JOURNAL OF CLINICAL MICROBIOLOGY, Feb. 1997, p. 504–507 Vol. 35, No. 2
0095-1137/97/$04.00+0

Fulminating Bacteremia and Pneumonia Due to *Bacillus cereus*

J. MICHAEL MILLER,[1]* JAMES G. HAIR,[2] MARY HEBERT,[2] LYNETTE HEBERT,[3] AND FLOYD J. ROBERTS, JR.[4]

Hospital Infections Program, National Center for Infectious Diseases, Centers for Disease Control and Prevention, Atlanta, Georgia,[1] and Rapides Regional Medical Center, Alexandria,[2] and Louisiana Reference Laboratory,[3] and Baton Rouge General Medical Center,[4] Baton Rouge, Louisiana

Received 23 September 1996/Returned for modification 16 October 1996/Accepted 30 October 1996

We present two cases of rapidly progressing, fatal pneumonia caused by *Bacillus cereus*. These cases are interesting in that *B. cereus*, even from blood or sputum specimens, may often be considered a contaminant and receive inadequate attention. Also of interest was the fact that the two patients resided in the same area of the state, were welders by trade, and became ill within a few days of each other, yet there was no epidemiologic link between them.

The finding of a *Bacillus* sp. is often considered not clinically relevant because these organisms are ubiquitous in the environment and can easily contaminate culture material as well as improperly handled specimen collection devices. *Bacillus anthracis* is a frank pathogen, but it is uncommon in most clinical laboratories. While *Bacillus cereus* is occasionally associated with food-borne illness, its presence in cultures is often considered a contaminant, and the organism is not further characterized beyond a descriptive identification and may not be reported at all (11). We present two unusual cases in which *B. cereus* is the apparent cause of fatal respiratory disease in otherwise healthy adults.

Patient 1. The patient was a 46-year-old male welder from central Louisiana who had been healthy until 5 days prior to his 27 December hospital admission. During this 5-day period, he had experienced a cough, congestion, and chills and fever, all of which began to subside 24 h prior to admission. During the 24 h before his presentation in the emergency room, he experienced episodes of hemoptysis that became particularly intense 6 to 8 h prior to seeing his family physician early on the morning of 27 December. He was referred directly to the emergency room of the local hospital.

By 10:00 a.m., he was examined in the emergency room and found to have an oral temperature of 98.1°F, a pulse of 128 bpm, a respiratory rate of 26/min, and a blood pressure of 170/98 mm Hg. He was immunocompetent, with no family history of tuberculosis; he reportedly was a nonsmoker and only rarely consumed alcohol; and he was well developed, well nourished, and in no acute distress. Upon admission, he was alert, calm, and responsive, with the ability to provide an accurate medical history. In his profession as a welder he was exposed to significant amounts of dust and fumes, but had no known exposure to asbestos. A physical examination revealed normal breath sounds in all quadrants of the lungs, although the sounds decreased in the lower quadrants, with only occasional coarse rales and rhonchi. Heart sounds were normal, although the rate was rapid. The skin exhibited no cyanosis, pallor, or rashes but was positive for diaphoresis. Ear, throat, and sinus examinations were unremarkable.

A chest X ray was performed and was markedly abnormal, revealing a confluent alveolar infiltrate in the right lung, with only a small amount of aeration noted in the apex (Fig. 1). The left lung had a confluent density in the midline suggestive of a mass. Remarkable laboratory findings on admission included the following: platelet count, 55,000/mm^3; total bilirubin, 2.6 mg/dl; lactate dehydrogenase, 1,322 U/liter; creatinine, 3.6 mg/dl; blood urea nitrogen, 43 mg/dl; and leukocyte count, 26,900 cells/mm^3 with a left shift. A sputum specimen and one set of aerobic and anaerobic blood specimens were collected for analysis. The Gram stain of the sputum revealed <10 squamous epithelial cells and >25 leukocytes per low-power field but no predominating organism upon further examination.

