Case Report

Effect of vasa vasorum in cerebrovascular compensation: 2 case reports

Rongfei Wang, Luankun Weng, Mengzhen Li

Brain Center, Guangdong Provincial Hospital of Chinese Medicine, Guangzhou 510120, China

Correspondence to: Rongfei Wang. Brain Center, Guangdong Provincial Hospital of Chinese Medicine, Dade Road, Yuexiu District, Guangzhou 510120, China. Email: wangrongfei30@126.com.

Abstract: Intracranial vasa vasorum (VV) are rare and develop predominantly in the proximal segments of the internal carotid artery (ICA) and vertebral artery (VA). The typical appearance of intracranial VV has not yet been reported in clinical practice. Although VV in the ICA have been found to re-institute the collateral flow, bypassing the obstructive segment, far less attention has been paid to the manner in which VV can connect the two ends of the obstructive segment through the plaque. In this study, we present two cases and discuss the positive effects of VV. In our first case, a patient with basilar artery (BA) occlusion and multiple infarcts in the posterior circulation territory received endovascular treatment. Digital subtraction angiography (DSA) showed the existence of VV, which originated from the proximal BA lumen, penetrated through the vessel wall, bypassed the obstructive segment, re-penetrated through the vessel wall, and reconnected to the distal BA lumen. Balloon angioplasty was performed, specifically avoiding the path of the VV, then the VV had disappeared in follow-up angiography. In our second case, a patient who had been diagnosed with occlusion in the initial segment of the left ICA two years ago suffered a stroke. DSA revealed that the VV collaterals penetrated directly through the plaque of obstructive site and reconnected to the distal vessel lumen, which caused low hemodynamic compensation. Angioplasty was performed directly following the VV path, then follow-up angiography showed the VV had disappeared. Arterial occlusion, including in the intracranial and extracranial artery, could trigger the occurrence of VV, which can improve downstream perfusion. VV also could play a role of signal light in endovascular treatment.

Keywords: Vasa vasorum (VV); intracranial artery; occlusion; collateral compensation; endovascular treatment

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The vasa vasorum (VV), also called the "vessel of the vessel", was first observed by Gimbert in 1865 (1). The majority of VVs are distributed in the tunica adventitia, and the rest extend from the adventitial layer to the tunica media (2). Intracranial VVs are rare and develop predominantly on the proximal segments of the internal carotid artery (ICA) and vertebral artery (VA) (2). However, the typical appearance of intracranial VV has not yet been reported in clinical practice. In this study, we reported the existence of VV in occlused basilar artery (BA) and ICA, which improved downstream perfusion. Based on these two cases, we discuss the positive effects of VV in cerebrovascular occlusion.

Case presentation

Case 1

A 74-year-old woman with hypertension was admitted after experiencing slurred speech and facial drooping on the left side for 6 hours previously. Two months before, she had started experiencing tiredness and decreased alertness. Magnetic resonance imaging (MRI) revealed serious stenosis of the BA and multiple acute infarcts in the right pons, cerebellum, thalamus, and the left cerebral peduncle. Unfortunately, the patient's family refused endovascular treatment. Three days later, the patient developed more severe symptoms and went into hypnosia.

