Abnormal atrial strain with speckle-tracking echocardiography predicts the arrhythmic substrate of atypical right atrial flutter

Hiroshi Kawakami, MD, Takayuki Nagai, MD, Katsuji Inoue, MD, Jitsuo Higaki, MD, Shuntaro Ikeda, MD

From the Division of Cardiology, Department of Cardiology, Pulmonology, Hypertension and Nephrology, Ehime University Graduate School of Medicine, Ehime, Japan.

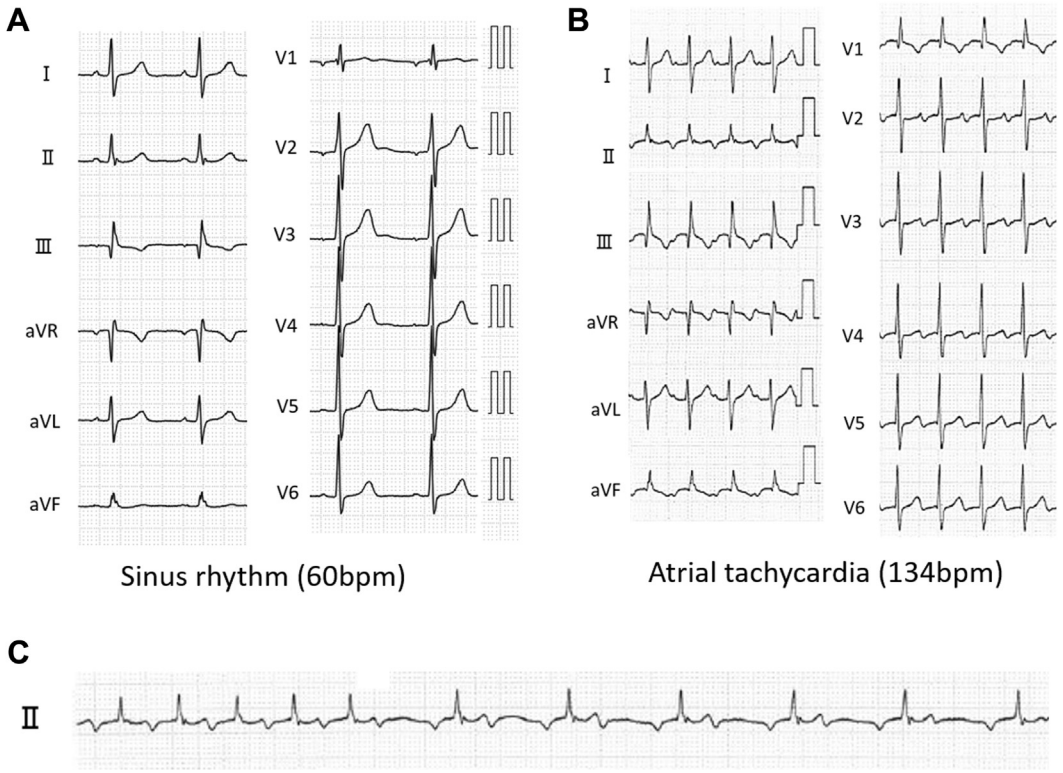

Figure 1 Twelve-lead electrocardiograms (ECG). **A:** The 12-lead ECG during sinus rhythm. **B:** The 12-lead ECG during atrial tachycardia showed long RP' tachycardia. **C:** Tachycardia showed 2:1 atrioventricular conduction when adenosine was administered as an intravenous bolus of 20 mg.

KEYWORDS Atrial macroreentrant tachycardia; Atypical flutter; Catheter ablation; Electroanatomic mapping; Speckle-tracking echocardiography (Heart Rhythm Case Reports 2017;3:251–254)

Address reprint requests and correspondence: Dr Hiroshi Kawakami, Division of Cardiology, Department of Cardiology, Pulmonology, Hypertension and Nephrology, Ehime University Graduate School of Medicine, Shitsukawa, Toon, Ehime 791-0295, Japan. E-mail address: kawaka@m.ehime-u.ac.jp.

Introduction

Atypical atrial flutter is defined as macroreentrant atrial tachycardia (AT) that does not involve the cavotricuspid isthmus; a variety of reentrant circuits may include reentry around the mitral valve annulus or scar tissue within the left and right atrium (RA).[1] Atypical RA flutter was observed in patients who had undergone prior cardiac surgery for obvious structural heart disease.[2–4] In recent years, a few

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KEY TEACHING POINTS

- Atypical atrial flutter in patients without obvious structural heart disease or previous surgical and catheter interventions is rare.
- In this case, the arrhythmogenic substrate of atypical right atrial flutter could be predicted by analyzing atrial strain using speckle-tracking echocardiography before catheter ablation.
- Atrial functional analyses with speckle-tracking echocardiography may help to determine the strategy of electrophysiology study and catheter ablation in patients with atypical atrial flutter.

reports have described atypical RA flutter in patients without obvious structural heart disease or previous cardiac surgery.[5–7] Because of the rarity of this occurrence, the arrhythmogenic substrate of atypical RA flutter in these patients is easily underrecognized before catheter ablation. In the present case, the arrhythmogenic substrate of atypical RA flutter in a patient without prior cardiac surgery was detected with speckle-tracking echocardiography before the procedure.

