Case Report

The Forgotten Valve: Pulmonary Valve Endocarditis in Late Pregnancy

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Infective endocarditis is a microbial infection of the cardiac endocardium, most often involving native or prosthetic valves. It commonly presents with persistent fever, malaise, anemia, and a new or changing murmur, along with possible cutaneous signs or systemic embolic features. The condition may progress to serious complications such as valvular regurgitation, heart failure, conduction abnormalities, stroke, and renal impairment. In this case report, we present a 21-year-old antenatal woman with an atrial septal defect who presented with fever and cough and was initially treated for community-acquired pneumonia.

Following an emergency caesarean section, the mother developed persistent fever despite the administration of antibiotics. Careful clinical examination prompted the evaluation of the heart, identifying a pulmonary valve pathology. Further investigations by obtaining blood cultures grew methicillin-resistant Staphylococcus aureus, which was then treated appropriately. The outcome was a successful recovery of the patient's usual state of health.

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Introduction

Infective endocarditis (IE) is an infection of the endocardial lining of the heart, predominantly affecting native or prosthetic valves, though it can also involve the mural endocardium, chordae tendineae, or intracardiac devices. It is characterized by the formation of vegetations composed of microorganisms, fibrin, and inflammatory cells. Population-based studies estimate the annual incidence of IE to range from 15 to 80 cases per million individuals [1] Right-sided IE accounts for approximately 5% to 10% of all IE cases and it is more often associated with intravenous drug use, intracardiac devices, and central venous catheters [2]

Isolated Pulmonary Valve Endocarditis (PVE) is extremely rare, affecting only about 1.5 to 2% of all IE cases.[3] Higher risk is seen in patients with underlying structural heart disease, previous rheumatic heart disease, congenital cardiac defects, prosthetic valves, prolonged intravascular access, intravenous drug use, or implanted cardiac devices. We report this case due to its rarity, native pulmonary valve endocarditis in an antenatal woman with an atrial septal defect, which was not detected on initial echocardiography but was identified through careful routine clinical examination.

Case Presentation

A 21-year-old antenatal female, who is a known ostium secundum atrial septal defect (ASD), which was diagnosed during her previous pregnancy, presented to the emergency department with chief complaints of intermittent fever for 2 days, associated with chills and rigor. The patient had a history of a dry cough for the past 1 day. On examination, the patient was conscious and oriented. Her vitals recorded a pulse rate of 110/minute, blood pressure of 100/60 mmHg, and a saturation of 96%

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Table 1: Laboratory investigations done at the time of presentation

Parameter	Value	Reference Value
Hemoglobin	9 g/dL	12–16 g/dL (F) / 13–17 g/dL (M)
Total Count (WBC)	10,100 /mm³	4,000–11,000 /mm³
Platelet Count	1,71,000 /mm³	1,50,000–4,50,000 /mm³
Serum Creatinine	0.7 mg/dL	0.6–1.2 mg/dL
Blood Urea	17 mg/dL	7–20 mg/dL
Serum Sodium	138 mEq/L	135–145 mEq/L
Serum Potassium	3.9 mEq/L	3.5–5.0 mEq/L
Random Blood Sugar	137 mg/dL	70–140 mg/dL

Table 2: Laboratory investigations done post-partum

Parameter	Value	Reference Value
C-Reactive Protein (CRP)	115.4 mg/L	< 5 mg/L
Erythrocyte Sedimentation Rate (ESR)	26 mm/hr	0–20 mm/hr
Total Count (WBC)	22,000 /mm³	4,000–11,000 /mm³
Hemoglobin	10.4 g/dL	12–16 g/dL (F) / 13–17 g/dL (M)
Malarial Parasite Test	Negative	Negative
Blood Culture	Negative	No growth
Urine Culture	Negative No growth	

in room air. Respiratory system examination revealed inspiratory crepitations in the right mammary region. The abdominal examination showed a term uterus, an unengaged fetal head, and a good fetal heart rate. Laboratory investigations revealed an anemic picture with slightly elevated WBC counts Table *1*. The patient was started on Intravenous fluids, Ringer's lactate in maintenance dose (100 ml/hr), along with injection Ceftriaxone 1g IV BD.

An echocardiogram showed an Atrial septal defect ostium secundum type of 24 mm Left to Right shunt. The Right Atrium and Right ventricle were dilated, with mild pulmonary hypertension (Tricuspid regurgitation pressure gradient (TRVG) of 30 mmHg), and the left ventricular Ejection fraction of 60%. On day 2 of admission, the patient's vitals recorded a Pulse rate of 110/minute, blood pressure of 114/80 mm Hg, but the Respiratory Rate was 30 breaths/minute. In view of oligohydramnios, the patient was taken for emergency

C-section and delivered a male baby of weight 2.5kg. Post C-section the patient was shifted to post antenatal care unit and a CT Chest was taken in view of desaturation which showed features of consolidation in the bilateral middle and lower lobes Figure 1. The Antibiotics were escalated to Injection Piperacillin-tazobactam 4.5 g IV QID, Tablet Azithromycin 500 mg BD, and Capsule Oseltamivir 75 mg BD were added. In view of persistent fever spikes, further investigations were done Table 2.

