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Case Report

When hyperthyroidism goes unchecked: A rare case of cardiac tamponade induced by acute pericarditis in Graves' disease ☆,☆☆

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ABSTRACT

Graves' disease, the most prevalent cause of hyperthyroidism, is an autoimmune disorder associated with several cardiovascular complications. The occurrence of acute pericarditis within the context of Graves' disease, is very rare. We present an uncommon case of cardiac tamponade in a patient diagnosed with Graves' disease. A female patient, was admitted with symptoms of dyspnea and palpitations. Clinical examination revealed Beck's triad. Transthoracic echocardiography showed a large pericardial effusion with significative respiratory variations, indicating an urgent pericardiocentesis. Thyroid function tests revealed hyperthyroidism with positive TSH-receptor antibodies, and elevated anti-TPO antibodies. Cardiac tamponade, induced by acute pericarditis in Graves' disease is an uncommon entity. Typically, the diagnosis is established on a detailed history and physical examination, supported by specific investigations, including suppressed levels of TSH along with elevated free T4 levels and thyroid receptor antibodies. Pericardiocentesis, NSAIDs, betablockers, associated with antithyroid medications constitute the mainstay of treatment.

Introduction

Acute pericarditis, a condition often seen in clinical practice, is characterized as an inflammation of the pericardium. Its main causes are typically infections, malignant tumors, myocardial infarction, and autoimmune diseases. It may lead to cardiac tamponade, a critical and potentially life-threatening situation [1].

Graves' disease, defined as an autoimmune disorder that leads to excessive thyroid gland activity, is associated with several cardiovascular complications. These include arrhythmia, hypertension, mitral valve prolapse and tachycardia-associated cardiomyopathy. Considering the autoimmune na-

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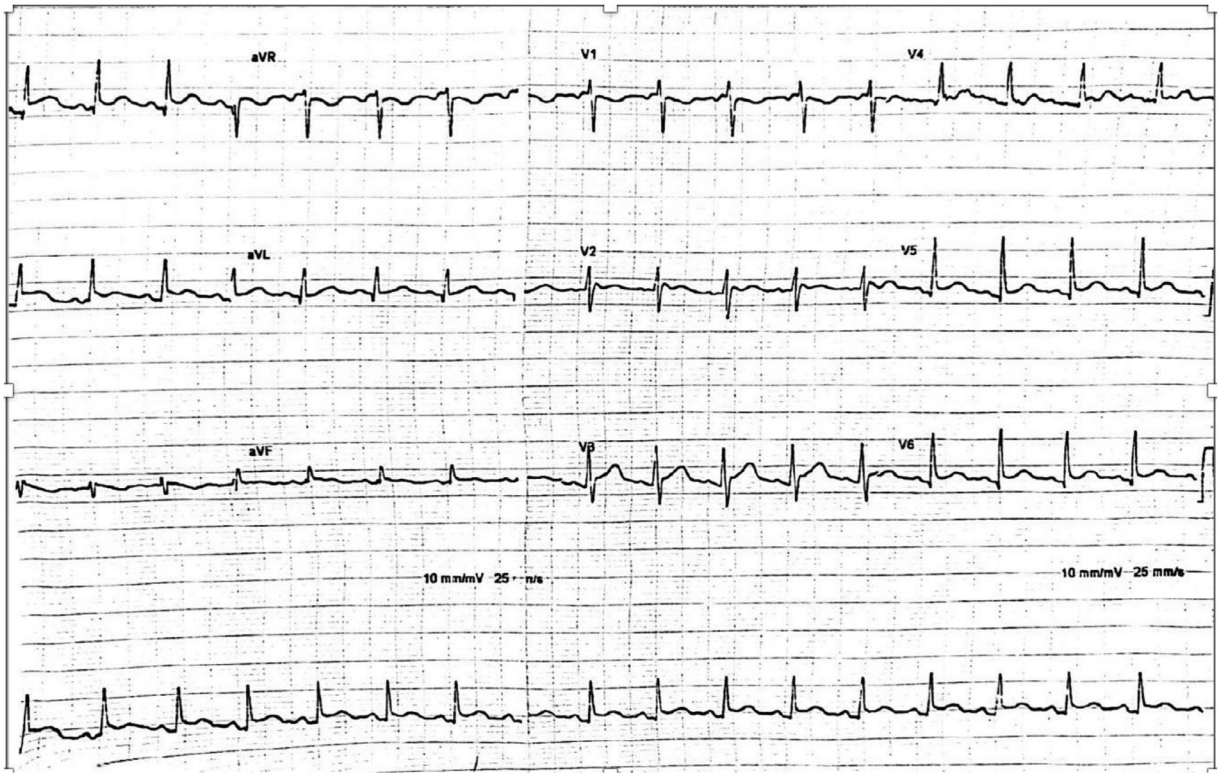

