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The Association of Insurance Status and Neighborhood Deprivation with Mortality in Hospitalized Adults with Comorbid Heart Failure and Diabetes

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2018

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Abstract

The Association of Insurance Status and Neighborhood Deprivation with Mortality in Hospitalized Adults with Comorbid Heart Failure and Diabetes

By Kaitlyn Long

Background: Heart failure and diabetes are significant public health issues, individually and simultaneously. Social determinants play a major role in the incidence of both conditions, access to health care and services, and poor health outcomes. The impact of race, individual socioeconomic position, and geography on diabetes incidence have been thoroughly studied. Few studies examine the joint role of healthcare access and neighborhood characteristics with health outcomes in patients hospitalized with heart failure and co-morbid diabetes.

Objective: The purpose of this study was to evaluate the relationship of insurance status and neighborhood deprivation with mortality among hospitalized patients with comorbid acute heart failure (HF) and diabetes mellitus (DM).

Methods: A retrospective cohort of Black and White patients with acute HF and DM with a recorded hospitalization between 2010-2018 within a single healthcare system was created. Among 10,598 patients (mean age 72 years, 47.4% female, 55.0% Black), we used log binomial regression to evaluate the association of individual insurance status and quartiles of a neighborhood social deprivation index (SDI; measured at the zip code level, where quartile 1 indicated lowest deprivation) with death, stratifying by race and sex.

Results: From 2010 to 2018, the absolute risk of all-cause mortality was 20.8%. In Black women, those with Medicaid insurance were less likely to die than those with private insurance (RR=0.63; 95% CI 0.40-0.98). In White women, those living in SDI quartile 2 were less likely to die than those living in SDI quartile 1 (RR-0.72; 95% CI 0.52-0.98). There were no other significant differences in mortality between insurance status or SDI quartiles across race and sex stratifications.

Conclusions and Recommendations: Among hospitalized patients with a history of HF and DM, insurance status and neighborhood deprivation were not significantly associated with all-cause mortality in most demographic groups. Access to quality healthcare may even out racial disparities in mortality outcomes in patients with comorbid HF and DM. Further research should examine a composite of death and hospital readmission and employ time-to-event methods.

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CHAPTER 1: Introduction

Diabetes mellitus (DM) and heart failure (HF) are major public health issues with significant morbidity and mortality. Approximately 34.2 million adults in the U.S. have diabetes and an estimated 6.2 million adults have HF.[1,2] The prevalence of HF continues to rise over time.[2] Diabetes and HF frequently occur together because of shared risk factors and also because diabetes has cardiotoxic effects.[3] The prevalence of diabetes in adults with HF ranges from 10% to 47%, depending on specific characteristics of the population (i.e., age, country, and severity of HF diagnosis).[4–9] The proportion of patients hospitalized for HF with diabetes is higher than the general population, estimated to be at 40% and higher.[10]

As with other cardiometabolic conditions, both diabetes and HF disproportionately affect racial and ethnic minority populations. In the U.S., Asian American adults (9.2%), non-Hispanic Black adults (11.7%), Hispanic adults (12.5%), and American Indian/Alaskan Native adults (14.7%) had higher rates of diabetes compared to non-Hispanic White adults (7.5%).[1] In addition to the high burden of disease, racial/ethnic minorities are more likely to die or experience complications due to diabetes.[11,12] In the U.S., Black adults have the highest incidence and prevalence of HF, as well as the worst clinical outcomes.[13]

Research indicates that the high burden of both diabetes and HF among Black adults arises largely from underlying modifiable CV risk factors.[14] Among these modifiable contributors to outcome disparities is management and treatment for these conditions. In diabetes management, there has been evidence of gender and racial disparities. It was found that women are less likely than men to receive the care recommended by guidelines.[15] Evidence also suggests that racial/ethnic disparities in diabetes care process exist within individual treatment facilities.[16]

Social determinants of health must also be considered as they are related to the incidence of diabetes in the U.S.[17] Social determinants are conditions in which people are born, live, and age that affect a wide range of health outcomes and risks.[17,18] Current interventions aim to address biologic and behavioral factors, but it is important to address physical and social factors such as low income, employment insecurity, low educational attainment, and poor living conditions, all of which are associated with diabetes and HF.[17,19]

This thesis is part of a larger body of work examining management and health care related to HF and diabetes within a social determinants' framework. This thesis focuses specifically on the association between social deprivation and all-cause mortality among adults with acute HF and comorbid diabetes. This research was motivated by the hypothesis that social determinants of health, including neighborhood deprivation and underinsurance, increase the risk of death among patients hospitalized for HF with comorbid diabetes. Identifying potential disparities in mortality will contribute to conversations surrounding the role of social determinants in optimizing outcomes among patients with HF and diabetes, as well as inspiring interventions that would address inequities.

CHAPTER 2: Literature Review

Heart Failure and Diabetes

Heart failure (HF) and diabetes are significant public health issues in the United States (U.S.), specifically in the Southeastern U.S. where HF and diabetes are the most prevalent.[20,21] An estimated 6.2 million American adults had heart failure between 2013 and 2016, an increase from 5.7 million in 2009-2012.[2] According to data collected from National Health and Nutrition Examination Survey (NHANES) 2013-2016, an estimated 26 million American adults (9.8% of the population) had diagnosed diabetes, 9.4 million (3.7%) had undiagnosed diabetes, and 91.8 million (37.6%) had prediabetes.[2] Although diabetes and HF are individually associated with substantial morbidity and mortality, they often occur simultaneously. Among those hospitalized with HF, approximately 40% of patients have diabetes.[10] Among adults with diabetes, the prevalence of HF is between 9% and 22%, fourfold higher than the general population.[22]

Diabetes is a risk factor for incident HF through multiple mechanisms, including via myocardial ischemia/infarction as well as the development of diabetic cardiomyopathy which can occur even in the absence of major epicardial coronary artery disease (CAD).[23,24] In the Framingham Heart Study, Kannel and McGee followed a cohort of 5,209 adults (aged 45-74 years) over a 20-year surveillance period relating various cardiovascular events to prior evidence of diabetes.[25] They found that diabetes was associated with a nearly twofold increase in the risk of incident HF in men (risk ratio [RR]=1.82) and a fourfold increase in women (RR=3.75). Both morbidity and mortality due to HF were higher for diabetic women compared to diabetic men. Another study utilizing data from NHANES evaluated risk factors of HF in a cohort of 13,643 adults (aged 25-74 years).[26] Over an average of 19 years of follow-up, there was evidence of increased risk of HF among those with a history of diabetes compared to those without a history

of diabetes (RR=1.85; 95% confidence interval [CI] 1.51-2.28; p<0.001).[26] Unlike the Framingham Heart Study, He and colleagues did not find a difference in HF risk by sex.[25,26]

