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Guidelines to Authors
Dear editor,

Inactivation of β-lactam antibiotics by enzyme is a major mechanism of resistance in gram-negative bacteria. Although a variety of β-lactamases has been described, class A and C are the most important. *Pseudomonas aeruginosa*, one of the most common pathogens responsible for hospital infection, is intrinsically resistant to many antibiotics. It also shows an increasing pattern of resistance towards β-lactam antibiotics, especially by production of class C chromosomal β-lactamases.[1] Hence, this study was designed to determine the prevalence rate of inducible AmpC β-lactamase-producing *P. aeruginosa* in a tertiary care hospital in North India as well as to detect *in vitro* susceptibility pattern of antipseudomonal antibiotics.

In a duration of six months (November 2005 to April 2006), 162 consecutive non-repetitive isolates of *P. aeruginosa* were obtained from SS hospital, Banaras Hindu University (BHU), Varanasi, India. The sources of isolates were pus (92), swab (33), urine (32) and blood (5). An *in vitro* susceptibility pattern to common antipseudomonal antibiotics was also determined according to CLSI guidelines[2] for all the strains. Screening of AmpC β-lactamase was performed by disc antagonism test. A 0.5 McFarland of test isolate was spread over Mueller-Hinton agar (Hi-Media, Mumbai, India) plate, and cefotaxime (30 μg) and cefoxitin (30 μg) (Hi-Media) discs were placed 20 mm apart from centre to centre. The isolates that showed blunting of cefotaxime zone of inhibition adjacent to cefoxitin discs were considered screen positive and were selected for confirmation of inducible AmpC β-lactamase production by the modified three-dimensional test described previously.[3] *E. coli* ATCC 25922 was used as negative control.

Among the test isolates, 36 (22%) were suspected to be AmpC β-lactamase producers, which were further confirmed by modified three-dimensional test. Among positives, 34 were isolated from hospitalized patients, and two were from out-patients who attended the clinic. Antibiotic susceptibility testing showed piperacillin/tazobactam, imipenem and cefoperazone/sulbactam to be the most effective (Table).

β-Lactamase-producing bacteria can cause major therapeutic failure if they remain undetected. Although clinicians often treat infections based on the results of antibiotic susceptibility tests available, the number of infections caused by AmpC β-lactamase-producing organism, particularly *P. aeruginosa*, is on the rise and poses a threat to patients due to treatment failure.[4] This emphasizes the need for detection of isolates that produce this enzyme so as to avoid therapeutic failures and nosocomial outbreaks. It should also be mentioned that there is currently no clear consensus regarding guidelines for phenotypic screening or confirmatory tests for AmpC β-lactamase-producing organisms.[5] Although comparison between studies is difficult to do since the patient populations in these centers and methods of study differ, interestingly, we found a slightly high prevalence of AmpC β-lactamase-producing *P. aeruginosa* (22%) in our centre as compared to earlier studies in India. It was 17.3% in Kolkata,[4] whereas in a study conducted in Aligarh, it showed 20%.[3]

The referral hospital had always shown a high prevalence of *P. aeruginosa* infection in the recent past.[5] In our study, all the 34 AmpC-producing isolates from the patients admitted at different wards of the hospital could be clonal dissemination of the same β-lactamase gene, although genetic analyses have not been performed. Antibiogram pattern of these isolates showed that there were cross-resistance between aminoglycosides and quinolones. Although carbapenems remain the first choice for the treatment of patients infected with ESBLs or AmpC β-lactamase, our study shows that β-lactam/β-lactamase inhibitor (sulbactam or tazobactam) combinations can also be a good option.

<table>
<thead>
<tr>
<th>Table: <em>In vitro</em> susceptibility pattern of <em>P. aeruginosa</em> in SS Hospital, Banaras Hindu University</th>
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<tbody>
<tr>
<td>Antibiotics</td>
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<td>----------------------------------</td>
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<tr>
<td>Piperacillin/tazobactam</td>
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<tr>
<td>Imipenem</td>
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<td>Cefoperazone/sulbactam</td>
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<td>Tobramycin</td>
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<td>Netilmicin</td>
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n* - Number of susceptible isolates

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Parental History of Ulcer and the Prevalence of \textit{Helicobacter pylori} Infection in their Offspring

Dear editor,

\textit{Helicobacter pylori} infection is present in almost all patients with duodenal ulcers and gastric ulcers.\(^1\) The pathogenic role of \textit{H. pylori} in peptic ulcer disease is well known. Up to 95\% of patients with duodenal ulcers, and 80\% of patients with gastric ulcers suffer from this infection.\(^2\) The present study was carried out in the population of south India, which is considered the population at high risk of stomach cancer.\(^3\) We assessed the relationship between subjects with a history of gastric or duodenal ulcer and the risk of infection in their offsprings with the help of PCR assay targeting the 16S rRNA gene. The 16S rRNA gene is a highly specific target for amplification and has been previously of help in reclassifying the organism.\(^4\)

Another scientist demonstrated the specificity of unique \textit{H. pylori} gene primer in identifying the organism in paraffin-embedded gastric biopsy specimen.

The subjects referred to for upper gastrointestinal endoscopy at Deccan College of Medical Sciences and Research Center, Hyderabad, were interviewed about their mother or father had been referred for endoscopy with the same symptoms or any history of ulcer. The questionnaire sought details on risk factors for \textit{H. pylori} infection, such as housing conditions, family demographics and socioeconomic factors. By 16S rRNA amplification, the status of \textit{H. pylori} was confirmed. A total of 160 subjects were enrolled in the study, of which 70 subjects reported a parental history (mother or father) of ulcer, and 90 were without any history of ulcer. Of a total of 70 subjects, 14.2\% were \textit{H. pylori} negative and 85.7\% were \textit{H. pylori} positive (10 and 60, respectively). In those with no family history of ulcer, the prevalence of \textit{H. pylori} was 80\% and 20\% \textit{H. pylori} negative (72 and 18, respectively, of 90). The results propose the hypotheses that the transmission of \textit{H. pylori} may be influenced by the presence of ulcer or that \textit{H. pylori} strains causing peptic ulcer may be more infective than other strains as published in earlier studies.\(^5\) This may be because of the relation between a history of ulcer and \textit{H. pylori} infection in his or her family or due to common environmental or genetic factors that influence susceptibility to infection. In addition, the high prevalence of \textit{H. pylori} infection in subjects with no family history of ulcer suggests how the living conditions, socioeconomic factors and cultural background of the subjects are important in mounting the prevalence and transmission of \textit{H. pylori} infection.

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