Original Article

ISSN (0): 2347-3398; ISSN (P): 2277-7253

Assessment of Electrolyte Abnormality in Acute Stroke Patients

Shaik Rabbani[1], N Sathish Reddy[2]

[1]Assistant Professor, Department of General Medicine, Fathima Institute of Medical Sciences, Kadapa, Andhra Pradesh, India.

[2]Associate Professor, Department of General Medicine, Fathima Institute of Medical Sciences, Kadapa, Andhra Pradesh, India.

Abstract

Background: To determine electrolyte abnormality in acute stroke patients. **Subjects and Methods:** 102 patients of stroke of both genders were enrolled. All were subjected to assessment of electrolytes level. **Results:** Age group 30-40 years comprised of 12 patients, 40-50 years had 28, 50-60 years had 25 and >60 years had 47 patients. A significant difference was observed (P< 0.05). Type of stroke was hemorrhagic in 48 and ischaemic in 54 patients. A non- significant difference was observed (P> 0.05). Dyselectrolytaemia in haemorrhagic stroke patients was seen in 40% and in ischaemic stroke patients in 65%. The mean serum osmolality in haemorrhagic stroke patients was 292.1 mmol/kg and in ischaemic stroke was 306.4 mmol/kg, urine sodium was 62.4 mEq/L/24 hours in haemorrhagic stroke patients and 67.4 mEq/L/24 hours in ischaemic stroke patients and urinary potassium was 78.6 mEq/L/24 hours in haemorrhagic stroke patients and 74.8 mEq/L/24 in ischaemic stroke patients. The difference was non- significant (P> 0.05). Sodium level found to be normal in 24 patients, hyponatraemia in 56 and hypernatremia in 22 patients. The difference was significant (P< 0.05). **Conclusion:** Sodium level was altered with hyponatremia was seen in most of the patients. The level of electrolytes should be assessed in stroke patients. Maximum cases of ischemic stroke in age group >60 years was seen.

Keywords: Stroke, Dyselectrolytaemia, hyponatraemia.

Corresponding Author: Shaik Rabbani, Assistant Professor, Department of General Medicine, Fathima Institute of Medical Sciences, Kadapa, Andhra Pradesh, India.

Received: 31 July 2021 **Revised: 11 September 2021** **Accepted: 25 September 2021** **Published: 30 November 2021**

Introduction

Stroke or cerebrovascular accident or CVA is defined as rapidly developing clinical symptoms and/or signs of focal and at times global loss of brain function, with symptoms lasting more than 24 hours or leading to death, with no apparent cause other than that of vascular origin.[1] About 85% of all first ever stroke are ischaemic, 10% are due to primary intracerebral haemorrhage and 5% are due to subarachnoid haemorrhage.[2] The incidence of stroke has declined in the western population during the last three decades. On the contrary, the burden of the disease in South Asian countries, such as India, Pakistan, Bangladesh, and Sri Lanka, is likely to rise.[3]

Stroke is the second leading cause of death. Many important factors related to ischemic stroke are modifiable. Quit smoking, increased physical activity, and proper diet, so, controlled diabetes and hypertension as well- known risk factors for stroke can be effective in reducing the rate of stroke.[4] Hyponatraemia, hypernatremia resulting from inappropriate secretion of antidiuretic hormone (ADH), increase in Brain Natriuretic-peptide (BNP), Atrial Natriuretic peptide and inappropriate fluid intake and loss; can lead to complications like seizures and death.[5] Hyponatremia is frequently observed in patients with stroke either on admission or during their hospitalization. Briefly, “baseline” hyponatremia identified upon admission is mainly attributed to the presence of comorbidities such as diabetes mellitus, chronic renal failure, and heart failure.[6] Besides, stroke patients, the elderly in particular, are frequently treated with medications that can induce hyponatremia, including antihypertensive agents, antidepressants, and nonsteroidal anti-inflammatory drugs.[7] Considering this, we attempted present study to assess electrolyte abnormality in acute stroke patients.

