Original Article ISSN (O):2395-2822; ISSN (P):2395-2814

Identification and Characterization of Various Pseudomonas Species from Distinct Clinical Specimens: Antibiogram and Resistance Pattern.

Karan Bahadur Singh[1], Abdul Wahab Haji Dawood Mirza[2], Naikey Minarey[3], Namrata Shrivastava[4]

[1] Associate Professor, Department of Microbiology, Index Medical College Hospital & Research Centre, Index City, Nemawar Road, NH-59A, Indore, Madhya Pradesh 452016, India.

[2]Assistant Professor, Department of TB & Chest, Index Medical College Hospital & Research Centre, Index City, Nemawar Road, NH-59A, Indore, Madhya Pradesh 452016, India.

[3]Assistant Professor, Department of Paediatrics, Index Medical College Hospital & Research Centre, Index City, Nemawar Road, NH-59A, Indore, Madhya Pradesh 452016, India.

[4]Assistant Professor, Department of Obstetrics & Gynaecology, Index Medical College Hospital & Research Centre, Index City, Nemawar Road, NH-59A, Indore, Madhya Pradesh 452016, India.

Received: March 2019
Accepted: March 2019

ABSTRACT

Background: *P. aeruginosa* accounts for a significant proportion of nosocomial infections. This study was conducted to assess the prevalence, levels of antimicrobial susceptibility and resistance mechanisms of *Pseudomonas* from various clinical samples. P. aeruginosa accounts for a significant proportion of nosocomial infections. This study was conducted to assess the prevalence, levels of antimicrobial susceptibility and resistance mechanisms of Pseudomonas from various clinical samples. **Methods:** The study was conducted in a tertiary care hospital, over a period of 1 year. After identification of genus Pseudomonas, the speciation was done by biochemical tests and by VITEK 2. Antibiotic susceptibility was determined by disc diffusion method. Extended-spectrum β-lactamases (ESBLs) and metallo-β-lactamases (MBLs) production were detected by the combined disc diffusion test. **Results:** They were predominantly isolated from pus (44.3%), followed by blood (24.05%), body fluids (13.92%) and sputum (12.03%). The highest number of isolates were pseudomonas aeruginosa (64.56%) followed by p. fluroscence 19.62%, p. putida 7.6%, p. stutzeri 1.9%, p. alcaligens 1.9%, burkhelderia cepacia complex (BCC) (previous designation: pseudomonas cepacia) 1.9% and 2.53% isolate of burkhelderia pseudomallei (previous designation: pseudomona spseudomallei). **Conclusion**: This study examined the prevalence of pseudomonl infections, and its susceptibility patterns to different antibiotics. The presence of antibiotic-resistant P. aeruginosa isolates could be attributed to β-lactamase production and the use of multiple drug resistance efflux pump. It therefore calls for a very judicious, rational treatment regimens prescription by the physicians to limit the further spread of antimicrobial resistance among the P. aeruginosa strains.

Keywords: Pseudomonas, Pseudomonas aeruginosa, Burkholderia cepacia complex, Sphingomonas paucimobilis, Strenotrophomonas maltophilia, Multi drug resistance.

INTRODUCTION

Pseudomonas aeruginosa is an important opportunistic pathogen in hospital-acquired infections. As an environmental bacterium, it colonizes soil and water as well as hospital water supply and shares its natural habitat with free-living amebae. The Gram-negative bacterium Pseudomonas aeruginosa is a ubiquitous aerobe that is present in water, in soil and on plants. Moreover, P. aeruginosa can be frequently isolated from tap water in patient rooms.[1] However, clinical isolates of P . aeruginosa appear to be more resistant to amoebal ingestion than environmental isolates.[2] P . aeruginosa accounts for a significant proportion of nosocomial infections.[3] A general problem with nosocomial infections is the tendency of nosocomial pathogens to acquire new antibiotic resistance.[4] Multidrug-resistant (MDR) strains of P. aeruginosa are often isolated among patients suffering from nosocomial infections, particularly those in the intensive care unit (ICU).[5] Thus, infections caused by P. aeruginosa are particularly problematic because the organism is inherently resistant to many

