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Serum Albumin Levels: A Potential Biomarker for Predicting Acute Ischemic Stroke

Dr. P. Rajendra Kumar

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Abstract:

- ***Background:***
  Acute ischemic stroke (AIS) is a leading cause of morbidity and mortality worldwide. Serum albumin levels have been proposed as a potential prognostic indicator for functional outcomes in stroke patients.

- ***Objective:***
  This study aims to evaluate the role of serum albumin levels as a prognostic indicator of acute ischemic stroke, using the modified Rankin scale (mRS) at a 3-month follow-up.

- ***Methods:***
  A cross-sectional study was conducted over 18 months at Katuri Medical College, involving 100 patients diagnosed with AIS within the first 72 hours of symptom onset. Clinical and biochemical parameters, including serum albumin levels, were assessed. The severity of stroke was measured using the Scandinavian Stroke Scale (SSS) and outcomes were evaluated using the mRS.

- ***Results:***
  The study found that 35% of patients had moderate disability, 26% had mild disability, and 9% died. A significant association was observed between low serum albumin levels and increased stroke severity, with lower albumin levels correlating with poorer functional outcomes at 3 months. The mean serum albumin level was significantly lower in patients with severe disability compared to those with mild disability.

- ***Conclusion:***
  Serum albumin levels are significantly associated with the severity of acute ischemic stroke and can serve as a useful prognostic marker for predicting functional outcomes. Monitoring serum albumin levels in stroke patients may help in assessing their nutritional status and guiding treatment strategies. Further studies are warranted to explore the therapeutic implications of albumin supplementation in stroke management.

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I. INTRODUCTION

Stroke is a leading cause of neurological disability in adults. Approximately two-thirds of those who survive a stroke experience lasting neurological problems that affect their ability to function. Around half of all stroke survivors are left with disabilities that make them reliant on others for help with everyday tasks.2

Following a stroke, the central nervous system (CNS) reorganizes itself as part of the functional recovery process.1 However, the extent of this recovery varies, and the underlying mechanisms are not fully understood.2 Neurophysiological changes related to recovery typically begin within one to two weeks post-stroke, potentially peaking between two and three months, depending on the severity and scope of the initial neurological impairment.3 Various factors like admission incontinence, age, functional ability, hemineglect, and others may affect functional outcome of stroke patients.4

Among undernourished people, stroke like acute illness can cause negative energy balance and more nutritional demand. Stroke patients may not meet increased demands.5 Serum albumin level was considered as a marker of nutritional status.6 It can detect acute changes in nutrition. Baseline measurements of serum albumin may not influence acute stress response after stroke. Hypoalbuminaemia during the time of admission may be linked to pre- morbid nutritional status attributable to high half-life of the albumin.7

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Albumin helps patients with acute ischemic stroke (AIS) due to its hemodilution, antioxidant, and neuroprotective effects. Hypoalbuminemia during the time of admission may have negative influence on neuroprotection among stroke patients. If this hypothesis is true, treating patients having hypoalbuminemia with acute stroke might improve their functional outcomes. Hence the current study was undertaken.

II. AIM & OBJECTIVES

➢ *Aim:*

- To determine if blood albumin is a reliable predictor of acute ischemic stroke using a modified Rankin scale at the three-month patient follow-up.

➢ *Objectives:*

- To determine, using a modified Rankin scale at a 3-month follow-up, the clinical and biochemical parameters influencing the prognosis of acute ischemic stroke.
- To use the Scandinavian stroke scale to gauge the severity of the stroke at admission.

III. REVIEW OF LITERATURE

A. Stroke:

A stroke is characterized as a state of sudden, localized neurological impairment brought on by vascular damage to the central nervous system (CNS), such as an infarction or hemorrhage.

B. Classification:

Stroke is of two types mainly. Ischemic stroke (IS) is a common stroke that occurs due to interruption of blood flow to a part of the brain. It constitutes 85% of total strokes.

Haemorrhagic strokes constitute 15% of strokes.22 They are caused by acute haemorrhage. Intracerebral haemorrhage (ICH) and subarachnoid haemorrhage are types of haemorrhagic strokes.

As per the TOAST classification, there are 5 types of ischemic strokes.

➢ *8They are:*

- Large artery atherosclerosis
- Small vessel occlusion
- Cardio-embolism
- Stroke of undetermined causes
- Stroke of other determined cause

C. Etiology:

Arteriosclerosis, hypertension and emboli due to atrial fibrillation or rheumatic heart disease, cerebral amyloid angiopathy are some of the reasons in elderly patients. Causes in younger patients include clotting disorders, cervical arterial dissection, and vasculitis.

Other risk factors include diabetes mellitus (DM), increasing age, obesity, smoking and genetics.

Alcohol intake shows a J-shaped relationship with IS. Mild to moderate drinking may decrease the risk of IS but heavy drinking enhances the risk significantly.

Lack of vascular structural integrity like aneurysms, arteriovenous malformations, cavernous malformations, capillary telangiectasias, venous angiomas, and vasculitis could lead to haemorrhagic stroke.

D. Clinical Features:

Features will depend on the artery that was affected. If Middle cerebral artery (MCA) is involved, it causes contra-lateral hemiparesis with gaze towards the side of lesion. In case of anterior cerebral artery(ACA) stroke, speech is preserved but there is d is-inhibition. Mental status and judgement may be impaired. In case of posterior cerebral artery (PCA) stroke, cortical blindness contralateral homonymous hemianopia, visual agnosia along with altered mental status will be seen.

In case of basilar artery stroke, vertigo, nystagmus, ataxia, syncope may be seen.

➢ *Findings as Per the Artery Involved:9-13 Anterior Cerebral Artery (ACA)*

- Leg more than arm involvement with hand sparing Gait apraxia
- Urinary incontinence Akinetic mutism.

✓ *Middle Cerebral Artery Involvement (MCA)*

- Homonymous hemianopia/quadrantanopia Face– arm– leg involvement
- Aphasia (Broca's if there is involvement of superior division; Wernicke's if there is inferior division involvement)
- Inattention
- Gaze paralysis

✓ *Vertebrobasilar*

- Cerebellum – ataxia, nystagmus
- Occipital lobe – homonymous hemianopia,
- Brainstem cranial nerve palsies – diplopia, along with facial numbness, dysphagia, vertigo, dysphonia
- Spinal tracts – hemiparesis

✓ *Lacunar Stroke Syndromes*

- Pure motor hemiparesis Ataxic hemiparesis
- Pure sensory stroke Sensori-motor stroke

E. Evaluation:

- Imaging: Emergency CT without contrast is the first imaging test to confirm the diagnosis and rule a hemorrhagic stroke.

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A diffusion-weighted MRI is the most diagnostic of acute ischemic infarction. A CT angiogram aids in knowing the location of blockage in the vasculature.

CT perfusion studies identify the ischemic core - may be a guide to a decision on revascularization after 6 hours.

Doppler studies help to know the degree of carotid stenosis.[14]

Other tests include complete blood count with differential, lipid profile, hemoglobin A1c (HbA1c), blood urine nitrogen (BUN), creatinine, albumin, and glomerular filtration rate (GFR) to rule out the cause.

Random blood glucose, platelet count (PT) and PT/PTT (Plasma thromboplastin time) helps to determine if the patient is eligible IV thrombolysis candidate.

F. Prognosis:

The prognosis depends on the degree, area involved, involved structures, time of diagnosis, time of starting treatment, intensity of physical and baseline functioning.[15,16]

G. Albumin

The most abundant plasma protein accounts for 55-60% of serum protein content. It consists of 585 amino acids, comprising a single polypeptide chain.[17]

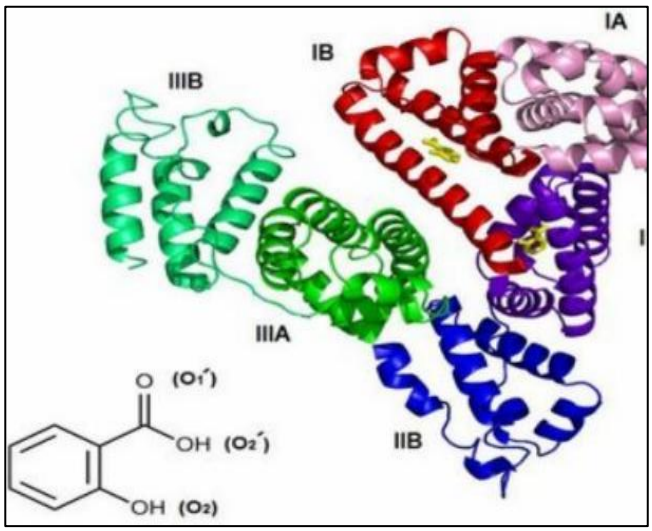

Image 1: Structure of Albumin

It has an abundance of charged particles but no residues of tryptophan or methionine. Its tertiary structure, as shown by X-ray crystallography, has a heart-like shape with a molecular weight of 66500Da.

It has great flexibility and can easily change form. Its three domains form an ellipsoid pattern in solution, which results in a structure with low viscosity. Its ability to readily restore its form under physiological circumstances is due to the existence of disulfide bridges. Only extreme non-physiological changes in temperature, pH, or chemical environment can cause denaturation.

H. Functions of Albumin:

➢ *Maintaining Oncotic Pressure:*[18]

With a molecular weight of 66.5 kDa, albumin is more abundant in plasma than other plasma proteins. Despite having a lesser molecular weight (147 kDa) than the typical globulin, albumin has a major osmotic effect.

Because of its direct osmotic action, albumin accounts for 60% of the usual oncotic pressure in a healthy condition. The remaining 40% is due to the molecule's negative charges, which cause positively charged solute particles to be retained intravascularly (Gibbs-Donnan effect).

Albumin's huge extravascular pool, water solubility, and net negative charge make it an essential component in controlling the distribution of tissue fluid.

I. Attachment of Substances:

Because of its adaptable structure, albumin can hold a variety of substances; bound molecules are frequently buried inside it. There is no relationship between the chemicals' charge and the strength of binding. Hematin, bilirubin, and long-chain fatty acids are examples of medium-sized hydrophobic organic anions that bind to albumin the strongest.

Additionally, albumin binds to ascorbate and tryptophan specifically, but less strongly.

The substance's chirality affects the binding strength as well. Divalent cations, such as calcium and magnesium, are bound by albumin, whereas monovalent cations are not. Albumin tends to bind to basic medications.

Eicosanoids, copper, zinc, folate, aquacobalamin, and bile acids are examples of endogenous compounds that bind to albumin. Moreover, albumin acts as a secondary or tertiary carrier for chemicals like thyroxine, vitamin D, and steroids that have particular binding proteins.

J. Metabolic Functions:

Certain substances, such as antibiotics from the penem group and d isulfiram, are inactivated by albumin. Its ability to bind aids in the metabolism of compounds such as eicosanoids and lipids. Albumin promotes lipoxygenase activity over cyclooxygenase activity, releases arachidonic acid from macrophages, and stabilizes eicosanoids such as prostaglandin I2 and thromboxane A2.

➢ *Function of Acid-Base:*[19]

Because albumin is abundant in plasma and has charged residues on its molecules, it functions as an efficient buffer. Albumin has a net negative charge of 19 and contributes to a half-normal anion gap physiologically.

An anion gap is reduced by 3 mmol/L when serum albumin is reduced by 1 g/dl, improving standard bicarbonate by 3.4 mmol/L.

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➢ *The Function as Antioxidant:*[20]

Albumin has antioxidant qualities under physiological settings by scavenging free radicals linked to a number of illnesses. Because albumin contains sulfhydryl groups, it prevents active polymorphonuclear leukocytes from producing oxygen free radicals through the enzyme myeloperoxidase.

➢ *Preserving the Integrity of Micro-vasculature:*[21]

Albumin controls the rise in capillary permeability brought on by stress, possibly via altering the distribution and makeup of glycoproteins in the vessel wall. The molecule's negative charge or the albumin molecule's lowering of the channel size may be the cause of this activity. Additionally, endothelial cell apoptosis is prevented by albumin, which works best at normal concentrations. By attaching to sulfhydryl groups and producing S-nitrosothiol groups that are not quickly broken down, it also modifies the onset and improves the vasodilatory response to nitric oxide. In order to control the vasodilatory tone of vessels, albumin is essential.

➢ *Impact as Anticoagulant:*[22]

Because of the many negatively charged groups in its molecular structure, albumin has characteristics similar to those of heparin.

By attaching its negatively charged sulfate groups to positively charged antithromb in III groups, heparin produces its anticoagulant actions. Additionally, albumin strengthens antithromb's neutralization of factor Xa in III. The observed inverse relationship between albumin levels and heparin needs in hemodialysis patients may be explained by this mechanism. It might contribute to the explanation of the hypercoagulable condition seen in nephrotic syndrome. Both reliant and independent of the cyclo-oxygenase system, albumin exhibits inhibitory effects on platelet aggregation.

➢ *Value of Serum Albumin in Prognosis:23*

Serum albumin predicts mortality independently in a variety of clinical settings. Higher blood albumin levels in older adults may be a sign of subclinical illness. Low blood albumin concentrations have been linked to longer hospital stays, more problems, and greater death rates, according to studies done on hospitalized patients.

Additionally, extended stays in the critical care unit, increased need for a ventilator, and a higher risk of infection are all associated with low blood albumin levels. It may be possible to anticipate the weaning ability of patients on mechanical breathing by looking at the daily trend of blood albumin levels.

When it comes to predicting mortality, serum albumin levels within 24 to 48 hours following intensive care unit admission have shown predictive powers on par with APACHE II scores.

Serum albumin levels are incorporated into the APACHE III system, which provides better mortality prediction in critical illness. Serum albumin is not a good indication of nutritional status in critically sick patients, despite its use as a nutritional marker.

K. Treatment with Intravenous Albumin:

For volume replacement in critically sick patients, this treatment has not been shown to be superior to alternative colloids. However, it had a positive effect on burn victims, especially in the first 24 hours, when capillary permeability and fluid changes significantly rise. After this period, intravenous albumin facilitates the reabsorption of plasma[24-27]

Burns that involve less than 15% of the body's surface area usually do not require albumin, according to the Paris Conference's Consensus Guidelines, whereas burns that affect more than 50% of the body surface area call for rapid albumin intervention. It is possible to postpone albumin supplementation for a full day in situations that lie in between.

