ABSTRACT

The present study was carried out in order to detect the production of ESBLs in Pseudomonas aeruginosa and their susceptibility pattern against different antibiotics. A total of 100 isolates of P. aeruginosa were analyzed for the production of ESBLs by using phenotypic confirmatory test and the results were determined. The antibiotic susceptibility against various antibiotics was performed by Kirby Bauer Disc diffusion method on Mueller Hinton Agar. Out of the 100 isolates tested 20 isolates were found to be positive for the production of ESBLs, with a high prevalence of 36.3% found in the pus samples followed by the sputum 26%, blood 22.2% and urine 5.4% respectively. Imipenem was found to be the most effective antibiotic with cent percent susceptibility followed by Ofloxacin. A drug resistance of 57% for ceftazidime was noted against P. aeruginosa followed by cefotaxime. A high prevalence rate of P. aeruginosa was detected in males. This study showed the rising tendency of multidrug resistant strains in the hospital settings, which is a great concern for clinicians and patients to treat these resistant strains.

Keywords: P. aeruginosa, Drug resistance, ESBLs, β-lactams.

INTRODUCTION

One of the central mechanisms of resistance in gram-negative bacteria is the manufacturing of beta-lactamases, which hinder protein transpeptidases participating in creation of bacterial cell wall [1, 2]. Presently there are several such enzymes known but ESBLs and AmpC beta lactamas es are of particular clinical and epidemiological significance that are capable of inactivating the broad spectrum cephalosporins and penicillins. Pseudomonas aeruginosa is a primary cause of nosocomial infections, including pneumonia, urinary tract infections, and bacteremia. The infections can be predominantly brutal in patients with impaired immune systems, such neutropenic or cancer patients [3]. Extended spectrum β-lactamases (ESBLs) are enzymes that arbitrate resistance to extended spectrum cephalosporins (ESCs), such as cefotaxime (CTX), ceftriaxone, and ceftazidime (CAZ), and the monobactam aztreonam (ATM) [4]. Although modern advances in sanitation amenities and the introduction of wide range of antimicrobial agents with antipseudomonal activities, life threatening infections caused by this agent continue to cause devastations in the hospitals. The resistance in Pseudomonas aeruginosa is primarily mediated by β-Lactamases [5]. Although, the most important ones are metallo beta lactamases, but several studies indicates the presence of (ESBLs) in Pseudomonas as well [6, 7].

MATERIALS AND METHODS

A total of 225 different specimens were processed for the isolation of P. aeruginosa. Out of them 100 isolates of P. aeruginosa were isolated by using various conventional methods. The identification was based on the various biochemical tests and colony morphology of the organism [8]. The antibiotic susceptibility was performed against
different antibiotics including, cefotaxime (30 µg), ceftazidime (30µg), ofloxacin (5µg), gentamicin (10µg), amikacin (30µg), tobramycin (10µg), piperacillin (100 µg), piperacillin/tazobactum (100µg/10µg), meropenem (10µg) and imipenem (10µg) as per Kirby disc diffusion method [9]. Results were interpreted as per CLSI's latest guidelines [10]. All the isolates of \textit{P. aeruginosa} were tested for the production of ESBLs by using phenotypic confirmatory test [11]. A 0.5 McFarland’s suspension of every isolate was spread on a Mueller Hinton agar (MHA) plate (Hi-Media) and a 30µg disc of ceftazidime and ceftazidime/Clavulanic acid 30 µg/10µg discs were positioned aseptically on the agar plate. The two discs were kept apart at a distance of about 15mm and the plates were incubated at 37\textdegree C for overnight. The results were interpreted and a distance of ≥ 5mm increase in the zone diameter of the antimicrobial agent that was tested in combination with clavulanic acid, against its zone diameter when tested alone, confirmed the organism as ESBL producer [Figure 1].

