Alcohol septal ablation in hypertrophic cardiomyopathy leading to permanent complete heart block; a case report

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Introduction

Hypertrophic cardiomyopathy (HCM) is a disease state characterised by unexplained left ventricular hypertrophy in the absence of another cardiac or systemic disease which can explain it (1). HCM is inherited as an autosomal dominant trait and has diverse clinical manifestations ranging from asymptomatic patients with normal life span to sudden cardiac death (2). A considerable proportion of patients with HCM have left ventricular outflow tract obstruction (LVOT) at rest or on provocation. Medical management of HCM involves beta blockers and calcium channel blockers. Patients with LVOT obstruction, refractory to medical management are considered for septal reduction therapy with alcohol septal ablation (ASA) or myomectomy. Complete heart block is a known complication of ASA which is usually transient. We report a HCM case with LVOT obstruction needing permanent pacing following ASA.

Case report

In 2005, a 51-year-old male presented with shortness of breath on moderate exertion and systolic murmur was found on clinical examination. His ECG revealed bizarre ST and T wave changes. Transthoracic echocardiogram (TTE) confirmed the diagnosis of HCM with interventricular septum diameter of 20mm, LVOT obstruction of 85mmHg and systolic anterior motion of mitral valve. He was started on beta blockers following which the patient became asymptomatic. Meanwhile, the family screening with echocardiography revealed negative results.

In January 2013, patient presented with worsening dyspnoea accompanying chest pain on moderate exertion. He underwent coronary angiogram which showed a prominent first septal branch in left anterior descending artery (LAD). There was no evidence of syncopy and his left ventricular (LV) ejection fraction was satisfactory during the course of illness. Since he continued to be symptomatic despite maximum medical therapy it was decided to proceed with ASA.

Pre procedure simultaneous pressure recordings at LVOT and LV apex revealed a difference of 72mmHg. We proceeded with the coronary angiogram to identify the first septal perforator of LAD which was subsequently engaged with 2mm over the wire balloon (OTW). With the inflation of OTW balloon the pressure gradient across LVOT and LV reduced dramatically to 20mmHg. Area supplied by the first septal branch and absence of backflow to LAD were confirmed with contrast injection. A temporary pace maker (TPM) lead was placed in the right ventricular apex anticipating atrioventricular blocks. 1ml of 100% alcohol was slowly injected with 0.5ml at a time and kept for 15 minutes. There was a dramatic reduction in pressure gradient across the LVOT (4mmHg) and the patent went into complete heart block and became dependent on TPM.

During the seven day stay at coronary care unit, the patient continued to have intermittent complete heart block and was TPM dependent. After consulting cardiac electrophysiology team, it was decided to insert a permanent dual chamber pacemaker. However following the insertion of permanent pacemaker he became asymptomatic and gradually

resumed his usual life style. TTE done 1month later revealed only 30mmHg pressure gradient across the LVOT which is expected to reduce in next few months.

Discussion

ASA is accepted as a reliable modality of treatment for symptom relief in patients with HCM since 1994 (1,3). The indications for ASA are refractory symptoms despite medical treatment, resting or provocable pressure gradient across LVOT more than 50mmHg and inter-ventricular diameter more than 18mm. As our patient fulfilled these criteria we considered him for ASA (1,3).

Tributaries of the first septal branch of the LAD have significant individual variations. They supply a large area of myocardium including right ventricular septum and the LV apex. Therefore, the exact localisation of the septal branch supplying basal septum is paramount in ASA. Anatomical variations of LAD can result in post procedural atrioventricular conduction defects but they usually recover spontaneously within 24 - 48 hours while 10 - 20% will persist and require permanent pacing (1). The myocardial contrast echocardiography (MCE) during procedure gives an opportunity to select the target septal branch in each individual, minimizing above complication (3,4). Thus the reduction of the incidences of post procedure pacemaker implantation is a proven advantage of MCE guided ASA which is yet to establish in our country (4).

Alternatively the dual chamber pacing is considered as an effective mode of treatment for symptom relief in HCM (1). The right ventricular apical pacing with the maintenance of atrioventricular synchrony has been reported to decrease the LVOT pressure gradient. Therefore, our patient was benefitted in both aspects with permanent pacing.

References

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