

Aortic Coronary Anomaly Causing Acute Myocardial Infarction in an Adolescent: Emergency Percutaneous Revascularization as a Bridge to Surgical Repair

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Case Report

Keywords: Anomalous aortic origin of the coronary artery, Acute myocardial infarction, Adolescent, Percutaneous coronary intervention, Stent restenosis, Coronary artery bypass grafting

Posted Date: September 30th, 2026

DOI: <https://doi.org/10.21203/rs.3.rs-10642803/v1>

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Additional Declarations: No competing interests reported.

Abstract

Background: Anomalous aortic origin of the left main coronary artery (AAOCA) with an interarterial course is a rare but high-risk congenital anomaly, predisposing adolescents to acute myocardial infarction (AMI) and sudden cardiac death during exertion. The optimal management of AAOCA-related AMI with cardiogenic shock in pediatric patients remains poorly defined, with no established consensus in current guidelines. This case highlights the novel strategy of emergency percutaneous coronary intervention (PCI) as a critical bridging therapy to stabilize hemodynamics prior to definitive surgical repair.

Case presentation: A previously healthy 14-year-old male with a history of exercise-induced chest tightness presented with recurrent anginal symptoms, progressive dyspnea, and hemodynamic collapse following vigorous physical activity. Electrocardiography revealed acute ischemic changes, and laboratory tests showed severe lactic acidosis. Emergency coronary angiography identified a slit-like ostial stenosis of the left main coronary artery arising anomalously from the right coronary sinus with an interarterial course. Given the critical instability and impending circulatory failure, emergency stenting was performed, achieving immediate restoration of TIMI 3 flow and rapid hemodynamic improvement. However, at one-year follow-up, the patient developed severe in-stent restenosis with stent malapposition due to persistent mechanical compression from the anomalous anatomy, necessitating subsequent coronary artery bypass grafting.

Conclusion: This case demonstrates that emergency PCI can serve as an effective and life-saving bridge to stabilize adolescents with AAOCA-related AMI and cardiogenic shock, enabling timely surgical correction. However, the late occurrence of stent failure underscores the biomechanical limitations of percutaneous intervention in this setting and reinforces that surgery remains the definitive treatment. This approach offers a practical management algorithm for this rare and high-risk emergency scenario.

Background

AAOCA with an interarterial course is a rare but high-risk congenital anomaly, frequently associated with exercise-induced sudden cardiac death in adolescents. The underlying ischemia results from dynamic compression of the slit-like ostium and intramural segment during exertion, leading to critical luminal narrowing and an acute supply-demand mismatch. Although some affected individuals remain asymptomatic, others present with catastrophic events such as acute myocardial infarction, cardiogenic shock, or malignant arrhythmias.

Current management of symptomatic AAOCA primarily favors surgical correction, including unroofing or bypass grafting, as recommended by major society guidelines. However, specific recommendations for acute presentations with hemodynamic instability are lacking, and surgical options may not be immediately available in all centers. Percutaneous coronary intervention, while established in adult atherosclerotic disease, has an uncertain role in congenital coronary anomalies, particularly in the emergency setting.

This case report describes a 14-year-old male with AAOCA-related acute myocardial infarction and refractory cardiogenic shock, managed with emergency stenting as a bridging strategy to stabilize hemodynamics prior to definitive surgery. The case illustrates both the acute viability of percutaneous revascularization and its long-term biomechanical limitations, reinforcing that surgery remains the gold standard for this congenital anomaly.

Case presentation

A 14-year-old male middle school student with 5-year history of exercise-induced chest tightness (typically lasting around 5 minutes and self-resolving) presented with recurrent anginal symptoms during a Physical Education (PE) class. Preceding his hospital admission by 20 minutes, the patient encountered a resurgence of dyspnea and profuse sweating following vigorous exercise in PE class, with persistent and unrelieved symptoms. He remained conscious throughout, and emergency medical services promptly transferred him to our Emergency Department (ED) within 10 minutes of symptom onset due to the hospital's close proximity. The patient had no medical history of diabetes mellitus, hypertension, congenital disorders, autoimmune diseases, or substance abuse.

