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Reply:

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REPLY:

We would like to thank Wu and colleagues for their critical analysis of our recently published study. As the authors point out, our meta-analysis found a strong association between deep cerebral venous anomalies and nonaneurysmal perimesencephalic SAH.¹ As demonstrated in our meta-analysis, patients with aneurysmal SAH might also have deep cerebral venous drainage anomalies, thus making the presence of this imaging finding, especially in the case of a subarachnoid hemorrhage extending beyond the perimesencephalic cisterns, nonspecific. However, in case of a typical perimesencephalic hemorrhage with negative DSA findings for aneurysm, the identification of deep cerebral venous drainage is an additional imaging finding in favor of nonaneurysmal perimesencephalic SAH.

Contrary the suggestion of Wu et al, the literature does not recommend abandonment of DSA in the initial phase of perimesencephalic hemorrhage.² In a recently published study of 230 patients with CTA negative for hemorrhage, Heit et al³ found that DSA identified vascular pathology in 13% of patients. Among patients with perimesencephalic hemorrhage, an aneurysm was found in 3% of cases and vasculitis was found in 1.5% of cases. While admittedly the diagnostic yield of angiography was low (4.5%), the risks of DSA are even lower and the importance of establishing the correct diagnosis is high.^{4,5} We do agree, however, that second-look DSA does not appear to have a sufficient diagnostic yield for the detection of a causative aneurysm in case of negative findings on 6-vessel DSA with 3D rotational angiography.⁶ If some authors maintain the futility of DSA in cases of typical perimesencephalic hemorrhage, the drainage venous pattern may still be evaluated with cross-sectional imaging (ie, CTV or MRV).⁷

One of the most interesting aspects of studying venous anomalies and disease in perimesencephalic hemorrhage is that we may be slowly arriving at a better understanding of the nature of the disease. The association between venous anomalies and nonaneurysmal perimesencephalic hemorrhage suggests that this could be secondary to a venous rupture/leak. In case of primitive venous drainage, the direct connection of the thin-walled perimesencephalic veins with the dural sinuses may predispose to sudden increases in venous pressure with engorgement and rupture of the veins as a result.⁸⁻¹⁰ However, because the anatomic venous drainage pattern is not likely to change after bleeding, it is surprising that the incidence of rebleeding is very low in patients after the initial perimesencephalic SAH.¹¹ Matsumaru et al¹² speculated

that the spontaneous healing of the venous rupture by fibrous tissue reaction would reinforce the wall of the vein, decreasing the risk of rupture.⁹

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