A common source outbreak of *Acinetobacter* pulmonary infections traced to Wright respirometers

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Summary

Over a 30-day period, *Acinetobacter calcoaceticus* var. *anitratus* was the responsible pathogen for hospital-acquired pneumonia in 10 patients, and resulted in the colonization of the upper respiratory tract in an additional 9 patients. Wright respirometers contaminated by this organism were shown to be the common source for the outbreak as indicated by the recovery of a single serotype (8J), the inability to recover *Acinetobacter* from any other environmental source, and the demonstration that moisturized Wright respirometers are capable of 'aerosolizing' fluids containing *Acinetobacter*.

Materials and methods

Patients

Hartford Hospital, Connecticut, is a 1000-bed, university affiliated, teaching hospital with 6 intensive care units. The outbreak consisted of 10 patients who developed evidence of *Acinetobacter* pneumonia, and an additional 9 patients who were colonized over a period of 30 days in 4 intensive care units. Patients with a lobar pulmonary infiltrate on chest film, fever, leucocytosis and a purulent sputum containing *Acinetobacter* as the sole or predominant organism were considered to have *Acinetobacter* pneumonia.

Microbiology

Sputum specimens were collected by nasotracheal aspiration, examined in the laboratory by Gram stain and cultured on MacConkey and 5% sheep blood agar plates. Swabs of respiratory equipment were transported to the laboratory in Amies transport medium and plated on the above media. After overnight incubation in air with 5% CO₂ added, the agar plates were examined. When 6 or more colonies of presumptive Gram-negative organisms were observed, a representative colony of each type was inoculated into an API 20 E biochemical strip for identification. The antibiotic susceptibility of *Acinetobacter* to ampicillin, cephalothin, gentamicin, kanamycin, tobramycin, co-trimoxazole, minocycline and chloramphenicol was determined by the Bauer-Kirby method (National Committee for Laboratory Standards, Los Angeles, 1976). Isolates identified as *Acinetobacter* were serotyped in the Analytical Bacteriology Branch at the Center for Disease Control.

Introduction

Acinetobacter calcoaceticus var. *anitratus* is a relatively frequent cause of upper respiratory tract colonization in hospital patients (Irving and Herrick, 1967; Inclan *et al.*, 1965; Green, Johnson and Shively, 1960; Gardner *et al.*, 1970; Ramphal and Kluge, 1979). *Acinetobacter* is a hydrophilic organism, and respiratory infections due to this organism have been associated with fine particle nebulizers and humidifiers. These outbreaks have always involved multiple *Acinetobacter* serotypes (Kelsen *et al.*, 1977; Smith and Massanan, 1977). In this communication, the clinical and epidemiological features are reported of a large outbreak of respiratory tract colonization and infection, in patients in hospital, due to a single *Acinetobacter* serotype (8J). Contaminated Wright respirometers were found to be the common source of the outbreak.

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FIG. 1. *Acinetobacter* infections and colonizations at Hartford Hospital, Connecticut, from April to May 1977. □, one case, colonization; ■, one case infection.

Epidemiology

Description of the outbreak

The usual incidence of *Acinetobacter* isolates from the sputum at Hartford Hospital is 235.8 cases/10⁵ patients or an average of 7.4 isolates per month. During routine epidemiological surveillance in April, an abrupt and unexplained increase in the recovery of *Acinetobacter* from sputum cultures was noted with 732.9 cases/10⁵ or 19 cases occurring during the month of the outbreak (Fig. 1).

During this outbreak, 10 patients developed *Acinetobacter* pneumonia approximately 8 days (range 6–10 days) after the introduction of Wright respirometers.

Acinetobacter was recovered from purulent sputum approximately 13 days (range 10–19 days) after the introduction of the respirometers. The fever peaked on the 14th day (range 10–19 days), and ranged from 100.6° to 106.4° F. A patchy infiltrate appeared on chest X-ray about 10 days (range 8–12 days) after the introduction of the respirometers. The initial radiological abnormality detected in all patients was that of a diffuse infiltrate in one or both of the lower lobes. All patients eventually developed spread to the contralateral lower lobe, and 2 of the patients also developed upper lobe involvement. Additionally, 2 patients developed diffuse bronchopneumonia during the course of their infection. No patient developed a pleural effusion or sign of consolidation by X-ray or physical examination. White blood cell counts also peaked on day 13 (range 8–25 days) of the outbreak. Peak WBC counts averaged $19.8 \times 10^9/l$ (range $15.4 \times 10^9/l$ – $2.6 \times 10^9/l$) with the exception of a pre-leukaemic patient who remained leucopenic while in hospital. Shaking chills or chest pain were not present during the illness of

any patient. *Acinetobacter* was not recovered from the blood of any patient during the outbreak.

Investigation

When the occurrence of the outbreak was recognized, extensive sampling of equipment and personnel was quickly carried out. Nasal and skin cultures of medical and para-medical personnel in the intensive care units were obtained, as were cultures of the suction apparatus, respirators, respirator tubing, respirator air effluent, and intravenous polyethylene catheters.

No organisms were recovered from any of the environmental sources cultured, but *Acinetobacter* was recovered from patients and respirometers in the intensive care units. Further investigation revealed that the Department of Respiratory Therapy had removed the Bourns* respirometers for repairs and substituted 5 Wright† respirometers. Because the number of respirometers available as interim replacements was limited, the respirometers were shared by 4 intensive care units.

Control measures

The initial steps to control the outbreak included restricting the use of each Wright respirometer to a single intensive care unit thereby eliminating the inter-intensive care unit sharing of equipment. The importance of hand washing precautions was

* The Bourns LS 75 respirometer has no moving parts, and operates on the vortex principle. A flow sensor in the device detects vibrations in the airstream by an ultrasonic beam and, via a miniature computer, converts wave motion into volume flow rates.