Upon emergency assessment, the patient exhibited a body temperature of 36.5°C, a heart rate of 125 bpm, tachypnea at 35 breaths per minute, and hypotension measuring 73/47 mmHg. The patient was alert but exhibited tachypnea, dusky skin discoloration, diaphoresis with clammy extremities, and thready peripheral pulses. Respiratory distress was evident characterized by manifested as bilateral pulmonary edema with diffuse crackles, while the cardiac examination revealed no murmurs. The patient had a soft and non-tender abdomen, a normal neurological exam, but presented with severe lactic acidosis (pH 7.25, Lac 8.6 mmol/L, BE -10.2) and hypoxemia (PaO₂ 48 mmHg; PaO₂/FiO₂ 165.5). Electrocardiography indicated acute ischemia patterns, including ST-elevation (0.1–0.3 mV) in aVR/V1/aVL with reciprocal ST-depression (0.3–0.8 mV) across inferolateral leads (**see** Fig. 1A). Initial Troponin and BNP levels, measured 30 minutes post-onset, did not provide a definitive diagnosis.

The differential diagnoses initially considered were acute myocardial infarction (AMI), aortic dissection, ruptured aortic sinus aneurysm, acute fulminant myocarditis, and acute pulmonary embolism. Due to the rapidly deteriorating clinical condition and urgency of the situation, contrast-enhanced thoracic computed tomography (CT) was promptly conducted due to suspicion of aortic dissection or aneurysmal rupture, taking precedence over bedside echocardiography. The CT scan revealed bilateral pulmonary infiltrates and interstitial congestion, along with normal contrast opacification of the aortic lumen and pulmonary arterial system without significant filling defects, ruling out aortic dissection (including Marfan syndrome variants), pulmonary embolism, and ruptured aortic sinus aneurysm. The patient's condition progressively deteriorated, presenting as worsening respiratory distress characterized by lethargy, perioral and facial cyanosis, diaphoresis with cutaneous hypothermia, and expectoration of pink frothy sputum. Continuous rhythm monitoring recorded frequent multifocal premature ventricular complexes with polymorphic QRS morphologies. Despite maximal catecholamine support (norepinephrine infusion at 25 µg/kg/min and dopamine at 20 µg/kg/min), hemodynamic instability

persisted, with worsening hypotension unresponsive to vasopressor therapy. Following the initial exclusion of aortic dissection, ruptured sinus of Valsalva aneurysm, and pulmonary embolism, emergency coronary angiography was performed while awaiting three-dimensional coronary artery reconstruction, with readiness for percutaneous coronary intervention (PCI) if deemed necessary.

Following ACC/AHA Class I guidelines managing STEMI, emergent coronary angiography was conducted after obtaining parental consent by his parents and prior to intra-aortic balloon pump (IABP) placement for mechanical circulatory support. The angiogram revealed a unique anatomy where the left main coronary artery (LMCA) arose from the right coronary sinus and displayed a slit-like ostium with 99% diameter stenosis, characterized by an anomalous take-off configuration. The left anterior descending (LAD) and left circumflex (LCx) arteries showed TIMI 2 flow, while the LAD, LCx, and right coronary artery (RCA) appeared free of atherosclerotic plaques on angiography (**see Fig. 1B**). The confirmed diagnosis was anomalous aortic origin of the left coronary artery (AAOCA) with an interarterial course between the ascending aorta and the pulmonary trunk, complicated by AMI (**see Fig. 2A**).