24 hours post-partum, the patient's saturation dropped to 93% in room air and during the cardiovascular system auscultation, an ejection systolic murmur of grade 3 was heard in the pulmonary area. A 3D echocardiogram was done which showed a slender vegetation of size 24*6 mm attached to the pulmonary valve Figure *2*. Three sets of blood cultures were sent according to the infective endocarditis protocol and the patient was started on Injection Vancomycin 1g IV BD. Two blood cultures came back positive for Methicillin-Resistant Staphylococcus Aureus (MRSA) sensitive to Vancomycin and Gentamicin. Thus a diagnosis of Infective endocarditis of the pulmonary valve was made based of Dukes 2023-The International Society for Cardiovascular Infectious Diseases (ISCVID) Criteria [4] The patient's antibiotics were changed according to the culture sensitivity. After day 5 of starting Vancomycin, the patient had no fever spikes, her total counts also reduced and the patient's condition improved. Her repeat cultures came out to be negative and she was discharged after 6 weeks of Antibiotic therapy.

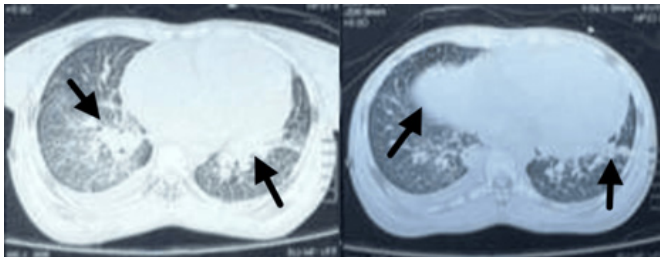

Figure 1: CT Chest of the patient. (Left) The arrows depict consolidation of the middle lobe of the right lung and the consolidation of the lower lobe of the left lung. (Right) The arrows depict consolidation of the both lower lobes.

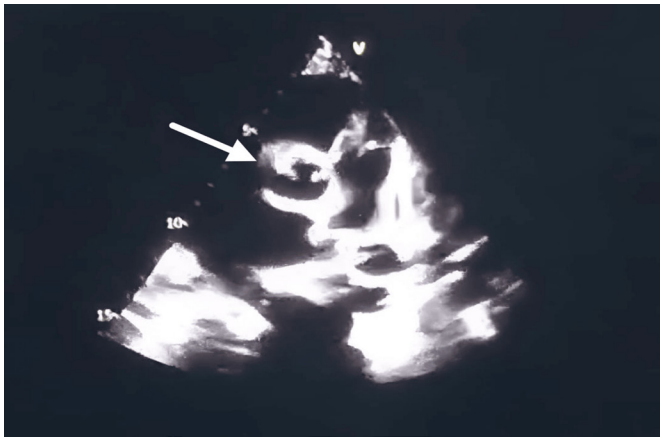

Figure 2: Parasternal short axis view with a focus on the pulmonary valve revealing a vegetation

Discussion

Infective endocarditis is an inflammatory condition involving the endocardial lining of the heart and the valvular structures separating its four chambers. Most often caused by bacterial organisms, it presents with varied clinical manifestations and potential complications. Failure to recognize and treat the condition promptly can result in significant intracardiac damage as well as systemic complications [5] Therefore, thorough clinical evaluation, including detailed history-taking and physical examination, is crucial for early diagnosis, appropriate management, and reduction of related morbidity and mortality.

Many species of bacteria and fungi cause sporadic episodes of IE although the predominant etiologic agents in infective endocarditis are gram-positive organisms such as streptococci, staphylococci, and enterococci, which collectively account for 80-90% of cases. Staphylococcus aureus was found to be the most common microorganism, being the most frequent. Viridans group streptococci (VGS), coagulase-negative staphylococci (CoNS), Enterococcus species are amongst the most common causes.[5] Apart from various streptococcal species, less common causes include oropharyngeal commensals belonging to the HACEK group (Haemophilus, Actinobacillus, Cardiobacterium, Eikenella, and Kingella species). Culture-negative organisms account for approximately 20% of cases [6], while fungal endocarditis represents nearly 1% and is associated with significant mortality, particularly in immunocompromised patients with disseminated Candida or Aspergillus infections.[7]

Multiple clinical studies report ventricular septal defect (VSD) as the most common congenital heart disease associated with infective endocarditis, especially in patients with unrepaired or residual shunts. In one retrospective cohort of 45 patients with congenital heart disease and infective endocarditis, 31% had VSD, making it the most frequent underlying lesion in that series.[8] A key mechanism predisposing to infective endocarditis is endothelial injury resulting from high-velocity turbulent blood flow. Such turbulence damages the endocardium, leading to platelet and fibrin deposition that provides a surface for bacterial adherence during episodes of transient bacteremia [9] In ASD, the left-to-right shunt occurs between two low-pressure atrial chambers and does not produce high-velocity jets or significant turbulence across valves or endocardial surfaces, unlike lesions such as ventricular septal defect (VSD) or patent ductus arteriosus (PDA) [9]

