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P2374 Characterising a *Staphylococcus aureus* bacteraemia outbreak in a large UK tertiary hospital: a whole-genome sequencing based study

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Background: *Staphylococcus aureus* bacteraemia (SAB) causes severe morbidity and mortality. During 2017, the monthly rate of SAB at Brighton and Sussex University Hospital (BSUH) markedly exceeded the expected rate for three consecutive months. This study aims to investigate whether bacterial or host factors were associated with this outbreak.

Materials/methods: We conducted a retrospective cohort study of all SAB cases during the 3-month outbreak and those identified over a similar time period one year previously, when expected SAB rates were observed. Data were collected on patient demographics, risk factors, management and outcome. Speciation was determined using MALDI-TOF and antibiotic susceptibility by disk diffusion using BSAC breakpoints. Typing data (MLST sequence type (ST)), virulence-profiling (PVL, *tsst-1*, enterotoxins) and isolate relatedness (single nucleotide variants (SNV)) were determined through whole-genome sequencing (WGS).

Results: During the outbreak 26 SAB (from 26 patients) were identified and 17 SAB (from 17 patients) in the preceding year. There were no significant differences in age or gender. During the outbreak SAB were more often community-onset (11/17(65%) vs 23/26(88%), p=0.15) and affect intravenous drug users (IVDU) (0/17(0%) vs 8/26(31%), p=0.016). Prior to the outbreak SAB were more likely hospital-onset and affect patients with chronic renal failure (8/17(47%) vs 5/26(19%), p=0.08) receiving haemodialysis (4/17(24%) vs 1/26(4%), p=0.07). There were no differences in rates of metastatic infection or mortality. Methicillin resistance was comparable across time-periods (1/17(6%) vs 1/26(4%), p=1.0) as were other antibiotic susceptibilities. 34 isolates underwent WGS: 17 from 2016, 17 from 2017. During both time-periods 12 STs were identified. Median genetic diversity of isolates from 2016 was 21,872SNV (min 326, IQR10,230-24,472) and 2017 was 23,149SNV (min 72, IQR11,049-25,209, p=0.1). Genotypic analysis revealed Staphylococcal enterotoxin A (*sea*) was found more often in outbreak isolates (2/17(12%) vs 11/26(42%), p=0.04). No significant differences were found in other virulence genes.

Conclusions: The SAB outbreak at BSUH was associated with community-onset disease in IVDUs. Whilst the high genomic diversity of isolates does not support clonal expansion an over-representation of *sea* might represent a surrogate marker for mobile element acquisition. Further characterisation of genotypic and host factors are required to develop targeted methods to tackle SAB.