J Surg Med. 2026;10(11):e8535.
https://doi.org/10.28982/josam.8535

JOSAM

Case Report
https://jsurgmed.com/

Journal of Surgery and Medicine
e-ISSN: 2602-2079

Abiotrophia defectiva Infective Endocarditis in a Patient with Hairy Cell Leukemia: Accelerated Aortic Valve Destruction in a Bicuspid Anatomy

Noor Riaz[1], Selena Dahabreh[2], Mehran Sourehnissani[3]

Institutional Affiliations
[1] Medical University of the Americas, Nevis, Saint Kitts and Nevis
[2] St. George's University School of Medicine, True Blue, Grenada
[3] PIH Health Good Samaritan Hospital, Los Angeles, California, USA

ORCID IDs
NR: https://orcid.org/0009-0004-9390-3467
SD: https://orcid.org/0009-0006-8634-3216
MS: https://orcid.org/0009-0004-1135-3693

Corresponding Author
Noor Riaz
Medical University of the Americas, Nevis, Saint Kitts and Nevis
E-mail: n.riaz@mua.edu

Abstract

Abiotrophia defectiva is a rare, fastidious cause of infective endocarditis associated with destructive valvular disease. Both bicuspid aortic valve and immunocompromised status increase susceptibility. A 55-year-old man with a history of hairy cell leukemia in remission and prior right lung carcinoid tumor presented with fever, cough, dyspnea, fatigue, and left-sided neck and chest pain. Blood cultures grew *A. defectiva*. Echocardiography revealed a bicuspid aortic valve with new vegetations and moderate aortic insufficiency. He underwent aortic valve replacement, which confirmed extensive leaflet destruction. This case illustrates how underlying immune dysfunction from hairy cell leukemia combined with a structural valve abnormality can predispose patients to severe *A. defectiva* endocarditis and emphasizes the need for early recognition and timely surgical management.

Keywords: abiotrophia defectiva, infective endocarditis, bicuspid aortic valve, aortic valve replacement, aortic insufficiency, hairy cell leukemia, nutritionally variant streptococci

Ethical Approval
No institutional approval/exemption was required at the treating institution.

Informed Consent
Informed consent was obtained from the patient for publication of this case and the accompanying image.

Conflict of Interest
No conflict of interest was declared by the authors.

Financial Disclosure
The authors declared that this case received no financial support.

CRediT Author Statement
Noor Riaz, Selena Dahabreh:. Writing, reviewing, editing and data curation, Mehran Sourehnissani: supervision, conceptualization, reviewing and editing

Use of Artificial Intelligence (AI) Statement
No AI or AI assisted technology was used in manuscript preparation

Data Availability
All data generated or analyzed during this case report are included in this published article.

Published
2026 November 18

Creative Commons Attribution (CC BY 4.0)
Copyright © 2026 by Author(s). Published by Journal of Surgery and Medicine (JOSAM).
This work is licensed under a Creative Commons Attribution 4.0 International License.
To view a copy of this license, visit https://creativecommons.org/licenses/by/4.0/

How to cite: Riaz N, Dahabreh S, Sourehnissani M. Abiotrophia defectiva Infective Endocarditis in a Patient with Hairy Cell Leukemia: Accelerated Aortic Valve Destruction in a Bicuspid Anatomy. J Surg Med. 2026;10(11):e8535.

Introduction

Infective endocarditis due to nutritionally variant streptococci, particularly *Abiotrophia defectiva*, is a rare but aggressive infection caused by a fastidious organism that presents major diagnostic challenges and is associated with poor clinical outcomes. These organisms often evade early detection because of their complex growth requirements and are disproportionately associated with extensive valvular destruction and the need for surgical intervention [1-3]. *A. defectiva* accounts for approximately 2% of all infective endocarditis cases and is associated with higher rates of complications, bacteriologic failure, and mortality compared with other streptococcal species [2, 4].

