# Fulminant *Bacillus cereus* food poisoning with fatal multi-organ failure

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## SUMMARY

This case represents a rare fulminant course of fried-rice associated food poisoning in an immunocompetent person due to pre-formed exotoxin produced by *Bacillus cereus*, with severe manifestations of sepsis, including multi-organ (hepatic, renal, cardiac, respiratory and neurological) failure, shock, metabolic acidosis, rhabdomyolysis and coagulopathy. Despite maximal supportive measures (continuous renal replacement therapy, plasmapheresis, N-acetylcysteine infusion and blood products, and broad-spectrum antimicrobials) and input from a multidisciplinary team (consisting of infectious diseases, intensive care, gastroenterology, surgery, toxicology, immunology and haematology), mortality resulted. This case is the first to use whole genome sequencing techniques to confirm the toxigenic potential of *B. cereus*. It has important implications for food preparation and storage, particularly given its occurrence in home isolation during the COVID-19 pandemic.

## BACKGROUND

*Bacillus cereus* is an aerobic and facultatively anaerobic, spore-forming, gram-positive *Bacillus*.[1] *B. cereus* is ubiquitous in the environment, and is commonly found in small quantities in raw, dried and processed foods, particularly in rice or pasta. Bacterial food poisoning due to *B. cereus* is uncommon though well known[1 2]; however, it is rarely fatal in immunocompetent individuals. It usually results in a self-limited gastroenteritis, requiring supportive treatment.[3–5] *B. cereus* causes two distinct syndromes: emetic and diarrhoeal. The emetic syndrome, which resembles *Staphylococcal aureus* enterotoxin foodborne illness, develops after a short incubation period (0.5–6 hours)[1 6] due to the emetic toxin, cereulide. The diarrhoeic syndrome is caused by heat labile enterotoxins produced in the gut following the consumption of contaminated food. The incubation period of diarrhoeal syndrome is longer (6–24 hours).[1 2] Previous cases implicated cereulide as a mitochondrial toxin,[5 7–11] with both positive and fatal outcomes. Cereulide is responsible for a dysfunction of the mitochondrial beta-oxidative process and thereby can evoke organ failure.

We describe a rare fatal case of emetic food poisoning of *B. cereus* causing multi-organ failure in an immunocompetent person, and the first to use whole genome sequencing (WGS) techniques to confirm its toxigenic potential.

## CASE PRESENTATION

A 40-year-old woman of Chinese ethnicity was admitted from the emergency department with 12-hour history of profuse vomiting, abdominal pain and diaphoresis, followed by syncope, tonic and decorticate positioning, bilateral exotropia and unresponsive state. Symptom onset started 1 hour after consumption of reheated fried rice meal consisting of peas, turkey, eggs and rice, which was prepared 1 week prior. The meal was left to cool overnight (approximately 12-hour duration) and kept in the fridge since then. She had also eaten canned pumpkin and mushroom soup, with added fresh button mushrooms. Her four children (aged 9 years, 11 years, 14 years and 17 years) consumed the same food, however, experienced mild abdominal pain and vomiting shortly after the meal, not requiring medical attention. Her medical history included gastric ulcer during first pregnancy and vitamin D deficiency. She took no regular over-the-counter medications. She consumed packet chicken herbal soup the day prior. She had no known allergies. There was no recent travel. She was of Chinese origin, and lived in Australia for at least 15 years.

At presentation, she appeared unwell, peripherally shut down with Glasgow Coma Scale 3. Her vital signs included low-grade fever and blood pressure of 69/49 mm Hg, despite metaraminol and oxygen saturations of 98% on 100% fractional inspired oxygen via non-rebreather mask, with normal respiratory and heart rate. Initial examination revealed a distended abdomen, that was firm, with feculent output from nasogastric tube. There were decreased breath sounds bilaterally, and normal cardiac examination. Point of care abdominal sonography revealed free fluid in abdomen supra-pubically.

## INVESTIGATIONS

Investigations revealed high leucocyte count with predominantly neutrophilia. Blood film revealed toxic granulation and marked neutrophil vacuolation, left shift of granulocytes, red cell fragments and thrombocytopenia, concerning for microangiopathic haemolytic anaemia.

