stands for Glycerol Phosphate Oxidase,
- whereas DAP stands for Dihydroxy Acetone Phosphate.
- ATP is for Adenosine Tri Phosphate.
- 4AAP stands for 4Amino Anti Pyrine.
- DHBS stands for 3,5Dichloro-2Hydroxy Benzene Sulfonate.

Lipoprotein lipase catalyzes the breakdown of triacylglycerol into glycerol. Glycerol is then phosphorylated by glycerol kinase using ATP to form glycerol-3-phosphate. Glycerol-3-phosphate is oxidized to produce dihydroxyacetone phosphate and hydrogen peroxide. A chemical reaction occurs between hydrogen peroxide and phenolic substance, resulting in the formation of a colored complex when combined with 4-amino antipyrine.

The magnitude of the Quinoneimine dye produced is directly proportional to the content of triglycerides in the sample when measured at a wavelength of 505 nm (within the range of 500-540 nm).

➢ *Reagent:*

- Reagent 1 consists of enzymes or chromogen. Buffer solution is Reagent 2.
- The standard concentration of triglycerides is 200mg/dl.

✓ Preparing the working reagent includes combining one part of R2 with four parts of R1.It is stable for 90-days between 2-8 degrees Celsius.

- **Sample:** Twelve hours after fasting, unhemolysed serum was drawn.

Table 1: Assay Methodolgy

| Pipette into Tubes Marked | Blank | Standard | Test |
|---|---|---|---|
| Working reagent | 1000µl | 1000µl | 1000µl |
| Distilled water | 10µl | - | - |
| Standard | - | 10µl | - |
| Sample | - | - | 10µl |

Ten minutes of room temperature incubation followed mixing. Standard and sample absorbances against reagent blank were measured at 505 nm.

$$\text{Triglycerides(mg/dl)} = \frac{\text{Absorbance of test}}{\text{Absorbance of standard}} \times \text{Concentration of standard}$$

Fig 21: Calculation of Triglycerides

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| | |
|---|---|
| Serum/plasma | 37°C |
| Normal fasting level | 25-160mg/dl |

Fig 22: Reference Range for Triglycerides Estimation

Triglycerides/5 estimates VLDL.

LDL = TC - HDL - (Triglycerides/5).

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# CHAPTER FOUR
# RESULTS AND STATISTICS

Comparative analysis of average High-Density Lipoprotein (HDL) levels in Individuals with Ischemic and Hemorrhagic stroke:

Table 2: Variable Factor Significance

| VARIABLES | TYPES OF STROKE | N | MEAN | SD | t Stat | P Value |
|---|---|---|---|---|---|---|
| **AGE** | Ischemic | 50 | 52.54 | 13.6744 | - 2.2489 | 0.0115 |
| | Hemorrhagic | 50 | 57.96 | 10.1699 | | |
| **RBS** | Ischemic | 50 | 150.44 | 53.5652 | 1.4832 | 0.0722 |
| | Hemorrhagic | 50 | 134.68 | 55.7613 | | |
| **TOTAL CHOLESTEROL** | Ischemic | 50 | 161.32 | 37.3376 | 0.2353 | 0.4075 |
| | Hemorrhagic | 50 | 159.56 | 33.2745 | | |
| **HDL** | Ischemic | 50 | 32.30 | 7.7070 | - 3.9693 | 0.0001 |
| | Hemorrhagic | 50 | 39.02 | 8.5440 | | |
| **TGL** | Ischemic | 50 | 159.56 | 66.6811 | 0.2273 | 0.4106 |
| | Hemorrhagic | 50 | 157.08 | 39.9504 | | |
| **LDL** | Ischemic | 50 | 95.22 | 36.3903 | 0.9587 | 0.1712 |
| | Hemorrhagic | 50 | 88.94 | 31.3697 | | |
| **VLDL** | Ischemic | 50 | 31.60 | 14.1782 | 0.0950 | 0.4624 |
| | Hemorrhagic | 50 | 31.38 | 8.1438 | | |
| **SYSTOLIC BP** | Ischemic | 50 | 136.58 | 14.5925 | - 7.0398 | 0.0000 |
| | Hemorrhagic | 50 | 161.84 | 16.7933 | | |
| **DIASTOLIC BP** | Ischemic | 50 | 83.46 | 13.0325 | - 5.2439 | 0.0000 |
| | Hemorrhagic | 50 | 98.10 | 13.3038 | | |

Table 3: Average HDL Level in Patients with Ischemic and Hemorrhagic Stroke

| PARAMETER | ISCHEMIC STROKE | HEMORRHAGIC STROKE |
|---|---|---|
| HDL | 32.30 | 39.02 |

This table presents the average HDL levels in patients with ischemic stroke, which are lower compared to those with hemorrhagic stroke. The P value of 0.000 indicates a statistically significant difference between the two groups.

Table 4: Average Lipid-Profile in Ischemic & Hemorrhagic Stroke

| PARAMETER | ISCHEMIC STROKE | HEMORRHAGIC STROKE |
|---|---|---|
| Total Cholesterol | 161.32 | 159.56 |
| Triglycerides | 159.56 | 157.08 |
| VLDL | 31.6 | 31.38 |
| LDL | 95.22 | 88.94 |

An analysis of the average lipid profile is conducted to compare individuals who have had an Ischemic stroke with those who have had a Hemorrhagic stroke.

This table displays the Mean Total Cholesterol, Triglycerides, VLDL, and LDL levels, which are elevated in Ischemic stroke compared to Hemorrhagic stroke.

Table 5: Average Blood Pressures in Ischemic and Hemorrhagic Stroke

| MEAN BLOOD PRESSURE | ISCHEMIC STROKE | HEMORRHAGIC STROKE | |
|---|---|---|---|
| SYSTOLIC BP | 136.58 | 161.84 | |
| DIASTOLIC BP | 83.46 | 98.1 | |

Comparative analysis of average blood pressure levels in patients with Ischemic and Hemorrhagic stroke

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The average blood pressure readings after an ischemic and hemorrhagic stroke are shown in the table above. There is a statistically significant difference between hemorrhagic and ischemic strokes in terms of mean systolic and diastolic blood pressure (P = 0.000).

Table 6: Mean Age & RBS in Ischemic & Hemorrhagic Stroke

| PARAMETER | ISCHEMIC STROKE | HEMORRHAGIC STROKE |
|---|---|---|
| AGE | 52.54 | 57.96 |
| RBS | 150.44 | 134.68 |

An Analysis of Hemorrhagic and Ischemic Stroke Mean Age and RBS

- This table displays the statistically significant difference in the mean age between Hemorrhagic stroke and Ischemic stroke, with a P value of 0.030.
- This data indicates that the average RBS (Random Blood Sugar) level for ischemic stroke is higher than the average RBS level for hemorrhagic stroke.

