

## SARS Patients and Need for Treatment

**To the Editor:** We read with interest the case report by Wong et al. (1). Three similar cases of serologically confirmed severe acute respiratory syndrome (SARS) were treated in our hospital; all of the patients recovered uneventfully without specific treatment. They had either negative results on polymerase chain reaction (PCR) tests for SARS-associated coronavirus (SAR-CoV), or they were admitted when such rapid diagnostic tests were not yet available; hence, SARS-specific treatment was not prescribed.

The first patient was a 35-year-old previously healthy female tourist from Guangzhou, China, who was admitted to our hospital in late February 2003. She had visited several family members who had atypical pneumonia; some eventually died from the disease. The patient had fever, chills, and dry cough approximately 1 week after exposure but experienced no myalgia, diarrhea, or shortness of breath. On physical examination, scanty crepitations were heard in her right lower chest, and chest radiographs showed right lower zone consolidation. Blood tests showed a slightly low platelet count of  $119 \times 10^9/L$  and mildly elevated alanine transaminase at 59 U/L (normal  $<55$  U/L), but total and differential leukocyte counts were normal. Tests for etiologic agents included blood and sputum bacterial cultures; sputum for acid-fast bacilli; and nasopharyngeal aspirates for influenza, parainfluenza, adenovirus, and respiratory syncytial virus. Serologic titers for *Mycoplasma*, *Chlamydia psittaci*, and *Legionella* were negative. Reverse transcription (RT)-PCR tests for SARS-CoV were not available at that time.

Oral clarithromycin and intravenous amoxicillin-clavulanate (subsequently switched to levofloxacin) were prescribed. Her high fever (tempera-

ture  $39.5^\circ C$ ) lasted for 4 days and then gradually subsided; the radiologic abnormality also improved progressively after the first week. Oxygen supplementation of 2 L/min was necessary for the first 2 days. The diagnosis of SARS was made when the patient's convalescent-phase serum sample, collected 33 days after discharge (day 45 of illness), showed an elevated anti-SARS immunoglobulin (Ig) G titer of 1:800 by immunofluorescence.

The second patient was a 34-year-old previously healthy man; his father had shared a hospital cubicle with a patient who was subsequently diagnosed with SARS. Fever (temperature  $39^\circ C$ ), chills, and rigors developed in patient 2 on December 3, 2003, approximately 4 days after his first hospital visit to his father; he had no cough or gastrointestinal symptoms. Chest radiographs showed right lower zone consolidation. Blood tests showed low platelet count of  $91 \times 10^9/L$ , elevated creatinine kinase (370 U/L), and elevated lactate dehydrogenase levels (1,060 U/L). Total and differential leukocyte counts were normal. Tests for etiologic agents of pneumonia had negative results. His fever (the highest temperature was  $39.5^\circ C$ ) subsided after day 2 of admission, with a transient spike on day 11 that coincided with a slight increase in right lower zone consolidation. Both abnormalities subsequently resolved promptly, and no oxygen supplement was necessary. RT-PCR test for SARS-CoV was not available in our hospital when he was admitted. However, SARS was diagnosed when his convalescent-phase serum, collected on day 21 of illness, demonstrated a SARS-CoV IgG titer (by immunofluorescence) of 1:3,200, from an initial baseline of  $<1:25$ , taken on day 12 of his illness. No treatment was given, since the patient had already fully recovered when the results arrived.

The last patient was a 74-year-old, previously healthy man, who had vis-

ited a sick relative; the relative was later diagnosed with SARS. Fever, chills, and cough developed in our patient 4 days later. Chest radiograph showed left lower and middle zone consolidations. Intravenous ceftriaxone and oral clarithromycin were started. Blood tests showed elevated alkaline phosphatase of 226 U/L and alanine transaminase of 126 U/L. His initial leukocyte count was  $18.8 \times 10^9/L$  with neutrophilia ( $16 \times 10^9/L$ , 85.2%) and a normal lymphocyte count of  $1.2 \times 10^9/L$ ; platelet count was normal. No causative agent was identified, including by RT-PCR test for SARS-CoV. His fever had subsided upon admission, and serial chest radiographs, liver function, and leukocyte counts showed progressive improvement without specific treatment. The diagnosis of SARS was made from two elevated SARS-CoV IgG levels of both 1:3,200 (by immunofluorescence), taken at days 5 and 24 after his admission (days 19 and 38 of illness).

Although SARS was diagnosed in these three patients retrospectively, and they were not treated with antiviral agents, they were managed in isolation wards. Patients reported adhering to droplet precautions after discharge (mainly, wearing surgical face masks when in close contact with others), and none was believed to have transmitted the virus to others.

SARS can be associated with a substantial death rate (2). Ribavirin and systemic corticosteroids were used in our hospital during the SARS epidemic. However, the efficacy of this regimen has not been proven, and concerns exist about side effects of both drugs (3,4). Some retrospective analyses suggested using lopinavir/ritonavir and integrative Chinese and Western medicine were associated with improved outcomes (5,6). In vitro (7,8) and animal (9) studies have suggested that interferon and monoclonal antibodies might have some effects on the disease. However, data

from randomized controlled trials are lacking. All of our patients had been previously healthy, with no coexisting conditions identified as poor prognostic risk factors (2,10). These three cases, together with the case of Wong et al. (1), suggested that at least a subset of SARS adult patients can have a relatively benign clinical course and uneventful recovery, without any specific treatment other than antimicrobial agents.

