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Case Report

Abiotrophica Defectiva-Causing Endocarditis in a TAVR Prosthetic Patient: Hidden Inflammation and Slow Clinical Progression

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Abstract

Introduction: Among streptococcus species, *Abiotrophia defectiva (A. Defectiva)* is a subspecies with strict growth requirements and is specifically designated as a Nutritional Variant Streptococcus (NVS). The special growth factors utilized by these bacteria are for cell wall synthesis and replication. These bacteria are normal commensals of the oral, gastrointestinal, and genitourinary tract. Against the backdrop of specific risk factors, these bacteria can enter the bloodstream, thereby opening the door to systemic infections, including endocarditis.

Clinical case: 75-year-old female with a PMH of AFIB [Atrial Fibrillation], atrial flutter, TAVR [Transaortic valve replacement], HTN [Hypertension], CVA [Cerebrovascular accident], and HLD [Hyperlipidemia] presented with left MCA [Middle Cerebral Artery Occlusion] occlusion, which was successfully treated with embolectomy. She was eventually discharged but returned 1 week later with shortness of breath. Evaluation revealed elevated troponin levels and pulmonary edema, necessitating a cardiology consult for possible cardiac catheterization. Routine workups, including bacterial cultures, revealed bacteremia. Echocardiography was considered because of bacteremia in the context of TAVR. The TEE showed large mobile vegetations in the aortic valve, the largest being 1.5x1.0cm. Repeated blood culture later grew *A. defectiva,* thus confirming the diagnosis of NVS provoked endocarditis. Considering the infection with *A. Defectiva,* IV [intravenous] Amipicillin-Sulbactam for 6 weeks, was recommended by the Infectious Disease Team. Cardiothoracic surgery was also consulted for possible surgical intervention. Long-term antibiotic clinical suppression is recommended given the high relapse rate seen in these cases.

Conclusion: By presenting this clinical case, we would like to emphasize the clinical relevance of *A. Defectiva*-induced endocarditis in patients with relevant risk factors. Well-honed clinical intuition and a high degree of preconception are almost always necessary for early diagnosis of this rare endocarditis. A delayed diagnosis is likely, given the strict growth requirements and the protracted clinical course. Despite antibiotic administration, a successful therapeutic response is stonewalled by antibiotic resistance and a higher relapse rate. On grounds of this, rapid clinical progression, and valvular complications command surgical management, thereby triggering amplified mortality and morbidity.

Keywords: Nutritionally variant streptococci; Heart disease; Endocarditis; Vegetations; Septic embolism; Stroke; Osteomyelitis; Brain abscesses

Abbreviations: *A. Defectiva: Abiotrophia Defectiva;* NVS: Nutritionally Variant Streptococcus; PMH: Past Medical History; AFIB: Atrial Fibrillation; TAVR: Transaortic Valve Replacement; HTN: Hypertension; CVA: Cerebrovascular Accident; HLD: Hyperlipidemia; MCA: Middle Cerebral Artery; TEE: Transesophageal Echocardiography; NSTEMI: Non-ST Elevation Myocardial Infarction; EF: Ejection Fraction; AI: Aortic Incompetence; IV: Intravenous; 16S rRNA: 16 Svedberg Units Ribosomal Ribonucleic Acid; B6: Pyridoxine; IE: Infective Endocarditis; MALTI-TOF-MS: Matrix-Assisted Laser Desorption Ionization-Time of Flight Mass Spectrometry; mNGS: Megagenomic Next-Generation Sequencing; PCR-RFL: Polymerase Chain Reaction-Restriction Length Polymorphism; Cath Lab: Cardiac Catheterization Laboratory

Introduction

A. defectiva was originally classified as Nutritionally Variant Streptococci (NVS). The endocarditis induced by *A.Defectiva* accounts for roughly for 4.3-6% of cases of streptococcus endocarditis and 1-2% of all infective endocarditis cases [1,2]. On the account of harboring special moonlighting proteins, *A. Defectiva* has the unique potential to forestall host cellular immunity, impair cellular hoemostasis mechanisms and amplify pro-inflammatory pathways, hence greasing the wheels for rapid multiplication, bacteremia and valvular infection [3]. As the infection flourishes, there is high incidence of peri-annular complications in *A. defectiva*

induced endocarditis [1]. The clinical presentation can be as non-specific as anemia, weight loss and stroke [2]. Scrupulous growth requirements and protracted clinical courses can mask the smoldering infection in the early stages, thus bringing about negative blood cultures. Therefore, a high index of clinical suspicion and transparent communication with the microbiologist is imperative particularly in patients with pertinent risk factors like congenital heart disease and valve replacement [2].

