LBBAP Unmasks Severe Aortic Stenosis and Percutaneous Closure of Post-AVR Iatrogenic VSD

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A 38-year-old male with a bicuspid aortic valve and a right-sided dual-chamber pacemaker (implanted at age 10 for congenital atrioventricular block) presented with New York Heart Association (NYHA) Class III dyspnea and fatigue. Physical examination revealed bilateral basal rales. Electrocardiography demonstrated an atrial-sensed, ventricular-paced rhythm with a paced QRS complex duration of 185 ms (Figure 1a). Pacemaker interrogation confirmed 100% pacing dependency without arrhythmias. Despite optimal medical therapy, transthoracic echocardiography (TTE) revealed a dilated left ventricle [left ventricular end-diastolic diameter (LVEDD), 58 mm], severe systolic dysfunction, and a left ventricular ejection fraction (LVEF) of 31%. A heavily calcified bicuspid aortic valve with restricted motion was also observed (Figure 1c). Doppler assessment showed peak and mean transaortic gradients of 40 and 20 mmHg, respectively. Despite severe calcification, the reduced LVEF and discordant gradients supported a diagnosis of classical low-flow, low-gradient (LFLG) aortic stenosis. Given the priority to address pacing-induced cardiomyopathy (PICM) over performing dobutamine stress echocardiography (DSE), an upgrade to cardiac resynchronization therapy-pacemaker (CRT-P) via left bundle branch area pacing (LBBAP) was undertaken to improve ventricular mechanics and flow reserve. Under deep sedation, the procedure was performed using a Selectra 3D delivery sheath (Biotronik, Germany) and a Solia S60 active-fixation lead (Biotronik). During the procedure, non-selective left bundle branch pacing (NS-LBBP) was confirmed by a transition in QRS morphology from NS-LBBP to selective LBBP at 0.8 V, a V6 R-wave peak time of 68 ms (≤ 80 ms), and a V6-V1 interpeak interval of 45 ms. The ventricular pacing threshold was 0.9 V, with a unipolar pacing impedance of 721 Ω. A significant reduction in paced QRS duration from 185 ms to 105 ms was also observed (Figure 1b).[1]

At the 3-month follow-up, the patient's NYHA functional class improved to I-II, and signs of congestion had resolved, although exertional dyspnea persisted. TTE demonstrated significant reverse remodeling, with a LVEF of 54% and a LVEDD of 49 mm. This improvement unmasked true severe aortic stenosis, with transaortic gradients increasing to 86/45 mmHg and an aortic valve area of 0.8 cm². Following surgical aortic valve replacement (AVR) with a 23-mm On-X mechanical valve, the patient continued to experience dyspnea. Postoperative TTE confirmed normal LVEF and appropriate prosthetic valve function but revealed a new 5-mm iatrogenic subaortic ventricular septal defect (VSD) with a left-to-right shunt (Figure 1d). Cardiac computed tomography and catheterization (Qp/Qs: 1.19) confirmed the presence of the 5-mm iatrogenic VSD. Intra-procedural simulation was used to guide device sizing by assessing the distance to the prosthetic valve ring (Figure 1e, f). Although the Qp/Qs ratio of 1.19 indicated a relatively small shunt, the close anatomical proximity of the VSD to the mechanical prosthetic valve was considered to pose a significant long-term risk of infective endocarditis due to turbulent flow. Given this risk, along with the patient's persistent dyspnea, the heart team opted for percutaneous closure rather than conservative management. Under general anesthesia and transesophageal echocardiography guidance, a 6 × 4 mm VSD occluder (Konar MFO; Lifetech, China) was successfully implanted via the right femoral vein (Figure 1g, h). Postprocedural imaging demonstrated no residual shunt. At the 3-, 6-, and 12-month follow-up visits, the patient remained asymptomatic, LVEF was within normal limits, and no residual shunt was detected.

