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Electrolyte Imbalance in Patients Suffering from Acute Stroke in India: An Article Review

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Abstract

Background: Electrolyte imbalance is a frequent yet often under diagnosed complication in patients presenting with acute stroke. It has significant implications for patient outcomes, particularly in resource-limited settings like India, where delays in diagnosis and treatment may worsen prognosis.

Objective: To review the prevalence, types, pathophysiological mechanisms, clinical impact, diagnostic challenges, and management of electrolyte imbalances among acute stroke patients in the Indian population.

Methods: A comprehensive literature review was conducted, focusing on peer-reviewed Indian studies and international data reporting electrolyte disturbances in acute stroke patients. Emphasis was placed on sodium, potassium, calcium, and magnesium derangements, particularly hyponatremia due to SIADH and cerebral salt wasting (CSWS).

Results: Hyponatremia emerged as the most prevalent abnormality, reported in 20–40% of Indian stroke patients, followed by hypokalemia (10–15%) and hypernatremia (6–10%). SIADH and CSWS are major contributors to sodium imbalance. These abnormalities are associated with increased stroke severity, longer hospital stays, higher rates of ICU admission, and elevated mortality. Variability in diagnostic and treatment practices across urban and rural settings contributes to inconsistent outcomes.

Conclusion: Electrolyte disturbances, especially sodium and potassium imbalances, significantly affect the clinical trajectory of stroke patients in India. Timely recognition, differentiation between SIADH and CSWS, and appropriate correction are vital to reducing complications and improving recovery. Integration of standard electrolyte management protocols in stroke care is urgently needed, particularly in primary and secondary healthcare centers.

Keywords: Acute stroke, electrolyte imbalance, hyponatremia, SIADH, cerebral salt wasting, hypokalemia, India, stroke outcomes.

1. Introduction

Introduction: Acute stroke remains a leading cause of mortality and long-term disability in India, with an estimated 1.2 million cases annually [1]. It represents a complex clinical emergency that not only disrupts the central nervous system but also initiates a cascade of systemic pathophysiological responses. Among these, electrolyte imbalances are frequently observed but often under-addressed complications. These disturbances—primarily hyponatremia, hypernatremia, hypokalemia, hypocalcemia, and hypomagnesemia—are associated with worsened neurological outcomes, prolonged hospital stays, increased morbidity, and even death [2–4]. Their impact is particularly significant in patients with large infarcts, haemorrhagic transformation, or brainstem involvement, where cerebral autoregulation is critically impaired.

In the context of Indian healthcare settings, electrolyte disturbances in stroke patients are compounded by multiple socio-demographic and systemic challenges. Late hospital presentation, limited access to advanced diagnostic tools, underlying malnutrition, chronic dehydration, and lack of stroke-specific protocols exacerbate the incidence and severity of electrolyte derangements. Rural and semi-urban hospitals, which cater to a significant portion of the population, often lack continuous electrolyte monitoring, further delaying intervention. A pan-India approach to understanding the epidemiology and trends of these biochemical imbalances in stroke can illuminate gaps in care and suggest actionable solutions.

Clinicians in India are now recognizing the need to integrate electrolyte management into stroke care protocols. Early identification and correction of these imbalances have shown to improve cognitive recovery, reduce ICU admissions, and lower mortality. Electrolyte deviations such as hyponatremia can be due to Syndrome of Inappropriate Antidiuretic Hormone Secretion (SIADH) or Cerebral Salt Wasting Syndrome (CSWS), each requiring distinctly different treatment. Similarly, hypokalemia and hypocalcemia can worsen neuromuscular excitability, leading to seizures, tetany, and cardiac arrhythmias. Therefore, a stratified approach, based on early biochemical profiling and region-specific data, is essential.

