CASE REPORT

Improvement of cardiac function and neurological remodeling in a patient with tachycardia-induced cardiomyopathy after catheter ablation

Chikaya Omichi (MD, PhD)a,*, Takeshi Tanaka (MD, PhD)b, Yoshiko Kakizawa (MD)b, Ayako Yamada (MD)b, Yasuhiro Ishii (MD, PhD)b, Hirotaka Nagashima (MD, PhD)b, Katsuo Kanmatsuse (MD, PhD, FJCC)b, Masahiro Endo (MD, PhD, FJCC)b

a Division of Cardiology, Cardiovascular Center, Kitahara Hospital, 1-7-23 Owada-cho, Hachioji, Tokyo 192-0045, Japan
b Division of Cardiology, Osaki Hospital, Tokyo Heart Center, Tokyo, Japan

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Abstract
Incessant ventricular tachycardia and long-standing ectopic beats lead to tachycardia-induced cardiomyopathy. Catheter ablation eliminates ventricular tachycardia and reverses left ventricular (LV) dysfunction. 201-Thallium (201Tl) scintigraphy demonstrates perfusion defects with ischemic cardiomyopathy. Reversible perfusion defects are observed even in non-ischemic cardiomyopathy, related to regional flow or metabolism derangements. 123-I-metaiodobenzylguanidine (123I-MIBG) scintigraphy delineates regional cardiac sympathetic denervation and heterogeneity. We demonstrated the progression of tachycardia-induced cardiomyopathy in a patient with idiopathic LV outflow tract tachycardia using 201Tl and 123I-MIBG scintigraphic findings. Regional defects were reversed predominantly in the basal interventricular septal wall in 201Tl scintigraphy and 123I-MIBG scintigraphic findings. This report suggests that incessant ventricular tachycardia or long-standing ventricular ectopic beats may develop adverse myocardial remodeling and sympathetic neurological remodeling. Treatment with catheter ablation for tachycardia-induced cardiomyopathy can reverse sympathetic neurological remodeling as well as myocardial structural remodeling.

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* Corresponding author. Tel.: +81 42 645 1110; fax: +81 42 645 1140.
E-mail address: omichi@kitaharahosp.com (C. Omichi).

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Introduction

Incessant ventricular tachycardia (VT) and longstanding ectopic beats lead to tachycardia-induced cardiomyopathy. Catheter ablation eliminates ventricular arrhythmia and reverses left ventricular (LV) dysfunction. We evaluated reverse remodeling processes in a patient with tachycardia-induced cardiomyopathy after catheter ablation of LV outflow tract VT, using 201-Thallium (201Tl) and 123-I-metaiodobenzylguanidine (123I-MIBG) scintigraphy.

Case report

An 80-year-old man visited our hospital and complained of dyspnea on exertion. Twelve-lead surface ECG showed ventricular ectopic beats of left bundle branch block pattern with inferior-axis. Holter ECG recorded 50,212 ventricular ectopic beats per day and incessant VT. Echocardiography showed LV global dysfunction from LV ejection fraction (EF) 30% to 16% during 6 months follow-up despite optimized medical therapy including beta-blocker, atenolol, or carvedilol. Brain natriuretic peptide (BNP) level increased from 191 to 792 pg/ml over 6 months of follow-up. He agreed with a treatment of catheter ablation for ventricular tachyarrrhythmias. The processes of adverse and reverse remodeling were assessed before and after catheter ablation by 201Tl and 123I-MIBG scintigraphy.

Coronary angiography was performed before catheter ablation and no significant stenosis of coronary artery was observed. Left ventriculography showed severe diffuse LV hypokinesis. Sustained VT was not induced by extra-ventricular stimuli from RV apex and LV outflow tract. He underwent catheter ablation of LV outflow tract ectopic beats. Paced and clinical VT QRS complexes were similar regarding the pattern of left bundle branch block, cardiac axis, and R/S relationship in precordial leads (Fig. 1A). Intensive mapping with a 7Fr, 4 mm-tip ablation catheter revealed that the local

