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Infective Endocarditis on Ventricular Septal Defect Complicated by Septic Pulmonary Emboli During Antibiotic Therapy: A Case Report and Literature Review

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Abstract | **Case Report**

Background: Infective endocarditis (IE) remains a serious complication of congenital heart disease, particularly ventricular septal defect (VSD), which predisposes to endothelial injury and vegetation formation. Septic pulmonary embolism is a feared complication that may occur despite appropriate antimicrobial therapy. ***Case Presentation:*** We report the case of a 20-year-old woman with a known perimembranous VSD admitted for prolonged fever. Blood cultures isolated Streptococcus mitis, and transthoracic echocardiography revealed mobile vegetations on the right ventricular aspect of the septal defect. Despite favorable initial clinical and biological evolution under appropriate antibiotic therapy, she developed bilateral septic pulmonary emboli on day 14 of treatment. After completing six weeks of antibiotics, she underwent surgical closure of the VSD with good outcome and no residual vegetations. ***Conclusion:*** IE complicating VSD may present with atypical features and may evolve toward embolic complications even under adequate treatment. Echocardiography plays a central role in diagnosis, monitoring, and detection of complications. Early recognition of embolic events is essential to guide timely surgical management.

Keywords: Infective endocarditis, ventricular septal defect, congenital heart disease, septic pulmonary embolism, echocardiography.

INTRODUCTION

Infective endocarditis (IE) is a life-threatening disease characterized by microbial infection of the endocardial surface, most frequently affecting cardiac valves but also involving congenital cardiac defects, prosthetic material, and intracardiac devices. Despite advances in antimicrobial therapy and imaging, IE remains associated with significant morbidity and mortality, particularly in young patients with congenital heart disease (CHD).

Ventricular septal defect (VSD) is one of the most common congenital heart malformations and represents the leading substrate for IE among CHD patients. The turbulent high-velocity jet through the defect causes endothelial injury, facilitating platelet-fibrin deposition and bacterial adherence during episodes of bacteremia. Although systemic embolization predominates in left-sided IE, right-sided IE or IE involving intracardiac shunts is more commonly associated with septic pulmonary embolism, which may complicate the disease course even during appropriate antimicrobial therapy.

We report a case of IE complicating a perimembranous VSD in a young adult, complicated by septic pulmonary emboli during antibiotic therapy, and provide a comprehensive review of the literature.

CASE PRESENTATION

A 20-year-old woman with a known congenital perimembranous ventricular septal defect followed since childhood, with no prior surgical correction and no relevant medical history, presented with a one-month history of prolonged unquantified fever associated with asthenia and malaise. She denied intravenous drug use, recent dental procedures, or invasive medical interventions.

On admission, the patient was febrile at 38°C, blood pressure was 120/77 mmHg, heart rate 100 bpm, and oxygen saturation 97% on room air. Cardiac auscultation revealed a grade 5/6 holosystolic murmur with thrill, best heard along the left sternal border and radiating toward the apex. The pulmonary and abdominal examinations were unremarkable, and there were no peripheral stigmata of endocarditis.

Electrocardiography showed sinus rhythm with incomplete right bundle branch block and secondary repolarization abnormalities. Laboratory investigations revealed elevated inflammatory markers with leukocytosis and elevated C-reactive protein. Two sets of blood cultures grew Streptococcus mitis, sensitive to penicillin and ceftriaxone.

Transthoracic echocardiography demonstrated a restrictive perimembranous VSD measuring approximately 7 mm with a left-to-right shunt (maximum velocity 7 m/s). Two mobile echogenic masses consistent with vegetations were visualized on the right ventricular aspect of the septal defect, the largest measuring 6 × 5 mm (Figures 1 and 2). No valvular involvement was detected, and ventricular function was preserved.

