

Microbiologic Characteristics, Serologic Responses, and Clinical Manifestations in Severe Acute Respiratory Syndrome, Taiwan¹

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The genome of one Taiwanese severe acute respiratory syndrome-associated coronavirus (SARS-CoV) strain (TW1) was 29,729 nt in length. Viral RNA may persist for some time in patients who seroconvert, and some patients may lack an antibody response (immunoglobulin G) to SARS-CoV >21 days after illness onset. An upsurge of antibody response was associated with the aggravation of respiratory failure.

In November 2002, cases of a life-threatening and highly contagious febrile respiratory illness of unknown cause were reported from Guangdong Province in southern China, followed by reports from Vietnam, Hong Kong, Singapore, Canada, the United States, and other countries (1–4). This illness was identified as a new clinical entity and designated as severe acute respiratory syndrome (SARS) in late February 2003. This disease has a high propensity to spread to healthcare workers and household members and may cause outbreaks in the community (1–4). Recent reports have demonstrated that a novel coronavirus, SARS-associated coronavirus (SARS-CoV), is associated with the pathogenesis of SARS (5–7).

Laboratory diagnostic tests to analyze clinical specimens for SARS-CoV include reverse-transcriptase polymerase chain reaction (RT-PCR) specific for RNA and detection of specific antibody by using indirect fluorescence antibody and enzyme-linked immunosorbent assays (8,9). However, data on the timing and intensity of serologic responses after illness onset and the association of these responses with clinical manifestations of the disease are lacking.

In Taiwan, the first case of SARS occurred in a businessman working in Guangdong who was admitted to National Taiwan University Hospital (NTUH) on March 8, 2003. As of May 18, 2003, a total of 308 probable cases of SARS were reported by the Center for Disease Control, Department of Health, Taiwan (10).

The Study

This study included seven Taiwanese patients, treated at the National Taiwan University Hospital from March 8 to May 3, 2003, whose illness met the recent Centers for Disease Control and Prevention (CDC) and World Health Organization (WHO) case definition for probable cases of SARS (11,12). The patients were 26–53 years of age, and six were men. The incubation period ranged from 2 to 12 days. Of the seven patients, four had recently returned from China: two patients (patients 1 and 7) from Guangdong Province and two (patients 5 and 6) from Beijing. In addition, two family members (patients 2 and 3), and one healthcare worker (patient 4) were from a cluster, which had household or healthcare contact with a SARS patient, and two patients (patient 5 and 6) were from another cluster, which had close contact with a SARS patient in an airplane.

All patients had fever (body temperature >38°C) and dry cough. Other symptoms included malaise (five patients), myalgia (five patients), and rigor (four patients). All but one patient (patient 7) had loose stools or diarrhea 2–10 days after febrile episodes, and five, including the four cluster A patients, had aggravating diarrhea 9–14 days after febrile episodes. The mean interval between onset of symptoms and hospitalization was 7.3 days (range 4–12 days).

Pneumonia developed in all seven patients, acute respiratory distress syndrome (ARDS) developed in four (patients 1, 2, 3, and 6), and ventilator support was given 10–12 days after the onset of illness. Pancytopenia compatible with hemophagocytosis syndrome developed in

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patient 2. Five patients (patients 2, 3, 4, 5, and 6) received ribavirin, intravenous corticosteroid (methylprednisolone, 2 mg/kg/d), and intravenous immunoglobulin (IVIg, 1 gm/kg/d for 2 days). Interstitial pneumonia developed in patient 7, who responded well to ribavirin and antibiotic treatment. All patients survived.

Urinary antigen detection for *S. pneumoniae* and *Legionella pneumophila* serogroup I was negative in all seven patients. Serum from patient 5 was positive for *Mycoplasma pneumoniae* immunoglobulin (Ig) M (enzyme-linked immunosorbent assay [ELISA]) antibody with a fourfold increase in complement fixation (CF) antibody titer in acute- (<1:40) and convalescent-phase sera (1:160). An elevated *Chlamydia pneumoniae* CF antibody (1:32) but negative reaction for *C. pneumoniae* IgM (ELISA) antibody was found in the acute-phase serum sample from patients 1 and 6 and in the acute- (1:32) and convalescent-phase serum (1:32) samples from patients 5 and 7. The antibody titers of acute- and convalescent-phase serum samples for *C. pneumoniae*, *C. trachomatis*, *C. psittaci*, and *L. pneumophila* in the other patients showed no significant increase. Five patients (patients 1, 2, 4, 5, and 6) had elevated CF antibody levels ($\geq 1:16$) against parainfluenzavirus 1, 2, or 3. Cultures for influenza virus, parainfluenzavirus, mumps, respiratory syncytial virus, adenovirus, enterovirus, herpes simplex virus, varicella-zoster virus, and cytomegalovirus were negative from various clinical samples of these patients.

