Research Article

Disorders of electrolyte and acid-base and its impact association in intensive care patients

Pradeep Kumar Dabla[1], Kumar Saurabh[1], Nilima Das[2], Jyoti Bala[3], Chandan Jha[1], Charbel Diwan[4], Ankit Sharma[2], Anirban Choudhuri[2]

[1]Department of Biochemistry, Govind Ballabh Pant Institute of Postgraduate Medical Education and Research, New Delhi, India, [2]Department of Anaesthesia and Intensive Care, Govind Ballabh Pant Institute of Postgraduate Medical Education and Research, New Delhi, India, [3]Department of Laboratory Medicine, Shree Aggarsain International Hospital, Rohini, Delhi, India, [4]Medical, Scientific and Clinical Affairs, Nova Biomedical Corporation, Waltham, MA, USA

Correspondence:
Dr. Anirban Choudhuri, Department of Anaesthesia and Intensive Care, Govind Ballabh Pant Institute of Postgraduate Medical Education and Research, New Delhi, India. E-mail: anirban.homchoudhuri@gmail.com

How to cite this article: Dabla PK, Saurabh K, Das N, Bala J, Jha C, Diwan C, *et al*. Disorders of electrolyte and acid-base and its impact association in intensive care patients. Innov Pharm Pharmacother 2021;9(2):37-43.

Source of Support: Nil.
Conflicts of Interest: None declared.

ABSTRACT

Aim: This study was aimed to assess acid-base and electrolyte disturbances among intensive care patients. **Materials and Methods:** The study was conducted in the Department of Biochemistry with the Department of Anaesthesia intensive care unit (ICU), *Govind Ballabh Pant* Institute of Postgraduate Medical Education and Research, Delhi, India. Thirty critically ill patients from the ICU were enrolled in the study and arterial blood gas (ABG) with serum electrolytes was performed within 24 h of admission. **Results:** Out of 30 patients, 14 (46.67%) patients had no respiratory disturbance and 16 (53.33%) developed respiratory complications; 3 (10%) had metabolic alkalosis, 7 (23.33%) had metabolic acidosis, 4 (13.33%) had respiratory alkalosis, and 2 (6.66%) suffered from respiratory acidosis. Hyponatremia was present in ICU patients both that got discharged (130.95 ± 11.62 mEq/L) after improvement and those who expired (133.31 ± 7.71 mEq/L) during hospital stay. Mean serum potassium levels and chloride levels were normal among discharged and expired patients. **Conclusion:** The present study showed that hyponatremia was the most common electrolyte disorder seen in our patients and metabolic acidosis was the most common acid-base disturbance. Electrolyte and acid-base disturbances, especially hyponatremia and acidosis, were highly associated with ICU mortality. These disturbances should be monitored closely, diagnosed early, and managed correctly during hospitalization and iatrogenic factors should be avoided.

Keywords: Arterial blood gas, critically ill patients, electrolyte, Glasgow Coma Scale, intensive care unit

Introduction

Electrolyte and acid-base disturbances are frequent and potentially dangerous complications seen commonly in critically ill patients admitted to the intensive care unit (ICU). The incidence of electrolyte and acid-base disturbances varies depending on the different underlying diseases and comorbidities.[1-3] ICU patients commonly suffer from disease states such as acute respiratory distress syndrome, uncontrolled diabetes mellitus, acute or chronic renal failure, acute or chronic respiratory failure, shock, severe cardiovascular events, trauma, multiple organ failure, and sepsis.[4,5] Critically ill patients with hypoxemia, deteriorating oxygen saturations, increasing breathlessness, or hypercapnia typically require measurement of blood gases for their management.[6]

Among the various diagnostic investigations carried out in ICU patients, arterial blood gas (ABG) analysis is one of the most common.[7] It is essential for assessing clinical oxygenation, acid-base status, and the degree of compensation that have occurred in these patients.[8] It provides important information about ventilation,

Access this article online

Website: www.innpharmacotherapy.com
Doi: 10.31690/ipp.2021.v09i02.003
e-ISSN: 2321-323X
p-ISSN: 2395-0781

oxygenation, and acid-base status.[9] It is essential to identify these changes to correct and treat the underlying conditions which cause them, and to maintain pH homeostasis and prevent organ dysfunction.

