# FREQUENCY OF CAUSES OF ASCITES IN PATIENTS ADMITTED AT MEDICAL UNIT OF A TERTIARY MEDICAL CARE FACILITY

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**Background:** Ascites can be defined as accumulation of free fluid in the peritoneal cavity. It is the most common complication of cirrhosis and is associated with a poor quality of health, increased risks of infections, renal failure and a poor long-term outcome. This descriptive analytical study was conducted on one hundred and fifty diagnosed patients of ascites consecutively admitted in medical unit of tertiary care facility attached to Muhammad Medical College, Mirpur Khas, Sindh, from Oct 2006 to Sep 2008. **Methods:** Special proforma was prepared containing patients' basic information, history, clinical findings and investigations necessary to diagnose the cause of ascites. Patients with ascites due to perforation and intraperitoneal bleeding were excluded from the study. Serum-ascitic albumin gradient (SAAG) was calculated and patients were grouped into high and low SAAG groups. The obtained data was analysed using SPSS-11. **Results:** In this study 150 patients of ascites were included. Patients were arranged in two groups. High SAAG 'high portal pressure' group and low SAAG 'low portal pressure' group. In high SAAG group patients included were 128 (85.33%), and in low SAAG group patients included were 22 (14.66%). In high SAAG group, out of 128 (85.33%) patients 122 (81.33%) were of cirrhotic ascites, [(viral hepatitis B, C and combined 105 (70%), alcoholic 7 (4.66%), cryptogenic 10 (6.66%)], heart failure ascitic patients were 5 (3.33%), and constrictive pericarditis 1 (0.66). In low SAAG group out of 22 (14.66%) patients malignant ascites was (primary peritoneal carcinomatosis and metastasis) 11 (7.33%), tuberculous ascites was 10 (6.66%), and ascites due to nephrotic syndrome was 1 (0.66%). **Conclusion:** As large number of cases of ascites are due to cirrhosis of liver that has little or no cure in developing countries. Media and NGO's should further work to increase awareness of this deadly problem.

**Keywords:** Ascites, SAAG, cirrhosis

## INTRODUCTION

In medicine ascites can be defined as accumulation of free fluid in the peritoneal cavity. Ascites is the most common complication of cirrhosis and is associated with a poor quality of health, increased risks of infections, renal failure and a poor long-term outcome.[1] Cirrhosis, most frequently caused by hepatitis C or alcoholism, was the 12th leading cause of death in the United States in 2000, accounting for more than 25,000 deaths.[2] Currently, pegylated interferon and ribavirin produce sustained viral remission in only 50% of patients. Additional agents are needed to increase the cure rate. In vitro experiments show strong antiviral effects of fluvastatin against hepatitis C virus. Fluvastatin used as monotherapy in vivo showed suppressive effects of HCV that are modest, variable, and often short-lived. Statins, fluvastatin in particular, appear to be safe for use in hepatitis C.[3] Over a million persons die annually due to HBV related complications around the world.[4,5] In patients with one type of viral hepatitis, other viral infections should be sought as they share a common mode of spread and may affect the overall response to treatment.[6] Mild ascites is hard to notice, but severe ascites leads to abdominal distension. Peritoneal fluid of less than 500 ml is difficult to detect clinically, and ultrasound is useful in defining small amounts of ascites. Patients with ascites generally complain of progressive abdominal heaviness and pressure as well as shortness of breath due to mechanical impingement on the diaphragm. Ascites is detected on physical examination of the abdomen by visible bulging of the flanks in the reclining patient 'flank bulging', 'shifting dullness' or in massive ascites with a 'fluid thrill'. Extra-abdominal signs of ascites may be present due to its underlying etiology. For instance in portal hypertension, patients may have leg swelling, bruising, gynecomastia, hematemesis, or mental changes due to encephalopathy. A study[7] has documented that in current clinical practice the classical signs of cirrhosis expected to be present in advanced cases are observed quite infrequently and therefore the diagnosis of cirrhosis should be supported by other means including ultrasound examination of the abdomen etc. Another study shows that the frequency of some of the clinical features of cirrhosis likes jaundice, pyrexia, spider angiomas, gynecomastia, palmer erythema and dupuytren`s contracture is different in our population as compared to that reported in western literature probably due to difference in etiology of cirrhosis.[8] Both ALT/AST ratio reversal (AST/ALT >1) and prolonged prothrombin time are separate indicators of hepatic cirrhosis. The high positive predictive value

here shows that almost all the patients with reversed ratio and prolonged PT will have cirrhosis.[9] Further it was found that the platelet count less than $65\times10^3/mm^3$, serum albumin less than 2.2 g/dl and portal vein diameter more than 13 mm on ultrasound are independent and significant predictors of oesophageal varices on endoscopy.[10]