The patient presents with acute heart failure stemming from congenital left main coronary artery stenosis deemed inoperable, with the condition now critical and nearing circulatory collapse. To salvage the child's life, an urgent decision was made to proceed with stent implantation for reperfusion therapy. Revascularization was executed by consecutively deploying two overlapping PARTNER 4.0×12 mm drug-eluting stents (Lepu Medical, Beijing, China) from the LMCA ostium to the proximal LAD, followed by post-dilation using a non-compliant NC TREK 5.0×8 mm balloon (Abbott Laboratories, CHI,USA) inflated to 14–18 atmospheres. The initial stent was positioned from the left main coronary artery ostium to the proximal LAD segment, with a dual-layer scaffolding deployed to counteract aortic compressive forces for adequate radial support (**see Fig. 1B**). Subsequent post-PCI electrocardiography revealed normalization of sinus rhythm with complete ST-segment resolution to baseline, albeit with persistent poor R-wave progression in precordial leads (**see Fig. 1C**). Post-intervention intravascular ultrasound (IVUS; Volcano Eagle Eye® Platinum) confirmed optimal stent-vessel wall apposition without residual gaps, resulting in a final LMCA ostial luminal area of 10.2 mm² (**see Fig. 2A and 2B**). Documentation showed restoration of TIMI 3 flow in both LAD and LCx territories.

Clinical improvement occurred promptly after PCI, leading to the resolution of respiratory distress within one hour, along with hemodynamic stabilization. The comprehensive critical care approach involved continuous IABP assistance, a combination of three antithrombotic medications (100 mg aspirin once daily, 90 mg ticagrelor twice daily, and 1 mg/kg enoxaparin twice daily), inotropic support, and heart failure treatment following established guidelines. Sequential analysis of arterial blood gases indicated a gradual correction of metabolic parameters: normalization of pH from 7.25 to 7.40, reduction of lactate levels from 8.6 to 2.59 mmol/L (69.9% decrease within 4.5 hours), and enhancement of the oxygenation index to 220.7 mmHg (PaO₂ 59 mmHg on FiO₂ 0.5). The evolution of myocardial injury biomarkers over time from symptom onset is detailed in Table 1. Echocardiography conducted 72 hours post-procedure revealed maintained left ventricular systolic function (LVEF 58.5%) with anterolateral hypokinesis, mild

mitral regurgitation, and normal chamber dimensions (LA 26 mm, LVEDD 40 mm). Removal of IABP was successfully carried out on the seventh day post-PCI following consistent hemodynamic improvement.

Table 1
Temporal changes in myocardial injury biomarkers following symptom onset

Biomarker (Post-onset)	0.5h	7h	20h	44h	70h
CK-MB (ng/mL)	< 1.0	297.0	264.9	80.3	52.3
cTnl (ng/mL)	< 0.05	> 80	> 80	38.3	31.6
BNP (ng/mL)	17.5	266	797	1998	1880

Two weeks after PCI, coronary computed tomography angiography (CCTA) showed the left main coronary artery (LMCA) arising anomalously from the right coronary sinus with an acute take-off angle < 45° and an intramural course; its proximal segment passed between the aortic root and the main pulmonary artery trunk, and stent struts were visible within the LMCA (see Fig. 2C and 2D). On post-PCI day 28, pre-discharge echocardiography showed a left atrial diameter of 30.2 mm, left ventricular end-diastolic diameter (LVDD) of 46 mm, segmental hypokinesis of the apical anterior left ventricular wall, and an ejection fraction (EF) of 63.7% (detailed echocardiographic parameters are summarized in Table 2).

Table 2
Serial echocardiographic assessment of left ventricular function following percutaneous coronary intervention

Parameters (Post-PCI)	Day 3	Week 1	Week 2	Year 1
LVDD (mm)	40	41	46	50
LVEF (%)	58.5	66.5	63.7	55

Approximately six months after PCI, the patient developed recurrent exertional chest tightness that resolved with rest. The patient declined follow-up examinations while preparing for the senior high school entrance examination. At the 1-year post-PCI visit, the patient reported chest tightness with mild exertion. Echocardiography at one year after PCI showed a left atrium (LA) of 32 mm, left ventricular end-diastolic diameter (LVDD) of 50 mm, mild mitral regurgitation, reduced anterior wall motion, and an LVEF of 55%; BNP was 199.94 pg/mL. Scheduled coronary angiography at one year revealed severe stent malapposition at the distal left main stem with approximately 90% luminal loss and TIMI grade 1 flow in the distal segments of the LAD and the LCX (see Fig. 3A and 3B). Because the stent was compressed by a congenital anomaly of the left main coronary artery, we determined that coronary artery bypass

grafting (CABG) was the definitive next step in the treatment strategy with left internal mammary artery (LIMA)-to-LAD anastomosis and an aorta-to-saphenous vein graft (SVG)-to-intermediate branch bypass.

