

When the Right Heart Leads to the Pelvis: Advanced Tricuspid Regurgitation Revealing an Ovarian Mass

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Abstract

Case Report

Background: Severe tricuspid regurgitation (TR) is usually functional, secondary to left-sided heart disease, pulmonary hypertension, or atrial fibrillation. Organic TR due to carcinoid heart disease is rare, and ovarian carcinoid tumors represent an uncommon primary source. **Case summary:** An 84-year-old woman with no prior cardiovascular history was admitted for decompensated right heart failure. She had a 2-year history of severe TR, now presenting with NYHA class III dyspnea, paroxysmal nocturnal dyspnea, and bilateral lower limb edema. ECG revealed atrial fibrillation with right axis deviation and incomplete right bundle branch block. Echocardiography showed a dilated right ventricle with preserved function, markedly enlarged right atrium, and severe TR due to leaflet maladaptation and annular dilation (66 mm). CT imaging identified a right latero-uterine cystic mass (53 × 56 mm) suspicious for ovarian neoplasm, along with ascites and pericardial effusion. Laboratory testing revealed elevated tumor markers, including carcinoembryonic antigen (CEA) and carbohydrate antigen 19-9 (CA 19-9). Given her advanced TR and high TRI-SCORE (≥6), surgery was deemed high risk with limited expected benefit. She was managed medically with high-dose furosemide (500 mg daily), spironolactone (75 mg daily), and hydrochlorothiazide (25 mg daily), alongside oncologic evaluation for suspected carcinoid tumor. **Discussion:** Ovarian carcinoid tumors can cause isolated right-sided valvular disease by releasing serotonin directly into the systemic circulation, bypassing hepatic metabolism. In advanced TR with high TRI-SCORE, registry data show poor survival regardless of surgical intervention, supporting a medical management strategy. **Conclusion:** Isolated severe TR without left-sided or pulmonary involvement should prompt investigation for systemic or neoplastic causes. Ovarian carcinoid tumor, though rare, should be considered, particularly when surgical correction of TR is not feasible. “When the Right Heart Leads to the Pelvis: Advanced Tricuspid Regurgitation Revealing an Ovarian Mass”.

Keywords: Severe tricuspid regurgitation, Ovarian carcinoid tumor, Right heart failure, Organic TR, Serotonin, NYHA class III dyspnea.

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INTRODUCTION

Severe tricuspid regurgitation (TR) is no longer considered a benign finding; it is now recognized as an independent predictor of poor outcomes, even in the absence of left-sided disease [1]. Most cases are functional, secondary to atrial fibrillation, pulmonary hypertension, or left-sided valve pathology. Organic TR is less common and may result from systemic or neoplastic causes.

Carcinoid heart disease is a classic example, typically affecting right-sided valves through serotonin-induced fibrotic retraction. While gastrointestinal neuroendocrine tumors are the usual source, ovarian carcinoid tumors are rare but important, as pelvic venous

drainage can bypass hepatic metabolism and lead to isolated right-sided involvement without hepatic metastases [2,3].

We report the case of an elderly woman with advanced severe TR and right heart failure, in whom imaging revealed a suspicious ovarian mass. This case illustrates the link between isolated TR and ovarian neoplasia, and the challenges of management in advanced disease.

CASE PRESENTATION

An 84-year-old woman with no known cardiovascular history was admitted for progressive signs of right heart failure. She had been followed for two

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years for severe tricuspid regurgitation (TR), initially considered functional. Over recent weeks, she developed worsening dyspnea (NYHA class III), paroxysmal nocturnal dyspnea, and prominent lower limb edema extending to the mid-thighs (Figure 1). Clinical

examination revealed jugular venous distention with a positive hepatojugular reflux and a holosystolic murmur best heard at the lower left sternal border, accentuated with inspiration.



Figure 1 : Clinical image showing bilateral lower limb edema extending to mid-thigh level, consistent with congestive right-sided heart failure

Electrocardiogram showed atrial fibrillation, a right axis deviation, and incomplete right bundle branch block (Figure 2).

