INTRODUCTION

*Acinetobacter* spp has been considered to be an organism of low pathogenicity and rarely associated with invasive disease. However, during the last decade, several reports of nosocomial outbreaks, especially in intensive care units (ICU) have been described (Bergogne-Berezin and Towner, 1996). Relatively few studies of bacteremia due to *Acinetobacter* spp have been published (Siau *et al*., 1999; Wisplinghoff *et al*., 2000). We report an outbreak of bacteremia due to *Acinetobacter* spp from the neonatal intensive care unit. This is the first such large study reported from India, to the best of our knowledge.

MATERIALS AND METHODS

During a 6-month period (February to July 1997), an increased number of infections due to *Acinetobacter baumanii* were noted from the neonatal ICU of Lok Nayak Hospital, New Delhi, India. All strains of *Acinetobacter* spp were identified in the clinical microbiological laboratory using standard methods (Collee, 1996). Speciation was performed with the simplified identification scheme of Bouvet and Grimont (1987). Records of the previous 5 years revealed no similar seasonal or regional (ie ward-specific) clustering of *A. baumanii* infections. Bacteremia was considered to be clinically significant when it is associated with a clinical finding, such as fever and leukocytosis, of more than 8 hours' duration.

Antimicrobial susceptibility was determined by standard disc diffusion method to amoxicillin (10 µg), cefotaxime (30 µg), ceftazidime (30 µg), gentamicin (10 µg), netilmicin (30 µg), amikacin (30 µg), cephalexin (30 µg), piperacillin (75 µg) and ciprofloxacin (5 µg).

Environmental sampling of various inanimate surfaces in frequent contact with patients or healthcare workers was performed. The following sites were sampled: ventilator equipment of each patient, oxygen saturation monitor, bed rails, suction catheters, telephone handsets, soap dispensers, counter-tops, sinks, walls, etc.

RESULTS

The mean age of the neonates from which *Acinetobacter* spp was isolated was 8.2 days. The male:female ratio was 4:3. The minimum duration of hospitalization before acquisition of *Acinetobacter* spp septicemia was 5 days and the maximum was after the second week of hospitalization. Mortality was 27.7% (13/47) in this population.

A total of 68 strains of *A. baumanii* isolates
were recovered from the blood and CSF of 47 neonates admitted to the intensive care unit. Of these, 9 strains were isolated from the CSF of these patients. Bacteremia was considered to be clinically significant in 36 patients.

Although during the initial phase of the outbreak, most isolates were sensitive to all of the drugs tested, acquisition of resistance occurred rapidly. Overall, 68.8% of the isolates were multidrug-resistant resistant to 2 or more than 2 drugs. The highest resistance was seen against amoxicillin (87.6%) and netilmicin (49.7%). Ciprofloxacin and cefotaxime were the mainstays of treatment. However, with the emergence of resistance to these antimicrobials (18.8% and 26.1% respectively), other regimes had to be used empirically.

Isolates of *A. baumannii* with a similar antibiogram were recovered from 2 intravascular catheters and the washbasin. No other environmental/personnel sample was found to be positive.

**DISCUSSION**

In recent years, *Acinetobacter* spp, especially *A. baumannii*, has assumed importance as a nosocomial pathogen. The persistence of strains of *A. baumannii* in the ICU seems to be related to their ability for long-term survival on inanimate surfaces in the patient’s immediate environment and to their widespread resistance to the major antimicrobial agents (Wisplinghoff *et al.*, 2000; Koeleman *et al.*, 2001). The spread of these strains of *A. baumannii* has been related to contaminated respiratory therapy equipment, intravascular devices, the hands of medical and paramedical personnel, etc (Wisplinghoff *et al.*, 2000). In the present study, we describe an outbreak of bacteremia due to *A. baumannii* in a neonatal ICU. In accordance with other reports, we also isolated *Acinetobacter* spp of similar antibiogram from intravascular devices and a washbasin. We could not do genotypic typing because of the lack of facilities.

Although, during the initial phase of the outbreak, most isolates were sensitive to all of the drugs tested, acquisition of resistance occurred rapidly. This rapid increase in the antibiotic profile of *Acinetobacter* spp has been documented in other studies (Bergogne-Berezin and Towner, 1996). Thus it is an important cause of nosocomially acquired infection in immunocompromized hosts. In general, aminoglycosides, carbapenems and fluoroquinolones remain the mainstay of therapy. However there are reports of resistance to these agents, including imipenem (Heinemann *et al.*, 2000; Fierobe *et al.*, 2001; Koeleman *et al.*, 2001)

As found by other investigators (Wisplinghoff *et al.*, 2000), mortality of 27.7% of children with *A. baumannii* bacteremia was demonstrated in the present study. The present outbreak continued for 6 months and was controlled only after temporary closure of the unit for disinfection purposes. This outbreak proved to be one of the most difficult and costly challenges in infection control in our hospital. *A. baumannii* will undoubtedly continue to post problems in the future also, which is disturbing because of the extent of its ever-increasing antibiotic resistance profile. Thus, the isolation of multi-drug-resistant *A. baumannii* should alert the hospital infection control team and prompt implementation of strict infection prevention measures to prevent further spread.

**REFERENCES**


