Radiofrequency Catheter Ablation in Type I Atrial Flutter : Preliminary Experience of 10 Cases

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Abstract

Common atrial flutter results from macroreentry in the right atrium. Catheter ablation of slow conduction, between tricuspid annulus and inferior vena cava (TA-IVC) or tricuspid annulus and coronary sinus ostium (TA-CS os) has been reported to terminate and prevent recurrence of this arrhythmia. We reported 10 consecutive patients, 7 men and 3 women, who underwent radiofrequency catheter ablation of common atrial flutter. The mean age was 59.4 ± 11.2 years (range 42 - 82 years). During the paroxysmal atrial flutter, all patients had palpitation, 4 had dyspnea on exertion, 3 patients had syncope and 1 patient had presyncope. The mean duration of symptoms was 5.7 ± 4.9 years (range 0.5 - 13 years). Two patients had dilated cardiomyopathy, 1 Ebstein's anomaly and 1 chronic obstructive pulmonary disease. Four patients (40%) had history of atrial fibrillation (AF) before ablation. The mean cycle length of atrial rhythm was 257.2 ± 36.6 ms. Ablation was done by anatomical approach and could terminate arrhythmia in 9 patients (90%), 7 from TA-IVC, 2 from TA-CS os without major complication. The mean number of applications was 20.4 ± 16.9 and turned atrial flutter to normal sinus rhythm in 13.5 ± 10.7 seconds. Fluoroscopic and procedure times were 38.4 ± 31.4 and 157.2 ± 68.8 minutes, respectively. During the follow-up period of 24.0 ± 28.7 weeks, 2 patients had recurrent atrial arrhythmia, 1 atrial fibrillation and 1 atrial flutter type I, giving the final success rate of 70 per cent. All patients who had recurrence or failure had a history of paroxysmal AF before ablation.

In conclusion, radiofrequency catheter ablation in atrial flutter type I, using anatomical approach, is an effective treatment to terminate and prevent this arrhythmia in short term follow-up. It may be considered as an alternative treatment in patients with atrial flutter who were refractory to antiarrhythmic agents.

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Atrial flutter type I or common atrial flutter (AFL), defined as inverted P waves in lead II, III and aVF, biphasic in I and V_6 and upright in V_1, is considered as a macroreentrant arrhythmia both in animal model[1,2] and human beings[3,4]. The reentrant circuit is counterclockwise in the right atrium[5,6]. The narrow isthmus of slow conduction, identified by prolonged low-amplitude fragmented electrical activity or double potentials and long stimulus-to-P wave interval during pacing entrainment, has been identified in the low posteroseptum of right atrium between the inferior vena cava (IVC) and the tricuspid annulus (TA)[6,7]. Catheter ablation, both direct current[8,9] and radiofrequency (RF)[10-12], at this area is able to terminate and prevent recurrence of this arrhythmia. Endocardial direct current shock can cause a significant barotrauma and converts AFL to sinus rhythm with high recurrence rate. In contrast, RF energy produces a more discrete myocardial lesion and the application of this energy at TA-IVC isthmus has high success and low recurrence rate. In this study, we performed radiofrequency catheter ablation (RFCA) in AFL, by using anatomical approach, at TA-IVC isthmus or TA-CS os isthmus and reported its result.

PATIENTS AND METHOD

Patients

The study population consisted of 10 consecutive patients who were referred to Her Majesty's Cardiac Centre, Siriraj Hospital, between October 1995 and January 1997 for RFCA of chronic paroxysmal AFL. All patients were symptomatic and refractory to various antiarrhythmic agents, including amiodarone. Complete history taking, physical examination, baseline electrocardiography (ECG), chest roentgenography, echocardiography and coronary angiography were performed in all patients. All gave informed consent.

