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Original Research

ASSESSMENT OF ELECTROLYTE IMBALANCE AMONG HYPOTENSIVE PATIENTS

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***ABSTRACT*:**
Background: Fluid and electrolyte disorders are considered one of the most common clinical problems observed in any clinical setup. Critical situations such as severe burns, trauma, sepsis, brain damage, and heart failure might cause disturbances in fluid and electrolyte homeostasis. The most common mechanisms include decreased perfusion to the kidney due to hypovolemia or hypotension. Thus, leading to activation of hormonal systems such as renin-angiotensin-aldosterone system and vasopressin; and tubular damage observed due to ischemic or nephrotoxic kidney damage, including renal insult caused by a myriad of medications used in the intensive care. **Material and method**: A study analysis was conducted in the ICU department of our medical college and hospital to evaluate the electrolyte imbalance in hypotensive patients. All patients selected were adults. A total of 55 patients were analysed and evaluated according to the electrolytic variation in the blood over a period of 10 months. **Results**: Out of the 55 patients, 37 were male and 18 were female. 21 patients were suffering from road side injury (38%), 8 were suffering from serious burns. Out of 29 patients of the traumatic category chances of hyponatremia and hypomagnesemia were high, i.e in 27 cases. In non-traumatic category out of 26 patients, majority had hyponatremia, hypokalemia **Conclusion**: Fluid and electrolyte abnormalities in critically ill patients might cause fatal consequences. More caution to electrolyte disturbances should be practiced in intensive care because it is nearly impossible to adequately assess symptoms and signs of critically ill patients. So as to provide optimal management, clinicians should be through with fluid and electrolyte homeostasis and the underlying pathophysiology of the respective disorders.

Keywords: Electrolyte, Fluid, homeostasis

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INTRODUCTION

Fluid and electrolyte disorders are considered one of the most common clinical problems observed in any clinical setup. Critical situations such as severe burns, trauma, sepsis, brain damage, and heart failure might cause disturbances in fluid and electrolyte homeostasis. The most common mechanisms include decreased perfusion to the kidney due to hypovolemia or hypotension. Thus, leading to activation of hormonal systems such as renin-angiotensin-aldosterone system and vasopressin; and tubular damage observed due to ischemic or nephrotoxic kidney damage, including renal insult caused by a myriad of medications used in the intensive care. Also, inappropriate administration of fluid and electrolytes should be considered in the diagnosis and treatment of fluid and electrolyte disturbances. Patients suffering from hypovolemia need an early goal-directed therapy, which is brought in action during the initial phase of management of patients with severe sepsis or septic shock, has been shown to improve overall survival chances of the patient. [(1)] Studies are suggestive that, patients with acute lung or kidney injury have reported that fluid overload has been associated with negative results. [(2-4)] Although, any uniform definitions of fluid overload and well-designed randomized clinical trials are absent. There seems to be a need to avoid over-use of fluid resuscitation in a subset of patients. [(5)] Novel techniques with invasive monitoring of extracellular fluid volume have been proposed long time back, but none of the technique have been actively validated in clinical care. [(6)] The present study was conducted with the aim to determine the electrolyte imbalance amongst hypotensive subjects.

MATERIAL AND METHOD

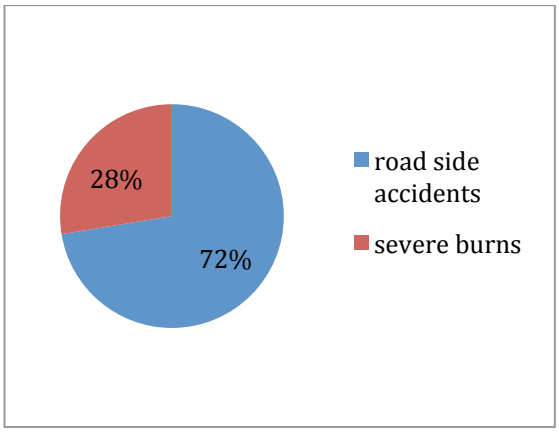
A study analysis was conducted in the ICU department of our medical college and hospital to evaluate the electrolyte imbalance in hypotensive patients. All patients selected were adults. A total of 55 patients were analysed and evaluated according to the electrolytic variation in the blood over a period of 10 months. This study was pre-approved by the ethics committee of our college. An

informed consent was obtained from the patients or the guardian in case the patient was unable to do so. All 55 patients were categorised according to the hypo-natremia, hyper-natremia, hypo-kalemia, hyper-kalemia, hypo-phosphatemia, hypo-calcemia and hypo-magnesemia. The patients were broadly classified as traumatic case and non-traumatic cases. For every patient blood serum analysis was done so as to analyse electrolyte levels once every 15 days for the 3 months. All the data was arranged in a tabulated form and analysed statistically. SPSS software was used for statistical analysis.

RESULTS

Out of the 55 patients, 37 were male and 18 were female. 21 patients were suffering from road side injury (38%), 8 were suffering from serious burns (15%). Both these types of patients were classified under the class of trauma (53%). (graph1) Under non traumatic category, 18 patients were suffering from severe renal disorder (33%), 6 patients were suffering from cardiac condition (11%) and 2 patients suffered from sepsis (4%), thus comprising a total of 47% of the total patients. (graph2) Out of 29 patients of the traumatic category chances of hyponatremia and hypomagnesemia were high, i.e in 27 cases. In non-traumatic category out of 26 patients, majority had hyponatremia, hypokalemia, and hypo phosphatemia. (table1).

