Case Report ISSN 2833-034X

Women's Health Care and Issues

Massive Tricuspid Regurgitation Revealing a Suspicious Ovarian Tumor (ORADS 5): A Typical Case of Carcinoid Cardiopathy

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Received: 08 Apr 20256; **Accepted:** 10 May 2026; **Published:** 20 May 2026

Citation: Khaoula LAKHDAR, Naoufal ELHARBILI, Mouna ANIBRI, et al. Massive Tricuspid Regurgitation Revealing a Suspicious Ovarian Tumor (ORADS 5): A Typical Case of Carcinoid Cardiopathy. Womens Health Care Issues. 2026; 5(1): 1-5.

ABSTRACT

Ovarian carcinoid syndrome is a rare entity in which vasoactive substances (serotonin) reach the right heart without hepatic inactivation. We report the case of a 70-year-old woman referred for evaluation of right heart failure. Echocardiography revealed massive tricuspid regurgitation (TR) due to bivalvular restriction, pathognomonic of carcinoid involvement. Further investigations revealed a left ovarian mass measuring 85 x 55 mm, classified as ORADS 5. This case highlights the importance of a multidisciplinary approach involving cardiologists and gynecological oncologists.

Keywords

Carcinoid heart disease, Carcinoid syndrome, Neuroendocrine tumors, Tricuspid valve disease, Serotonin.

Introduction

Carcinoid cardiomyopathy occurs in approximately 50% of patients with carcinoid syndrome. While the origin is classically digestive with liver metastases, primary ovarian involvement allows for direct passage of chemical mediators into the systemic circulation. Cardiac involvement is characterized by the deposition of fibrous plaques on the endocardium, leading to severe valvular dysfunction, primarily of the tricuspid valve.

Case Presentation

We report the case of a 70-year-old female patient with good performance status (OMS 0). She had no significant personal or familial medical history, with no known family history of malignancy. Regarding cardiovascular risk factors, the patient had none. Her gynecological history was notable for menarche at age 14, gravida 3 para 3, combined oral contraceptive use for over 10 years, menopause 15 years prior, and no hormone replacement therapy.

The patient presented with progressively worsening dyspnea (NYHAII), accompanied by lower back pain refractory to symptomatic treatment. Physical examination revealed arterial hypertension (blood pressure 170/80 mmHg). Cardiac auscultation revealed no pathological murmurs or additional heart sounds. Electrocardiography demonstrated sinus rhythm at 85 beats per minute, with normal PR interval and electrical axis, absence of ventricular hypertrophy, and negative T waves in leads V1 and V2 (Figure 1).

Transthoracic echocardiography revealed massive tricuspid regurgitation secondary to bivalvular leaflet restriction and diastasis, highly suggestive of carcinoid valvular disease. This was complicated by right chamber dilation with a dilated right ventricle of preserved systolic function and difficult-to-quantify pulmonary hypertension. The left atrium and aortic root were normal in size. The left ventricle was non-dilated without hypertrophy, with preserved systolic function and no segmental wall motion abnormalities apart from paradoxical septal motion secondary to right ventricular volume overload (Figure 2).

