Acute myocardial infarction after radiofrequency catheter ablation of typical atrial flutter: histopathological findings and etiopathogenetic hypothesis

Biagio Sassone, Ornella Leone*, Giuseppe Nicola Martinelli*, Giuseppe Di Pasquale

*Division of Cardiology, Hospital of Bentivoglio, Bentivoglio (BO), *Institute of Pathological Anatomy, University of Bologna, Bologna, Italy*

Key words:
Atrial flutter; Catheter ablation; Occlusion.

The right atrial inferior cavotricuspid isthmus represents the targeting site for radiofrequency (RF) current application during ablation treatment of typical atrial flutter. Despite the vicinity of the right coronary artery (RCA) to the RF application site and the long energy exposure needed to achieve electrophysiological success, reports about direct thermal damage of the coronary vessel during ablation of the cavotricuspid isthmus are rare and anecdotal. The present is the first case report describing the cardiac macroscopic and histological examination in a patient who died of cardiac rupture, as a complication of a myocardial infarction occurring after a standard procedure of RF ablation of typical atrial flutter. In consideration of the proximity we found between the RF energy-dependent tissue damage and the RCA, thermal-related damage of RCA during ablation of typical atrial flutter should always be considered as a potentially harmful risk of the procedure.

(Ital Heart J 2004; 5 (5): 403-407)

Received December 5, 2003; revision received February 26, 2004; accepted March 2, 2004.

Address:

Dr. Biagio Sassone

U.O. di Cardiologia
Ospedale di Bentivoglio
Via Marconi, 35
40010 Bentivoglio (BO)
E-mail:
biagio64@libero.it

Introduction

Radiofrequency (RF) catheter ablation is widely considered the treatment of choice for patients suffering from recurrent typical atrial flutter refractory to pharmacological therapy. Acute success is presumed in nearly 90% whereas severe complications secondary to the ablation procedure are rare and mainly represented by atrioventricular block, cardiac tamponade, and stroke[1-3]. Acute right coronary artery (RCA) damage is an additional potential severe complication of RF ablation in typical atrial flutter but, to date, reports are sparse and anecdotal.

This is the first documentation of the macroscopic and histological study of the heart in a patient treated by RF catheter ablation for typical atrial flutter complicated by an acute myocardial infarction, who died of cardiac rupture 3 days after the procedure.

Case report

A 79-year-old male was referred to our institution for symptomatic paroxysmal atrial flutter refractory to pharmacological therapy. The only risk factor for coronary artery disease was hypertension and no history of chest pain was present. An essentially normal structure of the heart had been documented during a recent Doppler echo-cardiographic examination. The patient underwent electrophysiological study and a typical atrial flutter (caudocranial septal and counterclockwise right atrial activation) was induced. In accordance with our ablation procedure protocol, venous access to the heart was achieved via a right femoral and internal jugular vein approach, under local anesthesia and fluoroscopic control. A diagnostic steerable 7F decapolar catheter (EP Technologies, Inc., Sylmar, CA, USA) was used to record the right atrial activation sequence around the tricuspid annulus. A diagnostic pre-curved (Josephson) 6F quadripolar catheter (St. Jude Medical, Inc., Natick, MA, USA) was inserted into the coronary sinus with the proximal pole placed on the ostium for pacing and recording. Finally, a steerable 8 mm tip ablation catheter (EP Technologies, Inc.), equipped with a thermocouple, was positioned within the right inferior isthmus and used to draw a line of RF lesions extending from the tricuspid annulus to the inferior vena cava. The patient was in chronic treatment with war-

farin (INR 2.0-3.0) and, at the time of admission, was kept hypocoagulable with intravenous heparin; warfarin was stopped only when the activated partial thromboplastin time value was double the basal value. Heparin was stopped 6 hours before the procedure and a bolus of an additional dose of 5000 IU was administered at the time of the insertion of the catheters.

