Original Research

STUDY OF CEREBRAL SALT WASTING SYNDROME AND SYNDROME OF INAPPROPRIATE ANTIDIURETIC HORMONE SECRETION AMONG STROKE PATIENTS

Dr. K.P. Selvarajan Chettiyar[1], Dr. Abinesh N.L[2*]
[1]Professor, Department of General Medicine, Sree Mookambika Institute of Medical Sciences, Kulasekharam, Tamilnadu, India.
[2*]Junior Resident, Department of General Medicine, Sree Mookambika Institute of Medical Sciences, Kulasekharam, Tamilnadu, India.
Corresponding Author: Dr. Abinesh N.L
Junior Resident, Department of General Medicine, Sree Mookambika Institute of Medical Sciences, Kulasekharam, Tamilnadu, India.

ABSTRACT

Background: Hyponatremia is a common electrolyte abnormality in patients with Stroke and is associated with increased morbidity and poor neurological outcomes. Syndrome of inappropriate antidiuretic hormone secretion (SIADH) and cerebral salt wasting syndrome (CSWS) are the major causes of hyponatremia in stroke patients. Early differentiation between these conditions is important because their management differs significantly. This study aimed to evaluate the occurrence of SIADH and CSWS among patients with stroke.

Methodology: This hospital-based observational study was conducted in the Department of General Medicine at Sree Mookambika Institute of Medical Sciences from January 2025 to December 2026. Fifty patients with clinically and radiologically confirmed stroke were included in the study. Detailed clinical evaluation, biochemical investigations, serum sodium levels, urine sodium, and assessment of volume status were performed. Data were analyzed using SPSS version 20.0. Chi-square test and Student's t-test were used for statistical analysis, with $p < 0.05$ considered statistically significant.

Results: Hyponatremia was observed in 14% of stroke patients. Among patients with hyponatremia, 85.7% had SIADH and 14.3% had CSWS. Ischemic stroke constituted 80% of cases, and most patients had middle cerebral artery territory involvement. Severe hyponatremia (<125 mEq/L) was observed in 10% of patients. SIADH patients had normal volume status, whereas CSWS patients demonstrated hypovolemia and elevated BUN/creatinine ratio.

Conclusion: Hyponatremia is a significant complication in stroke patients, with SIADH being the most common etiology. Early diagnosis and differentiation between SIADH and CSWS are essential for appropriate management and improved neurological outcomes.

Keywords: Stroke, Hyponatremia, Siadh, Cerebral Salt Wasting Syndrome, Sodium Imbalance, Ischemic Stroke.

INTRODUCTION

Stroke is one of the leading causes of mortality and long-term neurological disability worldwide. It is associated with significant morbidity due to complications affecting multiple organ systems, including disturbances in fluid and electrolyte balance [1]. Among these complications, hyponatremia is one of the most frequently encountered electrolyte abnormalities in patients with acute stroke and is associated with increased mortality, prolonged hospital stay, neurological deterioration, and poor functional outcome [2].

www.ajmrhs.com
eISSN: 2583-7761

Date of Received: 22-05-2026
Date Acceptance: 30-05-2026
Date of Publication: 29-06-2026

Hyponatremia in stroke patients commonly results from either syndrome of inappropriate antidiuretic hormone secretion (SIADH) or cerebral salt wasting syndrome (CSWS), both of which present with similar biochemical findings but require entirely different treatment approaches [3].

SIADH is characterized by excessive release of antidiuretic hormone (ADH), resulting in water retention, dilutional hyponatremia, low serum osmolality, and inappropriately concentrated urine [4]. It commonly occurs in patients with intracranial disorders such as stroke, traumatic brain injury, infections, and malignancies. The persistent secretion of ADH despite normal or low plasma osmolality leads to euvolemic or mildly hypervolemic hyponatremia [5]. In contrast, cerebral salt wasting syndrome is characterized by excessive renal sodium excretion leading to hypovolemia and hyponatremia. CSWS is believed to result from impaired sympathetic neural input to the kidneys and increased release of natriuretic peptides following central nervous system injury

[6]. Although both conditions present with hyponatremia, elevated urine sodium, and high urine osmolality, accurate differentiation between SIADH and CSWS is essential because fluid restriction is the treatment of choice for SIADH, whereas aggressive fluid and sodium replacement are required in CSWS [7].