Subjects and Methods

We enrolled one hundred two adult patients of stroke of either gender admitted to emergency department. The study protocol was approved from ethical review committee. The written consent from first degree relatives of all enrolled patients was obtained.

Data such as name, age, gender etc. was CNS examination was performed in all patients. All were subjected to baseline laboratory parameters such as complete blood count (CBC), blood sugar level (RBS), liver function test (LFT), renal function test (RFT), lipid profile, serum sodium, potassium, chlorides, urine sodium and potassium and serum osmolality. CT scan Brain and MRI Brain was performed. Results were statistically analysed. P value less than 0.05 was considered significant.

Results

Age group 30-40 years comprised of 12 patients, 40-50 years had 28, 50-60 years had 25 and >60 years had 47 patients. A significant difference was observed (P< 0.05) [Table 1]

Table 1: Distribution of patients based on age group

Age group (years)	Number	P value
30-40	12	0.05
40-50	18	
50-60	25	
>60	47	

Table 2: Type of stroke

Stroke type	Number	P value
Hemorrhagic	48	0.91
Ischaemic	54	

Type of stroke was hemorrhagic in 48 and ischaemic in 54 patients. A non- significant difference was observed (P> 0.05) [Table 2].

Table 3: Assessment of Dyselectrolytaemia in both patients

Dyselectrolytaemia	Hemorrhagic	Ischaemic	P value
Present	40%	65%	0.05
Absent	60%	35%	

Dyselectrolytaemia in haemorrhagic stroke patients was seen in 40% and in ischaemic stroke patients in 65%. The difference was significant (P< 0.05) [Table 3].

Table 4: Assessment of laboratory parameters

Parameters	Hemorrhagic	Ischaemic	P value
Serum osmolality (mmol/kg)	292.1	306.4	0.91
Urine sodium (mEq/L/24 hours)	62.4	67.4	0.86
Urinary potassium (mEq/L/24 hours)	78.6	74.8	0.94

The mean serum osmolality in haemorrhagic stroke patients was 292.1 mmol/kg and in ischaemic stroke was 306.4 mmol/kg, urine sodium was 62.4 mEq/L/24 hours in haemorrhagic stroke patients and 67.4 mEq/L/24 hours in ischaemic stroke patients and urinary potassium was 78.6 mEq/L/24 hours in haemorrhagic stroke patients and 74.8 mEq/L/24 in ischaemic stroke patients. The difference was non- significant (P> 0.05) [Table 4].

Table 5: Occurrence of hyponatraemia

Sodium level	Number	P value
Normal	24	0.05
Hyponatremia	56	
Hypernatremia	22	

Sodium level found to be normal in 24 patients, hyponatraemia in 56 and hypernatremia in 22 patients. The difference was significant (P< 0.05) [Table 5]

Discussion

Electrolyte disturbance are commonly found in acute stroke setting. Hypernatremia, hyponatremia and hypokalaemia was the commonest type of disturbance.[8] Electrolyte disturbances are commonly found among other metabolic problems in patients with acute ischemic stroke.[9] It is a potential cause of patient death unless corrected promptly. The disorders of sodium (Na) and potassium (K) balance are identified as the most common electrolyte abnormalities in patients with acute stroke.[10] Patients with hemorrhagic stroke present with symptoms like headache and vomiting, which in turn is a potential cause of electrolyte imbalance.[11] This disturbance in the electrolyte balance is due to deranged secretion of antidiuretic hormones (ADHs), rise in the levels of atrial and brain natriuretic peptides, and inappropriate fluid input and output, causing complications like seizures and death.[12] We attempted present study to assess electrolyte abnormality in acute stroke patients.

