STUDY ON CORRELATION BTEWEEN SERUM CORTISOL AND SEVERITY OF ACUTE ISCHEMIC STROKE IN PATIENTS

BY

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STUDY ON CORRELATION BETWEEN SERUM CORTISOL AND SEVERITY OF ACUTE ISCHEMIC STROKE IN PATIENTS

MD IN GENERAL MEDICINE

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ABSTRACT

INTRODUCTION: Numerous clinical factors, such as the number of symptoms present and advanced age, have been suggested as potential indicators of prognosis in acute stroke patients. But finding a biomarker to forecast the course of an acute stroke is really important. The hypothalamic-pituitary-adrenal (HPA) axis is activated by the stress response that follows an acute stroke. According to several research, patients with acute stroke and higher serum cortisol levels have a greater infarct volume, more severe strokes, and worse outcomes, including death

AIM AND OBJECTIVE :

To assess the relationship of serum cortisol levels to the severity of acute ischemic stroke.

MATERIALS AND METHODS:

About 70 patients of acute isch emic str oke who met the inclusion criteria and were admitted to the ICU or Wards of the BLDEUS Shri BM Patil Medical College and Research Center, Vijayapura, within 72 hours after the acu te neurol ogical eve nt Patients who met the inclusion criteria and attended the medical OPD or executive health check-up programmes were included in the study. At admission, a stroke protocol was obtained to confirm an acute ischemic stroke. All patients had their NI HSS (National In stitute of He alth Stroke Scale) scores evaluated for severity upon admission and on days three and

five. On the AM of the following day, serum cortisol concentrations were assessed. Chi-Square test is used to determine whether there is a relationship between serum co rtisol levels and stroke severity. Every statistical analysis is carried out utilising SPSS (so ftware pa ckage used fo r sta tistical an alysis) package.

KEYWORDS: Clinical severity, Functional outcome, A cute Isc hemic Str oke Scales, HP A Axis, Seru m Corti sol

INTRODUCTION

Numerous clin ical factors, such as the number of symptoms present and advanced age, have been suggested as potential indicators of prognosis in acute stroke patients. But finding a biomarker to forecast the course of an acute stroke is really important. After an acute stroke, the period that follows can be seen as a response to the stressful event. The sympathetic nervous system and hypothalamic-pituitary-adrenal (HPA) axis are activated as a result of this stress response. Because of the altered HPA axis, the endocrine changes are the first detectable changes in acute stroke. Cortisol is a hormone that is connected to the HPA axis and has a strong circadian rhythm, with levels peaking early in the day and troughing later.

The metabolism of glucose[2], fat, and protein, as well as cardiovascular reactivity, are significantly impacted by cortisol. There are concentrates on which sho wed that raised serum corti sol level related with a lot of diminished actual capability and hindered degree of cognizance. According to Fiorentino et al.'s findings, the levels of cortisol in a patient's saliva ca n be u sed as a bio logical ma rker to

identify patien ts who are more likely to receive less benefit from inpatient rehabilitation services. Numerous studies also demonstrate that; Acute ischemic stroke and SAH have been linked to elevated cortisol levels. In some studies, elevated levels of cortisol in the blood and urine are linked to worse outcomes, including death[4,5], a larger infarct volume, and greater stroke severity. The acute confusional state is significantly correlated with elevated serum cortisol levels following the acute event.

This study dissertation's main goal is to evaluate the theoretical association between elevated single serum cortisol levels and enhanced acute ischemic stroke severity. Despite the fact that cortisol levels vary throughout the day due to disturbances in the HPA axis, it has been demonstrated that acute stroke causes the normal circadian rhythm of cortisol to be suspended.

AIM OF THE STUDY:

To assess the relationship of serum cortisol levels to the

Severity of acute ischemic stroke

SUPPORT FOR THE STUDY:

The allocation of therapeutic options depends on the early prediction of the result. One of the earliest changes brought on by stress following cerebral ischemia is the hypothalamus-pituitary-adrenal axis. Therefore, in order to gauge the severity of an acute ischemic stroke, we assessed serum cortisol levels. Wen Jie Zi and Jie Shuai have undertaken studies on cortisol.

Cortisol's ability to serve as an indep endent sho rt-term prog nostic ma rker of fun ctional out come and de ath in Chin ese patients with acu te ische mic stroke was demonstrated as a prognostic marker of short-term outcome in Chinese patients with acute ischemic stroke despite the presence of confounding variables. The Cortisol clinical score's prognostic data can be significantly expanded with the combined model.

REVIEW 0F LITERATURE

CORTISOL

STRUCTURE OF CORTISOL:

Cortisol, the most powerful gluco corticoid, is responsible for 95% of glucocorticoid activity. The zona fasciculata and zona reticularis secrete this hormone from the adrenal cortex, but the former is largely responsible for its synthesis. The substance that produces cortisol is cholesterol. There is the

cyclopentanoperhydrophenanthrene nucleus. It is a C21 steroid with a Delta-4-3-keto configuration in the A ring and 17 hydroxy groups at carbons 11 and 21.

BIOSYNTHESIS OF CORTISOL:

All steroids have cholest erol as their precursor. T he majority of cholesterol comes from LDL, which is a type of blood fat. But the acetate also contributes to the production of some cholesterol. LDL receptors are abundant in adrenocortical cells. Lipid droplets hold the cholesterol once it has been esterified. Cholesterol ester hydrolase works in the lip id dr oplets to catalyse the production of free chole sterol. The cholesterol is transported to mitochondria by sterol carrier protein.Additionally, the side chain cleavage enzyme known as cholesterol desmolase

The conversion of cholesterol into pregnenalone is catalysed by P450scc or CYP11A1, a member of the cytochrome P450 superfamily. This process takes place in the mitochondria.Pregnenalone formed when it penetrates the smooth endoplasmic reticulum.3 beta hydroxysteroid dehydrogenase catalyses the dehydrogenation of progesterone to produce the hormone. The enzyme 3 beta hydroxysteroid dehydrogenase has a molecular weight of 46000 and a unique characteristic in that it does not belong to the cytochrome P450 superfamily.

By hydroxylating progesterone, 11-deoxycorticosterone and the smooth endoplasmic reticulum is where 17 alpha hydroxyprogesterone is converted to deoxy cortisol. 21beta-hydroxylase, commonly known as P450c21 or CYP21A2, is a member of the cytochrome P450 superfamily and catalyses the aforementioned reaction.

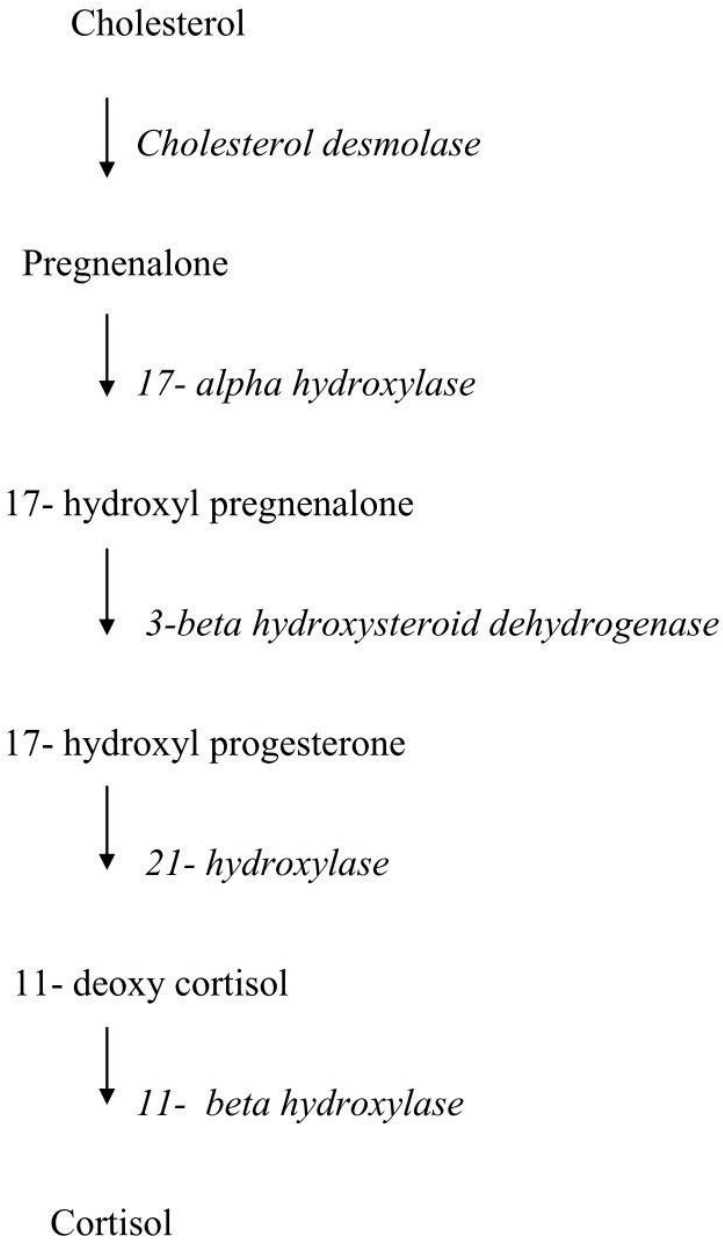
11-deoxycortisol and 11-deoxycorticosterone are released into the mitochondria. P450c11, also known as CYP11B1 or 11beta-hydroxylase, is a member of the

cytochrome P450 superfamily. The conversion of 11-deoxycorticosterone to corticosterone and 11-deoxycortisol to cortisol is catalysed by this enzyme. This happens in the adrenal cortex's zona reticularis and zona fasciculata. As a result, substrates are often shuttled in and out of mitochondria during steroidogenesis.

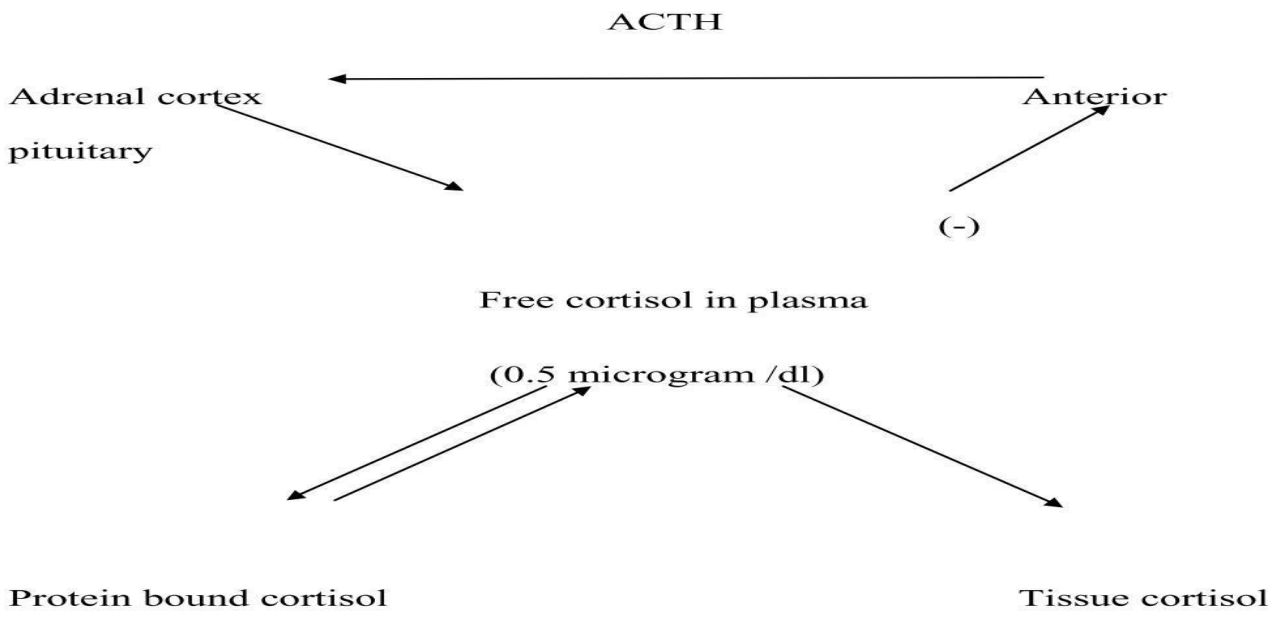
Plasma membranes of the adrenocortical cell are bound by ACTH with high affinity. Through Gs, the adenylyl cyclase is triggered by this. Pregnenalone and its derivatives are rapidly increasing in synthesis and secretion downstream of the adenylyl cyclase activation.

However, the release of inflammatory cytokines can impair adrenal steroidogenesis in sepsis.

SYNTHESIS OF CORTISOL



TRANSPORTATION OF CORTISOL



In the bloodstream, corticoste roid-bin ding glo bulin or al pha globu lin bi nds to cor tisol. Albumin is also less strongly bound to cortisol. The binding of cortico sterone is also similar, but to a lesser extent. Compared to corticos terone, which has a half-life of approximately 50 mi nutes, cor tisol has a half-life of approximately 60 to 90 min utes. However, bound steroids lack physiological activity. Urine only contains a small amount of free cortisol and corticosterone due to protein binding.

The preceding flowchart shows the equilib rium betwe en cor tisol and its binding protein as well as how binding affects tissue supplies and ACTH secretion. The tissues are constantly provided with free cortisol because the bound cortisol serves as a hormone reservoir that circulates.

Although the binding sites of cort isol bin ding glob ulin (CBG) become saturated when the total levels of plasma corti sol rises above 20 microgram/dl, there is very little cortisol present in the plasma at normal levels (13.5

microgram/dl or 375 Nmol/L). Cortisol's tendency to bind to albumin rises with plasma concentration, with the unbound fraction experiencing the greatest increase.

Cortisol is made in the liver, and estrogen makes more of it.During pregnancy, the levels of Cortisol Binding Globulin rise. Multiple myeloma, nephrosis, and cirrhosis of the liver all result in lower levels.

More of the cortisol is bound as CBG levels rise. The levels of free cortisol begin to decrease. Until a new equilibrium is reached, at which the bound cortisol is elevated but the free cortisol is normal, this event stimulates ACTH and increases cortisol secretion.

When CBG levels fall, changes occur in the opposite direction. This explains why pati ents with nep hrosis have low total pl asma corti sol without symp toms of gluco corticoid defici ency and pregnant women have high total plasma cortisol levels without symptoms of excess glucocorticoids.

