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Low-Dose Slow-Infusion Alteplase for a Thrombus-Dominant ST-Segment Elevation Myocardial Infarction in a Young Adult: A Case Report and Narrative Review

CASE REPORT

INTRODUCTION

Although acute coronary syndrome (ACS) is uncommon in young adults, coronary artery disease (CAD) may develop particularly in the 20-40 age group, even in the absence of conventional risk factors. Genetic, inflammatory, thrombotic, psychosocial, and environmental determinants may contribute to the distinct pathophysiology of premature CAD in this population.[1]

No standardized treatment approach or guideline-directed strategy exists for thrombotic CAD. Reported therapeutic options include emergency coronary artery bypass grafting, stent implantation, intracoronary or intravenous (IV) thrombolytic agents, anticoagulation, potent antiplatelet agents such as glycoprotein IIb/IIIa inhibitors, thrombus aspiration, and conservative management.[2,3] Recently, low-dose slow-infusion tissue plasminogen activator (t-PA) has been reported as a component of a multimodal therapy in selected angiographically thrombus-dominant coronary lesions.[4] Low-dose, slow-infusion t-PA therapy was administered in accordance with the literature.[4,5]

The clinical decision-making process and angiographic outcome are described in a 22-year-old patient with ST-Segment Elevation Myocardial Infarction (STEMI) and a large angiographic thrombus burden who underwent balloon angioplasty followed by low-dose slow-infusion alteplase. This regimen was used as an exceptional, individualized strategy after partial restoration of coronary flow in a hemodynamically stable patient.

LITERATURE SEARCH

To provide clinical context for the present case, a narrative review of the literature was conducted using PubMed/MEDLINE. Because premature myocardial infarction (MI) in young adults may occur through diverse atherosclerotic and non-atherosclerotic mechanisms, multiple independent searches were performed rather than a single predefined Boolean search strategy. The search included combinations of the following terms: premature CAD (PCAD), young MI, young adult STEMI, young adult, coronary angiography, coronary thrombosis, thrombus, thrombotic coronary lesion, young male smoker, and glycoprotein IIb/IIIa inhibitor.

Eligible case reports and case series were reviewed manually, and additional relevant publications were identified through citation tracking (snowball sampling) by screening the reference lists of retrieved articles. Cases with sufficient clinical, angiographic, therapeutic, and follow-up information were included in the descriptive summary presented in Table 1.

CASE REPORT

A 22-year-old male patient presented to the emergency department with severe chest pain that began during sleep. He was conscious, and his blood pressure was in the low-normal range (100/60 mm Hg). He reported intense exercise earlier that

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Table 1. Clinical Characteristics of Patients with Premature Coronary Artery Disease in the Literature

