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Anomalous Left Coronary Artery from the Pulmonary Artery Presenting as Acute Respiratory Distress after Feeding in a 3-Month-Old Infant: A Case Report

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Abstract

Background: Anomalous origin of the left coronary artery from the pulmonary artery (ALCAPA), also known as Bland–White–Garland syndrome, is a rare but life-threatening congenital coronary anomaly that may present during infancy with nonspecific symptoms such as feeding difficulty, irritability, failure to thrive, respiratory distress, or heart failure. Because the initial presentation may mimic respiratory or gastrointestinal disease, delayed diagnosis remains an important clinical risk.

Case Presentation: We report a 3-month-old term female infant who presented to the emergency department with acute respiratory distress immediately after vomiting and suspected aspiration of feeds. She had suboptimal weight gain, weighing 3.5 kg below the 3rd centile. Initial examination showed severe respiratory distress, tachypnea, subcostal retractions, head nodding, and oxygen desaturation, but no murmur or signs of poor peripheral perfusion. Chest radiography demonstrated marked cardiomegaly with a cardiothoracic ratio of approximately 67% without pulmonary congestion. Laboratory evaluation showed elevated cardiac biomarkers, including troponin I of 80 ng/mL and creatine kinase-MB of 28.9 U/L. Electrocardiography demonstrated ischemic changes with pathological Q waves and lateral ST-segment depression with T-wave inversion. Echocardiography revealed a severely dilated left ventricle with reduced systolic function, estimated ejection fraction of approximately 30%, anomalous origin of the left main coronary artery from the main pulmonary artery, retrograde flow in the left anterior descending coronary artery, mild mitral regurgitation, patent foramen ovale with left-to-right shunt, and mild pulmonary hypertension. The infant was stabilized with non-invasive ventilatory support, heart-failure therapy, antiplatelet treatment, nutritional support, and referral for definitive surgical correction.

Conclusion: This case highlights the importance of considering ALCAPA in infants with unexplained cardiomegaly, failure to thrive, respiratory distress, or apparent feeding-related respiratory symptoms. Chest radiography and electrocardiography are valuable early screening tools, but prompt echocardiographic assessment of coronary origins is essential for diagnosis and timely referral for surgical repair.

Keywords: ALCAPA; Bland–White–Garland syndrome; Congenital coronary anomaly; Infant heart failure; Respiratory distress; Case report

Introduction

Anomalous origin of the left coronary artery from the pulmonary artery (ALCAPA), also known as Bland–White–Garland syndrome, is a rare congenital coronary artery anomaly in which the left coronary artery arises from the pulmonary artery rather than the left coronary sinus of the aorta. It is estimated to occur in approximately 1 in 300,000 live births and accounts for about 0.25–0.5% of congenital heart disease cases [1,2]. However, the true incidence may be underestimated because some affected infants may die before diagnosis or may initially be misclassified as having respiratory, gastrointestinal, myocarditis-like, or nonspecific infantile illness [2,3].

The pathophysiology of ALCAPA evolves after birth. During fetal life, high pulmonary arterial pressure and relatively oxygenated pulmonary arterial blood may allow adequate myocardial perfusion despite the anomalous coronary origin. After birth, pulmonary vascular resistance falls and pulmonary arterial oxygen content decreases. As a result, perfusion through the left coronary artery becomes inadequate, and blood may flow retrogradely from the right coronary artery through collateral vessels into the left coronary system and then into the low-pressure pulmonary artery. This “coronary steal” phenomenon causes chronic myocardial ischemia, left ventricular dysfunction, mitral regurgitation due to papillary muscle ischemia, and progressive congestive heart failure [1,4].

Infants with ALCAPA commonly present within the first few months of life with nonspecific symptoms including poor feeding, diaphoresis during feeds, irritability, failure to thrive, tachypnea, recurrent respiratory distress, or signs of heart failure. Because these symptoms overlap with common pediatric conditions such as gastroesophageal reflux, infantile colic, bronchiolitis, pneumonia, aspiration, myocarditis, or dilated cardiomyopathy, clinical suspicion may be delayed [3,5]. Without surgical correction, infantile ALCAPA has historically been associated with very high mortality, whereas timely surgical establishment of a two-coronary-artery system can markedly improve survival and allow recovery of ventricular function [4,6]. Recent surgical series continue to support coronary reimplantation as the preferred corrective approach when anatomically feasible, with favorable intermediate- and long-term outcomes even among children presenting with severe left ventricular dysfunction [6,7]. Initial investigations may provide important diagnostic clues. Chest radiography may show cardiomegaly, while electrocardiography classically demonstrates features of anterolateral myocardial ischemia or infarction, including deep Q waves, ST-segment changes, and T-wave inversion in leads I, aVL, and the left precordial leads [8].

