Refractory Hypotension after Cardioversion in a Patient with Atrial Fibrillation and Congestive Heart Failure

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Abstract

A 63-year-old woman with atrial fibrillation visited our hospital due to decompensated heart failure. Because atrial fibrillation was considered a remarkable precipitating factor for heart failure, cardioversion was performed. After cardioversion, refractory hypotension and cardiopulmonary arrest occurred. An arterial blood gas analysis showed marked lactic acidosis. Chronic kidney disease, heart failure, sedatives, and hypoventilation might have contributed to refractory hypotension due to severe acidosis in this case.

Key words: refractory hypotension, cardioversion, acidosis


Introduction

Atrial fibrillation and congestive heart failure are commonly observed medical conditions that are closely related to one another (1). In the case of patients with congestive heart failure in whom the onset of atrial fibrillation is associated with severe hemodynamic deterioration, an initial attempt for the maintenance of the sinus rhythm is required (2). Cardioversion for atrial fibrillation has long been considered to be a safe and efficient option (3). We herein experienced a rare case of refractory hypotension and cardiopulmonary arrest (CPA) that occurred after cardioversion.

Case Report

A 63-year-old woman who had been treated for hypertension and chronic kidney disease (CKD) felt dyspnea for 2 days after cleaning out her shop and was referred to our hospital for the first episode of atrial fibrillation and heart failure. A physical examination showed that her blood pressure was 125/105 mmHg with a pulse rate of 103/min. Auscultation showed normal respiratory and heart sounds. The results of an examination of her blood sample showed mild liver injury [aspartate aminotransferase (AST) level 44 IU/L, alanine aminotransferase (ALT) level 32 IU/L], CKD (serum creatinine level) 1.47 mg/dL, and estimated glomerular filtration rate (eGFR) 28.6 mL·min⁻¹·1.73 m²⁻¹, and a high plasma brain natriuretic peptide (BNP) level (441.2 pg/mL). The thyroid function was normal. A chest radiograph showed a cardiothoracic ratio of 60% with mild pulmonary congestion. A 12-lead electrocardiogram (ECG) showed atrial fibrillation with a heart rate of 117 beats/min; a high R-wave in the left precordial leads; and ST segment depression in leads II, III, aVF, and V6 (Fig. 1). Transthoracic echocardiography showed left atrial (LA) dilation (43 mm) without obvious LA thrombus. The thicknesses of the interventricular septum and LV posterior wall were 13 mm and 10 mm, respectively. The LV systolic function was preserved with an ejection fraction (EF) of 56% (Fig. 2). Color flow Doppler echocardiography showed moderate mitral regurgitation (MR) without any findings of rheumatic heart disease, prolapse, or endocarditis (Fig. 3). Dilation of the mitral annulus due to congestive heart failure was considered to be the cause of MR. Continuous Doppler echocardiography showed that the estimated systolic pressure gradi-
ent at the tricuspid valve was 47 mmHg, suggesting mild pulmonary hypertension. Pulsed Doppler echocardiography at the mitral inflow showed short deceleration time of E wave (4) and the presence of mid-diastolic flow (5), suggesting LV diastolic dysfunction. The patient was admitted to our hospital and was treated for heart failure using diuretic and anticoagulant drugs. After the treatment, her symptoms were alleviated, and her plasma BNP level decreased.

Figure 1. A 12-lead electrocardiogram showing atrial fibrillation with a heart rate of 117 beats/min. A high R-wave in the left precordial leads and ST segment depression in leads II, III, aVF, and V6 were also observed.

Figure 2. A transthoracic M-mode echocardiogram showing a left ventricular (LV) ejection fraction of 56% (upper panel). A pulsed Doppler echocardiogram showing short deceleration time of E wave (125 ms) and additional mid-diastolic L wave (arrows) (lower panel).
Figure 3. A color flow Doppler echocardiogram showing moderate mitral regurgitation (arrow) (left panel). A chest radiograph before cardioversion showing a cardiothoracic ratio of 59% with mild pulmonary congestion (right panel).

Figure 4. The potential mechanism underlying refractory hypotension followed by cardiopulmonary arrest in this case. See details in the text.

to 182.5 pg/mL. She refused cardioversion for atrial fibrillation and was discharged. However, she visited our hospital 2 weeks later with a complaint of dyspnea and agreed to undergo cardioversion. Her plasma BNP level was approximately 2-fold the level at her initial visit (816.4 pg/mL). Her chest radiograph showed a cardiothoracic ratio of 59% with mild pulmonary congestion (Fig. 4). Because atrial fibrillation was considered a remarkable precipitating factor for heart failure, cardioversion was performed. After the intravenous administration of 150 mg of thiamylal sodium and cardioversion with 50 J, ECG monitoring showed restoration of her sinus rhythm.

