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The "Well-Appearing Trap": Cardiac Point-of-Care Ultrasound (POCUS) Prompting the Diagnosis of Anomalous Left Coronary Artery From the Pulmonary Artery (ALCAPA)

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Abstract

Anomalous left coronary artery from the pulmonary artery (ALCAPA) is a rare congenital coronary anomaly associated with high mortality when left untreated. A key diagnostic pitfall of ALCAPA is that infants may appear relatively well despite profound underlying myocardial dysfunction, creating a "well-appearing trap." To address this risk, bedside point-of-care ultrasound (POCUS) can be employed to identify occult ventricular dilation and dysfunction, prompting definitive cardiac evaluation. We describe two infants with ALCAPA who presented to our emergency department: a nine-month-old boy with a 12-hour history of decreased urine output and a two-month-old girl with a one-day history of inconsolable crying. In both patients, cardiac POCUS revealed marked left ventricular dilation and severely reduced systolic function that were not apparent from their initial clinical appearance, prompting urgent definitive cardiac imaging. These cases highlight a central diagnostic challenge: infants may appear relatively stable while harbouring life-threatening myocardial dysfunction. Cardiac POCUS provides a rapid bedside means of recognizing this clinical-structural mismatch, identifying occult ventricular dysfunction, and accelerating definitive cardiac assessment.

Categories: Emergency Medicine, Pediatrics
Keywords: anomalous origin of the left coronary artery from pulmonary artery (alcapa), cardiac pocus, inconsolable cry, pediatric emergency & acute care, pediatric emergency department (ped), well-appearing trap

Introduction

Anomalous left coronary artery from the pulmonary artery (ALCAPA), also known as Bland-White-Garland syndrome, is a rare congenital coronary anomaly in which the left coronary artery arises from the pulmonary artery rather than the aortic root. Its estimated prevalence is 1 in 300,000 live births (0.00033%) [1], though its true incidence may be higher than historically recognized. A five-year study from midwestern Ireland reported an incidence of 1 in 4,243 live births (0.023%) [2], while a prospective study from a cardiology center in Poland identified ALCAPA in 0.021% of 62,320 children who underwent routine echocardiography [3]. Without surgical correction, infantile ALCAPA is associated with a first-year mortality rate approaching 90% [4], making early recognition essential.

The pathophysiology of infantile ALCAPA is closely linked to the postnatal decline in pulmonary vascular resistance and pulmonary arterial pressure, which reduces perfusion pressure within the anomalous left coronary artery. Pulmonary vascular resistance decreases rapidly after birth and continues to decline during the subsequent weeks [5]. Infants with ALCAPA are therefore typically asymptomatic at birth, when pulmonary arterial pressure remains high enough to drive forward perfusion [6]. As this pressure decreases, retrograde flow from the left coronary artery into the pulmonary artery may develop, producing a coronary steal phenomenon [6]. This results in progressive myocardial ischemia and left ventricular dysfunction. In one series, the median age at the first clinical manifestation attributable to ALCAPA was two months, whereas the median age at diagnosis was five months [7].

As a compensatory mechanism, intercoronary collateral vessels from the right coronary artery may develop and provide perfusion to the left coronary territory. The timing and severity of symptoms are influenced by the extent of this collateral network. More extensive collateral circulation may permit survival into adulthood, but inadequate collateralization generally leads to presentation during infancy.

Despite severe underlying cardiac dysfunction, infants with ALCAPA often present with subtle, nonspecific clinical features. Numerous case series and reports have described the associated diagnostic challenges, particularly when ALCAPA is mistaken for respiratory disease or myocarditis. Notably, Levitas et al. reported failure to thrive in 78% of patients, while 55.5% had previously been diagnosed with asthma or wheezing, and 44% were initially suspected of having myocarditis [7]. Other reported manifestations include feeding-

associated distress, diaphoresis, tachypnea, vomiting, and respiratory distress [8]. This "well-appearing trap" may divert attention from a cardiac etiology, delaying diagnosis and life-saving surgical intervention.

