



## Anomalous Origin of the Left Coronary Artery from the Pulmonary Artery (ALCAPA): Post-Gestational Decompensation in an Adult Patient

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### Abstract

Anomalous Origin of The Left Coronary Artery from The Pulmonary Artery (ALCAPA), also known as Bland-White-Garland syndrome, is a rare and potentially lethal congenital heart disease. Although it is typically diagnosed during infancy, the presence of collateral circulation between the right and left coronary arteries may allow survival into adulthood. We report the case of a 36-year-old woman with late-diagnosed ALCAPA following hemodynamic decompensation in the post-gestational period, despite preserved ventricular function. This case highlights the importance of recognizing this anomaly in adults presenting with atypical ischemic symptoms and underscores the need for early surgical correction to prevent fatal outcomes.

**Keywords:** ALCAPA; Bland-White-Garland syndrome; congenital coronary anomaly; collateral circulation; adult survival; surgical correction

### Introduction

Anomalous Origin of The Left Coronary Artery from The Pulmonary Artery (ALCAPA) is a rare congenital cardiac anomaly accounting for approximately 0.25% to 0.5% of all congenital heart diseases [1-4]. First described anatomically by Brooks in 1885 and later clinically characterized by Bland, White, and Garland in 1933, the condition results from an embryological failure in the connection between the left coronary artery and the ascending aorta. Consequently, the left coronary artery arises from the pulmonary artery, leading to abnormal coronary perfusion and progressive myocardial ischemia [5]. In the neonatal period, pulmonary arterial pressure and oxygen content are relatively high, allowing temporary myocardial perfusion through the anomalous vessel. However, as pulmonary vascular resistance decreases after birth, the pressure gradient reverses [6], resulting in reduced perfusion of the left coronary artery and the development of myocardial ischemia. Most symptomatic infants with ALCAPA develop severe myocardial ischemia and heart failure, with mortality rates approaching 90% during the first year of life if untreated. Survival into adulthood is rare and largely depends on the development of extensive collateral circulation between the right and left coronary systems [7-14]. These collaterals allow retrograde perfusion of the left coronary artery, partially compensating for the anomalous origin. Despite this compensatory mechanism, chronic myocardial ischemia persists due to the so called “coronary steal” phenomenon, in which oxygenated blood flows retrogradely from the right coronary artery into the pulmonary artery via the anomalous left coronary artery. Over time, this may result in myocardial fibrosis, ventricular remodelling, mitral valve dysfunction, ventricular arrhythmias, and sudden cardiac death. Herein, we report the case of an adult patient with ALCAPA diagnosed following hemodynamic decompensation during the post-gestational period, emphasizing the diagnostic and therapeutic challenges associated with this rare congenital anomaly in adulthood [15].

### Case Presentation

A 36-year-old female patient presented with a history of seizure episodes between the ages of 5 and 10 years, initially interpreted as epilepsy [16-18]. These episodes resolved spontaneously and did not require long-term pharmacological therapy. There was no known family history of diagnosed congenital heart disease; however, her mother had systemic arterial hypertension, and an aunt had died from acute myocardial infarction under a similar clinical context, which prompted further cardiological investigation. The patient reported a history of exertional dyspnea since the age of seven, although no diagnostic investigation was performed at that time. At the age of 32,

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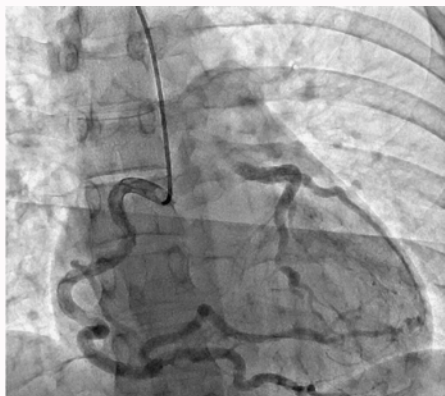
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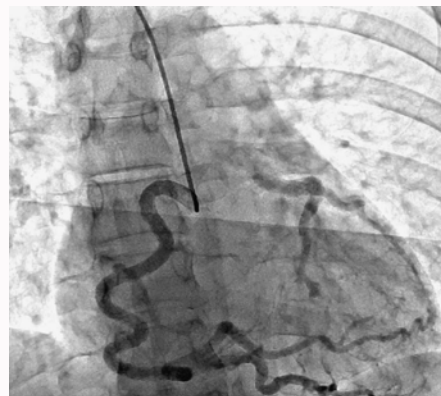
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**Figure 1:** Right coronary angiography demonstrating retrograde filling of the left coronary artery through collateral vessels, with subsequent drainage of the left coronary artery into the pulmonary artery trunk.



**Figure 2:** Right coronary angiography demonstrating retrograde filling of the left coronary artery through collateral vessels, with subsequent drainage of the left coronary artery into the pulmonary artery trunk.

following pregnancy, she experienced progressive worsening of symptoms with functional limitation characterized by dyspnea during minimal exertion. During this period, she began experiencing recurrent episodes of oppressive precordial chest pain without radiation, associated with tachycardia. These episodes typically lasted approximately five minutes, were triggered by mild physical exertion (Figure 1), and resolved spontaneously. Over the following four years, the patient remained symptomatic, with significant impact on her quality of life and daily activities. On physical examination, the patient was in fair general condition, conscious and oriented, and hemodynamically stable. Blood pressure was 120/70 mmHg, heart rate was 73 beats per minute, and peripheral oxygen saturation was 97% on room air.

