ASCITES IN INFANCY AND CHILDHOOD

A THESIS

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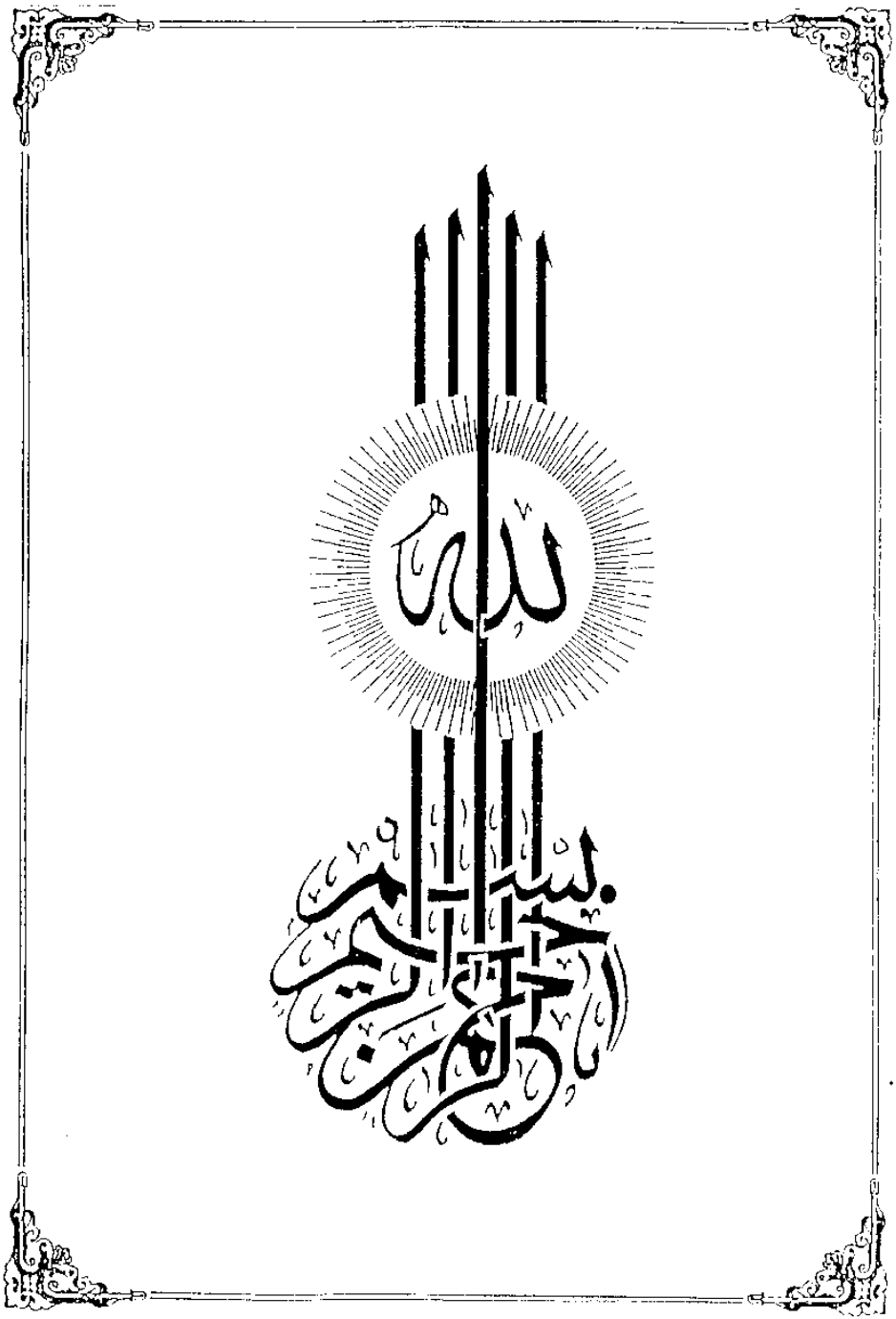

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INTRODUCTION AND AIM OF THE REVIEW

The term "Ascites" is derived from the Greek word askites, meaning bladder or bag. It refers to the accumulation of free fluid within the peritoneal cavity. The peritoneal fluid formation is a dynamic process. The net volume of fluid is the result of an equilibrium maintained between production and absorption.