PICM results from electromechanical dyssynchrony caused by right ventricular apical pacing, with an estimated incidence ranging from 10% to 26%.[2] A high ventricular pacing burden and prolonged paced QRS duration are well-established risk factors for PICM development.[3] Both conduction system pacing and conventional cardiac resynchronization therapy using biventricular pacing (BiV-CRT) have been shown to improve systolic function and clinical outcomes.[4] In patients with heart failure and concomitant (LFLG) aortic stenosis, the hemodynamic improvement associated with BiV-CRT has

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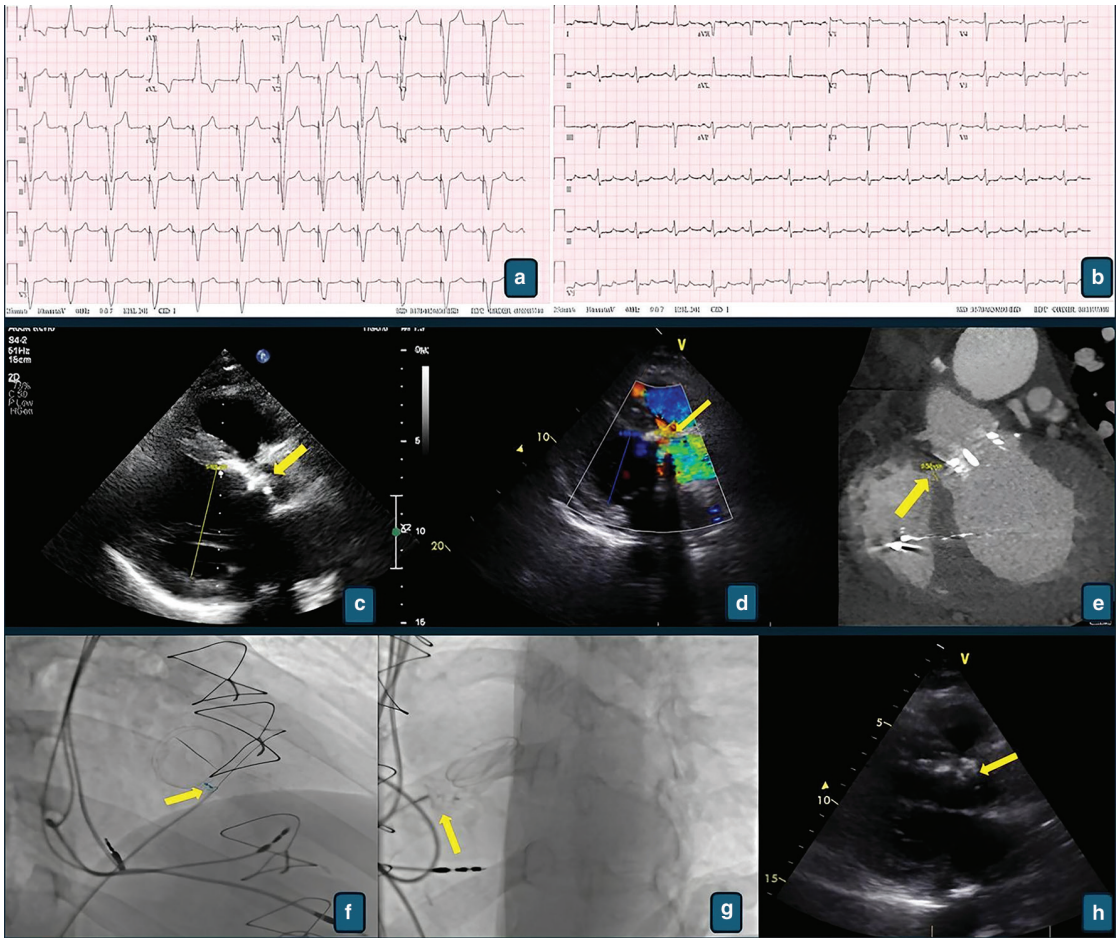

