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Dietary sodium intake is predicted by anti-hypertensive medication regimen in patients with heart failure.

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Abstract

Background: Low-sodium diet (LSD) adherence is foundational to heart failure (HF) self-management. Altered salt taste perception caused by angiotensin converting enzyme (ACE) inhibitors commonly prescribed to patients with HF may increase sodium consumption. We hypothesized sodium intake, indicated by dietary sodium density, would be significantly higher among patients with HF prescribed ACE inhibitor compared to those not prescribed the drug.

Objective: To assess the association between prescribed ACE inhibitor and dietary sodium density in patients with HF.

Methods: We conducted a secondary analysis of baseline data from patients with HF in an observational longitudinal study. Sodium density was derived by dividing averaged daily sodium intake from 4-day food diaries by averaged kilocalories consumed. Medical chart review was conducted to ascertain prescribed medications. Patients were categorized as prescribed and not prescribed an ACE inhibitor. T-tests were conducted to compare sodium intake between groups and linear regression to examine if prescribed ACE inhibitor independently predicted sodium density controlling for age, gender, New York Heart Association (NYHA) class, prescribed diuretics and beta blockers.

Results: Analyses included 255 patients with HF age 61 ± 12 , 67% male, 44% NYHA Class III/IV, 68% prescribed an ACE inhibitor. Compared to those not prescribed an ACE inhibitor, 13% more sodium per kcal was consumed by patients prescribed an ACE inhibitor. Prescribed ACE inhibitor independently predicted dietary sodium density ($\beta = .238$, $p = .009$).

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Conclusions: Sodium intake was higher among patients prescribed ACE inhibitors. Interventions to assist patients with HF with dietary sodium adherence can be informed by assessing medication regimens.

Introduction

In patients with heart failure, self-care is pivotal to appropriate management of the condition including adherence to a low sodium diet.¹ Unfortunately, many patients with heart failure do not follow the recommended low sodium diet with rates of adherence reported to be as low as 22%.¹⁻⁹ Patients have identified taste of food as one reason for not adhering to this recommendation.^{10,11} Most patients report that reducing salt makes food unpalatable and limits the enjoyment of eating, negatively affecting quality of life.^{10,11}

Salt taste sensitivity, defined as the ability of an individual to identify the taste of sodium, is comprised of two dimensions: recognition and detection.¹² Recognition is the minimum amount of salt required to identify sodium in a food; detection is the minimum amount of salt required to detect a change in amount of sodium in a food.¹² Salt taste sensitivity is a complex phenomenon that is affected by factors that include neural, chemical, and structural inputs which can be altered by external factors such as medications, dietary salt intake, or health conditions such as heart failure.¹²⁻¹⁴

Several medications prescribed to treat cardiovascular disease (CVD) have been associated with disturbances in salt taste sensitivity as well as other taste disturbances. For example, diuretics are known to reduce saliva production resulting in dry mouth.¹⁵ Because saliva is essential for breaking down food into taste substances, reduced saliva production adversely affects ability to taste food. Calcium channel blockers also adversely affect taste by interfering with the availability of zinc.¹⁵ Zinc is a central component of the gustin protein that has a major role in the development and maintenance of fungiform papillae. Reduced availability of zinc may reduce papillae involved in taste.¹⁶ Adrenergic receptor antagonists are thought to cause a disturbance in taste due to competition over binding sites with catecholamines, which are neurotransmitters that are involved with taste buds.¹⁵ Angiotensin converting enzyme (ACE) inhibitors and angiotensin II receptor blockers (ARBs), both commonly used treatments in patients with heart failure, have also been shown to cause taste disturbances. In a randomized control crossover trial, seven participants were given either an ACE inhibitor or an ARB and tested for taste ability.¹⁷ ACE inhibitors and ARBs were found to worsen taste ability over placebo. While underlying mechanisms that cause the taste disturbances are not clear, the investigators theorized that it might be related to alteration of the renin-angiotensin-system, with angiotensin II and aldosterone both associated with increased salt appetite.^{13,17}

Given that decreased salt taste sensitivity may result in increased salt consumption,¹⁷⁻²⁰ people with heart failure who are prescribed an ACE inhibitor may be at greater risk for higher salt consumption than those not prescribed an ACE inhibitor. However, the relationship between ACE inhibitors and salt consumption has not been well-studied. Therefore, the purpose of this study was to examine if prescription of an ACE inhibitor predicted sodium consumption. We hypothesized that sodium consumption (calculated based

on amount of sodium consumed per kilocalorie or sodium density), as an indicator of salt preference, would be higher in patients with heart failure prescribed ACE inhibitors than in patients who were not prescribed ACE inhibitors.

Methods

Design.

