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Abstract

BACKGROUND: A low-sodium diet is a core component of heart failure self-care but patients have difficulty following the diet.

AIM: The aim of this study was to identify predictors of higher than recommended sodium excretion among patients with heart failure.

METHODS: The World Health Organization Five Dimensions of Adherence model was used to guide analysis of existing data collected from a prospective, longitudinal study of 280 community-dwelling adults with previously or currently symptomatic heart failure. Sodium excretion was measured objectively using 24-hour urine sodium measured at three time points over six months. A mixed effect logistic model identified predictors of higher than recommended sodium excretion.

RESULTS: The adjusted odds of higher sodium excretion were 2.90, (95% confidence interval (CI): 1.15-4.25, *p*p=0.007) for patients with diabetes; and 2.22 (95% CI: 1.09-4.53, *p*=0.028) for patients who were cognitively intact.

CONCLUSION: Three factors were associated with excess sodium excretion and two factors, obesity and diabetes, are modifiable by changing dietary food patterns.

Keywords

Aged, Cohort Studies, Confidence Intervals, Diabetes Mellitus, Type 2, Diet, Sodium-Restricted, Disease-Free Survival, Female, Heart Failure, Humans, Longitudinal Studies, Male, Middle Aged, Obesity, Odds Ratio, Patient Compliance, Patient Education as Topic, Predictive Value of Tests, Prognosis, Prospective Studies, Risk Assessment, Severity of Illness Index, Sodium, Dietary, Statistics as Topic, Survival Analysis, Urinalysis, World Health Organization

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Identifying predictors of high sodium excretion in patients with heart failure: A mixed effect analysis of longitudinal data

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Abstract

Background—A low-sodium diet is a core component of heart failure self-care but patients have difficulty following the diet.

Aim—The aim of this study was to identify predictors of higher than recommended sodium excretion among patients with heart failure.

Methods—The World Health Organization Five Dimensions of Adherence model was used to guide analysis of existing data collected from a prospective, longitudinal study of 280 community-dwelling adults with previously or currently symptomatic heart failure. Sodium excretion was measured objectively using 24-hour urine sodium measured at three time points over six months. A mixed effect logistic model identified predictors of higher than recommended sodium excretion.

Results—The adjusted odds of higher sodium excretion were 2.90, (95% confidence interval (CI): 1.15–4.25, $p<0.001$) for patients who were obese; 2.80 (95% CI: 1.33–5.89, $p=0.007$) for patients with diabetes; and 2.22 (95% CI: 1.09–4.53, $p=0.028$) for patients who were cognitively intact.

Conclusion—Three factors were associated with excess sodium excretion and two factors, obesity and diabetes, are modifiable by changing dietary food patterns.

Keywords

Heart failure; diet therapy; urine sodium; diet; sodium-restricted

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Conflicts of interest

The authors declare there that there are no conflicts of interest.

Introduction

Heart failure is a major public health concern because it is associated with high morbidity, mortality and cost. Self-care is recognized as a means of improving these outcomes.[1] Consuming a low-sodium diet is one of the most frequently recommended self-care behaviors; [2–5] however, it is estimated that only 22–55% of patients with heart failure are adherent to a low-sodium diet.[6–8] The rationale for not consuming excessive sodium is that it can lead to fluid retention, higher ventricular filling pressures and symptoms of congestion[9]—all of which put patients with heart failure at risk for acute decompensation and hospitalization.[10,11]

The general recommendation for all adults in the United States is to consume less than 2300 mg of sodium a day; however, the average American consumes approximately 3400 mg of sodium per day.[12] Sodium is one of the primary cations in extracellular fluid. By influencing the shifting of water between body compartments, it plays an important role in maintaining body fluid tonicity, blood volume and pressure. The Heart Failure Society of America (HFSA) guidelines recommend that patients with symptoms of heart failure restrict daily sodium intake to 2000–3000 mg/ day.[6] The most recent 2013 American Heart Association (AHA) guideline for the management of heart failure recommends sodium restriction (<3000 mg/day) for patients with stage C and D heart failure.[13]

Patients with heart failure face a number of barriers to effectively quantifying sodium intake and maintaining a low-sodium diet. A major barrier is that many patients do not find low-sodium foods palatable and consequently never adjust to the taste of low-sodium foods and change their eating habits.[14] A lack of knowledge about how to identify high-sodium foods, even when labeled, is also a barrier for patients.[14–17] Few restaurants offer low-sodium options, which decreases dining out opportunities for socialization with friends.[14–16] Overall, there are multiple barriers that can impede patients with heart failure from consistently following a low-sodium diet.