METABOLISM OF CORTISOL:

Significant site of digestion of cor tisol is Liver. Dihydrocortisol replaces the majority of cor tisol. which is changed into tetrahydrocortisol later. It is then bound to glucuronic acid. The glucuronyl transferase system is the catalyst for this reaction. Additionally, it acts as a catalyst for the synthesis of several hormones and drugs, including bilirubin glucoronides. The enzyme system is notably inhibited by a competitive inhibition between these substrates.

The enzyme 11 – be ta hyd roxyl Ste roid dehyd rogenase is found in the liver and other tissues in at least two different forms. Cortisol to cortisone conversion and the reverse reaction are both catalyzed by Type 1, but this enzyme's primary role is as a reductase, making cor tisol from cor ticosterone. However, only the conversion of cor tisol to cor tisone is triggered by type 2.

Due to its extensive use in medicine, cortisone is well-known as an ac tive glucoc orticoid because it is con verted into cor tisol. It isn't discharged in that frame of mind in adrenal organs.

Cortisol and corticosterone's tetrahydroglucoronide derivatives are water-soluble. After entering the circulation, they do not bind to the protein. They are partly quickly excreted in the urine through tubular secretion.

The 17 ketoste roid deri vatives of co rtisol and cort isone are converted into about 10% of the secreted cortisol in the liver. After being mostly bound to sulfate, the ketosteroids are excreted in the urine. The 20-hydroxyl derivatives and other metabolites are produced.

A piece of the cortisol likewise enters the entero hepatic dissemination. Stool contains approximately 15% of the cortisol that is secreted. Corticosterone's metabolism is similar to that of cortisol, but it does not produce a 17-k etosteroid deriva tive. Glucocorticoid inactivation in the liver slows down in conditions like liver disease, surgery, and stress. As a result, plasma free cortisol levels rise more in stressed humans than when maximal AC TH stimu lation is used in the absence of stress.

REGULATION OF CORTISOL SECRETION:

ACTH'S ROLE:

Both the basal cortisol secretion and the stress-induced increased cortisol secretion are dependent on ACTH from the anterior pituitary. The adrenal gland becomes more responsive to subsequent doses of ACTH, which results in an immediate increase in cortisol secretion**.**

CIRCADIAN RHYTHM:

In response to ACTH secretion, which occurs in irregular bursts throu ghout t he d ay, plasma cortisol tends to fluctuate. The early morning bursts are more frequent and account for 75% of daily cortisol production between 4AM and 10AM. This diur nal musicality in the emitting example of AC TH is found in patients with adrenal deficiency getting steady portions of the glucocorticoids. The hypothalamic suprachiasmatic nuclei house the biologic clock that is in charge of the diurnal secretion of ACTH. The adrenal cycle also lengthens when the experimental day is extended to more than 24 hours, but the rise in AC TH release occurs during sleep.

CORTISOL FEEDBACK:

The levels of free glucocorticoid are what prevent ACTH from being secreted. Cortisol levels in the blood are inversely proportional to the degree of pituitary inhibition. The inhibitory effect occurs at both the hypothalamus and pituitary levels. Although more rapid fast feedback does occur, the inhibition is mainly caused by an act ion on D NA. The maxim al inhib ition takes hours to deve lop. Similar to how various steroids inhibit ACTH, glucocorticoids are potent.

The sum of two opposing forces determines the rate of ACTH secretion. glucocorticoids' braking effect on ACTH secretion, which is proportional to their level in the blood, and the culmination of neu ral and conceivably other sti muli that pass through the hypoth alamus and cause an inc rease in ACTH production.

PHYSIOLOGICAL EFFECTS OF CORTISOL:

The bindi ng of co rtisol to the glucoc orticoid recep tors enables cortisol to perform its many functions. The steroid receptor complex then promotes the transcription of particular DNA segments by acting as transcription factors. The hormone receptor complex and the glucocorticoid response elements must also interact appropriately with other proteins in the cell call ed transcri ption fac tors. Cortisol alters the synt hesis of mRNA for the protein that mediates the numerous physiologic effects by increasing or decreasing many genes. As a result, the physiologic effects of cortisol don't happen right away; instead, it takes 45 to 60 min utes fo r th e pro teins to be made and several hours to days for them to fully develop. Cortisol also has actions that aren't related to DNA.

STROKE

DEFINITION:

A stroke is a sudden onset of neur ological impairment that lasts for more than 24 hours and is caused by a focal vascular cause. Cerebral ischemia occurs when blood flow is reduced for more than a few seconds. If blood flow stops for more than a few minutes, the tissue in the brain can suffer an infarction or die.

A condition is called a "Transient Ischemic Attack" wh0en every neurological sympt om and s ign go away within 24 hours. An embolus from an arterial source that is proxi mal or from the heart, as well as throm bosis of major cere bral ve ssels, can result in focal ischemia. Intracranial discharge is brought about by seeping into cerebrum parenchyma and delivering mass outcomes prompting indication of neurological side effects.

EPIDEMIOLOGY:

From 1969 to 1971, a study on stroke was carried out in and aro und Vel lore. From 1971 to 1974, a study was carried out in Roh tak in N orth India15. In comp arison to Chinese and Cauca sians, India's population has a lower rate of stroke. It has been determined that the mo rtality rate from stroke is only 1.2% when comp ared to that from other causes. The age-adjus ted preva lence rate of stro ke was 250 -350 /100000. Age increases the proportion of stroke-related deaths. One stroke death per woman is the result of a stroke.

RISK FACTORS FOR STROKE:

NON-MODIFIABLE RISK FACTORS:

AGE: - The mo st significant risk fa ctor for str oke is age. After the age of 55, the rate of stroke 2 times in men and women for every 10 years after that.

SEX: Men have a stroke incidence that is 1.25 times higher than that of women. Nonetheless, in light of the fact that ladies live longer than men, a larger number of ladies pass on from stroke than men.

RACE: - Blacks have a higher stroke mortality rate than Whites do. Between the ages of 45 and 55, African-American mortality rates are four to five times higher than those of white people. As people get older, this gap narrows. Jap anese and Chi nese, in particular, have extre mely hig h rates of str oke, and Chinese have a higher prevalence of intracranial atherosclerosis.

Inheritance: - Stroke is more likely to run in fa milies beca use of stroke ten dency, genetic determination of stroke risk factors, and common familial exposure to lifestyle and environmental risks.

MODIFIABLE RISK FACTORS:

HYPER TENSION: When a person is said to have hypertension, their relative risk of stroke increases. Strong stroke risk factors include hypertension. The odds ratio is 4 for 50-year-olds and 1 for 90-year-olds. Treatment of hypertension resulted in a 40% reduction in fatal strokes and a 38% reduction in all strokes in a global study involving 50.000 patients from 17 hypertension treatment trials. Antihypertensive treatment of systolic hypertension in the elderly greatly reduces the risk of stroke.

Disease of the heart: One of the most potent risk factors for stroke is atrial fibrillation. Atrial fibrillation has a higher incidence and prevalence with age. The Fra mingham st udy demonstrated that individuals' attribu table ri sk of atrial fibrillation for st roke inc reased from 1.5% in people between the ages of 50 and 59 to 23.5% in people between the ages of 80 and 89. It is established that atrial fibrillation is the cause of nearly half of cardio embolic strokes. Warfarin anticoagulation reduces stroke risk by 68% in a pooled analysis of AF trials.

A stroke is more likely to occur in people who have heart valve abnorm alities like mitr al valve prolapse and mit ral valve ste nosis. Mitral annular calcification is another stroke risk factor. With both AF and mitral annular calcification, the chance of having a stroke goes up by five times.

According to the Framingham study, the age ad justed risk of stro ke doubles in male and female for ev0ery ten mm increase in le ft atr ial si ze. Valvular strands are a significant stroke-related finding. The mitral and aortic valves are connected

to these filamentous processes. Two fundamental examinations showed that these valv ular stran ds incline toward st roke, yet more imminent investigations are required.

Myocardial disease and a patent foramen ovale are also stroke risk factors. After cardiac catheterization and angioplasty, there is a 0.2 percent to 0.3 percent risk of stroke. Cardioversion, pacing, electrophysiological procedures, and radiofrequency ablation all have the potential to cause embolic complications. Prospective epidemiological studies revealed that diabetics have a relati ve ris k of stro ke that ran ges from 1.8 to 3.0 because of the increased prevalence of atherosclerosis and atherogenic risk factors like ob esity, hyperl ipidaemia, and hypertension. According to the Fram ingham study, people with glucose intolerance have twice the risk of stroke in diabetics than in non-diabetics. Diabetes plays a significant role as a stroke risk factor due to hyperinsulinemia and insulin resistance.

LIPIDS: Both the inci dence of st roke and the prote ctive rol e of H DL cho lesterol in extr acranial athero sclerosis are positively correlated with LDL and total cholesterol. The lovastatin group had fewer strokes than the placebo group in an asymptomatic carotid artery plaque study.

SMOKING: Stroke and smoking have a clear dose-response relationship. The stroke-causing rel ative ri sk (RR) of s moking is 2. According to the Fra mingham study, quitting smoking immediately lowers the risk of stroke.

ALCOHOL: In a review of stroke studies, a J-shaped association curve was found to be associated with moderate alcohol consumption and ischemic stroke. With increased alcohol consumption comes an increased risk of brain hemorrhage.

CAUSES OF ISCHEMIC STROKE:

COMMON CAUSES:

- THROMBOSIS:
- Lar ge ve ssel throm bosis
- Lacu nar stroke De hydration
- EMBO LIC O CCLUSION:
- ARTE RY TO AR TERY OCCL USION
- Caro tid bifurca tion

- Ar terial dis section
- Aortic arch
- CARDI O EM BOLIC
- At rial fibrill ation
- Mura l thro mbus
- Myo cardial Infar ction
- Dila ted cardiom yopathy
- Valvular lesions
- Mitral stenosis
- Mechanical valve

UNCOMMON CAUSES:

- Hyperc oagulable Dis orders
- Prote in C and S defic iency
- Antith rombin 3 defi ciency
- Antiph ospholipid anti body syndro me
- Fact or 5 Lei den mu tation
- Prothrombin G20210 mutation
- Cancer
- Sicklecell anaemia

- Betathalassemia
- Polycythaemia
- Homo cysteinemia

STROKE PATHOPHYSIOLOGY

The two most significant mechanisms that result in bra in dam age in stro ke are ischem ia and haemo rrhage. However, ische mic str oke accounts for 80 percent of strokes, in which neurons are denied the necessary substrates due to reduced or absent blood flow. The brain is incapable of anaerobic metabolism and does not store the primary energy substrate, glucose; consequently, focal ischemia occurs quickly. Ischemia occurs in the vascular territory when an embolus or thrombus blocks a cerebral artery. It is difficult to tell the difference between an embolus-caused lesion and a thrombus-caused lesion. Numerous factors influence the severity and progression of ischemic injury

DURATION AND ONSET RATES: An ische mic ev ent with a slow ons et and sh ort du ration is tolerated by the brain.

CIRCULATION COLLATERAL: There is improved result related with guarantee course

Pulse: For cerebral perfusion pressure to remain constant, adequate systemic blood pressure is required. Global ischemia of the brain can result from systemic hypotension.

TEMPERATURE: raised body temperature is associated to increased cerebral injury.

FACTORS HEMATOLOGICALS: The growth and size of microscopic thrombi, which are made worse by the hypercoagulable state.

Metabolism of glucose: Hypo- and hyperglycemia have a negative impact on an infarct's size.

CEREBRAL DRAINAGE: 50-60 ml/100g/mi is the normal cerebral blood flow. When cerebral blood flow (CBF) decreases, cerebral autoregulatory mechanisms cause local vasodilation. In order to safeguard the energy reserves, synaptic activity decreases when cerebral blood flow falls below 20 ml/100 g/min. When the cereb ral b lood flow is less than 10 ml/100g /mi n, ne uronal damage can't be fixed.

MECHANISM AS A CAUSE OF NEURONAL DAMAGE:

Ischemia prompts the arrival of horrendous vasoactive compounds by plate lets, leu cocytes, end othelium and other neu ronal cells, brings about development of a microthro mbi. The impairment of circulation in cerebral arterioles and capillaries is the result of these microthrombi.

Hypoxic ischemic neuronal injury develops when certain neurotransmitters, such as glutamate28-32 and aspartate, overreact. Excitotoxicity is a process that occurs when cellular energy stores are depleted. Normally, glutamate is found within the synaptic terminals. It is removed from the extracellular space by an energy-dependent process. When there is a lack of energy, glutamate builds up outside of cells, causing calcium channels to open. N-methyl-D-aspartate (NMDA) and alpha-amino-3-hydroxy-5-methyl-4 isoxanolepropionate (AMPA) receptors are linked to this calcium channel. Persistent membrane depolarization results in the influx of calcium, chloride, sodium, and potassium ions as well as potassium efflux.

Intracellular calcium activates a number of destructive enzymes, including protea ses, endonu cleases, and lipa ses. This causes arrival of cyt okines bringing about lo ss of cell honesty.

Leuko cyte recruit ment to the is chemic area begins as early as 30 minutes after the event of isch emia and repe rfusion.

Vasoactive substances like oxygen free radicals and arachidonic acid metabolites are activated by the leukocyte. Vasoc onstriction, inc reased perm eability, incre ased leuk ocyte adhesion to the endot helial wall, increased platelet aggre gation, and immu noregulation are the outcomes of these factors.

Response of Endothelial Cells: The first cell to respond to hypoxia is the endothelial cell. It expands and forms a microvilli on the cell's luminal side. Leukocytes, erythrocytes, and platelets mechanically plug the capillary vessel as a result of this decreased luminal patency. The endoth elin pept ides, NO, and eicos anoids that are me diated by endoth elial cells alter the vascular tone of the microcirculation. When the endothelial adhesion molecules are activated, they help

the leukocyte stick to the endothelial wall, which is a big part of how the inflammation process starts.

ISCHEMIC PENUMBRA

Ische mic obscuration is a zone of oligemia which encompasses the center of localized necrosis in something like an hour of hypoxic-ischemic affront, where autoregulation is ineffectual. — The crucial time period in which this volume of brain tissue is vulnerable is known as the "window of opportunity." because reperfusion of the ischemic but viable brain tissue within a critical period of time (2-4 hours) can completely or partially reverse the neurological effects of ischemia.

The cere bral bl ood stream is 25% to half of typical in the ischemic obscuration (IP), consequently there is protection of some energy digestion. In this ischemic penumbra, the function and integrity of the cells are maintained for varying lengths of time. The pathophysiology of IP is closely linked to the production of spontaneous waves of depolarization (SWD). Ischemic foci in the periinfarct zone and the ischemic core are the sources of this spontaneous wave of depolarization.

