Aortic pulsatility drives microvascular organ damage in essential hypertension: New evidence from choroidal thickness assessment

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COMMENTARY

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Aortic pulsatility drives microvascular organ damage in essential hypertension: New evidence from choroidal thickness assessment

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1 | INTRODUCTION

In a healthy cardiovascular system, the compliant properties of the large arteries ensure that pulsations in pressure and flow generated by cyclic left ventricular contraction are dampened at the site of the ascending aorta into a continuous pressure (and flow) downstream at the site of arterioles. This allows for the delivery of a steady flow of blood during organ perfusion, and the microvasculature of target organs is protected from the damaging effects of pressure pulsatility.[1] The dampening of the pressure/flow is achieved via the windkessel effect whereby the aorta expands during systole and temporarily stores a portion of the stroke volume, which is then propelled into the systemic circulation during diastole via recoil of the elastic arterial wall. However, in response to aging, hypertension, and other disease states, arterial stiffening limits the buffering capacity of the elastic arteries. The wear-and-tear of elastic fibers in the aortic wall, due to the mechanical stress induced by each heartbeat, is amplified by high blood pressure.[2] This results in increased transmission of pulsatile pressure/flow to the microvasculature of target organs, which may lead to capillary rarefaction, ischemia, and ultimately, target organ damage.[3-6]

Microvascular damage in hypertension has been demonstrated by invasive techniques in humans, mostly as increased media-lumen ratio, the so-called eutrophic remodeling.[7,8] However, it is now possible to image directly and non-invasively the microcirculation in the retina.[9] Non-invasive near-histological analysis of retinal microcirculation confirmed in vivo the impact of systolic and pulse pressure on arteriolar wall thickness and wall cross-sectional area, suggesting arteriolar hypertrophy as a consequence of an increased pulsatile stress.[10]

More recently, novel accurate and non-invasive techniques have allowed evaluating the anatomy and function of another important vascular layer: the choroid. The inner retinal and choroidal vasculature are completely independent vascular systems, and present specific anatomical and physiological peculiarities. The anatomical configuration of the choroid is very intricate, including up to 5 separate layers, containing different types of vessels: From the terminal branches of posterior ciliary arteries, some "feeder" arterioles are connected to the choriocapillaris in a triangular pattern, making of choroidal vasculature an end-arterial circulation.[11] As a result of this complex organization, the choroid received 80% of blood supply from systemic circulation, compared to 5% of the retina; furthermore, the choroidal vasculature is less protected and autoregulated than the retinal one.[12] Thus, it is conceivable that choroidal vasculature is largely affected by the pulsatility transmitted by large vessels.

This hypothesis is confirmed by Mulé and coauthors in the present issue of Journal of Clinical Hypertension.[13] In this paper, Mulé explored choroidal thickness in 155 hypertensive patients undergoing a 24-hour ambulatory blood pressure monitoring for the evaluation of central and brachial 24 hour-pulse pressure.[14] Patients with a central pulse pressure > 35 mm Hg exhibited a thinner choroidal thickness. These individuals were older, had a worse renal function, and showed higher systolic central and peripheral blood pressure. However, pulse pressure (central > peripheral) was the only predictor of choroidal thickness impairment, independent of confounders.

In this study, choroidal thickness was evaluated by optic computed tomography-angiography, one of the most innovative techniques for the evaluation of microvascular flow in deeper retinal structures.[9,15] However, some technical limitations of the technique need to be acknowledged. It is still difficult to distinguish the many vascular contributors of this complex vascular layer, even with ameliorated techniques as the swept-source used in the study. Furthermore, signal attenuation may be difficult to overcome when it comes to the detailed structural analysis. This lack of specificity makes it difficult to clearly define physiopathological mechanisms underlying changes in choroidal thickness in hypertension, which may be induced either by capillary rarefaction (as demonstrated in the retina[16]) or by small muscular artery vasoconstriction from the deeper vascular layers, as well as by non-vascular smooth muscle contraction. Neighbor structures as the retinal pigment epithelium, involved in the crosstalk with choriocapillaris, may also play a role.[17]

In conclusion, the provided results indicate a strong association between 24-hour central pulse pressure and choroidal thickness in essential hypertensive patients. Large population studies have shown that choroidal structural remodeling is largely influenced by aging.[18] These results shed a new light on this phenomenon, indicating that age-induced changes in central hemodynamics are most likely responsible for choroidal thickness reduction. Further research is needed to elucidate the mechanisms responsible and the anatomical structures involved in the hypertensive choroidal phenotype.

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