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CASE REPORT

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Isolated pulmonary valve endocarditis in a pediatric patient with down syndrome

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Abstract

Background Isolated pulmonary valve endocarditis (IPE) accounts for less than 2% of all infective endocarditis patients. It is commonly associated with several predisposing factors, including intravenous drug use (IVDU) and congenital heart disease. The most common causative pathogens of IPE are *Staphylococcus aureus* and Streptococcus *viridans*. We report a Down's syndrome patient with IPE and with no standard risk factors caused by the rare pathogen Acinetobacter spp. This led to respiratory failure and systemic infection due to septic pulmonary emboli. Early elective surgery was decided upon as the patient was no longer responding to medical therapy, and his clinical condition was worsening over time.

Case presentation A 15-year-old male with Down syndrome and no underlying heart defect presented with a 3-month history of episodic fever, nausea, vomiting, and diarrhea. Transthoracic echocardiography (TTE) revealed large vegetation on the pulmonary valve leaflet, another mobile mass at the pulmonary artery bifurcation, and severe pulmonary regurgitation. Serial blood cultures isolated Acinetobacter spp. Despite initial antibiotic therapy, the patient continued to have sepsis, unresolved vegetations, and developed life-threatening complications and respiratory distress, which convinced us to perform a pulmonary valve replacement surgery with a homograft. After surgery, the patient recovered and was discharged on the ninth postoperative day (POD).

Conclusion This report highlights IPE's diagnostic and therapeutic challenges, alongside the importance of a comprehensive cardiopulmonary workup in patients with unexplained fever, sepsis, and pulmonary symptoms, even without typical risk factors. Based on the patient's aggravating condition despite medical treatment, early surgical intervention and pulmonary valve replacement were deemed crucial. However, there still needs to be a definitive guideline on when and how surgery should be performed in patients with complicated IPE, especially in pediatric patients.

Keywords Isolated pulmonary valve endocarditis, Right-sided infective endocarditis, Septic pulmonary embolism, Early surgery, Pulmonary valve replacement, Down syndrome, Acinetobacter spp., Homograft

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Introduction

Right-sided infective endocarditis (RSIE) is considerably less common than left-sided infective endocarditis (LSIE), accounting for only 5–10% of all cases of infective endocarditis (IE), and primarily affects the tricuspid valve in almost 90% of RSIE patients [1–4]. The pulmonic valve, the least commonly involved in IE, accounts for less than 1.5–2% of all IE cases. The isolated form of pulmonary valve (PV) endocarditis, which is even more uncommon, was reported in only 70 patients between 1979 and 2013 [5, 6]. Isolated pulmonary valve endocarditis (IPE) is mainly associated with intravenous drug use (IDU), central venous catheters, congenital heart disease, alcoholism, and sepsis [1–3]. *Streptococcus viridans* is the most common pathogen causing PV endocarditis in non-IDU patients (55–65%). At the same time, *Staphylococcus aureus* is the predominant organism in IDUs, followed by coagulase-negative staphylococci, group B streptococci, and enterococci spp [3, 5]. Septic pulmonary embolism and respiratory failure caused by septic pulmonary emboli or other etiologies are the most common cardiac complications of IPE and require intensive care and prompt treatment to decrease morbidity and mortality rates [7, 8]. We describe a 15-year-old boy with Down syndrome who developed multiple septic pulmonary emboli following IPE caused by an exceedingly rare microorganism without a history of congenital heart disease or predisposing factors that are commonly associated with IPE.