Bicuspid aortic valve is a congenital structural abnormality that predisposes patients to infective endocarditis [5, 6]. Endocarditis involving the aortic valve may lead to acute or subacute aortic insufficiency, a complication associated with high morbidity and mortality [7]. Rapid progression of regurgitation due to leaflet destruction is particularly concerning because it can precipitate heart failure and necessitate urgent surgical intervention. Although *A. defectiva* has been reported in patients with underlying structural heart disease, cases resulting in accelerated leaflet destruction and early progression to severe valvular dysfunction remain uncommon [8].

In addition to structural abnormalities, host immune status plays a critical role in susceptibility to invasive infections. Patients with hematologic malignancies, including those in remission, may have persistent immune dysfunction that predisposes them to atypical or aggressive infections [9]. Hairy cell leukemia, in particular, is associated with long-standing defects in cell-mediated immunity as well as profound monocytopenia, which may increase vulnerability to fastidious organisms and severe infectious complications [10].

We present a case of *Abiotrophia defectiva* infective endocarditis in a patient with a history of hairy cell leukemia in remission and previously unrecognized bicuspid aortic valve, complicated by rapid aortic valve destruction and progression to severe aortic insufficiency requiring surgical valve replacement. This case highlights the combined impact of structural valve disease and underlying immune dysfunction on disease severity and emphasizes the importance of early recognition of atypical pathogens in immunologically vulnerable patients, even in the absence of traditional risk factors.

Case presentation

A 55-year-old man with a past medical history of right lung carcinoid tumor requiring right middle and lower lobectomy and hairy cell leukemia in remission presented with neck pain and left-sided chest pain radiating to the left upper extremity. He reported 3 weeks of intermittent fever, productive cough, dyspnea, fatigue, poor appetite, and difficulty sleeping. He denied any history of intravenous drug use or tobacco use.

On admission, he was febrile, and blood cultures were obtained. Cultures subsequently grew *Abiotrophia defectiva*, a nutritionally variant streptococcus. Review of an outpatient computed tomography angiogram performed 5 months earlier revealed no coronary artery stenosis, a coronary artery calcium score of 66 (76th percentile for age- and sex-matched controls), a left ventricular ejection fraction (LVEF) of 55%, bileaflet aortic valve morphology, a small right pleural effusion, and multiple hepatic cysts.

A transthoracic echocardiogram (TTE) obtained during admission demonstrated an LVEF of 60-65%, no regional wall motion abnormalities, mild aortic insufficiency, and possible vegetations on the aortic valve. Compared with an echocardiogram obtained 6 months earlier, a new aortic valve vegetation was noted.

The patient subsequently underwent transesophageal echocardiography (TEE), which showed an LVEF of 55-60% and no regional wall motion abnormalities. A bicuspid aortic valve was identified. A definite vegetation was visualized on the bicuspid aortic valve leaflets and along the subvalvular region (Figure 1), accompanied by moderate aortic insufficiency. Compared with prior imaging, these findings confirmed bicuspid aortic valve endocarditis with progression to moderate aortic insufficiency.

Given these findings, particularly progressive aortic insufficiency, cardiothoracic surgery was consulted, and the patient proceeded to aortic valve replacement. Intraoperatively, the aortic valve was noted to be bicuspid, with one leaflet approximately half destroyed by endocarditis and containing vegetations. The remaining leaflet exhibited thickening and additional vegetations, without outflow obstruction. Moderate-to-severe aortic insufficiency was present due to leaflet destruction. The annulus was noncalcified, and no annular abscess was identified.

Figure 1. Transesophageal echocardiogram (TEE) demonstrating a bicuspid aortic valve with definite vegetations along the valve leaflets and subvalvular region, consistent with *Abiotrophia defectiva* infective endocarditis. Moderate aortic insufficiency was noted secondary to leaflet destruction.



