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# Bacillus cereus endocarditis: a case-based literature review

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## Summary

*Bacillus cereus*, a gram-positive, rod-shaped bacterium known for both its environmental resilience and its pathogenic potential, has been increasingly recognised as a serious health threat outside the traditional contexts of food poisoning. This narrative review, anchored by a detailed case study, highlights the pathogen's role in rare but severe infections like endocarditis, especially among intravenous drug users, who are particularly vulnerable, among other identified risk factors. The case of a 62-year-old female with a history of intravenous cocaine use who developed *Bacillus cereus* endocarditis underscores the complexities of diagnosing and managing such infections. Despite the challenges posed by the patient's adverse reactions to vancomycin, the mainstay treatment, successful management was achieved through persistent administration adjusted for tolerance and side effects.

This review meticulously compiles all known cases of *Bacillus cereus* endocarditis from the past decades, beginning with the first identified case fifty years ago in 1974. It provides a thorough analysis, identifying various risk factors and outlining the evolution of treatment protocols. This comprehensive approach not only enhances understanding of the pathogen's clinical impact but also clarifies the progression of therapeutic strategies, highlighting the individual adaptations necessary to address this challenging infection effectively.

## Introduction

*Bacillus cereus*, a gram-positive rod-shaped bacterium, exhibits aerobic and facultative anaerobic characteristics. It generates dormant spores in response to adverse environmental conditions like heat and dryness, enabling its survival for extended periods. *B. cereus* belongs to the *Bacillus cereus* group, which also includes *Bacillus anthracis*, the causative agent of anthrax [1]. *B. cereus* was recognised as a pathogenic organism in 1963, and is no longer considered solely a contaminant [2]. It causes gastrointestinal issues, primarily through food-poisoning toxins [3], but is also an opportunistic pathogen that causes local infections and, less frequently, severe systemic infections [1]. Local infections can arise from post-surgical wounds, traumatic injuries, or burns [4]. Systemic infections include bacteraemia (more common in immunocompromised patients than intravenous drug users) [5], meningitis [6], encephalitis, respiratory infections, osteomyelitis, brain and liver abscess, pericarditis [7], and endocarditis [8–10].

*Bacilli* are ubiquitous and therefore frequently cause contamination in the laboratory and of paraphernalia used in intravenous drug consumption [11]. *Bacillus sp.* has historically been the most common bacterial contaminant, being found on 47% of injection paraphernalia [12].

## Case presentation

A 62-year-old female presented to our emergency department with fever and chills. She reported feeling weak for several days and experiencing night sweats. She had a history of weekly intravenous cocaine use.

In 2021, the patient had *Staphylococcus aureus* bacteraemia with an epidural abscess in the cervical spine due to spondylodiscitis. At that time, there was also suspicion of tricuspid valve endocarditis. In 2022, the patient experienced a recurrence of *S. aureus* bacteraemia with probable mitral valve endocarditis, gonarthritis, and a cervical spine abscess.

Clinical examination revealed Janeway lesions and Osler nodes (figures 1 and 2) and possibly splinter haemorrhages, which were difficult to assess due to dry and brittle nails. Transoesophageal echocardiography showed a vegetation on the aortic valve (on the right coronary cusp, sized 8 × 6 mm) and changes of the mitral and tricuspid valve suggestive of involvement of these valves (figure 3). Laboratory results showed a slightly elevated CRP (25 mg/l) with a normal white blood cell count. Due to initial suspicion of recurrent *S. aureus* endocarditis, antibiotic therapy with co-amoxicillin 2.2 g quad 4 h was initiated upon admission.

*B. cereus* was identified in 6/6 blood cultures collected upon the patient's admission, and blood cultures obtained 48h later also yielded positive results. The antibiogram indicated that *B. cereus* was susceptible to vancomycin and clindamycin, and with increased dosage, also susceptible to ciprofloxacin and levofloxacin but resistant to imipenem and co-amoxicillin. With 2 positive major Duke criteria [13] (vegetations in echocardiography, positive blood cultures in 4 sets) and 4 fulfilled minor criteria (fever,

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Janeway lesions, previous suspicion of endocarditis, intravenous drug use), the diagnosis of endocarditis was confirmed.

The therapy was supplemented with vancomycin after the first positive blood culture result. Vancomycin monotherapy was continued throughout with twice daily administration with close monitoring of serum levels (2–3 g/d, target level 15–20 mg/l). Under this therapy, there were recurrent subfebrile temperatures without an increase in inflammatory markers. No new aspects such as septic emboli or other infection foci were identified.