No	Authors	Year	Age/Sex	Clinical Presentation	Coronary Anatomy	Traditional CV Risk Factors	Non-atherOsclerotic Etiologic Factors	Thrombophilic Factors	Genetic Variant	İntervention Treatment, Thrombolytic or Anticoagulant Agent and Dose	Complications and Follow-up
1	Alhroub et al[13]	2025	19/M	Anterolateral STEMI	LM distal 70% LAD osteal 99% Cx osteal 90%	Mild dyslipidaemia (LDL-C: 122 mg/dL, HDL-C: 28 mg/dL)	No	No	No	CABG	4 months/good health
2	Hill et al[14]	2016	16/F	Anterior STEMI	LAD mid 100%	Dyslipidaemia (LDL-C: 159 mg/dl) Obesity (BMI: 32.1 kg/m^2)	No	No	No	PCI	No information
3	Akçay et al[4]	2022	43/M	NSTEMI (Anterior & lateral STdep)	Semi-mobile thrombus leading to 80-90% stenosis in LM	Smoker; family history of premature CAD	No	No	No information	Tirofiban, 80 hours; t-PA dose 25 mg, 6 hours	Repeat coronary angiography after 24 hours revealed that thrombus had disappeared
			54/M	NSTEMI (ST depression in Anterior STdep)	Semi-mobile thrombus formation leading to 70%-80% stenosis in LM	No	Psoriasis, history of deep venous thrombosis	No	No information	Absiximab, 72 hours; t-PA dose 25 mg, 6 hours/25 mg, 25 hours	In the control coronary angiography after 24 hours of the last dose of t-PA, the thrombus has dissolved
			45/M	NSTEMI (STdep in D1, aVL, V5, V6)	Thrombus formation leading to 70%-80% stenosis in mid LAD	Smoker; Family history of premature coronary artery disease	History of deep venous thrombosis	*Factor V Leiden* heterozygote mutation	*Factor V Leiden* heterozygote mutation	Tirofiban, 48 hours; t-PA dose 25 mg, 6 hours/25 mg, 25 hours	In the control coronary angiography after 24 hours of the last dose of t-PA, the thrombus has dissolved
			45/M	NSTEMI (STdep in inferior derivations;	Thrombus formation leading to 80% stenosis in mid LAD	Smoker	No	No	No information	Tirofiban, 48 hours; t-PA dose 25 mg, 6 hours/25 mg, 25 hours	In the control coronary angiography, after 24 hours of the last dose of t-PA, the thrombus has dissolved
			41/M	Inferior STEMI (DII, DIII, aVF)	Intraluminal thrombus leading to 80%-90% stenosis in prox LAD	Smoker	No	No	No information	Tirofiban, 24 hours; t-PA dose 25 mg, 6 hours/25 mg, 25 hours	48 hours after the end of t-PA infusion, control coronary angiography was performed and the thrombus was dissolved completely
4	Schlaifer et al[15]	2001	36/F	Anterolateral STEMI	LM distal, LAD & Cx trombose lesion	No	No	Antiphospholipid syndrome	No	Abciximab 0.25 mg/kg IC 10-min bolus infusion + IV 12-h infusion	No information
5	Sharma et al[20] (A retrospective analysis/5 cases)	2016	39; 56/F	Anterior/ inferior STEMI	LAD proximal/RCA proximal	DM, HTN	Peripartum state	No	No	Optimal medical therapy (antiplatelet, statins, beta-blocker, angiotensin-converting enzyme inhibitors)	SCAD in RCA after 17 years
			18/M	Anterior STEMI	LAD distal	No	No	No	No		5 years
			19/M	Anterior STEMI	LAD Proximal &mid	No	Vigorous activity	No	No		5 years
			40/M	Inferior STEMI	RCA Proximal, mid & distal	DM, HTN	No	↑Homocysteine	No		2 years
			31/M	Anterior STEMI	LAD Proximal & mid	Tobacco	No	↑Homocysteine	No	PCI	2 years
6	Sahoo et al[21]	2026	26/M	Anterolateral STEMI	Large thrombus in the distal LM	Family history of premature coronary artery disease	Nephrotic syndrome secondary to biopsy -proven focal segmental glomerulosclerosis; history of venous thrombosis	No	Heterozygous frameshift variant in *THBD* & a heterozygous missense variant in *CBS*	A reduced-dose bolus fibrinolytic regimen (18 mg reteplase; ½ of standard IV dose) in 30 min apart in divided doses (9 mg/dose diluted in 10 mL 0.9%7normal saline) via an aspiration catheter positioned in the distal LAD and LM+ IV abciximab (0.125 μg/kg/ min for 12 h)	Repeat angiography at 48-h showed complete thrombus resolution in the LAD and partial resolution in the LM with restoration of TIMI III flow. A thrombus in the left ventricular apex (21×27 mm) at 1 month (Systemic anticoagulation with warfarin was initiated)
7	Tudurachi et al[22]	2025	40/M	Anterior STEMI	LAD proximal 100%	Dyslipidaemia (LDL-C: 188 mg/dL)	Sedentary lifestyle	No	No	PCI	No information
			27/M	Anterolateral STEMI	Subocclusive lesion in the LAD	Smoker	User of ecstasy and cocaine	No	No	PCI	Favorable early clinical outcome
			50/F	Inferior STEMI	SCAD in the third segment of LAD (70% lesion)	Dyslipidaemia (LDL-C: 162 mg/dL); HTN	No	No information	No information	Conservative treatment (fail) + PCI	At discharge, a slightly circumferential pericardial effusion.
			35/M	Postero-infero-lateral STEMI	Extensive 3-vessel CAD (LAD 90%, Cx CTO, RCA %95)	No	HIV infection	No	No information	CABG	No