Fig. 1 – Electrocardiogram showing a sinus tachycardia with a heart rate of 120 beats per minute.

ture of Graves' disease, autoimmune pericarditis may also emerge as a possible cardiovascular complication of the condition. Nevertheless, the association between acute pericarditis and Graves' disease, as the most common form of thyrotoxicosis, is very rare, with a few documented cases [2].

In this article, we report a rare case of cardiac tamponade complicating acute pericarditis in a female patient diagnosed with Graves' disease.

Case presentation

A 50-year-old female was admitted to our hospital's emergency department, presenting with a 2-day history of experiencing difficulty breathing at rest accompanied by palpitations. This was preceded by an acute episode of chest pain, that radiated to the trapezius ridge and left arm, and increased by position changes and deep breathing.

One month previously, the patient was diagnosed with hyperthyroidism with suppressed thyroid-stimulating hormone (0.003 mUI/L, normal values 0.340-3.330 mUI/L), and elevetad free thyroxine (3.15 ng/dL, normal values 0.61-1.12 ng/dL), following a paroxysmal episode of atrial fibrillation (CHADSVASC score of 0) and was prescribed Carbimazole 60 mg/jour. She was not taking any other medications, and she did not use any herbal medicines. Her family history was essentially unremarkable.

She also reported having a fever, generalized tiredness, myalgia, and arthralgia, which had started 15 days before admission. The patient complained of insomnia, and a significant weight loss of 23 kg over the past 7 months. Additionally, she mentioned symptoms of heat intolerance, hand tremors, and anxiety. However, there was no history of prior chest pain, or sore throat, or rigors, or sputum, nor skin rash, or other systemic signs. Furthermore, the patient denied any chest trauma.

Upon general examination, the patient appeared anxious. She presented with a fever, having a temperature of 38°C, with a blood pressure of 87/55 mmHg and a heart rate of 125 beats per minute. She was tachypneic with a respiratory rate of 24 breaths per minutes and maintained an oxygen saturation of 98% on room air.

The cardiovascular examination showed tachycardia along with muffled heart sounds. No murmurs, or gallops or rubs were detected. Central jugular pressure was elevated, and there was no pulsus paradoxus. On pulmonary auscultation, the lung fields were clear bilaterally, with no wheezing, rhonchi, or crackles detected. No enlargement of the thyroid gland was observed. There was no pain in the neck or vein engorgement, nor any signs of ophtalmopathy. The remainder of systematic examination was normal.

12-lead electrocardiogram (ECG) (Fig. 1) showed sinus tachycardia at 120 beats per minute. Chest radiography (Fig. 2) revealed an enlarged cardiac silhouette.

A transthoracic echocardiography (Fig. 3) was performed, revealing a large pericardial effusion. Significant respiratory variation in mitral and aortic valve velocities was noted (Fig. 4). The systolic function of the left ventricle was preserved, and the dimensions and function of the right ventricle were normal. No valvular diseases were identified.

