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Case Report

Left bundle branch pacing in hypertrophic cardiomyopathy-a novel approach

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Abstract: Symptomatic bradycardia attributed by sick sinus syndrome in hypertrophic cardiomyopathy (HCM) is not commonly seen. Dual chamber pacing with right ventricular apical lead placement is conventional strategy in such scenario. Now physiological pacing which includes left bundle branch (LBB) pacing emerging as new technology for pacemaker implantation. Use of this technique is difficult in HCM due to septal hypertrophy. There is no such case reported so far in the literature where LBB pacing was performed in adult HCM for sick sinus syndrome. Here we present a novel approach of treating irreversible, symptomatic sinus node dysfunction in non-obstructive HCM with implementation of left bundle pacing strategy. Pacing parameters remain stable after 3 months of follow-up.

Keywords: Sick sinus syndrome, hypertrophic cardiomyopathy, dual chamber pacing, left bundle pacing

Introduction

Hypertrophic cardiomyopathy (HCM) is the most common genetic cardiovascular disease with diverse clinical manifestations, but sinus node dysfunction resulting in symptomatic bradycardia is relatively uncommon [1]. Atrioventricular (AV) sequential pacing with right ventricular (RV) apical lead placement is an age-old method to reduce left ventricular outflow gradient in hypertrophic obstructive cardiomyopathy [2]. In the long run there might be decreased left ventricular function due to either hypertrophic cardiomyopathy or due to detrimental effects of chronic right ventricular apical pacing. There is gradual shift of ventricular pacing site from RV apex to more and more adoption of physiological pacing sites i.e. conduction system pacing. Left bundle branch pacing (LBBP) may be nascent in respect to RV pacing but having a great potentiality to flourish with time. Thus to pre-empt left ventricular systolic dysfunction we explored the possibility of left bundle branch area pacing (LBBAP) in this difficult subset. LBBAP is a technique of conduction system pacing, application of this strategy in the background of hypertrophic cardiomyopathy (HCM) has made it even more unique not only because of the technical difficulties but also the rarity of evidences in the similar scenario [3]. In this case report we have used LBB pacing as novel pacing technique in HCM to treat symptomatic sinus node disease.

Case report

58 years old gentleman, known to have primary hypertension was admitted with recurrent syncope, no family history of similar illness or sudden cardiac death was present. On general examination, pallor was absent; pulse rate was 50 bpm, regular in rhythm; blood pressure was 120/80 in sitting posture without any evidence of postural hypotension; jugular venous pressure was not raised with normal wave pattern. In cardiovascular system examination apex was located at 5th intercostal area at left midclavicular line with palpable S4 at apex, on auscultation left ventricular S4 was present, and no murmur was detected even with valsalva. Other system examination was normal. Electrocardiogram (ECG) showed left ventricular hypertrophy with strain and premature atrial complex (**Figure 1A**). Frequent sinus pauses of more than 3 sec were present in 24 hours holter

