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LETTER TO THE EDITOR

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Renal resistive index: Beyond the hemodynamics

We read with great interest the original article entitled "*Prognostic significance of the renal resistive index in the primary prevention of type II diabetes*" by Delsart et al[1] In this issue of *The Journal*, the authors interestingly underlined the prognostic value of renal resistive index (RRI) in diabetic subjects and its role as an independent predictor of a first cardiovascular or renal event.

Similar conclusions were previously obtained by others in different subsets of patients (hypertension, chronic kidney disease, or heart failure),[2-4] in which intrarenal hemodynamic alterations showed to be independent predictors of cardiovascular events.

It is likely that the mechanisms underlying renal hemodynamic alterations in diabetics are similar to those involved in hypertensive subjects and that "*renal resistance indices reflect systemic rather than renal vascular damage*," as previously postulated by Heine et al.[5]

In this article, Delsart et al emphasize the idea that RRI is influenced by upstream hemodynamic factors such as left ventricular ejection fraction or heart rate,[1] which both induced modifications on intrarenal circulation. However, although systemic hemodynamics certainly affects kidney, it is vascular damage that plays a major role in determining direct or indirect RRI changes, being RRI itself a marker of morphofunctional vascular impairment rather than a mere "victim" of upstream hemodynamic factors: In other words, the vascular damage represents a "*passepartout*" that allows systemic hemodynamic alterations to impact on intrarenal circulation.[6-8]

According to this hypothesis, Tublin et al[9] observed in experimental studies that RRI was dependent on vascular compliance rather than on hemodynamic changes, and a close link between RRI and pulse wave velocity (a well-known measure of arterial stiffness) was observed by our group in 264 hypertensive patients regardless of renal function[10]: The reduced elastic properties of large vessels might predispose the renal circulation to a greater hemodynamic burden.[6,10] Through histological studies, Ikee et al observed structural impairment also in renal small vessels (hyalinosis, intimal thickening, and increased intima/media ratio) in subjects with higher RRI.[11] Moreover, in the abovementioned study a positive correlation between histopathologic findings of renal inflammation and ultrasonographic RRI was reported in patients with chronic kidney disease: The inflammatory state might determine endothelial dysfunction, so promoting atherosclerosis (in situ and systemic structural impairment) and changes in autoregulation of intrarenal vessel motility, thus predisposing the kidney to hemodynamic damage.[6,11]

In this regard, RRI also proved to be correlated with choroidal vasculature and choroidal thickness[12] as well as with carotid intima-media thickness in several studies,[13,14] being the renal hemodynamic alterations able to occur at a very early stage in the temporal evolution of atherosclerotic damage. Recently, we demonstrated that intrarenal hemodynamics was associated with coronary atherosclerotic burden assessed by coronary angiography in patients with hypertension with mild coronary disease[15]; however, to prove the above, this relationship was independent by left ventricular ejection fraction and was not affected by other hemodynamic factors as heart failure.

In conclusion, the complex connections between macro- and microcirculation, as well as hemodynamic burden and vascular damage, suggest the presence of a dynamic integration of which RRI is an important crossroads, and this might explain its role as an overall prognostic predictor of cardiovascular events also in diabetic population.

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