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A Clinical-Bacteriological Profile of Multidrug Resistant *Pseudomonas aeruginosa* at Tertiary Care Hospital Shahjahanpur, Uttar Pradesh

Jitendra Kumar Chaudhary*

Department of Microbiology, Associate Professor, VarunArjun Medical College and Rohilkhand Hospital, Banthra, Shahjahanpur, Pin –242307, Uttar Pradesh, India

**Corresponding author*

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A B S T R A C T

The *Pseudomonas aeruginosa* is predominant agent causing nosocomial infections. In recent time, it develops resistance continuously to the antibiotics becomes Multidrug-resistant *P. aeruginosa*. So, in cystic fibrosis patents it difficult to eradicate *P. aeruginosa* infections with antimicrobial treatment. Therefore, focus on alternative mechanisms for treating *P. aeruginosa* infections. On the basis of growth, morphological and biochemical characteristics, *P. aeruginosa* strains were isolated from the clinical samples in this work. After that the antibiotic sensitivity was performed and the Multidrug resistant *Pseudomonas aeruginosa* was identified according to CLSI standard guideline chart by measuring the zone of inhibition for *P. aeruginosa*. The isolated strains showed resistance against three or more antibiotics, considered as MDR *Pseudomonas aeruginosa*. By antibiogram pattern 51 showed Multi drug resistant strains out of 102 isolated strains. As *P. aeruginosa* abide to develop resistance to the antibiotics, the quorum sensing increased transcriptional regulator QscR might performs another target. Thus the prevalence of MDR strains of *Pseudomonas aeruginosa* was investigated on current study. The antibiogram pattern revealed 51 MDR strains *of P. aeruginosa*. In which the majority of strains exhibited resistance towards Piperacillin (98%), Ciprofloxacin (90%), Ofloxacin (90%), Levofloxacin (80%) and Tobramycin (60%).

Introduction

The *Pseudomonas aeruginosa* is the most important agent causing nosocomial infections. It is causes predominantly bacteremia, meningitis, pneumonia and urinary tract infection. It is more common in patients with neutropenia, burns, ventilators and cystic fibrosis.[1] It is continuously raising human pathogen, accountable for 12% of urinary tract infections, 10% of blood stream infections, and 8% of surgical wound infections.[2] In incubated patients, it causes mostly ventilator-associated pneumonia which directly responsible to death of patients.[3]*P.aeruginosa* infection becomes chronic infection in cystic fibrosis patients, which is highly illness and death of this

population. Pseudomonas have ability to invade a potential host that monitors cell density through a mechanism which control virulence factors and communicate between bacteria.[4,5] Cystic fibrosis is more vulnerable nowadays because recent antibacterial treatments are unable to eradicate multidrug-resistant *P. aeruginosa* infection which is more prevalent. Thus, in many research focused on alternate mechanisms to target the *P. aeruginosa*. [6]According to therapeutics that target inhibit QS in *P. aeruginosa* might diminish the virulence of the bacterium and that probably help to clear the infection by host immune system. There are Two QS networks in which LasR and RhIR is ideal target in therapeutics.[7]

The Las system made up of LasR transcriptional regulator and LasI synthase protein. LasI synthase is necessary for the producing of the AHL signal molecule N-(3-oxododecanoyl)-l-homoserine lactone (3O-C12-HSL). [8,9]LasR needs3O-C12-HSL for activation of LasR transcription factor.

It was recently denoted that, LasR forms multimers with 3O-C12-HSL, and only that multimeric form of LasR protein is capable to bind DNA and control the transcription of multiple genes. The second QS system to *P. aeruginosa* contains of the RhlI and RhlR proteins. [10,11]The RhlI synthase is essential for producing the AHL N-butyryl-l-homoserine lactone (C4-HSL), and RhlR is transcriptional regulator. When RhlR is combined with C4-HSL this controls the expression of several genes only. This has been determined that both 3O-C12-HSL and C4-HSL are freely diffuse out from bacterial cells. But, 3O-C12-HSL diffusion is comparatively slower than that of C4-HSL.

