Prevalence and Antimicrobial Drug Resistance of Acinetobacter baumannii Infection in a Tertiary Care Teaching Hospital of Rural Gujarat, India

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A B S T R A C T

Acinetobacter baumannii has emerged as a highly troublesome pathogen for many institutions globally. As a consequence of its immense ability to acquire or upregulate antibiotic drug resistance determinants, it has justifiably been propelled to the forefront of scientific attention. The clinical specimens over a period of 3 years (January 2014 to December 2016) were analyzed and the A. baumannii isolates obtained by an automated identification system (Vitek 2 Compact) were segregated for further study. Their antibiograms were studied and a clinical correlation was made to assess their pathogenic status and mode of acquisition. Further, the nosocomial infections acquired during this period were studied and the contribution made by A. baumannii was calculated to assess its nosocomial status. Prevalence of A. baumannii was 7.72% from the entire hospital and 75.22% in intensive care unit. Maximum isolates were from respiratory secretions (52.08%) followed by blood (17.9%). Of all isolates 27.78% proved to be pathogenic. A. baumannii contributed to 69.18% ventilator associated pneumonia, 16.94% Catheter Associated Blood Stream Infections, 15.82% Surgical Site Infections and 10.95% Catheter Associated Urinary Tract Infections. Overall resistance of A. baumannii towards carbapenems was 88.5% from all hospital isolates. ICU isolates showed higher resistance (95.04%) as compared to Inpatient Department (79.87%) and Out-patient Department (80.0%). In this study, 27.78% of A. baumannii isolates showed a pathogenic potential in with high rate of carbapenem resistance. We must be cognizant of the fact that all A. baumannii isolates doesn’t necessarily mean infection and antibiotics should only be given in clinically proven infections.

Keywords
A. baumannii, Prevalence, Resistance patterns, Clinical correlation, Nosocomial status.

Introduction

Members of the genus Acinetobacter have emerged from organisms of questionable pathogenicity to pan resistant nosocomial pathogens worldwide in the past two or three decades, especially since 2005-2006.¹ There are more than 30 genomic types of Acinetobacter identified so far, of which more than two third are due to Acinetobacter baumannii. A. baumannii colonizes healthy humans transiently at a low density on the warm and moist skin of axilla, groin, between toes, throat, nares and intestinal tract but it generally does not cause infection.²

In the hospital environment, A. baumannii can colonize the respiratory, urinary,
gastrointestinal tract and wounds of the patients and can cause infections in burn, trauma, mechanically ventilated and immunocompromised patients. It shows a special predilection for the ICU. The epidemiological, clinical, prognostic, and therapeutic characteristics of A. baumannii isolated from infected patients have been studied widely in the last decade. The most alarming problems encountered during this period are the organism’s ability to accumulate diverse mechanisms of resistance and the emergence of strains that are resistant to all commercially available antibiotics coupled with the lack of new antimicrobial agents in the pipeline. This has resulted in a limited choice of antibiotics for treatment of multidrug resistant isolates of A. baumannii, one of the most important therapeutic challenge. Acinetobacter baumannii being cause of human illness particularly in hospitalized patients and lack of information on the prevalent types responsible for infections in this area of Gujarat, prompted us to undertake this study.

Materials and Methods

This retrospective study was carried out in a tertiary care hospital over a period of 3 years from Jan 2014 to Dec 2016. Samples collected and processed during the course of routine diagnostic work up from patients in the ICU, wards and outpatient department (OPD) of the hospital for the identification of pathogens using routine microbiological techniques were analyzed and A. baumannii isolates were picked up for further studies.

The specimens studied were urine, respiratory samples (sputum, endo-tracheal aspirate and bronchoalveolar lavage), blood, pus and body fluids (pleural fluid, cerebrospinal fluid etc) Specimens were plated using appropriate culture media. Standard culture methods were used and the isolates were processed for identification and antibiotic sensitivity tests by the Vitek 2 Compact system (BioMe´rieux, Marcy l’Etoile, France), following CLSI guidelines.

