Dobutamine Facilitates Reversion of Atrial and Ventricular Stunning in a Patient after Catheter Ablation of Persistent Atrial Flutter

Shih-Chung Huang, 1,2 Tsung-Neng Tsai, 1 Shih-Ping Yang, 1 Kai-Min Chu1 and Wei-Shiang Lin 1

The development of cardiomyopathy may be correlated to atrial or to ventricular arrhythmias. The associated heart failure generally starts to improve within days to months of achieving ventricular rhythm control. However, transient myocardial stunning after transcatheter ablation leading to congestive heart failure with cardiogenic shock in tachycardia-induced cardiomyopathy was less mentioned before. A 33-year-old man, presented with a persistent atrial flutter with a rapid ventricular rate for three months, had complications with tachycardia-induced cardiomyopathy [left ventricle (LV) ejection fraction (EF) = 35%]. Atrial and ventricular (A/V) stunning with deterioration of the LV function (EF = 20%) developed after successful restoration of the sinus rhythm by catheter ablation. His A/V contractility improved after short-term intravenous treatment with dobutamine at 5 μg/kg/min. Four months later, the patient was still in sinus rhythm with an improved LV contractility (EF = 50%) and increased atrial contraction wave from 31.5 ± 5.9 cm/sec to 58.7 ± 8.5 cm/sec. This suggests that dobutamine may facilitate reversion of the A/V stunning in patients with tachycardia-induced cardiomyopathy.

**Key Words:** Atrial and ventricular stunning • Atrial flutter • Dobutamine • Tachycardia-induced cardiomyopathy

**INTRODUCTION**

Tachycardia-induced cardiomyopathy (TIC) has been described as a distinct clinical entity, and is generally accepted to be the most frequently unrecognized curable cause of heart failure. An increasing number of reports indicate that improvement of dilated cardiomyopathy can be achieved after control of the arrhythmia by pharmacologic, surgical and, more recently, transvenous catheter ablation techniques.1 However, conversion of atrial arrhythmia to sinus rhythm is often associated with transient atrial dysfunction, which has been termed “stunning”, leading to congestive heart failure associated with ventricular stunning. atrial/ventricular (A/V) stunning after conversion of atrial fibrillation (AF) or atrial flutter (AFL) has been reported in various methods, including transthoracic electrical cardioversion, low-energy internal electrical cardioversion, pharmacological cardioversion, spontaneous conversion, conversion by overdrive pacing and radiofrequency ablation. Unfortunately, the effective prevention or treatment of A/V stunning remains a challenge. Several cardioactive agents have been studied in this regard, such as verapamil, acetylsalicylic acid, isoproterenol, dobutamine and sotalol.2,3 Here, we report an unique case of AFL-related TIC, who developed A/V stunning after radiofrequency catheter ablation leading to rapid deterioration of the left ventricular (LV) systolic function and cardiogenic shock. His atrial and ventricle

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contractility [ejection fraction (EF) = 29%] improved after infusion of dobutamine at a low dose (5 μg/kg/min).

CASE REPORT

A 33-year-old man had a two-year history of hypertensive cardiovascular disease and chronic glomerulonephritis without regular medication. Three months before admission, he presented with chest tightness, palpitation and exertional dyspnea. The patient visited the outpatient department, where serum biochemistry analysis revealed chronic renal failure in the uremic state (blood urine nitrogen = 168 mg/dl, creatine = 16.1 mg/dl). In addition, electrocardiography showed the presence of AFL, with a ventricular rate of 142 beats/min (Figure 1A). The echocardiography further revealed a dilated four-chamber and generalized hypokinesis of the left ventricle, with a low LV EF (35%, Table 1). A diagnosis of uremia with fluid overload was tentatively suggested to be the cause of the patient’s dyspnea. Unfortunately, regular hemodialysis failed to improve the dyspnea. AFL-related TIC was impressed and the procedure of catheter ablation was scheduled.

On admission, the patient’s vital signs revealed a blood pressure of 124/80 mmHg, a heart rate of 144 beats/min and a respiratory rate of 21 breaths/min. Physical examination disclosed clammy extremities with pedal edema. The jugular venous pressure was elevated and the third heart sound was dominant. An electrophysiological study performed on the following day disclosed a counterclockwise AFL with an atrial cycle length of 208 ms (Figure 1B). Radiofrequency catheter ablation with anatomically guided linear ablation of the cava-tricuspid isthmus was performed with a 8-mm-tip ablation catheter (Safire, St. Jude Medical, Minnetonka, MN, USA) with the temperature preset at 60 °C and radiofrequency pulse duration of 120 seconds. Atrial flutter was terminated during linear catheter ablation of the inferior vena cava-tricuspid valve annulus (IVC-TV)-dependent isthmus, with a bidirectional conduction block (Figure 1C). After the procedure, severe shortness of breath and hypotension (blood pressure = 96/54 mmHg) developed, the transthoracic echocardiography revealed no pericardial effusion, but hypokinesis of the left atrium with low transmitral velocity of the atrial contraction wave (A-wave = 31.5 ± 5.9 cm/sec) (Figure 2A) and deterioration of the LV function were

Figure 1. (A) Atrial flutter with ventricular rate of 142 beats/min before ablation, (B) Counterclockwise AFL with atrial cycle length of 208 ms, (C) Termination of AFL during RFCA, (D) The 12-lead ECG showed sinus rhythm after RFCA 4 months later. Abbreviations: I, II and V1 = surface ECG leads I, II and V1, HRAP, high right atrium proximal; HRAD, high right atrium distal; HISP, His bundle proximal; HISD, His bundle distal; CSO, coronary sinus ostium; CSM, coronary sinus middle; CSD, coronary sinus distal; RVAP, right ventricle apex proximal; RVAD, right ventricle apex distal.
noted (LVEF = 20%, Table 1). Postablation myocardial stunning with cardiogenic shock was impressed. In addition to fluid resuscitation, dobutamine was administered at a rate of 5 μg/kg/min. The myocardial contractility improved dramatically after 30 min of dobutamine infusion. The patient’s vital signs resumed and dobutamine was discontinued 36 hours later. The patient remained in sinus rhythm (Figure 1D) four months after the procedure and the dyspnea was relieved too. The follow-up echocardiography showed a regressive atrial chamber diameter, preserved ventricular contractility, and improved transmitral velocity of the atrial contraction wave (A-wave = 58.7 ± 8.5 cm/sec) (Figure 2B).