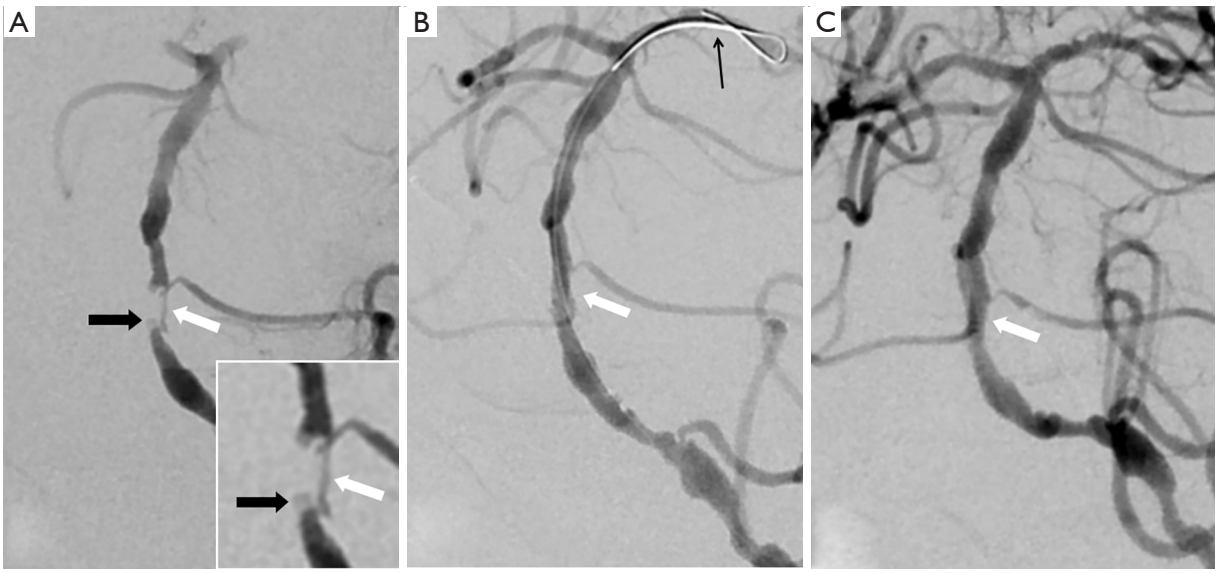

Figure 1 Digital subtraction angiography (DSA) showed basilar artery (BA) occlusion and a stump remaining in the proximal end [black arrowhead in (A)]. The vasa vasorum (VV) originated from the proximal BA lumen, penetrated through the vessel wall, bypassed the occlusion, re-penetrated through the vessel wall, and re-connected to the distal BA lumen [white arrowhead in (A)]. The microguidewire [slender arrowhead in (B)] threaded through the occluded segment from the stump to the left posterior cerebral artery, specifically avoiding the path of the VV, then balloon angioplasty was performed. After the BA was recanalized, the following DSA images showed the VV had disappeared [white arrowheads in (B,C)].

The second MRI showed larger infarcts. We performed an emergency operation once informed consent had been signed. Digital subtraction angiography (DSA) revealed BA occlusion with a stump in the proximal end (*Figure 1A*) and existence of VV originating from the proximal BA lumen, which penetrated through the vessel wall, bypassed the occlusion, re-penetrated through the vessel wall, and reconnected to the distal BA lumen (*Figure 1A*). The microguidewire was threaded directly through the occluded segment from the stump to the left posterior cerebral artery in endovascular treatment, avoiding the VV path. Balloon angioplasty was then performed. After these procedures, follow-up DSA images showed that the VV had disappeared (*Figure 1B,C*).

Case 2

A 62-year-old woman with hypertension and a history of stroke was admitted after the onset of severe right hemiparesis. Two years before, she was diagnosed with occlusion in the initial segment of the left ICA (L-C1), and was left with mild aphasia and weakness in her right limbs associated with stroke. She maintained the ability to take care of herself after medical treatments. We performed cerebrovascular assessments. Perfusion-weighted MRI showed a delay in diffuse perfusion in the left cerebral hemisphere. High resolution MRI revealed a narrowed lumen and wall thickening in the left ICA and M1 segment of the middle cerebral arterial (MCA), which was defined as negative reconstitution, and there was no enhancement in the thickened wall. Spectral and color Doppler imaging showed a narrow lumen in L-C1, with a single peak in the blood flow spectrum and a peak velocity of 46 cm/s. VV collaterals were revealed by DSA to penetrate directly through the plaque of obstructive site and to connect to the distal vessel lumen, and low hemodynamic compensation caused by VV and atrophy and collapse of the left ICA were also showed (*Figure 2A*). There was no collateral circulation from the anterior or posterior communicating arteries. Angioplasty was then performed to establish vessel recanalization in L-C1 and improve the perfusion within the left hemisphere (*Figure 2B,C*). The patient was prescribed aspirin (100 mg) and clopidogrel (75 mg) after being discharged home. Two months later, DSA confirmed that the VV had disappeared and the lumen of the left ICA had widened (*Figure 2D*).

