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Understanding Painful Left Bundle Branch Block Syndrome: Where Does the Evidence Lead Us?

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Introduction

Painful left bundle branch block (LBBB) syndrome is an uncommon and often under-recognised clinical entity with no available epidemiological data to define its prevalence in the United Kingdom (UK) or elsewhere. Although first described in 1976 (1), it remains a diagnostic challenge in conventional chest pain evaluation. Growing case series, advances in imaging, and the emergence of physiological pacing have renewed interest in this syndrome, providing insights into its mechanisms and therapeutic options. Nonetheless, key uncertainties persist, underscoring the need to synthesise current evidence.

Take Home Messages

- Painful left bundle branch block (LBBB) syndrome is a rare, often misdiagnosed cause of chest pain.
- Case series show limited benefit of pharmacologic therapy, whereas conduction-system pacing provides consistent symptom relief.
- Electromechanical dyssynchrony appears central to its mechanism.
- However, data remain observational. Larger, systematic investigations are needed to clarify mechanisms, validate diagnostic criteria, compare pacing strategies, and optimise long-term outcomes.

What is Painful LBBB Syndrome?

The defining feature is abrupt onset of chest pain temporally linked to the appearance of LBBB, in the absence of objective myocardial ischaemia. Symptoms begin and resolve within seconds of LBBB initiation and termination, distinguishing it from typical angina (2).

Mechanistic Concepts: What Might Explain the Pain?

Although the precise mechanism remains incompletely understood, several complementary hypotheses have emerged. The syndrome is associated with conduction-threshold phenomenon, in which abrupt left bundle refractoriness leads to sudden LBBB onset (2,3). The primary driver appears to be abrupt electromechanical dyssynchrony: LBBB delays lateral-wall activation relative to the septum and right ventricle, producing septal flash, apical rocking, and inefficient contraction (4–6). Dyssynchrony may also transiently impair septal subendocardial perfusion through elevating filling pressures and redistributing myocardial blood flow. This reflects the functional, reversible perfusion abnormalities rather than fixed epicardial coronary disease (7) (Figure 1).

Symptoms are reproducibly triggered by abrupt changes in ventricular activation, highlighting the role of activation pattern rather than absolute heart rate (2). A secondary neurogenic mechanism may amplify pain: altered wall stress and cardiac afferent signalling engage central pathways, including the insula and anterior cingulate cortex, enhancing symptom perception (2,8,9).

Approach to Diagnosis

Diagnosis of painful LBBB syndrome requires a clear temporal association between episodic LBBB and chest pain, alongside exclusion of myocardial ischemia (2). Functional evaluation via stress testing or coronary imaging should show no myocardial ischemia, with invasive angiography reserved for high-risk or ambiguous cases. Transient microvascular or subendocardial perfusion abnormalities may occur during LBBB due to electromechanical dyssynchrony, but these changes are reversible. Symptoms begin abruptly with LBBB onset and resolve upon restoration of narrow QRS conduction. New-onset LBBB features a low precordial S/T ratio (<1.8) (Figure 2) and inferior QRS axis (2). Baseline and inter-episode ECGs are usually normal, though T-wave inversion reflecting cardiac memory may occur. Left ventricular function is generally preserved, although rare non-ischemic cardiomyopathy cases suggest conduction-dyssynchrony rather than structural mechanism (5).

Adjunct testing can strengthen diagnostic confidence. Exercise or ambulatory monitoring may reproduce rate-related LBBB and symptoms. In selected cases, controlled pacing or electrophysiology study reproduces LBBB-associated pain (2,5,10), with symptom relief upon

correction of conduction (e.g., via conduction-system pacing), supporting a conduction-dyssynchrony mechanism.