Discussion and Conclusions

AMI is rare in children and adolescents and arise from causes distinct from adult type 1 myocardial infarction, including Kawasaki disease, Takayasu's arteritis, coagulation-related thrombophilia, and coronary artery origin or course anomalies[1]. Cases of AMI associated with anomalous coronary origins - especially coronaries originating from the contralateral sinus of Valsalva and following an interarterial course between the aorta and pulmonary artery - has increased markedly and now represents a leading cause of sudden cardiac death in young athletes because these anomalies confer high susceptibility to acute cardiovascular events[2]. Earlier explanations attributed ischemia to exercise-induced expansion of the aorta or pulmonary artery that compresses interarterial coronaries and reduces myocardial perfusion. Recent computational fluid dynamics (CFD) studies, however, show that ischemia principally results from dynamic flow restriction imposed by anatomical abnormalities: slit-like ostia and intramural segments have substantially reduced luminal areas relative to normal elliptical or circular coronaries. During exertion, increased stroke volume and blood pressure raise aortic wall stress and acutely angulate the coronary ostium, further narrowing its effective opening, while the higher aortic pressure directly compresses intramural segments; together, these effects markedly exacerbate myocardial hypoperfusion[3].

Hatoum H's study[3] examined three right AAOCA cases using patient-specific 3D-printed models and simulated exercise hemodynamics to compare ischemic and non-ischemic states and to evaluate surgical outcomes. The results indicate that exercise-induced hemodynamic compromise arises principally from ostial and intramural flow restriction at anatomically narrowed segments. When the lesion involves the left main or right coronary ostium, ischemia affects large myocardial territories. Exercise sharply increases myocardial oxygen demand, and the resulting aggravation of the supply - demand imbalance accelerates ischemia and can precipitate acute heart failure or malignant arrhythmias. Thus, AMI in these patients often presents catastrophically as syncope, cardiogenic shock, or sudden death, consistent with the anomaly's high-risk pathophysiology[4].

A 14-year-old presented with exertional chest tightness and post-exercise syncope, rapidly progressing to refractory pulmonary edema and cardiogenic shock. Coronary angiography and 3D-CT demonstrated an anomalous LMCA originating from the right sinus of Valsalva with a slit-like ostial stenosis, an intramural course, and an interarterial trajectory between the aortic and pulmonary trunks. Although management of AAOCA currently lacks standardized guidelines, available evidence favors surgical correction (e.g., unroofing, CABG)[5], which is typically available only at specialized centers. This case highlights the critical challenge of acute myocardial infarction complicated by cardiogenic shock, a setting in which conventional myocardial reperfusion strategies are poorly characterized. Exercise-induced compression of the slit-like ostium and the intramural segment produced extensive left ventricular ischemia. Emergency PCI with dual-stent implantation produced immediate luminal

expansion, resolved symptoms and restored hemodynamic stability, as demonstrated by lactate clearance and improved oxygenation. The successful acute outcome suggests that PCI may serve as a minimally invasive alternative to surgical reconstruction in selected AAOCA emergencies, but long-term outcomes remain to be defined.

