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بالرسالة صفحات
لم ترد بالأصل

PREVALENCE OF HEPATITIS C VIRUS ANTIBODIES AMONG DIALYSED PATIENTS AND STAFF IN SOME DIALYSIS UNITS IN ALEXANDRIA

Thesis

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By

Ehab Mohamed Amin Ahmed
MBBCh Alex

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SUPERVISORS

Prof. Dr. Atef Ahmed Hozain

Professor of Internal Medicine,
Chairman of Nephrology Department,
Faculty of Medicine,
University of Alexandria.

Prof. Dr. Salah Said Naga

Professor of Internal Medicine,
Faculty of Medicine,
University of Alexandria.

Dr. Nagwa Gamil Rizk

Lecturer of Microbiology,
Faculty of Medicine,
University of Alexandria.

بسم الله الرحمن الرحيم

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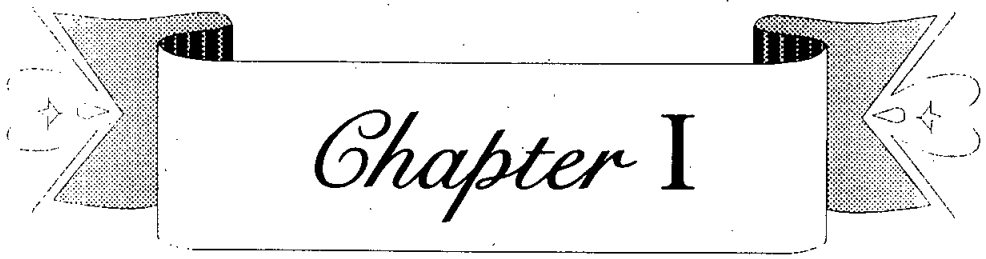
CONTENTS

To My Family



Chapter I

INTRODUCTION

INTRODUCTION

The term hepatitis is applied to a broad category of clinicopathological conditions that result from the damage produced by viral, toxic, pharmacologic or immune-mediated attack upon the liver.[1]

Clinically, the liver may be enlarged, tender with or without jaundice and laboratory evidence of hepatocellular damage is invariably found in the form of elevated aminotransferase levels (alanine aminotransferase, or ALT and aspartate aminotransferase, or AST).[1]

Independent of its cause, the clinical course of hepatitis may range from mild or asymptomatic to a dramatic illness with evidence of severe hepatocellular dysfunction, marked jaundice, impairment of coagulation and disturbance of neurologic functions.[1]

On the basis of clinical and pathologic criteria, hepatitis is further divided into acute and chronic types.

Acute hepatitis implies a condition lasting less than 6 months, culminating either in complete resolution of the liver damage with return to

normal liver function and structure or in rapid progression of the acute injury toward extensive necrosis and a fatal outcome.[1]

Chronic hepatitis is defined as a sustained inflammatory process in the liver lasting longer than 6 months. The histological and clinical picture may be that of a relatively benign process, chronic persistent hepatitis, or that of a process which usually leads to cirrhosis or chronic active hepatitis.[1]

In these years, more light has been brought into the field of hepatitis viruses, particularly with regard to their biology and etiopathogenesis. Besides, first attempts of specific therapy have been done.

Involvement of hepatic parenchyma by viral agents may be primary or secondary in the course of general disease. Among viruses with the liver as primary localization, there must not be forgotten non-A, non-B viruses which, even after the C virus discovery, represent 20% of post-transfusional and 30-50% of sporadic hepatitis.[2]

Hepatitis A

Virology :

Hepatitis A virus (HAV) is a small 27 nm picornaviurs. Its virion is composed of four polypeptides (VP1-VP4) which forms a tight protein shell, or capsid, containing the RNA.

All strains of this virus identified to date are immunologically indistinguishable and belong to one serotype. Unlike other hepatitis viruses, hepatitis A virus can be grown readily in epithelial cell lines.[3] Such characteristics favour the preparation of vaccines.

Epidemiology :

The agent is transmitted almost exclusively by the fecal-oral route. So spread of HAV is enhanced by poor personal hygiene and overcrowding. Large outbreaks as well as sporadic cases have been traced to contaminated food, water, milk and shell fish. In developed countries, the incidence of type A hepatitis has been declining.[4]

Clinical course :

The incubation period of HAV is short, 2-3 weeks. Hepatitis A is mild or subclinical in children. In adults, the disease is often more serious and prolonged, relapses occasionally occur. Fulminant hepatitis is rare. No HAV carrier state has been identified and chronicity has been only recently reported.[5]

Diagnosis :

Diagnosis is made by detection of anti-HAV IgM during the acute illness. Anti-HAV IgM persists for several months (2-6 months). During convalescence, anti-HAV IgG becomes the predominant and persists for years and gives immunity.[6]

Prevention :

Immunoglobulin should be given to persons without antibodies (for contacts and visitors travelling to endemic areas). Vaccine will be soon commercially available.[6]

Pathogenesis of HAV hepatitis :

A direct cytopathic effect on the hepatocytes has been suggested. However, HAV induces persistent infection in vitro without cytopathic