Cornock noted that the ABG is used to describe a set of values that permit the assessment of an individual's ability to maintain normal cell function.[10] Acute respiratory failure is a common problem seen among ICU patients with a mortality rate as high as 45%.[11] The incidence of adult primary respiratory failure in the United States accounts for 137 hospitalizations per 100,000 residents annually.[12] In India, the overall prevalence of chronic cough, chronic phlegm, and dyspnea was 2.0%, 1.2%, and 3.4%, respectively, and for chronic obstructive pulmonary disease has a reported prevalence ranging from 1.4% to 9.4% in males and 1.3–4.9% in females.[13]

Proper ABG interpretation allows for the assessment of oxygenation, acid-base status, whether or not it is respiratory, metabolic or mixed, and the degree of compensation.[6] Interpreting ABGs are a crucial skill for physicians, nurses, respiratory therapists, and other health-care personnel to prevent complications such as electrolyte imbalance, muscle weakness, narcosis, respiratory failure, organ failure, seizures, chronic kidney disease, arrhythmias, coma, shock, and death.[14]

Electrolyte disorders are among the most common clinical problems encountered in ICU patients. Conditions such as severe burns, trauma, sepsis, brain injury, and heart failure lead to disturbances in fluid and electrolyte homeostasis. There are numerous potential causes of fluid and electrolyte imbalances in critically ill patients: Renal hypoperfusion leading to pre-renal azotemia or tubular necrosis activation of hormonal systems such as renin-angiotensin-aldosterone system and vasopressin (ADH), third spacing of fluid or insensible fluid loss, and medication such as cyclosporine and aminoglycoside antibiotics. In addition, inappropriate administration of fluid and electrolytes should be considered in the diagnosis and treatment of fluid and electrolyte disturbances.[15]

Limited data exist on the prevalence of these disturbances in critically ill patients. The presence of these disorders may not only signal severe underlying pathophysiology but could also be a significant marker of adverse outcomes. It is currently not established whether electrolyte and acid-base disturbances are independent contributors to mortality in ICU patients. The purpose of this study is to assess acid-base and electrolyte disturbances among ICU patients and their relation to outcomes, specifically survival.

Aims and objective

The primary objective of this study was to assess electrolyte and acid-base imbalances commonly seen in ICU patients and to assess survival outcome of electrolyte imbalance associated with low Glasgow Coma Scale (GCS) patients in the medical ICU.

Materials and Methods

The study was conducted in the Department of Biochemistry in collaboration with the Department of Anaesthesia ICU, Govind Ballabh Pant Institute of Postgraduate Medical Education and Research, Delhi, India. Thirty critically ill patients in the ICU were enrolled in the study with informed consent. The study is approved with Institutional Ethics Committee as F.1/IEC/MAMC/65/No-372, December 2018. ABG analysis along with other relevant investigations was done within the first 24 h of ICU admission. The hospitalization details and progress of the patients were collected from the in-hospital records. Patients were followed-up until the end points, that is, discharge by the treating physician, discharge against medical advice, or in-hospital death.

The arterial whole blood sample was taken heparinized syringe and transported in ice packs for ABG analysis within 15 min of collection as per protocol. The sample was immediately analyzed on NOVA Stat Profile Prime Plus Critical Care Analyzer in the laboratory for pH, partial pressure of dissolved oxygen in blood (pO2), partial pressure of carbon dioxide in blood (pCO2), and bicarbonates. The results obtained were tabulated and recorded accordingly.

The normal ranges for the estimated ABG parameters are given below.[16]

1. Blood pH=7.35–7.45
2. Partial pressure of O_2 (pO2)=75–100mm Hg
3. Partial pressure of CO_2 (pCO2)=35–45mm Hg
4. Bicarbonate ions (HCO_3-)=22–26mmol/L.

The measurement of electrolytes – sodium, potassium, and chloride was done on NOVA Stat Profile Prime Plus Critical Care Analyzer. The reference range for sodium, potassium, and chloride is 135–145 mEq/L, 3.5–5.5 mEq/L, and 96–106 mEq/L, respectively.[17] Blood glucose levels were estimated by fully automated GOD POD method at the time of admission of patients to the ICU.

Data were presented as percentage (%) for frequencies and mean and standard deviation for continuous variables. Independent Student's t-test was done for comparison of continuous variables using SPSS 13 software.

