Original Research

Association of Hyponatremia with Diabetes Mellitus in CVA Patients

Suman Chatterjee[1], Rajashree Pradhan[2], Lekha Biswas[3], Sajeeb Mondal[4]

[1]Assistant Professor, Department of Biochemistry, Rampurhat Government Medical College & Hospital, Rampurhat, https://orcid.org/0009_0005_4383_3365, [2]Associate Professor, Department of Pathology, College of Medicine & Sagore Dutta Hospital, Kolkata, West Bengal, India, https://orcid.org/0000_0001_6770_7367, [3]Associate Professor, Department of Biochemistry, Rampurhat Government Medical College & Hospital, Rampurhat, https://orcid.org/0009_0008_0077_7449, [4]Associate Professor, Department of Pathology, Rampurhat Govt. Medical College, Rampurhat, West Bengal, India, https://orcid.org/0000_0002_1597_8584.

How to cite this article: Suman Chatterjee, Rajashree Pradhan, Lekha Biswas et al. Association of Hyponatremia with Diabetes Mellitus in CVA Patients. Indian Journal of Public Health Research and Development / Vol. 16 No. 3, July-September 2025.

Abstract

Background: The prevalence of CVA in Type II Diabetes Mellitus and is significantly high due to its macrovascular complication. Hyponatremia is a common electrolyte disorder seen in patients of cerebrovascular accident (CVA) usually either due to Syndrome of inappropriate secretion of Antidiuretic hormone (SIADH) or cerebral salt wasting syndrome (CSWS). The aim of the study was to find out whether there is any relationship between the Type II Diabetes Mellitus and hyponatremia in stroke patients and its etiology.

Method: For determining the incidence and etiology of hyponatremia in patients of stroke admitted in the hospital, that study was conducted in College of Medicine & Sagore Dutta Hospital, Kolkata over a period of six months including 100 established cases of CVA diagnosed on the basis of history, clinical examination and CT scan after fulfilling the inclusion criteria.

Conclusion: Out of these 100 CVA patients, 67 had cerebral infarction and rest had hemorrhagic stroke. Also out of these 100 patients, 41 developed hyponatremia. Out of those 41 hyponatremic patients, 30 had SIADH and rest had CSWS. Sixty seven suffered from Type II Diabetes Mellitus out of these 100 stroke patients. SIADH was seen in 8 diabetic and 8 non diabetic ischemic stroke patients. As well as, SIADH was also seen in 11 diabetic and 3 non diabetic haemorrhagic stroke patients. CSWS was seen in 2 diabetic and 2 non diabetic ischemic stroke patients. CSWS was also seen in 4 diabetic and 3 non diabetic haemorrhagic stroke patients. The mean sodium level was low in diabetic CVA patients (119.45 ± 11.12 mEq/L) than nondiabetics (127.76 ± 09.34 mEq/L) and statistically significant ($p < 0.05$). There was a negative correlation between the fasting blood sugar and sodium. So, diabetic CVA patients were suffered more from hyponatremia than nondiabetic stroke patients.

Keywords: CVA, Hyponatremia, S.I.A.D.H., C.S.W.S..

Corresponding Author: Sajeeb Mondal, Associate Professor, Department of Pathology, Rampurhat Govt. Medical College, Rampurhat, India.

E-mail: sjbmondal@gmail.com

Submission date: August 6, 2024 **Revision date:** October 24, 2024 **Published date: June 7,2025**

Introduction

Cerebrovascular accident is a major cause of hospital admission causing significant mortality and morbidity. The World Health Organization [WHO] definition of Stroke or Cerebrovascular accident (CVA) is-A clinical syndrome consisting of 'rapidly developing clinical signs of focal (at times global) disturbances of cerebral function, lasting more than 24hrs or leading to death with no apparent cause other than that of vascular origin'[1]. In India approx. 1.44-1.64 million new cases are reported every year[2].The prevalence rate of stroke range was 84-262/100,000 in rural and 334-424/100,000 in urbanareas[3].

