"A CROSS-SECTIONAL STUDY TO DETERMINE THE SHORT TERM OUTCOME OF CEREBRAL SALT WASTING SYNDROME AND SYNDROME OF INAPPROPRIATE ANTIDIURETIC HORMONE SECRETION IN STROKE PATIENTS"

By Dr. KAVYA B K



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

In partial fulfillment of the requirements for the degree of DOCTOR OF MEDICINE

IN

GENERAL MEDICINE

Under the Guidance of Dr. VIDYASAGAR C R
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JUNE 2023

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ABSTRACT

A CROSS-SECTIONAL STUDY TO DETERMINE THE SHORT TERM OUTCOME OF CEREBRAL SALT WASTING SYNDROME AND SYNDROME OF INAPPROPRIATE ANTIDIURETIC HORMONE SECRETION IN STROKE PATIENTS

Introduction: Hyponatremia is common in patients with stroke. Mortality rates in hyponatraemic stroke patients have been reported to be as larger as 60%. The causes of hyponatremia are varied but are most commonly attributed to the syndrome of inappropriate anti-diuresis, and cerebral salt wasting syndrome Differentiation between SIADH and CSW is important because treatment of one may be hazardous to the other.

Objectives:

- **1.** To measure the magnitude of hyponatremia in acute stroke.
- 2. To measure the frequency of occurrence of Syndrome of Inappropriate Anti-Diuretic Hormone Secretion and cerebral salt wasting syndrome in patients with hyponatremia in Acute Stroke.
- **3.** To compare short-term outcomes in patients with SIADH and Cerebral Salt Wasting syndrome.

Methodology: A cross sectional study was conducted with the minumun sample size of 102. Purposive sampling was conducted among the patients with stroke who had hyponatremia in Sri Devraj Urs Medical College and Hospital between January 2021- May 2022. After taking consent data collection was done using pretested proforma. Microsoft Excel software was used for the analysis. The level of significance[α] was 5% i.e., p-value <0.05 is considered statistically significant.

Results: 60.8 % of the patients were males. 70 % had ischemic stroke. 74.5% of the stroke patients had SIADH, while 25.5% of them had CSW. 56.6% of the stroke patients improved in the group of SIADH while only 19.2% improved among CSW. There is an association

between SIADH and CSW and the improvement of the patients. This association is statistically significant with p value = 0.001. Odd's Ratio for Improvement among SIADH is 1.466.

Conclusion: Hyponatremia should be evaluated in all the acute stroke patients. Reversal of severity of the stroke, survival of the patients is better in patients with SIADH than with patients with CSW

Keywords: Stroke, Hyponatremia, CSW, SIADH, NIHSS score

ABBREVIATIONS

ADH ANTI-DIURETIC HORMONE

ANP ATRIAL NATRIURETIC PEPTIDE

AVP ARGININE VASPRESSIN HORMONE

BUN BLOOD UREA NITROGEN

CSW CEREBRAL SALT WASTING

CVA CEREBROVASCULAR ACCIDENT

ECF EXTRACELLULAR FLUID

EABV EFFECTIVE ARTERIAL BLOOD VOLUME

ICF INTRACELLULAR FLUID

K POTASSIUM

mRS MODIFIED RANKLIN SCALE

Na SODIUM

NaCl SODIUM CHLORIDE

NATIONAL INSTITUTE OF HEALTH STROKE

NIHSS

SCALE

SYNDROME OF INAPPROPRIATE ANTI-

SIADH

DIURETIC HORMONE

SAH SUBARACHNOID HEMORRHAGE

TBW TOTAL BODY WATER

TSH THYROID STIMULATING HORMONE

< LESS THAN

> >	MORE THAN MORE THAN EQUAL TO
%	PERCENTAGE
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INTRODUCTION

INTRODUCTION

Stroke being a non-communicable disease lead to significant disability and loss of quality of life is the second cause of death after ischemic heart disease and the leading cause of disability worldwide. Stroke has also made significant contributions to the economy and society's burden on patients and their families. In all adulthood ${\bf s}$

obvious stroke neuropathy in terms of frequency. According to WHO, about 15 million people have a stroke worldwide each year. Of these, 5 million died and 5 million were permanently disabled. Electrolyte abnormalities are common in acute stroke cases. [1]

Hyponatremia is common in patients with stroke. The incidence of hyponatremia in stroke has been reported to range from 11% to 35% in the literature. Mortality rates in hyponatraemic stroke patients have been reported to be as more significant as 60%. The numerous underlying causes warrant a careful differential diagnosis, considering comorbidities, medications, outcome of clinical examination and management .[2]

The causes of hyponatremia are varied but are most commonly attributed to the SIADH and CSW. Inappropriate use of hypotonic solutions, poor fluid intake, infections and mannitol may also reduce sodium levels in acute stroke patients.

Sodium levels improve outcomes in patients with stroke and volume repletion is still the cornerstone of hypovolemic hyponatremia treatment. In cases of normal and elevated hyponatremia, except for correcting the underlying cause (e.g., stopping the irritating medication), fluid restriction, using hypertonic saline, loop diuretics and vaptans are among the treatment options. [3]

CSW is a volume-depleted state which occurs due to decreased sympathetic outflow and increased natriuretic peptides. So treatment includes volume replacement with isotonic saline but in severe cases hypertonic saline.

Hyponatremia especially cerebral salt wasting has been shown to worsen the prognosis of stroke, mortality and cause a poorer discharge disposition.

SIADH is a volume-expanded state due to inappropriate anti-diuresis causing euvolemic hyponatremia. In stroke, SIADH occurs due to anti-diuretic hormone secretion inappropriate to the osmotic threshold. The suppressed proximal convoluted tubule transport can cause hypouricemia and bicarbonate. The treatment include fluid restriction.

SIADH and CSW are two potential causes of hyponatremia in patients with Stroke. The main difference lies in the evaluation of EABV. SIADH is a euvolemic state due to renal water retention mediated by antidiuretic hormone. CSW is featured by EABV contracted by renal salt loss. Appropriate salt replacement is needed in patients with CSW while the fluid restriction is the treatment in SIADH. The difference between these two disorders is of particular importance as the treatment indicated for one disease but used in another can lead to adverse outcome. [4]

Differentiation between SIADH and CSW is important because treatment of one may be hazardous to the other. Hyponatremia is prevalent in acute stroke patients and is independently associated with higher mortality. In hospitalized acute stroke patients, persistent hyponatremia is associated with worse functional outcome[5]

There is a lack of prospective studies that evaluate the frequency and causes of hyponatremia in patients with stroke and the effect on short-term outcome.

AIMS & OBJECTIVES

AIMS AND OBJECTIVES

- 1. To measure the magnitude of hyponatremia in acute stroke.
- 2. To measure the frequency of occurrence of SIADH and CSW syndrome in patients with hyponatremia in Acute Stroke.
- 3. To compare short-term outcomes in patients with SIADH and CSW syndrome

REVIEW OF LITERATURE

REVIEW OF LITERATURE

The review of the literature of the current study will be presented under the following side headings:

- 1. Hyponatremia in Stroke
- 2. SIADH and CSW
- 3. Articles related to the studies

1. Hyponatremia in Stroke

Pathogenesis:

ADH is synthesized in hypothalamus and stored in posterior pituitary gland. Particles until their release is by osmosis or non-osmotic stimulation. Increased serum osmolality and effective hypovolemia represent the main cause stimulating ADH secretion. Its release can also be influenced by factors including nausea, stress (pain) and medications. ADH promotes reabsorption of water in the renal cortex and medullary collecting ducts .It plays a vital role in the mechanism of hyponatremia. Water retention causing hyponatremia occurs only when there is impaired renal excretion of water, this does not occur in patients with primary polydipsia. Considering that inhibition of ADH secretion is required to excrete any amount of water, the presence of abnormally high serum ADH levels in relationship with low plasma osmolality should be considered as an essential criteria to evaluate the etiology of hyponatremia. Most causes of hyponatremia are associated with an absolute or relative excess of ADH (despite the presence of hypotonicity) is mainly caused by SIADH or effective arterial blood volume depletion.

Incidence:

Figure 3.1: Summary of the Incidence of Hyponatremia in stroke.

Study	Design	Sample	Definition of hyponatremia	Prevalence of hyponatremia, %	Associations of hyponatremia
Ischemic stroke Fofi et al. [4], 2012	Prospective	n = 475; 53.9% male; mean age 67.0 years	Na <136 mmol/L	6.3	Increased in-hospital mortality
Huang et al. [5], 2012	Prospective	n = 925; 52.5% male; mean age 69.5 years	Na <135 mmol/L	11.6	Higher 3-year mortality (but not short-term mortality within 3 months)
Rodrigues et al. [6], 2014	Retrospective	n = 3,585; 49.6% male; mean age 71.0 years	Na <135 mmol/L	16.0	Higher admission NIHSS values, lower admission mBI values, worse disposition at discharge from hospital, and higher 3- and 12-month mortality
Lasek-Bal et al. [7], 2014	Prospective	n = 464; 46.1% male; mean age 70.4 years	Na <136 mmol/L	18.9	Higher mortality within 1 month, more severe neurological patients' state in both the acute and subacute phases of stroke
Bei et al. [8], 2017	Prospective	n = 3,314; 57.7% male; mean age 68.6 years	Na <135 mmol/L	3.9	Higher admission mRS and NIHSS scores No association with in-hospital mortality
Gao et al. [9], 2018	Retrospective	n = 718; 21.4% male; median age 73 years	Na <135 mmol/L	15.2	Association with 1-month mortality only in univariate analysis
Acute ischemic	or hemorrhagic str	oke			
Soiza et al [10], 2015	Retrospective	n = 8,540; 47.4% male; mean age 77.3 years	Na <135 mmol/L	13.8	Higher mortality (within 1 week – 11 years) in patients <75 years
Hemorrhagic str	roke				
Kuramatsu et al. [11], 2014	Retrospective	n = 464; 45.0% male; mean age 69.6 years	Na <135 mmol/L	15.6	Increased in-hospital mortality and within 90 days of index stroke; higher admission NIHSS values; lower Glasgow Coma Scale
Gray et al. [12], 2014	Retrospective	n = 99; 61.6% males; mean age 58 years	Na <135 mmol/L	24	Increased in-hospital complications (fever, infection and a longer hospitalization); no association with in-hospital mortality
Carcel et al. [13], 2016	Retrospective analysis of a randomized trial	n = 3,002; 62.8% males; 64±13 years	Na <135 mmol/L	12	Increased mortality within 3 months; larger baseline intracerebral hemorrhage volume

 $NIHSS, National\ Institutes\ of\ Health\ Stroke\ Scale; mBI,\ modified\ Barthel\ Index; mRS,\ modified\ Rankin\ Scale.$

ETIOLOGY OF HYPONATREMIA IN STROKE PATIENTS

	ETIOLOGY	MECHANISM	
STROKE RELATED CAUSES	SIADH	INCREASED HYPOTHALAMIC ADH PRODUCTION	
	CSW	INAPPROPRIATE RENAL SODIUM LOSS	
		- IMPAIRED SYMPATHETIC NEURAL INPUT	
		- INCREASED BNP LEVELS	
	SECONDARY ADRENAL	PITUTARY ISCHEMIA	
	INSUFFICIENCY	PITUTARY HEMORRHAGE	
NON-STROKE RELATED	COMORBIDITIES	DIABETES MELLITUS	
		CHRONIC RENAL FAILURE	
		CONGESTIVE HEART FAILURE	
	EXCESS INTRAVENOUS	HYPOTONIC FLUIDS	
	FLUIDS		
	INFECTIONS	ASPIRATION PNEUMONIA	
		LUNG ABSCESS	
		BRONCHIECTASIS	
		ASPERGILLOSIS	
DRUG INDUCED	MANNITOL	- Thiazide, Loop, Potassium sparing diuretics	
HYPONATREMIA	DIURETICS	- Tricyclic	
	ANTIDEPRESSANTS	antidepressants, Monoaminooxidase	
	BENZODIAZEPINES	inhibitors, SSRI ,SNRI	
	ANTIPSYCHOTICS	- Carbamazepine , Oxcarbamazepine, sodium	
	ANTICONVULSANTS	valproate, lamotrigine, levetericetam,	
	NSAIDs	gabapentin, phenytoin, topiramate	
	PROTON PUMP INHIBIORS	- Ciprofloxacin . cotrimoxazole	
	ANTIBIOTICS	- Amiodarone, propafenone	
	ANTIARRHYTHMICS		