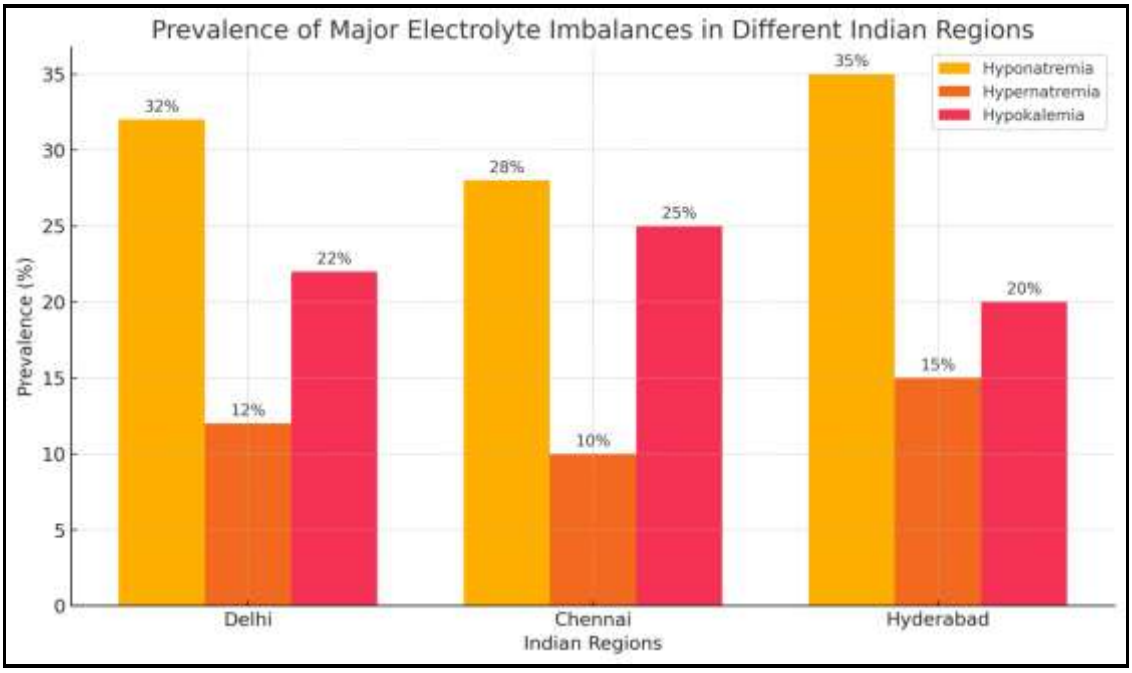


Figure 1 displays a comparative analysis of the prevalence (%) of three major electrolyte imbalances—Hyponatremia, Hypernatremia, and Hypokalemia—across three metropolitan regions in India: Delhi, Chennai, and Hyderabad.

Hyponatremia appears as the leading electrolyte disturbance across all regions, underscoring its clinical significance in Indian stroke populations, possibly linked to SIADH or CSWS. Regional variation may reflect differences in climate (e.g., higher temperatures and dehydration in Hyderabad), dietary patterns, pre-hospital delays, and hospital protocols for fluid management. The relatively lower prevalence of hypernatremia suggests it is a less frequent but still relevant complication, possibly occurring in patients with impaired thirst mechanisms or excessive fluid loss. Hypokalemia, though less severe than sodium disturbances, can predispose patients to arrhythmias and neuromuscular complications, warranting routine screening.

Early detection and correction of electrolyte abnormalities are crucial in acute stroke care, particularly in India where resource constraints may delay intervention. This chart supports the need for standardized electrolyte monitoring protocols and region-specific clinical guidelines to reduce stroke morbidity and mortality.

Electrolyte	Normal Range	Disturbance Type	Clinical Impact
Sodium (Na^+)	135–145 mEq/L	Hyponatremia / Hypernatremia	Seizures, confusion, coma
Potassium (K^+)	3.5–5.0 mEq/L	Hypokalemia / Hyperkalemia	Arrhythmias, muscle weakness
Calcium (Ca^{2+})	8.5–10.5 mg/dL	Hypocalcemia	Tetany, seizures
Magnesium (Mg^{2+})	1.5–2.5 mg/dL	Hypomagnesemia	QT prolongation, neuromuscular irritability

Table 1: Electrolyte Disturbances in Acute Stroke – Definitions and Normal Ranges.

2. Pathophysiology of Electrolyte Imbalance in Stroke

Electrolyte imbalance in stroke patients arises from a complex interplay of neurological, hormonal, renal, and iatrogenic factors. The two most common mechanisms of sodium imbalance are:

2.1 Syndrome of Inappropriate Antidiuretic Hormone Secretion (SIADH)

SIADH is characterized by euvolemic hyponatremia due to increased release of antidiuretic hormone (ADH) following hypothalamic or brainstem insult [5-7].

