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Severe tricuspid regurgitation mimicking constrictive pericarditis

Authors' Contribution:
Study Design A
Data Collection B
Statistical Analysis C
Data Interpretation D
Manuscript Preparation E
Literature Search F
Funds Collection G

ABCDEF 1 Ebru Ozpelit
ABDE 1 Bahri Akdeniz
DEF 2 Mehmet Emre Ozpelit
ABD 1 Özhan Göldeli

1 Department of Cardiology, Dokuz Eylul University, School of Medicine, Izmir, Turkey
2 Department of Cardiology, Izmir University, School of Medicine, Medicalpark Hospital, Izmir, Turkey

Corresponding Author: Ebru Ozpelit, e-mail: ebru.ozpelit@gmail.com
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Patient: Female, 62
Final Diagnosis: Tricuspid regurgitation
Symptoms: Dyspnea exertional • fatigue • leg edema
Medication: —
Clinical Procedure: —
Specialty: Cardiology

Objective: Challenging differential diagnosis

Background: Tricuspid regurgitation (TR) can mimic some hemodynamic findings of constrictive pericarditis (CP), due to the restraining effect of the enlarged right heart on intact pericardium and on the left ventricle. In this article, we report a case of severe tricuspid regurgitation in which hemodynamic findings were consistent with CP.

Case Report: A 62-year-old Caucasian woman presented with right heart failure symptoms. Echocardiography showed enlarged right heart chambers and severe tricuspid regurgitation. Right heart catheterization surprisingly demonstrated a constrictive physiology. Diastolic pressures of both ventricles were elevated and equalized, with a prominent deep and plateau pattern. The patient was re-evaluated with a further focus on constrictive pericarditis. However, echocardiography, thorax CT, and cardiac MRI did not demonstrate any pathological finding related to pericardium. The remaining explanation was that the severe TR itself and secondary right heart enlargement caused the restraining effect on the intact pericardium and on the left ventricle. The pericardium was normal and tricuspid annulus was severely dilated on surgical inspection. The tricuspid valve was replaced with a bioprosthetic valve. The patient did well just after the surgery, with a rapid decrease in cardiac pressures; however, she died due to respiratory failure on the 15th postoperative day.

Conclusions: This was a case with right heart failure symptoms in which invasive hemodynamic findings were consistent with constrictive pericarditis and the noninvasive imaging modalities were not. This case illustrates that severe TR can mimic some hemodynamic findings of constrictive pericarditis, due to restraining effect of the enlarged right heart on intact pericardium and on the left ventricle. Lack of significant respiratory changes in hemodynamic parameters that can safely be demonstrated by echocardiography and cardiac MRI suggest a normal pericardium.

MeSH Keywords: Pericarditis, Constrictive • Cardiac Catheterization • Tricuspid Valve Insufficiency

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Background

Significant tricuspid regurgitation (TR) is an independent predictor of reduced event-free and overall survival [1]. Patients may remain asymptomatic for a long time; however, signs of reduced cardiac output and right ventricular (RV) failure usually become apparent after a while. Moreover, the clinical consequences are not always limited to the right heart – occasional cases in the literature prove that severe TR can also influence the left ventricle (LV) and even the entire heart. In this report, a patient with right heart failure is presented, which had a constrictive physiology due to severe TR.

