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Severe Acute Respiratory Syndrome (SARS)

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**ABSTRACT**

Severe acute respiratory syndrome (SARS) first appeared in November 2002 and ultimately resulted in 8096 probable human infections and 774 deaths worldwide. By July 2003, the global outbreak was declared over. A new coronavirus, SARS-associated coronavirus (SARS-CoV), was identified as the causative agent; this virus appeared to have a zoonotic origin, as genetically similar coronaviruses have been identified in several animal species. The global response to the outbreak was extensive. Within a short period, the pathogen had been identified, new diagnostic tests were developed, surveillance systems were created, infection control and prevention measures were instituted, and transmission among humans stopped. It is unclear if and when person-to-person SARS-CoV transmission will reappear. However, procedures have been established by public health organizations, including the World Health Organization (WHO) and the Centers for Disease Control and Prevention (CDC), to help guide diagnosis, reporting, surveillance, and prevention.

**GEOGRAPHIC DISTRIBUTION AND MAGNITUDE OF DISEASE BURDEN**

**Background**

In 2002 and 2003, a previously unknown infectious agent caused a widespread, global outbreak of life-threatening respiratory infections. The illness was called *severe acute respiratory syndrome* (SARS) and was one of the first emerging infections in recent history to test the global public health response. Strategies used to prevent and control SARS helped to shape future public health response to infectious disease emergencies such as the 2009 H1N1 influenza pandemic. SARS emerged in Guangdong Province, China, in mid-November 2002 and was first officially reported to WHO in February 2003. In mid-March 2003, WHO issued an alert calling attention to several outbreaks of severe atypical pneumonia in Hong Kong, Hanoi, and Singapore. Many of the initial SARS infections were traced to a guest staying at a Hong Kong hotel, and global spread occurred quickly with multiple outbreaks reported in China, Southeast Asia, Europe, and North America. By mid-April 2003, the causative agent was identified as a new coronavirus, SARS-CoV. An unprecedented global outbreak response (including the implementation of surveillance systems, epidemiologic studies, appropriate infection control measures, and development of laboratory diagnostics) was swiftly initiated. In July 2003, WHO announced that the SARS outbreak was over. Although a few laboratory-associated SARS infections were reported in Asia after the outbreak was declared over, no infections have been reported worldwide since early 2004. Almost all persons with SARS were reported from China, Hong Kong, Taiwan, Singapore, or Toronto, but overall, as of December 2003, WHO had received reports of SARS from 29 countries and regions: 8096 persons with probable SARS, resulting in 774 deaths, a case-fatality rate of 9.6%. In the United States, eight infections were documented by laboratory testing, and an additional 19 probable infections were reported. The syndrome arose and vanished within several months, and it is unclear when or if SARS will return.

**Coronaviruses**

Coronaviruses are enveloped, single-stranded positive strand ribonucleic acid (RNA) viruses that infect a wide spectrum of mammals and birds. There are three coronavirus groups: groups I and II affect mammals and group III affects birds. In humans, coronaviruses are primarily associated with upper respiratory infections. During the 2002-2003 SARS outbreak, several strains of a coronavirus unrelated to previously described coronaviruses were identified and isolated from clinical samples, including respiratory secretions, urine, and autopsy tissues. This new virus was identified as a novel group II coronavirus and named *SARS-associated coronavirus*; of all coronaviruses, it is responsible for causing the most severe human disease.

Bats have been identified as natural reservoirs of SARS-CoV–like viruses and are most likely the natural reservoirs for SARS-CoV; therefore SARS is a zoonosis. Studies of animal ecology and virus evolution have revealed that SARS-CoV–like viruses are also present in other animals, including those commonly traded at live animal markets in southern China (e.g., masked palm civet, raccoon dog, red fox). At this time, it is unclear if these animals are susceptible hosts or carriers of SARS-CoV, but recent research indicates that the masked palm civet probably served as intermediate host between bats and humans during the 2002-2003 outbreak. Sequence analyses of SARS-CoV have shown that SARS-CoV–like viruses that infect masked palm civets are very similar (genome identity >99.6%) to the SARS-CoV that infected humans in the 2002-2003 SARS outbreak, suggesting that the virus had only recently circulated in the masked palm civet. The increased prevalence of SARS-CoV immunoglobulin G (IgG) antibodies among animal traders compared with a control group (13% versus 1% to 3%) further supports this theory. In addition, the absence of SARS-CoV antibodies in the general population without clinical evidence of SARS suggests that SARS-CoV did not widely circulate before the 2002-2003 SARS outbreak.

