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CLINICAL REASONING: CASE REPORT

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First reported human *Castellaniella defragrans* bacteraemia: a case report

Morgane Nédellec[1*], Titouan Cerruti[1*], Damien Jacot[2], Gilbert Greub[2,3], Malik Benmachiche[1]

1 Division of Internal Medicine, Lausanne University Hospital, Lausanne, Switzerland; 2 Institute of Microbiology, Lausanne University Hospital and University of Lausanne, Lausanne, Switzerland; 3 Division of Infectious Diseases, Lausanne University Hospital, Lausanne, Switzerland

Summary

Castellaniella defragrans is an environmental Gram-negative, non-fermenting β-proteobacterium that is not known as a human pathogen. Here, we report the first documented case of *C. defragrans* bacteraemia in an 82-year-old man with monoclonal B-cell lymphocytosis, subsequently diagnosed with chronic lymphocytic leukaemia, who presented with fever, confusion and clinical signs of sepsis. Blood cultures showed sustained growth of *C. defragrans* for 7 days. Imaging revealed pulmonary consolidations and mediastinal infiltration suggestive of mediastinitis. Further investigations did not identify a clear additional infectious focus. In addition, an immunological evaluation revealed severe cellular and humoral immunosuppression. Antimicrobial therapy with high-dose meropenem led to bacteraemia clearance and progressive clinical improvement. Treatment was completed with oral sulfamethoxazole–trimethoprim for a total of five weeks. This case highlights the potential of environmental microorganisms to cause severe infections in immunocompromised hosts.

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Correspondence
Morgane Nédellec
Morgane.Nedellec[at]chuv.ch

***** Equal contribution as first authors

Background

A few bacterial species are responsible for most sepsis cases, a common cause of mortality worldwide. *Escherichia coli*, staphylococci, *Pseudomonas aeruginosa* and *Klebsiella* spp. represent about 68% of microorganisms identified in positive blood cultures at the microbiology laboratory of the University Hospital of Lausanne, whilst no other single microbial species accounts for more than 5% of positive blood cultures [1]. Immunocompromised individuals are particularly susceptible to severe bacterial infections because of impaired host defences [2]. Here, *Castellaniella defragrans* (previously categorised under the *Alcaligenes* genus) was isolated from a patient with chronic lymphocytic B-cell leukaemia (see the Presentation section below). This Gram-negative, non-fermenting beta-proteobacterium is commonly found in agricultural environments and is not known as a human pathogen [3]. Thus, this case represents the first documented *C. defragrans* bacteraemia to date.

Case presentation

An 82-year-old European white male was transferred to the Lausanne University Hospital, Switzerland, following sepsis of undetermined origin, with sustained bacteraemia due to *Castellaniella defragrans*. Relevant medical history included monoclonal B-cell lymphocytosis, moderate aortic stenosis and well-controlled arterial hypertension.

The patient presented in a secondary care hospital with deterioration of his general condition, fever, intermittent chest pain and acute confusion. Clinical examination revealed a systolic aortic murmur, psychomotor slowing and oropharyngeal candidiasis. He was hypotensive without the need for vasopressor support, and scored 14 on the Glasgow Coma Scale (GCS). He was admitted to the intensive care unit for sepsis with a Sequential Organ Failure Assessment (SOFA) score of 6. Blood cultures revealed *Castellaniella defragrans* (4 positive bottles out of 8), susceptible to penicillins and carbapenems. This bacterium was also isolated in one sputum sample. Laboratory work-up showed agranulocytosis with a white-cell count of 16.5 G/l (normal range of 4–10 G/l), consisting of lymphocytes (15.9 G/l) and neutrophils (0.4 G/l), and marked inflammation

with a C-reactive protein level of 307 mg/l (normal <10 mg/l). Transthoracic echocardiography confirmed previously known moderate aortic stenosis without any finding compatible with infective endocarditis. A thoraco-abdominopelvic CT scan revealed mediastinal fat infiltration suggestive of mediastinitis and alveolar consolidation in the right upper lobe. An empirical antibiotic therapy with cefepime (1.5 g every 12 hours) was initiated. Given the lack of prior human infections involving *C. defragrans*, clinical management was challenging, particularly because of the absence of data regarding typical secondary dissemination, infection severity and recommended treatment duration. The patient was thus transferred to our tertiary care hospital for expert evaluation.

