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Case Report

Acute Anterior ST-Elevation Myocardial Infarction in a Patient with Mechanical Aortic Valve and Subtherapeutic Anticoagulation: Successful Revascularization Using Microcatheter-Guided Distal Visualization

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Abstract

Background:

Coronary embolism is an uncommon but important cause of ST-elevation myocardial infarction (STEMI), particularly in patients with mechanical heart valves and inadequate anticoagulation.

Case Presentation:

A 58-year-old male with a history of mechanical aortic valve replacement presented with acute chest pain and hemodynamic instability. Electrocardiography revealed anterolateral STEMI, and laboratory investigations showed markedly elevated troponin levels (204.6 ng/L) and subtherapeutic INR (1.25). Coronary angiography demonstrated complete occlusion of the mid left anterior descending artery (LAD) with TIMI 0 flow. Conventional reperfusion strategies, including thrombus aspiration and balloon predilatation, failed. A Finecross microcatheter was advanced distal to the lesion, enabling visualization of the distal LAD. A 3.0 × 28 mm drug-eluting stent was subsequently deployed, restoring TIMI III flow.

Conclusion:

This case highlights the importance of recognizing thromboembolic STEMI in patients with mechanical valves and demonstrates the utility of microcatheter-guided distal visualization as an effective bailout strategy in complex PCI.

Introduction

Acute ST-elevation myocardial infarction (STEMI) is a critical cardiovascular emergency, most commonly caused by atherosclerotic plaque rupture leading to coronary thrombosis. Rapid restoration of coronary blood flow through primary percutaneous coronary intervention (PCI) remains the cornerstone of management and significantly reduces mortality and morbidity [1,4].

However, in a subset of patients, STEMI may result from non-atherosclerotic mechanisms, including coronary embolism. Coronary embolism is an underrecognized cause of acute myocardial infarction, accounting for approximately 3% of STEMI cases [2,3]. It is particularly relevant in patients with predisposing conditions such as atrial fibrillation, infective endocarditis, and prosthetic heart valves.

Mechanical heart valves require lifelong anticoagulation to prevent thrombus formation. Subtherapeutic anticoagulation significantly increases the risk of thromboembolic events, which may manifest as stroke, peripheral ischemia, or, rarely, coronary artery occlusion [5]. The diagnosis of coronary embolism is often inferred from clinical context, angiographic findings, and absence of significant underlying coronary artery disease.

Interventional management of embolic STEMI poses unique challenges. Unlike atherosclerotic lesions, embolic occlusions may demonstrate high thrombus burden and resistance to standard PCI techniques such as thrombus aspiration and balloon angioplasty [6]. In such cases, adjunctive tools and innovative strategies are essential.

Microcatheters have emerged as valuable devices in complex PCI, allowing improved guidewire support, distal vessel visualization, and precise lesion crossing. Their role becomes particularly important when intravascular imaging modalities such as IVUS or OCT are unavailable[7].

We present a case of anterior STEMI in a patient with a mechanical aortic valve and subtherapeutic anticoagulation, successfully treated using a microcatheter-guided bailout strategy.

Case Presentation

A 58-year-old male presented with acute onset chest pain of 2 hours duration. His medical history was significant for mechanical aortic valve replacement performed 14 years earlier for aortic stenosis. He also had chronic heart failure, left lower limb amputation, and ongoing non-vascular gangrene of the right lower limb, with preserved dorsalis pedis and posterior tibial pulses and a normal ankle–brachial index, suggesting non-arterial etiology.

On admission, the patient was hemodynamically unstable with a blood pressure of 80/50 mmHg and required intravenous norepinephrine support.

Electrocardiography demonstrated ST-segment elevation in the anterior and lateral leads, consistent with acute anterolateral STEMI (Figure 1,2). Figure 3 is on discharge.

Laboratory investigations revealed:

- Troponin: 204.6 ng/L
- INR: 1.25 (subtherapeutic)
- D-dimer: 4.04 µg/mL
- Mild leukocytosis and electrolyte imbalance

These findings strongly suggested a thromboembolic etiology.

Parameter	Details
Age	58 years
Gender	Male
Valve History	Mechanical aortic valve (14 years)
Comorbidities	Heart failure
Vascular Disease	Amputation + gangrene
Presentation	Acute chest pain
Hemodynamics	Shock (BP 80/50 mmHg)

Table 01: Clinical Profile

Parameter	Value
Troponin	204.6 ng/L
INR	1.25
D-dimer	4.04 µg/mL
Potassium	5.2 mmol/L
WBC	11.89 K/µL

Table 2: Laboratory Findings

Step	Details
Access	Femoral
Catheter	7F
Wire	Sion
Microcatheter	Finecross
Stent	Supraflex 3.0 × 28 mm
Outcome	TIMI III flow

Table 03: Procedural Summary

Angriographic Interpretation

Coronary Angiography Findings

Coronary angiography revealed a complete occlusion of the mid segment of the left anterior descending artery (LAD) with absence of distal flow (TIMI 0) (Figure 6). The vessel appeared abruptly cut off, without clear evidence of underlying atherosclerotic stenosis, supporting a possible thromboembolic etiology.

