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Case Report

From "Joy" to "Ischemia": Coronary Spasm Masquerading as STEMI in a Healthy Woman: Case Report

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Abstract

Background:

Acute coronary syndrome (ACS) most often results from rupture of an atherosclerotic plaque and thrombosis. However, in some patients with normal or non-obstructive coronary arteries, transient coronary vasospasm can cause ischemia and even myocardial necrosis.

Case summary:

We report the case of a 52-year-old woman with no prior cardiac history who presented with acute chest pain and inferior ST-segment elevation following an episode of intense Joy. Coronary angiography revealed angiographically normal coronary arteries. Intracoronary acetylcholine testing provoked transient vasospasm of the right coronary artery, confirming the diagnosis of vasospastic angina. The patient was successfully treated with calcium channel blockers and nitrates, with complete symptom resolution.

Discussion:

This case highlights a rare presentation of myocardial infarction with non-obstructive coronary arteries (MINOCA) precipitated by positive emotional stress. Emotional arousal—whether negative or positive—can trigger vasospasm through sympathetic activation, endothelial dysfunction, and vascular smooth muscle hyperreactivity. Differentiating vasospastic angina from atherosclerotic myocardial infarction is essential, as treatment strategies differ significantly.

Conclusion:

Vasospasm should be suspected in ACS patients with normal coronary arteries, particularly when emotional stress precedes symptom onset. Recognition of this entity is crucial for appropriate diagnosis, management, and secondary prevention.

Keywords: vasospastic angina; coronary spasm; minoca; endothelial dysfunction; emotional stress; stemi

1.Introduction

Acute coronary syndromes (ACS) are typically caused by rupture or erosion of an atherosclerotic plaque with superimposed thrombosis, resulting in partial or complete obstruction of coronary flow [1]. However, in approximately 5–10% of cases, coronary angiography reveals no significant obstruction (defined as <50% luminal narrowing) [2]. These cases are grouped under the term myocardial infarction with non-obstructive coronary arteries (MINOCA), a heterogeneous clinical entity encompassing various pathophysiological mechanisms, including coronary vasospasm, microvascular dysfunction, spontaneous coronary artery dissection, and myocarditis [3,4].

Coronary vasospasm, or vasospastic angina (VSA), represents a dynamic, reversible narrowing of the coronary arteries caused by hypercontraction of vascular smooth muscle. When prolonged, vasospasm can result in ischemia, infarction, life-threatening arrhythmia, or sudden cardiac death [5].

Among the various triggers for vasospasm, emotional stress is particularly important. While negative emotional states such as anxiety, anger, or grief are well-established precipitants of coronary spasm [6], the role of positive emotions—so-called "joyful stress"—has only recently gained attention. The "happy heart syndrome," a positive-stress variant of Takotsubo

cardiomyopathy, has been documented, yet similar mechanisms may also precipitate coronary spasm [7].

Here, we present a case of ST-segment elevation myocardial infarction (STEMI) triggered by a Joyful emotional event, in a patient later diagnosed with coronary artery spasm. We discuss its clinical significance and underlying pathophysiological mechanisms, with emphasis on endothelial dysfunction.

2. Case Presentation

A 52-year-old woman, non-smoker, with no history of hypertension, diabetes, or dyslipidemia, presented to the emergency department with sudden-onset, intense substernal chest pain radiating to her left arm. The pain began within minutes of receiving unexpectedly Joyful family news.

On examination, the patient appeared anxious but hemodynamically stable: blood pressure 125/80 mmHg, heart rate 78 bpm, respiratory rate 18/min, and oxygen saturation 98% on room air. Cardiopulmonary examination was unremarkable.

2.1. Electrocardiogram and Laboratory Findings

The initial 12-lead ECG demonstrated ST-segment elevation in the inferior leads (II, III, aVF) with reciprocal ST depression in leads I and aVL,V1, V2 and V3. Serial ECGs over the next hour showed transient normalization of the ST segments. (Figure 1)
High-sensitivity troponin I was elevated to 0.82 ng/mL (reference < 0.04), consistent with myocardial injury. Complete blood count, renal, and hepatic panels were normal.