Sustained release of glutamate and extracellular potassium is also linked to the emergence of spontaneous waves of depolarization. The glutamate receptor antagonists can dampen this spontaneous wave of depolarization.

Rapid or hypoxic depolarization eventually triumphs over irreversible neuronal death.

DEATH IN THE NEURON: The injured neurons die through two processes: Necrosis due to coagulation Apoptosis

COAGULATION NECROSIS:

A process known as consolidation necrosis (CN) occurs when a cell dies without triggering an inflammatory response in its living neighbors. This kind of cell death is linked to the effects of damage to the plasma membrane from chemicals, physical damage, or osmotic damage. This is in contrast to liquefaction necrosis, in which the inflammatory process fills the empty space left by dying cells with pus.

In coagulative necrosis, the cell first expands, then contracts, and then it goes through pyknosis, which is the condensation of nuclear chromatin. More than six to twelve hours this cycle develops. Within a day, extensive chromatolysis causes pan necrosis. The astrocytes swell and break up. Myelin sheath degeneration follows. Eosinophilic cytoplasm and slender nuclei indicate irreversible cell injury between 8 and 12 hours after the occlu sion of the ar tery.

APOPTOSIS:

Ischemia is one of the conditions in which brain neurons are programmed to die. Atomic harm happens first during apoptosis. The plasma membrane and mitochondrial membrane remain intact until very late in the process. Latent suicide proteins, triggered by ischemia, initiate the autolytic process, resulting in cell death. This autolysis is mediated by cleavage of the DNA.

Apoptosis begins after one hour of ischemic injury, whereas coagulative necrosis begins six hours later. Neuronal death could be avoided by altering the DNA cleavage process, according to the hypothesis.

ISCHEMIC STROKE:

There are three main causes of ischemic stroke: 1. 2. A thrombosis Embolism No. 3 Gl obal isc hemia, but not all types of ische mic str oke fall into these ca tegories.

Throm bosis:The main neurotic component of vascular deterrent is atherosclerosis.

The struc ture, consi stency, and comp osition of an atherosclerotic plaque determines its suscep tibility to ulceration, disruption, and fracture. Other pathological changes include calcification, intraplaque hemorrhage, thrombosis, ulcerations, and calcification. Endothelium is disrupted as a result, activating numerous vasoactive enzymes. The adhesion and aggregation of platelets that results in platelets and fibrin nidi follow next. Within one hour, the inflammatory process is started by the leukocytes.

In addition to atherosclerosis, hypercoagulable state, fibromuscular dysplasia, arthritis, and vessel wall dissection are other conditions that can result in thrombotic occlusion.

Occlusion of the deep penetrating artery results in lupus infarcts. The small arteriole becomes tortuous due to long-term hypertension and diabetes mellitus, resulting in subintimal dissections and microaneurysms. Because of this, tiny

thrombi can occlude the arteriole. Due to the deposition of fibrin, lipohyalinosis is the underlying pathological mechanism.

EMBOLISM:

Embolic stroke happens because of embolization from various wellsprings of a course of focal dissemination. Mate rials that can emb olize are athe romatous pla que pi eces, fi brin, cl ot,a ir,fa t,tum or or meta stases, unfamiliar bodies, bacterial bunches. The superficial branches of the superior cerebellar and cerebral arteries are the most common targets. The majority of emboli settle in the distribution of the middle cerebral artery because nearly 80% of the blood carried by the large neck arteries flows through it.

EMBOLIC STROKE OF THE CARDIO:

Cardio embolism is the cause of 20% of all ischemic strokes. T he thro mbotic m aterial that is att ached to the l eft ventri cle, atria, or valves disintegrates and enters the circu lation. A TIA can only be caused by the thrombi lysing or fragmenting too quickly. Stroke occurs when the occlusion lasts longer. An embolic stroke occurs suddenly and causes the most damage to the brain at the tim0e of pre sentation. Pet echial haemor rhage typically occurs at the site of isc hemic territ ory following reperfusion. How ever, it must be distinguis hed from haemorrhage into the isc hemic ar ea, which can result in mass effect and a decline in neurological function, and it has no neurolo gical sign ificance.

Mi ddle cere bral ar tery, post erior cer ebral artery or its branches, and occasionally anterior cerebral artery are the vessels most frequently affected by heart embolism. Nonrheu matic AF, myocar dial infa rction (MI), rheu matic he art dis ease (RHD), and isch emic cardio myopathy are the most significant causes of cardioembolism.

When venous thrombi migrate into the arterial circulation via the patent foramen ovale or ASD, paradoxical embolization occurs. Other than a venous clot, paradoxical embolization can also be caused by fat, air, amniotic fluid, or bacterial endocarditis. Septic emboli can arise from bacterial vegetation. Bacterial endocarditis is more likely to be present in a stroke patient with multiple symptoms.

Embolic Stroke:

Over an atherosclerotic plaque, a thrombus can form, which can then embolise into an intracranial artery, resulting in an embolic stroke from one artery to another. Atherosclerosis of the carotid bifurcation is the most prevalent cause of artery-to-artery embolic stroke. The prevailing component of cerebrum ischemia is supply route to conduit embolism as opposed to nearby apoplexy dissimilar to in myocardial vessels.

Intracra nial athe rosclerosis, disse ction of inte0 rnal carotid or ver tebral arte ries, or even the vessels outside of the Willis circle, are additional causes of artery-to-artery embolic stroke.

The ability of the embolus to cause vasospasm by acting as a vascular irritant is crucial to the outcome of the embolic stroke. Due to the more pliable and less atherosclerotic vessels, young patients are more likely to experience it.

The term "haemorrhagic transformation of an ischemic infarct" refers to bleeding that occurs within necrotic cerebral tissue. Haemorrhagic transformation occurs when an embolus is lysed spontaneously and blood flow is restored, resulting in reperfusion. Reperf usion can result from coll ateral circu lation from leptom eningeal vess els when arterial occlusion persists.

The most important factors that contribute to haemorrhagic infarctions are: Dimensions of the infarct 3. Circulation of collateral Utilization of anticoagulants treatment with thrombolytics as part of an intervention.

GLOBAL – ISCHEMIC OR HYPOTENSIVE STROKE:

Any cause of profoundly low systemic blood pressure results in a hypotensive stroke. The number of viable neurons is 1. The second layer of the hippocampus's pyramidal cells 3. The cerebellar cortex's Purkinje cell layer Cerebral Dim matter

It is the more plentiful glutama te in th ese ne urons which makes them more helpless against worldwide is chemia. The water shed areas and the bou 0ndary zone created by the territo ries of the cere bellar and cere bral arteries also suffer the most damage. The pa rieto-tem poro-occi pital tri angle, which is formed at

the junction of the an terior, post erior, and mid dle cereb ral art eries, is the area that is most frequently affected.

A clinical syndrome is caused by a water shed infarction in this area, which results in sensory loss and arm paralysis, but speech and face remain unaffected. Almost 10 % of the multitude of ischemic strokes are water shed infarcts and 40% of the watershed infarct happens because of carotid impediment or stenosis.

STROKE SYNDROMES

The clinical picture produced by an occlusion of any one artery varies from patient to patient in some ways. However, there is sufficient uniformity to support the designation of a specific syndrome for each major cerebral artery and its branch. The clinical neurologist's most important skill is identifying specific neurovascular syndromes through careful examination. The clinical effects of infarction caused by embolism and thrombosis are particularly relevant for the purposes of the following descriptions.

CAROTID ARTERY NEOVASCULAR SYNDROME:

There are thr ee m ajor ar teries in the carotid system:

The internal carotid artery, the external carotid artery, and the common carotid artery. Occlusion of the common carotid artery occurs in less than 1% of cases. Disease of the internal carotid artery accounts for the remaining cases. However, an atheromatous plaque at the origin, typically on the left side, can obstruct the common carotid. After radiation therapy for thyr oid, lar yngeal, or other head and neck c ancer, atherosclerotic stenosis of the middle part of the common carotid also occurs.

Due to the retrog rade fl ow fro m the ex ternal car otid, which maint ains the flow of the internal carotid artery and, as a result, the brain's perfusion, if the bifurcation is patent, few symptoms occur.

The inter nal caro tid ar tery is connected to the vessels of the orbit and the circle of Willis in most people, and no part of the brain is completely dependent on it. As a result, the occlusion, which typically occurs in the first section of the internal carotid artery just beyond the carotid bifurcation, will typically go unnoticed in between 30% and 40% of cases.

Occlusion of the other internal carotid artery may result in bilateral cerebral infarction if one internal carotid artery was previously blocked. In such instances, the clinical effects include a coma accompanied by quadriplegia and continuous horizontal "metronomic" conjugate eye movements.

When the circulation of one internal carotid artery is partially impaired, decreasing blood flow in the middle and anterior cerebral arterial territories on the ipsilateral side, the zone of maximum ischemia is located between the two vascular territories (the "cortical watershed") or in the deeper regions of the cerebral hemisphere between the territories of the penetrating arteries from the convexity and the lenticostriate branches (the "interior" or "deep watershed").

The first infarction occurs in a region of the subcortical white matter and the hi gh fro ntal and par ietal co rtex. The degree to which the collateral circulation is sufficient determines the size of the infarct. The shoulder and hip are clinically found to be weaker than the face and hand. If the caro tid ste nosis has been present for a long time, the cor tical wat ershed area shifts toward the perisylvian regions of the mid dle ce rebral terri tory. As a result, a stroke may result in nonaffluent aphasia or weakness in facial movement. Impaired perfusion of the

deep watershed area is caused by infarctions of varying sizes in the subparietal and subfrontal regions of the centrum semiovale.

ANTERIOR CEREBRAL ARTERY STROKE SYNDROME:

Through the cortical branches, the anterior cerebral artery provides, 1. The frontal lobe's medial surface in its anterior three-quarters, The frontal axis, number 3. The frontal lobe's medial orbital surface, 4. The superior border, a strip of the cerebral hemisphere on its lateral surface, and 5. four-fifths of the corpus callosum anteriorly.

The anterior limb of the internal capsule is supplied by extensive branches that emerge close to the circle of Willis, 1. The portion of the globus pallidus [2] that is anterior Occlusion of the anterior cerebral artery stem proximal to the connection of the anterior communicating artery is tolerated well due to adequate collateral flow provided by the opposite side anterior cerebral artery. The inferior part of the head of the caudate nucleus

Occlusion proximal to the anterior communicating artery results in a sensorimotor deficit on the opposite side of the foot and leg, as well as on the arm and shoulder, with the face and hand remaining unaffected.

There will be a grasp reflex on the opposite side, urinary incontinence, and paratonic rigidity in the opposite limbs. With left-sided occlusion, there may be "sympathetic apraxia" of the left arm and leg. Language aggravations, as transcortical engine aphasia might happen.

MIDDLE CEREBRAL ARTERY STROKE SYNDROMES:

Through its superficial and deep hemisphere branches, the middle cerebral artery (MCA) 0supplies the majority of the cerebral hemisphere. The cortical branches that encompass supply the lateral (convexity) portion of the cerebral hemisphere.

(1) the cortex and white matter of the substandard and sidelong pieces of the cerebrum — which incorporates engine regions 4 and 6, the engine discourse area of Broca and the discussions places for horizontal look.

(2) the primary and secondary sensory cortices, as well as the white matter and cortex of the parietal lobe, which includes angular and supramarginal gyri.

(3) the superior insula and temporal lobe, including Wernicke's receptive language area.

A significant portion of the head and body of the caudate nucleus, the putamen, the posterior limb of the internal capsule, the outer glob us pa llidus, and the co rona ra diate are supplied by the M CA's lenticulostriate branches.

MCA STEM OCCLUSION:

It's possible that the MCA is blocked at the stem, which is close to the bifurcation. The superficial cortical branches and the small deep penetrating vessel are

prevented from receiving blood flow because of this location's occlusion. However, if the occlusion occurs at the distal end of the MCA stem but does not affect the deep penetrating vessels, it will affect the orifices of the divisions of the artery at the sylvian sulcus.

Contral ateral hem iplegia, homo nymous hem ianopia, and hemianesthesia with the he ad and ey es deviating to the side of the lesion make up the clinical picture. In addition, lesions on the right side cause anosognosia and amorphousness, while lesions on the left side cause global aphasia.

FIRST DIVISION:

An infarction in the superior division results in ipsilateral deviation of the head and eyes as well as mo tor wea kness and sensory loss in the opposite arm, face, and sometimes the leg. It is similar to the syndrome of MCA stem occlusion, but the leg and foot are not completely affected, and if they are, the weakness is less severe than in the arm and face (brachiofacial or chierobrachial paralysis). However, the alertness is maintained.

There will be very slow improvement if the occlusion is persistent. The patient will be able to walk with a spastic leg after a few months because the arm and face continue to have motor deficits. The motor deficit will be more severe than the sensory deficit, which will be profound and resemble a thalamic infarct

and manifest as stereoan esthesia, imp aired tac tile localiza tion, impaired position sense, agrap hesthesia, impai red two-point discr imination, as well as variable changes in pain, touch, and temperature sense.

Initial left-sided lesions cause a global aphasia that evolves into a predominantly nonaffluent aphasia, also known as Broca's aphasia.

DIVISION INFERIOR:

The majority of the time, embolism is the cause of occlusion of the MCA's inferior division, which occurs less frequently than the superior division. Wernicke's aphasia is the clinical sign of left-sided lesions, and it stays the same for days or weeks. After that, some improvement is to be expected.

The ability to comprehend written and spoken language is severely impaired in selective distal branch occlusions (superior parietal , posterior temporal , and angular). The deficits decrease after a few months.

Homonymous hemianopia or superior quadrantanopia is present in left-hemispheric lesions, while amorphosynthesis and left visual neglect are present in right-hemispheric lesions. A agitated confusional state caused by damage to the

temporal lobe may occasionally be a symptom of dominant hemisphere and even nondominant lesions.

POSTERIOR CEREBRAL ARTERY STROKE SYNDROME:

The basilar artery bifurcation forms both the posterior cerebral arteries and the thin poste rior comm unicating arteries that connect the above system to the internal carotid arteries in about 70% of people. One posterior cerebral artery originates from the basilar in approximately 20% to 25% of people, while the other originates from the internal carotid artery, a pattern of persistent foetal circulation. However, the pattern in which both the posterior cerebral artery and the corresponding carotid artery arise from each other is unusual in less than 5% of people.