Name & Address of Corresponding Author
Dr Abdul Wahab Haji Dawood Mirza
Assistant Professor,
Department of TB & Chest,
Index Medical College Hospital & Research Centre,
Index City, Nemawar Road, NH-59A,
Indore, Madhya Pradesh 452016,
India.

drug classes and is able to acquire resistance to all effective antimicrobial drugs.[6]

The isolation rate of Pseudomonas has been increasing recently in tertiary care hospital. Moreover, they pose a great threat to mankind as they are resistance to common antibiotics. These organisms are inherently resistance to many antibiotics by developing various efflux mechanisms and other methods. Pseudomonas species resistance to ampicillin, amoxicillin, amoxicillin-clavulanate, narrow spectrum and expanded-spectrum cephalosporin, cefotaxime and ceftriaxone and several efflux pump system.[7] However, due to unpredictable multidrug resistance patterns of clinical strains of Pseudomonas, it is imperative to know the institutional prevalent susceptibility profiles. Hence, this study was conducted to isolate the Pseudomonas species from various clinical samples by a simplified phenotypic identification protocol and to determine the antibiotic susceptibility pattern of these isolates.

MATERIALS AND METHODS

This was a prospective study. The study was conducted in the Microbiology Department of IMCHRC, Indore, over a period of 1 year (i.e. Jan 2018 to Dec 2018). A total 15,169 clinical samples of pus, blood, body fluid (pleural fluid, peritoneal fluid, synovial fluid etc.), urine, sputum, cerebral spinal fluid (CSF) & throat swab were carried out. The blood samples from the suspected patients of sepsis were collected in the adult and paediatric bottles of BACT/ALERT 3D system. The samples were taken from the suspected patients, admitted to different wards and various intensive care units (ICU) of this hospital. A detailed history was taken. The study was approved by the Institutional Ethical Committee. The statistical analysis was performed with the help of Microsoft EXCEL for WINDOWS 2017.

Samples were processed for culture by standard conventional methods. Genus Pseudomonas was identified by gram staining (gram negative bacilli), cell and colony morphology, pigment production positive catalase test, positive citrate test, triple sugar iron (alkaline slant/ no change butt), positive oxidase test and strongly motile by of motility test . Speciation of Pseudomonaswas performed on the basis of Hugh and Leifson oxidative-fermentative test (O-F) for glucose, sucrose, lactose, mannitol; gelatin liquefaction, beta haemolysis on blood agar media, nitrate reduction test, urease hydrolysis test (Christensen), Decarboxylation of Arginine, Lysin and Ornithine and growth at 35 ^{0}C and at 42^0 C for 18-24 hours on two tubes of trypticase soy agar (TSA). The final identification and confirmation was done by the Vitek 2 system.[8]

Antibiotic susceptibility testing was determined by Kirby - Bauer disc diffusion method: Muller-Hinton agar media was used.[8,9] Commercially available Himedia discs were used. The strength of the discs used and their zone size interpretation were carried out by National Committee for Clinical Laboratory Studies (NCCLS) guideline. The first line antibiotics, which were tested, Piperacillin (10mcg/disc), Carbenicillin, Cefotaxim (30mcg/disc), Ceftriaxone (30mcg/disc), Ceftazidime (30mcg/disc), Ciprofloxacin(5 mcq/disc) Gentamicin (10mcg/disc) Amikacin (30mcg/disc) and Imipenem (10mcg/disc). The second line antibiotics, which were tested, Tobramycin (10mcg/disc), Ofloxacin (5mcg/disc), Amoxicillin/Clavulanic acid (20/10mcg/disc), Piperacillin/Tazobactam (100/10mcg/disc), Tigecycline (15mcg/disc), Colistin (10mcg/disc) and Ertepenem (10mcg/disc).

Detection of multidrug drug resistance (MDR) strain

The isolates which were resistance to three or more than three groups of drugs were considered as MDR strain.[10] The groups of drugs we were tested are: penicillin (piperacillin, carbenicillin,), cephalosporin (cefotaxim, ceftriaxone, ceftazidime), aminoglycosides (gentamicin, amikacin, tobramycin), carbapenem (imipenem, ertepenem), fluroquinolones (ciprofloxacin, ofloxacin) and glycylcyclines (tigecycline).

