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Efficacy of Imipenem for the Treatment of Bacteremia Due to an OXA-48-Producing Klebsiella pneumoniae Isolate

To the Editor—Resistance to carbapenems in Enterobacteriaceae is emerging worldwide [1, 2]. The enzymes that are responsible for the resistance are mostly clavulanic acid–inhibited carbapenemase (KPC), metallo-β-lactamases (Verona integron-related metallo-β-lactamase [VIM], IMP-type carbapenemases, New Delhi metallo-β-lactamase), and β-lactamases of the OXA-48 type [3]. The bacterial strains that produce these enzymes are multidrug-resistant [1, 3]. The efficacy of carbapenems for treating infections due to carbapenemase producers with low-level resistance or susceptibility to several carbapenem molecules remain debatable [4]. We report now a case of successful treatment with imipenem in bacteremia that was due to OXA-48-producing Klebsiella pneumoniae.

A 61-year-old female was admitted in December 2010 to the intensive care unit for septic shock syndrome and acute pancreatitis. On day 12, a rectal screening swab was positive for a multidrug-resistant K. pneumoniae isolate. At 24 days after her admission, the patient was febrile (39°C). A blood culture withdrawn from a catheter was positive for a K. pneumoniae with the same multidrug-resistant pattern. Ertapenem (1 gram every day) was administered before the results of identification and susceptibility testing. Both isolates were found to be clonally related or identical according to pulse-field gel analysis (data not shown), and of the sequence type 101 [5]. They were resistant to ertapenem (minimum inhibitory concentration [MIC] 4 mg/L) and susceptible to imipenem (MIC 0.38 mg/L) according to results of the Etest techniques following the updated Clinical and Laboratory Standards Institute guidelines [6]. They were resistant to expanded-spectrum cephalosporins (cefotaxime, cefazidime, and ceftriaxone), fluoroquinolones, cotrimoxazole, kanamycin, gentamicin, and tobramycin. A molecular-based analysis identified a narrow-spectrum β-lactamase TEM-1, a clavulanic-acid inhibited CTX-M-15, and the carbapenemase OXA-48 using described techniques.

Ertapenem was then replaced by imipenem (1 gram every 8 hours) over 12 days. Then, all blood cultures withdrawn during the imipenem treatment tested negative for K. pneumoniae.

This report underlines that laboratory detection of OXA-48 producers may be difficult. Indeed, the extended spectrum β-lactamase–producing strain isolated first from the rectal swab had not been detected as OXA-48-producing K. pneumoniae since it was susceptible to imipenem. Additional testing for ertapenem may be helpful for detecting this mechanism of resistance. In addition, this clinical case shows that certain carbapenem molecules could be used to treat infections with Enterobacteriaceae that produce OXA-48-type carbapenemases. In animal infection models, carbapenems are effective against VIM-producing K. pneumoniae with carbapenem MICs up to 4 mg/L. Results are similar with patients that are infected with VIM- or KPC-producing K. pneumoniae and are treated with carbapenems when the MIC is less than 4 mg/L [4]. This report may be interesting, especially in many countries where strains carrying OXA-48-type enzymes are spreading, such as those currently from the Mediterranean region and Europe [2, 7, 8].

Note

Potential conflicts of interest. All authors: No reported conflicts.

All authors have submitted the ICMJE Form for Disclosure of Potential Conflicts of Interest. Conflicts that the editors consider relevant to the content of the manuscript have been disclosed.

Anne-Claire Maherault,1 Patrice Nordmann,2 Audrey Therby,3 and Béatrice Pangon1

1 Service de Microbiologie, Centre Hospitalier de Versailles, Le Chesnay; 2Service de Bactériologie-Virologie, INSERM U914 Emergence Resistance to Antibiotics, Hôpital de Bicêtre, Assistance Publique/Hôpitaux de Paris, Faculté de Médecine et Université Paris-Sud, Kremlin-Bicêtre; and 3 Service de Médecine Interne et Maladies Infectieuses, Centre Hospitalier de Versailles, Le Chesnay, France

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Correspondence: Beatrice Pangon, PhD, Service de Microbiologie, Centre Hospitalier de Versailles, 78157 Le Chesnay, Cedex, France (bpangon@ch-versailles.fr).

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