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Case Report

Management Challenge of Culture-Negative Infective Endocarditis in an Adolescent with Uncorrected Patent Ductus Arteriosus: A Case Report

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Abstract

Infective Endocarditis (IE) remains a life-threatening complication for patients with uncorrected Congenital Heart Disease (CHD). This case report illustrates the complex clinical management of a 39 kg pediatric patient presenting with a five-day history of fever and acute heart failure. Clinical examination and Transthoracic Echocardiography (TTE) confirmed the presence of a Patent Ductus Arteriosus (PDA) with associated bacterial vegetation, consistent with IE. The patient's condition was further complicated by sepsis and hypochromic microcytic anemia. Management required a high-precision, multi-modal approach to balance competing physiological demands. To eradicate the infection, a synergistic regimen of broad-spectrum antibiotics (Ceftriaxone and Gentamicin) was initiated. Simultaneously, heart failure was addressed using a combination of diuretics (Furosemide, Spironolactone) and afterload reduction via ACE-inhibitors (Captopril). A critical component of the intervention was strict fluid restriction (1900 ml/day) to prevent pulmonary volume overload, despite the concurrent systemic sepsis. Supportive therapies included corticosteroids and gastrointestinal care. This case demonstrates that while sepsis typically necessitates fluid resuscitation, the presence of a PDA-induced shunt requires cautious fluid titration to maintain hemodynamic stability. The successful stabilization of this patient underscores the necessity of aggressive antimicrobial therapy and meticulous fluid management in complex CHD complications. Ultimately, this report emphasizes the vital importance of early PDA closure as a primary preventive measure against the development of high-morbidity Infective Endocarditis.

Keywords: Heart Failure, Infective Endocarditis, Patent Ductus Arteriosus, Pediatric Cardiology, Sepsis.

1. Introduction

Infective Endocarditis (IE) is a severe infection of the cardiac endothelium that remains a formidable challenge in pediatric medicine, carrying high morbidity and mortality rates (Marín-Cruz et al., 2024; Vicent et al., 2022). While IE is relatively rare in children, its incidence has shown an increasing trend in recent years, particularly among those with pre-existing cardiac conditions (Abramczyk et al., 2024; Dias et al., 2025). Unlike adults, where degenerative valve disease is a common precursor, Congenital Heart Disease (CHD) is the predominant predisposing factor in the pediatric population, accounting for approximately 50-70% of cases (Rahayuningsih et al., 2023; Rushani et al., 2013). In Indonesia, studies indicate that non-cyanotic CHD, such as Patent Ductus Arteriosus (PDA), is a significant contributor to pediatric IE cases (Yosy & Nova, 2019).

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The pathophysiology of IE in PDA is driven by high-velocity turbulent blood flow through the ductal shunt. This turbulence creates "jet lesions" and mechanical stress on the pulmonary artery endothelium, leading to the formation of a sterile thrombotic nidus (Baltimore et al., 2015; Mahajan et al., 2022). During episodes of bacteremia, microorganisms colonize this damaged area, resulting in vegetations that are difficult for the immune system and pharmacological agents to penetrate (Rashed et al., 2023; Vicent et al., 2022).

Diagnosis in children is frequently complex due to nonspecific clinical manifestations, often overlapping with systemic inflammatory responses or sepsis (Marín-Cruz et al., 2024). While the Modified Duke Criteria remain the diagnostic standard, contemporary management emphasizes multimodal imaging to improve sensitivity (Baltimore et al., 2015; Vicent et al., 2022). Furthermore, culture-negative IE which often caused by prior antibiotic use presents a therapeutic dilemma necessitating aggressive empirical therapy (Rahayuningsih et al., 2023).

Effective management of IE complicated by heart failure and sepsis requires a delicate hemodynamic balance. Traditional sepsis resuscitation must be carefully titrated to avoid volume overload, which can exacerbate heart failure in the presence of a left-to-right shunt (Baltimore et al., 2015; Mahajan et al., 2022).