Stroke-related hyponatremia is particularly important because it can worsen cerebral edema, increase intracranial pressure, precipitate seizures, and impair neurological recovery [8]. Several studies have demonstrated that hyponatremia is more common in hemorrhagic stroke compared to ischemic stroke, likely due to greater hypothalamic and neuroendocrine involvement [9]. Furthermore, the severity of sodium imbalance has been associated with poor prognosis and increased risk of mortality among stroke patients [10]. Early recognition and appropriate management of electrolyte abnormalities may therefore improve neurological outcomes and reduce complications.

The differentiation between SIADH and CSWS remains clinically challenging because both disorders share overlapping laboratory features, including hyponatremia, elevated urine sodium concentration, and increased urine osmolality [11]. Assessment of volume status plays a crucial role in distinguishing the two conditions. Patients with SIADH are usually euvolemic, while patients with CSWS are hypovolemic with signs of dehydration such as hypotension, tachycardia, reduced skin turgor, and elevated hematocrit [12]. However, accurate clinical assessment of volume status may be difficult in critically ill neurological patients, resulting in underdiagnosis or misdiagnosis.

Despite the clinical significance of these disorders, limited studies have evaluated the prevalence and clinical profile of SIADH and CSWS among stroke patients in the Indian population. Understanding the occurrence and distinguishing features of these conditions is important for prompt diagnosis and appropriate therapeutic intervention. The present study was therefore undertaken to evaluate cerebral salt wasting syndrome and syndrome of inappropriate antidiuretic hormone secretion among patients with stroke and to analyze their clinical and biochemical characteristics [13].

Aim

To evaluate the occurrence and clinical profile of cerebral salt wasting syndrome and syndrome of inappropriate antidiuretic hormone secretion among patients with Stroke.

Objectives

1. To determine the prevalence of cerebral salt wasting syndrome (CSWS) and syndrome of inappropriate antidiuretic hormone secretion (SIADH) among patients with stroke.
2. To compare the occurrence of CSWS and SIADH between ischemic and hemorrhagic stroke patients.
3. To assess the clinical and biochemical characteristics of patients with CSWS and SIADH.

METHODOLOGY

This hospital-based observational study was conducted in the Department of General Medicine at Sree Mookambika Institute of Medical Sciences during the study period from January 2025 to December 2026. The study aimed to evaluate cerebral salt wasting syndrome (CSWS) and syndrome of inappropriate antidiuretic hormone secretion (SIADH) among patients diagnosed with Stroke. Patients aged 18 years and above with clinical features suggestive of stroke and confirmation by neuroimaging were included in the study after obtaining written informed consent. Diagnosis of stroke was established based on clinical examination and radiological investigations including computed tomography (CT) scan or magnetic resonance imaging (MRI) of the brain.

A pre-structured and pretested proforma was used for data collection. Baseline demographic details including age and sex were recorded for all participants. Detailed medical history including conventional risk factors such as hypertension, diabetes mellitus, smoking, alcohol consumption, dyslipidemia, and previous cerebrovascular events was obtained. Comprehensive clinical examination was performed, including neurological assessment and evaluation of volume status. Relevant laboratory investigations including serum electrolytes, serum osmolality, urine sodium, urine osmolality, renal function tests, blood glucose levels, and other routine biochemical parameters were carried out. Patients with hyponatremia were further evaluated for the presence of SIADH or CSWS based on clinical findings, biochemical profile, and assessment of volume status.

