Ascites with Renal Failure: Prevalence and Management

Thesis

Submitted in Partial Fulfilment of
M.D. Degree in *Internal Medicine*

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1992

Acknowledgement

I would like to express my deepest gratitude and appreciation to Dr. WAHID MOHAMED EL-SAID, professor of internal medicine and Head of Nephrology Department, Ain Shams University, for his indispensable guidance and his invaluable suggestions that greatly enriched this work. His parental attitude and moral support cannot be regarded enough by words.

I am much obliged to Dr. FADILA HASAN SABRY, professor of clinical pathology, Ain Shams University, for her continuous, generous and sincere contributions to this work.

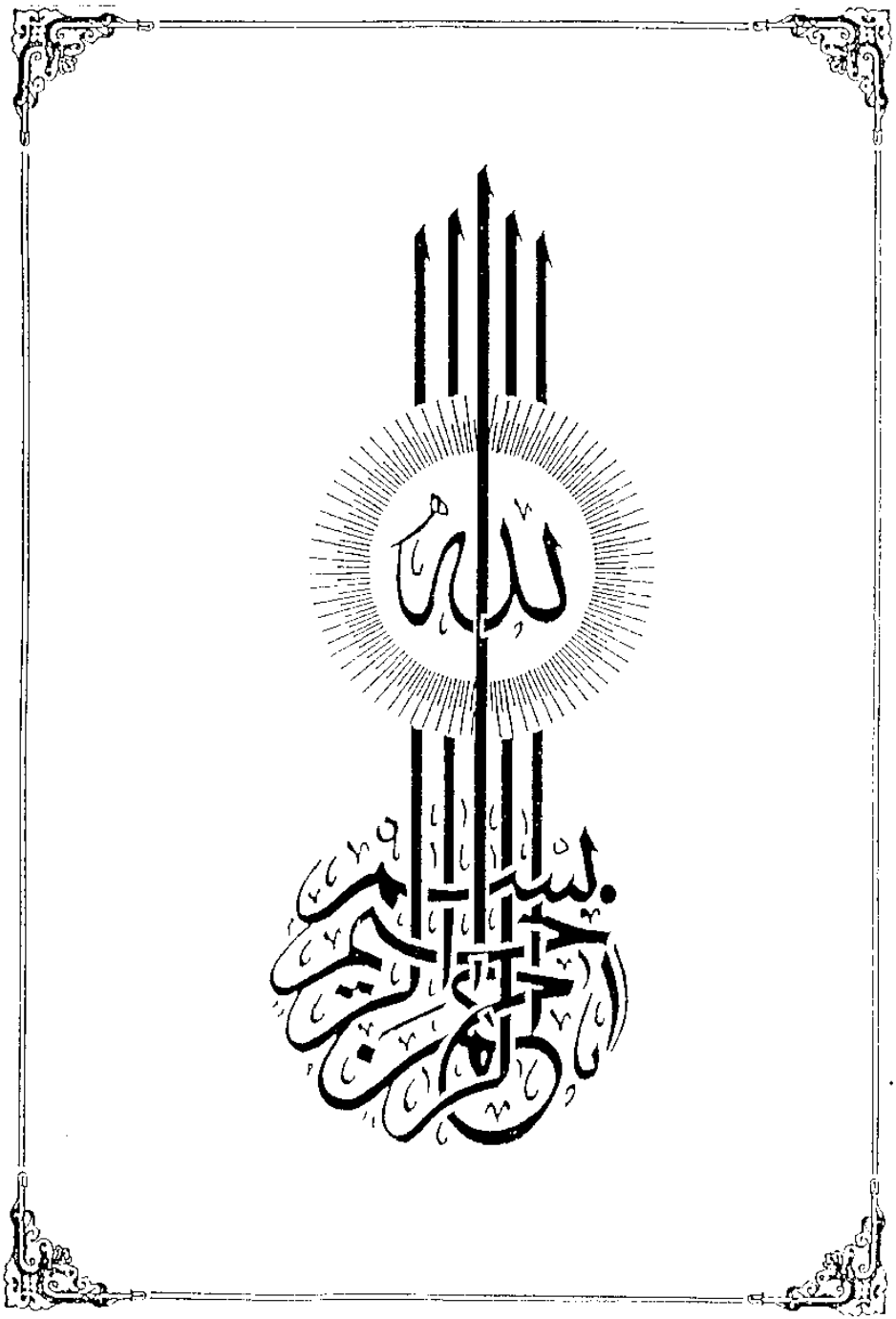
I am very grateful to Dr. MAHMOUD ABDEL FATAH ABDULLA, assistant professor of internal medicine and nephrology, Ain Shams University, for is kind support, valuable supervision, expert advice and his sincere efforts throughout the whole work. Indeed, this study could not have been done without his constant and close support.

