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Serial Histopathologic Myocardial Findings in a Patient With Ectopic Atrial Tachycardia-Induced Cardiomyopathy

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Abstract

A 17-year-old woman was found to have ectopic atrial tachycardia by her physician. Echocardiography and cardiac catheterization revealed findings resembling dilated cardiomyopathy at the time of initial presentation. The tachycardia was controlled with atenolol only at a dose of 50 mg/day. However, at the age of 22, the presence of ectopic atrial tachycardia was once again confirmed. We successfully performed catheter ablation for persistent ectopic atrial tachycardia. Serial echocardiographic findings showed the left ventricular dimension and function appeared to return to normal 1 year postablation. However, despite pharmacologic control and catheter ablation therapy, histopathology revealed myocardial fibrosis presumably representing permanent damage of the heart secondary to tachycardia 1 year postablation.

Key Words

Cardiomyopathies (tachycardia-induced cardiomyopathy), Arrhythmias, treatment of (catheter ablation), Myocardium (myocardial fibrosis), Pathology (endomyocardial biopsy)

INTRODUCTION

Catheter ablation has been used for the treatment of paroxysmal supraventricular tachycardia and is considered to be curative therapy[1–3]. We successfully performed catheter ablation of persistent ectopic atrial tachycardia in a patient with tachycardia-induced cardiomyopathy. The patient underwent myocardial biopsy and assessment of left ventricular function.

CASE REPORT

A 17-year-old girl was found to have atrial tachycardia by her physician. Echocardiography and cardiac catheterization revealed findings resembling dilated cardiomyopathy at initial presentation. The tachycardia was controlled only with atenolol

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Selected abbreviations and acronyms

ECG=electrocardiography

at a dose of 50 mg, but other medications, including verapamil, digoxin, and metoprolol, were ineffective. Furthermore, cardioversion was ineffective. By the age of 18, repeat examination revealed the absence of left ventricular dilatation. Treatment with atenolol continued at a dose of 50 mg/day. At the age of 22, the patient developed general fatigue and consulted a physician.

The presence of atrial tachycardia was once again confirmed, and verapamil was prescribed in addition to atenolol. However, these were ineffective. She was subsequently referred to our hospital and was diagnosed as having an ectopic atrial tachycardia followed by tachycardia-induced cardiomyopathy.

Physical examination revealed no other abnormal findings. Laboratory tests also revealed no abnormalities.

Catheter ablation was performed using two ablation catheters (Webster, 7F 2.5 inch curve type B, grooved tip and large tip). The ablator (Novaflame, 13.56 MHz) was manufactured by Internova. The final successful ablation site was located at the posterior septum near the foramen ovale by femoral approach (5–35 W, 60 sec). Sinus rhythm appeared after a 15 W application. A 30 min isoproterenol drip infusion failed to induce ectopic atrial tachycardia.

Electrocardiography (ECG) recorded at the age of 17 demonstrated atrial tachycardia (rate of 170 bpm) (**Fig. 1–upper**). After treatment with atenolol at a dose of 50 mg/day, her heart rate returned to normal. The P wave polarity appeared to be somewhat different from that noted during normal sinus rhythm in lead aVF. Prior to ablation at the age of 22 (in 1994), recurrence of atrial tachycardia was noted at a heart rate of 150–180 bpm with Wenckebach type AV block (**Fig. 1–lower–left**). Holter monitoring demonstrated atrial tachycardia that persisted throughout the day (heart rate 150–180 bpm). On the day following ablation, the patient had normal sinus rhythm with P wave morphology somewhat different from that of atrial tachycardia (**Fig. 1–lower–middle**). Seven days after ablation, the heart rate was normal, although P wave appeared to be somewhat different from normal sinus rhythm (**Fig. 1–lower–right**). During sinus rhythm, small change of P wave morphology seemed to occur.

Echocardiography showed left ventricular dilatation with reduced wall motion at the age of 17 (left ventricular end-diastolic dimension 61 mm, ejection fraction 27%; **Fig. 2–upper–left**). At the age of 18 (in 1989) after treatment with atenolol, the left ventricular dimension and function returned to normal (left ventricular end-diastolic dimension 50 mm, ejection fraction 57%; **Fig. 2–upper–right**). At the age of 22 (**Fig. 2–lower**), prior to catheter ablation, left ventricular dilatation and depressed ventricular function were once again noted (left ventricular end-diastolic dimension 57 mm, ejection fraction 41%). Two days after catheter ablation, the left ventricular dimension almost returned to normal. However, the ejection fraction remained slightly depressed (ejection fraction 54%) until 7 weeks after catheter ablation (ejection fraction 60%).