**Figure 1:** Janeway lesions.

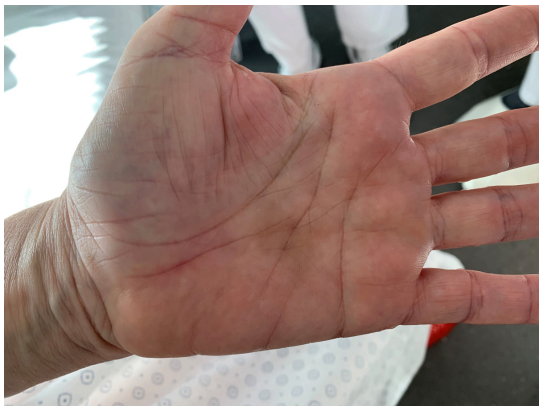


**Figure 2:** Osler node.

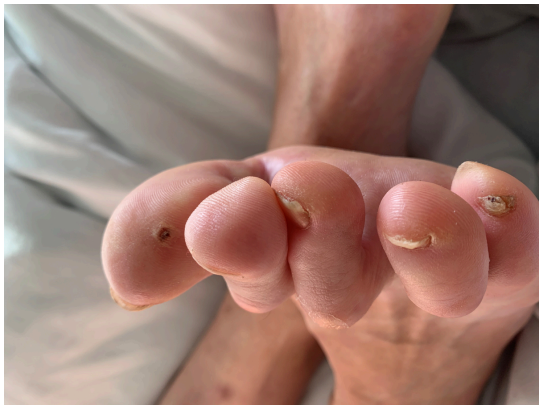
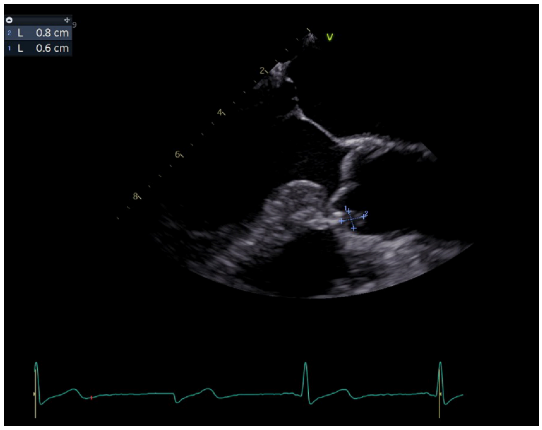


**Figure 3:** Transoesophageal image of vegetation.




After 4 weeks of vancomycin treatment, a symmetrical generalised exanthema occurred, which we interpreted as vancomycin infusion reaction, which is a common adverse non-allergic reaction to vancomycin. It is characterised by flushing, erythema, and maculopapular rash as seen in our patient. There were no signs such as pustules or mucosa involvement, making a more severe differential diagnosis like acute generalised exanthematous pustulosis (AGEP) or drug reaction with eosinophilia and systemic symptoms (DRESS) highly unlikely. Due to the mild reaction, we followed Guidelines and continued vancomycin at a reduced infusion rate. We also initiated therapy with prednisone and an antihistamine, which resulted in a reduction of the rash.

From two days following the start of treatment, all subsequent blood cultures remained negative. After 5 weeks, the patient developed a fever, and SARS-CoV-2 was diagnosed. The infection progressed without complications in the triple-vaccinated patient. Vancomycin therapy was discontinued after 6 weeks. Follow-up echocardiography still showed possible small floating residual structures but no complications. Blood cultures 1 and 3 weeks after the end of antibiotic therapy were negative.

## Discussion

The hypothesis of pathogen introduction into the bloodstream through the utilisation of contaminated drug paraphernalia appears highly plausible. Additionally, a risk arises from the storage of cocaine in basements, where the conditions could support the growth of pathogenic spores, especially considering the widespread presence of *B. cereus* spores in soil and dust. A microbial assessment of the cocaine and paraphernalia used, though pivotal in elucidating causative agents, was unattainable in our case.

*B. cereus* endocarditis typically involves the mitral valve, succeeded by the aortic and tricuspid valves [14]. Vegetations on the right side of the heart have been associated with intravenous drug use, while those on the left side are predominantly linked to prosthetic valves or implanted devices [15]. In our case, the involvement of the aortic valve could be due to pre-existing damage associated with previous endocarditis, although involvement of the aortic valve was not detected echocardiographically in the earlier episodes.

*B. cereus* bacteraemia does not always result in severe disease, as evidenced by case reports in which patients refused antibiotic therapy and the bacteraemia was self-limiting and relatively benign [5, 16]. However, cases of fatal bacteraemia have also been described in immunocompetent patients without signs of endocarditis [17]. Therefore, treatment should be considered in patients even without signs of endocarditis in transoesophageal echocardiography, depending on the risk factors. Despite *B. cereus*'s occurrence as a common contaminant in hospital blood cultures, the presence of multiple positive bottles should be regarded as true bacteraemia [18]. In summary, endocarditis appears to be an unusual consequence of *B. cereus* bacteraemia [19].

