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Transient Cardiac Dysfunction Due to New-Onset Mitral Chordal Rupture With Concomitant Congenital Absence of the Right Coronary Artery: A Case Report

Authors' Contribution:
Study Design A
Data Collection B
Statistical Analysis C
Data Interpretation D
Manuscript Preparation E
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Patient: Female, 58-year-old
Final Diagnosis: Congenital absence of the RCA and newonset mitral chordal rupture
Symptoms: Exertional chest pain
Clinical Procedure: —
Specialty: General and Internal Medicine

Objective: Rare disease

Background: Congenital absence of the right coronary artery (RCA) is a rare anomaly often identified incidentally. We report a case of transient left ventricular systolic dysfunction followed by recovery of ejection fraction and new-onset mitral chordal rupture, which became the primary driver of pronounced N-terminal pro-B-type natriuretic peptide (NT-proBNP) elevation. The coexistence of 2 distinct pathologies complicated causal attribution; we discuss proposed mechanisms while recognizing their limitations.

Case Report: A 58-year-old woman presented with exertional chest pain. At initial admission, she exhibited sinus tachycardia, elevated NT-proBNP (910 pg/mL), and reduced left ventricular ejection fraction (LVEF; 43% [M-mode]) with hypokinesis of the anterior wall and anteroseptum. Symptoms improved with anti-ischemic therapy. Three weeks later, LVEF recovered to 56% (biplane Simpson method), but NT-proBNP increased to 2440 pg/mL. Echocardiography revealed rupture of a small chord of the anterior mitral leaflet with mild-to-moderate regurgitation and elevated filling pressures (E/e'=13.8). Coronary angiography demonstrated congenital absence of the RCA ostium, with an enlarged left circumflex artery supplying the RCA territory and no evidence of atherosclerotic stenosis. Retrospective quantitative mitral regurgitation indices from archived images were examined. Management was conservative; symptoms resolved and NT-proBNP levels decreased during follow-up.

Conclusions: In patients with rare coronary anomalies, newly acquired common valvular disease may dominate the clinical presentation. Transient systolic dysfunction and subsequent chordal rupture occurred in our patient; a direct causal relationship remains speculative. Multimodality imaging is essential to accurately attribute hemodynamic changes and guide therapy. Long-term surveillance of coronary anatomy and valve function is recommended.

Keywords: Cardiology • Case Reports • Coronary Vessel Anomalies • Echocardiography • Heart Failure • Mitral Valve Insufficiency

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Introduction

Congenital coronary artery anomalies have an overall prevalence of approximately 1.33% in the general population [1]. Congenital absence of the right coronary artery (RCA) is an extremely rare subtype in which the RCA fails to develop, and perfusion of its territory entirely depends on compensatory supply from the left coronary system [2]. Because compensatory capacity varies, many affected individuals remain asymptomatic and are diagnosed incidentally; a minority develop ischemic symptoms when myocardial demand transiently exceeds supply or when additional cardiac pathology coexists [3]. Importantly, the presence of a rare congenital anomaly does not confer immunity against common acquired cardiac diseases; instead, these conditions may coexist and create a complex clinical presentation. Attributing all new symptoms or findings to the congenital anomaly risks overlooking more common and treatable conditions. In this report, we describe a patient with congenital absence of the RCA who exhibited transient left ventricular systolic dysfunction followed by recovery of ejection fraction and subsequent new-onset mitral chordal rupture. We emphasize that the chordal rupture, likely a degenerative event, became the predominant cause of biomarker elevation and hemodynamic changes. We also discuss the diagnostic challenge of distinguishing coincident pathologies from a unified causal process.