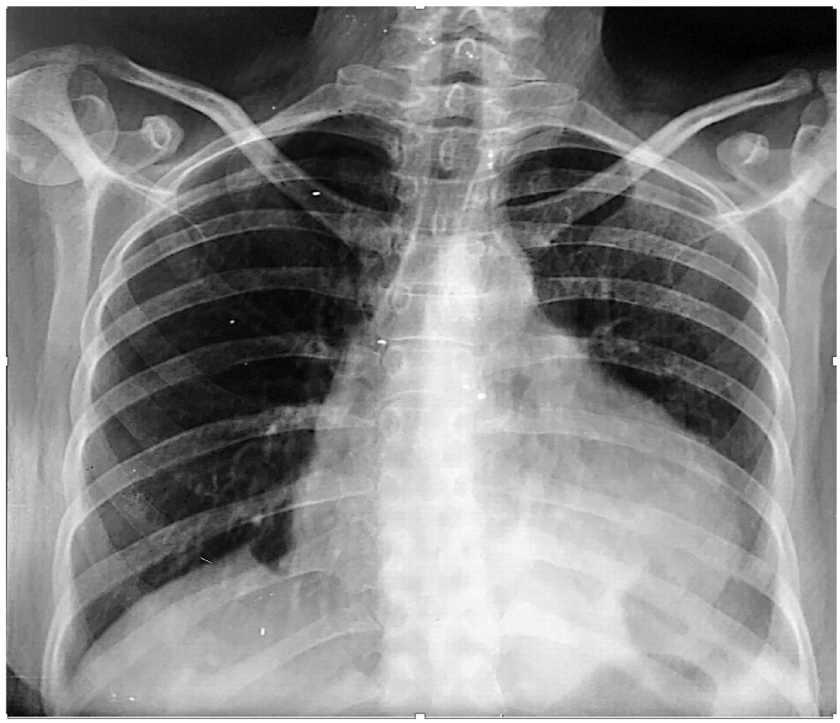

Fig. 2 – Chest X ray revealing cardiomegaly.

An emergency pericardiocentesis was performed, resulting in the drainage of 450 mL of citrine-yellow fluid, leading to prompt hemodynamic recovery. Analysis of the pericardial fluid showed 300 leukocytes/mm^3, with 50% neutrophils and 50% lymphocytes. The protein concentration was 50 g/L, and cultures were negative.

The laboratory analysis revealed a normal blood cell count but an increased C-reactive protein (CRP) level of 110 mg/L (normal range < 5 mg/L). Troponin levels were within normal limits, ruling out any associated myocarditis. Renal function was unaffected, excluding uremic pericarditis, and there were no notable abnormalities in the metabolic panel results.

However, thyroid function blood tests showed a suppressed thyroid-stimulating hormone (TSH) level of 0.01 mUI/L (normal reference range 0.35-4.94 mUI/L), with a normal free thyroxine (T4) level of 12.82 pmol/L (normal reference range 9.01-19.05 pmol/L). Strongly positive TSH-receptor antibodies were detected at 33.42 UI/L (normal range <2.58 UI/L), and anti-thyroperoxidase antibodies were elevated at 169.67 UI/mL (normal range <5.61 UI/mL). Thyroid ultrasound with Doppler showed increased vascularity in a normal-sized gland (Fig. 5).

The viral serology tests, specifically for HIV and hepatitis C, were negative. The polymerase chain reaction (PCR) for SARS-CoV-2 RNA also returned a negative result. No bacterial growth was detected in the blood cultures. A sputum culture for Mycobacterium tuberculosis was conducted, with negative results. Similarly, the GeneXpert MTB/RIF test, the IGRA, and tuberculin skin tests were all negative. The laboratory tests conducted included an antinuclear antibody (ANA) panel, anti-actin smooth muscle antibodies, antimitochondrial antibodies, and anti-Scl-70 antibodies, all of which returned negative results. A cerebro-thoraco-abdominal CT scan was performed, and the results showed no significant abnormalities.

Based on the patient's medical background and diagnostic tests, the diagnosis of cardiac tamponade as a complication of acute pericarditis in the context of Graves' disease was made.

The patient was prescribed a daily treatment for pericarditis, including Aspirin at 3g, colchicine at 0.5 mg, and omeprazole at 20 mg. In addition, Propranolol at 20 mg once daily was initiated to manage tachycardia, along with a lower dose of Carbimazole at 20 mg per day.

