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Prolonged outbreak of *Staphylococcus aureus* surgical site infection traced to a healthcare worker with psoriasis

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SUMMARY

Background: Meticillin-sensitive *Staphylococcus aureus* (MSSA) is a frequent cause of surgical site infection (SSI), but point-source outbreaks are rarely recognized.
Aim: To describe an outbreak of MSSA SSI in a thoracic surgical unit.
Methods: An outbreak investigation was started following two postoperative bacteraemias with MSSA resistant to fusidic acid (MSSA FusR). Patients with MSSA FusR were identified from microbiology records and through prospective case finding. Healthcare workers (HCWs) were screened. Isolates were characterized by phage typing, spa typing, pulsed-field gel electrophoresis and toxin gene profiling. A case–control study examined the association between one HCW with MSSA FusR and the patients involved in the outbreak.
Findings: Nineteen patients were identified with MSSA FusR over 16 months. Four isolates were available for typing and all belonged to the same lineage. Seventy-six HCWs were screened. One was a carrier of the outbreak strain (a nurse with psoriasis). All 19 cases were exposed to this HCW compared with only 40/66 controls ($P = 0.003$) and cases had a greater duration of exposure ($P = 0.00001$, chi-squared for trend). Direct patient contact was documented in 15 cases. The outbreak was halted by thorough cleaning of the ward and removal of the HCW from clinical duty.
Conclusion: The HCW with psoriasis was the source of this outbreak. MSSA FusR may be a marker for strains associated with skin conditions. HCWs with significant skin conditions may pose an infection risk in surgical settings. Recommendations are made for occupational health teams regarding screening of HCWs with dermatitis.

Introduction

The overall prevalence of surgical site infection (SSI) is estimated to be 4.65% and this is associated with increased morbidity, mortality, length of stay, and costs.[1,2] It is well established that *Staphylococcus aureus* is the most prevalent pathogen.[3,4] Approximately one in four of the adult general population carries *S. aureus* in their nares and the incidence of *S. aureus* SSI is known to be higher in preoperative carriers, as most patients are infected with their own strains.[5,6]

There are numerous reports of outbreaks of *S. aureus* infections on surgical wards dating back through the literature. These may arise from an index patient, for example at the time

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of wound dressing by way of contaminated hands and instruments.[7] Healthcare workers (HCWs) have long been recognized as possible sources with a report from 1939 of a study of SSIs arising from perforations in the operating gloves of an individual surgeon known to carry *S. aureus* in his nose and on his skin.[8] Air-borne infection during an operation is a well-recognized mode of spread, with a report from 1967 describing a cluster of postoperative wounds infected with a *S. aureus* originating from a theatre orderly with eczema, who was shown to be a disperser of the outbreak strain.[9] Whereas these and other reports have focused on individuals, the epidemic strain implicated in an outbreak can be carried by a number of members of staff, suggesting multiple possible modes of transmission.[10–12]

More recently, there have been reports of outbreaks of meticillin-resistant *S. aureus* (MRSA) SSI infection attributable to HCWs, and indeed, many of the issues related to aspects of carriage of MRSA in HCWs also apply to carriage of meticillin-sensitive *S. aureus* (MSSA).[13] Examples include an outbreak involving seven patients on two surgical wards in France which was traced to a worker with chronic sinusitis in the operating theatre.[14] An outbreak of postcardiac surgery wound infections and mediastinitis in northern Taiwan was linked to a surgeon who was an MRSA carrier.[15] Recently, the application of molecular analyses [*spa* typing, pulsed-field gel electrophoresis (PFGE) and multi-locus variable-number tandem-repeat analysis] showed that a prolonged outbreak of MRSA in an English cardiac surgery unit was linked to a single colonized HCW.[16] A systematic search of the literature has reported that 11 out of 191 nosocomial outbreaks of MRSA were likely to have been initiated by HCWs, based on strong epidemiological evidence. In three of these outbreaks, asymptomatic carriers were thought to be the cause.[17]

Although MSSA is no lesser pathogen than MRSA, there has been perhaps less emphasis on its surveillance compared with MRSA over recent years. We describe a relatively prolonged MSSA outbreak which raises important questions regarding our ability to detect such incidents in a timely fashion and the risks posed by *S. aureus*-colonized HCWs with chronic skin conditions in certain patient settings.

Methods

The outbreak

In September 2008, two postoperative thoracic surgical patients developed MSSA bacteraemia in a 22-bed thoracic surgical unit. Both isolates were fully sensitive to the standard antistaphylococcal antibiotics except for fusidic acid (MSSA FusR). Patient 1 was a 57-year-old female, post oesophagectomy, who was readmitted three days after discharge with severe sepsis and died. MSSA FusR was isolated from her wound and blood cultures. The second patient, a 57-year-old male, had an infected open wound following an open biopsy of a mediastinal mass. MSSA FusR was isolated from a wound swab and blood cultures. The blood culture isolates from both patients were sent for typing and found to belong to the same lineage (*spa* type t1892). A look-back exercise over the previous three months revealed four other MSSA FusR-positive thoracic patients (positive wound swabs and/or chest drain sites). Two further cases were detected prospectively as a result of enhanced surveillance. MSSA FusR was grown from a thoracotomy wound swab from a 61-year-old male post-oesophagogastrectomy patient and from chest drain pus from a 77-year-old female. This last patient had a chest drain inserted on the ward preoperatively and infection was then noted at the time of surgery for a thymoma resection.

Control measures

An outbreak meeting was convened. It was decided to close the unit to admissions and elective surgery was cancelled. Current inpatients and all staff were screened (nose swabs and swabs from wounds; staff were also issued with a health question screen regarding significant skin conditions, and, if identified, swabs were performed). The ward was thoroughly cleaned. During the outbreak meeting, concerns were raised about one particular ward-based member of the nursing staff with a skin condition.

Outbreak investigation

The case definition was MSSA FusR from a sterile site or wound swab in a patient who had been via the thoracic surgical unit. Retrospective analysis of microbiology records for all MSSA FusR isolates from the wider Nottingham area from the beginning of July 2007 was undertaken to establish the incidence of MSSA FusR in different units across the hospital. Prior to this, records were on a previous system, which was no longer available for data extraction. Available isolates were sent to the Staphylococcal Reference Unit, Health Protection Agency (now Public Health England), Colindale, for characterization.

Case–control study

A retrospective case–control study was performed to examine the epidemiological association between one HCW who was found to be a carrier of MSSA FusR and the patients involved in the outbreak. Sixty-six randomly selected controls were identified from patients who had an inpatient stay of >48 h on the thoracic surgical unit during the time of the outbreak, but who had not grown MSSA FusR. Admission and discharge data were obtained from the hospital system and the nursing rota and individual case notes were reviewed. The number of days on which cases and controls were exposed to the HCW carrying the outbreak strain was calculated (excluding day of admission and day of discharge, and days spent on intensive therapy unit).

Laboratory methods

As described previously, *S. aureus* bacteria were characterized by phage typing and a range of molecular techniques [*spa* typing, PFGE, multiplex polymerase chain reaction (PCR) for the detection of 14 toxin genes: enterotoxins A–E and G–J, exfoliatins A, B and D, toxic shock syndrome toxin-1 and Panton–Valentine leucocidin, plus PCR-based detection of *mecA*].[18]

Screening swabs (nose and lesions) from inpatients and staff were directly plated with no enrichment step.