

FIRST OUTBREAK OF METHICILLIN-RESISTANT *STAPHYLOCOCCUS AUREUS* ST398 IN A DUTCH HOSPITAL, JUNE 2007

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We describe the first outbreak of non-typable methicillin-resistant *Staphylococcus aureus* on a surgical ward in the Netherlands in June 2007. Nine cases of infection and/or colonisation were found among patients and healthcare workers.

Background

In the Netherlands, the proportion of methicillin-resistant *S. aureus* (MRSA) among clinical isolates of *S. aureus* is still low [1], but community-acquired MRSA occurs more frequently [2]. This increase is mainly caused by so called 'non-typable' MRSA (NT-MRSA, i.e. not typable by pulsed-field gel electrophoresis (PFGE) with *Sma*I restriction digest [3]) belonging to multilocus sequence typing (MLST) type ST398 [4].

These strains are widely disseminated among pigs, veal calves and people in contact with pigs [5-8]. An association between the use of antibiotics in pig farming and the dissemination of these strains has been suggested [6,8], since the majority of ST398 MRSA are tetracycline-resistant and oxytetracyclins are the most frequently used antibiotics in pig farming.

Transmission within families, as well as single cases of colonised healthcare workers, have been described [5]. One report indicates possible healthcare-acquired infections with a Panton-Valentine leukocidin (PVL)-positive ST398 strain in China [9], but no nosocomial transmission to multiple patients or healthcare workers has occurred in the Netherlands to date.

Outbreak description

In June 2007, MRSA was cultured from a diabetic foot ulcer of a patient on a surgical ward. Subsequent screening of contacts among patients and healthcare workers revealed four additional patients with MRSA infection and/or colonisation and five healthcare workers who carried MRSA.

Two of the five affected patients (one with prostate carcinoma and one with a diabetic foot) were successfully decolonised with mupirocin nasal ointment, chlorhexidine wash, and treatment with trimetoprim/rifampicin.

A further colonised patient with a gastro-intestinal malignancy and two patients with infected diabetic foot ulcers remained colonised, despite several decolonisation regimens.

Of 238 healthcare workers who were screened, five were colonised in the nose and/or throat and had no skin conditions. All five have been treated with mupirocin nasal ointment and chlorhexidine wash and successfully decolonised.

All strains were resistant to tetracycline and non-typable by PFGE. Spa-typing showed that all strains were spa-type t567. This spa-type corresponds to MLST type 398, a type previously found in pigs.

None of the patients had had contact with pigs or veal calves. One healthcare worker lived on the grounds of a pig farm but neither she nor her partner came into contact with pigs themselves. While we presume that this health care worker was the source of the infection, this could not be proven. Permission to sample the pigs on this farm was not granted.

Conclusions

The NT-MRSA strain responsible for this outbreak was spa-type t567, which corresponds to MLST type ST398, the clonal complex to which most of NT-MRSA strains belong. This outbreak shows that transmission on a larger scale than a one-on-one transmission between caretaker and patient can occur with NT-MRSA in a hospital setting.

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