Following guidewire crossing using a Sion wire and multiple unsuccessful attempts with thrombus aspiration and balloon predilatation, a Finecross microcatheter was advanced distal to the lesion. Contrast injection through the microcatheter demonstrated a patent distal LAD, confirming the presence of a true lumen beyond the occlusion (Figure 7).

Subsequent stent deployment (Supraflex 3.0 × 28 mm) resulted in restoration of TIMI III flow with good distal vessel opacification (Figure 8), indicating successful reperfusion.

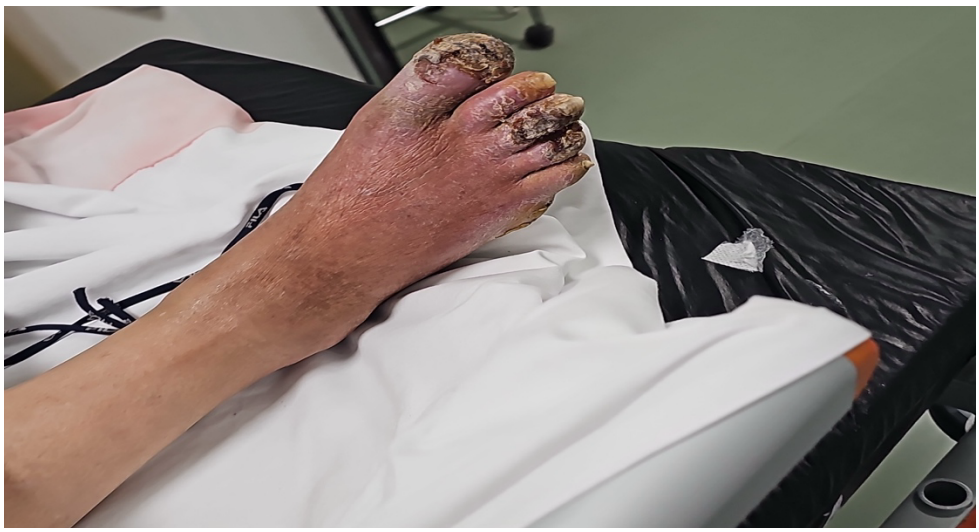

Figure 1: Right lower limb gangrene demonstrating severe peripheral ischemia and probable systemic thromboembolic disease

