Case Report

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Evaluation Of The Therapeutic Effects Of Cardiac Resynchronization Therapy In Patients With Heart Failure With Reduced Ejection Fraction (Hfref) And Concomitant Hypotension

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1. Abstract

We report a case of a male patient with dilated cardiomyopathy (DCM) leading to heart failure (HF). Following one month of cardiac resynchronization therapy pacemaker (CRT-P), a reevaluation indicated the persistence of heart failure symptoms accompanied by sustained hypotension (90/70 mmHg), necessitating rehospitalization. After proactive nursing care and medication adjustments, there was a marked improvement in the patient's heart failure symptoms, and the left ventricular ejection fraction (LVEF) significantly increased. Upon stabilization, an assessment deemed the patient suitable for discharge with continued observation. While there was significant improvement in cardiac structure one month post CRT-P, evident heart failure symptoms persisted with sustained hypotension, suggesting a potential association with inadequate post-CRT-P management. This report aims to discuss the feasibility of CRT-P in improving heart failure symptoms in patients with reduced ejection fraction accompanied by low blood pressure, as well as to explore post-CRT-P management strategies and approaches.

2. Keywords:

Cardiac resynchronization therapy; dilated cardiomyopathy; heart failure with reduced ejection fraction.

3. Introduction

Cardiac resynchronization therapy (CRT) is an instrumental approach to the management of chronic heart failure (CHF), specifically tailored for patients exhibiting a significant reduction in left ventricular ejection fraction (LVEF) coupled with asynchronous ventricular contraction. Serving as a non-pharmacological intervention for heart failure patients, CRT has gained widespread clinical application. Its mechanism involves biventricular pacing, orchestrating synchronized ventricular contractions to ameliorate cardiac pumping function in individuals with heart failure. This intervention not only enhances the quality of life for patients but also, when applied over an extended period, exhibits the potential to reverse ventricular remodeling [1]. Nevertheless, despite its clinical efficacy, there remains a subset of patients who exhibit suboptimal responses to CRT.

4. Case report

4.1. Patient's condition:

The patient, a 45-year-old male, presented over a year ago with chest tightness, shortness of breath, and nocturnal dyspnea, notably exacerbated during nighttime. Approximately five years prior, he received a diagnosis of "coronary heart disease" following coronary angiography at another hospital. The specific details of the coronary artery stent implantation procedure are unavailable. Postoperatively, he underwent prolonged management involving antiplatelet aggregation, lipid regulation, and coronary artery dilation, resulting in a gradual alleviation of symptoms compared to the initial presentation. Ten days ago, the patient experienced a worsening of shortness of breath without an apparent trigger, accompanied by paroxysmal nocturnal dyspnea. Ineffectiveness of symptomatic treatment prompted the decision for hospitalization. The patient has a seven-year history of hypertension and a five-year history of coronary heart disease.

4.2. Laboratory examinations:

Upon admission, the 12-lead electrocardiogram (ECG) (Figure 1) revealed an acute anterior myocardial infarction, moderate ST-segment depression, a QRS duration of 122 milliseconds, and left atrial enlargement. Holter exhibited paroxysmal ventricular tachycardia. The echocardiogram

Annals of Clinical and Medical Case Reports

demonstrated generalized hypokinesis of the left ventricular wall; enlargement of the left atrium (42mm), dilation of the left ventricle: end-diastolic diameter (LVEDD) 70mm, end-systolic diameter (LVESD) 62mm; restricted opening of the mitral valve leaflets with accelerated forward flow and moderate regurgitation (MR) (regurgitant velocity 430cm/s); minimal pericardial effusion; impaired left ventricular systolic function, characterized by a reduced LVEF (27%). The NT-proBNP level was measured at 21300pg/ml.

