

Association between Non-Alcoholic Steatohepatitis and Left Ventricular Diastolic Dysfunction in Type 2 Diabetes Mellitus (*Diabetes Metab J* 2020;44:267-76)

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Non-alcoholic fatty liver disease (NAFLD) is the most prevalent liver disease, with an estimated prevalence of 20% to 30% [1]. NAFLD in type 2 diabetes mellitus (T2DM) is a common condition that can act synergistically to produce adverse outcomes [2]. NAFLD has subtypes from simple steatosis characterized by little to no inflammation to non-alcoholic steatohepatitis (NASH), characterized by hepatic steatosis accompanied by inflammatory activity, fibrosis progression, and cirrhosis [3,4].

Heart failure (HF) represents a major health burden, with a prevalence of more than 23 million patients and an annual medical cost of approximately 108 billion dollars worldwide [5]. Clinical interest in HF has traditionally focused on left ventricular (LV) systolic dysfunction [5].

In this article entitled, "Association between non-alcoholic steatohepatitis and left ventricular diastolic dysfunction in type 2 diabetes mellitus," Lee et al. [6] investigated the association between liver fibrosis and LV diastolic dysfunction in T2DM. They showed that LV diastolic dysfunction was significantly more prevalent in the NAFLD compared to the non-NAFLD group, and liver fibrosis was associated with LV diastolic dysfunction in patients with T2DM. These findings are consistent with previous reports that have shown an association between diastolic dysfunction and NAFLD characterized by impaired ventricular relaxation, increased myocardial thickness, and epicardial fat content [7-9].


However, there are issues not covered in this study. First, liver fibrosis was associated with diastolic dysfunction only in pa-

tients without insulin resistance. Insulin resistance is a key mechanism in NAFLD and T2DM. NAFLD is recognized as the hepatic component of metabolic syndrome, as these conditions share insulin resistance as a common pathophysiological mechanism. Therefore, NAFLD is strongly associated with T2DM and abdominal obesity [2,10]. Because NAFLD and diastolic dysfunction share multiple metabolic risk factors for cardiovascular diseases [11], several studies have suggested that NAFLD is associated with LV diastolic dysfunction [7,12]. However, why this result is meaningful only in patients without insulin resistance was not discussed in the present study. Second, there was no adjustment for duration of hypertension in this study. LV remodeling depends on age, type of hypertension, blood pressure values, and duration of hypertension [13]. Therefore, it is reasonable to apply duration of hypertension as an adjustment factor.

In my perspective, it would be interesting to evaluate if improvement in NASH fibrosis with weight loss or medical intervention may result in improved diastolic function in T2DM. Further studies are also needed to better understand the mechanisms by which diastolic dysfunction progresses with progression of NASH.

CONFLICTS OF INTEREST

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

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