Occlusion of the posterior cerebral artery has the greatest variety of clinical effects because it supplies the upper brainstem, which contains the majority of important structures, as well as the occipital lobe and the inferomedial parts of the temporal lobes. The size and location of the resulting infarct will be significantly influenced by the occlusion site and the circle of Willis arrangement. For instance, an occlusion that is proximal to the posterior communicating artery may not cause symptoms at all, or it may only cause very brief clinical effects because the collateral flow from the opposite posterior cerebral vessel is sufficient. Additionally, if sufficient border zone collaterals from the ante rior and

mid dle cere bral arteries are present, an occlusion that is distal to the posterior commu nicating art ery may cause less da mage.

CORTISOL AND STROKE:

The hypothalamo-pituitary-adrenal axis (HPA) is early and heavily activated during the hyperacute phase of stroke. The typical response pattern is biphasic. At first, both cortisol and ACTH are elevated simultaneously. Cortisol levels continue to rise despite the rapid drop in ACTH levels in the second phase.

The preceding pattern is explained by the rapid decrease in ACTH levels induced by cortisol following the initial activation of the HPA axis. Additionally, the adrenal gland's increased vulnerability helps to maintain the elevated levels of cortisol. Surprisingly, the adrenal gland is also hyperresponsive to ACTH during the early recovery stage of the postoperative condition. When tissue is injured, the cytokines that cause inflammation have activity similar to ACTH or corticotrophin releasing hormone (CRH). This explains why there is a significant response from the adrenal glands even when there is no corresponding increase in ACTH and how much brain damage there is. Numerous in vitro and in vivo studies have established that HPA hormones can be neurotoxic. It states unequivocally that cortisol hurts the mind by

1. increasing the damage that hypoxia does to neurons and astrocytes.

2. causing disruption to the brain's glucose metabolism and absorption.

One of the reasons higher cortisol levels are linked to cognitive dysfunction is that hypercortisolism can exacerbate ischemic damage to the neurons in the hippocampus.

In addition, it has been discovered that people with acute ischemic stroke frequently experience repeated pressures such as infec tions, emo tional rea ctions, and cardi ovascular issues. The adrenal glands can become more sensitive to ACTH if stressors are repeated. As a result, hypercortisolism might continue.

CYTOKINES IN CORTISOL AFTER STROKE:

After the brain infarction, a cascade of proinflammatory cytokines 9, 10 is released. After ischemia, the cascade that activates interleukin 1 and 6 is initiated by TNF alpha. The effect is also influenced by activation of the sympathetic nervous system. In acute ischemic stroke, interleukin-6 can be regulated at multiple levels. Additionally, the hypothalamus-pituitary-adrenal axis can be activated by any psychological or physical stressor, resulting in an increase in interleukin-6

levels. After the occasion of intense stroke numerous provocative cytokines are set free from the fringe platelets. Additionally, abnormal leptin11 levels are associated with a flattening of diurnal variations in stroke patients. Leptin is linked to neuroendocrine balance, which also includes the regulation of the cortisol axis, according to additional evidence. Initi al IL-6 l evels and abnormal cortisol diurnal rhythm icity can predict stroke outcome, according to some studies.

MATERIALS AND METHODS

STUDY GROUP:

All 70 new cases of acute ischemic stroke admitted in Shri.B.M.Patil medical college and hospital within 72 h ours of acu te neuro logical event satisfying the inclusion and exclusion criteria .

TYPE OF STUDY:

Prospective Cross- Sectional Study

SAMPLE SIZE OF STUDY: 70

DURATION OF STUDY :2020 to 2023

BACKGROUND

Inclusion standards:

1. Patients older than 18 years of age

2. Patients admitted to ICU/Wards who have been diagnosed with an acute ischemic stroke (via MRI and CT Brain Plain)

Exclusion standards:

1. 18 years of age

2.Pregna ncy

3. Li ver illness

4. Patients who are taking immunosuppressive medications, steroids, rifampicin, or phenytoin.

5. Having no cancer

7. Stroke with hemorrhage

8. Severe febrile illness

9. Major Surgery in the Next Three Weeks

METHODOLOGY: Each patient's data was gathered using a specific proforma. which comprised,

1.The pati ent's n ame, a ge, sexual orientation, de mographic information, current comp laints, ri sk fa ctors, family history, and drug usage

2. General assessment

3.Vital signs .

4.System examination

5.Using the Nation al Ins titute of Hea lth Scie nces Scale (NI0HSS) to assess severity at the tim e of adm ission

6.Se rum C ortisol levels were measured the following day (0, 3, and 5).

7. MRI and CT Brain Plain

BIOCHEMICAL ANALYSIS Serum cortisol levels were measured from blood samples taken the morning after admission the following day. Enzyme Immuno Assay is used to quantitatively measure serum cortisol.

CONCEPT OF THE TEST: The competition between the enzyme-labeled antigen and the unlabeled antigen for a limited antibody binding site on the microwell plates is the test's underlying principle. Washing and decanting are used to get rid of the unbound materials. Add the enzyme substrate. The enzymatic reaction is stopped by adding stop solution. The absorbance is measured by the microtiter plate reader. Cortisol concentration in the sample has an inverse

relationship with the intensity of the formed color. A standard curve is plotted using a set of standards. which directly reads the amount of cortisol.

TEST : 50 ml of venous0 blood is gathered into the gathering tube and permitted it to clump and centrifuged and the serum layer is taken out. The horseradish peroxidase conjugate and wash buffer are prepared as working solutions . In a microwell plate coated with polyclonal antibody, the required number of microwell strips are removed. 100 microliters of conjugate working solution is pipetted into each well, and 20 microliters of each calibrator , control , and specimen sample are pipetted into the corresponding labelled wells in duplicate. After that, it is incubated for 45 minutes at room temperature in a plate shaker with about 200 rpm. After that, the wells are dried and washed with diluted wash buffer. At regular intervals, 150 microliters of tetra me thyl be nzidine subs trate are pipetted into each well. After that, it is incubated once more on a plate shaker at room temperature for twenty minutes. At predetermined intervals, 50 microliters of stop solution are pipetted into the well. A micro well plate reader at 450 nm can read the plate within 20 minutes of adding the stop solution. The mean optical density and the calibrator curve are used to calculate the results, which are then multiplied by a dilution factor.

STATISTICAL ANALYSIS OF DATA:

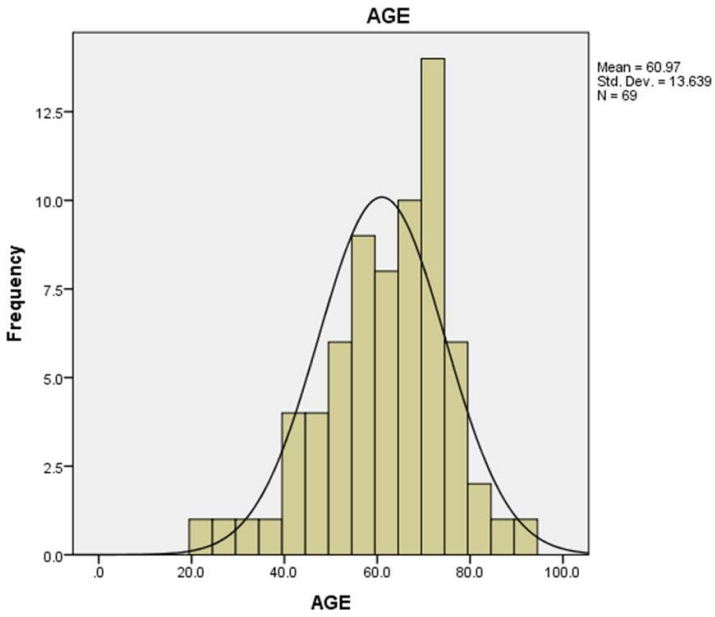
Number of patients and percentage of patients are used to express categorical variables. The Mann-Whitney U test or Kruskal Wallis test is used as necessary to compare continuous variables across groups. Continuous variables are expressed as Mean, Median, and Standard Deviation. Wilcoxon Signed Ranks Test was used to compare two data sets over time. The analysis was conducted using SPSS version 22 statistical software. A 5% alpha level was used, which means that any p value less than 0.05 was deemed significant.

TABLE 1 AGE WISE DISTRIBUTION

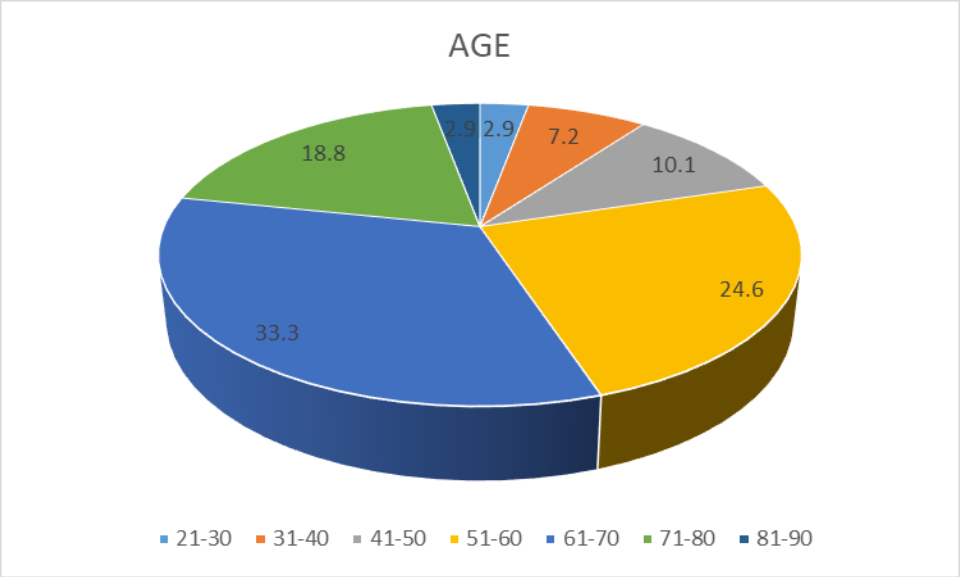
AGE	Frequency	Percent
21-30	3	2.9
31-40	5	7.2
41-50	7	10.1
51-60	17	24.6
61-70	23	33.3
71-80	13	18.8
81-90	2	2.9
Total	70	100.0

	AGE
Minimum	22.00
Maximum	90.00
Mean	60.97
Median	62.00
Std. Deviation	13.64

GRAPH 1



Among the 70 patients in the study group minimum age is 2 years and maximum age group is 90 yrs. Age group from 21 to 30 accounts for 2%, 31 to 40 yrs. is 5%, 41 to 50yrs is 7%, 51 to 60 yrs. is 17%, 61 to 70 yrs. is 23%, 71 to 80 yrs. is 13%, 81 to 90 yrs. is 2.9%

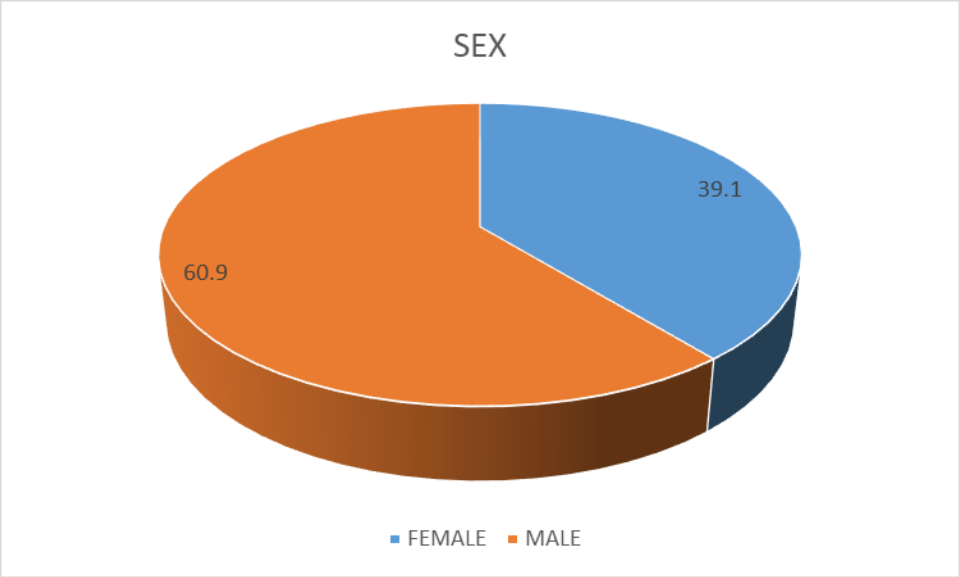


GRAPH .2

SEX WISE DISTRIBUTION OF CASES

TABLE 2

SEX	Frequency	Percent
FEMALE	27	39.1
MALE	43	60.9
Total	70	100.0



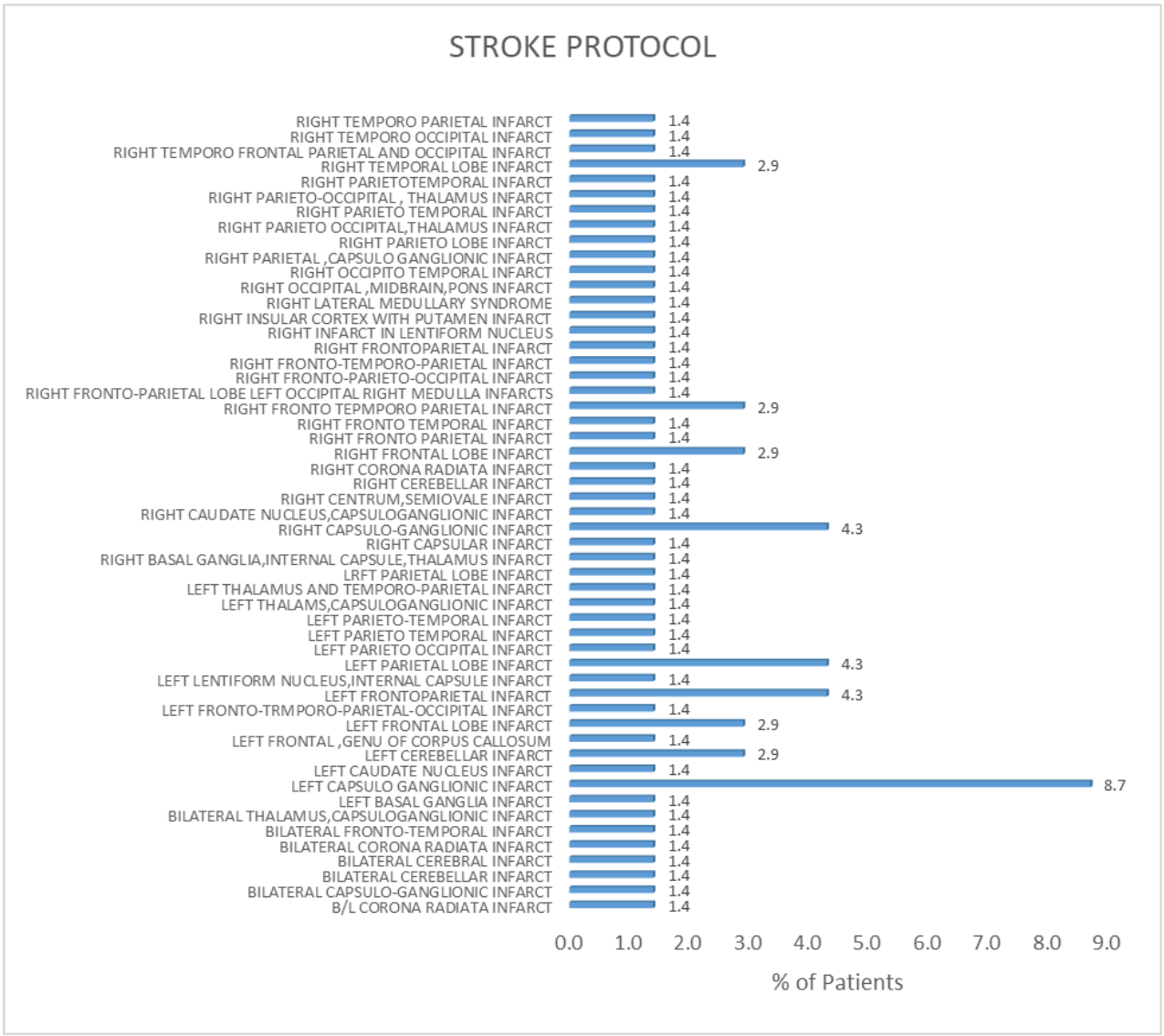
Total number of cases were 70 out of which 60.9 percent were males and 39.1 percent were female