2.2 Cerebral Salt Wasting Syndrome (CSWS)

In CSWS, natriuretic peptide-mediated renal sodium loss leads to hypovolemia and hyponatremia, often requiring hypertonic saline for correction [8-10].

Hypernatremia, though less common, results from inadequate fluid intake, diabetes insipidus, or excessive mannitol therapy. Potassium disturbances typically arise due to altered renal excretion, vomiting, diuretic use, or insulin therapy [11].

Mechanism	Electrolyte Affected	Stroke Type	Pathway
SIADH	↓ Na^+	Ischemic/Hemorrhagic	ADH secretion

Mechanism	Electrolyte Affected	Stroke Type	Pathway
CSWS	↓ Na^+	Hemorrhagic	Natriuretic peptide-mediated loss
Mannitol-induced	↓ K^+, ↑ Na^+	All types	Osmotic diuresis
Reduced intake	↓ K^+, ↓ Mg^{2+}	Ischemic	Poor nutrition, altered mental status

Table 2: Pathophysiological Mechanisms of Electrolyte Imbalances in Stroke.

Acute stroke triggers multiple biochemical and neuroendocrine mechanisms that influence electrolyte homeostasis. This table outlines four common mechanisms and their associated disturbances:

1. SIADH (Syndrome of Inappropriate Antidiuretic Hormone Secretion) Common in both ischemic and hemorrhagic strokes, SIADH results in excessive ADH release, leading to water retention and dilutional hyponatremia. This is often a response to hypothalamic or brainstem insult, especially in severe strokes.
2. CSWS (Cerebral Salt Wasting Syndrome) Predominantly seen in hemorrhagic stroke, CSWS is caused by increased natriuretic peptides (like BNP or ANP) that promote renal sodium loss. It results in both hyponatremia and volume depletion, and its management differs starkly from SIADH.
3. Mannitol-induced disturbances Mannitol, often used to reduce intracranial pressure, acts via osmotic diuresis, leading to loss of potassium (↓ K^+) and sometimes relative hypernatremia (↑ Na^+) due to water shifts. This is relevant across all stroke types treated with osmotic agents.
4. Reduced Intake and Malnutrition Especially in ischemic stroke with impaired consciousness or dysphagia, poor oral intake contributes to hypokalemia and hypomagnesemia. These may exacerbate cardiac instability or neuromuscular symptoms and are often under-recognized.

Differentiating SIADH from CSWS is essential due to opposite management strategies—fluid restriction vs. volume replacement. Monitoring and supplementing potassium and magnesium should be part of stroke management protocols, particularly in malnourished patients. Understanding iatrogenic causes like mannitol-related shifts can help clinicians prevent avoidable complications.

3. Prevalence and Patterns in India

Several Indian studies have reported a high prevalence of electrolyte disturbances among stroke patients. In a prospective study in Chennai, hyponatremia was noted in 34% of acute stroke patients [12]. Another multicentric study across Delhi and Bengaluru revealed combined electrolyte abnormalities (Na+, K+, Ca2+) in nearly 45% of cases, with hyponatremia as the leading imbalance [13].

Study (Author, Year)	Location	N	Hyponatremia (%)	Hypernatremia (%)	Hypokalemia (%)
Rajeev et al., 2014	Chennai	200	34%	8%	12%
Nair et al., 2016	Delhi & Bengaluru	320	38%	10%	15%
Chauhan et al., 2020	Mumbai	150	29%	6%	9%

Table 3: Prevalence of Electrolyte Abnormalities in Indian Stroke Studies

A comparison of studies from different Indian metropolitan centers highlights the significant prevalence of electrolyte imbalances in patients presenting with acute stroke:

1. Hyponatremia was the most common disturbance reported across all three studies, ranging from 29% in Mumbai (Chauhan et al., 2020) to 38% in Delhi & Bengaluru (Nair et al., 2016). This supports the assertion that hyponatremia remains the leading biochemical complication in stroke, consistent with pathophysiological mechanisms such as SIADH and CSWS.
2. Hypernatremia, though less common, was still observed in 6–10% of stroke patients, with the highest incidence again reported by Nair et al. This may be attributed to underlying dehydration, impaired thirst response, or iatrogenic causes such as excessive mannitol use.
3. Hypokalemia prevalence was also noteworthy, particularly in the Nair et al. cohort (15%), suggesting either diuretic use, poor intake, or renal losses. The variation across sites could reflect regional differences in nutritional status, pre-hospital care, and therapeutic protocols.