Case Report

A 58-year-old Han Chinese woman presented with 1 week of intermittent retrosternal pressure-like chest pain that had worsened over the preceding 2 days. The pain lasted 5 to 10 minutes per episode, was provoked by exertion, and did not clearly improve with rest. It was not associated with radiation, diaphoresis, dyspnea, or nausea. The patient had no history of smoking, alcohol use, hypertension, diabetes mellitus, or hyperlipidemia, as well as no family history of congenital heart disease or premature cardiovascular disease. On physical examination, her temperature was 36.4°C, heart rate was 98 beats/min, respiratory rate was 20/min, and blood pressure was 137/97 mmHg. The lungs were clear to auscultation, heart sounds were regular without murmurs, and no peripheral edema was evident.

During the first admission (October 15, 2024), electrocardiography showed sinus tachycardia at 101 bpm, high left ventricular voltages, poor R-wave progression, and ST-segment depression in leads V4 to V6 (**Figure 1A**). Laboratory tests revealed mildly elevated inflammatory markers (C-reactive protein and neutrophil percentage), substantially elevated N-terminal pro-B-type natriuretic peptide (NT-proBNP; 910 pg/mL), and normal myocardial injury markers (troponin I and creatine kinase–myocardial band [CK-MB] mass). Renal function was preserved (serum creatinine 73.0 µmol/L; estimated glomerular filtration rate [eGFR] 78.46 mL/min/1.73 m^2). Bedside transthoracic echocardiography demonstrated a left ventricular end-diastolic diameter (LVEDD) of 52 mm, left atrial dimension of 38 mm, and reduced left ventricular ejection fraction (LVEF) of 43% (estimated by M-mode), with regional wall motion abnormalities characterized by hypokinesis of the anterior wall and anteroseptum. Mild mitral regurgitation and mild tricuspid regurgitation were qualitatively reported. No quantitative mitral regurgitation parameters were recorded during this bedside study. Chest computed tomography excluded pulmonary inflammation and pleural effusion (**Figure 2**). A presumptive diagnosis of coronary atherosclerotic heart disease with acute heart failure was made; the patient was treated with aspirin, isosorbide dinitrate, metoprolol succinate (23.75 mg once daily), furosemide, spironolactone, and rosuvastatin. Her chest pain improved within 3 days, and NT-proBNP decreased to 680 pg/mL. She declined the recommended coronary computed

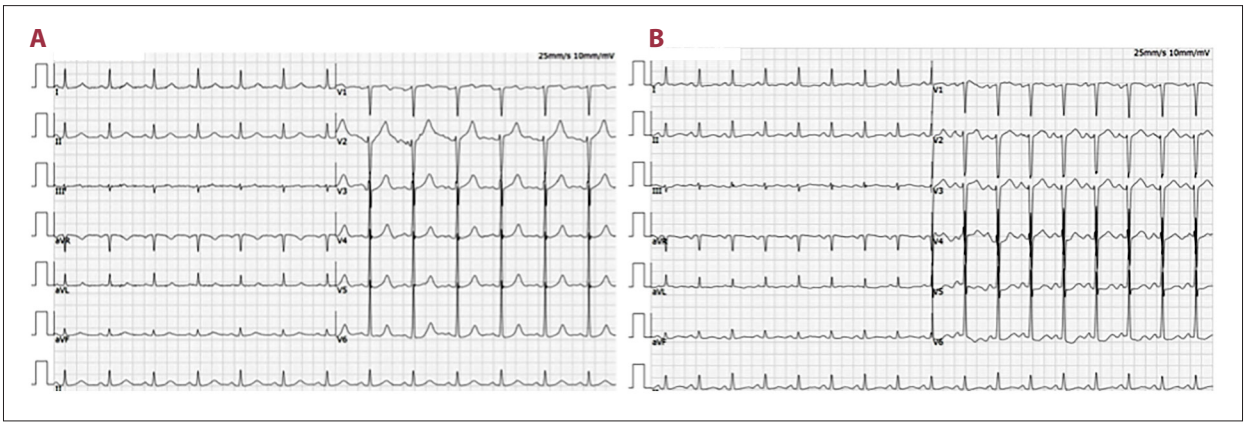

Figure 1. Electrocardiograms. (**A**) First admission: sinus tachycardia (101 bpm), high left ventricular voltages, poor R-wave progression, and ST-segment depression (0.1 mV) in leads V4-V6. (**B**) Second admission: sinus rhythm (72 bpm), occasional premature ventricular contractions, and no dynamic ST-T changes.

