



Systolic aortic regurgitation in rheumatic carditis: Mechanistic insight by Doppler echocardiography



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ARTICLE INFO

Article history:

Received 14 May 2016

Accepted 15 August 2017

Available online 24 August 2017

Keywords:

Systolic aortic regurgitation
Reversed aorto-ventricular pressure gradient
Flail mitral valve

ABSTRACT

Background: Aortic regurgitation (AR) usually occurs in diastole in presence of an incompetent aortic valve. Systolic AR is a rare phenomenon occurring in patients with reduced left ventricular systolic pressure and atrial fibrillation or premature ventricular contractions. Its occurrence is a Doppler peculiarity and adds to the hemodynamic burden.

Aim: Rheumatic carditis is often characterised by acute or subacute severe mitral regurgitation (MR) due to flail anterior mitral leaflet and elongated chords. In patients with acute or subacute MR, developed left ventricular systolic pressure may fall in mid and late systole due to reduced afterload and end-systolic volume and may be lower than the aortic systolic pressure, causing flow reversal in aorta and systolic AR.

Material and methods: 17 patients with acute rheumatic fever were studied in the echocardiography lab during the period 2005–2015. Five patients had severe MR of which two had no AR and hence were excluded from the study. Three young male patients (age 8–24 years) who met modified Jones' criteria for rheumatic fever with mitral and aortic valve involvement were studied for the presence of systolic AR. **Results:** In presence of acute or subacute severe MR, flail anterior mitral valve and heart failure, all three showed both diastolic and late systolic AR by continuous-wave and color Doppler echocardiography.

Conclusion: Systolic AR is a unique hemodynamic phenomenon in patients with acute rheumatic carditis involving both mitral and aortic valves and occurs in presence of severe MR.

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1. Introduction

Aortic regurgitation (AR) usually occurs during diastole when ventriculo-aortic pressure gradient is reversed and the aortic valve is incompetent. Diastolic AR may continue in systole in case the developed pressure in the left ventricle (LV) is lower than that of the aorta. This hemodynamic change is possible during atrial fibrillation with fast ventricular rate and in presence of premature ventricular contractions provided the aortic valve shows mal-coaptation.^{1,2} Presence of systolic dysfunction perpetuates it. It is also possible to observe this phenomenon in the failing LV in sinus rhythm wherein the ventricle develops pressure very slowly during isovolumic contraction period which is prolonged and the aortic systolic pressure exceeds the LV systolic pressure during early systole.³ In presence of atrial fibrillation and premature

ventricular contractions, aortic regurgitation can be pan-cyclic if the aortic valve fails to open for antegrade flow ejection during systole. In isolated LV systolic dysfunction associated with incompetent aortic valve, diastolic AR continues in early systole to a variable extent but is not observed in late diastole. In acute severe mitral regurgitation (MR), the LV systolic pressure falls rapidly during late systole.⁴ and may potentiate systolic AR in presence of aortic valve disease. Some patients of rheumatic carditis develop acute or subacute MR due to chordal rupture or flail anterior mitral leaflet combined with annular dilatation.⁵ A subset of patients with acute or subacute MR may present solely with new-onset dyspnea, without evidence of impending cardiovascular collapse.⁶ Aortic valvulitis with some degree of AR is frequent in rheumatic carditis.⁷ The purpose of this study was to detect systolic AR due to pressure gradient reversal in patients with rheumatic carditis who had acute severe MR.

2. Material and methods

Over a period of 2005–2015, 17 patients with first episode of acute rheumatic fever fulfilling modified Jones' criteria⁸ were evaluated by echocardiography. Of these, five patients had acute or

Abbreviations: LV, left ventricle; LA, left atrium; AV, aortic valve; AO, aorta; DAR, diastolic aortic regurgitation; SAR, systolic aortic regurgitation; RA, right atrium; RV, right ventricle; MR, mitral regurgitation.

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subacute severe MR. Three of these patients had associated aortic valvulitis with AR and were included in further analysis.

