

Intracardiac Thrombus Following Rivaroxaban Treatment in a Patient with Atrial Fibrillation Associated with Rheumatic Heart Disease

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We report a case of atrial fibrillation with rheumatic heart disease (RHD) who had intracardiac thrombus and cardiogenic cerebral embolism with rivaroxaban therapy. Intracardiac thrombus disappeared after switching from rivaroxaban to warfarin. Patients of RHD have the possibility of gradual progression of valvular disease even if they are old, so we need to distinguish nonvalvular atrial fibrillation from RHD before starting direct oral anticoagulants.

Key Words: Direct oral anticoagulants—atrial fibrillation—rheumatic heart disease—intracardiac thrombus

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Introduction

It is well known Direct Oral Anticoagulants (DOAC) is more effective to nonvalvular atrial fibrillation (NVAf) than warfarin in prevention of recurrence of stroke and hemorrhagic complications,¹ but it is unknown of efficacy of DOAC to atrial fibrillation (Af) with rheumatic heart disease (RHD). We report a case of Af with RHD who had intracardiac thrombus and cardiogenic cerebral embolism with rivaroxaban therapy.

Patient

A 75-year-old man had a cardiogenic cerebral embolism due to NVAf with moderate mitral regurgitation, and

started warfarin therapy 7 years ago. On subsequent annual surveillance, he remained asymptomatic and his mitral regurgitation remained moderate, but his mitral arteriosclerosis progressed gradually on echocardiography. His medicine was switched from warfarin to rivaroxaban (10 mg/day due to creatinine clearance 42.4 mL/minute) 2 months ago to avoid frequent blood tests. His adherence was good. A recent transthoracic echocardiography (TTE) performed 2 months before the commencement of rivaroxaban had shown no intracardiac thrombus or mitral stenosis.

He presented to our hospital due to transient right hemiparesis. Brain magnetic resonance imaging revealed acute infarcts in the right frontal lobe and the left cerebellum. TTE revealed a doming of anterior mitral leaflet (Fig 1A), a shortening of posterior mitral leaflet, an agglutination of mitral valve commissure (Fig 1B), and mild mitral stenosis (mitral valve area: 1.9 cm²), so we diagnosed RHD. It indicated administration of rivaroxaban to the current condition had been wrong when referring to guideline. We started continuous intravenous infusion of heparin and warfarin therapy. On day 6, transesophageal echocardiography demonstrated a thrombus in the left atrial appendage (18 × 15 mm) (Fig 1C). Left atrial appendage thrombus shrank on day 13 (Fig 1D), and disappeared on day 83 (Fig 1E).

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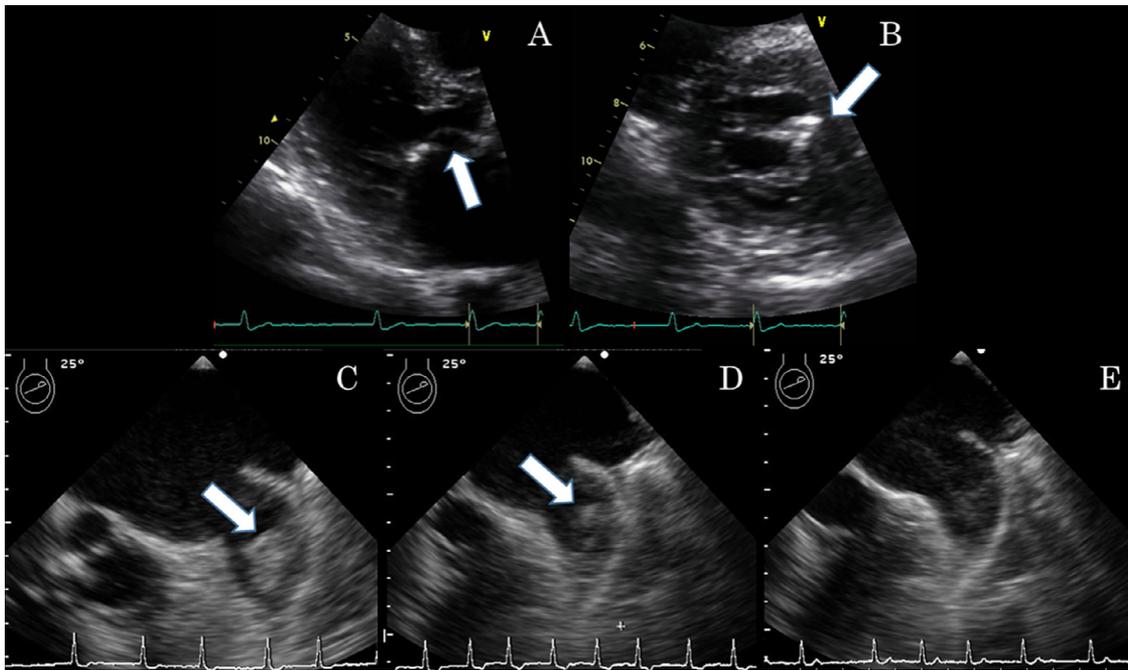


Figure 1. (A) TTE showed a doming of anterior mitral leaflet (arrow), and (B) an agglutination of mitral valve commissure (arrow). (C) Transesophageal echocardiography showed thrombus formation (arrow) in the apex of the left atrial appendage on day 6. (D) On day 13, LAA thrombus shrank and (E) disappeared on day 83.

Discussion

In this case, he had intracardiac thrombus after switching from warfarin to rivaroxaban, and we confirmed reduction and disappearance of intracardiac thrombus after switching from rivaroxaban to warfarin. This fact suggests warfarin is more effective to Af with RHD than DOAC in prevention of embolism. It is reported Af with RHD is more thrombogenic tendency than NVAF,² and we speculated, in such condition, warfarin which effect continuous anticoagulant may be more favorable. The mechanism of thrombolysis is thought to be based on the relative predominance of plasma fibrinolytic activity over thrombin activity.³

In this case, he was diagnosed with NVAF by TTE 7 years ago, but in this time TTE revealed progression of valvular disease and RHD. Patients of RHD have the possibility of gradual progression of valvular disease even if

they are old,⁴ so we need to distinguish NVAF from RHD by TTE. When we diagnose Af with RHD, we should choose warfarin.

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