Case presentation

A 15-year-old male with Down syndrome, a history of hypothyroidism, and no underlying heart disease was referred to our hospital with a three-month history of fever, nausea, vomiting, and diarrhea. The patient had no dental work during the last year and could not communicate well or speak fluently due to his unique condition. The patient's overall intelligence level was also significantly lower than the general population. Upon admission, he had experienced persistent fever and chills for one month following a prior two-week hospitalization at another hospital. During his initial hospitalization, he was diagnosed with septicemia and started on intravenous (IV) antibiotics, but his symptoms were not resolved. Finally, the patient presented to our hospital with dyspnea and persistent fever. Physical examination revealed no heart murmurs, but low-pitched rhonchi were auscultated. However, the chest X-ray revealed notable cardiomegaly and bilateral infiltration, while the spiral chest computed tomography (CT) indicated mild pericardial and pleural effusion, patchy ground-glass opacities suggestive of pneumonia, evidence of septic embolism and bilateral enlargement of the para hilar and mediastinal lymph nodes (Fig. 1). Subsequent transthoracic echocardiography (TTE) revealed a sizable hypoechoic mobile mass (measuring 20×11 mm) suggestive of vegetation attached to pulmonary valve leaflet (Fig. 2,), resulting in severe pulmonary regurgitation.

Additionally, another hypoechoic mobile mass (measuring 16×9 mm) was identified near the origin of the left pulmonary artery at the pulmonary artery bifurcation [see Video 1]. The patient exhibited a normal LV size with an ejection fraction of 45% in the left ventricle, severe right ventricular (RV) enlargement with moderate RV systolic dysfunction, and a pulmonary artery pressure of 52 mmHg. No evidence of ventricular septal defect (VSD) or patent ductus arteriosus (PDA) was detected. Two blood cultures were obtained, with the second identifying Acinetobacter spp. The patient was diagnosed with isolated pulmonary valve infective endocarditis (IPE) and subsequently initiated an antibiotic regimen consisting of ceftriaxone 2 g/day and vancomycin 750 mg twice daily before the blood culture results were available.

On the third day after admission, he developed severe respiratory distress, necessitating transfer to the intensive care unit (ICU). Physical examination revealed no heart murmurs, but diffuse rhonchi were heard bilaterally in the lungs. The patient exhibited a pulse rate of

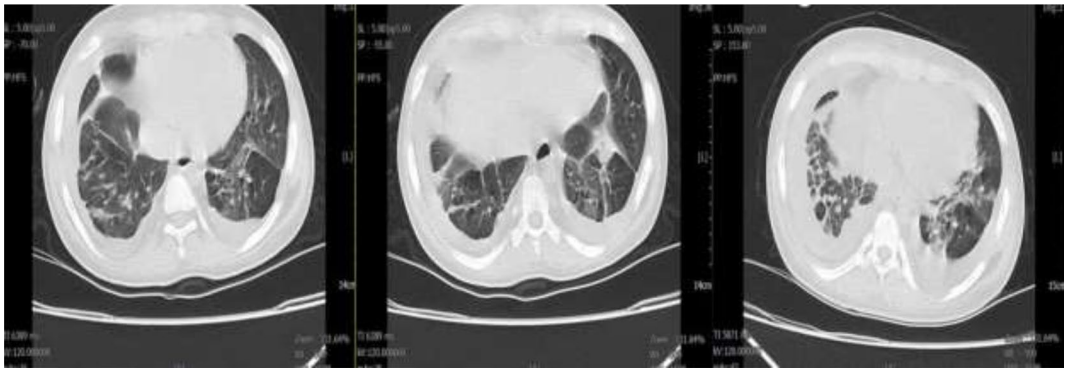

Fig. 1 Spiral chest CT showing mild pericardial and pleural effusion, patchy ground-glass opacities suggestive of pneumonia, evidence of septic embolism, and bilateral enlargement of the para hilar and mediastinal lymph nodes

