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Thymoma-Induced Severe Biventricular Failure without Myasthenia Gravis: Investigating Tachycardia-Induced Cardiomyopathy

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Patient: Male, 33-year-old
Final Diagnosis: Tachycardia induced cardiomyopathy
Symptoms: Heart failure
Clinical Procedure: —
Specialty: Cardiology • Immunology • Neurology • Oncology • Pathology
Objective: Rare disease
Background: Cardiomyopathy associated with thymoma is thought to be a cardiac manifestations of myasthenia gravis (MG). However, there are case reports of newly diagnosed thymoma presenting with cardiomyopathy without MG, and the mechanism remains unclear. The purpose of this report is to explore tachycardia-induced cardiomyopathy (TIC) as a potential mechanism for cardiomyopathy in thymoma without features of MG.
Case Report: A 31-year-old man presented with atrial flutter with right bundle branch block and severe biventricular heart failure. Echocardiogram revealed severe left ventricle ejection fraction (LVEF) of 15% with biventricular dilation with impaired systolic function. Computer tomography coronary angiography demonstrated normal coronary artery disease. Cardiac magnetic resonance imaging showed normal T1 and T2 mapping, without inflammation or edema. A large anterior mediastinal mass was found on computer tomography chest. Mediastinal mass biopsy identified type B3 thymoma (WHO classification) with dual population of large, uniform epithelial thymic cells and immature T cell phenotype. Acetylcholine receptor antibody was positive without clinical features of MG and hypogammaglobulinemia indicating Good syndrome. He was treated with antiarrhythmic and heart failure pharmacotherapy, carboplatin and paclitaxel, and intravenous immunoglobulin. He demonstrated reversible heart failure following abolishment of tachyarrhythmia, consistent with tachycardia-induced cardiomyopathy.
Conclusions: We report a rare case of a newly diagnosed thymoma and Good syndrome without clinical features of MG presenting with tachyarrhythmia and severe biventricular failure. The reversibility of the cardiomyopathy following abortion of tachyarrhythmia with treatment highlights TIC as a potential cause.
Keywords: Cardiomyopathies • Cardiovascular Diseases • Heart Failure • Myasthenia Gravis • Tachycardia • Thymoma
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Introduction

Thymoma is a rare neoplasm arising from thymic tissue, with an estimated incidence of 1 to 3 cases per million people per year worldwide [1]. Thymomas typically occur in those aged 40 to 60 years and occur equally in men and women [2]. There are no known risk factors, but thymomas are strongly associated with myasthenia gravis (MG) and other paraneoplastic syndromes [3]. Thymomas can present as an incidental finding in an asymptomatic individual, discovered during MG, or with symptoms due to mass effect, such as cough, superior vena syndrome, chest pain, or dysphagia [1]. Thymomas are usually located ventral to the pericardium, aorta, pulmonary artery, and superior vena cava and are not typically associated with lymphadenopathy [3]. Tissue biopsy is required for definitive diagnosis of thymoma and can be obtained from either surgical resection or biopsy [2]. Surgical resection is the primary treatment of thymomas [4]. For unresectable disease, radiotherapy and chemotherapy can improve symptoms and outcomes [2]. Multidisciplinary involvement to create individual surgical approaches and systemic therapy improve outcomes [4].

Thymoma is associated with autoantibody formation and paraneoplastic syndrome, most commonly with acetylcholine receptor antibodies, leading to MG [3]. MG is a neuromuscular autoimmune disease with defective neuromuscular transmission in skeletal muscles due to antibodies to the acetylcholine receptor at the neuromuscular junction [5]. Other commonly associated syndromes with thymoma are pure red cell aplasia and Good syndrome [2]. Pure red cell aplasia is caused by severe normocytic anemia, reticulocytopenia, and absence of erythroblasts in the bone marrow [3]. Good syndrome is a characterized by acquired hypogammaglobulinemia with thymoma [6].

