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Research Article

Evaluation of Changes in the Blood Ionogram of Patients Admitted to Intensive Care and Correlation with Vital Prognosis at the CHC Military Hospital (Burkina Faso)

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Abstract

Introduction: Electrolyte disorders are a major issue in the management of intensive care unit (ICU) patients. However, the blood ionogram is often performed only at admission, which limits its use in intensive care, where the patient's condition changes constantly with treatment. This study aimed to evaluate changes in the blood ionogram of patients admitted to the ICU of the Capitaine Halassane Coulibaly Military Hospital (HM-CHC) and to analyse their association with prognosis. *Material and methods:* This was an analytical cross-sectional study with a retrospective phase (January to September 2025) and a prospective phase (October 2025 to April 2026), conducted in the ICU of the HM-CHC in Ouagadougou (Burkina Faso). Sodium, potassium, chloride, bicarbonate, calcium, magnesium and phosphate were measured at admission (T0), 24 h, 48 h, 72 h and 168 h (day 7, D7). Proportions were compared with the chi-square or Fisher's exact test and means with Student's t test. The association between D7 values and 28-day mortality was assessed with Spearman's correlation coefficient ($p < 0.05$). *Results:* Forty-four patients were included (mean age 37.6 ± 13.7 years; 81.8% men). The 28-day mortality was 36.4%. At admission, 95.5% of patients had at least one electrolyte disorder, the most frequent abnormalities involving phosphate (72.7%), calcium (70.5%) and magnesium (65.9%). Calcium disorders peaked at 24 h (81.8%, including 77.3% hypocalcaemia), phosphate disorders at 48 h (75.0%), and dysnatraemia reached 70.5% at 72 h. At D7, bicarbonate and magnesium abnormalities reached 84.1% and chloride abnormalities 81.8%. At D7, phosphate ($\rho = +0.790$), calcium ($\rho = -0.784$), chloride ($\rho = +0.773$) and bicarbonate ($\rho = -0.738$) were strongly correlated with mortality ($p < 0.001$), whereas sodium, magnesium and potassium were not. *Conclusion:* Electrolyte disorders were frequent, persistent and evolving during the first week in the ICU. Hyperphosphataemia, hypocalcaemia, hyperchloraemia and low bicarbonate at D7 could constitute a simple and accessible electrolyte dashboard to stratify the risk of mortality.

Keywords

Blood Ionogram, Intensive Care, Prognosis, Phosphate, Calcium, Burkina Faso

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1. Introduction

Homeostasis refers to the mechanisms by which the organism remains close to a point of equilibrium [1]. Among these balances, that of body water and electrolytes is essential for cell function: sodium, chloride, potassium, calcium, magnesium, phosphate and bicarbonate determine membrane potential, the excitability of nerve and muscle tissue and enzyme activity [2]. In clinical practice, this balance is assessed by the blood ionogram [3]. Although often regarded as a routine test, its results can drive vital decisions: dyskalaemia can cause severe cardiac arrhythmias [4], dysnatraemia can cause seizures and impaired consciousness [5], hypocalcaemia can destabilise haemodynamics [6], and severe hypophosphataemia can compromise respiratory function [7]. These disorders significantly increase morbidity and mortality in vulnerable patients, particularly the critically ill [8].

International data quantify the extent of the problem. In 151,486 ICU patients, Funk et al. showed that dysnatraemias present at admission were independent risk factors for hospital mortality, with a risk rising with severity, from an odds ratio of 1.32 for borderline hyponatraemia to 3.64 for severe hypernatraemia [9]. In the meta-analysis by Upala et al., hypomagnesaemia was associated with higher mortality in ICU patients (relative risk 1.90; 95% CI 1.48–2.44) [10]. In sub-Saharan Africa, the situation is more severe: patients often arrive late and severe malaria, infectious diarrhoea and protein-energy malnutrition further weaken water and electrolyte balance [11, 12]. At Gabriel Toure Teaching Hospital in Bamako (Mali), 93.3% of 75 brain-injured patients had water and electrolyte disorders at admission [11]. In Burkina Faso, the only recent hospital data concern children: in 193 children hospitalised for acute poisoning at the Charles de Gaulle Paediatric University Hospital in Ouagadougou, Soudre et al. reported a predominance of metabolic acidosis (64.23%), hypercalcaemia (43.59%) and hyponatraemia (33.78%) [13]. These data cannot be transposed to adult ICU patients.