Data were entered into Microsoft Excel and analyzed using Statistical Package for Social Sciences (SPSS) software version 20.0. Continuous variables were expressed as mean ± standard deviation (SD), while categorical variables were represented as frequencies and percentages. Results were presented using tables and figures wherever appropriate. Univariate analysis of dichotomous variables was performed using the Chi-square test with Yates correction whenever required. Student's t-test was used for comparison of continuous variables between groups. Statistical significance was considered at a p-value of less than 0.05.

RESULTS

Out of 50 patients, 50% were in the age group 41-60 years and 40% in the age group 61-80 years. The mean age of the study population was. Among 50 patients,45(90%) had motor weakness, 9(18%) had cranial nerve involvement, 7(14%) had speech disturbances, 5(10%) had loss of consciousness, 6(12%) had sensory disturbance,2(4%) had seizures, 2(4%) had cerebellar involvement and 2(4%) had bowel and bladder involvement.

Table 1: Association of Demographic Variables & Clinical Variables with Hyponatremia

Clinical Variables	**HYPONATREMIA**		
	Without [n=43]	**SIADH [n=1]**	**CSW [n=6]**
AGE			
21 -40	7%	0%	0%
%			
41 – 60	46.5%	66.7%	100%
%			
61 – 80	44.2%	16.7%	0%
%			
>80	2.3%	16.7%	0%
%			
GENDER			
Male	67.4	50	100
%			
Female	32.6	50	0
%			
Types of Stroke			
Ischemic % Hemorrhagic %	79.1 20.9	83.3 16.7	100 0
TERRITORY			
ACA	4.7	0	0
%			
MCA	83.7	66.7	100
%			
PCA	4.7	16.7	0
%			
MCA-ACA	2.3	0	0
%			
MCA-PCA	4.7	16.7	0
%			
SIDE			
RIGHT	34.9	33.3	0
%			
LEFT	62.8	66.7	100
%			
BILATERAL	2.3	0	0
%			

Among 50 patients with stroke, 52% had DM, 50% had HTN, 30% had both DM and HTN, 6% had IHD and 4% had epilepsy.

Among 50 patients, 32% were smokers and 30% were alcoholics.

Among 50 patients, 40 (80%) patient had ischemic stroke and 10(20%) had hemorrhagic stroke. The majority of patients in our study i.e., 41 patients (82%), had stroke corresponding to the Middle Cerebral Artery territory.

In our study, most of the patients had Right sided stroke 64% (n=32), 34% had left sided stroke (n=17) and 2% (n=1) had bilateral involvement.

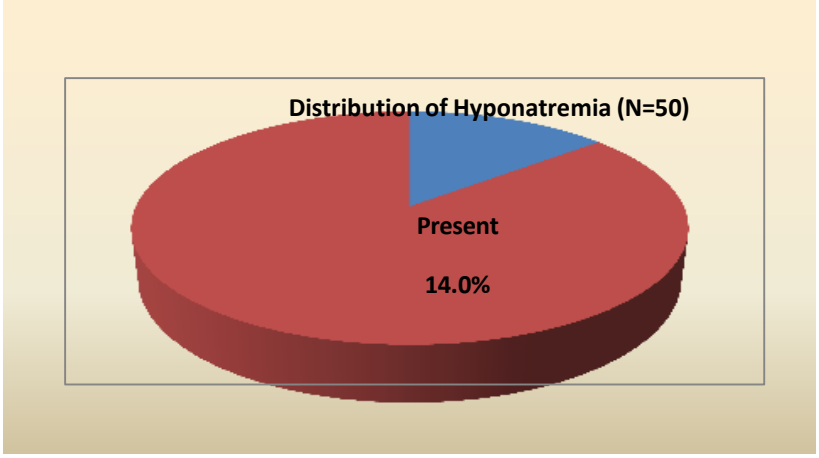

Figure 1. Incidence of Hypoatremia

Out of 50 patients, hyponatremia was present in 7(14%) of patients.

