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Paradoxical Coronary Artery Embolism via Patent Foramen Ovale in Acute Myocardial Infarction: A Narrative Mini Review of the Cardio-Neurological Interface

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PFO Closure
Coronary Angiography

ABSTRACT

Background: Paradoxical coronary artery embolism (PCAE) via a Patent Foramen Ovale (PFO) with a Right-to-Left Shunt (RLS) is an underrecognized cause of myocardial infarction with nonobstructive coronary arteries (MINOCA). Because it is a systemic embolic process, PCAE is frequently associated with cryptogenic neurological events. This review synthesizes current evidence to provide an operational diagnostic framework linking neurovascular and cardioembolic pathways.

Methods: A comprehensive, reproducible literature search of PubMed/MEDLINE was performed from January 2000 through June 2026 using validated Medical Subject Headings (MeSH) and precisely nested Boolean text strings to capture the intersection of paradoxical embolism, interatrial shunts, and myocardial infarction. Given the scarcity of large clinical trials, the eligibility criteria were expanded to include case series and individual case reports.

Key Findings: Diagnosing PCAE requires sequential multi-modality evaluations: invasive angiography to document nonobstructive disease, intracoronary imaging (Optical Coherence Tomography [OCT] or Intravascular Ultrasound [IVUS]) to exclude plaque dynamics, cardiac magnetic resonance imaging (CMRI) to confirm ischemic tissue distribution, and contrast-enhanced shunt evaluations via transesophageal echocardiography (c-TEE) or transcranial Doppler (c-TCD). Management requires balancing acute antiplatelet therapy with individualized, long-term secondary anticoagulation or structural PFO device closure.

Conclusions: Because recommendations for PFO closure in coronary presentations are largely extrapolated from ischemic stroke guidelines and expert consensus, close coordination within a dedicated multidisciplinary Heart-Brain Team is essential to optimize patient selection and prevent recurrent systemic embolic events across multiple vascular territories.

Introduction

Myocardial infarction with nonobstructive coronary arteries (MINOCA) encompasses a heterogeneous group of acute ischemic syndromes occurring in the absence of flow-limiting epicardial coronary artery disease (defined as less than 50% luminal stenosis) on invasive coronary angiography. While microvascular dysfunction and localized vasospasm are common etiologies, coronary artery embolism represents an important nonatherosclerotic mechanism. Specifically, paradoxical coronary artery embolism (PCAE), traveling via a Patent Foramen Ovale (PFO) with a Right-to-Left Shunt (RLS), remains an underrecognized clinical entity.

The objective of this narrative Mini Review is to critically appraise the current literature, clarify the underlying pathophysiology, and establish a clear, consolidated diagnostic and therapeutic workflow for identifying and managing PCAE while avoiding diagnostic over-attribution. Building directly upon our recently reported case series of two patients presenting with acute myocardial infarction (AMI) and angiographically normal coronary vessels[1], this review integrates evidence from both the cardiovascular and cerebrovascular literature to provide an operational, multi-modality framework for interdisciplinary Heart-Brain Clinical Teams.

Literature Search Methodology

To ensure transparency, reproducibility, and comprehensive database coverage, a systematic search strategy was executed in PubMed/MEDLINE for articles published from January 2000 through June 2026[2-40]. To correct previous Boolean precedence errors and optimize vocabulary sensitivity, the search utilized explicitly parenthesized string clusters combining Medical Subject Headings (MeSH) and expanded free-text keywords:

1. **String 1. Embolic Mechanism:** ("Embolism, Paradoxical"[MeSH] OR "paradoxical coronary embolism" OR "PCAE" OR "coronary artery embolism")
2. **String 2. Anatomical Shunt:** AND ("Foramen Ovale, Patent"[MeSH] OR "patent foramen ovale" OR "PFO" OR "Right-to-Left Shunt"[MeSH] OR "right-to-left shunt" OR "RLS")
3. **String 3. Ischemic Presentation:** AND ("Myocardial Infarction"[MeSH] OR "myocardial infarction" OR "acute myocardial infarction" OR "AMI" OR "MINOCA" OR "Stroke"[MeSH] OR "cryptogenic stroke" OR "embolic stroke of undetermined source" OR "ESUS")

The three strings were integrated using strict intersection logic (String 1 AND String 2 AND String 3). To capture the complete evidence base of this relatively rare pathological entity, literature eligibility was expanded beyond clinical trials and cohort studies to encompass case series, expert consensus statements, and detailed individual case reports. Only peer-reviewed, English-language articles were included in the final narrative synthesis.