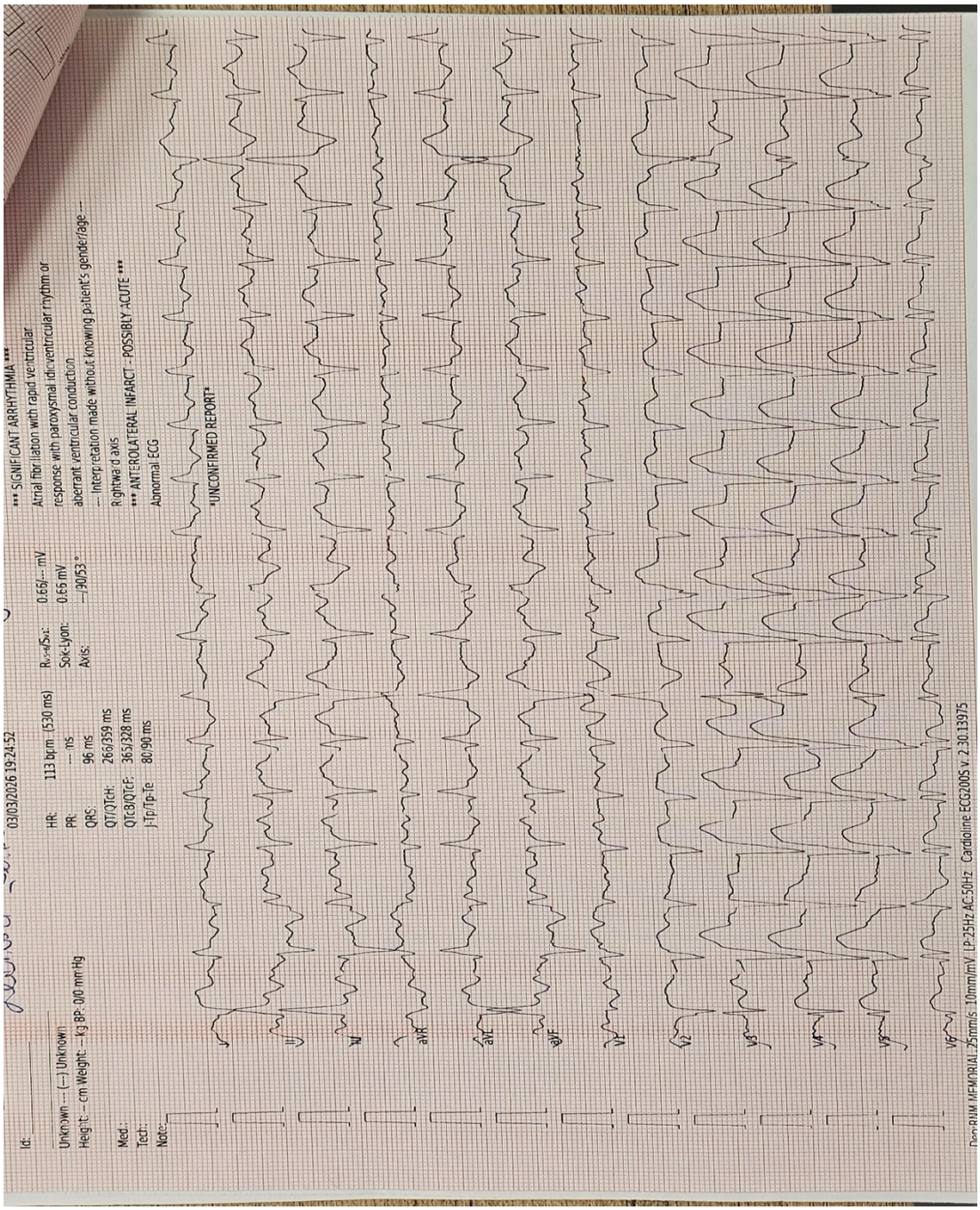

Figure 2: Baseline electrocardiogram showing ST-segment elevation in anterior and lateral leads consistent with acute anterolateral STEMI

