"THE EVALUATION OF EPICARDIAL FAT THICKNESS AND PLASMA FREE FATTY ACID LEVEL IN ACUTE ISCHEMIC STROKE- ONE YEAR CASE-CONTROL STUDY AT KLES Dr. PRABHAKAR KORE HOSPITAL AND MRC."

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DR. REKHA S PATIL
MD (GENERAL MEDICINE)
Professor and Head,
Department of General Medicine,
J. N. Medical College,
Nehru Nagar, Belagavi – 10

Date: 31/12/22
Place: Belagavi

KAHER * BELAGAVI * J.N. MEDICAL COLLEGE

DR.(Mrs.) N.S. MAHANTSHETTI
MD (PAEDIATRICS)
PRINCIPAL
J.N. Medical College,
BELAGAVI- 590 010
Principal
J. N. Medical College,
Nehru Nagar, Belagavi – 10

Date: 2/1/23
Place: Belagavi

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REG NO: BG0120018

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Nehru Nagar, Belagavi- 590 010, Karnataka, INDIA

0831 - 2471350 0831 - 2470759 www.jnmc.edu principal@jnmc.edu

Ref No: MDC/PG/ Date: 14-12-2022.

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J. N. Medical College, Belagavi.

To,
Reg. No. BG0120018,
Postgraduate Student,
2020-21 Batch,
Department of General Medicine,
J. N. Medical College, Belagavi.

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K.L.E. ACADEMY OF HIGHER EDUCATION AND RESEARCH
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JNMC INSTITUTIONAL ETHICS COMMITTEE
JAWAHARLAL NEHRU MEDICAL COLLEGE,
NEHRU NAGAR, BELAGAVI-590010 (KARNATAKA-INDIA)
Website: http//www.jnmc.edu
E-Mail : dome@jnmc.edu
Phone: (+ 91-(0)831 Office : 2472550
Principal: 2471701
Fax No. +91 (0)831 – 2470759

Ref No.MDC/JNMCIEC/ 254 **Date: 12/12/2022**

To,
REG NO: BG0120018
PG Student in General Medicine,
J. N. Medical College,
BELAGAVI.

Sub: Institutional Ethical Clearance for the study.

With reference to the above, we wish to inform you that your proposed research project titled **"THE EVALUATION OF EPICARDIAL FAT THICKNESS AND PLASMA FREE FATTY LEVEL IN ACUTE ISCHEMIC STROKE- ONE YEAR CASE –CONTROL STUDY AT KLES DR. PRABHAKAR KORE HOSPITAL AND MRC.",** is ethical and justifiable. The proposed research project has been cleared by the JNMC Institutional Ethics Committee.

(Dr. Smita Sonoli)
Member Secretary
JNMC Institutional Ethics Committee
J.N.Medical College, Belagavi.

(Dr. Harsha Hegde)
Chairman,
JNMC Institutional Ethics Committee
J.N.Medical College, Belagavi

ABSTRACT

Background: Stroke, a chronic disease with acute events causes significant morbidity among individuals. It increases the risk of psychological, emotional problems, physical disability and overall quality of life. The present study was conducted to evaluate the relationship between epicardial fat thickness (EFT) and plasma free fatty acid (PFFA) level in acute ischemic stroke and to further evaluate the effect of demographic and clinical characteristics of stroke on EFT and PFFA levels

Methods: The present case control study was conducted at KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belagavi between January 2021 to December 2021. Thirty-three adult patients admitted clinical and radiologically proven diagnosis of acute ischemic stroke and 33 patients age and sex matched healthy adults were included as controls. Patient demographics, family and medication history, blood parameters including total cholesterol, low-density lipoprotein cholesterol (LDL), high-density lipoprotein cholesterol (HDL), triglycerides, fasting blood glucose levels, epicardial fat thickness and plasma Free fatty acid levels were measured.

Results: Mean (SD) age of patients was significantly higher among cases as compared to controls (60.9 [11.8] years vs 51.4 [12.7] years; p=0.005).60.6% had anterior circulation stroke, 36.3% had posterior circulation stroke, and 3.1% had watershed area infarction. The mean (SD) FBS (173.5 [78.3] vs 110.3 [15.9%] mg/dl; p=0.000), TC (164.2 [50.5] vs 141.1 [31.4] mg/dl; p=0.022), LDL (94.2[45.9] vs 99.5 [9.8] mg/dl; p=0.514) and triglycerides (164.2 [50.5] vs 141.1 [31.4] mg/dl; p=0.022 were significantly higher in cases than those of controls while the mean (SD) of HDL (41.6 [32.7] vs 59.5 [8.6] mg/dl; p=0.024), was greater in controls than the cases .The mean (SD) EFT (11.87 [3.14] mm vs 7.11 [0.93]mm; p=0.000) and PFFA (0.24 [0.05]

vs 0.20 [0.06]; p=0.018) was much higher among cases than controls. Intergroup comparisons showed, female gender, statin therapy, increase in FBS, LDL, triglycerides, and decrease in HDL were significantly associated with EFT and PFFA levels ($p<0.05$). However, intragroup comparisons of effect of clinical and laboratory parameters on EFT and PFFA showed no relationship between variables such as sex, diabetes, statin therapy, FBS, HDL, LDL and triglyceride levels on EFT and PFFA ($p>0.05$).

Conclusion: EFT and PFFA levels can be considered an independent risk factor of ischemic stroke among adult patients. Further, large epidemiological, prospective studies are warranted to establish the relationship.

Keywords: epicardial fat thickness, ischemic stroke, low-density lipoprotein cholesterol, plasma free fatty acid levels, risk factor

ABBREVIATIONS

AF- atrial fibrillation

ARIC- Atherosclerosis Risk in Communities

CI- confidence interval

CE- cardioembolic

CT- computed tomography

DALY- disability adjusted life years

DIP- dipping effect

EFT- Epicardial fat thickness

ESUS- embolic stroke of undetermined score

FBS- fasting blood sugar

HDL- High Density lipoprotein cholesterol

HR- Hazard ratio

ICH- intracerebral hemorrhage

IS- ischemic stroke

LDL- low-density lipoprotein cholesterol

MRI- magnetic resonance imaging

NLR- neutrophil/lymphocyte ratio

pEATT- periatrial epicardial adipose tissue thickness

PFFA- plasma free fatty acids

SD- standard deviation

TC-Total cholesterol

Mmol/l- micromoles per liter

TABLE OF CONTENTS

LIST OF TABLES

List of Graphs

List of Figures

INTRODUCTION

"THE EVALUATION OF EPICARDIAL FAT THICKNESS AND PLASMA FREE FATTY ACID LEVEL IN ACUTE ISCHEMIC STROKE- ONE YEAR CASE-CONTROL STUDY AT KLES Dr. PRABHAKAR KORE HOSPITAL AND MRC."

INTRODUCTION

Stroke, also known as a cerebrovascular accident occurs secondary to rapid loss of brain function due to disturbance in the blood supply of the brain. It is one of the leading causes of morbidity and mortality worldwide [1]. According to GBD 2019 report, there was approximately 12 million incident strokes, 101 million prevalent strokes, 143 million disability adjusted life years (DALYs) and 6 million deaths from stroke reported in 2019. The incidence of ischemic stroke was substantially higher constituting 62% compared to other types of strokes, namely; intracerebral haemorrhage (28%) and subarachnoid haemorrhage (10%) [2]. The burden of stroke in India is on the rise. The crude incidence and prevalence rates of stroke in India ranges from 108 to 172 per 1,00,000 person per year and 26 to 757 per 1,00,000 person per year, respectively [3].

Waist circumference and echocardiographic epicardial fat are identified as the independent risk factors of stroke [4]. Epicardial fat is a metabolically active tissue which is present between the outer wall of myocardium and the visceral layer of pericardium with the free fatty acids being its major component [5]. While, under physiological conditions the epicardial fat protects and supports the heart during normal function, increased amounts of epicardial fat are related to increased

production of proinflammatory and proatherogenic mediators, leading to development of atherosclerosis, coronary artery disease, metabolic syndrome and obesity [6, 7]. However, since the atherosclerotic process begins years before the development of clinical cerebrovascular disease, and hence the markers such as epicardial fat thickness (EFT) and plasma free fatty acid can be used as a screening tool in early prediction of stroke.

Stroke not only increases morbidity of an individual, but also increases the risk of psychological problems which are often neglected and inadequately treated. Studies indicate a direct relationship between severity of stroke and physical disability, psychological and emotional problems and overall, the quality of life in patients. Moreover, there is increased healthcare and social burden [8, 9]. It is said that “time is brain”; therefore, early screening is required to prevent stroke. Additionally, understanding the pathophysiology and identifying the risk factors are of paramount importance. This further enables the policymakers to propose and initiate preventive and screening programs which helps to identify the population at risk. Although the relationship between EFT and cardiovascular disease is apparent, however, its role in ischemic stroke is not clear. In view of this, the present study was conducted to evaluate the relationship between epicardial fat thickness and plasma free fatty acid level in acute ischemic stroke.

OBJECTIVES

- To evaluate the association of epicardial fat thickness and plasma free fatty acid levels in patients with acute ischemic stroke.

REVIEW OF LITERATURE

Stroke is a cerebrovascular disorder characterized by sudden onset of clinical signs and symptoms. Occurrence of stroke negatively impacts the individual's and caretaker's psychological, physical and social health. Furthermore, with associated costs of treatment it affects the healthcare system and socioeconomic status of the country. The below section outlines the brief overview of stroke, risk factors and role of epicardial fat thickness and plasma free fatty acid in acute ischemic attack.

Definition of stroke

The World Health Organization defines stroke as "rapidly developing clinical signs of focal (or global) disturbance of cerebral function, with symptoms lasting 24 hours or longer or leading to death, with no apparent cause other than vascular origin" [10]. Stroke council of the American Heart Association and American Stroke Association updated the definition as "a transient episode of neurological dysfunction caused by focal brain, spinal cord or retinal ischemia, without acute infarction" [11].

History

The diagnostic term "apoplexy" is in existence for a long time and can be referred back from the Hippocratic writings. Apoplexy describes a condition in which patient experiences "sudden abolition of all activities of mind with the preservation of pulse and respiration"[12]. The lay term stroke related to apoplexia was first used in 1599 as "a stroke of God's hande" to describe the sudden onset of symptoms. Over the years, with the increase in number of autopsies led to better understanding of the pathologic and clinical characteristics of apoplexy. However, only in the mid-20th Century, apoplexy was replaced by the term stroke in the medical lexicons. Figure 1 briefly depicts the historical timeline.

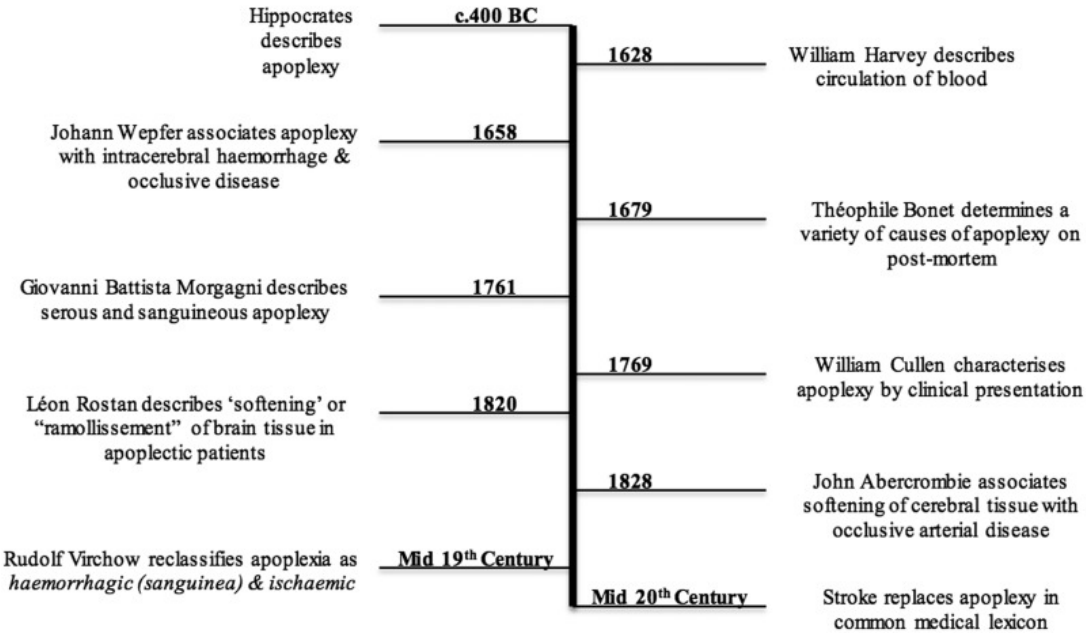


Figure 1: Timeline of stroke in medical history [13]

Epidemiology of stroke

Globally, stroke is the second leading cause of death after ischemic heart disease. According to the 2019 Global Burden of Disease Study, the reported incident and prevalent stroke in the year 2019 were 12.2(95% UI: 11.0-13.6) million and 101(95% UI: 93.2-111) million, respectively. Additionally, there was 143 (95% UI: 133-153) disability adjusted life years and 6.6 (95% UI: 6.0 to7.0) million deaths related to stroke [2]. In a systematic review of population based studies, Feigin et al reported a 42% decrease in incidence of stroke in high income countries. On the contrary, the incidence of stroke in low to middle income countries was more than 100%, suggesting the burden in developing countries [14]. In India, the burden of stroke is on the rise. The crude incidence and prevalence rates of stroke ranges from 108 to 172 per 100,000 person per year and 26 to 757 per 100,000 person per year, respectively [3].

Types of stroke

Stroke is broadly classified as

- Ischemic stroke which occurs secondary to blood clots causing blockage of blood and oxygen supply to the affected area. It accounts for 80-90% of the cases. Ischemic strokes are mainly related to small artery occlusion, large artery atheroma and cardiac sources of embolism.
- Hemorrhagic stroke occurs secondary to rupture of intracerebral blood vessels and leakage of blood to the surrounding area. It accounts for 10-20% of the cases. Hemorrhagic stroke is related to subarachnoid hemorrhage and intracerebral hemorrhage of small arteries [15,16]. (Figure 2)

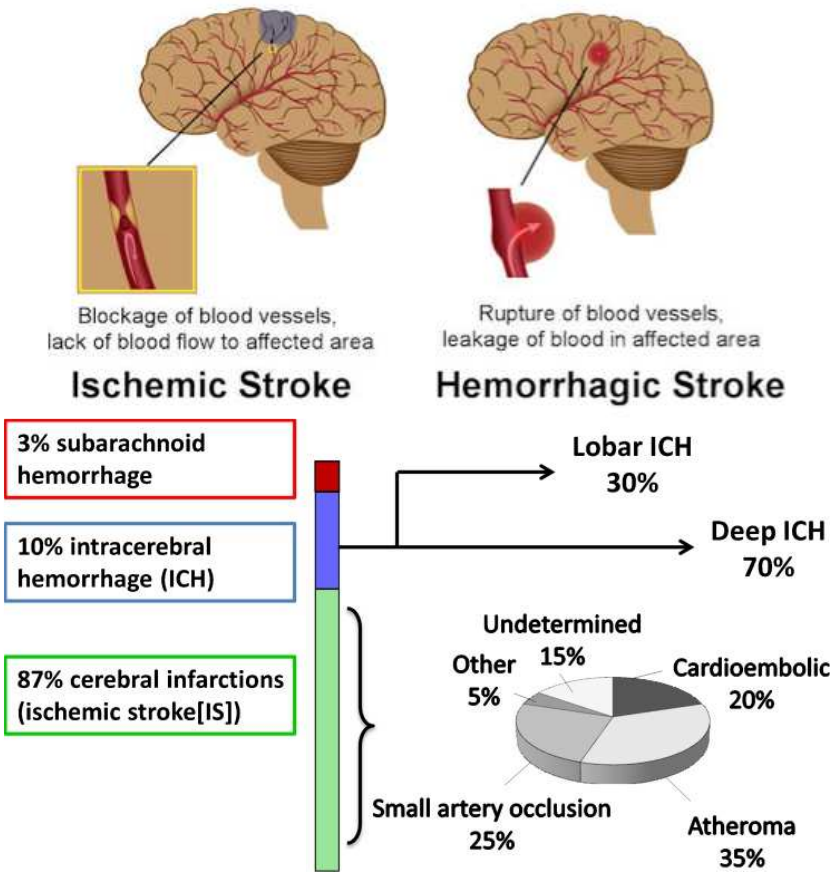


Figure 2: Types of stroke

Pathophysiology of stroke

In the brain, ischemic infarct results due to the occlusion of brain vessels secondary to thrombosis and embolism . In thrombosis, constant deposition of atherosclerotic plaque on the walls of blood vessels results in narrowing of the blood vessels, constriction of vascular lumen. This eventually results in thrombotic occlusion of the artery. Further, there is disruption of plasma membrane, organelle swelling and expulsion of content to extracellular space resulting in loss of neuronal function. Apart from these, inflammation, energy failure, loss of haemostasis, acidosis, increased intracellular calcium levels, excitotoxicity, free radical-mediated toxicity, cytokine-mediated cytotoxicity, complement activation, impairment of the blood–brain barrier, activation of glial cells, oxidative stress and infiltration of leukocytes are also related to pathophysiology of ischemic stroke [17, 18].

Haemorrhagic stroke occurs secondary to rupture of intracranial vessels and is associated with high mortality rate. Rupture of the blood vessels is due to hypertension, disrupted vasculature, excessive use of anticoagulants and thrombolytic agents . The rupture of blood vessels cause abnormal accumulation of blood within the brain, resulting in changes in vascular system and tissue necrosis. In subarachnoid haemorrhage, blood accumulates in the subarachnoid space of the brain due to a head injury or cerebral aneurysm [19]. The mechanism of stroke is depicted in Figure 3.

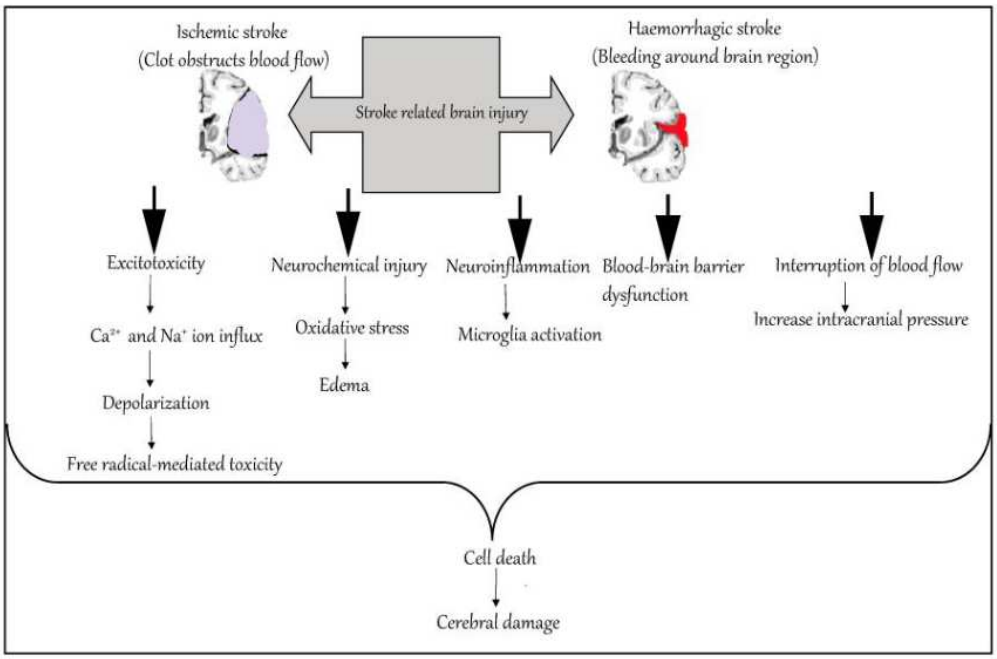


Figure 3: Pathophysiologic mechanism of stroke [17]

Pathophysiologic changes in ischemic stroke

At the onset of cerebral ischemia, there is continued consumption of ATP despite the reduced synthesis resulting in decreased ATP levels, lactic acidosis and loss of ionic haemostasis. This ionic imbalance further leads to neurotransmitter release and reuptake inhibition. Specifically, glutamate promotes increased calcium, sodium and water influx. While, the calcium overload results in degradation of cell membranes and proteins; excessive sodium and water influx results in swelling of cells, oedema and extracellular space shrinkage. Additionally, increased calcium, sodium and ATP levels in ischemic cells will stimulate mitochondrial production of oxygen free radicals which directly damage the carbohydrates, nucleic acid, lipids and proteins. The mechanism of ischemic cell death in white matter and gray matter differs. While the excessive calcium entry is via glutamate pathway in gray matter as

explained above, the increased intracellular calcium in white matter is associated with increased intracellular sodium and membrane depolarization [20-22].

Furthermore, following ischemic stroke or injury, cerebral inflammatory response sets in including primary activation of infiltrating immune cells including neutrophils, macrophages and lymphocytes with resultant sterile inflammation. Inflammation further triggers the tissue repair and is considered host defence mechanism [23]. The process is depicted in Figure 4.

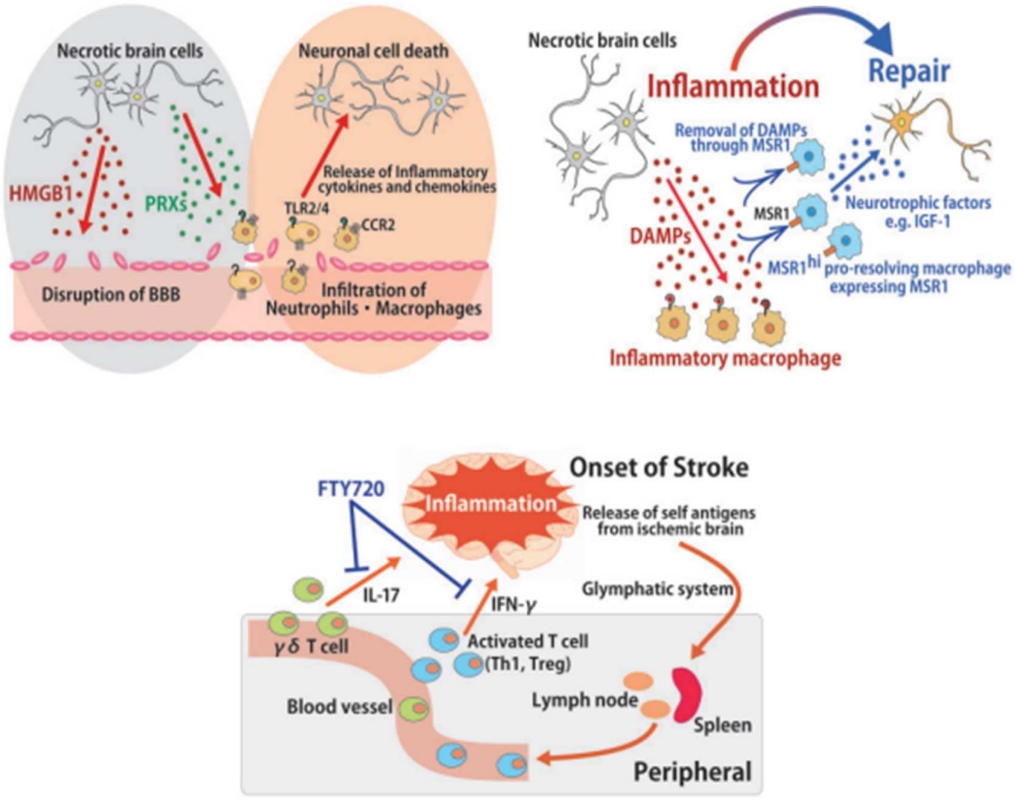


Figure 4: Immune response to ischemic stroke showing (a) Initiation of sterile inflammation (b) Change of macrophagic function from inflammation to repair (c) T cells showing neuroprotective effect against ischemic brain injury [23]

Risk factors of ischemic stroke

Previous studies have reported the following factor to pose higher risk of ischemic stroke [24, 25], which includes

- Age
- Males
- History of stroke and atrial fibrillation
- Family history of stroke
- Hypertension
- Diabetes
- Previous history of heart diseases
- Smoking
- Alcohol intake
- Obesity
- Lower physical activity
- Stress
- Lack of daily fruit and vegetable intake
- Dyslipidemia
- Altered parameters including increase in Lipoprotein A, C-reactive protein, Von Willebrand factor, homocysteine, and white blood cell count.

