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Anterior-Wall ST-Elevation Myocardial Infarction and Intracoronary Thrombolysis with Tenecteplase in a Young Adult; Massive Coronary Thrombus Burden: A Case Report

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Abstract

Background: Acute myocardial infarction (AMI) in adults younger than 45 years is rising globally, and in South Asian populations it is a leading cause of disability-adjusted life-years lost. The pathophysiology is often distinct. Dyslipidemia, and in particular an elevated lipoprotein(a), together with hyperhomocysteinemia, thrombophilia and pro-thrombotic lifestyle factors, makes a greater contribution in older patients. Angiography frequently demonstrates a high thrombus burden in an otherwise minimally diseased coronary tree. In such patients, primary Percutaneous Coronary Intervention (PCI) alone may be complicated by distal embolization, no-reflow and stent thrombosis. Adjunctive strategies such as manual aspiration, glycoprotein IIb/IIIa inhibitors and, in selected cases, Intracoronary (IC) thrombolysis remain relevant.

Case presentation: We report a 38-year-old previously healthy South Indian man who presented with anterior-wall ST-Elevation Myocardial Infarction (STEMI, Killip class I). Emergency coronary angiography demonstrated single-vessel disease with an 80–90% thrombotic lesion extending from the distal left main coronary artery into the proximal Left Anterior Descending artery (LAD), with distal TIMI II flow. Given the massive thrombus burden with preserved distal flow, 10 mg of IC tenecteplase was administered through the guide catheter into the left coronary system. A planned check angiogram on day 5 demonstrated complete recanalization of the LAD with no residual thrombus, and the patient was discharged on guideline-directed medical therapy without stent implantation. Metabolic evaluation identified marked elevation of lipoprotein(a) (151.5 nmol/L; reference 0–75), homocysteine (33.3 µmol/L; reference 5.4–16.2), LDL cholesterol (176 mg/dL) and mildly elevated thyroid-stimulating hormone (4.9 µIU/mL), providing a mechanistic explanation for coronary thrombosis in a young patient without traditional risk factors.

Conclusion: This report contextualizes the case within contemporary evidence on young MI, non-traditional risk factors and IC thrombolysis, and proposes an algorithmic approach for the young STEMI patient presenting with a massive coronary thrombus burden.

Keywords: Young myocardial infarction, ST-elevation myocardial infarction, Intracoronary thrombolysis, Tenecteplase, Coronary thrombus burden, Lipoprotein(a), Hyperhomocysteinemia, Primary percutaneous coronary intervention, Secondary prevention

Introduction

Acute myocardial infarction (AMI) in young adults represents a distinct and increasingly important clinical phenotype [1,2]. It is variably defined in the literature as onset at 45 years or younger, or at 55 years or younger for women and 45 years or younger for men. Contemporary registry data from the United States (Arora et al., ARIC surveillance, 2019) demonstrate that the proportion of AMI hospitalizations occurring in adults aged 35 to 54 years rose from 27% in 1995–1999 to 32% in 2010–2014 [3]. In a nationwide United States analysis of 1,462,168 young adults hospitalized with a first Myocardial Infarction (MI), Yandrapalli and colleagues reported that 90.3% of those aged 18 to 44 years had at least one modifiable cardiovascular risk factor, the commonest being smoking (56.8%), dyslipidemia (51.7%) and hypertension (49.8 %) [1]. In India and other South Asian populations, both the absolute burden and the population-attributable fraction of premature coronary events are substantially higher than the European or East-Asian populations, reflecting a combination of adverse lipid phenotypes, higher genetically-driven lipoprotein(a) levels, insulin resistance, and dietary and lifestyle factors [4].

The INTERHEART case-control study identified nine classical modifiable risk factors, namely smoking, dyslipidemia, hypertension, diabetes mellitus, abdominal obesity, adverse dietary and psychosocial factors, physical inactivity, low fruit and vegetable intake, and alcohol excess. Together these accounted for approximately 90% of the population-attributable risk of a first MI in men and 94 % in women [5]. Beyond these, young patients with AMI are more likely than older patients to have identifiable non-traditional risk factors. These include a markedly elevated lipoprotein(a), hyperhomocysteinemia, inherited or acquired thrombophilia, autoimmune inflammatory disease, cocaine or amphetamine use, and pregnancy-related coronary events [1,2].

Primary Percutaneous Coronary Intervention (PCI) is the reperfusion strategy of choice for ST-elevation myocardial infarction (STEMI), with a Class I recommendation in both the 2023 ESC Acute Coronary Syndrome guidelines and the 2021 ACC/AHA/SCAI coronary revascularization guidelines [6,7], and reaffirmed in the 2025 ACC/AHA/ACEP/NAEMSP/SCAI acute coronary syndrome guideline, which is now the current United States guidance for acute coronary syndromes [8]. However, in patients with a large intracoronary thrombus burden (TIMI Thrombus Grade 4–5), immediate stent deployment may be complicated by distal embolization, no-reflow and subsequent stent thrombosis [9], and Sianos and colleagues reported that a large thrombus burden independently predicted mortality (hazard ratio 1.76) and major adverse cardiac events (hazard ratio 1.88), with angiographic stent thrombosis occurring in 8.2% versus 1.3% at two years [10]. In this angiographic setting, adjunctive intracoronary pharmacotherapy continues to be used at the discretion of the operator, and the small randomized trial by Sezer and colleagues (2007) provided proof-of-concept that a single intracoronary bolus of a fibrinolytic agent following primary PCI can improve coronary microvascular function without increasing bleeding risk [11].

We report the case of a 38-year-old previously healthy South Indian man who presented with anterior-wall STEMI, was found to have a massive thrombotic lesion involving the distal left main coronary artery and proximal LAD, and was successfully treated with intracoronary tenecteplase, achieving complete angiographic recanalization. We use this case as the anchor for a contemporary review of the literature and propose an algorithmic approach for the young STEMI patient presenting with a heavy coronary thrombus burden. The instructive value of this case lies in three areas. First, it illustrates the recognition of a massive thrombotic burden with preserved distal flow as a distinct angiographic phenotype in a young adult. Second, it demonstrates intracoronary tenecteplase as a stent-sparing reperfusion option in this specific setting. Third, it shows how a structured search for non-traditional risk factors can uncover the mechanistic substrate and direct secondary prevention.

