

Fulminant *Bacillus Cereus* Endogenous Panophthalmitis and Flood Water: A Case Report and Literature Review

Abdullah MA^{ab}, Abdullah NA^a, Kamarudin Z^a, Liza-Sharmini AT^b

^aDepartment of Ophthalmology, Hospital Selayang, Selangor, Malaysia

^bDepartment of Ophthalmology and Visual Science, School of Medical Sciences, Universiti Sains Malaysia, Kelantan, Malaysia

ABSTRACT

Bacillus cereus is a spore-forming Gram-positive bacillus found in soil and water that can cause severe ocular infections. This is a case of a 53-year-old woman with uncontrolled diabetes who developed sudden, painful vision loss in her left eye after flood exposure. Examination revealed no light perception, ophthalmoplegia, and elevated intraocular pressure (IOP) of 52 mmHg. Within eight hours, her condition worsened with severe proptosis, chemosis, and IOP rising to 87 mmHg, requiring emergency canthotomy and cantholysis. Blood cultures confirmed *B. cereus*, and blood tests showed leucocytosis and elevated C-reactive protein. Imaging revealed necrotizing infection with orbital gas. Despite intravenous antibiotics, the eye ruptured and evisceration was performed. She completed two weeks of antibiotics and recovered without systemic or intracranial spread. This case underscores the fulminant progression of *B. cereus* pan-ophthalmitis, the need for early intervention, and the importance of PPE during post-flood clean-up to prevent serious infections, particularly in vulnerable individuals.

Keywords

bacillus cereus panophthalmitis,
bacillus cereus infection, *bacillus cereus* and
floodwater, fulminant panophthalmitis

Corresponding Author

Prof. Dr. Liza Sharmini Ahmad Tajudin
Department of Ophthalmology and Visual
Sciences, School of Medical Sciences,
Universiti Sains Malaysia, Health Campus,
Kelantan, Malaysia
Email: liza@usm.my

Received: 9th June 2025; Accepted: 11th
August 2026

Doi:
<https://doi.org/10.31436/10.31436/imjm.v25i04/2992>

INTRODUCTION

A 53-year-old woman with suboptimal controlled type 2 diabetes mellitus (HbA1c: 7.1%) was referred to the ophthalmology team for sudden-onset painful vision loss in the left eye (OS) on the seventh day of hospitalisation. She was initially admitted for lethargy, bilateral upper limb numbness, and tremors, which were diagnosed as electrolyte imbalances secondary to poor oral intake. At the time of admission, she was afebrile and hemodynamically stable, although hyperglycaemic with a capillary blood glucose of 16 mmol/L. Initial investigations revealed a normal full blood count, but confirmed significant biochemical derangements: fasting blood sugar of 15 mmol/L, hyponatremia (128 mmol/L), hypokalaemia (2.8 mmol/L), and hypomagnesemia (0.45 mmol/L). Chest radiography

was deferred as the patient lacked respiratory symptoms. Upon further history, the patient revealed she was a recent flood victim and had waded through floodwaters to reach an evacuation centre. Physical examination noted a small, healing ulcer on her leg, though the patient denied any history of chronic or non-healing ulcers.

Ocular examination of the left eye: her vision was no perception of light (NPL) with a positive relative afferent pupillary defect. There was eyelid swelling with complete ophthalmoplegia but no obvious proptosis initially. Her conjunctiva was injected with a clear cornea and no anterior chamber activity (Figure 1). Fundoscopy examination showed the presence of vitreous haemorrhage (VH) obscuring the optic disc with

superior preretinal haemorrhages. The right eye was sound. The intraocular pressure (IOP) was 52mmHg OS and 10mmHg right eye (OD). B-scan ultrasound showed scleral thickening (2.35mm), presence of VH, and intact retina with no sign of loculation. Based on the initial clinical findings, she was treated as orbital cellulitis with scleritis and central retinal vein occlusion (CRVO) secondary to ocular hypertension.

