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Electrolyte Disorders in the Acute Phase of Ischemic Stroke in the University Hospitals of Ouagadougou, Burkina Faso: Prevalence and Association with In-Hospital Mortality

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Abstract

Introduction: The objective of this study was to assess electrolyte disorders among patients hospitalized for ischemic stroke (IS) in the University Hospitals (UHs) of Ouagadougou. **Methods:** We conducted a retrospective, cross-sectional, descriptive, and analytical study of adult patients hospitalized for ischemic stroke—confirmed by brain CT or MRI—between January 1, 2022, and May 31, 2024. Socio-demographic, clinical, neuroimaging, blood laboratory (including electrolytes at admission), therapeutic, and in-hospital outcome data were analyzed using Epi Info 7.1.5.2. To identify electrolyte disorders associated with in-hospital mortality, we performed univariate analyses followed by multivariate analysis using backward logistic regression. The dependent variable was in-hospital mortality (yes/no), and the electrolyte abnormalities at admission served as independent variables. Statistical significance was set at p-value <0.05. **Results:** A total of 249 patients were included. The mean age was 63.2 years, and males accounted for 60.2% of cases. At admission, impaired consciousness (25.7%) and moderate neurological deficit (73.5%) were frequently observed. Middle cerebral artery ischemia (73.1%) was the most common stroke territory. The most frequent electrolyte disorders were hypokalemia (26.5%), hypocalcemia (26.5%), hypoproteinemia (22.6%), hyponatremia (18.3%), hypomagnesemia (17.5%), and hypochloremia (14.2%). The mean length of hospital stay was 8.6 days, and in-hospital mortality was 20.5%. Hyperkalemia (OR 1.40; p = 0.01), hyperchloremia (OR 4.91; p = 0.04), and hypermagnesemia (OR 6.48; p = 0.04) were significantly and independently

associated with in-hospital mortality. **Conclusion:** Electrolyte disorders are common in the acute phase of IS and are associated with an increased in-hospital mortality. Early detection, prevention, and management of these abnormalities could help improve outcomes in IS.

Keywords

Ischemic Stroke, Electrolyte Disorders, In-Hospital Mortality, University Hospitals, Ouagadougou

1. Introduction

Stroke is a severe neurological condition and a major global public health problem. Its burden is particularly heavy in sub-Saharan Africa, where long delays in medical care, the absence of stroke units, the lack of proven effective therapies such as thrombolysis and mechanical thrombectomy, and limited resuscitation resources significantly worsen outcomes. In this context, in-hospital mortality has been estimated at 22% in a recent meta-analysis [1]. In addition to the morbidity directly related to brain injury, biological and metabolic abnormalities occurring during the acute phase of ischemic stroke (IS)—particularly electrolyte disorders—appear to be associated with early poor vital and functional outcomes [2].

The high mortality and morbidity associated with stroke are partly due to serious complications, among which electrolyte imbalances play an important role [3] [4]. Electrolytes such as sodium, potassium, calcium, and magnesium are essential for normal neuronal function [5]. Their disruption during the acute phase of stroke is common and directly influences prognosis. Hyponatremia (11% - 33%) is associated with an increased risk of death; hypokalemia (18%) is linked to higher mortality and poorer functional outcomes; hypocalcemia (11% - 28%) is associated with a higher risk of hemorrhagic transformation of the infarct; and hypomagnesemia (10% - 20%) correlates with worse outcomes and larger volumes of IS [6] [7].

While several international studies have established an association between these electrolyte disorders and unfavorable outcomes, no specific study on this topic has been conducted in Burkina Faso, despite the high prevalence of stroke and its significant mortality rate.

This study, therefore, aims to fill this gap by determining the prevalence of electrolyte disorders during the acute phase of IS and evaluating their impact on in-hospital mortality among patients admitted to the University Hospitals (UHs) of Ouagadougou. The ultimate goal is to improve patient management and potentially reduce stroke-related morbidity and mortality in our setting through targeted correction of these abnormalities.

2. Patients and Methods

This was a descriptive and analytical study based on a retrospective collection of

data. It was conducted in the three main UHs of Ouagadougou, Burkina Faso—Yalgado Ouédraogo UH, Bogodogo UH, and Tengandogo UH—over the period from January 1, 2022, to May 31, 2024. These hospitals serve as national referral centers for the management of neurological disorders.