Case Presentation

Written informed consent for the publication of this case report, including the anonymized clinical details, laboratory data, and coronary angiographic images, was obtained from the patient. Formal institutional ethics committee approval was not required for a single de-identified case report at our institution.

History of presenting illness

A 38-year-old previously healthy gentleman, a resident of Thiruvallur district in the state of Tamil Nadu, southern India, was brought to the emergency department of our tertiary-care university hospital on 5th March 2026 with a burning, retrosternal chest discomfort of several hours' duration that had begun in the early morning. The pain was initially of moderate intensity, non-radiating, and was associated with three episodes of non-bilious vomiting and two episodes of loose stools during the morning, features that in the immediate pre-hospital setting had prompted a preliminary diagnostic consideration of acute gastroenteritis rather than an acute coronary syndrome. He had first attended a peripheral hospital at approximately 7:15 p.m., where loading doses of dual antiplatelet therapy and a high-intensity statin were administered on the basis of an outside-hospital 12-lead electrocardiogram that demonstrated sinus rhythm with 1:1 atrioventricular conduction, T-wave inversion in leads II, III and aVF, and ST-segment elevation in leads I, aVL and V4–V5 (**Figure 1A**). He was subsequently referred to our institution with ongoing chest pain, reported to be of approximately 20–30% of its peak intensity at the time of transfer.

Past medical, personal, family and lifestyle history

The patient had no known cardiovascular comorbidities: no prior history of systemic hypertension, diabetes mellitus, dyslipidemia, ischemic heart disease, cerebrovascular disease or peripheral arterial disease. His only past surgical history was an operative repair of a perforated duodenal ulcer performed in 2012. He was a lifelong non-smoker, did not consume alcohol regularly, and denied use of recreational or illicit drugs including cocaine and amphetamines. Family history was non-contributory for premature coronary artery disease, sudden cardiac death, or inherited thrombophilia. His socioeconomic profile corresponded to modified Kuppuswamy Class II.

Clinical examination on admission

On presentation the patient was conscious, alert, orientated and hemodynamically stable. He was afebrile, with a regular pulse rate of 80 beats per minute, a respiratory rate of 20 breaths per minute, a blood pressure of 110/70 mmHg, and oxygen saturation of 100% on room air. General examination revealed no pallor, icterus, cyanosis, clubbing,

peripheral lymphadenopathy, pedal oedema, or thyromegaly. Cardiovascular examination demonstrated normal first and second heart sounds with no added sounds and no audible murmur; the jugular venous pressure was not elevated. Respiratory examination confirmed bilaterally symmetrical air entry with clear breath sounds. Neurological and abdominal examinations were unremarkable. The patient was classified as Killip Class I.

Investigations

The admission 12-lead electrocardiogram at our institution confirmed an acute anterior ST-elevation myocardial infarction, with ST-segment elevation in leads V2 to V6, I and aVL (**Figure 1B**). The complete set of admission and inpatient laboratory investigations, cardiac biomarkers, echocardiographic findings and discharge medications is summarized in **Table 1**. Clinical events from symptom onset to hospital discharge, biomarker levels versus assay upper-reference limits, and lipid profile versus current secondary-prevention guideline thresholds are jointly depicted in **Figure 2**.

Coronary angiography and intracoronary thrombolysis

Emergency coronary angiography was performed via a right-radial approach using TIGER diagnostic catheter, with 30 mL of iodinated non-ionic contrast (iohexol; Omnipaque, GE Healthcare). Coronary anatomy demonstrated left-dominant circulation. The left main coronary artery was a good-sized vessel that bifurcated into the LAD and left circumflex artery (LCX). An 80–90% thrombotic lesion was identified extending from the distal left main into the proximal LAD, with distal TIMI II flow through the LAD; the mid and distal LAD and the first diagonal branch were free of significant disease. The LCX was a good-sized, dominant vessel that continued as the posterolateral and posterior descending branches; the first obtuse marginal branch (OM1) was of moderate size and free of significant disease. The right coronary artery was a moderate-sized, non-dominant vessel free of significant disease (**Figures 1D–1F**).

Table 1. Complete clinical, laboratory, echocardiographic and pharmacological profile of the patient at admission, during in-hospital stay and at discharge.

Parameter	Value
DEMOGRAPHIC AND CLINICAL PROFILE	
Age	38 years
Sex	Male
Ethnicity	South Indian
Socioeconomic class (modified Kuppuswamy)	Class II
Chief complaint	Burning retrosternal chest pain (several hours)
Associated symptoms	Vomiting × 3; loose stools × 2
Past medical history	Nil (no hypertension, diabetes mellitus, ischemic heart disease or dyslipidemia)
Past surgical history	Perforated duodenal ulcer repair (2012)
Tobacco / alcohol / illicit drug use	None
Family history of premature CAD	None
Killip class at admission	I
Heart rate	80 beats per minute (regular)
Respiratory rate	20 breaths per minute
Blood pressure	110/70 mmHg
Temperature	Afebrile
Oxygen saturation on room air	100%
Outside-hospital ECG	Sinus rhythm; ST-elevation in leads I, aVL, V4-V5; T-inversion in leads II, III, aVF (STEMI, anterolateral)
Admission ECG at our institution	Sinus rhythm; ST-elevation V2-V6, I, aVL
HAEMATOLOGY	
Hemoglobin	13.8 g/dL
Total leukocyte count (admission → follow-up)	14,460 → 10,340 cells/μL

Parameter	Value
Neutrophils	73.5%
Lymphocytes	17.1%
Eosinophils	1.5%
Monocytes	7.2%
Platelet count	3.48 → 3.08 lakhs/μL
ESR	9 mm/hr
COAGULATION PROFILE AND THROMBOPHILIA SCREEN	
Prothrombin time (PT)	12.4 s (control 12.8)
Activated partial thromboplastin time (aPTT)	22.8 s (control 27.2)
INR	1.02
Antithrombin III	83.8%
Protein C	112.8
Protein S	96.2%
Factor V Leiden mutation	Normal
RENAL PROFILE AND ELECTROLYTES	
BUN (day of admission → day 3)	16 → 11 mg/dL
Serum creatinine	1.0 → 1.1 mg/dL
Sodium	137 → 139 mmol/L
Potassium	4.0 → 3.8 mmol/L
Chloride	101 → 102 mmol/L
Bicarbonate	20 → 25 mmol/L
LIVER FUNCTION TESTS	
Total bilirubin	1.64 mg/dL
Direct bilirubin	0.46 mg/dL
Indirect bilirubin	1.18 mg/dL
SGOT (AST)	71-72 IU/L
SGPT (ALT)	30 IU/L
Alkaline phosphatase	75 IU/L
Gamma-GT	27 U/L
Total protein / albumin / globulin / A:G ratio	6.7 / 4.2 / 2.5 g/dL / 1.7
LIPID PROFILE	
Total cholesterol	239 mg/dL
Triglycerides	238 mg/dL
HDL cholesterol	48 mg/dL
LDL cholesterol	176 mg/dL
Total cholesterol : HDL ratio	5.0
NON-TRADITIONAL CARDIOVASCULAR RISK FACTORS	
Lipoprotein(a)	151.5 nmol/L
Homocysteine	33.3 μmol/L

