Editorial

From Arterial to Cardiac Dysfunction

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Razik et al,[1] in this issue of *Angiology*, provide an interesting contribution with regard to the relationship between arterial and cardiac dysfunction in patients with premature coronary artery disease (CAD). Briefly, cross-sectional aortic distensibility, an index of local arterial stiffness, was lower at baseline in patients with CAD who developed cardiovascular (CV) events. This finding is in agreement with the concept that increased arterial stiffness is a CV risk factor. Moreover, a significant negative correlation between severity of CAD (calculated using the SYNTAX score) and distensibility of the aortic root and descending aorta was reported by Razik et al.[1] In presence of increased aortic stiffness, the chronic decrease in coronary artery perfusion pressure and blood flow during diastole may lead to flow-mediated decreases in shear stress and inward arterial remodeling (narrowing of lumen diameter).[2] Arterial diameter is dependent on several dynamic factors, among which are the level of vascular tone in the short term and vascular remodeling in the long term.[3] Therefore, the presence of both CAD and increased aortic stiffness can be a further limiting factor for coronary artery perfusion predisposing to myocardial ischemia.

Increased arterial stiffness mediates, in part, the effect of several pathological (eg, CV risk factors,[4] chronic kidney disease,[5] and chronic inflammatory diseases[6]) and physiological conditions (eg, the aging process) on heart and CV events. Considering that in the Razik et al study,[1] patients with CAD with known CV risk factors (creatinine clearance <30 mL/min, diabetes, hypertension, previous coronary artery bypass graft, significant hypercholesterolemia, aortic regurgitation, and aortic aneurysm) were excluded, other factors may have played a role in the development of aortic stiffening and CAD in this setting. A contribution of subclinical chronic inflammation[7] in these processes cannot be excluded. Unfortunately, no marker of inflammation was reported by Razik et al.[1]

The article by Razik et al[1] is of interest for the technique and the site used for the determination of local distensibility. Pulse wave velocity (PWV) at the level of the aorta (carotid-femoral PWV, aortic stiffness) is a validated method for the measurement of regional arterial stiffness in clinical practice.[2,8] Using this method, the mean stiffness of the arterial segment can be obtained. However, the viscoelastic properties change from the ascending to the descending aorta, as reported in the controls enrolled in the Razik et al study.[1] Moreover, arterial applanation tonometry does not allow to study the aortic arch, and this segment makes an important contribution to total arterial compliance.[9] To overcome these limits, the study of local arterial stiffness in different aortic sections can be helpful. According to current guidelines,[8] echo tracking devices are frequently used to calculate several local stiffness parameters in superficial muscular and elastic arteries (ie, brachial, common carotid, and femoral artery). However, aorta and deep arteries are not explorable with these devices.[8] Therefore, Razik et al[1] measured systolic and diastolic aortic area with magnetic resonance imaging (MRI) and brachial pulse pressure with a sphygmomanometer and calculated cross-sectional aortic distensibility. One of the main advantages of the use of MRI for the calculation of local arterial stiffness is that this technique allows the study of both superficial and deep arteries, including the aorta in its main sections (including the aortic arch).

Thanks to their technique, Razik et al reported that local arterial stiffness in patients with premature CAD was not uniformly altered in the main sections of the aorta (reduced distensibility at the level of aortic root and descending aorta but not of ascending aorta).[1] Moreover, at baseline, patients who developed an event during follow-up had lower aortic distensibility at the level of the 3 aortic sections whereas those without events had an increased ascending aorta distensibility. These findings suggest that an increased ascending aorta distensibility may offset the effects on heart of reduced aortic root and descending aorta distensibility in patients with CAD. Further studies are needed to confirm this hypothesis.

A limitation of the Razik et al study[1] is that peripheral pulse pressure (and not local pulse pressure) was used for the calculation of aortic distensibility. Considering that central and peripheral blood pressure are not perfectly interchangeable for the pulse wave amplification phenomenon (pressure wave is higher in peripheral arteries than in central elastic arteries because reflection sites are closer to the former arteries[10]), Razik

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et al[1] might have slightly underestimated the aortic distensibility in patients with CAD and even more so in controls. Therefore, the difference in aortic distensibility between controls and patients with CAD might have been even higher.

Future studies with MRI could be designed using a more accurate noninvasive technique for the estimation of local pulse pressure (eg, applanation tonometry of the radial artery paired with a validated transfer function processing).[8] The link between increased aortic stiffness and CV events is widely recognized but only one study reported that the improvement in aortic stiffness independent of blood pressure changes is associated with a reduction in CV events.[11] Therefore, future studies should be designed to assess whether improvement in aortic function is associated with better prognosis in patients with premature CAD beyond the impact of blood pressure lowering alone.

Authors' Note

All authors have contributed significantly to the submitted work in conception and design, manuscript writing, and have read and approved the submission of the manuscript; the manuscript has not been published and is not being considered for publication elsewhere, in whole or in part, in any language.

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