

**P2588 Molecular epidemiology of carbapenemase-producing *Enterobacteriaceae* at a United Kingdom teaching hospital, 2014-2018**

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**Background:** Carbapenem-producing *Enterobacteriaceae* (CPE) are a major clinical concern; they are difficult to treat, have a propensity to spread in healthcare settings, and cause significant morbidity and mortality.

**Materials/methods:** We conducted a prospective, observational cohort study of all inpatients with CPE isolated in screening and clinical samples from 1 October 2014 to 30 September 2018. Clinical and laboratory data were extracted from the hospital information system. Prior to June 2016, all patients with risk factors for CPE were screened, in accordance with national CPE guidelines. In addition, from June 2016 onwards, all patients admitted to the adult Intensive Care Unit and the Neurosciences Critical Care Unit were screened on admission, and weekly thereafter, during their critical care unit stay. Isolates were identified using conventional methods and underwent PCR testing for carbapenemase genes, prior to sequencing using the HiSeq and MINION platforms.

**Results:** CPE were isolated in 56 samples from 48 patients, most commonly from rectal swabs (n=42). Clinical infections were identified in 8 patients (16.7%), with a 30-day mortality of 25%. The median age was 63 years (range 9 months to 92 years). Multiple patient risk factors for CPE were identified. Patients tended to have multiple co-morbidities, with a median Charlson comorbidity index of 5, and 28 (58.3%) had been previously admitted to ICU. Broad-spectrum antibiotics were given to 38 (79.2%) patients in the preceding 3 months. Long-term immunosuppression was given to 8 (16.7%) patients and 25 (52.1%) had indwelling medical devices. *Klebsiella pneumoniae* and *Escherichia coli* were the most commonly isolated species. There were two outbreaks of bla<sub>NDM-1</sub> producing *K. pneumoniae* in June and December 2016. CPE isolates were often multidrug-resistant, but no colistin resistance was detected. Initially bla<sub>NDM-1</sub> was the most prevalent resistance mechanism, but bla<sub>OXA-48</sub> has become dominant since 2017. Genomic data will be presented.

**Conclusions:** We observed an increase in the incidence of CPE colonisation and infection between 2014 and 2018. The majority of patients had recognised risk factors for CPE and multiple co-morbidities. Infection with CPE was associated with significant mortality. The dominant resistance mechanism shifted from bla<sub>NDM-1</sub> to bla<sub>OXA-48</sub> over this time period.

