Heart Failure: A Severe Risk Factors in Women

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Abstract

Heart Failure (HF) hospitalizations are more common in women with Ischemia and No Obstructive Coronary Artery Disease (INOCA), which is mostly HF with intact Ejection Fraction (HFpEF) with INOCA and long-term prospective follow-up, we wanted to find predictors for the development of heart failure HF in a highly phenotypic population of women. The clinical history, medicines, physical exam, laboratory data, and angiographic data of women recruited in the NHLBI-sponsored Women's Ischemia Syndrome Evaluation (WISE) were all reviewed. We examined the relationship between baseline variables and the incidence of HF hospitalizations in 493 women with signs of ischemia but no obstructive coronary disease, no prior history of HF, and accessible follow-up data using a multivariate Cox analysis.

Keywords: Heart failure • Women's ischemia syndrome evaluation • Women

Introduction

In the United States, 5.1 million people suffer from Heart Failure (HF), with a 5-year death rate ranging from 50% to 70%. Heart Failure with Preserved Ejection Fraction (HFpEF) affects about half of all patients with HF, and it is more common in women than in males. Hypertension, diabetes, cigarette use, and coronary artery disease have all been identified as risk factors for HF in both men and women in previous research: HFpEF has arisen as a separate clinical entity from heart failure with decreased ejection fraction (HFrEF) in the last two decades, with rising incidence, prevalence, and distinctive risk factors. Unlike HFrEF, current HFpEF therapy are restricted and mostly focused on symptom management and the avoidance of related disorders. The variability and vast clinical spectrum exhibited in HFpEF are likely to blame for the challenge in developing guideline-directed therapy. Multiple risk variables have been discovered in various cohort studies, but the underlying mechanisms for the increased frequency of HFpEF in females are still unknown [1]. Obesity and a history of Coronary Artery Disease (CAD) without myocardial infarction were identified as HFpEF-specific risk factors in the Women's Health Initiative and Prevention of Renal and Vascular End-Stage Disease (PREVEND) study discovered a substantial link between the development of HFpEF and female gender, Atrial Fibrillation (AF), and age. Body Mass Index (BMI), Hypertension (HTN), diabetes, Chronic Kidney Disease (CKD), anemia, and statin usage were all identified as risk factors for HFpEF in the SCREEN-HF trial. Higher BMI, cigarette use, AF, and female gender all predicted HFpEF in the Framingham Heart Study [2].

Description

Ischemia with No Obstructive Coronary Disease (INOCA) is becoming more common, with half to two-thirds of women with angina who undergo coronary angiography for suspected ischemic heart disease having no obstructive coronary disease. INOCA can be caused by a variety of factors, including Coronary Microvascular Dysfunction (CMD), which is defined as aberrant dilatation and constriction of the heart's tiny arteries. Endothelial dysfunction, reduced nitric oxide bioavailability, and cardio myocyte damage induced by coronary microvascular dysfunction have all been linked to inflammation and myocardial stiffness in individuals with HFpEF, according to studies [3]. The Women's Ischemia Syndrome Evaluation (WISE) project, funded by the National Heart, Lung, and Blood Institute (NHLBI), looked at the aetiology and effects of ischemic heart disease in women. One of the most important results of WISE is that women with INOCA signs and symptoms frequently have CMD and are at a higher risk for Major Adverse Cardiovascular Events (MACE), the most common of which is HF hospitalization, which has been proven to be mostly HFpEF. In a previous WISE study, 25 (11%) of 223 women with indications and symptoms of ischemia who underwent coronary angiography had HF hospitalizations [4]. For 13 of the 25 cases, medical records were accessible. In these confirmed cases, the Left Ventricular Ejection Fraction (LVEF) was assessed and found to be maintained in 12/13 (92%) of the patients. The goal of this study was to look at classic and new baseline clinical variables in a cohort of extensively phenotypic women with INOCA who were followed prospectively for HF hospitalization for a long time. Our objective is to gain a better

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understanding of potential molecular pathways so that future therapy options for HFpEF in women can be identified [5].

During a 6 years follow-up, 18 (3.7%) of the women were admitted to the hospital with HF. Hospitalization for heart failure was linked to diabetes and cigarette usage. New predicted factors included greater resting heart rate, parity, and IL-6 levels, as well as worse Coronary Flow Reserve (CFR) and poor functional status, in a multivariate analysis correcting for established HFpEF predictors such as age, diabetes, hypertension, cigarette use, and statin usage. Traditional risk factors such as diabetes and cigarette use were linked to the development of HF in women with INOCA, according to the findings [6]. In addition, we discovered new independent risk factors for HF in our group, including increased parity and IL-6 levels, as well as low CFR and poor functional status. The greatest independent predictor of HF hospitalization was a low CFR consistent with CMD.

Conclusion

Our long-term prospective analysis of extensively phenotypic women with INOCA finds both established and unexpected risk variables for HF hospitalization. While further research is needed to corroborate these findings, both classic and new risk variables point to a pro-inflammatory environment and CMD as contributing factors to HFpEF development. The concept that structural pregnancyrelated recurrent ventricular remodeling may also contribute is supported by a female-specific feature of increased parity. Future research should focus on elucidating the processes that cause low CFR and HFpEF, as well as whether invasive and non-invasive CFR may be used as a surrogate disease marker for HFpEF. The combination of classic, innovative, and sex-specific risk variables discovered in our study serves as a foundation for the development of risk prediction tools for identifying high-risk women, as well as therapy targets for prospective therapies to reduce or prevent the development of HFpEF.

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