Response to Letter by Charidimou et al Regarding Article, “Blood Pressure Reduction, Decreased Diffusion on MRI, and Outcomes After Intracerebral Hemorrhage”

Response:

We thank Dr Werring’s group in London for their recent letter. We are glad that the issue of cerebral ischemia after intracerebral hemorrhage (ICH) interests so many.

We did not find that cerebral amyloid angiopathy was associated with decreased diffusion (DD) on MRI, but it was an uncommon clinical cause of hemorrhage in ours and another published urban cohort study. We did not specifically assess white matter changes but agree this is an important topic for future research. Although there may be confounders that accounts for both DD and worse outcomes, we were unable to find one as detailed in the article. Indeed, the effect of DD on outcomes was independent of the most potent predictor of outcomes, the ICH score.

We agree that the timing of MRI is also important as it relates to the timing of the acute ischemic events. MRI performed more than 1 month after symptom onset, as commonly done in the European study, would be less likely to detect most acute ischemia. The only way to determine if acute ischemia is clustered around the time of symptom onset is to prospectively identify patients early after onset and perform MRI at defined intervals from the acute to chronic phases; we have submitted a grant proposal with repeated MRI to determine the timing of DD and quantitative MR perfusion to determine if reduced perfusion coexists and/or precedes acute infarction seen on diffusion imaging.

Dr Werring’s group notes that the admission blood pressure (BP) was not different in patients with ischemia on MRI. However, admission BP is not a risk factor for DD. Rather, change in BP, not baseline BP, was associated with cerebral ischemia on MRI in the two independent cohorts of acutely symptomatic ICH patients in whom BP lowering was provided as standard of care. These consistent observations indicate that acute blood pressure reduction after ICH is associated with DD. Furthermore, the link to outcomes as noted in our study raises important concerns about the impact of BP lowering in ICH patients. Although vasculopathy (ie, amyloid angiopathy or hypertensive) may contribute toward a predisposition for ischemic injury after ICH, BP lowering may play a more direct and causal role.

If the funds can be found, it is time to reconsider adding an MRI to the protocols for INTERACT-2 and ATACH-2 to evaluate for DD and assess the interaction between aggressive BP lowering, DD, and functional outcomes after ICH. Although INTERACT-1 showed some efficacy in reducing hematoma growth, an increased risk of cerebral ischemia and DD can only reduce its potential benefit in improving functional outcomes. We postulate that an interaction might exist such that the benefit of BP lowering depends on whether or not DD is present: BP lowering may reduce hematoma growth and lead to improved functional outcomes in those who do not have DD, but BP lowering may lead to worse functional outcomes in those with DD.

Disclosures

None.

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