Case report

A 54-year-old man was admitted to our hospital for AT treatment. Twelve-lead electrocardiogram results obtained during sinus rhythm and tachycardia are shown in Figure 1. He did not have a history of cardiovascular disease or surgical or catheter interventions. Although he had normal left ventricular function and did not have valvular heart disease, RA dilatation was observed on transthoracic echocardiography during sinus rhythm (Figure 2A and B, and supplemental movie, available online). We used speckle-tracking echocardiography for objective assessment of right and left atrial function and found that atrial strain was regionally decreased at the RA free wall (Figure 2C). Electrophysiological study and catheter ablation were performed 1 day after admission. An electroanatomic bipolar voltage map of the RA during

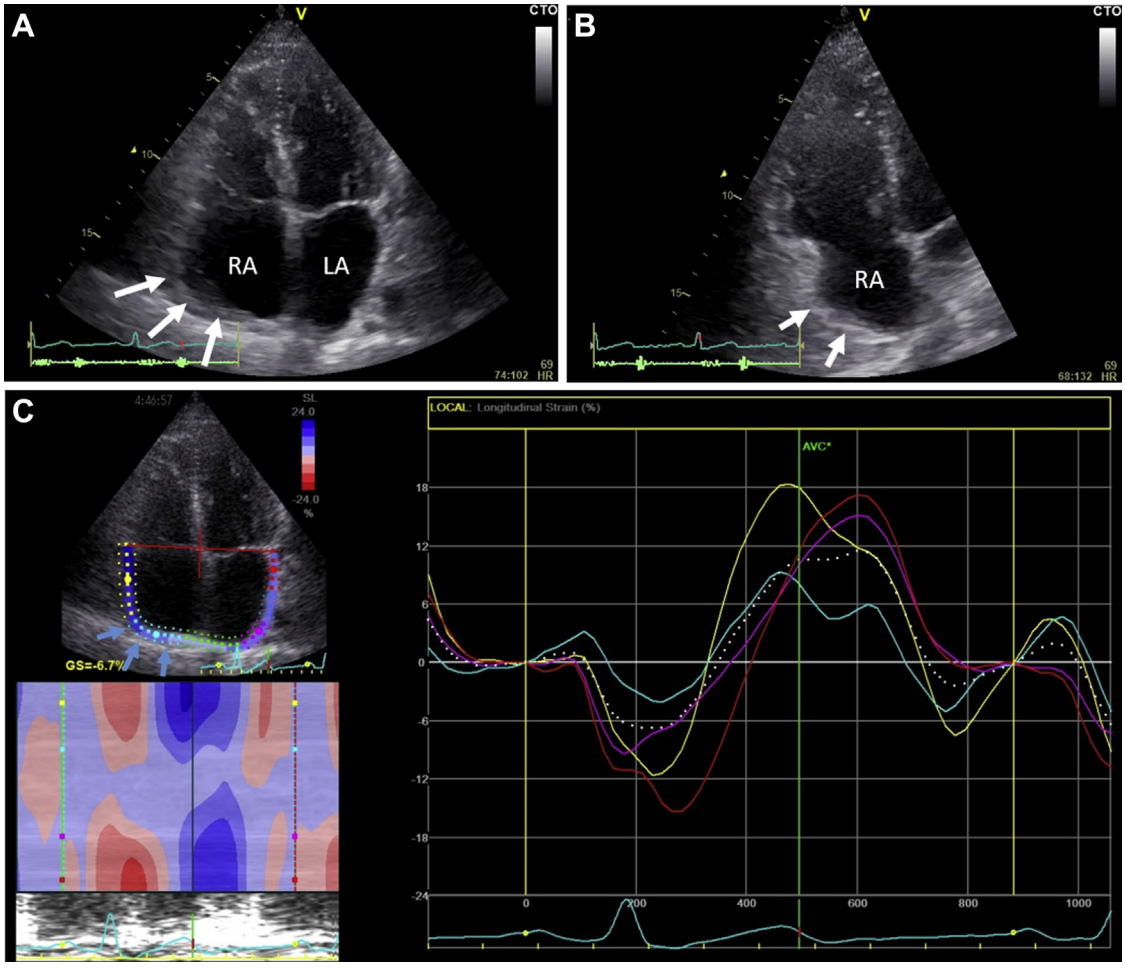

Figure 2 Transthoracic echocardiography during sinus rhythm. **A, B:** Apical 4-chamber view (**A**) and focused view of the right side of the heart (**B**). The right atrium is dilated compared to the left atrium. The right atrial free wall expands outward (*white arrows*). **C:** Atrial strain curves with speckle-tracking echocardiography. Atrial function is regionally decreased at the right atrial free wall (*light blue arrows*). In other atrial areas, including the left atrium, the atrial function is preserved. LA = left atrium; RA = right atrium.

