

Ischemic Stroke Patients with Non-Valvular Atrial Fibrillation have a Risk for Aortogenic Embolizations

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We have a great interest in the article in *Journal of Atherosclerosis and Thrombosis* by Suzuki *et al.* titled *Complex Aortic Arch Atherosclerosis in Acute Ischemic Stroke Patients with Non-Valvular Atrial Fibrillation*. The authors demonstrated that 38.7% transesophageal echocardiography-derived complex aortic arch plaques (CAPs) among 106 patients with acute ischemic strokes with atrial fibrillation (AF), suggesting that patients with acute ischemic stroke and AF often had CAPs. The atheromatous lesions at the aortic arch are one of the causes of ischemic strokes. The cause of acute ischemic strokes in patients with AF could not only be cardiogenic embolisms due to AF but also aortogenic embolisms due to CAPs. The possibility of concomitant CAPs should be considered for stroke patients with AF. Non-obstructive general angioscopy has the possibility to detect aortic plaques in the aortic arch more accurately than TEE and might help to diagnose atheromatous plaques and embolic materials in the aortic arch. Further studies are needed to elucidate the causes of ischemic strokes and are expected to improve the outcomes for acute ischemic strokes in patients with AF.

Key words: Aortic atherosclerosis, Atrial fibrillation, Non-obstructive general angioscopy, Stroke

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We have great interest in the article by Suzuki *et al.*¹⁾ titled “Complex Aortic Arch Atherosclerosis in Acute Ischemic Stroke Patients with Non-Valvular Atrial Fibrillation (AF).” The authors identified 41 (38.7%) transesophageal echocardiography (TEE)-derived complex aortic arch plaques (CAPs) among 106 patients with acute ischemic strokes and AF, suggesting that patients with acute ischemic stroke and AF often had CAPs. The atheromatous lesions at the aortic arch are one of the causes of ischemic strokes. If patients with acute ischemic strokes have both AF and CAPs, it becomes unclear whether the cause of the strokes is due to cardiogenic embolisms from AF or aortogenic embolisms from CAPs.

Cardiogenic strokes due to AF occupied 22% of all-causes of strokes. Embolic strokes of undetermined sources also included latent AF. Therefore, patients with strokes frequently have AF²⁾. On the other hand, several studies have reported that patients with AF

often have co-existing CAPs, which could be an embolic stroke. In an international multicenter registry including 770 patients with AF, CAPs were identified by TEE in 25% of the cohort³⁾. In the study of Suzuki *et al.*, 28% of the cohort did not undergo TEE because they had severe NIHSS scores and an old age. Older individuals have a high risk of CAPs, so if all patients in the study underwent TEE, the percentage of patients with CAPs could have been higher than what was reported.

TEE is a useful imaging modality to detect CAPs; however, its resolution is not sufficient to observe small embolic sources. Non-obstructive general angioscopy (NOGA), which is a newly developed method to evaluate aortic atheromatous plaques⁴⁾ with real-time and high spatial resolution images, can clearly detect the atherosclerotic vulnerability of the aortic wall in more detail than TEE⁵⁾. An interesting report showed that NOGA detected embolic sources as aortogenic embolic strokes in the aortic arch when TEE could not detect CAPs⁶⁾. We, herein, present a NOGA-derived aortic arch

