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CASE REPORT

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Unexpected Grave's-induced acute myocardial infarction in a young female, a literature review based on a case report

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Abstract

Introduction Myocardial ischemia can occur due to several causes, which result in an imbalance between the supply and demand of oxygen to cardiac muscles. One potential reason for this condition is the overwork of the heart due to hyperstimulated thyroid function.

Case presentation The patient was a 36-year-old woman who presented with left-sided chest pain, dyspnea, palpitation, and tremor. The initial evaluation showed evidence of myocardial ischemia (positive high-sensitivity troponin) caused by a hyperactive thyroid gland. The treatment for myocardial infarction, along with anti-thyroid medications, improved the patient's condition and subsided the symptoms. The coronary angiography revealed no pathologic finding, and the hypokinetic left ventricle, observed in the first echocardiogram, was resolved. The patient was discharged with an excellent clinical condition, and after the 4-month taking of a calcium channel blocker and tapering carbimazole, the thyroid function became normal, and her symptoms resolved completely.

Conclusion Patients without evident risk factors for ischemic heart disease, such as non-diabetic, nonsmoker, and young individuals who presented with acute coronary syndrome, should be evaluated for a potential background reason for the imbalance between the oxygen demand and supply of the myocardium. The presence of palpitation, weight loss, tremors, insomnia, and anxiousness, along with ischemic signs, should make the physician think about the probability of the hyperthyroid-induced cardiovascular disorder.

Clinical key point The initial presentation of hyperthyroidism might be accompanied by severe cardiac symptoms. When the demographic features are not aligned with usual ischemic heart disease, other probable symptoms and signs should be investigated, and thyroid function should be checked. The control of thyroid hyperactivity would result in the resolution of both cardiac and non-cardiac symptoms.

Keywords Myocardial ischemia, Hyperthyroidism, Acute coronary syndrome, Interventional cardiology, Case report

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Introduction

Type II myocardial infarction (MI) is defined by the activation of imbalance triggered by secondary causes, including accelerated metabolism and demand for more blood circulation due to sepsis, drugs, hypoxemia, and hyperthyroidism [1]. The increased demand for oxygen is caused by increased metabolism in myocardial tissue, which works faster and stronger in response to the overstimulation of thyroid gland products. This effect is caused by the enhancement of mitochondrial production, increased activity of intrinsic enzymes, and the development of intracellular proteins [2]. These all can lead to imbalanced myocardial bioenergetic condition and the consequent ischemia [3]. Moreover, overwork of the thyroid gland can cause coronary vasospasm, which is a predisposing factor for coronary thrombosis secondary to and stasis of blood [4]. Another potential declared etiologies is the probability of thrombotic status activation by thyroid hormones [4]. This type of cardiovascular disease is also categorized as myocardial infarction due to non-obstructive coronary artery (MINOCA) [5].

It has been shown that the risk of cardiovascular diseases, including MI, has been considerably higher in patients with hyperthyroidism, regardless of their gender and age [6]. Interestingly, the initial presentation in 6% of patients with hyperthyroidism was myocarditis and even more than with other cardiovascular presentations such as arrhythmia, congestive heart failure (CHF), and dilated cardiomyopathy [7]. These patients prevalently experience noncardiac symptoms, including weight loss, insomnia, anxiousness, and tremors [8]. The severity of symptoms and the effect on the myocardial tissue varies between patients, ranging from intermittent chest pains without any significant injury to the cardiac muscle to a severely destroyed myocardium by ischemic attack [9, 10]. It is crucial to differentiate the cause of these ischemic attacks from the usual obstructive ones, considering the completely different management they require [11].

In this study, we present a case of undiagnosed and mismanaged thyrotoxicosis-induced myocardial infarction, presented with chest pain, palpitation, and dyspnea.