Nucleic acid was extracted from the sputum and serum samples and the infected Vero E6 cells by using a viral RNA kit (QIAamp, Qiagen Inc., Valencia, CA). Reverse transcription polymerase chain reaction (RT-PCR) for SARS-CoV was performed with 3 sets of primers (IN-6 and IN-7; Cor-p-F1 and Cor-p-R2; and BNIinS and BNIAs) developed by CDC and WHO Network Laboratory. The RT-PCR products were analyzed, and the unique fragment was cloned and sequenced (6,11). RT-PCR test results for SARS-CoV were positive in oropharyngeal swabs from patients 6 and 7; sputum from patients 1, 2, 3, 4, and 5; and serum specimens from patients 1, 2, 3, 4, 5, and 7. Cultures of all oropharyngeal swabs and serum specimens were negative.

Cytopathic effects in the Vero E6 cells were first found between day 3 and day 4 after injection of serum specimens from patients 3 and 4. The initial cytopathic effect was focal, with cell rounding, and was followed by cell detachment. Similar cytopathic effects developed rapidly (between day 2 and day 3) after subculture.

Ultra-thin sections were prepared for electron microscopy by fixing a washed infected Vero E6 cell pellet with 2.5% glutaraldehyde and embedding in Spurr's resin. The SARS-CoV (range 60–80 nm in diameter) was identified by electron microscopy (Figure 1 A and B). RT-PCR from the infected Vero-E6 cells identified the same amplicon. Sequences of the amplicons from all patients were identical and were also identical to those from infected Vero E6 cells.

The genome of the SARS-CoV (TW-1) (GenBank accession no., AY291451) strain from patient 3 was 29,729 nt in length. A comparison of TW1 sequences to the sequences described previously is summarized in the Table. The number of nucleotide differences between this TW1 isolate and the Urbani (AY278741), TOR-2 (AY274119), HKU-39849 (AY278491), and CUHK-W1 (AY278554) strains was 6, 3, 12, and 10, respectively.

IgG antibody to the SARS-CoV was detected by a standard indirect fluorescence antibody assay (IFA) with serial serum specimens from the seven patients. Spot slides for IFA were prepared by applying the suspension mixed with SARS-CoV-infected Vero E6 cells from one patient (patient 4) and uninfected cells. Slides were dried and fixed in acetone. The conjugates used were goat antihuman IgG conjugated to fluorescein isothiocyanate (Organon Teknika-Cappel, Turnhout, Belgium). The starting dilution of serum specimens was 1:25 (5). Ten serum samples obtained from 10 pregnant women during routine prelabor check-ups were used as control sera. Two IVIG products, one domestic (from Taiwanese donors) and one imported (Bayer, Leverkusen, Germany), were also tested for the presence of antibody.

All serum samples from the 10 pregnant women and the two IVIG products were negative for IgG antibody (<1:25) to SARS-CoV. Six patients had detectable IgG antibody to SARS-CoV during the course of illness, and all of them