By lowering plasma renin activity, albumin treatment helps cirrhotic patients with ascites undergoing paracentesis avoid post-paracentesis circulatory dysfunction.

L. Limitations:

More risk of fluid overload from plasma expansion, myocardial depression potentially linked to calcium ion binding, allergic reactions primarily attributed to solution contaminants or storage-related polymers and a theoretical risk of viral transmission, albeit minimized by stringent preparation methods.

Moreover, albumin therapy is cost-intensive, warranting further research and interventional studies to resolve controversies and establish its efficacy as a standard treatment modality for reducing mortality in critically ill patients.

IV. PATIENTS & METHODS

The current study was conducted in the Department of General Medicine, Katuri Medical College, Guntur, Andhra Pradesh, India.

- Study Period: 18 months from November 2022 to April 2024.
- Type of Study: Cross-sectional study.
- Source of Data: After getting approval from Institutional Ethics Committee, patient admitted in the ward of General Medicine department with acute ischemic stroke with 1st 72 hours were taken as study sample.
- Sampling Procedure: Simple random sampling.
- Sample size calculation: As per the previous study done, the prevalence of stroke in India was 1.53%.

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➢ *The Sample Size was Calculated as Follows:*

N=Z2 PQ/E2

N-sample size
P-Prevalence

Q= 1-P

E-Error: 3%

Confidence Intervals=98%

N=91

18

The minimum sample size came to be 91. Hence we included 100 patients in the current study considering 10% of drop outs.

➢ *Inclusion Criteria:*

- Male and female patients
- Patients aged more than 18 years
- Patients with AIS- confirmed BY CT/MRI SCAN OF BRAIN
- Patients who provided consent to participate in the study

➢ *Exclusion Criteria:*

- l Patients with chronic kidney disease, liver disease, heart failure and dementia.
- l Patients with malignancies
- l Patients with acute haemorrhagic stroke, ischemic stroke with hemorrhagic transformation or stroke related to intracranial space occupying lesion (ICSOL).
- l Patients with past history of stroke
- l Patients presenting more than 72hrs hr after the onset of stroke
- l Patients with fever or infections.
- l Patients were excluded based on previous medical records and oral history given by the patient or relatives.19

➢ *Parameters Assessed:*

- Age
- Gender
- BMI
- Smoking
- Alcoholism
- Type of lesion
- Glassgow coma scale(GCS)
- Scandinavian stroke scale (SSS) score
- Serum albumin levels
- Presence of diabetes
- Presence of hypertension
- Presence of dyslipidemia
- Presence of CAD
- MRS SCORE
- Artery involved
- Mortality rate
- Duration of hospital stay
- Re-admission need 20

➢ *Definitions:*

- l DM : FBS above 126 mg/dl or PPBS above 200 mg/dl or HBA1C above 6% or if the patient was using antidiabetic medications.
- l HTN: Patient was using antihypertensives or BP above 140/90 – 3 readings after 5 min of sitting.
- l Outcomes assessed: Mortality rate, duration of hospitalization, need for re-admission during one week of follow up.

➢ *Statistical Analysis*

The data were entered in Excel 2023 and data analysis were done using statistical software called Epi Info version 7.2.5

Frequencies, percentages were also used.

Stroke patients were categorized as per mRS score and all parameters were compared among various groups of strokes.

Numerical parameters were compared using ANOVA test and categorical parameters were compared using chi-square test. P value <0.05, was considered as statistically significant.[21]

➢ *Ethical Considerations:*

Permission from the Institutional Ethical Committee attached to Katuri Medical College, Chinakond rupadu was taken before conducting the study. Informed consent was taken from all study patients.

V. RESULTS

➢ *Disability:*

35% of the patients had moderate disability, 26% had mild disability and 9% died in the current study. This classification is as per modified rankin scale.

Table 1: Disability

DISABILITY	Frequency	Percent
DEATH	9	9.00%
MILD	26	26.00%
MODERATE	35	35.00%
SEVERE	30	30.00%

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Total	100	100.0%

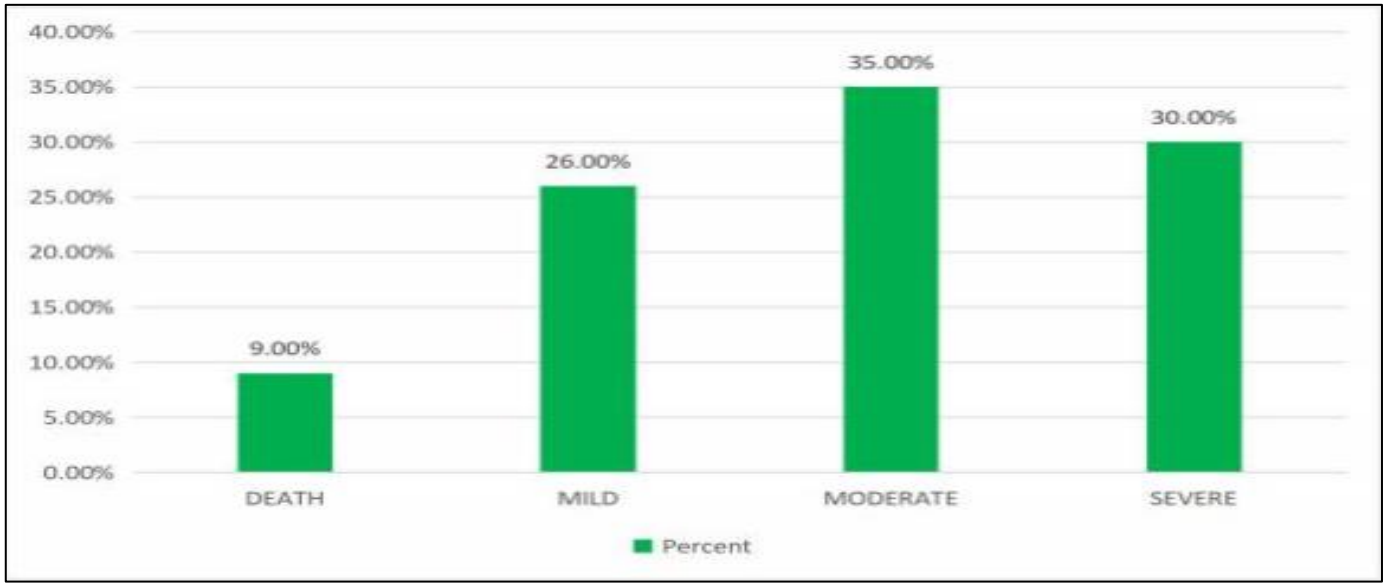

Fig 1: Bar Diagram Showing Disability

- *Age of Patients:*
  There is no significant difference in mean age in between groups of patients in the current study.

Table 2: Variations in Ages of Patients

MEANS				
GROUP	**OBS**	**MEAN**	**VARIANCE**	**STD. DEV**
Death	9.0000	53.7778	107.6944	10.3776
MILD	26.0000	55.5769	49.6138	7.0437
MODERATE	35.0000	53.1429	58.4790	7.6472
SEVERE	30.0000	52.4333	68.5299	8.2783

ANOVA

Variation	**SS**	**df**	**MS**	**F statistic**
Between	150.00591	3	50.00197	0.78982
Within	6077.55409	96	63.30786	
Total	6227.56000	99		

P-Value = 0.50248

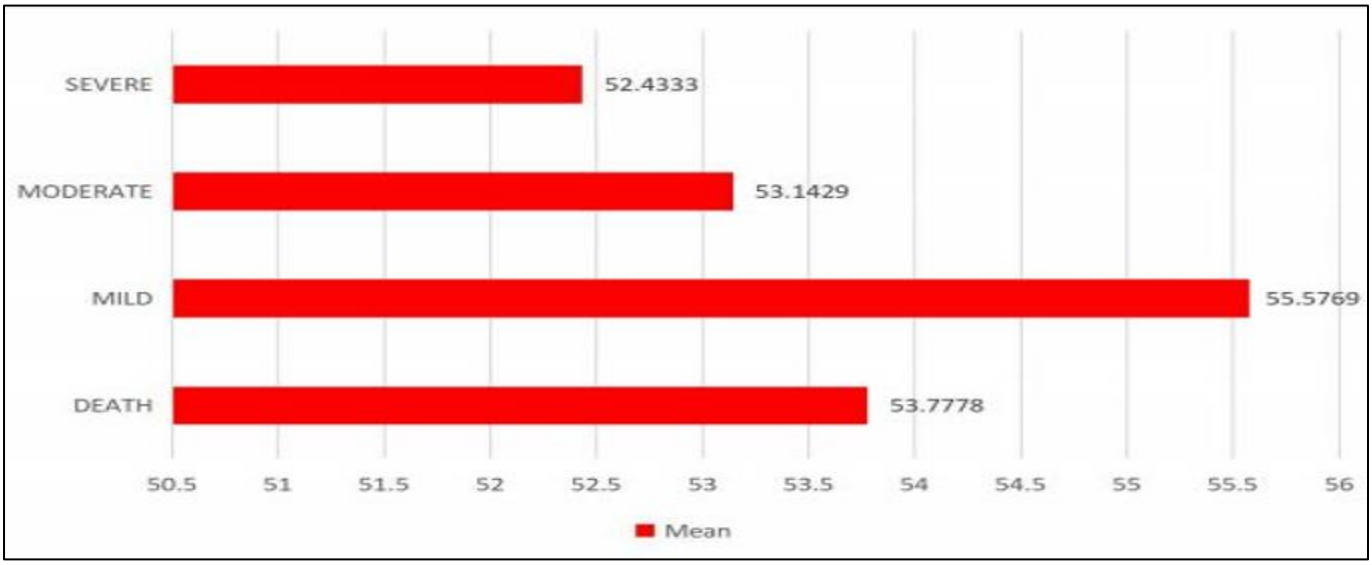

Fig 2: Bar Diagram Showing Variation Amon Ages

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➢ *Gender of Patients:*
There is no significant association between gender and groups of patients in the current study. Overall 69 patients were males.

Table 3: Variations in Gender of Patients

	Disability				
Gender	**Death**	**Mild**	**Moderate**	**Severe**	**Total**
F	3	7	14	7	31
M	6	19	21	23	69
Total	**9**	**26**	**35**	**30**	**100**

Single Table Analysis

Chi-Squared	df	Probability
2.3747	3	0.4984

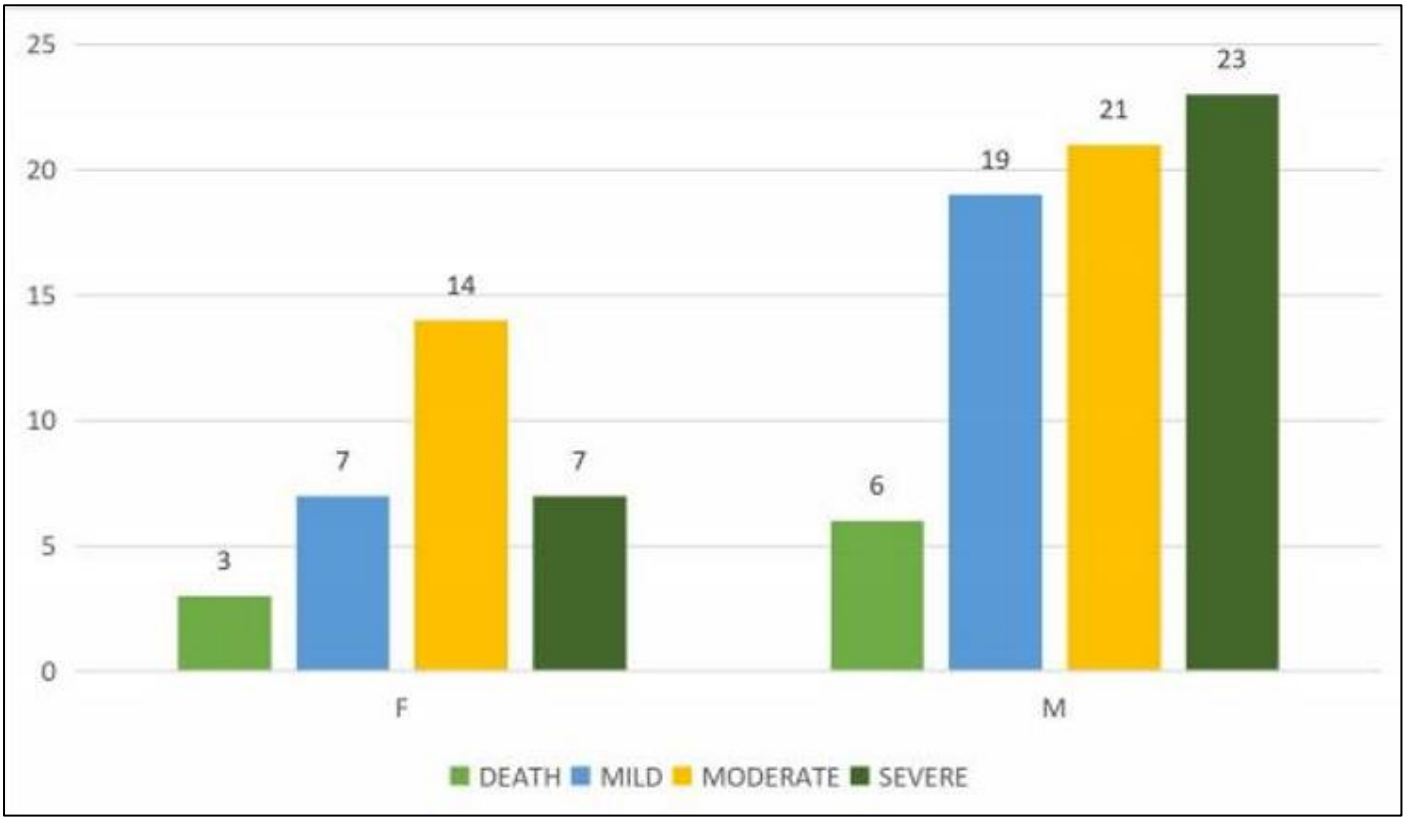

Fig 3: Bar Diagram Showing Variation Amon Genders

➢ *Smoking:*
There is no significant association between smoking and severity of stroke or severity of disability as per mRS in the current study. Overall 26% of patients were smokers.

