



Original Research Article

A Comparative Study of Antibigram of *Pseudomonas aeruginosa* in Hospital and Community Acquired Infections

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ABSTRACT

Pseudomonas aeruginosa is one of the leading causes of hospital as well as community acquired infections. Due to significant changes in microbial genetic ecology as a result of indiscriminate use of antibiotics, the spread of multidrug resistance is now a global problem. The study aimed at comparing the antibiograms of hospital and community acquired *Pseudomonas aeruginosa* infections and determination of ESBL production in the same isolates. A total of 90 isolates of *Pseudomonas aeruginosa* were isolated from different samples during a period of eight months, from January 2014 to August 2014. Antibigram of the isolates was determined by Kirby Bauer disc diffusion method as per the CLSI guidelines. ESBL producing strains were identified by the Disc Combination method. Out of the 90 isolates, hospital acquired isolates accounted for 65 while community acquired isolates came out to be 25 in number. On comparing the antibiograms of the two categories, it was found that the nosocomial isolates showed more resistance to antibiotics like ceftazidime (60%), tobramycin (46.5%), ticarcillin (58.46%), ticarcillin/clavulanic acid (38.46%) and piperacillin/tazobactam (26.15%) as compared to the community isolates which showed resistance of 32%, 0%, 40%, 24% and 8% respectively. However, all isolates were found to be 100% sensitive to imipenem. Also, a higher percentage of ESBL producing strains was observed among the hospital acquired isolates (26.50%) as compared to the community acquired (12%). The infections caused by hospital strains of *Pseudomonas aeruginosa* are more resistant to the commonly used antibiotics than the community strains. This is perhaps due to injudicious and aggressive use of antibiotics in the hospital set up.

Keywords

Pseudomonas aeruginosa, Nosocomial, ESBL

Introduction

Pseudomonas aeruginosa is an epitome of opportunistic nosocomial pathogens, which causes a wide spectrum of infections and leads to substantial morbidity in immune-compromised patients.

It is also being increasingly implicated in community acquired infections. Unfortunately, *Pseudomonas aeruginosa* demonstrates resistance to multiple antibiotics, thereby jeopardizing the

selection of appropriate treatment (Javiya *et al.*, 2008). Resistance to most antipseudomonal agents has increased by >20%, over the last five years (Jung *et al.*, 2004). The heightened level of drug resistance is a result of the de novo emergence of resistance in a specific organism after exposure to antimicrobials as well as of patient-to-patient spread of resistant organisms (Valerie *et al.*, 2006). *Pseudomonas aeruginosa* develops resistance by various mechanisms like multi-drug resistance efflux pumps, biofilm formation, production of β -lactamases and aminoglycoside modifying enzymes (Ahmed Bakr Mahmoud *et al.*, 2013). Extended spectrum beta lactamases (ESBLs) have been described in *Pseudomonas aeruginosa* only recently. These belong to various families as TEM and SHV types (which are also common among Enterobacteriaceae) and PER and VEB types which have been reported from various parts of the world (Amutha *et al.*, 2009). The threat of infections caused by MDR and ESBL producing *Pseudomonas aeruginosa* has become a major concern in hospital settings and implementation of infection control strategies are the mainstay to avoid the spread of this threat. With prior knowledge of susceptibility pattern in a particular area, it becomes easy to choose appropriate antimicrobial against these resistant strains. The present study therefore was carried out to find out and compare the susceptibility pattern of *Pseudomonas aeruginosa* in hospital and community acquired infections in a tertiary care hospital in Western UP, India. Determination and comparison of ESBL production in the same isolates was also aimed for.

Materials and Methods

The study was conducted in the Department of Microbiology in a tertiary care hospital in

Western UP. It was a cross sectional analytical study conducted over a period of eight months from January 2014 to August 2014.

A total of 90 isolates of *Pseudomonas aeruginosa* were isolated from various samples, out of which 65 were from nosocomial infections and 25 from community acquired. Identification of the organism was done according to the standard bacteriological procedures available (Mackie and McCartney, 2006). Seven ambiguous strains were identified by the VITEK 2-compact automated system (BioMerieux, France) following the manufacturer's instructions. The antimicrobial susceptibility pattern of isolated strains was performed by Kirby Bauer disc diffusion method according to the guidelines of Clinical and Laboratory Standards Institute (CLSI, 2010). The antibiotics used were ceftazidime (30 μ g), tobramycin (10 μ g), gentamicin (10 μ g), levofloxacin (5 μ g), imipenem (10 μ g), ticarcillin (75 μ g), piperacillin (100 μ g), piperacillin/tazobactam (100/10 μ g) and ticarcillin/clavulanic acid (75/10 μ g). Quality control of the test was done by standard ATCC strain *Pseudomonas aeruginosa* 27853.

