

# **Relation Between Hepatic Steatosis In Hepatitis C Virus Infection And Genotype (4)**

## **Thesis**

Submitted for partial fulfillment of Master Degree  
in Internal Medicine

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**2009**

# رصد العلاقة بين مستوى التغير بدهون الكبد والنوع الجيني رقم (4) لفيروس سى الكبدى

رسالة مقدمة لنيل درجة الماجستير فى امراض الباطنة

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بمستشفيات القوات المسلحة

كلية الطب

جامعة عين شمس

# **Acknowledgement**

He, and will always be, Allah who always blessed my work and who sent me those who were of help .

I would like to thank Allah a lot for his kindness patience and strength he gave to me to achieve this work and made me able to finish it.

I wish to express my sincere gratitude to **Dr. Amr Abd El Kader Ahmed Fateen** professor of Internal Medicine Faculty of Medicine, Ain Shams University. He patiently gave me much of his time, experience, knowledge, directions, supervision and support that can not be expressed by words. From him, I learnt the way of scientific research.

My deep thanks and appreciation to **Dr. Amany Talaat Kamal Hanna**, Assistant Professor of Internal Medicine, Ain Shams University, for her strict supervision and revision of this work, She gave me much of her time, experience and support, her valuable comments, efforts and collaboration were the causes to complete this work properly, so no words can express my gratitude to her.

I am greatly honored to express my deep gratitude and faithfulness to **Dr. Ahmed Mohamed El Sawy**, Consultant of Gastroenterology and Hepatology, Military Hospitals. He gave me his valuable advices and support that cannot be expressed in words. His fatherhood attitude and encouragement were so supportive for the completion of this work.

Words stand short to express my deep appreciation for the staff members of the **Central Military Laboratory** and my colleagues by whom all, I was very much impressed by the noble characters, generous attitude and kind sympathy.

My deep thanks to all my colleagues, all patients included in this work and every one made any effort for this work to be a reality.

A special dedication to my family for their never ending care. They were always supporting me and encouraging me to continue and finish this work.

***Ahmed Ibrahim***

# **LIST OF TABLES**

<b>NO</b>	<b>Title</b>	<b>Page</b>
<b>Table (1)</b>	Demonstrated HCV estimated prevalence and number infected by WHO region.	8
<b>Table (2)</b>	Factors that could have an impact on progression of chronic hepatitis C.	21
<b>Table (3)</b>	Degrees between steatosis and fibrosis	57
<b>Table (4)</b>	Modified HAI grading: necroinflammatory score.	97
<b>Table (5)</b>	Modified HAI grading: fibrosis score.	99
<b>Table (6)</b>	Grading of steatosis severity.	99
<b>Table (7)</b>	Excluded cases	102
<b>Table (8)</b>	Studied cases as regard general data.	108
<b>Table (9)</b>	Studied cases as regard laboratory investigations.	110
<b>Table (10)</b>	Studied cases as regard lipid profile.	112
<b>Table (11)</b>	Studied cases as regard degree of steatosis, inflammatory score, fibrotic score, subtypes of genotype 4 and PCR.	114
<b>Table (12)</b>	Comparison between both studied groups as regard general data.	116
<b>Table (13)</b>	Distribution of the studied non steatotic cases as regard liver functions, PCR, FBS, PPBS.	116
<b>Table (14)</b>	Distribution of the studied steatotic cases as regard liver functions, PCR, FBS, PPBS.	116
<b>Table (15)</b>	Comparison between studied groups as regard liver function, PCR, Albumin, FBS, PPBS.	117
<b>Table (16)</b>	Distribution of the studied non steatotic cases as regard lipid profile.	117