Cardiomyopathy with thymoma is rare and usually occurs with MG [7]. It typically presents as giant cell myocarditis or Takotsubo cardiomyopathy, which are recognized manifestations of MG [8]. Giant cell myocarditis is a severe form of myocarditis with myonecrosis attributed to T-cell lymphocyte-mediated inflammation of the heart muscle associated with systemic autoimmune disease [9]. The mechanism of giant cell myocarditis with MG is unclear but well reported [10]. Takotsubo cardiomyopathy is a temporary dysfunction of the left ventricle (LV) in the absence of coronary stenosis precipitated by emotional or physical stress, which causes catecholamine surge [8]. MG has been reported to precipitate severe stress and subsequent Takotsubo cardiomyopathy [11].

In cases of thymoma presenting with cardiomyopathy without MG, the mechanism remains uncertain [7,12,13]. Anti-Kv1.4-antibody has been identified as a potential cause of autoimmune myocarditis in thymoma, but this is based on studies of patients with MG [7]. Tabet et al reported a patient with a

thymoma presenting with right heart failure and treated with surgical resection [12]. Priester et al reported a patient with malignant thymoma presenting with biventricular failure unsuitable for surgical resection and treated with systemic chemotherapy [13]. All patients reported had thymoma presenting with tachycardia and heart failure without symptoms related to mass effect of thymoma or clinical features of MG. We hypothesize that the mediastinal involvement by the thymoma may cause local irritation driving tachycardia-induced cardiomyopathy (TIC).

TIC is defined as reversible heart failure caused by persistent tachyarrhythmia, resulting in ventricular dysfunction [14]. The incidence is not well-defined and often underdiagnosed [15]. Heart failure symptoms, including shortness of breath, fatigue, and edema occur between 3 and 120 days of onset of tachycardia and manifest earlier with high rates of tachycardia [15]. The definitive diagnosis is established by recovery or improvement of LV systolic function within first 6 months after eliminating the culprit tachyarrhythmia [15]. The primary treatment involves suppressing the tachyarrhythmia with antiarrhythmic medication or ablation as well as initiating and optimizing heart failure medications [16].

We present a case of thymoma presenting with tachyarrhythmia and biventricular heart failure and an anterior mediastinal mass diagnosed thymoma with Good syndrome but without clinical features of MG. TIC was considered the potential mechanism.

Case Report

A 33-year-old man presented with 2 weeks of worsening dyspnea and orthopnea. His past medical history included asthma, and he took fluticasone 250 mcg and salmeterol 50 mg inhalation twice daily. There was no family history of cardiac disease. He was a non-smoker without a history of alcohol or illicit drug use. Physical examination revealed clinical evidence of left- and right-sided heart failure, with gross upper and lower limb edema, ascites, and bilateral chest crepitations. Electrocardiogram showed rapid atrial flutter at 134 beats per minute, with right bundle branch block and left anterior fascicular block (**Figure 1**). There were no significant abnormalities in complete blood count with differentials, electrolytes, troponin I, kidney, liver, and thyroid function found (**Table 1**). Respiratory viral swabs were negative. Chest X-ray (CXR) revealed bilateral pleural effusions and pulmonary edema (**Figure 2A**). Computer tomography (CT) chest revealed an anterior mediastinal mass measuring 118×79 mm with right pleural metastasis and trans-diaphragmatic extension, without direct myocardial involvement (**Figure 2B**). Echocardiogram demonstrated a severe LV and right ventricle (RV) dilation