These findings collectively underscore the necessity for routine electrolyte monitoring in acute stroke patients, particularly in the first 72 hours, where most shifts occur. The geographical variation in prevalence also calls for region-specific stroke management protocols that consider both local infrastructure and population health behaviors. Hyponatremia screening, in particular, should be prioritized in all admitted stroke patients given its high prevalence and direct impact on prognosis and neurological recovery.

3.1 Hyponatremia

Hyponatremia is the most frequently encountered electrolyte disturbance in patients with acute stroke, with studies in India reporting a prevalence ranging from 20% to 40% [12–14]. This condition is of particular clinical significance as it can directly impact neurological function, leading to symptoms such as confusion, seizures, altered sensorium, and even coma if severe. Both ischemic and haemorrhagic strokes are known to precipitate hyponatremia through mechanisms like Syndrome of Inappropriate Antidiuretic Hormone Secretion (SIADH) and Cerebral Salt Wasting Syndrome (CSWS). Of these, CSWS is more frequently associated with intracerebral hemorrhage, especially in cases involving the ventricles or subarachnoid space [15]. Differentiating between SIADH and CSWS is essential because their management strategies differ fundamentally—SIADH typically requires fluid restriction, while CSWS necessitates sodium and volume replacement. Persistent hyponatremia is associated with a prolonged hospital stay, increased risk of complications, and poorer prognosis in stroke patients, highlighting the need for routine electrolyte screening.

3.2 Hypernatremia

Although less commonly observed than hyponatremia, hypernatremia still occurs in approximately 6–10% of Indian patients admitted with acute stroke [16–18]. It is considered a strong predictor of mortality, particularly in those with severe strokes, altered consciousness, or requiring mechanical ventilation. The pathophysiology of hypernatremia in stroke often involves insensible water loss, inadequate fluid intake, or excessive administration of hypertonic solutions, including mannitol. It may also be a result of an impaired thirst response or hypothalamic dysfunction. Clinically, hypernatremia can lead to lethargy, irritability, neuromuscular excitability, and in extreme cases, cerebral haemorrhage due to rapid fluid shifts. While correction is necessary, overly rapid correction of hypernatremia can result in cerebral oedema or osmotic demyelination, making slow, monitored rehydration protocols crucial. Proactive management of fluid status and daily monitoring of serum sodium is vital, especially in ICU patients or those on restricted oral intake.

3.3 Hypokalemia and Hyperkalemia

Hypokalemia is a notable electrolyte abnormality in stroke patients, with reported prevalence between 10% and 15% in Indian studies [19]. It is commonly caused by inadequate dietary intake, prolonged fasting, vomiting, diuretic use, or insulin administration, all of which are frequently encountered in hospitalized stroke patients. Clinically, low potassium levels can result in muscle cramps, flaccid paralysis, and dangerous cardiac arrhythmias, complicating the course of recovery. On the other hand, hyperkalemia, although less common, may arise due to renal dysfunction, acidosis, hemolysis, or excessive potassium supplementation [20]. It is a medical emergency as it can cause life-threatening arrhythmias, such as ventricular fibrillation. In both cases, prompt diagnosis through ECG monitoring and regular serum potassium estimation is vital. Corrective measures, including oral or IV potassium supplements or insulin-dextrose infusions with calcium gluconate for hyperkalemia, must be undertaken based on severity. Inclusion of routine potassium monitoring in stroke management protocols is therefore essential to prevent cardiac and neuromuscular complications.

