Prevalence of Multi Drug Resistant Non-fermenter Acinetobacter baumannii and Pseudomonas aeruginosa in Narayana Medical College and Hospital, Nellore, AP, India

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A B S T R A C T

The severity and extent of disease caused by multidrug-resistant organisms (MDROs) varies by the population(s) affected and the institution(s) at which these organisms are found; therefore, preventing and controlling MDROs are extremely important. A retrospective study of patients who were infected with Acinetobacter baumannii or Pseudomonas aeruginosa was performed at Narayana Medical College & Hospital from 2015 to 2016. A total of 88 A. baumannii isolates and 215 P. aeruginosa isolates were identified during the period. Imipenem 73 (88%) and colistin 62 (88%) were the most active agents against A. baumannii. Most MDR isolates were resistant to more than three classes of antibiotics. P. aeruginosa was recovered more frequently from the pus, followed by the soft tissue, urine and blood. Piperacillin/tazobactam 76 (81.86%), doripenem 172 (80%) imipenem 170 (79%) were active against P. aeruginosa isolates. In summary, A. baumannii was more rare than P. aeruginosa but was more commonly MDR. Epidemiological data will help to implement better infection control strategies, and developing a local antibiogram database will improve the knowledge of antimicrobial resistance patterns in our region.

Keywords
Acinetobacter baumannii, Pseudomonas aeruginosa, Infection, Antibiotics, Antibiotic resistance

Introduction

Nosocomial infections are one of the most common complications of hospitalization and lead to increased morbidity and mortality (Geffers et al., 2008; Aranaz-Andres et al., 2008).

These infections prolong hospitalization, require more extensive diagnostics and treatment and are associated with additional costs (Pittet et al., 1994; Beyersmann et al., 2006). Infection with multidrug-resistant pathogens can also complicate treatment.

Antibiotic resistance is a daunting phenomenon with a growing impact on patient safety, particularly in ICUs (Bonten, 2011). Critically ill patients are prone to colonization and infection by antibiotic-resistant Bacteria because of the frequent exposure of these patients to antibiotics and the presence of multiple, often invasive, devices. This dangerous array of risk factors drives a vicious
cycle of increased Infection incidence, increased need for broad-spectrum antibiotics, reduced antimicrobial efficacy and increased selection of antibiotic resistance.

Multidrug-resistant organisms (MDROs) are resistant to one or more classes of antimicrobial agents, such as β-lactams (penicillins, cephalosporins, monobactams and carbapenems), fluoroquinolones and aminoglycosides. During the past several decades, a shift in the MDR dilemma from gram-positive to gram-negative bacteria has been noted, which is in part due to the small number of new antimicrobial agents that are active against resistant gram-negative strains (Boucher et al., 2009). Gram-negative pathogens that have acquired epidemiological importance among nosocomial infections include Acinetobacter baumannii and Pseudomonas aeruginosa.

A. baumannii is a cause of outbreaks in hospitals (Morgan et al., 2009; Pournaras et al., 2006), and the MDR patterns observed among isolates often leave carbapenems as the only effective treatment for severe infections (Pournaras et al., 2006). However, carbapenem-resistant A. baumannii is emerging worldwide and has been observed in different countries (Morgan et al., 2009; Livermore et al., 2010; Peleg et al., 2008; Ying et al., 2006). There are limited therapeutic options for infections caused by these isolates. P. aeruginosa is also a common gram-negative nosocomial pathogen. This organism is an important cause of hospital-acquired pneumonia and urinary tract, wound and bloodstream infections (Walkty et al., 2008). Infections caused by this pathogen are often difficult to treat because of the multidrug-resistant nature of this bacterial species, and P. aeruginosa strains are often carbapenem resistant, which can severely limit the available therapeutic choices (Scheffer et al., 2010).

The purposes of this study were the following:

To determine the prevalence of A. baumannii and P. aeruginosa in patients with nosocomial infections at NMC & Hospital Nellore

To analyze the antimicrobial susceptibility patterns of these two microorganisms determined as part of an internal laboratory surveillance study from 2015 to 2016.

