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Review Article

Restricted Daily Sodium Intake as Heart Failure Management: A Systematic Review

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ABSTRACT

***Objective*:** To evaluate mortality and morbidity in HF patients receiving sodium restriction.

Study Design: Systematic Literature Review (SLR) of RCTs and Cohort studies worldwide published between Jan 2013 to Jan 2023.

***Methodology*:** We systematically selected RCTs and Cohort studies with sodium restriction as an intervention published within the last decade from five databases (PubMed, Scopus, Cochrane Library, Embase, and Medline). The full texts retrieved were assessed for risk of bias, and then a systematic review was performed. The endpoints analyzed were mortality and morbidity.

Result: Five RCTs and two cohort studies were included in this study, with 2,505 participants. The endpoints retrieved were mortality and morbidity (hospitalization, NYHA functional class, BNP or NT-proBNP level, quality of life, and congestion). No improvement was seen in mortality and hospitalization, but sodium restriction improved the rest of the morbidity indicators. Over-restricted and overconsumption of sodium intake might worsen HF presentation. When analyzed based on the HF groups, there was not enough evidence to recommend sodium dose based on ejection fraction; however, there was potentially more benefit for patients with higher NYHA classes. However, more evidence is still needed.

***Conclusion*:** Sodium restriction did not benefit mortality and hospitalization but improved quality of life, systolic blood pressure, and BNP or NT-pro BNP levels in all HF classes.

Keywords: Heart failure, Hypertension, Management, New York heart association (NYHA) functional class, Sodium restriction.

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INTRODUCTION

Heart failure (HF) is one of the leading health problems, affecting 26 million individuals globally.[1] In Southeast Asia, nine million people are estimated to have HF.[2] Baseline Health Research in Indonesia showed that cardiovascular disease in Indonesia reached 1.5% in prevalence, with HF still being the highest in number among adults; this is a concern because HF has the highest mortality risk in one year.[3] In 2030, an estimated eight million people will develop HF and increase prevalence by 46%.[4]

Standard HF drugs, including angiotensin-converting enzyme inhibitors, angiotensin receptor antagonists, β-adrenoceptor antagonists, and aldosterone receptor antagonists, are well-proven to improve the quality of life, symptoms, and longevity of HF patients.[5] However, lifestyle modification is still needed to support pharmacological treatments. Lifestyle changes help to relieve HF symptoms, improve day-to-day physical performance, and slow down the progression of the disease, especially in milder classes of HF.[6] physicians often recommend sodium restriction for dietary management, especially to correct congestion, a common sign and symptom in HF.[7]

However, there are still disputes around limiting sodium intake in HF patients due to the varying data: Guideline for the Management of Heart Failure 2013 from American College of Cardiology/American Heart Association recommended sodium restriction to reduce congestion[8] while Guidelines for the prevention, detection and management of heart failure in Australia 2018 mentioned that sodium restriction was not recommended because it led to worsening mortality as well as rehospitalization, and did not give significant difference in prognosis when compared to the non-restricted HF population.[7,9] The lack of consistency regarding the efficacy of sodium restriction in HF hence called for high-quality synthesis of trials and cohort studies to fill in the gap.

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METHODOLOGY

The systematic review was conducted according to the PRISMA (Preferred Reporting Items for Systematic Reviews and Meta-Analyses) Guidelines (Figure-1). This systematic literature review included RCTs and Cohort studies worldwide published between January 2013 to January 2023.

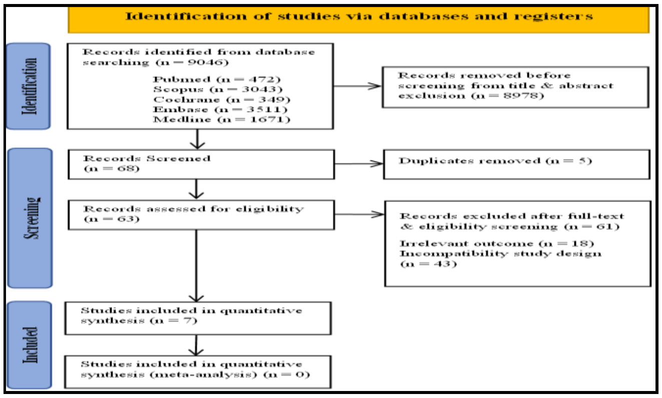

Figure-1: PRISMA Flowchart

LITERATURE SEARCH

The study was performed using a wide and comprehensive search through medical databases: PubMed, Scopus, Cochrane Library, Embase, and Medline. The keywords used were heart failure, sodium, restriction, and intake. We included randomized controlled trials (RCTs) and cohort studies to analyze later. Reference lists and articles cited in the studies were also reviewed thoroughly and added to our analysis if they met the criteria.