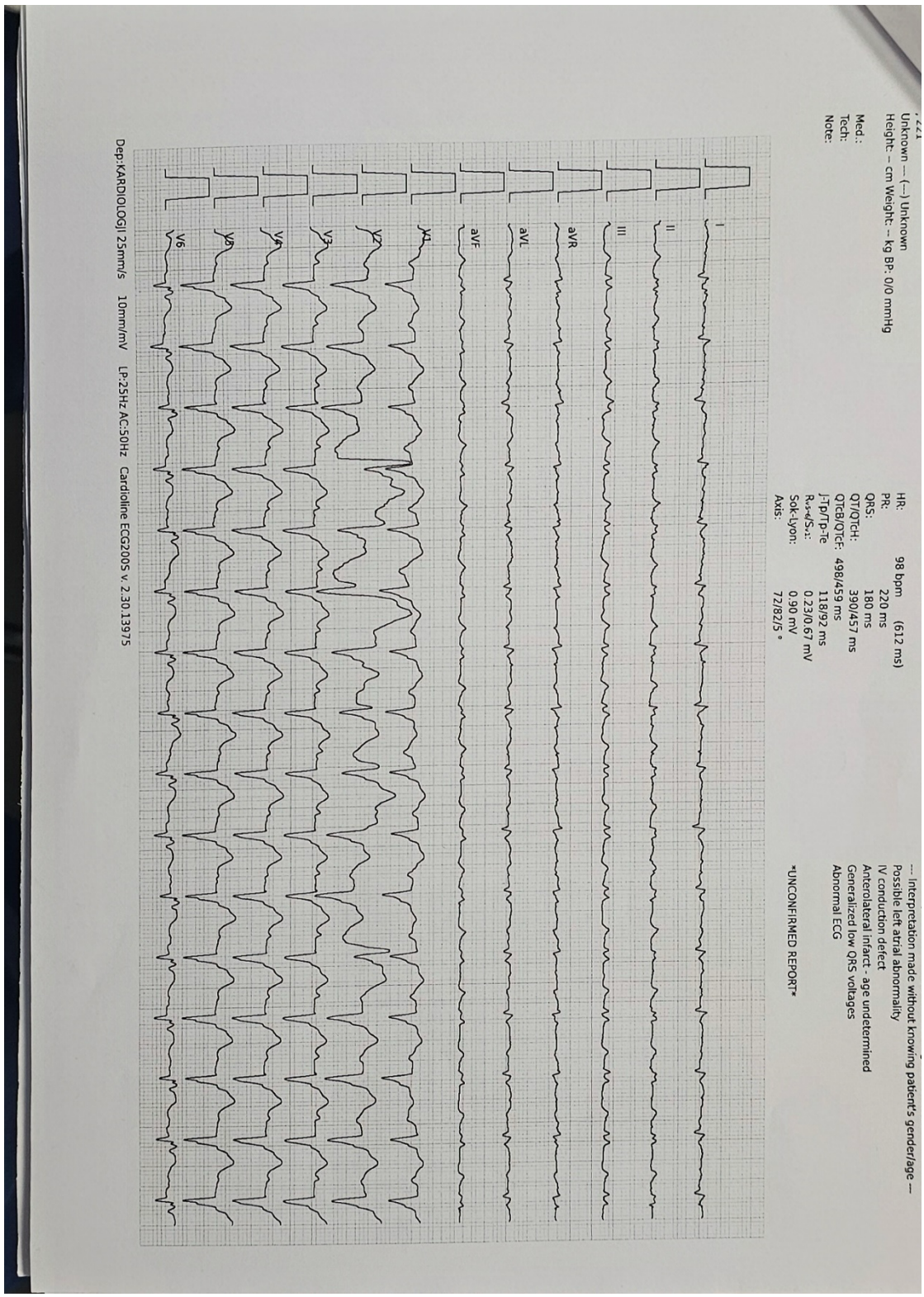

Figure 3: ECG Showing of ST Sepment After Restroing Flow

Quantity	Test Name	Result	Flag	Unit	Min Value
Name: BIOKIME (13 Record)					
	Glicemia	125,0	↑	mg/dl	74.00
	Troponin	204.6	↑	ng/L	
	Azotemia	38,52		mg/dl	18.00
	Kreatinemi	1,15		mg/dl	0.72
	Bilirubinë totale	1,1		mg/dl	0.30
	Bilirubinë e fraksion...	0,4		mg/dl	0.10
	Lipase	22,67		U/L	
	ALT-SGPT	10,0		U/L	
	AST-SGOT	23,0		U/L	5.00
	AMYLASE	29,86		U/L	25.00
	Na Natrium	138,0		mmol/L	132.00
	K Kalium	5,2	↑	mmol/L	3,5
	Cl Klor	102,0		mmol/L	98.00
Name: KOAGULIM (3 Record)					
	PT	72			70.00
	INR	1,25	↑		0.85
	D-Dimer	4.04ug/ml	↑		
Name: Gjak komplet (21 Record)					
	RBC	5.09		10^6/µL	4.40
	HGB	12.3	↓	g/dl	13
	HCT	37.1	↓	%	42.00
	MCV	72.9	↓	fL	80.00
	MCH	24.1	↓	pg	27.00
	MCHC	33.1		g/dl	32.00
	RDW-CV	14.4	↑	%	10.8
	WBC	11.89	↑	K/uL	4.00
	NEU#	8.76	↑	%	1.60
	LYM#	2.26		K/uL	

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Figure 4: Laboratory findings demonstrating elevated troponin levels and subtherapeutic INR.

