Introduction

Viral hepatitis is a major public health problem throughout the world, hepatitis infection is particularly endemic in certain areas of the world including North Africa *(WHO, 1991)*. It is a systematic viral infection in which hepatic cell necrosis and hepatic inflammation lead to a characteristic constellation of clinical, biochemical, immunoserologic, and morphologic features *(Koff, 1992)*.

The identification of the major agents causing human hepatitis (Hepatitis A, B, C, D, and E viruses) was achieved during the last 30 years, but there are still new viruses, responsible for human hepatitis cases without evidence of infection by any of these viruses, that could be called hepatitis G virus and hepatitis F virus *(Pinho and Silva, 1998)*.

Hepatitis C virus (HCV) is a positive single stranded RNA virus with genomic structure and organisation similar to those of Pestiviruses and Flaviviruses *(Altamarino et al., 1995)*.

Additionally, HCV is characterised by a high mutation rate and considerable genomic heterogenicity *(Houghton et al., 1991)*.

Kuo et al., (1989), reported that HCV was coloned and characterised as an RNA virus which was the major cause of post-transfusion and community aquired (or sporadic) non-A, non-B hepatitis, with approximately 80% of patients having antibodies (anti-HCV) against the virus.

Clinically HCV is associated with acute hepatitis, chronic active hepatitis, cirrohsis of the liver and hepatocellular carcinoma. It is transmitted mainly by blood transfusion, intravenous drug users, and less frequently from mother to offspring, or to organ transplant recepient ***(Cuthber, 1994)***.

The data of ***Brillianti et al., (1993)***, supported the concept of existence of healthy HCV carrier state. However, considerable controversy exists regarding the existance of a "true" healthy HCV carrier state. ***Prieto et al., (1995)***, reported that their data were in agreement with previous studies with regard to the high prevalence of chronic liver disease in apparently healthy HCV positive donors .

Hagiwara et al., (1993), noticed the presence of low viral level in serum of anti HCV positive subjects with normal liver tests. On the basis of anti HCV screening symptoms free individuals with positive HCV antibody and normal liver functions have been proved to exist.

The diagnosis of hepatitis C should be based upon clinical history of exposure to blood or blood products and the absence of any other apparent cause of liver disease. Testing for anti HCV can be used to confirm the diagnosis. Currently, the commercially available assay is ELISA for antibody to HCV.

PCR can be used for detection of HCV RNA in serum and measuring any low level of viremia occurs during the incubation period and extends into the symptomatic phase of the disease ***(Lindsay and Hoofnagle, 1992)***.

Numerous techniques to genotype HCV isolates have been discribed, these methods generally use the PCR to ampilify subgenomic HCV RNA. Genotype identification has been done by using specific primers for PCR *(Okamoto et al., 1992)*.

Hepatitis C virus demonstrates a high degree of variability HCV isolates have been classified into at least six genotypes. *(Nousbaum, 1998)*.

In Western Europe, HCV infection in patients with liver disease and in blood donors is predominately caused by genotypes 1b, 2b, and 3a, with some variation in frequency. In Japan and Taiwan, genotypes 1b, 2a and 2b are seen most frequently; elsewhere in Asia, type 3 is the most common. Type 4 is found frequently in the Middle East. *(Dusheiko et al., 1994)*.

Evidence that the genotype of HCV is predictive of the response to interferon-alpha therapy suggests that typing methods are clinically useful *(Lee et al., 1997)*. Also, the variability of HCV has major implications for the design of new vaccines strategies *(Nousbaum, 1998)*.