1. Abstract

Background: *Acinetobacter* spp. resistance towards *β*-lactam antibiotics is mediated mainly by different classes of *β*-lactamases production; detection of some genes responsible for production of *β*-lactamases is the objective of the study.

Methods: One hundred fifty bacterial isolates were recovered from blood, sputum, and urine specimens from different hospitals in Egypt. Sixty-nine isolate were identified as *Acinetobacter baumannii* using traditional biochemical tests, CHROM agar, MicroScan and PCR amplification of $bla_{oxa\text{-}51like}$ gene. *Acinetobacter baumannii* isolates were grouped into carbapenem resistant group (GP1), cefotaxime, ceftazidime and cefoxitin resistant group (GP2) and carbapenem and cephalosporin non-resistant group (GP3). Carbapenemase activity was screened using modified Hodge test (MHT) for GP1. Metallo-β-lactamases screening was performed for MHT positive isolates using double disk synergy test (DDST) and combined disk test (CDT). Amp C activity was screened using Amp C disk test with Tris-EDTA, DDST and CDT for GP2. Finally, PCR amplification of $bla_{oxa\text{-}51like}$, $bla_{oxa\text{-}23like}$, $bla_{IMP\text{-}like}$, $bla_{VIM\text{-}like}$, and $bla_{ADC\text{-}like}$ genes was performed for isolates that showed at least two positive results of three for both AmpC and carbapenemases phenotypic screening tests(obvious activity), in addition to GP3 (for comparison). Detection of $bla_{oxa\text{-}51like}$ and $bla_{ADC\text{-}like}$ genes preceded by *ISAba1* was also performed.

Results: Antibiogram of 69 pure *Acinetobacter baumannii* isolates resulted in 57, 64, and 2 isolates enrolled into GP1, GP2 and GP3, respectively. Carbapenemase activity was shown by 49(85.9%) isolate using MHT. Metallo-*β*-lactamases screening revealed 32(65.3%) and 35(71.4%) using DDST and CDT, respectively. AmpC activity was shown by 43(67.2%) and 50 (78.1%) isolates using AmpC disk test with Tris-EDTA, and both DDST and CDT, respectively. Twenty-seven isolates showed obvious activity, all of them (100%) were harboring $bla_{oxa\text{-}51like}$

and *bla*$_{ADC\text{-}like}$ genes, while *bla*$_{oxa\text{-}23like}$, *bla*$_{IMP\text{-}like}$ and *bla*$_{VIM\text{-}like}$ genes were harbored by 23(85.2%), 9 (33.%) and no isolate respectively. Only 12 (44.4%) isolates harbored *bla*$_{oxa\text{-}51like}$ and *bla*$_{ADC\text{-}like}$ genes preceded by *ISAba1*. GP3 isolates showed only positive *bla*$_{oxa\text{-}51like}$ and *bla*$_{ADC\text{-}like}$ genes.

Conclusion: It isn't possible to correlate resistance with presence of *bla*$_{oxa\text{-}51like}$ and *bla*$_{ADC\text{-}like}$ genes and presence of *ISAba1* was important as transcriptional promotor. A *bla*$_{oxa\text{-}23like}$ gene played an important role in carbapenem resistance when compared by bla$_{IMP\text{-}like}$ and *bla*$_{VIM\text{-}like}$ gene.