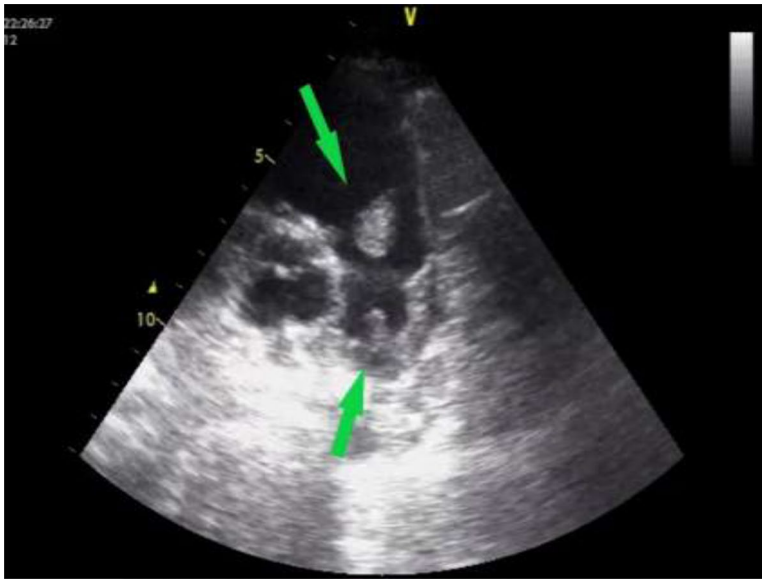

Fig. 2 A sizable hypoechoic mobile mass (measuring 20×11 mm) suggestive of vegetation attached to pulmonary valve leaflet resulting in severe pulmonary regurgitation. Additionally, another hypoechoic mobile mass (measuring 16×9 mm) was identified near the origin of the left pulmonary artery at the pulmonary artery bifurcation

Table 1 Laboratory results of the patient upon admission

	Results	Normal range
White blood cell count	10.4×10^9/L	4.4–11.3×10^9/L
Neutrophil %	76.5%	55–70%
Red blood cell count	3.70 * 10 ^ 6/μL	4.2–5.6 * 10 ^ 6/μL
Hemoglobin	10.2 g/dl	12.5–16 g/dl
Platelets	194×10^9/L	150–450×10^9/L
C-reactive protein	55 mg/dl	< 10 mg/dl
Erythrocyte sedimentation rate	44 mm/hr	< 20 mm/hr
Prothrombin time	13.9 s	11–14 s
Procalcitonin	2 ng/ml	
Lactate	21.6 mmol/L	< 1 mmol/L

130 and a respiratory rate of 35. The laboratory findings are detailed in Table 1. A repeat bedside transthoracic echocardiogram in the ICU corroborated the previous TTE findings. Blood cultures were repeated three times, all yielding Acinetobacter spp. sensitive to meropenem and trimethoprim-sulfamethoxazole. Peripheral blood smear indicated anisocytosis with a significant variation in RBC size and poikilocytosis with no schistocytes or blasts observed. White blood cells and platelets show no considerable morphological abnormalities and are within normal range. Urine analysis indicated 6 to 10 white blood cells with 2+bacteria in the urine. No casts or nitrites were seen or measured, and the catheterized urine culture showed no growth after 72 h of culture. Electrocardiography demonstrated sinus tachycardia. Arterial blood gas (ABG) analysis revealed severe hypoxemia, with a PaO2 ranging from 33.2 to 39.8 mmHg (normal: 75–100 mmHg) and oxygen saturation ranging from 65 to 76% (normal: >95%), along with a normal pH of 7.4. The patient also presented with hypokalemia (potassium level of 3 mmol/L; normal: 3.5–5 mmol/L) and hypocalcemia (ionized calcium level of 0.66 mmol/L; normal: 1.15–1.29 mmol/L) according to arterial blood electrolyte analysis.

Based on the blood culture sensitivities, the antibiotic regimen was adjusted to meropenem 1 g thrice daily plus vancomycin 750 mg twice daily. Additionally, furosemide 20 mg/day was administered to address pulmonary congestion and volume overload before scheduled surgery. Although the preoperative administration of furosemide has no effect on renal protection postoperatively or is even associated with an increased potential risk of AKI when it's taken in high dosage [9], we decided to give a low dose of furosemide to our patient with careful monitoring in a continuous course to promote diuresis and reduce fluid overload [10, 11]. Given the persistent fever coupled with the presence of large vegetation causing severe pulmonary insufficiency and life-threatening complications (e.g., septic pulmonary emboli and respiratory distress), surgical intervention was deemed imperative. On the fourth day after admission, the patient underwent pulmonary valve replacement with a pulmonary homograft. A median sternotomy was performed to establish cardiopulmonary bypass between the ascending aorta, superior vena cava, and inferior vena cava. A cold cardioplegia with antegrade delivery through the aortic root was applied. Subsequent steps included incision of the pulmonary artery, excision of large vegetations, extensive debridement, and thorough irrigation of the surrounding infected tissues. The surgeon confirmed the absence of VSD, PDA, or other congenital abnormalities. The