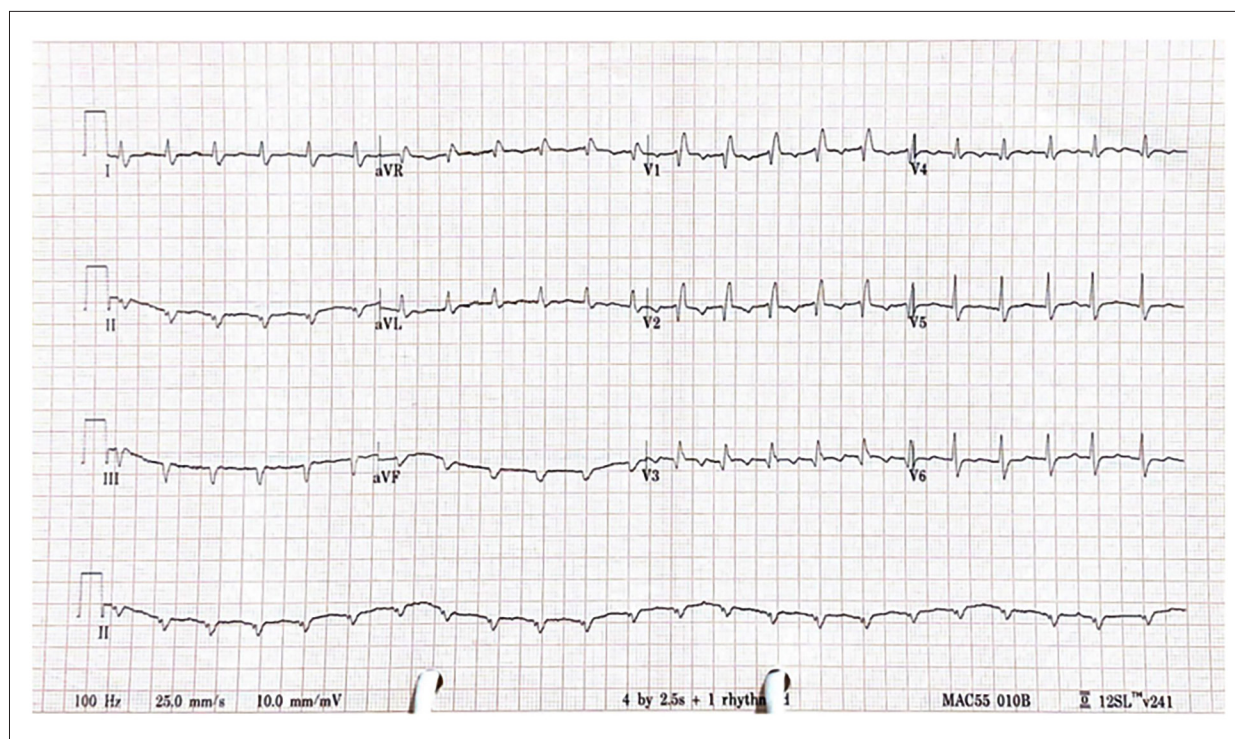


Figure 1. Electrocardiogram demonstrating atrial flutter with right bundle branch block and left anterior fascicular block.

with impaired systolic function; the estimated LVEF was 15% (**Video 1**). There was moderate mitral regurgitation and severe tricuspid regurgitation (**Videos 2, 3**).

The differentials for his acute failure included ischemic and non-ischemic causes, including idiopathic dilated, tachycardia-induced, and arrhythmogenic cardiomyopathies. Myocarditis secondary to viruses or autoimmune conditions were also considered. Given his clinical instability and low pre-test probability for ischemia and coronary artery disease based on the absence of chest pain, minimal cardiac risk factors, normal troponin, and no ischemic changes on ECG, we selected CT coronary angiogram for minimally invasive risk stratification. The results showed normal coronaries reducing the likelihood of ischemic cardiomyopathy. He then underwent cardiac magnetic resonance imaging to investigate for non-ischemic cardiomyopathy, which displayed severe biventricular dilatation with severe systolic impairment, both LV and RV EF were 18%, without inflammation or edema (**Video 4**). Overall, investigations showed a structurally normal heart without fibrosis, inflammation, or edema as evidence by normal T1 and T2 mapping making causes such as hypertrophy, myocarditis, arrhythmogenic, and infiltrative disorders such as amyloidosis, sarcoidosis, iron overload, or Fabry disease less likely. The leading differentials were idiopathic dilated and tachycardia-induced cardiomyopathy.

