A Study On Pattern Of Antimicrobial resistance in Clinical Isolates Of Acinetobacter Spp at a Tertiary Care Hospital

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ABSTRACT

Background and Objective: Acinetobacter has emerged as an important nosocomial pathogen, and can cause a wide spectrum of clinical infections in the ICU. In recent years the incidence of Acinetobacter infection is a great concern due to its ability to develop multiple resistance mechanisms against major antibiotic classes including cephalosporins, aminoglycosides, carbapenems, and quinolones. Hence this study was undertaken to determine the antibiotic resistant pattern of Acinetobacter isolates from clinical samples.

Materials and Methods: A total of 63 isolates of Acinetobacter spp collected from various clinical samples during the six month period (January 2014-June 2014) were included in this study. Isolates were subjected to antibiotic susceptibility testing by Kirby Bauer disk diffusion method and the results were interpreted as per recommended by the CLSI. Results: Among 63 isolates studied, 47 (74.6%) were identified as A. baumannii, 16 were A. lwoffii (25.4%). Urine was the most common source of Acinetobacter (36.51%) followed by respiratory specimens (26.98%). About 39 (61.90%) isolates were found to be multidrug resistant by disc diffusion method. The most active drug against MDR Acinetobacter was found to be Polymyxin B (93.65%). Conclusion: To prevent the occurrence and transmission of MDR Acinetobacter in the ICU, antibiotic control strategies and infection control measures are essential.

1. Introduction

Acinetobacter baumannii has emerged as an important, opportunistic pathogen and is involved in various nosocomial infections, especially in intensive care units (ICUs). Its clinical significance is due to the ability to easily acquire resistance genes from the environment, making it one of the organisms threatening the currently available therapeutic panel of antimicrobials [1]. Patients with Acinetobacter colonization often have a history of prolonged hospitalization or antimicrobial therapy, particularly in those who are intubated and have recent surgery and invasive procedures [2].

The virulence of these strains is enhanced by the presence of polysaccharide capsule made up of Lrhamnose, the property of adhesion to human epithelial cells in the presence of fimbriae or capsular polysaccharide, production of enzymes that may damage tissue lipids, lipopolysaccharide component of cell wall and lipid A [3]. The potential source of contamination with Acinetobacter in hospital environment are medical equipments used for therapy or from contamination of environment by airborne route or by contact with patients. Infection control measures and strict isolation procedure of colonized or infected patients prevents the dissemination of these strains to the environment [1,3].

Antimicrobial resistance in A. baumannii is due to antimicrobial-inactivating enzymes, reduced access to bacterial targets and mutations that change targets or cellular functions (alterations in penicillin-binding proteins; PBPs) [4]. Carbapenem resistance has been observed frequently in Acinetobacter spp, due to less outer membrane permeability, increased efflux systems, and carbapenem hydrolyzing enzymes- Carbapenemase and increased Amp C β lactamase production [5,7]. Many carbapenemase-producing A. baumannii isolates are resistant to all the available therapeutic agents except the Polymyxins and to the drugs with significant toxicity and poor penetration to respiratory secretions [6].

There are increasing number of reports of the variable susceptibility of Acinetobacter isolates against the multiple antibiotics around the world and only few therapeutic options are available for the treatment of the infections caused by this organism [8]. Hence this study was done to know the prevalence of Acinetobacter isolated from different clinical samples and to determine their antimicrobial susceptibility profile.
2. Materials and Methods

A total of 63 isolates of Acinetobacter spp collected from various clinical samples like urine, blood, sputum, bronchial washing, endotracheal tip, wound swabs, pleural fluid, cerebrospinal fluid etc during the six month period (January 2014–June 2014) were included in this study. The samples were collected from medical ICU, surgical ICU and outpatient department. The specimens were inoculated on Blood agar and MacConkey agar and incubated for 24 to 48 hours. Acinetobacter isolates were primarily identified by Gram-staining, colony characteristics, motility and oxidase test. Further identification to species level was done by indole production, OF glucose, urease test, nitrate reduction, gelatin liquefaction, malonate and arginine dihydrolase [9].

All the isolates were subjected to antibiotic susceptibility testing by Kirby Bauer disk diffusion method and the results were interpreted as per recommended by the CLSI (Clinical Laboratory Standard Institute) guidelines [10]. The isolates were tested for four basic classes of antibiotics viz beta lactam/beta lactamase inhibitors, aminoglycosides, fluoroquinolones and carbapenems. Multidrug resistance is defined as resistance to atleast three classes of antimicrobial agents which are considered as effective against Acinetobacter spp including carbapenems, antipseudomonal cephalosporins and penicillins, aminoglycosides, fluoroquinolones, monolactams, sulbactams and polymyxins [10].