44

Evaluation and treatment of hyponatremia in stroke:

- 1. Pearl of diagnostic method
 - "Pseudohyponatremia" should be excluded. Hyponatremia ("false" refers to non-hypotonic hyponatremia with normal plasma osmolality [P osm 275–290 m
 Osmol/kg]) Hyperproteinemia, Hypertriglyceridemia
- In the presence of osmotic active mannitol or glucose serum osmolality is normal or increased
- 3. Approach to management
 - Discontinue hypotonic medication or solution
 - Treatment of underlying cause (eg, hyperglycemia, infection)
 - Acute hyponatremia (defined within <48 hours) and severe symptomatic hyponatremia: 3% NORMAL SALINE
 - Volumetric hyponatremia: Fluid restriction and 3% saline
 - Hypervolemic hyponatremia: Diuretics
 - Hypovolemic hyponatremia: Isotonic saline

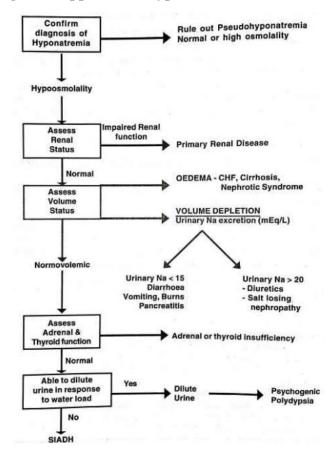


Figure 3.2: Diagnostic approach to hyponatremia.

2. SIADH and CSW

Amongst the electrolyte imbalances, hyponatremia is the commonest. SIADH is characterized by hyponatremia secondary to improperly concentrated urine, Abnormal or Slightly Elevated Concentrations, and Symptoms of raised extravascular volume. On the other hand, in such patients' Intracranial disease develops hyponatremia Similar properties but different in that they have clinical properties: Detection of extracellular fluid (ECF) contraction volume. CSW syndrome was first introduced in a 1950 report by Peters and his colleagues' These results were subsequently confirmed in a wide range of additional patients. Clinical Division of CSW very rare disease or a misnomer for SIADH. CSW was considered a separate entity in the recent years. This Recognition was particularly impressive in the following areas: Neurosurgery.

SIADH:

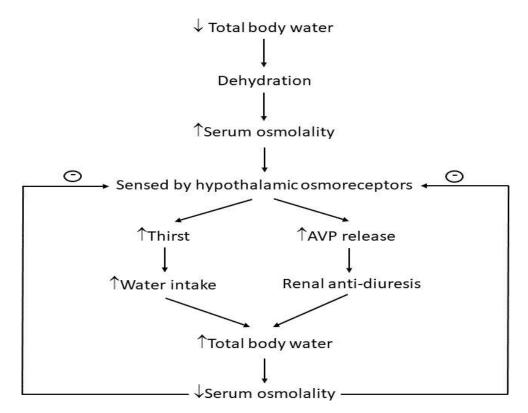
It is a volume expansion state where major pathogenesis include excessive release of ADH causing renal water reabsorption and consequent ECF volume expansion. Proof of volumetric expansion SIADH was originally born from research on healthy subjects. Exogenous pitrescine was administered. In these experiments administration of pitrescine led to a sharp decrease in volume and osmolarity of urine causing water retention due to this antidiuretic effect leading to weight gain and loss in serum Na concentration. A few days later, with pitrescine administration, a rapid increase in urinary sodium and chloride excretion was measured. Incremental expansion of reflected total ECF volume by gaining weight. if hydration is maintained Underweight, body weight during pitrescine administration changes and urinary electrolyte excretion does not increase. A case reported in two patients where fluid intake is not restricted and weight is tolerated during vasopressin administration increases and eventually reaches a steady state, sodium excretion in urine stabilizes and equals her Na in diet recording. Substances such as uric acid and BUN which are absorbed proximally also tend to decrease for the following reasons—proximal absorption.

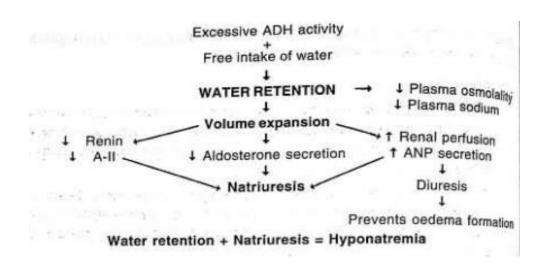
Pathophysiology of SIADH:

In SIADH, the increase in ADH secretion is independent of normal. Osmotic or hemodynamic stimulation. Due to increased secretion of ADH, water cannot be discharged freely with normal excretion. Water retention leads to dilution hyponatremia and gradual enlargement of inner and outer cells' cell fluid. Increased ECF volume leads to increased secretion of ANP (atrial natriuretic peptide) and decreased aldosterone secretion. These changes stimulate sodium excretion with loss of ECF isotonicity, returning the ECF volume to its reference volume. Natriuresis in the presence of water retention leads to concentrated urine. (Usually, in patients with hyponatremia the loss of Na is negligible, in the same way as volume reduction associated with oliguria). Natriuresis prevents the correction of

hyponatremia out of habit add salt (in case of no water restriction)

Figure 3.3: Pathophysiology of SIADH.





CSW – Cerebral Salt Wasting:

CSW is in a low volume state, a new understanding of the diagnosis of CSW is due to reduced blood and plasma volume in patients who met Conventional inspection criteria for SIADH. Patientswith intracranial haemorrhage developed hyponatremia by day 10 of illness and was associated with elevated sodium concentration in urine (0.25 mEq) and improperly concentrated urine. Hyponatremia and other clinical criteria The SIADH diagnostic of having a volume status. Not compatible with this diagnosis. Rather, the evidence of Negative salt balance and depletion of both blood and plasma Total blood volume is more consistent in these patients With CSW diagnostics. The beginning of this affliction is usually within the first 10 days after the accident After a neurosurgical intervention or a definable event such as subarachnoid hemorrhage and stroke. very late Onset of disability (postoperative day 35).

Pathophysiology of CSW:

Increased urinary sodium excretion in the setting of reduced ECF volume would be expected to result in renal Potassium loss because of increased serum aldosterone levels. The lack of renal Potassium wasting in CSW is because of failure to increase serum aldosterone.

Figure 3.4: Pathophysiology of CSW Central nervous system disease BNP, ANP, other Sympathetic nervous system 🗻 natriuretic factors? Outflow

Proximal Na* Proximal urate Renin ◄ reabsorption reabsorption Distal Na+ delivery Aldosterone Hypouricemia Natriuresis without Na* K+ wasting reabsorption in EABV - AVP → Urinary concentration Hyponatremia

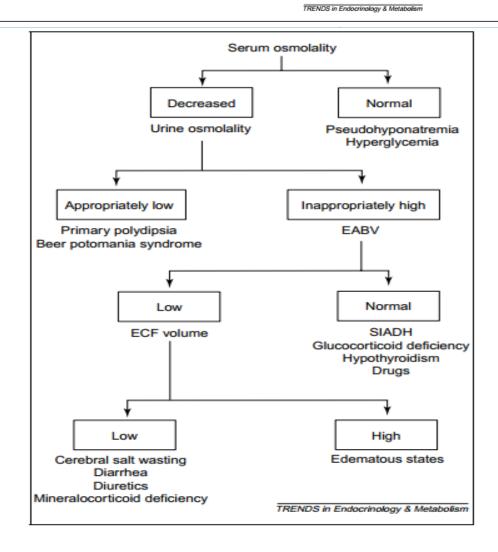


Figure 3.6: CSW and SIADH.

	CSW	SIADH
Extracellular fluid volume ^b	Decreased	Increased
Hematocrit	Increased	Normal
Plasma albumin concentration	Increased	Normal
Plasma BUN/creatinine	Increased	Decreased
Plasma K ⁺	Normal or increased	Normal
Plasma uric acid	Normal or decreased	Decreased
Treatment	Normal saline	Fluid restriction

^{*}Abbreviations: BUN, blood urea nitrogen; CSW, cerebral salt wasting; SIADH, syndrome of inappropriate antidiuretic hormone secretion.

Treatment of CSW and SIADH

Pandemic Restrictions are used in SIADH because the main that is unusual is the volume expansion of the ECF with water. NaCl administration is indicated in CSW because volume depletion due to renal salt excretion. The possibility of fluid restriction aggravates underlying neurological status in patients of CSW .Decrease in plasma volume has the potential to worsen cerebrovascular status by increasing the risk of cerebral ischemia. The intravascular volume should be maintained with IV Normal saline. Once the Patient can take oral medication, salt Tablets can be used. Management with fludrocortisone can also used.

Articles related to the studies

K Kusuda et al in their study saw that Serum sodium and potassium levels were measured in 196 patients with acute brain failure. infarction and 56 cerebral hemorrhages. [6]

According to Sivakumar Karunanandham et al in their study to estimate sodium levels in patients with CVA. [7]

M A Kabir et al. study to see the pattern and outcome of electrolyte imbalance in acute stroke patients showed that of the 80 patients. Of the 20 patients with hyponatremia, 14 deaths, and the 28 patients with hypokalemia, 14 died. Out of a total of 80 patients, 48 (60%) improved and 4 (5%) remained unchanged and moved to a higher center. [8]

^bDetermination of extracellular fluid volume is the primary way to differentiate CSW from SIADH.