This was a secondary analysis of data from patients with heart failure enrolled in an observational longitudinal study to examine the relationships between body fat mass, body fat distribution, cytokine activity, and nutritional intake.²¹ Patients were recruited into the study and followed for a period of 1 year. For the purposes of our study, we analyzed data collected at baseline.

Sample.

Data from 255 patients with heart failure were included in this analysis. Patients were recruited from outpatient facilities at three large academic healthcare facilities between 2006 and 2009. Two of the facilities were located in the southeastern region of the U.S. and one was located in the Midwestern region. Inclusion criteria were patients who: (1) had a confirmed diagnosis of chronic heart failure; (2) had a New York Heart Association (NYHA) classification of I–IV; (3) were stabilized on medication for 3 months prior to recruitment; (4) had no history of hospitalization in the 3 months before recruitment; and (5) were able to read and speak English. Patients were excluded if they had: (1) valvular heart disease as a cause of the heart failure; (2) a myocardial infarction within the last 3 months; (3) been referred for heart transplant; (4) uncontrolled diabetes mellitus (HbA1c > 8%); (5) an illness associated with systemic inflammation, weight loss, or decreased appetite; or (6) a cognitive impairment that prevented giving informed consent.

Measurement of variables.

Demographic and clinical data.—Participant demographic data including age, gender, race/ethnicity, and smoking status were collected through self-report via a sociodemographic questionnaire. New York Heart Association (NYHA) class and prescribed medications were ascertained by trained research nurses using a structured patient interview. Body mass index (BMI) was calculated using the standard kg/m² formula.

Dietary sodium intake.—Nutritional data were collected using food diaries that patients completed for a four-day period, including three weekdays and one weekend day. The use of four-day food diaries is a valid method from which nutrients in the diet, including sodium, can be measured.^{19,22} Prior to recording food diaries, patients met with a research assistant in their homes who, using food models and pictures of appropriate portion sizes, instructed the patients how to accurately weigh and record dietary information. Each participant was given an electronic digital scale for accurate recording of portion sizes. At the end of the four-day food diary collection, patients met with a dietitian who collected missing information, verified serving sizes, and verified food preparation methods. Food diaries were analyzed using the Nutrition Data System for Research software (NDS-R 2007; NRCC, Minneapolis, MN).

Sodium density.—Sodium density was defined as the averaged four-day sodium intake divided by averaged four-day kcal intake. This was chosen as the metric for salt consumption because it controls for differences in sodium intake due to differences in amount of food consumed and is a better indicator of patients' preferred saltiness of food. This approach has been utilized by the United States Department of Agriculture (USDA) and the Centers for Disease Control (CDC) to estimate sodium consumption adjusted for energy consumption because higher sodium intake is often accompanied by higher caloric intake.^{23,24}

Procedure.

Approval for this study was obtained from the Institutional Review Boards at each study site. Patients were recruited from affiliated outpatient clinics after referral from a nurse or physician. After providing written informed consent, patients were visited in their homes by a trained research nurse and instructed how to complete four-day food diaries. The morning after completing the diaries, patients visited one of the three clinical research centers at which time demographic and clinical data were collected. A dietitian also reviewed the food diaries with participants during this visit as a way of validating completion and accuracy of the information provided.

Data Analysis.

Patients were categorized into two groups: those who were prescribed ACE inhibitors and those who were not. Differences in demographic and clinical characteristics and in dietary sodium density between the two groups were compared using independent t tests or chi square as appropriate. A multivariable linear regression was conducted to determine whether prescribed ACE inhibitors independently predicted dietary sodium density. In the model we controlled for age, gender, ethnicity (non-Hispanic Whites vs other races), NYHA class (I-IV), smoking status (smokers vs non-smokers), prescribed beta blockers, prescribed angiotensin II receptor blockers (ARBs) and prescribed diuretics. All independent variables were entered into one model for analysis. An alpha of 0.05 was set *a priori*.

Results

Sample characteristics.

The majority of patients were White males with slightly more than half in NYHA class I/II. Mean BMI was 29.7 ± 6.8 . Diuretics (74.5%), beta blockers (88.6%), and ACE inhibitors (69.8%) were commonly prescribed but less than a quarter of patients were prescribed an ARB. In our sample, 178 patients were prescribed ACE inhibitors (69.8%). Sample characteristics and comparisons between patients who were and were not prescribed an ACE inhibitor are presented in Table 1. The only significant between-group differences were that a greater number of those prescribed an ACE inhibitor were male and were also prescribed a beta blocker.