ESBL production among the isolates was determined by the disc combination method using ceftazidime (30 μ g) and ceftazidime/clavulanic acid (30/10 μ g) (Bradford, 2001).

Statistical analysis of the data was done by using Fisher's exact test. All p values < 0.05 were considered as statistically significant.

Result and Discussion

Out of the 90 isolates of *Pseudomonas aeruginosa*, 30 were isolated from pus

constituting the major portion followed by sputum, urine, foley's catheter tip, endotracheal tube, ear swab, pleural fluid and semen (Table 1).

On comparing the antibiograms of hospital and community acquired isolates, it was observed that the hospital acquired strains showed higher resistance to all antibiotics except piperacillin to which the nosocomial isolates were found more sensitive (Table 2). A statistically significant difference was observed between the sensitivity of hospital and community isolates to antibiotics like tobramycin ($p=0.00$), gentamicin ($p=0.02$), and ceftazidime ($p=0.03$). It was found that tobramycin, gentamicin and ceftazidime showed 53.85%, 61.54%, and 40% sensitivity respectively against hospital acquired isolates as compared to 100%, 88% and 68% sensitivity respectively against community acquired isolates. Also, all isolates from both the groups were found to be 100% sensitive to imipenem. Although an appreciable difference was observed between the sensitivity pattern of levofloxacin, ticarcillin, piperacillin, ticarcillin/clavulanic acid and piperacillin/tazobactam to hospital and community acquired isolates but this difference was not statistically significant.

As far as ESBL production was concerned, out of the total 90 isolates, 20 (22.22%) were found to be associated with ESBL production. Amongst these, 17 (18.88%) were from hospital acquired infections and 3 (3.33%) from community acquired infections. When the two groups were compared, it was observed that out of 65 nosocomial isolates, 17 isolates i.e. 26.50% were ESBL producers and amongst the 25 isolates of community acquired infections 12% i.e. 3 isolates were ESBL producers although this difference was not found to be statistically significant ($p=0.24$) (Table 3).

There has been a rapid emergence of multidrug resistant *Pseudomonas aeruginosa* in recent times, which is an important concern for clinicians who treat these infections. Hospitalized patients are particularly susceptible to infections because the normal skin and mucosal barriers are compromised by use of invasive devices.

In our study we isolated 90 strains of *Pseudomonas aeruginosa* out of which 65 were from hospital acquired infections and 25 were community acquired. The rate of isolation of *Pseudomonas aeruginosa* was higher in hospitalized patients (72.22%) as compared to the outdoor patients (27.77%). A similar observation was made in a study conducted in Varanasi, where the isolation of *Pseudomonas aeruginosa* was found to be more common in indoor patients (73.42%) as compared to that in the outdoor patients (26.57%) (Anupurba *et al.*, 2006). A similar finding was observed in a study at Bijapur where they isolated 84.92% of *Pseudomonas aeruginosa* strains from hospitalized patients as compared to 15.07 % from outdoor patients (Prashant *et al.*, 2011).

Pseudomonas aeruginosa has increasingly been associated with wound infections. In our study, we isolated maximum strains of *Pseudomonas aeruginosa* from pus samples i.e. 30 (33.33%). This was consistent with the finding of a study done in Bhubaneswar in which maximum strains were isolated from pus samples (Pathi *et al.*, 2013). Pus was again the predominant sample from which maximum strains of *Pseudomonas aeruginosa* were isolated in a study conducted in Maharashtra (Satyajeet *et al.*, 2014) and also at Davangere (Mohan *et al.*, 2013). Another study from Gujarat reported higher isolation rates from urine, pus and sputum which accounts to 27% each, followed by ET secretion 14% (Javiya *et al.*, 2008). This variation among these studies could be due to the difference in study

period and sample size, geographical location and patient population.