Electrophysiologic study

Electrophysiologic test (EPS) was performed in fasting state and after discontinuation of all antiarrhythmic agent more than 3 half-lives. Under local anesthesia and mild sedation, standard invasive EPS was performed. Three 6 Fr quadripolar electrode catheters were passed from the right and left femoral veins and positioned in the high right atrium, His bundle area and right ventricular apex. An octapolar catheter was inserted into the left subclavian vein and advanced into the coronary sinus. The most proximal pair of electrodes was positioned at the coronary sinus ostium. The catheters were positioned under fluoroscopic guidance and the exact side determined by the intracardiac electrogram. Surface ECG lead I, aVF, V_1 and V_6 and intracardiac electrograms were simultaneously displayed and recorded on a multichannel oscilloscopic recorder (Cardiolab, Prucka Engineering). Electrical stimulation was performed *via* a stimulator with a pulse duration of 2 ms at twice diastolic threshold. Standard 12-lead ECG was recorded for all AFL episodes or as needed. Three patients who were in sinus rhythm were given incremental atrial pacing at progressively shorter cycle lengths (from 400 to 200 ms) or atrial extrastimulation pacing, up to 3 extrastimuli at three different cycle lengths, from the high right atrium and proximal coronary sinus in order to induce AFL. For patients with spontaneous and sustained AFL at the beginning of the study, atrial mapping and RFCA were performed before any programmed atrial pacing or stimulation.

Ablation procedure

After intravenous infusion of propofol and fentanyl, a 7 Fr quadripolar deflectable-tip catheter (Webster or EP Tech) was inserted into the right femoral vein and advanced to the low posteroseptum of right atrium and used as mapping and ablation catheter. The target site was the isthmus bounded by the IVC and atrial junction inferiorly, the coronary sinus ostium superiorly and tricuspid annulus anteriorly (Fig. 1). In all cases, RF current was applied during sustained atrial flutter. The initial power was 30 W for 30 seconds at the IVC-TA isthmus, where a large ventricular potential and a small atrial electrogram were recorded. Radiofrequency applications were applied at multiple points along the way during slow withdrawal of the ablation catheter toward the IVC. If the atrial flutter did not terminate, ablative technique was done at the same location, from TA to IVC or TA to CS os. At the site of successful termination of atrial flutter (Fig. 2), one additional application was delivered with the same power for 60 seconds.

If the impedance rose, the ablation catheter was withdrawn and cleaned and repositioned at the target site. After successful ablation, each patient was observed in the electrophysiological laboratory for at least 30 minutes and electrophysiologic

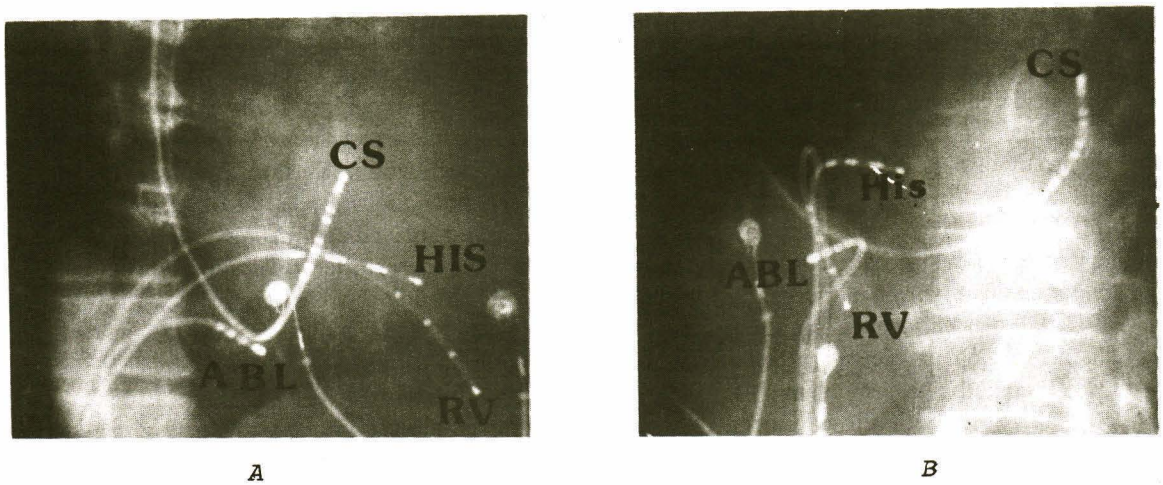

Fig. 1. Site of ablation catheter to create a line of conduction block from tricuspid annulus-inferior vena cava isthmus in A) right anterior oblique and B) left anterior oblique views.
ABL = ablation catheter, HIS = His position catheter, CS = coronary sinus catheter, RV = right ventricular catheter

