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Reply:

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REPLY:

We thank Dr Mugikura and colleagues for their comments regarding our recent article "Patient Outcomes and Cerebral Infarction after Ruptured Anterior Communicating Artery Aneurysm Treatment."[1] Patients who undergo ruptured anterior communicating (AcomA) aneurysm treatment by microsurgical clipping have been shown to have worse clinical outcomes compared with coil embolization,[2] and these worse outcomes include reduced levels of functional independence and more severe neurocognitive outcomes. In our study, we found that ischemic infarction was more common after microsurgical clipping compared with coil embolization. Our observation that cerebral infarction in the recurrent artery of Heubner (RAH) territory was much more common in patients who underwent microsurgical clipping compared with coil embolization (33% versus 2%) does suggest an interesting mechanism by which surgical clipping might result in worse outcomes compared with coil embolization. We agree that the limitations of retrospective analyses result in much difficulty in discerning the exact causes of patient dependency after such a neurologically complicated event as rupture of a cerebral aneurysm.

In response to the first question raised by our colleagues, we did not detect any subcallosal artery infarctions in our study population, as they note in their letter. Patients in our study underwent posttreatment imaging evaluation by both CT and MR imaging, and the reduced sensitivity of CT to cerebral infarction, especially with subarachnoid and intraventricular hemorrhages, may limit the detection of subcallosal artery infarctions (including the fornix) in our study. This limitation is perhaps more pronounced because more patients who underwent surgical clipping had imaging follow-up by CT rather than MR imaging, and these were the patients more likely to have an RAH infarction and possibly a subcallosal artery infarction, as our colleagues argue. We agree that a more thorough discussion of these (and other) limitations could have been mentioned in our discussion. However, as any author of a retrospective study must do, we chose to list the most important limitations to our overall results; the lack of randomization, lack of blindness to treatment technique on follow-up imaging, and limited patient follow-up were deemed more important to emphasize to our readers.

We would like to further highlight the important methodologic and patient population differences between our colleagues' prior study[3] and our study.[1] The prior study of Mugikura et al[3] that identified subcallosal infarctions was performed in patients who developed amnesia following ruptured or elective AcomA aneurysm clipping, and the imaging evaluation of these patients was performed several months after treatment using only MR imaging. By contrast, our study[1] and another study[4] included all patients who underwent ruptured AcomA treatment, patients treated by both clipping and coiling, and no specific posttreatment symptom (ie, amnesia) was required for subsequent analysis. We would argue that these differences in patient populations limit direct comparison between these studies when attempting to compare the frequency of infarctions related to the various perforating vessels that arise from the anterior communicating artery complex. We would also advise caution in directly applying the results from Mugikura et al to the population in our study, given these differences.

In response to the second point raised by our colleagues, reduced functional independence may be due to memory loss and amnesia, which is the focus of the prior study of Mugikura et al.[3] However, there are many other causes of patient dependency after rupture of a cerebral aneurysm. As they note in their letter and we note in our limitations, we do not routinely perform neuropsychological testing after AcomA aneurysm rupture and treatment. We are therefore unable to comment on the specifics of memory loss, decision-making, and other executive function deficits. As we discussed in our article, striatum infarction has been linked to all these deficits,[5-7] whereas subcallosal artery infarction may be more specific for memory deficits.[3] Thus, we would argue that RAH infarction may be an overall more important contributor to patient outcome in patients with ruptured AcomA aneurysms.

Last, we agree with our colleagues that a prospective study of patients with ruptured AcomA aneurysms is necessary to understand better the cause of worse neurologic outcomes after surgical treatment compared with coil embolization. Clearly, detailed MR imaging evaluation before and after treatment (at several time points) in concert with detailed neuropsychological evaluation would provide the best data to answer these questions.

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