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Molecular epidemiology and clonality of Acinetobacter spp in a Lebanese hospital over a period of one year

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Abstract

Introduction: The worldwide emergence of antimicrobial resistance in Acinetobacter spp and their clonal dissemination call for the investigation into Acinetobacter spp epidemiology.

Methodology: 100 nonrepetitive Acinetobacter spp isolates were recovered from patients admitted at Saint-George-Hospital-University-Medical-Center-Beirut, in a one-year period. Identification of the isolates was determined by the API20NE and confirmed by PCR amplification of blaOXA-51-like. Susceptibility to carbapenems and colistin were determined by the microdilution method and interpreted according to the CLSI, 2015. The β-lactamase inhibitors: PBA, EDTA, and Cloxacillin were used for the detection of KPC, MBL and AmpC, respectively. ESBL producers were detected whenever a keyhole effect was observed between 3rd generation cephalosporin and Augmentin®. Simplex PCR was conducted for the genotypic detection of β-lactamases. ERIC and 3LST-PCR were performed to determine the clonality of the isolates.

Results: Our findings showed that 84% were carbapenem resistant. Only one isolate was resistant to colistin. Phenotypically, 23 were ESBL, 15 KPC, 5 AmpC, and 4 MBL producers. PCR analysis showed that 99%, 93%, 77% and 3% of the isolates harbored blaOXA-51-like, blaADC, blaOXA-23-like, and blaOXA-40-like, respectively. ERIC-PCR analysis showed that A.baumannii isolates were clustered in 19 possibly related and 30 closely related subtypes. The 3-LST-PCR showed that 86.2% of the A.baumannii isolates pertained to the ICII (international clone II).

Conclusion: Our study showed a predominance of OXA-23-like producers and dissemination of ICII. Inhibitor based method was shown not to be accurate for the prediction of carbapenemases in Acinetobacter spp. Infection control measures are needed for management of Acinetobacter spp infections.

Key words: Acinetobacter spp; β-lactamases; ICII.


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