Acute Pulmonary Edema Associated With Direct Current Cardioversion in a Structurally Normal Heart

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The transthoracic application of synchronized direct current cardioversion (DCC) is widely used to terminate atrial fibrillation, atrial flutter, and other supraventricular tachyarrhythmia. DCC is a highly effective method for acute restoration of sinus rhythm. Although DCC is a relatively safe and frequently performed procedure, data on potential side effects are very rarely reported in the literature. The most serious complications associated with DCC are thromboembolism and intracranial hemorrhage. The true incidence of postcardioversion pulmonary edema is not known, but it is estimated to occur in 1% to 3% of patients, particularly those with coexistent heart disease. We report on a patient with a structurally normal heart who developed acute pulmonary edema after undergoing DCC. The patient had no evidence of myocardial injury according to an electrocardiogram and cardiac biomarkers. The patient was treated with intravenous diuretics. After 4 days, the pulmonary edema resolved.
[Rev Cardiovasc Med. *2008;9(2):137-141*]

Key words: Direct current cardioversion • Supraventricular tachyarrhythmia • Sinus rhythm • Acute pulmonary edema

The patient was a 62-year-old man who had experienced persistent atrial fibrillation with symptoms of palpitation and fatigue for 7 weeks. He presented for elective direct current cardioversion after 1 month of therapy to achieve an international normalized ratio greater than 2. He had a history of hypertension, dyslipidemia, and type II diabetes mellitus. The preprocedure electrocardiogram (ECG) exhibited atrial fibrillation with rapid ventricular response and a heart rate of 154 beats/min (Figure 1). The patient had no history of coronary artery disease, congestive heart failure, rheumatic heart

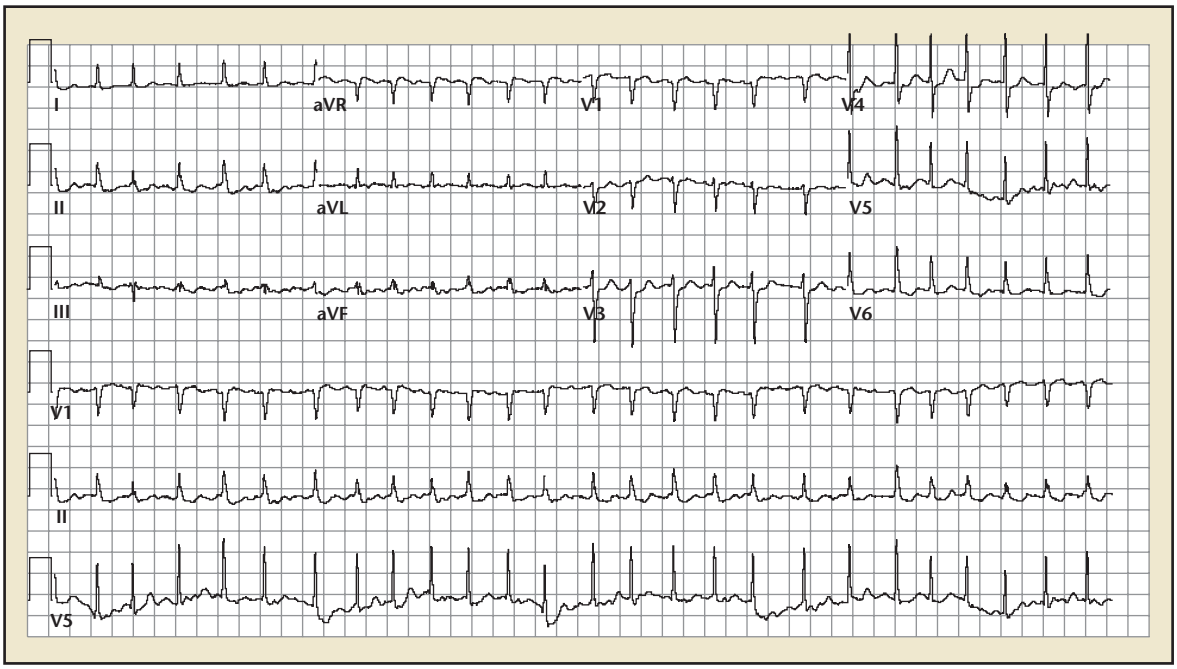

Figure 1. *The preprocedure electrocardiogram showed atrial fibrillation with a rapid ventricular response rate of 154 beats/min.*
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disease, thyroid dysfunction, or transient ischemic attacks or strokes. A recent 2-dimensional echocardiogram (2-D ECHO) revealed mild concentric hypertrophy, a left ventricular ejection fraction (LVEF) of 60% to 65%, a left atrial dimension of 4.5 cm, and Doppler evidence of diastolic dysfunction.

