

Dilated Cardiomyopathy And Valvular Dysfunction: A Pathway To Heart Failure With Reduce Ejection Fraction.

Dr. N. K. Pandey
(HOD MEDICINE)

Dr. Thakur Mani
(HOU 1 & Guide)

Dr. Shivkumar Chaurasia
(DNB Resident JR1)

Abstract:

Background:

Dilated cardiomyopathy is a frequent manifestation of advanced cardiac disease and may results from long-standing volume or pressure overload. Valvular heart disease becomes and remains an important and potentially reversible cause of left ventricular dilatation and systolic dysfunction leading to heart failure with reduced ejection fraction.

Case Presentation:

We report the case of middle-aged female patient who presented with progressive exertional dyspnea, orthopnea, PND, and Bilateral pedal edema. Clinical examination revealed the signs of congestive heart failure along with a pathological murmur. Chest radiography demonstrates marked cardiomegaly. The transthoracic echocardiogram revealed dilated left cardiac chambers with reduced ventricular systolic function consistent with HFrEF. Detailed echocardiography assessment identified significant valvular heart disease as an primary etiology of ventricular remodelling and systolic dysfunction. The patient was managed with guidelines-directed medical therapy for HFrEF along with targeted treatment for underlying valvular pathology resulting in clinical improvement.

Conclusion:

This case highlights valvular disease as an important cause of dilated cardiomegaly and HFrEF. Early recognition of valvular etiology through comprehensive clinical and echocardiographic evaluation is crucial as timely intervention and prevent irreversible myocardial damage and improves outcomes.

Keywords: Dilated cardiomyopathy; Valvular heart disease; heart failure with reduced ejection fraction; Echocardiography.

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I. Introduction:

Dilated cardiomyopathy is a common radiological and echocardiographic finding in patient with advanced heart disease and is often associated with poor clinical outcomes. Chronic valvular disease, particularly lesions resulting in volume overload, can lead to progressive ventricular dilatation, adverse remodeling, and eventually systolic dysfunction. Heart failure with reduced ejection fraction (HFrEF) represents the final outcomes pathway of such maladaptive changes if left untreated.

Despite advances in diagnostic imaging and medical therapy, valvular heart disease remains an under-recognized cause of dilated cardiomyopathy, especially in patients presented late with heart failure symptoms and signs. Identification of the underlying valvular pathology is essential, as timely intervention can halt or reverse ventricular remodeling. We report a case of dilated cardiomegaly secondary to valvular heart disease presenting as HFrEF, highlighting the diagnostic approach and clinical implications.

II. Case Presentation:

A 50-year old female patient came to CHD Medicine opd with progressively worsening of dyspnea on exertion (NYHA class III, Orthopnea, PND, and bilateral lower-limb swelling for several months. Patient also

complaints of occasionally palpitation with no diurnal variation. There was no history of chest pain, syncope, or fever. Past medical history was non contributory and there was no history of alcohol exposure or cardiotoxic drug exposure.

III. Clinical Examination:

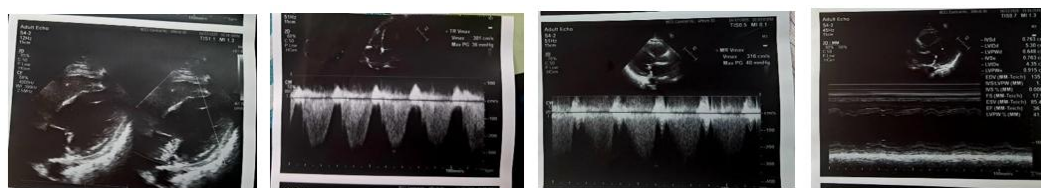
On Examination, the patient was tachycardic with pulse around 116bpm with fatigue and lower side blood pressure with signs of volume overload, including elevated jugular venous pressure and bilateral pitting pedal edema. Cardiac examination revealed a displaced point of maximal apical impulse (PMI), with Auscultatory findings suggestive of significant valvular regurgitations. JVP distended with hepatomegaly with bibasilar crepitations were noted on respiratory examination.

Diagnostic Assessment:

Routine laboratory investigations were within normal limits. Chest radiograph demonstrated marked cardiomegaly with increased cardiothoracic ratio with pulmonary venous congestion. Echocardiography showed features suggestive of chamber enlargement.