Epidemiology and Structural Definitions

To prevent diagnostic over-attribution, clinicians must distinguish between nested diagnostic classifications within the acute coronary syndrome (ACS) spectrum:

- **MINOCA Prevalence:** MINOCA is identified in approximately 2% to 10% of all AMI presentations. This baseline variance is driven by historical differences in diagnostic definitions, institutional usage of early cardiac MRI, and standard angiography thresholds.
- **General Coronary Embolism:** Embolic mechanisms account for roughly 4% to 13% of the broader MINOCA cohort. In unselected general AMI populations, coronary embolism represents a smaller fraction (~3%).
- **PFO-Mediated PCAE:** Robust epidemiological quantification of true PFO-mediated PCAE is currently constrained by a paucity of randomized trial data specifically isolating this narrow pathological subset. Its true incidence remains underestimated because transient venous thrombi frequently undergo spontaneous endogenous lysis before definitive angiography can be performed.

High-risk clinical demographics driving suspicion for PCAE include younger patients (under 60 years old), individuals with low conventional cardiovascular atherosclerotic risk metrics, active prothrombotic conditions (such as systemic hormone therapy or inherited hypercoagulability), or angiographic evidence showing isolated filling defects or abrupt distal cutoffs in otherwise smooth epicardial vessels.

Pathophysiology and Hemodynamic Mechanisms

PCAE requires an interrelated triad: a venous thromboembolic source, an anatomical interatrial communication (typically a PFO), and a transient or chronic right-to-left pressure gradient acting as the embolic trigger.

Hemodynamic Shunt Dynamics

The passage of a venous thrombus into the systemic arterial circulation depends directly on transient right-to-left pressure gradients. Under normal resting physiological conditions, left atrial pressure exceeds right atrial pressure by 1 to 5 mmHg, with an average difference of about 3 mmHg, keeping the PFO primitive flap closed against the interatrial septum.

However, brief reversals of this gradient occur during Valsalva-like maneuvers (e.g., severe coughing, straining during defecation, heavy lifting, or vomiting). These maneuvers abruptly increase intra-abdominal and intrathoracic pressure. Upon relaxation (Phase IV of the Valsalva maneuver), right atrial venous return rises, transiently elevating right atrial pressure above left atrial pressure, separating the septum primum from the septum secundum, and permitting embolic transit during this transient pressure overshoot.

Mechanical Thrombus Clot Dynamics

Thrombi may be quite small or break apart during interatrial transit, leading to micro-embolic showers in the coronary bed. Crucially, histological insights from neuro-interventional thrombectomy specimens demonstrate that cardioembolic and paradoxical thrombi are structurally fibrin-rich and platelet-poor. This contrasts sharply with the platelet-rich "white clots" seen in typical atherothrombotic plaque ruptures. This composition renders paradoxical emboli highly cohesive and uniquely resistant to standard manual aspiration catheters or conventional fibrinolytic regimens.

Concomitant Coronary Vasospasm Link

An alternative or coexisting mechanism involves localized epicardial coronary vasospasm provoked by vasoactive substances. Venous thrombi are rich in bioactive agents like serotonin and thromboxane A_2 (TXA_2). When a thrombus bypasses the pulmonary capillary bed—where these substances are normally cleared or metabolized—via an active right-to-left shunt, high concentrations are delivered directly into the coronary circulation. This can trigger intense, localized smooth muscle constriction at the embolic impact site, worsening the acute myocardial ischemia even if the thrombus undergoes subsequent rapid spontaneous lysis.

Establishing Causality: PFO/PCAE Tiers

Because PFO is common in the general population (approximately 25%), its presence alone does not prove causality in patients with MINOCA. To support clinical decision-making, we propose a standardized framework for grading the likelihood of PFO-mediated PCAE:

1. **Definite PCAE:** Patient meets all MINOCA criteria, has a confirmed PFO/RLS, and has visual proof of a thrombus caught straddling the PFO or documented simultaneous systemic venous (DVT/PE) and arterial (coronary/cerebral) embolic events.
2. **Probable PCAE:** Patient meets MINOCA criteria with an embolic angiographic profile (abrupt distal cutoff), a documented high-risk PFO/RLS, an absence of alternative MINOCA etiologies, and a clear history of a preceding Valsalva maneuver or underlying hypercoagulability.
3. **Suspected PCAE:** Patient meets MINOCA criteria, has a PFO detected during routine workup, but lacks high-risk anatomical features, an embolic angiographic pattern, or a history of a preceding Valsalva maneuver, leaving the PFO as a possible incidental finding.