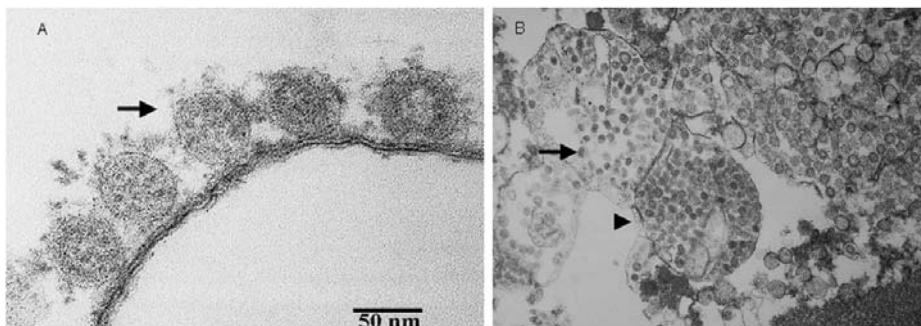


Figure 1. Thin-section electron micrograph of severe acute respiratory syndrome-associated coronavirus grown in Vero E6 cells. Panel A shows extracellular viral particles (arrow) lining the surface of the plasma membrane. Some spikes projecting from the envelope of the virus are seen. Panel B shows numerous spherical coronavirus particles (arrow) within dilated cytoplasmic vacuoles (arrowhead).

Table. Nucleotide base differences among the TW-1, TOR-2, HKU-39849, CUHK-W1, and the Urbani sequences of SARS-CoV

Base	SARS-associated coronavirus sequence					A. a. change ^b TW1/Urbani
	TW-1	TOR-2	HKU-39849	CUHK-W1	Urbani	
2,601	T	T	C	T	T	Val/Val
3,165	G	A	A	A	A	Ser/Ser
7,746	G	G	T	T	G	Pro/Pro
7,919	C	C	C	C	T	Ala/Val
9,404	T	T	C	C	T	Val/Ala
9,479	T	T	C	C	T	Val/Ala
16,622	C	C	C	C	T	Ala/Ala
17,564	T	T	G	G	T	Asp/Glu
17,846	C	C	T	T	C	Arg/Arg
19,064	A	A	G	G	G	Glu/Glu
21,721	G	G	A	A	G	Gly/Asp
22,222	T	T	C	C	T	Ile/Thr
23,220	T	G	T	T	T	Ser/Ala
24,872	T	T	T	T	C	Leu/Leu
25,298	G	A	G	G	G	Gly/Arg
26,867	T	T	T	T	C	Ser/Pro
27,827	T	T	C	C	T	Cys/Arg

^aSARS, severe acute respiratory syndrome.

^bIndicates a base difference resulting in an amino acid change.

had at least fourfold elevation of antibody levels in acute- and convalescent-phase serum samples (peak levels range 1:400– \geq 1:1600) (Figure 2). Antibody titers ($>$ 1:25) of these six patients could be detected 9–18 days (mean 12.3 days) after the onset of illness. The antibody titer increased to a plateau 4–10 days after the appearance of antibody. The high antibody levels might last for 1 to $>$ 2 months after onset of illness (Figure 2). One previously healthy patient (patient 7) with positive SARS-CoV RNA by RT-PCR from both sputum and serum specimens had no detectable antibody to SARS-CoV in serum specimens obtained 7, 10, 14, and 24 days after illness onset. Although the antibody levels reached a plateau in all patients, viral RNA persisted in the serum samples from patients 1 and 2 and sputum from patients 1 and 4 for 19 to 29 days after onset of their illness.

Although four patients had received ribavirin, corticosteroid, and IVIG treatment in the early stage of the disease, antibody was detected as early as 10–12 days after the onset of illness. The peak level of antibody was 1:400 in patients 2 and 6, 1:800 in patient 3, and \geq 1:1600 in patient 1.

Conclusions

Serologic study indicated that the antibody to SARS-CoV appeared as early as 9 days after disease onset and that a high level of antibody could last for 1–2 months after disease onset. Previous reports indicated that the mean time for IgG seroconversion was 20 days and may start as early as 9–10 days. Our finding supported the results of Peiris et al. (7,12). Levels and appearance of antibody to SARS-CoV did not seem to be influenced by the use of

ribavirin and immunosuppressive or immunomodulatory agents (corticosteroid or IVIG, a blood product prepared from the serum of 1,000 to 15,000 donors per batch) (13).

Third, the long-term persistence (19–29 days after illness onset) of viral RNA in the serum and sputum specimens of the SARS-CoV-specific IgG seroconverters is an important finding. Prolonged shedding of viral RNA in respiratory secretions (11 days after illness onset), plasma (up to 9 days), and stool specimens (25 days) was documented previously (6). Further studies are needed to determine whether the viable viral particles existed in body fluids in the presence of high antibody to the virus. Finally, one SARS patient, who did not have an underlying coexisting condition and did not receive any immunosuppressive agents during hospitalization, did not have detectable antibody to SARS-CoV 24 days ($>$ 21 days) after illness onset. The serum and sputum RT-PCR for SARS-CoV were positive in this patient, and the sequence was confirmed. Whether the patient was anergic to SARS-CoV infection is unknown. A later serum sample taken in the convalescent stage should be tested to determine whether this patient subsequently seroconverts (7).