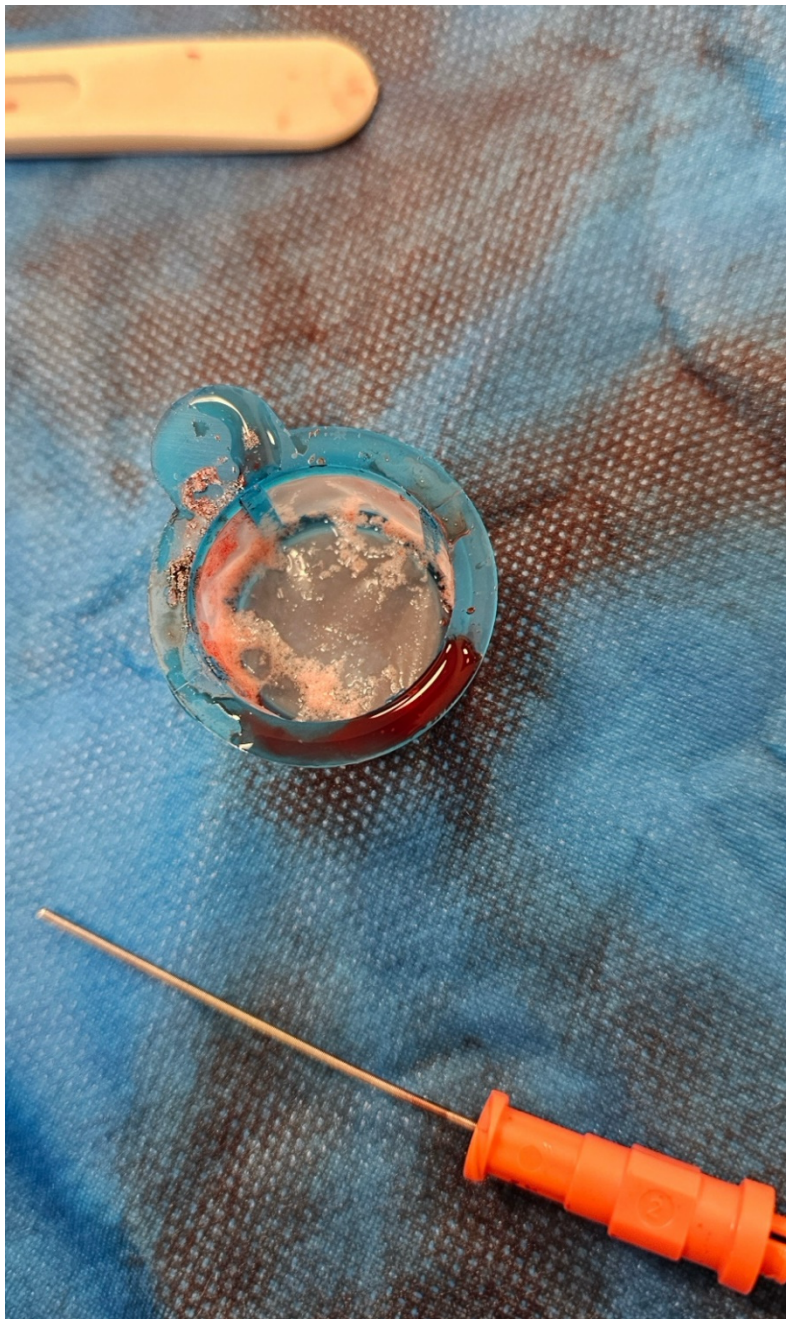

Figure 5: Extracted intracoronary thrombotic material obtained during PCI, supporting thromboembolic etiology

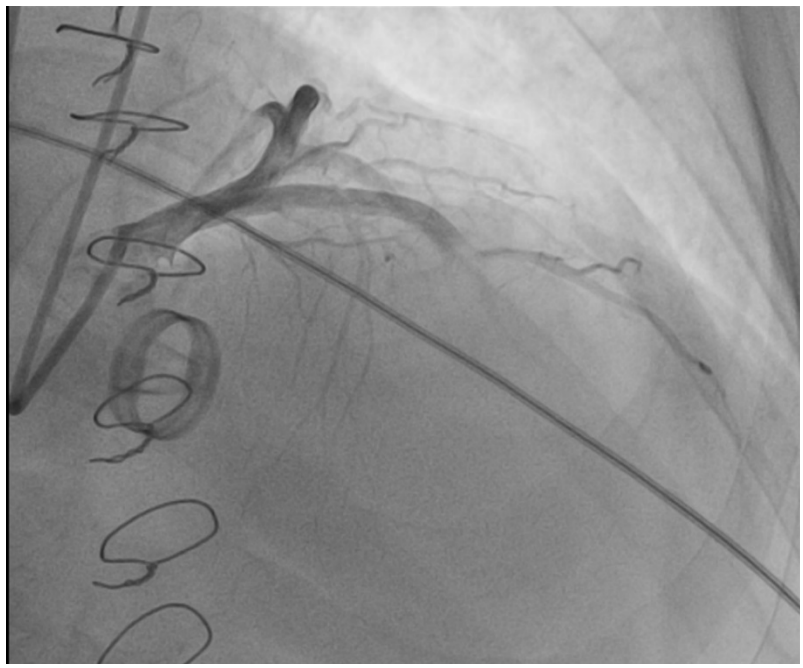

Figure 6: Coronary angiography showing abrupt occlusion of the mid left anterior descending artery (LAD) with absence of distal flow (TIMI 0).

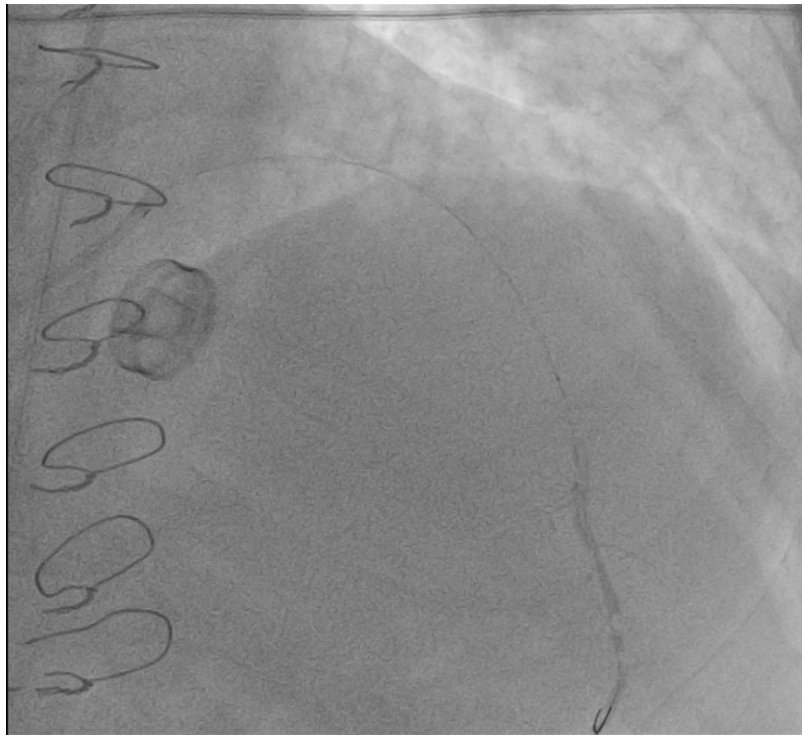

Figure 7: Microcatheter-guided distal contrast injection demonstrating patent distal LAD, confirming true lumen beyond the occlusion