Case presentation

A 36-year-old woman with no prior medical illness presented to the Emergency Department (ED) with a sudden onset of severe left-sided chest pain that started 30 min before her arrival at the ED, present at rest and worsened by exertion, associated with palpitations, diaphoresis, and dyspnea. Over the preceding three months, she had palpitations and tremors and lost 4 kg of weight. However, the condition was misdiagnosed as general anxiety disorder, and he was referred for psychological treatment, which was ineffective. She denied the use of illicit drugs. She had never been a smoker and worked in an office with minimal physical activity. There was no remarkable point in her family history except for diabetes mellitus (DM II) in her mother. She had a history of bariatric surgery due to obesity. On general appearance, she was anxious, diaphoretic, and shaking in the upper and lower extremities. In the vital signs evaluation, a blood pressure of 158/96 mmHg, respiratory rate of 25 per minute, heart rate of 122 beats per minute, and temperature of 36.8. Cardiovascular and respiratory system examinations were otherwise unremarkable. She had exophthalmos and a diffuse goiter measuring 9×6 cm, without any bruit. No other remarkable finding was detected during the patient's physical exam.

Immediately after the initial presentation and with the provisional diagnosis of Hyperthyroidism-Induced acute coronary syndrome, an electrocardiogram (ECG) was obtained, which showed sinus tachycardia without any significant ST-segment change in any of the precordial and limb leads (Fig. 1) The patient was monitored with cardiopulmonary monitoring. Lab data were requested after taking the blood sample. The patient was given oxygen via nasal canola 4-6lit/min, Metoprolol (50 mg), Aspirin (325 mg), Clopidogrel (300 mg), Atorvastatin (80 mg) orally, and Nitroglycerin (intravenous, 5 µg/min), losartan (25 mg twice a day), and UFH (5000IU stat, and 1000IU/hour). The patient's condition improved significantly after these interventions. The patient's blood results came back, demonstrating positive high-sensitivity cardiac Troponin I (hs-cTnI) with 98.5 (pg/ml), very high T3, T4, and considerably below the range of TSH (Table 1) A transthoracic echocardiogram (TTE) showed a hypokinetic anteroseptal wall with an ejection fraction of 35%, no valvular pathology, normal chamber sizes, and no remarkable abnormality in ventricular dimensions without any evidence of left ventricular hypertrophy (Table 2). She was treated for non-ST elevation myocardial infarction and hyperthyroidism. The serial ECG was done every 30 min, and it showed non-specific ST-segment changes in limb and precordial leads, which were detectable on the 4th ECG (Fig. 2) The changes were suggestive of probable ischemic changes in the myocardium.

The neck (thyroid and lymph nodes) ultrasound imaging showed a diffusely enlarged thyroid, increased vascularity, and diffused hyperactivity (Table 3) The coronary angiography (CA) 48 h after the patient's presentation revealed no abnormal findings (Fig. 3).

Due to the unavailability of the anti-TSH receptor antibody in the hospital's laboratory, it could not be checked. However, these antibodies were evaluated, and the thyroid radionuclide scan was performed after discharge, which was compatible with the diagnosis of Grave's disease. The laboratory workup showed that the patient had decreased thyroglobulin (Tg), 0.10 µg/L, with positive thyroglobulin antibody (Tg-Ab), positive thyroid