The relationship between DM and HF is likely bidirectional. Insulin resistance is present in approximately 60% of patients with HF.[27] Because insulin resistance is a leading mechanism in the development of prediabetes and type 2 diabetes, HF has been investigated as a risk factor for diabetes.[28] In a cohort of 1,620 non-diabetic adults (aged 18 years and older) with HF from the Candesartan in Heart Failure Assessment of Reduction in Mortality and Morbidity (CHARM) program, Preiss and colleagues found the incidence of diabetes as 27.8 cases per 1,000 patients per year.[29] They had an unexpected yet interesting finding that younger age was an independent predictor of diabetes and found that younger patients with HF had a greater chance of developing diabetes. They gave potential explanations of longer survival times and higher BMI.[29] In a study of 3,748 nondiabetic adults (aged 65 years and older), Guglin and colleagues found that HF significantly increased the odds of developing overt diabetes (odds ratio [OR]=4.78; 95% CI 1.84-12.4; p<0.001) in participants with normal fasting glucose baseline.[30] They also found HF to be significantly associated with worsening diabetes status (OR=2.43; 95% CI 1.38-4.29; p=0.002) adjusting for gender, age, BMI, height, weight, smoking, exercise, alcohol consumption, psychosocial factors, and other diagnoses.[30] Despite finding significant associations, the authors noted a potential limitation that the original study was designed to examine cardiovascular outcomes and not diabetes and the analysis was limited from the original nondiabetic cohort (78.4% of subjects from original cohort included).[30]

Disparities in Diabetes Burden and Complications

While the prevalence of diabetes among adults in the U.S. is quite high, the burden of disease varies across racial and ethnic groups. Cheng and colleagues utilized three cycles of NHANES data (2011-2012, 2013-2014, and 2015-2016) to examine the prevalence of total diabetes, including self-reported diabetes or undiagnosed diabetes, among a cross-section of 7,575 U.S. adults. They demonstrated the the prevalence of total diabetes was 12.1% (95% CI 11.0%-13.4%) for non-Hispanic White, 20.4% (95% CI 18.8%-22.1%) for non-Hispanic Black, 22.1% (95% CI 19.6%-24.7%) for Hispanic, and 19.1% (95% CI 16.0%-22.1%) for non-Hispanic Asian adults (overall $p < 0.001$).[31] Because of self-reporting and use of a one-time measurement of glucose level for defining undiagnosed diabetes, there is a potential for misclassification, so estimates may vary.[31] Another study examined whether health indicators have improved or worsened among minority adults. Odlum and colleagues extracted 4,856,326 participant records of adults aged 45 years and older from the Behavioral Risk Factor Surveillance System (BRFSS) between January 1999 and December 2018.[32] Black adults showed an increase in diabetes over the past 20 years ($\beta = 0.52\%$; $p < 0.001$). When comparing the two trend lines between Black and White adults, the Black-White gap worsened in diabetes (0.14%; $p<0.001$).[32] Racial and ethnic disparities exist in diabetes and appear to be widening.

In addition to racial and ethnic disparities in incident and prevalent diabetes, there also is growing evidence of disparities in diabetic complications. In a cohort study of 67,544 diabetic adults (aged 35-95 years) from three health care systems located in Southern Louisiana, four major diabetic complications (coronary heart disease [CHD], HF, stroke, and end-stage renal disease [ESRD]) were assessed.[12] It was found that Black participants had higher rates of HF, stroke, and ESRD compared to White participants after adjusting for age, but lower CHD rates. Interaction by sex was also assessed. Black women had a higher risk for HF (hazard ratio

[HR]=1.26; 95% CI 1.18-1.34), stroke (HR=1.15; 95% CI 1.08-1.22), and ESRD (HR=1.32; 95% CI 1.24-1.40) compared to White women while Black men had a higher risk for HF (HR=1.33; 95% CI 1.25-1.43) and ESRD (HR=1.47; 95% CI 1.37-1.57) than White men after adjusting for different health care systems, age, smoking, BMI, systolic blood pressure, HbA1c, LDL and HDL cholesterol, triglycerides, use of antihypertensive drugs, use of glucose-lowering medications, and use of lipid-lowering medications.[12] Another study examined the prevalence of diabetic retinopathy, one of the most common microvascular complications of diabetes, using a cross-section of 1,006 diabetic adults (aged 40 years and older) from NHANES.[33] The results suggest the prevalence of diabetic retinopathy and vision-threatening retinopathy was high, especially among Black adults. Black adults had a significantly larger (p=0.01) crude prevalence of diabetic retinopathy (38.8%; 95% CI 31.9%-46.1%) compared to White adults (26.4%; 95% CI 21.4%-32.2%). Black adults also had a significantly larger (p=0.01) crude prevalence of vision-threatening retinopathy (9.3%; 95% CI 5.9%-14.4%) compared to White adults (3.2%; 95% CI 2.0%-5.1%).[33] These studies strengthen the evidence of the racial disparities in diabetes complications in the U.S.

Diabetes Management and Treatment

Although diabetes is not curable, it is treatable and manageable. The Diabetes Control and Complications Trial (DCCT) was designed to determine whether vascular complications in insulin-dependent diabetic patients could be delayed or prevented through a primary prevention study and a secondary intervention study.[34] The DCCT was a controlled clinical trial of 1,441 participants comparing intensive therapy (use of insulin infusion pump or multiple insulin injections) to conventional therapy consistent with the standard of care in the 1980s (one or two

injections of insulin per day with no predefined target ranges for glycemic control).[34,35] More than 99% of participants completed the study after an average of 6.5 years. The researchers found a 35%-76% reduction in the early stages of microvascular disease with intensive therapy, with a median HbA1c of 7% compared to a median HbA1c of 9% for conventional therapy. There was an adverse effect of intensive therapy, a threefold increased risk of hypoglycemia, but this was not associated with a decline in cognitive function or quality of life.[35] An observational follow-up was done to determine the durability of the DCCT effects on more advanced complications such as cardiovascular disease. The Epidemiology of Diabetes Interventions and Complications (EDIC) showed a stable effect of initial assigned therapies and reduction in severe complications and cardiovascular disease (CVD).[35] The clinical trial and observational study provided strong evidence of the effectiveness of intensive therapy in reducing long-term complications of diabetes such as adverse CVD outcomes.