<b>Table (17)</b>	Distribution of the studied steatotic cases as regard lipid profile.	118
<b>Table (18)</b>	Comparison between studied groups as regard lipid profile.	118
<b>Table (19)</b>	Comparison between studied groups as regard gender.	118
<b>Table (20)</b>	Comparison between studied groups as regards fibrotic score.	119
<b>Table (21)</b>	Comparison between studied groups as regards inflammatory score.	119
<b>Table (22)</b>	Distribution of the total group as regard subtypes of genotype 4.	119
<b>Table (23)</b>	Comparison between studied groups as regard subtypes of genotype 4.	120
<b>Table (24)</b>	Comparison between steatotic groups as regard general data.	120
<b>Table (25)</b>	Comparison between steatotic groups as regard liver function, PCR, Albumin, FBS, PPBS.	120
<b>Table (26)</b>	Comparison between steatotic groups as regard lipid profile.	121
<b>Table (27)</b>	Comparison between steatotic groups as regard gender.	121
<b>Table (28)</b>	Comparison between steatotic groups as regards fibrotic score.	122
<b>Table (29)</b>	Comparison between steatotic groups as regards inflammatory score.	122

# **LIST OF FIGURES**

<b>NO</b>	<b>Title</b>	<b>Page</b>
<b>Figure (1)</b>	Natural History of HCV Infection.	20
<b>Figure (2)</b>	Worldwide geographic distribution of HCV genotypes and subtypes.	48
<b>Figure (3)</b>	Histologic appearance of hepatic fatty change.	56
<b>Figure (4)</b>	Correlation between severity of histopathological steatosis and serum albumin.	123
<b>Figure (5)</b>	Correlation between severity of histopathological steatosis and serum bilirubin.	124
<b>Figure (6)</b>	Correlation between severity of histopathological steatosis and serum triglyceride.	125
<b>Figure (7)</b>	Correlation between severity of histopathological steatosis and FBS.	126
<b>Figure (8)</b>	Correlation between severity of histopathological steatosis and fibrosis score according to Modified HAI grading.	127
<b>Figure (9)</b>	Correlation between severity of histopathological steatosis and inflammatory score according to Modified HAI grading.	128
<b>Figure (10)</b>	Correlation between severity of histopathological steatosis and HCV RNA quantitative PCR.	129

# **TABLE OF CONTENTS**

<b>NO</b>	<b>Title</b>	<b>Page</b>
	Acknowledgment	I
	Abstract	VII
	Introduction	1
	Aim of work	3
<b>Chapter (1)</b>	HCV Infection.	4
<b>Chapter (2)</b>	Steatosis	56
<b>Chapter (3)</b>	Relation between HCV and Steatosis.	68
<b>Chapter (4)</b>	Patient and methods	91
<b>Chapter (5)</b>	Results	102
<b>Chapter (6)</b>	Discussion	130
<b>Chapter (8)</b>	Summary and Conclusion	144
<b>Chapter (9)</b>	Recommendation	146
	References	148
	Arabic Summary	



# **ABSTRACT**

## **Background:**

Liver steatosis is a common finding in patients infected with hepatitis C virus (HCV). Host and viral factors have been associated with steatosis, but their relative contributions have not been clearly addressed. It has been suggested that steatosis plays a role in the progression of liver fibrosis.

## **Aim:**

The aim of this study is to assess Steatosis in chronic hepatitis C patients in Egypt, with re-evaluation of viral genotyping as an independent risk factor.

## **Patients and methods:**

This study was conducted on fifty cases of chronic HCV hepatitis genotype (4). They were clinically assessed and investigated (Laboratory including complete blood count, liver biochemical profile, lipid profile, fasting blood glucose. imaging by abdominal ultrasonography and histopathologically according to modified Knodell score).

Body mass index was assessed. Logistic regression and multivariate analysis were used to identify variables independently associated with steatosis.

## **Results:**

The frequency of hepatic steatosis was 54 %. The univariate analysis revealed that steatosis was significantly associated with elevated bilirubin, high serum triglycerides, low serum albumin level, high FBS high necroinflammatory score and high fibrosis stage.

## **Conclusion:**

Hepatic steatosis is common in patients with chronic HCV hepatitis. Prevalence of steatosis is higher with HCV genotype 4. Factors associated with hepatic steatosis are serum TGs, stage of fibrosis, necroinflammatory score. It was not related to HCV RNA level and might be due to associated non-alcoholic steatosis or steatohepatitis.