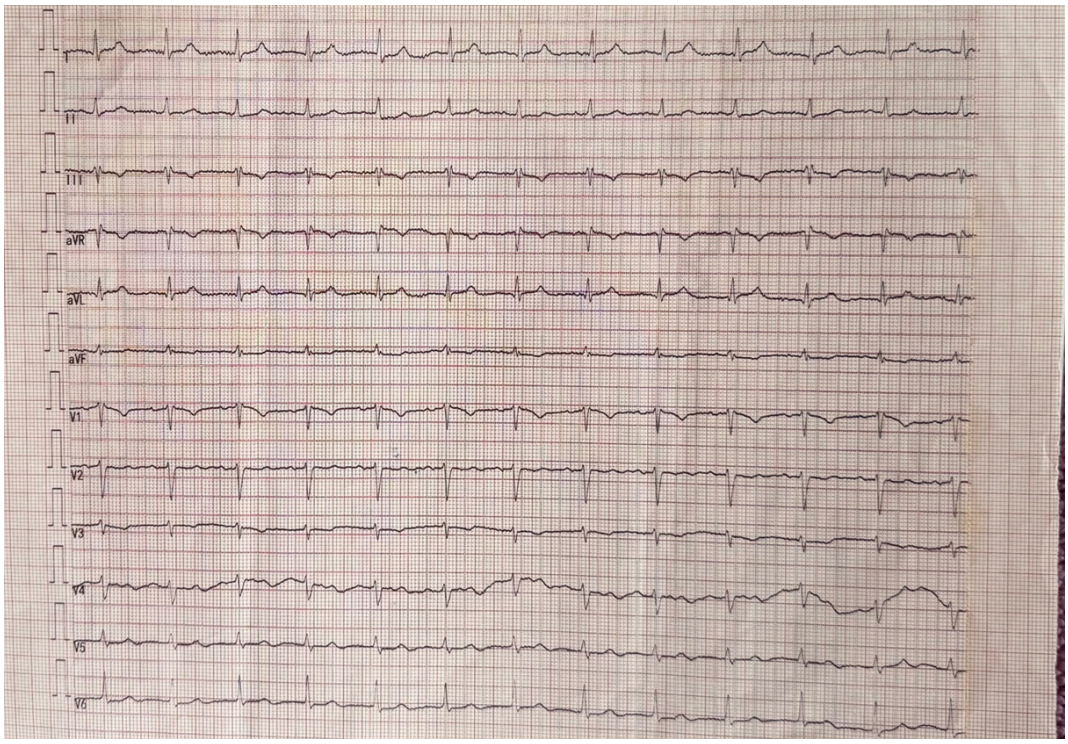

Figure 1: ECG showing a regular sinus rhythm with signs of right ventricular overload.

