A Multistate Outbreak of Salmonella enterica Serotype Saintpaul Infections Linked to Mango Consumption: A Recurrent Theme

Sir—We read with interest the report of a multistate outbreak of Salmonella enterica serotype Newport infection in 1999 by Sivapalasingam et al. [1]. The outbreak was associated with the consumption of mangoes imported from a single farm in Brazil. An environmental investigation at the farm revealed that inadequately chlorinated water was used in the hot water immersion treatment to exterminate fruit fly larvae. At the time, the importance of adequately chlorinating the treatment water had not been recognized [1, 2]. We write to report a second outbreak of Salmonella enterica infection associated with consumption of mangoes; however, the serotype in this outbreak was Saintpaul. Also imported, these mangoes were possibly contaminated through a mechanism similar to that described by Sivapalasingam et al. [1].

In March of 2001, the Massachusetts and Connecticut departments of public health reported 19 patients with culture-confirmed S. Saintpaul infections. The PFGE patterns of the DNA from these isolates were indistinguishable, suggesting an outbreak. By 31 March 2001, S. Saintpaul isolates from 7 additional patients, including residents of California, Delaware, New Jersey, New York, and Rhode Island, were identified. The mean age of patients was 35 years (range, 1–89 years); 48% were female.

To identify risk factors for infection, we conducted a case-control study with 13 cases and 25 controls frequency-matched for age, sex, and city of residence. Telephone interviews were conducted by state health department and Centers for Disease Control and Prevention officials using a standardized questionnaire. Raw mango consumption was the only exposure significantly associated with illness (OR, 28.8; 95% CI, 2.1–888; P = .003).

Three patients had adequate purchase records for the US Food and Drug Association (FDA) to initiate a “trace-back” investigation; however, only 1 patient’s purchase could be traced beyond the retail seller. This patient’s receipt indicated that the mangoes were imported from Peru, but there was inadequate information obtained to complete the trace-back to the farm level.

During an unrelated site-visit to mango-producing regions in Peru, USA Animal and Plant Health Inspection Service (APHIS) inspectors noted that producers were using untreated water in the final step of the fruit fly control program (P. C. Witherell, US Department of Agriculture, APHS-Plant Protection and Quarantine, personal communication). Subsequent to the outbreak of mango-associated S. Newport infection in 1999 reported by Sivapalasingam et al. [1], APHIS recommended a concentration of 50–200 ppm chlorine in the water used for the hot water immersion treatment [1]. This change was not published until 2002 [3]. It is likely that the mango producers in Peru had not yet learned of the need to chlorinate the water used for hot water immersion treatment, and a second outbreak occurred before the recommendation became widely adopted.

In conclusion, mangoes were implicated as the vehicle during a multistate outbreak of S. Saintpaul in February and March of 2001. These mangoes were likely exposed to inadequately chlorinated water, which may have led to contamination with S. Saintpaul. APHIS recommendations currently include using adequately chlorinated water in mango processing for the prevention of fruit fly infestation [3]. As of 1 January 2003, no subsequent foodborne outbreaks have been associated with mangoes. The outbreak we report demonstrates the need for thorough microbiologic evaluation of new methods of food processing prior to their implementation, as Sivapalasingam et al. [1] suggest.

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References

Persistence of Physical Symptoms in and Abnormal Laboratory Findings for Survivors of Severe Acute Respiratory Syndrome

Str—We performed a cross-sectional study to assess the physical symptoms in and abnormal laboratory findings for survivors of severe acute respiratory syndrome (SARS) at their first follow-up visit after discharge from Princess Margaret Hospital (Hong Kong, China). Sixty-two patients who experienced the onset of SARS symptoms during the period from 18 March 2003 through 30 March 2003 were recruited. All patients had pneumonia and positive SARS-associated coronavirus (SARS-CoV) seroconversion. The mean age (±SD) was 37.07 ± 12.96 years, the ratio of male subjects to female subjects was 0.82, and the intubation rate was 9.6%. In this cohort, 90.3% of patients were recruited after discharge from Princess Margaret Hospital, Hong Kong, China.

Symptoms reported at the first follow-up visit included palpitation (45.1% of patients), exertional dyspnea (41.9%), malaise (40.3%), easy forgetfulness (30.6%), chest discomfort (22.5%), hand tremor (21%), dizziness (17.7%), depression (16.1%), myalgia (12.9%), headache (9.6%), diarrhoea (8.1%), cough (8.1%), insomnia (6.5%), and hair loss over the scalp (3.2%). No patient reported sputum production. Patients described palpitation as a paroxysmal, fast heart beat or extra heart beat sensation. A sinus tachycardia with resting heart rate of 100–110 beats/min was identified in 18% of patients complaining of palpitation.

Laboratory findings included the following mean values (±SD): hemoglobin level, 12.93 ± 1.42 g/dL; WBC count, 6.71 × 10^9 ± 2.00 × 10^9 cells/L; neutrophil count, 4.58 × 10^9 ± 1.75 × 10^9 cells/L; lymphocyte count, 1.51 × 10^9 ± 0.46 × 10^9 lymphocytes/L; platelet count, 308 × 10^9 ± 89.26 × 10^9 cells/L; erythrocyte sedimentation rate, 11.86 ± 14.47 mm/h; albumin level, 42.56 ± 3.58 g/L; globulin level, 30.58 ± 3.05 g/L; bilirubin level, 8.67 ± 5.18 umol/L; alkaline phosphatase level, 83.2 ± 22.44 U/L; alanine aminotransferase level, 28.9 ± 13.96 U/L; creatinine kinase level, 104 ± 268.9 U/L; lactate dehydrogenase level, 242 ± 64.29 U/L. At the first follow-up visit, 46.7% of patients were found to have a lactate dehydrogenase level of ≥230 U/L. Abnormal chest radiograph findings were reported by the Department of Radiology for 35.4% of patients. These findings included patchy densities, linear atelectasis, ground glass appearance, reticular marking, and streaky opacities. There was no significant difference in the rate of exertional dyspnea between patients with and patients without abnormal chest radiograph findings (P = .54). For all patients, PCR of urine, nasal, and throat swab samples was negative for SARS-CoV RNA. However, for 1 female patient, PCR of a stool sample obtained 35 days after the onset of SARS symptoms was positive for SARS-CoV RNA. No person who had close contact with that patient after she was discharged from the hospital contracted SARS.

From what we have learned, some SARS survivors still had physical symptoms up to 6 weeks after the onset of SARS symptoms, although their complete blood counts, the results of their liver and renal function tests, and their erythrocyte sedimentation rate were largely normalized. The finding of abnormal lactate dehydrogenase levels may imply that patients still had not fully recovered from SARS-related tissue damage at the first follow-up visit. We should not overlook the effect of therapy with ribavirin and corticosteroids, which might have contributed to the symptoms and to the abnormal laboratory values. Physicians providing care to patients with SARS during the convalescent period should be aware of the possibility of such abnormal findings.

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References


Clinical Infectious Diseases 2004; 38:1338-8
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