Results

Mean age, HR, and SBP were comparable in both discharged patients and expired patients. Expired patients (192.13 ± 124.65 mg/dL) had mean serum glucose levels at the time of hospital admission were higher than those who got discharged (142.27 ± 27.51 mg/dL) after hospital stay, although this did not reach statistical significance ($P = 0.125$) [Figure 1]. This indicated that mortality rate becomes higher as glucose levels increase in critically ill patients. Creatinine levels were comparable in both discharged patients and expired patients. Hyponatremia was present in ICU patients who got discharged (130.95 ± 11.62 mEq/L) and those who expired (133.31 ± 7.71 mEq/L) during hospital stay. Mean serum potassium levels and chloride levels were normal among discharged and expired patients. Hypocalcemia was present in critically ill patients those got discharged (1.10 ± 0.19 mmol/L) after improvement as well as those who got expired (1.02 ± 0.15 mmol/L).

Out of 30 patients admitted in ICU, 8 patient's expired (26.66%) and 22 (73.33%) patients were discharged in stable conditions.

Out of 30 patients, 14 (46.67%) patients were normal without any respiratory disturbance and 16 (53.33%) patients developed respiratory complications; 3 (10%) had metabolic alkalosis, 7 (23.33%) had metabolic acidosis, 4 (13.33%) had respiratory alkalosis, and 2 (6.66%) suffered from respiratory acidosis. There was no statistically significant difference in pH between those who were discharged and those who expired ($P = 0.88$).

Out of 22 patients who got discharged from ICU, 12 (54.54%) had hyponatremia, 11 (50%) had hypokalemia. and 5 (22.7%) had hypochloremia. Out of eight patients who expired during hospital stay, 3 (37.5%) had hyponatremia, 4 (50%) had hypokalemia, and also 1 (12.5%) had hypochloremia.

Discussion

Respiratory disturbances are common among ICU patients as shown in our study. Out of 30 critically ill patients admitted to our ICU, 16 (53.33%) presented with respiratory disturbances. Out of the 16 patients with respiratory disturbance, 3 (10%) had metabolic alkalosis, 7 (23.33%) had metabolic acidosis, 4 (13.33%) had respiratory alkalosis, and 2 (6.66%) suffered from respiratory acidosis [Table 1].

Electrolyte and acid-base disturbances present differently among ICU patients depending on various underlying disease states. Table 2 shows broad categorization of critically ill patients according to different disease states. Baseline characteristics of critically ill patients (discharged and expired) are shown in Table 3. Out of 30 patients admitted in ICU, 8 patient's expired (26.66%) and 22 (73.33%) patients were discharged in stable conditions [Figure 2].

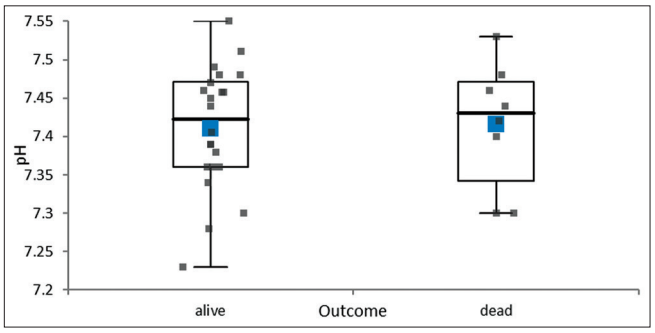

Figure 1: pH levels in discharged and expired patients ($P = 0.88$)

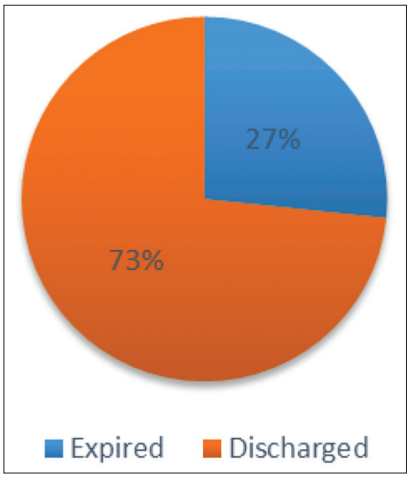

Figure 2: Outcome of critically ill patients

In the critical cardiac unit, the incidence of hyponatremia but not hypernatremia was higher in non-survivors than survivors (61.5% vs. 24.8%, respectively, $P = 0.000$). Hyponatremia in these patients may be a marker of neurohormonal activation that reflects the severity of heart failure (HF),[18] but it may occur following HF therapy.[19] Diuretics are one of the most common causes of drugs induced hyponatremia.[20] Respiratory acidosis is not the only acid-base disturbance observed in patients with acute and chronic respiratory failure. Both metabolic acidosis and metabolic alkalosis can coexist with respiratory acidosis. Heart failure, acute pulmonary edema, renal failure, and the onset of sepsis or severe hypoxia are the most common causes of metabolic acidosis associated with hypercapnia.[21] Metabolic alkalosis may also be the consequence of a too high minute ventilation in patients undergoing mechanical ventilation.[22]

Our results showed that of the 22 patients who got discharged from ICU, 12 (54.54%) had hyponatremia, 11 (50%) had hypokalemia, and 5 (22.7%) had hypochloremia. Out of the 8 patients who expired, 3 (37.5%) had hyponatremia, 4 (50%) had hypokalemia, and 1 (12.5%) had hypochloremia [Table 4, Figure 3].