For the direct current cardioversion (DCC) procedure, an anesthesiologist administered propofol and fentanyl. The paddles were placed in anterior and posterior positions for the delivery of the current. The patient was successfully converted to normal sinus rhythm by 100 joules of biphasic current.

The patient developed respiratory distress and diaphoresis 30 minutes after the DCC. On examination, blood pressure was 170/90 mm Hg, respiratory rate was 38 breaths/min, and pulse was 120 beats/min. Diffuse rales were noted during auscultation of the lungs. An apical systolic murmur and a fourth heart sound were heard on cardiac auscultation. Chest x-ray was consistent with new pulmonary edema (Figure 2). Sinus tachycardia was recorded on an ECG (Figure 3).

The patient was admitted to cardiac intensive care and treated with oxygen and intravenous diuretics. Serial cardiac biomarkers (creatine phosphokinase and troponin T) were

Figure 2. *After the direct current cardioversion procedure, the patient developed acute pulmonary edema.*
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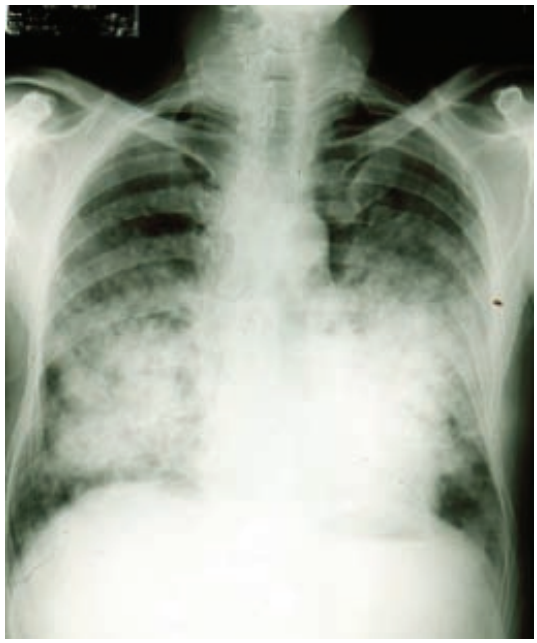

