

communicate quickly and easily with provincial and municipal health authorities was needed to ensure that the most up-to-date information concerning the outbreak was available. The intergovernmental relationships necessary for such rapid communication should be established in advance. Third, accurate and timely communication with frontline staff members is the best way to minimize their fears. Finally, personal protective equipment procedures should be maintained until assurance that the exposure risk is negligible. The SARS outbreak is unlikely an isolated occurrence; therefore, sound advance planning on the basis of experience will increase the ability to protect both EMS staff and the public in the future.

#### Acknowledgments

We thank the Toronto Emergency Medical Services staff who helped manage the outbreak and Paula Chabanais for editing this manuscript.

**Alexis Silverman,\*<sup>1</sup> Andrew Simor,†<sup>1</sup> and Mona R. Loutfy,‡<sup>1</sup>§<sup>1</sup>**

\*Toronto Emergency Medical Services, Toronto, Ontario, Canada; †Sunnybrook and Women's College Health Sciences Centre, Toronto, Ontario, Canada; ‡McGill University, Montreal, Quebec, Canada; and §North York General Hospital, Toronto, Ontario, Canada

#### Reference

1. Varia M, Wilson S, Sarwal S, et al. Hospital Outbreak Investigation Team. Investigation of a nosocomial outbreak of severe acute respiratory syndrome (SARS) in Toronto, Canada. *CMAJ*. 2003;169:285–92.

Address for correspondence: Alexis Silverman, Toronto Emergency Medical Services, 4330 Dufferin Street, Toronto, Ontario, Canada M3H 5R9; fax: 416-392-2149; email: asilver@toronto.ca

<sup>1</sup>All authors contributed equally to developing, drafting, and revising this letter.

## SARS during Pregnancy, United States

**To the Editor:** Two of eight persons with laboratory-confirmed severe acute respiratory syndrome– associated coronavirus (SARS-CoV) infection in the United States during 2003 were pregnant women. Robertson et al. (1) reported data describing one pregnant patient who recovered and delivered a healthy infant. We report data concerning the second patient, with follow-up 1 month after the child's birth.

The patient, a healthy, 38-year-old woman in the 7th week of pregnancy, traveled with her husband to Hong Kong. From March 1 to March 6, 2003, they stayed at the Hong Kong hotel where it is believed a physician from China spread SARS-CoV to several guests. These guests were the index case-patients for subsequent outbreaks in Hong Kong, Vietnam, Singapore, and Toronto, Canada (2). The woman and her husband returned to the United States on March 6; the husband had onset of SARS illness on March 13. On March 19, the patient had onset of an illness with fever (temperature 37.8–40°C), muscle aches, chills, headache, runny nose, productive cough, wheezing, and shortness of breath. A chest radiograph showed a diffuse infiltrate in the left lung. The patient was hospitalized for 9 days and given broad-spectrum antimicrobial drugs. She recovered from her illness, and enzyme immunoassay and immunofluorescent assays conducted on serum samples on days 28 and 64 after illness onset were positive for antibodies to SARS-CoV.

The patient had an uneventful pregnancy until the last trimester, when her blood glucose levels were elevated. Early spontaneous rupture of membranes initiated preterm labor, and a cesarean section was performed at 36 weeks' gestation because of fetal distress. A 5-pound, 7-ounce, healthy

boy was delivered without complications. Apgar scores were 7 at 1 minute and 8 at 5 minutes. The newborn had no illness, abnormalities, or congenital malformations. Serum samples from the patient at delivery were positive for antibodies to SARS-CoV, but cord blood and placenta samples were negative. Breast milk samples on postpartum days 12 and 30 were also negative for SARS-CoV antibodies. Blood, stool, and nasopharyngeal swab samples from the patient and cord-blood samples showed no viral RNA by reverse transcription–polymerase chain reaction. Stool samples from the newborn, collected on days 12 and 30 after delivery, were also negative for viral RNA.

Although other countries have reported cases of severe illness and poor outcome associated with SARS-CoV infection during pregnancy (3–5), neither of the two pregnant SARS case-patients in the United States had serious adverse outcomes. The presence of antibodies to SARS-CoV in breast milk might be influenced by the time of infection in relation to gestation. Robertson et al. (1) reported that antibodies to SARS-CoV were detected in the breast milk of a patient who was infected at 19 weeks' gestation; however, the patient in this case was infected at 7 weeks' gestation, and antibodies to the virus were not detected in her breast milk. No reports have indicated vertical transmission of SARS-CoV, a finding that is supported by our data. However, too few cases have been studied to clearly define the risks and provide guidance for treating pregnant women infected with SARS CoV.

