Mimicking Acute Coronary Syndrome: Cardiac Thyrotoxicosis - A Rare Case Report

ABSTRACT

This case report details the clinical presentation, diagnosis, and multidisciplinary management of a 68-year-old man with cardiac thyrotoxicosis mimickingan acute coronary syndrome (ACS). The patient presented with persistent anginal chest pain, systemic symptoms, and electrocardiographic abnormalities suggestive of ACS. However, a comprehensive evaluation revealed thyrotoxicosis-associated dilated cardiomyopathy, challenging the initial diagnostic impressions. The case highlights the intricate relationship between thyroid dysfunction and cardiovascular manifestations, emphasizing the need for a multidisciplinary approach. Management included standard ACS protocols, careful titration of antithyroid medications, and the transition from intravenous to oral heart failure medications. Follow-up assessments demonstrated the success of personalized treatment strategies, with the normalization of thyroid function and the emergence of atrial fibrillation. This case underscores the importance of considering diverse cardiac presentations in hyperthyroid patients and the effectiveness of individualized management approaches in navigating the complexities of thyroid-cardiac interactions.

Keywords: Cardiac thyrotoxicosis, Acute coronary syndrome, Heart failure, Dilated cardiomyopathy

Introduction :

An acute coronary syndrome (ACS) is a constellation of signs and symptoms associated with insufficient flow through the coronary tree and the resultant acute ischemia of the myocardium. The cardinal sign of a patient with ACS is chest pain that is usually described as central, substernal, like a pressure, and it can be nonradiating or can radiate to the left shoulder, to the jaw, or to the left arm [9,10].

CASE PRESENTATION

We present the case of a 68-year-old man, hypertensive and chronic smoker, who sought emergency care for persistent anginal chest pain evolving over 10 days and worsening over the last 14 hours. The patient's medical history revealed no particular pathological antecedents or coronary heredity, and no other modifiable cardiovascular risk factors. The retrosternal, constrictive pain irradiating to the jaw and interscapular region occurred at rest, was prolonged, nitrate-resistant, and worsened 14 hours before admission to the emergency department, accompanied by concurrent palpitations. The comprehensive history revealed a generalized feeling of asthenia with excessive sweating and diarrhea over the past 10 days, without abdominal pain, vomiting, or fever.

On general clinical examination, the patient was conscious, hemodynamically stable with a blood pressure of 141/82 mmHg, a heart rate of 140 bpm, eupneic at rest, and a saturation of 98% in ambient air. Auscultation revealed bilateral symmetric crepitant rales at the lung bases, with no signs of right heart failure. The patient was afebrile at 37.1°C. Thyroid palpation yielded normal results. The admission ECG showed a regular sinus rhythm at 140 bpm, a fixed and constant PR interval at 160 ms, left-axis deviation, complete left bundle branch block (LBBB) with secondary repolarization abnormalities (negative Sgarbossa criteria). Serial laboratory tests revealed elevated troponins at 429.3 ng/L (30 times the upper limit of normal), a suppressed TSH below 0.01 mIU/L with increased free T3 and T4 levels at 20 ng/L and 3.6 ng/dL, respectively. Inflammatory markers were elevate. Echocardiography demonstrated features of dilated cardiomyopathy with global severe hypokinesis and left ventricular dysfunction (left ventricular ejection fraction: 25%),

elevated left ventricular filling pressures, and no mitral or aortic valve pathologies. The probability of pulmonary hypertension was low.

This case underscores the clinical complexity, combining acute coronary syndrome, dilated cardiomyopathy, and thyrotoxicosis. A multidisciplinary approach is crucial for effective management of these diverse components.

The patient was admitted to the cardiology intensive care unit and closely monitored. He has received the loading dose of clopidogrel 300 mg and aspirin 300 mg, along with heparin therapy. The gradual introduction of the rest of the heart failure management regimen is underway.

The patient's case was discussed in a multidisciplinary meeting, during which endocrinologists did not authorized performing a coronary angiography due to the context of thyrotoxic crisis. Treatment with synthetic antithyroid drugs and corticosteroids was initiated, showing a significant improvement in both clinical and biological aspects. Coronary angiography was authorized 10 days after the initiation of the treatment, revealing no abnormalities (figure 1).