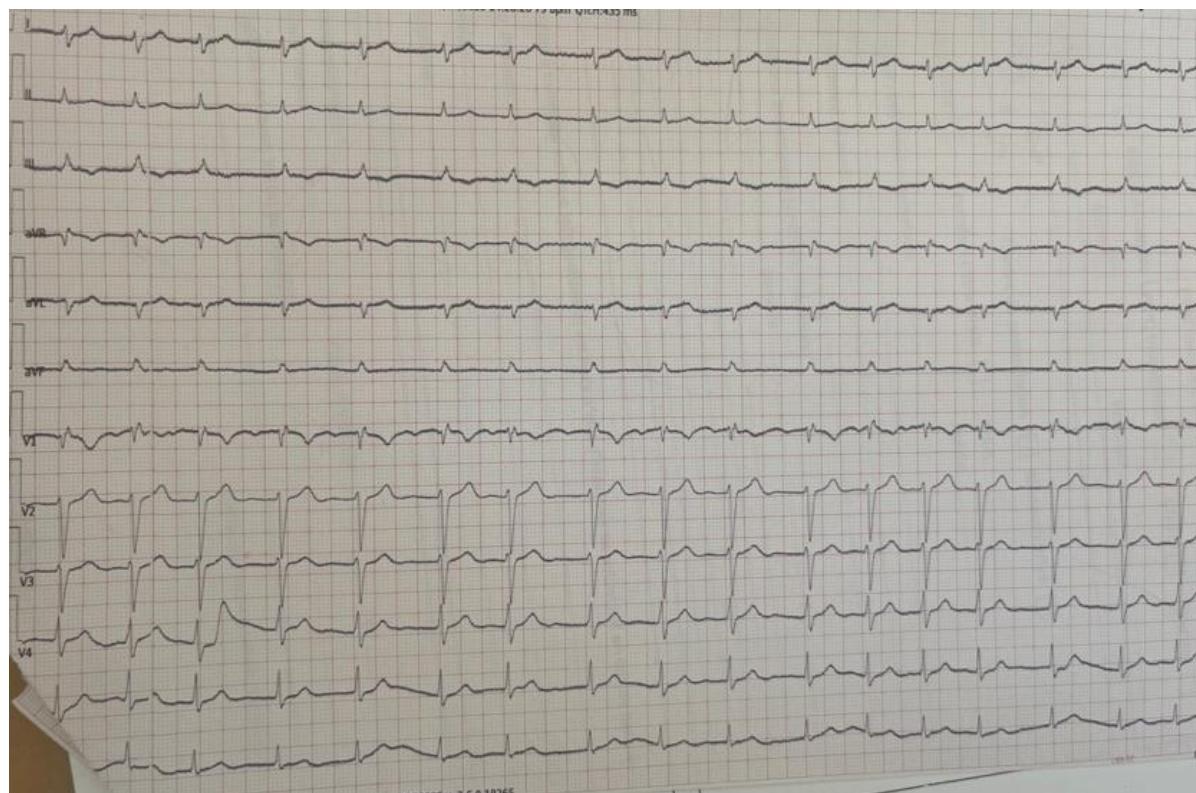


Figure 2 : 12-lead electrocardiogram showing atrial fibrillation with right axis deviation and incomplete right bundle branch block

Transthoracic echocardiography (TTE) demonstrated a dilated right ventricle with preserved systolic function (RV fractional shortening 55%, strain – 10%) and paradoxical septal motion. The right atrium was markedly enlarged (area: 84 cm²), and the inferior vena cava was severely dilated. The tricuspid valve appeared anatomically distorted, with poor leaflet

copartition and a massively dilated annulus (66 mm). Color Doppler confirmed severe tricuspid regurgitation with a triangular-shaped dense jet and no PISA. Left ventricular size and function were normal (EF 55–60%), with no significant left-sided valvular disease apart from mild functional mitral regurgitation. A minimal pericardial effusion was also noted (Figure 3-4).

