ANALYSIS OF THE ASCITIC FLUID
IN DECOMPENSATED HEPATIC SCHISTOSOMIASIS

THESIS

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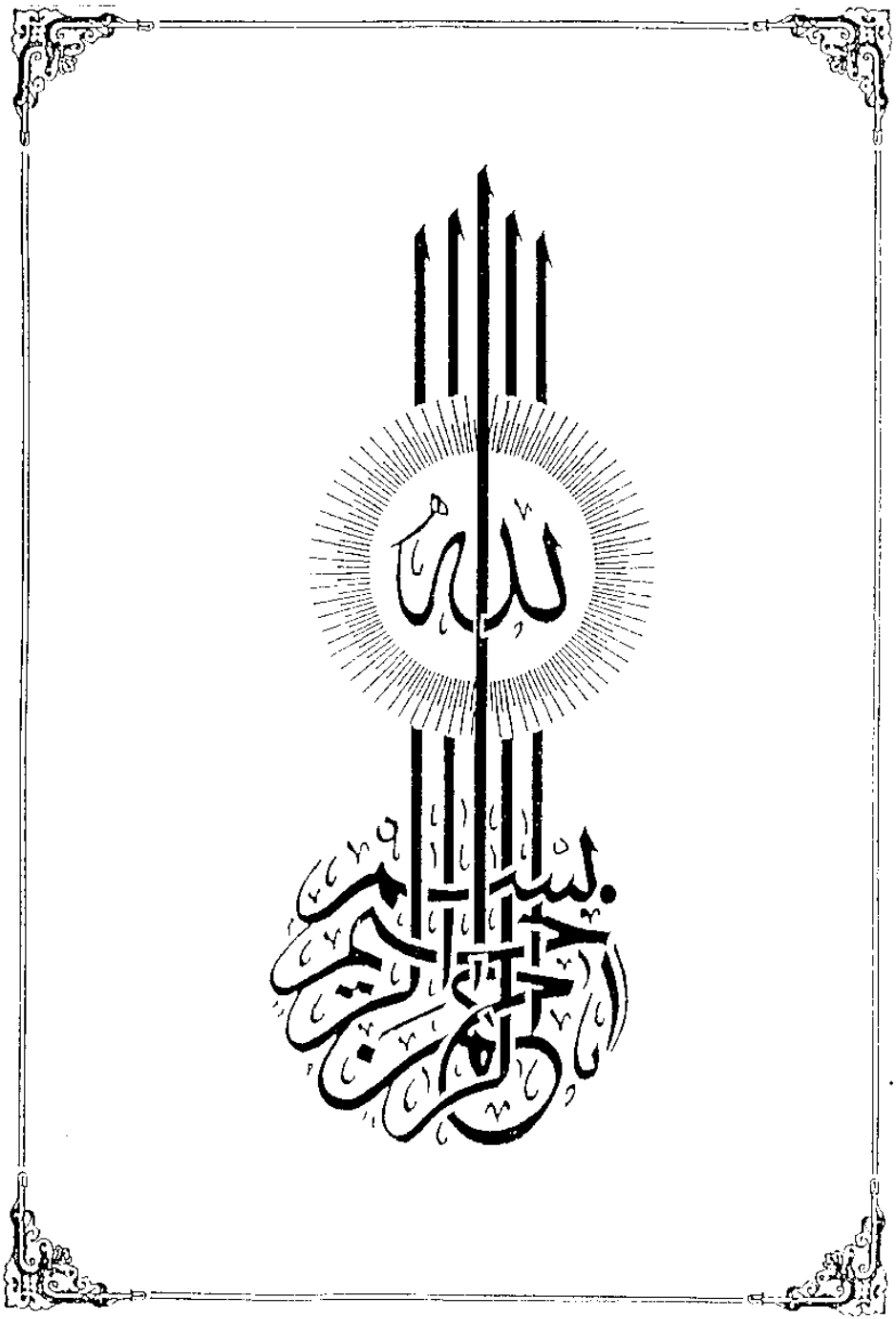

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AIM OF WORK

INTRODUCTION

Analysis of the ascitic fluid has been valuable in the diagnosis of different causes of ascites which may occur as a part of generalized anasarca or as a lone ascites.

