Carbapenemase-producing Acinetobacter baumannii, Algeria

Drissi, M.; Poirel, L.; Mugnier, P. D.;Z. Baba Ahmed; Nordmann, P.

Abstract :

Acinetobacter baumannii is increasingly reported as a nosocomial pathogen [1]. Carbapenem resistance is now observed worldwide in A. baumannii isolates, leading to limited therapeutic options. Several mechanisms are responsible for resistance to carbapenems in A. baumannii [1].

The carbapenem-hydrolyzing β-lactamases in A. baumannii are either metallo-β-lactamases (MBLs) or oxacillinases (carbapenem-hydrolyzing class D β-lactamases [CHDLs]) [2]. Four major subgroups of acquired CHDLs have been identified in A. baumannii, being OXA-23, OXA-40, OXA-58, and OXA-143 β-lactamase groups in addition to the naturally-occurring OXA-51 ß-lactamase [2,3]. The blaOXA-58 gene has been identified worldwide, but mostly from Europe [3].

In this study, the molecular basis of the carbapenem resistance in Acinetobacter spp. was investigated in human isolates collected from April to November 2008 at the University Hospital of Tlemcen (Algeria). A total of 12 Acinetobacter spp. isolates have been recovered from seven different patients (urine, broncheal and rectal specimens) hospitalized at the intensive care unit (ICU). In addition, 4 isolates recovered from the environnemental screening performed in that ICU ward were included. MICs of ticarcillin, ticarcillin plus clavulanic acid (4 mg/L), piperacillin, piperacillin plus tazobactam (4 mg/L), ceftazidime, cefepime, aztreonam, imipenem, gentamicin, tobramycin, amikacin and ciprofloxacin were determined by the agar dilution technique in Mueller Hinton medium. Results were interpreted according to the guidelines of the Clinical and Laboratory Standards Institute [4].

All isolates were multidrug resistant, including resistance to the broad-spectrum cephalosporins, aztreonam, imipenem (MIC of 16 mg/L), fluoroquinolones, and aminoglycosides. All the 16 isolates studied here showed identical pulsed-field gel electrophoresis patterns, suggesting that they were closely related. The presence of blaOXA carbapenemase genes was searched by PCR using previously described primer pairs for the blaOXA-23, blaOXA-40, and blaOXA-58 genes, representative of the four OXA clusters [5]. All of these carbapenem-resistant strains were positive for the blaOXA-58 gene, suggesting that ß-lactamase OXA-58 significantly contributed to the imipenem resistance in these Acinetobacter strains. The blaOXA-58 amplicons were fully sequenced, demonstrating 100% identity to the blaOXA-58 sequence, although the OXA-58 point-mutant derivative OXA-97 had been recently identified in Tunisia [6]. Plasmid extraction followed by electro-transformation into an A. baumannii recipient strain as described revealed that the blaOXA-58 gene was located on a plasmid that transferred a decreased susceptibility to carbapenems but not to ceftazidime.

Genetic investigations showed that the blaOXA-58 gene was bracketed by insertion sequences (ISAba3-blaOXA-58-ISAba3), which were likely at the origin of its acquisition and expression [5]. The plasmid mediated blaOXA-58 gene described in this study could contribute to a rapid spread of carbapenem resistance in Acinetobacter in our hospital. This work constitutes the first evidence of an outbreak situation involving CHDL-producing A. baumannii strains in Algeria.

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