Table 2: Severity of Hyponatremia

Serum sodium in mEq/l	n	(%)
>134	42	84%
130-134	1	2%
125-129	2	4%
<125	5	10%
Total	50	100%

Among 50 patients, 42 (84%) had normal serum sodium, 1(2%) had serum sodium 130-134, 2(4%) had serum sodium 125-129 and 5(10%) had serum sodium <125Meq/l Of the 50 patients, 85.7% had SIADH (n=6) and 14.3% had CSW (n=1).

Table 3. Mean Sodium Levels

	n	mean	SD	MIN	MAX
SIADH	6	121.67	7.94	106	125
CSW	1	123	-	123	123
Normal	43	138.74	3.88	134	148
Total	7	136.38	7.37	106	148

Patients with SIADH group 6(100%) had normal volume status and patients with CSW group 1(100%) had decreased volume status. Patients with SIADH group 6(100%) had normal BUN/creatinine ratio and patients with CSW group 1(100%) had increased BUN/creatinine ratio. Patients with SIADH group 4(60%) had normal HCT,2(40%) had decreased HCT and patients with CSW group 1(100%) had increased HCT. Patients with SIADH group 6(100%) and CSW group 1(100%) had normal serum albumin. Patients with SIADH group 6(100%) and CSW 1(100%) had increased urine sodium levels.

In general, the occurrence of hyponatremia was noted most in the 41-60 year age group i.e., 71.4% (n=5), 14.2%(n=1) in 61-80 year age group and 14.2%(n=1) in the > 80 year age group. The difference in the hyponatremia prevalence among the various age groups didn't reached statistical significance (p>0.05)

In the SIADH group, males 50% (n=3) and females 50% (n=3) equally distrubuted.

In the CSW group males comprised 0f 100%(n=1). The variation in sex distribution among the various groups did not reach statistical significance.(p > 0.05).

Out of the Ischemic stroke patients 15% had hyponatremia. (n=6). Hyponatremia was seen in 10% (n=1) of patients with haemorrhagic stroke.

Among patients with hyponatremia, 85.7% had ischemic stroke (n=6) and 14.3% had haemorrhagic stroke (n=1).

Among SIADH patients, 83.3% had ischemic stroke and 16.7% had hemorrhagic stroke. Among cerebral salt wasting patients, ischemic stroke was found in 100% of patients.

The difference in stroke type seen in the various conditions did not

Reach statistical significance (p>0.05).
In the MCA territory stroke patients, 12% developed hyponatremia. 33% of MCA-PCA territory stroke patients developed hyponatremia, while Hyponatremia was seen in 33% of the Posterior Cerebral Artery territory stroke.