There were features of inflammation with high procalcitonin and ferritin; however, C reactive protein (CRP), synthetic liver failure and encephalopathy with rhabdomyolysis, renal failure with mixed metabolic acidosis and coagulopathy were normal (table 1). There was evidence of hepatitis B past infection and immunity (hepatitis B core Ab positive, Ag negative and surface Ab positive at 578.0 mIU/mL). There was no evidence of acute hepatitis A infection, or hepatitis C (hepatitis C Ab negative). Testing for hepatitis D and E was not performed.

**Table 1** Laboratory details and clinical course

| Time from admission (hour) | 0 | 4 | 12 | 21 | 38 | 45 | 72 |
|---|---|---|---|---|---|---|---|
| Venous base excess, mmol/L | −21 | | | | | | |
| Venous pH | 6.71 | | | | | | |
| Arterial base excess, mmol/L | | −18 | −10 | −10 | −13 | −10 | 25 |
| Arterial pH | | 7.13 | 7.31 | 7.25 | 7.22 | 7.31 | 7.35 |
| Bicarbonate, mmol/L | 11 | <10 | 14 | 17 | 14 | 16 | 14 |
| Anion gap, mEq/L | 37 | >38 | 32 | 23 | 28 | 27 | 25 |
| Haemoglobin, g/L | 132 | 125 | 111 | 109 | 94 | 87 | 94 |
| Haematocrit, % | 43 | 39 | 34 | 32 | 29 | 25 | 28 |
| White cell count, $\times 10^9$/L | 27.7 | 26.3 | 22.3 | 20.0 | 17.1 | 13.4 | 11.0 |
| Neutrophils, per µL | 22 500 | 21 000 | 20 200 | 18 900 | 16 100 | 12 500 | 10 000 |
| Platelets, $\times 10^9$/L | 229 | 205 | 132 | 102 | 62 | 44 | 22 |
| Glucose, mmol/L | 3.2 | 15.2 | 11.1 | 4.6 | 9.9 | 7.6 | 6.5 |
| Sodium, mmol/L | 143 | 136 | 141 | 138 | 138 | 139 | 139 |
| Potassium, mmol / L | 4.8 | 7.5 | 5.5 | 4.8 | 3.6 | 3.8 | 4.4 |
| Chloride, mmol/L | 101 | 96 | 101 | 103 | 103 | 100 | 104 |
| Carbon dioxide, mm Hg | 110 | 33 | 30 | 37 | 34 | 31 | 25 |
| Creatinine, µmol/L | 311 | 232 | 203 | 114 | 37 | 64 | 59 |
| Urea, mmol/L | 10.8 | 10.8 | 9.7 | 5 | 4.5 | 3.9 | 3.6 |
| AST, U/L | 5473 | 9726 | 11 166 | 11 600 | 8664 | 4681 | 3195 |
| ALT, U/L | 3452 | 5446 | 5511 | 7579 | 5205 | 2270 | 2423 |
| GGT, U/L | 92 | 105 | 85 | 88 | 78 | 44 | 41 |
| ALP, U/L | 102 | 112 | 102 | 112 | 137 | 109 | 127 |
| Total bilirubin, umol/L | 18 | 19 | 35 | 47 | 70 | 56 | 70 |
| Amylase, U/L | | 403 | | | | | |
| Lipase, U/L | | 115 | | | | | |
| Lactate dehydrogenase, U/L | | 11 087 | | >4500 | 8770 | 4000 | 3465 |
| Creatine kinase, U/L | | 12 200 | | | 115 082 | 31 876 | 65 113 |
| Phosphate, mmol/L | 5.5 | 4.23 | | 1.29 | 1.79 | 1.11 | 0.87 |
| Lactate, mmol/L | 24 | 22 | 15 | 12.4 | 16 | 13.8 | 12.8 |
| Ammonia, ug/dL | | | | 320 | | 147 | 229.2 |
| CRP, mg/dL | <4 | <4 | | <4 | | <4 | <4 |
| Ferritin, ug/L | | | 27 657 | | | | |
| Procalcitonin, ug/L | | 5.57 | | 3.6 | | | |
| INR | 5.5 | 5.9 | 4.2 | 6.7 | 8.10 | 2.2 | >10.0 |
| PT, s | 75 | 80 | 58 | 90 | 108 | 31 | >120 |
| APTT, s | 118 | 144 | 63 | 69 | 76 | 42 | 55 |
| D-Dimer, mg/L | | >10.00 | | >10.00 | >10.00 | >10.00 | >10.00 |
| Fibrinogen, g/L | | 1.8 | 1.7 | 1 | 1.3 | 1.9 | 0.9 |

ALP, alkaline phosphatase; ALT, alanine aminotransferase; APTT, activate partial thromboplastin time; AST, aspartate aminotransferase; CRP, C reactive protein; GGT, gamma-glutamyl transferase; INR, international normalised ratio; PT, prothrombin time.