The aim of this study was to identify socio-demographic and clinical characteristics that predict higher than HFSA-recommended sodium intake, estimated by urinary sodium excretion, using longitudinal data of patients with heart failure. For the purposes of this study, we assumed that sodium excretion was a reflection of sodium consumption. The World Health Organization (WHO) Five Dimensions of Adherence model was used to identify factors potentially associated with poor dietary adherence.[18] This holistic model acknowledges the multi-dimensionality of adherence including social and economic conditions, the health care system, as well as condition-, therapy- and patient-related factors.[18] Previously, this model has been used to guide the assessment of medication adherence in patients with heart failure.[19]

Methods

Study population

This study was a secondary analysis of data collected in an observational prospective cohort study of 280 community-dwelling adults with previously or currently symptomatic heart

failure who were followed over a six-month time period. This study was in compliance with the Declaration of Helsinki.[20] Institutional Review Board approval was obtained from all three sites and all participants gave written informed consent. The parent study was conducted to investigate the relationship between excessive daytime sleepiness and heart failure self-care. The detailed methodology of this study has been reported elsewhere.[21] In brief, this was a prospective cohort study with patients assigned to one of four cohorts based on excessive daytime sleepiness and cognitive decline. Multiple heart failure self-care behaviors were measured including eating a low-sodium diet, which was assessed with 24-hour urine sodium specimens at three time-points (enrollment, three- and six-months). Patients with heart failure were prospectively enrolled from three outpatient settings, two in Pennsylvania and one in Delaware, between 2007 and 2010. The basic eligibility criteria for participation included: (a) chronic heart failure with prior or current symptoms, (b) ability to read and speak English, and (c) sufficiently able to complete questionnaires and study procedures (i.e. adequate cognition, English fluency, ability to read). Patients were ineligible to participate in the study if they had dementia, as measured by the Telephone Interview for Cognitive Status[22] along with other criteria specified in the parent study.[21] Most participants in this study were followed in specialty heart failure clinics and were on standard medical therapy. The usual care provided by heart failure specialty clinics was not augmented in this observational study.

Variables and measures

Clinical information (e.g. etiology and type of heart failure, left ventricular ejection fraction (LVEF), and comorbid illnesses) was abstracted from the medical record by a registered nurse at each of the three enrollment sites. These nurses were familiar with the unique electronic medical record systems in their own setting. Each nurse received extensive training to assure consistency of data collection. The principal investigator assured the fidelity of the data collection protocol at each site and was available for questions throughout the study period.

Comorbidities (including diabetes) were scored with the Charlson Comorbidity Index, a commonly used measure,[23] directly from the medical record. In this sample, all patients had a score of at least one because they all had heart failure. Validity was demonstrated by the instrument authors when comorbidity scores (categorized as low, medium, and high) predicted risk of 10-year mortality, complications, health care resource use, length-of-hospital-stay, discharge disposition and cost.[23–26]

New York Heart Association (NYHA) functional class was scored Class I–IV by a single board certified cardiologist using data obtained from a standardized interview.[27] Body mass index (BMI) was calculated based on the patient's weight at the baseline visit and self-reported height. Detailed information on participants' medications, with doses, was collected at each data collection period. Sociodemographic characteristics were self-reported.