Differential Diagnosis of MINOCA

A critical clinical challenge is avoiding the premature attribution of an AMI to a PFO simply because an interatrial shunt is present. A thorough diagnostic protocol must differentiate PCAE from other frequent MINOCA drivers (Table 1).

Neurological Relevance & Interdisciplinary Coordination

The clinical spectrum of PCAE directly intersects with the diagnostic workup for Cryptogenic Stroke and Embolic Stroke of Undetermined Source (ESUS). Because an anatomical PFO provides systemic arterial entry, a single paradoxical embolic event can seed the cerebral and coronary trees simultaneously or sequentially.

Overlapping Neuro-Cardiologic Workup

Any patient presenting with an embolic-appearing AMI and nonobstructive coronary arteries should be screened using the same diagnostic intensity applied to a cryptogenic stroke cohort. This includes brain MRI/MRA to rule out clinically silent cerebral infarcts.

Conversely, neurologists should request a comprehensive cardiology evaluation and specialized cardiac imaging (such as CMRI) for PFO-positive patients who exhibit atypical chest pain, unexplained electrocardiographic changes, or biomarker elevations following a stroke. This approach helps rule out concurrent, silent myocardial infarctions.

When concurrent or sequential cerebro-cardiac embolic events occur, immediate coordination within a dedicated Heart-Brain Team—comprising stroke neurologists, interventional cardiologists, non-invasive imaging specialists, and hematologists—is mandatory. Neurologists bring vital expertise in evaluating shunt-related neurological risks, quantifying overall embolic burden via clinical tools like the Risk of Paradoxical Embolism (RoPE) score, and identifying high-risk septal structures.

Cardiologists provide endovascular options and structural closure strategies. Joint decision-making ensures that secondary prevention plans balance long-term systemic bleeding risks against the danger of recurrent events across both vascular beds.

Operational Diagnostic Workflow

To establish a diagnosis of PCAE while systematically ruling out mimics, clinicians should follow a structured, stepwise approach (Figure 1. Part I):

1. **Emergent Coronary Angiography & Intracoronary Imaging:** perform urgent angiography to exclude obstructive atherosclerosis. When findings are

Table 1: Differential Diagnostic Clues in MINOCA

Underlying Mechanism	Key Demographic & Clinical Clues	Preferred Imaging / Diagnostic Modality	Diagnostic Hallmarks
Paradoxical Coronary Embolism (PCAE)	Young age (<60 years) Low atherosclerotic risk Valsalva strain pre-infarct Prior VTE or stroke history	Coronary Angiography c-TEE / c-TCD Cardiac MRI (CMR)	Distal vessel cutoff Right-to-left shunt with high-risk PFO features Subendocardial or transmural LGE pattern
Occult Plaque Disruption	Traditional CV risk factors (smoking, dyslipidemia, HTN)	Intracoronary OCT Intravascular Ultrasound (IVUS)	Intramural hematoma Lipid-rich plaque rupture Superficial erosion (not seen on standard angiogram)
Coronary Vasospasm	Recurrent chest pain at rest (often nocturnal) Tobacco use Prompt response to nitrates	Provocative testing (intracoronary acetylcholine or ergonovine)	Transient epicardial constriction (≥ 90%) Reproduction of clinical symptoms Ischemic ECG shifts
Spontaneous Coronary Artery Dissection (SCAD)	Female predominance Peripartum period History of FMD Physical or emotional stressors	Coronary Angiography Intracoronary OCT / IVUS	Radiolucent false lumen Abrupt vessel narrowing (Type 2 SCAD) Intramural hematoma visualization
Coronary Microvascular Dysfunction (CMD)	Postmenopausal females Anginal symptoms with objective ischemia Normal epicardial arteries	Invasive coronary physiology testing (CFR, IMR) Positron Emission Tomography (PET)	Coronary Flow Reserve (CFR) <2.0 Abnormal Index of Microcirculatory Resistance (IMR)
Myocarditis *(MINOCA Mimic)*	Recent viral prodrome or fever Pleuritic chest pain Elevated inflammatory markers	Cardiac MRI (CMR) with LGE profiling	Non-ischemic injury pattern Patchy, mid-wall, or subepicardial LGE Subendocardial sparing
Takotsubo Syndrome *(MINOCA Mimic)*	Profound emotional or physical stressor Postmenopausal females	Echocardiography Cardiac MRI (CMR)	Transient apical ballooning Mid-ventricular dyskinesis Absence of corresponding LGE distribution