Parameter	Value
GLYCAEMIC PROFILE	
HbA1c	5.9 %
Estimated average glucose	123 mg/dL
THYROID FUNCTION	
TSH	4.9 µIU/mL
Free T4	1.20 ng/dL
VIRAL MARKERS, BLOOD GROUP AND OTHER INVESTIGATIONS	
HIV-1/2, HBsAg, anti-HCV	Non-reactive
Blood group	B positive
Urine routine	pH 6.0; SG 1.005; no protein/glucose/ketones/blood; pus cells 0.30/HPF
Ultrasound abdomen and pelvis	Grade I fatty liver
CARDIAC BIOMARKERS AT PRESENTATION	
CK-MB	10.2 ng/mL
Troponin I	0.74 ng/mL
BNP	12.1 pg/mL
2D-ECHOCARDIOGRAPHIC FINDINGS	
2D-echo chamber dimensions	Normal
Regional wall motion	Apical septum and LV apex mildly hypokinetic
LV ejection fraction (Simpson biplane)	58 → 60%
Diastolic function	Grade I dysfunction
Valvular / PA pressure / pericardium	Trivial MR & TR; normal PA pressure; no effusion
Inferior vena cava	1.1 cm, collapsing >50 %
DISCHARGE MEDICATIONS	
Ticagrelor ($P2Y_{12}$ inhibitor)	90 mg twice daily
Aspirin (COX inhibitor)	75 mg once daily
Rosuvastatin (high-intensity statin)	40 mg once daily
Ezetimibe (cholesterol-absorption inhibitor)	10 mg once daily
Metoprolol succinate ER (β-blocker)	25 mg once daily
Methylcobalamin + folic acid + pyridoxine	1 tablet once daily
Pantoprazole (PPI)	40 mg once daily
Sucralfate suspension	10 mL twice daily
Lactulose	15 mL at bedtime
Alprazolam	0.25 mg at bedtime

AG: Albumin Globulin Ratio; ALT: Alanine Aminotransferase; AST: Aspartate Aminotransferase; BNP: B-type Natriuretic Peptide; BUN: Blood Urea Nitrogen; CAD: Coronary Artery Disease; CK-MB: Creatine Kinase MB isoenzyme; COX: Cyclooxygenase; DAPT: Dual Antiplatelet Therapy; DM: Diabetes Mellitus; ECG: Electrocardiogram; ER: Extended-release; ESR: Erythrocyte Sedimentation Rate; GI : Gastrointestinal; HDL: High-Density Lipoprotein; HPF: High-power Field; HTN: Hypertension; IHD: Ischemic Heart Disease; INR : International Normalized Ratio; LAD: Left Anterior Descending Artery; LDL: Low-density Lipoprotein; LV: Left Ventricle; MI: Myocardial Infarction; MR : Mitral Regurgitation; PA: Pulmonary Artery; PPI: Proton-pump Inhibitor; RA: Right Atrium; SG: Specific Gravity; SGOT: Serum Glutamic Oxaloacetic Transaminase; SGPT: Serum Glutamic Pyruvic Transaminase; TR: Tricuspid Regurgitation; TSH: Thyroid-stimulating Hormone; URL: Upper Reference Limit.