DISCUSSION

The present study evaluated the occurrence of hyponatremia, syndrome of inappropriate antidiuretic hormone secretion (SIADH), and cerebral salt wasting syndrome (CSWS) among patients with Stroke. Hyponatremia was observed in 14% of stroke patients, which is consistent with previous studies reporting that electrolyte imbalance is a common complication in acute stroke and significantly influences neurological outcome and mortality [14]. Hyponatremia in stroke patients may occur due to neuroendocrine dysfunction, cerebral edema, increased intracranial pressure, and altered hypothalamic regulation following brain injury [15].
In the present study, SIADH was identified as the predominant cause of hyponatremia, accounting for 85.7% of cases, whereas CSWS was observed in 14.3% of patients. Similar observations have been reported in earlier studies where SIADH was found to be more common than CSWS among patients with acute neurological disorders [16]. The higher prevalence of SIADH may be explained by inappropriate secretion of antidiuretic hormone secondary to hypothalamic stimulation caused by cerebral ischemia or hemorrhage [17]. Correct differentiation between SIADH and CSWS is clinically important because the management strategies differ significantly. Fluid restriction is recommended in SIADH, while aggressive salt and fluid replacement is necessary in CSWS [18].
The majority of patients with hyponatremia in the present study belonged to the 41–60 years age group. However, the association between age and occurrence of hyponatremia was not statistically significant. Similar findings have been reported by previous studies where middle-aged and elderly stroke patients were more prone to electrolyte disturbances due to associated comorbidities and impaired physiological reserve [19]. Males constituted the majority of stroke patients in the present study, although gender distribution among SIADH and CSWS groups did not show statistical significance. This may be attributed to the higher prevalence of vascular risk factors such as smoking, hypertension, and alcohol consumption among males [20]. Among the study population, ischemic stroke was more common than hemorrhagic stroke, accounting for 80% of cases. Hyponatremia was slightly more frequent among ischemic stroke patients compared to hemorrhagic stroke patients. Similar findings have been observed in studies suggesting that ischemic stroke involving the hypothalamic and middle cerebral artery territories may precipitate SIADH due to neurohormonal dysregulation [21]. In the present study, the majority of strokes involved the middle cerebral artery territory, which is known to be associated with autonomic dysfunction and disturbances in sodium regulation [22].
The severity analysis showed that most hyponatremic patients had severe sodium reduction, with 10% of patients demonstrating serum sodium levels below 125 mEq/L. Severe hyponatremia in stroke patients is clinically significant because it may worsen cerebral edema, increase intracranial pressure, precipitate seizures, and impair neurological recovery [23]. The mean serum sodium level in the SIADH group was significantly lower compared to patients without hyponatremia, reflecting the severity of water retention associated with inappropriate ADH secretion.
Volume status assessment played an important role in differentiating SIADH from CSWS in the present study. All patients with SIADH had normal volume status, whereas the patient with CSWS had clinical evidence of hypovolemia and elevated blood urea nitrogen (BUN)/creatinine ratio. Increased hematocrit observed in the CSWS patient further supported the diagnosis of volume depletion. Similar findings have been described in previous literature emphasizing the role of clinical assessment and biochemical parameters in distinguishing these two entities [24].
The present study highlights the importance of routine monitoring of serum sodium levels and careful evaluation of hyponatremia among stroke patients. Early identification and differentiation of SIADH and CSWS are essential for initiating appropriate therapy and preventing neurological deterioration. Prompt correction of sodium imbalance may improve clinical outcomes, reduce complications, and shorten hospital stay in patients with acute stroke [25].

CONCLUSION

The present study demonstrated that hyponatremia is a relatively common electrolyte abnormality among patients with Stroke, with syndrome of inappropriate antidiuretic hormone secretion (SIADH) being the predominant underlying cause compared to cerebral salt wasting syndrome (CSWS). Hyponatremia was more frequently observed in patients with ischemic stroke and middle cerebral artery territory involvement. Although the associations between demographic variables, stroke type, and hyponatremia were not statistically significant, severe hyponatremia was

associated with important clinical and biochemical abnormalities. Accurate differentiation between SIADH and CSWS based on volume status and laboratory parameters is essential because the management strategies for these conditions differ significantly. Early recognition and prompt correction of sodium imbalance may help reduce neurological complications, improve clinical outcomes, and decrease morbidity among stroke patients.