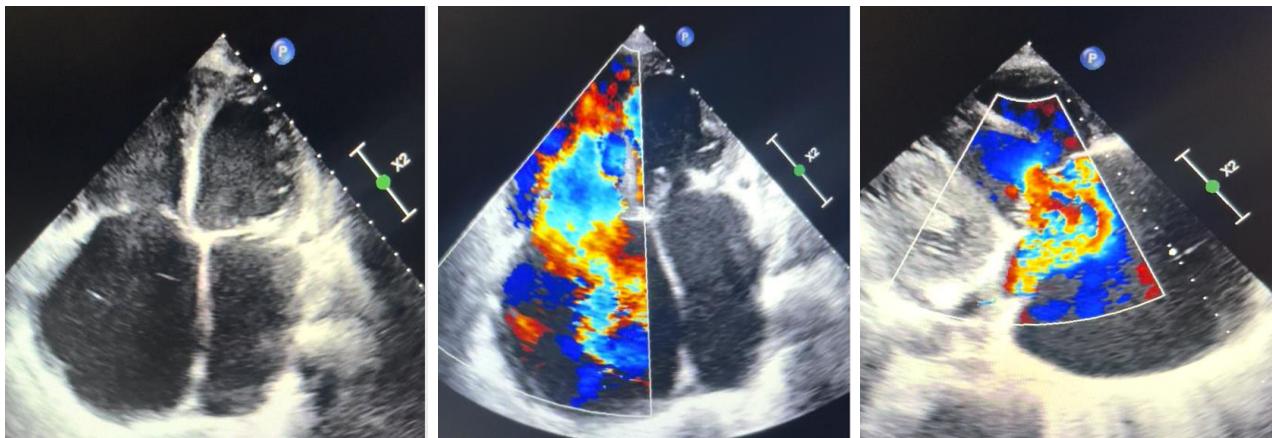


Figure 3 : Transthoracic echocardiography (apical four and two-chamber view) demonstrating a dilated right ventricle and severely enlarged right atrium with a severe tricuspid regurgitation

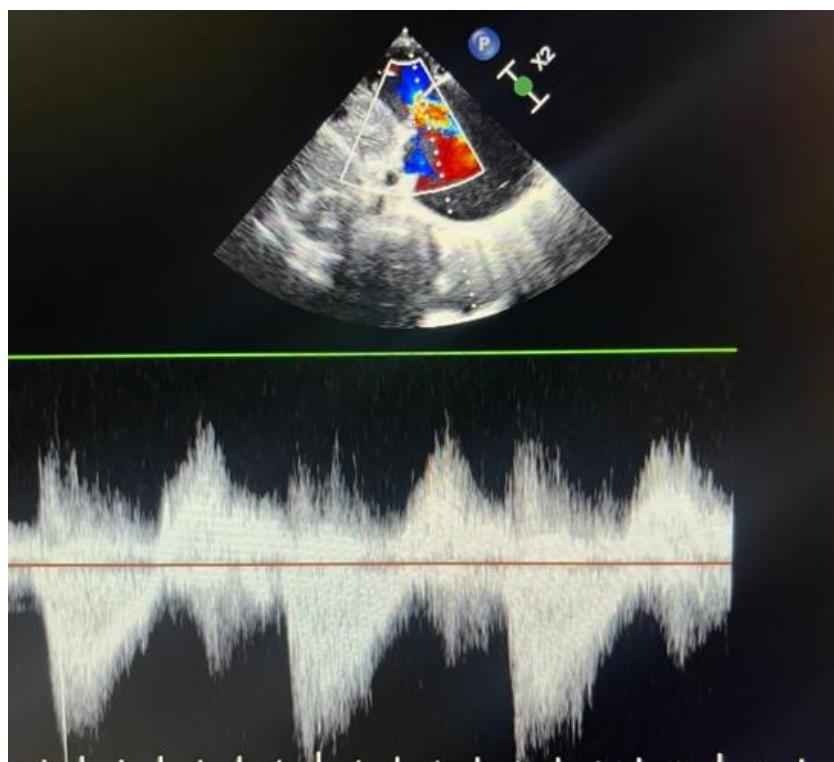


Figure 4 : Continuous wave Doppler profile of severe tricuspid regurgitation showing the characteristic dagger shaped spectrum

