Viral hepatitis E epidemiology and prevention

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Viral hepatitis E: epidemiology and prevention

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Abstract

The identification in the 1970s of the hepatitis A virus (HAV) and the hepatitis B virus (HBV) allowed to individualize hepatitis with the characteristics of viral hepatitis, related to neither of these two viruses and baptized by exclusion viral hepatitis "no A-no B" [1 , 2 , 3]. Non-A-non-B hepatitis covered two distinct epidemiological and clinical entities. Parenteral non-A-non-B hepatitis was primarily related to blood and blood products transfusion and venous drug use in industrialized countries; they were also present in developing countries; the causative agent, hepatitis C virus (HCV), was identified in 1989 by an exclusively molecular approach [4]. Non-A-non-B enteral, fecal-oral hepatitis was responsible for large epidemics of acute hepatitis and high mortality in pregnant women in developing countries.

The first experimental demonstration of the existence of a transmissible agent responsible for enteral non-A non-B hepatitis dates back to 1983 [5]. In electronic immunomicroscopy, Balayan visualized virus-like particles in his own stool after ingesting purified stool extracts from non-A non-B enteral-transmitted hepatitis patients collected during an outbreak in Tashkent, Uzbekistan. . Secondary inoculation of monkeys Cynomolgus macaques with particles of this new virus, dubbed hepatitis E virus (HEV), established an animal model of infection. It was not until 1990, one year after HCV, that the HEV genome was in turn cloned and sequenced [6].]. Since then, HEV research has remained relatively confidential, probably because the disease mainly affects developing countries and is benign in the majority of cases. HEV, however, is an original virus, in terms of its virological characteristics as well as its epidemiology or its pathogenicity. The World Health Organization (WHO) estimates that HEV is today the most common cause of acute hepatitis in adults in developing countries[7].

References

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