When sweet becomes too sweet: Left ventricular longitudinal strain in focus

To the Editor,

Left ventricular (LV) remodeling in patients with diabetes mellitus has been recognized since a long time (1, 2). However, the influence of insulin resistance on cardiac remodeling has not been sufficiently investigated (3). Our study group showed that metabolic syndrome and prediabetes, conditions based on insulin resistance, were associated with LV functional and mechanical changes (4, 5).

Akavan-Khaleghi et al. (6) in this issue of the journal title provided a new piece of information regarding the influence of insulin resistance on LV longitudinal deformation. The authors reported no difference in LV global longitudinal strain (GLS) and systolic and diastolic strain rates between participants with glycated hemoglobin (HbA1c) level <5.7% and 5.7%–6.4% (6).

Several points of this investigation deserve to be commented. The authors included a limited number of participants with relatively small absolute difference in HbA1c, which made statistical analysis more difficult. Although the participants did not have coronary artery disease, they had many other potential confounding factors (obesity, hypertension, hyperlipidemia, and smoking) that could significantly deteriorate LV mechanics. Even in these circumstances, LV GLS showed a trend toward deterioration in participants with higher HbA1c (–16.1%±2.0% vs. –16.8%±2.4%). Moreover, LV structural parameters (wall thickness and LV mass index) and LV diastolic (e', E/e') and systolic (s') indices showed a trend toward LV remodeling in participants with higher HbA1c. The involvement of a control group with no cardiovascular risk factors would provide the missing information.

Ceyhan et al. (3) reported that LV GLS gradually decreased from controls, across participants with isolated increased fasting glucose, to participants with combined increased fasting glucose, glucose tolerance impairment, and diabetes mellitus. Interestingly, LV GLS significantly dropped when controls were compared with participants with isolated increased fasting glucose, whereas there was no significant difference between individuals with various levels of glucose intolerance (3). In other words, the most important feature that induced LV remodeling was the existence of insulin resistance but not its severity, which could partly explain the results of the study by Akavan-Khaleghi et al. (6).

Ceyhan et al. (3) found a significant association between HbA1c level, E/e', and peak early diastolic strain but not with LV GLS. Our investigation reported that LV mass index, LA longitudinal strain, and three-dimensional LV area strain were independently associated with HbA1c in the study population that involved prediabetic and diabetic patients (5). Akavan-Khaleghi et al. (6) did not find an independent association between LV GLS and HbA1c. However, the authors did not consider simple correlation analysis between HbA1c and LV GLS. Furthermore, the range of HbA1c in this study was narrow, which significantly reduced the chance to obtain statistical significance in correlation and regression analyses.

In conclusion, it should be emphasized that even though the present study did not show significant difference in LV longitudinal mechanics between participants with various HbA1c levels within the prediabetic range (mainly because of narrow HbA1c range and small sample size), one should be aware of the fact that LV remodeling not only occurs in prediabetic and diabetic patients but also significantly determines outcomes in these patients.

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