vegetation specimens were sent to the pathology department for culture and isolation. A pulmonary homograft (size 21) was implanted into the right ventricular outflow tract on the proximal site, and distal anastomosis was performed at the pulmonary artery bifurcation. Following the closure of the pulmonary artery, cardioplegia was terminated, cardiopulmonary bypass was discontinued, and the sternum was closed. The cross-clamp and cardiopulmonary bypass times were 56 min and 106 min, respectively.

On the first postoperative day (POD) in the ICU, the patient developed a high fever, accompanied by a decrease in arterial oxygen saturation to 70%, necessitating intubation. However, his condition improved, leading to his discharge from the ICU on the sixth POD. Meropenem and vancomycin were continued until the end of the hospital stay, with the addition of amikacin 1.5 g/day to the regimen.

The patient was transferred nine days after surgery to the infectious disease ward. After four weeks of antibiotic therapy, he was discharged with improvements in vital signs and general condition, negative blood and urine cultures, a creatinine level of 0.7 mg/dl, and increased arterial oxygen saturation with clear lung sounds. Postoperative follow-up revealed the patient was in stable condition, with no reported complications six months after the operation.

Discussion

PV endocarditis often occurs in younger male patients, yet it remains a rare occurrence among the pediatric population. This condition is linked to various risk factors, particularly congenital heart anomalies, IDU, and chronic vascular access [4, 12, 13]. The isolated form of PV endocarditis, which constitutes less than 2% of the total reported cases of IE when it is associated with no well-established risk factors, has been reported in only a few case reports in the literature. 28% of the patients who had PV endocarditis, whether with or without the involvement of other valves, experienced IPE [3–6, 14]. According to TTE findings, out of 2739 patients diagnosed with infective endocarditis investigated in a 5-year prospective cohort, only 29 had vegetations observed over their pulmonary valve, highlighting the very low prevalence of PV endocarditis with or without the involvement of other valves [15]. Our pediatric patient did not have any underlying heart disease or other identified predisposing risk factors for developing PV endocarditis.

IPE in pediatric patients is not widely recognized, but studies report cases of IPE mainly caused by underlying heart defects or corrective surgeries. The nonspecific symptoms and insidious nature make diagnosis extremely challenging. The most frequently reported symptoms include fever, chills, respiratory illness, and chest pain lasting several days. Transthoracic echocardiography (TTE), chest X-ray, and computed tomography are the most commonly used diagnostic imaging modalities [16–18]. *Staphylococcus aureus*, coagulase-negative *staphylococci*, *group B streptococcus*, and *streptococcus viridans* are the main pathogens leading to IPE in all age groups. Case studies show most pediatric patients respond well to four weeks of targeted antibiotic therapy. Surgical intervention needs to be studied in children more thoroughly. Thus, the same indications applied to adults—such as large, persistent vegetations, prevention of embolic events or heart failure, vegetations extending into the pulmonary trunk and pulmonary complications, and infection resistant to medical treatment—may prompt surgery [16, 17, 19]. There is no specific debate over surgical outcomes in children. Still, a study on IPE surgical management suggests that the surgery has excellent early and late outcomes regardless of whether intervention is timely [20]. While there are established guidelines for managing IPE in adults, pediatric treatment often requires personalized approaches. In this case study, early surgical intervention was necessary as the child's condition worsened despite receiving medical treatment.