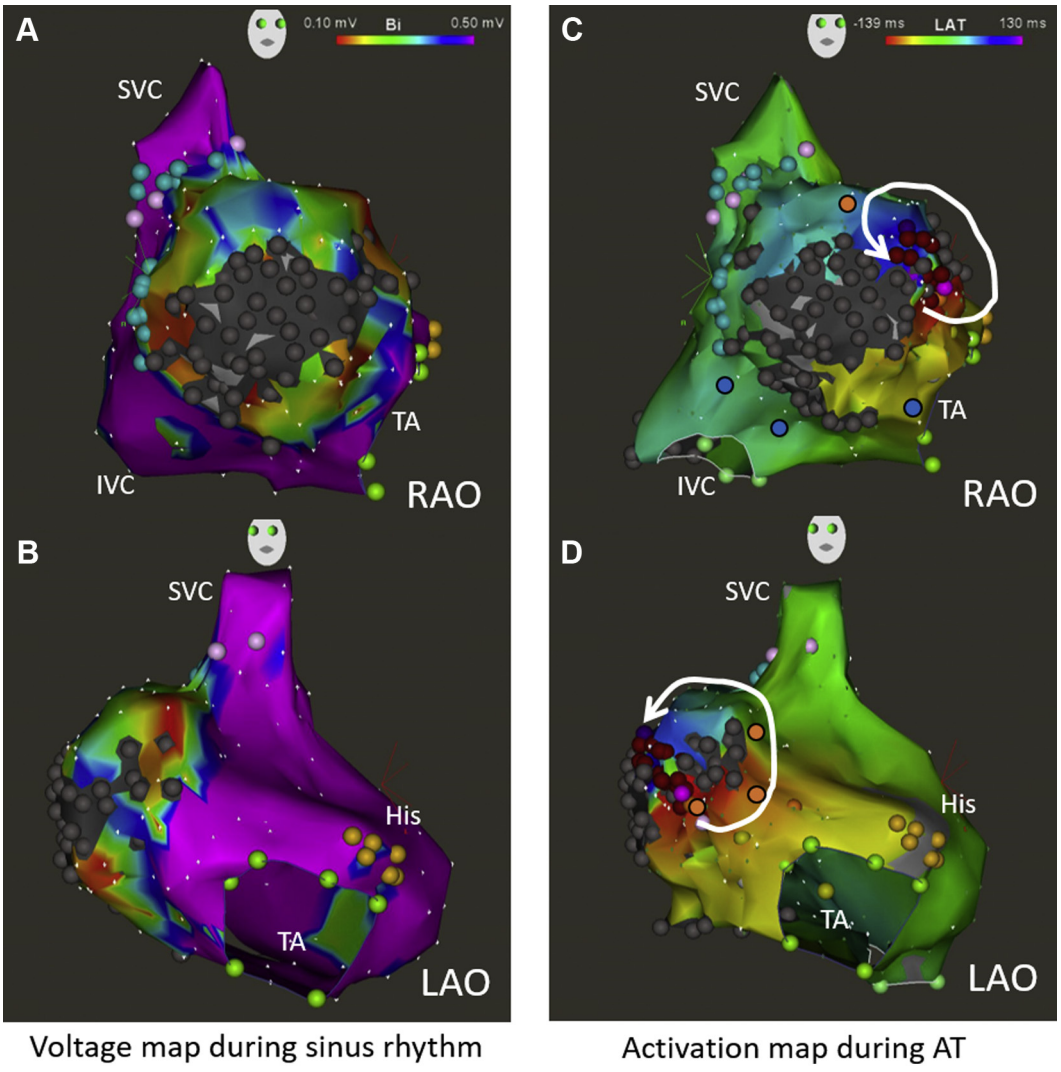

Figure 3 Electroanatomic mapping. **A, B:** Bipolar voltage maps during sinus rhythm. A wide, low-voltage scar area is observed at the right atrial free wall. The other areas have normal voltage. The gray points indicate the dense scar. **C, D:** Activation maps during atrial tachycardia (tachycardia cycle length, 280 ms). Tachycardia circling is noted in a counterclockwise fashion through a "channel" between the 2 areas of the scar zone. The postpacing interval is matched at the orange points, but not at the blue points. The pink point indicates the initial ablation point. LAO = left anterior oblique; IVC = inferior vena cava; RAO = right anterior oblique; SVC = superior vena cava; TA = tricuspid annulus.

sinus rhythm was constructed using CARTO3 (Biosense Webster, Diamond Bar, CA). A large scar (<0.1 mV) and low-voltage area (<0.5 mV) were observed at the RA free wall (Figure 3A and B). The other areas had normal voltage. The low-voltage area was consistent with the abnormal area detected using strain echocardiography.