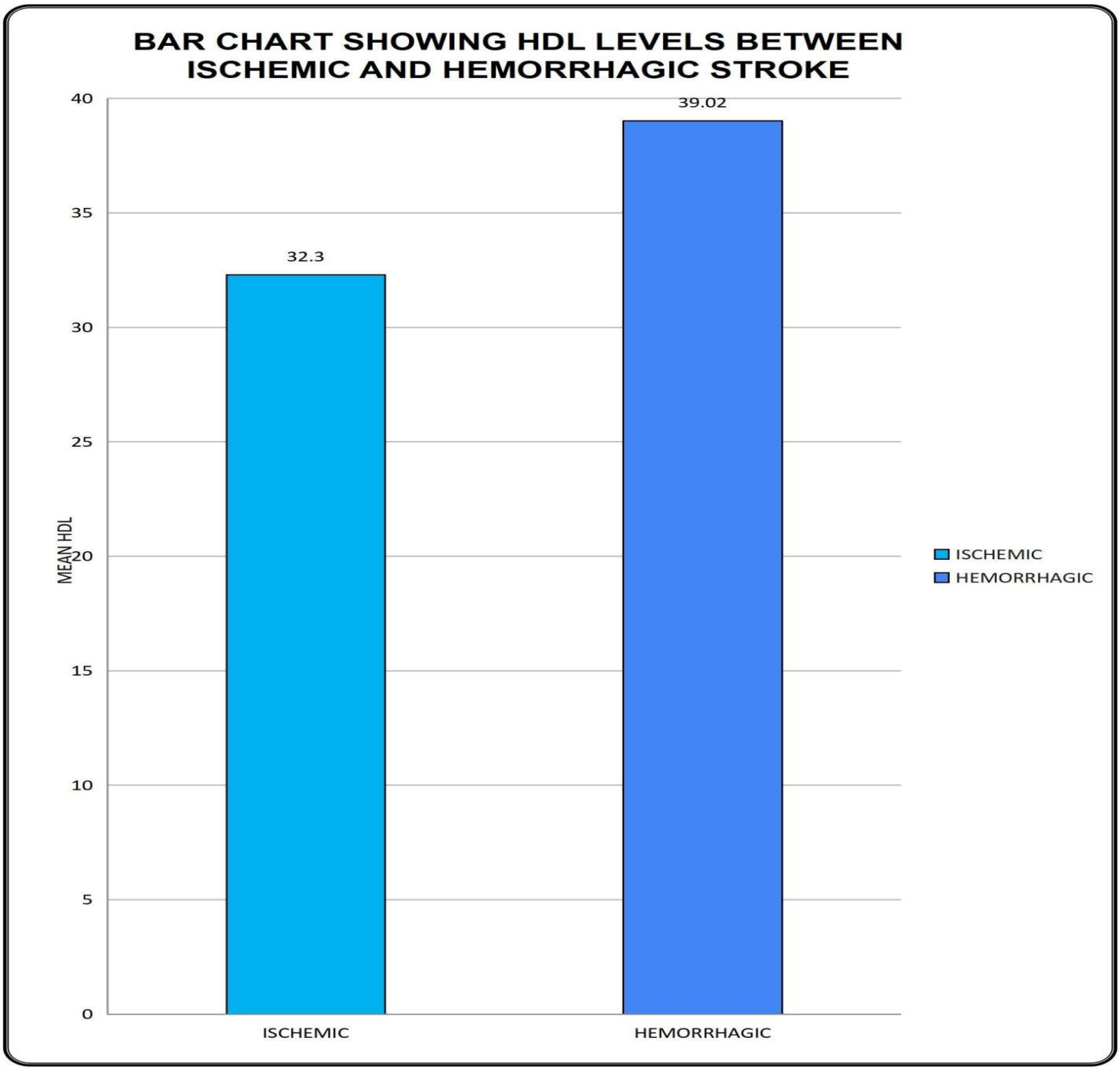


Fig 23: Bar-Chart Showing HDL Levels between Ischemic and Hemorrhagic Stroke

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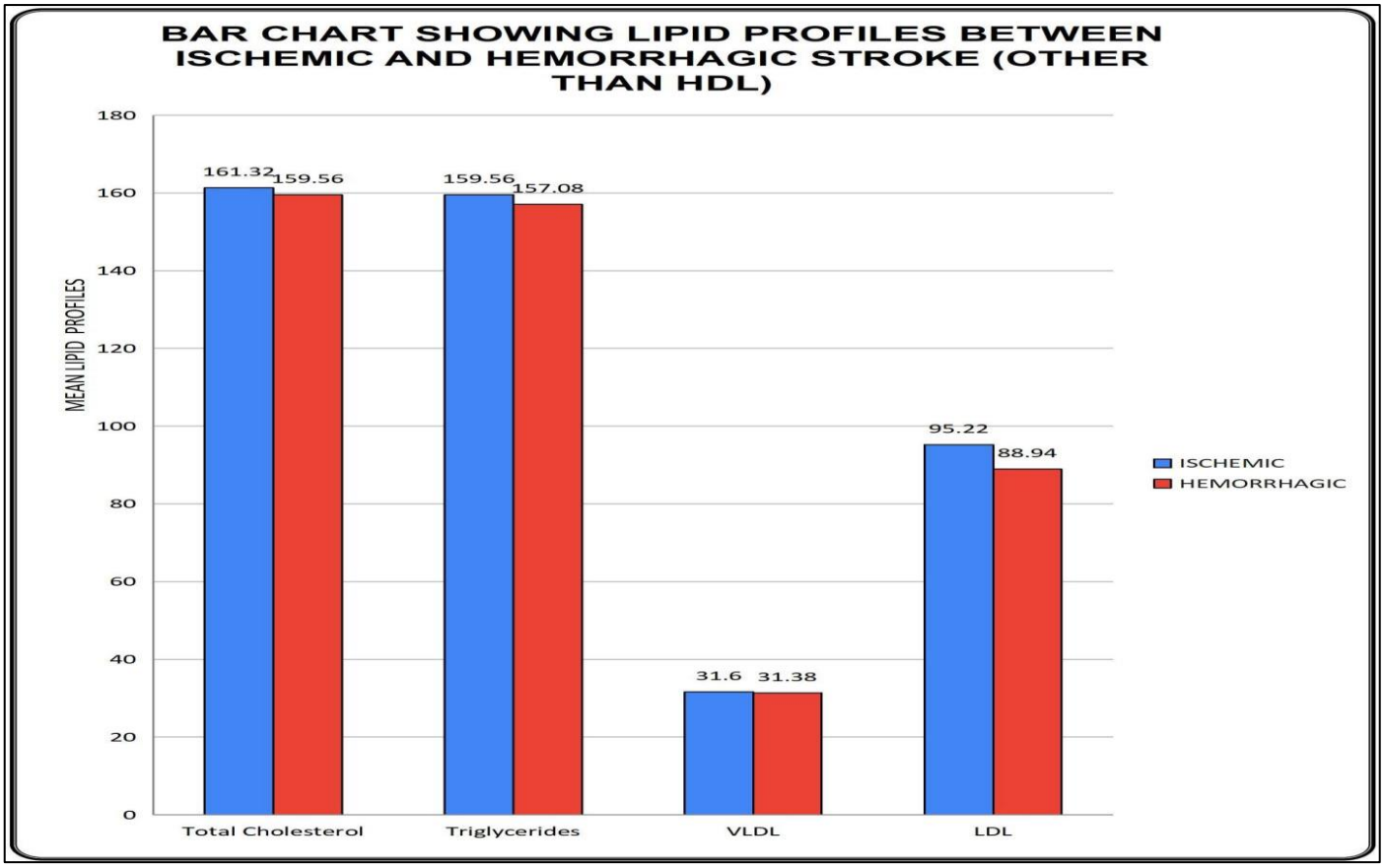


