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CASE REPORT

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A recurrent typical angina pectoris without any finding in coronary angiography: Microvascular angina

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Key Clinical Message

Microvascular angina (MVA) can present with recurrent chest pain and normal coronary angiography. Recognizing MVA is crucial as it significantly impacts patient morbidity and mortality. Early diagnosis and management with antianginal medications are essential for improving outcomes and quality of life.

Abstract

Cardiovascular diseases are still the main cause of death in many parts of the world. Chest pain and dyspnea are always concerning due to the implications of cardiovascular disease. However, in patients with the involvement of the small coronary vessels (Microvascular Angina), symptoms might be recurrent and persistent despite the presence of normal coronary vessel evaluations. A 45-year-old man with a 25-year smoking history presented with recurrent chest pain, especially during physical activity, and mild shortness of breath. He was admitted, and a coronary angiography the next day appeared normal. However, a cardiac PET scan revealed the involvement of small coronary vessels not visible on angiography. The Patient was a 45-year-old man who presented with recurrent chest pain, more prominent during physical activity. He also had mild shortness of breath. The patient was admitted, and the next day, he underwent normal coronary angiography. The cardiac positron emission tomography (PET scan) showed the involvement of small coronary vessels that were not obvious on angiography.

KEYWORDS

acute coronary syndrome, cardiovascular disease, microvascular angina, microvascular dysfunction, nonobstructive coronary disease

1 | INTRODUCTION

The definition of ischemic heart disease (IHD) is an imbalance between the blood supply and the demand of the heart muscle (myocardium) due to pathologic narrowing or blockage of the coronary vessels.[1] Coronary artery disease can present with a wide range of manifestations, from ST-elevation myocardial infarction (STEMI) caused by large thrombosis or significant atherosclerosis to chronic coronary syndrome caused by gradual partial

obstruction of the coronary arteries.[2,3] According to the 2024 American Heart Association reports, coronary artery diseases are one of the main causes of death in the United States (US)[4,5] and all over the world.[6] Chest pain (angina pectoris), shortness of breath (SOB), and diaphoresis are the most common and cardinal symptoms observed in patients with these conditions.[7,8] The obstruction of coronary arteries can also be caused by severe thrombosis, atherosclerosis of the coronary arteries, or sometimes the obstruction in minor and small coronary arteries that cannot be detected in specialized coronary imaging.[2,9]

One of the rarely reported causes of typical chest pain is microvascular angina (MVA) which has been attributed to several probable etiologies and is yet to be completely understood.[10] It has been shown that one of the causes of this condition might be the transient narrowing of the vessels, mainly caused by the dysfunction of the endothelium of these arteries.[8] This condition is more commonly seen among postmenopausal women.[11] Although the etiology of this condition seems to be different from obstructive coronary diseases, the risk factors of the MVA are the same, including diabetes, hypertension, aging, and smoking.[12] The diagnosis of the MVA is not always straightforward.[13] The coronary angiography and electrocardiogram between episodes are mostly normal, but the myocardial perfusion scan might reveal diminished blood flow.[9,10] MVA is caused by an increase in vascular tone; after that, it can cause compromise in myocardial perfusion.[14] Patients with MVA are at high risk for major cardiovascular adverse events, including cardiovascular mortality.[15] Considering that MVA is mostly overlooked in daily practice, the patient would probably be exposed to repetitious imaging, angiograms, emergency room visits, hospital admissions, and diminished quality of life.[16]

In this case, we present a 45-year-old man without any past medical history (PMH) who presented to the emergency department (ED) with a complaint of chest pain and diaphoresis that began 20 min ago while he was shoveling the snow.