Our results showed that Age group 30-40 years comprised of 12 patients, 40-50 years had 28, 50-60 years had 25 and >60 years had 47 patients. Fawal et al,[13] evaluated the effect of stress induced by acute stroke on the blood glucose and serum electrolytes and its relationship to the type, severity, and prognosis of stroke in 100 patients. Stress hyperglycemia was noted in 21 patients, out of which 62% presented with hemorrhagic stroke. Undiagnosed diabetes was found in 47% of cases. Also, 13 % of our patients had both stress hyperglycemia and electrolyte disturbance simultaneously. Mortality among hemorrhagic versus ischemic patients was 46% and 22%, respectively. The independent predictors of mortality were stress hyperglycemia, electrolytes disturbance, ischemic heart diseases, and type and admission severity of stroke.

Our results showed type of stroke was hemorrhagic in 48 and ischaemic in 54 patients. Dyselectrolytaemia in haemorrhagic stroke patients was seen in 40% and in ischaemic stroke patients in 65%. Alam et al,[14] in their study in 110 cases of stroke (55 ischemic and 55 haemorrhagic strokes) the serum concentration of Na^+, K^+, and Cl^- were measured. Out of 55 patients 29 (52.72%) had abnormal sodium level, of them 23 (41.8%) had hyponatremia, 6 (10.9%) had hypernatremia. 23 (41.80%) out of 55 had abnormal sodium level, of them 21(38%) had hyponatremia. The result showed that hyponatremia is almost equally common in both haemorrhagic and ischaemic group without significant difference (p>0.05). The study also revealed that hyponatremia is more common than hypernatremia in both groups. Mean ± SD of age of the haemorrhagic group was 60.80 ± 15.97 while the age of ischaemic group was 59.89 ± 15.84 years. Male, female ratio in haemorrhagic and ischaemic group 1:0.62 and 1:0.89 respectively. Mean ± SD of serum Na^+, K^+, Cl^- in haemorrhagic group were 136.18 ± 10.5, 3.83 ± 0.65, 97.96 ± 16.74 mmol/L, in ischaemic group 135.08 ± 9.08, 4.00 ± 0.75, 100.27 ± 8.39 mmol/L.

Our results showed the mean serum osmolality in haemorrhagic stroke patients was 292.1 mmol/kg and in ischaemic stroke was 306.4 mmol/kg, urine sodium was 62.4

mEq/L/24 hours in haemorrhagic stroke patients and 67.4 mEq/L/24 hours in ischaemic stroke patients and urinary potassium was 78.6 mEq/L/24 hours in haemorrhagic stroke patients and 74.8 mEq/L/24 in ischaemic stroke patients. In a study of 100 stroke patients, CSW was the most common cause of hyponatremia (44.2%), whereas only 7% of hyponatremia cases were attributed to SIAD.[15] On the contrary, in another series of 100 patients with mild/moderate subarachnoid hemorrhage, SIAD (71.4%) and acute glucocorticoid insufficiency (8.2%) were found to be the leading causes of hyponatremia.[16]

Sodium level found to be normal in 24 patients, hyponatraemia in 56 and hypernatremia in 22 patients. Mitchell,[17] showed an association between increased BMI and stroke. High daily dietary intake of fat is associated with obesity and may act as an independent risk factor or may affect other stroke risk factors such as hypertension, diabetes, and hyperlipidaemia.

Conclusion

Sodium level was altered with hyponatremia was seen in most of the patients. The level of electrolytes should be assessed in stroke patients. Maximum cases of ischemic stroke in age group >60 years was seen.