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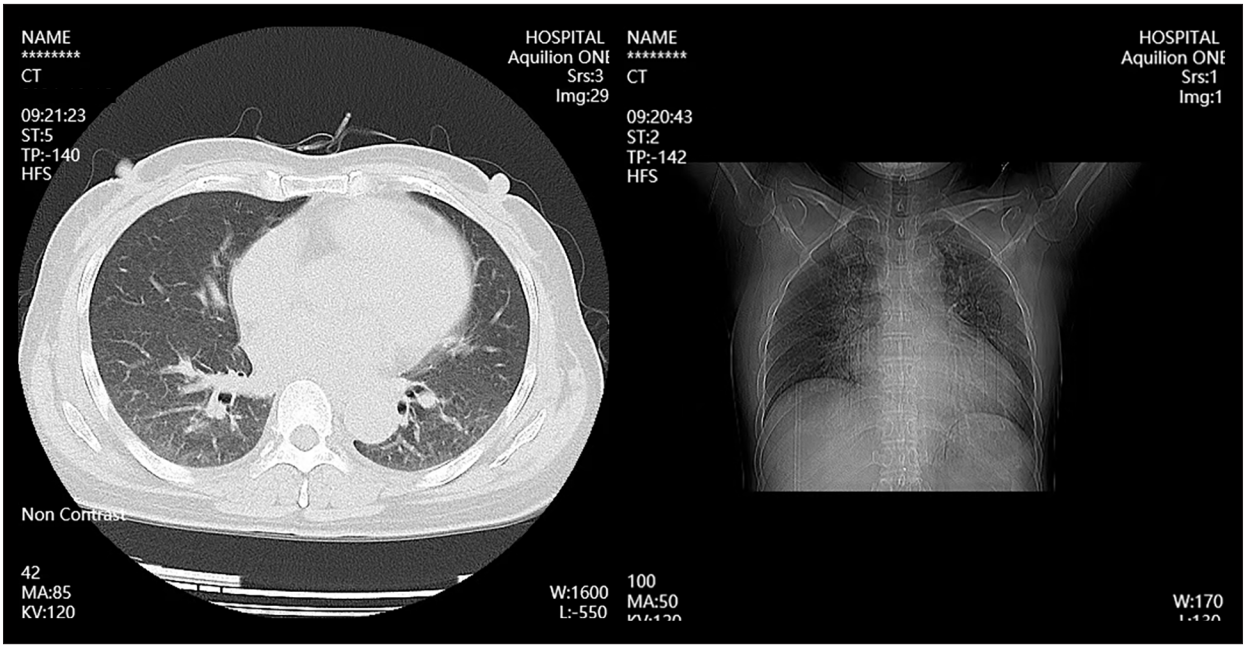

Figure 2. Chest computed tomography demonstrating no evidence of pulmonary inflammation or pleural effusion. This study was performed to exclude pulmonary causes of chest pain.