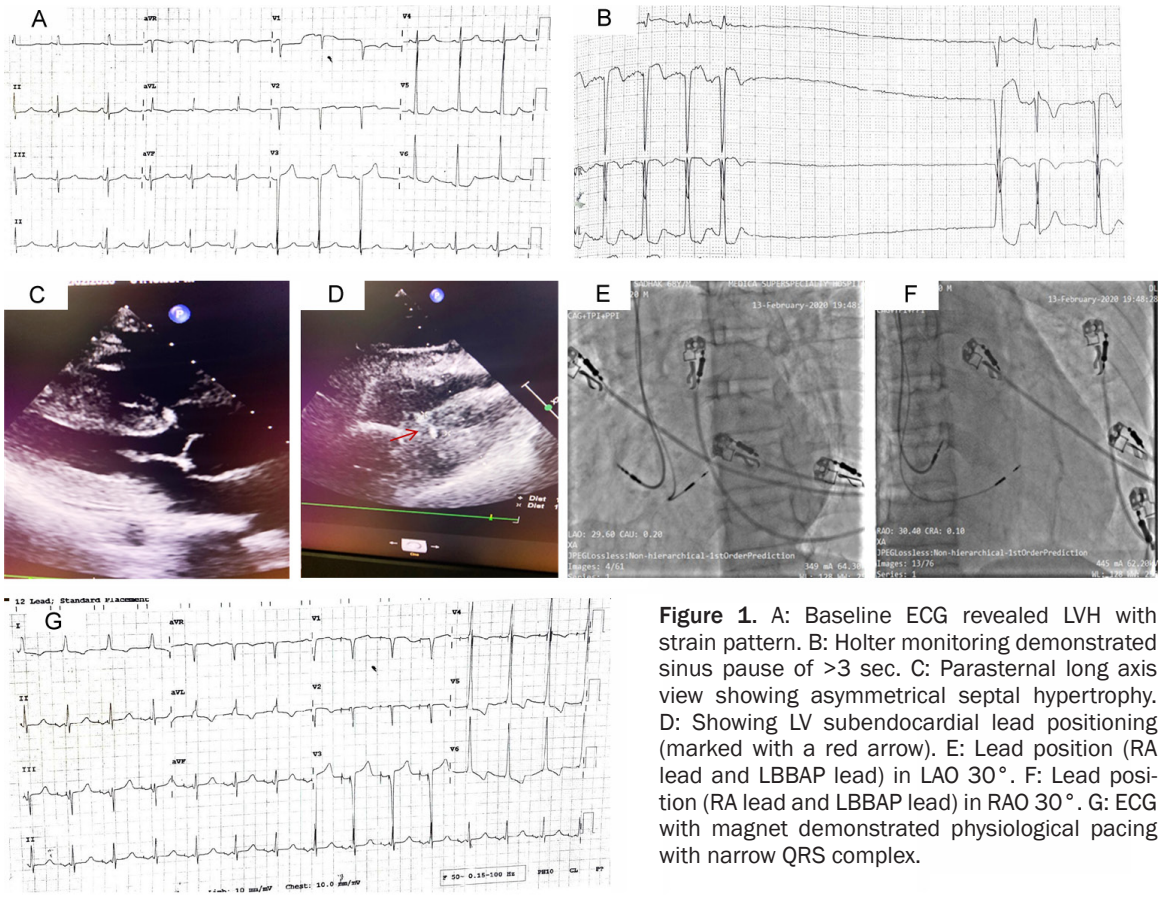

Figure 1. A: Baseline ECG revealed LVH with strain pattern. B: Holter monitoring demonstrated sinus pause of >3 sec. C: Parasternal long axis view showing asymmetrical septal hypertrophy. D: Showing LV subendocardial lead positioning (marked with a red arrow). E: Lead position (RA lead and LBBAP lead) in LAO 30°. F: Lead position (RA lead and LBBAP lead) in RAO 30°. G: ECG with magnet demonstrated physiological pacing with narrow QRS complex.

monitoring with established correlation between symptoms and bradycardia, no ventricular tachycardia was detected (**Figure 1B**). In Echocardiography asymmetrical septal hypertrophy was present (**Figure 1C**, **1D**) with maximum interventricular septum thickness (diastole) of 24 mm and left ventricular posterior wall thickness (diastole) of 15 mm, left atrial diameter was 34 mm, left ventricular ejection fraction 65% and there was grade 1 diastolic dysfunction. No significant left ventricular outflow tract obstruction (LVOTO) was demonstrated at rest or with Valsalva or after exercise, systolic anterior motion of anterior mitral leaflet and mitral regurgitation or apical aneurysm were not seen, all of these were suggestive of non-obstructive hypertrophic cardiomyopathy. Coronary angiography was normal. Electrophysiological study showed evidence of infra-Hisian block with incremental atrial pacing at paced cycle length of 500 ms with isoprenaline. So we proceeded to implant dual chamber pacemaker for this patient after taking proper consent. Quadripolar mapping catheter was used to identify the His signal. Left bundle branch area pacing (LBBAP) was performed after positioning C315 His sheath and Select Secure lead 3830 (Medtronic, Mineapolis, MN) assembly approximately 1-1.5 cm distal to the His mapping catheter tip along the line joining the quadripolar catheter tip and RV apex in RAO 30° and screwing the lead into basal transventricular septum subsequently (**Figure 1E**). Lead stability was checked and septogram was performed in LAO 30° showing lead depth of 13 mm into the interventricular septum (**Figure 1F**). ECG with magnet showed physiological pacing with narrow QRS complex (**Figure 1G**).

Following parameters were seen after pacing: R wave was 14 mV, LBB pacing threshold was 0.5 V at 0.5 ms pulse width, paced QRS duration of 110 ms, paced left ventricular activation time (LVAT in lead V6) was 70 ms. Atrial lead is also secured in left atrial appendage; finally leads were connected to Medtronic pulse generator. Complications like vascular injury,

pneumothorax, ventricular septal perforation, mural haematoma (intervetricular septum), tamponade, lead dislodgement were absent. Patient was discharged in stable condition and pacing parameters were stable after 3 months of follow-up.

