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Editorial

Subvalvular disease in patients undergoing balloon mitral valvotomy: A strong base is not always good[☆]



Balloon mitral valvotomy or percutaneous transvenous mitral commissurotomy (BMV/PTMC) is currently the treatment of choice for severe and/or symptomatic mitral stenosis. It has few contraindications, namely presence of a thrombus in left atrium, significant mitral regurgitation (MR), and severely calcified commissures in the mitral apparatus (though many would operate now even with commissural calcification¹). BMV as a procedure has its own problems however. Long-term event rates including death or re-intervention (either redo BMV or open heart surgery) approach 30–50% at 5- to 10-years follow-up. More dangerous is the threat of acute severe MR, mostly due to anterior mitral leaflet tear (2–10% of all cases). Taken together, these two form the Achilles' heel of this procedure. Therefore, a pre-intervention assessment by transthoracic echocardiogram is crucial for selecting the right patient, as both the complications are rather best avoided. To standardize this evaluation, several different scoring systems have been proposed which are useful in predicting long-term efficacy of the procedure, acute MR, or both.

While the determinants of acute MR and long-term procedural success do overlap, there are some important distinctions. It would seem that excessive fibrosis and/or calcification in the mitral valvular apparatus and its components individually predict acute failure to open the valve as well as long-term success. This would include the leaflets, commissures, and the subvalvular tissue. For the prediction of acute MR, there is considerable disagreement however.

In this issue of the journal, Dr Balghat et al.² present the results of a prospectively carried out study (of patients undergoing BMV in their tertiary care institution) of preoperative echocardiographic evaluation of a new subvalvular grading technique in comparison to traditional echocardiographic scores and its correlation with outcome of procedural failure (unsuccessful dilatation or severe MR requiring urgent surgery). Besides the usual evaluation and assessment of Wilkins' score³ and MR Score,⁴ the authors also carried out a detailed echocardiographic evaluation of subvalvular apparatus attached to each of the papillary muscle, postero-medial, and antero-lateral (or infero-septal and supero-lateral, respectively

in attitudinally correct nomenclature). This was then used to make a simple score (SVA score) with three categories: 1 being lack of severe involvement of both papillary muscles (and chordate attached to them), 2 denoting severe involvement of either papillary muscle, and 3 being severe involvement of both. This evaluation was prospective, carried out by 4 cardiologists blinded to the procedure, and quality of data checked by inter- and intra-observer variation assessment.

Of the 356 individuals undergoing BMV over a one-year period, procedural success was 88%, while 8% had severe MR requiring surgery, and in 4% the valve could not be satisfactorily dilated. The authors noted that nearly all of these (95%) had an SVA score of 3, and also all patients with a score of 3 developed complications. This was true irrespective of leaflet morphology, which varied depending on the type of complication. The overall performance of SVA score was much better than Wilkins and Padiol MR score. A good correlation of echocardiographic findings was seen with pathology in excised valves of patients undergoing surgery. Of note, the valve specimen also showed that while extensive commissural disease was always accompanied by severe subvalvular disease, the converse was not always true. The authors subsequently hypothesize that commissural involvement is only a surrogate for the extent of subvalvular disease, which is the only real predictor of procedural success as well as severe post-procedural MR. They also believe that it is the inadequacy of SVD evaluation (for e.g., evaluating only one papillary muscle rather than both), which is the cause of poor performance of scores like the Wilkins Score and the Padiol MR Score. They propose the utility of their SVA score as being crucial for identifying those patients who should not be offered BMV.