**Transmission**

The estimated incubation period for SARS is 2 to 10 days (median 5 to 6 days). The virus is detected at low levels in
respiratory secretions during the initial days after the onset of illness, and peak viral levels occur during the second week of illness (e.g., 10 days). This viral replication phase is followed by the immune hyperreactive phase, which occurs when disease severity increases and viral load decreases. Disease progression is variable, and not all patients progress to the final pulmonary destruction phase.

The primary route of SARS-CoV transmission is via the respiratory tract; during close contact with an infected patient, respiratory droplets may come into contact with mucous membranes either directly or indirectly through contaminated fomites. Studies have determined that SARS-CoV can remain stable on environmental surfaces for several days, although the virus can be easily inactivated by disinfectants. The virus has been isolated from respiratory secretions, saliva, tears, urine, and stool. Viral shedding generally does not persist beyond 4 weeks, except in stool, in which the virus can be detected by reverse transcription-polymerase chain reaction (RT-PCR) for longer than a month. Isolation of the virus more than a month after the onset of illness is rare. Virus detection in nasopharyngeal specimens using quantitative RT-PCR found that the level typically peaks during the second week of illness, often when severely ill patients are seeking medical care. As patients improve clinically and the viral load decreases, transmission of the virus also decreases. Unlike other respiratory viral infections, such as influenza, transmission before symptom onset has not been reported. During the recent SARS outbreak, transmission occurred primarily in hospitals, less so within households, and to an even lesser extent within communities.

**RISK FACTORS**

In the 2002-2003 outbreak, the primary risk factor was contact with a person who was infected with SARS-CoV. Twenty-one percent of all reported SARS-CoV infections occurred among healthcare workers. Nosocomial transmission of SARS-CoV was common early in the outbreak but subsequently decreased significantly as a result of early diagnosis and reinforcement of infection control practices. Nosocomial spread is theorized to occur via aerosolization during patient procedures, such as intubation and bronchoscopy. Transmission has also been documented on an airplane, in an apartment complex (probably secondary to faulty plumbing and aerosolization of fecal matter), and among laboratory workers handling SARS-CoV. Transmission of the virus has not been reported via food-borne or waterborne sources, nor from an infected patient whose fever had resolved more than 14 days previously.

As seen with other infectious diseases, environmental and host factors influence the risk of transmission. Although the virus was initially thought to be highly infectious, the rate of secondary transmission of SARS-CoV is estimated to be low to moderate. Transmission modeling studies have estimated that each patient will infect an average of three persons. However, some SARS-infected patients designated as “superspreaders” have been documented to have very high secondary transmission rates (infecting an average of 36 contacts [range 11 to 74 contacts]), a phenomenon not unique to SARS. Transmission of SARS-CoV by superspreaders primarily occurred in hospital settings and was associated with a greater number of close contacts, delayed diagnosis, older age, more severe illness, and poor infection control practices.

**CLINICAL FEATURES**

Severity of disease among SARS patients varies from asymptomatic infection to fatal acute respiratory distress syndrome (ARDS). Seroprevalence surveys have documented asymptomatic infection, especially among animal traders in Guangdong, China, but overall, asymptomatic or mild disease is relatively uncommon (<1%).

SARS affects persons of all ages; however, most infections occur among adults (median age approximately 42 to 57 years). Infections among children, especially those younger than 12 years of age, are uncommon. Compared with adults, the disease is considerably less severe among children, and the outcome is much more favorable. Infections during pregnancy have been documented, with an increased risk of spontaneous abortion, preterm labor, severe pulmonary disease, and death. No reports of perinatal transmission have been noted.

**Figure 89-1** Mechanism of acute respiratory distress syndrome.
The initial symptoms of SARS are nonspecific and consistent with an influenza-like illness. A prodrome that includes fever, headache, chills, rigors, malaise, and myalgias occurs approximately 1 to 2 days after exposure (Figure 89-2). Nearly all patients report fever (with temperatures frequently exceeding 101°F), which typically precedes other prodromal symptoms but can also occur after the prodrome. The elderly and those with a history of chronic comorbid conditions, such as diabetes mellitus or chronic renal failure, may have atypical presentations (e.g., lack of fever).