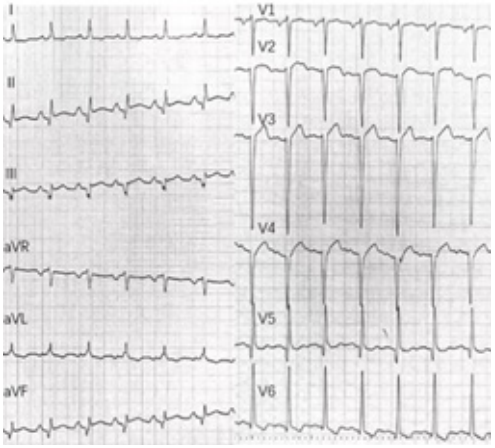

Figure 1: Admission ECG

4.3. Course of treatment:

The patient previously underwent coronary stent implantation. Currently, there are prominent symptoms of heart failure accompanied by paroxysmal ventricular tachycardia. Temporary treatment includes inotropes, diuretics, vasodilators, and other measures to correct heart failure. Coronary angiography was performed after the correction of heart failure, indicating good blood flow in the LAD stent, with no indication for intervention in other vessels. Ischemic cardiomyopathy as a cause of heart failure was ruled out, establishing an indication for CRT. The patient and their family were explicitly informed about the relevant risks. Three days later, the patient underwent CRT-P. Postoperative ECG (Figure 2) showed sinus tachycardia, anterior wall myocardial infarction, left atrial enlargement, and a QRS duration of 100 ms. NT-proBNP: 2670 pg/ml. The patient's symptoms improved, and they were discharged on the fifth day postoperatively. After discharge, the patient has been on a long-term regimen of regular medications, including bisoprolol 5 mg, sacubitril valsartan 50 mg, dapagliflozin 10 mg, spironolactone 20 mg, amiodarone 200 mg, aspirin 100 mg, and rosuvastatin calcium 10 mg.

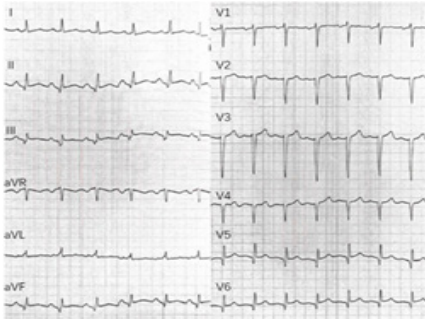

Figure 2: Postoperative ECG

4.4. Outcome and follow-up:

One month post CRT-P, a reassessment revealed that the patient continued to experience chest tightness and shortness of breath, with no lower limb edema. Continuous cardiac monitoring indicated that the patient's blood pressure remained at a relatively low level (90/70 mmHg), and ECG (Figure 3) showed a paced heart rate with a QRS duration of 112 ms. NT-proBNP levels were elevated at 6160 pg/ml. Chest X-ray (Figure 4) confirmed the proper position of the CRT-P device and normal lead placement.

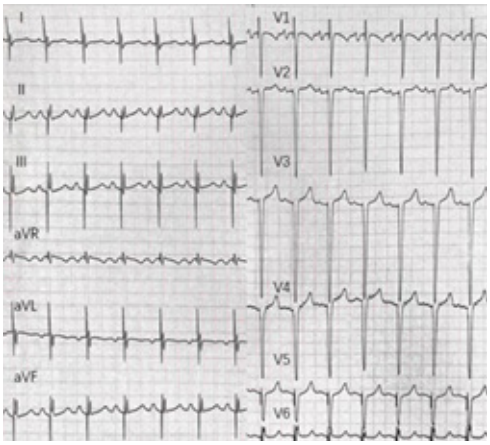

Figure 3: Postoperative 1-Month ECG

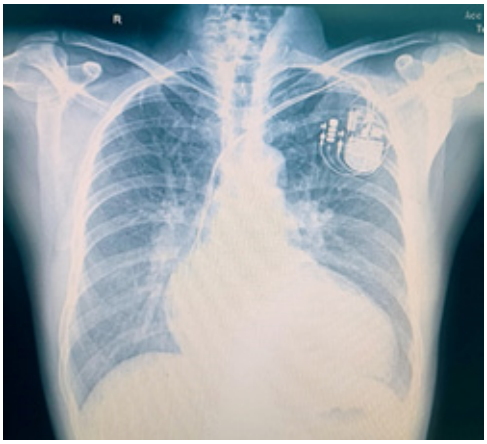

Figure 4: Postoperative 1-Month Chest X-ray

Interventions were implemented, including inotropic support and vasodilation, to address the ongoing heart failure. The prescribed medications upon admission included bisoprolol 5 mg, dapagliflozin 10 mg, digoxin 0.125 mg, spironolactone 20 mg, amiodarone 200 mg, aspirin 100 mg, and rosuvastatin calcium 10 mg. Additionally, the patient received recombinant human brain natriuretic peptide at a dose of 0.54 mg via microinfusion pump and a microinfusion pump injection of noradrenaline bitartrate at 4 mg. Upon stabilization of blood pressure (110/73 mmHg) and other vital signs, a subsequent echocardiogram

revealed left atrial enlargement (42 mm), LVEDD 58 mm, LVESD 41 mm, severe MR (regurgitant velocity 307 cm/s), and a LVEF of 40%. The patient's heart failure symptoms significantly improved compared to previous status, with restructuring of cardiac architecture and an improved ejection fraction. After careful evaluation, it was deemed appropriate to discharge the patient with continued observation. Relevant instructions and discharge prescriptions were provided to the patient and their family. The patient was discharged as per the plan.