8	Padda et al[23]	2024	19/M	Ventricular fibrillation / inferior STEMI	Obtuse marginal 1 100%	No	Marijuana use	No	No information	PCI	No
9	Kumral et al[24]	2023	36/M	Anterior STEMI	Subtotal dissected lesion in the LAD	No	No	No	A heterozygous pathogenic variant, *pGly1074Arg* mutations in the *COL3A1* gene, leading to a diagnosis of vascular Ehlers-Danlos syndrome	PCI	No
10	Melhem Jr et al[25]	2020	26/M amateur bodybuilder	Anterior STEMI	LAD 100%	Dyslipidaemia (LDL-C: 204 mg/dL), family history of premature coronary artery disease	He had been self-injecting t trenbolone acetate, stanozolol, and testosteron; ↓Luteinizing and follicle-stimulating hormones; estrone and estradiol and ↑dehydroepiandrosterone (DHEA)	No	No information	PCI	4 months/symptomatic
11	Ganta et al[26]	2022	38/M	Anterolateral STEMI	LAD osteal 100%	Smoker and alcohol abuse	High platelet count	No	Essential thrombocythemia; *JAK2 V617F* mutation	PCI	No
12	Zhang et al[27]	2025	28/M	Anterior STEMI	RCA mid 100%	Smoker	Thromboangiitis obliterans	No	No	Conservative management	3 years/symptomatic
13	Al Yaarubi et al[28]	2020	26/M	Inferior STEMI	Thrombotic occlusion of the mid-segment of the right posterior interventricular branch.	Smoker	No	Protein C deficiency	No	Aspiration thrombectomy + Rivaroxaban o.d	18 months later/ symptomatic
14	Tresenza et al[29]	2022	22/F	Anterior STEMI	LAD lesion	HTN	Systemic lupus erythematosus; nephropathy	No	No information	PCI	No
15	Soussi et al[30]	2025	27/M	Anterior STEMI	No obstructive coronary lesions/ MINOCA (confirmed by CMRI.)	DM, smoker, obesity, family history of premature coronary artery disease	No	No	No information	Due to the unavailability of a catheterization lab within 120 min of first medical contact, fibrinolysis was initiated + Coronary angiography; Conservative treatment	İnitially experienced symptomatic improvement; recurrent angina 2 weeks later; no new ischemic changes, Trimetazidine and diltiazem were added to treatment
16	Bengel et al[31]	2023	33/F	Inferior STEMI	Thrombotic occlusion of the Cx	No	Further diagnostic work up identified a highly active ulcerative pancolitis	No	No information	Bolus of Tirofiban; Due to the localization of the thrombus in the vascular periphery, stent implantation was not feasible. DAPT (acetylsalycylic acid and ticagrelor)	Lower gastrointestinal bleeding; Dressler syndrome
17	Khan et al[32]	2024	37/M	Inferior STEMI	RCA ostial aneurysm of ~t 8 mm in size with heavy thrombus formation & occluded distal PDA	No	No	No	No information	2 boluses of a tirofiban, followed by a tirofiban infusion (12.5 mg) in a 250 mL solution at a rate of 0.15 micrograms/kg/minute for 6-hours; enoxaparin 70 mg bid, rivaroxaban 20 mg od and clopidogrel	No information after discharge
18	Present case	2026	22/M	Anterior STEMI	LAD osteal trombotic lesion 99%	Dyslipidaemia (LDL-C: 126 mg/dL) and smoker	Vigorous activity	No	No pathogenic variant (*SGCD* VUS of uncertain clinical significance)	Low-dose slow infusion t-PA therapy	9 months/symptomatic

CABG, coronary artery bypass grafting; CMRI, cardiac magnetic resonance imaging; CTO, chronic total occlusion; CV, cardiovascular; Cx, circumflex artery; DAPT, dual antiplatelet therapy; DM, diabetes mellitus; F, female; HDL-C, high-density lipoprotein cholesterol; HTN, hypertension; IV, intravenous; LAD, left anterior descending artery; LDL-C, low-density lipoprotein cholesterol; LM, left main coronary artery; M, male; NSTEMI, non-ST-segment elevation myocardial ınfarction; o.d, once a day; PCI, percutaneous coronary intervention; PDA, posterior descending artery; RCA, right coronary artery; SCAD, spontaneous coronary artery dissection; SGCD VUS, SGCD gene variant of uncertain significance; STEMI, ST-segment elevation myocardial ınfarction; TIMI, thrombolysis in myocardial infarction; t-PA, tissue plasminogen activator.

