

## Editorial



# Atrial Fibrillation and Brain Health: Bridging the Heart–Brain Connection Beyond Stroke Prevention

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► See the article “Atrial Fibrillation, Brain Structure and Cognitive Function: A Mediation Analysis” in volume 56 on page 301.

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Atrial fibrillation (AF) is a major cause of stroke and systemic embolism.<sup>1)</sup> In recent years, however, attention has shifted toward its broader neurological consequences. Growing observational evidence suggests that AF is associated with subtle yet potentially clinically relevant alterations in brain structure and cognitive performance beyond overt thromboembolic events; however, these relationships remain associative, and causal pathways have not been definitively established.<sup>2-4)</sup> In this issue of the *Korean Circulation Journal*, Yang and colleagues<sup>5)</sup> report that AF is associated with both smaller brain volumes and lower cognitive scores, and that brain atrophy partly mediates this relationship. Using brain magnetic resonance imaging (MRI) and comprehensive neuropsychological testing, their analysis provides evidence that AF can contribute to cognitive decline even in individuals without a history of stroke.

This study distinguishes itself by its clear design and analytical depth. Earlier work from the Framingham Offspring and ARIC cohorts established that AF is related to lower brain volume,<sup>6,7)</sup> but Yang et al.<sup>5)</sup> extend these findings through formal mediation modeling. They quantify, for the first time in an Asian population, how much of the AF–cognition link is explained by MRI-based measures of atrophy—about 11% in this cohort. The use of the KoGES-CAVAS-C dataset is another strength, offering well-characterized data from Korean participants and addressing the need for ethnically diverse evidence in brain–heart research.

Importantly, the investigators combined region-specific morphometry with cognitive domain analysis. AF-related changes were most evident in gray matter, hippocampus, and frontal–temporal regions—areas responsible for executive and memory functions. The cognitive effects were particularly notable in attention and executive domains, and the authors point out that the magnitude of AF’s impact was roughly twice that of diabetes mellitus. These findings suggest that AF’s neurological burden may rival that of traditional vascular risk factors.

The implications are clear. If brain atrophy accounts for only a small part of AF-related cognitive decline, other mechanisms—microemboli, chronic hypoperfusion, systemic inflammation, or atrial myopathy—must be involved. Managing AF therefore requires a broader perspective that integrates both cardiovascular and cognitive protection. Emerging

**Data Sharing Statement**

The data generated in this study is available from the corresponding author upon reasonable request.

The contents of the report are the author's own views and do not necessarily reflect the views of the *Korean Circulation Journal*.

data hint that early rhythm control and sustained sinus rhythm could reduce dementia risk, but confirmation will require prospective studies incorporating serial imaging and biomarker assessments.

As the authors acknowledge, the cross-sectional design and small number of AF cases limit causal inference. Even so, the consistency of their results and the careful handling of confounders make the conclusions credible. The study reinforces the view that AF is not confined to the atria—it is a systemic disorder with consequences for the brain.

In summary, Yang et al.<sup>5)</sup> provide persuasive evidence linking AF to brain atrophy and cognitive impairment and show that structural brain changes mediate part of this association. Their work highlights the heart–brain continuum and calls for integrating cognitive evaluation into AF care. With aging populations and rising AF prevalence, protecting cognitive health should stand alongside stroke prevention as a core therapeutic goal.

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