In most African and many international studies, electrolytes are assessed at a single time point, generally at admission [9, 14]. In the ICU, however, the clinical condition changes continuously: a patient may have normal potassium at admission and develop hyperkalaemia within 24 hours as acute kidney injury progresses [15]. Recent work has begun to explore this kinetic dimension; for example, small early increases in creatinine not meeting the criteria for acute kidney injury were associated with subsequent severe acute kidney injury and mortality [16]. This kinetic approach remains almost absent from monitoring practices in African ICUs. We therefore conducted this study in the ICU of the Capitaine Halassane Coulibaly Military Hospital (HM-CHC) in Ouagadougou to evaluate changes in the blood ionogram (Na^+, K^+, Cl^-, HCO_3^-, Ca^{2+}, Mg^{2+} and phosphate) at T0, 24 h, 48 h, 72 h and 168 h (D7) and to analyse their association with 28-day mortality.

2. Material and Methods

2.1. Study Setting, Design and Period

The study was conducted in the ICU of the HM-CHC in Ouagadougou (Burkina Faso), a referral hospital for critically ill patients, notably polytrauma, shock, acute respiratory distress and severe infections. This was an analytical cross-sectional study with a retrospective data collection phase (January to September 2025) and a prospective phase (October 2025 to April 2026).

2.2. Patients

The study population consisted of all patients admitted to the HM-CHC ICU during the study period, with exhaustive sampling of those meeting the inclusion criteria. Patients were included if they had a blood ionogram at admission, at least one repeated ionogram during their stay, a hospital stay longer than 72 hours and a usable medical record. Patients who died or were transferred before the first ionogram, those hospitalised for less than 24 hours and those with incomplete or unusable medical records were not included.

2.3. Data Collection and Variables

Data were collected with a standardised form from patients' medical records, ICU registers and the registers of the HM-CHC biomedical laboratory. Variables comprised sociodemographic data (age, sex, occupation), clinical data (reason for admission, main diagnosis, APACHE II score, SOFA score on day 1, mechanical ventilation, length of stay, outcome) and biological data: serum sodium, potassium, chloride, bicarbonate, total calcium, magnesium and phosphate at T0, 24 h, 48 h, 72 h and 168 h (D7). Each value was classified as low, normal or high with respect to the following reference values [2, 3]: sodium 135–145 mmol/L, potassium 3.5–5.0 mmol/L, chloride 95–105 mmol/L, bicarbonate 22–26 mmol/L, total calcium 2.20–2.60 mmol/L, magnesium 0.75–0.95 mmol/L and phosphate 0.87–1.45 mmol/L. The outcome was vital status at day 28.

2.4. Data Analysis and Processing

Data were entered and analysed with Python 3.10.4. Quantitative variables were described as mean ± standard deviation when normally distributed, or as median and interquartile range otherwise; qualitative variables as absolute and relative frequencies. The frequency of each electrolyte abnormality was determined at each time point. Proportions were compared with the chi-square test (or Fisher's exact test when conditions were not met) and means between two groups with Student's t test after checking normality and homogeneity of variances. The association between electrolyte values at D7

and 28-day mortality was assessed with Spearman's correlation coefficient (ρ). The significance level was set at $p < 0.05$.

3. Results

3.1. Characteristics of the Patients

A total of 44 patients were included (Table 1). The mean age was 37.6 ± 13.7 years; patients aged 20 to 30 years accounted for 45.5% of the cohort and those over 50 years for 15.9%. Men accounted for 81.8% (sex ratio 4.5). Trauma was the leading reason for admission (36.4%), followed by postoperative care (27.3%) and sepsis or septic shock (6.8%). The mean APACHE II score was 16.1 ± 6.1 (median 14.5) and the mean SOFA score on day 1 was 6.8 ± 2.9. More than half of the patients (56.8%) required mechanical ventilation. The mean ICU length of stay was 12.9 ± 17.2 days. The 28-day mortality was 36.4% (16 deaths).

