Cureus
Part of SPRINGER NATURE

Open Access Case Report

Native Aortic Valve Infective Endocarditis Involving the Mitral-Aortic Intervalvular Fibrosa Caused by Lacticaseibacillus paracasei Presenting With a Prolonged Constitutional Syndrome and Non-diagnostic FDG/PET-CT: A Case Report and Literature Review

Arthiom Cambour [1], Mihaela Malanca Onica [1], Nawel Rezzoug [1], Céline Goffinet [1]

1. Cardiology, Clinique Saint Jean, Brussels, BEL

Corresponding author: Arthiom Cambour, a.cambour@gmail.com

Review began 10/12/2026
Review ended 10/27/2026
Published 11/01/2026

© **Copyright** 2026
Cambour et al. This is an open access article distributed under the terms of the Creative Commons Attribution License CC-BY 4.0., which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

DOI: 10.7759/cureus.118942

Abstract

Lacticaseibacillus paracasei is a rare cause of infective endocarditis (IE) and is frequently regarded as a blood culture contaminant, which may delay diagnosis. A 75-year-old man presented with a six-month history of progressive weight loss, drenching night sweats, and chills without fever. Initial investigations revealed an inflammatory syndrome, while FDG/PET-CT showed only nonspecific findings, prompting evaluation for inflammatory, infectious, and hematologic disorders. Transthoracic echocardiography (TTE) raised suspicion for IE, and transesophageal echocardiography (TEE) demonstrated a 9 mm vegetation on the mitral-aortic intervalvular fibrosa (MAIVF), without aortic leaflet involvement seen on imaging and with mild aortic regurgitation. Four pairs of blood cultures came back positive for *Lacticaseibacillus paracasei*, confirming persistent bacteremia. No conventional predisposing factors, including immunosuppression, probiotic use, dental disease, or significant gastrointestinal pathology, were identified. Intravenous antimicrobial therapy achieved microbiological clearance; however, repeat transesophageal echocardiography demonstrated persistent vegetation with MAIVF thinning and cavity formation, suspicious for pseudoaneurysm, prompting referral for cardiac surgery at another institution. The postoperative course was favorable, and the patient was discharged 10 days after surgery. This case illustrates that *L. paracasei* should not automatically be dismissed as a contaminant and that native aortic valve infective endocarditis involving the MAIVF may present without fever, while FDG/PET-CT may be falsely reassuring. Repeated blood cultures and serial echocardiography remain essential for establishing the diagnosis of endocarditis and guiding management.

Categories: Cardiology, Infectious Disease
Keywords: blood culture contaminant, fdg/pet-ct, infective endocarditis, lacticaseibacillus paracasei, lactobacillus, maivf, native aortic valve

Introduction

Lacticaseibacillus paracasei (formerly *Lactobacillus paracasei*) is a Gram-positive, facultatively anaerobic bacillus that is part of the normal oral and gastrointestinal microbiota and is widely used in probiotic preparations and fermented dairy products [1]. Although generally considered a microorganism of low virulence, it has rarely been implicated in invasive infections, including infective endocarditis (IE) [1,2]. In the largest review to date, only 7 of 82 reported cases of *Lactobacillus* endocarditis were caused by *L. paracasei* [1]. Because *Lactobacillus* species are frequently regarded as blood culture contaminants, recognition of true infection may be delayed [1].

Reported cases of *Lacticaseibacillus endocarditis* are uncommon and are typically associated with predisposing conditions such as prosthetic heart valves, immunosuppression, gastrointestinal pathology, dental disease, or probiotic exposure [1,3-6]. Diagnosis may be particularly challenging in native valve IE because patients can present with an indolent clinical course [2,7], and FDG/PET-CT, a major imaging criterion in the 2023 Duke-International Society for Cardiovascular Infectious Diseases (ISCVID) criteria mainly for prosthetic valve and device infection, has limited sensitivity in this setting [8,9]. The mitral-aortic intervalvular fibrosa (MAIVF) is a fibrous part of the cardiac skeleton lying between the aortic root, at the level of the non-coronary and left coronary cusps, and the anterior mitral leaflet. Infection at this site is uncommon but predisposes to perivalvular abscess, pseudoaneurysm, and fistula formation [9].

We report a rare case of native aortic valve infective endocarditis involving the mitral-aortic intervalvular fibrosa (MAIVF), caused by *L. paracasei* in a patient without identifiable predisposing risk factors, presenting as a prolonged constitutional syndrome with repeatedly positive blood cultures and initially non-contributory PET imaging. This case highlights the importance of considering *Lacticaseibacillus* species as

How to cite this article
Cambour A, Malanca Onica M, Rezzoug N, et al. (November 01, 2026) Native Aortic Valve Infective Endocarditis Involving the Mitral-Aortic Intervalvular Fibrosa Caused by Lacticaseibacillus paracasei Presenting With a Prolonged Constitutional Syndrome and Non-diagnostic FDG/PET-CT: A Case Report and Literature Review. Cureus 18(11): e118942. DOI 10.7759/cureus.118942

true pathogens when repeatedly isolated from blood cultures and illustrates the limitations of FDG/PET-CT in native valve infective endocarditis.

