Case Report

Cardiac Resynchronization Therapy Optimization in a Patient with Left Ventricular Non-Compaction Cardiomyopathy and Morbid Obesity

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ABSTRACT: Left ventricular non-compaction cardiomyopathy (LVNC) is a rare myocardial disorder associated with heart failure and arrhythmias. Cardiac resynchronisation therapy (CRT) is an established treatment in patients with electrical dyssynchrony; however, achieving optimal response remains challenging in complex clinical scenarios. We report a 51-year-old male with LVNC, severe left ventricular dysfunction, morbid obesity, and recurrent atrial and ventricular arrhythmias. Despite initial improvement following CRT-D implan-tation, the patient developed progressive heart failure and recurrent ventricular tachycardia with high atrial arrhythmia burden, resulting in ineffective biventricular pacing. Following unsuccessful device optimisation strategies, a multidisciplinary decision was made to proceed with atrioventricular node ablation. This resulted in marked clinical and echocardiographic improvement, including recovery of left ventricular ejection fraction and symptomatic status. This case highlights the importance of ensuring adequate CRT delivery and demonstrates the pivot-al role of atrioventricular node ablation in patients with refractory atrial arrhythmias and suboptimal CRT response.

KEYWORDS: *LVNC, CRT, HF, AVN, AT, VT.*

Introduction

Left ventricular non-compaction cardiomyopathy (LVNC) is characterized by prominent trabeculations and deep intertrabecular recesses, thought to arise from incomplete myocardial compaction during embryogenesis [1,2].

It is associated with progressive systolic dysfunction, heart failure, and a high burden of atrial and ventricular arrhythmias [3,4,5].

Cardiac resynchronisation therapy (CRT) is an established treatment for patients with heart failure and electrical dyssynchrony, particularly in the presence of left bundle branch block (LBBB) [6-11].

However, the effectiveness of CRT depends on achieving a high percentage of biventricular pacing, which may be compromised by atrial arrhythmias and suboptimal device programming.

In addition, morbid obesity presents unique challenges, including technical procedural difficulties, impaired imaging quality, and increased arrhythmic burden, further complicating management.

Objective

To demonstrate the importance of a multidisciplinary approach in optimizing CRT response in a complex patient with LVNC, focusing on the role of atrioventricular node ablation.

Case Report

In this case report, we aim to emphasize the pivotal role of a multidisciplinary approach in optimizing the clinical response to cardiac resynchronization therapy (CRT).

We present the case of a 51-year-old male patient with a diagnosis of familial left ventricular non-compaction cardiomyopathy (LVNC), initially established in 2008 based on comprehensive echocardiographic and cardiac magnetic resonance imaging findings.

The patient has a significant family history of cardiomyopathy; notably, his brother was found to carry variants of uncertain significance (VUS) in the *MYH7* gene (c.1045A>G, p.Met349Val) and the *DSP* gene (c.7847C>T, p.Ser2616Leu).

However, genetic testing in the patient himself did not identify any pathogenic or likely pathogenic variants.

At the time of diagnosis, left ventricular (LV) systolic function was only mildly impaired.

However, this was followed by a relatively rapid clinical deterioration, culminating in severe LV systolic dysfunction with an ejection fraction (EF) of approximately 20%, alongside hospitalization for decompensated heart failure.

Ischaemic aetiology of his LV dysfunction was ruled out by an invasive coronary angiogram, Figure 1.

In the context of a concomitant left bundle branch block (LBBB) with a QRS duration of 165ms, the patient underwent implantation of a

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cardiac resynchronization therapy defibrillator (CRT-D) device in 2008, with the aim of providing both ventricular resynchronization and primary prevention of sudden cardiac death.

Following device implantation, the patient demonstrated a favorable clinical and echocardiographic response, with improved functional capacity and partial recovery of LV systolic function, with EF improving to 35-40%.

Guideline-directed medical therapy for heart failure was initiated and optimized, resulting in a prolonged period of clinical stability.

