Neuropathologically defined cerebral amyloid angiopathy (CAA) cohort suggests that cortical superficial siderosis (CSS) and APOE ε2 are related to the hemorrhagic expression of the disease; APOE ε4 is enriched in nonhemorrhagic CAA. The study emphasizes the concept of different CAA phenotypes, suggesting divergent pathophysiologic mechanisms [1].

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418 consecutive patients admitted with primary lobar hemorrhage or deep ICH to a single tertiary care medical center between January 1, 2000, and October 1, 2012, were analyzed on March 4, 2016. Participants were consecutive patients with computed tomographic images allowing ICH volume calculation and MRI allowing imaging markers of SVD assessment.

The ICH volumes at baseline and within 48 hours after symptom onset were measured in 418 patients with spontaneous ICH without anticoagulant therapy, and hematoma expansion was calculated. Cerebral microbleeds, cortical superficial siderosis, and white matter hyperintensity volume were assessed on MRI. The associations between these SVD markers and ICH volume, as well as hematoma expansion, were investigated using multivariable models.

This study analyzed 254 patients with lobar ICH (mean [SD] age, 75 [11] years and 140 [55.1%] female) and 164 patients with deep ICH (mean [SD] age 67 [14] years and 71 [43.3%] female). The presence of cortical superficial siderosis was an independent variable associated with larger ICH volume in the lobar ICH group (odds ratio per quintile increase in final ICH volume, 1.49; 95% CI, 1.14-1.94; P = .004). In multivariable models, the absence of cerebral microbleeds was associated with larger ICH volume for both the lobar and deep ICH groups (odds ratios per quintile increase in final ICH volume, 1.41; 95% CI, 1.11-1.81; P = .006 and 1.43; 95% CI, 1.04-1.99; P = .03; respectively) and with hematoma expansion in the lobar ICH group (odds ratio, 1.70; 95% CI, 1.07-2.92; P = .04). The white matter hyperintensity volumes were not associated with either hematoma volume or expansion.

In patients admitted with primary lobar or deep ICH to a single tertiary care medical center, the presence of cortical superficial siderosis was an independent variable associated with larger lobar ICH volume, and the absence of cerebral microbleeds was associated with larger lobar and deep ICHs. The absence of cerebral microbleeds was independently associated with more frequent hematoma expansion in patients with lobar ICH. We provide an analytical framework for future studies aimed at limiting hematoma expansion [2].

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