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LEFT BUNDLE PACING FOR THIRD-DEGREE ATRIOVENTRICULAR BLOCK IN A PATIENT WITH OBSTRUCTIVE HYPERTROPHIC CARDIOMYOPATHY

Diana Andreea Ionescu[1], Florin Barbu[1], Elisabeta Bădilă[1,2], Alexandru Deutsch[1]
[1]Colentina Clinical Hospital, Bucharest
[2]Carol Davila University of Medicine and Pharmacy, Bucahrest
Correspondin author: Diana Andreea Ionescu

Rezumat

Introducere. *Aritmiile ventriculare sunt frecvente la pacienții cu cardiomiopatie hipertrofică obstructive (CMHO) și pot să determine sincopă și moarte subită, însă tulburările de conducere sunt complicații rare associate CMHO.*

Prezentarea cazului*. Pacient în vârstă de 57 de ani se internează în vederea cardiostimulării permanente pentru bloc atrioventricular (BAV) de gradIII simptomatic prin fatigabilitate și scăderea toleranței la efort. Nu a avut sincope. Acesta era sub tratament betablocant care s-a oprit de 10 zile, însă simptomatologia și tulburarea de conducere au persistat. Din antecedentele personale patologice reținem: fibrilație atrială paroxistică pentru care s-a efectuat ablație cu radiofrecvență, stenoză aortică largă, accident vascular cerebral cardioembolic și diabet zaharat tip II insulinonecesitant. Pe electrocardiogramă se vizualizează BAV gradIII cu QRS larg. Ecocardiografia transtoracică a evidențiat hipertrofie asimetrică la nivelul septului interventricular (diametrul telediastolic bazal de 22 mm) ce realizează, în repaus, un gradient în tractul de ejecție al ventriculului stâng de 150 mmHg cu mișcarea sistolică anterioară a valvei mitrale și, secundar, regurgitare mitrală severă. Astfel s-a formulat diagnosticul de CMHO. Riscul de moarte subită al pacientului este de 4.4%, ceea ce nu a impus implantarea unui defibrilator intern. S-a decis implantarea unui stimulator cardiac bicameral permanent folosind stimularea la nivelul ariei țesutului de conducere, cu evoluție bună post-implant. Având în vedere prezența CMHO și numărul mare de extrasistole ventriculare izolate, am introdus în medicație terapia betablocantă în doză maximă.*

Particularitatea cazului *este dată atât de asocierea rară a CMHO cu BAV gradIII, cât și de cardiostimularea permanentă cu succes la nivelul ariei țesutului de conducere, la un pacient cu CMHO.*

Cuvinte cheie*: cardiomiopatia hipertrofică obstructivă, bloc atrioventricular de grd III, stimulare la nivelul țesutului de conducere.*

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Abstract

Ventricular arrhythmias are common in patients with obstructive hypertrophic cardiomyopathy (HOCM) and can cause syncope and sudden death, but conduction disturbances are rarely associated with obstructive hypertrophic cardiomyopathy.

Cardiac physiologic pacing can be achieved by stimulating the intrinsic conduction system via conduction system pacing (Hiss bundle, left bundle branch-LBB- or left bundle branch area pacing). This technique can be challenging in HOCM due to septal hypertrophy. Our literature review did not find such a case reported so far where LBB area pacing was performed in a patient with HOCM with satisfying results and with the improvement of outflow obstruction of the left ventricle. Here we describe a case of a 57-year-old man with obstructive hypertrophic cardiomyopathy and third-degree atrioventricular (AV) block who presented in our cardiology department for dyspnea and fatigue. This patient was treated with LBB area pacing and recovered well.

Keywords*: obstructive hypertrophic cardiomyopathy, third-degree atrioventricular block, left bundle brunch area pacing*

Introduction

Hypertrophic cardiomyopathy is an autosomal dominant inherited genetic disease that includes a group of myocardial diseases characterized by left ventricular hypertrophy (LVH) unexplained by secondary causes[1,2].

LVH commonly manifests as asymmetric septal hypertrophy even though other patterns can occur (concentric, apical, and right ventricular hypertrophy)[2]. Bradyarrhythmias are rarely associated with hypertrophic cardiomyopathy and can cause syncope which is a risk factor for sudden death in these patients. A well-established technique for lowering the left ventricular outflow gradient in HOCM is atrioventricular sequential pacing with right ventricular apical lead implantation[3].

Long-term right apical pacing may cause a decrease in left ventricular function. Therefore, in our patient, we investigated the possibility of left bundle branch area pacing to prevent left ventricular systolic dysfunction.

