Pulmonary Edema After Cardioversion for Paroxysmal Atrial Flutter

— Left Ventricular Diastolic Dysfunction Induced by Direct Current Shock —

Nobuhiko Kobayashi, MD; Mari Takayama, MD; Shuichi Yamaura, MD;
Hiroyasu Ushimaru, MD; Tomio Ochi, MD; Muneyasu Saito, MD*

This report describes a patient with the pulmonary edema after cardioversion for paroxysmal atrial flutter without organic heart disease. A 68-year-old man was admitted to hospital for paroxysmal atrial flutter. Antiarrhythmic agents were not effective, and direct current cardioversion was performed on the 4th hospital day. Three hours after cardioversion, the patient complained of dyspnea, and a chest X-ray showed pulmonary edema. He responded to oxygen, intravenous furosemide and drip infusion of nitroglycerine. During tapering of the medication, his condition remained stable. The patient was discharged on the 7th day after admission. Echocardiographic findings indicated that transient left ventricular diastolic dysfunction due to direct current shock was the most likely cause of the lung edema. (*Jpn Circ J* 2000; 64: 76–79)

Key Words: Atrial flutter; Cardioversion; Left ventricular dysfunction; Pulmonary edema

Direct current cardioversion for atrial arrhythmias occasionally causes pulmonary edema and the mechanism of this is still controversial. Pulmonary edema mainly occurs in patients with chronic atrial fibrillation and organic heart disease, such as valvular disease and cardiomyopathy^{1,2}. We report a patient with pulmonary edema after cardioversion for lone atrial flutter that continued for 4 days, and discuss the mechanism of this complication.

Case Report

A 68-year-old man was admitted to hospital for paroxysmal atrial flutter. He had been well until the evening prior to admission, when he suddenly experienced chest discomfort and palpitation. When the symptoms did not improve after several hours, he came to hospital.

Three years before he had undergone a left upper lobectomy of the lung because of lung cancer. He had no other relevant history, such as heart disease or hypertension. On examination, his pulse was 130 beats/min and irregular. Electrocardiography (ECG) showed atrial flutter with atrioventricular conduction of 2 to 1 and 3 to 1 (Fig 1A). He was treated with intravenous infusion of procainamide, but sinus rhythm was not restored. After admission, oral administration of pilsicainide was also not effective, and direct current cardioversion was performed on the 4th hospital day. Chest X-ray showed no abnormal lesions except for the sequela of the left upper lobectomy (Fig 1A).

Transthoracic echocardiography revealed a kinetically well left ventricle (LV) without asynergy and hypertrophy. The LV end-diastolic dimension (LVDd) and LV end-systolic dimension (LVDs) were 38.7 and 23.7 mm, respectively. The LV ejection fraction (LVEF) was calculated at 70% using the Teichholz method³. Both the aortic and mitral valves appeared normal. The right side of the heart was also normal. The isovolumic relaxation time (IVRT), which was measured by phonocardiography from aortic closure to the onset of mitral flow, was 65 ms at a heart rate of 130 beats/min. Transesophageal echocardiography confirmed no intra-atrial thrombi prior to the countershock. After an intravenous injection of thiopental, the patient was cardioverted to sinus rhythm with a single shock of 50 J. Three hours after cardioversion, the patient complained of severe dyspnea. His blood pressure was 150/90 mmHg, and his pulse was 88 beats/min and regular. Chest examination revealed wheezing and moist rales throughout both lungs. Pulseoxymeter indicated 68% arterial oxygen saturation. ECG showed normal sinus rhythm (Fig 1B) and no significant ST–T changes. Chest X-ray showed pulmonary edema and pleural effusion (Fig 1B), but echocardiography detected no LV asynergy although LVDd and LVDs were slightly increased to 41.8 and 27.1 mm, respectively. The LVEF was calculated at 65%. Mild tricuspid regurgitation was observed at a pressure gradient of 47 mmHg. On pulsed wave Doppler measurement, the transmitral flow velocity was 0.97 m/s in early diastole and 0.37 m/s in late diastole; IVRT was shortened to 36 ms (Fig 2A). The right ventricular inflow velocity was 0.45 m/s in early diastole and 0.42 m/s in late diastole. The color M-mode flow propagation velocity (Vp) during LV early filling was 36.5 cm/s, using the method advocated by Takatsui et al⁴ (Fig 2B). The patient responded to oxygen, intravenous furosemide and drip infusion of nitroglycerine. The next day, he had no dyspnea and chest examination revealed