In adding to LasR and RhlR, there is also third Las system, orphan LasR-RhlR homolog, QscR, but which have no cognate acyl-HSL synthase gene. [12]The qscR mutant is hyper virulent. It is observed that expression of some genes controlled by the LasR-I and RhlR-I that stimulus of QscR and are prematurely triggered in a qscR mutant and enclose genes the phz1 and phz2 phenazine synthesis operons; hcnAB, the hydrogen cyanide synthesis operon; lasB, that codes for elastase; rhlI; and lasI. The mechanism for temporary suppression of these genes by QscR is not clear. [13,15] When acyl-HSL concentrations are low, the QscR might create heterodimers with LasR and RhlR. This would inactivate LasR and RhlR.

It is a greater alarming public health has aroused due to appearance and spread of multidrug resistance bacteria.

For therapeutic options, Multidrug resistant strains have very restricted option for treatment because antibiotic resistance of *P.aeruginosa* infection frequently complicated so need urgency for identification of novel antimicrobial targets and evolution of new antimicrobial drugs for better treatment.[16] *P.aeruginosa* can resistance to broad-spectrum antibiotics quickly because it is omnipresent micro-organism. MDR strains of *P. aeruginosa* are an increasing cause of morbidity and mortality in burn case.

In developing nations *P. aeruginosa* causes remarkable trouble among hospitalized patients because emergence of MDR strains frequently. [17,18]So, prime focus of researchers and practitioners worldwide are to identify MDR strains and contributing mechanism to give it resistance because there is limited number of studies has been done to discover MDR *P. aeruginosa* in India. Thus in the present study, *Pseudomonas aeruginosa* was identified and isolated from the clinical samples taken from tertiary care hospital and the Multidrug resistant strains were assessed by antibiogram pattern.

Materials and Methods

Sampling and processing

The Clinical samples were collected in clinical hospital laboratory during March 2019 to February 2020 and each sample was taken in Peptone water containing tubes and then sent to the Microbiology laboratory. Then, the samples were streaked on Nutrient agar plates and incubated at 37°C for 24 hours. After 24 hours, the separate characteristic colonies were subjected to Gram's staining and then also it should sub-culture on MacConkey agar[19]. The pure isolated *Pseudomonas aeruginosa* were stored in the refrigerator at 4°C.

Identification of *P. aeruginosa*

The sub-culture isolates in MacConkey media were identified on the basis of Bergey's manual of determinative bacteriology,[20]the characterize identification by Gram staining, catalase, oxidase and motility by Hanging drop method and further by performing various biochemical tests such as sugar fermentation tests for glucose, sucrose, lactose, maltose and mannitol, Triple Sugar Iron agar test, Indole production test, citrate test, urease test, methyl red test, Voges-Proskauer test, Nitrate reduction test, H_2S production and Inulin test.

Antibiotic Susceptibility

Antibiotic susceptibility was performed with disc diffusion or Kirby-Bauer test method. [21]The commercially available standard antibiotic discs were used for the Antibiotics sensitivity pattern identification. From sub-culture some bacterial colonies were inoculated into Nutrient broth and incubated at 37°C for 2-4 hours and then the turbidity was adjusted to 0.5 McFarland standards. After that, sterilized Muller Hinton agar media was prepared and solidify on petridishes then incubated up to 30 minute in incubator for remove the excess moisture from the surface and then inoculated by cotton swabs uniformly over the surface of MHA media. Antimicrobial antibodies susceptibility of each isolate was assessed by disk diffusion method i.e., Piperacillin (100g), Piperacillin-tazobactam (100/10g), Tobramycin (10g), Meropenem (10g), Imipenem (10g), Ceftazidine (30g), Cefepime (30g) Ofloxacin (5g), Ciprofloxacin (5g), Levofloxacin (5g) and Norfloxacin (10g).

The antibiotic discs were thoroughly placed on the surface of petri-dishes by sterile forceps and kept for 30 minutes at refrigerator to diffuse antibiotic in the media and then incubated at 37°C for 24hours. The diameter of the clear zones of inhibition was measured by using zone measuring ruler and recorded; the measurement was according to the standard tables given CLSI.

Multidrug Resistant Strains

According to CLSI guideline chart, Multidrug resistant *Pseudomonas aeruginosa* was identified by measuring the zone of inhibition for *Pseudomonas aeruginosa*. [22]The isolated strains that showed resistance against three or more antibiotics, considered as MDR *Pseudomonas aeruginosa*.