Basically, CVAs are of –2 types, namely ischemic & haemorrhagic. Hyponatremia is a common electrolyte disorder seen in patients of Cerebrovascular accident (CVA) and it is related with SIADH and CSWS[4]. It is also a major cause of mortality and morbidity in CVA patients

The inappropriate and constant secretion of ADH in spite of hypotonicity causes hyponatremia in SIADH because of water retention. Water permeability of the collecting tubules and ascending limb of loop of Henle is basically regulated by ADH and also allow reabsorbing water or rising in serum osmolality. As negative feedback mechanism is hampered, which ultimately unable to control the ADH secretion and an unsuppressed and excessive ADH release. This hypo-osmolality occurs due to dilution of solutes in plasma and ultimately results in hyponatremia. CSWS can be caused by intracranial trauma and lesions are characterized by the excretion of excessive sodium in urine, resulting in dehydration and ultimately hyponatremia[4].

The association between Type II Diabetes Mellitus and CVA is also significantly high.DM causes various microvascular and macrovascular alternations resulting a number of complications, one of which is stroke.

Some studies were done to relate these factors in different locations, but no similar studies were performed in this region. The aim of the study was to find out whether there is any relationship between the Type II Diabetes Mellitus and hyponatremia in stroke patients and its etiology.

Materials & Methods

This cross-sectional descriptive study with retrospective study design was conducted at College of Medicine & Sagore Dutta Hospital, Kolkata West Bengal, India from February, 2022 to July,2022 over 100 stroke patients (Diagnosed by CT Scan or MRI) admitted in General Medicine ward included as study population based on the inclusion & exclusion criteria. The biochemical analysis of samples was performed in the Department of Biochemistry, College of Medicine & Sagore Dutta Hospital, Kolkata. Informed consents were also taken from the patients or the relatives. As the study design was retrospective in nature, hence ethical clearance was not taken. The patient data was anonymised.

Inclusion criteria:

1. Ischemic and hemorrhagic stroke diagnosed on clinical & radiological basis.
2. Both male and female patients were included according to convenience.
3. Patients having age above 18 years were included.
4. Total 100 patients were included.

Exclusion criteria:

1. Known active infections like AGE etc.
2. Any hematological or other malignancy
3. Head injury or brain tumor
4. Previous stroke/ uncertain stroke patients
5. Patients referred from primary/secondary health centers after initial treatment
6. Patients reporting after 3 days of onset of symptoms
7. Patients with serious co-morbidities such as pulmonary or endocrine disease, hepatic failure, renal failure with dialysis
8. Patients who use medicines causing hyponatremia before the stroke attack
9. Patients with uncertain clinical diagnosis and non-reassuring imaging
10. Transient ischemic attack patients.
11. Patients with serum triglycerides more than 300 mg/dL.

The parameters studied in this study were serum sodium, potassium, fasting blood glucose,

serum albumin and hematocrit. Blood glucose was estimated by Glucose Oxidase-Peroxidase (GOD-POD) method and serum albumin was estimated by Modified BCG method. Serum sodium levels were measured in ion selective electrode (Roche, 9180 Electrolyte analyser).

The Normal serum Sodium level is 136-145 mEq/L and Hyponatremia is the situation when serum sodium level is less than 135 mEq/L. Generally, a normal range for hematocrit is considered to be: For men, 38.3 to 48.6 percent and For women, 35.5 to 44.9 percent. The normal range of serum albumin is 3.4 to 5.4 g/dL

The result was analyzed by suitable statistical software.

Results and Analysis:

Total 100 CVA patients were admitted in Deben Mahata Govt. Medical College and Hospital, Purulia among them 76 were male and 24 were female (Figure:1).

Table 1: Distribution of CVA patients according to various parameters

CVA Patients			
	Total	**Ischemic**	**Haemorrhagic**
Sex	100	65	35
Male	76	40	36
Female	24	15	09
Age(in Year)	54.98 ± 11.18	57.88 ± 12.59	52.08 ± 9.76
With Type 2 DM		70	30

Out of 100 CVA patients, 24 were female and rests were male.(Table 1 & Figure 1).