Case Presentation

A 75-year-old man was referred to our institution for evaluation of a six-month history of progressive constitutional symptoms characterized by involuntary weight loss exceeding 10% of his body weight, drenching night sweats, and recurrent chills. He denied documented fever throughout the course of his illness and reported preservation of his appetite. He also described chronic mild lower urinary tract symptoms, including dysuria and urinary urgency, without flank pain or gross hematuria.

His medical history was notable for recurrent unprovoked pulmonary embolism in 2011, 2015, and 2019, requiring lifelong anticoagulation with rivaroxaban. Additional comorbidities included hypertension and benign prostatic hyperplasia. His regular medications consisted of rivaroxaban, perindopril/amlodipine, sildenafil, and tamsulosin.

On admission, the patient was afebrile and hemodynamically stable. Cardiovascular examination revealed a soft systolic murmur and no peripheral manifestations of infective endocarditis. The remainder of the physical examination was unremarkable. Initial laboratory investigations demonstrated a hemoglobin concentration of 11.8 g/dL, leukocytosis of 12.9 × 10^9/L with neutrophil predominance, and a markedly elevated C-reactive protein level of 103 mg/L. HIV serology and an interferon-γ release assay for tuberculosis were negative. Given the prolonged constitutional syndrome with elevated inflammatory biomarkers, the initial diagnostic workup focused on inflammatory, infectious, and hematologic disorders. FDG/PET-CT was performed after dedicated preparation for cardiac imaging, before the initiation of any antimicrobial therapy, and demonstrated diffuse mild hypermetabolism of the bone marrow and spleen without focal valvular or metastatic infectious uptake, findings considered nonspecific. Subsequent hematologic evaluation with bone marrow aspiration identified a small monoclonal B-cell population but found no evidence of hematologic malignancy or another explanation for the persistent inflammatory syndrome. Immunoglobulin levels were normal.

Because infective endocarditis remained a differential diagnosis, transthoracic echocardiography (TTE) was performed and raised suspicion for a vegetation on the mitral-aortic intervalvular fibrosa (MAIVF) (Figure *1*). Transesophageal echocardiography (TEE) performed two days after the TTE subsequently confirmed a highly mobile vegetation measuring approximately 9 mm attached to the MAIVF, without visible aortic leaflet vegetation and with mild aortic regurgitation, consistent with native left-sided infective endocarditis involving the MAIVF (Figure *2*). Four pairs of blood cultures grew *Lacticaseibacillus paracasei*, identified by matrix-assisted laser desorption/ionization time-of-flight (MALDI-TOF) mass spectrometry. The repeated isolation of the same organism confirmed persistent bacteremia and strongly supported its role as the causative pathogen rather than a contaminant. Together with the echocardiographic vegetation, these findings fulfilled two major Duke-ISCVID criteria, and the case was classified as definite infective endocarditis.