This case report aims to achieve the following objectives: 1) Illustrate how uncorrected PDA serves as a primary nidus for vegetation in the pulmonary artery, even in adolescent patients. 2) Discuss the challenges of managing culture-negative IE where clinical criteria and echocardiographic findings must guide aggressive empirical treatment. 3) Detail the delicate balance required to treat concurrent sepsis and heart failure specifically the titration of fluid resuscitation against the need for preload reduction. 4) Reinforce the clinical necessity of early surgical or transcatheter closure of PDAs to eliminate the long-term risk of catastrophic endocardial infection. By presenting the successful management of a 16-year-old patient through a combination of tailored pharmacotherapy and strict fluid monitoring, this report contributes to the clinical framework for treating high-risk IE in the setting of uncorrected congenital defects.

2. Literature Review

Infective Endocarditis (IE) in the pediatric population is a rare but potentially devastating multisystemic disease characterized by the infection of the endocardial surface of the heart, including cardiac valves and great vessels (Baltimore et al., 2015). Over the past few decades, the clinical landscape of pediatric IE has shifted from being a complication of rheumatic fever to primarily affecting children with Congenital Heart Disease (CHD) (Marín-Cruz et al., 2024; Rashed et al., 2023). This transition is largely due to improved surgical outcomes for complex heart defects, which paradoxically creates a larger population of survivors at risk for endocardial infection (Rushani et al., 2013). Understanding the interplay between altered hemodynamics in defects like Patent Ductus Arteriosus (PDA) and the modern diagnostic frameworks is essential for improving survival rates, which still face significant challenges due to complications like heart failure and sepsis (Aldrich et al., 2025; Yosy & Nova, 2019).

2.1. Epidemiology and Predisposing Factors

The incidence of pediatric IE is estimated at 0.43 to 0.69 cases per 100,000 children annually (Baltimore et al., 2015). In tertiary care settings, CHD remains the leading risk factor, present in up to 90% of cases (Rashed et al., 2023). Left-to-right shunt lesions, such as Patent Ductus Arteriosus (PDA) and Ventricular Septal Defects (VSD), are particularly susceptible due to the high-pressure gradients they generate (Rushani et al., 2013).

However, emerging data suggests an increasing prevalence of IE in children without underlying heart disease (Lin et al., 2013). This "new" cohort often includes neonates and children with chronic debilitating conditions who require long-term central venous catheters (Dias et al., 2025; Vicent et al., 2022). Furthermore, specific populations, such as children with Down Syndrome, face an elevated risk of IE following invasive procedures, such as dental extractions, due to their unique immune profiles and higher rates of associated CHD (Isezuo et al., 2025).

2.2. Pathogenesis: Three Core Theories

The literature identifies three primary mechanisms that facilitate the development of IE in pediatric patients: In patients with PDA, blood flows at high velocity from the high-pressure aorta into the lower-pressure pulmonary artery. This "jet" creates intense turbulence that mechanically traumatizes the endocardium (Baltimore et al., 2015; Mahajan et al., 2022). This injury leads to the exposure of subendothelial collagen and the subsequent deposition of platelets and fibrin, forming a sterile nidus known as Non-Bacterial Thrombotic Endocarditis (NBTE) (Zhang et al., 2025).

The sterile NBTE serves as a landing site for circulating bacteria. Transient bacteremia can occur through everyday activities like tooth brushing or medical interventions (NICE, 2008). In a child with a pre-existing NBTE, these bacteria adhere to the fibrin-platelet matrix using surface adhesins, effectively colonizing the lesion and transforming it into an active vegetation (Rashed et al., 2023).

Once colonized, the vegetation grows as the bacteria stimulate further fibrin deposition. This creates a "biofilm" environment where pathogens are encased in a protective matrix (Vicent et al., 2022). This structure prevents host phagocytes from reaching the bacteria and significantly limits the penetration of systemic antibiotics, explaining why IE requires prolonged high-dose antimicrobial therapy (Abramczyk et al., 2024; Rahayuningsih et al., 2023).