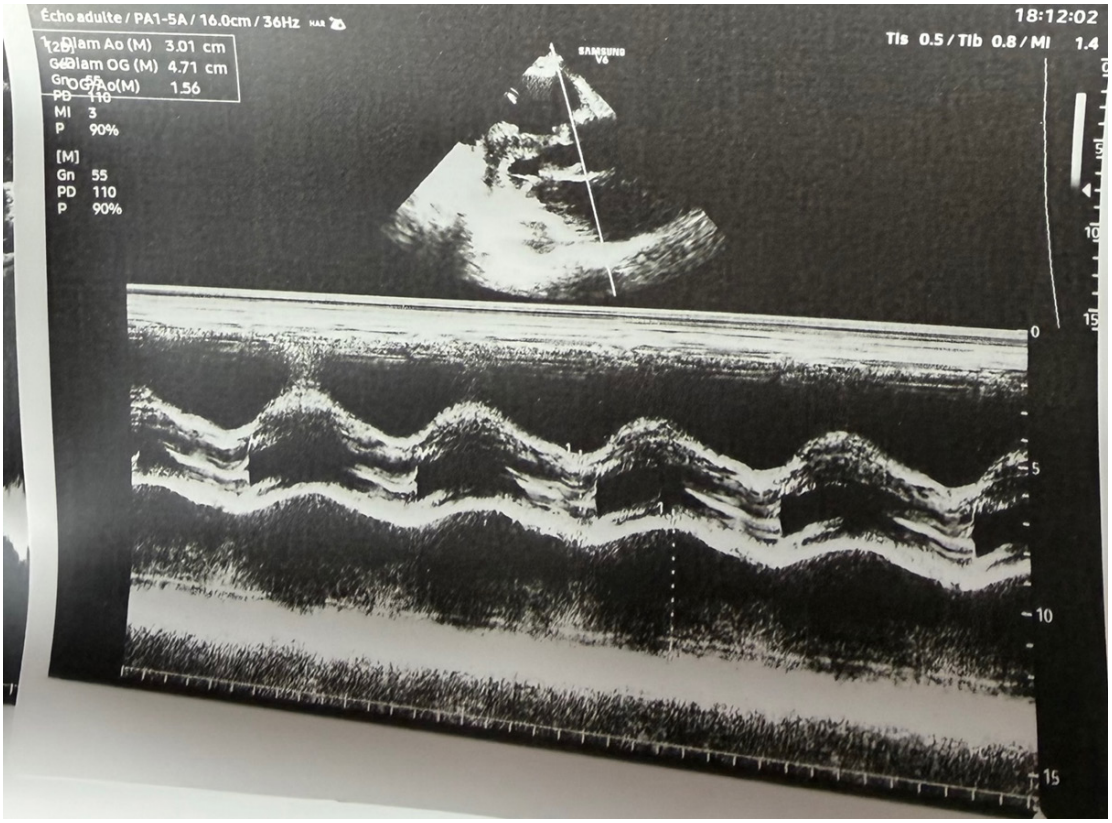

Figure 2: Transthoracic echocardiogram showing preserved left ventricular dimensions and preserved systolic function.

In addition, there was marked dilation of the right-sided chambers, including a severely dilated right ventricle and right atrium, with flattening of the interventricular septum, consistent with right ventricular volume overload (Figure 3). The echocardiographic findings also showed massive tricuspid regurgitation secondary to leaflet restriction and diastasis, highly suggestive of carcinoid valvular disease.

Gynecological examination revealed, on vaginal palpation, a fixed, painless left lateral uterine mass without parametrial invasion. The remainder of the pelvic examination was unremarkable.

Pelvic ultrasonography demonstrated a 85× 55 mm heterogeneous left lateral uterine tissue mass with irregular borders and marked Doppler vascularity, associated with mild ascites.

Pelvic magnetic resonance imaging confirmed a heterogeneous left lateral uterine/adnexal mass (O-RADS 5 classification) with mild ascites. Tumor marker evaluation showed CA-125 at 30.6 IU/mL. Thoraco-abdomino-pelvic CT demonstrated no evidence of distant metastases.

