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Hepatitis G: Not Quite Another Step in the Alphabet

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Approximately 10% to 15% of patients with parenterally transmitted non-A, non-B hepatitis have no evidence of hepatitis C virus (HCV) infection and are classified as having non-A, B, C, D, E, or non–A-to-E, hepatitis. Compared to patients with acute HCV, patients with acute non–A-to-E hepatitis are less likely to be jaundiced, have lower peak alanine aminotransferase levels, and have a lower frequency of chronic hepatitis. Recently, a newly discovered virus isolated from patients with hepatitis, hepatitis G virus (HGV), has been described. Dr. Miriam J. Alter and coinvestigators from the CDC's Hepatitis Branch conducted a study to determine the characteristics of acute non–A-to-E hepatitis in the United States and the possibility that it is caused by HGV. Using the sentinel surveillance system, the Hepatitis Branch has operated since 1982, involving counties in four states. Patients were selected who had acute disease during 1985 to 1986 or 1991 to 1995. Serum samples were tested for HGV ribonucleic acid (RNA) by polymerase chain reaction. Hepatitis G virus RNA was detected in 4 of 45 patients with a diagnosis of non–A-to-E hepatitis (9%), 23 patients with HCV (20%), 25 of 100 patients with hepatitis A (25%), and 32 of 100 patients with hepatitis B (32%). The clinical characteristics of the acute illness were similar for patients with HGV alone and those with hepatitis A, B, or C with or without HGV infection. During a follow-up period of 1 to 9 years, chronic hepatitis did not develop in any of the patients with HGV alone, but 75% were persistently positive for HGV RNA, as were 87% of those with both HCV and HGV infection. The rates of chronic hepatitis were similar in patients with HCV alone (60%) and those with both HCV and HGV infection (61%). It was concluded that HGV may not be a hepatotropic agent, but may induce hepatitis only under certain circumstances, as seen with other viruses such as cytomegalovirus and yellow fever virus. HGV was not implicated as an etiologic agent of non–A-to-E hepatitis. Persistent infection with HGV was common, but it did not lead to chronic disease and did not affect the clinical course or patients with hepatitis A, B, or C.

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