Angiotensin-II induced insulin resistance

Resistência à insulina induzida por angiotensina-II

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We read with great interest the article by Lima-Martínez and cols. (1). They studied the relationship between epicardial adipose tissue (EAT) thickness and plasma levels of adiponectin in Venezuelan patients. And they found a significant association between EAT thickness and both metabolic syndrome components and adiponectin concentration. The authors also reported a strong correlation between left ventricular mass and EAT thickness. The article has important messages. But there are some items to be clarified.

Angiotensin II (AII), the major hormone of the renin-angiotensin system, plays an important role in the pathogenesis of hypertension and atherosclerosis. Evidence has suggested that AII impairs insulin sensitivity (2). Hypertensive subjects and animal models have shown improvements in insulin resistance in response to treatment with angiotensin I converting enzyme (ACE) inhibitors or AII type 1 receptor (AT1R) blocker (3). The exact mechanisms for the AII-induced insulin resistance remain largely unknown. But Ran and cols. previously showed that long-term AII infusion decreased the circulating adiponectin concentration without affecting the gene expression in rats, and this may facilitate the development of insulin resistance. And AT1R blocker ameliorated the AII-induced hypoadiponectinemia (4).

Left ventricular hypertrophy (LVH) is well known to be associated with increased cardiac risk. Regression of LVH over a period of a few months has been reported with ACE inhibitors and angiotensin receptor blockers (ARBs) (5). Regression of LVH continues gradually over time (three years or more) and may be associated with complete reversal of LVH and other abnormalities induced by hypertension such as left atrial enlargement and diastolic dysfunction (5).

Lima-Martínez and cols. mentioned that 16 out of 27 patients in their series were on ACE inhibitor or ARB therapy (1). Considering the above mentioned data, antihypertensive therapy may have influenced left ventricular measurements and plasma levels of adiponectin. For these reasons, the authors have better mentioned this point as a limitation of the study.

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