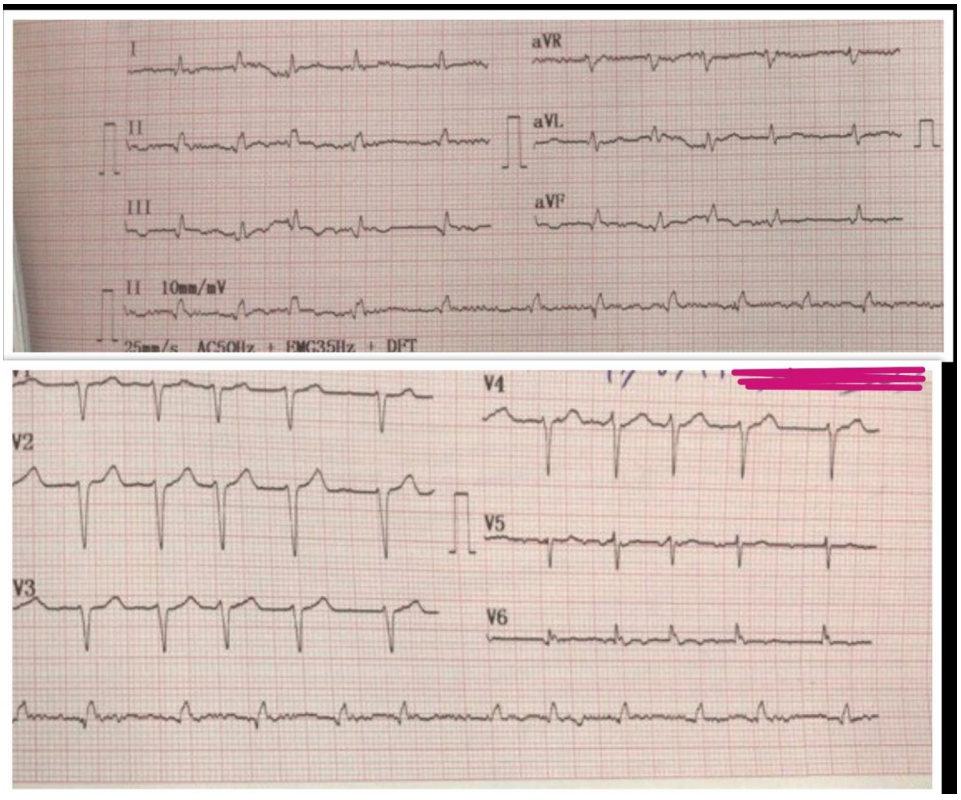

Fig. 1 The initial electrocardiogram showed sinus tachycardia without any other remarkable finding

peroxidase antibody (TPO-Ab), and positive thyroid-stimulating hormone (TSH) receptor antibody (TR-Ab). The thyroid radionuclide scan was performed, which showed disseminated hyperactivity of the thyroid, which was compatible with the diagnosis of Grave's disease. The tablet carbimazole 30 mg daily, which was started