**Materials and Methods**

**Bacterial isolates**

A retrospective study of all A. baumannii and P. aeruginosa isolates from different clinical specimens collected from patients with nosocomial infections and processed by the microbiology laboratory between 2015 and 2016 was conducted at the NMC & Hospital Nellore, major hospital approximately 20,000 clinical specimens are received in medical microbiology laboratory per year. Infections were considered nosocomial if they first appeared 48 h after admission. Infections that were likely to have been acquired before hospital admission were not considered nosocomial. Blood, urine, tracheal aspirate, bal-broncho alveolar lavage, sputum, purulent wound, skin ulcer and catheter tip samples collected from patients admitted to all units. Duplicate isolates were excluded.

The study was carried out in the central laboratory of Microbiology Narayana Medical College Nellore South India from August 2015 to September 2016. Relevant clinical specimens sputum, blood, pus, urine, were collected from patients by standard collection procedures. No specific exclusion criteria envisaged. Specimens were processed by standard microbiological techniques. (Collee et al., 1999). In Gram stain of direct smears Acinetobacter appeared as tiny, Gram-negative coccobacillary cells often appearing
as diplococci. (Koneman et al., 2006) All specimens were inoculated on 10% sheep blood agar and MacConkey agar and incubated at 37°C for 18-24 h. (Collee et al., 1999) Colonies on blood agar were 0.5-2 mm diameter, translucent to opaque (never pigmented), convex and entire. On MacConkey agar a faint pink tint was produced. (Koneman et al., 2006) Gram stain, catalase, oxidase and motility tests were performed. Acinetobacter are Gram-negative Coccobacilli, non-motile, strictly aerobic, catalase positive and oxidase negative. Rapid utilization of 10% glucose was seen with O-F medium. Acinetobacter baumannii identification done. Antimicrobial susceptibility testing (Collee et al., 1999) was performed by modified Kirby Bauer method (Bauer et al., 1966) as per the Clinical and Laboratory Standards Institute guidelines. (Wayne, 2008) Antibiotics tested were ampicillin, cephoxime, cefixime, Co-Trimoxazole, ciprofloxacin, Ofloxacin, gentamicin, amikacin, tigecycline, amoxycillin with clavulanic acid, cefoperazone with sulfactam, ticaricillin with clavulanic acid, piperacillin with tazobactam. Zone of inhibition diameter was measured using calibrated ruler and interpreted as susceptible, intermediate or resistant in accordance to CSLT guidelines. Multidrug resistance is defined as isolates resistance to more than three classes of drug.

**Results and Discussion**

In total, 88 Acinetobacter baumannii strains were isolated. Out of these 88Acinetobacter isolates, 36 isolates were from general wards and 52 were from ICU. Significantly higher percentage of Acinetobacter strains were found in ICU 56(59.095) compared with general ward 36(40.90%). The most common Acinetobacter isolates are from blood 45 (51.14%) followed by pus 18(20.15%), urine 15(17.05%) and sputum (11.36%). Table 1. Imipenem was most sensitive drug 73 (82.95%) followed by colistin 62(70.45%), Tigecycline 59(67.05%), ciprofloxacin 55(62.50%), ofloxacin 54(61.36%), amikacin 53(60.23%) gentamycin 52(59.09%). highest resistance is seen in ampicillin. 70(79.35%) followed by cefixime 66(75.00%), ceftriaxone 62(70.45%) amoxicillin + clavulanic acid (61(69.23%), cephoxime 57(64.77%), ticaricillin + clavulanic acid 54(61.35%) co-trimoxazole 49(55.68%), piperacillin + tazobactam 40(45.45%).

