

# Associations among Obesity Degree, Glycemic Status, and Risk of Heart Failure in 9,720,220 Korean Adults (*Diabetes Metab J* 2020;44:592-601)

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Heart failure (HF) is a growing public health concern with increasing prevalence worldwide. The estimated prevalence of HF is estimated to be 1.53% in 2013, and is expected to double in 20 years in Korea [1]. Elevated body mass index (BMI) and insulin resistance are major risk factors for HF [2]. Recognition of individuals at risk for HF is critical because early treatment at the preclinical stage may prevent HF progression and improve outcomes.

Rhee et al. [3] analyzed the risk of HF according to baseline glycemic status and BMI in a Korean population using National Health Insurance Data. This nationwide population study showed a 1.86-fold increased risk of HF in diabetic patients compared to those with normoglycemia, which is consistent with previous data [4]. The incidence of HF increased with duration of diabetes. Risk of HF showed a J-shaped curve according to BMI regardless of baseline glycemic status. Those with class II obesity and those who were underweight had significantly increased risk for developing HF compared to normal and overweight groups. Rhee et al. [3] showed an overall trend among obesity degree, glycemic status, and HF in the Korean population. However, there are many unrevealed confounding factors that must be taken into account, such as sex, age, and subtypes of HF (preserved vs. reduced ejection fraction [EF], ischemic vs. non-ischemic, and chronic stable vs. acute decompensated). The results of this study raise important questions to be answered to appreciate clinical implications between BMI and HF, especially from therapeutic and prognostic perspectives.

HF is a clinical syndrome that results from structural or functional impairment of ventricular filling and/or ejection of blood [5]. HF is often classified according to EF; HF with reduced EF (HFrEF) vs. HF with preserved EF (HFpEF). The presenting symptoms of HFrEF and HFpEF are similar; however, the pathologic mechanisms are different, reflected by the finding that mortality reducing treatment in HFrEF failed to improve outcomes in HFpEF. A previous large population-based study from the United States showed that obesity and associated cardiometabolic traits increase risk of HFpEF compared to that of HFrEF [6]. Notably, there was a different effect of obesity on HF subtypes between men and women. Among men, higher BMI was independently associated with both HF subtypes (hazard ratio [HR], 1.34; 95% confidence interval [CI], 1.18 to 1.52;  $P < 0.0001$  for HFpEF) (HR, 1.24; 95% CI, 1.14 to 1.35;  $P < 0.0001$  for HFrEF). On the contrary, among women, BMI was associated with incident HFpEF but not HFrEF (HR, 1.38 per 1-standard deviation; 95% CI, 1.24 to 1.54,  $P < 0.0001$  for HFpEF) (HR, 1.09; 95% CI, 0.96 to 1.24;  $P = 0.18$  for HFrEF;  $P$  for difference = 0.01). The J-shaped incidence curve of HF described by Rhee et al. [3] could change when HF subtype or sex is taken into account.

The risk of HF associated with obesity seems to be modifiable, suggesting another important question. Is there a therapeutic role of weight loss in obese patients in prevention of HF? Recent studies from Sweden have demonstrated that weight loss over longitudinal follow-up is associated with a lower risk of HF by comparing risk of HF in obese patients with and

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without bariatric surgery [7]. Also, a recent meta-analysis by Reddy et al. [8] demonstrated a hemodynamic effect of weight loss intervention in obese patients without HF. Weight loss in obese patients was associated with significantly reduced heart rate ( $-9$  beats/min), mean arterial pressure ( $-7$  mm Hg), resting oxygen consumption, and reduced left and right heart filling pressures. This finding suggests that weight loss could attenuate hemodynamic derangement that can lead to HF, especially HFpEF. However, future prospective trials designed to evaluate the efficacy of weight loss intervention on modifying risk of HF are needed. Based on previous studies and growing prevalence of obesity and HF, more evidence is warranted to demonstrate the efficacy of weight loss intervention to prevent HF in different subgroups of the population. The previous study by Rhee et al. [3] provides a good starting point.

## CONFLICTS OF INTEREST

No potential conflict of interest relevant to this article was reported.

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