The management of diabetes has changed over time, in part due to the introduction of various antihyperglycemic agents. The most predictable agent for patients with diabetes is insulin. In 1996, the first rapid-acting human insulin analog, insulin lispro, was approved though other forms of insulin were available much earlier.[36] Newer forms of insulin came in following years.[37] Metformin was introduced as an antihyperglycemic agent in 1959 but was not officially approved for use in the U.S. until the mid 1990s. Since its introduction, metformin has become one of the most widely used antihyperglycemic agents in the world.[36] Two common sulfonylureas agents, glipizide and glyburide, were introduced in the U.S. in 1984.[38] Another sulfonylurea, glimepiride, was released in 1995.[36] Thiazolidinediones (TZDs) were initially introduced to the U.S. market in 1996. Soon after the introduction, research linked TZDs to nonhyperglycemic issues, such as HF.[36] The first agent in the class meglitinides, which lowers

blood glucose levels by stimulating insulin release from the pancreas, was approved in the U.S. in 1997 with a second agent approved in 2000.[36,39] Dipeptidyl peptidase-4 (DPP-4) inhibitors, some of the first oral agents, were first introduced in 2006 when sitagliptin was approved.[40] This was shortly followed by Saxagliptin and linagliptin in 2009 and 2011, respectively.[36] Sodium glucose co-transporter 2 (SGLT2) inhibitors are a novel group of compounds that were first introduced in 2013 when canagliflozin was approved.[41] A second and third agent, dapagliflozin and empagliflozin, were approved in the U.S. in 2014.[36] Since the introduction of SGLT2 inhibitors, there has been growing evidence that they reduce cardiovascular events, including evidence of reducing risks of HF events by 25% to 40%, as well as renal events which may be of particular importance for patients with diabetes.[42–44]

Despite the development and introduction of new antihyperglycemic therapies, real-world prescription practices do not necessarily follow guideline-based recommendations, in part due to medication cost and provider and patient preferences. One study of 1,023,340 diabetic adults (aged 18-80 years) who recently initiated an antidiabetic drug explored temporal trends in prescribing.[45] Between 2005 and 2016, Montvida and colleagues found that metformin remained the most popular first-line agent and sulfonylureas remained the most popular second-line agent. The proportional share of metformin increased (60% to 77%) as the first-line therapy, while the proportional share of sulfonylureas decreased (60% to 46%) despite remaining the most popular second-line therapy throughout the study.[45] DPP-4 inhibitors and SGLT2 inhibitors grew in popularity as the second and third choice of therapy but it was so early in their introduction that it was expected to see them hold small proportional shares.[45] Another observational study examined antihyperglycemic therapies in a cohort of 4,970 adult patients with HF and diabetes from 152 U.S. sites in the Change the Management of Patients with Heart Failure (CHAMP-HF)

registry.[46] A key difference between this study and Montvida's study is the study period (2015-2017) includes only the years when all antihyperglycemic therapies were on the market. Vaduganathan and colleagues found that 46% of participants were taking one antihyperglycemic and 23% were taking two therapies. The therapies used by the largest proportion of participants to the smallest were metformin (39.6%), insulin (33.1%), sulfonylureas (23.9%), DPP-4 inhibitors (10.4%), GLP-1RA (4.0%), SGLT2 inhibitors (2.4%), and TZDs (1.6%).[46] The proportion of patients using each class of therapies did not vary substantially over the one-year follow-up. It was expected TZD use was low due it's increased risk of HF.[47] Neither of these studies examined therapies stratified by race or sex, which merits the need for further research.

The Role of Social Determinants in Diabetes Outcomes

Social determinants of health are increasingly being recognized for their role in the incidence of diabetes.[17] Agardh and colleagues conducted a systematic review and meta-analysis examining the associations between diabetes incidence and socio-economic position (SEP) worldwide.[48] SEP was measured by educational level, occupation, and income. From 23 studies, they found that low educational level (RR=1.44; 95% CI 1.28-1.51), occupation (RR=1.31; 95% CI 1.09-1.57), and income (RR=1.40; 95% CI 1.04-1.88) were associated with an increased risk of diabetes when compared to high levels of these determinants.[48]

Social determinants play a major role in access to health care. Health insurance has been found to be the strongest predictor of whether US adults have access to diabetes care.[49] In a cross-sectional study utilizing NHANES data, Kazemian and colleagues found adults who have health insurance coverage had higher odds of being linked to care (OR=3.96; 95% CI 2.34-6.69).[49] The American Diabetes Association conducted a study to quantify the health resource

use associated with diabetes.[50] They found that uninsured individuals have 60% fewer office visits with a physician, are prescribed 52% fewer medications, and have 168% more emergency department visits.[50]

There is growing evidence that differential access to health care and services is a contributing factor to the disparities in diabetes. A cross-sectional study utilizing the 2013 Medical Expenditure Panel Survey aimed to assess disparities in diabetes care.[51] Diabetes care was defined as adherence to recommendations for quality care (HbA1c measured twice yearly, yearly foot exam, yearly dilated eye exam, yearly blood cholesterol test, and yearly flu vaccination). Black participants were less likely to have at least two HbA1c tests (OR=0.59; 95% CI 0.40-0.88) and receive an annual flu vaccine (OR=0.68; 95% CI 0.49-0.93) compared to White participants. Those who were uninsured were less likely to receive care that aligned with the quality of care recommendations.[51] Canedo and colleagues found that lack of insurance coverage explained some of the racial disparities observed in quality care, and linked the importance of quality care and its impact on reducing rates of diabetes complications and mortality.[51]

Individual social determinants as well as access to care vary by geography, with disparities in access in many regions with the highest prevalence of diabetes and socioeconomic disadvantage.[52,53] A person's neighborhood has been shown to be associated with the prevalence of diabetes. Gaskin and colleagues used data from NHANES and the U.S. Census to determine the role of neighborhood poverty and racial composition on race disparities in diabetes prevalence.[54] They found that individual poverty increased the odds of diabetes in both Black participants and White participants. They also found that living in a poor neighborhood increased the odds of diabetes in nonpoor Black participants, poor Black participants, and poor White

participants.[54] In addition, resources available to people with diabetes are geographically disparate. A study examining the distribution of diabetes self-management education (DSME) programs in rural counties found that odds of having at least one DSME program is higher in counties with higher percent of the population with at least a high school education, lower percent of uninsured individuals, lower unemployment rate, and higher numbers of people with diabetes.[55]

Summary of Problem and Study Relevance

Previous literature has provided convincing evidence of the relationship between diabetes and HF, as well as the racial and ethnic disparities in disease, complications, and care. The interconnectedness of race, ethnicity, insurance status, neighborhood socioeconomic status (SES), therapies/treatments, and poor outcomes complicate the burden of diabetes and HF. Currently, there is little research that examines the association of social deprivation and mortality in individuals with both diabetes and HF. This study seeks to assess potential associations between insurance status, social deprivation, and mortality, while stratifying by race and sex. Doing so may provide insight into where interventions should occur to ensure healthcare professionals have the skills to assess social determinants of health and consider them in clinical care.