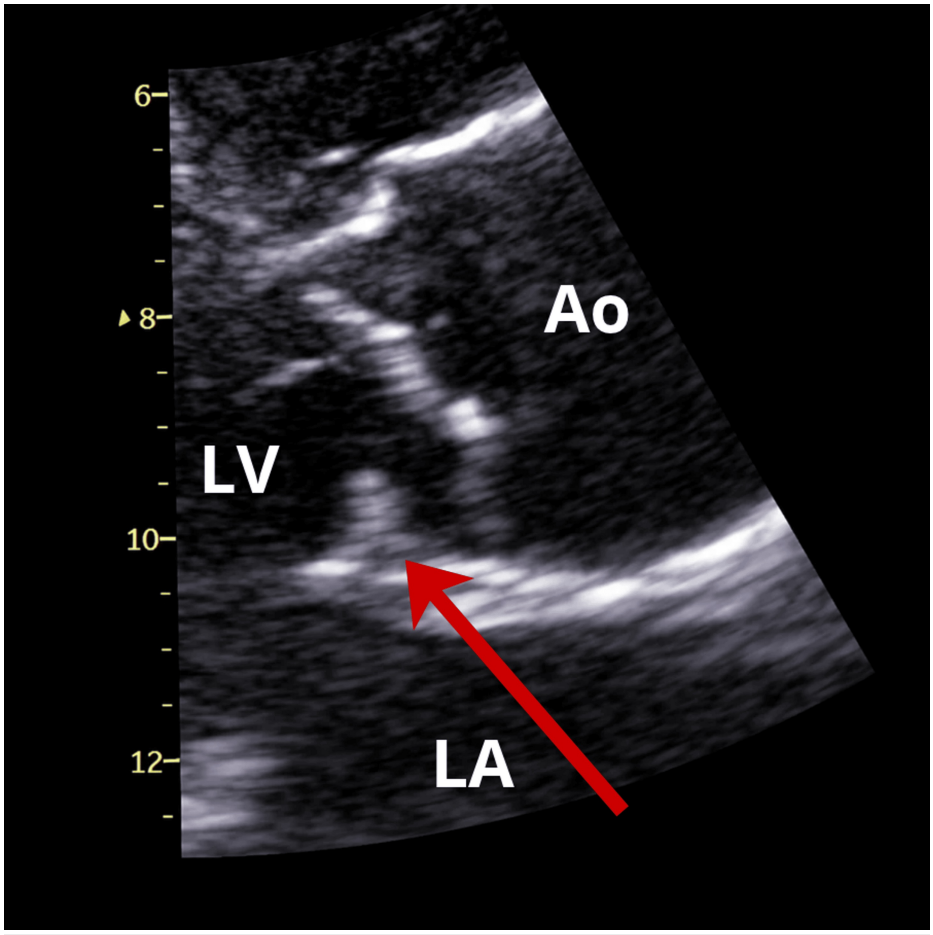


FIGURE 1: Initial transthoracic echocardiography, PLAX view, showing a suspicious lesion on the MAIVF (arrow), with no aortic leaflet vegetation visible

Ao: aorta, LA: left atrium, LV: left ventricle, MAIVF: mitral-aortic intervalvular fibrosa, PLAX: parasternal long-axis

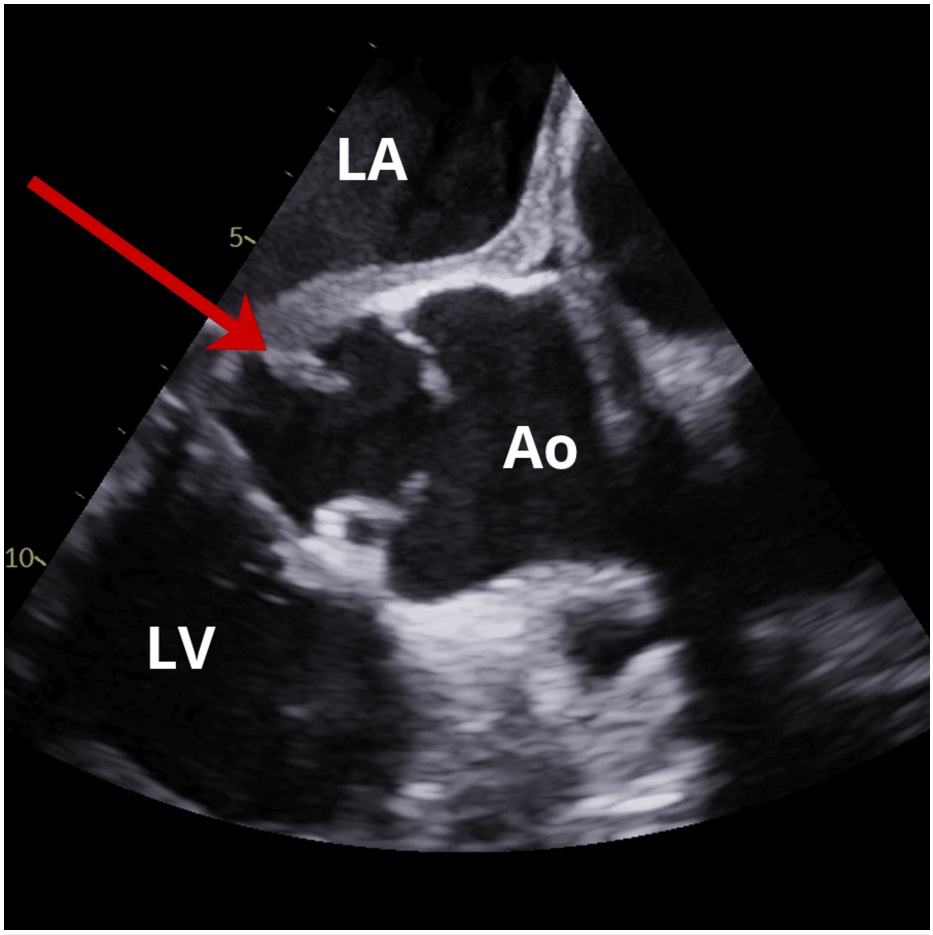


FIGURE 2: Initial transesophageal echocardiography performed two days after the first transthoracic echocardiography, mid-esophageal three-chamber (long-axis) view, showing a 9 mm vegetation on the mitral-aortic intervalvular fibrosa (MAIVF) (arrow), with no aortic leaflet vegetation visible

