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<u>Case Report</u> A case of Symptomatic Severe Left Ventricular Dysfunction– Combo Device (CRT-D) Implantation

Authors

Dr Jyotsna .P, Dr Subramanyam, Dr Dilip Johny

Department of General Medicine and Cardiology, K.S. Hegde Medical Academy, Deralakatte, Mangalore *Corresponding Author

Dr Subramanyam

Abstract

More than 22 million people globally suffer from the rapidly worsening condition, congestive heart failure (CHF). In those with impaired left ventricular (LV) dysfunction, cardiac resynchronization therapy (CRT) is clinically effective in about 70% of the patients. CRT can improve exercise capacity and well-being of the patient, minimize re-hospitalization and potentially, lower mortality.

Introduction

Heart failure is the quickest growing cardiovascular diagnosis in the world, and it includes a poor prognosis. To enhance the survival in heart failure patient, treatment plans should focus on minimizing both sudden cardiac death (the most common reason of death in sufferers with New York Association (NYHA) Class I or II symptoms) or progressive heart failure (the predominant reason of loss of life in those with NYHA Class III or IV symptoms). Electrical conduction disturbances are common in coronary heart failure and are affiliated with elevated mortality Atrial- synchronized risk. biventricular pacing (cardiac resynchronization addresses many therapy [CRT]of the pathophysiological changes seen in patients with wide QRS complexes in whom delayed activation of the left free wall results in mechanical dyssynchrony.⁽¹⁾

Case Report

69 year old male, came with complaints of dyspnea on exertion (Class III NYHA) over a duration of 3 months and history of one episode of syncope. On examination, he was conscious, oriented and obeying commands. Vitals : Heart rate - 76/ min, blood pressure - 90/50mmHg, oxygen saturation - 98 % in room air, respiratory rate - 19/min, JVP - normal, No pedal odema Systemic examination of cardiovascular and respiratory system was normal. Hb-11.2g%, TC-6,800cells/mm3, DC-68% neutrophils, 32% lymphocytes, 6% eosinophils, platelet count -2.78 lakh cells/mm3. Urea - 34.4mg/dl, Creatinine -1.04 mg/dl, Sodium-135mEq/L, Potassium 4.22mEq/L,LFT- total protein – 7.2g/dl, Bilirubin (mg/dl) - 0.3 (total), 0.211 (direct), 0.19 (indirect), SGOT-35U/L, SGPT- 34U/L, ALP-81U/L, Albumin - 3.6 (g/dl), Globulin - 3.6 (g/dl). Urine routine- pH 6.0, protein - NIL, 1-2 epithelial

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cells, RBC- NIL, WBC- 2-4, No granular casts present.

2D ECHO - Severe LV dysfunction, LVEF -20%, Global hypokinesia of LV, Grade II LVdiastolic dysfunction, Grade II MR, Grade I-II dilated LA and LV, other cardiac TR. chamberdimensions normal, no clot/ vegetation/ pericardial effusion.CAG showed normal coronaries.ECG (on admission) - Left bundle branch block. Holter monitoring - 24 hour ambulatory Holter monitoring revealed baseline LBBB with maximum heart rate of 135bpm, and minimum heart rate of 62bpm, occasional VE's and SVE's were observed.

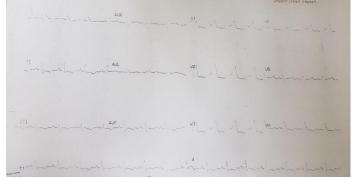
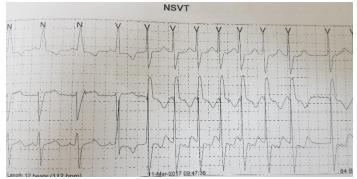
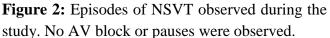


Figure 1: ECG (on admission) - LBBB, wide QRS complex v1 to v4





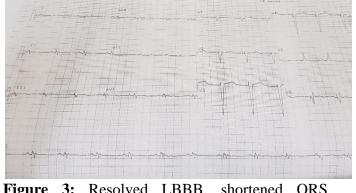


Figure 3: Resolved LBBB, shortened QRS complex duration v2 to v4

After one day of hospital stay, he was discharged on anti-platelets, statins, diuretics, anti-arrythmic drugs and re-admitted after a few days for CRT- D implantation.