FIG. 1. (a) The 12-lead electrocardiogram demonstrating the right ventricular pacing with a wide paced QRS complex (185 ms). (b) The 12-lead electrocardiogram following left bundle branch area pacing demonstrating marked paced QRS narrowing to 105 ms. (c) Parasternal long-axis view showing dilated left ventricle with severely reduced ejection fraction and heavily calcified bicuspid aortic valve with restricted leaflet motion. (d) Apical four-chamber view with color Doppler demonstrating a 5 mm subaortic iatrogenic ventricular septal defect (VSD) with left-to-right shunt. (e) Two-dimensional reconstruction showing the anatomical relationship between the 23-mm On-X mechanical aortic valve prosthesis and the 5 mm subaortic VSD. (f) Fluoroscopic image demonstrating catheter passage through the VSD during pre-procedural simulation. (g) Fluoroscopic image showing successful deployment of the 6 x 4 mm VSD Occluder in optimal position without impingement on the mechanical aortic valve. (h) Transthoracic echocardiography confirming the closure of the VSD.

been reported to unmask the true severity of valvular stenosis by increasing stroke volume.[5,6] To the best of our knowledge, this is the first reported case demonstrating this unmasking phenomenon specifically following an upgrade to LBBAP.

The decision to implant a CRT-P rather than a CRT-defibrillator was based on the reversible, non-ischemic nature of the ventricular dysfunction. Evidence suggests that patients with PICM have a relatively low arrhythmic risk, and implantable cardioverter-defibrillator (ICD) therapy has not been shown to independently improve survival in this population.[7] Moreover, given the anticipated recovery of LVEF following correction of dyssynchrony, the indication for ICD implantation was expected to resolve after device upgrade, as observed in this case. LBBAP was preferred over conventional BiV-CRT via a coronary sinus (CS) lead for several reasons. First, CRT implantation from the right side is technically more challenging than the standard left-sided approach. In particular, CS cannulation is more difficult due to the steep and unfavorable entry angle into the CS ostium, often requiring specially curved catheters. Additionally, achieving stable lead positioning is more challenging because of the anatomical relationship between the superior vena cava and the right atrium. Consequently, procedure time and radiation exposure are typically increased.[8,9] Second, LBBAP enables more physiological ventricular activation through direct engagement of the His-Purkinje system. Comparative studies have demonstrated that LBBAP is associated with a narrower paced QRS duration, greater improvement in LVEF, and superior clinical outcomes, while avoiding the procedural risks associated with CS lead placement.[10,11]

Distinguishing between true severe and pseudosevere aortic stenosis in patients with reduced ejection fraction remains a major diagnostic challenge. Although DSE is recommended as the standard method for assessing contractile reserve,[12] its interpretation may be limited in patients with 100% pacemaker dependency, as a fixed

paced rhythm can confound chronotropic and inotropic responses to dobutamine. Moreover, even if DSE had confirmed true severe aortic stenosis in this young patient, proceeding directly to surgical AVR would have entailed substantial operative risk in the setting of severely reduced LVEF (31%). Therefore, optimization of ventricular function prior to any valvular intervention would remain the most appropriate next step, regardless of the DSE findings. In this context, the upgrade to LBBAP served not only as a therapeutic intervention but also as a physiological diagnostic tool. The subsequent increase in mean transaortic gradient from 20 to 45 mmHg confirmed true severe aortic stenosis without the need for pharmacological stress testing.

The proximity of the VSD to the mechanical prosthesis posed a technical challenge, necessitating meticulous preprocedural planning. In the absence of a standardized approach, we performed intra-procedural catheter-based simulation, which demonstrated a 5-mm distance between the VSD margin and the prosthetic valve ring, thereby guiding optimal device sizing. Previous studies have reported the feasibility of percutaneous VSD closure following prosthetic valve replacement.[13] In the present case, the device was successfully deployed without residual shunt or interference with the On-X mechanical valve.

This case highlights that LBBAP upgrade may not only reverse PICM but also unmask true severe aortic stenosis in patients with LFLG physiology, thereby guiding the timing of definitive valvular intervention. It further demonstrates that complex structural complications arising after surgical management can be effectively addressed using a percutaneous approach.

Informed Consent: Informed consent was obtained from the patient for publication of this case report and the accompanying images.

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