EFFECT OF LOOP DIURETICS AND TAPPING ON INTRAPERITONEAL PRESSURE AND SODIUM EXCRETION IN URINE IN CIRRHOTIC PATIENTS WITH ASCITES

T H E S I S

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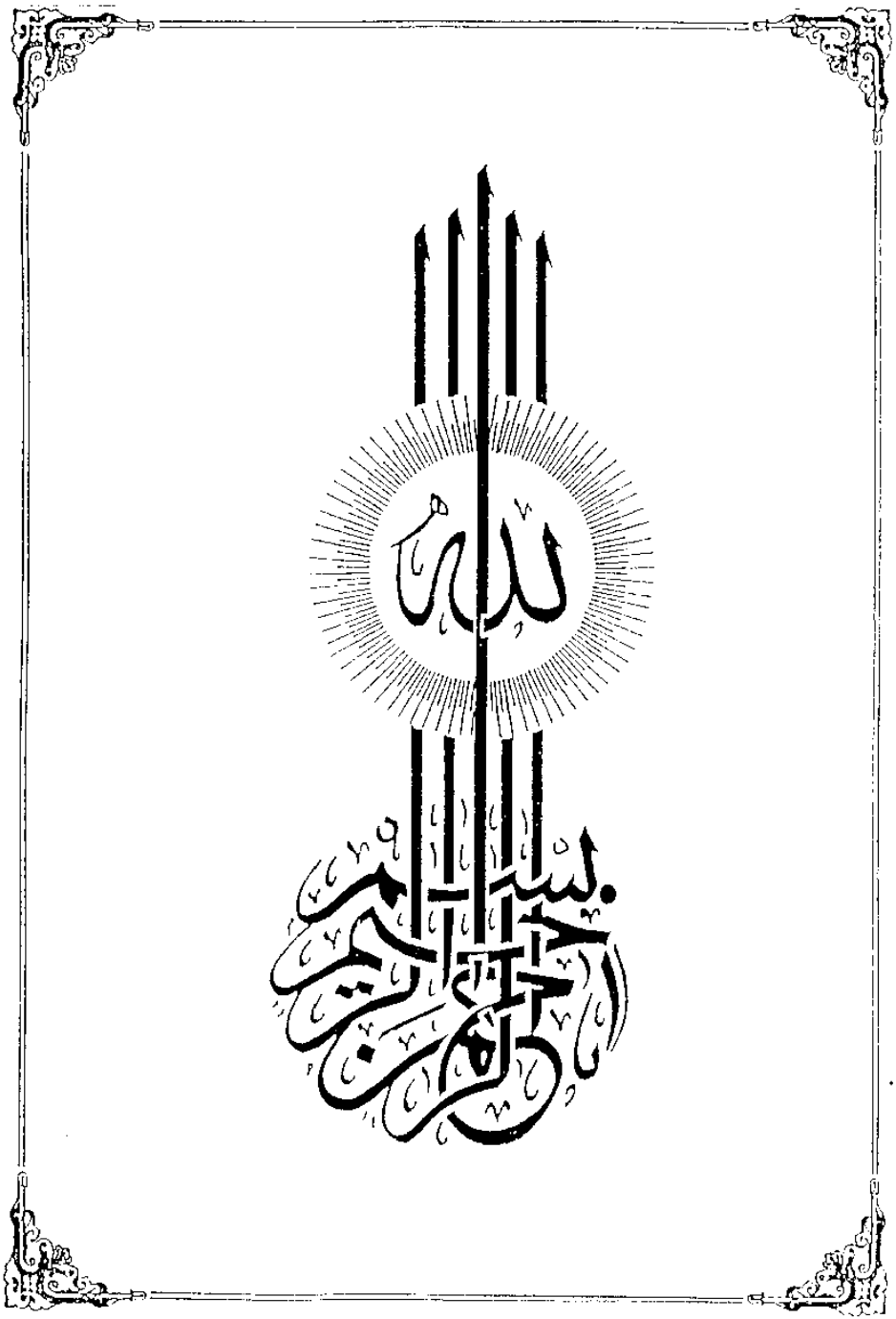

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AIM OF WORK

Patients who do not respond to the standard therapeutic regimen are designated as having refractory ascites. A number of factors may precipitate the development of refractory ascites in a patient with liver disease including: deterioration of liver function as a result of active liver cell damage or the development of a complicating primary hepatocellular carcinoma, spontaneous bacterial peritonitis, chylous ascites and hepatic venous thrombosis (Budd Chiari Syndrome).
In these resistant cases alternative therapy must be considered.

Aim of the Work:

The aim of this work is to study the correlation between the intraperitoneal pressure and sodium excretion in the urine of patients with ascites secondary to chronic liver disease and to correlate the effect of various therapeutic combinations on intraperitoneal pressure and sodium excretion in urine.

REVIEW OF LITERATURE

ASCITES

Peritoneal fluid & ascites:

The adult peritoneal cavity contains normally from 100-200 ml. of free non purulent fluid. Ascites is usually detectable clinically when 500 ml. or more of free fluid has accumulated in the peritoneal cavity (Trisdale et al., 1974).

Ascites is not a disease in itself but a manifestation of many diseases of various origins. It complicates all forms of cirrhosis and implies a poor prognosis (Ratnoff and Patek, 1942).

