O097

2-hour Oral Session

Trends in antimicrobial resistance

Emergence of carbapenemase-producing Enterobacteriaceae in a general hospital in Spain: rapid spread of OXA-48 (2012-2014)

E. Cercenado[1], M. Marín[1], B. Padilla[1], M. Grande[1], T. Vicente[1], E. Bouza[1]

[1]*Hospital General Universitario Gregorio Marañón, MADRID, Spain*

Objectives: Carbapenemase-producing *Enterobacteriaceae* (CPE) are an increasing problem not only in healthcare institutions but also in the community. In our institution, the first CPE was detected in 2011 from a patient transferred from Greece that carried KPC-2-positive-*K. pneumoniae* and VIM-2-positive-*P. stuartii*. Continuous monitoring was started, and in 2011 two additional patients with CPE were detected (both with VIM-carbapenemases). In this study we analyze the emergence and spread of CPE in our institution over the last 3 years (January 2012-October 2014).

Methods: Over the period of study, all *Enterobacteriaceae* isolated in our laboratory from any source were subjected to identification and susceptibility testing by microdilution using commercialized ComboNeg MicroScan panels (Siemens). All isolates non-susceptible to carbapenems (ertapenem and/ or meropenem MIC >0.125 mg/l, and /or imipenem MIC >1 mg/l, EUCAST guidelines) were subjected to PCR for the detection of carbapenemase genes.

Results: A total of 388 CPE were detected from 148 patients (54.72% males) as follows: 8 isolates (6 patients) in 2012; 96 isolates (48 patients) in 2013; and 284 isolates (94 patients) in 2014. Among the CPE, 202 (52.06%) were isolated from clinical samples [110 (54.45%) urine; 42 (20.79%) wounds and abscesses; 24 (11.88%) blood; 13 (6.43%) respiratory samples; 13 other sources (6.43%)], and 186 (47.93%) from rectal swabs. The carbapenemases were: 313 OXA-48-like (80.67%), 61 VIM-like (15.72%), and 14 KPC-like (3.6%). The CPE species were *K. pneumoniae* (n=311; 80.15%), *Enterobacter cloacae* (n=19; 4.89%), *Enterobacter aerogenes* (n=19; 4.89%), *Escherichia coli* (n=14; 3.6%), *Serratia marcescens* (n=11; 2.8%), *K. oxytoca* (n=34; 2.3%), and *Citrobacter freundii* (n=5; 1.28%). The overall prevalence of CP-*K. pneumoniae* was 0.32% in 2012; 3.72% in 2013; and 12.67% in 2014, and was mainly due to OXA-48 (prevalence of 12.51% in 2014). The evolution of OXA-48, VIM, and KPC, over the study period was, respectively: 1.28% (n=5), 0.51% (n=2), and 0.25% (n=1), in 2012; 16.23% (n=63), 7.98% (n=31), and 0.51% (n=2), in 2013; and 63.14% (n=245), 7.21% (n=28), and 2.83% (n=11), in 2014. Among the 148 patients with CPE, 109 (73.64%) were infected and 39 (26.35%) were colonized. Infections/colonizations were nosocomially-acquired in 58.1% of patients, and community-acquired or transferred from other institutions in 41.89%. OXA-48 was more frequent in *K. pneumoniae* and in adults, and VIM was distributed in different species and was more frequent in children <1 year-old due to an outbreak in a neonatology unit (63.93%).

Conclusions: Over the last 3 years we have observed a rapid spread of CPE, mainly due to OXA-48-like-producing *K. pneumoniae* isolates that were disseminated in the community and among hospitalized patients, with a high rate of intercenter spread. Although still infrequent, OXA-48-producing *E. coli* is a cause of concern. VIM- and KPC-carbapenemases were less frequent, distributed in different species, and mainly hospital-acquired.