**DISCUSSION**

In 1913, Gossage et al. first described tachycardia-induced cardiomyopathy in a patient with AF. Brill et al. later reported another case of AF with reversion of

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LAD, left atrium diameter; LVED, left ventricle end diastolic diameter; LVES, left ventricle end systolic diameter; LVEF, left ventricle ejection fraction.

*Figure 2.* Transmitral velocity of the atrial contraction wave (A-wave) measured to be: (A) 31.5 ± 5.9 cm/sec, one hour after ablation, and (B) 58.7 ± 8.5 cm/sec, 4 months after ablation.
heart failure after restoration of sinus rhythm. Tachycardia-induced cardiomyopathy can be defined as a condition characterized by atrial or ventricular myocardial dysfunction resulting solely from increased atrial or ventricular rates. The rate and duration of the tachycardia are the major determinants of the onset, progression and reversibility of this condition. At the cellular level, myocytes in tachycardia-induced cardiomyopathy showed loss of contractility, altered alignment, increased resting length, decreased beta-adrenergic density, and decreased sensitivity to beta-agonists and extracellular calcium. However, once the tachyarrhythmia is under control, this is a reversible form of heart failure.

TIC can involve atrial and ventricular stunning. Atrial stunning, a transient mechanical dysfunction of the atrium and atrial appendage, has been reported when atrial arrhythmia is converted to sinus rhythm, regardless of the method employed. The suggested mechanisms are: tachycardia-induced atrial cardiomyopathy, atrial cytosolic calcium alterations with downregulation of the L-type Ca\(^{2+}\) channels and upregulation of the Na\(^{+}/\)Ca\(^{2+}\) exchanger, atrial hibernation with myocyte dedifferentiation, myolysis and atrial fibrosis. Atrial stunning can occur immediately after conversion to sinus rhythm and improve gradually, and complete resolution with the duration from a few minutes to as long as 2-6 weeks later, depending on the duration of the preceding atrial arrhythmia, atrial size, and the presence of structural heart disease. The prevalence of atrial stunning after atrial arrhythmia varies considerably, with a maximum prevalence of 80% occurring after AFL conversion using RFCA.

Topics regarding ventricular stunning with rapid deterioration after tachycardia were less mentioned before. It can occur as soon as 24 hr, with continued deterioration in ventricular function for up to 3 to 5 weeks, resulting in end-stage heart failure. The mechanism for ventricular cardiomyopathy could be due to (1) myocardial energy depletion and impaired energy utilization; (2) myocardial ischemia; (3) abnormalities of cardiac calcium regulation; and (4) myocyte and extracellular matrix remodeling. Unfortunately, a definitive treatment for atrial or ventricular stunning is still lacking. Several cardioactive compounds have been studied as potential protective agents against myocardial stunning, including verapamil, acetylstrophenathidine, isoproterenol, sotalol, and dobutamine. However, some of these agents may exacerbate hypotension when myocardial stunning occurs in association with cardiogenic shock.

Dobutamine, a sympathomimetic amine inotropic agent that has been used for cardiogenic shock, can improve myocardial contractility via activation of cAMP/protein kinase A-mediated facilitation of intracellular Ca\(^{2+}\) mobilization. In the case reported here, a chronic renal failure patient in uremic state who had suffered from persistent AFL with tachycardia-induced cardiomyopathy underwent linear catheter ablation of the IVC-TVA-dependent isthmus with bidirectional conduction block. Transient deterioration of LVEF was found after ablation, and transthoracic echocardiography disclosed poor ventricular contractility and atrial stunning. The myocardial stunning deteriorated and resulted in cardiogenic shock. Since large fluid supplements were contraindicated because of the patient’s uremic state, and other medications for A/V stunning were not suitable because of the patient’s shock state, dobutamine infusion was considered. This was based on the assumption that dobutamine could not only overcome the atrium stunning, which is supported by results obtained in an animal model, but could also improve the myocardial contractility associated with myocardial stunning. After dobutamine infusion, the contractility of the patient’s LA and LV was dramatically improved. Therefore, we suggest that dobutamine could be one of the agents of choice to treat myocardial stunning after reversion of TIC. However, the role of short-term use of dobutamine in the recovery phase of TIC remains unclear. It could be only transient effect of dobutamine which corrected the status of cardiogenic shock by overcoming the myocardial stunning after RFCA. The improvement of A/V function show 4 months later may be due to progressive contractile, electrical or structural remodeling and have nothing to do with dobutamine.

In conclusion, we emphasize the fact that A/V stunning can develop soon after catheter ablation in patients presenting with AFL-induced cardiomyopathy. More importantly, we showed that dobutamine could facilitate the process of reversion of A/V stunning by transiently abolishing the vicious cycle of heart failure and may be considered as a bridge therapy for this type of cardiogenic shock. However, the long-term effect of dobutamine for TIC needs further study.
REFERENCES