The patient experienced significant improvement in symptoms and was discharged from the hospital after a few days.

The patient was asymptomatic at the latest follow-up. Her thyroid function tests indicated biochemical euthyroid status, with free T4 level at 14.24 pmol/L (normal range 9.01-19.05). Additionally, her C-reactive protein levels were normal. A transthoracic echocardiography performed during the follow-up showed the absence of pericardial effusion.

Discussion

Graves' disease is characterized as an autoimmune thyroid disorder which stimulate the overproduction of thyroid hormones, via the thyroid antibodies receptor, resulting in thyrotoxicosis [3]. The typical Graves' cardiac manifestations are hypertension, tachycardia-induced cardiomyopathy, tachyarrhythmias (such as atrial fibrillation, atrial flutter and supra ventricular tachycardia), and heart failure [4].

Defined as an inflammation of the pericardium, acute pericarditis may occur with or without a pericardial effusion. This

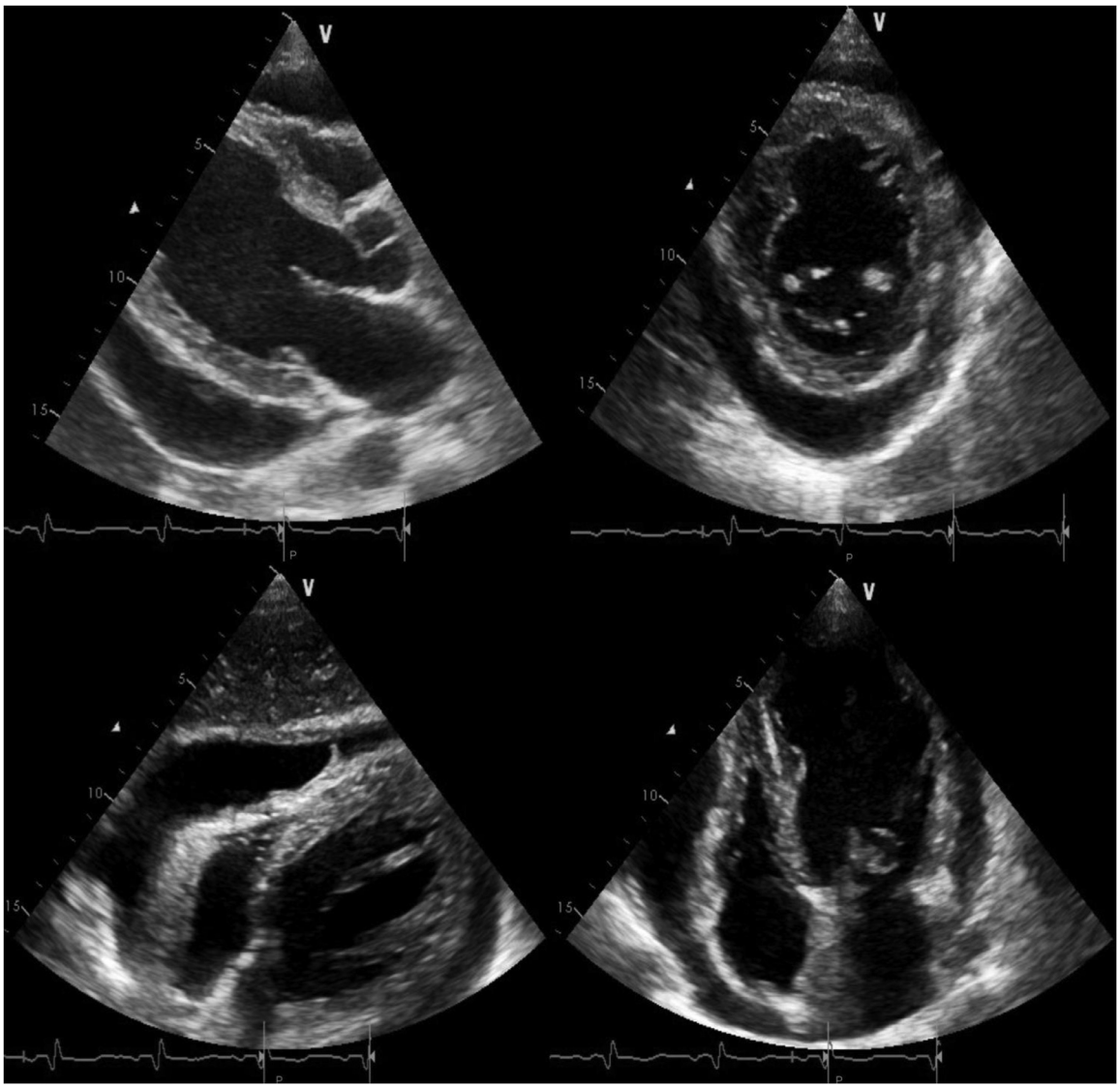

Fig. 3 – Transthoracic echocardiography revealing a large pericardial effusion.

condition can progress to cardiac tamponade, a critical situation, in which the heart is compressed due to excessive fluid buildup in the pericardium, posing a life-threatening risk. Diagnosis of acute pericarditis typically relies on the presence of at least 2 of the following criteria: (a) chest pain relieved by leaning forward and sitting upright; (b) pericardial rub; (c) electrocardiogram modifications like PR depression or widespread ST elevation; and (d) evidence of pericardial effusion via ultrasound or other imaging techniques. It is mainly caused by infections, myocardial infarction, malignant tumors, and autoimmune disorders [1].

Although acute pericarditis commonly occurs as a complication of hypothyroidism, its occurrence in association with thyrotoxicosis is less common and generally considered rare [5]. Hyperthyroidism can result on a variety of pericardial disease, including pericarditis with or without effusion, myopericarditis and cardiac tamponade [6,7]. The occurrence of cardiac tamponade, as a medical emergency, is more uncommon in this context. Cardiac tamponade as a complication of acute pericarditis in Graves' disease has been documented in only a few cases [3].

The first cases of hyperthyroidism associated with acute pericarditis were reported by Treusch and Jaffe in 1958. However, the link between the 2 conditions was not identified [8]. From 1958 to 2022, only 10 additional cases have been noted in the English literature. Most of these cases concern patients diagnosed with acute pericarditis alongside Graves' disease. Cardiac tamponade was described in only 6 case reports [9].

The pathophysiology of pericarditis associated with Graves' disease remains unclear [10,11]. Determining whether these 2 conditions are coincidental or have an underlying etiologic association is challenging. Various mechanisms have been suggested to elucidate their causal relationship [12]. The majority of previous reports of acute pericarditis associated with Graves' disease, indicating an autoimmune process in the pathogenesis of pericardial inflammation.

