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Utilization Of Eptifibatide for Treatment of Acute Coronary Thrombus After Severe Dissections as a Bail-Out Procedure in Anterior Stemi

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Abstract
Acute coronary thrombus formation during primary percutaneous coronary intervention (PCI) for ST-elevation myocardial infarction (STEMI) remains a challenging complication, particularly when associated with severe dissections. Optimal strategies to manage this life-threatening scenario are not well established. We report a case of a 58-year-old male presenting with anterior STEMI who underwent primary PCI. During intervention, severe iatrogenic dissection occurred in the distal left anterior descending artery, resulting in large thrombus burden and impaired distal flow. Multiple aspiration thrombectomy attempts and balloon dilatations failed to resolve the thrombus. As a bail-out strategy, intracoronary eptifibatide bolus followed by intravenous infusion was administered, leading to marked thrombus resolution and restoration of TIMI 3 flow 24 hour. After OCT approach in the next day, patient was stented with good results. The patient remained hemodynamically stable with no periprocedural complications, and follow-up echocardiography demonstrated preserved left ventricular function. Eptifibatide, a glycoprotein IIb/IIIa inhibitor, is generally reserved for high thrombus burden situations in STEMI. Its role as a rescue therapy post-dissection-induced thrombosis is rarely reported. In this case, eptifibatide effectively lysed the thrombus and improved microvascular perfusion, avoiding additional mechanical interventions. This aligns with prior studies indicating eptifibatide's rapid platelet aggregation inhibition and potential to reduce thrombus burden (Marlino et al., 2019; Stone et al., 2001). Intracoronary and intravenous eptifibatide can serve as a safe and effective bail-out therapy for acute coronary thrombus formation secondary to severe dissections in STEMI patients when conventional mechanical strategies fail. Further studies are needed to establish standardized protocols for its utilization in such complex scenarios.

Keywords: *STEMI, eptifibatide, coronary thrombus, dissection, glycoprotein IIb/IIIa inhibitor, bail-out therapy.*

1. INTRODUCTION

Percutaneous coronary intervention (PCI) for chronic total occlusion (CTO) remains one of the most complex procedures in interventional cardiology, with higher risks of procedural complications such as dissection, perforation, and the no-reflow phenomenon. No-reflow, defined as inadequate myocardial perfusion despite successful opening of the epicardial coronary artery [1], is a serious event associated with poor clinical outcomes, including increased rates of myocardial infarction, arrhythmia, and mortality. Its pathophysiology is multifactorial, involving microvascular obstruction due to distal embolization of thrombotic or atheromatous debris, platelet aggregation, and ischemia-reperfusion injury [2], [3].

The management of no-reflow includes pharmacologic agents such as adenosine, nitroprusside, and glycoprotein IIb/IIIa inhibitors. Eptifibatide, a fast-acting intravenous glycoprotein IIb/IIIa receptor blocker, is known to be effective in rapidly inhibiting platelet aggregation, facilitating thrombus resolution, and restoring microvascular flow [4]. Although its routine use has declined with the emergence of potent oral antiplatelet agents, eptifibatide remains valuable in selected high-risk or bailout scenarios, particularly when thrombotic complications are suspected [5].

In addition, intravascular imaging plays a pivotal role in optimizing PCI outcomes, especially in complex lesions such as CTO. Optical coherence tomography (OCT), with its high-resolution cross-sectional imaging, allows precise evaluation of stent expansion, apposition, plaque morphology, and procedural complications such as dissection or intramural hematoma [6].

This case report describes a patient with distal LAD CTO who developed acute no-reflow following stent implantation, successfully managed with intravenous eptifibatide [7]. Subsequent OCT imaging revealed underexpansion, plaque prolapse, and dissection—findings that were crucial for guiding a successful second-stage PCI [8].

2. Case Report

A 68-year-old male presented with a history of intermittent chest pain, primarily occurring during strenuous physical activity. The patient described the chest discomfort as a heavy or tight sensation, sometimes accompanied by shortness of breath. He has a known history of hypertension. On physical examination, his vital signs were within normal limits, with a blood pressure of 137/65 mmHg without pharmacologic support and a heart rate of 55 beats per minute.

Electrocardiography revealed sinus bradycardia at a rate of 55 bpm, normal cardiac axis, T-wave inversions, and poor R wave progression in leads V1 to V4. A chest X-ray showed no significant abnormalities. Transthoracic echocardiography demonstrated normal left ventricular dimensions and volume (EDV 109 mL; LVIDd 4.9 cm), with no evidence of left ventricular hypertrophy (LVDMi 69.3 g/m^2; RWT 0.28). The left ventricular ejection fraction, assessed using the biplane method, was preserved at 48%. Segmental wall motion analysis revealed hypokinesia in the inferoseptal (basal to mid), anterolateral (basal to mid), and anterior (basal) segments, while the remaining segments were normokinetic.

