The balloon impasse sign in percutaneous transvenous mitral valvuloplasty

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Abstract

Percutaneous transvenous mitral valvuloplasty (PTMV) is an established therapy for rheumatic mitral stenosis (MS). While the Wilkins score standardizes the description of valve anatomy and predicts successful PTMV, echocardiographic assessment has some limitations. The ‘balloon impasse’ sign is the inability to cross a stenotic valve with a deflated Inoue balloon. This sign was described in the 1990s as an indicator of severe subvalvular thickening (regardless of the echocardiographic findings), portending an increased risk of severe mitral regurgitation (MR) post-PTMV. Despite its implications for management, it has been seldom reported. A 57-year-old woman with symptomatic, severe MS and a Wilkins score of 7 underwent PTMV. The ‘balloon impasse’ sign was observed when attempting to cross the stenotic valve. When the balloon was fully inflated, severe MR was noted, and the patient required mitral valve replacement. This case demonstrates the continued importance of the ‘balloon impasse’ sign and its implications for the therapeutic efficacy of PTMV.

INTRODUCTION

Percutaneous transvenous mitral valvuloplasty (PTMV) is an established therapy for rheumatic mitral stenosis (MS) [1]. PTMV is primarily considered in the setting of favorable valve morphology in patients that are either symptomatic with severe MS or asymptomatic with very severe MS [1]. While echocardiography and the Wilkins score provides a standardized means of describing mitral valve (MV) anatomy and predicting successful PTMV [2], the characterization of the subvalvular anatomy echocardiographically is imperfect. As such, other clues to challenging MV anatomy, such as the ‘balloon impasse’ sign [3], may indicate the presence of severe subvalvular disease during PTMV.

CASE REPORT

A 57-year-old Caucasian female presented with recurrent episodes of decompensated heart failure and progressive New York Heart Association (NYHA) class III dyspnea. She was a remote smoker with hypertension and antiphospholipid antibody syndrome complicated by prior peripheral embolic events. Medications included warfarin, telmisartan, digoxin, metoprolol and furosemide. Invasive angiography revealed no obstructive coronary artery disease. Transesophageal echocardiogram (TEE) confirmed the presence of severe rheumatic MS (mitral valve area 1.0 cm² [normal 4–6 cm²], mean gradient 17.6 mmHg [normal ≤1 mmHg]) and severe pulmonary hypertension (right ventricular systolic pressure 87.6 mmHg [normal <35 mmHg]).
Figure 1: Echocardiographic valvular assessment. (A, B) Pre-procedural transthoracic echocardiogram demonstrating leaflet calcification, ‘hockey stick’ appearance of mitral valve leaflet opening in long-axis and characteristic ‘fish mouth’ appearance in short axis. TEE demonstrating (C) flow acceleration through the mitral valve consistent with severe MS and (D) mild mitral regurgitation (orange upwards jet). (E) PTMV with inflated balloon visualized within the valvular apparatus. (F) Severe mitral regurgitation post PTMV (upwards aliasing jet).

The Wilkins score was documented as 7 (range 4–16) on the basis of leaflet thickness (1/4), mobility (2/4), calcification (2/4) and subvalvular thickness (2/4) without significant mitral regurgitation (Fig. 1A–D). Hence, PTMV was pursued to increase the MV area. The patient provided written consent for the procedure and subsequent publication.

During the procedure, there was notable difficulty crossing the stenotic valve with the deflated balloon catheter—the ‘balloon impasse’ sign (Fig. 2A, Video) [3]. During the first two inflations, a notable waist remained in the balloon at the level of the stenotic valve. Despite a reduction in mean gradient from 14 to 6 mmHg without any significant MR, the increase in MV area was suboptimal (Fig. 2B). On the final inflation, the balloon assumed its fully inflated shape (Fig. 2C). At this point, a large V-wave was also noted on the left atrial pressure tracing, and the intra-procedural TEE confirmed the presence of severe MR. Hemodynamics remained stable, and an intra-aortic balloon pump was placed. The patient underwent same-day mechanical MV replacement with intra-operative findings of A2 chordal rupture in the setting of a severely calcified subvalvular apparatus—not appreciated on the screening echocardiograms. Following valve replacement, the patient was transferred to the cardiac surgical intensive care unit with normal mechanical MV function and recovered. In clinical follow-up to 3 years post-procedure, she remained NYHA class I with normal pulmonary pressures and mechanical prosthesis function.

DISCUSSION

The ‘balloon impasse’ sign refers to the inability of a deflated Inoue balloon to cross a stenotic valve and portends an increased risk of severe MR post-PTMV during standard inflation protocols [4]. Previously established as a sign of severe subvalvular thickening, initial studies have noted shortened chordae and thickened papillary muscles out of keeping with echocardiographic findings when this sign is observed [3, 5]. Indeed, while the Wilkins score provides some insight into MV anatomy, it remains relatively subjective, and assessment of the subvalvular apparatus largely depends on the views achieved on screening echocar-
diography. If this sign is encountered, operators should not pursue aggressive strategies to cross the valve and instead exchange for a smaller balloon to mitigate the risk of severe MR [3]. Despite its implications for management, since first being described in the early 1990s, further reports have been sparse [4, 6].

CONCLUSION
Clinicians performing PTMV should be cognizant of the ‘balloon impasse’ sign. Recognition of this sign should signal the likelihood of significant subvalvular disease and raise the possibility of severe MR post-PTMV, requiring alternative approaches to avoid subsequent surgical intervention.

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CONFLICT OF INTEREST STATEMENT
No conflict of interest.

ETHICAL APPROVAL
Not applicable.

CONSENT
The patient provided informed consent prior to undergoing the procedure.

GUARANTOR
Benjamin Hibbert

REFERENCES
1. Nishimura RA, Otto CM, Bonow RO, Carabello BA, Erwin JP 3rd, Guyton RA et al. AHA/ACC guideline for the management of patients with valvular heart disease: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines. J Am Coll Cardiol 2014, 2014;63:e57–e185.
2. Wilkins GT, Weyman AE, Abascal VM, Block PC, Palacios IF. Percutaneous balloon dilatation of the mitral valve: an analysis of echocardiographic variables related to outcome and the mechanism of dilatation. Br Heart J 1988;60:299–308.
3. Lau KW, Hung JS. Balloon impasse: a marker for severe mitral subvalvular disease and a predictor of mitral regurgitation.
in Inoue-balloon percutaneous transvenous mitral commissurotomy. Catheter Cardiovasc Diagn 1995;35:310–9 discussion 20.

4. Hung JS, Lau KW, Lo PH, Chern MS, Wu JJ. Complications of Inoue balloon mitral commissurotomy: impact of operator experience and evolving technique. Am Heart J 1999;138:114–21.

5. Hung JS, Chern MS, Wu JJ, Fu M, Yeh KH, Wu YC et al. Short- and long-term results of catheter balloon percutaneous transvenous mitral commissurotomy. Am J Cardiol 1991;67:854–62.

6. Turgeman Y, Atar S, Suleiman K, Bloch L, Rosenfeld T. Percutaneous balloon mitral valvuloplasty in patients with severe mitral stenosis and low transmitral diastolic pressure gradient. Int J Cardiovasc Interv 2003;5:200–5.