AT (tachycardia cycle length, 280 ms) was reproducibly induced by programmed stimulation of the RA. The activation map created during tachycardia suggested a reentry activation pattern associated with the scar and low-voltage area (Figure 3C and D). Entrainment mapping was performed during AT. The postpacing interval with an equal AT cycle length is noted at the orange points, but not at the blue points. Therefore, we considered that tachycardia circled in a counterclockwise fashion through a "channel" between the 2 areas of the scar zone (Figure 3C and D). This tachycardia was terminated immediately after delivery of radiofrequency energy at the channel exit (NaviStar ThermoCool; Biosense Webster, Diamond Bar, CA; maximum power, 30 W). Unmappable nonsustained AT was induced by programmed stimulation after clinical AT termination; therefore, bonus ablation was performed at an adjacent site. Finally, no further tachycardia could be induced by programmed stimulation with isoproterenol infusion. No complications were observed and there was no recurrence of AT during 1-year follow-up. After catheter ablation, cardiac computed tomography, magnetic resonance imaging, and fluorine-18-fluorodeoxyglucose positron emission tomography were performed to identify the etiology of the RA low-voltage area; however, there was no specific finding underlying cardiac disease.

Discussion

Atypical RA flutter in patients without obvious structural heart disease or previous surgical or catheter interventions

is rare, and the cause of the scar and low-voltage area in these patients remains speculative.[5–7] In our case, the etiology of regional scarring remained unknown even after various cardiac imaging studies. The majority of these ATs are scar-related macroreentrant tachycardias and typical atrial flutters.[5–7] Stevenson and colleagues[5] reported the first case series of RA macroreentrant tachycardia in 8 patients without prior atrial surgery. They described stable circuits around the scar in 7 patients, stable circuits through a "channel" within the scar in 4 patients, and typical atrial flutter in 5 patients. Two previous reports indicated that typical and atypical RA flutter could sometimes be observed in the same patients.[6,7] Typical RA flutter and other tachycardia were not induced in our case. However, Wieczorek and colleagues[7] reported that further tachycardias including atrial fibrillation could arise during long-term follow-up despite successful catheter ablation in these patients. Therefore, careful follow-up is needed. Whether the presence of atypical atrial flutters indicates underlying atrial cardiomyopathy or focal substrate is not known. The aforementioned study indicated that 5 of 8 patients had mild RA enlargement; however, the relationship between RA enlargement and the arrhythmogenic substrate was not discussed.[5] Recently, some studies demonstrated that speckle-tracking echocardiography is useful for detecting advanced atrial dysfunction in patients with or without cardiomyopathy.[8,9] In addition, some studies indicated that strain echocardiography is useful for predicting the arrhythmic risk of ventricular arrhythmias and atrial fibrillation.[10,11] In the present case, 2-dimensional echocardiography showed the RA dilatation relative to that of the left atrium, but judgment of regional wall motion abnormalities in the atrial wall was inconclusive with visual assessment alone. However, additional analysis with speckle-tracking echocardiography revealed that the atrial strain was regionally decreased at the RA free wall. Atrial strain mainly consists of negative strain due to atrial contraction and positive strain due to atrial reservoir function. Because both of these atrial strains were deteriorated in the RA free wall, the arrhythmogenic substrate of atypical RA flutter could be predicted before catheter ablation. The current findings would have more clinical impact if strain could identify regional wall motion abnormalities in patients without RA dilatation. However, this method has several limitations. First, it is difficult to detect atrial functional abnormalities during tachycardia. Second, we could not reconstruct the 3-dimensional strain images from the 2-dimensional data. The integrated imaging approach using 3-dimensional strain imaging might be capable of investigating the precise correlation between the low-voltage area and the area with regional abnormality.

Conclusion

If atrial enlargement is visually observed by conventional echocardiography in patients with possible atypical atrial flutter, then additional functional analyses with speckle-tracking echocardiography may provide more diagnostic information with regard to the substrate of atypical atrial flutter. Atrial functional analyses with speckle-tracking echocardiography may help to determine the strategy of electrophysiology study and catheter ablation in patients with atypical atrial flutter.

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Appendix

Supplementary data

Supplementary data associated with this article can be found in the online version at http://dx.doi.org/10.1016/j.hrcr.2017.02.001.

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