Severe Tricuspid Regurgitation Leads to Underestimation of the Severity of Mitral Stenosis by Doppler

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INTRODUCTION

Rheumatic mitral stenosis (MS) is a progressive cardiac disease characterized by the obstruction of blood flow through the mitral valve (MV).[1] Echocardiography is crucial for assessing the severity of MS. Low flow through the MV can lead to the underestimation of MS severity by echocardiographic methods.[2] Here, we present a case illustrating the underestimation of MS severity due to severe tricuspid regurgitation (TR).

CASE PRESENTATION

A 50-year-old patient with known rheumatic MS and severe TR presented with shortness of breath (New York Heart Association functional class III), easy fatiguability, and palpitations. Electrocardiography revealed atrial fibrillation (AF) with controlled ventricular rate, and chest radiography indicated signs of pulmonary congestion. The patient had previously undergone closed mitral commissurotomy and percutaneous balloon mitral commissurotomy. The preoperative transthoracic echocardiographic examination showed a dilated right atrium, right ventricle, tricuspid annulus (indexed diameter 23 cm/m^2), and inferior vena cava, with severe eccentric secondary TR (Figure 1, Video 1). The measured RV systolic pressure was 35 mm Hg, the mean transmitral gradient (TMG) was 4 mm Hg, and the measured MV area (MVA) was 1.16 cm^2 by two-dimensional planimetry (Figure 1C), and 1.8 cm^2 by the pressure half-time (PHT) method. As the patient had symptomatic MS and severe TR, MV replacement with tricuspid valve (TV) repair was planned.

During surgery, following the induction of anesthesia, the monitored parameters showed AF with a ventricular rate of 84 beats/min, arterial blood pressure of 106/54 mm Hg, pulmonary artery pressures of 43/17 mm Hg, and a cardiac index of 1.2 L/min/m^2. Prebypass transesophageal echocardiography showed a dilated right atrium, right ventricle, and tricuspid annulus, with qualitatively estimated moderate to severe secondary TR, and an average mean mitral TMG of 2 to 3 mm Hg (Figure 2). Three-dimensional (3D) echocardiographic examination revealed a MVA of 0.52 cm^2 by the volume-rendering technique and 0.85 cm^2 by the multiplanar reconstruction method (Figure 3). During examination in the midesophageal long-axis view, leftward bowing of the interventricular septum (IVS) was noted in diastole (Video 2). Additionally, the midesophageal four-chamber view also demonstrated leftward bowing of the IVS (Video 3). These findings were consistent with right ventricular volume overload. Subsequently, the MV was replaced with a mechanical prosthesis, and the TV was repaired using a 28-mm annuloplasty ring. After bypass, transesophageal echocardiographic examination did not show leftward bowing of the IVS. The rest of the postoperative course was uneventful. At 1-year follow-up, the patient was performing daily housework with mild TR, and the prosthetic MV function was normal.

DISCUSSION

Echocardiographic methods used to assess the severity of MS include mean TMG, PHT, and two-dimensional and 3D planimetry. The TMG depends on the flow velocity across the MV during diastole.[3] PHT measures the time taken for the peak left atrial to left ventricular (LV) pressure gradient to decrease by half and is influenced by the rate at which increasing LV diastolic pressure and decreasing left atrial pressure come into equilibrium.[2] Both TMG and PHT are influenced by the flow velocity across the MV, which in turn is affected by the pressures proximal and distal to the MV and the rate of change in these pressures. Left atrial pressure is influenced by factors such as left atrial wall stiffness, diastolic time, net atrioventricular compliance, and the volume of blood ejected into the pulmonary vasculature and left atrium during systole.[4] Clinical conditions such as AF with a fast ventricular rate and volume overload (e.g., pregnancy, mitral regurgitation) increase the left atrial pressure. Conversely, interatrial shunt and low cardiac output decrease pressure proximal to the stenotic MV.[5] Conditions such as diabetes, chronic hypertension, obesity, and coronary artery disease can affect operational compliance of the left atrium because of atrial fibrosis,[6] leading to increased pressure during filling and rapid decrease of pressure during emptying. In hypertensive and elderly patients, the left ventricle often becomes hypertrophied and less compliant. In the presence of reduced LV compliance, even moderate filling of the left ventricle can lead to a sharp increase in LV pressure. This decreased compliance of the left ventricle and the left atrium can result in a shortened PHT and a reduced mean TMG.

