

## Original Contributions

### EATING A LARGER NUMBER OF HIGH-SALT FOODS IS NOT ASSOCIATED WITH SHORT-TERM RISK OF ACUTE DECOMPENSATION IN PATIENTS WITH CHRONIC HEART FAILURE

Jesse A. Borke, MD\*† and Peter C. Wyer, MD‡

\*Advocate Christ Medical Center, Oak Lawn, Illinois, †New York Presbyterian Hospital, New York, New York, and ‡Columbia University College of Physicians and Surgeons, New York, New York

Reprint Address: Jesse A. Borke, MD, Emergency Medicine, Advocate Christ Medical Center, 4440 W. 95th Street, Room 185W, Oak Lawn, IL 60453

**Abstract—Background:** Risk factors for exacerbation of congestive heart failure have not been consistently validated. **Objective:** Our objective was to examine the role of short-term dietary sodium intake in acute decompensated heart failure. **Methods:** Patients with chronic congestive heart failure presenting to the Emergency Department for either acute decompensated heart failure (cases) or for other reasons (controls) were included in a case-control study. Cases and controls were compared with respect to age, smoking, recent sodium intake, medication nonadherence, coronary artery disease, and hypertension. A food frequency questionnaire was utilized to estimate recent sodium intake, defined as the number of food types consumed in the previous 3 days from the 12 highest-sodium food categories. **Results:** There were 182 patients enrolled. One patient was excluded due to uncertainty about the primary diagnosis. When adjusted for age, smoking, medication nonadherence, coronary artery disease, and hypertension, acute decompensated heart failure was not associated with short-term dietary sodium intake. The odds ratio for acute decompensated heart failure for each increase in the number of high-sodium food types consumed was 1.1 (95% confidence interval 0.9–1.3;  $p = 0.3$ ). Acute decompensated heart failure was associated with medication nonadherence, with an odds ratio for decompensation of 2.5 (95% confidence interval 1.2–5.1;  $p = 0.01$ ). **Conclusions:** Patients with chronic congestive heart failure who presented to the Emergency Department with acute decompensated heart failure were no more likely to report consuming a greater number of high-sodium foods in the

3 days before than were patients with chronic congestive heart failure who presented with unrelated symptoms. On the other hand, those who presented with acute decompensated heart failure were significantly more likely to report nonadherence with medications. © 2013 Elsevier Inc.

**Keywords—**heart failure; dietary sodium; patient adherence

#### INTRODUCTION

Risk factors for exacerbation of congestive heart failure have not been consistently validated. The role of dietary sodium, in particular, is poorly understood. Six to ten percent of people in the United States older than 65 years of age have congestive heart failure, and the prevalence is increasing due in part to an increasingly aged population (1–5). This has led to an increase in the number of presentations for heart failure, with concomitant costs coming primarily from Emergency Department (ED) and inpatient hospital services (6). The Acute Decompensated Heart Failure National Registry confirms that the management of acute decompensated heart failure is an ED problem, as >75% of patients admitted with heart failure arrive through the ED (7). Exacerbation of congestive heart failure results in considerable mortality and morbidity, including frequent hospitalizations and lowered quality of life (8,9).

Given the high stakes of potential outcomes of patients presenting with acute decompensated heart failure, it is particularly important for Emergency Physicians to have as much information as possible about the factors that contribute to decompensation of chronic heart failure to efficiently search for the underlying causes of decompensation events and, reciprocally, to avoid unnecessary workups. On occasion, Emergency Physicians can also contribute to effective preventive counseling.

Clinical studies to date have provided little information about preventable predictive factors for acute decompensated heart failure (10). Dietary sodium restriction nonadherence has been found to occur in up to 77% of patients with chronic congestive heart failure (11–18). Other authors have reasoned that “salt binging” can precipitate acute decompensated heart failure, and that one excessive sodium load from a single high-salt meal could lead to decompensation and an ED visit (19–22). However, to our knowledge, there has been no controlled study of the association between acute salt loads and presentation for acute decompensated heart failure. Observational studies examining precipitants of acute decompensated heart failure have found nonadherence to be present in patients with decompensation (23–27). However, those studies did not compare chronic heart failure patients presenting with acute decompensated heart failure to congestive heart failure patients not experiencing decompensation, or to other controls. We sought to examine the role of behavioral factors associated with acute decompensated heart failure, particularly dietary salt indiscretion, in patients presenting to the ED.

## METHODS

### *Study Design*

We performed a case-control study to evaluate the association between acute decompensated heart failure and recent sodium intake. All data were collected by research associates (RAs) who interviewed all patients while still in the ED. The study was approved by the Institutional Review Board of the university medical center.

### *Setting*

Our facility serves an ethnically diverse population, including a large number of patients from the surrounding predominantly Dominican Hispanic community. ED volume is approximately 65,000 per annum. Patients 21 years of age and older are triaged to the adult ED.

### *Selection of Participants*

All patients with established congestive heart failure presenting to our university tertiary referral center adult ED for acute decompensated heart failure or for other reasons between March 31, 2007 and August 22, 2008 were eligible for this study.

*Cases.* We identified patients with a history of congestive heart failure from any etiology, by either the medical record or by report, from those who presented to the ED with acute decompensated heart failure (based on the diagnostic assessment of the medical teams assigned to them). The etiology of congestive heart failure included ischemic, hypertensive, valvular, cardiomyopathic, and dysrhythmic heart disease. We excluded patients presenting with dyspnea due to a different cause, such as pneumothorax or chronic lung disease.

*Controls.* We identified patients with pre-existing congestive heart failure from any etiology, by the medical record or patient report, from those who presented to our ED with symptoms unrelated to congestive heart failure or dyspnea. The control patients had ED diagnoses that were not congestive heart failure, or assessments of other causes of dyspnea. RAs or medical residents screened patients' charts and medical records to identify patients eligible for inclusion. RAs were Columbia University premed post-baccalaureate students during the spring and fall semesters. There were 12 per semester. During the summer months, a smaller number of RAs were available. All RAs underwent significant orientation training by the authors. They sought to enroll every eligible patient they identified. We excluded patients who were unable (e.g., patients who were on a ventilator or with altered mental status) or unwilling to answer the survey questions.

### *Protocol and Measurements*

Research Assistants blinded to the purpose of the study enrolled patients at the time of their visits to the ED. They then collected data using standardized patient questionnaires. We used this method of survey administration to maximize the response rate. It has been previously shown that a patient's self-report in a structured interview correlates well with objective measures, such as pill counts (28). The RAs were trained by the investigators to ask the questions in a standard order using standardized phrasing. They used the hospital translation services as needed. To reduce recall bias, the RAs interviewed all patients while they were still in the ED and as soon as possible after they were adequately stabilized and able to participate.

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