Ao: aorta, LA: left atrium, LV: left ventricle, MAIVF: mitral-aortic intervalvular fibrosa

A comprehensive search for predisposing factors and a potential portal of entry was undertaken. The patient had no history of immunosuppressive disease or immunosuppressive treatment, and HIV testing was negative. He denied probiotic supplementation, excessive consumption of fermented dairy products or yogurt, recent dental procedures, or symptoms suggestive of active dental disease. Dental examination found no evidence of dental infection or other oral pathology. Colonoscopy revealed diverticulosis without evidence of colorectal neoplasia or inflammatory bowel disease. No alternative source of bacteremia could be identified. Computed tomography of the chest, abdomen, and pelvis showed no evidence of septic embolization, and brain magnetic resonance imaging revealed only chronic ischemic lesions without acute cerebral emboli.

Empiric intravenous amoxicillin-clavulanate 2.2 g four times daily was initiated, and following susceptibility testing, gentamicin was added for synergy. On the advice of the infectious diseases specialist, gentamicin was subsequently replaced by rifampicin. Follow-up blood cultures became sterile five days after the initiation of antimicrobial therapy.

Clinical status progressively improved, and inflammatory markers declined. Despite microbiological clearance and biochemical improvement, repeat transesophageal echocardiography performed 17 days after the initial TEE demonstrated persistence of the MAIVF vegetation, measuring approximately 10 mm, together with thinning of the MAIVF and cavity formation suspicious for pseudoaneurysm (Figure *3* and Figure *4*). Following multidisciplinary discussion by the Endocarditis Heart Team, surgical intervention was recommended because of the persistent vegetation and the risk of MAIVF rupture. Surgery was performed at another institution, and no detailed operative or histopathological report was available to us. The procedures communicated were cusp plication, Cabrol commissural sutures, bovine pericardial patch

reconstruction, and sinotubular junction annuloplasty. Although detailed intraoperative findings are unavailable, the nature of these procedures suggests that the aortic valve cusps may also have been involved, which was not apparent on preoperative echocardiography. The postoperative course was uneventful. Antimicrobial therapy was completed for a total of six weeks starting from the first negative blood culture, without interruption or drug-related adverse events. One month after discharge, the patient was asymptomatic, inflammatory markers had fully normalized, and blood cultures were negative. Transthoracic echocardiography showed a competent, well-functioning repaired aortic valve without residual vegetation. Follow-up is ongoing.

