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ORIGINAL ARTICLE

Acinetobacter baumannii isolates in a tertiary care hospital: Antimicrobial resistance and clinical significance

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ABSTRACT

Objectives: To carry out a retrospective study on *Acinetobacter baumannii* isolates from various clinical samples in a tertiary care hospital in India and analyze its epidemiology, antibiotic susceptibility patterns, pathogenic potential and nosocomial status.

Materials and methods: The clinical specimens over a period of 14 months from December 2008 to January 2010 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.

Results: *A.baumannii* was isolated in 155 samples out of 1632 gram negative isolates (9.4% prevalence) from the entire hospital. Maximum isolates were from respiratory secretions (57.4%) followed by blood (23.8%). Prevalence of *A.baumannii* rose to 22.7% (119 out of 525) in intensive care unit (ICU) and 65 isolates (54.6%) proved to be pathogenic. *A.baumannii* contributed to 30.4% ventilator associated pneumonia, 35.2% Catheter Associated Blood Stream Infections, 12.5% Surgical Site Infections and 2.94% Catheter Associated Urinary Tract Infections. Overall resistance of *A.baumannii* towards carbapenems was 90% from all hospital isolates. ICU isolates showed higher resistance (93.2%) as compared to Inpatient Department (82.7%) and Out-patient Department (57.1%).

Conclusion: In this study, *A.baumannii* isolates showed a pathogenic potential of around 54.6% and a majority were found to be carbapenem resistant. We must be cognizant of the fact that all *A.baumannii* isolations doesn't necessarily mean infection and antibiotics should only be given in clinically proven infections. *J Microbiol Infect Dis 2012; 2(2): 57-63*

Key words: *A.baumannii*; prevalence; resistance patterns; clinical correlation; nosocomial status

Üçüncü basamak bir hastanede Acinetobacter baumannii izolatları: Antimikrobiyal direnç ve klinik önemi

ÖZET

Amaç: Retrospektif bir çalışma ile değişik klinik örneklerden izole edilen *Acinetobacter baumannii* suşlarının değerlendirmesi yapılarak epidemiyolojileri, antibiyotik duyarlılık paternleri, patojen potansiyelleri ve hastane kaynaklı oluşları analiz edildi.

Gereç ve yöntem: Aralık 2008'den Ocak 2010'a kadar 14 ay süreyle klinik örnekler incelendi ve otomatik tanı sisteminin (Vitek 2 Compact) tanımladığı *A.baumannii* suşları alınarak ileri çalışmalar için ayrıştırıldı. Bu suşların antibiyogramları çalışıldı, patojenlik durumlarına alındıkları kaynaklarla klini uyumları karşılaştırılarak karar verildi. Ayrıca bu dönemde nozokomiyal enfeksiyonları hesaplanarak *A.baumannii*'nin bunlara katkısı değerlendirildi.

Bulgular: Toplam olarak izole edilen 1632 gram negatif suşun 155'i (% 9,4) *A.baumannii* idi. En fazla suş solunum salgılarından (% 57,4) ve kandan (% 54,6) elde edildi. *A.baumannii* prevalansı yoğun bakım ünitesinde (YBÜ) % 22,7'ye çıkarken (525'de 119) bunların 65'i (% 54,6) patojen olarak teyit edildi. *A.baumannii* ventilator ilişkili pnömonilerin % 30,4'üne, kateter ilişkili kan dolaşımı enfeksiyonlarının % 35,2'sine, cerrahi alan enfeksiyonlarının % 12,5'ine ve kateter ilişkili üriner sistem enfeksiyonlarının % 2,94'ünde etken olarak bulundu. Tüm hastane izolatlarında toplam olarak *A.baumannii* suşlarının karbapenemlere direnci % 90 idi. YBÜ suşları karbapenemlere karşı Yatan Hasta Departmanı suşlarına ve Ayaktan Takip Departmanı suşlarına göre daha yüksek bir direnç göstermekteydiler (sırasıyla, % 93,2, % 82,7 ve % 57,1).