It has been observed in various studies that nosocomially acquired *Pseudomonas aeruginosa* isolates tend to be more resistant to antimicrobial agents than community acquired strains, frequently displaying resistance and cross resistance to many antibiotics (Arshi Syed *et al.*, 2007). This is due to a continuous selective pressure of regularly used antibiotics. This selective antibiotic pressure leads to development of bacterial resistance by favouring rapid evolution of the bacterial genome. This was the fact that was observed in our study also in which the hospital acquired strains were more resistant to almost all the antibiotics as compared to the community strains. We observed that imipenem and tobramycin were the most sensitive drugs against *Pseudomonas aeruginosa* infections. A similar finding was observed in another study at Davangere in which *Pseudomonas aeruginosa* showed highest sensitivity to imipenem (94.30%) followed by tobramycin (72.15%) (Mohan *et al.*, 2013). As consistent with our findings, Shaikh *et al.* (2014) also observed 100% sensitivity to imipenem. In our study ceftazidime showed 40% sensitivity against nosocomial isolates of *Pseudomonas aeruginosa* which was quite similar to the study done in New Delhi where 38.32% sensitivity to ceftazidime was observed (Indu *et al.*, 2014). The resistance to drug combination like piperacillin/tazobactam was 26.15% in our study which was similar to the resistance observed by another study done in Gujarat (Javiya *et al.*, 2008).

In our study, we observed that 22.22% (20) of the isolates were ESBL producers. This was quite similar to the findings of Shaikh *et al.* (2014) who observed 25.13% (n =47) *Pseudomonas aeruginosa* to be ESBL producers in their study. Another study

conducted at Haryana also showed similar results (Aggarwal *et al.*, 2008).

We could deduce from our study that *Pseudomonas aeruginosa* being a stubborn multidrug resistant pathogen leaves carbapenems as the last resort for treatment of life threatening infections in hospital. Judicious use and constant monitoring are essential to check the spread of carbapenem resistant *Pseudomonas aeruginosa* in hospitals and its subsequent spread in the community. The use of imipenem for the treatment of *Pseudomonas aeruginosa* should be reserved for situations where the infection is polymicrobial or for pseudomonas isolates resistant to other antibiotics.

In cases where imipenem is selected as the antipseudomonal antibiotic, the potential for emergence of resistance should be anticipated. In appropriate circumstances, routine culture and susceptibility tests should be performed to detect the emergence of resistance to *Pseudomonas aeruginosa* as early as possible. Attention by the hospital infection control team is essential to implement stringent preventive measures to contain the spread of the infection and promote the judicious use of antimicrobial agents.

Antibiotic resistance is increasing at an alarming rate, leading to increased morbidity, mortality and treatment costs. A key factor in the development of antibiotic resistance is the inappropriate use of antibiotics. The medical fraternity needs to understand that antibiotics constitute a precious and finite resource. Unless conscious efforts are made to contain the menace of drug resistance, multi-drug resistant organisms, untreatable by every known antibiotic, may emerge, reversing the medical progress made by mankind and throwing us back to the pre-antibiotic era.

Table.1 Distribution of various sources of *Pseudomonas aeruginosa* isolates

Sample	Hospital acquired (n)	Community acquired (n)
Pus	28	2
Sputum	13	10
Urine	13	8
Foley's Catheter	8	0
Endotracheal Tip	2	0
Ear Swab	0	2
Pleural Fluid	2	0
Semen	0	2

Table.2 Sensitivity pattern of isolates of *Pseudomonas aeruginosa* to antibiotics

Antibiotics	Hospital acquired % (n)	Community Acquired%(n)
ceftazidime	40 (26)	68 (17)
Tobramycin	53.85 (35)	100 (25)
Gentamicin	61.54 (40)	88 (22)
Levofloxacin	80 (52)	92 (23)
Imipenem	100 (65)	100 (25)
Ticarcillin	41.55 (27)	60 (15)
Piperacillin	69.23 (45)	52 (13)
piperacillin/Tazobactam	73.85 (48)	92 (23)
Ticarcillin/ Clavulanic acid	61.54 (40)	76 (19)

Table.3 ESBL production in hospital and community acquired isolates

	Hospital Acquired % (n)	Community Acquired % (n)
ESBL	26.15 (17)	12 (3)
Non ESBL	73.85 (48)	98 (22)

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