A literature search found 35 reported cases, summarised in table 1. The first case with *B. cereus* bacteraemia and endocarditis was from 1974 in a female drug addict with atri-

al septal defect [20]. One year earlier, a case of endocarditis caused by *B. subtilis* had been reported with a patient with intravenous drug use [21]. Decades earlier three cases (1933–1951) with gram-positive Bacillus endocarditis were described [22]. Steen et al. [11] describes 10 cases of *B. cereus* endocarditis having been reported up to 1991, six of which were among people with intravenous drug use, and one each with rheumatic heart disease, mechanical mitral valve, porcine aortic valve, and permanent pacemaker. More cases of *B. cereus* endocarditis have been published since, describing intravenous devices and intravenous drug use as risk factors, as well as valvular and rheumatic heart disease [23] and immunosuppression [24]. In a summary of 38 cases of serious infectious caused by *B. cereus*, all but one patient had a risk factor [19], but multiple case reports have reported *B. cereus* endocarditis among patients without known risk factors [14, 25–30].

The first case of endocarditis with *B. cereus* was treated with intravenous clindamycin [20]. Most *B. cereus* strains seem to be in-vitro susceptible to clindamycin, vancomycin, imipenem, ciprofloxacin, erythromycin, tetracycline, and aminoglycosides (e.g. gentamicin, kanamycin) and chloramphenicol [11, 16, 19, 31]. According to Wright et al. [32], evidence from three studies [31, 33, 34] suggests *B. cereus* susceptibility to gentamicin, imipenem, and vancomycin, with all 240 strains tested responding to these antibiotics. Characteristically, the bacterium is resistant to beta-lactam antibiotics such as penicillin and cephalosporins due to the secretion of beta-lactamase enzymes [9, 11, 19, 31]. An exception to this appears to be susceptibility to mezlocillin. [31] Sensitivity to newer cephalosporins such as cefazolin has been described and successfully used in treatment [35]. Due to pronounced

**Table 1:**
Overview of previously reported cases.

| Year | Author | Risk factors | Treatment | Outcome |
|---|---|---|---|---|
| 1974 | Craig et al. [20] | Intravenous drug use | Clindamycin | Recovered |
| 1978 | Block et al. [38] | Mechanical valve | Tobramycin, chloramphenicol | Died |
| 1978 | Tuazon et al. [8] | Intravenous drug use | Nafcillin | Recovered |
| | | Intravenous drug use | Clindamycin | Recovered |
| | | Intravenous drug use | Clindamycin | Recovered |
| | | Intravenous drug use | Chloramphenicol, gentamicin, erythromycin (patient suffered from endocarditis and endophthalmitis) | Recovered |
| 1979 | Wanvarie et al. [23] | Rheumatic heart disease | Penicillin, gentamicin, streptomycin | Died |
| 1979 | Weller et al. [16] | Intravenous drug use | Ampicillin, oxacillin, and gentamicin, then clindamycin and kanamycin, total 4 weeks, no vegetations in echo | Recovered |
| 1982 | Oster et al. [39] | Porcine aortic valve | Clindamycin, surgery | Recovered |
| 1987 | Sliman et al. [19] | ICD and breast implant | Clindamycin, surgery, 4 weeks | Recovered |
| 1992 | Steen et al. [11] | Mechanical valve | Vancomycin, surgery, 6 weeks | Recovered |
| 1993 | Tomomasa et al. [25] | No risk factors | Not reported | Recovered |
| 1994 | Yamamura et al. [40] | Mechanical valve | Amikacin, minocycline | Recovered |
| 1998 | Martin Cadenas et al. [41] | Mechanical valve | Vancomycin, gentamycin, rifampicin, surgery | Recovered |
| 1999 | Castedo et al. [42] | Mechanical valve | Vancomycin, gentamicin, rifampicin, surgery, 6 weeks | Recovered |
| 2005 | Cone et al. [24] | Leukaemia | Penicillin, vancomycin, ciprofloxacin | Died |
| 2007 | Shalev et al. [36] | ICD | Vancomycin, gentamicin, 6 weeks | Recovered |
| 2008 | Abusin et al. [35] | Pacemaker | Cefazolin, 6 weeks | Recovered |
| 2012 | Barraud et al. [43] | Pacemaker | Vancomycin (later replaced with amoxicillin, then piperacillin), gentamicin (later replaced with ofloxacin), surgery | Died |
| 2012 | Thomas et al. [37] | Not intravenous drug use | Daptomycin, ampicillin (later replaced with ceftriaxone), 6 weeks, surgery | Recovered |
| 2012 | Oh et al. [27] | No risk factors | Ceftriaxone, vancomycin, 6 weeks, surgery | Recovered |
| 2013 | Sharma et al. [44] | Leukaemia | Vancomycin, meropenem | Recovered |
| 2013 | Ngow et al. [15] | Former intravenous drug use | Cefuroxime, 6 weeks | Recovered |
| 2015 | Shah et al. [45] | Pregnant intravenous drug use | Daptomycin, then vancomycin, 5 weeks | Recovered |
| 2015 | Kitazawa et al. [26] | No risk factors | Vancomycin, 9 weeks, surgery | Recovered |
| 2016 | Wright et al. [32] | Central venous catheter | Vancomycin, piperacillin-tazobactam, 6 weeks, surgery | Recovered |
| 2017 | Soudet et al. [28] | No risk factors | Piperacillin-tazobactam + teicoplanin, changed to rifampicin + levofloxacine, 6 weeks | Recovered |
| 2018 | Gopinathan et al. [14] | Baby with VSD repair | i.v. vancomycin 10 days, then oral linezolid for 4 weeks | Recovered |
| | | No risk factors | i.v. vancomycin 6 weeks | Recovered |
| 2018 | Ren et al. [29] | No risk factors | Ampicillin, clindamycin, and vancomycin, then only vancomycin, 6 weeks | Recovered |
| 2020 | Nallarajah et al. [30] | No risk factors | Ciprofloxacin, 8 weeks | Recovered |
| 2021 | Meledathu et al. [46] | Mitral valve repair | Vancomycin and piperacillin-tazobactam, then meropenem, then ciprofloxacin, 6 weeks | Recovered |
| 2022 | Ribeiro et al. [47] | Central venous catheter | Vancomycin, 6 weeks | Recovered |
| 2023 | De Carvalho et a. [48] | Pacemaker | Daptomycin, 4 weeks | Recovered |
| 2023 | Current case | Intravenous drug use | Co-amoxicillin, then vancomycin, total 6 weeks | Recovered |
| 2024 | Fukushima [49] | Prosthetic aortic valve | Vancomycin, 6 weeks | Recovered |