Detection of Extended spectrum β-lactamases production.[9,11]

The Combine disk diffusion test (CDDT) was used to determine the prevalence of extended spectrum β-lactamases (ESBL) production. Muller-Hinton agar media was used. One Ceftazidime (CAZ) (30µg) disc was placed on a lawn culture of test isolates and at the distance of 15 mm on both side of CAZ disc, a combination disc of Ceftazidime/ Tazobactam (30/10 µg) and Ceftazidime / Clavulanic acid (30/10 µg) were placed. A≥ 5 mm increased in a zone diameter for either antimicrobial agent tested in combination with Clavulanic acid or Tazobactam versus the zone diameter of the agent when tested alone = ESBL producer.

Detection of metallo β-lactamases production

Muller-Hinton agar media was used. One Imipenem (10µg) disc was placed on a lawn culture of isolates and at the distance of 15 mm a combination disc of 10µg of Imipenem and 100µl of EDTA disc was placed. Then it was incubated at 35^{0}C for 18 - 24 hours. An increase in zone size ≥ 7 mm around the Imipenem -EDTA disc as compared to Imipenem disc alone was recorded as positive.[11]

RESULTS

In this study, out of 2439 clinical samples, total number of culture positive isolates were 769 (31.53 %) among which 326 (42.39%) were gram positive cocci and 443 (57.6%) were gram negative bacilli (GNB). Out of the total 769 isolates, 158 (20.55 %) were different species of pseudomonas.

Table 1: Distribution of different species of Pseudomonas in different clinical samples (n=158)

Name of the organism	Pus	Blood	Urine	Sputum	Body fluid	Total (n=158)	%
Pseudomonas aeruginosa	52	26	4	9	11	102	64.56%
Pseudomonas fluroscence	12	7	2	4	6	31	19.62%
Pseudomonas putida	3	2	2	2	3	12	7.6%
Pseudomonas stutzeri	1	1	1	0	0	3	1.9%
Pseudomonas alcaligens	1	1	0	1	0	3	1.9%
Burkholderia cepacia complex	0	0	0	1	2	3	1.9%
Burkholderia pseudomallei	1	1	0	2	0	4	2.53%
Total	70 (44.3%)	38 (24.05%)	9 (5.7%)	19 (12.03%)	22 (13.92%)	158(100%)	

Table 2: Diagnosis wise distribution of the Pseudomonas (n=158)

Name of the organism	SSI	Surface non healing ulcers	Burn	Sepsis	CSOM	RTI	CA	UTI	GIT	Total
Pseudomonas aeruginosa	39	1	1	3	12	29	4	6	1	96
Pseudomonas fluroscence	7	0	1	1	7	3	2	1	1	23
Pseudomonas putida	5	1	0	1	3	1	3	0	0	14
Pseudomonas stutzeri	4	0	0	0	3	2	2	0	1	12
Pseudomonas alcaligens	1	0	0	1	0	1	0	0	1	4
Burkholderia cepacia complex	0	0	0	1	0	0	1	1	0	3
Burkholderia pseudomallei	1	0	1	0	1	1	1	1	0	6
Total	57	2	3	7	26	37	13	9	4	158
%	36.08%	1.27%	1.9%	4.43%	16.55%	23.4%	8.23%	5.7%	2.53%	100%

[SSI= Surgical site infection, RTI= Respiratory tract infection, UTI= Urinary tract infection, GIT= Gastrointestinal tract infection]

Table 3: Risk factors wise distribution of the organisms (n=158)

Name of the organism	Diabetes	Hospital Acquired Infection	Chemo therapy due to malignancy	Indwelling Intra vascular catheters	Ortho pedic implant	Venti lation	Others	Total (%)
P. aeruginosa	33	12	19	11	11	25	13	124
P. fluroscence	17	2	2	2	0	4	3	30
P. putida	4	3	1	4	2	1	0	15
P. stutzeri	4	2	2	1	0	4	1	14
P. alcaligens	0	0	1	0	0	1	0	2
Burkholderia cepacia complex	1	0	2	0	0	0	0	3
B.pseudomallei	0	0	0	1	0	0	0	1
Total	43	32	7	16	12	23	25	158
%	27.22%	20.3%	4.43%	10.13%	7.6%	14.6%	15.8%	100%

Table 4: Antibiotic susceptibility pattern of Pseudomonas aeruginosa (n=102)

