Pediatric case of fatal necrotizing pneumonia due to Panton-Valentine leukocidin-positive methicillin-resistant *Staphylococcus aureus* in Spain

Caso pediátrico de neumonía necrotizante fatal debida a Staphylococcus aureus *productor de Leucocidina de Panton-Valentine en España*

Methicillin-resistant *Staphylococcus aureus* (MRSA) is a leading cause of hospital-acquired and health care-associated infection worldwide. However, over recent decades, MRSA epidemiology has changed causing infections in the healthy community.[1] Production of Panton-Valentine leukocidin (PVL), a cytotoxin that causes leukocyte destruction and tissue necrosis in these strains is considered a virulence factor. PVL genes are consistently associated with skin and soft-tissue infections[2] in immunocompetent young patients although its role in invasive disease with poor prognosis is controversial.[2–4] PVL toxin can be produced by both methicillin-susceptible *S. aureus* (MSSA) and MRSA strains and is associated with more severe infections, regardless of methicillin resistance.[3] We report the case of a 12-years old, previously healthy girl born in Venezuela, triplet from a multiple gestation with two healthy sisters and family with low socioeconomic status. She had been living in Spain for the last seven months.

The patient started with progressive worsening flu-like symptoms, high fever and cough that progressed to haemoptysis in less than 24 h presenting sudden death at her home while sleeping. The autopsy established multiple organ failure after bilateral abscessed pneumonia as a cause of death, with pleurisy fibrinopurulent and bilateral adrenal hemorrhage. Blood culture, pleural fluid, cerebrospinal fluid and tissue biopsy of lungs, spleen, adrenal and pharynx were collected and sent for microbiology cultures postmortem. MRSA, also resistant to clindamycin and erythromycin, was isolated in all samples. Typing of MRSA strains was performed by phage typing and detection of PVL genes by the polymerase chain reaction technique, with the following results: fagogroup III, phage types 84 and 85, with positive PVL and *mecA* genes. All strains isolated showed the same pulsed-field gel electrophoretic pattern.

The risk of infection and/or colonization of the adolescent based on any current skin infections or previous healthcare contact during the previous year of the patient or her relatives were evaluated with no findings.

The status of colonization of the patient's cohabitants was studied. Screening was performed on 4 family members (mother, 2 sisters and brother) and 2 roommates originating from Ecuador. Screening included a swab of any suspicious lesion, nose, throat, perineum and inguinal area. Contacts underwent a five-day decolonization treatment including an intranasal application of mupirocin ointment three times a day, gargle with an antiseptic solution and chlorhexidine 4% as liquid soap in place of body wash and shampoo.[5] A sister of the deceased was found to be carrying MRSA in mucous, which was negative for the PVL gene. New decolonization-treatment was not indicated as the efficacy of decolonization in patients with community-associated MRSA has not been established by any data and the possibility of relapsing following decolonization treatment has been described.[6] Additional measures to reduce the risk of cross-infection within household as regular vacuuming, dusting, cleaning hard surfaces and soft furnishings with soap and water and/or 1:10 diluted bleach were provided.

The increase of morbidity and mortality associated with PVL-SA is of concern in Spain. A significant increase in the incidence of community-associated MRSA from 0.7% in 2004 to 8.8% in 2012 has been reported. The emergence of PVL-MRSA is more recent in Spain (mainly from South America immigrants) than in the rest of Europe.[1] One of the most important life-threatening conditions due to PVL-*Staphylococcus aureus* (PVL-SA) reported is hemorrhagic necrotizing pneumonia with a high mortality rate.[4] Characteristic patterns include a rapidly extensive pneumonia occurring in young patients after an influenza-like illness.[7,8]

A PVL-SA infection should be suspected if a patient with influenza-like illness associate haemoptysis, hypotension, high fever, leukopenia and/or multilobal lung infiltrates, which can be cavitated,[9] especially in epidemic flu period. The rareness in our country of severe cases of PVL-SA infection, especially when we found a MSSA, needs a greater awareness,[10] establishing an action guide with preventive measures and systematic notification to the epidemiological surveillance network as exist in other countries.[5]

Conflict of interest

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