By Mu Chi Chung et al reported that severe hyponatremia patients are at a higher risk of dementia than patients with non-severe hyponatremia.[9]

Sarfraz Mahesar et al. evaluated ischemic stroke patients for hyponatremia. [10]

Moiz Ehtesham et al. conducted a study to see the clinical spectrum of hyponatremia in patients with stroke. Their mean serum sodium concentration was 130.4 ± 3.5 (m Eq/L). Ischemic stroke was more common in hyponatremia (67.7%) and SIADH. groups were the more common cause of hyponatremia (71.1%). [11]

Shogo Shima et al. in their systematic review and meta-analysis have evaluated the significance of hyponatremia as a prognostic indicator among patients of acute stroke. The popularity rate of Stroke patients had hyponatremia ranging from 7.0 to 59.2%. Patients with hyponatremia tend to die in the hospital higher than those without hyponatremia. Hyponatremia may be a significant predictor of poor outcome after stroke. [12]

E F Wijdicks et al measured atrial sodium diuresis factor and vasopressin and sodium levels steady state for 5 days in 14 consecutive patients after aneurysm subarachnoid hemorrhage. Elevated plasma concentrations of atrial sodium diuresis on admission in the subarachnoid space bleeding patients (mean +/- SD 106 +/- 59 pg/ml) compared with severely ill controls (39 +/- 30pg/ml). In 8 patients, peak levels of sodium diuresis factor were higher than 300 pg/ml or doubling from baseline, followed by sodium excretion and negative sodium balance. Three patients, two of whom had hyponatremia, had cerebral infarction after sodium excretion. Vasopressin levels increase slightly immediately after bleeding but then decrease to normal values. [13]

Farah Mansoor et al. The purpose of our study was to determine electrolyte imbalance in patients who suffered an acute stroke in a tertiary care hospital. Average sodium levels are significantly lower in the ischemic group than in the hemorrhagic group. Significantly higher potassium levels in the bleeding group versus the ischemic group . [14]

Maliha Hakim, et al. conducted a study to observe the effect of hyponatremia on mortality and functional outcomes in a hospitalized patients stroke patient. This prospective analytical cohort study, performed had 229 patients hospitalized for acute stroke (65 ischemic, 164 hemorrhagic). Patient characteristics, hospitalization Measures, mortality, and functional outcomes on the mRS sclale (at discharge and age 30 and 90 days) were analyzed for hyponatremic eyes (Na<135 mmol/L). multivariable Regression analyzes were calculated for 90-day mortality predictors. Among 229 patients, 65 (28.4%) were hospitalized for acute ischemic stroke and 144 (62.9%) for acute stroke. Intracranial hemorrhage (ICH) and 20 (8.7%) for acute subarachnoid hemorrhage (ASH). five people died before serum can be sent for electrolyte testing (3 ICH, 1 SAH, and 1 ischemia). Furthermore, hyponatremia was detected in 39 (17.4%).SIADH was the most common cause of hyponatremia (51.3%), followed by brain salt loss (CSW; 30.8%). Although hyponatremia in both types of strokes is associated with higher mortality and poorer functional outcomes at discharge, 30 days and 90 days, which do not statistical significance. [15]

Ali Shah et al. evaluated the frequency of hyponatremia in patients with hemorrhagic stroke and determine its impact on their morbidity outcomes in the hospital. [16]

Huang W.-Y et al. analyzed if hyponatremia in stroke contributes to an increased risk of death or recurrent stroke in these patients. Among the risk factors for stroke, the incidence of diabetes was significantly higher in patients with hyponatremia. Hyponatremia was a significant predictor of 3-year mortality in these patients. [17]

Sheikh Saleem et al. in this study was conducted in a tertiary care hospital to determine the incidence and etiology of hyponatremia in hospitalized stroke patients. Hospital. Out of 1000 patients, 353 had hyponatremia. Of these 353 patients, 238 (67%) had SIADH and 115 (33%) CSWS. Statistical analysis showed that hyponatremia significantly affected the outcome of stroke and is especially caused by CSWS and not SIADH. [18]

MATERIAL AND METHODS

MATERIAL AND METHODS

Study design: Cross-Sectional study [Observational study]

Study Setting: Sri Devraj Urs Medical College and Hospital [R.L Jalappa hospital and

Research Centre.]

Study Population:

- **Population element**: Patients diagnosed with Acute Stroke
- Sampling element: Patients diagnosed with Acute Stroke in the study setting.

Study Period: January 2021- May 2022

Sample Size Estimation:

Assuming that random sampling and conditions warrant approximate normality of the distribution of p, sample size determination when a population proportion is to be estimated leads to the following formula for n, given by

$$n=z^2 \frac{pq}{d^2}$$

where,

n = Sample size

z = Standard Normal Deviate [z value] for a given level of confidence

p = prevalence or proportion

q = 1-p

d = absolute allowable error

Hereby taking, the **proportion of hyponatremia in stroke patients was 38.61%** from the study by Karunanandham S et al, p = 38.61%, 95% level of confidence, z = 1.96 for $\alpha = 5\%$ and d = 10%,

$$n = \frac{1.96^2 (38.61)(100 - 38.31)}{10^2} = 92$$

Considering a 10% non-response rate i.e., 92+9.2 = 102.

A minimum sample size of 102 was required for the study.

Sampling technique:

Purposive sampling was used. Specifically, patients who have been diagnosed with Acute Stroke [Diagnosis with CT or MRI proven Ischemic or Hemorrhagic Stroke] were considered for the study.

Eligibility Criteria:

• Inclusion Criteria:

- 1. Age > 18yrs
- 2. Confirmed cases of Stroke:
- 3. History, Neurologic and Imaging modalities presenting within 24hrs of the onset of stroke

• ExclusionCriteria

•	CT/MRI SHOWING CNS INFECTION
•	HISTORY OF HEAD INJURY
•	INFECTIONS - DIARRHOEA, PNEUMONIA
•	DRUG INTAKE
•	MALIGNANCY
•	HYPERGLYCEMIA (RBS>140)
•	HYPOTHYROIDISM (TSH>4)
•	DERANGED RENAL AND LIVER FUNCTION
•	COVID 19 POSITIVE STATUS
•	HISTORY OF RECENT SURGERY

Tools Used:

1. A proforma which was predesigned, pretested, and semi-structured proforma was used after piloting and an expert validation of the proforma. Content and Face

validation of the proforma was done by experts. Piloting of the proforma was done for finalizing the proforma and to see the feasibility of the study. Proforma includes the following details: Demographics, history, examination, and investigation. The proforma is annexed.

2. Investigations

RANDOM BLOOD SUGAR
SERUM URIC ACID
HEMATOCRIT
SERUM ALBUMIN
BUN/CREATININE RATIO
THYROID FUNCTION TEST
NIHSS SCORE ON DAY 1,3,7

3. National Institute of Health Stroke Scale [NIHSS] score:

"The NIHSS is a 15-item neurological examination stroke scale used to evaluate the effect of acute cerebral infarction on the levels of consciousness, language, neglect, visual-field loss, extraocular movement, motor strength, ataxia, dysarthria, and sensory loss. Ratings for each item are scored on a 3- to 5-point scale, with 0 as normal, and there is an allowance for untestable items. Scores range from 0 to 42, with higher scores indicating greater severity."

Stroke severity may be stratified based on NIHSS scores as follows:

• Very Severe: >25

• Severe: 15 – 24

• Mild to Moderately Severe: 5 − 14

• Mild < 5

1a—Level of consciousness	0 = Alert; keenly responsive
	1 = Not alert, but arousable by minor stimulation
	2 = Not alert; requires repeated stimulation
	3 = Unresponsive or responds only with reflex
1b—Level of consciousness questions:	0 = Answers two questions correctly
What is your age?	1 = Answers one question correctly
What is the month?	2 = Answers neither questions correctly
1c—Level of consciousness commands:	0 = Performs both tasks correctly
Open and close your eyes	1 = Performs one task correctly
Grip and release your hand	2=Performs neither task correctly
2—Best gaze	0 = Normal
2—Best gaze	
	1 = Partial gaze palsy 2 = Forced deviation
3—Visual	0 = No visual lost
	1 = Partial hemianopia
	2 = Complete hemianopia
	3 = Bilateral hemianopia
4—Facial palsy	0 = Normal symmetric movements
	1 = Minor paralysis
	2 = Partial paralysis
	3 = Complete paralysis of one or both sides
5—Motor arm	0 = No drift
Left arm	1 = Drift
Right arm	2 = Some effort against gravity
Kight aim	3 = No effort against gravity
	4 = No movement
6—Motor leg	0 = No drift
Left leg	1 = Drift
Right leg	2 = Some effort against gravity
	3 = No effort against gravity
	4 = No movement
7—Limb ataxia	0 = Absent
	1 = Present in one limb
	2 = Present in two limbs
8—Sensory	0 = Normal; no sensory loss
	1 = Mild-to-moderate sensory loss
	2 = Severe-to-total sensory loss
9—Best language	0 = No aphasia; normal
>—Dest language	1 = Mild-to-moderate aphasia
	2 = Severe aphasia
10 D 11	3 = Mute; global aphasia
10—Dysarthria	0 = Normal
	1 = Mild-to-moderate dysarthria
	2 = Severe dysarthria
11—Extinction and inattention	0 = No abnormality
	1 = Visual, tactile, auditory, spatial, or personal inattention
	2 = Profound hemi-inattention or extinction
Score = 0-42	•

• Operational Definition:

- **STROKE** -"An abrupt onset of a neurological deficit that is attributable to a focal vascular cause".
- **HYPONATREMIA** "Hyponatremia is defined as sodium level < 135mEq/L"
- TRUE HYPONATREMIA is defined as those patients with a sodium level of 135mEq/L and plasma osmolality less than 275 m osm/kg
- **PLASMA OSMOLALITY**: calculated by- 2(Na)+ Glu/18+ BUN/2.8
- **SODIUM CORRECTION**: Total body water (TBW) 50% of body weight in females and 60% of body weight in males
 - \circ Free water deficit [(Na+ 140) / 140] * TBW
 - \circ Free water clearance V * [1- (U. Na +UK) / P. Na]
 - o Timing of Sodium correction:>125mEq/L oral salt supplements, <125mEq/L
 - IV Sodium correction

NORMAL RANGES OF PARAMETERS USED IN THE STUDY

- ➤ Plasma Osmolality 275 295 mosm/kg
- ➤ Serum sodium 135-145 mEq/L
- Serum Potassium 3.5 5 mEq/L
- ➤ Serum Albumin 3.5- 5 g/dl
- ➤ Hematocrit Male: 40.7-50%: Female: 36.1- 44.3%
- ➤ BUN/ Creatinine 10:1- 20:1
- **CEREBRAL SALT WASTING SYNDROME [CSW]:** is defined as any 2 of the following features will be needed for diagnosis:
 - "Clinical findings of hypovolemia hypotension, dry mucous membranes, tachycardia, postural hypotension
 - Lab evidence of dehydration- elevated hematocrit, hemoglobin, serum albumin, blood urea
 - Negative fluid balance or weight loss"
- **SIADH:** is defined as any 2 of the following features in a patient with hyponatremia will be needed for diagnosis
 - "No signs of hypovolemia like hypotension, dry mucous membranes, tachycardia, postural hypotension
 - No lab evidence of dehydration- elevated hematocrit, hemoglobin, serum albumin, blood urea
 - o Normal or Positive fluid balance with the absence of weight loss"

DIAGNOSTIC CRITERIA BASED ON LABORATORY VALUES:

Parameter	CSW	SIADH
Extracellular fluid volume	Decreased	Increased
Haematocrit	Increased	Normal
Serum albumin	Increased	Normal
Plasma BUN/Creatinine ratio	Increased	Decreased
Plasma potassium	Normal/increased	normal
Serum uric acid	Decreased	Decreased
Treatment	Normal Saline	Fluid restriction

• TREATMENT PROTOCOL: CSW received fluid correction and sodium supplementation. SIADH received fluid restriction as treatment.

Data collection:

Patients who have been diagnosed with Acute Stroke [Diagnosis with CT or MRI proven Ischemic or Hemorrhagic Stroke], who satisfy the inclusion criteria were included in the study. Informed consent taken. Detailed history, Examination, and investigations were done on the selected study participants. The severity of the stroke was assessed by NIHSS score. Investigations like Serum electrolytes, Serum albumin, Renal Function Tests, urine osmolarity, serum uric acid, and hematocrit were done. Later, observation was made on patients developing hyponatremia and true hyponatremia. Based on the diagnostic algorithm and operational definition mentioned patients were classified as SIADH and CSW. Treated patients were followed up on days 1, 3, and 7 of hospitalization. The outcome of the patient was determined using NIHSS (National Institutes of Health Stroke Scale) to know the severity of the stroke on all 3 days i.e., Days 1,3,7 of hospitalization. A short-term follow-up was done to asses the outcome of the patient.

Statistical methods to be employed:

Data was entered in the licensed version 2016 of the Microsoft excel spreadsheet. To address the objectives, descriptive statistics like frequency, percentages, and graphs were used. Microsoft Excel software was used for the analysis. The level of significance[α] was 5% i.e., p-value <0.05 is considered statistically significant.