Fig 24: Bar-Chart Showing Lipid Profiles Between Ischemic and Hemorrhagic Stroke(other than HDL)

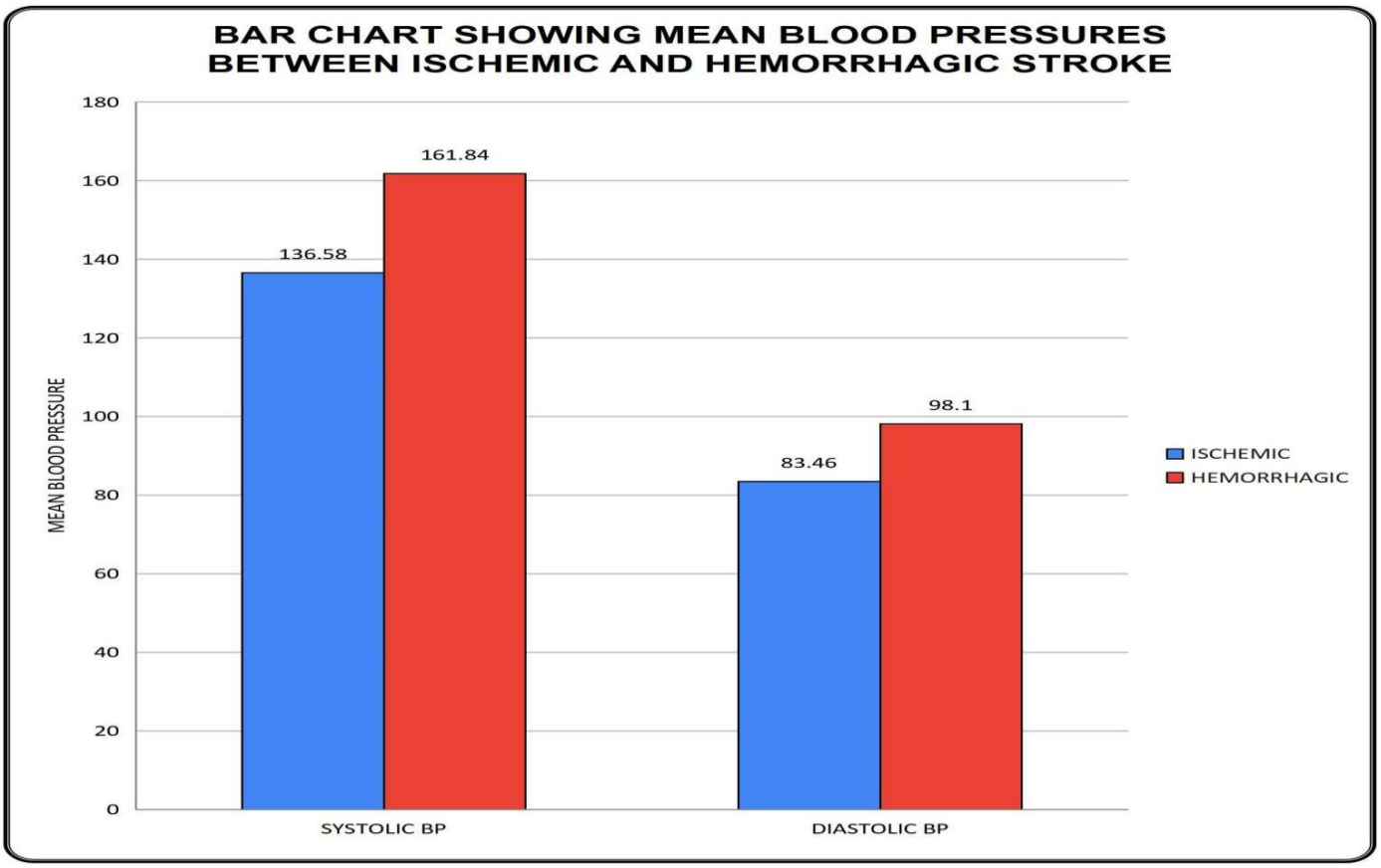


Fig 25: Bar-Chart Showing Mean Blood Pressures between Ischemic And Hemorrhagic Stroke

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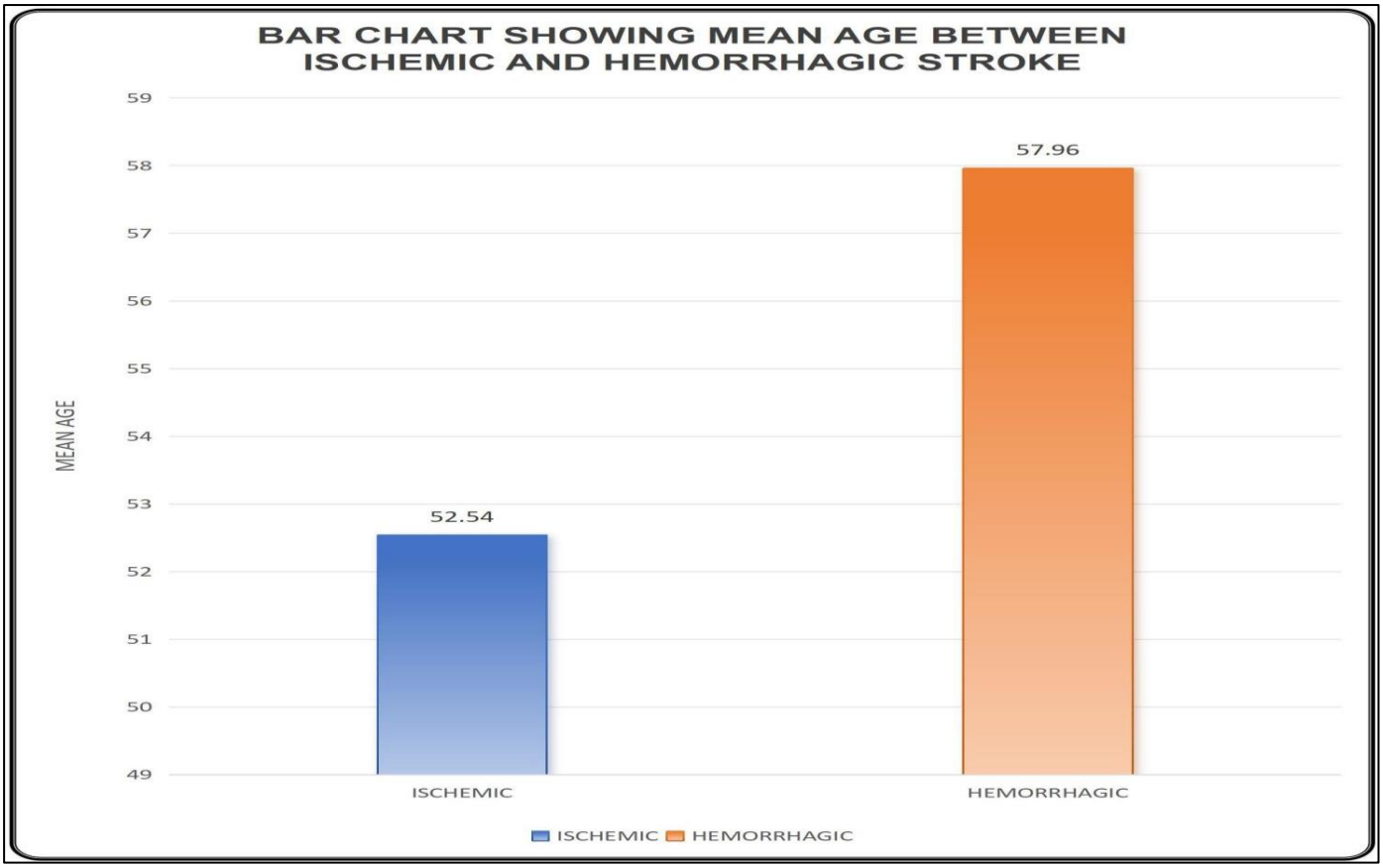