I am indebted to Dr. MAMDOUH GHONEIM , assistant professor of radiodiagnosis, Ain Shams University, for his guidance and help in different radiodiagnostic methods.

I am grateful to Dr. HANI ALI REFAAT , assistant professor of internal medicine Ain Shams University, for his advice and guidance.

I also wish to express my appreciation to Dr. ALI MOSSA ALI and Dr. MAHMOUD MOHAMED ALI ZAKI, lecturers of internal medicine, Ain Shams University, for their generous co-operation and continuous help in the practical part of the work.

I want to extend my thanks and gratitude to all colleagues in nephrology unit in Ain Shams University Hospital for their assistance and help throughout the work.



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INTRODUCTION AND AIM OF THE WORK

Nephrogenic ascites was first described in 1970 as refractory ascites associated with end-stage renal disease *(Cinque & Lettori 1970 and Gutch et al., 1970)*. The pathogenesis of nephrogenic ascites remains unexplained and the prognosis remains uncertain *(Mauk et al., 1988)*. The reported frequency varies between 0.7 to 26% of patients on hemodialysis *(Gotleib & Servadio 1976 and Gluck & Nolph, 1987)*. Many forms of treatment have been tried.

The aim of this work is to:

1. Define the prevalence of nephrogenic ascites.
2. To study the possible etiologic factors and,
3. To compare some of the modalities of treatment.

Review of Literature

REVIEW OF LITERATURE

The Pritoneum and the Peritoneal Cavity

The peritoneum is a living membrane that covers visceral organs forms the visceral mesentery that connects loops of bowel, and reflects over the inner surface of the abdominal wall *(Cunningham, 1926)*.

The peritoneum is continuous and forms a closed sack, which, because the space within contains only small amounts of fluid (probably less than 100ml), usually is only collapsed. In an adult of normal size, the space can be enlarged by instillation of fluid, two or more liters of fluid can be accommodated without causing discomfort. The surface of the member is a shiny layer of mesothelial cells, beneath which lie supporting interstitium containing exteracellular fluid, connective tissue fibers, blood vessels, and lymphatics. The visceral peritoneum is that part of the membrane that courses over the surfaces of the visceral organs. As visceral peritoneum reflects from loops of bowel to form the visceral mesentery, the interstitium becomes interspersed between adjacent mesothelial layers. The parietal peritoneum is that portion of the membrane that covers the inner surface of the abdominal wall. The total surface area of the peritoneal mesothelium (parietal and visceral) is believed to approximate the surface area of the skin, which, in most adults, is 1 to 2 m^2 *(Henderson, 1976)*.

Peritoneal Lymphatics

The anatomy of the peritoneal lymphatics has been reviewed by *Khanna et al. (1986)*. The lymphatics which probably have the greatest impact on peritoneal transport kinetics are those immediately below the mesothelial

layer on the caudal surface of the diaphragm. Mesenteric lymphatics drain fluid and nutrients from the intestine, diffusive and convective movements of solutes and water from mesenteric lymphatics into the peritoneal cavity may occur during peritoneal dialysis. The extent of such lymphatic contribution is unknown but assumed to be small. In contrast, diaphragmatic contractions may actively pump fluid from the peritoneal cavity into and through the diaphragmatic lymphatics and mainly via the right lymphatic duct into the venous circulation.

