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شبكة المعلومات الجامعية



شبكة المعلومات الجامعية

التوثيق الالكتروني والميكرو فيلم

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التوثيق الالكتروني والميكرو فيلم

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2000

17/7/00

IMMUNE COMPLEXES STUDY IN SOME CHRONIC VIRAL LIVER DISEASES

Thesis

Submitted to the Faculty of Medicine
Tanta University
In partial fulfilment of the
Requirements of the M.D degree of
Tropical Medicine and Hygiene

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*To my Parents
who prayed for my success,
To my Husband and
my lovely daughter*



Acknowledgement

Thanks to **ALLAH**, greater of all, for giving me the power & courage to undertake and achieve this work.

I would like to express my deep appreciation and sincere thanks to **Prof. El-Sayed Ahmed Abd El-Halim Wasfy**, Professor and Head of the Dept. of Tropical Medicine, Faculty of Medicine, Tanta University, for his valuable assistance, continuous help, unending cooperation and efforts, I am specially indebted to his valuable efforts in supervising, planning and revising the whole work and for which I will remain always grateful.

I am greatly indebted to **Prof. Mohamed Abd El-Hamid**, Professor and Head of Pharmacology Department, Faculty of Medicine, Tanta University, who gave generously his ideas and time in planning this work, assistance and devotion in the supervision of all parts of this work are appreciated. I am greatly indebted for his continuous encouragement.

I am delighted to express my deep gratitude to **Prof. Saad El-Din Abd El-Fattah Abou El-noeman**, Prof of Medical Biochemistry, Faculty of Medicine, Tanta University for his brilliant suggestion of the topic, planning & direction of the thesis, fruitful ideas unmatched guidance, constrict criticism, continous encouragement, valuable advice and sincere help. It was a great honour to work under his supervision.

No words can adequately express my sincere gratitude and great appreciation to **Prof. Atef El-Sayed Awad**, Ass. Prof. of Tropical Medicine, Faculty of Medicine, Tanta University for his patience, marvelous efforts, endless help, unlimited cooperation, kind support, valuable guidance precious suggestion, supervision throughout the whole thesis.

*My deepest gratitude to **Dr. Mohamed Zakaria**, Lecturer of Bacteriology, Faculty of Medicine, Tanta University for his guidance, supervision and valuable advice during the course of this study.*

*My thanks and gratitude to the **Staff members** of Tropical Medicine for their support and encouragement.*

*I would like to thank **all the patients** included in this work for their contribution.*

*Finally, thanks also must go to the **Staff of Scientific Service Center** for the good effort in writing the thesis and preparing presentation slides.*

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INTRODUCTION

INTRODUCTION

Viral hepatitis

Virus hepatitis is a common and serious infectious disease caused by several viral agents and marked by necrosis, fatty changes and lympholytic infiltration of the portal zone. The spectrum of chronic inflammatory disease of the liver extends from acute hepatitis to chronic hepatitis and finally to cirrhosis. The same basic underlying liver histology is seen whatever the etiology (Sbabid et al., 1995).

Viral hepatitis may result from the following viruses:

A. Hepatotropic viruses:

Liver is mainly affected but other organs may be involved; these include hepatitis viruses A,B,C,D,E,F and G.

B. Non-hepatotropic viruses:

The liver is involved as a part of the disease. These include: Epstein-Barr virus (EBV), Cytomegalovirus (CMV), herpes simplex type 1, varicella zoster virus, measles, coxsackie-B viruses (Sbabid et al., 1995).

C. Exotic viruses:

These very dangerous newly identified and unusual viruses have the liver as the primary target. They include Marburg, Lassa and Ebola viruses. They are becoming increasingly important as man encroaches into underdeveloped areas, as ecology changes and as a source of infection to medical or laboratory staff dealing with patients or their blood (Howard 1984). While these agents can cause diagnostic confusion by producing some degree of liver inflammation and dysfunction, they are not considered to be a primary cause of acute or chronic hepatitis (Mc-Cormick et al., 1986).

A. Hepatotropic virus**I. Hepatitis A virus:**

It accounts for 20-25% of clinical hepatitis in the developed world, it is due to a small 27 nm cubically symmetrical RNA of Picorna viridae family. It is single-stranded, transmitted faecally, self-limited with no chronicity or liver cirrhosis (Feinstone, 1973). A serum antibody (Anti-HAV) appears, as the stool becomes negative for virus, reaches a maximum in several months and is detectable for many years. IgG anti-HAV probably gives immunity from further infection with hepatitis A (Glickson et al., 1992). The appearance of serum IgM anti-HAV is more helpful diagnostically and implies a recent infection.

This antibody persists for only 2-6 months (Mishu et al., 1990). The virus is not directly cytopathic and liver cell damage is not caused by viral replication but by T-cell mediated immune responses to infection (Battegay et al., 1995).

II. Hepatitis B Virus:

This is one of the commonest and most widespread infections of the human beings. It is classified as a DNA viruses of the family Hepadna viridae (Lau and Wright, 1973). The virion of hepatitis B consists of a surface and core. The core is formed ⁱⁿ of hepatocyte nucleus and contains a DNA polymerase. The surface particles are made in the cytoplasm. The core contains a core antigen and another antigen called e' which is a protein subunit of the core. It is parenterally transmitted disease that often becomes chronic (Miller and Robinson 1986).

a. Serological diagnosis:

HBs Ag appears in the blood about six weeks after infection and disappears by three months. Persistence for more than six months implies a carrier state. Anti-HBs appears late, some three months after the onset and persists. Anti-HBs levels are rarely high and 10-15% of patients with acute type B hepatitis never develop the antibody. Anti-HBs accounts for recovery and immunity. (Kaneko et al., 1990).

b. Clinical course

The course may be anicteric. The non-icteric case is more liable to become chronic than the icteric one. Jaundice rarely exceeds four weeks. Occasionally, a prolonged benign course is marked by increased serum transaminase value for more than 100 days. Relapses are rare. Cholestatic hepatitis with prolonged deep jaundice is unusual (Brecht et al., 1984).

c. Prevention

Hepatitis B immunoglobulin (HBIG) is a special hyperimmune serum globulin with a high antibody titre. It is effective for passive immunization against hepatitis B if given prophylactically or within hours of infection. Hepatitis vaccine should always be given with HBIG, particularly if the subject is at risk of re-infection (Seeff and Koff 1984).

III. Hepatitis D virus:

This is a very small RNA virus of viroid family. It is parenterally spread and affects only those with hepatitis B infection. It is coated with HBsAg and is unable to replicate on its own but is capable of infection when activated by the presence of HBV. (Aragona et al., 1987).