Table 4: Variation in Smokers

	Disability				
Smoking	**Death**	**Mild**	**Moderate**	**Severe**	**Total**
N	5	22	25	22	74
Y	4	4	10	8	26
Total	**9**	**26**	**35**	**30**	**100**

Single Table Analysis

Chi-Squared	df	Probability
3.2414	3	0.3559

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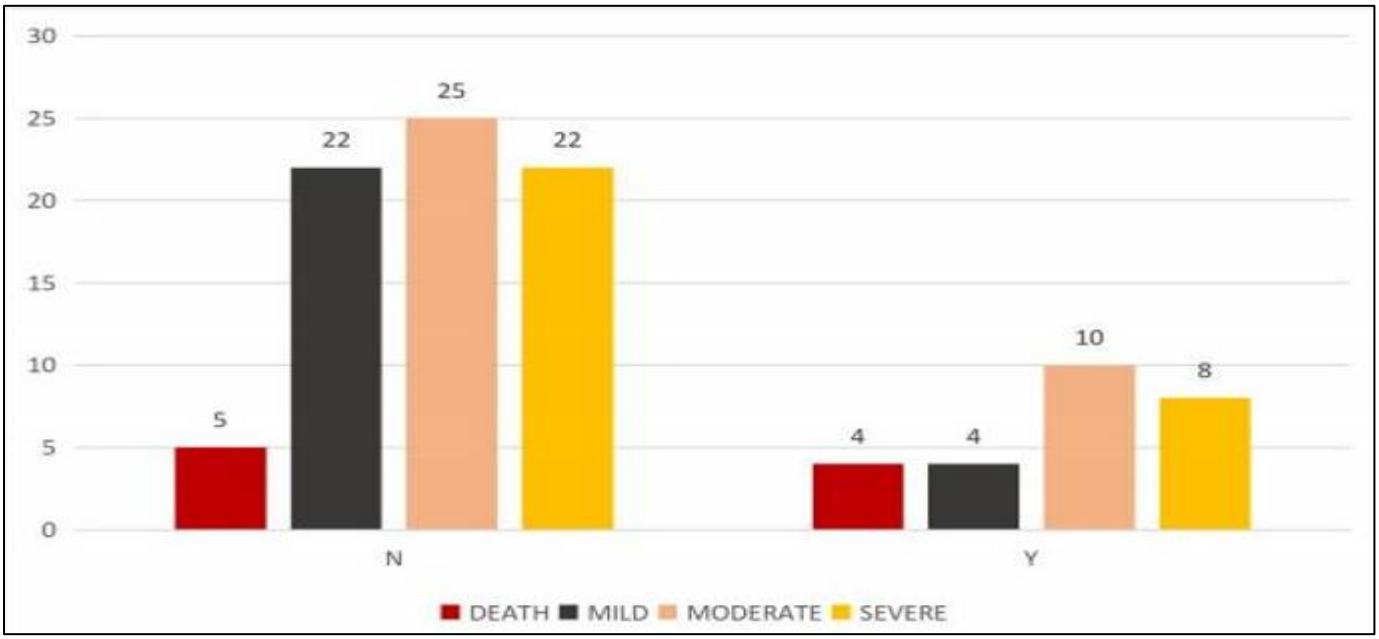

Fig 4: Bar Diagram Showing Variation Amon Smokers

➢ *Alcoholism:*

There is no significant association between alcholism and severity of stroke or severity of disability as per mRS in the current study. Overall 20% of patients were smokers.

Table 5: Variation in Alcoholics

	Disability				
Alcoholism	**Death**	**Mild**	**Moderate**	**Severe**	**Total**
N	7	22	30	21	80
Y	2	4	5	9	20
Total	**9**	**26**	**35**	**30**	**100**

Single Table Analysis

Chi-Squared	**df**	**Probability**
2.9632	3	0.3973

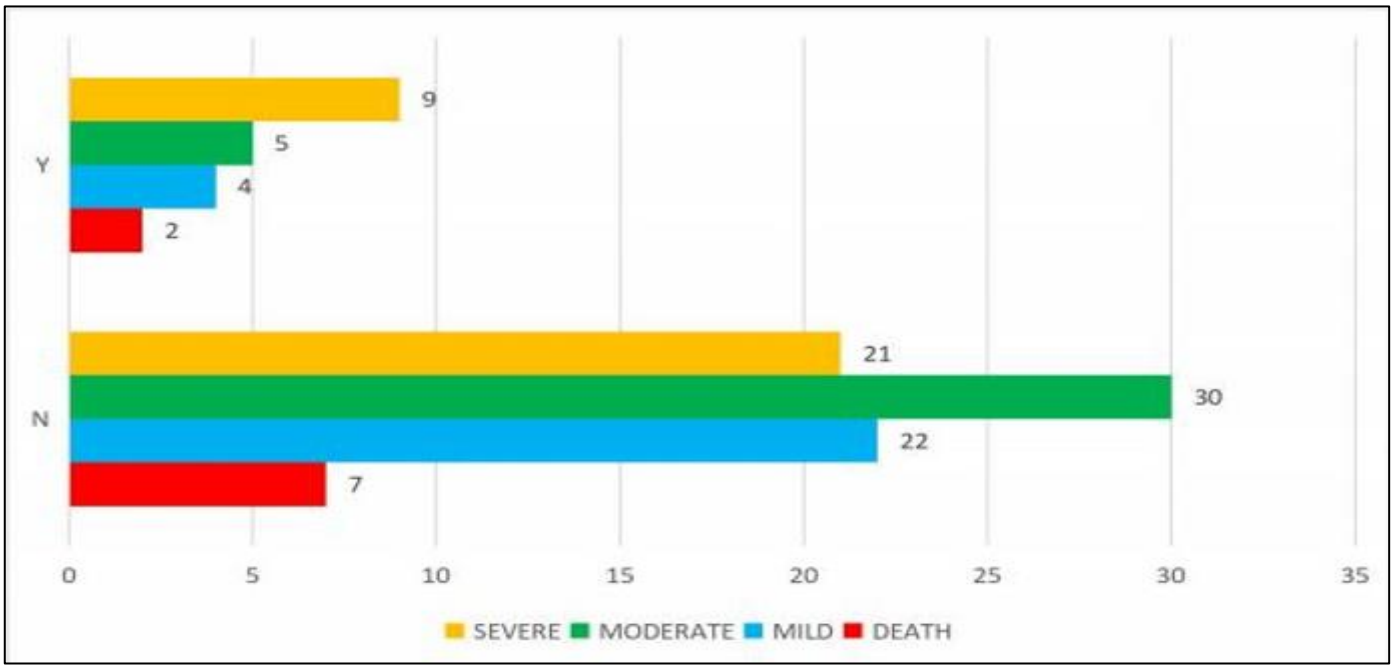

Fig 5: Bar Diagram Showing Variation Amon Alcoholics

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➢ *HTN:*

There is no significant association between presence of hypertension and severity of stroke or severity of disability as per mRS. Overall 20% of patients were hypertensives.

Table 6: Variation in Hypertensives

	Disability				
HTN	**Death**	**Mild**	**Moderate**	**Severe**	**Total**
N	7	22	28	23	80
Y	2	4	7	7	20
Total	**9**	**26**	**35**	**30**	**100**

Single Table Analysis

Chi-Squared	df	Probability
0.5823	3	0.9005

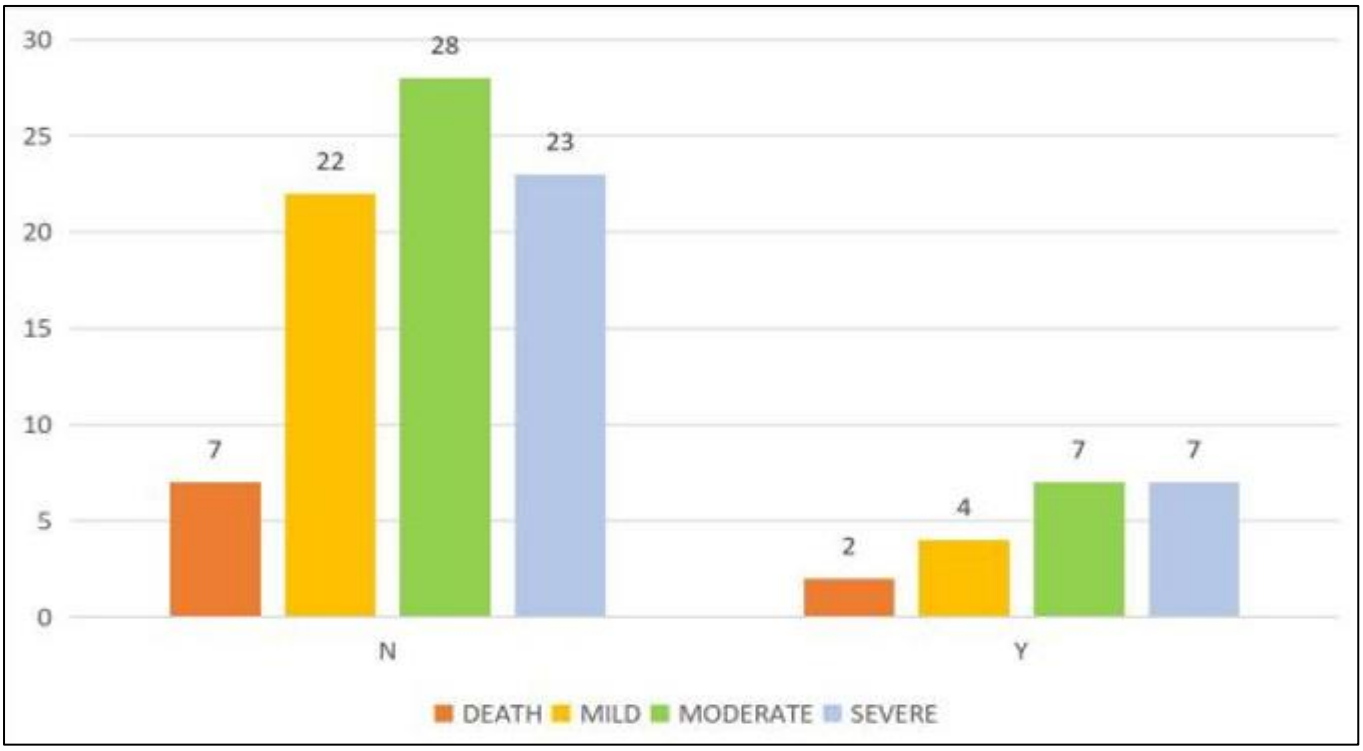

Fig 6: Bar Diagram Showing Variation Amon Hypertensives

➢ *Dm:*

There is no significant association between presence of diabetes and severity of stroke or severity of disability as per mRS. Overall 23% of patients were diabetic patients.

Table 7: Variation in Diabetics

	Disability				
DM	**Death**	**Mild**	**Moderate**	**Severe**	**Total**
N	4	23	27	23	80
Y	5	3	8	7	20
Total	**9**	**26**	**35**	**30**	**100**

Single Table Analysis

Chi-Squared	df	Probability
7.317	3	0.0625

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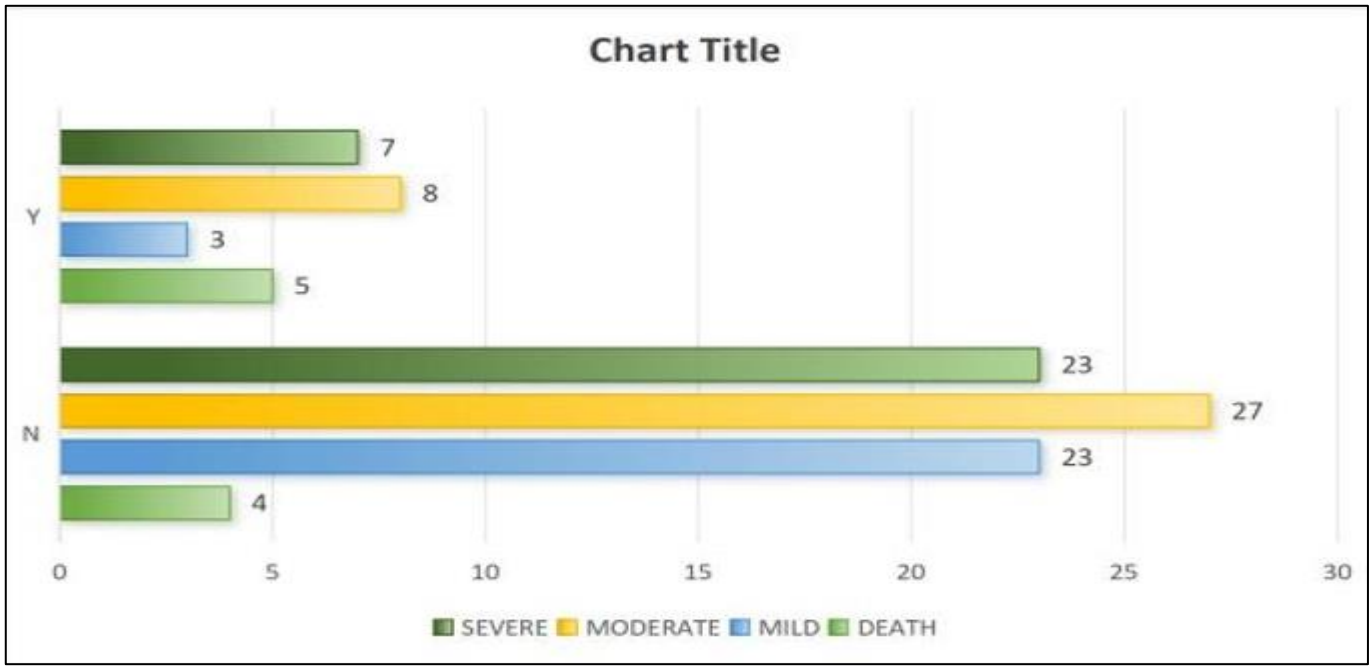

Fig 7: Bar Diagram Showing Variation Amon Diabetics

➢ *CAD:*

There is no significant association between presence of coronary artery disease and severity of stroke or severity of disability as per mRS. Overall 11% of patients had CAD.

