

## Coronary vascular age comes of age

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The majority of cardiovascular risk estimates are strongly influenced by chronological age and, starting from 50 years, age turns out to be the predominant cardiovascular risk factor.<sup>1</sup> The foremost explanation of this evidence is the progressive accumulation of atherosclerotic plaques over time, and the use of chronological age in cardiovascular risk assessment is a surrogate for atherosclerotic burden. If the cardiovascular risk estimate is primarily driven by age, a young person with a significantly elevated risk factor burden is still likely to have a low risk score. To overcome the short-term estimate pitfalls, it has been proposed to use a time horizon much longer than 10 years in risk calculation, such as the 30-year risk, or even the lifetime risk, instead of the conventional 10-year risk.<sup>2</sup> Specific score vascular age charts useful for the full range of absolute risk and age have also been proposed.<sup>3</sup>

As the atherosclerotic burden of individuals with the same chronological age can be considerably different, due to contribution of the other traditional risk

factors,<sup>4</sup> a technique that better measures atherosclerotic impairment could represent a helpful clinical tool.<sup>5</sup> Two of the most promising procedures for the evaluation of subclinical atherosclerosis are coronary artery calcium (CAC) scanning by computed tomography (CT) and carotid intima-media thickness (CIMT) assessment by B-mode ultrasonography.<sup>6,7</sup>

“Vascular age,” also known as arterial age, heart age, or cardiovascular risk age, is an emerging concept based on the theory that the conversion of chronological age to an age derived from vascular imaging data would lead to a more accurate assessment of individual cardiovascular risk. CAC score and CIMT assessment can be used to define vascular age. In particular, CIMT vascular age is determined by linear regression modeling using published nomograms of CIMT percentiles or the age at which the individual’s measurement would represent the 50th percentile of a reference database. Conversely, the CT approach provides a convenient transformation of CAC score from Agatston units in years. It has been reported that CAC and CIMT provide similar information in the assessment of vascular age.<sup>8</sup> On the other hand, the rationale to use these tools in cardiovascular risk prediction models is still debated. Vascular age may contribute to a superior understanding of long-term cardiovascular risk especially in young adults, but despite the growing interest in the clinical utility of this novel concept, only few data are available.<sup>9</sup> In particular, the impact of vascular age to predict myocardial ischemia has not been evaluated and there is the need to investigate the potential role of vascular age in the prediction of stress-induced myocardial ischemia in patients with suspected coronary artery disease.

Coronary calcium accumulation is the result of an active process related to atherosclerosis, and thus detection of coronary artery calcifications corresponds to an atherosclerosis advancement report. The strong predictive value of calcium deposit evaluation, regardless of the risk category estimated by traditional risk scores, has been widely demonstrated.<sup>10–12</sup>

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The emerging idea to assign a vascular age, according to observed CAC, shoots for taking into account the relationship between calcium deposit process and actual obsolescence of vascular system, tailoring individual cardiovascular risk aside from chronological age.<sup>13</sup>

Prognostic data from MESA<sup>14,15</sup> showed that the risk associated with vascular age was a much stronger predictor of cardiovascular events than predictions based on chronological age. Moreover, in the same cohort, chronological age did not provide additional information after controlling for vascular age. Other investigations showed that there is a significant inverse relationship between telomere length and coronary artery calcifications in individuals with no clinical history of CAD suggesting a potential association of telomere length, a marker of biological aging, with cumulative lifelong burden of oxidative stress detected with coronary artery calcification increase.<sup>16,17</sup>

Yet, vascular age would be a useful tool to communicate test results to patients and to apply stricter therapeutic strategies to freeze cardiovascular disease progression in patients with an vascular age higher than the respective chronological age. Indeed, the communication of a given vascular age would have a superior emotive impact improving observance of therapies and healthier lifestyles.

## Disclosure

*The authors have indicated that they have no financial conflict of interest.*

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