Graph 1: Frequency of trauma cases



Graph 2: Incidence of non trauma cases

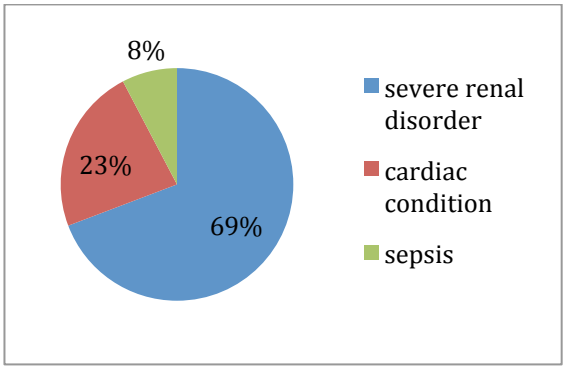


TABLE.1: The electrolyte status amongst the subjects

Category	***Traumatic***	***Non-Traumatic***
Condition		
Hypo-natrimia	HIGH	LOW
Hyper-natrimia	LOW	HIGH
Hyper-calcimia	HIGH	LOW
Hypo-calcimia	HIGH	LOW
Hyper-kalemia	LOW	HIGH
Hypo-kalemia	HIGH	LOW
Hypo-magnesia	HIGH	LOW

DISCUSSION

Patients in the ICU are always at a high risk of developing hypernatremia. Causative factors include the administration of sodium bicarbonate solutions to correct metabolic acidosis; renal water loss through a concentrating defects from renal disease. The use of diuretics or solute diuresis from glucose or urea and gastrointestinal fluid losses through nasogastric suction and lactulose administration, and water loss due to fever, various drainages, and open wounds also lead to fluid loss. Acute diabetes insipidus with hypernatremia can also complicate traumatic brain injury, occurring within 5-10 days after brain injury and disappearing within a few days to 1 month. [7] Moving on to hypokalemia, the major cause of hypokalemia include low dietary potassium intake, shift into the intracellular compartment, extrarenal potassium loss, and renal potassium loss. Medications most commonly prescribed in the ICU are associated with hypokalemia. [8] Sympathomimetics, insulin, methylxanthines, and dobutamine drive extracellular potassium into cells by stimulating Na^+,K^+-ATPase. Considering factors of hyperkalemia, the renal failure, adrenal insufficiency, insulin deficiency and resistance, and tissue damage from rhabdomyolysis, burns, or even trauma are among the most common factors for hyperkalemia in critically ill patients. A lot of medications used in the ICU might also cause hyperkalemia, including beta-blockers, inhibitors of renin-angiotensin-aldosterone system, potassium-sparing diuretics, heparin and its derivatives, trimethoprim, and non-steroidal anti-inflammatory drugs. Critical illness and anatomical or functional degeneration of muscle will result in (A) increased distribution of acetylcholine receptors throughout the muscle membrane beyond the neuromuscular junction, (B) altered subunit composition of acetylcholine receptors, and (C) the expression of a newly identified class of acetylcholine receptors which have longer duration of potassium efflux into the extracellular fluid. [9] Sodium bicarbonate should be avoided at all times in patients with extracellular fluid volume overload and is never the first choice in the treatment of hyperkalemia, as data on the usefulness of sodium bicarbonate are equivocal. [10] The condition of hypophosphatemia has been linked with critical conditions such as Gram-negative sepsis and open heart surgery. [11-13]The prevalence of hypophosphatemia is high in the ICU. [14] Hypophosphatemia (plasma phosphate concentration < 2.5 mg/dL or 0.81 mmol/L) may be due to decreased intestinal phosphate absorption, increased renal phosphate losses, and a shift of phosphate to intracellular

space. Hypocalcemia happens to be one of the most frequent electrolyte abnormalities encountered in the ICU. Low total concentrations of calcium have been reported to be affecting as many as 90% of critically ill patients, and the prevalence of hypocalcemia measured as ionized calcium is estimated to be about 15-20% [15] Hypocalcemia is associated with increased mortality in patients cared in the ICU, especially women. [16]The dilution of plasma induced by administration of massive amounts of intravenous fluid in a resuscitative effort is observed as an important cause of hypocalcemia in trauma patients. [17] Hypomagnesemia is frequently observed in critically ill patients, and its prevalence in the ICU is reported to be as high as 50%. [18] Severe hypomagnesemia can result in electrocardiographic changes, arrhythmias including torsades de pointes, seizures, coma, and death. Hypomagnesemia is associated with concomitant electrolyte disturbances such as hypokalemia and hypocalcemia. Considering causes of hypomagnesemia which include excess gastrointestinal or renal losses, surgery, trauma, infection or sepsis, burns, transfusion of blood products preserved with citrate, alcoholism, starvation or malnutrition, and certain medications. Fractional excretion of magnesium is a helpful index for differential diagnosis of hypomagnesemia also. [19]

CONCLUSION

Fluid and electrolyte abnormalities in critically ill patients might cause fatal consequences. More caution to electrolyte disturbances should be practiced in intensive care because it is nearly impossible to adequately assess symptoms and signs of critically ill patients. So as to provide optimal management, clinicians should be through with fluid and electrolyte homeostasis and the underlying pathophysiology of the respective disorders.

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