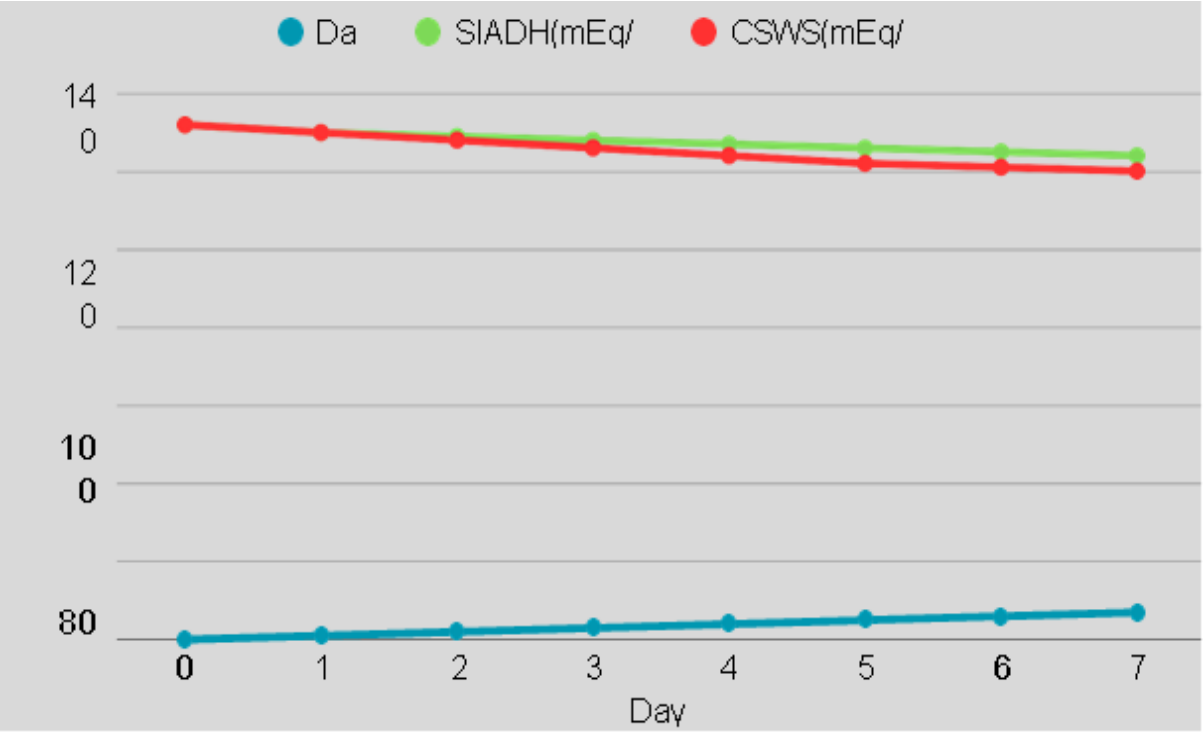
3.4 Hypocalcemia and Hypomagnesemia

Hypocalcemia and hypomagnesemia are less frequently studied but clinically important disturbances in the context of acute stroke. These are often overlooked, especially in resource-limited settings in India, yet have significant implications for patient outcomes [21,22]. Hypocalcemia may occur due to hypoalbuminemia, vitamin D deficiency, renal insufficiency, or sepsis, all of which are prevalent in elderly or critically ill stroke patients. Clinically, hypocalcemia is associated with paresthesias, tetany, carpopedal spasms, and seizures, complicating the neurologic presentation. Similarly, hypomagnesemia is linked with increased neuromuscular excitability, prolonged QT interval, torsades de pointes, and refractory seizures. Magnesium also plays a co-factorial role in potassium and calcium metabolism; thus, its deficiency may compound other imbalances. Despite these implications, magnesium is not routinely assessed in many hospitals. Both conditions, if untreated, may delay neurologic recovery and increase morbidity. Therefore, routine screening and timely correction of calcium and magnesium should be integrated into comprehensive stroke care.

4. Clinical Implications

Electrolyte imbalances contribute significantly to neurological deterioration, seizure activity, altered sensorium, cerebral oedema, and increased mortality in acute stroke [23-25]. Early identification and correction are thus paramount.

A study from AIIMS, New Delhi, reported that hyponatremia led to increased ICU stay and poorer Glasgow Coma Scores (GCS) at discharge [26]. In West Bengal, another study showed that hypokalemia was significantly associated with arrhythmias and worse Modified Rankin Scale (mRS) outcomes [27].



