Sporadic hepatitis E in southern India

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The definite existence of a second enterically transmitted hepatitis virus (non-A) was recognized in 1980 and retrospectively diagnosed to have caused the 1955 Delhi epidemic (KHUROO, 1980; TICEHURST, 1995). The name hepatitis E virus (HEV) was proposed for the agent in 1988; by the year 1990 its identity was firmly established when its genomic structure was elucidated (REYES et al., 1990). Until 1990, the virus was epidemiologically linked to epidemic hepatitis mainly in Asia, Africa and Central America. Since then, with evolving diagnostic techniques, it has been shown to be responsible for most sporadic and epidemic hepatitis in developing countries (TAN et al., 1995; TICEHURST, 1995). India has had 16 epidemics, some quite large (ARANKALLE et al., 1994). In addition, HEV is estimated to cause around 60% of the sporadic illness in India (ARANKALLE et al., 1993). Most of the reports from the subcontinent contributing to these figures have been from the northern, and some from the western, parts in south India, where there has been an occasional report (FAGAN et al., 1994), the role of HEV in sporadic or epidemic viral hepatitis has not been documented. Hence we decided to investigate its presence in this area as evidenced by serological testing. While the possibility of hepatitis E disease has been considered for some time, easily applicable diagnostic tests for HEV acute phase antibodies have only recently become available. In this paper we report the prevalence of sporadic cases of the disease in southern India as indicated by the occurrence of anti-HEV immunoglobulin M (IgM) in the absence of any recognizable epidemic.

Between May and December 1996, sera from 69 cases of acute viral hepatitis in Tamil Nadu and Andhra Pradesh States, India, were tested for anti-hepatitis A virus IgM using a commercial assay (Abbott, USA). Samples giving negative results were subjected to an enzyme-linked immunosorbent assay for IgM antibodies to HEV (Genelabs Diagnostics, Singapore); the test was performed according to the manufacturer's instructions. Briefly, sera were added in a 1:21 dilution to the wells of a plate coated with recombinant structural proteins of HEV. Following washing, the plates were incubated with labelled mouse monoclonal anti-human IgM. Unbound antibodies were removed by a second wash and a substrate solution added. A stop solution was then added and the optical density (OD) measured at 490 nm. All samples reactive above the cut-off OD value were retested in duplicate; only those that gave repeatedly reactive results were considered positive.

Among the 69 samples from as many patients, 8 (11.5%) contained anti-HAV IgM antibodies. On subsequent testing 40 of the 61 negative sera for anti-HEV IgM, 13 samples (32.5%) were found to be repeatedly reactive and hence positive for anti-HEV; 6 samples were from females and 7 from males; their ages ranged between 18 and 42 years.

This is a preliminary study and had a single objective, to investigate the occurrence of HEV in sporadic cases of acute hepatitis in southern India. Our results showed that one-third of the patients presenting with acute hepatitis, in whom hepatitis A was ruled out, had HEV infection. Therefore HEV can be considered to be an important pathogen in south India. Considering the high mortality associated with HEV infection in pregnancy, the epidemiology of this virus needs to be further investigated. The finding that HAV accounts for only about 12% of all cases of acute hepatitis is contrary to the general perception of the role of this virus as a major cause of acute hepatitis. Furthermore, a large number of samples gave negative test results for both viruses. This highlights the need for a comprehensive investigation of viral pathogens, especially in sporadic cases of acute hepatitis.

References


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