Studies indicate a high prevalence of congenital heart disease (CHD) in patients with Down syndrome (DS), ranging from 44 to 62% [21]. Although data on the prevalence of infective endocarditis (IE) in children with DS is limited, the high incidence of CHD in DS suggests a similar pattern of increased IE risk. Sun et al. reported that 11.13 per 10,000 person-years developed IE among children with CHD, with those undergoing heart surgeries being at greater risk [22]. *Staphylococcus aureus* and *Streptococcus viridans* are the primary causative agents of IE in children with CHD, including those with DS. There is also a report of *Leptotrichia buccalis* causing IE in DS patients [23, 24]. Children with DS can have a more complex and subacute IE clinical course, presenting with anemia, pulmonary embolism, and immunologic symptoms [24]. IE mortality rates, mainly assessed in CHD patients, are still relevant for DS children. A study comparing CHD and non-CHD patients in terms of IE mortality rate found an in-hospital mortality rate of 12.7% in CHD patients, lower than that in non-CHD patients with IE [25]. The severity of the underlying cardiac defect and virulence of the causative agent are the main predictors of mortality in children with concomitant CHD and IE [23]. Fudge et al. reported that DS children are more likely to develop postoperative complications such as systemic infections, pulmonary hypertension, and respiratory problems after heart surgeries [26]. Our DS patient did not have any underlying cardiac defect, which complicated the diagnosis and understanding of the IE etiology. This underscores the need for future studies on

the prevalence and clinical course of IE in DS patients without CHD. Children with Down syndrome (DS) frequently have weakened immune systems, resulting in more frequent infections and necessitating more comprehensive treatment compared to children without DS. This immune deficiency is associated with genes on chromosome 21, such as SOD1 and RCAN1, which impair immune responses. Various medical and anatomical comorbidities in DS, defective neutrophil chemotaxis, diminished humoral immune responses, zinc deficiency, and increased immunosenescence increase their vulnerability to infections. These factors probably explain our patient's infection with a rare bacterial species causing IPVE despite having no known risk factors [27].

The most common pathogen in non-IV drug users with PV endocarditis is *Streptococcus viridans* [3]. However, our patient's blood cultures isolated HACEK group (HACEK: *Haemophilus*, *Aggregatibacter*, *Cardiobacterium*, *Eikenella*, *Kingella*) bacteria, which account for only 5-10% of community-acquired native valve endocarditis (NVE) in non-IV drug users [28]. A review by Ioannou et al. found that the aortic valve is the most commonly infected in NVE caused by Acinetobacter spp., with fever, sepsis, heart failure, and embolic phenomena being the most common manifestations and TTE being the best diagnostic tool for IE-developed by this agent group. Acinetobacter spp. Infections are usually hospital-acquired and linked to invasive medical devices like central venous catheters, cardiac surgeries, poor dental hygiene and recent dental work, and IV drug use [29]. To this day, no patients with isolated PV endocarditis by Acinetobacter spp. were reported.

The Modified Duke Major Criteria for diagnosing IE requires three blood cultures from different sites, with the first and last samples drawn one hour apart, to isolate typical microorganisms causing IE [28, 30]. We obtained three blood cultures from different sites, all yielding Acinetobacter spp. Persistent fever and sepsis, pulmonary events such as septic pulmonary embolism, anemia, and microscopic hematuria are the most common clinical manifestations in patients with RSIE. In some studies, the term 'tricuspid syndrome' is used to outline the presence of the abovementioned symptoms in patients with RSIE [31]. When septic pulmonary embolism occurs in the setting of IPE, the patient presents with pneumonia-like symptoms, including fever, shortness of breath, and pleuritic chest pain, accompanied by radiographic and laboratory findings suggestive of pulmonary embolism. Pulmonary hypertension is another manifestation of septic pulmonary embolism that can occur in some patients. Autopsy analysis of nine patients revealed that vegetation extended into the pulmonary trunk and its branches in 67% of the patients explored, showing how common pulmonary events are in IPE [6, 19]. However, most patients are overlooked due to the subtle and nonspecific clinical signs of IPE, such as pulmonic regurgitant murmur, which may be auscultated in approximately 50% of the patients during the later stages of the disease [3, 6, 32–36]. We established our patient's diagnosis using the modified Duke criteria, recognized as the most specific and sensitive diagnostic tool for IE. The presence of a positive blood culture, along with evidence of endocardial involvement (vegetation) observed via echocardiography, fulfilled the major criteria, while fever exceeding 38 °C and septic pulmonary infarcts constituted the minor criteria, collectively confirming the diagnosis of IE.

