INTRODUCTION
Panton-Valentine leukocidin (PVL) is a pore-forming cytotoxin produced by some clones of Staphylococcus aureus that causes leukocyte destruction and tissue necrosis (1). It is associated with infections ranging from uncomplicated skin and soft tissue infections to life-threatening necrotizing pneumonia (2). We describe an outbreak of recurrent cutaneous infections caused by PVL-producing methicillin resistant S. aureus (MRSA) that affected 6 out of 8 members of 2 related families.

MATERIALS AND METHODS
From December 2014 recurrent cutaneous abscesses occurred in 6 of 8 members of 2 families (Fig.1). Family A: parents and 5 years and 7 months old children, family B: parents and 2 years and 1 month old children, who shared the same recreation area and 1 infant from each family was born in the same delivery room 2 and 8 months earlier. The events started with the mother of both families.

In October and November 2015 wound swabs from available active lesions (n=2) and nasal swabs from all members (n=6) of the two families, were processed at the Laboratory of Clinical Microbiology, Virology and Bioemergencies of the University Hospital “L. Sacco” in Milan, Italy. Nasal swabs from relatives (n=5) frequently spending time with the two families were also investigated. Isolates from clinical samples were identified and tested for antimicrobial susceptibility (Vitek.2 system BioMérieux, Marcy l’Etoile, France).

Methicillin resistance was assessed using the oxacillin susceptibility according to the European Committee on Antimicrobial Susceptibility Testing (EUCAST) breakpoints. S. aureus isolates were tested with the Real Time PCR RealCycler SAMAPV (Progenie Molecular, Valencia, Spain) to confirm the presence of the mecA gene and to evaluate the presence of the Panton-Valentine leukocidin gene.

RESULTS
Nine out of 15 clinical samples tested were positive for S. aureus: 8 were MRSA-PVL positive and 1 was methicillin susceptible (MSSA) but PVL negative. In particular (Tab.1):

Family A: the lesions, the nasal swabs of both parents and the nasal swab of the younger child were MRSA-PVL positive. The nasal swab of the older son was MSSA-PVL negative.

Family B: the nasal swabs of both parents and of the younger child were MRSA-PVL positives whereas the nasal swab of the older son resulted negative.

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

Two adults with cutaneous abscesses were treated with antibiotics according to susceptibility tests; afterwards, the families were advised to apply neomycin plus chlorhexidine nasal ointment 4 times a day for 10 days (the strains were resistant to mupirocin) and to bathe with 4% chlorhexidine scrub for 1 week.

CONCLUSIONS
The presence of PVL-producing S. aureus clones in nasal cultures in 6 of the 8 members of the 2 families suggests that, in this niche, they were able to persist and cause recurrent infections in a large number of family members. Our report highlights the high intrafamiliar transmissibility of PVL-producing S. aureus clone, its high attack rate, and its virulence. Further molecular investigations are required in order to genotype the MRSA-PVL positive strains to assess the transmission among the 2 families.

Tab.1 Microbiologic data collected form Oct. to Nov. 2015 among the two families. MRSA: methicillin resistant S. aureus; MSSA: methicillin susceptible S. aureus; PVL: Panton-Valentine leukocidin; NT: not tested

REFERENCES