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Painful Left Bundle Branch Block Syndrome Successfully Treated With Left Bundle Branch Area Pacing

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ABSTRACT

Chest pain may be rarely associated with left bundle branch block (LBBB)-mediated ventricular dys-synchrony has been reported. This article reports 2 such cases, where left bundle branch area pacing resulted in resolution of the LBBB and associated symptoms. By adjusting the atrioventricular delays, the QRS duration was narrowed further by achieving fusion with the intrinsic activation wavefront. (**Level of Difficulty: Beginner.**) (J Am Coll Cardiol Case Rep 2020;2:568–71) © 2020 The Authors. Published by Elsevier on behalf of the American College of Cardiology Foundation. This is an open access article under the CC BY-NC-ND license (http://creativecommons.org/licenses/by-nc-nd/4.0/).

Chest pain attributed to left bundle branch block (LBBB) in the absence of ischemia is a rare clinical entity with <60 cases reported (1). The exact mechanism is unclear, but the hypothesis of LBBB-mediated dyssynchrony and alteration of activation wavefront in the ventricles resulting in nonspecific symptoms of chest pain, fatigue, and worsening exercise capacity has been postulated (2). Symptoms may vary from self-limiting discomfort to severe pain with exertion or even at rest. Often these patients are misdiagnosed for years resulting in extensive evaluation for their chest pain including stress tests, coronary angiograms, cardiac magnetic resonance, and so forth. The present study is the first to report 2 cases of painful LBBB syndrome treated successfully with left bundle branch area pacing (LBBAP).

LEARNING OBJECTIVES

- Painful LBBB syndrome should be considered as a possible differential in patients with chest pain and LBBB after ischemia has been ruled out.
- These patients have conventionally been treated with limited efficacy using exercise regimens, atrioventricular nodal blockers, or right ventricular or biventricular pacemakers.
- Conduction system pacing using His bundle pacing and left bundle branch area pacing is a feasible and effective treatment modality for these patients.

CASE 1

PRESENTATION. A 63-year-old woman with no known cardiac risk factors had worsening chest pain with exercise for the past 4 years.

INVESTIGATIONS. She underwent an extensive workup including a nuclear stress test and cardiac catheterization that ruled out obstructive epicardial coronary artery disease. CMR was unremarkable. Her baseline electrocardiography showed normal sinus rhythm at 62 beats/min with a narrow QRS duration.

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She was taking high-dose diltiazem, 360 mg daily, for possible microvascular disease. Due to refractory exertional chest pain, she underwent a repeated exercise stress echocardiogram. She complained of chest pain with the appearance of a new LBBB at faster heart rates (above 75 beats/min) during exercise that resolved completely within minutes of resolution of LBBB at rest (below 60 beats/min), confirming the diagnosis of painful LBB syndrome. Eventually, the chest pain and the LBBB became persistent even at rest (Figure 1A).

MANAGEMENT. She underwent a dual-chamber pacemaker implantation with ventricular lead in an LBBA position. The His bundle region was mapped for a His potential by using a C315 His delivery sheath (Medtronic, Dublin, Ireland) and a 3830 lead (Medtronic). The His bundle-ventricular interval (HV interval) was 62 ms. The sheath was then advanced approximately 1.5 to 2 cm distally along the septum in the right ventricle. Pacing at this site revealed a "notch" in the descending limb of the paced QRS complex in lead V_1. The R-wave in lead II was taller than that in lead III, aVR was negative, and aVL was positive, suggestive of para-Hisian location. With the sheath oriented perpendicularly to the septum (confirmed in right anterior oblique and left anterior oblique views), rapid clockwise turns were given. Pacing impedance and QRS morphology were monitored carefully. After 6 to 8 turns, a rSr′ pattern was seen in lead V_1, along with stimulation artifact to QRS latency of 26 ms. Stim to peak of R-wave in V_5 was 60 ms at high- and low-outputs when pacing unipolar from the tip electrode. In addition, a retrograde LB potential was seen 40 ms after the initiation of QRS complex during sinus rhythm (Figure 1B). These findings suggested selective LBBA capture. The septogram (1 to 2 ml of contrast using the His sheath) revealed that the lead was approximately 1.1 cm deep in the septum (Supplemental Figure 1). The paced QRS duration was 110 ms. By adjusting the AV delays, the QRS duration was normalized (80 ms) by facilitating fusion of the native impulse conducting anterogradely over the right bundle with the anterograde activation of the left bundle branch by pacing (Figure 1C, Supplemental Figure 3A). On the LBB pacing lead, R waves were

