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Cortical Laminar Necrosis Following ALCAPA Repair Complicated by Intracardiac Thrombus: A Pediatric Postoperative Educational Clinical Scenario

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Abstract

Background: Anomalous origin of the left coronary artery from the pulmonary artery (ALCAPA) is a rare but life-threatening congenital coronary anomaly that may present with myocardial ischemia, left ventricular dysfunction, mitral regurgitation, and heart failure in infancy. Surgical repair aims to restore a dual-coronary circulation and is usually associated with gradual myocardial recovery. However, patients with severe preoperative instability may develop complex postoperative complications, including persistent ventricular dysfunction, thrombus formation, neurological injury, and multisystem deterioration.

Case Presentation: We report a pediatric patient with ALCAPA managed at a tertiary cardiac center in Riyadh, Saudi Arabia. The patient required extracorporeal membrane oxygenation on 26 August 2025 because of severe cardiorespiratory compromise and subsequently underwent ALCAPA repair on 28 August 2025, with ECMO decannulation on the same day. Delayed sternal closure was performed on 30 August 2025. Early postoperative echocardiography demonstrated severe left ventricular dysfunction with an ejection fraction of 33%, severe mitral regurgitation, and a large left ventricular thrombus. The thrombus was treated with enoxaparin and showed interval improvement. However, the postoperative course remained stormy, complicated by seizures controlled with antiepileptic medications, hypoxic–ischemic encephalopathy, and suspected thromboembolic neurological injury. Brain CT on 6 October 2025 showed hypoxic–ischemic changes involving the basal ganglia and thalami, with pineal region hemorrhage. Subsequent CT on 11 October 2025 demonstrated cortical laminar necrosis. MRI on 13 October 2025 showed generalized brain atrophy, an old left frontoparietal ischemic insult, and scattered small hemorrhagic foci. Respiratory complications included three failed extubation trials, left lower lobe collapse, focal narrowing of the left main bronchus, and bilateral reactive small airway disease. The course was further complicated by resolved fungal and Serratia marcescens infections. The patient sustained cardiopulmonary arrest requiring 4 minutes of CPR on 8 October 2025 and died on 15 October 2025 after prolonged CPR lasting 36 minutes.

Conclusion: This case highlights a severe postoperative neurocardiac cascade after ALCAPA repair, linking persistent ventricular dysfunction, intracardiac thrombosis, hypoxic–ischemic brain injury, cortical laminar necrosis, respiratory failure, and mortality. It emphasizes the need for vigilant postoperative echocardiographic, neurological, respiratory, and anticoagulation surveillance in high-risk pediatric ALCAPA patients.

Keywords: ALCAPA; Cortical laminar necrosis; Intracardiac thrombus; Hypoxic–ischemic encephalopathy; Pediatric cardiac surgery; Postoperative complications; Left ventricular dysfunction; 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 but potentially fatal congenital coronary anomaly in which the left coronary artery arises from the pulmonary arterial circulation rather than from the aorta. Its estimated incidence is approximately 1 in 300,000 live births, and although uncommon, it represents one of the most clinically important surgically correctable causes of infantile myocardial ischemia, dilated cardiomyopathy, mitral regurgitation, and heart failure [1]. The pathophysiological burden of ALCAPA becomes particularly evident after the normal postnatal decline in pulmonary vascular resistance, when perfusion pressure within the anomalous left coronary artery falls, resulting in coronary steal from the left coronary system into the pulmonary artery. This produces chronic subendocardial ischemia, progressive left ventricular dysfunction, papillary muscle ischemia, and variable degrees of functional mitral regurgitation [2]. Without timely recognition and surgical correction, the natural history is poor; historical series have reported that most untreated infants die within the first year of life [1,3].

The modern therapeutic objective in ALCAPA is restoration of a dual-coronary circulation, most commonly through direct reimplantation of the anomalous left coronary artery into the aorta, although alternative procedures such as the Takeuchi intrapulmonary baffle technique may be used depending on anatomy and institutional practice [2,4]. Contemporary surgical series generally report favorable survival and recovery of ventricular function after repair, even among patients presenting with severe preoperative left ventricular dysfunction [4,5]. However, postoperative recovery may be prolonged and heterogeneous, particularly in infants and in patients with markedly depressed preoperative left ventricular ejection fraction. In a recent single-center series of 136 surgically treated patients, Yu et al. reported a mortality rate of 6.6%, with low preoperative left ventricular ejection fraction and younger age associated with delayed postoperative recovery of left ventricular function [4]. Similarly, Cao and Wang emphasized that preoperative left ventricular dysfunction remains a major determinant of in-hospital mortality after ALCAPA repair, despite generally excellent early and midterm outcomes [1]. These observations highlight that anatomical correction does not immediately eliminate the risk of postoperative myocardial stunning, low-output physiology, arrhythmia, residual mitral regurgitation, or thromboembolic complications.

Intracardiac thrombosis after pediatric cardiac surgery is uncommon but clinically significant, particularly when severe ventricular dysfunction, chamber dilation, endocardial injury, systemic inflammation, cardiopulmonary bypass, central venous instrumentation, or extracorporeal membrane oxygenation are present. In the setting of ALCAPA, profound ischemic cardiomyopathy may create a low-flow left ventricular environment that predisposes to mural thrombus formation.

Freud et al. previously reported a large left ventricular thrombus after ALCAPA repair in an infant with ischemic cardiomyopathy, underscoring the possibility of postoperative thrombosis in this population [6]. However, published descriptions of ALCAPA repair complicated by intracardiac thrombus remain limited, and cases linking postoperative thrombus to suspected cerebral embolic injury, hypoxic–ischemic encephalopathy, and cortical laminar necrosis are especially rare. This gap is clinically important because postoperative anticoagulation in infants and critically ill children is often challenging: clinicians must balance thrombus progression and embolization against bleeding risk, recent sternotomy, invasive lines, coagulopathy, infection, and possible intracranial hemorrhage.

