Fluid and Electrolyte Disturbances in Critically Ill Patients

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Disturbances in fluid and electrolytes are among the most common clinical problems encountered in the intensive care unit (ICU). Recent studies have reported that fluid and electrolyte imbalances are associated with increased morbidity and mortality among critically ill patients. To provide optimal care, health care providers should be familiar with the principles and practice of fluid and electrolyte physiology and pathophysiology. Fluid resuscitation should be aimed at restoration of normal hemodynamics and tissue perfusion. Early goal-directed therapy has been shown to be effective in patients with severe sepsis or septic shock. On the other hand, liberal fluid administration is associated with adverse outcomes such as prolonged stay in the ICU, higher cost of care, and increased mortality. Development of hyponatremia in critically ill patients is associated with disturbances in the renal mechanism of urinary dilution. Removal of nonosmotic stimuli for vasopressin secretion, judicious use of hypertonic saline, and close monitoring of plasma and urine electrolytes are essential components of therapy. Hypernatremia is associated with cellular dehydration and central nervous system damage. Water deficit should be corrected with hypotonic fluid, and ongoing water loss should be taken into account. Cardiac manifestations should be identified and treated before initiating stepwise diagnostic evaluation of dyskalemias. Divalent ion deficiencies such as hypocalcemia, hypomagnesemia and hypophosphatemia should be identified and corrected, since they are associated with increased adverse events among critically ill patients.

Key Words: intensive care; hyponatremia; hypernatremia; hypokalemia; hyperkalemia; hypocalcemia; hypophosphatemia

Introduction

Fluid and electrolyte disorders are among the most common clinical problems encountered in the setting of intensive care. Critical disorders such as severe burns, trauma, sepsis, brain damage, and heart failure lead to disturbances in fluid and electrolyte homeostasis. Possible mechanisms include reduced perfusion to the kidney due to hypovolemia or hypotension; activation of hormonal systems such as renin-angiotensin-aldosterone system and vasopressin; and tubular damage caused by ischemic or nephrotoxic kidney damage, including renal insult caused by a myriad of medications used in the intensive care. In addition, inappropriate administration of fluid...
and electrolytes should be considered in the diagnosis and treatment of fluid and electrolyte disturbances.

This article is intended to provide readers with relevant information on fluid and electrolyte problems frequently found in the intensive care unit (ICU), as well as on medications associated with fluid and electrolyte disorders.

**Fluid management**

Volume resuscitation of a patient with hypovolemic shock or sepsis is an essential component of patient care. Massive amounts of intravenous fluid are usually administered to replace intravascular volume deficit and to minimize complications attributed to hypovolemia such as tachycardia, hypotension, acute kidney injury, and multiorgan failure. Goal-directed therapies focused on restoration of normal blood pressure and organ perfusion have been advocated in the management of critically ill patients. Early goal-directed therapy, which is instituted in the initial phase of management of patients with severe sepsis or septic shock, has been shown to improve overall survival\(^1\). Clinicians should bear in mind that assessment of hemodynamic response to volume resuscitation and vasopressors should be based on specific hemodynamic and oxygenation parameters such as mean arterial pressure, central venous pressure, and central venous oxygen saturation, not solely on symptoms and physical findings.

In contrast to the notion of aggressive and liberal volume resuscitation, a growing body of evidence strongly suggests that fluid overload may be detrimental to critically ill patients. Relatively little attention has been paid to the consequences of fluid overload such as respiratory failure, increased cardiac demand, and peripheral edema. Recent studies on patients with acute lung or kidney injury have reported that fluid overload has been associated with adverse outcomes\(^2-4\). Although uniform definitions of fluid overload and well-designed randomized clinical trials are lacking, there seems to be a need to avoid overzealous fluid resuscitation in a subset of patients\(^5\).

As a general rule, daily input and output of fluid should be closely monitored, and loss into “third spaces” should be taken into account. Vital signs, findings from physical examination, and chest radiographs are of great importance in assessing the volume status of the patient. Invasive monitoring of central venous pressure or pulmonary capillary wedge pressure may be useful. Novel techniques involving invasive monitoring of extracellular fluid volume have been proposed, but none of them have been rigorously validated in clinical care\(^6\).

**Hyponatremia**

Disturbances in plasma sodium concentrations are a common clinical problem in patients admitted to the intensive care unit. Many cases of dysnatremia are acquired after a patient is admitted to the ICU, and the presence of dysnatremia is associated with poor prognosis. A recent study involving 151,486 adult patients from 77 intensive care units over a period of 10 years has demonstrated that many cases of dysnatremia are acquired in the intensive care unit, and that the severity of dysnatremia is associated with poor outcome in a graded fashion. Another study on the ICU patients with dysnatremias corroborated these findings, reporting that ICU-acquired hyponatremia and ICU-acquired hypernatremia were associated with increased mortality\(^7\).

Low plasma \([\text{Na}^+]\) represents a relative water excess in conjunction with impaired ability of the kidney to excrete electrolyte-free water. Removal of excess water by the kidney requires urinary dilution, which is compromised in virtually all patients in the ICU: (1) Heart failure, sepsis, shock, and multiple organ dysfunction syndrome impair glomerular filtration and enhance sodium and water reabsorption at the proximal tubule, thereby diminishing delivery of the filtrate to the diluting segment, i.e., the thick ascending limb of the loop of Henle and the distal convoluted tubule; (2) loop diuretics, thiazides, osmotic diuretics, and tubulointerstitial pathology reduce the reabsorption of sodium and chloride in the diluting segment; (3) and nonosmotic stimuli for vasopressin