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Epidemiological Investigation of Hepatitis F Viruses (HFV)

Haradhan Kumar Mohajan[1]

[1] Associate Professor, Department of Mathematics, Premier University, Chittagong, Bangladesh

Correspondence: Haradhan Kumar Mohajan, Associate Professor, Department of Mathematics, Premier University, Chittagong, Bangladesh.

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Abstract

The hepatitis is a liver inflammation that is related to hepatocellular necrosis. Viral hepatitis may be caused by various hepatitis viruses, such as A, B, C, D, E, F, and G. Hepatitis F is a hypothetical virus linked to viral hepatitis. Sporadic non-A, non-B hepatitis, such as hepatitis F is the most common, presumed viral that may cause acute liver failure. A novel agent called hepatitis French virus (HFV) was present as 27-37nm particles in the infectious stool extract of French patients. The virology, epidemiology, hepatotropism, and clinical importance of HFV are quite uncertain, and are not determined yet. This study tries to discuss the known structure and other clinical features of HFV.

Keywords: Hepatitis F virus, non-A-E hepatitis, liver failure, rhesus monkeys

1. Introduction

Hepatitis F is a hypothetical virus linked to viral hepatitis. At present seven viruses: A, B, C, D, E, F, and G have been described as agents of acute or chronic hepatitis infection. Of these five hepatitis viruses A, B, C, D, and E are all well-characterized (Yasmin et al., 1997). An enteric agent responsible for sporadic non-A, non-E hepatitis is tentatively called hepatitis F virus (HFV) or hepatitis French virus (HFV) and has been described by two groups. In 1992, the first group has realized that a toga virus like agent was visualized by electron microscopy from liver biopsies of patients with unexplained fulminant viral hepatitis (Fagan et al., 1992). In 1994, the second group has realized that a virus from the feces of a patient with hepatitis and was transmitted to primates. But the role of this virus remains unclear (Deka et al., 1994).

Some cases of hepatitis transmitted through contaminated food or water are attributed to the HFV, which was first reported in 1994. Several hepatitis F candidates emerged in the 1990s; however, none of these claims were substantiated. In December 1994, researchers published evidence for the transmission of the enteric agent responsible for sporadic non-A, non-E hepatitis to rhesus monkeys that measuring 27-37nm in diameter (Kim et al., 1995).

2. Literature Review

The literature review section is an introductory region of research, which shows the works of previous researchers in the same field within the existing knowledge (Polit & Hungler, 2013). Niren Deka and his coauthors have indicated that 27 to 37-nm virus like particles are responsible for sporadic non-A, non-B hepatitis in rhesus monkeys. They have shown that sporadic non-A, non-B hepatitis in humans is serially transmitted in rhesus monkeys by intravenous inoculation of the stool extract from a patient (Deka et al., 1994). Scott Bowden has found that the HFV has a high prevalence in cases of non-A-E hepatitis, it also has a high prevalence in the appropriate control groups and convincing evidence for its replication in the liver is lacking (Bowden, 2001).

Krisnan Rajeshwari has taken communication attempts to summarize the recent relevant information in relation to the newer discovered hepatitis viruses through the application of the latest molecular technology to the

primate model (Rajeshwari, 1997). Elizabeth Ann Fagan and Tim J. Harrison have determined whether HAV, HEV, HCV, or HBV were detectable in prospectively stored hepatectomies from seven British patients grafted for acute liver failure attributed to sporadic non-A, non-B hepatitis. They have considered it candidate hepatitis F virus (HFV) in sporadic non-A, non-B acute liver failure (Fagan & Harrison, 1995). W. Keith Paver and Philip P. Mortinzer have indicated that a 60-nm particle is seen in the livers of patients with fulminant hepatitis, aid in subsequent graft necrosis following liver transplantation, and is originally proposed as hepatitis F virus (Paver & Mortinzer, 1996).

3. Research Methodology of the Study

Research is an essential device to the academicians for the leading in academic area (Pandey & Pandey, 2015). Methodology provides the research design and analysis procedures to perform a good research (Hallberg, 2006). Hence, research methodology is the collection of a set of principles for organizing, planning, designing and conducting a good research (Legesse, 2014). In this paper, I have depended on the secondary data sources of optimization, such as journal articles, books of famous authors, conference papers, internet, websites, etc. (Mohajan, 2017, 2018, 2020, 2024a-l).

4. Objective of the Study

Main objective of this article is to discuss the basic concept of hepatitis F virus (HFV). The exact role of HFV in human disease is not clear and needs more exploration (Rajeshwari, 1997). Other minor objectives of the study are as follows:

- to focus on the historical background of HFV,
- to highlight virology and symptoms of HFV, and
- to show diagnosis and treatment of HFV.

5. Historical Background of HFV

The 1980s' investigators of England, Italy, France, the USA, and India have studied on the sporadic non-parenteral non-A, B, C, D, E hepatitis (Deka et al., 1994). In 1987, the disease was transmitted to cynomolgus macaques and tamarins, and 27-34nm viral-like particles that consist of double-stranded DNA of approximately 20kb were observed in stool samples by electron microscopy (Bradley et al., 1987). The HFV is substantially different from HAV and HEV, and both of which consist of single-stranded RNA of approximately 7.5kb (Bowden et al., 1996). In December 1994, a group from New Delhi, India claimed to have transmitted the enteric agent responsible for sporadic non-A, non-E hepatitis in humans to rhesus monkeys using a stool extract from a patient. They have reported that the viral particles represented a "novel agent" that they called HFV (Deka et al., 1994).

6. Virology of HFV

Hepatitis F virus (HFV) first appeared as togavirus-like 60-70nm enveloped particles that were recovered from the hepatocytes of a number of patients transplanted for fulminant hepatic failure (Kim et al., 1995). The HFV is a purified viral 27-37nm consists of a double-strand DNA of 20kb that is incongruous considering the size of the virion, and is detected in infected monkey liver (Deka et al., 1994).

7. Symptoms and Transmission of HFV

The liver morphology of HFV infected persons may be of an acute hepatitis, and 20% of the cases the fatality of the disease becomes sever. In infected animals viral antigens and elevation of transaminases appear in an average of 20 days (Deka et al., 1994). The infection was not only sporadic but also enterically transmitted (Bradley et al., 1987).

8. Diagnosis and Treatment of HFV

The HFV antigen has been detected by enzyme-linked immunosorbent assay (ELISA) in 66% of coded specimen (Deka et al., 1994). The first- and second- round primers in a polymerase chain reaction (PCR) to amplify conserved regions of HAV, HEV, and HCV; the E1/S (gp35) region of HCV, and surface and core regions of HBV (Fagan & Harrison, 1995).

9. Conclusions

From this study, I have realized that HFV is transmitted in humans through the rhesus monkeys. The HFV genome is 27-37nm in diameter about 20kilobases of double-stranded DNA. More epidemiological studies are required to determine the complete knowledge on HFV.

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