In the presence of severe TR, the right ventricular stroke volume is ejected into both the pulmonary circulation and the right atrium, which raises the right atrial pressure and hinders systemic venous return. This results in dilatation of the vena cava and further reduction in MV flow. Our patient had severe TR, with a severely decreased cardiac index (1.2 L/min/m^2) and a relatively low pulmonary arterial

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Keywords: Mitral stenosis, Tricuspid regurgitation, Doppler gradients, 3D echocardiography, TEE

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2468-6441

https://doi.org/10.1016/j.case.2024.08.003

VIDEO HIGHLIGHTS

Video 1: Two-dimensional transthoracic echocardiography, apical four-chamber zoomed view focused on the right heart with color flow Doppler, demonstrates severe TR.

Video 2: Two-dimensional transesophageal echocardiography, midesophageal long-axis (138°) view without *(left)* and with *(right)* color-flow Doppler of the mitral and aortic valves, demonstrates movement of the IVS toward the left ventricle (right ventricular volume overload), restricted opening of the MV leaflets with mild mitral regurgitation, normal aortic valve leaflet motion, and spontaneous echo contrast in the left atrium. A cauterization artifact is seen.

Video 3: Two-dimensional transesophageal echocardiography, midesophageal four-chamber (2°) view focused on the right heart, demonstrates a dilatated right atrium, right ventricle, and tricuspid annulus with leftward bowing of the ventricular and atrial septa consistent with volume overload; spontaneous echocardiographic contrast is seen in the left atrium.

View the video content online at www.cvcasejournal.com.

pressure (43/17 mm Hg). These parameters reflects a state of low flow through the pulmonary circulation, reduced blood volume reaching the left atrium, resulting in low flow across the MV and a decreased TMG[2] and PHT. Furthermore, the pulmonary blood flow is affected by general anesthesia, intermittent positive pressure ventilation, and positive end-expiratory pressure. These factors further decrease the systemic venous return and increase the right ventricular afterload, compounding the decrease in pulmonary blood flow and the flow across the MV. Additionally, these effects are exaggerated if a large tidal volume is selected for ventilation. The summative effect on TR is variable and may cause increased TR severity because of increased afterload to the right ventricle or may cause decreased TR severity because of reduced systemic venous return. We believe that these physiologic factors ultimately resulted in a significant decrease in TMG and PHT in our patient.

In our patient, the averaged mean TMG indicated mild to moderate MS, but the clinical symptoms did not align with the TMGs or the MVA measured by planimetry. The severe TR and decreased pulmonary blood flow was an important reason for low TMGs and overestimation of MVA. Additionally, a leftward bowing of the IVS was noted. In the presence of severe TR, the right atrial blood volume is severely increased in systole; during diastole, the increased right atrial volume flows across the TV into the right ventricle, whereas, the flow across the MV is decreased because of stenosis and low flow. An overloaded right ventricle and an underfilled left ventricle create a pressure differential and leftward bowing of the IVS into the left ventricle. Silbiger[2] previously reported leftward bowing of the IVS in the presence of severe TR. Kresoja *et al.*[7] studied the effects of TV edge-to-edge repair in 20 patients with severe TR and heart failure with preserved ejection fraction. They found that following the procedure, there was an increase in LV end-diastolic volume, without a significant change in LV end-diastolic pressure (LVEDP). There was also a significant increase in the ratio of LV