Despite these improvements, significant comorbidities persisted.

The patient had morbid obesity, with a body mass index (BMI) of 52.1 kg/m², which remained a major contributor to his overall cardiovascular risk profile.

He was therefore referred to a specialist bariatric service for structured weight management and consideration of surgical intervention.

In addition, he was diagnosed with obstructive sleep apnoea and commenced on continuous positive airway pressure (CPAP) therapy.

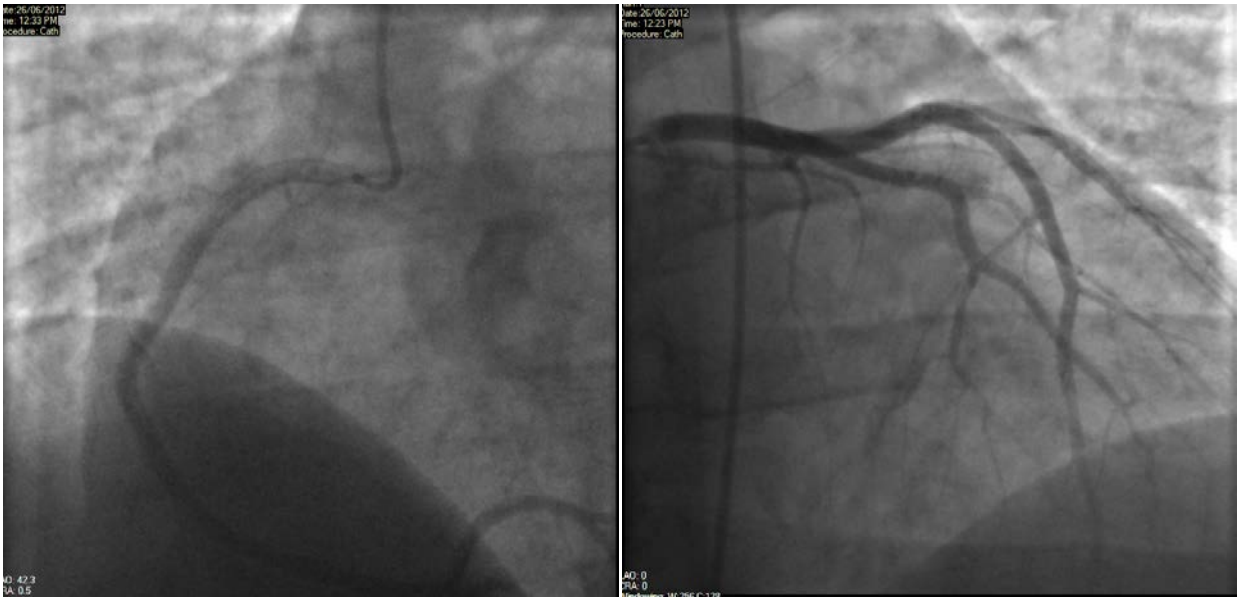

Figure 1. Diagnostic coronary angiography demonstrating unobstructed coronary arteries. Left panel: right coronary artery. Right panel: left coronary system with patent left main stem, left anterior descending, and left circumflex arteries.

In 2021, the patient experienced a significant clinical deterioration, accompanied by a marked decline in left ventricular systolic function, with the left ventricular ejection fraction (LVEF) reduced once again to 15-20%.

Concomitantly, he was found to be in persistent typical atrial flutter, which was considered a key contributing factor to his worsening heart failure status, likely through a combination of tachycardia-mediated cardiomyopathy and loss of effective atrioventricular synchrony, thereby compromising the efficacy of cardiac resynchronization therapy.

Initial rhythm control strategies included two attempts at inpatient direct current cardioversion (DCCV), both of which were unsuccessful in achieving sustained sinus rhythm.

In view of the refractory nature of the arrhythmia and its haemodynamic impact, the patient was referred for electrophysiological intervention.