Neurological injury remains one of the most devastating complications after congenital cardiac surgery. Mechanisms are usually multifactorial and may include perioperative hypoperfusion, embolic stroke, hypoxemia, cardiac arrest, extracorporeal circulation-related inflammation, coagulation disturbance, and postoperative hemodynamic instability [7]. Recent pediatric cardiac surgery data continue to identify seizures, ischemic injury, and hypoxic–ischemic encephalopathy as important postoperative neurological events associated with increased morbidity, prolonged hospitalization, and mortality [7,8]. Children undergoing complex cardiac surgery may be particularly vulnerable because cerebral oxygen delivery depends on the interaction between cardiac output, arterial oxygen content, hemoglobin concentration, perfusion pressure, and cerebral autoregulation. In patients with severe ventricular dysfunction after ALCAPA repair, this vulnerability may be amplified by low systemic output, recurrent arrest, thromboembolic risk, and the need for mechanical circulatory or ventilatory support.

Cortical laminar necrosis is a distinctive pattern of selective cortical neuronal injury involving metabolically vulnerable cortical layers, most classically associated with profound oxygen-glucose deprivation. It may occur after hypoxic–ischemic encephalopathy, cerebral infarction, prolonged seizures, hypoglycemia, metabolic disorders, or severe systemic illness [9,10]. In children, cortical laminar necrosis is uncommon and often reflects severe cerebral energy failure. Magnetic resonance imaging, particularly diffusion-weighted imaging and susceptibility-weighted imaging, can help characterize the timing and hemorrhagic components of the injury [9]. A recent pediatric retrospective study further emphasized that hypoxic–ischemic, vascular, infectious, traumatic, and metabolic etiologies represent important causes of cortical laminar necrosis in children, with outcomes depending on the extent and underlying mechanism of brain injury [10]. In the postoperative cardiac setting, the appearance of cortical laminar necrosis should prompt careful reconstruction of hemodynamic, thromboembolic, seizure-related, and resuscitation events.

This case report describes a pediatric patient who developed cortical laminar necrosis and hypoxic–ischemic encephalopathy after ALCAPA repair complicated by severe left ventricular dysfunction, mitral regurgitation, and intracardiac thrombus. The case is noteworthy because it illustrates a severe postoperative neurocardiac cascade in which myocardial dysfunction, thrombus formation, suspected embolic or hypoxic–ischemic cerebral injury, respiratory failure, infection, and recurrent cardiopulmonary deterioration converged despite surgical correction of the coronary anomaly. By presenting this case, we aim to emphasize the need for vigilant postoperative surveillance using serial echocardiography, early neurological assessment, timely neuroimaging, individualized anticoagulation strategies, and multidisciplinary pediatric cardiac intensive care in high-risk ALCAPA patients.

Case Presentation

Patient Background

A pediatric patient was managed at a tertiary care cardiac center in Riyadh, Saudi Arabia, with a confirmed diagnosis of anomalous origin of the left coronary artery from the pulmonary artery (ALCAPA). The patient had a complex perioperative course requiring advanced pediatric cardiac intensive care, including mechanical circulatory support, surgical correction of the coronary anomaly, serial cardiac imaging, neurological evaluation, respiratory support, and multidisciplinary postoperative management.

The patient underwent ALCAPA repair on 28 August 2025. Prior to definitive surgical repair, extracorporeal membrane oxygenation (ECMO) support was initiated on 26 August 2025, reflecting significant preoperative cardiorespiratory instability and the need for mechanical circulatory support. ECMO decannulation was successfully performed on 28 August 2025, and delayed sternal closure was completed on 30 August 2025. Despite successful anatomical repair and early postoperative stabilization measures, the subsequent clinical course was complicated by severe left ventricular dysfunction, mitral regurgitation, intracardiac thrombus formation, neurological injury, respiratory failure, infectious complications, and recurrent cardiopulmonary deterioration.

The case was further complicated by the development of hypoxic–ischemic encephalopathy, seizures controlled with antiepileptic medications, radiological evidence of cortical laminar necrosis, repeated failed extubation attempts, and persistent postoperative myocardial dysfunction. The patient was later referred back to the referring maternity and children's hospital on 7 October 2025. The final clinical course was unfavorable, with mortality recorded on 15 October 2025 following prolonged cardiopulmonary resuscitation lasting 36 minutes.

Preoperative Status and ECMO Support

Before definitive surgical correction, the patient demonstrated significant cardiorespiratory instability in the setting of ALCAPA-associated myocardial ischemia and ventricular dysfunction. Given the severity of the clinical condition and the need for advanced circulatory support, extracorporeal membrane oxygenation was initiated on 26 August 2025 as a bridge to surgical repair. The requirement for ECMO reflected a high-risk preoperative state, most likely related to impaired myocardial performance, compromised systemic perfusion, and the anticipated vulnerability of the left ventricle before restoration of a dual-coronary circulation.