Drug	Cipro	Oflox	Genta	Amika	Imipe	Piper	Carbeni	Ceftaz	Tobra	Tige	Pip +Taz	Ertep
Sensitive	51.32	58.5	61.12	65.08	87.02	59.08	46.6	55.44	68.4	78.7	61.5	91.03
Resistant	48.68	41.5	38.88	34.92	12.98	40.92	53.4	44.56	31.6	21.3	38.5	8.97

[Piper- piperacillin, Carbeni- carbenicillin, Ceftaz- ceftazidime, Genta- gentamicin, Ami- amikacin, Tobra- tobramycin, Imipe- imipenem, Ertep -ertepenem, Cipro- ciprofloxacin, Oflox- ofloxacin, Tige- tigecycline]

Table 5: Antibiotic susceptibility pattern of Pseudomonas fluroscence (n=31)

Drug	Cipro	Oflox	Genta	Amika	Imipe	Piper	Carbeni	Ceftaz	Tobra	Tige	Pip +Taz	Ertep
Sensitive	47.8	55.8	69.4	62.4	89.6	76.8	57.6	54.7	68.4	82.5	69.5	93.5
Resistant	52.2	44.2	30.6	37.6	10.4	23.2	42.4	45.3	31.6	17.5	30.5	6.5

Table 6: Antibiotic susceptibility pattern of Pseudomonas putida (n=12)

Drug	Cipro	Oflox	Genta	Amika	Imipe	Piper	Carbeni	Ceftaz	Tobra	Tige	Pip +Taz	Ertep
Sensitive	59.8	51.4	61.4	70.8	90.03	52.8	44.6	56.9	61.6	67.4	58.8	90.8
Resistant	52.2	44.2	30.6	37.6	10.4	23.2	42.4	45.3	31.6	17.5	30.5	6.5

They were predominantly isolated from pus (44.3%), followed by blood (24.05%), body fluids (13.92%) and sputum (12.03%) [Table 1]. The highest number of isolates were pseudomonas aeruginosa (64.56%) followed by p. fluroscence 19.62%, p. putida 7.6%, p. stutzeri 1.9%, p. alcaligens 1.9%, burkhelderia cepacia complex (BCC) (previous designation: pseudomonas cepacia) 1.9% and 2.53% isolate of burkhelderia pseudomallei (previous designation: pseudomona spseudomallei) [Table 1].

Highest number of Pseudomonas species was isolated from surgical site infection (SSI) 36.08%, followed by respiratory tract infection (23.4%) and CSOM 16.55% [Table 2].

In this study 27.22% isolates were obtained from the patients who were suffering from Diabetes Mellitus. Around 20.3% isolates were obtained from the patients, who acquired infection during their hospital stay. About 14.6% of isolates were from those patients who on ventilator [Table 3].

[Table 4] shows Pseudomonas aeruginosa was having a good sensitivity to ertepenam (91.03%) followed by imipenem (87.02%), tigecycline (78.7%), tobramycin (68.4%) and amikacin (65.08%).

[Table 5] shows Pseudomonas fluroscence was having a good sensitivity to ertepenam (93.5%), followed by imipenem (89.6%), tigecycline (82.5%), pipercillin (76.8%) and gentamicin (69.4%).

[Table 6] shows Pseudomonas putida was having a good sensitivity to ertepenam (90.8%), followed by imipenem (90.03%), amikacin (70.8%), tigecycline (67.4%), and ceftazidime (61.6%).

DISCUSSION

Nabamita Chaudhury et al,[7] 2018 study showed a total of 15,169 clinical samples of pus, wound swab, different body fluid, blood, sputum and urine were carried out. Out of these total sample processed, 5096 (33.59 %) were culture positive. A total of 505 (15.9%) NFGNB were obtained from the culture positive samples. Pseudomonas aeruginosa is the most common isolate, accounting for 189 (61.56%), Pseudomonas fluroscence accounting for 42 (13.68%). Next to it was Pseudomonas putida 29 (9.44%), Pseudomonas stutzeri 21 (6.84%), Strenotrophomonas maltophilia 17 (5.54%), next to it were Burkholderia cepacia complex (BCC) and Sphingomonas paucimobilis accounting for 3 isolates (0.98%) and Pseudomonas alcaligens 2 (0.65%) respectively.[7] We have yielded also similar reports. Similar result were obtained by Patel P. H. et al in 2013, yield 76.97% Pseudomonas aeruginosa,which was the commonest one, followed by Pseudomonas species 0.54%, Strenotrophomonas maltophilia 0.2%, and Pseudomonas putida 0.8%.[12] Another similar study done by Memish Z.A. et al in 2012 yield 72.9% Pseudomonas aeruginosa, which was the commonest one, followed by Stenotrophomonas maltophilia 1.8%.[13]