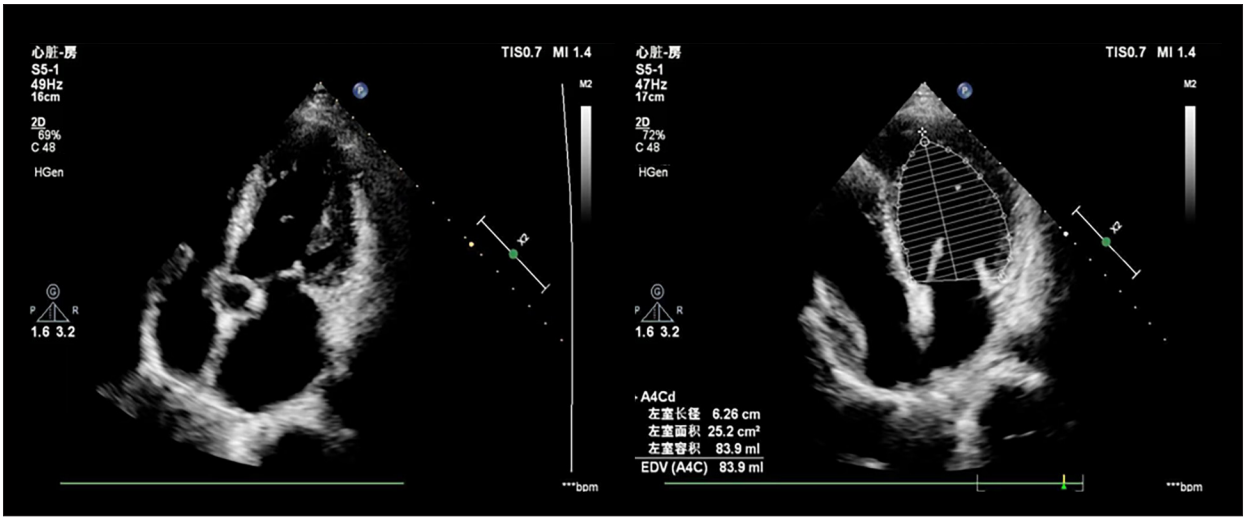

Figure 3. Transthoracic echocardiography. Echocardiographic findings at the second admission demonstrated rupture of a small chord of the anterior mitral leaflet with poor leaflet coaptation, resulting in mild-to-moderate mitral regurgitation. Doppler analysis showed impaired left ventricular relaxation and elevated filling pressures.

tomography angiography and invasive coronary angiography, then was discharged.

Despite initial improvement, the patient returned 3 weeks later (November 6, 2024) with persistent exertional chest pain, now occurring 3 to 4 times per week and lasting up to 15 minutes. Physical examination findings were similar, and no new murmurs were detected. Repeat electrocardiography showed sinus rhythm with occasional premature ventricular contractions and no dynamic ST-T changes (**Figure 1B**). Myocardial injury markers remained normal, but NT-proBNP had substantially risen to 2440 pg/mL; renal function was preserved (serum creatinine 59.4 µmol/L; eGFR 97.11 mL/min/1.73 m²). A second transthoracic echocardiogram (**Figure 3**), performed using the biplane Simpson method, demonstrated an LVEDD of 49 mm, left atrial dimension of 39 mm, and recovered LVEF of 56% (end-diastolic volume 84 mL; end-systolic volume 37 mL). Although different methods were used to assess LVEF (M-mode vs biplane Simpson), the substantial improvement, together with normalization of the previously observed wall

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Table 1. Key transthoracic echocardiography parameters at first and second admissions.

Parameter	First admission (October 15, 2024)	Second admission (November 6, 2024)
LVEDD (mm)	52	49
LA (mm)	38	39
LVEF (%)	43 (M-mode)	56 (biplane Simpson)
Regional wall motion	Anterior and anteroseptal hypokinesis	Normal
Mitral regurgitation	Mild (qualitative)	Mild-to-moderate; vena contracta 3 mm, EROA 0.15 cm^2, regurgitant volume 25 mL
E-wave velocity (m/s)	—	0.69
A-wave velocity (m/s)	—	0.95
E/A ratio	—	0.7
Septal e' velocity (cm/s)	—	4.41
Lateral e' velocity (cm/s)	—	5.61
E/e' ratio	—	13.8

Abbreviations: EROA, effective regurgitant orifice area; LA, left atrial dimension; LVEDD, left ventricular end-diastolic diameter; LVEF, left ventricular ejection fraction.

