Sodium Intake and Heart Failure

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Sodium is an essential mineral and nutrient used in dietary practices across the world and is important to maintain proper blood volume and blood pressure. A high sodium diet is associated with increased expression of β—myosin heavy chain, decreased expression of α/β—myosin heavy chain, increased myocyte enhancer factor 2/nuclear factor of activated T cell transcriptional activity, and increased salt-inducible kinase 1 expression, which leads to alteration in myocardial mechanical performance. A high sodium diet is also associated with alterations in various proteins responsible for calcium homeostasis and myocardial contractility. Excessive sodium intake is associated with the development of a variety of comorbidities including hypertension, chronic kidney disease, stroke, and cardiovascular diseases.

Keywords: sodium,salt,heart failure,ambulatory heart failure,epidemiological studies

1. Introduction

Salt is an ionic compound made up of cation and anion. Edible salt consists of 40% sodium and 60% chloride by weight. Salt was historically used as a preservative since bacteria cannot flourish in the presence of high salt concentrations. Human cells require approximately 0.5 g/day of sodium to maintain vital functions. Most food preservatives have high sodium content and are major causes of increased dietary intake of sodium. The average sodium intake in most Americans is 3.4 g/day or 1.5 teaspoons of salt, which is greater than the physiological requirement for the human body. High sodium or salt intake can lead to chronic comorbidities including hypertension, heart failure (HF), chronic kidney disease, stroke, cardiovascular diseases, and increase mortality. Hence, current guidelines recommend restricting sodium consumption to 2–3 g/day[1].

HF is a major burden of morbidity and mortality on the health care system and is classified into two major groups, heart failure with reduced ejection fraction (HFrEF) and heart failure with preserved ejection fraction (HFpEF). Treatment of HFrEF involves both pharmacologic and non-pharmacologic strategies, while mainly heart rate and blood pressure control strategies are used in HFpEF since multiple clinical trials have not shown significant benefits of pharmacologic therapy[2]. Sodium restriction has historically been taught in textbooks as a cornerstone of the management of HF patients. However, data on this management strategy are controversial. In addition, the adherence to following a low sodium diet is challenging, especially after a recent hospitalization, as shown by Riegel et al.[3]. Before we vigorously start educating HF patients to limit sodium intake in their diet, we need to understand the evidence behind such recommendations. In this paper, we review evidence relating sodium to HF, pathophysiological mechanisms of increased sodium intake, and the relation of sodium intake to HF outcomes.

2. Sodium Intake and Ambulatory Heart Failure

Low-sodium diet recommendations not only apply to hospitalized patients but also to ambulatory patients to prevent acute worsening of symptoms. However, the evidence behind these recommendations is not conclusive. Alvelos et al. reported that in patients with chronic HFrEF with Ejection Fraction (EF) ≤40%, sodium restriction was not associated with improvement in NYHA functional class during 15-day follow-up[4]. Colin-Ramirez et al. in 2004 showed that in patients with HFrEF or HFpEF, 2.0–2.4 g/d of sodium restriction was associated with an improvement in NYHA functional class and less reported signs of HF on 6-months follow up [5]. However, Colin-Ramirez et al. in 2015 showed no significant difference in NYHA functional class between the intervention group with sodium restriction of 1.5 g/d in patients and the control group of moderate sodium intake of 2.4 g/d in patients with HFrEF and HFpEF who are on optimal medical therapy during 6-months follow up[6]. In a study by Philipson et al., sodium and fluid restriction of 2.3 g/d and 1500 mL/d respectively were associated with lower NYHA functional class and symptoms of edema in patients with a history of HF in NYHA classes II and IV over a 12-week follow-up[7]. Hummel et al. reported that 30-day readmissions were lower in the group with sodium restriction of 1.5 g/d in patients with a history of hypertension and recent admission or acute decompensated HF who are followed by discharge into the community [8]. However, they reported that the Kansas City

Cardiomyopathy Questionnaire clinical summary score was not different between the two groups over 12 weeks of follow-up[8]. Amongst 123 ambulatory HFrEF patients from two outpatient HF clinics over a median follow-up of three years, higher sodium tertile was associated with a 39% increased risk for all-cause hospitalization and a 3.5-fold increase in risk for mortality[9]. A recent propensity-matched analysis from the HF Adherence and Retention Trial showed that sodium restriction to <2.5 g/d in NYHA class II/III HF patients is associated with a 72% higher risk of death or HF hospitalization compared to a higher sodium intake of >2.5 g/d, especially in patients not receiving therapy with renin–angiotensin antagonists with a hazard ratio of 5.23[10]. However, sodium intake was determined from a food-frequency questionnaire, which is subject to recall bias.

3. Serum Sodium Values and HF

Research has shown that low serum sodium value (hyponatremia) is seen in about 20% of hospitalized patients with acute HF[11]. Serum sodium concentration is closely regulated by water homeostasis, which in turn is regulated by thirst, arginine vasopressin, and kidney function[12]. Hyponatremia can be caused by excessive water retention from neurohormonal activation as well as by negative sodium balance from loop diuretics and with a low sodium intake diet. Serum sodium values can be used to prognosticate outcomes in both HFrEF and HFpEF. Low serum sodium is a risk factor for poor long-term outcomes in acute HF, regardless of ejection fraction[13]. The Organize Program to Initiate Lifesaving Treatment in Hospitalized Patients with Heart Failure registry (OPTIMIZE-HF) involving 48,612 patients recruited from 259 hospitals revealed that each 3 mmol/L drop in serum sodium values below 140 mmol/L in hospitalized patients is associated with a 19.5% increased risk of in-hospital mortality, 10% increased risk of mortality on follow-up, and 8% increase risk of death or rehospitalization on follow-up[11]. A meta-analysis of HF patients showed that low serum sodium values are associated with an increased risk of mortality[14]. We have previously shown in a national Veterans Affairs database study of 25,540 HFpEF patients that a J-shaped relationship is observed between serum sodium levels and a higher risk of number of days of HF hospitalizations and all-cause hospitalizations per year[15]. Such a relationship exists with baseline measurements of serum sodium levels at the time of diagnosis of HF as well as during longitudinal follow-up. Among 50,932 HFpEF patients with a median follow-up of 2.9 years, a J-shaped relationship was seen between serum sodium values and all-cause mortality, HF hospitalizations, and all-cause hospitalizations[16]. These data are further supported by the fact that the improvement of hyponatremia in HF patients is associated with long-term clinical outcomes.

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