Pseudomonas aeruginosais a pathogen associated with a wide range of nosocomial infection. This organism can cause disease in hospitalized patients, predominantly surgical site infection (SSI), pneumonia, septicemia, urinary tract infection, soft tissue infections, non-healing ulcers and chronic suppurative otitis media (CSOM).[14] Nabamita Chaudhury et al,[7] 2018 study majority of P. aeruginosa (42.32%) were isolated from pus and wound discharge and from different types of body fluid (55 isolates) like pleural fluid, peritoneal fluid, knee aspiration etc. Another previous study byand Yoshodhara et. al. isolated majority of the Pseudomonas aeruginosa from the pus.[16] In this present study 39.68 of Pseudomonas aeruginosawere encountered as Surgical site infection (SSI), followed by 21.16% isolates yield from respiratory tract infection.Moreover, around 11.11% isolates of P.aeruginosa were isolated from the blood cultures of the patients diagnosed with septicemia. The National Nosocomial Infection Surveillance system from 1986-2003 reported that Pseudomonas aeruginosais the second most common cause of pneumonia (18.1%), third most common cause of urimary tract infection (16.3%) and eight most frequently isolated pathogen from blood stream (3.4%).[15]

Vallés J et al 2004 identified 1,607 isolates pertaining to 35 different pulsotypes. Overall 54.2% of patients presented colonization, and tracheal colonization was present in 30.5%. Ten patients had colonization at intubation, and four of these developed ventilator-associated pneumonia (VAP) after a mean of 4+/-2 days. ICU-acquired colonization occurred in 31 patients, and 4 of these developed VAP after a median of 10+/-5 days. P. aeruginosa was isolated from the room's tap water in 62.4% of samples. More than 90% of tap water samples had pulsotypes 1 and 2, which were frequently isolated in the stomach (59%) but were only rarely associated with VAP.1

Tassios PT et al 1997 revealed 14 of the ICU outbreak isolates were indeed identical with respect to their serogroup, O:11, pyocin type, 10/a, and PFGE type, A. Clone A was endemic and dominant throughout the hospital, even though, within the ICU, it underwent phenotypic alterations, such as loss of cell wall lipopolysaccharide side-chains, or

acquisition of ceftazidime and imipenem resistance. Bacteriocin typing was more discriminatory than serotyping, but PFGE could differentiate further among phenotypically identical strains. It also allowed the tracking of an O:6 strain, as it was becoming gradually more resistant and undergoing a bacteriocin-type conversion while remaining genotypically unaltered.2 Gad GF et al 2007 showed that out of the 445 clinical specimens, 107 Pseudomonas strains (24%) and 81 Pseudomonas aeruginosa strains were isolated (18.2%). Out of the 200 environmental specimens, 57 Pseudomonas strains (28.5%) and 39 P. aeruginosa strains were isolated (19.5%). Amikacin was the most active drug against P. aeruginosa followed by meropenem, cefepime and fluoroquinolones. P. aeruginosa was highly resistant to all other antibiotics tested. The environmental isolates of P. aeruginosa exhibited higher antibiotic resistance than clinical isolates. Mechanisms of resistance used by P. aeruginosa included beta-lactamase production and multiple drug resistance efflux pumps. Our results showed that 29 (36%) of the clinical P. aeruginosa isolates and 37 (95%) of the environmental P. aeruginosa isolates were beta-lactamase producers. In addition, P. aeruginosa isolates effectively used an efflux-mediated mechanism of resistance against ciprofloxacin and meropenem, but not gentamicin or cefotaxime.[3]

