

O0566 **Successful control of a hospital-wide carbapenemase-producing *Serratia marcescens* outbreak related to contaminated hand basin waste-water systems**

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Background: *Serratia marcescens* is a common healthcare associated infection that is intrinsically resistant to colistin and is occasionally linked to environmental contamination. After the identification of a number of clinical (n=2) and faecal colonization (n=5) isolates of carbapenem-resistant *S. marcescens* producing *bla*_{IMP-4} (SM-IMP4), we investigated potential environmental reservoirs in our hospital.

Materials/methods: Following an initial broad-based assessment of potential reservoirs, the drains of clinical hand basins (CHBs) in key high-risk wards (ICU, neurosurgery, etc), then CHBs throughout all clinical wards, were cultured. Samples were collected with a flocced swab, which underwent broth culture amplification (tryptone soy broth) followed by culture on CHROMagar ESBL media. Potential ESBL-producing Gram-negative isolates were identified (MALDI-TOF) and underwent antibiotic susceptibility testing. Carbapenem-resistant isolates were assessed for carbapenemase genes (Carba-blue test and PCR; CPE) and by whole genome sequencing (WGS).

Results: A total of 400 CHBs on 22 wards were cultured, with 72/400 (18%) testing positive for CPE (70/72 [97%] SM-IMP4; 1/72 *K. pneumoniae*-IMP4; 1/72 SM-OXA1). Although these CPE were identified on 11/22 wards, most isolates were from the 3 wards (ICU, Neurosurgery, Spinal) with high rates of infection/colonisation where 58-72% of CHBs were positive for SM-IMP4 and CHBs were noted to be heavily choked with biofilm material. WGS confirmed that these environmental and clinical isolates were highly related. A detailed manual CHB cleaning program using routine dishwashing detergent to remove visible biofilm was conducted throughout the hospital with dramatic pre- vs post-cleaning (4-19 days later) reductions in CHB SM-IMP4 colonisation (e.g. ICU: 25/33 vs 10/33; Spinal: 10/17 vs 3/17), although aerosolization of SM-IMP4 during cleaning was detected, requiring careful barrier control measures. No further infections or faecal colonisations with SM-IMP4 were noted during 4 months follow-up.

Conclusions: We identified biofilm-laden CHBs to be a major reservoir for SM-IMP4 and to be tightly linked to patient cases of SM-IMP4 colonisation/infection. Simple, but detailed, cleaning resolved the

outbreak but education campaigns of healthcare workers to avoid using CHBs for disposal of biologic material and parenteral nutrition that encourage biofilm production are ongoing.