2 | CASE PRESENTATION

A 45-year-old man presented to the emergency department (ED) with severe retrosternal chest pain, shortness of breath (SOB), and diaphoresis. His symptoms had begun 20 min earlier while he was shoveling snow. The pain persisted but subsided after resting in the car on the way to the ED. The patient had a history of similar episodes that were previously evaluated and were diagnosed as noncardiac (musculoskeletal) pain. During those investigations, he had undergone multiple tests, including electrocardiograms (ECGs), echocardiograms, stress tests, and blood tests, all of which showed no remarkable abnormality. He had been smoking for 25 years, averaging 10 cigarettes per day (12.5 pack-year). He stated a history of hypertension and ischemic heart disease (IHD), in his father's medical history, starting at age 55. The patient denied any alcohol or illicit drug use and declared that he had a sedentary lifestyle with minimal physical activity.

3 | METHOD

Upon arrival, the patient was anxious and diaphoretic. Vital signs were stable with a blood pressure of 130/85 mmHg, heart rate of 75 beats per minute, respiratory rate of 18 breaths per minute, and oxygen saturation of 98% on ambient air. Cardiopulmonary examination revealed no abnormal findings; the lungs were clear to auscultation, and heart sounds were normal without murmurs, gallops, or rubs. Abdominal examination was unremarkable, and there were no signs of peripheral edema. Neurological examination showed the patient was alert and oriented with no focal deficits.

Continuous cardiopulmonary monitoring showed a normal heart rate (75/min) and sinus rhythm. Serial ECGs showed no significant ST-T changes (Figure 1), and an echocardiogram revealed no significant abnormalities (Table 1), with a normal left ventricular ejection fraction. Initial blood tests (Table 2), including complete blood count (CBC), basic metabolic panel (BMP), lipid profile, and cardiac enzymes (Troponin I and high-sensitivity Troponin) were within normal Ranges. The hs-cTnI levels were checked upon arrival and 6 h later. Both tests showed levels below the 99th percentile upper reference limit

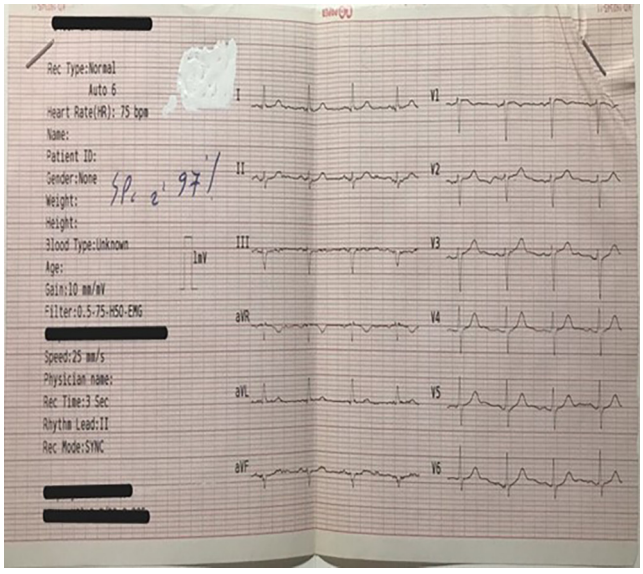

FIGURE 1 No significant abnormality was detected in the patient's initial ECG in the emergency department.

TABLE 1 Comprehensive echocardiographic findings.

Category and measurement	Finding
M-mode dimensions (mm): RV	32 mm
M-mode dimensions (mm): AO	35 mm
M-mode dimensions (mm): LA	34 mm
M-mode dimensions (mm): IVS	11 mm
M-mode dimensions (mm): LVPW	11 mm
M-mode dimensions (mm): TAPSE	23 mm
M-mode dimensions (mm): LV	52 mm/39 mm
2D Study: mitral valve	WNL
2D Study: aortic valve	Mildly thickened & dome
2D Study: tricuspid and pulmonary valves	WNL
EF (and RWMA): EF	55%, No RWMA
Doppler & Color Flow Study: mitral valve	Mild MR, E/A > 1
Doppler & Color Flow Study: aortic valve	Mild AI, Width of AI/LVOT = 6/34, AIVC = 3 mm, AI PHT = 751 ms, AVPG = 8 mmHg
Doppler & Color Flow Study: tricuspid and pulmonary valves	Trivial TR, TRG = 27 mmHg, Trivial PI, PVPG = 6 mmHg
Doppler & Color Flow Study: mitral valve	Mild MR
Doppler & Color Flow Study: aortic valve	Mild AI
Doppler & Color Flow Study: tricuspid and pulmonary valves	Trivial TR, PAPs = 32 mmHg
Tissue Doppler Study: PEV	76 cm/s
Tissue Doppler Study: PAV	69 cm/s
Tissue Doppler Study: RVSM	15 cm/s