2.3. Clinical Management and Prevention

The management of pediatric IE is multimodal, involving aggressive antibiotic therapy for 4 to 6 weeks (Aldrich et al., 2025). For patients with PDA, the focus is on balancing infection control with hemodynamic stability. While medical management is the first line, surgical intervention is indicated for persistent infection, heart failure, or large, mobile vegetations at risk of embolization (Djer & Madiyono, 2016; Zhang et al., 2025). Prevention remains critical; although NICE guidelines have narrowed the scope for antibiotic prophylaxis, the AHA continues to recommend it for high-risk CHD groups undergoing dental procedures (Baltimore et al., 2015; NICE, 2008).

3. Methods

This study is a clinical case report describing the diagnostic and therapeutic journey of a 16-year-old female patient. The clinical management and data collection took place at August 2025 inpatient in a primary hospital specializing in pediatric. This report follows the established ethical standards for clinical documentation, ensuring patient anonymity while providing sufficient detail for clinical replication.

3.1. Diagnostic Procedures and Criteria

The diagnosis of Infective Endocarditis (IE) was established using a multimodal approach in accordance with the 2023 Duke-ISCVID Criteria (Fowler et al., 2023; Marín-Cruz et al., 2024).

A. Clinical Assessment: Continuous monitoring of vital signs, including temperature patterns and hemodynamic stability.
B. Imaging: Transthoracic Echocardiography (TTE) was performed to visualize the Patent Ductus Arteriosus (PDA) shunt and identify the presence, size, and mobility of vegetations on the pulmonary artery endothelium (Baltimore et al., 2015).
C. Laboratory Analysis: Serial blood cultures were collected prior to the escalation of antimicrobial therapy. Complete Blood Count (CBC) and peripheral blood smears were utilized to characterize the microcytic hypochromic anemia.

3.2. Therapeutic Intervention and Management

The management strategy was based on the Pediatric Acute Care Cardiology Collaborative (PAC3) Clinical Practice Guidelines (Aldrich et al., 2025). A synergistic intravenous regimen consisting of Ceftriaxone (supplier: generic/hospital formulary) and Gentamicin was administered for 6 weeks to address the suspected pathogens in a culture-negative context. Heart failure is managed through afterload reduction using Captopril and preload control using a combination of Furosemide and Spironolactone. A strict fluid restriction protocol of 1900 ml/day was implemented. This was calculated to balance the metabolic demands of sepsis against the risk of pulmonary congestion caused by the left-to-right PDA shunt (Mahajan et al., 2022).

4. Results and Discussion

4.1. Research Results

4.1.1. Clinical Presentations and Baseline Characteristics

A 16-year-old female weighing 39 kg (underweight) presented with a 5-day history of worsening dyspnea, orthopnea, and intermittent fever. Physical examination confirmed tachycardia (125 bpm) and tachypnea (28 breaths/minute). A characteristic continuous machinery murmur was audible at the left 2nd-3rd intercostal space, consistent with the patient's known history of Patent Ductus Arteriosus (PDA).

4.1.2. Laboratory and Pathological Findings

Initial hematological analysis revealed a significant inflammatory response and anemia, as summarized in Table 1.

Table 1. Initial Laboratory and Pathological Findings

Investigation	Result	Unit	Normal Range*
Hematology			
Hemoglobin	8.7	g/dL	12.0 - 16.0
Leukocytes	17.110	/µL	5.000 – 10.000
Neutrophils	73.8	%	50 - 70
Absolute Neutrophil Count	12.63	\times 10³/µL	1.5 - 7.0
MCV	74.8	fL	80 - 100
MCH	22.4	pg	27 - 32