Sonuç: Bu çalışmada *A.baumannii* suşları % 54,6 civarında bir patojen potansiyeli gösterdiler ve ekseriyeti karbapenemlere dirençli idi. Tüm *A.baumannii* izolasyonlarının enfeksiyon anlamına gelmediği ve antibiyotiklerin sadece klinik olarak teyit edlien enfeksiyonlarda kullanılması gerektiği gerçeğinin farkında olmalıyız.

Anahtar kelimeler: *A.baumannii*, prevalans, direnç paternleri, klinik uyum, nozokomiyal

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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 of Acinetobacter infections 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.[3] The epidemiological, clinical, prognostic, and therapeutic characteristics of *A.baumannii* isolated from infected patients have been studied widely in the last decade.[4] 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.[5] This has resulted in a limited choice of antibiotics for treatment of multidrug resistant isolates of *A.baumannii*. The most active agents in vitro against the multidrug resistant *A.baumannii* are the polymixins- polymixin B and polymixin E (Colistin) and tigecycline.[6]

In our study undertaken over a period of 14 months (Dec 2008-Jan 2010) at a tertiary care super speciality hospital, we report the prevalence of *A.baumannii* isolates, their antibiograms and their clinical significance. Their contribution as causative agents of nosocomial infections has also been evaluated.

MATERIALS AND METHODS

This retrospective study was carried out in a 215 bedded tertiary care hospital over a period of 14 months from Dec 2008 to Jan 2010. 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, body fluids (pleural fluid, cerebrospinal fluid etc) and stool. Specimens were plated using appropriate culture media (Mac-Conkey agar, Blood agar, Chocolate agar and Cysteine Lactose Electrolyte Deficient (CLED) agar and Xylose Lysine Deoxycholate agar). Standard culture methods were used and the isolates, both Gram positive and Gram negative were processed for identification and antibiotic sensitivity tests by the Vitek 2 Compact system (BioMe´rieux, Marcy l'Etoile, France), following CLSI guidelines.[7]

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, gentamicin, netilmycin, tobramycin, ceftazidime, cefipime, cefoparazone-sulbactam, piperacillin-tazobactam, ciprofloxacin, levofloxacin, imepenem, meropenem, colistin, polymixin B and tigecycline. The susceptibility results were compiled with the WHONET 5.4 programme.

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 [8] and analyzing the role of *A.baumannii* in the causation of hospital acquired infections.

RESULTS

The culture samples from the entire hospital processed during the 14 months study period from Dec 2008 to Jan 2010 were 10079. Of the total cultures processed, the number of pathogenic bacterial isolates was1961 (19.4%) which

constituted 1632 (83.2%) gram negatives and 329 (16.7%) gram positive organisms. Overall, *A.baumannii* isolates constituted 9.4% of the total gram negative load (155 out of 1632). This included the maximum number of *A.baumannii* isolates from respiratory secretions (89 out of 155) (57.4%) followed by 37 in blood (23.8%), 21 in pus (13.5%) and 4 each in fluids and urine specimens (2.5%). The carbapenem resistance in these *A.baumannii* isolates was seen maximum in respiratory isolates (93.2%) as compared to non-respiratory isolates.

Of the 155 isolates of *A.baumannii* from the entire hospital, 119 belonged to the ICU (76.7%). The ICU also had a higher relative proportion of *A.baumannii* isolates among gram negative isolates as 22.6% (119 out of 525) as compared to overall 9.4% of *A.baumannii* in the hospital among gram negative isolates (155 out of 1632). The inpatient department (IPD) and the outpatient department (OPD) contributed to 18.7% (29 out of 155) and 4.5% (7 out of 155) 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*, 71 out of 119 (56.9%) followed by 30 out of 119 isolates (25.2%) from blood,12 out of 119 isolates (10%) from pus, 4 isolates from fluids (3.36%) and 2 from urine (1.68%).