Fig. 1 (A) Paced and clinical VT QRS complexes were similar regarding the pattern of left bundle branch block, cardiac axis, and R/S relationship in precordial leads. (B) Intracardiac recordings show ectopic beats of LV outflow occurring spontaneously. The initial deflection from ablation catheter in LVOT preceded the beginning of the QRS by 23 ms. All local ventricular activations within RVOT were delayed. (C) Successful ablation site: Fluoroscopic images show the site of successful ablation just beneath aortic valve in the right anterior oblique (RAO 30°) and left anterior oblique (LAO 50°) projections. Radiofrequency ablation terminated LV outflow ectopic beats. ABL, Ablation catheter; ABLuni, ablation catheter unipolar; AoP, aortic pressure; LAO, left anterior oblique projection; PVC, premature ventricular ectopic beat; RAO, right anterior oblique projection; LVOT, left ventricular outflow tract; RVOT, right ventricular outflow tract; Stim, stimulus.
ventricular ectopic beat preceded the onset of the QRS by 23 ms just beneath aortic valve, followed by successful ablation despite a lack of optimal pace map score (Fig. 1B and C). Holter ECG demonstrated no further episodes of incessant VT or ectopic beats after catheter ablation.

Scintigraphic assessments of reverse ventricular remodeling

We performed early and delayed cardiac $^{201}$Tl and $^{123}$I-MIBG scintigraphy at rest before and after catheter ablation. We used a single-head rotating gamma camera equipped with a low-energy, general-purpose collimator (ECAM Signature, Toshiba, Tokyo, Japan). $^{201}$Tl scintigraphy can demonstrate perfusion defects with ischemic cardiomyopathy. In non-ischemic cardiomyopathy reversible perfusion defects can be observed corresponding to potential regional flow or metabolic derangements. $^{123}$I-MIBG scintigraphy can delineate regional cardiac sympathetic denervation and heterogeneity. Early imaging began 20 min after an intravenous injection of 111 MBq of $^{201}$Tl and $^{123}$I-MIBG. Before STATIC acquisition, an anterior chest image was acquired for 180 s in a $512 \times 512$ matrix. Immediately after planar imaging, 60 images were obtained in a $64 \times 64$ matrix for 40 s, with a 360° rotation and an energy window of 20% centered at 70 keV for $^{201}$Tl and 158 keV for $^{123}$I-MIBG. Tomographic images were reconstructed using a Butterworth filter with a cutoff frequency of 0.55 cycle/cm and an order of 8.

$^{201}$Tl scintigraphy demonstrated regional defects predominantly in the basal interventricular septal wall 6 months before catheter ablation (Fig. 2A, arrows) and the regional defect further progressed to the anterolateral wall with LV dilatation immediately before catheter ablation (B, arrows). The regional uptake predominantly increased in the basal interventricular septal wall 1 month after catheter ablation (C, arrows). The regional uptake reversed with concomitant improvement of LVEF from 16% to 47% after catheter ablation. ANT, Anterior wall; INF, inferior wall; LAT, lateral wall; SEP, septal wall; LVEF, left ventricular ejection fraction. Anterior, lateral, and septal regions were further defined as shown in Table 1.

Fig. 2 $^{201}$Tl scintigraphy SPECT. Short axial images demonstrate lower regional uptake in the basal interventricular septal wall 6 months before catheter ablation (A, arrows). The regional defect further progressed to the anterolateral wall with LV dilatation immediately before catheter ablation (B, arrows). The regional uptake predominantly increased in the basal interventricular septal wall 1 month after catheter ablation (C, arrows). The regional uptake reversed with concomitant improvement of LVEF from 16% to 47% after catheter ablation. ANT, Anterior wall; INF, inferior wall; LAT, lateral wall; SEP, septal wall; LVEF, left ventricular ejection fraction.
Improvement of functional and neurological remodeling after catheter ablation

Fig. 3 123I-MIBG scintigraphy SPECT. Regional uptake in the basal interventricular septal wall decreased 6 months before catheter ablation (A, arrows). The regional uptake improved predominantly in the interventricular septal basal wall 1 month after catheter ablation (B, arrows). ANT, Anterior wall; INF, inferior wall; LAT, lateral wall; SEP, septal wall.

ters. 123I-MIBG scintigraphy similarly demonstrated that the predominant regional defect in the basal interventricular septal wall reversed 1 month after catheter ablation (Fig. 3A and B, arrows).