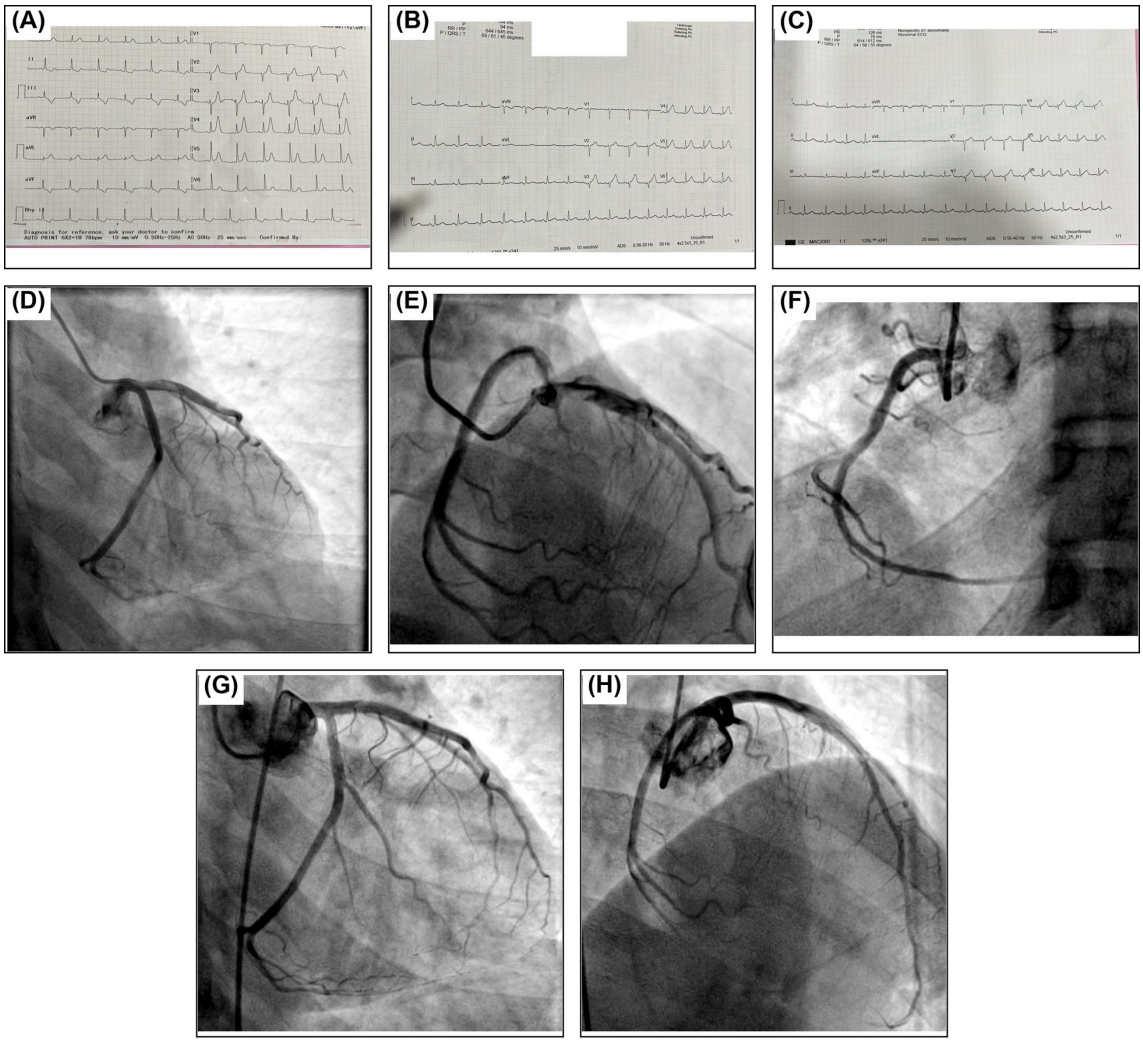

Figure 1. Electrocardiographic and coronary angiographic findings. (**A**) Outside-hospital 12-lead electrocardiogram at first presentation, showing sinus rhythm with ST-segment elevation in leads I, aVL and V4 to V5 and T-wave inversion in the inferior leads. (**B**) Admission 12-lead electrocardiogram at our institution, showing an acute anterior ST-elevation myocardial infarction with ST-segment elevation in leads V2 to V6, I and aVL. (**C**) Post-procedure 12-lead electrocardiogram showing resolution of the anterior ST-segment elevation. (**D, E**) Baseline coronary angiogram of the left coronary system in two projections, showing an 80% to 90% thrombotic lesion extending from the distal left main coronary artery into the proximal left anterior descending artery (LAD), with distal TIMI II flow. (**F**) Baseline right coronary artery angiogram, a moderate-sized non-dominant vessel free of significant disease. (**G, H**) Check coronary angiogram on day 5 after intracoronary tenecteplase (10 mg single bolus), in two projections, showing complete recanalization of the LAD with TIMI III flow and no residual thrombus in the left main coronary artery, LAD or left circumflex artery. Patient identifiers have been removed.

In view of the massive thrombotic burden extending from the distal left main into the proximal LAD, the preserved distal TIMI II flow, and the operators' concern that immediate stent deployment could precipitate distal embolization, no-reflow and periprocedural infarct-extension, a strategy of adjunctive pharmacological reperfusion was selected. Ten milligrams of tenecteplase were administered as a single intracoronary bolus via the guiding catheter into the left main coronary artery. The procedure was uneventful; total procedural and fluoroscopy times were minimal, the recorded radiation exposure was 0.60 Gy·cm^2 and hemodynamics remained stable throughout (aortic systolic 120 mmHg, diastolic 80 mmHg). The patient was transferred to the coronary care unit on standard post-STEMI intensive monitoring, on dual antiplatelet therapy (ticagrelor and aspirin), high-intensity rosuvastatin with ezetimibe, β-blocker (metoprolol succinate), pantoprazole, and adjunctive supportive medication.

In-hospital course and check angiography

The patient's chest pain settled progressively over the first 24 hours. Serial 12-lead electrocardiograms demonstrated stepwise resolution of ST-segment elevation across the anterior precordial leads (**Figure 1C**). Repeat 2D transthoracic echocardiography confirmed persistent mild apical and apical-septal hypokinesia with a left ventricular ejection fraction of 60% and no new mechanical complications. Renal function remained stable throughout the admission. There were no clinical or laboratory features of major or minor bleeding, no cerebrovascular event, and no allergic or anaphylactoid reaction attributable to tenecteplase.

A protocol-planned check coronary angiogram was performed on day 5 (9th March 2026). This demonstrated complete recanalization of the LAD, with no residual thrombus in the left main coronary artery, LAD or LCX; the RCA remained a moderate-sized, non-dominant vessel free of significant disease; and TIMI III flow was achieved throughout the left coronary system. In view of the patient's symptomatic improvement, absence of residual angiographic disease, preserved left ventricular systolic function, and stable hemodynamic status, a decision was taken to continue definitive medical management without stent implantation (**Figures 1G** and **1H**). The complete chronology from symptom onset through discharge is shown in **Figure 2A**.

Discharge and follow-up plan

The patient was discharged on 11th March 2026 in stable clinical condition. Discharge medications are enumerated in the terminal section of **Table 1**. In view of the identified spectrum of non-traditional risk factors, aggressive secondary prevention was instituted, including maximally-tolerated high-intensity statin (rosuvastatin 40 mg once daily) plus ezetimibe 10 mg once daily targeting an LDL cholesterol below 55 mg/dL [12,13], cardioprotective β-blockade with metoprolol succinate, dual antiplatelet therapy with aspirin and ticagrelor for at least 12 months, and a three-month course of the methylcobalamin-folic acid-pyridoxine combination directed at his hyperhomocysteinemia. Structured lifestyle counselling addressing diet, physical activity, weight management and psychosocial factors was provided, and follow-up appointments were scheduled at two, six, and twelve weeks and thereafter every three months in the cardiology outpatient department.

Discussion

Myocardial infarction in the young: an emerging clinical entity

Although myocardial infarction remains predominantly a disease of the older adult, the epidemiological literature of the past decade has documented a concerning secular trend towards a growing share of AMI in younger populations, particularly in South Asia [1–4]. Yandrapalli and colleagues, analyzing 1,462,168 young adults aged 18 to 59 years hospitalized in the United States with a first myocardial infarction, reported that the great majority had established, modifiable cardiovascular risk factors, with 90.3% of those aged 18 to 44 years carrying at least one such factor [1]. Arora and colleagues, analyzing the Atherosclerosis Risk in Communities (ARIC) Surveillance Study, reported that the proportion of AMI hospitalizations occurring in adults aged 35 to 54 years rose from 27% in the late 1990s to 32% in 2010–2014, young adults accounting for 30% of the surveillance cohort [3]. In a comprehensive Mayo Clinic Proceedings review, Gulati and colleagues emphasized the distinct and heterogeneous etiology of myocardial infarction in young individuals, encompassing plaque rupture and erosion, spontaneous coronary artery dissection, coronary spasm and myocardial infarction with non-obstructive coronary arteries, and underlined the need for an etiology-directed evaluation in this subgroup [2].

