Introduction

An estimated 130–170 million people worldwide are infected with hepatitis C. The existence of hepatitis C (originally "non-A non-B hepatitis") was postulated in the 1970s and proven in 1989 **(*Houghton, 2009*).**

Countries with particularly high rates of infection include Egypt (22%), Pakistan (4.8%) and China (3.2%) ***(WHO,2011).***

It is believed that the high prevalence in Egypt is linked to a discontinued mass-treatment campaign for schistosomiasis, using improperly sterilized glass syringes **(*Alter, 2007*).**

There is a growing evidence suggesting an association between hepatitis C virus (HCV) infection and diabetes, two common disorders that cause devastating long-term complications in a significant number of patients. Several reports have found a high prevalence of HCV infection among diabetic patients ***(Mason et al., 1999)***.

Insulin resistance seems to be related to poor response to antiviral treatment in chronic hepatitis C *patients* ***(Romero-Gomez et al., 2005).***

The specific mechanisms by which HCV leads to type 2 diabetes are not fully understood. There is a suggestion that insulin resistance (mediated by proinflammatory cytokines) and not a deficiency of insulin secretion, is the main mechanism involved in the pathogenesis of diabetes associated with HCV infection ***(Lecube et al., 2006).***

Lecube & **colleagues** found that impaired fasting glucose and type 2 diabetes influenced the response to antiviral therapy with interferon plus ribavirin in treatment-naive patients with chronic hepatitis C. Fasting plasma glucose was measured prior to starting interferon-based therapy ***(Lecube et al.,2004).***

The relationship between hyperglycemia and the reduced treatment response in HCV infected patients may be mediated through several mechanisms; **hyperinsulinemia** blocks the inhibition of HCV virus replication by interferon, thus reducing the efficacy of antiviral therapy ***(Sanyal et al., 2004).***

Hyperglycemia itself could be involved in the impairment of HCV clearance. Hyperglycemia can impair a wide range of functions in neutrophils and macrophage including chemotaxis, adherence , phagocytosis and intracellular killing of microorganisms ***(Pozzilli and Leslie, 2004).***