STUDY SELECTION

Inclusion criteria:

The studies included in this systematic review need to fit the inclusion criteria: 1) published between January 2013 and January 2023, 2) studying a population of stable chronic HF with standard care, 3) with the intervention of sodium restriction, and 4) with endpoints of mortality (all-cause or cardiovascular death) and morbidity (hospitalization, NYHA functional class, BNP or NT-proBNP level, quality of life, and congestion).

Exclusion criteria:

The exclusion criteria are 1) literature review studies, 2) studying hospitalized, unstable, or exacerbated HF patients, and 3) giving interventions outside of sodium restriction, fluid restriction, or diuretic drugs.

Data extraction and analysis

Authors analyzed each selected study and extracted the following data: 1) author, 2) publication year, 3) study characteristics (location/country, study design, sample size, length of follow-up period), 4) participant characteristics (type of heart failure), 5) intervention and control (daily sodium dose; fluid and diuretics dose if available), and 6) endpoints observed, which are mortality (all-cause or cardiovascular death), morbidity (hospitalization, NYHA classes, NT-proBNP level, quality of life, and congestion), and others (if available).

RESULTS

Search results

At the start of our search, we selected five electronic databases that included a total of 9,046 research articles, consisting of 472 articles from PubMed, 3,043 articles from Scopus, 349 articles from Cochrane Library, 3,511 articles from Embase, and 1,671 articles from Medline. Several 8,978 studies were removed from the title and abstract exclusion. Five duplicate records were obtained and were immediately excluded. Of the remaining 68 records assessed for eligibility, 61 were excluded during screening; these records consisted of 18 studies with irrelevant outcomes and 43 with incompatible study designs. Finally, seven studies met our inclusion and exclusion criteria, comprised of 5 RCT studies and two prospective cohort studies.

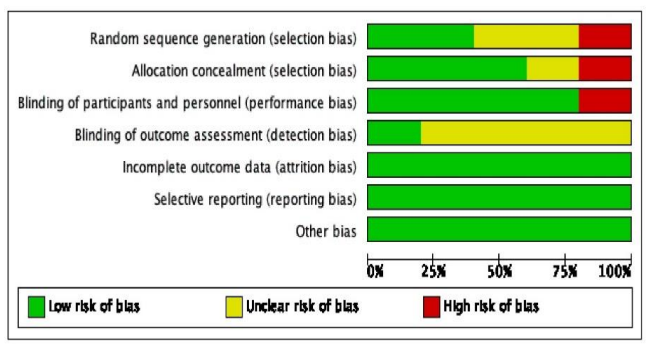

Figure-2: Risk of Bias Assessment Using Cochrane Risk of bias tool. Risk of bias graph showing review authors judgement about each risk of bias item presented as percentages across included studies

For quality assessment of the studies, we used Version 1 of the Cochrane risk-of-bias tool for randomized trials (RoB) and the Newcastle-Ottawa Scale for cohort studies. The scoring of the RoB tool was separated into individual elements. Each study was judged by "high", "low", or "unclear" risk of bias.[10] The scoring of the Newcastle-Ottawa Scale is based on three domains: selection, comparability and exposure.[11]

The quality of the studies, as shown in Figure-2 and Table-I, was then categorized according to the overall number of stars.

New York Heart Association (NYHA) functional classification to classify participants, while three other studies used ejection fraction (heart failure with reduced ejection fraction, HFrEF; heart failure with preserved ejection fraction, HFpEF).

Table-I: Risk of Bias Assessment using Cochrane Risk of Bias Tool for included Studies

Indicators	Collin Ramirez *et al*, 2015	Doukky *et al*, 2016	Ivey-Miranda *et al*, 2021	Li *et al*, 2022	Philipson *et al*, 2013
Random Sequence generation	Yellow	Red	Green	Yellow	Green
Allocation concealment	Green	Red	Green	Yellow	Green
Blinding of participants and personnel	Red	Green	Green	Green	Green
Blinding of outcome assessment	Green	Yellow	Yellow	Yellow	Yellow
Incomplete outcome data	Green	Green	Green	Green	Green
Selective reporting	Green	Green	Green	Green	Green
Other bias	Green	Green	Green	Green	Green

Red = High risk, Yellow = Unclear risk, Green = Low risk

The authors assessed the studies risk of bias collaboratively through group discussion. One study conducted by Doukky *et al*.[15] showed a high risk of bias for random sequence generation, and two studies by Colin-Ramirez *et al*.[16] and Li *et al*.[12] showed an unclear risk of bias for random sequence generation. Allocation concealment bias assessment showed one study with a high risk of bias by Doukky *et al*.[15] and one study with an unclear risk of bias by Li *et al*.[12] In accordance with performance bias, one out of five studies showed a high risk of bias, which was the study by Colin-Ramirez *et al*.[16] However, all five studies showed a low risk of bias for attrition bias, reporting bias, and other biases.

