Hepatitis C virus infection in sporadic fulminant viral hepatitis in North India: cause or co-factor?

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Hepatitis C virus infection in sporadic fulminant viral hepatitis in North India: cause or co-factor?

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Abstract

Introduction: The role of hepatitis C virus (HCV) infection in fulminant hepatitis (FH) is poorly understood and the available data are conflicting. We have examined the aetiological role of HCV in 50 consecutive patients with sporadic FH by employing serology and reverse transcription-polymerase chain reaction (RT-PCR).

Materials And Methods: A total of 50 consecutive patients with sporadic FH were included. After an initial clinical and biochemical assessment, tests were performed for detection of HBsAg, IgM anti-HBc, IgM anti-HAV, IgM anti-HEV and anti-HCV. RT-PCR was carried out for detection of HCV RNA in sera of all the patients and in post mortem liver biopsy tissue of 20 subjects, using primers selected from the conserved 5' non-coding region of the HCV genome.

Results: Hepatitis E virus (HEV) was found to be the most common viral infection (21/50; 42%) followed by HBV (14/50; 28%), HCV (7/50; 14%) and HAV (2/50; 4%). No viral markers could be detected in nine patients (18%) and multiple infections were seen in seven (14%). Of the seven subjects who tested positive for HCV-related markers, two had both anti-HCV and HCV RNA, three had HCV RNA alone and the remaining two had anti-HCV alone. Interestingly, all the HCV-infected subjects were co-infected with other hepatotropic viruses and the most common co-infecting agent was found to be HBV (5/7). Liver tissue was available in 20 cases and HCV RNA was detected in three of them. All of these patients were also positive for the viral genome in their serum samples. Comparison of the biological attributes of HCV-positive and HCV-negative cases revealed that haemorrhagic symptomatology (haematemesis, melaena and purpurae) was significantly more common, prothrombin time more deranged and mortality was much higher in the former group. The overall mortality was 68% and the most common cause of death was cerebral oedema (70.6%). No significant correlation was observed between mortality and the duration of the icterus-encephalopathy interval. The study included a total of 21 pregnant females; HEV infection was found to be significantly greater in this group and was associated with a higher mortality rate.

Conclusions: The results clearly suggest that HCV is not an important aetiological factor for FH in North India. However, it may act as a co-factor in the development of FH leading to a

higher mortality. HEV appears to contribute substantially to the causation of sporadic FH in India and advanced stage pregnancy is a potential risk factor for HEV-induced FH and high rate of mortality. Our study also suggests that the length of the icterus-encephalopathy period may not have significant prognostic implications in Indian patients with FH.

Reference

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