Apart from these well-known risk factors, recent literature also suggests the role of Epicardial fat thickness and plasma free fatty acids in ischemic stroke.

Epicardial adipose tissue

The adipose tissue of heart consists of epicardial and pericardial fat which are embryologically different. Epicardial fat is located between the myocardium and visceral pericardium, represents 20% of the heart mass. It comprises mainly of mesothelial cells, and is supplied by the branches of coronary arteries. In adult human heart it is mainly located in the atrioventricular and interventricular grooves and may extend to epicardial surface of myocardium. As there is no fascia separating the myocardium and epicardial fat could interact with each other via paracrine and vasocrine secretion of proinflammatory adipokines [26, 27]. Epicardial fat is actively involved in the lipid energy homeostasis. Some of the physiological and pathophysiological functions of epicardial fat includes [28-30]:

Some physiological functions-

- Acts as an energy source to myocardium and provides mechanical protection to coronary artery.
- It produces anti-angiogenic and anti-inflammatory adipokine.
- Modulates intra-myocardial fat content and protects against excess free fatty acids.

Pathological effects of increase epicardial fat thickness

- Source to produce proatherogenic and proinflammatory adipokines.
- Plays a role in excess free fatty acid synthesis and release.
- Previous studies have also reported a correlation between epicardial fat thickness and coronary artery disease, bi-ventricular hypertrophy, and atrial fibrillation.

The activity of lipoprotein lipase and acetyl CoA carboxylase are low in epicardial fat. Energy production of heart is dependent on the oxidation of FFA in the myocardium. Epicardial fat acts as a buffer by protecting the heart against exposure to excessive FFA levels [31].

Clinical implications of epicardial fat

Epicardial fat thickness increases with increasing age and is approximately 20% thicker in patients aged over 65 years and is linked to females [32, 33]. Previous studies have shown a direct relationship between epicardial fat and disease wherein, it produces various inflammatory factors. Epicardial fat is directly related to obesity and higher body mass index. Studies have reported positive relationship between increased epicardial fat and metabolic syndrome. Increased epicardial fat thickness is also associated with cardiac risk factors including higher fasting glucose, increase in C-reactive protein, decreased HDL. Furthermore, epicardial fat thickness is directly correlated with coronary artery diseases and cancers. [34, 35]. Epicardial fat thickness can be quantified using imaging techniques such as echocardiography, magnetic resonance imaging and non-contrast computed tomography [36].

Relationship between epicardial fat thickness and ischemic stroke

Studies have also evaluated the relationship between EFT and risk of stroke. In a systematic review of 80 studies conducted by Rosa IM, et al (2021) [37] evaluated the association between EFT and stroke, the following conclusions were drawn.

- Increased EFT may be associated with stroke.
- Echocardiography, CT or MRI can be used for the evaluation, results remain same.
- Concomitant presence of metabolic syndrome and atrial fibrillation increases the risk of stroke.

Plasma free fatty acids

Free fatty acids are non-esterified fatty acids circulating in the plasma and are bound to albumin. Based on the aliphatic tail length they are classified as short chain fatty acids (<6 carbon atoms), medium chain fatty acids (6-12 carbon atoms) and long chain fatty acids (>12 carbon atoms). Free fatty acids are one of the important energy sources for the body tissues. In addition to being an energy source, other critical functions of FFA's include, receptor signalling, gene expression, regulation of energy homeostasis. FFA's also act as natural ligand for group of G protein coupled receptors termed as FFA receptors, which regulate inflammation and secrete peptide hormones [38, 39]. Pathologically, FFA increases the oxidative stress and induces hypercoagulability [40]. Previous studies have suggested an association between FFA's and thrombus formation via platelet aggregation and activation of clotting factors. FFA's are also associated with metabolic syndrome, obesity, insulin resistance, myocardial dysfunction and cardiac diseases including cardiac arrythmias [41-43].

Free fatty acid as risk factor of ischemic stroke

Elevated FFA levels are also associated with atherosclerotic cerebrovascular accidents and is considered a predictor for atrial fibrillation associated stroke. Studies

have suggested an association between cerebrospinal FFA levels and functional outcome and recurrence in ischemic stroke patients [44, 45]. The link between FFA and stroke is mainly attributed to atherosclerosis and arrhythmia. While some studies have suggested impaired FFA metabolism to be related to arrhythmia [46], however the exact mechanism of FFA on stroke is yet to be elucidated.

Related studies

Seo WK, et al (2011) [47] conducted a cross-sectional study to evaluate the association between FFA concentration and ischemic stroke with further emphasis on ischemic stroke subtypes. Data of a total of 715 stroke patients from a hospital-based acute stroke registry were included for analysis. Compared to different stroke types, the cardioembolic stroke type has higher concentrations of FFA. After adjusting for covariates, the logistic regression analysis showed a positive association between the cardioembolic subtype and higher FFA concentration. Additionally, patients with AF had higher FFA concentrations than those patients without AF. However, presence of atherosclerotic stenosis did not affect the FFA concentrations in patients with intra and extracranial arterial atherosclerosis. The authors concluded that there is a significant correlation between the fasting FFA concentrations and cardioembolic stroke subtype.

Yamagishi K, et al (2013) [48] conducted a prospective study with an aim to evaluate the association between plasma free fatty acids and ischemic stroke among whites. A total of 3870 white patients, both males and females aged between 45 years and 64 years from the Minneapolis field centre of the Atherosclerosis Risk in Communities (ARIC) Study having a baseline measurement of plasma cholesterol ester and phospholipid during 1987 to 1989 were included in

the study. Patients were followed up till 2008 for incident ischemic stroke. Cox proportional model was used for the analysis. Over 22 years of follow up, a total of 168 ischemic strokes were reported. Upon adjusting for age and sex, plasma free fatty acids were positively associated with stroke with HR (95% CI) of 1.9 (1.2 to 3.0). Additionally, the study reported a positive linear association between palmitoleic acid and stroke (HR [95% CI]: 1.9 [1.2-2.9]), negative correlation between linoleic acid and stroke (HR [95% CI]: 0.6 [0.4-1.0]) and no relationship between ω-3 and ω-6 polyunsaturated fatty acids and ischemic stroke. These relations unchanged after adjusting for other cardiovascular risk factors. The authors concluded that among US cohorts of white patients, ischemic stroke is correlated with plasma saturated and monounsaturated fatty acids.

Akıl E, et al (2014) [49] carried out a cross-sectional study to evaluate the levels of EFT and neutrophil/lymphocyte ratio (NLR) in patients with ischemic stroke and to further evaluate the relationship between these inflammatory markers and ischemic stroke incidence. The study included cases having diagnosed as ischemic stroke (n=38) and age and sex matched controls (n=47). EFT was measured using Echocardiography and automated haematology analyser was used for blood parameters. The mean EFT was significantly lower in control group as compared to cases (4.9 vs 6.0 mm; $p<0.001$). Similarly, the mean NLR was also significantly lower among controls as compared to the cases (1.8 vs 2.5; $p<0.001$). Spearman's correlation analysis showed a mild but significant correlation between EFT and NLR ($p=0.006$). The study did not identify any confounding factors. Based on the positive association between EFT, NLR and ischemic stroke, the authors concluded that EFT is an inexpensive and readily available clinical marker which can be included as risk estimation marker for ischemic stroke.

Choi JY, et al (2014) [50] conducted a retrospective registry-based observational study to evaluate the relationship between plasma FFA levels and recurrent stroke in cardioembolic (CE) stroke patients. A total of 669 patients with acute ischemic stroke were included in the study. Patients were further divided into CE stroke and non- CE stroke groups for comparative analysis. Baseline mean plasma FFA concentration was higher in the CE stroke patients as compared to non-CE patients (1.0 vs 0.7 mEq/L). Based on a multivariate logistic regression analysis, higher FFA levels had higher risk of CE stroke with a hazard ratio (95% CI) of 2.1 (1.5 to 3.0). At a median follow up of 25 months, stroke recurrence rate was 8.4% and recurrence rate was comparable between CE and non-CE group (11% vs 8%; p=0.396). While there was no association between baseline FFA and risk of recurrence among non-CE patients, among the CE stroke group, elevated baseline FFA was an independent risk factor of recurrence with a hazard ratio (95% CI) of 2.7 (1.1 to 7.0). The authors concluded that elevated baseline FFA concentration could be considered an independent risk factor of recurrence and is a useful indicator to predict the recurrences in CE patients.

Khawaja O, et al (2014) [51] conducted a prospective cohort study to evaluate the association between plasma free fatty acids and incident stroke among elderly population. In this Cardiovascular Health Study, a total of 4369 patients at or above 65 years with both sex and age were included in the study. Plasma free fatty acid levels were measured based on 1992-93 examination and was further confirmed by a committee of experts including neurologists and neuroradiologists. Relative risk of stroke associated with free fatty acids was tested using cox regression analysis. The mean age of patients was 75 years. At a median follow up of 11 years, a total incident stroke was 732. With the increasing titres of plasma fatty acids, there was corresponding increase in crude incidence rates from 14

to 18 per 1000 person-years with an associated hazard ratio (95% CI) of 1.05 (0.97 to 1.14). Restriction to ischemic stroke and change in adiposity and diabetes had no effect on ischemic stroke incidence. The authors concluded that there is an association between plasma free fatty acids and ischemic stroke.

Choi JY, et al (2016) [52] conducted a study to evaluate the association between baseline FFA concentration with different stroke types including any stroke, ischemic stroke/systemic embolism (ISSE), or ischemic stroke among stroke survivors with atrial fibrillation (A-fib). A total of 279 stroke patients with A-fib were included in the study. At a median follow up of approximately 18 months, 22, 21, and 17 patients had incidence of any stroke, ISSE, and ischemic stroke, respectively. At the end of 1 and 3 years, the cumulative risk for any stroke, ISSE, and ischemic stroke were 5% vs 15%, 5% vs 12%, and 4% vs 11%, respectively. After adjusting covariates baseline FFA concentration (HR [95% CI]: 1.7 [1.1 to 2.8]) was associated with recurrence of any stroke. Although FFA showed a trend association with ISSE (HR [95% CI]: 1.6 [1.0 to 2.6]) and ischemic stroke (HR [95% CI]: 1.6 [1.0 to 2.7]), it was non-significant. Further analysis using adjusted models including CHADS2 or CHA2 DS2 -VASc score as a covariate confirmed that FFA as an independent predictor of any stroke and ischemic stroke, but not for ISSE.

Cho KI, et al (2018) [53] conducted a study to evaluate the association between echocardiographic epicardial fat thickness and plasma FFA levels and occurrence of ischemic stroke in atrial fibrillation. A total of 214 patients diagnosed with ischemic stroke from March 2011 to June 2014 were included in the study. The patients were further divided into 2 groups, ischemic stroke with atrial fibrillation and ischemic stroke without atrial fibrillation (AF) with 35 and 179 patients, respectively. The

study comprised of 40% women and the mean age of the patients was 66.8 years. Serum FFA and EFT was significantly higher among ischemic stroke with AF as compared to ischemic stroke without AF (1379.7 vs. 757.8 mEq/L, $p < 0.0001$) and (6.5 vs. 5.3 mm, $p < 0.001$), respectively. Based on the Multivariate logistic regression analysis, age, serum FFA and EFT were independently associated with ischemic stroke group with AF. The authors concluded that high levels of EFT and serum FFA levels were associated with ischemic stroke among AF patients.

Golovko SA, et al (2018) [54] conducted a preclinical study in an animal model to test the hypothesis that plasma free fatty acids are altered at the early stages of acute ischemic stroke. Upon two- hours onset of permanent middle cerebral artery occlusion, plasma sample was collected from the affected mice and from control animals. The FFA sample was compared between the sham mice and control mice. The authors reported two-to-three-fold increase in very long n-3 and n-6 FFA including 20:4n-6, 22:4n-6, 22:5n-6, and 22:6n-3 in mice with cerebral occlusion. These changes correspond to the FFA liberation from brain phospholipid hydrolysed under ischemic insult. The results of this preclinical experiment identified plasma FFA profile to be a potential biomarker for early ischemic stroke.

Gurdal A, et al (2018) [55] conducted a study to investigate the epicardial fat measurement among young patients with embolic stroke of undetermined source (ESUS). A total of 77 volunteers (patients with ESUS, n=40; controls, n=37) were included in the study. Mean age of patients were 43 years vs 38 years in cases and control groups, respectively. Echocardiography was used to assess the EFT. EFT was significantly higher among the patients with ESUS as compared to control group (5.5 vs 4.0 mm; $p<0.01$). Additionally, the authors reported a positive correlation between

EFT and serum C reactive protein ($p<0.05$). A cut off value of 4.6mm of EFT was identified as optimum to predict the ESUS, which had 88% sensitivity and 81% specificity. The authors concluded that echocardiographic EFT was significantly higher in young patients with ESUS than healthy controls and therefore, can be used as a novel marker in these patients.

Cosansu K, et al (2020) [56] conducted a cross-sectional study to evaluate the effectiveness of EFT in predicting acute ischemic stroke among patients with atrial fibrillation. The study included a total of 160 patients (cases: n=80, controls [age and sex matched]: n=80) were included in the study. Echocardiographic evaluations was performed within the three days of hospitalization and EFT was measured using standard methods from previous studies. Mean ± standard deviation (SD) of EFT among cases was 8.55 ± 1.08 mm and among controls was 5.90 ± 1.35 mm. The difference was statistically significant ($p < 0.0001$). This was further substantiated by a multivariable logistic regression analysis which confirmed EFT to be an independent predictor of acute ischemic stroke in patients with atrial fibrillation. The authors concluded that EFT can be used as a predictor of development of acute ischemic stroke in patients with AF.

A registry based retrospective observational study was conducted by **Eun MY, et al (2020)** [57] to evaluate the predictive value of FFA in ESUS. A total of 110 patients with ESUS confirmed based on laboratory findings and imaging were included in the study. Patients were further divided into two groups: ESUS with PES (n=49) and ESUS without PES (n=61). Clinical and laboratory findings were compared between 2 groups. Compared to patients with ESUS with PES, plasma FFA levels, systolic and diastolic blood pressure and left atrial enlargement were higher in patients with ESUS

without PES. This was further confirmed by a multivariable analysis that showed FFA levels (odds ratio [OR, 95% CI]: 10.4 [1.01 to 1.06]), diastolic blood pressure (OR [95% CI]: 1.4[1.4 to 1.9]) and left atrial volume index (OR [95% CI]:1.1[1.0 to 1.1]) were higher in patients with ESUS without PES. The authors concluded that higher levels of FFA, diastolic blood pressure and left atrial volume index can be considered risk factors of ESUS without PES and more large scale studies are warranted to further quantify the results.

Drozd A, et al (2022) [58] conducted a prospective study to evaluate the impact of FFA and the associated inflammatory metabolites on the risk of sleep apnoea in stroke patients. A total of 64 patients with ischemic stroke were included in the study. Based on the physiologic dipping effect (DIP), patients were further divided into DIP and non-DIP groups with 33 and 31 patients, respectively. Epworth sleepiness scale (ESS) was used for sleep analysis and all patients underwent 24-hour blood pressure measurement, FA gas chromatography and inflammatory metabolite measurements. In patients with non-DIP, C16:0 palmitic acid levels were comparatively higher while, C20:0 arachidic acid, C22:0 behenic acid and C24:1 nervonic acid levels were lower. Additionally, leukotriene B4 level was lower among non-DIP patients. In the DIP group, a positive correlation between ESS and FAs and derivatives was noted. Decreased inflammation and increased anti-inflammatory mediators from FAs favoured DIP. The authors concluded the potential association between FA levels and eicosanoids in the pathogenesis of the non-DIP phenomenon. Further studies are warranted.

Edsen F, et al (2022) [59] conducted a retrospective study to evaluate the relationship between epicardial adipose tissue and incidence of acute ischemic stroke. A total of

215 patients (atrial fibrillation related stroke, n=121; non cardioembolic stroke, n=94) were included in the study. On admission of these patients with large vessel occlusion, peri atrial epicardial adipose tissue thickness (pEATT) was measured using computed tomography angiography. Compared to non-cardioembolic patients, pEATT was high among patients with atrial fibrillation related stroke. The study reported that computed tomography measured left sided pEATT was an independent predictor of atrial fibrillation related stroke with an adjusted odds ratio (95% CI) of 1.3(1.1 to 1.5; p=0.012). The authors concluded that there is a positive relationship between epicardial adipose tissue and incidence of acute ischemic stroke.

In a prospective Kadoorie Biobank study, China, **Sun L, et al (2022)** [60] evaluated the associations of different polyunsaturated fatty acids (PUFA) and incidence of total stroke, ischemic stroke (IS), and intracerebral haemorrhage (ICH). A total of 10,563 Chinese patients who attended the 2013-14 resurvey were included in the study. Gas chromatography was used to measure the erythrocyte PUFA's. Mean age of patients was 58 years and mean BMI was 24 kg/m^2. At a median follow up of 4 years, 342 IS and 53 ICH incidences were reported among 8159 participants without prior history of diabetes or vascular diseases. Based on multivariable analysis, a positive correlation was noted between 18:2n-6 and ICH (HR = 2.3 [95% CIs 1.4 to 3.8]) was observed, however this parameter was inversely correlated with IS (0.7 [0.5 to 0.9]). Similarly, a positive correlation was noted between 20:3n-6 and IS (1.6 [1.3 to 2.0]) but not with ICH. These associations remained unchanged after adjusting for conventional CVD risk factors and dietary factors. The authors concluded that variable associations between PUFA and different stroke types needs further understanding of the stroke aetiology.

MATERIALS AND METHODS

Source of the data

All the patients admitted at KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belagavi with clinical and radiologically proven diagnosis of acute ischemic stroke.

Study Design

Case-Control study.

Study Period

January 2021 to December 2021

Sample size:

We used the below formula to estimate the sample size.

$$n = \frac{(r+1)\left(Z_{\alpha/2} + Z_{\beta}\right)^2}{r\left(\frac{|\mu_1 - \mu_2|}{\sigma}\right)^2}$$

$$d = \frac{|\mu_1 - \mu_2|}{\sigma}$$

Where,

$\mu 1$ is the mean of the first group

μ_2 is the mean of the second group

σ^2 is the common error variance, for 95% confidence level,

$Z_{\alpha/2}$ values are 1.96 and

Z_{β} value is 1.28 (for 90% power)

r is the allocation ratio, which is given as $r = n_1/n_2$.

Here we assume the difference between epicardial fat thickness between the groups as large, i.e., d = 0.8, with this assumed d, 95% confidence level and 90% power, we take allocation ratio for control to case as 1:1.

By imputing the values to the above formula an overall sample size of **66** was estimated.

- For cases group: n_1= **33 patients**
- For control group: n_2 =**33 patients**

Sampling Method

- Cases: Consecutive patients admitted with the diagnosis of acute ischemic stroke.
- Controls: Healthy controls with no history of diseases visiting the hospital.

Selection Criteria

Inclusion criteria

- All adult patients over 18 years of age admitted with the diagnosis of acute ischemic stroke

Exclusion criteria

Patients with the history the following diseases were excluded

- Transient ischemic attack, Recurrent stroke, Intracranial haemorrhage
- Congestive heart failure
- Hepatorenal dysfunction
- Rheumatologic disorder and immunological disorder
- Malignancy

Methodology

A detailed information on demographics, patient history and clinical baseline information was collected from all cases and controls. Following specific examinations and investigations were carried out.

Neurological assessment

On arrival to the hospital, all the patients were clinically assessed, with thorough assessment of symptom evolution. A complete neurological examination was performed and the patient underwent a Computed Topography of the brain or Magnetic Resonance Imaging of the brain with or without CT Angiography or MRI Angiography. Confirmed cases of acute ischemic stroke, after informed consent were enrolled in the study.

Laboratory parameters

On the first day of hospitalization, the patient's morning venous blood sample was taken after overnight fasting for investigations. The parameters that were tested with the standardized techniques were total cholesterol, low-density lipoprotein cholesterol (LDL), high-density lipoprotein cholesterol (HDL), triglycerides, fasting blood glucose levels. Plasma Free fatty Acid levels were measured by the enzymatic colorimetric test. The kit that was utilized for the estimation of the plasma free fatty acid level was Sigma-Aldrich MAK044-1KT.

Echocardiographic measurement

Standard two-dimensional echocardiography was done in all the cases and controls using GE Vivid S60N and probe MFSc. The echocardiography technician was blinded to the patient information. The patient was made to lie in the left lateral

decubitus position. Epicardial fat thickness is the space between the outer wall of the myocardium and the visceral pericardium, an echo-free space. It was measured from the free wall of the right ventricle at the end-systole in three cardiac cycles. The maximum EFT was measured from the point on the free wall of the right ventricle along the midline of the ultrasound beam perpendicular to the aortic annulus as the anatomic landmark.

Data collection

The following data was collected and entered in a case history proforma

- Baseline demographics: age and sex, education, number of spoken languages
- History of hypertension, diabetes, and related data
- Family history
- History of statin therapy
- History of smoking, alcohol, and tobacco consumption
- Type of diet
- Sleep adequacy
- General examination mainly focusing on the signs of atherosclerosis like arcus senalis, xanthoma, xanthelasma, locomotor brachialis.
- Vital signs including blood pressure, pulse rate, respiratory rate, and temperature.
- Laboratory investigations such as fasting blood sugar (FBS), Total cholesterol, HDL, LDL, and triglyceride levels
- Plasma free fatty acids
- Epicardial fat thickness

Ethical considerations

Institutional ethical clearance was obtained prior to initiation of the study. The details of the study were explained to the cases and controls and an informed consent was obtained from all the cases and controls.

Data handling

The collected data was entered in Microsoft excel and the related records were stored safely with no access to other study personnel.

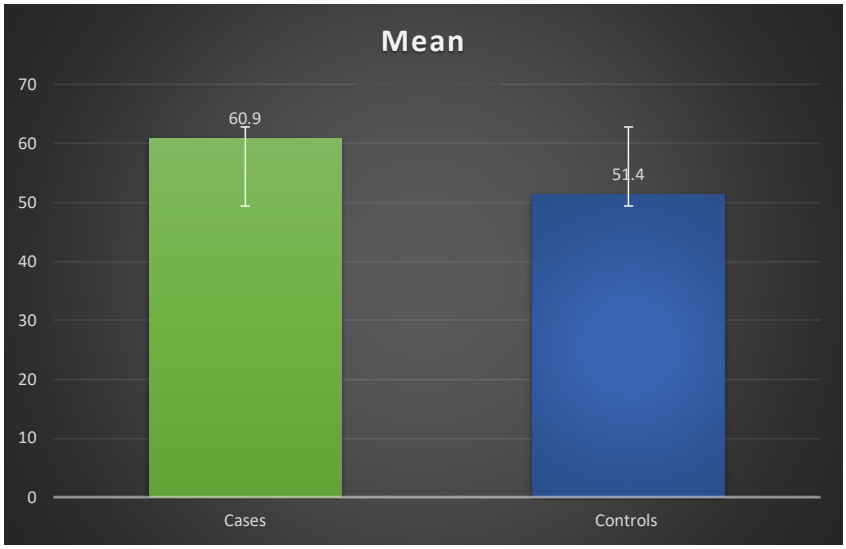
STATISTICAL ANALYSIS

All data was entered in the Microsoft excel sheet and then imported to SPSS version 22 software for statistical analysis. Categorical variables were summarized as frequency and percentages. Continuous variables were presented as Mean and standard deviation or median (minimum, maximum) values. Chi-square test was used to check the association of outcome among categorical variables. ANOVA test was used to compare the association between categorical and continuous variable. p-value less than or equal to 0.05 indicates statistical significance.