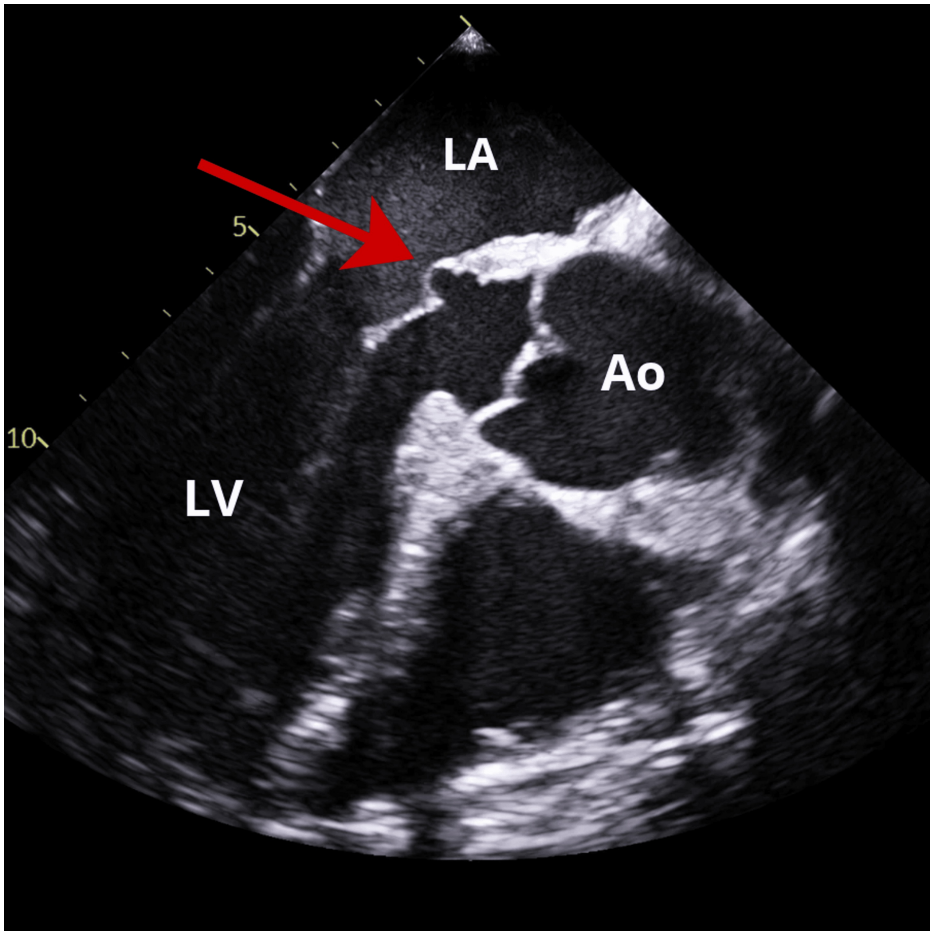


FIGURE 3: Control transesophageal echocardiography (performed 17 days after the first transesophageal echocardiography) showing thinning of the MAIVF with cavity formation (arrow), suspicious for early pseudoaneurysm formation

Ao: aorta, LA: left atrium, LV: left ventricle, MAIVF: mitral-aortic intervalvular fibrosa

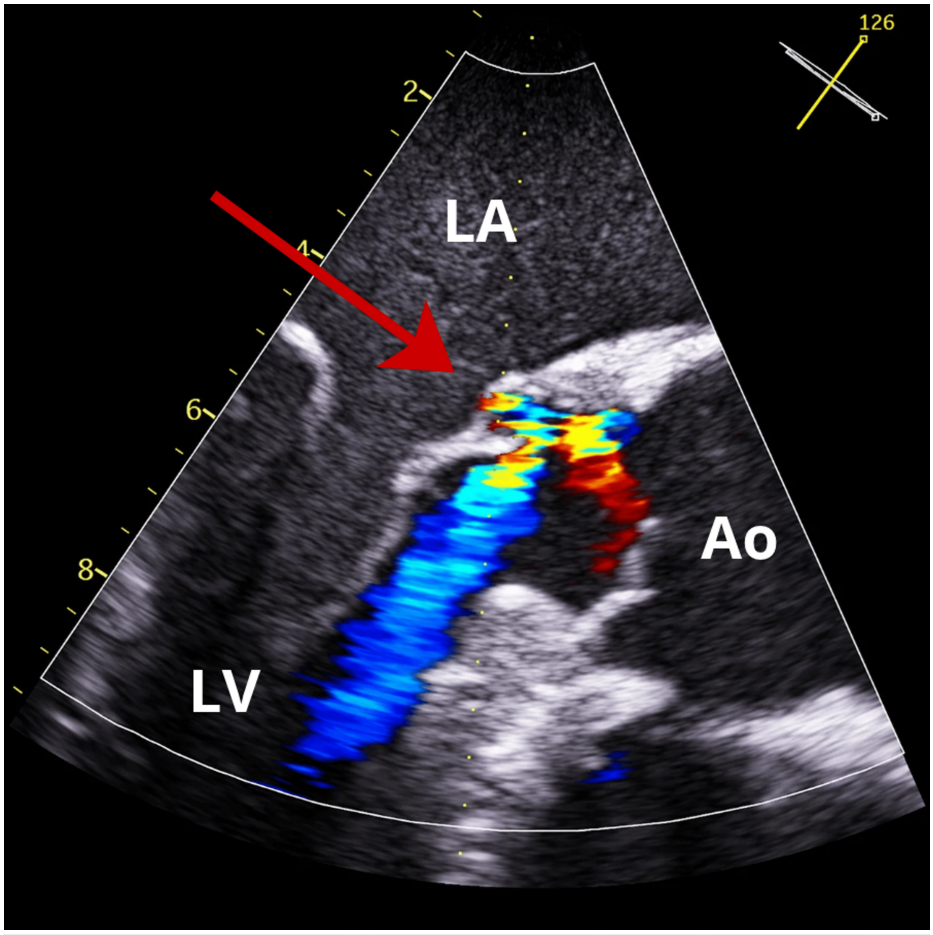


FIGURE 4: Control transesophageal echocardiography with color Doppler imaging of the MAIVF, showing flow within the thinned MAIVF cavity, suspicious for pseudoaneurysm formation

Ao: aorta, LA: left atrium, LV: left ventricle, MAIVF: mitral-aortic intervalvular fibrosa