Course in the Hospital

The patient was evaluated to have left bundle branch block (QRS duration of 160ms) on electrocardiogram, dilated cardiomyopathy with severe LV dysfunction (LVEF – 20%), grade II mitral regurgitation on echocardiography, non sustained ventricular tachycardia on Holter monitoring. He was taken up for coronary angiogram that showed normal coronaries. A CRT device with an implantable cardioverter defibrillator (CRT-D) implantation was done under local anesthesia.

Procedure: TPI done through right femoral artery route. Through left venous route, coronary sinus was intubated with a MB 2 catheter, LV lead was placed. Subsequently RA lead and ICD lead was placed in the RV apex. All the leads were then connected to the Medtronic Egida pulse generator and were placed in the left mammary pocket. It was closed in layers. The procedure was successful, with no complications. Subsequent ECG following the implant placement showed shortening of QRS duration to 124ms, (as shown in figure 1 and figure 3). The patient was monitored in the ward, continuing anti platelets, diuretics. He did not develop any surgical complications and was discharged on day 4 of admission.

Diagnosis

- 1) Dilated Cardiomyopathy with Severe Left Ventricular Dysfunction (LVEF – 20%)
- 2) Coronary Angiogram Normal Coronaries
- 3) Holter Monitoring Multiple Episodes of NSVT
- 4) S/P CRT- D Implantation

Discussion

Cardiac resynchronization therapy (CRT) devices limits mortality by pacing induced cardiac

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resynchronization and treating ventricular arrythmias (Vas) by using implantable internal defibrillator (ICD). Cardiac resynchronization pacemakers (CRT-P) and therapy CRT defibrillators (CRT-D) each impart improvements mortality in groups of patients with in dyssynchronous heart failure.⁽²⁾ The intention of CRT ins to enhance electromechanical coupling the heart via generating a greater efficient sequence of impulse generation and conduction. The instantaneous hemodynamic benefits of the technique encompasses the expanded diastolic filling and more efficient systolic contractility. A gradual breakdown of the pump or sudden death caused by arrythmias is the result of mortality due to CHF. The CRT, in tandem with an ICD, can delay the pump failure process and deter sudden cardiac death.⁽³⁾ The joint recommendations 2002 of the American College of Cardiology, the American Heart Association and the Pacing and Electrophysiology American North Society approve the use of CRT in patients with medically refractory, symptomatic, NYHA class III or IV disease and a ORS interval of atleast 130msec, a left ventricular end-diastolic diameter of atleast 55mm, and LVEF of 30 % or less. The American Heart Association Research Recommendation in April 2005 updated these recommendations that "optimal candidates for CRT have a dilated cardiomyopathy on an ischemic or non ischemic basis, an LVEF <0.35, a ORS complex >120 ms, and sinus rhythm and are NYHA functional class III or IV despite maximal medical therapy for heart failure."⁽⁴⁾ The benefits of CRT include the following: •Improved cardiac contractility and increase ejection fraction •Reduced mitral regurgitant fraction, which enhances cardiac output •Improved exercise tolerance in the 6minute walk test •Improved New York Heart Association functional class • Improved quality of life •Reduced re-hospitalization for worsening heart failure The role of CRT without an ICD in reducing mortality from heart failure is still unresolved.⁽³⁾ While CRT by itself obviously improves heart failure symptoms, the addition of a

defibrillator tends to increase survival dramatically.⁽⁵⁾ The most frequent complication is the malfunction of the lead positioning, and the most serious adverse circumstances are heart perforation and coronary sinus dissection.⁽⁶⁾

On complete evaluation of our patient, a diagnosis of dilated cardiomyopathy with severe LV dysfunction, Holter monitoring showing multiple episodes of NSVT was made. ECG showed LBBB with widened QRS complexes. CAG was normal. In view of these findings, the patient underwent CRT- D implantation, following which ECG changes were found to be reverted.

Conclusion

Heart failure is a complex syndrome orchestrated by multiple complex and overlapping pathways that can result in modes of death secondary to pump failure or ventricular arrythmias. CRT has represented a revolution in the treatment of HF. Adaptive CRT is a potential therapeutic option in patients with dilated cardiomyopathy and severe functional MR. Modern goals of CRT are to enhance response to CRT in those patients who respond to the therapy as well as to reduce the proportion of patients not responding to therapy.

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