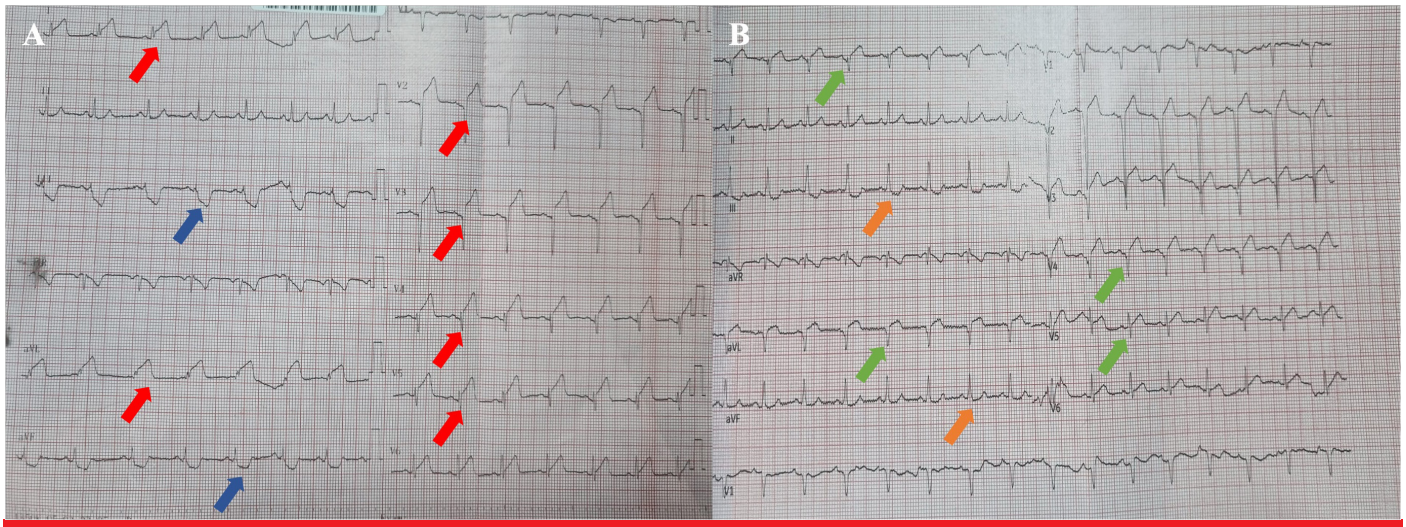

Figure 1. Electrocardiogram images. (A) The initial electrocardiogram shows ST-segment elevation in the anterior and lateral leads (red arrows) and reciprocal ST-segment depression in the inferior leads (blue arrows). (B) The post-percutaneous coronary intervention control electrocardiogram demonstrates regression of the ST-segment elevation (green arrows) and resolution of the reciprocal ST-segment depression (orange arrows).

day. The electrocardiogram was consistent with an anterolateral MI (Figures 1A-B). Following the administration of antiplatelet and anticoagulant therapy (180 mg ticagrelor, 300 mg acetylsalicylic acid, and 5000 IU IV heparin), the patient was immediately transferred to the angiography laboratory. He had no known medical history or family history of cardiovascular disease. He was a smoker (5 pack-years), denied illicit drug use, and had a body mass index of 24.7 kg/m^2.

Coronary angiography performed via the radial approach revealed normal right coronary and circumflex arteries, while the left anterior descending (LAD) artery exhibited a suspected thrombus-dominant ostial lesion causing 99% luminal obstruction. After crossing the LAD lesion with a floppy guidewire (PTCA guidewire, Shunmei®), predilatation was performed using a 2.5 × 15 mm semi-compliant balloon (Solarice, Medtronic®) (Figure 2). Repeated thrombus aspiration yielded no visible thrombotic material. Manual thrombus aspiration was performed using a conventional coronary aspiration catheter (APT Medical®); a mechanical aspiration system such as Penumbra was not utilized. Because of the angiographically suspected high thrombus burden and concern about distal embolization, intravascular imaging (IVI), including intravascular ultrasound (IVUS) or optical coherence tomography (OCT), was not performed. Given the angiographic suspicion of a predominant thrombotic burden and the restoration of coronary flow after balloon angioplasty, a pharmacological thrombus resolution strategy was favored over immediate stent implantation in this hemodynamically stable young patient. Following evaluation by the heart team, the patient who achieved thrombolysis in myocardial infarction (TIMI) grade 2 flow, was hemodynamically stabilized, and reported improvement in chest pain was transferred to the coronary intensive care unit (ICU) for low-dose slow-infusion thrombolytic therapy (25 mg t-PA over 6 hours followed by an additional 25 mg t-PA over 25 hours intravenously). The alteplase regimen (25 mg over 6 hours followed by 25 mg over 25 hours) was selected in accordance with previously published low-dose slow-infusion protocols. Transthoracic echocardiography (TTE) showed hypokinesis of the left ventricular anterior and apical segments, left ventricular ejection fraction (LVEF) of 30%, normal valve structures, normal ascending aorta, and normal pericardium (Figure 3).

In the coronary ICU, the patient received guideline-directed medical therapy, including dual antiplatelet therapy (DAPT), low-molecular-weight heparin, high-intensity statin therapy, and guideline-directed medical therapy (GDMT). Vital signs remained stable during thrombolytic therapy. Laboratory parameters are shown in Table 2. During intensive care monitoring, the patient remained free of chest pain. On day 5, repeat TTE demonstrated an LVEF of 30%, with persistent anterior wall hypokinesia but no mechanical complications. A follow-up coronary angiography performed on day 5 revealed normal coronary arteries (Figure 4). Although follow-up IVI could have provided valuable mechanistic information, it was not pursued because the vessel appeared angiographically normal and the treating heart team considered that additional imaging was unlikely to alter immediate clinical management.

