CASE REPORT

Reversible cardiomyopathy after radiofrequency ablation of 30-year persistent atrial tachycardia

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SUMMARY

Tachycardia-induced cardiomyopathy (TIC) is a reversible form of the left ventricular (LV) systolic dysfunction and is believed to be a relatively acute process. We report a TIC case with a 30-year history of long-lasting persistent atrial tachycardia involving a 44-year-old man previously diagnosed with dilated cardiomyopathy and a low LV ejection fraction (LVEF) of 20%. ECG revealed atrial tachycardia at 110–120 bpm. He was hospitalised with a worsening heart failure. His clinical status was New York Heart Association functional class III, and echocardiography revealed LV dilation and an LVEF of 9%. A two-dimensional speckle-tracking strain measurement revealed LV mechanical dyssynchrony. He underwent radiofrequency ablation for atrial tachycardia. After restoring sinus rhythm, his cardiac symptoms improved immediately. The LV mechanical dyssynchrony decreased a week after ablation, without changes in the LV dilation or LVEF. Thereafter, the LV dilation and systolic function gradually improved, and atrial tachycardia and heart failure remained absent.

BACKGROUND

Tachycardia-induced cardiomyopathy (TIC) is a reversible form of left ventricular (LV) dysfunction in which decreased wall motion and dilation of the LV are caused by chronic tachycardia. The symptoms of TIC improve after HR normalisation. Persistent tachycardia is caused by supraventricular or ventricular tachyarrhythmia and has a variety of symptoms. TIC is believed to be a relatively acute process that acts to limit the enlargement of the LV.¹ Until now, few studies have assessed the association between the duration of tachycardia and reduced cardiac function. In addition, there have been no reported cases of TIC that showed early improvement in LV function after 30 years of impairment after arrhythmia treatment.

CASE PRESENTATION

A 44-year-old Japanese man was admitted to our hospital for mildly symptomatic heart failure in 1980 (at the age of 15 years). A cardiac catheterisation study revealed no coronary artery stenosis, but his LV ejection fraction (LVEF) was 20%, leading to a diagnosis of non-ischaemic dilated cardiomyopathy. His heart rate (HR) during hospitalisation was generally 100–120 bpm according to ECG, and his basal rhythm was diagnosed as compensatory sinus tachycardia due to his low LV function. The patient was discharged from the hospital with prescriptions for 40 mg of furosemide daily and 0.125 mg of digoxin daily. Propranolol was also introduced at

up to 30 mg, but the patient did not tolerate the medication due to fatigue. He chose to discontinue the prescribed medications because he experienced no symptom progression. Although an HR of 100–120 bpm persisted, he was in the New York Heart Association (NYHA) functional class I, and his plasma brain natriuretic peptide (BNP) level remained below 100 pg/mL until 2009.

However, he was referred to our hospital again in 2010 because of the development of respiratory discomfort on exertion, and he was in NYHA functional class II at that time. A 12-lead ECG revealed atrial tachycardia at 120–140 bpm, and an ambulatory ECG revealed persistent atrial tachycardia throughout the day. He was therefore restarted on 40 mg of furosemide daily and 0.125 mg of digoxin daily. However, his symptoms worsened, and he was admitted to our hospital. On admission, he was in NYHA functional class III, and his blood pressure was 98/60 mm Hg. ECG revealed atrial tachycardia at 144 bpm (Figure 1). Chest radiography revealed cardiomegaly, and his plasma BNP level had increased to 1807 pg/mL. Echocardiography revealed significant LV dilation and systolic dysfunction; his LV dimension in diastole (LVDD) was 77 mm, and his LVEF (according to the biplane Simpson's method) was 9% (figure 2). After admission, he reported laboured breathing, and his blood pressure decreased to 76 mm Hg. Intravenous dobutamine was initiated due to worsening heart failure, and intravenous heparin was also initiated.

INVESTIGATIONS

A 12-lead ECG revealed atrial tachycardia with a positive P wave in leads I, II, III and aVF; a biphasic P wave in lead V1; left axis deviation (−45°) and poor R wave progression in V2–4; and a widened QRS complex (120 ms) and T wave inversion in leads I, aVL, V5 and V6 (figure 1).