Electrolytes such as sodium (Na), potassium (K), and chloride (Cl) are critical in allowing cells to generate energy, maintain the stability of their walls, and allow the body to work. Studies had reported that electrolyte imbalances are associated with increased morbidity and mortality in critically ill patients. Development of hyponatremia in ICU patients is associated with disturbances in the renal mechanism of urinary dilution and can lead to cellular dehydration and central nervous system damage. Serious disorders such as burns, trauma, sepsis, acute liver disease, heart failure, and CNS damage along with a myriad of medications used in the ICUs can lead to disturbances in fluid and electrolyte homeostasis.[17]

Table 1: Acid-base disturbances among critically ill patients

Respiratory disturbance	*n*=No. of cases	Percentage
Metabolic alkalosis	3	10
Metabolic acidosis	7	23.33
Respiratory alkalosis	4	13.33
Respiratory acidosis	2	6.66

Table 2: Broad categorization of critically ill patients

Disorders	*n*=No of patients/30 (%)
Gastrointestinal disorders (e.g., upper GI bleed, acute necrotizing pancreatitis)	13 (43.33)
Carcinoma (Ca esophagus, periampullary carcinoma)	07 (23.3)
Cardiac disorders (e.g., left MCA)	05 (16.66)
Miscellaneous (e.g., sepsis)	05 (16.66)

Hyponatremia can result from abnormal vasopressin production (SIADH), loop diuretics, thiazides, osmotic diuretics, and tubular damage that reduce Na and Cl in diluted part of urine. Signs and symptoms of hyponatremia are seen mainly when Na levels are rapidly reduced to less than 125 mEq/L.[23] Seizures and coma caused by cerebral edema are seen when Na is less than 110 mEq/L. Hyponatremia symptoms may not be seen in ventilated patients and, therefore, may exacerbate cerebral edema and lead to significant sequel such as herniation if not carefully monitored.[24]

Our results were similar to a study by Padhi *et al*.[25] who reported that the frequency of hyponatremia in ICU admission was 34.3%. Friedman *et al*. estimated the prevalence of hyponatremia in the ICU as high as 30%–40%.[26] Another study done by Laczi *et al*. found mild-to-moderate hyponatremia and severe hyponatremia in 15–30% and 1–4% of hospitalized patients, respectively.[17]

Hypokalemia can occur because of decreased potassium intake or inadequate replacement, medications such as sympathomimetics, insulin, dobutamine, diuretics, amphotericin B, and non-absorbed anions (penicillin and aminoglycosides). Signs and symptoms of hypokalemia are more neuromuscular, including paralysis, weakness, constipation, weakness of the respiratory muscles, and rhabdomyolysis. Cardiac arrhythmia, especially in patients with hypertension and heart disease, can also be seen.[27]

In ICU patients, hypokalemia can be a result of intracellular shifts, increased losses and decreased ingestion, or administration of potassium. Serum potassium levels do not correlate well with intracellular potassium levels and may not correlate with total body potassium. Therefore, hypokalemia may not imply a depletion of body potassium stores. Critically ill patients may have underlying conditions or receive medications that can cause hypokalemia. Metabolic alkalosis causes an intracellular shift of potassium; however, the total body potassium does level will not change. Correction of the underlying metabolic alkalosis should result in redistribution of potassium into the extracellular space and correction of hypokalemia.[28]

The balance of chloride is closely regulated by the body. Significant increases or decreases in chloride can have deleterious or even fatal consequences: Chloride is also involved in regulating blood pressure. Hypochloremia in critically ill patients can be caused by active Cl− loss, for example, through the GI tract (e.g., vomiting and diarrhea), through inadequate renal Cl− reabsorption, or through dilution following infusion of hypotonic fluids.[29,30] In addition, Cl− can be lost through the kidneys in cases of increased bicarbonate reabsorption in either chronic respiratory acidosis or hyperaldosteronism. High-volume bicarbonate infusion may result in Cl− being exchanged for bicarbonate to maintain electroneutrality. Studies have reported an incidence of hypochloremia between 6.7% and 37% among ICU patients.[31-34]