Ethical Considerations:

Ethical clearance was taken from the institutional ethical committee. The ethical Clearance letter is annexed. [Annexure 1]

The four universal ethical principles in biomedical research are followed in the present study, as described in the landmark book Principles of biomedical ethics by Beauchamp and Childress. They are

a) Respect for autonomy:

The study subjects were explained about the study and prior written informed consent was taken in the local language [Kannada] and English. Only those who gave consent were included in the study.

b) Beneficence:

All the participants explained the importance of the study and assurance was given there will be no breach in the care given if refused to consent to the study.

c) Nonmaleficence:

The patient will not be exposed to any additional risk as a result of this study. Due care was taken to protect the privacy of the study subjects. Confidentiality of the information collected was maintained.

d) Justice:

Due care was taken while recruiting the participants. Fairness and Equity were maintained in selecting the study participants.

Sample Size Estimation

Assuming that random sampling and conditions warrant approximate normality of the distribution of p, sample size determination when a population proportion is to be estimated leads to the following formula for n, given by

$$n=z^2 \frac{pq}{d^2}$$

where,

n = Sample size

z = Standard Normal Deviate [z value] for a given level of confidence

p = prevalence or proportion

q = 1-p

d = absolute allowable error

Hereby taking, the **prevalence of hyponatremia in stroke patients was 38.61%** from the study by Karunanandham S et al, p = 38.61%, 95% level of confidence, z = 1.96 for $\alpha = 5\%$ and d = 10%,

$$n = \frac{1.96^2 (38.61)(100 - 38.31)}{10^2} = 92$$

Considering a 10% non-response rate i.e., 92+9.2 = 102.

A minimum sample size of 102 was required for the study.

RESULTS

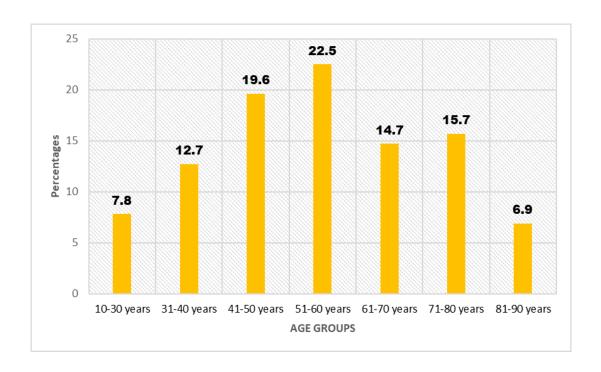
RESULTS

Table 6.1: Demographic characteristics of the study participants. [n=102]

Sl no	Variable Freq		Frequencies	Percentages
		Female	40	39.2
1	Gender	Male	62	60.8
		Total	102	100.0
		10-30 years	8	7.8
		31-40 years	13	12.7
		41-50 years	20	19.6
2	A C	51-60 years	23	22.5
2	Age Group	61-70 years	15	14.7
		71-80 years	16	15.7
		81-90 years	7	6.9
		Total	102	100.0

Table 6.1 shows the demographic characteristics of the study participants. 60.8 % of the patients were males and the rest were females. Age group-wise distribution shows that majority of the patients were in age 51-60 years, followed by 41-50 years and 71-80 years 19.6 and 15.7 percentages respectively. It is worth noting that nearly 20% of patients belong in the early 41-50 years.

Figure 6.1: Gender distribution of the study participants.



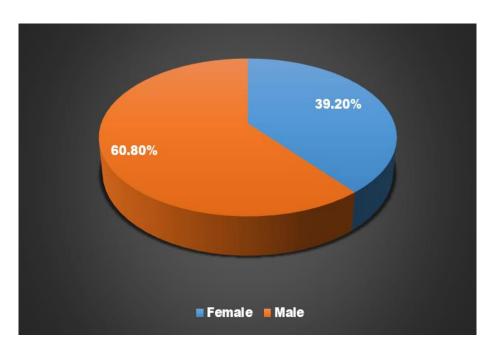


Figure 6.2: Age group distribution of the study participants

Table 6.2: Type of stroke among the patients in the study. [n=102]

Variable	Frequencies	Percentages
Ischemic stroke	71	69.6
Hemorrhagic stroke	31	30.4
Total	102	100.0

Figure 6.3: Type of stroke among the patients in the study

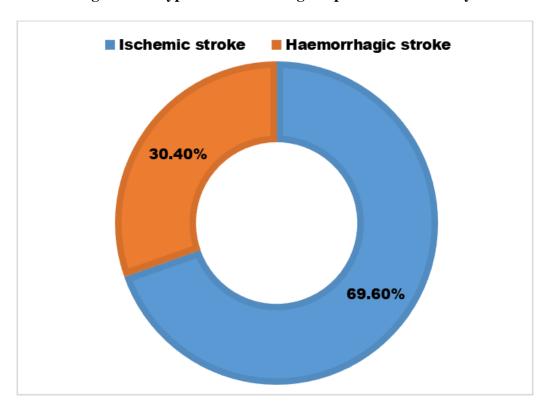


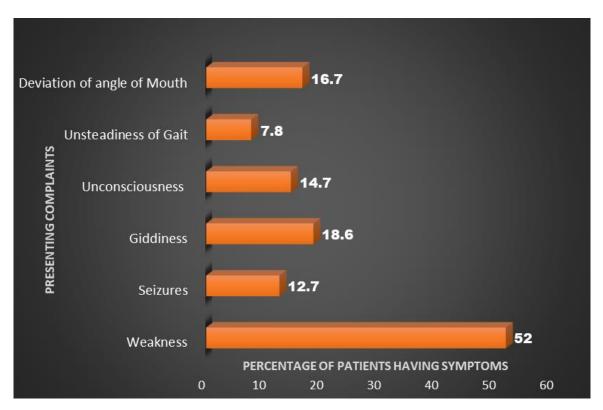
Table 6.2 and the above figure shows that nearly 70 % of the stroke were due to Ischemic events in the vessels of the brain, rest 70 % were due to hemorrhage.

Table 6.3: Clinical presentation of the patients in the study. [n=102]

Sl no	Variable	Frequencies	Percentages*
1	Weakness	53	52.0
2	Seizures	13	12.7
3	Giddiness	19	18.6
4	Unconsciousness	15	14.7
5	Unsteadiness of Gait	8	7.8
6	Deviation of the angle of the Mouth	17	16.7

^{*} Percentages do not add up to 100% as each patient may have more than one symptom

Figure 6.4: Clinical presentation of the patients in the study



As depicted in the above table and figure, weakness [52%] is the most common presenting among the patients diagnosed with stroke followed by deviation of angle of mouth [16.7%], giddiness [18.6%], unconsciousness [14.7%] and seizures [12.7%].

Table 6.4: Co-morbidities among the patients in the study. [n=102]

Sl no	Variable	Frequencies	Percentages*
1	Prior Stroke	12	11.8
2	Diabetes mellitus	38	37.3
3	Hypertension	57	55.9
4	Cardiac Disease	12	11.8
5	Epilepsy	6	5.9
6	Tuberculosis	3	2.9

^{*} Percentages do not add up to 100% as each patient may have more than one co-morbidity

Prior Stroke
Cardiac Disease
Epilepsy
Prior Stroke

Prior Stroke

Cardiac Disease

Diabetes mellitus
Epilepsy
Tuberculosis

Prior Stroke

Cardiac Disease

Epilepsy
Tub...

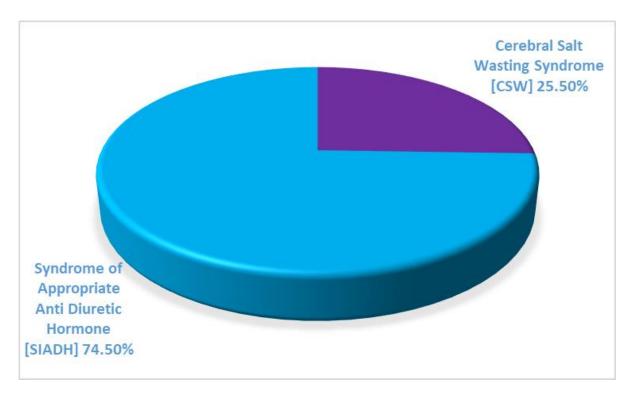
Figure 6.5: Co-morbidities among the patients in the study.

Hypertension was the most common comorbidity among the patients with stroke [55.9%]. Next in line is diabetes mellitus with 37.8%. Other co-morbidities are prior stroke and previous cardiac disease. Few of the patients had a history of epilepsy and tuberculosis.

Table 6.5: Classification of stroke patients into CSW and SIADH. [n=102]

Variable	Frequencies	Percentages
Cerebral Salt Wasting Syndrome [CSW]	26	25.5
Syndrome of Appropriate Anti-Diuretic Hormone [SIADH]	76	74.5
Total	102	100.0

Figure 6.6: Classification of stroke patients into CSW and SIADH. [n=102]



According to the above table 6.5 and figure 6.6, 74.5% of the stroke patients had Syndrome of Appropriate Anti Diuretic Hormone [SIADH], while 25.5% of them had Cerebral salt wasting syndrome.

Table 6.6: Age group-wise and gender-wise classification of stroke patients into CSW and SIADH. [n=102]

Sl No	Variable		CSW	SIADH
			Frequencies [Percentages #]	
		male	7 [26.9]	33 [43.4]
n	ıder	ale	19 [73.1]	43 [56.6]
		tal	26 [100]	76 [100]
		-30 years	3 [11.5]	5 [6.6]
		-40 years	1 [3.8]	12 [15.8]
		-50 years	3 [11.5]	17 [22.4]
	Crown	-60 years	4 [15.4]	19 [25.0]
ţe.	Group	-70 years	5 [19.2]	10 [13.2]
		-80 years	7 [26.9]	9 [11.8]
		-90 years	3 [11.5]	4 [5.3]
		tal	26 [100]	76 [100]

Percentages in the parenthesis are column percentages

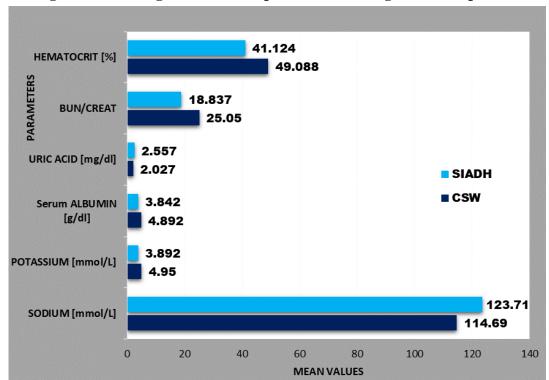
Table 6.6 shows that in both CSW and SIADH, males were the majority of the patients. Among patients with CSW, a large share of patients was in the age group between 61-80 years, and among patients with SIADH, the majority of patients are in the age group of 31-60 years. This may indicate that more older patients have CSW and relatively younger patients may have SIADH.

Table 6.7: Average values of the parameters among the stroke patients.

