Acute ST-elevation Myocardial Infarction due to Coronary Embolism from Left Atrial Thrombus: Challenges in Mechanical Recanalization and the Role of Pharmacological Therapies.

Amaury Sogorb¹, Timothée Noterdaeme¹, Lucien Finianos¹, Pieter-Jan Palmers¹, and Olivier Gach¹

¹Centre Hospitalier Chrétien

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Authors and Affiliations:
Amaury Sogorb¹, M.D.: amaury.sogorb@gmail.com
Timothée Noterdaeme¹, M.D.: timothee.noterdaeme@chc.be
Lucien Finianos¹, M.D.: lucien.finianos@chc.be
Pieter-Jan Palmers¹, M.D.: pieter-jan.palmers@chc.be
Olivier Gach¹, M.D. PhD.: oliviergach70@gmail.com
¹: Department of Cardiology, CHC Montlégia, Liège, Belgium

Short Running Title:
Coronary Embolism from Atrial Thrombus: STEMI Management Challenges

Corresponding Author:
Amaury Sogorb
Department of Cardiology, CHC Montlégia
Bd Patience et Beaujonc 2
4000 Liège, Belgium
Email: amaury.sogorb@gmail.com

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**Key Clinical Message**
In select unusual STEMI cases, combined antiplatelet and anticoagulation therapy can achieve reperfusion despite mechanical intervention failures. Profound knowledge of these drug combinations is key.

**Abstract**
In patients with ST-elevation myocardial infarction, early revascularization plays a crucial role and is widely acknowledged as the primary treatment option. However, various underlying etiologies can lead to a STEMI, and it’s of utmost importance to recognize the specific etiology since initial treatment strategies can differ significantly.

In this case report, we present a patient with atrial fibrillation and LAA thrombus. The patient’s initial symptoms were indicative of an acute ST-segment elevation myocardial infarction (STEMI) caused by a distal obstruction in the left anterior descending artery (LAD). This obstruction proved to be resistant to angioplasty intervention but was successfully treated and cleared using conservative medical measures, eliminating the need for stenting.

**Classification Keywords:**
Cardiology, Cardiovascular Disorders, Pharmacology and Pharmacy
ST Elevation Myocardial Infarction, Reperfusion, Platelet Glycoprotein GPIIb-IIIa Complex, Antiplatelet Agents, Anticoagulants

**Abbreviations:**
ECG – Electrocardiogram
STEMI - ST-elevation myocardial infarction
UFH - Unfractionated Heparin
ASA - Acetylsalicylic acid
LAD - Left Anterior Descending artery
ACT - Activated Clotting Time
GPIIb/IIIa - Glycoprotein IIb/IIIa
CT - Computed Tomography
CICU - Cardiac Intensive Care Unit
TOE - Transesophageal Echocardiogram
LAA - Left Atrial Appendage
ACS-ST+ - Acute Coronary Syndrome-ST elevation myocardial infarction

**Case report**
A 59-year-old male patient presented to our emergency department with an acute onset of severe chest pain upon awakening. The first electrocardiogram (ECG) taken three hours after symptom onset displayed a regular sinuss rhythm with an ST-elevation myocardial infarction pattern in both the anterior and inferior leads (figure 1). The patient’s hemodynamics were stable.
He was promptly treated pharmacologically with an intravenous bolus of unfractionated heparin (UFH) at 70U/Kg, acetylsalicylic acid (ASA), and oral Ticagrelor 180mg. He was then transferred for coronary angiography. Upon arrival at the cath-lab, his symptoms had already spontaneously improved. A subsequent ECG exhibited persistent ST-segment elevation in the anterior leads, but the ST-segment elevation in the inferior leads had resolved.

Via the right radial access, injection of the right coronary artery displayed a smooth, lesion-free vessel. The left coronary angiography revealed large-caliber, smooth-walled coronary arteries with, however, a mid-segment thrombo-embolic occlusion in the left anterior descending artery (LAD). Before initiating the intervention on the LAD, UFH was readministered, adjusted to the ACT result. Although a wire and a balloon catheter easily passed the occlusion site, multiple balloon angioplasties and passages failed to reestablish distal blood flow in the LAD (figure 2). Administration of two distinct intracoronary vasodilators, Isosorbide dinitrate and Papaverine, yielded no significant improvement, leaving distal perfusion entirely obstructed. Thromboaspiration was not attempted due to worries about thrombus mobilization and the subsequent stroke risk. No intravascular imaging was utilized.