Point-of-care ultrasound (POCUS) is increasingly used in pediatric emergency medicine to answer focused clinical questions and support time-sensitive decision-making [9]. Cardiac POCUS can help assess cardiac function and fluid status, as well as identify causes of shock. Although focused cardiac POCUS is not intended to map coronary anatomy, it readily identifies gross ventricular dysfunction, chamber dilation, or pericardial effusion requiring urgent comprehensive echocardiography. Illustrating its potential role in recognizing ALCAPA, Shaahinfar et al. described an infant in whom POCUS demonstrated dilated cardiomyopathy and a regional wall-motion abnormality [10].

We describe two infants whose nonspecific complaints and relatively reassuring initial appearances concealed severe myocardial dysfunction. In both cases, subtle respiratory findings prompted cardiac POCUS, which revealed marked ventricular dilation and dysfunction, facilitating prompt definitive imaging of ALCAPA and early cardiology consultation. These cases aim to raise awareness of this rare but potentially life-threatening condition and highlight the role of POCUS as an adjunct in clinical assessment.

Case Presentation

Case 1

A nine-month-old boy presented to the emergency department (ED) after his parents noted no urine output for approximately 12 hours; no other associated symptoms were reported on review of systems. He was born at 38 weeks' gestation following an uncomplicated pregnancy. His past medical history was notable for failure to thrive, frequent arching, and fussiness during feeding. He had been treated with lansoprazole with minimal improvement. His weight had fallen from the 75th percentile at birth to below the third percentile at presentation. He was fed both breast milk and formula and had begun complementary solid foods at six months of age. Family history was positive for the sudden death of his maternal grandfather at 46 years of age of unclear etiology. There was otherwise no known family history of cardiac disease.

On initial assessment in the ED, the patient had subcostal retractions and nasal flaring; his lungs had good breath sounds with no crackles or wheezing. His parents reported that the increased work of breathing had been his baseline breathing effort and that they had come to regard it as normal for him. His vital signs were normal (Table *1*). Cardiac, abdominal, and other systems examinations were otherwise unremarkable, with no audible murmur, hepatomegaly, or peripheral edema.

Vital parameters	Readings	Normal range
Heart rate	148 beats/minute	100–180 beats/minute
Systolic blood pressure	92 mmHg	80–100 mmHg
Respiratory rate	44 breaths/minute	30–53 breaths/minute
SpO_2	97%	>95%

TABLE 1: Case 1: Vital signs.

All vital signs are within the normal range.

Renal and bladder POCUS was performed using a Mindray ME8 ultrasound system (Mindray, Shenzhen, China) equipped with a low-frequency curvilinear transducer of 2 to 5 MHz to evaluate the reported absence of urine output; the bladder was empty, providing no evidence of urinary retention. Because the degree of respiratory effort was not explained by the initial urinary presentation, the examination was extended to focused cardiac POCUS using the same Mindray ME8 system and a low-frequency phased-array transducer (1-5 MHz). Marked left ventricular dilation, severely reduced global left ventricular systolic function, and poor mitral leaflet coaptation were observed (Figure *1*, Video *1*). Minimal pericardial effusion was identified, and the inferior vena cava collapsed with respiration (Video *2*).

