Pacing to treat low cardiac output syndrome following elective aortic valve replacement

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ABSTRACT

We report a case of low cardiac output syndrome caused by dynamic left ventricular (LV) outflow obstruction after aortic valve replacement (AVR). This recognized phenomenon probably occurs more frequently than appreciated, and the author suggests that this should be considered when managing patients with severe hemodynamic instability after AVR. In addition, we also focus on the fact that invasive pacemaker systems have significant effects on cardiac output augmentation postoperatively and in long-term management of patients with LV outflow tract (LVOT) obstruction following AVR. The possible mechanisms and subsequent treatments are discussed.

Key words: *Aortic valve replacement, DDD pacing, echocardiography*

INTRODUCTION

Dynamic left ventricular (LV) outflow tract (LVOT) obstruction occurs in a number of patients who have had aortic valve replacement (AVR). Following AVR for aortic stenosis, the LVOT gradient may worsen and can consequently cause significant hemodynamic compromise in the immediate postoperative period.

We present a case of LVOT obstruction complicating AVR. The cause of this was a combination of septal muscular hypertrophy and systolic anterior motion (SAM) of the mitral anterior leaflet. In this case, the patient was initially managed by Transvenous dual chamber (DDD) pacing. The mechanisms and treatment of this phenomenon are discussed here.

CASE REPORT

A 71-year-old man fit and well other than treated hypertension, was referred for AVR and coronary artery bypass grafting (CABG). Echocardiography demonstrated severe aortic stenosis with a heavily calcified aortic valve, peak gradient of 100 mmHg. The left ventricle was hypertrophied with good systolic function (septal thickness 16 mm). The coronary angiogram showed left anterior descending (LAD) and right coronary artery disease. His logistic Euro-SCORE was 3.29%.

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The patient underwent CABG plus AVR, using left internal mammary artery (LIMA) to the left anterior descending (LAD) coronary artery and Saphenous Vein Graft (SVG) to the right coronary artery (RCA). The aortic valve was replaced with St. Jude Epic standard 23 mm. On-table transesophageal echocardiography(TOE) post-Cardiopulmonary bypass (CPB) demonstrated LVOT obstruction, no SAM, and postoperative mean gradient of 18 mmHg. The patient was weaned off of CPB without any inotropic support. He was extubated in intensive care unit and 2 h later he developed low cardiac out put syndrome assessed by Swan–Ganz catheter cardiac index (CI) was 1.5. The patient was started on intravenous fluid filling, adrenalin plus noradrenalin inotropic support and received few bolus doses of metaraminol.

TOE showed obstruction of the LVOT and the presence of SAM of the mitral valve [Figure 1a].

He had temporary epicardial atrial and ventricular leads *in situ* (routinely inserted during valve operations), the atrial leads were only intermittently capturing. He was in sinus bradycardia and left bundle branch block (LBBB). A decision was made to institute dual chamber pacing a DDD pacing system was expeditiously inserted and he was sequentially paced with an A–V delay of more than 100 ms. The patient immediately showed marked hemodynamic

improvement and after a few days stay in hospital was discharged home. TOE revealed no SAM [Figure 1b].

DISCUSSION

In certain patients following AVR, the dynamic intraventricular flow velocities are worsened and create a significant LVOT with severe hemodynamic compromise. The locations of these abnormal flow velocities include the LVOT and the area below the mitral valve itself and above the insertion of the papillary muscles[1] The mechanisms of abnormal flow velocities (AFVs) after AVR for stenosis in the presence of a hypertrophied left ventricle are either systolic cavity obliteration or SAM of mitral valve.[1] In patients with aortic stenosis, the afterload is increased and the ventricle is often small and hypertrophied. Post-AVR, the after load dramatically decreases and that further decreases the LV volume and increase fibres shortening.[2] These factors may combine to cause cavity squeezing, which may lead to increased systolic flow velocities. Increasing myocardial contractility and vasodilatation by the use of inotropic and other agents may cause unloading of the left ventricle and worsen these abnormal flow velocities. Systolic anterior motion of the mitral valve apparatus may also independently cause AFV.[1]

Echocardiography factors predictive of postoperative AFVs are as follows:

- LV geometry and function
- High transvalvular gradient
- Discrete asymmetric hypertrophy
- Raised septal to posterior wall thickness ratio.