(Received July 7, 1999; revised manuscript received September 16, 1999; accepted September 21, 1999)

Department of Internal Medicine, Yodakubo Hospital, Nagano and *Omiya Medical Center, Jichi Medical School, Omiya, Japan

Mailing address: Nobuhiko Kobayashi, MD, Department of Internal Medicine, Yodakubo Hospital, 2857 Nagato Town, Nagano 386-0603, Japan. E-mail: nkoba@valley.ne.jp

Fig 1. (A) Chest X-ray and electrocardiogram before cardioversion. Atrial flutter with atrioventricular conduction of 2 to 1 and 3 to 1 (V₁ lead). (B) Chest X-ray and electrocardiogram at the onset of lung edema after cardioversion. Pulmonary edema and pleural effusion on sinus rhythm (V₁ lead).

Fig 2. Echocardiography at the onset of lung edema. (A) Transmittal flow spectrum by pulsed wave Doppler with phonocardiography. The isovolumic relaxation time is shortened to 36 ms (white broken lines). (B) Color M-mode Doppler imaging. The slope of the aliasing flow shows the flow propagation velocity of 36.5 cm/s during left ventricular early filling (white arrow).

moist rales only in the lower dorsal region. Echocardiography showed an improved LVEF up to 75%, and IVRT was 52 ms. The Vp had also improved to 65 cm/s. During

tapering of the medication, his condition remained stable and the patient was discharged on the 7th day after admission.

Discussion

Direct current cardioversion for atrial arrhythmias occasionally causes pulmonary edema; however, it is rare in patients without organic heart disease, as in the present patient.^{1,2} Upshaw reviewed the literature concerning cardioversion of atrial arrhythmias and reported that post-cardioversion lung edema occurred in 14 of 1,130 patients (1.2%).¹ Among these 14 patients, only one exhibited atrial fibrillation alone. The remaining patients had organic heart disease or a history of hypertension.

The mechanism of this complication is not well-known. Some investigators consider that cardioversion induces cardiac dysfunction, which causes cardiogenic pulmonary edema;^{5,6} whereas others suggest that direct lung injury causes noncardiogenic pulmonary edema by increasing the permeability of the pulmonary capillary.⁷ To differentiate these conditions, the evaluation of the left atrial (LA) pressure is necessary. Although it is difficult to precisely estimate LA pressure by echocardiography, IVRT is a useful index that inversely correlates with LA pressure.⁸ In general, aging and bradycardia prolong IVRT.^{8,9} Mantero et al investigated the LV diastolic parameters in 288 healthy subjects and their value of IVRT was 77.3 ± 12.8 ms (mean $\pm$ SD) in those aged 50–59 years.⁹ At the onset of lung edema in the present patient, echocardiography detected a shortening of the IVRT from 65 to 36 ms despite a lower heart rate compared with before cardioversion (Fig 2A). This shortened value is considered to reflect a marked increase of LA pressure after cardioversion.^{8,9} In the clinical course, intravenous nitroglycerine and furosemide gave rapid relief of dyspnea. These findings indicate that the pulmonary edema in the present patient was congestive edema due to increased LA pressure, and some kind of cardiac dysfunction was the likely cause.

A thromboembolism of the coronary artery¹⁰ was unlikely because no intraatrial thrombi were noted before cardioversion by transesophageal echocardiography and LV asynergy was not detected at the onset of lung edema. Delayed recovery of LA contraction compared with the right atrium may cause cardiogenic lung edema.¹ Mayosi et al were unable to detect LA contraction with Doppler echocardiography despite a recovered right atrium in their patient with postcardioversion lung edema.¹¹ In the present patient, this mechanism was not the main cause because Doppler echocardiography revealed similar transmitral flow velocity in late diastole to right ventricular inflow.