He subsequently underwent a successful cavotricuspid isthmus (CTI) ablation in July 2022 at Oxford University Hospitals NHS Foundation Trust (John Radcliffe Hospital).

The procedure resulted in bidirectional isthmus block and restoration of sinus rhythm, with subsequent symptomatic improvement and partial clinical stabilization.

Despite an initially favorable post-ablation course, the patient experienced a further abrupt deterioration in December 2022.

He presented in cardiogenic shock and required admission to the intensive care unit (ITU).

This acute decompensation was precipitated by multiple appropriate therapies delivered by his implantable cardioverter-defibrillator (ICD) in response to recurrent episodes of sustained ventricular tachycardia (VT).

Device interrogation provided important mechanistic insights into the arrhythmic substrate.

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As illustrated in Figure 2.1, the intracardiac electrogram (EGM) initially demonstrated an irregular rhythm characterized by a predominance of atrial sensed events (As) relative to ventricular sensed events (Vs), suggestive of a supraventricular tachyarrhythmia.

This subsequently evolved into a more regular and organized tachycardia, with a subtle but discernible change in the near-field ventricular EGM morphology, accompanied by a shift towards a predominance of ventricular events (Vs>As).

This transition raised the possibility of a dual tachycardia mechanism, with degeneration from a supraventricular arrhythmia into sustained ventricular tachycardia.

Figure 2.2 and Figure 2.3 illustrate electrocardiographic evidence of the ventricular tachycardia (VT) and atrial tachycardia (AT).

Antitachycardia pacing (ATP) was delivered but was unsuccessful in terminating the arrhythmia, necessitating high-energy ICD shock therapy.

The shock was appropriately delivered and successfully terminated both the ventricular tachycardia and the concomitant atrial arrhythmia, restoring sinus rhythm.

This episode highlights the complex interplay between atrial and ventricular arrhythmias in advanced cardiomyopathy, as well as the critical role of device therapy in preventing sudden cardiac death in this high-risk population.