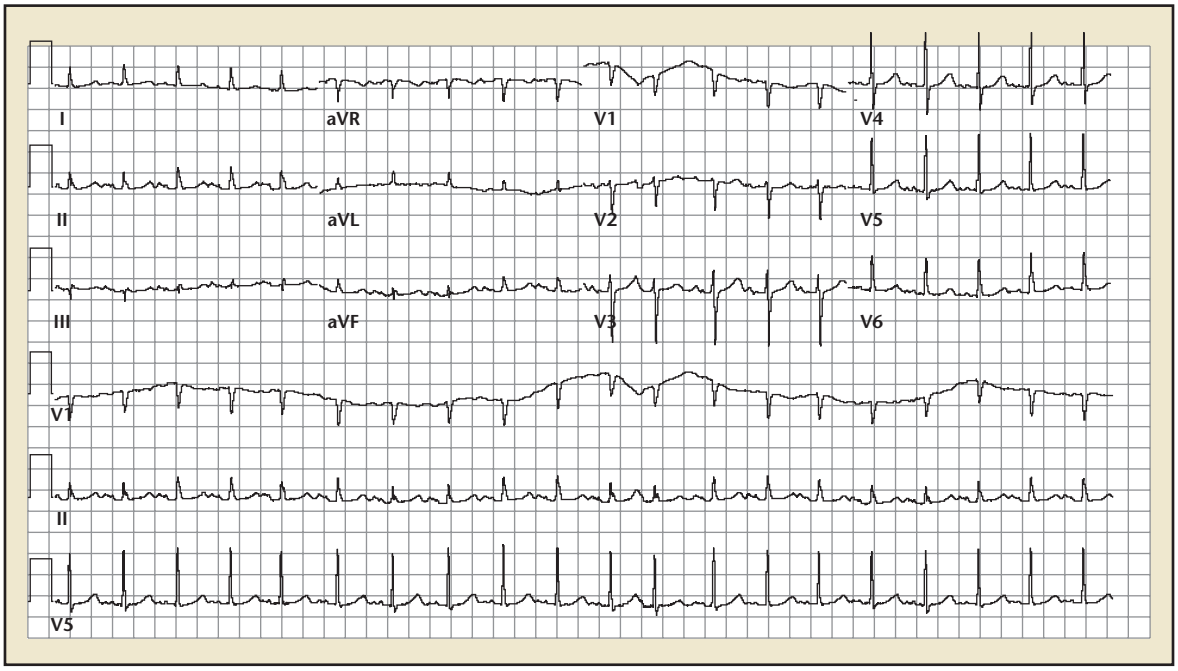

Figure 3. *An electrocardiogram showed sinus tachycardia, with a heart rate of 120 beats/min.* www.medreviews.com

negative. The B-type natriuretic peptide (BNP) level was elevated to 300 pg/mL. (The normal value is < 100 pg/mL.) A repeat 2-D ECHO performed mainly to assess LVEF and wall motion abnormalities was unremarkable when compared with the baseline.

The patient did well with intravenous diuretics. BNP trended to a normal value. After 4 days in the hospital, the patient was discharged home in a clinically stable state. A radiograph showed resolution of the pulmonary edema.

Discussion

In the United States, alternating defibrillation was first employed by Zoll and colleagues[1] in 1956. Lown and coworkers[2] introduced DCC in the early 1960s. Cardioversion of various arrhythmias is often performed to restore sinus rhythm and is usually followed by an improvement in symptoms and hemodynamics. DCC can be safely and effectively performed on an ambulatory basis. The risks of cardioversion are mainly related to the embolic events. There are no significant differences in the safety and complications associated with outpatient versus inpatient DCC.[3] DCC can be safely performed if the rhythm is synchronized with the peak of the R wave. This approach will avoid the delivery of shock during the vulnerable period of repolarization—around the peak of the T wave—which can result in ventricular fibrillation and cardiac arrest. It is particularly important to check in other connected leads for artifacts when unusual QRS-T complexes are present to prevent inadvertent delivery of shock around the peak of the T wave on ECG.

The most serious complications associated with DCC are thromboembolism and intracranial hemorrhage. The incidence of thromboembolic complications after DCC that occur because of atrial stunning is up to 7% in patients with atrial fibrillation who have not received prior anticoagulation treatment.[4] Less serious adverse effects that leave no permanent sequelae include skin burns due to paddle misplacement, reversible muscle pain, subcutaneous hematoma due to warfarin, and slow recovery from anesthesia.[5]

Acute pulmonary edema is an uncommon complication associated with DCC. The true incidence of postcardioversion pulmonary edema is not known, but it is estimated to occur in 1% to 3% of patients, particularly those with coexistent heart disease. Rates of pulmonary edema do not correspond with the energy used for cardioversion. Pulmonary edema can occur immediately after cardioconversion or up to 96 hours after.

In a case series of 712 patients with chronic atrial fibrillation, a significant number of patients developed reduced cardiac output after cardioversion, but this outcome rarely led to acute pulmonary edema.[6] The mechanism for the development of

pulmonary edema after cardioversion is not well known. Patients who remain in sinus rhythm after cardioversion of atrial fibrillation and flutter experience a gradual, significant increase in cardiac output accompanied by an increase in the strength of the left atrial contraction. Moreover, as the heart rate slows down after cardioversion of tachyarrhythmias, the left ventricular end-diastolic pressure rises, increasing left ventricular fiber length and left ventricle contraction according to the Frank-Starling principle.