2.1. Inclusion Criteria

The study included adult patients (>16 years) hospitalized for ischemic stroke (IS) confirmed by brain computed tomography (CT) performed within 72 hours of symptom onset, with complete medical records including serum electrolytes at admission.

2.2. Exclusion Criteria

Excluded were incomplete medical files, patients hospitalized for neurological conditions other than IS, and cases lacking interpretable serum electrolyte measurements at admission.

2.3. Data Collection and Analysis

Data were extracted from medical charts, hospitalization registries, and follow-up forms between August and October 2024. A comprehensive set of variables was analyzed, including socio-demographic characteristics, clinical data, laboratory parameters (including serum electrolyte profiles), brain CT findings obtained at admission, stroke etiologies, treatment modalities, and in-hospital outcomes. Statistical analyses were performed using Epi Info version 7.1.5.2. The analysis included descriptive statistics as well as univariate analysis and multivariate logistic regression to identify electrolyte abnormalities independently associated with in-hospital mortality. Variables for which p-value was <0.20 were included in the multivariate logistic regression model.

Authorization from the administrative authorities of all participating UHs was obtained prior to the study. Patient confidentiality was strictly preserved through anonymization and coded data collection procedures.

2.4. Operational Definitions

Due to the unavailability of certain laboratory tests, the study focused on the following serum electrolytes: sodium, potassium, calcium, magnesium, chloride, and total proteins.

In this study:

- Hyponatremia was defined as serum sodium <135 mmol/L and hypernatremia as >145 mmol/L;
- Hypokalemia as serum potassium <3.5 mmol/L and hyperkalemia as >5.0 mmol/L;
- Hypochloremia as serum chloride <96 mmol/L and hyperchloremia as >106 mmol/L;
- Hypocalcemia as serum calcium <2.20 mmol/L and hypercalcemia as >2.60

mmol/L;

- Hypomagnesemia as serum magnesium <0.50 mmol/L and hypermagnesemia as >0.95 mmol/L;
- Hypoproteinemia as total serum proteins <60 g/L.

3. Results

Over a 29-month period (January 2022 to May 2024), 1539 patients were admitted to the neurology departments of the UHs of Ouagadougou for stroke, including 1016 ischemic strokes, of which 249 patients were ultimately included in the study.

Male patients were predominant (60.2%), with a male-to-female sex ratio of 1.1. The mean age was 63.2 years (±14.0), with the 60-75-year age group being the most represented (45.4%). Hypertension, dyslipidemia, previous stroke, and sedentary lifestyle—with 166 cases (66.7%), 89 cases (35.7%), and 43 cases (17.3%), respectively—were the most frequently observed vascular risk factors. Comorbidities were found in 26 patients (10.4%). Table 1 below presents the distribution of patients according to sociodemographic characteristics, vascular risk factors, and comorbidities.

Table 1. Distribution of patients according to sociodemographic characteristics, vascular risk factors, and comorbidities (n = 249).

Variables	Number (n = 249)	Percentage (%)
Sex		
Male	150	60.2
Female	99	39.8
Age groups		
≤30 years	6	2.4
31 - 45 years	25	10.0
46 - 60 years	61	24.5
61 - 75 years	113	45.4
>75 years	44	17.7
Vascular risk factors		
Hypertension	166	66.7
Dyslipidemia	89	35.7
History of stroke	43	17.3
Sedentary lifestyle	43	17.3
Alcohol use	38	15.3
Smoking	33	13.3

Continued

Diabetes mellitus	26	10.4
Obesity	17	6.8
Oral contraception	1	0.4
Comorbidities	26	10.4
Chronic psychosis	8	3.2
Peptic ulcer disease	7	2.8
History of stroke with neurological sequelae	5	2.0
Cardiomyopathy	5	2.0
HIV infection	3	1.2
Gout	2	0.8
Prior myocardial infarction	1	0.4
Chronic kidney disease	1	0.4
History of herpes zoster	1	0.4

The mean time to hospital admission was 26.4 ± 8 hours, with extremes ranging from 4 to 72 hours. The mean NIHSS at admission was 12 ± 4.3, with values ranging from 1 to 30. At admission, 64 patients (25.7%) presented with altered consciousness, and 183 patients (73.5%) had a moderate neurological deficit (NIHSS between 5 and 15). The middle cerebral artery territory was the most frequently affected, with 182 cases (73.1%). Early signs of ischemia were observed in 35 patients (14%). Atherosclerosis (27.3%) and cardioembolic disorders (19.3%) were the most common etiologies. Table 2 below presents the distribution of patients according to clinical findings, brain CT scan results at admission, and the etiologies of ischemic stroke.