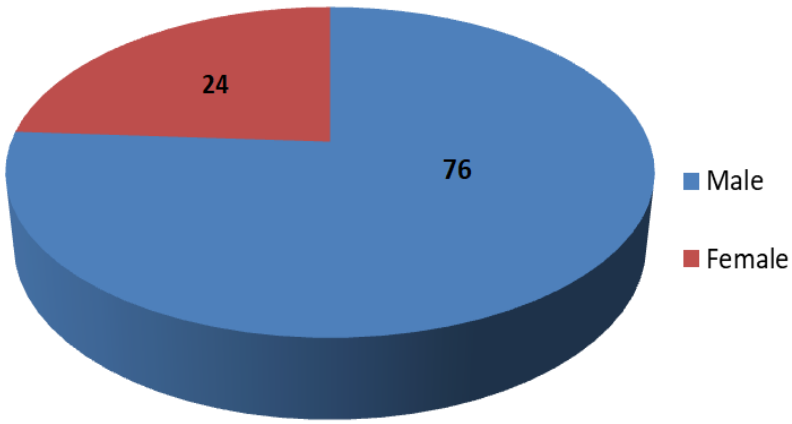

Figure 1: Distribution of CVA patients according to sex

Among 100 CVA patients, 65 were ischemic and rest 35 were hemorrhagic (Figure:2).

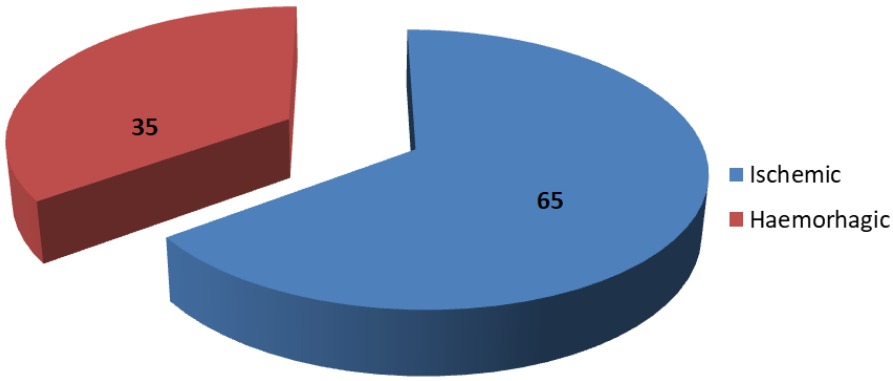

Figure 2: Distribution of CVA patients according to type

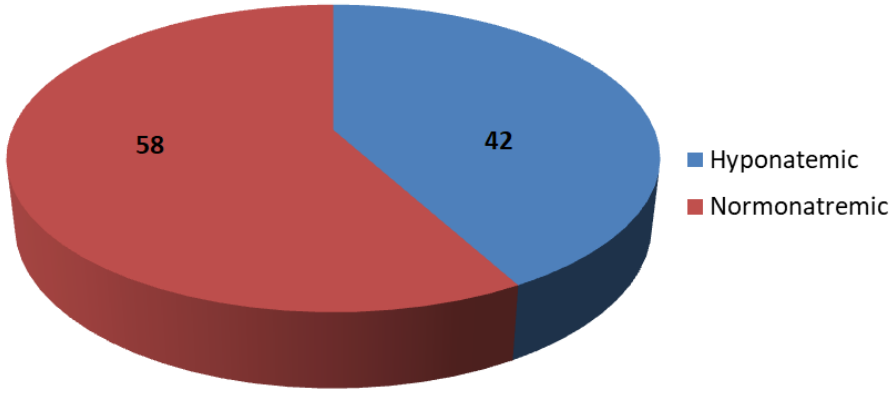

Figure 3: Distribution of CVA patients according to serum sodium level

42 were hyponatemic and rest were normonatremic out of 100 CVA patients (Figure:3).

Figure: 4 showed that 70 were hyperglycemic and rest were normoglycemic out of 100 CVA patients.