CHAPTER 3: Methods

Data Source

This study utilized the Emory Healthcare Clinical Data Warehouse (EHC-CDW), a repository that integrates patient data from electronic medical records across the Emory Healthcare system. The EHC-CDW contains data from multiple business and clinical applications, and some external benchmarking databases across the Emory Healthcare system. Available information within the EHC-CDW includes patient and provider data, diagnoses and procedures, laboratory results, medications, emergency data, and clinical documentation. For this analysis, data from Emory University Hospital (EUH) and Emory University Hospital Midtown (EUHM) were examined as they are equipped with general medicine, general cardiology, and advanced heart failure services. This study was approved by the Emory Institutional Review Board.

Study Population

The study population included all patients aged 18-100 years admitted to EUH or EUHM with a primary or secondary discharge diagnosis of acute heart failure (based on *International Classification of Diseases –Ninth or Tenth Revision, Clinical Modification* codes 428.x and I50.x) from January 1, 2010 to December 31, 2018. Patients who did not have diabetes were excluded (based on *International Classification of Diseases –Ninth Revision, Clinical Modification* codes 250.x and 251). Self-reported race-ethnicity were extracted from electronic medical record information in the EHC-CDW. Only Black and White patients were included. Patients with other race-ethnic identifiers were excluded due to limited numbers (N=1,143).

Outcome

The primary outcome of interest was all-cause death experienced before December 31, 2018. Deaths occurring outside of the hospital were ascertained through a variety of mechanisms. One mechanism is through information received from the National Death Index at pre-specified intervals. A second mechanism is Emory's patient death notification email address that family members or caregivers can provide information about a death. A third mechanism is through DCM Services for Healthcare, a private company that specializes in estate recoveries. DCM Services helps complete death ascertainment for patients followed in the system through weekly updates. Information received from DCM Services is validated by Emory Healthcare personnel.

Social Deprivation Index

The Social Deprivation Index (SDI) summarizes seven sociodemographic indicators taken from the U.S. Census American Community Survey to quantify the socioeconomic variation in health outcomes.[56] The SDI was developed through a factor analysis of the percentage of the population that lives in poverty, percentage with less than 12 years of education, percentage of single parent households, percentage living in rented housing units, percentage living in overcrowded housing units, percentage of households without a car, and percentage of non-employed adults under 65 years of age. For this analysis, SDI at the census tract level was used. Patient addresses were geocoded at street level accuracy using the U.S. Census Bureau's geocoder. Census tracts were used as proxies for neighborhoods due to similar physical and social features and characteristics.[57] SDI scores range from 1 to 100, with a higher score indicating greater census tract deprivation and more disadvantaged.

Covariates

Additional individual-level clinical factors and hospital characteristics at the time of index hospitalization extracted from the EHC-CDW were considered. These characteristics include age, insurance type, heart failure type (heart failure with reduced ejection fraction [HFrEF], heart failure with preserved ejection fraction [HFpEF], other), medical comorbidities (hypertension, chronic kidney disease, coronary artery disease, atrial fibrillation, chronic obstructive pulmonary disease, peripheral vascular disease, and cerebrovascular accident/transient ischemic attack), vital signs (systolic blood pressure, diastolic blood pressure, heart rate, and respiratory rate), laboratory values (serum sodium, creatinine, estimated glomerular filtration rate [eGFR], blood urea nitrogen, hemoglobin, hemoglobin A1c [HbA1c], B-type natriuretic peptide [BNP] and troponin [TNI]), antihyperglycemic therapies, year of index hospitalization, discharging specialty (cardiovascular, hospitalist/internal medicine, other), and hospital location (EUH and EUHM). For patients with missing eGFR values (N=4,608), the Modification of Diet in Renal Disease equation was used to derive eGFR from serum creatinine.[58]

Statistical Analysis

Baseline patient characteristics were derived from available data at the time of the index hospitalization for acute heart failure. Data are presented as mean (standard deviation) for normally distributed continuous variables, median (interquartile range) for non-normally distributed continuous variables, or count (percentage) for categorical variables. Baseline characteristics were compared using t-tests (normally distributed variables), Wilcoxon rank sum

tests (skewed continuous variables), or chi-square tests (categorical variables). Neighborhood SDI quartiles were analyzed as a categorical variable (least deprived quartile [1-41], quartile 2 [42-65], quartile 3 [66-85], and most deprived quartile [86-100]).

The risk of mortality over the 8-year study period was estimated in the sample stratified by race and sex. Unadjusted and adjusted risk ratios (RRs) of mortality comparing insurance status and social deprivation using log binomial regression. Model 1 adjusted for age and year of admission. Model 2 additionally adjusted for clinical characteristics at the time of index hospitalization (HF type, hypertension, coronary artery disease, chronic kidney disease, atrial fibrillation, chronic pulmonary disease, peripheral vascular disease, systolic blood pressure, diastolic blood pressure, heart rate, respiration, estimated glomerular filtration rate, blood urea nitrogen, hemoglobin, HbA1c, sodium, and antihyperglycemic therapies). Model 3 additionally adjusted for discharging specialty and hospital location. BNP and TNI were not included in analyses due to a large amount of missingness.

All statistical analyses were conducted using SAS statistical software version 9.4 (Cary, NC). All p-values are two-tailed with a significance threshold of <0.05.