A comprehensive thrombophilia workup was performed, including protein C and protein S activity, antithrombin III levels, plasminogen activator inhibitor 1, antiphospholipid antibodies (lupus anticoagulant, anticardiolipin, and anti-β2 glycoprotein I), homocysteine levels, and screening for inherited thrombophilia (Factor V Leiden variant). All results were within normal limits. Toxic exposures such as drug use or chronic infections like HIV, as well as rheumatologic diseases, vasculitis, and hematologic disorders, were ruled out based on patient history, physical examination, laboratory

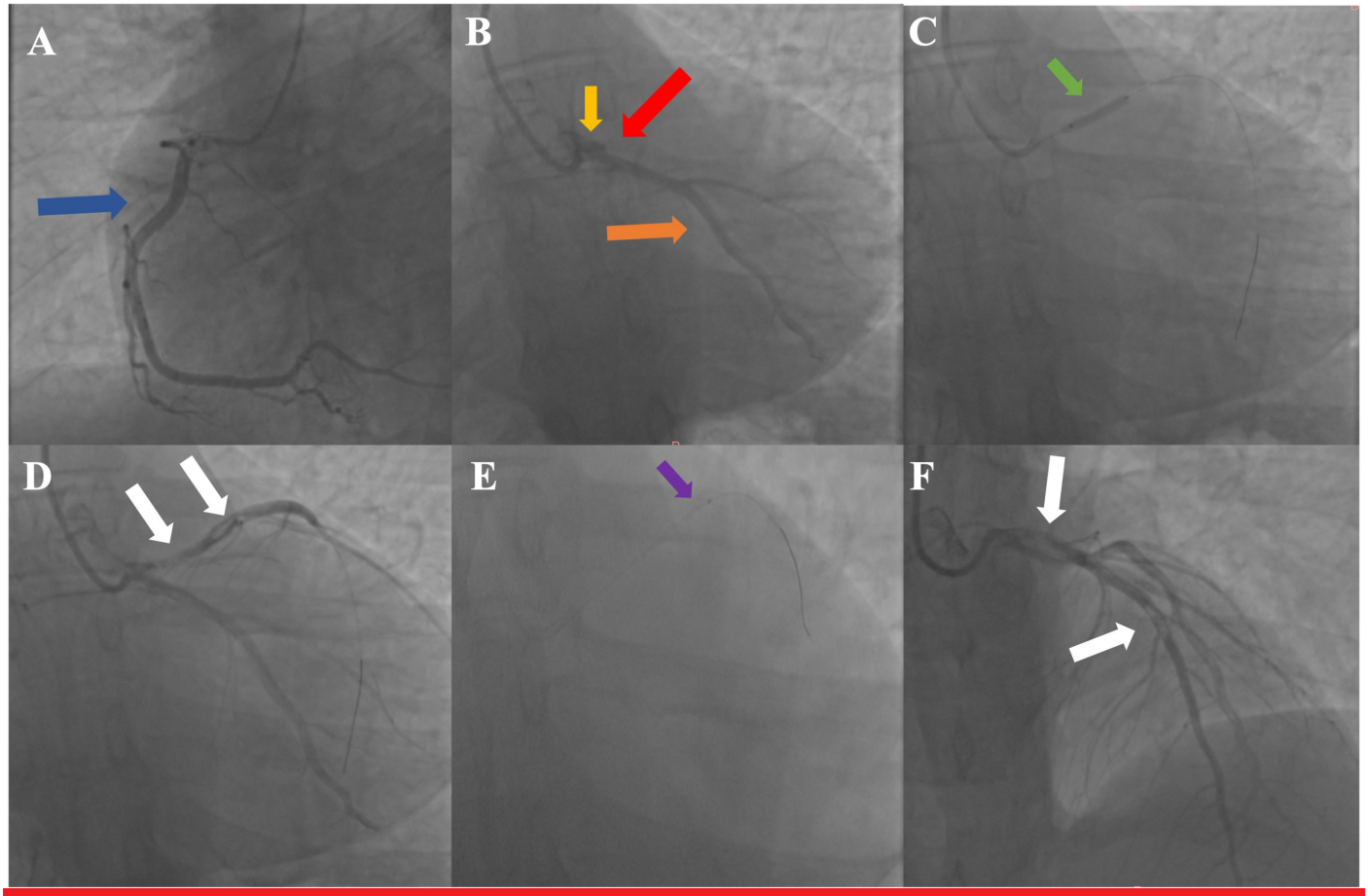

Figure 2. Emergency coronary angiography images. (A) The right coronary artery appears normal (blue arrow). (B) The left main (yellow arrow) and circumflex artery (orange arrow) are normal, whereas the LAD is completely occluded at the ostium (red arrow). (C) After the LAD lesion was crossed with a floppy wire, balloon angioplasty was performed using a 2.5 × 15 mm balloon (green arrow). (D) In the caudal view, dense thrombus is observed in the ostial and mid segments of the LAD following balloon angioplasty (white arrow). (E) Aspiration of the thrombus using a coronary aspiration catheter is shown (purple arrow). (F) In the cranial view, a thrombus migrating distally in the ostial and mid LAD segments is visualized (white arrow). LAD, left anterior descending artery.

