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Coronary Artery Embolism From Atrial Flutter Presenting as Cardiac Arrest and ST-Elevation Myocardial Infarction: Diagnostic and Therapeutic Challenges

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Study Design A
Data Collection B
Statistical Analysis C
Data Interpretation D
Manuscript Preparation E
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Patient: Female, 82-year-old
Final Diagnosis: Atrial flutter • cardiac arrest • embolic myocardial Infarction • STEMI
Symptoms: Cardiac arrest
Clinical Procedure: —
Specialty: Cardiology

Objective: Challenging differential diagnosis

Background: Coronary artery embolism (CAE) is an infrequent but clinically significant non-atherosclerotic cause of ST-elevation myocardial infarction (STEMI). It presents unique diagnostic and therapeutic challenges due to its varied etiologies and the frequent absence of significant coronary artery disease. While atrial fibrillation is the most recognized cardiac arrhythmia associated with coronary embolism, atrial flutter as an embolic source remains underreported. This report highlights the complexities of diagnosing and managing CAE manifesting as an atypical STEMI and sudden cardiac arrest in a patient with previously undiagnosed atrial flutter, emphasizing the critical importance of identifying underlying embolic sources, particularly atrial flutter, to guide appropriate therapy.

Case Report: An 82-year-old woman with non-ischemic cardiomyopathy presented after an out-of-hospital ventricular fibrillation cardiac arrest. Her post-resuscitation electrocardiogram revealed an inferior STEMI. Emergency coronary angiography demonstrated abrupt embolic occlusions in the distal right coronary artery, without evidence of significant underlying atherosclerosis. Percutaneous coronary intervention was performed to restore blood flow. After the procedure, telemetry revealed new-onset atrial flutter, the presumed embolic source. Her hospital course was complicated by severe global hypokinesis out of proportion to the infarct territory and progressive respiratory failure, ultimately leading to a family decision to transition to comfort care.

Conclusions: This case underscores the critical need to consider coronary artery embolism in the differential diagnosis for STEMI, particularly in patients lacking significant atherosclerotic disease burden. The presence of arrhythmias, such as atrial flutter, should raise strong clinical suspicion for an embolic etiology. While a good outcome was not achieved in this specific case, early recognition of CAE remains crucial for guiding appropriate revascularization strategies and initiating prompt anticoagulation to prevent recurrent thromboembolic events in surviving patients.

Keywords: Atrial Flutter • Cardiology • Case Reports • Coronary Embolism • Percutaneous Coronary Intervention • ST Elevation Myocardial Infarction

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Introduction

Coronary artery embolism (CAE) is an uncommon but clinically significant cause of ST-elevation myocardial infarction (STEMI). While traditionally grouped under the broader umbrella of acute coronary syndrome (ACS), recognizing CAE specifically within STEMI presentations is critical. Recent data by Popovic et al (2024) demonstrate that while CAE accounts for approximately 5% of all STEMI cases, it is the culprit in up to 20% of STEMI patients presenting with concomitant atrial fibrillation [1]. However, while the association between coronary embolism and atrial fibrillation has been extensively described, atrial flutter as an isolated embolic source remains highly underreported and underrecognized. Despite this high prevalence in specific subgroups, it is often overlooked due to its variable presentation and the frequent absence of overt embolic sources.

Unlike traditional atherosclerotic coronary occlusions, CAE arises from embolic material originating from cardiac or, in the case of a patent foramen ovale, systemic sources, leading to abrupt vessel occlusion and myocardial ischemia. The etiologies of CAE are diverse, with atrial fibrillation and infective endocarditis being the most common underlying conditions [2,3]. While atrial flutter shares a similar thrombogenic pathophysiology with fibrillation, its specific role as a primary etiology for CAE is far less documented. Other notable causes include arrhythmias like atrial flutter, cardiomyopathy with the potential for left ventricular thrombus, prosthetic valve thrombosis, paradoxical embolism, and hypercoagulable states like antiphospholipid syndrome or factor V Leiden mutation, and malignancy-associated thrombosis [4]. The diagnosis remains challenging, as conventional coronary angiography may not readily differentiate embolic events from thrombotic occlusions secondary to atherosclerosis. To address this, Shibata et al (2015) proposed diagnostic criteria to aid in recognizing CAE, emphasizing the importance of identifying an embolic source and ruling out significant atherosclerosis [2].