However, ECG findings may vary and should not be used alone to exclude the diagnosis. Echocardiography with color Doppler is the key diagnostic modality, allowing assessment of coronary artery origin, retrograde flow into the pulmonary artery, left ventricular dilation and dysfunction, mitral regurgitation, and associated pulmonary hypertension [1,5]. Computed tomography angiography or cardiac catheterization may be used when echocardiographic findings are inconclusive or when additional anatomical definition is required before surgical repair [1,5].

We report a 3-month-old infant who presented with acute respiratory distress after vomiting and suspected aspiration, but was subsequently diagnosed with ALCAPA after cardiomegaly, elevated cardiac biomarkers, ischemic electrocardiographic changes, and severe left ventricular dysfunction prompted echocardiographic evaluation. This case emphasizes the need to consider congenital coronary anomalies in infants with unexplained respiratory distress, failure to thrive, or cardiomegaly, even when the initial presentation appears respiratory or gastrointestinal.

Case Presentation

A 3-month-old female infant, born at term by spontaneous vaginal delivery, presented to the emergency department with acute shortness of breath immediately after vomiting and suspected aspiration of feeds. There was no reported fever, cyanosis, recurrent vomiting, or known congenital heart disease. Feeding history was notable for suboptimal weight gain, with a current weight of 3.5 kg, below the 3rd centile for age. There was no documented family history of congenital heart disease, cardiomyopathy, or sudden unexplained infant death.

On arrival, the infant appeared in severe respiratory distress with tachypnea, subcostal retractions, and head nodding. Oxygen desaturation was noted on room air. Cardiovascular examination revealed tachycardia with good peripheral perfusion and no audible murmur. Breath sounds were clear bilaterally. Initial chest radiography demonstrated marked cardiomegaly, with a cardiothoracic ratio of approximately 67%, without clear radiographic evidence of pulmonary congestion (Figure 1). A point-of-care venous blood gas showed compensated hypercapnic respiratory failure without metabolic acidosis. Initial laboratory investigations showed a normal leukocyte count, microcytic hypochromic anemia, normal platelet count, normal serum electrolytes, and normal renal and liver function tests. Serum lactate was mildly elevated at 2.43 mmol/L.

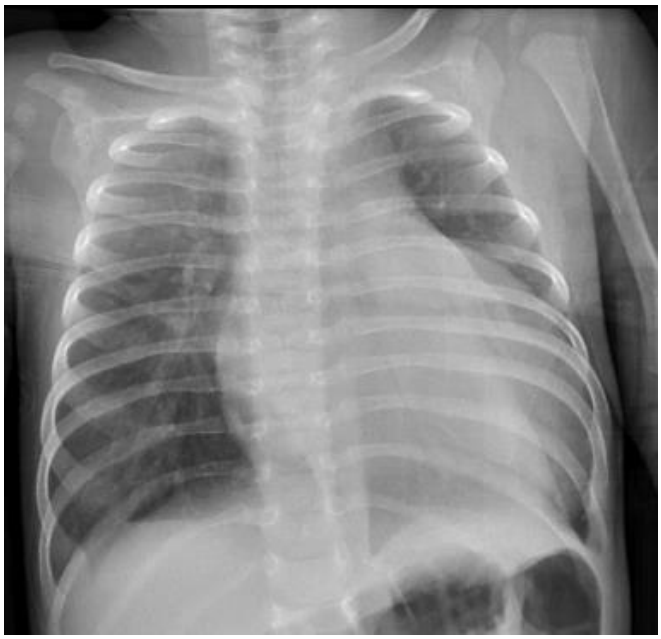


Figure 1: Chest radiograph at presentation showing marked cardiomegaly with a cardiothoracic ratio of approximately 67%, without clear pulmonary congestion.

The infant was admitted to the pediatric intensive care unit for non-invasive mechanical ventilation, empirical intravenous antibiotics, and supportive care for presumed aspiration-related respiratory distress. Sedation with dexmedetomidine and intermittent ketamine boluses was required because of agitation and ventilatory intolerance. Blood cultures remained negative. Because of the marked cardiomegaly on chest radiography, cardiac biomarkers and electrocardiography were obtained. Troponin I was markedly elevated at 80 ng/mL, and creatine kinase-MB was 28.9 U/L. A 12-lead electrocardiogram showed sinus rhythm with normal intervals and ischemic changes, including pathological Q waves and lateral ST-segment depression with T-wave inversion in leads V4–V6 (Figure 2). These findings raised concern for myocardial ischemia rather than isolated respiratory disease.