The intraperitoneal collection of fluid, arises as a result of one or more than three basic mechanisms:

1- Reduction in plasma oncotic pressure.

2- Obstruction to drainage - venous or lymphatic.

3- Irritation of peritoneum by infection, trauma or neoplasia (Forfar et al., 1978).

Ascites in childhood is usually due to a medical illness, such as nephrosis, heart failure, or cirrhosis of the liver. Peritoneal fluid in a new born or young infant is almost always from a perforation in the genito-urinary, biliary or gastrointestinal tracts. Haemolytic diseases of the new born, Syphillis, peritonitis secondary to ruptured ovarian cyst, and chyle leakage are other rare causes of ascites.

Ascites secondary to congestion or obstruction (transudate) is either of posthepatic (heart failure), or intrahepatic (cirrhosis of the liver), or prehapatic (tumor, compression of the portal vein, or thrombosis of the portal artery) origin. Ascites always arises in a rapidly evolving obstruction.

Ascites occurs in decreased colloid osmotic pressure as a sequel of hypoproteinaemia. Hypoproteinemia can be secondary to insufficient protein production, such as in cirrhosis of the liver. In children it usually results from increased protein losses either through the kidneys (nephrotic syndrome) or through the gastrointestinal tract (protein losing enteropathy). It can occur in all chronic disorders of the gastrointestinal tract whenever plasma protein loss into the intestinal tract exceeds protein synthesis by the liver. This is the case in chronic diarrheas of various etiologies, in malabsorption, such as cystic fibrosis, in severe celiac disease, starvation, Kwashiorkor, or in rare disorders such as the blind loop syndrome, in Ménétrier's disease or in Whipple's disease.

Abdominal distension is the usual presentation of ascites. When distension is great, there is flattening or pouting of the umbilicus. Fluctuation can be detected on palpation; a wavelike impulse is obtained by sharp tapping on one side of the abdominal wall while the other hand is placed on the opposite side of the abdomen and an assistant's hand compresses it in the midline; shifting percussion dullness can often be demonstrated.

Ascites must be differentiated from other conditions that cause distension of the abdomen. These include gaseous distension of the intestines, fecal distension as in

megacolon, tumor masses, including cysts of the mesentery, acute or chronic peritonitis, peritoneal hemorrhage, extreme distension of the bladder, and simple obesity.

The course, prognosis and treatment of ascites depend entirely upon the cause (Nelson et al., 1979).

Aim of the Review:

This is intended to be a review on the subject of ascites most recent point of view as regards the causes, prognosis and management.

This is because ascites is considered a complication of many diseases and therefore knowing the pathology, mechanism of formation of ascites and types of ascites from the most recent researches might add some up to date information to the subject.

HISTORICAL REVIEW

Ascites is discussed in some of the earliest medical literature (Dawson, 1960). Hippocrates (fifth century B.C.) suggested that ascites resulted from the leakage of hepatic fluid into the peritoneal cavity. Erasistratus of Alexandria in the third century B.C. postulated that ascites was due to the stonelike hardness of the liver commenting that "the blood is prevented from going forward into the liver owing to the narrowness of the passages." He also postulated that the "narrowness of the blood vessels going through the liver" was the major factor in peritoneal fluid accumulation. Galen later refuted the Alexandrian and proposed a theory of cold and moist humors which dominated medical thought for the next 12 centuries. Modern studies were initiated in the seventeenth century by Lower (Lower, 1679), an Oxford physiologist, initiated the investigative approach, producing ascites in dogs by ligating the inferior vena cava. Sydenham (1850), in the same century, suggested a relationship between alcohol consumption and ascites. Flint in 1863 first described functional renal failure as a complication of ascites in hepatic disease (Flint, 1863).