The present patient exemplifies the phenotype increasingly recognized in South Asian young MI: absence of traditional risk factors (no smoking, no hypertension, no diabetes, no family history of premature coronary disease), yet with a combination of markedly elevated lipoprotein(a), homocysteine, LDL cholesterol and mild subclinical hypothyroidism (**Figures 2B** and **2C**).

Lipoprotein(a), homocysteine and thyroid dysfunction

Lipoprotein(a) [Lp(a)] is an LDL-like particle in which a single apolipoprotein(a) molecule is covalently linked to apolipoprotein B-100. Its plasma concentration is largely genetically determined by the LPA gene and is essentially unmodifiable by diet, exercise or standard statin therapy. In the Copenhagen studies analyzed by Kamstrup and colleagues (2009), a measured Lp(a) above the 95th percentile was associated with a 2.6-fold increased risk of myocardial infarction (95 % confidence interval 1.6–4.1), while genetically elevated Lp(a) carried a hazard ratio of 1.22 per doubling, supporting a causal relationship [14]. In the European Atherosclerosis Society (EAS) 2010 consensus statement, Nordestgaard and colleagues proposed a desirable Lp(a) below the 80th percentile (approximately 50 mg/dL), with intensive management of all other modifiable risk factors in those above it [15]. The subsequent 2022 EAS consensus statement recommends that Lp(a) be measured at least once in every adult's lifetime and regards it as a causal, largely genetically determined and continuous risk factor for atherosclerotic cardiovascular disease [16]. In the present case, the patient's Lp(a) of 151.5 nmol/L was approximately twice the upper limit of the reporting laboratory's reference interval (0–75 nmol/L) and constitutes an important non-modifiable contributor to his premature coronary event.

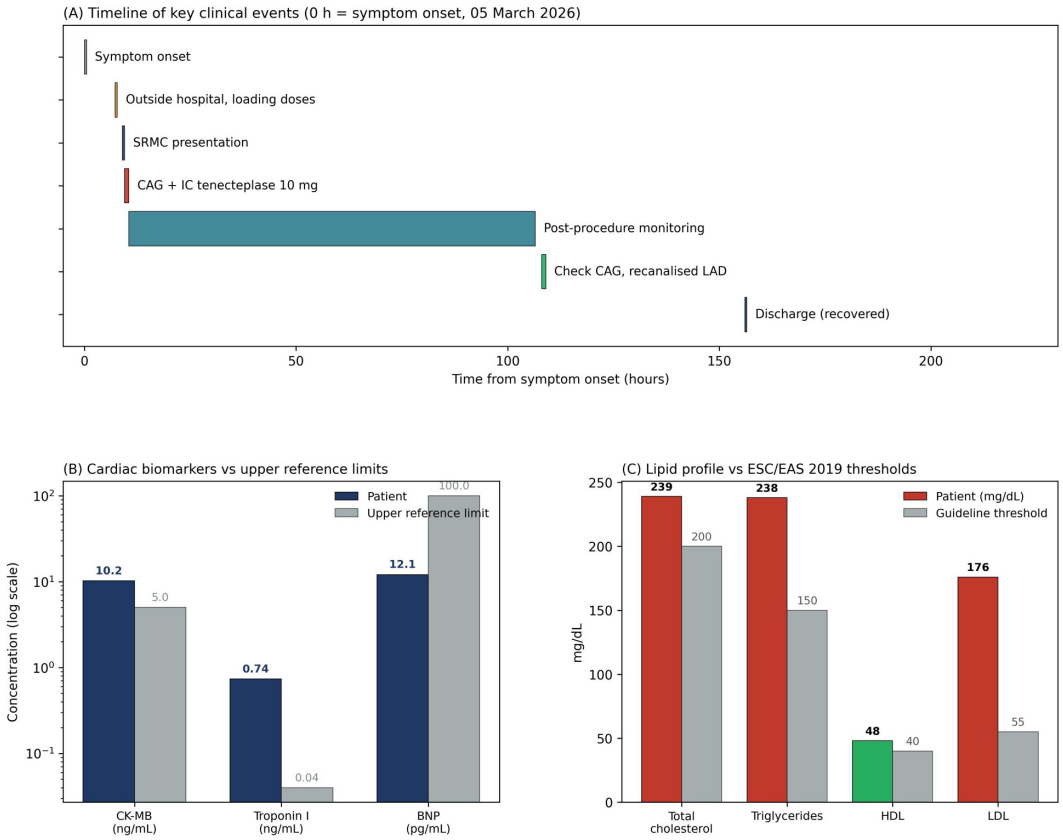

Figure 2. Combined clinical panel showing the timeline of events, the cardiac biomarker profile and the lipid profile. (**A**) Timeline of key clinical events from symptom onset through outside-hospital loading, presentation, emergency coronary angiography with intracoronary tenecteplase, in-hospital monitoring, the day-5 check angiogram showing recanalization, and discharge. (**B**) Cardiac biomarkers at presentation (CK-MB, troponin I and B-type natriuretic peptide) plotted on a logarithmic scale against the assay upper reference limit. (**C**) Lipid profile at admission compared with contemporary guideline thresholds. Red bars indicate values above the threshold and green bars values within target.

Hyperhomocysteinemia has been the subject of extensive epidemiological and interventional investigation. The Homocysteine Studies Collaboration meta-analysis (2002), which combined data from 30 prospective and retrospective observational studies, concluded that a 25% lower usual homocysteine level was associated with approximately an 11% lower risk of ischemic heart disease and an approximately 19% lower risk of stroke [17]. However, the Heart Outcomes Prevention Evaluation (HOPE) 2 randomized trial reported by Lonn and colleagues (2006), which randomized 5,522 patients with vascular disease or diabetes to a folic-acid-vitamin-B12-vitamin-B6 combination or placebo, did not demonstrate a reduction in the composite primary outcome of cardiovascular death, myocardial infarction, and stroke over a mean follow-up of five years (18.8% versus 19.8%; relative risk 0.95), despite substantial reductions in homocysteine, although stroke alone was modestly reduced [18]. Homocysteine is therefore most reasonably interpreted, in a young patient with premature coronary thrombosis, as a marker of overall vascular vulnerability and a driver of symptomatic B-vitamin repletion rather than as a target for prognosis-modifying therapy in isolation.