Discussion

Abiotrophia species, previously known as nutritionally variant streptococci, are gram-positive, catalase-negative cocci that colonize the oral cavity, gastrointestinal tract, and genitourinary tract. *A. defectiva* is implicated as the causative organism in 1-2% of infective endocarditis cases [5]. This species is particularly virulent because it produces exopolysaccharides that facilitate binding to fibronectin in the extracellular matrix [11]. *A. defectiva* is a fastidious species that requires growth media enhanced with vitamin B6 or cysteine [3], and routine culture media typically cannot detect it. *A. defectiva* preferentially adheres to damaged or prosthetic heart valves and most commonly affects

This work is licensed under a Creative Commons Attribution 4.0 International License.
To view a copy of this license, visit https://creativecommons.org/licenses/by/4.0/

the mitral valve, although aortic valve involvement occurs in a minority of cases [12, 13]. Common predisposing factors include prior infective endocarditis, chronic kidney disease, advanced age, bicuspid aortic valve, corticosteroid use, immunosuppression, pregnancy, or recent invasive procedures [14].

Infective endocarditis has variable clinical features and may present as an acute, subacute, or chronic condition, with up to 90% of patients experiencing fever, fatigue, or chest pain [15]. Embolic phenomena occur in 20-50% of patients and are a notable clinical feature that can cause cardiac and extracardiac injury [16]. This patient met contemporary criteria for definite infective endocarditis based on positive blood cultures and echocardiographic evidence of endocardial involvement with new valvular vegetation [14].

Bicuspid aortic valve endocarditis warrants particular attention because of its association with accelerated valvular destruction. Turbulent flow and increased shear stress across a bicuspid valve, even when normally functioning, cause chronic endothelial damage and inflammation that promote sterile thrombus formation, creating an ideal nidus for bacterial adhesion. Bicuspid aortic valve endocarditis is associated with larger vegetations, more frequent perivalvular extension, and higher rates of heart failure and surgical intervention compared with tricuspid aortic valve endocarditis. Notably, this patient's CT angiogram and echocardiogram obtained only 5 months earlier showed only bileaflet morphology without vegetations or significant regurgitation, underscoring the rapidity of valvular destruction, a hallmark of the accelerated course described in this case.

No clear portal of entry was identified; the patient denied recent dental procedures, invasive interventions, or intravenous drug use. *A. defectiva* is a commensal organism of the oral cavity and gastrointestinal tract, and transient bacteremia from routine activities such as chewing or toothbrushing may have been sufficient to seed the damaged bicuspid valve endothelium.

Antimicrobial therapy remains the cornerstone of infective endocarditis treatment; however, surgical intervention is necessary in cases of severe valvular damage or recurrent embolism. Current European Society of Cardiology guidance recommends penicillin G, ceftriaxone, or vancomycin for 6 weeks for infective endocarditis caused by Granulicatella or Abiotrophia; for prosthetic-valve endocarditis, an aminoglycoside is added for at least the first 2 weeks [14]. Antimicrobial susceptibility testing is critical because of the organism's unpredictable resistance patterns [17]. Unfortunately, susceptibility testing results were not available in this case; therefore, resistance patterns could not be further evaluated.

The decision to proceed with surgical valve replacement was driven by progression to moderate-to-severe aortic insufficiency with leaflet destruction, which met established surgical indications for heart failure prevention. However, the known propensity of *A. defectiva* for bacteriologic failure with antibiotic therapy alone, with reported failure rates higher than those of other streptococcal species, combined with the patient's underlying immune dysfunction from hairy cell leukemia, further supported early surgical intervention rather than prolonged medical management.

Although no explicit large-scale data support a direct relationship between hairy cell leukemia and the development of infective endocarditis, the immunologic consequences of this malignancy are highly relevant. Hairy cell leukemia is a chronic B-cell malignancy associated with profound and characteristic monocytopenia, present in nearly all cases, as well as disease-related neutropenia. Standard treatments for hairy cell leukemia commonly include purine analogues, such as cladribine and pentostatin. Although these treatments are highly effective, they cause significant myelosuppression and immunosuppression, which translate into greater cytopenia and increased susceptibility to infection. Specifically, treatment with purine analogues results in significant neutropenia and CD4+ T-lymphopenia, contributing to underlying immune dysfunction. Long-lasting CD4+ T-cell depletion may persist for more than 2-3 years, and defects in the monocyte-macrophage lineage and dendritic cell compartment further compromise innate immune surveillance. Approximately 20-30% of patients with hairy cell leukemia develop opportunistic infections during the disease course. These specific immune deficits, rather than generalized immunosuppression alone, likely rendered this patient particularly vulnerable to a fastidious organism such as *A. defectiva*, even while in clinical remission. Abnormal B- and T-lymphocyte function in the setting of leukemia further increases susceptibility to bloodstream infections from opportunistic pathogens [17, 18].