Cognition was measured with a battery of neuropsychological tests administered in person by trained research assistants. The number of tests on which participants scored below their age-based norm was used as an indicator of cognitive status. Any participant who scored more than 1.5 standard deviations (SDs) beyond the range of normal on at least two of the

paper-and-pencil tests was considered to have mild cognitive decline. For analysis, the cognition variable was dichotomized to indicate those with or without cognitive impairment. The details of the neurocognitive battery and methodology for categorizing cognitive status are reported in detail in the parent study.[21]

In this study, dietary sodium intake was approximated using urine sodium excretion. Sodium is under tight homeostatic control and is only lost from the body as sweat (20–80 mmol/day), stool (5–10 mmol/day) or urine (1–500 mmol/day).[28] Patients with heart failure do not typically engage in strenuous physical activity; therefore do not lose sodium from heavy perspiration. Variations in sodium excretion are primarily due to variations in recent sodium intake.[29] In this study, we made the assumption that the 24-hour urinary sodium excretion samples reflected dietary sodium intake across the study period, consistent with previous studies.[10,30,31] The 24-hour urine collection method has been validated by urine recovery of oral doses of para-amino benzoic acid [32–34] and the collection procedures followed in the parent study were consistent with recommendations for collection by the Institute of Medicine.[35]

Based on criteria set forth by the 2010 HFSA practice guidelines and published papers, urine sodium in mmoles was converted to mg (mg= mmole×22.99).[5,10] The urine sodium binary outcome in this study was created using 3000 mg/day 24-hour urine sodium cut-off point for patients in NYHA Class I/II and a 2000 mg/day 24-hour cut-off point for patients in NYHA Class III/IV. Expected dietary sodium categories were compared between participants whose dietary sodium excretion was within recommended levels versus those in higher than recommended levels.

Procedures

Data were collected at baseline, three-months, and six-months by research assistants during home visits. Training for urine specimen collection was provided for the patient and family member, if available, at the baseline data collection interval. Prior to each subsequent urine collection, patients were telephoned to remind them and reinforce training for the procedure. Patients were given urine containers, collection devices, verbal and written instructions (with pictures) specifying when they should start and finish collecting the urine specimen. Participants were instructed to start the collection at 08:00, discard that specimen, and collect the final specimen at 08:00 on the following day. Prior to the patient collecting each urine specimen, the research assistant inquired about changes in the medication regimen. If the participant had experienced a recent medication change the collection was delayed for three days starting from the date the medication was changed. Participants recorded when their urine sodium collection started and finished. A courier picked up the specimens from the participant's homes after the 24-hour sample was completed.

The central laboratory at the Hospital of the University of Pennsylvania analyzed the urine samples to determine the amount of sodium in each specimen using the ion-selective electrode method (Beckman LX 20 Chemistry Analyzer, Beckman Coulter, Inc., Fullerton, California, USA).

Statistical analysis

Descriptive statistics were used to calculate frequencies with percentages and means with standard deviation (SD) for the total sample, urine sodium in g per day, and sodium consumption. Chi-square and Kruskal-Wallis tests were used to compare baseline characteristics of binary, categorical and ordinal variables between groups. For the purposes of between group comparisons, the doses for patients on loop diuretics (*n*=83) were converted to furosemide equivalents using the following formula: furosemide 80 mg=torsemide 40 mg=bumetanide 2 mg.[36] The other participants on diuretics (*n*=8) were prescribed hydrochlorothiazide and compared separately. This method has been used in other studies.[10]

Over the three points of data collection, data on the urine sodium excretion were missing overall in 23% of the participants (193/840 samples), 19% at baseline, 29% at three-months and 23% at six-months. Multiple imputations were performed for urinary sodium excretion and BMI data to account for missing time-varying covariates.[37–39] Multiple imputation is a principled, likelihood-based method using statistical modeling to impute data using the method of chained equations.[40] In short, this method creates 10 copies of the data, as recommended by Rubin.[39] The multiple imputations were based on baseline age, gender, etiology of heart failure, cognitive status and LVEF and complete data for time-varying covariates, including NYHA class, BMI and urine sodium excretion. Each dataset was then analyzed separately.[41] Estimates of the parameters of interest were averaged across all the datasets to give a single, final estimate.[41,42] After imputation, data were checked to confirm reasonable values. Standard errors and 95% confidence intervals (CIs) were estimated by calculating within and between components of variance using the method of Rubin.[39]

To determine predictors of higher than recommended sodium intake, a mixed effect logistic model with a random intercept was tested for each patient. Adjusted odds ratios (ORs), 95% CIs and *p*-values are presented for statistically significant predictors of higher than recommended sodium intake fit on the imputed data.[43] The selection of model covariates was based on four of the five dimensions of adherence from the WHO conceptual model: condition, therapy, patient-related factors and social and economic conditions as well as statistical association with the outcome variable, urine sodium excretion. The final 12 covariates included in the multivariable regression models are presented in Table 1.[44]