Table 2. Distribution of patients according to clinical characteristics, brain ct findings at admission, and etiologies of ischemic stroke (n = 249).

Variables	Number	Percentage (%)
Clinical characteristics at admission		
Impaired consciousness (Glasgow Coma Score <13)	78	32.3
Non-comatose impaired consciousness	64	25.7
Coma (GCS 4 - 8)	14	5.6
Fever	20	8.0
Oxygen desaturation	17	6.8
Hypertension at admission	150	60.2

Continued

NIHSS		
1 - 4 (minor neurological deficit)	52	20.9
5 - 15 (moderate deficit)	183	73.5
16 - 20 (severe deficit)	8	3.2
>20 (very severe deficit)	6	2.4
Brain CT findings		
Early signs of cerebral ischemia	35	14.0
Hyperdense arteries	21	8.4
Sulcal effacement	4	1.6
Loss of gray-white matter differentiation	10	4.0
Leukoaraiosis	43	17.3
Old ischemic scars	13	5.2
Ischemic stroke territory		
Middle cerebral artery territory	182	73.1
Anterior cerebral artery territory	23	9.2
Anterior choroidal artery	14	5.6
Vertebrobasilar territory	36	14.4
Posterior cerebral artery	25	10.0
Cerebellar artery	6	2.4
Multilevel vertebrobasilar	8	3.2
Multifocal	14	5.6
Etiologies of ischemic stroke		
Atherosclerosis	68	27.3
Cardioembolic	48	19.3
Lacunar (small-vessel disease)	26	10.4
Undetermined etiology	107	43.0

At admission, several biological abnormalities were identified, predominantly electrolyte disorders. Hypokalemia and hypocalcemia were the most frequent disorders, each observed in 65 patients (26.5%). Hypoproteinemia was recorded in 50 patients (22.6%), while hyponatremia, hypomagnesemia, and hypochloremia were noted in 45 patients (18.3%), 42 patients (17.5%), and 35 patients (14.2%), respectively. In addition, other laboratory investigations revealed anemia in 32% of pa-

tients, hyperglycemia in 37.3%, an elevated C-reactive protein (CRP) level in 75.8% of those tested, and elevated serum creatinine in 25.8% of cases. Table 3 summarizes the distribution of electrolyte abnormalities and other biological disorders at admission.

Table 3. Distribution of patients according to electrolyte disturbances and other biological abnormalities at admission (n = 249).

Parameter	Tested (n)	% Tested	Abnormality	n	%
Sodium	246	98.8%	Decreased	45	18.3%
			Increased	16	6.5%
Potassium	245	98.4%	Decreased	65	26.5%
			Increased	17	7.0%
Chloride	247	99.2%	Decreased	35	14.2%
			Increased	24	9.7%
Magnesium	240	96.4%	Decreased	42	17.5%
			Increased	16	6.7%
Calcium	243	97.6%	Decreased	65	26.7%
			Increased	5	2.1%
Total proteins	221	88.8%	Decreased	50	22.6%
			Increased	12	5.4%
Test	**Tested (n)**	**%**	**Abnormality**	**n**	**%**
Complete blood count (CBC)	244	98.0%	Anemia	78	32.0%
			Leukocytosis	63	25.8%
			Leukopenia	5	2.0%
			Thrombocytopenia	36	14.8%
			Thrombocytosis	12	4.9%
Blood glucose	241	96.8%	Hyperglycemia	93	37.3%
			Hypoglycemia	12	4.8%
C-reactive protein (CRP)	66	26.5%	Elevated CRP	50	75.8%
Renal function tests	240	96.4%	Elevated creatinine	62	25.8%
			Elevated blood urea nitrogen	52	21.7%
Coagulation profile	23	9.2%	Low prothrombin time (PT)	9	39.1%
			Prolonged activated partial thromboplastin time (aPTT)	3	13.0%
			Elevated INR	1	4.3%

Correction of electrolyte disorders was performed in 41% of the affected patients. Antiplatelet therapy was administered to 95.6% of cases.

