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Acute Pulmonary Edema During Cardiac Resynchronization Therapy Device Implantation: Management With Intraaortic Balloon Pump Insertion

Kardiyak Resenkronizasyon Tedavi Cihazı Takılması Sırasında Gelişen Akut Akciğer Ödeminin İntraaortik Balon Pompası İle Tedavisi

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In this case report, we present two patients who experienced acute pulmonary edema during cardiac resynchronization therapy device implantation. In both patients, insertion of an intraaortic balloon pump immediately improved clinical condition and the operation could be completed, which would otherwise had to be postponed.

Key Words: ***Acute pulmonary edema,cardiac resynchronization therapy,intraaortic balloon pump***

Bu vaka takdiminde, kardiyak resenkronizasyon tedavi cihazı takılması esnasında akut akciğer ödemi gelişen iki hastayı sunuyoruz. Her iki hastada da intraaortik balon pompası takılması klinik durumu hemen düzeltmiş ve operasyon tamamlanabilmiştir.

Anahtar Kelimeler: ***Akut akciğer ödemi,kardiyak resenkronizasyon tedavisi,intraaortik balon pompası***

Cardiac Resynchronization Therapy (CRT) is a safe and effective method for improving functional status, left ventricular (LV) function, quality of life, morbidity and mortality in a selected group of patients with New York Heart Association (NYHA) functional class III and IV heart failure despite optimal medical therapy (1, 2). Occasionally, the implantation procedure may be hampered by adverse clinical events (3) which might cause the procedure to be postponed. In this report, we describe 2 patients that experienced acute pulmonary edema during CRT device implantation in whom intraaortic balloon pump (IABP) insertion provided us to complete the procedure.

Case report

The clinical profiles of the 2 patients are summarized in the Table.

Case 1

Mrs EO, 50 years old hypertensive patient with idiopathic dilated cardiomyopathy (CMP) was admitted to our hospital with rescusitated cardiopulmonary arrest secondary to ventricular fibrillation. She had a history of multiple hospital admissions due to decompensated heart failure. She remained in NYHA class III despite optimal medical therapy. Her coronary angiogram was normal. In her ECG, a left bundle branch block (LBBB) was present. Echocardiography revealed LV systolic dysfunction and

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Table 1: Clinical and echocardiographic characteristics of the patients.

Case	Age, years	QRS, msec	LVEF, %	Septal-lateral wall delay, msec	Procedure time, min	Contrast dose, mL
1	50	180	11	68	135	140
2	63	120	20	100	170	150

an intraventricular dyssynchrony with Tissue Doppler Imaging (TDI). In the electrophysiologic study (EPS), no arrhythmia was induced. A biventricular implantable cardioverter-defibrillator (ICD) implantation was attempted in the EPS laboratory. During implantation of the LV lead, acute pulmonary edema developed. Her blood pressure was 100/80 mmHg and heart rate was 150 bpm. The operation was ended. Ten days later, a second implantation was attempted after the patient was stabilized. During the procedure, pulmonary edema developed again and an intraaortic balloon pump (IABP) (Datascope) was inserted through femoral artery and counter pulsation was started at 1:1 augmentation. A bolus of 5000 iu intravenous heparin was applied. In a few minutes the patient became comfortable. After the patient recovered in a short time, we decided to continue to the procedure. The LV lead could not be located due to the small size of the target vessel. The ICD lead and right atrial lead with a biventricular ICD generator (CONTAK RENEWAL 4 HE, Guidant Corp, St Paul, MN, USA) were implanted. There were no other procedure related complications. The IABP could be weaned off 3 hours after the procedure. The LV lead was implanted epicardially via thoracotomy in another day. The patient improved symptomatically and at 24 months follow-up she had no further hospitalization. The EF rised to 35% and her NYHA class decreased to II.

Case 2

Mrs AS, 63 years old hypertensive, diabetic patient was admitted with a diagnosis of ischemic CMP. The patient had a history of old myocardial infarction and sustained monomorphic ventricular tachycardia (SMVT) that was converted to sinus rhythm by amiodarone infusion and electrical cardioversion, respectively. She had multiple hospital admissions due to worsening heart failure in the previous 3 months. ECG showed a LBBB pattern. She had low ejection fraction and ventricular dyssynchrony evidenced by a significant intraventricular delay with TDI in the echocardiographic evaluation. In the EPS, SMVT was induced. Because she was in NYHA class III despite optimal medical therapy, a biventricular ICD implantation was indicated. During implantation, first, the LV lead was inserted. Then, during ICD lead implantation, acute pulmonary edema with hypotension developed. Because she did not recover with intravenous medication including inotropic support, an IABP (Datascope) was inserted through femoral artery, and counterpulsation with 1:1 augmentation was started. A bolus of 5000 iu intravenous heparin was applied. The patient recovered immediately, and she tolerated the procedure with no further complications. The LV lead was repositioned because it was dislocated during management of acute pulmonary edema. Then, right ventricular and atrial leads and the biventricular ICD generator (CONTAK RENEWAL 4 HE, Guidant Corp, St Paul, MN, USA) were implanted. The IABP was removed 4 hours after the procedure. During 12 months follow-up, the patient was readmitted to the hospital twice due to decompensated heart failure.

Discussion

The success rate of CRT implantation procedure is about 90%, however the operation may be hampered by some complications like coronary sinus dissection, cardiac tamponade, and acute patient decompensation (3). Up to our knowledge, the incidence of acute pulmonary edema during CRT implantation procedure is uncertain. In this case report, we presented two patients who experienced acute pulmonary edema that immediately recovered by insertion of an IABP, during CRT device implantation.

IABP has been proved beneficial in patients with cardiogenic shock with mechanical complications due to AMI. It may be used as a bridge to heart transplantation. IABP reduces afterload component of the cardiac work and reduces myocardial oxygen consumption, thus improves LV function and cardiac output (4).

There are some possible mechanisms to explain why acute pulmonary edema developed in our patients during operation. First, both of the patients had poor LV function, and lying in supine position during long lasting CRT implantation increases venous return to the heart. In healthy people, the heart respond to increased venous return with an augmented stroke volume (Frank-Starling relationship) (5). However, in patients with heart failure, stroke volume can not be further augmented due to exhaustion of Frank Starling reserve (6). Both of the patients had been tested if they could stay in supine position for 2 or 3 hours in the day before the procedure, so this mechanism can not solely explain the situation. Secondly, a considerable amount of contrast material was used for LV lead placement. Contrast media may have a depressant effect on LV function (7). Finally, anxiety related to the operation might have aggrevated the clinical condition. Anxiety is widely seen in patients with chronic heart failure (8), and may make the clinical scenario more complicated (9).

Both of the patients improved hemodynamically after IABP insertion and the procedures were completed. There were no other procedure related complications. Another choice would be postponing the procedure and implanting the CRT device in another session. However, reoperation poses the patient to additional risks associated with wound site and subclavian puncture.

In conclusion, IABP insertion may be a safe and beneficial option for completing the procedure in a small proportion of patients experiencing acute cardiac decompensation during CRT implantation. This approach may eliminate the need for reoperation and associated risks.

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