Abdominal paracentesis was commonly performed during the time of Erasistratus although he disagreed with the practice. Celsus described methods of paracentesis about twenty B.C., and Paul of Aegina (625-690 AD) wrote a detailed description of the procedure including the warning:

"avoid by all means a sudden evacuation, for some ignorant persons having evacuated the vital spirit with the fluid have immediately killed the patient."

PATHOPHYSIOLOGY OF ASCITES

Osmotic and hydrostatic forces, as described by the Starling equilibrium, control the net flux of fluid (and proteins) between the peritoneal capillary bed and the peritoneal cavity. Movement of materials between the compartments is modified by the peritoneal membrane and influenced by the lymphatic drainage system. In ascites there is net increase in abdominal fluid accumulation but a dynamic equilibrium is maintained between the plasma and the peritoneal cavity. Forty to 80% of the peritoneal fluid is exchanged with the plasma every hour; radiolabelled albumin administered intravenously accumulates linearly in the ascitic fluid for four hours following administration (Dykes and Jones, 1968).

Pathological mechanism resulting in ascites include inflammation, venous, and lymphatic obstruction, decreased plasma colloid oncotic pressure, and a ruptured viscus. Inflammation of the peritoneum promotes capillary permeability and leakage of high-protein fluid into the peritoneal cavity. This increases the colloid osmotic pressure of the intra-abdominal fluid which decreases ascitic fluid absorption. Abdominal viscera drain primarily through the portal venous system. Obstruction of the portal vein causes an increase in portal hydrostatic pressure which may result in intraperitoneal fluid accumulation. Cirrhosis is one common cause of portal vein obstruction, others include obstruction of the hepatic vein of inferior vena cava, and

elevated right ventricular pressure. Prehapatic or presinusoidal portal hypertension is rarely associated with ascites.

Congenital defects of the lymphatic system, mesenteric adenitis, and tuberculosis and neoplasms of the abdomen, retroperitoneum, thorax, and mediastinum have all been associated with the development of chylous ascites. The pathophysiologic mechanisms involved superficially appear to be associated with obstruction or leakage of the thoracic duct, cisterna chyli, or intestinal lymphatic vessels. Experimental observations, however, suggest a more complex relationship.

Hypoalbuminemia with decreased plasma colloid osmotic pressure is characteristic of many disease states. Hepatic, gastrointestinal (with protein-losing enteropathy), and renal diseases may be associated with hypoalbuminemia and ascites. Controversy exists concerning the relative importance of altered albumin metabolism and the disruption of Starling forces. Hypoalbuminemia and even analbuminemia alone are rarely associated with ascites, and there is no statistical difference in the plasma albumin concentration of cirrhotics with or without ascites (Summerskill et al., 1975).

The protein concentration of ascitic fluid in patients with ascites cannot be explained by simple transudation; hence,

other considerations besides the Starling equilibrium have been introduced to explain the accumulation of intraperitoneal fluid associated with portal hypertension and abnormalities of lymph circulation.

A perforated abdominal viscus or cyst may also cause fluid to accumulate in the abdomen. Blunt trauma associated with rupture of the spleen or liver are not uncommon. Neonatal ascites is often associated with urinary tract abnormalities involving a direct communication between the renal collecting systems and the abdominal cavity.

Movements of materials between the abdominal cavity and the circulation are modified by the lymphatic system and mediated by the peritoneal membrane. It is established that the transport kinetics of the peritoneum can be affected by both physical and pharmacologic agents (Shear, 1966). Abnormalities in membrane function might therefore theoretically contribute to ascites formation, but have not been conclusively demonstrated.

Both intra-abdominal and systemic factors are involved in ascites formation. Intra-abdominal factors localize accumulation of fluid mainly within the abdomen, whereas systemic factors cause retention of sodium and water throughout the body. Interaction between both intra-abdominal and systemic mechanisms, with concomitant hormonal, metabolic,