Sl no	Parameter	CSW		SIADH	
		Mean	SD	Mean	SD
1	PULSE [beats per minute]	88.19	6.705	88.14	7.552
2	SODIUM [mmol/L]	114.69	8.284	123.71	7.379
3	POTASSIUM [mmol/L]	4.950	.8599	3.892	.5564
4	Serum ALBUMIN [g/dl]	4.892	.9139	3.842	.5259
5	URIC ACID [mg/dl]	2.027	.5647	2.557	.5936
6	BUN/CREAT	25.050	18.0485	18.837	7.8008
7	HEMATOCRIT [%]	49.088	8.8574	41.124	6.8635

SD- Standard Deviation

Figure 6.7: Average values of the parameters among the stroke patients





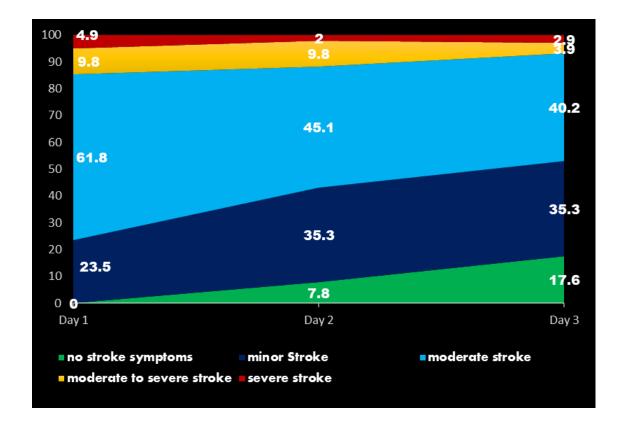


Figure 6.8 depicts the categories of the severity of stroke based on NIHSS score among the study population. As we can see that starting from the day one there were more patients of moderate stroke and higher severity, as the treatment initiated, and days progressed to day 3 and day 7 there are more patents of minor severity and there are patients who do not show any signs of stroke according to NIHSS score.

Table 6.8: Severity of stroke due to CSW according to NIHSS scores of the patients in the study. [n=102]

		on Day 1	on Day 3	on Day 7
l no	CSW		<u> </u>	
		Freq	uencies [Percentag	ges #]
1	stroke signs	0	1 [3.8]	0
2	Minor Stroke	0	1 [3.8]	2 [7.7]
3	oderate stroke	19 [73.1]	17 [65.4]	18 [69.2]
4	oderate to Severe stroke	3 [11.5]	5 [19.2]	3 [11.5]
5	vere stroke	4 [15.4]	2 [7.7]	3 [11.5]
	tal	26 [100]	26 [100]	26 [100]

[#] Percentages in the parenthesis are column percentages.

Table 6.8 shows the regression in the severity of the stroke as the days pass by among the patients of the CSW. On day 1 there are more patients at the worse end i.e., more moderate and severe stroke patients. However, on day 7 there are few patients with minor strokes. But largely there is not much change in the categories of severity of stroke among those diagnosed with CSW.

Table 6.9: Severity of stroke due to SIADH according to NIHSS scores of the patients in the study. [n=102]

l no	SIADH	on Day 1	on Day 3	on Day 7
		Frequencies [Percentages #]		
1	stroke signs	0	7 [9.2]	18 [23.7]
2	Minor Stroke	24 [31.6]	35 [46.1]	34 [44.7]
3	oderate stroke	44 [57.9]	29 [38.2]	23 [30.3]
4	oderate to Severe stroke	7 [9.2]	5 [6.6]	1 [1.3]
5	vere stroke	1 [1.3]	0	0
	tal	76 [100]	76 [100]	76 [100]

[#] Percentages in the parenthesis are column percentages

Table 6.9 shows the regression in the severity of the stroke as the days pass by among the patients with SIADH. On day 1 there are more patients at the worse end i.e., more moderate and severe stroke patients. However, on day 7 there are a large number of patients with minor strokes and 23.7% of them depicted no signs of stroke. There is considerable change in the categories of severity of stroke among those diagnosed with SIADH towards faster improvement.

Table 6.10: Improvement* in the NIHSS score from Day 1 to Day 7.

Variable		CSW	SIADH	Total
		Frequencies [Percentages #]		
provement	proved	5 [19.2]	43 [56.6]	48 [47.1]
	t improved	21 [80.8]	33 [43.4]	54 [52.9]

^{*}Improved is defined as the change in one category of severity from worse to better.

Percentages in the parenthesis are column percentages.



Figure 6.9: Improvement in the NIHSS score from Day 1 to Day 7.

The condition of the patient was considered improved if there was a change in one category of severity from worse to better. For example, day 1 moderate to severe stroke and on day 7 moderate stroke. Table 6.10 and figure 6.9 shows that 56.6% of the stroke patients improved in the group of SIADH while only 19.2% improved among CSW. Improvement seems better among the patients with SIADH than CSW.

Table 6.11: Association between the Improvement and CSW versus SIADH. [n=102]

Variable		CSW	SIADH	Total
			Frequencies	
	proved	5	43	48
provement	t improved	21	33	54
	tal	26	76	102
	Chi-Square	test of association:	p-value = 0.001	•
	Odd's Ratio for	· improvement am	ong SIADH is <mark>1.46</mark>	66

p-value <0.05 is considered significant.

The difference in the improvement between the SIADH and CSW was tested for its association and strength of association by estimating its odd's Ratio. Table 6.11 shows that there is an association between SIADH and CSW and the improvement of the patients. This association is statistically significant with p value = 0.001.

Odd's Ratio for Improvement among SIADH is 1.466. This means that patients with stroke and hyponatremia having been diagnosed with SIADH have 1.46 times higher chances of improvement when compared to patients diagnosed with CSW. Also, can be said that patients with SIADH have 46.6% higher chances of faster and better improvement compared to patients with CSW.

Table 6.12: Short-term outcome of the patients in the study. [n=102]

Variable -		CSW	SIADH	Total
		Frequencies [Percentages #]		
taoma	ath	2 [7.7]	1 [1.3]	3 [2.95]
itcome	red	24 [92.3]	75 [98.7]	99 [97.05]

Percentages in the parenthesis are column percentages.

Figure 6.10: Short-term outcome of the patients in the study



Table 6.12 and figure 6.10 shows that 7.7% of the stroke patients died in the group of CSW while only 1.3% died among SIADH. Improvement seems better among the patients with SIADH than CSW. There are definitive fewer deaths among the patients with SIADH compared to CSW.

DISCUSSION

DISCUSSION

The discussion of the current study is discussed under the following headings:

- 1. Demographics
- 2. Type of stroke
- 3. Co-morbidities
- 4. SIADH and CSW
- 5. Improvements in stroke patients

1. Demographics

According to the results of our study 60.8 % of the patients were males and the rest were females. Age group-wise distribution shows that majority of the patients were in age 51-60 years, followed by 41-50 years and 71-80 years 19.6 and 15.7 percentages respectively. It is worth noting that nearly 20% of patients belong in the early 41-50 years.

According to Sivakumar K et al [6] in their study 86.6% of the participants were male and only 13.3% were females. Average age of the patients is 57.5 years. These results are slightly differed from our study.

In the study by MA Kabir et al [8] 62.5% of the patients were males and rest were females. Majority of the patients were in the 51-60 years followed by >70 years. In terms of age distribution, the results are similar to our results.

In can be said that our study has better distribution of the both the genders compared to the other studies which adds to the strength of our study since it has better generalisability across the gender. In terms of age distribution, our study has wider range of age which is different from the other studies. It also depicts that even younger patients are having stroke and differentiating between SIADH and CSW will help in improvement irrespective of the age of onset of stroke.

2. Type of stroke

Among the stroke patients in the current study, 70 % of the stroke were due to ischemic events in the vessels of the brain, rest 30 % were due to haemorrhage.

According to Sivakumar K et al [6] 80% of the participants had ischemic stroke while 15% had haemorrhagic stroke and 5% had transient ischemic attack. These results are comparable to the findings of our study.

Findings of study by Moiz Ehtesham et al. [11] show that, 67.7% of patients had ischemic stroke and 32.2% of them had haemorrhagic stroke. The findings are similar to the finding of our study.

On comparison with the other studies, it can be said that, proportion of ischemic stroke is more common than haemorrhagic stroke among stroke patients. These results are shown in our study also which guarantees good representation of the study population in the present study.

3. Co-morbidities

Hypertension was the most common comorbidity among the patients with stroke [55.9%]. Next in line is diabetes mellitus with 37.8%. Other co-morbidities are prior stroke and previous cardiac disease. Few of the patients had a history of epilepsy and tuberculosis.

Moiz Ehtesham et al. [11] in their study showed that 23.1% of the patients had hypertension, 39.6% of them had neurological disease, 7.4% had diabetes mellitus and 14.1% had ischemic heart diseases as a comorbidity.

Mu-Chi Chung et al [5] in their study, shows that 40.8% have diabetes,68.4% have hypertension, 40.2% have ischemic heart diseases, 32.9% have hyperlipidaemia and 17.1% have heart failure.

All the studies including our study shows the presence of comorbidities in the stroke patients. However, the type of comorbidities differs in their proportion. The most common one among all are hypertension, diabetes and ischemic heart diseases.

4. SIADH and CSW

74.5% of the stroke patients in our study had SIADH while 25.5% of them had CSW.

In the study by Moiz Ehtesham et al. [14] we can see that 71.1% of the stroke patients had SIADH and 28.9% of them had CSW syndrome. These findings are similar to the results of our study.

Sivakumar K et al [3] in their study has published that 21.2% of the patients of stoke with hyponatremia had SIADH and 7.4% of them had CSW while 9.9% of their cause for hyponatremia was unknown.

Even according to Ali Shah et al [16], 26.4% of the patients had hyponatremia due to SIADH and 18.8% of them had hyponatremia due to CSW. 54.7% of the stroke patients did not have hyponatremia.

The proportional difference between occurrence of SIADH and CSW are similar although exact percentage of occurrence is difference. This is because Sivakumar K et al and Ali shah et al has patients with stroke who have normal sodium levels in their study.

5.Improvements in stroke patients

In the present study, 56.6% of the stroke patients improved in the group of SIADH while only 19.2% improved among CSW. Improvement seems better among the patients with SIADH than CSW, there is an association between SIADH and CSW and the improvement of the patients. This association is statistically significant with p value = 0.001. Odd's Ratio for Improvement among SIADH is 1.466. This means that patients with stroke and hyponatremia having been diagnosed with SIADH have 1.46 times higher chances of improvement when compared to patients diagnosed with CSW

Mu-Chi Chung et al [9] in their study, shows that hyponatremia cohort has higher hazard

ration of developing dementia than normal sodium levels. The hazard ratio is increases with the increase in the severity of the hyponatremia. As dementia can be one of the factors determining the quality of life of the patients post stroke. It also affects the outcome of the patients.

Ali Shah et al [16] in their studied the improvement of the stroke patients who has SIADH and CSW. It can be seen that 27.6% of the patients recovered completely among SIADH while it was only 6.3% among CSW. In hospital mortality was seen among 36.8% of SIADH patients and 50% of CSW patients. These results clearly indicate that improvement is better among patients with SIADH.

There is consistency among the studies regarding the better improvement and prognosis of the stroke patients who have hyponatremia due to SIADH than patients due to CSW.

Based on the results of the present study and findings from the other studies it can be said that hyponatremia should be evaluated in all the acute stoke patients. On proper diagnosis and treatment of CSW and SIADH in stroke patients with hyponatremia can have better prognosis and outcome. Reversal of severity of the stroke, survival of the patients is better in patients with SIADH than with patients with CSW

CONCLUSION

CONCLUSION

- 1. Hyponatremia should be evaluated in all the acute stroke patients.
- 2. On proper diagnosis and treatment of CSW and SIADH in stroke patients with hyponatremia can have better prognosis and outcome.
- 3. Reversal of severity of the stroke, survival of the patients is better in patients with SIADH than with patients with CSW.

SUMMARY

SUMMARY

INTRODUCTION:

Stroke being one of the major non-communicable diseases leading to significant disability and loss of quality of life. Hyponatremia is common in patients with stroke. The causes of hyponatremia are varied but are most commonly attributed to SIADH and CSW. Differentiation between SIADH and CSW is important because treatment of one may be hazardous to the other. Hyponatremia is independently associated with higher mortality. There is a paucity of prospective studies that evaluate the frequency, severity, and causes of

hyponatremia in patients with stroke and its effect on short-term outcomes

OBJECTIVES:

1. To measure the magnitude of hyponatremia in acute stroke.

2. To measure the frequency of occurrence of SIADH and CSW in patients with

hyponatremia in Acute Stroke.