Investigation	Result	Unit	Normal Range*
Inflammatory Markers			
C-Reactive Protein (CRP)	205 - 209	mg/L	< 5.0
Biochemistry			
Random Blood Glucose	91	mg/dL	< 140
Renal Function Test	Within Normal Limits	-	-
Liver Function Test	Within Normal Limits	-	-
Microbiology			
Blood Culture	No Growth	-	Sterile
Peripheral Blood Smear			
RBC Morphology	Microcytic Hypochromic, Anisocytosis, Poikilocytosis	-	Normocytic Normochromic
WBC Morphology	Toxic Granulation (Hypergranulation)	-	Normal Granulation

Peripheral blood smear evaluation demonstrated microcytic hypochromic cells with anisocytosis and poikilocytosis. Toxic granulation in neutrophils was observed, supporting the presence of a severe systemic infection despite the negative blood cultures.

4.1.3. Imaging and Diagnostic Confirmation

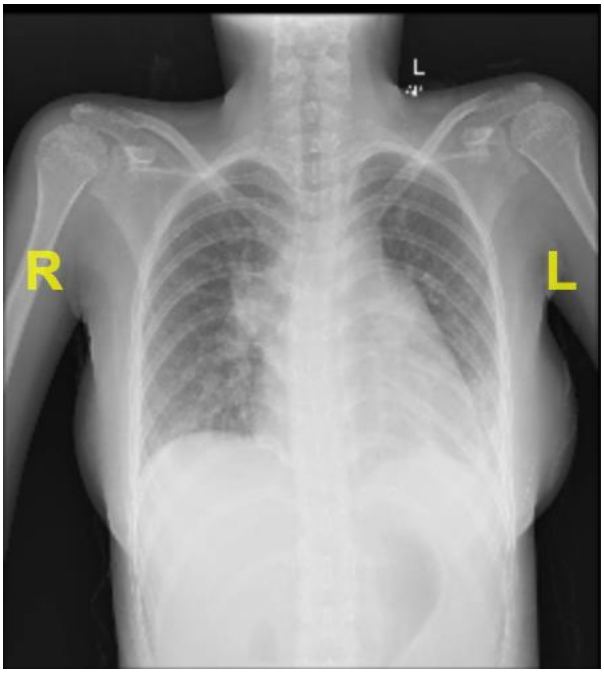

Figure 1. Chest radiography

Figure 1 revealed cardiomegaly (Cor prominent) and cephalization of the pulmonary vasculature, consistent with pulmonary venous congestion (congestive pulmonum). Additionally, patchy infiltrates were observed in the right paracardial region, suggestive of pneumonia. The costophrenic angles were clear with no evidence of pleural effusion, and the bony thorax appeared intact, indicating pulmonary venous congestion and congestive heart failure. Patchy infiltrates in the right paracardial area suggested a superimposed pneumonia.