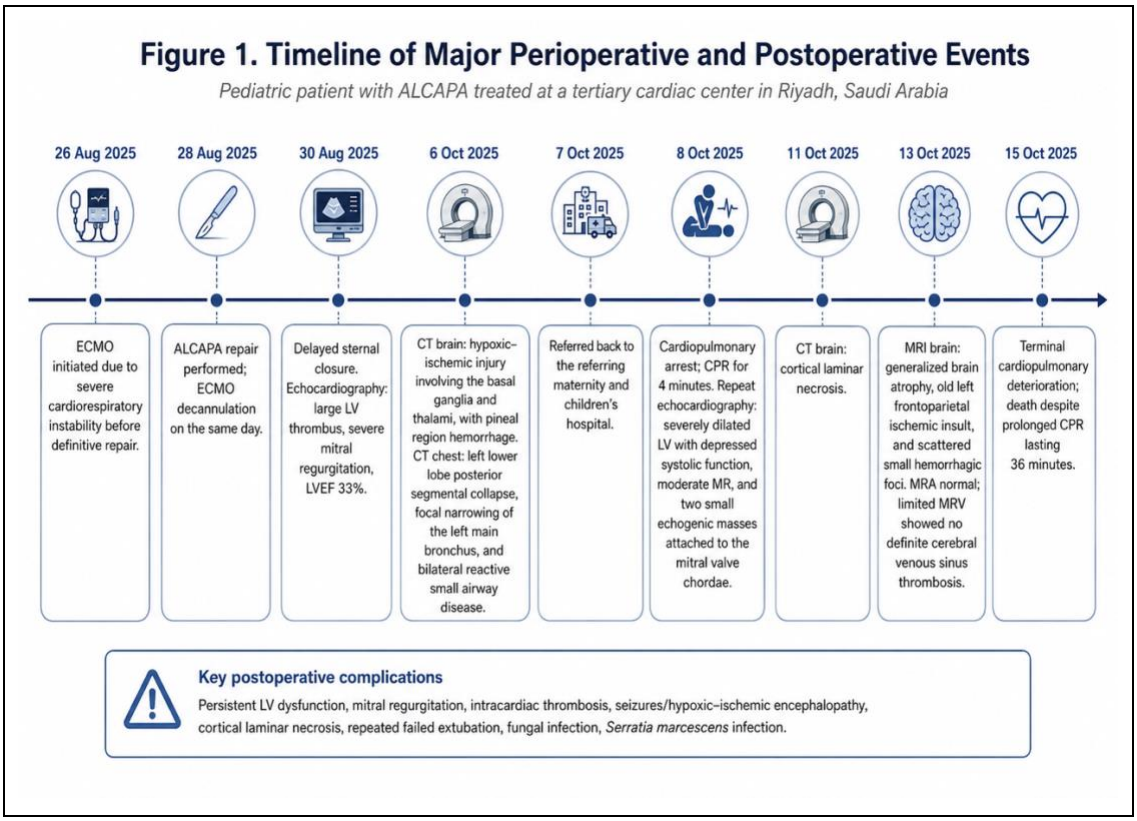
The patient subsequently underwent ALCAPA repair on 28 August 2025, with successful ECMO decannulation performed on the same day. This sequence suggested an initial postoperative improvement sufficient to permit separation from mechanical circulatory support. However, the need for ECMO before repair indicated the severity of the preoperative myocardial compromise and placed the patient at increased risk for a complicated postoperative course, including low cardiac output physiology, persistent left ventricular systolic dysfunction, thrombus formation, and multisystem complications. Delayed sternal closure was completed on 30 August 2025, consistent with the need for cautious postoperative management in the context of recent ECMO support and complex congenital cardiac surgery.

Surgical Repair and Early Postoperative Course

The patient underwent surgical repair of ALCAPA on 28 August 2025 at a tertiary cardiac center in Riyadh, following preoperative stabilization with extracorporeal membrane oxygenation. ECMO was successfully discontinued on the same day as the corrective procedure, indicating an initial capacity to separate from mechanical circulatory support after restoration of coronary perfusion. Given the complexity of the case and the anticipated risk of postoperative myocardial edema, hemodynamic instability, and low cardiac output physiology, delayed sternal closure was performed on 30 August 2025.

Despite successful surgical correction and ECMO decannulation, the early postoperative course was complicated and clinically unstable. Serial echocardiographic assessment soon after repair demonstrated significant residual myocardial dysfunction. Echocardiography performed on 30 August 2025 revealed a severely compromised left ventricle with an ejection fraction of 33%, associated with severe mitral regurgitation and a large left ventricular thrombus. These findings suggested persistent postoperative ischemic cardiomyopathy and a low-flow ventricular state despite anatomical correction of the coronary anomaly. The presence of a large intracardiac thrombus early after repair introduced a major risk for systemic embolization and further complicated postoperative anticoagulation and hemodynamic management.

The early postoperative period was therefore characterized by a high-risk combination of severe left ventricular systolic dysfunction, mitral valve regurgitation, intracardiac thrombosis, and the need for intensive cardiorespiratory monitoring. Although the patient was initially separated from ECMO, the subsequent course remained stormy, requiring ongoing pediatric cardiac intensive care, serial echocardiography, anticoagulation therapy, ventilatory support, and multidisciplinary evaluation (Figure 1).



