Une traduction française suit cet article

Viral hepatitis: know your D, E, F and Gs

Over 90% of cases of acute viral hepatitis in the US (and presumably in Canada) are caused by hepatitis A, B or C virus (HAV, HBV, HCV).[1] As described below, there are other hepatitis viruses, some of which have been identified only recently. Still, the majority of non-A–C hepatitis cases in Canada are caused by as yet unidentified agents.

Hepatitis D

Hepatitis delta virus (HDV) requires the presence of HBV to replicate.[2] Therefore, HDV infection can occur as a coinfection at the time of HBV acquisition or as a superinfection of chronic HBV infection. HDV coinfection tends to increase the severity of acute HBV infection but decrease the risk of becoming an HBV carrier. HDV superinfection tends to enhance the frequency and severity of clinical sequelae of chronic HBV infection. Sexual and vertical transmission is less common with HDV than with HBV.

HDV infection can be detected by serologic tests. Interferon treatment is of uncertain benefit, and should be given for HDV infection only by physicians with extensive experience.[3] Prevention of HDV infection relies on the prevention of HBV infection (e.g., through immunization) or, if HBV infection is already present, reducing exposure to blood (e.g., by treating an addiction to injection drugs). The few reported studies indicate that the overall prevalence rate of HDV infection among people with HBV infection in Canada is generally low (< 5%); injection drug users may be at increased risk.[4]

Hepatitis E

Hepatitis E virus (HEV) is transmitted by the fecal–oral route, often through contaminated water. It is endemic and at times epidemic in the developing world.[5] A few cases have been identified in Canada; all were acquired during travel to endemic areas.[5] Clinically, HEV infection cannot be distinguished from HAV infection. The incubation period is from 2 to 9 weeks, and the disease is usually mild, resolving in a few weeks with no sequelae. The case-fatality rate is low except in pregnant women, among whom it may approach 20%.

There is no specific treatment for HEV infection. Immune globulin has not been found to be useful for prophylaxis. Prudent travel hygiene and sanitation are important to reduce the risk of infection. Laboratory diagnosis of HEV infection includes polymerase chain reaction (PCR) and serologic antibody detection. HEV infection should be considered in patients with acute hepatitis for whom HAV, HBV and HCV have been ruled out and who have travelled to the developing world within the incubation period.

Hepatitis F

Hepatitis F virus (HFV) has been described in only a handful of cases (from France) with subsequent experimental transmission to primates.[6] The virology, epidemiology, hepatotropicity and clinical importance of HFV are quite uncertain.

Hepatitis G

Hepatitis G virus (HGV; also called hepatitis GB virus C or HGBV-C) was fully characterized in early 1996.[7] HGV is a flavivirus and a distant relative of HCV. At this time, HGV infection can be identified only through PCR testing, which indicates current infection; such testing is not readily available or standardized. An antibody test for HGV is under development and, when available, will elucidate the epidemiology of HGV infection more fully than HGV RNA testing can. It appears that once antibodies are found HGV RNA is usually no longer present.[8]

The nature and frequency of HGV infection are unclear; there is also uncertainty about risk factors and means of prevention. Transmission through blood transfusion has been documented (the only Canadian study indicated that infection may occur in 1 in 1500 transfusion recipients and account for 9% of post-transfusion hepatitis[9]), and from mother to child in the perinatal period.[10] There is an increased prevalence of HGV RNA among groups with frequent exposure to blood or blood products (e.g., people with hemophilia or thalassemia, patients on hemodialysis and injection drug users).[7] Other modes of transmission (e.g., sexual) are possible but have not been well documented. Coinfection with HBV, HCV or both is common and likely represents similar modes of transmission.[7] In 0.3% of cases of community-acquired acute viral hepatitis, HGV is the only identified agent.[11]

It remains unclear what disease state HGV infection causes acutely and in the long term.[12] Although caution and vigilance must be maintained, there is a growing consensus that HGV is "a virus looking for a disease" and may in fact prove not to be a cause of viral hepatitis.[12–14] Acute HGV

infection is generally reported to be clinically and biochemically mild and transient. Although HGV RNA can be detected for years after infection in perhaps a minority of people who have been infected,[8] there is no compelling evidence that HGV infection has important sequelae. However, the role of HGV in fulminant hepatitis is an unresolved question.[12] HGV infection does not seem to worsen coinfection with HBV or HCV.[12] There is no proven treatment for HGV infection; at this point, guidelines for its investigation and management cannot be developed.

The threat that HGV may pose to the Canadian blood supply is an important issue. Among healthy blood donors, 1–2% may have HGV RNA,[7] and transmission through transfusion has been documented.[9] However, there is no commercial test available for screening; antibody testing, when it becomes available, may not be of particular value because it will not identify current infection; donors coinfected with HCV or HBV will largely be excluded already; and the infection seems largely benign. Rational risk management decisions regarding this issue will be difficult.

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Testing of clinical samples for HDV, HEV and HGV can be obtained from the Laboratory for Viral Hepatitis, Bureau of Microbiology, Laboratory Centre for Disease Control (613 957-0180). The specimen should be forwarded through the provincial laboratory.

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