motion abnormalities, strongly suggested true myocardial recovery rather than measurement artifact. Furthermore, the study revealed rupture of a small chord of the anterior mitral leaflet with poor coaptation, resulting in mild-to-moderate mitral regurgitation. Doppler measurements included an E-wave velocity of 0.69 m/s, A-wave velocity of 0.95 m/s, E/A ratio of 0.7, septal e' velocity of 4.41 cm/s, lateral e' velocity of 5.61 cm/s, and E/e' ratio of 13.8, indicating impaired left ventricular relaxation with mildly elevated filling pressures [4]. Retrospective quantitative analysis of archived images showed a vena contracta width of 3 mm, effective regurgitant orifice area (EROA) of 0.15 cm^2, and regurgitant volume of 25 mL, confirming mild-to-moderate severity (**Table 1**). On November 8, 2024, coronary angiography demonstrated a normal left coronary origin but no RCA ostium on selective or nonselective injection. The left circumflex artery was greatly enlarged and supplied the entire RCA territory, consistent with left-dominant circulation. No atherosclerotic stenosis was identified. A grade 1 myocardial bridge of the mid-left anterior descending artery was also noted but was considered hemodynamically insignificant [5] (**Figure 4**).

Given the diagnosis of congenital absence of the RCA and new-onset mitral chordal rupture, medical therapy was optimized: metoprolol succinate was increased to 47.5 mg once daily, furosemide was adjusted to 20 mg twice daily, and valsartan 80 mg once daily was added. The patient's chest pain resolved, and NT-proBNP decreased to 920 pg/mL within 5 days. She was discharged with a plan for close follow-up. Three months later, she remained free of chest pain, dyspnea, and edema. NT-proBNP had further decreased to 620 pg/mL; echocardiography demonstrated a stable LVEF of 58% with persistent mild-to-moderate mitral regurgitation and no evidence of progression. Premature ventricular contractions were no longer observed.

Discussion

The diagnosis of congenital absence of the RCA in our patient was clearly established by coronary angiography, which demonstrated absence of the RCA ostium and a hyperdominant left circumflex artery perfusing the right ventricular and inferior left ventricular walls. Although coronary computed tomography angiography is the noninvasive gold standard for excluding other anomalies, such as anomalous RCA origin from the pulmonary artery (ARCAPA), the angiographic findings and absence of retrograde collateral filling made ARCAPA unlikely [6,7].

The present case is instructive because the patient experienced 2 distinct cardiac events—transient left ventricular systolic dysfunction and new-onset mitral chordal rupture—in the setting of a rare congenital coronary anatomy. The key point is not to establish a continuous pathophysiological sequence, but rather to recognize that these events were most likely coincidental. The initial episode of chest pain, ST-segment depression, regional wall motion abnormalities, and reduced LVEF is consistent with transient myocardial ischemia due to demand-supply mismatch in a single-coronary system [8]. The absence of elevated troponin and rapid recovery of systolic function with anti-ischemic therapy and afterload reduction support a

