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Newly Diagnosed Graves Disease Presenting as Suspected Myocardial Injury With Nonobstructive Coronary Arteries

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Joel Thomas [1]

1. Internal Medicine, UCLA Health, Torrance, USA

Corresponding author: Joel Thomas, jthoma18@gmail.com

Abstract

Myocardial injury with ischemic symptoms and electrocardiographic abnormalities can occur in patients without obstructive coronary artery disease. We report a case of newly diagnosed Graves disease presenting with suspected myocardial injury with nonobstructive coronary arteries. A 55-year-old woman with hypertension presented with intermittent, nonexertional chest pressure and shortness of breath. High-sensitivity cardiac troponin I was elevated to 437 ng/L and subsequently downtrended. Thyroid testing demonstrated a suppressed thyroid-stimulating hormone level with elevated free thyroxine and positive thyrotropin receptor and thyroid-stimulating immunoglobulin testing, consistent with Graves disease. Transthoracic echocardiography showed preserved left ventricular systolic function without regional wall motion abnormalities. During regadenoson myocardial perfusion imaging, she developed chest discomfort with transient anterolateral T-wave inversions and submillimeter ST-segment depression despite normal myocardial perfusion. Her symptoms resolved promptly after sublingual nitroglycerin. Coronary computed tomography angiography performed after discharge demonstrated widely patent epicardial coronary arteries without obstructive disease and a coronary artery calcium score of 3. The combination of transient ischemic electrocardiographic changes, nitrate-responsive symptoms, elevated cardiac biomarkers, and nonobstructive coronary arteries raised suspicion for coronary vasomotor dysfunction associated with thyrotoxicosis. However, invasive coronary angiography, cardiac magnetic resonance imaging, and provocative testing for coronary spasm were not performed; therefore, the mechanism remains unconfirmed. This case highlights the importance of considering thyroid dysfunction in patients presenting with suspected myocardial injury despite the absence of obstructive coronary disease.

Categories: Cardiology, Internal Medicine, Endocrinology/Diabetes/Metabolism
Keywords: coronary vasospasm, graves disease, myocardial injury, nonobstructive coronary arteries, regadenoson, thyrotoxicosis

Introduction

Myocardial injury with nonobstructive coronary arteries (MINOCA) is a working diagnosis applied to patients who meet the criteria for acute myocardial injury but have no obstructive epicardial coronary artery disease and no alternative explanation for the myocardial injury. MINOCA encompasses multiple ischemic mechanisms and should prompt further evaluation rather than be regarded as a final diagnosis [1-3].

Potential mechanisms include plaque disruption, coronary artery spasm, coronary microvascular dysfunction, coronary embolism or thrombosis, and spontaneous coronary artery dissection. Nonischemic conditions that can mimic acute myocardial injury, particularly myocarditis and stress cardiomyopathy, must also be considered during the diagnostic evaluation [1-3]. Contemporary recommendations emphasize a systematic, multimodality approach that may include coronary angiography, intracoronary imaging, cardiac magnetic resonance imaging, and provocative testing for coronary vasomotor dysfunction when clinically appropriate [2,3].

Coronary artery spasm is an important potentially reversible cause of myocardial ischemia in patients with nonobstructive coronary arteries. It can produce transient ischemic electrocardiographic changes and chest discomfort and may respond promptly to nitrates. Intracoronary acetylcholine or ergonovine provocation can identify epicardial or microvascular spasm in selected patients, although such testing is not routinely performed in all clinical settings [4,5].

Hyperthyroidism has important cardiovascular effects, including increased heart rate, myocardial contractility, cardiac output, and myocardial oxygen consumption. Thyroid hormone excess also alters adrenergic signaling and vascular physiology and has been associated with coronary vasospasm [6,7]. Severe coronary artery spasm has been described in patients with Graves disease, and case reports have documented myocardial injury associated with thyrotoxicosis and coronary vasospasm [7,8].

We present a 55-year-old woman with previously unrecognized Graves disease who developed chest pain,

elevated cardiac biomarkers, and transient ischemic electrocardiographic changes during pharmacologic stress testing. Subsequent coronary computed tomography angiography demonstrated widely patent epicardial coronary arteries without obstructive disease. The case raises the possibility of thyrotoxicosis-associated coronary vasomotor dysfunction and illustrates the diagnostic limitations encountered when invasive angiography, cardiac magnetic resonance imaging, and provocative testing are not performed.