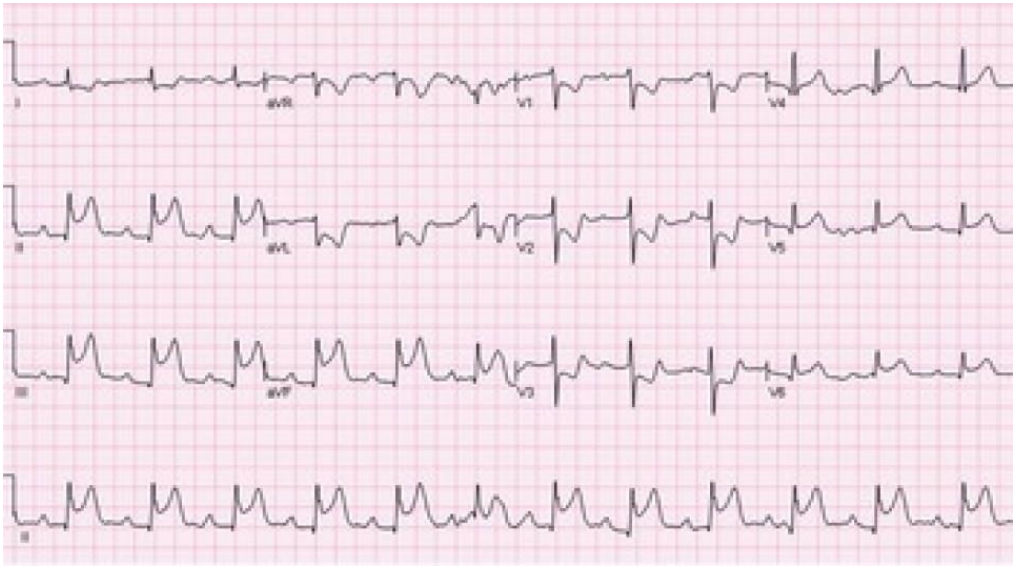

Figure 1: ECG showing ST-segment elevation in the inferior leads (II, III, aVF) with reciprocal ST depression in leads I and aVL,V1, V2 and V3

2.2. Imaging and Coronary Angiography

Given the presumed diagnosis of inferior STEMI, emergent coronary angiography was performed. Surprisingly, the initial angiogram demonstrated a total occlusion of the left anterior descending artery (LAD), which was discordant with the inferior ST-segment elevations observed on the ECG (Figure 2). After advancement of the guidewire, spontaneous restoration of coronary flow occurred (Figure 3). Ultimately, the coronary arteries appeared completely normal, with no evidence of plaque, thrombus, or dissection. Left ventricular ejection fraction was preserved (60%), with no regional wall motion abnormalities.

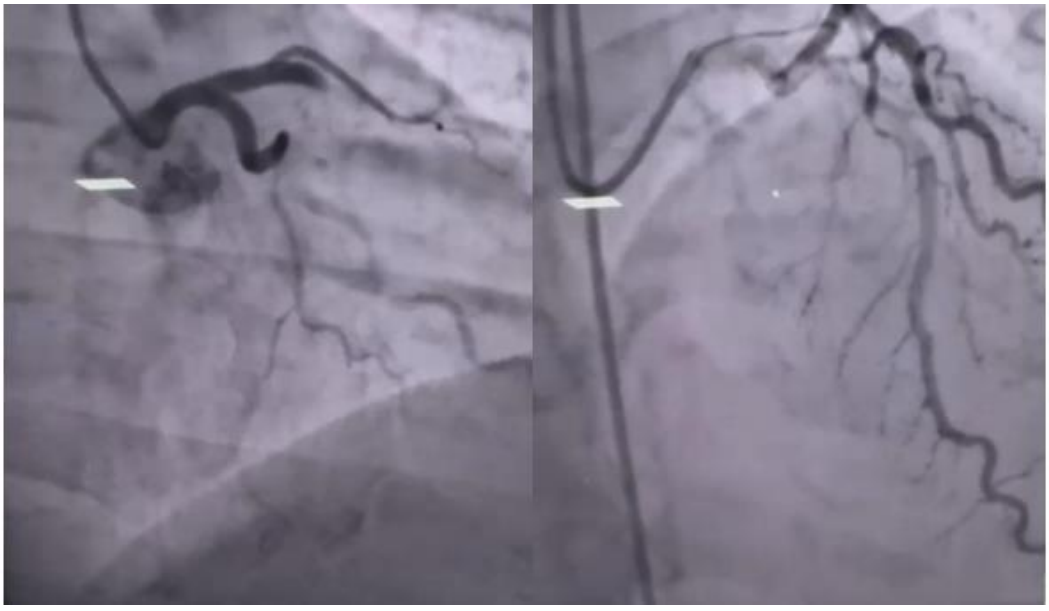

Figure 2: Figure showing a total occlusion of the left anterior descending artery (not crossable with the guidewire