RF current was delivered by a radiofrequency generator (EP Technologies, Inc. - 1000 XP) in the monopolar mode and under temperature guidance with a target temperature of 70°C and an upper power output limit of 100 W. Standard RF current application was by sequential points, lasting 60 s for each point. Atrial flutter ended during the third RF current application and restoration of sinus rhythm was achieved. However, twelve current applications were necessary to obtain a bidirectional isthmus conduction block. During the last RF current application, the patient complained of epigastric pain, irradiating to the precordial area in the absence of ECG alterations. RF current delivery was immediately interrupted and the catheters removed from the right atrium. The maximum temperature and energy reached were 67°C and 80 W respectively, with the impedance within normal values (78-85 W). A few minutes later, 12-lead ECG showed ST-T segment elevation (maximum 3 mV) in leads II, III and aVF with a persistent epigastric/precordial pain. Heparin, aspirin and intravenous nitrate were promptly administered and the patient, in stable hemodynamic conditions, was transferred to the coronary care unit where his chest pain as well as the ECG alterations began decreasing. An echocardiographic examination showed neither asynergic segments of the heart nor pericardial effusion. Because of the gradual normalization of the ECG and clinical symptomatology, and the relatively small size of the ischemic area, no intervention for acute coronary revascularization was adopted. One hour later the clinical picture had resolved and an acute non-Q wave myocardial infarction was diagnosed on the basis of cardiac enzyme release (troponin T blood level 0.6 ng/ml, normal value < 0.01 ng/ml); in view of this, an elective coronary angiography was planned. In spite of the absence of any symptoms during the following 2 days, the patient suddenly died of electromechanical dissociation 72 hours after the acute myocardial infarction. An echocardiographic examination showed a massive pericardial effusion.

Macroscopic and histological observation. Autopsy was performed 48 hours later confirming, as the cause of death, a cardiac rupture secondary to acute myocardial infarction. The cleft was identified at the middle sector of the left ventricular inferior wall, where a medium size myocardial infarction was noted. More than 350 ml of hemorrhagic effusion were evacuated from the pericardial sac.

The lumen of the RCA appeared patent along the proximal and middle segments, whereas at the distal segment, near the crux cordis and immediately before the emergence of the interventricular posterior descending artery, a 15 mm long fibrous atheromatous plaque occupying > 90% of the lumen and complicated by an acute occlusive thrombosis, was found (Fig. 1). The left coronary tree was subnormal whereas the lumen of the coronary sinus was unaffected.

Macroscopic examination of the right atrial inferior cavotricuspid isthmus showed that the RF lesion consisted of a rounded subendocardial hemorrhagic infiltration, localized at the posterior-middle sector of the region[4] (Fig. 2).

Multiple formalin-fixed specimens taken from this lesion were routinely processed and 2 mm thick sections were cut and stained with hematoxylin-eosin.

At histology, the RF lesion was found to consist of a clear-cut coagulative necrosis of the atrial myocardium and of a fibrino-hemorrhagic infiltration extending from the endocardial surface at the ablation site, through a very thin atrial myocardium, to the subepicardial fat. The maximum depth of damaged tissue was 6.5 mm and the minimum distance from the RCA edge,

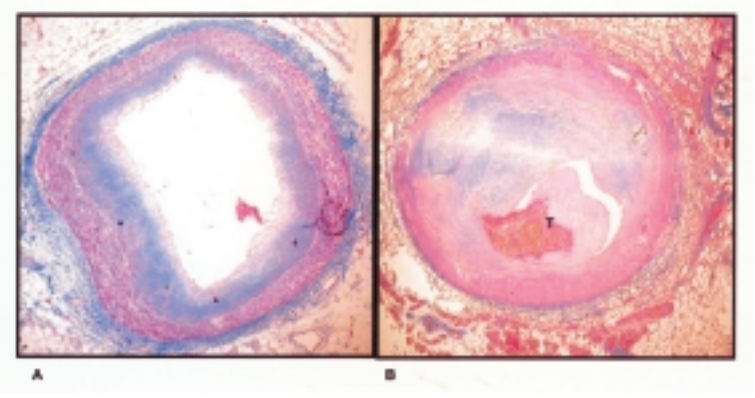

Figure 1. *In* panel A *the patent lumen of the right coronary artery along the proximal segment is shown. Only slight intimal hyperplasia is evident (Mallory trichrome, original magnification 100×). In* panel B, *the fibrous atheromatous plaque complicated by an acute occlusive thrombosis is represented (Mallory trichrome, original magnification 100×). T = thrombus.*