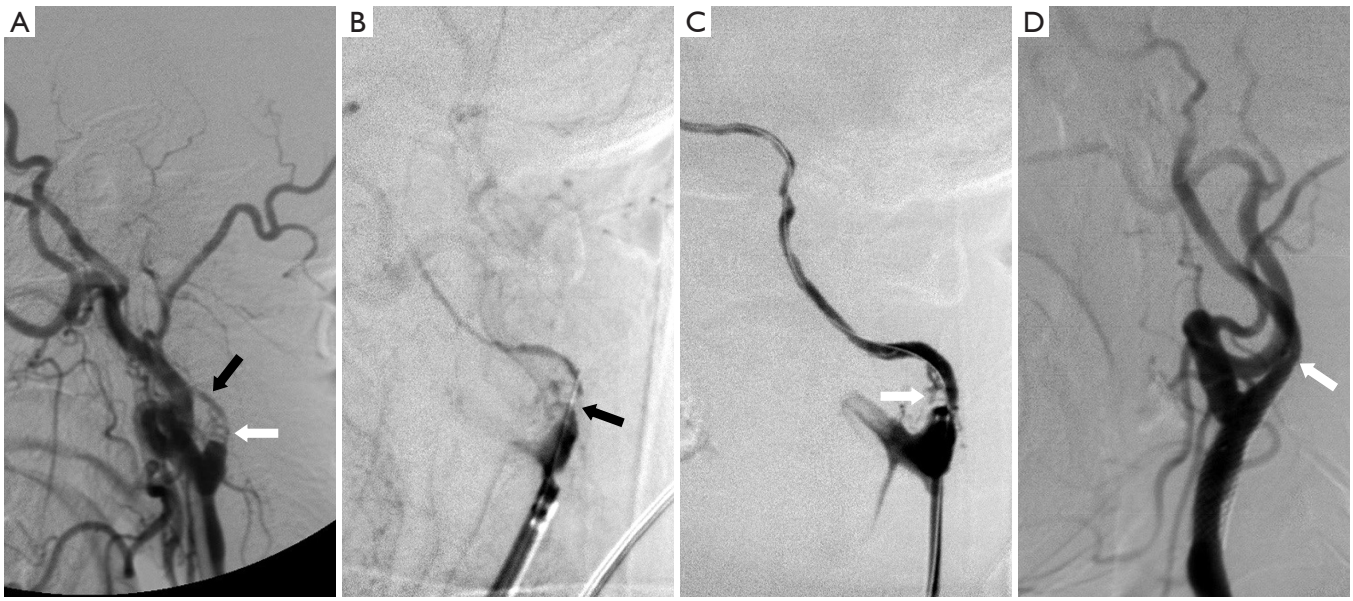

Figure 2 Digital subtraction angiography (DSA) showed vasa vasorum (VV) collaterals [white arrowhead in (A)] directly penetrated through the plaque leading to occlusion from the proximal vessel lumen to the distal [black arrowhead in (A)] and caused low hemodynamic compensation. In the neurointerventional operation, the microguidewire [slender arrowhead in (B)] was threaded directly through the plaque to the distal vascular lumen. Partial VV collaterals were also observed in the plaque after balloon angioplasty [white arrowhead in (C)], and then a stent was implanted. Two months later, the DSA showed the disappearance of VV [white arrowheads in (D)] and the larger lumen of internal carotid artery (ICA).

Discussion

Scarcity of intracranial VV

While rare, intracranial VV are essential for supplying nutrients and oxygen (3). VV are found in 67%, 45%, and 13% of VA, ICA, and BA, respectively, as well as in the distal cerebral arteries (MCA, ACA). Furthermore, the distribution of VV is very sparse (M1: 5%, A1: 2%) (2). Intracranial arteries have thinner media and adventitia than comparably sized extracranial vessels (4). The nutrient-rich cerebrospinal fluid (CSF) that bathes the adventitial surface probably contributes to the decreased demand for supplemental nourishment from the VV (5). Therefore, differences in the vascular structure and external environment of the arteries may be relative to the rarity of intracranial VV.

Methods of observing VV

In clinical practice, VV could be visualized directly by several imaging modalities, including microcomputed tomography (micro-CT), contrast-enhanced ultrasound, intravascular ultrasound, optical coherence tomography, contrast agent-enhanced MRI (6) and DSA. However, for observing intracranial VV, only contrast agent-enhanced MRI and DSA were the feasible modalities. There were challenges in interpreting the MR images. Notably, the plaque could be enhanced by the contrast agent as well as the VV. Although the VV were an important contributing factor to contrast agent uptake in the plaque, the mechanism of plaque enhancement was complex, and the VV were not the sole contributors. The small size of intracranial vessels can also increase the difficulty in identifying vessel wall enhancement. In addition, cerebral aneurysms, intracranial dissections, and intracranial vasculitis, which are related to the occurrence of VV, can express contrast enhancement (7). DSA seemed to be a prospective approach for observing intracranial VV, because although the VV were usually very small, often below the resolving power of DSA, thickened VV could be shown by DSA.

