



شبكة المعلومات الجامعية

بِسْمِ اللَّهِ الرَّحْمَنِ الرَّحِيمِ





شبكة المعلومات الجامعية



شبكة المعلومات الجامعية

التوثيق الإلكتروني والميكروفيلم



شبكة المعلومات الجامعية

جامعة عين شمس

التوثيق الالكتروني والميكروفيلم



نقسم بللّاه العظيم أن المادة التي تم توثيقها وتسجيلها
علي هذه الأفلام قد اعدت دون أية تغييرات



يجب أن

تحفظ هذه الأفلام بعيداً عن الغبار

في درجة حرارة من 15 – 20 مئوية ورطوبة نسبية من 20-40 %

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



شبكة المعلومات الجامعية
@ ASUNET



بعض الوثائق الأصلية تالفة



شبكة المعلومات الجامعية
@ ASUNET



بالرسالة صفحات

لم ترد بالأصل

B.V.VI

**STUDY OF THE PREVALENCE OF
PORTAL VEIN THROMBOSIS IN
PATIENTS WITH HEPATOCELLULAR
CARCINOMA**

**Thesis
Submitted to Faculty of medicine
University of Alexandria
In Partial Fulfillment of the Requirements
Of the Degree of
Master of Internal Medicine**

**By
Amany Saleh Abd El Aziz El Yamany
MBBCh (Alex)**

**FACULTY OF MEDICINE
UNIVERSITY OF ALEXANDRIA
2002**

Supervisors

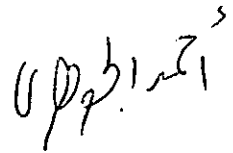
Prof. Dr. Ali Abdel Moety Soliman

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



Prof. Dr. Ahmed Mohamed El Gohary,

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



Prof. Dr. Nagla Moustafa Mashaal,

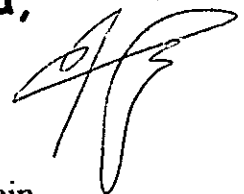
Assistant Professor of Internal Medicine,
Faculty of Medicine,
University of Alexandria



Sharing Supervisors

Prof. Dr. Mohamed Ihab Samy Reda,

Assistant Professor of Radiodiagnosis
Faculty of Medicine,
University of Alexandria



For his experience in Doppler examination of the portal vein.

ACKNOWLEDGMENT

ACKNOWLEDGMENT

Thanks to **Allah** that this research came to its end with his will and help.

It is my greatest pleasure to express my sincere gratitude and appreciation to Professor **Aly Abdel Moety Soliman**, Professor of Internal Medicine, for suggesting, supervising and guiding this work from the start to the end.

I am deeply indebted to Professor **Ahmed Mohamed El Gohary**, Professor of Internal Medicine, for his encouragement and helpful instructions throughout the work.

I am also more than delighted for the great support and continuous advice obtained from Professor **Mohamed Yousry Taher**, Professor of Internal Medicine and Head of Hepatobiliary Department.

I am very thankful to Dr. **Nagla Moustafa Mashaal**, Assistant Professor of Internal Medicine, for her sincere help and continuous care, advice and encouragement.

I am so grateful to Dr. **Mohamed Ehab Samy Reda**, Assistant Professor of Radiology, for his endless help and continuous support.

CONTENTS

Contents

1- Introduction	1-20
2- Aim of work	21
3- Subjects	22
4- Methods	23-26
5- Results	27-50
6- Discussion	51-59
7- Summary	60-61
8- Conclusions	62
9- Recommendations	63
10- References	64-77
11- Protocol	78-89
12- Arabic summary	90-93

INTRODUCTION

INTRODUCTION

Hepatocellular carcinoma (HCC) is a malignant neoplasm originating from liver cells, in which the neoplastic cells resemble, to some extent, the morphologic features of hepatocytes . Although it is classified in several types, the morphologic distinctions have no reflection in biological behavior ,the exception being fibrolamellar carcinoma that appear in younger individuals ,it often arises in non -cirrhotic livers, and has a more benign course.^(1,2)