RESULTS

Table 1: Comparison of mean and median ages

Age, in years	Cases (n=33)	Controls (n=33)	p value
Mean (SD)	60.9 (11.8)	51.4 (12.7)	0.005
Median (min-max)	63 (36-82)	52 (26-75)	-

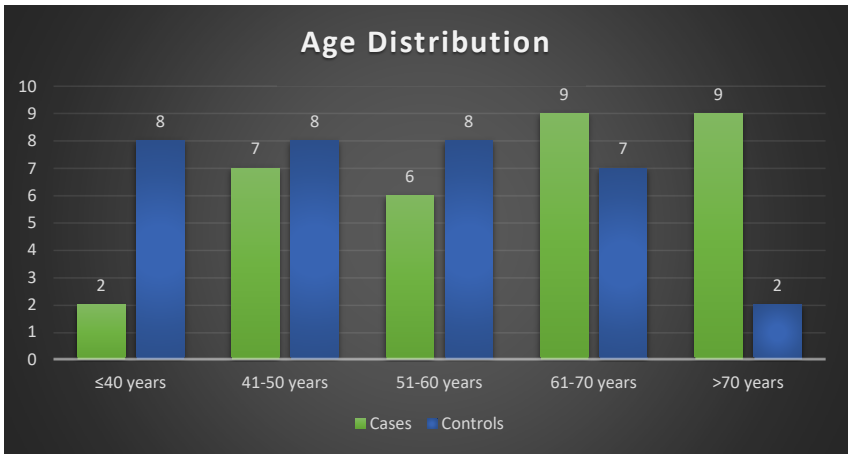


Graph 1: Bar diagram showing comparison of mean age (Error bar shows standard deviation)

In our study a total of 66 patients were included (cases, n=33 and controls, n=33). Mean (SD) age of patients was significantly higher among cases as compared to controls (60.9 [11.8] years vs 51.4 [12.7] years; p=0.005).

Table 2: Age distribution of cases and controls

Age, n (%)	Cases (n=33)	Controls (n=33)	p value
≤40 years	2 (6.1%)	8 (24.2%)	0.070
41-50 years	7 (21.2%)	8 (24.2%)	
51-60 years	6 (18.2%)	8 (24.2%)	
61-70 years	9 (27.3%)	7 (21.2%)	
>70 years	9 (27.3%)	2 (6.1%)	
Total	**33 (100%)**	**33 (100%)**	

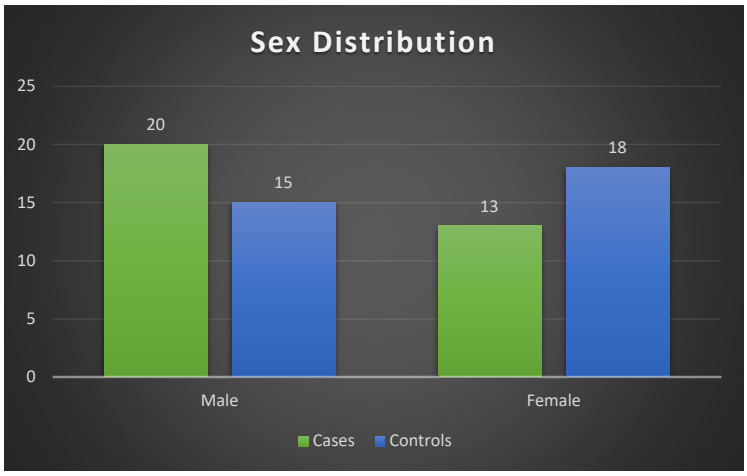


Graph 2: Bar diagram showing age distribution

Among cases, most patients belonged to the age group of 61-70 years and >70 years followed by 51-60 years. Among controls, subjects were younger and belonged to the age group of ≤40 years, 41-50 years and 51-60 years. However, the difference was not statistically significant (p=0.070)

Table 3: Sex distribution among study population

Sex, n (%)	Cases (n=33)	Controls (n=33)	p value
Male	20 (60.6%)	15(45.5%)	0.324
Female	13 (39.4%)	18 (54.5%)	
Total	**33 (100%)**	**33 (100%)**	

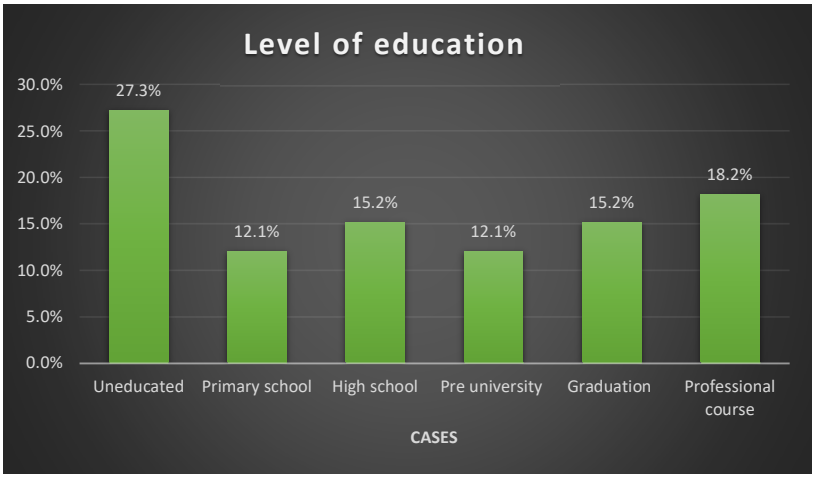


Graph 3: Bar diagram showing Sex distribution among study population

In our study, case groups comprised of 20 (60.6%) male patients and 13 (39.4%) female patients and control groups comprised of 15 (45.5%) males and 18 (54.5%) females. The difference was not statistically significant (p=0.324).

Table 4: Distribution of cases based on level of education among cases

Cases (n=33)		Frequency	Percentage
Level of education	**Uneducated**	9	27.3%
	Primary school	4	12.1%
	High school	5	15.2%
	Pre university	4	12.1%
	Graduation	5	15.2%
	Professional course	6	18.2%
	Total	**33**	**100.0%**

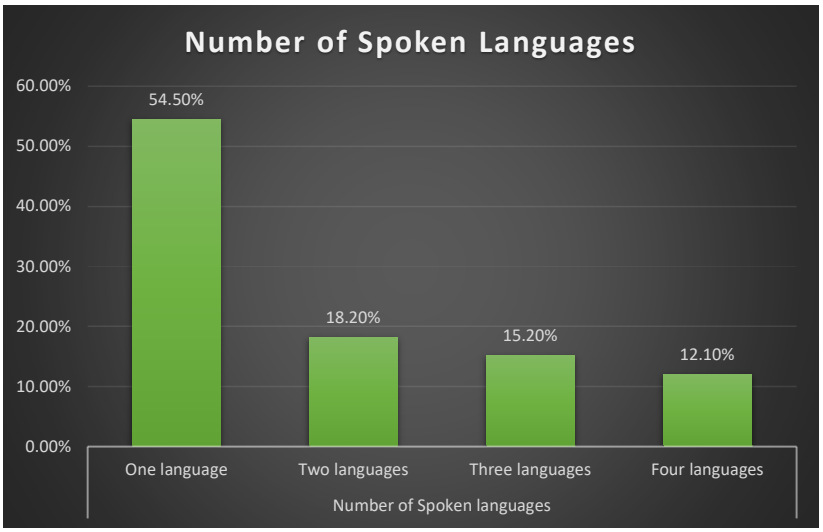


Graph 4: Frequency distribution of cases based on level of education

Out of 33 cases, 9 (27.3%) were uneducated, 4 (12.1%) had completed primary school, 5 (15.2%) had completed high school, 4 (12.1%) had completed preuniversity, 5 (15.2%) had completed graduation, and 6 (18.2%) were professionals.

Table 5: Distribution of cases based on number of spoken languages

Cases (n=33		Frequency	Percentage
Number of Spoken languages	**One language**	18	54.5
	Two languages	6	18.2
	Three languages	5	15.2
	Four languages	4	12.1
	Total	**33**	**100.0**

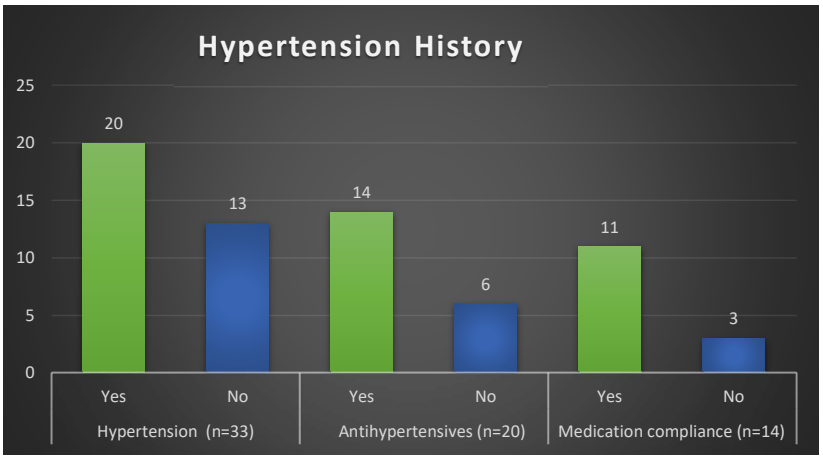


Graph 5: Bar diagram showing distribution of cases based on number of spoken languages

Most patients among cases spoke one language (n=18, 54.5%), followed by two languages (n=6, 18.2%), three languages (n=5, 15.2%) and four languages (n=4, 12.1%). Most common spoken languages were either Kannada or Marathi, while English and Hindi were spoken by limited patients.

Table 6: History of hypertension among cases

Cases (n=33)		Frequency	Percentage
Hypertension	**Yes**	20	60.6%
	No	13	39.4%
Duration of hypertension	**Mean (SD)**	6.8 (8.4) years	
Antihypertensives therapy (n=20)	**Yes**	14	70%
	No	6	30%
Medication compliance (n=14)	**Yes**	11	78.6%
	No	3	21.4%



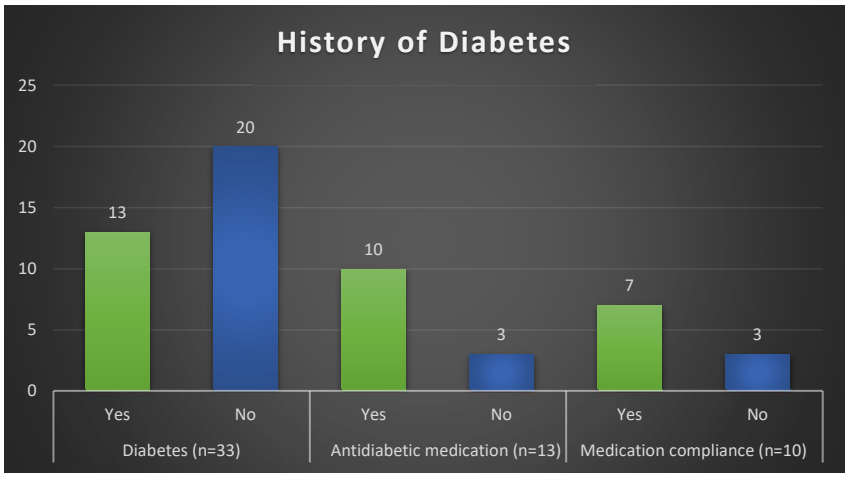
Graph 6: Frequency distribution of hypertension among cases

Among cases, 20 (60.6%) of patients had history of hypertension. The mean (SD) duration of hypertension was 6.8 (8.4) years. Among patients with hypertension, 14 (70%) were on antihypertensives and 6 (30%) were not on anti- hypertensive medication. Of the 14 patients, medication compliance was seen in 11 (78.6%) patients and 3 (21.4%) patients were not taking medications.

Commented [K1]: Please confirm

Table 7: History of diabetes among cases

Cases (n=33)		Frequency	Percentage
Diabetes	**Yes**	13	39.4%
	No	20	60.6%
Duration of Diabetes	**Mean (SD)**	4.5 (7.1) years	
Antidiabetic medication (n=13)	**Yes**	10	76.9%
	No	3	23.1%
Medication compliance (n=10)	**Yes**	7	70%
	No	3	30%



Graph 7: Frequency distribution of diabetes among cases

Among cases, 13 (39.4%) of patients had history of diabetes. The mean (SD) duration of hypertension was 4.5 (7.1) years. Among patients with diabetes, 10 (76.9%) were on anti-diabetics and 3 (23.1%) were not on anti- diabetic medication. Of the 10 patients, medication compliance was seen in 7 (70%) patients and 3 (30%) patients were not taking medications.

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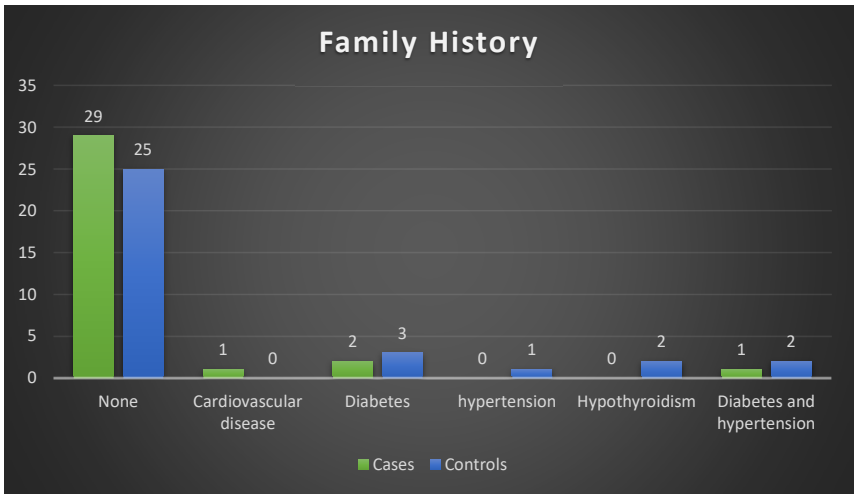
Table 8: Comparison of family history between cases and controls

Family history, n (%)	Cases (n=33)	Controls (n=33)	p value
None	29 (87.9%)	25 (75.8%)	0.437
Cardiovascular disease	1 (3.0%)	0 (0%)	
Diabetes	2 (6.1%)	3 (9.1%)	
hypertension	0 (0%)	1 (3.0%)	
Hypothyroidism	0 (0%)	2 (6.1%)	
Diabetes and hypertension	1 (3.0%)	2 (6.1%)	
Total	**33 (100%)**	**33 (100%)**	

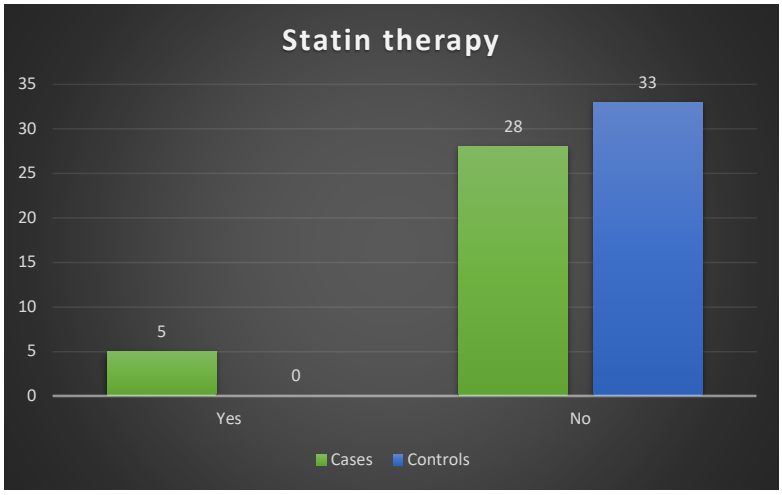


Graph 8: Bar diagram showing comparison of family history between groups

Among cases, 2(6.1%) patients had family history of diabetes, 1 (3.0%) each had history of cardiovascular disease and combination of diabetes and hypertension. Among controls, 3 (9.1%) subjects had diabetes, 2 (6.15) each, subjects had hypothyroidism and combination of diabetes and hypertension. The distribution of family history between cases and controls were comparable (p=0.437).

Table 9: Comparison of statin therapy between cases and controls

Statins, n (%)	Cases (n=33)	Controls (n=33)	p value
Yes	5 (15.2%)	0 (0%)	0.027
No	28 (84.8%)	33 (100%)	
Total	**33 (100%)**	**33 (100%)**	

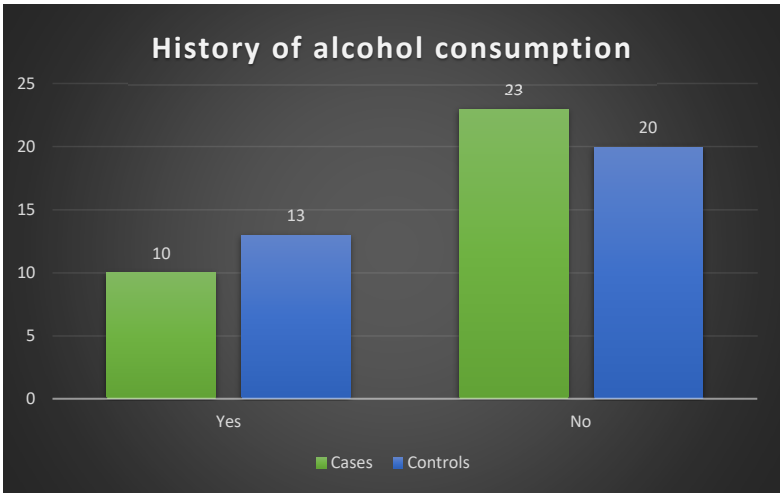


Graph 9: Bar diagram showing distribution of statin therapy between groups

None of the subjects in control group were on statin therapy. Among cases, out of 33 patients, 5 (15.2%) patients were on statins. The difference between groups was statistically significant (p=0.027).

Table 10: Comparison of history of alcohol consumption between cases and controls

Alcohol consumption, n (%)	Cases (n=33)	Controls (n=33)	p value
Yes	10 (30.3%)	13 (39.4%)	0.606
No	23 (69.7%)	20 (60.6%)	
Total	**33 (100%)**	**33 (100%)**	

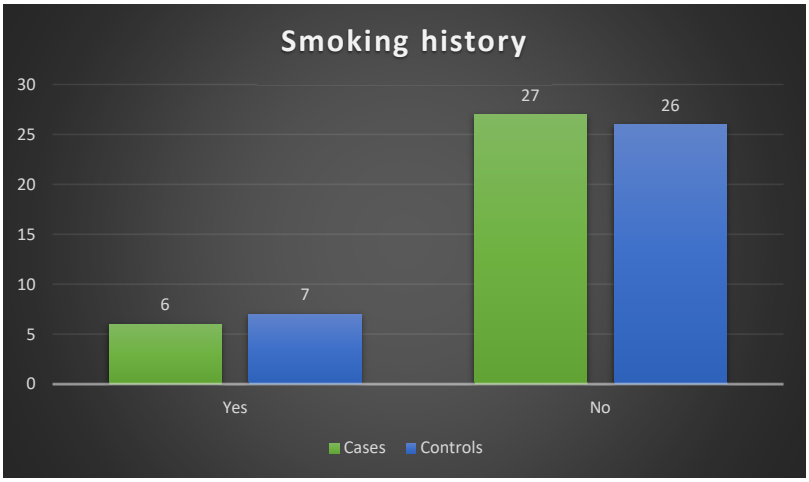


Graph 10: Bar diagram showing distribution of cases and controls based on history of alcohol consumption between groups

Among cases, 10 (30.3%) patients had history of alcohol consumption. Among controls, 13 (39.4%) of subjects had history of alcohol consumption. The difference between groups was not statistically significant (p=0.606).

Table 11: Comparison of history of smoking between cases and controls

Smoking history, n (%)	Cases (n=33)	Controls (n=33)	p value
Yes	6 (18.2%)	7 (21.2%)	1.000
No	27 (81.8%)	26 (78.8%)	
Total	**33 (100%)**	**33 (100%)**	

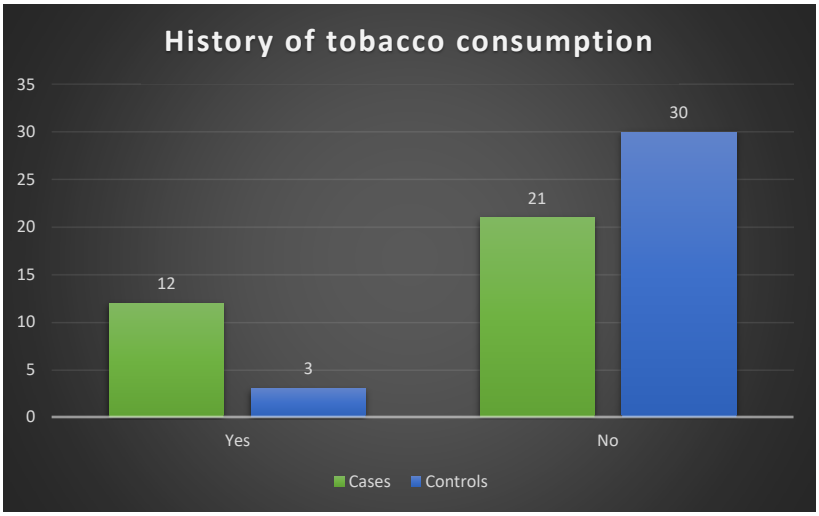


Graph 11: Bar diagram showing distribution of cases and controls based on history of smoking between groups

Among cases, 6 (18.2%) patients had history smoking. Among controls, 7 (21.2%) had history of smoking. The difference between groups was not statistically significant (p=1.000).

Table 12: Comparison of history of tobacco consumption between cases and controls

History of tobacco consumption, n (%)	Cases (n=33)	Controls (n=33)	P value
Yes	12 (36.4%)	3 (9.1%)	0.017
No	21 (63.6%)	30 (90.9%)	
Total	**33 (100%)**	**33 (100%)**	

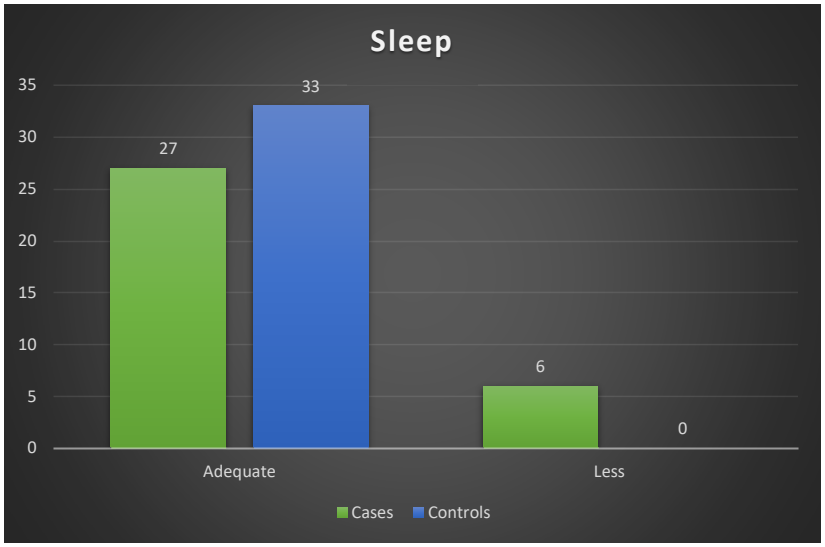


Graph 12: Bar diagram showing distribution of cases and controls based on history of tobacco consumption between groups

In our study, frequency of patients with tobacco consumption was much higher among cases as compared to controls (12 [36.4%] vs. 3 [9.1%]). The difference between groups was statistically significant (p=0.017).