The following antibiotics were used namely Amikacin (30 μg), ceftazidime (30 μg), cefotaxime (30 μg), cefaperazone / sulbactam (75/30 μg), Ceftipime (30 μg), Imipenem (10 μg), Levofloxacin (5 μg), netilmicin (30 μg), Piperacillin/tazobactam (100 μg), Ciprofloxacin (5 μg), gentamicin (10 μg), tigecycline (15 μg), colistin (10 μg) and, Polymyxin B (300 units). Escherichia coli ATCC 25922 and Pseudomonas aeruginosa ATCC 27853 were used as a quality control [10].

The interpretation of diameter of zone of inhibition of tigecycline is done by using the US FDA susceptible breakpoints (resistance ≤ 14 mm, intermediate 15–18 mm, susceptibility ≥ 19 mm) [8].

Results:

Of the 63 clinical isolates of Acinetobacter studied, 55 (87.30%) were from inpatients and 8 (12.70%) were from outpatients [Fig 1]. Among inpatients studied 65.08% isolates were from medical ICU and 22.22% were from surgical ICU. Out of 63 isolates studied, 47 (74.6%) were identified as A. baumannii, 16 were A. lwoffii (25.4%).

Majority of isolates were obtained from urine sample (36.51%) and respiratory specimens (26.98%) [Fig 2]. The highest percentage of isolates were obtained from patients in the age group of 40 to 60 years. Among the outpatients, Acinetobacter baumannii were obtained from 5 urine samples, 2 pus samples and from 1 blood sample. Out of 190 gram negative bacilli studied from inpatients, 55 (28.95%) were identified as A. baumannii, 16 were A. lwoffii (25.4%).

More than 70% isolates were found to be resistant to aminoglycosides. Cephalosporin resistance was seen in ≥80% isolates. About 70% strains were found to be resistant to fluoroquinolones. Among the isolates from inpatients, carbapenem resistance was found to be 63.64(35/55). Low carbapenem resistance of 12.5% was observed among the isolates obtained from outpatients. Among inpatients 5/35 (14.29%) imipenem resistant strains were resistant to both colistin and tigecycline and 4/35 (11.43%) showed resistance to polymyxin B and colistin. The overall percentage resistance to polymyxin B, tigecycline and colistin were 6.35, 12.70 and 15.87 respectively.

Fig 1: Percentage distribution of Acinetobacter isolates

Fig 2: Sample-wise distribution of Acinetobacter isolates

Fig 3: Percentage isolation of Acinetobacter spp from inpatients
Acinetobacter spp. plays a significant role in the colonization and causing infections in hospitalized patients. In the present study 87.30% isolates were from ICU patients. Acinetobacter spp. can cause a wide spectrum of clinical infections in the ICU, including pneumonia, meningitis, bacteremia, urinary tract infection, endocarditis, peritonitis, and soft-tissue infections. The use of broad spectrum antibiotics, advanced age, immunosuppression, chronic lung disease, presence of invasive devices such as endotracheal tubes, indwelling catheters etc are the risk factors for colonization by Acinetobacter spp [3].

In our study majority of the isolates were obtained from urine (36.51%) followed by respiratory samples (26.58%). This finding is similar to another Indian study reported from Pune [12]. There is an increased incidence of nosocomial pneumonia caused by Acinetobacter in intubated patients on mechanical ventilation [3].

The antimicrobial resistance pattern of these organism vary from country to country and from one hospital to another. The prevalence rate of the multidrug resistance in the Acinetobacter spp. isolates was 61.90%, which is comparable to the study conducted by Patel et al (77.78%). Carbapenems are used as a last resort to treat multidrug-resistant Acinetobacter infections in India. In recent years, there have been reports of reduced susceptibility to carbapenems from various parts of the country [13,14,15,16]. In our study, 57.14% of isolates were resistant to Imipenem. These strains were also resistant to several other antibiotics including penicillins, cephalosporins, quinolones, aminoglycosides and third generation cephalosporins, which is consistent with reports from different studies [14,16,17]. High percentage strains were resistant to cefotaxime (84.13%) and ceftazidime (84.13%). Among the aminoglycosides, 77.78% were resistant to amikacin, 76.19% to netilmicin and 73.02% to Gentamicin. Levofloxacin (65.08%) was superior to ciprofloxacin (77.78%) among the fluoroquinolones.

The most active drug against multidrug resistant Acinetobacter spp was found to be Polymyxin B (93.65%) followed by tigecycline (87.30%) and colistin (84.13%). However, varying reports of tigecycline resistance were found in literature [18,19,20,21]. In a study conducted by Patel et al, 100% Acinetobacter strains were found to be sensitive to polymyxin B. The emerging resistance to colistin and tigecycline in recent years limit their therapeutic options for MDR Acinetobacter spp.

Acinetobacter have been considered as uncommon cause of community acquired infection. About 12.70% of Acinetobacter isolated were from outpatients in our study. A. baumannii is increasingly recognized as a cause of community acquired pneumonia [22]. MDR strains spreads through contaminated hands of health care workers and the colonization and invasiveness depends on the severity of patient condition, use of invasive devices and effect on normal flora.

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