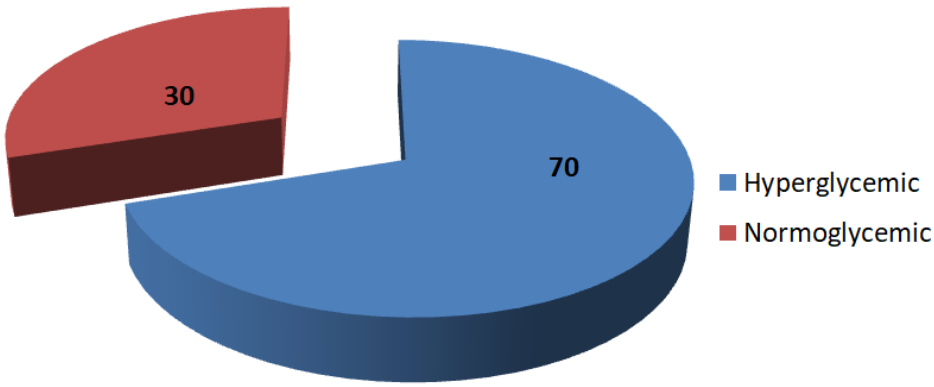

Figure 4: Distribution of CVA patients according to diabetic status

Table 2: Laboratory Characteristics of stroke patients with hyponatremia
Different laboratory parameters were shown in Table 2.

Characteristics	SIADH	CSWS	p-value
Serum Sodium (mEq/L)	126.09 ± 1.68	124.86 ±1.54	0.11
Serum uric acid (mg/dL)	3.62 ± 0.84	5.89 ±0.42	0.04
Serum Osmolality (mOsmol/kg)	247.76 ± 6.33	246.32 ± 7.28	0.13
Urine Osmolality (mOsmol/kg)	1036 ± 41	978 ± 36	0.09
Urine sodium (mEq/L)	69.71 ± 17.23	197.94 ± 23.14	0.001
Urine Specific gravity	1023.46± 10.36	1026.25 ± 9.28	0.13
Urine Volume (ml/kg/hr)	4.54 ± 0.96	2.78 ±0.58	0.03

Among 42 hyponatremic patients, 12 were due to CSWS and rests were due to SIADH.(Table 3)

Table 3: Distribution of hypontaremic CVA patients according to cause of hyponatremia

