Asymptomatic Severe Acute Respiratory Syndrome–associated Coronavirus Infection

To the Editor: An outbreak of severe acute respiratory syndrome (SARS) began in Hong Kong in March 2003. As of May 29, 2003, a total of 1,732 cases were confirmed; 381 case-patients were healthcare workers and medical students. Clinical features, treatment protocols, and outcomes have been previously reported by various local experts (1–3). The etiologic agent is a SARS-associated coronavirus (SARS-CoV) (1). However, no asymptomatic case of SARS-CoV infection has been previously reported (4). In addition, in Hong Kong, blood donors have not shown any detectable antibody to SARS-CoV (1). We report a case of possible asymptomatic SARS-CoV infection in Hong Kong.

The case-patient is a registered nurse working in Princess Margaret Hospital, the major infectious diseases hospital that treated >600 SARS patients in Hong Kong. Within this hospital, >800 frontline staff members have participated in direct care of SARS patients, and SARS developed in 62 of these staff members. All healthcare workers working in SARS wards followed the same infection control measures, wearing a N-95 respirator, eye shield, disposable cap, water-resistant gown, and gloves. Gowns and equipment were removed before the staff left the SARS wards.

We performed serologic testing of the first 101 healthcare workers (doctors, nurses, healthcare assistants) who worked in the SARS wards but in whom SARS did not develop. The serologic testing was performed 7–8 weeks after the healthcare workers were first exposed to SARS patients.

We identified a nurse who was asymptomatic for SARS-CoV infection, worked in the SARS ward since the disease outbreak, and used full infection control procedures as recommended by the World Health Organization (WHO). The nurse performed procedures, including nasopharyngeal aspiration, handling of fecal matter, and oral feeding of SARS patients. SARS developed in six colleagues who worked in the same ward. She had unprotected exposure to a colleague who contracted SARS and required hospitalization. Serologic testing for SARS-CoV antibody was performed in the microbiology laboratory of Princess Margaret Hospital on week 8 of the nurse's SARS ward duty. The result of the test was positive by enzyme-linked immunosorbent assay. The test was repeated by the Government Virus Unit of the Department of Health, one of the reference laboratories in Hong Kong. The second test also showed a positive result with an antibody titer of 400 by immunofluorescence assay (normal: <25). We performed another serologic test on week 10 of her SARS ward duty; the result was again positive. The nurse was interviewed by two physicians and questioned about her health condition since February 2003. She did not report any symptoms typical of SARS, such as fever, chills, rigors, malaise, myalgia, cough, dyspnea, and diarrhea (1,3) during and after her SARS ward duty. She did have a mild, short-term headache, which she has had periodically for many years. She did not take sick leave since February 2003. She did not record any rise in body temperature >37°C and had a leukocyte count of 5.9×10^9/L and a lymphocyte count of 1.6×10^9/L. Results of liver and renal function tests were all normal. Reverse transcription-polymerase chain reaction results for SARS-CoV in stool, urine, throat, and nasal swabs collected during weeks 10 and 14 of her SARS ward duty were all negative. No abnormal radiologic change was identified in the lungs. She lived with four family members and had close contact with them. None of her family members contracted SARS, and all showed a negative result in the serologic testing for SARS-CoV.

We think that asymptomatic and subclinical infection of SARS-CoV exists and can result in seroconversion; however, this kind of asymptomatic seroconversion is probably uncommon. Why a person infected with SARS-CoV did not have typical symptoms, and the infectivity of an asymptomatic person is unknown. A person's genetic makeup may determine susceptibility to SARS-CoV and the final clinical outcome. We agree with Seto et al. (5) that recall bias is a concern. However, recall bias probably had little effect since the events took place recently. Moreover, the hospitalization of the nurse's infected colleague would have made her more alert and aware of symptoms of the illness.

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Hepatitis C Antibodies among Blood Donors, Senegal, 2001

To the Editor: Prevalence of chronic hepatitis C virus (HCV) among blood donors has been assessed in a few West African countries; most recent estimates range from 1.1% to 6.7% (1–4). A recent meta-analysis of studies, including a confirmation test, yielded an average prevalence of HCV infection of 3.0% (5). Until 2001, no systematic screening of HCV infection occurred among blood donors in Senegal, and blood donation legislation is still pending. We report an assessment of the proportion of blood donors from the Hôpital Principal de Dakar who had HCV antibodies in 2001.

