Simultaneous presence of an invasive and a carrier strain of methicillin-resistant Staphylococcus aureus (MRSA) in a family

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Simultaneous carriage of multiple clones of MRSA has rarely been reported. We describe a case of bacteraemia and osteomyelitis due to CA-MRSA and the simultaneous presence of 2 clones of MRSA in a family, 1 strain with an invasive capacity and another strain colonizing several family members.

Introduction
Community acquired methicillin-resistant Staphylococcus aureus (CA-MRSA) is emerging and the incidence is rapidly increasing in Europe [1]. In low prevalence areas, such as Scandinavia, almost half of the MRSA are community acquired, regardless of whether contracted abroad or domestic (www.smittskyddsinstitutet.se). Intra-familial spread is common [2] and high prevalence has been reported from the US among, for example, military recruits and prison inmates indicating crowded living conditions as a risk factor. Methicillin resistance is mediated by the mecA gene located on the staphylococcal cassette chromosome mec (SCCmec) of S. aureus. To date, 6 structurally different types of SCCmec (types I, II, III, IV, V, and VI) have been identified among MRSA, and types IV and V are predominantly associated with CA-MRSA. These MRSA are primarily related to skin and soft-tissue infections and serious, often necrotizing, pneumonia [3], and an association between these clinical conditions and the presence of the genes lukS-PV and lukF-PV encoding Panton-Valentine leukocidin (PVL) has been highlighted [3]. However, additional virulence determinants may also be of importance [4].

We here describe a case of bacteraemia and osteomyelitis due to CA-MRSA and the simultaneous presence of 2 clones of MRSA in a family, 1 strain with an invasive capacity and another strain acting as a colonizer of several family members.

Case report
In August 2007 a 10-y-old boy was admitted to Örebro University hospital due to increasing pain of the left hip for 4 d and also, on the last d, occurrence of fever. On physical examination the left hip joint was normal but redness of the right ankle was noted and the temperature was 39.4°C. C-reactive protein was 106 mg/l and the sedimentation rate 38 mm but the leukocyte count including neutrophils was normal. An X-ray of the left hip was normal although a scintigraphic investigation revealed an increased activity of the major trochanter region of the left hip and of the medial malleolus of the right ankle. Biopsies from the left hip and right ankle were performed in general anaesthesia at the operating theatre. Cultures subsequently showed growth of MRSA, both from the hip and the ankle, as well as aerobic and anaerobic blood cultures. The patient...
was empirically treated with cefuroxime intravenously but the treatment was changed to clindamycin intravenously in combination with rifampicin orally when culture results were available. The patient rapidly improved and the temperature normalized. A follow-up performed 3 weeks later with X-ray showed a minor osteolytic process of the major tuberculum. Rifampicin was administrated for 4 weeks and altogether treatment with clindamycin orally was given for 3 months. A follow-up after discontinuation of antimicrobial treatment was uneventful.

The patient’s family consisted of the patient’s mother, father, grandmother and 3 siblings. The family were refugees from Burma and had spent 9 y in a refugee camp in Thailand before arriving in Sweden in September 2006.

Screening for MRSA from the nares, throat and perineum of all the family members was performed by PCR following broth enrichment [5] and was positive for all members except for the grandmother. However, the antibiotic susceptibility pattern as determined by Etest (AB Biodisk, Solna, Sweden) differed between the isolates, with 2 isolates found to be resistant to trimethoprim/sulfamethoxazole and tetracycline, but all isolates were otherwise susceptible to clindamycin, fusidic acid, gentamicin, rifampicin, ciprofoxacin and vancomycin. MIC values for oxacillin performed on Iso Sensitest Agar (Oxoid) supplemented with 5% defibrinated horse blood (SVA) and 2% β-nicotinamide adenin dinucleotide (Sigma-Aldrich Inc, St. Louis, MO, USA) ranged from 0.5 to 1.0 mg/l.

Further characterization and typing of the MRSA isolates revealed the presence of 2 clones of MRSA. Spa typing [6] showed that 2 different spa types could be detected among the MRSA isolates of the index patient: t878 (26–23–17–34–21–25–33–16) and t2882 (26–23–17–34–17–20–17–12–20–17–12–16), respectively. The t2882 MRSA isolate was found in the blood and biopsy cultures, and t878 isolate was found to colonize the throat. Four family members were colonized (nares, throat, and/or perineum) with the same strain, t878, but the fifth was colonized with the invasive strain, t2882. Also, pulsed-field gel electrophoresis (PFGE) of chromosomal SmaI digests (7) showed 2 pulsotypes (Figure 1). In addition, determination of the SCCmec types I, II, III, IV, and V was performed as described [6]. Two isolates could be assigned as SCCmec type V and the rest as SCCmec type IV (although not possible to subtype as a, b, c, or d) (see Figure 1).

Neither the genes for Panton-Valentine leukocidin (PVL), lukS-PV and lukF-P [8] nor the gene for toxic shock syndrome toxin-1 (TSST-1), tst [9], were detected among any of the isolates.

**Discussion**

Carriage of multiple subtypes of MRSA in a hospital setting such as an intensive care unit [10] and following nosocomial acquisition [11] has been described. However, reports describing simultaneous carriage of various clones are scarce [11], especially in the community.

We here describe a case in which 2 different clones of MRSA simultaneously colonized a family. The 2 strains showed different patterns as determined by PFGE and also unrelated spa types according to BURP analysis [12]. One clone was prevalent and colonized 5 out of 6 family members, and 1 additional clone, colonizing only 1 family member but also caused an invasive disease, bacteraemia and osteomyelitis, of the index case. This latter, invasive clone may exhibit specific virulence determinants [4] but was found to be negative for the PVL locus that has specifically been proposed to be responsible for the enhanced virulence of CA-MRSA [13]. Neither were any of the strains positive for TSST-1. Perhaps this clone was recently introduced into this family setting and therefore the experience of the immune system of the index patient was naïve regarding expressed virulence factors of this MRSA strain. The SCCmec associated with CA-MRSA are relatively small compared with HA-MRSA, and CA-MRSA are commonly lacking additional resistance genes. Accordingly, the CA-MRSA may not depend on

![Figure 1. Pulsed-field gel electrophoresis (PFGE) patterns and typing characteristics of the MRSA. The index patient is represented by the blood isolate, 07B479 and an isolate colonizing the nares, 07T860. The family members are represented by 1 carrier isolate each.](image-url)
exposure to antibiotic pressure for selection and are obviously capable of existing as human colonizers. The 2 MRSA clones were assumed to be CA-MRSA since there was no history of hospital contacts although the family had lived for a long period in a crowded refugee camp. Also, according to the SCCmec characterization, types associated with CA-MRSA were found, i.e. types IV and V. However, the epidemiological information about CA-MRSA in Thailand [14], and especially in Burma, is very limited.

This case illustrates the simultaneous presence of 2 different strains of MRSA in a patient, 1 acting as a harmless colonizer or commensal and the other as an invasive pathogen causing septic metastatic disease.

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References