Here, we present this clinical case to of rare form of endocarditis triggered by *Abiotrophia Defectiva,* a nutritional variant of streptococci in a patient with trans-aortic valve replacement. The patient was initially presented with an acute occlusion of the left MCA, which was successfully treated and discharged. But he later presented within 1 week with shortness of breath. Further workup revealed, pulmonary edema, high troponin levels and bacteremia which prompted echocardiography revealing large mobile vegetations in the aortic valve. Treatment with antibiotics along with placement on long term antibiotic regimen was recommended for reducing recurrence as well as improving mortality & morbidity.

Clinical Case

75-year-old male with past medical history significant for paroxysmal A-fib, paroxysmal atrial flutter (s/p successful atrial ablation on 01/05/2023), aortic stenosis s/p TAVR, HTN, CVA, & HLD. Patient was admitted on 09/10/2024 & was seen by a neuro-interventionalist and taken to the Cath Lab [Cardiac Catheterization Laboratory] on 09/10/2024, where he was found to have an acute occlusion of the left MCA trifurcation at the distal M1 level. A successful embolectomy was performed, achieving complete reperfusion. He was discharged on 09/12/2024 on a regimen of Amiodarone, Apixaban, Aspirin, & Statin for management of atrial fibrillation and stroke prevention. Initial evaluation revealed severe pulmonary edema on chest X-ray and highly elevated cardiac enzymes (Troponin: 3200), indicating a likely non-ST-segment elevation myocardial infarction (NSTEMI). The patient denied chest pain but was noted to be uncomfortable at home. Cardiology has been consulted, and he is being considered potential cardiac catheterization. He was on anticoagulation therapy, started recently after his stroke. He has been admitted to the ICU for further management. Infectious disease consulted, blood cultures grew Abiotrophia defectiva, and the patient was initiated on IV Unasyn 3g IV every 6. Cardiology recommended an echocardiogram, which showed severely reduced LV function and an estimated EF of 25 to 30%. Patient was placed on Lasix IV 40 IV daily, with monitoring of renal functions and input output chart. In view of bacteremia and a history of TAVR for aortic stenosis, the patient was planned for TEE. The TEE showed multiple vegetations on the aortic valve, and a prosthetic TAVR valve in place. Mobile vegetation was detected (Figure 1). The largest vegetation measured 1.5x1.0cm. Severe aortic stenosis, mild AI, and a peak gradient of 64 mmHg were also documented. Cardiothoracic surgery was consulted for surgical intervention, but they advised no active intervention at this moment. Infectious disease consultant, and they recommended long-term IV antibiotics with IV Ampicillin-Sulbactam 3g Q6 for 6 weeks, followed by lifelong oral antibiotic suppression.