Figure 1. *Baseline ECG*

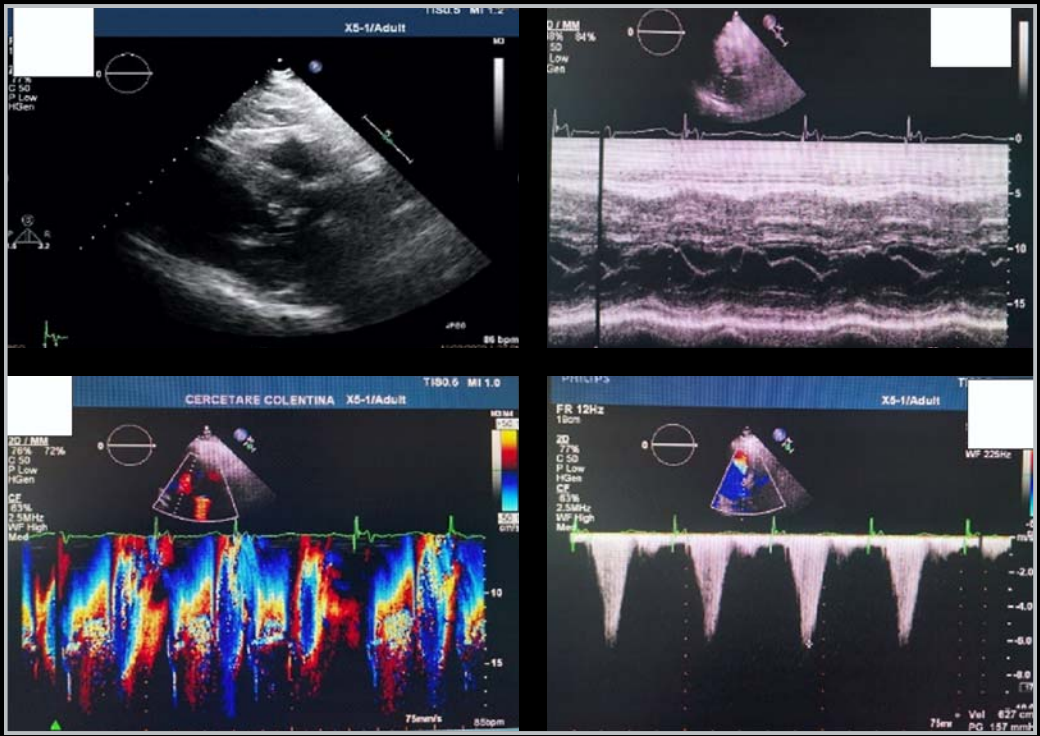

Figure 2. *Echocardiographic evaluation; 2A - asymmetric septal hypertrophy; 2B - SAM and severe mitral regurgitation; 2C - significant left ventricle outflow tract gradient*

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Case Presentation

A 57-year-old man was admitted to our Cardiology Department for dyspnea with exercise and fatigue for two weeks. He had no history of syncope. The patient was receiving beta-blocker treatment at home (metoprolol succinate 50mg), stopped for 10 days, but with persistence of symptoms. No family history of similar illness or sudden cardiac death was present. From his personal medical history, we note: primary hypertension, paroxysmal atrial fibrillation for which radiofrequency ablation was performed, and cardioembolic stroke.

On physical examination, his heart rate was 40 beats/min and his blood pressure was 150/70 mmHg. There was a grade II/VI holosystolic ejection murmur best auscultated in the apex and axilla. Initial electrocardiogram (ECG) revealed third-degree atrioventricular block with ventricular rate of 40 beats/min and with intermittent wide QRS escape rhythm (Figure 1).

A two-dimensional echocardiographic examination was performed and showed HOCM with asymmetric septal hypertrophy (basal end-diastolic thickness. of the septum was 22 mm, the posterior wall 15 mm, Figure 2A). A significant left ventricle outflow tract (LVOT) gradient was found at rest (150 mmHg) with anterior systolic motion of the mitral valve (SAM) which causes severe mitral regurgitation. (Figure 2C, Figure 2B). Left atrium was severely dilated (left atrial volume index 80 ml/m^2), but systolic function (ejection fraction -EF- 57%) and cavity dimensions of the left ventricle were within normal range.

As a result of this echocardiographic examination, the patient was diagnosed with obstructive hypertrophic cardiomyopathy.

Considering the presence of HOCM and third-degree AV block, cardiac pacing was necessary. As a novel pacing strategy, LBB area pacing was taken into account. So, we decided to implant a dual chamber pacemaker for this patient after obtaining proper consent. We used a dual-chamber pacemaker in DDDR mode.

