Research Article

A study of antibiotic sensitivity pattern of *Pseudomonas aeruginosa* isolated from a tertiary care hospital in South Chhattisgarh

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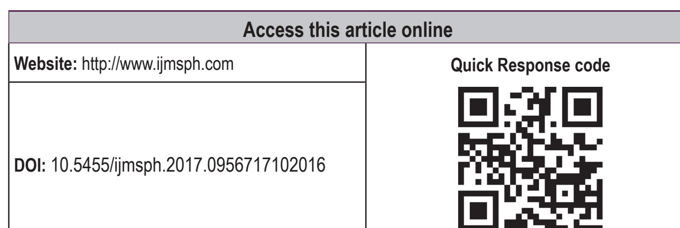
ABSTRACT

Background: *Pseudomonas aeruginosa* is one of the leading causes of hospital-acquired 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 (MDR) is now a global problem. Its general resistance is due to a combination of factor. Several different epidemiological studies indicate that antibiotic resistance is increasing in clinical isolates. **Objectives:** The present study was conducted to find out the current antimicrobial susceptibility pattern of *P. aeruginosa* isolates obtained from various clinical samples at our tertiary care hospital. **Materials and Methods:** The present study was conducted in a tertiary care hospital in South Chhattisgarh, India, from June 2014 to May 2016. One hundred and ninety-eight clinical isolates of *P. aeruginosa* obtained from various clinical samples were studied. They were identified by routine standards and operative procedures, antimicrobial susceptibility testing was done using Kirby-Bauer disc diffusion method, and the results were interpreted according to the CLSI guidelines. Quality control of the test was done by standards ATCC strain *P. aeruginosa* 27853. Data obtained were analyzed and presented in counts and percentage. **Results:** Piperacillin-tazobactam was the most sensitive chemotherapeutic agent with 93% susceptibility rate, followed by imipenem 91% and levofloxacin 83.5%. Amikacin showed better susceptibility rate 78% when compared to that of gentamicin 53%; the susceptibility rate to cephalosporin and aztreonam was relative very low. Most of the *P. aeruginosa* strains were isolated from clinical samples such as pus 73, urine 42, and respiratory secretion 16, and sputum 26. Out of 198 clinical isolates, 34 (17%) clinical isolates of *P. aeruginosa* were found to be MDR. Most of the MDR *P. aeruginosa* strains were isolates from pus, urine, and respiratory sample. **Conclusions:** To prevent the spread of the resistant bacteria, it is critically important to have strict antibiotic policies in our country. There should be surveillance programs for the detection of MDR organisms in every locality. Infection control programs need to be implemented with quality control in every hospital.

KEY WORDS: Multidrug-resistant; Antibiotic Susceptibility Testing; Antipseudomonal Agents; Tazobactam; Aztreonam



INTRODUCTION

Pseudomonas aeruginosa is one of the leading causes of hospital-acquired as well as community-acquired Infections.[1] *Pseudomonas aeruginosa* is an aerobic, motile, Gram-negative rod that belongs to the family, Pseudomonadeceae.[2] It is an opportunistic pathogen,

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meaning that it exploits some break in the host defenses to initiate an infection.[3] It is one of the important bacterial pathogens isolated from various samples. Despite advances in medical and surgical care and introduction of wide variety of antimicrobial agents having antipseudomonal activities, life-threatening infection caused by *P. aeruginosa* continues to cause complications in hospital-acquired infections.[4] Penicillin when first discovered and used for the treatment was a magic drug. A single injection of penicillin could cure a life-threatening infection. Unfortunately, with time due to malpractices of natural causes, most of the cheaper antibiotics have lost their efficacy, and more expensive and complicated antibiotics were introduced and marketed to combat simple infection.[5]

The microbial pathogens, as well as their antibiotic sensitivity pattern, may change from time to time and place to place. Therefore, knowledge of current drug resistance pattern of the common pathogenic bacteria in a particular region is useful in clinical practice. Unfortunately, *P. aeruginosa* demonstrates resistance to multiple antibiotics, thereby jeopardizing the selection of appropriate treatment.[6] 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 patient to patient spread of resistant organism.[7]

