بسم الله الرحمن الرحيم

شبكة المعلومـــــات الجامعية

التوثيق الالكتروني والميكروفيلم

شبكة المعلومات الجامعية

جامعة عين شمس

التوثيق الالكتروني والميكروفيلم

﴿ قـــسـم ﴾

نقسم بالله العظيم أن المادة التي تم توثيقها وتسجيلها
علي هذه الأفلام قد اعدت دون آية تغيرات

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 %

بعض الوثائق الأصلية تالفة

بالرسالة صفحات

لم ترد بالأصـــل

Effect of Repeated Large Volume Paracentesis on Portal Hemodynamics and Risk of Bleeding in Patients with Mixed Liver Cirrhosis and Tense Ascites

Thesis

Submitted to The Faculty of Medicine – University of Alexandria
in partial fulfillment of the requirements for the Degree of

Master of Internal Medicine

By

Manal Al Said Abdel Motelb Mohamed

MBBCh – Faculty of Medicine – University of Alexandria - 1997

Faculty of Medicine
University of Alexandria
2002

SUPERVISORS

Prof. Dr. Mohamed Ahmed El-Bialy

Professor of Internal Medicine

Medical Research Institute

University of Alexandria

Dr. Mohamed Youssef El-Hassaafy

Assistant Professor of Internal Medicine

Faculty of Medicine

University of Alexandria

Dr. Gamal Ahmed Mohamed Amin

Assistant Professor of Internal Medicine

Medical Research Institute

University of Alexandria

Co-Worker

Dr. Mahmoud Saad Ragab

Assistant Professor of chemical Pathology

Medical Research Institute

University of Alexandria

To Share in Laboratory Investigation

CONTENTS

INTRODUCTION

INTRODUCTION

Schistosomiasis

Schistosomiasis is a chronic debilitating disease of widespread distribution in Africa, South America and the Far East. It is estimated to affect at least 200 million people, and endanger another 600 million in more than seventy subtropical and tropical countries all over the world.[1]

Schistosomiasis is caused by a parasitic helminth of the genus schistosoma and is characterized by the presence of adult worms in the portal and mesenteric veins of man and various other migratory cycle initiated by the cutaneous penetration of water living cercaria.[2]

The main schistosomal species that affect humans are *S. haematobium*, *S. mansoni* and *S. Japonicum*. Recently, a fourth species was discovered, *S. mekongi*. Other species which infect man include *S. bovis* and *S. mateei* which are usually parasites of cattle and sheep. Schistosoma intercalatum naturally infecting sheep, goats and rats can also affect man.[2,3]

In Egypt, it has been estimated that 16 million amongst the population suffer from schistosomiasis or its complications. This number amounts to 37% of the population infected with the disease at that time. However, this rate is not the real incidence of the disease as children in preschool age are not included and method of diagnosis are those used routinely in rural units.[4] It has been proved that in rural areas all exposed inhabitants are usually infected.[5]

Hepatic Schistosomiasis

Prevalence

Hepatic schistosomiasis is probably the world's most prevalent chronic liver disease.[6] Schistosomiasis is the cause of 70% of the Egyptian liver cirrhosis which is complicated mostly by the sequelae of viral hepatitis.[7]

Hashem,[8] by study of autopsy material, found that hepatic schistosomiasis occurrs in 15% of cases infected with *Schistosoma haematobium*, in 72% of *S. mansoni* and in 36% of mixed infection. Yet, Elwi and Attia.[9] reported an incidence of 10,37 and 32% respectively.

Pathology

The intrahepatic schistosomal process starts with the formation of granulmatous cellular infiltration of portal tracts lodging the living ova. On the extent of liver affection: either isolated discrete granulomata with clinically normal liver or widespread granulomatous cellular infiltration of multiple portal tracts resulting in hepatomegaly. This stage is reversible under antischistosomal treatment.