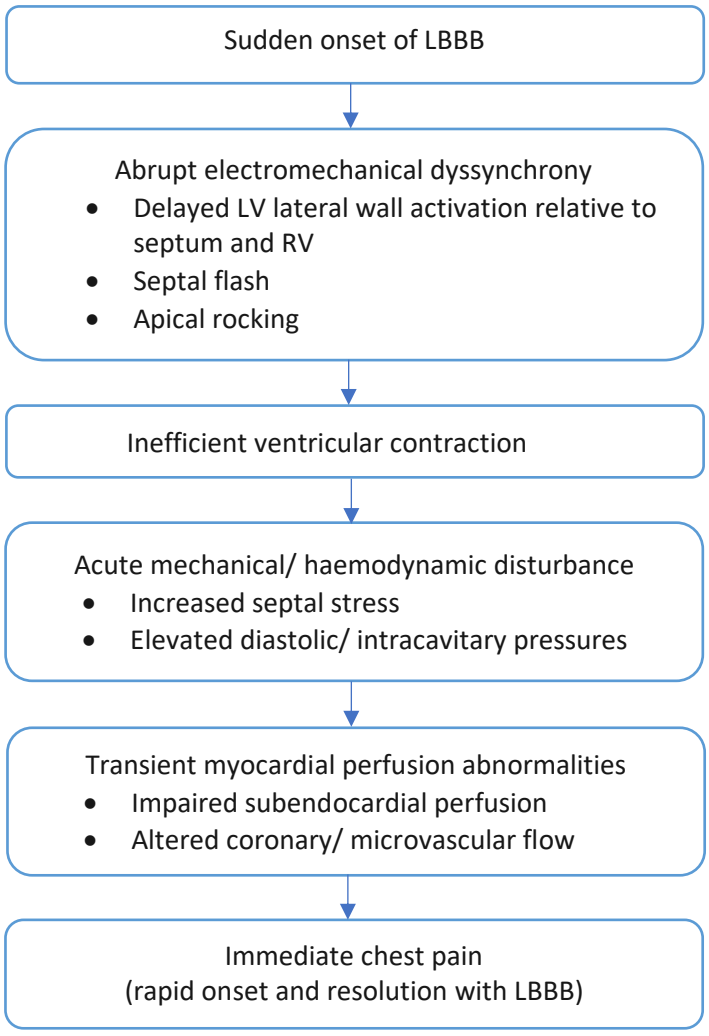

Figure 1. Proposed mechanism of painful LBBB syndrome: abrupt electromechanical dyssynchrony leads to parallel downstream perfusion abnormalities and mechanically mediated functional myocardial ischaemia, distinct from epicardial coronary artery disease.
LBBB, Left bundle branch block; LV, Left ventricle; RV, Right ventricle.

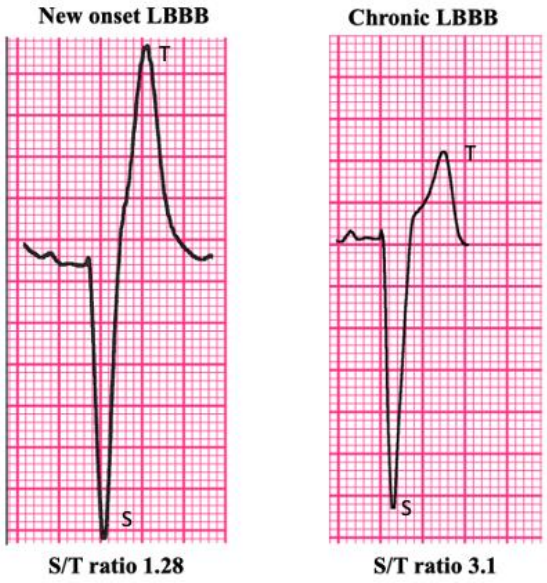

Figure 2. New onset versus chronic LBBB. New-onset LBBB is characterised by a low S/T ratio (<1.8). Lead V3, 25 mm/s, 10 mm/mV (adapted from Shvilkin et al. (3).

Therapeutic Options: What Does the Evidence Suggest?

Management of painful LBBB syndrome is guided primarily by case reports and small observational series, but several therapeutic patterns are consistent.

Medical Therapy

For patients with preserved systolic function and rate-dependent LBBB, betablockers or non-dihydropyridine calcium-channel blockers are commonly used to avoid the heart-rate threshold at which LBBB emerges (2,5). Their effectiveness, however, is limited and there are no standardised dosing schedules. In a pooled analysis of 45 published cases (11), only 33% improved with initial medical therapy, with high non-response rates to betablockers (88%) and antianginals (83%). These findings suggest that rate control alone is frequently inadequate, particularly when LBBB occurs at low heart rates.

Pacing Therapy

Pacing has emerged as the most consistently effective option for severe or refractory symptoms (2,5,11) (Table 1). In 21 reported cases, whether as first-line therapy (n=9) or after medical failure

(n=12), nearly all patients achieved substantial or complete symptom resolution, including 12 of 13 previously refractory patients (5,11). Pacing strategies have included right-ventricular, biventricular, and, more recently, conduction-system approaches such as His bundle pacing (HBP) and left bundle branch area pacing (LBBAP). Conduction-system pacing (CSP) effectively restores physiologic His–Purkinje conduction, correct ventricular dyssynchrony and rapidly relieve symptoms. Contemporary case reports (2018-2025) consistently describe immediate and sustained symptom relief (3,5,10–13).

Table 1. Summary of Reported Therapeutic Approaches and Response Rate in Painful LBBB Syndrome.

Treatment Strategy	No. of Patients	Response Rate	Notes
Betablockers / CCB	25	12%	First-line medical therapy; limited efficacy
Antianginals	6	17%	Often refractory; adjunctive use
Pacemaker (all types)	21	95–100%	Includes RV pacing, biventricular pacing, HBP, LBBAP; most effective for refractory cases
Other strategies	8	Variable	Exercise, amiodarone, betablocker + antianginal, chloroquine discontinuation, phlebotomy; patient-specific

Abbreviations: CCB, Calcium channel blocker; RV, Right ventricle; HBP, His bundle pacing; LBBAP, Left bundle branch area pacing.

Other Approaches

Less common strategies (e.g., exercise training, treatment of contributing conditions, medication adjustments) have been reported in isolated cases but show variable efficacy and are generally adjunctive (11).

What Remains Uncertain?

Key knowledge gaps persist despite expanding clinical recognition. The epidemiology of painful LBBB is unclear, and proposed diagnostic criteria lack formal validation. The precise electrophysiological mechanism underlying pain perception during LBBB is still debated. Current

therapeutic evidence, particularly conduction system pacing, is limited to small case series without controlled comparative data. Long-term outcomes, optimal patient selection, and the role of adjunctive testing require further investigation.

Conclusions

Painful LBBB syndrome is challenging due to its under-recognition and the absence of standardised diagnostic or therapeutic pathways. Available evidence indicates that pharmacologic therapy benefits only a minority of patients, whereas pacing particularly conduction system pacing provides more consistent and substantial symptom relief. However, the heterogeneity of pacing approaches underscores the need for individualised treatment and well-designed studies to establish evidence-based recommendations.

Disclosures

None

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