Transient LV damage by direct current (DC) shock has been considered to cause postcardioversion lung edema.^{5,6,12} Experimental studies showed that thermal stress by electrical discharge induced microscopic cardiac damage, such as myocardial interstitial edema and infiltration of granulocytes, especially after excessive doses of electrical energy.^{5,13} In addition to the morphologic derangement, some investigators observed deterioration of LV contractility,^{12,14} but others have reported no alteration.¹⁵ We detected a small depression in LVEF after the electrical discharge compared with the LVEF before cardioversion. However, a LVEF of 65% was not likely to cause lung edema independently without any other causative factors. Yasuda et al examined the LV pressure–volume relationship in a canine model before and after direct current countershock.¹⁶ They reported that electrical discharge elevated LV end-diastolic pressure by impairing LV relaxation despite no significant impairment of LV contractility. In their study, the LV dias-

tolic pressure did not change 3 min after DC shocks, but increased progressively 10–15 min after the DC shock with a marked prolongation of the time constant of LV pressure decay, the invasive index of LV relaxation. In contrast, no significant change was observed in the slope of the LV end-systolic pressure–volume relationship, the index of LV contractility. Their evaluation of the water content of the ventricular myocardium indicated that myocardial interstitial edema was a likely potential cause of the LV diastolic dysfunction after DC shocks. To investigate whether or not such a mechanism accounted for the pulmonary edema in the present patient, clinical assessment of the LV diastolic function was necessary. Pulsed wave Doppler measurement of transmitral flow is the usual noninvasive method of assessment of LV diastolic function. It is, however, not reliable in the decompensated state with insufficient recovery of atrial function. The Vp during LV early filling was proposed as the index reflecting LV diastolic function with the least influence of the loading condition.^{4,17} This index seems to be useful also in the condition of insufficient atrial recovery because the value reflects LV early relaxation independent of atrial kick in late diastole. Takatsuji et al investigated LV function in subjects with and without organic heart disease, such as an old myocardial infarction and dilated cardiomyopathy, by means of echocardiography and cardiac catheterization,⁴ and showed that the Vp correlated with the invasive variables of LV relaxation. The value of Vp was 74.3 ± 17.4 cm/s (mean $\pm$ SD) in subjects without significant cardiac disease. In the present patient, the Vp was 36.5 cm/s at the onset of lung edema (Fig 2B), which is relatively low, indicating impaired LV early relaxation.⁴ The Vp improved to 65 cm/s the next day. Consequently, direct current shock probably induced transient LV diastolic dysfunction in the present patient and is the likely cause of the lung edema.

Transthoracic impedance (TTI) is an important factor that determines the actual energy dose through the chest during countershock.¹⁸ The transmural current is inversely proportional to the TTI.¹⁹ Some investigators have studied the influence of the phase of ventilation on the TTI and defibrillation success in animal models,^{20,21} which revealed a significantly higher TTI during inspiration, and a significant decrease in defibrillation success rate when shocks were delivered during inspiration, compared with expiration. Deakin et al investigated the influence of lung volume on the TTI by means of a continuous positive airways pressure circuit.²² In 12 healthy subjects, increased lung volume using positive end-expiratory pressure increased the TTI significantly. These findings indicate that lung volume reduces the transthoracic current during electrical discharge. The present patient had undergone left upper lobectomy 3 years before, and according to the chest X-ray, the left lung volume surrounding the heart had diminished (Fig 1A). This decreased lung barrier may have resulted in the excessive transmural current causing the cardiogenic lung edema.

However, some limitations should be considered. We used echocardiographic findings, such as IVRT and Vp, to evaluate the hemodynamic state and cardiac function. A large number of studies support the validity of these noninvasive indices,^{4,8,17} but they are not fully standardized because several factors influence these echocardiographic findings.^{4,8,9} Invasive studies, such as cardiac catheterization, may have confirmed the mechanism of the postcardioversion lung edema more definitely, but they were not performed on this patient.

