ACUTE MYOCARDIAL INFARCTION

Acute myocardial infarction in a patient with anomalous left main coronary artery origin with a hypoplastic left anterior descending artery: A diagnostic and therapeutic challenge

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ABSTRACT

Congenital coronary artery anomalies are rare but clinically significant. We present a 41-year-old Caucasian male who presented with acute ST-elevation myocardial infarction (STEMI) involving two distinct vascular territories. Initial electrocardiogram (ECG) findings showed ST-segment elevation in the inferior and anterolateral leads, raising suspicion of multivessel involvement. Delayed access to a cardiac catheterisation laboratory warranted thrombolysis as the primary reperfusion strategy; however, this was unsuccessful. Emergent coronary angiography revealed an anomalous left main coronary artery (LMCA) originating from the right coronary cusp, sharing a common ostium with the right coronary artery (RCA). A critical stenosis of the mid-RCA was identified, attenuated proximally by a thrombolysis in myocardial infarction (TIMI) 3 thrombus burden and complete occlusion of its distal branches. Coronary computed tomography angiography (CCTA) further revealed a hypoplastic left anterior descending artery (LAD). The patient was initially managed with medical therapy alone, and subsequent percutaneous coronary intervention (PCI) was performed for ongoing stable angina, resulting in complete coronary revascularisation. This case highlights the importance of considering coronary anomalies in patients with atypical clinical presentations and the need for individualised treatment approaches. The coexistence of congenital and atherosclerotic coronary artery disease poses significant challenges, and further studies are needed to refine screening and management guidelines.

Keywords: congenital coronary artery anomal, anomalous left main coronary artery origin, anomaly, hypoplastic left anterior descending artery, myocardial infarction, coronary computed tomography angiography

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INTRODUCTION

Coronary artery anomalies are rare congenital abnormalities, with a reported prevalence of 0.15–0.84%, often discovered incidentally or at autopsy.[1,2] While many anomalies are benign and asymptomatic, certain types – particularly those involving an anomalous origin or course of coronary artery – are associated with myocardial ischaemia, arrhythmias, and sudden cardiac death, especially in younger individuals or during exertion.[2] Their clinical relevance becomes particularly significant in the setting of acute coronary syndromes, where they may contribute directly to ischaemia or complicate diagnosis, risk stratification, and revascularisation planning.[3] In such cases, the presence of an anomalous artery may obscure the identification of the culprit lesion, delay timely reperfusion, or alter the choice of intervention.

We report an exceptionally rare case of STEMI involving two vascular territories in a patient with coexisting congenital coronary anomalies and multiple cardiovascular risk factors. Initial ECG showed ST-segment elevation in the inferior and anterolateral leads, raising suspicion for multivessel involvement. Despite thrombolysis, failed reperfusion necessitated coronary angiography, which revealed an anomalous LMCA originating from the right coronary cusp and suspected chronic total occlusion (CTO) of the LAD. The RCA demonstrated severe mid-vessel stenosis with a high thrombus burden and was initially

managed medically. CCTA excluded the presence of a CTO and instead identified a rudimentary, hypoplastic LAD, while also confirming a prepulmonic course of the anomalous LMCA originating from a common ostium with the RCA.

The coexistence of an anomalous LMCA originating from the right coronary cusp and a hypoplastic LAD is extremely rare, with no reported cases in living patients. This combination not only complicates diagnosis and clinical decision-making but also poses challenges in selecting an appropriate management strategy. Current guidelines support an individualised approach, including medical therapy, PCI, or surgical correction, depending on the anatomical course, presence of ischaemia, and patient-specific factors. This case underscores the importance of recognising coronary anomalies in atypical STEMI presentations and highlights the diagnostic and therapeutic complexities posed by the coexistence of congenital and atherosclerotic coronary disease. Further research is needed to refine screening protocols, improve early recognition, and optimise treatment strategies in this patient population.

CASE REPORT

A 41-year-old Caucasian male presented to the emergency department of a peripheral hospital 10 hours after the initial onset of acute retrosternal chest pain radiating to the left jaw associated with nausea and diaphoresis. Relevant medical history and cardiovascular risk factors included untreated diabetes mellitus, active cigarette smoking, and a family history of ischaemic heart disease.