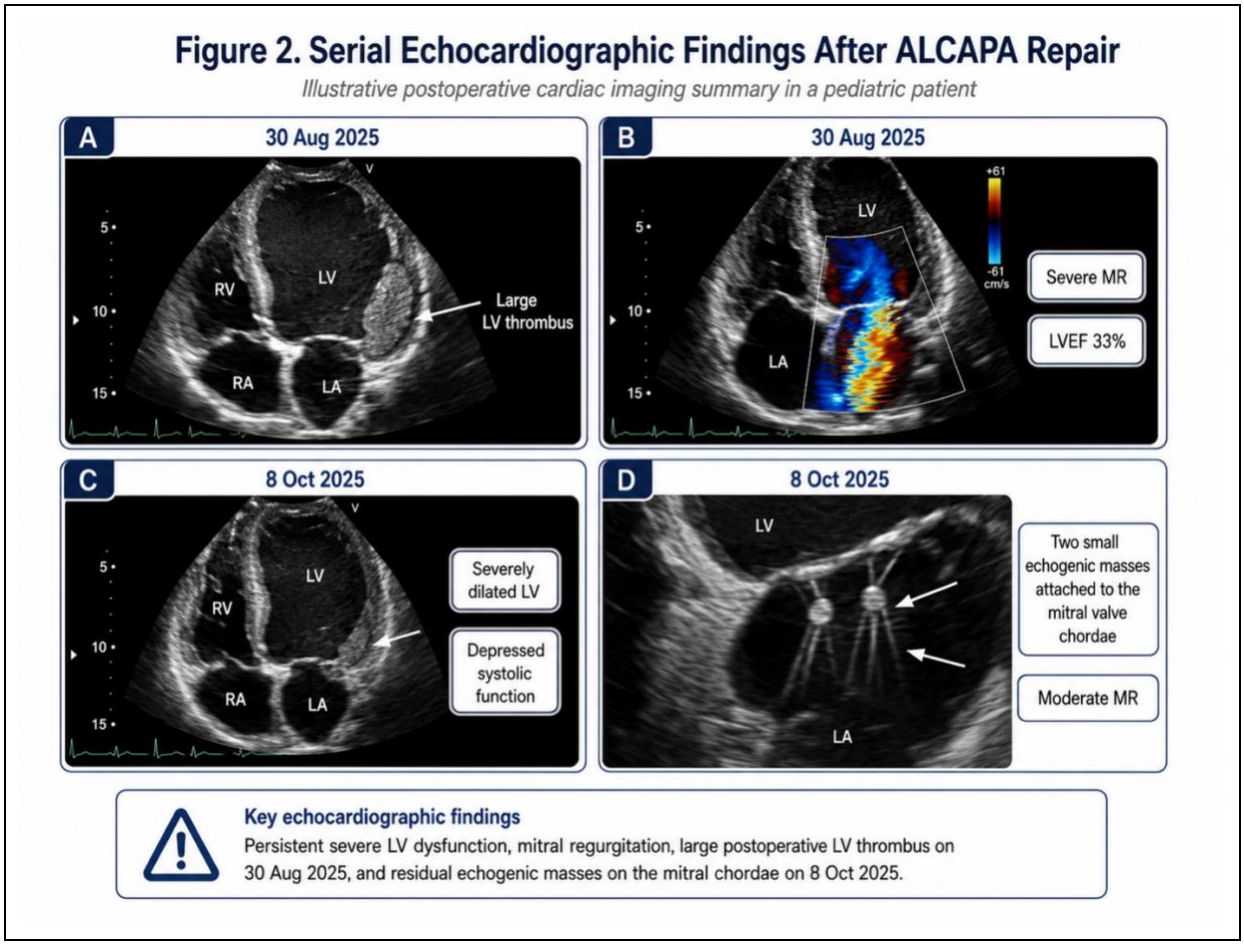
Cardiac Complications and Intracardiac Thrombosis

The postoperative cardiac course was dominated by persistent severe left ventricular dysfunction, mitral regurgitation, and intracardiac thrombus formation. On 30 August 2025, shortly after ALCAPA repair and delayed sternal closure, transthoracic echocardiography demonstrated a large left ventricular thrombus, severe mitral regurgitation, and markedly impaired left ventricular systolic function with an estimated ejection fraction of 33%. These findings were consistent with ongoing postoperative ischemic cardiomyopathy and a low-flow ventricular state, despite anatomical correction of the anomalous coronary circulation.

The presence of a large left ventricular thrombus represented a major postoperative complication, particularly in the context of a dilated and poorly contracting left ventricle. The thrombus was managed with enoxaparin anticoagulation, with subsequent documentation suggesting interval improvement. However, the thrombotic burden remained clinically significant because of the potential risk of systemic embolization, including cerebral embolic events. In this case, the later development of seizures and hypoxic–ischemic brain injury raised concern for a possible thromboembolic contribution, although the neurological injury was likely multifactorial and may have also reflected perioperative hypoperfusion, cardiac arrest, and prolonged critical illness.

Repeat echocardiography on 8 October 2025 continued to show significant postoperative cardiac pathology. The left ventricle remained severely dilated with depressed systolic function, and mitral regurgitation persisted, although it was described as moderate rather than severe. In addition, two small echogenic masses were noted attached to the mitral valve chordae, raising concern for residual thrombotic material, fibrinous deposits, or other postoperative echogenic lesions. These findings emphasized the ongoing complexity of the patient's cardiac status and the need for continued serial echocardiographic surveillance, anticoagulation monitoring, and multidisciplinary discussion between pediatric cardiology, cardiac surgery, intensive care, hematology, and neurology teams.

Overall, the cardiac course illustrated a severe postoperative neurocardiac risk profile: persistent left ventricular systolic failure, mitral regurgitation, intracardiac thrombosis, and possible embolic vulnerability following ALCAPA repair. Although the thrombus showed improvement on anticoagulation, the patient's global postoperative trajectory remained unfavorable, with progressive multisystem deterioration and later fatal cardiopulmonary arrest (Figure 2).



