Case Report

Reverse left ventricular remodeling reversed left bundle branch block: A case report

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ABSTRACT

Left bundle branch block (LBBB) is frequently associated with cardiomyopathy and is generally considered irreversible, often prompting cardiac resynchronization therapy. However, emerging evidence suggests that optimal medical therapy may lead to reverse remodeling and resolution of LBBB. A 60-year-old female with heart failure presented with severe left ventricular (LV) dysfunction (ejection fraction 25%) and LBBB. Coronary angiography was normal. Cardiac magnetic resonance showed no fibrosis, and genetic testing was unremarkable. Guideline-directed medical therapy was initiated. Over 9 months, serial evaluations demonstrated progressive improvement in LV volumes and systolic function. Notably, LBBB resolved spontaneously, with a normalization of QRS duration. This case illustrates that reverse LV remodeling with optimal medical therapy may be accompanied by resolution of LBBB in non-ischemic cardiomyopathy. These findings highlight the dynamic relationship between myocardial recovery and electrical conduction and may have implications for the timing of device therapy.

Key words: Electrocardiography, Guideline-directed medical therapy, Left bundle branch block, Reverse left ventricular remodeling

Left bundle branch block (LBBB) is a common conduction disorder that is found frequently in patients with cardiomyopathy and left ventricular (LV) dysfunction, and it is associated with severe adverse cardiac events and increased morbidity and mortality [1]. While LBBB is most commonly considered an irreversible state requiring interventions such as cardiac resynchronization therapy (CRT) [2], particularly in non-ischemic cardiomyopathy (NICM), aggressive heart failure treatment can lead to marked reverse LV remodeling and indeed spontaneous resolution of LBBB [3-5]. Recent clinical reports have highlighted the potential for delayed spontaneous resolution of LBBB following successful medical treatment, emphasizing the dynamic nature of cardiac remodeling in this patient population [3-5]. The mechanisms underlying such changes are multifaceted, with both direct effects on myocardial structure and indirect impact on the cardiac conduction system [6].

This case report contributes to the growing body of evidence that effective heart failure management may not only restore ventricular function but also normalize conduction in some patients with NICM, challenging the traditional view of LBBB as an irreversible condition.

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CASE REPORT

A 60-year-old female patient with heart failure was referred to the Heart Function Clinic for further evaluation in early 2024. She was diagnosed with breast cancer in 2013 and underwent a lumpectomy followed by chemotherapy and radiotherapy. She has rheumatoid arthritis and was on methotrexate. She had no history of hypertension, diabetes mellitus, dyslipidemia requiring treatment, thyroid dysfunction, chronic kidney disease or uremia, known coronary artery disease, or significant valvular heart disease. She did not report alcohol abuse, recreational drug use, or exposure to other cardiotoxic medications. There was no family history of cardiomyopathy or premature sudden cardiac death. Her heart failure did not occur in relation to pregnancy or the early postpartum period, and there was no prior episode consistent with stress-induced (Takotsubo) cardiomyopathy.

Her initial echocardiogram (Echo) in January 2024 showed an LV end-diastolic volume index (LVEDVi) of 127.2 mL/m^2 and an ejection fraction (EF) of 25% (Table 1). An electrocardiogram (ECG) revealed sinus rhythm with LBBB (Fig. 1a). Following guideline-based treatment and normal coronary angiography results, her

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Table 1: The changes in patients' left ventricular size and function based on echocardiographic measurements over months

Date	LVEDD (cm)	LVEDV (mL)	LVEDVi (mL/m²)	LVESD (cm)	LVEF (%)
Jan 2024	6.7	208.7	127.2	5.6	25
May 2024	6.7	189.6	113.4	5.5	23
Sep 2024	6.2	147.1	88.6	4.9	35
Apr 2025	4.2	110.5	66.3	3.4	37

LVEDD: Left ventricular end-diastolic diameter, LVESD: Left ventricular end-systolic diameter, LVEDV: Left ventricular end-diastolic volume, LVEDVi: Left ventricular end-diastolic volume index, LVEF: Left ventricular ejection fraction

