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ORIGINAL ARTICLE

Antimicrobial Resistance in Carbapenem Resistance Pseudomonas

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ABSTRACT

Aim: To figure out the antimicrobial sensitivity effect of multidrug resistant *Pseudomonas aeruginosa* obtained from several type of clinical specimens.
Study setting: Department of Microbiology and Resource laboratory, University of Health Sciences Lahore.
Methods: A sum total of 53 isolates of multi-resistant *Pseudomonas aeruginosa* were selected from Jinnah hospital Lahore during the period of 1st January 2016 to 2nd February 2017.
Nutrient agar slants were used for the transportation of resistant strains. In accordance with the CLSI manuals re-confirmation and processing of the strains were accomplished. The sub culturing and incubation was done on culture media such as MacConkey and blood agar at room temperature for 1 day. Standard confirmation of isolates under went by the graded morphological, cultural and biochemical techniques. In order to achieve this, Gram staining, culture media such as blood, oxidase test, motility test were executed.
Results: The resistance pattern of *Pseudomonas aeruginosa* against antibiotics was as follows: Meropenem 53(100%), 51(96%) to piperacillin–tazobactam, 49(92%) to ceftazidime, 43(81%) to amikacin, 41(77%) showed resistance to aztreonam, 48(91%) to quinolones as shown in figure. Almost all the *Pseudomonas aeruginosa* were resistant to aztreonam except for 23% (n=12 isolates). Colistin was predominant as the major strength of treatment for *Pseudomonas aeruginosa* with sensitivity of 48(91%).
Keywords: Disk-diffusion, Carbapenem, McFarland.

INTRODUCTION

Antimicrobial resistance emergence and spread continues to threaten our ability to treat common infections[1]. According to WHO, antimicrobials is one of the prominent global health threat imposed on humanity[2]. Recent issue of growing concern is the deadly spread of carbapenem resistant organisms also known as "superbugs"[3]. The cost of carbapenem resistant microbes to national economies and health system alters treatment of patients, lead to extensive hospital stays and need for more expensive, intensive treatment[4].

The objective of the study was to figure out the antimicrobial sensitivity effect of multidrug resistant *Pseudomonas aeruginosa* obtained from several type of clinical specimens.

METHODOLOGY

Drug susceptibility By Disk-diffusion: First of all bacterial growth was encouraged in TSB at 35^0 C for 1 day. After that, bacteria were added to emulsion sample in comparisons to 0.5 McFarland standards. With sterilized cotton swabs the antibiotic disks cropped up over the MH media for 1 day at 35^0C. Following growth period, length of the zone of inhibition were assessed. By Kirby-Bauer testing phenotypic characterization of isolates were determined. Susceptibility testing was executed as stated by standardized CLSI 2015 protocols in all the multidrug resistant isolates[5]. Pseudomonas aeruginosa ATCC27853 was used as control for the susceptibility tests. Chosen antibacterial for *Pseudomonas aeruginosa* amikacin (AK), ceftazidime (CAZ), ciprofloxacin (CIP), Meropenem (MEM), aztreonam (ATM) and piperacillin/tazobactam (TZP)[3,6]. The zone of complete inhibition of colistin for *Pseudomonas* species had also been performed by Kirby diffusion technique.

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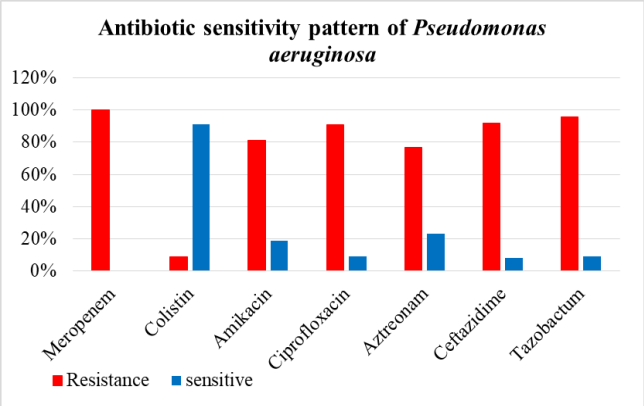
RESULTS