Acinetobacter baumannii. is Gram-negative Coccobacilli that contribute profoundly to the burden of modern medicine. Acinetobacter spp. is the second most commonly isolated non-fermenter in human specimens (after Pseudomonas aeruginosa). They rank fourth (after P. aeruginosa, Staphylococcus aureus and Klebsiella pneumoniae) among the most frequent hospital acquired infectious agents. (Shete et al., 2009) Acinetobacter spp. have emerged as a cause of ICUs infection. Multiresistant Acinetobacter spp. have become established as “alert” pathogens, particularly in ICUs and are associated with outbreaks of infection (Agodi et al., 2006). Their ubiquitous nature in the ICU environment and inadequate infection control practice have continuously raised the incidence of Acinetobacter infections over the past two decades. The understanding and recognition of Acinetobacter infections in the ICU is critically needed (Rungruanghiranya et al., 2005).

In our study, a total number 88 Acinetobacter strains were isolated from processed clinical specimens. (Houang et al., 2001) reported a total of 1.32%. Patients in ICU are sicker and require more invasive monitoring and therapeutic procedures to survive. ICU environmental contamination appears to be another important source of Acinetobacter infection. (Rungruanghiranya et al., 2005) The
development of ICU-acquired infections is strongly related to prolonged ICU stay and is associated with worse outcomes including increased morbidity and mortality. (Falagas et al., 2008) Our study isolated acinetobacter from blood 45-(51.14%) followed by wound infections (pus18.-(20, 25%) pneumonia(11.36%) urinary tract infections (urine15-17.05%). (Joshi et al., 2006) reported that 27.5 wound infections were caused by Acinetobacter. Acinetobacter ICU-acquired infections during the last decade represent a growing concern among clinicians and researchers. These infections most frequently involve the respiratory tract of intubated patients. (Falagas et al., 2008)

As noted by the Infectious Disease Society of America, Acinetobacter is “a prime example of mismatch between unmet medical need and the current antimicrobial research and development pipeline.” Acinetobacter spp. are notorious for their ability to acquire antibiotic resistance. (Lee et al., 2004) Antimicrobial resistance among Acinetobacter spp. has increased substantially in the past decade and has created a major public health dilemma. The most potent antibiotic drug class currently available are the carbapenems, but resistant strains have emerged. We have studied the antimicrobial resistance pattern among Acinetobacter isolates by Kirby-Bauer disc diffusion method. In our study, Acinetobacter isolates showed resistance to most of the antibiotics available. Acinetobacter is universally resistant to penicillin, ampicillin and cephalothin. Various susceptibility to second and third generation cephalosporins have been reported. (Houang et al., 2001) Acinetobacter possess a wide array of β-lactamases that hydrolyze and confer resistance to penicillins, cephalosporins and carbapenems. AmpC cephalosporinases are chromosomally encoded and confer resistance to broad-spectrum cephalosporins. Class D oxacillin-hydrolyzing-type enzymes, Class B metallo β-lactamases (MBLs), hydrolyze a broad array of antimicrobial agents, including carbapenems. Increasing antimicrobial resistance leaves few therapeutic options for MDR Acinetobacter infection. In the present study, imipenem was most sensitive drug 73 (82.95%) followed by colistin 62(70.45%), Tygycyclin 59(67.05%), ciprofloxacin 55(62.50%), ofloxacin 54(61.36%), amikacin 53(60.23%) gentamycin 52(59.09%). Highest resistance is seen in ampicillin. 70(79.35%) followed by cefixime 66(75.00%), ceftriaxone 62(70.45%) amoxicillin + clavulanic acid 61(69.23%), cephapaxime 57(64.77%), ticarcillin + clavulanic acid 54(61.35%) cotrimoxazol 49(55.68%), pipercillin + tazobactim 40(45.45%) Table 2. (Sinha et al., 2006) reported 35.00% IMIPENEM resistant Acinetobacter. (Lee et al., 2004) reported 21.18% (Corbella et al.,) reported 36.00% carbapenem resistant A. baumannii from the patients admitted to ICU.

