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CLINICAL PROFILE OF ASCITES

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ABSTRACT

Background: Ascites is defined as the accumulation of excess fluid in the peritoneal cavity. Fluid accumulates when it enters the peritoneal cavity from the mesenteries, the peritoneum and hepatic surface at a rate greater than can be returned to the circulation via the capillaries and lymphatics. Objective: To determine the causes of Ascites and the characteristics of adult patients with Ascites in our hospital and the rate of complications like Hepatic Encephalopathy, Spontaneous Bacterial Peritonitis and Upper Gastrointestinal Bleed in our settings. Methods: Blood sugar estimated by Glucose oxidase- peroxidase method. Blood urea estimated by Bertheld method. Serum creatinine estimated by Jaffe's kinetic method. Serum Na+ and serum K+ estimated by flame photometer. ALT estimated by kinetic method- IFCC. Ascitic fluid Glucose estimated by GOD POD method. Ascitic fluid protein estimated by Sulpho-salicylic acid method. 24 hrs Urine protein estimated by sulpho-salicylic acid method.

KEYWORDS: capillaries and lymphatics.

INTRODUCTION

The term ascites is derived from the greek word "ASKOS" (bladder, belly, bag) and denotes the presence of excessive fluid in the peritoneal cavity.[1] Many diseases are known to lead to the formation of free fluid within the peritoneal cavity. Basically the causes of ascites may be grouped into those conditions in which the pathological process does not directly affect the peritoneum and those in which the peritoneum itself is involved. The first group includes diseases associated with sinusoidal portal hypertension, hypoalbuminaemia and a variety of disorders that may cause ascites through different mechanisms, such as myxoedema, ovarian diseases, chronic pancreatitis, biliary-tract leakage diseases affecting the lymphatic system of the splanchnic area and chronic renal failure. In the second group, ascites is formed as a consequence of primary peritoneal disease or as a result of peritoneal involvement in systemic process; tuberculous, fungal parasitic and granulomatous peritonitis, primary or metastatic peritoneal tumours, vasculitis, eosinophilic gastroenteritis, and Whipples disease are the most characteristic causes of ascites in this group.[2]

The evaluation of a patient with ascites requires that the cause of the ascites to be established. In most cases ascites appears as part of a well recognized illness such as cirrhosis, congestive heart failure, nephrosis or disseminated carcinomatosis, in these situations the physician should determine that the development of ascites is indeed a consequence of the basic underlying disease and not due to the presence of a separate or related disease process. This distinction is necessary even when the cause of ascites seems obvious.

Diagnostic paracentesis (50-100ml) should be part of the routine evaluation of the patient, with ascites. The fluid should be examined for its gross appearance, protein content, albumin level, cell count, and differential cell count, should be determined and gram's and acid fast stains and culture should be performed. Cytologic and cell block examination may disclose an otherwise unsuspected carcinoma. Serum ascites albumin gradient (SAAG) should be calculated to determine whether the fluid has features of a transudate or an exudate. The gradient correlates directly with portal pressure, a gradient >1.1 gm/dl, high gradient ascites is characteristic of uncomplicated cirrhotic ascites and differentiates ascites due to portal hypertension > 97% of the time. Other etiologies of high gradient ascites include alcoholic hepatitis, congestive heart failure, hepatic metastasis constrictive pericarditis and Budd chiari Syndrome. A gradient < 1.1 gm/dl (Low gradient) suggests that the ascites is not due to portal hypertension

with > 97 % accuracy and mandates a search for other causes such as peritoneal carcinomatosis, tuberculous peritonitis, pancreatitis, serositis, pyogenic peritonitis, and nephrotic syndrome.

Blood stained fluid with > 2.5gm / dl protein is unusual in uncomplicated cirrhosis but is consistant with tuberculous peritonitis or neoplasm. Cloudy fluid with predominance of polymorphonuclear cells > 250 / micro liter and a positive Gram's stain are characteristic of bacterial peritonitis, which requires antibiotic therapy, if most cells are lymphocytes tuberculosis should be suspected. Chylous ascites refers to a turbid milky or creamy peritoneal fluid due to presence of thoracic or intestinal lymph. A turbid fluid due to leukocytes, or tumor cells may be confused with chylous fluid (pseudochylous) and it is often helpful to carry out alkalinization and ether extraction of the specimen. Alkali tend to dissolve cellular proteins and thereby reduce turbidity, ether extraction leads to clearing if the turbidity of the fluid is due to lipid. Chylous ascites is often the result of lymphatic disruption, or obstruction from cirrhosis, tumor, trauma, tuberculosis, filariasis, or congenital abnormalities. It may also be seen in nephrotic Syndrome.