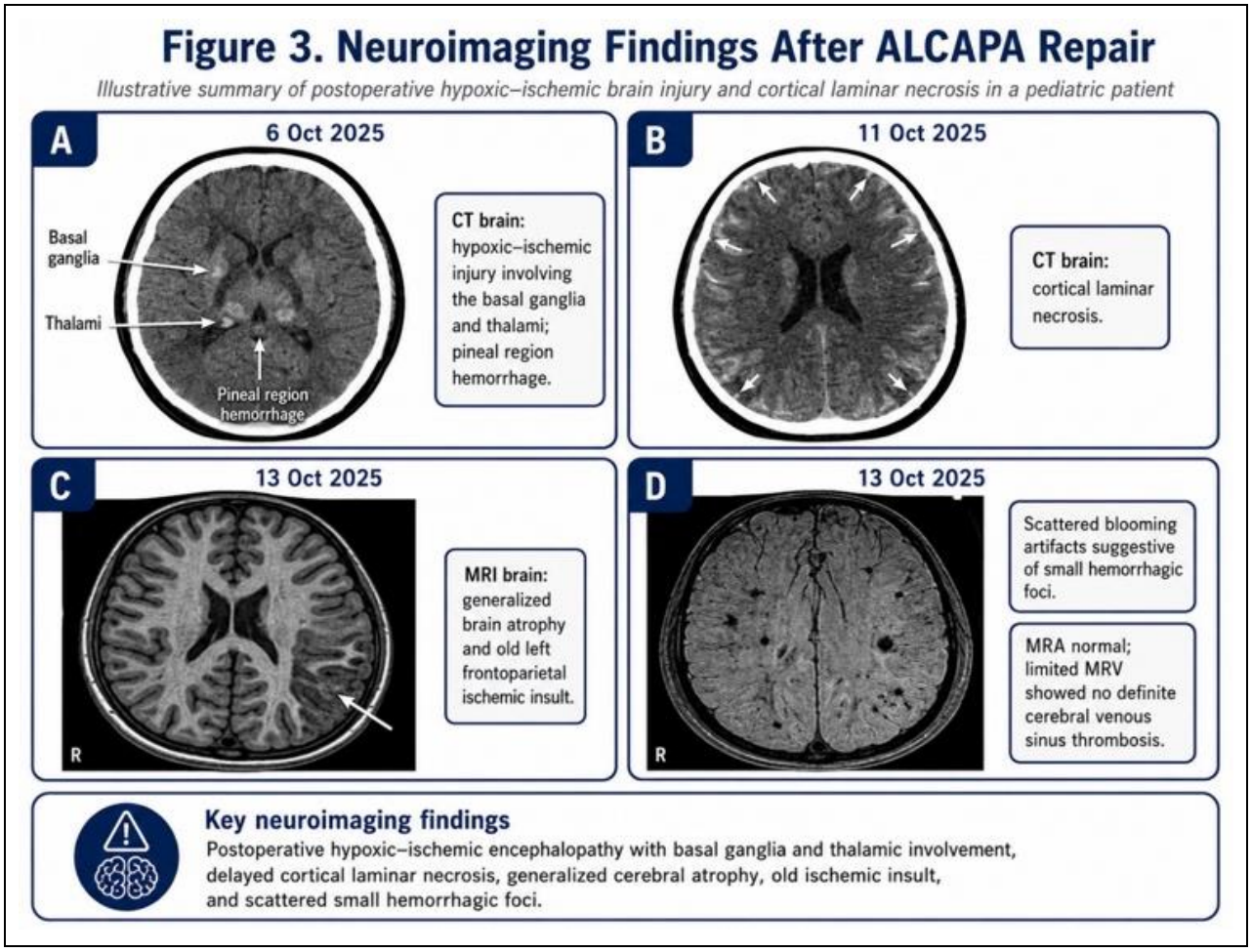
Neurological Deterioration and Neuroimaging Findings

The postoperative course was further complicated by significant neurological deterioration, manifested primarily by convulsive episodes requiring antiepileptic medications. The neurological insult was considered most consistent with hypoxic–ischemic encephalopathy, with a possible thromboembolic contribution in the context of the documented intracardiac thrombus and severe postoperative left ventricular dysfunction. Given the patient's complex cardiac course, including ALCAPA repair, ECMO support, persistent ventricular dysfunction, intracardiac thrombosis, and later cardiopulmonary arrest, the mechanism of brain injury was likely multifactorial, involving impaired cerebral perfusion, hypoxemia, and possible embolic phenomena.

Brain CT performed on 6 October 2025 demonstrated features of hypoxic–ischemic brain injury, with involvement of the basal ganglia and thalami. The scan also showed a pineal region hemorrhage, without evidence of hydrocephalus or significant mass effect. These findings supported the presence of severe global cerebral injury in a vulnerable postoperative cardiac patient. Subsequent CT imaging on 11 October 2025 demonstrated cortical laminar necrosis, indicating delayed cortical injury following severe hypoxic–ischemic or ischemic insult. This radiological evolution was clinically relevant because cortical laminar necrosis is typically associated with profound cerebral oxygen-glucose deprivation and may reflect irreversible neuronal damage.

Further neuroimaging with brain MRI on 13 October 2025 showed generalized brain atrophy with an old left frontoparietal ischemic insult. Scattered foci of blooming artifact were also observed, suggestive of small hemorrhagic foci. Magnetic resonance angiography of the brain was reported as normal. Magnetic resonance venography was limited, but no definite filling defect was identified within the cerebral venous sinuses to suggest venous sinus thrombosis. The radiology report noted that CT brain venography would be the preferred modality if cerebral venous sinus thrombosis remained clinically suspected.

Collectively, the neurological and imaging findings described a severe postoperative brain injury pattern characterized by hypoxic–ischemic encephalopathy, seizure activity, basal ganglia and thalamic involvement, cortical laminar necrosis, cerebral atrophy, and small hemorrhagic foci. In the context of severe left ventricular dysfunction and intracardiac thrombosis, these findings raised concern for a combined hypoxic–ischemic and possible thromboembolic mechanism. This neurocardiac association represents a central feature of the case and highlights the importance of early neurological surveillance, seizure control, serial neuroimaging, and careful anticoagulation decision-making in high-risk pediatric patients after ALCAPA repair (Figure 3).



