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Carbapenemase-producing Acinetobacter baumannii, Algeria

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Sir,

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 β -lactamase [2,3]. The bla_{OXA-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, 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 bla_{OXA-23}, bla_{OXA-40}, and bla_{OXA-58} genes, representative of the four OXA clusters [5]. All of these carbapenem-resistant strains were positive for the bla_{OXA-58} gene, suggesting that β lactamase OXA-58 significantly contributed to the imipenem resistance in these Acinetobacter strains. The bla_{OXA-58} amplicons were fully sequenced, demonstrating 100% identity to the *bla*_{OXA-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 bla_{OXA-58} gene was located on a plasmid that transferred a decreased susceptibility to carbapenems but not to ceftazidime. Genetic investigations showed that the bla_{OXA-58} gene was bracketed by insertion sequences (IS $Aba3-bla_{OXA-58}$ -ISAba3), which were likely at the origin of its acquisition and expression [5]. The plasmid mediated bla_{OXA-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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Transparency declaration

None to declare

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