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Paper Poster Session

Emergence and worldwide outbreaks of carbapenemase-producing bacteria

Carbapenem-resistant *Klebsiella pneumoniae* carriage, colonization and infection in intensive care units in Catania, Italy

Antonella Agodi*¹, Martina Barchitta¹, Annalisa Quattrocchi¹, Andrea Maugeri¹, Maria Clara La Rosa¹, Celestino Bonura², Aurora Aleo³, Caterina Mammina³

¹*Department of Medical and Surgical Sciences and Advanced Technologies “Gf Ingrassia”, University of Catania, Italy, Catania, Italy*

²*Department of Sciences for Health Promotion « G. D’alessandro », University of Palermo, Palermo, Italy*

³*Department of Sciences for Health Promotion « G. D’alessandro », University of Palermo, Palermo, Italy, Palermo, Italy*

Background: Carbapenem-Resistant *Klebsiella pneumoniae* (CRKP) is becoming increasingly common in Europe, and especially in Italy as reported by the SPIN-UTI project (Agodi et al., 2015). Our study aimed to describe the epidemiological and molecular relationships of CRsKP in four Intensive Care Units (ICUs) in Catania.

Material/methods: From the 1st October 2014 to the 31st March 2015, in the framework of the SPIN-UTI project, all the consecutive isolates of *K. pneumoniae* from any site were collected. Resistance to carbapenems was defined according with the European Committee on Antimicrobial Susceptibility Testing (EUCAST) guidelines. Patterns of *K. pneumoniae* acquisition in ICU were carriage, colonization and infection. PCR screening for carbapenemase encoding gene of *bla*_{KPC}, for extended-spectrum β-lactamases encoding gene *bla*_{CTX-M} and for plasmid-mediated quinolone resistance (PMQR) genes *qnrB* and *aac(6′)-Ib-cr* was performed. Molecular typing of *K. pneumoniae* isolates by PFGE analysis of the XbaI-digested genomic DNA, according to the PulseNet protocol, and MLST analysis using the Institute Pasteur MLST scheme, were evaluated.

Results: A total of 123 *K. pneumoniae* were collected and 50 of them (41%), from 35 patients, were CRsKP. Using standard definitions, infection was associated to 39.5% of isolates, carriage to 31.6%, colonization to 18.4%, and colonization/infection to 10.5% of isolates. Among the resistance genetic determinants under screening, KPC3-type enzymes were detected in all *K. pneumoniae* isolates as a single determinant of β-lactam resistance (76.3%) or in association with CTX-M-15 (23.7%). Among the PMQR genes, *aac(6′)-Ib-cr* and *qnrB* were found in 78.9% and 42.1% isolates, respectively. PFGE analysis led to the identification of 6 different PFGE-types (named A to F). A major clone was identified, involving 51.47% of isolates, showing intra-ICU and inter-hospital spread. Besides, some clones spread within single ICUs. MLST analysis of representative isolates with same PFGE-type and resistance genetic determinants pattern, recognized 4 different STs: ST 258, ST 512, ST 35 and ST 307.

Conclusions: Our study indicated that multiple CRsKP clones were disseminated through different hospitals, suggesting clonal spread of some PFGE-types and STs and lateral transmission of genetic resistance determinants. These local findings suggest the need to extend the systematic surveillance of CRsKP acquisition at the nationwide level while implementing infection control strategies in order to evaluate their effectiveness in minimising the risk of transmission between ICUs and the spread of this dramatic antimicrobial resistance trait.

Collaborators: Anna Maria Longhitano, Carmelo Denaro, Giorgio Scrofani, Maria Antonietta Romeo - AO Cannizzaro, Catania, Italy; Anna Rita Mattaliano, Patrizia Bellocchi, Giacomo Castiglione, Anna

Elisa Marchese, Alida Imbriani - AOU "Policlinico-Vittorio Emanuele", Catania, Italy; Santa Adele Carini, Marinella Astuto, Giuseppa La Camera, Agata Sciacca - AOU "Policlinico-Vittorio Emanuele", Catania, Italy; Giuseppe Giammanco, Graziella Manciangli, Salvatore Coniglio, Savino Borracino, Gaetano Strano, Giuseppe Sciuto - ARNAS Garibaldi, Catania, Italy.