Journal of Cardiovascular Disease Research

ISSN: 0975-3583,0976-2833 VOL 17, ISSUE 1, 2026

ISOLATED NATIVE PULMONARY VALVE ENDOCARDITIS DUE TO ABIOTROPHIA DEFECTIVA IN A YOUNG WOMAN WITH SUB-PULMONARY VENTRICULAR SEPTAL DEFECT

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Keywords:
Abiotrophia defectiva, Infective Endocarditis, Ventricular Septal Defect
Word count: 1320 words
Declaration of interest: None

ABSTRACT:

A girl in her late adolescence, with known multiple sclerosis, restrictive sub pulmonary ventricular septal defect (VSD) with history of steroid usage and dental caries presented with fever, loss of appetite and weight loss for 3 months and pleuritic chest pain for 2 weeks. Trans-thoracic echocardiography showed a large mobile pulmonary valve vegetation with moderate regurgitation. Extended incubation blood cultures grew *Abiotrophia defectiva*, and MALDI-TOF-MS and VITEK-2 systems were used for speciation and antibiotic susceptibility respectively. Chest imaging revealed septic pulmonary emboli and infarcts in the right lung. She was managed with intravenous ampicillin, gentamycin and vancomycin, following which fever subsided and blood cultures became sterile. She subsequently underwent extensive surgical treatment in the form of VSD closure, vegetectomy, right ventricular infundibular muscle resection and bioprosthetic pulmonary valve replacement. At 3 month follow up, she remains afebrile with improved functional status.

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INTRODUCTION:

Infective endocarditis (IE) refers to infection of the endothelial surface of the heart or intravascular devices like prosthetic valves and has an estimated incidence of 13.8 cases per 100,000 person-years [1]. While left-sided IE predominates, right-sided IE accounts for 10% of cases, typically in association with risk factors like injection drug use, intra-vascular device or congenital heart defects [2]. More than two-thirds of right-sided IE cases have been attributed to *Staphylococcus aureus*, followed by *Streptococci* and *Enterococci* (accounting for 5-30% and 2-5% cases respectively) [3].

Abiotrophia defectiva (AD), formerly classified under 'nutritionally variant streptococci (NVS)' is part of the normal flora of the oral cavity, urogenital and gastro-intestinal tracts. Diagnosis of AD IE involves various challenges like extended incubation time, variable Gram staining and need for molecular techniques for speciation [4]. AD IE has higher morbidity and mortality compared to viridans group streptococcal (VGS) or enterococcal IE, despite its slow and indolent clinical course, and predominantly occurs in the setting of pre-existing heart disease and dental manipulation or dental caries [4]. Stein and Nelson, in a review of 30 NVS IE cases, reported high rates of embolization (27%), relapse after therapy (17%), bacteriological failure despite appropriate antibiotic therapy (41%) and high mortality (17%), highlighting the importance of prompt identification and aggressive therapy [5]. Here, we report a case of isolated native pulmonary valve (PV) IE caused by *Abiotrophia defectiva* in an immuno-compromised young Indian female with pre-existing sub-pulmonary ventricular septal defect (VSD) with septic pulmonary embolism who was successfully managed with appropriate antibiotic therapy and surgery.

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CASE PRESENTATION:A 19-year-old female, resident of North India, with known restrictive sub-pulmonary ventricular septal defect (VSD) presented with complaints of intermittent low-grade fever spikes for the past year, which had increased in frequency (3-4 spikes/day) and intensity (101°-102°F) over the past 3 months. She had history of loss of appetite and loss of weight (around 6 kilograms in past 3 months). She also complained of pleuritic right sided chest pain for the past 2 weeks.

Her past medical history was significant for a diagnosis of multiple sclerosis 18 months earlier, when she developed slurring of speech, sudden onset paraparesis, numbness of bilateral lower-limbs and low-backache. Magnetic resonance imaging (MRI) of brain and spine had revealed the suggestive findings of Dawson's fingers and cervical myelitis at C2-C3 level. Cerebro-spinal fluid (CSF) analysis had revealed significant oligo-clonal bands. She had responded to intravenous corticosteroids. Four months prior to this admission, she again developed lower back-ache, for which she was evaluated in another hospital. MRI spine did not reveal any contrast enhancement (resolution of cervical myelitis) and on the basis of positive rheumatoid factor (anti-CCP negative), she had been labelled as a case of rheumatoid arthritis and started on weekly methotrexate and oral steroids (ongoing at the time of current admission).