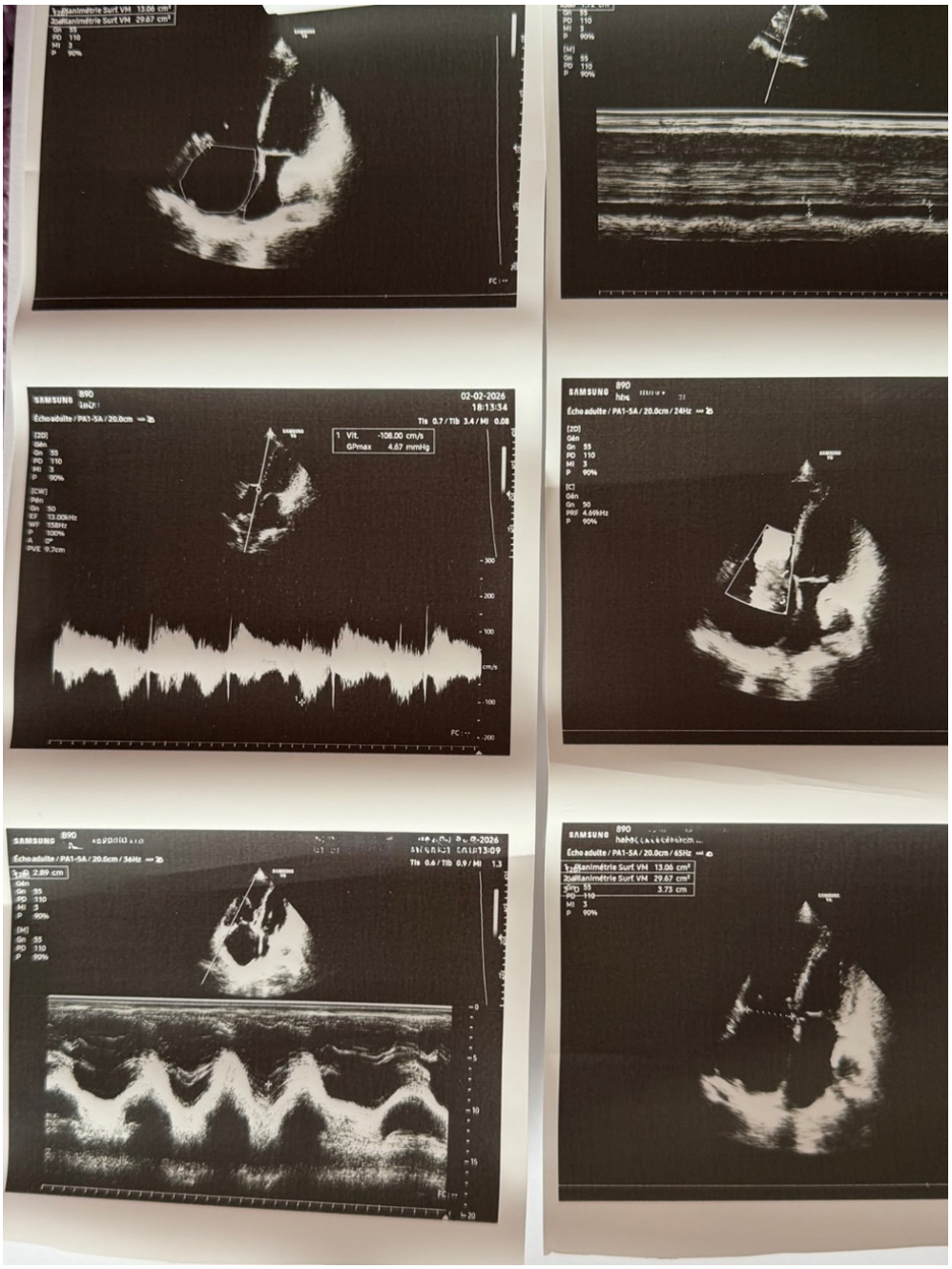

Figure 3: Severe dilation of the right ventricle, with dilation of the right atrium and flattening of the interventricular septum.

Management

The patient was scheduled to undergo optimal cytoreductive surgery with the goal of complete (R0) resection, in conjunction with appropriate medical management of her right-sided heart failure.

Discussion

Pathophysiology and Mechanisms of Carcinoid Heart Disease

Carcinoid heart disease constitutes one of the most formidable complications of carcinoid syndrome, occurring in 20 to 60% of patients with functional metastatic neuroendocrine tumors [1]. This cardiac involvement is directly linked to the deleterious action of vasoactive substances secreted by the tumor, notably serotonin (5-hydroxytryptamine), bradykinin, histamine, and tachykinins (substance P, neurokinin A) [2].

The central pathophysiological mechanism involves serotonin, the principal metabolite synthesized from tryptophan by the enterochromaffin cells of the tumor. Circulating serotonin activates 5-HT2B receptors expressed on cardiac valves, triggering an intracellular signaling cascade leading to fibroblast proliferation and collagen synthesis [3]. This process results in the formation of characteristic white, thick, retractile fibrous plaques that predominate on the ventricular surface of the tricuspid and pulmonary valves. The absence of left-sided involvement in the majority of cases is explained by the pulmonary inactivation of vasoactive substances by monoamine oxidase (MAO) enzymes present in the pulmonary capillary endothelium. Left-sided valvular involvement (mitral or aortic) is therefore only observed in the presence of a persistent right-to-left shunt (patent foramen ovale, interatrial septum defect) or significant pulmonary shunt, allowing vasoactive amines to bypass the pulmonary filter [4].

The fibrotic extension is not limited to the valves but may involve the chordae tendineae, papillary muscles, right ventricular endocardial wall, inferior vena cava, and coronary sinus. This retractile fibrosis progressively leads to mixed valvular lesions combining regurgitation and stenosis, culminating in refractory right heart failure, which represents the leading cause of death in these patients [5].

Diagnostic Aspects and Screening

Cardiovascular manifestations must be systematically sought in every patient with neuroendocrine tumor and carcinoid syndrome, as they may be revealing of the disease, as in our clinical observation. It is important to emphasize that the functional signs of right heart failure — lower limb edema, ascites, jugular venous distension, painful hepatomegaly — appear only late in the disease course.

Transthoracic echocardiography remains the reference examination for the diagnosis and follow-up of carcinoid heart disease [6]. It allows visualization of characteristic valvular thickening, assessment of the severity of regurgitation and stenosis, and evaluation of right ventricular function. The echocardiographic criteria include: thickening and retraction of the tricuspid and/or pulmonary valves, restriction of valvular motion, and valvular calcifications. Transesophageal echocardiography may be necessary for more precise evaluation, particularly in the preoperative setting.

