







شبكة المعلومـــات الجامعية التوثيق الالكتروني والميكروفيا.



جامعة عين شمس

التوثيق الالكتروني والميكروفيلم



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STUDY OF

BISIN

INTERLEUKIN - 8 - AND GRANULOCYTE

MACROPHAGE - COLONY STIMULATING FACTOR IN

CHRONIC HEPATITIS B & C VIRAL INFECTION

Thesis

Submitted in partial fulfillment for Master degrée in internal medicine

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LIST OF ABBREVIATIONS

AIDS Acquired immune defeciency syndrome.

ALT Alanine transaminase

APCs Antigen presenting cells.

AST Aspartate transaminase.

CAH Chronic active hepatitis.

CPH Chronic persistant hepatitis.

DHBV Duck hepatitis B virus.

DNA Deoxy ribonucleic acid.

DNA-P Deoxy ribonucleic acid - polymerase

ELISA Enzyme linked immunosorbent assay

GM-CSF Granulocyte macrophage - colony stimulating factor

GSHV Ground squirrel hepatitis virus

HAV Hepatitis A virus

HBcAg Hepatitis B core antigen.

HBcAb Hepatitis B core antibody

HBeAg Hepatitis B'envelope antigen

HBeAb Hepatitis B enevelope antibody

HBsAg Hepatitis B, surface antigen.

HBsAb Hepatitis B surface antibody.

HBV Hepatitis B virus.

HBV. DNA Hepatitis B virus deoxy ribonucleic acid.

HCC Hepatocellular carcinoma.

HCV Hepatitis C virus.

HCV ab 🧳 🌼 Hepatitis C-virus antibody.

HDV Hepatitis delta virus.

HEV Hepatitis E virus.

HFV Hepatitis F virus.

HGV Hepatitis G virus.

IFN Interferone

IgG Immunoglobulin G

IgM Immunoglobulin M

IL Interleukin

LCI Liver cell inflammation

MHC Major histocompatibility complex.

NK cells Natural Killer cells.

ORF Open reading frame.

PCR Polymerase chain reaction.

RNA Ribonucleic acid

RIBA Recombinant immunoblot assay.

SGOT Serum glutamic oxaloacetic transaminase.

SGPT Serum glutamic Pyruvic transaminase.

SD Standard deviation.

T.C T.cytotoxic.

T.H T. helper.

TNF- α Tumour necrosis factor alpha.

WHV Woodchuck hepatitis virus.

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MTRODUCTION & AIM OF THE WORK

INTRODUCTION & AIM OF THE WORK

Viral hepatitis is a major public health problem in all parts of the world specially in tropical and subtropical areas (Gust and Crowe, 1986).

Viral hepatitis is caused by infection by one of several viruses which produce varying degrees of hepatic inflammation and necrosis (Weisiger, 1989). At least five different types of hepatic viruses can cause hepatitis: A, B,C,D, and E in addition to less common viruses cytomegalovirus. Ebstien Barr virus and herpes simplex virus (Knauer and Cichael, 1993).

Hepatitis B is a parenterally transmitted disease that often becomes chronic. Hepatitis D is parenterally spread and affects only those with hepatitis B infection. Hepatitis C is a parenterally spread disease with a high chronicity rate. There will undoubtedly be other members of the hepatitis alphabet (Sherlock and Dooley, 1993).

The parenterally transmitted B,C,D and G viruses can progress into chronic infection, cirrhosis or hepatocellular carcinoma "HCC" (Kabil et al., 1991).

Chronic viral hepatitis is defined as chronic inflammatory reaction in the liver continuing without improvement for at least six months (Scheuer, 1933).

Chronic hepatitis B is not usually preceded by recognizable acute attack of hepatitis B, however in some, the acute episode progress directly

into chronicity, in others, although apparent as an acute illness chronic hepatitis already exist (Sherlock and Dooley, 1993).

Nowadays hepatitis C is much more important problem in Egypt than hepatitis B. Hepatitis B is on the way out with better hygiene, vaccines, all B is going but C is coming. The carrier rate of HCV in Egypt is increasing. The patients has about a 60% chance to develop chronic disease (Sherlock and Dooley, 1993).

Cytokines are polypeptides that possess a wide spectrum of inflammatory, metabolic, haematopoietic and immunologic regulatory properties (Arai et al., 1990).

Interleukin-8 (IL-8) and granulocyte- macrophage colony stimulating factor (GM -CSF) are important mediators of inflammation and immune response in human disease (Al - Wabil et al., 1995).

Our aim in this study is to determine whether IL-8 and GM-CSF are elevated in patients with chronic hepatitis B and C viral infection and their importance in pathophysiological processes in these patients.



VIRAL HEPATITIS

It is a disease of major public health significance all over the world not only in terms of over all morbidity but also in terms of economic consequences (Mc Quilan, et al.,1989)

Viral hepatitis caused by a wide range of hepatotropic viruses including hepatitis A virus (HAV), hepatitis B virus (HBV), hepatitis C virus (HCV), hepatitis D (delta) virus (HDV), hepatitis E virus (HEV), (Chopra et al., 1994)

Hepatitis A virus (HAV): Spread by the faecal -oral route causing self limiting acute disease with low mortality and no progression to chronic disease (Forbes and Williams 1990)

Hepatitis B virus (HBV): Transmitted parenterally and by other non-faecal-oral routes, associated with chronic hepatitis, cirrhosis and eventually hepatocellular carcinoma (Papaevangelou 1987)

Hepatitis C virus (HCV): Transmitted parenterally and by other non-faecal-oral routes, also associated with chronic liver disease and hepatocellular carcinoma (Houghton et al., 1991).

Hepatitis D (Delta) virus (HDV): Superinfects patients with HBV infection since it requires HBV for its own replication. It may lead to exacerbation of hepatitis. A patient is rarely infected with HBV and HDV at the same time (Co-infection) (Monjardino and saldanho, 1990).