شبكة المعلومات الجامعية

التوثيق الإلكتروني والميكروفيلم

بسم الله الرحمن الرحيم

HANAA ALY

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جامعة عين شمس

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قسم

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

يجب أن

تحفظ هذه الأقراص المدمجة بعيدا عن الغبار

HANAA ALY

Measure the level of Ascetic Fluid Mannose Binding Lectin in Patients with Spontaneous Bacterial Peritonitis

And possibility for using as an indicator for inflammation

Thesis

Submitted for partial fulfillment of the m.D.
In internal medicine

By

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بسم الله الرحمن الرحيم
قَالوُاسُبحَانَك لاَعلِمَ لنَا إلا مَاعَلمتَناَ
إنكَ أَنتَ العَلِيمُ الحَكِيمُ
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Hazem elsayed mohamed

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INTRODUCTION

Spontaneous bacterial peritonitis is a very common bacterial infection in patients with cirrhosis and ascites .(***Wong etal.,2005***) when first described its mortality exceeded 90% but it has been reduced to approximately 20% with early diagnosis and treatment .(***Tandon and Garcia-Tsao.,2008***).

Mannose - binding lectin (MBL)is an important protein of humoral innate immune system with multiple carbohydrate recognition domain ,it is able to bind to sugar group displayed on the surface of wide range of micro organism and therepy provide first line defense ,it also activate complement MBL produced in liver in response to infection and is a part of many other factor termed acute phase protein.(***Herpes etal.2009***).MBL deficiency has been reported as risk factor for infection.(***Eisen et al.2008***) .

Finally the MBL deficiency predisposes patient with liver cirrhosis to develop spontaneous Bacterial peritonitis (***chong et al .2005***).

AIM OF THE WORK

To asses the role of Ascitic fluid mannose binding lectin as apossible marker of spontaneous bacterial peritonitis

REVIEW OF LITERATURE

Cirrhotic Ascites

Ascites is the most common complication of cirrhosis, and 60% of patients with compensated cirrhosis develop ascites within 10 years during the course of their disease. Ascites only occurs when portal hypertension has developed and is primarily related to an inability to excrete an adequate amount of sodium into urine, leading to a positive sodium balance **(Ripoll et al., 2007)**.

Tandon and Garcia-Tsao (2008) suggested that renal sodium retention in patients with cirrhosis is secondary to arterial splanchnic vasodilation. This causes a decrease in effective arterial blood volume with activation of arterial and cardiopulmonary volume receptors, and homeostatic activation of vasoconstrictor and sodium-retaining systems (i.e., the sympathetic nervous system and the renin–angiotensin–aldosterone system). Renal sodium retention leads to expansion of the extracellular fluid volume and formation of ascites and edema. The development of ascites is associated with a poor prognosis and impaired quality of life in patients with cirrhosis.

Uncomplicated ascites:

Approximately 75% of patients presenting with ascites in Western Europe or the USA have cirrhosis as the underlying cause. For the remaining patients, ascites is caused by malignancy, heart failure, tuberculosis, pancreatic disease, or other miscellaneous causes **(EASL,2010)**.

Diagnosis of ascites:

The initial evaluation of a patient with ascites should include history, physical examination, abdominal ultrasound, and laboratory assessment of liver function, renal function, serum and urine electrolytes, as well as an analysis of the ascitic fluid.

The International Ascites Club proposed to link the choice of treatment of uncomplicated ascites to a classification of ascites on the basis of a quantitative criterion. A diagnostic paracentesis with an appropriate ascitic fluid analysis is essential in all patients investigated for ascites prior to any therapy to exclude causes of ascites other than cirrhosis and rule out spontaneous bacterial peritonitis (SBP) in cirrhosis **(EASL, 2010)**.

When the diagnosis of cirrhosis is not clinically evident, ascites due to portal hypertension can be readily differentiated from ascites due to other causes by the serum–ascites albumin

gradient (SAAG). If the SAAG is greater than or equal to 1.1 g/dl (or 11 g/L), ascites is ascribed to portal hypertension with an approximate 97% accuracy **(Runyon, 2009)**.

Ascitic fluid inoculation (10 ml) in blood culture bottles should be performed at the bedside in all patients. Other tests, such as amylase, cytology, PCR and culture for mycobacteria should be done only when the diagnosis is unclear or if there is a clinical suspicion of pancreatic disease, malignancy, or tuberculosis **(Moore et al., 2003)**.

A diagnostic paracentesis should be performed in all patients with new onset grade 2 or 3 ascites, and in all patients hospitalized for worsening of ascites or any complication of cirrhosis. Neutrophil count and culture of ascitic fluid (by inoculation into blood culture bottles at the bedside) should be performed to exclude bacterial peritonitis **(EASL,2010).**

It is important to measure ascitic total protein concentration, since patients with an ascitic protein concentration of less than 15 g/L have an increased risk of developing spontaneous bacterial peritonitis (Level A1) and may benefit from antibiotic prophylaxis (Level A1) **(European Association for the Study of the Liver, 2010).**

Measurement of the serum–ascites albumin gradient may be useful when the diagnosis of cirrhosis is not clinically

evident or in patients with cirrhosis in whom a cause of ascites different than cirrhosis is suspected (EASL,2010**).**

Prognosis of patients with ascites:

The development of ascites in cirrhosis indicates a poor prognosis. The mortality is approximately 40% at 1 year and 50% at 2 years. The most reliable factors in the prediction of poor prognosis include: hyponatremia, low arterial pressure, increased serum creatinine, and low urine sodium **(EASL, 2010)**

These parameters are not included in the Child-Turcotte-Pugh score (CTP score) and among them, only serum creatinine is included in the Model for end-stage liver disease (MELD score). Furthermore, since serum creatinine has limitations as an estimate of glomerular filtration rate in cirrhosis, these scores probably underestimate the mortality risk in patients with ascites. Since allocation for liver transplantation is based on the MELD score in several countries, patients with ascites may not receive an adequate priority in the transplant lists. Therefore, there is need for improved methods to assess prognosis in patients with ascites **(Heuman et al., 2004)**.

Recommendations: Since the development of grade 2 or 3 ascites in patients with cirrhosis is associated with reduced