testing, and toxicology screening. Lipoprotein(a) levels were not assessed during hospitalization. Genetic testing identified a heterozygous variant of uncertain significance (VUS) in the SGCD *gene (c.154A>G, p.Met52Val)*. As this variant has no established association with PCAD or coronary thrombosis, it was considered an incidental finding without relevance to the patient's clinical presentation. He was discharged on DAPT (acetylsalicylic acid 100 mg daily and ticagrelor 90

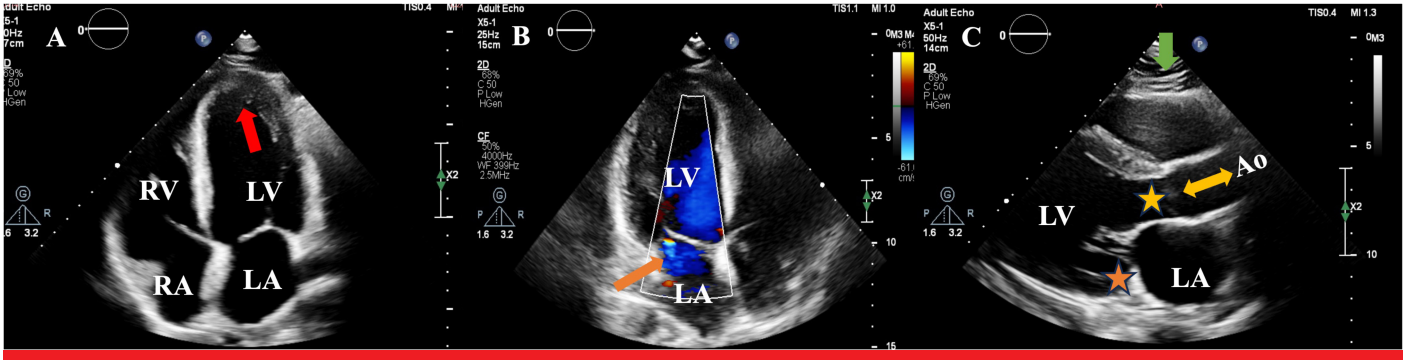

Figure 3. Transthoracic echocardiography images. (A) In the apical 4-chamber view, hypokinesia of the apical segment is observed (red arrow). (B) Color Doppler echocardiography demonstrates mild mitral regurgitation (orange arrow). (C) In the parasternal long-axis view, the aortic (yellow star) and mitral (orange star) valves appear normal, with no pathology detected in the ascending aorta (double-headed yellow arrow) or pericardium (green arrow).

Table 2. Laboratory Test Results of the Patient

Parameters	Day 0	Day 5
WBC (10^3/μL)	18.7	6.9
Hgb (g/dL)	16.1	13.1
PLT (10^3/μL)	349	306
Glucose (mg/dL)	117	
Creatinine (mg/dL)	0.84	0.84
Sodium (mmol/L)	136	139
Potassium (mmol/L)	4.6	4.5
Calcium (mg/dL)	9.7	8.9
ALT (U/L)/AST (U/L)	31/27	29/24
D-Dimer (μg/mL)	0.53	
CRP (mg/L)	5.8	
HbA1c (%)	6	
hs-TnT (μg/L, ref 0-0,34)	>50	33.7
BNP (pg/mL)	225.4	175.7
Total cholesterol (mg/dL)	174	
LDL (mg/dL)	126	
HDL (mg/dL)	30	
Triglyceride (mg/dL)	86	
Anti-HCV	Negative	
Anti-HIV	Negative	
HbsAg	Negative	
Fibrinojen (mg/dL)	417.6	
TSH (uIU/mL)	0.97	
Romatoid Factor (IU/mL) ANA, ANCA, anti ds-DNA, SS-A, SS-B, Scl70, Jo-1, APA	<20 Negative	

ALT, alanine transaminase; ANA, antinuclear antibody; ANCA, antineutrophil cytoplasmic antibodies; Anti-dsDNA, anti-double stranded DNA; Anti-HCV, hepatitis C virus antibody; Anti-HIV, human immunodeficiency virus antibody; APA, anti-phospholipid antibody; AST, aspartate aminotransferase; BNP, B-type natriuretic peptide; Crp, C-reactive protein; Hgb, hemoglobin; HbA1c, hemoglobin A1C; hs-TnT, high-sensitivity cardiac troponin; HDL, high-density lipoprotein; LDL-C, low-density lipoprotein; PLT, platelet count; Scl70, scleroderma antibodies; SS-A/B, Sjögren's syndrome antigen A/B; TSH, thyroid-stimulating hormone; WBC, white blood count.

mg twice daily), high-intensity statin therapy, and GDMT. At the 9-month follow-up, he remained asymptomatic without recurrent chest pain or ischemic events. Transthoracic echocardiography demonstrated improvement in LVEF to 45%, indicating partial recovery of systolic function. He remains on DAPT and GDMT, with improved exercise tolerance and no limitations in daily activities.

