Wound Infection Caused by *Branhamella catarrhalis*

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*Branhamella catarrhalis* was isolated from sputum, tracheal secretions, and a nonhealing and infected thoracic surgical wound in a 59-year-old woman who had a history of a chronic, interstitial fibrosis and who had undergone an open lung biopsy procedure. The patient’s upper respiratory tract was the likely source of the organism. To our knowledge, this is the first report of a wound infection caused by *B. catarrhalis*.

*Branhamella catarrhalis* is a commensal of the upper respiratory tract of humans and is only rarely associated with human infections. However, over the last 20 years, *B. catarrhalis* has become increasingly recognized as a pathogen that can cause lower respiratory tract infections (6, 10, 14, 18; G. V. Doern and R. E. Schmid, Clin. Microbiol. Newsl. 8:34–36, 1986) and acute otitis media (4, 13, 20), and it has been implicated in maxillary sinusitis (3). *B. catarrhalis* also has been reported to cause meningitis (8), cellulitis (11), endocarditis (17), keratitis (22), conjunctivitis (12), bacteremia (14, 18; Doern and Schmid, Clin. Microbiol. NewsL.), septic arthritis (5), urethritis (9), and exacerbation of bronchitis (15).

The laboratory identification of *B. catarrhalis* is based on Gram stain, colonial morphology, growth characteristics, inability to produce acid from selected carbohydrates, and the production of oxidase and DNase (6).

Most isolates of *B. catarrhalis* produce at least one β-lactamase, and production of these β-lactamases is not usually plasmid mediated (6, 19). Strains producing β-lactamases are resistant to penicillin and ampicillin, but in vitro susceptibility results may be misleading (7). *B. catarrhalis*, including β-lactamase-producing strains, is usually susceptible to erythromycin, tetracycline, chloramphenicol, trimethoprim-sulfamethoxazole, ampicillin-sulbactam, utoxopenicillins, aminoglycosides, cephalosporins, and fluoroquinolones (1, 6, 7, 16; Doern and Schmid, Clin. Microbiol. NewsL.) and is resistant to clindamycin.

In this report we describe the isolation of *B. catarrhalis* from a nonhealing and infected thoracic surgical wound in a 59-year-old woman who had a history of a chronic, interstitial fibrosis and who had undergone an open lung biopsy procedure. To our knowledge, this is the first report of a wound infection caused by *B. catarrhalis*.

**CASE REPORT**

A 59-year-old woman with a history of over 50 pack years of smoking and alcohol abuse was hospitalized in 1982, 1984, and 1986 for recurrent bronchitis and pneumonias, control of adult-onset diabetes mellitus, and various combinations of chronic obstructive pulmonary disease (bronchitis), fatigue, anorexia, weight loss, chronic cough, dehydration, and malnutrition. At various times in 1986, the patient was treated with doxycycline, erythromycin, gentamicin, cefaclor, and prednisone. Before the hospitalization described below, the patient had dyspnea, fatigue, and cough productive of yellow sputum. Chest X ray revealed extensive bullous changes with increased interstitial markings. Pulmonary function tests demonstrated a restrictive pattern with reduction in all lung volumes and reduced carbon monoxide diffusing capacity to 10 ml/min per mm Hg (1 mm Hg is equal to ca. 133.3 Pa) (normal capacity is 17 to 28 ml/min per mm Hg).

In December 1986, a bronchoscopy performed on an outpatient basis revealed purulent secretions and inflammatory changes in the bronchioles and provided brush and washing specimens that were culture negative for bacteria, fungi, and mycobacteria. Cytological examination of washings obtained during the bronchoscopy procedure revealed mildly atypical squamous epithelial cells and pronounced inflammatory reactivity. The patient was treated with trimethoprim-sulfamethoxazole and clindamycin for 1.5 months.