On the under surface of the diaphragm there are intercellular gaps or stomata located between lateral borders of the mesothelial cells that overlie lymphatic lacunae *(Yoffy & Courtice, 1970)* . Submesothelial connective tissue is interrupted at the site of the stoma and contains masses of microfibrills. Also the cytoplasm of mesothelial cells and lymphatic endothelial cells have abundant fine filamints *(Tsilibary & Wissig, 1979)*. In 1983 the same authors demonstrated that when the diaphragm is relaxed with succinylcholine, numerous stomata are observed, and when the diaphragm is contracted with carbacol, the stomata are less apparent and often appear nonpatent. Flaps of mesothelial and underlying lymphatic endothelial cells may influence the patency of the stomata based on the status of the actin components of the cytoplasm of these cellular extentions.

Lymphatic endothelium forms a continuous layer in the roof of each lacuna. It appears that overlapping lymphatic endothelial cell junctions may separate to allow passage of cells or particles into the lymphatic vessels and they may act as flap valves preventing the passage of the lacunae contents back into the peritoneal cavity *(Casley-Smith, 1964)*. The diaphragm and

lymphatics therin act as pumps with the mesothelial and endothelial flaps as "inlet valve". The flow of fluid from the peritoneal cavity into and through the system would thus represent a convective transport system with little or no molecular size discrimination *(Flessner et al., 1984; Flessner et al., 1985 and Flessner & Fenstermacher et al., 1985).*

ASCITES

Ascites was defined by *Thomas La Mont et al. (1980)* as the accumulation of excessive volumes of fluid within the peritoneal cavity *(Gr. Askites, from askos bag).*

History

Ascites was discussed in some of the earliest medical literatures *(Dawson, 1960).* Hippocrates recognized an association between liver and dropsy, Celsus detailed the methods of paracentesis most likely to succeed; and Erasistratus of Alexandria postulated that ascites was due to stonelike hardness of the liver, commenting that "the blood is prevented from going forward into the liver owing to narowness of the passages". *Lower (1679)* initiated the investigative approach, producing ascites in dogs by ligating the vena cava above the liver, *Flint (1863)* first described functional renal failure as a complication of ascites in hepatic disease.

Murchison (1885) had listed physical findings in ascites including progressive abdominal enlargement, eversion of hernia of the umbilicus, exertional dyspnea and thoracic breathing. He drew attention to the importance of percussing for dullness over the flanks with the patient in supine position and of evoking the shift of dullness in response to gravity as

the patient assumed lateral or knee-elbow position. The method of eliciting a fluid thrill also was described, together with tendency of the umbilicus to approximate more closely the pubic symphysis than the sternum, in contrast to upward displacement of the umbilicus by overian cyct.

Scientific medicine has since generated extensive data bearing on ascites, but greater precision in diagnosis, knowledge of pathogenesis and improvement in treatment still constitute major challenge.

Consequences of Ascites *(Dworken, 1982)*

Ascites causes adverse effects largely through mechanical alterations related directly to the volume and mass of the accumulated fluid.

The major consequences of ascites are: cosmotic aberrations, changes in the truncal center of gravity, aggravation of incipient hernia, reduced capacity for food, respiratory embarrassment, and masking of other intra-abdominal pathology.

Cosmotic aberrations refer principally to the concern that their bodily contours are changing unfavorably. This is a fortunate observation, because it usually leads to early recognition and treatment of the underlying disorders.

Changes in the center of gravity caused by ascites will often force the patients to lean backward as they walk, thereby placing added stress on the vertebral column. It is not a serious problem, but recognition of the unusual posture will occasionally lead to early diagnosis of ascites.

Incepient hernias in the inguinal and umbilical areas are commonly enlarged by the increased abdominal pressure from ascites. Scrotal and penile edema are common concomitants. Umbilical hernias may become massive

and resemble the contoures of an elephant's trunk. Tight stretching of the skin overlying umbilical hernia may lead to pressure necrosis and to spontaneous tearing of the skin, with sudden external drainage of ascites, but adverse consequences are rare.

Reduction in food capacity is a serious complication and may lead to the superficially paradoxical clinical picture of a person who, while eating little, continues to gain weight. One notes marked decrease in muscle mass, particularly in the upper extremities, shoulder girdles, neck, face, and chest. The patient looks cachectic above the diaphragm and massively obese below. Muscular weakness is prominant, and the situation is self-perpetuating. Prompt treatment of the ascites can, if successful, lead to increased caloric intake and improvement in muscle mass.