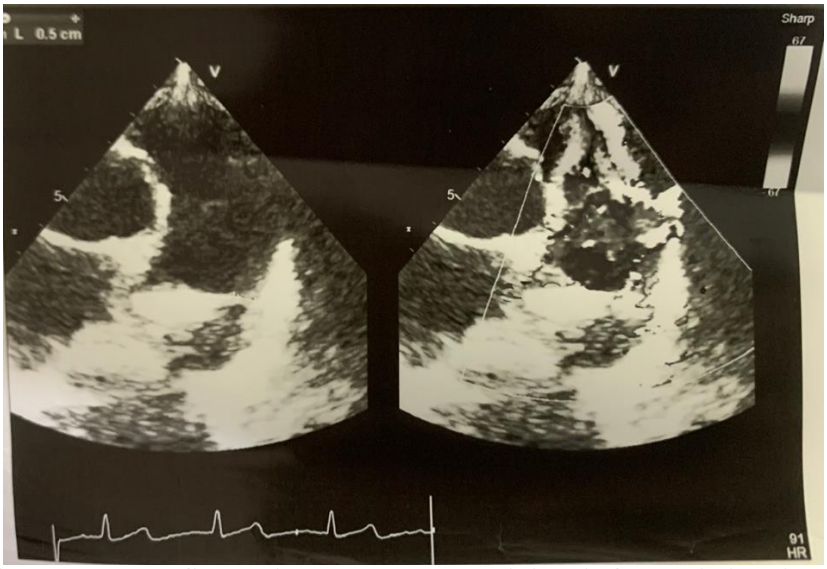

Figure 2. Confirmed a 5 mm PDA with a left-to-right shunt

Transthoracic Echocardiography then performed upon admission to confirm the presence of a Patent Ductus Arteriosus and revealed a vegetation on the pulmonary artery, establishing the diagnosis of Infective Endocarditis (IE). A 5 mm vegetation was visualized on the pulmonary artery wall, fulfilling the major Duke criteria for Infective Endocarditis (IE). Ventricular function remained preserved (LVEF 57%). An additional abdominal ultrasound was performed to screen for septic emboli or other sources of infection and to investigate physical examination finding that is tenderness in epigastric area upon arrival, which returned normal findings for the liver, spleen, and kidneys.

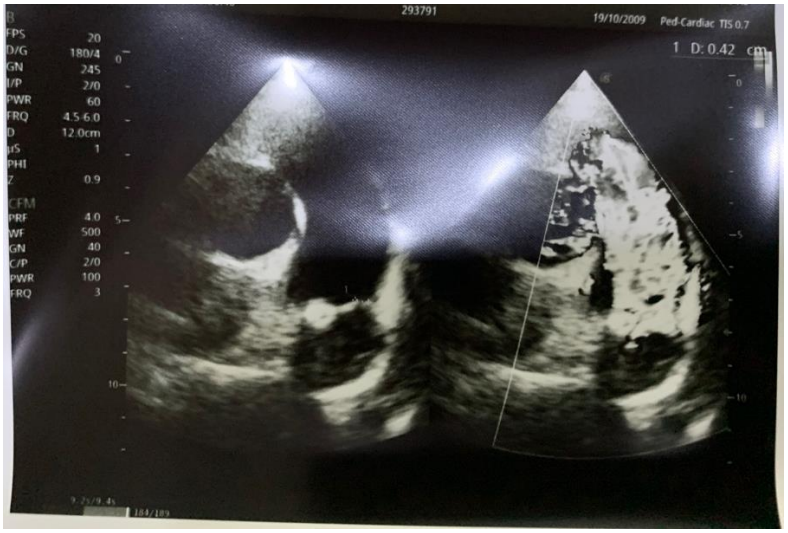

Figure 3. Follow-up echocardiography revealed a 4 mm patent ductus arteriosus (PDA) with hyperdynamic systolic function (EF 76%) and the absence of vegetation

Transthoracic Echocardiography (TTE) then performed again after 14 days of management to evaluate the vegetation progression and to assess LVEF. Follow-up echocardiography showed a patent ductus arteriosus (4-5 mm) with a high pressure gradient (80 mmHg), indicating low pulmonary artery pressure. Left ventricular systolic function was hyperdynamic (EF 76%). Most importantly, the previously noted vegetation was no longer

visualized (no vegetation visualized), suggesting successful resolution of the infective endocarditis.

4.1.4. Therapeutic Outcomes and Clinical Course

The patient underwent a strict 14-day intensive management protocol. Following the administration of intravenous Ceftriaxone and Gentamicin, coupled with a strict fluid restriction of 1,900 ml/day and anti-congestive therapy (Furosemide, Spironolactone, and Captopril), the following results were observed:

A. Clinical Stabilization: Resolution of fever, dyspnea, and tachycardia within the first week.
B. Hematological Improvement: Stabilization of hemoglobin levels following a 200 ml PRC transfusion and a downward trend in inflammatory markers.
C. Follow-up Echocardiography (Day 14): Revealed the complete absence of vegetation (vegetation negative). The PDA persisted (4-5 mm), but the left ventricular systolic function improved to a hyperdynamic state (EF 76%) with a high pressure gradient (80 mmHg), indicating successful resolution of the acute infectious process.