## **Key Words:**

Liver steatosis

Hepatitis C patients

Genotype (4)

## **INTRODUCTION**

Hepatitis C virus (HCV) is a major cause of chronic liver disease with approximately 3% of the world's population (170 million people) infected worldwide. Over 85% of the world's hepatitis C viruses (HCV)-infected subjects exist in regions of Africa, Southeast Asia and Middle Eastern countries. **(Candotti D.et al., 2003)**

HCV genotype 4 is highly prevalent in Egypt with no accurate number of prevalence of the infection. Overall prevalence of antibody to HCV in the general population is around 15-20%. Chronic HCV representing one of the top five leading causes of death. **(Kamal SM.et al., 2005)**

The spectrum of severity of the liver disease associated with HCV varies widely from nonspecific, minimal inflammatory changes to cirrhosis and hepatocellular carcinoma. The rate of progression of chronic hepatitis C is also variable, depending on many cofactors, mostly host-related, such as age, gender, alcohol consumption, overweightness and co-infections. **(Nicot F.et al., 2005)**

Liver biopsy provides the most accurate information on the stage of fibrosis and grade of necroinflammation. The value of liver biopsy in predicting treatment response is incompletely defined and the relation of pre-treatment liver biopsy findings to standard interferon (IFN) and ribavirin (RBV) treatment outcomes is heterogeneous. **(Ikeda M.et al., 2006)**

## ***Introduction***

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Impaired secretion of lipids from the infected hepatocyte has been the first proposed mechanism of HCV-induced Steatosis. In fact, serum levels of apolipoprotein B (ApoB) and cholesterol are reduced in chronic hepatitis C patients in whom Steatosis responds to antiviral therapy. (**Liu C J.et al., 2005**)

Liver Steatosis (LS) has been variably associated with chronic hepatitis C (CHC) but whether it affects sustained virological response to antiviral treatment and by what mechanisms is a question still under debate, at least for some genotypes. (**Marcello P.et al., 2007**)

***Aim of the work***

The aim of this study is to study prevalence and level of Steatosis in chronic hepatitis C within our cohort of patients, with re-evaluation of viral genotyping as an independent risk factor.

## **Historical review of HCV**

Viral hepatitis is almost as old as human beings, at least as old as known human history, however, viruses as distinct biological entities have been known for little more than a century. Consequently, efforts to understand and control these important agents of disease are phenomena of the 20<sup>th</sup> century. Nevertheless, evidence of viral infection can be found among the earliest recordings of human activity, and methods for combating viral disease were practiced long before the first virus was recognized (Oldstone et al., 1998 and Szabo et al., 2003).

Reconstruction of the prehistoric past to provide a plausible account of when or how viruses established themselves in human populations is a challenging task. However, extrapolating from current knowledge, we can deduce that some modern viruses undoubtedly were associated with the earliest precursors of mammals and co-evolved with populations. Others entered human populations only recently. It is instructive to consider the last 10,000 years of human development, a time of radical change for humans and viruses; animals were domesticated, the humans' population increased dramatically, large population centers appeared and commerce drove interactions among unprecedented numbers of humans. We can infer from scattered glimpses of

ancient history that viruses have long been a part of human experience, and evidence of several viral diseases can be found in ancient records. The Greek Poet Homer characterizes Hecto, as “rabid” in the Iliad. Mesopotamian laws describing the responsibilities of the owners of rabid dogs date from before 1000 B.C. Egyptian hieroglyphs illustrating what appear to be the consequences of poliovirus infection or pustular lesions characteristic of smallpox also date from that period (**Brothwell and Sandison et al., 1967**).

In **1892** the **Russian Dimitrii Ivanowsky** gave the first report on a discovery of a pathogenic agent smaller than any known bacterium and 6 years later the Dutsch **Martinus Beijernick** did the same. In the same year (**1898**) the German Scientists **Friedrich Loeffler** and **Paul Frosch** observed that the causative agent of foot and mouth disease was also not retained by the porcelain filters used at that time to remove bacteria. However, the first identification of a human virus was done by **Reed and Carroll** in **1901** when they reported that a filterable virus is the cause of yellow fever (**Hughes et al., 1977**).

The terms hepatitis A and B were first used in 1947, however, proof that viruses are responsible for this disease was first published in **1968** with the description of hepatitis B virus particles in nature (**Havens et al., 1947, and Bayer et al., 1968**).