Endomyocardial biopsy findings of two samples of myocardium were obtained at each biopsy procedure from both right and left ventricles. At the age of 17 light microscopic examination (hematoxylin-eosin stain, ×200) of the left ventricular endomyocardial biopsy specimen demonstrated mild interstitial tissue fibrosis and mild myocardial cell hypertrophy (**Fig. 3–a**). The second left ventricular endomyocardial biopsy specimen at the age of 22 demonstrated advanced interstitial fibrosis, mild inflammatory cell infiltration, endocardial thickening and myocardial hypertrophy (**Fig. 3–b**). The third left and right ventricular endomyocardial biopsy done a year later after catheter ablation was almost the same as the second one except for the slight progress of hypertrophy of myocites in the left ventricular biopsy specimens (**Fig. 3–c_1, c_2**).

DISCUSSION

Studies of animal models of tachycardia-induced cardiomyopathy[4,5] have shown that supraventricular tachycardias lasting for a month cause a reduction in collagen support of adjoining myocytes. A month of recovery from supraventricular tachycardia is associated with left ventricular hypertrophy and increased concentration of collagen. No histopathologic findings of left and right ventricular myocardial biopsy specimens associated with tachycardia in humans have been reported in detail. Compared with animal model studies, the histologi-

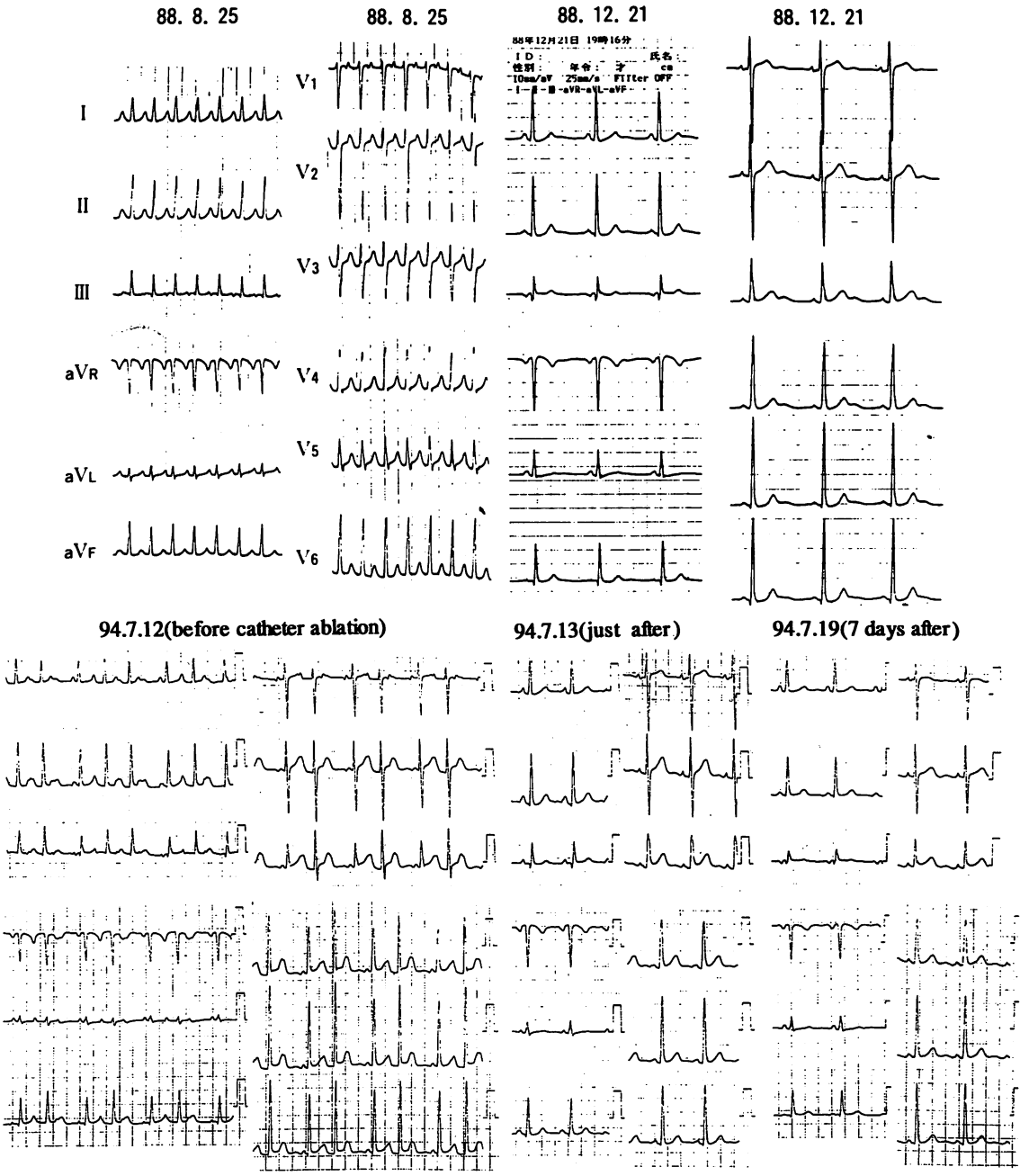

Fig. 1 Electrocardiographic findings

Upper: ECG obtained at the age of 17 demonstrating atrial tachycardia. The heart rate was 170/min. The P wave polarity is negative in lead III and the first part of P wave is negative in lead V_1. After treatment with atenolol at a dose of 50 mg/day (December 21, 1988), the heart rate decreased to normal. However, the P wave polarity is somewhat different from that noted during sinus rhythm.

Lower: ECG at the age of 22 showing atrial tachycardia with Wenckebach block. The P wave polarity is somewhat different from normal sinus rhythm. The heart rate is 150–180/min. Just after ablation, the ECG showed normal sinus rhythm. Seven days after ablation, the heart rate is in the normal range. However, the P wave polarity is somewhat different from that noted during sinus rhythm.

cal findings of the myocardium of our patient at the age of 17 appear to correspond to the phase of tachycardia and that at the age of 22 comply with the recovery phase from tachycardia in animal models. There is no report of the time-course of myocardial histological findings in human atrial tachycardia-induced cardiomyopathy to our knowledge. In a patient with chronic ventricular tachycardia, left

