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



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سوره البقرا

آله ۲۲



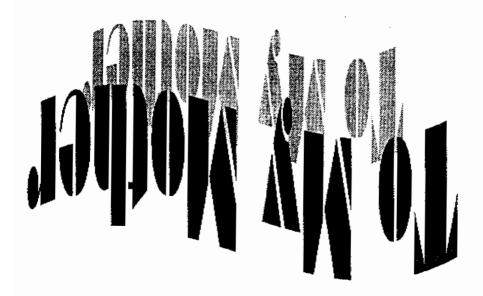
ACKNOWLEDGEMENT

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 Ei Makhzangy, 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



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Introduction Introduction Sim of the Work

<u>Introduction</u> 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 acquird viral hepatitis (stevens, et al., 1990). Recently the genome of non-A, non-B hepatitis designated hepatitis C virus(HCV) was moleculary 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 antibode 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 describid 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 evalutions of the magnitude of vertical transmissibility as a public health issue will evantually depend on studies first establishing the actual risk of such occurences on the basis of seroprevalence data in the population as a whole. We also, examined the value of risk factors identification for predicting HCV anti-body positivity among pregnant women.

I iterateure Ot Mainterateure

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 accasionally seen. Liver cell damage

consists of hepatic cell degenration and necrosis, ballooning of cells, and acidophilic degeneration of hepatocytes forming socalled (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, Ag and can be identified histochemically with orcein or aldehyde fuchsin. In viral hepatitis, the reticulin framwork is uncomplicated 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 Isselbacker, 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 adjecent portal tracts. The meaning of