Letter by Gutierrez and Elkind Regarding Article, “Patterns and Implications of Intracranial Arterial Remodeling in Patients With Stroke”

To the Editor:

We read with interest the article by Qiao et al,1 in which the authors affirm that there exists evidence of brain arterial outward remodeling that accommodates an expanding intracranial plaque. Our published data from a postmortem sample of brain arteries, however, suggests that such compensatory responses are not found.2 The reasons for this discrepancy may lie in the methods used for each study. First, and most important, the definition of plaque burden used in this study does not represent true plaque burden (ie, intima area) but rather it represents the proportion of the interadventitial area occupied by the wall area. Given this limitation, stating that brain arteries can accommodate plaque burden ≤55% before the lumen narrows is not supported by the data presented. In fact, the linear fit observed in supplemental Figure III is most likely the result of colinearity between wall area and stenosis, that is, as the stenosis increases so does the intima area, and intima area becomes a more important component of the total arterial area as stenosis progresses. Plotting lumen area against stenosis would better serve this purpose.3 Second, the tapering correction needs further clarification. The slope of the regression line (S) is reported as positive (article’s supplemental Figures I and II) but it should be negative, as the arterial caliber normally decreases from proximal to distal locations within the same arterial segment.4 Furthermore, it is reported that plaques were measured in the proximal segments of intracranial arteries, but then the authors report that the lesion site was identified as the portion with the thickest plaque without mentioning the proximal location as a criterion. It is unclear whether these terms were used interchangeably. The positivity or negativity of the β coefficient should vary depending on whether the referent site preceded or followed the lesion site, and it is unclear whether this was actually incorporated into the calculations. Yet, another concern for the tapering correction arises from the selection bias for symptomatic patients, which would select cases with more widespread thicker wall and neighboring intima disease as is usually the case in advanced atherosclerosis.5 Finally, although the 400 to 450 μm resolution of intracranial arterial wall imaging is impressive for clinical magnetic resonance imaging techniques, this resolution may not be ideal for the A2, M2, and P2 arterial segments included in this study in which the wall thickness measures 200 to 300 μm.4 Comparing the article’s Figure 2 (M1) against the article’s Figure 3 (basilar artery) offers an example of poorer delimitation of the outer wall area dependent on size.

We welcome the uncertainty that this study introduces to what is already published about brain arterial remodeling. Outward remodeling occurs in brain arteries with aging and pathologically in the setting of dolichoectasia. Whether it occurs also as a result of growing plaque, and to what extent, should be confirmed with prospective data. The feasibility of this confirmation seems more likely as techniques like the one used in this study become more prevalent and successfully validated within the boundaries imposed by the resolution achieved.

Disclosures

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