(URL) of 0.04 ng/mL, indicating no significant myocardial injury.

Based on the presenting symptoms, a provisional diagnosis of acute coronary syndrome (ACS) was made. The patient was given aspirin, clopidogrel, atorvastatin, and nitroglycerin. The chest pain resolved completely within 5 min of nitroglycerin administration. Coronary angiography (CAG) was performed 12 h after admission, showing no significant narrowing or thrombotic lesions in any of the coronary arteries, indicating normal coronary arteries (Figure 2).

The patient was observed in the Coronary Care Unit (CCU) for 24 h. Continuous monitoring showed resolution of the initially observed ST-segment elevation within 24 h. Repeat ECGs and echocardiograms showed no significant changes. The patient was discharged with prescriptions for atenolol (50 mg daily), atorvastatin (40 mg nightly), aspirin (80 mg daily), and isosorbide dinitrate (10 mg two to three times a day). He was advised to follow up in the outpatient clinic.

Given the normal results from standard diagnostic tests and the persistence of symptoms, a more advanced diagnostic tool was needed to assess microvascular function. A cardiac positron emission tomography (PET) scan was chosen and subsequently revealed MBF at 1.5 mL/g/min and MPR at 1.2, confirming the diagnosis of MVA. The patient reported no abnormal symptoms at follow-up appointments 2 weeks, 1 month, 3 months, and 6 months post-discharge. Follow-up ECGs and echocardiograms remained normal. In addition to regular clinical assessments, the patient underwent a cardiovascular magnetic resonance (CMR) scan 3 months post-discharge, which showed no evidence of ischemia or fibrosis, further confirming the stability of the condition. The patient was encouraged to adopt lifestyle changes, including smoking cessation and increased physical activity. Educational materials and counseling sessions were provided to support these changes, and the patient's adherence to these recommendations was regularly assessed during follow-up visits.

4 | CONCLUSION AND RESULTS

The patient's symptoms resolved after taking standard antianginal medications and observing the recommended lifestyle changes. The patient was discharged with medications, and follow-up appointments revealed no recurrent symptoms and normal ECGs and echocardiograms. Additional monitoring, including bimonthly blood pressure checks and a CMR scan, showed normal results, confirming the stability of the condition.

5 | DISCUSSION

About two-thirds of patients with angina pectoris do not show any significant obstruction in their invasive diagnostic procedures.[17] These episodes of pain might be accompanied by evidence of ischemia in the myocardium despite normal coronary angiography. This condition is named “angina with non-obstructive coronary angiography” (ANOCA) and “ischemia with non-obstructive coronary arteries” (INOCA).[15,18] These sequels' etiology is epicardial coronary vasospasm and coronary microvascular dysfunction (CMD).[14,19] The clinical manifestation of this condition can be highly variable, ranging from atypical chest pain at rest or night to typical angina pectoris commencing or worsening by exertion.[20] The diagnostic criteria of microvascular angina Coronary

TABLE 2 Laboratory findings of the patient.

