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(CASE REPORT)

Implantation of ICD in a patient with heart failure with mildly reduced ejection fraction

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Abstract

Heart failure affects approximately 2% of the adult population in developed countries, with prevalence escalating to 10% among those aged over 70 years. Patients with Heart Failure with Mildly Reduced Ejection Fraction (HFmrEF) exhibit a risk profile comparable to those with reduced ejection fraction (EF), necessitating their classification as high-risk and tailoring therapeutic goals accordingly. The clinical case presented here of a patient with HFmrEF and elevated arrhythmic risk underscores the need for refining indications for primary prevention of sudden cardiac death (SCD). Furthermore, there is an imperative to identify and validate a panel of investigations to guide decision-making for patients with borderline indications for high-voltage device implantation. This approach aims to optimize the benefit-to-risk ratio for these individuals.

Keywords: Left Ventricular Dysfunction; Myocardial Fibrosis; Implantable Defibrillator

1. Introduction

Heart failure (HF) is a clinical syndrome characterized by fatigue and shortness of breath, often accompanied by elevated jugular venous pressure, auscultatory findings of pulmonary congestion, and peripheral edema [1]. Based on the assessment of left ventricular systolic function, HF is categorized into three phenotypes: HF with reduced ejection fraction (HFrEF), HF with preserved ejection fraction (HFpEF), and HF with mildly reduced ejection fraction (HFmrEF).

Data from randomized trials indicate that patients with HFmrEF benefit equally from the recommended HF therapies as those with HFrEF. Additionally, patients presenting with congestive HF symptoms are at an increased risk of fatal cardiovascular events, including sudden cardiac death (SCD). The primary cause of adverse outcomes in HF patients is disease decompensation, with the second most common cause being sudden arrhythmic cardiac death, which is not directly related to the ejection fraction (EF) [2].

These findings suggest the need for reassessment of risk in patients with EF greater than 35% to reduce cardiovascular mortality within this patient population.

2. Case Description

A 50-year-old woman presented to the outpatient clinic with symptoms of congestive heart failure (CHF), classified as NYHA functional class III, with a duration of 2–3 weeks. She had no prior history of cardiac disease and had not previously sought medical care.

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On physical examination, moist crackles were noted at the bases of both lungs. The heart rate was 70 bpm, with a systolic murmur at the cardiac apex radiating to the axilla. Her blood pressure was 100/60 mmHg. An electrocardiogram (ECG) revealed sinus rhythm, left axis deviation, and negative T waves in leads I, aVL, and V4–V6 (Figure 1).

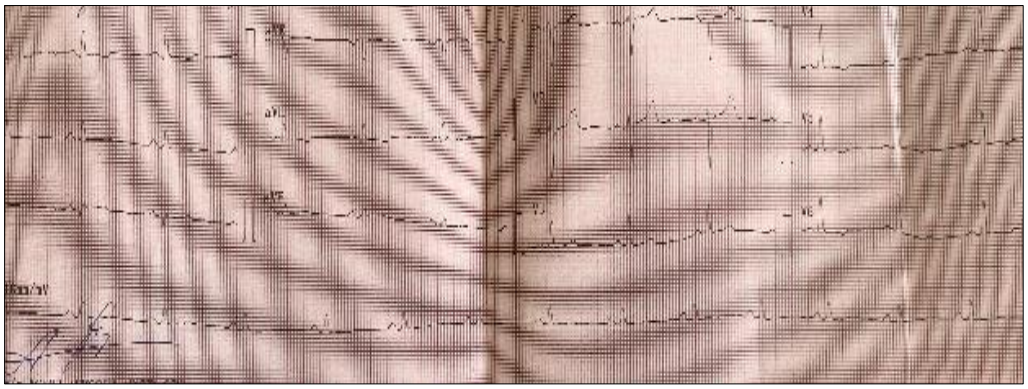

Figure 1 ECG of the patient in the outpatient clinic

Echocardiographic evaluation revealed a non-dilated left ventricle (LV) with a moderately reduced ejection fraction (EF) of 42% (Figure 2). The LV showed symmetrical wall motion with diffusely depressed contractility. Moderate mitral regurgitation was observed, along with restrictive diastolic filling patterns, evidenced by an E/A ratio of 2.01 and an E/E' ratio of 15. The left atrium was mildly dilated, with an indexed volume of 45 mL/m^2.

