Case report

MRSA eradication in a health care worker with cystic fibrosis; re-emergence or re-infection?

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Abstract

Methicillin-resistant Staphylococcus aureus (MRSA) is an emerging infection in patients with cystic fibrosis (CF). MRSA may be a management dilemma for healthcare workers (HCWs) with CF. Eradication of MRSA with long-term rifampicin and fusidic acid can be achieved in patients with CF. We describe a case of recurrent MRSA infection in a HCW with CF. Molecular typing of the MRSA isolates supported re-infection rather than re-emergence of an earlier MRSA infection. Infection control advice for HCWs with CF who acquire MRSA remains controversial.

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1. Introduction

Methicillin-resistant Staphylococcus aureus (MRSA) was first identified in 1960 [1]. Risk factors for the acquisition of MRSA include undergoing a surgical procedure, admission to an intensive care unit, chronic and/or long-term hospitalisation and admission to health care facilities with high rates of endemic MRSA [2].

The incidence of MRSA is increasing in patients with cystic fibrosis (CF) [3]. The North American Cystic Fibrosis Data Registry has reported that 9.2% (individual centres range from 0% to 26%) of children and adults with CF had MRSA isolated from their respiratory secretions [4]. A 1994 survey of patients with cystic fibrosis (CF) in the UK showed that 6.6% worked as health care workers (HCWs), which represented as many as 160 HCWs at that time [5]. It is likely that this number has grown [6] and reflected in other countries.

This leads to concerns of infection control between patients and HCWs and what advice should be given. We report our experience of a health care worker with CF and MRSA infection.

2. Case report

The patient was a 29-year-old HCW with CF who attended the regional adult CF unit in The Prince Charles Hospital (TPCH), Brisbane, Australia. She was diagnosed with CF at 3 years old in the UK and moved to Australia in 2003. She had moderate lung disease (FEV1 63% predicted, FVC 77%) and Pseudomonas aeruginosa infection requiring yearly intravenous (i.v.) antibiotics. She acquired MRSA in 2001 following a surgical procedure and was treated with i.v. vancomycin and oral fusidic acid for 2 weeks. Sputum culture and screening swabs in the UK prior to moving to Australia were negative for MRSA. She was first seen as an outpatient in Brisbane in March 2003. Sputum bacteriological analysis in April and May 2003 revealed a light to moderate growth of two P. aeruginosa strains and a heavy growth of MRSA. The MRSA identified...
was classified as non-multi-resistant exhibiting resistance to only the penicillins, erythromycin and ciprofloxacin. This was similar to the antibiotic resistance pattern of the common endemic MRSA strains in the UK. It was also notably urease negative (Microscan Pos Combo Panel Type 1A biotype number: 317157-Dade Behring Inc., West Sacramento, USA) and thus phenotypically consistent with that of the previously reported EMRSA-15 strain, which has spread throughout the UK since the early 1990s [7]. Swabs from nose, axilla and perineum over this period were negative for MRSA. As there was prolonged increased sputum production, an attempt at MRSA eradication was initiated using a combination of oral rifampicin (600 mg daily) and fusidic acid (500 mg bid) for 6 months. This treatment was commenced in May 2003. Monthly sputum cultures during therapy and until early January 2004 were negative for MRSA. There were no hospitalisations from March 2003 to March 2004. Three weeks after completion of rifampicin and fusidic acid, she had a mild pulmonary exacerbation of CF requiring oral ciprofloxacin and nebulised tobramycin. A scant growth of MRSA was isolated in sputum cultures on three occasions from late January 2004 until March 2004. The organism was phenotypically dissimilar to the isolate identified in 2003. It produced a different Microscan PC1A biotype number 717172 (Dade Behring Inc.), along with a three class change in the antibiotic susceptibility profile (i.e. resistant to gentamicin, tetracycline and trimethoprim-sulfamethoxazole). The multi-resistant susceptibility profile was similar to the endemic MRSA strains seen in South East Queensland [8]. Pulsed-field gel electrophoresis analysis of SmAl digested DNA from each isolate demonstrated the existence of two distinct strains differing by greater than 6 bands (Fig. 1—isolates A and B) as proposed by Tenover et al. [9]. This suggested a newly acquired MRSA infection. Nose and perineum swabs were also positive for MRSA. Following further discussions with the patient, treatment with rifampicin and fusidic acid was recommenced in March 2004 for 6 months. Sputum samples were negative for MRSA during treatment. However, following completion of treatment, a sputum single sample was positive for MRSA followed by three MRSA negative sputum cultures.

3. Discussion

This case describes MRSA infection in a HCW with CF. There is controversy as to the clinical importance of MRSA in CF, but it is felt that the pathogenicity of MRSA is considered to be equal to that of methicillin-sensitive S. aureus [10]. At TPCH, patients with CF and MRSA are routinely segregated from those without this organism. As has been suggested with Burkholderia cepacia complex (BCC) infection, this may be associated with a sense of social isolation and could possibly result in a similar adverse psychological effect upon some affected patients [11–13].

Does this case represent a new infection or re-emergence of MRSA? The vastly different phenotypic and genotypic profiles of each MRSA isolate suggest that this is a newly acquired infection, but what was the likely source? Our patient did not have an admission to the CF unit and was seen separately from other CF patients as an outpatient. Previous work at our hospital in adults with CF with MRSA demonstrated that 47% of MRSA isolates were genetically distinct from local endemic strains [14]. As she worked as a HCW and close contact is required for MRSA acquisition [15], it is possible that she reacquired MRSA from patient contact at work.

What advice should be given to a HCW in this situation and what risk do they pose in turn to their patients? The CF Trust UK state that HCWs who carry or are infected with MRSA should not be allowed to come into contact with patients until the problem has been adequately treated [6]. Reports have suggested antibiotic regimes for MRSA eradication, including linezolid [16,17] and vancomycin (55% successful) [18]. However, in a recent study from our group, 5 out of 7 patients treated for MRSA with rifampicin and fusidic acid did not culture MRSA from sputum after the initiation of therapy and during 6 months after treatment [19]. Therefore, our patient continued to work during this time and used a gown, mask and gloves, while in direct contact with patients. However, her ongoing employment in the health care profession is likely to be a significant risk for MRSA infection and a potential source of further infection to other patients.
To our knowledge, this is the first reported case of likely transmission of MRSA from a patient to a HCW with CF. As the patient initially acquired MRSA in the UK, this case provides a unique opportunity to demonstrate that the recurrence of MRSA may be due to a new infection rather than merely re-emergence. As there are increasing numbers of patients with CF that are opting for careers in the health care profession, we will be faced with increasingly challenging situations.

References