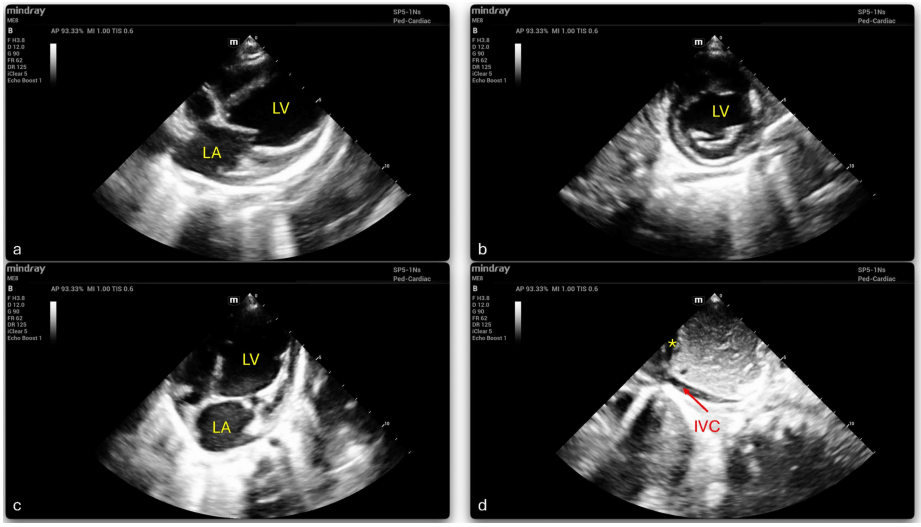


FIGURE 1: Case 1: Cardiac POCUS.

Dilated LV and LA in parasternal long-axis (a) and apical four-chamber view (c). Short-axis view (b) shows a dilated LV. Small pericardial effusion (*) in the subxiphoid IVC view (d).

POCUS: point-of-care ultrasound; LV: left ventricle, LA: left atrium, IVC: inferior vena cava



VIDEO 1: Case 1: Cardiac POCUS.

Apical four-chamber view shows dilated left ventricle and left atrium, along with poor mitral leaflet coaptation.

POCUS: point-of-care ultrasound

View video here: https://youtu.be/BAHtatZ5PFk



VIDEO 2: Case 1: Cardiac POCUS.

Subxiphoid view shows the collapsed inferior vena cava with respiration, along with minimal pericardial effusion.

POCUS: point-of-care ultrasound

View video here: https://youtu.be/AOeHru0fGHs

Chest radiography (CXR) showed cardiomegaly with diffuse bilateral interstitial and perihilar opacities

suggestive of pulmonary edema, along with a trace right pleural effusion (Figure 2). An electrocardiogram (ECG) demonstrated a sinus rhythm with deep Q-waves, left-axis deviation, T-wave inversions, and ST-segment abnormalities in leads I, aVL, and the lateral precordial leads (Figure 3). Laboratory investigations showed a markedly elevated N-terminal pro-brain natriuretic peptide (NT-proBNP) level, but troponin I remained within the normal range (Table 2), and the remaining laboratory results were unremarkable.