Case Presentation

A 55-year-old woman with a history of hypertension treated with amlodipine 5 mg daily presented with a two-day history of intermittent left-sided chest discomfort and shortness of breath. The discomfort was not clearly exertional. She denied diaphoresis, palpitations, syncope, dizziness, cough, or fever. She was a lifelong nonsmoker. She denies use of alcohol or illicit substances. On presentation, blood pressure was 151/67 mmHg, and the remainder of the vital signs were unremarkable. The initial electrocardiogram demonstrated normal sinus rhythm with nonspecific ST-segment/T-wave abnormalities (Figure *1*).

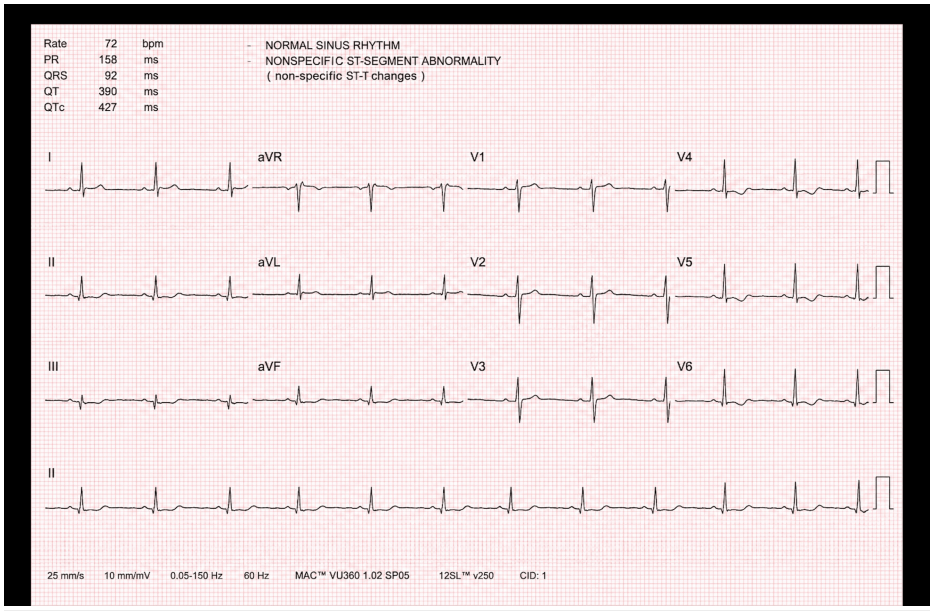


FIGURE 1: Baseline electrocardiogram

Baseline 12-lead electrocardiogram demonstrating normal sinus rhythm with nonspecific ST-segment/T-wave abnormalities and no diagnostic ST-segment elevation.

bpm: beats per minute; ms: milliseconds

Initial laboratory evaluation demonstrated an elevated high-sensitivity cardiac troponin I level, which peaked at 437 ng/L and subsequently downtrended. Hemoglobin A1c was 5.6%, and low-density lipoprotein cholesterol was 65 mg/dL. Transthoracic echocardiography demonstrated preserved left ventricular systolic function without regional wall motion abnormalities.

Because of the presenting symptoms and elevated cardiac biomarkers, further evaluation for myocardial ischemia was pursued. During regadenoson pharmacologic stress testing, the patient developed chest discomfort accompanied by new anterolateral T-wave inversions and approximately 0.5 mm of ST-segment depression (Figure *2*). Her symptoms resolved promptly after sublingual nitroglycerin. Despite the electrocardiographic changes and symptoms, myocardial perfusion imaging demonstrated normal perfusion without reversible or fixed perfusion defects. Gated single-photon emission computed tomography showed normal wall motion and a stress left ventricular ejection fraction of 86% (Figure *3*).