Although SARS primarily affects the pulmonary system, respiratory symptoms (typically including nonproductive cough and shortness of breath) appear more often during the second week of illness. In one reported patient series, gastrointestinal symptoms, primarily diarrhea, were prominent (73%), with high-volume diarrhea occurring in the second week of illness. In a separate report, diarrhea, nausea, and vomiting were less common (<25%). Mucus and blood in stool are uncommon, and the diarrhea is often self-limiting. Lymphadenopathy, rhinorrhea, sore throat, rash, and purpura are unusual.

In persons with SARS, inspiratory crackles at the lung bases and, less commonly, wheezing may be noted on auscultatory examination. Initially a consistent finding is the paucity of auscultatory findings relative to the degree of abnormalities displayed on chest radiographs. By the second week of illness, clinical deterioration may occur, with pneumonia and hypoxemia that necessitate hospitalization. During the 2002-2003 outbreak, respiratory failure and ARDS were the most common reasons for admission to an intensive care unit (ICU). In several studies, approximately 20% to 30% of patients hospitalized with SARS were admitted to an ICU; about 75% of ICU patients required mechanical ventilation.

Initial chest radiographic findings may be unremarkable or indistinguishable from those of other causes of infectious pneumonia in up to 30% of patients. However, serial chest radiographs and high-resolution computed tomography (CT) scans may offer valuable information during evaluation of a patient with suspected SARS, as abnormalities appear in a large proportion of patients by day 7 to 10 of illness. Typical chest radiographs have a ground glass appearance with focal opacities or consolidations in the peripheral lower lung fields, which often progress to bilateral patchy consolidations (Figure 89-2). Peripheral lung involvement was a very common finding in most case studies of the 2002-2003 outbreak, and pulmonary cavitation, hilar lymphadenopathy, nodular infiltrates, and pleural effusion were unusual (Figure 89-3).

Evidence of extrapulmonary dissemination of SARS-CoV can be found by laboratory and pathologic diagnostic methods. The virus has been detected in several extrapulmonary organs, including the gastrointestinal tract, kidneys, liver, and spleen. Studies to improve our understanding of SARS pathogenesis and immune response are ongoing.

**LABORATORY FINDINGS**

Common laboratory findings in patients with SARS include moderate lymphocytopenia with a low to normal white blood cell count (primarily caused by a decrease in T-cell lineages); mild thrombocytopenia; increased serum lactate dehydrogenase (i.e., more than three to five times the upper limit of normal); and elevated serum hepatic transaminases. Decreased CD4 and CD8 T-cell counts can be significant: up to 30% of infected patients have a CD4 T-cell count of <200 cells/mm³. Elevated serum creatinine phosphokinase (noncardiac) and electrolyte...
abnormalities, including hypocalcemia, hypokalemia, hypomagnesemia, and hypophosphatemia, are also commonly seen in SARS.

**DIAGNOSTIC APPROACH**

It is important to note that since 2004 there has been a worldwide absence of human-to-human transmission of SARS-CoV. As a result, resources for the laboratory diagnosis of SARS infection are limited, and testing is reserved for situations in which a high level of suspicion exists for possible SARS-CoV infection. In the United States, the CDC has developed guidelines for the clinical and public health management of SARS, including specific criteria and algorithms for laboratory testing (see the section on prevention and control for a detailed explanation).

In response to the SARS outbreak, laboratory diagnostics were rapidly developed to detect the newly identified coronavirus. Methods of choice currently include detection of SARS-CoV-specific antibodies in serum using enzyme immunoassays (EIAs) or immunofluorescence assays (IFAs) and detection of SARS-CoV RNA in clinical specimens using real-time RT-PCR.

Other diagnostic methods, such as virus isolation, electron microscopy, and immunohistology, are also available but are not routinely used because of specific technologic requirements and safety issues (e.g., virus isolation requires a Biosafety Level 3 laboratory).

The virus that causes SARS can be detected from a number of clinical specimens including respiratory fluids and tissue (i.e., nasopharyngeal and oropharyngeal [NP/OP] swab, nasal aspirate, sputum, bronchoalveolar lavage [BAL] specimen or lung tissue) and from serum, stool, urine, and hepatic tissue. To optimize laboratory diagnosis, the type of specimen obtained and the timing of specimen collection relative to illness onset are important. In the 2002-2003 SARS outbreak, rapid detection of SARS was initially hampered by several factors: diagnostic assays had to be developed, high-level biosafety was required for working with the live virus, and low viral load in clinical specimens and the time required by a host to mount antibody limited the sensitivity of assays to detect early SARS-CoV infection.