ICD: implantable cardioverter-defibrillator; VSD: ventricular septal defect; i.v.: intravenous.

side effects (e.g. with aminoglycosides, and chloramphenicol), not all susceptible antibiotics are suitable for therapy.

The patient's adverse reaction to vancomycin highlights the delicate equilibrium clinicians must maintain between drug efficacy and patient tolerance. Such side effects, while not life-threatening, can severely impact patient compliance and comfort. These concerns are particularly pronounced among intravenous drug users due to the challenges in ensuring adherence to treatment regimens. To mitigate these issues, we maintained the original antibiotic despite side effects and administered a six-week treatment within the hospital. This conservative approach was necessitated by limited literature on alternative antibiotics for this specific pathogen. Outpatient intravenous antibiotic treatment emerges as a possibility worthy of consideration. However, this is complicated by the need for twice-daily administration and for regular therapeutic drug monitoring to ensure appropriate dosing and monitoring for toxicity, thus reinforcing the need for inpatient oversight in complex cases such as this.

The successful non-surgical treatment of our patient highlights that conservative management with antibiotics is effective for native valve endocarditis and intravenous drug users. While *B. cereus* endocarditis typically carries a higher risk in prosthetic valve patients and often necessitates material replacement, conservative treatment can still succeed [35, 36], particularly in those who respond quickly to antibiotics or as an alternative among patients at high risk for peri-operative complications [36]. Nonetheless, valve replacement is sometimes imperative for native valve infections unresponsive to antibiotics alone [14, 27, 37].

One notable limitation is the lack of data, which makes it unclear whether all the reported cases met the Duke criteria for definitive endocarditis. Our case was diagnosed according to the updated Duke criteria 2023 [13].

## Conclusion

In the clinical management of endocarditis, consideration of atypical aetiologies like *B. cereus* is critical, especially in intravenous drug users, who face an escalated risk of developing uncommon infections due to exposure to contaminated paraphernalia. Prompt initiation of antimicrobials attuned to the specific susceptibilities of the pathogen, toxicity profile, and local resistance patterns is essential. It is equally important to carefully assess potential adverse effects associated with the selected antibiotics. This case report, reinforced by a comprehensive literature review, underscores the imperative for ongoing research and the dissemination of knowledge to refine therapeutic strategies for rare infectious diseases and to provide alternatives when severe side effects arise.

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