Case Report

A 62-year-old Caucasian woman presented with profound leg edema, increased abdominal girth, exertional dyspnea, and fatigue. Her symptoms were prominent for the past 20 days after an upper respiratory tract infection. Her past medical history included open heart surgery for pulmonary stenosis (PS) when she was 19 years old. She was diagnosed with RV failure 15 years ago. Physical examination revealed signs of severe RV failure and a 3/6 systolic murmur in the tricuspid region. Transthoracic echocardiography (TTE) showed enlarged right heart chambers and severe TR with a 45 mmHg estimated systolic pulmonary artery pressure (PAP) (Figure 1A, 1B). The tricuspid leaflets had no organic pathology, so the severe TR was thought to be secondary to annular dilatation. Despite the history of PS, the appearance of the pulmonary valve was normal. Right heart catheterization (RHC) was performed to identify the etiology of functional TR and pulmonary hypertension. RHC findings surprisingly demonstrated a constrictive physiology. Pressures of right atrium (RA) and superior and inferior vena cava (SVC) curves were almost totally identical, with prominent V waves, and mean pressures of 20 mm Hg, which was also equal to the RV end diastolic pressure (RVEDP), LVEDP, and pulmonary capillary wedge pressure (PCWP). Diastolic pressures of both ventricles were elevated and equalized, with a prominent deep and plateau pattern (Figure 2A–2C). Although respiratory ventricular discordance was negative, all of the other catheter findings were consistent with constrictive pericarditis (Figure 2D). We re-evaluated the patient with a further focus on CP. However, echocardiography, thorax CT, and cardiac MRI did not demonstrate any pathological finding related to the pericardium. Paradoxical septal movement was noted in both echo and cardiac MRI, but respiratory septal bounce was absent. There was also no respiratory variation in mitral and tricuspid inflow velocities (Figure 3). We therefore thought the pericardium was not responsible for these hemodynamic findings. The remaining explanation was the severe TR itself and secondary right heart enlargement causing a restraining effect on the intact pericardium and on the left ventricle during diastole. Tricuspid valve surgery was planned. On surgical inspection, the pericardium and tricuspid valve leaflets were completely normal, but the tricuspid annulus was severely dilated. There was no pericardial thickening or adhesion. Therefore, the tricuspid valve was replaced with a bioprosthetic valve. The patient did well just after the surgery, with a rapid decrease in central venous pressure and PCWP. Supportive medical therapy, including intravenous antibiotics, diuretics, and heparin was given postoperatively. However, her respiratory functions progressively deteriorated postoperatively. She developed hospital-acquired pneumonia due to prolonged invasive ventilation and on the 15th day after the surgery she died due to respiratory failure.

Discussion

This case illustrates how severe TR can cause hemodynamic findings consistent with CP. The equalization of diastolic pressures of all 4 chambers, deep and plateau diastolic filling

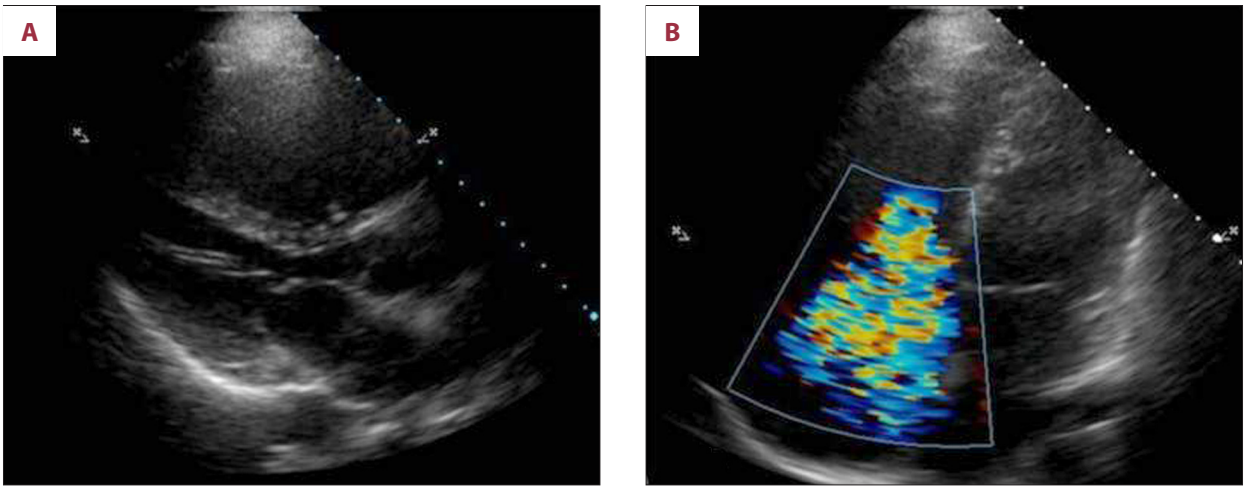

Figure 1. Echocardiography showing enlargement of right heart and severe tricuspid regurgitation.