P. aeruginosa develops resistance by various mechanisms such as MDR, efflux pumps, biofilm formation, production of β-lactamases (extended-spectrum beta-lactamases [ESBLs]), and aminoglycoside modifying enzymes.[8] The *Pseudomonas* sp. is also the second most common causative organism in post-operative infection in surgical wound. It is a common nosocomial infection after *Staphylococcus aureus* having variable sensitivity pattern for antibiotics.[9]

It is a significant cause of nosocomial infection of the respiratory tract, urinary tracts, wounds, bloodstream, and even the central nervous system. In immunocompromised patients, the infections are often severe and frequently life-threatening. It can survive harsh environmental conditions and displays intrinsic resistant to a wide variety of antimicrobial agents that facilitate the organisms ability to survive in hospital setting.[10] In addition to its intrinsic resistance to various antibiotics, it also readily acquires resistance to the potentially active agents.[11] Since some of the resistance markers are carried by plasmids, the threat to human health is compounded by the possibility of transmission of markers to other Gram-negative pathogens.[12] Resistance to antipseudomonal antibiotics is increasing worldwide. This situation has been compounded by the lack of new classes of antipseudomonal drugs.[13]

Therefore, area-wise studies on antimicrobial susceptibility profiles are essential to guide policy on the appropriate use of antibiotics. The present study was conducted to find out the antimicrobial susceptibility pattern of *P. aeruginosa* isolates obtained from various clinical samples at Microbiology Department in Tertiary Care Hospital, Jagdalpur, South Chhattisgarh, India.

MATERIALS AND METHODS

Materials

The present research work was conducted at Microbiology Department, LSBK Memorial Government Medical College, Jagdalpur, Chhattisgarh, during June 2014 to May 2016. The clinical samples were collected from indoor patients (IDPs) and outdoor patients from different wards of the hospital. Samples were processed for culture, and sensitivity pattern for *P. aeruginosa* was determined against commonly used antibiotics by disc diffusion method.[14] The hospital's laboratory samples comprised blood, pus, swabs different body sites from outdoor patients department (OPD) as well as IDPs from different wards of the hospital.

Sample Collection and Processing

Blood, pus, and urine samples were collected from patients visiting OPD and different wards of the hospital (IDP) and were brought to the Microbiology Clinical Laboratory. Freshly drawn, 3-5 ml blood sample was immediately transferred to 50 ml of tryptone soy broth (CM0129-OXOID) and incubated at 37°C for 24 h. In case of no growth, incubation period was extended for another 24 h. In case of growth positive, it was sub-cultured on cetrimide agar (CM0055-OXOID) and MacConkey agar (CM0007-OXOID) plates, incubated for 24 h at 37°C. Pus samples were directly inoculated on certified agar (CM0055-OXOID) and MacConkey agar (CM0007-OXOID) plates, incubated for 24 h at 37°C. Urine samples were cultured on blood agar, MacConkey agar, and cetrimide agar, incubated for 24-48 h at 37°C. Isolated colonies, after purification, were initially Gram-stained. The isolates were biochemically characterized and identified up to species level according to Bergey's Manual of Determinative Bacteriology.[15]

Antibiotic Susceptibility Test (Disc Diffusion)

The disc diffusion test for *P. aeruginosa* was carried on Mueller-Hinton agar (CM0337-OXOID) as growth medium. Media were prepared according to manufacturer's instructions and sterilized by autoclaving at 121°C for 15 min. Sterilized medium was then cooled in a water bath, and about 25 ml of medium was poured into 90 mm diameter sterile Petri-plates to a depth of 4 mm on a level surface to make the depth of the medium uniform and left at room temperature overnight to check sterility. Inoculum was spread evenly over the entire surface of the Mueller-Hinton agar plates by swabbing back and front across the agar in three directions to give a uniform inoculum to the entire surface. These plates were allowed

to dry before applying the discs. The disc of given potency was applied on the inoculated plates with the help of sterile forceps. Then, the plates were placed in an incubator at 37°C for 18 h in inverted position. After 18 h of incubation, plates were examined and the diameters of zone of inhibition were measured in mm.[14]