The right ventricle (RV) was non-dilated, with evidence of moderate tricuspid regurgitation and an indirectly measured systolic pulmonary artery pressure (SPAP) of 30 mmHg.

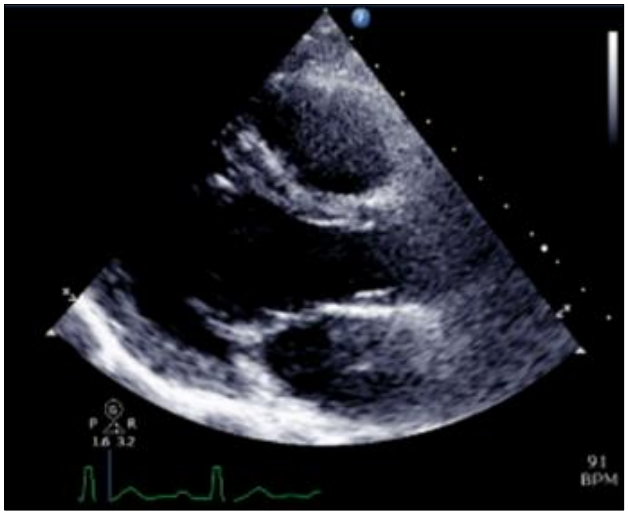

Figure 2 Baseline echocardiographic examination—parasternal long-axis view

To determine the underlying etiology of the systolic dysfunction, coronary angiography was performed after the stabilization of heart failure symptoms (Figure 3). The results showed no evidence of stenotic coronary atherosclerosis.

Laboratory evaluations, including thyroid-stimulating hormone (TSH), were within reference ranges. Treatment was initiated in accordance with current guidelines to improve symptoms: diuretics (Class I), Dapagliflozin/Empagliflozin (Class I), ACE inhibitors/ARNI/ARB (Class IIb), and beta-blockers (Class IIb) [1].

The prescribed therapy included torasemide 10 mg in the morning, bisoprolol 2.5 mg in the morning with dose titration to 5 mg daily, eplerenone 25 mg in the morning, ramipril 2.5 mg in the evening, and an SGLT2 inhibitor. Therapy with an angiotensin receptor-neprilysin inhibitor (ARNI) was initiated but discontinued due to hypotension and replaced with ramipril at the minimal effective dose.

At a follow-up examination three months later, the patient demonstrated an improved functional status to NYHA class I. However, echocardiographic assessment showed no significant change in left ventricular (LV) systolic function. A reduction in mitral and tricuspid regurgitation to grade I was observed, along with a decrease in indirectly measured systolic pulmonary artery pressure to 20 mmHg.

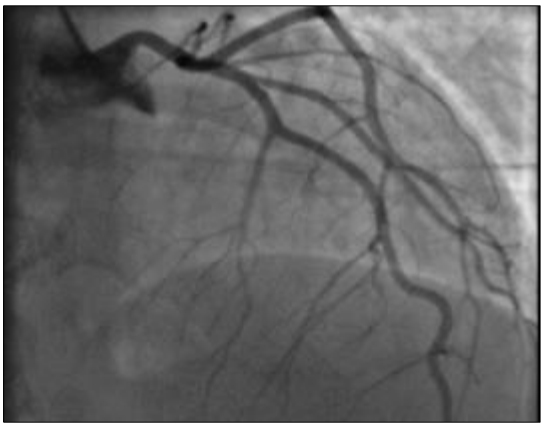

Figure 3 Invasive coronary angiography. Right anterior oblique (RAO) projection at 20°, showing no evidence of stenosis in the left main artery (LM), left anterior descending artery (LAD), or left circumflex artery (LCx)

Given the presence of LV systolic dysfunction (ejection fraction [EF] below 45%) without LV cavity dilation, and in the absence of pressure or volume overload, such as hypertension or valvular pathology, or ischemic coronary artery disease as underlying causes, the diagnosis of non-dilated left ventricular cardiomyopathy was established [3].