The Acinetobacter isolates, thus identified were studied for their antibiotic sensitivity patterns in the Vitek 2 Compact. The antibiotics tested against the organism were Amikacin, Amoxyclave, Ampi-sulbactum, Cefotaxime, Gentamicin, Netilmicin, Nitrofurantoin, Norfloxacin, Tobramycin, Ceftazidime, Cefipime, Cefoparazole-sulbactam, Piperacillin-tazobactam, Ciprofloxacin, Levofloxacin, Imepenem, Meropenem, Colistin, Polymixin B and Tigecycline.

The role of A. baumannii as a pathogen or a colonizer in the respective infectious cases was determined by clinical correlation involving discussion with the clinicians to assess the pathogenic status of the isolate.

The role of A. baumannii in causing the nosocomial infections- Ventilator Associated Pneumonia (VAP), Catheter Associated Blood Stream Infections (CA-BSI), Surgical Site Infections (SSI) and Catheter Associated Urinary Tract Infections (CA-UTI) was evaluated. This was done by following the standard definitions of nosocomial infections according to CDC guidelines and analyzing the role of A. baumannii in the causation of hospital acquired infections.

Results and Discussion

Of the total cultures processed (8669), A. baumannii constituted 23.1% of the total Gram negative load (670 out of 2900). This included the maximum isolates from respiratory secretions (364 out of 670) (54.3%) followed by 125 in blood (18.7%), 95 in pus (14.3%) and 49 urine specimens (7.4%) as shown in Figure 1.
Of the 670 isolates of *A. baumannii* from the entire hospital, 504 belonged to the ICU (75.22%). The inpatient department (IPD) and the outpatient department (OPD) contributed to 22.24% (149 out of 670) and 1.94% (13 out of 670) of the total *A. baumannii* isolates respectively. In the ICU isolates, similar to the entire hospital isolates, respiratory samples showed a maximum yield of *A. baumannii*, 345 out of 504 (68.45%) followed by 94 out of 504 isolates (18.65%) from blood, 35 out of 504 isolates (6.94%) from pus, and 30 from urine (5.95%).

To assess whether *A. baumannii* was actually causing clinical infection or was an innocent bystander, a clinical correlation was done in the 504 isolates in the ICU. Of these 122 proved to be pathogenic (24.20%), 382 (75.8%) appeared to be colonizers as shown in (Table 1).

Of the 94 *A. baumannii* isolates from the blood, 7 (6.58%) were proven for their pathogenic status of the samples isolated from pus and drain fluid (35 isolates), 3 (8.1%) isolates were proven as pathogens and rest of the 32 (91.9%) were skin colonizers.

Of the 30 isolates from urine 6 (20.00%) isolates were proven as pathogens.

*A. baumannii* contributed to 61.62% of VAP (119 out of 172 cases), 16.94% of CA-BSI (10 out of 59 cases), 15.82% of SSI (44 out of 278 cases) and 10.95% of CA-UTI (8 out of 73 cases).

The resistance patterns of the *A. baumannii* isolates towards carbapenems was studied for ICU, IPD and OPD patients separately (Table 2). Resistance rates in various locations ranged from 86.6% in OPD to 100% in ICU.

*A. baumannii* was isolated in 23.10% of the total Gram-negative isolates. This corresponds to similar study carried out by Sameera *et al.*, where *A. baumannii* isolates were 31.7% of the total Gram negative isolates.

**Table 1** Distribution of *A. baumannii* isolates based on the type of specimen in the ICU isolates (n=504)

| Samples               | Total No. of Isolates | Pathogenic | Non-Pathogenic | % Pathogenic |
|-----------------------|-----------------------|------------|----------------|--------------|
| Respiratory Secretions| 345                   | 106(VAP)²  | 239            | 30.73        |
| Blood                 | 94                    | 7(CLABSI)¹ | 87             | 6.58         |
| Pus                   | 35                    | 3(SSI)²   | 32             | 8.1          |
| Urine                 | 30                    | 6(CAUTI)¹ | 24             | 20.00        |

a Ventilator Associated Pneumonia, b Catheter Associated Bloodstream Infections, c Surgical Site Infections, d Catheter Associated Urinary Tract Infections