In this case, the patient's congenital bicuspid aortic valve provided a susceptible site for bacterial adherence, while his history of hairy cell leukemia may have contributed to impaired host immunity and the subsequent development of *A. defectiva* infective endocarditis. His prior right lung carcinoid tumor was retained as part of the relevant medical history but is not independently established here as an immunosuppressive risk factor.

This case illustrates the combined impact of structural valve abnormality and impaired host immunity on the development of severe *Abiotrophia defectiva* infective endocarditis. The patient's bicuspid aortic valve provided a susceptible site for bacterial adherence, while his history of hairy cell leukemia, with its characteristic monocytopenia, T-cell depletion, and dendritic cell deficits, likely permitted rapid valvular destruction even in clinical remission. The absence of vegetations or significant regurgitation on imaging only 5 months earlier underscores the accelerated nature of this process. Because *A. defectiva* is a fastidious organism with a subtle, subacute presentation, diagnosis can be delayed and complications can progress quickly. This case reinforces the importance of early recognition, appropriate antimicrobial therapy, and timely surgical intervention for optimizing clinical outcomes in this rare but highly destructive cause of infective endocarditis.

References

1. Ohara-Nemoto Y, Tajika S, Sasaki M, Kaneko M. Identification of Abiotrophia adiacens and Abiotrophia defectiva by 16S rRNA gene PCR and restriction fragment length polymorphism analysis. J Clin Microbiol. 1997;35(10):2458-63. https://doi.org/10.1128/JCM.35.10.2458-2463.1997
2. Seguiti C, Piacentini E, Fraghì A, Zappa M, Croce E, Meloni A, et al. Abiotrophia defectiva and Granulicatella: a literature review on prosthetic joint infection and a case report on A. defectiva PJI and concurrent native valve endocarditis. Microorganisms. 2025;13(5):1113. https://doi.org/10.3390/microorganisms13051113
3. Christensen JJ, Facklam RR. Granulicatella and Abiotrophia species from human clinical specimens. J Clin Microbiol. 2001;39(10):3520-3. https://doi.org/10.1128/JCM.39.10.3520-3523.2001

This work is licensed under a Creative Commons Attribution 4.0 International License.
To view a copy of this license, visit https://creativecommons.org/licenses/by/4.0/

