Percutaneous Cardiopulmonary Support Aids Resuscitation From Sustained Ventricular Tachycardia

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A 67-year-old man was transferred to hospital because of acute circulatory failure resulting from sustained left ventricular tachycardia (LVT) and dysfunction. Transthoracic echocardiography revealed severely impaired left ventricular contraction and dyskinesis of the apical wall. Neither anti-arrhythmic agents nor direct current cardioversion was effective; the patient was resuscitated by immediate use of percutaneous cardiopulmonary support and intraaortic balloon counterpulsation. Ventricular contraction returned to normal following restoration of normal sinus rhythm with amiodarone and cibenzoline. The pathogenesis of LVT accompanied by transient ventricular dyskinesis is discussed with regard to the efficient use of a mechanical circulatory support system in resuscitation. (Circ J 2003; 67: 1061–1063)

Key Words: Cardiogenic shock; Percutaneous cardiopulmonary support; Ventricular tachycardia

The use of percutaneous cardiopulmonary support (PCPS) with intraaortic balloon pump (IABP) counterpulsation is an effective treatment for acute circulatory failure. We present a rare case of cardiogenic shock caused by sustained left ventricular tachycardia (LVT) and left ventricular apical dyskinesis, which was treated successfully with mechanical circulatory support system. It is sometimes difficult to uncover the immediate causes of life-threatening arrhythmia or LV dysfunction and using a mechanical circulatory support system can provide primary physicians with adequate time to successfully diagnose and treat reversible causes of cardiogenic shock.

Case Report

A 67-year-old man was transferred to the intensive care unit (ICU) because of sustained LVT and cardiogenic shock. He had been well until the day of admission when he felt sudden intermittent chest discomfort and nausea. He had a history of hypertension and had been treated with nifedipine and diltiazem for 5 years. There were no symptoms attributable to infectious disease nor a personal or family history of cardiac disease.

On arrival at the primary hospital, his blood pressure was 60/23 mmHg and his heart rate was 260 beats/min. Electrocardiography showed sustained monomorphic LVT with a QRS pattern of right-bundle branch block and a superior axis (Fig 1A, B). Medical termination was attempted initially because he was drowsy. The LVT did not respond to intravenous verapamil (10 mg) and lidocaine (100 mg), so direct current cardioversion was performed; however, the LVT continued. Cardioversion was attempted several times after the intravenous administration of each of propranolol (2 mg), disopyramide (50 mg) and procainamide (600 mg). Brief episodes of asystole occurred, followed by LVT. Temporary right ventricular pacing was initiated. Coronary angiography showed normal coronary arteries. The patient had to undergo cardiopulmonary resuscitation during transfer to the ICU at our hospital.

Upon arrival in the ICU, the patient’s cardiac rhythm alternated between LVT and cardiac standstill with ventricular pacing. His lungs were clear and no murmur or extra heart sounds were heard. Chest X-ray was normal. Two-dimensional echocardiography showed diffuse left ventricular (LV) hypokinesis and dyskinesis of the apical wall (Fig 2). His white blood cell count was 17,910/mm³, serum creatine kinase was 410 IU/L, magnesium was 2.2 mg/dl, and C-reactive protein was 0.07 mg/dl.

PCPS (CAPIOX SX custom pack, Terumo, Tokyo, Japan) was begun immediately through the right femoral artery and vein with extracorporal circulatory support at 3.0 L/min. Under circulatory support, we performed a right ventricular endomyocardial biopsy and found normal myocardium. The IABP was subsequently inserted through the left femoral artery and was set to 1:1 assistance. Anticoagulation therapy was intravenous administration of heparin to achieve an activated coagulation time of 200s. After these procedures, the patient’s hemodynamic status had improved: blood pressure increased to 97/50 mmHg, he became alert, and there was a substantial increase in urinary volume. However, repeat administration of verapamil and ventricular overdrive pacing had no effect on the LVT.

On the second hospital day, sotalol was given orally at 80 mg/day, but the cardiac rhythm still alternated between sinus rhythm and LVT. In addition to the sotalol, continuous intravenous infusion of cibenzoline at a dose of 350 mg/day was begun. The LVT was finally terminated successfully on day 3. The PCPS was weaned to 2.5 L/min on day 2 and then 1.0 L/min on day 3. On day 4, we were
able to stop the PCPS flow and decrease IABP support to 2:1 assistance, then 4:1, and then cease it in accordance with the increased cardiac output.

