P1129 An outbreak of sepsis due to extensively drug-resistant Pseudomonas aeruginosa originating from an environmental source in an Italian haematologic unit

Anna Knezevich[1], Marina Busetti[1], Cristina Skert[2], Gianluca Festini[2], Marta Vidus[2], Roberto Luzzati*[3], Jacopo Monticelli[3], Lucia Pelusi[4], Alfredo Perulli[4], Rossana Piani[4], Daniela Monteverdi[4], Cristina Lagatolla[5], Raffaella Bressan[5]
[1] Microbiology Unit, ASUITS, , [2] Haematologic unit, ASUITS, , [3] Infectious Diseases ward, ASUITS, , [4] Medical Direction, ASUITS, , [5] Depatment of Life sciences, University of Trieste,

Background: Multidrug-resistant (MDR) or extensively-drug-resistant (XDR) Pseudomonas aeruginosa (PSA) strains infections are a major concern in nosocomial environments being associated both with worse outcomes and with high mortality rates especially in oncologic and haematogic settings.
In the Trieste University hospital, we experienced an outbreak of XDR-PSA bacteremia in the haematology unit probably caused by a reservoir in the toilets faucets.
Materials/methods: The haematology unit in Trieste hospital has twelve double-bed rooms and a protected area with three single-bed rooms reserved to patients undergoing marrow transplants and severe neutropenia (neutrophils <0.5 x10^3/µL). From August until September 2017 we found 5 patients with sepsis or septic-shock due to an XDR-PSA. The bacterial identification was performed by Vitek-2 (bioMérieux). Minimal inhibitory concentrations (MICs) were determined by a micro-dilution method (Sensititre Diagnostic System, Trek), and interpreted according to the EUCAST criteria. MIC for ceftolozane/tazobactam was determined by e-test. Genotyping to determine genetic relatedness between isolates was performed by analysis of pulsed-field gel electrophoresis (PFGE) profiles of chromosomal DNA digested with SpeI.
Results: All 5 consecutive strains of PSA from blood culture were resistant to piperacillin/tazobactam, ceftazidime, cefepime, ciprofloxacin, gentamicin, imipenem, meropenem and ceftolozane/tazobactam and susceptible to colistin and amikacin. None of these patients had any apparent source of bacteremia. Four out of five patients occupied the three isolation rooms. Two out of five patients had CVC-related sepsis and two died because of the XDR-PSA bacteremia (40% in-hospital mortality). No culture from rectal swabs and samples from ventilation filters was found positive for XDR-PSA which was instead found in the faucets of the toilets in the protected area. Pulse field analysis demonstrated that the strains both from blood cultures and from the toilets tabs were highly related suggesting that the outbreak was due to a single clone, probably originating from the tap water. The environmental disinfection of the ward succeeded in clearing the XDR-PSA strain and allowed to resume the regular transplant activity.
Conclusions: The investigation of this dramatic outbreak of XDR PSA highlights the potential role of environmental sources acting as a reservoir of MDR/XDR nosocomial pathogens.