

Transient left bundle branch block due to massive increase of His bundle pacing threshold associated with acute heart failure in a patient with complete heart block



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Introduction

Transient left bundle branch block (LBBB) may occur in association with high left ventricular (LV) filling pressure and/or global systolic LV dysfunction in patients with heart failure.¹ We herein present a patient who had undergone permanent His bundle pacing (HBP) for complete infranodal heart block and developed transient LBBB because of a massive increase in the His bundle (left bundle) pacing threshold [ie, HB(LB) pacing threshold] associated with acute heart failure.

Maintenance of electrical synchrony with recruitment of LBBB at higher pacing output might have led to prompt myocardial recovery and improvement of the HBP threshold.

Case report

An 81-year-old man with no remarkable medical history presented for evaluation of upper abdominal pain. He was a previous smoker. He had no history of chest pain, palpitation, or syncope. His blood pressure was 120/80 mm Hg, and his heart rate was 46 beats per minute. Laboratory examination findings, including cardiac enzyme levels, were normal. An electrocardiogram (ECG) showed complete heart block (CHB) with right bundle branch block and left anterior fascicular block morphology escape rhythm (Figure 1A). Precordial abnormal Q waves were also noted, suggesting silent myocardial infarction (MI). An echocardiogram revealed a mildly impaired LV ejection fraction (LVEF) of 48% with anteroseptal wall thinning, consistent with the ECG findings. The cause of the abdominal pain was thought to be cholecystitis, for which conservative treatment was planned by a gastroenterologist. Permanent

KEY TEACHING POINTS

- Transient left bundle branch block can be associated with high filling left ventricular pressure in patients with acute heart failure. The postulated mechanism is mechanical injury of the left bundle branch. The main His bundle may be vulnerable to mechanical stretch owing to its fibrous surroundings.
- High-output His bundle pacing could correct left bundle branch block associated with acute heart failure or cardiomyopathy. Recruitment of the left bundle branch block might lead to prompt myocardial recovery.
- His bundle pacing is feasible and effective for infranodal heart block, although the procedure may be challenging.

HBP for CHB and subsequent coronary angiography (CAG) were scheduled. At the time of pacemaker implantation, CHB with LBBB morphology escape rhythm was also observed (Figure 1B). The HB was mapped using unipolar recordings from a Medtronic 3830 lead (Medtronic, Minneapolis, MN) through a C315 His sheath, and His-ventricle (HV) block was confirmed (Figure 1C). However, pacing at 5 V @ 1 ms at this site did not capture the distal HB. Thus, we performed repeated pace mapping along the presumed distal course of the HB to identify the site at which nonselective HBP with recruitment of both bundles was obtained at an acceptable HBP threshold of 2 V @ 1 ms (Figure 1D).

Two days after the pacemaker implantation, CAG was performed, and the severely stenotic proximal left ascending artery was stented. Both beta-blocking agents and angiotensin-converting enzyme inhibitors were started.

KEYWORDS Cardiac resynchronization therapy; Complete heart block; Heart failure; His bundle pacing; Left bundle branch block (Heart Rhythm Case Reports 2019;5:143–147)

