JACC Vol. 32, No. 1
July 1998:205–10

Resolution of Cardiomyopathy After Ablation of Atrial Flutter

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Objectives. **We sought to serially assess left ventricular (LV) function before and after catheter ablation of atrial flutter (AFl).**

Background. **The relation of tachycardia-induced cardiomyopathy to AFl and its response to direct catheter ablation are unknown.**

Methods. **LV function was assessed in a series of 59 consecutive patients with successful radiofrequency ablation (RFA) of AFl before and after the procedure. Eleven patients had dilated cardiomyopathy (LV ejection fraction [LVEF] <50%) and congestive heart failure (CHF) symptoms and are the subject of this report. LV function was assessed by LVEF on two-dimensional echocardiography and functional status by New York Heart Association (NYHA) CHF classification.**

Results. **Patients were 59 ± 8 years old, and were all male. Five patients had a preablation diagnosis of idiopathic cardiomyopathy. The preablation LVEF was 30.9 ± 11.0% and improved to 41.3 ± 16% (p = 0.005) when measured 7 months after successful ablation. NYHA CHF class improved from 2.6 ± 0.5 to 1.6 ± 0.9 (p = 0.002). Six (55%) of 11 patients had normalization of the LVEF, with complete resolution of CHF symptoms. A lower preablation LVEF and functional class predicted nonresolution of dilated cardiomyopathy (p = 0.002 and 0.001, respectively).**

Conclusions. **Restoration of normal sinus rhythm by RFA in patients with chronic AFl and cardiomyopathy substantially improved LV function. Resolution of dilated cardiomyopathy occurred in the majority of patients. Tachycardia-induced cardiomyopathy may be a more common mechanism of LV dysfunction in patients with AFl than expected, and aggressive treatment of this arrhythmia should be considered.**

(J Am Coll Cardiol 1998;32:205–10)

Tachycardia-induced cardiomyopathy has been described as a distinct clinical entity and has been referred to as the most frequently unrecognized curable cause of heart failure (1). Timely recognition and correct diagnosis can lead to improvement in ventricular function, relief of congestive heart failure (CHF) and, usually, cure if effective rate slowing measures are undertaken. Animal models of tachycardia-induced cardiomyopathy demonstrate impaired systolic and diastolic function, reduced response to catecholamine stimulation and defective myocyte contractile function; all may improve after control of tachycardia (2–4). There are numerous case reports and small series describing reversal or improvement of dilated cardiomyopathy after control of ventricular response in a variety of supraventricular and ventricular tachycardias in adults and children by pharmacologic, surgical and, more recently, transvenous catheter ablation techniques (5–16). Control of tachycardia has been achieved by permanently reducing ventricular conduction or by sustained restoration of normal sinus rhythm (NSR).

Recently, radiofrequency catheter ablation of common atrial flutter (AFl) has been described as a safe and effective means of cure of refractory AFl, with good long-term outcomes in several series (17–21). In the present series, prompted by a previous observation (21), we investigated the contribution of persistent AFl to the development of left ventricular (LV) dysfunction and the evolution of tachycardia-induced dilated cardiomyopathy in a group of patients undergoing successful radiofrequency ablation (RFA) of AFl. At the time of referral, this group of patients was presumed to have AFl secondary to structural heart disease. The results of our study suggest that in these patients a tachycardia-induced mechanism was the cause of, or contributed to, LV dysfunction.

Methods

Patient selection. Patients with refractory type 1 (common) AFl were referred for RFA as definitive treatment. *Refractory AFl* was defined as the failure of more than two cardioversions and more than two antiarrhythmic medications to maintain NSR. All patients had electrocardiographically documented type 1 AFl, defined as inverted P waves (sawtooth pattern) in leads II, III, aVF and upright P waves in lead V_1. Patients were specifically excluded for the presence of, or history of, documented atrial fibrillation.