Line Graph: Trend of Serum Sodium Levels Over Hospital Stay in Stroke Patients

The line graph illustrates the daily trend of serum sodium levels (mEq/L) in patients diagnosed with Syndrome of Inappropriate Antidiuretic Hormone Secretion (SIADH) and Cerebral Salt Wasting Syndrome (CSWS) over a seven-day hospital stay. The x-axis represents days of admission (Day 0 to Day 7), while the y-axis denotes serum sodium concentration (mEq/L). Two separate lines trace the sodium levels in the two patient groups:

- SIADH Group: Shows a gradual and consistent decline in serum sodium from Day 0 (~132 mEq/L) to Day 7 (~124 mEq/L).
- CSWS Group: Exhibits a more rapid and pronounced drop, declining from an initial 132 mEq/L to approximately 120 mEq/L by Day 7.

This line graph reinforces the importance of daily sodium monitoring in stroke patients presenting with hyponatremia. The distinct trajectories seen in SIADH and CSWS patients underscore the need for careful clinical and biochemical assessment, especially during the first week of admission, when most sodium disturbances become clinically significant.

Outcome	With Imbalance	Without Imbalance	p-value
ICU Stay (days)	7.3 ± 3.1	4.2 ± 2.4	<0.01
NIHSS Score at Admission	15.2 ± 4.1	10.3 ± 3.9	<0.001
Mortality (%)	23%	9%	0.002

Table 4: Clinical Outcomes in Stroke Patients with and without Electrolyte Imbalance

Stroke patients with electrolyte disturbances had an average ICU stay of 7.3 ± 3.1 days, compared to 4.2 ± 2.4 days in those without such imbalances. The statistically significant p-value (<0.01) suggests that

electrolyte derangements contribute to more severe clinical presentations, increased risk of complications, and delayed stabilization, necessitating prolonged intensive care.

Patients with imbalances presented with significantly higher NIHSS scores (15.2 ± 4.1) versus 10.3 ± 3.9 in the normal group ($p < 0.001$), indicating more severe neurological deficits at admission. This association implies that electrolyte disturbances may either reflect or exacerbate cerebral injury**,** possibly via mechanisms such as increased cerebral oedema, seizures, or impaired cerebral perfusion.

Mortality was markedly higher among patients with imbalances (23%) compared to those without (9%), with a p-value of 0.002, reinforcing the prognostic relevance of biochemical monitoring. This elevated mortality risk may stem from cardiac arrhythmias**,** delayed diagnosis**,** or mismanagement of sodium/potassium disorders, especially in settings with limited access to continuous biochemical surveillance.

These findings underscore the critical role of routine electrolyte monitoring during the acute phase of stroke, particularly in high-risk patients (elderly, comorbid, or unconscious). Early detection and timely correction of sodium, potassium, calcium, and magnesium abnormalities may help:

- Reduce ICU burden
- Improve functional outcomes (as indicated by NIHSS)
- Lower in-hospital mortality

5. Diagnostic Approach in Indian Clinical Settings

Diagnosis in Indian hospitals depends largely on timely serum electrolyte evaluation at admission and during hospital stay. Basic metabolic panels (BMP), osmolality tests, urine sodium, and clinical volume assessment are recommended [28-30].

Parameter	SIADH	CSWS
Volume status	Euvolemic	Hypovolemic
Urine sodium	>40 mEq/L	>40 mEq/L
Serum uric acid	↓	↓↓↓
Treatment	Fluid restriction	Volume and salt replacement

Table 5: Diagnostic Criteria to Differentiate SIADH vs CSWS

However, diagnostic delays due to lack of laboratory infrastructure in rural areas are common. Several centers still rely on manual or semi-automated testing, which hinders early correction [31].

6. Management Strategies

The treatment of electrolyte imbalance varies depending on the underlying mechanism:

6.1 Hyponatremia

- SIADH: fluid restriction, oral salt tablets, tolvaptan in resistant cases [32].
- CSWS: isotonic or hypertonic saline with fludrocortisone [33].

6.2 Hypernatremia

Managed with hypotonic fluids and correcting fluid deficits while monitoring serum sodium rise carefully to avoid cerebral oedema [34].

6.3 Hypokalemia

Supplemented with oral or IV potassium chloride, considering ECG monitoring for arrhythmias [35].

6.4 Hypocalcemia and Hypomagnesemia

IV calcium gluconate or magnesium sulphate, especially in seizure-prone patients [36]. Despite these standard protocols, adherence varies across Indian institutions due to financial constraints and uneven training [37-39].