The study has been done with rigorous methodology and has acceptable inter- and intra-observer variability, meaning that once understood, the new SVA score is likely to be reproducible. The question that remains to be answered is whether the new SVA score provides any incremental value to our pre-BMV assessment of mitral valve or even replace the traditional scores employed. Starting with the Wilkins Score, published in 1988,³ there have been several scores that

[☆] This editorial is pertaining to the article: Subvalvular apparatus and adverse outcome of balloon valvotomy in rheumatic mitral stenosis.

attempt to predict the outcomes of BMV. Soon after the Wilkins article, data emerged from several investigators that the score (probably still the commonest used around the world) had several flaws. One of them was the omission of information about the commissural fusion and calcification in the score, which seemed to dichotomize procedural success among those with 'good' Wilkins score. This was subsequently acknowledged and incorporated by the Harvard group into a new score in 2014, which takes into account the above, along with commissural asymmetry and subvalvular disease.⁵ The other major flaw with Wilkins Score is its inability to predict severe post-procedural MR necessitating urgent or semi-urgent mitral valve surgery. This then prompted development of new scores for the prediction of severe MR, one of them being the Padiol score,⁴ designed by the same Harvard group. However, these scores have only average performance characteristics and are poorly reproducible across diverse populations and studies.

It is pertinent here to look mechanistically at the causes of failure of this otherwise excellent procedure. Stiff valvular apparatus (due to diffuse thickening, calcification, severe subvalvular disease, or all of them) is less likely to give way causing either failure or under-dilatation. Less acute gain in the valve area as well as fundamentally 'bad' valve will also cause more restenosis in the long run. This is why the Wilkins Score is good at this kind of prediction. However, it is the unevenness of fibrosis/calcification in the valvular apparatus that will predispose certain points (mostly the thinner part of

the leaflet, and other interfaces of the mitral valve complex sometimes) for tearing and severe regurgitation. As the thicker portions refuse to give way, the brunt of asymmetric and large forces generated during balloon dilatation is borne by these areas. This asymmetry can exist within the leaflets and commissures, as well as between two components, for e.g., excessive thickening in commissures and/or subvalvular tissue in relation to leaflets with the latter being less thick. It is the AML that mostly tears at a 'hinge' point. This is why the Wilkins score will not predict procedural severe MR while the Padiol score (which takes into account leaflet heterogeneity) or the proposed score by Bhalgat et al. might. Obviously, an inappropriately large balloon (or balloon in high pressure zone) can cause regurgitation with diffuse thickening also.

While most of the investigators believe that leaflet and commissural thickening have a strong correlation with long-term outcomes, it is the effect of subvalvular disease that has been a major point of contention. While some, including the present study, claim subvalvular disease to be the major determinant of success, others (including from India⁶) have found no correlation between the two. Besides differing population and procedural characteristics, another reason for this discrepancy can be the way in which the subvalvular apparatus is assessed on echocardiogram. As pointed out by the authors, evaluation of only one of the two papillary muscles can potentially cause both under-diagnosis and over-diagnosis of severe SVD (Fig. 1). However, contrary to the assertion of the authors, the original Wilkins score³ actually