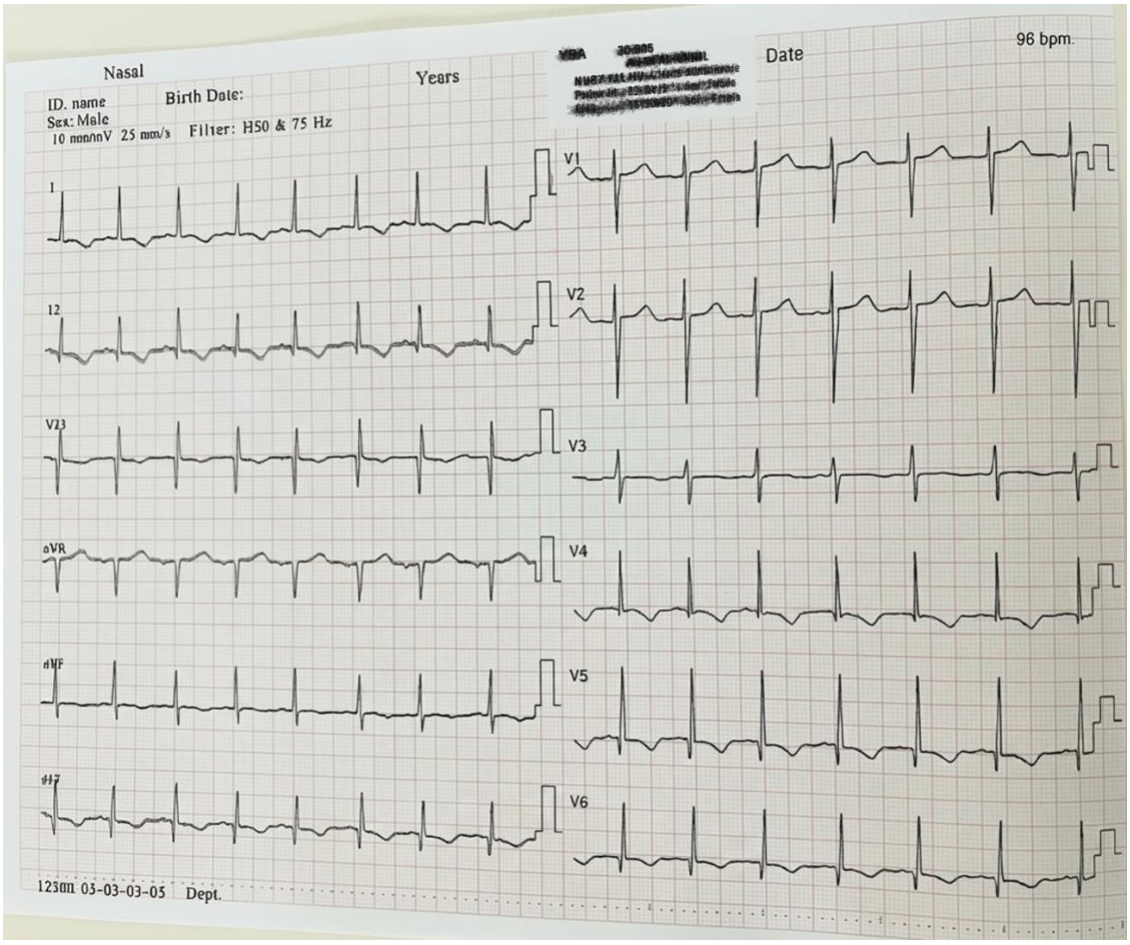
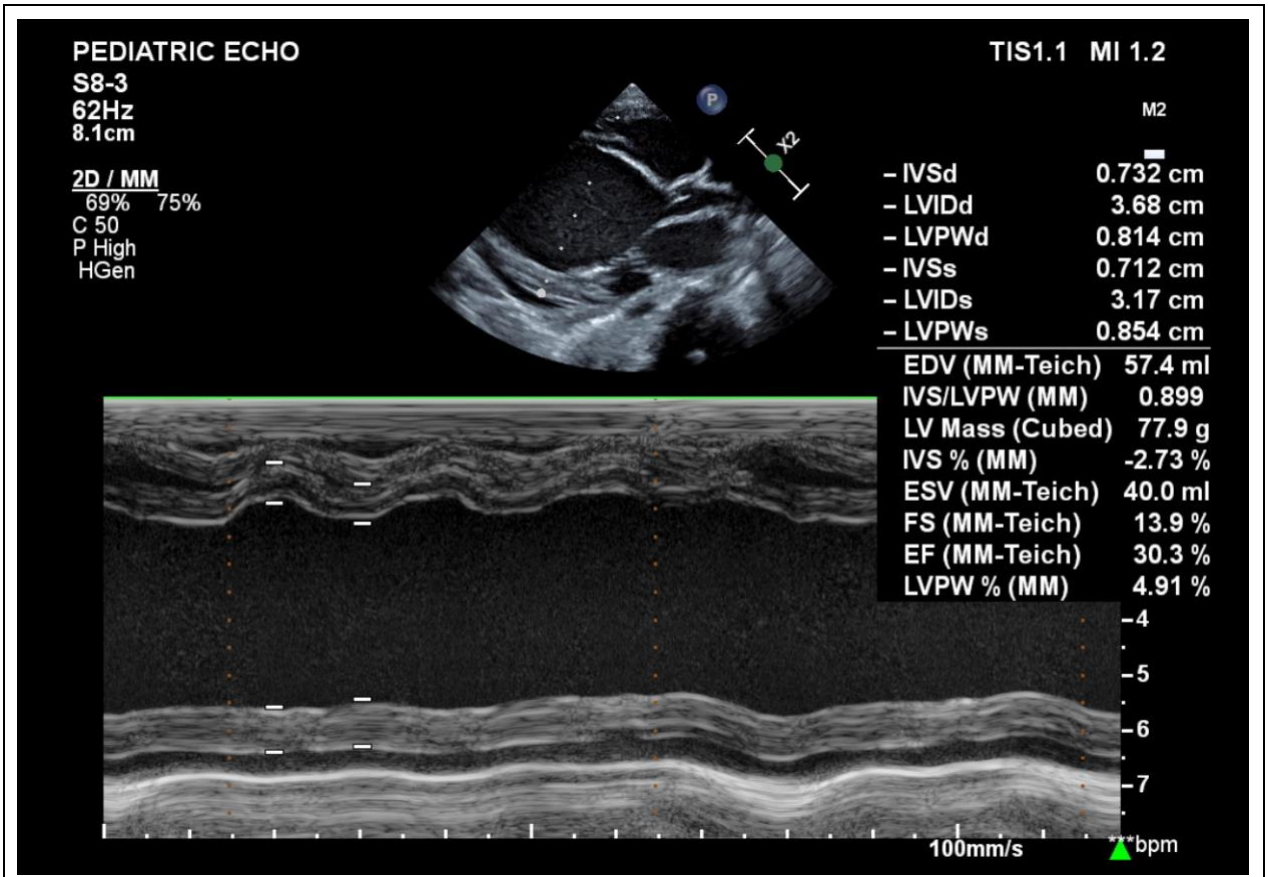