Table 8: Variation in Patients With Cad

	Disability				
CAD	**Death**	**Mild**	**Moderate**	**Severe**	**Total**
N	7	22	34	26	89
Y	2	4	1	4	11
Total	**9**	**26**	**35**	**30**	**100**

Single Table Analysis

Chi-Squared	**df**	**Probability**
4.2057	3	0.2401

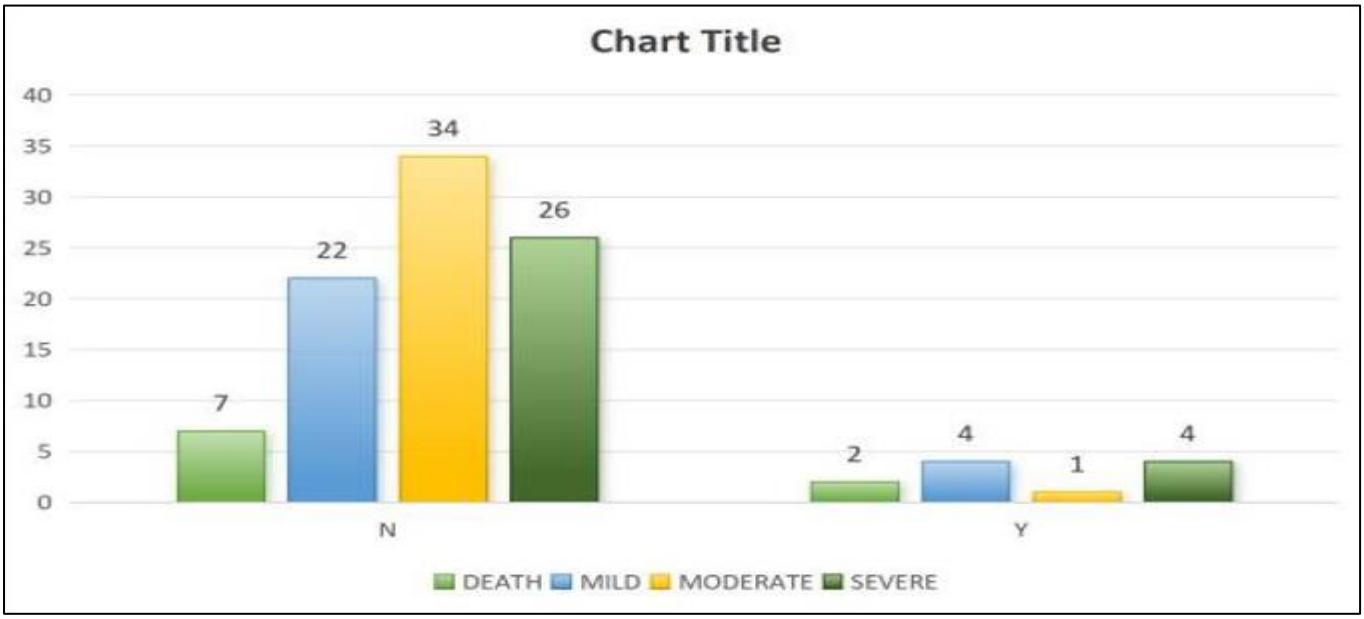

Fig 8: Bar Diagram Showing Variation Amon Patients with CAD

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➢ *Dyslipidemia:*

There is no significant association between presence of dyslipidemia and severity of stroke or severity of disability as per mRS. Overall 18% of patients were hypertensives.

Table 9: Variation in Patients with Dyslipidemia

	Disability				
Dyslipidemia	**Death**	**Mild**	**Moderate**	**Severe**	**Total**
N	8	21	30	23	82
Y	1	5	5	7	18
Total	**9**	**26**	**35**	**30**	**100**

Single Table Analysis

Chi-Squared	**df**	**Probability**
1.2213	3	0.7479

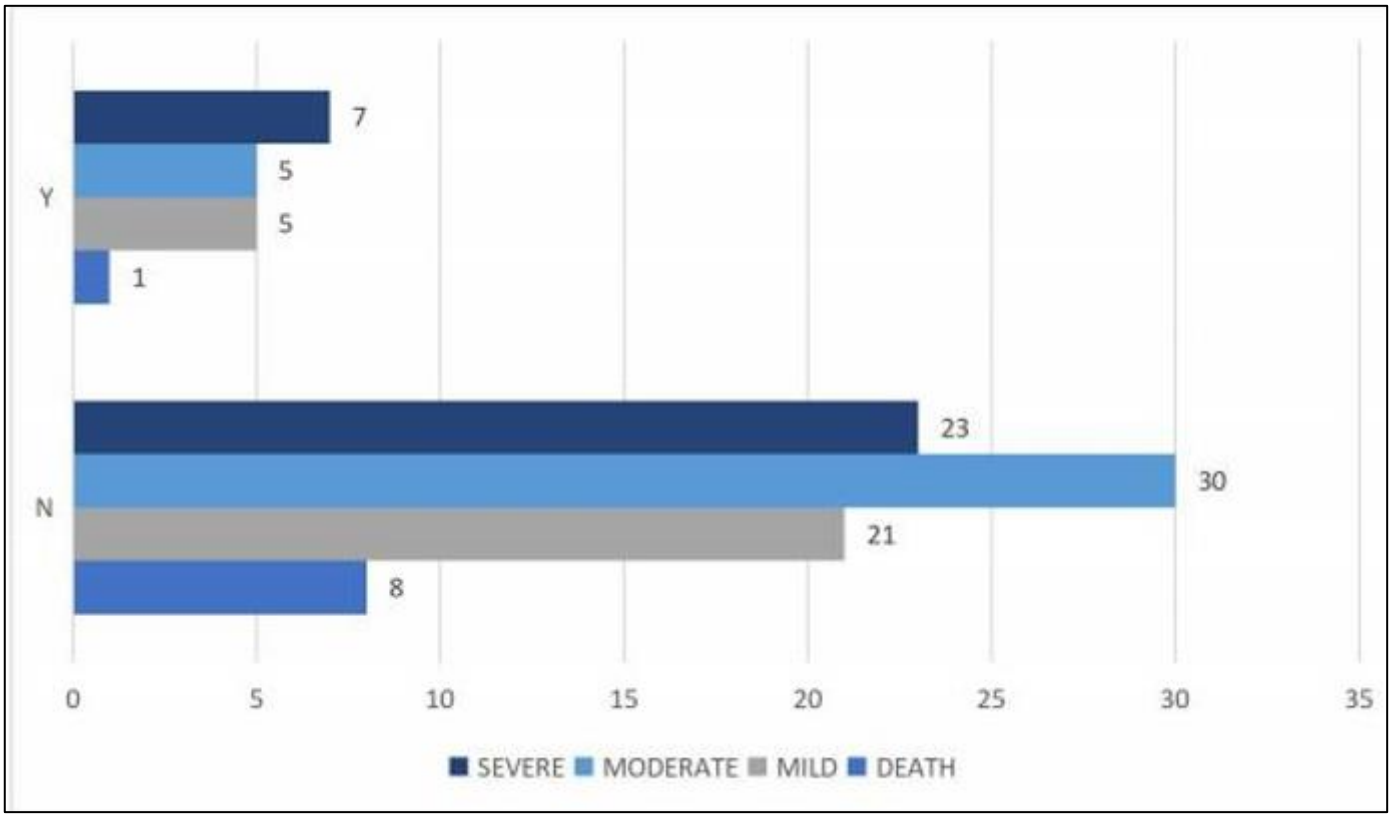

Fig 9: Bar Diagram Showing Variation Amon Patients with Dyslipidemia

➢ *GCS Scale:*

There is significant difference in mean GCS scale in between groups of patients in the current study. GCS score was less in patients who died or having severe stroke as per mRS scale.

Table 10: Mean Values of GCS Among Different Study Groups

MEANS				
GROUP	**Obs**	**Mean**	**Variance**	**Std Dev**
DEATH	9.0000	8.3333	4.0000	2.0000
MILD	26.0000	12.8077	4.7215	2.1729
Moderate	35.0000	11.5429	4.4319	2.1052
SEVERE	30.0000	8.5333	5.4299	2.3302

ANOVA

Variation	**SS**	**df**	**MS**	**F statistic**
Between	331.56916	3	110.52305	23.15675
Within	458.19084	96	4.77282	
Total	789.76000	99		

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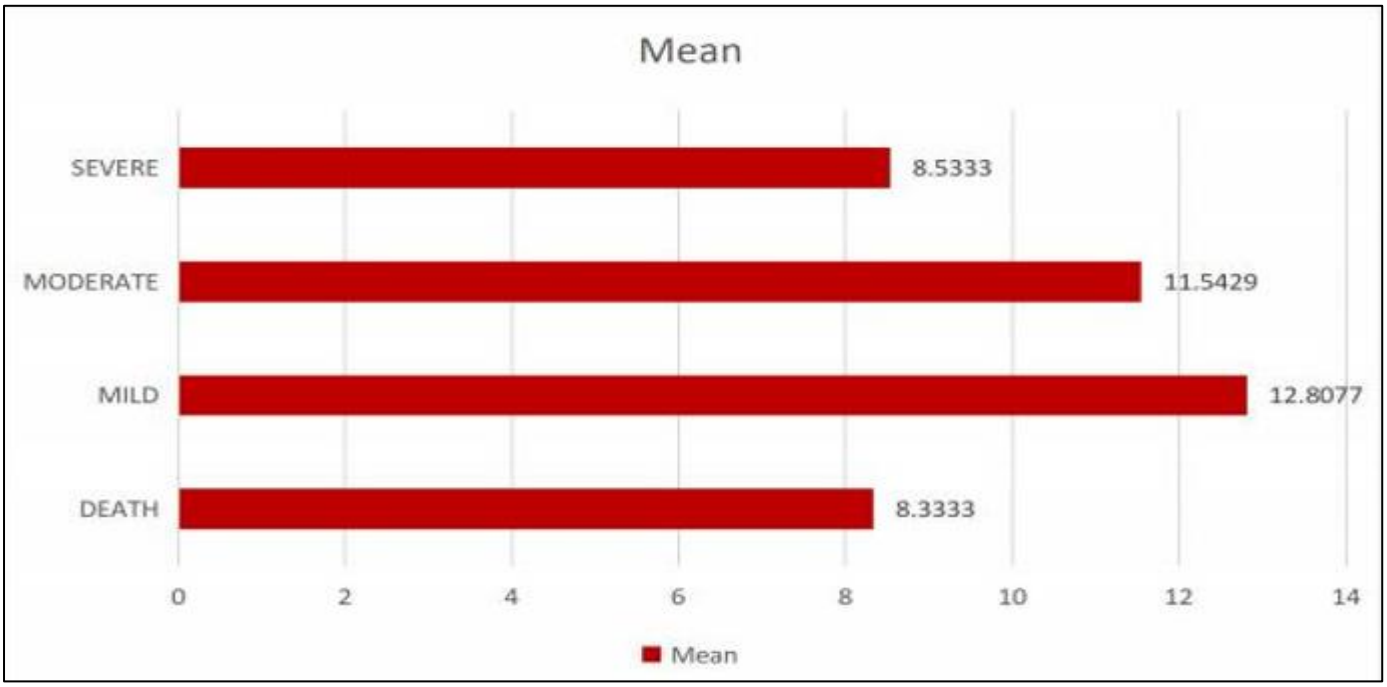

Fig 10: Mean Values of GCS Among Different Study Groups

- *GCS Scale CAT:*

31% of the patients had belonged to severe GCS scale category in the current study (score below 9).

Table 11: Mean Values of GCS Among Current Study Groups

GCS SCALE CAT	Frequency	Percent
MILD	40	40.00%
MODERATE	29	29.00%
SEVERE	31	31.00%
Total	100	100.00%

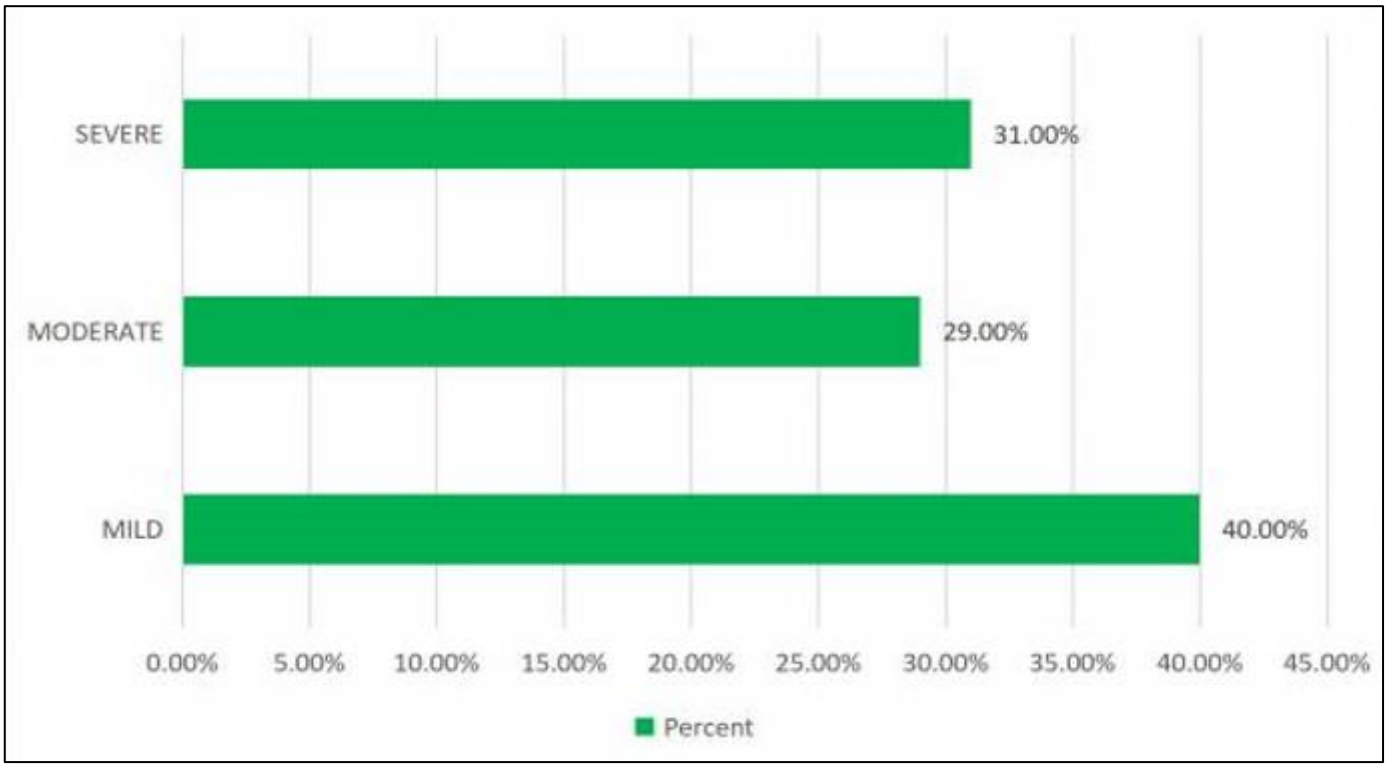

Fig 11: Mean Values of GCS Among Current Study Groups

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- *SSS CAT:*
  26% of the patients belong to severe SSS category in the current study.