At six-month follow-up, the patient reported exertional chest discomfort, which progressed to severe angiographically confirmed in-stent restenosis (ISR) with stent malapposition at one year post-PCI, despite post-procedural IVUS initially confirming optimal stent apposition. The ISR mechanism likely reflects sustained aortic wall stress in this physically active adolescent, which mechanically deformed stent struts and produced progressive malapposition. Rigatelli and Hatoum[3] showed that angulated coronary ostia and intramural segments exhibit pathologically elevated wall shear stress (WSS), which drives endothelial injury and pro-atherogenic flow patterns. Accelerated stenotic flow velocities combined with distal deceleration create zones of thrombogenic turbulence. Although stenting restores luminal diameter, it does not correct the underlying ostial angulation that surgical unroofing addresses. Persistent high WSS and abnormal hemodynamics in the LMCA therefore likely promoted neoatherosclerosis and thrombus accumulation, culminating in ISR and highlighting the biomechanical limits of endovascular approaches for AAOCA with dynamic compression.

AAOCA-induced acute myocardial infarction progresses rapidly. In this case, PCI with stenting proved an effective salvage strategy, achieving immediate LMCA expansion (door-to-balloon $\leq$ 90 minutes) that restored perfusion, resolved pulmonary edema, and reversed cardiogenic shock within 6 hours, thereby establishing the hemodynamic stability required for subsequent surgery. Although long-term outcomes indicate the biomechanical limitations of PCI and its inability to definitively treat AAOCA, PCI functions as a crucial bridge during the acute window, as shown by 23-month survival with preserved ventricular function prior to restenosis.

Abbreviations

AAOCA

Anomalous aortic origin of the left main coronary artery

ACC/AHA

American College of Cardiology / American Heart Association

AMI

Acute myocardial infarction

BNP

B-type natriuretic peptide

CABG

Coronary artery bypass grafting

CCTA

Coronary computed tomography angiography

CFD

Computational fluid dynamics
CK-MB
Creatine kinase-MB
CT
Computed tomography
cTnl
Cardiac troponin I
ECG
Electrocardiography
ED
Emergency department
EF
Ejection fraction
ETR
Emergency transcatheter revascularization
IABP
Intra-aortic balloon pump
ISR
In-stent restenosis
IVUS
Intravascular ultrasound
LAD
Left anterior descending artery
LCx
Left circumflex artery
LIMA
Left internal mammary artery
LMCA
Left main coronary artery
LVDD
Left ventricular end-diastolic diameter
LVEF
Left ventricular ejection fraction
PCI
Percutaneous coronary intervention
PE
Physical education
RCA
Right coronary artery
STEMI

ST-segment elevation myocardial infarction

SVG

Saphenous vein graft

TIMI

Thrombolysis in Myocardial Infarction

WSS

Wall shear stress

Declarations

Ethics approval and consent to participate

This case report was conducted in accordance with the applicable local ethics guidelines. The study involved human participant data and was performed in accordance with the Declaration of Helsinki. Ethical approval was obtained from the Ethics Committee of the 983rd Hospital of the Joint Logistics Support Force of the Chinese People's Liberation Army. Written informed consent to participate was obtained from the patient's parents or legal guardians.

Consent for publication

Written informed consent for publication of this case report and any accompanying images was obtained from the patient's parents or legal guardians.

Competing interests

The authors declare that they have no competing interests.

Funding

The authors received no financial support for the research, authorship, and/or publication of this article.

Author Contribution

YW and CS contributed equally to this work. YW and CS were involved in patient management, data collection, and drafting the manuscript. TL conceived the study, supervised the clinical management, and revised the manuscript critically for important intellectual content. All authors read and approved the final manuscript.

Acknowledgements

The authors thank the patient and their family for their valuable contributions to this study.

Availability of data and materials

The datasets used and/or analyzed during the current case report are available from the corresponding author on reasonable request.