Respiratory and Infectious Complications

The postoperative course was also complicated by significant respiratory morbidity, requiring prolonged ventilatory support and repeated attempts at liberation from mechanical ventilation. The patient experienced three failed extubation trials during the postoperative intensive care course, reflecting a complex interaction between persistent cardiac dysfunction, impaired pulmonary mechanics, airway pathology, neurological impairment, and overall critical illness. In the setting of severe left ventricular systolic dysfunction and mitral regurgitation, respiratory recovery was likely further compromised by pulmonary congestion, reduced cardiopulmonary reserve, and vulnerability to recurrent decompensation.

Chest CT performed on 6 October 2025 demonstrated posterior segmental collapse of the left lower lung lobe, associated with focal narrowing of the left main bronchus. The same study also showed features suggestive of bilateral reactive small airway disease. These findings provided a radiological explanation for part of the patient's difficult respiratory course and repeated extubation failure. The left lower lobe collapse and left main bronchial narrowing may have contributed to impaired ventilation, atelectasis, secretion retention, and reduced respiratory reserve, while the background of small airway disease could have increased the risk of bronchospasm and prolonged dependence on respiratory support.

In addition to respiratory complications, the patient developed infectious complications during the postoperative period, including a documented fungal infection and Serratia marcescens infection. Both infections were later described as resolved. These infections occurred in the context of prolonged critical care admission, invasive monitoring, mechanical ventilation, recent cardiac surgery, prior ECMO support, and systemic vulnerability. Although the available data do not specify the source, timing, or antimicrobial regimen, the occurrence of fungal and Gram-negative infection likely contributed to the overall inflammatory burden, prolonged intensive care course, and difficulty achieving stable recovery.

Overall, the respiratory and infectious complications formed an important part of the patient's multisystem postoperative deterioration. Recurrent extubation failure, radiological evidence of lobar collapse and airway narrowing, bilateral small airway reactivity, and resolved fungal and Serratia marcescens infections collectively reflected the severity of the postoperative course following ALCAPA repair. These complications further compounded the already high-risk cardiac and neurological status and contributed to the complexity of ongoing pediatric cardiac intensive care management.

Clinical Deterioration and Outcome

Despite surgical correction of ALCAPA and initial separation from extracorporeal membrane oxygenation, the patient's postoperative course remained clinically unstable and was characterized by progressive multisystem complications. The major ongoing issues included severely depressed left ventricular systolic function, mitral regurgitation, intracardiac thrombus formation, hypoxic–ischemic encephalopathy with seizures, radiological evidence of cortical laminar necrosis, repeated failed extubation attempts, pulmonary collapse with airway narrowing, and resolved infectious complications including fungal infection and Serratia marcescens. Although the intracardiac thrombus showed interval improvement under enoxaparin therapy, the overall clinical trajectory remained unfavorable.

The patient experienced an episode of cardiopulmonary arrest on 8 October 2025, requiring approximately 4 minutes of cardiopulmonary resuscitation before return of circulation. This event occurred in the setting of persistent postoperative myocardial dysfunction and severe neurological and respiratory vulnerability. Repeat echocardiography on the same date demonstrated a severely dilated left ventricle with depressed systolic function, moderate mitral regurgitation, and two small echogenic masses attached to the mitral valve chordae, indicating persistent structural and functional cardiac abnormalities despite ongoing management.

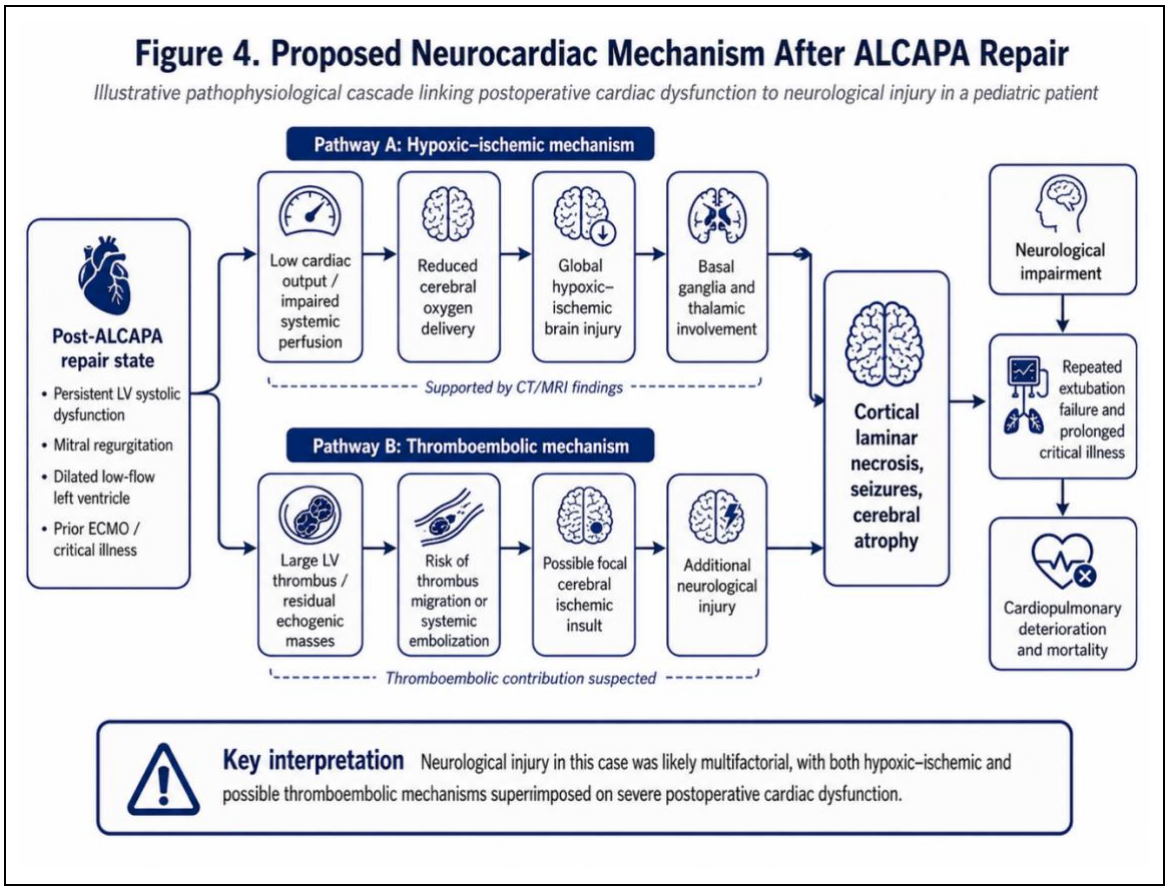
Following referral back to the referring maternity and children's hospital on 7 October 2025, the patient continued to deteriorate clinically. Neuroimaging performed during this period confirmed extensive hypoxic–ischemic brain injury, including cortical laminar necrosis, generalized brain atrophy, old ischemic insult, and scattered small hemorrhagic foci. The patient subsequently developed terminal cardiopulmonary collapse and died on 15 October 2025 despite prolonged resuscitative efforts lasting 36 minutes. The final outcome reflected a severe postoperative neurocardiac cascade after ALCAPA repair, in which persistent myocardial dysfunction, intracardiac thrombosis, neurological injury, respiratory failure, and critical illness collectively contributed to mortality.