The right ventricular lead was passed through the right ventricular septum and reached the sub-endocardium of the left ventricle side. The next step was to evaluate the paced QRS morphology obtained at this initial position; the lead stopped when QRS morphology was transformed in unipolar configuration from a complete LBB pattern to a right bundle branch block.

The following parameters were seen after pacing: R wave was 8mV, LBB pacing threshold was 0.75V/1ms, impedance 440Ω, paced QRS duration of 120ms, and paced left ventricular activation time (LVAT in lead V6) was 90 ms. Atrial lead was secured in the right atrial appendage.

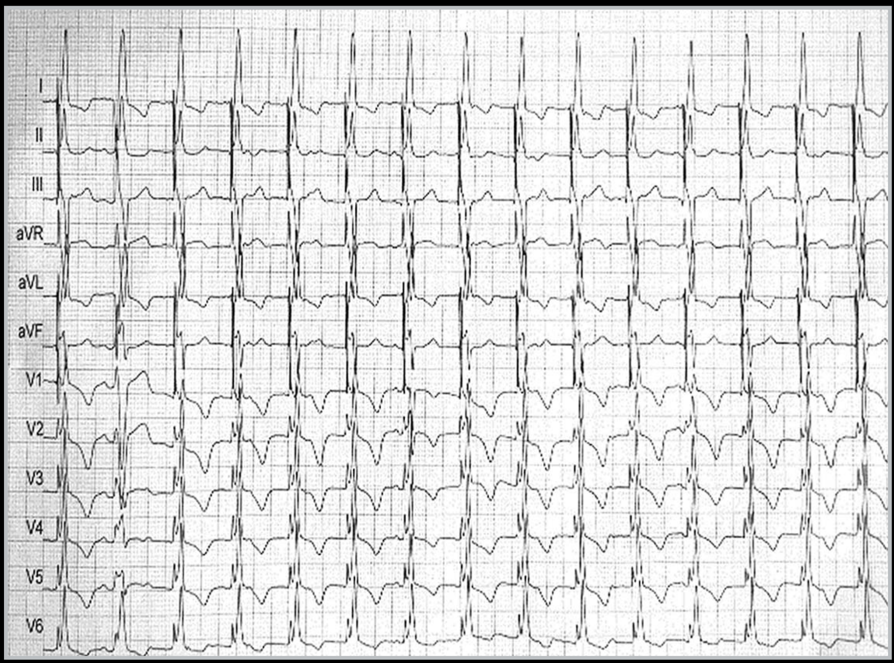

Figure 3. *ECG after LBP*

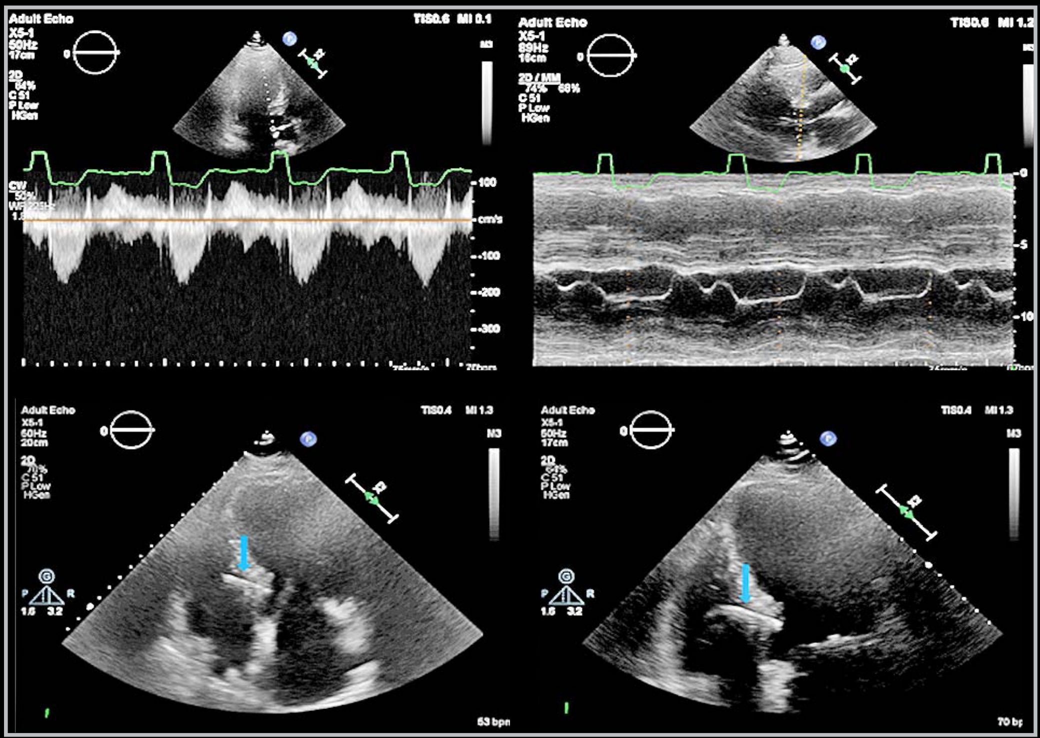

Figure 4. *Echocardiophic evaluation after LBP*

Finally, the leads were connected to the pulse generator and a significantly narrow QRS complex (Figure 3) was achieved. No ventricular septal defect, vascular injury, pneumothorax, and other complications occurred.

After implanting the pacemaker, we initiated beta-blocker treatment which was up-titrated to the maximum dose (metoprolol succinate 200mg daily). The patient was discharged in stable condition. At the 3-month follow-up, the pacemaker parameters were stable and a significant improvement in left ventricle outflow tract gradient was observed both at rest and during provocation maneuvers (Figure 4).

At the 6-month follow-up, the ECG shows intrinsic conduction with right bundle branch block alternating with left bundle branch block. We wanted to determine whether the left ventricle outflow tract gradient changes when there is intrinsic conduction versus when the patient is stimulated.

Provocation maneuvers (Valsalva) caused the left ventricle's outflow tract to rise, as shown by the echocardiographic examination. The greatest gradient was 80 mmHg when there was 1:1 intrinsic conduction with LBB, compared to 30 mmHg when LBB area stimulation was present. During the rest period, we observed no variation in the gradient both in the presence of intrinsic conduction and LBB area pacing.

Discussion

The mainstay of treatment for third-degree AV block is pacemaker implantation[4]. Selecting the appropriate implantable electronic device can be challenging when third-degree AV block is present along with obstructive hypertrophic cardiomyopathy. This is due to the significance of concurrently analyzing the risk of sudden cardiac death. Our patient did not require cardiac defibrillator implantation, because he did not fall into the high-risk category for sudden cardiac death- the hypertrophic cardiomyopathy risk score was 4.34%.