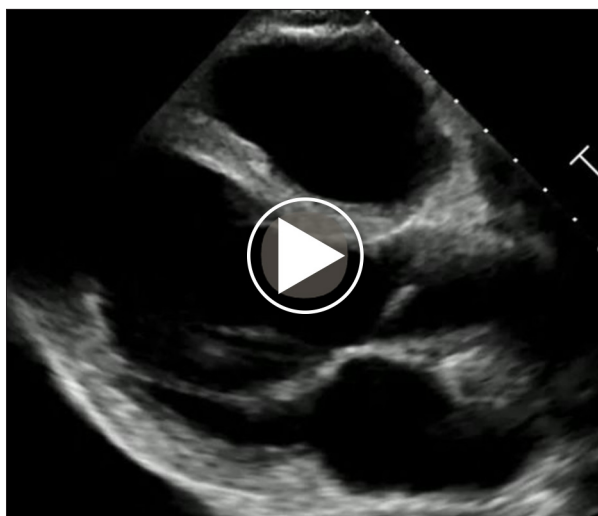
The clinical and radiological differential diagnosis for his mass includes thymic tumor, lymphoma, or germ cell tumor. Fluorodeoxyglucose-positron emission tomography revealed increased activity in the corresponding anterior mediastinal mass, with contiguous involvement of the right lower pleural space and right lobe of liver. The serum lactate dehydrogenase was 498 mU/L, alpha fetoprotein, 3.8 ug/L, and beta-human chorionic gonadotrophin, <2 IU/L. There were no focal testicular lesions or scrotal masses on ultrasound. Mediastinal mass biopsy indicated features consistent with a type B3 thymoma (WHO Classification), with a dual population of large but uniform epithelial thymic cells, without cytological pleomorphism or necrosis, and prominent background lymphocytes showing an immature T-cell phenotype, with immunohistochemistry showing CD99 and CD1a positivity despite equivocal TdT (**Figure 3**). CD117 was negative in epithelioid thymic cells. PLAP, CD30, and Oct3/4 were also negative, ruling out a germ cell tumor. Thymic carcinomas would tend to have positive CD117, and an infiltrate of mature T lymphocytes (meaning TdT, CD99, and CD1a should be negative). There were also no overt features of squamous cell carcinoma. However, due to aggressive clinical and radiological features, thymic carcinoma could not be excluded when correlating histopathological results and clinical features. MG screening demonstrated positive acetylcholine receptor antibodies, with a value of 1.98 nmol/L, but was negative for muscle-specific kinase antibodies. Neuronal antibodies, autoimmune screen, and myositis

Table 1. Laboratory results.

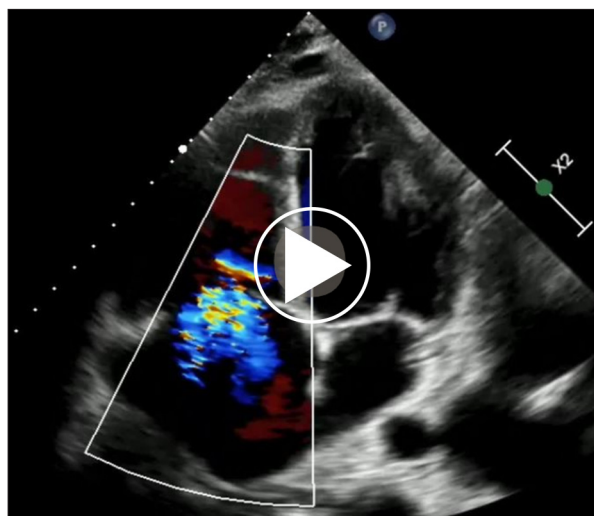
Blood work	Results	Reference range
White blood cells	5.9×10 ⁹ /L	4.0-11.0×10 ⁹ /L
Red blood cells	4.9×10 ¹² /L	4.5-6.5×10 ¹² /L
Hemoglobin	141 g/L	130-180 g/L
Hematocrit	0.43	0.40-0.54
Platelets	216×10 ⁹ /L	150-400×10 ⁹ /L
Neutrophils	5.0×10 ⁹ /L	4.0-11.0×10 ⁹ /L
Lymphocytes	0.6×10⁹/L	1.5-4.0×10⁹/L
Monocytes	0.2×10 ⁹ /L	0.2-1.0×10 ⁹ /L
Eosinophils	0.0×10 ⁹ /L	0.0-0.4×10 ⁹ /L
Basophils	0.0×10 ⁹ /L	0.0-0.1×10 ⁹ /L
Sodium	141 mmol/L	135-145 mmol/L
Potassium	4.4 mmol/L	3.5-5.2 mmol/L
Chloride	100 mmol/L	95-110 mmol/L
Bicarbonate	27 mmol/L	22-32 mmol/L
Urea	8.7 mmol/L	3.5-8.0 mmol/L
Creatinine	83 umol/L	60-110 umol/L
Estimated glomerular filtration rate	>90 mL/mn/1.73 m ²	>60 mL/mn/1.73 m ²
Phosphate	1.8 mmol/L	0.7-1.5 mmol/L
Calcium	2.3 mmol/L	2.2-2.6 mmol/L
Magnesium	0.9 mmol/L	0.7-1.1 mmol/L
Albumin	36 g/L	33-48 g/L
Total protein	64 g/L	60-80 g/L
Total bilirubin	20 umol/L	0-20 umol/L
Alanine transaminase	40 U/L	0-40 U/L
Aspartate aminotransferase	33 U/L	0-35 U/L
Alkaline phosphatase	91 U/L	30-110 U/L
Gamma-glutamyl transpeptidase	36 U/L	0-50 U/L