RFA. All patients gave written informed consent for the ablation procedure. Antiarrhythmic medications were withheld for at least 7 days before RFA. RFA was performed according to an anatomically guided approach, as published previously

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Manuscript received April 4, 1997; revised manuscript received March 16, 1998, accepted March 20, 1998.

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0735-1097/98/$19.00
PII S0735-1097(98)00183-1

JACC Vol. 32, No. 1
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Abbreviations and Acronyms

ACE = angiotensin-converting enzyme
AFl = atrial flutter
AV = atrioventricular
CHF = congestive heart failure
ECG = electrocardiogram, electrocardiographic
LV = left ventricular
LVEF = left ventricular ejection fraction
NYHA = New York Heart Association
NSR = normal sinus rhythm
RFA = radiofrequency ablation

(18,21). Patients were in the fasting state and received midazolam and fentanyl for sedation.

Patient follow-up. Patients with successful RFA were discharged without administration of any antiarrhythmic medication. They were examined as outpatients after 3 to 4 weeks and every 3 months thereafter. Any symptoms suggestive of arrhythmia were investigated with an electrocardiogram (ECG), 24-h Holter ECG recording or transtelephonic ECG event recording. Patients were treated with aspirin (325 mg daily) for 3 months as prophylaxis against thromboembolic events.

LV function assessment. LV function was assessed by visual estimation of LV ejection fraction (LVEF) with two-dimensional echocardiography in all patients, measurement of end-diastolic and end-systolic LV volumes and fractional shortening before and after the ablation procedure. Two-dimensional echocardiography has been shown (22–24) to be a valid and reliable method of measuring LV function. The echocardiograms were obtained in the days preceding the ablation procedure and again 7 months (median) after the successful ablation. Echocardiograms were acquired at the ablation center or at the referring hospital if the patient was from a geographically distant site. Echocardiographers were unaware of the patient's ablation status. In addition, assessment of clinical functional status and use of medications to combat CHF were noted.

Statistics. Results are reported as mean value ± SD. The paired Student *t* test was used to compare variables before and after RFA. Clinical characteristics of patients with versus those without improvement in LV function were compared using unpaired Student *t* test. The Fisher exact test was used for analysis of categoric variables as predictors of end points.

Results

Patient characteristics. In our laboratory, 59 of 61 patients underwent successful RFA of AFl. Eleven patients had an LVEF <50% before ablation (all successful) and are the subjects of the present report. All 11 patients were male and were of 59 ± 8 years old. The duration of AFl before ablation was 13 ± 5 months (range 6 to 24), and all patients had chronic AFl. Ventricular rates >100 beats/min had been present in all patients, with a mean heart rate before ablation of 102.3 ± 24.7 beats/min. All patients had symptoms of LV dysfunction. Mean New York Heart Association (NYHA) CHF class before ablation was 2.6 ± 0.5, and LVEF was 30.9 ± 11.0%.

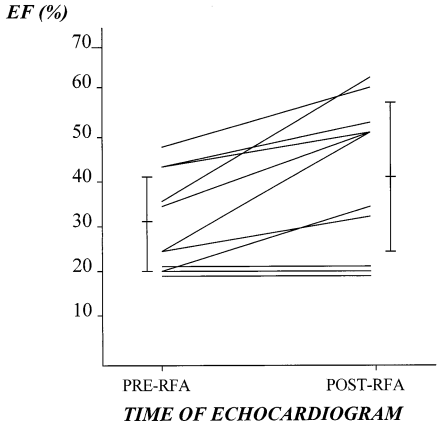


Figure 1. Comparison of LVEF before and after RFA. Mean LVEF improved from 30.9 ± 11% to 41.4 ± 16%.