Exploratory laparotomy yielded slightly turbid peritoneal fluid, which revealed no microorganisms, otherwise healthy small and large bowel, liver, stomach and gall bladder. Nasogastric aspirate culture yielded moderate growth of *Pichia kudriavzevii*, which was thought to be not pathogenic. Stool examination was negative for enteropathogenic bacteria and enteroviruses. Three blood cultures were negative.

Concern for *B. cereus* poisoning prompted a search for left-over rice, which was located and thought to be consumed from a container that only the patient had eaten from, and not her children. This was sent to the laboratory for analysis. This sample was inoculated directly onto blood agar and underwent ethanol shock treatment with absolute ethanol for 1 hour to select out toxin producing organisms prior to plating onto blood agar. The plates were then incubated at 37°C in $O_2$ overnight. Pure growth of beta-haemolytic colonies 6 mm in diameter were present after 14 hours of incubation on both plates and initial identification was performed using Matrix-assisted laser desorption/ionisation time of flight mass spectrometry (Bruker) with a score of 2.43 for *B. cereus*. No other samples directly from the patient or the patient's family isolated *B. cereus* despite them having the same diet.

The isolate underwent WGS to confirm the identification and the presence of genes conferring emetic toxin production. Genomic DNA was extracted using a QIAGEN DNeasy Blood and Tissue Mini Kit (QIAGEN) from the pure culture of *B. cereus*. Sequencing libraries were prepared using Nextera XT DNA Library Prep Kit (Illumina) and sequenced on a NextSeq 500. Reads were quality trimmed and filtered, then processed through the Nullarbor V.2.0 pipeline,[12] for assembly (skesa V.2.3.0) and multilocus sequence typing (V.2.6). The assembled genome was annotated using Prokka.[13] *Bacillus* toxin genes were identified directly from quality-trimmed reads using SRST2 V.0.2.0,[14] with a version of the VFDB *Bacillus* database

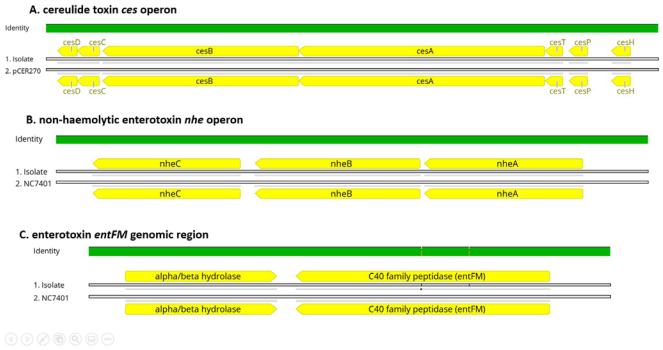


**Figure 1** Alignment of *cesHPTABCD*, *nheABC* and *entFM* genes. The isolate is 100% identical (green bar) to the reference sequences for the *ces* and *nhe* operons. There are two single nucleotide polymorphisms relative to the reference in the *entFM* gene (one synonymous and one non-synonymous).

in which the *ces* genes from *B. cereus* strain NC7401 were added.[15] The presence of additional toxin genes of interest, *cytK*, *hblA* and *entFM* were assayed by blasting the assembly. Identified toxin genes were manually examined using Geneious Prime.[16]

WGS of the isolate assigned it to sequence type (ST) 26, which is the typical sequence type for international emetic *B. cereus* isolates.[17] The isolate possessed complete operons for synthesis of the cereulide emetic toxin (*cesHPTABCD*) and non-haemolytic enterotoxin (*nheABC*) (figure 1). It was also found to possess the enterotoxin *entFM* gene, but did not carry the haemolysin BL (*hblA*) or cytotoxin K (*cytK*) genes more commonly found in *B. cereus* isolates associated with a diarrhoeal form of food poisoning.[18]

## TREATMENT

Initial treatment with intravenous (IV) piperacillin–tazobactam, metronidazole and azithromycin, which was changed to IV vancomycin continuous infusion 2 g/24 hours, meropenem 2 g/8 hours and metronidazole 500 mg/12 hours. Supportive measures were employed, including endotracheal intubation, significant vasopressor support, continuous venovenous haemodiafiltration (CVVHDF), plasmapheresis (day 2), cryoprecipitate, fresh frozen plasma, vitamin K and N-acetylcysteine (NAC) infusion. Charcoal was not given.