The patient was rehospitalized in February 1987 because of her continuing illness and gradual deterioration in health, and an open lung biopsy procedure was performed. The thoracotomy procedure revealed dense pleural adhesions, and the right middle and upper lobes were encased in inflammatory reaction. Subsequent decortication of the right lung revealed a middle lobe empyema overlying a bronchoalveolar fistula. Approximately 15 ml of purulent exudate was removed from the empyema cavity, which subsequently was closed with an anterior serratus muscle flap. Multiple surgical specimens were obtained for Gram stain, KOH examination, acid-fast stains, *Pneumocystis* stain, and fluorescent stain for *Legionella* spp. Cultures were obtained for anaerobes, aerobes, fungi, acid-fast bacilli, and *Legionella* organisms. All stains and cultures were negative. Two representative biopsy specimens of lung parenchyma also were obtained. The patient was treated with gentamicin, clindamycin, and imipenem-cilastatin for 2 of the following 4 weeks, during which time she continued to show no improvement and to have intermittent fevers. She also developed tenderness, erythema, and pressure necrosis along the margins of the surgical wound. Approximately 4 weeks after the thoracotomy, the wound was opened and a large amount of pus was expressed from surrounding subcutaneous tissues. The wound communicated with the chest cavity. Drainage of the wound abscess was accompanied by rapid defervescence. Samples of sputum, tracheal secretions, and pus from deep in the thoracotomy wound were cultured for bacteria and fungi. During the weeks 5 and 6 of her hospitalization, the patient remained afebrile and was treated with trimethoprim-sulfamethoxazole and a 4-day course of clin.

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damycin. During the next 3 weeks of hospitalization, she continued to be treated with trimethoprim-sulfamethoxazole, and her wound was debrided and continually dressed and packed. The patient showed some gradual improvement even though she had intermittent episodes of fever. Throughout her hospitalization, blood samples were cultured for bacteria and fungi. The patient was discharged on her own and her family's volition 9 weeks after the open lung biopsy procedure with a bronchocutaneous fistula that was culture positive for \textit{B. catarrhalis}, chronic intrafolliculmonary fibrosis, and adult-onset diabetes mellitus. She convalesced in a nursing home facility until she died 6 weeks after discharge. A postmortem examination was not performed.

### MATERIALS AND METHODS

For bacterial cultures, sputa and tracheal secretions were inoculated onto sheep blood agar, chocolate blood agar, and eosin-methylene blue agar plates; pus expressed from the wound was inoculated onto colistin-nalidixic acid agar in addition to the media used for sputa. Cultures were incubated in 5 to 10% CO₂ at 35°C and examined daily for 2 days. Pus was cultured for anaerobic bacteria by inoculation of rabbit blood agar, laked rabbit blood agar with gentamicin and vancomycin, phenylethyl alcohol-sheep blood agar, and hemin-thioglycolate broth. Plates were inoculated anaerobically at 35°C for 48 h, and hemin-thioglycolate broth was inoculated in 5 to 10% CO₂ for 7 days. For fungal cultures, the three specimen types were inoculated onto (i) inhibitory mold agar with chloramphenicol, (ii) inhibitory mold agar with ciprofloxacin, (iii) brain heart infusion agar containing sheep blood, chloramphenicol, and gentamicin, and (iv) brain heart infusion agar with cycloheximide. Fungal cultures were incubated in room air at 30°C for 30 days. Blood samples were inoculated into a nonvented tryptic soy broth (Difco Laboratories, Detroit, Mich.), a Septi-Chek biphasic system (Roche Diagnostics, Div. Hoffmann-LaRoche, Inc., Nutley, N.J.), and a 10-ml Isolator tube (E. I. du Pont de Nemours and Co., Wilmington, Del.) as previously described (21). Bacterial colonies were Gram stained and tested for oxidase activity. Colonies which consisted of aerobic, gram-negative, diplococci were tested for acid production in glucose, maltose, lactose, and sucrose and for DNase production by using RIM kits for the rapid identification of \textit{Neisseria} and \textit{ Branhamella} species (Austin Biological Laboratories, Austin, Tex.). β-Lactamase production was determined by using a chromogenic cephalosporin test (Cefinase disks; BBL Microbiology Systems, Cockeysville, Md.). MICs of antibiotics were determined by an agar-dilution technique (2).