Figure 2. (A) Chest X-ray showing patchy opacities in right mid to lower zone with associated large right pleural effusion and increased interstitial markings, vascular congestion, and peribronchial cuffing in both lungs suggestive of pulmonary edema. (B) Computed tomography of the chest demonstrating anterior mediastinal mass with invasion of the mediastinal fat and extensive right pleural and trans-diaphragmatic invasion.



Video 1. Echocardiogram of parasternal long axis view showing severe left ventricular dilatation with severe impairment of systolic function.



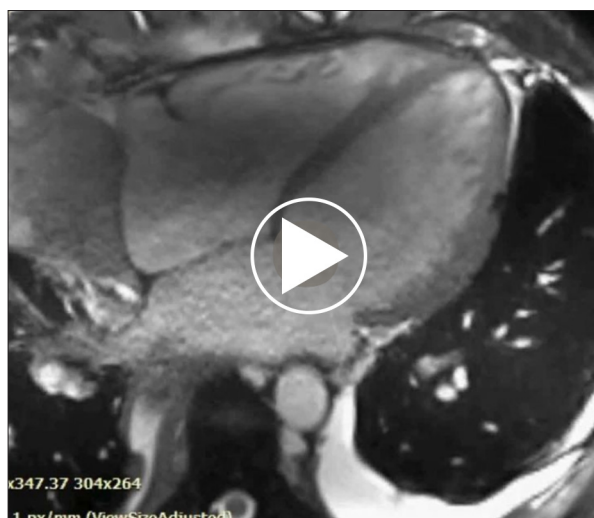
Video 3. Echocardiogram of apical four chamber view showing severe right ventricular dilatation with severe impairment of systolic function, and severe tricuspid regurgitation.



Video 2. Echocardiogram of apical four chamber view showing moderate mitral regurgitation.

panels were negative. Immunoglobulin G levels were found to be low at 5.4g/L, consistent with Good syndrome.

The patient was admitted under the heart failure service and commenced on intravenous dobutamine 5 mcg/kg/h and frusemide 10 mg/h infusions. He underwent cardioversion from atrial flutter to sinus rhythm with amiodarone and anticoagulation with enoxaparin 80 mg subcutaneously twice daily. Oncology, neurology, and immunology teams were involved in his care. Given hypogammaglobulinemia, clinical instability, positive acetylcholine antibodies, and thymic malignancy-MG association, intravenous immunoglobulin was commenced despite lacking clinical features of MG. Multidisciplinary consensus deemed the mass unsuitable



Video 4. Cardiac magnetic resonance demonstrating severe impairment of systolic function with left ventricle and right ventricle ejection fraction of 18% without inflammation or edema.

for local therapy, including radiation or surgery, based on size and invasion of major vessels, pleura, and diaphragm. Chemotherapy began on a cardiac monitor with carboplatin and paclitaxel, chosen over anthracycline-based regimen due to potential cardiotoxicity.