Ascites is a common problem seen in medical wards. Those patients with ascites due to malignancy may complain of chronic fatigue or weight loss. Those with ascites due to heart failure may also complain of shortness of breath as well as wheezing and exercise intolerance. Ascites exists in three grades: Grade-1: mild, only visible on ultrasound; Grade-2: detectable with flank bulging and shifting dullness; and Grade-3: directly visible, confirmed with fluid thrill. Refractory ascites, which occurs in 5 to 10 percent of patients with ascites, is defined as a lack of response to high doses of diuretics (400 mg of spironolactone plus 160 mg of furosemide per day). Also patients in whom lower doses of diuretics produce recurrent side effects like hepatic encephalopathy, hyponatremia, hypokalemia or azotemia are considered to have refractory ascites.[11] After clinical diagnosis, diagnostic paracentesis help in further breakdown of various causes of ascites. The fluid is reviewed for its gross appearance, protein level, albumin, and cell counts. Additional tests will be performed if indicated such as Gram stain and cytology. The Serum-ascites albumin gradient (SAAG) is probably a better discriminant than older measures (transudate versus exudate) for the classification of ascites.[12] A high gradient (>1.1 g/dL) indicates that ascites is due to portal hypertension. A low gradient (<1.1 g/dL) indicates ascites of non-portal hypertensive aetiology. The concept of serum ascites-albumin gradient (SAAG) differentiates the ascites into portal hypertensive and non-portal hypertensive etiology.[13] Causes of high SAAG ('transudate') are; Cirrhosis, Heart failure, Budd-Chiari syndrome or veno-occlusive disease, Constrictive pericarditis & Kwashiorkor. Causes of low SAAG 'exudate' are: Malignancy, Tuberculosis 2%, Pancreatitis 1%, Serositis, Hereditary angioedema[14]; chylous ascites is sometimes caused by trauma, abdominal surgery, tuberculosis, or another peritoneal infection, it is usually a symptom of lymphoma or some other malignancy. Ascites due to hypothyroidism and renal disease and ascites due to systemic lupus erythematosus are rare disorders.[15]

## PATIENTS AND METHODS

This descriptive analytical study was conducted on one hundred and fifty patients of ascites admitted in medical unit of tertiary care facility attached to Muhammad Medical College (MMC), from Oct 2006 to Sep 2008. Special proforma was prepared containing the basic information of patients. Patients' history including history of jaundice and alcoholism, and clinical findings were recorded. Relevant investigations to diagnose the cause of ascites were carried out including blood complete count, urine detailed report, blood sugar, blood urea and serum creatinine, liver function tests, serum proteins, coagulation profile, viral serology for Hepatitis B, C and D, alpha fetoprotein, ascitic fluid albumin, ascitic fluid culture and sensitivity in suspected cases of peritoneal infection, ascitic fluid for malignant cells in case of suspected malignancy, ultrasound and Doppler examination of abdomen, X-Ray chest PA view, sputum for AFB and Mauntoux test was done in suspected cases of tuberculous ascites, CT scan abdomen in suspected cases of abdominal malignancy and electrocardiogram and echocardiography in suspected cases of heart failure. Upper gastrointestinal endoscopy was done to diagnose the oesophageal varices and malignancy. SAAG was calculated and patients were grouped into high and low SAAG groups. The data was analysed using SPSS-11. Criteria to diagnose cirrhotic ascites were clinical features of cirrhosis of liver, high SAAG ascites, increased echotexture of liver and splenomegaly on ultrasound examination. Criteria to diagnose malignant ascites were low SAAG ascites, absence of cirrhosis of liver, malignant cells in ascitic fluid, and imaging techniques and endoscopy to find out primary malignancy. Criteria to diagnose tuberculous ascites were absence of cirrhosis of liver, low SAAG ascites, excess of lymphocytes in ascites and other techniques as X-Ray chest, sputum for AFB and Mauntoux test.

## RESULTS

Out of 150 patients of ascites in this study 105 (70%) were male and 45 (30%) were female. Their ages were between eighteen and seventy years. Mean age was thirty seven years. 90 (60%) patients were < 40 years of age and 60 (40%) patients were > 40 years of age. Patients were arranged in two groups. High SAAG 'high portal pressure' group and low SAAG 'low portal pressure' group (Table-1). In high SAAG group, out of 128 (85.33%) patients 122 (81.33%) were of cirrhotic with ascites, [viral hepatitis B, C and combined 105 (70%), alcoholic 7 (4.66%), cryptogenic 10 (6.66%)], heart failure ascitic patients were 5 (3.33%) and constrictive pericarditis 1 (0.66). In low SAAG group out of 22 (14.66%) patients; malignant ascites was present in 11 (7.33%) patients, tuberculous ascites in 10 (6.66%) and ascites due to nephrotic syndrome in 1 (0.66%) (Table-1).