As shown in Table-II, for the included cohort studies, Kalogeropoulos *et al.* and Song *et al*. were categorized as good-quality studies with a score of 7 on the Newcastle-Ottawa Scale.

Table-II: Newcastle-Ottawa Scale for included Cohort Studies

Indicators	Selection	Comparability	Outcome	Overall
Kalogeropoulos *et al*, 2020	***	**	**	7/9
Song *et al*, 2013	***	*	***	7/9

The populations included in this study were from China, Mexico, the United States of America, Canada, South Korea, and Sweden, as shown in Table-III. The follow-up period ranged from three months to three years. A total of 2,505 patients were included in this analysis. All participants were adults with stable heart failure with standard therapy. Four studies used the

Intervention and Control Group Characteristic

The intervention given was sodium restriction; in one out of seven studies, the intervention included fluid restriction as well. Five studies compared two groups of intervention and control groups; one study compared three groups with high, moderate, and low sodium intake; and one study used cooking salt scores to measure daily salt intake in teaspoons.

Mortality

Mortality was observed in three studies, as shown in Table-IV. Doukky *et al*.[15] showed that, although non-significant, there was an increase in cardiac death with a hazard ratio [HR]: 1.62; 95% CI: 0.70 to 3.73; P=0.257 and all-cause mortality of P=0.074 in the sodium-restricted group. In accordance with that, one study by Song *et al*. (2014) showed a significantly lower death rate in the group with higher sodium intake in NYHA class I-II patients (HR=0.39, 95% CI 0.16 to 0.98, P=0.047). However, based on the same study by Song *et al*. (2014), it differed with the higher class of the NYHA (III-IV) population, which showed higher consumption of sodium had a 2.1 times risk of hospitalization (P=0.044).[17] While one study by Li *et al*.[12] signified that there was no significant increase in all-cause mortality (HR=0.838, 95% CI 0.684 to 1.027, P=0.088) or cardiovascular death (HR=0.782, 95% CI 0.598 to 1.020, P=0.071) in lower or higher sodium intake groups.

Morbidity

Hospitalization

Results varied for the hospitalization endpoint. Hospitalization was observed in five out of seven studies, signifying that it was the most observed endpoint. Three studies showed that the non-restricted sodium group posed a significantly lower risk for HF hospitalization, in research by Doukky *et al*.[15] (32.3% vs 20.0%; HR: 1.82; 95% CI: 1.11 to 2.96; *P*= 0.015); Li *et al*.[12], that showed that patients with cooking salt score >0 significantly had a lower number of HF hospitalization (HR=0.737, 95% CI 0.603 to 0.900, *P*=0.003); and Song *et al*. signified that NYHA I-II patients with lower sodium intake had lower HF