TABLE 1. Biochemical reactions[a] of two strains of *B. cereus* isolated from two cases of fatal respiratory infection

Test material or process[b]	Reaction
Catalase	Positive
Oxidase	Positive
MacConkey agar	No growth
SS agar	No growth
Urea	Partial positive
Simmons citrate	Positive
Nitrate	Positive, no gas
Nitrite (0.1%)	Negative
Nitrite (0.01%)	Positive, no gas
Indole	Negative
MR	Positive
VP	Positive
TSI slant	K/A
Gelatin	Positive
Esculin hydrolysis	Positive
Litmus milk	Clot, peptonization
Pigment, insoluble	Green
Fat stain	Positive, safranin stain
Casein hydrolysis	Positive
Starch hydrolysis	Negative
Lecithinase	Positive
Penicillin disk	No zone, 6 mm
D-Glucose	Acid
D-Xylose	Negative
D-Mannitol	Negative
Lactose	Negative
Sucrose	Acid
Maltose	Acid

[a] These results, identical for both isolates, were generated by the Special Bacteriology Laboratory of the Centers for Disease Control and Prevention.
[b] SS, salmonella-shigella; MR, methyl red; VP, Voges-Proskauer reaction; K/A, alkaline/acid.

* Corresponding author.

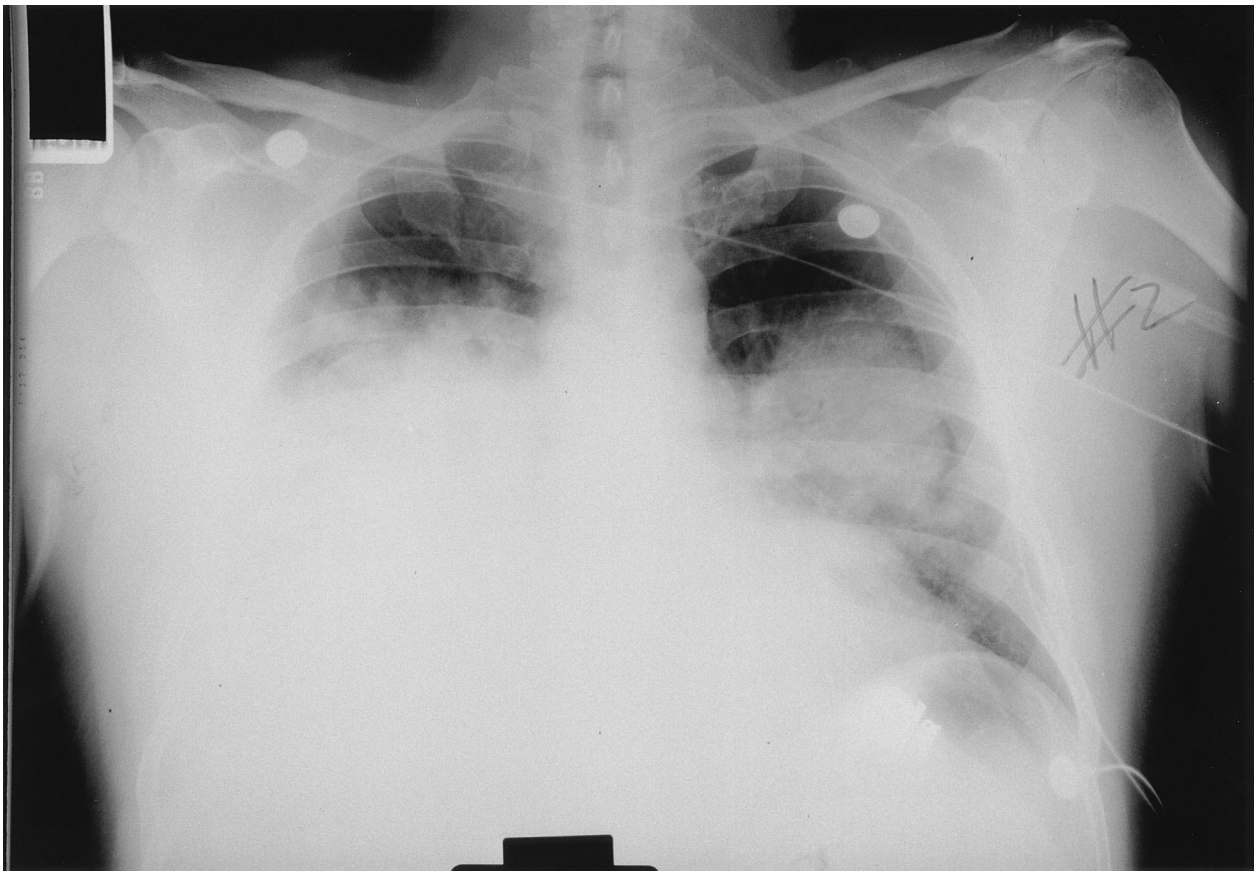

FIG. 1. Chest X ray of 46-year-old male welder showing confluent infiltrates. *B. cereus* was isolated from both sputum and blood from this patient.

