Commentary: Cardio-ankle vascular index: The how and why for an ophthalmologist

Arterial stiffness is a well-known indicator of cardiovascular function. It implies reduced contractility and distensibility of the arterial wall in response to pressure changes. Arterial stiffness can be affected by both *atherosclerosis*—primarily affecting the arterial intima resulting in increased intima-media thickness secondary to deposition of atherosclerotic plaques; and *arteriosclerosis*—an age-related process of medial degeneration that involves increase in collagen and calcium deposition along with fragmentation of elastin lamellae in the medial layer.[1] With the growing interest in arterial wall properties in the pathogenesis of various cardiovascular diseases, many methods have been developed to measure arterial stiffness. The current gold standard is the pulse wave velocity (PWV), which is the velocity of propagation of a pressure wave along the arterial tree, calculated by dividing the distance between the two points by the time taken to cover the distance. Carotid-femoral PWV is the most commonly used reference and has been shown to be an independent predictor of cardiovascular mortality and morbidity. However, an innate drawback of using PWV is its dependence on the pressure of blood exerted on the arterial wall. Hence, the measured stiffness can change even without a concomitant structural change due to changes in blood pressure.[2]

To overcome this challenge, cardio-ankle vascular index (CAVI) was introduced as an indicator of arterial stiffness.[3] CAVI is measured by recording the distance between the level of the aortic valve (brachial level) to the measuring point (ankle) and the time delay between the closing of the aortic valve to the detected change in the arterial pressure wave at the measuring point. Data required to compute CAVI, including PWV, systolic and diastolic blood pressure as well as arterial pulse waveforms, are obtained through electrocardiogram, cardiac phonogram, and the pressure cuffs at the reference points. A CAVI of less than 8 is considered normal, between 8 and 9 as borderline and equal to or more than 9 is considered as highly suspicious of arteriosclerosis.[1-3] There has been increasing interest in the role of CAVI in predicting cardiovascular events in not just patients with known cardiovascular risk factors but also the healthy population. However, given that the measurement of CAVI takes place in the larger vessels including the aorta, femoral and tibial arteries, its correlation to the changes happening in the microvasculature of end organs like the eye cannot be directly extrapolated.

The literature on the application of CAVI in predicting the risk of ocular vascular disease is limited. Shiba *et al*. studied the optic nerve head (ONH) flow using laser speckle flowgraphy and demonstrated a positive correlation between CAVI and changes in the ONH blood flow.[4] Diabetes is an important condition which affects both the large and small vessels in the body. Researchers have shown that CAVI is higher in younger patients with diabetic retinopathy (DR) in comparison to those with without DR. Furthermore, the derangement of CAVI correlated with the severity of DR.[5] Kim *et al*. studied the retinal and choroidal microvasculature in eyes with DR and deranged CAVI. They found a significant correlation between arteriosclerotic changes in large vessels and choroidal vascular changes in DR.[6] Giving further push to the importance of arteriosclerosis over atherosclerosis in the pathogenesis of ocular disease, Taniguchi *et al*. demonstrated a positive correlation between CAVI and exudative age-related macular degeneration in comparison to atherosclerotic changes like carotid intimal plaques.[7] Branch retinal vein occlusion (BRVO) is another ocular disorder in which the arterial wall stiffness plays a direct role in the pathogenesis of the disease. The study by Kocayigit *et al*. further adds credence to this hypothesis by demonstrating increased CAVI in patients with acute BRVO in comparison to age-matched controls with similar risk factors like hypertension.[8]

In conclusion, CAVI appears to be a novel noninvasive surrogate marker of predicting risk of various ocular vascular diseases. However, the current evidence is based on cross-sectional studies with no temporal risk association. Prospective, randomized, and longitudinal studies are required to validate the ability of CAVI in predicting the risk of ocular complications in individuals with systemic risk factors.

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