

Case Report

Dual Valve Destructive Fungal Endocarditis.

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Abstract

Fungal endocarditis is rare and highly lethal, particularly in immunocompromised patients. We report a 36-year-old woman with advanced HIV infection (CD4 45 cells/mm³) who presented with progressive heart failure following treatment for presumed pneumonia and tuberculosis. Transthoracic and transoesophageal echocardiography demonstrated large vegetations on both the aortic and pulmonary valves, severe regurgitation, and an aortic root-to-right ventricular fistula. Blood cultures remained negative. Urgent surgery with metallic aortic and bioprosthetic pulmonary valve replacement was performed. Valve cultures subsequently grew *Aspergillus fumigatus*. Despite intensive antifungal therapy, the patient died post-operatively from methicillin-resistant *Staphylococcus aureus* infection, highlighting the aggressive course of dual-valve disease.

A 36 year old, HIV positive female patient presented to a peripheral hospital three months prior with a 2week history of respiratory symptoms. She was admitted and treated for pneumonia there. Due to her lack of clinical response, she was started on empiric anti-tuberculosis treatment despite having negative sputum for tuberculosis (TB). Anti retroviral therapy was subsequently started with a baseline CD4 count of 45 cells/mm³. A month post-discharge from hospital, she noticed her legs began to swell and complained that it was becoming more difficult to breath.

On arrival to her referral hospital, she had signs and symptoms of left heart failure. She had a displaced volume loaded apex with peripheral signs of severe aortic regurgitation. Her auscultatory findings were that of a to-and-fro, machinery murmur at the base of the heart. At the time, she was afebrile and had no clinical features of acute infective endocarditis (IE). Her inflammatory markers were not elevated and blood cultures (including fungal) were all negative. Her transthoracic echocardiographic findings showed a thickened rheumatic aortic valve with a large (1.67 x 0.47cm) right coronary cusp vegetation resulting in severe aortic regurgitation. The parasternal short axis view revealed a similar sized pulmonary valve vegetation (1.79 x 1.12cm) with severe pulmonary regurgitation.

In search of an explanation for both side endocarditis, a trans-esophageal echocardiograph was performed. A

communication between the aortic root and right ventricle was made. This was confirmed in the catheterization-lab, where a pigtail catheter was placed above the aortic valve and contrast injected here was noted to track down into the right ventricle. The presumptive diagnosis of chronic infective endocarditis with a sinus-of-valsava fistula was made. It is remarkable that such destructive endocarditis was silent for so long. It would seem that this is partly due to the antibiotics she received previously as well as the anti-tuberculosis medication. Due to her refractory heart failure, she was sent for surgery where a metallic aortic valve replacement was performed along with a bio-prosthetic pulmonary valve replacement. Post-operatively she was unstable, requiring inotropic support and then developed Systemic Inflammatory Response Syndrome (SIRS). Her native valve cultures came back with *Aspergillus fumigatus*. Despite giving her goal directed therapy, our patient never really showed clinical response and demised 10 days post valve replacement from a newly acquired methicillin resistant *Staphylococcus aureus* infection.

The precise incidence of IE is difficult to ascertain because case definitions have varied over time between authors and between clinical centers. HIV-infection itself places one at a higher risk of developing IE, with reports in the literature of patients with a CD4 cell count < 50 cells/mm³ and high viral loads (>100 000copies/ml) having a 4-fold increased risk of developing IE[1]. Both side endocarditis is far less common

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and is predominantly described in the congenital heart disease group with an incidence of 2.3% [2].

The incidence of fungal endocarditis has been reported to comprise of 1-10% of all IE cases where *Candida* accounts for almost half of fungal cases and *Aspergillus* is the second most common with approximately 25% [3]. Diagnosing this condition proves a challenge with a clinical dilemma as only 50% of blood cultures are positive for *Candidiasis* and is hardly ever positive for *Aspergillus* species[4]. A review of the literature by Kalokhe et al., shows that fungal endocarditis predominately affects the left side of the heart (72%) with right sided endocarditis accounting for 19%. There were no reports of both sided endocarditis in this review[5].

Mortality of fungal endocarditis is more than 50% and treatment requires antifungal therapy and surgical valve replacement[6]. The recommended antifungal therapy

for invasive *Aspergillus* infections is voriconazole. The superiority of voriconazole to amphotericin B deoxycholate was demonstrated in a large, randomized controlled trial of invasive *Aspergillus* infections[7]. Compared to amphotericin B, voriconazole was associated with improved survival, and less nephrotoxicity, electrolyte abnormalities, and infusion-related event [7]. Suppressive long term therapy with oral azoles is recommended for life once fungal IE is acquired [8]. In our patient, it is possible that her fungemia was kept at bay by the Rifampacin she was taking for her mis-diagnosed TB. Interestingly, Rifampacin has not only shown to have some antifungal activity but also offers synergistic effects to Amphotericin B thereby becoming fungicidal rather than only being fungistatic[9]. Our case is a unique one and highlights the aggressive nature of fungal endocarditis.