Discussion

This case illustrates a severe postoperative neurocardiac cascade following ALCAPA repair, characterized by persistent left ventricular dysfunction, mitral regurgitation, intracardiac thrombosis, hypoxic–ischemic encephalopathy, cortical laminar necrosis, respiratory failure, and eventual mortality. Although surgical restoration of a dual-coronary system is the definitive treatment for ALCAPA, postoperative recovery is not always immediate, particularly in patients who present with severe myocardial dysfunction or require mechanical circulatory support. Previous surgical outcome studies have shown that left ventricular function and mitral regurgitation may improve over time after repair; however, early postoperative morbidity remains substantial in high-risk infants with ischemic cardiomyopathy [1,2]. In the present case, the early postoperative echocardiographic finding of an ejection fraction of 33%, severe mitral regurgitation, and a large left ventricular thrombus reflected a profoundly vulnerable low-flow state despite anatomical correction.

The development of intracardiac thrombosis in this setting is pathophysiologically plausible. Severe ventricular dilation, impaired systolic contraction, endocardial injury, systemic inflammation, postoperative low cardiac output, and prior ECMO exposure all contribute to thrombus formation. Pediatric intracardiac thrombi are uncommon but clinically important because embolization, obstruction, and anticoagulation-related bleeding may all occur, and management is often individualized rather than standardized [3]. Recent pediatric data emphasize that anticoagulation strategies for intracardiac thrombi vary widely, with most patients requiring prolonged therapy and serial imaging to document thrombus resolution [3]. Neonatal and infant intracardiac thrombosis literature similarly highlights the importance of early recognition, echocardiographic surveillance, and individualized anticoagulation, particularly when hemorrhagic risk is high [4]. In this patient, enoxaparin therapy was associated with interval improvement of the thrombus; however, improvement in thrombus burden did not translate into clinical recovery, likely because irreversible neurological and multisystem injury had already occurred.

The neurological injury in this case was likely multifactorial. The presence of severe left ventricular dysfunction, intracardiac thrombus, postoperative instability, ECMO exposure, and cardiopulmonary arrest created several potential pathways for cerebral injury, including global hypoperfusion, hypoxemia, embolic ischemia, seizure-related metabolic demand, and impaired cerebral autoregulation. Recent studies of pediatric cardiac surgery have confirmed that neurological complications, particularly seizures and central nervous system injury, are associated with poorer short-term outcomes, longer hospitalization, and increased mortality [5,6]. In one contemporary pediatric cardiac surgery cohort, seizures were the most frequent neurological manifestation, and central nervous system insult correlated with worse clinical outcomes [6]. This aligns with the present case, in which seizures were a prominent clinical manifestation and were followed by neuroimaging evidence of extensive brain injury (Figure 4).



The radiological evolution toward cortical laminar necrosis is particularly important. Cortical laminar necrosis represents selective cortical neuronal death, typically involving metabolically vulnerable cortical layers after profound oxygen-glucose deprivation. It may follow hypoxic–ischemic encephalopathy, prolonged seizures, infarction, metabolic crisis, or severe systemic illness [7]. In postoperative cardiac patients, cortical laminar necrosis should not be interpreted as a nonspecific imaging finding; rather, it indicates severe cortical energy failure and should prompt careful review of perioperative hemodynamics, oxygenation, thromboembolic risk, seizure burden, and resuscitation events. The associated basal ganglia and thalamic abnormalities in this patient further support severe hypoxic–ischemic injury, because these deep gray matter structures are highly vulnerable to profound global ischemia. The scattered hemorrhagic foci seen on MRI also complicate management, as they increase the difficulty of balancing anticoagulation for intracardiac thrombus against the risk of intracranial bleeding.

The history of ECMO support adds another layer of complexity. ECMO is lifesaving in pediatric cardiac failure but is strongly associated with hemostatic disturbance, including both bleeding and thrombosis. Contemporary ECMO anticoagulation literature emphasizes that optimal monitoring and target ranges remain uncertain in children, and that institutional practice varies considerably [8]. Heparin remains widely used, while alternatives such as bivalirudin have been increasingly discussed, although prospective pediatric evidence remains limited [9]. In this context, a patient transitioning from ECMO to postoperative severe ventricular dysfunction may remain at persistent thrombotic risk even after decannulation. Therefore, serial echocardiography is essential, especially in patients with dilated ventricles, poor contractility, and mitral regurgitation.