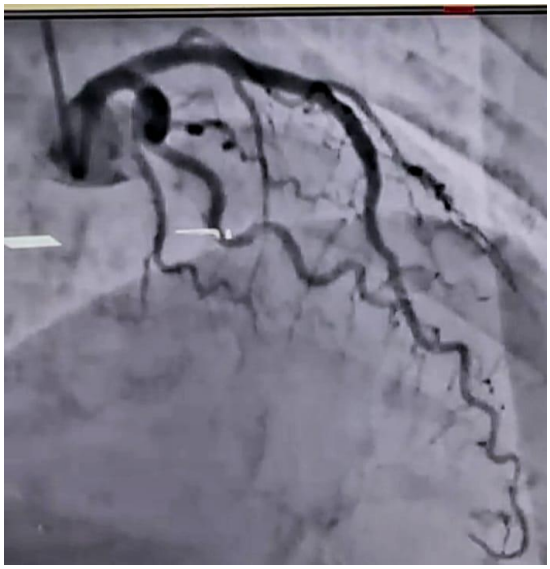

Figure 3: Coronary angiography showed arteries without significant lesions. (The occluded artery spontaneously returned to normal)

2.3. Provocative Testing and Final Diagnosis

In view of the normal angiogram, vasospastic angina was suspected. After the patient stabilized, an intracoronary acetylcholine provocation test was performed under careful monitoring. Acetylcholine injection into the right coronary artery provoked a focal, high-grade constriction accompanied by chest pain and transient ST elevation, both of which resolved promptly after intracoronary nitroglycerin administration.

A definitive diagnosis of vasospastic angina causing MINOCA was established

2.4. Management and Follow-up

The patient was initiated on oral diltiazem (120 mg daily) and long-acting nitrates. Aspirin and statins were continued for secondary prevention given the mild troponin elevation. Beta-blockers were avoided to prevent unopposed α-adrenergic vasoconstriction.

At three-month follow-up, the patient remained asymptomatic. ECG and echocardiography were normal. Repeat troponin was negative, and she reported improved stress management through relaxation therapy.

3. Discussion

3.1. Vasospastic Angina and the MINOCA Spectrum

Vasospastic angina (VSA), first described by Prinzmetal in 1959, results from transient coronary vasoconstriction leading to ischemia in the absence of fixed atherosclerotic obstruction [8]. It can occur at rest, often in the early morning hours, and may produce transient ST elevation during episodes.

VSA accounts for a significant portion of MINOCA cases. In a large European registry, invasive acetylcholine testing identified coronary vasospasm in up to 40% of patients with MINOCA [9]. This underscores the diagnostic importance of provocation testing, which remains underutilized in clinical practice.

3.2. Emotional Stress as a Trigger for Coronary Spasm

Emotional stress—negative or positive—can provoke myocardial ischemia via complex neuroendocrine mechanisms. The acute sympathetic surge releases catecholamines, augments heart rate and blood pressure, and increases myocardial oxygen demand while inducing coronary vasoconstriction through α-adrenergic receptor activation [10].

Interestingly, recent data have highlighted that "positive stress" can produce similar cardiovascular effects. The Happy Heart Syndrome described by Ghadri et al. [11] represents a form of Takotsubo cardiomyopathy precipitated by Joyful events. In our patient, a similar autonomic surge likely caused coronary vasospasm rather than transient ventricular dysfunction. Thus, "emotional extremes," regardless of valence, may perturb the delicate balance between sympathetic and parasympathetic tone, triggering coronary vasomotion abnormalities in predisposed individuals.

3.3. Pathophysiology: Endothelial Dysfunction and Smooth Muscle Hyperreactivity

The pathogenesis of coronary vasospasm involves a complex interplay between endothelial dysfunction and vascular smooth muscle (VSMC) hyper reactivity [12].

3.3.1. Endothelial Dysfunction

The endothelium maintains vascular homeostasis through the release of vasodilators such as nitric oxide (NO), prostacyclin, and endothelium-derived hyperpolarizing factor. In endothelial dysfunction, reduced NO bioavailability impairs vasodilation, while increased oxidative stress and inflammation enhance sensitivity to constrictive stimuli [13].