Cardiac catheterisation was performed, and coronary angiography revealed no significant stenosis. Left ventriculography revealed enlargement (LV end-diastolic volume index 170 mL/m²) and impaired LV systolic function (LVEF 26%). A right ventricular endomyocardial biopsy was also performed. A histopathological examination revealed mildly hypertrophied myocytes and myocardial degeneration with interstitial fibrosis but no inflammatory cell infiltration, which were not remarkably different from the findings obtained for the patient in 1980 (figure 3).

Two-dimensional (2D) speckle-tracking strain analysis revealed 224 ms of anteroposterior to posterior wall strain delay (values ≥130 ms indicate

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Figure 1 Twelve-lead ECGs showing atrial tachycardia with a positive P wave in leads I, II, III and aVF and a biphasic P wave in lead V1 suggested an origin in the lateral right atrium.

intraventricular mechanical dyssynchrony) despite a narrow QRS duration (figure 4).

TREATMENT

The patient underwent radiofrequency ablation (RFA) to treat the tachycardia. Based on the potentials measured using a duodecapolar catheter, the site of earliest activation was considered to be the right atrium, originating from the superior vena cava (SVC) and the high right atrium. The excitement propagation was confirmed using developing geometry with an EnSite balloon catheter placed in the upper right atrium. EnSite non-contact mapping-guided activation revealed a focal pattern from the high lateral right atrium (figure 5A). A duodecapolar catheter was placed between the septum of the right atrium and the SVC to obtain detailed information on the adjacent area of the earliest activation site in the atrium. The site of origin was identified at the high lateral right atrium (figure 5B).

The area adjacent to the earliest activation site was mapped using a reference to the earliest site of activation on EnSite. Immediately after contact with the high lateral right atrium, atrial tachycardia recurred. RFA was performed at this site (figure 5C). Atrial tachycardia ceased 4 s after the start of RFA, and sinus rhythm returned. There was no subsequent recurrence

of atrial tachycardia, and mapping during sinus rhythm using the EnSite balloon confirmed that the sinus node and current-carrying site were distant from each other.

OUTCOME AND FOLLOW-UP

The patient was treated with perindopril, carvedilol and warfarin after RFA. His ECG showed sinus rhythm, normalisation of the QRS axis ($+65^\circ$) and shortening of the QRS duration (100 ms; figure 6), with no recurrence of atrial tachycardia 2 years after RFA.

One week after RFA, echocardiography revealed no apparent changes in the LVDD (74 mm) or LVEF (14%). However, the 2D speckle-tracking strain analysis revealed a decrease in the degree of intraventricular mechanical dyssynchrony (anteroseptal to posterior wall motion delay: 116 ms; figure 7). His cardiac status was NYHA functional class II, and his plasma BNP level was 631 pg/mL.

Four months after RFA, echocardiography revealed an improvement in the LVDD (57 mm) and LVEF (43%); his cardiac status was NYHA functional class I; and his plasma BNP level was 14 pg/mL. Warfarin was discontinued 5 months after RFA. One year after RFA, echocardiography revealed a normal LVDD (51 mm) and an increased LVEF (48%; figure 8), and his

Long-Axis
2D

M-mode

LVDD	77mm
LVDS	69mm
LVEF(Simpson)	9%

Figure 2 Echocardiography during atrial tachycardia. IVS, interventricular septum; LV, left ventricle; LVEF, left ventricular ejection fraction; LVDD, left ventricular internal dimension in diastole; LVDS, left ventricular internal dimension in systole; PW, posterior wall.

plasma BNP level was 6 pg/mL. Currently, the patient is taking 5 mg of carvedilol and 2 mg of perindopril, both daily, and his cardiac status has been maintained for more than 2 years following RFA.