Table 13: Comparison of sleep between cases and controls

Sleep, n (%)	Cases (n=33)	Controls (n=33)	p value
Adequate	27 (81.8%)	33 (100%)	0.024
Less	6 (18.2%)	0 (0%)	
Total	**33 (100%)**	**33 (100%)**	

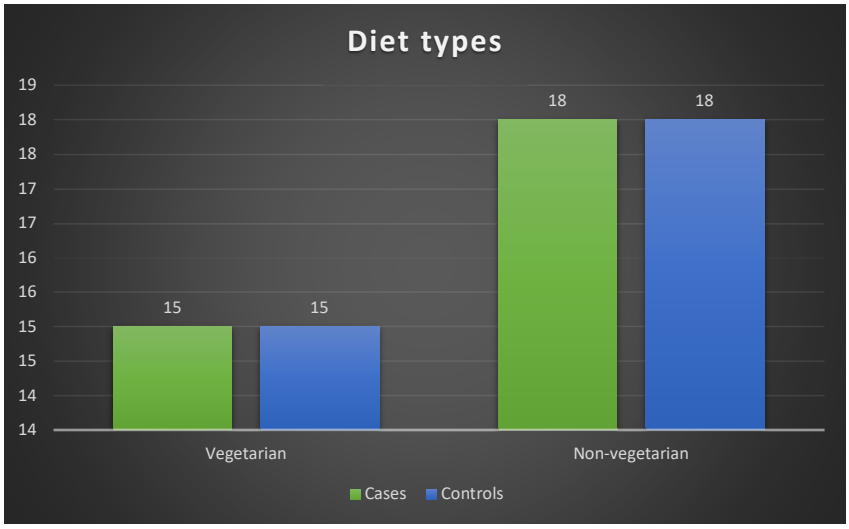


Graph 13: Bar diagram showing distribution of cases and controls based on sleep

All subjects in the control group had adequate sleep. Among cases, 27 (81.8%) had adequate sleep and only 6 (18.2%) had inadequate sleep. The difference between groups was statistically significant (p=0.024).

Table 14: Comparison of diet types between cases and controls

Diet, n (%)	Cases (n=33)	Controls (n=33)	p value
Vegetarian	15 (45.5%)	15 (45.5%)	1.000
Nonvegetarian	18 (54.5%)	18 (54.5%)	
Total	**33(100)**	**33(100)**	

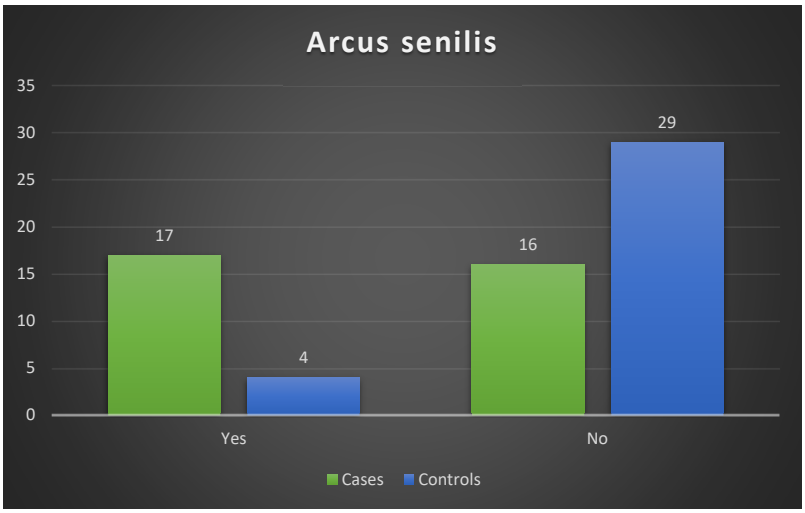


Graph 14: Bar diagram showing distribution of cases and controls based on diet types

Among cases, 15 (45.5%) patients were vegetarians and 18 (54.5%) patients were non-vegetarians. Similarly, 15 (45.5%) subjects were vegetarians and 18 (54.5%) subjects were non-vegetarians. The difference was not statistically significant (p=1.000).

Graph 15: Comparison of cases and controls with or without Arcus senilis between groups

Arcus senilis, n (%)	Cases (n=33)	Controls (n=33)	p value
Present	17(51.5)	4(12.1)	0.001
Absent	16(48.5)	29(87.9)	
Total	**33(100)**	**33(100)**	

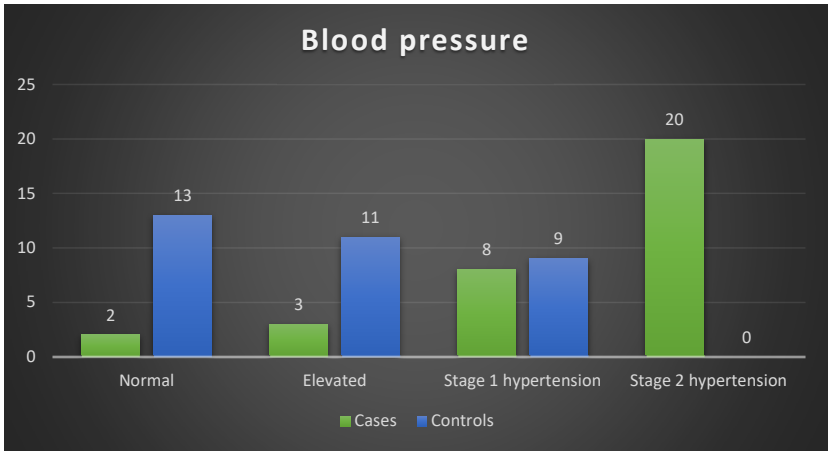


Graph 15: Bar diagram showing distribution of cases and controls based on presence or absence of arcus senilis.

In our study, 17 (51.5%) patients among cases and 4 (12.1%) subjects among controls had arcus senilis. The difference was statistically significant (p=0.001)

Table 16: Comparison of blood pressure between cases and controls

Blood pressure, mmHg, n (%)	Cases (n=33)	Controls (n=33)	p value
Normal	2 (6.1%)	13 (39.4%)	0.000
Prehypertension	3 (9.1%)	11 (33.3%)	
Stage 1 hypertension	8 (24.2%)	9 (27.3%)	
Stage 2 hypertension	20 (60.6%)	0 (0%)	
Total	**33(100)**	**33(100)**	

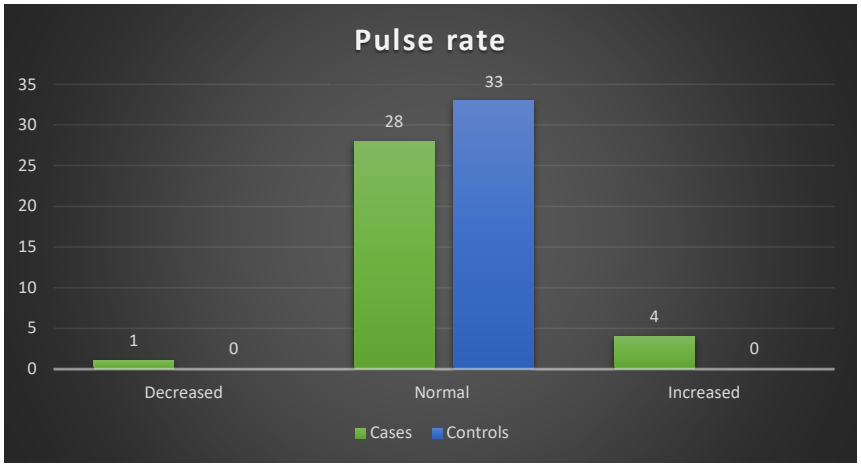


Graph 16: Distribution of cases and controls based on blood pressure reading

Among cases, 2 (6.1%) patients had normal blood pressure (<120/<80 mmHg), 3 (9.15%) patients had prehypertension BP (120-129/<80 mmHg), 8 (24.2%) patients had stage 1 hypertension (130-139/80-89 mmHg) and 20 (60.6%) had stage 2 hypertension (≥140/>90 mmHg). Among cases 13 (39.4%), 11 (33.3%), 9 (27.3%) had normal, elevated, and stage 1 hypertension, respectively. The difference between groups was statistically significant (p=0.000).

Table 17: Comparison of pulse rate between cases and controls

Pulse rate, per minute		Cases (n=33)	Controls (n=33)	p value
Mean (SD)		86.7 (14.8)	83.7 (8.4)	0.273
n (%)	**decreased**	1 (3.0%)	0 (0%)	0.000
	Normal	28 (84.8%)	33 (100%)	
	Increased	4 (12.1%)	0 (0%)	
	Total	**33(100%)**	**33(100%)**	

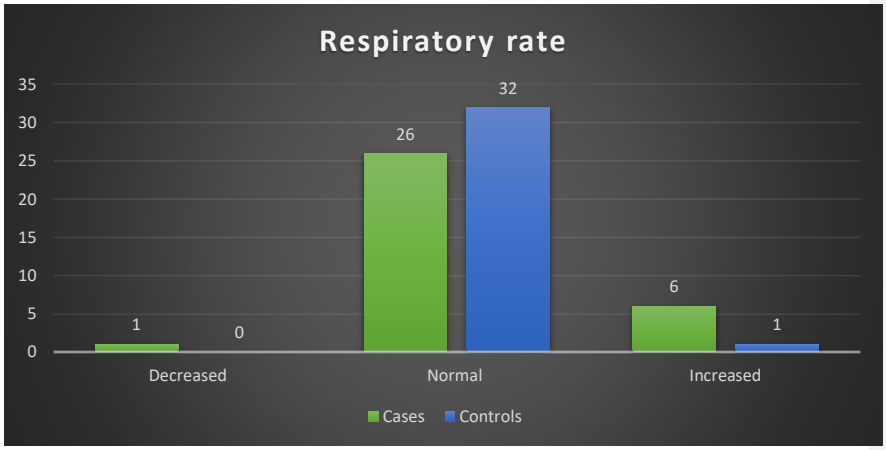


Graph 17: Distribution of cases and controls based on pulse rate

Mean (SD) pulse rate between cases and control was comparable (86.7 [14.8] vs 83.7 [8.4] per minute; p=0.273). Among controls, all subjects had normal pulse rate (60-100 per minute). Among cases, 28 (84.8%) patients had normal pulse rate, 1(3.0%) patient had decreased pulse rate (<60 per minute) and 4 (12.1%) patients had increased pulse rate (>100 per minute). The difference was statistically significant (p=0.000).

Table 18: Comparison of respiratory rate between cases and controls

Respiratory rate, cycles/min		Cases (n=33)	Controls (n=33)	p value
Mean (SD)		19.0 (3.4)	18.2 (1.3)	0.183
n (%)	**decreased**	1 (3.0%)	0 (0%)	0.075
	Normal	26 (78.8%)	32 (97.0%)	
	Increased	6 (18.2%)	1 (3.0%)	
	Total	**33(100%)**	**33(100%)**	

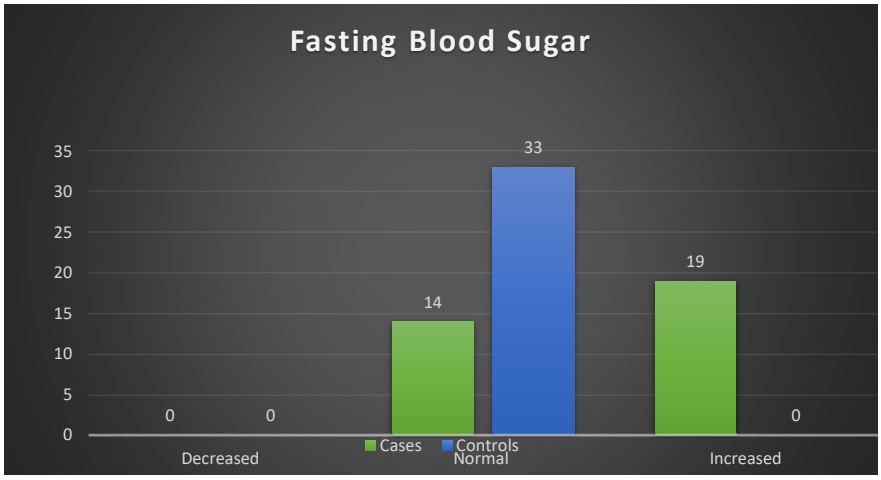


Graph 18: Distribution of cases and controls based on respiratory rate

Mean (SD) respiratory rate between cases and control was comparable (19.0 [3.4] vs 18.2 [1.3] cycles/min; p=0.183). Among controls, 32 (97.0%) subjects had normal pulse rate (16-20 cycles/min) and 1 (3.0%) subject had decreased respiratory rate (<16 cycles/min). Among cases, 26 (78.8%) patients had normal respiratory rate, while, 1(3.0%) patient and 6 (18.2%) patients had decreased and increased respiratory rate (>20 cycles/minute), respectively. However, the difference was statistically non-significant (p=0.075).

Table 19: Comparison of fasting blood sugar between cases and controls

FBS, mg/dl		Cases (n=33)	Controls (n=33)	p value
Mean (SD)		173.5 (78.3)	110.3 (15.9%)	0.000
n (%)	**Decreased**	0 (0%)	0 (0%)	0.000
	Normal	14 (42.4%)	33 (100%)	
	Increased	19 (57.6%)	0 (0%)	
	Total	**33(100%)**	**33(100%)**	

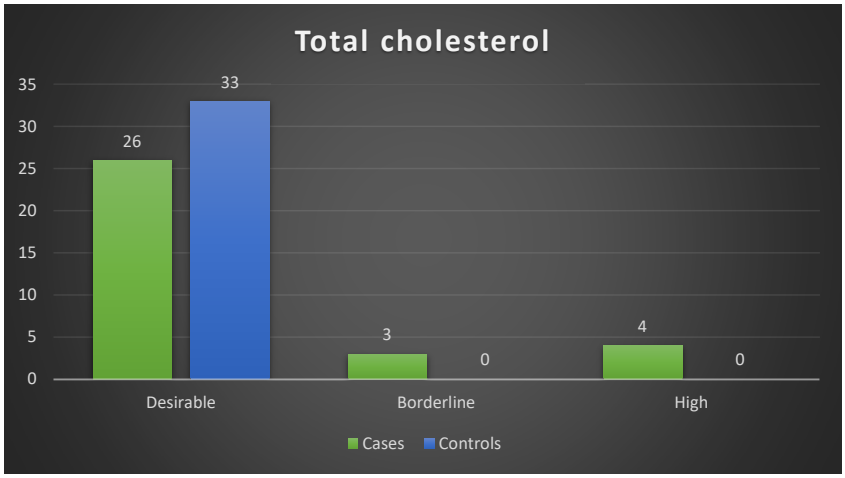


Graph 19: Distribution of cases and controls based on fasting blood sugar

The mean (SD) FBS among cases was significantly higher than those of controls (173.5 [78.3] vs 110.3 [15.9%] mg/dl; p=0.000). Normal FBS of 70-140 mg/dl was noted among 14 (42.4%) of cases and 33 (100%) of controls. Increased FBS of >140 mg/dl was seen among 19 (57.6%) of cases and none in the control group. The difference was statistically significant (p=0.000).

Table 20: Comparison of total cholesterol between cases and controls

Total cholesterol, mg/dl		Cases (n=33)	Controls (n=33)	p value
Mean (SD)		164.2 (50.5)	141.1 (31.4)	0.022
n (%)	**Desirable**	26(78.8%)	33(100%)	0.020
	Borderline	3(9.1%)	0(0%)	
	High	4(12.1%)	0(0%)	
	Total	**33(100%)**	**33(100%)**	

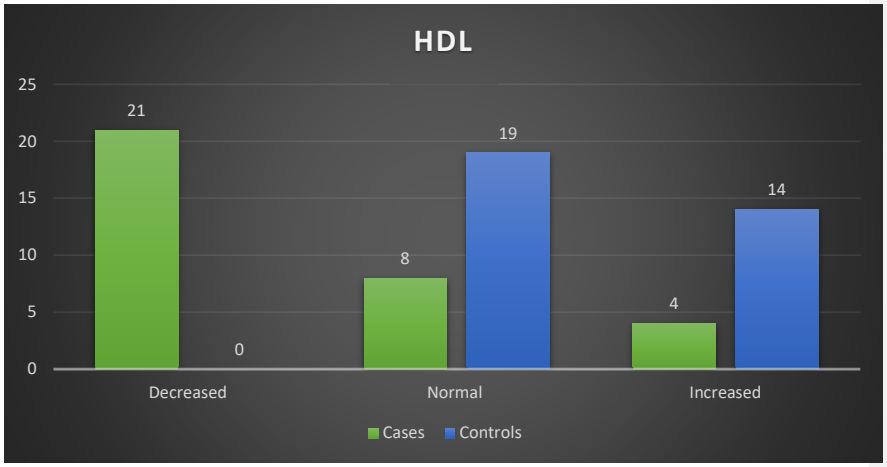


Graph 20: Distribution of cases and controls based on total cholesterol levels

The mean (SD) TC among cases was significantly higher than those of controls (164.2 [50.5] vs 141.1 [31.4] mg/dl; p=0.022). Desirable TC of <200 mg/dl was noted among 26 (78.8%) cases and 33 (100%) controls. Borderline TC of 200-239 mg/dl was noted in 3 cases(9.1%) and high TC count of ≥240 mg/dl was seen among 4 (12.1%) cases. The difference was statistically significant (p=0.020).

Table 21: Comparison of HDL between cases and controls

HDL, mg/dl		Cases (n=33)	Controls (n=33)	p value
Mean (SD)		41.6 (32.7)	59.5 (8.6)	0.024
n (%)	**Decreased**	21(63.6%)	0(0%)	0.000
	Normal	8(24.2%)	19(57.6%)	
	Increased	4(12.1%)	14(42.4%)	
	Total	**33(100%)**	**33(100%)**	

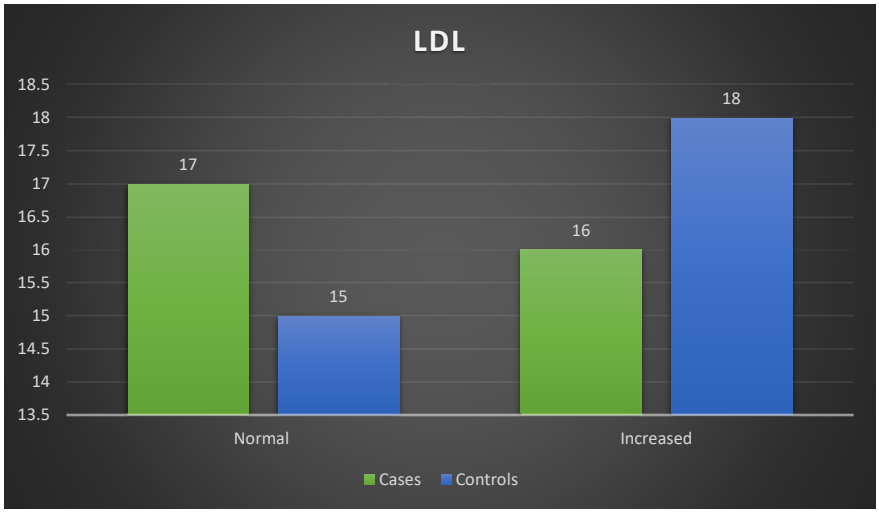


Graph 21: Distribution of cases and controls based on HDL

The mean (SD) HDL among cases was significantly lower than those of controls (41.6 [32.7] vs 59.5 [8.6] mg/dl; p=0.024). Normal HDL of 40-60 mg/dl was noted among 8(24.2%) cases and 19(57.6%) controls. Decreased HDL of <40 mg/dl was seen among (21[63.6%] vs 0[0%]) and increased HDL of >60 mg/dl (4[12.1%] vs 14[42.4%]) cases vs controls, respectively. The difference was statistically significant (p=0.000).

Table 22: Comparison of LDL between cases and controls

LDL, mg/dl		Cases (n=33)	Controls (n=33)	p value
Mean (SD)		94.2 (45.9)	99.5 (9.8)	0.514
n (%)	**Normal**	17(51.5%)	15(45.5%)	0.806
	Increased	16(48.5%)	18(54.5%)	
	Total	**33(100%)**	**33(100%)**	

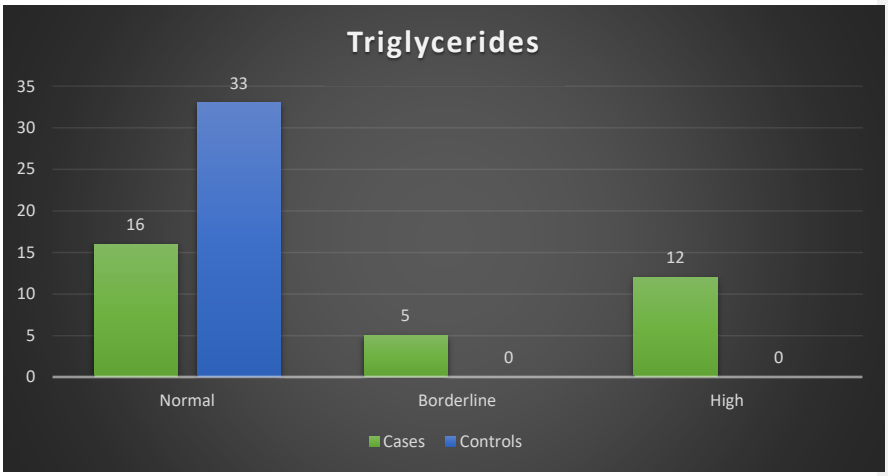


Graph 22: Distribution of cases and controls based on LDL

The mean (SD) LDL between groups was similar (94.2[45.9] vs 99.5 [9.8] mg/dl; p=0.514). Normal LDL of <100 mg/dl was noted among 17 (51.5%) cases and 15 (45.5%) controls. Increased LDL of ≥100 mg/dl was seen among 16 (48.5%) cases and 18 (54.5%) controls. The difference was not statistically significant (p=0.806).

Table 23: Comparison of triglycerides between cases and controls

Triglycerides, mg/dl		Cases (n=33)	Controls (n=33)	p value
Mean (SD)		175.4 (93.8)	106.6 (20.7)	0.000
n (%)	**Normal**	16(48.5%)	33(100%)	0.000
	Borderline	5(15.2%)	0(0%)	
	High	12(36.4%)	0(0%)	
	Total	**33(100%)**	**33(100%)**	



Graph 23: Distribution of cases and controls based on triglycerides

The mean (SD) Triglycerides among cases was significantly higher than those of controls (164.2 [50.5] vs 141.1 [31.4] mg/dl; p=0.022). All subjects in control group had normal triglyceride levels of <150mg/dl. Among cases, 16(48.5%) had normal triglycerides, 5(15.2%) had borderline high levels of 150-199 and 12(36.4%) had high triglyceride levels of 200-499 mg/dl. The difference was statistically significant (p=0.000).

Table 24: Comparison of mean EFT between groups

	Cases (n=33)	Controls (n=33)	P value
EFT, Mean (SD)	11.87 (3.14)	7.11 (0.93)	0.000

The mean (SD) EFT among cases was 11.87 mm (3.14) which was much higher than the mean EFT among controls (7.11mm [0.93]). The difference was statistically significant (p=0.000).

Commented [K3]: Please give unit of measurement for EFT

Table 25: Comparison of mean PFFA between groups

	Cases (n=33)	Controls (n=33)	p value
PFFA, Mean (SD)	0.24 (0.05)	0.20 (0.06)	0.018

In our study, the mean (SD) plasma free fatty acid (PFFA) among cases was significantly higher (0.24 mmol/L [0.05]) as compared to the control group (0.20 mmol/L [0.06]). The difference was statistically significant (p=0.018).