These factors and our observations suggest that small, hyperdynamic, and asymmetrical hypertrophied left ventricles are prone to develop AFV whenever afterload is reduced following AVR.

Treatment should initially be targeted toward manipulation of the physiology; dual chamber pacing causes beneficial atrioventricular delay in LVOT obstruction. Pre-excitation of the right ventricular apex is achieved by short AV delay DDD pacing. The atria are sensed and then trigger right ventricular pacing ahead of spontaneous AV conduction.[3] In addition to an altered ventricular contraction pattern, pacing results in redistribution of wall stress, probably causing modification of coronary blood flow.[4] The altered LV activation pattern with late activation of the basal part of the septum and decreased LV contractility increases the LV systolic diameter and reduces the systolic anterior motion of the mitral valve, which leads to a lowering of the LVOT gradient.[5] Pre-existing left bundle branch block is compatible with severe LVOT obstruction, and DDD pacing is also beneficial in this subgroup. DDD pacing reduces both resting and functional LVOT obstruction.[6] In this case, after 3 months, obstruction was reduced and symptoms were improved.

In conclusion, this condition should be considered, after an uncomplicated AVR, if hemodynamic instability develops particularly in the presence of risk factors for postoperative AFV. The following measures should be followed.

1. Increase preload with volume to force the ventricle to work according to the Frank–Starling curve
2. Avoid using inotropic agents or systemic vasodilators to maintain stroke volume without cavity squeezing
3. DDD permanent pacing should be considered if the patient has LVOT obstruction confirmed on TOE and LBBB on preoperative ECG
4. AFV occurs most often at midcavity level rather than in the LVOT, so septal myectomy should be avoided even in the case of risk factors for developing postoperative AFV
5. Doppler echocardiography examination should be carried out, with special attention paid to turbulent flow

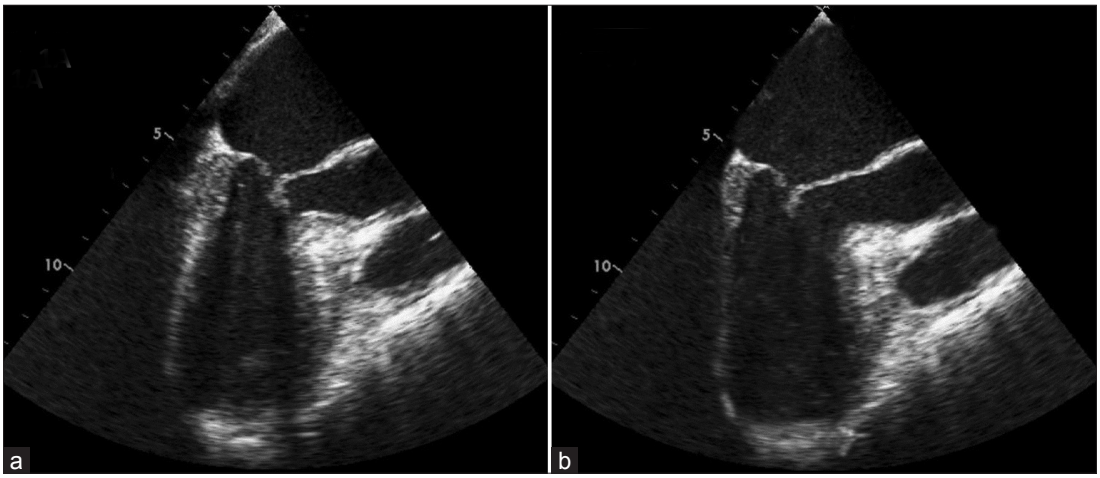

Figure 1: a) TOE shows obstruction of the LVOT caused by SAM of the anterior leaflet of mitral valve and septal hypertrophy b) TOE shows absence of systolic anterior motion (SAM) of mitral valve after pacing

in the midcavity or LVOT. Because image quality in the postoperative phase is often poor, a transesophageal approach is usually required

6. Volume loading and administration of β-blockers should be considered in the case of AFV with hemodynamic consequences.

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