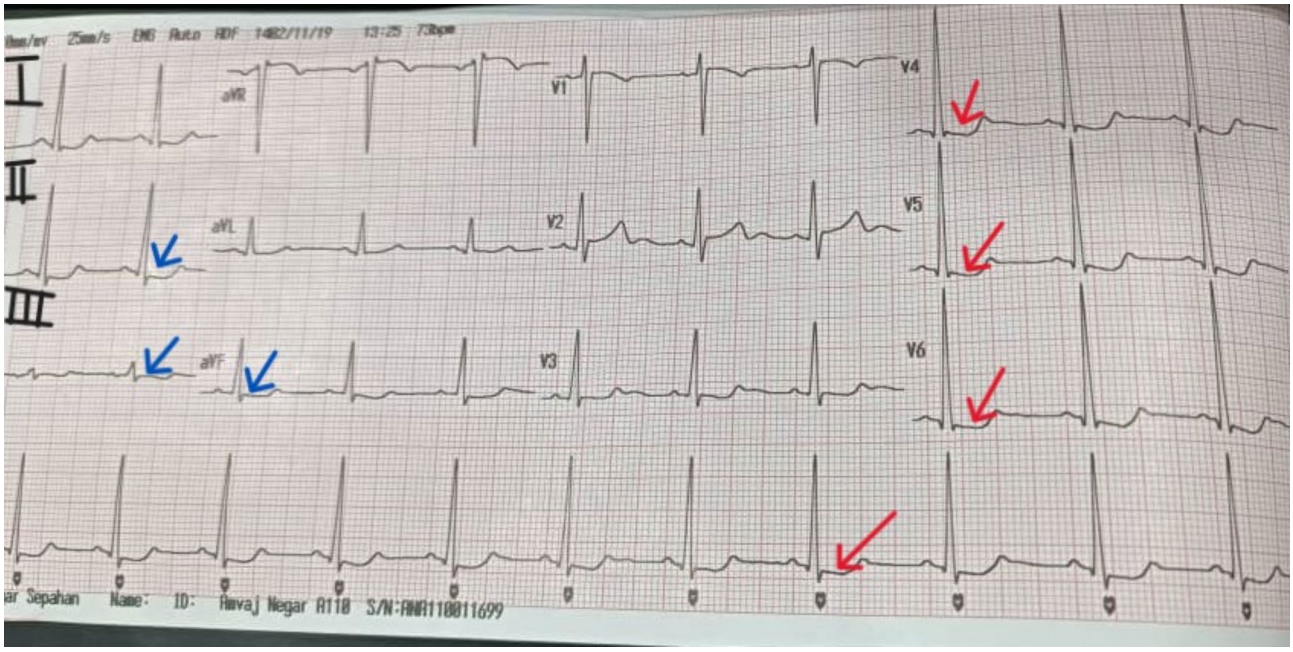

Fig. 2 The 4th electrocardiogram, performed 90 min after the initial electrocardiogram, showed dynamic changes in limbs (blue arrows) and precordial (red arrows) leads

Table 1 Index laboratory data

Test	Result	Reference Range
Complete Blood Count		
RBC (10^6/µl)	5.51	4.2–5.5
Hemoglobin (gr/dL)	14.0	12–16
WBC (per µl)	5200	4.000–11.000
MCV (fL)	75.7	80–99
Hematocrit (%)	41.7	37–47
Platelet (per µl)	230.000	150.000-400.000
Neutrophils (%)	30.8%	40–75%
Lymphocytes (%)	58.7%	20–45%
Monocyte (%)	9.9%	2–8%
Eosinophils (%)	0.5%	0–6%
BMP and Cardiac Biomarkers		
Fasting Blood Glucose (mg/dl)	88	Up to 126
Cholesterol	151	Up to 200
K^+ (meq/lit)	4.0	3.5–5.3
Na^+ (meq/lit)	144	135–145
Ca^+ (mg/dl)	10.2	8–11
Creatinine (mg/dl)	0.5	0.6–1.4
Phosphorus (mg/dl)	4.1	2–5
BUN (mg/dl)	11	8.22
Hb A1C %Hb	5.4	3.5–5.9
PT	15.3s	11–13 s
PTT	31s	25–38 s
INR	1.31	1-1.5
CK-MB	29 u/l	< 25u/l
hs-cTnI (pg/ml)	98.5	<16
T4 Total (ug/dl)	Over 24.86	5.1–14.1
T4 Total (ng/ml)	6.51	0.7-2
TSH (ulU/ml)	0.005	0.3–4.2
DHEAS (ug/dl)	325.7	25.5-460.2
ESR (Millimeters per hour)	45	0–20
CRP (qualitative)	Negative	3+
Cortisol	5.07	6.2–20

Urine Analysis: No remarkable pathologic finding was detected except 1 + glucosuria.

Abbreviations: WBC: white blood cells, RBC: Red blood cell count, MCV: Mean corpuscular volume, BUN: Blood urea nitrogen, PT: prothrombin time, INR: International Normalized ratio, CK: Creatinine kinase, TSH: Thyroid stimulating hormone, DHEAS: Dehydroepiandrosterone sulfate, ESR: Erythrocyte Sedimentation Rate, CRP: C-Reactive Protein, hs-cTnI: High-Sensitivity Cardiac Troponin I

after stabilizing the patient's condition in the hospital, was gradually tapered over six months, and verapamil 80 mg was continued despite the discontinuation of all other medications. Considering the differential diagnosis of myocarditis, an echocardiogram and cardiac magnetic resonance imaging (CMRI) were performed, which showed no sign of myocarditis. During the follow-up sessions 2, 4, and 8 weeks later, she had no new complaints and remarked on good activity tolerance. The thyroid function was normal at the 4th follow-up visit four months after the admission. Repeat ECG (Fig. 4) and TTE showed no remarkable abnormality. Normal chambers, valvular function, and an ejection fraction of 50% were detected on the TTE. The detected hypokinetic area in the initial ER echocardiogram was not observed in the follow-up TTE.