Electrolyte Disorder	Treatment	Monitoring Frequency	Caution
Hyponatremia (SIADH)	Fluid restriction, Tolvaptan	6–12 hr	Overcorrection risk
CSWS	3% saline, fludrocortisone	6 hr	Volume overload
Hypokalemia	Oral/IV K^+ replacement	6 hr	Arrhythmia risk
Hypernatremia	0.45% saline, water intake	12 hr	Cerebral oedema

Table 6: Management Protocols for Electrolyte Imbalances in Stroke

7. Regional Disparities and Challenges in India

India's vast geographical and socioeconomic diversity introduces disparities in diagnosis and treatment of electrolyte imbalances. In tertiary care centers like AIIMS or PGIMER, sophisticated diagnostic modalities are available. In contrast, district hospitals often lack consistent electrolyte monitoring capabilities [40].

Rural patients also face delays in reaching stroke-ready hospitals, further worsening prognosis [41]. Poor nutrition, dehydration due to hot climate, and underlying comorbidities such as diabetes or hypertension complicate electrolyte profiles [42-43].

8. Mortality and Outcome Correlations

Multiple Indian studies affirm that patients with electrolyte imbalances, particularly hyponatremia and hypernatremia, have significantly worse outcomes [44-46]. For instance, a Hyderabad-based cohort showed that hyponatremic stroke patients had twice the mortality risk compared to normonatremic counterparts [47].

Moreover, abnormal sodium levels at admission were associated with a higher NIH Stroke Scale (NIHSS) score, prolonged ICU stay, increased ventilator need, and a poor functional outcome at 30 days [48].

9. Conclusion and Future Directions

In Indian clinical settings, Electrolyte imbalances are often exacerbated by late hospital arrival, pre-existing malnutrition, dehydration, and lack of awareness. Given India's heterogeneity in healthcare infrastructure and access, understanding electrolyte trends in the acute stroke population can guide clinical decisions and improve survival and recovery. Routine electrolyte screening, early identification of SIADH or CSWS, and timely correction are essential to improve outcomes. Given India's diversity, region-specific strategies are necessary to address resource gaps and improve clinician awareness.

Further large-scale, multicenter prospective studies are warranted to establish standardized protocols and improve care equity.

References:

1. Pandian JD, Sudhan P. Stroke epidemiology and stroke care services in India. J Stroke. 2013;15(3):128–34.
2. Sharma VK, Tsivgoulis G, Tan JC, et al. The impact of electrolyte imbalance in stroke. Int J Stroke. 2008;3(2):122–7.
3. Saleem S, et al. Electrolyte abnormalities in patients with stroke. J Ayub Med Coll Abbottabad. 2009;21(2):34–6.
4. Kumar S, et al. A study on hyponatremia in patients of cerebrovascular accidents. Indian J Neurosci. 2018;4(1):18–23.
5. Palmer BF. Hyponatremia in patients with central nervous system disease: SIADH vs. CSWS. Nat Clin Pract Nephrol. 2003;1(1):17–27.
6. Sherlock M, et al. The incidence and pathophysiology of hyponatraemia after subarachnoid haemorrhage. Clin Endocrinol (Oxf). 2006;64(3):250–4.
7. Tapia J, et al. SIADH in stroke: cause or consequence? Rev Neurol. 2005;40(5):259–64.
8. Leonard J, et al. Cerebral salt wasting vs SIADH. Nephrol Dial Transplant. 2002;17(10):1684–8.
9. Harrigan MR. Cerebral salt wasting syndrome. Crit Care Clin. 2001;17(1):125–38.
10. Misra UK, Kalita J. Cerebral salt wasting syndrome: an update. Neurol India. 2008;56(1):49–54.
11. Patil G, et al. Electrolyte disturbances in acute stroke. Int J Adv Med. 2017;4(3):763–6.
12. Rajeev A, et al. Prevalence and pattern of hyponatremia in acute stroke. J Assoc Physicians India. 2014;62(10):29–31.
13. Nair N, et al. Electrolyte abnormalities in acute stroke: a multicentric study. J Clin Diagn Res. 2016;10(5):OC07–OC10.
14. Aggarwal A, et al. Hyponatremia in stroke. J Indian Acad Clin Med. 2010;11(1):24–9.
15. Subramanian S, et al. Hyponatremia in stroke: SIADH or CSWS? Indian J Endocrinol Metab. 2013;17(4):761–3.
16. Shukla G, et al. Hypernatremia in stroke patients: incidence and outcome. Neurol India. 2016;64(4):709–12.
17. Chauhan P, et al. Clinical profile of electrolyte imbalance in stroke. J Assoc Physicians India. 2020;68(6):21–6.
18. Thakur SK, et al. Hypernatremia as a predictor of mortality in stroke. Indian J Crit Care Med. 2019;23(12):556–61.
19. Joshi R, et al. Hypokalemia in acute stroke. J Indian Med Assoc. 2015;113(7):36–9.
20. Ghosh S, et al. Electrolyte imbalance and stroke severity. Indian J Med Sci. 2012;66(1):27–34.
21. Mehta S, et al. Hypocalcemia in acute stroke: clinical significance. J Neurosci Rural Pract. 2015;6(3):325–9.
22. Reddy S, et al. Hypomagnesemia in stroke patients. Indian J Neurotrauma. 2011;8(1):21–5.
23. Sharma PK, et al. Electrolyte disturbances and their impact on stroke outcome. J Clin Neurosci. 2016;33:1–6.
24. Lal A, et al. SIADH in acute stroke. J Assoc Physicians India. 2010;58(11):689–91.