To better evaluate the patient's prognosis and risk of arrhythmic events, the team decided to perform cardiac magnetic resonance imaging with late gadolinium enhancement (CMR-LGE). The results revealed extensive areas of myocardial fibrosis predominantly affecting the subepicardial and intramural regions of the LV (Figure 4). Moderate LV systolic dysfunction with an EF of 41% was confirmed.

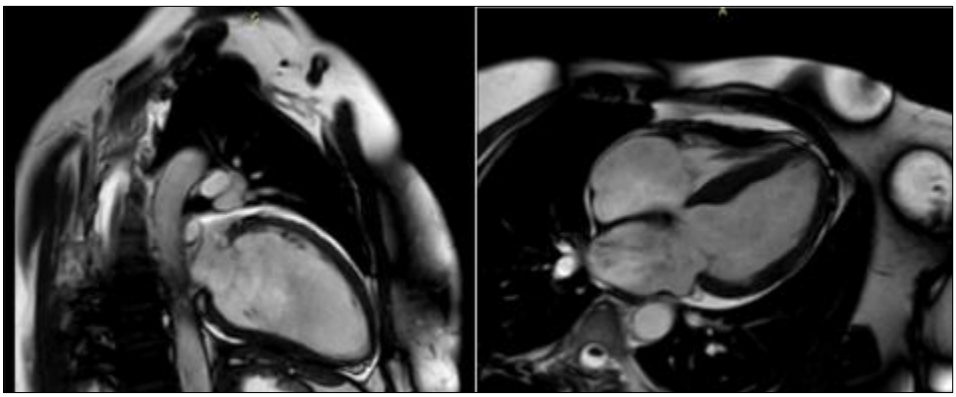

Figure 4 Cardiac MRI with late gadolinium enhancement showing a longitudinal section of the left ventricle (LV) and a four-chamber view, visualizing the LV, right ventricle (RV), left atrium (LA), and right atrium (RA). (LV - left ventricle, RV - right ventricle, LA - left atrium, RA - right atrium)

A genetic test was discussed with the patient, but due to personal considerations, it was deferred to a later stage. Given the patient's history of palpitations, 24-hour Holter ECG monitoring was performed, revealing polymorphic ventricular ectopy occupying approximately 10% of the day. Long-term treatment with amiodarone was deemed unsuitable due to patient intolerance. Electrophysiological study and potential ablation of ventricular ectopy were also considered but declined by the patient.

As the Holter ECG findings were recorded while the patient was on the maximum tolerated dose of bisoprolol, the decision was made to implant a loop recorder to assess the risk of arrhythmogenic events in the context of significant intramural myocardial fibrosis detected on cardiac MRI.

Follow-up telemetry from the implanted device one week later revealed several episodes of non-sustained monomorphic ventricular tachycardia (VT), lasting 4–5 seconds with a heart rate of 180 bpm.

Given these findings, the patient was classified as high-risk, and an ICD-DDDR was implanted for primary prevention of sudden cardiac death (SCD) (Figure 5).