Test	Result	Reference range
RBC (10^6/μl)	4.8	4.2–5.5
Hemoglobin (gr/dL)	15	12–16
WBC (per μl)	8600	4.000–11.000
MCV (fL)	87.2	80–99
Hematocrit (%)	45.6	37–47
Platelet (per μl)	184.000	150.000–400.000
Neutrophils (%)	66%	40–75
Lymphocytes (%)	35.1%	20–45
Eosinophils (%)	4.4%	0–6
MCH (pg/cell)	30.3	27–31
MCHC (g/dL)	32.7	32–36
Troponin I (Fist sample)	Negative	Negative
Lipid profile, coagulation factors, and troponin		
Cholesterol	155	Up to 200
TG (mg/dl)	91	Up to 150
LDL (mg/dl)	63	Up to 130
HDL (mg/dl)	42	>45 mg/dL
Blood sugar (mg/dl)	98	74–106 mg/dL
LDH (Iu/L)	335	235-470Iu/L
K^+ (meq/lit)	4.0	3.5–5.3 meq/lit
Creatinine (mg/dl)	0.95	0.5–1.00
Urea (mg/dl)	19	13–43
Hb A1C %Hb	5.3	4.8–5.9
PT	12S	11–13 s
PTT	29 s	25–38 s
INR	1.3	1–1.5
CK-MB	16 u/l	<25u/l
Troponin I (Second sample in 6 h)	Within normal range	<0.04 ng/mL
High-sensitivity troponin (Upon arrival)	Within normal range	<14 ng/L (females) < 22 ng/L (males)
High-sensitivity troponin (6 h later)	Within normal range	<14 ng/L (females) < 22 ng/L (males)

Vasomotor Disorder International Study group (COVADIS) are (1) disturbed flow in the microvascular coronary circulation, (2) no obstruction in coronary artery disease, (3) clinical symptoms of myocardial infarction, (4) Objective evidence of myocardial ischemia (not mandatory).[19]

Several previous studies have tried to explain the main reason behind this syndrome. Based on the anatomy of the coronary arteries, consisting of four subgroups of (1) epicardial arteries (>400 μM), the periarteriolar vessels (400–100 μM), arterioles (<100 μM), and capillaries (<10 μM),[21] it seems that involvement of coronary microvascular dysfunction (CMD), epicardial and microvascular endothelial and nonendothelial dysfunction that limits myocardial perfusion is the main reason for this condition.[19]

This pathologic condition can be caused by structural (like inflammation or atherosclerosis, coronary microembolization) or functional epicardium abnormality.[22,23] It is believed that in CMD, microvascular dysfunction happens due to the secretion of cytokines from the injured endothelium.[24] Consequently, abnormal release of inflammatory cytokines leads to vascular spasms and a decline in pain tolerance, which results in pain and chest discomfort.[25] The feature distinguishing between structural CMD and other pathologies is the observation of normal rest CBF, a reduction in CBF under stress and in coronary