### RESULTS

Gram-stained smears of tracheal secretions, sputa, and deep thoracotomy wound specimens showed numerous polymorphonuclear leukocytes and both intra- and extracellular gram-negative diplococci. Abundant aerobic, gram-negative, oxidase-positive diplococci were isolated from the three aforementioned specimen types. The isolate did not utilize glucose, maltose, lactose, or sucrose; it degraded DNA and was identified as \textit{B. catarrhalis}. The isolate produced β-lactamase, and antimicrobial MICs were as follows: cefazolin, ≤ 8 μg/ml; erythromycin, ≤0.5 μg/ml; and trimethoprim and sulfamethoxazole, <0.5 and 9.5 μg/ml, respectively. A few colonies of \textit{Aspergillus fumigatus} also were isolated from each of the three specimen types, and a few colonies of an unidentified yeast (not \textit{Cryptococcus} sp.) were isolated from tracheal secretions. Numerous blood cultures taken throughout hospitalization were negative for bacteria and fungi.

Microscopic examination of the two biopsy specimens revealed interstitial fibrosis, honeycombing, and bronchiolar metaplasia.

### DISCUSSION

To our knowledge, this is the first report of a wound infection caused by \textit{B. catarrhalis}. The patient's upper respiratory tract was the likely source of the \textit{B. catarrhalis} isolate, which was isolated from the infected wound as well as from sputum and tracheal secretions.

The pathogenesis of the \textit{B. catarrhalis} wound infection is not known. However, tissues surrounding the surgical wound probably became infected with \textit{B. catarrhalis} when the fistula closure failed and the resulting open empyema cavity intermittently touched the wound or when \textit{B. catarrhalis}-containing adhesions developed between the lung and the wound. Subsequently, one or more subcutaneous abscesses developed at the wound site. Other reports have documented \textit{B. catarrhalis} infections that probably resulted from the direct spread of \textit{B. catarrhalis} from the site of a surgical procedure that involved the upper respiratory tract to the site where the infection occurred, e.g., cellulitis at a cerebrospinal fluid shunt site after a tracheostomy (11), meningitis after neurosurgery that involved the right ethmoid sinus (8), and endocarditis after a tooth extraction (17).

Several observations suggested that our patient probably had some degree of \textit{B. catarrhalis} pneumonia or at least some form of \textit{B. catarrhalis}-induced pulmonary disease. The patient had several predisposing factors for respiratory infection, including an age of greater than 50 years, steroid therapy, chronic obstructive pulmonary disease (bronchitis), alcohol and tobacco abuse, and diabetes mellitus. At various times, the patient showed many of the signs and symptoms of acute exacerbation of chronic bronchitis and of pneumonia such as low-grade fevers, productive cough, dyspnea, tachycardia, and chest infiltrates. The patient's sputa contained high numbers of polymorphonuclear leukocytes and many intracellular gram-negative diplococci. \textit{B. catarrhalis} was the only potential bacterial pathogen isolated from the patient's specimens. All of the aforementioned observations have been previously reported as being characteristic of \textit{B. catarrhalis}-induced pulmonary disease (6, 10).

It is difficult to know when \textit{B. catarrhalis} pneumonia developed in the patient. We were not able to obtain evidence of positive lower respiratory tract cultures during any of her hospitalizations in 1986 or before.

It is possible that the pulmonary abscess and fistula developed over a long period before admission and were at least partially responsible for the chronic and debilitating lung process. Alternatively, and as evidenced by preadmission roentgenograms that did not show evidence of abscess formation, \textit{B. catarrhalis} pulmonary infection could have been precipitated by the open lung biopsy procedure.

The significance of \textit{B. catarrhalis} infection in this patient is not known, but \textit{B. catarrhalis} wound infection probably played a role in eliciting fever in the patient because she was afebrile for 10 days after the wound was drained, debrided, and dressed.

\textit{B. catarrhalis} is a part of the normal flora of the upper respiratory tract and is a pathogen capable of producing severe disease. To our knowledge, this is the first report of a
wound infection caused by *B. catarrhalis*. Labortorians and clinicians should be aware that *B. catarrhalis* infections are possible after surgical procedures that involve the respiratory tract.

**LITERATURE CITED**