Table 1. *Characteristics of the patients (N = 44).*

Characteristic	n (%) or mean ± SD
Age (years), mean ± SD	37.6 ± 13.7
Age group	
20–30 years	20 (45.5)
31–50 years	17 (38.6)
> 50 years	7 (15.9)
Male sex	36 (81.8)
Reason for admission	
Trauma	16 (36.4)
Postoperative care	12 (27.3)
Sepsis or septic shock	3 (6.8)
Acute or chronic kidney failure	1 (2.3)
Rhabdomyolysis	1 (2.3)
Other	11 (25.0)
APACHE II score, mean ± SD (median)	16.1 ± 6.1 (14.5)
SOFA score on day 1, mean ± SD	6.8 ± 2.9
Mechanical ventilation	25 (56.8)
ICU length of stay (days), mean ± SD	12.9 ± 17.2
Death at day 28	16 (36.4)

SD: standard deviation; APACHE II: Acute Physiology and Chronic Health Evaluation II; SOFA: Sequential Organ Failure Assessment; ICU: intensive care unit.

3.2. Frequency of Electrolyte Disorders from T0 to D7

Electrolyte disorders were frequent throughout the first seven days (Table 2). At admission, 95.5% of patients had at least one disorder, and no electrolyte had an abnormality frequency below 45% at any of the five time points. At T0, the most frequent abnormalities involved phosphate (72.7%), calcium (70.5%) and magnesium (65.9%), dominated by hyperphosphataemia (68.2%) and hypocalcaemia (68.2%). At 24 h, calcium disorders peaked at 81.8% (77.3% hypocalcaemia) and dysnatraemia rose to 61.4%, whereas bicarbonate (45.5%) and potassium (47.7%) abnormalities decreased. At 48 h, phosphate disorders peaked at 75.0% and dysnatraemia reached 65.9%. At 72 h, dysnatraemia reached 70.5%, bicarbonate abnormalities 68.2% (25.0% high values) and hypomagnesaemia 61.4%. At D7, bicarbonate (84.1%) and magnesium (84.1%) abnormalities reached their highest frequencies, and chloride abnormalities rose to 81.8% (43.2% hypochloraemia and 38.6% hyperchloraemia). Between T0 and D7, the frequency of abnormalities increased by 22.8 percentage points for sodium, 31.8 for bicarbonate, 27.3 for chloride and 18.2 for magnesium; potassium was the only electrolyte whose abnormality frequency decreased (−4.5 points).

Table 2. *Frequency of blood ionogram abnormalities from admission (T0) to day 7 (D7), n (%), N = 44.*

Electrolyte / abnormality	T0	24 h	48 h	72 h	D7
Sodium (Na^+)					
Low	15 (34.1)	16 (36.4)	20 (45.5)	20 (45.5)	20 (45.5)
High	6 (13.6)	11 (25.0)	9 (20.5)	11 (25.0)	11 (25.0)
Total abnormal	21 (47.7)	27 (61.4)	29 (65.9)	31 (70.5)	31 (70.5)
Potassium (K^+)					
Low	9 (20.5)	8 (18.2)	9 (20.5)	9 (20.5)	11 (25.0)

