earlier in gestation [9], when such responses are absent or low.

In acknowledgment of safety concerns highlighted in recent studies, the CDC no longer recommends that pregnant women with BV be treated with intravaginal clindamycin therapy, because 5 of 6 trials that have evaluated this therapy for women with (or without) BV showed significant increases in the rate of prematurity and neonatal sepsis [10].

Klebanoff et al. [1] emphasize the importance of an accurate diagnosis of BV before any antibiotic therapy is administered, and they warn that harm may result from treating women with metronidazole who do not have BV [2–4, 13]. We agree, and we included, in both the CDC guidelines and the background article, methods for the proper diagnosis of BV [9, 10].

The CDC’s STD treatment guidelines [9] are developed by a panel of experts who review published studies, rate the quality of the evidence, and determine the strength of recommendations [14]. The guidelines contain detailed information that is relevant for clinicians who must make treatment decisions. They state, “Because treatment of BV in asymptomatic pregnant women at high risk for preterm delivery (i.e., those who have previously delivered a premature infant) with a recommended regimen has reduced preterm delivery in three of four randomized controlled trials, some specialists recommend multiple dosing: oral and rectal. In: Phillips R, Collier J, eds. Metronidazole: proceedings of the 2nd International Symposium on Anaerobic Infections (Geneva, Switzerland). New York: Academic Press, 1979:55–8.


Very Rapid Evolution of Infection with Hepatitis C Virus Transmitted by an Accidental Needlestick

Sir—Hepatitis C virus (HCV) is one of the most common etiologic agents of acute and chronic liver disease [1]. HCV infection results in acute hepatitis usually associated with mild symptoms. The mean incubation period for acute HCV infection is 7 weeks, and symptoms, if present, last for 2–12 weeks [2]. The main route of HCV transmission is parenteral. Transmission has also been documented after receipt of needlestick injuries (estimated risk, 1.8%), but the prevalence of HCV infection among health care workers is no greater than the prevalence among the general population [3]. Nevertheless, transmission of HCV after receipt of a needlestick is an important threat to health care workers. We describe a 53-year-old female nurse who received a
Table 1. Clinical and virological features of a nurse who was exposed to hepatitis C virus (HCV) after she received a needlestick injury.

<table>
<thead>
<tr>
<th>Laboratory value or test</th>
<th>0</th>
<th>13</th>
<th>16</th>
<th>24</th>
<th>26</th>
<th>27</th>
<th>28</th>
<th>29</th>
<th>30</th>
<th>31</th>
</tr>
</thead>
<tbody>
<tr>
<td>AST, IU/L</td>
<td>14</td>
<td>216</td>
<td>327</td>
<td>4149</td>
<td>7791</td>
<td>5070</td>
<td>2197</td>
<td>673</td>
<td>412</td>
<td>204</td>
</tr>
<tr>
<td>ALT, IU/L</td>
<td>14</td>
<td>314</td>
<td>791</td>
<td>5927</td>
<td>8434</td>
<td>6140</td>
<td>4521</td>
<td>2989</td>
<td>2229</td>
<td>1607</td>
</tr>
<tr>
<td>Bilirubin, mg/dL</td>
<td>1.08</td>
<td>0.76</td>
<td>0.77</td>
<td>13.78</td>
<td>17.90</td>
<td>18.67</td>
<td>22.83</td>
<td>20.27</td>
<td>18.07</td>
<td>13.08</td>
</tr>
<tr>
<td>HCV load, a × 10⁶ copies/mL</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>0.7</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>Antibodies against HCV</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>+</td>
<td>+</td>
<td>—</td>
<td>—</td>
</tr>
</tbody>
</table>

**NOTE.** ALT, alanine aminotransferase; AST, aspartate aminotransferase.

*a* Determined by RT-PCR.

needlestick injury and developed severe acute hepatitis C, which had an incubation period of only 9 days.

The needlestick accident occurred when the nurse was obtaining a blood sample from a patient who had undergone liver transplantation to treat HCV-associated cirrhosis. The accident occurred when the nurse's fingertip hit the tip of the needle, which punctured her skin and caused a superficial wound of approximately 2–3 mm that bled spontaneously. The donor had a histologically proven recurrence of HCV infection noted by a posttransplantation liver biopsy, as well as high viral replication titers in serum (2.2 × 10⁶ copies/mL) at the moment of the accidental needlestick. Soon after the needlestick injury occurred, a blood sample was obtained from the nurse. Laboratory analysis confirmed that the nurse was positive for hepatitis B virus antibodies, that she did not have antibodies to HCV or HIV, and that her alanine aminotransferase (ALT) level was normal (14 IU/L). Eight days after she received the needlestick, she developed fever, arthralgias, asthenia, and general discomfort; 24 h later, these symptoms were followed by ictericia, vomiting, and pain in the right hypochondrium. Follow-up testing revealed evidence of an elevated ALT level (194 IU/L), and a diagnosis of clinical acute HCV infection was suggested. Two weeks after the exposure, the nurse’s ALT level was continuing to increase, up to 791 IU/L (table 1). The results of virological assays for the detection of viruses other than HCV (e.g., hepatitis B and D viruses, cytomegalovirus, and HIV) were negative. Instead of relying only on the conventional anti-HCV antibody test to detect HCV infection after

