

Response to: effect of vasa vasorum in cerebrovascular compensation: 2 case reports

Long Li¹[^], Adam A. Dmytriw², Liqun Jiao^{1,3}

¹Department of Neurosurgery, Xuanwu Hospital, Capital Medical University, Beijing, China; ²Neuroradiology & Neurointervention Service, Brigham and Women's Hospital, Harvard Medical School, Boston, MA, USA; ³Department of Interventional Neuroradiology, Xuanwu Hospital, Capital Medical University, Beijing, China

Correspondence to: Liqun Jiao. Xuanwu Hospital, Capital Medical University, No. 45, Changchun Street, Beijing 100053, China. Email: liqunjiao@sina.cn. Response to: Wang R, Weng L, Li M. Effect of vasa vasorum in cerebrovascular compensation: 2 case reports. Ann Transl Med 2020;8:508.

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We read with great interest the paper by Wang et al. (1) in a previous issue of Annals of Translational Medicine. The authors report two cases of vasa vasorum (VV) on digital subtraction angiography (DSA) and suggest that VV collaterals would benefit revascularization of previously occluded cerebral arteries with improvement of downstream perfusion. However, their presentations and accompanying angiograms do not provide convincing evidence that the vascular channels developing in chronically occluded arteries are really VV.

First, the so-called VV collaterals penetrating the plaque on DSA may in fact represent luminal recanalization whereby thrombus is replaced by fibrous tissue in the chronic phase. In previous studies (2-4), multiple vascular channels which run distinctly outside of the expected lumen of occluded arteries were generally recognized as bridging collaterals developed from VV. In the process of maturation, the adventitial collaterals grow both in width and length and therefore develop a typical corkscrew-like or bead-like morphology (5,6), which are not consistent with the angiography presented by the authors.

Second, the VV is defined functionally as a network of microvessels that deliver trophic and nutritive factors, as well as regulatory signals. In different stages of atherosclerosis, VV neovascularization may exert both positive and negative physiological effects on plaque

stability (7). From this point of view however, improving downstream perfusion is beyond its definition and capacity. Both immature neovascularization leading to local arterial dissection (i.e., inside the vessel wall) and mature neovascularization forming bridging collaterals (i.e., adventitial to the artery wall) are no longer considered VV, but rather the sequelae of local dissection and of bridging collaterals respectively.

Third, the authors comment that DSA could be used prospectively as an approach for observing intracranial VV, which seems extremely unlikely. Literature has extensively documented the approximate range of VV diameters. In coronary arteries, the mean diameter of first-order VV is $160.9\pm5.10~\mu m$ and that of second-order VV is $67.99\pm2.72~\mu m$ (8). However, the detector resolution of contrast angiography is about $250~\mu m$, which makes it impossible to render the genuine contour of VV.

Finally, the authors comment that only contrast agentenhanced MRI and DSA are feasible modalities for observing intracranial VV. Our article work on a similar topic was published in 2020 and readily reveals firstorder VV (9). Using a higher resolution (10 µm) imaging modality, optical coherence tomography, we reported in vivo visualization of the human native intracranial arterial VV and intraplaque neovasculature, as well subsequently this year of neointimal VV with the same technology (10).

[^] ORCID: 0000-0003-2807-5728.

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