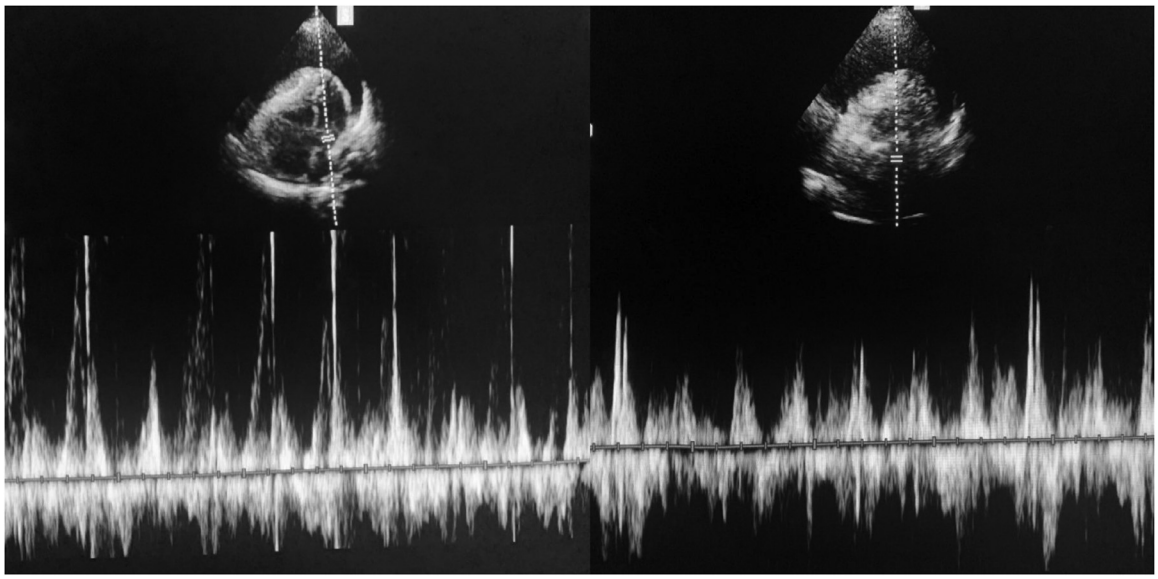

Fig. 4 – Transthoracic echocardiography showing significant respiratory variation in mitral and aortic valve velocities.

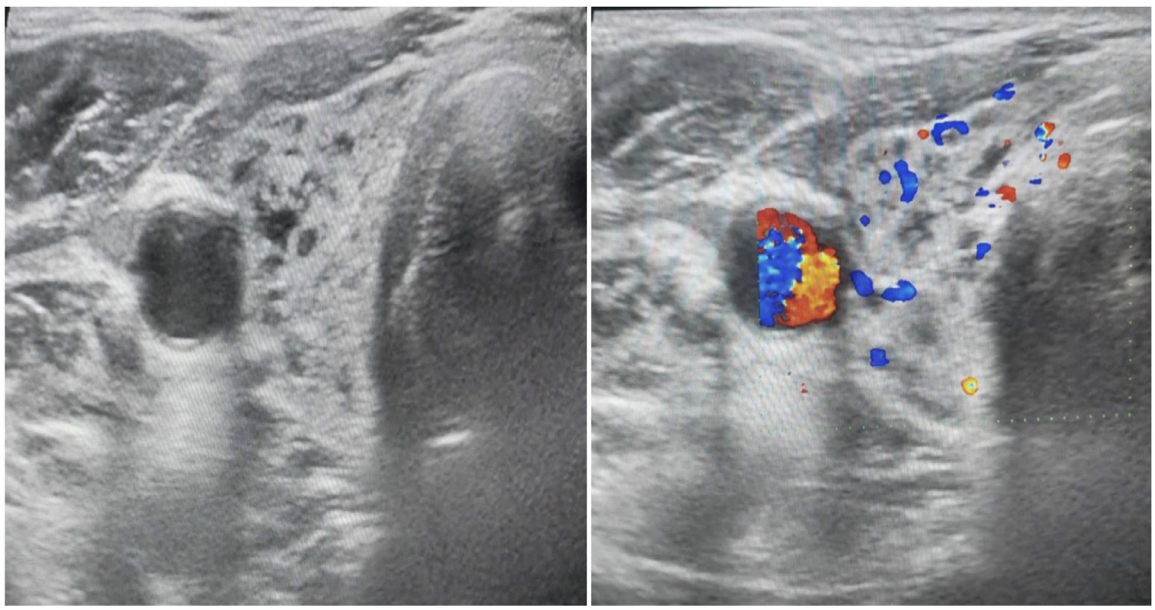

Fig. 5 – Thyroid ultrasound with Doppler showing increased vascularity in a normal-sized gland.

Clarke et al. proposed that the etiology of pericarditis in Graves' disease might be autoimmune, similar to dermopathy and ophthalmomyopathy [11]. Another theory proposes that Graves' disease and acute pericarditis are frequently associated with viral infections, particularly the Epstein Barr virus [12]. Indeed, there is a complex relationship between autoantibodies and systemic viral infections, which is implicated in the pathogenesis of Graves' disease and acute pericarditis. One conceivable explanation is that in Graves' disease, autoantibodies or viral infections could interact with specific receptors on the pericardium, potentially inducing inflammation in the pericardial tissue [13]. An alternate explanation indicates that thyroid hormones may impact myocardial fat metabolism [14]. In certain clinical scenarios, cases of drug-induced pericarditis have been observed following the initiation of anti-thyroid medications such as propylthiouracil. Other anti-thyroid drugs like iodine and carbimazole have also been implicated [15].