Cardiac magnetic resonance imaging (MRI) was also realized (figure 2), revealing dilated cardiomyopathy without specific signs and no evidence supporting myocarditis.

2. DISCUSSION

The presented case of a 68-year-old hypertensive and chronic smoker reveals a complex interplay between cardiac thyrotoxicosis and symptoms mimicking acute coronary syndrome (ACS). The patient's atypical clinical presentation, characterized by persistent anginal chest pain, systemic symptoms, and unique electrocardiographic abnormalities, prompted a comprehensive diagnostic and therapeutic approach.

The diagnostic challenges in this case stemmed from the overlapping features of cardiac thyrotoxicosis and ACS. The coexistence of palpitations, excessive sweating, diarrhea, and asthenia, raised suspicion for an alternative etiology. The admission ECG, indicative of sinus tachycardia, left bundle branch block (LBBB), and repolarization abnormalities, along with elevated troponin levels, complicated the diagnostic landscape. The subsequent revelation of thyrotoxicosis, in conjunction with echocardiographic evidence of dilated cardiomyopathy, highlighted the intricate relationship between thyroid dysfunction and cardiovascular manifestations.

The phenomenon of thyrotoxicosis-induced dilated cardiomyopathy is a rare but recognized manifestation of hyperthyroidism. Thyroid hormones influence myocardial contractility, heart rate, and peripheral vascular resistance, and the hyperadrenergic state induced by thyrotoxicosis can lead to dilated cardiomyopathy in severe cases (1)(2). The unique combination of dilated cardiomyopathy, ACS-like symptoms, and thyrotoxicosis in this case aligns with existing literature emphasizing diverse cardiac presentations in hyperthyroid patients(3)(4).

The multidisciplinary approach employed in the management of this case was crucial. Admission to the cardiology intensive care unit, initiation of antiplatelet therapy, and anticoagulation followed standard ACS protocols. However, the disturbed thyroid profile warranted caution, leading to the postponement of coronary angiography. The subsequent introduction of antithyroid medications and corticosteroids, coupled with careful titration of beta-blockers and the transition from intravenous to oral diuretics, reflected the dynamic nature of the treatment strategy.

The successful outcomes observed, including the normalization of thyroid function and the gradual introduction of heart failure medications, support the individualized treatment approach in complex clinical scenarios. Follow-up assessments, encompassing thyroid function tests and electrocardiography, played a pivotal role in monitoring the patient's progress. The detection of atrial fibrillation on follow-up ECG underscored the dynamic nature of the relationship between thyroid dysfunction and cardiac manifestations(5).

The predominant manifestation of cardiothyrotoxicosis is heart failure rather than acute coronary syndrome(6)(7). While both conditions may share some clinical features, the underlying pathophysiological mechanisms differ significantly. In cardiothyrotoxicosis, the excessive levels of thyroid hormones, particularly triiodothyronine (T3), can lead to a hyperdynamic circulatory state. This state is characterized by increased heart rate, elevated cardiac output, and reduced systemic vascular resistance.

The impact of thyroid hormone excess on the cardiovascular system includes direct effects on myocardial contractility and relaxation, as well as alterations in peripheral vascular resistance(8). These changes collectively contribute to the development of a high-output heart failure syndrome. The increased demand on the heart, coupled with potential structural changes such as myocardial hypertrophy, can result in dilated cardiomyopathy and compromised cardiac function over time.

In contrast, acute coronary syndrome primarily involves a disruption in coronary blood flow, leading to myocardial ischemia or infarction. The clinical presentation often includes chest pain or discomfort, shortness of breath, and other symptoms related to insufficient blood supply to the heart muscle. This condition is commonly associated with atherosclerotic plaque rupture, thrombus formation, and subsequent coronary artery obstruction.

While there can be overlapping symptoms between cardiothyrotoxicosis and acute coronary syndrome, distinguishing between the two is crucial for appropriate management. The characteristic feature of heart failure in cardiothyrotoxicosis, in the absence of significant coronary artery disease, highlights the importance of considering thyroid function evaluation in patients presenting with heart failure symptoms. This differentiation is vital to tailor the treatment approach, with a focus on addressing the underlying thyroid dysfunction in cardiothyrotoxicosis rather than the coronary artery pathology seen in acute coronary syndrome.