Table 2 The patient's echocardiogram showed evidence of hypokinetic myocardium

Variable	Result
Chambers sizes and ventricular function	Normal size (LVEDD:45 mm), LV systolic dysfunction (LVEF:35%), global hypokinesia, Hypokinesia in the apical aspect of the LV, RVE, and RV systolic dysfunction were noted.
Valves	Trileaflet AV, No AS, No AI, Normal ascending aorta and root, No COA, No MS, Moderate MR, No TR (within normal range TVG, PAP), No TS
IVC size	Top normal of normal size IVC and respiratory collapse < 50%
Pericardial Effusion	No PE, No compressive sign
Recommendation	According to the findings, Myocardial Ischemia should be ruled out, and CAG is recommended.

LVEDD: Left ventricle end-diastolic diameter, LVEF: Left ventricle ejection fraction, GLS: Global longitudinal strain, LVH: Left ventricle hypertrophy, LVSD: Left ventricle systolic diameter, PWP: Pulmonary wedge pressure, E/A: E-wave on A-wave ratio, LAE: Left atrial enlargement, LAVI: Left-atrial volume indexed, RAE: Right atrium enlargement, RVE: Right ventricle enlargement, TAPSE: Tricuspid annular plane systolic excursion, FAC: Fractional area change, AV: Aortic valve, AS: Aortic stenosis, AI: Aortic insufficiency, COA: Coarctation of Aorta, MS: Mitral stenosis, MR: Mitral regurgitation, TVG: Tricuspid valve gradient, PAP: Pulmonary Atrial Pressure, TS: Tricuspid stenosis, IVC: Inferior vena cava.

Discussion

In this case report, we presented a patient with undiagnosed graves, presented with cardiovascular symptoms and NSTEMI. Although the prevalence of thyrotoxicosis-induced MI is less than 2%, it has substantial importance considering the cruciality of differentiation from obstructive ischemia [9, 10]. The broader term for such cardiovascular disorders is MINOCA, defined by 1) myocardial infarction approved by documents, 2) approval of

Table 3 Thyroid ultrasound showed the diffuse hyperactivity without any nodule

Section	Size	Volume
Right Lobe	24*26*55	17.5 cc
Left Lobe	24*23*53	15.4 cc
Isthmus	4 mm	
Right Zone III lateral neck	Three defined-border L.N, 16*6, 17*5, 17*6 mm	without increased vascularity
Left Zone III lateral neck	Three defined-border L.N, 18*5, 17*5, 13*4 mm	Three defined-border L.N, without increased vascularity

Both thyroid lobes have decreased parenchymal echo, are heterogeneous, include pseudo-nodules, and have linear and enlarged echogenicity. Mild hypertrophy of isthmus was seen. In color Doppler evaluation, increased vascularity of both lobes was detected. There is no nodule or space-occupying lesion.

