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Major article

Infection control strategies that successfully controlled an outbreak of *Mycobacterium abscessus* at a cystic fibrosis center

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Background: *Mycobacterium abscessus* infection in patients with cystic fibrosis (CF) can result in accelerated clinical decline and the potential for direct or indirect transmission between patients has been recently demonstrated. Data on the outcomes of *M abscessus* outbreaks and the efficacy of specific infection control procedures in patients with CF remain limited. This study provides follow-up from an outbreak of pulmonary *M abscessus* in our center, highlighting outcomes and strategies that appear to have prevented further spread of the organism.

Methods: Data from our adult CF center (1989-2015) were analyzed, including chart reviews of all patients with positive mycobacterial sputum cultures, cultures from environment surfaces, and epidemiologic evaluation of infected patients. Following an *M abscessus* outbreak in 2009, infection control policies were intensified based on CF guidelines and surveillance data were collected and reviewed.

Results: Five cases of *M abscessus* were involved in the outbreak; 3 patients died during follow-up. An environment search failed to reveal an intermediary source of transmission between patients. After implementation of infection control measures composed of staff/patient education, environment sterilization, and patient isolation, no new cases were detected.

Conclusions: Direct or indirect patient-to-patient transmission of *M abscessus* is a threat in the CF population. A multifaceted infection control strategy based on CF guidelines was effective in halting transmission in our center.

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Mycobacterium abscessus is an organism increasingly recognized as causing chronic lung disease, often in patients with altered host defenses or disrupted airway clearance mechanisms. In cystic fibrosis (CF), airway integrity is compromised by abnormal mucus composition, reduced mucociliary clearance, and chronic bacterial colonization leading to progressive bronchiectasis. Due to structural airway damage and other host factors, *M abscessus* is a threat to infect the airway in patients with CF, and has been found in the sputum of 3%-10% of patients with CF in the United States and Europe, with suggestion of increased prevalence in recent years.[1,2] It can behave as an invasive pathogen leading to progressive pulmonary decline, it can preclude safe lung transplantation, and treatment is often unsuccessful or poorly tolerated.[2-4]

Acquisition of *M abscessus* has been traditionally attributed to environmental exposure, including soil, dust, and water. Although the extent of its environmental niche remains unclear, the organism has been cultured from various water sources,[5] and prior studies have supported this mechanism of acquisition based on the identification of genetically unique strains within single-center CF cohorts with *M abscessus* infection.[2,6] More recently, patient-to-patient cross-infection has been demonstrated with *M abscessus*, either via direct exposure, or more likely indirect exposure from shared intermediary sources. This was recently described in the non-CF population, where an outbreak of *M abscessus* took place in an intensive care unit among patients with a variety of comorbidities.[7] The problem of patient-to-patient transmission is especially relevant in the CF population, and in 2012 our group reported an outbreak involving an index patient and 4 secondary cases of patients with CF who developed *M abscessus* subsp *massiliense* pulmonary disease. The 5 individual patient isolates were not

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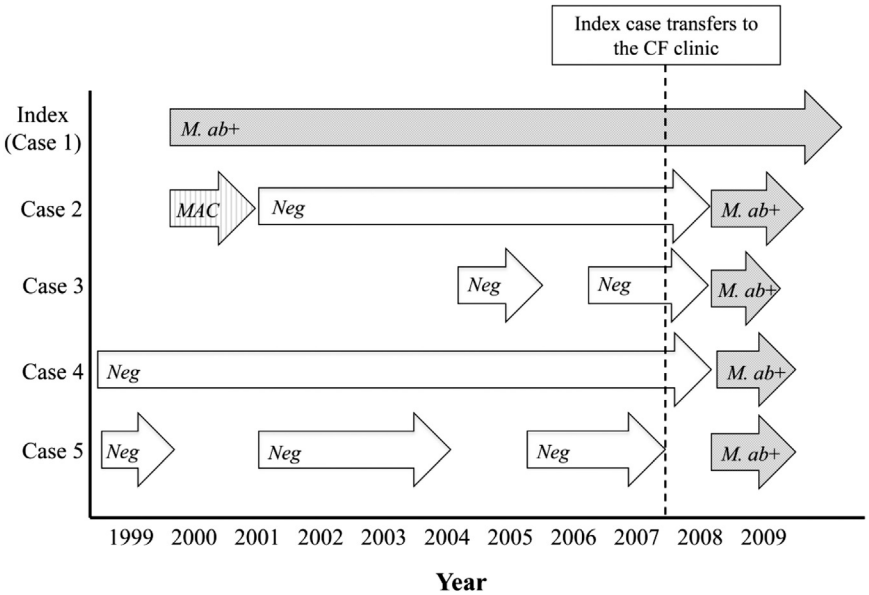