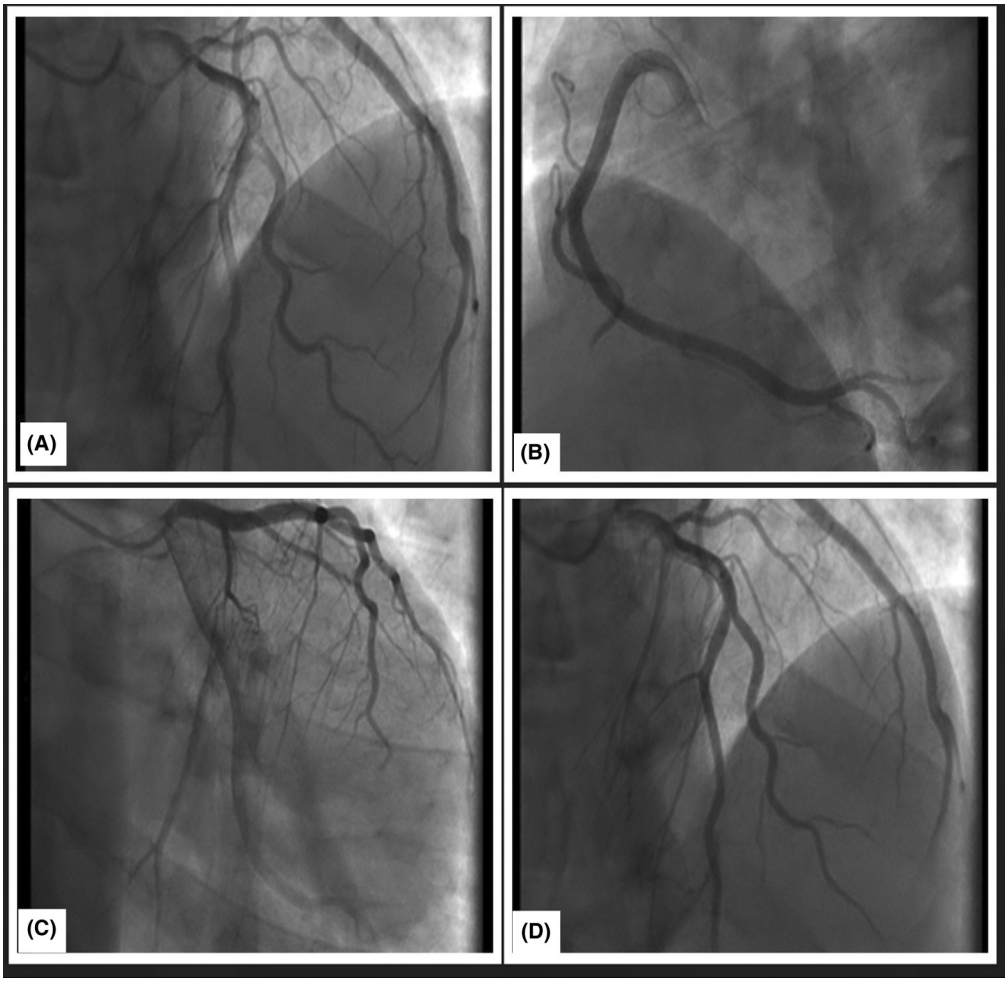

FIGURE 2 Coronary angiography of the patient shows 2A, 2C, and 2D: LAD and LCX without any specific lesion, narrowing, or abnormality. 2B: Shows normal RCA without remarkable narrowing, lesion, or abnormality. Right coronary Artery: Dominant without stenosis.

flow reserve (CFR), and higher resistances under stress in structural CMD.[26] On the contrary, in functional CMD, hyperstimulation of alpha-adrenergic stimulators during sympathetic activation causes exhaustion of the muscular part of the involved coronary vessels. This leads to declined vasodilatory capacity characterized by lower vascular tone at rest and under stress, resulting in a higher CBF at rest, normal stress CBF, and normal resistance under hyperemic stimulus but reduced CFR.[23,26] The definite cause of MVA is not thoroughly identified; however, the three factors of increased oxygen requirement, alteration of nitric oxide pathway, and pathologic change in the structure of the vessels are the main contributing factors.[27]

The diagnosis of MVA is not always straightforward since, in most cases, no abnormality can be seen in electrocardiogram, transthoracic echo, chest X-ray, CT angiography, or coronary angiography. There are two main diagnosis categories for MVA: invasive and noninvasive methods.[28] The most accurate invasive modalities are intracoronary Doppler wire or an intracoronary thermodilution-derived method that measures CBF's response under vasoactive stimuli.[29] The most used stimulator vasoactive medication is adenosine. However, the intracoronary acetylcholine has shown better outcomes.[13,30] According to 2019 ESC guidelines, after injection of acetylcholine, an increase of less than 50% indicates CMD.[18] The main noninvasive method for the diagnosis of MVA (CMD) is positron imaging tomography (PET), which evaluates the myocardial blood flow (MBF), the myocardial perfusion reserve (MPR), and the myocardial flow reserve (MFR). The MPR is defined as the MBF at the maximum stress, while the MFR is the ratio of MBF at maximal coronary vasodilation and resting MBF.[27] CMD is correlated with the MFR <1.5.[31,32] Cardiac magnetic resonance, cardiac CT scan, and pulsed Doppler echo can also guide the diagnosis of MVA.[28]

