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Recovery of Right Ventricular Apical Pacing-Induced Cardiomyopathy with Left Bundle Branch Pacing

Sağ Ventriküler Apikal Uyarıma Bağlı Kardiyomiyopatinin Sol Dal Uyarımı ile Düzelmesi

CASE REPORT
OLGU SUNUMU

ABSTRACT

Right ventricular pacing (RVP) is conventionally preferred in the treatment of patients with atrioventricular block. However, long-term RVP may lead to pacing-induced cardiomyopathy (PICM), characterized by new-onset or worsening ventricular functions due to dyssynchronous ventricular electrical activation, abnormal ventricular remodeling, and increased energy expenditure. Historically, biventricular pacing (BVP) and guideline-directed medical therapy were the only treatment option for PICM. Recently, conduction system pacing, including left bundle branch area pacing (LBBaP), has emerged as a physiological alternative to BVP, showing better results in electro-mechanical ventricular synchronization and hemodynamic parameters compared to BVP. We present a case involving a patient from whom the PICM was successfully recovered shortly after LBBaP.

Keywords: Left bundle branch pacing, left ventricular ejection fraction, pacing-induced cardiomyopathy

ÖZET

Atriyoventriküler bloklu hastaların tedavisinde geleneksel olarak sağ ventriküler uyarım tercih edilmektedir. Bununla birlikte, uzun süreli sağ ventriküler uyarım, senkron olmayan ventriküler elektriksel aktivasyon, anormal ventriküler yeniden şekillenme ve artan enerji harcaması nedeniyle ventriküler fonksiyonların bozulmasıyla karakterize, kalp pili uyarımına bağlı kardiyomiyopatiye neden olabilir. Biventriküler uyarım, kalp pili uyarımına bağlı kardiyomiyopatide kılavuzun önerdiği tıbbi tedavinin yanı sıra tek tedavi seçeneğiydi. Bununla birlikte, sol dal alanı uyarımını içeren iletim sistemi uyarımı yakın geçmişte ortaya çıktı ve biventriküler uyarıma kıyasla elektro-mekanik ventriküler senkronizasyon ve hemodinamik parametreler açısından daha iyi sonuçlar sağlayarak biventriküler uyarıma fizyolojik bir alternatif olarak benimsendi. Bu yazımızda sol dal alanı uyarımından kısa süre sonra kalp pili uyarımına bağlı kardiyomiyopatinin toparladığı bir hastayı sunduk.

Anahtar Kelimeler: Sol dal uyarımı, sol ventrikül ejeksiyon fraksiyonu, kalp pili uyarımına bağlı kardiyomiyopati

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Permanent pacemaker therapy is a crucial therapeutic option for patients diagnosed with atrioventricular block (AVB).[1] However, conventional right ventricular pacing (RVP) is not physiologically optimal, and chronic RVP can lead to the development of pacing-induced cardiomyopathy (PICM) by causing electro-mechanical dyssynchrony.[2,3] A recent meta-analysis identified male sex, a history of myocardial infarction (MI), chronic kidney disease (CKD), atrial fibrillation (AF), baseline left ventricular ejection fraction (LVEF), native QRS duration (QRSd), RVP percentage, and paced QRS duration (pQRSd) as key risk factors for PICM.[4] Although there is no consensus on managing these patients beyond guideline-directed medical therapy (GDMT) for heart failure, biventricular pacing (BVP) and conduction system pacing (CSP) through His bundle pacing (HBP) or left bundle branch area pacing (LBBaP) have demonstrated improvements in clinical and echocardiographic abnormalities associated with PICM.[2,5-7] We present a case of a patient who developed symptomatic PICM after RVP for symptomatic 2nd-degree Mobitz type 2 atrioventricular block and subsequently showed significant clinical and echocardiographic improvement shortly after transitioning to LBBaP.