The relatively low turbulence and limited endothelial injury decrease the likelihood of bacterial colonization and vegetation formation. Traditional surgical and epidemiological studies commonly identify ventricular septal defects and other high shear stress lesions as frequent congenital heart diseases associated with infective endocarditis, whereas uncomplicated atrial septal defects are rarely implicated [10] Although ASD itself is a low risk condition, certain situations may increase the chance of IE in ASD patients such as associated other valve lesions like mitral regurgitation which would introduce turbulence and predispose to infective endocarditis [11], Intravenous drug use or indwelling devices/catheters create sustained bacteremia that can lead to the seeding of endocardial surface[12] Post-device closures particularly early post-implant can also rarely be a substrate for IE because of incomplete endothelialisation around the device.

The mitral valve followed by the aortic valve (AV) is the most commonly affected in IE. Right-sided IE overall is less common than left-sided IE amongst the right -sided valves the tricuspid valve predominates leaving the pulmonary valve as the least frequent site .[13] Pulmonary valve endocarditis is an uncommon manifestation of infective endocarditis; however, its occurrence may be relatively increased in the presence of certain risk factors, including intravascular devices, immunocompromised states, and congenital abnormalities involving the right ventricular outflow tract (RVOT) or pulmonary valve. Prosthetic valve replacements, particularly transcatheter pulmonary valves, can lead to residual gradients and altered hemodynamics following repair. These factors predispose to bacteremia, endothelial injury, or colonization of prosthetic material, thereby facilitating infection of the pulmonary valve [14]

Pulmonary valve infective endocarditis (PV IE) is an uncommon entity and is frequently recognized late, contributing to its comparatively higher mortality. Early manifestations such as low-grade fever, malaise, or respiratory symptoms are often nonspecific, and classic peripheral signs of endocarditis may be absent.

Owing to the anterior and superior location of the pulmonary valve and the low-pressure, low-velocity flow across it, vegetations are often difficult to visualize on routine transthoracic echocardiography, leading to delayed diagnosis. Predisposing factors include intravenous drug use, central venous catheters, congenital abnormalities of the right ventricular outflow tract, and prosthetic or transcatheter pulmonary valves. Late detection permits virulent organisms such as Staphylococcus aureus to progress, resulting in septic pulmonary emboli, persistent bacteremia, and right-sided heart failure; moreover, the technical complexity of surgical management further adds to the associated morbidity and mortality.[15]

The transthoracic echo can miss around 20% of early vegetations. The probable cause for infective endocarditis in the above-mentioned case would be a pneumonia leading to bacteremia and infective endocarditis. The diagnosis is based on Duke's ISCVID 2023 Criteria.[4] The treatment for native pulmonary valve endocarditis is based on the culture sensitivity findings for 6-8 weeks starting from the first negative blood culture.[4]

Conclusions

This case highlights the rare occurrence of isolated pulmonary valve infective endocarditis in a patient with an atrial septal defect, emphasising the importance of meticulous clinical examination in detecting uncommon cardiac complications. Pulmonary valve involvement in infective endocarditis is fairly uncommon, and association with congenital heart disease such as ASD further underscores its rarity.

Careful auscultation and prompt echocardiographic evaluation enabled early diagnosis in our patient, preventing potential complications such as septic embolism and right-sided heart failure. This report reinforces that even in the era of advanced imaging, thorough bedside clinical assessment remains pivotal in identifying rare presentations of infective endocarditis and guiding timely management.

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work. Concept and design: Yogesh Subramanian, Preeti Nandakumar, K R Murugan, S Sureya Vayshnava Kumar, S Shivamalarvizhi, Keerthi K, Sahasyaa Adalarasan, Jayaprakash N, Hariharan C Acquisition, analysis, or interpretation of data: Yogesh Subramanian, Preeti Nandakumar, K R Murugan, S Sureya Vayshnava Kumar, S Shivamalarvizhi, Keerthi K, Sahasyaa Adalarasan, Jayaprakash N, Hariharan C Drafting of the manuscript: Yogesh Subramanian, Preeti Nandakumar, K R Murugan, S Sureya Vayshnava Kumar, S Shivamalarvizhi, Keerthi K, Sahasyaa Adalarasan, Jayaprakash N, Hariharan C Critical review of the manuscript for important intellectual content: Yogesh Subramanian, Preeti Nandakumar, K R Murugan, S Sureya Vayshnava Kumar, S Shivamalarvizhi, Keerthi K, Sahasyaa Adalarasan, Jayaprakash N, Hariharan C Supervision: Yogesh Subramanian, Preeti Nandakumar, K R Murugan, S Sureya Vayshnava Kumar, S Shivamalarvizhi, Keerthi K, Sahasyaa Adalarasan, Jayaprakash N, Hariharan C

Human subjects

Informed consent for treatment and open access publication was obtained or waived by all participants in this study.

Conflicts of interest

In compliance with the ICMJE uniform disclosure form, all authors declare the following:

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All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work.

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