The upsurge of IgG antibody to SARS-CoV and its correlation with the progression of ARDS, necessitating ventilator support in four of the seven patients, was evident. Previous study suggested that an overexuberant host response rather than uncontrolled viral replication, contributed to severe clinical symptoms and progressive lung damage (12). Whether the addition of SARS-CoV-specific antibody in SARS patients further aggravated the preexisting overactive immune-mediated deterioration was unclear.

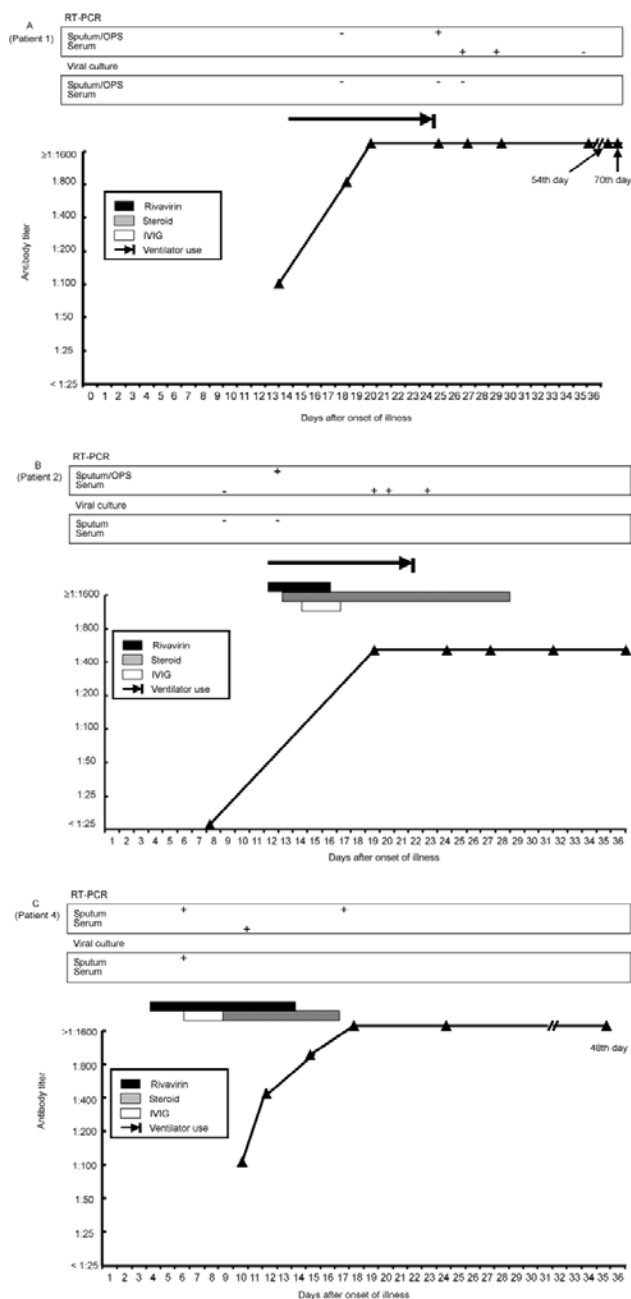


Figure 2. Timelines of positive reverse transcription polymerase chain reaction, antibody responses and treatment regimens (ribavirin, corticosteroid, and intravenous immunoglobulin) after onset of disease in seven patients with severe acute respiratory syndrome. Panels A–C indicate patients 1, 2, and 4.

High concentrations of viral RNA, up to 100 million molecules per milliliter, were detected in a sputum sample from an index patient on day 9 (6). In the present series, a physician contracted the infection from a patient (patient 2) 12 days after the onset of symptoms, indicating that shedding of the virus from the respiratory tract of symptomatic SARS patients may last for ≥ 12 days. Viral RNA in

the sputum samples of patient 2 collected 12 days after the onset of symptoms supports this clinical finding.

