Bacillus cereus Pneumonia with Empyema Complicating Aplastic Anemia —A Case Report—

Hisashi FUNADA, Toshihiko MACHI and Tamotsu MATSUDA

The Protected Environment Unit and Third Department of Medicine, Kanazawa University School of Medicine, Kanazawa 920, Japan

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Introduction

More evidence has recently been presented suggesting that *Bacillus* species other than *Bacillus anthracis* should not be readily discarded as contaminants, especially when isolated from neutropenic patients with cancer[1~3]. Although very rare, infections with *Bacillus cereus*, such as bacteremia, pneumonia or meningitis, are most likely to occur in patients with acute leukemia, and often follow a rapidly fatal course[1~4]. We describe here a successfully treated case of *B. cereus* pneumonia with empyema complicating aplastic anemia.

Case Report

A 60-year-old housewife with aplastic anemia of six years' duration was admitted on September 18, 1989, to Kanazawa University Hospital with a one-week history of malaise, and left-sided pleuritic chest pain (Fig. 1).

On admission, she had a temperature of 38.8°C. Scattered petechiae were found over the extremities and trunk. Her breath sounds had decreased at the left lung base, but no pleural rubs were heard.

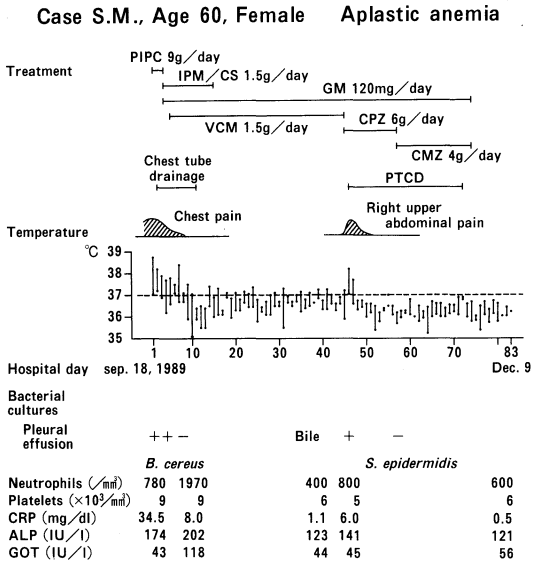

Fig. 1 Clinical course of a patient with aplastic anemia who developed *Bacillus cereus* pneumonia with empyema.
Abbreviations: PIPC, piperacillin; IPM/CS, imipenem/cilastatin; GM, gentamicin; VCM, vancomycin; CPZ, cefoperazone; CMZ, cefmetazole; PTCD, percutaneous transhepatic cholangial drainage; CRP, C-reactive protein; ALP, alkaline phosphatase; and GOT, glutamic oxaloacetic transaminase. Reference ranges for laboratory tests: CRP, <0.5 mg/dl; ALP, 86~272 IU/l; and GOT, 9~42 IU/l.

別刷請求先：（〒920）金沢市宝町13番1号
金沢大学医学部付属病院高密度無菌治療部
舟田　久

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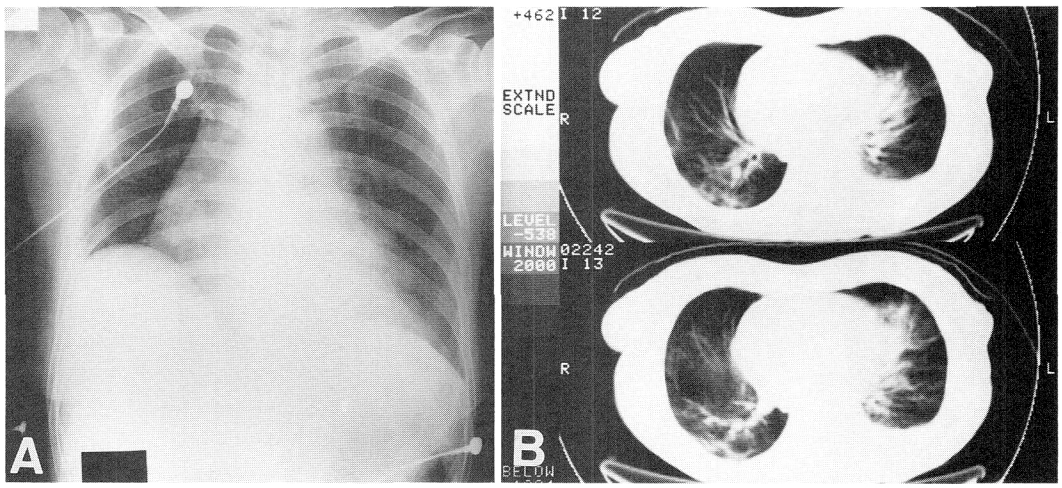
Fig. 2 Chest roentgenogram on admission (taken in the supine position), showing a left lower lobe infiltrate with a pleural effusion (A), as revealed clearly by computed tomography (B), which also demonstrates a faint infiltrate in the right lung.



Laboratory data showed anemia (hemoglobin, 9.5 g/dl), leukopenia (1200/mm^3, with 65% neutrophils), and thrombocytopenia (9000/mm^3). A chest roentgenogram showed a left lower lobe infiltrate associated with a moderate pleural effusion, as revealed clearly by computed tomography (Fig. 2). Thoracentesis revealed a bloody, serosanguinous fluid. Large gram-positive rods were seen with neutrophils and erythrocytes on the gram-stained smear (Fig. 3), subsequently identified as *B. cereus* on culture. A throat culture grew only normal flora, and blood cultures were negative.

