SCIENTIFIC LETTER

Left Bundle Branch Pacing in a Patient with Orthotopic Heart Transplantation and Pacemaker-Induced Cardiomyopathy: First Case in Argentina

Estimulación de la rama izquierda en paciente con trasplante cardíaco ortotópico con miocardiopatía inducida por marcapasos: primer caso en Argentina

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Sinus node dysfunction (SND) and atrioventricular block may require pacemaker (PM) implantation in patients with orthotopic heart transplantation (OHT). This is a case of a female patient with post-OHT SND who underwent DDR PM implantation and developed pacemaker-induced cardiomyopathy and left ventricular systolic dysfunction, which required an upgrade to left bundle-branch pacing (LBBP).

The patient was a 26-year-old female, who underwent an OHT in 2012 due to dilated cardiomyopathy requiring extracorporeal membrane oxygenation (ECMO) support for 14 days. In 2019, a dual-chamber pacemaker was implanted due to SND, and during the last year, she was hospitalized for dengue and infectious endocarditis. She required explantation and reimplantation of the pacemaker. An endomyocardial biopsy (EMB) was indicated in the context of acute dengue because of a drop in left ventricular ejection fraction (LVEF). The biopsy showed mild, grade 1R rejection (ISHLT 2005), pAMR1 (+). The patient did not receive treatment; the subsequent EMB showed no rejection and a LVEF of 52%. She showed an increase in the percentage of ventricular pacing (VP 92%) and a deterioration in LVEF, so the following studies were performed:

- Cardiac Doppler ultrasound: Left ventricular dilatation with left ventricular diastolic diameter (LVDD) of 54 mm, indexed 33.5 mm/m². LVEF 40%. Akinesia of the basal inferoseptal, inferior, and basal and mid inferolateral walls. Abnormal interventricular septum motion. Right ventricle (RV) normal. Moderate mitral regurgitation (MR) due to restrictive closure of the posterior leaflet. Moderate tricuspid regurgitation (TR) with central jet. Systolic pulmonary artery pressure (sPAP) 30 mmHg. E-wave deceleration time 91 ms. Pulmonary acceleration time 148 ms. Isovolumic relaxation time 83 msec.
- Holter electrocardiography (ECG): PM rhythm (atrial sensing (AS)-ventricular pacing (VP) at 80 bpm (60-88); 655 premature supraventricular beats (PSVBs) with no runs of tachycardia (<1%). No pauses.
- ECG: atrial sensing and ventricular pacing at 60 bpm with isolated PSVBs (Figure 1A).
- Cine-coronary angiography (cine-CAG): no significant angiographic lesions. Mild lesion in the mid-segment of the circumflex artery.
- Intervalometer data: DDD mode (60-130 bpm). ECG AS/VP (AP 23%, VP 92.4%). Battery 10A/3.02 V. AV interval 180-150 ms. Sensing: P wave 0.4 mV, R wave dependent. Outputs: RA 3.5V/0.4 ms, RV 2.5 V/0.4 ms. Thresholds: RA NR (AT/AF), RV 0.5 V/0.4 ms. Impedances: RA 361 Ω, RV 380 Ω. From September 20 to September 27: AT/AF episodes with RA lead sensing failure.
- Endomyocardial biopsy: EM G0 ISHLT.

Current treatment: tacrolimus, meprednisone, rapamycin, acetylsalicylic acid, bisoprolol, B complex, rosuvastatin, folic acid, ferrous sulfate, magnesium, potassium, lamotrigine,brivaracetam, trimetoprime, sulfametoxazol, warfarin.

Progression to left ventricular systolic dysfunction was interpreted as pacemaker-induced cardiomyopathy due to the high percentage of ventricular pacing. It was decided to upgrade to physiological left bundle-branch pacing and to reposition the RA lead. A right deltopectoral incision was made, the generator was exposed, and the RA lead was extracted by simple traction. A right jugular puncture was performed, and

