Multidrug-resistant acinetobacter infection and their susceptibility patterns in a tertiary care hospital

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ABSTRACT

Background: Antibiotic-resistant Acinetobacter nosocomial infection is a leading problem. It acts as an opportunistic pathogen to cause a wide spectrum of infection including nosocomial pneumonia, meningitis, endocarditis, skin and soft tissue infections, urinary tract infection, conjunctivitis, burn wound infection and bacteremia. Multidrug-resistant Acinetobacter infection creates a great problem in hospital setting. Materials and Methods: The clinical specimens obtained from ICU and different surgical and medical wards were investigated using standard microbiological techniques to know the distribution of and their resistant profile. Antimicrobial resistance was studied using the modified Kirby Bauer disk diffusion technique following the CLSI protocol. Results: Major infections found in different medical wards, surgical wards and ICU were due to Acinetobacter baumannii (74.02%), A. lowfii (14.2%), A. haemolyticus (7.79%), A. junii (3.8%) among Acinetobacter spices. Acinetobacter showed increased resistant against majority of commercially available drugs imipenem (5.2%), meropenem (9.75%), piperacillin-tazobactum (18.2%), netilmicin (16.24%), amikacin (14.29%), ceftazidime (74.1%), gentamicin (70.13%), ofloxacin (42.21%). Conclusion: A. baumannii was found to be associated with UTI, RTI, septicemia, bacteremia, and meningitis and wound infection. A. baumannii displayed higher resistance to more number of antibiotics than other nosocomial pathogens from ICU.

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Key words: Acinetobacter, antibiotic resistance, nosocomial infection

INTRODUCTION

Acinetobacter baumannii is an opportunistic pathogen that is frequently involved in outbreaks of infection occurring mostly in intensive care units.¹ Members of genus *Acinetobacter* is gram negative, nonmotile nonspore forming encapsulated coccobacilli belonging to family Neisseriaceae.² It is an opportunistic pathogen found to be associated with wide spectrum of infection including nosocomial pneumonia, meningitis, endocarditis, skin and soft tissue infections, urinary tract infection, conjunctivitis, burn wound infection and bacteremia posing risk for high mortality.^{3,4} *Acinetobacter* pneumonia generally occurs in patients with diminished host defenses (e.g. alcoholism, tobacco use, diabetes mellitus, and renal failure, underlying pulmonary disease).⁵⁻⁷ Outbreak

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of Acinetobacter infections is linked to contaminated respiratory equipments, intravascular access devices, bedding materials and transmission via hands of hospital personal.⁸ It typically colonizes skin and indwelling plastic devices of the hospitalized patients.9 MDR strains of Acinetobacter isolates are a growing problem and have been widely reported.¹⁰ Most A. baumannnii are now resistant to ampicillin, carbenicillin, cefotaxime, and chloramphenicol, with some centers reporting up to 91% of nosocomial Acinetobacter resistant to Resistance to tobramycin and amikacin is increasing. Fluoroquinolones, colistin, imipenem, and meropenem may retain activity against nosocomial Acinetobacter.¹¹ Ertapenem, the newest of the carbapenems, has little intrinsic activity against Acinetobacter and should not be used.¹² However, the rapid development of significant quinolone resistance in France, aminoglycoside resistance in Germany, and carbapenem resistance in selected regions worldwide raises an important therapeutic problem.^{13,14}

MATERIALS AND METHODS

The study was conducted for a period of 2 years. A total 4180 specimens like blood sample, pus, CSF, and other body

Table 1: Identification scheme of Acinetobacter species								
Species total no	Haemolysis	Growth		OF test	Arg	Mal	Gelatin	C sensitivity
	on BA	37°C	42°C				liquefaction	
Ac b complex	—	+	+(50%)	saccharolytic	+	+(79.4%)	—	R
A. lowfii	—	+	—	S (72%)	—	+(02%)	—	S
A. haemolyticus	+	+	_	NS	+	—	+	R
A. junii	_	+	_	NS	+	_	_	R

BA – Blood agar; OF – Oxidation-fermentation test; Arg – Arginine; Mal – Malonate; C – Chloramphenicol; S – Sensitive; R – Resistant

fluids were subjected to simplified phenotypic identification scheme [Table 1]. Antimicrobial susceptibility testing was done. Presumptive identification of Acinetobacter was made by inoculation on MacConkey agar medium incubated at 37°C. Urine samples were inoculated on CLED also. All nonlactose fermenting members subjected to gram staining, oxidase, catalase, and hanging drop preparation. Acinetobactor are gram-negative bacilli or coccobacilli, oxidase negative, nonmotile, catalase positive. Speciation was done on the basis of glucose oxidation, gelatine liquefaction, haemolysis, growth at 35° and 42°C. Antimicrobial susceptibility testing was done by modified Kirby-Bauer disk diffusion method with imipenem, meropenem, piperacillin-tazobactum, netilmicin, amikacin, ceftazidime, gentamicin, ofloxacin, chloramphenicol disks. The screening test for detection of inducible beta lactamases (IBL), extended spectrum beta lactamases (ESBL), and MBL was done by the disk approximation method and double disk synergy respectively.