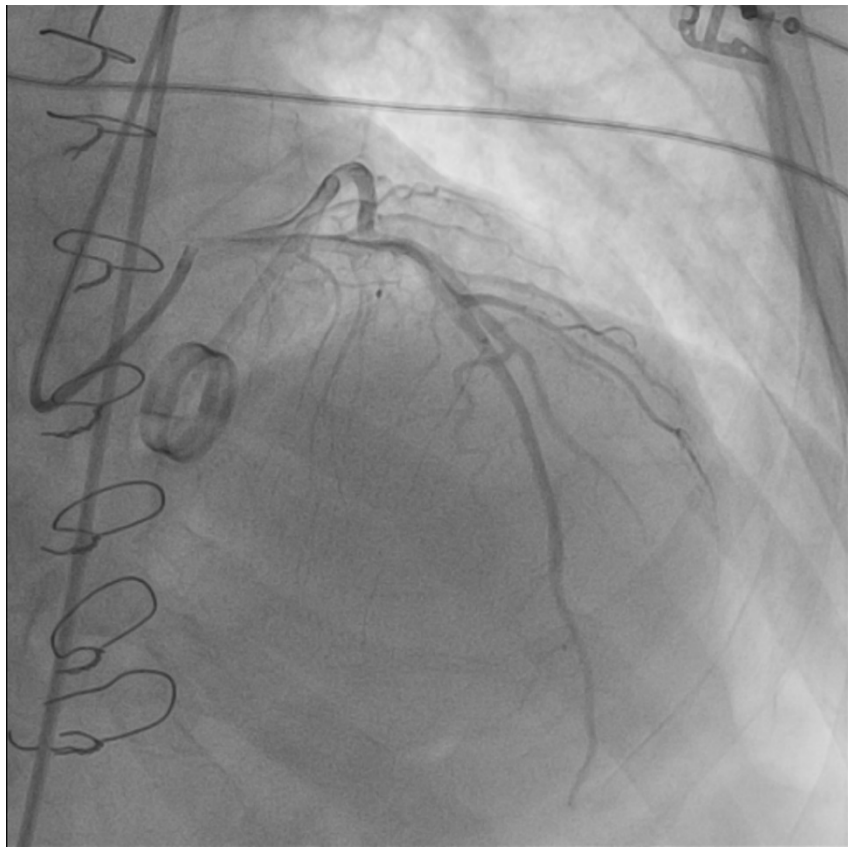

Figure 8: Final angiographic result after deployment of a drug-eluting stent showing restoration of TIMI III flow in the LAD with good distal perfusion

Discussion

This case illustrates a rare but clinically important presentation of acute anterior ST-elevation myocardial infarction (STEMI) in a patient with a mechanical aortic valve prosthesis, markedly subtherapeutic anticoagulation, and evidence of systemic peripheral ischemic disease[8]. Although the majority of STEMI presentations are attributable to atherosclerotic plaque rupture with superimposed thrombosis, this patient's clinical background, angiographic appearance, and laboratory findings strongly suggest an alternative mechanism, most likely coronary embolism. In this context, the case is particularly valuable because it combines both a distinctive pathophysiological substrate and a technically challenging percutaneous coronary intervention (PCI) that was ultimately rescued by microcatheter-guided distal vessel visualization.

Coronary embolism remains an uncommon and frequently underrecognized cause of acute myocardial infarction. In routine practice, STEMI is often immediately attributed to plaque disruption in the setting of obstructive coronary artery disease[9]. However, in selected patients, especially those with prosthetic heart valves, atrial fibrillation, endocarditis, intracardiac thrombus, or hypercoagulable states, embolic coronary occlusion must be actively considered in the differential diagnosis. In the present case, the patient had a mechanical aortic valve implanted 14 years previously and an INR of 1.25 at presentation, which is clearly below the expected therapeutic range for most mechanical aortic prostheses. This finding significantly increases the likelihood of thrombus formation on or around the prosthetic valve, with subsequent embolization into the systemic circulation. The clinical suspicion of an embolic mechanism is further reinforced by his history of left lower limb amputation and the presence of gangrenous changes in the contralateral lower limb, both of which suggest prior or ongoing severe peripheral ischemic events[10].

An important feature supporting a thromboembolic mechanism in this patient is the overall clinical pattern rather than any single finding in isolation. The abrupt onset of chest pain, the angiographic picture of a sudden mid-LAD occlusion, the lack of documented prior coronary disease, the absence of diffuse multivessel obstructive atherosclerotic changes in the images provided, and the coexistence of subtherapeutic anticoagulation collectively favor embolic occlusion over conventional plaque rupture. While definitive differentiation between embolic and atherothrombotic STEMI often requires intravascular imaging or histopathological confirmation, many real-world cases must be interpreted on the basis of clinical probability. In this patient, the extracted thrombotic material, the abrupt coronary cutoff, and the preserved distal vessel architecture seen after distal contrast injection all strengthen the hypothesis that a thrombus, rather than complex underlying calcified plaque, was the dominant mechanism of vessel closure[11].

