

INTRODUCTION

The liver is the major site for cholesterol and triglyceride metabolism, and the thyroid hormones play an integral part in hepatic lipid homeostasis. Thyroid hormones increase the expression of low density lipoprotein receptors on the hepatocytes, and increase the activity of lipid-lowering liver enzymes, resulting in a reduction in low-density lipoprotein levels (Malik and Hodgson 2002)

Autoimmune disorders have been described in association with chronic hepatitis C infection (HCV), including thyroid disease, including hypothyroidism, sialadenitis, autoantibody formation and autoimmune idiopathic thrombocytopenic purpura (*Maribel et al; 2008*)

Patients with chronic hepatitis C and severe fibrosis have higher probability of prior history of thyroid dysfunction(TD) and develop more frequently TD events during treatment with Peg-IFN Alfa-2a than patients with mild fibrosis. The number of TD markers at baseline in the patients with severe fibrosis is among the higher levels reported This suggests a relationship between liver damage (fibrosis) progression in chronic hepatitis C and the risk for TD (*Antonelli et al ;2004*)

Anti thyroid antibodies are present in 5-17% of patients with HCV infection and thyroid dysfunction occurs in 2-13% of patients A higher prevalence in older women has been reported that this finding is controversial (*Antonelli et al ;2004*)

Thyroid dysfunction is also a known adverse event or complication during interferon treatment. It has been reported to be more frequent in females, and to be mostly hypothyroidism (both clinical and biochemical) and to resolve after end of therapy (*Tran et al ., 2005*)

It has been recently reported that HCV patients developing thyroid dysfunction during IFN- α treatment displayed circulating markers of a Th1 immune reaction, as assessed by IFN- γ expression by peripheral blood lymphocytes (*Mazzaiotti et al 2005*).

The appearance of autoantibodies or clinical manifestations of autoimmunity, during the treatment with interferon α , is associated with a statistically significant improvement in the relapse-free survival and in the overall survival of the patients with melanoma (*Mario et al 2007*)

In acute hepatitis of mild or moderate severity, patients have elevated serum levels of total T4, due to increased thyroid-binding globulin, which is synthesized as an acute-phase reactant, but normal levels of free T4. In more severe cases with impending liver failure, the data is variable, and low total T4 levels may reflect reduced hepatocellular synthesis of thyroid-binding globulin(*Malik And Hodgson 2002*)

patients developing thyroid dysfunction were also more likely to display a favourable response to IFN- α treatment, showing nearly a three times higher rate of success (*Mario et al 2007*)

AIM OF THE WORK

The aim of our work is to study thyroid dysfunction among chronic hepatitis c patients and its relation to hepatic fibrosis .