Figure 2: Twelve-lead electrocardiogram showing sinus rhythm with ischemic changes, including pathological Q waves and lateral ST-segment depression with T-wave inversion in the left precordial leads.

Transthoracic echocardiography performed on 17 August 2025 demonstrated a severely dilated left ventricle with reduced systolic function and an estimated ejection fraction of approximately 30% by M-mode (Figure 3). The left main coronary artery was visualized arising from the main pulmonary artery, with retrograde flow seen in the left anterior descending coronary artery. Additional findings included mild mitral regurgitation, patent foramen ovale with left-to-right shunt, and mild pulmonary hypertension with an estimated pulmonary artery pressure of 41 mmHg. Based on these echocardiographic findings, the diagnosis of anomalous origin of the left coronary artery from the pulmonary artery was established.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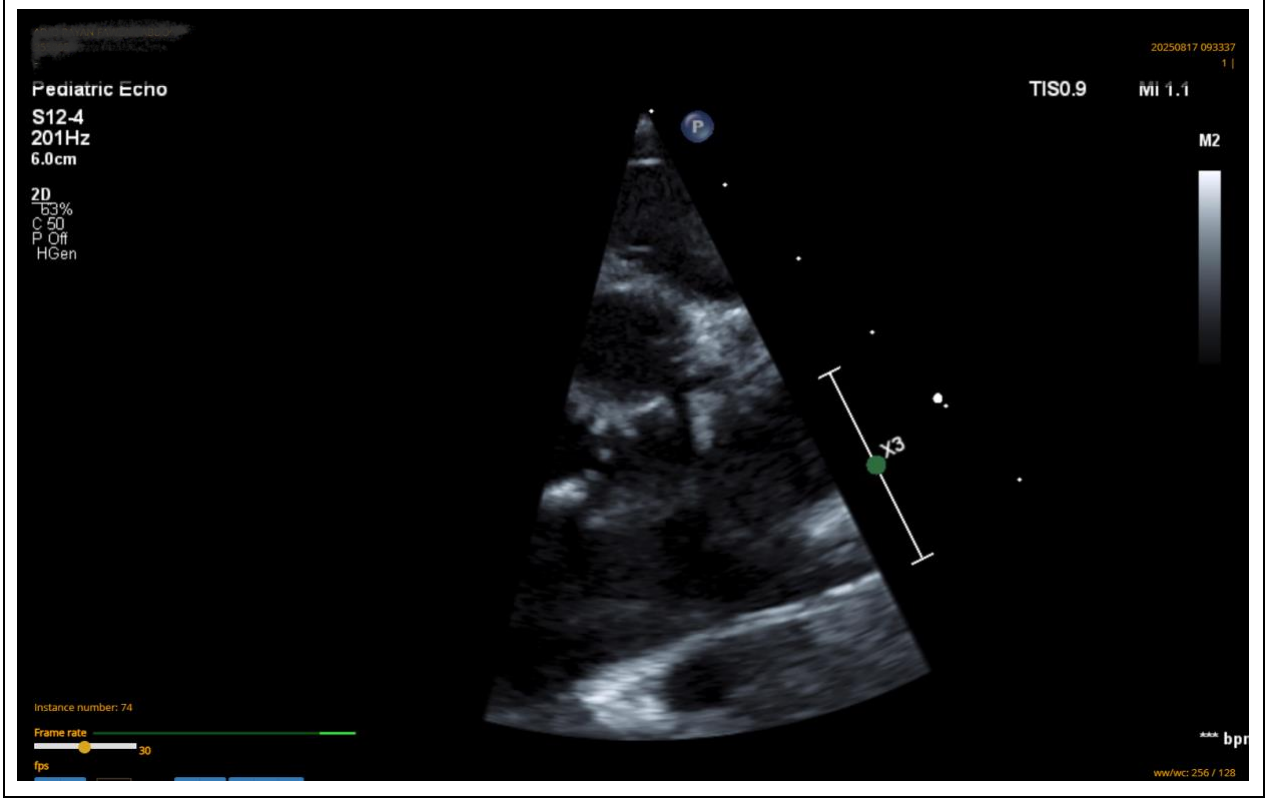
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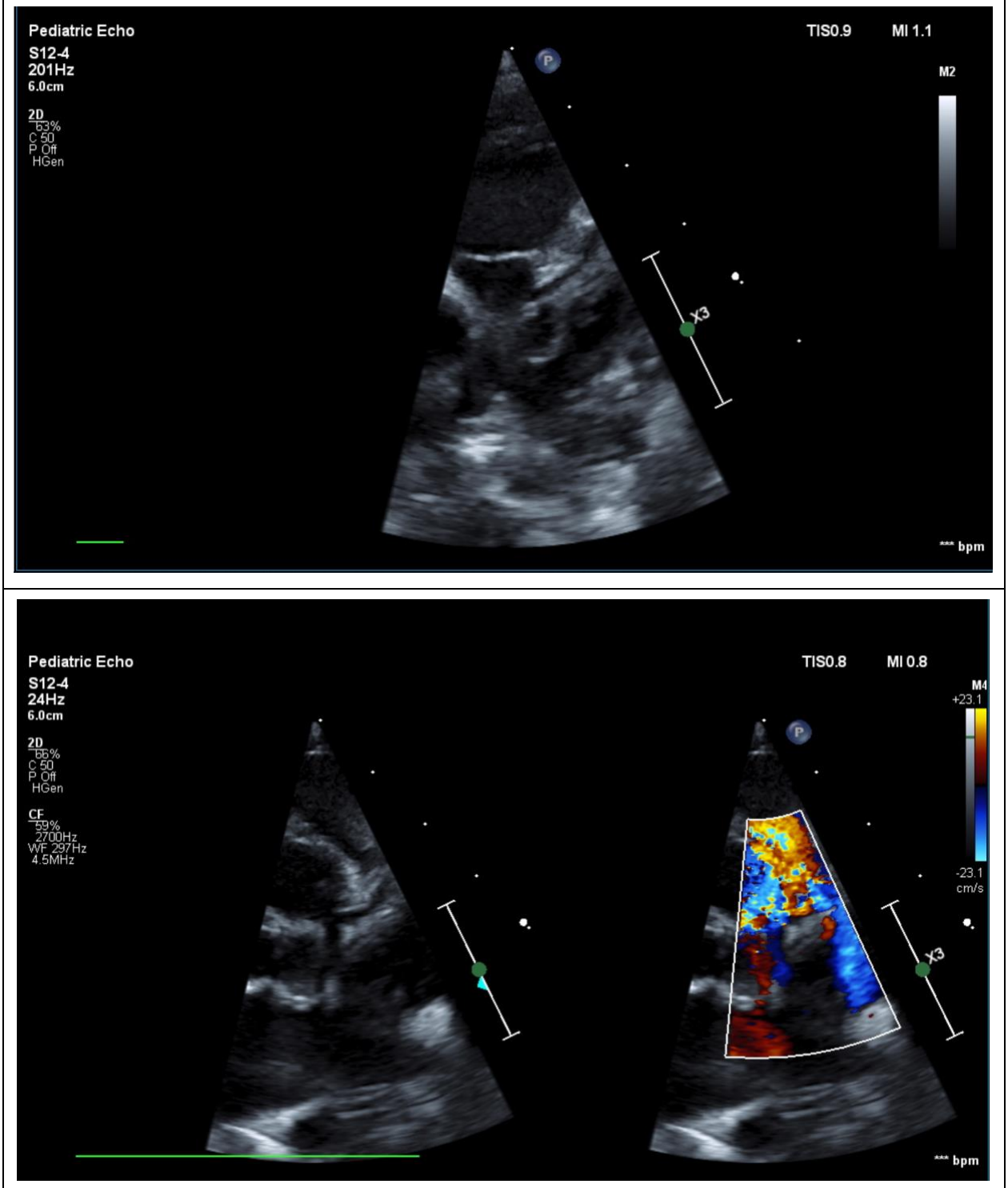