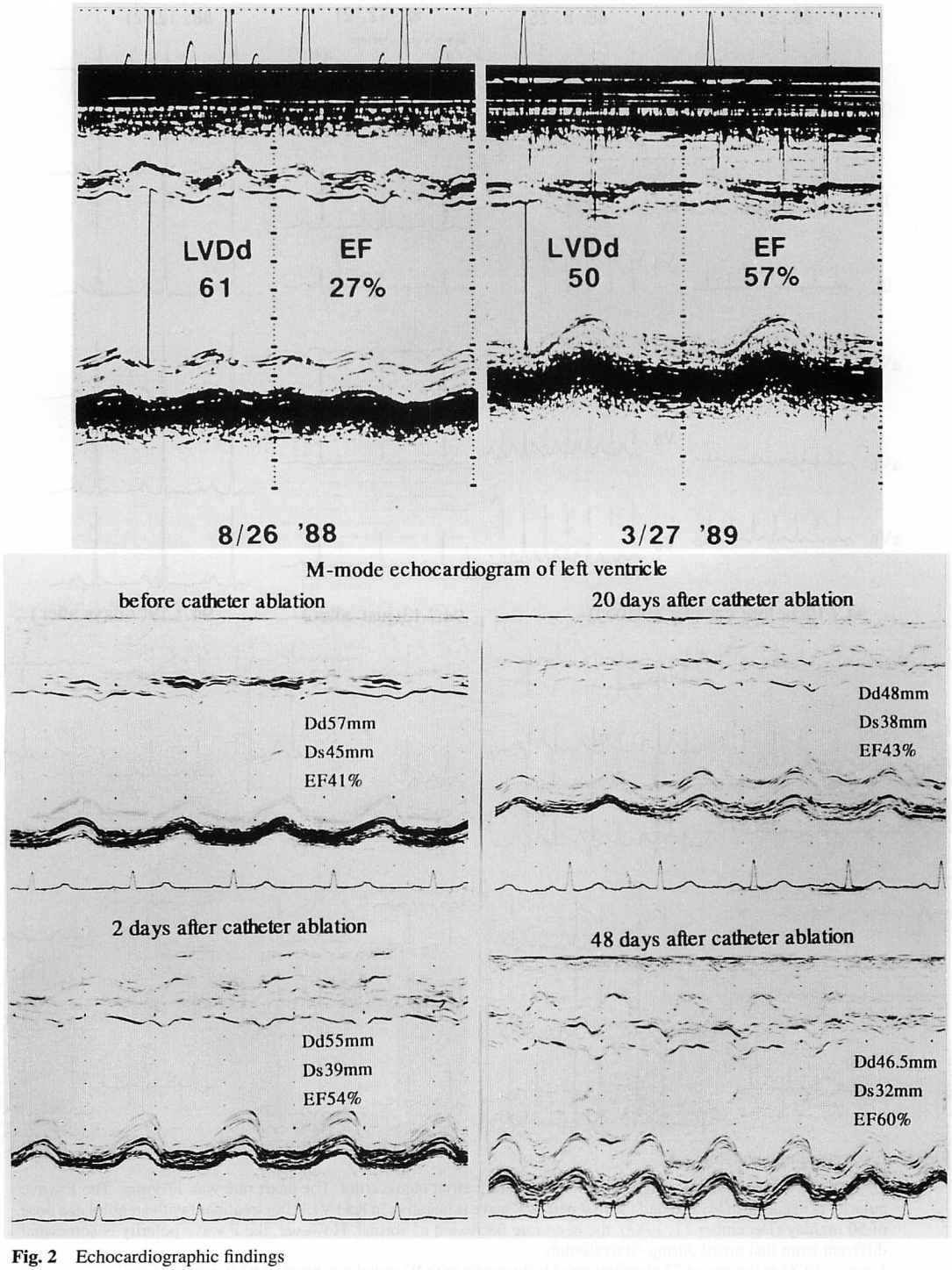

Fig. 2 Echocardiographic findings

Upper : M-mode echocardiogram of the left ventricle.
Echocardiogram obtained at the age of 17 demonstrating left ventricular dilatation and impaired wall motion (left ventricular end-diastolic dimension of 61 mm and ejection fraction of 27%; *left*). After treatment with atenolol at a dose of 50 mg/day, both left ventricular end-diastolic dimension and wall motion returned to normal (left ventricular dimension of 50 mm, ejection fraction of 57%; *right*).
Lower : Echocardiogram prior to catheter ablation showing left ventricular dilatation and impaired wall motion (left ventricular end-diastolic dimension of 57 mm and ejection fraction of 41%). Following catheter ablation, both left ventricular dilatation and wall motion gradually returned to normal.
LV=left ventricular; Dd=end-diastolic dimension; Ds=systolic dimension; EF=ejection fraction.