Figure A. Trans-esophageal echocardiographic picture showing the large aortic valve vegetation.

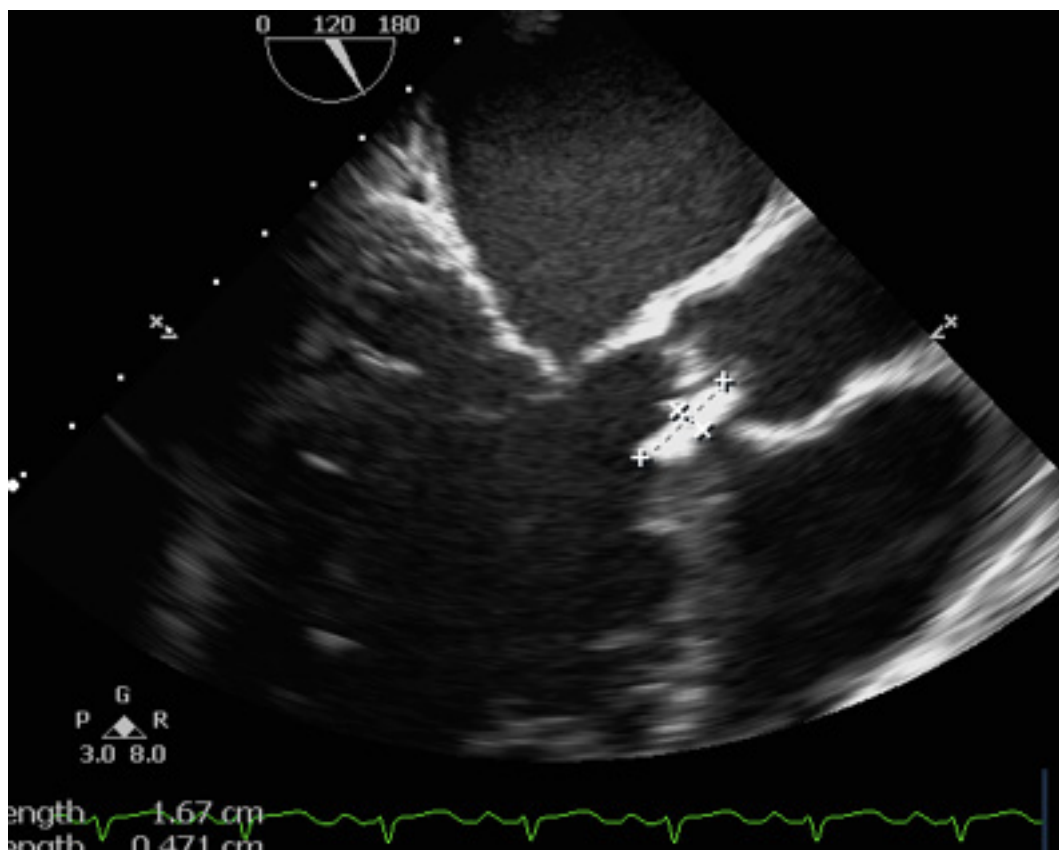


Figure B. Trans-esophageal echocardiographic picture depicting the large pulmonary valve vegetation in relation to the aortic valve.