Table-III: Study Characteristics

No.	Study design	Daily sodium dose	Observed variables	Outcome
Based on ejection fraction				
1.	Li *et al*. (2022) (12) Location: China; Multicenter, international, randomized, double-blind, and placebo-controlled trial; 2.93-years follow-up; 1.713 samples (propensity-matching) with HFpEF.	Cooking salt score was used (with scoring, which was no table salt consumption= 0, 1/8 ts*P*= 1, 1/4 ts*P*= 2, ≥1/2 ts*P*= 3) with 1 tsp of table salt= 2235.5 mg sodium	Primary endpoint: composite of cardiovascular death, HF hospitalization, and cardiac arrest. Secondary endpoint: all-cause death, cardiovascular death, HF hospitalization.	Patients with the score of >0 had significantly lower primary endpoints and HF hospitalization, but there was no difference for cardiovascular death or all-cause death
2.	Ivey-Miranda *et al*. (2021) [13] Location: Mexico; Randomized, double-blind, parallel-group trial; 20 weeks follow-up; 70 samples with stable HFrEF.	Restricted (2 grams/day) vs non-restricted (3 grams/day)	NT-proBNP, quality of life, and blood pressure	There were no differences of NT-proBNP and quality of life between both groups; but it was found that consumption of <2.5 grams/day still improved NT-proBNP level and quality of life. Blood pressure was found lower in restricted patients (2 grams/day).
3.	Kalogeropoulos *et al*. (2020) [14] Location: United States of America; Prospective observational pilot study; 12-weeks follow-up; 83 samples with HFrEF.	Restricted (1.5 grams/day) vs non-restricted (3 grams/day)	Food satisfaction, adherence, quality of life, NT-proBNP, hospitalization, blood pressure, and serum creatinine	Both groups felt satisfied with their food, with consumption of either 1.5 grams or 3 grams per day. Quality of life improved for the restricted group (1.5 grams/day). Serum creatinine was found better in the restricted group (1.5 grams/day) and was elevated from the baseline in the non-restricted group (3 gram/day). Adherence, NT-proBNP, hospitalization rate, and blood pressure were not found different between both groups.
Based on NYHA functional class				
4.	Doukky *et al*. (2016) (15) Location: United States of America; Behavioral RCT, partially blinded, multicenter; 36 months follow-up; 260 samples (prospensity-matched) NYHA II-III heart failure with standard therapy.	Restricted (<2.5 grams/day) vs non-restricted (≥2.5 grams/day)	Death and HF hospitalization	Sodium restriction (<2.5 grams/day) significantly correlated with higher risk of death and HF hospitalization.
5.	Colin-Ramirez *et al*. (2015) (16) Location: Canada; RCT pilot, open-label, adjudicated end-point; 6-months follow up; 38 samples NYHA II-III heart failure with optimal therapy.	Low (1.5 grams/day) vs moderate (2.3 grams/day)	Quality of life and BNP level	There was an improvement of quality of life on both intervention groups; BNP level was lower on the low sodium intake group (1.5 grams/day). There were no differences on the NYHA class between both groups.
6.	Song *et al*. (2014) (17) Location: South Korea; Prospective observational study; 365-days follow up; 244 outpatient samples with NYHA I/II and NYHA III/IV heart failure.	<2 grams/day, 2-3 grams/day, or >3 grams/day	Hospitalization, death, and event-free survival.	In NYHA I/II patients, patients who consumed <2 grams/day had higher risk and patients who consumed >3 grams/day had lower risk for hospitalization and death compared to 2-3 grams/day. Meanwhile, for NYHA III/IV patients, consumption of >3 grams/day had shorter event-free survival; there was no differences between <2 grams/day and 2-3 grams/day.
7.	Philipson *et al*. (2013) (18) Location: Sweden; Prospective, randomized, multicenter intervention trial; 12-weeks follow up; 97 samples with NYHA II-IV stable heart failure with furosemide (>40 mg for NYHA III-IV or >80 mg for NIYHA II-IV).	The intervention group was given instructions on sodium diet of <5 grams/day; and fluid restriction (<1.5 L/day). The control group was not given restriction instruction.	Composite of NYHA class, hospitalization, peripheral edema, quality of life	The intervention group had better composite, which was mainly due to NYHA class improvement and leg edema; and there was no bad effect on thirst, hunger, or quality of life.

Table-IV: Outcome Variables Observed in the Studies

Sr	Studies	CVD death	All-cause mortality		Hospitali-zation	QoL	NYHA class	Peripheral edema	BNP or NT-proBNP	BP
1.	Li *et al*. (2022) [12]	✓	✓		✓					
2.	Ivey-Miranda *et al*. (2021) [13]					✓			✓	✓
3.	Kalogeropoulos *et al*. (2020) [14]				✓	✓			✓	✓
4.	Doukky *et al*. (2016) [15]	✓	✓		✓					
5.	Colin-Ramirez *et al*. (2015)[16]					✓✓	✓		✓	
6.	Song *et al*. (2014) [17]		✓*✓**		✓✓**					
7.	Philipson *et al*. (2013)[18]				✓***	✓✓	✓***	✓***		
	Number of studies	2	3		5	4	2	1	3	2

Notes: Favoring sodium restriction group (✓)
Favoring non-sodium restriction group (✓)
Not showing difference between sodium restriction and non-sodium restriction groups (✓)
*Notes (2): * = in HF NYHA I-II*
*** = in HF NYHA III-IV*
**** = composite endpoint*
Notes (3): CVD= cardiovascular risk; QoL= quality of life; NYHA class= New York Heart Association functional classification; BP= blood pressure; Cr= creatinine

hospitalization rate (HR=0.39, 95% CI 0.16 to 0.98, *P*=0.047). However, hospitalization was higher in the NYHA III-IV group with higher sodium intake intervention.[17]

However, the restricted group posed a better composite endpoint in 51% of patients, with hospitalization being one of the endpoints (*P*<0.001) in a study by Philipson *et al*. (2013)[18], while Kalogeropoulos *et al*.[14] showed that the number of hospitalizations between control and intervention group did not differ significantly.