Despite maximal supportive measures, significant vasopressor requirements, metabolic acidosis and anuria persisted. Coagulopathy was partially corrected, with mild improvement of rhabdomyolysis. Atrial fibrillation with rapid ventricular rate developed, requiring amiodarone.

## OUTCOME AND FOLLOW-UP

Serial ECG revealed ST elevation in inferior leads with reciprocal changes, with troponin 69 020 ng/L (normal high ≤50 ng/L). Decision was made not for angiography, thrombolysis, antiplatelets or anticoagulants due to ongoing coagulopathy and multi-organ failure (see table 1). Diffuse broad complex ventricular tachycardia developed, despite IV magnesium replacement. Gradually declining mean arterial pressure was noted from 53 to 39, despite uptitration of norepinephrine infusion, resulting in asystolic arrest and death. A surgical autopsy was not performed at the family's request.

## DISCUSSION

This case details an extreme presentation of food poisoning due to pre-formed emetic toxin producing bacteria *B. cereus,*[1] with severe manifestations of sepsis, including multi-organ (hepatic, renal, respiratory and neurological) failure, shock, metabolic acidosis, rhabdomyolysis and coagulopathy, ultimately resulting in a fatal ischaemic myocardial insult. There are only nine cases of similar catastrophic disease progression described in immunocompetent individuals,[5 7–11 19–22] with only four above 16 years of age.[5 8 10 22] Culprit foods contaminated by *B. cereus* included fried rice, pasta, noodles and salami.

Given consumption of one-week-old fried rice and mushrooms, both bacterial and mushroom toxins were suspected. Food poisoning and hepatotoxicity due to consumption of mushrooms are caused by amatoxin in *Galerina*, *Lepiota* and especially *Amanita* species. Gastrointestinal effects occur typically 6–12 hours after ingestion, followed by a quiescent period of 12 hours with symptomatic improvement, then progressive hepatotoxicity and death within a week, or require liver transplantation.[23]

The detection of cereulide has been confirmed in six case reports, with variable concordance between food and patient samples. Only Shiota *et al* describes a family of 3 cases with serial measurements of cereulide levels, with falling levels correlating with clinical improvement, and undetectable levels with milder symptoms. This case demonstrates the novel use of WGS, to confirm the toxigenic potential of *B. cereus*. Given *B. cereus* is ubiquitous in the environment, its culture from a clinical sample or food leftovers may be otherwise difficult to interpret. Furthermore, cereulide is responsible for mitochondrial beta-oxidative dysfunction, which can evoke organ failure, particularly microvesicular steatosis of the liver. This may be due to a genetic or acquired impairment, which perhaps may have been present in this patient and account for a differential expression of toxicity among family members.[24]

There have been variable time intervals from food preparation to ingestion (1–5 days), time left at room temperature (hours to 5 days) and reheating, following leaving at room temperature or refrigeration. Cereulide is known to be a heat-stable toxin, resistant to proteolysis. By spore formation, *B. cereus* may survive pasteurisation and reheating. It tolerates extreme pH values between 2 and 11 and is stable to pepsin and trypsin.[25] Naranjo *et al* draw reference to the importance of food storage to prevent *B. cereus* growth; however, note that toxin production is not strictly correlated with bacterial counts but rather temperature, with higher amounts seen at ambient (23°C) than warmer (30°C) temperatures. Pickering *et al* suggest to prevent foodborne outbreaks, food should be kept at temperatures higher than 60°C or rapidly cooled to less than 10°C after cooking. This coincides with other reports that some *B. cereus* strains are known to have the highest emetic toxin production between 12°C and 15°C,[26] and may grow despite refrigeration (at 14°C).[9] This has important bearings on food preparation particularly as this case occurred amid the COVID-19 pandemic and during a time of strict home isolation. Starch-based foods should not be left to cool prior to refrigeration.

Several treatment options were offered with known benefit. *Bacillus* species are known to produce beta-lactamases and are usually resistant to penicillin and cephalosporins. Vancomycin, aminoglycosides, carbapenem, clindamycin and ciprofloxacin are usually active in vitro,[1 2] and have been effective in a clinical setting.[21] In view of this, our patient was promptly switched from piperacillin–tazobactam to meropenem and vancomycin.