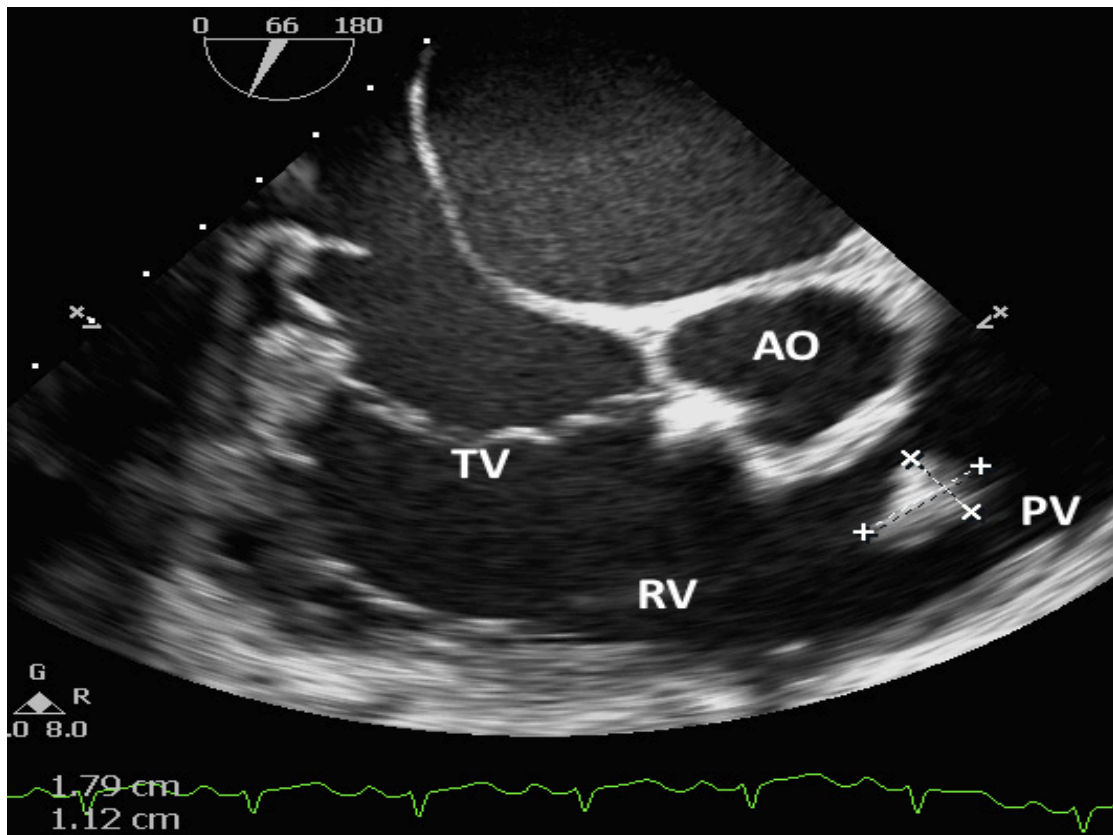
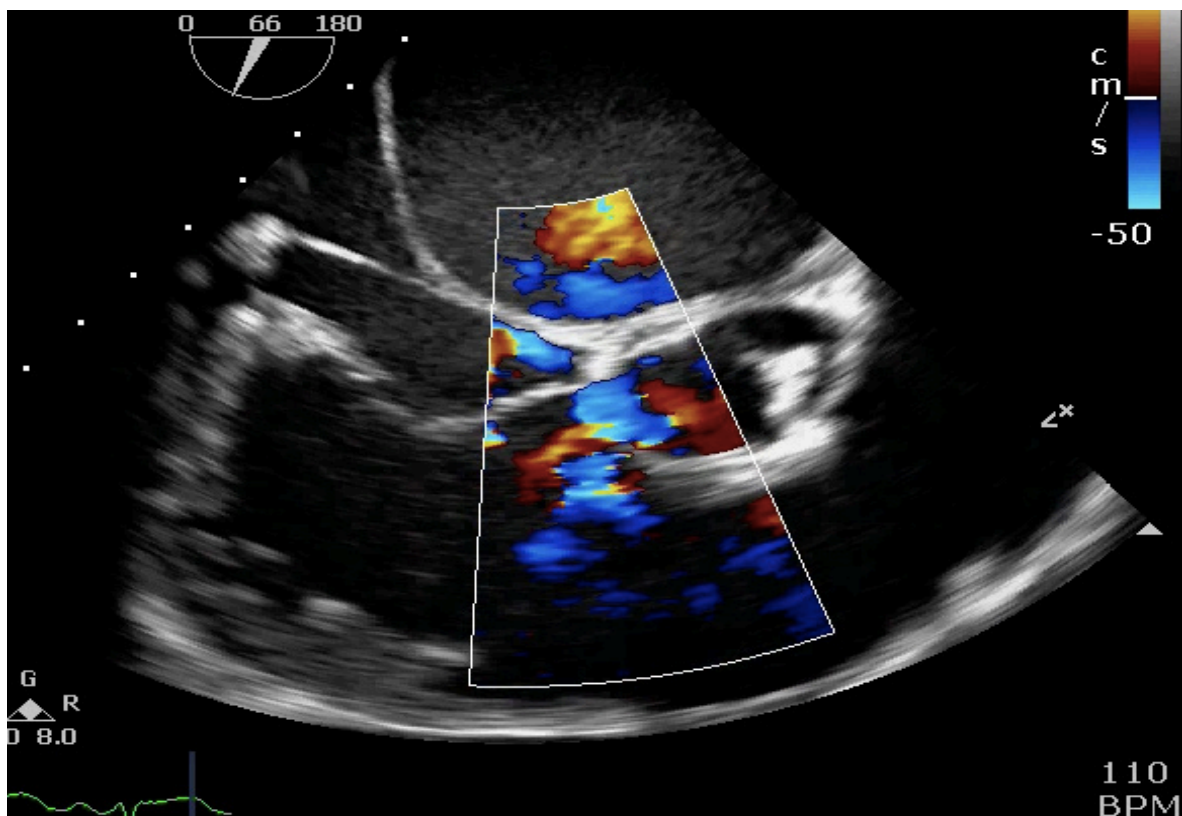


Figure C. Trans-esophageal echocardiographic picture showing the communication from the aortic root to the right ventricle .



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