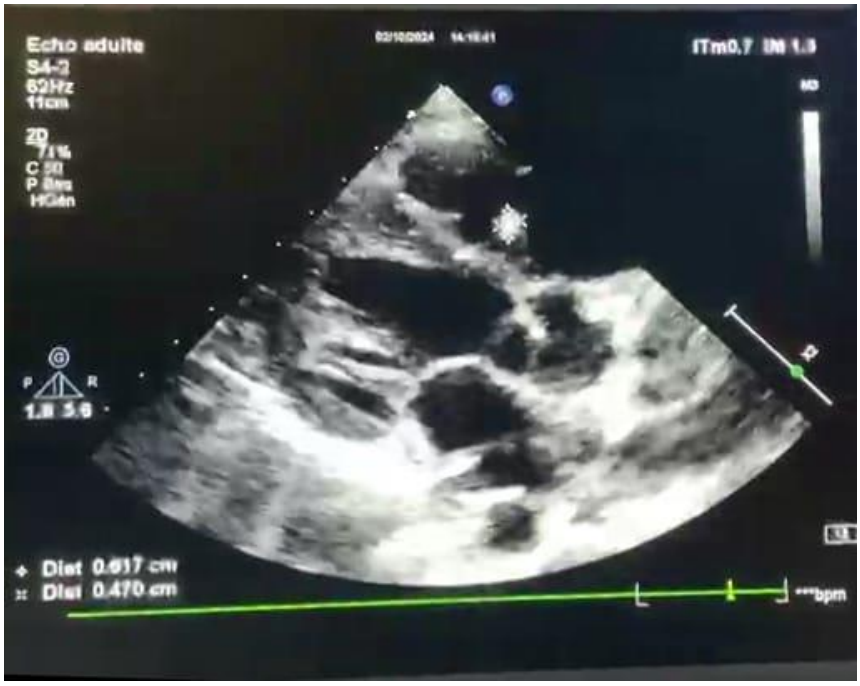

Figure 1: Parasternal long-axis transthoracic echocardiographic view showing a restrictive perimembranous ventricular septal defect with left-to-right shunt and a mobile vegetation on the right ventricular side

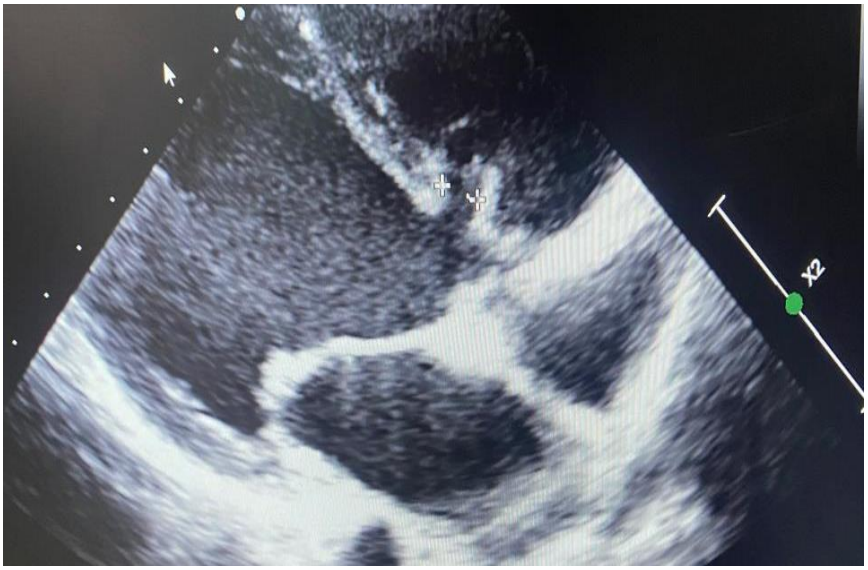

Figure 2: Zoomed parasternal view demonstrating two mobile echogenic masses attached to the free edge of the septal defect, the largest measuring 6 × 5 mm

Figure 1+2: parasternal long axis section on transthoracic echocardiography showing a perimembranous interventricular communication measuring 7 mm restrictive with a flow through the VSD at 7 m/s, shunting left - right with the presence of 2 mobile elements on the free edge of the interventricular communication on the side of the right ventricle, the largest measuring = 6 mm * 5 mm

The patient was started on intravenous beta-lactam therapy according to sensitivity testing, with close clinical and biological monitoring. She initially showed favorable evolution with defervescence and decreasing inflammatory markers. However, on day 14 of antibiotic therapy, she developed a new febrile episode associated with pleuritic chest pain and mild dyspnea. Laboratory tests showed a secondary rise in inflammatory markers.