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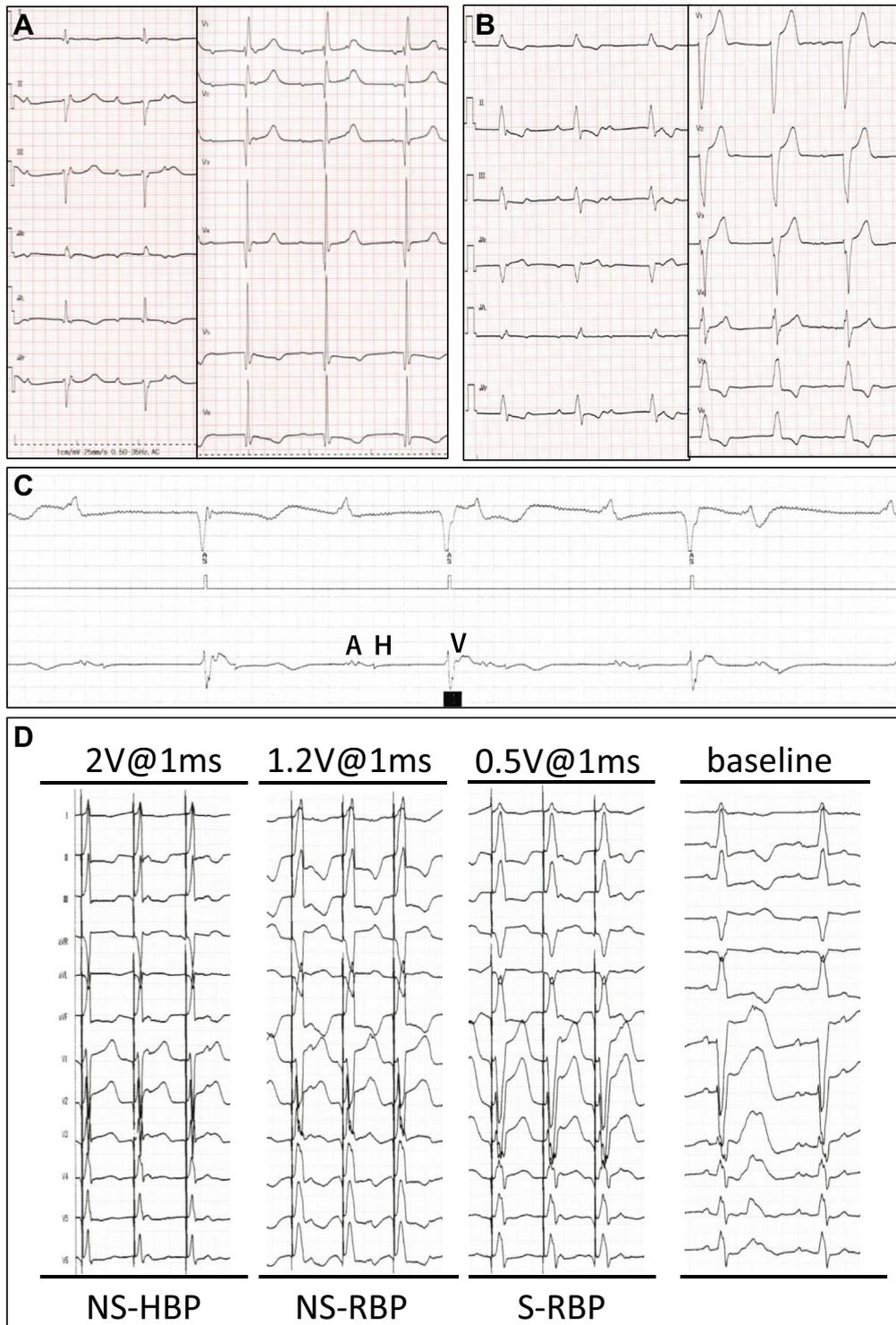


Figure 1 **A:** Baseline electrocardiogram (ECG) showing complete heart block (CHB) with right bundle branch block and left anterior fascicular block morphology escape rhythm. Precordial abnormal Q waves are also noted, suggesting old anterior myocardial infarction. **B:** Baseline ECG at the time of the first procedure, showing CHB with left bundle branch block (LBBB) morphology escape rhythm. **C:** Intracardiac electrograms demonstrating His-ventricle block. **D:** Intraoperative ECG at the final successful site. Pacing at 2.0 V @ 1 ms and above resulted in nonselective His bundle pacing (NS-HBP). Left bundle, right ventricle, and right bundle capture was lost at 1.9 V @ 1 ms, 1.1 V @ 1 ms, and 0.4 V @ 1 ms, respectively. NS-RBP = nonselective right bundle pacing; S-RBP = selective right bundle pacing.