Methods Pseudomonas 215 greenish pigmented, non-duplicate consecutive P. aeruginosa isolated from the clinical specimens were identified by standard bacteriological methods (colonial morphology, citrate, and oxidase etc). The isolates were recovered from wound, urine, pus, endotrachial tube, catheter tips and body fluids. Demographic information on the isolates includes the age of the patient, sex, type of clinical specimens and wards. Antibiotic susceptibility testing was determined by disc diffusion method using Mueller-Hinton agar plates. Bacterial suspension was prepared in Andrasss peptones water to give concentration an equivalent of 0.5 McFarland standards. The bacterial suspension were inoculated on the Mueller-Hinton agar plate by swabbing to give a smooth lawn, and antibiotic discs were placed on it, incubated at 37°C overnight.
**Table 1** Various clinical samples of *Acinetobacter baumannii*

| SAMPLE   | NO | %    |
|----------|----|------|
| BLOOD    | 45 | 51.14% |
| PUS      | 18 | 20.45% |
| URINE    | 15 | 17.05% |
| SPUTAM   | 10 | 11.36% |

**Table 1A** Distribution in various clinical specimens of *Pseudomonas*

| Clinical specimen | frequency % |
|-------------------|-------------|
| Body fluids       | 10(4.65%)   |
| Endotrachial tube | 32(14.88%)  |
| Pus               | 17(7.91%)   |
| Sputum            | 34(15.80%)  |
| Wounds            | 54(25.12%)  |
| Catheter tip      | 14(6.51%)   |
| Urine             | 54(25.12%)  |
| **Total**         | **215**     |

**Table 2A** Antimicrobial susceptibility pattern of *P. aeruginosa*

| Antibiotics            | Sensitivity | Resistance  |
|------------------------|-------------|-------------|
| Amoxicillin            | 12 (5.58%)  | 203 (94.42%)|
| Cefixime               | 45 (20.93)  | 170 (79.07%)|
| Ceftazidime            | 106 (49.30) | 109 (50.70%)|
| Cefipime               | 90 (41.86)  | 125 (58.14%)|
| Co-trimoxazole         | 85 (39.53)  | 130 (60.47)|
|                        |             |             |
| Ciprofloxacin          | 135 (62.79) | 80 (37.21)  |
| Ofloxacin              | 115 (53.49) | 100 (46.51) |
| Amikacin               | 140 (65.12) | 73 (38.88)  |
| Tigecycline            | 119 (55.35) | 96 (44.65)  |
| Polymyxin-B            | 152 (70.70) | 63 (29.30)  |
| Cefperazone+Sulbactam  | 123 (57.21) | 92 (42.79)  |
| Ticarcillin            | 92 (42.79)  | 122 (57.21) |
| Piperacillin+Tazobactam| 176 (81.86) | 39 (18.14)  |
| Imipenem               | 170 (79.07) | 45 (20.93)  |
| Doripenem              | 172 (80.00) | 43 (20.00)  |
| ANTIMICROBIAL AGENT | NO OF RESISTANCE/SENSIVITY | TOTAL |
|---------------------|---------------------------|-------|
| Ampicillin          | 70/18                     | 88/100.00 |
| Cephalexine         | 57/31                     | 88/100.00 |
| Cefitazime          | 66/22                     | 88/100.00 |
| Co-Trimoxazole      | 49/39                     | 88/100.00 |
| Ciprofloxacin       | 33/55                     | 88/100.00 |
| Ofloxacin           | 34/54                     | 88/100.00 |
| Gentamicin          | 36/52                     | 88/100.00 |
| Amikacin            | 35/53                     | 88/100.00 |
| Tigecyclin          | 29/59                     | 88/100.00 |
| Amoxicillin+clavulanic | 61/27                 | 88/100.00 |
| Ceperaseone+sulbactam | 33/55                | 88/100.00 |
| Tececarilin+clavulanicacid | 54/34            | 88/100.00 |
| Piparacillin+Tazobactam | 40/48              | 88/100.00 |
| Imipenem            | 15/73                     | 88/100.00 |
| Cefitaxone          | 62/26                     | 88/100.00 |
| Colistin            | 26/62                     | 88/100.00 |
|                     | 29.55%/70.45%             | %/100.00 |
**Table 3** Shows the antibiotic susceptibility pattern of non-fermentors *A. baumannii* and *P. aeruginosa*