Abbreviations: MINOCA, Myocardial Infarction with Non-Obstructive Coronary Arteries; CFR, Coronary Flow Reserve; MRI, Magnetic Resonance Imaging; c-TEE, contrast Transesophageal Echocardiography; c-TCD, Contrast-Enhanced Transcranial Doppler; CV, Cardiovascular; ECG, Electrocardiogram; HTN, Hypertension; IMR, Index of Microcirculatory Resistance; LGE, Late Gadolinium Enhancement; OCT, Optical Coherence Tomography; PFO, Patent Foramen Ovale; VTE, Venous Thromboembolism; Fibromuscular Dysplasia, FMD

ambiguous or SCAD/plaque erosion is suspected, perform OCT/IVUS to verify vessel wall integrity and rule out hidden mechanical plaque dynamics.

2. **CMRI:** schedule CMRI early (ideally within 2 to 7 days of admission). Evaluation via Late Gadolinium Enhancement (LGE) is essential to confirm an ischemic subendocardial or transmural pattern of myocardial injury, differentiating it from the non-ischemic mid-wall or subepicardial patterns characteristic of myocarditis or Takotsubo cardiomyopathy.

3. **Shunt Screening & Quantification:** perform an agitated-saline contrast bubble study via contrast-Transthoracic Echocardiography (cTTE), c-TEE, or c-TCD. To maximize sensitivity, these studies must include standardized, calibrated Valsalva maneuvers to reliably trigger transient right-to-left shunting. TEE is the gold standard for defining PFO tissue anatomy, including tunnel length, hypermobile components, and the presence of an Atrial Septal Aneurysm (ASA) defined by tissue excursion of 10 mm or more.

4. **Venous Thromboembolism (VTE) Surveillance:** conduct lower extremity venous duplex ultrasonography and pelvic magnetic resonance venography when clinically indicated. Importantly, failing to detect a deep vein thrombosis (DVT) does not exclude PCAE, as the entire thrombus may have embolized before evaluation. Conversely, detecting a DVT confirms a venous source but does not automatically prove a causal link to the AMI without structural shunt confirmation.

5. **Thrombophilia & Rhythm Monitoring:** order a targeted hypercoagulability panel : Antiphospholipid Syndrome [Lupus Anticoagulant, Anticardiolipin

antibodies, and Beta-2-Glycoprotein I antibodies], Factor V Leiden mutation, Prothrombin (Factor II) G20210A Mutation, Protein C/S and Antithrombin III functional activity. To definitively exclude occult atrial fibrillation (AF), implement prolonged ambulatory electrocardiographic monitoring (minimum 14-day patch or an insertable cardiac monitor).

Graded Therapeutic Management Matrix

Therapeutic strategies must be divided into distinct clinical phases to manage acute ischemia and mitigate long-term recurrence risks, using graded recommendations adapted from expert consensus and stroke prevention guidelines (Figure 1. Part II):

Acute Ischemic Phase

- Follow standard acute coronary syndrome guidelines, including dual antiplatelet therapy (DAPT) and weight-adjusted heparinization (Class I, Level C).
- If a mobile, flow-limiting embolus is angiographically visible, mechanical options like manual thrombus aspiration or microcatheter techniques may be attempted (Class IIb, Level C). Clinicians must remain mindful of the technical challenges posed by distal, friable, and highly cohesive fibrin-rich clots.

Secondary Prevention Strategy

Long-term antithrombotic and structural management pathways diverge based on the patient's individual clinical profile:

- **Documented VTE or High-Risk Thrombophilia:** initiate long-term therapeutic anticoagulation (using direct oral anticoagulants

Figure 1. Consolidated Operational Diagnostic Workflow and Graded Therapeutic Management Matrix.

Part I — Diagnostic adjudication

Clinical entry point: suspected MINOCA, defined as acute myocardial infarction with angiographically non-obstructive coronary arteries.

1. **Coronary definition:** confirm <50% stenosis and use OCT/IVUS when needed to exclude plaque rupture, plaque erosion, intramural hematoma, or SCAD.
2. **Myocardial tissue characterization:** perform early cardiac MRI, preferably within 2–7 days, to confirm ischemic injury and exclude myocarditis or Takotsubo syndrome.
3. **Interatrial shunt assessment:** evaluate right-to-left shunt burden and high-risk PFO anatomy using contrast TTE/TEE or transcranial Doppler with standardized Valsalva maneuver.
4. **Embolic source and competing-mechanism screen:** assess venous thromboembolism, thrombophilia, pelvic venous sources when indicated, and occult atrial fibrillation.