Preliminary diagnoses included left lung tumor, hemoptysis, pneumonia, and renal insufficiency.

At 1:30 p.m., the patient was admitted to the hospital and placed on ciprofloxacin and cefotaxime as therapy for a potential bacterial pneumonia. Oxygen therapy was started, and the patient remained alert with stable vital signs. At 4:00 p.m., the patient vomited coffee ground-colored emesis and continued to produce a bloody, frothy sputum. Shortly thereafter, the patient was taken to the radiology suite for a computed tomography scan where he had a cardiorespiratory arrest. Resuscitation attempts were unsuccessful, and the patient was pronounced dead at 5:30 p.m.

Both sputum and blood cultures grew a large, beta-hemolytic gram-positive rod with central spores, identified biochemically as *B. cereus* (Table 1). The isolate was initially identified by the API 50CH card (bioMerieux Vitek, St. Louis, Mo.) and later confirmed by a reference laboratory. Pathology sections of lung tissue stained by tissue Gram stain and Gomori methenamine silver revealed that the alveoli of the lung contained numerous gram-positive bacilli (Fig. 2), indicating an overwhelming infection. There was a mild-to-moderate inflammatory response, much less than would be expected from the number of bacteria present in the tissue.

Patient 2. The patient was a 41-year-old male welder from south Louisiana who had been healthy until 3 days prior to admission. During this 3-day period he had experienced a "bloody" cough, chills, and pleuritic pain in his right chest. Upon presentation to the hospital on 15 January, his temperature was 97.2°F, his pulse was 135 bpm, his respiratory rate was 18/min, and his blood pressure was 118/82 mm Hg. Although alert and responsive, the patient appeared ill, with rhonchi noted more prominently on the right posterior hemithorax than on the left. He had no medical history of illness and reportedly did not smoke, use drugs, or drink alcohol. All other physical examination parameters were normal.

A chest X ray revealed a right middle and upper lobe infiltrate that was globular in appearance. Laboratory findings on admission included the following: leukocyte count, 8,800 cells/mm^3 (that rose to 17,000 cells/mm^3 by day 3); hemoglobin, 18.2 gm/dl; hematocrit, 55.9%; and platelets, 141,000/mm^3. Abnormal chemistry results were a blood urea nitrogen level of 27 mg/dl, a creatinine level of 3.6 mg/dl, and a total bilirubin level of 4.3 mg/dl. At this time there was a question of his inability to mount an adequate leukocyte count in light of his clinical picture. The patient was admitted to the hospital, given oxygen supplementation, and started on ampicillin-sulbactam, erythromycin, and clindamycin. Sputum and blood specimens were obtained, and stains and a culture for *Mycobacterium tuberculosis* were requested. Microscopy results showed gram-positive rods in the sputum with >25 leukocytes/low-power field. An auramine-rhodamine stain for *M. tuberculosis* was negative as was a culture. Routine blood and sputum cultures grew gram-positive rods subsequently identified with conventional biochemicals as *B. cereus* (Table 1). Vancomycin was added to the antimicrobic regimen.

Over the next 3 days, the patient's condition deteriorated. Chest X rays showed a progression to infiltrates in both lung fields with effusions. On 18 January, the patient began to ex-