Discussion

Lacticaseibacillus paracasei (formerly *Lactobacillus paracasei*) is an exceptionally rare cause of infective endocarditis (IE). In the largest review published to date, Ioannou et al. identified only seven cases of *L. paracasei* infection among 82 reported cases of *Lactobacillus* endocarditis, emphasizing the rarity of this pathogen and the limited clinical experience available to guide management [1]. To identify previously published cases, we searched PubMed for "*Lacticaseibacillus paracasei*" or "*Lactobacillus paracasei*" combined with "endocarditis" (from 2000 to July 2026) and added the cases included in the review by Ioannou et al. [1]. Together, these sources yielded 16 articles, of which 8 were excluded (non-English language, non-clinical articles, systematic reviews, incomplete data, not endocarditis, or full text unavailable). Of the eight remaining cases, seven are summarized in Table *1*; the eighth (DeMarco et al. (2023) [10]) was excluded from the table because it was a mixed infection with *L. casei*, *L. paracasei*, and *L. zeae*. The tabulated cases were generally associated with identifiable predisposing conditions, including structural valvular disease, prosthetic valves, or probiotic consumption [2-7,11]. In contrast, our patient had no recognized predisposing condition despite extensive investigation, which included negative HIV testing, no probiotic use, a normal dental history and examination, and a colonoscopy showing only uncomplicated diverticulosis, considered an incidental finding at his age.

Study	Age/sex	Valve	Predisposing factors	Clinical presentation	Valve involved	Treatment	Outcome
Soleman et al. (2003) [2]	75/female	Native	Severe aortic and mitral valve stenosis	Acute heart failure, chills, afebrile	Mitral	Penicillin + gentamicin → oral linezolid	Recovered; minor embolic stroke
Franko et al. (2013) [3]	77/male	Native	Mitral valve prolapse; daily probiotics	Recurrent chills without fever	Mitral	Amoxicillin + gentamicin; mitral valve repair	Recovered
Kato et al. (2016) [4]	78/male	Native	Severe bicuspid aortic stenosis; daily probiotics; previous dental extraction	Chest pain, later fever	Aortic	Clindamycin; AVR + CABG	Recovered
Osman et al. (2019) [5]	65/male	Bioprosthetic	Previous IE; bioprosthetic AVR; dental extraction	Recurrent embolic strokes; initially afebrile	Bioprosthetic aortic	Penicillin + gentamicin	Recovered without repeat surgery
Ajam et al. (2019) [6]	75/female	Bioprosthetic	Bioprosthetic AVR	Acute heart failure	Bioprosthetic aortic	Penicillin + gentamicin; surgery planned	Died before surgery
Campbell et al. (2020) [7]	45/male	Native	Severe mitral regurgitation; dental disease; daily dairy consumption for >2 years	Night sweats, intermittent fever, weight loss	Mitral	Penicillin + gentamicin; mitral valve surgery	Recovered
Tang et al. (2021) [11]	47/male	Native	History of cardiac disease (details not specified); no immunosuppression or probiotic use reported	2 months of fever, headache, myalgia, arthralgia, malaise; palpitations and dyspnea	Aortic	Ceftriaxone and cefoperazone-sulbactam, then piperacillin-tazobactam; mechanical AVR after 3 weeks	Recovered
Present case	75/male	Native	No immunosuppression; no probiotics; no dental procedure	Weight loss, night sweats and chills, afebrile	Aortic + MAIVF	Amoxicillin-clavulanate + gentamicin/rifampicin; valve-sparing repair	Recovered

TABLE 1: Reported cases of Lacticaseibacillus paracasei infective endocarditis in the literature compared with the present case

AVR: aortic valve replacement, CABG: coronary artery bypass graft, IE: infective endocarditis, MAIVF: mitral-aortic intervalvular fibrosa

The diagnosis was particularly challenging because of the patient's atypical presentation. Instead of developing the classical manifestations of IE, he presented with a six-month history of progressive weight loss, night sweats, and chills, without documented fever, and an FDG/PET-CT scan that was not suggestive of endocarditis, opening the differential diagnosis to inflammatory or hematologic disorders. Although fever remains the most common presenting symptom of *Lactobacillus* endocarditis, occurring in approximately two-thirds of reported patients, a substantial proportion present atypically, illustrating that its absence should not delay consideration of IE when persistent inflammatory markers and constitutional symptoms remain unexplained [1].

One of the principal messages of this case concerns the interpretation of blood cultures. Because *Lactobacillus* species are commensal organisms of the oral cavity and gastrointestinal tract, their isolation is frequently regarded as contamination. However, repeated recovery of the same organism from multiple independently collected blood cultures should always raise suspicion for true invasive infection. In our patient, multiple blood culture bottles repeatedly yielded *L. paracasei*, indicating persistent bacteremia and strongly supporting a pathogenic rather than contaminant role. This microbiological pattern fulfilled a major Duke-ISCVID criterion for infective endocarditis and proved decisive in establishing the diagnosis [8]. Previous reports similarly emphasize that repeated positive blood cultures are a hallmark of true *Lactobacillus* endocarditis and should never be dismissed without careful clinical correlation [1-7].