Upon arrival, the patient experienced worsening neurological status (GCS of 13 points), had persistent high-grade fever (up to 40 °C) and sustained bacteraemia for 7 days. Profound cellular and humoral immunosuppression, attributed to the transformation of monoclonal lymphocytosis into chronic lymphocytic leukaemia, was documented, with agranulocytosis, a CD4+ T-cell count of 10 cells/mm³ (normal range of 490–1640 cells/mm³) and IgG levels of 3.84 g/l (normal range of 6.10–16.16 g/l). Infective endocarditis was considered unlikely because the Duke criteria were not fulfilled, including the absence of vegetation on both transthoracic (TTE) and transoesophageal echocardiography (TEE). An extensive infectious screening excluded any additional concomitant infection that could have contributed to the patient's severe clinical presentation. Upper endoscopy confirmed intact oesophageal mucosa, with no evidence of oesophageal candidiasis, and excluded a digestive origin for the mediastinal infiltration. Cerebro-thoraco-lumbar magnetic resonance imaging (MRI) and a second CT scan ruled out a secondary site of infection and showed a stable mediastinal infiltration. The analysis of a cerebrospinal fluid sample obtained through lumbar puncture revealed lymphocytic infiltration consistent with chronic lymphocytic leukaemia (CLL). Lymphocytic infiltration was also identified in the pleural fluid, without evidence of concurrent infection. A positron emission tomography-CT (PET-CT) scan was performed to complete the extension assessment and confirmed diffuse mediastinitis and multiple consolidations in the middle lobe and the lingular segments of the left lower lobe (figure 1), without evidence of infective endocarditis or any other infectious focus explaining the sustained bacteraemia. We performed a mediastinoscopy with biopsy of the mediastinum and adjacent lymph nodes to understand the aetiology of the mediastinal infiltration. The mediastinoscopy revealed lymphocytic infiltration consistent with CLL. Both culture and DNA analyses of these samples showed no evidence of *Castellaniella defragrans* presence or infection. However, the mediastinal biopsy was performed only 22 days after the initiation of antibiotic therapy.

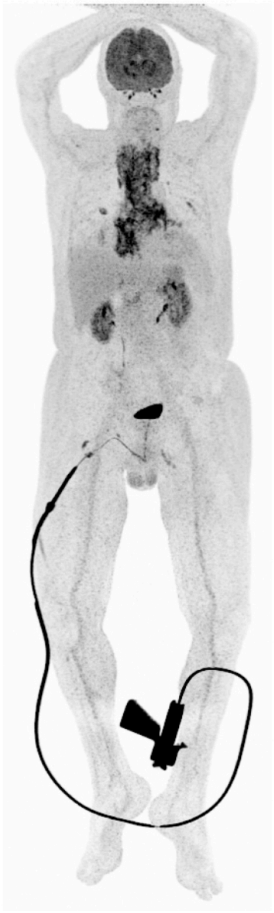

Figure 1: PET-CT showing diffuse mediastinitis and multiple consolidations in the middle lobe and the lingular segments of the left lower lobe.

The antibiotic therapy selected for this patient initially consisted of cefepime and was shortly escalated to piperacillin–tazobactam (4.5 g every 8 hours), based on the minimum inhibitory concentration (MIC) of the isolated strain (see the Bacteriology section below). Given the patient's neurological deterioration, the unclear source of infection and the need for enhanced central nervous system penetration, therapy was subsequently switched to high-dose meropenem (2 g every 12 hours). This regimen resulted in effective infection control, with blood cultures turning negative after 8 days and progressive improvement in neurological function. Following favourable clinical evolution, antimicrobial therapy was de-escalated to oral sulfamethoxazole-trimethoprim (800/160 mg every 12 hours). The total antibiotic treatment lasted for 5 weeks, including 4 weeks following the last negative blood cultures, ensuring sufficiently prolonged therapy given the possibility of infectious mediastinitis.

In sum, based on clinical, microbiological and radiological findings, a diagnosis of sepsis with sustained *Castellaniella defragrans* bacteraemia was established in an immunocompromised patient with chronic lymphocytic leukaemia. However, the exact source of infection was never formally identified. Pneumonia of the right upper lobe, infectious mediastinitis or an unidentified primary bacteraemic source with secondary infectious foci were possible explanations.

Bacteriology

C. defragrans was isolated from aerobic blood cultures (BD BACTEC™ Plus Aerobic/F) within 24 h to 4 days, and identified by MALDI-TOF (Biotyper® Sirius, Bruker). After 24 h of subculture, milky, slightly mucoid colonies grew on Chocolate II and Columbia 5% sheep blood agar (Figure 2A and B), with faint growth on MacConkey. MICs (mg/l) (blood culture vs sputum isolates)

determined by Etest (Liofilchem®) were comparable between strains: piperacillin–tazobactam, 0.75 vs 1; ceftazidime, 1.5 vs 2; cefepime, 8 vs 8; meropenem, 0.047 vs 0.047; co-trimoxazole, 0.047 vs 0.064; ciprofloxacin, 0.032 vs 0.032.