Determination of Minimum Inhibitory Concentration (MIC) (Turbidimetric Method)

Individual antibiotics were dissolved in nutrient broth and diluted (nutrient broth). The MIC range varied with different drugs. All MIC range was followed according to the NCCLS guidelines.[16] Inoculums were obtained from an overnight agar culture of the test organism. Inoculums for the MIC test were prepared by taking at least 3-5 well-isolated colonies of the same morphology from an agar plate culture. The top of each colony was touched with a sterile loop, and the growth was transferred into a tube containing 4-5 ml of normal saline. The broth culture was incubated at 35°C until it achieved the turbidity of the 0.5 McFarland standards (usually 2-6 h). This results in a suspension containing approximately 1-2 × 10^8 cfu/ml. The turbidity of the actively growing broth culture was adjusted with the broth to obtain turbidity as compared to that of 0.5 McFarland standards.

A 0.5 McFarland standard was prepared as described in NCCLS one percent V/V solution of sulfuric acid was prepared adding 1 ml of concentrated sulfuric acid to 99 ml of water and mixed well. A 1.75% W/V solution of barium chloride ($BaC1_2$) was prepared dissolving 2.35 of dehydrated $BaC1_2$ and 1 ml of concentrated sulfuric acid to 99 ml of water and mixed well (1.75% H_2O in 200 ml of distilled water). To make the turbidity standard, 0.5 ml of the $BaC1_2$ solution was added to 1% 99.5 ml sulfuric acid solution and mixed well. A small volume of those turbid solutions was transferred to a screw-capped tube of same type as used for preparing the control inoculum and stored in the dark at room temperature. Test tube containing different concentrations (2, 4, 8, 16, and 32 µg/ml) of antimicrobial agent and the control test done without antimicrobial agent were spotted and inoculated with a 2 µl suspension with micropipette. Inoculation was done from the plate containing lowest concentration of antimicrobial agent and the control plate was inoculated finally. Inoculated test tube was incubated at 35°C for overnight. The MIC is the lowest concentration of the anti microbial agent that completely inhibits visible growth. The concentration in which the test tube showed no growth was considered as the MIC of the specific antimicrobial agent.

Determination of Minimum Bactericidal Concentration (MBC)

The MBC is the concentration of antibiotic that kills at least 99.9% of standardized bacterial inoculums. From each of the test tube where MIC was performed, 1 ml was taken in a sterile Petri plate and dispensed 15 ml of melted presterilized Mueller-Hinton media, mixed by rotating both clockwise and counterclockwise, allowed to solidify, and incubated at 35°C for overnight. The lowest concentration at which no growth occurs that was considered as the MBC of the specific antimicrobial agent.

RESULTS

The present study was carried out in the Department of Microbiology, LBRKM Government Medical College, Jagdalpur, for 2 years. A total of 5760 samples were received for culture and sensitivity in Bacteriology Diagnostic Laboratory, of which 1530 samples showed growth on culture. Among 1530 cultured organism, 198 isolates were identified as *P. aeruginosa* (13%). The samples from which we cultured were pus, wound swab, blood, urine, ascitic fluid, pleural fluid, sputum, ear swab, and others. Out of 198 isolated *P. aeruginosa*, the most common samples given positive growth were pus 73 (37%), urine 42 (21%), sputum 26 (13%), other respiratory secretion 16 (8%), blood 14 (7%) pleural 10 (5%), ascetic fluid 06 (3%), ear swab 4 (2%), and other samples 7 (4%) as shown in Table 1.

In the present study, all isolated *P. aeruginosa* were processed for the determination of sensitivity against antibiotics, piperacillin, piperacillin + tazobactam, ticarcillin + tazobactam, ceftazidime, cefepime, aztreonam, imipenem, meropenem, gentamicin, amikacin, ciprofloxacin, levofloxacin, on Mueller-Hinton agar. Quality control of the test was done by standard ATCC strain *P. aeruginosa* 27853.