Transthoracic two-dimensional echocardiography revealed severe mitral regurgitation and severe tricuspid regurgitation. The left atrium and ventricle were markedly dilated. Left ventricular systolic function were severely impaired, with global hypokinesia and dyskinetic interventricular septal motion.

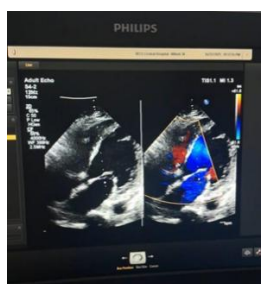
The estimated left ventricular ejection fraction was 35% consistent with heart failure with reduced ejection fraction. No intracardiac thrombus were visualized.



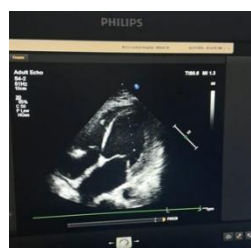
Chest radiograph (posteroanterior view) demonstrating marked cardiomegaly with increased cardiothoracic ratio and features suggestive of pulmonary venous congestion.

CT Scan of thorax shows evidence of cardiomegaly with LV configuration moderate bilateral pleural effusion and features suggestive of pulmonary edema.

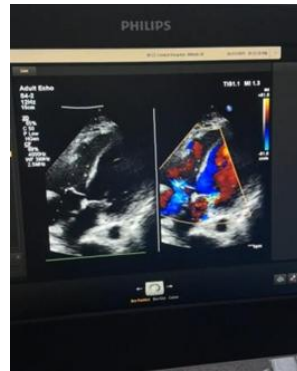
Two-dimensional transthoracic echocardiography, apical four-chamber view, showing markedly dilated left atrium and left ventricle with severe mitral regurgitation visualized on colour Doppler imaging.



Two-dimensional transthoracic echocardiography, parasternal long-axis view, demonstrating dilated left ventricle with reduced systolic function, global hypokinesia, and dyskinetic interventricular septal motion.



Apical four-chamber view with color Doppler showing severe tricuspid regurgitation with right atrial enlargement, consistent with advanced valvular heart disease



Diagnosis:

Based on clinical presentation and echocardiographic findings, a diagnosis of dilated cardiomegaly secondary to severe mitral and tricuspid regurgitation resulting in heart failure with reduced ejection fraction was established.

Therapeutic Intervention:

The patient was initiated on guideline-directed medical therapy for HFrEF, including loop diuretics, beta-blockers, angiotensin-converting enzyme inhibitors/angiotensin receptor–neprilysin inhibitors, and mineralocorticoid receptor antagonists, with careful titration. The patient was referred for cardiology evaluation for definitive management of the underlying valvular disease.

Follow-Up and Outcome:

Following initiation of therapy, the patient showed significant symptomatic improvement with reduction in dyspnea and peripheral edema. The patient remains under close follow-up for optimization of heart failure therapy and further assessment for potential surgical or percutaneous valvular intervention.

IV. Discussion:

Chronic valvular regurgitation imposes sustained volume overload on cardiac chambers, leading to eccentric hypertrophy, chamber dilatation, and progressive systolic dysfunction. Severe mitral regurgitation results in left atrial and left ventricular dilatation, while chronic tricuspid regurgitation contributes to right-sided volume overload and systemic congestion. When prolonged, these changes lead to adverse ventricular remodeling and reduced ejection fraction.

Echocardiography remains the cornerstone of diagnosis, allowing comprehensive assessment of chamber dimensions, ventricular function, and valvular severity. The presence of global hypokinesia and dyskinetic septal motion in this patient suggests advanced myocardial involvement. Early identification and timely correction of valvular lesions are crucial, as delayed intervention may result in irreversible myocardial damage and poor prognosis.

V. Conclusion:

Severe mitral and tricuspid regurgitation can lead to dilated cardiomegaly and HFrEF through chronic volume overload and adverse ventricular remodeling. This case underscores the importance of detailed echocardiographic evaluation in patients presenting with heart failure, as early recognition and appropriate management of valvular heart disease may significantly improve clinical outcomes.

Patient Consent:

Written informed consent was obtained from the patient for publication of this case report.

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