Although mask-bag ventilation was performed, the pulse oximeter saturation level transiently decreased to 60%, potentially due to deep sedation and congestive heart failure. After the recovery of her consciousness, the patient complained of dyspnea. Her blood pressure was 71/49 mmHg, and maneuvers such as leg elevation, drip infusion, atropine sulfate injection, and dopamine drip infusion were ineffective. Eventually, her pulse was not palpable, and she lost consciousness. ECG monitoring showed asystole. Cardiopulmonary resuscitation was initiated with an intravenous administration of adrenaline, noradrenaline, and dopamine. She was intubated and artificially ventilated. Cardioversion was performed once for ventricular tachycardia. Emergent echocardiography during cardiac massage showed a left ventricu-
lar ejection fraction (LVEF) of 47%. The results of her arterial blood gas analysis showed marked acidosis due to respiratory and lactic acidosis (partial pressure of arterial oxygen (PaO₂), 445.0 mmHg; partial pressure of carbon dioxide in arterial blood (PaCO₂), 51.9 mmHg; standard base excess, -15.9 mmol/L; pH, 7.026; anion gap, 13.1 mEq/L; lactate level, 75 mg/dL; and HCO₃ level, 12.9 mmol/L) (6). The patient was later administered sodium bicarbonate. Because the hemodynamic status remained unstable, she was transferred to an emergency hospital for intensive care. Chest radiography showed pulmonary congestion and transthoracic echocardiography showed a decreased LVEF of 32%. She was under mechanical ventilation for one day. Then, her condition gradually improved and transthoracic echocardiography a week later showed a fairly improved LVEF of 37%. A rest thallium scintigram showed no LV perfusion defect with diffuse LV hypokinesis (EF 37%). Although the patient’s ribs were fractured and ECG showed atrial fibrillation, she was discharged without any remarkable complications.

In the present case, although atrial fibrillation appeared to have worsened the patient’s heart failure, refractory hypotension followed by CPA occurred after cardioversion. Although cardioversion for atrial fibrillation is generally safe, asystole may occur, albeit rarely, as a complication. Grönberg et al. reported that asystole after bradycardia was observed in 9 cases out of 7,660 cardioversions and was related to unsuccessful cardioversion (7).

In the present case, however, cardioversion was transiently successful. We suspected some pathophysiologic mechanisms underlying these events (Fig. 4).

Some studies have shown that sedation by propofol administration may cause lactic acidosis and cardiogenic shock (8), known as propofol infusion syndrome. Because the mechanism of the action of thiamylal is similar to that of propofol (9), thiamylal might have contributed to lactic acidosis. Other factors might also have contributed to hypotension and acidosis. Sedation decreases the blood pressure. Thus, sedation in our case might have caused tissue hypoxia and lactic acidosis. Cardiac ischemia might have worsened LV dysfunction and hypotension. Renal ischemia might have worsened CKD and decreased urinary acid secretion. Sedation, in addition to potentially causing hypoxia, might have promoted hypercapnia caused by hypoventilation. Hypoxia might have worsened tissue ischemia, and hypercapnia might have caused respiratory acidosis. Severe acidosis then may have caused refractory hypotension which thus led to a vicious cycle, eventually causing CPA. This case suggests that some caution must be exercised when cardioversion is indicated to avoid serious complications in patients with atrial fibrillation. Patients with decompensated heart failure should be admitted to a hospital and be strictly treated for heart failure before cardioversion. The sedative should be carefully selected and small doses of the drug should be used. Bag-mask ventilation should be performed in case apnea occurs. The heart rate and blood pressure should be monitored, and appropriate measures should be immediately taken whenever necessary. Moreover, McMullan et al. reported a case of prolonged asystole after cardioversion for atrial flutter, stressing the need for managing complications after cardioversion (10).

In conclusion, we experienced a case of refractory hypotension and cardiopulmonary arrest after cardioversion. Advanced treatment of accompanying diseases, selection of the proper sedative, maintenance of sufficient ventilation, and monitoring of vital signs are important in the prevention of adverse events during cardioversion.

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References