During the outbreak, studies of the first-generation quantitative RT-PCR assay for detection of virus levels in respiratory specimens found that SARS-CoV RNA was detected in less than

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**Figure 89-3** SARS is a cause of noncardiac pulmonary edema. The infection causes the lung’s capillaries to leak more fluid than normal into the air sacs (alveoli).
50% of specimens obtained in the first 4 to 5 days of illness and that viral load peaked by about 10 to 12 days before gradually declining. Additional studies confirmed that after 7 to 10 days, RNA was detectable by RT-PCR in the majority of both respiratory and stool specimens. Second generation RT-PCR assays, developed after the outbreak ended, can detect SARS-CoV RNA in more than 80% of respiratory specimens within the first 3 days of symptom onset. When upper respiratory tract specimens are negative, sputum and BAL specimens may be useful for diagnosis, as viral loads may be greater in the lower respiratory tract. In stool, RT-PCR assays are able to detect SARS-CoV RNA for more than 4 weeks after illness onset; however, virus has not been isolated from stool after the third week.

Serologic testing is a useful tool for diagnosis because it permits detection of anti-SARS antibodies, as well as helping to confirm or exclude a SARS diagnosis and better characterize the SARS immune response. Host immune response includes the development of IgG and IgM SARS-CoV–specific antibodies as well as anti–SARS-CoV neutralizing antibodies. Longitudinal studies conducted after the 2002-2003 outbreak using IFA and/or EIA on specimens obtained from outbreak-related SARS patients showed that in the first 7 days of illness, only about 15% had detectable anti–SARS-CoV IgG or IgM. However, by the second week of illness, IgG was detected in approximately 40% to 50% of patients and IgM in about 40% to 65%. More than 90% of patients had detectable anti–SARS-CoV IgG by day 28 after illness onset. This milestone is important because an undetectable anti-SARS antibody in a serum specimen obtained more than 28 days after onset of illness is one of three exclusion criteria recommended by the CDC when defining an infection as a probable SARS case. A 3-year study that followed the progression of antibody titers in 56 patients with SARS found that levels of anti–SARS-CoV IgG peaked 4 months after onset of illness, then decreased; 3 years after infection, IgG was still detectable in 26% of patients. Second-generation immunoassays are being developed, some focusing on recombinant SARS-CoV nucleoprotein antigens. However, cross-reactive antigenic epitopes between SARS-CoV and other human coronaviruses (some of which are newly discovered and also cause respiratory illness) may complicate serologic test results and merit continued investigation.

A diagnosis of SARS should not be based on a single reactive laboratory test. Any reactive result should be confirmed by a laboratory that participates in the WHO SARS International Reference and Verification Laboratory Network. Because of apparent lack of circulating SARS-CoV in humans, a single reactive serologic test may support a diagnosis, but the positive predictive value of serologic testing is low, and confirmation by an experienced laboratory is still recommended. Test results that suggest a diagnosis of SARS must be evaluated in the context of clinical findings, exposure risk factors, and epidemiologic data. The positive predictive value of a laboratory diagnosis increases with the collection and testing of multiple and different specimen types.

**CLINICAL MANAGEMENT AND DRUG TREATMENT**

The clinical management of SARS primarily relies on providing supportive care for the acute respiratory illness and its complications and preventing and treating secondary bacterial infections. During the 2002-2003 SARS outbreak, ribavirin and corticosteroids were used with little apparent success. The effectiveness of other therapeutic options, such as interferon, intravenous immunoglobulin, and antiviral drugs is poorly understood. Several laboratories worldwide are conducting research on SARS-CoV to improve our understanding of the virus and its pathogenesis, which may lead to possible future treatment options. Currently no effective vaccine exists.

**PROGNOSIS**

Approximately 30% of patients with SARS clinically improve within a week or two of the onset of their illness, whereas 70% develop persistent fever and worsening respiratory symptoms and may require hospitalization; some will be admitted to an ICU. The length of hospital stays has varied, but several studies have reported a median length of stay of approximately 2 weeks.