Dual infection caused by *M. pneumoniae* and SARS-CoV was found in patient 5. No evidence of *M. pneumoniae* infection existed in patient 6 from the same cluster. This finding is similar to a previous report (6). Four of our patients had elevated IgG antibody titers for *C. pneumoniae*, and five had elevated antibody titers against parainfluenzavirus 1, 2, or 3 in acute-phase serum samples without a fourfold rise of titers in convalescent-phase serum samples. Whether the antibody responses of these patients reflected past infections from *C. pneumoniae*, parainfluenzavirus, or both, or merely a cross-reaction with antibody against SARS-CoV virus remains unclear.

As of May 16 2003, data of complete genomic sequences for 13 SARS-CoV strains isolated from Hong Kong, Singapore, China, Canada, Vietnam, and Taiwan were available in GenBank. The number of nucleotides ranged from 29,705 (SIN2677 strain) to 29,751 (TOR2) (14,15). Since February 2003, at least three different clusters of SARS outbreaks occurred in different parts of Taiwan, and five strains were identified from patients in these clusters. The availability of the sequence data of different strains in a given geographic area will have an immediate impact on the effort to trace the origins and transmission of SARS-CoV and develop novel rapid diagnostic tests and a vaccine.

In summary, analysis of these seven patients with virologically or serologically documented infections caused by SARS-CoV in Taiwan not only extended the knowledge of this emerging novel disease but also provided microbiologic and immunologic clues for the physicians caring for patients suspected of having this disorder. Viral RNA may persist for some time in patients who seroconvert, and some patients may lack an antibody response to SARS-CoV >21 days after illness onset. An upsurge of antibody response is associated with the aggravation of respiratory failure that required ventilator support.

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HEALTH ALERT NOTICE
Health Alert Notice for International Travelers
Arriving in the United States
from China, Vietnam, and Singapore

TO THE TRAVELER: During your recent travel, you may have been exposed to cases of severe acute respiratory disease syndrome (SARS). You should monitor your health for at least 10 days. If you become ill with fever, cough, or difficulty in breathing, you should consult a physician. In advance of your visit to the physician, tell him or her about your recent travel to these regions and whether you were in contact with someone who had these symptoms. Please save this card and give it to your physician if you become ill.

TO THE PHYSICIAN: The patient presenting this card may have recently traveled to China, Vietnam, or Singapore, where cases of SARS have been identified. If you suspect that this patient may have SARS, please contact your city, county, or state health officer (see <http://www.cdc.gov> or call the CDC Emergency Operations Center at 770-488-7100).

English

緊急保健通告
从中国、越南和新加坡抵达美国的国际旅行者紧急保健通告

旅行保健通告。 您在最近旅行期间可能接触了严重急性呼吸综合征病例。您应当观察自己的健康状况，为期至少十天。如果您出现发热、咳嗽、或呼吸困难，您应当咨询医生。在您就诊之前，告知医生您最近到过上述地区旅行，并告知医生您是否接触过有上述症状者。请保存本卡片，如果您患病，请带本卡片交给医生。

医生保健通告。 交给本卡片的患者最近可能曾前往中国、越南和新加坡旅行。这些地区已发现SARS（即严重急性呼吸综合征）病例。如果您认为此患者可能患有SARS，请咨询。去：州保健官员联系（请参见<http://www.cdc.gov>，或致电CDC Emergency Operations Center，770-488-7100）。

Simplified Chinese

KHUYẾN CÁO Y TẾ
THÔNG BÁO QUÉ HÀNH KHÁCH QUỐC TẾ ĐI BẾN HOA KÝ
TỪ TRUNG-QUỐC, VIỆT-NAM VÀ SINGAPORE.

THÔNG BÁO QUÉ HÀNH KHÁCH: Trong chuyến du hành vào mỗi đây, quý vị có thể đã tiếp xúc nhân chứng bệnh hô hấp cấp tính nghiêm trọng (SARS). Quý vị nên theo dõi kỹ lưỡng các triệu chứng bệnh ít nhất là 10 ngày. Nếu quý vị bị mắc bệnh liên quan đến bệnh hô hấp cấp tính, thì nên tham khảo với bác sĩ ngay. Trước khi đến bác sĩ, quý vị nên cho bác sĩ biết về những nơi quý vị đã đi hành và mỗi đây tiếp xúc gần gũi với ai. Quý vị cũng nên cho bác sĩ biết nếu quý vị đã có tiếp xúc gần gũi với những người có những chứng bệnh này. Xin quý vị hãy giữ gìn phiếu khuyến cáo này để trình cho bác sĩ nếu quý vị bị mắc bệnh.