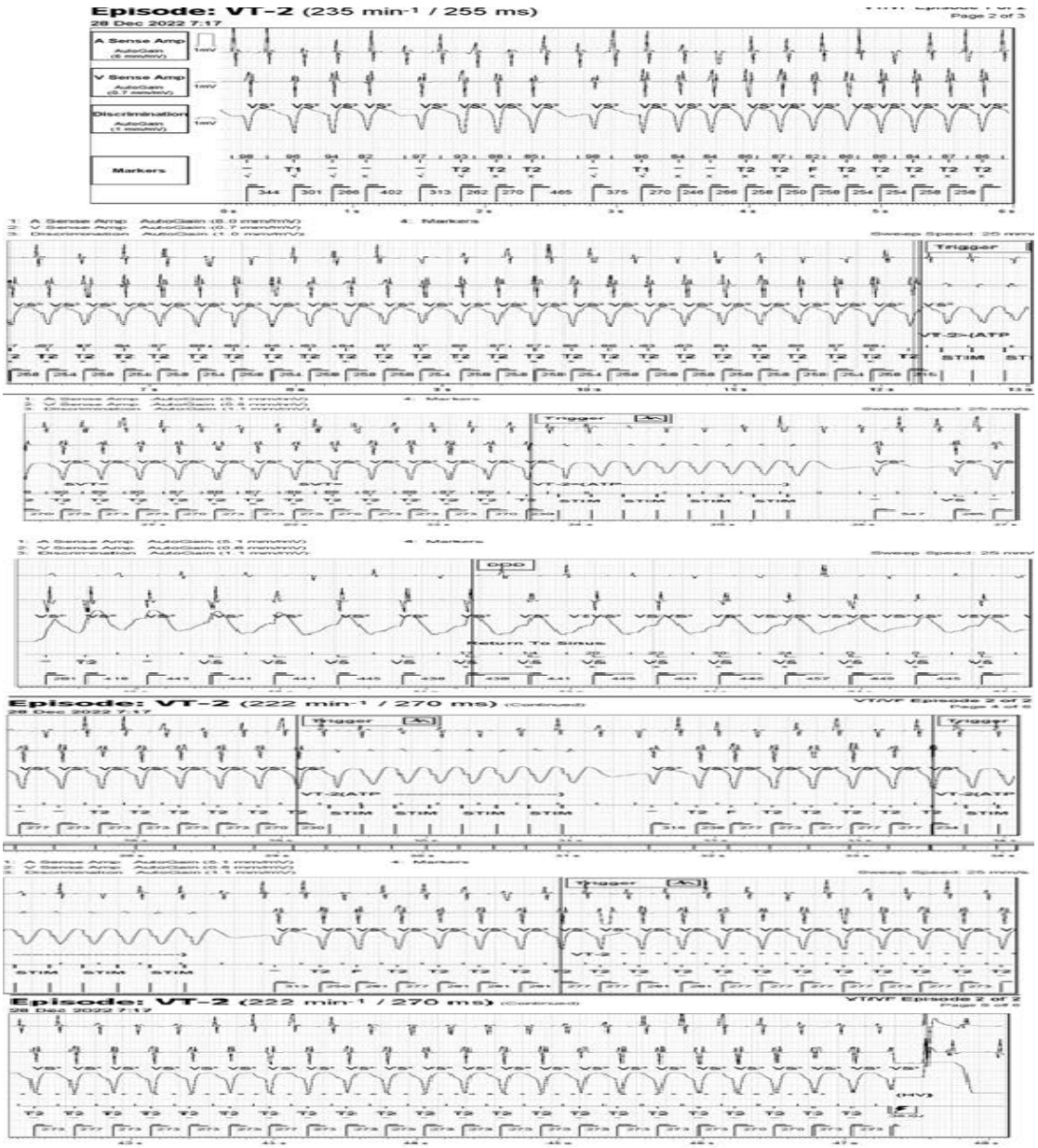

Figure 2.1. Device electrograms during tachycardia episodes illustrating dual arrhythmia mechanism. (A) Initial supraventricular tachycardia with atrial activity exceeding ventricular activity. (B) Transition to organized ventricular tachycardia with change in near-field electrogram morphology. (C-G) Failed antitachycardia pacing attempts followed by successful termination with ICD shock.