Fig 26: Bar-Chart Showing Mean Age between Ischemic and Hemorrhagic Stroke

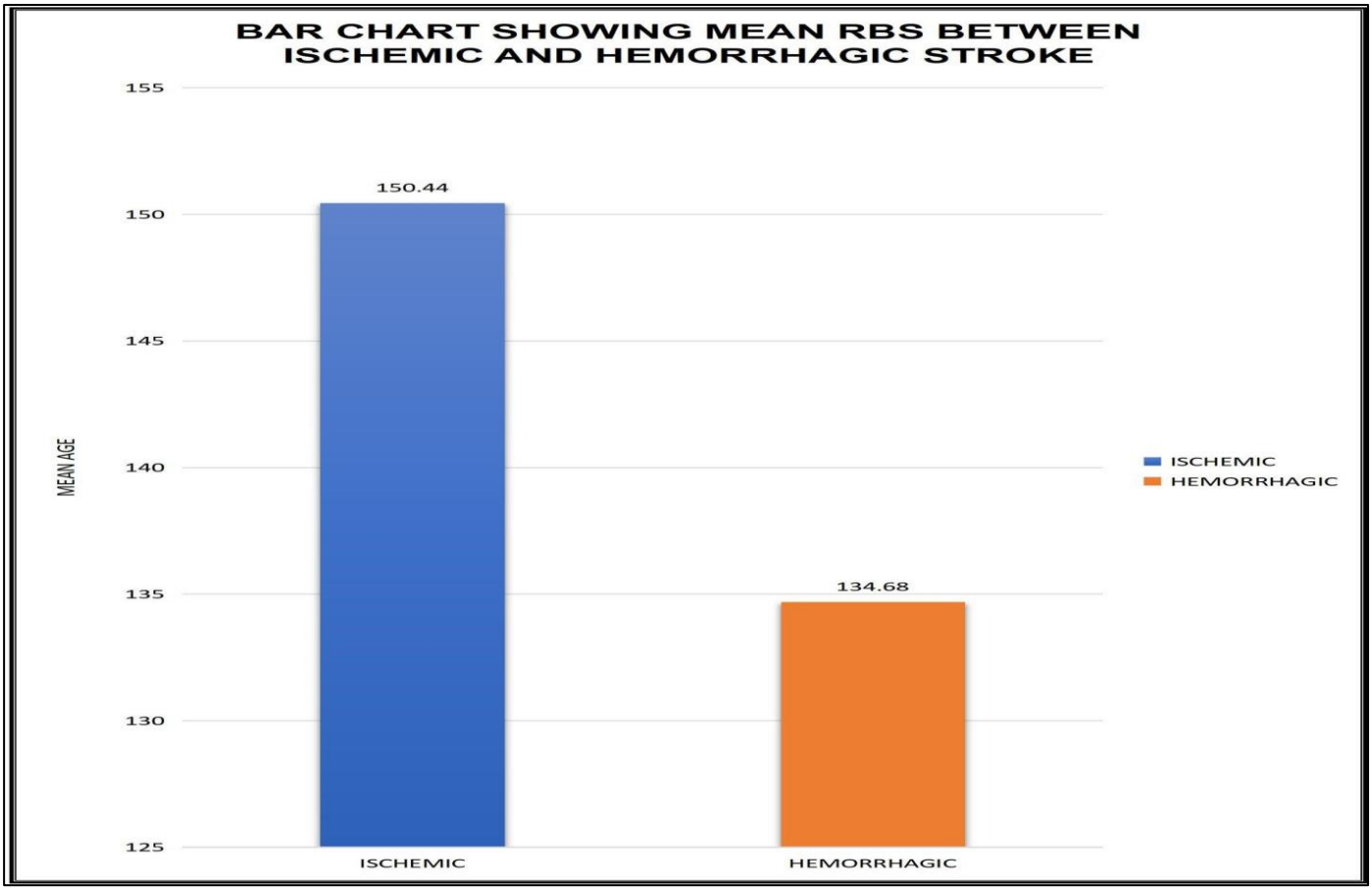


Fig 27: Bar-Chart Showing Mean RBS between Ischemic And Hemorrhagic Stroke

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Table 7: Percentage of Site of Lesion in Hemorrhagic Stroke

| PERCENTAGE OF SITE OF LESION IN HEMORRHAGIC STROKE | |
|---|---|
| SITE OF THE LESION | PERCENTAGE |
| PUTAMEN | 32% |
| THALAMUS | 16% |
| MULTIPLE SITES | 12% |
| TEMPORO- PARIETAL | 14% |
| BRAIN STEM | 10% |
| FRONTO-PARIETAL | 10% |
| CEREBELLUM | 6% |
| **TOTAL** | **100%** |