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Table 26: Intergroup comparison of mean EFT among gender

Variable		EFT, mean (SD)		p value
		Cases (n=33)	Controls (n=33)	
Sex	**Male**	11.30(3.39)	7.06(1.00)	0.000
	Female	12.77(2.62)	7.17(0.90)	

The mean (SD) EFT was lower among males than females in both groups [cases: 11.30 (3.39) vs 12.77 (2.62); controls: 7.06 (1.00) vs 7.17 (0.90)]. The difference was statistically significant (p=0.000).

Table 27: Relationship between statin and EFT among study groups

Variable		EFT, mean (SD)		p value
		Cases (n=33)	Controls (n=33)	
Statins	**Yes**	10.80(2.59)	-	0.000
	No	12.07(3.24)	7.12(0.93)	

This is the Intergroup comparison of effect of statins on mean EFT. Mean EFT was significantly higher among cases than controls. And among the cases those without statins had higher mean EFT. (p=0.000).

Table 28: Relationship between FBS and EFT between groups.

Variable		EFT, mean (SD)		p value
		Cases (n=33)	Controls (n=33)	
FBS	**Normal**	11.72(3.49)	7.12(0.93)	0.000
	Increased	12.00(3.00)	-	

Among patients the mean EFT was significantly higher among cases with increased FBS values. [11.72 (3.49) vs 7.12 (0.93); p=0.000].

Table 29: Relationship between HDL, LDL, triglycerides and EFT

Variable		EFT, mean (SD)		p value
		Cases (n=33)	**Controls (n=33)**	
HDL	**Decreased**	12.23(3.72)	-	0.000
	Normal	11.75(1.75)	6.96(1.01)	
	Increased	10.25(1.25)	7.34(0.81)	
LDL	**Normal**	10.99(3.14)	7.11(1.01)	0.000
	Increased	12.81(2.97)	7.12(0.90)	
Triglycerides	**Normal**	11.94(3.27)	7.11 (0.93)	0.000
	Borderline	12.00(2.00)	-	
	High	11.73(3.58)	-	

Overall, mean (SD) EFT was higher among cases than controls within HDL (p=0.000), LDL (p=0.000) and triglyceride (p=0.000) subgroups. Among cases, the mean (SD) EFT decreased with increase in HDL levels whereas, the mean (SD) EFT increased with increasing HDL levels. In both groups, mean (SD) EFT increased with increased LDL levels. Among triglyceride subgroup in cases, mean EFT was higher in borderline group (12.00 [2.00]) than in patients with normal (11.94[3.27]) and high (11.73[3.58]) Triglyceride levels.

Table 30: Intergroup comparison of mean PFFA among gender

Variable		PFFA, mean (SD)		p value
		Cases (n=33)	Controls (n=33)	
Sex	**Male**	0.22(0.06)	0.19(0.01)	0.015
	Female	0.25(0.03)	0.21(0.08)	

The mean (SD) PFFA was lower among males than females in both groups [cases: 0.22(0.06) vs 0.25(0.03); controls: 0.19(0.01) vs 0.21(0.08)]. The difference was statistically significant (p=0.015).

Table 31: Relationship between statin and PFFA among study groups

Variable		PFFA, mean (SD)		p value
		Cases (n=33)	Controls (n=33)	
Statins	**Yes**	0.24(0.04)	-	0.015
	No	0.24(0.05)	0.20(0.06)	

Mean PFFA was significantly higher among cases than controls without statins (p=0.015).

Table 32: Relationship between FBS and PFFA between groups

Variable		PFFA, mean (SD)		p value
		Cases (n=33)	Controls (n=33)	
FBS	**Normal**	0.23(0.05)	0.20(0.06)	0.015
	Increased	0.24(0.05)	-	

Among patients with normal FBS, the mean PFFA was significantly higher among cases as compared to controls [0.23(0.05) vs 0.20(0.06); p=0.015].

Table 33: Relationship between HDL, LDL, triglycerides and PFFA

Variable		PFFA, mean (SD)		p value
		Cases (n=33)	**Controls (n=33)**	
HDL	**Decreased**	0.24(0.57)	-	0.015
	Normal	0.22(0.02)	0.21(0.08)	
	Increased	0.23(0.37)	0.19(0.01)	
LDL	**Normal**	0.23 (0.03)	0.20(0.02)	0.015
	Increased	0.25(0.06)	0.21(0.08)	
Triglycerides	**Normal**	0.23(0.05)	0.20(0.06)	0.015
	Borderline	0.28(0.07)	-	
	High	0.23(0.03)	-	

Overall, mean (SD) PFFA was higher among cases than controls within HDL (p=0.015), LDL (p=0.015) and triglyceride (p=0.015) subgroups. Among cases, the mean (SD) PFFA increased with increase in HDL levels whereas, the mean (SD) PFFA decreased with increasing HDL levels. In both groups, mean (SD) PFFA increased with increased LDL levels. Among triglyceride subgroup in cases, mean PFFA was higher in borderline group (0.28[0.07]) than in patients with normal (0.23[0.05]) and high (0.23[0.03]) Triglyceride levels.

DISCUSSION

Stroke, also known as a cerebrovascular accident occurs secondary to rapid loss of brain function due to disturbance in the blood supply of the brain. It is one of the leading causes of morbidity and mortality worldwide [1]. The incidence of stroke is increasing over the years and therefore, it is essential to identify the predictive factors related to stroke for early identification and prevention of stroke. Previously established non-modifiable risk factors include advanced age, male sex, duration of diabetes and modifiable risk factors of stroke include higher glycaemic index, higher blood pressure, smoking and hyperlipidaemia [61]. Recently, EFT and plasma FFA have also been identified as the risk factors of ischemic stroke. As severity of stroke is inversely related to physical, psychological well-being as well as associated with increased healthcare burden, it is important to identify the risk factors and screen patients. In view of this, we conducted the present study to evaluate the relationship between EFT and plasma FFA in patients with ischemic stroke.

In our study a total of 66 subjects were included (cases, n=33 and controls, n=33). Age and sex are independent risk factors of ischemic stroke. It influences the stroke epidemiology, pathophysiology and treatment efficacy [62]. In our study, the Mean (SD) age of patients was significantly higher among cases as compared to controls (60.9 years vs 51.4 years). Mean age in our study population accordance with a large population-based study conducted by Muhammad IF, et al [63] where mean age of patients with ischemic stroke and those without ischemic stroke was 61.7 years and 57.2 years, respectively, whereas much lower than 71.4 vs 68.6 years reported by Altun I, et al [64]. In this study controls were healthy, young subjects.

Sex as a risk factor of stroke has also been vastly reviewed. It is believed that males are at higher risk of ischemic stroke during childhood and early adulthood and

women of middle age and elderly women have higher risk of ischemic stroke than men. This is related to the onset of menopause and loss of female sex hormones [65, 66]. In our study, frequency of males was higher among cases than controls (60.6% vs 45.5%) and frequency of females was higher in control groups (54.5% vs 45.5%).

Mendelian randomization analysis has reported the protective effect of education on the stroke incidence [68]. In another 2 sample mendelian randomization analysis, Xiuyun W, et al [69] showed that patients with an advanced education had 27% and those with intermediate education had 18% lesser hazards of ischemic stroke suggesting a negative causal relationship between education and ischemic stroke. In our study, 27.3% of ischemic stroke patients were uneducated, 12.1% had completed primary school, 15.2% had completed high school, 12.1% had completed preuniversity, 15.2% had completed graduation, and 18.2% were professionals.

Number of spoken languages help to assess the effect of stroke on cognitive function. It is suggested that bilingualism is associated with slower cognitive aging and delays the onset of dementia [70]. Alladi S, et al [71] reported that among stroke patients, frequency of normal cognition was significantly higher among bilinguals as compared to monolinguals (41% vs 20%). 54.5% of the patients spoke only one language and 18.2%, 15.2% and 12.1% patients spoke two, three and four languages, respectively.

Comorbidities including hypertension and diabetes are independent risk factors of ischemic stroke. Frequency of hypertension among ischemic patients varies widely among different studies. Duan XX, et al [67] reported hypertension among 57% of stroke patients while Muhammed IF, et al [63] reported 23.8% patients taking antihypertensives. In our study, among cases, 60.6% of patients had history of

hypertension with a mean duration of 6.8 years; 70% were on medication and medication compliance was seen in 78.6% patients. Similarly, frequency of diabetes among stroke patients ranges from 7.3% reported by Muhammed IF, et al [63] to 29% by Duan XX, et al [67]. In our study, history of diabetes was reported by 39.4% patients for a mean duration of 4.5 years. 77% were on medications and medication compliance was seen in 70% patients.

In our study out of 33 cases, 20 (60.6%) had anterior circulation stroke, 12 (36.3%) had a posterior circulation stroke, and 1(3.1%) presented with watershed area infarct. While 30 (90.9%) cases had unilateral infarcts on radiological imaging,3 (9.1%) presented with bilateral infarcts.

In our study frequency of smoking was slightly higher among controls than ischemic stroke patients (21% vs 18%) and is lower than reported by Muhammed IF, et al (30%) [63], Duan XX, et al (20.6%) [67], Altun I, et al (42.6%) [64]. We noted that, the frequency of tobacco consumption was significantly higher among cases than controls (36.4% vs 9.1%), and compared to other studies [63],[67] in India, particularly rural parts, the tobacco chewing overrides the smoking habit. Considering our study population comprised of rural population, tobacco chewing was higher than smoking habit.

Habitual moderate alcohol consumption is associated with reduced risk of mortality, coronary artery disease, congestive heart failure and stroke, whereas high alcohol intake is a risk factor of both ischemic and haemorrhagic stroke [72, 73]. In our study, 30.3% vs 39.4% patients in cases vs controls had history of alcohol consumption. However, we did not assess the number of drinks per day.

Nutrition is another important risk factor of stroke. Previous studies have suggested that a vegetarian diet consisting of whole grains, legumes, fruits and

vegetables reduces the risk of stroke by >40% [74]. Baden MY, et al [75] reported that patients who adhered to a healthful plant based diet had decreased risk of total stroke. In our study, no significant difference in the diet types were noted between cases and controls.

Obstructive sleep apnoea is considered an independent risk factor of stroke [76]. Additionally, abnormal sleep duration also poses a higher risk of incidence of stroke. In a study by Zhou L, et al, compared to patients with adequate sleep, those with longer duration of sleep had higher risk of stroke while those with inadequate or shorter sleep had no significant effect. Moreover, poor quality of sleep had 28% higher changes of ischemic stroke as compared to patients with good quality sleep [77]. In our study, there was significant difference in the sleep adequacy between cases and controls, while all subjects among controls had adequate sleep 18% patients with ischemic stroke had inadequate sleep.

Statins are commonly used treatment for the primary and secondary prevention of cardiovascular diseases which control the lipid levels in the body. It also has anti-inflammatory properties [78]. Aznaouridis K, et al [79] reported that statin treatment reduces the ischemic stroke events in patients with high cardiovascular risk and established atherosclerotic disease. In our study, none of the subjects in control group were on statins therapy and 15.2% patients with ischemic stroke were on statins.

Abrupt increase in the blood pressure is a characteristic finding of acute ischemic stroke. Acute BP elevations within the first hours to days after acute ischemic stroke influence the clinical decisions and also affect the risk of complications and future events [80]. This was correlated in our study, wherein we observed Stage 2 hypertension was more common among cases (60.6%) while most

subjects in control group had either normal (39.4%) or prehypertension (33.3%). The cerebral changes may further impact the cardiac changes [81]. However, in our study, the mean pulse rate between cases and control was comparable (86.7 vs 83.7 per minute). Further, brain inflammation secondary to ischemic stroke leads to potential lung damage and altered respiratory parameters [82]. However, no significant difference in the mean respiratory rate was noted between cases and controls.

Total cholesterol, triglycerides, HDL, LDL and glucose levels are modifiable risk factors of ischemic stroke. A meta-analysis conducted by Cui and Naikoo [83], reported a significant association between triglyceride and glucose levels with ischemic stroke, whereas the association between Total cholesterol, HDL, LDL with ischemic stroke was non-significant. According to Tziomalos K, et al [84], lower triglyceride and higher HDL levels are related to stroke severity and predict the outcomes of stroke. Kurth T, et al [85] reported a correlation between total cholesterol, LDL and ischemic stroke risk among healthy women. In our study, apart from LDL (94.2 vs 99.5 mg/dl; p=0.514), the mean FBS (173.5 vs 110.3 mg/dl; p=0.000), Total cholesterol (164.2 vs 141.1 mg/dl; p=0.022), and triglycerides (164.2 vs 141.1 mg/dl; p=0.022) were significantly higher in cases than those of controls. While lower HDL (41.6 vs 59.5 mg/dl; p=0.024) was noted in the cases group.

FFA independently predict the mortality and morbidity in patients with coronary artery disease. Wei XJ, et al [86], have reported that FFA is an independent short term prognostic marker of acute ischemic stroke. According to Pilitsis JG, et al [87] increased concentrations of polyunsaturated fatty acids within 48 hours of stroke onset is associated with worse outcome in acute ischemic stroke patients. Duan XX, et

al [67] estimated that serum FFA level of ≥0.71 mmol/L was an independent predictor of functional outcome and mortality in acute ischemic stroke. In our study, the mean PFFA was much higher among cases than controls (0.24 [0.05] vs 0.20 [0.06]; p=0.018) suggesting the link between PFFA and stroke. The results are in accordance with the reports by Duan et al [67] who indicated significantly higher median serum FFA levels in stroke patients as compared to normal controls [0.53 vs 0.24 mmol/L), respectively].

Epicardial fat is a metabolically active tissue is considered an important risk factor of metabolic and cardiovascular diseases including stroke. Previous studies have shown an association between increased EFT and occurrence of embolic stroke; with every 1mm increase in peri atrial EFT was associated with 7% increased hazard of post-ablation embolic stroke [88]. Additionally, a relationship between EFT and ischemic stroke is also reported by Akil E, et al [49] and Altun I, et al [64]. We found the mean EFT was significantly higher among ischemic stroke patients than controls (11.87 mm vs 7.11 mm; p=0.000). Our results are in accordance with reports by Altun I, et al [64] (4.8 ± 0.9 vs 3.8 ± 0.7) and Korkut M, et al [7] (6.33 ± 1.47 mm vs 3.74 ± 0.61 mm). Although our results are comparable, the mean EFT in our study group is much higher than previous reports [7, 64].

A correlation between EFT, insulin resistance and impaired arterial function has been shown previously [89]. According to Dogan M, et al, in hypertensive patients, increased EFT is significantly related to impaired aortic elastic properties [90]. Previous study by Parisi P et al [91] reported that statin therapy significantly reduces the EFT thickness and this has anti-inflammatory effect as a result of its direct action on cardiac visceral fat. In our study, among cases, only 15.2% patients were on statin therapy and this subset of patients had less EFT compared with patients without

statin therapy. Our reports are in accordance with these facts wherein we observed female gender, no statin therapy, higher FBS, LDL, triglycerides, and lower HDL to be significantly associated with increase in EFT and PFFA levels ($p<0.05$). However, intragroup comparisons within the cases of effect of clinical and laboratory parameters on EFT and PFFA showed no relationship between variables such as sex, diabetes, statin therapy, FBS, HDL, LDL and triglyceride levels on EFT and PFFA ($p>0.05$).

Results of our study further support that EFT and PFFA can be considered independent predictors of the ischemic stroke, further follow up studies are warranted to validate the results. Additionally, these markers can be used for early detection of the risk of stroke which may help in the early preventive treatment initiation to reduce EFT and PFFA levels in high-risk patients. Furthermore, the relation of these markers can be studied in the first degree relatives of the patient.

CONCLUSION

The conclusions drawn from our study

- There is significant increase in the mean EFT and mean PFFA among patients with ischemic stroke than healthy controls suggesting a strong association between EFT and PFFA among ischemic stroke patients.
- Higher age, male sex, lower level of education, monolinguals, concomitant hypertension and diabetes, tobacco consumption, inadequate sleep had higher risk of ischemic stroke.
- The female gender and laboratory parameters including increase in the levels of FBS, TC, LDL and triglycerides, and lower values of HDL showed statistically significant co-relation with increase in EFT and PFFA levels in acute ischemic stroke.

The results of our study suggest that EFT and PFFA levels can be considered an independent risk factor of ischemic stroke among adult patients. Further, large epidemiological, prospective studies are warranted to establish the relationship. We further suggest incorporation of EFT and PFFA levels in the screening programmes for ischemic stroke, for reducing the burden of stroke on the families, society and nation at large and hence curbing the morbidity and mortality that follows with it.

SUMMARY

The present case-control study was conducted at KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belagavi with an aim to evaluate the association of epicardial fat thickness and plasma free fatty acid in patients with acute ischemic stroke.

- A total of 33 adult patients admitted at KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belagavi between January 2021 to December 2021with clinical and radiologically proven diagnosis of acute ischemic stroke were included in the study.
- A total of 33 healthy adults with no history of any diseases visiting the hospital were included as controls.
- After obtaining an informed consent, a detailed information on demographics, patient history and clinical baseline information was collected from all cases and controls.

  Blood parameters including total cholesterol, low-density lipoprotein cholesterol (LDL), high-density lipoprotein cholesterol (HDL), triglycerides, fasting blood glucose levels were examined. Plasma FFA levels were measured by the enzymatic colorimetric test. Standard two-dimensional echocardiography was used to measure EFT.

- All data was entered in the Microsoft excel sheet and then imported to SPSS version 22 software for statistical analysis. Comparative analysis was performed using chi square test and ANOVA test. p-value less than or equal to 0.05 indicates statistical significance.

1. In our study a total of 66 subjects were included (cases, n=33 and controls, n=33). Mean (SD) age of patients was significantly higher among cases as compared to controls (60.9 [11.8] years vs 51.4 [12.7] years; p=0.005). Frequency of males was higher among cases than controls (60.6% vs 45.5%; p=0.324).
2. Among cases, 27.3% were uneducated, 12.1% had completed primary school, 15.2% had completed high school, 12.1% had completed preuniversity, 15.2% had completed graduation, and 18.2% were professionals.
3. 54.5% of the patients spoke only one language and 18.2%, 15.2% and 12.1% patients spoke two, three and four languages, respectively.
4. Among cases, 60.6% of patients had history of hypertension with a mean duration of 6.8 years. Among these, 70% were on medication and medication compliance was seen in 78.6% patients.
5. History of diabetes was reported by 39.4% patients for a mean duration of 4.5 years. 77% were on medications and medication compliance was seen in 70% patients.
6. Family history of diseases including diabetes, cardiovascular diseases, hypertension were seen in <10% of patients, in cases and controls.
7. 60.6% of the cases had anterior circulation stroke, 36.3% had a posterior circulation stroke, and 3.1% presented with watershed area infarct. 90.9% of them had unilateral infarcts on radiological imaging, while 9.1% presented with bilateral infarcts.
8. Among cases, only 15.2% patients were on statin therapy. Frequency of alcohol consumption (30.3% vs 39.4%; p=0.606), smoking (18.2% vs 21.2%; p=1.000) between cases and controls was comparable, while the frequency of tobacco

consumption was significantly higher among cases than controls (36.4% vs 9.1%; p=0.017).

9. Sleep adequacy was significantly higher among controls than cases (100% vs 81.8%; p=0.024). No significant difference in the diet types was noted between groups.
10. General examination revealed higher incidence of arcus senilis among cases than controls (51.5% vs 12.1%; p=0.001).
11. Stage 2 hypertension was noted among cases (60.6%).
12. Significantly higher levels of the mean (SD) of FBS (173.5 [78.3] vs 110.3 [15.9%] mg/dl; p=0.000), TC (164.2 [50.5] vs 141.1 [31.4] mg/dl; p=0.022), LDL (94.2[45.9] vs 99.5 [9.8] mg/dl; p=0.514) and triglycerides (164.2 [50.5] vs 141.1 [31.4] mg/dl; p=0.022 was seen among the cases as compared to the controls. While decrease in HDL (41.6 [32.7] vs 59.5 [8.6] mg/dl; p=0.024) was seen in the cases as compared to the controls.
13. The mean (SD) EFT (11.87 [3.14] vs 7.11 [0.93]; p=0.000) and PFFA (0.24 [0.05] vs 0.20 [0.06]; p=0.018) was much higher among cases than controls.
14. Intergroup comparisons between the cases and controls showed female gender, increased values of FBS, LDL, triglycerides and decreased values of HDL were significantly associated with increase in EFT and PFFA levels ($p<0.05$).
15. Intragroup comparisons in cases on effect of clinical and laboratory parameters on EFT and PFFA showed no relationship between variables such as sex, diabetes, FBS, HDL, LDL, and triglyceride levels ($p>0.05$).

STRENGTHS AND LIMITATIONS OF THE STUDY

Strengths of the study

- One of the few studies done in the Indian subcontinent to assess the epicardial fat thickness and plasma free fatty acid levels as independent risk factors for stroke.

Limitations of the study

- Small sample size.
- Short study duration.
- Case-control nature of the study and we did not follow up patients for re-measurement of EFT and PFFA levels after medication administration.

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ANNEXURE II: CONSENT

INFORMED CONSENT

Dear Mr./Mrs./Dr. _____, you are

kindly requested to enroll yourself in a research study tilted, **"THE EVALUATION OF EPICARDIAL FAT THICKNESS AND PLASMA FREE FATTY ACID LEVEL IN ACUTE ISCHEMIC STROKE- ONE YEAR CASE-CONTROL STUDY AT KLES Dr. PRABHAKAR KORE HOSPITAL AND MRC."** being conducted by REG NO: BG0120018 , a post graduate student in M.D. General Medicine and the study will be carried out under the direct supervision and guidance of Professor and Head of Unit, Department of General Medicine, Jawaharlal Nehru Medical College, Belagavi.

You have been requested to participate in this as you fit into the laid-out criteria for a study 'subject'/ participant.

Your participation in study is voluntary. During the study you will be undergoing blood investigations and a transthoracic echocardiography. Your decision whether or not to participate in the study will not affect your treatment in any form. If you decide not to participate you are free to withdraw at any time.

TITLE OF THE STUDY: **"THE EVALUATION OF EPICARDIAL FAT THICKNESS AND PLASMA FREE FATTY ACID LEVEL IN ACUTE ISCHEMIC STROKE- ONE YEAR CASE-CONTROL STUDY AT KLES Dr. PRABHAKAR KORE HOSPITAL AND MRC."**

PURPOSE OF THE STUDY: To study the association of epicardial fat thickness and plasma free fatty acid in patients with acute ischemic stroke.

PROCEDURES INVOLVED:

If you agree to enroll yourself in my study, you will be clinically examined in detail and investigated for the below said investigations accordingly.

- Plasma Free Fatty Acid (FFA)
- Random Blood Sugar
- Total Cholesterol
- High Density Lipoprotein (HDL)
- Low Density Lipoprotein (LDL)
- Very Density Lipoprotein (VLDL)
- Triglycerides
- Transthoracic Echocardiography- Epicardial fat thickness

RISKS AND BENEFITS:

There are no potential risks involved in this study. Benefits of taking part in this research:

To study the association of epicardial fat thickness and plasma free fatty acid in acute ischemic stroke.

VOLUNTARY PARTICIPATION / WITHDRAWAL FROM THE STUDY:

Taking part in the study is voluntary. You may choose not to enroll yourself in this study and may choose to leave the study anytime in between.

ALTERNATIVES:

Your decision regarding participation in study will not change present or future health care services offered to you at KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum. You would simply be excluded from the study if you wish to, and all your details shall be kept confidential and you will get the routine line of management.