Comparison of sodium density between those prescribed and not prescribed an ACE inhibitor demonstrated that patients prescribed an ACE inhibitor consumed approximately 13% more sodium per kcal than patients who were not prescribed an ACE inhibitor (1.8

± 0.55 mg sodium/kcal vs. $1.6 \text{ mg} \pm 0.43$ sodium/kcal). In our regression model, prescription of an ACE inhibitor predicted higher sodium density ($b = .230$, $t(234) = 2.566$, $p = .011$), controlling for age, smoking status, ethnicity, NYHA class, and other prescribed medications (Table 2). No other medications predicted sodium density. Among the controlled variables, only BMI predicted sodium density ($b = .018$, $t(234) = 3.045$, $p = .002$).

Discussion

In support of our hypothesis, patients who were prescribed an ACE inhibitor had a significantly higher consumption of sodium per kcal compared to those who were not prescribed an ACE inhibitor. In accord with current practice guidelines,^{25,26} patients with heart failure are commonly prescribed medications including ACE inhibitors in conjunction with a low sodium diet.²⁵ However, as indicated by the results of our study, prescribed ACE inhibitors may present a barrier to low sodium diet adherence. Supporting our findings are studies that have shown ACE inhibitors are associated with taste disturbances.^{12,17,27} Taste disturbances most commonly associated with ACE inhibitors range from loss of taste to alteration of salty, sweet, or metallic tastes that include a lingering taste in the mouth or taste confusions such as sweet for salty or sweet for bitter.²⁸⁻³⁰ These disturbances may result in lowered salt taste sensitivity and increased salt consumption.¹⁹

Other medications are also suspected to produce taste disturbances, such as ARBs, diuretics, and beta blockers.^{12,17} However, similar to other studies,¹² only ACE inhibitors predict dietary sodium density in our patients with heart failure. The effect of ACE inhibitors may be exacerbated by heart failure induced over-activation of the renin-angiotensin-aldosterone system (RAAS), which is linked to water and salt consumption behaviors.¹³ ACE inhibitors work by preventing the conversion of angiotensin I to angiotensin II and increasing levels of bradykinins. ACE inhibitors are also known to chelate with serum zinc which results in depletion of zinc levels.³¹ It is possible that the depletion of zinc that occurs with the chelating action of ACE inhibitors causes a disturbance in the functioning of gustin, which in turn produces a disturbance in salt taste. The influence of ACE inhibitors on taste may be due to the reduction of serum zinc concentration through this mechanism.^{31,32}

In contrast, ARBs work by selectively blocking angiotensin II from binding to receptors but have not been linked to serum zinc depletion.^{17,29} This may explain why prescribed ARBs did not predict dietary sodium density. Although some diuretics have also been linked to salt taste sensitivity alterations, this has only been demonstrated for diuretic medications that are less commonly prescribed today. For example, the potassium-sparing diuretic amiloride, rarely used today among patients with heart failure, is thought to block ion channels in the taste receptor membrane altering the flow of sodium ions.²⁹ In a literature review, Doty and colleagues demonstrate that amiloride can alter salt taste intensity and sensitivities.²⁹ The majority of patients in our study were prescribed loop diuretics, which have not been associated with salt taste sensitivity, as supported by our findings.

de Souza et al. report that patients with heart failure may have a greater preference for salt in their food.¹³ These authors suggest that because most patients with heart failure have taken multiple medications over a long period of time, the medication could affect the palatability

of food, although they did not define the length of time or the particular medication.¹³ Consequently, patients with heart failure taking a medication that reduces the ability to taste the salt in their food¹² may need to use more salt to meet their already heightened preference. This behavior, in turn, can increase the risk of heart failure exacerbation. Our results suggest that ACE inhibitors may be one of those medications.

The only other variable in the model that was found to be a significant predictor of sodium density in the diet was BMI. This is not a surprising finding as sodium has been linked to BMI previously. In particular, excessive sodium consumption has been associated with high calorie foods and the consumption of sugar sweetened beverages which can lead to overweight or obesity^{33,34} Specifically, sodium consumption in patients with heart failure has been linked to fluid retention which in turn has been shown to result in increased weight.³⁵ Our results confirm these previously reported associations between BMI and sodium consumption.³³⁻³⁵

Our finding that gender was not a significant predictor of sodium density may appear to be contrary to findings from previous studies in which men were shown to have higher rates of non-adherence to low sodium diet.⁶ However, because the amount of sodium consumed is tied to the amount of food consumed, using sodium density instead of total sodium intake controlled for differences in amount of food consumed between men and women. Consequently, this method allowed us to directly compare preferred level of sodium in food between men and women.

A limitation of our study was that we did not use a measure of medication adherence. Rather, patients reported a list of which medications they were taking during the structured patient interviews. It is unknown whether a certain level of adherence to ACE inhibitors is necessary for ACE inhibitor use to be associated with sodium density. Further research is required to further explore this link. Another limitation was that there was not an objective measure of salt taste function such as salt taste sensitivity. Although the addition of such a measure would provide more specific data about taste, sodium density is a proxy for sodium consumption and supports conclusions about taste function based on eating behavior.