Table 11: Mean Values of SSS Category Study Groups

SSS CAT	Frequency	Percent
MILD	63	63.00%
MODERATE	11	11.00%
SEVERE	26	26.00%
Total	100	100.00%

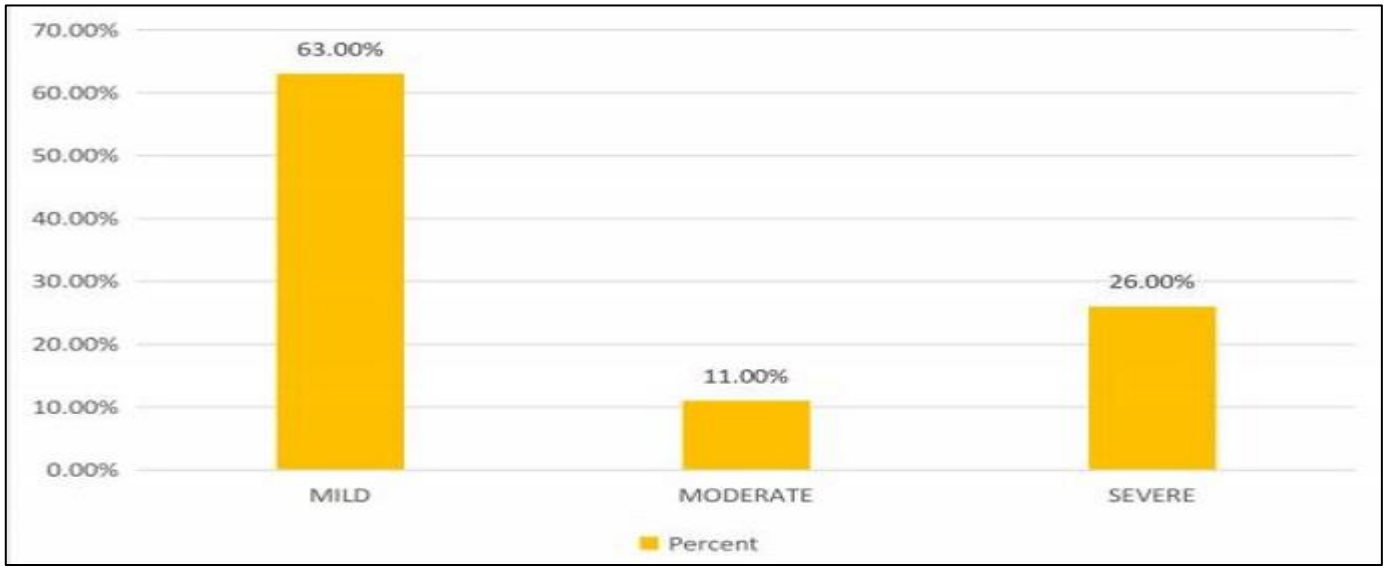

Table 12: Mean Values of SSS Category Study Groups

- *Type of Lesion:*
  24% of the patients had multiple infarcts. 24% had lacunar infarct and 52% had macular infarct in the current study.

Table 13: Types of Lesions in Study Groups

TYPES OF LESIONS	Frequency	Percent
LACUNAR INFARCT	24	24.00%
MACULAR INFARCT	52	52.00%
MULTI INFARCT	24	24.00%
Total	100	100.00%

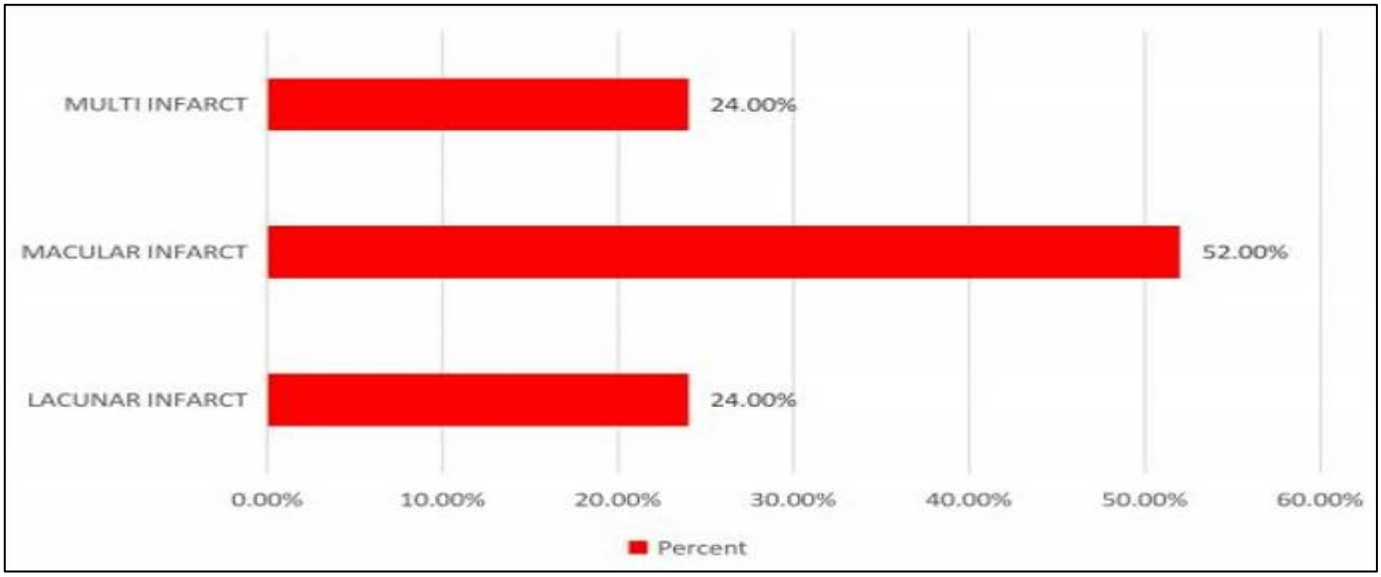

Fig 13: Types of Lesions in Study Groups

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➢ *GCS Scale CAT & Serum Albumin:*

There is significant difference in mean GCS score & serum albumin in the current study. Mean serum albumin was less in patients with severe GCS category.

Table 14: Table Showing GCS CAT & Serum Albumin in Study Groups

MEANS				
GROUP	**Obs**	**Mean**	**Variance**	**Std Dev**
mild	40.0000	4.1153	0.0797	0.2823
Moderate	29.0000	3.6628	0.1705	0.4129
SEVERE	31.0000	3.1558	0.1063	0.3260

ANOVA

Variation	SS	df	MS	F statistic
Between	16.10011	2	8.05006	70.54734
Within	11.06853	97	0.11411	
Total	27.16864	99		

P-Value = 0.00000

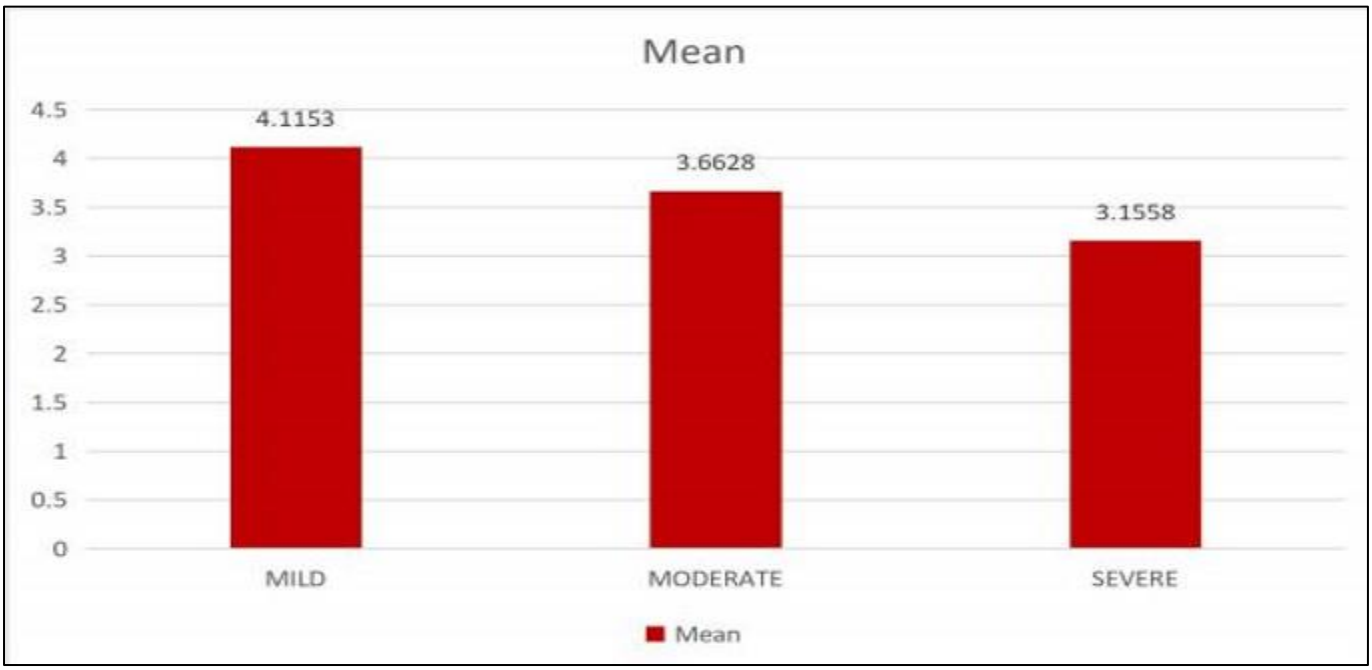

Fig 14: Bar Diagram Showing GCS Cat & Serum Albumin in Study Groups

➢ *SSS CAT & Serum Albumin:*

There is significant association between mean SSS and serum albumin in the current study.

Table 15: Table Showing Serum Albumin in SSS CAT Study Group

MEANS				
GROUP	**Obs**	**Mean**	**Variance**	**Std Dev**
mild	63.0000	3.9402	0.1568	0.3960
Moderate	11.0000	3.6009	0.2679	0.5176
SEVERE	26.0000	3.1085	0.0779	0.2791

ANOVA

Variation	SS	df	MS	F statistic
Between	12.82152	2	6.41076	43.34272
Within	14.34713	97	0.14791	
Total	27.16864	99		

P-Value = 0.00000

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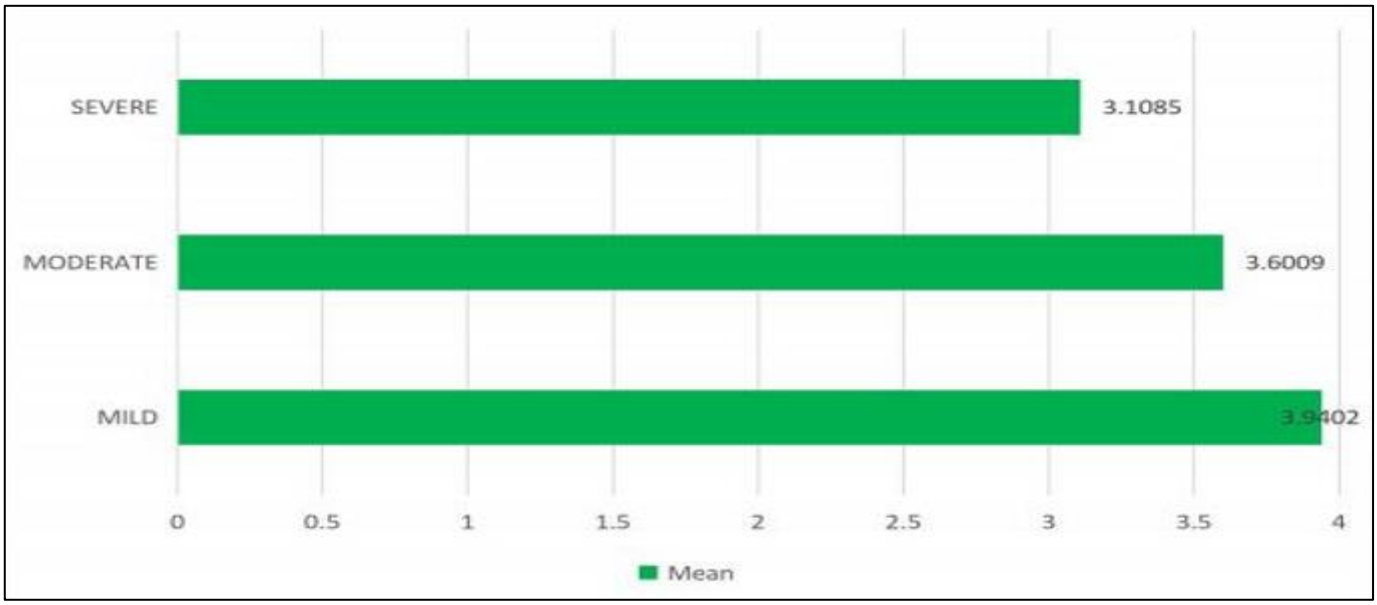

Fig 15: Bar Diagram Showing Serum Albumin in SSS CAT Study Group

➢ *Type of Lesion:*

There is significant difference in mean serum albumin between various types of lesions. Mean serum albumin was less in patients with multiple infarcts.