One week after admission, amiodarone was substituted for sotalol because of occasional bouts of non-sustained LVT. Ultimately, the ventricular arrhythmia was abolished with both amiodarone at 200 mg/day and cibenzoline at 400 mg/day. Stable normal sinus rhythm was restored. 201Tl and 99mTc-pyrophosphate scintigraphy showed myocardial injury in the apical portion of the left ventricle. Repeated measurement of several viral antibody titers, such as Coxsackievirus, adenovirus, cytomegalovirus, echovirus, mumps and varicella, disclosed no evidence of recent viral infection. On day 28, the electrocardiography findings were normal except for negative T waves in the precordial leads (Fig 1C). The diffuse LV hypokinesis and apical dyskinesis normalized after the ventricular arrhythmia was abolished. A follow-up echocardiogram performed 4 weeks after admission showed an ameliorated left ventricular ejection fraction of 67% (Fig 3). Five weeks later, LVT could not be induced by programmed electrical stimulation and the patient was discharged from hospital.

Six months later, the patient was quite well with no recurrence of LVT, so we ceased the administration of amiodarone and only the cibenzoline (300 mg/day) was continued. He has been in good health with normal activity of daily life for the 2 years since his discharge.

**Discussion**

PCPS is an accepted therapeutic device for selected patients with cardiogenic shock accompanied by fulminant myocarditis; acute massive myocardial infarction or circu-
latory insufficiency after open-heart surgery. Alternate support from ventricular assist devices is necessary when cardiac function does not recover and powerful long-term assistance is needed. PCPS provides immediate life-sustaining circulatory and oxygenation support until diagnostic and therapeutic interventions can be initiated. Rapid deployment of such percutaneous devices is useful in the critical care setting, as described in the American Heart Association’s guidelines 2000 for cardiopulmonary resuscitation and emergency cardiovascular care; however, there has not been an adequate randomized clinical trial of the device. In the present case, immediate circulatory support was crucial to maintain the patient’s circulation and to secure enough time for treatment as well as diagnosis.

Generally, cardioversion, anti-arrhythmic drugs or ventricular overdrive pacing successfully terminates LVT in otherwise healthy patients. Despite having no previous cardiac abnormalities, the present patient had sustained LVT refractory to these treatments. Some types of LVT with right-bundle branch block pattern are verapamil sensitive, but because intravenous verapamil was not effective and the mechanism of the LVT was unclear, multiple antiarrhythmic drugs were administered on a trial-and-error basis. However, careful attention must be given to the pro-arrhythmic and negative inotropic effects of these agents, especially in patients with depressed LV function and fortunately there was no evidence of drug-induced heart failure or proarrhythmia in the present case.

The Sicilian Gambit Guideline recommends direct current cardioversion in patients suffering sustained LVT with circulatory failure; however, it was ineffective on several occasions in the present patient. The immediate use of PCPS enabled us to maintain an adequate hemodynamic status and gave us time to find effective drugs. The use of IABP with PCPS augmented coronary perfusion and reduced the left ventricular afterload. Myocardial ischemia caused by cardiogenic shock increases electrical irritability, which makes LVT more and more refractory. Thus, it is possible that this mechanical support ameliorated the myocardial ischemia and contributed to the termination of the LVT itself.

This case is characterized by acute circulatory failure caused by sustained LVT concomitant with a transient LV apical dyskinesia. The morphologic characteristics of the LVT indicated an origin from the apical portion of the left ventricle. We consider that a reversible myocardial dysfunction occurred there with arrhythmogenic change because both 201Tl and 99mTc-pyrophosphate scintigrams showed apical myocardial injury.

Reversible LV dysfunction has a wide spectrum of pathophysiologies: coronary vasospasm, coronary thromboembolism, myocardial stunning, neurohumoral abnormality, acute myocarditis, severe prolonged anemia, emotional stress, and tachycardia-induced cardiomyopathy. Tachycardia-induced cardiomyopathy could have caused the diffuse LV dysfunction in the present case; but could not account for the regional LV dyskinesia. The patient’s coronary arteries were normal, and none of the laboratory findings suggested myocardial damage; however, the endomyocardial biopsy specimens were taken only from the right ventricle. It may be that a focal acute myocarditis was present in the LV apical wall because the reversible clinical course followed that of typical acute myocarditis. Because the sustained LVT of this case was considered to be the result of an acute reversible myocardial injury, in accordance with ACC/AHA Practice Guidelines an implanted automatic cardio-defibrillator was not indicated. It is sometimes difficult to uncover the immediate cause of acute circulatory failure and we recommend the use of PCPS by the primary physician in the treatment of circulatory insufficiency caused by life-threatening LVT.

References