Electrolyte / abnormality	T0	24 h	48 h	72 h	D7
High	16 (36.4)	13 (29.5)	12 (27.3)	14 (31.8)	12 (27.3)
Total abnormal	25 (56.8)	21 (47.7)	21 (47.7)	23 (52.3)	23 (52.3)
Chloride (Cl^-)					
Low	11 (25.0)	11 (25.0)	12 (27.3)	12 (27.3)	19 (43.2)
High	13 (29.5)	13 (29.5)	14 (31.8)	12 (27.3)	17 (38.6)
Total abnormal	24 (54.5)	24 (54.5)	26 (59.1)	24 (54.5)	36 (81.8)
Bicarbonate (HCO_3^-)					
Low	21 (47.7)	19 (43.2)	20 (45.5)	19 (43.2)	19 (43.2)
High	2 (4.5)	1 (2.3)	6 (13.6)	11 (25.0)	18 (40.9)
Total abnormal	23 (52.3)	20 (45.5)	26 (59.1)	30 (68.2)	37 (84.1)
Calcium (Ca^{2+})					
Low	30 (68.2)	34 (77.3)	33 (75.0)	31 (70.5)	25 (56.8)
High	1 (2.3)	2 (4.5)	2 (4.5)	3 (6.8)	8 (18.2)
Total abnormal	31 (70.5)	36 (81.8)	35 (79.5)	34 (77.3)	33 (75.0)
Magnesium (Mg^{2+})					
Low	18 (40.9)	23 (52.3)	23 (52.3)	27 (61.4)	36 (81.8)
High	11 (25.0)	5 (11.4)	6 (13.6)	3 (6.8)	1 (2.3)
Total abnormal	29 (65.9)	28 (63.6)	29 (65.9)	30 (68.2)	37 (84.1)
Phosphate (PO_4^{3-})					
Low	2 (4.5)	4 (9.1)	7 (15.9)	8 (18.2)	11 (25.0)
High	30 (68.2)	26 (59.1)	26 (59.1)	23 (52.3)	21 (47.7)
Total abnormal	32 (72.7)	30 (68.2)	33 (75.0)	31 (70.5)	32 (72.7)

Low and high values were defined with respect to the reference values given in section 2.3. D7: 168 h.

3.3. Longitudinal Changes and Association with Mortality

Comparison of mean electrolyte values between patients who died by day 28 and survivors showed three patterns: a progressive divergence from 24 h onwards for phosphate, bicarbonate and calcium; a similar decrease in both groups for magnesium; and relative stability for potassium. At D7, four of the seven electrolytes were strongly correlated with 28-day mortality ($|\rho| \geq 0.73$; $p < 0.001$) (Table 3): phosphate ($\rho = +0.790$; 4.46 ± 1.22 mmol/L in non-survivors versus 1.02 ± 0.52 mmol/L in survivors), calcium ($\rho = -0.784$; 0.97 ± 0.37 versus 2.32 ± 0.48 mmol/L), chloride ($\rho = +0.773$; 115.92 ± 5.42 versus 94.82 ± 9.42 mmol/L) and bicarbonate ($\rho = -0.738$; 9.27 ± 6.98 versus 28.15 ± 4.95 mmol/L). Sodium ($\rho = -0.192$; $p = 0.213$), magnesium ($\rho = +0.160$; $p = 0.299$) and potassium ($\rho = +0.075$; $p = 0.631$) did not significantly discriminate survivors from non-survivors.

Table 3. *Spearman correlation between electrolyte values at D7 and 28-day mortality (N = 44).*

Electrolyte (mmol/L)	Survivors (n = 28)	Non-survivors (n = 16)	ρ	p
Sodium (Na^+)	139.92 ± 11.83	134.69 ± 12.25	−0.192	0.213
Potassium (K^+)	4.29 ± 1.13	4.64 ± 1.56	+0.075	0.631
Chloride (Cl^-)	94.82 ± 9.42	115.92 ± 5.42	+0.773	< 0.001*

Electrolyte (mmol/L)	Survivors (n = 28)	Non-survivors (n = 16)	ρ	p
Bicarbonate (HCO_3^-)	28.15 ±4.95	9.27 ±6.98	−0.738	< 0.001*
Total calcium (Ca^{2+})	2.32 ±0.48	0.97 ±0.37	−0.784	< 0.001*
Magnesium (Mg^{2+})	0.62 ±0.13	0.70 ±0.23	+0.160	0.299
Phosphate (PO_4^{3-})	1.02 ±0.52	4.46 ±1.22	+0.790	< 0.001*

Values are mean ± SD. A negative ρ indicates that a low value is associated with death; a positive ρ that a high value is associated with death. *p < 0.05.

4. Discussion

This study has limitations. It was single-centre with a modest sample of 44 patients, which limits statistical power and generalisability. The diversity of reasons for admission (trauma, postoperative care, sepsis) introduces clinical heterogeneity that may influence electrolyte trajectories differently. Because only patients hospitalised for more than 72 hours were included, a survival bias cannot be excluded. The correlation analysis was univariate and not adjusted for severity scores, and confounding factors such as parenteral nutrition, diuretics and corticosteroids, which can modify the ionogram independently of prognosis, were not taken into account. Its strengths are the combination of retrospective and prospective phases, repeated measurements at five standardised time points, the use of statistical methods suited to non-normal distributions and the simultaneous study of seven electrolytes over a full week, an approach rarely reported in sub-Saharan Africa.