The findings from our study can inform educational efforts performed by nurses interacting with patients with heart failure. Understanding that ACE inhibitor use is associated with an increase in sodium consumption per kcal possibly because of alteration in salt taste sensitivity will allow providers to preemptively plan for this side effect. Nurses can educate patients who are taking ACE inhibitors regarding the expected decrease in salt taste sensitivity. To offset this effect, nurses can also emphasize means by which the taste of food can be enhanced such as exploring the use of spices that can enhance flavor without increasing the use of salt. Involving a trained dietitian on the care team could be of particular benefit for patients prescribed ACE inhibitors. Also, some researchers have shown that prescribing zinc with ACE inhibitors may decrease the drug's influence on salt taste sensitivity.³¹ The reason for this may be due to an effect of ACE inhibitors that is thought to alter zinc at the receptor level which changes the conversion of angiotensin I to angiotensin II. This is because zinc is necessary for the catalytic action of angiotensin converting enzyme.^{13,29,36} In addition, taste disturbance is linked to zinc deficiency which may explain

why this drug produces the effect.^{12,27,31,37} Future research efforts should explore the use of zinc supplements to offset this effect. Likewise, when educating patients with heart failure, consideration of possible taste disturbances may allow nurses to be more specific when discussing changes in taste that may present a barrier when patients attempt to alter their diets. Nurses may be able to offer advice on how to acclimate to decreased sodium in the diet while also dealing with a possible decrease in taste sensitivity, suggesting seasoning substitutes that will enhance the flavor of food without raising sodium content. More research is required to further refine information regarding the association of ACE inhibitor use and sodium consumption as well as to develop educational interventions aimed at helping those on ACE inhibitors to decrease their sodium consumption. Future research should include measures of salt taste sensitivity to further elucidate the effects of ACE inhibitor use on taste perception.

Conclusions

The finding that prescribed ACE inhibitors predicted the sodium consumption of patients with heart failure has implications for interventions to improve heart failure self-care. Aware of the potential alteration of sodium consumption associated with ACE inhibitors, nurses and other health professionals can closely monitor dietary sodium intake of patients prescribed ACE inhibitors and provide appropriate education and interventions. These measures can better support adherence to low sodium diet recommendations among patients with heart failure thereby decreasing the risk of heart failure exacerbation and rehospitalization.

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Table 1.

Sample Characteristics by Angiotensin Converting Enzyme inhibitor (ACEi) grouping.

Characteristic	Total Sample (N=255)	Not prescribed ACEi (n=77)	Prescribed ACEi (n=178)	P-value
Age (years)	61.1 ± 11.8	61.2 ± 12.2	61.0 ± 11.7	.39
Gender (male)	174 (68.2%)	42 (54.5%)	132 (74.2%)	.003
Ethnicity (White)	185 (72.5%)	54 (70.1%)	131 (73.6%)	.64
Smoker (current or recent)	66 (25.9%)	23 (29.8 %)	43 (24.2%)	.35
NYHA class (III/IV)	112 (43.9%)	32 (41.6%)	80 (44.9%)	.78
BMI (kg/m ²)	29.7 ± 6.8	29.4 ± 7.0	29.8 ± 6.7	.51
Prescribed diuretic (yes)	190 (74.5%)	59 (76.6%)	131 (73.6%)	.87
Prescribed beta blocker (yes)	226 (88.6%)	64 (83.1%)	162 (91.0%)	.08
Prescribed angiotensin receptor blocker (yes)	48 (18.8%)	41 (53.2%)	7 (3.9%)	.001

Data are mean ± SD or n (%)

Table 2.

Linear Regression Results

	<i>b (CI)</i>	SE	β	P value
Age (years)	.000 (–.006, .006)	.003	–.007	.925
Gender (male reference group)	.047 (–.097, .190)	.073	.042	.523
Ethnicity (non-Hispanic White reference group)	–.113 (–.274, .047)	.082	–.096	.166
Smoking (past or never smoke reference group)	.101 (–.062, .264)	.083	.085	.225
NYHA Class (Class I/II reference group)	.021 (–.115, .158)	.069	.020	.758
BMI (kg/m ²)	.018 (.007, .028)	.005	.224	.002
Prescribed diuretic (not prescribed reference group)	.077 (–.082, .237)	.081	.064	.339
Prescribed beta blocker (not prescribed reference group)	–.092 (–.296, .112)	.104	–.056	.376
Prescribed Angiotensin II Receptor Blockers (not prescribed reference group)	.005 (–.201, .212)	.105	.004	.959
Prescribed Angiotensin Converting Enzyme Inhibitors (not prescribed reference group)	.230 (.053, .406)	.089	.204	.011

Note. The dependent variable was sodium density. R^2 for the full model = .070, p = .044.