Table 3: Baseline characteristics of critically ill patients (discharged and expired)

Parameters	Critically ill patients (*n*=30)	Discharged (*n*=22)	Expired (*n*=8)
Age (years)	49.73±16.57	48.95±14.6	51.88±22.15
HR (min)	93.00±15.07	93.50±15.19	91.63±15.67
SBP (mmHg)	114.03±11.82	115.14±10.47	111.00±15.33
pH	7.41±0.08	7.41±0.08	7.42±0.08
PCo2	33.61±8.91	33.23±6.25	34.66±14.49
T Co2	22.96±5.18	22.75±4.97	23.55±6.06
PaO2	106.11±44.06	101.30±39.11	119.34±56.38
Glucose (mg/dL)	155.57±78.79	142.27±27.51	192.13±124.65
Creatinine (mg/dL)	0.84±0.23	0.82±0.21	0.88±0.28
HCT	30.43±6.94	29.90±7.80	31.88±3.72
WBC	9250.0±4569.45	9777.27±4999.23	7800.0±2872.03
Platelet	2.69±1.75	2.97±1.92	1.95±0.86
Na (mEq/L)	131.58±10.64	130.95±11.62	133.31±7.71
K (mEq/L)	3.57±0.86	3.49±0.90	3.80±0.74
Cl (mEq/L)	101.79±9.83	100.34±8.14	105.77±13.29

Table 4: Mean electrolyte levels in discharged and expired patients

Patients	Hyponatremia	*n*=No. of patients	Hypokalemia	*n*=No. of patients (%)	Hypochloremia	*n*=No. of patients (%)
Discharged (*n*=22)	124.45±11.83	12 (54.54%)	2.89±0.37	11 (50)	90.98±6.04	5 (22.7)
Expired (*n*=8)	125.10±4.12	3 (37.5%)	3.24±0.07	4 (50)	84.4±0.0	1 (12.5)

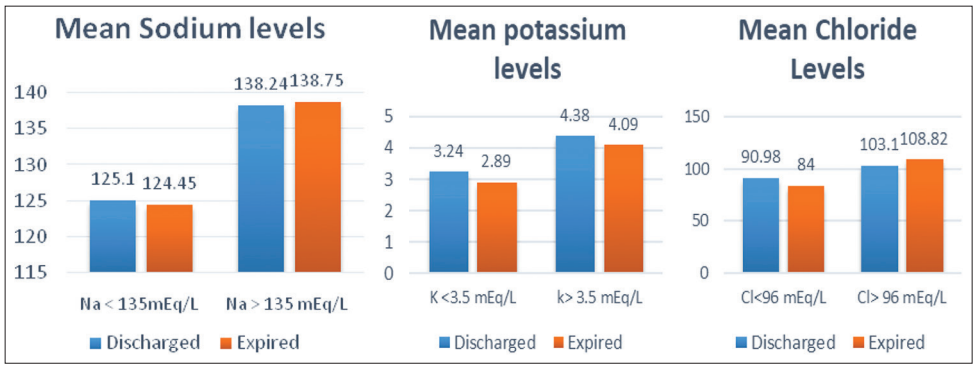

Figure 3: Mean sodium, potassium, and chloride levels in discharged and expired patients

Expired patients (192.13 ± 124.65 mg/dL) had elevated mean serum glucose levels at the time of hospital admission than those who got discharged (142.27 ± 27.51 mg/dL) after hospital stay, although this did not reach statistical significance (P = 0.125) [Figure 4]. This indicated that mortality rate becomes higher as glucose levels increases in critically ill patients. Hyperglycemia in these critical patients may associated with adverse outcome as reported by Van Den Berghe *et al*.[35]

There was no statistically significant difference in pH between those who were discharged and those who expired (p=0.88) [Figure 1].

Out of 30 patients, according to GCS, 08 (26.67%) had mild, 15 (50.0%) had moderate, and 07 (23.33%) had severe critical illness [Table 5]. Mean electrolyte levels were shown according to GCS in Table 6.