In our study, the sensitivity patterns of *P. aeruginosa* are shown in Table 2. We found that the more sensitive drug was piperacillin-tazobactam 93% (184), imipenem 91.41% (181), ticarcillin + tazobactam 87% (172), meropenem 83.5% (165), levofloxacin 83% (162), amikacin 78% (155), piperacillin 75% (148), aztreonam 64% (126), gentamicin 53% (105), ceftazidime 52% (102), ciprofloxacin 51% (101),

Table 1: *P. aeruginosa* strains isolated from different samples

Sample	Cases (%)
Pus	73 (36.8)
Urine	42 (21.21)
Blood	14 (7.07)
Sputum	26 (13.13)
Pleural	10 (5.05)
Ascetic fluid	06 (3.03)
Ear swab	04 (2.02)
Respiratory secretions	16 (8.08)
Others	07 (3.53)

P. aeruginosa: Pseudomonas aeruginosa

Table 2: Antimicrobial susceptibility of *P. aeruginosa* isolation (no -198) to various antibiotics

Name of antibiotics	Total number of sample	Number of sample	Sensitive %
Piperacillin	198	148	75
Piperacillin+tazobactam	198	184	93
Ticracillin+tazobactam	198	172	87
Ceftazidime	198	102	51.5
Cefepime	198	94	47.5
Aztreonam	198	126	63.7
Imipenem	198	181	91.4
Meropenem	198	165	83.5
Gentamicin	198	105	53
Amikacin	198	155	78
Ciprofloxacin	198	101	51
Levofloxacin	198	165	83.5

P. aeruginosa: Pseudomonas aeruginosa

Table 3: Distribution of MDR *P. aeruginosa* isolates among clinical specimens

Clinical sample	Total number of isolation	Number of MDR strain	Number of MDR isolation (%)
Pus	73	22	30
Urine	42	8	20
Blood	14	2	15
Sputum	26	7	27
Respiratory	16	3	20
Pleural	10	1	10
Ascetic	06	0	00
Ear swab	04	1	25
Others	07	0	0

MDR: Multidrug resistance, *P. aeruginosa: Pseudomonas aeruginosa*

and cefepime 48% (94). There was no significant difference *P. aeruginosa* infection in sex-wise ratio. We found 52.5% in female, i.e., 104 out of 198 cases, and 94 cases in male, 47.5%.

We also found in our study that Pseudomonas aeruginosa MDR Strians from clinical sioltaes are as shown in table 3

DISCUSSION

In this study, a total of 198 isolates of *P. aeruginosa* were isolated and identified from various clinical samples from the IDP and outpatient and their antimicrobial susceptibility patterns were determined. In our study, we found that most of them belonged to older age group of 41-60 years (41%) and elderly age group of >60 years (32%). This could be explained as due to decreased immunity, prolonged hospitalization, and other associated comorbidities in these age groups. A study done in Ahmadabad, India,[4] showed that 29% of patients were aged between 31 and 45 years.

Ahmed et al.[17] reported a higher prevalence rate among elderly persons of age 61-80 years (43.92%) and sex-wise reported an increased incidence in male sex (77.7%). In our study, there is no significant difference in sex-wise incidence; similarly, a high prevalence of *Pseudomonas* infection was found in the 35-50 years age group.[18]

The distribution of specimen of *P. aeruginosa* may vary with each hospital as each hospital has a different environment and facility associated within. In our study, more than 70% of the *P. aeruginosa* isolates were obtained from pus, wound, swab, urine, sputum, and tracheal aspirates; similar results had been obtained in different studies in other parts of the country reported by Mohanasoundaram[18] and Arora et al.[19] Increasing resistance to different antipseudomonal drugs has been reported worldwide,[20,21] and this is a serious therapeutic problem in the management of disease due to the organisms. We studied resistance pattern of *P. aeruginosa* against 12 antimicrobial agents; *P. aeruginosa* isolates were found most sensitive to imipenem (92%) and piperacillin + tazobactam (93%); this may be due to the restricted use of piperacillin + tazobactam and imipenem in this hospital. This type of finding also reported from other authors. 100% sensitive against imipenem was reported from Mangalore in 2002 by Shenoy et al.[22]