She was treated with intravenous (IV) ceftriaxone 2g STAT and 1g twice a day and IV metronidazole 500mg thrice a day with topical pressure-lowering drugs including prostaglandin analogue, acetazolamide, alpha agonist and beta blocker. She was also given oral acetazolamide 500mg stat followed by 250mg QID. Despite this treatment, she developed left orbital compartment syndrome within eight hours of initiation of treatment. She developed sudden worsening periorbital swelling, conjunctival chemosis, and non-axial proptosis with corneal exposure (Figure 1). There was also presence of total hyphema, and the IOP shot up to 87 mmHg. Emergency canthotomy and cantholysis were done to release the pressure; her IOP remained high at 69 mmHg. Chloramphenicol ointment QID and a moist chamber were applied to protect the exposed cornea.

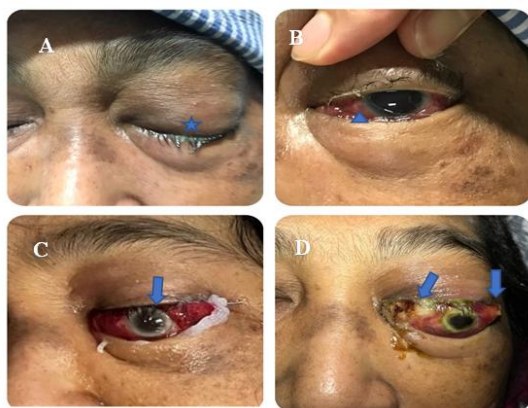


Figure 1: (A, B) shows swollen left upper and lower lids (star) with thick mucus discharges and injected conjunctiva with shallow chemosis with clear cornea (triangle). (C) shows proptosis 8 hours later with severe lagophthalmos exposing whole cornea after lateral canthotomy and cantholysis procedure (arrow). (D) Shows deformed left eye globe after sclera perforation at temporal and medial region (arrow).

Laboratory investigations and imaging performed on day seven following the onset of ocular symptoms revealed leucocytosis and an elevated C-reactive protein. Blood cultures confirmed the presence of *Bacillus cereus*, which was sensitive to ciprofloxacin, meropenem, penicillin, and vancomycin, but resistant to metronidazole. Chest radiography subsequently revealed bilateral lower lobe consolidation (Figure 2). A computed tomography (CT) scan of the orbit and brain demonstrated left eye proptosis with orbital cellulitis, the presence of an intraocular gas bubble, and vitreous haemorrhage, with no intracranial or cavernous sinus involvement (Figure 2). Based on the blood culture result (Figure 3) and the rapid progression of the disease, the patient was diagnosed with *B. cereus* panophthalmitis complicated by orbital compartment syndrome. The infectious disease team initiated broad-spectrum antimicrobial therapy with intravenous vancomycin and ceftriaxone. However, due to an allergic reaction to vancomycin, the regimen was adjusted to oral linezolid 600 mg twice daily.

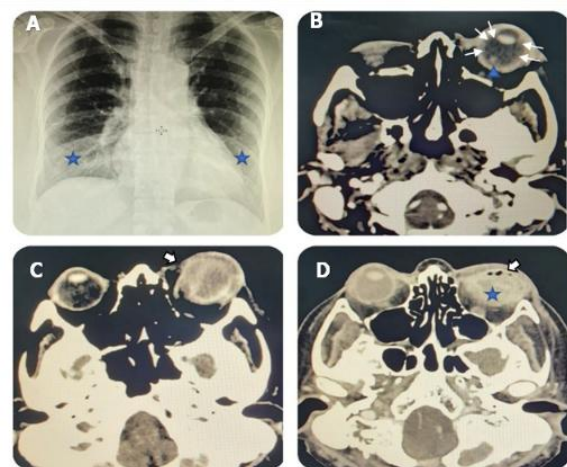


Figure 2: (A) Shows inferior lower lobes consolidation (star), (B) CT scan Brain and Orbit showed left eye proptosis. Presence of intraocular gas bubble (arrow), focal of hyperdensity at the vitreous region, suggestive of hemorrhage (triangle). No definite collection or subperiosteal collection seen. (C) Non contrasted CT scan brain and orbit on day seventh after onset of the infection showed distorted left eye globe with irregular wall and presence of crescent-shaped collection (arrow) at medial posterosuperior which communicating with discontinuity of lateral wall, suggestive of globe ruptured. (D) Non contrasted CT scan brain and orbit post evisceration showed small rim-enhancing collection (arrow) at preseptal region and associated with multiple air pockets within.

