OLB07
2-hour Oral Session
Late breaker session: Colistin resistance

Colistin resistance can be unrevealed by semi-automated systems

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Background: Carbapenems represent the drug of choice for infections caused by multi-drug resistant (MDR) Gram negative bacteria, particularly *Pseudomonas aeruginosa, Acinetobacter baumannii* and *Klebsiella pneumoniae*. The worrisome increase of carbapenem resistant bacteria observed over the last two decades has led to consider other therapeutic options. In this context, colistin is being considered as the last resort therapeutic option to treat infections due to MDR bacteria. Colistin is an old antimicrobial compound, which acts by disrupting the bacterial membrane, resulting in cellular death. Increasing use of colistin revealed the emergence of colistin resistance around the world. This resistance not only results from chromosomal mutations, but recently also plasmid-mediated polymixin resistance has been described.

Material/methods: Three-hundred and eleven strains of carbapenem resistant *Klebsiella pneumoniae* (KPC) were isolated from various biological samples in the Microbiology Unit, Pisa, Italy during 2015. Samples were cultured on common isolation media and incubated at 37 °C. Suspected colonies were identified using a matrix-assisted laser desorption ionization–time of flight mass spectrometer (MALDI-TOF MS, Bruker Daltonics). Antimicrobial susceptibility tests were performed using the VITEK®2 system (Biomérieux) and the Phoenix™ system (Becton Dickinson). For confirmation, the Sensititre system (Thermo Fisher Scientific) was used. The Minimal Inhibitory Concentration results were interpreted using the EUCAST breakpoints (2014).

Results: Three-hundred and eleven strains of KPC were screened for colistin-resistance. One hundred and seven KPC strains resulted resistant to colistin using automated systems (VITEK®2 and Phoenix™) or broth microdilution assay. For 46 samples, mostly bronchoalveolar lavages, blood cultures, wound swabs and urine, the antimicrobial susceptibility test with automated system was flanked by broth microdilution assay. Thirty-seven colistin resistant strains were confirmed resistant by broth microdilution assay, while 9 strains interpreted as colistin susceptible with automated systems, resulted colistin resistant by broth microdilution assay.

Conclusions: Increased use of colistin as the last therapeutic option has immediately led to the spread of resistant strains. Considering the recently described plasmid-mediated resistance, which other microorganisms may acquire, the reliable detection of colistin resistance becomes mandatory. Our data on KPC strains showed an underestimation of the colistin resistance for 19% of samples using automated systems, thus leading to very major errors in clinical interpretation of antibiogram, which can cause therapeutic failure. Further analysis will define the most advisable diagnostic method for colistin resistance detection.