Table 1. Contact investigation results.

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>n</th>
<th>No. tested</th>
<th>No. (%) positive by TST</th>
</tr>
</thead>
<tbody>
<tr>
<td>St. Croix contacts</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Household</td>
<td>4</td>
<td>4</td>
<td>1 (25)</td>
</tr>
<tr>
<td>Relatives/social</td>
<td>12</td>
<td>12</td>
<td>4 (33)</td>
</tr>
<tr>
<td>Hospital B medical staff</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Signed chart</td>
<td>20</td>
<td>18</td>
<td>3 (17)</td>
</tr>
<tr>
<td>Others</td>
<td>17</td>
<td>11</td>
<td>1 (9)</td>
</tr>
<tr>
<td>Patient at Hospital B</td>
<td>1</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Clinic A medical staff</td>
<td>3</td>
<td>3</td>
<td>0</td>
</tr>
<tr>
<td>Florida contacts</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Household</td>
<td>3</td>
<td>3</td>
<td>3 (100)</td>
</tr>
<tr>
<td>Social</td>
<td>5</td>
<td>5</td>
<td>1 (20)</td>
</tr>
<tr>
<td>Totals</td>
<td>65</td>
<td>57</td>
<td>13 (23)*</td>
</tr>
</tbody>
</table>

NOTE: TB, tuberculosis; TST, tuberculin skin test

* Includes 4 contacts with documented TST conversions (1 among medical staff at the US Virgin Islands [USVI] Hospital A and 3 among household contacts in Florida) and 2 contacts with initial positive TSTs who were young, healthy, and had no known prior TB exposure (1 household and 1 social contact in the USVI).

The index case patient had primary, rather than acquired, drug resistance. The fact that the DNA fingerprint of his isolate exactly matched that of a person with active MDR TB admitted to the same Puerto Rico hospital during the case patient’s 1994 hospitalization strongly suggests that nosocomial transmission occurred at that facility in 1994.

Factors associated with outbreaks of MDR TB in other settings include delayed diagnosis of TB, inadequate infection-control measures, delayed recognition of drug resistance, and interruption of therapy [1–3]. All of these factors were present in the USVI.

The case patient described in this report was successfully treated for 18 months with daily directly observed therapy (DOT). During the week, he came to Clinic A to receive oral and injectable medication, and on weekends the USVI Health Department performed DOT at his home. Telephone calls were made to the patient, his family, and the health department on the few occasions when he did not arrive promptly at Clinic A. Dedicated health department and clinic staff and a supportive family contributed to this successful endeavor.

Thus far there have been no further cases of MDR TB in the USVI. Maintaining an effective surveillance system, which includes surveillance for drug resistance, is critically important.

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References


Acute Hepatitis E Infection Acquired in California

Outbreaks of hepatitis E virus (HEV) infection have occurred in India, Bangladesh, Nepal, Pakistan, Burma, China, Mexico, the central Asian republics of the former Soviet Union, and parts of the Middle East and Africa [1]. Until recently, acute HEV infection in the United States has been documented sporadically among travelers returning from HEV-endemic areas [1–3]. There were very few reports of acute HEV infection acquired in the United States [4, 5]. Here we describe a case of acute HEV infection in a California resident with no history of travel.

On 28 September 1998, we examined a 43-year-old man with a 3-week history of nausea, vomiting, diarrhea, and abdominal discomfort. That examination revealed a nontender liver of normal consistency, palpable 1 inch below the right costal margin with full inspiration. Evaluation of a serum sample obtained on 28 September indicated an alanine aminotransferase (ALT) level of 699 U/L (normal range, 0–45 U/L); an aspartate aminotransferase (AST) level of 416 U/L (normal range, 0–40 U/L); an alkaline phosphatase level of 144 U/L (normal range, 0–40 U/L); and a total bilirubin level of 0.7 mg/dL (normal...
range, 0.2–1.5 mg/dL). His liver enzymes returned to normal levels when tested on 8 October 1998.

Serological tests for hepatitis A (IgM and IgG anti-hepatitis A virus [HAV]), hepatitis B (hepatitis B surface antigen [HBsAg], anti-HBsAg, IgG and IgM anti-hepatitis B core antigen [HBcAg]), and hepatitis C (anti-hepatitis C virus [HCV]) were all negative. On 5 October, his serum tested positive for IgM anti-HEV by use of a commercial laboratory using an EIA. In January 1999, a follow-up serum sample tested positive for IgG anti-HEV and negative for IgM anti-HEV by EIA at the Centers for Disease Control and Prevention (CDC; Atlanta, Georgia).

The patient had no history of travel outside California in 1998. Approximately 1 month before the onset of illness, he had gone camping with his 3 children (aged 10, 12, and 16 years) in the Sierra Nevada mountains for 1 week. At the campsite, they drank unboiled water from a well that was located ~60 ft from a toilet. The patient (but not his children) also drank water from a lake where he was fishing. His children did not develop any illness after the trip. In January 1999, the children’s serum samples tested negative for IgG anti-HEV by use of EIA at the CDC.

The patient denied previous blood transfusion, injection drug use, or excessive alcohol consumption. Within the 12 weeks before illness onset, he reported no consumption of raw shell-fish, raw fish, raw meat, or raw eggs or egg products. He had no history of exposure to farm animals, contact with persons having jaundice, or contact with visitors from other countries. He ate in restaurants occasionally but could not recall the names of those that he had visited.

To our knowledge, this is the first definite acute HEV infection acquired in California. The clinical presentation followed by serological evidence of IgM and IgG anti-HEV established the case. The source of infection cannot be established but was clearly within California, given that the patient did not travel out of the state during the incubation period (i.e., 2–9 weeks, average ~45 days) [6]. Although the patient had been exposed to well and lake water during the incubation period, other modes of transmission (e.g., contaminated food) were also possible.

A previous report described an HEV case in a Minnesota resident who had traveled to San Jose, California, during part of the incubation period, but could not determine where the infection was acquired [4]. Given these case reports, HEV infection should be considered in patients with hepatitis of unknown etiology, even if they had no history of international travel. Physicians and laboratories should report acute HEV infections to local public health departments so that potential local sources can be determined and interventions conducted if necessary.

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References

Severe Hypertension and Renal Atrophy Associated with Indinavir

The addition of protease inhibitors to antiretroviral therapy has been a major advance in the treatment of HIV infection, delaying disease progression and death, and returning people to productive lives. Indinavir, one of the most widely prescribed HIV protease inhibitors, is relatively safe and well-tolerated, even though its use is associated with nephrolithiasis in ~5% of patients [1, 2]. This adverse effect, generally presenting early in therapy, is not usually serious and resolves rapidly with drug discontinuation. We describe a case of severe hypertension and renal atrophy that developed during long-term therapy with indinavir.

The patient was a 39-year-old woman known to be HIV positive for 7 years who was infected through heterosexual contact. In March 1998, her clinical condition was compromised, and her laboratory data were as follows: CD4+ cell count, 18/μL (1.8%), and plasma HIV-1 RNA level, 123,000 copies/mL. A combination antiretroviral therapy with zidovudine, lamivudine, and indinavir (800 mg orally 3 times daily) was instituted. Before starting indinavir therapy, renal ultrasonography...