بسم الله الرحمن الرحيم

شبكة المعلومـــــات الجامعية

التوثيق الالكتروني والميكروفيلم

شبكة المعلومات الجامعية

جامعة عين شمس

التوثيق الالكتروني والميكروفيلم

﴿ قـــســـم ﴾

نقسم بالله العظيم أن المادة التي تم توثيقها وتسجيلها
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يجب أن

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To be kept away from dust in dry cool place of
15 – 25c and relative humidity 20-40 %

بعض الوثائق الأصلية تالفة

بالرسالة صفحات

لم ترد بالأصـــل

HEPATITIS E VIRUS INFECTION AMONG CASES OF ACUTE VIRAL HEPATITIS IN ALEXANDRIA (EGYPT)

Thesis

Submitted to the High Institute of Public Health
University of Alexandria
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By

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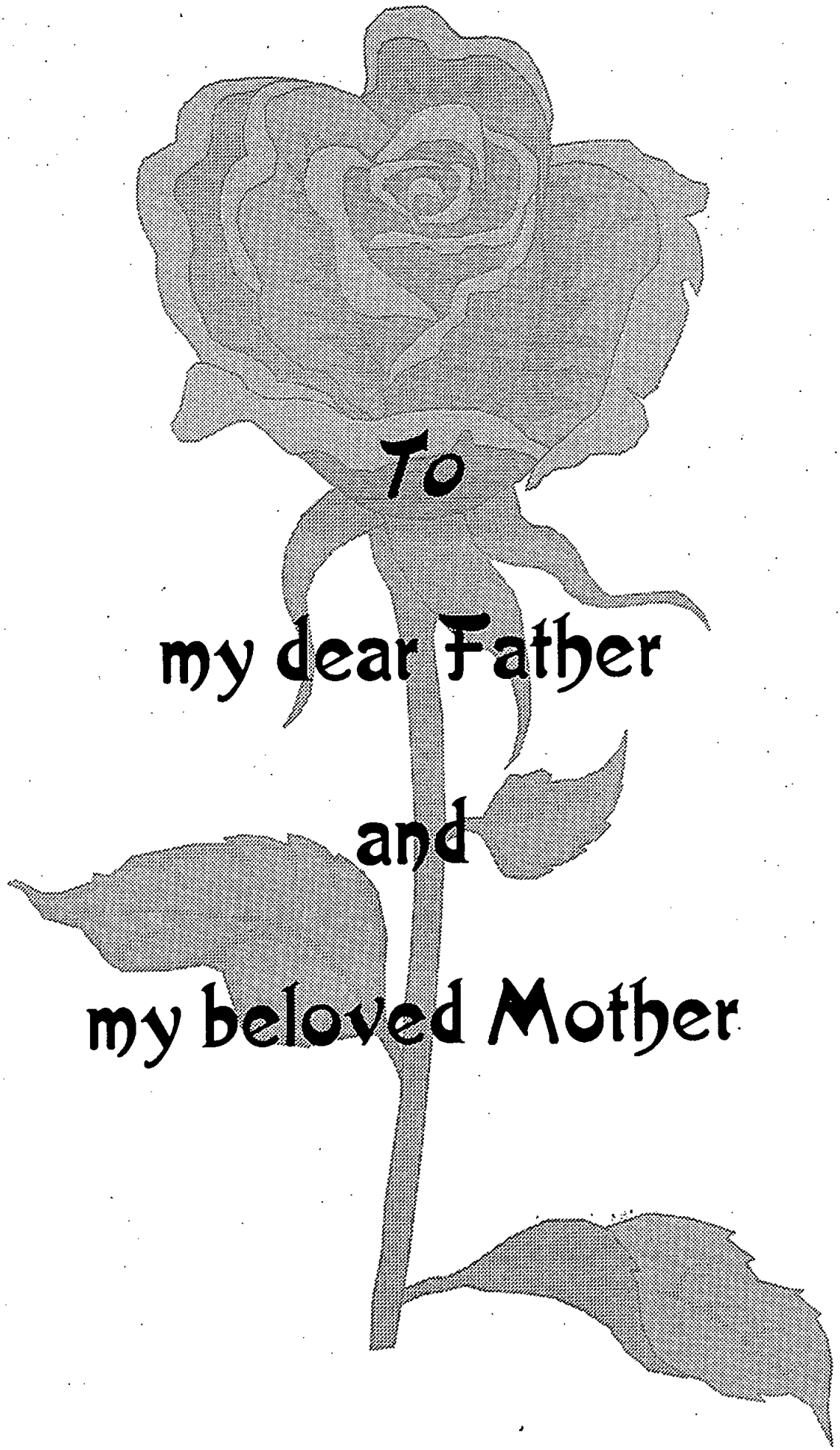