To assess whether *A.baumannii* was actually causing clinical infection or was an innocent bystander, a clinical correlation was done in the 119 isolates of *A.baumannii* in the ICU. Of the total 119 isolates of *A.baumannii* from the ICU, 65 proved to be pathogenic (54.6%) as shown in Table 1. Of the 71 isolates from respiratory samples in the ICU, 46 (64.7%) appeared to be colonizers without contributing to the signs and symptoms of infection and only 25 (35.2%) contributed to the infection. 21 out of 71 (29.5%) were clinically proven ventilator associated pneumonia (VAP) cases and 4 were admitted with community acquired infections. Of these 21 isolates, 7 (33.3%) were proven as having been acquired from our tertiary care hospital and 14 (66.6%) were brought in at the time of admission from other hospitals.

Of the 30 *A.baumannii* isolates from the blood, 26 (86.6%) were proven for their pathogenic status and in the remaining 4 patients who showed no symptoms of blood stream infection (BSI), the culture positivity may have been due to contamination with *A.baumannii* colonized on skin during sample collection. Of the 26 isolates, 11 (42.3%) were responsible for nosocomial CA-BSIs, 6 of these were acquired from the tertiary care centre and 5 patients came in with an infected line from another hospital. The other 15 (57.6%) infections caused by *A.baumannii* were secondary to the primary infection elsewhere.

Of the samples isolated from pus and drain fluid (16 isolates), 12 (75%) isolates were proven as pathogens and rest of the 4 (25%) were skin colonizers. Out of the 12 pathogens, 4 were attributed to having been hospital acquired from our tertiary care centre, and 8 were brought in from the community or other hospitals.

Of the 2 isolates from urine, both were found pathogenic and 1 was observed as acquired from our tertiary care hospital and the other was community acquired.

When this data was compared to the total data of hospital acquired infections at our tertiary care hospital in the fourteen month period, it was seen that *A.baumannii* contributed 30.4% of VAP (7 out of 23 cases), 35.2% of CA-BSI (6 out of 17 cases), 12.5% of SSI (4 out of 32 cases) and 2.9% of CA-UTI (1 out of 34 cases).

The resistance patterns of the *A.baumannii* isolates towards carbapenems was studied (Table 2) for ICU, IPD and OPD patients separately and it was found that out of the total of 155 isolates 139 were carbapenem resistant indicating a high resistance rate of 89.6%. In the ICU, the overall resistance rate was 93.2% where 100% resistance was found in urine and fluids and 94.3%, 91.6% and 90% in respiratory, pus and blood samples respectively. The IPD *A.baumannii* isolates showed overall resistance of 82.7% and 88.8% were found carbapenem resistant from the respiratory specimens. *A.baumannii* isolated from OPD specimens (represented the prevalence in community) showed a comparatively lower resistance of 57.1% and the highest carbapenem resistance was obtained in isolates from pus as 80%.

The antibiotic susceptibility patterns of *A.baumannii* isolates and other gram negative isolates were also studied as given in Table 3.

Table 1. Distribution of A baumannii isolates based on the mode of acquisition of infection from Dec 2008 to Jan 2010 (14 month) in the ICU isolates (Total No=119).

Samples	Total No. of isolates	Our Hospital Acquired	Other Hospital Acquired	Community Acquired	Colonizers/ Contaminants	Secondary Infection
Respiratory secretions	71	7 VAP[a]	14	4	46 (Colonizers)	-
Blood	30	6 CA-BSI[b]	5	-	4 (Contaminants)	15 BSI[c]
Pus & fluids*	16	4 SSI[d]	2	6	4 (Contaminants)	-
Urine	2	1 CA-UTI[e]	-	1	-	-

[a] Ventilator Associated Pneumonia, [b] Catheter Associated Bloodstream Infections, [c] Bloodstream Infections

[d] Surgical Site Infections, [e] Catheter Associated Urinary Tract Infections, * Pleural fluid, cerebrospinal fluid, drain fluids etc

Table 2. *A.baumannii* resistance to Carbapenems in different areas of hospital from Dec 2008 to Jan 2010

Sample Type	*A.baumannii*		ICU		IPD		OPD	
	No. of isolates	C R**	No. of isolates	C R**	No. of isolates	C R**	No. of isolates	C R**
Urine	4	3	2	2	1	1	1	0
Blood	37	31	30	27	6	4	1	0
Resp*	89	83, 93.2%	71	67, 94.3%	18	16, 88.8%	0	0
Pus	21	18	12	11	4	3	5	4, 80%
Fluids	4	4	4	4	0	0	0	0
Total	155	139, 89.6%	119	111, 93.2%	29	24, 82.7%	7	4, 57.1%