Despite an initial period of enthusiasm, right ventricular apical pacing failed to show in randomized trials significant LVOT gradient reductions and objective improvements in exercise capacity. Physiological pacing stimulates normal cardiac conduction which leads the ventricle to contract in synchrony. His bundle pacing (HBP) is a physiological pacing technique that is effective, although it can be challenging to implement technically and lead to issues during follow-up. LBBP has been suggested as an advantageous alternative to HBP because it provides lead stability and low and stable pacing threshold[6]. LBBP results in correction of left bundle branch block, quick synchronized left ventricular activity, and narrow-paced QRS duration. This method has difficulties when applied to patients with HOMC. Issues may arise from the following: fibrosis may result in thresholds that are inadequate, and the trans ventricular septal approach may present challenges due to its thickness.

In our case, we utilized LBB area pacing and the implantation procedure was with satisfactory results, and a favorable postoperative recovery was observed. Even though the paced LVAT parameter was not excellent, the presence of distal conduction disorders may have affected it.

After implanting the pacemaker, we were able to give the patient the maximal dose of beta-blockers while maintaining good tolerance.

The pacemaker parameters were stable during the three-month follow-up, and the left ventricular outflow tract gradient had significantly improved both at rest and during provocation maneuvers.

In our case, we also noticed a reduction of the left ventricle's outflow tract gradient after LBB area pacing. Therefore, we wanted to know if this could have been influenced by LBB area pacing.

At 6 months after the implant, we evaluated the patient echocardiographically both in the presence of 1:1 intrinsic conduction with LBB and stimulated. We detected a decrease in the left ventricle's outflow tract gradient in the presence of LBB area stimulation compared to intrinsic conduction, while the patient was receiving maximal beta-blocking therapy in both circumstances. This might suggest that LBBAP might have a role in the reduction of the left ventricle's outflow tract gradient in addition to beta-blocker therapy in patients with HOCM.

We wanted to highlight that all these observations were based on a single case; however, additional data is required to confirm the effect of LBB area pacing in patients with HOCM.

One of the limitations is that, as this is a relatively new procedure, large-scale randomized trials have not yet provided a clear explanation of the long-term effects.

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