REFERENCES

1. Feigin VL, Norrving B, Mensah GA. Global burden of stroke. Circ Res. 2017;120(3):439-448.
2. Liamis G, Barkas F, Megapanou E, Elisaf M, Milionis H. Hyponatremia in acute stroke patients: pathophysiology, clinical significance, and management. Clin Neurol Neurosurg. 2019;179:1-7.
3. Sherlock M, O'Sullivan E, Agha A, Behan LA, Rawluk D, Brennan P, et al. Incidence and pathophysiology of severe hyponatraemia in neurosurgical patients. Postgrad Med J. 2009;85(1002):171-175.
4. Ellison DH, Berl T. The syndrome of inappropriate antidiuresis. N Engl J Med. 2007;356(20):2064-2072.
5. Verbalis JG, Goldsmith SR, Greenberg A, Korzelius C, Schrier RW, Sterns RH, et al. Diagnosis, evaluation, and treatment of hyponatremia. Am J Med. 2013;126(10 Suppl 1):S1-S42.
6. Palmer BF. Hyponatraemia in patients with central nervous system disease: SIADH versus cerebral salt wasting. Trends Endocrinol Metab. 2003;14(4):182-187.
7. Harrigan MR. Cerebral salt wasting syndrome: a review. Neurosurgery. 1996;38(1):152-160.
8. Saleem S, Anwar F, Hussain M, Rehmani MA. Frequency of hyponatremia in stroke patients and its impact on clinical outcome. Cureus. 2020;12(9):e10391.
9. Kusuda K, Saku Y, Sadoshima S, Fujishima M. Disturbances of fluid and electrolyte balance in patients with acute stroke. Jpn Circ J. 1989;53(9):1092-1097.
10. Soiza RL, Cumming K, Clarke JM, Wood KM, Myint PK. Hyponatremia: special considerations in older patients. J Clin Med. 2014;3(3):944-958.
11. Yee AH, Burns JD, Wijdicks EF. Cerebral salt wasting: pathophysiology, diagnosis, and treatment. Neurosurg Clin N Am. 2010;21(2):339-352.
12. Misra UK, Kalita J, Singh SK. A study of hyponatremia in tuberculous meningitis. J Neurol Sci. 2016;367:152-157.
13. Sherlock M, Thompson CJ. The syndrome of inappropriate antidiuretic hormone: current and future management options. Eur J Endocrinol. 2010;162 Suppl 1:S13-S18.
14. Liamis G, Barkas F, Megapanou E, Elisaf M, Milionis H. Hyponatremia in acute stroke patients: pathophysiology, clinical significance, and management. Clin Neurol Neurosurg. 2019;179:1-7.
15. Saleem S, Anwar F, Hussain M, Rehmani MA. Frequency of hyponatremia in stroke patients and its impact on clinical outcome. Cureus. 2020;12(9):e10391.
16. Sherlock M, O'Sullivan E, Agha A, Behan LA, Rawluk D, Brennan P, et al. Incidence and pathophysiology of severe hyponatraemia in neurosurgical patients. Postgrad Med J. 2009;85(1002):171-175.
17. Ellison DH, Berl T. The syndrome of inappropriate antidiuresis. N Engl J Med. 2007;356(20):2064-2072.
18. Palmer BF. Hyponatraemia in patients with central nervous system disease: SIADH versus cerebral salt wasting. Trends Endocrinol Metab. 2003;14(4):182-187.
19. Soiza RL, Cumming K, Clarke JM, Wood KM, Myint PK. Hyponatremia: special considerations in older patients. J Clin Med. 2014;3(3):944-958.
20. Feigin VL, Norrving B, Mensah GA. Global burden of stroke. Circ Res. 2017;120(3):439-448.
21. Kusuda K, Saku Y, Sadoshima S, Fujishima M. Disturbances of fluid and electrolyte balance in patients with acute stroke. Jpn Circ J. 1989;53(9):1092-1097.
22. Yee AH, Burns JD, Wijdicks EF. Cerebral salt wasting: pathophysiology, diagnosis, and treatment. Neurosurg Clin N Am. 2010;21(2):339-352.
23. Verbalis JG, Goldsmith SR, Greenberg A, Korzelius C, Schrier RW, Sterns RH, et al. Diagnosis, evaluation, and treatment of hyponatremia. Am J Med. 2013;126(10 Suppl 1):S1-S42.
24. Harrigan MR. Cerebral salt wasting syndrome: a review. Neurosurgery. 1996;38(1):152-160.
25. Sherlock M, Thompson CJ. The syndrome of inappropriate antidiuretic hormone: current and future management options. Eur J Endocrinol. 2010;162 Suppl 1:S13-S18.

How to cite this article: Dr. K.P. Selvarajan Chettiyar, Dr. Abinesh N.L, STUDY OF CEREBRAL SALT WASTING SYNDROME AND SYNDROME OF INAPPROPRIATE ANTIDIURETIC HORMONE SECRETION AMONG STROKE PATIENTS, Asian J. Med. Res. Health Sci., 2026; 4 (2):1315-1321.
Source of Support: Nil, Conflicts of Interest: None declared.