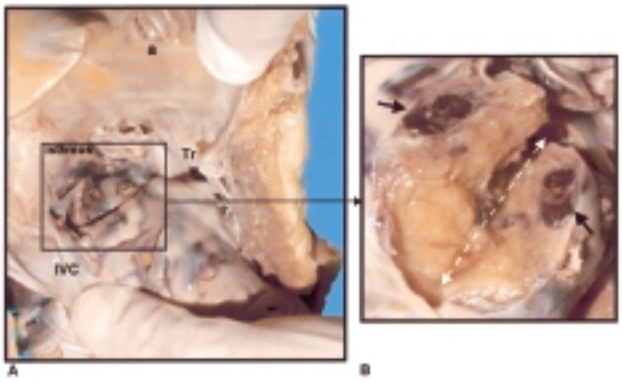

Figure 2. A: *macroscopic picture of the radiofrequency lesion.* B: *detail. A rounded subendocardial hemorrhagic lesion* (black arrows) *located at the right atrial inferior cavotricuspid isthmus. a = right auricular appendage; IVC = inferior vena cava; Tr = tricuspid valve.*

found in the epicardial fat of the vestibule, was 1.7 mm (Fig. 3A). No involvement of the RCA wall, such as any inflammatory and/or necrotic reaction, was found (Fig. 3B).

Discussion

This is the first report including a cardiac macroscopic and histological study in a patient who died of an acute myocardial infarction due to an acute RCA occlusion secondary to cavotricuspid isthmus RF catheter ablation for typical atrial flutter.

In 1993, the Multicenter European Radiofrequency Survey (MERFS), reporting on 4398 transcatheter ablation procedures, did not mention acute coronary artery occlusion as a complication[3]. Nevertheless, there are several later reports on coronary artery complications due to RF catheter ablation procedures in adults, but almost exclusively confined to the left coronary artery and mainly related to the treatment of accessory atrioventricular pathways[5-13]. On the other hand, despite the vicinity of the endocardial ablation site to the RCA and the longer energy exposure needed to achieve electrophysiological success, reports about vessel damage during cavotricuspid isthmus RF ablation for typical atrial flutter are anecdotal and sparse.

In an experimental model, Paul et al.[14] first demonstrated, 48 hours after RF current application in a temperature-guided mode (75°C) at the right atrial portion of the tricuspid valve annulus, transmural coagulative necrosis with perifocal hemorrhage and lymphocytic infiltration around the right atrial lesions and extending into the layers of the RCA in 4 of 5 pigs. As a late sequela of RF current application, 6 months later a 25 and 45% stenosis of the RCA lumen due to intimal thickening respectively occurred in 2 pigs. Strikingly, the authors did not find any correlation between the lesion size and the quantity of energy delivered.

Recently in animal studies, Madrid et al.[15,16] investigated the effect of an irrigated-tip catheter, capable of making deeper lesion lines by RF ablation, compared to the standard catheter with regard to the successful acute electrophysiological achievement of cavotricuspid isthmus bidirectional block and the subacute anatomic characteristics of the lesions. They demonstrated that cooled catheters achieved a complete line of electrophysiological and anatomical block in a significantly higher percentage than the standard catheters while histological evaluation showed an inflammatory infiltration around the site of RF current application extending into the adventitia, but sporadically even in the muscular wall, of the RCA, although the endothelium was never involved and necrosis never occurred.

Jais et al.[17] performed cavotricuspid isthmus transcatheter RF ablation in 50 patients (30 with a cooled irrigated-tip catheter and 20 with a standard catheter) suffering from typical atrial flutter. All patients underwent a pre-ablation and post-ablation coronary angiography. Complete bidirectional isthmus block was achieved for all patients and no change was demonstrated in the post-ablation coronary angiogram,

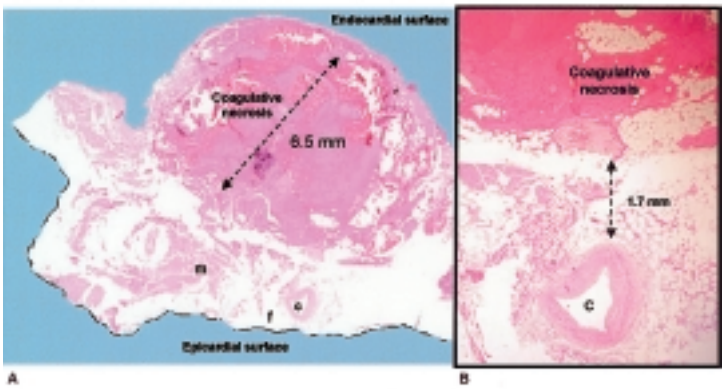

Figure 3. *The histological aspect of the radiofrequency lesion, consisting of coagulative necrosis of the atrial myocardium (m) and fibrino-hemorrhagic infiltration. In* panel A*, the maximum depth of the lesion is shown (hematoxylin-eosin, original magnification 25×); in* panel B*, the minimum distance between the lesion rim and the right coronary artery is illustrated (hematoxylin-eosin, original magnification 100×). c = right coronary artery lumen; f = subepicardial fat.*

whichever catheter was used, including patients with documented pre-ablation RCA disease.