† The Wright respirometer utilizes a small rotor placed in the airstream of the device. The small air turbine is calibrated so that its revs/sec correspond to volume flow rates.

emphasized to intensive care personnel, and strict enforcement of careful hand washing technique was observed. Because the cleaning of Wright respirometers with alcohol is technically not possible, it was decided to modify respirometer usage in an attempt to minimize the exposure of each patient to respirometers. This was accomplished by obtaining volume measurements with the respirometers placed at the most distal portion of the respirator tubing instead of interposing the respirometer between the patient and the endotracheal or tracheostomy tubing. This manoeuvre minimized the possibility of 'aerosolizing' the organism into the bronchioles of patients not previously colonized or infected by *Acinetobacter*.

Respirometer investigation

Laboratory tests were designed to simulate the high moisture content of the air exhaled by patients while on a respirator. Gas-sterilized Wright respirometers were exposed to sterile saline via a sterile nebulizer attached to the same type of volume respirator used in the intensive care units. Subsequently, sterilized Wright respirometers were exposed to a suspension of *Acinetobacter* via a sterile nebulizer attached to a volume respirator. Effluent air samples were obtained for culture using the Andersen air sampler.

Results

Patients

All patients colonized or infected had severe underlying disease requiring assisted ventilation in an intensive care unit. Because of the presence of unexplained fever, all patients received empiric antibiotic therapy before the diagnosis of *Acinetobacter* pneumonia. Two patients received cephazolin and 4 received both cephazolin and gentamicin for 1 to 4 days before the correct diagnosis was made. When the diagnosis of *Acinetobacter* pneumonia was established, patients with pneumonia were treated with either tobramycin, 80 mg i.v. every 8 hr, or minocycline, 150 mg intravenously every 12 hr, for 4 to 14 days based on antibiotic disc susceptibility tests.

Although no patient died directly as a result of *Acinetobacter* pneumonia it is likely that the organism contributed to the deaths of 3 patients. The remaining patients with pneumonia became afebrile after approximately 6 days (range 2–12 days) of antimicrobial therapy, and were discharged without *Acinetobacter* in the sputum. Sputum cultures of patients colonized by *Acinetobacter* eventually became negative after 6–18 days without antimicrobial therapy.

Microbiology

Sensitivity testing by disc diffusion technique

demonstrated uniform resistance of *Acinetobacter* to ampicillin, cephalosporins, gentamicin, and chloramphenicol. Some strains were sensitive to kanamycin or to tobramycin. The only antibiotics tested to which all the *Acinetobacter* strains tested were consistently sensitive were minocycline and co-trimoxazole. *Acinetobacter* recovered from the respirometers and colonized patients demonstrated the same sensitivity patterns recovered from infected patients except for considerable variation in sensitivity to kanamycin and tobramycin by disc testing. Throughout the outbreak, *Acinetobacter* was recovered only from sputum and the Wright respirometers. The common source nature of the outbreak is further supported by finding only one serotype (8J) involved in the entire outbreak.

Epidemiology

Recovery rate of the organism from sputum began to decrease, and no new cases occurred 6 days after the institution of control measures. At this time the repaired Bourns respirometers were returned, and the Wright respirometers were removed from service. An additional 18 days were required fully to eliminate the carriage of *Acinetobacter* from the sputum of the colonized patients. Retrospectively, the outbreak lasted 30 days from the introduction of the Wright respirometers to the elimination of respiratory carriage of the organism.

Respirometer investigation

Studies were conducted to determine if moisturized Wright respirometers, contaminated with *Acinetobacter*, could be responsible for the transmission of the organism by 'aerosolization'. Air samples obtained through Wright respirometers exposed to *Acinetobacter* as described in materials and methods, consistently revealed colonies of *Acinetobacter* too numerous to count. Air samples obtained through Wright respirometers exposed to sterile saline were either sterile or yielded air contaminants only.

Discussion

A large outbreak of respiratory tract infection and colonization due to *Acinetobacter* occurred in 4 intensive care units over a 30-day period.

Patients who developed *Acinetobacter* pneumonia were all pre-treated with antibiotics, had serious underlying diseases, and required assisted ventilation in an intensive care setting.

Acinetobacter pneumonia was characterized by leucocytosis, fever, purulent sputum, and diffuse lower lobe infiltrates without consolidation or effusion. Blood cultures were negative in all patients.

These findings are consistent with previously published reports indicating that bacteraemia is not a characteristic feature of the disease (Glew, Moellering and Kunz, 1977; Glick *et al.*, 1959). Appropriate antibiotic selection, based on sensitivity disc testing, included minocycline, tobramycin, and cotrimoxazole (Maderazo *et al.*, 1975). Patients with pneumonia were treated with either parenteral minocycline or tobramycin.

Studies performed during the outbreak demonstrate that moisturized Wright respirometers were capable of aerosolizing *Acinetobacter*. Although these studies did not involve patients the data suggest that this may explain the role of the respirometers in the patient-to-patient transmission of *Acinetobacter* in intensive care units where Wright respirometers are directly attached to the patient's endotracheal or tracheostomy tubing.

Control measures included rigorous hand washing techniques by intensive care personnel, restricting the use of individual Wright respirometers to only one intensive care unit, and the taking of volume measurements with the respirometer from the ventilator tubing instead of directly from the patient's endotracheal or tracheostomy tube. Since the elimination of the patients' colonization by the organism, and the re-introduction of the Bourns respirometers, the recovery of *Acinetobacter* from sputum has returned to pre-outbreak levels.

Serotyping confirmed the initial clinical and epidemiological impression that the Wright respirometers containing *Acinetobacter* were solely responsible for the outbreak.

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