



## Pre-Operative Systolic Anterior Motion of the Mitral Valve in a Patient Undergoing Mitral Valve Replacement

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### Abstract

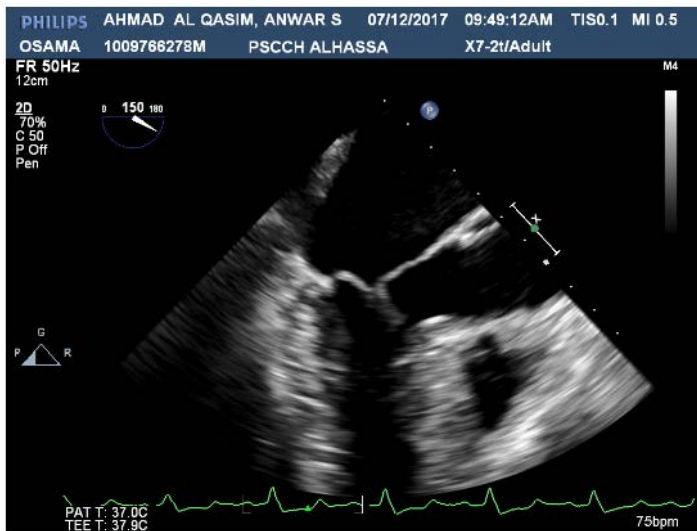
A patient with myxomatous mitral valve prolapse underwent mitral valve repair due to severe mitral regurgitation. Pre-operative echocardiography demonstrated Systolic Anterior Motion (SAM) of the mitral valve. As the patient was considered at high risk of developing post-repair SAM, he was operated on using surgical techniques aimed at lowering the risk of this complication. Despite this, post-repair SAM did develop and could only be eliminated by a mitral valve replacement. A brief review of the literature regarding the prediction, prevention and treatment of post-operative SAM is presented.

### Introduction

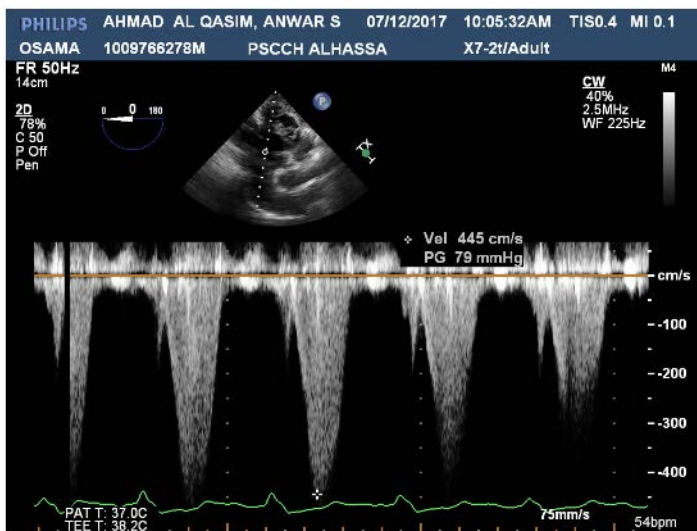
Systolic Anterior Motion (SAM) of the Mitral Valve (MV) can be a life-threatening condition. The SAM can result in severe left ventricular outflow tract obstruction and/or mitral regurgitation and is associated with an up to 20% risk of sudden death. The mechanisms of SAM are complex and depend on the functional status of the ventricle. The SAM can occur in the normal population, but is typically observed in patients with Hypertrophic Obstructive Cardiomyopathy (HOCM) or following MV repair. We report a unique case of a patient with myxomatous MV disease and MV regurgitation with SAM. As the patient was considered at high risk of developing post-MV repair SAM, he was operated on using surgical techniques aimed at lowering the risk of this complication. Despite this, post-repair SAM did develop and could only be eliminated by a mitral valve replacement.

### Case Presentation

A 46-year-old male patient, Diabetic, was referred for transthoracic echocardiography due to progressive dyspnoea. Clinically, with a Pan Systolic murmur in the aortic area. Notably, there was no history of hypertension or family history of cardiomyopathy. Electrocardiogram showed sinus tachycardia. Transthoracic echo done for the patient revealing good LV systolic function but high gradient on LVOT (MAX PG 135 mmHg), SAM (long anterior mitral leaflet obstructing LVOT), moderate to severe mitral regurgitation. Chest X Ray was normal. Pre-Cardio Pulmonary Bypass, Transoesophageal Echocardiography (TEE) revealed normal left ventricular contractility, mild concentric left ventricular hypertrophy, SAM with LVOT maximum Pressure Gradient of 79 mmHg, and moderate to severe mitral valve regurgitation (Figure 1A and 1B) (Video).



