Ain Shams University

Faculty of Medicine

Internal Medicine Department

ROLE OF SERUM AND ASCETIC FLUID HIGH SENSITIVITY C-REACTIVE PROTIEN IN DIAGNOSIS OF SPONTANOUS BACTERIAL PERITONITIS

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By

IhabAbdelazizAbdelmotyMesbah

M.B.,B.CH

Supervisors

Professor Doctor/ Said Mohamed Shalaby

Professor of Internal Medicine
Faculty of Medicine
AinShams University

Professor Doctor / RawyaAbd El salamAlfeky

Professor of Internal Medicine
Faculty of Medicine
AinShams University

Doctor / Wesam Ahmed Ibrahim Mohamed

Assistant Professor of Internal Medicine
Faculty of Medicine,
Ain Shams University

Faculty of Medicine
AinShamsUniversity
2014

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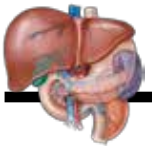

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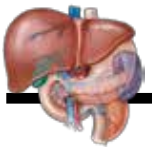

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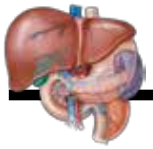

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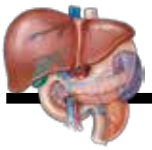

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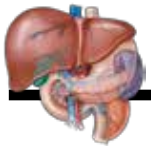

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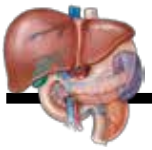
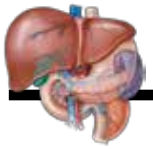
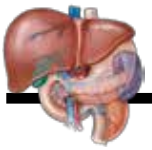


ABBREVIATIONS

AASLD	***American Association for the study of liver diseases***
AFB	***Acid fast bacilli***
ALT	***Alanine Transaminase***
APC	***Antigen presenting cells***
AST	***Aspartate Transamiase***
BP	***Blood pressure***
BT	***Bacterial Translocation***
CAP	***Community Acquired pneumonia***
CNNA	***Culture-Negative Neurtocytic Ascitis***
CPNA	***Culture-positive Neutrocytic Ascitic***
CRP	***C-Reactive protein***
CT	***Computed Tomography***
CTP	***Child-Turcotte pugh***
EASL	***European Association for the study of liver disease***
ESR	***Erythrocyte Sedimentation Rate***
ESRD	***End stage Rend disease***
GALT	***Gut-Associated Lymphoid Tissue***
G.I	***Gastro-intestinal***
HAV	***Hepatitis A Virus***



HBsAg	***Hepatitis B Surface Antigens***
HcvAb	***Hepatitis C virus Antibody***
HE	***Hepatic Enchephalopathy***
HRS	***Hepato-renal syndrome***
HsCRP	***High sensitivity C-Reactive protein***
IL6	***Interleukin-6***
INR	***International Normalizied Ratio***
I.V	***Intravenous***
LDH	***Lactate Dehydrogenase***
LVP	***Large volume paracentesis***
MAF	***Machrophage Activity factor***
MELD	***Model for End-Stage Liver Diseases***
MIF	***Migration Inhibitory Factor***
MLN	***Mesenteric Lymph Node***
MNP	***Monomicrobial Non neutrocytic bacterascitis***
NK	***Natural Killer***
PAMP	***Pathogen-Associated Molecular pattern***
PMN	***Polymorphnuclear lrycocytic count***
PT	***Prothrombin Time***
PSI	***Pneumonia severity Index***
PVS's	***Peritoneo-venous shunt***
PRR	***Pattern Recognition Receptor***
SAP	***Serum Amyloid p compound***



SAA6	***Serum-Ascitis albumin gradient***
SIRS	***Systemic inflammatory Response syndrome***
SBp	***Spontaneous Bacterial peritonitis***
T.B	***Tuberculosis***
TIPs	***Transjugular Intrahepatic portosystemic shunt .***
TLR	***Tool like Receptor***
US	***Ultra sonography***

Aime of the Work

The aime of the work to evaluate the role of both serum and ascitic fluid high sensitivity C - reactive protein in diagnosis of spontaneous bacterial peritonitis .

Introduction

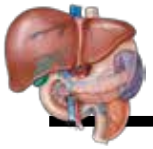
Liver cirrhosis is a frequent phenomenon in chronic liver disease such as hepatitis B, hepatitis C, alcoholic – related liver damage, autoimmune hepatitis and hemochromatosis **(Van Erpecum, 2006)**.

Ascites is a collection of extracellular fluid in the peritoneal cavity resulting from imbalance between inflow and outflow through peritoneal membrane **(Bataller et al., 1997)**.

Ascites is the most common complication in patients with decompensated cirrhosis. Approximately 50% of patients with compensated cirrhosis will develop ascites over a 10 – year's period **(Saadeh and Davis, 2004)**.

Patients with cirrhosis and ascites show a higher susceptibility to bacterial infections mainly because of the inadequate defence mechanisms. The most Frequent and the most severe one begin spontaneous bacterial peritonitis (SBP) **(Garcia – Tsao, 2005)**

SBP is bacterial infection of the ascitic fluid without any intra abdominal source of infection **(Frances et al., 2004).**



The Prevalence of SBP in cirrhotic patients with ascites has been estimated at 10 to 30% **(Evans t al., 2003).**

There are some mechanisms that are being proposed to explain bacterial translocation (BT) in cirrhosis: the intestinal bacterial overgrowth, The structural and functional alterations of the intestinal mucosal barrier and the deficiencies of the local immune response **(Guarner and Soriano, 2005).**

Symptoms of SBP include: fevers, chills, nausea, vomiting, abdominal tenderness and general malaise. Patients may complain of abdominal pain and worsening ascites (Filik and Unal, 2004).

For SBP Diagnosis, the number of polymorphonuclear leucocytes (PMN) from the ascitic fluid obtained by paracentesis must exceed 250 cells / mm3 and from bacteriological cultures only one germ must be isolated **(Mandell et al., 2005).**

Cefotaxime or other third-generation cephlosporins have been considered the first-choice empirical antibiotics in the treatment of cirrhotic patients with SBP and is efficacius in approximately 90% of cases (Strauss and Caly, 2006). Broad-spectrum quinolones which almost completely absorbed after oral administration and diffuse rapidly through the ascitic fluid

are currently used for oral treatment of uncomplicated SBP **(Strauss and Caly, 2006).**

Prophylactic oral norfloxacin is extremely useful in preventing SBP in patients that are at high risk for developing SBP such as hospitalized cirrhotic patients with gastrointestinal hemorrhage or low ascitic fluid protein **(Guarner and Soriano, 1997).**

Liver Cirrhosis

History:

Cirrhosis was first described in the fourth century B.C. hippocratic aphorism: "In case of jaundice, it is a bad sign when the liver becomes hard **(Chen and Chen, 1984)**. The word "cirrhosis" is a neologism that derives from Greek kirrhos, meaning "tawny" (the orange-yellow colour of the diseased liver). While the clinical entity was known before, it was Rene Leannec who gave it the name "cirrhosis **(Rogun, 2006).**

Definition:

Cirrhosis is a slowly progressive disease, causing irreversible scarring and nodularity of the liver in response to chronic injury from a variety of causes **(Rimola et al, 2000)**. This process distorts the normal liver architecture, interferes with blood flow through the liver and disrupts the biochemical functions of the liver **(Mathews et al, 2006)**.