Rapidly controlled outbreak of *Serratia marcescens* infection/colonisations in a neonatal intensive care unit, Pescara General Hospital, Pescara, Italy, April 2011

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Article published on 16 June 2011

In April 2011, an outbreak of *Serratia marcescens* infection/colonisations occurred in the neonatal intensive care unit of Pescara General Hospital. Rapid microbiological investigations lead to identification of five cases of likely cross-transmission from a neonate hospitalised for *S. marcescens* sepsis: four infections and one neonate colonised post-mortem. Two low birth weight neonates died. The environmental investigation detected *S. marcescens* from two soap dispensers. Strict hygiene measures lead to early interruption of the outbreak, without recurrences to date.

*Serratia marcescens* is an opportunistic pathogen able to rapidly spread in the nosocomial environment, being identified in up to 16% of nosocomial Gram-negative bloodstream infections [1]. Several outbreaks of *S. marcescens* in neonatal intensive care unit (NICU) were documented in recent years [2-6], causing potentially fatal sepsis, meningitis or pneumonitis in very premature or low birth weight neonates with mortality rates as high as 44%, significantly higher compared with those caused by *Enterobacter cloacae*, another well-known cause of disease and death in premature neonates [2,7].

**Outbreak description and control measures**

In early April 2011, a normal weight neonate was born at 41 weeks of gestational age and was transferred to NICU of Pescara General Hospital because of fever, failure to thrive and increasing C-reactive protein (CRP) values. Blood cultures grew *S. marcescens* fully susceptible to quinolones and carbapenems. The neonate was treated with antibiotics for 11 days and discharged after full recovery on day 11. Three days after, four of 16 neonates in the NICU developed a clinical picture suggestive of sepsis, with rising CRP values. All four were cared for in the same room. Aware of the recent case of *S. marcescens*, clinicians on duty asked for immediate testing and molecular characterisation by Septifast® (Roche) on blood samples which detected *S. marcescens* DNA from all four cases. Antibiotics were administered, based on susceptibility data from the possible source. Two of the four neonates died, after 20 and 22 days of hospitalisation. Both had been premature births (week 26 and 29) with birth weight <1,200 g. Due to respiratory distress at birth, they had been transferred to NICU directly from delivery rooms, 10 and four days before becoming symptomatic. The other two septic neonates were born at 31 and 38 weeks of gestational age, one with low and one with normal birth weight. They had been delivered through Caesarian section and transferred to NICU for respiratory distress, nine and five days before becoming septic. They were discharged in good condition after 49 and 25 days of hospitalisation respectively, after their CRP values were normal.

A fifth very low birth weight (<800 g) neonate, cared for in the same room, had died the day before the four neonates became septic. Post-mortem sampling from his umbilical cord catheter grew *S. marcescens*, suggestive of colonisation.

**Environmental sampling**

On the day after the four neonates became symptomatic, extensive environmental microbiological investigations were started. Swabs were taken from numerous surfaces, including walls, floors with their edges and corners, doors and door handles, shelves, benches, hoods, sinks, cradles and ventilators, stethoscopes and other personal medical devices, milk
neonates with clinical signs of sepsis were successfully infected and one colonised post-mortem. Two were hospitalised for the infection and so far no recurrence. In recent NICU reports, greater numbers of patients were involved, the fraction of infected/colonised neonates among all neonates present in the wards higher, and environmental colonisation more protracted, with more than one epidemic peak; mortality rates were however comparable.

For this reason, molecular cluster analysis of the eight available isolates (six from the neonates and two from the soap dispensers) was not requested. Fortunately, this outbreak was limited to a small number of cases, with a single epidemic peak of colonisation/infection and so far no recurrence. In recent NICU reports, greater numbers of patients were involved, the fraction of infected/colonised neonates among all neonates present in the wards higher, and environmental colonisation more protracted, with more than one epidemic peak; mortality rates were however comparable.

Prompt clinical suspicion of *S. marcescens* and the immediate use of a molecular assay allowed for early etiological diagnosis, supported by an epidemiological link with a recent case and paved the way to immediate measures for containment of nosocomial infections. These measures adopted before any evidence from traditional microbiological cultures, enabled the rapid interruption of the outbreak. Moreover, this report serves as a reminder of the importance to keep up hygiene precautions at any time specifically in high risk settings such as a NICU.

*Erratum:* At the moment of publication, the names of V Cortesi and V Fortunato were left out in the list of authors. This mistake was corrected on 21 June 2011. We apologise to the authors.