Results and Discussion

Pseudomonas aeruginosa is a most important agent causing nosocomial infections associated mainly with neutropenia, burns, ventilators and cystic fibrosis. Recent time, it is challenging for clinician to treat *P. aeruginosa* infection because there is frequently get resistant to many antibiotics which is lethal for patients lead to death. [23]The multidrug resistant strains of *P. aeruginosa* develop rapidly. For controlling it, we need to

use alternative method that inhibiting the QS in *P. aeruginosa* by enhancing the transcriptional regulator.

In the present study, total 1575 samples were collected from Clinical hospital laboratory at VAMC and RH during the year March 2019 to February 2020. On the basis of the growth characteristics on Peptone water, 525 samples (33.33 %) were observed for turbid growth with surface pellicle on Peptone water.

Out of 525 samples, 102 samples (19.43 %) were conformed as *P. aeruginosa* based on their growth characteristics [Fig. 1] and numerous biochemical tests were mentioned in [Table 1] and [Fig. 2].

The study report of 60 admitted patients at Vardhman Mahavir Medical College and Safdarjang hospital New Delhi in 2014[24] 56 *P. aeruginosa* isolated positive from 525 samples of Blood and wound swabs. Similarly, 495 *P. aeruginosa* isolated from 1548 clinical samples at Mangalore in 2002. [25]From numerous samples study from clinical hospital infections at a tertiary care hospital Andhra Pradesh reported 290 *P. aeruginosa* isolates in 2012.[26] A remarkable number of *P. aeruginosa* isolated at Bangalore in 2007 according to reference.[7] Reference[27] P. aeruginosa reported out of 85 patients in New Delhi, India.

In the present study, the total 102 isolated *P. aeruginosa* were subjected to the antimicrobial susceptibility testing with different types of antibiotics such as penicillin, β-Lactam/β-Lactamase Inhibitor Combinations, aminoglycosides, carba-penems, cephems and Fluoroquinolones. According to the CLSI standard guide line chart for enterobacteriaceae, each antibiotic was isolated as resistant or intermediate or sensitive to measure the diameter of zone of inhibition against the antibiotics [Table 2]. If the diameter of the zone of inhibition of ≤ 14mm for Piperacillin, ≤ 14 for Piperacillin-tazobactam, ≤ 12mm for Tobramycin, ≤ 15mm for Meropenem, ≤ 15 mm for Imipenem, ≤ 14mm for Ceftazidine, ≤ 14mm for Cefepime, ≤ 12mm for Oflaxacin and ≤ 15mm for Ciprofloxacin, ≤ 13 mm for Levofloxacin, ≤ 12 mm for Norfloxacin then the isolate was scrutinized as resistant.

Resistance of isolated *P. aeruginosa* with three or more antibiotics was assessed as multidrug resistant strains. Out of isolated MDR, 98% was noted resistant against Piperacillin and 75% sensitivite against Ceftazidine, Cefepime, 85% sensitivity against Pepracillin-tazobactam and 98% sensitivity noted against Imipenem, Meropenem. The more than three antibiotics resistant isolated and their zone of inhibition pattern was exhibited in [Fig. 3]. Ramana *et al.*, study in 2012[26] they found the highest number of infections in urinary catheters, followed by endotracheal tips and central venous catheters. The antibiotic resistance was maximum incefotaxime and gentamycin (40%) followed by ciprofloxacin (39%), amikacin (26%), cefoperazone- sulbactum (22%), piperacillin-tazobactum (16%), and imipenem (14%).

The studies of Shenoy *et al.,* in 2002 [29]They found that *P. aeruginosa* strains were highly resistant to most anti-pseudomonal antibiotics, 68.01% sensitivity of Amikacin was most suitable for routine use and Netilmicin was most resistant antibiotic showing 70.04% resistance but Gentamycin found a relatively higher sensitivity of 55.87%. Imipenem and Meropenem was 100% sensitivity. Swetha *et al.*, in 2017[28] was isolated 19 *P. aeruginosa* and studied the susceptibility against 12 antibiotics.