The patient was discharged with a recommendation for elective definitive closure of the PDA to prevent future recurrence of endocarditis.

4.2. Discussion

The clinical trajectory of this 16-year-old female underscores the persistent threat of Infective Endocarditis (IE) in adolescents with uncorrected Congenital Heart Disease (CHD). Despite the global shift in IE epidemiology, CHD remains the primary predisposing factor in 50-70% of pediatric cases, particularly in developing regions where surgical correction may be delayed (Rashed et al., 2023; Yosy & Nova, 2019). This patient's history of a Patent Ductus Arteriosus (PDA) diagnosed in infancy, yet left untreated until adolescence, created a prolonged window of vulnerability for endocardial infection.

The localization of the vegetation on the pulmonary artery in this case perfectly illustrates the "Jet Lesion" theory. In PDA, the high-pressure gradient between the aorta and the pulmonary artery generates a high-velocity turbulent jet that strikes the pulmonary endocardial surface (Baltimore et al., 2015). This mechanical trauma leads to the formation of a sterile fibrin-platelet nidus (Non-Bacterial Thrombotic Endocarditis), which, during episodes of transient bacteremia, becomes colonized by pathogens (Mahajan et al., 2022; Zhang et al., 2025). The presence of toxic granulation in the patient's neutrophils and a CRP level exceeding 200 mg/L further confirm a state of severe systemic inflammation triggered by this colonized vegetation.

A defining challenge in this case was the Culture-Negative Infective Endocarditis (CNIE) status. Literature suggests that 5% to 7% of IE cases yield no growth, often due to prior antibiotic administration or infection by fastidious organisms such as the HACEK group (Baltimore et al., 2015; Rahayuningsih et al., 2023). Despite the negative cultures, the patient met the Modified Duke Criteria through one major criterion (TTE visualization of a 5 mm vegetation) and three minor criteria (fever ≥38◦C, predisposing heart condition, and immunologic phenomena evidenced by massive CRP elevation) (Fowler et al., 2023).

The patient's presentation of concurrent sepsis and congestive heart failure (CHF) created a management paradox. While sepsis typically requires fluid resuscitation, the left-to-right shunt of the PDA causes pulmonary overcirculation, where excess fluids can rapidly lead to pulmonary edema and respiratory failure (Aldrich et al., 2025). The clinical decision to implement a strict fluid restriction of 1,900 ml/day (with a further 10% reduction) alongside aggressive diuresis (Furosemide and Spironolactone) was pivotal. This strategy successfully

reduced the cardiac preload and stabilized the pulmonary vasculature, as evidenced by the resolution of cephalization on follow-up chest radiography.

The successful resolution of the 5 mm vegetation after 14 days of empirical therapy with Ceftriaxone highlights the efficacy of early, broad-spectrum bactericidal intervention in CNIE (Vicent et al., 2022). The transition from an initial ill appearance to a hyperdynamic systolic state (EF 76%) indicates a robust recovery. However, as noted in recent pediatric guidelines, medical stabilization is only a temporary measure; the uncorrected PDA remains a permanent "nidus" for recurrence (Aldrich et al., 2025; Zhang et al., 2025). Definitive transcatheter or surgical closure is mandatory to eliminate the turbulent flow and safeguard the patient from future embolic or infectious catastrophic events.

5. Conclusion

This case underscores that an uncorrected Patent Ductus Arteriosus (PDA) serves as a primary risk factor for Infective Endocarditis (IE) in adolescent patients. The study demonstrates that a 5 mm vegetation can be treated and maintain within 14 days through a multimodal strategy of synergistic antibiotic therapy and aggressive heart failure management.

This case highlights the critical importance of individualized fluid titration when heart failure and systemic infection coexist in a patient with a left-to-right shunt. Consequently, early definitive closure of a PDA is the primary recommendation to prevent life-threatening endocardial infections. Furthermore, clinicians should prioritize meticulous fluid titration and prompt empirical treatment in complex, culture-negative IE cases to ensure favorable clinical outcomes.

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