***Abiotrophia defectiva* Infective Endocarditis in Hypertrophic Obstructive Cardiomyopathy**

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Abstract: A 74-year-old male with hypertrophic obstructive cardiomyopathy (HOCM), moderate-severe mitral regurgitation, and CKD3a presented with six weeks of fevers, chills, and malaise. *Abiotrophia defectiva (A. defectiva)* grew in 2/2 sets of blood cultures. He had no peripheral stigmata of infective endocarditis (IE), and transthoracic echocardiogram (TTE) showed no vegetations. Recent dental work suggested an odontogenic source. He received seven days of ceftriaxone with culture clearance, followed by outpatient amoxicillin-clavulanate. Seven months later, after further dental work, he presented to an outside hospital with two weeks of dyspnea and fatigue. Labs showed leukocytosis (13,000/µL) and a creatinine of 3.9 mg/dL (baseline 1.8 mg/dL). Blood cultures were again positive for *A. defectiva*, and TTE was negative for vegetations. Upon transfer to our hospital, ceftriaxone was started based on susceptibilities. Given clinical deterioration, TTE was repeated and revealed an echodensity on the mitral valve (Figure 1), which was confirmed on transesophageal echocardiogram (TEE). The patient developed hemolytic anemia and glomerulonephritis requiring hemodialysis. A multidisciplinary review found no indication for urgent valve surgery. He was discharged on six weeks of vancomycin after dental extractions for source control. His IE resolved without surgery. This case highlights the importance of sustained clinical suspicion in evaluating IE, particularly when an uncommon yet virulent organism grows in the setting of structural heart disease. To our knowledge, this is also the first reported case of *A. defectiva* IE in HOCM. *A. defectiva* is a rare cause of IE (1-2% of cases), but carries high rates of progression from bacteremia to IE. This risk was compounded in our patient by HOCM, atrial enlargement, valvular regurgitation, and ongoing dental procedures. *A. defectiva* frequently demonstrates reduced penicillin susceptibility, with high rates of embolization and valvular destruction often attributed to diagnostic delay. Here, TTE was negative for vegetations during the initial presentation and early during the second. Continued evaluation for IE was therefore guided by ongoing clinical suspicion, recognizing TTE's limited sensitivity (50-90%) and patient deterioration in the presence of multiple pathogen- and host-related risk factors. Interestingly, his recurrent bacteremia with a penicillin-resistant organism represents a scenario where first-line IE prophylaxis, if indicated, would have been ineffective. This case reinforces guidelines calling for individualized evaluation and a low threshold to repeat TTE and escalate to TEE when clinical context renders post-test probability for IE high despite negative initial testing.

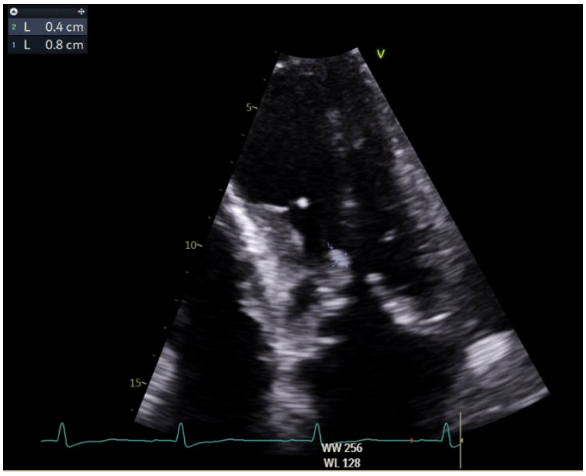

Figure 1. TTE.