This pathologic circulation is most often detected as reduced CFR detected by invasive Doppler or noninvasive advanced imaging such as PET or cardiac MRI. In line with the guidelines by Camici and Crea (2015), PET was selected for its ability to noninvasively measure CFR. This

approach minimizes the need for invasive procedures while effectively diagnosing microvascular dysfunction, ensuring accurate and patient-friendly diagnostic practices.[20,33] CMR is a highly valuable tool in the diagnosis and management of various cardiovascular conditions, including vasospastic angina (VSA) and CMD. CMR provides detailed imaging and functional assessment of the myocardium, which is crucial in cases where other diagnostic methods are inconclusive. It can also be effectively used for follow-up to monitor disease progression and treatment efficacy.[34]

MVA is usually associated with increased endothelial inflammation or atherosclerosis due to cardiovascular risk factors such as diabetes, hypertension, and dyslipidemia. The mainstay of treatment in these patients is antianginal, aiming to balance the demand and supply of blood to the myocardium through coronary arteries.[35–37] The choice of medications, such as beta-blockers, calcium channel blockers, nitrates, and statins, aligns with the guidelines for managing microvascular angina as detailed in recent studies. These recommendations provide a solid evidence base for the therapeutic strategy, ensuring effective management of the condition with appropriately justified dosages.[28,38] The overall prognosis of the patients with CMD has been reported to be poor. This condition is correlated with an increased risk of major cardiovascular adverse events, declined quality of life, and higher healthcare expenditure due to multiple diagnostic procedures and hospital admissions.[39–41]

5.1 | Clinical learning point (conclusion)

Patients with recurrent chest pain who have had no remarkable abnormality in their routine evaluation of cardiac diseases, especially in postmenopausal women, should be examined for microvascular coronary pathologies. It is advisable to consider invasive or noninvasive evaluation of small coronary vessels. Due to the high risk of major adverse cardiac events and mortality, these patients should be treated as soon as possible. The mainstay of these patients' treatment is antianginal medications such as beta-blockers and calcium channel blockers.

AUTHOR CONTRIBUTIONS

Homa Taheri: Conceptualization; data curation; investigation; methodology; writing – original draft. **Maryam Taheri:** Conceptualization; data curation; investigation; methodology; writing – original draft. **Pouya Ebrahimi:** Data curation; software; supervision; writing – original draft; writing – review and editing. **Parnian Soltani:** Data curation; writing – review and editing. **Amin Zaki Zadeh:** Data curation; writing – review and editing. **Mohsen Anafje:** Data curation; software; supervision; writing – original draft; writing – review and editing.

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CONFLICT OF INTEREST STATEMENT

The authors declare no conflicts of interest.

DATA AVAILABILITY STATEMENT

There is no supporting data, and all available information is included in the manuscript.

CONSENT

Written informed consent was obtained from the patient to publish this report under the journal's patient consent policy.

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REFERENCES

1. Institute of Medicine (US) Committee on Social Security Cardiovascular Disability Criteria. *Cardiovascular Disability: Updating the Social Security Listings*. National Academies Press (US); 2010.
2. Safi M, Nazari R, Senobari N, Taheri H, Ebrahimi P. A woman with eptifibatide (integrilin)-induced thrombocytopenia following treatment of a clot in her coronary artery: a case report and literature review. *Clin Case Rep*. 2024;12(4):e8694.
3. Ahmed B. New insights into the pathophysiology, classification, and diagnosis of coronary microvascular dysfunction. *Coron Artery Dis*. 2014;25(5):439-449.
4. Martin SS, Aday AW, Almarzooq ZI, et al. 2024 heart disease and stroke statistics: a report of US and global data from the American Heart Association. *Circulation*. 2024;149(8):e347-e913.
5. Behnoush AH, Maleki S, Arzhangzadeh A, et al. Prediabetes and major adverse cardiac events after acute coronary syndrome: an overestimated concept. *Clin Cardiol*. 2024;47(4):e24262.
6. Khan MA, Hashim MJ, Mustafa H, et al. Global epidemiology of ischemic heart disease: results from the Global Burden of Disease Study. *Cureus*. 2020;12(7):e9349.
7. Brown JC, Gerhardt TE, Kwon E. *Risk Factors for Coronary Artery Disease*. StatPearls Publishing; 2024.
8. Jensen RV, Hjortbak MV, Bøtker HE. Ischemic heart disease: an update. *Semin Nucl Med*. 2020;50(3):195-207.
9. Kounis NG. Kounis syndrome (allergic angina and allergic myocardial infarction): a natural paradigm? *Int J Cardiol*. 2006;110(1):7-14.
10. Jha S. Cardiac syndrome X: the sensitive heart of a young adult man. *Cureus*. 2021;13(12):e20669.
11. Murthy VL, Naya M, Taqueti VR, et al. Effects of sex on coronary microvascular dysfunction and cardiac outcomes. *Circulation*. 2014;129(24):2518-2527.