25. Sharma A, et al. Impact of hyponatremia on stroke severity. J Neurosci Rural Pract. 2013;4(2):139–42.
26. Gupta A, et al. Electrolyte abnormalities and ICU stay in stroke. AIIMS J Med. 2019;2(1):45–9.
27. Chatterjee S, et al. Impact of hypokalemia in stroke: A tertiary centre study. J Indian Acad Clin Med. 2017;18(2):125–8.
28. Bassi P, et al. Electrolyte management in stroke: practical protocols. J Clin Med. 2020;9(2):431.
29. Agarwal R, et al. Guidelines for hyponatremia in neurological diseases. Indian J Crit Care Med. 2019;23(Suppl 1):S22–S30.
30. Singh AK, et al. Role of laboratory monitoring in stroke. Indian J Med Res. 2018;147(5):389–92.
31. Mishra UK, et al. Challenges in rural stroke care. Neurol India. 2012;60(5):541–4.
32. Ellison DH, et al. Treatment of SIADH. N Engl J Med. 2007;356(20):2064–72.
33. Berendes E, et al. CSWS management in stroke patients. Acta Neurochir Suppl. 2006;96:225–30.
34. Adrogué HJ, et al. Hypernatremia therapy in critical illness. N Engl J Med. 2000;342(20):1493–9.
35. Smolarz A, et al. ECG monitoring in hypokalemia. J Electrocardiol. 2021;66:45–50.
36. Polderman KH. Calcium and magnesium in critical illness. Crit Care Med. 2006;34(6):1885–93.
37. Singh R, et al. Healthcare inequalities in stroke care. Lancet Glob Health. 2020;8(10):e1239–e1240.
38. Pandian JD, et al. National stroke care audit. Indian J Med Res. 2015;141(4):486–91.
39. Narayan SK, et al. Medical education and stroke management. Natl Med J India. 2017;30(1):18–21.
40. Saxena SK, et al. Rural stroke units in India. Stroke. 2015;46(2):476–80.
41. Dandona R, et al. Stroke burden in India: GBD study. Lancet Neurol. 2018;17(11):877–89.
42. Bansal A, et al. Malnutrition and stroke risk in India. Nutr Neurosci. 2021;24(5):396–403.
43. Chhabra P, et al. Role of comorbidities in stroke outcomes. Indian Heart J. 2020;72(1):31–6.
44. Mohan KM, et al. Hyponatremia and stroke outcome: a study in South India. J Stroke Cerebrovasc Dis. 2019;28(2):398–404.
45. Yadav KS, et al. Impact of electrolyte derangement in stroke: A tertiary care experience. J Clin Diagn Res. 2022;16(4):OC15–OC19.
46. Kumar D, et al. Sodium abnormalities and mortality in acute stroke. J Assoc Physicians India. 2019;67(12):29–33.
47. Rao S, et al. Predictors of poor prognosis in stroke. Indian J Stroke. 2016;8(3):165–70.
48. Singh V, et al. Electrolyte imbalance at admission and outcomes in stroke. J Indian Med Assoc. 2023;121(3):15–21.