Figure 1. The coronary angiography showing a left dominancenetwork with no significant atheromatous lesions.

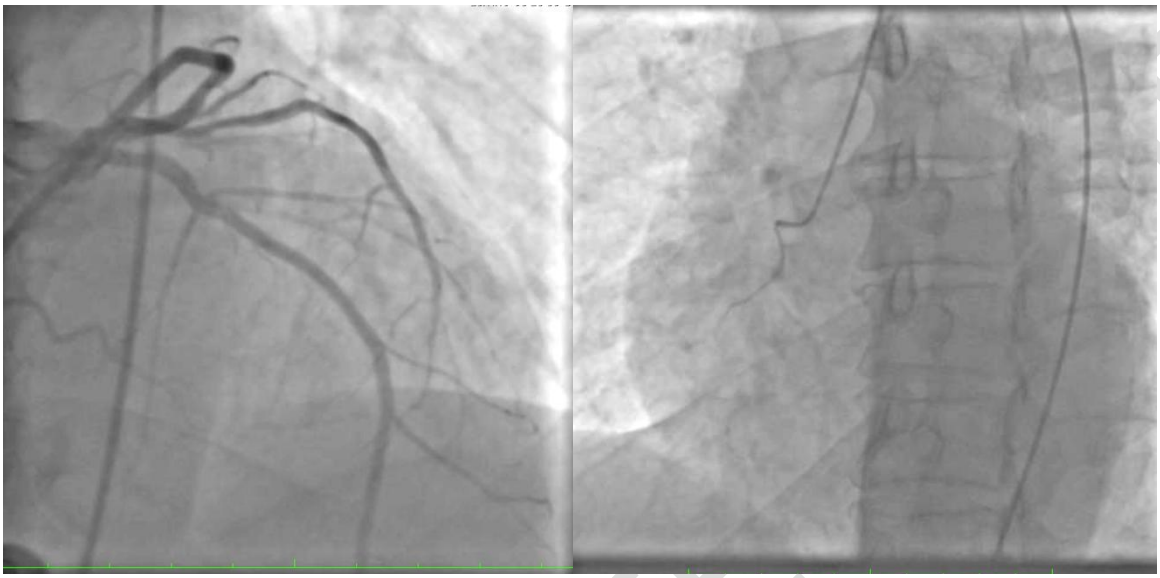
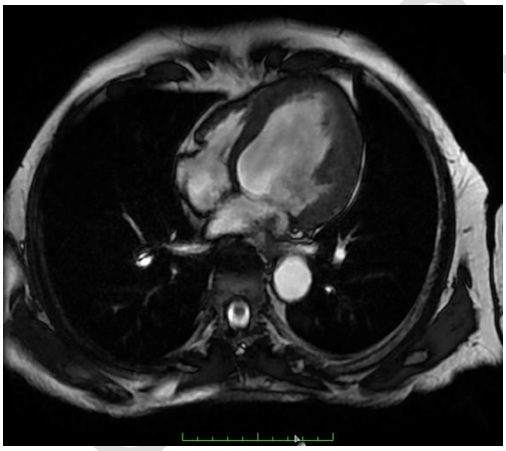


Fig.2.Cardiac MRI showing dilated cardiomyopathy without specific pattern



4. CONCLUSION

In conclusion, this case illustrates the intricate interplay between cardiac thyrotoxicosis and ACS-like symptoms, emphasizing the challenges in diagnosis and the significance of a multidisciplinary approach. The successful management, guided by a nuanced understanding of thyroid function and cardiovascular dynamics, reinforces the importance of individualized treatment strategies in complex clinical scenarios.

Consent

As per international standards or university standards, patient written consent has been collected and preserved by the authors.

Ethical approval

All authors hereby declare that all experiments have been examined and approved by the appropriate ethics committee and have therefore been performed in accordance with the ethical standards laid down in the 1964 Declaration of Helsinki.

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