There was good response to diuresis with reduction in pleural effusion and pulmonary edema seen on progress chest X-ray (**Figure 4A**). Once euvolemic status was achieved, heart failure pharmacotherapy with bisoprolol 5 mg oral twice daily, dapagliflozin 10 mg oral daily, perindopril 2.5 mg oral twice daily, and

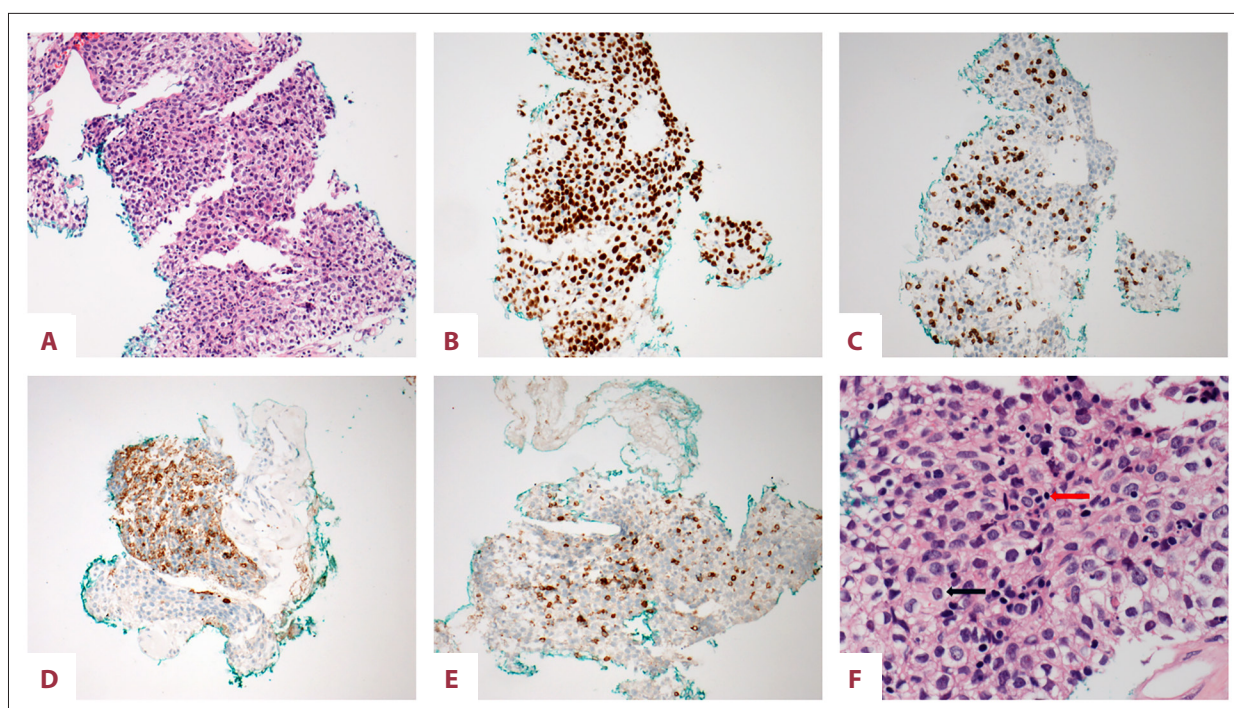


Figure 3. (A) Small fragmented biopsy showing type B3 thymoma (H&E ×200); (B) large epithelioid cells (highlighted with P40 immunohistochemistry 200); (C) lymphocytic component with an immature T-cell phenotype CD3 ×200; (D) Factor1A ×200; (E) CD99 positive ×200; (F) High power view of uniform, large clear thymic epithelial cells (black arrow) and scattered small mature lymphocytes (red arrow) (H&E ×600).

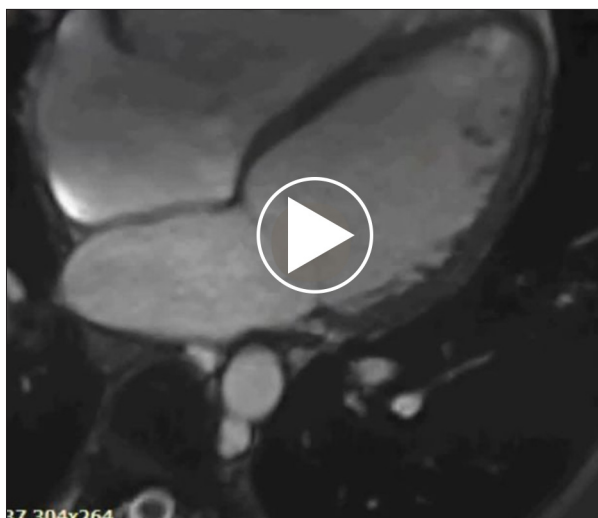


Figure 4. (A) Progress chest X-ray after 2 weeks demonstrating improved pulmonary edema and pleural effusion. (B) Progress computed tomography of the chest after 4 months showing reduction in size of anterior mediastinal mass after chemotherapy.