CHAPTER 4: Results

Between January 1, 2010 and December 31, 2018, there were 44,333 patients discharged with a primary or secondary diagnosis with acute HF from two hospitals in the Emory Healthcare System. After excluding 26,258 patients with no diabetes or unknown diabetes status, 94 patients older than 100 years of age, 1,143 patients for racial identifiers besides Black or White, 3,061 patients with an eGFR less than 20 mL/min/1.73m^2 or missing, 2,806 patients missing SDI scores, and 392 patients missing information on important clinical covariates, the final analytic cohort included 10,598 Black and White patients discharged with HF with diabetes (**Figure 1**). Patient characteristics were largely similar between those included in the analysis and those excluded (**Table 4**). Overall, the mean age of the analytic cohort was 72 years, 47.4% were women, 55.0% were Black and 69.2% had Medicare insurance. Most clinical covariates differed by race when stratified by sex (**Table 1**). Of note, Black men and women were more likely to be younger, have a higher HbA1c level, have a higher Charlson comorbidity index, and have a higher SDI. The absolute risk of all-cause mortality was 20.8% for all patients analyzed (**Table 2**). The risk of mortality among women was 19.1% and the risk of mortality among men was 22.3%. There was no difference in mortality by race in men (p=0.069) or women (p=0.292).

Insurance Status

Table 3 shows the unadjusted and adjusted relative risks of mortality associated with insurance status and social deprivation by race and sex. The fully adjusted model accounted for demographics, medical comorbidities, and hospital factors. Among Black men, the relative risk of death comparing those with Medicare to those with private insurance was 1.22 (95% CI 0.93-1.60). The relative risk of death comparing those with Medicaid insurance to those with private

insurance was 1.17 (95% CI 0.80-1.71) among Black men. In White men, the associations were 1.22 (95% CI 0.95-1.55) and 0.99 (95% CI 0.60-1.63), respectively. None of these associations were statistically significant.

Among Black women, the relative risk of death comparing those with Medicare insurance to those with private insurance was 0.81 (95% CI 0.60-1.09). The relative risk of death was statistically significantly lower for those with Medicaid insurance compared to those with private insurance in Black women (RR=0.63; 95% CI 0.40-0.98). In White women, the associations were 0.74 (95% CI 0.53-1.05) and 0.53 (95% CI 0.26-1.06), respectively, comparing Medicare and Medicaid insurance status with private insurance.

Overall, there was no significant difference in mortality between those with Medicare insurance and private insurance in any of the race and sex stratifications. There was no interaction between race and insurance status for men (p=0.862) or women (p=0.477).

Neighborhood Deprivation

When examining the association of social deprivation and death , the relative risks of death comparing patients living in SDI quartile 2, quartile 3, and quartile 4 to patients living in SDI quartile 1 were 1.10 (95% CI 0.80-1.52), 1.06 (95% CI 0.77-1.45), and 1.05 (95% CI 0.78-1.43), respectively, among Black men. In White men, the associations were 0.86 (95% CI 0.71-1.04), 0.95 (95% CI 0.78-1.17), and 0.85 (95% CI 0.63-1.15), respectively, comparing patients living in SDI quartile 2, quartile 3, and quartile 4 to patients living in SDI quartile 1.

Among Black women, the relative risks of death comparing patients living in SDI quartile 2, quartile 3, and quartile 4 to those living in SDI quartile 1 were 0.96 (95% CI 0.69-1.32), 0.73 (95% CI 0.53-1.00), and 0.80 (95% CI 0.59-1.08), respectively. In white women, the

relative risk of death was significantly lower for patients living in SDI quartile 2 compared to patients living in SDI quartile 1 (RR=0.72; 95% CI 0.52-0.98), while the relative risks of death of patients living in SDI quartile 3 and quartile 4 were not significantly different from patients living in SDI quartile 1 (RR=0.86; 95% CI 0.64-1.17 and RR=1.13; CI 0.76-1.67, respectively).

Other than white women living in SDI quartile 2, there was no significant difference in mortality between SDI quartiles across all race/sex stratifications. There was no interaction between race and social deprivation for men (p=0.583) or women (p=0.069).

CHAPTER 5: Discussion and Recommendations

The purpose of this study was to examine the relationship between insurance status, neighborhood deprivation, and mortality among adults with diabetes and HF. It was hypothesized that underinsurance and neighborhood deprivation would increase the risk of death among patients hospitalized for HF with comorbid diabetes. In contrast to expectation, Black women with Medicaid insurance and White women living in SDI quartile 2 had a lower risk of mortality than Black women with private insurance and White women living in SDI quartile 1. There were no other significant differences observed between insurance status and mortality or social deprivation and mortality, accounting for patient demographics, medical comorbidities, and hospital characteristics. In addition, there was no evidence of interaction between race and insurance status or race and social deprivation.

Many observational studies have analyzed the association between insurance status and all-cause mortality.[59–62] Using data from NHANES, Bittoni et al found that those with public or no insurance had a 54% increased risk of mortality compared to those with private insurance controlling for demographic and inflammation-related lifestyle factors.[60] Song et al and Sorlie et al used data from national surveys and found that those with private insurance had a decreased risk of mortality and those with public insurance had an increased risk compared to those with no insurance.[61,62] Though the prior research by Song et al and Sorlie et al did not account for uninsured patients, our results differed from these studies in that we did not detect associations between insurance type and all-cause mortality among insured patients hospitalized with HF and DM. Unlike our patient population, these previous studies were conducted on healthy adults across the nation and included lifestyle factors and other demographics.

Previous studies have shown that socioeconomic factors, lifestyle factors, and accessing care when needed play an important part in the mechanism relating insurance status and mortality.[63,64] We did not have access to individual socioeconomic or lifestyle factors. Because our study focused on a patient population treated within the same healthcare system, any effects of differential access to care may have been was attenuated. This may explain why there was no measurable variation in risk of mortality by insurance status in our study.

Past studies that examined neighborhood deprivation and mortality have shown differing risks of mortality as those from the current study.[65,66] Using data from the Southern Community Cohort Study, Warren Anderson et al. found that men living in neighborhoods with the greatest socioeconomic disadvantage had a 9% increased risk of mortality, and women had a 26% increased risk of mortality.[65] In the NIH-AARP Study, Major et al found that men living in the highest quintile of deprivation had a 17% increased risk of mortality compared to those living in the lowest quintile of deprivation, and women had a 13% increased risk of mortality.[66] These were community-based cohort studies of the general population. Similar mortality risks have also been reported for HF patients. Bikdeli et al found the odds of mortality at 6 months was 0.75 (95% CI 0.48-1.17) when comparing living in low SES neighborhoods to high SES neighborhoods.[67] When examining death or readmission at 6 months, the odds of mortality increased to 1.50. The authors decided to consider a composite of death or readmission because of the potential for death to act as a competing event (i.e., less readmissions in case of more deaths). There is a possibility that some of our patients experienced the adverse outcome of readmission but not death, and this should be considered in future research.