Multidisciplinary adjudication: classify causality as definite, probable, or suspected PCAE after integration of coronary, neurovascular, and hematologic findings.

Part II — Therapeutic management after multidisciplinary assessment

Acute ischemic phase	**Pathway A** Documented VTE or high-risk thrombophilia	**Pathway B** High-risk PFO plus recurrent systemic embolic events	**Pathway C** Isolated PCAE without VTE or high-risk anatomy
- standard ACS antiplatelet therapy; - consider weight-adjusted heparinization when clinically appropriate; - avoid routine manual clot aspiration unless justified.	- long-term oral anticoagulation, using DOAC or VKA according to clinical context, indication, and contraindications.	- consider percutaneous PFO closure plus short-term DAPT after individualized multidisciplinary risk–benefit assessment.	Individualize SAPT versus OAC according to recurrent embolic risk, bleeding risk, and Heart–Brain Team consensus.

Figure 1:

[DOACs] or vitamin K antagonists) as the primary treatment (Class I, Level B).

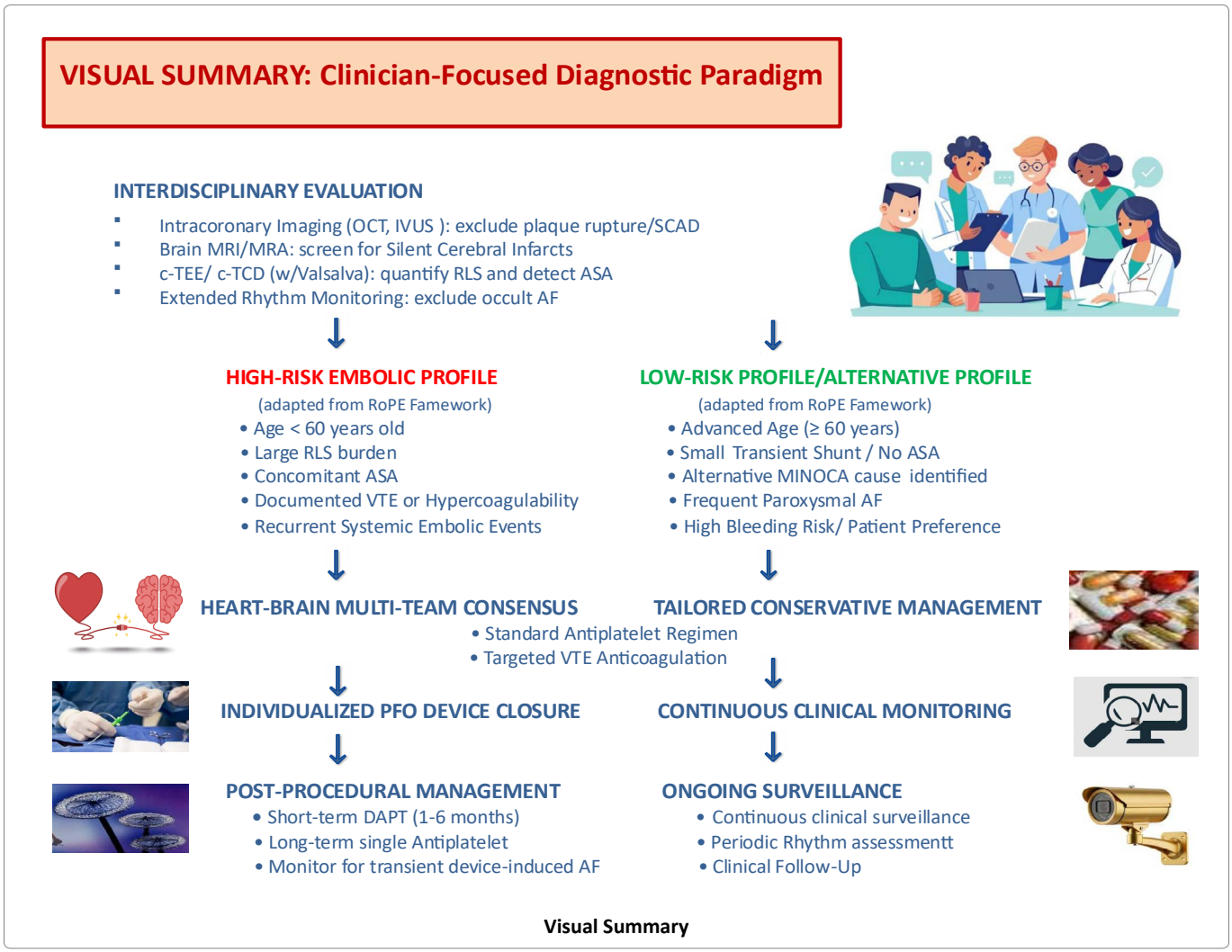
- **Absence of Documented VTE:** significant clinical uncertainty persists regarding the optimal duration and composition of antithrombotic therapy when a venous source cannot be confirmed. In these cases, decisions regarding prolonged anticoagulation versus antiplatelet therapy must be tailored by the Heart-Brain Team, factoring in individual bleeding risks (Class IIa, Level C).
- **Post-PFO Closure Regimen:** following percutaneous structural device closure, standard protocols generally require short-term DAPT (typically 1 to 6 months), followed by long-term single antiplatelet therapy (SAPT) (Class I, Level C). Clinicians should conduct short-term post-procedural monitoring to screen for transient, device-induced atrial fibrillation.