Case Report

A 62-year-old male with a history of Takayasu arteritis, coronary artery disease (underwent coronary artery bypass graft surgery 5 years prior), and a percutaneous carotid artery intervention for carotid stenosis was admitted to our Cardiology department with presyncope. Upon admission, the 12-lead electrocardiography (ECG) revealed a 2^{nd}-degree Mobitz type 2 AVB with a ventricular rate of 38 bpm (Figure 1). Transthoracic echocardiography (TTE) showed an LVEF of 48%, moderate mitral and tricuspid regurgitation, and an estimated systolic pulmonary artery pressure (sPAP) of 35 mmHg. A dual-chamber permanent pacemaker was implanted using a conventional method (RVP), achieving a pQRS duration of 160 ms (Figure 2A-B). The patient's hospital stay was uneventful. Ten months after permanent pacemaker implantation, the patient was admitted to another hospital with acute decompensated heart failure and was diagnosed with heart failure with reduced ejection fraction (HFrEF). He was referred to our Cardiology department for additional treatment strategies beyond GDMT. At this time, he was still symptomatic (New York Heart Association [NYHA] class 3) despite optimal treatment for HFrEF for four months. Echocardiography revealed a left ventricular end-diastolic diameter (LVEDD) of 64 mm, an LVEF of 27%, severe mitral and tricuspid regurgitation, and an sPAP of 60 mmHg (Video 1A-B). The 12-lead ECG displayed an atrial sensed, ventricular paced rhythm with a pQRSd of 160 ms. The serum brain natriuretic peptide (BNP) level was 3222.6 pg/mL. Coronary computed tomography angiography revealed no new coronary artery stenosis and intact coronary bypass grafts. Thus, we diagnosed our patient with PICM and planned to upgrade his dual-chamber pacemaker to a cardiac resynchronization therapy pacemaker (CRT-P) via LBBaP. The left upper extremity venography showed no venous obstruction. Following the left axillary venous access, the Selectra 3D 55-39 (Biotronic, Berlin, Germany) delivery sheath was advanced into the right ventricle over a 0.038-inch guidewire. The Solia S60 ventricular electrode was prepared for LBBaP (the helix was activated on the table).[8] The procedure was conducted under fluoroscopy, continuous 12-lead ECG (Artis Zee angiography system, Siemens Healthcare GmbH, Forchheim, Germany), and pacing system analyzer (PSA) intracardiac electrogram (EGM)/modified 3-lead ECG recording (The Renamic Neo programmer, Biotronik). We optimized the position of the delivery sheath using the right anterior oblique (RAO) 30° view and performed the penetration of the interventricular septum after achieving optimal unipolar pace-

ABBREVIATIONS

AF	Atrial fibrillation
AVB	Atrioventricular block
BVP	Biventricular pacing
CKD	Chronic kidney disease
CSP	Conduction system pacing
ECG	Electrocardiography
GDMT	Guideline-directed medical therapy
LBBaP	Left bundle branch area pacing
LVEDD	Left ventricular end-diastolic diameter
MI	Myocardial infarction
PICM	Pacing-induced cardiomyopathy
RVP	Right ventricular pacing
sPAP	Systolic pulmonary artery pressure

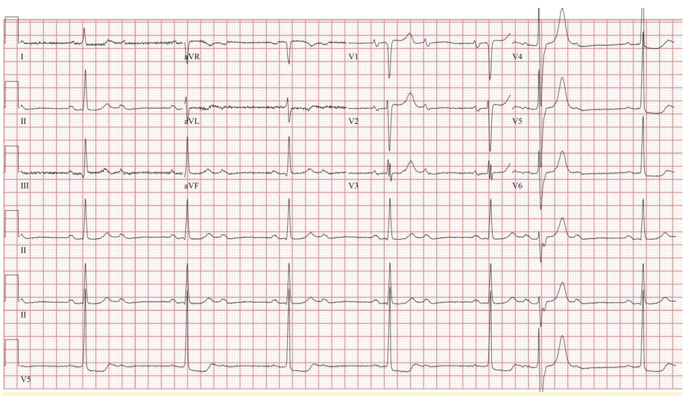

Figure 1. The 12-lead electrocardiogram (ECG) from the initial presentation displaying a Mobitz type 2 (2:1) atrioventricular block with a ventricular rate of 39 bpm.

