Abdominal Obesity and Arterial Stiffness: The Differential Role of Adipokines

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Abdominal obesity, a major public health problem, is an important risk factor for arterial stiffness, and both adiposity and arterial stiffness are independently associated with increased cardiovascular risk. Recently, increasing attention is paid to adipokines, a family of polypeptides mainly produced by adipose tissue–related leukocytes and mononuclear cells. Thus, many studies have reported an association of adipokines with other factors known to modulate arterial stiffness such as inflammation, sympathetic activation, hypertrophy, and proliferation of vascular smooth muscle cells. Focusing in the clinical setting of essential hypertension, an unfavorable effect of hypoadiponectinemia and increased leptin levels on aortic stiffness has been demonstrated, although data regarding resistin are lacking.¹

In this issue of the American Journal of Hypertension, Windham et al. examined the influence of adipokines leptin, adiponectin, or resistin, independent of other factors known to affect vascular stiffness, on the relationship between abdominal adiposity (by means of dual-energy X-ray absorptiometry) and carotid–femoral pulse wave velocity (PWV) using a mediation pathway approach.² The study confirms the relationship between abdominal adiposity and adipokines levels, as well as the associations between leptin and adiponectin with PWV. Particularly, the relationships between adipokines and PWV did not vary by amount of abdominal adiposity. Notably, the relationship of abdominal adiposity with arterial stiffness was completely eliminated when leptin was included in the analysis. In contrast, neither adiponectin nor resistin signifi­cantly altered the relationship between arterial stiffness and adiposity.

Noteworthy, this was the first study to examine the association between resistin and PWV, finding the two to be inversely related. This is in contrast with other reports of resistin as a potential mediator in cardiovascular disease, potentially through inflammatory pathways. Associations of resistin with carotid atherosclerosis as well as with the presence and severity of coronary artery disease and chronic kidney disease support even more the active role of resistin in the mani­festation of atherosclerotic cardiovascular and renal damage.³–⁵

In order to explain this discordance, a feedback mechanism in which increasing arterial stiffness signals a reduction in resistin to avoid further stiffening and/or a type I error have been proposed by the authors.

The cross-sectional nature of the study, the inclusion of participants with a broad age range and wide spectrum of cardiovascular disease, as well as the lack of data regarding the specific type of antihypertensive medications and hypertension control represent potent limitations. The statistical methodology used, although complex, may compensate for the limitations imposed by the cross-sectional nature of the study providing novel insights in the examination of the influence of adipokines on the relationship between abdominal adiposity and PWV. In addition, measurement of abdominal adiposity through dual-energy X-ray absorptiometry and arterial stiffness by carotid–femoral PWV using the Complior SP device (Artech Medical, Pantin, France) strengthens this study.

In conclusion, leptin explains in part the observed relationship between abdominal adiposity and arterial stiffness. It is speculated that the changes in arterial stiffness that accompany the changes in weight could be also explained by leptin. Future research focusing on large selected populations will elucidate the potential role of resistin in the pathophysiology of arterial stiffness.

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