2.1. Case #1

This 24-yr old male presented with fever and polyarthralgia of two months duration. He had progressive dyspnea for the last 15 days. Physical examination revealed a thin-built person in respiratory distress; pulse rate was 110/min with low volume, supine blood pressure of 110/84 mmHg, distended jugular veins, mild hepatomegaly, basal rales, hyperdynamic apical impulse and a pan-systolic apical murmur. A 12-lead electrocardiogram showed sinus tachycardia, left ventricular hypertrophy by voltage criteria and PR interval of 200 msec. The chest skiagram revealed enlarged cardiac silhouette and pulmonary venous congestion. His biochemistry was as follows: hemoglobin 9.2 gm%, ESR 92 mm/first h, white cell count of 7800/mm³ with 82% polymorphs, hs-CRP 19 mg/L anti-streptolysin titre of 320 units, negative throat and blood cultures.

2D echocardiography showed mildly enlarged left atrium, low normal LV systolic (ejection fraction 48%) function, flail anterior mitral leaflet with a torn primary chord, mildly thickened aortic valve and no pericardial effusion (Fig. 1). Color Doppler flow mapping revealed severe eccentric mitral regurgitation.

Continuous-wave (CW) Doppler examination of the LV inflow showed dense spectrum of MR with triangular appearance and rapid late systolic deceleration (Fig. 2). CW interrogation of the LV outflow tract revealed two Doppler spectra of retrograde flow into the LV (Fig. 2A), one in diastole (peak velocity of 5 m/s with rapid deceleration) and the other in systole (peak velocity of 2 m/s). M-mode of color Doppler flow across the LV outflow (Fig. 2C) showed systolic retrograde flow in later half of systole penetrating just beyond the opened aortic valve and diastolic AR extending deep

into the LV cavity. Fig. 3 compares CW spectra of LV inflow and outflow in two non-simultaneous but equal cycle length beat.

The patient was treated with corticosteroids and decongestive therapy. No aspirin was used. He subsequently underwent successful mitral valve replacement with a bileaflet mechanical valve. Aortic valve was spared as the aortic regurgitation was not considered severe.

2.2. Case #2

This 10-year old male child presented with fever, generalised fatigue and dyspnoea of 3 weeks' duration. Physical examination revealed sinus tachycardia (pulse rate 106/minute, regular), supine blood pressure of 90/76 mmHg, presence of mitral regurgitation and he was in heart failure. He had polymorphonuclear leucocytosis (white cell count 12600/cmm), ESR of 52 mm, positive CRP and anti-streptolysin O titre of 250 units. Throat and blood cultures were negative for group A streptococci. 2D echocardiography revealed enlarged LV, mildly thickened aortic valve, flail anterior mitral leaflet with severe eccentric MR (Fig. 4). CW interrogation of the LV outflow showed dense spectrum of DAR and late systolic retrograde flow with slow acceleration and a peak velocity of 1.1 m/s (Fig. 5).

The patient was treated with corticosteroids and bed rest. He showed regression of MR and AR and disappearance of SAR.

2.3. Case 3#

This 8-year old boy was being treated for rheumatic carditis in pediatric service. He present in heart failure with heart rate of 120/minute, had severe MR and had anti-streptolysin O titre of 400 units. 2D echocardiography with color and CW interrogation showed slightly flail anterior leaflet, severe mitral regurgitation,