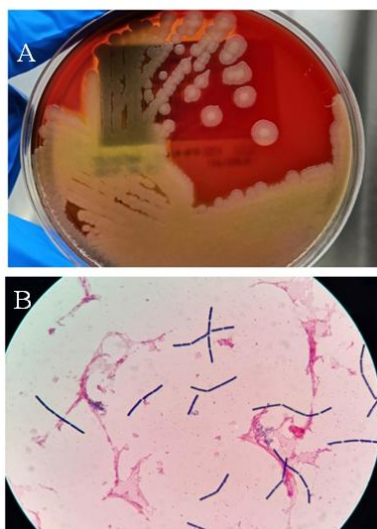


Figure 3: (A) shows large rounded dry looking grey-white, weakly haemolytic bacterial colony on blood agar. (B) microscopically *B. cereus* appears as gram-positive, rod-shaped bacilli.

Clinically, her ocular condition showed slight improvement, but her leucocyte level and inflammatory markers improved drastically. The patient remained afebrile, and repeated blood cultures showed no growth on day-6 post initiation of treatment. However, despite responding well to systemic antibiotics, her left eye perforated on day-7 (Figure 1D and Figure 2C, D). Emergency evisceration was done. Postoperatively, serial CT Scan brain and orbit showed resolution of the preseptal pus collection and air pocket (Figure 2D). She was then given a two-week course of ceftriaxone and linezolid, achieving full systemic recovery without contralateral eye involvement or septic complications. Her pneumonia was clinically resolved. She is currently fitted with a left ocular prosthesis and remains under follow-up for the right eye.

DISCUSSION

Bacillus cereus is a Gram-positive, spore-forming, rod-shaped aerobic bacterium that is commonly found in soil and water.¹ Its ability to form spores allows it to survive in harsh environmental conditions, including contaminated floodwater. The bacterium produces multiple virulence factors such as phospholipases, hemolysins, and enterotoxins that damage cell membranes, induce tissue necrosis, and cause intense

inflammation, making *B. cereus* infections particularly aggressive and destructive.² Its ability to form biofilms further enhances survival and antibiotic resistance.

In ocular infections, *B. cereus* can rapidly invade intraocular structures. Enzymes like collagenases and proteases damage the cornea, sclera, and vitreous, often leading to fulminant panophthalmitis.³ In our case, a 53-year-old diabetic patient developed sudden painful vision loss and orbital compartment syndrome following flood exposure. Her immunocompromised status and exposure to contaminated floodwater made her highly susceptible.⁴ The diagnosis of *B. cereus* panophthalmitis relies on clinical suspicion and microbiological confirmation. Hallmarks include rapid progression, typically within hours⁵, and profound ocular inflammation. In our case, the patient presented with no light perception, high IOP, and chemosis. Within hours, her IOP rose to 87 mmHg, necessitating emergency canthotomy and cantholysis. Imaging revealed the presence of intraocular gas bubbles on CT scan, an important diagnostic clue, as gas formation within the globe is highly suggestive of infection with gas-producing organisms such as *B. cereus*.⁶ This finding reflects rapid intraocular tissue necrosis and anaerobic metabolic activity. Culture of blood samples confirmed *B. cereus*. Microscopic examination revealed Gram-positive rods. While definitive diagnosis is made through culture, PCR and next-generation sequencing are also valuable for rapid identification. Clinically, *B. cereus* panophthalmitis often presents with severe ocular pain, chemosis, proptosis, hypopyon, vitreous haze, and loss of vision. The infection may extend to the orbit, causing cellulitis or abscess formation.⁷ In some cases, systemic spread to the central nervous system can occur. Our patient showed bilateral lower lobe consolidation, possibly from inhalation of spores during post-flood clean-up. Although she remained afebrile, she developed sepsis-like changes with elevated white cell count and C-reactive protein. Foot examination revealed a healing diabetic foot ulcer, suggestive of suboptimal glycaemic control rather than an active infectious source, as there was no evidence of purulent discharge, foul odour, or

necrotic tissue indicative of ongoing infection. Nosocomial transmission was excluded due to the absence of an epidemiological link to concurrent cases within the ward during the same period of admission, supported by a negative ward screening conducted by the hospital infection control team. The patient's significant history of exposure to contaminated floodwater prior to the manifestation of systemic and ocular symptoms instead suggests a community-acquired environmental aetiology.

