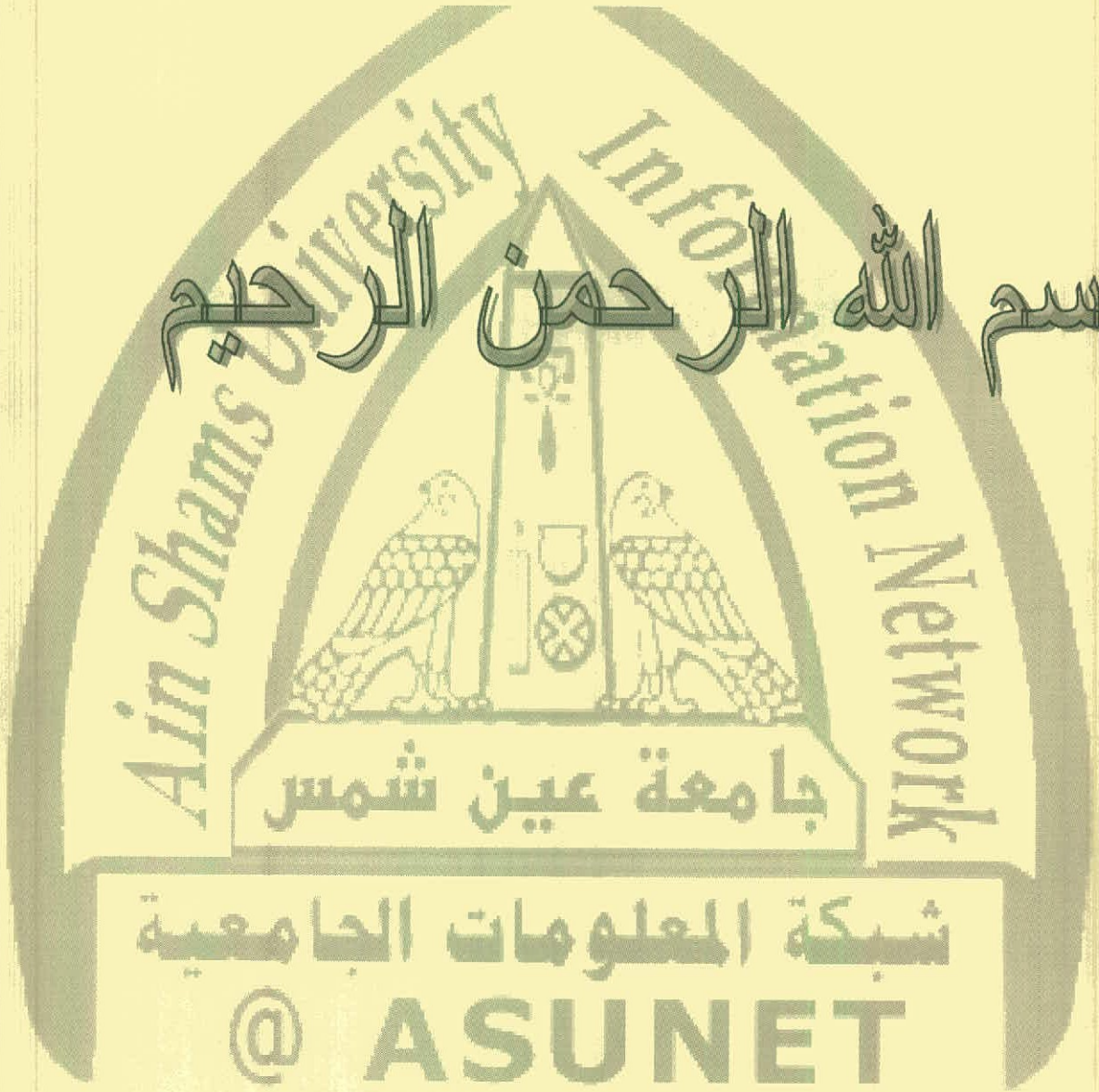
شبكة المعلومات الجامعية



بسم الله الرحمن الرحيم

شبكة المعلومات الجامعية

التوثيق الالكتروني والميكروفيلم

شبكة المعلومات الجامعية

جامعة عين شمس

التوثيق الالكتروني والميكروفيلم

قسم

نقسم بالله العظيم أن المادة التي تم توثيقها وتسجيلها
علي هذه الأفلام قد أعدت دون أية تغيرات