Due to the non-atherosclerotic appearance of the other coronary arteries and the unsuccessful balloon reperfusion, a conservative treatment approach was chosen without stent placement. Pharmacological therapy with GPIIb/IIIa inhibitors, specifically eptifibatide in our case, was initiated and maintained for 24 hours.

Several days after the incident, a coronary computed tomography (CT) scan was performed, revealing the normal appearance of all coronary arteries and the complete restoration of blood flow in the left anterior descending (LAD) artery (figure 3).

During the patient’s stay in our cardiac intensive care unit (CICU), he was diagnosed with a first episode of atrial fibrillation. A transesophageal echocardiogram (TOE) performed prior to electrical cardioversion revealed a left atrial thrombus (figure 4), which was likely the cause of the patient’s acute coronary syndrome. Fragments of the thrombus had probably embolized, explaining the initial anterior and inferior ST-elevation myocardial infarction. The evolving changes in the ECG might have been due to the pharmacological dissolution of the blood clot in an arterial branch.

A transthoracic echocardiogram revealed moderately impaired function, showing anteroseptal, inferoseptal, and lateral-apical hypokinesia. Furthermore, the patient was diagnosed with severe aortic stenosis. Consequently, aortic valve replacement surgery has been scheduled, pending confirmation of the complete resolution of the LAA thrombus.

Discussion

This atypical case underscores the importance of recognizing and considering the diverse etiologies of acute ST-segment elevation myocardial infarction, given the vast differences in potential treatment strategies. The observed thrombotic event, attributed to an embolism from a left atrial thrombus, coupled with the secondary recanalization of the implicated coronary artery evident on a subsequent coronary CT scan—without the intervention of stenting or thromboaspiration—suggests a pivotal role of the GPIIb/IIIa inhibitor in facilitating this favorable outcome.

Given that multiple balloon angioplasty attempts were unsuccessful, our team deduced that the inability to reopen the LAD might indicate a significant thrombotic load at both the epicardial coronary and microcirculatory levels, known as the ‘no-reflow phenomenon’.

The long-term benefits of culprit artery reopening in STEMI and subsequent coronary reperfusion hinge on the total duration of ischemia. Early recanalization is linked with a marked improvement in prognosis, especially in terms of survival. However, it’s important to note that the quality of reperfusion is equally significant.

Various treatment strategies can be implemented for patients with ST-elevation myocardial infarction and a substantial thrombus burden to reduce the risk of microcirculatory obstruction. These strategies encompass
thrombus aspiration, utilization of distal protection devices, and/or administration of glycoprotein IIb/IIIa inhibitors (GPIs)². Yet, even with these approaches, achieving a complete thrombus removal might remain elusive.

Moreover, patients with acute coronary syndrome typically exhibit elevated stress levels, inflammation, and a tendency for hypercoagulability. Excessive intravascular manipulation during procedures can potentially exacerbate the inflammatory state and activate the coagulation cascade, which may, in turn, precipitate further thrombus formation³. Implanting a stent under such conditions can instigate clotting, potentially leading to thromboembolism distal to the stent location.

Since recanalization attempts were unsuccessful and considering the uncertainty surrounding the nature of the obstruction (especially with other coronary arteries appearing angiographically intact), our team chose not to pursue stent placement. This decision aimed to mitigate the potential risks tied to more invasive interventions. Instead, we opted solely for medical treatment, which, in retrospect, proved to be the right choice.

In the present era, when stent implantation for STEMI is standard and current guidelines predominantly advocate for immediate stenting as the preferred strategy⁴, the angiographic appearance of the arteries, mechanisms of obstruction, and clinical presentation should compel us to reconsider the suitability of stenting to mitigate potential complications. Implanting a stent in this patient might have exacerbated the local inflammatory processes, thrombotic burden, and the already-present no-reflow phenomenon, further diminishing the chances of recanalizing the left anterior descending artery.

In reflecting on the treatment regimen employed, it becomes evident that the aggressive pharmacological strategy had a significant role in achieving the favorable outcome observed. Notably, the combination of unfractionated heparin (UFH) with a trio of antiplatelet agents—ASA, P2Y12 inhibitor, and GPIIB/IIIA—may have been pivotal in restoring full coronary flow. Each of these agents plays a distinct role in preventing thrombus formation and progression. UFH, an anticoagulant, inhibits the conversion of fibrinogen to fibrin, thereby halting clot propagation. Meanwhile, ASA and P2Y12 inhibitors prevent platelet activation and aggregation, with GPIIB/IIIA further inhibiting platelet cross-linking. The combined effect of these drugs could likely disrupt existing thrombi while simultaneously preventing new thrombus formation, thus achieving the reperfusion seen in our patient. This highlights the importance of tailoring treatment approaches based on the specific clinical context, even when mechanical interventions are considered the norm.