4. Rhodes HM, Hirigoyen D, Shabnam L, Williams DN, Hansen GT. Infective endocarditis due to Abiotrophia defectiva and Granulicatella spp. complicated by infectious intracranial cerebral aneurysms: a report of three cases and review of the literature. J Med Microbiol. 2016;65(6):493-9. https://doi.org/10.1099/jmm.0.000260
5. Téllez A, Ambrosioni J, Llopis J, Pericàs JM, Falces C, Almela M, et al. Epidemiology, clinical features, and outcome of infective endocarditis due to Abiotrophia species and Granulicatella species: report of 76 cases, 2000-2015. Clin Infect Dis. 2018;66(1):104-11. https://doi.org/10.1093/cid/cix752
6. Cai S, Zhou C, Shan Y, Bao R, Hu L, Pan J, et al. Epidemiology, clinical characteristics, and outcome of infective endocarditis due to Abiotrophia and Granulicatella in a tertiary hospital in China, 2015-2023: a retrospective study. BMC Infect Dis. 2024;24(1):1022. https://doi.org/10.1186/s12879-024-09943-4
7. Chambers HF, Bayer AS. Native-valve infective endocarditis. N Engl J Med. 2020;383(6):567-76. https://doi.org/10.1056/NEJMcp2000400
8. Zegri-Reiriz I, de Alarcón A, Muñoz P, Martínez Sellés M, González-Ramallo V, Miró JM, et al. Infective endocarditis in patients with bicuspid aortic valve or mitral valve prolapse. J Am Coll Cardiol. 2018;71(24):2731-40. https://doi.org/10.1016/j.jacc.2018.03.534
9. Kraut E. Infectious complications in hairy cell leukemia. Leuk Lymphoma. 2011;52(Suppl 2):50-2. https://doi.org/10.3109/10428194.2011.570819
10. Bourguin-Plonquet A, Rouard H, Roudot-Thoraval F, Bellanger C, Marquet J, Delfau-Larue MH, et al. Severe decrease in peripheral blood dendritic cells in hairy cell leukaemia. Br J Haematol. 2002;116(3):595-7. https://doi.org/10.1046/j.0007-1048.2001.03318.x
11. Sasaki M, Shimoyama Y, Ishikawa T, Kodama Y, Tajika S, Kimura S. Contribution of different adherent properties of Granulicatella adiacens and Abiotrophia defectiva to their associations with oral colonization and the risk of infective endocarditis. J Oral Sci. 2020;62(1):36-9. https://doi.org/10.2334/josnusd.19-0021
12. Elashery AR, Stratidis J, Patel AD. Double-valve heart disease and glomerulonephritis consequent to Abiotrophia defectiva endocarditis. Tex Heart Inst J. 2020;47(1):35-7. https://doi.org/10.14503/THIJ-17-6575
13. Je H, Song D, Chang CL. Bacterial endocarditis caused by Abiotrophia defectiva in a healthy adult: a case report with literature review. Ann Clin Microbiol. 2019;22(1):23-7. https://doi.org/10.5145/ACM.2019.22.1.23
14. Delgado V, Ajmone Marsan N, de Waha S, Bonaros N, Brida M, Burri H, et al. 2023 ESC Guidelines for the management of endocarditis. Eur Heart J. 2023;44(39):3948-4042. https://doi.org/10.1093/eurheartj/ehad193
15. Abdelgawad H, Azab S, Abdel-Hay MA, Almaghraby A. Clinical features and outcomes of infective endocarditis: a single-centre experience. Cardiovasc J Afr. 2023;34(2):82-8. https://doi.org/10.5830/CVJA-2022-027
16. Kasper DL, Fauci AS, Hauser SL, Longo DL, Jameson JL, Loscalzo J, editors. Harrison's principles of internal medicine. 19th ed. New York: McGraw-Hill Education; 2015. p. 816-26. ISBN: 9780071802154.
17. Naing PT, Acharya U, Kumar A. Hairy cell leukemia. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2026 [updated 2023 May 22]. Available from: https://www.ncbi.nlm.nih.gov/books/NBK499845/
18. Goldman D. Chronic lymphocytic leukemia and its impact on the immune system. Clin J Oncol Nurs. 2000;4(5):233-4, 236. Available from: https://pubmed.ncbi.nlm.nih.gov/11111455/

Disclaimer/Publisher's Note: The statements, opinions, and data presented in publications in the Journal of Surgery and Medicine (JOSAM) are exclusively those of the individual author(s) and contributor(s) and do not necessarily reflect the views of JOSAM, the publisher, or the editor(s). JOSAM, the publisher, and the editor(s) disclaim any liability for any harm to individuals or damage to property that may arise from implementing any ideas, methods, instructions, or products referenced within the content. Authors are responsible for all content in their article(s), including the accuracy of facts, statements, and citations. Authors are responsible for obtaining permission from the previous publisher or copyright holder if re-using any part of a paper (e.g., figures) published elsewhere. The publisher, editors, and their respective employees are not responsible or liable for the use of any potentially inaccurate or misleading data, opinions, or information contained within the articles on the journal's website.

This work is licensed under a Creative Commons Attribution 4.0 International License.
To view a copy of this license, visit https://creativecommons.org/licenses/by/4.0/