The patient's mildly elevated TSH (4.9 µIU/mL) with normal free thyroxine is consistent with subclinical hypothyroidism. In the Thyroid Studies Collaboration analysis of 55,287 participants, the excess risk of coronary heart disease events was confined to a TSH of 10.0–19.9 mIU/L (hazard ratio 1.89), with no excess risk at a TSH of 4.5–6.9 mIU/L (hazard ratio 1.00), and risks did not differ significantly by age, sex or pre-existing cardiovascular disease [19]. The present value therefore does not in itself confer excess coronary risk; nevertheless, thyroid function will be re-evaluated at follow-up and, if persistent, will inform a shared-decision consideration of low-dose levothyroxine.

The angiographic phenotype: heavy thrombus burden and the challenge of primary PCI

In a landmark analysis, Sianos and colleagues (2007) demonstrated that in patients undergoing primary PCI for STEMI with drug-eluting stent implantation, a large intracoronary thrombus burden (defined as a thrombus of at least two vessel diameters) was independently associated with adverse outcomes, and specifically with an increased rate of angiographically defined stent thrombosis (8.2% versus 1.3%) and major adverse cardiac events at two years [10]. These findings have led to the recognition that in the specific angiographic phenotype of massive thrombus burden with preserved distal flow, immediate stent deployment may not be the optimal reperfusion strategy, and adjunctive antithrombotic and pharmacological strategies deserve considered application.

Routine manual thrombus aspiration was extensively evaluated in the TAPAS, TASTE, and TOTAL trials [20,21]. In the TOTAL trial (10,732 patients undergoing primary PCI), routine upfront manual thrombus aspiration did not reduce the composite of cardiovascular death, recurrent myocardial infarction, cardiogenic shock, or New York Heart Association class IV heart failure at 180 days, and was associated with an increase in stroke within 30 days (0.7% versus 0.3%) [20]; the TASTE trial (7,244 patients) likewise showed no reduction in all-cause death at 30 days (2.8% versus 3.0%) [21]. Routine upfront aspiration is therefore no longer recommended: the 2023 ESC guidelines classify routine thrombus aspiration as Class III, whereas the 2021 ACC/AHA/SCAI guidelines additionally retain a Class 2b recommendation for selective bailout aspiration in the setting of high residual thrombus burden after initial guidewire crossing [6,7].

Mechanistic rationale and evidence base for intracoronary thrombolysis

Intracoronary administration of a fibrinolytic agent delivers a locally high concentration of drug directly to the site of thrombus while minimizing systemic exposure and the associated hemorrhagic risk. In the small randomized trial of Sezer and colleagues (2007), 41 patients with acute myocardial infarction who had undergone primary PCI were randomized, following stent deployment, to a single intracoronary bolus of streptokinase (250,000 IU) or matching placebo; the primary endpoint of coronary flow reserve at two days was significantly improved in the streptokinase group (2.01±0.57 versus 1.39±0.31), with a correspondingly lower index of microvascular resistance, although no long-term left ventricular functional benefit was demonstrated at six months [11]. Although these microvascular gains are of interest, and subsequent small studies of intracoronary tenecteplase, reteplase, and urokinase in the specific setting of massive thrombus burden have reported qualitatively similar angiographic benefits, the evidence base for intracoronary thrombolysis remains too small and heterogeneous to support a routine indication in contemporary guidelines. Its use is therefore considered case-by-case decision by the operator in specifically defined angiographic and clinical settings, of which the present case is representative.

Tenecteplase, a bioengineered variant of tissue plasminogen activator with three targeted amino-acid substitutions, offers pharmacological advantages that support its use in this setting: an approximately 15-fold greater fibrin specificity than alteplase, an approximately 80-fold greater resistance to inactivation by plasminogen activator inhibitor-1, and a longer initial plasma half-life (approximately 17–24 minutes in patients) permitting single-bolus administration [22]. The large ASSENT-2 randomized trial of 16,949 patients with STEMI demonstrated 30-day mortality equivalence to accelerated-infusion alteplase (6.18% versus 6.15%), with a modest reduction in non-cerebral bleeding (26.43% versus 28.95%) [23]. These pharmacokinetic and clinical properties support the choice of intracoronary tenecteplase over first-generation fibrinolytics in the setting of massive coronary thrombus.

Comparison of the present case with the published literature

Table 2 summarizes the position of the present case in relation to key published studies and guidelines on young MI, non-traditional risk factors and adjunctive intracoronary thrombolysis in the last ten years, with each cited datum verified against the original PubMed-indexed source.

Proposed management algorithm

On the basis of the present case and the reviewed literature, we propose the algorithmic approach summarized in **Figure 3**. The algorithm foregrounds early recognition of STEMI in the young patient, structured angiographic assessment with formal grading of thrombus burden, and selective use of intracoronary thrombolysis as an adjunctive strategy in the specific setting of massive thrombus with preserved distal flow. Aggressive secondary prevention with lipid-lowering, antiplatelet, β-blocker and B-vitamin therapy is combined with systematic evaluation for non-traditional risk factors, namely lipoprotein(a), homocysteine, thyroid function and a thrombophilia screen, which we consider a mandatory component of the young-MI workup. The algorithm has been aligned with the 2023 ESC acute coronary syndrome guidelines, the 2025 ACC/AHA/ACEP/NAEMSP/SCAI acute coronary syndrome guideline and the 2025 focused update of the ESC/EAS dyslipidemia guidelines [6,8,13].

