

INTRODUCTION

Hepatitis C virus (HCV) infection is a global health problem, with an infection rate of 3% of the world's population equating to 170-200million individuals (*WHO, 2009*).in the US,approximately 3 million people are chronically infected,many of whom are still undiagnosed. Egypt has the highest prevalence of hepatitis C in the world, reaching 13% of the population equating to an estimated 10 million anti-HCVpositive persons (*Deuffic-Burban et al., 2006, Mohamed,2004*).Almost 90% of HCV infections are caused by genotype 4, which is the major cause of the high prevalence of liver cirrhosis, hepatocellular carcinoma and liver transplantation in the country (*Wall et al 2003 ;Tanaka et al.,2004;Ezzat et al,2005 ; Kamal et al., 2008;NCR,2007*).

the major clinical consequence of acute hepatitis c infection is evolution into chronic hepatitis in 70-80% of infected individuals which leads to liver cirrhosis in 20-40% with an accompanying risk of hepatic decompensation, HCC, deaths (Seef,2002; MeHutchison,2004). the fibrosis progression in chronic hepatitis c genotype 4 has been assessed in few longitudinal studies(**Kamal et al 2004;Kamal et al 2006**).patients with chronic HCV genotype 4 had a disease progression rate of 0.1 ± 0.06 fibrosis units per year,which is not significantly different from rates reported in genotype 1,2 or 3 (**poynard et al 2001**).

steatosis is a condition characterized by the deposition of fat within the liver,triggering inflammation of the liver.It is also known as fatty liver.steatosis is a common feature of many liver diseases(namely non alcoholic steatohepatitis (NASH) and hepatitis c virus infection(**Ramalho,2003**).Hepatic steatosis is a frequent histological finding in patients with chronic HCV and it is one of the characteristic features of chronic hepatitis c (**Patel et al,2005**).chronic hepatitis c steatosis is characterized by excessive accumulation of triglycerides in hepatocytes in the absence of significant alcohol consumption,defined as more than 5% fat by weight (**Neuschwander et al 2003**).

Introduction

The overall prevalence of steatosis in patients with Hepatitis C virus (HCV) chronic infection is 55.5% (range 34.8–81.2%). Several studies have demonstrated that both host and viral factors may contribute to the development of steatosis with relative importance of each varying with each HCV genotypes (*Poynard et al., 2003*).

Steatosis is detected in 40-50% of chronic hepatitis genotype 4 compared to 72% in genotype 3 (*Poynard et al., 2003*). One of the features of chronic hepatitis genotype 4 is the presence of moderate to severe steatosis (*El-Zayadi et al., 2007; Kamal et al. 2007; Kamal et al., 2008*) with no associated sinusoidal fibrosis (*Kamal et al., 2007*). The steatosis associated with HCV genotype 4 is probably attributed to metabolic factors (*EL-Zayadi et al., 2007*).

The currently recommended therapy of chronic HCV infection is the combination of pegylated interferon and ribavirin. Randomized controlled studies of pegylated interferon alfa 2b and ribavirin demonstrated higher SVRs rates in patients infected with genotype 2 or 3 and those with lower body weight and absence of bridging fibrosis and cirrhosis (**Fried et al.2002**).sustained virologic response rates in chronic hepatitis c genotype 4 are better than those achieved in genotype 1 (**Kamal et al.2005; El-Zayadi et al.2005 ; Kamal et al. 2008 ; Ferrenci et al, 2008**).

several factors have been suggested as predictors of response and non response to the therapy.

Younger age,female gender,lower viral load,non 1 genotype,lower histologic staging have been linked with higher sustained virologic response rates.