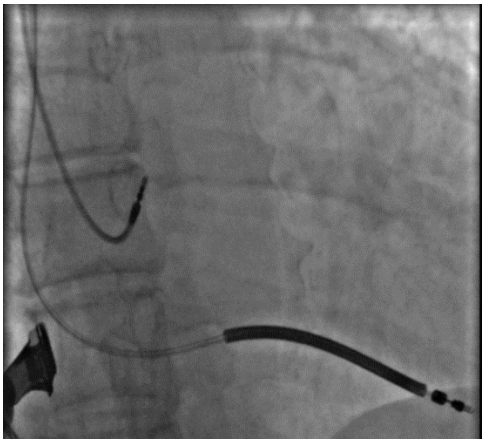

Figure 5 Implanted with a biventricular defibrillator with an electrode in the right atrial appendage and a high-voltage electrode in the apex of the left ventricle

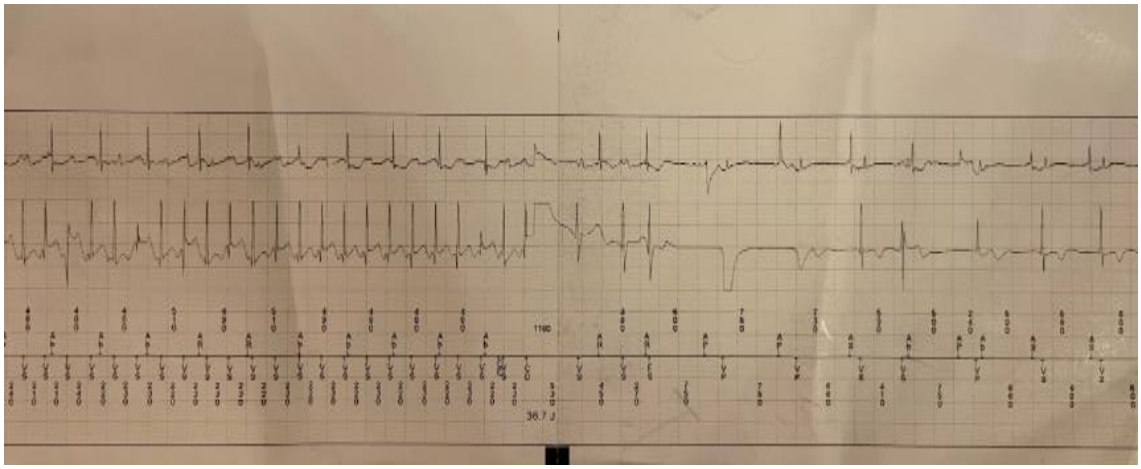

Figure 6 Telemetry data from the implanted defibrillator. The first row shows the atrial channel recording. The second row shows the ventricular channel, where the ventricular rate is 270 bpm and the atrial rate is 98 bpm, which are among the determinants for the device's detection of ventricular tachycardia (VT). Due to the confirmed episode of high-frequency VT falling within the VF zone, a 36J shock was delivered, successfully terminating the episode

At the 3-month follow-up visit after implantation, several episodes of non-sustained ventricular tachycardia (VT) were recorded by the device. At the 5-month follow-up, the patient presented to the emergency clinic with a history of a syncopal episode.

Telemetry data revealed sustained VT with a heart rate of 270 bpm, and because it fell within the ventricular fibrillation (VF) zone, the device successfully defibrillated the patient with a 36J shock (Figure 6).

3. Discussion

The 12-lead electrocardiogram (ECG) is a fundamental diagnostic tool for a range of cardiac diseases. However, documenting arrhythmias associated with existing symptoms often proves challenging. Depending on the frequency of the symptoms, both the appropriate recording device and the duration of the recording should be selected. Holter ECG monitoring for a period of 24 to 48 hours is applicable when patients present with daily symptoms [3]. Implantable loop

recorders (ILR) are considered when episodes of paroxysmal tachyarrhythmias and a history of syncopal symptoms with an unclear etiology are suspected.

In the case of the patient presented here, polymorphic ventricular ectopy was recorded while on the maximum tolerated dose of a beta-blocker. We hypothesized that the documented left ventricular dysfunction might be a result of the observed ectopy, but we lacked data on the burden of ectopic beats prior to initiating pharmacological therapy. Given that the heart failure treatment over a 3-month period did not lead to normalization of left ventricular pumping parameters, coupled with MRI findings indicating myocardial fibrosis affecting extensive intramural regions, we concluded that the left ventricular remodeling process had advanced, with a corresponding loss of contractile tissue.