Ascites is classically divided into two main categories, transudates and exudates.

Ascites has been defined as a transudate, a fluid of low specific gravity (<1015), and a low protein concentration (<3g/dl) that has passed through a membrane or extruded from a tissue.

Moreover, the recognition of spontaneous bacterial peritonitis as a common problem, has focused attention on the ascitic fluid and its constituents.

Untill recently, only a few papers had presented data about the number of leukocytes in "normal" ascitic fluid.

In this study, the available investigations were used to analyse the composition of the ascitic fluid in cirrhotic patients with a special attention to the number and type of leukocytes in an attempt to detect which are the most accurate criteria for the differentiation between infected from unifected fluid and to put a rational basis on which to make urgent therapeutic decisions.

REVIEW OF LITERATURE

ASCITES

Historical Notes On Ascites:

The term ascites was derived from the Greek word "askos", meaning bag and was introduced in 1398 by trenisa.

The Ancient Egyptian 3000 B.C. were aware of abnormal collections of abdominal fluid associated with disease of the liver (Hyatt and Smith, 1954).

Hippocrates (460-377 B.C.) and Erasistratos of Alexandria (250 B.C.) had also respectively recognized the association between liver disease and dropsy.

One of the aphorisms attributed to Hippocrates states "When the liver is full of fluid and this overflows into the peritoneal cavity so that belly becomes full of water, death follows".

Erasistratos of Alexandria postulated that ascites was due to the stone like hardness of the liver, commenting that "Blood is prevented from going forward into the liver owing of the narrowness of the passages (Dawson, 1960).

Galen (130-200 A.D.) contrary to eariler suggestions, refused the saying that water can not accumulate in the skin in any other way than liver disease, because he had seen ascites resulting from disease of the spleen,

and other organs, he summed up with a view on the pathogenesis which reigned for the next 10 centuries "Water in skin (anasarca or dropsy) arises from too many humours, we hold that water in skin is cold and moist and it arises from abundancy of cold and moist humours" (Dawson, 1960).

Definition:

Ascites is the term applied to accumulation of abnormal volume of free fluid within the peritoneal cavity, it may be produced by a large variety of conditions.

Ascites is classically divided into two categories:

I- Transudates: Which are morbid accumulation of fluid due to a hydrostatic (elevated portal or systemic venous pressure) or osmotic etiology, despite normal permeability.

II- Exudates: Which are morbid accumulation of fluid due to increased vascular permeability of the peritoneum.

Dynamics of ascitic fluid:-

Ascitic fluid is far from resembling a stagnant pool, its constituents once formed are in dynamic equilibrium with those in plasma, through an enormous capillary bed under the visceral peritoneum.

Albumin diffuses from plasma to ascites at a rate of approximately 4 per cent per hour (Dykes and Jones, 1968).

In presence of portal hypertension, the maximum rate of reabsorption of ascitic fluid is limited and is much less than the maximum rate of reabsorption of oedema this rate can not be exceeded despite vigorous diuresis, rather, such diuresis only serves to remove fluid from other body compartments and may cause hypovolaemia (Losowsky and Scott, 1973).

Causes of Ascites:

Ockner, R.K., (1979), classified the causes of ascites into diseases not involving the peritoneum on one hand and diseases of peritoneum on the other hand.

A) Causes of ascites not associated with peritoneal disease:-

I- Portal Hypertension:

1. Cirrhosis.

2. Hepatic congestion:-

a) Congestive heart failure.

b) Constrictive pericarditis.

c) Inferior vena cava obstruction.

d) Hepatic vein obstruction (Budd-Chiari Syndrome).

3- Portal vein occlusion.

II- Hypoalbuminaemia:

1- Nephrotic syndrome.

2- Protein losing enteropathy.

3- Malnutrition.

III- Miscellaneous:

1- Myxedema.

2- Ovarian disease:

a) Meig's syndrome.

b) Straum ovarii.

c) Ovarian over stimulation syndrome.

3- Pancreatic ascites.

4- Chylous ascites.

