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Paper Poster Session V

More and more third generation cephalosporin-resistent enteric bacteria everywhere?

High diversity of mechanisms of amoxicillin-clavulanate-resistance in *Klebsiella pneumoniae* and emergence of the OXA-1-beta-lactamase

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Objectives: In recent years we have observed at our institution an increase in the resistance to amoxicillin-clavulanate (AMC) among *K. pneumoniae* isolates that reached 37.0% in 2013 and 29.6% in 2014 (January-September).The aim of this study was to analyze the phenotypes of resistance to beta-lactams of all *K. pneumoniae* isolates resistant to AMC recovered in our institution over the period January 2013-September 2014, and to characterize the mechanisms of resistance to AMC. **Methods**: Over the period of study, a total of 3,769 *K. pneumoniae* isolates were recovered in our institution (1,931 in 2013, and 1,838 in 2014). Identification and susceptibility testing was performed by microdilution using commercialized ComboNeg MicroScan panels (Siemens). We analyzed all isolates in which the MIC of AMC was ≥16/8 mg/L. The characterization of the different mechanisms of resistance was determined phenotypically and by PCR. **Results**: A total of 1,269 isolates (33.66%) showed an AMC MIC of ≥16/8 mg/L. Among these, 752 (59.1%) were ESBL-producers, 260 (20.5%) were carbapenemase-producers (21 isolates were VIM-positive, and 239 were OXA-48-positive), and 171 (13.19%) were SHV-1-hyperproducers. Resistance to AMC among ESBL-producers suggested the presence of an additional AMC resistance mechanism. The remaining 86 isolates (6.8%), showed different phenotypic resistance profiles (PRP) that included the following: PRP1: only AMC (26 isolates); PRP2: AMC + cefazolin and /or cefuroxime (34 isolates); PRP3: AMC+ cefazolin and/or cefuroxime+ piperacillin/tazobactam (23 isolates); and PRP4: AMC+ cefazolin + cefuroxime+ piperacillin/tazobactam + cefepime (3 isolates). Among these, 27 isolates were OXA-1-producers (2.1%), and 59 (4.6%) were OXA-1-negative by PCR. Among the 59 isolates, none of them showed the plasmidic-AmpC phenotype, and 4 isolates tested by PCR were TEM-positive. The PRP of the 27 OXA-1-positive isolates were PRP1: 9 isolates; PRP2: 8 isolates; PRP3: 6 isolates; PRP4: 3 isolates; and other PRP: 1 isolate; and corresponded to 19 patients. Among the patients with OXA-1-producing *K. pneumoniae* isolates, 9 had community-acquired infections (9 urinary tract infections-UTI-), and 10 had nosocomial-acquired infections: UTI (3 patients), skin and soft-tissue (3), lower respiratory tract (2), and catheter-related bacteremia (2). **Conclusions**: We observed a high diversity in the mechanisms of resistance to AMC in *K. pneumoniae*. Although resistance to AMC was mainly present in isolates producing ESBLs and/or carbapenemases, we also observed the emergence and spread of the OXA-1-beta-lactamase among this species in the community and in the hospital that, to our knowledge, has previously only been described in *K. pneumoniae* sporadically. The high variability in the phenotypic resistance profiles of isolates producing the OXA-1-beta-lactamase, precludes the use of the antibiogram profile to predict the presence of this mechanism of resistance.