Another notable aspect of this case was the initially non-contributory FDG/PET-CT examination. Despite active infective endocarditis, FDG/PET-CT demonstrated only diffuse mild splenic and bone marrow hypermetabolism without convincing cardiac uptake. Although FDG/PET-CT has become an established

adjunctive imaging modality for prosthetic valve endocarditis, its diagnostic sensitivity is considerably lower in native valve disease because of the relatively small inflammatory burden of vegetations, physiological myocardial glucose uptake, and technical limitations related to cardiac motion [9]. Consequently, a negative or nonspecific PET examination should not be considered sufficient to exclude native valve infective endocarditis when clinical suspicion persists. In our patient, transesophageal echocardiography ultimately established the diagnosis, highlighting its central role in accordance with current European Society of Cardiology recommendations [9].

An additional clinically relevant feature was the discordance between microbiological clearance and persistent structural cardiac infection. Following antibiotic therapy, inflammatory markers progressively diminished and blood cultures became sterile, indicating blood culture clearance. Nevertheless, repeat transesophageal echocardiography demonstrated persistence of the mitral-aortic intervalvular fibrosa (MAIVF) vegetation, with thinning of the MAIVF and cavity formation suspicious for pseudoaneurysm. This discordance between microbiological and structural response underscores the importance of serial imaging during treatment, as a small perivalvular cavity may not be visible on a single examination, and repeated studies in several planes can reveal it. Eradication of bacteremia does not necessarily indicate resolution of structural infection, and progressive structural damage may continue despite apparent clinical improvement. Repeat echocardiography therefore remains essential for reassessing embolic risk and identifying complications requiring surgical intervention [9].

The anatomical extent of infection in our patient deserves comment. Neither TTE nor TEE showed vegetation or other abnormality of the aortic leaflets: both the initial vegetation and the subsequent cavity formation were localized to the MAIVF. However, surgery was performed at another institution, and the only information available to us is a brief communication listing the procedures performed (plication of the free edge of the right coronary cusp, Cabrol commissural sutures, bovine pericardial patch reconstruction of the mitral-aortic trigone, and sinotubular junction annuloplasty). As these techniques address the aortic cusps and root, it is possible that the surgeons identified extension of the infection from the MAIVF to the aortic valve or aortic root that was not apparent on preoperative imaging. Such spread would be anatomically plausible given the continuity of the MAIVF with the non-coronary and left coronary cusps. This interpretation remains speculative in the absence of a detailed operative or histopathological report, and it also illustrates that echocardiography may underestimate the true extent of periannular and valvular involvement. Given that aortic valve repair was required, we consider this case to represent native aortic valve endocarditis with MAIVF involvement, although the vegetation was visualized only on the MAIVF on echocardiography.

Comparison with previously published *L. paracasei* cases further highlights several distinctive aspects of our patient. Most reported patients had an identifiable portal of entry or recognized predisposing factor, including probiotic consumption, prosthetic valve material, significant valvular disease, or recent dental manipulation [2-7]. Conversely, no source of bacteremia could be identified in our patient despite comprehensive evaluation. Furthermore, while surgery has sometimes consisted of valve replacement in previously reported cases, our patient underwent a repair-based surgical approach with an uneventful postoperative course, although the details could not be verified, as discussed above.

This report has several limitations. Surgery was performed at another institution, and no operative or anatomopathological report was available to us. The pseudoaneurysm therefore could not be confirmed, the reason for the aortic cusp repair is unknown, and aortic leaflet involvement, which was not visible on preoperative TTE or TEE, could not be formally confirmed. Our classification of this case as native aortic valve endocarditis rests on the fact that the aortic valve was surgically repaired. In addition, no intraoperative cultures or molecular analysis of the excised tissue were available to confirm the presence of the organism at the site of infection. Finally, as a single case report, our observations cannot be generalized, and the absence of an identifiable portal of entry does not exclude an undetected source. Moreover, follow-up is still ongoing, and we do not know the long-term outcome, in particular the risk of late relapse or recurrent valvular or periannular complications.

The recently published narrative review by Ioannou et al. demonstrated that the aortic valve is the most frequently involved intracardiac site in *Lactobacillus* endocarditis. Surgery is required in more than half of reported patients, and overall mortality remains approximately 17% [1]. Prosthetic valve infection and presentation with shock were independently associated with mortality [1]. Our patient experienced a favorable outcome despite prolonged diagnostic delay, likely owing to persistent microbiological investigation, early echocardiographic diagnosis, appropriate antimicrobial therapy, and timely surgical intervention before the development of overt echocardiographic evidence of extensive MAIVF destruction or systemic complications.