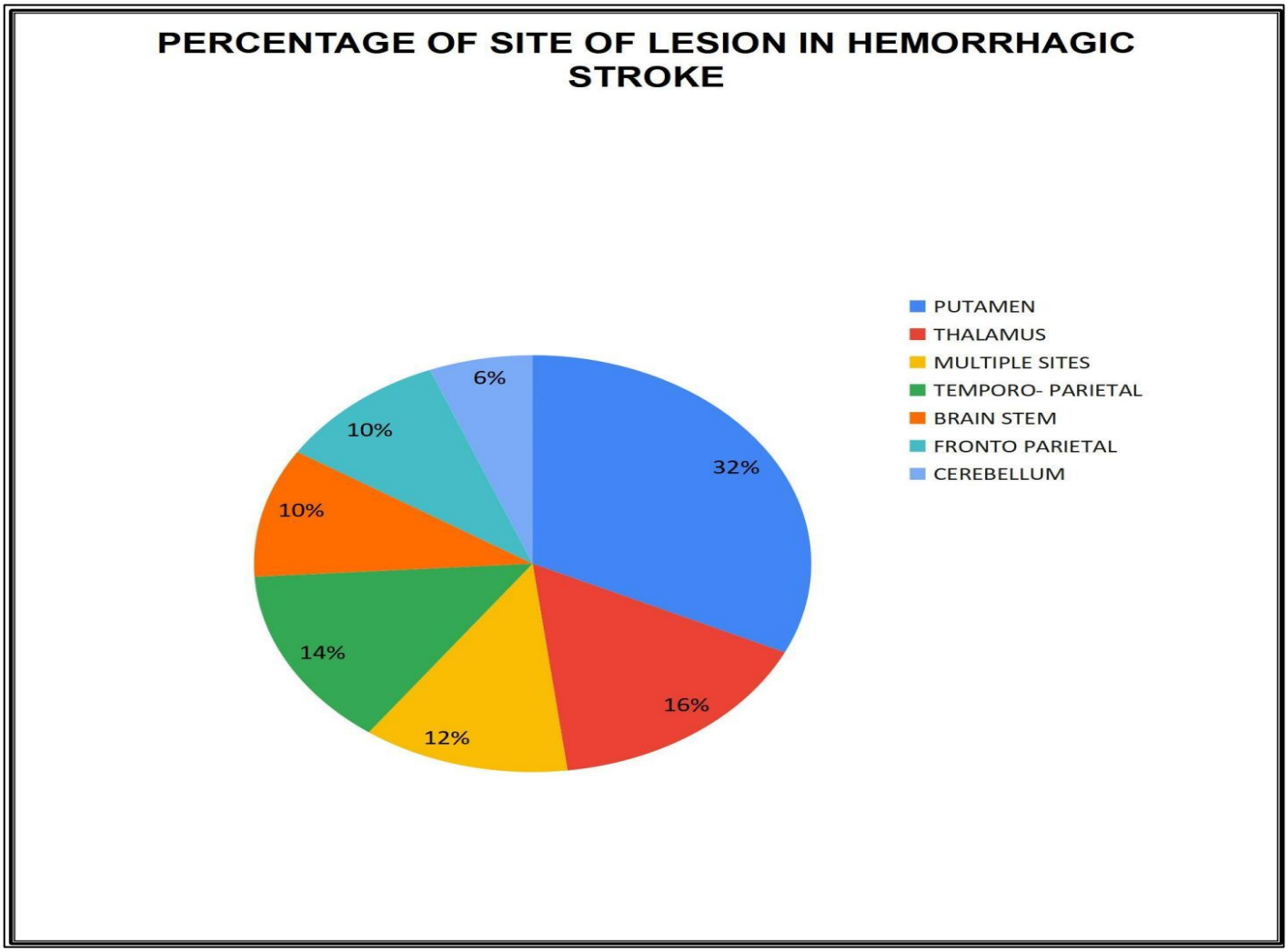


Fig 28: Percentage of Site of Lesion in Hemorrhagic Stroke

Table 8: Percentage of Artery Involvement in Ischemic Stroke

| PERCENTAGE OF ARTERY INVOLVEMENT IN ISCHEMIC STROKE | |
|---|---|
| ARTERY INVOLVED | PERCENTAGE |
| MIDDLE CEREBRAL ARTERY | 42% |
| ANTERIOR CEREBRAL ARTERY | 16% |
| VERTEBRO- BASILAR ARTERY | 18% |
| INTERNAL CAROTID ARTERY | 14% |
| MULTIPLE SITES | 10% |
| TOTAL | 100% |

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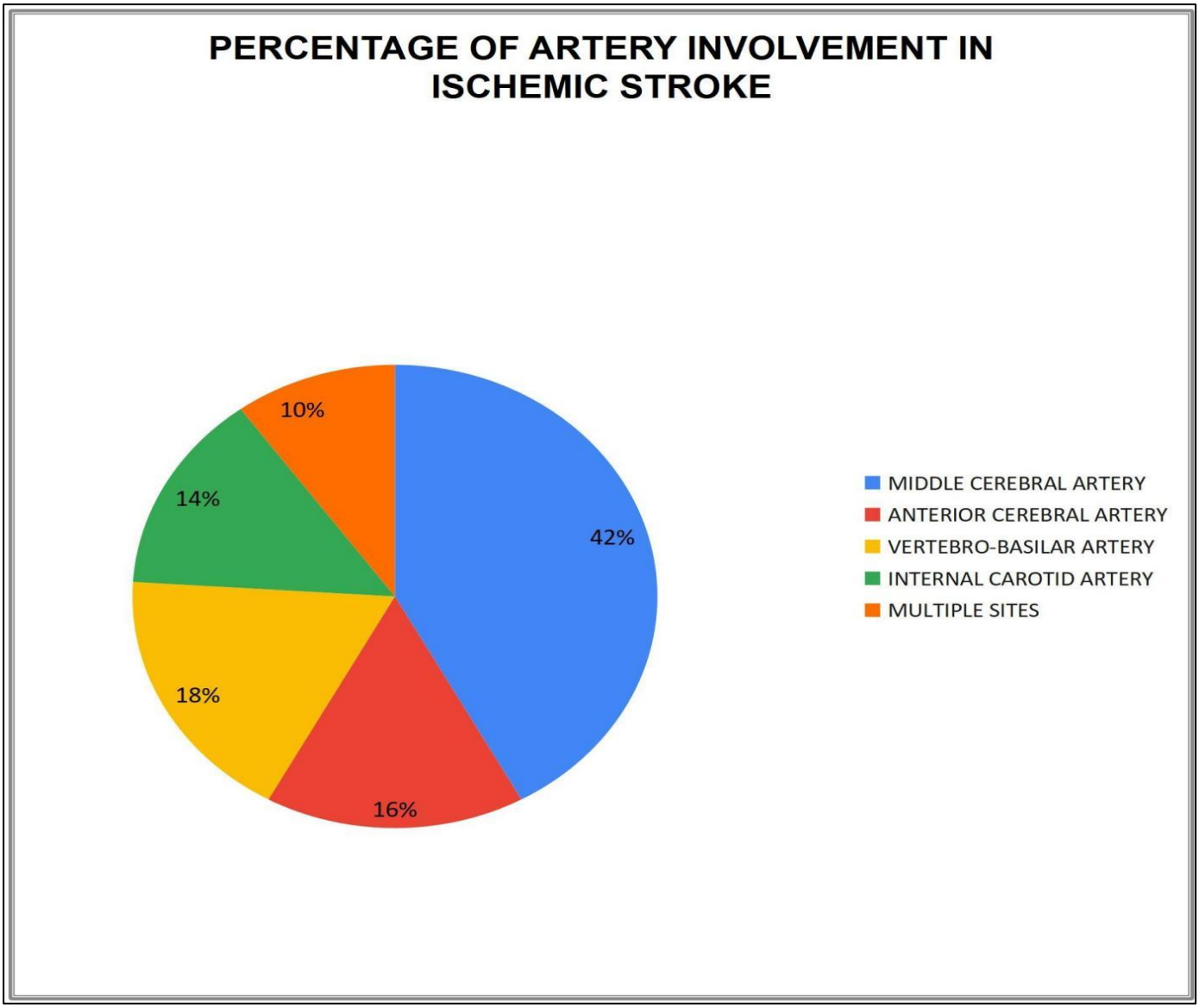


Fig 29: Percentage of Artery Involvement in Ischemic Stroke

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# CHAPTER FIVE
# DISCUSSION

The current research, conducted with a sample size of 100 individuals (50 with Ischemic stroke and 50 with Hemorrhagic stroke), provides evidence that assessing the lipid profile is a valuable tool for predicting the risk factors associated with stroke.

The average HDL value of patients with Hemorrhagic stroke (39.02) was greater than the average HDL value of patients with Ischemic Stroke (32.30), and this difference was statistically significant with a p-value of less than 0.05.

The average levels of Total Cholesterol (161.32), Triglycerides (159.561), VLDL (31.60), and LDL (95.22) were greater in cases of ischemic stroke compared to hemorrhagic stroke, where the average levels of Total Cholesterol (159.563), Triglycerides (157.08), VLDL (31.38), and LDL (88.94) were observed.

