

Hyponatremia in Patients with Acute Decompensated Heart Failure – Marker of a Poor Prognosis

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Hyponatremia (defined as a serum Na < 135 mEq/L) is the most common electrolytic disorder in hospitalized patients, which poses diagnostic and therapeutic challenges. Hyponatremia, either at admission or hospital-acquired, is associated with increased duration of hospitalization, increased risk of adverse events and high risk of mortality. Patients who present to the emergency departments with acute decompensated heart failure are often hyponatremic. The question of hyponatremia in patients with acute decompensated heart failure is a biomarker of advanced and severe disease or a therapeutic target has not been completely answered yet by the medical science. In the OPTIMIZE-HF registry, which included almost 50,000 patients with acute decompensated heart failure, 20% of patients presented hyponatremia at admission [1]. In acute heart failure, hyponatremia is rather dilutional than depletional. The extensive use of diuretic agents in patients with heart failure makes the differentiation between dilutional and depletional hyponatremia very difficult in these patients. In acute heart failure, hyponatremia is more likely to appear due to impaired water excretion and not to Na⁺ depletion. In patients with acute decompensated heart failure, several studies have demonstrated that arginine vasopressin level is elevated, thus promoting reabsorption of excessive free water, with subsequent dilutional hyponatremia. Depletional hyponatremia is rather rare in acute decompensated heart failure, if the patients are not treated with diuretics. However, sodium restriction in patients with heart failure may lead to a negative Na⁺ balance and may increase the risk of hyponatremia. There are currently many opinions that this strict dietary sodium restriction increases the risk of hospitalizations in patients with heart failure and is associated with higher mortality rates, especially if these patients receive high doses of diuretic agents.

Regarding the treatment, differentiation of dilutional versus depletional hyponatremia is very important, because it may lead to different therapeutic approaches. If depletional hyponatremia is confirmed, treatment with sodium solutions must be initiated. In case of dilutional hyponatremia, free water excretion must be promoted. The first step in patients with hyponatremia is to evaluate the plasma osmolality, in order to exclude false hyponatremia, induced by high levels of triglycerides, cholesterol, immunoglobulins or monoclonal gammopathies, such as multiple myeloma, in which plasma osmolality is normal. In uncontrolled diabetic patients with high levels of seric glucose, hyponatremia with normal or even high plasma osmolality is encountered.

In depletional hyponatremia, administration of hypertonic saline solution should be started, with slowly correction of hyponatremia. In mild hyponatremia, serum Na⁺ should be corrected by a maximal rate of 5 mEq/L/day. If hyponatremia is severe (< 125 mEq/L) and the patient is symptomatic, the correction rate may be no more than 10 mEq/L/day. Too rapid correction of hyponatremia may lead to increased risk of central pontine myelinolysis.

In dilutional hypotonic hyponatremia, in order to restore a

normal level of serum sodium, free water excretion should be promoted. Further therapeutic efforts should be concentrated in preventing a positive free water balance in these patients. A study on 322 patients with acute decompensated heart failure and hyponatremia has demonstrated that long-term changes of serum sodium are predictors of mortality and the correction of hyponatremia is associated with better outcomes [2]. Other study has found that normalization of hyponatremia in heart failure patients before discharge was not associated with a better survival rate [3]. The results of these studies may be interpreted that only a durable correction of hyponatremia may lead to increased survival in patients with heart failure. To achieve this durable correction of hyponatremia, a long term optimal treatment should be applied.

The acute treatment of dilutional hyponatremia usually includes loop diuretics with or without hypertonic saline solution, acetazolamide in combination with loop diuretics and AVP antagonists. Arginine vasopressin (AVP) antagonists are the only therapeutic class that actively promotes free water excretion. In patients with acute decompensated heart failure and hyponatremia, three oral V2 receptor antagonists have been studied: tolvaptan, satavaptan and lixivaptan [4-7], together with an intravenous AVP antagonist, conivaptan [8]. For the long term management of dilutional hypotonic hyponatremia in patients with acute decompensated heart failure, all the guidelines recommend water restriction. A study has found that water restriction in patients with acute decompensated heart failure and mild hyponatremia is associated with improved quality of life (9). However, water restriction is not well tolerated by most of the patients and adherence to this recommendation may be a real problem. The efficacy of AVP antagonists for the long term treatment of hyponatremia in heart failure is not well known. Renin-angiotensin system blockers are effective in the treatment of dilutional hypotonic hyponatremia in patients with heart failure and may be used, if there are no contraindications, such as renal dysfunction.

In conclusion, in patients with acute decompensated heart failure, hyponatremia is a marker of bad prognosis and should be intensively and carefully addressed. The differentiation between dilutional and depletional hyponatremia is essential for the optimal management of these conditions.

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