

TITLE: KPC-2-producing *Escherichia coli* causing pneumonia. Molecular characterization of isolates from patients admitted in a tertiary-care hospital in São José do Rio Preto, São Paulo, Brazil

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Hospital-acquired pneumonia (HAP) is a very common health-care associated infection, and a leading cause of death. In the last years, *Escherichia coli* emerged as a prominent agent of HAP, and in this regard, its ability to acquire genes codifying virulence and resistance to antibiotics is of clinical concern. Carbapenems have been widely prescribed for the empirical treatment of HAP; therefore, pneumonia by carbapenem-resistant bacteria represents a considerable challenge. Production of *Klebsiella pneumoniae* carbapenemase (KPC) is the main mechanism of resistance to carbapenems in Enterobacteriaceae. Around the globe, detection of KPC producing *E. coli* is increasing. At Hospital de Base de São José do Rio Preto – São Paulo, seven carbapenem resistant *E. coli* were identified as agents of HAP from January, 2014, to April, 2018. Isolates obtained from tracheal aspirate cultures were identified by an automated method and tested for antimicrobial susceptibility according to CLSI. The presence of genes codifying carbapenemase and virulence, as well as the phylogenetic groups was investigated by PCR. Clonal relatedness was accessed by *Xba*I-Pulsed-field gel electrophoresis (PFGE) and Multilocus Sequence Typing (MLST). The seven *E. coli* presented resistance to all tested beta-lactams and penicillin/beta-lactamase inhibitors. Also, 57.1% of the isolates were resistant to ciprofloxacin and 42.9% to gentamicin. All *E. coli* were susceptible to amikacin, fosfomycin, polymyxins B and E, and tigecycline. The *bla*_{KPC-2} gene was identified in all isolates. Six isolates were identified as phylogroup D and one as B2. The potential for pathogenicity was endorsed by the presence of genes for type 1 fimbriae and capsule's polysaccharide (*fimH* and *kpsMT II*) in all isolates. Furthermore, 71.5% presented *iutA* and *fyuA* genes, 28.5% presented *iroN*, and 14.3% presented *papEF*, *papG II*, *vat*, *hlyA* or *usp* as virulence determinants. According to PFGE, *E. coli* isolates are not clonally related. MLST showed that three isolates belong to the Sequence Type (ST) 648, two to ST38/CC38, and one to ST69/CC69 or ST95/CC95, all previously associated to extraintestinal infections. These preliminary show that KPC-2 producing clones of *E. coli* can cause HAP. Further surveillance of clinical cases and deep characterization of isolates in terms of pathogenicity and response to treatment will be important for infection control purposes and to improve knowledge about pneumonia caused by *E. coli*.

Keywords: *Escherichia coli*, KPC-2, pneumonia, virulence

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