The coexistence of systemic peripheral ischemic manifestations adds further clinical relevance to this case. The patient had already undergone left lower limb amputation and was noted to have non-vascular gangrene of the right lower limb. Even though the precise vascular workup of the limb ischemia was not available, these findings suggest a severely compromised vascular state and possibly repeated thromboembolic events or profound chronic ischemia. In the setting of a mechanical valve and insufficient anticoagulation, the possibility of recurrent embolization becomes especially concerning[12]. Thus, this case not only demonstrates an isolated coronary event but may reflect a

broader thromboembolic disease burden. From a discussion standpoint, this is important because it shifts the interpretation of the myocardial infarction from a purely coronary event to part of a multisystem vascular pathology.

From the interventional perspective, the case is equally noteworthy. Primary PCI in thromboembolic lesions can be substantially more challenging than PCI for conventional atherosclerotic culprit lesions. Embolic occlusions may be associated with high thrombus burden, poor distal visualization, distal embolization, no-reflow phenomena, and uncertainty regarding the true extent of the lesion. In the present case, standard procedural approaches were attempted first, including mechanical thrombus aspiration and multiple balloon predilatation attempts, yet these failed to restore flow. This failure is itself informative[13]. It suggests that the lesion was not easily compressible or traversable in the way typical soft plaque-related occlusions often are, and that visualization of the distal vessel was inadequate for confident and safe definitive treatment. Such situations place the operator in a technically difficult position: proceeding without understanding the distal vessel may risk suboptimal stent placement, while stopping the procedure leaves the patient with ongoing myocardial ischemia.

The key procedural turning point in this case was the use of a Finecross microcatheter advanced distal to the occlusion. This maneuver enabled contrast injection beyond the lesion and demonstrated a patent distal LAD, thereby confirming true lumen continuity and providing a roadmap for definitive intervention. This aspect is highly important and represents the major technical message of the report. In centers where intravascular ultrasound (IVUS) or optical coherence tomography (OCT) is unavailable, the microcatheter may serve as a simple and practical alternative tool to clarify anatomy in selected cases. Distal vessel visualization through a microcatheter can help determine whether the wire is in the true lumen, assess the caliber and course of the distal vessel, and guide appropriate stent sizing and landing zones. In the present case, this strategy transformed a failed reperfusion attempt into a successful procedure with restoration of TIMI III flow[14].

The angiographic sequence adds strong support to the technical success of this strategy. The initial images show abrupt mid-LAD occlusion with absent distal opacification, corresponding to TIMI 0 flow. After the microcatheter was advanced distally, contrast injection outlined the distal LAD and demonstrated that the distal vessel remained patent. This finding not only confirmed the feasibility of further intervention but also reduced the uncertainty regarding the distal landing zone. Following stent

deployment with a 3.0 × 28 mm Supraflex drug-eluting stent, final angiography demonstrated restoration of TIMI III flow with satisfactory distal perfusion. This progression from complete occlusion to full reperfusion provides a coherent procedural narrative and significantly strengthens the educational value of the case.

Another important consideration is whether stent implantation was the optimal strategy in a lesion suspected to be primarily embolic. In some embolic myocardial infarctions, aspiration or pharmacologic therapy alone may be sufficient if no underlying fixed stenosis exists. However, real-world practice often involves uncertainty, especially in unstable patients, in lesions with persistent obstruction after aspiration, or when distal anatomy cannot initially be assessed. In this case, repeated attempts at reperfusion failed, and the eventual demonstration of distal lumen continuity allowed definitive stent implantation. Given the hemodynamic instability at presentation, ongoing ischemia, and the need for prompt restoration of flow, stenting appears to have been a reasonable and life-saving choice[15]. The favorable in-hospital outcome and discharge by day 7 further support the effectiveness of this approach.

The laboratory findings also contribute meaningfully to the discussion. Troponin elevation confirmed acute myocardial injury, while the INR of 1.25 strongly suggested inadequate anticoagulation in a patient at inherently high thromboembolic risk. The elevated D-dimer may reflect active thrombosis or systemic coagulation activation, although it is not specific. Mild leukocytosis and anemia were present but do not alter the central interpretation of the case. Taken together, the laboratory profile is consistent with acute coronary thrombosis occurring in a prothrombotic context. Importantly, the subtherapeutic INR is not merely an incidental laboratory abnormality in this patient; it is central to the pathophysiological interpretation and one of the most preventable contributors to the event.

