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Paper Poster Session
Emergence and worldwide outbreaks of carbapenemase-producing bacteria

Carbapenemase-producing Enterobacteriaceae and their clinical impact in an outbreak context in the university hospital of Angers

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Background: The spread of Carbapenemase-producing *Enterobacteriaceae* (CPE) is an emerging problem in France. In the University hospital of Angers, CPE are circulating since 2012, with two main outbreaks: one with OXA-48 producing *Klebsiella pneumoniae* and another one with KPC-producing *K. pneumoniae*. The objective here was to assess the characteristics of CPE carriage and their clinical impact on patients in our hospital.

Material/methods: From the 01/01/2012 to the 06/11/2015, all the patients hospitalized in the University hospital of Angers for whom a CPE was isolated were included in the study. Retrospectively, information such as the type of carbapenemase, the bacterial species producing the enzyme, and the anatomical sites corresponding to positive samples were recorded. Each patient was classified as “colonized” and/or “infected” by a CPE, according to the clinical and microbiological data. At last, deaths were recorded for infected patients.

Results: Overall, 72 patients have been labeled as CPE carriers during the period studied, with 62 patients positive with OXA-48, 9 with KPC and 1 with NDM. In these patients, 89 CPE were isolated (16 patients were colonized by more than one CPE). Bacterial species producing carbapenemases were distributed as follows: *K. pneumoniae* (68.5%), *E. coli* (18.0%), and other *Enterobacteriaceae* (13.5%). Sixty-two patients had a digestive colonization with CPE. Concurrently, the rectal screenings were negative for 5 patients who were colonized or infected in another site. For the 5 remaining patients, the digestive carriage had not been investigated. Sixteen patients were colonized in another site than the digestive tract (mainly urine and cutaneous samples). Within the 72 CPE carriers, 23 patients developed an infection with CPE: around one-third of patients with OXA-48-producing *Enterobacteriaceae* (N=19) and one-third of patients with KPC-producing *Enterobacteriaceae* (N=3). In 52% of the infected patients, the CPE infection occurred without any prior documentation of CPE carriage. Within the infected patients, 8 deaths (35%) were recorded during the infection period: 6 deaths were directly attributable to the CPE whereas 2 deaths occurred without direct link with the CPE. The proportion of deaths attributable to CPE in infected patients was similar for those infected with OXA-48 (5/19) and those infected with KPC (1/3).

Conclusions: A high rate (around a third) of CPE-carrier patients developed an infection. Among the infected patients, one quarter directly died as a consequence of this infection. This underlines the deep clinical impact of the CPE spread in our hospital. Furthermore, one half of the infected patients developed an infection without any prior information about CPE carriage, demonstrating the importance of the hidden transmission during this outbreak and the limits of the rectal CPE screening.

At last, no difference in the proportion of infection cases or mortality was observed between OXA-48 and KPC-carrier patients.