Two sensitivity analyses were conducted. First, mixed effect logistic estimates were compared between the complete case and imputed data models. Estimates based on imputed data were more conservative in terms of effect sizes and had narrower CIs. This was expected because CIs and model variance depend on the amount of missing data, sample size and number of imputed datasets.[40,45] Second, estimates from the mixed effects logistic models were confirmed with generalized estimating equations models with robust standard errors. Data are not shown for these two sensitivity analyses because there were no significant differences between the models. All data analyses were performed using Stata v. 11.2.[46]

Results

This sample of patients with heart failure had a mean age of 62 years (SD=12), was predominantly male (64%), functionally compromised (77% in NYHA class III or IV) with a mean LVEF of 35 (SD: 17). Over one-third of the sample had diabetes. Among participants with diabetes, 60% were also obese. Over two-thirds of the sample had hypertension, and about 20% had chronic obstructive pulmonary disease. Sample demographic and clinical characteristics are shown in Table 2. The urine sodium levels were relatively normally distributed so mean and standard errors are presented. The majority of the sample was prescribed furosemide or an equivalent loop diuretic.

The median and interquartile range values for urine sodium excretion on the complete cases at all three time points were 2770 mg (1750–3950 mg) at baseline, 2630 mg (1690–4000 mg) at three-months and 2780 mg (1900–3790 mg) at six-months. There was no trend in sodium excretion across time that would suggest that participants modified their diets in anticipation of these measurements. We found no difference in the pattern of observed values for participants with missing data and those with complete observations.

In the final model identifying patient characteristics associated with higher than recommended sodium intake (Table 3), patients with heart failure who were obese (BMI>30) had nearly three times higher adjusted odds of consuming more than recommended sodium intake compared to patients with a normal body weight (BMI<25). The adjusted odds of sodium intake above recommended level were over two times higher in patients who were cognitively intact compared to patients with some cognitive impairment. Patients with heart failure and diabetes had almost three times the adjusted odds of consuming more than the recommended sodium intake.

Discussion

The purpose of the study was to identify characteristics of patients with heart failure who had higher than recommended 24-hour urine sodium excretion. In this study, we used 24-hour sodium excretion as an estimate of dietary sodium intake, or adherence to sodium guidelines. Considering possible predictors drawn from the WHO adherence model, the three most important predictors of higher sodium excretion were patient- and condition-related factors. Patients with heart failure who were obese or had diabetes and those who were cognitively intact were more likely to excrete higher amounts of sodium.

The association between higher sodium and obesity is most likely explained by both direct and indirect effects.[31,47] One explanation for the relationship between obesity and higher sodium consumption is that high-calorie foods often contain high sodium.[48] Increased dietary sodium intake is directly correlated with increased calorie consumption.[30,49] According to the Salted Food Addictive Hypothesis, salted foods act in the brain like an opiate agonist, producing a hedonistic reward, which becomes associated with foods being judged as “delicious” or “flavorful.”[47] Withdrawal of salty foods acts like an opiate receptor withdrawal and causes perceived “cravings” for salted foods.[47] Over time, daily

consumption of salted foods can produce an addiction, which escalates and can stimulate overeating.[50]

The association between high sodium intake and obesity has also been found in rat animal models[51] and has a physiological explanation: chronic salt overload induces adipocyte hypertrophy, which enhances insulin sensitivity for glucose uptake and insulin-induced glucose metabolism.[51] Song and colleagues report a direct effect between sodium intake and risk of being overweight in humans, after adjusting for energy, water, and soda consumption;[31] however, their study sample did not include patients with heart failure, hypertension or diabetes. The relationship between obesity and sodium consumption is consistent with results of our previous study in which we found that patients with heart failure who were overweight had a four-fold increased odds of consuming more than 3000 mg of sodium per day.[52]