Other studies have shown varying degrees of resistant to imipenem.[6,18,19,23] Meropenem 83.5% sensitive, followed by levofloxacin 83% sensitivity while ciprofloxacin only 51% sensitive, amikacin 78% sensitive were detected to be the most effective drugs for routine use against the *P. aeruginosa* strains investigated. In this study, high percentage of resistance to aminoglycosides had been reported in other studies from other parts of India,[19] Bangladesh,[24] Malaysia,[23] and Turkey.[25]

In our study, we detected the 49% resistance to ciprofloxacin; similarly, higher rate of resistance to fluoroquinolones such as ciprofloxacin (40.5%) had been reported in a study in Kerala, India,[16] and 92% resistance was reported in a study from Malaysia.[26] An earlier study reported from Kathmandu, Nepal,[27] showed amikacin 81.4% sensitive. In our study also, we get 78% sensitive among *P. aeruginosa*. In other study, piperacillin alone tested showed a resistance rate of 55% reported from Kathmandu, Nepal.[26] In our study, piperacillin + tazobactam showed 93% sensitive whereas piperacillin alone showed 74.8% sensitive, followed by ticarcillin + tazobactam 87% sensitive; therefore, it indicates beta-lactamase inhibitor markedly expands the spectrum of activity of drug.

A similar finding is also reported;[23] thus, it determines that the combination drug should be the preferred choice against *P. aeruginosa*. In another study, resistance rated for piperacillin 54.66% had been reported by Shenoy et al.[22] Relatively, low piperacillin resistance (11.5%) had been

reported in inpatients isolates of *P. aeruginosa* from Saudi Arabia.[28] We found the *P. aeruginosa* 49.5% resistant to ceftazidime third-generation cephalosporin drug and 52.5% resistant to cefepime.

Reported from another study, a much higher resistant third-generation cephalosporin ceftriaxone (68.96%) from Kathmandu, 75%, 86%, and 95% had been reported in studies done in India,[19] Bangladesh,[24] and Nepal.[27] Higher sensitivity to ceftriaxone 60% had been reported in studies done in India,[19] Bangladesh,[24] Nepal.[27] Higher sensitivity to ceftriaxone 60% had been reported in another study from Andra Pradesh.[29]

We found sensitivity to 83.2% to levofloxacin. These levofloxacin should be included in the treatment regimen for the *P. aeruginosa*. Another significant finding in this study was the rate of multidrug resistance (MDR) to be 17%; similarly, MDR rate of 19.6% from Malaysia[2] and 20.69% from Kathmandu, Nepal, had reported from studies; a higher rate of MDR had been reported from studies conducted from Nepal (89.4%)[27] and 100% *P. aeruginosa* isolates from Iran.[30] This study has a few limitation that is molecular typing plasmid profile and study of mechanism of development of MDR strain would provide much-needed detail information and these should be also analysis of ESBL-producing *P. aeruginosa* which is a major cause of nosocomial infection with MDR strains in hospital.[31,32]

CONCLUSION

In our study, we found results clearly indicate the evolution of MDR stains and the occurrence of resistance to various antipseudomonal agents among the *P. aeruginosa* isolates. We suggest a more restricted and more rational use of this drug in the treatment of *P. aeruginosa* infection in hospital setting. Regular antimicrobial susceptibility monitoring is essential for local, regional and national level isolates. In the clinical diagnostic laboratory level in all the CHC, District Hospital should have proper diagnostic laboratories to determine the culture and sensitivity report. This would help prescribing the right combination of chemotherapeutic agent and prevent the emergence of MDR strains of *P. aeruginosa*.

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