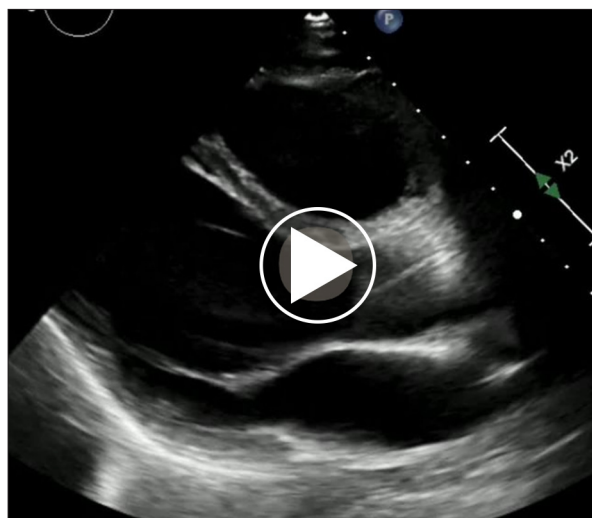
spironolactone 25 mg daily was gradually introduced. He had several runs of asymptomatic non-sustained ventricular tachycardia, up to 15 beats. Bilateral upper limb deep vein thromboses were diagnosed, precluding implantation of a transvenous defibrillator. The cardiothoracic surgical team was unable to implant a subcutaneous defibrillator, due to thoracic malignancy. In discussion with the electrophysiology team, the patient elected to have a wearable defibrillator fitted. Progress echocardiography 2 weeks later showed improved LV systolic function, with an EF of 25% to 35%. A repeat cardiac magnetic

resonance showed improvement in biventricular systolic function, with 37% LVEF and 39% RVEF (**Video 5**). He was clinically stable and discharged after a 3-week hospital admission.

He continued 3-weekly carboplatin and paclitaxel with partial response, as evidence by follow-up chest CT after 6 cycles (**Figure 4B**), with anterior mediastinal mass measuring 70×60 mm and reduction of liver and pulmonary involvement. Empirical 4-weekly intravenous immunoglobulin continued. He did not develop clinical features of MG.



Video 5. Progress cardiac magnetic resonance after 3 weeks demonstrating improved 37% left ventricle ejection fraction and 39% right ventricle ejection fraction with moderate biventricular systolic impairment improved, compared with previous imaging.



Video 6. Progress echocardiogram of parasternal long axis view at 5 months showing mild left ventricular dilatation with left ventricle ejection fraction of 45% and mildly dilated right ventricle with normal systolic function improved, compared to previous imaging.

At the 5-months follow-up, the patient remained well and eu-volemic, without further hospitalizations. He remained on bisoprolol, dapagliflozin, spironolactone, and frusemide, with successful introduction of sacubitril/valsartan. Amiodarone prevented further symptomatic arrhythmias, and his wearable defibrillator was discontinued. Echocardiography showed improvement with LVEF at 45%, and mildly dilated RV with normal systolic function (**Video 6**). Mitral and tricuspid regurgitation significantly improved.

Discussion

We suggest TIC as a likely cause for the thymoma-associated cardiomyopathy without clinical features of MG in our presented patient. Thymoma typically occurs in patients aged 40 to 60 years and present incidentally on chest X-ray, during MG work-up, or with symptoms due to mass effect, such as cough, superior vena syndrome, chest pain, and dysphagia [2]. The patient presented here had heart failure symptoms and was younger than the typical age, without mass effect symptoms or clinical findings of MG. Thymoma-associated cardiomyopathy typically occurs with MG and presents giant cell myocarditis or Takotsubo cardiomyopathy [8]. However, our patient had negative neuronal antibodies, autoimmune screen, and myositis panels, and had no evidence of cardiac inflammation or edema on cardiac magnetic resonance imaging to suggest giant cell myocarditis. There was also no echocardiographic evidence of Takotsubo cardiomyopathy. TIC is heart failure caused by persistent tachyarrhythmias resulting in ventricular dysfunction and diagnosed by evidence of reversibility within first 6

months after eliminating the culprit tachyarrhythmia [15]. The presentation of heart failure with tachyarrhythmia with subsequent reversibility of ventricular dysfunction following resolution of tachyarrhythmia and introduction of heart failure pharmacotherapy is diagnostic of TIC.