Pseudomonas aeruginosa shows a good sensitivity to ertapenem (90.8%), imipenem (86.77%), tobramycin (66.66%) and amikacin (64.02%). This is almost similar to the study by Patel P.H. et al,who reported 94% sensitivity to this drug.12A study by Rit K et al reported that P.aeruginosa were highly susceptible to colistin (100%), imipenem (91.8%) and amikacin (69.3%).[16] Similarly In case of Pseudomonas fluroscence, ertapenam (95%)and imipenem (88%) show the highest sensitivity. Similarly, a study by Rit K et al reported 100 % sensitivity to imipenem.[16] Nabamita Chaudhury et al,[7] 2018 study revealed gentamicin and piperacillin each of them show 71.4% sensitivity. Unlikely, Rit K et al revealed a low sensitivity rate to gentamicin (33.33%).[16]

P. Yasodhara et al 1997 study showed antibiogram of 400 Pseudomonas species, 337 were P. aeruginosa and majority of these isolates were sensitive to imipenem 323 (95.86%) followed by amikacin 248 (73.59%). Marked resistance was observed to ampicillin 329 (97.62%), gentamicin 298 (88.42%) and nitrofurantoin 295 (87.53%).[17] In their study amikacin showed sensitivity against 77.19% isolates. Veenu et al. in another study found amikacin to be the most active drug against P. aeruginosa and gentamicin to be the least active drug.[18] Thomas SS et al,[19] 2016 showed the overall resistance pattern of all isolates reveal the most resistance to flouroquinolones with only 72% of the total isolates showing susceptibility to ciprofloxacin. This finding correlates with the study results of Senthamarai et al from Kanchipuram in South India,[20] who showed resistance rates of 64% with ciprofloxacin and Chaudhari et al who demonstrated least susceptibility to ciprofloxacin among their isolates.[21]

CONCLUSION

A large number of different species of Pseudomonasare isolated as primary pathogen from different clinical specimens of the patients, admitted in different wards and ICUs. Most of the patients had high risk factors, like prolonged hospitalization, immunocompromised due to chemotherapy, indwelling catheters and orthopedics implants in situ or other catheterization (urinary or intravenous), diabetics and burns. Pseudomonas aeruginosa is the commonest pathogen obtained from SSI. Most effective antibiotics are imipenem, amikacin, gentamicin, ciprofloxacin, cefotaxim and cotrimaxazole. Constant surveillance of antimicrobial resistance trends, administration of appropriate antibiotics, use of combination therapy and simple measures like hand washing have become quintessential for the control of this organism.

REFERENCES

1. Vallés J, Mariscal D, Cortés P, Coll P, Villagrá A, Díaz E, Artigas A, Rello J. Patterns of colonization by Pseudomonas aeruginosa in intubated patients: a 3-year prospective study of 1,607 isolates using pulsed-field gel electrophoresis with implications for prevention of ventilator-associated pneumonia. Intensive Care Med. 2004 Sep;30(9):1768-75.
2. Fenner L, Richet H, Raoult D, Papazian L, Martin C, La Scola B. Are clinical isolates of Pseudomonas aeruginosa more virulent than hospital environmental isolates in amebal co-culture test? Crit Care Med. 2006 Mar;34(3):823-8.
3. Gad GF, El-Domany RA, Zaki S, Ashour HM. Characterization of Pseudomonas aeruginosa isolated from clinical and environmental samplesin Minia, Egypt: prevalence, antibiogram and resistance mechanisms. J Antimicrob Chemother. 2007 Nov;60(5):1010-7.
4. Livrelli V, De Champs C, Di Martino P, Darfeuille-Michaud A, Forestier C, Joly B. Adhesive properties and antibiotic resistance of Klebsiella, Enterobacter, and Serratia clinicalisolates involved in nosocomial infections. J Clin Microbiol. 1996 Aug;34(8):1963-9.
5. Tassios PT, Gennimata V, Spaliara-Kalogeropoulou L, Kairis D, Koutsia C, Vatopoulos AC, Legakis NJ. Multiresistant Pseudomonas aeruginosa serogroup O:11 outbreak in an intensive care unit. Clin Microbiol Infect. 1997 Feb;3(6):621-628.
6. Gales AC, Jones RN, Turnidge J, Rennie R, Ramphal R. Characterization of Pseudomonas aeruginosa isolates: occurrence rates, antimicrobial susceptibility patterns, and molecular typing in the global SENTRY Antimicrobial Surveillance Program, 1997-1999. Clin Infect Dis. 2001 May 15;32 Suppl 2:S146-55.
7. Chaudhury N, Mirza S, Misra RN, Paul R, Chaudhuri SS, Sen S. Isolation and identification of various pseudomonas species from distinct clinical specimens and the study of their