DISCUSSION

TIC was first reported in 1913 in a case of cardiomegaly and heart failure of unknown aetiology.² In 1986, Packer *et al*³ reported the improvement of cardiac function in a patient with cardiomyopathy due to persistent tachycardia. They described seven patients with incessant tachycardia that had been present for 6 months to 6 years (mean, 2 years) and one patient with supraventricular tachycardia that was virtually uninterrupted, as documented by continuous monitoring for 15 years. TIC is a reversible condition in which decreased wall motion and dilation of LV are caused by chronic tachyarrhythmia; however, the symptoms resolve (or at least improve) after heart rate normalisation.^{1 4 5} Van Gelder *et al*⁶ reported that LV function essentially recovered 4 weeks after cardioversion (and significant recovery was observed 1 week after cardioversion) and remained normal for 6 months thereafter. In the present case, a patient with a 30-year history of tachyarrhythmia with 100 or more beats/min according to ECG developed LV dysfunction, resulting in worsening heart failure and hospitalisation. After restoring sinus rhythm, his cardiac function gradually improved and had normalised 1 year later. Therefore, we diagnosed this case as TIC. In this case, interestingly, the LV mechanical dyssynchrony decreased rapidly after RFA, leading to an improvement in the standard indicators of LV systolic function, such as the LV size and LVEF. The ECG showed a widened QRS complex in the left anterior fascicular block pattern during atrial tachycardia, but it disappeared after RFA. This intraventricular conduction delay may have resulted from LV mechanical dyssynchrony and heart failure. Furthermore, Fauchier *et al*⁷ reported that a high proportion of patients with left anterior hemiblock, which may be related to a proximal or fascicular conduction delay, exhibited a marked intra-LV dyssynchrony. Left anterior hemiblock, which appeared during atrial tachycardia, may have contributed to LV dyssynchrony in this patient. Until now, it is still not well understood if LV mechanical dyssynchrony exists in patients with TIC or affects the relationship between decreased dyssynchrony and reverse remodelling of TIC. In this case, mechanical dyssynchrony may have contributed partially to LV dysfunction.

The differential diagnosis of TIC instead of dilated cardiomyopathy is sometimes difficult to determine when patients present with reduced cardiac function. Fujino *et al* reported that

A

200 µm

B

200 µm

Figure 3 Endomyocardial biopsy specimens obtained in 1980 (A) and in 2010 (B; H&E).

Figure 4 Two-dimensional speckle-tracking imaging before radiofrequency ablation. Regional radial strain imaging (distinctively coloured area for 6 left ventricular wall segments; left panel) and time-strain curves (right panel). Strain is a measure of myocardial deformation and is defined as the percentage change from the original dimension of the object. It analyses motion by tracking speckles (natural acoustic markers) within the two-dimensional ultrasonic image from frame to frame. The systolic strain is positive. The time difference (arrow) between the time to peak strain in the antero-septal wall and that in the posterior wall was 224 ms.

TIC was defined as the rapid improvement of cardiac function (over the course of less than 6 months), whereas dilated cardiomyopathy was defined by persistent LV dysfunction after the management of tachycardia with atrial fibrillation. Fujino *et al*⁸ also noted that the LV size was significantly smaller in TIC than in dilated cardiomyopathy (LVDD, 57 ± 7 mm vs 63 ± 9 mm) in a comparison of the two conditions in 63 heart failure patients with LV dysfunction and atrial fibrillation. Jeong *et al*⁹ also reported that an LVDD <66 mm in patients with an LVEF of 30% or less could be used as an index to facilitate the differential diagnosis of TIC from dilated cardiomyopathy. In most

patients with TIC, the LV dimension is small because heart failure occurs suddenly in association with tachycardia before the development of LV dilation. However, our patient presented a large LV dimension (LVDD 77 mm) compared with those of previously reported TIC cases. The patient may have had sufficient time to develop LV dilation during his long-lasting (30 years) tachycardia. Interestingly, his histopathological examination revealed only mild changes, despite the long disease duration and long-lasting impairment of LV function. Although the BNP value was not shown to differ between TIC and dilated cardiomyopathy,⁸ it decreased rapidly after restoring sinus

