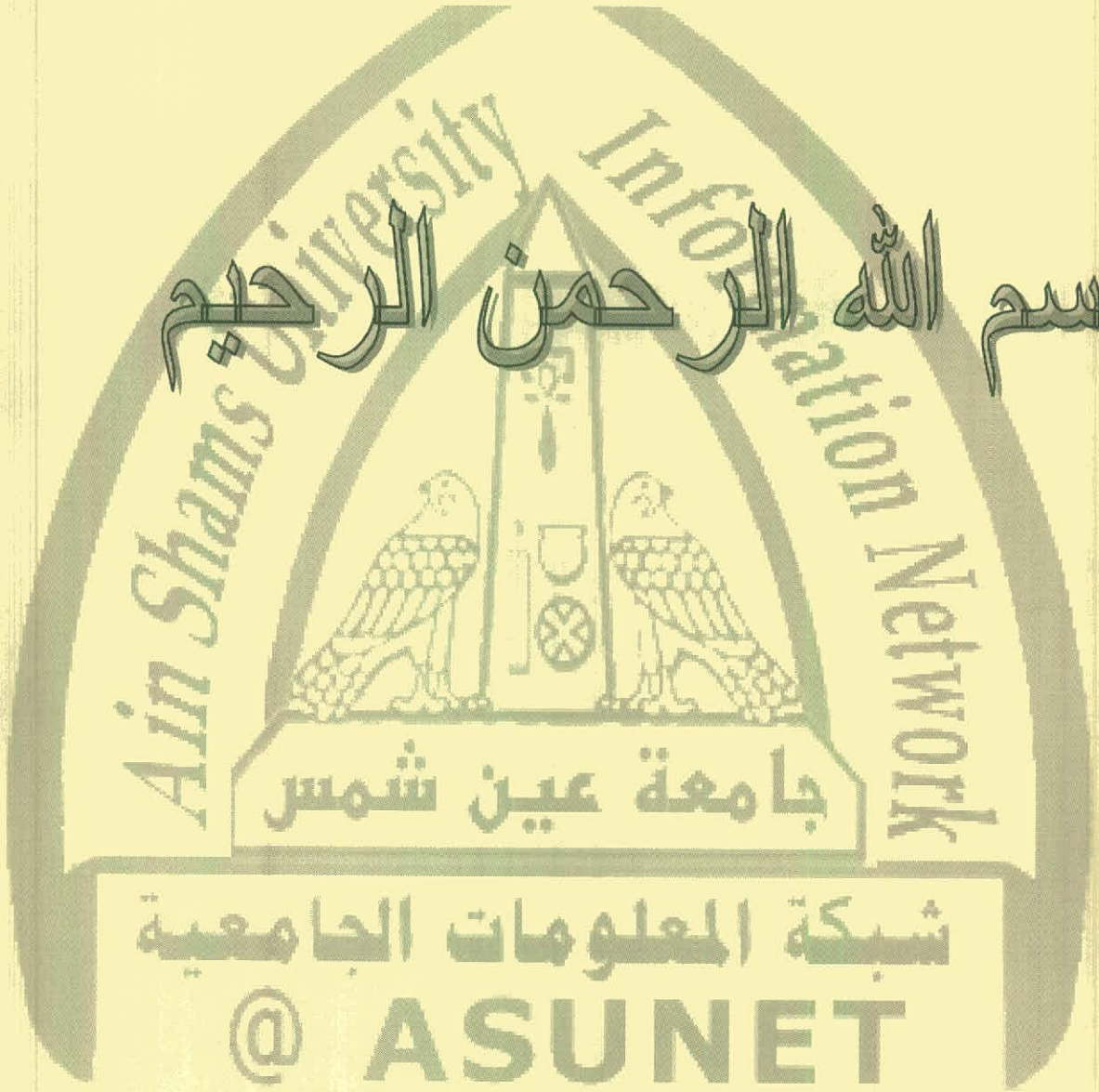
شبكة المعلومات الجامعية



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

تحفظ هذه الأفلام بعيدا عن الغبار

في درجة حرارة من ١٥-٢٥ مئوية ورطوبة نسبية من ٢٠-٤٠%

To be Kept away from Dust in Dry Cool place of 15-25- c and relative humidity 20-40%

بعض الوثائـــــــق الاصليـــــة تالفـــة

بالرسالة صفحات لم ترد بالاصل

Functional and structural cardiac abnormalities in cirrhotic patients with and without ascites

THESIS

Submitted for the partial fulfillment of the
Master degree in general medicine

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بسم الله الرحمن الرحيم

وقل رب زدني علما

صدق الله العظيم

To my father

Acknowledgment

In the beginning thanks to God, who helped me to complete this study.

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List of contents

Review of literature

Haemodynamic changes in liver cirrhosis

(I) Systemic haemodynamic changes in liver cirrhosis:

Liver cirrhosis is associated with several circulatory abnormalities.These include hyperkinetic systemic and splanchnic circulation, hepatopulmonary syndrome including pulmonary hypertension and cirrhotic cardiomyopathy.