The decision not to attempt thrombus aspiration was a topic of debate within our team. Fears of thrombus mobilization during aspiration and the subsequent risk of a stroke due to massive intracoronary thrombus led to the decision against this technique. Another reason to avoid thromboaspiration was its potential ineffectiveness on distal thrombus fragments already lodged in the microcirculation.

Thromboaspiration has been extensively studied as a treatment option for ACS-ST+. This technique is viewed as a potential complement to angioplasty, enhancing both epicardial and myocardial perfusion and preserving microvascular integrity by preventing distal embolization of thrombotic material.

The decision not to use thromboaspiration was a contentious one. Studies like TAPAS⁵ showed the potential benefits, but the later TASTE⁶ and TOTAL⁷ studies indicated it didn’t significantly reduce adverse events and might increase the risk of stroke. Given the balance of evidence, thromboaspiration is not routinely recommended⁸, and our patient’s perceived risk profile made us lean against it.

It is also important to note that in our patient, revascularization of the left anterior descending artery (LAD) using only pharmacological treatment (with a likely appropriate non-stenting strategy) does not offer insight into the duration of ischemia prior to reperfusion. This might partially explain the observed segmental kinetic abnormalities and echocardiographic functional impairment. Hence, the genuine long-term clinical outcome is still ambiguous.

However, at the same time, the epicardial coronary network, which captures all the attention of the interventional cardiologist during the urgent angioplasty procedure in STEMI, represents only 5 to 10% of the
overall coronary circulation. The microcirculation and the potential no-reflow phenomenon, which appears in 11-41% of STEMI cases depending on the series studied\(^9\), remains less manageable by the interventional cardiologist.

**Conclusion**

Our clinical case exemplifies a nuanced and less frequent STEMI scenario with occlusion of the LAD due to coronary embolism, wherein mechanical recanalization of the artery proved elusive despite repeated attempts. What stands out prominently in our case is the seemingly profound impact of the GPIIb/IIIa inhibitor used in conjunction with full anticoagulation by UFH. Within the myriad of treatment strategies available, this combination has emerged as a potential game changer. While we have acknowledged the substantial thrombotic load and recognized the 'no-reflow phenomenon', it was this particular pharmacological strategy that may have tipped the scales in our patient’s favor.

Indeed, while many discussions revolve around mechanical interventions and their merits, our case underscores the potency of combining GPIIb/IIIa inhibitors with UFH. It’s plausible to suggest that while the GPIIb/IIIa inhibitor prevented further platelet aggregation and enhanced thrombus dissolution, the UFH provided robust anticoagulation, preventing new thrombus formation. Their synergistic effect could have facilitated the reperfusion observed days later in the LAD, without any invasive coronary intervention. The success achieved by solely relying on this pharmacological approach, amidst failed mechanical interventions, echoes the importance of understanding and harnessing the full potential of drug combinations in managing acute ST-segment elevation myocardial infarction cases. It’s a testament to the fact that, in certain contexts, medication alone, when judiciously chosen, can dramatically influence outcomes.

**Author Contributions**

Amaury Sogorb: Conceptualization, data collection, manuscript drafting, final approval of the version to be published, and overall responsibility for the accuracy and integrity of the work.

Olivier Gach: Critical review of the manuscript, provided intellectual input, final approval of the version to be published, and ensured accuracy and integrity of a portion of the work.

Timothée Noterdaeme: Critical review of the manuscript, provided intellectual input, final approval of the version to be published, and ensured accuracy and integrity of a portion of the work.

Lucien Finianos: Minor contribution in manuscript review, final approval of the version to be published, and ensured accuracy and integrity of a portion of the work.

Peter-Jan Palmers: Minor contribution in manuscript review, final approval of the version to be published, and ensured accuracy and integrity of a portion of the work.

**Figure legend**

**Figure 1.** Admission electrocardiogram from the ER.

**Figure 2.** Unsuccessful revascularization during coronary angiography.

**Figure 3.** CT scan depicting LAD restoration.

**Figure 4.** Left atrial thrombus observed in transesophageal echocardiography (TEE).

**References**