Table 16: Table Showing Type of Lesions Study Group

MEANS				
GROUP	**Obs**	**Mean**	**Variance**	**Std Dev**
LACUNAR INFARCT	24.0000	3.9867	0.1722	0.4150
MACULAR INFARCT	52.0000	3.6319	0.2962	0.5443
MULTI INFARCT	24.0000	3.5050	0.2170	0.4659

ANOVA

Variation	**SS**	**df**	**MS**	**F statistic**
Between	3.10790	2	1.55395	6.26470
Within	24.06074	97	0.24805	
Total	27.16864	99		

P-Value = 0.00276

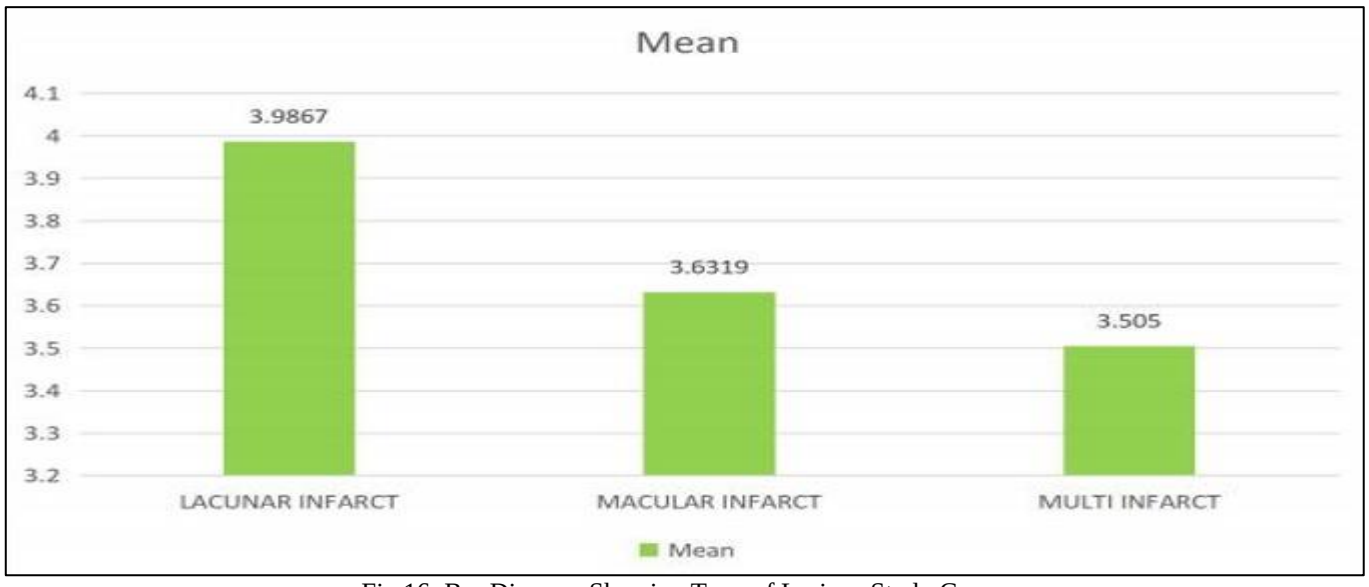

Fig 16: Bar Diagram Showing Type of Lesions Study Group

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➢ *Re Admission:*

There is no significant difference in the mean serum albumin levels among patients who got re admitted and not in the current study.

Table 17: Table Showing Readmission Rates in Current Study Groups

MEANS				
GROUP	**Obs**	**Mean**	**Variance**	**Std Dev**
N	92.0000	3.7163	0.2761	0.5255
Y	8.0000	3.3450	0.1466	0.3829

T-Test

Method	**Variances**	**DF**	**t Value**	**Pr > |t|**
Pooled	Equal	98	1.95	0.0540

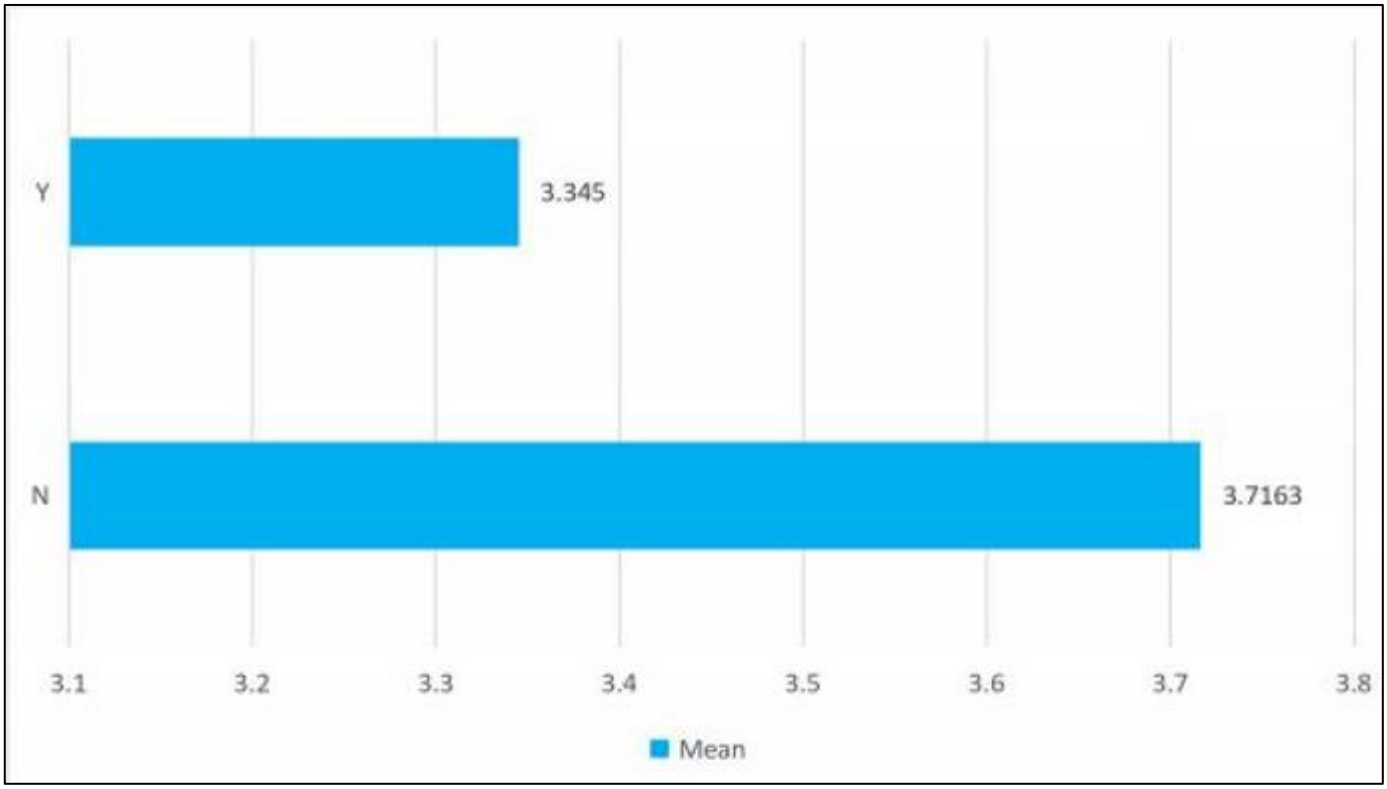

Fig 17: Bar Diagram Showing Readmission Rates in Current Study Groups

➢ *Gender & Serum Albumin:*

There is no significant association between gender & serum albumin in the current study.

Table 18: Table Showing Gender & Serum Albumin Correlation Current Study Groups

MEANS				
GROUP	**Obs**	**Mean**	**Variance**	**Std Dev**
F	31.0000	3.7026	0.2639	0.5138
M	69.0000	3.6794	0.2829	0.5319

T-Test

Method	**Variances**	**DF**	**t Value**	**Pr > |t|**
Pooled	Equal	***98***	0.20	0.8392

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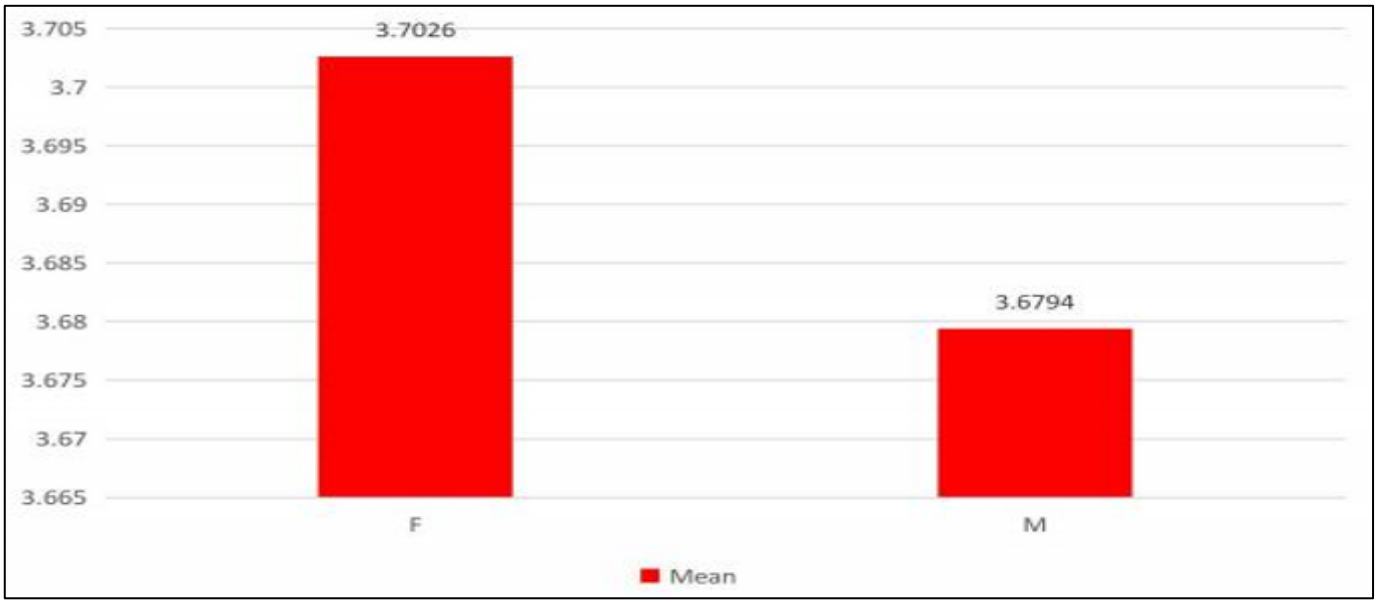

Fig 18: Bar Diagram Showing Gender & Serum Albumin Correlation Current Study Groups

VI. DISCUSSION

This study included 100 patients with acute ischemic stroke admitted at tertiary care center.

Patients who assessed initially and categorized with modified rankin scale and then all parameters were compared between categories.

- *Modified Rank in Scale is as Follows:*

Points	Grade of disability
0	No symptoms
1	No significant disability. Some symptoms but able to carry out all usual activities
2	Slight disability. Able to perform daily activity without assistance, but unable to carry out previous activities.
3	Moderate disability. Requires some help, unable to walk alone without assistance.
4	Moderate severe disability. Needs for assistance for own bodily needs, unable to walk alone without assistance.
5	Severe disability. Unable to attend own body needs without constant assistance, nursing care and attention. Incontinent.
6	Dead.

Image : Modified Rank in Scale

In the current study, patients were divided into 4 groups, as there were no patients with 0 and 1 point.

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➢ *NIHSS Scale*

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NIH STROKE SCALE

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

PATIENT IMPRINT

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

INITIAL	SIGNATURE	INITIAL	SIGNATURE	INITIAL	SIGNATURE

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➢ *Role of Albumin in Stroke:*

Serum albumin levels were also assessed and the prognostic influence of albumin was assessed. Low serum albumin levels indicate <35 mmol/L in the current study.

Albumin induces hemodilution. In one rat model study done on transient focal cerebral ischemia induced by MCA occlusion, administering 5% albumin at the onset of ischemia led to a decrease in ischemic brain damage, evidenced by reduced hematocrit, infarct volume, and cerebral edema.[31]

In human subjects, **Dziedzic et al.** examined 759 patients with acute ischemic stroke. They measured serum albumin levels between 12 and 36 hours after stroke onset and assessed functional outcomes three months later using a modified Rankin scale. Their findings revealed that patients with poorer outcomes had lower serum albumin levels compared to those with better outcomes. Serum albumin level remained an independent predictor of poorer outcomes in logistic regression analysis [32] In the current study also, results showed that low serum albumin was associated with severe stroke and poor outcomes.

Abubakar et al.[33] conducted a study involving 75 acute stroke patients, comparing admission serum albumin levels with 30-day mortality and functional outcomes assessed by the modified Rankin scale. They found that the mean serum albumin level of 3.0 g/dL in patients with favorable outcomes was significantly higher than the 2.0 g/dL observed in those with unfavorable outcomes. Patients who died had lower serum albumin levels, with a mean of 1.66 g/dL, compared to survivors. In the current study also, the mean serum albumin levels were more significant among survivors compared to patients who expired.

Ruiwen che et al- [34] investigated the relationship between serum albumin levels and hemorrhagic transformation in acute stroke patients following intravenous thrombolysis. Data from 428 patients who got intravenous rt-PA treatment between 2013 and 2016 were collected. The patients were split into two groups according to their blood albumin levels: low level and normal level. Data on hemorrhagic transformation (HT) incidence and functional results, as well as demographic, clinical, and laboratory information, were evaluated. Within seven days, HT was verified by CT or MRI, and at seven and ninety days, functional outcomes were assessed using the modified Barthel Index and mRS.

The study found that patients with lower albumin levels had a significantly higher risk of HT and symptomatic intracerebral hemorrhage compared to those with normal albumin levels. Uni variate analysis identified atrial fibrillation and albumin level as significant factors for HT. Multivariate logistic regression analysis, serum albumin level is found to be an independent predictor of HT. There were no significant difference observed in clinical outcomes at 7 and 90 days between the two groups. So, a low serum albumin level within 24 hours may independently predict post-thrombolytic HT.