Computed tomography pulmonary angiography revealed bilateral distal pulmonary emboli with features suggestive of septic embolization. No evidence of right ventricular failure was present. Antibiotic therapy was continued and adapted according to multidisciplinary discussion. After completing six weeks of intravenous antibiotics, repeat blood cultures were sterile, and follow-up echocardiography showed disappearance of vegetations.

Given the occurrence of embolic complications and the persistence of the anatomical substrate, the patient underwent surgical closure of the VSD with patch repair. Postoperative echocardiography confirmed complete closure without residual shunt or vegetations (Figure 3). The postoperative course was uneventful, and the patient remained asymptomatic at follow-up.

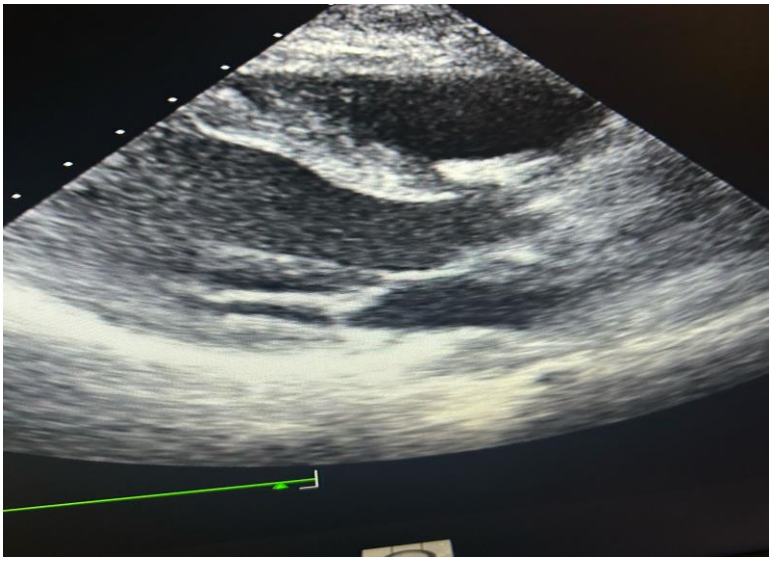

Figure 3: Postoperative parasternal long-axis echocardiographic view showing patch closure of the ventricular septal defect without residual shunt or vegetations

DISCUSSION

Infective endocarditis in patients with congenital heart disease represents a unique clinical challenge. VSD is the most frequently implicated lesion and confers a significantly increased risk of IE compared with the general population. The mechanism involves turbulent flow-induced endothelial injury on the right ventricular side of the septum, facilitating bacterial adherence during transient bacteremia.

Clinical presentation may be nonspecific, often limited to prolonged fever and constitutional symptoms, resulting in diagnostic delays. Blood cultures remain essential for etiological diagnosis, and viridans group streptococci remain among the most common pathogens in community-acquired IE associated with CHD.

Echocardiography plays a central role in diagnosis and monitoring. Transthoracic echocardiography is usually sufficient to detect vegetations in VSD-related IE, particularly when lesions are located on the right ventricular aspect of the defect.

Septic pulmonary embolism is a serious complication of right-sided IE or IE involving intracardiac shunts. It typically presents with fever recurrence, pleuritic chest pain, and respiratory symptoms and often occurs during the first two weeks of antibiotic therapy, when vegetations may fragment before complete bacterial sterilization.

Management relies on prolonged intravenous antibiotic therapy tailored to microbiological findings. Surgical intervention is indicated in cases of uncontrolled infection, embolic complications, or persistence of the anatomical substrate, as in our patient, where definitive closure of the VSD eliminated the underlying risk factor for recurrence.

CONCLUSION

Infective endocarditis complicating ventricular septal defect remains a significant clinical entity, particularly in young adults with unrepaired congenital heart disease. Presentation may be atypical, highlighting the importance of echocardiography and blood cultures in diagnosis and follow-up. Septic pulmonary embolism is a severe complication that may occur despite appropriate antimicrobial therapy and warrants early recognition and multidisciplinary management. Surgical closure of the defect after infection control provides definitive treatment and reduces recurrence risk.

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