At the 1-week routine follow-up, the HBP threshold increased to 3.25 V @ 1 ms. At the 4-week follow-up, the HBP threshold further increased to 4.5 V @ 1 ms and we decided to perform elective lead revision.

At the time of the second procedure, because of the difficulty in achieving the original site, we mapped the HB region using a new pacing lead without removing the original lead. At a slightly proximal position (approximately

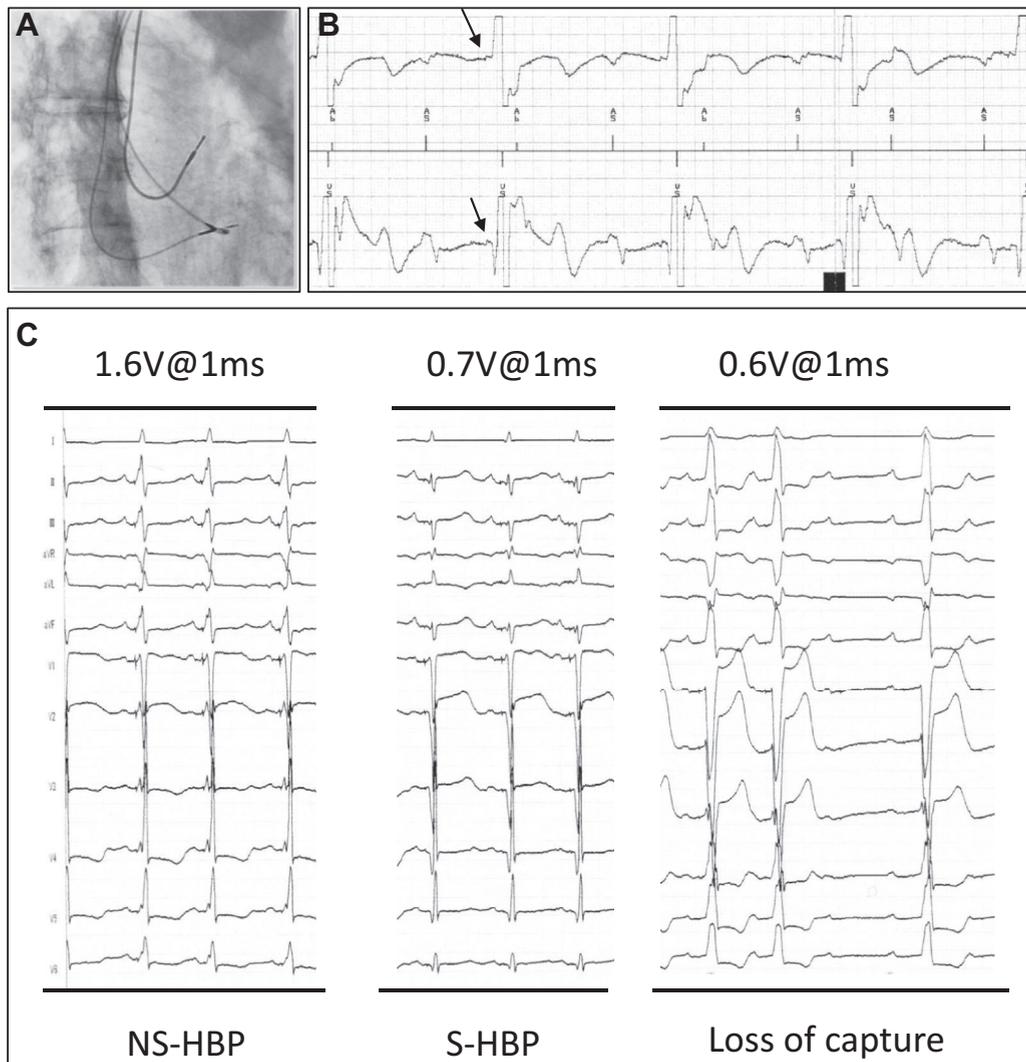


Figure 2 A: Fluoroscopic image (right anterior oblique projection) at the time of lead revision, showing a new His bundle pacing (HBP) lead placed slightly proximal and inferior to the original lead. B: Intracardiac electrograms after a new lead fixation, showing distal HB potential with HB injury current (arrows). C: Intraoperative electrocardiogram at the successful site. Pacing at 1.6 V and above results in nonselective HBP (NS-HBP). Below 1.5 V, there is selective HB capture (S-HBP). At pacing output 0.6 V @ 1 ms, selective right bundle capture is seen just before loss of capture (right panel).

