COMPARISON OF ORAL VERSUS INTRAVENOUS THERAPY OF TENSE ASCITES IN LIVER CIRRHOSIS

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By

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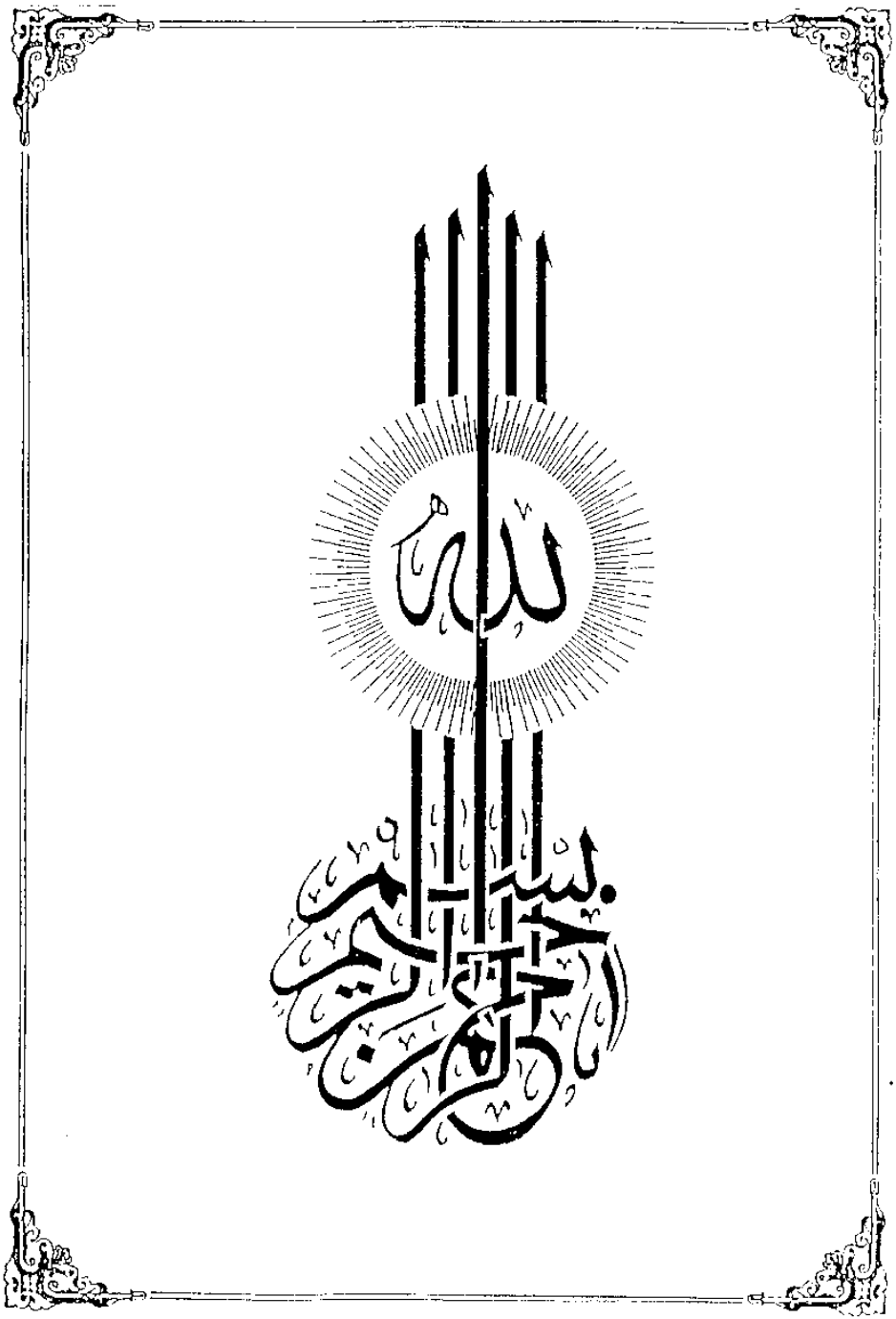

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REVIEW OF LITERATURE

CHAPTER I.

CHRONIC LIVER CELL FAILURE

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CHRONIC LIVER CELL FAILURE

The presentation of the decompensated cases of liver cirrhosis is by liver cell failure or portal hypertension, singly or in combination. At the same time, the well compensated liver cirrhosis is usually asymptomatic being often discovered accidentally on physical examination (Maher, I977).

The clinical picture of chronic liver cell failure may be variable and non-specific. It includes: (1) Hepatocellular jaundice which in patients with cirrhosis indicates activity of the disease and is a presenting sign of hepatic cell failure (Leon, I969); (2) Fever, chills and malaise which are not affected by antibiotics or by altering the protein content of the diet (Sherlock, I975); (3) Gastrointestinal symptoms, such as pain, diarrhoea, constipation, malnutrition, impaired appetite and loss of weight; (4) Neuro-psychiatric states, such as psychoneurotic manifestations, peripheral neuritis, headaches and impending or terminal hepatic coma; (5) Haematologic manifestations,

such as haemorrhage, hypersplenism and anaemias of various types; (6) Many clinicians include ascites, oedema and portal hypertension in addition to the endocrinal and renal changes (Martin, 1960).

Renal failure which is found in patients with chronic liver cell failure, is called "hepatorenal failure". It is one of the reversible chronic renal failures (Souidan, 1969).