Figure 3: Transthoracic echocardiography with color Doppler showing anomalous origin of the left main coronary artery from the main pulmonary artery, retrograde flow in the left anterior descending coronary artery, severe left ventricular dilation, and reduced systolic function.

Medical treatment for heart failure was initiated, including furosemide 1 mg/kg orally three times daily, spironolactone 4 mg orally once daily, captopril 1 mg orally three times daily, and aspirin 12 mg orally once daily. Nutritional support was provided through high-calorie feeding, breastfeeding support, and anti-reflux formula as needed. The infant gradually improved clinically, was weaned from non-invasive ventilation to nasal cannula oxygen and then to room air, and was transferred from the pediatric intensive care unit to the pediatric ward on day 4 of admission. On day 6 of admission, she was accepted and transferred to Prince Sultan Cardiac Center in Al Ahsa for definitive surgical correction by coronary artery reimplantation.

Timeline

Time Point	Clinical Event
Birth	Term female infant delivered by spontaneous vaginal delivery. No known congenital heart disease was documented.
Before admission	Suboptimal weight gain; current weight 3.5 kg, below the 3rd centile for age.
Day 0: Emergency department presentation	Acute respiratory distress immediately after vomiting and suspected aspiration of feeds. Examination showed severe respiratory distress and oxygen desaturation. Chest radiography showed marked cardiomegaly with cardiothoracic ratio approximately 67%.
Day 0: Initial PICU admission	Non-invasive mechanical ventilation, empirical antibiotics, and supportive care were started.
Day 0–1: Cardiac evaluation	Troponin I was 80 ng/mL and CK-MB was 28.9 U/L. ECG showed ischemic changes with pathological Q waves and lateral ST-segment depression with T-wave inversion.
17-Aug-25	Echocardiography showed severely dilated left ventricle, ejection fraction approximately 30%, anomalous left main coronary artery arising from the main pulmonary artery, retrograde flow in the left anterior descending artery, mild mitral regurgitation, patent foramen ovale, and mild pulmonary hypertension.
After diagnosis	Heart-failure therapy, aspirin, respiratory support, and nutritional optimization were initiated.
Day 4	Clinical improvement allowed transfer from PICU to the pediatric ward.
Day 6	The infant was transferred to Prince Sultan Cardiac Center, Al Ahsa, for definitive surgical correction.

Diagnostic Assessment

The initial working diagnosis was aspiration-related respiratory distress because the acute symptoms occurred immediately after vomiting and feeding. However, several findings were not fully explained by aspiration alone, particularly marked cardiomegaly on chest radiography, failure to thrive, elevated cardiac biomarkers, and ischemic electrocardiographic changes. These findings prompted urgent echocardiographic assessment.

The main differential diagnoses considered included aspiration pneumonia, viral bronchiolitis, myocarditis, dilated cardiomyopathy, congenital structural heart disease, and congenital coronary artery anomaly. The absence of fever, normal leukocyte count, negative blood cultures, clear breath sounds, absence of radiographic pulmonary infiltrates, and the presence of severe left ventricular dilation with coronary origin abnormality made isolated respiratory infection or aspiration less likely as the primary diagnosis. The echocardiographic demonstration of the left main coronary artery arising from the main pulmonary artery with retrograde flow in the left anterior descending coronary artery established the diagnosis of ALCAPA.