Abbreviations: LN: Lymph nodes, MM: millimeters

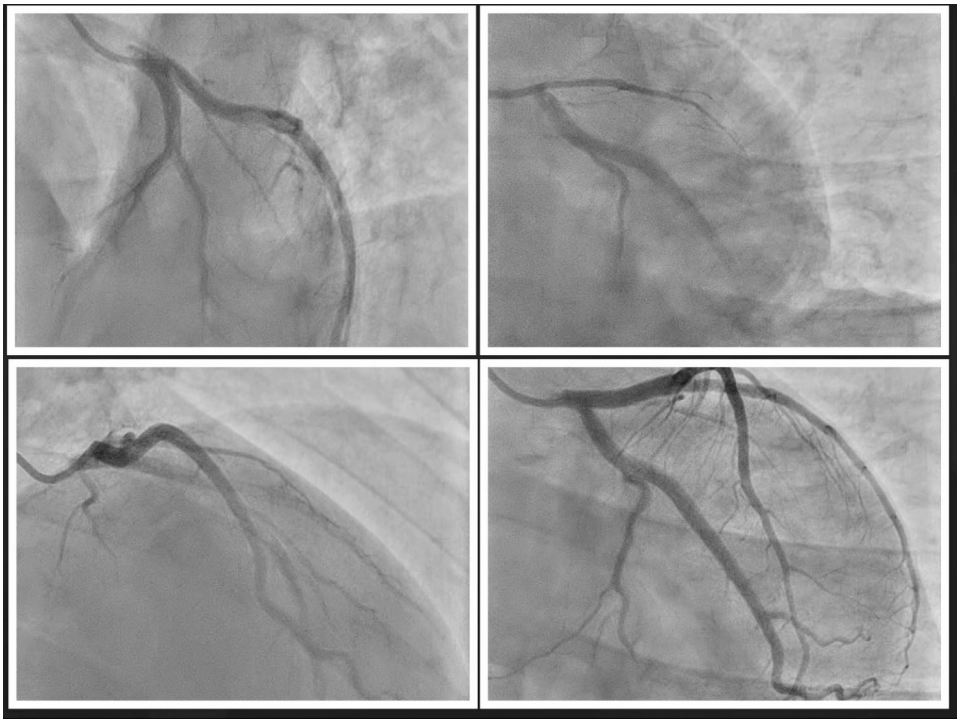

Fig. 3 The coronary angiography of the patient showed no abnormal finding

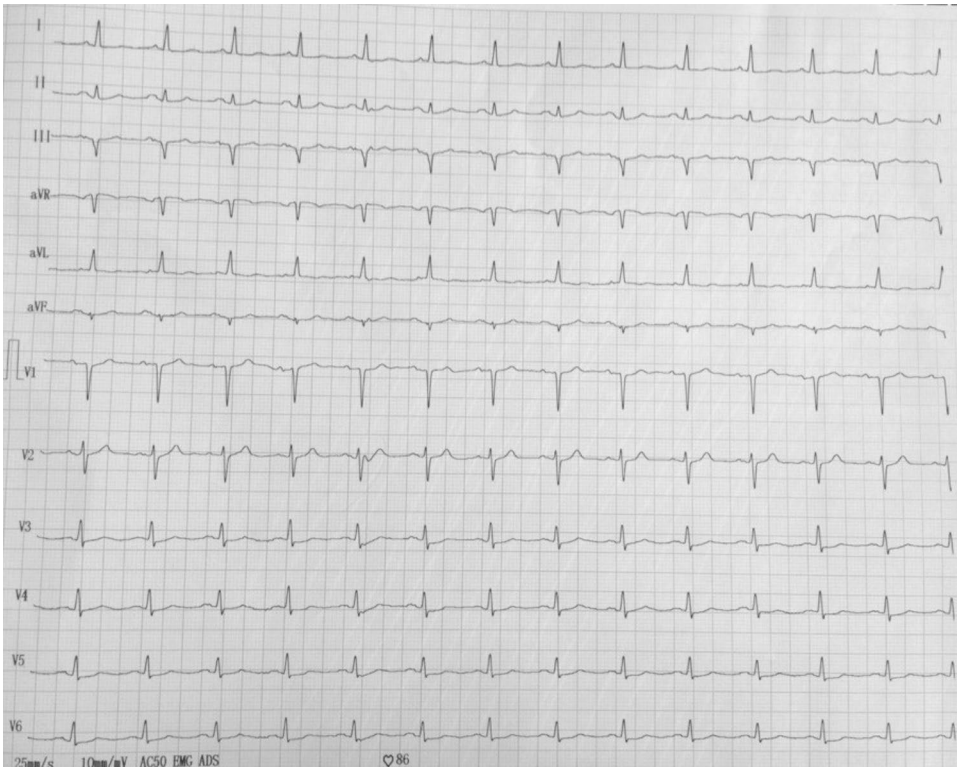

Fig. 4 Resolution of tachycardia and ST-segment changes in the follow-up ECGs

the absence of obstructive coronary arteries, and 3) the exclusion of any other background cause e for the AMI presentation, such as cardiac trauma [12]. However, the symptoms and clinical manifestations might be less severe, causing recurrent angina, known as thyrotoxicosis angina. The diagnostic criteria include four conditions: (I) lack of clinical presentations of hyperthyroidism at presentation, (II) rapid and progressive angina, iii) presence of pain at rest, and IV) resolution of symptoms after treating hyperthyroidism [13].

The most common background cause of thyrotoxicosis is Grave's disease, which is related to myocardial ischemia [14]. The underlying pathophysiology of this condition is the abnormal production of thyroid-stimulating Antibody (TSAb), also known as thyroid-stimulating immunoglobulin (TSI). The attachment of the immunoglobulin to the TSH receptors results in both the enlargement and increased secretion of the thyroid hormones, causing thyromegaly and hyperthyroidism [15]. The diagnosis of this condition relies on the history and physical examination along with laboratory data and imaging. Suppressed TSH, highly elevated T3, T4, and positive TSAb are diagnostic methods laboratory indicators [15].

The vasospasm caused by thyrotoxicosis might be intermittent and not be seen during the CAG. However, it is the main method of diagnosis. The absence of occlusion and probable vasospasm, which are reactive to the intracoronary infusion of nitroglycerine, are other associated diagnostic hints of Grave's-induced vasospasm and myocardial ischemia. In case of inconclusive CAG results, intravascular ultrasound (IVUS) or optical coherence tomography (OCT) can be beneficial for the differentiation of the obstructive and non-obstructive causes of MI [14]. Stress-induced cardiomyopathy is another differential diagnosis that should be ruled out in these cases (Takotsubo). It can be achieved by imaging strategies such as TTE, Cardiac magnetic resonance imaging (CMRI), and CAG [16].

The main recommended medications for treating these conditions are combined antithyroid medications (Methimazole, Carbimazole), Calcium channel or beta blockers, corticosteroids, and symptomatic therapy. Although treatment with beta-blockers is a standard part