يجب أن

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بعض الوثائـــــــق الاصليـــــة تالفـــة

بالرسالة صفحات لم ترد بالاصل

The Relationship between Helicobacter Infection and Serum Ammonia Level in patients with Hepatic Encephalopathy

Thesis

Submitted to the Faculty of Medicine
University of Alexandria
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Requirements of the degree of

Master of Internal Medicine

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INTRODUCTION

Liver cirrhosis

Definition[1]

Cirrhosis is defined anatomically as a diffuse process of hepatocellular necrosis with fibrosis and nodule formation. Although the causes of necrosis are many the end result is the same.

Pathogenesis[1]

Necrosis follows hepatocellular insult this may be:

- Piece meal necrosis leads to portal-portal fibrous bridges.
- Confluent necrosis leads to central-portal bridging and fibrosis.
- Spotty necrosis is followed by focal fibrosis

The necrosis is followed by nodule formation which disturb the hepatic architecture and a full clinical picture of cirrhosis develops.[2] Moreover, the increased collagen converts sinusoids to capillaries with sinusoidal stenosis, impeding metabolic exchange through the basement membranes between liver cells and blood, and causing portal hypertention.[3]

Classification of cirrhosis[4]

Three anatomical types of cirrhosis are recognized:

1- Aetiological

2- Clinical:

- Compensated - Decompensated

3- Functional :

- Mesenchymal: present by portal hypertension.
- Parenchymal: present by liver failure.

4- Morphological

- Micronodular < 3mm, uniform, separated by thin fibrous septa.
- Macronodular > 3 mm, variable, separated by broad scar.
- Mixed cirrhosis which result when regeneration occurs in micro noduler cirrhosis that leads to mixed appearance.
- Micronodular cirrhosis characterized by thick regular septa, regenerating small nodules and represent impaired capacity for regrowth.
- Macro noduler cirrhosis characterized by septa, nodules of variable size and by normal lobules in the large nodules.

Aetiology[1]

1- Viral hepatitis B; ± Delta; C.
2- Alcohol.
3- Metabolic e.g hemochromatosis, wilson's disease, α_1-antitrypsin deficiency, diabetes mellitus, glycogen storage disease (type IV), glactosemia, congenital tyrosinosis.
4- Prolonged cholestasis, intra and extra-hepatic.
5- Hepatic venous outflow obstruction e.g veno-occlusive disease, budd-chiari syndrome, constrictive pericarditis.
6- Disturbed immunity.
7- Toxins and drugs e.g methotrexate, amiodaron.
8- Intestinal bypass.
9- Indian childhood cirrhosis.

Other possible factors to be considered include:

- Malnutrition.
- Infection e.g. schistosomiasis where the ova excite a fibrous tissue reaction in the portal zone, the association with cirrhosis is probably related to other aetiological factors.
- Granulomatous lesion e.g brucellosis, tuberculosis.
- Cryptogenic cirrhosis where the aetiology is unknown.

Clinical picture[5]

The onset of symptoms may be insidious or less often abrupt. Weakness, fatigability and weight loss are common, anorexia is usually present with associated nausea and vomiting. Abdominal pain may be present and is related eigther to hepatic enlargement and stretching of Glisson's capsule or to the presence of ascites. Menstrual abnormalities, impotance, loss of lipido, sterility and painful enlargement of male breast.

As regard signs the liver is enlarged, palpable and firm and has blunt or nodular edge. Skin manifestation consists of spider navi, palmer erythema and pigmentaiton. Jaundice is mild at first and increasing lately.Ascites, pleural effusion, peripheral edema and purperic lesions are late findings. Clinical splenomegaly is present in 35-50% of cases.

Complications[6]

1- OF portal hypertension

- Esophageal varices
- Ascites
- Hypersplenism
- Spontaneous bacterial peritonitis.

2- Anemia

3- Hemorrhagic tendancy due to hypoprothrombinemia.

4- Hepatocellular carcinoma.

5- Hepatic osteodystrophy.

6- Pigment gall stones.

7- Hepatic encephalopathy.

8- Hepatorenal syndrome.