Respiratory embarrassment is a direct result of diaphragmatic elevation from ascites, with lowered lung compliance decreased vital capacity, hyperventilation, and occasionally respiratory alkalosis.

Masking of other intra-abdominal pathology, such as acute appendicitis or pancreatitis, is an occasional consequence of ascites. The parietal peritoneum, being separated from the inflamed abdominal viscus, does not become inflamed promptly, and localizing symptoms and signs do not developed. One has to rely often on the appearance of fever and leucocytosis to confirm clinical impressions.

Diagnosis of Ascites

As mentioned by *Bender (1985)* , small amounts of ascites may be asymptomatic, but as it increases the patient becomes aware of abdominal

distention and a sense of fullness and discomfort. Larger amount of ascites, especially if the abdomen is tensely distended, may cause respiratory distress, anorxia,, nausea, early satiety, or frank pain. Body weight may vary, depending on the state of nutrition and the underlying disease process. On physical examination the flanks bulge, and a fluid wave may be demonstrable. Shifting dullness is somewhat more sensitive but may be non-specific.

Although, it is difficult to detect less than 1.5 to 2 liters of fluid, placing the patient on his hands and knees and perccussing flatness over the dependent abdomen (puddle sign) may demonstrate smaller amounts *(Lawson and Weissbein, 1959)*.

Radiologically, ascites may be manifested by abdominal haziness, separation of bowel loops, or widening of the flank stripe on plain abdominal film. Ultrasonography and computed tomography may be useful in demonstrating relatively small amounts of peritoneal fluid and specially in distinguishing free fluid from cystic masses.

Diagnostic parcentesis *(Glickman & Isselbacher, 1980)*: 50, to 100ml of ascitic fluid should be examined from it's gross appearance, protein content, cell count, as well as Gram's and acid fast stains and culture. Cytologic examination may disclose an otherwise unsuspected carcinoma. The characteristics of ascites fluid in various disease states are shown in table (A).

Causes of Ascites

Although a wide variety of disease processes may be associated with the presence of ascites, more than 90% of patients with this complication are

found to have cirrhosis, congestive heart failure, neoplasm or tuberculosis. For purposes of classification, causes of ascites may be divided into two main groups. First group are disease not involving the peritoneum, second group are diseases of the peritoneum.

1. ***Causes of Ascites not Associated with Peritoneal Disease (Bender & Ockner, 1983)***

I. Portal hypertension
   - A. Liver cirrhosis
   - B. Hepatic congestion:
     1. Congestive heart failure.
     2. Constrictive pericaditis.
     3. Inferior vena cava obstruction.
     4. Hepatic vein obstruction (Budd-Chiari)
   - C. Portal vein occlusion

II. Hypoalbuminemia
   - A. Nephrotic syndorme.
   - B. Protein-loosing entropathy.
   - C. Malnutrition.

III. Miscellaneous:
   - A. Myxedema
   - B. Ovarian disease.
   - C. Pancreatic ascites.
   - D. Bile ascites.
   - E. Chylous ascites.
   - F. Urine ascites.

G. Nephrogenic ascites.

2. Causes of ascites associates with diseases of the peritoneum *(Modified from Bender and Ockner, 1983).*

I. Infections

A. Bacterial peritonitis.

B. Tuberculous peritonitis.

C. Fungal diseases.

D. Parasitic diseases.

II. Neoplasms

A. Secondary malignancy.

B. Primary mesothelioma.

C. Pseudomyxoma peritonei.

III. Granulomatous peritonitis

IV. Miscellaneous

A. Vasculities.

B. Familial Mediateranean fever.

C. Gynecologic disease.

1. Endometriosis.

2. Dermoid cyct.

D. Peritoneal lymphageictasia.

Relation Between Ascites and Renal Disease

The relation between ascites and kidney disease can be discussed under the following headings.

1. Renal impairment complicating pre-existing ascites.
2. Ascites due to specific pathology associated with renal disease.
3. Nephrogenic (idiopathic) ascites.