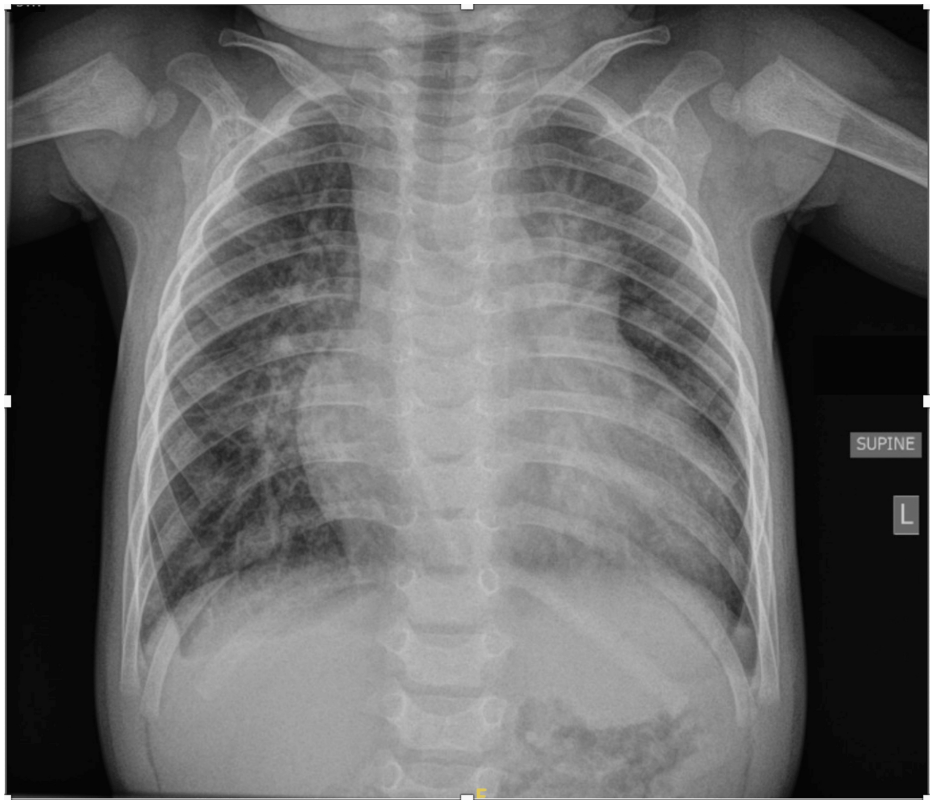


FIGURE 2: Case 1: Chest X-ray.

Chest X-ray showing cardiomegaly and pulmonary edema.

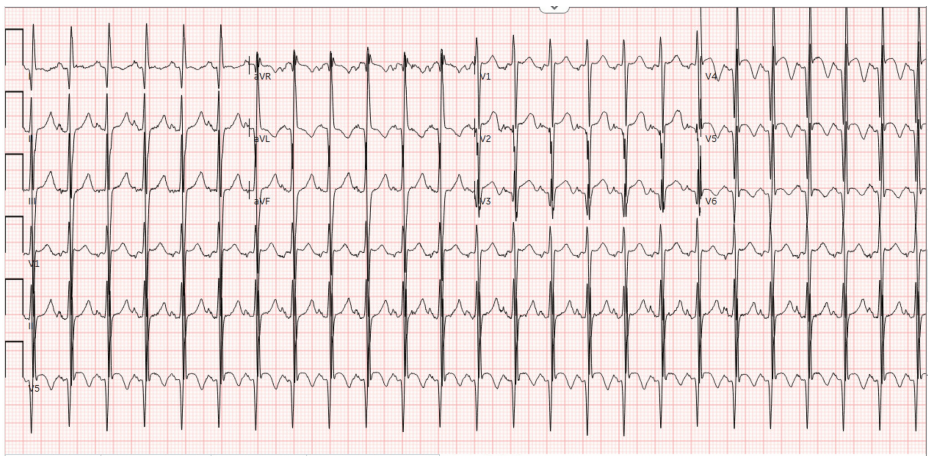


FIGURE 3: Case 1: Electrocardiogram.

Electrocardiogram showing normal sinus rhythm, left axis deviation, deep Q-wave with ST-T changes in I, aVL, and lateral precordial leads.

Cardiac biomarkers	Result (ng/L)	Normal range (ng/L)
Troponin I	22	≤97.7
NT-proBNP	7,994	42.4–1,475.3

TABLE 2: Case 1: Cardiac biomarkers.

NT-proBNP: N-terminal pro-B-type natriuretic peptide

Cardiology was urgently consulted in the ED. Formal echocardiography confirmed severe left ventricular dilation with severely reduced systolic function and severe mitral regurgitation. Subsequent contrast-enhanced ECG-gated cardiac CT demonstrated ALCAPA. The left coronary artery originated from the undersurface of the right pulmonary artery and coursed close to the aorta before bifurcating into the left anterior descending and circumflex arteries. The right coronary artery was enlarged, arising from the ascending aorta above the left aortic sinus, with extensive collateral circulation supplying the left coronary system. Marked dilation of the left ventricle and left atrium with associated left ventricular hypertrophy was noted.

The patient was admitted to the cardiac critical care unit and started on milrinone infusion at 0.5 µg/kg/minute along with intravenous furosemide. He subsequently underwent surgical repair consisting of left coronary artery reimplantation, right pulmonary artery plasty, and mitral valve repair. Due to persistent severe left ventricular dysfunction and substantial inotropic requirements post-bypass, his sternum was left open. The postoperative echocardiogram confirmed a successful mitral valve repair with only mild residual mitral regurgitation.