The patient underwent a diagnostic coronary angiography (DCA), which revealed the following findings: the left main (LM) coronary artery was normal. The left anterior descending artery (LAD) showed diffuse disease from the proximal to distal segments, with a chronic total occlusion (CTO) at the distal LAD. The left circumflex artery (LCX) had a non-significant stenosis of approximately 60% in the proximal segment. The right coronary artery (RCA) was dominant and appeared normal, with collateral circulation observed from the right posterior descending artery (RPDA) supplying the distal LAD (Rentrop Grade II).

During the procedure, an ASAHI GAIA Next 3 guidewire was successfully advanced to the distal LAD with the aid of an APT microcatheter, successfully crossing the lesion. Sequential balloon dilatations were performed from distal to proximal LAD: first with a semi-compliant Mecross CTO balloon (1.0 x 15 mm) at 10 atm for 13 seconds, 10 atm for 10 seconds (twice), and then 15 atm for 20 seconds and 15 seconds, respectively, this was followed by further dilatation using a Sapphire 3 balloon (1.5 x 12 mm). A non-compliant Sapphire II balloon (2.0 x 15 mm) dilatations was performed at proximal to mid LAD. A drug-eluting stent (DES), XIENCE Xpedition (Everolimus), sized 2.75 x 33 mm, was deployed in the distal LAD at 10 atm for 18 seconds. Post-deployment, no flow was observed. In response, a bolus loading dose of Eptifibatide was administered intravenously at 180 mcg/kg body weight, followed by a continuous intravenous infusion at a maintenance dose of 2 mcg/kg/min for 18 hours.

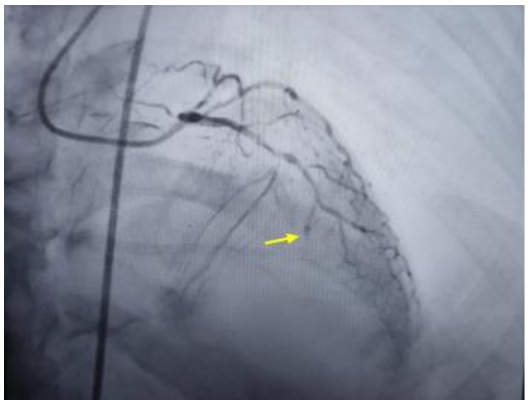

Figure 1. Rao Cranial projection of LAD branch show chronic total occlusion at distal LAD (Yellow Arrow)