DISTRIBUTION OF PATIENTS BASED ON LOCATION OF INFARCTS ON STROKE PROTOCOL

TABLE 3

STROKE PROTOCOL	Frequency	Percent
B/L CORONA RADIATA INFARCT	1	1.4
BILATERAL CAPSULO-GANGLIONIC INFARCT	1	1.4
BILATERAL CEREBELLAR INFARCT	1	1.4
BILATERAL CEREBRAL INFARCT	1	1.4
BILATERAL CORONA RADIATA INFARCT	1	1.4
BILATERAL FRONTO-TEMPORAL INFARCT	1	1.4
BILATERAL THALAMUS, CAPSULOGANGLIONIC INFARCT	1	1.4
LEFT BASAL GANGLIA INFARCT	1	1.4
LEFT CAPSULO GANGLIONIC INFARCT	6	8.7
LEFT CAUDATE NUCLEUS INFARCT	1	1.4
LEFT CEREBELLAR INFARCT	2	2.9
LEFT FRONTAL, GENU OF CORPUS CALLOSUM	1	1.4
LEFT FRONTAL LOBE INFARCT	2	2.9
LEFT FRONTO-TRMPORO-PARIETAL-OCCIPITAL INFARCT	1	1.4
LEFT FRONTOPARIETAL INFARCT	3	4.3
LEFT LENTIFORM NUCLEUS, INTERNAL CAPSULE INFARCT	1	1.4
LEFT PARIETAL LOBE INFARCT	3	4.3
LEFT PARIETO OCCIPITAL INFARCT	1	1.4
LEFT PARIETO TEMPORAL INFARCT	1	1.4
LEFT PARIETO-TEMPORAL INFARCT	1	1.4
LEFT THALAMS, CAPSULOGANGLIONIC INFARCT	1	1.4
LEFT THALAMUS AND TEMPORO-PARIETAL INFARCT	1	1.4
LRFT PARIETAL LOBE INFARCT	1	1.4
RIGHT BASAL GANGLIA, INTERNAL CAPSULE, THALAMUS INFARCT	1	1.4

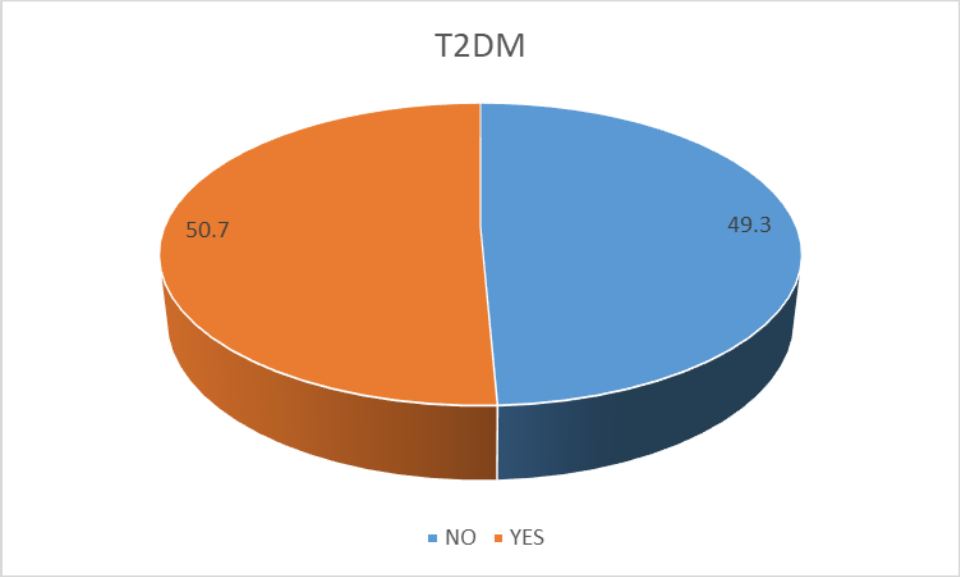
RIGHT CAPSULAR INFARCT	1	1.4
RIGHT CAPSULO-GANGLIONIC INFARCT	3	4.3
RIGHT CAUDATE NUCLEUS, CAPSULOGANGLIONIC INFARCT	1	1.4
RIGHT CENTRUM, SEMIOVALE INFARCT	1	1.4
RIGHT CEREBELLAR INFARCT	1	1.4
RIGHT CORONA RADIATA INFARCT	1	1.4
RIGHT FRONTAL LOBE INFARCT	2	2.9
RIGHT FRONTO PARIETAL INFARCT	1	1.4
RIGHT FRONTO TEMPORAL INFARCT	1	1.4
RIGHT FRONTO TEPMPORO PARIETAL INFARCT	2	2.9
RIGHT FRONTO-PARIETAL LOBE LEFT OCCIPITAL RIGHT MEDULLA INFARCTS	1	1.4
RIGHT FRONTO-PARIETO-OCCIPITAL INFARCT	1	1.4
RIGHT FRONTO-TEMPORO-PARIETAL INFARCT	1	1.4
RIGHT FRONTOPARIETAL INFARCT	1	1.4
RIGHT INFARCT IN LENTIFORM NUCLEUS	1	1.4
RIGHT INSULAR CORTEX WITH PUTAMEN INFARCT	1	1.4
RIGHT LATERAL MEDULLARY SYNDROME	1	1.4
RIGHT OCCIPITAL, MIDBRAIN, PONS INFARCT	1	1.4
RIGHT OCCIPITO TEMPORAL INFARCT	1	1.4
RIGHT PARIETAL, CAPSULO GANGLIONIC INFARCT	1	1.4
RIGHT PARIETO LOBE INFARCT	1	1.4
RIGHT PARIETO OCCIPITAL, THALAMUS INFARCT	1	1.4
RIGHT PARIETO TEMPORAL INFARCT	1	1.4
RIGHT PARIETO-OCCIPITAL, THALAMUS INFARCT	1	1.4
RIGHT PARIETOTEMPORAL INFARCT	1	1.4
RIGHT TEMPORAL LOBE INFARCT	2	2.9
RIGHT TEMPORO FRONTAL PARIETAL AND OCCIPITAL INFARCT	1	1.4
RIGHT TEMPORO OCCIPITAL INFARCT	1	1.4
RIGHT TEMPORO PARIETAL INFARCT	1	1.4
Total	70	100.0



NUMBER OF DIABETIC AND NON-DIABETIC PATIENTS

TABLE 4

T2DM	Frequency	Percent
NO	34	49.3
YES	36	50.7
Total	70	100.0

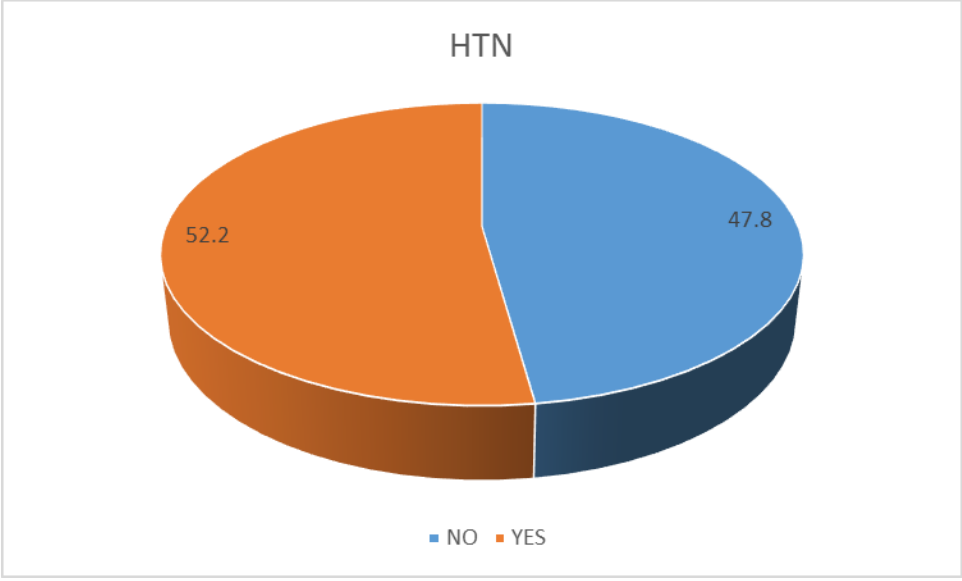


Among the 70 patients 36 patients were diabetic 50.7 % and 34 patients were non diabetic 49.3%

DISTRIBUTION OF PATIENTS BASED ON HYPERTENSION

TABLE 5

HTN	Frequency	Percent
NO	34	47.8
YES	36	52.2
Total	70	100.0

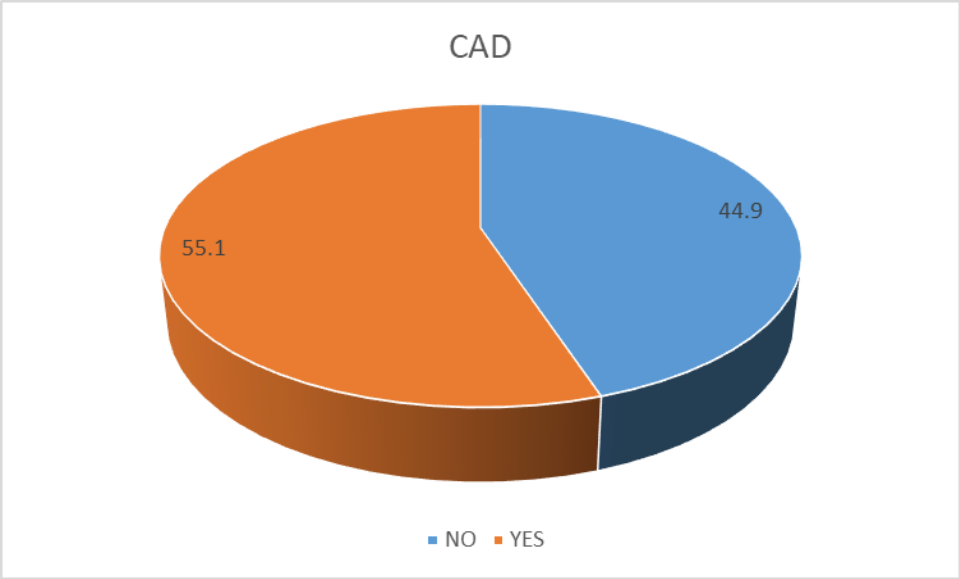


Among the 70 patients 52.2 % were hypertensive and 47.8 % were non hypertensive

DISTRIBUTION OF PATIENTS BASED ON CORONARY ARTERY DISEASE

TABLE 6

CAD	Frequency	Percent
NO	31	44.9
YES	38	55.1
Total	70	100.0



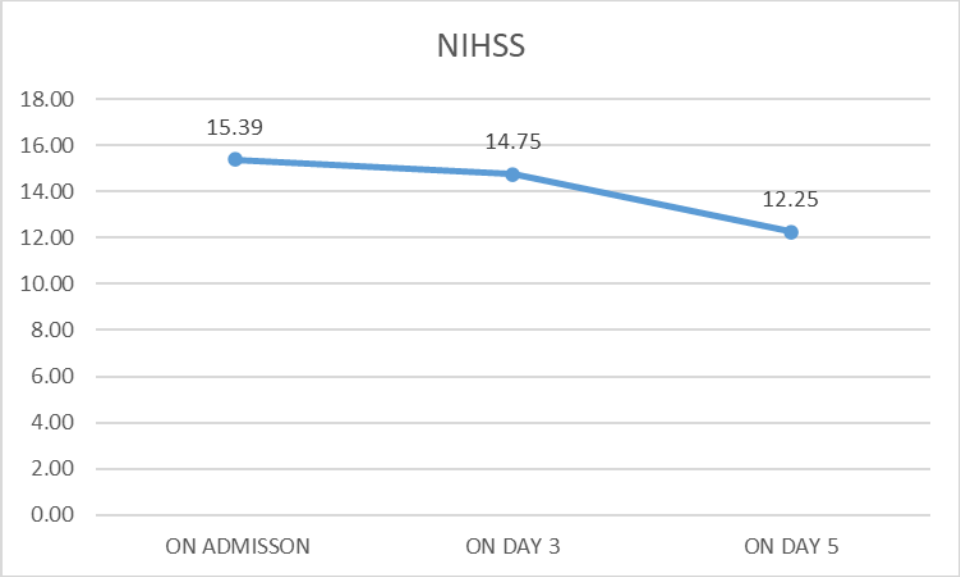
Among the 70 patients 55.1 % were having coronary artery disease and 44.9% were not having coronary artery disease