PRIVACY AND CONFIDENTIALITY:

All data collected or disclosed by you during the course of

participation of study, will be kept fully confidential. If, however during the course it becomes necessary for the progress of the course to disclose the identity, it would be done so only after your informed & written consent.

The only people to know that you are a research subject are members of the research team. No information about you will be disclosed to other without your written permission except:

- In emergency to protect your rights AND welfare.
- If required by la

AUTHORIZATION TO PUBLISH RESULT:

The results of the study may be used to publish an article. When the results of research published or discussed, in a conference, no information will be displayed that would disclose your identity. Any information obtained in connection with this study and that can be identified with you will remain confidential.

FINANCIAL INCENTIVES FOR PARTICIPATION:

No additional costs shall be incurred upon you for the purpose of this study.

It is purely being done with the idea of research and all the cost of study will be borne by the investigator.

COMPENSATION:

In the event that you become injured as a result of taking part in this study, treatment will be offered to you at KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belagavi, or you will be given information about where to receive medical care.
However, no reimbursement, compensation or free medical care will be given.

QUESTIONS/CONTACT DETAILS:

You shall be free to contact the below mentioned name & addresses anytime during the study period for any clarification or help as you may desire for.

In case of the queries during study or in future you may contact following persons,

Dr. Harsha Hegde,
The Chairperson,
Ethical Committee for Human Subject Research
Jawaharlal Nehru Medical College,
KAHER, Belagavi – 590010

2.
Professor and Unit Head, Dept of
General Medicine, JNMC, Belagavi.

3 **REG NO: BG0120018**
Investigator,
Postgraduate in General Medicine, JNMC,
Belagavi.

CONSENT FORM

I voluntarily agree to take part in this study by signing below. I may withdraw at any time. I am not giving up any of my legal rights by signing this form. My signature below indicates that I have read this consent form, or it has been read to me, this consent form and have had all the questions answered

Signature / Left Thumb print of the Participant or legally authorized representative

Participant's name .....

Signature / Left thumb impression .....
of the participant

Name of the legally authorize .....
representative / guardian

Signature / Left thumb impression .....

Witness' name .....

Signature / Left thumb impression .....

Investigator's name and signature .....

Date:

Place:

ತಿಳುವಳಿಕೆಯ ಸಮ್ಮತಿ

ಆತ್ಮೀಯ ಶ್ರೀ / ಶ್ರೀ. / ಡಾ. _____, ಒರೆಯಾಗಿರುವ ಸಂಶೋಧನಾ ಅಧ್ಯಯನಕ್ಕೆ ನಿಮ್ಮನ್ನು ಸೇರಿಸಲು ನಿಮ್ಮನ್ನು ದಯೆಯಿಂದ ವಿನಂತಿಸಲಾಗಿದೆ, "ತೀವ್ರವಾದ ಇಸ್ಕೆಮಿಕ್ ಸ್ಟ್ರೋಕ್ನಲ್ಲಿ ಎಪಿಕಾರ್ಡಿಯಲ್ ಫ್ಯಾಟ್ ಮತ್ತು ದಪ್ಪ ಪ್ಲಾಸ್ಮಾ ಮುಕ್ತ ಕೊಬ್ಬಿನಾಮ್ಲ ಮಟ್ಟದ ಮೌಲ್ಯಮಾಪನ. " ಎಂ.ಡಿ. ಜನರಲ್ ಮೆಡಿಸಿನ್‌ನಲ್ಲಿ ಸ್ನಾತಕೋತ್ತರ ವಿದ್ಯಾರ್ಥಿ REG NO: BG0120018, ಅವರು ನಡೆಸಲಿದ್ದಾರೆ ಮತ್ತು ಬೆಳಗಾವಿನ ಜವಾಹರಲಾಲ್ ನೆಹರು ವೈದ್ಯಕೀಯ ಕಾಲೇಜಿನ ಜನರಲ್ ಮೆಡಿಸಿನ್ ವಿಭಾಗದ ಪ್ರಾಧ್ಯಾಪಕ ಡಾ. _____, ಪ್ರಾಧ್ಯಾಪಕ ಮತ್ತು ಘಟಕದ ಮುಖ್ಯಸ್ಥ ಅವರ ನೇರ ಮೇಲ್ವಿಚಾರಣೆ ಮತ್ತು ಮಾರ್ಗದರ್ಶನದಲ್ಲಿ ಈ ಅಧ್ಯಯನವನ್ನು ನಡೆಸಲಾಗುವುದು.

ಅಧ್ಯಯನದ 'ವಿಷಯ' / ಭಾಗವಹಿಸುವವರಿಗೆ ನೀವು ನಿಗದಿಪಡಿಸಿದ ಮಾನದಂಡಗಳಿಗೆ ಸರಿಹೊಂದುವಂತೆ ಇದರಲ್ಲಿ ಭಾಗವಹಿಸಲು ನಿಮ್ಮನ್ನು ವಿನಂತಿಸಲಾಗಿದೆ.

ಅಧ್ಯಯನದಲ್ಲಿ ನಿಮ್ಮ ಭಾಗವಹಿಸುವಿಕೆ ಸ್ವಯಂಪ್ರೇರಿತವಾಗಿದೆ. ಅಧ್ಯಯನದ ಸಮಯದಲ್ಲಿ ನಿಮ್ಮನ್ನು ಕೆಲವು ಪ್ರಶ್ನೆಗಳನ್ನು ಕೇಳಲಾಗುತ್ತದೆ ಮತ್ತು ನಿಮ್ಮ ಉತ್ತಮ ಜ್ಞಾನಕ್ಕೆ ನೀವು ಉತ್ತರಿಸಬೇಕಾಗುತ್ತದೆ. ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸಬೇಕೆ ಅಥವಾ ಬೇಡವೇ ಎಂಬ ನಿಮ್ಮ ನಿರ್ಧಾರವು ನಿಮ್ಮ ಚಿಕಿತ್ಸೆಯ ಮೇಲೆ ಯಾವುದೇ ರೂಪದಲ್ಲಿ ಪರಿಣಾಮ ಬೀರುವುದಿಲ್ಲ. ನೀವು ಭಾಗವಹಿಸಲು ನಿರ್ಧರಿಸಿದರೆ ನೀವು ಯಾವುದೇ ಸಮಯದಲ್ಲಿ ಹಿಂತೆಗೆದುಕೊಳ್ಳಬಹುದು.

ಅಧ್ಯಯನದ ಶೀರ್ಷಿಕೆ:

"ತೀವ್ರವಾದ ಇಸ್ಕೆಮಿಕ್ ಸ್ಟ್ರೋಕ್ನಲ್ಲಿ ಎಪಿಕಾರ್ಡಿಯಲ್ ಫ್ಯಾಟ್ ಮತ್ತು ದಪ್ಪ ಪ್ಲಾಸ್ಮಾ ಮುಕ್ತ ಕೊಬ್ಬಿನಾಮ್ಲ ಮಟ್ಟದ ಮೌಲ್ಯಮಾಪನ. "

ಅಧ್ಯಯನದ ಉದ್ದೇಶ:

ತೀವ್ರವಾದ ರಕ್ತಕೊರತೆಯ ಪಾರ್ಶ್ವವಾಯು ಹೊಂದಿರುವ ರೋಗಿಗಳಲ್ಲಿ ಎಪಿಕಾರ್ಡಿಯಲ್ ಕೊಬ್ಬಿನ ದಪ್ಪ ಮತ್ತು ಪ್ಲಾಸ್ಮಾ ಮುಕ್ತ ಕೊಬ್ಬಿನಾಮ್ಲಗಳ ಸಂಯೋಜನೆಯನ್ನು ಅಧ್ಯಯನ ಮಾಡಲು.

ಒಳಗೊಂಡಿರುವ ಕಾರ್ಯವಿಧಾನಗಳು:

ನನ್ನ ಅಧ್ಯಯನಕ್ಕೆ ನಿಮ್ಮನ್ನು ಸೇರಿಸಲು ನೀವು ಒಪ್ಪಿದರೆ, ನಿಮ್ಮ ಪ್ರಸ್ತುತ, ಹಿಂದಿನ ಮತ್ತು ಕುಟುಂಬದ ಇತಿಹಾಸದ ಬಗ್ಗೆ ನಿಮ್ಮನ್ನು ಸಂದರ್ಶಿಸಲಾಗುವುದು, ನಂತರ ನಿಮ್ಮನ್ನು ಪ್ರಾಯೋಗಿಕವಾಗಿ ವಿವರವಾಗಿ ಪರಿಶೀಲಿಸಲಾಗುತ್ತದೆ ಮತ್ತು ಅದಕ್ಕೆ ಅನುಗುಣವಾಗಿ ತನಿಖೆ ಮಾಡಲಾಗುತ್ತದೆ.

ಪ್ಲಾಸ್ಮಾ ಫ್ರೀ ಫ್ಯಾಟಿ ಆಸಿಡ್ (ಎಫ್ಎಫ್ಎ)
ಯಾದೃಚ್ಛಿಕ ರಕ್ತ ಸಕ್ಕರೆ
ಒಟ್ಟು ಕೊಲೆಸ್ಟ್ರಾಲ್
ಹೈ ಡೆನ್ಸಿಟಿ ಲಿಪೊಪ್ರೋಟೀನ್ (ಎಚ್ಡಿಎಲ್)
ಕಡಿಮೆ ಸಾಂದ್ರತೆಯ ಲಿಪೊಪ್ರೋಟೀನ್ (ಎಲ್ಡಿಎಲ್)
ವೆರಿ ಡೆನ್ಸಿಟಿ ಲಿಪೊಪ್ರೋಟೀನ್ (ವಿಎಲ್ಡಿಎಲ್)
ಟ್ರೈಗ್ಲಿಸರೈಡ್ಗಳು
ಟ್ರಾನ್ಸ್ಥೊರಾಸಿಕ್ ಎಕೋಕಾರ್ಡಿಯೋಗ್ರಫಿ- ಎಪಿಕಾರ್ಡಿಯಲ್ ಕೊಬ್ಬಿನ ದಪ್ಪ

ಅಪಾಯ ಮತ್ತು ಪ್ರಯೋಜನಗಳು:

ಈ ಅಧ್ಯಯನದಲ್ಲಿ ಯಾವುದೇ ಸಂಭಾವ್ಯ ಅಪಾಯಗಳಿಲ್ಲ.

ಈ ಸಂಶೋಧನೆಯಲ್ಲಿ ಪಾಲ್ಗೊಳ್ಳುವ ಪ್ರಯೋಜನಗಳು:

ಎಪಿಕಾರ್ಡಿಯಲ್ ಕೊಬ್ಬಿನ ದಪ್ಪ ಮತ್ತು ಪ್ಲಾಸ್ಮಾ ಮುಕ್ತ ಕೊಬ್ಬಿನಾಮ್ಲ ಇನಾಕ್ಯುಟ್ ಇಸ್ಕೆಮಿಕ್ ಸ್ಕೋರ್ನ ಸಂಯೋಜನೆಯನ್ನು ಅಧ್ಯಯನ ಮಾಡಲು.

ಸ್ವಯಂಪ್ರೇರಿತ ಭಾಗವಹಿಸುವಿಕೆ / ಅಧ್ಯಯನದಿಂದ

ಹಿಂತೆಗೆದುಕೊಳ್ಳುವಿಕೆ:

ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸುವುದು ಸ್ವಯಂಪ್ರೇರಿತವಾಗಿದೆ. ಈ ಅಧ್ಯಯನಕ್ಕೆ ನಿಮ್ಮನ್ನು ಸೇರಿಸಿಕೊಳ್ಳದಿರಲು ನೀವು ಆಯ್ಕೆ ಮಾಡಬಹುದು ಮತ್ತು ಈ ನಡುವೆ ಯಾವುದೇ ಸಮಯದಲ್ಲಿ ಅಧ್ಯಯನವನ್ನು ಬಿಡಲು ಆಯ್ಕೆ ಮಾಡಬಹುದು.

ಪರ್ಯಾಯಗಳು:

ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸುವ ಬಗ್ಗೆ ನಿಮ್ಮ ನಿರ್ಧಾರವು ಕೆಎಲ್ಇಎಸ್ ಡಾ. ಪ್ರಭಾಕರ್ ಕೋರೆ ಆಸ್ಪತ್ರೆ ಮತ್ತು ಬೆಳಗಾವಿ ವೈದ್ಯಕೀಯ ಸಂಶೋಧನಾ ಕೇಂದ್ರದಲ್ಲಿ ನಿಮಗೆ ನೀಡುತ್ತಿರುವ ಪ್ರಸ್ತುತ ಅಥವಾ ಭವಿಷ್ಯದ ಆರೋಗ್ಯ ಸೇವೆಗಳನ್ನು ಬದಲಾಯಿಸುವುದಿಲ್ಲ. ನೀವು ಬಯಸಿದರೆ ನಿಮ್ಮನ್ನು ಅಧ್ಯಯನದಿಂದ ಹೊರಗಿಡಲಾಗುವುದು, ಮತ್ತು ನಿಮ್ಮ ಎಲ್ಲಾ ವಿವರಗಳನ್ನು ಗೌಪ್ಯವಾಗಿಡಲಾಗುತ್ತದೆ ಮತ್ತು ನೀವು ವಾಡಿಕೆಯ ನಿರ್ವಹಣೆಯನ್ನು ಪಡೆಯುತ್ತೀರಿ.

ಗೌಪ್ಯತೆ ಮತ್ತು ಗೌಪ್ಯತೆ:

ಅಧ್ಯಯನದ ಭಾಗವಹಿಸುವಿಕೆಯ ಸಮಯದಲ್ಲಿ ನೀವು ಸಂಗ್ರಹಿಸಿದ ಅಥವಾ ಬಹಿರಂಗಪಡಿಸಿದ ಎಲ್ಲಾ ಡೇಟಾವನ್ನು ಸಂಪೂರ್ಣವಾಗಿ ಗೌಪ್ಯವಾಗಿಡಲಾಗುತ್ತದೆ. ಕೋರ್ಸ್ ಸಮಯದಲ್ಲಿ ಪ್ರಗತಿಗೆ ಗುರುತನ್ನು ಬಹಿರಂಗಪಡಿಸುವುದು ಅಗತ್ಯವಿದ್ದರೆ, ನಿಮ್ಮ ಮಾಹಿತಿ ಮತ್ತು ಲಿಖಿತ ಒಪ್ಪಿಗೆಯ ನಂತರವೇ ಇದನ್ನು ಮಾಡಲಾಗುತ್ತದೆ.

ನೀವು ಸಂಶೋಧನಾ ವಿಷಯ ಎಂದು ತಿಳಿದುಕೊಳ್ಳುವ ಏಕೈಕ ಜನರು ಸಂಶೋಧನಾ ತಂಡದ ಸದಸ್ಯರು. ನಿಮ್ಮ ಲಿಖಿತ ಅನುಮತಿಯಿಲ್ಲದೆ ನಿಮ್ಮ ಬಗ್ಗೆ ಯಾವುದೇ ಮಾಹಿತಿಯನ್ನು ಇತರರಿಗೆ ಬಹಿರಂಗಪಡಿಸಲಾಗುವುದಿಲ್ಲ:

- ನಿಮ್ಮ ಹಕ್ಕುಗಳು ಮತ್ತು ಕಲ್ಯಾಣವನ್ನು ರಕ್ಷಿಸಲು ತುರ್ತು ಪರಿಸ್ಥಿತಿಯಲ್ಲಿ.
- ಕಾನೂನಿನ ಪ್ರಕಾರ ಅಗತ್ಯವಿದ್ದರೆ.

ಫಲಿತಾಂಶಗಳನ್ನು ಪ್ರಕಟಿಸಲು ಅಧಿಕಾರ:

ಅಧ್ಯಯನದ ಫಲಿತಾಂಶಗಳನ್ನು ಲೇಖನವನ್ನು ಪ್ರಕಟಿಸಲು ಬಳಸಬಹುದು. ಸಂಶೋಧನೆಯ ಫಲಿತಾಂಶಗಳು ಪ್ರಕಟವಾದ ಅಥವಾ ಚರ್ಚಿಸಿದಾಗ, ಸಮ್ಮೇಳನದಲ್ಲಿ, ನಿಮ್ಮ ಗುರುತನ್ನು ಬಹಿರಂಗಪಡಿಸುವ ಯಾವುದೇ ಮಾಹಿತಿಯನ್ನು ಪ್ರದರ್ಶಿಸಲಾಗುವುದಿಲ್ಲ. ಈ ಅಧ್ಯಯನಕ್ಕೆ ಸಂಬಂಧಿಸಿದಂತೆ ಪಡೆದ ಯಾವುದೇ ಮಾಹಿತಿಯು ಮತ್ತು ಅದನ್ನು ನಿಮ್ಮೊಂದಿಗೆ ಗುರುತಿಸಬಹುದು.

ಭಾಗವಹಿಸುವಿಕೆಗೆ ಆರ್ಥಿಕ ಪ್ರೋತ್ಸಾಹ:

ಈ ಅಧ್ಯಯನದ ಉದ್ದೇಶಕ್ಕಾಗಿ ಯಾವುದೇ ಹೆಚ್ಚುವರಿ ವೆಚ್ಚಗಳು ನಿಮ್ಮ ಮೇಲೆ ಆಗುವುದಿಲ್ಲ. ಇದನ್ನು ಸಂಪೂರ್ಣವಾಗಿ ಸಂಶೋಧನೆಯ ಆಲೋಚನೆಯೊಂದಿಗೆ ಮಾಡಲಾಗುತ್ತಿದೆ ಮತ್ತು ಅಧ್ಯಯನದ ಎಲ್ಲಾ ವೆಚ್ಚವನ್ನು ತನಿಖಾಧಿಕಾರಿ ಭರಿಸುತ್ತಾರೆ.

ಪರಿಹಾರ:

ಈ ಅಧ್ಯಯನದಲ್ಲಿ ಪಾಲ್ಗೊಂಡ ಪರಿಣಾಮವಾಗಿ ನೀವು ಗಾಯಗೊಂಡರೆ ಬೆಳಗಾವಿನ ಕೆಎಲ್ಇಎಸ್ ಡಾ. ಪ್ರಭಾಕರ್ ಕೋರೆ ಆಸ್ಪತ್ರೆ ಮತ್ತು ವೈದ್ಯಕೀಯ ಸಂಶೋಧನಾ ಕೇಂದ್ರದಲ್ಲಿ ನಿಮಗೆ ಚಿಕಿತ್ಸೆ ನೀಡಲಾಗುವುದು ಅಥವಾ ವೈದ್ಯಕೀಯ ಆರೈಕೆಯನ್ನು ಎಲ್ಲಿ ಪಡೆಯಬೇಕು ಎಂಬ ಬಗ್ಗೆ ನಿಮಗೆ ಮಾಹಿತಿ ನೀಡಲಾಗುವುದು. ಆದಾಗ್ಯೂ, ಯಾವುದೇ ಮರುಪಾವತಿ, ಪರಿಹಾರ ಅಥವಾ ಉಚಿತ ವೈದ್ಯಕೀಯ ಸೌಲಭ್ಯವನ್ನು ನೀಡಲಾಗುವುದಿಲ್ಲ.

ಒಪ್ಪಿಗೆ ಪತ್ರ

ಕೆಳಗೆ ಸಹಿ ಮಾಡುವ ಮೂಲಕ ಈ ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸಲು ನಾನು ಸ್ವಯಂಪ್ರೇರಣೆಯಿಂದ ಒಪ್ಪುತ್ತೇನೆ. ನಾನು ಯಾವುದೇ ಸಮಯದಲ್ಲಿ ಹಿಂತೆಗೆದುಕೊಳ್ಳಬಹುದು. ಈ ಫಾರ್ಮ್ಗೆ ಸಹಿ ಮಾಡುವ ಮೂಲಕ ನಾನು ನನ್ನ ಯಾವುದೇ ಕಾನೂನು ಹಕ್ಕುಗಳನ್ನು ಬಿಟ್ಟುಕೊಡುತ್ತಿಲ್ಲ. ಕೆಳಗಿನ ನನ್ನ ಸಹಿ ನಾನು ಈ ಒಪ್ಪಿಗೆಯ ಫಾರ್ಮ್ ಅನ್ನು ಓದಿದ್ದೇನೆ ಅಥವಾ ಈ ಸಮ್ಮತಿಯ ಫಾರ್ಮ್ ಅನ್ನು ನನಗೆ ಓದಿದ್ದೇನೆ ಮತ್ತು ಎಲ್ಲಾ ಪ್ರಶ್ನೆಗಳಿಗೆ ಉತ್ತರಿಸಿದೆ ಎಂದು ಸೂಚಿಸುತ್ತದೆ

ಭಾಗವಹಿಸುವವರ ಅಥವಾ ಕಾನೂನುಬದ್ಧವಾಗಿ ಅಧಿಕೃತ ಪ್ರತಿನಿಧಿಯ ಸಹಿ / ಎಡ ಹೆಬ್ಬೆರಳು ಮುದ್ರಣ
ಭಾಗವಹಿಸುವವರ ಹೆಸರು: ....

ಸಹಿ / ಎಡ ಹೆಬ್ಬೆರಳು ಅನಿಸಿಕೆ: ....

ಭಾಗವಹಿಸುವವರ ಕಾನೂನುಬದ್ಧವಾಗಿ ಅಧಿಕಾರ ಪಡೆದವರ ಹೆಸರು: ....

ಪ್ರತಿನಿಧಿ / ರಕ್ಷಕ ಸಹಿ / ಎಡ ಹೆಬ್ಬೆರಳು ಅನಿಸಿಕೆ: ....

ಸಾಕ್ಷಿ ಹೆಸರು: ....

ಸಹಿ / ಎಡ ಹೆಬ್ಬೆರಳು ಅನಿಸಿಕೆ: ....

ತನಿಖಾಧಿಕಾರಿ ಹೆಸರು ಮತ್ತು ಸಹಿ: ....

ದಿನಾಂಕ:

ಸ್ಥಳ:

माहितीपूर्ण संमती

प्रिय श्रीमती / श्रीमती / डॉ. _____, आपणास विनम्र विनंती आहे की आपणास झुकलेल्या एका संशोधन अभ्यासामध्ये नाव नोंदवावे "तीव्र इस्केमिक स्ट्रोकमध्ये एपिकार्डियल फॅ□ची जाडी आणि प्लाझ्मा फ्री फॅ□ी ॲसिड पातळीचे मूल्यांकन."

REG NO: BG0120018, एम.डी. जनरल मेडिसीन मधील पदव्युत्तर विद्यार्थी घेत आहेत आणि हा अभ्यास, जवाहरलाल नेहरू मेडिकल कॉलेज, बेळगाव येथील डॉ_____, प्राध्यापक आणि युनि□ हेड, सामान्य चिकित्सा विभाग, जवाहरलाल नेहरू मेडिकल कॉलेज, बेळगाव यांच्या थे□ देखरेखीखाली आणि मार्गदर्शनानुसार केला जाईल.

आपण अभ्यासाच्या 'विषय' / सहभागीच्या निकषांनुसार बसत असल्यामुळे यामध्ये सहभागी होण्याची विनंती केली गेली आहे.

अभ्यासात आपला सहभाग ऐच्छिक आहे. अभ्यासादरम्यान आपल्याला काही प्रश्न विचारले जातील आणि आपल्या सर्वोत्तम उत्तरासाठी आपल्याला उत्तर द्यावे लागेल. अभ्यासामध्ये भाग घ्यायचा की नाही या निर्णयाचा तुमच्या उपचारांवर कोणत्याही प्रकारचा परिणाम होणार नाही. आपण सहभागी होण्याचे ठरविल्यास आपण कधीही माघार घेण्यास मोकळे आहात.

बेळगावी.

अभ्यासाचे शीर्षकः

"तीव्र इस्केमिक स्ट्रोकमध्ये एपिकार्डियल फॅ□ची जाडी आणि प्लाझ्मा फ्री फॅ□ी ॲसिड पातळीचे मूल्यांकन."