Patients with hyperthyroidism-induced pericarditis typically display symptoms compatible with both acute pericarditis and hyperthyroidism [16]. Pleuritic chest pain is a major sign of acute pericarditis, the diagnosis of which relies on well-defined criteria. The Beck's triad, including arterial hypotension, muffled heart sounds, and elevated jugular venous pressure, is commonly observed in patients presenting with cardiac tamponade. Graves' disease is generally di-

agnosed through a combination of clinical features indicating hyperthyroidism, such as weight loss, palpitations, anxiety and exophthalmia, along with laboratory finding including decreased TSH levels, and elevated concentrations of thyroid hormone concentrations, TSH receptor antibodies, and thyroid-stimulating immunoglobulins [17]. In addition, a characteristic pattern of an enlarged gland with elevated vascularity on Doppler imaging is frequently seen with thyroid ultrasonography [18]. Radioactive iodine uptake tests are essential diagnostic tools used to evaluate thyroid function and identify various thyroid pathologies, including Graves' disease, by demonstrating the uptake patterns of the radioactive tracer in the thyroid gland. However, their use may be limited due to factors such as restricted availability, high cost, and exposure to ionizing radiation. Consequently, these scans may be reserved for cases where the diagnosis is uncertain [13].

At present, there are no evidence-based recommendations for managing patients diagnosed with acute pericarditis as a manifestation of Graves' disease [7]. Similar treatment approaches have been adhered in the treatment of both acute pericarditis and hyperthyroidism across all published cases in the literature. As per the European Thyroid Association guidelines [19], the management of thyrotoxicosis in the setting of Graves' disease primarily involves the application of antithyroid drugs like Carbimazole or Propylthiouracil. Beta blockers are also administered to alleviate the adrenergic symptoms associated with the hyperthyroid state. Additional therapeutic interventions, including radioactive iodine therapy or surgery, should be evaluated depending on the individual's unique clinical presentation [7]. The primary strategy, according to the guidelines provided by the European Society of Cardiology for managing pericarditis, involves limiting physical activity and using colchicine alongside either Aspirin or nonsteroidal anti-inflammatory drugs (NSAIDs). Corticosteroids are recommended as second-line agents for individuals who do not respond adequately to initial treatments, or for those who have contraindications. When acute pericarditis is complicated by cardiac tamponade, pericardiocentesis should be performed. In the absence of clear standards, a combination of the therapeutic alternatives mentioned above remains a practical approach for managing patients with acute pericarditis induced by Graves' disease. Nevertheless, it's crucial to consider individual patient factors, customize management accordingly, and ensure the effective treatment of underlying hyperthyroidism, to successfully resole pericarditis [20].

Conclusion

Acute pericarditis complicated by cardiac tamponade is an uncommon manifestation of Graves' disease. Various mechanisms have been suggested to explain this association. Nevertheless, it remains challenging to determine whether these 2 conditions are causally related or occur coincidentally. Thorough history taking and physical examination are crucial to consider Graves' disease in the setting of acute pericarditis. Management should involve pericardiocentesis along with concurrent treatment of pericarditis and thyrotoxicosis.

Patient consent

Written informed consent was obtained from the patient for publication of this case report and accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal on request.