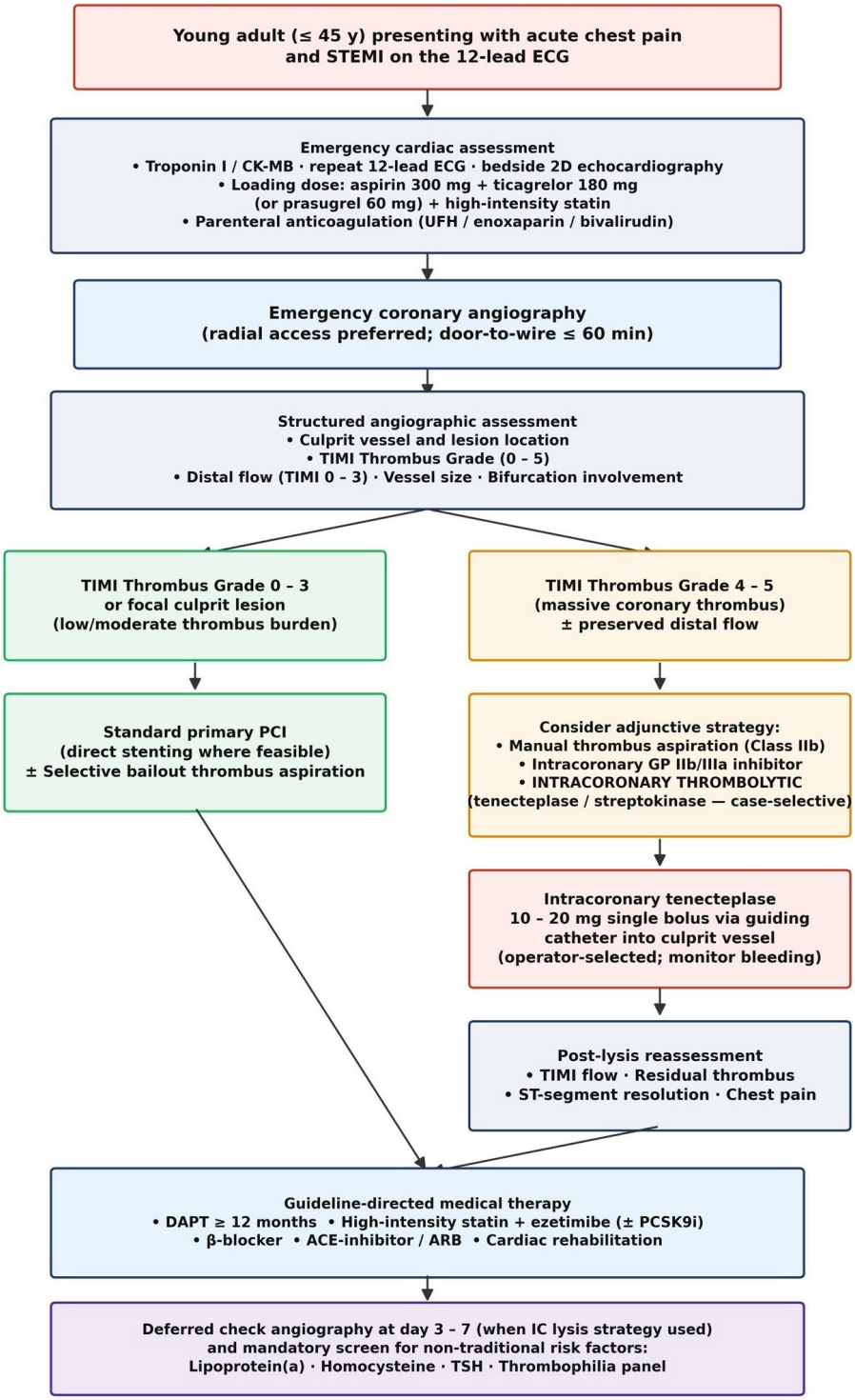

Figure 3. Proposed management algorithm for young ST-elevation myocardial infarction with a heavy coronary thrombus burden. The pathway comprises emergency cardiac assessment, structured angiographic evaluation with formal TIMI thrombus grading, and a reperfusion strategy stratified by thrombus burden. Intracoronary tenecteplase (10 to 20 mg single bolus) is shown as an operator-selected adjunctive option in TIMI thrombus grade 4 to 5 lesions with preserved distal flow. This is followed by guideline-directed medical therapy, deferred check angiography at day 3 to 7, and screening for non-traditional cardiovascular risk factors.

Table 2. Comparison of the present case with landmark published literature on young MI, non-traditional risk factors and adjunctive intracoronary thrombolysis.

Study / guideline (year) [ref]	Design / setting	Key finding as reported	Relevance to and comparison with present case
Yandrapalli et al., 2019 [1]	Nationwide US inpatient analysis; 1,462,168 young adults aged 18–59 y with a first AMI	90.3% of those aged 18–44 y had ≥1 modifiable cardiovascular risk factor; commonest were smoking (56.8%), dyslipidemia (51.7%) and hypertension (49.8%).	Our patient (38 y) had zero traditional modifiable risk factors, placing him in the residual minority requiring dedicated evaluation for non-traditional causes.
Arora et al., 2019 [3]	ARIC Surveillance; hospitalized AMI in adults 35–54 y, 1995-2014	Proportion of AMI hospitalizations in young adults rose from 27 % (1995–1999) to 32 % (2010–2014); young adults comprised 30% of the surveillance cohort.	Consistent with the rising South Asian and Indian burden of young MI; frames the case within a broader epidemiological trend.
Gulati et al., 2020 [2]	Narrative review of young AMI	Reviews the distinct and heterogeneous etiology of young AMI, including plaque rupture and erosion, spontaneous coronary artery dissection, coronary spasm and MINOCA.	Supports an etiology-directed evaluation in the young patient without traditional risk factors.
Yusuf et al., 2004 [5]	Case-control, 15,152 cases and 14,820 controls, 52 countries	Nine modifiable risk factors accounted for ≈ 90% of the population-attributable risk of a first MI in men and 94% in women.	Our patient falls into the ≈ 10% of first MIs not principally attributable to the nine classical INTERHEART risk factors.
Kamstrup et al., 2009 [14]	Copenhagen City Heart Study + Copenhagen General Population Study, prospective cohorts	Measured Lp(a) above the 95th percentile carried a 2.6-fold increased MI risk (95 % CI 1.6-4.1); genetically elevated Lp(a) HR 1.22 per doubling, supporting causality.	Our patient's Lp(a) of 151.5 nmol/L (≈ 2× upper reference limit) is a probable causal contributor.
Nordestgaard et al., 2010 [15]	Consensus statement	Recommends Lp(a) measurement in patients at intermediate or high CV risk; desirable level below the 80th percentile (≈ 50 mg/dL).	Our patient's Lp(a) was ≈ 2× the laboratory upper reference limit, supporting aggressive LDL-C reduction to<55 mg/dL.
Homocysteine Studies Collaboration, 2002 [17]	Meta-analysis of 30 observational studies	A 25 % lower usual homocysteine associated with≈11% lower IHD risk and≈19% lower stroke risk.	Our patient's Hcy of 33.3 µmol/L represents ~2× the upper reference limit, potential contributor.
Lonn et al., HOPE-2, 2006 [18]	Randomized, 5,522 vascular-disease/diabetes patients; B-vitamin combination vs placebo	B-vitamin therapy lowered homocysteine but did not reduce composite CV death / MI / stroke; reduced stroke risk.	Justifies symptomatic B-vitamin repletion but not as a prognosis-modifying therapy in isolation.
Sianos et al., 2007 [10]	Registry of 812 STEMI patients undergoing primary PCI with DES	Large thrombus burden (≥2 vessel diameters) independently predicted mortality (HR 1.76) and MACE (HR 1.88); stent thrombosis 8.2% vs 1.3% at 2 years.	Provides the primary rationale for adjunctive antithrombotic strategy rather than immediate stenting in our patient.
Sezer et al., 2007 [11]	Randomized, 41 patients with acute MI; IC streptokinase 250,000 IU vs placebo after primary PCI	IC streptokinase significantly improved coronary flow reserve at 48 h (2.01 vs 1.39); no long-term LV functional benefit at 6 months.	Proof-of-concept supporting selective adjunctive IC thrombolytic strategy.
ASSENT-2 Investigators, 1999 [23]	Randomized, 16,949 STEMI patients; single-bolus tenecteplase vs accelerated alteplase	30-day mortality equivalent between the two arms; modest reduction in non-cerebral bleeding with tenecteplase.	Supports tenecteplase pharmacology when a fibrinolytic strategy is selected.