**Table 2** *A. baumannii* resistance to carbapenems in different areas of hospital (n=504)

| Sample type | ICU | IPD | OPD |
|-------------|-----|-----|-----|
|             | CR² | CR² | CR² |
| Res.²       | 345 | 323 (93.17%) | 45 | 31 (68.88%) | -- |
| Blood       | 94  | 91 (96.8%) | 29 | 23 (79.31%) | 1 |
| Pus         | 35  | 34 (97.14%) | 46 | 44 (95.65%) | 15 | 13 (86.66%) |
| Urine       | 30  | 28 (93.33%) | 17 | 10 (58.82%) | 2 | 2 (100%) |

a Respiratory Secretions, b Carbapenem Resistance
We isolated *A. baumannii* most commonly from respiratory secretions (41.3%), similar to findings by Ashu et al.,\(^9\) where 59% isolates were from respiratory secretions. Of the total 670 isolates of *A. baumannii*, a maximum relative percentage (53.28%) was obtained in the respiratory secretions. Sudhaharan S et al.,\(^10\) have also reported a predominance of *A. baumannii* in tracheo-bronchial secretions as 40% and Anitha et al.,\(^11\) as 43% respectively in their studies.

The proportion of *A. baumannii* isolates was higher in the ICU (75.22%) as compared to the IPD (22.24%) and OPD (1.94%) pointing towards *A. baumannii* being a predominantly ICU bug. This result corroborates the fact that a lot of risk factors associated with *Acinetobacter* infection exist in the ICU like potential environmental reservoirs, opportunities for cross transmission, and highly susceptible patient population.

We made an attempt to distinguish clinical infection from colonization. In ICU, 24.2% isolates proved to be pathogenic. When pathogenic potential from different sample was analysed, 30.73% were recognized as pathogens in respiratory secretions, Literature also reports 43% isolation from tracheo-bronchial secretions.\(^10\)

From blood only 10.83% of *A. baumannii* were found to be pathogenic. We identified 89.17% as the contaminants where-as, Lahiri et al.,\(^12\) have reported only 33% of *A. baumannii* isolates from blood as skin contaminants.

Pus and fluids analysis showed 15.82% of *A. baumannii* as pathogens. Sengupta et al.,\(^13\) reported a lower isolation rate of 11.5% of *A. baumannii* from wounds. High isolation rate in our hospital could be because of a smaller sample subset of pus and body fluid samples or more infected patients coming into a tertiary care center.

*A. baumannii* emerged as a predominant pathogen in VAPs (61.62%) in our study, similar to 59% by Seyed et al.,\(^14\). We had 16.94% of CA-BSI caused by *A. baumannii* while Zakuan et al., reported it to be 6.1% only.\(^15\) *A. baumannii* caused about 15.82% of the SSIs in the hospital while Jones et al.,\(^16\) have reported it to be 11.7%.
The overall carbapenem resistance in our study was 88.5% which was 79.86% for IPD patients and 93.6% for ICU. Resistant to tigecycline in our study was 66.13%, similar to 66% by Navon et al.,20 and 58% by Bijayini Behara et al.,21. Colistin/Polymyxin is one agent which is active against A. baumannii and resistance rates are still low, 1.32% in ours and 3.3% from the Western Pacific region.19

Antibiotic resistance in A. baumannii is increasing at an alarming rate leading to increased morbidity, mortality and treatment costs in ICU settings as revealed by surveillance studies from various countries, over the last 3-5 years.17 Earlier studies18 in India had reported lower resistance rates (9.8-18.5%) and increasing resistance in A. baumannii towards carbapenems is a critical finding in our study.

Thus, Acinetobacter baumannii has evolved as a prominent human pathogen, more so in critical area and with high rates of drug resistance. A judicious use of antibiotic with an attempt to distinguish colonizers from pathogens is necessary for better patient outcome.

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