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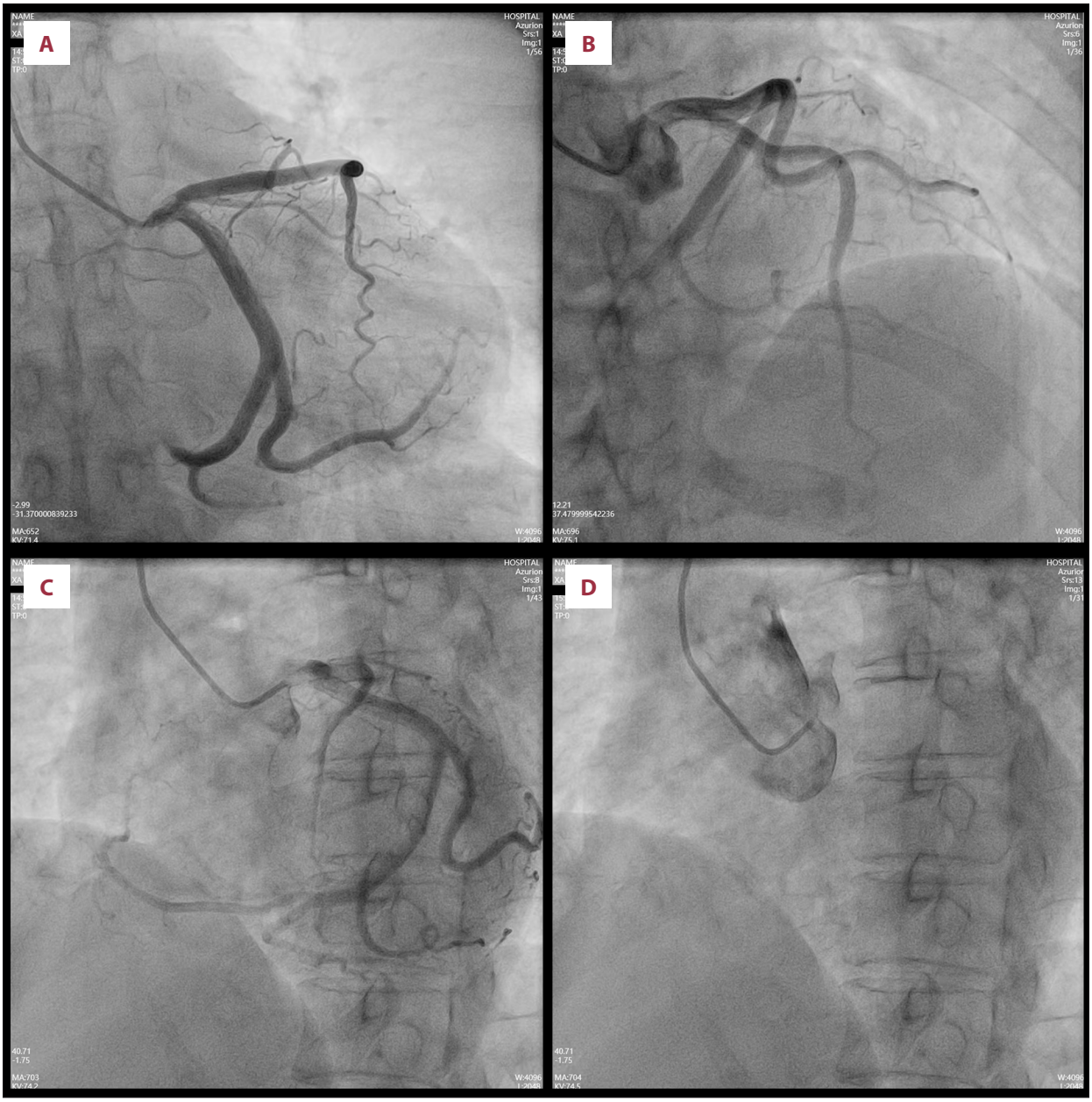

Figure 4. Coronary angiography demonstrating congenital absence of the right coronary artery (RCA). Panels show (**A**) cranial view, (**B**) caudal view, (**C**) left anterior oblique view, and (**D**) nonselective aortic root injection demonstrating absence of the RCA ostium. The left circumflex artery is substantially enlarged and supplies the RCA territory.

reversible ischemic insult rather than irreversible myocardial injury. Upon restoration of hemodynamic balance, ventricular wall motion normalized and LVEF recovered to 56%.

The subsequent pronounced elevation of NT-proBNP to 2440 pg/mL, despite normalization of ejection fraction, is best explained by the newly diagnosed mitral chordal rupture. Acute or subacute mitral regurgitation leads to sudden volume overload, increased left atrial pressure, and robust secretion of natriuretic peptides even when systolic function is preserved [9]. Diastolic parameters (E/e' 13.8 and reduced tissue Doppler velocities) confirmed elevated left ventricular filling pressures, consistent with this mechanism.