Fig 1. Serial annual mycobacterial cultures from the outbreak case patients: The 5 patients are labeled as index (case 1) and cases 2 through 5 based on chronological order of identification of *Mycobacterium abscessus* subsp *massiliense* (M. ab) in their sputum. Blank spaces indicate that either specimens were not sent or the results are unknown. Dotted line denotes the date of transfer of the index case patient to the clinic. *MAC*, *Mycobacterium avium* complex; *Neg*, negative.

distinguishable by DNA analysis, suggesting direct or indirect spread of an identical strain.[8] Additional evidence in the CF population was provided by Bryant et al,[9] who reported 2 clustered outbreaks of *M abscessus* in the United Kingdom in which isolates were genetically indistinguishable by the methods used, again signifying direct or indirect cross-infection within the center.

There has been growing consciousness of the need for infection control in the setting of CF. Guidelines have been recently updated to emphasize the importance of avoiding cross-contamination between patients.[10] Despite this, data outlining the efficacy of interventions targeting pathogen spread in CF patients remain limited, as are data on clinical outcomes of infectious outbreaks in this population. Our center's *M abscessus* outbreak in 2009 was unique at that time, and upon discovery became the impetus for aggressive infection control and isolation processes.[8] We now report complete follow-up from the initial outbreak, including a description of measures that led to a sustained lack of transmission within our CF center.

METHODS

Study setting

Patients were aged ≥18 years and attended our academic adult CF clinic. Chart review was performed on CF patients with positive *M abscessus* screening cultures from the clinic's inception in 1989, including those identified in the previously reported outbreak.[8] Our university's Human Subjects Division approved the study without explicit subject consent (approval 49.287). Clinical data collected included age, gender, CF genotype, body mass index (BMI), gastroesophageal reflux disease (GERD) (defined by the need for acid suppressive medications), vitamin D levels (deficiency defined as 25-hydroxy level ≤30 ng/mL), sputum cultures, pulmonary function tests, and treatment regimen. Epidemiologic data among outbreak cases included medical provider contact, hospital admission dates, visit times to clinic and diagnostic locations, and source of domestic water supply. Infection control protocols were documented prospectively and reviewed for the study.

Sputum processing

Mycobacteriology specimen processing was performed as previously described, with isolates identified as *M abscessus* subsp *massiliense* by polymerase chain reaction-restriction enzyme analysis.[11] Susceptibility testing used the Clinical and Laboratory Standards Institute broth microdilution method for rapidly growing mycobacteria, with susceptibility breakpoints defined by Clinical and Laboratory Standards Institute guidelines.[12] Strain-relatedness between outbreak cases was assessed using repetitive unit sequence based polymerase chain reaction (rep-PCR), pulsed-field gel electrophoresis, and sequencing.[8,13,14]

RESULTS

Outbreak

The outbreak was briefly described previously.[8] The index case was a 22-year-old man with CF and an forced expiratory volume in 1 second (FEV_1) of 28% predicted. He was first documented to be positive for *M abscessus* subsp *massiliense* in 2000 while receiving care at another institution. Despite treatment, he was 4+ smear positive in January 2007, at which time he was referred to our center's lung transplantation clinic (located in a shared space with the CF clinic) for consideration for double lung transplantation. He was listed for transplant, and established care in our CF clinic in July 2007. Before this time there had been no previous reports of patient-to-patient transmission of pulmonary *M abscessus*, and the clinic adhered to the recommended general CF infection control guidelines.[15]

Eight months after the index patient entered the clinic, 4 additional CF patients were found to be newly colonized with *M abscessus* subsp *massiliense* after having been historically negative on screening over the previous decade (Fig 1). The age of the 5 affected patients ranged from 22-38 years. All were coinfected with *Pseudomonas aeruginosa* and had severe airflow obstruction with FEV_1 ranging from 27%-44% predicted. Four of the five were malnourished with abnormally low BMI (<20) (Table 1).

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