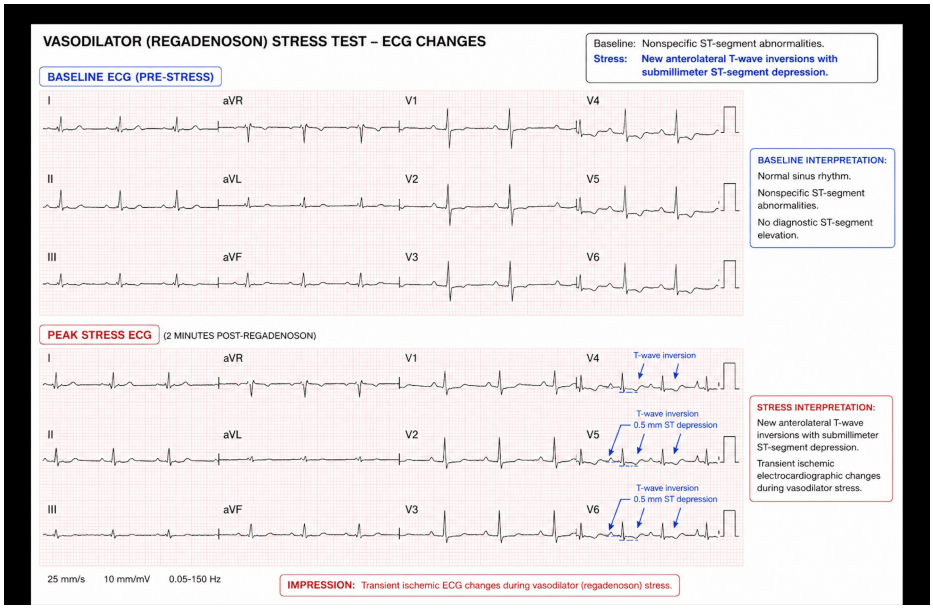


FIGURE 2: Transient electrocardiographic changes during regadenoson stress

Comparison of baseline and peak-stress electrocardiographic tracings demonstrating new anterolateral T-wave inversions with approximately 0.5 mm of ST-segment depression during regadenoson administration.

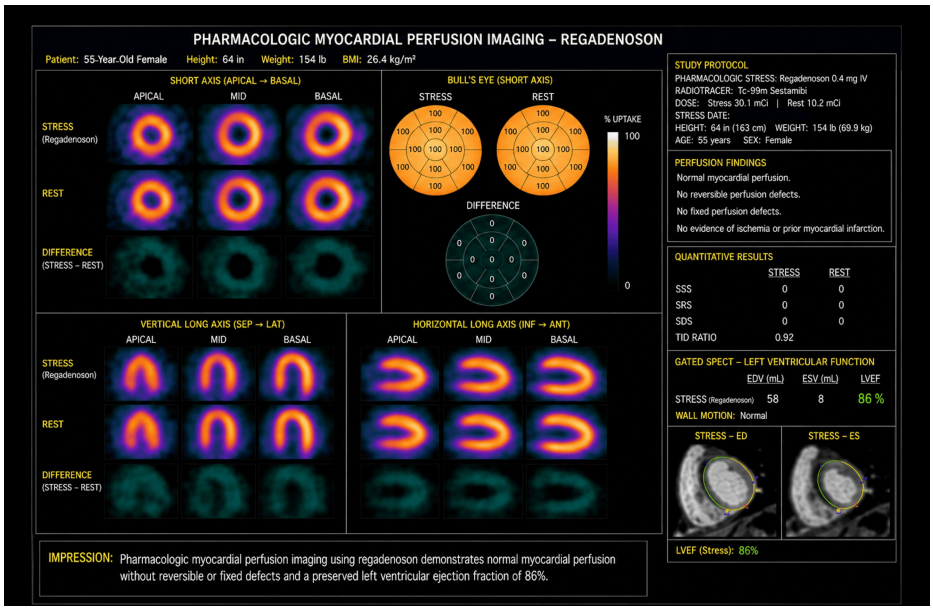


FIGURE 3: Regadenoson myocardial perfusion imaging

Pharmacologic myocardial perfusion imaging using regadenoson demonstrating normal myocardial perfusion without reversible or fixed defects. Gated single-photon emission computed tomography demonstrates normal wall motion and a stress left ventricular ejection fraction of 86%.

mCi: millicurie; SSS: Summed Stress Score; SRS: Summed Rest Score; SDS: Summed Difference Score; TID ratio: transient ischemic dilation ratio; EDV: end-diastolic volume; ESV: end-systolic volume; LVEF: left ventricular ejection fraction

Thyroid testing obtained during the evaluation demonstrated a thyroid-stimulating hormone level below 0.010 micro-international units/mL and an elevated free thyroxine level of 2.04 ng/dL. Thyrotropin receptor antibody and thyroid-stimulating immunoglobulin testing were positive, supporting a diagnosis of Graves disease. These findings provided a potential systemic contributor to the patient's dynamic coronary

vasomotor abnormalities.

Coronary computed tomography angiography performed approximately two weeks after discharge demonstrated right-dominant coronary circulation with widely patent epicardial coronary arteries and no obstructive coronary artery disease. A single eccentric calcified plaque was identified in the first diagonal branch without luminal stenosis. The total coronary artery calcium score was 3 (Figure *4*).