antibiogram. Sch. J. App. Med. Sci., Dec, 2018; 6(12): 4964-4976.
8. Winn W Jr, Allen S, Janda W, Koneman E, Procop G, Schreckenberger P, et al, The Non-fermentative Gram - Negative Bacilli. Koneman's Color Atlas and Textbook of Diagnostic Microbiology, 2006; 305-91.
9. National Committee for Clinical Laboratory standards Methods for dilution antimicrobial susceptibility tests for bacterial that grow aerobically. Informational supplement M100-S23. National Committee for Clinical Laboratory Standards 2013.Wayne, Pa.
10. Edith BH, Deborah AH, Speert DP, Pseudomonas. In: Baron EJ, Jorgensen JH. Landry ML, Pfaller MA (editors). Manual of Clinical Microbiology. 9thed.Washington DC: American Society for Microbiology; 2007, p.734-48.
11. Chong Y, Lee K, Shin H, Kim Y, Yong D, Yum J. Modified Hodge and EDTA disk synergy test to screen metallo β-lactamases producing strains of Pseudomonas and Acinetobacter species. Society of Clinical Microbiology 2001; 7(2):88-91.
12. Patel PH, Pethani JD, Rathod SD, Chauhan B, Shah PD. Prevalence of non fermenting Gram negative bacilli infection in tertiary care Hospital in Ahmedabad, Gujarat. Indian Journal of Basic and Applied Medical Research 2013; 6(2):608-13.
13. Menmish ZA, Shibl AM, Kambal AM, Ohaly YA, Ishaq AM, Livermore DM. Antimicrobial resistance among non-fermenting Gram- negative bacteria in Saudi Arabia. Journal of Antimicrobial Chemotherapy 2012; 67(7): 1701-5.
14. Juyal D, Prakask R, Shanakarmarayan SA, Sharma M, Negi V, Sharma N. Prevalence of non-fermenting gram negative bacilli in vitro susceptibility pattern in a tertiary care hospital of Uttarakhand: A study from foothills of Himalayas. Soudi Journal of Heath Sciences 2013; 2(2):108-12.
15. Yashodhara P, Shyamala P. Identification and characterization of non-fermenters from clinical samples. Indian Journal of Medical Microbiology1997;15(4):195-97.
16. Rit K, Nag F, Rar HJ, Maity PK. Prevalence and susceptibility profiles of nonfermentative gram-negative bacilli infection in a tertiary care hospital of Eastern India. Indian J Clin Pract 2013; 24:451-5.
17. Yasodhara P, Shyamala S. Identification and characterization of nonfermenters from clinical specimens, Ind. J. Med. Microbiol.,15, 1997, 195-197.
18. Veenu, R. Sikka, and D.R. Arora, Isolation and susceptibility pattern of nonfermenting gram negative bacilli from clinical samples, Ind. J. Med. Microbiol.,17(1), 1998, 14-18.
19. Thomas SS, Sreenath K, Sebastian S. Characterization of the antibiotic profile of Pseudomonas aeruginosa isolates from a tertiary care center. Int J Res Med Sci 2016;4:571-4.
20. Senthamarai S, Kumar SRA, Sivasankari S, Anitha C, Somasunder V, Kumudhavathi MS. Resistance Pattern of Pseudomonas aeruginosa in a Tertiary Care Hospital of Kanchipuram, Tamilnadu, India. J of Clin and Diagn Res. 2014;8(5):DC30-DC32.
21. Chaudhari V, Gunjal S, Mehta M. Antibiotic resistance patterns of Pseudomonas aeruginosa in a tertiary care hospital in Central India. Int J Med Sci Public Health. 2013;2(2):386-9

How to cite this article: Singh KB, Mirza AWHD, Minarey N, Shrivastava N. Identification and Characterization of Various Pseudomonas Species from Distinct Clinical Specimens: Antibiogram and Resistance Pattern. Ann. Int. Med. Den. Res. 2019; 5(3):MB14-MB19.

Source of Support: Nil, **Conflict of Interest:** None declared