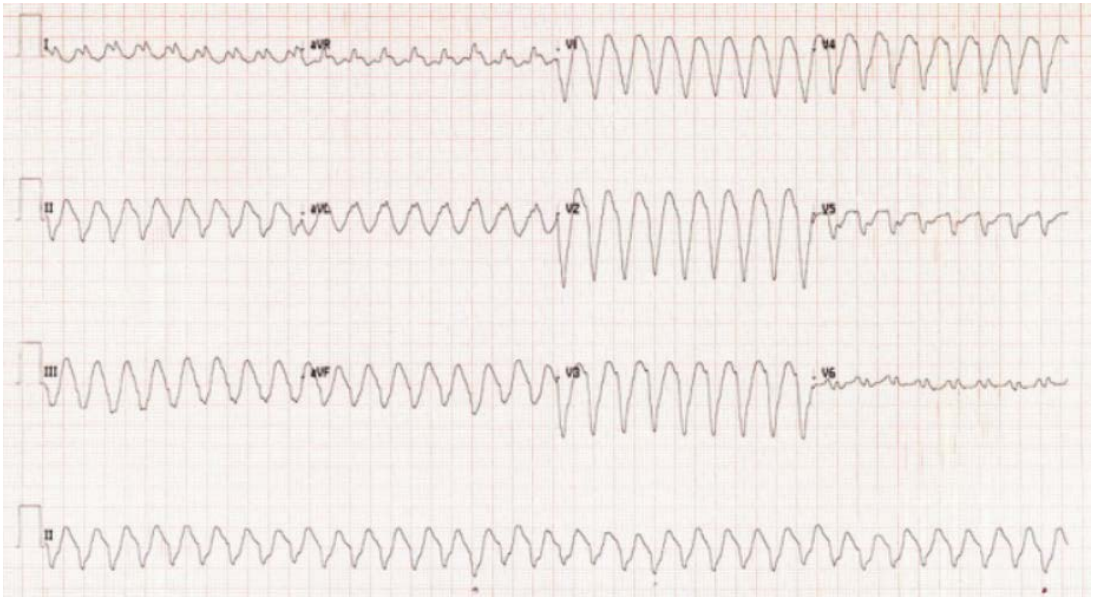

Figure 2.2. Monomorphic ventricular tachycardia, heart rate 210 bpm, LBBB morphology, Indeterminate axis in the inferior leads in keeping with mid-septal LV scar with a mid RV septal endocardial.

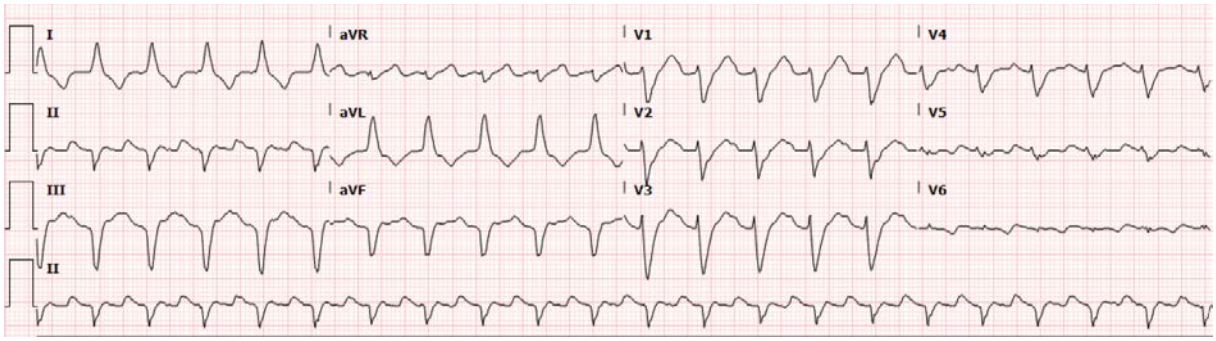

Figure 2.3. Left bundle branch block morphology, atrial tachycardia with an atrial cycle length of aproximately 450ms, QRS duration 180ms.

At the time of this admission, the patient was established on guideline-directed medical therapy for heart failure, including sacubitril/valsartan 24/26mg twice daily, dapagliflozin 10mg once daily, eplerenone 25mg once daily, bisoprolol 5mg twice daily, amiodarone 200mg once daily, and furosemide 40mg once daily.

The initial episode of ventricular tachycardia (VT) storm was managed with intravenous amiodarone, resulting in temporary stabilization and subsequent discharge following device interrogation and optimization.

However, within a few days of discharge, the patient was readmitted with further appropriate implantable cardioverter-defibrillator (ICD) therapies for recurrent sustained VT.

Once again, he required intravenous amiodarone infusion, which achieved rhythm stabilization.

At this stage, the patient exhibited advanced heart failure with New York Heart Association (NYHA) class IV symptoms and required ongoing management in the coronary care unit.

Although no further sustained ventricular arrhythmias were documented following treatment, he continued to experience a high burden of rapidly conducted atrial arrhythmias, with frequent transitions between atrial flutter and atrial tachycardia.

This resulted in suboptimal biventricular pacing and significantly impaired the effectiveness of cardiac resynchronization therapy, contributing to progressive haemodynamic compromise.

Optimisation of medical therapy was limited by persistent hypotension.

Consequently, sacubitril/valsartan was down-titrated to half the initial dose, and dapagliflozin was discontinued in order to facilitate up-titration of beta-blockade with bisoprolol, aiming to

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achieve better ventricular rate control during atrial arrhythmias.