Table.1 Identification of *Pseudomonas aeruginosa* by various Biochemical test

S. No.	Name of the test	Observations
1	Gram's staining	Gram negative rod
2	Motility test	Positive
3	Catalase test	Positive
4	Oxidase test	Positive
5	Glucose fermentation	Negative
6	Sucrose fermentation	Negative
7	Lactose fermentation	Negative
8	Maltose fermentation	Negative
9	Mannitol fermentation	Negative
10	Acid from glucose	Positive
11	Gas from glucose	Negative
12	Triple sugar iron test	K/K
13	Inulin test	Negative
14	Indole production test	Negative
15	Citrate utilization test	Positive
16	Urease hydrolysis test	Negative
17	Methyl red test	Negative
18	Voges - Proskauer test	Negative
19	Nitrate reduction test	Positive
20	H2S Production	Negative

Table.2 CLSI guide lines table for determining the *Pseudomonas aeruginosa* as Resistant or Intermediate or Sensitive against various Antibiotics

Types	Antibiotics	Resistant	Intermediate	Sensitive
Penicillin	Piperacillin	≤ 14	15 – 20	≥ 21
β-Lactam/β-Lactamase inhibitor Combinations	Piperacillin-tazobactam	≤ 14	15 – 20	≥ 21
Aminoglycosides	Tobramycin	≤ 12	13 – 14	≥ 15
Carbapenems	Meropenem	≤ 15	16 – 18	≥ 19
	Imipenem	≤ 15	16 – 18	≥ 19
Cephems	Ceftazidine	≤ 14	15 – 17	≥ 18
	Cefepime	≤ 14	15 – 17	≥ 18
Fluoroquinolones	Oflaxacin	≤ 12	13 – 15	≥ 16
	Ciprofloxacin	≤ 15	16 – 20	≥ 21
	Levofloxacin	≤ 13	14 – 16	≥17
	Norfloxacin	≤ 12	13 – 16	≥17

Fig.1 Culture characterization of isolated *Pseudomonas aeruginosa* from samples was showed as medium-sized yellowish green colored colonies. The gram' staining showed gram-negative, rod shaped and pink colored bacteria.

Fig.2 Biochemical behavior of *Pseudomonas aeruginosa* that are Sugar fermentation tests for glucose, sucrose, lactose, maltose and mannitol, Triple Sugar Iron agar test, Urease test, Citrate utilization test, Nitrate reduction test, Indole production test, methyl red test, Voges-Proskauer test, Oxidase test and Catalase test.

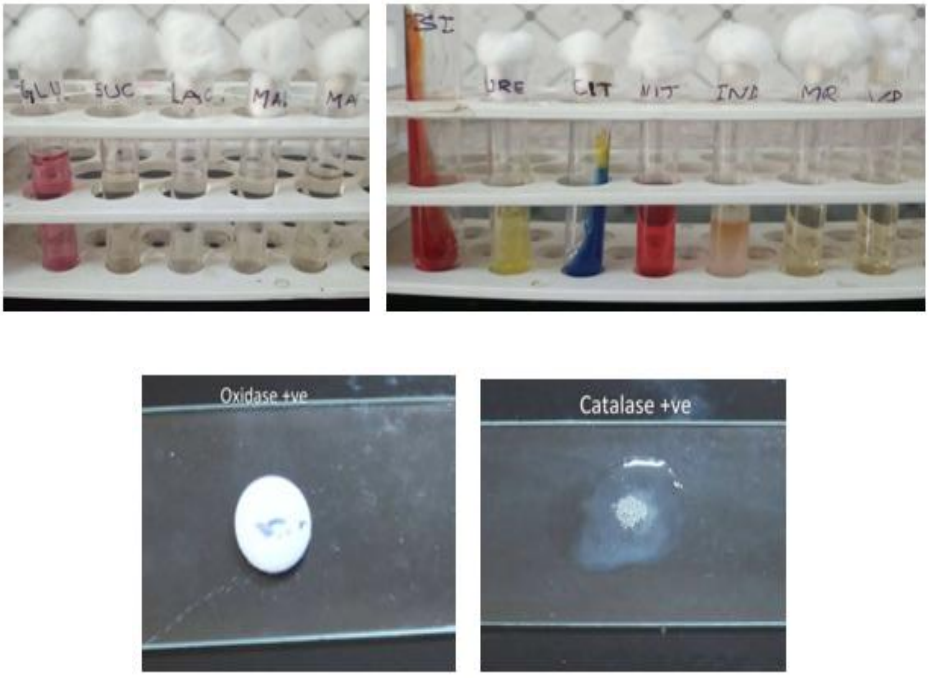
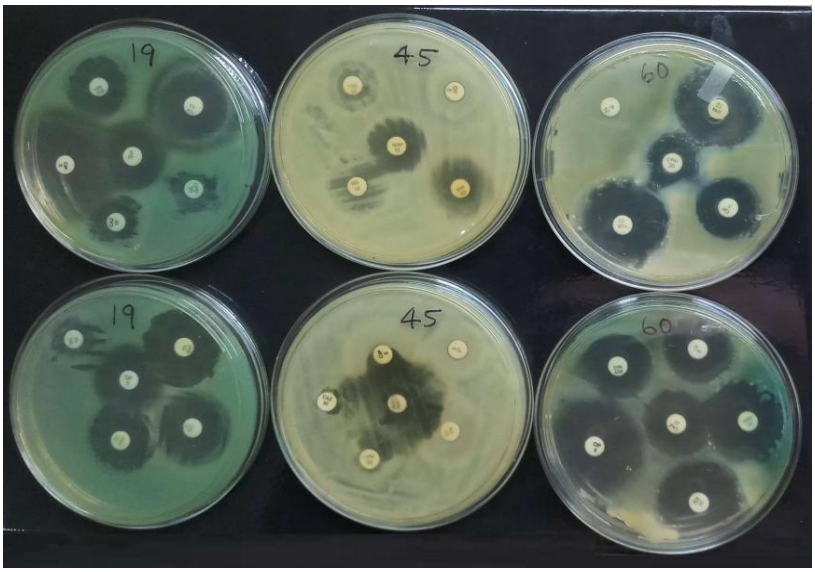