All patients were described by the referring physicians as having cardiovascular disease; two patients had a history of hypertension; three had a history of myocardial infarction, two of whom also had a history of hypertension; and one patient had previously undergone aortic valve replacement for aortic stenosis. Five patients were diagnosed as having idiopathic dilated cardiomyopathy on the basis of the presence of significant LV dysfunction without the presence of an identifiable etiology (e.g., coronary artery disease, hypertension, valvular heart disease, alcohol abuse) (25). In all patients, the AFl was believed to have resulted from the underlying cardiovascular disease and cardiomyopathy.

Follow-up of LV function after ablation. All patients remained in NSR during the follow-up period of 35.5 ± 10.4 months, without the need for antiarrhythmic treatment or a repeat ablation procedure. They were followed up by both the arrhythmia service and the referring physicians, and NSR was present at each visit, without reports of clinically recognizable AFl. After successful RFA, LVEF increased from 30.9 ± 11.0% to 41.4 ± 16.3% ($p = 0.005$). Heart rate decreased from 102.3 ± 24.7 to 78.7 ± 10.7 beats/min ($p = 0.005$). Figure 1 contrasts LVEF before and after ablation in the individual patients. Table 1 shows the differences in clinical characteristics before and after ablation. The overall improvement in LVEF correlated with the change in systolic LV dimensions. LV end-systolic dimension decreased from 4.8 ± 1.4 to 4.0 ± 1.3 cm ($p = 0.03$) and LV end-diastolic dimension from 5.8 ± 1.0 to 5.7 ± 0.7 cm (p = NS). Fractional shortening increased from 18.7 ± 10.1% before to 31.3 ± 14.4% after ablation ($p = 0.001$). There was no significant difference in LV wall thickness before and after ablation. Symptoms of CHF significantly decreased during follow-up as well. NYHA CHF class decreased from 2.6 ± 0.5 before to 1.6 ± 0.9 after ablation ($p = 0.002$).

Eight (73%) of the 11 patients showed an absolute improve-

Table 1. Clinical Characteristics Before and After Ablation for Atrial Flutter

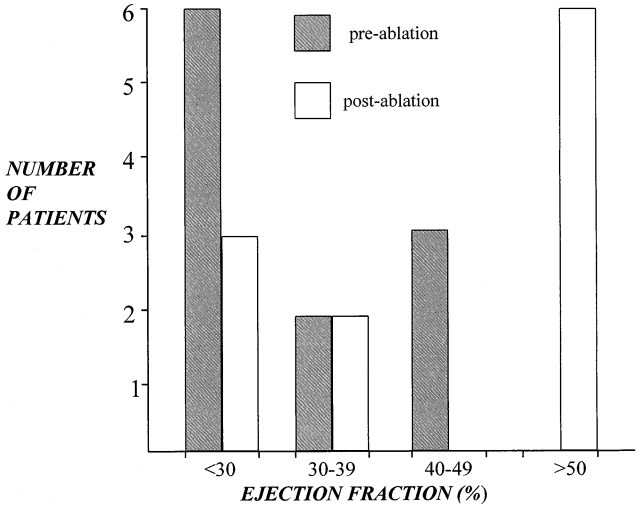
	Before RFA	After RFA	p Value
Heart rate (beats/min)	102.3 ± 24.7	78.7 ± 10.8	0.005
NYHA CHF class	2.6 ± 0.5	1.6 ± 0.9	0.002
LVEF (%)	30.9 ± 11.0	41.4 ± 16.3	0.005
LV ED dimensions (cm)	5.8 ± 1.0	5.7 ± 0.7	NS
LV ES dimensions (cm)	4.8 ± 1.4	4.0 ± 1.3	0.03
Fractional shortening (%)	18.7 ± 10.1	31.3 ± 14.4	0.001
LV wall thickness (cm)	1.3 ± 0.1	1.3 ± 0.2	NS

Data presented are mean value ± SD. CHF = congestive heart failure; ED = end-diastolic; ES = end-systolic; LV = left ventricular; LVEF = left ventricular ejection fraction; NYHA = New York Heart Association; RFA = radiofrequency ablation.