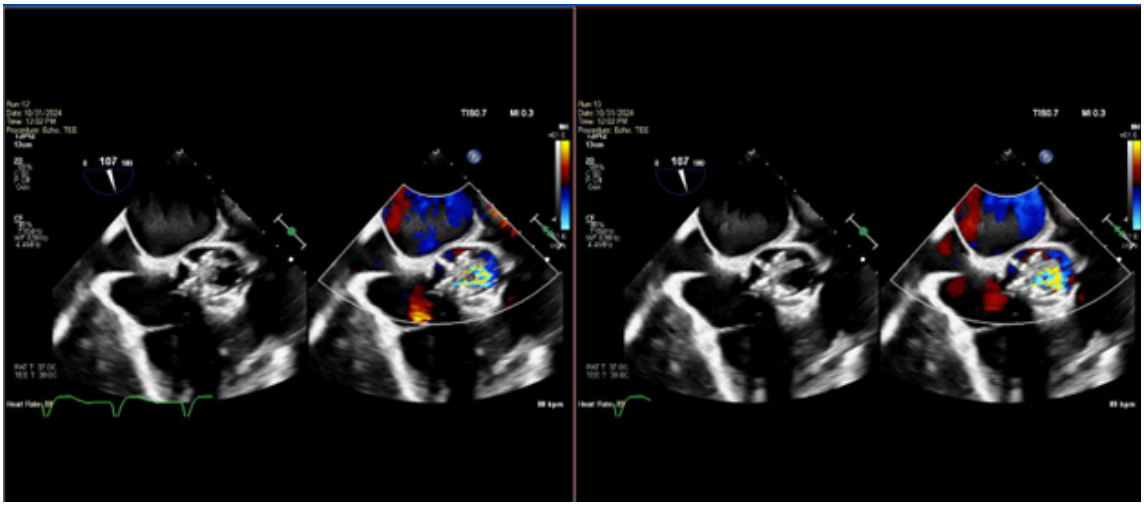

Figure 1: Transoesophageal Echocardiography (TEE): TEE showing large and mobile vegetation in the aortic valve. Largest 1.5x1.0cm. Mild AI. Severe aortic stenosis, peak gradient 64mmHg.

Discussion

We present an unusual case of infective endocarditis with a rare form of nutritionally variant streptococci *[Abiotrophia Defectiva]* in patient with TAVR and pertinent risk factors including paroxysmal A-fib, paroxysmal atrial flutter aortic stenosis, HTN, CVA, & HLD. Cardiothoracic surgery is consulted for surgical intervention, but ultimately patient was given IV antibiotics and recommended long-term antibiotic regimen.

A. defectiva are normal colonization bacteria of the human body, with their presence primarily localized to oral mucosa, gastrointestinal tract and genitourinary tract [4]. This NVS was subsequently reclassified as *Abiotrophia defectiva,* through 16S rRNA gene sequencing as they do not belong to streptococcus genus. There were originally discovered by Frankel & Hirsh in 1961 [5]. In their laboratory, colonies of non-hemolytic streptococci isolated from blood cultures of infective endocarditis and otitis

media were more inclined to grow as satellite colonies adjoining other auxiliary bacteria [5]. These satellite colonies have a propensity to enlarge in the outer layer within 48h as they absorb nutrients from the surrounding medium. Gram-positive bacteria, gram negative bacteria as well as yeast transude nutrients such as pyridoxine and cysteine into the medium that are ultimately taken up and assimilated by these *A. defectiva* [4]

Pertinently, most characteristic feature of these bacteria is their nutritional dependency on sulfhydryl compounds as well as some bacterial secretions [5]. Specifically, these bacteria need L-cysteine, Thio glycate, reduced glutathione, thiomalate, pyridoxine [B6] and other such factors for its proper growth [4]. Addition of blood enhances the recovery of NVS due to presence of pyridoxine in the erythrocytes. These nutritional factors are most probably used as building blocks for assembling the cell wall of these NVS [5]. If these nutritional compounds are not accessible, then are more liable to transform into L-shaped colonies (thickened and filamentous structure), due to abnormal development of cell [5]. These L-shaped colonies assume a fried egg appearance when present in an osmotically supportive medium and are usually resistant to cell wall lytic antibiotics. Morphologically in the blood cultures, there are more likely to be more gram variable and, in some instances, mimicking the appearance of fungal colonies [4]. These are non-motile, non-sporulating and facultative anaerobes that are usually difficult to be grown in the blood, unless blood cultures are spiked with pyridoxine or cysteine [6].

Reports suggest that, patients with predisposing factors including pre-existing heart disease, previous cardiac surgery and valvular heart disease are liable to be afflicted with A Defectiva endocarditis with [7]. Other relevant risk factors that provoke the causuation can range from immunosuppression, pregnancy, oral cavity infection (tonsillitis or pharyngitis), poor dental hygiene to recent dental procedures [8].

With NVS being localized to the oral cavity, dental caries or dental procedure will instigate and thrust these bacteria into the blood steam. The virulence of *A. defectiva* in triggering endocarditis can materlize secondary to multifarious factors. Disordered cellular morphology due to nutritional deficiency and exopolysaccharide production are the priming events that have a cascading effect for initiating the onset of endocarditis. Biochemically, exopolysaccharides secreted into the blood circulation are more accustomed to cling to the fibronectin of the extracellular matrix, thence underscoring their affinity towards vascular free collagen in cornea, joints and heart valves [9].

These aforementioned factors potentiate strong binding of bacterial colonies to the valvular interface, thus triggering a chain of events for transpiring endocarditis. Regardless of the sluggish clinical course and smaller valvular vegetations, endocarditis that emanates culminates into severe complications. On the grounds of this, unfolding of congestive heart failure, septic embolism, destruction of valves, valve incompetence, valve surgery during the clinical course of endocarditis are not uncommon developments [10]. Reports suggest that, children are more likely have systemic embolism (69%) as compared to adults [1].