The GCS is a 15-point clinical scoring system for assessing a patients' level of conscious. Causes for altered consciousness which causes low GCS include intracranial causes such as head injuries, tumors, hemorrhages, seizures, and degenerative conditions. Metabolic causes (acid-base and fluid/electrolyte imbalances) are one of the extracranial causes that can lead altered consciousness in a patient. Other extracranial causes include hypoxia, sedative drugs, cardiovascular instability, sepsis, shock, trauma; liver, renal and endocrine dysfunction, and multiorgan dysfunction. The GCS grades

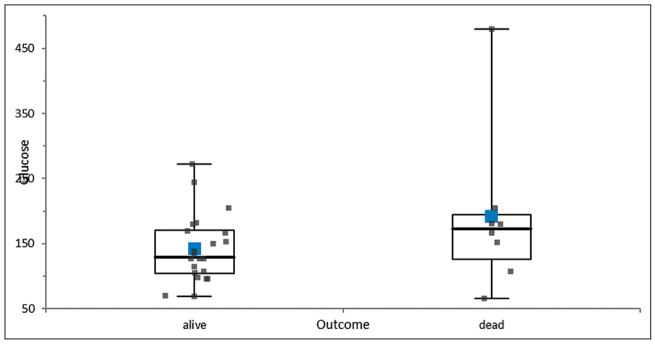

Figure 4: Glucose levels in discharged and expired patients (P = 0.125)

Table 5: Percentage distribution of critically ill patients according to Glasgow Coma Scale

Glasgow Coma Scale	*n*=No. of patients (percentage)
13–15 (Mild)	08 (26.67)
9–12 (Moderate)	15 (50.0)
3–8 (Severe)	07 (23.33)

Table 6: Mean electrolyte levels according to Glasgow Coma Scale

Glasgow Coma Scale	Sodium (mEq/L)	Potassium (mEq/L)	Chloride (mEq/L)
13–15 (Mild)	127.49±14.83	3.21±0.56	98.13±5.41
9–12 (Moderate)	134.41±9.51	3.74±1.03	102.16±9.39
3–8 (Severe)	130.20±5.95	3.63±0.72	105.20±13.98

severity of coma according to three categories of responsiveness: Eye opening, motor, and verbal responses.[36]

Sodium is involved in the transmission of nerve impulses and muscle contraction. Hyponatremia is an under-documented, inaccurately investigated, and suboptimally managed electrolyte disorder often leading to poor patient outcomes.[37] Critical care patients being treated for hyponatremia require diligent monitoring. Depending on the severity of the condition and the selected treatments, serum Na levels should be measured every 1–2 h during initial treatment and then at least every 4 h until signs and symptoms resolve.[38] Therapy must then be dictated not only by recognized reversible causes, such as advanced hypothyroidism, adrenal insufficiency, diuretics, or other medicines, but also by whether the hyponatremia occurred acutely or chronically and the degree of symptoms related to it.[39]

Rafat *et al*. showed that hyponatremia is a very common electrolyte derangement seen in the setting of the ICU.[40] Usually, sodium level correlates directly with the sensorium. As the serum sodium level falls, consciousness becomes progressively impaired reflecting by low GCS scores.[17]

In cancer patients, hyponatremia is a serious comorbidity that needs special care as its treatment varies by pathophysiologic groups, and its consequences can have a deleterious effect on the patient's health.[41] Critically, most evidence suggests that overenthusiastic treatment of SIADH is considerably more dangerous than treatment that is slow or relatively ineffective. It is vital that caregivers avoid too rapid normalization of serum sodium except in the most extreme circumstances.[37] Whether hyponatremia in a patient with cancer is just an indicator of poor prognosis or whether its presence may alter the patient's quality of life has not been definitively proven, but increasing evidence shows that hyponatremia must not be taken lightly.[42]

Chloride levels were normal in our study irrespective of low GCS in critically ill patients [Table 6]. This may be because of the small sample size of our study.

Studies show that electrolyte abnormalities were the most frequent occurrence (59% of patients) of low GCS but did not alter outcome.[36] Potassium ions play a major role in regulating fluid balance in cells, the transmission of nerve impulses, and in muscle contractions.[43] A symptom of hypokalemia is confusion or disorientation which, in turn, is related to a low GCS. Pomernaz *et al*. concluded that hypokalemia is the result of a large catecholamine discharge that is known to accompany severe head trauma, with resultant β2 adrenergic stimulation of the Na+-K+ pump, which reflects poor survival outcome in critically ill patients.[44]

Conclusion

The present study concluded that hyponatremia is the most common electrolyte disorder seen among critically ill patients that alter consciousness, which becomes progressively impaired, reflecting in a low GCS score. Hyponatremia and acidosis were highly associated with ICU mortality; these disturbances should be monitored closely,

diagnosed early, and managed correctly during hospitalization. Substantial additional work is still required to determine the true occurrence of hyponatremia in the various clinical settings.