Ouali et al.[18] described the first case of acute RCA occlusion in an adult patient during RF catheter ablation of typical atrial flutter. An acute total thrombotic occlusion of the posterior left ventricular branch of the RCA, responsible for an acute non-Q wave myocardial infarction, was observed at coronary angiography. Moreover, a severe stenosis in the mid portion of the RCA was also present. The authors hypothesized a relationship between the RF ablation site and the coronary thrombosis, by positioning a catheter at the targeted ablation site (right inferior cavotricuspid isthmus) during the acquisition of the angiogram. A precise superimposition between the tip of the catheter and the coronary occlusion was considered as proof of the direct thermal effect of RF delivery on the arterial damage.

In conclusion, coronary artery damage due to direct thermal energy from RF current has been a concern since the inception of RF ablation. Acute coronary artery occlusion due to a direct thermal effect has been thought to result from different mechanisms: 1) focal spasm or vessel wall edema, due to the thermal impact, in patients with normal coronary arteries at angiography[19-22]; 2) intravascular electrocoagulation with local vessel occlusion because of the extreme vicinity of the artery to the RF energy application site, in small heart size patients, especially when associated with long energy exposure[23]; 3) thrombus formation at the ablation target site with distal embolization[24].

In our case, the RCA segment closer to the RF energy-dependent tissue damage showed a patent lumen without any lesion of the arterial layers, whereas distally to the ablation site we found a severe stenosis (> 90%) at the RCA distal segment followed, proximally to the emergence of the interventricular posterior descending coronary artery, by an atheromatous plaque complicated by acute occlusive thrombosis responsible for the myocardial infarction. Probably the thermal side effects of RF energy on the arteries are limited by the luminal blood flow, which serves as a heat dispelling sink, keeping the vessel wall cool when the surrounding connective tissue is heated[25]. In our patient the severe narrowing in the distal segment of the RCA caused a slowing flow in the distal vessels and this condition may have amplified the RF energy thermal impact upon a complex morphology plaque, representing a nidus of shear forces that precipitated the hemocoagulative cascade leading to the acute occlusive thrombosis.

In consideration of the proximity between the RCA and the region targeted for the RF current application site, that is the right inferior cavotricuspid isthmus, thermal damage of the RCA should always be considered as potentially harmful during ablation treatment of typical atrial flutter, especially in elderly patients with suspected coronary artery disease.

Nevertheless, in the light of the large recently published series[26,27], where myocardial infarction following atrial flutter ablation has not been reported, occlusive damage of the RCA should be considered as a rare complication of such a safe and effective ablation procedure.

References

1. Poty H, Saoudi N, Abdel Aziz A, Nair M, Letac B. Radiofrequency catheter ablation of type I atrial flutter. Prediction of late success by electrophysiological criteria. Circulation 1995; 92: 1389-92.
2. Anselme F, Klug D, Scanu P, et al. Randomized comparison of two targets in typical atrial flutter ablation. Am J Cardiol 2000; 85: 1302-7.
3. Hindricks G, on behalf of the MERFS Investigators. The Multicenter European Radiofrequency Survey (MERFS): complications of radiofrequency catheter ablation of arrhythmias. Eur Heart J 1993; 14: 1644-53.
4. Cabrera JA, Sanchez-Quintana D, Ho SY, et al. Angiographic anatomy of the inferior right atrial isthmus in patients with and without history of common atrial flutter. Circulation 1999; 99: 3017-23.
5. Dinckal H, Yucel O, Kirilmaz A, Karaca M, Kilicaslan F, Dokumaci B. Left anterior descending coronary artery occlusion after left lateral free wall accessory pathway ablation: what is the possible mechanism? Europace 2003; 5: 263-6.
6. Kosinski DJ, Burket MW, Durzinsky D. Occlusion of the left main coronary artery during radiofrequency ablation for the Wolff-Parkinson-White syndrome. Eur J Card Pacing Electrophysiol 1993; 3: 63-6.
7. Chatelain P, Zimmermann M, Weber R, Campanini C, Adamec R. Acute coronary occlusion secondary to radiofrequency catheter ablation of a left lateral accessory pathway. Eur Heart J 1995; 16: 859-61.
8. Calkins H, Yong P, Miller JM, et al, for the ATAKAR Multicenter Investigators Group. Catheter ablation of the accessory pathway, atrioventricular nodal reentrant tachycardia, and the atrioventricular junction: final results of a prospective multicenter clinical trial. Circulation 1998; 98: 262-70.
9. Janeira LF. Coronary artery dissection complicating radiofrequency catheter ablation via the retrograde approach. Pacing Clin Electrophysiol 1998; 21: 1327-8.
10. Pons M, Beck L, Leclercq F, Ferriere M, Albat B, Davy JM. Chronic left main coronary artery occlusion: a complication of radiofrequency ablation of idiopathic left ventricular tachycardia. Pacing Clin Electrophysiol 1997; 20: 1874-6.
11. Lesh MD, Coggins DL, Ports TA. Coronary air embolism complicating transseptal radiofrequency ablation of left free-wall accessory pathways. Pacing Clin Electrophysiol 1992; 15: 1105-8.
12. Thakur RK, Klein GJ, Yee R. Radiofrequency catheter ablation in patients with Wolff-Parkinson-White syndrome. CMAJ 1994; 151: 771-6.
13. Hope EJ, Haigney MC, Calkins H, Resar JR. Left main coronary thrombosis after radiofrequency ablation: successful treatment with percutaneous transluminal angioplasty. Am Heart J 1995; 129: 1217-9.
14. Paul T, Bokenkamp R, Mahnert B, Trappe HJ. Coronary artery involvement early and later after radiofrequency current application in young pigs. Am Heart J 1997; 133: 436-40.
15. Madrid AH, Rebollo JM, Del Rey JM, et al. Randomized comparison of efficacy of cooled tip catheter ablation of atrial flutter: anatomic versus electrophysiological com-