DISCUSSION

Young patients with MI require a comprehensive etiological evaluation because PCAD may involve both conventional and nontraditional risk factors; the diagnostic framework proposed by Kayikcioglu et al[6] provides a useful approach to this assessment. In the present patient, smoking and mild dyslipidemia (LDL-C, 126 mg/dL) were the only identified conventional modifiable risk factors. Lipoprotein(a), an established risk factor for PCAD, was not measured during hospitalization; although its measurement would not have altered acute management, it remains relevant to the etiological assessment of young patients with MI.[7] Because inherited and acquired thrombophilic disorders, including hyperhomocysteinemia, may contribute to MI in young patients, a comprehensive thrombophilia evaluation was performed, with all results, including homocysteine, within normal limits.[8,9]

Acute coronary syndrome in young patients may result from heterogeneous mechanisms beyond classical plaque rupture, including spontaneous coronary artery dissection (SCAD), plaque erosion, vasospasm, inflammation, and non-atherosclerotic thrombosis. These may present as thrombus-dominant lesions with minimal residual stenosis. Importantly, angiography alone may be insufficient to distinguish thrombus, intramural hematoma, or plaque pathology, particularly in cases mimicking type 2 or 3 SCAD.[10] Intravascular ultrasound or OCT can provide greater diagnostic accuracy in these cases.[11] Given the patient's very young age, limited atherosclerotic burden, and ostial LAD involvement, SCAD remained an important differential diagnosis. As IVI was not

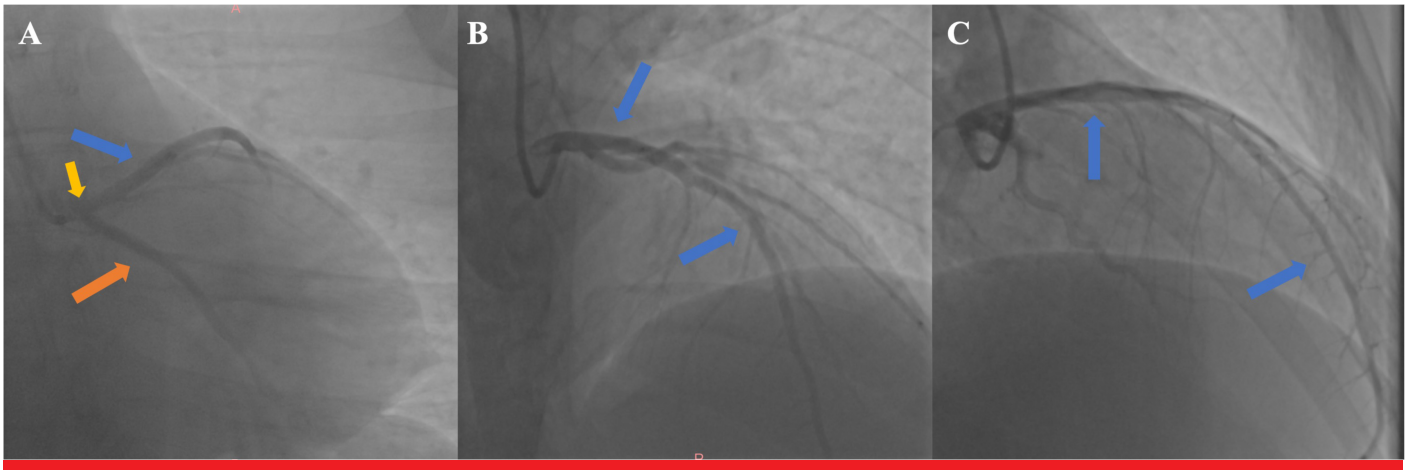

Figure 4. Follow-up coronary angiography images. (A) In the caudal view, the left main (yellow arrow), circumflex artery (orange arrow), and LAD appear normal, with complete resolution of the thrombus (blue arrow). (B) In the cranial view, the LAD and its branches are visualized as normal. (C) In the right cranial view, the LAD is also seen to be normal (blue arrow). LAD, left anterior descending artery.

performed during either the index procedure or follow-up angiography, SCAD and other underlying mechanisms could not be definitively excluded. The absence of IVI during both procedures represents a major limitation of the present report. Nevertheless, such lesions are encountered when imaging is unavailable, requiring decisions based on angiography and clinical status. Although complete thrombus resolution after thrombolysis supports a thrombus-dominant phenotype, it does not establish the underlying mechanism. This case should therefore be interpreted as a therapeutic observation rather than definitive pathophysiological evidence.