THÔNG BÁO QUÉ BÁC SĨ: Bệnh nhân trên phiếu khuyến cáo này có thể vừa đi hành đến Trung-Quốc-Việt-Nam, hoặc Singapore nơi đã phát hiện bệnh SARS. Nếu quý vị nghi ngờ bệnh nhân đã mắc bệnh SARS, xin liên lạc với cán bộ y tế của thành phố, quận, hoặc tiểu bang. (xem <http://www.cdc.gov> hoặc gọi cho Trung Tâm Kiểm Soát Bệnh Dịch, Phòng Cấp Báo Cứu CDC (CDC Emergency Operations Center), 606 số thuê: 770-488-7100).

Vietnamese

HEALTH ALERT NOTICE
건강 경보 공지사항
KHUYẾN CÁO Y TẾ
健康に関する注意喚起

Japanese

AVIS D'ALERTE MÉDICALE
AVIS DE ALERTA DE SALUD

緊急保健通告
緊急保健通告

French

AVIS D'ALERTE MÉDICALE
aux voyageurs internationaux arrivant aux États-Unis
en provenance de la Chine, du Vietnam et de Singapour

AU VOYAGEUR : Au cours de votre séjour réalisé récemment dans les régions susmentionnées, vous avez peut-être été en contact avec des personnes atteintes du syndrome respiratoire sévère (SARS). Par conséquent, vous devez surveiller votre état de santé pendant au moins dix (10) jours. Consultez un médecin si vous présentez l'un des symptômes du SRAS (fièvre, toux, difficulté à respirer). Appelez le bureau du médecin avant de vous rendre. Informez-le de votre voyage dans ces régions et s'il vous avez été en contact avec des personnes qui manifestaient ces symptômes. Conservez cette carte et présentez-la à votre médecin si vous devenez malade.

AU MÉDECIN : La personne qui vous présente cette carte a peut-être visité récemment la Chine, le Vietnam ou Singapour, soit des régions touchées par le SRAS. Si vous croyez que cette personne peut être atteinte du SRAS, contactez les autorités de la santé publique de votre ville ou de votre région. (Veuillez le site <http://www.cdc.gov> ou appelez le Centre des opérations d'urgence du Centers for Disease Control and Prevention au (770) 488-7100).

French

건강 경보 공지사항
중국, 베트남, 싱가포르로부터
미국으로 도착하는
국제 여행객들 건강 경보 공지사항

여행객에게: 최근 여행기간 동안 공중 보건 위기(SARS)에 노출되었을 수 있습니다. 적어도 10일 동안 건강을 주의하여야 합니다. 열, 기침 또는 호흡 곤란 등의 발이 발생하는 경우 의사의 진찰을 받아 보아야 합니다. 병원 방문하기 전에, 상급 의료기관으로 여행 사실을 알리고 증상, 발고 있는 환자요의 접촉 이력을 의사에게 알린 후 방문하십시오. 이 카드도 보관하십시오. 이 카드가 필요할 때 의사에게 보여주세요.

의사들에게: 이 카드를 제공하는 환자는 공중 보건 위기(SARS)에 노출된 발이 의심된 중국, 베트남 또는 싱가포르 최근 여행객일 수 있습니다. 관련 증상이 출현된다면 의심되는 경우, 시, 또는 주 보건 담당자에게 연락하십시오. (<http://www.cdc.gov>를 참조하거나, CDC 비상 센터 770-488-7100으로 전화 주십시오.)

Korean

As part of Centers for Disease Control and Prevntion's response to the severe acute respiratory syndrome (SARS) outbreak, from March 15 through July 17, 2003, quarantine officials at U.S. ports of entry distributed 2,721,965 health alert notices to passengers arriving from areas with SARS. The notices, translated into eight languages, advised travelers of SARS symptoms and provided physicians with reporting information.