RESULTS

During the period of study from January 2010 to December 2011 a total of 4180 samples were examined from of different age group admitted in various medical wards, surgical wards and ICU. Nonfermenter isolates account for 12% and Acinetobacter isolates account for 4.5% of total organism isolated during the study period. Pseudomonas was the most common nonfermenter (69.44% of total non fermenter isolated).

The male: female ratio was 1.5:1. Acinetobacter infection was more common in patients of aged over 40 yrs. Most of these patients had respiratory problems like COPD, bronchial asthma, respiratory failure. Infection in neonates was common in preterm babies. In 87.5% sample growth was monomicrobial. In 12.5% sample growth was polymicrobial. Escherichia coli was the most common associated organism with Acinetobacter in the case of UTI. Staphylococcus aureus was the associated organism in the case of wound infection, cellulitis, and abcess. Acinetobacter was isolated most commonly from surgical ward, medical ward, burn unit, and two isolates were isolated from humidifier ventilator and two isolates from the OT table.

The present study shows more strains belonging to Acinetobacter baumanii complex 114 (74.02%) of total

Table 2: Different spices of Acinetobacter isolated from clinical samples

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Acinetobacter sp	No. of isolates (%)		
A. baumannii	114 (74.02)		
A. lowfii	22 (14.2)		
A. haemolyticus	12 (7.79)		
A. junni	06 (3.8)		

Table 3: Sensitivity pattern of Acinetobacter isolate to different antibiotic

Antibiotics	Sensitivity patterns (%)	Resistant patterns (%)
Imipenem (10 mcg/ disk)	146 (94.80)	08 (5.2)
Meropenem (10 mcg/disk)	139 (90.25)	15 (9.75)
Piperacillin-tazobactum (100/10/mcg/disk)	126 (81.8)	28 (18.2)
Netilmycin (30 mcg/disk)	129 (83.76)	25 (16.24)
Amikacin (30 mcg/disk)	132 (85.71)	22 (14.29)
Ceftazidime (30 mcg/disk)	40 (25.9)	114 (74.1)
Gentamicin (10 mcg/disk)	46 (29.87)	108 (70.13)
Ofloxacin (5 mcg/disk)	89 (57.79)	65 (42.21)
Chloramphenicol (30 mcg/disk)	12 (07.79)	142 (92.20)

Acinetobacter isolates [Table 2]. Other species includes A.lowfii 22 isolates (14.2%), A. haemolyticus 12 (7.79%) isolates, A junni 06 (3.8%) isolates.

One of the striking features of genus Acinetobacter is the ability to develop antibiotic resistant extremely rapid in response to challenge with new antibiotics. In the present study, strains were resistant to imipenem (5.2%), meropenem (9.75%), piperacilin-tazobactum (18.2%), netilmicin (16.24%), amikacin (14.29%), ceftazidime (74.1%), gentamicin (70.13%), ofloxacin ((42.21%), chloramphenicol (92.20%) [Table 3]. IBL and ESBL were detected 10% and 8% isolates of Acinetobacter respectively. The difference in susceptibility pattern was due to environmental factors and different pattern of antimicrobial usage.

DISCUSSION

Acinetobacter species has emerged as an important pathogen causing life-threatening infections both in community and hospital. Rapid emergence of multidrug-resistant Acinetobacter has further made the situation critical.¹⁵ Acinetobacter is found ubiquitously in nature, soil and also

Table 4: Number of other nonfermenter andAcinetobacter isolated from various samples					
Specimen	Total No Nonfermenter Acinetobo				
Pus/Swab	1400	156	48		
Urine	1100	138	40		
Sputum	800	90	35		
Blood	700	60	22		

08

52

02

07

TA – Tracheal aspirate; ET tube – Endotracheal tube

80

100

CSF

Others (TA, ET tip)

in skin as commensal. Infection is commonly transmitted through aerosol. Prior use of broad spectrum antibiotics, cross infection by hand of hospital staff, ventilator machine are all potential risk factors for development of multidrug-resistant *Acinetobacter* infection in hospital.¹⁶ In one study at JIPMER hospital respiratory infections due to *Acinetobacter* in mechanically ventilated patients in ICU were 44.7%. One recent study revealed that *Acinetobacter* spp. was responsible for 35% of ventilator associated pneumonia (VAP), making it the most conspicuous and dominant pathogen among all other bacteria encountered in that study.¹⁷

In our study, a total of 4180 samples were studied, out of which 504 (12.05%, n=4180) nonfermenters were isolated [Table 4]. *Pseudomonas* was the most common isolated nonfermenter (69.44%, n=504). *Acinetobacter* species accounted for (30.55%, n=504) of total nonfermenter and 4.5% of total positive culture. Most of the isolated *Acinetobacter* species were sensitive to imipenem, meropenem. However, 10% of them were IBL producers and 8% were ESBL producers.

CONCLUSION

In conclusion, multidrug-resistant *A. baumannii* was responsible for majority of the *Acinetobacter* infections at our hospital. Injudicious use of antibiotics, mechanical ventilation, cross infection was found to be potential risk factors for development of *Acinetobacter* infection.

During routine microbiological work nonfermenter GNB other than *Pseudomonas aeruginosa* are not taken seriously and are dismissed as contaminants. But the rate of isolation of *Acinetobacter* in various studies indicates its role as nosocomial pathogen and also as an agent of community acquired infection. Traditional typing methods like phenotyping and antibiogram typing have advantage over genotyping as they are readily available and cost effective.

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