DISTRIBUTION OF PATIENTS BASED ON NIHSS SCORE

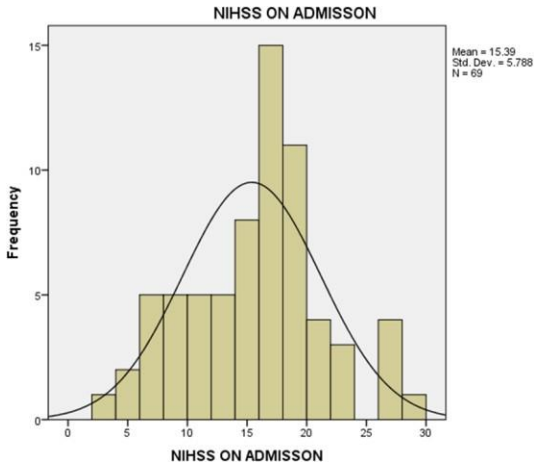
TABLE 7

	NIHSS ON ADMISSON	NIHSS ON DAY 3	NIHSS ON DAY 5
Minimum	3.00	3.00	3.00
Maximum	28.00	29.00	26.00
Mean	15.39	14.75	12.25
Median	16.00	15.00	12.00

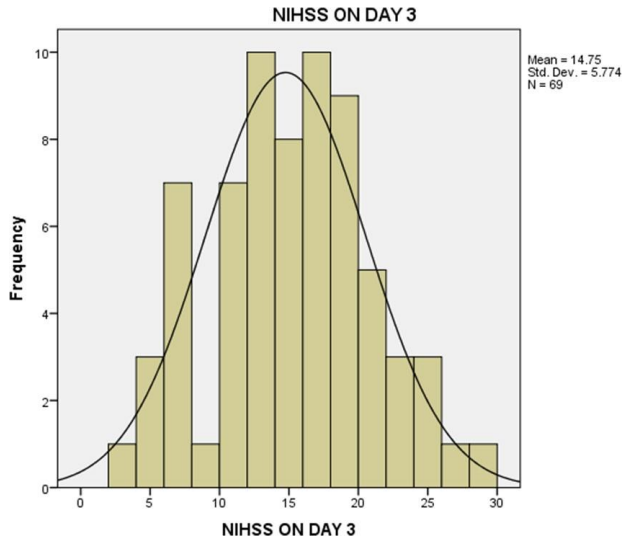
Std. Deviation	5.79	5.77	5.93



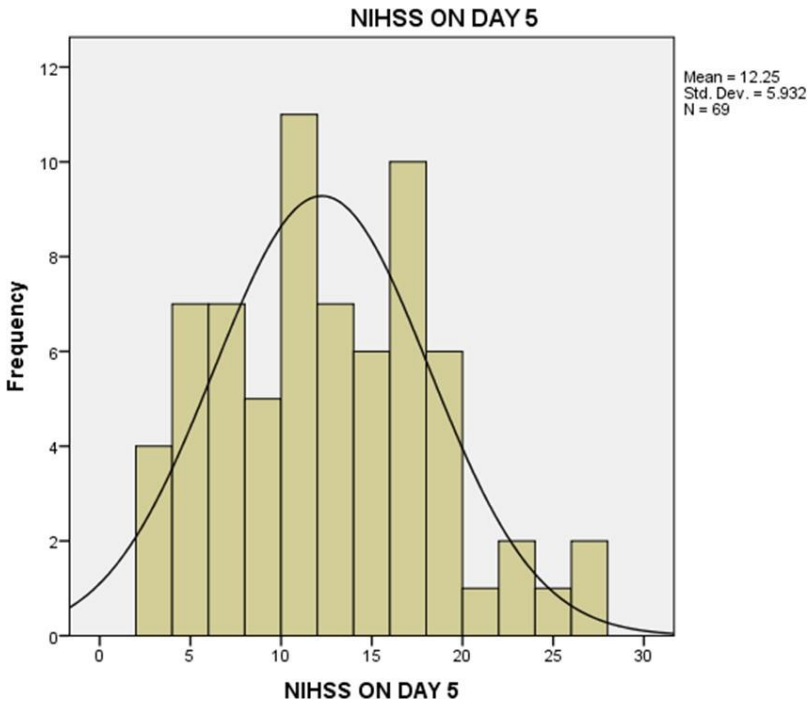
	NIHSS ON DAY 3 - NIHSS ON ADMISSON	NIHSS ON DAY 5 - NIHSS ON ADMISSON	NIHSS ON DAY 5 - NIHSS ON DAY 3
p Value	0.018	<0.001	<0.001



This graph depicts the NIHSS score of patients on day of admission



This graph depicts the NIHSS score of the patients on day 3 of admission

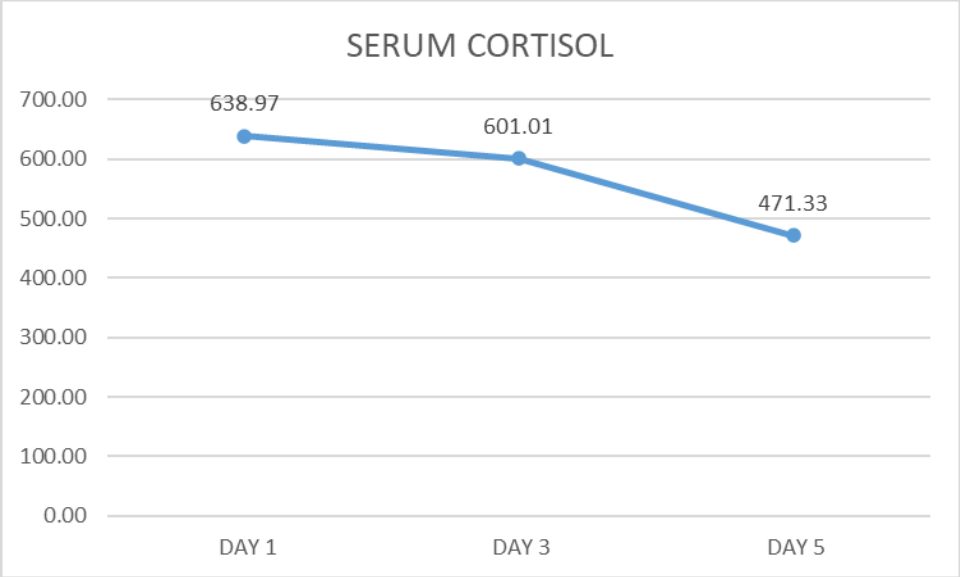


This graph depicts the NIHSS score on day 5 of admission

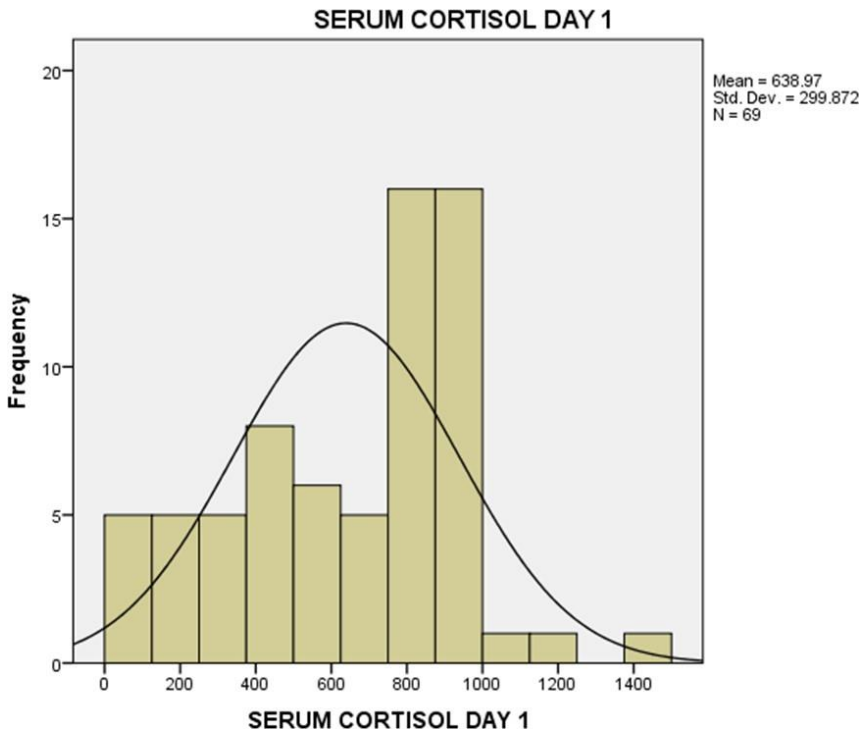
DISTRIBUTION OF PATIENTS BASED ON SERUM CORTISOL LEVELS

TABLE 8

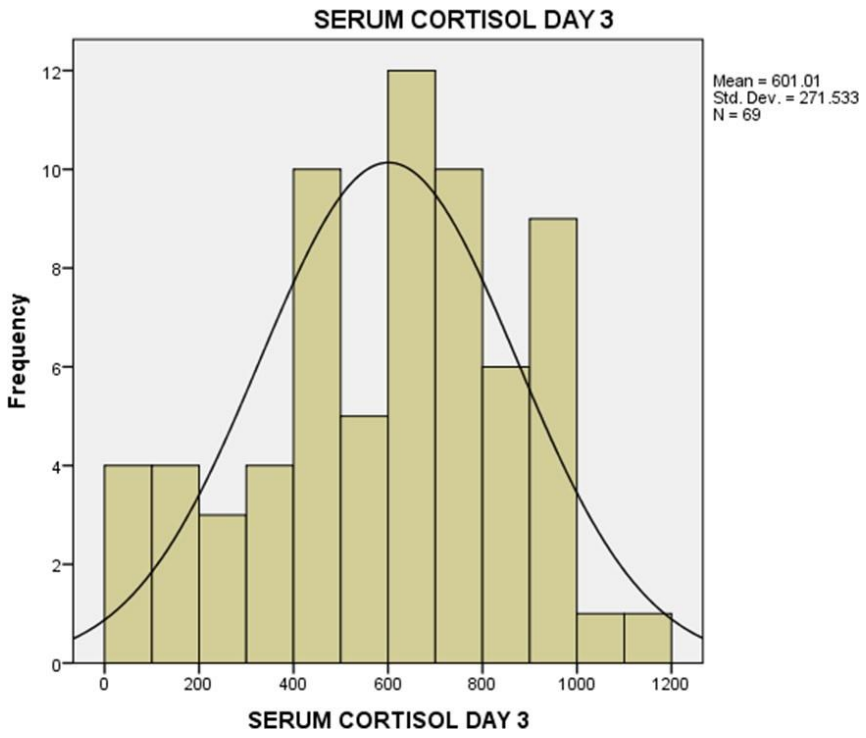
	SERUM CORTISOL DAY 1	SERUM CORTISOL DAY 3	SERUM CORTISOL DAY 5
Minimum	46.00	52.00	28.00
Maximum	1452.00	1135.00	947.00
Mean	638.97	601.01	471.33
Median	757.00	662.00	488.00
Std. Deviation	299.87	271.53	254.28



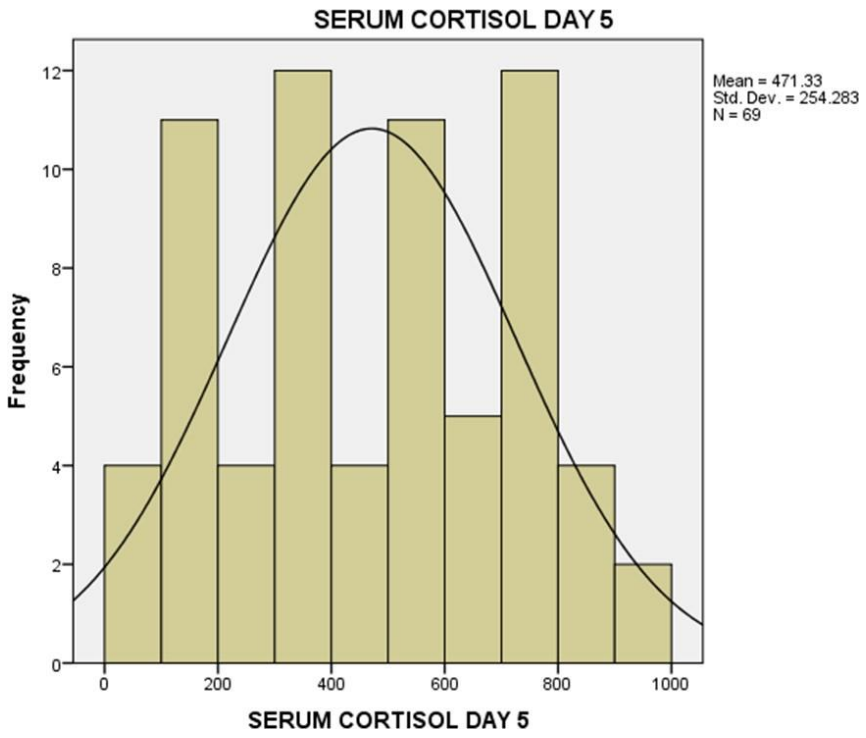
	SERUM CORTISOL DAY 3 - SERUM CORTISOL DAY 1	SERUM CORTISOL DAY 5 - SERUM CORTISOL DAY 1	SERUM CORTISOL DAY 5 - SERUM CORTISOL DAY 3
p Value	0.155	<0.001	<0.001