ABBREVIATIONS AND ACRONYMS

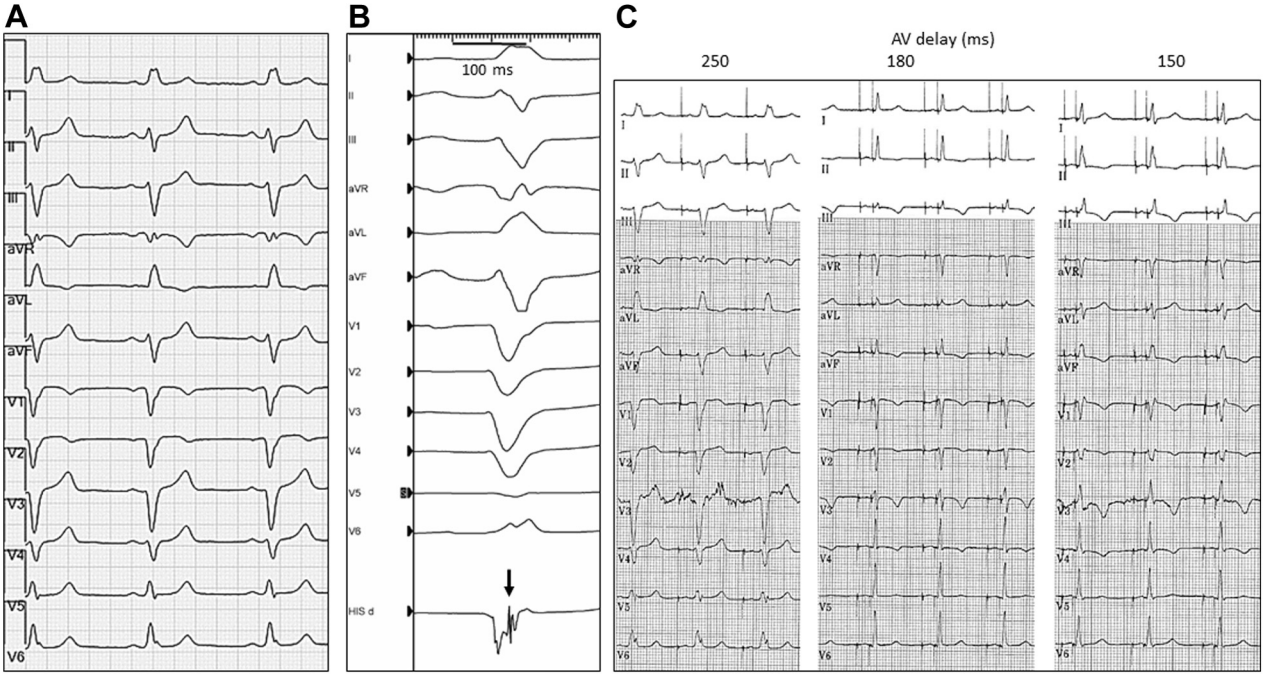
AV = atrioventricular

HBP = His bundle pacing

LBBAP = left bundle branch area pacing

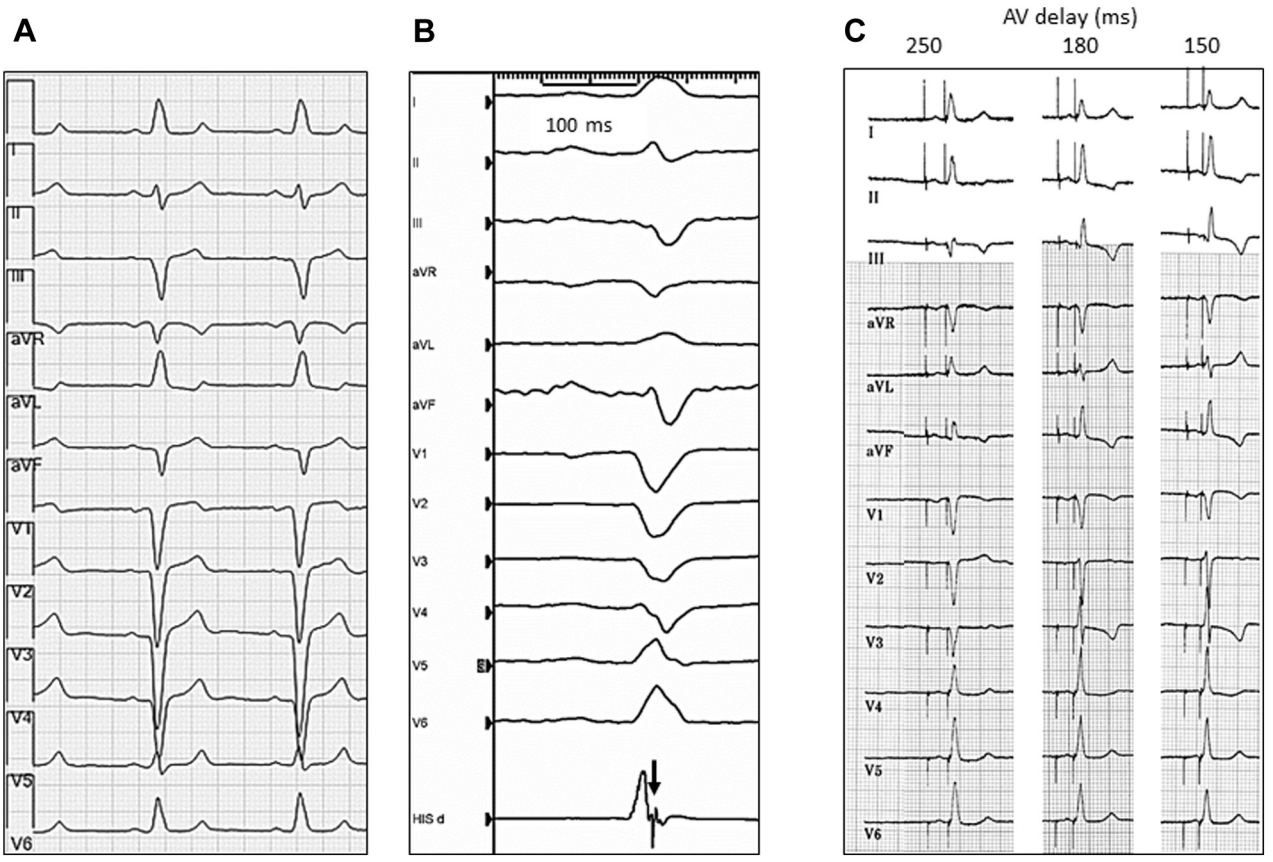
LBBB = left bundle branch block

FIGURE 1 Baseline ECG With Normal Sinus Rhythm, LBBB, and QRS Duration of 140 ms



(A) Baseline electrocardiography (ECG) with normal sinus rhythm, left bundle branch block (LBBB), and QRS duration of 140 ms. **(B)** Retrograde left bundle branch potential **(arrow)** seen 40 ms after QRS initiation. **(C)** Narrowing of QRSd to 80 ms with AV delay of 180 ms due to progressive fusion with shortening of atrioventricular (AV) delay. Further shortening causes selective left bundle branch area pacing with rSr′ in V_1.

FIGURE 2 Baseline ECG With LBBB and QRS Duration of 132 ms



(A) Baseline ECG with LBBB and QRS duration of 132 ms. **(B)** Retrograde left bundle branch potential **(arrow)** seen 46 ms after QRS initiation **(C)** Narrowing of QRSd to 104 ms with AV delay of 150 ms due to progressive fusion with shortening of AV delay. Further shortening causes nonselective LBBA pacing with qR in V_1. Abbreviations as in Figure 1.

measured at 8 mV, pacing impedance was 807 Ω, and capture threshold was 0.7 V at 0.5 ms. After the procedure, the patient was blinded to pacing, and she had repetitive reproducible chest pains when pacing was turned off.