To

my dear Father

and

my beloved Mother

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CONTENTS

LIST OF ABBREVIATIONS

ALT	Alanine aminotransferase
Anti-HAV	Antibodies against hepatitis A virus
Anti-HBc	Antibodies against hepatitis B virus core antigen
Anti-Hbe	Antibodies against hepatitis B virus e antigen
Anti-HEV	Antibodies against hepatitis E virus
Anti-hIgM	Anti-human immunoglobulin M
COV	Cut off value
ELISA	Enzyme linked immunosorbant assay
EM	Electron microscope
ET-NANB	Enterically transmitted non-A, non-B
FITC	Fluorescein isothiocyanate
GBV-C	GB virus C
HAV	Hepatitis A virus
HBV	Hepatitis B virus
HbeAg	Hepatitis B e antigen
HbsAg	Hepatitis B core antigen
HDV	Hepatitis D virus
HCV	Hepatitis C virus
HDV	Hepatitis D virus
HEV	Hepatitis E virus
HFV	Hepatitis F virus
HGV	Hepatitis G virus
HRP	Horse radish peroxidase
IEM	Immunoelectron microscope
NC	Negative control
OD	Optic density
OPD	O-Phenylenediamine
ORF	Open reading frame
PC	Positive coltrol
RT-PCR	Reverse transcription-polymerase chain reaction
TMB	Tetramethylbenzidine
VLPs	Virus like particles

INTRODUCTION

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Six major viruses are the etiologic agents responsible for most clinical cases of viral hepatitis: hepatitis A virus (HAV), hepatitis B virus (HBV), hepatitis C virus (HCV), hepatitis D virus (HDV), hepatitis E virus (HEV), and hepatitis G virus (HGV). Deka and colleagues [1] have described what they designate hepatitis F virus (HFV).

Acute and chronic necro-inflammatory liver disease caused by hepatotropic virus infection continues to be a major worldwide health problem. Approximately 5% of the world's population has chronic hepatitis B virus (HBV) [2] as many as 100% of the residents of nonindustrial nations and 70% of the residents of industrial nations have serologic evidence of prior exposure to hepatitis A virus (HAV), [3] and in the United States, end-stage liver disease caused by chronic hepatitis C virus (HCV) is the most common indication for liver transplantation. [4]

History of viral hepatitis

Inflammation of the liver is not a new disease. Early documents such as the Ebers papyrus (1552 BC) mentioned liver ailments, as did the Babylonian Talmud (5th century BC). The epidemic nature of viral hepatitis was manifested in battlefields. For example, approximately 72,000 cases of jaundice were diagnosed among the Union Army soldiers fighting the American Civil War (1861-1865). Similarly, large epidemics of jaundice occurred during the Japan-Russia war (1904-1905) and during World Wars II and I. [5] During World War II, more than 50,000 American soldiers who received yellow fever vaccine developed hepatitis, and 62 died. [6] In a follow-up study of 597 service men, and using modern serologic methods, Seeff and coworkers [7] confirmed that HBV had been the cause of this outbreak.

In 1947, MacCallum [8] proposed the names hepatitis A and hepatitis B for what was known as infectious and serum hepatitis respectively. Blumberg and associates [9] reported the discovery of the Australia antigen in 1965; this antigen is now known as the hepatitis B surface antigen (HBsAg). This pivotal discovery allowed the serologic differentiation of HBV from other causes of viral hepatitis.

The identification of the hepatitis A viral particle by Feinstone and coworkers [10] accelerated the development of laboratory assays for an antibody to hepatitis A (anti-HAV). [11] The discovery of the delta agent reported by Rizzetto and colleagues [12] in 1977 allowed prompt development of diagnostic techniques for this agent. [13] Initially, exclusion of HAV and HBV infections was the basis of the diagnosis of HCV and HEV infections, then known as blood-borne (HCV) or water-borne (HEV) non-A, non-B hepatitis. In 1989, a group of scientists at the Chiron Laboratory in California reported the cloning of HCV [14] and developed an assay for the detection of an antibody to hepatitis C (anti-HCV). [15] HEV was first observed and reported in 1983 by Balayan and associates. [16] In 1990, Reyes and colleagues [17] reported the sequencing of HEV. Assays for the determination of an antibody to HEV (anti-HEV) have been developed and described. [18,19,20] In 1995, investigators from Abbott Laboratories published the sequencing of three HGB agents: A, B, and C, [21] and investigators at Genelabs Technologies in California discovered the HGV. [22] The HFV has been reported to be a double-stranded DNA virus, transmissible to rhesus monkeys by inoculation with stool extracts from infected patients. [1]