The initial ECG findings (Figure 1) were those of ST-segment elevation in the inferior and anterolateral leads with reciprocal ST-depression, raising suspicion for thrombosis in two distinct vascular territories. Laboratory investigations revealed a high-sensitivity troponin T of 824 ng/L (World Health Organization rule-in criteria > 100 ng/L) and a haemoglobin A1c (HbA1c) of 7.9% (above the 7.0% therapeutic target). Serum chemistry, inflammatory markers, lipogram, and coagulation studies were all within normal range. Initial mortality risk stratification was low: Global Registry of Acute Coronary Events (GRACE) score of 76, TIMI score of 2, and Killip Class I.

Presentation within the 12-hour thrombolysis window and the absence of contraindications warranted fibrinolytic therapy as the primary reperfusion strategy, as prolonged transfer time to a PCI-capable facility precluded timely intervention. Tissue plasminogen activator, alteplase, was administered without any electrical or mechanical cardiac complications. ECG 90 minutes post-thrombolysis showed persistent ST-segment elevation with < 50% reduction in initial ST-segment elevation, suggestive of failed thrombolysis by ECG criteria. Loading doses of aspirin 300 mg, clopidogrel 300 mg, and atorvastatin 80 mg were administered, followed by maintenance doses and therapeutic enoxaparin.

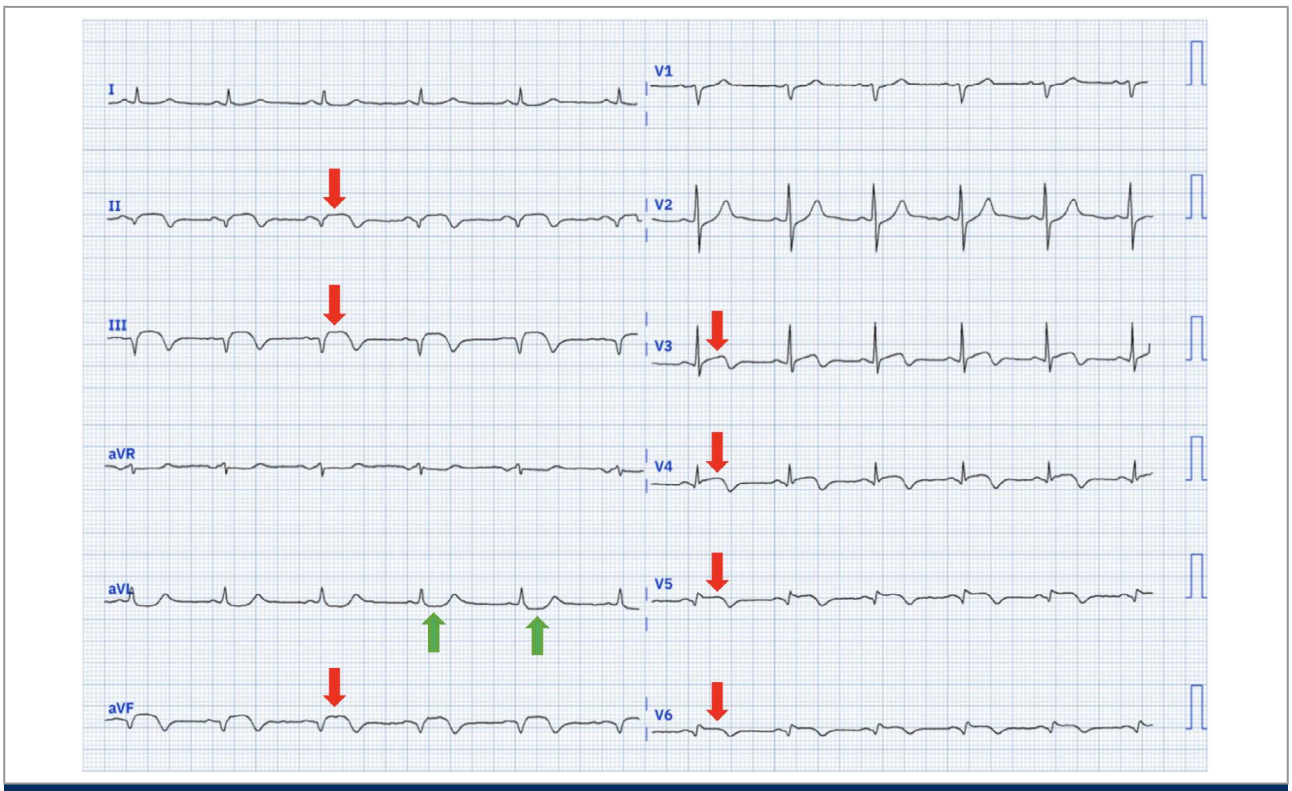

FIGURE 1: Electrocardiogram on initial presentation; 12-lead electrocardiogram showing ST-segment elevation (red arrows) in inferior (II, III, aVF) and anterolateral leads (V3–V6), with reciprocal ST-segment depression in lead aVL (green arrows).

