Early Defervescence and SARS Recovery

To the Editor: Severe acute respiratory syndrome (SARS) is an emerging disease first recognized November 2002 (1). Previous studies show patients with probable SARS on ribavirin and steroid therapy may experience a biphasic course, with clinical symptoms and changes shown on chest x-rays increasing in the second week of disease (2). We report a patient with probable SARS who had temporary defervescence for 7 days before rapidly progressing to respiratory failure.

The patient was a 54-year-old female nursing aide for a patient with fever and pneumonia who was diagnosed with probable SARS on the basis of the criteria proposed by World Health Organization (WHO) (3). Our patient did not have underlying disease, but fever of 38.6°C developed on May 10, 2003, a total of 3 days after her last contact with the patient she was caring for. Mild myalgia was noted. She was admitted that day with suspected SARS. Initial chest x-ray results were normal. Hemogram showed a normal leukocyte count with mild lymphopenia (absolute lymphocyte count 0.84 x 10^9/L) and a normal platelet count (253 x 10^9/L). Initial serum aspartate aminotransferase (AST) and alanine aminotransferase (ALT) levels were 38 U/L and 20 U/L, respectively (normal <35 U/L). Serum creatinine kinase (CK) level was 84 U/L (normal <190 U/L). Serum C-reactive protein (CRP) level was elevated (3.49 mg/dL; normal <0.8 mg/dL). Serum sodium level was normal.

After admission, she received oral ribavirin, 1,000 mg/day. No other antimicrobial agent was administered. Her fever persisted for 2 days, and she became afebrile spontaneously on May 13, 2003. The result of reverse transcription–polymerase chain reaction (RT-PCR) for SARS-associated coronavirus (SARS-CoV) on a throat swab specimen performed on May 10 was negative. All other testing, including blood culture; virus isolation; and serologic tests for SARS-CoV, chlamydiae, mycoplasmas, rickettsiae, influenza virus, parainfluenza virus, adenovirus, respiratory syncytial virus (RSV), and coxsackie virus were also negative. She did not take any nonsteroid antiinflammatory drugs (NSAIDs) or steroids during this period. Her chest x-ray results on May 13 remained normal. Other laboratory testing on May 13 showed resolution of lymphopenia, a lower serum CRP level (2.48 mg/dL), but an elevated lactate dehydrogenase (LDH) level (627 U/L; normal <460 U/L).

During the next 4 days, she remained afebrile. Results of a repeated chest x-ray on May 16 were still normal. The serum CRP level decreased to 2.29 mg/dL. However, borderline leukopenia (4.45 x 10^9/L), borderline thrombocytopenia (167 x 10^9/L), an elevated serum CK level (238 U/L), hyponatremia (128.2 mmol/L), and a progressively elevated serum LDH level (1,138 U/L) were noted. Because she had been afebrile for 5 days, she was discharged on May 17. After discharge, she continued to take ribavirin and be quarantined at home. Unfortunately, fever and rapidly progressive dyspnea developed on May 20. On the same day, chest x-ray showed diffusely increased infiltration over all lung zones. Hemogram showed leukocytosis (leukocyte count 14.03 x 10^9/L). Serum CRP level was elevated to 12.7 mg/dL. Serum sodium level was 129.5 mmol/L. Serum AST level was 135 U/L, serum CK level was 71 U/L, and serum LDH level was 1,719 U/L. All blood cultures and sputum culture for bacteria yielded nothing.

She was intubated on May 21 for respiratory failure. Under the assumption of probable SARS, she was given high-dose methylprednisolone (120 mg/day), and her clinical condition stabilized soon after. The results of RT-PCR for SARS-CoV on a throat swab specimen performed on May 21 were positive. The results of immunofluorescent assays testing for immunoglobulin (Ig) M and IgG against SARS-CoV (performed in the research laboratory at National Taiwan University Hospital on May 21 and 27) were all positive (both IgM titers >1:10; both IgG titers >1:1,000). Sputum culture and Gram stain were both negative. Urine tests were also negative for pneumococcal and *Legionella* antigens. Other serologic tests, including those for chlamydiae, mycoplasmas, rickettsiae, influenza virus, parainfluenza virus, adenovirus, RSV, and coxsackie virus were still negative. The ventilator was removed on June 9.