References

1. Upshaw CB Jr: Hemodynamic changes after cardioversion of chronic atrial fibrillation. *Arch Intern Med* 1997; **157**: 1070–1076
2. Budow J, Natarajan P, Kroop IG: Pulmonary edema following direct current cardioversion for atrial arrhythmias. *JAMA* 1971; **218**: 1803–1805
3. Teichholz LE, Kreulen T, Herman MV, Gorlin R: Problems in echocardiographic volume determinations: echocardiographic–angiographic correlations in the presence or absence of asynergy. *Am J Cardiol* 1976; **37**: 7–11
4. Takatsuji H, Mikami T, Urasawa K, Teranishi J, Onozuka H, Takagi C, et al: A new approach for evaluation of left ventricular diastolic function: Spatial and temporal analysis of left ventricular filling flow propagation by color M-mode Doppler echocardiography. *J Am Coll Cardiol* 1996; **27**: 365–371
5. Van Vleet JF, Tacker WA Jr, Geddes LA, Ferrans VJ: Acute cardiac damage in dogs given multiple transthoracic shocks with a trapezoidal wave-form defibrillator. *Am J Vet Res* 1977; **38**: 617–626
6. Paloheimo JA: Pulmonary edema after defibrillation. *Lancet* 1965; **2**: 439
7. Gomaa M, Dayem MKA, Eissa A, Ateyya M: Pulmonary edema after direct current countershock. *Chest* 1972; **62**: 623–625
8. Appleton CP, Hatle LK, Popp RL: Relation of transmitral flow velocity patterns to left ventricular diastolic function: new insights from a combined hemodynamic and Doppler echocardiographic study. *J Am Coll Cardiol* 1988; **12**: 426–440
9. Mantero A, Gentile F, Gualtierotti C, Azzollini M, Barbier P, Beretta L, et al: Left ventricular diastolic parameters in 288 normal subjects from 20 to 80 years old. *Eur Heart J* 1995; **16**: 94–105
10. Turner JRB, Towers JRH: Complication of cardioversion. *Lancet* 1965; **2**: 612–614
11. Mayosi BM, Commerford PJ: Pulmonary edema following electrical cardioversion of atrial fibrillation. *Chest* 1996; **109**: 278–280
12. Kerber RE, Martins JB, Gascho JA, Marcus ML, Grayzel J: Effect of direct-current countershocks on regional myocardial contractility and perfusion. *Circulation* 1981; **63**: 323–332
13. Tedeschi CG, White CW: A morphologic study of canine hearts subjected to fibrillation, electrical defibrillation and manual compression. *Circulation* 1954; **9**: 916–921
14. Fuenmayor AJ, Vidal E, Fuenmayor AM: The effects of cardioversion and defibrillation on left ventricular systolic function. *Jpn Circ J* 1992; **56**: 822–828
15. Koning G, Veefkind AH, Schneider H: Cardiac damage caused by direct application of defibrillator shocks to isolated Langendorff-perfused rabbit heart. *Am Heart J* 1980; **100**: 473–482
16. Yasuda S, Shishido T, Goto Y: Severe diastolic dysfunction with preserved energy conversion efficiency after countershock. *Am J Physiol* 1997; **273**: H583–H592
17. Garcia MJ, Palac RT, Malenka DJ, Terrell P, Plehn JF: Color M-mode Doppler flow propagation velocity is a relatively preload-independent index of left ventricular filling. *J Am Soc Echocardiogr* 1999; **12**: 129–137
18. Kerber RE, Kouba C, Martins J, Kelly K, Low R, Hoyt R, et al: Advance prediction of transthoracic impedance in human defibrillation and cardioversion: importance of impedance in determining the success of low-energy shocks. *Circulation* 1984; **70**: 303–308
19. Jones VC, Charbonnier FM, Long P: Determining transthoracic impedance, delivered energy, and peak current during defibrillation episodes. *Medical Instrumentation* 1981; **15**: 380–382
20. Ewy GA, Hellman DA, McClung S, Taren D: Influence of ventilation phase on transthoracic impedance and defibrillation effectiveness. *Crit Care Med* 1980; **8**: 164–166
21. Sirna S, Ferguson D, Charbonnier F, Kerber R: Factors affecting transthoracic impedance during electrical cardioversion. *Am J Cardiol* 1988; **62**: 1048–1052
22. Deakin CD, McLaren RM, Petley GW, Clewlow F, Dalrymple-Hay MJ: Effects of positive end-expiratory pressure on transthoracic impedance: implications for defibrillation. *Resuscitation* 1998; **37**: 9–12