Other lifestyle factors may help explain the relationship between obesity and sodium consumption. Processed and packaged foods are naturally high in sodium because sodium is a natural preservative that increases the shelf life of foods. Patients with heart failure often feel fatigued and may have less energy to dedicate to the purchase and preparation of low-sodium food. Originally, we thought that purchasing fresh produce and meat may be prohibitively expensive for patients on fixed or limited incomes; however, income was not a statistically significant predictor of higher sodium excretion. Another important question is whether the guidelines should recommend varying amounts of sodium intake for people with heart failure with different body weight and other clinical characteristics. According to Gupta and colleagues, reporting in 2012, there is still uncertainty as to whether dietary sodium intake recommendations should be individualized.[9]

Diabetes was a strong predictor of higher than recommended sodium intake. This may be explained by the fact that any form of kidney disease in which tubular reabsorption of filtered sodium does not match the filtered load plus dietary intake can cause renal sodium wasting (i.e. hyperfiltration occurring in earlier stages of diabetic nephropathy). The finding that diabetes is a predictor of higher sodium intake is consistent with a previous analysis in which patients with diabetes were four times as likely to consume more than 4 g of sodium per day over time.[52] This finding may be explained by the association between diabetes and obesity, in which case diabetes is acting as a marker of obesity.

In the literature, associations between mild cognitive impairment and heart failure self-care behaviors are mixed.[1,53,54] Mild cognitive impairment has been associated with worse self-care management[55,56] probably because of an inability to perform the complex decision-making required to manage signs and symptoms. An association between cognitive impairment and poorer self-care maintenance such as eating a low sodium diet has not been reported. In a study by Cameron and colleagues, the majority of patients (73%) had unrecognized or subclinical cognitive impairment and were as likely to engage in health-promoting behaviors, such as daily weighing, as those without cognitive impairment.[56] Dickson and colleagues reported that poorer cognition was associated with better self-care behaviors among patients with heart failure in a mixed-methods study.[57] It is important to note that these prior studies focused on the subjective assessment of multiple self-care

behaviors and not exclusively on dietary sodium. Further, there were considerable differences across studies in the methods of measuring cognition that may, in part, explain differences in study findings.[57]

While it may be assumed that patients with mild cognitive impairment will not be able to follow a low-sodium diet that was not the case in this study. Patients who were cognitively intact had more than double the odds of consuming higher than recommended sodium levels compared to patients with cognitive impairment. This seemingly counter-intuitive finding may be explained by the fact that patients with cognitive impairment often have caregivers who make and provide their meals. Patients who are cognitively intact may be more self-reliant in making dietary decisions. This finding could also be explained by the rigidity that is associated with cognitive impairment.[58,59] Rigidity refers to the tendency to form and repeat particular behaviors; eating the same foods is one form of rigidity. We cannot confirm either of these hypotheses because we did not collect data on caregivers or rigidity associated with specific dietary choices in this study.

Many patients with heart failure struggle to consume levels of sodium that are considered moderate. In a study of adults with heart failure from the Southern US, only 33% of individuals consumed 2000 mg/day or less with the wide range of 522–9251 mg/day.[60] Likewise, based on 24-hour urinary sodium excretion levels, another study reported that 34% of the sample was compliant with recommendations to consume less than 3000 mg/day and only 15% consumed less than 2000 mg/day.[10]

Recently, sodium restriction for patients with heart failure has been questioned due to wide variation in the study protocols, study samples, fluid intake, measurement of sodium intake and compliance.[13] Some studies have even questioned any sodium restriction;[61,62] however, this was largely due to a very rigid definition of sodium intake (80 mmol/day).[61] Paterna and colleagues recommend a normal sodium diet, defined as 120 mmol/ day (about 2800 mg/ day) [61,62] which is consistent with the recommendation of <3 g a day for patients with Class C and D heart failure.[13] Overall, the message across these studies and a recent review by Gupta and colleagues is consistent— high sodium intake is not optimal for patients with heart failure because excessive sodium intake is associated with fluid retention; however, the lower-dose range of sodium intake needs to be better defined, and possibly individualized to patients in future studies.[9]

Strengths and limitations

One of the strengths of this study was the repeated measures of sodium excretion on the same individuals over a six-month period. Multivariate statistical methods were used for these repeated measures to account for the correlation of measures within an individual, which reduced measurement error and individual variability. The use of longitudinal data in this study also provided a clearer picture of how well patients followed a low-sodium diet over an extended period of time, instead of one point in time. Another strength of the study is that we used robust and transparent imputation methods to address potential bias from incomplete data.[37,39]