B) Causes of ascites involving the peritoneum:-

I- Infection:

1- Acute peritonitis.

2- Tuberculous peritonitis.

3- Fungal diseases.

4- Parasitic diseases:

a) Schistosomiasis.

b) Amebiasis. c) Ascariasis.

II- Neoplasm:

1. Secondary malignancy.
2. Primary mesothelioma.
3. Pseudomyxoma peritonei.

III- Miscellaneous:

1. Vasculitis.
2. Granulomatous peritonitis:
    a) Sarcoidosis
    b) Crohn's disease.
    c) Starch peritonitis.
3. Familial paroxysmal peritonitis.
4. Whipple's disease.
5. Gynecologic diseases:
    a) Endometriosis
    b) Deciduosis.
    c) Dermoid cyst.
    d) Melanosis.
6. Splenosis.
7. Sclerosing peritonitis.
8. Peritoneal lymphangiectasis.
9. Eosinophilic gastro-enterilitis.

Pathogenesis of important causes

I- In liver cirrhosis

Ascites in liver cirrhosis results from disturbances of both local and systemic mechanisms that regulate the passage of fluid and solutes across vascular and serosal membrane (Lamont, Koff and Isselbacher, 1979).

A) Local Causes favoring ascites formation in cirrhosis:-

1- Portal hypertension:

The role of portal hypertension in the formation of ascites is still not clear. Patients with portal hypertension due to extra-hepatic vein block (Pre-simusoidal) do not generally have ascites. On the other hand, reduction in portal pressure by side to side porto-caval anastmosis result in improvement of ascites. It is likely that portal hypertension combined with other factors contributes to the formation and persistence of ascites in cirrhotic patients. Sherlock (1975), suggested that portal hypertension helps to localize the fluid retention in the peritoneal cavity rather than in the peripheral tissue.

2- Obstruction to hepatic vein radicles:

Post-sinusoidal block or diffuse block of hepatic venous system (by cirrhosis or infiltrative disease) leads to ascites.

3- Increased flow of hepatic lymph:

Weeping of lymph from the surface of the cirrhotic

liver can be observed at surgery, this presumably results from distortion and blockage of hepatic sinusoids and lymphatics resulting in extra-vasation of protein rich lymph into the peritoneal cavity.

Intrahepatic (post-sinusoidal) and extrahepatic (pre-sinusoidal) portal hypertension often coexist in patients with liver cirrhosis, distinction between them is readily apparent from high protein content of ascitic fluid and thoracic duct in the former disorder and low protein content of these fluids in the latter (Witte, M.H. et al., 1971).

Summer Skill and Bladus (1975), stated that, in Schistosomiasis, ascites arises with portal hypertension due to block in pre-sinusoidal veins, the fluid has the characteristics of a transudates.

B) Systemic factors favoring ascites formation in cirrhosis:

1- Decreased plasma colloidal osmotic pressure:

The serum albumin is low in cirrhotic patients because of a combination of expansion of plasma volume, causing dilution of serum proteins, impaired hepatic synthesis and loss of albumin from the vascular space into the peritoneal cavity.

This hypoalbuminaemia is associated with decrease

of plasma osmotic pressure and loss of water in the extra-vascular space.

2- Increased sodium retention:

Patients with cirrhosis and ascites have secondary hyperaldosteronism and consequently marked sodium retention. Secondary hyper-aldosteronism is presumably due to reduction in renal flow and impaired hepatic metabolism and excretion of aldosterone. Impaired activity of "third" factor secondary to liver cirrhosis with consequent increased proximal tubular reabsorption of sodium is believed to be another important factor than aldosterone.

3- Impaired water excretion:

Patients with ascites have impaired renal excretion of water load, which is due to reduced renal vascular flow and in some patients to excessive serum level of antidiuretic hormone (Lamont, Koff and Isselbacher,1979).

Read, (1978), stated that though hypoalbuminaemia and portal hypertension seem the major factors which determine the production of ascites, its persistence must be due to other factors, most propably those involving renal sodium excretion.

II- In heart disease:

Ascites is a late manifestation of right side heart failure, often occuring in conjunction with severe