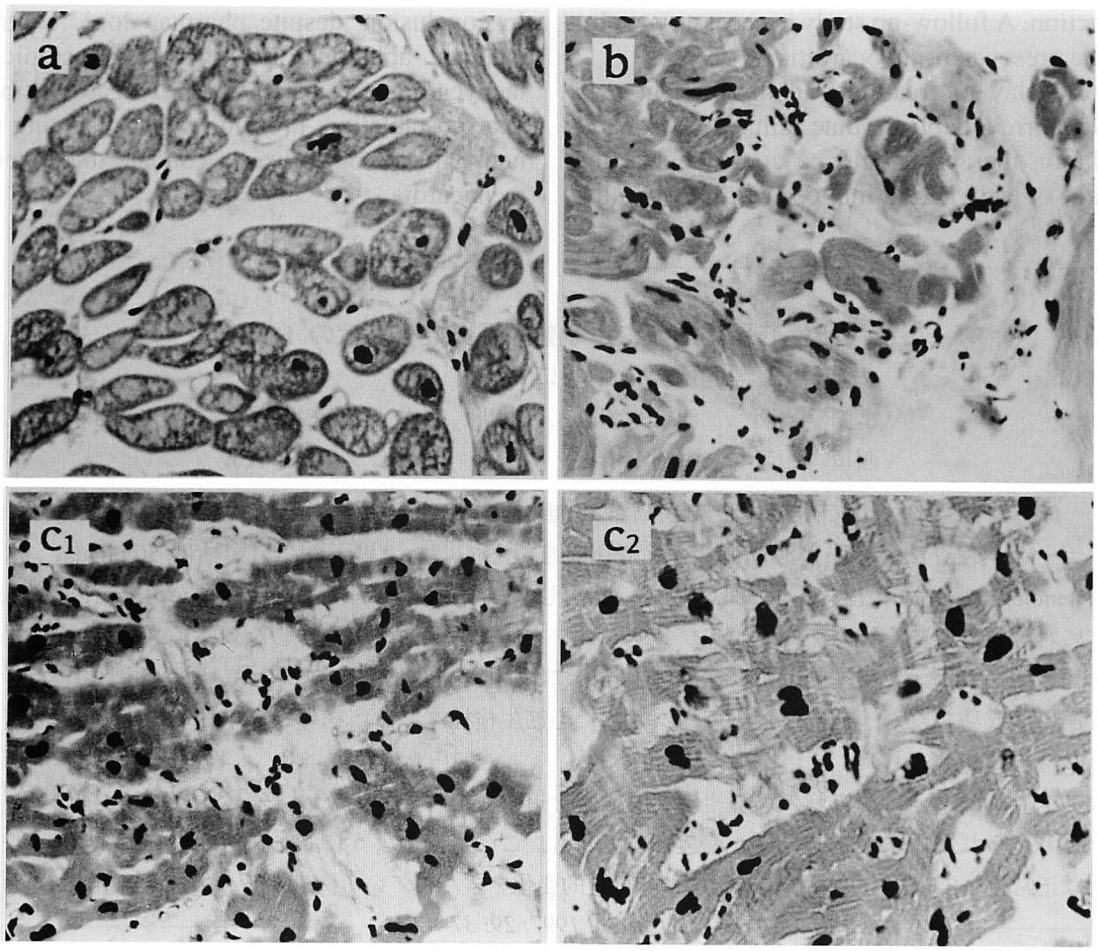

Fig. 3 Endomyocardial biopsy findings

a : Left ventricular endomyocardial biopsy specimen obtained at the age of 17 when the patient had tachycardia. Mild interstitial fibrosis and mild myocardial cell hypertrophy are seen (cell diameter 17±3 μm; ×200, hematoxylin-eosin stain).

b : Left ventricular endomyocardial biopsy specimen obtained at the age of 22 when ablation was performed. Advanced interstitial fibrosis, mild inflammatory cell infiltration, endocardial thickening, and myocardial hypertrophy are seen (cell diameter 17±4 μm; ×200, hematoxylin-eosin stain).

c_1 : Right ventricular endomyocardial biopsy specimen obtained 1 year after catheter ablation. Interstitial fibrosis and mild inflammatory cell infiltration are seen (×200, hematoxylin-eosin stain).

c_2 : Left ventricular endomyocardial biopsy specimen obtained 1 year after catheter ablation. Interstitial fibrosis, mild inflammatory cell infiltration, and slight progression of myocardial hypertrophy are seen compared with *b* (cell diameter 20±5 μm; ×200, hematoxylin-eosin stain).

ventricular function returned to normal following catheter ablation[6]. Furthermore, myocardial hypertrophy regressed back to normal 9 months after ablation. They did not observe any fibrosis in the specimens obtained from the right and left ventricular biopsy.

The myocardial biopsy specimen obtained at the age of 17 demonstrated mild hypertrophy of myocytes and mild fibrosis. Although echocardiography demonstrated normalization of left ventricular dimension and function following radiofrequency catheter ablation, the myocardial biopsy at the age of 22 showed mild