Risk factors such as smoking, hyperlipidemia, and inflammation exacerbate this imbalance. Moreover, subclinical endothelial dysfunction may persist in apparently healthy individuals, predisposing them to spasm under sympathetic stress [14].

3.3.2. Smooth Muscle Hyperreactivity

Even with mild endothelial dysfunction, heightened contractility of VSMCs can amplify vasospastic responses. Activation of the Rho-kinase pathway increases calcium sensitivity of contractile proteins, promoting sustained constriction independent of intracellular calcium levels [15]. Inhibiting Rho-kinase has been shown experimentally to reduce spasm severity, suggesting a potential therapeutic avenue [16].

3.3.3. Neurohumoral Modulation

Autonomic imbalance contributes significantly: excessive sympathetic activity (norepinephrine, epinephrine) or parasympathetic withdrawal can provoke spasm. Endothelin-1, serotonin, histamine, and thromboxane A_2

also act as potent vasoconstrictors released during stress or platelet activation [17]. The interplay of these mediators explains why vasospasm may occur even in angiographically normal arteries.

3.4. Diagnostic Approach

3.4.1. Coronary Angiography and Provocation Testing

In patients presenting with STEMI but normal coronary arteries, clinicians should consider vasospasm, Takotsubo cardiomyopathy, myocarditis, and microvascular dysfunction. Coronary angiography remains essential to exclude obstructive lesions.

The gold standard for diagnosing vasospasm is intracoronary acetylcholine or ergonovine testing [18]. A positive test is defined by transient ≥ 90% luminal constriction with ischemic ECG changes and chest pain, reversed by nitrates. The procedure is safe when performed in controlled settings, with complication rates below 1% [19].

3.4.2. Role of Non-Invasive Imaging

Cardiac magnetic resonance imaging (MRI) provides valuable complementary data, differentiating ischemic necrosis from myocarditis or Takotsubo patterns [20]. Absence of wall motion abnormalities in our patient supported isolated vasospasm rather than stress cardiomyopathy.

3.5. Treatment and Prognosis

3.5.1. Acute Management

During acute attacks, nitrates are the mainstay for rapid vasodilation. Calcium channel blockers (CCBs) should be initiated promptly to prevent recurrence. Beta-blockers, particularly non-selective agents, may worsen vasospasm by blocking β2-mediated vasodilation and leaving α-adrenergic constriction unopposed [21].

3.5.2. Chronic Management

Chronic therapy includes long-acting CCBs (diltiazem, amlodipine, or verapamil) and nitrates. In refractory cases, addition of nicorandil or Rho-kinase inhibitors may be considered [22].

Modifiable risk factors—smoking cessation, lipid control, and stress reduction—are crucial. Statins and ACE inhibitors improve endothelial function and may lower recurrence risk [23].

3.5.3. Prognosis

When appropriately treated, long-term prognosis is favorable, with annual mortality rates below 1% [24]. However, untreated or misdiagnosed vasospasm carries risks of recurrent Ischemia, arrhythmia, and sudden death. Early recognition and patient education are therefore essential.

3.6. Differential Diagnosis: Vasospasm vs. Takotsubo Syndrome

Although both entities can be triggered by emotional stress, key distinctions exist. Takotsubo syndrome primarily involves transient left ventricular systolic dysfunction (apical ballooning) without coronary obstruction, often confirmed by characteristic imaging findings [25]. In contrast, vasospasm produces localized ischemia with angiographically normal arteries and normal ventricular function.

Both conditions, however, share common pathophysiological pathways—sympathetic surge, endothelial dysfunction, and catecholamine toxicity—suggesting overlapping mechanisms within the “stress cardiomyopathy spectrum” [26].

3.7. Clinical Implications and Future Directions

This case emphasizes the importance of considering vasospasm in patients with chest pain and non-obstructive coronaries, especially when emotional stress precedes symptom onset. Provocative testing should be integrated into diagnostic pathways for MINOCA when available.

Future research should focus on:

• Biomarkers of endothelial dysfunction and vasospastic predisposition (e.g., endothelin-1, asymmetric dimethylarginine).

• Role of autonomic modulation therapies (biofeedback, stress reduction).

• Potential benefit of Rho-kinase inhibitors and antioxidant strategies in refractory cases.

4. Conclusion

ST-elevation myocardial infarction caused by coronary vasospasm may closely mimic atherosclerotic STEMI, even when triggered by positive emotional stimuli. Recognition of vasospasm as a potential etiology in MINOCA is essential for appropriate therapy. Endothelial dysfunction provides the substrate, while autonomic and neurohumoral factors serve as triggers.

