

***S.aureus* with Panton-Valentine leukocidin gene among two families in Milan**

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Background

Panton-Valentine leukocidin (PVL) is a cytotoxin produced by *Staphylococcus aureus* that causes leukocyte destruction and tissue necrosis.

Although produced by <5% of *S.aureus* strains, the toxin is detected in large percentages of isolates that cause necrotic skin lesions and severe necrotizing pneumonia. We describe a case of cutaneous infections caused by PVL-producing methicillin resistant *S.aureus* (MRSA) that affected two related families.

Material/methods

From December 2014 recurrent tissue abscesses occurred in 2 families: family A (parents and 2 children; 5 years and 7 months), family B (parents and 2 children, 2 years and 1 months). Families attended the same recreation areas and the two mothers given birth to the younger sons in the same delivery room in a hospital in Milan (sixth month apart).

As reported by the physicians, the recurrent tissue abscesses started with the mothers in both the families.

On October and November 2015 wound swabs from active lesions available and nasal swabs from family A and B respectively, were processed at the Laboratory of Clinical Microbiology, Virology and Bioemergencies of the University Hospital "L. Sacco" in Milan, Italy. Moreover nasal swabs from relatives attending the two families were investigated. Isolates from clinical samples were identified and tested for antimicrobial susceptibility (Vitek.2 system BioMérieux, Marcy l'Etoile, France). To assess methicillin resistance the

interpretation was performed on the oxacillin susceptibility according to the European Committee on Antimicrobial Susceptibility Testing (EUCAST) breakpoints.

S.aureus isolates were tested with the RealCycler SAMAPV (Progenie Molecular, Valencia, Spain) to confirm the presence of the *mecA* gene and to evaluate the presence of the Panton Valentine leukocidin (PVL) gene.

Results

Eight out of 15 clinical samples tested were positive for *S.aureus*: 7 were MRSA-PVL positives, 1 was methicillin susceptible (MSSA) PVL negative.

Family A: lesions swabs of the parents and the nasal swab of the youngest child were MRSA-PVL positives, nasal swab of the oldest child was MSSA-PVL negative.

Family B: nasal swabs of the parents and the wound swab of the youngest child were MRSA-PVL positives, nasal swab of the oldest child resulted negative.

None of the nasal swabs of the relatives tested were positive for *S.aureus*.

Patients with tissue abscesses were treated with antibiotics according to the susceptibility tests; nasal carriers were treated with chlorhexidine and neomycin.

Conclusions

The exact route of transmission between the two families was not identified.

As for the intrafamiliar transmission we presume that the close contact leads to skin or nasal colonization since only the youngest children of the families (< 1years), in close contact with their mothers (breastfeeding) and the husbands were MRSA-PVL positives.

Further molecular investigations are required in order to genotype the MRSA-PVL positive strains to assess the transmission among the two families.