Acknowledgments

Authors are thankful to *G. B. Pant* Institute of Postgraduate Medical Education and Research and NOVA Biomedical for their help to conduct the study.

Authorship Contributions

PKD and AMC – study conceptualization and design, supervision, manuscript editing, and approval; KS, ND, JB, and AS – data acquisition and analysis, manuscript drafting, and review; CAD and KCJ – statistical analysis and manuscript preparation; all authors have read and approve the final version of the manuscript.

References

1. Hu J, Wang Y, Chen R, Zhang X, Lin J, Teng J, *et al*. Electrolyte and acid-base disturbances in critically ill patients: A retrospective and propensity-matched study. Int J Clin Exp Med 2017;10:992-1003.
2. Spatenkova V, Bradac O, Skrabalek P. The impact of a standardized sodium protocol on incidence and outcome of dysnatremias in neurocritical care. J Neurol Surg Cent Eur Neurosurg 2015;76:279-90.
3. Kristof RA, Rother M, Neuloh G, Klingmuller D. Incidence, clinical manifestations, and course of water and electrolyte metabolism disturbances following transsphenoidal pituitary adenoma surgery: A prospective observational study. J Neurosurg 2009;111:555-62.
4. Bockenkamp B, Vyas H. Understanding and managing acute fluid and electrolyte disturbances. Curr Pediatr 2003;13:520-8.
5. Balci AK, Koksal O, Kose A, Armagan E, Ozdemir F, Inal T, *et al*. General characteristics of patients with electrolyte imbalance admitted to emergency department. World J Emerg Med 2013;4:113-6.
6. Kaur A, Charan GS. A study to assess the effectiveness of STP on knowledge and practice regarding ABGs among ICU nurses in selected hospitals at Jalandhar, Punjab. IJHSR 2018;8:182-8.
7. Lewis SM, Lewis SL, Heitkemper MM, Dirksen SR, O'Brien PG, Bucher L. Medical Surgical Nursing. Assessment and Management of Clinical Problems. 7th ed. Netherlands: Elseveir Publisher; 2010.
8. Smeltzer SC, Bare BG, Hinkle JL, Cheever KH. Brunner and Suddarth's Textbook of Medical Surgical Nursing. 10th ed. Philadelphia USA: Lippimkot Williams and Wilkins; 2010.
9. Barthwal MS. Analysis of arterial blood gases a comprehensive approach. JAPI 2004;52:573-7.
10. Mohammed HM, Abdelatief DA. Easy blood gas analysis: Implications for nursing. Egypt J Chest Dis Tuberc 2016;65:369-76.
11. Urden LD, Stacy KM, Lough ME. Priorities in Critical Care Nursing. Netherlands: Elsevier Health Sciences; 2015.
12. Nelmes P. Advanced critical care nursing--American association of critical-care nurses. Nurs Stand 2009;23:30-1.
13. Chhabra P, Sharma G, Kannan AT. Prevalence of respiratory disease and associated factors in an urban area of Delhi. Indian J Community Med 2008;33:229-32.
14. Mathew R, Hemavati G, Pillai S, Biswal A. A study to assess the effectiveness of structured teaching programme regarding arterial blood gas analysis and interpretation in terms of knowledge among nurses working in ICU in selected hospitals of Indore. IJNER 2014;2:286-89.
15. Lee JW. Fluid and electrolyte disturbances in critically ill patients. Electrolyte Blood Press 2010;8:72-81.
16. Clementine YF, Tar Choon AW. Arterial blood gases. Proc Singapore Healthc 2011;20:227-35.
17. Upadhyay S, Bhalerao N, Pratinidhi SA. Study of level of consciousness and electrolyte abnormalities in patients admitted to intensive care unit (ICU). Int J Contemp Med Res 2017;4:1739-42.
18. De Luca L, Klein L, Udelson JE, Orlandi C, Sardella G, Fedele F, *et al*. Hyponatremia in patients with heart failure. Am J Cardiol 2005;96:19-23.
19. Oren RM. Hyponatremia in congestive heart failure. Am J Cardiol 2005;95:2B-7.
20. Costache II, Alexandrescu DM, Cimpoesu D, Petris OR, Petris AO. Hyponatremia--risk factor in patients with chronic heart failure--clinical, evolutive and therapeutic implications. Rev Med Chir Soc Med Nat Iasi 2014;118:315-9.
21. Bruno CM, Valenti M. Acid-base disorders in patients with chronic obstructive pulmonary disease: A pathophysiological review. J Biomed Biotechnol 2012;2012:915150.
22. Tisdall M, Crocker M, Watkiss J, Smith M. Disturbances of sodium in critically ill adult neurologic patients: A clinical review. J Neurosurg Anesthesiol 2006;18:57-63.
23. Anderson RJ, Chung HM, Kluge R, Schrier RW. Hyponatremia: A prospective analysis of its epidemiology and the pathogenetic role of vasopressin. Ann Intern Med 1985;102:164-8.
24. Bennani SL, Abouqal R, Zeggwagh AA, Madani N, Abidi K, Zekraoui A, *et al*. Incidence, causes and prognostic factors of hyponatremia in intensive care. Rev Med Interne 2003;24:224-9.
25. Padhi R, Panda BN, Jagati S, Patra SC. Hyponatremias in critically ill patients. Indian J Crit Care Med 2014;18:83-7.
26. Friedman B, Cirulli J. Hyponatremia in critical care patients frequency, outcome, characteristics and treatment with the vasopressin V^2 receptor antagonist tolvaptan. J Crit Care 2013;28:219.
27. Buckley MS, Leblanc JM, Cawley MJ. Electrolyte disturbances associated with commonly prescribed medications in the intensive care unit. Crit Care Med 2010;38:S253-64.
28. Kraft MD, Btaiche IF, Sacks GS, Kudsk AK. Treatment of electrolyte disorders in adult patients in the intensive care unit. Clin Rev Am J Health Syst Pharm 2005;62:1663-82.
29. Berend K, van Hulsteijn LH, Gans RO. Chloride: The queen of electrolytes? Eur J Intern 2012;23:203-11.
30. Yunos NM, Bellomo R, Story D, Kellum J. Bench-to-bedside review: Chloride in critical illness. Crit Care 2010;14:226.
31. Shao M, Li G, Sarvottam K, Wang S, Thongprayoon C, Dong Y, *et al*. Dyschloremia is a risk factor for the development of acute kidney injury in critically ill patients. PLoS One 2016;11:e0160322.
32. Kimura S, Matsumoto S, Muto N, Yamanoi T, Higashi T, Nakamura K, *et al*. Association of serum chloride concentration with outcomes in postoperative critically ill patients: A retrospective observational study. J Intensive Care 2014;2:39.
33. Tani M, Morimatsu H, Takatsu F, Morita K. The incidence and prognostic value of hypochloremia in critically ill patients. ScientificWorldJournal 2012;2012:474185.
34. Van Regenmortel N, Verbrugghe W, Van den Wyngaert T, Jorens PG. Impact of chloride and strong ion difference on ICU and hospital mortality in a mixed intensive care population. Ann Intensive Care 2016;6:91.
35. Van den Berghe G, Wilmer A, Hermans G, Meersseman W, Wouters PJ, Milants I, *et al*. Intensive insulin therapy in the medical ICU. N Engl J Med 2006;354:449-61.
36. Buhary BM, Alrajhi SM, Abukhater M, Kyadudyn AS, Ali AF, Rahman AK, *et al*. Acid base electrolyte imbalance and survival outcome of low Glasgow coma scale (GCS) patients in the medical intensive care unit. Ann Med Health Sci Res