The mean cortisol levels on day of admission were 638.97



The mean cortisol levels on day 3 of admission was 601.01

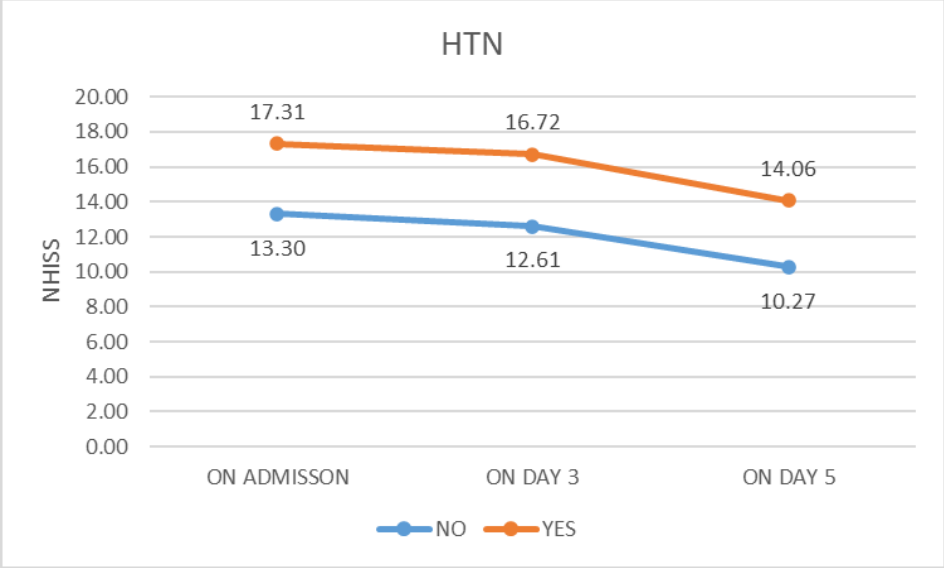


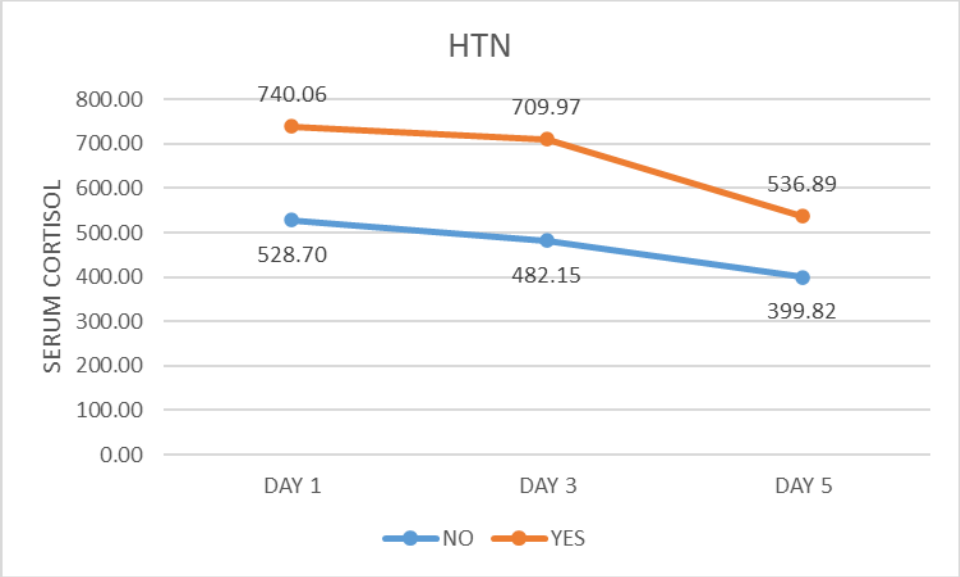
Mean cortisol levels on day 5 of admission was 471.33

CORRELATION BETWEEN HYPERTENSION AND CORTISOL

TABLE 10

HTN		NIHSS ON ADMISSON	NIHSS ON DAY 3	NIHSS ON DAY 5	SERUM CORTISOL DAY 1	SERUM CORTISOL DAY 3	SERUM CORTISOL DAY 5
NO	Mean	13.30	12.61	10.27	528.70	482.15	399.82
	Median	15.00	12.00	9.00	584.00	456.00	391.00
	Std. Deviation	5.98	6.04	5.99	310.14	286.83	256.62
YES	Mean	17.31	16.72	14.06	740.06	709.97	536.89
	Median	17.00	17.00	13.50	783.00	717.50	548.00
	Std. Deviation	4.95	4.80	5.34	254.54	206.14	237.01
	p Value	0.012	0.002	0.008	0.009	0.001	0.036
	Significance	Significant	Significant	Significant	Significant	Significant	Significant





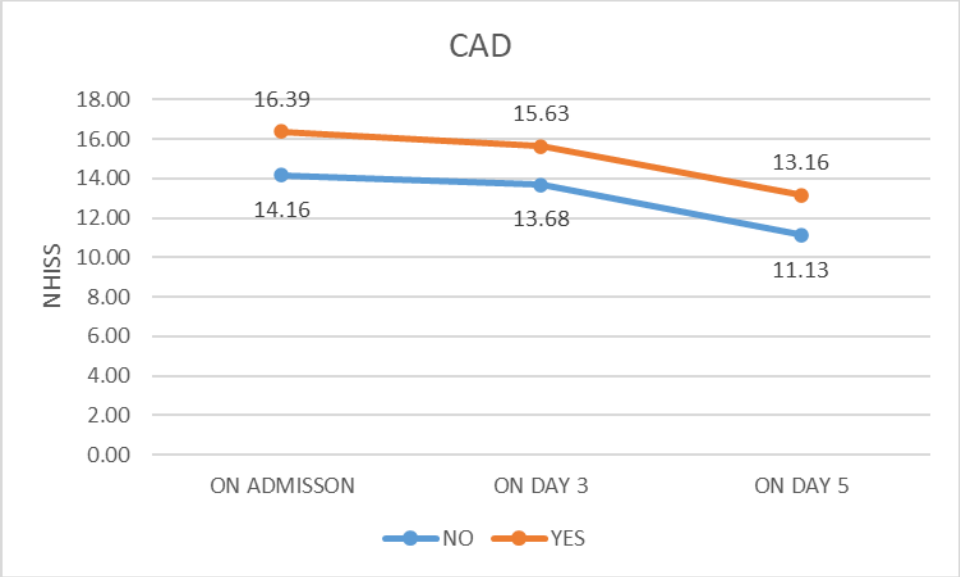
PATIENTS WITH HYPERTENSION HAVE HIGHER LEVELS OF CORTISOL LEVELS AND HIGHER RISK OF STROKE

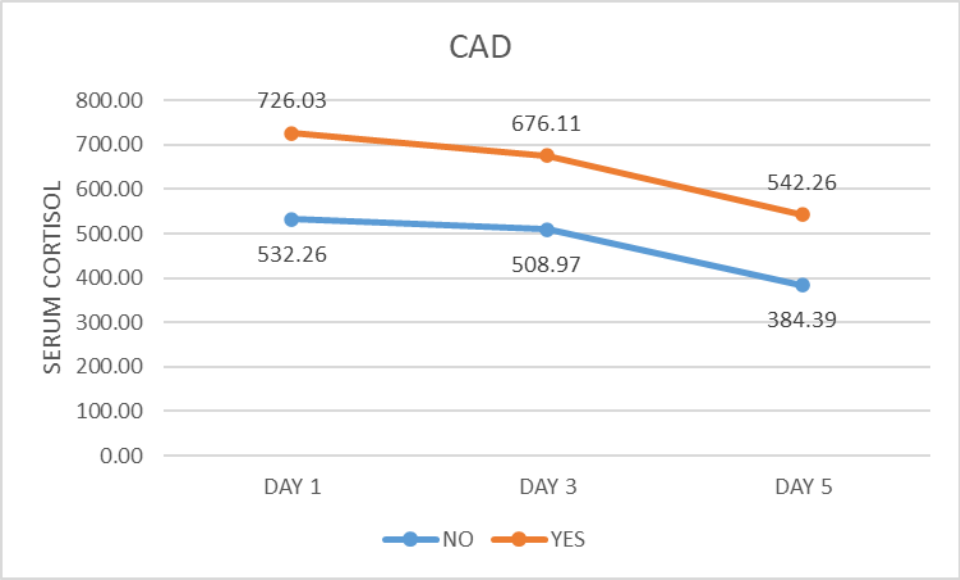
CORRELATION BETWEEN CORONARY ARTERY DISEASE AND CORTISOL LEVELS

TABLE 11

CAD	Frequency	Percent
NO	32	44.9
YES	38	55.1
Total	70	100.0

CAD		NIHSS ON ADMISSON	NIHSS ON DAY 3	NIHSS ON DAY 5	SERUM CORTISOL DAY 1	SERUM CORTISOL DAY 3	SERUM CORTISOL DAY 5
NO	Mean	14.16	13.68	11.13	532.26	508.97	384.39
	Median	15.00	13.00	10.00	586.00	550.00	361.00
	Std. Deviation	5.96	6.18	5.99	284.83	274.98	236.44
YES	Mean	16.39	15.63	13.16	726.03	676.11	542.26
	Median	16.00	15.50	12.00	813.00	709.50	568.00
	Std. Deviation	5.52	5.34	5.81	286.63	247.60	249.01
	p Value	0.189	0.241	0.211	0.005	0.013	0.009
	Significance	Not Significant	Not Significant	Not Significant	Significant	Significant	Significant





PATIENTS WITH CORONARY ARTERY DISEASE HAD HIGHER CORTISOL LEVELS WHICH ADDS AS A RISK FACTOR FOR DEVELOPING STROKE AND PROGNOSIS

DISCUSSION

In this study of total 70 patients were enrolled having Acute Ischemic Stroke by CT Brain which was taken at the time of admission . The min imum age was 22 yrs. and maximum age being 90 yrs. . Male patients accounted for 60.9% and females accounted for 30.1%

Of the 70 patients 50.7% of patients were diabetic and 49.3 % were non diabetic. In diabetic patients the mean cortisol levels were 675.77 and in non-diabetic patients the mean cortisol levels were 601.59.

52.2% patients were hypertensive and 47.8 % were non hypertensive the mean levels of cortisol were 740.06 and in non-hypertensive 528.70.

55.1% patients were known case of coronary disease and 44.9% were not having coronary artery disease. The mean cortisol levels were in CAD group was 726.2 and in non-CAD group the cortisol levels were 532.26.

The majority of cases had elevated serum cortisol levels. The NIHSS score was above 6 in 100% of the cases with a serum cortisol level greater than 638.7 nmol/L. This is found to be statistically significant with a p value of 0.001. Given that a NIH SS scor e of 6 or higher is consid ered to be mo derate to se vere stroke, the aforementioned observation makes it abundantly clear th at n early a ll c ases with raised cort isol levels had modera te to se vere stroke.

SIMILAR STUDIES:

According to Marklund N. et al.'s research, high cortisol levels are associated with severe stroke outcomes. Cortisol levels on day one were higher in patients with severe functional impairment than in patients with mild symptoms. Additionally, high levels of serum cortisol on day one significantly predicted mortality after 28 days.

According to a st udy by We n Jie Zi et al., pati ents with high seru m cort isol levels on the day of adm0ission have a poor prognosis (P 0.0001). Our study discovered a statistically significant correlation between ser0um cort0isol levels and the NIHSS sc ore in predi cting the sever ity of the st roke and the function al outco me (P0.001). This correlation was also found in the previous two studies.

As a result, it was abundantly clear that, at the ti me of admissi on, patients with high s erum c ortisol levels had extremely severe acute ischemic stroke, and that patients with high serum cortisol levels also had poor outcomes.

CONCLUSION:

High levels of serum cortisol at the time of admission correlate with, among patients with acute ischemic stroke: Prognosis and clinical severity, as measured by the National Institutes of Health Stroke Scale

CLINICAL SIGNIFICANCE:

The adre nal str ess resp onse increases blood glucose levels, cataboli sm, heart rate, and the likelihood of ischemic neuronal damage in humans. These effects could result in secondary brain damage in acute ischemic stroke. Nerve center Pituitary-adrenal pivot adjustments are one of the significant pressure actuated modifications after the occasion of cerebral ischemia. Even after adjusting for confounding factors, cortisol remains an independent short-term indicator of functional outcome and death in patients with acute ischemic stroke. After a stroke, elevated cortisol levels are clearly linked to morbidity, dependency, and death.

Because ea rly pre diction of stro ke out come is very import ant for the alloca tion of therapeu tic strateg ies, seru m co rtisol level mea surement at the time of admis sion can be sign ificant predicti ve information to the existing NIH SS score. However, the clinical score can be significantly enhanced by a combined model.

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SCHEME OF CASE TAKING

B.L.D.E. U'S SHRI B. M. PATIL MEDICAL COLLEGE HOSPITAL AND RESEARCH CENTRE, VIJAYAPURA.

DEPARTMENT OF MEDICINE

PROFORMA

Name IP number
Age: Sex

Address: Occupation:

Date of Admission: Date of discharge:

Chief Complaints:

History of present illness:

Past history:

Treatment History:

Personal History:

Physical Examination:

On Examination:

VITALS:
Temperature:
Pulse:
Respiratory rate:
Blood pressure:

GENERAL CONDITION:

Pallor:	Yes/ No
Icterus:	Yes/ No
Cyanosis:	Yes/No
Clubbing:	Yes/No
Lymphadenopathy:	Yes/No
Edema:	Yes/No
Pt condition on discharge	Improved/Worsened/same/Expired

SYSTEMIC EXAMINATION:

EXAMINATION OF THE CNS:

1.Higher Mental Functions:

2.Cranial Nerves:

3.Motor System:

Tone-

Reflexes-

Coordination-

Gait-

4.Sensory System:

5.Cerebellar signs:

6.Meningeal signs:

7.complete hemogram

8.Liver Function Test

9.Renal Function Test

10. Fasting and Post Prandial Blood Sugars

	Day 0	Day 3	Day 5
Serum Cortisol levels			
NIHSS			

STROKE PROTOCOL-

NIHSS SCORING: -

1a. Level of Consciousness

0: Alert

1: Not alert, but arousable with minimal stimulation

2: Not alert, requires repeated stimulation to attend

3: Coma

1.b. LOC questions (Ask patient the month and her/his age)

0: Answers both correctly

1: Answers one correctly

2: Both incorrect

1.c. LOC commands (Ask patient to open/close eyes & form/release fist)

0: Obeys both correctly

1: Obeys one correctly

2: Both incorrect

2. Best gaze (only horizontal eye movement)

0: Normal

1: Partial gaze palsy

2: Total gaze paresis or Forced deviation

3. Visual Field testing

0: No visual field loss

1: Partial hemianopia

2: Complete hemianopia

3: Bilateral hemianopia (blind including cortical blindness)

4. Facial Paresis (Ask patient to show teeth/ raise eyebrows & close eyes tightly)

0: Normal symmetrical movement

1: Minor paralysis (flattened nasolabial fold, asymmetry on smiling)

2: Partial paralysis (total or near total paralysis of lower face)

3: Complete paralysis of one or both sides (absence of facial movement in the upper and lower face)

5. Motor Function – Arm (5a left,

0: Normal (extends arms 90degree (or 45 degree) for 10 seconds)

1: Drift 2: Some effort against gravity

3: No effort against gravity

4: No movement

9: Untestable (Joint fused or limb amputated) (do not add score)

5b right)

6. Motor Function - Leg (6a-left 6b-Right)

0: Normal (hold leg in 300 positions for 5 sec without drift)

1: Drift

2: Some effort against gravity

3: No effort against gravity

4: No movement

9: Untestable (Joint fused or limb amputated) (do not add score).