The average systolic blood pressure (BP) for Hemorrhagic stroke (161.84) was greater than the average systolic BP for Ischemic stroke (135.58). Similarly, the average diastolic BP for Hemorrhagic stroke (98.10) was higher than the average diastolic BP for ischemic stroke (83.46).The P value, which indicates statistical significance, was less than 0.05.

The average age of individuals with Hemorrhagic stroke was 57.96, which was greater than the average age of those with ischemic stroke, which was 52.54. The P value, which indicates statistical significance, was less than 0.05.

The average random blood sugar levels of patients with ischemic stroke was 150.44, which was higher compared to patients with hemorrhagic stroke, whose average blood sugar level was 134.68.

The distribution of lesion sites in hemorrhagic stroke is as follows: putamen (32%), thalamus (16%), various sites (12%), temporoparietal (14%), brainstem (10%), frontoparietal (10%), and cerebellum (6%).

The degree of arterial involvement in Ischemic stroke is as follows: Middle cerebral artery - 42%, Anterior cerebral artery - 16%, Vertebrobasilar artery - 18%. The prevalence of stenosis in the Internal carotid artery is 12%, whereas stenosis in many places is 8%.

This research demonstrates that levels of HDL were considerably lower in cases of Ischemic stroke compared to those of Hemorrhagic stroke. In contrast, the levels of HDL were elevated in cases of Hemorrhagic stroke.

- Ischemic stroke had higher levels of total cholesterol, triglycerides, LDL, and VLDL compared to Hemorrhagic stroke.
- The Random Blood sugar level was higher in cases of ischemic stroke compared to hemorrhagic stroke.
- Hemorrhagic stroke had higher blood pressure levels compared to ischemic stroke.
- Hemorrhagic stroke patients were in an older age bracket compared to those with ischemic stroke.
- The order of lesion sites in Hemorrhagic stroke is as follows: putamen, thalamus, numerous sites, temporoparietal, brain stem, frontoparietal, and cerebellum.
- The sequence of Artery involvement in Ischemic stroke was as follows: Middle Cerebral Artery > Anterior Cerebral Artery > Vertebrobasilar Artery >Internal Carotid Artery > Multiple locations.

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# CHAPTER SIX
# CONCLUSION

This research demonstrates a significant reduction in HDL levels in cases of Ischemic stroke compared to Hemorrhagic stroke. The diminished Reverse Cholesterol Transport, Anti-inflammatory, Anti-oxidative, Anti-Apoptotic, Endothelial repair, Anti-Thrombotic, and Anti-infectious activities of HDL due to decreased HDL results in a higher risk of Ischemic stroke compared to Hemorrhagic stroke.

The findings of the current research indicate that HDL may be used as an early indicator of atherosclerosis and ischemic stroke. By doing early measurements of HDL, it is possible to implement early intervention strategies using pharmacological or nutritional methods to raise HDL levels and reduce the occurrence of stroke-related illness and death.

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# ANNEXURES

## PATIENT INFORM CONSENT FORM

Patient I.D. No.

Title of the Project: "FUNCTION OF HDL IN STROKE AS WELL AS COMPARISION OF ITS LEVELS IN ISCHEMIC AND HEMORRHAGIC STROKE".

Principal Investigator Name: **Dr.M.Naga pradeep**

Contact No:

Information Sheet: I have read the information sheet carefully/ explained in the language I understand, and I fully understand the contents. I confirm that I have had an opportunity to askquestions. I have been explained in detail about the nature and purpose of the study and its potential risks/benefits, and the expected duration of the study and other related details of the study. I understand that my participation is voluntary and i am free to withdraw at any time without giving any reason, my medical care and legal rights.

I agree to participate in the study.

.....
(signature / left thumb impression) Date:

Place:
Name of the participant:.....
Son/ Daughter/ Spouse of:.....
Complete postal address: .....

This certifies that the above consent has been taken in my presence.

.....
(Signature of principal investigator)

Date: .....Place: .....

witness- I..... 2) Witness - II .....

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## PROFORMA FORMAT

NAME: AGE/SEX:

IP NUMBER: ADDRESS: PHONE NUMBER:

PRESENTING COMPLAINTS:

WEAKNESS

GIDDINESS

SEIZURE

UNRESPONSIVENESS

UNSTEADINESS OF GAIT

DEVIATION OF ANGLE OF MOUTH DURATION OF SYMPTOMS:
H/O FEVER/TRAUMA PAST HISTORY:
PRIOR STROKE

DIABETES MELLITUS

HYPERTENSION

CARDIAC DISEASES

EPILEPSY

TUBERCULOSIS

TREATMENT HISTORY: FAMILY HISTORY: ADDICTIONS: VITALS:

PULSE-RATE AND RHYTHM

BLOOD PRESSURE• RESPIRATORY RATE

TEMPERATURE ,SPO2 GENERAL EXAMINATION
PALLOR, ICTERUS,CYANOSIS,CLUBBING,LYMPHADENOPATHY,PEDAL EDEMA SYSTEMIC EXAMINATION
CENTRAL NERVOUS SYSTEM :-

HIGHER MENTAL FUNCTIONS: CONSCIOUSNESS ORIENTATION
SPEECH

CRANIAL NERVES

MOTOR SYSTEM:-ATTITUDE OF LIMB TONE
POWER REFLEXES
SENSORY SYSTEM

CEREBELLAR SIGNS

MENINGEAL SIGNS

GLASGOW COMA SCALE- E V M (TOTAL SCORE -15) CARDIOVASCULAR SYSTEM
APEX BEAT

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HEART SOUNDS

MURMURS RESPIRATORY SYSTEM
BREATH SOUNDS

ADDED SOUNDS GASTROINTESTINAL SYSTEM
ANY TENDERNESS

LIVER SPAN

SHIFTING DULLNESS INVESTIGATIONS :-
COMPLETE HEMOGRAM

RANDOM BLOOD SUGAR

SERUM CREATININE

LIVER FUNCTION TEST

LIPID PROFILE

SERUM PROTEINS: TOTAL:
ALBUMIN:

ECG:

NCCT/MRI- BRAIN:

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# MASTER CHART-I
# ISCHEMIC STROKE