Despite these adjustments, the patient's clinical status remained largely unchanged over the following month.

Functionally, the patient was profoundly limited, unable to transfer from bed to chair without experiencing severe dyspnoea, presyncopal or vasovagal episodes, and frequent short runs of paroxysmal atrial tachycardia.

Repeated attempts at device optimization failed to yield meaningful clinical or electrical improvement.

A redo atrial arrhythmia ablation was considered but deemed unlikely to be effective, particularly in the context of extensive atrial remodeling.

Furthermore, the patient's markedly elevated body mass index and associated comorbidities rendered any further complex electrophysiological intervention prohibitively high risk.

Extensive cardiac resynchronization therapy optimization was undertaken, with multiple programming strategies explored to improve electrical synchrony.

The intrinsic QRS duration, measured in lead I, was 203ms. Sequential left ventricular (LV) to right ventricular (RV) pacing with delays of 15ms, 20ms, and 40ms did not result in any improvement, with QRS duration remaining at 203ms.

Similarly, RV-to-LV pacing with a 10ms delay also failed to shorten the QRS duration.

Alternative configurations, including LV1-LV2 to RV pacing with delays of 15ms and 10ms, resulted in further QRS prolongation to 238ms.

Simultaneous LV and RV pacing yielded a QRS duration of 215ms.

Ultimately, the most favorable configuration was achieved using DDT mode with LV-to-RV delays of 15ms and 40ms, resulting in a reduction of QRS duration to 156ms.

Despite this relative electrical improvement, the high burden of atrial tachyarrhythmia remained the predominant limiting factor, preventing consistent delivery of effective biventricular pacing.

Given persistent severe symptoms, failure of medical therapy, and unsuccessful device optimization, the case was discussed in a multidisciplinary team (MDT) setting.

A consensus decision was made to proceed with urgent inpatient atrioventricular node (AVN) ablation, with consideration of additional cavotricuspid isthmus (CTI) line touch-up if required.

Ventricular tachycardia ablation was considered to carry an unacceptably high procedural risk in this context; therefore, continuation of amiodarone therapy was recommended for ongoing ventricular arrhythmia suppression.

The AVN ablation was performed during the same admission under anaesthetist-led sedation.

At the time of the procedure, the patient was in atrial flutter with a ventricular rate of approximately 100 beats per minute.

Venous access was obtained via the right femoral vein under ultrasound guidance; this was technically challenging due to body habitus but was achieved without complication, and long sheaths were utilized to facilitate catheter stability.

Ablation was delivered at the level of the compact atrioventricular node, resulting in prompt onset of complete heart block.

This was confirmed following an observation period, with no recovery of intrinsic atrioventricular conduction.

Assessment of the prior CTI ablation line was undertaken using differential pacing techniques, confirming persistent bidirectional block with a trans-isthmus conduction time of 190ms.

Following the procedure, the CRT device was reprogrammed to DDDR mode at 50-110 beats per minute, with a maximum tracking rate of 125 beats per minute, in the context of restoration of sinus rhythm and the expectation of complete ventricular pacing dependency.

Post AV node ablation electrocardiogram is illustrated in Figure 3.

The patient demonstrated marked clinical improvement following AV node ablation, with stabilization of haemodynamics and resolution of rapid atrial conduction.

Within a few days, he was successfully discharged from hospital with a significant improvement in symptoms.