Quality of life (QoL)

QoL was measured in four studies. Sodium restriction significantly improved the quality of life in the restricted group when measured with the Kansas City Cardiomyopathy Questionnaire (KCCQ) score in two studies by Kalogeropoulos *et al*. (*P*<0.001) and Colin-Ramirez *et al*. (*P*=0.006)[14,16]. A study by Ivey-Miranda *et al*. might exhibit no differences between the 2-gram sodium/day group vs 3 grams/day (*P*=0.04) when measured with The Minnesota Living with Heart Failure Questionnaire (MLHFQ), but the scores improved when the consumption was <2.5 gram/day[13] .The study conducted by Philipson *et al*. showed an improvement in QoL scores in both restricted and non-restricted groups when measured with the Life Satisfaction Questionnaire (LiSat-11)[18]. However, Colin-Ramirez *et al*. (2015) also showed that there was an improvement in QoL in the non-restricted group, although not significant (*P*=0.07)[16].

NT-proBNP or BNP Level

Two studies signified that there was no difference in NT-pro BNP level between groups, both in a study by Ivey-Miranda *et al*. (2021) (95% confidence interval −27 to −73%; *p*= 0.002) and Kalogeropoulos *et al*. (2020) (with *P*=0.70 and *P*=0.69 for between-groups change).[13,14] Colin-Ramirez *et al*. (2015) presented a differing result, with the low-sodium group showing a lowered BNP level (*P*=0.06).[16]

NYHA Class and Congestion

One study presented no significant NYHA difference between the intervention and control group (*P*=0.87).[16] But Philipson *et al*. (2013) showed improved NYHA class (*P*=0.01) and oedema (*P*=0.01) in the intervention group that received <5 grams of sodium/day and fluid intake no higher than 1.5 L/day.[18]

Other Endpoints

Other endpoints were observed in the seven studies included in this research. The first was blood pressure, which was used to measure the safety of the intervention. If the intervention of sodium restriction caused SBP to drop below 90 mmHg, the patients would be withdrawn. One study showed no significant SBP drop in the intervention (*P*=0.24) and control group (*P*=0.29).[14] Another study by Ivey-

Miranda *et al*. (2021) showed that blood pressure decreased significantly in the intervention group (*P*=0.05) but not any lower than <90 mmHg.[13] The second observed endpoint was creatinine level, which showed a decrease in the sodium-restricted group (*P*=0.001).[14]

DISCUSSION

Among the selected studies, the recommended dose of daily sodium consumption for HF patients is difficult to conclude due to the widely varying results. There are several reasons for the inconsistency of the result: 1) the diverse characteristics and comorbidities of the HF patients included in the studies; 2) the most used method to measure sodium intake was with diet recall, which was not a precise technique to estimate sodium consumption; 3) the lack of homogeneity of daily sodium consumption classification; and 4) not all studies considered to measure daily fluid intake and furosemide dose.

The variabilities in the results also derived from the paradox of limiting sodium consumption in HF patients. Sodium restriction initially aims to break the vicious cycle in HF that leads to excessive water retention, as shown in **Figure-3**. In contrast, the excessive water retention in HF that later increases circulating volume is a compensation mechanism for the low cardiac output (CO) in HF[22]. However, this mechanism, combined with maladaptive RAAS, commonly causes congestion in HF patients that worsens morbidity and mortality.[20,23,24]

The recommendation based on the Ejection Fraction:

Heart failure with reduced ejection fraction (HFrEF)

Two studies by Ivey-Miranda *et al*. (2021) and Kalogeropoulos *et al*. comprised of HFrEF patients. [13,14] Sodium restriction of 1.5 to 2 grams/day posed no harm and even showed a possible benefit for HFrEF patients, especially for: QoL improvement [13,14]; the reduction of NT-proBNP level, although insigni-ficant; and the decline of systolic blood pressure. [13] Patients also expressed satisfaction with consuming 1.5 grams of sodium/day. [14] In relevance to the systolic blood pressure finding, hypertension is one of the most common comorbidities in HF. Another perspective to look at sodium restriction in HF is aimed at relieving congestion and controlling blood pressure.