**Table-1: Causes of High SAAG (HSA) and Low SAAG (LSA) Ascites (n=150)**

| Variables | n | % |
|---|---|---|
| **Causes of high (>1.1 SAAG) 'high portal pressure, transudate ascites' n=128 (85.33%)** | | |
| **Cirrhosis** | **122** | **81.33** |
| viral hepatitis B, C and coinfection | 105 | 70.0 |
| alcoholic | 7 | 4.66 |
| cryptogenic | 10 | 6.66 |
| **Heart failure** | 5 | 3.33 |
| **Constrictive pericarditis** | 1 | 0.66 |
| **Budd-Chiari syndrome or veno-occlusive disease** | 0 | 0 |
| **Myxedema** | 0 | 0 |
| **Causes of low (<1.1 SAAG) 'low portal pressure, exudate ascites' n=22 (14.66%)** | | |
| **Malignancy (primary peritoneal carcinomatosis and metastasis)** | 11 | 7.33 |
| **Tuberculosis** | 10 | 6.66 |
| **Nephrotic syndrome** | 1 | 0.66 |
| **Pancreatitis** | 0 | 0 |
| **Serositis** | 0 | 0 |

## DISCUSSION

Ascitic fluid can accumulate as a transudate or an exudate. Amounts of up to 25 litres are possible. Transudates are a result of increased pressure in the portal vein (>8 mmHg, usually around 20 mmHg), e.g., due to cirrhosis, while exudates are actively secreted fluid due to inflammation or malignancy. As a result, exudates are high in protein (>30 g/L), high in lactate dehydrogenase, have a low pH (<7.30), a low glucose level, and more white blood cells. Transudates have low protein (<30 g/L), low LDH, high pH, normal glucose, and less than 1 white cell per 1000 $mm^3$. Clinically, the most useful measure is the difference between ascitic and serum albumin concentrations. A difference of less than 1 g/dl (10 g/L) implies an exudate. Portal hypertension plays an important role in the production of ascites by raising capillary hydrostatic pressure within the splanchnic bed. Regardless of the cause, sequestration of fluid within the abdomen leads to additional fluid retention by the kidneys due to hyperaldosteronism and renin-angiotensin system triggered by decreased renal perfusion.

Ultrasound investigation is often performed prior to attempts to remove fluid from the abdomen. This may reveal the size and shape of the abdominal organs, and Doppler studies may show the direction of flow in the portal vein, as well as detecting Budd-Chiari syndrome and portal vein thrombosis. Additionally, the sonographer can make an estimation of the amount of ascitic fluid, and difficult-to-drain ascites may be drained under ultrasound guidance. Abdominal CT scan is a more accurate alternate to reveal abdominal organ structure and morphology. Laparoscopy is a safe diagnostic modality to establish the cause of low SAAG ascites, and one study showed using laparoscopy that tuberculous peritonitis was present in 22 patients, carcinomatous peritonitis in 5 and liver cirrhosis with hepatocellular carcinoma in 4 and Budd Chiari Syndrome in one patient.[16]

Our study revealed that most common cause of ascites is high SAAG ascites (85.33%), and most common aetiology of high SAAG ascites is cirrhsis of liver (81.33%), frequently caused by hapatitis viruses (70%) followed by alcohalism (7%). Our results are comparable to other studies and western figures. In west most common cause of high SAAG cirrhotic ascites (81%) is due alcoholism (65%) rather than viral hepatitis (10%). In our study in low SAAG group out of 22 (14.66%) patients; malignant ascites was (primary peritoneal carcinomatosis and metastasis) 11 (7.33%), tuberculous ascites was10 (6.66%), ascites due to nephrotic syndrome was 1 (0.66%). Hepatocellular carcinoma (HCC) has traditionally been considered a rare complication of cirrhosis secondary to autoimmune hepatitis (AIH), which subsequently occurs at a rate of 1.1% per year and affects men and women in equal proportions.[17] The risk of HCC can be predicted by male gender, features of portal hypertension, history of blood transfusions, immunosuppressive treatment for ≥3 yrs, treatment failure, and cirrhosis of ≥10 yr duration in cirrhosis secondary to AIH.[18]