12. Aldiwani H, Mahdai S, Alhatemi G, Bairey Merz CN. Microvascular angina: diagnosis and management. *Eur Cardiol*. 2021;16:e46.
13. Shimokawa H, Suda A, Takahashi J, et al. Clinical characteristics and prognosis of patients with microvascular angina: an international and prospective cohort study by the Coronary Vasomotor Disorders International Study (COVADIS) Group. *Eur Heart J*. 2021;42(44):4592-4600.
14. Beltrame JF, Crea F, Kaski JC, et al. International standardization of diagnostic criteria for vasospastic angina. *Eur Heart J*. 2017;38(33):2565-2568.
15. Jespersen L, Hvelplund A, Abildstrøm SZ, et al. Stable angina pectoris with no obstructive coronary artery disease is associated with increased risks of major adverse cardiovascular events. *Eur Heart J*. 2012;33(6):734-744.
16. Jespersen L, Abildstrøm SZ, Hvelplund A, Prescott E. Persistent angina: highly prevalent and associated with long-term anxiety, depression, low physical functioning, and quality of life in stable angina pectoris. *Clin Res Cardiol*. 2013;102(8):571-581.
17. Global, regional, and national life expectancy, all-cause mortality, and cause-specific mortality for 249 causes of death, 1980-2015: a systematic analysis for the Global Burden of Disease Study 2015. *Lancet*. 2016;388(10053):1459-1544.
18. Knuuti J, Wijns W, Saraste A, et al. 2019 ESC guidelines for the diagnosis and management of chronic coronary syndromes. *Eur Heart J*. 2020;41(3):407-477.
19. Ong P, Camici PG, Beltrame JF, et al. International standardization of diagnostic criteria for microvascular angina. *Int J Cardiol*. 2018;250:16-20.
20. Bairey Merz CN, Shaw LJ, Reis SE, et al. Insights from the NHLBI-Sponsored Women's Ischemia Syndrome Evaluation (WISE) Study: Part II: gender differences in presentation, diagnosis, and outcome with regard to gender-based pathophysiology of atherosclerosis and macrovascular and microvascular coronary disease. *J Am Coll Cardiol*. 2006;47(3 Suppl):S21-S29.
21. Kaski JC, Aldama G, Cosín-Sales J. Cardiac syndrome X. Diagnosis, pathogenesis and management. *Am J Cardiovasc Drugs*. 2004;4(3):179-194.
22. Kleinbongard P, Heusch G. A fresh look at coronary microembolization. *Nat Rev Cardiol*. 2022;19(4):265-280.
23. Rahman H, Ryan M, Lumley M, et al. Coronary microvascular dysfunction is associated with myocardial ischemia and abnormal coronary perfusion during exercise. *Circulation*. 2019;140(22):1805-1816.
24. Ezhumalai B, Ananthakrishnapillai A, Selvaraj RJ, Satheesh S, Jayaraman B. Cardiac syndrome X: clinical characteristics revisited. *Indian Heart J*. 2015;67(4):328-331.
25. Valeriani M, Sestito A, Pera DL, et al. Abnormal cortical pain processing in patients with cardiac syndrome X. *Eur Heart J*. 2005;26(10):975-982.
26. Kunadian V, Chieffo A, Camici PG, et al. An EAPCI expert consensus document on Ischaemia with non-obstructive coronary arteries in collaboration with European Society of Cardiology Working Group on Coronary Pathophysiology & Microcirculation Endorsed by Coronary Vasomotor Disorders International Study Group. *Eur Heart J*. 2020;41(37):3504-3520.
