The Lesser of Two Evils: Incidental Aortic Stenosis During Mitral Valve Replacement

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Introduction: Intraoperative transesophageal echocardiography frequently surprises us with incidental findings that can require intervention. We present a case where the discovery of severe aortic stenosis during a mitral valve replacement created a clinical dilemma: leave the patient with a stenotic aortic valve or replace it and potentially cause patient-prosthesis mismatch and AV dissociation? A thorough evaluation of the aortic valve in the context of the overall patient allowed us to make a decision that provided maximal benefit with minimal risk.

Case Presentation: A 65 yr old female with a history of rheumatic heart disease and atrial fibrillation presented to the hospital with new onset congestive heart failure. Cardiac echocardiographic workup revealed rheumatic deformity of the mitral valve with severe mitral stenosis and mitral regurgitation, a moderately thickened aortic valve with minimal stenosis, and a normal LVEF. Cardiac catheterization revealed single vessel coronary artery disease. After medical optimization, the patient was scheduled to undergo MVR, CABG, MAZE procedure, and left atrial appendage ligation. Intraoperative TEE, while confirming the presence of severe rheumatic mitral valve disease, also revealed a severely thickened, critically stenotic aortic valve (area = 0.7 cm²) with a peak gradient of 18 mmHg. Complicating matters, there was significant mitral annular calcification and a small aortic annulus. We decided to proceed without an AVR and reassess the aortic valve post-bypass. The aortic valve area improved from 0.7 cm² (stroke volume = 27 mL) pre-bypass to 1.1 cm² (stroke volume = 45 mL) post-bypass with improved leaflet excursion. The patient had an uneventful postoperative course and recently underwent a follow-up TTE showing mild AS.

Discussion: Grading the severity of AS can be difficult. Conventionally used methods (planimetry, continuity equation, and Gorlin equation) generally produce useful data, but are fundamentally different and rely on inaccurate assumptions. Furthermore, such methods frequently produce discordant data, where a valve may be deemed severely stenotic by area but not by gradient according to the ACC/AHA Guidelines.(1) In such patients, a low stroke volume index is thought to be the culprit.(2) Two types of low flow AS have been described in the literature. In pseudo-aortic stenosis, a depressed LV leads to a low cardiac output and reduced transvalvular flow unable to sufficiently open the aortic valve leaflets. In these patients, improving LV function may improve the degree of AS without surgical intervention.(3) In paradoxical low-flow low-gradient AS with a normal EF, a chronically increased afterload from a stenotic valve causes myocardial remodeling, leading to a reduced stroke volume despite a normal EF. These patients benefit from AVR.(4) We believe that our patient had a variation of pseudo-aortic stenosis, where replacing the mitral valve and thus increasing stroke volume through the aortic valve improved the degree of AS.