Given the absence of pulmonary hypertension or left-sided overload, an extended etiologic workup was undertaken. A contrast-enhanced thoraco-abdominopelvic CT scan revealed a well-circumscribed right latero-uterine cystic mass measuring 53 × 56 mm,

suggestive of an ovarian neoplasm, along with moderate ascites, cardiomegaly, and a 16 mm pericardial effusion (Figure 5). No hepatic lesions or metastases were identified.

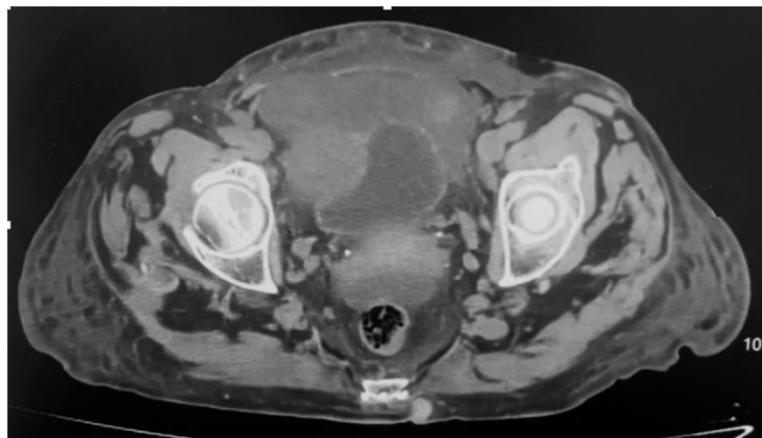


Figure 5 : Axial pelvic CT showing a well-defined right latero-uterine cystic mass, suspicious for an ovarian neoplasm

Laboratory tests showed elevated tumor markers, including carcinoembryonic antigen (CEA) and carbohydrate antigen 19-9 (CA 19-9). Based on clinical, imaging, echocardiographic, and laboratory findings, a paraneoplastic carcinoid syndrome of ovarian origin was suspected. Given the advanced stage of TR and a high TRI-SCORE (≥ 6), surgical intervention was not considered viable.

A medical management strategy was adopted, including aggressive diuretic therapy with furosemide 500 mg once daily, spironolactone 75 mg once daily, and hydrochlorothiazide 25 mg once daily, alongside dietary sodium restriction. A multidisciplinary team involving cardiology and gynecologic oncology was engaged for ongoing evaluation.

DISCUSSION

Severe tricuspid regurgitation (TR) is most frequently functional, resulting from right atrial and annular dilation due to atrial fibrillation, pulmonary hypertension, or left-sided valve disease. Organic TR is less common and typically arises from infective endocarditis, rheumatic disease, congenital abnormalities, or systemic disorders such as carcinoid heart disease.[4]

Carcinoid heart disease is caused by vasoactive substances—most notably serotonin—released by neuroendocrine tumors. These mediators stimulate fibrous tissue deposition on the endocardium, leading to leaflet thickening, retraction, and malcoaptation. The disease predominantly affects right-sided valves because serotonin is inactivated in the lungs. In gastrointestinal carcinoid tumors, clinically significant cardiac involvement generally requires hepatic metastases to bypass first-pass metabolism. In contrast, ovarian carcinoid tumors have a unique venous drainage pathway into the systemic circulation, allowing bioactive substances to reach the right heart directly without hepatic filtration, even in the absence of metastases. This explains why isolated right-sided valvular involvement may be the initial manifestation in such cases.[5,6]

Several reports have described ovarian tumor-associated carcinoid heart disease presenting as severe TR. Shin *et al.*, reported a patient with isolated severe TR as the first clue to an ovarian carcinoid tumor, confirmed histologically after surgical excision [3]. Engelsman *et al.*, described TR as the presenting symptom of a metastatic carcinoid tumor, highlighting that valvular involvement can precede other systemic manifestations [2]. Our patient's presentation—severe TR with a morphologically abnormal tricuspid valve, massive annular dilation, and a suspicious ovarian mass—is consistent with this rare entity.