Our patients were young (mean age 37.6 ±13.7 years) and predominantly male (81.8%). Comparable profiles have been reported in African ICUs: in the polyvalent ICU of the Souro Sanou University Hospital in Bobo-Dioulasso, Ilboudo et al. reported a mean age of 33.63 ±15.62 years and a sex ratio of 7.23 among patients with severe head injury [17], and in Zinder (Niger), Amadou et al. reported a mean age of 36.45 years with 56.1% men [18]. Male predominance is explained by men's greater exposure to trauma and road traffic accidents, all the more so in a military hospital in a context of armed conflict. The mean APACHE II (16.1 ±6.1) and SOFA (6.8 ± 2.9) scores reflect moderate to high severity. The 28-day mortality of 36.4% is consistent with data from resource-limited ICUs: Abate et al. reported a 28-day mortality of 49% among 630 mechanically ventilated patients in southern Ethiopia [19], and Baelani et al. highlighted the insufficient resources available in Africa to treat severe sepsis and septic shock [20].

One of the main findings is the near-universal nature of electrolyte disorders: 95.5% of patients had at least one disorder at admission, and the frequency of abnormalities never fell below 45% for any electrolyte at any time point. In 355 medical ICU patients, Mohsin Iqbal et al. found electrolyte imbalances in 78.3%, and multiple abnormalities independently predicted ICU mortality (odds ratio 3.1) [21]. Hypocalcaemia was the most frequent mineral disorder at T0 (68.2%) and peaked at 24 h (77.3%). Chernow et al. found hypocalcaemia in 64% of ICU patients, associated with higher mortality [22]; Vivien et al. found ionised hypocalcaemia in 74% of severe trauma patients at hospital arrival [23]; and hypocalcaemia is reported in 55–85% of ICU patients [24]. Its mechanisms are multiple, including alterations of the parathyroid hormone–vitamin D axis in sepsis [25]. The persistence of hypocalcaemia in 56.8% of patients at D7 is clinically relevant.

Phosphate abnormalities affected 72.7% of patients at T0, dominated by hyperphosphataemia (68.2%), as expected in acute hypercatabolic states (trauma, rhabdomyolysis, sepsis). At D7, the distribution became bimodal, with emerging hypophosphataemia (25.0%) alongside predominant hyperphosphataemia (47.7%), consistent with refeeding and the late anabolic phase described by van Zanten et al. [26]. In a large South African hospital, severe hypophosphataemia (≤ 0.5 mmol/L) was associated with an in-hospital mortality of 30%, and 45% of cases occurred in intensive care [27]. Dysnatraemias increased from 47.7% at T0 to 70.5% at D7; hyponatraemia remained the most frequent form (45.5%), while hypernatraemia increased from 13.6% to 25.0%. Lindner et al. reported hypernatraemia in 9% of medical ICU patients, mostly acquired after admission, and identified it as an independent risk factor for mortality [28]. In our cohort, this increase probably reflects insensible water losses under mechanical ventilation, hypertonic fluids and renal dysfunction [29].

Magnesium showed a progressive depletion, from 65.9% abnormalities at T0 to 84.1% at D7, with 81.8% hypomagnesaemia at D7. Magnesium deficiency is common in critical illness and results mainly from gastrointestinal and renal losses [30]; it was not systematically supplemented in our setting. Bicarbonate abnormalities showed the largest increase (+31.8 points, 84.1% at D7), with a growing bimodal distribution: acidosis in the most severely ill patients and alkalosis in those recovering. In septic patients followed for five days, Noritomi et al. showed that metabolic acidosis, mainly hyperchloraemic, was more pronounced in non-survivors and was corrected in survivors but not in non-survivors [31]; metabolic alkalosis is also common in critically ill patients [32].