[Liu H &Lee SS., 1999]

Liver cirrhosis is associated with marked abnormalities in systemic circulation and renal function that tend to increase with time.

[Gines et al., 1997]

Patients with liver cirrhosis exhibit characteristic haemodynamic changes with hyperdynamic circulation, abnormal distribution of blood volume and neurohumoral regulation.

[Miller & Henriksen, 1997]

Systemic hemodynamic changes occurring in preascitic cirrhosis are increased total blood volume, increased cardiac output and a decrease in total systemic vascular resistance with normal arterial blood pressure. These changes are most marked when the patients are in supine position due to maldistribution of total blood volume with increased volume in the splanchnic circulation that moves to the central circulation when the patient lies down. **[Bernardi & Trevisani , 1997]** . Systemic hemodynamic changes in cirrhotic patient with ascites include increased total blood volume, increased cardiac output, decreased systemic vascular resistance with low arterial blood pressure. **[Moller et al., 1997]**

The most striking hemodynamic difference between cirrhotic patients with and without ascites is the increased activity of renin angiotensin aldosterone system and sympathetic nervous system activity in the ascitic patients.

[Bichet et al., 1982]

(A) Cardiac output

In cirrhotic patients, cardiac output was subnormal at maximal exercise and increasing only by 96% and 97% in alcoholic and non alcoholic cirrhosis respectively and this was considered insufficient compared to normal subject exposed to the same amount of exercise in whom cardiac output is increased by 300% .

[Grose et al., 1995]

Cardiac index was considerably elevated in end stage liver disease giving evidence of marked hyperdynamic circulatory state. Cardiac index was decreased significantly after orthotropic liver transplantation and this produce significant decrease of the mean pulmonary artery pressure.

[Schott et al., 1999]

Cardiac output was increased in-patients with liver cirrhosis and it did not result from diminished afterload because of the increased left ventricular end systolic diameter observed in these patients. As the diameter of the left ventricle during diastolic filling correlate with the vascular volume, cardiac output may be primarily determined by an increase of the intravascular volume. **[Lewis et al., 1992].** They also found that no significant difference in cardiac output between patients with ascites and patients without ascites. And this was in agreement with finding of **Bosch et al., (1980)** who found that cardiac output was markedly increased in cirrhotic patients with and without ascites. And this was in controversy with finding of **Rector& Hossack ., (1988)** as they

found that cardiac output was increased in cirrhotic patients with ascites more than those without ascites.

Lee SS., (1989) found that the circulation in-patient with liver cirrhosis is a state of high cardiac output. And this was in agreement with finding of **Rapaport, (1982)** who found that cardiac output was increased in patients with liver cirrhosis and was attributed to the hyperkinetic state of cirrhosis, while **Schalant, (1980);** attributed the increased cardiac output in liver cirrhosis to the increased heart rate and to the less marked and less frequent increase of the stroke volume.

(B) Heart rate

In supine position, cirrhotic patient presented tachycardia and left ventricular hyperkinesy (increased velocity of left ventricular filling and emptying.

[Hartleb et al., 1997]

Lee SS., (1989) found that the heart rate was increased in-patient with liver cirrhosis and **Rector & Hossack., (1988)** found that the heart rate was increased in cirrhotic patients with ascites more than those without ascites. While **Kelbaek et al., (1984)** found that no significant difference between cirrhotic patients and control subject at rest except for the increased heart rate in cirrhotic patients.

No significant increase of the heart rate in response to tilting was observed in cirrhotic patients as compared to normal subject and this was attributed to impaired baroreflex arc rather than a direct end organ abnormality in the heart.

[Bernardi, et al 1983]

(C) Peripheral vascular resistance

Peripheral arterial vasodilatation is the main vascular dysfunction associated with the hyperdynamic circulation of liver cirrhosis

[Battista et al., 1997]

Systemic vascular resistance was reduced in-patients with liver cirrhosis and it has been suggested that peripheral vasodilatation was the cause as it correlates with the degree of central hypovolemia. Peripheral vasodilatation was attributed to various vasodilator substances that have been found to be increased in liver cirrhosis such as nitric oxide, adrenomedullin, atrial natriuretic peptide and calcitonin gene related peptide.

[Miller & Henriksen. , 1997]

Patients with hepatocellular disease showed marked vasodilatation with reduced systemic vascular resistance, low arterial pressure and increased cardiac output. These were attributed to vasodilator substances and whatever their natures, these substances might be formed by the sick hepatocyte, fail to be inactivated by it or fail to bypass it through the extra or intrahepatic portosystemic shunts. It has been found that, in liver cirrhosis the cardiac index and the reduced systemic vascular resistance correlate with the Child's grade of liver failure. **[Ponte & Cafagna ., 1996].** And this was in agreement with finding of **Braillon et al., (1986)** who found that systemic vascular resistance was reduced in cirrhotic patients and it was positively correlated with the degree of the Child Pugh score of liver cirrhosis.

Peripheral vascular resistance was markedly reduced in cirrhotic patients with and without ascites and this reflecting marked peripheral arteriolar vasodilatation. **[Bosch et al., 1980].** While