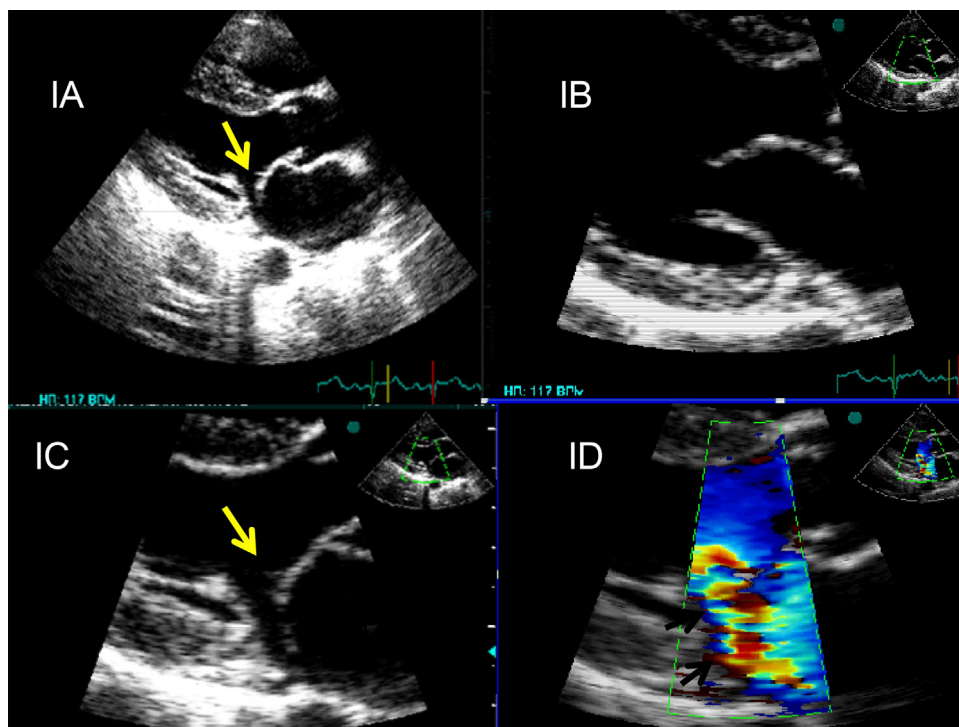


Fig. 1. 2D echocardiographic parasternal long axis views showing flail anterior mitral leaflet (yellow arrows) and severe eccentric mitral regurgitation by color Doppler (black arrows, ID).

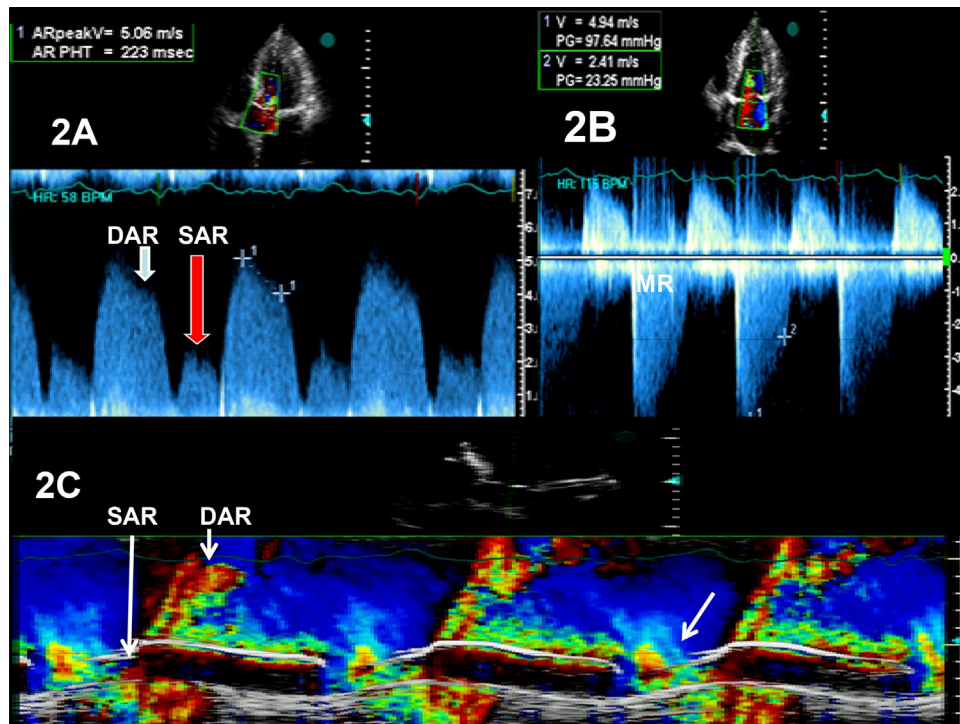


Fig. 2. CW interrogation of the LV outflow tract showing diastolic AR (DAR) and systolic AR (SAR) in Fig. 2A. Fig. 2B shows triangular CW spectrum of MR with rapid deceleration. Fig. 2C shows color Doppler M-mode spectrum of the LV outflow revealing SAR and DAR flows.

mild diastolic AR and brief signal of systolic retrograde flow in the LV outflow suggestive of SAR (Fig. 6). This jet signal of 1.0 m/s velocity was present in late systole distinct from that of early diastolic mitral flow and pan-diastolic spectrum of DAR. He made uneventful recovery following treatment with mild residual MR.