![Figure 1: Shows detection of ESBL in \textit{P. aeruginosa}](image)

**RESULTS**

Out of 100 \textit{P. aeruginosa} isolates 20 were found to produce ESBLs. The maximum prevalence of ESBLs was set up in the isolates detected from the pus specimens (36.3%) while as low 5.4% was detected in urine [Table 1]. All the ESBL producing strains showed drug resistance to maximum number of antibiotics. Imipenem was found to be the drug of choice with 100% sensitivity followed by ofloxacin, while as highest resistance of 57% was noted against the ceftazidimine followed by cefotaxeim 51% [Table 2]. The majority of the isolates 76% were obtained from male patients.

<table>
<thead>
<tr>
<th>Sample</th>
<th>No. of isolates</th>
<th>No of ESBL strains</th>
<th>ESBL %age</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sputum</td>
<td>23</td>
<td>6</td>
<td>26%</td>
</tr>
<tr>
<td>Pus</td>
<td>22</td>
<td>8</td>
<td>36.3%</td>
</tr>
<tr>
<td>Blood</td>
<td>18</td>
<td>4</td>
<td>22.2%</td>
</tr>
<tr>
<td>Urine</td>
<td>37</td>
<td>2</td>
<td>5.4%</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>100</strong></td>
<td><strong>20</strong></td>
<td><strong>20</strong></td>
</tr>
</tbody>
</table>

**Table 2: Antibiotic resistance of \textit{P. aeruginosa} strains**

<table>
<thead>
<tr>
<th>Antibiotic</th>
<th>Resistance</th>
</tr>
</thead>
<tbody>
<tr>
<td>cefotaxime (30µg)</td>
<td>51%</td>
</tr>
<tr>
<td>ceftazidime (30µg)</td>
<td>57%</td>
</tr>
</tbody>
</table>

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DISCUSSION

Extended spectrum β-lactamases (ESBLs) are rapidly developing β-lactamases which are talented of conferring bacterial resistance to penicillins, first, second, third generation cephalosporins and aztreonam (but not the cephemycins or carbapenems) by hydrolysis of these antibiotics [12]. Clavulanic acid can be used as β-lactamase inhibitor to screen for ESBL production. Because of possibility of inoculum effect, their use for treatment of ESBL producing bacteria is not encouraged [13]. In the present study the prevalence of ESBLs producing P. aeruginosa was found to be 20% which is quite similar to the studies conducted [14, 15], who have observed 20.27% and 22.22% respectively however, one more study have reported a 64% incidence of ESBL producing P. aeruginosa [16]. Yet another study has noted a low ESBL production of 7.7% [17]. During the ongoing study, imipenem was found to be the drug of choice with 100% sensitivity which is similar to the studies reported [18, 19]. The current investigation has recorded a prevalence rate of 76% of P. aeruginosa in male patients which is comparable to the findings [20], who reported 78% isolates from the male patients in her study. However, [21] reported predominance of males as (68%). In our study ceftazidime showed a resistance rate of 57% which is related to the conclusion of [22] who showed a resistance rate of 55.4% while as another finding have recorded 53.17% [15], whereas one more study has noted 63% ceftazidime resistance [23]. The emergence of ESBLs and their wide-ranging spectrums and unrivalled drug resistance is creating a therapeutic challenge for clinicians and microbiologists throughout the world. Therefore, we put forward that the detection of ESBL in Pseudomonas aeruginosa must be a regular practice. We suggest a routine inspection on antibiotic resistance in the hospitals to curb the growing resistance.

CONCLUSION

The current study highlights that Pseudomonas aeruginosa relics a significant cause of nosocomial wound infections worldwide. The incidence of β-lactamase producing Pseudomonas aeruginosa is on the rise. Although this study underlines the exceptional problem of ESBL mediated resistance, which has produced a therapeutic confront for the clinicians and microbiologists. To conquer the problem of appearance and the stretch of multidrug resistant P. aeruginosa, a joint communication and collaboration among the microbiologists, clinicians and the infection control team is desired. We suggest the regular surveillance of antibiotic resistance inside and outside the hospitals.

REFERENCES