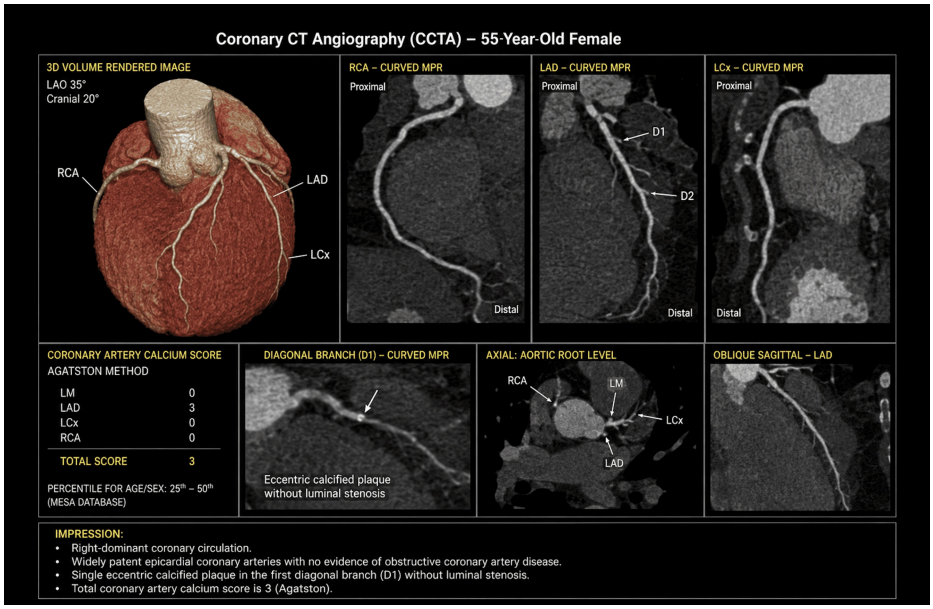


FIGURE 4: Coronary computed tomography angiography

Coronary computed tomography angiography demonstrating right-dominant coronary circulation and widely patent epicardial coronary arteries without obstructive coronary artery disease. A small eccentric calcified plaque is present in the first diagonal branch without luminal stenosis. The total coronary artery calcium score is 3 (Agatston).

LM: left main; LAD: left anterior descending artery; LCx: left circumflex artery; RCA: right coronary artery

The patient was discharged on medical therapy including aspirin, atorvastatin, amlodipine, and isosorbide mononitrate. Antithyroid therapy was not initiated at this time as it was recommended to follow up with endocrinology. At three months of follow-up, she had not reported recurrent anginal symptoms; therefore, further cardiac studies were not performed. At this time, she had not arranged endocrinology follow-up, so thyroid function tests had not been reassessed (Table *1*).

Selected laboratory and cardiac diagnostic tests	Result	Clinical relevance
High-sensitivity cardiac troponin I	Peak 437 ng/L; subsequently downtrended	Evidence of myocardial injury
Thyroid-stimulating hormone	<0.010 micro-international units/mL	Markedly suppressed
Free thyroxine	2.04 ng/dL	Elevated
Thyrotropin receptor antibody	Positive; 3.27 IU/L reported	Supports Graves disease
Thyroid-stimulating immunoglobulin	Positive; 2.00 IU/L reported	Supports Graves disease
Hemoglobin A1c	5.6%	No diabetes by this measure
Low-density lipoprotein cholesterol	65 mg/dL	Low atherosclerotic lipid burden
Stress myocardial perfusion	Normal; no reversible or fixed defect	No perfusion evidence of inducible ischemia
Stress left ventricular ejection fraction	86%	Preserved systolic function
Coronary artery calcium score	3 (Agatston)	Minimal calcified plaque burden

TABLE 1: Selected laboratory and cardiac diagnostic tests

Discussion

MINOCA represents a diagnostic working diagnosis rather than a single disease entity, and identifying the underlying mechanism is important because management differs according to the cause [1-3]. In the present case, the patient had symptoms compatible with myocardial ischemia, elevated high-sensitivity cardiac troponin I, and transient ischemic electrocardiographic changes. Subsequent coronary computed tomography angiography showed widely patent epicardial coronary arteries without obstructive disease. However, because invasive coronary angiography was not performed and nonischemic causes of myocardial injury were not definitively excluded with cardiac magnetic resonance imaging, the case is best characterized as suspected MINOCA rather than confirmed MINOCA.