Davalos et al [35] A research that measured albumin levels in 104 acute stroke patients within 24 hours after the beginning of symptoms found that hypoalbuminemia (<35 g/L) was present in 7.7% of cases. 45.5% of the 705 patients with ischemic and hemorrhagic strokes in another cohort research had hypoalbuminemia. [36]

The variations in study demographics, methods, and blood sample time may contribute to discrepancies in hypoalbuminemia rates between our investigation and earlier research.

Hypoalbuminemia among stroke patients may stem from factors like malnutrition or underlying conditions such as renal or hepatic insufficiency and malignancy. To ensure homogeneity, our study excluded patients with malignant tumors, severe renal or hepatic diseases, and heart failure. Reduced Patients with hypo-albuminemia faced a higher mortality risk six months after their initial stroke onset.

[38]In the current study also, mean albumin levels were less significantly among patients who expired. In the current study, 9% of patients expired.

While preclinical studies support albumin's neuroprotective effects, clinical trials investigating its role in humans have yielded mixed results. **Ginsberg et al.** found no improvement in neurologic function with 25% albumin in ischemic stroke patients. Further research is warranted, particularly focusing on different stroke patient subgroups.[39]

Less is known about the processes by which serum albumin influences the clinical outcomes of people who have had ischemic stroke. On the other hand, a number of pathophysiological routes have been suggested. In big artery occlusion stroke, the length of arterial occlusion and the maintenance of collateral blood flow above the infarction threshold determine the rate of infarct development. As a major modulator of colloidal osmotic pressure, decreased serum albumin levels may decrease collateral flow and worsen the prognosis of large artery occlusive stroke following thrombectomy.[40]

According to experimental research, albumin increases cerebral blood flow via mediating erythrocyte aggregation and lowering hematocrit levels. More neurons could be saved and infarct development could be stopped by this improved blood perfusion. To clarify this possible process, more studies including imaging data—such as infarct development and ultimate infarct volumes—are required.41, 42 Albumin may also help ischemic damage by reducing oxidative stress and inflammatory reactions, preventing platelet aggregation, lowering cytokine adhesion in the post-capillary

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microcirculation, and transferring free fatty acids after ischemia. Furthermore, ischemia-induced disruption of the blood-brain barrier may make it easier for albumin to enter the brain parenchyma, where cortical neurons with typical morphological characteristics may absorb it. Together, these results point to a possible advantage of serum albumin in ischemia damage.[41-46]

Zhou et al-[47] evaluated data from the Third China National Stroke Registry (CNSR-III), classifying patients into four categories according to their admission blood albumin levels. At three months and a year, they evaluated outcomes such as death and poor functional result. The connections were assessed using Cox regression models and logistic regression models, respectively. In the meta-analysis, risk ratios for death were computed using a random-effect model, whereas those for poor functional outcome were computed using a fixed-effect model.

The research covered 13,618 patients. Patients in the <35 g/L group showed higher odds of poor functional outcome and death throughout the 3-month follow-up period than those in the 40–44.9 g/L group. There was a persistent negative correlation between the prognosis and a 10 g/L drop in serum albumin as well. At one year, there was an independent correlation between low blood albumin and clinical outcomes. The meta-analysis found that the pooled HR for death across five trials was 1.07 and the pooled OR for poor functional outcome across three studies per 1 g/L reduction was 1.03.The authors came to the conclusion that patients with acute ischemic stroke (AIS) or transient ischemic attack (TIA) who had low blood albumin levels are more likely to have poor functional outcomes and to die. Serum albumin levels were shown to predict stroke severity and patient death in the current investigation as well.

Gao et al- [48]Data from the Chinese ischemic stroke multicenter registry was used in a research by Gao et al. [(48)]. They concentrated on individuals whose anterior circulation major artery blockage led to endovascular treatment. Serum albumin levels were measured 24 hours after admission. Poor functional result, which was defined as a modified Rankin scale score of 3–6 at the three-month point, was the main outcome that was measured. Three-month mortality and symptomatic intracranial hemorrhage (sICH) were secondary outcomes.

605 patients (mean age: 64 years; 59% male) were included in their analysis. 342 individuals had poor functional results within 3 months after their stroke. Patients in the lowest tertile of serum albumin levels had a significantly higher odds ratio of 2.43 for a poor functional outcome than those in the highest tertile, even after controlling for demographic factors, the National Institutes of Health stroke score, and other potential confounders. There was a linear correlation between low albumin levels and poor functional result, according to restricted cubic spline regression. Analysis of subgroups supported these conclusions.

Serum albumin levels showed a comparable substantial connection with mortality, but not with sICH. The study came to the conclusion that, in the anterior circulation treated with EVT, lower blood albumin levels were independently associated with a worse prognosis ninety days after an acute big artery occlusion stroke.

WANG[49] did a cross-sectional study to know the association between serum albumin levels and stroke risk among adults. They used data from the 2009 to 2018 National Health and Nutrition Examination Survey, involving 17,303 participants aged 40 years or older. A multivariate logistic regression model were employed to explore this relationship, with smoothed curve fitting used to detect potential nonlinear connections. Upon identifying nonlinear associations, the inflection point was determined through a recursive method.

Their analysis showed significant and inverse association between serum albumin levels and stroke risk, even after adjusting for potential variable. Subgroup analysis further demonstrated that this inverse relationship was statistically significant among men, participants under 60 years old, non- diabetic individuals, and those with hypertension. These findings suggest a negative association between serum albumin levels and stroke risk,implying lower levels of serum albumin associated with an increased risk of stroke.

Aptaker et al.[50] conducted a study on the relationship between albumin levels and functional results and medical problems in 79 stroke patients 65 years of age or older. These individuals had a unilateral thrombo-embolic stroke and received inpatient multidisciplinary rehabilitation. The study evaluated modified Barthel index scores at admission and discharge, as well as blood albumin levels at admission. The results imply that serum albumin levels and the course of medical problems as well as functional outcomes in elderly stroke patients are related.

Gariballa et al. [51] demonstrated that low blood albumin levels are a predictor of poor functional outcomes following a stroke. Only blood albumin levels, out of all the nutritional indicators they looked at, showed a meaningful and independent correlation with stroke outcomes.

Babu et al.[52] observed a correlation between poor functional outcomes during a three-month follow-up and low blood albumin levels upon admission. Additionally, they found that individuals who presented with low blood albumin levels had a greater likelihood of stroke recurrence.

Ramesh et al.[53] found that among research participants admitted to neurosurgical intensive care units, serum albumin levels were an independent predictor of survival.

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The current study found that AIS patients were more likely to be male. This might be because of social biases in India, as well as the fact that men are more likely to chew tobacco, smoke, and drink alcohol.

➢ *Comorbidities:*

In this study, diabetes mellitus and hypertension emerged as the most common risk factors, aligning with findings from studies like R James et al. and Kasundra et al. 54

➢ *Clinical Features*

The majority of patients had focal neurological impairments, mostly motor weakness, which were followed by convulsions (9%), headaches (16%), head spinning, impaired sensorium (9%), and slurred speech (18%). According to earlier studies, patients who appear with convulsions typically have poorer outcomes and greater fatality rates.

➢ *Age*

One week after admission, the mean age of patients with poor outcomes was 68 years, which was a statistically significant result. Additionally, the bad result group had lower mean serum albumin levels than the excellent outcome group, which is consistent with findings from the **Reinhardt et al**.[55] research. This contrasted with the results of the current study, which show no correlation between age and stroke severity.

➢ *Mortality:*

The study's mortality rate, at 9%, aligns with rates reported in other studies. Patients with low NIHSS and mRS scores and higher serum albumin levels tended to have better outcomes, indicating a significant correlation between ischemic stroke severity at presentation and serum albumin levels.

Serum albumin levels can be lowered by conditions such as liver disease, renal illness, and starvation. Changes in blood albumin levels after a stroke are also influenced by the neuroendocrine response and catabolic state. Albumin production may be impacted by malnutrition's downregulation of protein synthesis. Thus, measured blood albumin levels indicate the patients' nutritional condition and may be associated with increased post-stroke morbidity and death.

VII. SUMMARY

- 35% of the patients had moderate disability, 26% had mild disability and 9% died. This classification is as per modified ranking scale
- There is no significant difference in mean age in between groups of patients.
- There is no significant association between gender and groups of patients. Overall 69 patients were males.
- No significant relation between smoking and severeness of stroke or severity of disability as per mRS. Overall 26% of patients were smokers.
- There is no significant association between alcoholism and severity of stroke or severity of disability as per mRS. Overall 20% of patients were smokers.
- There is no significant association between presence of hypertension and severity of stroke or severity of disability as per mRS. Overall 20% of patients were hypertensives.
- There is no significant association between presence of diabetes and severity of stroke or severity of disability as per mRS. Overall 23% of patients were diabetic patients.
- There is no significant association between presence of coronary artery disease and severity of stroke or severity of disability as per mRS. Overall 11% of patients had CAD.
- There is no significant association between presence of dyslipidemia and severity of stroke or severity of disability as per mRS. Overall 18% of patients were hypertensives.
- There is significant difference in mean GCS scale in between groups of patients. GCS score was less in patients who died or having severe stroke as per mRS scale.
- 31% of the patients had belonged to severe GCS scale category.(score below 9)
- 26% of the patients belonged to severe SSS category.
- 24% of the patients had multiple infarcts. 24% had lacunar infarct and 52% had macular infarct.
- There is significant difference in mean GCS score & serum albumin. Mean serum albumin was less in patients with severe GCS category
- There is significant association between mean SSS and serum albumin.
- There is significant difference in mean serum albumin between various types of lesions. Mean serum albumin was less in patients with multiple infarcts.
- There is no significant difference in the mean serum albumin levels among patients who got re admitted and not n the current study.There is no significant association between gender & serum albumin.

VIII. CONCLUSION

The present study showed that predictive role of serum albumin levels among patients with acute ischemic stroke. There was significant association between serum albumin levels and outcomes like duration of hospitalization and severity of stroke.

Due to the high prevalence of hypo-albuminemia in patients with ischemic stroke, it can be considered as a risk factor for ischemic stroke.

We recommend to check serum albumin levels serially to monitor patients health condition.

- Sponsor: None
- Conflicts of interest: Nil

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ANNEXURES
WRITTEN INFORMED CONSENT FORM

S.NO: ……..…

Date: ……..

NAME: ……..

Age: ……..

OP/ IP No.: …

I, _____ hereby give my consent to participate in this study titled **"A STUDY OF SERUM ALBUMIN LEVEL AS A PROGNOSTIC INDICATOR OF ACUTE ISCHEMIC STROKE"**

The investigator explained to me the aim and objectives of the study and I had the opportunity to ask questions on the subject.

- I agree to undergo the relevant examination and testing as explained.
- I had been told that there are no blood tests, or any laboratory investigations required or administration of any medicines in this study.
- My Personal details will be kept as secret and will not be revealed to any other person without my written informed consent.

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

I … … … … … … … … ... willingly and voluntarily give this consent without undue pressure.

Signature of Participant Signature of Witness

Contact No:

Date:

Help Line: In case of any problem, you can contact Dr. VAKA ANUHYA VIDYA DEVI M.B.B.S on phone no-: 8297840203. You will receive prompt and appropriate medical attention.

Additional information:

You are entitled to ask questions at any time during your participation in the study. A copy of the agreement will be given for your records.

Responsibility:

In case of any loss, the investigator will bear compensation.

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INFORMED CONSENT ACCEPTANCE SHEET

Patient I.D. No.

Title of the Project: "AN INVESTIGATION OF SERUM ALBUMIN LEVELS AS A PREDICTOR OF ACUTE ISCHEMIC STROKE".

Principal Investigator Name: Dr.P.Rajendra kumar

Contact No: Information Sheet: I have read the information sheet carefully/ explained in the languageI understand, and I fully understand the contents. I confirm that I have had an opportunity toaskquestions. I have been explained in detail about the nature and purpose of the study anditspotential risks/benefits, and the expected duration of the study and other related detailsof thestudy.

I understand that my participation is voluntary and i am free to withdrawat any timewithout giving any reason, without any reason, without any reason, my medical care andlegal rights.

I agree to participate in the study. .....

(signature / left thumb impression)

Date:

Place:

Name of the participant: .....

Son/ Daughter/ Spouse of: .....

Complete postal address: .....

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

(Signature of principal investigator)

Date: .....

Place: .....

1) witness- I.....

2)Witness II .....

