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Systolic Aortic Regurgitation in Rheumatic Carditis: Mechanistic Insight by Doppler Echocardiography

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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.

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 cardio-vascular 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' criteria [8] 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 dyspnoea 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,

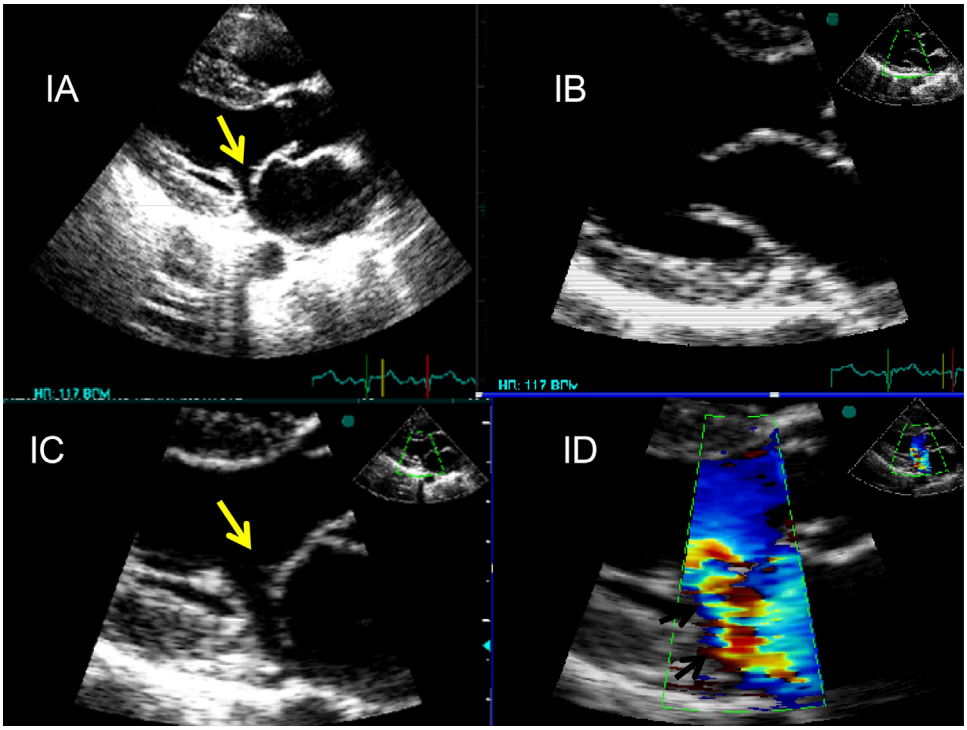

Fig. 1. 2D echocardiographic parasternal long axis views showing flail anterior mitral leaflet (yellow arrows) and sever eccentric mitral regurgitation by color Doppler (black arrows, ID).

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