Sodium restriction may benefit because the serum sodium can draw and hold water intravascularly and further worsen the congestion, as shown in **Figure-3** [25,26]; hence, sodium restriction is one of the most common recommendations for HF patients. The argumentation behind the recommen-dation is that sodium restriction also acts as a diuretic, reducing intravascular volume by decreasing fluid retention in the kidney.[23]

Heart failure with Preserved Ejection Fraction (HFpEF)

One study by Li *et al*. (2022) comprised of HFpEF patients.[12] The study showed that overstrict sodium restriction (with a cooking salt score of 0) was proven unbeneficial by increasing the rate of HF hospitalization, although it did not significantly affect mortality.[12] This is because low sodium diet leads to lower filtrated sodium and higher uptake of sodium in proximal tubules that cause a reduction of the sodium amount going through macula densa, stimulating the release of renin and later activating the RAAS) [16] as shown in **Figure-3**. Hence, an over-strict sodium diet may trigger a counter-regulatory mechanism that decreases CO and worsens HF patients' hemodynamics. [7,20,24]

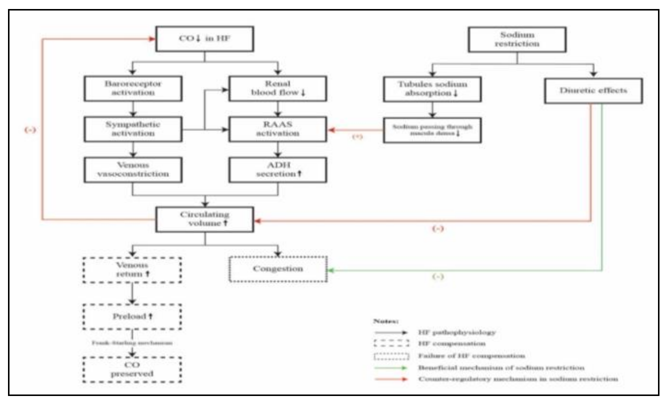

Figure-3: An overview of Pathomechanism between sodium restriction and Heart Failure

(a) HF pathophysiology. HF is a condition of reduced CO, which reduces intravascular volume, then triggers baroreceptor activation in left ventricle, aortic arch, and carotid sinus. This activates neurohormonal response (with sympathetic increase, parasympathetic reduction, and the release of ADH), inducing venous vasoconstriction. Reduced CO, coupled with vasoconstriction, reduces renal blood flow and triggers RAAS, which increases the release of ADH. (19–21) (b) HF compensation. All of these mechanisms increase circulating volume, improve venous return and preload, and eventually, the CO is preserved. HF is then compensated (22). (c) Failure of HF compensation. On the flip side, these mechanisms cause too much water retained in the body; coupled with maladaptive RAAS in HF, congestion occurs (23) (d) Beneficial mechanism of sodium restriction. Sodium restriction serves as diuretic effects, improving congestion in HF. (24) (e) Counter-regulatory mechanism in sodium restriction. The diuretic effects of sodium restriction decrease circulating volume, which later reduces CO, worsens the low CO in HF, and creates vicious cycle. (7,20,24)

The recommendation based on NYHA Functional Classification

Two studies observing mortality and HF hospitality classified the participants based on their

NYHA functional classification. One study with the NYHA II-III population signified that daily sodium consumption of <2.5 grams/day was associated with a higher risk of mortality or HF hospitalization.[15] In contrast to that, the NYHA II-III population in a study by Colin-Ramirez *et al*. (2015) had better QoL and BNP levels outcomes in the group with lower sodium consumption of 1.5 grams/day.[16] The varying results might be due to the need for studies to run subgroup analyses between each NYHA group.

One study by Song *et al*. (2014) showed that patients reacted differently to daily sodium doses based on their NYHA severity. NYHA I-II patients had better outcomes with higher sodium consumption; >3 grams of sodium/day lowered the risk of hospitalization and death, while in contrast, a dose of <2 grams of sodium/day caused a higher risk of hospitalization and death. On the contrary, NYHA III-IV patients with >3 grams of sodium/day had shorter event-free survival.[17] Hence, low sodium intake tends to trigger a counter-regulatory mechanism (**Figure-3**) in lower NYHA classes. Sodium restriction is better advised for higher NYHA classes patients, especially when their congestion does not improve significantly despite being given standard HF treatment.

However, Philipson *et al*. (2013) showed that >5 grams of sodium/day in NYHA II-IV patients showed worse composite outcomes when compared to <5 grams/day, be it the NYHA functional class, hospitalization, peripheral oedema, and QoL. Hence, daily sodium consumption in all NYHA classes should not exceed 5 grams/day.[18]

Our study showed that the recommendation of sodium daily dose could only be generalized among some HF patients since each HF group reacted differently to different sodium doses. Hence, physicians should tailor the sodium dose based on the HF type instead, whether based on the ejection fraction or the functional type.

Strengths and Limitations

This systematic review concluded the last decade's most recent trials and prospective cohorts. In comparison to other existing systematic reviews on the same subject, the authors discussed and analyzed endpoints based on 1) specific HF classification to reduce biased analysis due to the diverse characteristics of the HF patients included in this study and 2) sodium dosing instead of sodium grouping to limit the bias due to the inhomogeneity of the sodium restriction grouping.