Figure 5 Activation map of atrial tachycardia originating from the high lateral right atrium. Activation map of atrial tachycardia in the anteroposterior (AP) view. An EnSite non-contact mapping system displays a focal pattern from the high lateral right atrium (A). A negative electric potential recorded using unipolar electrodes is shown in colour ranging from blue to white, according to the potential difference. The point of the most negative electric potential is shown in white. This point is affected by the electric potential earlier than the other regions and is the arrhythmic origin. AP view of the ablation catheter at the successful ablation site on the high lateral side of the right atrium (B). During tachycardia, a distal unipolar ECG of the ablation catheter (ABL) indicates the earliest onset (arrow head) (C). Radiofrequency delivery to this earliest activation site terminated the tachycardia. Leads I, II and V1 refer to the surface leads; ABL, ablation catheter; ABL uni refer to the electrodes of the unipolar ablation catheter; ABL d and ABL p refer to the electrodes on the distal and proximal portions of the bipolar ablation catheter, respectively; CS, coronary sinus; CS d and CS p refer to the distal and proximal coronary sinus electrodes, respectively; HRA, high right atrium; HRA d and HRA p refer to the distal and proximal high right atrium electrodes, respectively; RFA, radiofrequency ablation; TV, tricuspid valve; TV d and TV p refer to the distal and proximal tricuspid valve electrodes, respectively.

Figure 6 Twelve-lead ECGs showing sinus rhythm after radiofrequency ablation. A widened QRS complex in the left anterior fascicular block pattern during atrial tachycardia disappeared after treatment.

rhythm in patients with TIC.¹⁰ In this case, the plasma BNP level decreased after RFA and normalised 4 months later.

The control of tachyarrhythmia during the management of TIC is essential. Recently, RFA was introduced as a powerful tool for symptom cessation and the control of supraventricular or ventricular tachyarrhythmia. Progress in intracardiac mapping systems has also helped to identify the exact locations and mechanisms of arrhythmias.

Atrial tachycardia has not been associated with increased risk of stroke or systemic embolism, and anticoagulant therapy is not usually required. Recently, the ASSERT study indicated that atrial tachyarrhythmia was associated with increased risk of ischaemic stroke or systemic embolism in elderly patients with pacing devices but no history of atrial fibrillation and that longer durations increased the rate of strokes or embolic events.¹¹ The MOST trial indicated that an atrial rate

Figure 7 Two-dimensional speckle-tracking imaging 1 week after radiofrequency ablation. The time difference (arrow) between the time to peak strain in the antero-septal wall and that in the posterior wall was reduced from 224 m/s to 116 m/s after treatment.

Long-Axis
2D

M-mode

LVDD	51mm
LVDS	37mm
LVEF(Simpson)	48%

Figure 8 Echocardiography 1 year after radiofrequency ablation. IVS, interventricular septum; LV, left ventricle; LVEF, left ventricular ejection fraction; LVDD, left ventricular internal dimension in diastole; LVDS, left ventricular internal dimension in systole; PW, posterior wall.

Learning points

- Tachycardia-induced cardiomyopathy is a reversible form of the left ventricular dysfunction caused by chronic tachycardia.
- In tachycardia-induced cardiomyopathy, cardiac function is typically improved within 4 weeks after the heart rate normalisation and has returned to baseline within 6 months.
- Cardiac dysfunction due to an extremely long period of persistent tachycardia can improve rapidly with appropriate tachyarrhythmia treatment.
- The left ventricular mechanical dyssynchrony may contribute to cardiac dysfunction in tachycardia-induced cardiomyopathy.

>220 bpm increased the risk of stroke in elderly patients with pacemakers.¹² However, there has been no evidence that atrial tachycardia, including the relatively slower atrial rate cases (such as that of the present patient), is associated with increased risk of stroke. Therefore, anticoagulant therapy should not routinely be required for patients with sustained atrial tachyarrhythmia unless low LVEF and heart failure (ie, conventional risk factors for stroke) are present. The present patient received anticoagulants from 3 weeks before to 5 months after RFA.

We described a patient with a 30-year history of TIC and impaired LV systolic function. His cardiac status improved rapidly with the restoration of sinus rhythm using RFA. This case suggests that cardiac function may improve with appropriate arrhythmia treatment, even in patients with impaired LV function due to an extremely prolonged (30-year) period of persistent tachycardia.

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Competing interests None.

Patient consent Obtained.

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