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SARS in a hospital visitor and her intensivist

Sir,

Severe acute respiratory syndrome (SARS) is a novel infectious disease in humans caused by a coronavirus that was first recognized in Southeast Asia in late February 2003.[1] The World Health Organization (WHO) has issued definitions for probable and suspected cases.[2] Most transmissions occurred by close hospital or household contact with infected individuals suggesting a predominant droplet mode of spread.[3] Widespread use of personal protective equipment (PPE) including N95 respirators is thought to have been instrumental in controlling the epidemic. We report a patient who had had no direct contact with SARS patients and who subsequently transmitted the infection to her intensivist who was wearing standard PPE during a bronchoscopy.

A 43-year-old woman was admitted to our hospital with a respiratory and systemic illness of 1-week duration. She had had no direct contact with any patients suffering from SARS, but had visited a friend with hepatitis in hospital who was at least two cubicles (approximately 10 m) away from a patient later confirmed to have SARS. On examination, she was tachypnoiec and febrile. Chest radiography revealed bilateral basal consolidations and the white blood cell count was 19.3×10^9/L (polymorphs 90%, lymphocytes 4%). Community-acquired pneumonia was diagnosed and the patient was isolated in a single room with negative pressure and started on intravenous antibiotics. The following day, she required endotracheal intubation and mechanical ventilation. Over the next four days, she developed multisystem organ failure and adult respiratory distress syndrome requiring haemofiltration and vasopressor support and died on the eighth day of admission. Cultures of blood, urine and bronchoalveolar lavage (BAL) were negative for micro-organisms including fungi and mycobacteria.

Three days after the bronchoscopy, the intensivist (K.-H.L.) who performed the procedure developed fever and myalgia and was subsequently diagnosed as having SARS with pneumonia, despite wearing a N95 mask, glasses, gown, and gloves throughout the procedure. He required admission to intensive care unit and mechanical ventilation, and survived the episode.

The patient's serology was positive for SARS by a dot-blot immunoassay using a viral lysate in a post-mortem analysis of a serum sample taken on day three of admission. Her intensivist was also seropositive 4 weeks after hospitalization.

Our patient met the revised WHO criteria[2] for SARS except that she did not 'care for, live with, or have had direct contact with respiratory secretions and body fluids of a person with SARS,' as the WHO defines close contact.[2] Her serology was only found to be positive at post-mortem. To our knowledge, fomite spread has not been well documented for SARS, although it has been demonstrated experimentally for other respiratory viral pathogens, and is the probable route of transmission for our patient. Environmental contamination is the most likely route of transmission in Hotel M, which triggered off the worldwide outbreak of SARS[1] and in the large Amoy Gardens outbreak.[4] Transmission to a bronchoscopist has not, to our knowledge, been documented before. During the procedure the doctor wore an N95 mask, glasses, gloves, footwear and disposable gown. However, he was not using specific eye protection and a powered air purifying respirator (PAPR) hood, which has become common practice since then.

This case suggests that if the viral load is high the N95 mask may not offer sufficient protection during high-risk procedures, including bronchoscopy, nebulizer therapy or intubation. This is similar to the experience in Canada in which transmission of SARS to protected healthcare workers occurred during a difficult intubation.[5] We believe that

high-risk procedures such as intubation or bronchoscopy may require a higher level of protection than N95 respirators. We also feel that more work needs to be done to establish the role of environmental surfaces in the indirect transmission of the virus to individuals without direct contact. These will be important for future strategies for SARS.

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Isolation of MRSA from communal areas in a teaching hospital

On 26 September 2003, the *Evening Standard* printed an article entitled: 'Killer bugs widespread in 'horrifying' hospital study'.[1] The work was carried out on behalf of Chemsol, UK, and reported by Mr C. Malyszewicz, Consultant Chemist/Microbiologist, using direct contact plates for the detection of methicillin-resistant *Staphylococcus aureus* (MRSA) made by Dimanco Ltd, Henlow, UK. The media comprised a dip-slide. One side was standard Baird Parker Agar, complete with egg yolk and potassium tellurite and the other, selective side, was mannitol salt agar with 4 mg/L methicillin (not oxacillin).

The survey reported on eight London hospitals from which 22 out of 34 swabs taken from public areas were reported positive for MRSA. At the Middlesex Hospital 'five out of seven showed MRSA' (71.4%). The prevalence of MRSA outside the clinical areas of hospitals has not been previously determined, but previous studies have shown that patient colonization varies from 0.2 to 1.3%[2] in the community, to 16.2%[3] on intensive care units. These results contrast markedly with the stated prevalence of 71.4% in the *Evening Standard* report.

In response to this article, we performed an internal investigation to verify these results. Twenty swabs were taken from areas accessible to members of the public, i.e. handles to entrances, corridors, canteen and toilet doors; internal and external lift control panels and banisters. Samples were taken from each wing of the Middlesex Hospital and most were obtained from the ground floor, which forms the only common route of access between wings. Swabs were taken from 5 cm^2 areas and these were incubated in nutrient enrichment broth at 37 °C for 48 h and then subcultured on to Colombia blood agar by streak plate method and incubated at 37 °C for 18 h. All Gram-positive cocci identified as catalase positive were identified by standard methods and tested for oxacillin resistance. Three swabs yielded no growth, and organisms were identified from the other 17, including 10 coagulase-negative staphylococci. Of these, three (30%) were resistant to oxacillin. No MRSA were identified. Gram-negative bacteria were isolated from two swabs.

The study was then repeated one week later at the same sites with the contact media used by Chemsol (kindly supplied by the manufacturer). Once again, no MRSA were identified. Coagulase-negative staphylococci were identified at 17 of the 20 sites, nine of which were methicillin resistant. There was one isolate of methicillin-sensitive *S. aureus*. Gram-negative bacteria were isolated at one site. Neither the cleaning procedures nor the contractor's cleaning staff changed between the original sampling by Chemsol and our own studies.

The current public and governmental interest in MRSA means that any story of widespread MRSA in the hospital environment will receive prominent coverage. Poor cleaning standards by outside contractors are often blamed. However, for such stories to effect an improvement in investment in cleaning they must be factually accurate. Unfortunately, newspapers do not generally subject such articles to expert review. This particular claim of