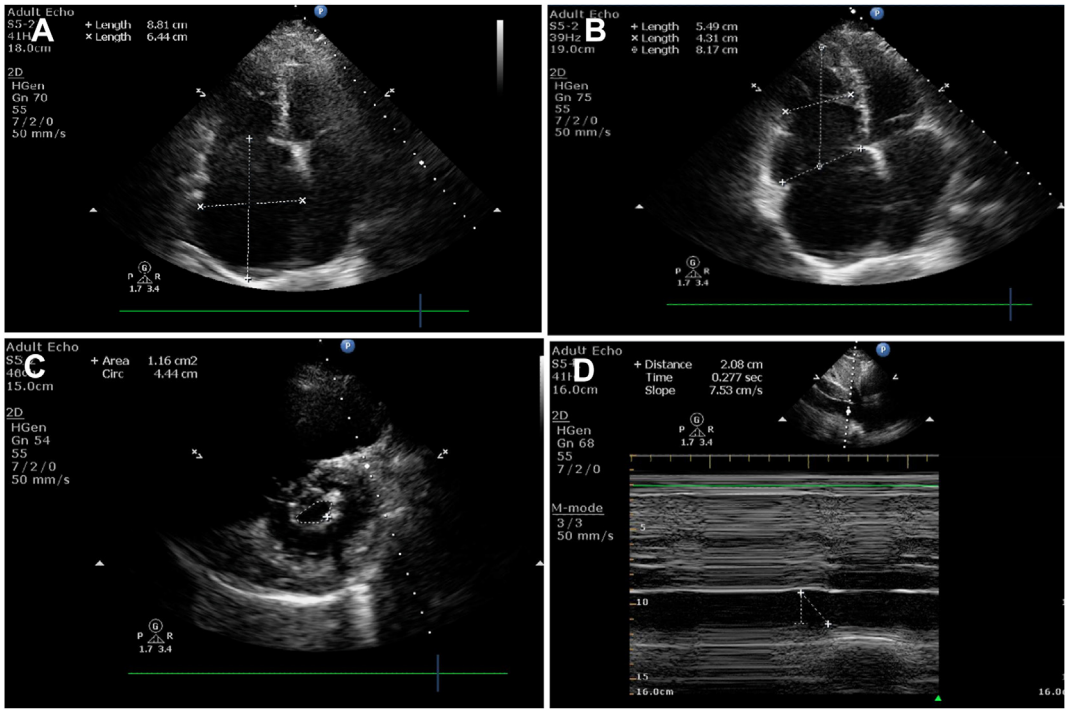

Figure 1 **(A** and **B)** Two-dimensional (2D) transthoracic echocardiography (TTE), apical four-chamber diastolic view with right heart focus, demonstrates a dilated right atrium and right ventricle. **(C)** Two-dimensional TTE, parasternal short-axis diastolic view of the MV leaflet tips, demonstrates moderate stenosis using a calculated MVA by the 2D planimetry method. **(D)** Two-dimensional TTE-guided M-mode, subcostal view, demonstrates a dilated, plethoric inferior vena cava with minimal respiratory variation.