Study / guideline (year) [ref]	Design / setting	Key finding as reported	Relevance to and comparison with present case
Byrne et al., 2023 ESC ACS Guidelines, 2023 [6]	Guideline	Primary PCI Class I; routine upfront thrombus aspiration Class III.	Our approach constituted operator-selected adjunctive IC fibrinolysis in a specific angiographic phenotype not covered by a positive Class recommendation.
Lawton et al., 2021 ACC/AHA/SCAI Revascularization Guideline, 2022 [7]	Guideline	Primary PCI Class I for STEMI; routine aspiration thrombectomy Class 3 (No Benefit); selective/bailout aspiration Class 2b.	Consistent with ESC 2023; frames the IC-lysis strategy as complementary rather than substitutive.
Rao et al., 2025 ACC/AHA/ACEP/NAEMSP/SCAI ACS Guideline, 2025 [8]	Guideline	Current United States guidance for acute coronary syndromes; primary PCI remains the reperfusion strategy of choice in STEMI.	Confirm that the reperfusion decision in this case sits within current recommendations, while adjunctive IC fibrinolysis remains outside any positive class recommendation.
Mach et al., 2025 Focused Update of the ESC/EAS Dyslipidaemia Guidelines, 2025 [13]	Guideline focused update	Most recent European guidance on lipid management, updating the 2019 ESC/EAS recommendations.	Directs the intensity of LDL-C lowering applied in this patient's secondary prevention.
Kronenberg et al., EAS Lp(a) Consensus, 2022 [16]	Consensus statement	Recommends measuring Lp(a) at least once in every adult's lifetime; Lp(a) is a causal, largely genetically determined, continuous ASCVD risk factor.	Provides the contemporary framework for interpreting this patient's markedly elevated Lp(a) of 151.5 nmol/L.

AMI: Acute Myocardial Infarction; CV: Cardiovascular; DES: Drug-Eluting Stent; IC: Intracoronary; IHD: Ischemic Heart Disease; PCI: Percutaneous Coronary Intervention; STEMI: ST-Elevation Myocardial Infarction.

Strengths and Limitations

Strengths of this report include complete, contemporaneous clinical documentation with fully-quantified laboratory data; angiographic confirmation of both the index lesion and the response to intracoronary thrombolysis with paired coronary angiograms; a comprehensive evaluation for non-traditional cardiovascular risk factors that identified two clinically-actionable abnormalities; and application of guideline-directed secondary-prevention therapy at discharge. Limitations include the absence of routine intracoronary imaging (optical coherence tomography or intravascular ultrasound) to characterize plaque morphology beneath the thrombus, the absence of formal extended thrombophilia testing beyond antithrombin III, protein C, protein S and Factor V Leiden, the single-center nature of the report, and the inherent limitations of a single-case observation. Long-term follow-up beyond hospital discharge is planned but not yet mature.

Conclusion

In a young adult presenting with ST-elevation myocardial infarction, a massive intracoronary thrombus with preserved distal flow should be recognized as a distinct angiographic phenotype in which immediate stenting carries a particular risk of distal embolization and no-reflow. Intracoronary thrombolysis deserves consideration as a stent-sparing adjunctive reperfusion strategy in carefully selected patients of this type, although the supporting evidence remains limited and its use should remain an individualized, operator-led decision. Every young patient with myocardial infarction warrants a systematic search for non-traditional and thrombophilic risk factors, because their identification directs long-term secondary prevention. Prospective studies are needed to define the role of adjunctive intracoronary fibrinolysis in this setting.

Conflicts of Interest

The authors declare that they have no conflicts of interest. The corresponding author confirms this declaration on behalf of all co-authors.

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Consent for Publication

Written informed consent for the publication of anonymized clinical details, laboratory data and coronary angiographic images was obtained from the patient.

Availability of Data and Materials

All data supporting this case report are contained within the article. Additional anonymized information is available from the corresponding author on reasonable request.

Reporting Guideline

This case report was prepared in accordance with the CARE (CAse REport) guidelines.

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Author Contributions Statement

L.R.K. conceived the report, acquired and interpreted the clinical, laboratory and angiographic data, and drafted the manuscript. B.J.V. performed the coronary angiography and intracoronary thrombolysis, supervised the patient's management, and critically revised the manuscript for important intellectual content. S.S.M.J. contributed to clinical management, the review of the literature, and the critical revision of the manuscript. All authors have read and approved the final version of the manuscript and agree to be accountable for all aspects of the work.

Conflicts of Interest

None declared.

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