3. Discussion

Mitral regurgitation (MR) occurs in 84–94% cases of acute rheumatic carditis and the jet is usually directed postero-laterally due to involvement of anterior mitral leaflet.⁷ Multiple

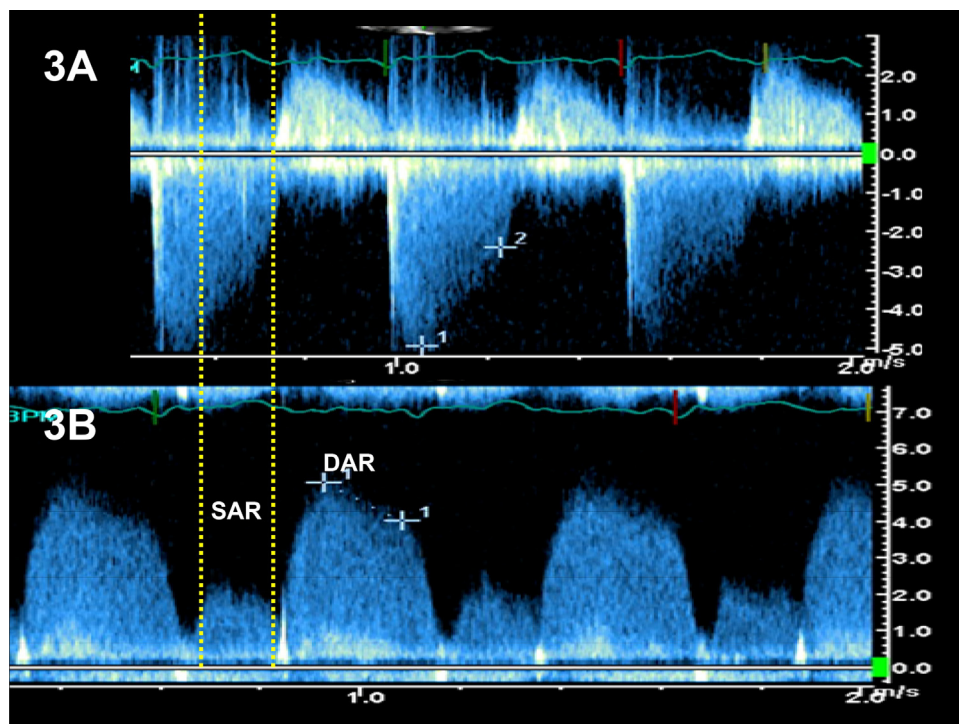


Fig. 3. CW spectrum of MR (3A) compared to that of LV outflow (3B). Note that SAR appears when the LV-LA gradient rapidly falls during later half of the systole. Maximum velocity of SAR is 2 m/s. A peak diastolic velocity of 2.2 m/s across the LV inflow suggests severe MR.