plete isthmus block. Pacing Clin Electrophysiol 2001; 24: 1525-33.
16. Madrid AH, Gonzales Rebollo JM, Del Rey JM, et al. Macroscopic and microscopic study of the right coronary artery after radiofrequency catheter ablation of cavotricuspid isthmus in an experimental model. Rev Esp Cardiol 2001; 54: 693-702.
17. Jais P, Shah DC, Haissaguerre M, et al. Prospective randomized comparison of irrigated-tip versus conventional tip catheter for ablation of common flutter. Circulation 2000; 101: 772-6.
18. Ouali S, Anselme F, Savoure A, Cribier A. Acute coronary occlusion during radiofrequency catheter ablation of typical atrial flutter. J Cardiovasc Electrophysiol 2002; 13: 1047-9.
19. Calkins H, Sousa J, El-Atassi R, et al. Diagnosis and cure of the Wolff-Parkinson-White syndrome or paroxysmal supraventricular tachycardias during a single electrophysiologic test. N Engl J Med 1991; 324: 1612-8.
20. Lesh MD, Van Hare GF, Sheinman MM, Ports TA, Epstein LA. Comparison of the retrograde and transseptal methods for ablation of left free wall accessory pathways. J Am Coll Cardiol 1993; 22: 542-9.
21. Strobel GG, Trehan S, Compton S, Judd VE, Day RW, Etheridge SP. Successful pediatric stenting of a nonthrombotic coronary occlusion as a complication of radiofrequency catheter ablation. Pacing Clin Electrophysiol 2001; 24: 1026-8.
22. Lesh MD, Van Hare GF, Schamp DJ, et al. Curative percutaneous catheter ablation using radiofrequency energy for accessory pathways in all locations: results in 100 consecutive patients. J Am Coll Cardiol 1992; 19: 1303-9.
23. Bokenkamp R, Wibbelt G, Sturm M, et al. Effects of intracardiac radiofrequency current application on coronary artery vessels in young pigs. J Cardiovasc Electrophysiol 2000; 11: 565-71.
24. Dinckal H, Yucel O, Kirilmaz A, Karaca M, Kilikaslan F, Dokumaci B. Left anterior descending coronary artery occlusion after left lateral free wall accessory pathway ablation. What is the possible mechanism? Europace 2003; 5: 263-6.
25. Delacretaz E, Stevenson WG, Winters GL, Friedman PL. Radiofrequency ablation of atrial flutter. Circulation 1999; 99: E1-E2.
26. Scheinman MM, Huang S. The 1998 NASPE prospective catheter ablation registry. Pacing Clin Electrophysiol 2000; 23: 1020-8.
27. Schmieder S, Ndrepepa G, Dong J, et al. Acute and long-term results of radiofrequency ablation of common atrial flutter and the influence of the right atrial isthmus ablation on the occurrence of atrial fibrillation. Eur Heart J 2003; 24: 956-62.