myocardial hypertrophy of myocytes, inflammatory cell infiltration, and moderate fibrosis. The difference in the myocardial histopathologic findings at 1 year after the treatment between the previously reported case and our case may reflect the duration of tachycardia. In our case, the tachycardia was persistent.

In our case, inflammatory cell infiltration was found in the myocardial biopsy specimens (**Figs. 3–b, c_1, c_2**). We could not rule out the possibility that myocarditis might cause atrial tachycardia and furthermore that atrial tachycardia exacerbated the car-

diac function. A follow-up study is necessary to elucidate the presence of myocarditis.

Further delay of treatment in this patient may have led to irreversible cardiac failure. Thus, catheter ablation therapy should be considered at the early stage of tachycardia-induced cardiomyopathy. In conclusion, despite pharmacologic control and catheter ablation therapy of an ectopic atrial tachycardia, this patient had myocardial fibrosis 1 year postablation, presumably representing permanent damage to her heart secondary to her persistent tachycardia.

要　約

異所性心房頻拍症による頻脈誘発性心筋症の1例：病理組織学的所見を中心に

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症例は22歳の女性で，17歳時に異所性心房頻拍を指摘された．当初は心エコー図法と心臓カテーテル検査法で拡張型心筋症様の検査所見を呈していた．異所性心房頻拍は 50 mg/day の atenolol 内服によりコントロールされていた．しかし22歳時に異所性心房頻拍が再発し，それに対し当院にて心臓カテーテルアブレーションを施行し，治療に成功した．心エコー図による経過観察では，左室内腔および機能は1年後には正常化した．このように異所性心房頻拍の治療には成功したが，期間を追って観察された心筋生検標本では，病理組織学的に中等度の心筋の線維化が示された．

本症例のように，心筋の病理組織学的検索を追跡した報告は少ない．一般的に可逆的であると考えられている頻拍誘発性心筋症といえども，この症例のように心エコー図検査では心機能は正常化していても，病理組織学的に非可逆的変化をきたしている例もあるので注意を要する．

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