Renal failure is a grave complication and generally is seen only in patients with advanced cases of chronic liver cell failure. It is encountered in patients who have had gastro-intestinal bleeding. In these, it has been attributed to the absorption of nitrogenous products and to impaired renal functions from shock causing ischaemia of the kidneys. It can also occur abruptly after abdominal paracentesis, presumably due to sudden reduction in extracellular fluid. However, the changes of renal functions often are moderate and usually do not account for the degree of failure (Leon, 1963).

Cirrhosis is the main cause of intrahepatic portal hypertension but there are various liver lesions

other than cirrhosis which can lead to an increase in the portal venous pressure (Table 1) (Jean, 1985).

Table 1. Chronic liver lesions commonly associated with portal hypertension

1. Schistosomiasis.	6. Congenital hepatic fibrosis.
2. Veno-occlusive disease.	7. Nodular regenerative hyperplasia.
3. Peliosis hepatis.	8. Partial nodular transformation.
4. Primary biliary cirrhosis.	9. Hepatoportal sclerosis.
5. Secondary biliary cirrhosis.	10. Idiopathic portal hypertension.

However, cirrhosis in elderly individuals is often clinically silent and the aetiology is usually inapparent. Also, the complications in elderly are much the same as in younger patients with the exception that chronic hepatic encephalopathy tends to occur more frequently and its presence is more often overlooked on the assumption of cerebral atherosclerosis (Mooney, 1985).

CHAPTER II.

PATHOLOGY OF ASCITES

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A. Aetiology Of Ascites

Ascites refers to accumulation of free fluid within the peritoneal cavity. While liable to occur with any liver disease, ascites is the commonest major complication of cirrhosis and implies a poor prognosis (Leon, I975).

Galambos (I979), reported that ascites may be due to portal hypertension, hypoalbuminaemia, infections, neoplasms or other miscellaneous causes, all are listed in table 2.

Table 2. Causes of ascites

I. Portal hypertension
   - A. Cirrhosis
   - B. Hepatic congestion:
     - . Congestive heart failure.
     - . Constrictive pericarditis.
     - . Budd-Chiari syndrome.

II. Hypoalbuminaemia:
   - A. Nephrotic syndrome.
   - B. Protein-losing enteropathy.
   - C. Malnutrition.

(Cont.)

III. Infection:
- Bacterial (Tuberculous)
- Fungal (Candida, Histoplasma)
- Parasitic (Schistosoma & Enterobius)

IV. Neoplasms:
- Primary mesothelioma
- Secondary carcinomatosis

V. Miscellaneous:
- Bile ascites
- Pancreatic ascites due to ruptured duct or pseudocysts
- Chylous ascites
- Myxoedema
- Ovarian diseases
- Eosinophilic ascites

There are rare causes of ascites including hypothyroidism and systemic lupus erytheromatosis (Wilkinson, 1979 and Mier, 1985). Moryan (1985), reported a case of ascites occurring early in the course of a patient with myeloma in whom there was no evidence of intra-abdominal plasmacytoma and the skeleton was relatively spared. Also Stocker (1985), has reported a case of congenital cytomegalovirus infection presenting with massive ascites.

In 1985, Berchuck reported a case of cervical adenoid cystic carcinoma associated with ascites.

Wilkins (I985), reported cases of S.L.E. which presented as massive ascites, and Mier (I985), also reported cases of S.L.E. with ascites in the absence of nephrotic syndrome, congestive heart failure or hepatic cirrhosis. There were three primary omental lieomyosarcoma which have been described to date and all were presenting as haemorrhagic ascites (Dixon, I984).

Also, Wheeler (I985), has reported that although bloody ascites due to sarcoidosis is rare, two patients in whom sarcoidosis was manifested as bloody ascites were reported, while five patients with non bloody ascites were diagnosed as abdominal sarcoidosis.

On the other hand, Rowland (I985), reported patients with E. coli spontaneous bacterial peritonitis with an 80% eosinophilia in the peritoneal fluid and no peripheral oesinophilia; they gave good response for antimicrobial therapy.

B. Pathogenesis & Dynamics Of Ascites

Practically, the determination of pathogenesis of ascites is a far more complex problem. Although the basic elements of Starling's equilibrium - portal hypertension and hypoalbuminaemia are valid, many additional factors participate. Many cirrhotic patients with portal hypertension do not have ascites and sometimes portal hypertension and hypoalbuminaemia occur together without ascites (Leon, 1982).

Wilkinson (1979), suggested that in the mechanism of ascites formation, both local factors and changes in renal function have to be considered. The local factors involve the raised pressure at the venous end of the splanchnic capillaries due to portal hypertension and a reduced plasma albumin concentration will both favour transudation of fluid into the peritoneal cavity.

Madden (1954), assumed that portal hypertension is the result of the marked alteration of the hepatic microcirculation and macrocirculation induced by cirrhosis and the marked decrease in the number of hepatic

venous radicals in cirrhotic liver resulting in outflow block.

The relationship between portal pressure and ascites is poor. For example, a group of patients with ascites were found to have lower portal pressure than a group without ascites (Teague, I966). However, Atkinson (I96I), found that the measured changes in portal pressure and plasma albumin concentration alone were sufficient to account for ascites formation, but on the other hand, Chenick (I960) and Witte (I969), found the correlation to be less close.

However, Wilkinson (I979), reported that it is not possible to predict accurately the likelihood of ascites from the portal hypertension and hypoalbuminaemia alone. Indeed, a patient with cirrhosis may have hypoalbuminaemia, portal hypertension and even oedema of lower limbs without clinically detectable ascites (Shearman, I982).

In cirrhosis, each day 5 - 20 gm. of albumin leave the plasma compartment and appear in the ascitic fluid; conversely about 2 - 15 gm. of albumin