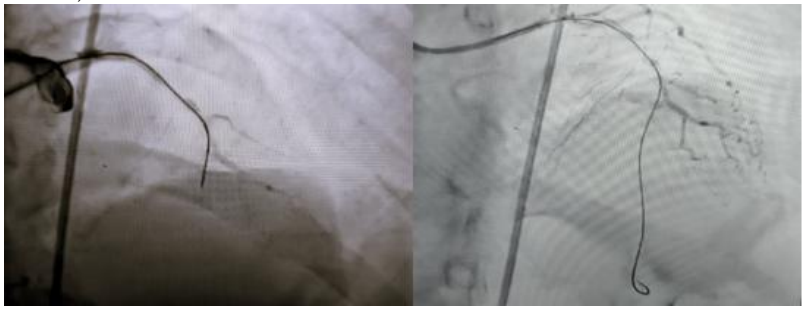

Figure 2. Wiring to distal LAD using ASAHI GAIA Next 3 guidewire + microcatheter

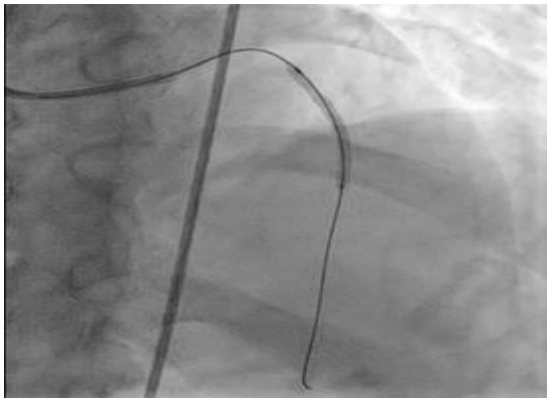

Figure 3. Balloon dilatation from mid to proximal LAD

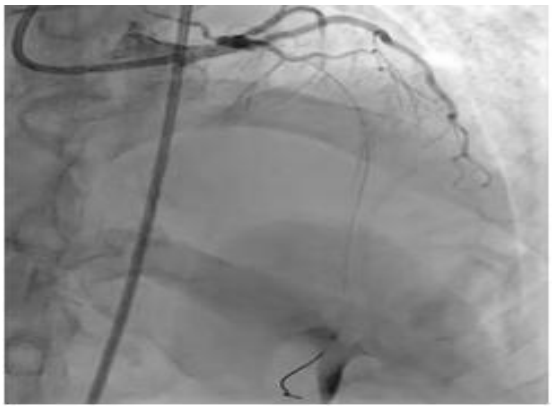

Figure 4. No flow phenomenon after stent deployed at proximal-mid LAD

Optical coherence tomography (OCT) was performed 24 hours after intravenous eptifibatide administration and restoration of TIMI 3 flow to assess lesion morphology and guide further intervention. In the mid-to-distal LAD in-stent segment, the stent appeared well apposed but showed significant underexpansion, with a stent expansion index of 47%. A substantial plaque prolapse was observed, measuring approximately 7.9 mm in length and 1.5 mm in diameter, with a minimum stent area (MSA) of 2.24 mm^2. In the distal after stent segment, OCT revealed the presence of an extraluminal hematoma and a medial dissection, although the stent remained well apposed throughout.

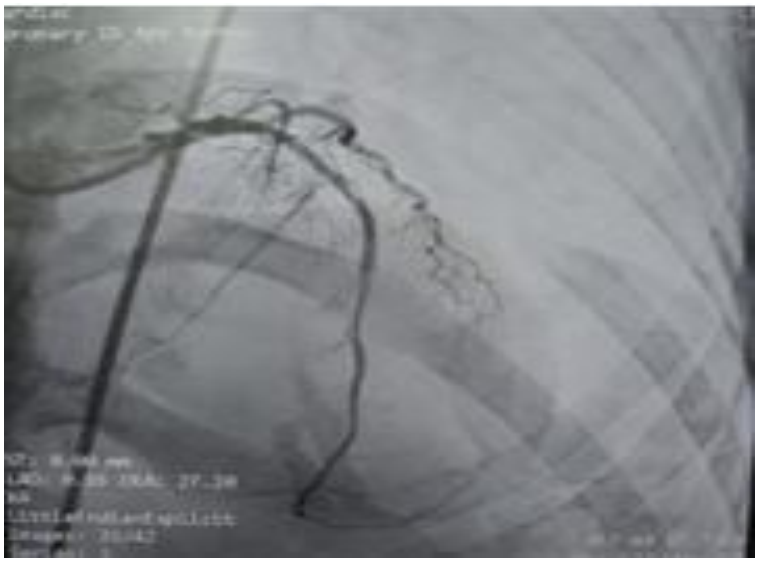

Figure 5. Coronary Angiography of LAD post 24 hours intravenous Eptifibatide

Figure 6. OCT evaluation after 24 hours intravenous Eptifibatide; Tissue Protusion and plaque prolaps inner stent at Proximal LAD.

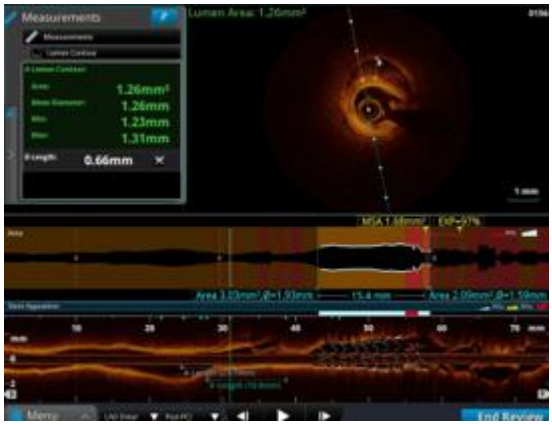

Figure 7. OCT evaluation after 24 hours of intravenous Eptifibatide; Hematoma and dissection at distal LAD.

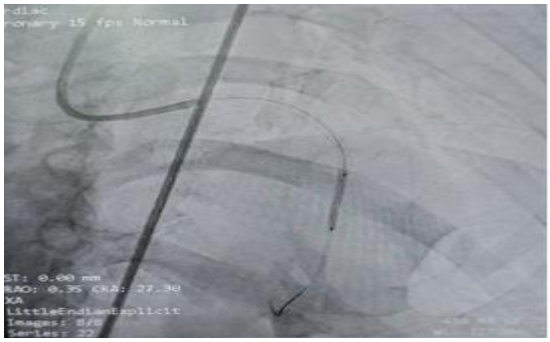

Figure 8. Stent placement at distal LAD, at the site of hematoma and dissection

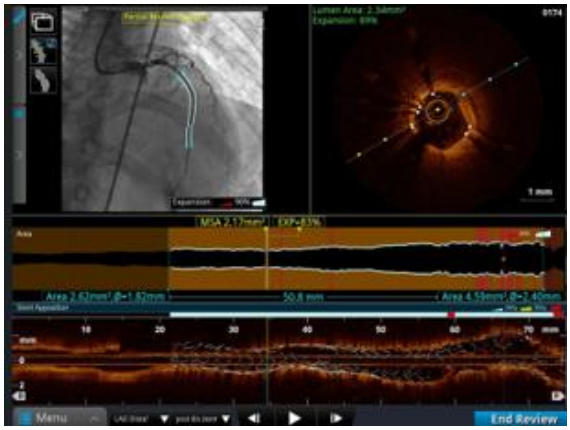

Figure 9. OCT evaluation after stent implantation at distal LAD and Balloon angioplasty at proximal-mid LAD