| S.NO | NAME | AGE | SEX | SBP | DBP | RBS | LIPID PROFILE(mg/dl) | | | | | ARTERY INVOLVED |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | (mm Hg) | | mg/dl | T.CHO | TGL | HDL | LDL | VLDL | |
| 1 | T RAJASEKHAR | 55 | M | 150 | 90 | 136 | 126 | 96 | 26 | 80 | 14 | **MCA** |
| 2 | SURESH KANNA | 27 | M | 146 | 92 | 182 | 156 | 88 | 32 | 106 | 18 | **MCA** |
| 3 | J ANASUYAMMA | 54 | F | 142 | 80 | 201 | 263 | 262 | 38 | 183 | 52 | **MCA** |
| 4 | KAVITHA K | 46 | F | 146 | 70 | 256 | 148 | 127 | 42 | 81 | 25 | **ICA** |
| 5 | P YESU RAJU | 47 | M | 130 | 89 | 186 | 199 | 117 | 44 | 122 | 23 | **Multi site** |
| 6 | U VENKAIAH | 70 | M | 110 | 72 | 146 | 124 | 111 | 35 | 69 | 22 | **MCA** |
| 7 | J VEERAMMA | 78 | F | 126 | 60 | 124 | 122 | 91 | 28 | 77 | 15 | **MCA** |
| 8 | D BALASWAMY | 70 | M | 137 | 81 | 83 | 161 | 107 | 28 | 133 | 21 | **MCA** |
| 9 | M CHAITANYA | 50 | M | 100 | 62 | 156 | 220 | 85 | 38 | 164 | 17 | **VBA** |
| 10 | P SRINIVASA RAO | 52 | M | 141 | 90 | 336 | 135 | 383 | 26 | 32 | 75 | **Multi site** |
| 11 | V PAPA | 60 | F | 130 | 94 | 109 | 116 | 66 | 22 | 82 | 13 | **ICA** |
| 12 | JAN BI | 25 | F | 144 | 90 | 232 | 110 | 148 | 23 | 57 | 30 | **MCA** |
| 13 | K KOTESWARA RAO | 54 | M | 160 | 99 | 142 | 121 | 188 | 28 | 52 | 38 | **VBA** |
| 14 | S JHANARDHAN | 43 | M | 136 | 84 | 128 | 108 | 144 | 23 | 56 | 30 | **ACA** |
| 15 | RAHMATULLA | 55 | M | 138 | 92 | 104 | 126 | 116 | 21 | 76 | 23 | **ACA** |
| 16 | SRIKANTH | 28 | M | 148 | 93 | 207 | 158 | 138 | 32 | 96 | 28 | **MCA** |
| 17 | S SAMBRAJYAM | 54 | F | 140 | 80 | 202 | 257 | 274 | 38 | 173 | 50 | **MCA** |
| 18 | K SUBBA RAO | 60 | M | 144 | 70 | 61 | 168 | 335 | 31 | 65 | 67 | **ACA** |
| 19 | M YEDUKONDALU | 70 | M | 133 | 83 | 127 | 177 | 242 | 38 | 91 | 48 | **MCA** |
| 20 | NURULLA | 55 | M | 132 | 82 | 217 | 132 | 108 | 29 | 83 | 22 | **MCA** |
| 21 | G KANAKAMMA | 78 | F | 122 | 55 | 125 | 132 | 92 | 27 | 87 | 18 | **VBA** |
| 22 | CH SUBAMMA | 58 | F | 137 | 74 | 83 | 161 | 97 | 16 | 125 | 19 | **ICA** |
| 23 | SK KHASIM | 50 | M | 99 | 65 | 154 | 219 | 95 | 30 | 162 | 12 | **MCA** |
| 24 | K MAHBOOBI | 47 | F | 143 | 94 | 106 | 173 | 212 | 33 | 100 | 40 | **ICA** |
| 25 | Y VENKATESWARA RAO | 63 | M | 133 | 96 | 92 | 111 | 123 | 27 | 47 | 25 | **MCA** |
| 26 | U PUSHPA LATHA | 56 | F | 154 | 100 | 197 | 186 | 179 | 36 | 114 | 36 | **MCA** |
| 27 | DAYARNA | 44 | M | 130 | 90 | 189 | 198 | 116 | 46 | 137 | 23 | **Multi site** |
| 28 | I MANIKYAM | 70 | M | 120 | 77 | 148 | 128 | 101 | 35 | 71 | 20 | **VBA** |
| 29 | S MALLESWARI | 78 | F | 126 | 58 | 124 | 122 | 104 | 25 | 74 | 11 | **ICA** |
| 30 | SK KARIMULLA | 70 | M | 131 | 83 | 82 | 162 | 98 | 24 | 119 | 19 | **Multi site** |
| 31 | K SUDHAKAR | 50 | M | 105 | 67 | 158 | 216 | 86 | 38 | 164 | 16 | **ACA** |
| 32 | N SURYA PRAKASH | 48 | M | 136 | 80 | 248 | 156 | 152 | 32 | 94 | 30 | **ACA** |
| 33 | SK GOUSIYA BEE | 42 | F | 128 | 88 | 135 | 207 | 189 | 34 | 135 | 38 | **MCA** |
| 34 | R SURYA CHANDRA RAO | 67 | M | 142 | 84 | 210 | 166 | 132 | 30 | 108 | 27 | **VBA** |
| 35 | G SAMRAJYAM | 57 | F | 149 | 105 | 174 | 169 | 178 | 39 | 96 | 36 | **MCA** |
| 36 | U YESUDANAM | 62 | M | 152 | 110 | 144 | 197 | 175 | 32 | 130 | 35 | **MCA** |
| 37 | L NARESH | 25 | M | 139 | 72 | 132 | 212 | 186 | 35 | 140 | 38 | **ICA** |
| 38 | PUTANI BAI | 62 | F | 155 | 108 | 108 | 154 | 168 | 40 | 82 | 34 | **MCA** |
| 39 | T ANJANEYULU | 39 | M | 118 | 98 | 202 | 152 | 228 | 31 | 72 | 48 | **VBA** |
| 40 | B KIRAN | 37 | M | 157 | 86 | 111 | 150 | 162 | 18 | 88 | 32 | **MCA** |
| 41 | S HANUMAIAH | 51 | M | 140 | 82 | 152 | 171 | 183 | 32 | 102 | 37 | **ACA** |
| 42 | SK SHABIRUN | 48 | F | 134 | 86 | 183 | 158 | 169 | 47 | 97 | 34 | **VBA** |

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| | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 43 | P NAGESWARA RAO | 45 | M | 145 | 70 | 153 | 118 | 170 | 25 | 54 | 34 | **ACA** |
| 44 | DURGA PRASAD | 53 | M | 162 | 84 | 138 | 164 | 239 | 37 | 75 | 52 | **MCA** |
| 45 | N ARUNA KUMARI | 33 | F | 138 | 97 | 93 | 131 | 124 | 57 | 48 | 25 | **VBA** |
| 46 | BOLLA RAO | 41 | M | 164 | 102 | 129 | 163 | 218 | 31 | 89 | 58 | **ACA** |
| 47 | RAMKRISHNA | 31 | M | 135 | 89 | 116 | 120 | 180 | 34 | 51 | 36 | **ICA** |
| 48 | SK PEERAVALI | 49 | F | 148 | 76 | 95 | 201 | 158 | 36 | 33 | 32 | **VBA** |
| 49 | T AKKA RAO | 70 | M | 134 | 68 | 126 | 180 | 256 | 38 | 91 | 48 | **Multi site** |
| 50 | DHUBLA | 50 | M | 120 | 76 | 80 | 162 | 182 | 28 | 98 | 36 | **MCA** |