Unravelling of these complications inflates the mortality rate to 17%, much higher than that is encountered in the streptococcal endocarditis. Apart from endocarditis, *A defectiva* can is known to be associated with other infections including osteomyelitis, meningitis, cerebral brain abscess, implantable cardioverter defibrillator lead infection, mycotic aneurysm, endophthalmitis, and septic arthritis [11].

In earlier case reports, the subacute *A. defectiva* IE [Infective Endocarditis] cases were most frequently reported due to their indolent clinical course. The clinical presentation of *A. defectiva* induced endocarditis can from mitral valve regurgitation, bleeding, sepsis, new onset heart failure to mycotic aneurysms [12]. As compared to viridans streptococcal group, endocarditis triggered by NVS is inclined to have lower rate of intravenous abuse, higher incidence of peri annular complications, greater propencity for surgical intervention, and increased mortality rate [1]. Aortic valve and mitral valve are equivalently effected by this bacterium, although some reports report preferential involvement of aortic valve [7]. In few instances, patient also presented with stroke and cerebral hemorrhage, a culmination that unfolds secondary to septic emboli, hemorrhage from cerebral infarction, rupture of pyogenic arteries as well as mycotic aneursyms [13].

This procastinated clinical trajectory in collusion with finicky growth requirements culminates into false negative blood cultures, thence forestalling the earlier diagnosis and subsequent treatment commencement. Therefore, a high index of clinical suspicion and early diagnosis is inescapable and become crucial in clinical practice for bringing about reasonable clinical outcomes. Identification of this organism entails utilization of 16 RNA gene amplification followed by PCR-RFL [Polymerase chain reaction-restriction length] polymorphism [11]. It is important keep in mind that in cases with culture negative endocarditis, Megagenomic Next-Generation Sequencing (mNGS) was deemed to more sensitive and invaluable for conforming the diagnosis [14]. A rather unique technique to quickly identify NVS entails the utilization of Matrix-assisted laser desorption ionization–time of flight mass spectrometry (MALTI-TOF-MS) [15]. 3-4 blood cultures over a period of 24 h, preferentially before the initiation of antibiotics would be a best possible strategy to unearth these organisms [16]. Possibly, in most instances gram staining might reveal streptococcal like organisms, but these organisms are inclined to show dawdling growth, unless specific growth factors are supplemented [17].

In patients suspected with endocarditis with *A. defectiva,* American College of Cardiology recommends initiating treatment with penicillin/vancomycin and gentamycin for 4-6 weeks [18]. The resistance rate of penicillin and macrolides is 53% and 90% respectively [7]. Even with prompt therapy with antibiotics, failure rate is as high as 41% [7]. As much as we can rely on the clinical potency of these antibiotics, it is a miscalculation to believe that a favorable therapeutic response is habitual state of affairs. Having said that, interplay of multifarious factors including, scrupulous growth pattern, special growth factor requirement, protracted multiplication time, and resistance to pencillins are inclined to disrupt the efficient targeting and dismantling of these atypical

bacterial colonies that are drifting around in the blood [10]. That being the case, treatment failure rate in *A. Defectiva* induced endocarditis has been as high as 41% [19]. Unfortunately, more often than not endocarditis forges ahead despite the timely administration of antibiotics, thus necessitating the involvement of cardiothoracic surgery in management of these cases [15]. Pertinently, the indications of surgery in endocarditis includes large vegetations, congestive heart failure and septic emboli and persistent sepsis [20]. It is estimated approximately 50% of cases needs surgical consultation. In children, radical surgical procedure (Ross-Kano procedure) with delayed sternal closure in combination with prolonged antibiotic treatment might be lifesaving [1].

Conclusion

We present this case report to bring the attention to this rare species, *A. defectiva* and its predilection for triggering endocarditis in vulnerable patients. Fastidious growth requirements, lingering clinical course and atypical symptoms/signs will give rise to false-negative blood culture results, thence fostering delayed diagnosis and treatment. Unfortunately, despite specific antibiotics, endocarditis progresses in severity due to antibiotic resistance, thus culminating in cardiac complications requiring surgery NVS endocarditis in patients with pertinent risk factors might require lifelong antibiotic therapy for limiting recurrence and improving clinical outcomes.

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