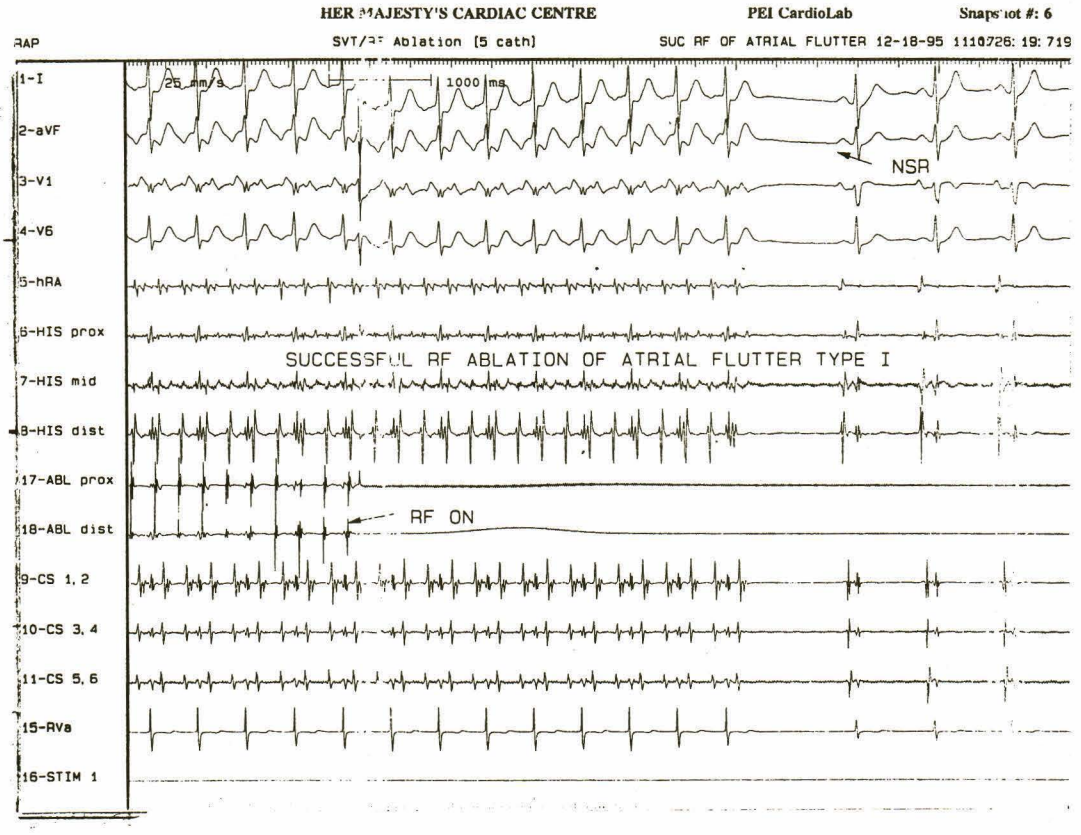

Fig. 2. Atrial flutter type I was terminated to sinus rhythm after application of radiofrequency current.

testing was performed by both rapid atrial pacing and programmed atrial extrastimulation, with and without isoproterenol infusion (1-6 μg/min) to ensure that atrial flutter was no longer inducible, to assess whether sustained atrial fibrillation could be induced and evidence of block at the IVC-TA isthmus during proximal coronary sinus pacing. The absence of inducible AFL was defined as successful. Total procedure time, defined as total time in the EPS laboratory and fluoroscopic time were recorded in all patients.

Follow-up

All patients were monitored continuously in hospital for 24 to 48 hours after the ablation procedure. In the successful cases, they were discharged without any antiarrhythmic agents. Each patient was observed on a regular basis every $^1/_2$ - 2 months by one of the first three authors. Surface ECG and Holter monitoring were done in all patients who had recurrent palpitation, presyncope and syncope.