The trajectories of calcium and bicarbonate diverged between survivors and non-survivors from 24 h and the gap widened until D7. In 15,409 ICU admissions from the MIMIC II database, Zhang et al. found that moderate hypocalcaemia at admission (odds ratio 1.94) and hypocalcaemia of the mean ionised calcium during the stay were associated with an increased risk of death [33]. Closer monitoring of calcium from the first 24 hours could therefore help identify high-risk patients. For phosphate, the divergence was the most marked: phosphate rose continuously to 4.46 mmol/L at D7 in non-survivors, whereas it normalised in survivors. This pattern is consistent with ongoing cell destruction and acute kidney injury limiting phosphate excretion [15].

At D7, phosphate showed the strongest positive correlation with mortality ($\rho = +0.790$), and calcium a strong negative correlation ($\rho = -0.784$), with severe hypocalcaemia (0.97 mmol/L) in non-survivors. Steele et al. observed that severely hypocalcaemic ICU patients whose ionised calcium had not normalised by day 4 had twice the mortality of the others (38% vs. 19%), although the difference was not significant [6]. In the South African EpiC study, hypocalcaemia (ionised calcium < 1.15 mmol/L) was found in 52% of trauma patients at hospital arrival, and severe hypocalcaemia was associated with higher 30-day mortality, ICU admission and transfusion [34]. Hypocalcaemia may, however, be a marker of severity rather than a direct cause [24]. Bicarbonate ($\rho = -0.738$) and chloride ($\rho = +0.773$) formed the second prognostic axis. The fall in bicarbonate to 9.27 mmol/L in non-survivors reflects severe, uncorrected metabolic acidosis, and the hyperchloraemia (115.9 mmol/L) is consistent with hyperchloraemic acidosis [31]; Yunos et al. showed that a chloride-restrictive fluid strategy was associated with less acute kidney injury in ICU patients [35].

Sodium ($\rho = -0.192$), potassium ($\rho = +0.075$) and magnesium ($\rho = +0.160$) were not significantly correlated with mortality at D7. For sodium, the coexistence of hypo- and hypernatraemia in both groups may cancel out the overall correlation, and even borderline deviations of serum sodium carry prognostic weight [5], which a single correlation coefficient does not capture. For magnesium, depletion was similar in both groups, in contrast with the meta-analysis by Upala et al. [10]. For potassium, repeated corrections in both groups probably removed its discriminating value, as protocolised supplementation effectively prevents hypokalaemia in critically ill patients [36].

These results have practical implications. First, bicarbonate, magnesium and chloride abnormalities reached their maximum frequency at D7 and not at 72 h; stopping electrolyte monitoring at 72 hours, as is still often the case in resource-limited settings, would miss the latest and most prognostic changes. Second, the four parameters identified at D7 (phosphate, calcium, bicarbonate and chloride) can be measured in any functional hospital laboratory, and their joint measurement could provide a simple biological dashboard complementary to clinical scores. Third, the progressive magnesium depletion supports early, protocolised correction [30] and attention to refeeding hypophosphataemia [37]. For calcium, direct evidence that supplementation improves outcomes in critically ill patients is lacking, and correction should be guided by ionised calcium and clinical context [24].

5. Conclusion

Electrolyte disorders were the rule rather than the exception in our ICU patients and evolved over the first week, with an acute phase at T0, an intermediate phase at 72 h and a late phase at D7. Hypocalcaemia and hypomagnesaemia were early and persistent, whereas chloride and bicarbonate abnormalities increased at the end of the first week. Phosphate, calcium, bicarbonate and chloride measured at D7 were strongly correlated with 28-day mortality. Their joint measurement, simple and inexpensive, could constitute an electrolyte dashboard complementary to severity scores in resource-limited settings, which should be validated in larger multicentre cohorts with multivariable analysis. These results support extending electrolyte monitoring beyond 72 hours.

Abbreviations

APACHE II	Acute Physiology and Chronic Health Evaluation II
D7	Day 7 (168 h)
HM-CHC	Capitaine Halassane Coulibaly Military Hospital
ICU	Intensive Care Unit
MIMIC	Medical Information Mart for Intensive Care
SD	Standard Deviation
SOFA	Sequential Organ Failure Assessment

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Conflicts of Interest

The authors declare no conflicts of interest.

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