A previous report pointed out the great variety in the clinical course of SARS (4). We emphasize that even a patient with suspected SARS who became afebrile in the first week and remained so for 7 days without steroid or NSAID treatment still risks deterioration in the second week, as long as some laboratory values remain abnormal. Therefore, defervescence, even up to 7 days, may not be the single indicator for discharging SARS patients. Obtaining normal results for previously abnormal laboratory parameters, including hemogram, CRP, CK, AST, ALT, and LDH levels should be considered when deciding whether a patient can be safely discharged (5).

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Babesiosis in Fairfield County, Connecticut

To the Editor: Human babesiosis, caused by *Babesia microti*, was initially described in the eastern United States in 1970 in a woman vacationing on Nantucket Island, Massachusetts (1). With few exceptions, almost all subsequent cases were recorded from islands in the northeastern United States and Cape Cod, Massachusetts (2), until this illness was diagnosed in 13 patients living in New London County in southeastern Connecticut (3,4). *B. microti* was isolated from white-footed mice, *Peromyscus leucopus*, captured from 1988 to 1990 in the yards of patients. Babesiosis also was diagnosed in persons living in Wisconsin (5) and in New Jersey (6) who acquired the organism locally. The number of cases of babesiosis reported by health departments on their Web sites and by personal communication in Massachusetts, Rhode Island, and New York State, was 330 from 1988 to 2002, 121 from 1994 to 2002, and 542 from 1986 to 2001, respectively. The number of cases reported by the New York City Health Department from 1991 to 2000 was 75.

From 1991 to 2000, babesiosis was diagnosed in 230 persons residing in New London County and adjacent Middlesex County, Connecticut (7). Fifty-three additional cases were reported in five other counties in Connecticut, but epidemiologic data did not indicate that these infections likely were acquired within Connecticut. We now note a new and distinct geographic focus by reporting the isolation of *B. microti* from rodents captured in the yards of two patients in whom babesiosis was diagnosed at Greenwich Hospital in 2002. These patients lived in Greenwich, Connecticut, which is located in Fairfield County in the extreme southwestern part of the state. Neither patient had traveled outside of the immediate area of Greenwich, Connecticut, before onset of illness. We also trapped rodents in the yards of two additional patients in whom babesiosis was diagnosed. These two patients had traveled to Rhode Island shortly before becoming ill. Patients became ill from June 23 to July 7, 2002, and none reported a tick bite.

Attempts to trap small mammals on the properties of the four patients were made on July 22, 23, and 29, 2002. Rodents were captured in Sherman box traps baited with peanut butter and apple. Approximately 0.3 mL of blood was drawn from the heart of each animal into a syringe coated with heparin or uncoated. Blood was kept on ice in the field and then returned to the laboratory. A 3- to 5-week-old male Syrian hamster was injected intraperitoneally with 0.1 mL of each blood sample.

Blood smears were obtained from a drop of blood taken from the tail of each hamster on weeks 3 to 6 after injection. Blood cells were stained with Giemsa and examined for *B. microti* at a magnification of 1,008x. Hamsters were considered uninfected when no parasites were found in 75 fields of stained erythrocytes.

B. microti was isolated from rodents captured at the residences of two of the patients who did not travel outside of the Greenwich area 6 weeks before onset of illness. Blood from two of three white-footed mice and from the two eastern chipmunks, *Tamias striatus*, captured in the yards of the patients, produced infections in injected hamsters. Infections did not develop in hamsters injected with blood from 10 white-footed mice captured at the residences of two patients who visited Wakefield and Charlestown, Rhode Island, shortly before becoming ill.

B. microti is prevalent in rodent populations in Greenwich, Connecticut, and causes human disease. Establishing evidence of *B. microti* in rodents and documenting this protozoan parasite as the cause of human disease in Greenwich are important. Relatively high populations of the vector tick, *Ixodes scapularis*, are present in Greenwich and nearby towns. In 2002, the health departments of Greenwich, Stamford, New Canaan, and Darien submitted 1,671 *I. scapularis* ticks removed from persons to the Connecticut Agricultural Experiment Station for identification and testing for *Borrelia burgdorferi*. Two hundred and thirty cases of Lyme disease were reported from these four towns in 2002 (Connecticut Department of Public Health, unpub. data). With such extensive human exposure to ticks and a relatively large number of Lyme disease cases in these four towns and elsewhere in Fairfield County, the number of cases of babesiosis is likely to increase appreciably in the future.