There are limitations to this study worth noting. There was a lack of information on individual-level SES factors such as individual income, level of education, and occupation.

However, there is often a high degree of correlation between individual- and neighborhood-level SES measures, especially in urban areas.[68] Additionally, other clinical or behavior variables such as duration of diabetes, BMI, and smoking status that can impact mortality were not included in the analysis.[69] Also, patients with low eGFR (less than 20 mL/min/1.73 m^2)—who are at higher risk of death—were excluded from analysis. This eGFR criterion may have also led to the exclusion of patients with chronic kidney disease, an important consequence of diabetes as well as a strong predictor of HF outcomes. The exclusion of these patients may have contributed to our null findings. Finally, there was no consideration for cardiovascular specific mortality, or hospital readmissions as outcomes. Since hospital readmissions and mortality may have complex correlations in hospitalized patients with HF, they are often considered together in analyses.

Public Health Recommendations

Findings from this study have implications for future research and practice. Future research should not limit outcomes to solely mortality, as there are many care processes and health outcomes that arise from inequalities in a hospitalized population. Future investigation could examine racial disparities in quality of care and treatments, including antihyperglycemic therapies among the hospitalized population. There is a need to also examine hospital readmissions as an adverse event, alongside mortality, that may occur after an index hospitalization. To study short- and long-term outcomes following a hospitalization, future research should examine measures at 30-day and 1-year. Another layer of stratification by age should be considered due to the potential differences in the relationship between insurance status and death by age group. The relationships studied here can be further examined through a time-to-event analysis. If possible, future research should incorporate clinical covariates in addition to

lifestyle and individual-level socio-demographic factors. Despite the lack of significant results, it is still important to have conversations surrounding the role of social determinants in health outcomes in a clinical setting. Continued research in approaches to improve the health and clinical management of patients with HF and DM within social ecological context will benefit the population with these conditions.

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Tables and Figures

Table 1. Baseline patient characteristics at time of index hospitalization.

	Overall (N=10,598)	Men			Women		
		Black (N=2,626)	White (N=2,947)	P-value[a]	Black (N=3,206)	White (N=1,819)	P-value[a]
Age, years	72.2 ± 13.3	69.1 ± 13.1	74.1 ± 12.1	<0.001	71.5 ± 13.9	74.6 ± 13.1	<0.001
HF Classification				0.146			0.002
HFrEF	5215 (49.2)	1492 (56.8)	1625 (55.1)		1393 (43.5)	705 (38.8)	
HFpEF	2884 (26.8)	557 (21.2)	609 (20.7)		1059 (33.0)	619 (34.0)	
Other [b]	2539 (24.0)	577 (22.0)	713 (24.2)		754 (23.5)	495 (27.2)	
Hypertension	7964 (75.2)	1991 (75.8)	2041 (69.3)	<0.001	2609 (81.4)	1323 (72.7)	<0.001
Coronary Artery Disease	5913 (55.8)	1304 (49.7)	2195 (74.5)	<0.001	1401 (43.7)	1013 (55.7)	<0.001
Chronic Kidney Disease	4152 (39.2)	1288 (49.1)	1133 (38.5)	<0.001	1231 (38.4)	500 (27.5)	<0.001
Cerebrovascular Disease	2177 (20.5)	568 (21.6)	559 (19.0)	0.014	706 (22.0)	344 (18.9)	0.009
Atrial Fibrillation	3266 (30.8)	654 (24.9)	1317 (44.7)	<0.001	644 (20.1)	651 (35.8)	<0.001
Chronic Pulmonary Disease	3660 (34.5)	745 (28.4)	993 (33.7)	<0.001	1163 (36.3)	759 (41.7)	<0.001
Peripheral Vascular Disease	1849 (17.5)	419 (16.0)	689 (23.4)	<0.001	384 (12.0)	357 (19.6)	<0.001
Charlson Comorbidity Index	5.1 ± 2.9	5.5 ± 3.0	5.1 ± 2.8	<0.001	5.1 ± 2.8	4.7 ± 2.8	<0.001
Systolic Blood Pressure, mm Hg	143.1 ± 32.6	145.1 ± 34.6	134.9 ± 26.8	<0.001	151.5 ± 35.4	139.0 ± 28.6	<0.001
Diastolic Blood Pressure, mm Hg	76.8 ± 17.0	81.2 ± 18.8	72.8 ± 13.6	<0.001	79.9 ± 18.1	71.3 ± 14.1	<0.001
Heart Rate, beats per minute	85.1 ± 23.1	87.6 ± 21.8	81.0 ± 19.9	<0.001	88.1 ± 27.5	83.0 ±19.5	<0.001
Respiratory rate, breaths per minute	20.1 ± 9.4	20.1 ± 8.8	19.9 ± 9.5	0.405	20.3 ± 9.6	20.0 ± 9.6	0.280
Creatinine, mg/dL	1.2 (0.9-1.6)	1.4 (1.1-1.9)	1.2 (1.0-1.6)	<0.001	1.1 (0.9-1.6)	1.0 (0.8-1.3)	<0.001
eGFR, ml/min/1.73m^2	58.4 ± 23.3	60.6 ± 25.1	58.5 ± 21.1	<0.001	57.9 ± 24.4	56.0 ± 21.8	0.007