In hospitalized patients, common complications of SARS include cardiovascular abnormalities (e.g., hypotension, tachycardia) and hepatic dysfunction. Deep vein thrombosis (DVT) has been less common, but in one Singapore case series 30% of hospitalized patients had evidence of a DVT. Disseminated intravascular coagulation, acute renal failure, and neurologic and other complications are uncommon.

In the 2002-2003 SARS outbreak, poor outcome was associated with increasing age and the presence of comorbid conditions (e.g., diabetes mellitus, hypertension, cardiovascular disease, chronic renal disease). Overall crude mortality rates ranged from about 4% to 15%, with age-specific mortality rates highest among older adults. Mortality rates were higher in those aged 60 years or older (43%) as compared with those younger than 60 years of age (13%). Most SARS patients who survive have a complete, sometimes prolonged recovery, but some severely ill patients have reported long-term decreased pulmonary function.

**PREVENTION AND CONTROL**

It is unclear when or if the world will experience another SARS epidemic. In the absence of person-to-person SARS-CoV transmission worldwide, recommendations have been published by several public health organizations, including WHO and the CDC. In the United States, the CDC has developed several documents that provide guidance on surveillance, clinical and laboratory evaluation, and reporting of suspected SARS infections (www.cdc.gov/ncidod/sars/). Being familiar with the clinical features of SARS-CoV disease, assessing travel history and exposure risk, and recognizing unusual clusters of unexplained pneumonia can help maximize early detection. In the absence of person-to-person transmission, public health and healthcare personnel should be aware of specific settings that should raise suspicion for SARS-CoV infection (www.cdc.gov/ncidod/sars/absenceofsars.htm). These situations include persons who are hospitalized for radiographically confirmed pneumonia or ARDS without an identifiable cause and who have one of the following three risk factors in the 10 days before the onset of illness:
• Travel to mainland China, Hong Kong, or Taiwan, or close contact with an ill person with a history of recent travel to one of these areas
• Employment in an occupation associated with a risk for SARS-CoV exposure (e.g., healthcare worker, worker in a laboratory that contains SARS-CoV)
• Illness in association with a cluster of atypical pneumonia without an alternative diagnosis

Clinicians evaluating patients who fit one of these three criteria should implement appropriate infection control measures, contact the local or state health department, and continue with a diagnostic evaluation. This evaluation should include testing for other respiratory pathogens (e.g., influenza, respiratory syncytial virus, Streptococcus pneumoniae, Legionella species). If no alternative diagnosis has been made after 72 hours or if a high index of suspicion for SARS exists, the clinician and health department should consider SARS-CoV testing and contact the CDC for consultation. Currently, laboratory-acquired SARS infection remains a possible scenario but a remote one, as adherence to strict biosafety and laboratory policies has significantly reduced this risk. The CDC is available for consultation and testing and has made guidelines for laboratory personnel working with SARS-CoV available (www.cdc.gov/ncidod/sars/guidance/f/pdf/app6.pdf).

Globally, SARS is one of a select few conditions that has been designated as immediately reportable by International Health Regulations. The local or state health department should be promptly notified if a suspected SARS case is identified. Prompt case detection, implementation of infection control measures including patient isolation and standard and droplet precautions (www.cdc.gov/ncidod/sars/guidance/i/pdf/i.pdf), and contact tracing have been shown to reduce transmission. Additional infection control measures should be instituted depending on the setting (e.g., healthcare, home, community) (www.cdc.gov/ncidod/sars/guidance/i/pdf/i.pdf).

One critical lesson learned from the 2002-2003 SARS outbreak is the apparent need for prompt collaboration and open communication among local, national, and international health agencies. Early diagnosis, timely reporting, implementation of infection control measures, and continued research, including research regarding treatment and vaccine development, will help identify and control possible future SARS outbreaks.

EVIDENCE

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Centers for Disease Control and Prevention (CDC): Supplement I. Infection control in healthcare, home, and community settings. Available at: www.cdc.gov/ncidod/sars/guidance/f/pdf/i.pdf. Accessed September 15, 2010. This document provides infection control recommendations for different settings, including the hospital, home, and community. Implementation of infection control measures is critical for reducing transmission. Also included are detailed recommendations for preparedness planning, and infection control guidelines for hospitalized SARS patients (e.g., standard and droplet precautions, isolation), healthcare workers (e.g., nurses, emergency medical services), and persons in the community (e.g., contacts, family members).

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