Letter to the Editor

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Letter by Mugikura and Takahashi Regarding Article, “Perfusion Characteristics of Moyamoya Disease: An Anatomically and Clinically Oriented Analysis and Comparison”

To the Editor:

We completely agree with the findings by Schubert et al:\(^1\): cerebrovascular reserve capacity was exhausted in the large area, including the middle cerebral artery (MCA) territory and its adjacent anterior and posterior watershed areas with resultant decreased cerebral blood flow (CBF), whereas cerebrovascular reserve capacity in the pericallosal area was not with relatively preserved CBF.

In their patients without posterior cerebral artery (PCA) involvement, deep leptomeningeal collaterals through the posterior pericallosal artery (Fisher’s anastomosis) from the proximal PCA as well as superficial leptomeningeal collaterals from the more distal PCA branches presumably developed to compensate for the reduced blood flow in the anterior circulation.\(^2\) In that case, the deep collaterals mainly perfuse the medial part of the anterior cerebral artery territory, including the pericallosal area,\(^1\) whereas the superficial collaterals perfuse the lateral part of the hemisphere, including the MCA and its adjacent anterior and posterior watershed areas.\(^2\) Therefore, we hypothesize that the results of the study by Schubert et al\(^1\) described above may be interpreted as indicating the insufficient compensation of the superficial collaterals to prevent relative shortage in CBF in MCA and its adjacent anterior and posterior watershed areas. We presume this insufficient compensation was ascribed to their lengthy and retrograde route through the end-to-end anastomoses between the most distal branches of the PCA and MCA to supply the region of the anterior circulation: the anterior watershed of that region would be the farthest supply area from the heart. Indeed, even in patients without PCA involvement, the anterior watershed area is most commonly involved by infarction in this disease.\(^3\)

In moyamoya disease, stenoocclusive changes initially affect the internal carotid artery system, followed by the PCA involvement.\(^2,4\) When the stenoocclusive changes affect the PCA usually involving its proximal segments, CBF in the MCA and its adjacent watershed areas, which was shown to decrease even in patients without PCA lesions by Schubert et al,\(^1\) would decrease drastically and might become even more causative of developing infarctions in these regions. Indeed, infarctions tended to be distributed in the anterior watershed in less-advanced PCA cases, whereas in more advanced PCA cases lesions were additionally found posteriorly in the territory of the MCA and the posterior watershed area. We postulated that decrease of the superficial leptomeningeal collaterals according to the advancement of PCA changes was closely related to such a pattern of developing cerebral infarction.\(^5\)

In contrast, the pericallosal area and its adjacent region of the anterior cerebral artery territory are relatively well preserved from infarction.\(^2\) As a reason for this, we hypothesize that the CBF in the pericallosal area would be well preserved even in patients with PCA involvement, because the posterior pericallosal artery would receive blood supply from the proximal PCA via the moyamoya vessels of the PCA (PCA moyamoya) including enlarged medial posterior choroidal arteries that are known to have frequent anastomoses with the posterior pericallosal artery. Thus, deep leptomeningeal collaterals through the posterior pericallosal artery are liable to get blood supply via a shortcut route or the PCA moyamoya from the proximal PCA. The PCA moyamoya gradually increases as the stenoocclusive PCA involvement advances until it becomes extremely severe, whereas the superficial leptomeningeal collaterals consistently decrease.\(^1\)

We believe the 2 different types of leptomeningeal collaterals from the proximal and distal PCA explain the territory-specific perfusion pattern as indicated by Schubert et al.\(^1\) Further studies examining the relationship between PCA changes and perfusion are required to validate the hypothesis.

Disclosures

None.

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