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Pleural Empyema Due to *Clostridium difficile* and *Clostridium cadaveris*

Pleuropulmonary infections with clostridial species, usually *Clostridium perfringens*, have been reported occasionally [1]. We describe a patient with a bronchogenic cyst who developed pleuropulmonary empyema due to *Clostridium difficile* and *Clostridium cadaveris* after surgical removal of the cyst.

A 33-year-old male pig farmer had had a bronchogenic cyst in the upper lobe of the left lung for ~8 years. Because the cyst was enlarging, segmental resection was performed on 31 January 1996. On the sixth postoperative day, the patient developed fever with leukocytosis (leukocyte count, 17,700 cells/mm³). A chest radiograph revealed pleural effusion on the left side. Foul-smelling fluid was obtained via transthoracic puncture on 11 February.

Culture of the material yielded two anaerobic gram-positive spore-forming rods with different colony morphologies and odors. With use of the API 20A system (bioMérieux, Marcy l'Etoile, France) and gas chromatography, the isolates were identified as *C. difficile* and *C. cadaveris* (the identity of the latter isolate was confirmed by Dr. M. McTeague, Wadsworth Anaerobe Laboratory, Los Angeles). The *C. difficile* strain produced cytotoxin B. The Etest (AB BIODISK, Solna, Sweden) showed that both organisms were susceptible to amoxicillin/clavulanate and metronidazole. Therapy with iv amoxicillin/clavulanate (1,000/200 mg t.i.d.) was started on 11 February.

On 19 February, the patient developed a bronchopleural fistula. A second thoracotomy was performed on 21 February. The rest of the left lung was found to be necrotic, and pneumonectomy was performed. Intravenous metronidazole (500 mg t.i.d.) was added to the patient's regimen of amoxicillin/clavulanate. Cultures of fluid from the thoracic drain were repeatedly negative after antimicrobial therapy was initiated. The drain was removed on 1 March, antibiotic therapy was stopped, and the patient was discharged from the hospital.

Four days later, he returned to the hospital with fever and pain on the left side of the thorax. A chest radiograph revealed a normal pleural cavity but enlarged cardiac contours. An echocardiogram revealed a 5-cm pericardial effusion. A third thoracotomy with pericardiectomy was performed on 25 March for debridement followed by drainage and daily rinsing with 5% povidone-iodine. Culture of the pleural fluid yielded only *C. cadaveris*; the isolate was susceptible in vitro to amoxicillin/clavulanate and metronidazole. The pericardial fluid was sterile. The patient again received

the intravenous combination of amoxicillin/clavulanate (1,000/200 mg q4h) and metronidazole (500 mg t.i.d.). Follow-up cultures of fluid from the drain were all sterile. Therapy with metronidazole was stopped on 12 April, and the drain was removed 1 week later. Therapy with amoxicillin/clavulanate was continued until 26 May (during the last 2 weeks, amoxicillin/clavulanate was administered orally at a dosage of 500/125 mg t.i.d.). A CT scan showed no signs of abscesses. The leukocyte count was 8,200/mm³. No β -lactamase production was detected in the *C. cadaveris* isolate. The patient was discharged from the hospital in good clinical condition with a prescription for oral amoxicillin (500 mg q.i.d.). As of this writing (3 months later), he is clinically stable.

Few cases of pleural infection with *C. difficile* have been reported [2, 3]. *C. cadaveris* is not known to produce a toxin and is considered to be nonpathogenic for humans and laboratory animals [4]. However, *C. cadaveris* bacteremia (in two immunocompromised patients) and *C. cadaveris*-associated spontaneous bacterial peritonitis have been described [5, 6]. Aspiration of aerosol from animal material while handling dead piglets seems to be a logical explanation for our patient's pulmonary infection. The severity of his infection with two anaerobic organisms that are both considered to be of little pathogenic importance, with destruction of a whole lung despite adequate antimicrobial therapy, is remarkable.

We believe that the *C. cadaveris* isolate persisted after 4 weeks of therapy because of its capacity to sporulate. The persistence of *C. cadaveris* despite adequate therapy was not described in the three previously mentioned case reports [5, 6]; those patients all died of complications during hospitalization, which obscured the possible role of *C. cadaveris*. We conclude that the pathogenicity of *C. cadaveris* needs further study.

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