Management strategies for CAE have not been well established by current guidelines, presenting a significant clinical challenge. While primary percutaneous coronary intervention (PCI) with aspiration thrombectomy is often favored to avoid unnecessary stent deployment in non-atherosclerotic vessels, definitive standardized protocols remain lacking [5].

Furthermore, while long-term systemic anticoagulation is essential for preventing recurrent embolic events, particularly in patients with atrial arrhythmias or thrombophilia, initiating such therapy is often complicated by severe clinical instability, cardiogenic shock, or early mortality [5]. This report highlights the complexities of diagnosing and managing CAE manifesting as an atypical STEMI and sudden cardiac arrest in a patient with previously undiagnosed atrial flutter. By distinguishing atrial flutter from more commonly reported arrhythmias, this case underscores the critical diagnostic gaps and the challenges of initiating therapy before catastrophic clinical deterioration occurs.

Case Report

We report the case of an 82-year-old woman who presented to the emergency department (ED) after an out-of-hospital ventricular fibrillation (VF) cardiac arrest. The patient had a known baseline history of non-ischemic cardiomyopathy following a diagnosis of infective myocarditis in 1997. She had chronic heart failure with a historically reduced left ventricular ejection fraction (LVEF) of 26% managed with medical therapy; she was not on baseline systemic anticoagulation and had previously declined an implantable cardioverter-defibrillator (ICD). On the day of admission, she had a VF arrest at home. Cardiopulmonary resuscitation (CPR) was immediately initiated by her daughter, who activated emergency medical services. Return of spontaneous circulation was achieved after a 10-minute downtime following a single defibrillator shock by paramedics.

Upon arrival at the ED, she was in cardiogenic shock requiring vasopressor support and was intubated for acute respiratory failure. A post-resuscitation electrocardiogram (ECG) revealed inferior lead ST-segment elevations with reciprocal changes in the lateral leads (**Figure 1**). The cardiac catheterization laboratory was urgently activated. Selective coronary angiography revealed a lack of significant obstructive atherosclerotic disease (10% stenosis in the left anterior descending artery and 20% in the left circumflex). However, there was an abrupt 100% occlusion in the right posterior descending artery (PDA) with a visible filling defect consistent with thrombus, as well as an occlusion in the posterolateral branch (PLB) (**Figure 2**). At the time of the procedure, an embolic etiology was not suspected, as the patient's atrial flutter was unknown. Consequently, the lesion was treated as a standard primary atherosclerotic STEMI. A 0.014-inch run-through wire was advanced into the distal PDA, pre-dilated with a 2.5×12 mm balloon, and a 2.25×12 mm drug-eluting stent was deployed. Thrombolysis in myocardial infarction (TIMI) 0 flow was successfully restored to TIMI III flow with only mild residual intracoronary thrombus (**Figure 3**). The patient was loaded with aspirin and clopidogrel and initiated on an eptifibatide infusion. A left ventriculogram performed during the procedure revealed a severely reduced LVEF of 10% to 15% with profound global hypokinesis, a finding disproportionate to the small myocardial territory impacted by the distal right coronary artery lesions.

The patient was transferred to the intensive care unit (ICU). To evaluate for concomitant paradoxical or systemic emboli