Blood urea nitrogen, mg/dL	20 (15-30)	20 (15-31)	22 (16-31)	<0.001	19 (14-28)	20 (14-30)	0.003
Hemoglobin, g/dL	11.8 ± 2.2	12.1 ± 2.4	12.2 ± 2.3	0.034	11.3 ± 2.0	11.4 ± 2.0	0.031
HbA1c, % [c]	6.9 (6.1-8.3)	7.1 (6.2-8.5)	6.9 (6.1-8.0)	<0.001	7.1 (6.2-8.6)	6.7 (6.0-8.0)	<0.001
Sodium, mEg/L	137.6 ± 4.3	137.5 ± 4.4	137.2 ± 4.1	0.009	138.1 ± 4.3	137.4 ± 4.2	<0.001
B-type Natriuretic Peptide, pg/mL [c]	370 (140-854)	396 (140-927)	389 (154-876)	0.825	336 (117-798)	361 (153-787)	0.060
Troponin I, pg/mL [c]	0.04 (0.01-0.14)	0.05 (0.02-0.13)	0.05 (0.02-0.23)	0.023	0.04 (0.01-0.10)	0.04 (0.01-0.19)	0.003
Discharging Specialty				<0.001			<0.001
Cardiovascular	4334 (40.9)	863 (32.9)	1754 (59.5)		824 (25.7)	893 (49.1)	
Internal Medicine	5025 (47.4)	1534 (58.4)	836 (28.4)		1990 (62.1)	665 (36.6)	
Other	1239 (11.7)	229 (8.7)	357 (12.1)		392 (12.2)	261 (14.3)	
Location of Admission				<0.001			<0.001
EUH Floor	3744 (35.3)	815 (31.0)	1204 (40.9)		947 (29.5)	778 (42.8)	
EUH ICU	1376 (13.0)	272 (10.4)	492 (16.7)		324 (10.1)	288 (15.8)	
EUHM Floor	4498 (42.4)	1261 (48.0)	1014 (34.4)		1606 (50.1)	617 (33.9)	
EUHM ICU	980 (9.3)	278 (10.6)	237 (8.0)		329 (10.3)	136 (7.5)	
Length of stays, days	6 (3-11)	6 (3-11)	6 (3-11)	0.222	6 (3-10)	6 (3-11)	0.007
Social Deprivation Measures							
Social Deprivation Index	67 (43-86)	77 (53-91)	51 (29-71)	<0.001	81 (59-92)	53 (31-73)	<0.001
SDI Quartiles				<0.001			<0.001
Q1	2419 (22.8)	353 (13.4)	1079 (36.6)		364 (11.4)	623 (34.3)	
Q2	2670 (25.2)	557 (21.2)	928 (31.5)		608 (19.0)	577 (31.7)	
Q3	2751 (26.0)	735 (28.0)	671 (22.8)		924 (28.8)	421 (23.1)	
Q4	2758 (26.0)	981 (37.4)	269 (9.1)		1310 (40.9)	198 (10.9)	
Insurance				<0.001			<0.001
Private	1957 (18.5)	543 (20.7)	644 (21.9)		471 (14.7)	299 (16.4)	
Medicare	7333 (69.2)	1641 (62.5)	2070 (70.2)		2243 (70.0)	1379 (75.8)	
Medicaid	831 (7.8)	248 (9.4)	112 (3.8)		371 (11.6)	100 (5.5)	
Other/Not Recorded	477 (4.5)	194 (7.4)	121 (4.1)		121 (3.8)	41 (2.3)	

HFrEF, heart failure with reduced ejection fraction, or systolic HF; HFpEF, heart failure with preserved ejection fraction, or diastolic HF; eGFR, estimated glomerular filtration rate; HbA1c, glycated hemoglobin; EUH, Emory University Hospital; EUHM, Emory University Hospital Midtown; ICU, intensive care unit; SDI, social deprivation index; Q1, lowest deprivation quartile; Q4, highest deprivation quartile
Values are mean ± standard deviation, median (interquartile range), or N (%).

[a] p-values compare statistical difference between characteristics by race for each sex separately
[b] HF other refers to ICD-9 and ICD-10 codes including HF unspecified, right heart failure, and high output HF
[c] Data missing as follows: n=4,083 HbA1c; n=4,266 B-type Natriuretic Peptide; n=2,914 Troponin

Table 2. Mortality in patients by race and sex.

		Men			Women		
	Overall (N=10,598)	**Black (N=2,626)**	**White (N=2,947)**	**P-value**	**Black (N=3,206)**	**White (N=1,819)**	**P-value**
Mortality risk in each group	2205 (20.8)	558 (21.3)	686 (23.3)	0.069	599 (18.7)	362 (19.9)	0.292

Values are N (%).

Table 3. Risk ratio of mortality associated with insurance status and social deprivation.

	Unadjusted RR (95% CI)	**Model 1 (95% CI)**	**Model 2 (95% CI)**	**Model 3 (95% CI)**
Black Men				
Insurance				
Private	Ref	Ref	Ref	Ref
Medicare	1.46 (1.19 – 1.79) [b]	1.22 (0.97 – 1.53)	1.19 (0.90 – 1.56)	1.22 (0.93 – 1.60)
Medicaid	0.94 (0.67 – 1.32)	0.93 (0.66 – 1.31)	1.11 (0.76 – 1.64)	1.17 (0.80 – 1.71)
SDI Quartiles				
Q1	Ref	Ref	Ref	Ref
Q2	1.10 (0.85 – 1.41)	1.06 (0.83 – 1.36)	1.16 (0.84 – 1.61)	1.10 (0.80 – 1.52)
Q3	0.97 (0.75 – 1.24)	0.94 (0.74 – 1.20)	1.04 (0.76 – 1.42)	1.06 (0.77 – 1.45)
Q4	1.01 (0.80 – 1.27)	0.97 (0.77 – 1.22)	1.05 (0.78 – 1.43)	1.05 (0.78 – 1.43)
White Men				
Insurance				
Private	Ref	Ref	Ref	Ref
Medicare	1.25 (1.05 – 1.49) [a]	1.17 (0.97 – 1.42)	1.22 (0.95 – 1.56)	1.22 (0.95 – 1.55)
Medicaid	0.99 (0.66 – 1.48)	0.99 (0.66 – 1.48)	0.92 (0.56 – 1.51)	0.99 (0.60 – 1.63)
SDI Quartiles				
Q1	Ref	Ref	Ref	Ref
Q2	0.91 (0.77 – 1.06)	0.89 (0.76 – 1.04)	0.84 (0.69 – 1.03)	0.86 (0.71 – 1.04)
Q3	0.87 (0.73 – 1.04)	0.88 (0.74 – 1.05)	0.95 (0.77 – 1.17)	0.95 (0.78 – 1.17)
Q4	0.81 (0.63 – 1.05)	0.84 (0.65 – 1.08)	0.84 (0.62 – 1.15)	0.85 (0.63 – 1.15)
Black Women				
Insurance				
Private	Ref	Ref	Ref	Ref
Medicare	1.28 (1.03 – 1.60) [a]	1.04 (0.82 – 1.33)	0.79 (0.58 – 1.06)	0.81 (0.60 – 1.09)
Medicaid	0.68 (0.48 – 0.97) [a]	0.69 (0.48 – 0.98) [a]	0.64 (0.41 – 1.01)	0.63 (0.40 – 0.98) [a]
SDI Quartiles				
Q1	Ref	Ref	Ref	Ref
Q2	0.82 (0.65 – 1.05)	0.87 (0.69 – 1.09)	0.91 (0.66 – 1.27)	0.96 (0.69 – 1.32)
Q3	0.68 (0.54 – 0.86) [a]	0.72 (0.57 – 0.90) [a]	0.71 (0.51 – 0.98) [a]	0.73 (0.53 – 1.00)
Q4	0.71 (0.57 – 0.88) [a]	0.73 (0.59 – 0.90) [a]	0.79 (0.58 – 1.08)	0.80 (0.59 – 1.08)