Directly after admission, she was placed on intravenous piperacillin, with a central venous catheter inserted to facilitate venous access. Therapy was changed two days later to intravenous gentamicin and imipenem/cilastatin, to which the isolate had in vitro sensitivity. A thoracostomy tube was inserted into the left side of her chest with the aid of platelet transfusions. On day 5 after admission, a repeat chest roentgenogram showed a right pleural effusion, which was also drained by placement of a chest tube. Intravenous vancomycin was therefore added to the antibiotic regimen. She became afebrile in keeping with the decrease in drainage from the tubes, which were removed on day 11. A few days later, however, hepatic dysfunction was noted, and imipenem/cilastatin was discontinued. The neutrophil count fluctuated between 400 to 1970/mm^3. Her general condition and hepatic function had considerably improved by day 45, when she developed acute cholecystitis with *Staphylococcus epidermidis*, which was successfully treated with antibiotics and drainage.

On day 82, she was discharged with bilateral mild pleural thickening and costophrenic angle blunting seen on the chest roentgenogram (Fig. 4).

Discussion

This is the first case, to our knowledge, in which *B. cereus* pneumonia has been reported in an adult with aplastic anemia, although *B. cereus* is widely distributed as the most common aerobic spore-bearer in nature[5]. We urge consideration of serious *Bacillus* infection in patients with aplastic anemia.

The breakdown of mucosal barriers, together with neutropenia, plays an important role in allowing ready access of the usually saprophytic organism to deeper tissues[6]. In our case, therefore, a small area of bronchial submucosal hemorrhage, resulting in disruption of the mucosal surface, may have been a

Fig. 3 Gram-stained smear of pleural effusion, showing large gram-positive rods (arrow) with neurtrophils. Many erythrocytes were faintly seen as ghost cells through the staining process. (Original magnification, ×400)

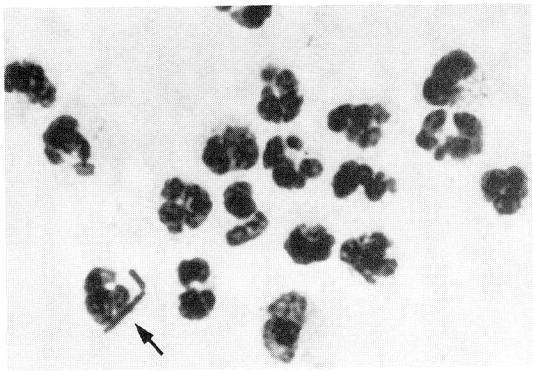
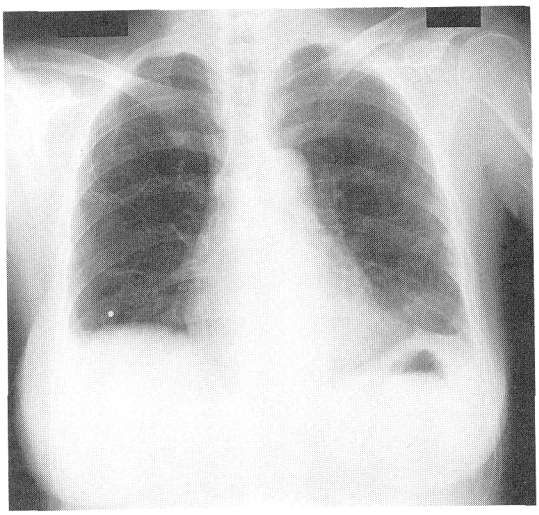


Fig. 4 Chest roentgenogram at discharge (taken in the upright position), showing bilateral pleural thickening with costophrenic angle blunting.



probable portal of entry for pneumonia, considering that the patient had petechiae and severe thrombocytopenia on admission.

The outcome of serious infection in patients with neutropenia is usually ominous in the absence of both early diagnosis and an increase in the neutrophil count during therapy[6,7]. In our case, a gram stain and culture of the pleural effusion obtained by thoracentesis revealed the causative organism. In retrospect, however, the non-encapsulated, wide, long, and straight gram-positive rods seen on the smear (Fig. 2) seemed to be morphologically suggestive, although not characteristic, of *B. cereus*. Such tentative diagnosis may have led to prompt choice of the antibiotics active against *B. cereus*, which include vancomycin, gentamicin, imipenem, ciprofloxacin, and clindamycin[1,3,4,8]. Because of β-lactamase production, the organism is usually resistant to penicillins and cephalosporins[7,8], which are commonly used for the empiric treatment of febrile patients with neutropenia. Anyway, the increase in our patient's neutrophil count during therapy (780/mm^3 on admission to 1970/mm^3 on day 10, as shown in Fig. 1), closely associated with the timely change to appropriate antibiotics, seems to have contributed to the successful outcome.

Incidentally, the pleural fluid related to *B. cereus* pneumonia in our patient was bloody and serosanguinous. The organism produces exotoxins that can cause hemolysis, and tissue necrosis[4], and has an affinity for blood vessels which may cause thrombosis and infarction followed by hemorrhage[2]. No doubt alteration in the nature of drainage was observed in parallel with eradication of the organism, although her platelet count remained less than 10000/mm^3 during the infection.

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再生不良性貧血の症例にみられた *Bacillus cereus* による膿胸をともなった肺炎

金沢大学医学部付属病院高密度無菌治療部・第3内科
舟田　久　真智　俊彦　松田　保

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要　旨

60歳，主婦の再生不良性貧血の症例に *Bacillus cereus* による膿胸をともなった肺炎がみられた．試験穿刺でえられた血性胸水中に好中球に混じって大きなグラム陽性桿菌がみられた．分離菌が感性を示したイミペネム，ゲンタマイシン，バンコマイシンの投与に加えて，胸腔ドレナージを施行して，治癒に導くことができた．治療中の好中球数の増加も治療の奏効に作用したと考えられた．