**Figure 1A:** Mid Esophageal (ME) long axis view showing typical SAM with LVOT obstruction.



**Figure 1B:** Deep trans-gastric view showing increase Pressure Gradient (PG) across LVOT with maximum recording PG 79 mm Hg.

MV repair included: plication of the posterior mitral leaflet, sliding annuloplasty with implantation of a large 33mm annuloplasty ring (Duran Ancore Medtronic), an intra-operative, post-repair TEE examination revealed persistent SAM with more increased pressure Gradient across LVOT, The maximum gradient recorded was 139 mm hg , which caused haemodynamic compromise despite of normal loading condition and discontinuation of inotropic agents. Another trial was done by Alfieri technique, Edge to Edge (EE) but with no improving of pressure gradient. However, despite the undertaking of these

preventive measures and after failed of two trials for repair, we decided to replace the valve (St. Jude medical Mechanical valve 27mm). Aortotomy was done, the septum was inspected, found to be hypertrophied, excised and hegar dilator size 18 was smoothly passed through the LVOT. During two years follow up, the patient remained completely asymptomatic and without echocardiographic signs of recurrence of LVOT Obstruction.

## Discussion

There are a number of medical causes of SAM including diabetes mellitus, myocardial infarction, hypertension and general anaesthesia. The mechanism of SAM in diabetics is complex .Nevertheless, there may be a specific mechanism as suggested by studies of beta-adrenoceptor stimulation .Upon beta-adrenoceptor stimulation by intravenous infusion of isoproterenol, and echocardiography revealed SAM in 65 % of diabetic subjects [1]. SAM can be a complication of MV repair surgery, occurring in approximately 5-10% of patients [2]. This relatively high incidence of SAM can be partially explained by haemodynamic changes that occur during this operation, such as increased contractility and/or reduced preload or afterload. Studies suggested that certain intrinsic properties of the MV itself and its orientation relative to the interventricular septum are associated with an increased risk for the development of SAM [3].

Maslow et al defined the preoperative parameters associated with an increased risk of developing post-MV repair SAM [3]. These include: (a) a lower ratio of the anterior to posterior MV leaflets length; and (b) a smaller distance between the coaptation point of the MV leaflets and the interventricular septum. Another feature in SAM-prone patients is MV leaflet elongation, a common finding in patients with myxomatous MV disease. Algorithms are available to guide the decision-making process when this complication is detected soon after weaning from cardiopulmonary bypass [4]. As a first step, intravascular volume is gradually expanded and any inotropic drug is discontinued. As a second step,  $\beta$ -blockers are administered. In a small percentage of patients, despite all the measures described above, significant SAM persists and surgical revision is required.

SAM can be eliminated with the EE technique [5, 6]. With excellent long-term results. Another rapid procedure to correct SAM is to reduce the height of the posterior leaflet. A partial ring seems to prevent SAM more than a complete one, as the complete rigid ring causes a narrower systolic LVOT diameter and favouring SAM [7]. Refractory SAM can also be treated by sliding plasty, if this procedure was not carried out at the initial repair. Under these circumstances, a prosthetic ring larger than the one previously implanted is often inserted [8]. Mitral valve repair is superior to replacemant, especially for younger and female patients. If SAM is severe and persistent despite exhaustive medical and surgical attempts at eliminating SAM, mitral valve replacemant can be the

only option [9]. In this setting, care must be taken to properly resect any remaining mitral tissue, to prevent SAM recurrence [10].

We believe that the case presented highlights some important aspects of post-MV repair SAM. First, the mild thickening of the LV walls in the absence of systemic hypertension suggests that our patient had an underlying hypertrophic cardiomyopathy, a disease which is commonly associated with MV SAM. Second, some patients, post-repair SAM develops even after the use of surgical techniques aimed at preventing its development. In the case presented here, despite the use of a large annuloplasty ring and shortening of the posterior leaflet then Alfieri technique, post-repair SAM developed. Finally, despite the good results of the Alfieri technique edge-to-edge for the surgical treatment of post-MV repair SAM. In our case this technique failed and we are obliged to replace the mitral valve.

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