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9. Haagmans BL, Kuiken T, Matrina BE, Fouchier RA, Rimmelzwaan GF, van Amerongen G, et al. Pegylated interferon-alpha protects type 1 pneumocytes against SARS coronavirus infection in macaques. *Nat Med.* 2004;10:290-3.
10. Booth CM, Matuka LM, Tomlinson GA, Rachlis AR, Rose DB, Dwosh HA, et al. Clinical features and short term outcomes of 144 patients with SARS in the Greater Toronto area. *JAMA.* 2003;289:2801-9.

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## References

1. Wong RSM, Hui DS. Index patient and SARS outbreak in Hong Kong. *Emerg Infect Dis* [serial on the Internet]. 2004 Feb [cited Jan 8 2004]. Available from <http://www.cdc.gov/ncidod/EID/vol10no2/03-0645.htm>
2. Chan JW, Ng CK, Chan YH, Mok TY, Lee S, Chu SY, et al. Short term outcome and risk factors for adverse clinical outcomes in adults with severe acute respiratory syndrome (SARS). *Thorax.* 2003;58:686-9.
3. Van Vonderen MGA, Bos JC, Prins JM, Wertheim-van Dillen P, Speelman P. Ribavirin in the treatment of severe acute respiratory syndrome (SARS). *The Netherlands Journal of Medicine.* 2003;61:238-41.
4. Oba Y. The use of corticosteroids in SARS. *N Engl J Med.* 2003;348:2034-5.
5. Chu CM, Cheng VCC, Hung IFN, Wong MML, Chan KH, Chan KS, et al. Role of lopinair/ritonavir in the treatment of SARS: initial virological and clinical findings. *Thorax.* 2004;59:252-6.
6. Li J, Li SD, Du N. Clinical study on treatment of severe acute respiratory syndrome with integrated Chinese and Western medicine approach. *Zhongguo Zhong Xi Yi Jie He Za Zhi.* 2004;24:28-31.
7. Stroher U, DiCaro A, Li Y, Strong JE, Plummer F, Jones SM, et al. Severe acute respiratory syndrome coronavirus is inhibited by interferon-alpha. *J Infect Dis.* 2004;189:1164-7.
8. Sui J, Li W, Murakami A, Tamin A, Matthews LJ, Wong SK, et al. Potent neutralization of severe acute respiratory syndrome (SARS) coronavirus by a human MAb to S1 protein that blocks receptor association. *Proc Natl Acad Sci U S A.* 2004;101:2536-41.

## Occupational Malaria Following Needlestick Injury

**To the Editor:** A 24-year-old female nurse was admitted to the emergency room at Bichat University Hospital in Paris, France, on July 4, 2001, with fever, nausea, and general malaise. She had no notable medical history, except spontaneously regressive Schönlein-Henloch purpura at 9 months of age. On admission, after she was given paracetamol, her axillary temperature was 37.6°C. She was slightly jaundiced and reported a mild headache but showed no resistance to head flexion. Her abdomen was depressible but tender. Urinalysis did not show hematuria or signs of urinary infection. Biologic tests indicated normal values except the following: platelets 47.4 x 10<sup>3</sup>/μL, aspartate aminotransferase 307 U/L (normal value <56), alanine aminotransferase 239 U/L (normal value <56), total bilirubin 58 μmol/L (normal value <24), and γ-glutamyl transpeptidase 57 U/L (normal value <35). Results of an abdominal echogram were normal. Result of a blood film to identify *Plasmodium falciparum* was positive

for parasitemia at 0.038 per 100 erythrocytes. The patient was given 500 mg of oral quinine three times daily; intravenous quinine was administered 15 hours after admission because she became nauseated. Her malaise persisted for 3 days, but she did not show any signs of malaria. She recovered completely and was discharged on day 6 of hospitalization.

The patient had not traveled outside France except to the United Kingdom years earlier. She did not live near an airport, nor had she been to one recently. She had vacationed in the south of France from June 23 to June 26 but had traveled by car. She had been certified as a registered nurse on May 28 and had been working as a substitute employee at various hospitals in the greater Paris area. On June 21, 2001, she sustained an accidental needlestick injury while taking a blood sample with an 18-gauge, peripheral venous catheter that had no safety feature. She removed the catheter stylet and stuck herself as she crossed her hands to discard the stylet in a sharps container. The needlestick pierced the nurse's glove and caused a deep, blood-letting injury on the anterior aspect of the left wrist. She had no previous history of needlestick injury. She notified the hospital occupational medicine department of her injury on the day it occurred and was given a postexposure interview. In accordance with national postexposure management guidelines, she was tested for HIV and hepatitis C virus (HCV) antibody, and results were negative at baseline; her immunization against hepatitis B virus (HBV) was confirmed. The risk of infection by pathogens other than HBV, HCV, or HIV following a needlestick injury was not discussed during her postexposure interview, and the nurse was not made aware of that risk. The injured nurse did not inform the managing physician that the injury had occurred while she was drawing