References

1. Coenraad MJ, Meinders AE, Taal JC, Bolk JH. Hyponatremia in intracranial disorders. Neth J Med. 2001;58(3):123-7. doi: 10.1016/s0300-2977(01)00087-0.
2. Moussa NA, Osman AR, Yahya TM. Acute hyponatremic encephalopathy after a cerebrovascular accident. Am J Med Sci. 1998;316(1):56-9. doi: 10.1097/00000441-199807000-00009.
3. Xie JX, Sasaki S, Joossens JV, Kesteloot H. The relationship between urinary cations obtained from the INTERSALT study and cerebrovascular mortality. J Hum Hypertens. 1992;6(1):17-21.
4. Hinkle JL, Guanci MM. Acute ischemic stroke review. J Neurosci Nurs. 2007;39(5):285-93, 310. doi: 10.1097/01376517-200710000-00005.
5. Turana Y, Tengkawan J, Chia YC, Nathaniel M, Wang JG, Sukonthasarn A, et al. Hypertension and stroke in Asia: A comprehensive review from HOPE Asia. J Clin Hypertens (Greenwich). 2021;23(3):513-521. doi: 10.1111/jch.14099.
6. Hasan MK, Hasan AB, Rubaiyat KA. Electrolyte Disturbances in Acute Phase of Stroke Patients. Dinajpur Med Col J. 2013;6(1):12-16.
7. Boutayeb A, Derouich M, Boutayeb W, Lamlili MEN. Cerebrovascular Diseases and Associated Risk Factors in WHO Eastern Mediterranean Countries, Cardiology and Angiology. An Inter J. 2014;2(1):62-75.
8. Mansoor F, Kumar J, Kaur N, Sultan S, Tahir H, Dilip A, et al. Frequency of Electrolyte Imbalance in Patients Presenting With Acute Stroke. Cureus. 2021;13(9):e18307. doi: 10.7759/cureus.18307.
9. Bhatti A B, Ali F, Satti S A. Association of Obesity with Stroke. Int J Biomed Res. 2013;4(8):422-426.
10. Rosamond WD, Folsom AR, Chambless LE, Wang CH, McGovern PG, Howard G, ET AL. Stroke incidence and survival among middle-aged adults: 9-year follow-up of the Atherosclerosis Risk in Communities (ARIC) cohort. Stroke. 1999;30(4):736-43. doi: 10.1161/01.str.30.4.736.
11. Gray JR, Morbitzer KA, Liu-DeRyke X, Parker D, Zimmerman LH, Rhoney DH. Hyponatremia in Patients with Spontaneous Intracerebral Hemorrhage. J Clin Med. 2014;3(4):1322-32. doi: 10.3390/jcm3041322.
12. Chen Z, Jia Q, Liu C. Association of Hyponatremia and Risk of Short- and Long-Term Mortality in Patients with Stroke: A Systematic Review and Meta-Analysis. J Stroke Cerebrovasc Dis. 2019;28(6):1674-1683. doi: 10.1016/j.jstrokecerebrovasdis.2019.02.021.
13. El-Fawal BM, Badry R, Abbas WA, Ibrahim AK. Stress hyperglycemia and electrolytes disturbance in patients with acute cerebrovascular stroke. Egypt J Neurol Psychiatry Neurosurg. 2019;55(1):1-6.
14. Alam MN, Uddin MJ, Rahman KM, Ahmed S, Akhter M, Nahar N, ET AL. Electrolyte changes in stroke. Mymensingh Med J. 2012;21(4):594-9.
15. Kalita J, Singh RK, Misra UK. Cerebral Salt Wasting Is the Most Common Cause of Hyponatremia in Stroke. J Stroke Cerebrovasc Dis. 2017;26(5):1026-1032. doi: 10.1016/j.jstrokecerebrovasdis.2016.12.011.
16. Hannon MJ, Behan LA, O'Brien MM, Tormey W, Ball SG, Javadpour M, ET AL. Hyponatremia following mild/moderate subarachnoid hemorrhage is due to SIAD and glucocorticoid deficiency and not cerebral salt wasting. J Clin Endocrinol Metab. 2014;99(1):291-8. doi: 10.1210/jc.2013-3032.
17. Mitchell AB, Cole JW, McArdle PF, Cheng YC, Ryan KA, Sparks MJ, ET AL. Obesity increases risk of ischemic stroke in young adults. Stroke. 2015;46(6):1690-2. doi: 10.1161/STROKEAHA.115.008940.

How to cite this article: Rabbani S, Reddy NS. Assessment of Electrolyte Abnormality in Acute Stroke Patients. Asian J. Med. Res. 2021;10(4):22-24.

DOI: dx.doi.org/10.47009/ajmr.2021.10.4.ME5

Source of Support: Nil, **Conflict of Interest:** None declared.