3 mm from the original site) (Figure 2A), the distal HB potential was recorded and excellent nonselective HBP with an HBP threshold of 0.7 V @ 1 ms was obtained with an HB injury current (Figure 2B and C). The right ventricular (RV) capture threshold was 1.6 V @ 1 ms, and the pacing output was programmed at 3 V @ 1 ms.

Until the 6-month follow-up after the lead revision, the HB capture threshold had remained stable at 0.5 V @ 1 ms, although the RV pacing threshold had progressively increased to 3.5 V @ 1 ms. Repeat echocardiography had also shown an almost unchanged LVEF of 46%.

One week after the last follow-up, the patient was admitted for acute heart failure. Upon admission, an ECG showed selective HBP without correction of LBBB (Figure 3A). Pacemaker interrogation revealed an acute increase of the HB(LB) pacing threshold to 3.75 V @ 1 ms, while the HB (right bundle) capture threshold remained

at 0.5 V @ 1 ms. Pacing output was reprogrammed at 5 V @ 1 ms to recruit LBBB.

Emergency echocardiography showed that the LVEF had severely declined to 18% with LV enlargement. Intravenous human atrial natriuretic peptides and diuretics were initiated, and the patient's heart failure symptoms almost fully resolved within 2 weeks. CAG showed no restenosis or new lesion.

Three weeks after admission, the patient was discharged, although both the HB(LB) pacing threshold and echocardiographic cardiac function still showed marginal improvement. Surprisingly, 4 weeks after discharge, the HB(LB) pacing threshold had dramatically improved to 0.5 V @ 1 ms and the LVEF had recovered to the preadmission value of 45%.

One year later, the patient had experienced no further episodes of heart failure, and his HBP threshold remained stable at 0.5 V @ 1 ms (Figure 3B).