ment in LVEF of at least 5%. When the clinical characteristics of the eight patients who showed improvement in LVEF were compared with those of the three who did not, no statistically significant difference was found in age and duration of AFl. However, there was a statistically significant difference in LVEF and NYHA CHF class before ablation between the two patient groups: Patients who did not improve had a significantly worse LVEF (20.3 ± 0.6% vs. 34.9 ± 1.7%, p = 0.048) and CHF class (3.0 ± 0.0 vs. 2.3 ± 0.5, p = 0.024) before ablation. Of the patients who improved, two patients had hypertension, and two had hypertension and a previous myocardial infarction; the remaining four had no history of cardiovascular disease other than idiopathic cardiomyopathy. In the three patients who did not improve, one had a history of myocardial infarction, one had a history of aortic valve replacement, and the third had no known cardiovascular history other than idiopathic cardiomyopathy.

Before RFA, most patients had severe LV dysfunction and 55% of the study group had an LVEF <30%. Figure 2 contrasts the distribution of LVEF before and after ablation.

Figure 2. Change in distribution of LVEF after RFA for AFl. Six of 11 patients had normalized LVEF after RFA.



After ablation, only 3 patients (27%) had an LVEF >30%, and strikingly, 6 (55%) had an LVEF ≥50%. In other words, LVEF improved in the majority of patients to within the normal range after ablation.

Resolution of dilated cardiomyopathy. Of the 11 patients in the present series, 6 (55%) had complete normalization of LV function, with an LVEF >50% after ablation. The absolute improvement in LVEF in this group was 17.8%. All patients reported improvement in functional capacity and complete resolution of CHF symptoms; medications for CHF were discontinued in all patients by their primary physicians. A detailed analysis was undertaken to identify the patient characteristics that were associated with normalization of LV function. There was no difference in age or AFl duration between the patients who showed normalization and those who did not. However, there was a statistically significant difference between the two groups in LVEF and NYHA CHF class. The preablation LVEF was 39.0 ± 8.7% in the group with resolution and 21.2 ± 2.2% in the group without normalization (p = 0.002). NYHA CHF class was 2.2 ± 0.4 in the former group and 3.0 ± 0.0 in the latter (p = 0.001).

The relation of preablation diagnosis to resolution of cardiomyopathy showed that four (66%) of six patients in the group with normalization had a preablation diagnosis of idiopathic dilated cardiomyopathy compared with only one (20%) of five in the group without normalization (p = NS, Fisher exact test). Of the patients in the group with normalization and no diagnosis of idiopathic cardiomyopathy, one had a history of hypertension, and another had a history of inferior wall myocardial infarction and hypertension.

Of the six patients who showed resolution of cardiomyopathy, five were taking digoxin, one quinidine, two procainamide, one flecainide and two amiodarone (all drugs were discontinued before echocardiography and ablation, according to procedure protocol). All six patients were taking angiotensin-converting enzyme (ACE) inhibitors before RFA. After ablation, quinidine, procainamide, amiodarone and digoxin were not restarted in those six patients. Of the five patients without resolution of cardiomyopathy, all were receiving digoxin and ACE inhibitors before ablation. One patient was taking flecainide and amiodarone and another amiodarone alone. Antiarrhythmic agents were not restarted after ablation. There were no patients taking beta-adrenergic blocking agents before or after ablation. One patient was taking amlodipine before ablation and also received this medication afterward. No other calcium channel blocking agents were used by any patient.