Blood donors were all volunteers, recruited independently from the hospitalized patients and registered in a local donors association. We screened for risk factors for bloodborne infections in potential donors through a clinical examination and a confidential questionnaire. Persons with a history of jaundice or a risk behavior were excluded. Serum samples collected from blood donors from June to December 2001 were screened for HCV antibodies by a third-generation enzyme immunoassay (EIA) (HCV Murex 4.0; Abbott Laboratories, Abbott, IL). Confirmation was performed by a recombinant-immunoblot assay (INNO-LIA HCV Ab III update; [Innogenetics, Gent, Belgium]). HCV RNA was detected by a qualitative reverse transcription–polymerase chain reaction (Roche Amplicor HCV test [Hoffman-LaRoche, Basel, Switzerland]). Genotype was determined by the INNO-LiPA HCV II assay (Innogenetics). Presence of hepatitis B surface antigen (HbsAg) and alanine-aminotransferase (ALAT) level are routinely assessed, as well as HIV and human T-lymphotropic virus type l infection.

The age of the 1,081 donors ranged from 18 years to 61 years (mean 35.6 years), and 81% were men. First-time donors accounted for 31% and were younger than repeat donors (mean 30.5 years vs. 37.8 years; $p < 10^{-4}$). EIA HCV antibodies were found in 18 donors (1.6%). Immunoblot assay was positive for nine, yielding an overall prevalence of 0.8% (exact 95% confidence interval 0.4% to 1.5%). Eight of the nine were repeat donors, but the difference in prevalence compared with first-time donors did not reach statistical significance (1.1% vs. 0.3%). HCV-infected donors tended to be older than uninfected donors (mean 42.3 years vs. 35.5 years, median 46.7 years vs. 34.6 years, Mann-Whitney test $p = 0.04$), and the trend with age was significant (18–29 years 0.3%; 30–39 years 0.6%; 40–49 years 1.5%; ≥50 years 1.8%; chi-square trend = 4.39; $p = 0.03$). ALAT levels of infected study participants were in the normal range (17–55 IU). One participant had an ALAT level above normal. Genotype 2ac has been identified on line immunoassay–positive samples (three samples not tested). HBsAg was detected in 13% of the new donors. No co-infection with HCV and hepatitis B virus was found.

The prevalence of HCV antibodies in blood donors in Dakar in 2001 appears to be one of the lowest in West Africa, close to published estimates for Mauritania and Benin (1.1% and 1.4%, respectively) and lower than in other West African countries such as Ghana or Guinea, where prevalence ranges from 2.8% to 6.7% (1–4). This finding is in keeping with results of a hospital case-control study on HCV infection and liver cirrhosis or cancer, conducted in 1995 in Dakar. While that study did not identify HCV infection in 73 controls, 2 of 73 case-patients (2.7%) had HCV antibodies (6). Conversely, high HCV prevalence was found in groups at risk: antibodies were present in 12 of 15 hemodialysis patients, and HCV RNA was found in 6 of the 12 HVC antibody-positive patients (genotype 2ac, the same as in our study); 7% of a cohort of 58 HIV-1 patients receiving highly active antiretroviral therapy had a positive HCV serologic result (7,8).

In the urban setting of Dakar, HCV infection seems still to be confined to groups at risk. The contribution of HCV to chronic liver diseases has not been yet demonstrated. Approximately 15,000 blood donations are annually made in Dakar. A systematic screening of HCV antibodies in blood donors could prevent, on average, 120 bloodborne HCV infections each year. Given these data and the price of EIA and LIA, the screening cost per HCV-positive sample identified, and infection subsequently averted, is approximately 200,300 CFA (U.S.$305). This estimate is low since it includes only the marginal cost of the reagent kits. This screening cost could be reduced by discarding blood units that test positive after only one enzyme-linked immunosorbent assay (156,000 CFA or U.S.$237), at the price of nearly 3% of blood units wrongly discarded. France has demonstrated that this strategy has the best cost-effectiveness ratio, as long as the prevalence remains below 8% (9). This cost compares favorably with the cost per HIV infection averted through improvement of blood safety (range U.S.$20–U.S.$1,000), assessed in some highly HIV-prevalent southern African countries (Tanzania, Zambia, Zimbabwe) (10). The HCV-positive discarded blood units will be added to