The study also had several limitations including the fact that this was a secondary analysis of a dataset that did not include measures of sodium intake. Data from carefully controlled studies show that at least seven days of direct measurement of sodium intake and excretion in the urine are needed to achieve correlations of 0.8,[63] which implies that food records and urine excretion provide complementary information about sodium intake. The relationship between sodium intake and excretion is analogous to fasting blood glucose and hemoglobin A1c in diabetic patients. These two measures provide similar, but different, information about dietary adherence in patients with diabetes. In this study, our intention was not to precisely estimate sodium intake over the previous 24 hours, but to estimate general adherence to dietary recommendations. For our purposes, 24-hour sodium excretion was the best available marker of dietary sodium consumption.

Another limitation of this study was that patients in this sample were on loop diuretics, which enhance urinary excretion of sodium by inhibiting tubular reabsorption at the thick ascending limb of the Loop of Henle, in the nephron. Arcand and colleagues, in 2010, found no statistically significant relationship between 24-hour urine sodium excretion and sodium intake from food records in patients with heart failure on a loop diuretic; however, they reported significant correlations for non-heart failure cardiac patients and for patients with heart failure who were not prescribed a loop diuretic.[64] While informative, limitations of the Arcand study were that it used a cross-sectional, correlational design in a young, relatively small sample of patients with heart failure in which other relevant variables that may explain the differences in sodium excretion were not controlled. Though there may be some imprecision from using urinary sodium excretion as a measure of dietary sodium intake for patients on loop diuretics, it is still considered the best measure for patients with heart failure.[10,30,65] In addition, we statistically controlled for diuretic use.

There are inherent limitations to a secondary data analysis, including the fact that this study was limited by the demographics and size of the original sample. For example this sample was younger than most other community-dwelling samples of patients with heart failure so the results may not be generalizable to older adults with heart failure. There may be additional predictors of higher sodium intake that were not measured in the parent study, such as fluid intake, and thus could not be controlled for. In addition, while it was a strength that we used four of the five dimensions of adherence in the WHO model, the dimensions were not measured comprehensively. For instance, due to the sample size, we were restricted to including only variables that were both conceptually consistent with the WHO dimensions as well as statistically associated with sodium excretion in the final model. Some dimensions may not have been fully captured by the variables selected.

Conclusion

Patients with heart failure who are cognitively intact, those who have diabetes or are obese are at risk for consuming more sodium than recommended. The 2013 report of the AHA guidelines for the management of heart failure recommends reasonable sodium restriction for patients with symptomatic heart failure to reduce congestive symptoms[13] and the HFSA guidelines recommend that all patients with heart failure be provided with dietary instruction.[5] Sodium dietary instructions should be contextualized within a longer

conversation with patients about their general sodium intake. Further research may support identifying higher risk patients and intervening to help them decrease their sodium intake. Ultimately interventions around dietary sodium intake may help decrease the risk of decompensation and re-hospitalization for patients with heart failure.[11,66]

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Implications for practice

- Patients who have diabetes, are obese or are cognitively intact might be at risk for consuming more sodium than recommended.
- Further research should identify patients and intervene to help them reduce high sodium consumption with tailored dietary interventions.

Table 1

World Health Organization five dimensions of adherence model.

Dimension of adherence	Variables used in this study
Social/economic factors	Race, income, highest level of education
Therapy-related factors	Diuretic (loop or thiazide)
Patient-related factors	Gender, age, body mass index, cognitive status
Condition-related factors	Heart failure type (diastolic or systolic), diabetes, hypertension, etiology (ischemic or non-ischemic)
Health-system factors	*Not available in this study*

Table 2

Demographic, social and clinical characteristics of patients, mean 24-hour urine sodium (Na+) samples and whether they were within or higher than recommended HFSA guidelines.