| ANTIMICROBIAL AGENT | Resistance/ % | Sensitivity/ % | OBIAL AGENT | NO. Resistant | NO Sensitivity |
|---------------------|---------------|----------------|--------------|---------------|----------------|
| Ampicillin/         | 70(79.55%)    | 18(20.45%)     | Amoxicillin  | 203 (94.42%)  | 12 (5.58)      |
| Co-Triomaxazole     | 49(55.68%)    | 39(44.32%)     | CEFIXIME     | 170 (79.07%)  | 45 (20.93)     |
| Cefotaxime          | 57(64.77%)    | 31(34.33)      | CEFAZIDI ME  | 109(50.70%)   | 106(49.30)     |
| Cefizime            | 66(75.00%)    | 22(25.00%)     | CEFIPIME     | 125 (58.14%)  | 90(41.86)      |
| CEPHOTAXIME         | 57(64.77%)    | 31(34.33)      | CO-TRIMO XAZOLE | 130(60.47%) | 85(39.53)      |
| Ciprofloxacin       | 33(37.50%)    | 55(62.50%)     | OFLOXACIN    | 100(46.51%)   | 115(53.49)     |
| Gentamycin          | 36(40.91%)    | 52(59.09%)     | AMIKACIN     | 73(88%)       | 140(65.12)     |
| Imipenem            | 15(17.05%)    | 73(82.95%)     | TEGICYCLINE POLIMYX | 96(44.65%) | 119(55.35)     |
| Ofloxaicin          | 34(38.64%)    | 54(61.36%)     | IN-B         | 63(29.30%)    | 152(70.70)     |
| Ticercyclin         | 29(32.95%)    | 59(67.05%)     | CEPHERAZONE +SULBACTAM | 92(42.79%) | 123(57.21)     |
| Ceperazone + Sulbactam | 61(69.32%) | 37(30.68%)     | -            | -             | -              |
| Gentamycin          | 36(40.91%)    | 52(59.09%)     | Doripenem    | 43(20.00%)    | 172(80.00)     |
| Cefixime            | 66(75.00%)    | 22(25.00%)     | -            | -             | -              |
| Cephalosporin + Sulbactam | 33(37.50%) | 55(62.50%)     | -            | -             | -              |
| Colistin            | 61(69.32%)    | 26(30.68%)     | TICARICLIN NUTCLAS AR | 122(57.21%) | 92(42.79)      |
| Pipacillin/Tazobactam | 61(69.32%) | 38.64%         | PIPERACIL LIN+TAZO BACTAM | 39(18.14%) | 176(81.86)     |
| Imipenem            | 15(17.05%)    | 73(82.95%)     | IMIPENEM     | 45(20.93%)    | 170(79.07)     |
| Cefixone            | 62(70.45%)    | 26(29.55%)     | -            | -             | -              |
| Colistin            | 26(29.55%)    | 62(70.45%)     | -            | -             | -              |

The following antibiotic discs were tested, amoxicillin (30ug), cefexime (30ug), ceftazidine (30ug), cefepine (30ug) co trimaxazole (30ug), ciprofloxacin (1ug), ofloxaicin (1ug), gentamycin (1ug), amikacin (30ug), polymixin-B (10ug), tegycycline (10ug) cefeperazone plus salbactem (75/30), peparacillin plus tazobactem (100/10 ug) imepenem (10ug), dorepenem (10ug).
The zone of inhibition diameter was measured using calibrated ruler and interpreted as susceptible, intermediate or resistant in accordance to CSLT guidelines. Multidrug resistance is defined as isolates resistance to more than three classes of drugs.

Results and Discussion

Over 12 month study period, *P. aeruginosa* isolates accounted for 215. Significant proportion of isolates were recovered from wounds specimen 54(25, 12%) and urine 54 (25. 12%) followed by sputum 34(15. 81) ET32 (14. 38%), pus 17 (7. 9%) catheter tips 14 (6. 51. %) urine) body fluids 10 (4. 65%) high in wound and urine least in body fluids (Table 1A).