Tabet et al reported a patient diagnosed with a thymoma presenting with right heart failure and treated with surgical resection [12]. Priester et al reported a patient with malignant thymoma presenting with biventricular failure unsuitable for surgical resection and treated with systemic chemotherapy of intravenous cisplatin 100mg/m², doxorubicin 60mg/m², and cyclophosphamide 60mg/m² [13]. All patients reported presented with tachyarrhythmia and cardiomyopathy and mediastinal mass diagnosed as thymoma without clinical features of MG [12,13]. **Table 2** summarizes the presenting symptoms and rhythm, imaging, and biopsy results. Heart failure resolved following treatment of the thymoma [12,13]. However, the previous case reports do not discuss the use of antiarrhythmic or heart failure pharmacotherapy or provide follow-up cardiac imaging to demonstrate improvement of ventricular function [12,13]. We hypothesize that the mediastinal involvement by the thymoma cause local irritation, driving tachyarrhythmias resulting in cardiomyopathy and heart failure. Cardiomyopathy in all reported patients resolved with treatment of thymoma, demonstrating reversibility in keeping with TIC.

Despite this, there is still the question of whether a primary cardiomyopathy associated with the thymoma as a paraneoplastic phenomenon is the primary diagnosis. Endomyocardial biopsies were not indicated in any of the cases; thus, there

Table 2. Comparing cases of thymoma without clinical MG presenting with heart failure.

Authors	Patient demographics	Presenting rhythm	Imaging	Pathology and immunohistochemistry	MG-related antibodies
Tabet, et al [12]	31-year-old man with 2-week history of worsening exertional dyspnea, ascites, and lower limb edema	Sinus tachycardia	CT: retrosternal irregular mass (69×46 mm) with pericardial effusion and bilateral pleural effusion	Lympho-epithelial thymoma with atypical and aggressive cells. Positive anti-LCA, anti-pan cytokeratin, and anti-CD1a antibodies	Acetylcholine and MusK antibodies negative
Priester, et al [13]	60-year-old man with biventricular cardiac failure	Sinus tachycardia	CT: mediastinal tumor with infiltration to both lungs, vascular structures, and dissemination to chest wall	Invasive cortical thymoma, no immunohistochemistry provided	Not provided
Presented Case	33-year-old male with 2-week history of dyspnea, orthopnea, ascites, and lower limb edema	Atrial flutter	CT: anterior mediastinal mass measuring 118×79 mm with right pleural metastasis and trans-diaphragmatic extension	Large cohesive epithelioid cells, forming sheets with significant proportion of infiltrating mature lymphocytes. Positive C3, CD5, CD99, and CD1a	Acetylcholine antibody positive, MusK antibody negative

are no histological samples to definitively rule out microscopic invasion of thymoma or evidence of an inflammatory process or myositis. Our hypothesis of TIC as the mechanism of biventricular failure of cases with thymoma without clinical feature of MG is limited and based on comparison of our presented patients with other case reports.

Conclusions

We report a rare case of a newly diagnosed thymoma with positive acetylcholine receptor antibody and Good syndrome, but without clinical features of MG, presenting with tachyarrhythmia and severe biventricular failure. The patient was treated with antiarrhythmic and heart failure pharmacotherapy, intravenous immunoglobulin, and chemotherapy, based on multi-disciplinary consensus. Reversibility of the cardiomyopathy was demonstrated following termination of tachyarrhythmia, with improved ventricular function with dilation progress imaging. The case highlights TIC as a cause of cardiomyopathy in thymoma without clinical features of MG.

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Informed Consent

The patient provided consent for publication.

Declaration of Figures’ Authenticity

All figures submitted have been created by the authors who confirm that the images are original with no duplication and have not been previously published in whole or in part.

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