

**REPLY:**

We sincerely thank Harrison X. Bai and colleagues for their interest and comments regarding our recent article, in which we demonstrated the diagnostic significance of cortical superficial siderosis (cSS) for Alzheimer disease in patients with cognitive impairment.<sup>1</sup>

Regarding the methodologic issues of the present study that their letter raises, the diagnosis of cSS seen on susceptibility-weighted imaging was based on collegial discussion with experienced neuroradiologists, and no data are available for interobserver variability. We defined cSS as linear hypointensities on the surface of the cerebral gyri on SWI. The appearance of cSS on SWI was obvious in this study; fortunately, we had no difficulty in distinguishing cSS from lobar cerebral microbleeds (MBs). Very superficial clusters of multiple MBs can be mistaken for cSS, but these would be distinguished by their irregular appearance.<sup>2</sup>

In relation to exclusions, 4 patients with previous symptomatic subarachnoid hemorrhage, 23 patients with traumatic subdural hematoma, and 1 patient with an intracranial operation were excluded according to medical charts. We therefore presented 12 cases with cSS that did not seem to have occult sources of bleeding, as the authors pointed out.

We agree with their statement that elucidating the pathogenesis of cSS from cerebral amyloid angiopathy (CAA) warrants further analysis. However, imaging-histopathologic correlations

shown in cases with CAA might indicate that recurrent blood leakage of meningeal vessels leads to the propagation of cSS.<sup>3</sup> To confirm the progression of CAA-related cSS, prospective studies are needed that recruit patients with CAA based on the Boston criteria.<sup>4</sup>

**REFERENCES**

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