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ePoster Viewing

MDR *Acinetobacter baumannii*

FIRST REPORT OF ISABA825 UPSTREAM OF BLAOXA-143 GENE IN CARBAPENEM-RESISTANT ACINETOBACTER BAUMANNII (ACB) CLINICAL ISOLATES

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Objectives: The aim of this study was to describe carbapenem-resistant Acb isolates producers of OXA-143, a carbapenem-hydrolyzing class D β -lactamase (CHDL), associated with the insertion sequence (IS) IS*Aba825* in Brazil. **Methods:** Three carbapenem-resistant *Acinetobacter* spp. isolates were recovered from distinct patients hospitalized at a tertiary teaching hospital located at São Paulo city, between the years 1997 and 1998. The identification of Acb isolates was confirmed by *rpoB* sequence. Susceptibility testing was performed by broth microdilution according to CLSI guidelines. The genetic relatedness was evaluated by Rep-PCR and automated ribotyping. The production of CHDLs and its association with IS*Aba1*, as well as other β -lactamases encoding genes was confirmed by PCR followed by DNA sequencing. Outer membrane proteins encoding genes *carO*, *ompW*, 33-36kDa and *oprD* were also evaluated by PCR and sequencing. **Results:** All isolates were identified as Acb and showed the same Rep-PCR pattern and ribogroup (52-1). As expected, these isolates possessed the intrinsic *bla*_{OXA-51} gene and *bla*_{TEM-1}. The IS*Aba1* was only observed upstream to the *bla*_{ADC} gene. A PCR amplification analysis of *bla*_{OXA-143-like} gene showed a single amplicon of nearly 1,822 bp instead of the expected fragment size of 889 bp. Sequencing analysis indicated the presence of an IS (876 bp), the IS*Aba825* (IS982 family), upstream of *bla*_{OXA-143} gene. All Acb isolates showing IS*Aba825*+*bla*_{OXA-143} were resistant to meropenem (MER; MICs, >8 mg/L), imipenem (IPM; MICs, 16 mg/L), levofloxacin (MICs, 16 mg/L), gentamicin (MICs, 16 mg/L), amikacin (MICs, 64 mg/L), ciprofloxacin (MICs, 128 mg/L), cefepime (MICs, 128 mg/L), ampicillin/sulbactam (MICs, 256 mg/L), cefotaxime (MICs, 256 mg/L), ceftriaxone (MICs, 256 mg/L) and ceftazidime (MICs, 256 mg/L). In opposite, these isolates were susceptible only to tigecycline (MICs, 0.125 mg/L) and polymyxin B (MICs, 0.25 mg/L). PCR analysis showed that the *oprD* gene was disrupted by an IS in all Acb isolates. **Conclusion:** To date, similarly what occurs with the OXA-24/40 group, the expression of OXA-143 has not been associated with the presence of IS. In fact, the role of IS for OXA-143 expression has been poorly studied. To our knowledge this is the first report of the presence of IS*Aba825* upstream of *bla*_{OXA-143} in a multidrug-resistant Acb isolates also showing the disruption of *oprD* gene. In the same way, the overexpression of ADC caused by IS*Aba1* contributed to the high MICs observed for the extended-spectrum cephalosporins among Brazilian OXA-143-producing Acb isolates. Although a clonal dissemination had been observed in our hospital between the years 1997 and 1998, none OXA-143-producing Acb isolate associated with IS*Aba825* had been observed subsequently, showing that this phenomenon rarely occur in this CHDL group.