However, the authors only concluded studies published in English, and the follow-up period between studies also varied widely (between three months and three years), which might contribute to endpoint bias.

CONCLUSION

Sodium restriction did not improve mortality and hospitalization in HF patients. However, it may benefit the quality of life (due to the relief of congestion and NYHA class improvement), systolic blood pressure, and BNP or NT-proBNP levels. Either very low (0 gram) or very high (>5 gram) daily sodium intake is not recommended for all HF types. The dose of daily sodium intake recommendation is still inconclusive, but the sodium restriction approach is more beneficial for HF with higher NYHA functional classes. More evidence is needed to recommend sodium dose for HF patients based on ejection fraction. For future research, we suggest running subgroup analyses so sodium recommendations can be given in an individualized manner based on HF characteristics.

Conflict of Interest: None.

Authors' Contribution

Following authors have made substantial contributions to the manuscript as under:

1,2: Conception, study design, drafting the manuscript, approval of the final version to be published.

3,4: Data acquisition, data analysis, data interpretation, critical review, approval of the final version to be published.

5,6: Data acquisition, drafting the manuscript, approval of the final version to be published.

Authors agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved.

REFERENCES

1. Savarese G, Lund LH. Global Public Health Burden of Heart Failure. Card Fail Rev 2017; 3(1): 7-11. https://doi.org/10.15420/cfr.2016:25:2
2. Lam CSP. Heart failure in Southeast Asia: facts and numbers. ESC Heart Fail 2015 ; 2(2): 46-49. https://doi.org/10.1002/ehf2.12036
3. Emmons-Bell S, Johnson C, Roth G. Prevalence, incidence and survival of heart failure: a systematic review. Heart 2022; 108(17): 1351-1360. https://doi.org/10.1136/heartjnl-2021-320131.
4. Benjamin EJ, Blaha MJ, Chiuve SE, Cushman M, Das SR, Deo R, et al. American Heart Association Statistics Committee and Stroke Statistics Subcommittee. Heart Disease and Stroke Statistics-2017 Update: A Report From the American Heart Association. Circulation 2017; 135(10): e146-e603. https://doi.org/10.1161/CIR.0000000000000485