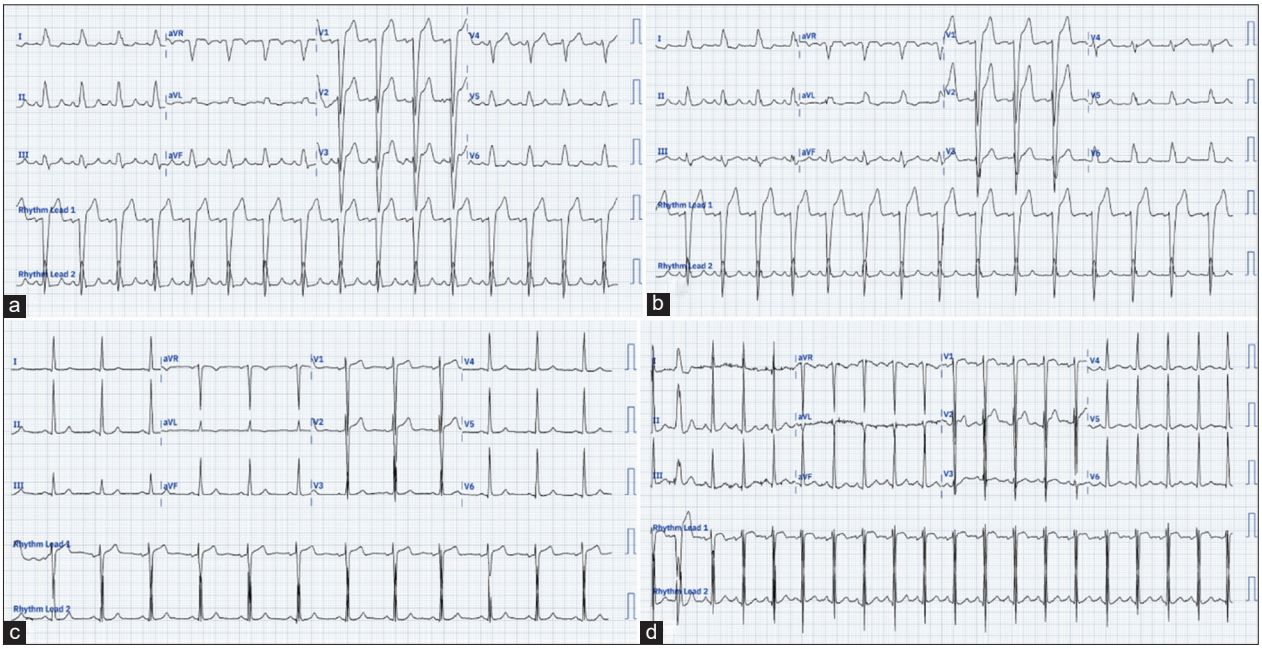


Figure 1: Electrocardiogram (ECG) in (a) (February 2024) and (b) (May 2024) show sinus rhythm with left bundle branch block. ECG in (c) (September 2024) shows sinus rhythm with resolved left bundle branch block. ECG in (d) (April 2025) is compatible with sinus rhythm and a narrow QRS complex

condition was monitored. Her second Echo in May 2024 showed improvement in LVEDVi without a discernible change in EF (Table 1), and her ECG was compatible with LBBB (Fig. 1b). Clinical improvement was noted, and medications were adjusted accordingly. In September 2024, her Echo showed significant improvement in both LVEDVi and EF (Table 1), and her ECG demonstrated resolution of LBBB (Fig. 1c). Her final Echo in April 2025 revealed further improvement in LV size and function (Table 1), (Figure 2). Moreover, the interventricular septal and posterior wall thicknesses were both 11 mm, indicating no significant hypertrophy. The left atrial volume index was 34 mL/m², suggesting only mild residual atrial enlargement, and the average mitral E/E' ratio was 14, compatible with borderline-elevated filling pressures in the context of substantially improved systolic function.

Her last ECG was in sinus rhythm and still had a narrow QRS (Fig. 1d). Since her second clinic visit, she has been maintained on spironolactone 25 mg, bisoprolol 5 mg, and sacubitril/valsartan 24/26 mg. Given her history of recurrent urinary tract infections, initiation of a sodium-glucose co-transporter 2 inhibitor was deferred. In addition, up-titration of sacubitril/valsartan was limited by borderline blood pressure. Three months after initiating guideline-directed medical therapy (GDMT), the decision was made to proceed with CRT-D implantation; however, the device was not implanted as the patient declined. Her functional capacity improved from New York Heart Association Class III to Class II during her treatment course and follow-up. There was no fibrosis or scar visible on cardiac magnetic resonance imaging. The results of a pan-cardiac genetic screening showed insignificant variance.

DISCUSSION

LV reverse remodeling (LVRR) refers to the process whereby the heart undergoes structural and functional improvements. It is generally evidenced by chamber volume reduction, normalization of ventricular geometry, and improvement of systolic and diastolic function [3,6]. Although LVRR can occur spontaneously, it is more commonly observed following therapeutic interventions that diminish the precipitating stressors or pathophysiological mechanisms of heart failure, such as hemodynamic overloading and abnormally elevated levels of neurohormonal activation [3].