From a biological standpoint, NT-proBNP (N-terminal pro–B-type natriuretic peptide) has emerged as the biomarker of choice for screening and surveillance. A threshold value greater than 260 ng/L suggests significant valvular involvement requiring thorough echocardiographic evaluation [6]. Concurrently, measurement of urinary 5-hydroxyindoleacetic acid (5-HIAA), the principal metabolite of serotonin, holds important prognostic value. Levels exceeding 300 µmol/24h constitute an independent predictive factor for the development of carcinoid heart disease and are associated with decreased survival [7].

Electrocardiography and chest radiography are generally non-contributory in the early stages, although they may reveal enlargement of the right cardiac silhouette or P-wave abnormalities in cases of right atrial hypertrophy.

Prognostic Impact

The presence of carcinoid heart disease represents a major prognostic event that considerably alters patient survival. Contemporary data confirm a median survival of approximately 3 years in affected patients, compared to more than 10 years in patients free of cardiac involvement [8]. This prognostic difference underscores the importance of early detection and appropriate management before the onset of refractory heart failure.

Classification of the disease into stages (mild, moderate, severe) based on echocardiographic and clinical criteria allows risk stratification and guidance of therapeutic management. Patients presenting with severe disease (massive tricuspid regurgitation, significant pulmonary stenosis, right ventricular dysfunction) have a particularly poor prognosis without surgical intervention.

Multidisciplinary Therapeutic Strategy

The management of carcinoid heart disease requires a multidisciplinary approach integrating oncologists, cardiologists, and cardiac surgeons [8].

Medical Treatment and Targeted Therapies

Symptomatic medical treatment includes a low-sodium diet and diuretics to control fluid and sodium retention. However, these measures represent only palliative approaches with transient efficacy.Targeted therapies aim to control both hormonal secretion and tumor progression:

- **Somatostatin analogues:** (lanreotide, octreotide): first-line agents that reduce serotonin secretion and may slow the progression of valvular fibrosis [9]
- **mTOR inhibitors:** (everolimus): demonstrate efficacy in controlling tumor growth and potentially in preventing cardiac progression
- **Peptide receptor radionuclide therapy:** (PRRT): represents a major advance in the management of metastatic

neuroendocrine tumors, with positive impact on carcinoid syndrome and potentially on prevention of cardiac involvement [9]

- **Chemotherapy:** reserved for high-grade tumors or in cases of rapid progression

These systemic treatments, while effective on the tumoral and symptomatic levels, have not demonstrated the ability to reverse established valvular lesions.

Surgical Treatment

Surgical valve replacement constitutes the only curative treatment, significantly improving quality of life and patient survival [10]. Surgery generally involves replacement of the tricuspid valve (often with a bioprosthesis) and sometimes the pulmonary valve.

Despite non-negligible perioperative morbidity and mortality — related notably to the risk of carcinoid crisis induced by tumor manipulation and anesthesia — surgical intervention should be considered early as soon as symptomatic medical treatment proves insufficient, before the onset of irreversible right heart failure. Modern anesthetic protocols include preventive treatment with somatostatin analogues to minimize the risk of perioperative carcinoid crisis [10].

Bioprosthesis is generally preferred over mechanical valve due to the morbidity associated with anticoagulation in these patients. However, the early degeneration of bioprostheses under the effect of persistent vasoactive substances constitutes a therapeutic challenge, justifying optimal control of carcinoid syndrome before and after surgery.

Conclusion

Carcinoid heart disease remains a formidable complication of carcinoid syndrome, associated with poor prognosis. Its complex pathophysiology involving vasoactive substances, notably serotonin, explains the preferential involvement of right-sided valves. Early detection through echocardiography and biomarkers (NT-proBNP, 5-HIAA) proves crucial for identifying at-risk patients before the onset of refractory heart failure. The integration of modern targeted therapies within a multidisciplinary approach, combined with early surgical indication, currently represents the best strategy for improving survival and quality of life in these patients.

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