What, then, caused the chordal rupture? Common etiologies include degenerative mitral valve disease (myxomatous degeneration), infective endocarditis, trauma, papillary muscle ischemia, and connective tissue disorders [10]. In our patient, troponin levels remained consistently normal; coronary angiography showed no occlusive disease or evidence of papillary

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muscle ischemia. There was no history of trauma, no clinical or echocardiographic evidence of endocarditis, and no systemic features suggestive of connective tissue disease. The most plausible explanation is spontaneous (likely degenerative) rupture of the anterior mitral leaflet chordae—a relatively common valvular event that happened to occur in a patient with a rare coronary anomaly. Any hypothesis proposing that chronic single-coronary perfusion directly caused subvalvular degeneration remains speculative and unsupported by current evidence. Accordingly, we regarded mitral chordal rupture as the primary cause of transient cardiac decompensation; congenital RCA absence served as a concomitant anatomical background.

This case carries an important clinical message: when a rare congenital anomaly is identified, there is a tendency to attribute all subsequent clinical findings to it, a form of diagnostic anchoring. Clinicians should resist this impulse and systematically evaluate acquired conditions that may coexist coincidentally. In our patient, detailed repeat echocardiographic examination identified the chordal rupture, thus clarifying the reason for paradoxical biomarker elevation and redirecting management toward heart failure therapy and serial valve surveillance.

Regarding measurement methodology, we acknowledge that the first LVEF was estimated using M-mode, whereas the second was assessed via the biplane Simpson method. Although M-mode may overestimate or underestimate true ventricular volumes, the concordant resolution of regional wall motion abnormalities provides additional confidence that genuine functional recovery occurred. Whenever possible, serial LVEF assessments should be performed using the same quantitative method to improve comparability. This limitation was unavoidable in the acute bedside setting but should be considered when interpreting the clinical trajectory.

Treatment of congenital RCA absence in the absence of atherosclerotic stenosis is generally conservative and aims to prevent ischemia using beta-blockers and nitrates, whereas heart failure therapies (angiotensin-converting enzyme inhibitors/angiotensin receptor blockers and mineralocorticoid receptor antagonists) may be used when ventricular remodeling or volume overload is present. For the mitral valve, mild-to-moderate regurgitation in a clinically stable patient warrants medical optimization and regular echocardiographic surveillance. If regurgitation progresses to severe disease or results in refractory symptoms or ventricular dilation, surgical or transcatheter intervention should be considered according to current guidelines [11]. Long-term follow-up with serial echocardiography, quantitative mitral regurgitation grading, and biomarker monitoring is recommended.

Conclusions

This case report describes a patient with congenital absence of the RCA who experienced transient cardiac dysfunction primarily driven by new-onset mitral chordal rupture rather than by the coronary anomaly itself. The transient reduction in LVEF was consistent with reversible ischemia in a single-coronary system, whereas the subsequent biomarker surge and diastolic abnormalities resulted from acute valvular incompetence. The chronological association did not establish a causal relationship between the congenital anomaly and degenerative chordal rupture; the 2 conditions were most likely coincidental. Multimodality imaging, including coronary angiography and quantitative echocardiography, was pivotal in clarifying the respective contributions of each lesion. This case underscores the danger of attributing all clinical findings to a rare anomaly and highlights the need to diligently investigate common acquired diseases that may ultimately determine prognosis. Individualized surveillance of coronary perfusion status and valve function remains essential.

Department and Institution Where Work Was Done

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Patient Consent

Informed consent was obtained from the patient for publication of this case report and any accompanying images.

Declaration of Figures' Authenticity

All figures submitted have been created by the authors who confirm that the images are original with no duplication and have not been previously published in whole or in part.

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