Current guidelines recommend primary percutaneous coronary intervention (PCI) as the standard of care for STEMI.[12] However, management is challenging in cases with predominant large thrombus burden without fixed atherosclerotic stenosis. Cases identified in the structured literature review illustrate substantial etiological and therapeutic heterogeneity of MI in young adults (Table 1), with no definitive cause established despite extensive diagnostic evaluation in several reports. Depending on the underlying etiology, angiographic findings, thrombus burden, and hemodynamic status, treatment strategies have included primary PCI with or without stenting, coronary artery bypass grafting, glycoprotein IIb/IIIa inhibitors, thrombolysis, and conservative medical therapy.

Published reports demonstrate diverse management strategies for PCAD. Alhroub et al[13] performed surgical revascularization in a 19-year-old patient, whereas Hill et al[14] treated a 16-year-old STEMI patient with PCI.[13,14] Schlaifer et al[15] achieved successful thrombus resolution using intracoronary abciximab (0.25 mg/kg) followed by a 12-hour IV infusion in a 36-year-old patient who had suffered an MI due to a thrombosed lesion in the distal main coronary. Boniface et al[16] reported successful thrombolysis in 2 young patients with cocaine-associated MI. Akçay et al[4] also reported favorable outcomes with low-dose slow-infusion t-PA in 5 patients with PCAD who presented with acute thrombotic coronary lesions.

Similarly, in this case, the absence of IVI necessitated treatment decisions based primarily on angiographic findings. The decision to defer ostial LAD stenting was guided by the angiographic appearance of a suspected thrombotic lesion, restoration of TIMI 2 flow after balloon angioplasty, and the patient's hemodynamic stability. In young patients, avoiding unnecessary stent implantation may reduce the lifelong risks of stent thrombosis, restenosis, and prolonged DAPT.[17] However, this strategy was highly individualized and should not be considered standard practice.

Low-dose thrombolysis is an established concept; higher-dose regimens were developed to achieve more rapid reperfusion, particularly before the primary PCI era or in unstable patients.[18] Unlike bolus fibrinolytics such as reteplase, alteplase allows controlled infusion and dose adjustment, which may be advantageous in hemodynamically stable patients with high thrombus burden. However, bleeding remains a major concern.[19] In selected hemodynamically stable patients with partial restoration of coronary flow, a slower and lower-dose thrombus-resolution strategy may represent a reasonable individualized approach. Although prolonged low-dose alteplase infusion has been associated with favorable safety profiles in small case series, the available evidence remains limited, and robust comparative data are lacking.[4] In this case, thrombolytic therapy was favored as the 22-year-old patient was hemodynamically stable, had no history of bleeding, stroke, hypertension, or other contraindications to fibrinolysis. No bleeding occurred during hospitalization. Nevertheless, this favorable outcome should not be interpreted as evidence that prolonged low-dose thrombolysis is universally safe, and careful patient selection remains essential when considering this individualized therapeutic approach.

An important consideration is that complete angiographic thrombus resolution cannot be attributed solely to low-dose slow-infusion alteplase. Balloon angioplasty restored partial coronary flow before initiation of fibrinolytic therapy, and the patient simultaneously received DAPT, anticoagulation, and GDMT. Therefore, the favorable angiographic outcome should be interpreted as the result of a combined interventional and pharmacological treatment strategy rather than the isolated effect of alteplase. The independent therapeutic contribution of alteplase cannot be determined from a single observational case.

It is important to emphasize that the observation is based on a single case and limited literature evidence. Therefore, low-dose slow-infusion thrombolytic therapy cannot be recommended as a routine treatment strategy. Instead, this case should be interpreted as hypothesis-generating and may encourage further investigation into individualized management approaches for selected patients with angiographically thrombus-dominant coronary occlusion.

CONCLUSION

In this carefully selected, hemodynamically stable young patient, low-dose slow-infusion alteplase was used as an individualized adjunctive strategy after partial restoration of coronary flow. Because intracoronary imaging was not performed, the underlying coronary mechanism could not be determined. This case should therefore be interpreted as a hypothesis-generating therapeutic observation rather than evidence supporting routine use of this regimen in STEMI.

Informed Consent: Written informed consent was obtained from the patient.

Declaration of Interests: M.T. has no conflict of interest to declare. M.K. received research grants, honoraria for consultancy or lectures/speaker's bureau from Abbott, Abdi-Ibrahim, Amgen, Chiesi, AstraZeneca, Daiichi-Sankyo, Ionis pharmaceuticals, Medpace, Libtherapeutics, Lilly, MSD, Novartis, NovoNordisk, Pfizer, Recordati, Servier, and Ultragenyx.

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