Following OCT-guided implantation of an overlapping drug-eluting stent in the distal LAD, repeat OCT demonstrated significant improvement. The stent in the mid-to-distal LAD was well apposed and adequately expanded, with an improved expansion index of 83%. Residual plaque prolapse was still present but reduced, measuring 5.2 mm in length with a diameter ranging from 1.16 to 1.5 mm. Importantly, the previously noted extraluminal hematoma and medial dissection in the distal LAD had completely resolved, and stent apposition was optimal. These post-PCI findings confirmed mechanical success and effective lesion coverage, supporting the favorable angiographic and clinical outcome.

3. RESULT AND DISCUSSION

This case highlights a successful multidisciplinary approach to managing acute no-reflow in a complex distal LAD chronic total occlusion (CTO) percutaneous coronary intervention (PCI). The patient developed no-reflow following drug-eluting stent (DES) implantation, which was promptly and effectively managed with intravenous eptifibatide [9]. Subsequent optical coherence tomography (OCT) imaging provided critical insight into the underlying pathology and guided a staged, imaging-optimized re-intervention.

The no-reflow phenomenon is a recognized complication in CTO interventions, particularly when dealing with long, calcified, or diffusely diseased segments [10]. It is characterized by impaired myocardial perfusion despite angiographic evidence of an open epicardial vessel. The underlying pathophysiology involves a combination of distal embolization of atherosclerotic and thrombotic debris, microvascular dysfunction, vasospasm, and ischemia-reperfusion injury [2], [3]. The presence of visible thrombus, prolonged balloon inflation, and plaque prolapse are known contributors.

In this case, no-reflow occurred immediately after stent deployment in the distal LAD CTO lesion. Although mechanical complications such as dissection and hematoma were not initially apparent on angiography, the clinical picture was consistent with thrombotic microvascular obstruction. Eptifibatide, a fast-acting glycoprotein IIb/IIIa receptor inhibitor, was administered as a bolus followed by continuous infusion. This agent blocks the final common pathway of platelet aggregation, thereby preventing fibrinogen cross-linking and reducing thrombus burden [4]. Within 24 hours, the patient showed clinical and angiographic improvement with restoration of TIMI 3 flow, confirming thrombotic no-reflow as the likely mechanism.

OCT performed after stabilization revealed critical mechanical insights [11]. The initial stent was well apposed but significantly underexpanded, with a stent expansion index of 47%. A large prolapsed plaque was present within the stent lumen, which likely served as a nidus for thrombus formation and distal embolization. In addition, the distal segment showed a medial dissection and extraluminal hematoma—findings that were not apparent angiographically. These observations emphasize the limitations of angiography alone in assessing stent-related complications and reinforce the importance of intracoronary imaging [12].

The decision to perform OCT-guided re-intervention led to implantation of a second overlapping DES [13]. Post-procedural OCT showed improved stent expansion (83%), complete resolution of the extraluminal hematoma and dissection, and adequate plaque containment. The favorable OCT and angiographic outcomes validated the imaging-guided approach and likely contributed to long-term procedural success.

This case illustrates several important clinical lessons. First, in the setting of no-reflow—particularly when thrombus is suspected—early use of glycoprotein IIb/IIIa inhibitors remains a valuable therapeutic option. Second, OCT imaging provides essential information that can uncover angiographically silent complications, allowing precise correction of mechanical issues. Lastly, integrating pharmacological and imaging tools in complex PCI cases can dramatically improve outcomes and reduce complications.

4. CONCLUSION

This case underscores the importance of a multimodal approach in the management of acute no-reflow during complex percutaneous coronary intervention, particularly in chronic total occlusion (CTO) cases. The timely administration of intravenous eptifibatide effectively restored coronary flow by addressing thrombotic microvascular obstruction, demonstrating its continued relevance in selected high-risk settings. Furthermore, the use of optical coherence tomography (OCT) provided invaluable diagnostic

insight, uncovering critical mechanical complications such as stent underexpansion, plaque prolapse, and vessel wall injury that were not visible on angiography. OCT-guided reintervention allowed for precise stent optimization and ensured favorable procedural and clinical outcomes. This case highlights the synergistic value of pharmacological and imaging strategies in achieving success in complex coronary interventions.

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