* Respiratory secretions, ** Carbapenem resistant isolates

Table 3. Antimicrobial susceptibility of *A.baumanni* and other gram negative Isolates obtained from infected patients

Antimicrobial agent	Break-points (µg/ml)	Percent Resistant	
		A.baumannii n=155	Other GNB n=1477
Amikacin	S<=16 R>=64	90.3	35.6
Gentamicin	S<=4 R>=16	85.8	55.9
Netilmycin	S<=16 R>=64	90.3	33.7
Tobramycin	S<=4 R>=16	80	62
Aztreonem	S<=8 R>=32	94.2	64.3
Cefipime	S<=8 R>=32	90.3	62.7
Ceftazidime	S<=8 R>=32	92.1	61.5
Cefaperazone-sulbactum	S<=16 R>=64	92.3	33.3
Piperacillin-Tazobactam	S<=16 R>=128	89.7	28.9
Ciprofloxacin	S<=1 R>=4	91.6	67.4
Levofloxacin	S<=2 R>=8	87.6	67.1
Imepenem	S<=4 R>=16	89.6	18.5
Meropenem	S<=4 R>=16	89.6	19.2
Tigecycline	S<=2 R>=8	74.8	6.7
Colistin	S<=2 R>=8	1.2	6.1
Polymixin B	S<=2 R>=4	1.9	5.7

DISCUSSION

A.baumannii was isolated in 155 samples forming 9.4% of the total gram-negatives. This corresponds to similar study carried out by H Siau et al[9] where figures of *A.baumannii* isolates were 11% of the total gram negative isolates.

Among the gram-negative isolates from the respiratory secretions *A.baumannii* was the most common (18%). Studies similar to this, were carried out by Pedersen et al[10] where the maximum isolates were obtained from sputum as 26.3%. Since Acinetobacter is a common commensal in the respiratory tract, it may be isolated without necessarily causing infection. Of the total 155 isolates of *A.baumannii*, a maximum relative percentage (57.4%) was obtained in the respiratory secretions. Villers et al[11] have also reported a predominance of *A.baumannii* in tracheo-bronchial secretions as 24.8% to 48.8% and Suri et al[12] as 45.6% respectively in their studies. The ICU also showed the maximum yield of *A.baumannii* from the respiratory samples (59.6%) followed by blood (25.2%). Siau et al[9] reported in their ICU isolates that respiratory tract was the most common site from which Acinetobacter was isolated.

The proportion of gram negatives and gram positives in ICU was similar to the entire hospital data but the proportion of *A.baumannii* isolates was higher in the ICU (76.7%) as compared to the IPD (18.7%) and OPD (4.5%) 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 for *A.baumannii*, opportunities for cross transmission, sick, immunocompromised patients who are colonized, patients having multiple wounds and indwelling devices, heavy use of broad spectrum antibiotics and frequent contamination of the hands of health care workers while patient care.

In the community, *A.baumannii* has been found to be associated with community acquired pneumonia, (in patients with COPD, renal failure, diabetes mellitus, heavy smokers or excessive alcohol consumers) or bacteremia in Australia and Asia, although rare in USA as evidenced by previous studies.[13] This supports our results of isolation of *A.baumannii* from the outpatient department (4.5%).

An attempt was made in this study to distinguish clinical infection from colonization. Of the total 119 isolates of *A.baumannii* from the ICU, 65 proved to be pathogenic (54.6%). This was done by correlating various clinical and lab parameters and discussion with the clinician. An analysis was also done of the pathogenic potential of *A.baumannii* in various samples like respiratory secretions, blood, pus and body fluids and urine specimens. Of the 71 isolates of *A.baumannii* from respiratory secretions only 32.5% were recognized as pathogens and rest were colonizers. Other studies in the world have reported a similar isolation from tracheo-bronchial secretions (24.8%-48.8%).[11] Since this organism is a fast colonizer of the respiratory tract, its percentage can increase from 7% to 45% in healthy subjects to those on ventilator respectively.[14] Of the *A.baumannii* isolates from blood 86.6% were found to be pathogenic and rest were the contaminants. As Acinetobacter cannot exist as a colonizer in blood, it would have a higher pathogenic potential at this site. We did isolate *A.baumannii* as contaminants in 13.3% cases. However, Lahiri et al[15] have reported 33% of *A.baumannii* isolates from blood as skin contaminants. Pus and fluids analysis showed 75% of *A.baumannii* as pathogens. Sengupta et al[16] 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.