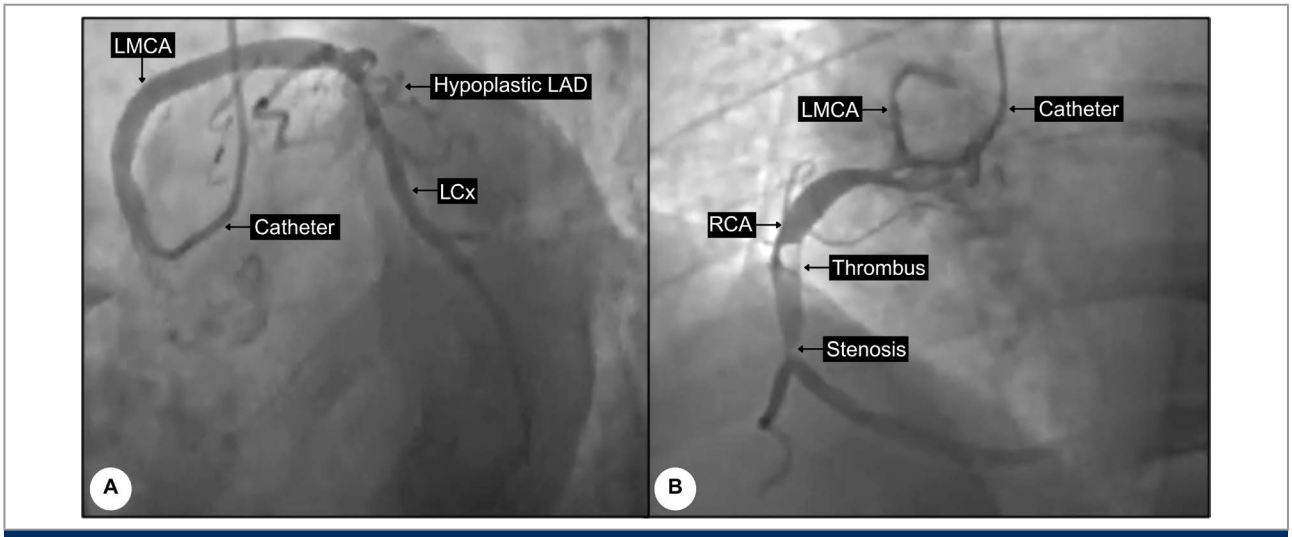

FIGURE 2: Angiogram of the right and left coronary arteries.
A: Invasive coronary angiography LAO caudal "spider view" revealing the LMCA arising from the right coronary cusp, a hypoplastic LAD, and the LCx. B: LAO cranial view showing that both the LMCA and RCA originate from a common ostium in the right coronary cusp. A subtotal stenosis of the RCA with complete occlusion of its distal branches – the PDA and posterolateral branches. Proximal to the stenosis, a persistent filling defect was visualised in multiple projections throughout the cardiac cycle and on repeat contrast injections, characterised by haziness and contrast staining. The defect was consistent with an intraluminal thrombus, thereby excluding an air bubble.
LAD: left anterior descending artery, LCx: left circumflex artery, LMCA: left main coronary artery, LOA: left anterior oblique, PDA: posterior descending artery, RCA: right coronary artery.

Upon arrival at our institution, the patient underwent emergent coronary angiography (Figure 2), revealing a LMCA arising from the right coronary cusp. Proximally, the left circumflex artery (LCx) was of adequate calibre, with a mid-type A lesion and 40% stenosis. A CTO of the LAD was suspected, as the vessel was not clearly visualised; instead, several rudimentary, short vessels extending anteriorly to the mid-ventricle were observed, supplying portions of the expected LAD territory. No definitive proximal cap or abrupt cut-off was identified, and there was no evidence of calcification. Interrogation of the right cardiac circulation showed a RCA of normal origin with a tight 90% mid-RCA stenosis, accentuated by a TIMI 3 thrombus burden proximal to the lesion, suggestive of an acute thrombotic event superimposed on a high-grade chronic lesion. The posterior descending artery (PDA) and posterolateral branches were poorly visualised, suggesting complete occlusion of these vessels. Ventriculography of the left ventricle demonstrated reduced ventricular contractility and inferior wall hypokinesia. This finding supports designating the RCA as the infarct-related artery, consistent with the clinical presentation and ECG changes, because the RCA potentially subtended both the inferior wall and anterior segments through collateral vessels.