These results suggest that negative LV remodeling occurred predominantly in the basal interventricular septal wall before catheter ablation of LV outflow tract ectopic beats and reversed after catheter ablation. Cardiac sympathetic neurological dysfunction also reversed after catheter ablation.

Discussion

The present case demonstrated the reverse processes of cardiac dysfunction and neurological dysfunction after catheter ablation of incessant VT and ectopic beats. Cardiac sympathetic denervation, “neurological remodeling” reversed after catheter ablation in the assessment of 123I-MIBG scintigraphy.

Prior reports described cardiomyopathy due to supraventricular tachycardia and reversibility after catheter ablation [1,2]. However, processes of adverse and reverse ventricular remodeling have not been fully understood in patients with tachycardia-induced cardiomyopathy. A few earlier reports demonstrated successful ablation of LV outflow tract ectopic beats or VT [3,4]. Our present report showed the reverse processes of LV remodeling after catheter ablation in both 201Tl and 123I-MIBG scintigraphic assessments. Improvement in the scintigraphic findings was associated with increase in the LVEF and decrease in the BNP level, which was concordant with processes of positive LV remodeling. Echocardiography identified global LV dysfunction and the improvement of LVEF after catheter ablation. However, we were not able to identify the LV segmental delay before ablation in the echocardiography. It is possible that the negative global LV remodeling was accomplished in tachycardia-induced cardiomyopathy at the time of echocardiography and consecutive echocardiography was needed to identify the LV segmental delay throughout the clinical course. The new imaging technique, tissue Doppler or three-dimensional echocardiography may provide accurate assessment of LV segmental delay.

Plasma levels of BNP correlate with LV remodeling [5]. In this patient, BNP levels significantly decreased after catheter ablation in association with LV functional remodeling and neurological remodeling. The decrease in BNP levels may be
associated with both functional and neurological normalization.

Anti-tachycardia therapies have been reported to improve altered cardiac adrenergic function and systolic dysfunction in patients with tachycardia-induced cardiomyopathy [6]. The study included one patient who had catheter ablation of VT and ventricular ectopic beats for tachycardia-induced cardiomyopathy. Anti-tachycardia therapy with catheter ablation and medications improved cardiac adrenergic function in that study. Our report is consistent with the study that sympathetic denervation, neurological remodeling reversed 2 months after catheter ablation.

VT and frequent ectopic beats reliably can reproduce the hemodynamic, structural, and neurohumoral alterations typically seen in humans with heart failure [7]. The autonomic nervous system has been shown to exert profound effects on ventricular arrhythmogenesis [8]. The heterogeneous sympathetic denervation occurs during some stage of adverse ventricular remodeling and is likely important in ventricular arrhythmogenesis. The heterogeneous sympathetic denervation of the ventricle results in altered sympathovagal balance and creates the milieu for sustained VT and fibrillation [9]. Our scintigraphic findings suggest that patients with LV outflow tract VT and ectopic beats were more susceptible to development of regional perfusion defect and heterogeneous sympathetic denervation. Reversal of regional defects may be caused by regional flow or metabolism derangements, related to tachycardia-induced microcirculatory myocardial impairment. Regional cardiac sympathetic denervation and heterogeneity in addition to ventricular structural remodeling may be further potential factors, which could induce malignant ventricular arrhythmias. Catheter ablation may reverse ventricular structural remodeling and sympathetic heterogeneity to reduce risk of further malignant ventricular arrhythmias.

There were potential limitations in the present study. Our study suffered from one limited sampling case and provided only limited information. The result of one case may not apply for all patients with outflow tract ventricular ectopic beats or ventricular tachyarrhythmias. Patients with left bundle branch block (LBBB) exhibit abnormal septal motion that may limit the interpretation of myocardial scintigraphy and can demonstrate false-positive abnormalities, especially in the septum.

In the present case, it is not clear whether ventricular ectopy with LBBB type morphology could result in artifact septal defects. We did not perform electrophysiological study when the reverse remodeling was observed after catheter ablation. Therefore, we could not evaluate electrical remodeling, after LV remodeling and neurological remodeling reversed.

In conclusion, this report indicates that VT and long-standing ectopic beats can induce not only LV functional remodeling but also sympathetic neurological remodeling. Catheter ablation may reverse the mechanical remodeling and neurological remodeling to prevent further malignant ventricular arrhythmias.

References