The mean length of hospital stay was 8.6 days. A total of 51 patients died during hospitalization, corresponding to an in-hospital mortality rate of 20.5%. The immediate causes of hospital deaths included direct stroke-related complications and respiratory distress (each accounting for 60.8%), often combined with infections or sepsis (52.9%).

At hospital discharge, among survivors, only 18.7% were functionally independent (mRS 0 - 2), whereas 81.3% had moderate to severe disability (mRS 3 - 5).

On univariate analysis, several electrolyte disorders were significantly associated with an increased risk of in-hospital mortality: hypernatremia (OR = 5.15; p = 0.01), hyperkalemia (OR = 2.68; p = 0.001), hyperchloremia (OR = 10.38; p < 0.001), hypomagnesemia (OR = 3.36; p = 0.03), hypermagnesemia (OR = 8.30; p < 0.001) and hypocalcemia (OR = 3.58; p = 0.01).

After multivariate analysis, hyperkalemia (adjusted OR = 1.40; 95% CI [1.12 - 5.79]; p = 0.01), hyperchloremia (adjusted OR = 4.91; 95% CI [1.04 - 23.14]; p = 0.04), and hypermagnesemia (adjusted OR = 6.48; 95% CI [1.07 - 39.19]; p = 0.04) were independently associated with in-hospital mortality (Table 4). Table 5 below presents the results of the multivariate analysis using logistic regression on the impact of serum electrolyte disturbances on hospital mortality in patients admitted for IS.

Table 4. The results of the univariate analysis assessing the impact of serum electrolyte disturbances on in-hospital mortality.

Variables	In-hospital mortality		Total (n = 249)	p-value
	Yes (n = 51)	No (n = 198)		
Hyponatremia				
Yes	2	43	45	0.80
No	49	155	204	
Hypernatremia				
Yes	3	13	16	0.01
No	48	185	233	
Hypokalemia				
Yes	5	60	65	0.30
No	46	138	184	
Hyperkalemia				
Yes	2	15	17	0.001
No	49	183	232	

Continued

Hypochloremia				
Yes	3	32	35	0.34
No	48	166	214	
Hyperchloremia				
Yes	6	18	24	0.000
No	45	162	225	
Hypomagnesemia				
Yes	5	37	42	0.03
No	46	161	207	
Hypermagnesemia				
Yes	4	12	16	0.000
No	47	186	233	
Hypocalcemia				
Yes	7	58	65	0.01
No	44	140	184	
Hypercalcemia				
Yes	2	3	5	0.60
No	49	195	244	

Table 5. Multivariate analysis of the impact of electrolyte disturbances on in-hospital mortality in patients admitted for IS.

Variables	In-hospital mortality		Total	OR [95% CI]	p-value
	Yes	No			
Hypernatremia					
Yes	2	43	45	1.00 [0.15 - 6.82]	0.99
No	49	155	204		
Hyperkalemia					
Yes	2	15	17	1.40 [1.12 - 5.79]	0.01
No	49	183	232		
Hyperchloremia					
Yes	6	18	24	4.91 [1.04 - 23.14]	0.04
No	45	162	225		

Continued

Hypomagnesemia					
Yes	5	37	42	3.92 [0.90 - 16.99]	0.06
No	46	161	207		
Hypermagnesemia					
Yes	4	12	16	6.48 [1.07 - 39.19]	0.04
No	47	186	233		
Hypocalcemia					
yes	2	3	5	2.15 [0.59 - 7.76]	0.24
No	49	195	244		

4. Discussion

Study Limitations

This study presents several potential biases that must be considered when interpreting the results:

Patient selection was limited to those admitted within ≤72 hours after stroke onset and who could undergo brain CT scanning within ≤12 hours of admission. This criterion excluded patients admitted later than 72 hours and/or those without rapid access to imaging.

Data quality depended on the completeness of electrolyte panels and medical records, which may have led to an underestimation of certain parameters.

Despite these limitations, the results are consistent and allow for meaningful comparison with the existing literature.

Electrolyte disorders such as hyponatremia, hypokalemia, hypocalcemia, and hypomagnesemia are common during the acute phase of stroke [8]. Data on other electrolytes (chloride, magnesium, etc.) are much more limited. There is a trend toward higher frequency and greater severity of electrolyte disorders in hemorrhagic strokes compared to ISs [9].

