

Etiological role of hepatitis E virus in sporadic fulminant hepatitis

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Abstract:

Non-A, non-B hepatitis viruses have been implicated as the etiological agent(s) in up to 60% of patients with fulminant hepatitis. These agents are reported to induce a higher mortality than other causes of fulminant hepatitis. Hepatitis E virus (HEV) and hepatitis C virus (HCV) at present constitute the major identifiable non-A, non-B hepatitis agents. Of these, HEV has been established as the sole cause of epidemic hepatitis in Afro-Asian countries, and fulminant hepatitis has been recorded during such epidemics. However, in sporadic cases, the etiological role of HEV in fulminant hepatitis has remained uncertain. The role of HCV in acute liver disease and fulminant hepatitis remains unclear. The present study was undertaken to investigate the association of HEV and HCV in patients with fulminant hepatitis by direct detection of the viral genome using reverse transcription-polymerase chain reaction (RT-PCR). Serum samples from 50 serologically identified non-A, non-B fulminant hepatitis cases negative for cryptic hepatitis B virus (HBV) infection examined via PCR were tested for HEV and HCV RNA using RT-PCR. For HEV primers from the nonstructural region (ORF-1) were used, and for HCV primers from the highly conserved 5' untranslated regions were used. The products were analysed using agarose gel electrophoresis and confirmed by hybridisation with radiolabelled internal oligonucleotide probes. HEV was detected in 31 (62%) of the 50 fulminant non-A, non-B hepatitis cases. In 18 (36%) cases, HCV RNA was detected. In 11 (22%) of the HCV cases, the HEV genome was also amplified. In 20 (40%) cases, HEV was detected alone. (ABSTRACT TRUNCATED AT 250 WORDS)