3. To compare short-term outcomes in patients with SIADH and CSW

METHODOLOGY:

Study design: Cross-Sectional study [Observational study]

Study Setting: Sri Devraj Urs Medical College and Hospital [R.L Jalappa hospital and

Research Centre.]

Study Population:

• **Population element**: Patients diagnosed with Acute Stroke

Sampling element: Patients diagnosed with Acute Stroke in the study setting.

Study Period: January 2021- May 2022

Sample Size Estimation:

Taking, the prevalance of hyponatremia in stroke patients was 38.61% from the study by

Karunanandham S et al., p = 38.61%, 95% level of confidence, z = 1.96 for α = 5% and d =

10%,

$$n = \frac{1.96^2 (38.61)(100 - 38.31)}{10^2} = 92$$

Considering a 10% non-response rate, i.e., 92+9.2 = 102.

A minimum sample size of 102 was required for the study.

Sampling technique:

Purposive sampling was used. Specifically, patients who have been diagnosed with Acute Stroke [Diagnosis with CT or MRI proven Ischemic or Hemorrhagic Stroke] were considered for the study.

Eligibility Criteria:

• Inclusion Criteria:

- 1. Age > 18yrs
- 2. Confirmed cases of Stroke within 24hrs of the onset of stroke

• Exclusion Criteria:

- 1. CT /MRI showing CNS infections
- 2. History of Head injury
- 3. Any Infections Diarrhea, Pneumonia
- 4. Drug history
- 5. Malignancy
- 6. Hyperglycemia (RBS >140mg/dL)
- 7. Hypothyroidism (TSH > 4mU/L)
- 8. Patients with deranged kidney and liver functions
- 9. Covid 19 positive status
- 10. Patients with a history of recent surgery.

Tools Used:

a. A proforma which was predesigned, pretested, and semi-structured proforma was used after piloting and an expert validation of the proforma. Content and Face validation of the proforma was done by experts. Piloting of the proforma was done for finalizing the proforma and to see the feasibility of the study. Proforma includes the following details: Demographics, history, examination, and investigation. The proforma is annexed.

b. Investigations

Data collection:

Ethical clearance was taken from the institutional ethical committee. The ethical Clearance letter is annexed. Patients who have been diagnosed with Acute Stroke [Diagnosis with CT or MRI proven Ischemic or Hemorrhagic Stroke], who satisfy the inclusion criteria and were included in the study. Informed consent taken. Detailed history, Examination, and investigations were done on the selected study participants. The severity of the stroke was assessed by NIHSS. Investigations like Serum electrolytes, Serum albumin, Renal Function Tests, urine osmolarity, serum uric acid, and hematocrit were done. Later, observation was made on patients developing hyponatremia and true hyponatremia. Based on the diagnostic algorithm and operational definition mentioned patients were categorised as SIADH and CSW. As per the classification and the protocol patients were treated. Treated patients were followed up on days 1, 3, and 7 of hospitalization. The outcome of the patient was determined using NIHSS (National Institutes of Health Stroke Scale) to know the severity of the stroke on all 3 days i.e., Days 1,3,7 of hospitalization.

Statistical methods to be employed:

Data was entered in the licensed version 2016 of the Microsoft excel spreadsheet. To address the objectives, descriptive statistics like frequency, percentages, and graphs were

used. Microsoft Excel software was used for the analysis. The level of significance[α] was 5% i.e., p-value <0.05 is considered statistically significant.

RESULTS:

The results shows that 60.8 % of the patients were males and the rest were females. Age group-wise distribution shows that majority of the patients were in age 51-60 years, followed by 41-50 years and 71-80 years 19.6 and 15.7 percentages respectively. It is worth noting that nearly 20% of patients belong in the early 41-50 years. Nearly 70 % of the stroke were due to Ischemic events in the vessels of the brain, rest 70 % were due to haemorrhage.

Weakness [52%] is the most common presenting among the patients diagnosed with stroke followed by deviation of angle of mouth [16.7%], giddiness [18.6%], unconsciousness [14.7%] and seizures [12.7%]. Hypertension was the most common comorbidity among the patients with stroke [55.9%]. Next in line is diabetes mellitus with 37.8%. Other comorbidities are prior stroke and previous cardiac disease. Few of the patients had a history of epilepsy and tuberculosis.

74.5% of the stroke patients had Syndrome of Appropriate Anti Diuretic Hormone [SIADH], while 25.5% of them had Cerebral salt wasting syndrome. From the day one there were more patients of moderate stroke and higher severity, as the treatment initiated, and days progressed to day 3 and day 7 there are more patents of minor severity and there are patients who do not show any signs of stroke according to NIHSS score.

56.6% of the stroke patients improved in the group of SIADH while only 19.2% improved among CSW. Improvement seems better among the patients with SIADH than CSW. 7.7% of the stroke patients died in the group of CSW while only 1.3% died among SIADH. Improvement seems better among the patients with SIADH than CSW. There are definitive fewer deaths among the patients with SIADH compared to CSW.

There is an association between SIADH and CSW and the improvement of the patients. This

association is statistically significant with p value = 0.001. Odd's Ratio for Improvement among SIADH is 1.466. This means that patients with stroke and hyponatremia having been diagnosed with SIADH have 1.46 times higher chances of improvement when compared to patients diagnosed with CSW. Also, can be said that patients with SIADH have 46.6% higher chances of faster and better improvement compared to patients with CSW.

CONCLUSION:

Based on the present study, it can be concluded that

- 1. Hyponatremia should be evaluated in all the acute stoke patients.
- 2. On proper diagnosis and treatment of CSW and SIADH in stroke patients with hyponatremia can have better prognosis and outcome.
- 3. Reversal of severity of the stroke, survival of the patients is better in patients with SIADH than with patients with CSW.

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ANNEXURE

ANNEXURE 1 PROFORMA DEPARTMENT OF GENERAL MEDICINE, SDUMC

NAME : AGE : SEX :

IP NUMBER : ADDRESS :

PHONE NUMBER:
Date of admission
Date of Disabarra/ Date

Date of Discharge/ Death Duration of Hospital stay

PRESENTING COMPLAINTS:

oWeakness

oSeizures

oGiddiness

oUnresponsiveness

oUnsteadiness of gait

oDeviation of angle of mouth

DURATION OF SYMPTOMS:

H/o FEVER/COUGH/LOOSE STOOLS/WEIGHT LOSS/ TRAUMA/DRUG INTAKE/RECENT SURGERY/ON DIALYSIS

PAST HISTORY:

oPrior stroke

oDiabetes mellitus

oHypertension

oCardiac disease

oEpilepsy

oTuberculosis

TREATMENT HISTORY:

FAMILY HISTORY:

ADDICTIONS:

VITALS: Pulse- Rate & Rhythm

Blood Pressure Respiratory Rate Temperature

Daily input and output

GENERAL EXAMINATION:

SYSTEMIC EXAMINATION:

CENTRAL NERVOUS SYSTEM

Higher Mental Functions: Consciousness

Orientation Speech

Cranial Nerves

Motor system: Tone

Power

Reflexes

Sensory system

Cerebellar signs

Meningeal signs

GLASGOW COMA SCALE: E V M Total score: /15

CARDIOVASCULAR SYSTEM

RESPIRATORY SYSTEM:

GASTROINTESTINAL SYSTEM:

General Parameters

oCompletehemogram

oPeripheral Smear

oRandom Blood Sugar

oBlood urea

oSerumcreatinine

oECG:

oChest X-Ray

SPECIFIC PARAMETERS:

CT scan Brain/ MRI Brain:

Day of hospitalization:

DAY 1

RANDOM BLOOD

SUGAR

BLOOD UREA

NITROGEN

SERUM SODIUM

SERUM POTASSIUM

SERUM OSMOLALITY

URINE SODIUM

URINE OSMOLALITY

SERUM ALBUMIN

SERUM URIC ACID BUN/CREATININE HEMATOCRIT NIHSS OUTCOME SCORE

NIHSS OUTCOME SCORE:

```
DAY 1
                                                    DAY 3
                                                                    DAY 7
1a.LEVEL OF CONSCIOUSNESS(0 1
23)
1b.LOC Questions(0 1 2)
1c.LOC COMMANDS(0 1 2)
2.Best Gaze(0 1 2)
3.VISUAL(0 1 2 3)
4.Fascial palsy(0 1 2 3)
5a.Motor arm -Left(0 1 2 3 4)
5b.Motor arm-Right(0 1 2 3 4)
6a.motor Leg-Left(0 1 2 3 4)
6b.Motor Leg-Right(0 1 2 3 4)
7.Limb ataxia(0 1 2)
8.Sensory(0 1 2)
9.Best Language(0 1 2 3)
10.Dysarthria(0 1 2)
11.Extinction and Inattention(0 1 2)
```

- 0 Normal functioning
- 4- completely impaired

OUTCOME OF THE PATIENT: Death / Recovery and discharge

ANNEXURE II PATIENT INFORMATION SHEET

SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION AND RESEARCH TAMAKA, KOLAR - 563101.

PATIENT INFORMATION SHEET

This information is to help you understand the purpose of the study

"A CROSS-SECTIONAL STUDY TO DETERMINE THE SHORT TERM OUTCOME OF CEREBRAL SALT WASTING SYNDROME AND SYNDROME OF INAPPROPRIATE ANTI-DIURETIC HORMONE SECRETION IN STROKE PATIENTS"

. You are invited to take part voluntarily in this research study, it is important that you read and understand the purpose, procedure, benefits and discomforts of the study.

What is the purpose of this study?

What are the various investigations being used? Are there any associated risks?

Absolutely no risks are associated with various investigations involved in this study such as Complete blood count.

Serum Electrolytes

Serum albumin

Urine osmolarity

What is the benefit for me as a participant?

Participation in this research study may not change the final outcome of stroke. However, patients in the future may benefit as a result of knowledge gained from this study. You will not be charged extra for any of the procedures performed during the research study. Your taking part in this study is entirely voluntary. You may refuse to take part in the study or you may stop your participation in the study at any time, without a penalty or loss of any benefits to which you were otherwise entitled before taking part in this study.

CONFIDENTIALITY

Your medical information will be kept confidential by the study doctor and staff and will not be made publicly available. Your original records may be reviewed by your doctor or ethics review board. For further information/ clarification please contact

DR. KAVYA.B.K ,SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION AND RESEARCH, TAMAKA, KOLAR - 563101.

Contact no: 9886972761 to DrKavya

ANNEXURE III

INFORMED CONSENT FORM

SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION AND RESEARCH, TAMAKA, KOLAR - 563101.

ANTI-DIURETIC HORMONE SECRETION IN STROKE PATIENTS "

Case no:

<u>IP no</u>:

TITLE:

Primary Investigator/ Doctor:

INFORMED CONSENT FORM

"A CROSS-SECTIONAL STUDY TO DETERMINE THE SHORT TERM OUTCOME OF CEREBRAL SALT WASTING SYNDROME AND SYNDROME OF INAPPROPRIATE

maat anadroota tusimaas in tha Danson			
post graduate trainees in the Depar	tment of General Medici	ne and authorize	e the collection
and disclosure of personal information	tion as outlined in this co	nsent form.	
I understand the purpose of this stu	dy, the risks and benefits	of the techniqu	e and the
confidential nature of the informati	ion that will be collected	and disclosed d	uring the study.
The information collected will be u	used only for research.		
I have had the opportunity to ask q	uestions regarding the va	rious aspects of	this study and my
questions have been answered to m	ny satisfaction.		
I understand that I remain free to v	withdraw the participation	n from this stud	y at any time and
this will not change the future care			
\mathcal{L}	•		
Participation in this study does not		me.	
S		Date	Time
Participation in this study does not	involve any extra cost to		Time

ಶ್ರೀದೇವರಾಜ್ ಯುಆರ್ಎಸ್ ಉನ್ನತ ಶಿಕ್ಷಣ ಮತ್ತು ಸಂಶೋಧನಾ ಸಂಸ್ಥೆ, ತಾಮಾಕ, ಕೋಲಾರ್ - 563101.