Two days later, he underwent delayed sternal closure and was successfully extubated to noninvasive positive-pressure ventilation. His respiratory and hemodynamic status improved gradually, allowing noninvasive respiratory support to be weaned. Heart failure therapy was then initiated, including enalapril, carvedilol, and spironolactone. Aspirin was also started considering his coronary reimplantation and severely reduced ventricular function (ejection fraction <30%).

Subsequent follow-up demonstrated continued clinical and echocardiographic improvement. NT-proBNP levels gradually declined, and repeat echocardiography showed an increase in left ventricular ejection fraction from 15% initially to 58% on follow-up, with only mild residual mitral regurgitation. His heart failure medications were gradually tapered over time.

Case 2

A two-month-old girl was brought to the ED after 24 hours of inconsolable crying and six hours of reduced oral intake. Her parents also reported a mild cough over the preceding week without associated fever. No additional symptoms were reported during the systemic review. She was born at 38 weeks' gestation following an uncomplicated pregnancy and delivery, with no significant medical history or known family history of cardiac disease.

On initial assessment, she was active and did not appear acutely distressed, with a capillary refill of less than two seconds. Vital signs were normal except for a slightly raised respiratory rate (Table *3*). Her respiratory assessment was reassuring with no audible respiratory noise, no chest recession, and she had good bilateral air entry with no added sounds on auscultation. Her anterior fontanelle appeared sunken, but the remainder of the cardiac and abdominal examinations were unremarkable.

Vital parameters	Readings	Normal range
Heart rate	154 beats/minute	100–180 beats/minute
Systolic blood pressure	86 mmHg	80–100 mmHg
Respiratory rate	60 breaths/minute	30–53 breaths/minute
SpO_2	98%	>95%

TABLE 3: Case 2: Vital signs.

All parameters are within the normal limits except for increased respiratory rate.

She was observed in the ED while oral feeding was attempted. A urinalysis performed to screen for occult urinary tract infection was negative. During ongoing observation, however, nursing staff noted intermittent grunting that had not been apparent during the initial assessment.

Although subtle, the grunting was inconsistent with an otherwise reassuring examination and prompted immediate lung and cardiac POCUS. Focused cardiac POCUS was performed using the Mindray ME8 ultrasound system with a phased-array transducer (1-5 MHz), revealing marked left ventricular and left atrial dilation, severely reduced global left ventricular systolic function, and a moderate pericardial effusion (Figure *4*; Videos *3*, *4*). Lung POCUS demonstrated B-lines. The unexpected severity of these findings prompted urgent laboratory investigations, CXR, and a cardiology consultation.

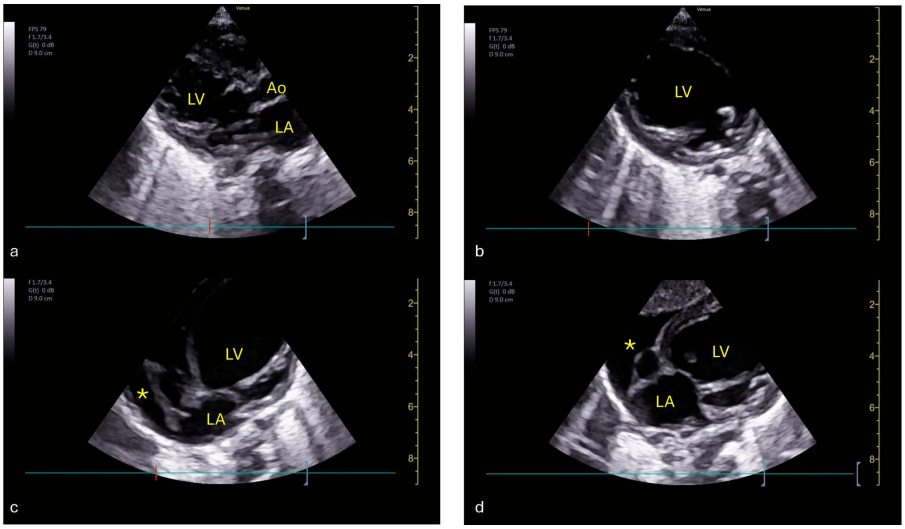
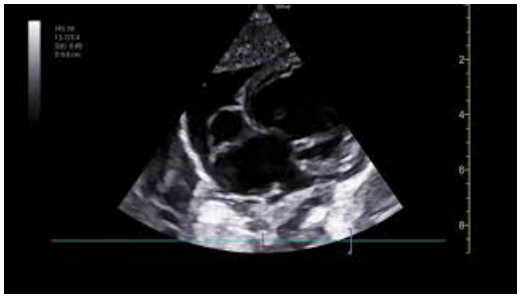