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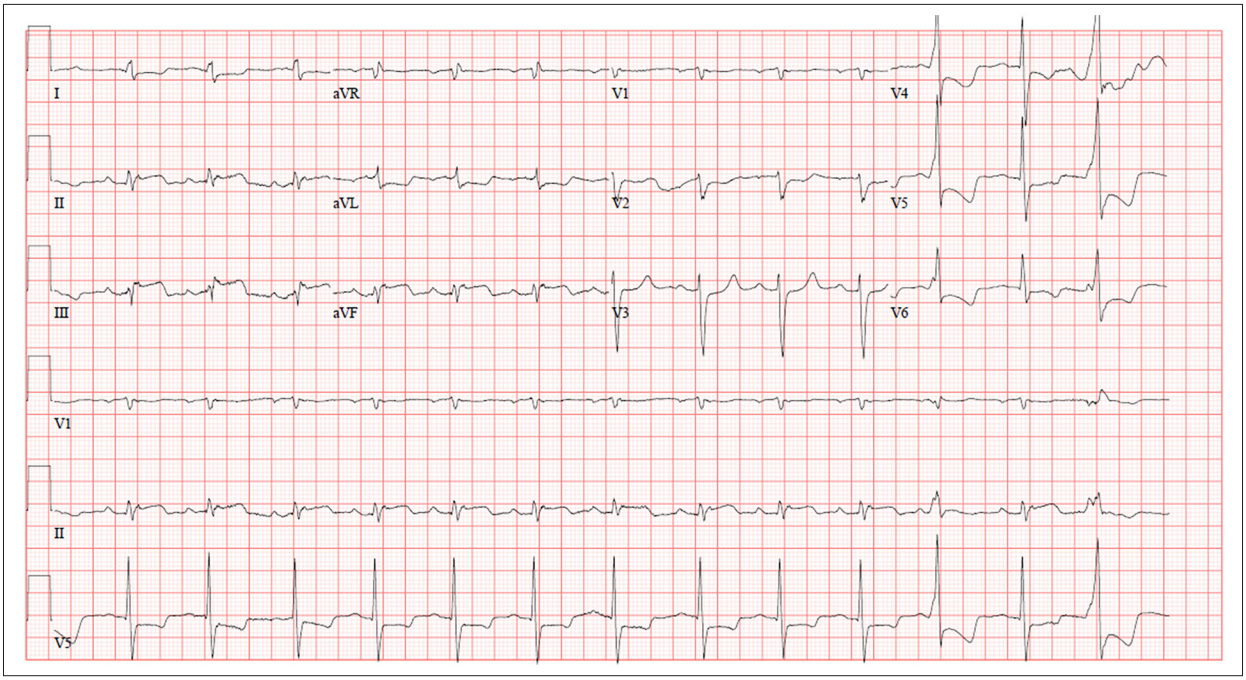

Figure 1. Baseline electrocardiogram demonstrating inferior ST-elevation myocardial infarction. Initial 12-lead electrocardiogram obtained upon presentation following return of spontaneous circulation. The tracing reveals significant ST-segment elevations in the inferior leads (II, III, aVF) with corresponding reciprocal ST-segment depressions in the lateral leads (I, aVL, V4-V6).

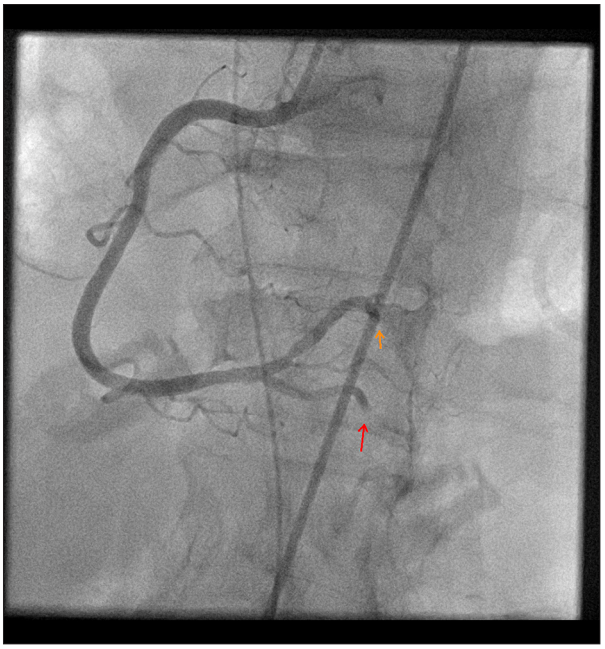

Figure 2. Coronary angiogram demonstrating abrupt embolic occlusion of the right coronary artery. The angiogram demonstrates the right coronary artery (RCA) without significant underlying atherosclerotic plaque. There is an abrupt total occlusion at the bifurcation of the posterior descending artery (PDA) (red arrow) and posterolateral branch (PLB) (orange arrow).