In view of anuric acute kidney injury, CVVHDF was promptly commenced, however had a dual role in potential toxin clearance. Continuous Renal Replacement Therapy (CRRT) has high efficacy in removing low-molecular-weight substances, and as such should aid in clearing cereulide, a small molecule.[27] Symptomatic and biochemical improvement has been seen exclusively with CRRT.[7 9]

The binding capacity of cereulide to plasma protein and distribution in human tissue is unknown. If a large amount of protein was bound to cereulide, plasmapheresis is preferred as it removes both large and small molecular weight substances.[5] The use of plasmapheresis is controversial in massive disseminated intravascular coagulation (DIC) or bacterial septicaemia,[28] with further studies ongoing to assess its efficacy.[29] It may prevent kidney exposure to massive quantities of haemolysed red cells, reduce release of red cell stroma and prevent intravascular haemolysis. It may move mediators of Systemic inflammatory response syndrome response that follow endotoxin exposure, for example, tumor necrosis factor. Reduced intravascular coagulation abnormalities have been seen in a rabbit model of endotoxin-induced DIC. Shiota *et al* describe that plasmapheresis should be used before CRRT to remove all toxins with and without plasma proteins, then haemodialysis to remove both the remaining and redistributed cereulide after plasmapheresis. This was used in their case of a 2-year-old girl; however, they report symptomatic improvement and decreased cereulide amount prior to plasmapheresis administration, suggesting the patient was already improving. Tschiedel *et al* describe a case of a 1-year-old boy that had hyperammonia and hyperkalaemia that was refractory to simultaneous CVVHDF and plasmapheresis, however, stabilised following hepatectomy. In our case, CVVHDF began prior to *B. cereus* infection being recognised, and plasmapharesis started 12 hours following the infection. Serial CRP measurements were normal (<4 mg/L) throughout admission. It, therefore, still remains unknown: the optimal timing for initiation and benefit of plasmapheresis.

Several treatments were trialled without proven benefit. NAC was used due to the hepatoprotective effect of acetylcysteine, based on its ability to replenish glutathione stores, and potential improvements on mitochondrial energy metabolism.[30] A meta-analysis of four studies was explored by Hu *et al*, looking at the efficacy and safety of NAC in non-acetaminophen acute liver failure was inconclusive. NAC showed conflicting results with prolonged survival in transplant-free patients, and survival after transplantation, but no effect on overall survival.

## Learning points

- Fulminant hepatitis with multi-organ failure is a rare and potentially fatal outcome of fried-rice-associated food poisoning due to the pre-formed exotoxin cereulide, produced by *Bacillus cereus*.
- There is value in targeted history-taking to guide clinical investigations and reach an ultimate diagnosis.
- After cooking, leftover starch-based foods, including rice, should be promptly cooled to temperatures <10°C, and not left to cool prior, to prevent production of Bacillus cereus and toxin production.
- Whole genome sequencing is valuable in confirming the toxigenic potential of implicated *B. cereus* isolates, which are ubiquitous in nature.

Liver transplantation in *B. cereus* fulminant hepatic failure has to date been described in one case report in a previously healthy 13-month-old child. Thirty hours into admission, the patient continued to have refractory hyperkalaemia (despite CVVHDF and plasmapheresis with 2 L/hour turnover) and hyperammonia. He underwent hepatectomy, and stabilised within 3 hour with normal potassium and blood pressure while on CVVHDF and noradrenaline <1 ug/kg/min. He then received partial liver transplantation of segments II and III from his father, with normalisation of liver function within 1 hour, renal function within 8 weeks and no evidence of brain damage.[20]

The use of activated charcoal has been explored by Mahler *et al*, describing a case of father and son with *B. cereus* gastroenteritis. The father tolerated the charcoal, and survived, while the son did not, and died within 2 days. Post-mortem examination identified high concentration of emetic toxin in the son's bile, suggesting biliary excretion and enterohepatic circulation.[8] This case suggests charcoal could have merits for primary detoxification in *B. cereus* poisoning, and accelerate elimination of absorbed toxin. If used, administration should begin as soon as possible, at 50–100 g, and can be given repeatedly.[31] In our case, a delay of >24 hour from symptom onset to recognising *B. cereus* infection limited the perceived benefit, and charcoal was not administered.

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