Antimicrobial susceptibility of clinical isolates. The isolates used in this study were ineffective to a minimum of three group of antibacterial like penicillins, cephalosporin, aminoglycosides, quinolones and carbapenems. All the isolates (n=53) were found to be resistant to carbapenem thus extensively drug resistant (XDR). Isolates of *Pseudomonas* were irresponsive to amikacin, ciprofloxacin, ceftazidime, piperacillin-tazobactam and Meropenem. The resistance pattern of *Pseudomonas aeruginosa* against antibiotics was as follows: Meropenem 100% (53 isolates), 96% (51 isolates) to piperacillin–tazobactam, 92% (49 isolates) to ceftazidime, 81% (43 isolates) to amikacin, 77% (41 isolates) exhibited defiance to aztreonam, 91% (48 isolates) to quinolones as depicted by the figure. A majority of *Pseudomonas aeruginosa* were irresponsive to aztreonam, excluding 23% 12 isolates a clear indication of presence of additional carbapenemase as MBL are particularly ineffective to aztreonam[7]. Colistin was predominant as the major strength of treatment for *Pseudomonas aeruginosa* with sensitivity of 48(91%). Except for the 5(9%) isolates expressing pan drug resistance, colistin succeeded as drug of choice amongst the Pseudomonas considered in our study as can be seen in chart I.

Figure I: Level of sensitivity pattern of *Pseudomonas aeruginosa* to various antibiotics.



DISCUSSION

High proportion of Pseudomonas were resistant to piperacillin/tazobactam, levofloxacin, aztreonam, ceftazidime, ciprofloxacin. One possibility against emergence of resistance towards antibiotics is if people do not change the way antibiotic are used therefore will lead to upcoming intrinsic resistance in *Pseudomonas aeruginosa* against the drugs[8]. Reportedly 19% amikacin sensitivity levels displayed on the other hand, this decreasing trend of amikacin is lowered than the conclusion drawn by Cavallo *et al in* 2007 and Hussain *et al* in 2015 who remarked 86% and 56% aminoglycoside susceptibility trends [9,10]. Currently overuse of aminoglycosides against gram negative bacteria in our hospital setups and dissemination of these pathogens by inappropriate hygienic measures is probably the reason[10]. We also concluded that aztreonam drug level in *Pseudomonas aeruginosa* was 88% in regulation with conclusive studies of Peymani *et al* in 2011 (95%), kalam *et al* in 2012 (92%), and Ameen *et al* in 2011 (86%) [11,12,13]. Resistance to all β–lactam drugs was imparted by MBL while excluding monobactams, such as aztreonam. However the aztreonam resistance observed in our study suggest some other modes of resistance such as concurrent presence of varied β-lactamases particularly the lactamases. Colistin remains as the mainstay of treatment for Pseudomonas with result shown up to of 91.8%. Seen in the studies of Jeya M *et al* in 2014 similar trends of 95% susceptibility towards Pseudomonas had been noted [14]. Whereas 5 (9%) of *Pseudomonas aeruginosa* exhibiting phenomenon of pan-drug resistance. These pan drug resistant strains are apparent because colistin will suffer the same fate as the previous antibiotics and become ineffective[16].

Moreover, the horizontal transfer of resistance gene through plasmids amongst microbes are the key against the acquirement of resistance to various drug under cosideration[20]. Carbapenem the best medication for multidrug resistant Gram-negative pathogens lead to it's over used in hospital ICUs contributing to increase carbapenem resistance[17].

Meropenem 53(100%), colistin 5(9%), amikacin 43(81%), ciprofloxacin 48(91%), aztreonam 41(77%), ceftazidime 49(92%), tazobactam 51(96%).

CONCLUSION

To conclude, Colistin is the only last resort treatment for life-threatening infections caused by carbapenem resistant Pseudomonas. This was also analyzed that bacteria resistant to colistin for which no effective antibacterial present currently**.**

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