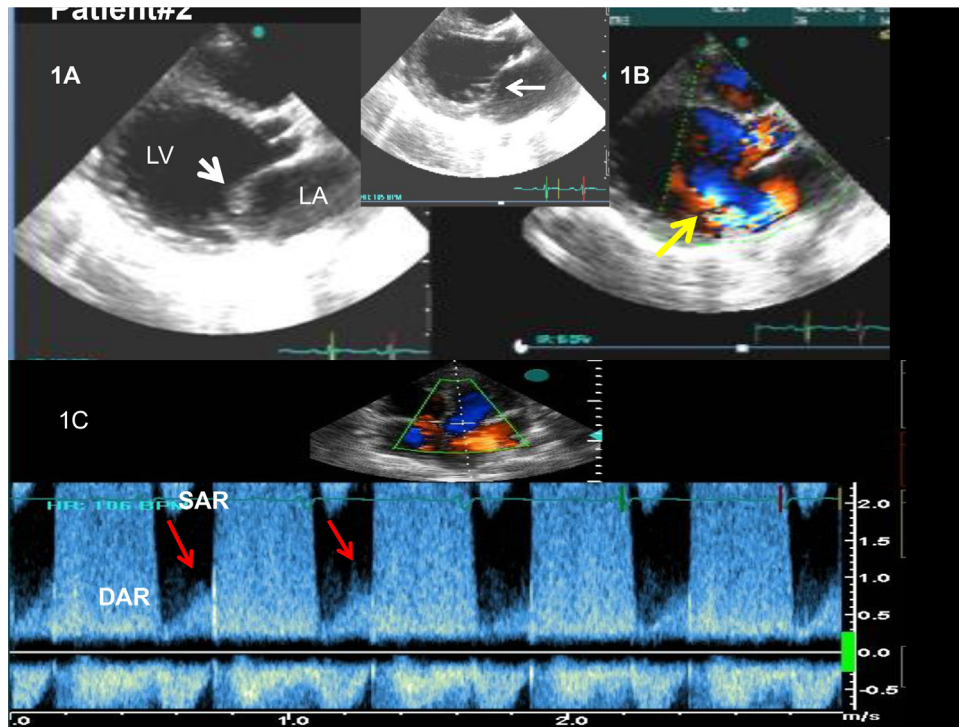


Fig. 4. Upper panels contains 2D echocardiographic parasternal long axis views with flail anterior mitral leaflet (white arrow) and severe eccentric MR (yellow arrow). Lower panel (1C) shows dense spectrum of diastolic AR (DAR) and systolic AR (SAR, red arrows) in late systole.

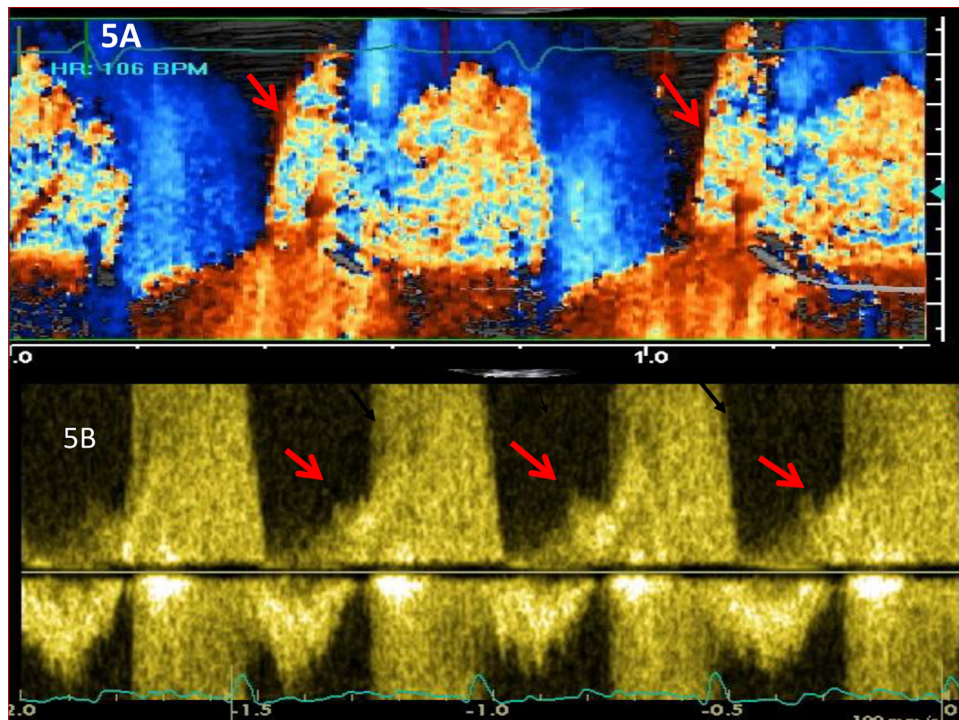


Fig. 5. Color Doppler M-mode (5A) and continuous-wave Doppler spectrum of the LV outflow showing late systolic AR (red arrows) with dense spectrum of diastolic AR.

mechanisms are involved in its pathogenesis but elongated and/or torn chords with flail anterior mitral leaflet play a major role.^{5,6,8} Severe MR occurs in 9–31% cases but aortic regurgitation is less common.⁷ MR in rheumatic carditis is usually acute or subacute and late systolic pressure falls significantly due to low end-systolic

wall stress and end-systolic volume.⁴ Unlike other causes of acute MR, there is no shock-like state in rheumatic carditis. In presence of aortic valvulitis, there may be systolic AR in association with acute or subacute severe MR due to late systolic pressure gradient reversal. The flow of systolic AR is confined to the left ventricular