LBBB is an abnormality of conduction that is widely associated with adverse cardiac remodeling and poor prognosis. LBBB leads to ventricular dyssynchrony that, in turn, worsens heart performance and can induce progression of cardiomyopathy [1]. In the past, LBBB in NICM was traditionally considered irreversible, as the conduction defects and myocardial dysfunction

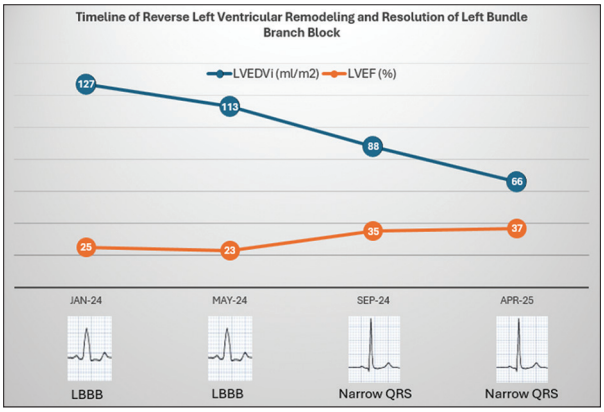


Figure 2: Timeline of reverse left ventricular remodeling and resolution of left bundle branch block

often worsen each other [2,4]. Nevertheless, there are reported cases that demonstrate both normalization of the QRS complex and improvement in cardiac function in conjunction with reverse remodeling, either naturally or as a result of optimized medicinal therapy [5,4,7]. This is an uncommon phenomenon but highlights the dynamic interaction between the cardiac conduction system and the myocardial structure. Resolution of LBBB in these patients is likely due to recovery of myocardial structure and function, which may alleviate the mechanical and electrical substrate for conduction delay [4,7].

Reversible etiologies such as electrolyte imbalances and metabolic disorders should be considered and excluded in the evaluation of LBBB [8]. No electrolyte abnormalities or other metabolic disturbances were identified as potential reversible causes of LBBB in our patient during the course of her evaluation. Because the patient had run out of bisoprolol 1 day before presentation, her heart rate on the most recent narrow QRS ECG was higher than that observed on prior ECGs demonstrating LBBB (Fig. 1d). This finding excludes a rate-dependent mechanism for the bundle branch block.

Several alternative etiologies of LV dysfunction warrant consideration in this patient. Ischemic cardiomyopathy is unlikely given normal coronary angiography, the absence of regional wall-motion abnormalities, and a pattern of global systolic impairment. Infiltrative or inflammatory cardiomyopathies are also less probable, as cardiac magnetic resonance imaging (MRI) showed no fibrosis, edema, or late gadolinium enhancement, and there were no systemic features to suggest amyloidosis, sarcoidosis, or myocarditis. Significant valvular or hypertensive heart disease was not present on echocardiography or clinical evaluation. The patient's remote history of breast-cancer therapy raises the possibility of chemotherapy-induced cardiomyopathy. Anthracyclines and trastuzumab are known to cause LV dysfunction, often within months to a few years after exposure, and may exert cumulative, partially irreversible effects [9]. In our case, the long latency of approximately 11 years, absence of myocardial fibrosis on cardiac MRI, normal coronary angiography, and substantial reverse remodeling with near-complete EF recovery argue against dominant, fixed chemotherapy-induced injury.

The absence of major systemic, metabolic, and lifestyle risk factors for cardiomyopathy – including diabetes, hypertension, thyroid or renal dysfunction, and alcohol or substance abuse – together with a clinical history that does not fulfill criteria for peripartum cardiomyopathy and lacks any acute stress-related, transient wall-motion pattern typical of takotsubo cardiomyopathy, further supports the classification of this case as NICM with LBBB-related dyssynchrony rather than cardiomyopathy secondary to other common extracardiac or specific clinical causes.

Spontaneous resolution of non-rate-dependent LBBB with concomitant recovery of LV function has been described only in a small number of case reports, underscoring the rarity of this phenomenon. Our case adds several distinctive elements: invasive coronary evaluation and cardiac MRI excluded ischemic and fibrotic substrates; comprehensive genetic testing did not identify a heritable cardiomyopathy; and reverse remodeling occurred under low-to-moderate doses of GDMT without CRT, with LBBB resolution temporally aligned with LV recovery. These features strengthen the concept that, in selected NICM patients, optimized medical therapy alone may reverse both mechanical and electrical remodeling.

CONCLUSION

This case adds to the emerging evidence that reverse LV remodeling can be correlated with spontaneous resolution of LBBB, particularly in patients with NICM. The relationship between structural and electrical remodeling demonstrates that, in appropriate patients, combined heart failure therapy not only improves ventricular performance but also normalizes conduction.

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