It is worth noting that while most *B. cereus* ocular infections present within 12 to 24 hours, delayed onset of up to 48 hours has been documented, and in hematogenous cases the eye may appear unremarkable during the early bacteraemia phase, making unexplained systemic signs such as electrolyte disturbances, particularly hyponatremia, an important early warning that should prompt urgent blood cultures and close ophthalmic surveillance even before overt ocular symptoms emerge.^{5,8,9} The uncontrolled diabetic status of our patient likely contributed to this delayed yet fulminant presentation, as chronic hyperglycaemia is well established to impair neutrophil chemotaxis, phagocytosis, and complement activation, thereby blunting the early host response and allowing subclinical bacteraemic seeding to progress silently until intraocular invasion had already occurred, a pattern that has been

previously documented in fatal *B. cereus* bacteraemia in a diabetic patient.¹⁻⁴

Panophthalmitis is an ocular emergency. Although systemic antibiotics are the mainstay of treatment, *B. cereus* produces beta-lactamases, making it resistant to many beta-lactam antibiotics. In our patient, empirical intravenous ceftriaxone and metronidazole were started, but adjusted once *B. cereus* was identified in the blood culture. Vancomycin, typically used for Gram-positive coverage, was contraindicated due to allergy, so oral linezolid was initiated alongside continued ceftriaxone. The infection rapidly progressed despite timely antibiotic escalation, leading to globe perforation. Emergency evisceration was performed to control the infection and prevent systemic spread. This outcome is frequently reported in *B. cereus* ocular infections due to their aggressive, necrotizing course and poor response to medical therapy alone.¹⁰ Regarding the pneumonia, as it served as a potential source of ocular infection via septicaemia, the antibiotic regimen was aligned with that used for panophthalmitis as described above. The patient was treated with oral linezolid and ceftriaxone, demonstrating a favourable clinical response, evidenced by resolution of lung consolidation on imaging, clearance of bacteraemia on repeat blood cultures, and stable hemodynamic parameters without progression to respiratory distress requiring intensive care support.

Table I: Literature review on cases of *B. Cereus* Endophthalmitis and Panophthalmitis from 1974 until 2021s

Author(s), Year	Gender, Age	Eye(s)	Route of Infection	IOFB ¥	Culture Sample	Organism Grew	Antibiotic Treatment	Eviscerated	Presenting Vision	Final Vision
Current case report	Female, 53	Left	Floodwater exposure and endogenous	No	Blood culture	Bacillus cereus	Linezolid, Ceftriaxone	Yes	NPL	NPL
Shamsuddin et al. 1974	Female, 25	Right	Intravenous drug use	No	Intraocular content	Bacillus cereus	Vancomycin, Chloramphenicol	Not specified	Not specified	Not specified
Hatem et al. 1979	Male, 32	Left	Intravenous drug use	No	Vitreous humor	Bacillus cereus	Clindamycin, gentamicin	Yes	Not specified	NPL
Bouza et al. 1979	Male, 43	Right	Intravenous vitamin injection	No	Vitreous humor	Bacillus cereus	Not specified	Yes	Not specified	NPL
Young et al. 1980	Male, 23	Left	Intravenous drug use	No	Vitreous humor and blood	Bacillus cereus	Cefazolin -> Clindamycin, Gentamicin	Yes	Not specified	NPL
Ho P et al. 1982	Male, 30	Left	Soil-contaminated injury	Yes	Vitreous humor	Bacillus cereus	Clindamycin, gentamicin	Yes	PL €	NPL
Cowan et al. 1987	Female, 28	Right	Intravenous drug use	No	Vitreous humor	Bacillus cereus	Vancomycin, gentamicin	Yes	Not specified	NPL
Wai-Man Chan et al. 2015	Male, 72	Left	Post-cataract surgery	No	Vitreous humor	Bacillus cereus	Vancomycin, Cefuroxime	Yes	PL €	NPL
Donovan S. et al. 2021	Male, 39	left	Intravenous drug use	No	Blood culture	Bacillus cereus	Not specified	Yes	NPL	NPL