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MASTER CHART-II
HEMORRHAGIC STROKE

| S.NO | NAME | AGE | SEX | SBP | DBP | RBS | LIPID PROFILE(mg/dl) | | | | | SITE OF LESION |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | (mm Hg) | | mg/dl | T.CHO | TGL | HDL | LDL | VLDL | |
| 1 | CH KOTESWARAMMA | 44 | F | 176 | 93 | 127 | 158 | 176 | 40 | 84 | 35 | PUTAMEN |
| 2 | P LAZAR | 50 | M | 136 | 80 | 92 | 160 | 92 | 42 | 99 | 19 | PUTAMEN |
| 3 | B BALA SWAMY | 52 | M | 146 | 92 | 87 | 154 | 93 | 46 | 89 | 19 | THALAMUS |
| 4 | J RAMESH | 54 | M | 156 | 82 | 114 | 121 | 188 | 34 | 57 | 38 | PUTAMEN |
| 5 | JANU VALI | 58 | M | 150 | 86 | 51 | 144 | 86 | 56 | 72 | 16 | FRONTOPARIETAL |
| 6 | MASTAN RAO | 59 | M | 174 | 90 | 264 | 175 | 138 | 38 | 109 | 28 | CEREBELLUM |
| 7 | K VENKATESWARLU | 63 | M | 155 | 91 | 159 | 236 | 256 | 51 | 136 | 48 | PUTAMEN |
| 8 | K SUBBA RAO | 45 | M | 149 | 100 | 124 | 186 | 116 | 48 | 115 | 23 | THALAMUS |
| 9 | G LEELAVATHI | 58 | F | 156 | 90 | 103 | 126 | 133 | 33 | 66 | 26 | BRAIN STEM |
| 10 | J DEVADASU | 55 | M | 141 | 108 | 140 | 200 | 199 | 49 | 111 | 40 | TEMPOROPARIETAL |
| 11 | S LAKSHMI | 49 | F | 152 | 102 | 106 | 165 | 143 | 28 | 108 | 29 | PUTAMEN |
| 12 | Y RAMESH | 59 | M | 160 | 84 | 86 | 143 | 166 | 33 | 78 | 33 | MULTI SITE |
| 13 | I SUBBAIAH | 70 | M | 140 | 86 | 143 | 178 | 124 | 52 | 102 | 25 | PUTAMEN |
| 14 | M ARUNA | 63 | F | 157 | 88 | 182 | 202 | 174 | 36 | 127 | 35 | FRONTOPARIETAL |
| 15 | S DEVI | 49 | F | 148 | 92 | 96 | 162 | 148 | 30 | 98 | 30 | CEREBELLUM |
| 16 | K PURNACHANDRA RAO | 53 | M | 150 | 104 | 132 | 122 | 189 | 37 | 46 | 38 | MULTI SITE |
| 17 | A LAKSHMI | 64 | F | 190 | 96 | 98 | 142 | 139 | 30 | 82 | 28 | BRAIN STEM |
| 18 | Y SHOWRI REDDY | 53 | M | 163 | 100 | 137 | 112 | 222 | 26 | 40 | 44 | PUTAMEN |
| 19 | K BHAGYAMMA | 70 | F | 166 | 89 | 68 | 183 | 132 | 31 | 125 | 27 | FRONTOPARIETAL |
| 20 | K KOTESWARA RAO | 55 | M | 154 | 82 | 130 | 129 | 186 | 40 | 50 | 37 | PUTAMEN |
| 21 | K RAMULU | 65 | M | 180 | 110 | 107 | 232 | 140 | 44 | 156 | 29 | THALAMUS |
| 22 | G DRAKSHAMMA | 62 | F | 142 | 101 | 126 | 182 | 163 | 38 | 110 | 32 | TEMPOROPARIETAL |
| 23 | MOIDDIN | 57 | M | 222 | 105 | 109 | 155 | 209 | 41 | 76 | 42 | BRAIN STEM |
| 24 | J KUMARI | 52 | F | 168 | 92 | 163 | 132 | 162 | 32 | 68 | 31 | PUTAMEN |
| 25 | K RAMULU | 72 | M | 170 | 90 | 232 | 92 | 106 | 35 | 35 | 21 | TEMPOROPARIETAL |
| 26 | N VARA LAKSHMI | 63 | F | 160 | 76 | 187 | 140 | 117 | 24 | 95 | 23 | PUTAMEN |
| 27 | B NAGARAJU | 55 | M | 156 | 101 | 144 | 190 | 199 | 34 | 112 | 40 | BRAIN STEM |
| 28 | M NAGESWARA RAO | 59 | M | 188 | 126 | 105 | 176 | 164 | 41 | 106 | 33 | THALAMUS |
| 29 | YESU KUMARI | 57 | F | 162 | 106 | 186 | 110 | 168 | 36 | 43 | 34 | MULTI SITE |
| 30 | HANUMANTHA RAO | 64 | M | 146 | 70 | 94 | 130 | 147 | 69 | 32 | 30 | TEMPOROPARIETAL |
| 31 | KASI NATH | 42 | M | 158 | 94 | 101 | 202 | 157 | 47 | 122 | 33 | PUTAMEN |
| 32 | B VENKATAMMA | 55 | F | 165 | 124 | 328 | 141 | 134 | 38 | 77 | 27 | CEREBELLUM |

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| | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 33 | L HANUMAYYA | 77 | M | 148 | 90 | 79 | 124 | 96 | 44 | 60 | 18 | THALAMUS |
| 34 | CH ANJANEYULU | 49 | M | 172 | 114 | 192 | 203 | 185 | 32 | 128 | 37 | MULTI SITE |
| 35 | N SIVA SRINU | 73 | M | 144 | 102 | 111 | 138 | 156 | 32 | 74 | 31 | PUTAMEN |
| 36 | D HYMAVATHI | 69 | F | 180 | 120 | 98 | 173 | 130 | 40 | 107 | 24 | FRONTOPARIETAL |
| 37 | T SRINIVASA RAO | 55 | M | 200 | 109 | 210 | 167 | 173 | 38 | 94 | 35 | THALAMUS |
| 38 | V THIRUMALESH | 40 | M | 154 | 96 | 117 | 141 | 136 | 43 | 72 | 26 | PUTAMEN |
| 39 | M NAGENDRA REDDY | 37 | M | 186 | 80 | 197 | 202 | 176 | 37 | 130 | 36 | TEMPOROPARIETAL |
| 40 | K HARI BABU | 65 | M | 148 | 110 | 136 | 220 | 145 | 46 | 144 | 24 | PUTAMEN |
| 41 | T CHENCHU LAKSHMI | 74 | F | 164 | 72 | 78 | 134 | 167 | 25 | 76 | 34 | MULTI SITE |
| 42 | B YEDUKONDALU | 49 | M | 152 | 112 | 104 | 198 | 149 | 39 | 138 | 32 | BRAIN STEM |
| 43 | J BHASKAR RAJU | 35 | M | 180 | 98 | 124 | 184 | 114 | 48 | 115 | 20 | TEMPOROPARIETAL |
| 44 | M SEETAIAH | 61 | M | 161 | 110 | 183 | 127 | 131 | 33 | 62 | 26 | THALAMUS |
| 45 | T CHAKRAPANI | 46 | M | 156 | 108 | 276 | 152 | 301 | 50 | 44 | 60 | PUTAMEN |
| 46 | M SUBBA LAKSHMI | 73 | F | 170 | 107 | 83 | 147 | 161 | 36 | 79 | 32 | FRONTOPARIETAL |
| 47 | K RAM MOHAN RAO | 75 | M | 140 | 99 | 102 | 122 | 154 | 36 | 52 | 36 | THALAMUS |
| 48 | T PATTABHI | 67 | M | 172 | 120 | 92 | 130 | 172 | 32 | 64 | 34 | TEMPOROPARIETAL |
| 49 | SK FATHIMA | 58 | F | 158 | 112 | 106 | 188 | 182 | 34 | 116 | 39 | MULTI SITE |
| 50 | NAGA BRAHMACHARI | 69 | M | 171 | 116 | 125 | 148 | 160 | 47 | 66 | 32 | PUTAMEN |