Patient Selection for Percutaneous PFO Closure

A critical limitation of current clinical practice is that recommendations regarding percutaneous PFO closure for isolated coronary endpoints are largely extrapolated from ischemic stroke literature and expert consensus opinion. While multiple large-scale randomized controlled trials (RCTs)[40-43] confirm that PFO closure reduces recurrence risks in younger patients with cryptogenic stroke, no dedicated RCTs exist for isolated PCAE.

Consequently, structural device closure should not be applied as a generalized, routine treatment pathway for all PFO-positive MINOCA patients. Instead, closure should be reserved for cases meeting strict risk-stratification parameters evaluated by the Heart-Brain Team:

- **High-Risk Anatomical Features:** Presence of ASA, hypermobile interatrial septum, a large resting right-to-left shunt (≥ 20microbubbles) appearing in the left atrium within 3 cardiac cycles), or a wide PFO tunnel diameter (≥ 2 mm).
- **Clinical Risk Frameworks:** Utilization of clinical scoring frameworks like the Risk of Paradoxical Embolism (RoPE) score to calculate the probability that the PFO is causally related to the embolic event



Visual Summary

rather than an incidental finding[44]. High RoPE scores (typically ≥ 7) identify younger patients with minimal conventional cardiovascular risk factors, indicating a higher probability of PFO-mediated causality.

Key Take-Home Points

1. **Maintain Clinical Vigilance:** suspect paradoxical coronary embolism in patients presenting with an AMI and nonobstructive coronary arteries, particularly younger individuals with low conventional cardiovascular risk factor profiles.
2. **Implement Multimodality Screening:** combine early diagnostic angiography and intracoronary imaging (OCT/IVUS) with subendocardial late gadolinium enhancement (LGE) profiling via CMRI, targeted venous imaging, and contrast-enhanced shunt studies (c-TEE/c-TCD).
3. **Acknowledge Evidence Limitations:** in the absence of dedicated RCTs evaluating PFO closure for coronary pathologies, contemporary clinical recommendations are inherently extrapolated from cryptogenic stroke data; consequently, standardized closure algorithms must be avoided in favor of highly individualized, multidisciplinary risk-benefit assessments.
4. **Engage the Heart-Brain Team:** centralize long-term antithrombotic choices and patient selection for percutaneous device closure within a formal, multidisciplinary framework involving stroke neurologists and cardiologists to minimize total systemic embolic risks.

Data Availability Statement

The original contributions presented in the study are included in the article/Supplementary Material, further inquiries can be directed to the corresponding author.

Ethics Statement

Written informed consent was obtained from the individual for the publication of any potentially identifiable images or data included in this article.

Author's Note

This article was the original work of the authors who have all seen and approved the paper and authorship. The article has not been published elsewhere and is not under consideration in any other journals.

Author Contributions

All authors listed have made a substantial, direct, and intellectual contribution to the work and approved it for publication.

Conflict of Interest

The authors declare no conflict of interest.