Variables	Total	%	Mean UrNa g/day (SE)	Na+ excretion levels Within or Higher		*p*-value
Age						
<65 years	165	41	3.08 (0.03)	48	63	**0.022**
≥65 years	115	59	2.61 (0.03)	52	37	
Gender						
Male	180	64	3.23 (0.24)	61	66	0.425
Female	100	36	2.81 (0.03)	39	34	
Race						
Black/other	105	38	2.95 (0.02)	33	37	0.597
White	175	62	3.30 (0.04)	67	63	
Income						
More than needed	98	35	2.75 (0.03)	38	33	0.269
Enough to meet needs	137	49	3.21 (0.03)	51	48	
Less than needed	45	16	3.35 (0.05)	11	19	
NYHA functional class						
Class I/II	66	23	3.03 (0.04)	33	17	**0.009**
Class III	164	59	3.02 (0.03)	49	68	
Class IV	50	18	3.28 (0.06)	18	15	
Diuretic use[a]						
Furosemide equ. (mg/day) *n*=183	183		3.07 (0.15)	72	69	0.703
≤40 mg/day	91	50	2.97 (0.14)			
>40 and ≤120 mg/day	63	34	3.33 (0.27)			
>120 mg/day	29	16	3.01 (0.27)			
Hydrochlorothiazide (mg/day)	8	3	3.30 (0.53)	1	3	0.400
Heart failure etiology (*n*=279)[b]						
Ischemic	102	36	3.11 (0.03)	35	38	0.625
Non-ischemic	177	64	3.05 (0.03)	66	61	
BMI (*n*=279)[b]						

Variables	Total	%	Mean UrNa g/day (SE)	Na+ excretion levels Within or Higher		*p*-value
Normal (18–24)	74	26	2.39 (0.03)	38	18	**0.001**
Overweight (25–29)	73	26	2.86 (0.05)	27	27	
Obese (30+)	133	48	3.53 (0.02)	35	55	
Highest level of education						
Less than high school	27	10	2.65 (0.07)	11	7	0.271
High school graduate	102	36	3.05 (0.03)	39	35	
More than high school	151	54	3.12 (0.03)	50	58	
Type of heart failure						
Systolic	194	69	3.00 (0.02)	76	69	0.475
Diastolic or mixed	86	31	3.19 (0.05)	24	31	
Charlson co-morbidity						
Low	149	53	2.96 (0.04)	56	50	0.268
Moderate	101	36	3.13 (0.06)	37	35	
High	30	11	3.26 (0.09)	7	15	
Diabetes						
Yes	107	38	3.57 (0.05)	23	47	**0.001**
No	173	62	2.72 (0.04)	77	53	
Hypertension						
Yes	181	65	3.06 (0.02)	65	63	0.729
No	99	35	3.07 (0.03)	35	37	
COPD						
Yes	222	79	2.53 (0.06)	73	81	0.129
No	58	21	3.20 (0.03)	27	19	
Cognition						**0.004**
Cognitively impaired	107	39	2.90 (0.02)	47	28	
Not cognitively impaired	165	61	3.14 (0.03)	53	72	

[a] Diuretic use: This only includes information on patients who were on diuretic medications. The proportions are only for those prescribed furosemide (*n*=183), not the entire population sample.

[b] Sample size for variables with missing values.

BMI: body mass index; COPD: chronic obstructive pulmonary disorder; equ.: equivalent; NYHA: New York Heart Association; SE: standard error, UrNa: urine sodium.

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Table 3

Factors associated with higher sodium excretion (*n*=280).

Variables	Adjusted OR	95% CI	*p*-value
Cognition			
Cognitively impaired	1.00	Reference	
Cognitively intact	2.22	1.09–4.53	**0.028**
Body mass index			
Normal (18–24)	1.00	Reference	
Overweight (25–29)	1.79	0.75–4.25	0.187
Obese (30+)	2.90	1.15–4.25	**<0.001**
Diabetes			
No	1.00	Reference	
Yes	2.80	1.33–5.89	**0.007**
Gender			
Female	1.00	Reference	
Male	1.16	0.54–2.51	0.703
Age			
≥65 years	1.00	Reference	
<65 years	1.95	0.97–3.90	0.059
Hypertension	0.51	0.25–1.04	0.068
Income	1.11	0.67–1.84	0.696
Diuretic use	1.05	0.48–2.33	0.897
Heart failure etiology	0.72	0.31–1.67	0.438
Type of heart failure (systolic, diastolic, mixed)	1.24	0.78–1.98	0.351
Education	1.14	0.67–1.96	0.607
Race	1.17	0.57–2.42	0.663

OR: odds ratio; CI: confidence interval.