27. Tonet E, Pompei G, Faragasso E, et al. Coronary microvascular dysfunction: PET, CMR and CT assessment. *J Clin Med*. 2021;10(9):1848.
28. Spione F, Arevalos V, Gabani R, Sabaté M, Brugaletta S. Coronary microvascular angina: a state-of-the-art review. *Front Cardiovasc Med*. 2022;9:800918.
29. Fearon WF, Kobayashi Y. Invasive assessment of the coronary microvasculature: the index of microcirculatory resistance. *Circ Cardiovasc Interv*. 2017;10(12):e005361.
30. Xaplanteris P, Fournier S, Keulards DCJ, et al. Catheter-based measurements of absolute coronary blood flow and microvascular resistance: feasibility, safety, and reproducibility in humans. *Circ Cardiovasc Interv*. 2018;11(3):e006194.
31. Dilsizian V, Bacharach SL, Beanlands RS, et al. ASNC imaging guidelines/SNMMI procedure standard for positron emission tomography (PET) nuclear cardiology procedures. *J Nucl Cardiol*. 2016;23(5):1187-1226.
32. Campisi R, Marengo FD. Coronary microvascular dysfunction in women with nonobstructive ischemic heart disease as assessed by positron emission tomography. *Cardiovasc Diagn Ther*. 2017;7(2):196-205.
33. Camici PG, Crea F. Coronary microvascular dysfunction. *N Engl J Med*. 2007;356(8):830-840.
34. Hokimoto S, Kaikita K, Yasuda S, et al. JCS/CVIT/JCC 2023 guideline focused update on diagnosis and treatment of vasospastic angina (coronary spastic angina) and coronary microvascular dysfunction. *Circ J*. 2023;87(6):879-936.
35. Khuddus MA, Pepine CJ, Handberg EM, et al. An intravascular ultrasound analysis in women experiencing chest pain in the absence of obstructive coronary artery disease: a substudy from the National Heart, Lung and Blood Institute-Sponsored Women's Ischemia Syndrome Evaluation (WISE). *J Interv Cardiol*. 2010;23(6):511-519.
36. Sucato V, Novo S, Manno G, et al. Ischemia with no obstructive coronary artery disease: microvascular angina and vasospastic angina. *G Ital Cardiol (Rome)*. 2020;21(12):954-960.
37. Ford TJ, Stanley B, Good R, et al. Stratified medical therapy using invasive coronary function testing in angina: the CorMicA trial. *J Am Coll Cardiol*. 2018;72(23 Pt A):2841-2855.
38. Smilowitz NR, Toleva O, Chieffo A, Perera D, Berry C. Coronary microvascular disease in contemporary clinical practice. *Circ Cardiovasc Interv*. 2023;16(6):e012568.
39. Taqueti VR, Shaw LJ, Cook NR, et al. Excess cardiovascular risk in women relative to men referred for coronary angiography is associated with severely impaired coronary flow reserve not obstructive disease. *Circulation*. 2017;135(6):566-577.
40. Kenkre TS, Malhotra P, Johnson BD, et al. Ten-year mortality in the WISE study (Women's ischemia syndrome evaluation). *Circ Cardiovasc Qual Outcomes*. 2017;10(12):e003863.
41. de Silva R, Cheng K. Microvascular angina: quo tendimus? *Eur Heart J*. 2021;42(44):4601-4604.

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