Coronary vasomotor dysfunction was considered the most likely mechanism in this patient. She developed chest discomfort and transient anterolateral T-wave inversions with submillimeter ST-segment depression during regadenoson stress testing, followed by prompt symptom resolution after sublingual nitroglycerin. Chest discomfort and transient ST-T changes can occur during regadenoson stress testing even when myocardial perfusion imaging is normal. Therefore, these findings, including nitrate responsiveness, are supportive but not specific for coronary vasospasm. Acetylcholine or ergonovine provocation can demonstrate epicardial or microvascular spasm in selected patients [4,5]. Nevertheless, the findings in this case are suggestive rather than diagnostic because provocative testing was not performed.

The patient's newly diagnosed Graves disease provides a plausible physiologic context for coronary vasomotor dysfunction. Thyroid hormone excess increases cardiac chronotropy and inotropy and alters systemic and vascular physiology [6,9]. Hyperthyroidism has also been associated with coronary artery spasm. In a series of patients with severe coronary artery spasm associated with hyperthyroidism, all patients had Graves disease, and several demonstrated coronary vasoconstriction during angiography [7]. Case reports have additionally described myocardial injury associated with Graves disease and angiographically demonstrated coronary vasospasm [8,10].

The present case has similarities to previously reported cases but differs in an important respect. In a previously reported patient with Graves disease, severe right coronary and left main coronary artery spasm was demonstrated during invasive angiography and resolved after intracoronary nitroglycerin; intravascular optical coherence tomography also excluded atherosclerosis, thrombus, and dissection [8]. In contrast, our patient did not undergo invasive angiography or provocative testing. Instead, the diagnosis was supported indirectly by transient ischemic electrocardiographic changes during pharmacologic stress, nitrate-responsive symptoms, elevated cardiac biomarkers, and subsequent demonstration of nonobstructive coronary arteries by computed tomography angiography.

The normal myocardial perfusion study despite transient electrocardiographic abnormalities is also noteworthy. This finding does not establish coronary spasm but illustrates that dynamic coronary vasomotor abnormalities may not necessarily produce a persistent perfusion defect on a single stress study. The

patient's preserved ventricular function and absence of regional wall motion abnormalities further argue against a large fixed myocardial injury, although they do not exclude transient ischemia.

The case also emphasizes the importance of distinguishing suspected MINOCA from other causes of myocardial injury. Cardiac magnetic resonance imaging was not performed, so myocarditis and other nonischemic myocardial injury could not be definitively excluded. Similarly, intracoronary imaging was not performed, limiting assessment for plaque disruption or spontaneous coronary artery dissection. These limitations prevent definitive assignment of the mechanism and should be considered when interpreting the association between Graves disease and the patient's myocardial injury.

The patient's absence of recurrent angina during three months of follow-up while receiving calcium-channel blocker and long-acting nitrate therapy is reassuring, although symptom resolution cannot establish the mechanism or prove treatment efficacy in an individual case. Further evaluation and management of the underlying Graves disease remain important.

Conclusions

Newly diagnosed Graves disease may be associated with coronary vasomotor dysfunction and should be considered in selected patients presenting with myocardial injury and suspected nonobstructive coronary disease. In this patient, transient ischemic electrocardiographic changes, nitrate-responsive chest discomfort, elevated cardiac biomarkers, and subsequent demonstration of nonobstructive coronary arteries raised suspicion for thyrotoxicosis-associated coronary vasomotor dysfunction.

Because invasive coronary angiography, cardiac magnetic resonance imaging, and provocative testing were not performed, the diagnosis and underlying mechanism could not be confirmed. The case nevertheless highlights the importance of considering thyroid dysfunction during the evaluation of patients with suspected myocardial injury and nonobstructive coronary arteries.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

Concept and design: Joel Thomas

Acquisition, analysis, or interpretation of data: Joel Thomas

Drafting of the manuscript: Joel Thomas

Critical review of the manuscript for important intellectual content: Joel Thomas

Supervision: Joel Thomas

Disclosures

Human subjects: Informed consent for treatment and open access publication was obtained or waived by all participants in this study. **Conflicts of interest:** In compliance with the ICMJE uniform disclosure form, all authors declare the following: **Payment/services info:** All authors have declared that no financial support was received from any organization for the submitted work. **Financial relationships:** All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. **Other relationships:** All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

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