RESULT

The clinical characteristics of the study population are shown in Table 1. There were 7 men and 3 women. The mean age was 59.4 ± 10.4 years (range 42-82 years). All of them were AFL despite being treated with many classes of antiarrhythmic agents including quinidine, flecanide, propafenone and amiodarone, with the duration range from $^1/_2$ - 13 years (mean 5.7 ± 4.9 years). All patients had palpitations, 4 patients had dyspnea on exertion, 3 and 1 had at least one episode of syncope and presyncope, respectively, during sustained AFL. Four patients (40%) had underlying diseases, 2 dilated cardiomyopathy, 1 Ebstein's anomaly, and 1 chronic obstructive pulmonary disease. All patients had ECG documented AFL. Four patients (40%), 2 dilated cardiomyopathy and 2 normal structural heart, also had history of atrial fibrillation (AF) from surface ECG before ablation. The mean left atrial diameter and ejection fraction of left ventricle were 41.6 ± 3.3 mm (range 39-48 mm) and 62.9 ± 15.7 per cent (range 40-85%), respectively. No patients had significant coronary artery stenosis from coronary angiography.

Seven patients (70%) were in AFL at the start of the EPS test and 3 (30%) were in sinus rhythm. AFL was induced by rapid atrial pacing with isoproterenol infusion and atropine injection in 1 patient. The characteristic of EPS and RFCA were shown in Table 2. The mean cycle length of AFL was 257.2 ± 36.6 ms (range 200-300 ms). RFCA was immediately successful in 9 patients (90%). The patient who was unsuccessful had dilated cardiomyopathy and AFL type I turned to AFL type II after success application of RF at TA-CS os isthmus. The successful ablation sites were located in TA-IVC isthmus in 7 patients and TA-CS os in 2 patients. The mean duration of AFL turned to sinus rhythm was 13.5 ± 10.9 seconds (range 2.0 - 29.2 seconds). The mean number of RF applications in patients with a successful result was 20.4 ± 16.9 times. Inducible of sustained AF after success ablation occurred in 1 patient who had

Table 1. Clinical characteristics of the study population.

Characteristics	n = 10 (%)	
Sex F:M	1:2.3	
Symptoms		
Palpitation	10	(100)
Dyspnea	4	(40)
Syncope	3	(30)
Presyncope	1	(10)
Underlying disease	4	(40)
Echocardiography		
Left atrium diameter (> 4 cm)	7	(70)
Ejection fraction (<45%)	2	(20)
History of atrial fibrillation	4	(40)

Table 2. Electrophysiological characteristics of the patients who had of atrial flutter successfully ablated.

Characteristics	n = 9
Atrial flutter cycle length (ms)	257.2 ± 36.6
Site of ablation	
TA - CS os	2 (22.2%)
TA - IVC	7 (77.8%)
Atrial flutter turn to sinus (sec)	13.5 ± 10.9
Attempts (times)	20.4 ± 16.9
Flu time (min)	38.4 ± 31.4
Procedure time (min)	157.2 ± 68.8

Abbreviation : TA = Tricuspid annulus, CS os = Coronary sinus ostium, IVC= Inferior vena cava, sec = seconds, min = minutes, ms = milliseconds.

dilated cardiomyopathy and needed cardioversion to terminate AF. The mean fluoroscopic and procedure times were 38.4 ± 31.4 times (range 15.0 - 75) and 157.2 ± 68.8 (range 50 - 240) minutes, respectively.

Hypotension, bradycardia and second degree, Mobitz I, atrioventricular block occurred in 1 patients, who had Ebstein's anomally, and needed dopamine infusion to maintain the blood pressure and heart rate and was discharged three days later without any antiarrhythmic agents.

During a mean follow-up of 24.0 ± 28.71 weeks, 2 patients had recurrent atrial arrhythmia, 1 AFL and 1 AF, respectively, giving the final success rate of 70 per cent. All the patients who had recurrence or failure had a history of paroxysmal AF before ablation.