Awareness of emotional stress—whether negative or Joyful—as a cardiovascular risk factor underscores the need for a holistic approach to cardiac health encompassing psychological well-being and vascular function.

References

1. Thygesen K, Alpert JS, Jaffe AS, et al. Fourth universal definition of myocardial infarction (2019). *Eur Heart J.* 0(3):237–269.
2. Tamis-Holland JE, Jneid H, Reynolds HR, et al. (2019). Contemporary diagnosis and management of patients with myocardial infarction in the absence of obstructive coronary artery disease. Circulation. 139:e891–e908.
3. Pasupathy S, Air T, Dreyer RP, et al. (2015). Systematic review of patients presenting with suspected myocardial infarction and non-obstructive coronary arteries. Circulation. 131(10):861–870.
4. Niccoli G, Scalone G, Crea F. (2015). Pathophysiology of acute coronary syndromes without coronary plaque rupture. *Nat Rev Cardiol.* 12(12):629–641.
5. Beltrame JF, Crea F, Kaski JC, et al. (2017). International standardization of diagnostic criteria for vasospastic angina. *Eur Heart J.* 38(33):2565–2568.
6. Vaccarino V, Shah AJ, Rooks C, et al. (2014). Sex differences in mental stress–induced myocardial ischemia in young survivors of acute myocardial infarction. *Psychosom Med.* 76(3):171–180.
7. Ghadri JR, Sarcon A, Diekmann J, et al. (2016). Happy heart syndrome: role of positive emotional stress in Takotsubo syndrome. *Eur Heart J.* 37(37):2823–2829.
8. Prinzmetal M, Kennamer R, Merliss R, et al. (1959). Angina pectoris. I. A variant form of angina pectoris. Am J Med. 27(3):375–388.
9. Montone RA, Niccoli G, Fracassi F, et al. (2018). Patients with acute myocardial infarction and non-obstructive coronary arteries: safety and prognostic relevance of invasive coronary provocative tests. *Eur Heart J.* 39(2):91–98.
10. Steptoe A, Kivimäki M. (2012). Stress and cardiovascular disease. Nat Rev Cardiol. 9(6):360–370.
11. Ghadri JR, Wittstein IS, Prasad A, et al. International expert consensus document on Takotsubo syndrome. Eur Heart J. 2018;39(22):2032–2046.
12. Lanza GA, Crea F. (2010). Primary coronary microvascular dysfunction: pathophysiology and management. Circulation. 121(21):2317–2325.
13. Sato K, Kaikita K, Nakayama N, et al. (2019). Coronary spasm and endothelial dysfunction: recent insights. J Cardiol. 74(5):361–369.

14. Hung MJ, Hu P, Hung MY. (2014). Coronary artery spasm: review and update. *Int J Med Sci.;*11(11):1161–1171.
15. Shimokawa H, Satoh K. (2014). Rho-kinase in the pathogenesis of coronary artery spasm and atherosclerosis. *J Mol Cell Cardiol*. 77:91–98.
16. Masumoto A, Mohri M, Shimokawa H. (2002). Suppression of coronary artery spasm by Rho-kinase inhibitor fasudil in patients with vasospastic angina. Circulation. 105(13):1545–1547.
17. Okumura K, Yasue H, Matsuyama K, et al. (1996). Diffuse disorder of coronary artery vasomotility in patients with coronary spastic angina. *J Am Coll Cardiol.* 27(1):45–52.
18. Ong P, Athanasiadis A, Sechtem U. (2013). Pharmacologically induced coronary spasm: critical role of acetylcholine testing. Heart. 99(15):1228–1233.
19. Ford TJ, Ong P, Sechtem U, et al. (2020). Assessment of vascular dysfunction in patients without obstructive coronary artery disease. *JACC Cardiovasc Interv.* 13(16):1847–1864.
20. Dastidar AG, Rodrigues JCL, Johnson TW, et al. (2017).Myocardial infarction with nonobstructed coronary arteries: CMR findings. *JACC Cardiovasc Imaging.* 10(11):1204–1206.
21. Yasue H, Mizuno Y, Harada E, et al. (2008). Coronary artery spasm—clinical features and treatment. *J Cardiol.* 51(1):2–17.
22. Takagi Y, Takahashi J, Yasuda S, et al. (2013). Prognostic stratification of patients with vasospastic angina. *J Am Coll Cardiol.*

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