Evaluation of aortic stiffness to predict and prevent the risk of atrial fibrillation in hypertensive patients in their 50’s

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Pulse wave velocity measurement (PWVM) is the current “gold standard” for assessment of aortic stiffness in hypertensive subjects. (1) PWV is included amongst the risk factors for cardiovascular disease (2) and is strongly predictive for cardiovascular events in hypertensive individuals. (3) An increase in aortic stiffness may increase the risk of stroke through several mechanisms such as an increase in central pulse pressure or an increase in carotid intima-media thickness, promoting the development of atherosclerotic lesions and thus the likelihood of plaque rupture.

In this issue, Lantelme et al. (4), propose an exciting, alternative physiopathological approach. In agreement with the precedent hypothesis, aortic stiffness is able to influence the diameter of the left atrium and expose the patient to embolic stroke by increasing their risk of atrial fibrillation (AF). Indeed, this study shows that in a hypertensive population recruited in their 50’s, to a referring center in arterial hypertension, there was a significant relationship between the diameter of the left atrium and aortic stiffness (estimated from PWV measurements and humeral pulse pressure). This relationship is independent from other confounding factors, particularly cardiac remodelling.

The interdependence between elastic aortic properties and the left ventricular mass is well established particularly with relative wall thickness, this relationship remains independent from mean arterial pressure. So the expected correlation is again found between the dimension of the left atrium and the ventricular geometry, expressed as the relative wall thickness (RWT) and the left posterior wall thickness measured in diastole (LPWT).

As suggested by P. Gosse and M. Safar in view of a common embryological origin, the aorta may be considered, along with the left atrium and ventricle, as the third chamber of the left sided cardiac pump transforming the systolic output of the left ventricle into a continuous flow. (5)

Although the physiopathological support for this work is attractive, several limitations which are underlined by the author, need to be discussed before considering the therapeutic angle. Correlations are established by accepting “surrogate criteria”; the diameter of the left atrium for AF, pro-BNP concentration for left ventricular diastolic function, peripheral pulse pressure (PP) for central pulse pressure.

The hypothesis of the role of inflammation, raised in the literature seems even more questionable and largely speculative at this point to explain the risk of AF.

The pulse pressure, and as such arterial stiffness, is closely associated with inflammation, as expressed by CRP (6) or other cytokines, and raises further questions to the development and the extent of atherosclerosis rather than on the cause of atrial fibrillation.
The study illustrates the importance of considering target organ damage at the sub-clinical stage in the hypertensive patient and reopens the question of a potential benefit of anti-hypertensive agents beyond reduction in arterial blood pressure. (7)

The favourable impact of antihypertensive treatment on aortic stiffness may contribute to reduce the extent of atherosclerosis, but also prevent the risk of the occurrence of AF. (8)

According to recent clinical trials, the blockade of the renin-angiotensin system (RAS) should be first-choice agents, whose efficacy remains to be established prospectively by targeting the relationship between aortic stiffness and the left atrium.

References