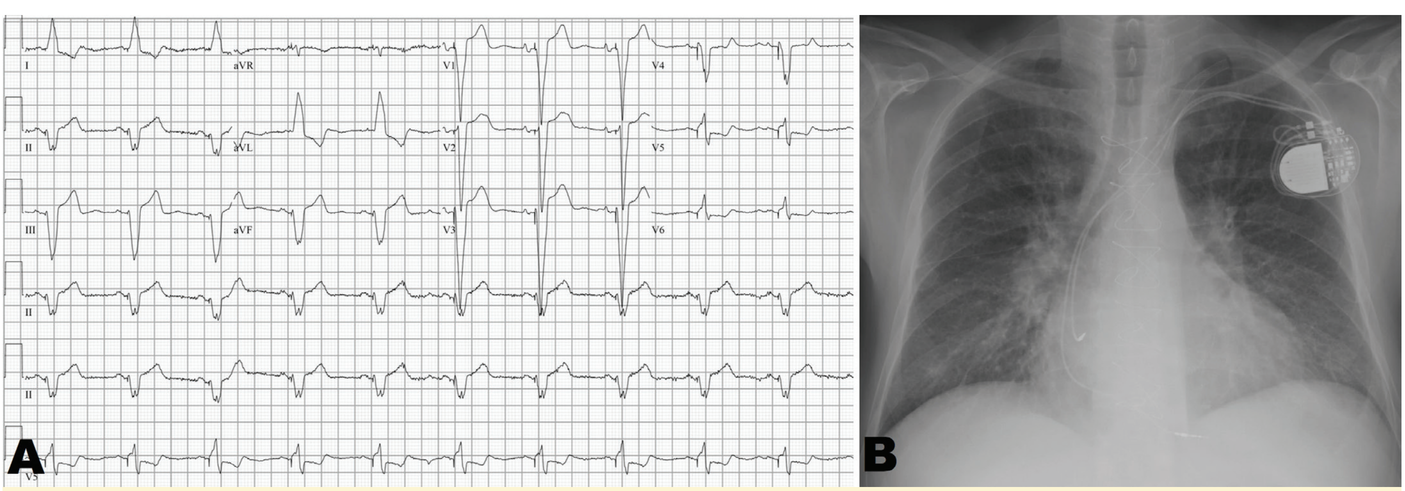

Figure 2. (A) The 12-lead ECG after the implantation of a conventional dual-chamber permanent pacemaker, showing an atrial sensed-ventricular paced rhythm with a paced QRS duration of 160 ms. (B) A post-procedural chest X-ray illustrating the apical placement of the right ventricular lead.