White Women				
Insurance				
Private	Ref	Ref	Ref	Ref
Medicare	1.02 (0.79 – 1.31)	0.94 (0.71 – 1.25)	0.74 (0.52 – 1.04)	0.74 (0.53 – 1.05)
Medicaid	0.70 (0.41 – 1.19)	0.70 (0.41 – 1.20)	0.51 (0.25 – 1.04)	0.53 (0.26 – 1.06)
SDI Quartiles				
Q1	Ref	Ref	Ref	Ref
Q2	0.84 (0.67 – 1.06)	0.85 (0.67 – 1.06)	0.72 (0.52 – 0.98) [a]	0.72 (0.52 – 0.98) [a]
Q3	0.84 (0.65 – 1.08)	0.85 (0.67 – 1.09)	0.84 (0.62 – 1.15)	0.86 (0.64 – 1.17)
Q4	0.99 (0.73 – 1.35)	1.03 (0.76 – 1.40)	1.09 (0.74 – 1.62)	1.13 (0.76 – 1.67)

[a] $p<0.05$
[b] $p<0.001$

Model 1 adjusts for age and year of admission.
Model 2 adjusts for Model 1 and HF type, hypertension, coronary artery disease, chronic kidney disease, atrial fibrillation, chronic pulmonary disease, peripheral vascular disease, systolic blood pressure, diastolic blood pressure, heart rate, respiration, estimated glomerular filtration rate, blood urea nitrogen, hemoglobin, HbA1c, sodium, and antihyperglycemic therapies
Model 3 adjusts for Model 1, Model 2, and discharging specialty and hospital location

SDI, social deprivation index; Q1, lowest deprivation quartile; Q4, highest deprivation quartile
RR indicates risk ratio; CI indicates confidence interval.

Table 4. Baseline patient characteristics who were included and excluded in the analysis.

	Included in Analysis (N=10,598)	**Excluded from Analysis (N=6,240)**
Age, years	72.2 ± 13.3	69.9 ± 13.6
Women	5025 (47.4)	3101 (49.7)
Black	5832 (55.0)	3868 (62.0)
HF Classification		
HFrEF	5215 (49.2)	2700 (43.3)
HFpEF	2884 (26.8)	1639 (26.3)
Other	2539 (24.0)	1901 (30.5)
Hypertension	7964 (75.2)	4275 (68.5)
Coronary Artery Disease	5913 (55.8)	3170 (50.8)
Chronic Kidney Disease	4152 (39.2)	3960 (63.5)
Cerebrovascular Disease	2177 (20.5)	1192 (19.1)
Atrial Fibrillation	3266 (30.8)	1600 (25.6)
Chronic Pulmonary Disease	3660 (34.5)	2055 (32.9)
Peripheral Vascular Disease	1849 (17.5)	1133 (18.2)
Charlson Comorbidity Index	5.1 ± 2.9	5.8 ± 2.9
Systolic Blood Pressure, mm Hg	143.1 ± 32.6	143.3 ± 36.1
Diastolic Blood Pressure, mm Hg	76.8 ± 17.0	76.1 ± 18.0
Heart Rate, beats per minute	85.1 ± 23.1	85.5 ± 22.3
Respiratory rate, breaths per minute	20.1 ± 9.4	20.1 ± 11.2
Creatinine, mg/dL	1.2 (0.9-1.6)	2.4 (1.1-5.2)
eGFR, ml/min/1.73m^2	58.4 ± 23.3	36.4 ± 30.3
Blood urea nitrogen, mg/dL	20 (15-30)	31 (19-52)
Hemoglobin, g/dL	11.8 ± 2.2	11.0 ± 2.3
HbA1c, % [a]	6.9 (6.1-8.3)	6.7 (5.8-7.9)
Sodium, mEg/L	137.6 ± 4.3	137.1 ± 4.6
B-type Natriuretic Peptide, pg/mL [a]	370 (140-854)	504 (182-1187)
Troponin I, pg/mL [a]	0.04 (0.01-0.14)	0.05 (0.02-0.17)
Discharging Specialty		
Cardiovascular	4334 (40.9)	1973 (31.6)

Internal Medicine	5025 (47.4)	3475 (55.7)
Other	1239 (11.7)	792 (12.7)
Location of Admission		
EUH Floor	3744 (35.3)	1986 (31.8)
EUH ICU	1376 (13.0)	858 (13.8)
EUHM Floor	4498 (42.4)	2738 (43.9)
EUHM ICU	980 (9.3)	654 (10.5)
Length of stays, days	6 (3-11)	6 (3-11)
Social Deprivation Measures		
Social Deprivation Index (SDI) [a]	67 (43-86)	74 (47-89)
SDI Quartiles [a]		
Q1	2419 (22.8)	510 (18.1)
Q2	2670 (25.2)	625 (22.2)
Q3	2751 (26.0)	807 (28.7)
Q4	2758 (26.0)	871 (31.0)
Insurance		
Private	1957 (18.5)	904 (14.5)
Medicare	7333 (69.2)	4587 (73.5)
Medicaid	831 (7.8)	487 (7.8)
Other/Not Recorded	477 (4.5)	262 (4.2)

HFrEF, heart failure with reduced ejection fraction, or systolic HF; HFpEF, heart failure with preserved ejection fraction, or diastolic HF; eGFR, estimated glomerular filtration rate; HbA1c, glycated hemoglobin; EUH, Emory University Hospital; EUHM, Emory University Hospital Midtown; ICU, intensive care unit; Q1, lowest deprivation quartile; Q4, highest deprivation quartile
Values are mean ± standard deviation, median (interquartile range), or N (%).

[a] Data missing from excluded as follows: n=2,641 HbA1c; n=2,932 B-type Natriuretic Peptide; n=1,888 Troponin; n=3,427 SDI

Figure 1. Flow diagram for study participants.