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reported to have SARS after exposure to an index case. One infected staff member had no exposure to any admitted patient with SARS and was classified as a community-acquired infection. All index patients and infected staff in the study, except for one, showed a four-fold rise in the number of antibodies to corona-like virus.

356 completed questionnaires were returned, covering 85% of the staff on roster. Non-responders were mostly those on leave or night shift, which is rotated among the staff. We excluded 102 staff who had no contact with index patients. Most of the infected staff were from the medical wards (table 1), and omitted at least one of the four measures queried. Two who were using a mask reported only paper masks.

Staff who used masks, gowns, and handwashing were less likely to develop SARS than those who did not use them, but the association for gloves was not significant (table 2). None of the 69 staff reporting use of all four measures became infected. By contrast, all 13 infected staff had omitted at least one of the measures (p=0.0224). However logistic regression of the four measures with forward stepwise selection showed that only use of masks was significant in the final model (table 2).

The staff who wore surgical masks and N95 masks were significantly associated with non-infection (table 2), but this was not seen for paper masks.

That use of masks and handwashing was associated with non-infection, and that no staff became infected when they used all four measures, suggest that precautions against droplets and contact are adequate for prevention of nosocomial SARS, where no aerosolisations are expected. The surgical and N95 masks were both effective in significantly reducing the risk of infection, which together with the finding that 30% of non-infected staff did not use masks (table 2) supports that transmission is not airborne. The finding that paper masks did not significantly reduce the risk is not unexpected. Such masks, being easily wet with saliva, are never recommended as a precaution against droplets.

In any survey, recall bias is a concern. However, it probably had little effect since the associations shown are clear and the information requested was about simple concrete behaviour and events that took place recently. Masks seem to be essential for protection, since only this measure was significant in stepwise logistic regression. Thus, in hospital, the other three measures add no significant protection to the mask. This finding fits well with droplets transmission because droplets are generated at the face level making the mask crucial for protection.

Table 2: Protective measures reported by infected and non-infected staff

| Protective measures | Infected Staff (n=13) | Non-infected staff (n=241) | p* | Odds ratio (95% CI)† |
|---------------------|-----------------------|---------------------------|----|---------------------|
| Masks§              | 2 (15%)               | 169 (70%)                 | 0·0001 | 13 (3–60)           |
| Paper mask          | 2                     | 26                        | 0·511‡ |                     |
| Surgical mask       | 0                     | 51                        | 0·007‡ |                     |
| N95                 | 0                     | 92                        | 0·0004‡|                     |
| Gloves              | 4 (31%)               | 117 (48%)                 | 0·364|                     |
| Gowns               | 0 (0%)                | 83 (34%)                  | 0·006|                     |
| Hand-washing        | 10 (77%)              | 227 (94%)                 | 0·047|                     |
| All measures        | 0 (0%)                | 69 (29%)                  | 0·022|                     |

Notes: NC=not calculatable. *Two-tailed. †Odds ratio of staff with specific protection not getting infected. ‡Yes and "most of the time" were grouped together. †Total cases 254 by forward stepwise (Waldesian) logistic regression using 0·05 as entry probability and 0·10 as removal probability. Forward and backward stepwise regression result in same model with mask in the model (p=0·011); ‡Comparing proportion of infected over non-infected staff, with those without mask (11 infected and 72 non-infected).

Contributors

W H Seto is the main investigator and was involved in all aspects of the study. D Tsang, R W H Yung, T K Ng, and M Ho helped in the survey, and T Y Ching coordinated the infection-control nurses. L M Ho did the statistical analysis, and J S M Peiris organised the laboratory tests and supervised the writing of the report. The Expert Group advised on the clinical aspects of the report.

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Conflict of interest statement

None declared.

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Haemorrhagic-fever-like changes and normal chest radiograph in a doctor with SARS

Eugene B Wu, Joseph J Y Sung

A 33-year-old doctor contracted severe acute respiratory syndrome presenting with features of disseminated intravascular coagulopathy without changes in the chest radiograph initially. A CT scan of his chest showed marked lung changes. His condition improved with intravenous methylprednisolone 500 mg daily and ribavirin 1-2 g orally thrice daily. The case illustrates the importance of a break in fever between the viraeitic and lung inflammatory phases of the illness that
occurs before radiographic changes and which may obscure diagnosis. Careful quarantine and follow-up of these patients are necessary. Coagulopathy is usually uncomplicated and early CT of the chest may elucidate hidden lung changes and facilitate a rapid diagnosis.