The antibiotic susceptibility pattern of *P. aeruginosa* isolates as presented in Tables 2A showed that the isolates were highly susceptible piperclillin plus tazobactem 176(81. 86%), doperenem 172(80%), imepenem 170(79. 07%) polymixin-B152 (70. 70%) amikacin 140 (65. 12%) ciprofloxacain 135 (62. 75%), cotrimaxazole 130(60. 47%) and moderately to cefepime 125(58%) ticarcillin 122 (57. 21%) cfetzazidine 109 (50. 70%) and least to ofloxacin100(46. 5i%).

High level of resistance was observed with amoxicillin 203(94%), cefixine 170 (79. 07%). Majority of the isolates that exhibited multidrug resistant pattern. *Pseudomonas aeruginosa* is ranked second among gram-negative bacteria isolated in hospital environmental, and leading cause of nosocomial infections responsible for morbidity and mortality rate. High prevalence of pseudomonal infections is common among critically ill patients on admission on intensive care unit and those with underlying clinical conditions (Raja et al., 2007) epidemiological data of bacterial pathogens as in this study might be difficult as there are other variables that influences the outcome of results such as, clinical specimens received for examination, studied population, type of hospitals and geographical locations.

In this study highest 39(18 sensitivity.14) was seen in combination drugs 45(20like.93) piperacillin and tazobactem (8143(20.86%). 00) sensitive to carbapenems like domperenem (80. 00%) and imepenem (79. 07%) was comparatively high. Prevalence of *P. aeruginosa* isolates varied similar studies like Aljesser and Elkhizzi with clinical conditions and specimens. In the (2004) sensitivity of imepenem (90. 1%) and European Prevalence of Infection in Intensive piperacillin and tazobactem (90. 6%). raja and Care (EPIC), *P. aeruginosa* was predominant Singh (2007) showed sensitivity to imepenem gram-negative bacteria isolated from (90. 1%), piperacillin and tazobactem (90. 6%). bronchopulmonary infections and accounts for Sensitivity to cefepime and cfetzazidine ranges 17% of health care-associated pneumonia and late onset ventilate associated pneumonia and from 40=50% is same as study conducted by accounts for significant cases of cystic fibriosis. Garba et al., The distribution of isolates differs with studies Highest resistance was seen to amoxicillin (97). and clinical specimens, In Zaria, Olayinka et al., 2004 reported 51. 1 % in urine, 41. 3 % in 4%) similar to Garba et al., resistance to wound and 1. 1% in sputum, while 4. 6% in coprofloxacin and oflaxacin ranges urine in Jos. In Ile-Ife, southwestern Nigeria, from 50-60%. Most disturbing pattern observed prevalence of 11. 1% in open musculoskeletal in this study was the multidrug resistance injuries', and in Ibadan, isolate rate of 16. 8% exhibited by most of the isolates (no pan drug with 41. 9% and 39. 35 from ear and wound resistance).

Although, similar pattern had been swab
respectively (Ogbolu et al., 2008). Reported in studied conducted in Zaria”, in Jamacia 29, in Italy”, Saudi Arabia; and Brazil.

However, the possibility of P. aeruginosa In conclusion, the multidrug resistance by P. contaminators of wounds and catheter tips cannot be ruled out. This is possible in hospital aeruginosa isolated in this study posed direct environment where strict hand washing procedure is not strictly adhered to clinical consequence in term of patient management and infection control approach in hospital environment. And also more restricted and rational use of these drugs is necessary. And unhygienic procedure especially in wound dressing and insertion of indwelling catheter may be a contributory factor. Majority of isolates were recovered from patient on admission, this observation affirmed the significant role of this organism in nosocomial infection, similarly was the pattern in wounds and catheter tip specimens.

The unique feature of P. aeruginosa isolates is the resistance to variety of antibiotics, primarily attributed to low permeability of the cell wall, production of inducible cephalosporinase, active efflux and poor affinity for the target (DNA gyrase) (Lim et al., 2009). The ongoing surveillance of Acinetobacter baumannii & Psuedomonas aeruginosa microorgan-isms is important to help direct antimicrobial therapy and monitor the emergence of potentially drug-resistant strains in NMC & Hospital Nellore.

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