Introduction and aim of the work

Introduction

Hepatitis C virus (HCV) is a major health problem and chronic infection with this virus remains one of the most prevalent chronic diseases as well as an economic burden worldwide. It has been estimated that globally over 170 million people are infected with chronic HCV infection and about 20-25% of them are at risk of developing cirrhosis or even end stage liver disease (Mazur et al., 2010). In Egypt, the problem is even more evident. HCV infection is the most important cause of liver disease in Egypt according to Habib et al., (2001) as the prevalence of antibodies to HCV is estimated to be 10 fold greater than Europe and the US (Alter et al., 1999).

It has been estimated that 10-20% of the population were infected with HCV in addition to it being the leading cause of cirrhosis and hepatocellular carcinoma (HCC) in this country (AbdelAziz et al., 2000). Although in its natural history, chronic HCV infection is a slowly progressive disease. It is important to remember that some factors are associated with a rapid progression of liver injury into fibrosis (Mazur et al., 2010).

Adipose tissue, previously thought of as a passive storage site for excess energy, is now recognized as a hormonally active system producing numerous molecules known as adipokines, which exert local, central and peripheral actions. During the last decade, interest has turned to the study of this group of molecules and their role in chronic liver diseases (El-Raziky et al., 2009)

Resistin is one such adipokine, it is formed of a 10 kDa protein composed of 94 amino acids. It was cloned in 2001 and is thought to be mainly expressed in adipose tissue. Resistin was shown to be involved in hepatic glucose and lipid metabolism and appears to play a pivotal role in hepatic insulin resistance (Murad et al., 2010). There have been a number of studies observing serum resistin levels in chronic liver diseases but they have mostly dealt with patients suffering from non-alcoholic fatty liver disease (NAFLD). In patients with NAFLD, abnormalities in the various adipokines (Resistin and leptin in particular) have been documented and possibly implicated in the progression of this disease towards cirrhosis (Wong et al., 2009).