Type of stroke	SIADH	CSWS	Unknown
Ischemic	15	5	2
Haemorrhagic	10	7	3

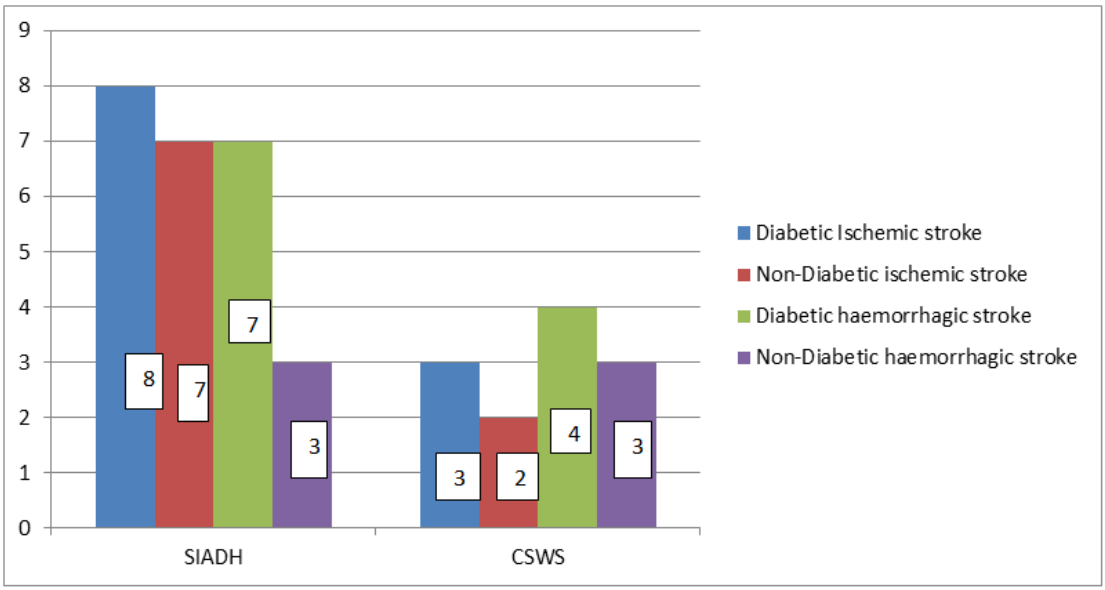

Figure 5: Distribution of hyponatremic CVA patients according to etiology

The number different categories of CVA patients were shown in Figure 5.

Table 4: Serum sodium level in CVA patients according to type of CVA

Parameter	Total	Ischemic	Haemorrhagic
Serum Sodium (in mEq/L)	129.73±4.98	126.01±3.32	125.45±5.04

The serum sodium levels were low in both ischemic and hemorrhagic type of CVA patients. (Table 4)

Table 5: Serum sodium level in CVA patients according to type of glycemic status

Sodium level in diabetic CVA patients	Sodium level in non-diabetic CVA patients	P-value
125.45 ± 11.12 mEq/L	130.76 ± 09.34 mEq/L	< 0.05

The mean sodium level was lesser in diabetic CVA patients than CVA patients without diabetes and the difference was statistically significant. (Table 5)

Table 6: Relationship between fasting blood glucose and serum sodium levels

Parameter	Correlation coefficient (r)	Significance (p value)
Fasting blood glucose Vs serum sodium level	-0.335	0.017

* Correlation is significant at the 0.05 level (2-tailed)

The fasting blood glucose and serum sodium levels carried a significant negative correlation. (Table 6)

Discussions

CVA is one of the common causes of mortality and morbidity in present era. Diabetic individuals are more prone to CVA than nondiabetics. The CVA also may get complicated if hyponatremia occurs. The patient may get drowsier and the prognosis becomes poor. The present was carried out to find out any relationship between the Type II Diabetes Mellitus and hyponatremia in stroke patients and its etiology, whether it is due to CSWS or SIADH.

Total 100 CVA patients were admitted in IPD, Department of General Medicine, College of Medicine & Sagore Dutta Hospital, Kolkata among them 76 were male and 24 were female.

Among 100 CVA patients, 65 were ischemic and rest35 were hemorrhagic. 42 were hyponatemic and rest was normonatremic out of 100 CVA patients. 70 were hyperglycemic and rest was normoglycemic out of 100 CVA patients. Among 42 hyponatremic patients, 12 were due to CSWS and rests were due to SIADH. The serum sodium levels were low in both ischemic and hemorrhagic type of CVA patients. The mean sodium level was lesser in diabetic CVA patients than CVA patients without diabetes and the difference was statistically significant.

In the study conducted by Saleem S et al.,[3] the incidence of hyponatremia was 35 % which is consistent with our study. Hyponatremia can cause cerebral oedema which ultimately results in different neurological dysfunctions. Hyponatremia is quite common complication in stroke may be due to SIADH or CSWS. In the present study, 59.52% and 28.57% cases were SIADH and CSWS respectively out of all the hyponaremic patients. But it is reversed in a study by Saleem S. et all[3] i.e. 7% and 33% had SIADH and CSWS respectively.

A study done by Hoorn EJ et al., the prevalence of hyponatremia in hospital ranges from 11% to 21% and it increases to 28.2% in severely ill patients[6].

Hyponatremia in SIADH is due to increased volume overload and hyponatremia is dilutional in nature. In CSWS, natriuresis is due to disruption of sympathetic neural input signal to kidney and it is also induced by natriuretic peptides like Arterial Natriuretic Peptide (ANP), Brain Natriuretic Factor (BNP), C-Type Natriuretic factor (CNP) and Dendroaspis Natriuretic Peptide (DNP)[7,8,9].