On examination, she was febrile, tachycardic, normotensive and maintained room air saturation of 95%. She appeared pale with bipedal edema. No Osler nodes, Janeway lesions, Roth spots or sub-conjunctival haemorrhage were noted. She had active untreated dental caries of her right 2^{nd} molar tooth. Cardiovascular examination revealed a grade 4/6 pan-systolic murmur, heard best at the left 3^{rd}-4^{th} intercostal area (without respiratory variation). Abdomen examination revealed hepato-splenomegaly. Neurological examination was unremarkable.

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Laboratory investigations (Table 1) showed severe anemia (hemoglobin- 6.1 g/dl), neutrophilic leukocytosis (total leukocyte count – 15,830/µL, N%) and elevated inflammatory markers (CRP 127.4 mg/L, procalcitonin 17.1 ng/mL). Chest X-ray at admission was unremarkable.

Trans-thoracic echocardiography (Figure 1) showed a large (1.5 x 1.0 cm) mobile vegetation attached to the pulmonary valve with moderate pulmonary regurgitation, no right ventricular outflow tract obstruction and restrictive sub-pulmonary VSD with a left to right shunt (peak gradient across VSD: 110 mmHg). Three sets of blood cultures grew *Abiotrophia defective* after extended incubation. Speciation was done by using the matrix-assisted laser desorption/ionization time-of-flight mass spectrometry (MALDI-TOF-MS) and antibiotic susceptibility using VITEK-2 system. The isolated organism was susceptible to ampicillin, cefepime, ciprofloxacin, imipenem, gentamicin and vancomycin.

She underwent contrast enhanced computed tomography (CT) of chest and abdomen along with CT pulmonary angiography (Figure 2) to look for evidence of septic emboli (which would necessitate urgent surgery), which revealed filling defects in right descending pulmonary artery and bilateral lower lobe segmental branches with multiple peripheral wedge-shaped consolidations (likely infarcts). The pulmonary valve was thickened with leaflet irregularities but no para-valvular abscess.

The patient was initiated on intravenous ampicillin, gentamicin, and vancomycin. Fever subsided and repeat blood cultures became sterile. Following infection control and stabilization (3 weeks of culture sterility), she underwent open surgical repair comprising trans-pulmonary artery closure of the VSD, vegetectomy, right ventricular infundibular muscle resection, and bioprosthetic pulmonary valve replacement. Intraoperative findings included a perforated, moth-eaten pulmonary valve with small vegetations. All intraoperative cultures (bacterial,

ISSN: 0975-3583,0976-2833 VOL 17, ISSUE 1, 2026

fungal, anaerobic) were negative. She made an uneventful recovery and was discharged in stable condition after 1.5 months of hospital stay. At 3-month follow-up, she remained afebrile with improved functional status.

TABLE 1: Laboratory investigations

Parameter	At admission	After 4 weeks
Hemoglobin (g/dL)	6.1	9.6
Total leukocyte count (/µL)	15830	6920
Differential leukocyte count (N/L) (%)	84/7	60/27
Iron (µg/dL)/ ferritin (ng/mL)/ TIBC (ug/dL)	20/1505/179	
Corrected reticulocyte count	1.6%	
Urea (mg/dL)/Creatinine (mg/dL)	25/0.9	31/0.39
Sodium (mmol/L)/Potassium (mmol/L)	131/3.9	138/4.6
Total bilirubin (mg/dL)/AST (U/L)/ALT (U/L)	0.78/20/17	0.3/29/22
Total protein (g/dL)/albumin (g/dL)	5.8/3.2	7.6/3.9
HIV I, II (ELISA)	Negative	
Hepatitis B surface antigen/Anti HCV	Negative	
Anti-Hepatitis C antibody	Negative	
CRP (mg/L)	127.4	2.4
ESR (mm/hr)	129	
Procalcitonin (ng/mL)	17.1	0.72

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ANA (by IIF)	Negative	
aCL-IgM, aCL-IgG **Lupus anticoagulant** **Anti-β2 glycoprotein 1 Ab**	Negative	
Blood culture	*Abiotrophia defectiva*	Sterile
Urine culture	sterile	

DISCUSSION:

Frenkel and Hirsch first described NVS in 1961 as fastidious Gram-positive cocci, exhibiting satellitism around other bacteria. The nomenclature of NVS underwent multiple changes over the years, based on DNA-DNA hybridization studies and 16S RNA sequence analysis to be finally grouped into two genera, namely *Abiotrophia* and *Granulicatella* [4]. The major portal of entry seems to be the oral cavity, with oral colonization rates of around 11.8% in healthy population [4]. Once *Abiotrophia* gains access to the bloodstream, higher affinity to endocardium is facilitated by factors like excessive production of exopolysaccharide and ability to bind to fibronectin in extra-cellular matrix [6].

