The overlap between atherosclerotic disease of the cerebrovascular, cardiovascular, and peripheral vasculature has long been recognized. Carotid atherosclerosis increases the odds of coronary events and of finding atherosclerotic changes in coronary, cerebral, aortic, and iliac arteries. Similarly, peripheral vascular disease is a risk factor for both stroke and myocardial infarction and is associated with extracranial carotid and intracranial atherosclerosis. Data from the Reduction of Atherothrombosis for Continued Health (REACH) registry show that atherosclerosis in 1 vascular bed increases the risk of adverse outcomes in each of the other locations, and that multiple disease locations at baseline increase the risk of subsequent cardiovascular and cerebrovascular events. Given shared underlying risk factors and pathophysiology, it is not surprising that those who present with atherosclerotic disease in 1 vascular bed are at increased risk for disease in others.

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It is fair to say that although considerable attention has been given to atherosclerotic disease affecting peripheral, cardiac, and carotid arteries, important gaps remain in our knowledge. For example, the intracranial circulation has received comparatively less attention as a marker of systemic vascular risk, and most research has neglected nonatherosclerotic vascular pathologies. Intracranial nonatherosclerotic diseases are important causes of stroke in young people that may provide key clues to understanding systemic vascular disease. Two examples of intracranial nonatherosclerotic arteriopathies are moyamoya and large artery dolichoectasia. Moyamoya is an obliterator vasculopathy that occurs either in association with vessel wall injury from a variety of sources (moyamoya syndrome) or without an identifiable trigger (moyamoya disease). In contrast, dolichoectasia is a dilative arteriopathy with marked vessel elongation, widening, and tortuosity that predominantly affects the intracranial verteobasilar arteries. Dolichoectatic vessels usually have a dilated lumen, thin arterial wall, degenerated internal elastic lamina, thinning of the media, and smooth muscle atrophy. Dolichoectasia is strongly associated with aging and vascular risk factors. Rare case reports describe diffuse involvement of all intracranial vessels, and onset in childhood and infancy has been reported, suggesting underlying genetic or congenital causes. This relatively rare vessel anomaly (affecting 1% to 6% of asymptomatic people in scan and autopsy series) is linked to vascular risk factors and thus is likely preceded by a more common forme fruste stage of vessel enlargement.

The study by Tanaka et al reported in this issue of Arteriosclerosis, Thrombosis, and Vascular Biology examines basilar artery dilation as a possible forme fruste of dolichoectasia and demonstrates the importance of this nonatherosclerotic intracranial vessel pathology for systemic vascular risk. In their prospective imaging study, they show that basilar artery dilation at baseline is a significant predictor of future vascular events, especially coronary events. This risk persists even after accounting for the effects of vascular risk factors, intracranial small vessel disease measures, and large vessel disease measures. The authors show that basilar artery diameter is related to multiple vascular risk factors for small and large vessel intracranial disease, all of which are also related to coronary artery disease. Basilar artery changes cannot directly cause cardiac disease, so there must be genetic, biochemical, or alternative mechanisms that affect the basilar artery wall and increase the risk for future cardiac events. Basilar dolichoectasia has been associated with coronary artery ectasia and abdominal aortic aneurysms. It has been argued that this diffuse ectasia represents an extracellular matrix pathology that shares risk factors with, but is not a direct consequence of, atherosclerosis. This raises the intriguing possibility that basilar and coronary arteries may dilate and remodel in similar ways, and each reflects risk of the other.

Importantly, the risk from basilar artery dilation only begins to accrue after 2 years of follow-up (Figure 1 in Tanaka et al), suggesting that basilar dilation is an early marker of future risk. Longitudinal follow-up of patients with basilar artery dilation will be important to detect and prevent incident cardiovascular or cerebrovascular events, and to determine how and why some patients develop dolichoectasia. The findings of Tanaka et al open a new, albeit cloudy, window to understanding systemic vascular risk. Arterial wall changes beyond atherosclerotic plaque features have been understudied. Reasons for predominantly posterior intracranial circulation involvement, for the connection between coronary and posterior circulation vasculature, for the specific involvement of the internal elastic lamina, to name but a few clues, suggest that there is likely a category of disease that has been poorly recognized. We may have been distracted by clinical presentations (eg, bleeding, ischemic stroke, myocardial infarction) instead of appreciating the more subtle similarities and overlaps between vessel walls in differing locations. Although
arterial wall research has slowly begun to move beyond the lumen, the article by Tanaka et al. is the first step in moving beyond the plaque toward an understanding of the genetic, biochemical, and physiological contributors to the dynamic life of intracranial, coronary, and peripheral vessel walls.

**Disclosures**

None.

**References**


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