The prevalence of hyponatremia in our study was 18.3%, which falls within the 7% - 59% range reported in the international literature [4] [10]-[12]. Syndrome of inappropriate antidiuretic hormone secretion (SIADH) and cerebral salt-wasting syndrome are among the main mechanisms of hyponatremia in the acute phase of stroke [13]. Additional contributing factors include low-sodium or unsalted diets, use of certain medications (antihypertensives, antidepressants, nonsteroidal anti-inflammatory drugs, some antibiotics), diabetes mellitus, and heart failure [14]. In the context of IS, hyponatremia is primarily studied as a prognostic factor and predictor of mortality. In our cohort, no statistically significant association was found between hyponatremia and IS-related mortality.

The prevalence of hypernatremia in our study was 6.5%, comparable to Lompo *et al*., who reported 6% among IS patients in Burkina Faso [4]. In the United States

and Germany, Aiyagari *et al*., Lindner *et al*., and Polderman *et al*. found hypernatremia prevalences ranging from 5% to 9% in stroke patients admitted to intensive care units [15] [16], placing our findings within this range. Causes of hypernatremia in hospitalized patients are diverse, including insufficient hydration, impaired thirst perception, renal (diuretics, diabetes) or extra-renal fluid losses (respiratory, gastrointestinal), and excessive sodium administration during correction attempts [17]. In the context of IS, hypernatremia is also evaluated as a prognostic factor and predictor of mortality, but no statistically significant association was observed in our study.

The prevalence of hypokalemia in our study was 26.5%, compared to 19.2% reported by Lompo *et al*. among IS patients [4]. Our higher prevalence may be explained by reduced oral intake related to dysphagia and underlying malnutrition, as well as stress-induced neuroendocrine responses (catecholamine surge, activation of the renin-angiotensin-aldosterone system) that enhance renal potassium loss. In addition, osmotic diuresis due to uncontrolled hyperglycemia and the use of β-agonists or insulin during acute management can shift potassium intracellularly and further contribute to hypokalemia in this setting [18]. In this study, the association between hypokalemia and in-hospital mortality after IS was not statistically significant.

The prevalence of hyperkalemia was 7%, close to the 6% reported by Lompo *et al*. [4]. Hyperkalemia in stroke patients remains relatively uncommon and is often influenced by medications used to manage vascular risk factors, such as angiotensin-converting enzyme inhibitors or potassium-sparing diuretics, which impair renal potassium excretion and thereby increase the risk of hyperkalemia. Additionally, too-tight tourniquet application or excessive fist-clenching during blood collection can lead to spurious (pseudo-) hyperkalemia, resulting in falsely elevated serum potassium levels [19] [20].

The prevalences of hypochloremia and hyperchloremia were 14.2% and 9.7%, respectively, comparable to 14.81% and 13.58% reported by Lompo *et al*. [4]. In a Chinese neuro-intensive care cohort of acute stroke patients, prevalence of hyperchloremia was 8.6% at admission and rose to 17.0% within the first 72 h [21]. In a Bangladeshi series, among ischaemic strokes, hypochloremia was present in 30.25% and hyperchloremia in 3.36% [12]. Hypochloremia may result from diuretic use for hypertension management, leading to excessive chloride loss, whereas hyperchloremia may be related to overzealous saline infusion [22].

Hypomagnesemia was observed in 17.5% of patients. In South Korea, Ryu *et al*. reported hypomagnesemia in 10% - 20% of stroke patients [23], while Pradhan *et al*. in India reported 32.81% [24]. Our prevalence falls within this global range. Contributing factors may include diuretic therapy for hypertension and malnutrition, which is common in hospitalized patients with swallowing difficulties or altered consciousness [25].

The prevalence of hypermagnesemia was 6.7% in our series, potentially due to high magnesium intake or renal insufficiency [26]. Hypermagnesemia remains rare or under-reported in this context [27].

Hypocalcemia was found in 26.7% of patients. Hossain *et al*. reported 11.8%, whereas Pradhan *et al*. reported 28.12% among IS patients [8] [24]. Our prevalence lies within this broad range. Hypocalcemia in stroke patients is often associated with worsened prognosis, although the exact mechanisms remain partially unclear [28]. However, in this study, the association between hypocalcemia and in-hospital mortality after IS was not statistically significant.