Conclusions

This case highlights the importance of recognizing *Lacticaseibacillus paracasei* as a potential cause of true native aortic valve infective endocarditis, here involving the mitral-aortic intervalvular fibrosa (MAIVF), rather than dismissing it as a contaminant. Diagnosis may be delayed by an indolent clinical presentation, absence of classical signs of infective endocarditis, and non-diagnostic FDG/PET-CT findings. Persistent

bacteremia, careful interpretation of microbiological results, and repeated transesophageal echocardiography were key to establishing the diagnosis and identifying complications. Finally, this case illustrates that blood culture clearance does not necessarily reflect resolution of structural cardiac infection, as MAIVF thinning with cavity formation suspicious for pseudoaneurysm was detected despite antimicrobial therapy, reinforcing the importance of serial imaging and multidisciplinary Heart Team assessment throughout the course of treatment.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

Concept and design: Arthiom Cambour, Mihaela Malanca Onica, Céline Goffinet, Nawel Rezzoug

Drafting of the manuscript: Arthiom Cambour

Critical review of the manuscript for important intellectual content: Mihaela Malanca Onica, Céline Goffinet, Nawel Rezzoug

Supervision: Mihaela Malanca Onica, Céline Goffinet, Nawel Rezzoug

Disclosures

Human subjects: Informed consent for treatment and open access publication was obtained or waived by all participants in this study. **Conflicts of interest:** In compliance with the ICMJE uniform disclosure form, all authors declare the following: **Payment/services info:** All authors have declared that no financial support was received from any organization for the submitted work. **Financial relationships:** All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. **Other relationships:** All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

References

1. Ioannou P, Ziogou A, Giannakodimos I, Giannakodimos A, Baliou S, Samonis G: Infective endocarditis by Lactobacillus species-a narrative review. Antibiotics (Basel). 2024, 13:10.3390/antibiotics13010053
2. Soleman N, Laferl H, Kneifel W, et al.: How safe is safe?--a case of Lactobacillus paracasei ssp. paracasei endocarditis and discussion of the safety of lactic acid bacteria. Scand J Infect Dis. 2003, 35:759-62. 10.1080/00365540310015872
3. Franko B, Vaillant M, Recule C, Vautrin E, Brion JP, Pavese P: Lactobacillus paracasei endocarditis in a consumer of probiotics. Med Mal Infect. 2013, 43:171-3. 10.1016/j.medmal.2013.01.007
4. Kato K, Funabashi N, Takaoka H, et al.: Lactobacillus paracasei endocarditis in a consumer of probiotics with advanced and severe bicuspid aortic valve stenosis complicated with diffuse left ventricular mid-layer fibrosis. Int J Cardiol. 2016, 224:157-61. 10.1016/j.ijcard.2016.09.002
5. Osman A, Taipale M, Najjar M, Osman B: Lactobacillus paracasei endocarditis of bioprosthetic aortic valve presenting with recurrent embolic strokes. Access Microbiol. 2019, 1:e000038. 10.1099/acmi.0.000038
6. Ajam M, Adam O, Yeddi A, Kahlid M, Shokr M, Afonso L: Prosthetic aortic valve endocarditis in a patient with Birt-Hogg-Dube syndrome due to Lactobacillus paracasei. Cardiol Res. 2019, 10:245-8. 10.14740/cr901
7. Campbell RE, Miller A, Afroze A: Native valve endocarditis secondary to Lactobacillus paracasei bacteremia. Consultant. 2020, 60:27-8. 10.25270/con.2020.06.00015
8. Fowler VG, Durack DT, Selton-Suty C, et al.: The 2023 Duke-International Society for Cardiovascular Infectious Diseases criteria for infective endocarditis: updating the modified Duke criteria. Clin Infect Dis. 2023, 77:518-26. 10.1093/cid/ciad271
9. Delgado V, Ajmone Marsan N, de Waha S, et al.: 2023 ESC guidelines for the management of endocarditis. Eur Heart J. 2023, 44:3948-4042. 10.1093/eurheartj/ehad193
10. DeMarco E, DePetrillo J, Qadeer F: Meropenem resistant Lactobacillus endocarditis in an immunocompetent patient. SAGE Open Med Case Rep. 2023, 11:2050313X231152709. 10.1177/2050313X231152709
11. Tang Q, Hao Y, Wang L, et al.: Characterization of a bacterial strain Lactobacillus paracasei LP10266 recovered from an endocarditis patient in Shandong, China. BMC Microbiol. 2021, 21:183. 10.1186/s12866-021-02253-8