Bacillus cereus causing widespread necrotising skin infection in a diabetic person

Flurina Michelotti
MBBS, St James’s University Hospital, Leeds, UK
(current post: Dewsbury and District Hospital, Dewsbury, UK)

H Jonathan Bodansky
MB ChB, MD, FRCP, St James’s University Hospital, Leeds, UK

Correspondence to:
Flurina Michelotti, Dewsbury and District Hospital, Halifax Road, Dewsbury WF13 4HS, UK; email: flurina.michelotti@gmail.com

Received: 31 January 2015
Accepted in revised form: 25 February 2015

Abstract

Bacillus cereus is a Gram-positive aerobic bacterium recognised for causing toxin-mediated gastrointestinal infections. We report a unique case of B. cereus cutaneous infection causing extensive skin necrosis in a person with type 2 diabetes mellitus. Examination revealed erythema, pitting oedema and right ankle tenderness. Elevated inflammatory markers supported a diagnosis of cellulitis, and intravenous flucloxacinillin was initiated as per local hospital guidelines. Clinical response was poor with the development of spreading cellulitis, desquamated blisters and superficial necrosis. In response to swab culture and sensitivity results, vancomycin was commenced resulting in gradual resolution.

Cutaneous necrotising B. cereus infection in a diabetic patient is unusual. The only other reported case was of a necrotising fasciitis affecting the arm, resulting in tissue loss. The challenge lies in recognising this atypical pathogen and initiating sensitive antimicrobial therapy which includes vancomycin, ciprofloxacin, doxycycline and novel agents such as linezolid. Copyright © 2015 John Wiley & Sons.


Key words

Bacillus cereus; necrosis; infection; diabetes; antibiotics

Introduction

Bacillus cereus is a widely distributed environmental Gram-positive aerobic bacterium. It is commonly recognised for its toxin mediated gastrointestinal infections, notably diarrhoea from infected warm buffet rice. Cases of severe and sometimes life-threatening systemic infections affecting the heart, eyes, respiratory tract and skin have been described. Here, we report an unusual presentation affecting a man with type 2 diabetes mellitus (T2DM) who developed a dramatic necrotising rash.

Case history

A 72-year-old man, with T2DM, presented following a fall. Past medical history included hypertension, cerebrovascular disease, obesity, chronic obstructive pulmonary disease, sleep apnoea and ischaemic heart disease. His drug history was extensive including bumetanide, bisoprolol, spironolactone, indapamide, atorvastatin, Lansoprazole, ramipril, aspirin, GTN spray, liraglutide and Novomix 30 insulin. He was an ex-smoker who enjoyed taking his dog for walks through long grass.

On examination, he was alert (GCS 15/15), pyrexial (38.1°C), tachycardic and normotensive. There was marked tenderness over the right medial malleolus (10/10), erythema and pitting oedema, with no other clinical signs.

Initial blood results were: white cells 19.8x10⁹/L, neutrophils 17.75 x10⁹/L, C-reactive protein 100mg/L, urea 9.8mmol/L, creatinine 127µmol/L, eGFR 48 (baseline 58), sodium 133mmol/L, potassium 4.8mmol/L. Random blood glucose was 20.1mmol/L. No recent HbA1c was available to compare, with the most up to date being 70mmol/mol four months prior to presentation. Blood cultures were negative and chest X-ray was unremarkable.

In light of physical examination and initial investigations, the clinical impression was of a right ankle cellulitis. This was managed as per local trust guidelines with IV flucloxacillin. Additionally, IV insulin was commenced and nephrotoxins omitted.

Clinical response was poor, with the development of two desquamated weeping blisters (measuring 2cm and 4cm in diameter) over the posterior right calf on the third day. Further blisters continued to appear and, by day eight, there were areas of painful haemorrhagic superficial necrosis up to the knee (as illustrated in Figure 1). Pain continued to be a key feature, as did persistent temperature spikes. C-reactive protein reached a peak on the fourth day at 314mg/L, and fell continuously thereafter. This
in addition to initial negative wound cultures explained why, despite a refractory clinical cellulitis, flucloxacillin was continued. Alternative diagnoses of erysipelas and necrotising fasciitis were considered.