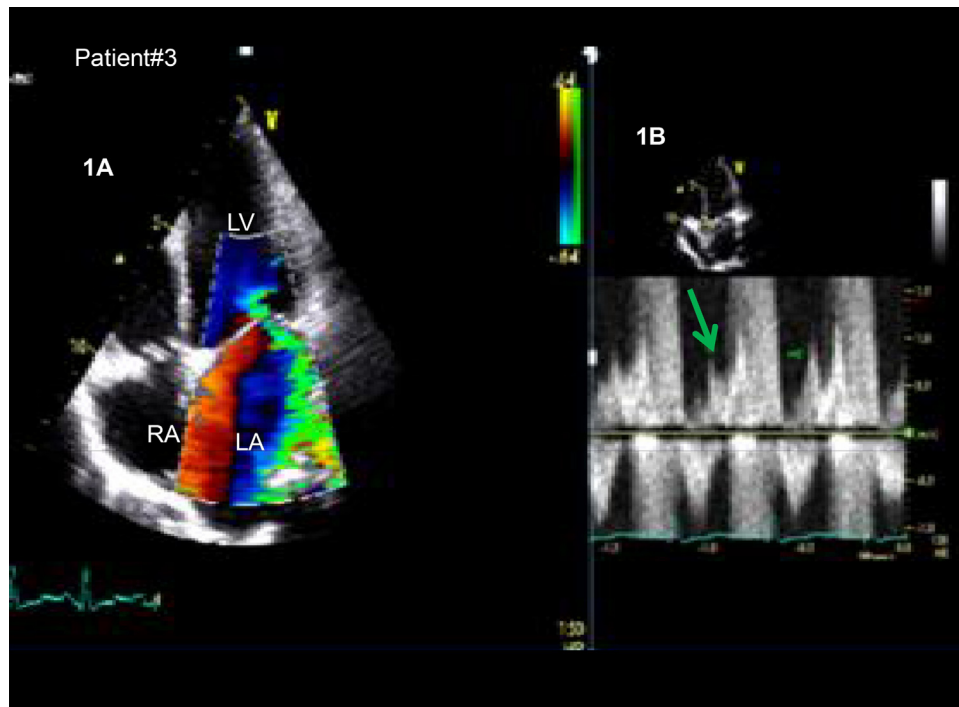


Fig. 6. 2D apical four-chamber view showing severe eccentric MR (1A) and SAR spectrum (green arrow, 1B).

outflow tract. This phenomenon was noted in three patients described in this study.

Aortic regurgitation (AR) is mostly a diastolic hemodynamic entity. Diastolic AR may encroach upon systole in case the LV does not generate adequate pressure in some beats. This hemodynamic phenomenon occurs during atrial fibrillation with fast ventricular rate and in presence of premature ventricular contractions if the aortic valve shows incomplete closure.^{1,2} Presence of systolic dysfunction potentiates its occurrence. None of our patients had atrial fibrillation or ectopic beats. It is also possible to observe this phenomenon in the failing LV in sinus rhythm wherein rate of development of pressure is very slow and the aortic systolic pressure exceeds the LV systolic pressure during prolonged isovolumic contraction.³ In presence of atrial fibrillation and premature ventricular contractions, aortic regurgitation can be pan-cyclic if the aortic valve fails to open for antegrade flow ejection during systole. In isolated LV systolic dysfunction associated with incompetent aortic valve, diastolic AR continues in early systole to a variable extent but is not observed in late diastole

Pressure gradient reversal in central aorta during late systole in acute or subacute severe MR is a complex process. It is easy to explain systolic pressure-flow reversal in aorta in premature ventricular contractions or atrial fibrillation with marked RR variability. In these conditions, Frank-Starling Law explains smaller stroke volume and developed LV systolic pressure in beats with shorter diastole. On the other hand, aortic systolic pressure is minimally affected by RR variability due to anti-oscillatory effect of the latter.⁹ Aortic systolic pressure is determined by aortic compliance, ventricular stroke volume and peripheral resistance.¹⁰ In severe MR and heart failure, aortic systolic pressure does not track LV systolic pressure as the former is controlled by baro-reflex activity and increased peripheral vascular resistance. This baro-reflex buffering of arterial systolic blood pressure may be responsible for transient pressure-flow reversal during later half of systole and

consequent systolic AR in patients with decompensated acute or subacute severe MR, as seen in our cases.

4. Limitations

The phenomenon of systolic AR in this study has been explained by Doppler echocardiographic examination. We did not conduct invasive pressure studies to substantiate our results. 4D MRI flow studies can also provide greater insight and differentiate systolic valvular versus supra-valvular AR. Retrospective nature of the study precluded such confirmation. Nevertheless, the study describes an interesting Doppler phenomenon of systolic flow –velocity reversal in aorta in presence of severe MR in rheumatic carditis.

Conflict of interest

None.

Financial support

None.

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