Extreme disruption of the renal blood flow can lead to hepatorenal syndrome. The hepatorenal syndrome is characterized by renal failure due to severe renal vasoconstriction. It occurs in up to 10 percent of patients with advanced cirrhosis and ascites and may follow either of two following clinical patterns. In some patients, there is progressive oliguria and a rapid rise of the serum creatinine concentration. This condition is known as type 1 hepatorenal syndrome. In other patients, most of whom have refractory ascites, the increase in the serum creatinine concentration is moderate and has no tendency to progress over time. This pattern is known as type-2 hepatorenal syndrome. The prognosis is poor, particularly among patients with type 1 hepatorenal syndrome, who have a median survival of less than one month without therapy. Vasoconstrictor drugs, in combination with albumin, are effective in approximately two thirds of patients.[19] Octreotide is ineffective when administered alone.[20] Patients who have a response to terlipressin have a higher rate of survival than patients who do not have a response, a benefit that may reduce post-transplantation morbidity and mortality.[21–23]

Spontaneous bacterial peritonitis (SBP) is quite a common and fatal complication of liver cirrhosis with ascites, 9.3% of patients in one study had asymptomatic SBP.[24] Patients usually present

with abdominal pain, abdominal tenderness, fever with or without rigors, jaundice, and hepatic encephalopathy. E Coli is the commonest organism followed by streptococcal pneumoniae.[25] The SBP can develop due to decreased antibacterial factors in the ascitic fluid such as complement. The total polymorph count of >250/μL, is diagnostic of SBP. SBP is characterised by the spontaneous infection of ascitic fluid in the absence of an intra-abdominal source of infection. Its prevalence among patients with ascites ranges between 10 and 30 percent. Aerobic gram-negative bacteria, primarily Escherichia coli, are the most common isolates, although the frequency of episodes caused by gram-positive bacteria has recently increased, SBP involves the translocation of bacteria from the intestinal lumen to the lymph nodes, with subsequent bacteremia and infection of ascitic fluid. Long-term antibiotic prophylaxis with quinolones reduces the rate of recurrence, but SBP caused by quinolone-resistant bacteria is an emerging problem.[26] In one study patients of cirrhosis with ascites were prospectively evaluated for the presence of bacteria in SBP. E. coli (65.3%) found to be the common pathogen while salmonella (7.69%) proved to be uncommon organism involved.[27] Recurrent ascites associated with the Budd-Chiari syndrome can lead to partial obstruction of the small bowel owing to intestinal adhesions.[28] The incidence of hepatopulmonary syndrome (HPs) among patients of cirrhosis of liver was found to be 26%.[29] The HPs is characterised by a defect in arterial oxygenation induced by pulmonary vascular dilatation in the setting of liver disease; patients of all ages can be affected. This clinical syndrome has three components: liver disease, pulmonary vascular dilatation, and a defect in oxygenation.[30] The patient has been reported having decompensated cirrhosis was found to have pleuropericardial effusion.[31]

Ascites is generally treated simultaneously while an underlying etiology is sought in order to prevent complications, to relieve symptoms and to prevent further progression. High SAAG ascites can be managed by salt and diuretics. Diuresis can be monitored by weighing the patient daily. In patients with mild ascites, therapy is usually as an outpatient. The goal is weight loss of no more than 1.0 kg/day for patients with both ascites and peripheral oedema and no more than 0.5 kg/day for patients with ascites alone. Patients with cirrhosis of liver receiving high dose of the diuretics, having oedema, ascites and high serum creatinine are at the greater risk of developing hyperkalemia during spironolactone therapy.[32] Water restriction is needed if hyponatremia <130 mmol/L develops.[33] In those with tense ascites, therapeutic paracentesis may be needed in addition to medical treatment. As this may deplete serum albumin level in the blood, albumin is generally administered intravenously in proportion to the amount of ascites removed. Total abdominal paracentesis with IV Albumin or Plasma expanders is a safe procedure and can be used to relieve patients with tense ascites.[34] In a minority of patients with advanced cirrhosis that have recurrent ascites, shunts may be used. Typical shunts used are portacaval shunt, peritoneovenous shunt, and the transjugular intrahepatic portosystemic shunt (TIPS). Ascites due to cirrhosis that is refractory to medical therapy is considered an indication for liver transplantation. Exudative ascites generally does not respond to manipulation of the salt balance or diuretic therapy. Repeated paracentesis and treatment of the underlying cause is the mainstay of treatment. In the United States, the MELD score (online calculator) is used to prioritise patients for transplantation. Recent advances in regenerative medicine, including haematopoietic stem cell transplantation, have brought hope for patients with severe alcoholic liver cirrhosis. A study was conducted to assess the safety and efficacy of administering autologous expanded mobilised adult bone marrow derived progenitor CD34+ cells into the hepatic artery of patients with alcoholic cirrhosis for potential improvement in the liver function. The clinical and biochemical improvement in the study group was encouraging and warrants further clinical trials.[35]

## CONCLUSION

As large number of cases of ascites are due to cirrhosis of liver, which has little cure in developing countries. Prevention is always better than cure. Media and NGOs are trying to increase awareness of this deadly problem but still more is needed to be done.

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