In our study, the prevalence of hypercalcemia was 2.1%. In other series hypercalcemia at the acute phase of ischemic stroke has been uncommon: a recent systematic review reported hypercalcemia in about 5.6% of acute IS patients [8] [29].

Single-center cohorts from Asia and other settings similarly report low rates (generally <6%) falling within this range. Beyond serum calcium levels, intracellular calcium disturbances are almost universally observed after IS. This is due to excess glutamate release during ischemia, which causes calcium overload in neurons, leading to neuronal injury. These intracellular disturbances are frequent and contribute to the expansion of the ischemic penumbra [30].

Calcium imbalances, particularly hypercalcemia, appear to be associated with worse prognosis in IS patients, with higher rates of mortality and complications [31].

Hypoproteinemia was present in 22.6% of patients in our cohort. Lompo *et al*. reported a prevalence of 15.2% [4]. In some cohort studies of acute ischemic strokes, serum albumin (the main component of plasma proteins) is reduced in 45.5% of patients [32]. The mechanisms implicated include leakage of plasma proteins into the ischemic brain parenchyma, accelerated catabolism (proteolysis) due to metabolic stress, and decreased protein synthesis due to systemic inflammation induced by stroke [33]. In our context, malnutrition, common among hospitalized patients, contributes significantly to hypoproteinemia [4]. Hypoproteinemia is associated with poorer outcomes in IS, including an increased risk of infections and prolonged hospital stay [4] [34].

In our study, the in-hospital mortality among patients admitted for IS was 20.5%, higher than the 17.9% reported by Lompo *et al*. [4]. Harada *et al*. in Japan and Lee *et al*. in the United States reported lower mortality rates of 5.7% and 6.8%, respectively [35] [36], likely due to early stroke unit care, intravenous thrombolysis, and mechanical thrombectomy in high-income countries.

In multivariate analysis, hyperkalemia (OR = 1.40; p = 0.01), hyperchloremia (OR = 4.91; p = 0.04), and hypermagnesemia (OR = 6.48; p = 0.04) were significantly associated with in-hospital mortality in IS patients. Elevated potassium levels can exacerbate ischemic neuronal injury. During acute stroke, neuronal depolarization increases extracellular potassium, and systemic hyperkalemia may worsen this effect, enhancing cell death. Huang *et al*. reported that hyperkalemia may indirectly contribute to cerebral edema, a major cause of mortality in IS [11].

Berend *et al*. in the Netherlands found that hyperchloremia is associated with poor prognosis in IS, as it may worsen metabolic acidosis, detrimental to ischemic brain tissue. Hyperchloremia can disrupt osmotic balance and cellular volume,

potentially aggravating neurological damage, prolonging hospitalization, and increasing complications [37].

Harada *et al*. in Japan and Hoca *et al*. in Türkiye reported that hypermagnesemia is associated with worse prognosis and increased mortality in IS due to cardiovascular, respiratory, and neurological complications [35] [38]. Hypermagnesemia can impair blood coagulation and viscosity, potentially slowing cerebral circulation and limiting oxygen and nutrient delivery to ischemic areas. Additionally, hypermagnesemia-induced hypotension may further reduce cerebral perfusion, aggravating ischemic injury [39]. Early detection and correction of these electrolyte disturbances may prevent additional morbidity and mortality.

Elevated C-reactive protein (CRP) levels on admission, a marker of systemic inflammation, are recognized in series of patients with ischemic stroke as an independent predictor of mortality or poor functional prognosis [40]. However, in our series, only a small proportion of patients (26.5%) were tested for CRP, making its elevation reported in 75.8% of those tested unrepresentative of our patient population as a whole. Thus, in our context, the potentially prognostic role of CRP is debatable.

5. Conclusion

In our study, patients admitted for ischemic stroke (IS) exhibited electrolyte disturbances upon admission, the most frequent being hypokalemia, hypocalcemia, hypoproteinemia, hyponatremia, hypomagnesemia, and hypochloremia. Among these disturbances, hyperkalemia, hyperchloremia, and hypermagnesemia were significantly associated with increased in-hospital mortality. Systematic screening for electrolyte disturbances in all patients admitted for acute IS and their rapid correction could significantly reduce hospital mortality in IS patients in our context. Larger prospective studies are needed to further validate these findings.

Conflicts of Interest

There are no conflicts of interest.

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