DISCUSSION

Atrial flutter is often associated with disabling symptoms and may be resistant to treatment with various antiarrhythmic agents. Recent studies have demonstrated that type I atrial flutter is due to macroreentry in the right atrium[5,6]. The reentrant circuit courses between the IVC and TA posteriorly and ablation in this area can terminate this arrhythmia[8-14]. In this study, we performed RFCA of AFL by using only anatomical approach and demonstrated that this protocol could be performed safely and effectively. The initial success rate in this study was 90 per cent, similar to the previous reports[12-16]. The major success ablation site in this study was TA-IVC isthmus, that was the area located inferior and posterior to the CS ostium. The recurrent rate of AFL after RFCA varied from 10 - 40 per cent in the previous reports [9-16]. This was probably related to in complete destruction of the atrial tissue providing reentrant circuit. A recent study by Nakagawa H[17] et al reported that eustachian valve/ridge (EVR) formed a line of fixed conduction block between the IVC and CS os and EVR and the TA provided boundaries for the AFL reentrant circuit. A creation of complete line of conduction block between TA and the EVR was a more reliable criteria for long-term ablation success than noninducible criteria[17]. The recurrent rate in this study was similar to the previous reports. In the patient who had recurrence we did not create line of block as Nakagawa proposed because the patient was one of our early experiences and we used only non inducible criteria.

Previous studies of RFCA of AFL have shown a high recurrence of AF after ablation [13-15]. The factors which may predict the risk for developing AF[15,17] are structural heart disease, right or left atrial enlargement, a clinical history of paroxysmal AF before ablation, inducibility of sustained AF after ablation and greater number of applications of RF current[17]. In this study, RFCA could not prevent atrial arrhythmia, either immediate or short term follow-up, in 2 of 4 patients who had a history of paroxysmal AF before ablation. These patients had a higher incidence of underlying heart disease and they also received a greater number of applications of RF current (31.3 ± 25.7 *vs* 18.2 ± 10.5) and had longer fluoroscopic and procedure times (60.9 ± 46.8 *vs* 32.5 ± 20.1 and 183.3 ± 85.1 *vs* 144.2 ± 63.8 minutes, respectively). However, ablation of AFL in the patients who had a history of AF still may be helpful because AF and/or the ventricular response rate may be tolerated better by the patient or easier controls by pharmacologic treatment. Moreover, 50 per cent of patients with AF before ablation had remained arrhythmic record free after ablation of AFL[15], similar to our study and 1 of our patients who had inducible sustained AF and needed electrical cardioversion to converts AF to sinus rhythm is still free of atrial arrhythmia. These may suggest that AF before and after successful ablation of AFL were not necessarily an indicator of occurring AF after ablation of atrial arrhythmia.

SUMMARY

RFCA of atrial flutter type I, using anatomical approach, is a safe technique and effective treatment to terminate and prevent this arrhythmia in short term follow-up. Recurrent AFL and AF are still high especially in patients with underlying heart disease and having a previous episode of AF before ablation. However, AF occurring before and after ablation of AFL are not necessarily an indicator of occurring AF after ablation. In conclusion, RFCA may be considered as alternative treatment in patients with AFL but long-term results are still lacking.

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การใช้ Radiofrequency Catheter Ablation ในผู้ป่วย type I atrial flutter

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คณะผู้รายงานได้ศึกษาผลการรักษาภาวะ type I atrial flutter (AFL) โดยใช้ radiofrequency catheter ablation (RFCA) ในผู้ป่วย 10 ราย อายุเฉลี่ย 59.4 ± 11.2 ปี ผู้ป่วยทั้งหมดมีอาการ และไม่ตอบสนองต่อยาต้านการเต้นผิดจังหวะเป็นเวลาเฉลี่ย 5.7 ± 4.9 ปี ผู้ป่วย 4 ราย (40%) มีโรคอื่นโดยที่ 2 ราย เป็น dilated cardiomyopathy, Ebstein's anomaly 1 ราย และหลอดลมอุดกั้นเรื้อรัง 1 ราย การทำ RFCA สามารถหยุดยั้งภาวะหัวใจเต้นผิดจังหวะได้ 90% โดยไม่มีภาวะแทรกซ้อนที่รุนแรง ผู้ป่วย 2 รายเกิด AFL และ atrial fibrillation ในระยะเวลา 24.0 ± 28.7 สัปดาห์ ทำให้การรักษาด้วย RFCA ได้ผล 70% ดังนั้น RFCA เป็นวิธีการรักษา AFL ที่ได้ผลดีอีกวิธีหนึ่งโดยเฉพาะผู้ป่วยที่ไม่ตอบสนองต่อยาต้านการเต้นผิดจังหวะของหัวใจ

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