Geographic distribution and Prevalence:

The different geographic distribution seems to be related to the prevalence of factors that carry a risk for the disease namely viruses , chemicals , alcohol , oral contraceptive pills and cirrhosis .⁽³⁾

Of the estimated 350000 new cases per year, one-third occur in China and another third elsewhere in Asia. There are about 30000 cases per year in Europe and 23000 in Japan; the United States has about 7000 cases per year and there are at least six times that number in Africa.⁽⁴⁾

Crude incidence rates do not tell the whole story , and a case can be made that **HCC** in different places is a different disease, primarily depending upon poorly defined host factors and the age when hepatitis virus was acquired.⁽⁵⁾

Pathogenesis :

a) Predisposing factors:

More than **80%** of patients with **HCC** have cirrhosis. Cirrhosis per se seems to predispose to **HCC** , the risk varying according to the etiology of the cirrhosis. Hepatocellular carcinoma is unusual in patients with primary biliary cirrhosis but common when the cirrhosis is secondary to chronic viral hepatitis .

Untreated genetic **haemochromatosis** is an especially severe premalignant state . The risk of death from **HCC** in patients with haemochromatosis has been as high as **45%** in some series.⁽⁶⁾ Iron depletion, if done before the onset of cirrhosis, reduces the incidence of **HCC**, but if cirrhosis has already developed , the risk of **HCC** persists despite iron depletion .⁽⁷⁾

Chronic infection with **Hepatitis B Virus(HBV)** is a strong risk factor for **HCC** .⁽⁸⁾ The lines of evidence associating **HBV** and **HCC** are; numerous , including the world-wide correlation between the incidence of **HCC** and the geographical distribution of hepatitis B surface antigen(**HbsAg**) carriers .^(9,10) Demonstration of the virus itself or its components are present within tumor tissue .⁽¹¹⁾

HBV DNA is integrated into the cellular genome at specific site(s) leads to activation of specific gene(s) responsible for carcinogenesis⁽¹²⁻¹⁴⁾, also other possibilities for **HBV**-induced carcinogenesis are the unregulated expression of the **HBV X** gene product or the **HBV** polymerase .^(15,16)

Hepatitis B virus is a cause of cirrhosis which is listed among the predisposing factors of **HCC** .⁽¹⁷⁾

The age of acquisition of **HBV** plays an important part in the eventual development of cancer . Infants and young children exposed to **HBV** have a much greater chance of becoming chronic carriers than newly infected adults do; and adults who experience acute **HBV** infection and recovery have little if any increased risk of **HCC** .⁽¹⁸⁾

HCC seems less common in **HbsAg** carriers with **delta** . Whether this represents inhibition by delta virus or the rapid progression of the liver disease let death to occur before cancer can develop is uncertain .⁽¹⁹⁾

Hepatitis C Virus (HCV) , unlike **HBV**, does not integrate into host DNA so the mechanism of hepatocarcinogenesis is likely to be different for the two viruses .The transforming potential of the **NS3** region of **HCV**(helicase and protease proteins) and the **HCV** core protein may have a role here. ⁽¹⁾

Chronic infection with hepatitis **C** appears to increase the risk of **HCC** at about the same rate as chronic **HBV** infection does. Treating chronic type **C** hepatitis with interferon decreases the risk of **HCC** ,if the patient responds to this therapy .⁽²⁰⁾

Aflatoxins are produced by some strains of the mould “*Aspergillus flavus* “and most of the strain “*Aspergillus parasiticus* “.The production of aflatoxin depends on the microclimate surrounding and within the cereals and is critically related to humidity .⁽²¹⁾

Alcohol is considered as a promoting agent to **HCC**. A long term alcohol abuse leads to the development of cirrhosis in about **15%** of cases