ತಿಳಿವಳಿಕೆಯ ಸಮ್ಮತಿ ನಮೂನೆ

ಕೇಸ್ ಸಂಖ್ಯೆ:

ಐಪಿ ಸಂಖ್ಯೆ:

ಈಮಾಹಿತಿಯುಅಧ್ಯಯನದಉದ್ದೇಶವನ್ನು ಅರ್ಥಮಾಡಿಕೊಳ್ಳಲುನಿಮಗೆಸಹಾಯಮಾಡುತ್ತದೆ,

"ಹೈಪೋನಾಟ್ರೆಮಿಯಾಸ್ಟ್ರೋಕ್ರೋಗಿಗಳಲ್ಲಿಎಸ್ಐಎಡಿಎಚ್ಮತ್ತುಸೆರಿಬ್ರಲ್ಸಾಲ್ಟ್ವಾಸ್ಟಿನ್ಸಿಂಡ್ರೋಮ್ಭಿನ್ನತೆಕಂಡುಹಿಡಿಯಬೇಕು".ಈ ಸಂಶೋಧನಾಅಧ್ಯಯನದಲ್ಲಿಸ್ವಯಂಪ್ರೇರಿತವಾಗಿಪಾಲ್ಗೊಳ್ಳಲುನಿಮ್ಮನ್ನು ಆಹ್ವಾನಿಸಲಾಗಿದೆ, ಅಧ್ಯಯನದಉದ್ದೇಶ,

- ಕಾರ್ಯವಿಧಾನ, ಪ್ರಯೋಜನಗಳುಮತ್ತುಅಸ್ವಸ್ಥತೆಗಳನ್ನು ನೀವುಓದುವುದುಮತ್ತುಅರ್ಥಮಾಡಿಕೊಳ್ಳುವುದುಮುಖ್ಯವಾಗಿದೆ.
- 1. ಈಅಧ್ಯಯನದಉದ್ದೇಶವೇನು?
- 2. ವಿವಿಧತನಿಖೆಗಳನ್ನು ಬಳಸಲಾಗುತ್ತಿದೆ? ಯಾವುದೇಸಂಬಂಧಿತಅಪಾಯಗಳಿವೆಯೇ? ಈಅಧ್ಯಯನದಂತಹಹಲವಾರುತನಿಖೆಗಳೊಂದಿಗೆಯಾವುದೇಅಪಾಯಗಳುಸಂಪೂರ್ಣವಾಗಿಸಂಬಂಧಿಸಿಲ್ಲ
- a)ಸಂಪೂರ್ಣರಕ್ಕಎಣಿಕೆ.
- b)ಸೀರಮ್ಅಲ್ಪುಮಿನ್
- c)ಮೂತ್ರದಆಸ್ಕ್ರೋಲರಿಟಿ
- d)ಸೀರಮ್ಕ್ರಿಯೇಟಿನೈನ್.
- e)ಎಕ್ಸ್ಪೇ-ಎದೆ
- 3. ಭಾಗವಹಿಸುವವನಾಗಿನನಗೆ ಎನುಪ್ರಯೋಜನ?

ಈಸಂಶೋಧನೆಯಅಧ್ಯಯನದಲ್ಲಿಭಾಗವಹಿಸುವಿಕೆಯುನಿಮ್ಮದೀರ್ಘಕಾಲದಬೆನ್ನಿನನೋವಿನಅಂತಿಮಫಲಿತಾಂಶವನ್ನುಬದಲಿ ಸಬಾರದು. ಆದಾಗ್ಯೂ,

ಭವಿಷ್ಯದಲ್ಲಿರೋಗಿಗಳುಈಅಧ್ಯಯನದಿಂದಪಡೆದಜ್ಞಾನದಫಲಿತಾಂಶವಾಗಿಪ್ರಯೋಜನಪಡೆಯಬಹುದು.

ಸಂಶೋಧನಾಅಧ್ಯಯನದಸಮಯದಲ್ಲಿನಡೆಸಿದಯಾವುದೇಪ್ರಕ್ರಿಯೆಗಳಿಗೆನಿಮಗೆಹೆಚ್ಚುವರಿಶುಲ್ಕವಿಧಿಸಲಾಗುವುದಿಲ್ಲ. ಈಅಧ್ಯಯನದಲ್ಲಿನಿಮ್ಮಪಾಲ್ಗೊಳ್ಳುವಿಕೆಯುಸಂಪೂರ್ಣವಾಗಿಸ್ವಯಂಪ್ರೇರಿತವಾಗಿದೆ.

ಅಧ್ಯಯನದಲ್ಲಿಪಾಲ್ಗೊಳ್ಳಲುನೀವುನಿರಾಕರಿಸಬಹುದುಅಥವಾಈಅಧ್ಯಯನದಲ್ಲಿಪಾಲ್ಗೊಳ್ಳುವುದಕ್ಕೆ ಮುಂಚಿತವಾಗಿನೀವುಯಾ ಪುದೇಅರ್ಹತೆಯಿಂದಯಾವುದೇದಂಡಅಥವಾನಷ್ಟವಿಲ್ಲದೆಯೇ,

ಯಾವುದೇಸಮಯದಲ್ಲಿನಿಮ್ಮಪಾಲ್ಗೊಳ್ಳುವಿಕೆಯನ್ನು ಅಧ್ಯಯನದಲ್ಲಿನಿಲ್ಲಿ ಸಬಹುದು.

ಗೌಪ್ಕತೆ

ನಿಮ್ಮವೈದ್ಯಕೀಯಮಾಹಿತಿಯನ್ನುಅಧ್ಯಯನದವೈದ್ಯರುಮತ್ತುಸಿಬ್ಬಂದಿಗೌಪ್ಯವಾಗಿಡಲಾಗುವುದುಮತ್ತುಸಾರ್ವಜ ನಿಕವಾಗಿಲಭ್ಯವಿರುವುದಿಲ್ಲ.

ನಿಮ್ಮಮೂಲದಾಖಲೆಗಳನ್ನು ನಿಮ್ಮವೈದ್ಯರುಅಥವಾನೈ ತಿಕವಿಮರ್ಶೆ ಮಂಡಳಿಪರಿಶೀಲಿಸಬಹುದು.

ಹೆಚ್ಚಿನಮಾಹಿತಿಗಾಗಿ / ಸೃಷ್ಟೀಕರಣಕ್ಕಾಗಿದಯವಿಬ್ಬಡಾ.ಜೌಲೌದುಪ್ರದೀಪ್, ಶ್ರೀ

ಶಿಕ್ಷಣಮತ್ತುಸಂಶೋಧನಾಸಂಸ್ಥೆ, ತಮಾಕಾ, ಕೋಲಾರ್ - 563101 ಅಕಾಡೆಮಿಸಂಪರ್ಕಿಸಿ

ಡಾ. ಕಾವ್ಯಾ.ಬಿ.ಕೆ 9886972761 ಸಂಪರ್ಕಿಸಿಶ್ರೀದಿವಾರಾಯುಆರ್ಎಸ್ಉನ್ನ ತಶಿಕ್ಷಣಮತ್ತುಸಂಶೋಧನಾಸಂಸ್ಥೆ, ತಾಮಾಕ, ಕೋಲಾರ್ - 563