The term *'Abiotrophia'* refers to 'life nutrition deficiency', highlighting its need for supplemental culture media enriched with pyridoxine and L-cystine for growth. If these nutrients are absent, a streak of *Staphylococcus aureus* or *Staphylococcus epidermidis* provides appropriate culture conditions [7]. Identification of *Abiotrophia*remains to be a challenge with insufficient accuracy of biochemical tests and phenotypic characteristics. In a study of antimicrobial susceptibility in NVS, Alberti et.al [8] reported susceptibility of all 132 isolates of NVS to vancomycin and no evidence of high-level aminoglycoside resistance among any of

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the isolates. *Abiotrophia* species were less susceptible to penicillin than *Granulicatella* species (10.8% vs 38.9%), however all isolates of *Abiotrophia* were susceptible to ceftriaxone.

Abiotrophia IE is typically slow and indolent in nature, but is associated with higher morbidity and mortality than other streptococcal IE cases. Aortic valve is most commonly involved, with high rates of valve destruction and requirement for surgery in approximately 30% of cases [4]. Mortality due to Abiotrophia IE is mostly due to refractory congestive cardiac failure and major systemic emboli [4]. Despite the high mortality (17%) in the review by Stein and Nelson [5], a more recent report by Garcia et.al [9] revealed a mortality rate of 9.2%. They also reported higher rates of peri-annular complications in NVS IE (28.9%) compared to VGS IE (22%) [9]. Abiotrophia has also been implicated in septic arthritis, osteomyelitis, endophthalmitis, brain abscess and mycotic aneurysms as well [4].

Dental caries, steroid use for back-ache and pre-existing restrictive sub-pulmonary VSD seem to be the predisposing factors for development of IE in our case. The delay in diagnosis of around 3 months in our case, despite extensive evaluation in multiple hospitals further emphasizes the difficulty in identification of *Abiotrophia* IE, due to its fastidious growth requirements.

The American Heart Association (AHA) recommends combination therapy with penicillin G and gentamicin for 4-6 weeks, similar to treatment of enterococcal IE; other treatment options being vancomycin or combination of ceftriaxone and gentamicin [10]. Our patient received a combination of ampicillin, vancomycin and gentamicin with her hospital course being complicated by septic pulmonary embolism and hospital acquired pneumonia, requiring antibiotic upgradation and ultimately surgical repair of the VSD and pulmonary valve IE.

Acknowledgement:

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None

Author contributions:

PRP, SKS, NG, MRM and AS were responsible for drafting of the text, sourcing and editing of clinical images, investigation results and critical revision for important intellectual content and RN, MRM and NG were involved in the review of the manuscript

Statements and declarations:

Not applicable

Declaration of conflicting interest:

The author(s) declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article

Consent statement:

Written informed consent was obtained from the patient for publication of this case report, including accompanying images

Funding statement:

Not applicable

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FIGURE 1: Trans-thoracic echocardiography (TTE) – A: Parasternal short axis (basal) view showing 1.5x 1.0 cm vegetation (arrow) attached to pulmonary valve; B: Parasternal long axis (PLAX) view showing the vegetation (arrow) and sub-pulmonary ventricular septal defect (VSD); C: PLAX view showing turbulent flow across the restrictive sub-pulmonary VSD; D: Gradient across the sub-pulmonary VSD (peak gradient 110 mmHg)