IPE is commonly missed because of nonspecific clinical manifestations, while proper assessment of PV in echocardiography is demanding; therefore, sensitivity and specificity are two essential features of a diagnostic strategy that is capable of providing an early and accurate diagnosis. TTE, with a sensitivity of 30–63% and specificity of 91–100%, is the definitive diagnostic and follow-up imaging tool for IPE [3, 28]. TTE should be the initial imaging modality for all patients suspected of having infective endocarditis. Moreover, TTE should be repeated after completing antimicrobial therapy, considering that patients with previous IE are at a high risk of recurrent infection.

Regarding patient treatment and follow-up, RSIE typically presents a more favorable prognosis with lower mortality rates than LSIE and is often managed conservatively with medical therapy, yielding a mortality rate of less than 5%. According to several studies, surgical intervention is not commonly pursued in RSIE management, with only 5 to 16% of patients with right heart valvular involvement undergoing cardiac surgery as part of their treatment plan. However, when surgery is performed on an appropriately selected patient within the optimal timeframe, it has been shown to reduce both in-hospital and long-term mortality rates significantly. Surgical intervention in patients with complicated RSIE is typically warranted in the presence of concomitant LSIE, recurrent septic pulmonary emboli regardless of antibiotic treatment with or without concurrent right heart failure, empyema complicated by septic pulmonary emboli, large bronchopleural fistula observed on chest computed tomography as a complication of septic pulmonary embolism, vegetations exceeding 15 mm in a single direction over pulmonary valve on TTE, lack of response to medical therapy with sepsis persisting for more than seven days, [13, 34, 37–42]. Our patient had a large vegetation measuring 20×11 mm over the pulmonary valve, exhibited unresponsiveness to medical therapy and spiral chest CT scan results revealed the presence of septic pulmonary emboli, all of which necessitated cardiothoracic surgery.

Early surgery is defined as a surgical intervention performed on a patient with IE during their in-hospital stay following the diagnosis [43]. Suspicions of *S. aureus* bacteremia, fungal or other microorganisms resistant to medical treatment, the development of life-threatening complications including severe congestive heart failure, abscess formation or hypoxemia due to massive septic pulmonary emboli, very large vegetation (>15 mm), and persistent infection despite adequate antibiotic treatment without extracardiac focus suggest the need for performing early surgery in RSIE patients [38]. Early surgical intervention is correlated with a more favorable prognosis, as the intensity and duration of preceding antibiotic therapy have no significant impact on the overall clinical outcome. Furthermore, delaying surgery may lead to an increased perioperative risk, RSIE-related mortality, and persistent bacteremia due to the accumulating burden of pulmonary embolism [13, 37, 38, 43]. Because of persistent sepsis, large vegetation, and impaired gas exchange due to massive pulmonary emboli in the pulmonary trunk, our patient underwent early elective surgery within two weeks of diagnosis during his in-hospital stay.

