ORIGINAL RESEARCH PAPER
Volume - 11 | Issue - 08 | August - 2022 | PRINT ISSN No. 2277 - 8179 | DOI : 10.36106/ijsr
INTERNATIONAL JOURNAL OF SCIENTIFIC RESEARCH

A STUDY OF 50 CASES OF ASCITES

Medicine

Dr. Pradeep k. Dudhrejiya M. D. General Medicine, Superintendent at General Hospital, Morbi

Dr. Divyesh D. Sherasiya M. D. General Medicine. General Hospital, Morbi

Dr. Himshikha Pandya M.B.B.S. Medical officer at General Hospital, Morbi

ABSTRACT

Aims and objective :

- To study the common and uncommon causes of ascites.
- To study the outcome and prognosis of ascites due to its causative factor.

KEYWORDS

Ascites, Liver Parenchymal Disease, Alcoholic Liver Disease, Dilated Cardiomyopathy, Chronic Renal Failure,

BACKGROUND :

Ascites is the pathologic accumulation of fluid within the peritoneal cavity. It is the most common complication of cirrhosis and occurs in about 50% of patient with decompensated cirrhosis in 10 years. The development of ascites denotes the transition from compensated to decompensated cirrhosis. Mortality increases from complications such as spontaneous bacterial peritonitis and hepatorenal syndrome.

PATHOGENESIS IN THE PRESENCE OF CIRRHOSIS Ascites in patients with cirrhosis is the result of portal hypertension and renal salt and water retention. Similar mechanisms contribute to ascites formation in heart failure. Portal hypertension signifies elevation of the pressure within the portal vein. According to Ohm's law, pressure is the product of resistance and flow. Increased hepatic resistance occurs by several mechanisms. First, the development of hepatic fibrosis, which defines cirrhosis, disrupts the normal architecture of the hepatic sinusoids and impedes normal blood flow through the liver. Second, activation of hepatic stellate cells, which mediate fibrogenesis, leads to smooth-muscle contraction and fibrosis. Finally, cirrhosis is associated with a decrease in endothelial nitric oxide synthetase (eNOS) production, which results in decreased nitric oxide production and increased intrahepatic vasoconstriction. The development of cirrhosis is also associated with increased systemic circulating levels of nitric oxide (in contrast to the decrease seen intrahepatically), as well as increased levels of vascular endothelial growth factor and tumor necrosis factor, that result in splanchnic arterial vasodilation. Vasodilation of the splanchnic circulation results in pooling of blood and a decrease in the effective circulating volume, which is perceived by the kidneys as hypovolemia. Compensatory vasoconstriction via release of antidiuretic hormone ensues; the consequences are free water retention and activation of the sympathetic nervous system and the renin-angiotensin-aldosterone system, which lead in turn to renal sodium and water retention.

PATHOGENESIS IN THE ABSENCE OF CIRRHOSIS Ascites in the absence of cirrhosis generally results from peritoneal carcinomatosis, peritoneal infection, or pancreatic disease. Peritoneal carcinomatosis can result from primary peritoneal malignancies such as mesothelioma or sarcoma, abdominal malignancies such as gastric or colonic adenocarcinoma, or metastatic disease from breast or lung carcinoma or melanoma. The tumor cells lining the peritoneum produce a protein-rich fluid that contributes to the development of ascites. Fluid from the extracellular space is drawn into the peritoneum, further contributing to the development of ascites. Tuberculous peritonitis causes ascites via a similar mechanism; tubercles deposited on the peritoneum exude a proteinaceous fluid. Pancreatic ascites results from leakage of pancreatic enzymes into the peritoneum.

Subjects :

From January 1,2021 to July 15, 2022 a 50 case of ultrasonography confirmed cases of ascites admitted to General hospital, Morbi.

Inclusion Criteria:

All case of USG confirmed ascites with or without liver parenchymal disease of age more than 18 years

Exclusion Criteria:

Pregnant women and patient age less than 18year are excluded from this study.

METHOD :

We performed a retrospective/analytical study to determine various causes and outcome of usg confirmed case of ascites admitted at General hospital morbi. We divide patient in three groups according to their cause (1. due to liver disease (2. due to renal disease (3. due to cardiac disease. Further In group 1 (due to liver disease) again we divide patient in group (a. due to alcoholism (b. due to NASH (c. due to autoimmune liver disease. Treated them according to guideline and Outcome were observed during followup.

Table-1

No. of patients	Male	Female
Alcoholic liver disease (ALD)	28	00
Non-alcoholic steatohepatitis/ fatty liver disease (NASH/NAFLD)	00	04
Autoimmune hepatitis	00	06
Chronic Renal Failure (CRF)	05	03
Dilated Cardiomyopathy (DCM)	04	00
Total	37	13

Table-2

	Total	Discharged	Death
Alcoholic liver disease	28	24	04
Autoimmune hepatitis	06	04	02
Non-alcoholic steatohepatitis/ fatty liver disease (NASH/NAFLD)	04	04	00
Chronic Renal Failure (CRF)	08	07	01
Dilated Cardiomyopathy (DCM)	04	04	00
	50	43	07

CONCLUSION:

From the above study, we reach on conclusion that

1) Male are more commonly suffering from ascites than the female.
2) Most common cause of ascites is liver cirrhosis > CRF > DCM.
3) The most common cause of ascites in male is alcoholic liver disease, 2nd most common is CRF and 3rd most common is DCM, whereas in female the most common cause is autoimmune hepatitis, 2nd most common is NASH, 3rd is CRF.
4) Mortality rate is more in autoimmune hepatitis (33%) than in alcoholic(14%) and CRF (12.5%) so Outcome is poor in autoimmune hepatitis than any other causes.

REFERENCES :

1. Harrison principle of internal of medicine, 21st edition.
2. API Textbook of medicine, 11th edition.
3. Greenberger's CMDT for gastroenterology and hepatology, 4th edition.
4. Schiff's diseases of the liver,
5. Brunward's heart disease: A textbook of cardiovascular medicine, 11th edition.
6. The Washington's manual of Nephrology.