From a management perspective, timely surgical or percutaneous intervention is crucial before irreversible right ventricular remodeling and severe annular dilation occur. In our patient, the combination of marked annular dilation (66 mm), advanced right atrial and ventricular enlargement, and a high TRI-SCORE (≥ 6) placed her in a poor-prognosis category. The TRIGISTRY multicenter registry demonstrated that, in such high-risk patients, 10-year survival remains dismal—28% after repair, 24% after replacement, and 17% with conservative therapy—without a statistically significant survival advantage for surgery over medical management [7]. Consequently, our patient was managed medically with aggressive diuretic therapy to control congestion, while addressing the underlying neoplastic process through oncologic evaluation.

In recent years, transcatheter tricuspid valve interventions (TTVI) have emerged as a therapeutic option for patients with severe TR deemed inoperable or at high surgical risk. Devices such as the TriClip and Pascal systems (edge-to-edge repair) and various transcatheter annuloplasty devices have demonstrated significant reductions in TR severity, symptomatic improvement, and better quality of life in selected patients.[8] Trials such as TRILUMINATE and early registry data have reported procedural success rates above 85% with sustained benefit at 1–2 years,

especially in functional TR with less advanced annular and right ventricular remodeling.[9,10]

However, these therapies are most effective when intervention occurs before extreme annular dilation, advanced right atrial/ventricular enlargement, or irreversible leaflet tethering.[11] In our patient, the annular diameter, massive right atrial enlargement, and leaflet malcoaptation reflect an end-stage anatomy, with limited likelihood of durable leaflet approximation or annular reduction via percutaneous means. Moreover, the suspected active neoplastic process further limits the potential benefit of a device-based approach.

Thus, while TTVI represents a valuable option for many high-risk TR patients, its benefit in very advanced carcinoid-related TR remains uncertain, and careful anatomical and clinical selection remains essential.

This case emphasizes two key lessons:

1. Isolated severe TR in the absence of left-sided or pulmonary disease warrants investigation for rare systemic and neoplastic causes, including ovarian carcinoid tumors.
2. In advanced high-risk TR, particularly with a TRI-SCORE ≥ 6 , the role of surgery is limited, and optimal medical therapy becomes the mainstay, alongside multidisciplinary care.

CONCLUSION

This case illustrates a rare but important association between isolated severe tricuspid regurgitation (TR) and a probable ovarian carcinoid tumor. In the absence of left-sided or pulmonary hypertension, TR of this severity should prompt an etiologic search for systemic or neoplastic causes. Ovarian carcinoid tumors can lead to isolated right-sided valvular disease through systemic release of serotonin, bypassing hepatic metabolism. In advanced high-risk TR (TRI-SCORE ≥ 6), surgical outcomes remain poor, and optimal management often relies on aggressive medical therapy and multidisciplinary coordination. Early recognition of this association is crucial to improve patient outcomes.

Ethical Statement

Written informed consent was obtained from the patient for publication of this case and accompanying images, in accordance with the journal's patient consent policy. The study was conducted in compliance with institutional and ethical standards and in accordance with the Declaration of Helsinki.

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Disclosure

The authors have nothing to report.

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Conflict of Interest

The authors declare that they have no conflicts of interest related to this publication.

Author Contributions:

S.T. and L.E. contributed to conceptualization, data collection, echocardiographic analysis, and manuscript writing.
S.D. and C.M. participated in investigation and methodology.
N.F. supervised and validated the manuscript.
M.C. provided critical revision and final approval.

All authors approved the final version and are accountable for all aspects of the work.

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