

# **SEROPREVALENCE OF HEPATITIS C VIRUS AMONG RURAL AND URBAN PREGNANT WOMEN**

## **THESIS**

Submitted in Partial Fulfilment for Requirement of the  
Master Degree in Obstetrics Gynecology

**BY**

***Mona El-Sayd Mohamed Hassan***

M.B.B.Ch.

Mansoura University, 1984

## **SUPERVISED BY**

***Professor Dr.***

***Mohamed Nagi El-Makhzangy***

Professor of Obstetrics and Gynecology  
Faculty of Medicine - Ain Shams University

***Dr.***

***Mohamed Ali Mohamed Ibrahim***

Assistant Professor of Obstetrics and Gynecology  
Faculty of Medicine - Ain Shams University.

***Professor Dr.***

***Farha Abdul Aziz El-Shennawy***

Professor of Clinical Pathology  
Faculty of Medicine - Mansoura University

Faculty of Medicine  
Ain Shams University  
1996



قالوا سبحنك لا علم لنا الا ما علمتنا انك انت العليم الحكيم  
صدق الله العظيم

سوره البقره

آيه ٣٢



### ACKNOWLEDGEMENT

I wish to express my sincere thanks to all those who made the completion of this work possible .

I am grateful to professor Mohamed Nagi El Maklizangy, professor of Obstetrics and Gynecology, Ain Shams University for his invaluable advise and constant supervision

I would like to express my sincere gratitude to Dr. Mohamed Ali Mohamed Ibrahim, assistant professor of Obstetrics and Gynecology, Ain Shams University for his great help and guidance

I would like to thank professor Farha Abdulaziz El Shennawy, professor of Clinical Pathology , Mansura University for her continued supervision and encouragement

---

# TO MY FATHER

## **LIST OF CONTENTS**

<u>Subject</u>	<u>Page</u>
Introduction and Aim of the work	1-2
Part I- Review of the literature	3-69
1- Viral hepatitis	3-17
• Historical aspect	3
• Pathology	3-5
• Clinical features	6-11
• Sequelae	11-15
• Investigation	16-17
2- Hepatitis A virus	18-21
3- Hepatitis B virus	22-28
4- Hepatitis D virus	29-32
5- Hepatitis E virus	33-35
6- Hepatitis C virus	36-63
7- Pregnancy and viral hepatitis	64-69
Part II : Patients and methods	70-77
Part III: Results	78-112
Part IV: Dissussion	113-123
Part V :Summary and conclusion	124-125
Part VI : References	126-159
Part VII : Arabic summary	1-2

---

## LIST OF TABLES AND FIGURES

### Figures

<u>Figure No</u>		<u>Page</u>
1	Diagram of the hepatitis A virus	19
2	Diagram of the hepatitis B virus	23
3	Diagram of the hepatitis D virus	30
4	Diagram of the hepatitis E virus	34
5	Diagram of the hepatitis C virus	37
6	Odds ratios of different HCV risk factors in all cases	108
7	Odds ratios of different HCV risk factors in rural area	109
8	Odds ratios of different HCV risk factors in urban area	110

### Tables

<u>Table No</u>		<u>Page</u>
1	Relationship between HCV antibody test reactivity and residency in all tested women	79
2	HCV antibody test reactivity in pregnant versus non pregnant women	80
3	Relationship between HCV antibody test reactivity and gravidity among all cases	81
4	Relationship between HCV antibody test reactivity and blood transfusion among all cases	82
5	Relationship between HCV antibody test reactivity and Ob./Gyn. surgery in all cases	83
6	Relationship between HCV antibody test reactivity and previous general surgery in all cases	84
7	Relationship between HCV antibody test reactivity and history suggestive of hepatitis among all cases	85
8	Relationship between HCV antibody test	86

---

pregnant women in rural area	
20 Relationship between HCV antibody test reactivity and frequent intravenous ( I.V.) drug use among pregnant women in urban area	98
21 Results of logistic regression of prevalence of HCV antibody among all pregnant women	99
22 Results of logistic regression of prevalence of HCV antibody ( all variable in rural area )	100
23 Results of logistic regression of prevalence of HCV antibody ( all variable in urban area )	101
24 Percent of grouped cases correctly predicted for all cases	102
25 Percent of grouped cases correctly predicted in rural area	103
26 Percent of grouped cases correctly predicted in urban area	104
27 Standerdized canonical discriminant function coefficients disriminating HCV infection	105
28 Unstanderdized canonical discriminant function coefficients disriminating HCV infection	106
29 Percent of grouped cases correctly classified from the previous discriminant analysis	107

---

	reactivity and intravenous ( I.V) drug use among all cases	
9	Relationship between HCV antibody test reactivity and gravidity among pregnant women in rural area	87
10	Relationship between HCV antibody test reactivity and gravidity among pregnant women in rural area	88
11	Relationship between HCV antibody test reactivity and previous history of blood transfusion among pregnan t women in rural area	89
12	Relationship between HCV antibody test reactivity and previous history of blood transfusion among pregnant women in urban area	90
13	Relationship between HCV antibody test reactivity and previous OB./ Gyn. surgery among pregnant women in rural area.	91
14	Relationship between HCV antibody test and previous Ob./Gyn. surgery pregnant women in urban area	92
15	Relationship between HCV antibody test reactivity and previous general surgery among pregnant women in rual area	93
16	Relationship between HCV antibody test reactivity and previous general surgery among pregnant women in urban area	94
17	Relationship between HCV antibody test reactivity and past history suggestive of hepatitis among pregnant women in rural area	95
18	Relationship between HCV antibody test reactivity and past history suggestive of hepatitis among pregnant women in urban area	96
19	Relationship between HCV antibody test reactivity and frequent intravenous ( I.V.) drug use among	97