References

1. Onorato EM, Grancini L, Casilli F, et al. Myocardial Infarction with Normal Coronary Arteries in Two Patients with Patent Foramen Ovale. JACC Case Rep. 2026; 28(12): 107870.
2. Travis JA, Fuller SB, Ligush J, et al. Diagnosis and treatment of paradoxical embolus. Journal of Vascular Surgery. 2001; 34(5): 860-865.
3. Dubiel M, Bruch L, Liebner M, et al. Exclusion of patients with arteriosclerosis reduces long-term recurrence rate of presumed arterial embolism after PFO closure. J Interv Cardiol. 2007; 20: 275-281.
4. Stortecky S, Cook S, Meier B, et al. Patent foramen ovale: a culpable pathway for myocardial infarction. J Am Coll Cardiol. 2011; 58(18): 1923.
5. Calvert PA, Rana BS, Kydd AC, et al. Patent foramen ovale: Anatomy, outcomes, and closure. Heart. 2011; 97: 812-818.
6. Dao CN, Tobis JM. PFO and paradoxical embolism producing events other than stroke. Catheter Cardiovasc Interv. 2011; 77: 903-909.
7. Beltrame JF. Assessing patients with myocardial infarction and nonobstructed coronary arteries (MINOCA). J Int Med. 2013; 273(3): 182-185.
8. Windecker S, Stortecky S, Meier B. Paradoxical embolism. J Am Coll Cardiol. 2014; 64: 403-415.
9. Capodanno D, Milazzo G, Vitale L, et al. Updating the evidence on patent foramen ovale closure. EuroIntervention. 2014; 9(11): 1342-1349.
10. Shibata T, Kawakami S, Noguchi T, et al. Prevalence, clinical features, and prognosis of acute myocardial infarction attributable to coronary artery embolism. Circulation. 2015; 132(4): 241-250.
11. Neisius U, Northridge DB, Cruden NL, et al. Myocardial infarction associated with patent foramen ovale and paradoxical embolism: a case series. Int J Cardiol. 2015; 180: 34-37.
12. Shibata T, Kawakami S, Noguchi T, et al. Prevalence, clinical features, and prognosis of acute myocardial infarction attributable to coronary artery embolism. Circulation. 2015; 132: 241-250.
13. Tukaye DN, Chandra A, Mowery A, et al. Paradoxical thromboembolism and ST-elevation myocardial infarction. Case Rep Cardiol. 2017; 7058578.
14. Sporns PB, Hanning U, Schwindt W, et al. Ischemic stroke: what does the histological composition tell us about the origin of the thrombus? Stroke. 2017; 48(8): 2206-2210.
15. Popovic B, Agrinier N, Bouchahda N, et al. Coronary embolism among ST-segment-elevation myocardial infarction patients: Mechanisms and management. Circ Cardiovasc Interv. 2018; 11: e00558.
16. Raphael CE, Heit JA, Reeder GS, et al. Coronary embolus: An underappreciated cause of acute coronary syndromes. JACC Cardiovasc Interv. 2018; 1: 172-180.
17. Alkhouli M, Busu T, Shah K, et al. Incidence and outcomes of patent foramen ovale closure in contemporary practice. JACC Cardiovasc Interv. 2018; 11: 727-37.
18. Raphael CE, Heit JA, Reeder GS, et al. Coronary embolus: an underappreciated cause of acute coronary syndromes. JACC Cardiovasc Interv. 2018; 11: 172-180.
19. Pristipino C, Sievert H, D'Ascenzo F, et al. European position paper on the management of patients with patent foramen ovale. Eur Heart J. 2019;14:1389-1402.

20. Kumar P, Tobis JM. The multiple clinical manifestations of patent foramen ovale. Structural Heart. 2020; 4(3): 159-168.

21. Boumaaz M, Moutaouakil F, Rachdi S, et al. Myocardial infarction caused by a proven paradoxical coronary embolism through a patent foramen ovale. Pan Afr Med J. 2020; 37: 198.

22. Tarantini G, D'Amico G, Baracchini C, et al. Documento di posizione della Società Italiana di Cardiologia Interventistica (SICI-GISE): Gestione del forame ovale pervio in presenza di tromboembolia cerebrale o sistemica criptogenetica – versione 2020. G Ital Cardiol. 2020; 21(4 Suppl. 2): 50S-59S.

23. Collet JP, Thiele H, Barbato E, et al. 2020 ESC Guidelines for the management of acute coronary syndromes. Eur Heart J. 2021; 42: 1289-1367.

24. Pristipino C, Germonpré P, Toni D, et al. European position paper on the management of patients with patent foramen ovale. Part II - Decompression sickness, migraine, arterial deoxygenation. Eur Heart J. 2021; 42(16): 1545-1553.

25. Mojaddedi S, Farahani AV, Abbasi SH, et al. Patent foramen ovale-related paradoxical embolism after noncardiac surgery. J Med Cases. 2022;13:105-109.

