



CASE REPORT

Thyrotoxicosis as the cause of acute recurrent perimyocarditis

Dino Mirić¹, Duška Glavaš¹, Ljubica Juretić Kuščić², Darija Baković Kramarić¹, Ajvor Lukin¹, Marisa Klančnik³, Maja Grgec³, Petar Ivanišević³

¹Department of Cardiology, University Hospital Split, Split, Croatia,

²Department of Nuclear medicine, University Hospital Split, Split, Croatia,

³Department of Otorhinolaryngology, University Hospital Split, Split, Croatia

Corresponding author:

Petar Ivanišević, Department of Otorhinolaryngology, University Hospital Centre Split, Spinčićeva 1, 21 000 Split, Croatia. Tel.: 0038521556410. e-mail: pivanisevic@kbsplit.hr

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Abstract

We present a case of a 32-year old patient with acute recurrent perimyocarditis associated with thyrotoxicosis. To the best of our knowledge, this is the first reported case of acute recurrent pericarditis associated with increased thyroid hormones in the bloodstream and accompanied by a significant increase in cardioselective enzymes and reduced ejection fraction. Although some authors have described a single episode of myopericarditis mimicking acute coronary syndrome, it was not recurrent. Clinical improvement and normalization of laboratory results following the successful management of thyrotoxicosis, suggests a possible link between these two clinical entities—myopericarditis and thyrotoxicosis.

Key words: Perimyocarditis; thyrotoxicosis

1. Introduction

Perimyocarditis is predominant myocarditis with pericardial involvement and is characterized by evidence of new-onset focal or diffuse reduction of left ventricular function in patients with elevated myocardial biomarkers and clinical criteria for acute pericarditis [1].

Thyrotoxicosis refers to a clinical state that results from inappropriately high-thyroid hormone action in tissues [2]. A few case reports suggest an association between acute pericarditis and Graves' disease, but there are no published case reports of acute recurrent perimyocarditis due to excess thyroid hormones in the bloodstream. In this case report, we describe acute recurrent perimyocarditis as a consequence of unrecognized thyrotoxicosis.

2. Case Report

A 32-year-old male patient was admitted to the Coronary Care Unit in the University Hospital Center Split due to intense chest pain, specific electrocardiography (ECG) findings, and elevated cardioselective enzymes, raising suspicion for the acute coronary syndrome. Medical history showed that he was admitted to the hospital a year earlier because of the same reason. During his previous stay, coronarography showed no pathological changes of the coronary arteries, and echocardiography revealed a hyperechogenic pericardium in the posterolateral area, with minimal pericardial effusion. Laboratory findings at that time showed subclinical hyperthyroidism, but no medical therapy was prescribed. Symptoms and clinical findings spontaneously improved within 3–4 days and the patient was discharged with the diagnosis of acute pericarditis.

During the examination, the only pathological finding was the presence of a pericardial friction rub, heard as a scratchy sound produced by the heart muscle rubbing against the inflamed pericardium. An ECG showed sinus rhythm with concave ST-elevation in leads I, II, and III, aVL, aVF, and V3-V6. Laboratory results showed increased creatine kinase (469 U/L, normal 50–177 U/L), and high-sensitive troponin I 229.1 ng/L (normal <34.2 ng/L). One day later, sensitive troponin increased to 15011.0 ng/L, and creatine kinase to 2432 U/L. Complete blood count, coagulation parameters, acid-base status, C-reactive protein, and other proteins and enzymes were normal. Due to the prior

medical history of subclinical hyperthyroidism, thyroid function tests were performed, revealing thyrotoxicosis. The thyroid-stimulating hormone level was 0.026 mIU/L (normal 0.3–3.6 mIU/L), T3 was 4.4 nmol/L (normal 1.2–3.6), and T4 was 202 nmol/L (normal 58–161 nmol/L). Echocardiography showed normal dimensions of heart chambers, but the ejection fraction was reduced from 75% to 54% in comparison to the year before. Echocardiography also showed a globally hyperechogenic pericardium with a 3-mm pericardial effusion [Figure 1]. Thyroid ultrasound showed a hypoechoic gland with multiple calcifications [Figure 2]. Coronarography showed no abnormalities of the coronary arteries, and ACS was once again excluded. Due to the similarities with the previous presentation the year before, another episode of perimyocarditis due to thyrotoxicosis was diagnosed.