Over the past two decades, numerous studies have been conducted to assess the necessity and benefits of implantable cardioverter-defibrillators (ICDs). The results have been more than controversial. On one hand, data from an observational study indicate that most victims of sudden cardiac death (SCD) do not have severe left ventricular systolic dysfunction and are generally not protected by ICDs, as they fall outside the indications for primary prevention with implantable devices [4]. On the other hand, data from the DEFINITE (Defibrillators in Non-Ischemic Cardiomyopathy Treatment Evaluation) trial show a low incidence of shock therapy from implanted defibrillators as primary prevention in patients with non-ischemic dilated cardiomyopathy (DCM) [5]. The results of these and other studies highlight the need for more precise indications for primary prevention of SCD with ICDs, particularly in non-ischemic dilated cardiomyopathy. It is crucial to identify appropriate imaging studies that can distinguish high-risk individuals and serum markers with sufficient sensitivity and specificity to be used for risk stratification.

In a meta-analysis conducted by Di Marco et al. on studies examining the relationship between the presence of significant myocardial fibrosis, detected with CMR and LGE, and the risk of sudden cardiac death (SCD), interesting findings were observed [6]. In studies reporting fibrosis above 35%, an arrhythmic endpoint was observed in 23.9% of patients with LGE on CMR and in 5.6% of patients without LGE. In cases with fibrosis below 35%, the arrhythmic endpoint was found in 19.6% of patients with LGE and 4.1% in those without LGE. A significant association was also noted between the presence of LGE and the manifestation of ventricular tachycardia (VT) or SCD, both in the group with reduced left ventricular ejection fraction (LVEF) below 35% and in those with LVEF above 35%. This meta-analysis showed that LGE is significantly associated with an arrhythmic endpoint (OR 7.8) among studies that included only patients with primary prevention of SCD and no significant coronary atherosclerosis. Patients with LGE had a relatively high annual rate of arrhythmic events (17.2%), while those without LGE, who accounted for approximately 58% of the individuals included in studies on primary prevention of SCD with ICD, had a relatively low rate of arrhythmic events (2.1% per year). Therefore, incorporating the presence of LGE into the criteria for primary prevention of SCD with ICD may help guide treatment toward a subgroup of patients with a high-risk profile. On the other hand, ICD implantation in low-risk patients exposes them to potential periprocedural complications from device implantation, which is unlikely to improve their prognosis.

Myocardial remodeling in the presence of heart failure is associated with the development of interstitial fibrosis and collagen deposition [7]. Both replacement and diffuse myocardial fibrosis are linked to adverse outcomes, both in patients with indications for implantation of cardiac devices and in those undergoing cardiac surgical interventions [8, 9].

Although there has been an active search for reliable biomarkers to assess activated collagen synthesis in recent years [10, 11], CMR-LGE has been established as the gold standard for evaluating myocardial fibrosis burden. This highlights that this imaging technique is a reliable method for determining risk and provides valuable information for patients with heart failure symptoms, as well as those with conduction abnormalities, implanted electronic devices, and ischemic heart disease (IHD) with operative revascularization.

4. Conclusion

Risk assessment in patients with moderately reduced systolic function is often a significant clinical challenge. To make the correct decision, a comprehensive approach should be employed, utilizing all available clinical, imaging, and laboratory tests. The implantation of monitoring devices provides valuable information about the existing rhythm and conduction pathology in this patient group, while performing CMR with LGE can serve as a powerful tool for differentiating high-risk individuals. The selection of reliable biomarkers for activated collagen synthesis, among the many known molecules, requires the conduction of extensive randomized trials.

Compliance with ethical standards

Disclosure of conflict of interest

No conflict of interest to be disclosed.

Statement of informed consent

Informed consent was obtained from all individual participants included in the study.

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