Borderzone Infarction: Stroke Topography Does Not Easily Equate With Stroke Mechanism

To the Editor:

We congratulate Yong and colleagues on their recent article about borderzone infarction. However, the findings from this detailed study can be interpreted differently and we would like to propose an alternative conclusion.

Originally, the interest in these stroke syndromes was that their recognition potentially suggested a hemodynamic mechanism and hence direction for medical therapy. Investigators emphasized the importance of severe carotid artery stenosis/occlusion coupled with hemodynamic compromise to the mechanism of internal borderzone (IBZ) infarction. However, this is not a constant finding, and others have found IBZ infarction occurring in the setting of mild, moderate and severe carotid artery stenosis. The cortical borderzone (CBZ) hypothesis was also questioned when it was found that there was variability in the classification of CBZ infarcts depending on whether the minimum or maximum middle cerebral artery map was used for topographic classification. Much later, hemodynamic compromise with impaired clearance of emboli was put forward as an explanation for microemboli found at autopsy and on transcranial Doppler in patients with “borderzone” infarction.

With this in mind, it was interesting to read that Yong and colleagues found severe stenotic/occlusive carotid artery disease of the internal carotid artery (ICA) and middle cerebral artery (MCA) in 60% patients with CBZ infarction. Based primarily on this, they concluded that “embolic pathogenesis appears to contribute greatly to the genesis of CBZ infarcts”. Moreover, they remained convinced that IBZ infarction was caused by hemodynamic compromise, based mainly on a higher proportion of people with IBZ infaracts having stenotic/occlusive ICA or MCA disease (75.5%) than those with CBZ (60%; see their Table 2). A limitation of this approach is their assumption that severe arterial stenotic disease equates with a hemodynamic mechanism even in the absence of recorded hemodynamic events immediately before stroke. Moreover, the difference in the proportion of patients with severe ICA stenosis/occlusion between the 2 groups is not significant. If one were to adopt the classic view that carotid disease is an important component of a hemodynamic mechanism, then their data do not support a different (potentially hemodynamic) mechanism between IBZ and CBZ infarcts. An embolic cause could not be excluded in the IBZ group because 15.6% of IBZ patients had potential sources of cardioembolism. In addition, aortic arch atheroma was not fully excluded as a stroke mechanism in their study. Thus, there is enough uncertainty in the data to suggest caution in adopting a strong view about differences in stroke mechanism between apparent IBZ and CBZ infarcts.

A consideration of infarct topography is useful in generating hypotheses about stroke pathophysiology but should not be the principal basis for inferring stroke mechanism. Borderzone infarcts may represent territorial infarcts secondary to emboli. The deeper brain structures within the MCA territory are supplied by the lenticulostriate arteries and the long penetrating medullary arteries with poor collateral blood supply and thus may be prone to the effect of emboli. The embolic occlusion of these arteries may lead to deprivation of blood supply to the smaller arterial branches of these vessels potentially giving rise to the apparent IBZ infarct distribution. The “embolic” postulate may also fit with the high frequency of carotid or MCA disease described in this study. Such mechanistic hypotheses need to be tested further and not be inferred based purely on topographic analysis of imaging studies. Careful measurement and identification of alternative (hemodynamic and nonhemodynamic) stroke mechanisms, and not lumping of stroke syndromes, will provide the direction of secondary stroke prevention in the individual patient.

Disclosures

None.

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