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Figures

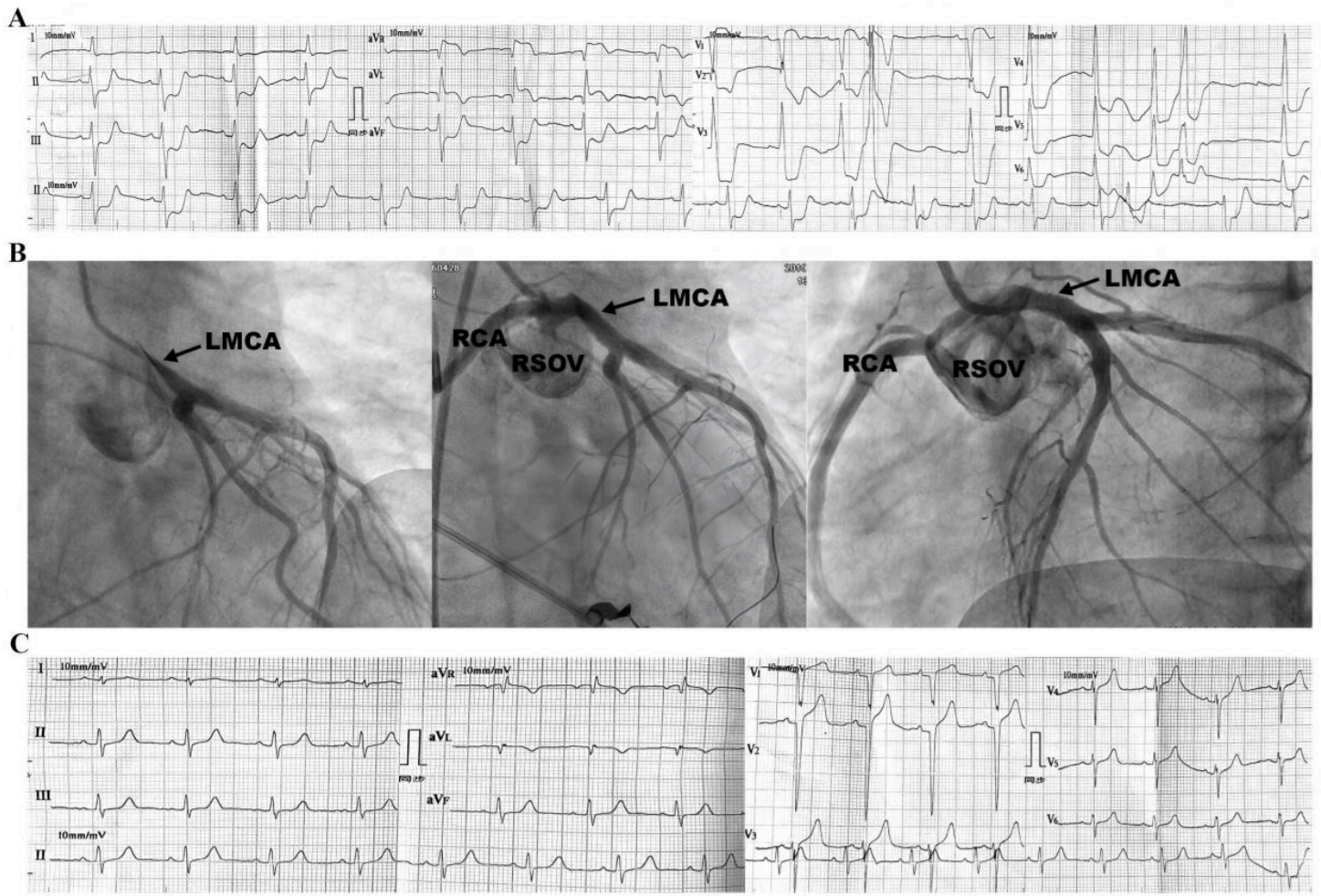


Figure 1

A: ECG at admission showing ST elevation in leads aVR - aVL and V1 with down-sloping ST depression in leads (V2-V6) and in II-III-aVF. **B:** Left panel: Coronary angiography demonstrating a severely narrowed LMCA with a slit-like ostium (black arrow). Middle and right Panels: Relief of LMCA stenosis after stent implantation (black arrow). LMCA and RCA originate from the right Valsalva sinus. LMCA, left main coronary artery; LAD, left anterior descending artery; RCA, right coronary artery; RSOV, right sinus of Valsalva. **C:** Normalized ECG after PCI.