Singh S et al. observed that CSWS is less common than SIADH, which is similar with our study, in which incidence of SIADH was more than CSWS[10].

But in Kalita J et al, hyponatremia due to CSWS was 44.2%, SIADH was 7% and miscellaneous causes were 32.6%[11], which is contrast to our study.

Hyponatremia due to CSWS can be treated with fluid replacement and Fludrocortisone, but hyponatremia due to SIADH can be treated with fluid restriction along with vasopressin-2 receptor antagonists[12,13,14]. The early detection of the diabetes mellitus in the community level is very much important as it can cause a number of complications and proper health education regarding diabetes mellitus and its complications must be initiated among the diabetes patients. The early diagnosis and treatment of the hyponatremia in diabetic CVA patients are very important to prevent morbidity and mortality.

Conclusion

Diabetes mellitus can lead to a number of micro and macrovascular complications which ultimately turn into morbidity and mortality. So, early detection of diabetes is very important as well as detection of complications in diabetic patients can ultimately reduce the morbidities and mortalities.

Conflict of interest: No.

Source of Funding: There was no financial support concerning this work.

Reference

1. Hatano S. Experiance from a multicentre stroke register: a preliminary report. Bulletin of the World Health Organization 1976;54(5):541-53.
2. Pandian JD, Sudhan P. Stroke epidemiology and stroke care services in India. J Stroke. 2013;15(3):128-34
3. Saleem S, Yousuf I, Gul A, Gupta S, Verma S. Hyponatremia in stroke. Ann Indian Acad Neurol. 2014;17:55–57.
4. Oh JY, Shin JI. Syndrome of inappropriate antidiuretic hormone secretion and cerebral/renal salt wasting syndrome: similarities and differences. Front Pediatr. 2014;2:146.
5. Nakajima H, Okada H, Hirose K, Murakami T, Shiotsu Y, Kadono M, et al. Cerebral salt-wasting syndrome and inappropriate antidiuretic hormone syndrome after subarachnoid haemorrhaging. Intern Med. 2017;56:677–80.
6. Hoorn EJ, Zietse R. Hyponatremia and mortality: moving beyond associations. Am J Kidney Dis. 2013;62:139–49.
7. Sorkhi H, Salehi Omran MR, Barari Savadkoohi R, Baghdadi F, Nakhjavani N, Bijani A. CSWS versus SIADH as the probable causes of hyponatremia

in children with acute CNS disorders. Iran J Child Neurol. 2013;7(3):34–39.

8. Leonard J, Garrett RE, Salottolo K, Slone DS, Mains CW, Carrick MM, et al. Cerebral salt wasting after traumatic brain injury: a review of the literature. Scand J Trauma Resusc Emerg Med. 2015;23:98.
9. Palmer BF. Hyponatraemia in a neurosurgical patient: syndrome of inappropriate antidiuretic hormone secretion versus cerebral salt wasting. Nephrol Dial Transplant. 2000;15:262–68.
10. Singh S, Bohn D, Carlotti AP, Cusimano M, Rutka JT, Halperin ML. Cerebral salt wasting: truths, fallacies, theories, and challenges. Crit Care Med. 2002;30:2575–79.
11. Kalita J, Singh RK, Misra UK. Cerebral Salt Wasting Is the Most Common Cause of Hyponatremia in Stroke. J Stroke Cerebrovasc Dis. 2017; 26:1026-32.
12. Laville M, Burst V, Peri A, Verbalis JG. Hyponatremia secondary to the syndrome of inappropriate secretion of antidiuretic hormone (SIADH): therapeutic decisionmaking in real-life cases. Clin Kidney J. 2013;6(Suppl 1):11-120
13. Zietse R, Van der Lubbe N, Hoorn EJ. Current and future treatment options in SIADH. NDT Plus 2009; 2(Suppl. 3): 312-319.
14. Gross P. Clinical management of SIADH. Ther Adv Endocrinol Metab. 2012;3(2):61-73.