The patient was transferred to the cardiac intensive care unit (CCU) for a 24-hour tirofiban infusion. Post-infusion transthoracic echocardiogram confirmed a reduced ejection fraction of 40–45% and inferior wall hypokinesia. CCTA (Figure 3) was requested to further study the anatomy of the LAD and the course of the anomalous LMCA. Imaging confirmed an anomalous origin of the LMCA arising from the RCA ostium with a prepulmonic course. This anomalous vessel gave rise to a

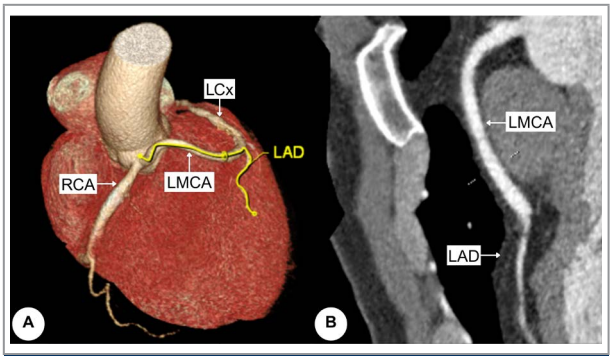

FIGURE 3: Coronary computed tomography angiography with reconstructed views (LMCA and LAD course is highlighted in yellow).
A: Three-dimensional reconstruction of the heart and coronary vessels showing the RCA, LMCA originating from the right coronary cusp, with a prepulmonic course, the LCx, and hypoplastic LAD.
B: Multiplanar reconstruction of the hypoplastic LAD originating from an anomalous LMCA.
LAD: left anterior descending artery, LCx: left circumflex artery, LMCA: left main coronary artery, RCA: right coronary artery.

diminutive LAD that coursed in the anterior interventricular groove but terminated mid-ventricle – findings that refuted the initially suspected CTO – and demonstrated collateral branches from the RCA supplying the LAD territory.

The remainder of the hospital admission was uneventful. The patient was discharged home on dual antiplatelet therapy, high-dose statin, and oral antihyperglycaemic therapy. At the routine 1-month follow-up, the patient reported ongoing angina,

prompting initiation of atenolol 25 mg daily. Due to persistent symptoms despite uptitration of medical therapy, the patient was scheduled for an elective coronary angiogram, which demonstrated a persistent mid-RCA stenosis exceeding 75%. The RCA was successfully engaged, dilated, and stented. The final angiogram confirmed successful revascularisation.

DISCUSSION

We report a rare case of two concurrent coronary anomalies confirmed by ECG, coronary angiography, and CCTA findings. Unusually, the patient's initial ECG showed ST-segment elevation in both the LAD and RCA territories. Angiography revealed an anomalous LMCA arising from the right coronary cusp, sharing a common ostium with the RCA. CCTA further demonstrated a rudimentary, hypoplastic LAD, refuting the initial suspicion of CTO. Failure to recognise an anomalous coronary origin or branching pattern can lead to misinterpretation during angiography, potentially resulting in an incorrect diagnosis or suboptimal management strategy. It also poses a risk of iatrogenic injury during cardiac interventions. Multiple cardiac risk factors accelerated coronary atherosclerosis, leading to mid-RCA and LCx stenosis, and eventual STEMI of the RCA territory. As the RCA supplied portions of the anterior and anterolateral walls via small collateral branches (typically supplied by the LAD), these regions were also affected.

Literature review

STEMI typically results from single-vessel occlusion, with simultaneous involvement of two arteries being exceptionally rare and associated with a poor prognosis.[4] Known risk factors include dyslipidaemia, diabetes, hypertension, tobacco use, and a family history of coronary artery disease. The mechanism behind dual-vessel STEMI remains unclear but is often linked to vasospasm, thrombophilia, or cocaine use.[4] To our knowledge, no prior reports describe ST-segment elevation in two vascular territories in a patient with dual congenital coronary anomalies, highlighting the rarity of our findings.