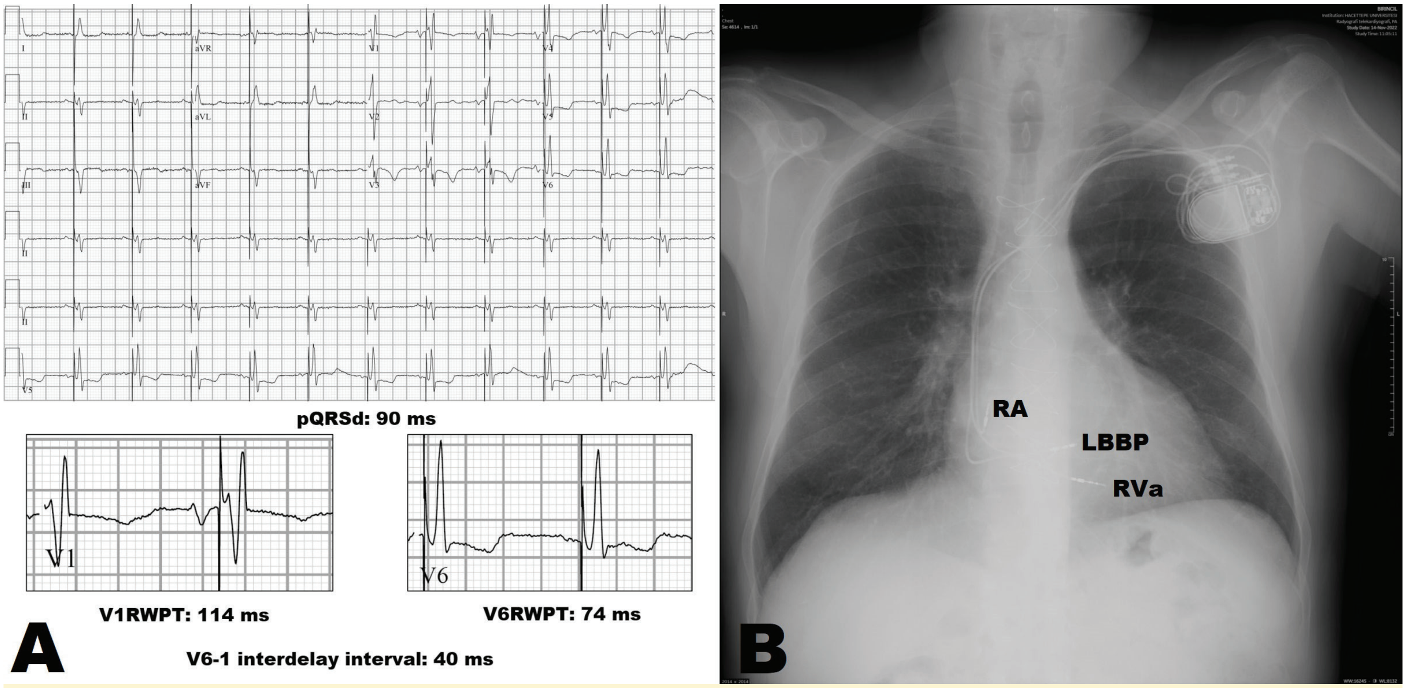

Figure 3. (A) The 12-lead ECG following an upgrade to cardiac resynchronization therapy pacemaker (CRT-P) via left bundle branch pacing (LBBP), showing an atrial sensed-ventricular paced rhythm with a paced QRS duration of 90 ms. (B) A post-procedural chest X-ray depicting the septal placement of the LBBP lead.

map ECG parameters (looking for W pattern at V1 derivation, aVL/aVR discordance, R amplitude lead II > lead III). During the septal penetration (Video 2), we monitored the unipolar impedance, fluoroscopic advancement of the electrode, and progressive changes in lead V1 from the W pattern to the terminal r/R wave. Once the terminal r/R wave appeared in lead V1, we measured the V6 R wave peak time (V6RWPT or left ventricular activation time (LVAT)) (< 80 ms), V6-1 interpeak delay (> 33 ms), and uni/bi-polar threshold test (< 1.5V@0.4s), all indicative of non-selective left bundle branch pacing (LBBP) as per the recent EHRA (European Heart Rhythm Association) consensus document.[9] A CRT-P device was implanted, with the LBBP lead inserted into the left ventricle socket, and programmed to LV-only pacing mode with unipolar pacing polarity. Postprocedural echocardiography confirmed that the LBBP electrode was positioned in the LV subendocardial region (Video 3). The pQRSd, V6RWPT, and V6-1 inter-delay times were measured at 90 ms, 74 ms, and 40 ms in the post-procedure ECG (Figure 3). The patient was discharged uneventfully on optimal GDMT and remained asymptomatic with good functional capacity (NYHA class 1) at the 1st and 3rd-month follow-up visits. At the third-month visit, echocardiography revealed an LVEDD of 54 mm, an LVEF of 45%, moderate mitral and tricuspid regurgitation, and an sPAP of 35 mmHg (Video 4A-B).

Discussion

High-degree AVB is one of the most common indications of permanent pacemaker therapy.[10] The implantation technique for dual-chamber permanent pacemaker is detailed in the recent European Heart Rhythm Association (EHRA) consensus document.[11] Among several locations for right ventricular (RV) lead placement—including the RV outflow tract, septum, and apex—the RV apex was the preferred site in half of the centers surveyed by the EHRA.[12] However, the main concerns about RV apical pacing include its long-term harmful effects on LV function, heart failure, and mortality.[13-15] Peri-procedural complications, such as perforation, also present higher risks with apically implanted RV leads.[16] RV apical myocardial pacing leads to non-physiological delayed ventricular activation, resulting in LV electro-mechanical dyssynchrony, decreased myocardial energy expenditure, impaired coronary perfusion, and reduced LV systolic function.[17] These adverse effects are more pronounced in patients with preexisting LV dysfunction.[4,18] However, there is no consensus on a standard definition of PICM. Previous studies have defined PICM as a ≥ 10% reduction in LVEF irrespective of baseline LVEF without another cause other than RVP, a decrease in LVEF to ≤ 40% if baseline LVEF was ≥ 50%, or a reduction in LVEF of ≥ 5-10% if the baseline was < 50%, or new-onset heart failure (HF), or the requirement for a CRT upgrade.[4,17,19] Male sex, history of MI, CKD, AF, baseline LVEF, native QRSd, RVP percentage, and pQRSd were identified as significant predictors of PICM in a recent meta-analysis.[4] Our patient, a male with a history of MI and preexisting LVEF dysfunction (48%), had a 100% RVP percentage and a pQRSd of 160 ms. The presence of several PICM risk factors likely accelerated the development of PICM shortly after the conventional dual-chamber pacemaker implantation. The baseline LVEF of 48% was reduced to 27% after RVP, aligning with the PICM definition from previous studies (an absolute reduction in LVEFof ≥ 5-10% if baseline LVEF was < 50%).