REFERENCES

[1] Adler Y, Charron P, Imazio M, Badano L, Barón-Esquivias G, et al. 2015 ESC Guidelines for the diagnosis and management of pericardial diseases: the Task Force for the Diagnosis and Management of Pericardial Diseases of the European Society of Cardiology (ESC) endorsed by: the European Association for Cardio-Thoracic Surgery (EACTS). Eur. Heart J. 2015;36:2921–64.
[2] Tsai MS, Yang CW, Chi CL, Hsieh CC, Chen WJ, Huang CH. Acute pericarditis: a rare complication of Graves' thyrotoxicosis? Am. J. Emerg. Med. 2006;24:374–5.
[3] Fonseca EM, Schonhofen I, Toralles MP, de Carvalho JF. Graves' disease inducing a massive cardiac tamponade. BMJ Case Rep 2021;14:e239772.
[4] Al-Futaisi A, Al-Aamri M, Bahowairath F, Al Jufaili MS. Acute pericarditis as an uncommon presentation of graves' disease. Oman Med. J. 2022;37:e359.
[5] Fabowale MO, Ogah OS, Kehinde AM, Olusola FI, Okafor IJ, Bakare TA, et al. Pericardial effusion in a patient with hyperthyroidism: a case report. Ann. Ib. Postgrad. Med. 2021;19:78–81.
[6] Tourniaire J, Sassolas G, Touboul P, Lejeune H, Berger M. [Tamponade caused by subacute pericarditis in Basedow's disease]. Presse Medicale Paris Fr. 1983 1983;12:1989–90.
[7] Thomson RJ, Rossberg N, Davar J, Whelan C. Myopericarditis and thyroiditis: a case report. Eur. Heart J. Case Rep. 2021;5:ytab192.
[8] Treusch JV, Jaffe HL. Hyperthyroidism associated with presumptive acute pericarditis; a report of three cases. Calif. Med. 1958;89:217–21.
[9] Chahine J, Jedeon Z, Chang KY, Jellis CL. Pericardial manifestations of thyroid diseases. Curr. Cardiol. Rep. 2022;24:893–904.
[10] Sugar SJ. Pericarditis as a complication of thyrotoxicosis. Arch. Intern. Med. 1981;141:1242.
[11] Clarke NRA, Banning AP, Gwilt DJ, Scott AR. Pericardial disease associated with Grave's thyrotoxicosis. QJM Mon. J. Assoc. Physicians 2002;95:188–9.
[12] 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.
[13] Gondal M, Hussain A, Yousuf H, Haider Z. Double trouble – thyro-pericarditis: rare presentation of Graves' disease as pericarditis—a case report. Eur. Heart J. Case Rep. 2020;4:1–5.
[14] Chhabra L, Khalid N, Spodick DH. [Letter on the article 'Pericardial effusion: an unknown expression of Graves' disease']. Presse Medicale Paris Fr. 1983 2014;43:1407.
[15] Airel PS, Steele MB, Lin AH, Seidensticker DF, Shwayhat AF. Pericarditis, thymic hyperplasia, and graves' thyrotoxicosis: case report and review of the literature. Mil. Med 2013;178:e865–9.
[16] Chiabrando JG, Bonaventura A, Vecchié A, Wohlford GF, Mauro AG, Jordan JH, et al. Management of acute and recurrent pericarditis: JACC State-of-the-Art review. J. Am. Coll. Cardiol. 2020;75:76–92.

[17] Cullen D, Munjal N, Chalal H, Ramgopal S, Tas E, Witchel S. Pericarditis as the presenting feature of graves disease in a pediatric patient. Pediatr. Emerg. Care 2017;33:268–70.
[18] Hari Kumar KV, Pasupuleti V, Jayaraman M, Abhyuday V, Rayudu BR, Modi KD. Role of thyroid Doppler in differential diagnosis of thyrotoxicosis. Endocr. Pract. Off. J. Am. Coll. Endocrinol. Am. Assoc. Clin. Endocrinol. 2009;15:6–9.
[19] Kahaly GJ, Bartalena L, Hegedüs L, Leenhardt L, Poppe K, Pearce SH. 2018 European Thyroid Association Guideline for the management of graves' hyperthyroidism. Eur. Thyroid J. 2018;7:167–86.
[20] Schwier NC, O'Neal K. Pharmacotherapeutic management strategies for thyroid disease-induced pericarditis. Ann. Pharmacother. 2020;54:486–95.