Review Article

Is Salt Restriction Really Necessary in Patients with Acute Decompensated Heart Failure?

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Introduction

Heart failure (HF) syndrome was first described as an emerging epidemic almost 30 years ago. Due to population growth and aging, the total number of patients with HF has increased over the past years. Acute decompensated HF (ADHF) is estimated to be the leading cause of hospitalization, based on data available from 50% of the South American population.[1]

The standard treatment for patients with ADHF includes diuretics, vasodilators, and sodium restriction.

The recommendation of sodium restriction has been historically and heuristically based on sodium-related fluid retention and, in a patient with congestive HF, one would expectedly avoid anything that promotes fluid retention.

Therefore, sodium restriction has undoubtedly been the most frequently recommended self-care strategy for patients with ADHF.[2]

However, with the contemporary understanding of the pathophysiology of HF, in which treatments involve inhibition or activation of neuroendocrine systems, sodium restriction could have a negative effect on patients due to the activation of the renin-angiotensin-aldosterone system (RAAS), heart rate increase, and reduction in energy and protein intake, leading to cachexia.

This review aims to analyze the existing literature including both randomized and nonrandomized clinical trials on the effects of sodium restriction in ADHF.

Why may sodium restriction not be beneficial?

We, *homo sapiens*, have been "designed" to retain sodium. Our kidneys have long serpentines, the loop of Henle, which has a complex and efficient mechanism of sodium retention. And thanks to this sodium retention capacity, particularly considering the mammals in the scale of evolution, we, human beings can live out of the water. Amphibians, for example, are not completely terrestrial as they have kidneys with a smaller loop, and hence a lower sodium retention capacity.[3]

Our sodium retention capacity is also due to the evolutionary development of the AARS. This system is particularly activated in situations that cause renal hypoperfusion, including dehydration, bleeding, burns, and as we know, HF. Today, thanks to four decades of advances in the understanding of HF, it is known that interventions that inhibit the RAAS are proven to be beneficial. These include angiotensin-converting-enzyme inhibitors (ACEI),[4] angiotensin receptor blockers (ARB),[5] aldosterone inhibitors (spironolactone and eplerenone),[6] betablockers (indirectly),[7] and angiotensin receptor-neprilysin inhibitor (ARNi).[8]

On the other hand, situations that activate the RAAS are proven to be harmful to HF patients. These include severe hypovolemia, hypotension, erythropoietin,[9] adrenaline (indirectly),[10] and furosemide.

In this balance between benefits and harms, we must consider that sodium restriction acts as an activating factor of the RAAS, whereas sodium administration acts as an inhibitor factor of the system.[11] Thus, in an era in which HF treatment is based on neurohormonal systems, interventions that activate the RAAS, such as sodium restriction, may be deleterious (Figure 1).

Analysis of the best available evidence

We selected studies that evaluated the effect of interventions for sodium intake (*e.g*, low sodium intake) for patients aged ≥ 18 years, admitted for treatment of ADHF.

Studies that evaluated sodium intake as a continuous exposure (i.e., not prescribed), studies conducted in the emergency department (without admission) and those without a control group were not included. We searched for peer-reviewed articles in Medline (via PubMed), Embase, ClinicalTrials.gov, CINAHL and Cochrane Database of Systematic Reviews until August 2023. Using these criteria, five clinical trials were analyzed and compared (Table 1).

Conclusions

To date, five studies (four clinical trials and one nonrandomized), with a total of 411 patients, compared a sodium-restricted diet with a high-sodium diet (unrestricted in four studies) in patients admitted with ADHF. Currently

Keywords

Salt Restriction; Heart Failure; Treatment; Salt; Randomized Clinical Trials.

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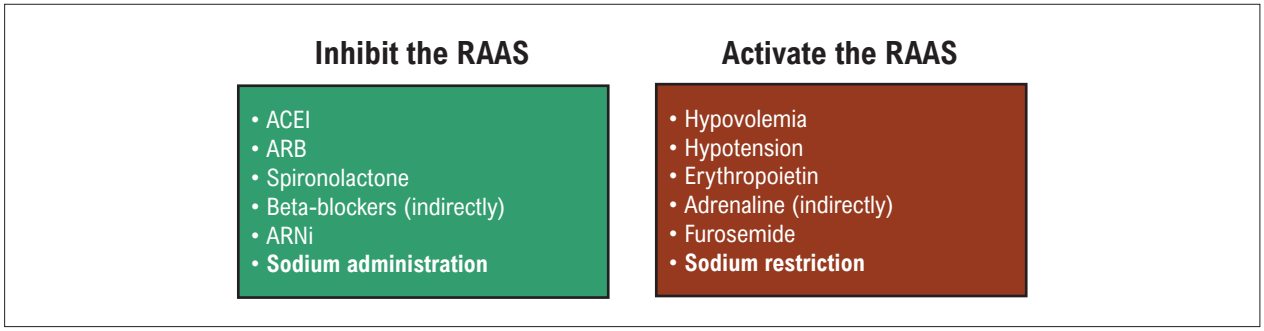