METHODS

In this study 80 cases were selected randomly in patients who presented with ascites and got admitted in the medical wards of VIMSAR, Burla, Odisha. After clinical Diagnosis, diagnostic paracentesis and ultrasonogram were done to confirm the same in all cases in this study. This study was conducted during the period of two years- October 2017 to October 2019.

The following investigations have been done in all the patients in this study.

Blood complete hemogram, Random Blood Sugar, Blood Urea, Serum Creatinine, Serum Sodium, Serum Potassium, Urine Albumin, Urine Sugar, Deposits, X-ray Chest PA view, ECG, USG abdomen, Liver function tests, Ascitic Fluid Analysis (bio chemical analysis and cytology), UGI Scopy.

Along with Ascites if the clinical picture was suggestive of heart disease, Echocardiogram was done, in nephrotic syndrome, 24hrs urine protein and serum lipid profile done and in case of malignant ascites, ascitic fluid cytology for malignant cells and α feto-protein were looked for.

RESULTS

Table 1: Showing Details Of Symptoms With Which Patients With Ascites Presented.

Symptoms	No. Of Patients	Percentage
Distention of Abdomen	80	100%
Swelling of Lower Limbs	28	35%
Jaundice	20	25%
Fever	16	20%
Abdominal Pain	20	25%
Hematemesis	4	5%
Altered sensorium	4	5%

Table 2: Showing Physicial Findings Amongst Cases Of Ascites.

Physical findings	No. Of Patients	Percentage
Ascites	80	100%
Anemia	60	75%
Icterus	20	25%
Hepatomegaly	16	20%
Splenomegaly	8	10%
Lymphadenopathy	5	6.25%
Edema	48	60%
Palmar Erythema	3	3.75%
Spider Angioma	2	2.5%
Testicular Atrophy	4	5%
Gynecomastia	4	5%
Tenderness of Abdomen	20	25%
Pleural effusion	16	20%
Doughy abdomen	8	10%
Fever	16	20%
Altered sensorium	4	5%
Asterixis	2	2.5%

Table 3: Showing Various Etiologies Of Ascites As Observed Form This Study.

Etiology	No. Of Patients	Percentage
Liver cirrhosis	60	75%
TB Abdomen	8	10%
Chronic Kidney Disease	4	5%
Systemic lupus erythematosus	3	3.75%
Heart failure	2	2.5%
Abdomen carcinoma	2	2.5%
NCPF	1	1.25%

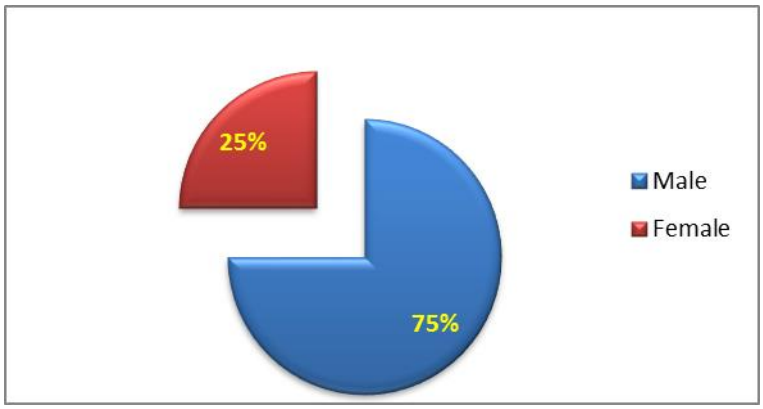

Fig. 1: Showing Sex Distribution Of Patients With Ascities.

Alcoholism is common in males. Alcoholic are more prone to cirrhosis with ascites. Alcoholism is less in females but when they consume alcohol lesser quantities will causes more damaged to liver leading to cirrhosis. In the present study 25% of cases were females.