Discussion

In the present series of patients undergoing RFA for AFl we demonstrated 1) an overall improvement in LVEF, with restoration of NSR; 2) resolution of dilated cardiomyopathy in a significant subset of these patients; and 3) a decreased likelihood of resolution of dilated cardiomyopathy when the preablation LV dysfunction and clinical status were more

severe. It is generally believed that chronic or persistent AFl occurs in the setting of organic heart disease, often valvular, ischemic or dilated cardiomyopathy (26,27), rather than in the absence of heart disease. In addition, it is believed that most AFl tends to be unstable, degenerating to atrial fibrillation or reverting to NSR (26,27). When AFl is recurrent or chronic, especially if antiarrhythmic drugs have failed to reestablish NSR, ventricular rate control is usually attempted. However, pharmacologic control of rate is very difficult to achieve, especially on a consistent basis. The belief that AFl occurs in the setting of cardiomyopathy, that it may ultimately transform to atrial fibrillation and the frequent difficulty in achieving effective rate control set up conditions ripe for the development of unrecognized tachycardia-induced cardiomyopathy in patients with chronic AFl. The present series, and a previous description of a single case of ours (21), represent the first report of resolution of tachycardia-induced cardiomyopathy by RFA for AFl.

Tachycardia-induced cardiomyopathy. Early reports in the first half of this century showed cardiac enlargement and CHF after onset of atrial fibrillation and resolution by pharmacologic cardioversion or rate control (28,29). These observations suggested tachycardia as the etiologic factor for cardiomyopathy, especially when other causes for CHF could not be found. Subsequent series of patients have shown improvement in LVEF after control of ventricular rates in different types of tachycardia, further reinforcing the concept of tachycardia-induced cardiomyopathy as a clinical entity. For example, improvement in LVEF was observed after control of tachycardia in a series of eight patients with ectopic atrial tachycardia or accessory atrioventricular (AV) pathway after His bundle and AV node ablation (13), 10 patients with atrial fibrillation after RFA of the His bundle (14) and a series of 10 patients with atrial fibrillation after different means of rate control (15). In animal models, dilated cardiomyopathy is produced by inducing tachycardia through chronic electronic pacing. After cessation of tachycardia, LV function normalizes along with the animals' neurohormonal profile, although decreased LV mass and myocyte contractile function may persist (2–4). In humans, the diagnosis of tachycardia-induced cardiomyopathy can only be made in retrospect, making clinical features that predict this diagnosis very important. A recent review (1) suggested a set of simple clinical characteristics that should raise the suspicion of tachycardia-induced cardiomyopathy: 1) dilation of the heart or clinical heart failure, and 2) chronic or very frequent cardiac arrhythmias. The group of patients studied in the present report fulfill both criteria.

AFl as a cause of tachycardia-induced cardiomyopathy. All patients who had resolution of cardiomyopathy in the present report were referred with a diagnosis of AFl caused by the dilated cardiomyopathy. The objective of treatment was to control the rapid ventricular rates generated by refractory AFl and alleviate tachycardia symptoms and CHF. The finding of improvement in LVEF in a substantial segment of the series was unanticipated. One patient with a diagnosis of idiopathic dilated cardiomyopathy did not improve and had an LVEF of 20%. This lack of improvement may have reflected irreversible myocardial damage not amenable to improvement or cure with ablation, despite the presence of tachycardia as a potential explanation for dilated cardiomyopathy (1), and is further supported by the findings of the comparison of clinical characteristics between those patients who showed improvement and those who did not. This comparison showed that the latter group had a lower preablation LVEF and a poorer functional status as measured by NYHA CHF class, further suggesting that in this group of patients, the cardiomyopathy may not be amenable to improvement because of irreversible structural changes that correlate with worse LVEF and functional status. Furthermore, the comparison between the group with and that without resolution of cardiomyopathy again showed a lower LVEF and worse functional status as negative predictors for resolution of cardiomyopathy. These findings may be explained by a disruption in myocardial architecture produced by tachycardia that could result in scarring and therefore irreversible myocardial damage, as seen in animal models of tachycardia-induced cardiomyopathy (4,30). This hypothesis suggests the importance of early detection of chronic AFl and institution of effective rate control measures to prevent permanent damage. Most patients with normalization of LV function had a preablation diagnosis of idiopathic dilated cardiomyopathy, but this variable failed to meet statistical significance as an indicator of resolution of cardiomyopathy. Because the number of patients in this group was small, it would be ideal to assess this relation in a larger series.