5. Gupta D, Georgiopoulou VV, Kalogeropoulos AP, Dunbar SB, Reilly CM, Sands JM, et al. Dietary sodium intake in heart failure. Circulation 2012; 126(4): 479-85. https://doi.org/10.1161/CIRCULATIONAHA.111.062430
6. Aggarwal M, Bozkurt B, Panjrath G, Aggarwal B, Ostfeld RJ, Barnard ND, et al. American College of Cardiology's Nutrition and Lifestyle Committee of the Prevention of Cardiovascular Disease Council. Lifestyle Modifications for Preventing and Treating Heart Failure. J Am Coll Cardiol 2018; 72(19): 2391-2405. https://doi.org/10.1016/j.jacc.2018.08.2160
7. Patel Y, Joseph J. Sodium Intake and Heart Failure. Int J Mol Sci 2020; 21(24): 9474. https://doi.org/10.3390/ijms21249474
8. Yancy CW, Jessup M, Bozkurt B, Butler J, Casey DE Jr, Drazner MH, et al. American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines. 2013 ACCF/AHA guideline for the management of heart failure: a report of the American College of Cardiology Foundation/American Heart Association Task Force on practice guidelines. Circulation 2013; 128(16): e240-327. https://doi.org/10.1161/CIR.0b013e31829e8776
9. Machado d'Almeida KS, Rabelo-Silva ER, Souza GC, Trojahn MM, Santin Barilli SL, Aliti G, et al. Aggressive fluid and sodium restriction in decompensated heart failure with preserved ejection fraction: Results from a randomized clinical trial. Nutrition 2018; 54:111-117. https://doi.org/10.1016/j.nut.2018.02.007
10. Higgins JP, Altman DG, Gøtzsche PC, Jüni P, Moher D, Oxman AD; et al. Cochrane Bias Methods Group; Cochrane Statistical Methods Group. The Cochrane Collaboration's tool for assessing risk of bias in randomised trials. BMJ 2011; 343: d5928. https://doi.org/10.1136/bmj.d5928
11. Luchini C, Stubbs B, Solmi M, Veronese N. Assessing the quality of studies in meta-analyses: Advantages and limitations of the Newcastle Ottawa Scale. World J Meta-Anal2017; 5(4): 80-84. http://doi.org/10.13105/wjma.v5.i4.80
12. Li J, Zhen Z, Huang P, Dong YG, Liu C, Liang W. Salt restriction and risk of adverse outcomes in heart failure with preserved ejection fraction. Heart 2022;108(17):1377-1382. https://doi.org/10.1136/heartjnl-2022-321167
13. Ivey-Miranda JB, Almeida-Gutierrez E, Herrera-Saucedo R, Posada-Martinez EL, Chavez-Mendoza A, Mendoza-Zavala GH, et al. Sodium restriction in patients with chronic heart failure and reduced ejection fraction: A randomized controlled trial. Cardiol J 2023;30(3):411-421. https://doi.org/10.5603/CJ.a2021.0098
14. Kalogeropoulos A, Papadimitriou L, Georgiopoulou VV, Dunbar SB, Skopicki H, Butler J, et al. Low- Versus Moderate-Sodium Diet in Patients With Recent Hospitalization for Heart Failure: The PROHIBIT (Prevent Adverse Outcomes in Heart Failure by Limiting Sodium) Pilot Study. Circ Heart Fail 2020; 13(1): e006389. https://doi.org/10.1161/CIRCHEARTFAILURE.119.006389
15. Doukky R, Avery E, Mangla A, Collado FM, Ibrahim Z, Poulin MF, et al. Impact of Dietary Sodium Restriction on Heart Failure Outcomes. JACC Heart Fail 2016; 4(1): 24-35. https://doi.org/10.1016/j.jchf.2015.08.007
16. Colin-Ramirez E, McAlister FA, Zheng Y, Sharma S, Armstrong PW, Ezekowitz JA, et al. The long-term effects of dietary sodium restriction on clinical outcomes in patients with heart failure. The SODIUM-HF (Study of Dietary Intervention Under 100 mmol in Heart Failure): a pilot study. Am Heart J 2015; 169(2): 274-281.e1. https://doi.org/10.1016/j.ahj.2014.11.013
17. Song EK, Moser DK, Dunbar SB, Pressler SJ, Lennie TA. Dietary sodium restriction below 2 g per day predicted shorter event-free survival in patients with mild heart failure. Eur J Cardiovasc Nurs 2014; 13(6): 541-548. https://doi.org/10.1177/1474515113517574
18. Philipson H, Ekman I, Forslund HB, Swedberg K, Schaufelberger M. Salt and fluid restriction is effective in patients with chronic heart failure. Eur J Heart Fail 2013; 15(11): 1304-1310. https://doi.org/10.1093/eurjhf/hft097
19. Colin-Ramirez E, Arcand J, Ezekowitz JA. Dietary Self-management in Heart Failure: High Tech or High Touch? Curr Treat Options Cardiovasc Med 2017; 19(3): 19. https://doi.org/10.1007/s11936-017-0515-9
20. DiNicolantonio JJ, Chatterjee S, O'Keefe JH. Dietary Salt Restriction in Heart Failure: Where Is the Evidence? Prog Cardiovasc Dis 2016 ; 58(4): 401-406. https://doi.org/10.1016/j.pcad.2015.12.002
21. Konerman MC, Hummel SL. Sodium restriction in heart failure: benefit or harm? Curr Treat Options Cardiovasc Med 2014; 16(2): 286. https://doi.org/10.1007/s11936-013-0286-x
22. Yancy CW. The Uncertainty of Sodium Restriction in Heart Failure: We Can Do Better Than This. JACC Heart Fail 2016; 4(1): 39-41. https://doi.org/10.1016/j.jchf.2015.11.005
23. Reilly CM, Anderson KM, Baas L, Johnson E, Lennie TA, Lewis CM, et al. American Association of Heart Failure Nurses Best Practices paper: Literature synthesis and guideline review for dietary sodium restriction. Heart Lung 2015; 44(4): 289-298. https://doi.org/10.1016/j.hrtlng.2015.03.003
24. Khan MS, Jones DW, Butler J. Salt, No Salt, or Less Salt for Patients With Heart Failure? Am J Med 2020; 133(1): 32-38. https://doi.org/10.1016/j.amjmed.2019.07.034
25. Wang K, Chu C, Hu J, Wang Y, Zheng W, Lv Y, et al. Effect of Salt Intake on the Serum Cardiotrophin-1 Levels in Chinese Adults. Ann Nutr Metab 2018; 7 3(4): 302-309. https://doi.org/10.1159/000494436
26. Rakova N, Kitada K, Lerchl K, Dahlmann A, Birukov A, Daub S, et al. Increased salt consumption induces body water conservation and decreases fluid intake. J Clin Invest 2017; 127(5): 1932-1943. https://doi.org/10.1172/JCI88530

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