Ascites - Plasma interchange:

It has been shown that ascites is in a dynamic equilibrium with the plasma . It is a continuously circulating fluid, about half of the amount present leaves and enters the peritoneal cavity every hour there being a rapid transit in both directions (Birkenfeld et al., 1958 and Shaldon 1958).

Prentice and his Colleagues (1952) have shown that the water content of ascitic fluid is exchanged with that of the blood at a rate of 40-80% each hour.

The proteins of ascitic fluid also readily interchange

with those of the plasma and this is known as dynamic equilibrium.

Both albumin & globulin participate in the interchange between blood and ascites, albumin turnover being 3 times more rapid than that of globulin (McKee et al., 1952).

Peritoneal permeability is an important factor in ascites production (Mankin and Lowell, 1984). Peritoneal permeability increases in cases of liver cirrhosis because of increased capillary permeability, probably due to associated malnutrition. (Kark, 1951).

AETIOLOGY OF ASCITES

Causes of ascites:

Ockner (1979) classified the causes of ascites into:

A. Diseases not involving the peritoneum.

B. Diseases involving the peritoneum.

A. Diseases not involving the peritoneum include:

1. Diseases causing portal hypertension.
   - Cirrhosis.
   - Hepatic congestion : from :
     a. Congestive heart failure.
     b. Constrictive pericarditis.
     c. Inferior vena caval obstruction.
     d. Hepatic vein obstruction (Budd-Chiari-syndrome)
   - Portal vein occlusion.

2. Diseases resulting in hypoalbuminemia:
   - Nephrotic syndrome.
   - Protein losing enteropathy.
   - Malnutrition.

3. Miscellaneous:
   - Myxedema.
   - Ovarian diseases as:
     a. Meig's syndrome.
     b. Strauma ovarii.

. Pancreatic diseases due to ruptured duct or pseudocyst.

. Chylous ascites.

B. Diseases involving the peritoneum:

1. Infection:

. Acute peritonitis.

. Tuberculous peritonitis.

. Fungal diseases.

. Parasitic diseases: as :

a. Schistosomiasis.

b. Amebiasis.

c. Ascariasis.

However these parasitic diseases were not verified in clinical practice

2. Neoplasms:

. Secondary carcinomatosis.

. Primary mesothelioma.

. Pseudomyxoma peritonii.

3. Miscelloneous:

. Vasculitis.

. Granulomatous peritonitis as:

a. Sarcoidosis.

b. Crohn's disease.

. Familial paroxysmal peritonitis.

. Whipple's disease.

. Gynaecological causes:

  a. Endometriosis.

  b. Dermoid cyst.

. Esinophilic gastro-enteritis.

There are rare causes of ascites including hypothyroidism and systemic lupus erythematosis (Wilkinson, 1979 and Mier, 1985). Also Stocker (1985), has reported a case of congenital cytomegalovirus infection presenting with massive ascites.

Wilkins (1985), reported cases of S.L.E which presented as massive ascites and Mier (1985) also reported cases of S.L.E ascites in the absence of nephrotic syndrome, congestive heart failure or liver cirrhosis. There were three primary omental lieomyosarcomata which have been described to date and all were presenting as hemorrhagic ascites (Dixon, 1984).

Also Wheeler (1985), has reported that although bloody ascites due to sarcoidosis is rare, two patients in whom sarcoidosis was manifested as bloody ascites, while five patients with non bloody ascites were diagnosed as abdominal sarcoidosis.

On the other hand, Rowland (1985), reported patients with E.coli spontaneous bacterial peritonitis with 80%

esinophilia in the peritoneal fluid and no peripheral esinophilia. They gave good response to antimicrobial treatment.

Boyer et al., (1977) divided the causes of ascites into those that are 'Transudative' and those that are "Exudative".

The usual way for differentiating these 2 types of ascitic fluid was the measurement of total protein content. However sampliner and Iber in 1974 declared that neither protein content levels of 2.5 or 3.0 g/dl. consistently differentiate portal hypertension and non portal hypertension fluids.

Reynolds and Campra (1985) prefer to use the difference between serum albumin and ascites albumin levels as the best indicator for whether or not a fluid is related to portal hypertension. The albumin levels of serum and ascites are closely related to their oncotic pressures. If the oncotic pressure difference between the serum and ascites is large, an equally large hydrostatic pressure difference can be assumed between the portal venous compartment and the intraperitoneal space (Hoefs, 1978).

Hoefs in 1980 compared the portal pressure (Transhepatic portal pressure minus vena caval pressure) with the albumin level difference between serum and ascites

in 54 patients and found an inverse relationship. When the serum albumin - ascites albumin difference exceeds 1.0 to 1.1 g/dl, there is appreciable portal hypertension & this is probably the cause of ascites in such patients (Hoefs, 1980).

Laparoscopy:

Larparoscopy is an ideal method for the differential diagnosis of ascites. In order to see through the laparoscopic telescope, considerable ascites must be removed & replaced by air, but fluid removal is no longer viewed as a dangerous maneuver. Cirrhosis can be readily recognized, as can peritoneal tuberculosis or carcinomatosis (Reynolds and Campra 1985).