Fig.3 Antibiogram pattern of 3 isolated MDR *Pseudomonas aeruginosa* that are Isolate-19, Isolate-45 and Isolate-60



All the isolated multidrug resistant *P. aeruginosa* showed range of resistance from 36.84% to 100% of the 12 antibiotics tested. Resistance was reported against Penicillin (100%), Oxacillin (100%), Ampicillin (100%), Vancomycin (89.47%), Cotrimoxazole (48.65%), Tetracycline (78.94%), Chloramphenicol (57.89%), Erythromycin (52.63%), Azithromycin (52.63%), Streptomycin (47.36%), Gentamycin (42.10%) and Ciprofloxacin (36.84%)

The exposure of these drug resistant isolates is more challenging to clinical health concern. In this study, among isolates we found 51(50%) multidrug resistant strains show high level of infections of MDR *P. aeruginosa*. Over the past few decades, the extensive use of antibiotics leads to the increase in different multi drug resistant bacterial strains. Among isolated MDR strains, majority of strains showed resistance such as Piperacillin (98%), Ciprofloxacin (90%), Ofloxacin (90%), Levofloxacin(80%), Norfloxacin (60%) and Tobramycin (60%) and the lowest resistance was detected such as Ceftazidine, Cefepime, Piperacillin-tazobactam, Imipenem and Meropenem. Thus need regular study of MDR strain of *Pseudomonas aeruginosa* and its antibiotics sensitivity patterns for better treatment in future.

Pseudomonas aeruginosa is the most important agent causing nosocomial infections and it frequently develops resistance to the many antibiotics which is challenging for clinicians to treat so, this study desires to maintain proper hygienic condition of wards and ICU with proper hand hygiene maintains to prevent nosocomial infections of MDR strains of *P. aeruginosa*. The clinical samples were collected from VAMC and RH and processed in the laboratory andon the basis of their morphological characteristics and numerous biochemicals test *P. aeruginosa* were isolated**.** From these isolates MDR strains of *P. aeruginosa* identified according to antibiotic sensitivity pattern. These resistant strains indicate the improper use of antibiotics in the hospital. During study, 102 *P. aeruginosa* strains were isolated from the

clinical samples out of these the 51 MDR strains of *P. aeruginosa* were decleared on antibiogram pattern. We foundthe majority of strains showed resistance such as Piperacillin (98%), Ciprofloxacin (90%), Ofloxacin (90%), Levofloxacin (80%) and Tobramycin (60%). So, in this study the MDR *P. aeruginosa* isolated these strains act as source for exploration the QscR protein. For controlling MDR strains of *P. aeruginosa*, we need to use alternative method that inhibiting the QS in *P. aeruginosa* by enhancing the transcriptional regulator.

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