Discussion

Pacemaker implantation is the cornerstone treatment modality of symptomatic sinus node dysfunction, but its arduous task to decide the precise cardiovascular implantable electronic device when sick sinus dysfunction is seen in presence of hypertrophic cardiomyopathy because simultaneous assessment regarding risk of sudden cardiac death is crucial. Our patient didn't have any conventional risk markers nor is he a potential high risk subset of sudden cardiac death, so defibrillation was not required for our patient [1].

Chronic right ventricular apical pacing alters the electrical and mechanical activation pattern, modify the metabolic milieu leading to left ventricular remodeling, thus resulting in deleterious consequences of increased risk of heart failure, left ventricular contractile dysfunction, mitral regurgitation, and atrial fibrillation [2]. These urges the cardiologists to search for the alternative pacing sites like RV septum, RV outflow tract, but were not very promising. Conduction system pacing was found to be very encouraging in the last decade. In spite of having several clinical benefits, his-bundle pacing (HBP) which is considered to be an ideal physiological pacing site is not beyond limitations. Low R wave amplitude leading to over sensing of atrial or his signal and under sensing of ventricular signal, high HBP capture threshold during implant and delayed rise above 2.5 V at 1 ms in 25-30% patients, early battery depletion, high lead revision rate (8-10%) and loss of conduction tissue capture (upto 10%) [3], all these have made the electro physiologists to pursue a surrogate conduction system pacing site instead this technically challenging HBP. To address these issues a novel pacing strategy i.e. LBB pacing was introduced by Huang et al. [4]. Though data are limited but wide target and favorable histology (LBB is surrounded by myocardium) facilitate LBB area pacing (LBBAP) whereas narrow target and unfavorable histology (His is encased by fibrous insulation) have made HBP a demanding procedure [4]. Low and stable pacing thresholds, large R wave amplitude, lower lead revision rate (1%), longer battery longevity; all these have rendered this technique appealing without any doubt [5]. LBB pacing also improves LV synchrony and can be done in patients with sinus node dysfunction and AV block [6].

Now it is very interesting to note that we have done LBB pacing in patient having hypertrophic cardiomyopathy with sinus node dysfunction. Traditionally in hypertrophic obstructive cardiomyopathy, AV sequential pacing with short AV delay can reduce left ventricular outflow obstruction and improve symptom status in selected patients by several mechanisms like; RV pre-excitation alters the activation pattern of LV and thus there will be delayed septal activation and thickening, decreased LV hyper contractility, restriction of systolic anterior motion of mitral valve, interaction with left ventricular filling [7]. But chronic RV apical pacing results in LV systolic dysfunction [11]. This holds true for non-obstructive cardiomyopathy too. In our case we expected RV pacing in view of existent infra-Hisian disease. We opted for LBBAP to avoid development of LV systolic dysfunction. Also, our patient didn't have any LVOT obstruction, so we comfortably implemented our plan to do LBBAP. LV synchrony can be restored with this novel technique. Short term follow up indicating stable pacing parameters are also rewarding in our patient. Even there are reports indicating dramatic improvement of symptoms correction of LBBB and echocardiographic parameters when LBBAP is done in heart failure patients with LBBB [8]. Overall success rate is 80-94%; though septal perforation, lead dislodgement has been reported but it is technically feasible and safe alternative to HBP [8]. Nevertheless few things need to be mentioned: (1) how much will this LBB pacing lead be able to withstand the LV strain? answer is not known, (2) possibility of mural haematoma, coronary artery injury, lead removal, (3) select Secure lead 3830 (Medtronic, Mineapolis, MN) and the C315 His sheath are not specifically designed for LBBAP so, fine-tuning to increase the success rate may be required in near future, (4) long term data are needed to compare this strategy with age old RV apical pacing. While

doing LBBAP in HCM we should be aware of the following: (1) hypertrophied septum may provide hindrance to lead penetration specially if associated with fibrosis, (2) LV threshold may not be satisfactory if fibrosis is marked, (3) hyper contractility of LV is a feature of HOCM, so LV subendocardial lead placement may threaten the integrity of the lead in the long term, (4) amount of lead penetration will be more depending on amount of septal hypertrophy as compared to the norm which is 8-10 mm. In literature there is only one case report of LBB pacing in HCM is available so far [9]. We have successfully implanted the LBB pacing lead overcoming all the impediments associated with hypertrophic cardiomyopathy but it is worthwhile to mention that more evidences are needed to confirm the feasibility and safety of this novel conduction system pacing in hypertrophic cardiomyopathy.

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Disclosure of conflict of interest

None.

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