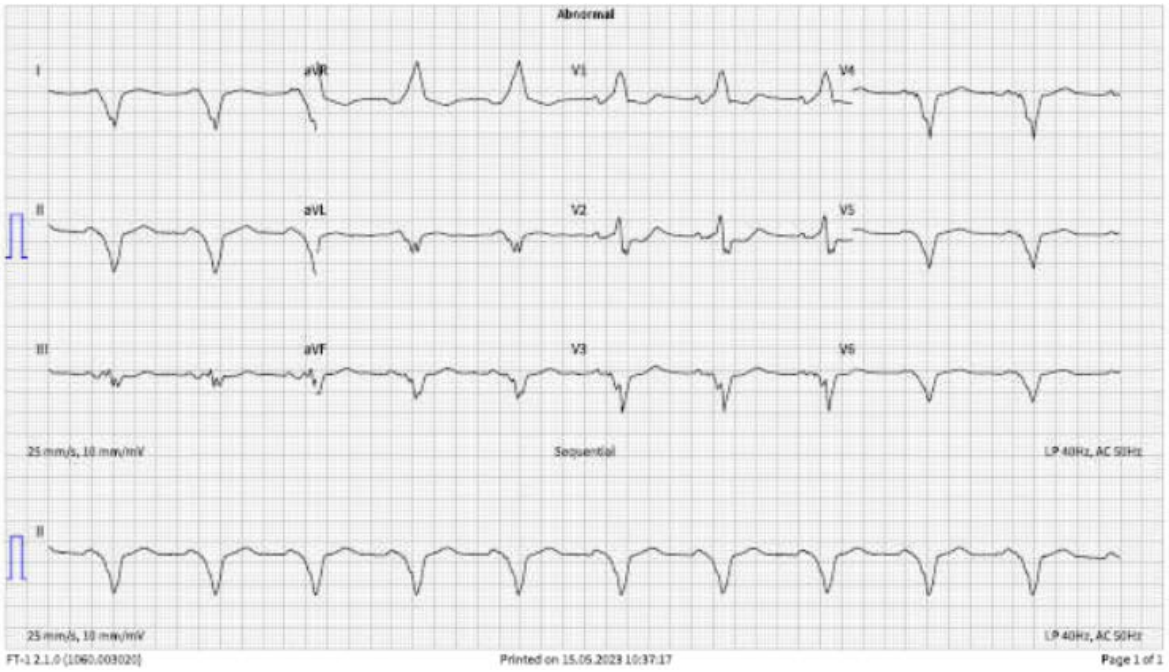

Figure 3. Electrocardiogram following atrioventricular node ablation demonstrating paced rhythm with improved ventricular synchrony.

A repeat transthoracic echocardiogram performed one month following atrioventricular node (AVN) ablation demonstrated a significant improvement in left ventricular systolic function, with the left ventricular ejection fraction (LVEF) increasing to 35-40%, compared to 15-20% prior to the intervention.

This was accompanied by a marked reduction in both left ventricular end-diastolic volume (LVEDV) and left ventricular end-systolic volume, consistent with favorable reverse remodeling.

These echocardiographic findings were paralleled by a substantial clinical improvement.

At one-month follow-up, the patient reported a notable increase in functional capacity, being able to ambulate approximately 100 yards, representing a significant improvement from his pre-procedural status.

However, repeat imaging also identified moderate-to-severe mitral regurgitation.

In light of this, an echocardiography-guided cardiac resynchronization therapy (CRT) optimization was undertaken.

This resulted in improved ventricular synchrony, with a corresponding reduction in the severity of mitral regurgitation to mild-to-moderate and further narrowing of the QRS complex, as illustrated in Figure 4.