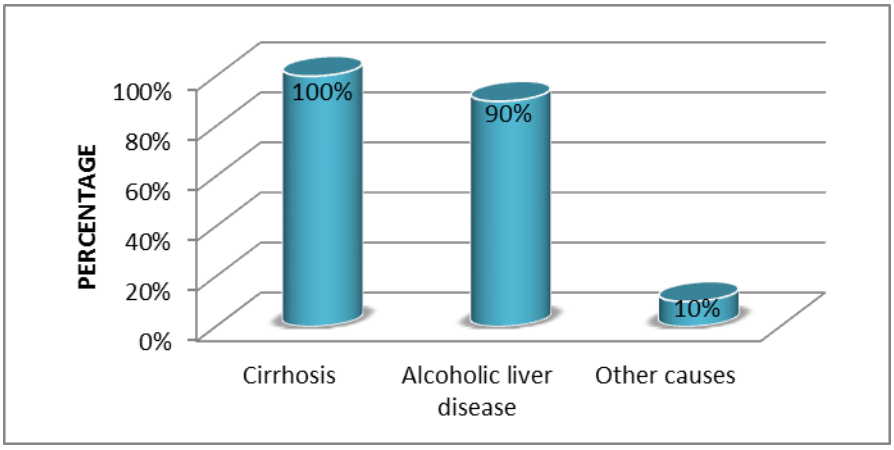

Fig. 2: Showing Number Of Patients In Which Alcoholism Was The Cause Of Liver Cirrhosis As Evidenced From History Of Alcohol Intake And Signs Of Alcoholic Liver Disease.

In this study out of 60 patients with SAAG >1.1 maximum were due to liver cirrhosis. Among the 80 patients with cirrhotic ascites which was confirmed by USG 16 patients were found positive for SBP, in this 13 patients had only PMN count >250/mm^3 but culture negative for any organism. Only 3 subjects were culture positive and isolated E.coli in 2 cases and in one cases Staphylococcus aureus organism by 72hrs, by culture. All the positive cases were males.

Table 4: Showing clinical signs in correlation with sbp.

Signs	No. Of patients	Positive for SBP	Percentage
Janudice	20	7	35%
Fever	20	9	45%
Abdomen tenderness	20	12	60%
Altered sensorium	4	4	100%

Comparing signs and incidence of SBP 20 patients had abdominal tenderness out of these 12 patients were positive for SBP. Out of 20 patients with fever 9 patients came out to be positive for SBP. Out of 4 patients with altered sensorium all of them were positive for SBP.

Table 5: Showing Incidence Of Various Complications In Ascites.

Incidence of complications	Total Patients	No. Of patients	Percentage
SBP	80	16	20%
Hematemesis	80	4	5%
Hepatic Encephalopathy	80	4	5%

Out of 80 patients in our study 16 patients developed spontaneous bacterial peritonitis, out of which 13 patients were positive by PMN>250/mm,[3] and 3 patients were positive by culture of ascitic fluid. 4 patients developed hematemesis out of the total patients. 4 patients developed Hepatic encephalopathy, all 4 of these had altered sensorium on examination and 2 patients had asterixis. Out of the studied cases 1 patient of chronic kidney disease was also HIV positive. In our study 2 patients of liver cirrhosis had hepatitis B as the underlying cause. Out of the studied cases 1 patient of chronic kidney disease was also HIV positive. In our study 2 patients of liver cirrhosis had hepatitis B as the underlying cause.

DISCUSSION

In this study of 80 cases of ascites, the etiology and its incidence is observed as Cirrhosis with portal hypertension was the most common cause for Ascites and Tuberculous peritonitis was the second most common cause of ascites. This coincides well with the following two studies: The study of Runyon BA, Montano AA, Akriviadis EA et al,[35] the etiology and its incidence for ascites is as Cirrhosis with portal Hypertension 85 %, Miscellaneous portal hypertension 8 %, Cardiac disease 3 %, Peritoneal Carcinomatosis 2 %, Miscellaneous normal HT related disorders 2 % and in the Study of Vicente Arroyo, Pere Gines, Ramon planas, Juan Rodes et a,[l2] the etiology and its incidence for ascites is as follows, hepatic cirrhosis 88 % neoplasms 6% and to a lesser extent congestive heart failure 3 %, tuberculous peritonitis 2 % and other 1 %. In our study of the 60 cases of cirrhosis with portal hypertension, 48 cases were male and 12 cases were female and out of the 48 male cases 44 cases were alcoholics: alcoholism is the commonest cause for cirrhosis with portal HT in male. This coincides well with the study of the Tuyns A Pequignot G: Greatest risk of ascitic cirrhosis in males in relation to alcohol consumption Int J Epidemiol 13:53, 1984.[40]

Our study shows along with ascites, pleural effusion was present in 16 cases (20%) of ascite out of which 12 cases presented with Right sided effusion (75 %), 2 case presented with Left sided effusion (12.5 %), 2 case presented with Bilateral effusion (12.5 %). In the study of Leuallen EC, Carr DT, [36] 4.8 % cases of cirrhosis with portal HT were having Pleural effusion and majority of cases 90 % were having Right sided Pleural effusion, 7 % were having Bilateral effusion 3 % having Left sided pleural effusion.