¥ Intraocular Foreign Body
€ Perception of light

Although early diagnosis and prompt antibiotic therapy can be sight- and life-saving in *B. cereus* panophthalmitis, preventive measures remain crucial, as *B. cereus* infections can lead to severe morbidity and potentially fatal outcomes. In this case, the lady likely acquired the infection via hematogenous spread from pneumonia after contact with contaminated floodwater. Spores can aerosolize during clean-up and be inhaled. Given that flooding is common in Malaysia during the monsoon season, this case highlights the need for strengthened public health guidance emphasizing the use of personal protective equipment (PPE), including gloves, N95 masks, goggles, and boots during flood recovery efforts. In addition, raising awareness of proper hand hygiene, avoiding eye contact with contaminated water, and ensuring adequate ventilation in enclosed spaces is equally important to reduce the risk of infection.

CONCLUSION

B. cereus panophthalmitis is a rapidly progressing ocular emergency. Prompt recognition, aggressive antibiotic therapy, and early surgical intervention are key to preventing systemic spread and mortality. Public health strategies focusing on prevention and awareness are crucial in post-flood scenarios, particularly for high-risk groups.

REFERENCES

1. Bottone EJ. *Bacillus cereus*, a Volatile Human Pathogen. Clin Microbiol Rev. 2010 Apr;23(2):382–98. doi:10.1128/CMR.00073-09
2. Callegan MC, Cochran DC, Kane ST, Gilmore MS, Gominet M, Lereclus D. Contribution of membrane-damaging toxins to *Bacillus* endophthalmitis pathogenesis. Infect Immun. 2002 Oct;70(10):5381–9. doi:10.1128/IAI.70.10.5381-5389.2002. PubMed PMID: 12228262.
3. Mursalin MH, Livingston ET, Callegan MC. The cereus matter of *Bacillus* endophthalmitis. Exp Eye Res. 2020 Apr;193:107959. doi:10.1016/j.exer.2020.107959
4. Orrett FA. Fatal *Bacillus cereus* bacteremia in a patient with diabetes. J Natl Med Assoc. 2000 Apr;92(4):206–8. PubMed PMID: 10976178.
5. Ho PC, O'Day DM, Head WS. Fulminating panophthalmitis due to exogenous infection with *Bacillus cereus*: report of 4 cases. Br J Ophthalmol. 1982 Mar;66(3):205–8. doi:10.1136/bjo.66.3.205. PubMed PMID: 6802173.
6. al-Hemidan A, Byrne-Rhodes KA, Tabbara KF. *Bacillus cereus* panophthalmitis associated with intraocular gas bubble. Br J Ophthalmol. 1989 Jan;73(1):25–8. doi:10.1136/bjo.73.1.25. PubMed PMID: 2493262.
7. Kumar N, Garg N, Kumar N, Van Wagoner N. *Bacillus cereus* panophthalmitis associated with injection drug use. International Journal of Infectious Diseases. 2014 Sep;26:165–6. doi:10.1016/j.ijid.2014.01.019
8. Davey RT, Tauber WB. Posttraumatic Endophthalmitis: The Emerging Role of *Bacillus cereus* Infection. Clinical Infectious Diseases. 1987 Jan 1;9(1):110–23. doi:10.1093/clinids/9.1.110
9. Liamis G, Milionis HJ, Elisaf M. Hyponatremia in patients with infectious diseases. Journal of Infection. 2011 Nov;63(5):327–35. doi:10.1016/j.jinf.2011.07.013
10. Shamsuddin D, Tuazon CU, Levy C, Curtin J. *Bacillus cereus* Panophthalmitis: Source of the Organism. Clinical Infectious Diseases. 1982 Jan 1;4(1):97–103. doi:10.1093/clinids/4.1.97