The magnitude of steatosis in non -obese patients with chronic HCV

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M.Sc. Degree of Endemic medicine

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Abstract

This study included 566 consecutive infected chronic HCV patients recruited from the Hepatology outpatient clinic in Al Quahira El-Fatemia Hospital between January2007 & January 2009. The patients' age ranged from 18 - 60 years old with the male: female ratio was 1:4.8. Patients BMI ranged between 19 to<30 Kg/m². They divided into groups according to histopathological examination of METAVIR group scoring system of hepatic steatosis into two groups (steatotic &non- steatotic patients).

This study aimed to evaluate detection of hepatic steatosis & its correlation with degree of hepatic necroinflammatory activity& fibrosis in the histopathological examination of liver biopsy according to the METAVIR group scoring system of hepatic steatosis in non–obese Patients(BMI 19 to<30 Kg/m²) chronic infected HCV patients.

Key Words :

Amino acid - Bile capillary - Cyto Keratin .

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List of Abbreviations

Aa	Amino acid
A1AT	Alpha 1- Antitrypsin Deficiency
AIH	Auto Immune Hepatitis
ALT	Alanine Transeaminase
AST	Aspartate Transaminase
ATP	Adenosine Triphosphate
Bc	Bile capillary
BMI	Body Mass Index
CAH	Chronic Active Hepatitis
CHC	Chronic Hepatitis C
CK	Cyto Keratin
CLH	Chronic Lobular Hepatitis
CPH	Chronic Persistent Hepatitis
ECM	Extra Cellular Matrix
Es	Examination Survey
FFA	Free Fatty Acids
GGT	Gamma Glutamyl transeferase
HAART	Highly Active Anti Retroviral Therapy
HBV	Hepatitis B Virus
HCC	Hepato Cellular Carcinoma
HCV	Hepatitis C Virus
HIV	Human Immunodeficiency Virus
HSCs	Hepatic Stellate Cells
INF-ALPHA	Inter Feron Alpha
IKKB	Inhibitor Kinase Kappa Beta

IL	Inter Leukin
LVPs	Lipo Viral Particles
MH	Mallory Hyaline
MSSH	Metabolic Syndrome SteatoHepatitis
MTP	Microsomal Triglycerides transfer Protein
NAFLD	Non -Alcoholic Fatty Liver Disease
NASH	Non- Alcoholic SteatoHepatitis
NHANES	National Health And Nutrition
PAT	Parenteral AntiSchistosomal Therapy
PBC	Primary Biliary Cirrhosis
PMNLs	Polymorph Nuclear Leucocytes
PPAR Gamma	Peroxisome Proliferator-Activated Receptor Gamma
RNA	Ribonucleic Acid
ROS	Reactive Oxygen Species
SREBP	Sterol Response Element Binding Protein
SVR	Sustained Virological Response
TNF ALPHA	Tumor Necrosis Factor Alpha
TUNEL	Tdt-mediated UTP-x Nick End Labeling
TzDs	ThiazoliDineiones
UNOS	United Network For Organ Sharing
UTR	UnTranslated Region
VLDL	Very Low Density Lipoprotein
VP	Vascular Pore
WHR	Waist Hip Ratio

Introduction

HCV is now recognized as one of the major causes of chronic liver diseases worldwide. One of the striking features of HCV infection is the very high rate of development of chronicity **(Yenigum and Durupinar, 2002).**

Persistence of the virus results in a wide spectrum of chronic liver lesions ranging from minimal inflammation to cirrhosis or hepato-cellular carcinoma **(Yee, 2004).**

Liver steatosis is a frequent finding in chronic hepatitis C. An association has been suggested between steatosis and fibrosis progression rate, but the pathogenesis mechanisms linking fatty infiltration and collagen deposition are unknown **(Adinolfi et al., 2001)**

Current data suggest that there are at least two types of steatosis in HCV patients:

1. Virally-related steatosis, which correlates with HCV replication level, is often associated with genotype 3, and disappears upon successful antiviral therapy.
2. Metabolic steatosis, whose presence and severity correlate with the body mass index (BMI), and that does not respond to antiviral treatment, even in virological responders **(Patton et al., 2004).**

Among patients infected with HCV, 13-33% developed type 2 diabetes mellitus. Whether this reflects an HCV- mediated effect remains to be established **(Knobler and schattner, 2005).**

Many research results have linked for the first time insulin resistance to fibrosis in patients with chronic hepatitis C and showed that insulin resistance syndrome was capable of influencing the degree of both steatosis and fibrosis in chronic hepatitis C. They have also suggested the presence of a factor? (HCV) other than overweight and/or obesity involved in the pathogenesis of insulin resistance **(Ratziu et al., 2003).**

The presence of steatosis on liver biopsy in patients with hepatitis C is more frequent when compared to other chronic liver diseases such as chronic hepatitis B and autoimmune hepatitis **(Hwang et al., 2001).**

It has been shown that HCV genotype -3 is independently associated with hepato-cellular steatosis in patients with chronic hepatitis C **(Rubbia-Brand et al., 2000).** Furthermore, the severity of steatosis in these patients is directly related to the burden of the HCV RNA load. This relationship between the HCV viral load and the magnitude of steatosis was not observed in other HCV genotypes **(Hezode et al., 2004).**

Aim of work

This study aimed to evaluate detection of hepatic steatosis degree (no, mild, moderate, severe) & its correlations with the degree of hepatic necro-inflammatory activity (HAI) & hepatic fibrosis in the histo-pathological examination of liver biopsies of non–obese BMI=19 to 29.9 kg/m^2) chronic HCV patients.