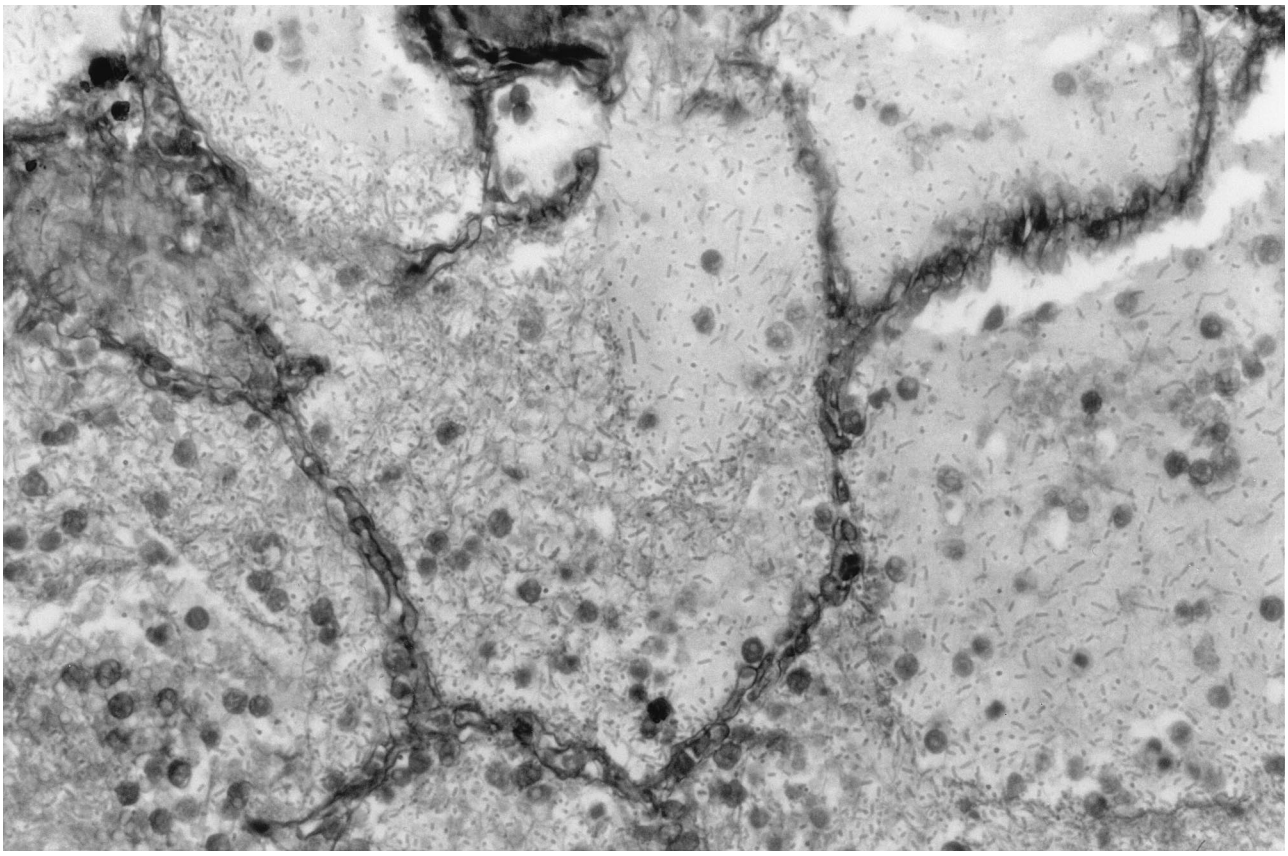

FIG. 2. Gomori methenamine silver stain of lung tissue showing alveoli containing numerous rods shown by culture to be *B. cereus*. Magnification, ×400.

perience sinus tachycardia and died 3 days after admission. Gomori methenamine silver and tissue Gram stained preparations of lung tissue revealed large numbers of gram-positive bacilli within the alveoli.

These two episodes are unusual in that two previously healthy middle-aged individuals, with no known immunocompromised status, succumbed to overwhelming sepsis and lung infiltrates associated with *B. cereus* and symptoms resembling those of *B. anthracis* respiratory disease. Also of interest is the fact that these two patients were both welders by trade, that they resided less than 3 h from each other, and that the deaths occurred about 2 weeks apart. As far as could be determined, there was no epidemiologic relationship between the two and no evidence in the literature that welders are significantly more at risk for *B. cereus* disease than are individuals in other occupations.

The identity of both isolates was confirmed by 7-day conventional fermentative biochemical reactions (Table 1). The antimicrobic test results generated by E-test (done for the isolate from patient 1 at a reference laboratory) and disk diffusion (done on the isolate from patient 2 at the local hospital) were identical. Although there are no National Committee for Clinical Laboratory Standards guidelines for interpreting and reporting the results of susceptibility tests on *Bacillus* sp., if the interpretive criteria for staphylococci are used as a guideline, both isolates appeared susceptible to amikacin, gentamicin, chloramphenicol, ciprofloxacin, clindamycin, erythromycin, tetracycline, and vancomycin. Both appeared resistant to ampicillin, cephalothin, ceftriaxone, trimethoprim-sulfamethoxazole, and penicillin.