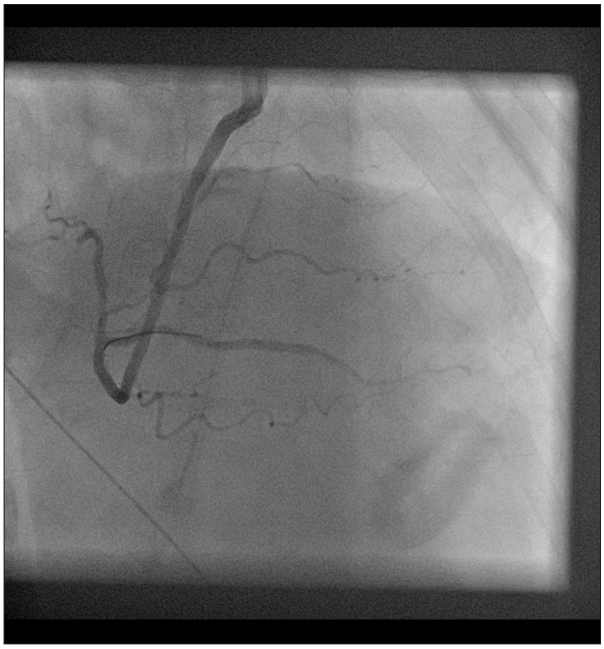

Figure 3. Post-intervention coronary angiogram demonstrating restored flow. The angiogram demonstrates the right coronary artery (RCA) following successful restoration of TIMI III blood flow to the distal vessel.

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given her shock and respiratory failure, computed tomography (CT) imaging was performed. A CT scan of the head revealed no acute intracranial hemorrhage or infarct, and a CT angiogram of the chest, abdomen, and pelvis ruled out pulmonary embolism and aortic dissection, but it did identify a moderate right pleural effusion and bibasilar atelectasis. A transthoracic echocardiogram confirmed the profound global hypokinesis (LVEF 10% to 15%) without evidence of a left ventricular thrombus. Her family noted she had recently experienced severe emotional distress following the death of a relative, raising secondary suspicion for a superimposed stress-induced cardiomyopathy.

The critical diagnostic turning point occurred on hospital day 3, when telemetry revealed new-onset atrial flutter. Given the lack of significant background atherosclerosis on angiography and this new arrhythmogenic finding, the PDA occlusion was retrospectively identified as a coronary artery embolism. Intravenous heparin and amiodarone therapy were immediately initiated. A transesophageal echocardiogram (TEE) ruled out left atrial appendage thrombus, and she was electrically cardioverted to normal sinus rhythm to optimize cardiac output.

Despite these interventions, her clinical course deteriorated. She was briefly extubated to non-invasive positive pressure ventilation (BiPAP) but required re-intubation within 24 hours for progressive respiratory failure exacerbated by her pleural effusions. The family declined pleurocentesis, expressing a desire to avoid further invasive therapies. Over the course of 10 days in the ICU, she remained ventilator-dependent with irreversible cardiogenic shock and multi-organ decline. Recognizing her poor prognosis and aligning with her previously stated goals of care, which are highlighted by her prior refusal of an ICD, multidisciplinary discussions were held with the family. The consensus decision was made to transition to comfort measures only. She underwent terminal extubation and died peacefully in the ICU.

Discussion

Coronary artery embolism (CAE) is an uncommon but critical non-atherosclerotic cause of ST-elevation myocardial infarction (STEMI). Studies suggest a CAE prevalence of roughly 3% to 5% among all acute myocardial infarction cases [1,2]. The etiologies are diverse; Lacey et al (2020) found infective endocarditis to be the most prevalent at 22.4%, followed by atrial fibrillation at 17%, whereas earlier database analyses by Shibata et al (2015) attributed up to 73% of cases to atrial fibrillation [2,3]. Other sources include cardiomyopathy with left ventricular thrombus, paradoxical emboli, and thrombophilic disorders [3]. More recently, Popovic et al (2024) reported 46 cases of coronary embolism associated with atrial fibrillation, highlighting this entity as underrecognized [1]. However, while atrial fibrillation is a heavily documented etiology, atrial flutter shares similar thromboembolic mechanisms but remains an underreported embolic source, demanding a heightened clinical index of suspicion.