This case also underscores the need for strict long-term anticoagulation management in patients with mechanical valve prostheses. Mechanical valves remain highly durable and effective, but they impose a lifelong requirement for careful anticoagulation monitoring. Deviations from the therapeutic range may not only predispose to stroke or valve thrombosis but can also result in less commonly recognized systemic embolic complications such as coronary embolism and limb ischemia. In this regard, the present case serves as a reminder that anticoagulation follow-up is not a routine administrative issue but a critical determinant of survival and major morbidity.

It would be reasonable, in the broader discussion of such cases, to emphasize the importance of patient education, regular INR monitoring, adherence support, and individualized anticoagulation planning.

The case additionally has value because it demonstrates how careful procedural adaptability can compensate for limited resources. Not all catheterization laboratories have access to IVUS, OCT, or advanced thrombectomy systems. In such settings, operators must rely on available devices and sound clinical judgment. The use of a microcatheter for distal contrast injection is technically simple, relatively accessible, and highly instructive. It may not replace intravascular imaging, but in selected bailout situations it can provide enough anatomic information to safely complete the procedure. This practical message increases the relevance of the report, particularly for interventional cardiologists working in resource-constrained environments.

Several limitations should also be acknowledged. First, definitive proof of coronary embolism is lacking because intravascular imaging, histopathological thrombus analysis, and prior coronary imaging were not available. Second, echocardiographic data regarding prosthetic valve function, ventricular function, and possible intracardiac thrombus were not provided. Third, the absence of detailed follow-up limits conclusions regarding longer-term outcomes, recurrent ischemia, or anticoagulation control after discharge. Nevertheless, case reports are valuable precisely because they highlight important clinical scenarios even when complete datasets are unavailable. In this instance, the coherence between history, laboratory profile, angiographic features, and procedural course makes the case sufficiently persuasive and educational.

Overall, this case contributes to the existing literature in two important ways. First, it reinforces the need to consider coronary embolism in STEMI patients with mechanical heart valves and inadequate anticoagulation, especially when accompanied by other embolic or ischemic manifestations. Second, it demonstrates the procedural usefulness of microcatheter-guided distal vessel visualization as a bailout strategy when conventional reperfusion techniques fail and intravascular imaging is unavailable. These points have direct implications for both diagnosis and management. Clinicians should maintain awareness that not all STEMI lesions are purely atherosclerotic, and interventionalists should remain prepared to adapt technique when standard methods do not achieve reperfusion.

In summary, the discussion generated by this case extends beyond a single successful PCI. It touches on the broader themes of embolic myocardial infarction, anticoagulation failure in mechanical valve recipients, systemic thromboembolic disease, and procedural innovation in complex coronary intervention. For these reasons, the case has substantial educational value and offers a practical message for everyday cardiology and interventional practice.

Conclusion

This case underscores a rare yet clinically significant presentation of acute ST-elevation myocardial infarction (STEMI) likely secondary to coronary embolism in the setting of a mechanical aortic valve and subtherapeutic anticoagulation. It highlights the importance of maintaining a high index of suspicion for non-atherosclerotic causes of myocardial infarction, particularly in patients with known thromboembolic risk factors.

The presence of a markedly subtherapeutic INR (1.25), combined with systemic manifestations of embolic disease such as prior limb amputation and ongoing gangrene, strongly supports a thromboembolic mechanism rather than classical plaque rupture. This reinforces the critical role of strict anticoagulation management in patients with mechanical heart valves, as inadequate anticoagulation remains a preventable cause of life-threatening complications.

From an interventional standpoint, this case demonstrates that conventional PCI strategies, including thrombus aspiration and balloon predilatation, may be insufficient in thromboembolic coronary occlusions due to high thrombus burden and atypical lesion characteristics. In such challenging scenarios, the use of adjunctive techniques becomes essential.

The successful use of a microcatheter (Finecross) to achieve distal vessel visualization represents a key procedural innovation in this case. By confirming the presence of a patent distal lumen, the microcatheter facilitated accurate guidewire positioning and optimal stent deployment, ultimately restoring TIMI III flow. This highlights the value of microcatheter-guided strategies as an effective, accessible, and potentially life-saving bailout option, particularly in settings where advanced intravascular imaging modalities such as IVUS or OCT are not available.

Furthermore, this case emphasizes the need for individualized and adaptive interventional approaches in complex coronary lesions, especially those with non-traditional etiologies. It also reinforces the importance of integrating clinical, laboratory, and angiographic findings to guide diagnosis and management.

In conclusion, clinicians should:

- Consider coronary embolism in STEMI patients with mechanical valves and inadequate anticoagulation
- Recognize subtherapeutic INR as a major modifiable risk factor
- Utilize microcatheter-guided techniques as a practical bailout strategy in difficult PCI cases

This case contributes to the growing body of evidence supporting innovative interventional approaches and highlights the need for heightened awareness of embolic myocardial infarction in high-risk patient populations.

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