Cardiac auscultation revealed normal heart sounds with a regular two-beat rhythm, without murmurs, friction rubs, or additional sounds. Pulmonary auscultation demonstrated bilaterally present and well-distributed vesicular breath sounds without adventitious noises. The abdomen was soft and non-tender on palpation, with normal bowel sounds and no evidence of organomegaly [19]. The extremities showed no peripheral edema or signs of venous congestion, and peripheral pulses were palpable with preserved perfusion. Electrocardiography revealed sinus rhythm with left anterior fascicular block. The initial transthoracic echocardiogram demonstrated preserved biventricular function with a left ventricular ejection fraction of 69% [20].

Systolic posterior bulging of the mitral valve leaflets was observed, consistent with mid-to-late systolic mitral valve prolapse without significant functional repercussion. A small pericardial effusion was also noted. A subsequent echocardiographic evaluation showed atrial situs solitus, concordant atrioventricular and ventriculoarterial connections, intact septa, and cardiac chambers with normal dimensions. Global systolic function remained preserved, with a left ventricular global longitudinal strain 17.2%, Tricuspid Annular Plane Systolic Excursion (TAPSE) of 2 cm, and Fractional Area Change (FAC) of 45% the mitral valve exhibited dysplastic features with mild prolapse of the P2 segment and minimal regurgitation.

The remaining cardiac valves showed only trivial regurgitation without hemodynamic significance. A markedly dilated right coronary artery measuring 4.6 mm (Z-score +2.8) was identified. Retrograde filling of the left coronary artery through collateral vessels was also observed, suggesting anomalous origin from the pulmonary artery,

consistent with ALCAPA. Additionally, a fistulous communication between the left coronary artery and the pulmonary trunk was identified, with a flow velocity of 1.69 m/s, along with multiple small coronary cavity fistulas. A laminar pericardial effusion was present posterior to the left ventricle without clinical repercussion (Figure 2). Exercise stress testing was positive for myocardial ischemia based on clinical criteria. The patient developed typical precordial chest pain during exertion and presented a plus-minus type electrocardiographic alteration during recovery. She achieved submaximal heart rate with an adequate blood pressure response and no arrhythmias. Coronary angiography confirmed the anomalous origin of the left coronary artery from the pulmonary trunk.

Extensive collateral circulation from the right coronary artery was observed, supplying the left coronary system retrogradely. The right coronary artery was ectatic, dominant, and free of significant obstructive lesions. The posterior descending and posterior ventricular branches were patent without stenosis. Left ventriculography demonstrated a mildly increased left ventricular volume, although global systolic function and contractility were preserved. Angiography further demonstrated retrograde filling of the left coronary artery via collateral vessels originating from the right coronary artery, followed by drainage into the pulmonary artery (Figure 3). Attempts to selectively catheterize the ostium of the left coronary artery were unsuccessful, further confirming the anomalous origin of the vessel from the pulmonary trunk. Taken together,



**Figure 3:** Aortography showing absence of the left coronary artery origin from the aorta.

these findings established the diagnosis of ALCAPA syndrome with hemodynamic compensation through robust collateral circulation. The condition was associated with dilation of the right coronary artery, mild mitral valve prolapse with minimal regurgitation, and a small pericardial effusion. Medical therapy with metoprolol 50 mg/day was initiated, resulting in symptomatic improvement. Surgical correction was subsequently recommended, consisting of reimplantation of the left coronary artery into the ascending aorta in order to restore dual coronary circulation and prevent potentially fatal long-term complications.

## Discussion

ALCAPA syndrome represents one of the most severe congenital coronary anomalies due to its strong association with myocardial ischemia, ventricular arrhythmias, and sudden cardiac death. Survival into adulthood is uncommon and depends primarily on the development of sufficient collateral circulation capable of perfusing the territory supplied by the left coronary artery [21].

In this case, the patient survived into adulthood owing to the formation of an extensive collateral network between the right and left coronary systems, which allowed adequate myocardial perfusion. However, the retrograde flow into the pulmonary artery perpetuates chronic myocardial ischemia through the coronary steal phenomenon. The patient's clinical decompensation in the post-gestational period was likely precipitated by the physiological increase in metabolic demand and cardiac output associated with pregnancy and the postpartum period [22]. These hemodynamic changes may unmask latent myocardial ischemia in individuals with previously compensated coronary anomalies.

The echocardiographic and angiographic findings observed in this case are characteristic of ALCAPA. These include marked dilation of the right coronary artery, retrograde filling of the left coronary artery, and anomalous origin from the pulmonary artery. Importantly, preserved ventricular function does not exclude the risk of life-threatening complications. Patients with ALCAPA remain at risk for malignant ventricular arrhythmias and sudden cardiac death, even when myocardial function appears normal. For this reason, surgical correction is recommended regardless of symptom severity. Direct reimplantation of the left coronary artery into the ascending aorta is currently considered the gold-standard surgical treatment, as it restores a two-coronary system and normal antegrade coronary perfusion.

Alternative surgical techniques include the Takeuchi procedure, which involves the creation of an intrapulmonary tunnel, or coronary artery bypass grafting combined with ligation of the anomalous origin. These techniques may be considered in cases with complex coronary anatomy. Long-term postoperative follow-up should include regular clinical evaluation and multimodality imaging, including echocardiography and cardiac magnetic resonance imaging. These modalities allow assessment of ventricular function, myocardial fibrosis, and long-term patency of the reimplanted coronary artery [23,24].

## Conclusion

ALCAPA should be considered in the differential diagnosis of adults presenting with atypical ischemic symptoms, particularly in young women with chronic exertional dyspnea and clinical deterioration during the post-gestational period. Although

survival into adulthood may occur due to the presence of collateral circulation, this compensatory mechanism does not eliminate the risk of myocardial ischemia or sudden cardiac death. Early diagnosis through multimodal imaging and prompt surgical correction with coronary reimplantation are essential to prevent late complications and improve long-term prognosis and quality of life.

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