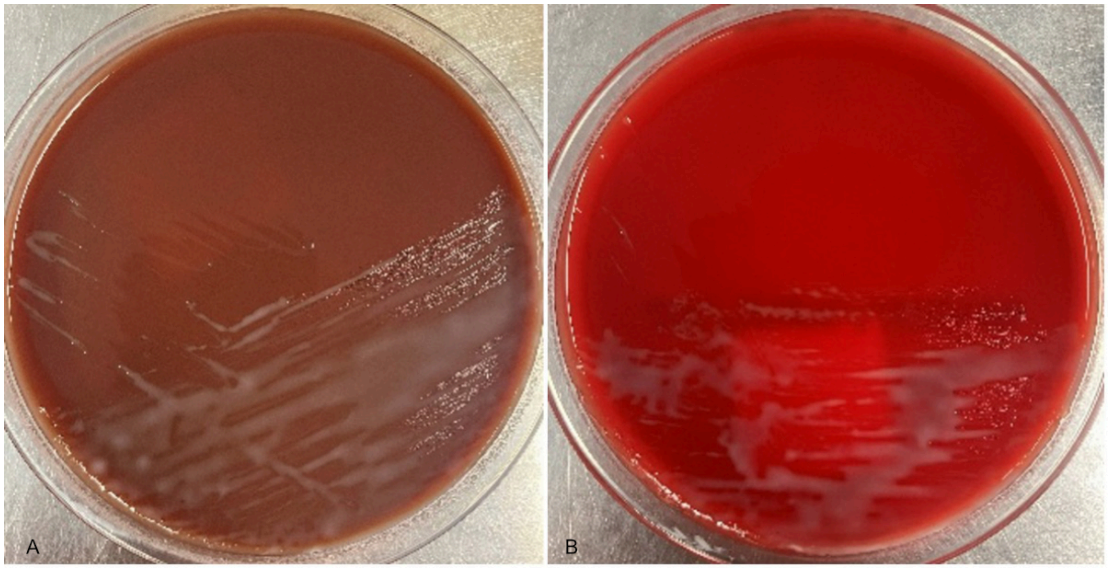

Figure 2: A: BD Chocolate II Agar plate showing milky and slightly mucoid colonies. B: BD Columbia Agar plate showing milky and slightly mucoid colonies.

Discussion

This is the first documented case of bacteraemia caused by *Castellaniella defragrans* in humans, representing the first confirmed report of this bacterium as a human pathogen. Although previously identified as part of the commensal lung microbiota in immunocompromised individuals, particularly in lung transplant recipients, its clinical significance had not been established [4]. A prior report described septic arthritis caused by a Gram-negative bacillus closely related to the *Alcaligenes - Bordetella* complex, with high genetic similarity to *Alcaligenes defragrans*; however, species-level identification was not possible with the molecular tools available at that time [5].

The origin of the sustained bacteraemia remains uncertain in the present case. Our differential diagnosis included right upper lobe pneumonia, infectious mediastinitis or an unidentified primary bacteraemic source with secondary infectious foci. The admission CT scan revealed consolidation in the right upper lobe, and *C. defragrans* was also isolated from a sputum sample, suggesting pneumonia. However, uncomplicated pneumonia is rarely associated with sustained bacteraemia, making this diagnosis uncertain. Infectious mediastinitis was also considered a possible source. However, no entry portal was identified, and upper endoscopy excluded oesophageal mucosal involvement as a potential translocation site. Mediastinal infiltration was most likely attributable to CLL involvement, supported by mediastinoscopy findings showing only leukaemic infiltration without evidence of *C. defragrans* infection. Nevertheless, cultures and eubacterial PCR of mediastinal tissue were conducted more than three weeks after antibiotic initiation, limiting the sensitivity of both microbiological approaches. Furthermore, DNA analyses were performed using a 16S rRNA gene PCR on paraffin-embedded tissue. Such materials are notoriously less sensitive than native specimens because of DNA fragmentation resulting from formalin cross-linking and paraffin inhibition. A follow-up PET-CT after completion of antimicrobial therapy could have helped differentiate an infectious from a chronic lymphocytic leukaemia-related aetiology; however, complete clinical recovery and healthcare resource considerations precluded additional imaging.

This case illustrates that environmental or commensal organisms, previously not considered pathogenic, can cause severe infections in immunocompromised hosts. Further studies are needed to better define the pathogenic potential, optimal management and clinical outcomes of *C. defragrans* infections in humans.

Notes

Informed consent: Written informed consent was obtained from the patient for the publication of the case report and the accompanying images.

Use of AI: Generative AI (ChatGPT-4) was used for grammar and language verification throughout the manuscript. The authors subsequently reviewed each sentence.

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Potential competing interests

All authors have completed and submitted the International Committee of Medical Journal Editors form for disclosure of potential conflicts of interest. No potential conflict of interest related to the content of this manuscript was disclosed.

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