In cirrhosis with Portal hypertension, serum protein ranges between 2 - 6 grams. 75.6 % have 4 - 6 grams, 19.5 % have 2 - 4 grams, 5 % have > 6 grams, and 5 % cases have normal protein value. In the study of Runyon total ascitic protein concentration ranges between 0.5 grams and more than 6 grams and is greater than 3 grams in up to 30 % of patients with other uncomplicated ascites.[37] In the study of Runyon, the proportions of albumin and globulin in the total protein concentration are approximately 45 and 55 % respectively and the value ranges between 0.225 grams to 2.7 grams.

In cirrhosis with Portal hypertension, serum albumin values ranges between 1.1 grams to 3.3 grams. In the study of Runyon, the proportions of albumin and globulin in the total protein concentration are approximately 45 and 55 % respectively and the value ranges between 0.225 grams to 2.7 grams.[37]

In this study in 60 patients of cirrhosis with Portal hypertension 52 cases (87%) were having SAAG value more than 1.1, 8 cases (13 %) were having SAAG value less than 1.1. This coincides with the study of Runyon BA, Montano AA, Akriviadis EA et al where SAAG value was more than 1.1 in 97 % cases of Cirrhotic ascites and less than 1.1 in Non cirrhotic ascites.[35]

In this study, 2 cases of ascites were caused by heart failure from which 1 cases was caused by DCM and 1 cases was caused by Rheumatic heart disease. In the study of Eugene Braunwald, CAHD followed by RHD are the common causes of heart failure.[9]

16 patients of Ascitic patients had Spontaneous Bacterial Peritonitis out of 60 Cirrhotic Ascitic cases presently studied from which 13 patients had only PMN count >250/mm3 but culture negative for any organisms. Only 3 patients were culture positive and isolated E.coli and Staphalococcus aureus organism on culture by 72hrs.

The common mode of presentation of SBP in our series was with fever, vomiting, altered sensorium and abdominal tenderness. Among 20 patients presented with fever 45% of cases had SBP and all the 4 patients presented with altered sensorium was positive for SBP. In Mihas AA, Toussaint J,[10] study the clinical features were fever (69%) range and abdominal pain range (59%). include hepatic encephalopathy (54%),

abdominal tenderness (49%), diarrhea (32%).

In our study 2 MNB cases (66%) was positive for E.coli and another case cultured Staphylococcus aureus (33%) which isolated in first 72 hours. In the study by Runyon et al,[74] E.coli was responsible for 27.3% of cases of SBP and Staphylococcus aureus for 6.8%, while Wilcox et al demonstrated Escherichia coli as the culprit in 45% cases and Staphylococcus aureus in 12% cases. In a study by A P Jain,[68] isolated 44.44 % growing coagulase positive stayphalococcus aureus. Rest grown E. Coli, pseudomonas and kleibseilla.

Among the 16 patients positive for SBP there was only 1 mortality was seen Rest of the SBP cases responded to antibiotic regimen. Among the rest 15 patients none of them were re admitted in our hospital in this period of 2 years.

Incidence of various complications as observed in our study was 20% incidence for development of SBP (16 cases), 5% each for hematemesis and hepatic encephalopathy (4 cases each).

Out of 80 cases, 1 patient was HIV positive and 2 patients were positive for Hepatitis B and the cause of cirrhosis in these 2 patients was the same.

LIMITATIONS

- As the sample size was smaller, it could have affected our observations and conclusions.
- Due to limited resources in our setting, we could not do CT angiography, MR angiography to localise the vessels.
- As the study was done only in our centre, there may be regional variations in etiology and also management strategies for which we cannot generalise to the whole population

CONCLUSION

This study shows Cirrhosis with portal hypertension was the most common cause for ascites (75 %) and the next common cause for ascites was TB abdomen (10 %) followed by renal diseases (5 %) – chronic kidney disease, nephrotic syndrome and other rare causes including peritoneal carcinomatosis, portal vein thrombosis, budd-chiari syndrome all together were 10 % only. In this study portal hypertension was present in all the case of ascites due to cirrhosis, as evidenced by oesophageal varices in UGI scopy, where as hypoproteinaemia was present only in 87 % which shows portal hypertension is the major cause for ascites in Cirrhosis. In cirrhosis with portal hypertension alcoholic liver disease was the commonest cause (90 %). In this study malignant ascites was only 2 cases 1 due to uterine malignancy another one due to hepatocellular carcinoma. The rate of complications for SBP, hematemesis, hepatic encephalopathy was 20%, 5%, 5% respectively.

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