अभ्यासाचा हेतू:

तीव्र इस्केमिक स्ट्रोक असलेल्या रूग्णांमध्ये एपिकार्डियल फॅ□ची जाडी आणि प्लाझ्मा फ्री फॅ□ी असिडच्या संगतीचा अभ्यास करणे.

प्रक्रिया समाविष्ट:

माझ्या अभ्यासामध्ये आपण स्वतःस नावनोंदणी घेण्यास सहमत असल्यास, आपल्यास आपल्या वर्तमान, भूतकाळाच्या आणि कौ□ुंबिक इतिहासाच्या संदर्भात मुलाखत दिली जाईल, त्यानंतर आपणास वैद्यकीयदृष्ट्या तपशीलवार तपासणी केली जाईल आणि त्यानुसार चौकशी केली जाईल.

प्लाझ्मा फ्री फॅ□ी असिड (एफएफए)
रँडम रक्तातील साखर
एकूण कोलेस्ट्रॉल
हाय डेन्सि□ी लिपोप्रो□ीन (एचडीएल)
लो डेन्सि□ी लिपोप्रो□ीन (एल डी एल)
वेअर डेन्सि□ी लिपोप्रो□ीन (व्हीएलडीएल)
ट्रायग्लिसेराइड्स
ट्रान्सस्□ोरॅसिक इकोकार्डियोग्राफी- एपिकार्डियल फॅ□ची जाडी

जोखीम आणि फायदे:

या अभ्यासामध्ये कोणतेही संभाव्य धोके गुंतलेले नाहीत.

या संशोधनात भाग घेण्याचे फायदेः

एपिकार्डियल फॅ□ची जाडी आणि प्लाझ्मा फ्री फॅ□ी असिड निष्क्रिय इस्केमिक स्ट्रोकच्या सहवासाचा अभ्यास करणे.

ऐच्छिक सहभाग / अभ्यासामधून पैसे काढणे:

अभ्यासामध्ये भाग घेणे ऐच्छिक आहे. आपण या अभ्यासामध्ये स्वत: ची नावनोंदणी न करणे निवडू शकता आणि दरम्यान अभ्यास कधीही सोडणे निवडू शकता.

विकल्पः

अभ्यासात सहभागासंदर्भातील तुमचा निर्णय केएलईएस डॉ. प्रभाकर कोरे हॉस्पि□ल आणि वैद्यकीय संशोधन केंद्र, बेळगाव येथे तुम्हाला देऊ केलेल्या सध्याच्या किंवा भविष्यातील आरोग्य सेवा बदलणार नाही. आपली इच्छा असेल तर आपल्याला अभ्यासापासून वगळले जाईल आणि आपले सर्व तपशील गोपनीय ठेवले जातील आणि आपल्याला व्यवस्थापनाची नियमित रूंदी मिळेल.

गोपनीयता आणि गोपनीयता:

अभ्यासाच्या सहभागादरम्यान आपण गोळा केलेला किंवा जाहीर केलेला सर्व डे□ा पूर्णपणे गोपनीय ठेवला जाईल. अर्थात कोर्सच्या दरम्यान ओळख जाहीर करणे आवश्यक झाले तर ते तुमच्या माहिती व लेखी संमतीनंतरच केले जाईल.

आपण संशोधन विषय आहात हे फक्त लोकांनाच माहित आहे की ते संशोधन पथकाचे सदस्य आहेत. आपल्या लेखी परवानगीशिवाय इतर आपल्याबद्दल कोणतीही माहिती उघड केली जाणार नाहीः

- आपत्कालीन परिस्थितीत आपले हक्क आणि कल्याण यांचे संरक्षण करण्यासाठी.
- कायद्याने आवश्यक असल्यास.

निकाल प्रकाशित करण्यासाठी अधिकृतताः

अभ्यासाचा निकाल लेख प्रकाशित करण्यासाठी वापरला जाऊ शकतो. जेव्हा एखाद्या संशोधनाचे निकाल कॉन्फरन्समध्ये प्रकाशित केले जातात किंवा त्यावर चर्चा केली जाते तेव्हा आपली ओळख उघडकीस आणणारी कोणतीही माहिती दर्शविली जाणार नाही. या अभ्यासाच्या संदर्भात प्राप्त केलेली कोणतीही माहिती आणि ती आपल्याशी ओळखली जाऊ शकते ती गोपनीय राहील.

सहभागासाठी आर्थिक प्रोत्साहन:

या अभ्यासाच्या हेतूने आपल्यावर कोणत्याही प्रकारची अतिरिक्त किंमत आकारली जाणार नाही.
हे निव्वळ संशोधनाच्या कल्पनेने केले जात आहे आणि अभ्यासाचा सर्व खर्च तपासनीस करेल.

भरपाई:

या अभ्यासामध्ये भाग घेतल्यामुळे आपण जखमी झाल्यास, केएलईएस डॉ. प्रभाकर कोरे हॉस्पि□ल आणि मेडिकल रिसर्च सें□र, बेळगाव येथे तुम्हाला उपचार देण्यात येतील किंवा तुम्हाला वैद्यकीय सेवा कोठून घ्यावी याविषयी माहिती दिली जाईल. तथापि, कोणतेही प्रतिपूर्ती, भरपाई किंवा विनामूल्य वैद्यकीय सेवा दिली जाणार नाही.

संमती फॉर्म

मी खाली स्वाक्षरी करून या अभ्यासात भाग घेण्यास स्वेच्छेने सहमत आहे. मी कधीही माघार घेऊ शकतो. या फॉर्मवर सही करून मी माझा कोणताही कायदेशीर हक्क सोडत नाही. खाली माझी स्वाक्षरी सूचित करते की मी हा संमती फॉर्म वाचला आहे किंवा हा संमती फॉर्म मला वाचला आहे आणि मला सर्व प्रश्नांची उत्तरे दिली आहेत

सहभागी किंवा कायदेशीररित्या अधिकृत प्रतिनिधीची सही / डावा अंगठा प्रिं

सहभागीचे नाव: ....

स्वाक्षरी / डावा अंगठा ठसा: ....

सहभागीचा

कायदेशीररीत्या अधिकृत नाव: ....

प्रतिनिधी / पालक

स्वाक्षरी / डावा अंगठा ठसा: ....

साक्षीचे नाव: ....

स्वाक्षरी / डावा अंगठा ठसा: ....

अन्वेषकांचे नाव आणि स्वाक्षरीः ....

तारीख:

ठिकाणः

सूचित सहमति

प्रिय श्री / श्रीमती / डॉ। _____, आपसे विनम्र अनुरोध है कि आप स्वयं को एक शोध अध्ययन में नामांकित करें, "एपिकार्डियल फै□ की मो□ाई का ल्यांकनऔर एक्यू□ इस्केमिक स्ट्रोक में प्लाज्मा फ्री फै□ी एसिड स्तर." एमडी जनरल मेडिसिन में स्नातकोत्तर छात्र REG NO: BG0120018 द्वारा संचालित किया जा रहा है और यह अध्ययन डॉ_____, प्रोफेसर और यूनि□ के प्रमुख, जनरल मेडिसिन विभाग, जवाहरलाल नेहरू मेडिकल कॉलेज, बेलगाम के प्रत्यक्ष पर्यवेक्षण और मार्गदर्शन में किया जाएगा।

आपसे यह अनुरोध किया गया है कि आप इसमें एक अध्ययन 'विषय' / प्रतिभागी के निर्धारित मानदंडों में फि□ हों। अध्ययन में आपकी भागीदारी स्वैच्छिक है। अध्ययन के दौरान आपसे कुछ प्रश्न पूछे जाएंगे और आप अपने ज्ञान का सबसे अच्छा जवाब देने वाले हैं। अध्ययन में भाग लेने या न लेने का आपका निर्णय किसी भी रूप में आपके उपचार को प्रभावित नहीं करेगा। यदि आप भाग लेने का निर्णय लेते हैं तो आप किसी भी समय वापस लेने के लिए स्वतंत्र हैं।

अध्ययन का शीर्षक :

"एपिकार्डियल फै□ की मो□ाई का ल्यांकनऔर एक्यू□ इस्केमिक स्ट्रोक में प्लाज्मा फ्री फै□ी एसिड स्तर."

अध्ययन का उद्देश्य:

तीव्र इस्केमिक स्ट्रोक वाले रोगियों में एपिकार्डियल वसा की मो□ाई और प्लाज्मा मुक्त फै□ी एसिड के सहयोग का अध्ययन करना।

शामिल प्रक्रियाएं :

यदि आप मेरे अध्ययन में खुद को नामांकित करने के लिए सहमत हैं, तो आपको अपने वर्तमान, अतीत और परिवार के इतिहास के बारे में साक्षात्कार दिया जाएगा, फिर आपकी चिकित्सकीय जांच की जाएगी और तदनुसार जांच की जाएगी।

प्लाज्मा फ्री फै□ी एसिड (एफएफए)
यादृच्छिक रक्त शर्करा
कुल कोलेस्ट्रॉल
उच्च घनत्व वाले लिपोप्रो□ीन (एचडीएल)
कम घनत्व वाले लिपोप्रो□ीन (एलडीएल)
बहुत घनत्व वाले लिपोप्रो□ीन (वीएलडीएल)
ट्राइग्लिसराइड्स
ट्रान्सथोरासिक इकोकार्डियोग्राफी- एपिकार्डियल वसा की मो□ाई

जोखिम और लाभ:

इस अध्ययन में कोई संभावित जोखिम शामिल नहीं हैं।

इस शोध में भाग लेने के लाभ:

एपिकार्डियल फै□ की मो□ाई और प्लाज़्मा फ्री फै□ी एसिड इनक्यू□ इस्केमिक स्ट्रोक के संबंध का अध्ययन करने के लिए।

अध्ययन से स्वैच्छिक भागीदारी / निकासी :

अध्ययन में भाग लेना स्वैच्छिक है। आप इस अध्ययन में खुद को नामांकित नहीं करना चुन सकते हैं और बीच में कभी भी अध्ययन छोड़ने का विकल्प चुन सकते हैं।

विकल्पः

अध्ययन में भाग लेने के बारे में आपका निर्णय के एल ई एस डॉ। प्रभाकर कोरे अस्पताल और चिकित्सा अनुसंधान केंद्र, बेलगाम में आपके लिए पेश की गई वर्तमान या भविष्य की स्वास्थ्य देखभाल सेवाओं को नहीं बदलेगा। यदि आप चाहें, तो आपको अध्ययन से बाहर रखा जाएगा और आपके सभी विवरणों को गोपनीय रखा जाएगा और आपको प्रबंधन की नियमित लाइन मिल जाएगी।

गोपनीयता और गोपनीयता :

अध्ययन की भागीदारी के दौरान आपके द्वारा एकत्र या प्रक□ किए गए सभी डे□ा को पूरी तरह से गोपनीय रखा जाएगा। हालांकि यदि पाठ्यक्रम के दौरान यह आवश्यक है कि पाठ्यक्रम की प्रगति के लिए पहचान का खुलासा करना आवश्यक है, तो यह आपकी सूचना और लिखित सहमति के बाद ही किया जाएगा।
केवल यह जानने के लिए कि आप एक शोध विषय हैं, अनुसंधान □ीम के सदस्य हैं। आपके लिखित अनुमति के बिना आपके बारे में कोई भी जानकारी का खुलासा नहीं किया जाएगा :
- अपने अधिकारों और कल्याण की रक्षा के लिए आपातकाल में।
- यदि कानून द्वारा आवश्यक हो।

परिणाम प्रकाशित करने के लिए प्राधिकरण :

अध्ययन के परिणामों का उपयोग एक लेख प्रकाशित करने के लिए किया जा सकता है। जब एक सम्मेलन में प्रकाशित या चर्चा की गई शोध के परिणाम, कोई भी जानकारी प्रदर्शित नहीं की जाएगी जो आपकी पहचान का खुलासा करेगी। इस अध्ययन के संबंध में प्राप्त की गई कोई भी जानकारी और जिसे आप के साथ पहचाना जा सकता है, गोपनीय रहेगी।

भागीदारी के लिए वित्तीय प्रोत्साहन :

इस अध्ययन के उद्देश्य से आपके ऊपर कोई अतिरिक्त लागत नहीं लगेगी।
यह विशुद्ध रूप से अनुसंधान के विचार के साथ किया जा रहा है और अध्ययन का सारा खर्च अन्वेषक द्वारा वहन किया जाएगा।

भरपाई : इस अध्ययन में भाग लेने के परिणामस्वरूप आप घायल हो जाते हैं, तो केएलईएस डॉ। प्रभाकर कोरे अस्पताल और चिकित्सा अनुसंधान केंद्र, बेलगाम में उपचार की पेशकश की जाएगी, या आपको चिकित्सा देखभाल कहाँ प्राप्त होगी, इसके बारे में जानकारी दी जाएगी। हालांकि, कोई प्रतिपूर्ति, मुआवजा या मुफ्त चिकित्सा देखभाल नहीं दी जाएगी।

सहमति पत्र

मैं स्वेच्छा से नीचे हस्ताक्षर करके इस अध्ययन में भाग लेने के लिए सहमत हूं। मैं किसी भी समय वापस ले सकता हूं। मैं इस फॉर्म पर हस्ताक्षर करके अपने किसी भी कानूनी अधिकार को नहीं छोड़ रहा हूं। नीचे दिए गए मेरे हस्ताक्षर से संकेत मिलता है कि मैंने इस सहमति फॉर्म को पढ़ा है, या यह मेरे लिए पढ़ा गया है, यह सहमति फॉर्म और उत्तर दिए गए प्रश्नों के उत्तर हैं

तिभागी या कानूनी रूप से अधिकृत प्रतिनिधि का हस्ताक्षर / बायाँ अंगूठा प्रिं

प्रतिभागी का नाम: ....।

हस्ताक्षर / बाएं अंगूठे का निशान: ....।

प्रतिभागी का

कानूनी रूप से अधिकृत का नाम: ....।

प्रतिनिधि / अभिभावक

हस्ताक्षर / बाएं अंगूठे का निशान: ....।

साक्षी का नाम: ....।

हस्ताक्षर / बाएं अंगूठे का निशान: ....।

अन्वेषक का नाम और हस्ताक्षर: ....।

दिनांक:

जगह:

CASE PROFORMA

CASE NO:

NAME:

AGE:

SEX:

IP NO:

ADDRESS:

PRESENTING COMPLAINTS:

PAST HISTORY:

Hypertension-

1. Duration-
2. Medications-
3. Compliance-

Diabetes Mellitus-

1. Duration-
2. Medications-
3. Compliance-

PERSONAL HISTORY:

- Alcohol use-
- Smoking-
- Tobacco chewer-
- Sleep-
- Bowel habits-
- Bladder habits-

DIETARY HISTORY:

- Vegetarian Diet-
- Non-Vegetarian Diet-

FAMILY HISTORY-

- Stroke-
- Cardiovascular Disease-
- Sudden Cardiac Death-
- Hypertension-
- Diabetes mellitus-

DRUG HISTORY:

PATIENT NO	ON STATIN	NOT ON STATIN

OTHER DRUGS:

PHYSICAL EXAMINATION:

- Conscious-
- Co-operative-
- Oriented-

VITALS:

- TEMPERATURE-
- PULSE RATE-
- RESPIRATORY RATE-
- BLOOD PRESSURE-

GENERAL CONDITION:

- PALLOR-
- ICTERUS-
- CYNOSIS-
- CLUBBING-
- LYMPHADENOPATHY-
- EDEMA-
- ARCUS SENALIS-
- XANTHELASMA-

SYSTEMIC EXAMINATION:

RESPIRATORY SYSTEM-

CARDIOVASCULAR SYSTEM-

CENTRAL NERVOUS SYSTEM-

PER ABDOMEN-

INVESTIGATIONS

- Plasma Free Fatty Acid (PFFA)
- Fasting Blood Sugar
- Total Cholesterol
- High Density Lipoprotein (HDL)
- Low Density Lipoprotein (LDL)
- Very Density Lipoprotein (VLDL)
- Triglycerides
- Transthoracic Echocardiography- Epicardial fat thickness

CONTROL PROFORMA

CONTROL NO:

NAME:

AGE:

SEX:

ADDRESS:

PAST HISTORY:

PERSONAL HISTORY:

- Alcohol-
- Smoking-
- Tobacco Chewer-
- Sleep-
- Bowel habits-
- Bladder habits-

DIETARY HISTORY:

- Vegetarian Diet-
- Non-vegetarian Diet-

DRUG HISTORY:

FAMILY HISTORY-

- Stroke-
- Cardiovascular Disease-
- Sudden Cardiac Death-
- Hypertension-
- Diabetes mellitus-

PHYSICAL EXAMINATION:

- Conscious-
- Co-operative-
- Oriented-

VITALS:

- TEMPERATURE-
- PULSE RATE-
- RESPIRATORY RATE-
- BLOOD PRESSURE-

GENERAL CONDITION:

- PALLOR-
- ICTERUS-
- CYNOSIS-
- CLUBBING-
- LYMPHADENOPATHY-
- EDEMA-
- ARCUS SENALIS-
- XANTHELESMA-

SYSTEMIC EXAMINATION:

RESPIRATORY SYSTEM-

CARDIOVASCULAR SYSTEM-

CENTRAL NERVOUS SYSTEM-

PER ABDOMEN-

INVESTIGATIONS

- Plasma Free Fatty Acid (PFFA)
- Fasting Blood Sugar
- Total Cholesterol
- High Density Lipoprotein (HDL)
- Low Density Lipoprotein (LDL)
- Very Density Lipoprotein (VLDL)
- Triglycerides
- Transthoracic Echocardiography- Epicardial fat thickness

CONTROLS

Control no	Age	Sex	Address	Past history	Alcohol	Smoking	Tobacco chewing	Sleep	Vegetarian	Non-vegetarian	Drug history	Familyhistory	BP	Pulse	RR	General Condition	Systemic Examination	Plasma Free Fatty Acid	Fasting Blood Sugar	Total cholesterol	HDL	LDL	Trigycerides	Epicardial Fat Thickness
1	45	M	Belagavi	None	Yes	None	None	Adequate		Yes	None	None	120/80	88	18	Normal	Normal	0.18	118	74	46	88	78	7
2	68	M	Belagavi	None	Yes	Yes	None	Adequate		Yes	None	None	110/70	98	18	Normal	Normal	0.2	109	139	58	96	90	7
3	37	F	Belagavi	None	None	None	None	Adequate	Yes		None	None	110/70	72	16	Normal	Normal	0.21	98	146	44	78	72	7
4	44	F	Belagavi	None	None	None	None	Adequate		Yes	None	None	100/70	84	18	Normal	Normal	0.54	120	160	54	102	112	8
5	64	M	Belagavi	None	None	Yes	Yes	Adequate	Yes		None	None	130/90	86	18	Normal	Normal	0.2	134	134	48	84	100	7
6	75	F	Belagavi	None	None	None	None	Adequate	Yes		None	None	110/70	82	18	Arcus senalis present	Normal	0.19	78	78	52	106	77	7
7	69	F	Belagavi	None	None	None	None	Adequate		Yes	None	None	110/70	98	20	Arcus senalis present	Normal	0.21	114	110	56	88	92	8.9
8	55	M	Belagavi	None	None	None	Yes	Adequate		Yes	None	DM in mother	110/70	76	18	Normal	Normal	0.2	130	145	64	102	104	7
9	40	F	Belagavi	None	None	None	None	Adequate	Yes		None	Hypothyroidism in mother	120/80	88	18	Normal	Normal	0.24	92	158	48	92	72	5
10	38	F	Belagavi	None	None	None	None	Adequate		Yes	None	DM and HTN in father	110/70	84	18	Normal	Normal	0.19	112	174	58	100	88	7
11	52	F	Belagavi	None	None	None	None	Adequate	Yes		None	None	110/70	90	16	Normal	Normal	0.18	128	164	62	99	102	8
12	62	M	Belagavi	None	Yes	Yes	None	Adequate		Yes	None	None	130/90	78	20	Arcus senalis present	Normal	0.18	112	136	64	104	123	7.8
13	63	F	Belagavi	None	None	None	None	Adequate		Yes	None	None	120/80	88	18	Normal	Normal	0.18	108	175	70	120	112	7
14	67	F	Belagavi	None	None	None	None	Adequate	Yes		None	None	110/70	86	18	Normal	Normal	0.18	114	179	74	131	142	8
15	54	F	Belagavi	None	None	None	None	Adequate	Yes		None	None	130/90	78	16	Normal	Normal	0.19	109	116	56	98	100	7
16	38	M	Belagavi	None	Yes	None	None	Adequate		Yes	None	DM in father	120/80	64	17	Normal	Normal	0.18	96	148	58	100	98	5
17	33	M	Belagavi	None	Yes	None	None	Adequate		Yes	None	None	120/80	98	18	Normal	Normal	0.18	88	98	61	102	114	7.5
18	48	M	Belagavi	None	Yes	Yes	None	Adequate		Yes	None	Htn in mother	120/80	88	20	Normal	Normal	0.19	100	118	64	100	120	8.2
19	56	F	Belagavi	None	None	None	None	Adequate	Yes		None	None	130/90	76	18	Normal	Normal	0.18	138	104	58	92	84	6.2
20	48	F	Belagavi	None	None	None	None	Adequate	Yes		None	DM and HTN in father	110/70	80	18	Normal	Normal	0.19	114	128	44	88	90	7.3
21	60	M	Belagavi	None	None	Yes	Yes	Adequate	Yes		None	None	130/90	84	18	Normal	Normal	0.19	99	144	58	100	108	7
22	43	M	Belagavi	None	Yes	None	None	Adequate		Yes	None	None	120/80	92	22	Normal	Normal	0.19	132	198	60	94	133	8
23	60	F	Belagavi	None	Yes	None	None	Adequate	Yes		None	None	130/90	76	16	Normal	Normal	0.19	119	184	64	104	118	7.8
24	42	M	Belagavi	None	Yes	Yes	None	Adequate		Yes	None	None	120/80	90	18	Normal	Normal	0.18	98	116	58	96	74	6.5
25	31	M	Belagavi	None	Yes	None	None	Adequate		Yes	None	DM in mother	110/70	74	20	Normal	Normal	0.18	100	164	66	102	110	5
26	58	F	Belagavi	None	None	None	None	Adequate		Yes	None	None	130/90	70	18	Normal	Normal	0.2	92	116	66	100	96	7
27	26	F	Belagavi	None	None	None	None	Adequate	Yes		None	None	120/80	78	18	Normal	Normal	0.2	78	191	60	98	115	5.8
28	40	F	Belagavi	None	None	None	None	Adequate	Yes		None	Hypothyroidism in mother	120/80	90	18	Normal	Normal	0.18	128	164	74	112	140	7
29	44	M	Belagavi	None	Yes	None	None	Adequate		Yes	None	None	110/70	94	18	Normal	Normal	0.22	110	154	78	108	138	7
30	71	M	Belagavi	None	None	None	None	Adequate		Yes	None	None	130/90	78	20	Arcus senalis present	Normal	0.2	124	174	60	98	128	8.5
31	64	F	Belagavi	None	Yes	None	None	Adequate	Yes		None	None	130/90	84	18	Normal	Normal	0.19	131	106	48	104	98	7
32	48	M	Belagavi	None	Yes	Yes	None	Adequate		Yes	None	None	110/70	88	18	Normal	Normal	0.18	119	134	62	96	115	7.4
33	52	F	Belagavi	None	None	None	None	Adequate	Yes		None	None	120/80	72	18	Normal	Normal	0.18	97	128	72	100	142	8