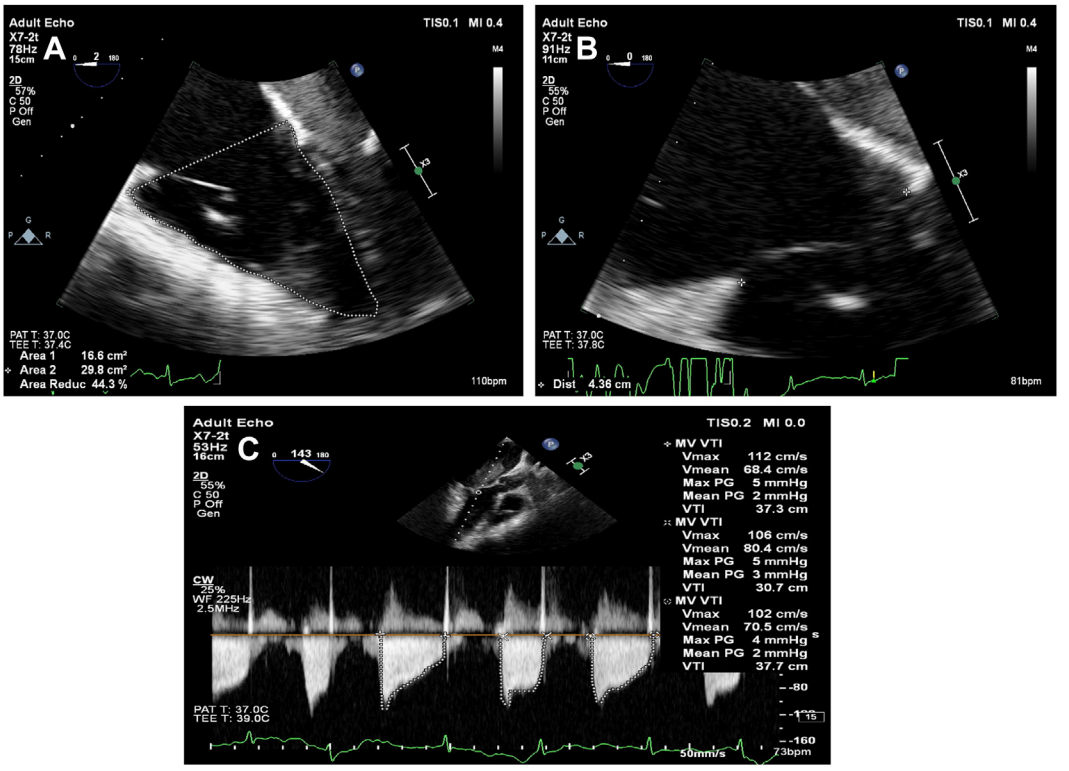

Figure 2 Two-dimensional transesophageal echocardiography, midesophageal zoomed four-chamber (2°) diastolic views focused on the right heart, demonstrates a dilated right ventricular end-diastolic area **(A)** and TV annular dimension **(B)**. **(C)** Midesophageal long-axis view with continuous-wave Doppler across the MV demonstrates a variable but normal mean gradient.