FIGURE 4: Case 2: Cardiac POCUS.

Left ventricle and atrium dilation in parasternal long-axis (a), apical four-chamber (c), and subxiphoid view (d). Dilated left ventricle in parasternal shot-axis view (b). Moderate pericardial effusion (*) in apical four-chamber (c) and subxiphoid view (d).

POCUS: point-of-care ultrasound; LV: left ventricle, LA: left atrium, Ao: aorta

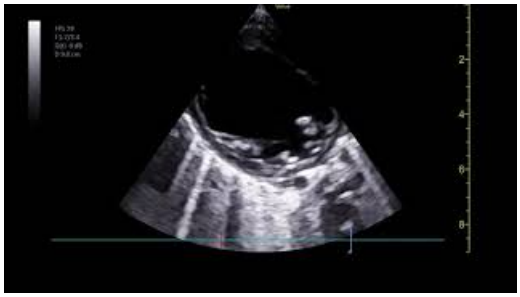


VIDEO 3: Case 2: Cardiac POCUS.

Subxiphoid view shows dilated left ventricle and left atrium, global reduced cardiac contractility, and moderate pericardial effusion.

POCUS: point-of-care ultrasound

View video here: https://youtu.be/bZbFP2N8UEY



VIDEO 4: Case 2: Cardiac POCUS

Short-axis view shows severely dilated left ventricle with poor contractility.

POCUS: point-of-care ultrasound

View video here: https://youtu.be/KbQ1Hn78nco

CXR revealed severe cardiomegaly with normal, symmetric pulmonary vascularity and no focal consolidation or pleural effusion (Figure *5*). An ECG demonstrated T-wave inversions in leads I, II, aVL, and V5-V6; ST-segment elevation in V4-V5; and Q-waves in leads I, aVL, and V5-V6 (Figure *6*). Laboratory investigations showed elevated troponin and NT-proBNP concentrations (Table *4*).



FIGURE 5: Case 2: Chest X-ray.

Chest X-ray showing severe cardiomegaly.

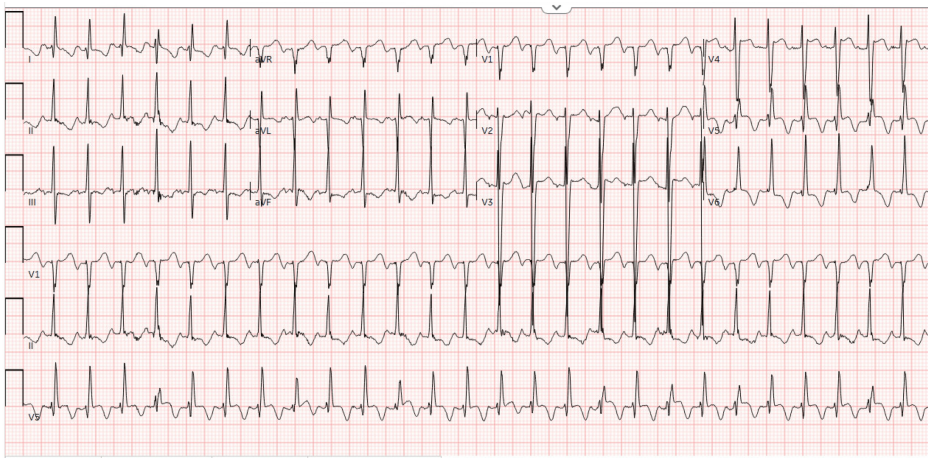


FIGURE 6: Case 2: Electrocardiogram.

