Salt Restriction in Heart Failure: The Great Debate!!

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“Let there be work, bread, water, and salt for all.” —Nelson Mandela

Salt is an essential nutrient used in dietary practices across the globe and without which food, especially Indian, can be tasteless. Edible salt consists of 40% sodium and 60% chloride by weight. Physiologically approximately 0.5 gm/day of sodium is sufficient for human cells to meet their vital functions. The average Indian adult consumes around 11 gm of salt/day which is double the amount of salt recommended (5 gm/day) and is way greater than the physiological requirement for the human body.

High sodium is associated with an increased risk of cardiovascular comorbidities, especially hypertension and heart failure (HF). HF prevalence is increasing globally and in India, it is 1.2/1,000 population as per India UK-India Education and Research Initiative study. HF is classified into two major groups, HF with reduced ejection fraction (HFrEF) and HF with preserved ejection fraction (HFpEF).

The Study of Dietary Intervention Under 100 mmol in HF (SODIUM-HF)—a pilot trial1, was a randomized controlled trial (RCT) that compared a low sodium (1.5 gm/day) to a moderate (2.3 gm/day) sodium diet in HF patients. It concluded dietary restriction of sodium intake was feasible, and achievement of this low sodium goal was associated with lower brain natriuretic peptide (BNP) levels and improved quality of life in patients with HF. The Prevent Adverse Outcomes in HF by Limiting Sodium,2 another pilot trial, concluded that the quality of life improved among patients in the 1500 mg group but remained unchanged in the 3000 mg group, and there was no difference in the change in N-terminal-pro BNP levels. Another trial concluded that sodium restriction below 2 gm/day is not warranted in mild HF patients, whereas excessive sodium intake above 3 gm/day may be harmful in moderate to severe HF patients.3 Colin Ramirez et al. in 2004 showed that in HF patients (both HFrEF or HFpEF), 2.0–2.4 gm/day of sodium restriction was associated with an improvement in New York Heart Association (NYHA) class and fewer reported signs of HF on 6-month follow-up.4 There were other clinical trials supporting the same notion of dietary sodium restriction in patients with HF.5,6

Evidences favoring low sodium intake demonstrated higher sodium intake was associated with an increased risk of cardiovascular events and death compared with moderate sodium intake in HF populations. Sodium restriction is appropriate in patients with stage I (at risk for HF) and II (asymptomatic) HF due to its effect on lowering blood pressure, the incidence of hypertension, left ventricular hypertrophy, cardiovascular disease, and even the incidence of HF. However, there is insufficient evidence for such a recommendation for stages III (with prior or current symptoms) and stage IV (refractory) HF.

Now let us focus on studies indicating the negative impact of sodium restriction in ambulatory HF patients

Avelos et al. reported that in patients with chronic HFrEF, with ejection fraction <40%, sodium restriction was not associated with improvement in functional class during 15-day follow-up.7 In 2015 Colin-Ramirez et al. showed no significant difference in NYHA class between the intervention group with sodium restriction of 1.5 gm/day compared to group of moderate sodium intake of 2.4 gm/day in patients with HF (both HFrEF and HFpEF) who are Optimal Medical Treatment during 6-months follow-up.1 In a recently published international, open-label, randomized controlled SODIUM-HFrEF trial, 806 patients with HF (preserved or reduced left ventricular systolic function) were randomly assigned to a low sodium diet (<1500 mg/day) vs usual care. It concluded that, among ambulatory patients with HF, a low sodium diet was not associated with a reduction in adverse cardiovascular events. A low sodium diet was associated with a modest improvement in quality of life; however, the 6-minute walk test was not different between treatment groups. Unfortunately, the trial was terminated early.

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Due to the coronavirus disease 2019 pandemic, limiting the interpretation of the findings. An earlier study by Doukky et al., in 2016 (adherence and retention trial) found that sodium restriction is not only disadvantageous but also has a rather detrimental impact on outcomes in symptomatic patients with chronic HF. They studied 902 chronic HF patients (both preserved or reduced left ventricular ejection fraction), which found no demonstrable evidence that dietary sodium restriction is associated with lower rate of death or HF hospitalization. They showed that sodium restriction to <2.5 gm/day 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 gm/day, especially in patients not receiving therapy with RAAS blockers with a hazard ratio of 5.23.

Now We Discuss Role of Sodium Restriction in Acute Decompensated HF (ADHF)

Aliti et al., studied the role of sodium restriction, which showed no effect on weight loss or clinical stability at 3 days and they concluded that sodium restriction in patients admitted for ADHF is unnecessary. In a large Italian study of patients admitted with HF, patients assigned to low sodium intake (1.84 gm/day) compared to moderate sodium intake (2.76 gm/day), had reduced diuresis, more HF readmissions, and a trend towards increased mortality. Unfortunately, patients in this study did not receive optimal neurohormonal blockade and received strict fluid restriction of 1 L/day and high dose diuretic (up to 100–1000 mg of furosemide) without adjustment of clinical status. In a meta-analysis of 17 RCTs, sodium restriction was not associated reduction in CV or all cause mortality or hospitalizations in patients with HF.

Role of Sodium Restriction in HFrEF

Prevalence of HF is increasing globally, and HF with HFrEF has gradually accounted for almost half of the HF population with near similar mortality rates. Although some observational studies and RCTs have focused on sodium intake in patients with HF, patients with HFrEF were frequently excluded from these studies. Moreover, patients with HFrEF have a different response to treatment and volume status than those with HFrEF. As salt intake could significantly affect volume status and neurohormonal status, it might play a role in response to treatment in HFrEF. However, contrary to the theoretical expectation, a recent study done in patients with HFrEF revealed that an overstrict dietary salt intake restriction could harm patients with HFrEF and is associated with a worse prognosis. A study done by Machado et al., showed that aggressive salt/water restriction does not provide clinical benefits in patients with HFrEF. Also, sodium restriction does not seem to have a neurohormonal effect in patients with HFrEF.

There are many potential reasons for conflicting evidence regarding the benefit/harm of sodium restriction. These include heterogeneity of the HF patient population studied, lack of uniformity in limiting the amount of sodium restriction per day, unclear data on the associated use of fluid restriction, and simultaneous usage of diuretics and neurohormonal blockade agents.

What Should We Do?

Given there is clear evidence of the benefit of limiting sodium intake to prevent various comorbidities leading to HF, it is recommended to limit sodium intake in those who are at risk (American College of Cardiology stages A and B) to prevent the onset of HF. In patients with HF, salt restriction is a double-edged sword, and the benefits and harm have to be balanced cautiously. Current data suggest a moderate salt restriction in ambulatory patients with HFrEF. Stringent salt restriction (I < 1.5 gm a day) is associated with worse outcomes in both ambulatory and decompensated patients with HFrEF. However, HFrEF data suggest that salt restriction does not confer any significant benefits. As the degree of dietary sodium reduction that would lead to a reduction in clinical events has not yet been defined, clinicians and patients should consider this dietary intervention similar to other medical therapies and balance the potential benefits on an individual basis.

References