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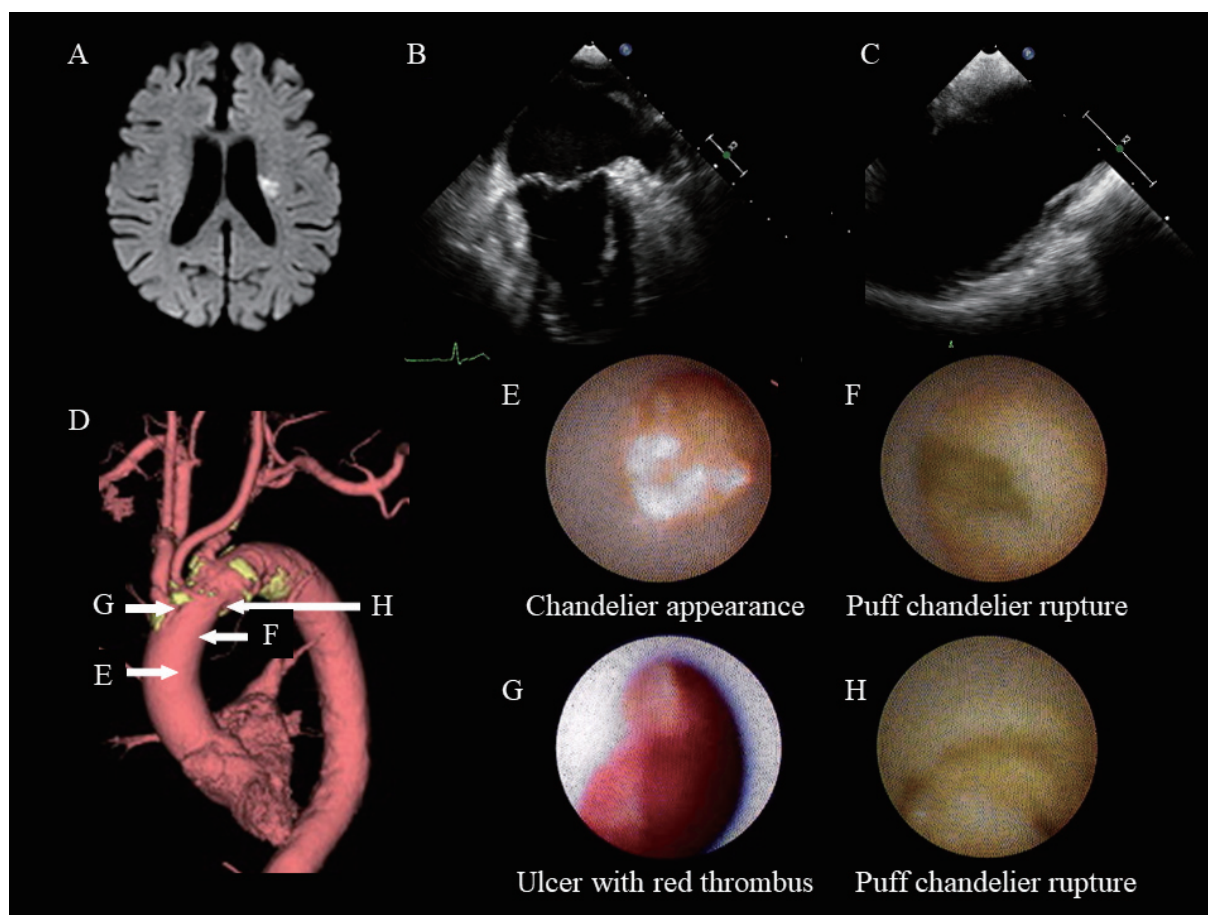


Fig. 1.

Diffusion-weighted magnetic resonance imaging shows high-intensity signals in the left striatum (A). Transesophageal echocardiography shows no thrombus in the left atrium and appendage (B) and indicates atherosclerotic plaques but no complex aortic arch plaque (C). Computed tomography angiography of the aortic arch (D). Non-obstructive general angiography detects atheromatous plaques (E–H).

observation in a case. An 82-year-old man had sudden right incomplete paralysis. Brain magnetic resonance imaging indicated an acute ischemic stroke in the left striatum (**Fig. 1A**). He underwent catheter ablation of paroxysmal AF 15 years before the stroke. No recurrence of AF had been detected until or during hospitalization. His CHADS2 score was low (1: age), and he was taking an anticoagulant (rivaroxaban 10 mg). TEE showed no thrombus in the left atrium and appendage and no CAPs (**Fig. 1B, C**). There were no significant lesions in the carotid or intra-cranial arteries. For seeking the etiology of the stroke, NOGA revealed several atheromatous plaques in the aortic arch (**Fig. 1D–H**). Remarkably, some ruptured plaques scattered atheromatous materials (**Fig. 1F, H, Supplemental Video 1–3**), which could become the cause of his embolic stroke, although TEE detected no CAPs. NOGA has the possibility to diagnose aortic plaques in the aortic arch more accurately than TEE.

Patients with acute ischemic stroke and AF often have CAPs. The cause of strokes could not only be cardiogenic embolisms due to AF but also aortogenic embolisms due to CAPs. Although anticoagulation therapy is the golden standard treatment for cardiogenic embolic strokes due to AF, appropriate therapies for patients with aortogenic embolic strokes have not been fully investigated. The possibility of concomitant CAPs should be considered for patients with stroke and AF. NOGA might help to diagnose atheromatous plaques and embolic materials in the aortic arch. Further studies are needed to elucidate the causes of ischemic strokes and are expected to improve the outcomes for acute ischemic strokes in patients with AF.

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