Table 4 Hyperthyroidism-Induced Acute Coronary Syndrome Literature Review

First Author and YOP	Age and Sex	Presentation and Work-Up	Tx and Progression of Disease
Wu L. et al. 2021 [8]	31-y.o Female	Hx of Hyperthyroidism from 3 years ago, stopped taking PTU and MTM, Admitted due to AbNL Thyro function, Poor R prog, High hscTnI	Aspirin, statins, methimazole, dexamethasone sodium phosphate (once) IV, and hs-cTnI surprisingly dropped rapidly. However, the level of his-cTnI rose dramatically to its peak due to the discontinuation of GC. IV MetPred was initiated: Trop declined, and the condition improved.
Widya S. et al. 2024 [11]	50-y.o Female	+ Hx of HTN, DLP, presented with prolonged worsening left chest discomfort, with a heavy sensation, elevated BP, PR, T, Thyroid Nodule, Sinus Tachycardia, Spodick's sign on ECG, very low TSH high TRAb, T3, T4, DX: Grave's	Mistreatment for ACS, worsening of the symptoms, paroxysmal AF was seen, Grave's is diagnosed, PTU is started on the 3rd day of admission, improved significantly, and recovered on the 5th day.
Klomp M. et al. 2020 [14]	49-y.o Female	2-day intermittent chest pain SOB, Nausea, Sweating, sinus tachycardia ST-depression in the inferior leads + V4, V6 with inverted T and ST-elevation in lead aVR and V1, lab: Grave's disease, BWPS:25	Aspirin, ticagrelor, IV Heparin, and metoprolol plus nitroglycerine continuous infusion, CAG: RCA, LM ostial vasospasm, OCT: optical coherence tomography, Intracoronary NG, thiamazole 30 mg replaced ACS TX, resolution of symptoms in 1 day, CCB is added,
Malarkiewicz E. et al. 2015 [19]	41-y.o Male	Clinical and TTE evidence of Ant STEMI, five episodes of SCA, and VF converted by a defibrillator, 30 pack-year smokers, elevated T, hscTnI, WBC, granulocyte, D-dimer, increased CKMB, CRP, Low TSH, STE: V2-V5, STD: II, III, AVF, CAG: Amputation of proximal ADA, Lab Data and Ultrasound: Grave's, TTE: akinesia of apex and anterior septum, significant hypokinesia of posterior septum, dilated LA, LVEF: 42–45,	TX: PCI + stent in proximal ADA, typical myocardium infarction, and anti-thyroid therapy; recovered and discharged in good condition two weeks later.
Anjum. et al. 2022 [10]	47-y.o Female	Exertional sudden-onset chest pain radiating to the neck, + Hx of exertional SOB CAG: LM spasm Declined TSH, Normal troponin	Tx: initially for ACS and then MTM, Referred for urgent PCI, IC NG was used during CAG, which improved the symptoms. Readmitted two months later due to non-compliance, MTM caused the improvement.