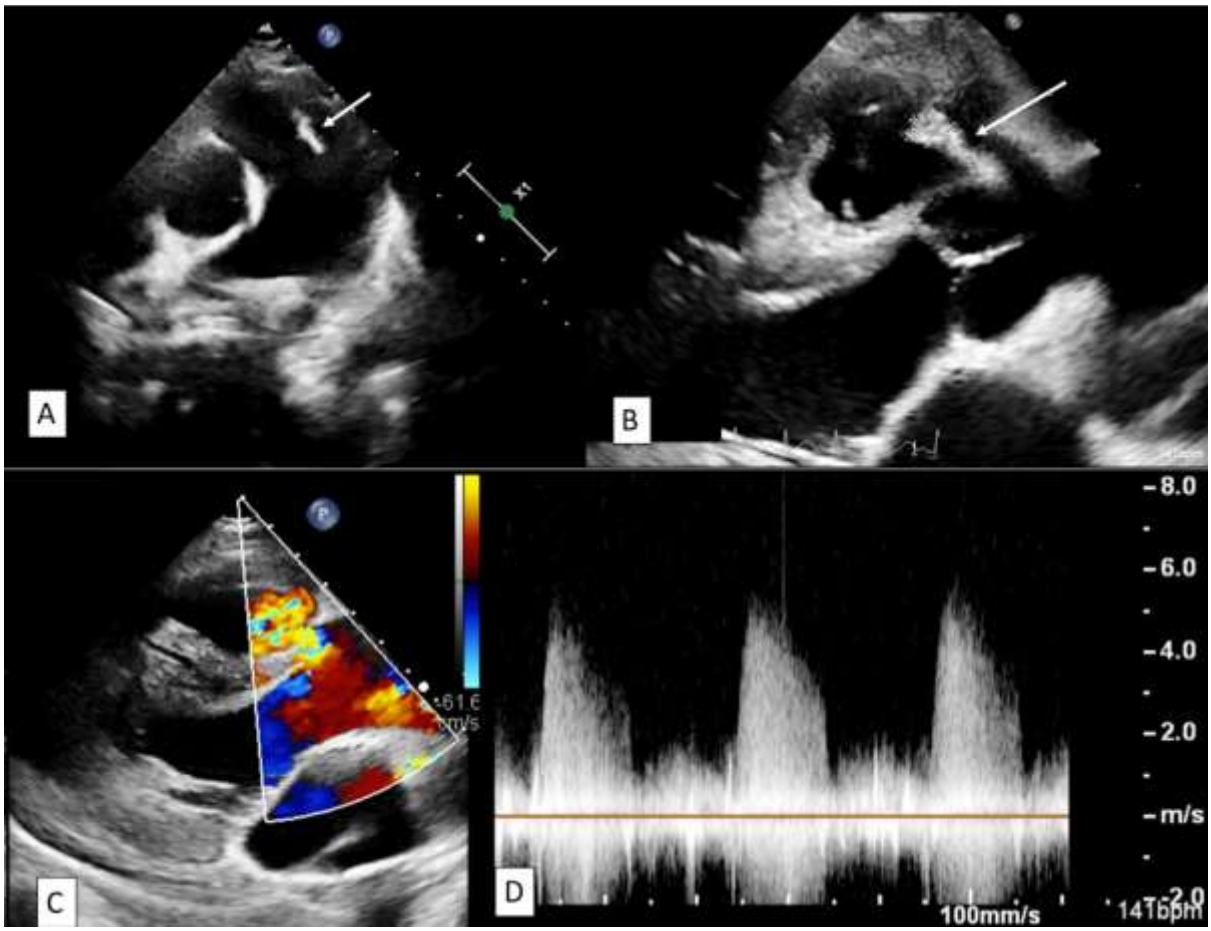

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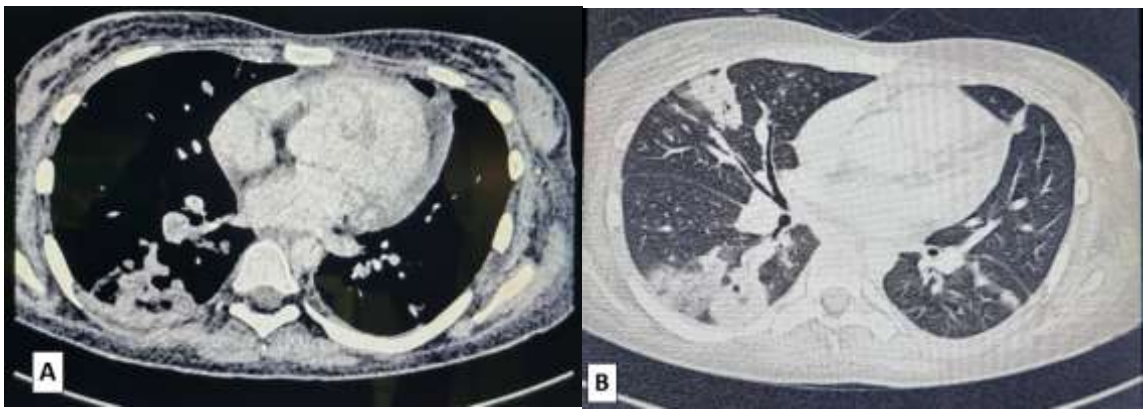
FIGURE 2: Contrast enhanced computed tomography (CECT) chest with CT pulmonary angiography (CTPA) – A: filling defect in right descending pulmonary artery (septic pulmonary embolism) and pulmonary infarct; B: multifocal consolidations in right middle and lower lobe (secondary to septic pulmonary emboli)



Date: 29/12/2025