The pathology is produced by repeated embolization of the liver with ova and worms that are shifted back from the terminal radicles of mesenteric and haemorrhoidal veins when they fail to emerge through their walls. Deposition of ova may occur either in large portal branch producing the coarse type of fibrosis [clay-pipe stem] or fine portal branch producing

the fine type form. Ova deposited get surrounded by intense eosinophilic and histocytic cellular reaction in and around the adjacent vessels with swelling of their endothelia and sometimes cellular reaction of their walls. The latter may be so intense as to block the vessel.[7] The sensitizing property was attributed to soluble material that can diffuse out through the intact egg. This material was called the soluble egg antigen (SEA) which induce delayed cellular response.[10] Moreover, this reaction is gradually replaced by fibroblasts with new capillary formation.[7]

The characteristic lesion shows wide bands of fibrosis around portal veins, so that the cut surface of portal tracts resembles cross section through the stem of clay-pipe. The parenchyma between fibrotic areas is typically well preserved correlating with the maintenance of nearly normal hepatic functions, one of the clinical hallmarks of hepatic schistosomiasis.[11,12]

In pure schistosomal hepatic fibrosis the lesion is mainly interstitial, the parenchyma is usually silent with no evidence of gross degeneration or regeneration where fibrosis is mainly periportal and lobular architecture is not altered.[13,14] Furthermore, there is a higher incidence of periportal fibrosis during the recent years which has been attributed to death of large number of worms by antischistosomal treatment and carried back to intrahepatic portal veins where they produce their reaction.[9]

Schistosomal periportal fibrosis obstruct portal blood flow, increasing the tension in portal radicles so portal hypertension may

develop.[15] Splenomegaly in schistosomiasis is congestive as consequence of portal hypertension. Also, it results from reticuloendothelial hyperplesia.[16]

Diagnosis of schistosomiasis

1- Direct methods

These entail identification of parasite itself in body fluids, tissues or excreta. Detection of eggs in stool or urine specimen is possible only after egg production has begun; Direct microscopic examination of stool smears is not very sensitive but may be useful for screening purpose. The Kato thick smear is a simple sensitive quantitation technique that has been used in the field. Rectal snip biopsies have been particularly useful in detection of eggs in patients with light, chronic or inactive infection.

2- Indirect methods

These include searching for schistosomal parasite itself (antigen), or the body's response to parasitic invasion(antibody).

The detection of antigens include

- Quantitation of schistosomal circulating anodic and cathodic antigens in urine and serum.
- Quantitative determination of circulating soluble egg antigen (SEA) in urine and serum by ELISA.
- Monitoring the efficacy of praziquantel by quantification of circulating anodic and cathodic antigens in serum and urine of schistosomal patients by monocolonal antibody based ELISA.

A large number of serological tests have been used in the diagnosis of schistosomiasis which include intradermal test, circumoral precipitation, complement fixation, indirect heamagglutination, and enzyme linked immunosorbent assay. Most of the tests are sensitive.[11]

Indirect heamagglutination test (IHA)

Agglutination reaction is the interaction between antibody and particular antigen that results in visible clumping.[11] In the IHA, red blood cells that have been coated with antigen are allowed to react with test serum. The red cells agglutinate in the presence of the specific antibody. The test is relatively simple to perform and requires only short time.[17] Dilution of the serum allows rough titration of the reaction.[11]

Hepatitis B Virus

Hepatitis B virus (HBV) is a double stranded DNA virus, consists of surface and core. The core contains DNA polymerase, double stranded DNA, core antigen and e-antigen. The surface consists of HBsAg[18] (Figure 1).

Hepatitis B virus infection is a global challenge, there being an estimated 300 million chronic carrier worldwide.[19] The endemicity of HBV infection throughout the world varies greatly. In the populous areas of developing world, in South East Asia, Middle East, Africa and Pacific Island, where the majority of people are seropositive (anti-HBc is more than 60%) and 8-15% are chronic carriers.[20]

In Egypt, the prevalence of HBsAg varies from 4-6% with a rate of exposure to infection ranging from 30-60%.[21] In 1996, Helaly[22] reported a carrier rate of 2% among Egyptian blood donors. Also, in 1998 Shaher reported a carrier rate of 3.3% among blood donors.[23]

Clinical course of Hepatitis B

Most HBV infections are subclinical, particularly during childhood, but about one-third of adult infections are icteric.[24] The course of acute viral hepatitis is conventionally divided into three phases:

1- Preicteric.

2- Icteric.

3- Convalescent.