In most patients, right atrial contractile activity returns faster than left atrial activity, which could be a possible contributing factor in the genesis of pulmonary edema after cardioversion of atrial fibrillation and flutter. A proposed hypothesis for this phenomenon is that the left atrium, being more muscular, assumes more of the burden of the chronic changes and has a relatively slow return of the contractile activity upon resumption of sinus rhythm. The right atrium is less stunned and resumes its normal mechanical activity earlier as compared with the left atrium. This differential atrial recovery related to atrial myopathy results in slow recovery of the left atrial contractility upon resumption of sinus rhythm. Also because of the lower pressure volume overload, the structural alterations in the right atrium are less marked as compared with the left atrium.

The earlier normalization of right atrial mechanical activity before the left atrium may be a factor in the pathogenesis of pulmonary edema after successful DCC. The other possible factors contributing to the occurrence of acute pulmonary edema after cardioversion include transient left ventricular diastolic dysfunction, idiosyncratic damage of the myocardium from the direct current shock, direct lung injury causing noncardiogenic pulmonary edema, reduction in cardiac performance caused by a combination of heart disease and the cardiac depressant effects of the anesthetic drugs, and pulmonary and/or coronary microemboli.[7] In addition, after DCC, the differential atrial recovery causes a preferential increase in blood flow to the lungs relative to the systemic circulation. This disruption can upset the hemodynamic balance, especially in patients who are marginally compensated before cardioversion.

The causes of acute pulmonary edema in our patient, who had no structural heart disease, are uncertain. It seems unlikely that the cause was noncardiogenic. Propofol and fentanyl, which were used for anesthesia, have not been linked to acute pulmonary edema or cardiac depressant adverse effects. Negative pressure pulmonary edema, which is commonly seen as an acute complication after relief of complete or partial upper airway obstruction, was excluded. The elevated BNP level of 300 pg/mL clearly supports a cardiac etiology as the cause of the acute respiratory distress (which can occur with levels $>$ 100 pg/mL).[8] A BNP level greater than 500 pg/mL indicates that heart failure is the likely cause of acute pulmonary edema occurring with systolic dysfunction.[9] We hypothesize that the moderately elevated BNP level of 300 pg/mL in our patient was probably related to low-pressure, high-volume stress on the right ventricle due to delayed left atrial recovery. In our own experience, BNP levels must be interpreted with caution. Right ventricular strain will increase BNP levels less than left ventricular strain will. We commonly observe BNP levels exceeding 600 pg/mL in patients with cardiomyopathy, LVEF less than 30%, and left bundle branch block in the

Main Points

- Direct current cardioversion (DCC) of various arrhythmias is often performed to restore sinus rhythm and is usually followed by an improvement in symptoms and hemodynamics.
- DCC can be safely performed if the rhythm is synchronized with the peak of the R wave. This approach will avoid the delivery of shock during the vulnerable period of repolarization—around the peak of the T wave—which can result in ventricular fibrillation and cardiac arrest.
- The incidence of thromboembolic complications after DCC that occur because of atrial stunning is up to 7% in patients with atrial fibrillation who have not received prior anticoagulation treatment.
- In a case series of 712 patients with chronic atrial fibrillation, a significant number of patients developed reduced cardiac output after cardioversion, but this outcome rarely led to acute pulmonary edema.
- The true incidence of postcardioversion pulmonary edema is not known, but it is estimated to occur in 1% to 3% of patients. Although the condition is more common in patients with coexistent heart disease, it can arise in patients with a structurally normal heart.

absence of clinical heart failure symptoms. In our patient, a limited 2-D ECHO obtained the day after admission revealed preserved left ventricular systolic function without wall motion abnormalities. A 2-D ECHO focusing on atrial kinetics, such as Doppler recording of simultaneous tricuspid and mitral annular inflow pattern, simultaneous tissue Doppler of mitral and tricuspid valve, and Doppler recording of pulmonary inflow pattern, could have been useful in confirming our hypothesis of atrial mechanical dyssynchrony but was not obtained.

Conclusion

Synchronized DCC can be safely and effectively performed on an ambulatory basis. The Association for the Advancement of Medical Instrumentation has published standards for cardiac monitoring.[10] For DCC, the guidelines note the necessity of safely detecting and distinguishing between QRS complexes and T waves to avoid shock during the vulnerable period of repolarization. DCC is a relatively safe and frequently performed procedure. Acute pulmonary edema is an uncommon complication associated with DCC. Even patients with a structurally normal heart can develop acute pulmonary edema after undergoing DCC. ■

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