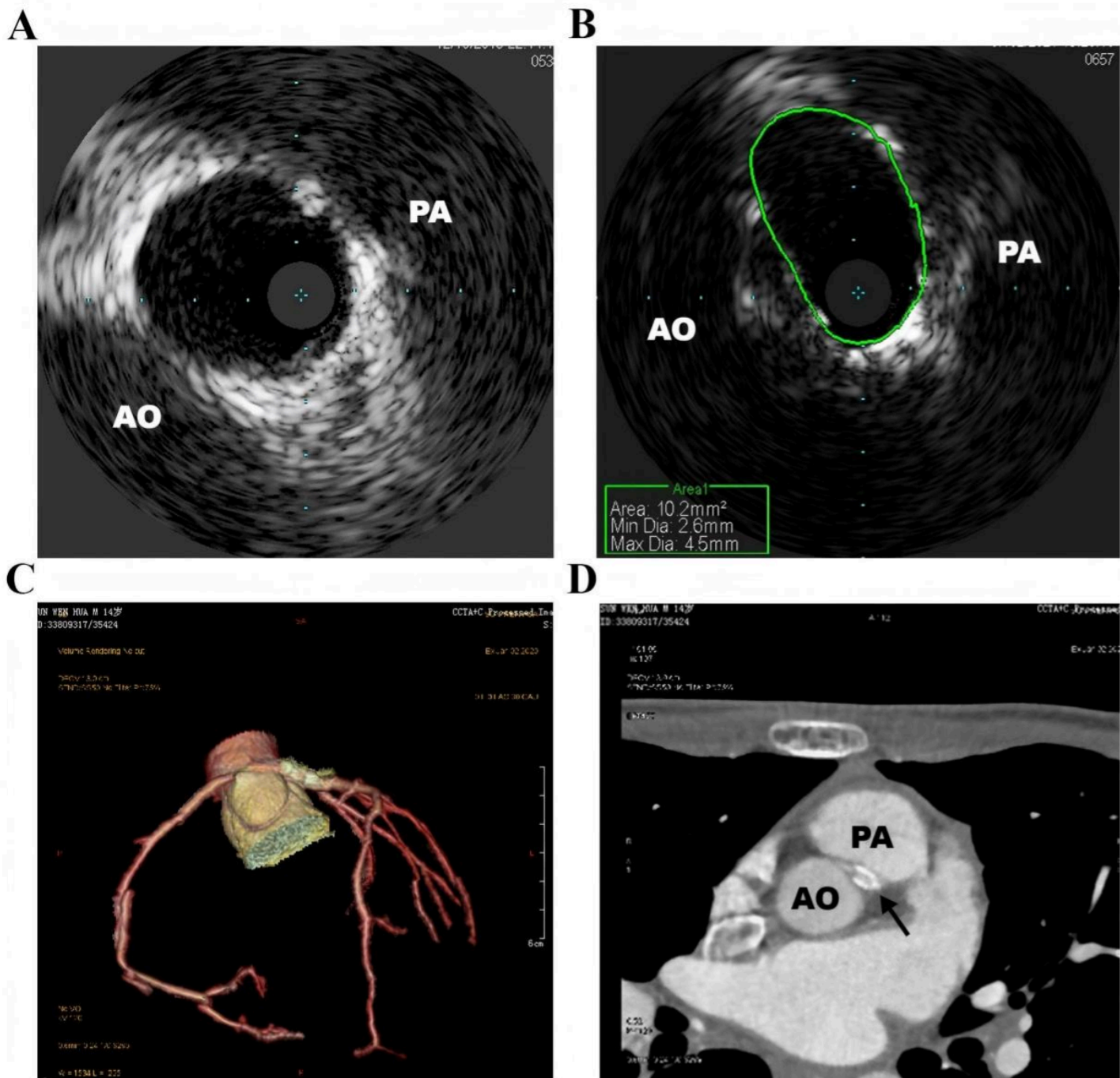


Figure 2

A: Intravascular ultrasound (IVUS) shows a fully expanded bilayer stent in the LMCA, located between the aorta (AO) and the pulmonary artery (PA). **B:** IVUS reveals an asymmetric LMCA orifice. **C:** 3D CCTA reconstruction corroborates the angiographic findings and confirms an anomalous origin of the LMCA from the right coronary sinus. **D:** CT angiography depicts the implanted stents (black arrow) within the LMCA, which follows an interarterial course between the aorta (AO) and pulmonary artery (PA).