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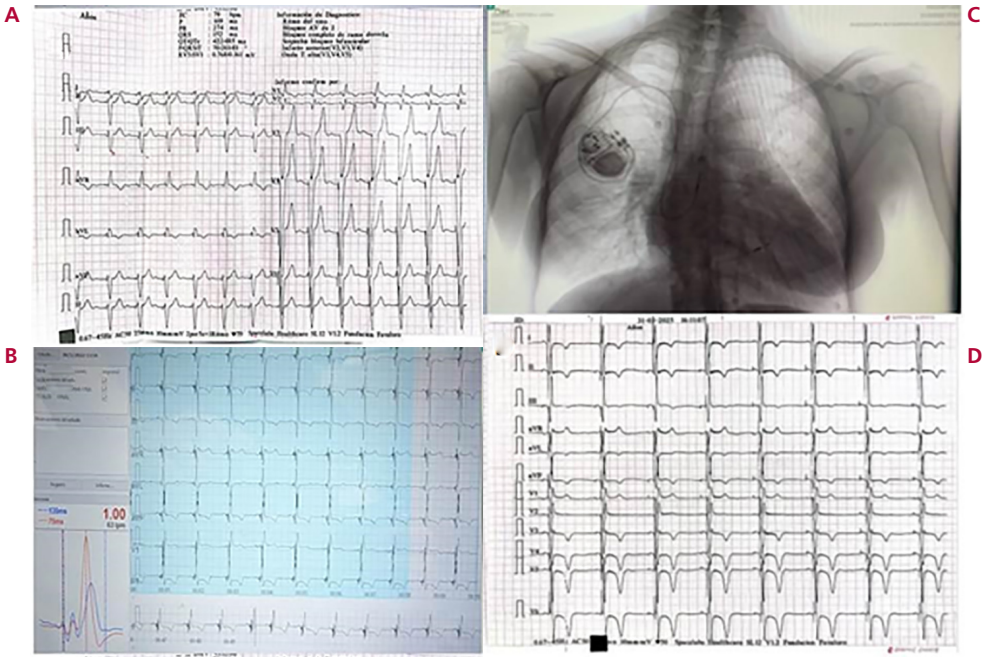
Fig. 1. A. Pre-implantation ECG: dual-chamber pacemaker rhythm with a pattern consistent with complete left bundle-branch block (LBBB). **B.** ECG during implantation showing QRS duration of 135 ms and a patent rSR' pattern in lead V1. **C.** Post-implantation chest X-ray. **D.** Post-implantation ECG showing QRS duration of 135 ms and negative T waves in the anterolateral leads (electrotonic modulation).



a 7 Fr introducer was advanced. A curved sheath was inserted into the right ventricle and positioned in the interventricular septum. An active fixation lead (Select Secure MRI Surescan, Medtronic) was advanced and fixed to the septum to achieve left bundle- branch. An 8 Fr introducer sheath was then inserted via the right subclavian puncture under the clavicle, and the lead was advanced and secured in the right atrium with an adequate threshold. It was connected to the DR generator in the pectoral pocket. The previous right ventricular lead was removed by simple traction without complications.

Post-implantation electrocardiography: Sinus rhythm 60 bpm, QRS 135 ms with rSR' pattern in V1 (Figures 1B and 1D).

Chest X-ray: Normally positioned leads without complications (Figure 1C).

The most common conduction abnormality following heart transplantation is right bundle-branch block. (1-2) In this case, the patient developed SND requiring permanent cardiac pacing and subsequently progressed to increased ventricular pacing requirements, leading to left ventricular systolic dysfunction (LVSD). (3) Common etiologies of LVSD were ruled out through cine-CAG and EMB. Although mild cellular rejection cannot be completely ruled out as the cause of late graft failure, the lack of improvement in LVEF with immunosuppression argues against it being the cause of LVSD. A pacemaker with LBBP was then implanted.

To our knowledge, this is the first case reported in Argentina of LVSD due to a high percentage of ventricular pacing in a transplanted heart treated with LBBP. We believe that the temporal relationship between the high percentage of pacing and the development of LVSD, followed by an adequate response, would demonstrate pacing-induced cardiomyopathy. (4-5) LBBP corrected the pacing-induced pseudo-left bundle branch block (LBBB), thus overcoming the electrical dyssinchrony caused by the pacemaker. Six months later, new echocardiographic measurements of LV function will be performed to assess its progression.

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Conflicts of interest
None declared.
(See conflicts of interest forms on the website).

Ethical considerations
Not applicable

REFERENCES

1. Golshayan D, Seydoux C, Berguer DG, Stumpe F, Hurni M, Ruchat P, et al. Incidence and prognostic value of electrocardiographic abnormalities after heart transplantation. Clin Cardiol. 1998;21:680-4.

https://doi.org/10.1002/clc.4960210914.
2. Ferretto S, Tafciu E, Giuliani I, Feltrin G, Bottio T, Gambino A, et al. Interventricular conduction disorders after orthotopic heart transplantation: risk factors and clinical relevance. Ann Noninvasic Electrocardiol. 2017;22:e12402. https://doi.org/10.1111/anec.12402.
3. Vaillant C, Martins RP, Donal E, Leclercq C, Thébault C, Behar N, et al. Resolution of left bundle branch block-induced cardiomyopathy by cardiac resynchronization therapy. J Am Coll Cardiol. 2013;61:1089–95. https://doi.org/10.1016/j.jacc.2012.10.053.
4. Sze E, Dunning A, Loring Z, Atwater BD, Chiswell K, Daubert JP, et al. Comparison of incidence of left ventricular systolic dysfunction among patients with left bundle branch block versus those with normal QRS duration. Am J Cardiol 2017;120:1900–7. https://doi.org/10.1016/j.amjcard.2017.08.003.
5. Do DH, Bailey KL, Beyer R, Neubuerger S, Bradfield J, Shivkumar K, et al. Outcomes in orthotopic heart transplantation following pacemaker implantation. Pacing Clin Electrophysiol 2023;46:583-91. https://doi.org/10.1111/pace.14716.