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The index case of severe acute respiratory syndrome (SARS) was admitted to ward 8A in the Prince of Wales Hospital, Hong Kong, on the March 4, 2003. On March 10, 2003, a 33-year-old doctor (EBW, figure) working on ward 8A developed a fever of 39·6°C. He was examined by JJYS. His fever had gone by March 12, and his chest radiograph was normal. His platelet count was 94 × 10^9/L and white-cell count was 3·4 × 10^9/L (monocytes 0·4 × 10^9/L). A nasopharyngeal swab grew no pathogens. He was admitted to the SARS triage ward on March 13, and was started on oseltamivir phosphate 75 mg twice a day and levofloxacin 500 mg daily. Further blood tests showed disseminated intravascular coagulopathy (platelets 61 × 10^9/L, D-dimer 630 ng/mL, prothrombin time 11·1 s, activated partial thromboplastin time (APTT) 43·3 s). His white-cell count was 1·8 × 10^9/L (neutrophils 1·1 × 10^9/L, lymphocytes 0·5 × 10^9/L, and monocytes 0·2 × 10^9/L). His chest radiograph showed a prominent right hilum. CT of his thorax showed an ill-defined opacity with an air bronchogram in the apical posterior segment of the right lower lobe and diffusely in the right middle lobe. He was started on oral ribavirin 1·2 g thrice daily and intravenous methylprednisolone 500 mg daily. His fever settled the next morning and his coagulopathy improved (APTT 40·7 s, platelet count 105 × 10^9/L, and D-dimer of 564 ng/mL). On March 19, 2003, oral prednisolone 1 mg/kg was started.

On the evening of March 20, he had a fever of 38·9°C. His white blood cell count rose to 15·7 × 10^9/L (predominantly due to an increase in neutrophils). A secondary bacterial chest infection was suspected, and cefipime 2 g was given intravenously. Over the next 2 days he became increasingly breathless and his coagulopathy became worse (D-dimer of 716 ng/mL, prothrombin time 11·9 s, platelets 199 × 10^9/L). The patient was given a single dose of methylprednisolone 500 mg intravenously and 4 L/min of oxygen. After this, he began to get better. Coagulation parameters returned to normal, he was weaned off of oxygen, and was discharged from hospital on March 31, 2003, on 0·3 mg/kg prednisolone and ribavirin 600 mg orally three times a day. On April 7, his chest radiograph showed worsening consolidation of the consolidation of the right middle zone and the prednisolone was increased to 0·5 mg/kg.

This illness shows several characteristic features of SARS. There is often a break in the fever between the viraemic stage (days 1–3) and the lung inflammation phase of the illness. Patients who become apyrexial for several days still need up to 10 days of quarantine and follow-up. Haemorrhagic-fever-like illness with disseminated intravascular coagulopathy does occur but is usually mild. Although 78·3% of SARS patients in our centre had an abnormal chest radiographs when feverish, some can have relatively normal radiographs. As shown in this case, a chest CT can show hidden pneumatic changes and facilitate a rapid diagnosis.

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Risk of vaccine failure after Haemophilus influenzae type b (Hib) combination vaccines with acellular pertussis

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An increase in invasive Hib disease incidence in the UK has coincided with the distribution of combination vaccines that contain acellular pertussis (DTaP-Hib). These vaccines have been associated with reduced immunogenicity of the Hib component, although there is little agreement on the clinical relevance of this finding. We retrospectively compared vaccine formulations given to fully vaccinated Hib cases with those administered to fully immunised age-matched controls using conditional logistic regression. More cases than control received all three doses of their infant primary course as DTaP-Hib, compared with two or three doses of another Hib vaccine (conditional odds ratio 6·77 [95% CI 3·26–14·07]).

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Haemophilus influenzae type b (Hib) was a leading cause of meningitis in infants in the UK until October, 1992, when Hib vaccine without a booster dose was introduced for children at age 2, 3, and 4 months. Despite a striking initial fall in the rate of disease in England and Wales from 22·9 per 100 000 children younger than 5 years in 1990, the incidence of invasive Hib infection has risen from the nadir of 0·65 per 100 000 in 1998 to 4·6 per 100 000 in 2002. From these data, the estimated direct protective effect of Hib vaccine against invasive disease in the first 2 years of life was substantially lower in children born in 2000 and 2001 than in those born between August, 1992 and December, 1999.