Figure 1. Two-dimensional transthoracic echocardiography showed a hyperechogenic pericardium with a 3 mm pericardial effusion

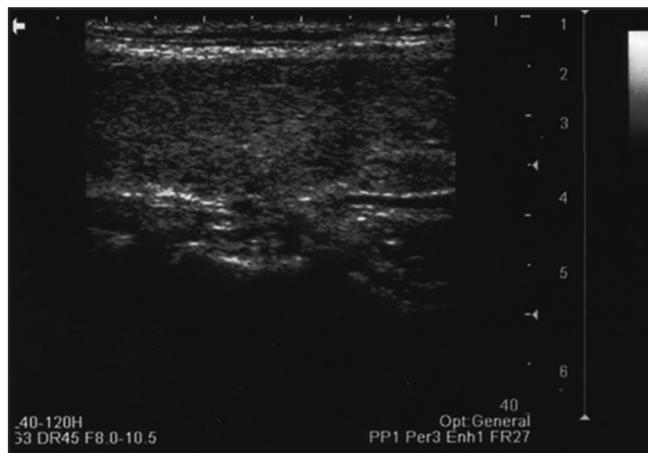


Figure 2. Inhomogeneous echo texture of the thyroid gland with multiple calcifications—diffuse thyroid disease

After the patient was diagnosed with thyrotoxicosis, thiamazole treatment was started at 10 mg 3 times per day for the first 10 days, then twice per day for the next 3 weeks, and finally 15 mg a day until control. Besides the thyrostatic therapy, colchicine 0.5 mg twice per day, propranolol 40 mg twice per day, and ibuprofen 600 mg 3 times per day was prescribed. Within 24 h, the patient's chest pain subsided, and the patient showed significant clinical improvement. ECG changes improved within 10 days. The patient was discharged with the prescribed therapy, in a clinically stable condition, without symptoms.

A month after discharge serum thyroid hormone examination showed normalized values. Magnetic resonance imaging of the heart performed 2 months later showed no structural abnormalities and no pericardial effusion or pericardial wall thickness changes. There was no clinical evidence of acute perimyocarditis recurrence the following 2 years after discharge.

3. Discussion

There have been a few articles published describing the possible association between pericardial and thyroid disease. Sugar [3] and Cullen et al. [4] reported pericarditis as a complication of thyrotoxicosis. Inami et al. [5] reported a case of acute pericarditis as unique comorbidity of thyrotoxic crisis with Graves' disease. Clarke et al. [6] reported four cases of acute pericarditis accompanied by pericardial effusion associated with Graves' disease, explained by a similar pathogenetic background of inflammation with other complications of Graves' disease. Kortekass [7] and Tsai [8] established acute pericarditis as an aggravation of Graves' disease due to noncompliance with treatment. However, the only case of acute recurrent pericarditis accompanied by Graves' disease ever reported has been published by Koo et al. [9].

The pathophysiology of Graves' disease-associated pericarditis is not known. Although one case was attributed to antithyroid medications, especially propylthiouracil [1], most reported cases have occurred in individuals who were poorly adherent to the treatment of known thyroid disease, or who were previously unknown to be hyperthyroid. Antineutrophil cytoplasmic antibodies and rheumatoid factor antibodies have also been implicated in the development of pericarditis in

previously published reports. The pericardial disease occurs similarly to other complications of Graves' disease, such as pretibial myxedema or ophthalmic myopathy [2]; however, most cases show resolution of pericarditis after initiation of appropriate treatment, in contrast to pretibial myxedema and ophthalmopathy, which generally persist despite treatment. An alternate theory suggests that autoantibodies or viral infections, which have been postulated as causative factors in Graves' disease, may also interact with pericardial receptors in certain individuals, resulting in concurrent acute pericarditis [4].

In this case report, we present a patient with acute recurrent perimyocarditis associated with thyrotoxicosis. To the best of our knowledge, this is the first published case of acute recurrent perimyocarditis due to increased thyroid hormones in the bloodstream, accompanied by a significant increase in cardioselective enzymes, pericardial effusion, and reduction of ejection fraction. Although Kukla et al. [10] described myopericarditis complicated with cardiogenic shock mimicking ACS with ST elevation in a patient with hyperthyroidism and diabetes mellitus, there were no signs of recurrence. Although no tissue diagnosis was done to confirm histological changes in the heart muscle, clinical improvement and normalization of laboratory results after successful management of thyrotoxicosis suggest a possible link between thyrotoxicosis and recurrent perimyocarditis. There was no sign of recurrence during the 2-year follow-up. This is the first case report of recurrent perimyocarditis associated with thyrotoxicosis presented in the University Hospital Split in Croatia. A limitation of this case report is that we did not perform viral serological analyses, and hence, we cannot exclude the autoimmune nature of the disease. Viruses may trigger both Graves' disease and subacute thyroiditis. This is an important issue since the patient had mild thyrotoxicosis that would not have such cardiac repercussions.