On day 10 the wound swab cultures and sensitivities were released identifying *B. cereus* (+++) with enteric flora (+++) sensitive to vancomycin. Following a change of antibiotics, there was a marked improvement with the necrotic rash receding. Thereafter, the patient’s clinical condition improved, with the necrotic rash slowly receding. By day five, vancomycin was switched to oral doxycycline to complete a total of 14 days of targeted antibiotic therapy. The patient went home after 16 days in hospital with resolving cutaneous lesions.

Four months after the initial presentation, the patient reported resolution of his condition.

**Discussion**

*B. cereus* is a widely distributed organism associated with a variety of toxin-mediated infections, including gastroenteritis, pneumonia, meningitis, endocarditis, osteomyelitis and necrotising fasciitis.1–5 Previously, other than enteritis, pneumonia, meningitis, endocarditis, osteomyelitis and necrotising fasciitis, it had been considered to be contaminated, and thus accurate epidemiological data are lacking.1

Cutaneous *B. cereus* skin infection is frequently related to open wounds, haematological malignancies and drug-induced neutropenia.1 Very rarely, *B. cereus* may produce infections in immunocompetent individuals,6 and has been associated with an outbreak among military cadets.7 Diabetes predisposes to cutaneous and other infections, but infection with *B. cereus* is rare. There is only one other reported diabetic case where *B. cereus* caused necrotising fasciitis and myonecrosis of the arm resulting in tissue loss.8 There is a wide spectrum of its effects on the skin ranging from superficial necrosis to necrotising fasciitis and myonecrosis. Tissue necrosis and haemolysis are in response to the toxins produced, which include phospholipase, lecithinase and enterotoxins.1,4,9

In non-traumatic cases, the mode of entry for the organism may be through microscopic skin abrasions in the hands and/or feet.1,4 *B. cereus* is environmentally widespread in many types of soils, sediments, dust and plants.1 This is significant as an entry point through minor skin abrasions. For example, in the case presented here, infection is likely to have occurred when the patient walked his dog in long grass, sustaining a minor abrasion to the legs. Moreover, his poor glycaemic control in itself is a risk factor for the development of infection.

The challenge in treating *B. cereus* infections lies initially in recognising the presence of this atypical pathogen. Helpful clinical features include severe pain and low-grade pyrexia, followed by spreading erythema and the development of non-pruritic vesicular lesions.5 In the previous reported case where *B. cereus* was isolated in a patient with diabetes mellitus, the initial clinical picture is similar to that which we have presented in this case. Pain out of proportion to the clinical picture and swelling of the left upper limb preceded a rapidly-spreading diffuse purpuric skin rash. Unlike the case discussed here, the rash extended at a more rapid rate involving the lower limbs, and resulted in severe necrotising fasciitis and myonecrosis of the upper limb requiring surgical exploration. Similarly, clinical response was correlated to commencing vancomycin in response to cultures.

Since the initial presentation would appear to be a cellulitis, β-lactam antibiotics would be the usual initial treatment, to which *B. cereus* is resistant.1,2,8–10 It is important to be aware of a failure to respond to antibiotic therapy and consider adding or switching antimicrobial therapy to cover for atypical infections, even if the culture results are initially negative or still awaited.

Studies have shown sensitivities to a range of antibiotics including chloramphenicol, ciprofloxacin, gentamicin, vancomycin, tetracycline, levofloxacin, linezolid, moxifloxacin, daptomycin and streptomycin.2,9,10 Choosing a suitable antibiotic will depend on individual culture and sensitivities and will require consideration of cost and adverse effects. Linezolid, for example, requires blood count and visual acuity monitoring; and with it having greater risks for toxicity is not a first-line choice. However, with reports of *B. cereus* resistance to erythromycin and tetracyclines emerging across Europe and in the United States,10 this may change and there may be a rise in the number of infections managed with more novel antibiotics including daptomycin and linezolid.

In conclusion, despite *B. cereus* cutaneous infection being uncommon, it is important to consider it as a differential diagnosis when presented with a worsening cellulitis unresponsive to conventional therapy. This is especially important as it can produce a dramatic necrotising rash that may progress to necrotising fasciitis and myonecrosis, thus making early recognition and appropriate therapy vital.

The challenge lies in prompt recognition and initiating sensitive antimicrobial therapy. As *B. cereus* is resistant to conventional β-lactams, consideration of alternative cost-effective, first-line empirical antibiotics sensitive to a number of atypical pathogens may be indicated in diabetic patients.

**Declaration of interests**

There are no conflicts of interest declared.

The patient gave informed consent for the publication of this article.

**References**

References are available online at www.practicaldiabetes.com.
References