7. Limb Ataxia

0: No ataxia

1: Present in one limb

2: Present in two limbs

8. Sensory (Use pinprick to test arms, legs, trunk and face-compare side to side)

0: Normal

1: Mild to moderate decrease in sensation

2: Severe to total sensory loss

9. Best Language (Ask patient to describe picture, name items, read sentences)

0: No aphasia

1: Mild to moderate aphasia

2: Severe aphasia

3: Mute

10. Dysarthria (Ask patient to read several words) 0: Normal articulation

1: Mild to moderate slurring of words

2: Near unintelligible or unable to speak

9: Intubated or another physical barrier (do not add score)

11. Extinction and inattention (Formerly Neglect) (Use visual or sensory double stimulation)

0: Normal

1: Inattention or extinction to bilateral simultaneous stimulation in one of the sensory modalities

2: Severe hemi-inattention or hemi-inattention to more than one modality

Total Score (0-42):

ABBREVIATION

ACTH - Adrenocorticotrophic Harmone

ASD - Atrial Septal defect

CBG - Cortisol Binding Globulin

CBF - Cerebral Blood Flow

CN - Coagulation Necrosis

CRH - Corticotrophin Releasing hormone

CYP - Cytochrome P 450

DIVC - Disseminated Intravascular Coagulation

DNA - Deoxyribose Nucleic Acid

EEG - Electro Encephalo Gram

HDL - High Density Lipoprotein

HPA - Hypothalamus Pituitary Adrenal Axis

IP - Ischemic Penumbra

ITT - Insulin Tolerance Test

LDL - Low Density Lipoprotein

LSD - Lysergic Acid Derivative

MCA - Middle Cerebral Artery

NMDA - N-methyl-d-Aspartate

NO - Nitric oxide

RHD - Rheumatic Heart Disease

RR - Relative Risk SAH – Haemorrhage

SWD - Spontaneous Wave Depolarisation

TIA - Transient Ischemic Attack

TSH - Thyroid Stimulating Harmone

NAME	AGE	SEX	IP NUMBER	STROKE PROTOCOL	T2DM	HTN	CAD	NIHSS ON ADMISSION	DAY 3	DAY 5	SERUM CORTISOL DAY 1	DAY 3	DAY 5
HUSEN BADAF	70	M	20712	LEFT CAPSULO GANGLIONIC INFARCT	YES	YES	NO	17	14	10	456	550	320
DUNDAWWA	80	F	19807	RIGHT IN FARCT IN LENTIFORM NUCLEUS	YES	YES	NO	20	20	17	654	330	280
LAXMIBAI	58	F	24229	RIGHT LATERAL MEDULLARY SYNDROME	NO	NO	YES	16	12	8	532	687	488
ANAPPA	70	M	142084	LEFT PARIETAL LOBE IN FARCT	NO	NO	NO	8	10	7	212	307	123
CHANDRASHEKAR	65	M	135367	LEFT PARIETO -TEMPORAL IN FARCT	NO	YES	YES	13	13	9	542	654	700
SHIVLINGAYYA	65	M	127365	BILATERAL CAPSULO GANGLIONIC IN FARCT	NO	YES	YES	28	26	26	1057	863	789
SHARNAPPA	65	M	26134	RIGHT FRONTO PARIETO OCCIPITAL INFARCT	YES	YES	NO	21	22	18	885	662	579
RAMESH BADIGER	40	M	37102	LEFT LENTIFORM NUCLEUS,INTERNAL CAPSULE INFARCT	NO	NO	NO	23	23	19	912	906	834
DRAKSHAYANI	67	F	156102	LEFT PARIETO OCCIPITAL INFARCT	YES	NO	YES	16	14	11	295	456	195
SHRIMANTH	70	M	15608	BILATERAL FRONTO -TEMPORAL INFARCT	NO	NO	YES	19	19	17	833	781	788
SHIVANAND	55	M	16501	RIGHT PARIETOTEMPORAL INFARCT	NO	YES	NO	12	12	6	452	488	309
SHAVANTRAWA	65	F	16507	BILATERAL CORONA RADIATA INFARCT	NO	NO	YES	17	19	12	879	931	597
JAIBANDAR	55	F	24625	LEFT THALAMUS AND TEMPORO -PARIETAL IN FARCT	YES	YES	NO	11	13	9	341	472	189
BASAMMA	73	F	22481	LEFT PARIETO TEMPORAL IN FARCT	NO	YES	YES	15	12	10	629	741	311
SHIVAYOGI	70	M	20075	LEFT CAUDATE NUCLEUS INFARCT	YES	NO	YES	16	12	6	349	286	162
RAMURATHOD	55	M	25337	RIGHT CAUDATE NUCLEUS,CAPSULOGANGLIONIC INFARCT	YES	YES	NO	9	11	5	446	561	361
SHANTAPPA	70	M	22336	LEFT FRONTO PARIETAL INFARCT	YES	YES	YES	27	25	22	1163	1004	943
KASHIBAI	61	F	149242	LEFT CAPSULO GANGLIONIC INFARCT	YES	YES	NO	13	8	6	782	494	502
SHARADABAI	26	F	25477	LEFT FRONTO PARIETAL INFARCT	NO	NO	NO	7	7	4	184	52	64
APPASAHEB	59	M	25346	LEFT THALAMS,CAPSULOGANGLIONIC INFARCT	YES	NO	YES	15	15	12	451	754	364
MOHAN	76	M	24718	RIGHT CENTRUM,SEMIOVALE INFARCT	YES	NO	YES	7	5	5	772	439	551
BASAVRAJ	40	M	24460	RIGHT BASAL GANGLIA,INTERNAL CAPSULE,THALAMUS INFARCT	YES	NO	YES	18	15	19	874	539	713
HANAMANTHM	46	M	160653	LEFT FRONTO PARIETAL INFARCT	NO	NO	NO	11	7	7	584	473	192
PREMSINGH K	85	F	185000	RIGHT PARIETO -OCCIPITAL , THALAMUS INFARCT	NO	YES	YES	23	19	11	906	884	738
LACHAPPA	44	M	184979	RIGHT PARIETO OCCIPITAL,THALAMUS INFARCT	NO	NO	NO	19	16	13	648	572	453
VIDYA B	32	F	277127	BILATERAL THALAMUS,CAPSULOGANGLIONIC INFARCT	NO	YES	YES	17	12	15	962	784	813
SHEKAPPA	55	M	250651	LEFT PARIETAL LOBE IN FARCT	YES	YES	YES	15	13	11	856	714	639
MANJULA	55	F	22064	RIGHT CAPSULAR INFARCT	YES	NO	YES	8	11	8	452	415	291
MAMTAZ K	50	F	42161	RIGHT TEMPORO PARIETAL INFARCT	YES	YES	NO	16	16	10	678	651	553
SHIASHANKAR	62	M	84990	RIGHT CAPSULO -GANGLIONIC INFARCT	NO	YES	NO	18	22	20	783	971	673
BAPU MANE	56	M	94865	LEFT BASAL GANGLIA INFARCT	NO	NO	NO	6	6	4	136	111	129
ASHOK P	55	M	101728	LEFT CAPSULO GANGLIONIC INFARCT	YES	NO	YES	11	14	10	284	451	372
SHRISHAIL	65	M	20090	LEFT CEREBELLAR INFARCT	YES	YES	NO	23	21	19	948	673	286
GANGARAM	68	M	94283	RIGHT CORONA RADIATA INFARCT	NO	NO	YES	5	7	3	193	179	202
SARUBAI	68	F	16707	LEFT CAPSULO GANGLIONIC INFARCT	YES	NO	YES	19	17	15	884	671	583
SHARANAPPA	70	M	162742	RIGHT PARIETO TEMPORAL INFARCT	YES	NO	YES	15	10	6	757	297	133
KASHIBAI	62	M	165783	RIGHT OCCIPITO TEMPORAL INFARCT	YES	NO	NO	8	5	5	189	212	164
VISHWARAPPA	72	M	136868	RIGHT FRONTO PARIETAL INFARCT	NO	YES	YES	18	18	16	893	812	768
TOOLAWWA	45	F	128772	RIGHT INSULAR CORTEX WITH PUTAMEN I NFARCT	NO	YES	NO	7	7	5	96	116	76
MANINOHAR	76	M	10086	RIGHT FRONTAL LOBE INFARCT	YES	NO	NO	13	17	19	661	649	781
SHANKARGOUDA P	40	M	97216	LEFT CAPSULO GANGLIONIC INFARCT	NO	NO	YES	16	16	14	877	931	753
NOOR JAAN	53	F	72684	RIGHT CAPSULO -GANGLIONIC INFARCT	YES	YES	NO	17	19	15	759	841	629
SAROJANEBAI	76	F	46360	LEFT PARIETAL LOBE IN FARCT	NO	NO	YES	4	4	3	46	58	112
LAGMAVVA	75	M	112386	LEFT CAPSULO GANGLIONIC INFARCT	YES	YES	YES	27	25	25	1452	1135	947
ASHOK P	60	M	11876	RIGHT FRONTO TEPMPORO PARIETAL INFARCT	YES	YES	NO	17	12	11	783	451	394
LATABAI	74	F	12610	B/L CORONA RADIATA INFARCT	NO	YES	YES	20	20	19	887	903	891
SAVITRI	72	F	112319	RIGHT FRONTAL LOBE INFARCT	YES	NO	NO	6	6	4	78	106	92
SHANTA	48	F	113535	LRFT PARIETAL LOBE INFARCT	YES	NO	NO	11	11	9	436	386	337
SIDDAVVA	65	F	122366	RIGHT FRONTO TEPMPORO PARIETAL INFARCT	YES	NO	NO	19	20	17	819	905	756
ARAVIND	75	M	73870	RIGHT FRONTOPARIETAL IN FARCT	NO	YES	YES	14	16	12	562	671	465
BAGAPPA	60	M	33277	RIGHT FRONTO TEMPORAL IN FARCT	NO	YES	NO	19	20	16	786	912	543
BASALINGAPPA	75	M	114563	LEFT FRONTAL ,GENU OF CORPUS CALLOSUM	NO	YES	YES	17	19	17	908	923	845
BASAMMA K	80	F	68037	LEFT FRONTAL LOBE INFARCT	YES	YES	YES	15	18	11	563	781	128
BERAPPA	52	M	56903	RIGHT TEMPORAL LOBE INFARCT	YES	NO	NO	9	7	3	98	74	105
HANAMANYHM	22	M	72895	LEFT FRONTO -TRMPOIRO PARIETAL -OCCIPITAL INFARCT	NO	NO	YES	17	15	14	897	662	391
ISMAIL	35	M	56992	LEFT FRONTAL LOBE INFARCT	NO	YES	YES	16	18	12	471	705	312
RAVI	45	M	65319	RIGHT PARIETAL ,CAPSULO GANGLIONIC INFARCT	NO	YES	NO	19	17	17	817	671	595
SHAKUNTALA	73	F	95939	BILATERAL CEREBELLAR INFARCT	NO	YES	YES	16	17	15	793	697	303
SHRISHAILAYYA	70	M	47384	RIGHT FRONTO -TEMPORO -PARIETAL INFARCT	YES	NO	YES	20	17	11	885	561	608
SIDAMMA	60	F	68646	RIGHT OCCIPITAL ,MIDBRAIN,PONS INFARCT	YES	NO	YES	27	25	22	789	721	664
SHIVANAND	53	M	38686	RIGHT PARIETO LOBE INFARCT	YES	NO	YES	27	29	26	904	998	402
SAHEB GHATE	90	M	160879	LEFT CEREBELLAR INFARCT	NO	NO	YES	11	11	7	351	346	553
KRISHNAJI	61	M	1764411	RIGHT CAPSULO -GANGLIONIC INFARCT	YES	NO	YES	19	19	17	789	763	701
DUNDAVVA NAVI	70	F	209378	RIGHT TEMPORAL LOBE INFARCT	NO	YES	YES	16	14	12	884	891	767
MUTAWWA	70	F	276929	RIGHT CEREBELLAR INFARCT	NO	NO	NO	3	3	3	51	95	28
TUKARAM	68	M	283991	BILATERAL CEREBRAL INFARCT	YES	YES	YES	14	15	17	774	781	516
ITABAI	60	F	289739	RIGHT TEMPORO OCCIPITAL INFARCT	NO	YES	YES	12	11	11	487	414	387
MAMTAZ B	50	F	306953	RIGHT TEMPORO FRONTAL PARIETAL AND OCCIPITAL I NFARCT	YES	NO	NO	15	12	13	586	612	541
SADASHIV S	54	M	40283	RIGHT FRONTO PARIETAL LOBE LEFT OCCIPITAL RIGHT MEDULLA INFARCTS	YES	YES	YES	19	17	17	947	884	749
MODINSAB	60	M	143770	RIGHT PARIETAL LOBE I NFARCT	NO	YES	NO	16	14	14	684	582	616

B.L.D.E. (DEEMED TO BE UNIVERSITY) IEC/NO-09/2021/ Date-22/01/2021

(Declared vide notification No. F.9-37/2007-U.3 (A) Dated. 29-2-2008 of the MHRD, Government of India under Section 3 of the UGC Act, 1956)

The Constituent College

SHRI. B. M. PATIL MEDICAL COLLEGE, HOSPITAL AND RESEARCH CENTRE

INSTITUTIONAL ETHICAL CLEARANCE CERTIFICATE

The Institutional ethical committee of this college met on 11-01-2021 at 11am to scrutinize the synopsis of Postgraduate students of this college from Ethical Clearance point of view. After scrutiny the following original/corrected and revised version synopsis of the Thesis has been accorded Ethical Clearance

Title: A study of correlation between serum cortisol and severity of acute ischemic stroke

Name of PG student: Dr Chirag Mallikarjuna Sajjanar, Department of Medicine

Name of Guide/Co-investigator: Dr R C Bidri, Professor of Medicine

DR .S.V.PATIL
CHAIRMAN
Institutional Ethical Committee
B L D E (Deemed to be University)
Shri B.M. Patil Medical College,
VIJAYAPUR-586103 (Karnataka)

Following documents were placed before Ethical Committee for Scrutinization:

1. Copy of Synopsis / Research project
2. Copy of informed consent form
3. Any other relevant documents.