26. Zanchin C, Ledwoch S, Bär S, et al. Acute coronary syndromes in young patients: phenotypes, causes and clinical outcomes following percutaneous coronary interventions. Int J Cardiol. 2022; 350: 1-8.

27. Ravi D, Parikh RV, Aboulhosn J, et al. A new syndrome of patent foramen ovale inducing vasospastic angina and migraine. J Am Coll Cardiol Case Rep. 2023; 28: 102132.

28. Meier C, Eisenblätter M, Gielen S. Myocardial Late Gadolinium Enhancement (LGE) in Cardiac Magnetic Resonance Imaging (CMR) An Important Risk Marker for Cardiac Disease. J Cardiovasc Dev Dis. 2024; 11(2): 40.

29. Monin A, Didier R, Leclercq T, et al. Coronary artery embolism and acute coronary syndrome: a critical appraisal of existing data. Trends Cardiovasc Med. 2024; 34: 50-56.

30. Sola M, Pozzi M, Tresoldi S, et al. Coronary Embolization After Massive Pulmonary Embolism Treated with Extracorporeal Membrane Oxygenation. Journal of Cardiothoracic and Vascular Anesthesia. 2024; 38(12): 3157-3161.

31. Wong R, Alenezi F, Altisent OAJ, et al. Paradoxical embolism leading to myocardial infarction: Revisiting the significance of patent foramen ovale. JACC Case Rep. 2025; 30: 05330.

32. Wong R, McClure T, Saleem D, et al. Paradoxical Embolism Leading to Myocardial Infarction: Revisiting the Significance of Patent Foramen Ovale. JACC Case Rep. 2025; 30(31): 105330.

33. Ibitamuno G, Ismayl M, Stanko K, et al. Conservative Acute Management of a Large Paradoxical Coronary Embolism: A Case Report and Systematic Literature Review. JACC: Case Reports. 2026; 31(1): 106208.

34. Gersony DR, Kim SH, Di Tullio M, et al. Acute myocardial infarction caused by paradoxical coronary embolization in a patient with a patent foramen ovale. J Am Soc Echocardiogr. 2001; 14(12): 1227-1229.

35. Singh S, Tawayha M, Sharma M, et al. Paradoxical Coronary Artery Embolism Through a Patent Foramen Ovale in A Young Adult. Hearts. 2026; 7(2): 12.

36. Barrios DD, Roncancio J, Avila AA, et al. Paradoxical embolism due to patent foramen ovale; a Case Report. Rev Esp Cardiol. 2017; 70: 207-209.

37. Hakim FA, Kransdorf EP, Abudiab MM, et al. Paradoxical coronary artery embolism: A rare cause of myocardial infarction. J Community Hosp Intern Med Perspect. 2014; 4: 24290

38. Boumaaz M, Moutaouakil F, Rachdi S, et al. Myocardial infarction caused by a proven paradoxical coronary embolism through a patent foramen ovale. Pan Afr Med J. 2020; 37: 198.

39. Pałuchowski M, Jakubik O, Tyślerowicz P, et al. Paradoxical embolism via patent foramen ovale: a rare cause of myocardial infarction in an air traveler. Cardiology Journal. 2025; 32(3): 332-333.

40. Saver JL, Carroll JD, Thaler DE, et al. RESPECT Investigators. Long-term out-comes of patent foramen ovale closure or medical therapy after stroke. N Engl J Med. 2017; 377: 1022-1032.

41. Søndergaard L, Kasner SE, Rhodes JF, et al. REDUCE Clinical Study Investigators. Patent foramen ovale closure or antiplatelet therapy for cryptogenic stroke. N Engl J Med. 2017; 377: 1033-1042.

42. Mas JL, Derumeaux G, Guillon B, et al. CLOSE Investigators. Patent foramen ovale closure or anticoagulation vs. antiplatelets after stroke. N Engl J Med. 2017; 377: 1011-1021.

43. Lee PH, Song JK, Kim JS, et al. Cryptogenic stroke and high-risk patent foramen ovale: the DEFENSE-PFO trial. J Am Coll Cardiol. 2018; 71: 2335-2342.

44. Kent DM, Saver JL, Ruthazer R, et al. Risk of Paradoxical Embolism (RoPE)-Estimate Attributable Fraction Correlates with the Benefit of Patent Foramen Ovale Closure: An Analysis of 3 Trials. Stroke. 2020; 51(10): 3119-3123.