CASES

Case No	Age	Sex	IP NO	Address	Level of Education	Language	Complaints	Hypertension	Duration	Medications	Compliance	DM	Duration	Medications	Compliance	Alcohol	Smoking	Tobacoo	Sleep	Vegeterian Diet	Non Vegeterian Diet	Family History	On statin	Not on Statin
1	72	M	1E+06	Belagavi	7 th Std	Kanada	Difficulty speaking, snoring,night time hypoxic episodes	no	No	No	No	No	No	No	No	Yes, 30 years	No	Yes	Adequate		Yes	No		Yes
2	75	M	1E+06	Belagavi	10 th Std	Marathi, kanada, Hindi	Left upper and lower limb weakness, difficulty speaking since 7.30 am	Yes	15 years	Yes	Yes	No	No	No	No	No	No	N	Adequate		Yes	No		Yes
3	57	M	1E+06	Belagavi	Uneducated	Marathi, Kannada	Dysarthria, Ataxia, Deviation of angle of the mouth to the right	No	No	No	No	Yes	6 years	No	No	Yes, 2 years	No	Yes	Less		Yes	No		Yes
4	60	M	1E+06	Belagavi	5 th Std	Kanada	Right upper and Lower limb weakness,Difficulty in speaking, deviation of angle of mouth to the left	No	No	No	No	No	No	No	No	Yes	Yes	Yes	Less		Yes	CVA in Mother		Yes
5	64	M	1E+06	Belagavi	SC Agricultu	Marathi, kanada, Hindi, English	Left upper limb tingling and numbness, Left upper limb weakness since 1 day, swaying to the left side while walking since 1 day, blurring and diplopia since 1 day	no	No	No	No	Yes	3 years	Yes	No	No	No	N	Adequate	Yes		No		Yes
6	63	F	1E+06	Belagavi	7 th Std	Marathi	Sudden onset giddiness, Tingling in bilateral upper limbs since 6.30 am	Yes	5 years	Yes	Yes	No	No	No	No	No	No	N	Adequate		Yes	No	Yes	
7	47	M	1E+06	Belagavi	BE	Kanada, English	Slurring of speeech since 2 days	no	No	No	No	No	No	No	No	Yes, 10 years	No	N	Adequate	Yes		DM in mother		Yes
8	53	M	1E+06	Belagavi	Engineer	Kanada,Hindi, English	Right Upper and lower limb weakness and slurring of speech since 5 am	no	No	No	No	Yes	5 Years	Yes	Yes	Yes, 15 years	Yes	Yes	Adequate		Yes	Hypertensio n and DM in Father		Yes
9	74	M	1E+06	Belagavi	10 th Std	Kanada	Fall at home at 9 am followed by loss of consiousness	Yes	7 years	No	No	Yes	7 years	No	No	No	Yes	Yes	Less	Yes		No		Yes
10	72	F	1E+06	Belagavi	7 th Std	Kanada	Right UL and LL weakness with difficulty in speaking, vomiting	Yes	1 year	Yes	Yes	No	No	No	No	No	No	N	Adequate	Yes		No		Yes
11	68	M	1E+06	Kurihal	Uneducated	Kanada	Right UL and LL weakness, difficulty walking	no	No	No	No	No	No	No	No	Yes, 30 years	Yes	Yes	Less		Yes	No		Yes
12	53	F	1E+06	Dharwad	BA	Kanada, marathi	Left sided headache, blurring of vision in the right eye, tingling and numbness of the right hand all since 7 days, mild confusional state and dizziness since 5 days	Yes	10 years	Yes	Yes	Yes	3 years	Yes	Yes	No	No	N	Adequate		Yes	DM in mother		Yes
13	74	M	1E+06	Belagavi	BA	Kanada, marathi	Acute onset confusional state and blurring of vision in both eyes since 11 am	Yes	15 years	Yes	Yes	Yes	25 years	Yes	Yes	No	No	N	Adequate	Yes		No	Yes	
14	40	M	1E+06	Belagavi	Engineer	Kanada,Hindi, English	Abrupt onset language impairement with loss of fluency, reduced verbal output, difficulty naming people and objects	no	No	No	No	No	No	No	No	Yes, 10 years	No	N	Adequate	Yes		No		Yes
15	69	F	1E+06	Belagavi	10 th Std	Marathi	Behavioural change since 3-4 days, Right sided UL and LL weakness with fall 1 day	Yes	15 years	Yes	Yes	No	No	No	No	No	No	N	Adequate	Yes		No		Yes
16	48	M	1E+06	Belagavi	SC Agricultu	Kanada	Acute onset vomiting, vertigo, headache, left sided ataxia, mild dysarthria	no	No	No	No	No	No	No	No	No	No	N	Adequate		Yes	No		Yes

Case No	other drugs	BP	Pulse	RR	General Examination	Neurological Examination	Plasma Free fatty Acid	Fasting blood sugars	Total Cholesterol	HDL	LDL	Trigycerides	Transthoracic Echo	MRI Brain	MRI Angiography	CT Brain	CT Angio	Other Co-morbidity	Territory of circulation	Side of the infarct
1	None	140/80	72	18	arcus Senalis	Right handed, Dysarthria	0.2	107	150	34	98	92	8	Hyperacute infarct in left corona radiata	Normal				Anterior	Unilateral
2	Antihypertensives (Temesartan , Hydrochlorthiazide)	130/80	94	16	arcus Senalis	Right handed, Deviation of angle of mouth to the right, Loss of left nasolabial fold, Left eye unable to close,Power in left shoulder 0/5, elbow 1/5 wrist 1/5, handgrip poor, left hip, knee, ankle toes 1/5, Pain, touch, temperature not felt in the left upper and lower limb,Areflexia on the left ul and ll, Plantars left upgoing	0.2	100	182	40	117	124	9	Hyperacute infarct in the right MCA territory.		Acute infarct in the right caudate, lentiform nucleus and anterior limb of internal capsule, hyperacute infarct in right temporal region			Anterior	Unilateral
3	Antiplatelet (Clopidogrel)	120/70	80	18	Normal	Dysarthria, Right mild facial palsy	0.25	131	74	31	27	81	12	Subacute infarct in PCA territory				CABG	Posterior	Unilateral
4	None	80/60	120	34	Drowsy, Arcus Senalis	Drowsy, Not co-operative to time,place and person, Right Upper and Lower limb hypotonia, Right UL shoulder, elbow, wrists and Right hip, knee and ankle power 0/5, Right UL and LL DTR- areflexia, Plantars Right extensors	0.25	196	100	6	7	436	7			Left MCA infarct			Anterior	Unilateral
5	Antidiabetic (Vildagliptin, Metformin)	150/100	76	18	arcus Senalis	Right handed, Diplopia, Left UL hypotonia,Left finger to nose impaired gait-swaying to the left, Bilateral plantars flexors	0.19	409	148	27	78	213	10.8	Late hyperacute infarct in pons	Normal				Posterior	Unilateral
6	Antihypertensive, Antiplatelets, Statin (Metoprolol,losartan, hydrochlorthiazide, ecosprin, clopidogrel, rosuvastatin)	130/80	82	16	Normal	Right handed, normal examination	0.24	99	161	32	100	143	11	Bilateral thalamic infarct				IHD	Posterior	Bilateral
7	None	150/90	78	18	Normal	Right handed, dysarthria,	0.22	98	177	37	118	112	18	Subacute infarct in the left lentiform nucleus, left corona radiata and left parietal region	Normal				Anterior	Unilateral
8	Antidiabetic (Metformin)	150/90	88	18	Normal	Right handed, Dysarthria, Left UMN facial palsy, Right UL and LL Hypotonia, Right UL shoulder, elbow, wrists 1/5, right handgrip poor, right hip, knee ankle toes 2/5, right UL and LL DTR 0, Right plantar extensore	0.2	251	240	35	157	240	10	Subacute infarct in left MCA territory	Occlusion of left MCA				Anterior	Unilateral
9	None	140/80	74	22	Normal	Right handed, E1V1M3, Bilateral pupils sluggishly reactive to light, Bilateral UL ans LL hypotonia, Bilateral UL and LL DTR 1, Right plantar extensor, left plantar flexor	0.2	129	282	27	160	400	20	Hperacute ischemic infarct in artery of percheron					Posterior	Unilateral
10	Antihypertensive (Telmisartan)	120/70	58	22	Normal	Right handed, Right UL and LL hypotonia, Right UL shoulder, elbow, wrists, and right hip, knee, ankle, toes power 0/5, Right UMN facial palsy, Right UL and LL DTR 0, Right conjunctival and corneal reflex absent, Right plantars extensors	0.26	160	138	34	89	75	11	Subacute infarct in the left fronto-parietal, temporal, caudate nucleus, eexternal capsule and left lentiform nucleus	Thrombosis in the intracranial portion of left internal carotid artery and M1 segment of left				Anterior	Unilateral
11	None	110/70	82	18	Arcus senalis, xanthelasma present	Right handed, drowsy, global aphasia, Left eye gaze preference, restricted right lateral gaze movement, right UMN palsy, Right UL and LL hypotonia, right UL shoulder, elbow, wrists, power 0/5, right hip, knee , ankle and toes 1/5, right UL and LL DTR 1, Right plantars mute	0.2	189	160	38	135	140	10	Subacute infarct in the left fronto-parieto- temporal region, left corona radiata, left centrum semi-ovale and left insular cortex	Thrombotic occlusion of M2, M3 and M4 segments of left MCA				Anterior	Unilateral
12	Antihypertensive, antidiabetic (Cilnidipine, metoprolol, Vildagliptin, glimiperide)	140/90	78	18	Normal	Right handed, oriented to time, place and person, calculation impaired, Right sided hemianopia, Right sided blurring of vision,	0.24	179	105	31	50	121	20	Subacute infarct in PCA territory, posterior limb of left internal capsule, left posterior parietal and left occipital region and left temporal region	Occlusion of the left PCA				Posterior	Unilateral
13	Antihypertensive, Antidiabetic,Antiplatelet, Statin (Cilnidipine, glimeperide, rosuvastatin, ecosprin)	130/80	88	20	Normal	Right handed, dressing apraxia, decreased attention, poor recall, repition normal, Left gaze preference, nystagmus present, right UL and LL shoulder, elbow, wrists, hip, knee and ankle 3/5 with right pronator drift, Right plantar extensor	0.2	232	146	28	71	235	7	Subacute infarct in left MCA -PCA watershed territory	Normal				Watershed area	Unilateral
14	None	180/90	88	22	Normal	Right handed, with impaired speech, loss of fluency, comprehension, repition and naming, right hemi-neglect,Right pronator drift, Right UL shoulder, elbow, wrists 4/5 , Right LL 5/5, left UL and LL 5/5Right plantar extensors	0.38	86	165	35	109	107	14	Subacute infarct in left posterior cerebellar lobe, left corona radiata, genu of the left internal capsule, left centrum semi-ovale and right high parietal region					Posterior	Unilateral
15	Antihypertensive, antiseizure (Frisium, Valproic acid, losartan)	130/80	98	20	Normal	Right handed, Apathy, abulia, Recent memory impaired, Right UMN facial palsy, Right UL and LL Hypotonia, Right shoulder 2/5, elbow 3/5, wrists 3/5, fingers 4/5, Right Hip 2/5, Knee 3/5, ankle and toes 3/5, right plantar extensor	0.2	175	150	45	112	140	12	Subacute infarct in left MCA territory				long standing temporal lobe epilepsy	Anterior	Unilateral
16	None	160/100	70	18	Normal	Right handed, horizontal nystagmus, with at rest and left gaze, mild dysarthria, left finger to nose test and knee to heel positive, left dysdiadokinesia present	0.18	195	134	185	71	26	9	Subacute infarct in the cerebellar lobe and vermis on the left side	Normal				Posterior	Unilateral

17	36	M	1E+06	Belagavi	MS	Kanada,Hindi, English	Acute onset vertigo and osilopis, tingling in the right UL and LL and difficulty swallowing since 10.30 pm	Yes	1 year	Yes	Yes	No	No	No	No	No	No	Yes	Adequate		Yes	No		Yes	Antihypertensive (Telmisartan, hydrochlorthiazide)	150/90	100	18	Normal	Right handed, Bilateral restriction of left agze, horizontal gaze evoked nystagmus, right> left palatal palsy, weak neck flexion, right ul and ll 4/5, right sided ataxia, with right finger to nose positive, bilateral plantars mute and gait ataxi\c	0.2	179	110	44	132	235	11	Subacute infarct in the right cerebellar lobe and right occipital region					Posterior	Unilateral
18	64	M	1E+06	Belagavi	Uneducated	Kanada	Weakness of the left UL and LL, with headache, vomiting and hiccups	Yes	7 days	No	No	Yes	1 year	Yes	Yes	No	No	N	Adequate	Yes		No		Yes	Antidiabetic(Metformin, glimeperide)	160/90	70	18	arcus Senalis	Right handed,dysarthria, loss of left nasolabial fold , deviation of angle of the mouth to the right, left upper and lower limb hypotonia, left shoulder, elbow and wrists 2/5left hip,knee and ankle 2/5, left biceps, triceps,,supinator reflexes brisk,bilateral plantars flexors	0.19	104	78	30	33	77	9.1	Acute infacrt in right MCA territory					Anterior	Unilateral
19	67	M	1E+06	Belagavi	10 th Std	Kanada	Loss of vision, left sided upper and lower limb weakness since 1 day	no	No	No	No	No	No	No	No	No	No	Yes	Adequate		Yes	No		Yes	None	180/100	72	20	Arcus senalis present	Right handed, drowsy, not following commands, bilateral pup[ils 3mm reactive to light, left biceps, supinator and triceps +1, left knee and ankle brisk, right plantar flexor and left plantar extensor	0.25	109	110	33	61	78	12	Subacute infarct in right PCA territory, subacute lacunar infarct in left centrum semiovale					Posterior	Unilateral
20	45	F	1E+06	Belagavi	Uneducated	Hindi	Headache, Inability to speak since 9 am	no	No	No	No	Yes	10 years	Yes	No	No	No	N	Adequate		Yes	No		Yes	Antidiabetic (Gylcomet 500)	130/90	76	22	Pallor present, arcs senalis present	Right handed, with decrease in spontaneous speech, loss of comprehension, inability to assess the naming, reading and writing	0.22	120	172	98	40	100	10	Acute infarct in MCA territory					Anterior	Unilateral
21	65	M	1E+06	Belagavi	Engineer	Marathi, kanada, Hindi, English	Slurring of speech, deviation of angle of the mouth since afternoon 2 pm today	no	No	No	No	No	No	No	No	No	No	N	Adequate	Yes		No		Yes	None	180/100	82	18	Arcus senalis present	Right handed, consious oriented, dysarthria present, Left shoulder,elbow, wrists power 2/5, left hip,knee and ankle power 3/5, Power left Upper limb and lower limb hypotonia, sensations normal, bilateral plantars mute	0.21	125	164	36	79	243	9	Subacute infarct in the right frontal region, and right peri-sylvian cortex.					Anterior	Unilateral
22	75	F	1E+06	Belagavi	Uneducated	Kanada	Difficulty in walking since 3 days and dysarthria since 3 days	Yes	10 years	Yes	No	No	No	No	No	No	No	N	Adequate		Yes	No		Yes	Antihypertensive, antihypothyroidism (Tab telmisartan + chlorthalidone 40/12.5, tab Thyronorm 100 mcg)	146/90	90	18	Arcus senalis present	Right handed, consious, irritable, Bilateral lower limbs hypotonia, Bilateral lower limbs hip knee and ankle power 1/5, bilateral plantars mute	0.24	276	139	20	46	364	14	Tiny hyperacute infacrt in bilateral corona radiata	Occlusion of the left internal carotid artery			Hypothyroi dism	Anterior	Bilateral
23	45	M	1E+06	Belagavi	Uneducated	Kanada	Left upper and lower limb weakness and difficulty in swallowing since 6 days	Yes	2 years	Yes	No	No	No	No	No	Yes, 8 years	es, 8 year	Yes	Adequate		Yes	No	Yes		Antihypertensive, Antiplatelet, Statin (Tab Rosycap gold 75/20, Tab Calciguard R 20 mg, Tab Moxidine) 0.3	200/100	66	18	Nil	Right handed, consious,oriented, Left shoulder,elbow, wrists power 4/5, left hip,knee and ankle power is 4/5, Left upper and lower limb hypotonia, Left sided DTR +1, Uvula deviated to the left side, Bilateral plantars mute	0.22	73	212	58	115	193	10	Subacute infarct in the high frontal-parietal region					Anterior	Unilateral
24	55	F	1E+06	Belagavi	Uneducated	Kanada	Headache and vomiting since 4 days, right eye ptosis and swaying to the right side while walking since 3 days	Yes	3 years	Yes	Yes	Yes	8 years	Yes	Yes	No	No	N	Adequate	Yes		No		Yes	Antihypertensive, Antidiabetic (Tab Telmesartan 40 mg, Tab Metformin+Voglibose+Glimeperide)	180/100	98	18	Arcus senalis present	Right handed, Consious, oriented, Right eye complete ptosis, left eye movements restricted in all directions right UL and LL Ataxia, Right shoulder, elbow, wrists, hip, knee and ankle power 4/5, Bilateral planatrs flexors	0.24	221	175	40	90	225	12	Subacute infarcts in the left thalamus and left midbrain					Posterior	Unilateral
25	68	F	1E+06	Belagavi	Uneducated	Kanada	Sudden onset of vertigo and postural dizziness, with imbalance while walking for 2 days, Difficulty in getting up and indicating needs since 1 day.	Yes	4 years	No	No	No	No	No	No	No	No	Yes	Less	Yes		No		Yes	None	160/70	88	20	Arcus senalis, xanthelasma present	Right handed, Consious, expressive aphasia, Left hypoglossal palsy, Bilateral lower limb power across hip knee and ankle joint 1/5, bilatyeral plantars mute	0.21	142	230	38	144	233	14	Subacute infarcts in the bilateral para-sagittal frontal and high frontal regions, left para-sagittal basifrontal and genu of corpus callosum on the left side					Anterior	Bilateral
26	82	M	1E+06	Belagavi	BE	Kanada, English, Hindi,marathi	Slurring of speech, Right sided facial weakness, since 30 minutes	Yes	20 years	Yes	Yes	No	No	No	No	Yes	No	N	Adequate	Yes		No		Yes	Antihypertensive (Tab Metoprolol 25mg)	190/100	96	12	Arcus senalis present	Right handed, consious, oriented, dysarthria, Right sided facial weakness, deviation of the mouth to the left side with blunting of the right nasolabial fold, Right upper limb pronator drift present, Right plantar extensor	0.22	104	241	56	159	132	12	Acute infarct in PCA territory					Posterior	Unilateral
27	72	M	1E+06	Belagavi	10 th STD	Kanada, marathi	Difficulty in expressing himself since 1 day, deviation of the angle of the mouth to the left side since 1 day	Yes	25 years	No	No	No	No	No	No	Yes, 30 years	Y	Yes	Adequate	Yes		No		Yes	None	180/100	112	22	Arcus senalis, xanthelasma present	Right handed, consious, oriented, intact comprehension, with decrease in spontaneous speech, bilateral plantars are mute	0.25	191	117	41	57	95	14	Subacute infarct in the posterior aspect of the left perisylvian fissure and posterior aspect of the left lentiform nucleus					Anterior	Unilateral
28	60	F	1E+06	Belagavi	Uneducated	Kanada	Inability to speak and right upper and lower limb weakness since 1 day	Yes	20 years	No	No	Yes	20 Years	No	No	No	No	N	Adequate	Yes		No		Yes	None	130/80	84	18	Arcus senalis present	Right handed, consious, oriented, dysarthria, right uataxic hemiparesis, right shoulder, elbow, wrists 3/5, right hip, knee and ankle 4/5, left sided deviation of angle of the mouth with loss of right nasolabial fold, right plantar extensor	0.33	190	160	32	112	180	12		Left MCA infarct				Anterior	Unilateral
29	62	F	1E+06	Belagavi	B Com	Kanada, English, Hindi,marathi	Slurring of speech and weakness of thr right upper limb since 1 day	Yes	25 years	Yes	Yes	Yes	25 years	Yes	Yes	No	No	N	Adequate		Yes	No	Yes		Antihypertensive, Antidiabetic, Antiplatelets, Statin, Anti-hypothyroid (Tab Amaryl M2, Tab Glavas met, Inj human mixtard 30/70, Tab thyronorm 75 mcg, tab Metosartan 40/50, Tab cilacar 5 mg, Tab Jupiros F, Tab stiloz 100 mg, Tab ivabrad 5 mg)	130/80	82	16	Bilateral non pitting edema	Right handed, consious, with loss of immediate and recent memory, Right shoulder, elbow, wrisits power 4/5, dysarthria present, right plantar extensor	0.27	256	190	110	34	200	12		Left MCA infarct			Hypothyroi d	Anterior	Unilateral
30	49	F	1E+06	Belagavi	12 Std	Kanada, marathi	Difficulty in speaking, weakness of right upper and lower lombs since 2 days	Yes	20 years	Yes	Yes	Yes	20 Years	Yes	Yes	No	No	N	Adequate		Yes	No		Yes	Antidiabetic, Antihypertensive (tab Amaryl M1, Inj human Mixtard 30/70, Tab Telmisartan + hydrochlorthiazide 40/12.5)	160/90	118	18	Arcus senalis present	Right handed, Consious, E3V2M5, bilateral pupils 2mm sluggishly reactive to light, right shoulder, elbow and wrists with right hip, knee and ankle hypotonia, power 2/5, right DTR +1, bilateral plantars mute	0.24	332	275	51	190	169	14	Subacute infarcts in the left MCA territory					Anterior	Unilateral
31	50	F	1E+06	Belagavi	12 Std	Kanada,Hindi, English	Weakness of the left upper and lower limbs, slurring of the speech and deviation of the angle of the mouth to the right since 1 day	no	No	No	No	No	No	No	No	No	No	N	Adequate	Yes		No		Yes	Ayurvedic preparation	140/90	112	16	Normal	Right handed, consious, oriented, with loss of left nasolabial fold and deviation of angle of the mouth to the right, left shoulder, elbow, wrists hypotonia with power shoulder, elbow and wrists 0/5, left hip, knee and ankle 1/5 left plantar extensor	0.23	98	160	90	44	184	10	Subacute infarct in right MCA territory	Occlusion of the M1 segment of the right middle cerebral arterywith apuscity of few of the cortical				Anterior	Unilateral
32	50	F	1E+06	Belagavi	12 th Std	Kanada	Right sided upper and lower limb weakness, difficulty walking since 2 days	Yes	15 years	Yes	No	Yes	15 years	Yes	No	No	No	N	Less		Yes	No	Yes		Antidiabetics, Antihypertensive, Antiplatelets, Statin (Tab rosuvastatin, aspirin, Clopidogrel 10/75/75, Tab telmisartan+ metoprolol 40/50, Tab Metformin/voglibose/glimeperide)	120/80	90	20	Normal	Right handed, consious, oriented, right upper and lower limb hypotonia, right shoulder, elbow, wrists 4/5, right hip,knee and ankle 4/5, bilateral plantars mute	0.29	300	200	32	158	238	14			Subacute infacrt in the left thalamus			Posterior	Unilateral
33	75	M	1E+06	Belagavi	12th Std	Kanada	Left sided upper limb and lower limb weakness, slurring of the speech since 8 hours	Yes	10 years	No	No	No	No	No	No	No	No	Yes	Adequate		Yes	No		Yes	None	130/80	98	20	Arcus senalis present	Right handed, consious, oriented to place and person not oriented to time, restriction of eye movement to the right side, right shoulder,elbow,wrists 4/5, left shoulder elbow, wrists, hip, knee and ankle joint 0/5 left upper and lower limb hypotonia, left sided dtr 0,bilateral plantars mute.	0.38	170	175	30	114	156	14	Subacute infarct in the right frontal , caudate nucleus, lentiform nucleus, thalamus,and insular cortex of the left side					Anterior	Unilateral