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**Figure 1.** Alignment of the sequences of the HCV hypervariable region 1 in virus recovered from the donor and from the nurse.
these incidents, we used PCR for earlier detection of the virus, so that we could treat the nurse as soon as possible. The first PCR test for detection of HCV RNA was performed 2 weeks after the needlestick accident, and the result was positive (virus load, $2 \times 10^7$ copies/mL).

Although we knew that the nurse had not had a previous HCV infection, we confirmed that the HCV genotype was 1b in both the donor and the nurse. It has been shown that HCV exists as a dynamic quasi species, which can be defined as a complex population of closely related HCV variants circulating simultaneously in each individual patient [4]. HCV quasi species vary by <5% in nucleotide sequences [5]. Then, to prove that the HCV recovered from the nurse came from the inoculum and not from a different source, cloning and sequencing of the 5′ untranslated (UTR) and hypervariable 1 (HV1) regions of the HCV genome from donor and nurse were performed. Viral sequences showed total homology in the UTR region and a homology of >97% in the HV1 region among all of the sequenced clones from both individuals (figure 1), which confirmed that the nurse’s circulating HCV was transmitted by the accidental needlestick.

Because it has been reported that receipt of treatment during the acute phase of HCV infection can prevent the development of chronic infection [6], we decided to initiate IFN treatment for the nurse with the goal of preventing progression of infection. She started receiving IFN therapy (3 MU 3 times per week) 24 days after she received the needlestick injury, and she started receiving combination therapy with ribavirin (400 mg b.i.d.) 2 months after she began receiving IFN treatment, for 6 months of combined therapy. Three years after the needlestick accident, the nurse has had a sustained virological response, with no detectable serum HCV RNA and a normal ALT level.

We report, to our knowledge, the first case of very rapid and severe acute hepatitis C occurring after receipt of an accidental needlestick injury, with an incubation period of only 9 days. Moreover, we have shown that HCV can be transmitted with high efficiency by the percutaneous route with a high HCV RNA concentration in a small volume of inoculum in a patient without a history of injection drug use or blood/blood product transfusion. Also, this case reveals the importance of detecting HCV RNA soon after receipt of a needlestick to provide prompt treatment to the patient, regardless of whether it is necessary, to prevent chronic HCV infection.

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No Evidence for a Major Risk of Roseolovirus Vertical Transmission During Pregnancy

Sir—Human herpesvirus (HHV) 6 and HHV-7 are 2 lymphotropic viruses representative of the Roseolovirus genus within the Betaherpesvirinae subfamily. As with the human cytomegalovirus (HCMV), which belongs to the same subfamily, HHV-6 may be transmitted from mother to child during pregnancy, raising the possibility of congenital infections. Nevertheless, Roseolovirus transmission in utero is still little understood, and its association with the transmission of other viruses remains unknown. We made a preliminary study of selected amniotic fluid samples in order to evaluate the rate of Roseolovirus vertical infection and the potential role of betaherpesvirus interactions in this process.

Fifty-four specimens of amniotic fluid were tested. Amniotic fluid was collected from pregnant women who were classified into 3 distinct groups according to their status for viral infections. The first group consisted of 18 women with established HCMV primary infection and proven fetal infection, the second group consisted of 19 women with established rubella virus primary infection and transplacental transmission of the virus, and the third group consisted of 17 women with abnormal ultrasonographic findings but without established viral infection. Amniocentesis was performed between 14 and 40 weeks after amenorrhea (median, 23 weeks). HHV-6 and HHV-7 genomic DNA was looked for in DNA extracts of amniotic fluid by means of specific PCR and hybridization, as previously described [1]. The threshold of DNA detection was 1 genome-equivalent copy/mm$^3$.

No amniotic fluid specimen tested positive for either HHV-6 or HHV-7 DNA. In parallel, to check that this negative result was not due to the presence of PCR inhibitors, all amniotic fluid samples were spiked with a minimal amount of reference HHV-6 DNA and subsequently sub-