The prevalence of anomalous coronary arteries originating from the opposite coronary cusp ranges from 0.15% to 0.84%, with LMCA arising from the right cusp in only 0.03–0.28% of cases.[1,2] Hypoplastic coronary artery disease (HCAD) is a rare congenital underdevelopment of a major epicardial artery – a rudimentary vessel with a shortened course or luminal diameter < 1.5 mm, reported in fewer than 35 cases, with only 10 involving the LAD.[1,2,5,6] HCAD increases the risk of myocardial ischaemia and sudden cardiac death. The coexistence of two coronary anomalies is extremely rare, given their usual isolation in congenital cases.[6]

While both anomalous coronary origins and hypoplastic coronary arteries have been described independently, their coexistence remains exceptionally rare. One case report described a hypoplastic left coronary artery with an anomalous origin from the left ventricular outflow tract, emphasising the role of advanced imaging in defining complex congenital

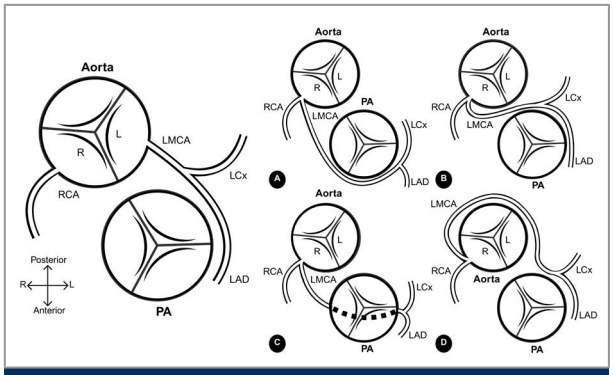

FIGURE 4: Schematic illustration of LMCA pathways.
Left panel: Normal LMCA originates from the left coronary cusp and its bifurcation into the LAD and LCx.
Right panel: Anomalous LMCA arising from the right coronary cusp with four potential courses: (A) prepulmonic, (B) interarterial, (C) subpulmonic, and (D) retroaortic.[9] In our patient, the anomalous LMCA followed the prepulmonic course (A).
L: left, LAD: left anterior descending artery, LCx: left circumflex artery, LMCA: left main coronary artery, PA: pulmonary artery, R: right, RCA: right coronary artery.

anatomy.[7] Another report documented a single coronary artery arising from the right sinus of Valsalva, in which the LAD was hypoplastic.[8] However, to our knowledge, no published cases to date have described an anomalous LMCA arising from the right coronary cusp in conjunction with a hypoplastic LAD in a living patient presenting with acute STEMI. This highlights the clinical novelty of the case and expands on the limited literature regarding dual congenital coronary anomalies.

Pathophysiological considerations

Coronary artery embryonic development begins with intramural vessels within the primordial myocardium joining a subepicardial vascular network, forming the right and left coronary arteries by day 14 of embryonic growth. Congenital coronary anomalies result from abnormal ingrowth of the initially formed subepicardial vascular plexus into the aortic root during embryonic development and include those arising from aberrant sinus origins, intrinsic arterial anatomical abnormalities, or termination defects.[9] In most instances, these anomalies support foetal myocardial development to ensure post-natal function.[9] Approximately 26% of coronary artery anomalies are associated with abnormalities of the aortic root, including bicuspid aortic valve or asymmetry in the size and morphology of the aortic valve leaflets.[10] In this patient, the coexistence of a bicuspid aortic valve was excluded using both transthoracic echocardiography and CCTA.

Anomalous LMCA from the right cusp can follow four potential courses (Figure 4): prepulmonic (anterior to the right ventricular outflow tract), interarterial (between the aorta and pulmonary artery), subpulmonic (intramyocardial within the interventricular septum), or retroaortic (posterior to the aortic root).[9] The prepulmonic course, as identified in this patient, is typically considered haemodynamically benign and is less likely to

predispose to myocardial ischaemia and sudden cardiac death. Unlike the interarterial course – where the artery passes between the aorta and pulmonary artery and may be subjected to lateral compression during systole, a phenomenon that can be exacerbated by physical activity due to increased great vessel wall stress – the prepulmonic course avoids external compression by major vascular structures.[2]

Clinical presentation and diagnostic challenges

Congenital coronary artery anomalies present variably, with symptoms in ~20% of cases.[11] Sudden cardiac death is often due to ventricular arrhythmia following exertion-induced myocardial ischaemia. In an anomalous LMCA, ischaemia may result from slit-like or stenotic ostial abnormalities or interarterial compression by major arteries.[2] The anomalous origin of the LMCA from the right coronary sinus is consistently associated with sudden cardiac death in 59% of reported cases, with 81% of these events triggered by physical activity.[12]

HCAD-related ischaemia arises from thrombosis, coronary spasm, or physical exertion, further restricting blood flow to an already narrowed arterial lumen.[6] Whether coronary anomalies independently contribute to obstructive atherosclerosis remains debated.[11] Though most anomalies are asymptomatic, clinical features include exertional dyspnoea, chest pain, syncope, palpitations, or dizziness.[6] The high prevalence of asymptomatic cases before critical events highlights the challenge of risk assessment and the lack of screening guidelines.