An excellent review of *B. cereus* has recently been published (3) that describes the organism and its association with human disease. Although most commonly seen as a food-borne pathogen, it is associated with other human disease, especially in immunocompromised patients and in those with other underlying illnesses. Its isolation as an etiologic agent of pneumonia is rare and usually associated with significant risk factors (Table 2). It is even more unusual in patients who otherwise have no known risk factors for severe respiratory illness. The phys-

TABLE 2. Reported episodes[a] of adult pneumonia associated with *B. cereus*

Patient	Risk factor	Outcome	Reference
1	None	Died	10
2	Leukemia	Died	2
3	Leukemia	Died	6
4	Leukemia	Recovered	8
5	None	Recovered	7
6	Alcohol abuse	Recovered	1
7	Leukemia	Recovered	9
8	None	Recovered	5
9	Aplastic anemia	Died	4

[a] These case reports include complete clinical, risk, and identification data. This table is not intended to represent a complete literature search.

ical symptoms experienced by the patients reported here were not unusual since many of the patients infected with *B. cereus* exhibit cough, fever, hemoptysis, positive blood and sputum cultures, and X ray evidence of infiltrates.

B. cereus is a spore former and is found normally in the soil. Presumably, soil and dust particles could be the source of the organism in these episodes. Welders are exposed to large amounts of dust on a daily basis. However, there is no evidence that might explain why these events occurred at this time and followed such a fulminant course. Since there was no indication of the symptoms of food-borne illness, one might assume that ingestion was an unlikely method of inoculation. Pulmonary infections with *B. cereus*, while unusual, are potentially life threatening. It has long been known that pulmonary anthrax caused by *B. anthracis* (woolsorter's disease) is often fatal and usually accompanied by overwhelming sepsis (11). Pulmonary anthrax begins abruptly with high fever, dyspnea, and chest pain; it progresses rapidly and often is fatal before treatment can halt the invasive aspect of the infection (6). These reported episodes of pneumonia and bacteremia due to *B. cereus* are reminiscent of a *B. anthracis*-like infection. The large numbers of *B. cereus* organisms coupled with the variety of toxins and enzymes they can produce (3) could have led to the rapid course and fatal outcome of this disease.

REFERENCES

1. **Bekemeyer, W. B., and G. A. Zimmerman.** 1985. Life-threatening complications associated with *Bacillus cereus* pneumonia. Am. Rev. Respir. Dis. **131:**466–469.
2. **Coonrod, J. D., P. J. Leadley, and T. C. Eickhoff.** 1971. *Bacillus cereus* pneumonia and bacteremia. Am. Rev. Respir. Dis. **103:**711–714.
3. **Drobniewski, F. A.** 1993. *Bacillus cereus* and related species. Clin. Microbiol. Rev. **6:**324–338.
4. **Funada, H., T. Machi, and T. Matsuda.** 1991. *Bacillus cereus* pneumonia with empyema complicating aplastic anaemia—a case report. J. Jpn. Assoc. Infect. Dis. **65:**477–480.
5. **Gascoigne, A. D., J. Richards, K. Gould, and G. J. Gibson.** 1991. Successful treatment of *Bacillus cereus* infection with ciprofloxacin. Thorax **46:**220–221.
6. **Ihde, D. C., and D. Armstrong.** 1973. Clinical spectrum of infections due to *Bacillus* species. Am. J. Med. **55:**839–845.
7. **Jonsson, S., J. Clarridge, and E. J. Young.** 1983. Necrotising pneumonia and empyema caused by *Bacillus cereus* and *Clostridium bifermentans*. Am. Rev. Respir. Dis. **127:**357–359.
8. **Leff, A., R. Jacobs, V. Gooding, J. Hauch, J. Conte, and M. Stulbarg.** 1977. *Bacillus cereus* pneumonia. Survival in a patient with cavitary disease treated with gentamicin. Am. Rev. Respir. Dis. **115:**151–154.
9. **Sliman, R., S. Rehm, and D. M. Shlaes.** 1987. Serious infections caused by *Bacillus cereus*. Medicine (Baltimore) **66:**218–233.
10. **Stapler, T., V. Caneuscu, and M. Voiculescu.** 1965. Bronchopneumonia with lethal evolution determined by a microorganism of the genus *Bacillus* (*B. cereus*). Rom. Med. Rev. **2:**7–9.
11. **Turnbull, P. C. B., and J. M. Kramer.** 1995. Bacillus p. 349–356. *In* P. R. Murray, E. J. Baron, M. A. Pfaller, F. C. Tenover, and R. H. Yolken (ed.), Manual of clinical microbiology, 6th ed. American Society for Microbiology, Washington, D.C.