Electrocardiogram showing ST abnormality and T-wave inversion in inferolateral leads, along with prolonged QT.

Cardiac biomarkers	Result (ng/L)	Normal range (ng/L)
Troponin I	3,399.5	≤97.7
NT-proBNP	>35,000	42.4–1,475.3

TABLE 4: Case 2: Cardiac biomarkers.

NT-proBNP: N-terminal pro-B-type natriuretic peptide

Cardiology was consulted, and formal echocardiography confirmed that the left coronary artery originated anomalously from the left-facing cusp of the pulmonary artery. It also demonstrated severe left ventricular dilation, profound systolic dysfunction, severe mitral annular dilation with severe mitral regurgitation, and a moderate pericardial effusion.

The patient was admitted to the cardiac critical care unit for planned surgical repair. At the time of surgical incision, she experienced a sudden cardiac arrest, requiring approximately 10 minutes of cardiopulmonary resuscitation and the immediate initiation of postoperative venoarterial extracorporeal membrane oxygenation support. Five days postoperatively, she developed pulseless ventricular tachycardia, requiring a further 44 minutes of cardiopulmonary resuscitation. She subsequently sustained a hypoxic-ischemic brain injury, resulting in intractable seizures, spasticity, and dystonia. Her cardiac function remained persistently impaired, with no signs of recovery. Following multidisciplinary discussions and consultation with her family, care was redirected toward comfort because of her poor neurological and cardiac prognosis. She died peacefully two weeks after surgery.

Discussion

We report on two infants with ALCAPA whose initial clinical appearances masked the severity of their underlying myocardial dysfunction. These cases illustrate the diagnostic perils of the “well-appearing trap”: both infants remained active, normotensive, and adequately oxygenated despite advanced myocardial dysfunction. In each case, subtle respiratory findings suggested that the apparent clinical stability was misleading. Cardiac POCUS rapidly exposed severe ventricular dilation and dysfunction, prompting accelerated definitive cardiac assessment.

Classic presentations of infantile ALCAPA include failure to thrive, feeding difficulty, and feeding-associated irritability. However, the initial presenting complaints in our cases, particularly decreased urine output, were atypical and did not immediately suggest a cardiac diagnosis. Keith reported that many infants with ALCAPA experience irritability lasting days to weeks, particularly after feeding [1], and Levitas et al. reported failure to thrive in up to 77% of patients with ALCAPA [7]. These symptoms are common in pediatric practice and may initially be attributed to more prevalent noncardiac conditions, delaying consideration of an underlying cardiac cause.

Failure to thrive is nonspecific and has a broad differential diagnosis, meaning that, in an otherwise well-appearing infant, inadequate caloric intake or gastroesophageal reflux is often assumed to be the primary cause. A prior assessment for failure to thrive may also contribute to diagnostic anchoring, making it less likely that the underlying explanation will be reconsidered during a subsequent emergency visit. Similarly, inconsolable crying or irritability is frequently attributed to gastroesophageal reflux, meningitis, abdominal pathology, or colic. Moreover, in a clinically stable infant, decreased urine output is routinely attributed to reduced intake, dehydration, or urinary tract pathology. In Case 1, decreased urine output may have indicated reduced intake or impaired renal perfusion associated with low cardiac output; however, a causal relationship cannot be established.

Diagnostic recognition is further complicated when clinical abnormalities develop gradually, leading caregivers to regard them as normal for the infant. In these cases, the relevant findings included tachypnea, retractions, nasal flaring, and intermittent grunting. In Case 2, the intermittent grunting observed during assessment had been perceived by her caregivers as part of her normal crying. The gradual evolution of these findings may have masked the severity of the underlying illness. Infants with significant cardiac pathology may appear relatively well and lack overt hemodynamic instability despite advanced myocardial dysfunction.