Figure 1 – *Factors that lead to inhibition or activation of the renin-angiotensin-aldosterone system (RAAS) in patients with acute decompensated heart failure. ACEI: angiotensin-converting-enzyme inhibitors; ARB: angiotensin receptor blockers; ARNi: angiotensin receptor-neprilysin inhibitor.*

Table 1 - Summary of the results of the mains studies on dietary sodium restriction in patients with acute heart failure available until August 2023

	Velloso et al.[12]	Aliti et al.[13]	Inuzuka et al.[14]	Machado d'Almeida et al.[15]	Fabricio et al.[16]
Year	1991	2013	2016	2018	2019
Country	Brazil	Brazil	Japan	Brazil	Brazil
N	Intervention: 14 Control: 18	Intervention: 38 Control: 37	207	Intervention: 30 Control: 23	Intervention: 22 Control: 22
Randomized	Yes	Yes	No	Yes	Yes
Centers	Single-center	Single-center	Non-available	Single-center	Single-center
Inclusion criteria	NYHA: III - IV	Boston criteria ≥ 8, EF ≤ 45% Less than 36h of hospitalization for randomization Age ≥18 years	NYHA: II-IV, Median BNP = 856 pg/mL	EF ≥50%, Age ≥18 years; clinical signs of congestion, dyspnea, orthopnea in the last week, BNP > 100 pg/mL	Framingham criteria, Age ≥18 years
Mean age (years)	54	60	79	72	58
NYHA	III-IV	III-IV	II-IV	II-IV	Non-available
LVEF (mean)	34,8	26	Not available	61	29
Intervention	Sodium-restricted diet (2 g/day)	Sodium-restricted (0.8 g/day) and fluid-restricted (800 mL/day) diet	Sodium-restricted diet (6 g/day)	Sodium-restricted (0.8 g/day) and fluid-restricted diet (800 mL/day)	Sodium-restricted (3 g/day) and fluid-restricted (1000 mL/day)
Control	Norma-sodium diet (10 g/day)	Diet with no restrictions	Norma-sodium diet (10 g/day)	Diet with no restrictions	Norma-sodium (7 g/day), fluid-restricted diet (1000 mL/day)
Intervention duration	7,5 days (median) for the intervention group and 6.6 days(median) for the control group	6 days (median)	Not available	5 days (median)	7 days
Follow-up duration	Not available	30 days	Not available	7 days for the primary outcome and 30 days for the secondary outcomes	7 days
Primary outcome	Clinical improvement (recovery to NYHA I or II)	Weight loss and clinical stability in three days, thirst sensation, hospital readmission in 30 days	Energy intake	Weight loss	Difference in serum sodium
Difference in the primary outcome	No	Increase in thirst sensation	Low energy intake in the group with sodium restriction	Lower energy-protein intake in the group with sodium restriction	Yes

EF: ejection fraction; BNP: brain natriuretic peptide; LVEF: left ventricular ejection fraction

available evidence allows us to conclude that a sodium-restricted diet:

1. Did not have a significant impact on symptoms (clinical congestion score and HF symptoms), on the dose of diuretics prescribed during hospitalization, on the hospital length of stay, or on readmission within 30 days;

2. Did not have a significant impact on intermediate outcomes like serum creatinine, urea, brain natriuretic peptide, diuresis, serum aldosterone or plasma renin activity;

3. Caused a significant reduction in energy intake as compared to an unrestricted diet;

4. Did not provide enough data to draw conclusions about mortality.

Author Contributions

Conception and design of the research, Acquisition of data and Writing of the manuscript: Beck-da-Silva L; Analysis and interpretation of the data: Butzke M; Critical revision of the manuscript for important intellectual content: Beck-da-Silva L, Butzke M.

Potential conflict of interest

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Study association

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Ethics approval and consent to participate

This article does not contain any studies with human participants or animals performed by any of the authors.

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