Table 1: Comparison of Key Cardiac Indicators Before and After Surgery.

		QRS ms	LVEDD (mm)	LVESD (mm)	LVEF (%)	MR	NT-pro BNP (pg/ml)
preoperative	122	70	62	27	severe	21300	
Postoperative 1-Month	112	58	41	40	severe	6160	

5. Discussion

Heart failure is a clinical syndrome characterized by a decrease in cardiac output due to abnormal cardiac structure or function, representing the terminal stage of various cardiac diseases. According to the LVEF, heart failure can be classified into three types: LVEF ≤ 40%, heart failure with reduced ejection fraction (HFrEF); LVEF 41%~49%, heart failure with mildly reduced ejection fraction (HFmrEF); LVEF ≥ 50%, heart failure with preserved ejection fraction (HFpEF) [2]. The 2021 ESC Guidelines on cardiac pacing and cardiac resynchronization therapy (CRT) suggest that CRT is a viable option for patients with reduced ejection fraction, aiming to alleviate symptoms and reduce heart failure mortality [3]. CRT includes CRT-P and CRT defibrillators (CRT-D). CRT-P is a standard CRT, while CRT-D, in addition to regular CRT functions, incorporates automatic defibrillation. In this case, the patient underwent CRT-P due to DCM and severe mitral regurgitation secondary to reduced ejection fraction. One month after CRT-P, the patient experienced persistent hypotension and worsening heart failure symptoms, with NT-proBNP levels increasing from 2670 pg/ml to 6160 pg/ml. Hospitalization and medication adjustments resulted in improved cardiac structure, remodeling, reduced left ventricular enlargement, improved cardiac function, and an increase in LVEF from 27% to 40%. However, post-discharge, heart failure symptoms recurred, and NT-proBNP levels rose, possibly attributed to inadequate post-CRT management.

Considering the patient's history of hypertension and long-term postoperative use of sacubitril valsartan, in addition to heart failure medications, the combined antihypertensive effects and the inherent reduction in ejection fraction might contribute to persistent hypotension and recurrent heart failure symptoms. Additionally, the outcome variation among patients undergoing CRT may be related to the degree of patient response. As a non-pharmacological targeted therapy for heart failure patients, the results differ significantly among those receiving CRT. In clinical practice, approximately 20% of CHF patients experience a gradual elevation of LVEF to normal levels after CRT implantation, and these individuals are referred to as "CRT super-responders"[4]. Furthermore, another subset of patients, constituting 20% to 40%, exhibits no improvement in New York Heart Association (NYHA) functional classification six months after CRT implantation, categorizing them as "CRT non-responders." However, defining CRT response lacks consensus, with assessments relying on postoperative symptoms, NYHA functional class, and/or echocardiography [5]. Furthermore, some scholars have pointed out that the introduction of the concept of "CRT response" is inappropriate. This is because CRT is just one of the adjunctive therapies aimed at slowing the progression of heart failure. Heart failure is a progressive disease, and even though CRT can reduce the occurrence of cardiac dyssynchrony during contraction, it cannot prevent the underlying disease's potential advancement [6]. Factors contributing to poor CRT response include inappropriate patient selection, device-related issues, arrhythmias, abnormal lead placement, suboptimal drug effects, and inadequate postoperative management. Prospective studies emphasize multidisciplinary post-CRT management, highlighting the importance of collaborative efforts and drug assistance for CRT patients to enhance outcomes and reduce readmission rates [8].

CRT as a pivotal intervention in the treatment of heart failure, remains a recommended therapeutic approach for ameliorating symptoms in heart failure patients, despite its inherent limitations. Given the current status of CRT, researchers have proposed optimized programming algorithms such as Adaptive CRT and SyncAV to enhance the therapeutic efficacy of CRT. Additionally, physiologically synchronized methods for cardiac resynchronization have emerged as a new research focus. Examples include His bundle pacing (HBP) and Left Bundle Branch area Pacing (LBBaP). In comparison to traditional pacing, physiological pacing offers advantages such as low thresholds, high sensing capabilities, high success rates, and electrode stability [9]. Finally, for heart failure patients after CRT implantation, strict adherence to pharmacological treatment is essential. The post-CRT management should involve a multidisciplinary, long-term collaboration to maximize the impact of CRT and achieve the ultimate therapeutic goals.

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