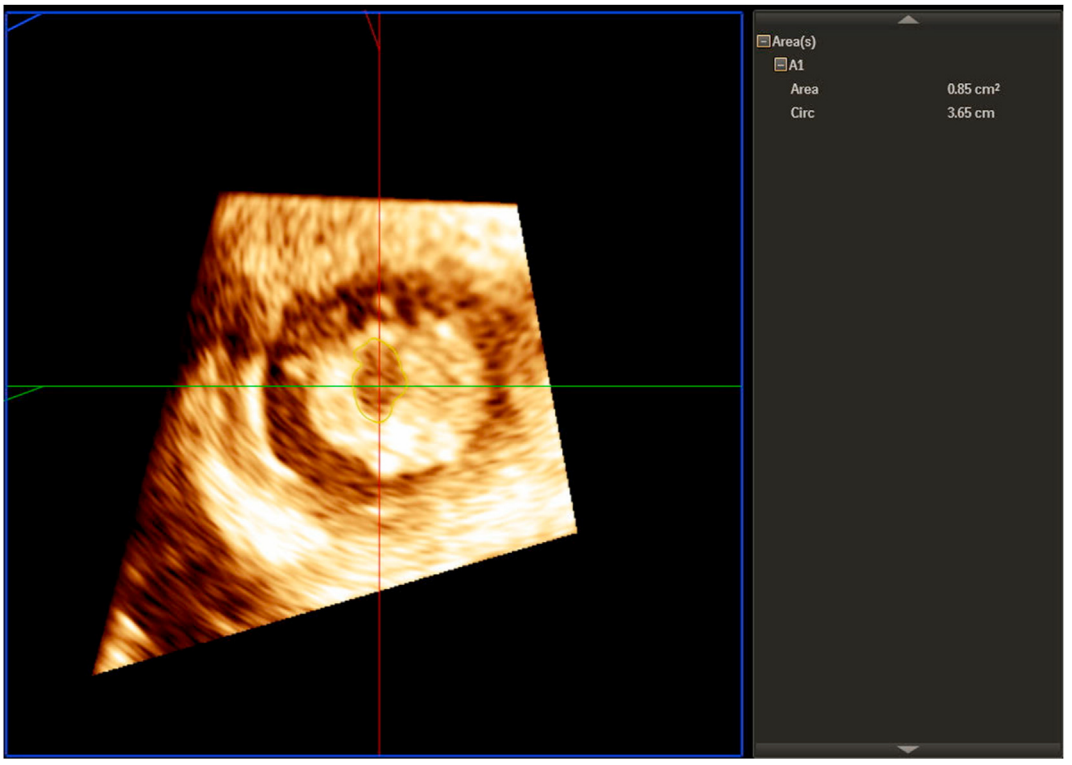

Figure 3 Three-dimensional zoomed multibeat acquisition of the MV, multiplanar reconstruction short-axis display, demonstrates calculation of MVA using the anatomic planimetry method.

end-diastolic volume to LVEDP, suggesting improved filling of the left ventricle. These changes were associated with an improvement in LV stroke volume per LVEDP, a decrease in the leftward bowing of IVS, and an increase in TMG. If a decrease in leftward bowing of IVS results in an increase in TMG, a leftward bowing, as observed in our patient, would decrease TMG and overestimate the MVA, provided, flow across the MV is not compromised. The concept is similar to "low-flow, low-gradient MS," in which flow across the MV is reduced because of decreased LV compliance.[2]

CONCLUSION

In the setting of an abnormal flow across the MV, the flow-dependent Doppler methods like TMG and PHT overestimate the MVA. MS severity should preferably be evaluated using flow-independent measurements such as 3D planimetry.

ETHICS STATEMENT

The authors declare that the work described has been carried out in accordance with The Code of Ethics of the World Medical Association (Declaration of Helsinki) for experiments involving humans.

CONSENT STATEMENT

Complete written informed consent was obtained from the patient (or appropriate parent, guardian, or power of attorney) for the publication of this study and accompanying images.

FUNDING STATEMENT

The authors declare that this report did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

DISCLOSURE STATEMENT

The authors report no conflict of interest.

SUPPLEMENTARY DATA

Supplementary data related to this article can be found at https://doi.org/10.1016/j.case.2024.08.003.

REFERENCES

1. Zühlke L, Engel ME, Karthikeyan G, Rangarajan S, Mackie P, Cupido B, et al. Characteristics, complications, and gaps in evidence-based interventions in rheumatic heart disease: the Global Rheumatic Heart Disease Registry (the REMEDY study). Eur Heart J 2015;15:1115-22.
2. Silbiger JJ. Advances in rheumatic mitral stenosis: echocardiographic, pathophysiologic, and hemodynamic considerations. J Am Soc Echocardiogr 2021;34:709-7221.
3. Nishimura RA, Otto CM, Bonow RO, Carabello BA, Erwin JP 3rd, Guyton RA, et al. 2014 AHA/ACC guideline for the management of patients with valvular heart disease: executive summary: a report of the American College of Cardiology/American Heart Association task force on practice guidelines. Circulation 2014;129:2440-92.
4. Neema PK, Rathod RC. Pulmonary artery hypertension in mitral stenosis: role of right ventricular stroke volume, atrio-ventricular compliance, and pulmonary venous compliance. J Anaesthesiol Clin Pharmacol 2012;28:261-2.
5. Tsiaras S, Poppas A. Mitral valve disease in pregnancy: outcomes and management. Obstet Med 2009;2:6-10.
6. Morris DA, Gailani M, Vaz Pérez A, Blaschke F, Dietz R, Haverkamp W, et al. Left atrial systolic and diastolic dysfunction in heart failure with normal left ventricular ejection fraction. J Am Soc Echocardiogr 2011;24:651-62.
7. Kresoja KP, Rosch S, Schöber AR, Fengler K, Schlotter F, Bombace S, et al. Implications of tricuspid regurgitation and right ventricular volume overload in patients with heart failure with preserved ejection fraction. Eur J Heart Fail 2024;26:1025-35.