Currently, there is no consensus on managing patients with PICM. Current guidelines recommend upgrading to cardiac resynchronization therapy (CRT) for patients who developed

PICM.[10] The CRT upgrade, either via biventricular pacing or CSP, has been shown to improve clinical and echocardiographic abnormalities.[2,4,20] Significant improvement in LVEF was observed following the biventricular pacing upgrade, which correlated with the narrowing of the pQRSd.[2] Additionally, CSP via HBP or LBBaP has been widely adopted in clinical practice as an important alternative to biventricular pacing, proving to be an effective and safe method.[5,7] Previous studies[21] have demonstrated that HBP led to better improvements in functional capacity, and electrocardiographic and echocardiographic outcomes compared to biventricular pacing in patients with PICM. Rademakers et al.[7] also showed significant improvements in electrical resynchronization, LVEF, and functional capacity with LBBaP in the short-term follow-up of patients with PICM. After diagnosing our patient with PICM, an upgrade to CRT-P using the physiological pacing method (LBBaP) was planned and successfully performed. Post-procedure, the pQRSd was reduced from 160 ms with RVP to 90 ms with LBBaP. Additionally, there was a notable improvement in functional capacity and echocardiographic parameters at the 3rd-month follow-up visit (NYHA class I, an LVEF of 45%, and an LVEDD of 52 mm).

According to the recent Heart Rhythm Society (HRS) guidelines on cardiac physiological pacing for the avoidance and mitigation of heart failure,[22] periodic assessment of ventricular function is recommended for patients requiring substantial RVP (> 20-40%). The guidelines also suggest considering CSP via HBP or LBBaP for patients with an indication for permanent pacing and an LVEF of 36-50% who require substantial RVP (> 20-40%), to reduce the risk of PICM. This recommendation is supported by recent studies[23,24] among patients with indications for anti-bradycardia pacing who underwent LBBaP, which showed that LBBaP prevented the development of PICM.

Conclusion

In conclusion, close follow-up is necessary after conventional RV lead placement in patients at risk for PICM who require high-burden RVP. CSP should be considered for patients who require high-burden ventricular pacing and have baseline LV systolic dysfunction (< 50%). Upgrading to CRT-P via LBBP may significantly improve functional capacity, electrocardiographic, and echocardiographic parameters in a short period for patients with PICM.

Before the case presentation, the patient was informed about the procedure results. It was explained that these results would contribute to scientific knowledge. Written informed consent was obtained from the patient for the case presentation.

Informed Consent: Written informed consent was obtained from the patient for the case presentation.

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Video 1. The presentation of the patient with pacing-induced cardiomyopathy revealed a left ventricular ejection fraction of 27%, global hypokinesia, and a dilated left ventricular cavity [left ventricular end-diastolic diameter (LVEDD) of 64 mm] from parasternal (A) and apical 4-chamber (B) views.

Video 2. Fluoroscopy demonstrating the deep septal penetration of the left bundle branch pacing (LBBP) lead following contrast agent injection from the delivery sheath.

Video 3. Post-procedural echocardiography showing the LBBP lead positioned at the subendocardial border of the left ventricle.

Video 4. Echocardiography from parasternal (A) and apical 4-chamber (B) views during the 3rd-month follow-up visit, showing an improved left ventricular ejection fraction (45%) and reverse left ventricular remodeling (LVEDD of 52 mm).

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