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PROFORMA

- WEAKNESS
- GIDDINESS
- SEIZURE
- UNRESPONSIVENESS
- UNSTEADINESS OF GAIT
- DEVIATION OF ANGLE OF MOUTHDURATION OF SYMPTOMS: H/O FEVER/TRAUMA PAST HISTORY
- PRIOR STROKE
- DIABETES MELLITUS
- HYPERTENSION
- CARDIAC DISEASES
- EPILEPSY
- TUBERCULOSIS TREATMENT HISTORY:
- FAMILY HISTORY:
- ADDICTIONS:
- VITALS:
- PULSE-RATE AND RHYTHM
- BLOOD PRESSURE
- RESPIRATORY RATE
- TEMPERATURE GENERAL EXAMINATION SYSTEMIC EXAMINATION
- CENTRAL NERVOUS SYSTEM
- HIGHER MENTAL FUNCTION: CONSCIOUSNESS ORIENTATION
- SPEECH
- CRANIAL NERVES
- MOTOR SYSTEM: TONE POWER REFLEXES
- SENSORY SYSTEM
- CEREBELLAR SIGNS
- MENINGEAL SIGNS
- GLASGOW COMA SCALE- E V MTOTAL SCORE: /15
- SCANDINAVIAN STROKE SCALE SCORE: CARDIOVASCULAR SYSTEM

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- APEX BEAT
- HEART SOUNDS
- MURMURS RESPIRATORY SYSTEM
- BREATH SOUNDS
- ADDED SOUNDS GASTROINTESTINAL SYSTEM
- ANY TENDERNESS
- LIVER SPAN INVESTIGATIONS
- SHIFTING DULLNESS

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KEY TO MASTER CHART

Y: YES

N: NO

MASTER CHART

S NO	DISABILITY	AGE	GENDER	SMOKING	ALCOHOLISM	HTN	DM	CAD	DYSLIPIDEMIA
1	MILD	59	F	N	N	N	N	N	N
2	MILD	53	M	N	N	N	N	N	N
3	MILD	63	M	N	N	N	N	Y	N
4	MILD	58	M	Y	Y	N	N	N	N
5	MILD	67	M	N	N	Y	N	N	N
6	MILD	61	M	N	Y	N	N	N	Y
7	MILD	54	M	N	N	N	Y	N	N
8	MILD	63	M	N	N	Y	N	Y	N
9	MILD	47	F	Y	N	N	N	N	N
10	MILD	51	M	N	N	N	N	N	N
11	MILD	70	M	N	N	N	N	N	Y
12	MILD	51	F	N	N	N	N	N	N
13	MILD	53	M	N	N	Y	N	N	N
14	MILD	51	M	Y	N	N	N	N	N
15	MILD	55	M	N	N	N	N	Y	N
16	MILD	50	F	N	Y	N	Y	N	N
17	MILD	48	M	N	N	Y	N	N	N
18	MILD	49	F	N	N	N	N	N	Y
19	MILD	48	F	N	N	N	N	N	N
20	MILD	58	M	N	N	N	N	Y	N
21	MILD	59	M	N	N	N	Y	N	N
22	MILD	59	F	N	N	N	N	N	N
23	MILD	44	M	N	N	N	N	N	Y
24	MILD	47	M	Y	N	N	N	N	N
25	MILD	68	M	N	N	N	N	N	N
26	MILD	59	M	N	Y	N	N	N	Y
27	MODERATE	43	F	N	N	N	N	N	N
28	MODERATE	58	M	N	N	Y	Y	N	N
29	MODERATE	45	M	N	N	N	N	N	N
30	MODERATE	50	M	N	N	N	N	Y	Y
31	MODERATE	53	F	Y	N	Y	N	N	N
32	MODERATE	54	F	N	N	N	N	N	N
33	MODERATE	57	M	N	N	N	N	N	N
34	MODERATE	46	M	Y	N	N	N	N	N
35	MODERATE	41	M	N	N	N	N	N	N
36	MODERATE	60	F	Y	N	N	N	N	N
37	MODERATE	61	M	N	Y	N	N	N	N
38	MODERATE	69	M	N	N	Y	N	N	N
39	MODERATE	59	M	N	N	N	N	N	N
40	MODERATE	45	M	Y	Y	N	Y	N	N
41	MODERATE	55	F	N	N	N	N	N	N
42	MODERATE	46	F	N	N	N	Y	N	N
43	MODERATE	62	M	Y	N	Y	Y	N	N
44	MODERATE	52	M	N	N	N	N	N	N
45	MODERATE	42	F	N	Y	N	N	N	N
46	MODERATE	53	M	Y	Y	Y	N	N	N
47	MODERATE	46	F	N	N	N	Y	N	N
48	MODERATE	57	M	N	N	N	N	N	N
49	MODERATE	56	F	Y	N	N	N	N	N

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50	MODERATE	69	F	N	N	Y	N	N	N
51	MODERATE	66	M	N	N	N	Y	N	Y
52	MODERATE	55	M	Y	N	N	N	N	N
53	MODERATE	51	M	N	Y	N	Y	N	Y
54	MODERATE	54	M	N	N	N	N	N	N
55	MODERATE	52	F	N	N	N	N	N	N
56	MODERATE	58	F	N	N	N	N	N	N
57	MODERATE	41	M	N	N	Y	N	N	N
58	MODERATE	61	M	N	N	N	N	N	Y
59	MODERATE	52	M	Y	N	N	Y	N	N
60	MODERATE	45	F	N	N	N	N	N	N
61	MODERATE	46	F	Y	N	N	N	N	Y
62	SEVERE	59	F	N	N	N	N	N	N
63	SEVERE	50	M	N	Y	N	N	N	Y
64	SEVERE	57	M	N	N	Y	N	N	N
65	SEVERE	58	F	N	Y	N	N	N	Y
66	SEVERE	48	M	Y	N	N	N	N	N
67	SEVERE	44	M	N	N	N	N	N	N
68	SEVERE	53	M	Y	N	N	N	N	N
69	SEVERE	42	M	N	N	Y	Y	N	N
70	SEVERE	56	F	N	Y	N	N	N	Y
71	SEVERE	59	F	N	N	N	N	N	N
72	SEVERE	41	M	N	N	N	N	N	N
73	SEVERE	59	M	N	N	N	N	Y	N
74	SEVERE	48	M	N	N	N	Y	N	N
75	SEVERE	41	F	N	Y	N	N	N	N
76	SEVERE	58	M	Y	N	N	N	N	N
77	SEVERE	66	M	N	N	N	N	N	N
78	SEVERE	42	M	N	N	N	N	N	Y
79	SEVERE	46	M	N	N	N	N	Y	N
80	SEVERE	51	M	Y	N	Y	N	N	N
81	SEVERE	66	M	N	Y	N	Y	N	N
82	SEVERE	50	F	N	N	Y	N	N	Y
83	SEVERE	43	M	Y	Y	N	Y	N	N
84	SEVERE	56	M	N	N	N	N	Y	N
85	SEVERE	63	F	N	N	Y	Y	N	N
86	SEVERE	42	M	N	N	N	N	N	Y
87	SEVERE	68	M	Y	Y	N	N	N	N
88	SEVERE	59	M	N	N	Y	N	N	N
89	SEVERE	57	M	Y	N	N	N	Y	N
90	SEVERE	42	M	Y	Y	Y	Y	N	Y
91	SEVERE	49	M	N	Y	N	Y	N	N
92	DEATH	42	F	N	N	Y	N	Y	N
93	DEATH	45	M	Y	N	N	N	N	N
94	DEATH	62	M	Y	Y	N	N	N	N
95	DEATH	47	M	N	N	N	Y	N	N
96	DEATH	69	M	N	N	Y	Y	N	Y
97	DEATH	50	M	Y	N	N	Y	N	N
98	DEATH	43	F	Y	N	N	N	N	N
99	DEATH	63	M	N	Y	N	Y	N	N
100	DEATH	63	F	N	N	N	Y	Y	N

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GCS SCALE	GCS SCALE CAT	SSS	SSS CAT	TYPE OF LESION	DURATION OF HOSPITALIZATION	RE ADMISSION	SERUM ALBUMIN
12	MILD	58	MILD	LACUNAR INFARCT	10	N	4.28
14	MILD	53	MILD	MACULAR INFARCT	8	N	4.22
14	MILD	44	MILD	LACUNAR INFARCT	9	N	4.23
13	MILD	52	MILD	LACUNAR INFARCT	3	N	4.26
15	MILD	34	MODERATE	LACUNAR INFARCT	10	N	4.23
13	MILD	46	MILD	MULTI INFARCT	4	N	4.28
12	MILD	52	MILD	MACULAR INFARCT	4	N	4.21
9	MODERATE	49	MILD	MACULAR INFARCT	5	N	4.3
13	MILD	57	MILD	MACULAR INFARCT	9	N	4.22
15	MILD	47	MILD	MACULAR INFARCT	7	N	4.25
12	MILD	48	MILD	LACUNAR INFARCT	5	N	4.3
13	MILD	51	MILD	MULTI INFARCT	9	N	4.21
14	MILD	56	MILD	MACULAR INFARCT	8	N	4.21
8	MODERATE	47	MILD	MACULAR INFARCT	9	N	4.25
12	MILD	47	MILD	LACUNAR INFARCT	3	N	4.2
13	MILD	56	MILD	MACULAR INFARCT	3	N	4.24
15	MILD	49	MILD	LACUNAR INFARCT	10	N	4.2
13	MILD	49	MILD	MULTI INFARCT	6	N	4.2
15	MILD	44	MILD	MACULAR INFARCT	3	N	4.21
13	MILD	48	MILD	MACULAR INFARCT	10	N	4.29
6	SEVERE	58	MILD	LACUNAR INFARCT	6	N	4.23
13	MILD	57	MILD	MACULAR INFARCT	5	N	4.3
14	MILD	33	MODERATE	LACUNAR INFARCT	8	N	4.2
13	MILD	46	MILD	LACUNAR INFARCT	4	N	4.2
15	MILD	44	MILD	LACUNAR INFARCT	5	N	4.24
14	MILD	55	MILD	LACUNAR INFARCT	10	Y	4.2
14	MILD	56	MILD	MACULAR INFARCT	10	N	4.27
13	MILD	45	MILD	MACULAR INFARCT	9	N	4.26
12	MILD	43	MILD	MACULAR INFARCT	7	N	4.3

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14	MILD	48	MILD	MACULAR INFARCT	5	N	4.23
12	MILD	54	MILD	LACUNAR INFARCT	10	N	4.02
12	MILD	47	MILD	MACULAR INFARCT	9	N	4.04
15	MILD	52	MILD	LACUNAR INFARCT	5	N	4.07
15	MILD	33	MODERATE	MULTI INFARCT	3	N	4.09
10	MODERATE	49	MILD	MACULAR INFARCT	8	N	4.06
15	MILD	43	MILD	LACUNAR INFARCT	8	N	4.09
15	MILD	57	MILD	MACULAR INFARCT	10	N	4.08
12	MILD	47	MILD	LACUNAR INFARCT	3	N	4.03
14	MILD	30	MILD	MULTI INFARCT	10	N	4.01
14	MILD	47	MILD	MACULAR INFARCT	9	N	4.08
13	MILD	46	MILD	MULTI INFARCT	3	N	4.08
10	MODERATE	29	MODERATE	LACUNAR INFARCT	5	N	4.1
10	MODERATE	50	MILD	MACULAR INFARCT	5	N	4.02
11	MODERATE	44	MILD	MULTI INFARCT	7	N	4.05
11	MODERATE	24	SEVERE	MACULAR INFARCT	9	N	4.05
11	MODERATE	48	MILD	LACUNAR INFARCT	8	N	4.05
11	MODERATE	45	MILD	MULTI INFARCT	10	N	4.01
10	MODERATE	48	MILD	MACULAR INFARCT	7	N	4.01
10	MODERATE	50	MILD	LACUNAR INFARCT	8	N	4.05
10	MODERATE	27	MODERATE	MACULAR INFARCT	7	N	4.01
12	MODERATE	45	MILD	MACULAR INFARCT	10	N	4.05
9	SEVERE	53	MILD	MACULAR INFARCT	4	N	4.02
12	MODERATE	46	MILD	LACUNAR INFARCT	5	N	4.07
9	MODERATE	47	MILD	MACULAR INFARCT	6	N	3.27
10	MODERATE	51	MILD	MULTI INFARCT	5	N	3.29
10	MODERATE	56	MILD	MULTI INFARCT	6	N	3.3
9	MODERATE	31	MODERATE	MULTI INFARCT	10	N	3.2
11	MODERATE	32	MODERATE	MACULAR INFARCT	9	N	3.32
12	MODERATE	46	MILD	MULTI INFARCT	5	N	3.23

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10	MODERATE	49	MILD	MACULAR INFARCT	6	N	3.29
6	SEVERE	55	MILD	MACULAR INFARCT	4	Y	3.34
9	MODERATE	58	MILD	MACULAR INFARCT	4	N	3.26
11	MODERATE	40	MODERATE	LACUNAR INFARCT	3	N	3.2
10	MODERATE	55	MILD	MULTI INFARCT	10	N	3.33
12	MODERATE	47	MILD	MULTI INFARCT	6	N	3.27
11	MODERATE	55	MILD	MACULAR INFARCT	7	Y	3.25
9	MODERATE	41	MODERATE	MACULAR INFARCT	7	N	3.31
12	MODERATE	57	MILD	MACULAR INFARCT	6	N	3.3
8	SEVERE	56	MILD	MULTI INFARCT	6	N	3.25
11	MODERATE	56	MILD	MACULAR INFARCT	7	N	3.32
8	SEVERE	46	MILD	MULTI INFARCT	9	Y	3.24
6	SEVERE	52	MILD	MULTI INFARCT	4	N	3.29
6	SEVERE	56	MILD	MACULAR INFARCT	13	Y	3.32
8	SEVERE	15	SEVERE	MULTI INFARCT	13	N	3.25
9	SEVERE	1	SEVERE	MACULAR INFARCT	7	N	3.3
6	SEVERE	17	SEVERE	MACULAR INFARCT	13	Y	3.25
13	MILD	18	SEVERE	MACULAR INFARCT	13	N	3.31
6	SEVERE	1	SEVERE	LACUNAR INFARCT	12	N	3.31
7	SEVERE	12	SEVERE	MULTI INFARCT	12	N	3.31
7	SEVERE	17	SEVERE	MACULAR INFARCT	10	N	3.3
14	MILD	19	SEVERE	MACULAR INFARCT	13	N	3.3
9	SEVERE	0	SEVERE	MULTI INFARCT	9	Y	3.33
9	SEVERE	7	SEVERE	MULTI INFARCT	7	N	3.31
6	SEVERE	12	SEVERE	MACULAR INFARCT	10	N	3.26
7	SEVERE	1	SEVERE	MACULAR INFARCT	5	N	2.92
9	SEVERE	15	SEVERE	MACULAR INFARCT	12	N	2.92
7	SEVERE	10	SEVERE	MACULAR INFARCT	6	N	2.83
6	SEVERE	14	SEVERE	MACULAR INFARCT	8	Y	2.83
6	SEVERE	18	SEVERE	MACULAR INFARCT	4	N	2.94

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6	SEVERE	9	SEVERE	MACULAR INFARCT	13	N	2.98
8	SEVERE	45	MODERATE	MACULAR INFARCT	13	N	2.96
7	SEVERE	5	SEVERE	MACULAR INFARCT	7	N	2.8
9	SEVERE	4	SEVERE	MULTI INFARCT	12	N	2.86
8	SEVERE	1	SEVERE	MACULAR INFARCT	11	N	2.94
13	MILD	6	SEVERE	LACUNAR INFARCT	9	N	2.97
8	SEVERE	34	MODERATE	MACULAR INFARCT	12	N	2.99
6	SEVERE	12	SEVERE	LACUNAR INFARCT	4	N	2.95
7	SEVERE	3	SEVERE	MULTI INFARCT	11	N	2.87
9	SEVERE	12	SEVERE	MULTI INFARCT	6	N	2.86
8	SEVERE	17	SEVERE	MACULAR INFARCT	6	N	2.87