Respiratory failure also contributed significantly to the patient's deterioration. Repeated failed extubation attempts were probably not due to a single cause but rather to the convergence of impaired cardiac output, pulmonary venous congestion from mitral regurgitation, left lower lobe collapse, left main bronchial narrowing, small airway reactivity, neurological impairment, and infection. This pattern is clinically relevant because extubation failure after congenital cardiac surgery often signals unresolved cardiopulmonary or neurological pathology rather than isolated respiratory weakness. In this case, respiratory compromise likely amplified cerebral vulnerability by increasing the risk of hypoxemia and recurrent hemodynamic instability. The main value of this case lies in its integration of cardiac, thrombotic, neurological, respiratory, and intensive care complications after ALCAPA repair. While postoperative LV dysfunction and mitral regurgitation are recognized after ALCAPA repair, the combination of large intracardiac thrombus, suspected embolic or hypoxic–ischemic brain injury, cortical laminar necrosis, repeated extubation failure, and mortality is rarely described as a unified postoperative syndrome. This case therefore supports a practical lesson: high-risk ALCAPA patients require proactive multidisciplinary surveillance after repair, including serial echocardiography for thrombus detection, early neurology consultation for seizures or encephalopathy, timely CT/MRI correlation, structured anticoagulation monitoring, and careful respiratory assessment before extubation. Anatomical correction is essential, but in patients with severe preoperative myocardial compromise, postoperative survival depends on early recognition and coordinated management of the downstream neurocardiac consequences.

Conclusion

This case highlights a severe postoperative neurocardiac cascade following surgical repair of ALCAPA in a pediatric patient. Despite successful anatomical correction of the coronary anomaly and initial separation from ECMO, the postoperative course was complicated by persistent left ventricular systolic dysfunction, mitral regurgitation, intracardiac thrombus formation, hypoxic–ischemic encephalopathy, cortical laminar necrosis, recurrent respiratory failure, infectious complications, cardiopulmonary arrest, and eventual mortality. The case emphasizes that ALCAPA repair, particularly in patients with profound preoperative myocardial compromise, should be followed by vigilant multidisciplinary surveillance. Serial echocardiography, early neurological assessment, timely neuroimaging, structured anticoagulation monitoring, and careful respiratory evaluation are essential to identify and manage potentially catastrophic postoperative complications.

Learning Points

• ALCAPA repair does not immediately eliminate postoperative risk, especially in patients with severe preoperative myocardial dysfunction or ECMO requirement.

• Persistent left ventricular dysfunction and mitral regurgitation after ALCAPA repair may create a low-flow state, increasing the risk of intracardiac thrombus formation.

• Intracardiac thrombus after pediatric cardiac surgery requires close serial echocardiographic monitoring because of the potential risk of systemic and cerebral embolization.

• Postoperative seizures in high-risk congenital cardiac surgery patients should prompt urgent neurological evaluation, including consideration of hypoxic–ischemic injury, embolic stroke, hemorrhage, and seizure-related cortical injury.

• Cortical laminar necrosis is an important imaging marker of severe cortical injury, most often reflecting profound hypoxic–ischemic or metabolic insult.

• The mechanism of brain injury in this case was likely multifactorial, involving severe ventricular dysfunction, possible thromboembolism, hypoperfusion, hypoxemia, critical illness, and cardiopulmonary arrest.

• Repeated extubation failure after congenital cardiac surgery should be interpreted as a warning sign, requiring reassessment of cardiac function, airway anatomy, lung collapse, infection, and neurological status.

• Anticoagulation in postoperative pediatric cardiac patients is clinically challenging, particularly when intracardiac thrombus coexists with intracranial hemorrhagic findings.

• A multidisciplinary pediatric cardiac ICU approach is essential, involving cardiology, cardiac surgery, intensive care, neurology, radiology, hematology, infectious disease, respiratory therapy, and rehabilitation teams.

• This case supports the need for early integrated neurocardiac surveillance protocols in high-risk ALCAPA patients after surgical repair.

Timeline

Date	Clinical Event
26-Aug-25	ECMO support initiated due to severe cardiorespiratory instability before definitive ALCAPA repair.
28-Aug-25	Surgical repair of ALCAPA performed. ECMO decannulation was completed on the same day.
30-Aug-25	Delayed sternal closure performed. Echocardiography showed large left ventricular thrombus, severe mitral regurgitation, and reduced left ventricular ejection fraction of 33%.
06-Oct-25	Brain CT showed hypoxic–ischemic brain injury involving the basal ganglia and thalami, with pineal region hemorrhage and no hydrocephalus or mass effect. Chest CT showed left lower lobe posterior segmental collapse, focal narrowing of the left main bronchus, and bilateral reactive small airway disease.
07-Oct-25	Patient was referred back to the referring maternity and children's hospital.
08-Oct-25	Cardiopulmonary arrest occurred, requiring approximately 4 minutes of CPR. Repeat echocardiography showed post-ALCAPA repair status with severely dilated and depressed left ventricular systolic function, moderate mitral regurgitation, and two small echogenic masses attached to the mitral valve chordae.
11-Oct-25	CT brain demonstrated cortical laminar necrosis.
13-Oct-25	Brain MRI showed generalized brain atrophy, old left frontoparietal ischemic insult, and scattered blooming artifacts suggestive of small hemorrhages. MRA was normal. MRV was limited but showed no definite cerebral venous sinus thrombosis.
15-Oct-25	Patient died following terminal cardiopulmonary deterioration despite prolonged CPR lasting 36 minutes.

Consent and Ethical Considerations

Written informed consent was obtained from the patient's legal guardian for publication of this case report and any accompanying anonymized clinical information. All patient identifiers were removed to preserve confidentiality. Institutional requirements for case-report publication were followed.

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