Post-operative Events

Following ALCAPA repair on 28 August 2025, after preoperative ECMO support initiated on 26 August 2025 with successful decannulation on 28 August 2025 and delayed sternal closure on 30 August 2025, the patient experienced a markedly stormy postoperative course. The clinical course was complicated by hypoxic–ischemic encephalopathy, most likely secondary to an embolic event from a migrated intracardiac thrombus, presenting with convulsions that were subsequently controlled with antiepileptic medications. Neuroimaging demonstrated significant ischemic and hemorrhagic brain injury: CT brain on 6 October 2025 showed hypoxic–ischemic changes involving the basal ganglia and thalami, with pineal region hemorrhage but without hydrocephalus or mass effect, while subsequent CT on 11 October 2025 reported cortical laminar necrosis. MRI brain on 13 October 2025 revealed generalized cerebral atrophy, an old left frontoparietal ischemic insult, and scattered foci of blooming artifact suggestive of small hemorrhages; MRA was normal, and no definite evidence of cerebral venous sinus thrombosis was identified within the limitations of MRV. The patient also had a difficult respiratory course, including three failed extubation attempts in PSCC. CT chest on 6 October 2025 demonstrated posterior segmental collapse of the left lower lobe with focal narrowing of the left main bronchus, in addition to features suggestive of bilateral small airway reactive disease.

Cardiac follow-up showed persistent severe myocardial dysfunction and thrombotic complications. Echocardiography on 30 August 2025 demonstrated a large left ventricular thrombus, severe mitral regurgitation, and reduced left ventricular ejection fraction of 33%. A repeat echocardiogram on 8 October 2025, following the complicated postoperative course, showed a severely dilated left ventricle with depressed systolic function, moderate mitral regurgitation, and two small echogenic masses attached to the mitral valve chordae. The intracardiac thrombus showed interval improvement while the patient was treated with enoxaparin. The postoperative period was further complicated by a brief cardiopulmonary arrest requiring four minutes of CPR on 8 October 2025, resolved right-sided hyphema requiring ophthalmology follow-up, and resolved infectious complications including fungal infection and Serratia marcescens. The patient was referred back to MCH on 7 October 2025 and subsequently deteriorated, with mortality recorded on 15 October 2025 after prolonged resuscitation lasting 36 minutes.

Discussion

ALCAPA is an uncommon but critical diagnosis in infants presenting with unexplained respiratory distress, cardiomegaly, failure to thrive, or dilated cardiomyopathy. The present case is clinically important because the initial presentation was temporally associated with vomiting and suspected aspiration, which could reasonably direct clinicians toward a respiratory or gastrointestinal diagnosis. However, the presence of marked cardiomegaly, poor weight gain, elevated cardiac biomarkers, and ischemic electrocardiographic changes indicated an underlying cardiac process and led to the diagnosis of ALCAPA by echocardiography.

This diagnostic pathway is consistent with contemporary pediatric ALCAPA literature, where affected children may initially be evaluated for nonspecific respiratory distress, cardiomyopathy, myocarditis-like illness, or heart failure before the coronary anomaly is recognized [9,10].

The diagnostic challenge in infantile ALCAPA is related to the nonspecific nature of early symptoms. Infants may present with irritability, feeding difficulty, diaphoresis, tachypnea, pallor, poor weight gain, or recurrent episodes that resemble colic, reflux, bronchiolitis, pneumonia, or aspiration. These symptoms reflect myocardial ischemia and left ventricular dysfunction that become clinically apparent after pulmonary vascular resistance falls during early infancy. In this setting, respiratory distress may be the visible manifestation of heart failure rather than primary lung disease. Therefore, cardiomegaly in an infant with respiratory symptoms should not be attributed to respiratory disease without further cardiac evaluation. Recent multicenter and cohort data emphasize that ALCAPA may present across a broad clinical spectrum, but left ventricular dysfunction, mitral regurgitation, cardiomegaly, and heart failure remain central diagnostic clues, particularly in symptomatic infants and young children [9-11].

The pathophysiology of ALCAPA explains the progressive nature of presentation. During fetal life and the early neonatal period, pulmonary arterial pressure and oxygen saturation may be sufficient to maintain some degree of antegrade perfusion through the anomalous left coronary artery. As pulmonary vascular resistance decreases after birth, perfusion pressure and oxygen delivery through the left coronary artery fall. Collateral vessels may develop from the right coronary artery, but blood can flow retrogradely through the left coronary system into the low-pressure pulmonary artery, producing a coronary steal phenomenon. The result is chronic left ventricular ischemia, myocardial dysfunction, papillary muscle ischemia, mitral regurgitation, and eventually congestive heart failure. The severity of clinical presentation is influenced by the degree of collateral circulation, baseline ventricular function, and the extent of ischemic myocardial injury at the time of diagnosis [10,11].

Electrocardiography remains an important screening tool. Classic ECG features of infantile ALCAPA resemble myocardial ischemia or infarction and may include pathological Q waves, ST-segment abnormalities, and T-wave inversion. Published pediatric cohorts have shown that ECG abnormalities are common in patients with impaired ventricular function, although ECG findings should not be considered sufficiently sensitive to exclude ALCAPA when clinical suspicion is high [9,12]. In the present case, the ECG demonstrated ischemic changes with pathological Q waves and lateral ST-segment depression with T-wave inversion in V4–V6, which supported the need for urgent echocardiography. Literature also supports suspicion of ALCAPA in infants with heart failure symptoms, abnormal ECG findings, reduced left ventricular systolic function, or mitral regurgitation [9,10,12].