Our surgical approach, in accordance with recommended methods, entailed radical debridement of the vegetation and all the infected tissue, followed by thorough irrigation and the replacement of the damaged pulmonary valve with a 21 mm homograft. Various studies recommend pulmonary valve repair/reconstruction as the most preferred surgical procedure for pulmonary valve treatment, considering the greater risk of reinfection of the prosthetic valve and worse survival in patients who undergo pulmonary valve replacement. If valve replacement is needed, there is no significant superiority of biological prosthetic or mechanical prosthetic valves over each other in terms of long-term survival, reoperation rate, 5-year valve failure, early mortality, or bleeding. Nevertheless, some studies show that thromboembolic events are strongly associated with mechanical valves requiring anticoagulants and thrombolytics [44, 45]. Based on the existing literature, in bioprostheses, homografts or stentless xenografts have the lowest rate of infection when pulmonary valve replacement is indispensable [13, 38, 46–48]. Pulmonary valve replacement was unavoidable in our patient due to the destruction of the valve and severe pulmonary insufficiency. In a study on patients with RSIE over 20 years by Musci et al., four surgeries were performed on patients with IPE, two of which involved pulmonary valve replacement with homografts, indicating how familiar and well-established homografts are in pulmonary valve replacement surgery [46]. Overall, postoperative outcomes of RSIE, with or without involvement of the left-sided valves, appear to provide good early, mid-term, and long-term results [13, 38, 46, 49].

Medical treatment of IE is initiated based on the causative microorganisms isolated in blood cultures. Before the blood culture results are prepared, empiric therapy such as vancomycin or daptomycin should be given for methicillin-resistant *S. aureus* [34]. Our patient was empirically treated with ceftriaxone and vancomycin. Ceftriaxone was replaced with meropenem once Acinetobacter spp. and gram-negative bacilli were isolated from the blood cultures. The patient was then continued on a carbapenem-aminoglycoside regimen until the end of his hospitalization. Acinetobacter spp. is susceptible to ceftriaxone and fluoroquinolones if the patient does not tolerate third or fourth-generation cephalosporins [28]. Some studies suggest that aminoglycosides and carbapenems can also be administered in Acinetobacter endocarditis [29]. By the prescribed duration of antibiotic therapy outlined in the existing guidelines, our patient received varying antibiotic regimens spanning a cumulative period of five weeks, both pre-and post-surgery [28].

Conclusion

The diagnostic challenges of IPE often lead to patients needing to be noticed and treated. Therefore, a comprehensive cardiopulmonary investigation should be pursued for any patient with recurrent fever, sepsis, and pulmonary symptoms, even if the patient has no common risk factors. The decision between early and late surgery and pulmonary valve repair or replacement in complicated IPE remains a significant challenge, as there are no definitive guidelines for providing a clear answer. Furthermore, the rarity of IPE as a clinical entity and the uncommon microbial pathogens causing it suggest further research in the future, particularly in patients with an unclear history of precipitating factors. Future reports should aim to include longitudinal follow-up data to assess the long-term efficacy of surgical interventions in pediatric IPE patients, especially those with complex conditions like Down syndrome.

Abbreviations

IPE	Isolated Pulmonary valve Endocarditis
PV	Pulmonary Valve
IE	Infective Endocarditis
TTE	Transthoracic Echocardiography
POD	Post-Operative Day
RSIE	Right-Sided Infective Endocarditis
LSIE	Left-Sided Infective Endocarditis
IDU	Intravenous Drug Use
CT	Computed Tomography
ICU	Intensive Care Unit
VSD	Ventricular Septal Defect
PDA	Patent Ductus Arteriosus
ABG	Arterial Blood Gas
NVE	Native Valve Endocarditis
DS	Down Syndrome

Supplementary Information

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Supplementary Material 1

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Not applicable.

Author contributions
MS performed the surgery on the patient and contributed to manuscript drafting. MF was the main supervisor of the patient, performed all physical examinations, and contributed to the literature review and data collection. SS contributed to manuscript drafting and revision and was responsible for intellectual data gathering and literature review. AB and BG reviewed the literature. FL performed the patient's echocardiography and collected data. FL also highly contributed to the drafting and revising of the manuscript for important intellectual content and reviewed the literature. All authors read and approved the final manuscript.

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Data availability
No datasets were generated or analysed during the current study.

Declarations

Ethics approval and consent to participate
Written informed consent was obtained from the patient's parents.

Consent for publication
Written informed consent was obtained from the patient's parents to publish this case report.

Competing interests
The authors declare no competing interests.

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