FOLLOW-UP. The patient did extremely well, and her chest pain resolved at rest and with exertion. The capture threshold in the LBB lead remained stable at 0.75 V at 0.4 ms at 6 months.

CASE 2

PRESENTATION. A 71-year-old man with a medical history of coronary artery disease, prior percutaneous coronary stents, and paroxysmal atrial fibrillation presented with intermittent exertional chest pain and shortness of breath. Coronary angiogram showed patent stents. Pain was refractory to antianginal medications (amlodipine, metoprolol, and nitroglycerine, as needed).

INVESTIGATIONS. The patient was noted to have an LBBB with QRS duration of approximately 132 ms (Figure 2A). Initially his symptoms were intermittent but later progressed to constant. After extensive work-up and excluding potential causes of chest pain, painful LBB syndrome was diagnosed.

MANAGEMENT. He underwent implantation of dual-chamber pacemaker with ventricular lead in LBBA position. A C315 His sheath and 3830 lead (Medtronic) were used to map the His bundle region. The HV interval was 70 ms. The lead was implanted in the LBBA in a fashion similar to that described in the previous case. After insertion of the lead to approximately 1.4 cm into the septum based on the septogram, a qR pattern was seen in V_1 on unipolar pacing

from the tip electrode (Supplemental Figure 2). In addition, a retrograde LB potential was seen 46 ms after the initiation of QRS complex during sinus rhythm (**Figure 2B**). In lead V_5, stim to peak of R-wave was 73 ms with high- and low-output pacing. All these features suggested a selective LBBA capture. AV delays were adjusted to achieve fusion with intrinsic activation wavefront conducting anterograde over the right bundle branch with the LBB activation by pacing the LBBA (**Figure 2C**), thus normalizing the QRS morphology and duration. Final QRS duration was 110 ms (Supplemental Figure 3B).

FOLLOW-UP. The LBB pacing lead threshold was 0.3 V at 0.5 ms and remained stable at 0.5 V at 0.4 ms at 1-month follow-up. The patient did very well after implantation and has not had any recurrence of his symptoms in 5 months.

DISCUSSION

Various treatment modalities have been used to alleviate symptoms in patients with painful LBB syndrome. Favorable results of a physical exercise regimen and a blunting heart rate response to exercise with AV nodal blockers have been reported (3,4). Right ventricular pacing, biventricular pacemaker implantation, and more recently, cases of resolution of LBBB and associated symptoms with His bundle pacing (HBP) have been reported (1,5,6,7). Routine use of HBP may be limited due to higher capture thresholds at implantation and increased risk for late rise in HBP thresholds, necessitating lead revisions (8). Recent studies by Vijayaraman et al. (9) and Zhang et al. (10) demonstrated that LBBAP can significantly narrow the QRS duration in patients with LBBB. To the best of the present authors' knowledge, these are the first cases with painful LBBB that were successfully treated with LBBAP leading to complete resolution of the LBBB, narrowing of QRS, and alleviation of symptoms. Conduction system pacing offers an attractive treatment modality for patients with painful LBB syndrome. LBBAP may circumvent the sensing- and threshold-related issues seen with HBP and hence may be the preferred method of pacing in such patients. Long-term studies are warranted to understand the best pacing modality in painful LBB patients.

CONCLUSIONS

Painful LBB syndrome is an underrecognized entity and should be suspected in patients with chest pain and LBBB in the absence of ischemia. Painful LBB syndrome may coexist in patients with coronary artery disease. Ventricular dyssynchrony due to LBBB is likely the cause of chest pain. Conduction system pacing (His bundle or LBBA) may result in complete resolution of LBB and associated symptoms.

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KEY WORDS cardiac pacemaker, chest pain, cardiac resynchronization therapy

APPENDIX For supplemental figures, please see the online version of this paper.