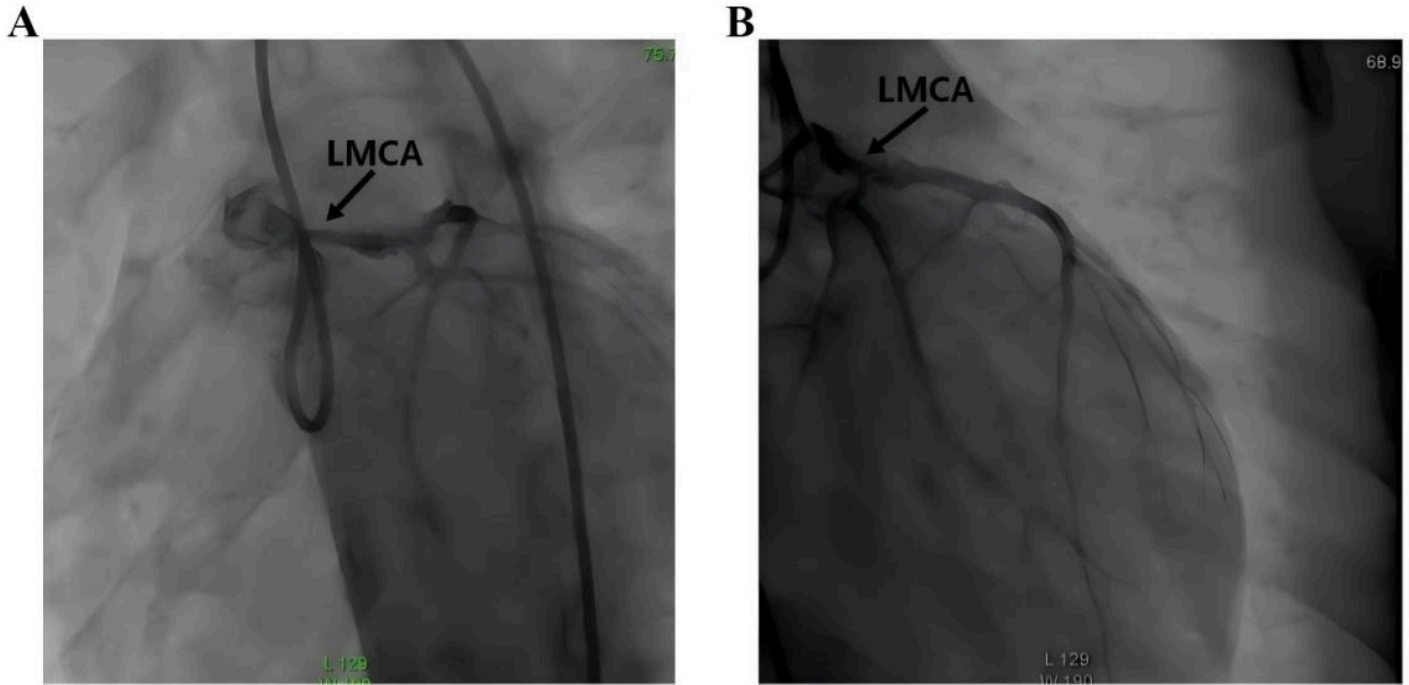


Figure 3

A and B: Scheduled follow up coronary angiography at one year showed severe stent malapposition in the distal left main stem with approximately 90% luminal loss, and TIMI grade 1 flow in the distal LAD and LCX. LMCA, left main coronary artery; LAD, left anterior descending artery; LCX, left circumflex artery.