**Lauren J. Stockman,\* Sara A. Lowther,\* Karen Coy,† Jenny Saw,‡ and Umesh D. Parashar\***

\*Centers for Disease Control and Prevention, Atlanta, Georgia, USA; †Santa Clara County Department of Public Health, San Jose, California, USA; and ‡Private pediatric practice, San Jose, California, USA

## References

1. Robertson CA, Lowther SA, Birch T, Tan C, Sorhage F, Stockman L, et al. SARS and pregnancy: a case report. *Emerg Infect Dis.* 2004;10:345–8.
2. Centers for Disease Control and Prevention. Update: outbreak of severe acute respiratory syndrome—worldwide, 2003. *MMWR Morb Mortal Wkly Rep.* 2003;52:241–8.
3. Shek CC, Ng PC, Fung GP, Cheng FW, Chan PK, Peiris MJ, et al. Infants born to mothers with severe acute respiratory syndrome. *Pediatrics.* 2003;112:254–6.
4. Zhang JP, Wang YH, Chen LN, Zhang R, Xie YF. Clinical analysis of pregnancy in second and third trimesters complicated severe acute respiratory syndrome. *Zhonghua Fu Chan Ke Za Zhi.* 2003;38:516–20.
5. Wong SF, Chow KM, deSwiet M. Severe acute respiratory syndrome and pregnancy. *BJOG.* 2003;110:641–2

Address for correspondence: Lauren Stockman, Centers for Disease Control and Prevention, 1600 Clifton Road, MS E78, Atlanta, GA 30333, USA; fax: 404-498-2720; email: bgu8@cdc.gov

## Eosinophilic Pleural Effusion in Gnathostomiasis

**To the Editor:** Moore et al. reported a case series of patients infected with imported gnathostomiasis who had typical intermittent, migratory skin manifestations or peripheral blood eosinophilia or both, as well as undiagnosed eosinophilia with non-specific symptoms (1). We would like to add some comments based on a recent patient treated in Marseille, France.

In April 2003, a 66-year-old man living in Marseille indicated a history of fever for 8 days. He had returned from Vietnam 1 month earlier, where he had stayed for 4 weeks in the Ho Chi Minh City area and 2 days in the Mekong Delta area. He was well dur-

ing his trip, and reported no arthropod bites except from mosquitoes. He had no direct skin contact with river water. Dietary intake included local dishes with rice, fish, pork, shrimp, and chicken. His symptoms started 3 weeks after he returned to Marseille and included fever (temperature 38°C), asthenia, chills (1 day), moderate dyspnea during exercise, transient bilateral pain of the testes, and an episode of hemospermia. He was referred by his family doctor to the Infectious and Tropical Diseases Unit, North Hospital.

On admission, the patient's temperature was 38°C. Physical examination of the patient, including the testes, was normal except for a systolic heart murmur (preexisting and known to the patient), and clinical signs of left pleural effusion. The effusion was subsequently confirmed by chest x-ray, which also showed a discrete diffuse bilateral lung infiltrate. Results of routine laboratory tests conducted on blood samples were normal except for an elevated eosinophil count of 5.2 10<sup>9</sup>/L. Blood smears for plasmodia and microfilaremia were negative. Urologic examination, including echography and prostate-specific antigen, showed no abnormalities except a prostatic adenoma (preexisting and known to the patient). No eggs or parasites were detected by microscopic examination in stools or in urine, although both sedimented and centrifuged urine specimens were studied and filtration techniques were used. After transthoracic aspiration of 100 mL of pleural effusion, cytologic examination showed an eosinophil count of 5,800/L without parasites. Bacterial culture, including mycobacteria, was negative. On day 4, the patient was afebrile and was discharged. On admission, results of a first set of examinations involving reactivities to schistosomiasis, paragonimiasis, strongyloidiasis, cysticercosis, trichinosis, gnathostomiasis, filariasis, and toxocarasis were negative.

One month later, the eosinophil count of our patient had decreased to 1.8 x 10<sup>9</sup>/L. He was afebrile, and his only complaint was asthenia. A new set of serologic examinations was conducted. The Western blot assay for gnathostomiasis conducted at the Swiss Tropical Institute (Socinstrasse 57, CH-4002, Basel, Switzerland) was positive, showing immunoglobulin G reactivity to four specific bands including the 24-kDa band, considered pathognomonic for the diagnosis of *Gnathostoma* infection (1). The seroconversion confirmed the diagnosis of gnathostomiasis. All other serologic tests remained negative, except an increase of antibodies against *Acanthocheilonema vitae* used as antigen for unspecific serologic screening for filariasis (Laboratoire Marcel Merieux, Lyon, France). After a 21-day course of albendazole and a single dose of ivermectin, the eosinophil count of our patient decreased to 0.8 x 10<sup>9</sup>/L.

Three aspects of gnathostomiasis as an emerging imported disease can complement the findings of Moore et al. (1). First, the clinical findings in our case are very unusual. Hemospermia is often benign with predominant causes including prostatic and seminal vesicle disease. Infections, including mainly schistosomiasis and tuberculosis, have been associated with these symptoms (2). Although our patient had a prostatic adenoma, this is the first time that hemospermia has been associated with gnathostomiasis. Because of the anxiety hemospermia caused, this symptom was the main reason that our patient consulted our center. Secondly, eosinophilic pleural effusion is also unusual in gnathostomiasis. Although reported as a potential cause in reference books (3), a Medline search (key words: gnathostomiasis and eosinophilia and pleural effusion or pleuritis or lung) disclosed only two references to pleural effusion as the main symptom of gnathostomiasis (4,5).