Clinical evidence of intracranial VV

There is not a typical description of intracranial VV in the literature of clinical practice. Muthusami *et al.* (8) reported a case of spontaneous revascularization caused

by collaterals reconstituting distal flow in a chronically occluded MCA branch of a 9-year old boy. However, the collateral segment manifested multiple tortuous channels on DSA images, mimicking a brain arteriovenous malformation. We presented the case of typical VV which improved downstream perfusion in occluded BA (Sig. 1a). The VV originated from the proximal BA lumen, penetrated through the vessel wall, bypassed the occlusion, re-penetrated through the vessel wall, and reconnected to the distal BA lumen. After the occluded BA was recanalized by endovascular treatment, the VV were not detected in the following cerebral angiogram. Harnoss *et al.* (9) produced a porcine model by minimally invasively implanting a copper stent into the coronary artery, resulting in a novel semi-acute vessel occlusion. The micro-CT angiogram imaging of the coronary arteries clearly showed VV collaterals, which closely resembled the DSA images of VV in our case. They considered that the hemodynamic pressure difference between the two ends of the occlusion played an important role in the occurrence and development of VV. In our case, the relief of hemodynamic pressure difference resulting from BA recanalization caused the immediate closure of the VV collaterals. This phenomenon indicated that the effect of hemodynamic pressure difference was reversible. Since the VV bypassed the occlusion from the tunica adventitia or the outer layer, the microguidewire could not follow the path of the VV in the operation.

Growing paths of VV in ICA

Several reports have studied revascularization in the ICA via VV after occlusion (10,11). The VV originated from the proximal vessel lumen and penetrated through the vessel wall, forming an irregular and tortuous vascular channel across the occlusion, then penetrated the wall again and reconnected to the distal ICA lumen. The connection points could be either close to the occlusion, or at the distantly cavernous segment (11). However, far less attention has been paid to the idea that VV connect the two ends of the occlusion through the plaque. In our case, the DSA images clearly showed that the VV collaterals penetrated directly through the plaque leading to occlusion from the proximal to the distal vessel lumen, causing low flow compensation (*Figure 2A*). Although the pathogenesis of atherosclerosis is inseparable from the appearance of VV, VV conventionally penetrate from the adventitia and outer layers of the media to the tunica intima, supplying them with nutrients and oxygen (3). Our case suggested that the VV did not necessarily follow the conventional growth path. Although the majority of the VV penetrate though the vascular wall, under certain conditions, the VV in plaque may become the mainstream and eventually reconnect the two ends of the occlusion. After VV reconnection, the difference in hemodynamic pressure between the two ends of the occlusion declined and the ischemia and hypoxia were improved. When they reached an equilibrium point, the angiogenesis would stop. As the VV reconnected the distal lumen from the plaque in the vessels, the microguidewire could be guided along the VV path in endovascular operation.

The occurrence of VV was not itself pathological, but was rather a normal physiological response triggered by the oxygenation and nutritional needs of the artery. Obstruction, hypoperfusion, or endothelial barrier leakiness could reduce oxygen and nutrient metabolism and trigger the occurrence of VV (3). The VV would follow paths which benefited revascularization and improvement of downstream perfusion. From this point of view, VV could be regarded as third-level collateral compensation in the cerebrovascular system. Moreover, VV could also prove useful in endovascular treatment, depending on whether the microguidewire is able to follow the VV path.

Conclusions

Arterial occlusion, including in the intracranial and extracranial artery, could trigger the occurrence of VV, which can improve downstream perfusion. VV could be regarded as third-level collateral compensation in the cerebrovascular system and play a role of signal light in endovascular treatment.

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Footnote

Conflicts of Interest: All authors have completed the ICMJE uniform disclosure form (available at http://dx.doi.org/10.21037/atm.2020.03.77). The authors have no conflicts of interest to declare.

Ethical Statement: The authors are accountable for all

aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved. Written informed consent was obtained from the patients for publication of this manuscript and any accompanying images.

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