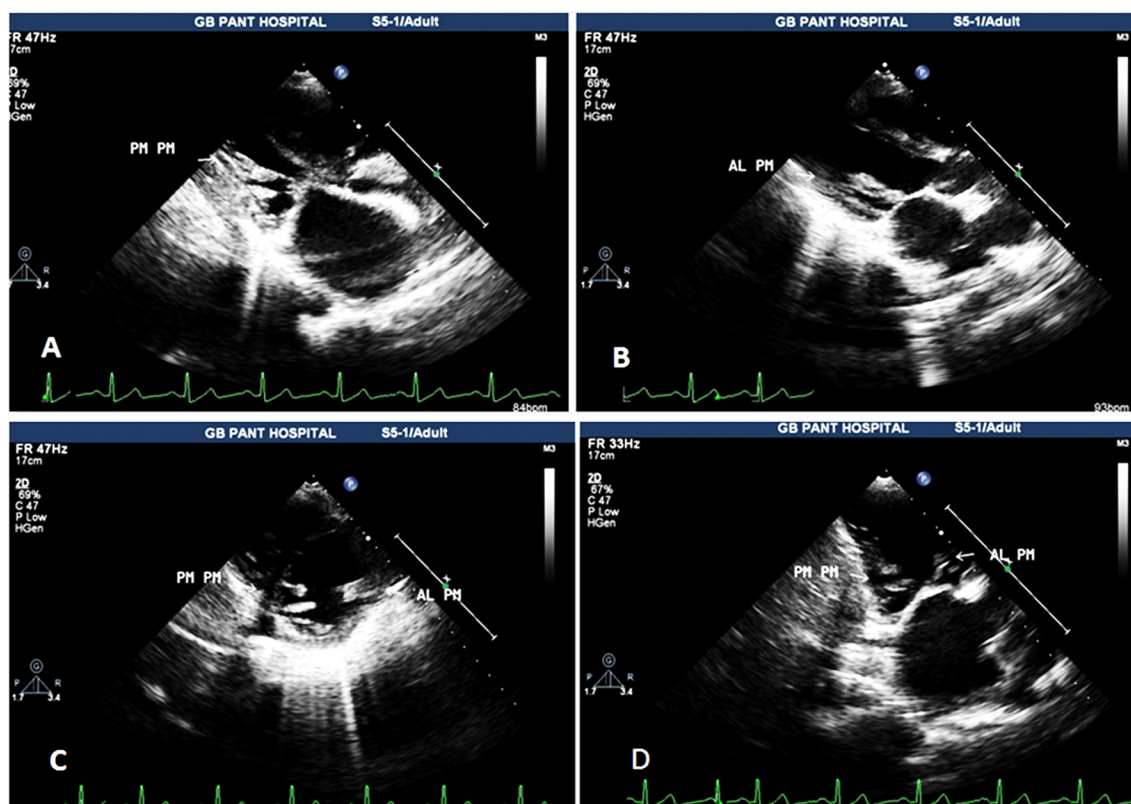


Fig. 1 – Assessment of subvalvular apparatus in mitral stenosis. While both the papillary muscles and chordae have disease, it is more extensive on the AL papillary muscle side. (A and B) Modified tilted parasternal long axis views; (C) parasternal short axis view at the papillary muscle level; (D) modified apical 2 chamber view showing both papillary muscles. AL: Anterolateral; PM: Posteromedial; PM: papillary muscle.

recommends inspecting both the papillary muscles (though it does not provide guidance for scoring in cases, where the involvement is asymmetric), and this has been the practice of many others,⁷ including this author. Nevertheless, it is a fact that the extent of subvalvular disease is often underdiagnosed and this has been repeatedly demonstrated in studies of patients undergoing emergency surgery for severe MR following BMV. These studies invariably show underappreciated severe subvalvular fibrosis and/or commissural calcification along with tears in the AML.^{8,9}

The authors are to be commended for developing a simple tool for subvalvular disease assessment, that is easy to standardize and perform and shows a good correlation with surgical findings in their study. They have rightly put the focus back on SVA, which should just not be relegated to a brief assessment of a single papillary muscle in parasternal long axis view. They are also correct in stating that SVD is a strong predictor of severe MR, a fact substantiated by previous studies as well. However, to conclude that, this assessment alone is sufficient for picking out the unsuitable BMV candidates, and that all other valvular abnormalities are in fact surrogate for severe SVD, might be premature. While it is likely that severe involvement of both papillary muscles might indeed preclude a successful procedure, absence of this parameter is unlikely to safeguard against complications. Their finding of commissural fibrosis/calcification being only a surrogate for SVA involvement may be due to small sample size and may not be reproducible. A larger sample size and validation of their scoring in an independent population would have been reassuring.

Hence, pending long-term data from the authors and independent validation of this score, we would much rather incorporate this elegantly simple and objective SVD evaluation in our assessment of mitral valve complex, instead of replacing current practices entirely. It is clear however that a 'strong base' is indeed counter-productive for the success of BMV, and the authors are to be lauded for putting the spotlight back on the subvalvular apparatus.

Conflicts of interest

The authors have none to declare.

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