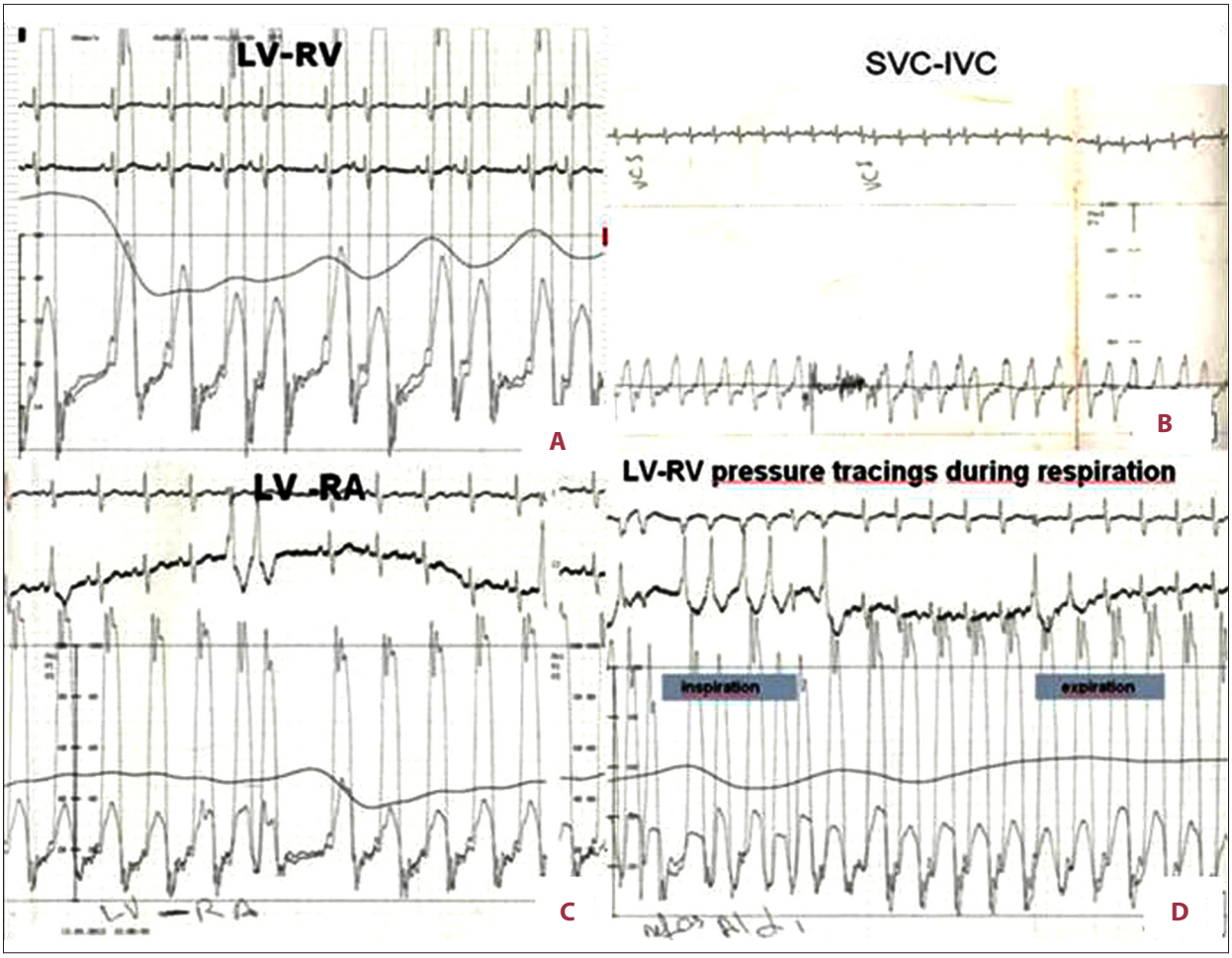

Figure 2. Pressure tracings of cardiac catheterization.