**Introduction  
&  
Aim of the Work**

## *Introduction* *and Aim of the Work*

A viral agent that is neither hepatitis A virus nor hepatitis B virus has been recognized as the major cause of community acquired viral hepatitis (Stevens, et al , 1990). Recently the genome of non-A , non-B hepatitis designated hepatitis C virus( HCV ) was molecularly cloned and identified as a positive strand RNA molecule ( Kuo et al , 1989 ).

An assay for circulating viral antibodies to HCV was developed using an antigen purified from recombinant yeast clones derived from the genome and result obtained with this assay suggested that antibodies to HCV could be used as a marker of HCV infection ( Stevens, et al , 1990 )

Since that time, the assay has become commercially available and published reports have described anti- HCV antibody seroprevalences in a variety of populations at both high and low risk for infection , most studies using the epidemiologic characteristics of hepatitis B virus infection as their model for risk predictions.

As hepatitis B virus “ HBV “ materno - fetal transmission was described in the pre-natal period and measures for immunoprophylaxis of the at - risk newborn proved effective

( Beasley, et al , 1983 ), concerns have been raised regarding the HCV vertical transmission ( Thaler , 1991 ) .

The aim of the work is to contribute in delighting the prevalence of HCV among both urban and rural pregnant women in some localities in Egypt ,as large - scale evaluations of the magnitude of vertical transmissibility as a public health issue will eventually depend on studies first establishing the actual risk of such occurrences on the basis of seroprevalence data in the population as a whole . We also, examined the value of risk factors identification for predicting HCV antibody positivity among pregnant women.

# Review of Literature

## **Viral hepatitis**

### **Historical Aspect :**

The first reference to epidemic jaundice has been ascribed to Hippocrates. The earliest record in Western Europe in a letter written in 751 AD by Pope Zacharias to St. Boniface, Archbishop of Mainz. Since then there have been numerous accounts of epidemics, particularly during wars. Hepatitis was a problem in the Franco- Prussian War, the American Civil War and World War I. In World War II huge epidemics occurred, particularly in the Middle East and Italy (Zuckerman, 1977).

In the last few years, important research developments have clarified the molecular biology, diagnosis, epidemiology and clinical features of five distinct hepatotropic viruses, that is A, B, C, D, and E (Gregorio et al, 1994).

### **Pathology :**

#### **I- Hepatic changes:**

All forms of viral hepatitis have basic pathology. The essential lesion is an acute inflammation of the entire liver (Dible et al, 1943).

The typical morphologic lesions of hepatitis A, B, C, D and E are often similar and consist of panlobular infiltration with mononuclear cells, hepatic cell necrosis, hyperplasia of Kupffer cells, and variable degrees of cholestasis.

Hepatic cell regeneration is present, as evidenced by numerous mitotic figures, multinucleated cells, and "rosette" or "pseudoacinar" formation. The mononuclear infiltration consists primarily of small lymphocytes although plasma cells and eosinophils are occasionally seen. Liver cell damage

consists of hepatic cell degeneration and necrosis, ballooning of cells, and acidophilic degeneration of hepatocytes forming so-called (Councilman-like bodies). Large hepatocytes with a ground glass appearance of the cytoplasm may be seen in chronic but not in acute hepatitis B virus (HBV) infection. These cells have been shown to contain HB<sub>s</sub>Ag and can be identified histochemically with orcein or aldehyde fuchsin. In uncomplicated viral hepatitis, the reticulin framework is preserved in hepatitis C, the histologic lesion is often remarkable for a relative paucity of inflammation a marked increase in activation of sinusoidal lining cells, the presence of fat, and occasionally, bile duct lesions in which biliary epithelial cells appear to be piled up without interruption of the basement membrane. Occasionally microvesicular steatosis occurs in hepatitis D. In hepatitis E, a common histologic feature is marked cholestasis. A cholestatic variant of slowly resolving acute hepatitis A also has been described. (Dienstag, and Isselbacher, 1994).

**Confluent bridging hepatic necrosis: (Koff, 1993)**

This term was suggested by an international group of hepatologists (Bianchi, et al, 1971) to designate zonal necrosis affecting extensive groups of adjacent hepatocytes in contrast to the usual spotty, focal, random distribution of necrosis in typical viral hepatitis. When hepatocellular necrosis or a necroinflammatory process involves contiguous groups of hepatocytes that connect recognizable anatomic structures this confluent lesion appears to bridge these structures and the term bridging necrosis is often used.

**Patterns of bridging:** the importance of bridging necrosis depends in part on the location as well as the extent of the injury.

**A-Portal to portal bridging:** preportal hepatitis increase the chance that random sections across hepatic lobules produce apparent confluence of adjacent portal tracts. The meaning of