Although this conclusion requires further investigation, it can be useful in the differential diagnosis of acute and recurrent perimyocarditis where the etiology is uncertain.

Author contributions

Dino Mirić - Substantial contributions to the conception or design of the work; acquisition, analysis and interpretation

of data for the work, Drafting the work for important intellectual content, Final approval of the version to be published. Ensured that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved.

Duška Glavaš - Substantial contributions to acquisition, analysis and interpretation of data for the work, Drafting the work and revising it critically for important intellectual content, Final approval of the version to be published. Ensured that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved.

Ljubica Juretić Kuščić - Substantial contributions to analysis and interpretation of data for the work, Revising the work critically for important intellectual content, Final approval of the version to be published.

Darija Baković Kramarić - Substantial contributions to analysis and interpretation of data for the work, Revising the work critically it critically for important intellectual content, Final approval of the version to be published.

Ajvor Lukin - Substantial contributions to analysis and interpretation of data for the work, Revising the work critically for important intellectual content, Final approval of the version to be published.

Marisa Klančnik - Substantial contributions to the conception or design of the work, Revising the work critically for important intellectual content, Final approval of the version to be published.

Maja Grgec - Substantial contributions to the conception or design of the work, Final approval of the version to be published.

Petar Ivanišević - Substantial contributions to the conception or design of the work, Final approval of the version to be published.

References

1. Imazio M, Brucato A, Barbieri A, Ferroni F, Maestroni S, Ligabue G, et al. Good prognosis for pericarditis with and without myocardial involvement: Results from a multicenter, prospective cohort study. *Circulation* 2013;128:42-9. <https://doi.org/10.1161/CIRCULATIONAHA.113.001531>.
2. Bahn Chair RS, Burch HB, Cooper DS, Garber JR, Greenlee MC, Klein I, et al. Hyperthyroidism and other causes of thyrotoxicosis: Management guidelines of the American thyroid association and American association of clinical endocrinologists. *Thyroid* 2011;21:593-646. <https://doi.org/10.1089/thy.2010.0417>.
3. Sugar SJ. Pericarditis as a complication of thyrotoxicosis. *Arch Intern Med* 1981;141:1242. <https://doi.org/10.1001/archinte.1981.00340090138042>.
4. Cullen D, Munjal N, Chalal H, Ramgopal S, Tas E, Witchel S, et al. Pericarditis as the presenting feature of Graves' disease in a pediatric patient. *Pediatr Emerg Care* 2017;33:268-70. <https://doi.org/10.1097/PEC.0000000000000439>.
5. Inami T, Seino Y, Goda H, Okazaki H, Shirakabe A, Yamamoto M, et al. Acute pericarditis: Unique comorbidity of thyrotoxic crisis with graves' disease. *Int J Cardiol* 2014;171:e129-30. <https://doi.org/10.1016/j.ijcard.2013.12.042>.
6. Clarke NR, Banning AP, Gwilt DJ, Scott AR. Pericardial disease associated with Grave's thyrotoxicosis. *QJM* 2002;95:188-89. <https://doi.org/10.1093/qjmed/95.3.188>.
7. Kortekaas KA, van der Lienden B, Jong S, Riezebos R. Graves' disease as an uncommon cause of acute pericarditis. *BMJ Case Rep* 2014;2014.
8. Tsai MS, Yang CW, Chi CL, Hsieh CC, Chen WJ, Huang CH, et al. Acute pericarditis: A rare complication of graves' thyrotoxicosis? *Am J Emerg Med* 2006;24:374-5. <https://doi.org/10.1016/j.ajem.2005.10.020>.
9. Koo EH, Kim SM, Park SM, Park JW, Kim EK, Lee GY, et al. Acute recurrent pericarditis accompanied by graves' disease. *Korean Circ J* 2012;42:419-22. <https://doi.org/10.4070/kcj.2012.42.6.419>.
10. Kukla P, Bryniarski L, Bromblik A, Szczuka K, Kawecka-Jaszcz K. Myopericarditis complicated with cardiogenic shock mimicking acute coronary syndrome with ST elevation in a patient with hyperthyroidism and diabetes mellitus. *Kardiol Pol* 2008;66:982-6.