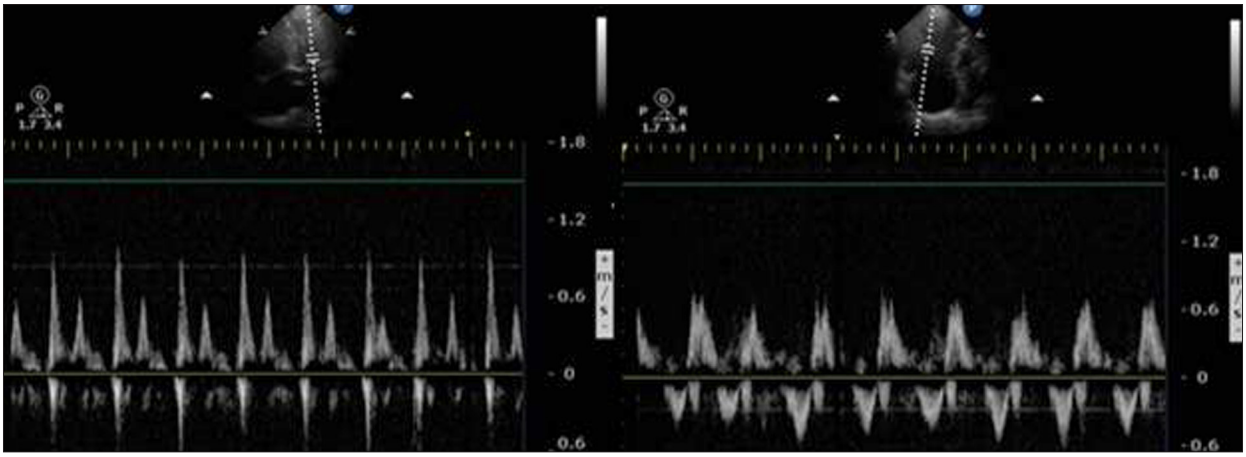

Figure 3. Pulsed wave Doppler analysis of mitral and tricuspid inflow.

pattern, and slightly increased PAP and RVEDP exceeding 1/3 of RV systolic pressure at first suggested CP. Understanding the underlying physiology and differentiating these 2 entities are important to appropriate management. This case also demonstrates how an ignored, severe TR can cause a very disabling clinical picture over the years, resulting in surgical mortality, probably due to a delayed operation.

Severe TR mimicking constrictive pericarditis is a very rare clinical condition due to the restraining effect of the enlarged

right heart on the pericardium and left ventricle. This phenomenon was first described by Cha et al., who reported the hemodynamic findings of 59 patients with severe TR [2]. In their series, 2 patients were found to have constrictive-like physiology, with equalization of diastolic pressures of the heart chambers, elevated PCWP, and RV diastolic pressure greater than 1/3 of the systolic pressure. Later, Studley et al. reported a case with severe TR showing signs of CP in invasive hemodynamic study, despite a totally normal pericardium in surgical inspection [3]. These data raised the question of how to discriminate between criteria for CP itself and severe TR with constrictive-like physiology. Jaber et al. searched for these criteria in their study comparing the hemodynamic findings of 14 severe TR patients with those of 14 patients with surgically proven CP [4]. They found that during deep inspiration, there was a marked increase in RVEDP and in transseptal pressure gradient (RVEDP became substantially higher than LVEDP) in the TR group. However, RV and LV diastolic pressure tracings, which come together during deep inspiration, were found to be more consistent with CP. Although not mentioned, the main limitation of that study is that the patients in the severe TR group were not selected according to the presence of constrictive-like physiology. Considering that this physiology was established only in 2 out of 59 severe TR cases in Cha et al's series, it is questionable whether the study population was appropriate for establishing such discriminating criteria. Also, considering our case and Studley's case, in which there were no significant respiratory changes in hemodynamic parameters, catheterization, or echocardiography, it is more convenient to use noninvasive imaging findings for discrimination. In these 2 cases, although the invasive hemodynamic were demonstrative of a constrictive-like physiology, noninvasive imaging modalities were not. In our opinion, in such patients it is safe to rely on the findings of TTE, thorax CT, and, especially, the cardiac MRI.

Conclusions

We presented a case with right heart failure symptoms in which invasive hemodynamic findings were consistent with CP and the noninvasive imaging modalities were not. This case illustrates that severe TR can mimic some hemodynamic findings of CP, due to the restraining effect of the enlarged right heart on intact pericardium and the left ventricle. Lack of significant respiratory changes in hemodynamic parameters that can safely be demonstrated in TTE and cardiac MRI must suggest a normal pericardium. Ignorance of the TR for a period of years and delayed intervention can cause such findings, exacerbating the right heart failure. Moreover, this causes a significantly poorer prognosis and higher operative mortality, as observed in our reported case.

Conflict of interest

The authors declare that they have no competing interests.

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