Left ventricular hypertrophy, inflammation, and insulin resistance

To the Editor,

I have read the article entitled "Relationship between extent and complexity of coronary artery disease and different left ventricular geometric patterns in patients with coronary artery disease and hypertension" by Uçar et al. (1) with great interest, which was published in Anatol J Cardiol 2015; 15: 782-8. In their study, the authors reported that the SYNTAX score is independently related with the left ventricular (LV) geometry in patients with hypertension and that LV remodeling is parallel to an increase in the extent and complexity of coronary artery disease (CAD).

Arterial hypertension with some nonhemodynamic factors, such as genetic, environmental, and metabolic factors, induce important structural changes in the ventricular myocardium. Among the metabolic factors, insulin resistance (IR) has been reported to be associated with the LV growth in patients with hypertension. Moreover, IR has been demonstrated to be a pathogenic cause that can predict the CAD occurrence (2, 3). Uçar et al. (1) reported that there is no information regarding plasma insulin levels. It would be helpful if the authors provided this information.

Finally, in the study by Uçar et al. (1), there are no data regarding the proinflammatory state of patients. LV hypertrophy is a low-level inflammatory state that may increase the risk of atherosclerotic heart disease. LV overload with an increased wall stress will result with a remodeling process, which is predominantly governed by various inflammatory cascades. Pathophysiology of the remodeling process includes increased proinflammatory cytokine expression, which is accompanied by leukocyte infiltration and proteolytic myocardial destruction by neutrophil originated enzymes (4, 5). Measuring IR and inflammatory marker levels could provide insights into the pathogenesis of different LV geometries and its relationship with CAD severity in patients with hypertension.

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Author`s Reply

To the Editor,

We thank the authors for their great interest in our work entitled "Relationship between extent and complexity of coronary artery disease and different left ventricular geometric patterns in patients with coronary artery disease and hypertension" that was published in the October 2015; 15: 789-794 issue of the Anatol J Cardiol (1). As reported, we found that the SYNTAX score is independently related with the LV geometry in patients with hypertension. Moreover, this result demonstrates that LV remodeling is parallel to the increase in the extent and complexity of CAD in our study patients (1). We discussed several mechanisms to explain the study results. We mentioned that in particular, the renin–angiotensin–aldosterone system can be the most important mechanism. Angiotensin II and angiotensin II type 1 receptor activation promote intracellular reactions that may lead to both cardiac hypertrophy and the progression of complex atherosclerotic lesions through the proliferation of vascular smooth muscle cells and the production of extracellular matrix protein (2). Furthermore, we discussed that oxidative stress contributes to the progression of atherosclerosis in patients with hypertension having different LV geometries (3).

As mentioned in the letter, IR and proinflammatory state have been reported to be associated with the LV growth and CAD in patients with hypertension (4, 5). However, we did not measure IR and any inflammatory marker. Furthermore, although we examined the hospital data, we did not find any values for these parameters. Measuring IR and inflammatory marker levels could provide insights into the pathogenesis of different LV geometries and its relationship with CAD severity in patients with hypertension. Further studies can be designed to determine the effects of IR and inflammatory markers for these patients.