The management of CAE presents unique challenges as it differs fundamentally from standard atherosclerotic STEMI. Current approaches favor percutaneous coronary intervention (PCI) with aspiration thrombectomy, ideally avoiding unnecessary stent placement unless significant underlying atherosclerosis or flow-limiting dissection is present [5]. However, because embolic etiologies are frequently unrecognized in the acute setting—as in this case, where the patient presented in extremis and the atrial flutter was not detected until hospital day 3—lesions are often treated definitively with balloon angioplasty and stenting to secure the vessel. Beyond acute revascularization, long-term systemic anticoagulation is crucial. The current literature suggests chronic anticoagulation should be initiated after a CAE event in the setting of atrial arrhythmias regardless of the baseline CHADS-VASc score [6]. Unfortunately, initiating such therapy is often precluded by profound clinical instability or early mortality.

Differentiating CAE from traditional atherosclerotic MI relies heavily on specific angiographic features. Shibata et al (2015) established diagnostic criteria for CAE, which our patient met [2]. In this case, several key angiographic findings supported the diagnosis and excluded simultaneous multivessel plaque rupture (MVPR): (1) abrupt vessel cutoff in the distal PDA and PLB without evidence of proximal atherosclerotic disease; (2) absence of collateral circulation; (3) lack of coronary calcification on fluoroscopy; and (4) presence of filling defects consistent with thrombus rather than ruptured plaque. While MVPR is a recognized cause of ACS, it is characterized by atheromatous changes, systemic inflammation, and a high atherosclerotic burden, making it highly unlikely in this patient's presentation [7].

Compared to previously reported cases of CAE, this presentation is uniquely characterized by the profound discordance between the anatomic lesion and the hemodynamic consequence. Distal right coronary artery occlusions typically impact a much smaller myocardial territory. However, this patient suffered a ventricular fibrillation arrest and developed irreversible cardiogenic shock with an ejection fraction of 10% to 15%. This catastrophic collapse was likely a combination of her baseline non-ischemic cardiomyopathy and a superimposed stress-induced (takotsubo) cardiomyopathy secondary to the recent loss of a relative. Identifying atrial flutter as the embolic source highlights an atypical etiology compared to standard atrial fibrillation cases. This case emphasizes that in patients with underlying structural heart disease, even a small

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distal coronary embolus can trigger devastating clinical deterioration, reinforcing the need for early recognition and aggressive diagnostic workup for occult arrhythmias.

Conclusions

This case underscores the critical necessity of considering coronary artery embolism (CAE) in the differential diagnosis of ST-elevation myocardial infarction (STEMI), particularly in patients presenting with atrial arrhythmias and lacking significant atherosclerotic disease burden. As evidenced by this patient's rapid clinical deterioration, a high index of suspicion is required when profound global hypokinesis and cardiogenic shock occur disproportionately to the territory of a distal coronary occlusion. While improved outcomes were not achieved in this specific case, early recognition of CAE remains crucial for guiding appropriate revascularization strategies and initiating prompt anticoagulation to prevent recurrent thromboembolic events in surviving patients.

Furthermore, this report highlights several concrete areas for future clinical research to optimize the management of this underrecognized condition. First, diagnostic and management guidelines specifically addressing atrial flutter as an embolic source need to be refined, as current protocols predominantly extrapolate from atrial fibrillation data. Second, further investigation is required to establish definitive revascularization algorithms for CAE, specifically delineating the precise indications for aspiration thrombectomy versus stent deployment in the absence of significant underlying atherosclerosis. Finally, improved risk stratification tools are essential to identify patients at the highest risk for recurrence and to guide the safety, intensity, and duration of long-term anticoagulation therapy in the setting of acute myocardial infarction.

Patient Consent

Patient permission was obtained for the preparation and submission of this manuscript.

Institution Where Work Was Done

Henry Ford Providence Hospital, Southfield, MI, USA.

Declaration of Figures' Authenticity

All figures submitted have been created by the authors who confirm that the images are original with no duplication and have not been previously published in whole or in part.

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