Of the two isolates from urine, both were found pathogenic and 1 was observed as acquired from our tertiary care hospital and the other was community acquired.

To see the relevance of *A.baumannii* in the causation of hospital acquired infections all the hospital acquired infections in this period were analyzed. *A.baumannii* emerged as a predominant pathogen in contributing to hospital acquired VAPs (30.4%) in our study. Similar incidents of *A.baumannii* VAP have been reported by Torres et al[17] as 39%. A similar situation was observed with CA-BSI where it contributed to 35.2% of BSI. Other studies have reported a prevalence of 6.1% (Zakuan et al).[18] Our hospital had slightly higher prevalence which has been attributed to the lack of implementation of the prevention bundle for CA-BSIs till that time. *A.baumannii* caused about 12.5% of the SSIs in the hospital in this

period. Other studies by Jones et al[19] have shown similar results (11.7%). In our study, *A.baumannii* showed relatively low prevalence in causing CA-UTI (2.9%).

The antibiograms of the isolates of *A.baumannii* from the entire hospital showed 89.6% carbapenem resistance in our study. When the isolates in the ICU were studied, the resistance to carbapenems rose to 93.2% whereas in IPD it was 82.7%. However, a comparatively low carbapenem resistance of 57.1% was observed in *A.baumannii* in the community (as represented by the OPD isolates). 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 Europe, the Asia Pacific region, Latin America and North America over the last 3-5 years.[20] However, lower rates of carbapenem resistance have been reported in studies carried out by Knam Soo Koo et al[20] as 8.3%. This could be explained by their stringent antibiotic policies and judicious use of carbapenems in their countries.

Earlier studies[21] in India have also reported lower resistance rates (9.8-18.5%) in *A.baumannii*. This clearly explains that our study brings up an important aspect of increasing resistance in *A.baumannii* towards carbapenems.

The antibiotic susceptibility patterns clearly showed the increasing resistance of *A.baumannii* to various antibiotics as compared to other gram negatives. Colistin (Polymixin E) is one agent which is active against *A.baumannii*. In the present study, colistin resistance has been reported as 1.2%. A recent study of clinical isolates from the Western Pacific region showed 3.3% resistance of *A.baumannii* to colistin.[22] Heteroresistance to colistin among *A.baumannii* isolates has also been described in earlier reports.[20] In a study in Korea [20], there was high resistance to colistin (30.6%) and polymixin (18.1%). However, as the resistance against colistin/polymyxin is not very high in our country, it can still be used as the drug of choice against multidrug resistant strains of *A.baumannii*.

A.baumannii was resistant to tigecycline in 74.8% cases in present study. This corresponds to other studies by Navon et al[23] where 66% of Acinetobacter were resistant to tigecycline. Studies by Bijayini Behara et al[24] in India have also shown only 42% susceptibility in A.baumannii isolates to tigecycline respectively. However issues about the breakpoints for susceptibility in the disk diffusion tests and standardized guidelines for in vitro testing of tigecycline need to be further evaluated.

In our study, antibiotics were prescribed for A.baumannii infection in the ICU for 119 patients, which was nearly twice the number of those with proven infection. The antibiotic overuse reflected this propensity to treat *A.baumannii* infections based on bacteriological reports alone and not the patient in entirety. The resistance patterns detected in Acinetobacter could reflect the antibiotic misuse and lack of regulations on the over the counter sale in some parts of the World. Our study suggested that due to the increasing resistance of *A.baumannii*, we should judiciously use antibiotics by making an attempt to distinguish colonization from infections and treatment should be only given to the clinically confirmed Acinetobacter infections and not merely colonization.

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