Progression of Brain Network Alterations in Cerebral Amyloid Angiopathy

In this interesting prospective longitudinal study of the natural history of cerebral amyloid angiopathy (CAA), Reijmer et al. explore the progression of white matter pathology over time and its correlation with cognitive decline. The authors quantified the efficiency of global white matter connections network by analysis of diffusion tensor imaging; CAA was dichotomized as moderate or severe based on the number of cerebral microbleeds, and a detailed battery of neuropsychological testing was performed in all 33 patients included in their final analysis. The authors found an unequivocal decline of the global network efficiency during a mean follow-up time of 1.3 years, to an extent significantly higher than previous studies in healthy adults. This decline was driven by the subgroup with severe CAA. A similar pattern was noted in the posterior–frontal and frontal–frontal connections, whereas the posterior–posterior connections were affected across the whole study. The decline in network connectivity was correlated with a parallel decline in executive functioning; no relationship was found with processing speed and memory. White matter hyperintensity volume also increased over time, but it was not associated with concurrent decline in cognitive performance. Overall, despite its small sample size, this study offers unique insight into the biology of CAA: It reveals a structural, measurable decline in the quality of white matter connections, commensurate with the severity of the disease and at a faster rate than what naturally occurs as part of normal ageing. In addition, it reveals a unique posterior–anterior gradient, suggesting that although CAA predominantly affects posterior cerebral regions, in more severe forms and over time, it becomes more diffuse, affecting more anterior regions as well. Future studies examining the disease progression in larger patient samples and over longer periods of time are expected to deepen our understanding of this complex disease even more. See p 2470.

Risk Factors and Cognitive Relevance of Cortical Cerebral Microinfarcts in Patients With Ischemic Stroke or Transient Ischemic Attack

In this subset of 231 of patients with acute ischemic stroke or transient ischemic attack from the prospective STRIDE study (Stroke Registry Investigating cognitive Decline), Mok et al. examined the prevalence, risk factors, and cognitive repercussions of cortical cerebral microinfarcts (CMIs) in 3-Tesla magnetic resonance imaging. The Montreal Cognitive Assessment was used to measure the global cognitive performance at baseline and over 28 months. Approximately 15% of the patients had CMIs. Both acute and chronic CMIs were significantly more frequent in patients with atrial fibrillation or those with cardioembolic stroke. On the contrary, only chronic CMIs were associated with confluent white matter hyperintensities, suggesting that the possible etiologic mechanisms for CMIs are likely to be diverse. On the basis of these findings, the authors propose that both cardioembolism and small-vessel disease might be implicated in the pathogenesis of CMIs. The authors found no differences in the global cognitive performance or its change over time. However, when exploring specific cognitive domains, patients with CMIs had significantly lower performance in the visuospatial domain; this was the only domain in which a significant decline was noted during the 28-month follow-up. The explanation for this preferential involvement of visuospatial performance might be the higher prevalence of parietotemporal CMIs in this patient group, although the reason for this spatial lesion distribution is not clear. Despite the use of 3-Tesla magnetic resonance imaging that might underestimate the total CMI burden compared with more sensitive imaging modalities such as 7-Tesla magnetic resonance imaging, this in vivo study sheds significant light in the pathophysiology of CMIs in acute ischemic stroke and highlights some of the many unanswered questions surrounding this interesting imaging biomarker. See p 2450.

Higher Risk of Ischemic Events in Secondary Prevention for Patients With Persistent Than Those With Paroxysmal Atrial Fibrillation

Using data from the SAMURAI-NVAF study (Stroke Management with Urgent Risk-Factor Assessment and Improvement-Nonvalvular Atrial Fibrillation), a prospective, observational study conducted in 18 Stroke centers in Japan, Koga et al. report on the difference in ischemic events in patients with NVAF-related stroke depending on the type of NVAF. The authors dichotomized 1192 patients into paroxysmal or sustained NVAF, the latter group including both persistent and permanent NVAF. The 2 groups were imbalanced with regard to several baseline risk factors, including congestive heart failure, previous stroke, or hemorrhage, which were higher in those with sustained NVAF. Despite comparable rates of anticoagulant therapy use reaching 96% for each group, the incidence of recurrent stroke or systemic embolism was ≈2-fold in the sustained NVAF group, even after adjusting for imbalances in baseline risk factors. The findings of the study are in agreement with post hoc analyses of randomized controlled trials of anticoagulation use in NVAF-related stroke but different from similar previous observational studies that found no difference in stroke recurrence risk based on the duration of NVAF. The reason for this discrepancy is not immediately clear. Although not specifically studied in the context of this study, the authors suggest that structural changes in the left atrial appendage in those with sustained NVAF make them more prone to emboli formation; this research thread merits further exploration. See p 2582.
Stroke: Highlights of Selected Articles

Stroke. 2016;47:2442
doi: 10.1161/STROKEAHA.116.015213
Stroke is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 2016 American Heart Association, Inc. All rights reserved.
Print ISSN: 0039-2499. Online ISSN: 1524-4628

The online version of this article, along with updated information and services, is located on the World Wide Web at:
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