Cardiac biomarkers may contribute to clinical suspicion but should not be considered definitive. Troponin elevation in ALCAPA may reflect ongoing myocardial injury, but the timing and chronicity of ischemia can make biomarker interpretation variable. In this infant, the markedly elevated troponin I and CK-MB helped shift the diagnostic focus from isolated aspiration-related respiratory distress toward myocardial ischemia. Biomarker elevation should therefore be interpreted as a supportive finding within the broader clinical context, particularly when accompanied by cardiomegaly, ECG abnormalities, and echocardiographic evidence of left ventricular dysfunction [11,12].

Transthoracic echocardiography with color Doppler is the key diagnostic test. Characteristic findings include failure to identify the left coronary artery from the left coronary sinus, visualization of the left coronary artery arising from the pulmonary artery, retrograde flow from the left coronary system into the pulmonary artery, right coronary artery dilation, left ventricular dilation and systolic dysfunction, papillary muscle echogenicity, and mitral regurgitation. In this patient, echocardiography directly demonstrated the anomalous origin of the left main coronary artery from the main pulmonary artery, retrograde flow in the left anterior descending coronary artery, severe left ventricular dilation, reduced ejection fraction, and mild mitral regurgitation. Computed tomography angiography or cardiac catheterization may be useful when echocardiography is technically limited or when more detailed preoperative anatomical mapping is required, particularly in patients with atypical anatomy or when surgical planning requires precise definition of coronary origin and course [11,13].

Initial treatment focuses on stabilization of heart failure, optimization of respiratory status, and nutritional support while urgent referral for definitive surgical repair is arranged. Medical therapy may include diuretics, afterload reduction, aldosterone antagonists, antiplatelet therapy, and careful feeding support. However, medical therapy is only a bridge to definitive correction. Surgical creation of a two-coronary-artery system, most commonly by direct reimplantation of the anomalous left coronary artery into the aorta when feasible, is the treatment of choice. Contemporary surgical series have demonstrated that repair of ALCAPA can achieve favorable survival, recovery of left ventricular function, and improvement in mitral regurgitation, although outcomes depend on preoperative ventricular function, age at diagnosis, associated lesions, and perioperative severity [12,13-15].

The main learning point from this case is that feeding-related respiratory distress should not automatically be attributed to aspiration, reflux, or pneumonia when cardiomegaly or failure to thrive is present. In such infants, early ECG and echocardiography can identify rare but life-threatening cardiac conditions. The threshold for echocardiographic evaluation should be low in infants with unexplained cardiomegaly, poor growth, elevated cardiac enzymes, ischemic ECG changes, or recurrent respiratory distress. Earlier recognition is clinically important because surgical repair before irreversible myocardial injury offers the best chance for ventricular recovery and favorable long-term outcome [14,15-16].

Limitations

This report has several limitations. First, postoperative surgical details and long-term follow-up were not available at the time of manuscript preparation because the patient was transferred to a specialized cardiac center. Second, some clinical parameters, including complete serial vital signs and serial echocardiographic measurements, were not available for inclusion. Third, the diagnosis was established by echocardiography, and additional anatomical confirmation by computed tomography angiography or cardiac catheterization was not documented before transfer. Despite these limitations, the case provides an important diagnostic lesson regarding ALCAPA presenting as apparent respiratory disease in early infancy.

Conclusion

ALCAPA should be considered in infants presenting with unexplained respiratory distress, feeding-related symptoms, failure to thrive, cardiomegaly, elevated cardiac biomarkers, or ischemic electrocardiographic changes. This case demonstrates how ALCAPA can initially mimic aspiration or gastrointestinal disease, delaying cardiac suspicion. Chest radiography and ECG are useful early warning tools, but echocardiography with careful assessment of coronary artery origins is essential for diagnosis. Prompt stabilization and urgent referral for surgical correction are critical to improving survival and ventricular recovery.

Learning Points

1. ALCAPA may present in early infancy as respiratory distress, feeding intolerance, poor weight gain, or apparent aspiration rather than obvious cardiac disease.
2. Cardiomegaly in an infant with respiratory symptoms should trigger cardiac evaluation, especially when accompanied by failure to thrive or elevated cardiac biomarkers.
3. ECG findings suggestive of myocardial ischemia in infants, including pathological Q waves, ST-segment changes, or T-wave inversion, should prompt urgent echocardiography.
4. Echocardiography with color Doppler assessment of coronary artery origins is essential for diagnosis.
5. Medical stabilization is a bridge to definitive surgical repair, most commonly coronary reimplantation to establish a two-coronary-artery system.

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