	AGE SEX		KE COMPLA	AINTSCOMOR	BID PULSE											SIADH DEATH/C
SHARF UNNISA	80 F	930650 I		12,3		80 140/90	127	3.3	2.4	2	12.8	35.7	10	10	9 S	C
LAKSHMAKKA	60 F	930420 I		1	2	84 100/60	127	3.8	4.2	4	22	38.6	14	13	11 S	C
BYRAMMA	90 F	932788 I		5	3	86 150/90	108	4.7	4.2	2.1	74	42.5	11	12	15 C	C
ERIYAMMA	65 F	936153 I		4 2,3		84 140/90	126	6.3	2.4	3	19.71	34.4	17	13	13 S	C
BASAPPA	60 M	942999 I	1,3		4	80 140/90	126	3.8	4.5	1.6	10	48.1	11	9	9 C	C
GIRIJA	35 F	943281 I		4 1,3		110 190/100	128	3.9	2.5	2.5	16	29.4	11	10	10 S	C
CHALAPATHY	50 M	943561 I	2,4		3	110 200/100	107	4.4	5.6	2	24	45.5	11	11	11 C	C
GIDDAPPA	76 M	944229 I		1 2,3		98 100/60	133	4.1	2.4	2.2	8.77	21.4	4	4	48	C
MUNISHAMAPPA	65 M	944241 I	1,6	1,2,3		100 112/60	134	4.2	4	2	31.53	32.6	11	11	9 S	C
VINODKUMAR	30 M	944588 H	1,0	1	5	84 140/90	128	4.5	5.6	1.2	21.7	48.1	13	11	8 C	C
KRISHNAMURTHY		944679 I		- ;	2											C
	62 M			1	2	84 110/70	132	4.7	3.5	2.4	22.32	38.7	6	6	48	
BHARATH	26 M	944901 I		- 1	3	84 160/100	127	3.5	3.5	3.7	8.3	42.7	- /	4	3 S	С
MARIYAPPA	60 M	944909 I		1 2,3		84 140/90	107	4.5	5.6	2	20.23	50.3	8	8	8 C	C
SOKAPPA	70 M	9454221		2,3		90 140/90	132	4.5	4.3	2.6	19.42	54	12	12	13 C	C
NANDINI	27 F	945502 H		1 2,3		84 110/70	120	3.9	3.9	2.2	14	38.6	12	10	10 S	C
NARASIMHAPPA	60 M	945520 H		12,3		84 160/100	126	4.1	3.5	2.5	14.6	46.9	10	10	10 C	C
MUNIYAMMA	70 F	945542 H		1	3	84 180/100	127	4.4	3.6	2.6	20	31.1	10	8	10 S	C
UJINAPPA	48 M	9457901		12.6		84 90/60	118	4.6	3.5	3	15.5	42.8	12		08	C
071111111														12		
MANJUNATH	36 M	945849 H		4	3	84 160/70	112	4.7	3.6	2.8	16.8	42	13	3	4 S	C
NAGAMMA	50 F	946779 I		1 2,3		84 140/90	114	4	5.6	3.2	18.68	34.2	12	12	12 C	C
RAJAMMA	67 F	947060 I		1	3	84 100/60	111	2.6	3.7	1.7	17.5	27	17	17	9 S	C
ANANDA	24 M	947094 I		1 2,3		82 90/50	113	4	4.7	2.3	43	40.8	14	16	18 C	C
MUNILAKSHMAMMA	60 F	947453 I		1	2	90 110/70	116	4.1	3.8	3	30	34.4	20	11	5 S	C
SIRISHA	47 F	938090 I		1	2	84 190/80	120	4.3	4.2	3.3	21.6	31.1	20	19	12 S	C
				1	3											
CHALAPATHI	50 M	948573 H		4	3	80 186/100	114	3.9	6	1.9	28.57	38	19	19	18 C	C
RADHA	85 F	927668 I		1	3	98 140/80	117	6.1	5	2.2	16.6	25.3	23	20	10 S	C
NARAYANA SHETTY	85 M	927507 I		4		86 140/90	112	3.9	3.8	3.4	18.75	35.8	20	16	8 S	C
SHIVAKUMAR	40 M	9449051	1,6	2,3		84 130/80	118	4.8	3.6	2.6	13.56	38.6	15	15	9 S	C
MUNIYAPPA	60 M	950545 I		1 2,3		80 130/70	115	4.5	4	2.5	15.1	39.9	19	19	20 S	D
LAKSHMAMMA	55 F	928751 I		3	2	86 160/100	112	3.7	3.8	2.3	12	36	10	8	3 S	C
		950881 I		í	1					2.4		45	14	16	18 C	C
VENKATLAKSHMAMM/				- 1	1	84 112/70	110	3.5	5.6		30					
KRISHNAMURTHY	75 M	50683 I		1	4	87 140/90	115	5.8	5.4	2.6	36.6	55	- 11	9	7 C	С
MUNIYAPPA	65 M	72239 I		3 1,2		86 160/100	118	4	4	2	27.5	50	4	0	18	C
KRISHNAMURTHY	72 M	76132 I		1	2	86 130/80	109	6.6	5.4	2.6	36.6	56	21	20	22 C	D
SUBRAMANI	38 M	66284 H		2	3	84 186/100	122	3.8	4.1	3	30	45	7	4	0 S	C
BHAGYAMMA	45 F	66757 H		2		86 180/100	120	3.5	4.1	3	20	45	7	4	0.5	C
SURENDRA	40 M	50265 H		4	5	98 210/100	128	5.5	5	2.3	18	55	6	0	3 C	C
				2	2				2.0	2.3		45	6	2		C
SHAMAN KUMAR	59 M	49955 H		4	3	96 190/100	120	4.5	3.5	3	55		0	3	0.8	-
NAGARATHNAMMA	54 F	49808 H		1 2,3		84 180/100	117	4	4.6	2.4	22	50	4	3	3 S	C
FAHAMIDA BANU	33 F	49657 I		1		84 140/90	124	3.5	4.1	3	25	42	6	4	48	C
BYRE REDDY	57 M	49436 I		5	1	80 130/80	112	3.9	3.4	2	15.5	50	3	2	2 S	C
SHIVAPPA	60 M	48892 H		2 3,4		84 190/110	126	4	3.4	2	15.7	50	3	3	3 S	C
KRISHNAPPA	40 M	48904 H		3	3	90 170/110	110	4.2	4.2	4	37.5	45	5	1	18	C
NAGAMANI	30 F	48529 H		1	3	84 180/100	128	5	5	1.8	27.2	50	9	2	2 C	C
				2	3			3.7					8	2		C
YASHODA	50 F	47801 I				84 140/70	129	3.6	3.6	2	35.6	46	4	0	0.8	С
NAGARAJA	45 M	21917 I		4	3	74 130/80	130	3.6	4.1	2.1	13.7	48	3	0	0 S	C
AMBAREESH	30 M	47090 H	1,6			84 144/90	120	3.6	4.2	2.1	15	45	6	6	4 S	C
KRISHNAPPA	50 M	469161		1 2,3		86 140/90	116	4.1	4.2	2	5	50	4	4	3 S	C
JESTIN	43 M	46334 I		5	5	86 130/80	112	3.7	5	2	11.8	50	3	3	18	C
MANJULA	45 F	462101		4	4	84 130/80	125	3.5	3.6	3.2	11.2	40	4	4	2 S	C
MOHAN	61 M	46036 I	1,3	1,3		84 140/90	108	5.5	5.2	1.9	13.6	52	12	0	6C	C
							110					58		10		C
ANAND REDDY	62 M	45802 I	1,4	1,4		86 160/100		5.2	5.1	1.5	11.53		18	10	10 C	
PRAMOD	47 M	45801 I		3	3	84 170/100	131	3.5	3.5	3.2	20	40	2	1	0 S	C
VENKATESHAPPA	55 M	45354 I		3	2	84 130/80	134	3.4	3.4	2.5	13.3	45	2	1	0 S	C
BHAGYAMMA	52 F	45268 H		5	3	84 130/90	130	3.5	3.8	2.2	11.36	42	2	1	1 S	C
VENKATESHAPPA	78 M	44938 I	1,6	2,3		84 150/90	110	5.8	5.2	1.2	10.7	60	10	8	7 C	C
SUBRAMANI	57 M	43911 H		23,5		84 190/110	122	3.8	4.1	2.4	10	40	14	4	3 S	C
ANWAR	47 M	43130 H		1	3	89 160/100	132	3.5	4.1	2	22	42	5	4	2 S	C
			1.7	2.4	- 3			3.3					17	12		-
NARAYANAPPA	72 M	42484 I	1,6	3,4		84 140/90	122	4	3.6	3.2	10.7	40	16	12	10 S	C
MOKSHITH	40 M	42026 H		2	3	86 210/110	128	4.2	4	2.5	14.4	45	4	4	0 S	C
DEENADAYALAN	76 M	41776 I	1,6	2,3,4		86 150/100	120	6	5.2	2.1	16.53	53	15	14	7 C	C
RAMAKKA	80 F	41566 I		3	3	80 140/90	125	3.5	4.2	2.6	20.83	50	4	2	18	C
RATNAMMA	60 F	41368 H		2	3	96 160/100	124	3.6	4.4	3.2	15.38	40	7	0	0.8	C
VENKATLAKSHMI	49 F	409161		1	2	90 130/80	130	3.8	5	2.1	25	41	3	3	2 S	C
RAMASWAMY	71 M	405451	1.4	2,3		86 150/100	114	3.6	4	2.5	11.1	49	15	14	10 S	C
			1,6	2,5	,				4				13			
RAMACHANDRAPPA	51 M	40330 I		5	4	90 140/90	130	3.5	4	2.5	12.5	46	4	3	18	C
SAMSON	63 M	40065 I	1,4	2,3		90 150/100	109	5.4	5.1	2	10.97	54	21	13	8 C	C
PRATAP	60 M	39997 H		1		86 160/100	126	3.8	4	3	10.66	44	6	6	48	C
PARVATAMMA	58 F	39614I		3 2,3		84 140/90	132	3.6	4.6	2.2	18.3	42	2	1	0 S	C
MUNIVENKATAMMA	80 F	38266 I	3,6	2,3		90 160/100	132	3.9	4.6	4	14.28	50	8	5	48	C
RAGHU	34 M	38039 I	-,"	5	4	92 140/100	111	4.2	4.1	2.2	27.5	49	7	5	2 S	C
MAHALAKSHMI	60 F	37947 I	1,6	-	2	90 140/90	102	6.2	6	1.4	83.3	60	11	10	10 C	C
SUMITRAMMA	70 F	37398 H	1,0	2	3	102 190/110	126	4.2	é	2.2	18.33	38	- 2	2	08	C
	70 F			122	3		112		5.4	2.8		58	1.4	14		C
MUNIYAPPA		952281		1 2,3	4	94 160/100		3.9	5.4		16.25		14	14	14 C	C
KRISHNAPPA	65 M	952008 I	-	3	4	90 140/90	130	3.6	4.1	2.1	13.75	46	2	1	18	C
LAKSHMIDEVI	45 F	951789 I	1,6		2	72 120/70	122	3.9	4	3.9	14.28	42	7	6	5 S	C
RAGHUNATH	60 M	73119 I		5 4,6		102 140/90	132	4.2	4	2.6	31.25	49	2	2	1 S	C
KRISHNAVENI	50 F	930925 I		1 2,3		94 130/80	130	4.6	4	2.2	20	41	10	10	8 S	C
ARCHANA	30 F	929807 I	1,6		5	96 130/80	132	3.6	3.7	2.5	20.83	38	6	6	5 S	C
RADHIKA	52 F	9366401	1,6		2	79 140/80	130	3.6	3.6	2.5	20	44	9	9	88	C
DILSHAD	40 F	731231	.,.,	3	4	94 130/80	131	3.6	3.6	2.5	16.25	50	6	- 6	08	C
VENKATESHAPPA			1.6	1.0	-									2		-
	80 M	731121	1,6	1,3		92 150/100	129	3.5	3.5	2	26.6	44	10	- /	7 S	C
ASHWINI	45 F	75612 I		3	5	96 126/70	131	3.6	4.1	3.1	16.2	36	5	4	18	C
VASANTHA	48 F	66751 H		2	3	120 190/110	133	3.9	3.8	2.1	18.84	40	6	6	48	C
MUNIRAJU	38 M	75588 I	1,6			89 152/90	127	4.2	4.1	2.3	20	50	8	6	5 S	C
VENKATACHALAPATHI	40 M	75606 H		3	3	98 184/110	115	3	3.2	2	7.14	44	4	3	18	C
ANJANAPPA	38 M	76084 H		6		90 170/100	126	3.2	3.5	2.2	20	48	6	4	3 S	C
			1.4		1							40	2	4		
ZAREENA BEGUM	60 F	66478 H	1,4		- 1	78 160/100	132	3.5	3.7	2.2	14.68		7	4	2 S	C
VENKATARAYAPPA	75 M	67381 H	1,3,6	2,3		96 190/102	109	5.2	3.2	0.9	9.37	54	23	21	21 C	D
NARASAPPA	55 M	67086 I		3	1	92 140/90	117	3.4	3.8	2	12.5	46	2	2	1 S	C
VIJAY KUMAR	45 M	67880 H		2	6	92 140/90	120	4	4.2	1.6	14.54	49	7	0	0 S	C
KRISHNAMOHAN	62 M	681661		1 2,3		94 160/100	107	3.2	3.5	1.9	19.23	40	8	8	5 S	C
HANUMAKKA	70 F	68604 I		1	1	92 140/90	107	5.4	3.6	2.1	19.23	58	16	16	15 C	C
				600	-			3.4					10	10		
VENKATANNA	85 M	66561 I		5 2,3		92 150/90	130	4	3.8	2.6	21.66	38	8	- 1	0.8	C
	72 M	70139 H		3	3	86 152/100	130	3.5	3.8	2.5	27.27	40	8	5	5 S	C
SOMEGOWDA	21 M	70810 I		6		90 130/70	130	3.8	3.5	3	20	28	3	0	0 S	C
SOMEGOWDA PONTULA SINGH	47 F	571241		3	2	90 130/90	128	3.6	3	3.1	21.42	22	4	0	0.8	C
PONTULA SINGH		9281831	1,4	2,3		90 190/100	112	5.2	2.2		18.9	34.6	23	23	23 C	C
PONTULA SINGH SARASWATHI			1,4	2,3			112	3.9	3.8	1.2	21.2	35.8	8	1		
PONTULA SINGH SARASWATHI VENKATESH GOWDA	75 M															
PONTULA SINGH SARASWATHI VENKATESH GOWDA NARAYAN SHETTY	75 M 85 M	927507 I		4		86 140/60									18	C
PONTULA SINGH SARASWATHI VENKATESH GOWDA NARAYAN SHETTY RADHA	75 M 85 M 85 F	927507 I 927668 I		1	3	98 140/80	117	6.1	5	2.5	18.33	25.3	15	15	15 C	C
PONTULA SINGH SARASWATHI VENKATESH GOWDA NARAYAN SHETTY	75 M 85 M	927507 I		1 2	3 1											

PARAMETER	KEY TO PARAMETER
COMPLAINTS	1-WEAKNESS
	2-SEIZURES
	3-GIDDINESS
	4-UNRESPONSIVENESS
	5-UNSTEADINESS OF GAIT
	6-DEVIATION OF ANGLE OF MOUTH
COMORBIDS	1-PRIOR STROKE
	2-DIABETES
	3-HYPERTENSION
	4-CARDIAC DISEASE
	5-EPILEPSY
	6-TUBERCULOSIS
OUTCOME OF PATIENT	D -DEATH
	R- RECOVERY AND DISCHARGE
CAUSE	S -SIADH
	C- CSW
STROKE TYPE	I - ISCHEMIC
	H- HEMORRHAGIC