YOP: Year of Publication, Y.O: years old, PTU: propylthiouracil, MTM: Methimazole, AbNL: abnormal, Thyro: Thyroid, hscTnI: High-Sensitivity Cardiac Troponin I, IV: Intravenously, GC: Glucocorticoids, MetPred: Methylprednisolone, HTN: Hypertension, DLP: Dyslipidemia, BP: Blood pressure, PR: Pulse rate, T: Temperature, ECG: Electrocardiogram, TSH: Thyroid Stimulating Hormone, TRAb: thyrotropin receptor antibody, ACS: Acute Coronary Syndrome, SOB: Shortness of Breath, CAG: Coronary Angiography, RCA: Right coronary artery, LM: Left main, OCT: optical coherence tomography, BWPS: Burch–Wartofsky Point Scale, CCB: Calcium channel blocker, STEMI: ST-Elevation Myocardial Infarction, TTE: Transthoracic Echocardiogram, SCA: Sudden Cardiac Arrest, VF: Ventricular Fibrillation, STE: ST-elevation, STD: ST-depression, ADA: Anterior-descending artery, LA: Left Atrium, LVEF: Left-Ventricle ejection fraction

of Hyperthyroidism-Induced tachycardia, it is controversial, considering the evidence of worsening symptoms in some cases. Aspirin and nitroglycerin long-time use, considering the uncertainty of their effectiveness, is not recommended, although there is no common consensus regarding their impact on these conditions [17]. A study conducted by Al Jaber et al. showed that these patients have an excellent prognosis with the application of appropriate anti-thyroid treatment [18]. Table 4 provides more information regarding the similar cases.

Clinical key point (conclusion)

Grave's disease, along with other types of hyperthyroidism, can result in ACS and, in severe cases, myocardial ischemia and infarction. The presentation might be without or with subtle extracardiac symptoms such as weight loss, tremors, insomnia, and anxiousness. It is recommended that patients with ACS symptoms be screened for secondary MI. Considering their different treatment, these MINOCA cases should be diagnosed and differentiated from the occlusive instances of myocardial infarction.

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Author contributions
FN and NN contributed to the conceptualization, resources, data curation and analysis, and project administration; SMM and AZ contributed to the supervision, validation, visualization, investigation, methodology, software, and writing the initial draft, and revision of the final draft of the manuscript. Both authors read and approved the final manuscript.

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Data availability
No datasets were generated or analysed during the current study.

Declarations

Ethical approval
Not Applicable.

Consent to participate
Not applicable.

Consent for publication
Written informed consent was obtained from the patient to publish this report following the journal's patient consent policy, and the procedure was performed by the center's ethical policy.

Competing interests
The authors declare no competing interests.

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