The lesson here is not that every irritable or poorly feeding infant requires cardiac imaging. Rather, subtle respiratory findings that are inconsistent with the apparent clinical picture should prompt clinicians to pause and reconsider underlying cardiac disease. In this setting, cardiac POCUS offers a rapid, radiation-free, and accessible bedside assessment that can reveal cardiac dysfunction in an infant who otherwise appears relatively well.

In both of our cases, subtle signs of respiratory distress prompted focused cardiac POCUS using a Mindray ME8 ultrasound system equipped with a low-frequency phased-array transducer (1-5 MHz). Parasternal long-axis, parasternal short-axis, apical four-chamber, and subcostal four-chamber views were obtained to assess global systolic function, chamber dimensions, and the presence of pericardial effusion. In both patients, cardiac POCUS demonstrated left ventricular and left atrial dilation with reduced global left ventricular systolic function, consistent with findings previously described in an infant with ALCAPA [10]. Cardiac POCUS also demonstrated poor mitral leaflet coaptation in Case 1 and a moderate-to-large pericardial effusion in Case 2. These findings prompted urgent cardiology consultation and comprehensive cardiac imaging, which established the diagnosis of ALCAPA. In infants presenting with otherwise unexplained dilated cardiomyopathy, echocardiographic findings that raise suspicion for ALCAPA include increased papillary muscle echogenicity and mitral regurgitation [11,12]. Definitive echocardiographic features include direct visualization of the anomalous coronary origin, retrograde flow within the left coronary artery, collateral coronary flow, and right coronary artery dilation [11].

CXR and ECGs can provide additional clues but are not diagnostic. CXR commonly demonstrates cardiomegaly [1,7], while characteristic ECG features in infantile ALCAPA include deep, narrow Q-waves and T-wave inversions in leads I, aVL, and the lateral precordial leads, reflecting anterolateral myocardial ischemia [1]. These changes were present in both of our cases.

Laboratory findings may support recognition of cardiac failure. NT-proBNP elevation may indicate heart failure, while troponin elevation may indicate myocardial injury, although neither biomarker is specific to ALCAPA. One case series reported troponin elevation in four of nine patients with ALCAPA [7], a variability mirrored in our case series: troponin was normal in Case 1 but elevated in Case 2.

Taken together, these cases demonstrate that ALCAPA in infants younger than one year may present with subtle, nonspecific symptoms. Clinicians working in the ED must remain vigilant for respiratory findings that are inconsistent with the infant's otherwise reassuring appearance. In this setting, cardiac POCUS can reveal occult ventricular dysfunction, helping clinicians avoid the "well-appearing trap" and facilitate definitive, life-saving care.

Conclusions

ALCAPA should be considered in the differential diagnosis of infants presenting with feeding-associated irritability, feeding difficulty, or unexplained failure to thrive, particularly when accompanied by subtle signs of increased work of breathing. Decreased urine output may represent an unusual manifestation of impaired systemic perfusion secondary to low cardiac output, but this requires further investigation. The two cases reported herein highlight the dangers of the "well-appearing trap," wherein severe cardiac disease is overlooked because infants initially appear clinically stable. Cardiac POCUS is a useful adjunct tool that serves as a readily available, noninvasive modality for clinical assessment, revealing occult ventricular dysfunction and prompting timely definitive imaging and cardiology consultation.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

Concept and design: Lee Wan Gan, Danielle Sweetnam-Holmes, Nouf Aljahdali

Acquisition, analysis, or interpretation of data: Lee Wan Gan, Nouf Aljahdali

Drafting of the manuscript: Lee Wan Gan, Nouf Aljahdali

Critical review of the manuscript for important intellectual content: Lee Wan Gan, Danielle Sweetnam-Holmes

Supervision: Danielle Sweetnam-Holmes

Disclosures

Human subjects: Informed consent for treatment and open access publication was obtained or waived by all participants in this study. **Conflicts of interest:** In compliance with the ICMJE uniform disclosure form, all authors declare the following: **Payment/services info:** All authors have declared that no financial support was received from any organization for the submitted work. **Financial relationships:** All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. **Other relationships:** All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

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