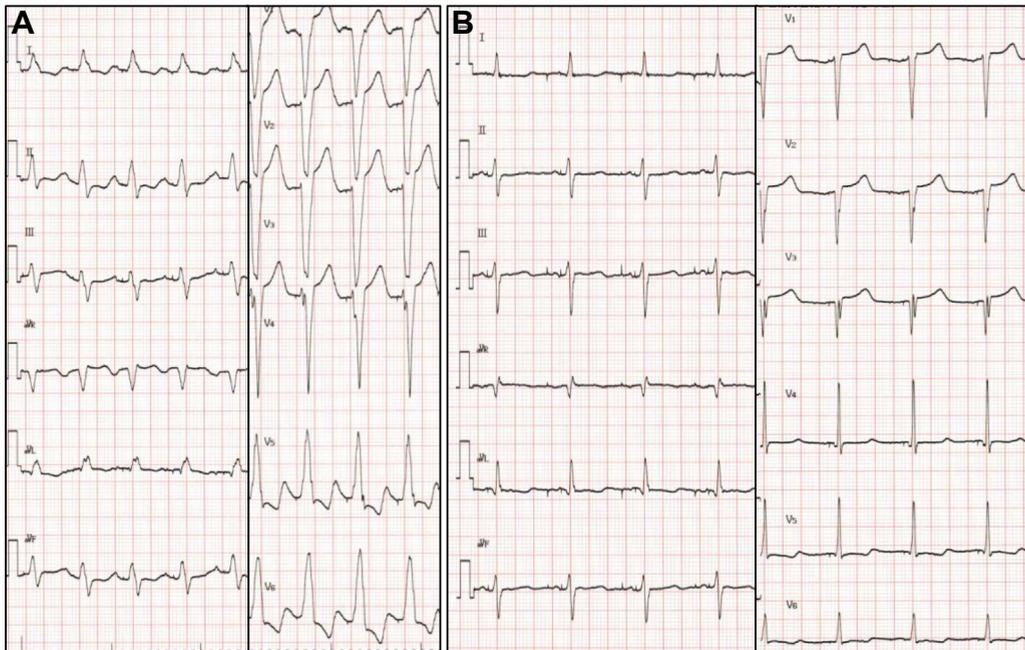


Figure 3 A: Electrocardiogram (ECG) at admission for acute heart failure, showing selective His bundle pacing (HBP) without correction of left bundle branch block because of an acute increase in the His bundle (left bundle) pacing threshold to 3.75 V @ 1 ms and programmed output setting at 3.0 V @ 1 ms. B: ECG at 1 year after last discharge, showing selective HBP with stable HBP threshold of 0.5 V @ 1 ms.

Discussion

Acute heart failure can occur when a sudden increase in the intracardiac pressure and/or acute myocardial dysfunction occurs.² The most common etiology of acute heart failure is cardiac ischemia. In the herein-described patient with silent MI, ischemia is an unlikely cause of the episode of acute heart failure because neither restenosis nor new lesions were observed on CAG. However, the possibility of coronary artery vasospasm as an initial triggering event could not be excluded. Patients with silent MI reportedly have a significantly higher risk of heart failure than those without MI.³

Additionally, high filling LV pressure and/or global systolic dysfunction in patients with heart failure, such as tachycardia-induced cardiomyopathy, might cause transient LBBB.¹ In this patient, the LBBB was due to a sudden increase in the HB(LB) pacing threshold, which was apparently associated with high filling LV pressure and/or global systolic dysfunction in association with acute heart failure.

The postulated mechanism of such new-onset LBBB in patients with heart failure is thought to be transient mechanical injury of the left bundle branch. When the left ventricle expands with high end-diastolic pressure in acute heart failure or cardiomyopathy, the left bundle branch may be stretched, and the mechanical injury may cause a conduction disturbance in this bundle branch. However, the most likely site of LBBB induced by mechanical stretch in this case was the main HB because it could be corrected by higher-output pacing in the distal HB. If the block had been further down, then high output would have been unlikely to correct the LBBB. The main HB may be vulnerable to mechanical stretch because of its fibrous surroundings.⁴

Whether the acute heart failure–associated LBBB itself further exacerbated the LV systolic function in our case is unclear, but electrical synchronization by higher-output HBP might have resulted in accelerated improvement in the patient's systolic function and HB capture threshold. RV pacing is well known to cause ventricular dyssynchrony and heart failure. If this patient had undergone conventional RV pacing, recovery of cardiac function might have been delayed or not obtained.

As described in this report, HBP for infranodal CHB might be challenging.⁵ At the time of the first procedure, the lead tip was probably fixed in the proximal right bundle branch, explaining the high HBP threshold of 2 V @ 1 ms and the low right bundle pacing threshold of 0.5 V @ 1 ms.⁶ Conversely, at the time of lead revision, the lead was placed beyond the blocked site in the HB, explaining the excellent HBP threshold and local His injury current.⁵ In patients with HV block undergoing HBP, it is critical to achieve a stable long-term threshold by pacing the distal HB beyond the site of block, where His capture threshold < 1 V @ 1 ms can be obtained.⁵ Alternatively, as recently reported, a novel method involving left bundle branch pacing may be considered for HV block or LBBB.⁷

Conclusion

In summary, we have herein presented a patient who underwent permanent HBP for complete infranodal heart block and developed transient LBBB because of a massive increase in the HB(LB) pacing threshold associated with acute heart failure. With the recovery of myocardial function, the LBBB

(from the increase in the HBP threshold) resolved and the HBP threshold remained stable during follow-up.

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