2017;7:10-5.

37. Grant P, Ayuk J, Bouloux PM, Cohen M, Cranston I, Murray RD, *et al*. The diagnosis and management of inpatient hyponatraemia and SIADH. Eur J Clin Investig 2015;45:888-94.
38. Decaux G, Soupart A. Treatment of symptomatic hyponatremia. Am J Med Sci 2003;326:25-30.
39. Verbalis JG, Goldsmith SR, Greenberg A, Korzelius C, Schrier RW, Sterns RH, *et al*. Diagnosis, evaluation, and treatment of hyponatremia: Expert panel recommendations. Am J Med 2013;126:S1-42.
40. Rafat C, Flamant M, Gaudry S, VidlaPetiot E, Ricard CD, Dreyfuss D. Hyponatremia in the intensive care unit: How to avoid a zugzwang situation? Ann Intensive Care 2015;5:39.
41. Onitilo AA, Kio E, Doi SA. Tumor-related hyponatremia. Clin Med Res 2007;5:228-37.
42. Schrier RW, Sharma S, Shchekochikhin D. Hyponatraemia: More than just a marker of disease severity? Nat Rev Nephrol 2013;9:37-50.
43. Halperin ML, Kamel KS. Potassium. Lancet 1998;352:220-8.
44. Pomeranz SH, Constantini SH, Rappaport ZH. Hypokalaemia in severe head trauma. Acta Neurochir 1989;97:62-6.