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Prevalence of Multi Drug Resistant Non- fermenter *Acientobacter baumanni* 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 t0 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. baumanni*. 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. bumannii* 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

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, suchas B-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 pat-terns 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 spec-imens collected from patients with nosocomial infections and processed by the microbiology lab-oratory 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, cephotaxime, cefizime, Co-Trimoxazole, ciprofloxacin, Oflaxacin, gentamicin, amikacin, tigecycline, amoxycillin with clavulanic acid, cefoperazone with sulbactam, ticarcillin with clavulanic acid, piperacillin with tazobzctim, imipenem, ceftriaxone, colistin. 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 88*Acinetobacter* 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%), ciproflaxacin 55(62.50%), oflaxacin 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.oo%), ceftriaxone 62(70.45%) amoxicillin + clavulanic acid (61(69.23%), cephotaxime 57(64,77%), ticarcillin + 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 acenitobacter from blood 45-(51.14%) followed by wound infections (pus18.-(20, 25%) pneumonea (sputum-10(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%), ciproflaxacin 55(62.50%), ofloaxacin 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.oo%), cefriaxone 62(70.45%) amoxicillin + clavulanic acid 61(69.23%), cephotaxime 57(64,77%), ticarcillin + clavulanic acid 54(61,35%) co-trimoxazole 49(55.68%), piperacillin + 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 Andrase peptones water to give concentration an equivalent of 0. 5McFarland 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 *Acenitobacter 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%0
Endotrachial tube	32(14. 88%0
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(88)	
tigecycline	119(55. 35)	96(44. 65)	
Polymyxin-B	152(70. 70)	63(29. 30)	
Cefperazone+Sulbactam	123(57. 21)	92(42. 79)	
ticarcilin	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)	

Table.2 Antimicrobial pattern of *A.baumanni*

ANTIMICROBIAL AGENT	NO OF Resistance/ %	NO OF Sensitivity/ %	TOTAL
Ampicillin	70	18	88
	79.55%	20.45%	100.00 %
Cephotaxime	57	31	88
	64.77%	35.23%	100.00 %
Cefizime	66	22	88
	75.00%	25.00%	100.00 %
Co-Trimoxazole	49	39	88
	55.68%	44.32%	100.00 %
Ciprofloxacin	33	55	88
	37.50%	62.50%	100.00 %
Oflaxacin	34	54	88
	38.64%	61.36%	100.00 %
Gentamycin	36	52	88
	40.91%	59.09%	100.00 %
Amikacin	35	53	88
	39.77%	60.23%	100.00 %
Tigecyclin	29	59	88
	32.95%	67.05%	100.00 %
Amoxicillin +clavulanic	61	27	88
	69.32%	30.68%	100.00 %
Ceperazone+sulbactam	33	55	88
	37.50%	62.50%	100.00 %
Tecarcillin +clavulanicacid	54	34	88
	61.36%	38.64%	100.00 %
Piparacillin+Tazobactam	40	48	88
	45.45%	54.55%	100.00 %
Imipenem	15	73	88
	17.05%	82.95%	100.00 %
Cefriaxone	62	26	88
	70.45%	29.55%	100.00 %
Colistin	26	62	88
	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)
			CEFIXIME	170 (79. 07%)	45 (20. 93)
Cephotaxime	57(64.77%)	31(34.33)	CEFTAZIDIME	109(50.70%)	106(49.30)
			CEFIPIME	125 (58. 14%)	90(41. 86%)
Cefizime	66(75.00%)	22(25.00%)	CO-TRIMOXAZOLE	130(60.47%)	85(39’53%%)
Co-Trimoxazole	49(55.68%)	39(44.32%)	CIPROFLOXACIN	80(37.25%)	135(62.74%)
			OFLOXACIN	100(46. 51%)	115(53. 49%)
Ciprofloxacin	33(37.50%)	55(62.50%)	AMIKACIN	73(88%)	140(65. 12%)
			TEGICYCLINE	96(44. 65%)	119(55. 35%)
Oflaxacin	34(38.64%)	54(61.36%)	POLYMYXIN-B	63(29. 30%)	152(70. 70%)
			CEFPERAZONE+SULBACTAM	92(42. 79%)	123(57. 21%)
Gentamycin	36(40.91%)	52(59.09%)	-	-	-
			-	-	-
Amikacin	35(39.77%)	53(60.23%)	-	-	-
			Doripenem	43(20. 00%)	172(80. 00%)
Tigecyclin	29(32.95%)	59(67.05%)	-	-	
Amoxicillin +clavulanic	61(69.32%)	27(30.68%)	-	-	
Ceperazone+sulbactam	33(37.50%)	55(62.50%)	-	-	
Tecarcillin +clavulanicacid	61.36%	38.64%	TICARCILINUTCLASAR	122(57. 21%)	92(42. 79%)
Piparacillin+Tzobactam	40(45.45%)	48(54.55%)	PIPERACILLIN+TAZOBACTAM	39(18. 14%)	176(81. 86%)
Imipenem	15(17.05%)	73(82.95%)	IMIPENEM	45(20. 93%)	170(79. 07%)
Cefriaxone	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), cefepime (30ug) co trimaxazole (30ug), ciprofloxacin (l ug), ofloxacin (1ug), gentamycin (I ug), amikacin (30ug), polymixin-B (10ug), tegycycline (10ug) cefeperazone plus salbactem (75/30), peperacillin 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 2 A showed that the isolates were highly susceptible pipercillin plus tazobactem 176(81. 86%), doperenem 172(80%), imepenem 170(79. 07%) polymixin-B152 (70. 70%) amikacin 140 (65. 12%) ciprofloxacin 135 (62. 75%), cotrimaxazole 130(60. 47%) and moderately to cefepime 125(58%) ticarcillin 122 (57, 21%) ceftazideine 109 (50. 70%) and least to ofloxacin100(46. 5i%).

High level of resistance was observed with amoxicillin 203(94%), cefixime 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) piperacllin 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 ceftazidine 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 clincal 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 Brazir.

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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