

Isolated tricuspid regurgitation secondary to annular dilatation: a rare case report

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Abstract

Isolated tricuspid regurgitation (TR) which is due to primary valvular pathology is a rare occurrence. Very few cases have been reported from India. Pathology may involve tricuspid valve leaflets, subvalvular apparatus or valve annulus. Tricuspid regurgitation is usually functional and associated with rheumatic heart disease or dilated cardiomyopathy. Here we are presenting a case of 55 years old man with primary tricuspid regurgitation secondary to dilatation of tricuspid annulus. Patient was started on diuretics and anticoagulation, 50% reduction in symptoms was noted after one week of treatment. In the given clinical context cause of this isolated tricuspid regurgitation appears to be secondary to annular dilatation. Patient was referred for myocardial biopsy and Annuloplasty. Patient's course seems to be rapidly progressive once he presents with the symptoms. But with the advances in percutaneous interventions there is a hope for these patients to be treated with significant clinical improvements.

Key words: Tricuspid valve leaflets, rheumatic heart disease, dilated cardiomyopathy.

Introduction

Tricuspid regurgitation (TR) can result from structural alterations of any of the components of the tricuspid valve apparatus which includes the leaflets, chordae tendineae, annulus, and papillary muscles or adjacent right ventricular (RV) muscle.

The pathophysiology of tricuspid regurgitation focuses on the structural incompetence of the valve. The incompetent nature of the valve can result from primary structural abnormalities of the leaflets and chordae or from secondary myocardial dysfunction and dilatation [1].

The lesion may be called as primary when it is caused by an intrinsic abnormality of the valve apparatus or as secondary when it is caused by right ventricular dilatation or dysfunction [2]. Incidence of tricuspid regurgitation is < 1% out of which incidence of primary isolated tricuspid regurgitation is very rare. The causes

of primary tricuspid regurgitation include rheumatic heart disease, infective endocarditis, Ebstein anomaly, prolapse, Carcinoid, papillary muscle dysfunction, trauma, connective tissue diseases or medications [5].

Case Presentation: A 55 years old man presented with history of class II dyspnoea since two years, oedema of both lower limbs since one year and facial puffiness since 6 months. No previous history of angina, chronic cough, pulmonary tuberculosis. No history of addictions.

Physical examination: Patients pulse was 82 per minute irregularly irregular radial artery; blood pressure was 128/76 mm of Hg. Jugular venous pressure was raised to 14 cm with prominence of CV complex. Oedema of both lower limbs is present which is pitting and extending up to the knee joint. Cardiovascular system examination revealed shift of apex laterally apex being in left fifth intercostal space 1 cm lateral to mid-clavicular line. Diastolic shock was absent. Right heart border was 1.5 cm lateral to right sternal border. Soft

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high pitched pan systolic murmur of grade III/VI was present in tricuspid area increasing on inspiration. Abdominal examination revealed hepatomegaly. Liver was soft non tender, pulsatile with span of 16 cm.

Investigations: Haemoglobin was 12.5mg/dl with total leukocyte count of 4700, platelets were adequate, random sugar was 90 mg /dl, Creatinine 0.6; AST was 41, ALT 36. Arterial blood gas showed PH 7.43, po2 88.9, pco2 39, so2 95, HCO3 25.5. Electrocardiogram showed atrial fibrillation with ventricular rate of 78. X-ray chest showed cardiomegaly.

2-dimensional echo revealed normal left ventricular function. Tricuspid regurgitation of grade III. TV annulus 55 mm. Valve leaflets was normal. Right atrium was 81x 75 mm. Right atrium and right ventricle was dilated. No Epstein anomaly, no vegetation, interatrial and interventricular septum was intact, left ventricular ejection fraction was 60%.

Urinary 5-hydroxyindolacetic acid levels were done to rule out carcinoid, it was marginally elevated 11.7 (Normal range: 2-6) although there were no previous symptoms of flushing, diarrhoea. Computerized tomography scan of chest and abdomen with contrast was performed, it was normal.

Management: During hospital stay patient was started on diuretics and anticoagulation, 50% reduction in symptoms was noted after one week of treatment. In the given clinical context cause of this isolated tricuspid regurgitation appears to be secondary to annular dilatation. Patient was then referred for myocardial biopsy and planned for annuloplasty.

Discussion

The natural history of isolated, severe organic TR has been patients initially without symptoms. Most of the cases reported yet have experienced the end points of heart failure, new atrial fibrillation, Tricuspid valve surgery or death within 5 years of diagnosis [3].

Severe TR is either functional or primarily organic. It does not have a benign prognosis over the long term as suggested in the earlier literature. Despite the fact that tricuspid regurgitation can result in significant symptoms, it remains undertreated. Patients are rarely referred for isolated surgical repair, and most repairs are done in the context of other planned cardiac surgery. Even in case of functional TR to avoid the development

of moderate to severe tricuspid regurgitation later in the course of disease aggressive management of TR needs to be done when operating on mitral valve [4].

2014 AHA/ACC Guideline for the Management of Patients With Valvular Heart Disease now recommend that Tricuspid valve repair be performed for severe TR in patients requiring left sided valve surgery as a class I indication. Symptomatic, severe primary TR (stage D) is a class IIa indication for Tricuspid valve repair or Tricuspid valve replacement (TVR) [5]. Tricuspid annuloplasty repair should also be considered for less than severe TR in patients undergoing mitral valve surgery in the presence of pulmonary hypertension or tricuspid annular dilation. Our patient had grade III TR due to annular dilatation. Although this recommendation carries a class IIb indication, it should be noted that tricuspid annular dilation is a progressive process commonly associated with the development of severe TR on long-term follow up. Studies show that Ring annuloplasty is a key aspect of current surgical techniques and its durability is superior to suture annuloplasty techniques, although this does not translate into a survival benefit [6]. Campelo-Parada F et al performed transcatheter Forma Repair System (Edwards Lifesciences, Irvine, California) on severe TR patients who were high risk for TVR and showed significant improvement in symptoms [7]. If left untreated Patients with moderate-to-severe TR are much more likely to have ablation failure with atrial arrhythmias [8].

Isolated primary TR is rare and progressively fatal Valvular disease. But with the advances in percutaneous interventions there is a hope for these patients to be treated with surgery once secondary causes are ruled out with significant clinical improvement.

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