One patient with a history of hypertension, but not coronary artery disease, myocardial infarction or valvular heart disease, had resolution of cardiomyopathy after ablation, which suggests that the tachycardia was the main mechanism for ventricular dysfunction rather than chronic hypertension. Another patient with hypertension and a history of an inferior wall myocardial infarction also showed resolution of dilated cardiomyopathy, also suggesting tachycardia-induced cardiomyopathy as the main mechanism for ventricular dysfunction. The echocardiogram in this patient showed global hypokinesia before ablation, with an LVEF of 35%; the postablation echocardiogram showed only inferior wall hypokinesia and an LVEF of 50%, supporting the assertion that the inferior wall myocardial infarction was not the main etiology of the LV dysfunction. These observations suggest that tachycardia should be considered a possible underlying cause of dilated cardiomyopathy in those patients with ventricular dysfunction or a ventricular motion pattern, out of proportion to the underlying anatomic substrate. A recent review of tachycardia-induced cardiomyopathy (1) introduced the concept of pure and impure tachycardia-induced cardiomyopathy, the former existing in those cases in which the tachycardia is the sole explanation for the ventricular dysfunction and the latter occurring in cases in which other factors may contribute to the reduction in LVEF. The patients in our series who showed improvement in LV function without achieving normalization and had a diagnosis other than idiopathic dilated cardiomyopathy could be classified as having impure tachycardia-induced

cardiomyopathy, and all those with normalized ventricular function could be classified as having pure tachycardia-induced cardiomyopathy, even the patient with a history of hypertension because the latter diagnosis probably had no bearing on the preablation LV dysfunction. The example mentioned previously stresses the need to consider correction of the arrhythmias, even in the presence of seemingly logical causes for cardiomyopathy, because of the possibility of several simultaneous underlying mechanisms of dilated cardiomyopathy.

Other therapeutic options exist for the treatment of AFl, but all have drawbacks. Control of ventricular response by pharmacologic means could help to improve LV function, but this goal is often not achieved. Some medications can also reduce LV function. AV junction ablation will permanently eliminate rapid rates but does not restore AV synchrony or normal chronotropic response and necessitates permanent pacemaker implantation. Antiarrhythmic therapy can assist in the maintenance of NSR but can have serious side effects (31).

Limitations of the study. The small number of patients in the present series and the referral bias for RFA precludes determining the true prevalence of dilated cardiomyopathy secondary to AFl. Echocardiography was not uniformly performed at a single laboratory, but all measurements were performed in a similar manner. In addition, most of the echocardiograms were rereviewed by a single observer, and the results were confirmed. Visual estimation of LVEF has been proven to be a highly accurate and reproducible measure of LV function (22–24) and correlates well with findings in gated nuclear angiography, contrast ventriculography and sophisticated computer techniques. The fractional shortening calculations confirm the study observations. Assessment of LV function was performed only once before the procedure and once after the procedure. Ideally, echocardiography would be performed serially after the procedure because previous studies indicate that recovery can occur late, especially in patients with more severe dysfunction (1). No postablation invasive studies were available to correlate hemodynamic variables with the LVEF findings in the present series.

Conclusions. In patients with both AFl and LV dysfunction, tachycardia may play a primary rather than secondary role. Given the prevalence of AFl relative to other incessant or chronic tachycardias, AFl may be a common etiology of tachycardia-induced cardiomyopathy. Cure or control of AFl should be seriously considered in all patients with the uncontrolled form of AFl, with or without LV dysfunction, to prevent or improve cardiomyopathy. Further studies are needed to assess the scope of this condition.

ADDENDUM. Since submission of the manuscript for this report, one additional patient with AFl of 9 months in duration and an LVEF of 20% underwent successful ablation. Six weeks after ablation the LVEF was 55%.

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