Management and therapeutic implications

Due to the complex pathophysiology, treatment must be individualised based on anatomy, coexisting disease, and imaging findings. The 2018 American Heart Association (AHA)/ American College of Cardiology (ACC) guidelines recommend beta-blockade, angioplasty with stenting, or surgical repair for an anomalous LMCA originating from the right coronary cusp.[13] Surgical intervention is a 2018 AHA/ACC class I recommendation for symptoms or diagnostic evidence of ischaemia and a class IIa recommendation for asymptomatic cases without ischaemia.[13] Though rare, coronary artery bypass grafting has been employed in single-vessel hypoplasia, with its feasibility largely dependent on anatomical considerations and disease distribution.[5] HCAD treatment is limited due to its diffuse nature. Implantable cardioverter-defibrillator placement is recommended for secondary prevention of arrhythmia and sudden cardiac death.[5,6]

The management of this patient highlights significant challenges faced within the public healthcare system in Johannesburg. The patient presented late to a peripheral hospital and was initially managed medically with fibrinolysis, given the anticipated delays in transfer to a PCI-capable facility. This reflects the ongoing difficulty in securing timeous ICU ambulance transport. Despite failed thrombolysis, rescue PCI could not be performed due to logistical constraints. At our facility, coronary angiography was performed 78 hours after symptom onset. A relook angiogram following tirofiban infusion was not done, given high patient volumes and the absence of ongoing symptoms more than a week after presentation. Further delays were encountered in obtaining a CCTA scan, which was only available at an external site at the time.

Consequently, the patient was managed as having chronic coronary syndrome. Recurrent anginal episodes despite optimal medical therapy necessitated PCI, directed at the atherosclerotic RCA stenosis, considered the primary culprit lesion, with a favourable outcome. Although surgical repair remains the recommended intervention for anomalous coronary arteries associated with ischaemia, treatment options for HCAD are limited due to its diffuse nature. In this patient, surgical revascularisation was not feasible, as the LAD was a rudimentary vessel and the distal RCA branches supplying the anterior left ventricle were too small to serve as suitable graft targets.

Though the patient's ischaemic symptoms were attributed to atherosclerotic RCA and LCx stenosis, surgery remains the recommended treatment for anomalous coronary arteries with ischaemia. In the limited literature describing similar anomalies, surgical or conservative strategies were typically pursued depending on anatomical feasibility and the presence of ischaemia. This case contributes to existing reports by demonstrating the feasibility of a non-surgical management pathway in the context of complex dual coronary anomalies with concomitant atherosclerotic coronary artery disease.

The lifetime risk associated with anomalous coronary arteries remains unclear, highlighting the need for multicentre registry data to inform evidence-based guidelines. Currently, there is no consensus on diagnostic or management strategies for congenital coronary variants. Further longitudinal studies are needed to guide patient selection for revascularisation versus conservative management.[14]

CONCLUSION

This case report highlights the rare coexistence of two distinct congenital coronary artery anomalies in a patient presenting with STEMI. Although the acute presentation was primarily attributed to atherosclerotic coronary artery disease and thrombosis, the incidental discovery of these anomalies complicated clinical decision-making by influencing both diagnostic interpretation and the timing of intervention. The case underscores the importance of recognising coronary anomalies, particularly in patients with atypical electrocardiographic findings. Furthermore, it emphasises the challenges in evaluating individuals at risk for sudden cardiac events, given the often asymptomatic nature of these anomalies.

Management should be individualised based on anatomical characteristics, coexisting coronary disease, and functional imaging. While surgical correction remains the definitive treatment for high-risk coronary anomalies, medical management and percutaneous interventions may be appropriate in select cases.

Moreover, this case reinforces the need for long-term follow-up and surveillance in patients with coronary artery anomalies, especially those involving the LMCA, due to the potential risk of sudden cardiac death – even in the context of a haemodynamically benign course. The rarity of this dual anomaly, with associated acute coronary syndrome, highlights the need for further research to better understand its clinical implications, guide management strategies, and clarify long-term outcomes.

DECLARATION OF COMPETING INTEREST

The authors declare no conflict of interest.

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PATIENT CONSENT STATEMENT

Informed consent was obtained from the patient before the publication of this case report with its accompanying images.

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