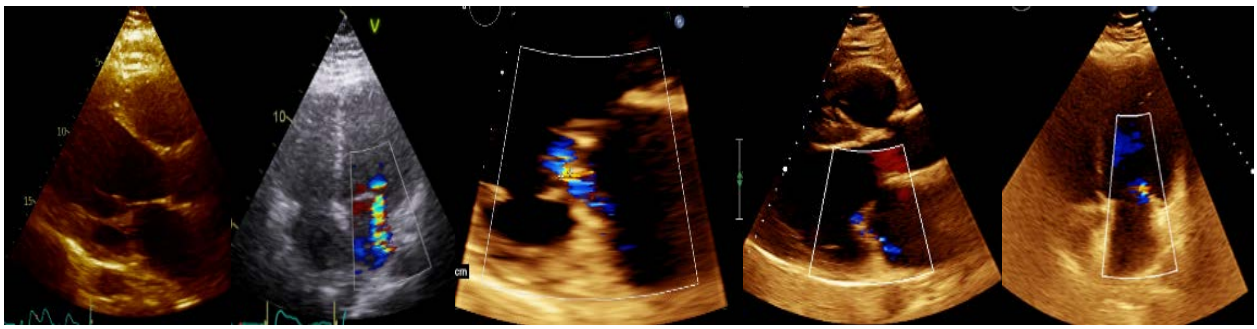

Figure 4. A: Parasternal long axis view and apical 4 chamber view showing moderate mitral regurgitation pre echo-guided CRT optimization. B: Parasternal short axis and apical 4 chamber view after echo-guided CRT optimization showing mild mitral regurgitation. Poor image quailities due to body habitus.

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Discussion

Evidence supporting the role of cardiac resynchronization therapy (CRT) in patients with left ventricular non-compaction cardiomyopathy (LVNC) remains limited but increasingly compelling.

In a study by Marco Bertini et al., CRT was associated with significant clinical and echocardiographic benefit in this subgroup, including greater degrees of left ventricular reverse remodeling, improvement in New York Heart Association (NYHA) functional class, and a meaningful increase in left ventricular ejection fraction (LVEF)[12].

These findings suggest that, despite the unique myocardial architecture and potential arrhythmogenic substrate in LVNC, CRT responsiveness may be preserved and, in selected patients, particularly pronounced.

This has important practical implications in cases where conventional transvenous left ventricular (LV) lead placement is unsuccessful or suboptimal.

In such scenarios, alternative strategies should be actively considered.

These include transseptal endocardial LV lead implantation, which may offer more physiological activation patterns, and surgical epicardial LV lead placement in anatomically challenging coronary venous systems.

More recently, conduction system pacing, including His bundle pacing and left bundle branch area pacing, has emerged as a safe and feasible alternative to traditional biventricular pacing.

As highlighted by Pugazhendhi Vijayaraman et al., conduction system pacing can achieve more physiological ventricular activation and may be particularly advantageous in patients with failed CRT or non-response to conventional approaches [13].

Similarly, Vivek Reddy and colleagues have demonstrated the feasibility and growing clinical adoption of these techniques in complex heart failure populations [14].

Beyond technical considerations, optimization of CRT requires a comprehensive and multidisciplinary framework.

As emphasized in the European Society of Cardiology guidelines, CRT response is not solely dependent on device implantation, but on a continuum of care involving appropriate patient selection, optimization of medical therapy, rhythm control strategies, and meticulous device programming.

A multidisciplinary team approach-integrating heart failure specialists, electrophysiologists, cardiac physiologists, imaging experts, and device specialists-is therefore essential to maximize therapeutic benefit [15,16].

In the present case, this integrated approach proved critical.

Despite initial non-response and clinical deterioration, systematic optimization-including arrhythmia control, device reprogramming, and ultimately atrioventricular node ablation-resulted in significant reverse remodeling and symptomatic improvement.

Conclusions

In patients with LVNC and advanced heart failure, CRT optimisation can be particularly challenging, especially in the presence of atrial arrhythmias and morbid obesity.

Atrioventricular node ablation plays a pivotal role in ensuring effective CRT delivery and can result in significant clinical and echocardiographic improvement when conventional optimization strategies fail.

A multidisciplinary approach is essential to achieve optimal outcomes in such complex cases.

Acknowledgement

None to declare.

Author Contribution

Arsalan Farhangee managed the patient, conceived the report, performed the literature review, and drafted the manuscript.

Ion Mindrila supervised the work, critically revised the manuscript, and approved the final version as the corresponding author. Both authors read and approved the final manuscript.

Conflicts Of Interest

No conflict of interest.

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Consent Statement

A written informed consent was obtained from the patient, prior to preparing this manuscript for publication.

Data Availability

Data available on reasonable request.

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