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General Medicine

# CLINICO-ETIOLOGICAL PROFILE OF PATIENTS WITH NEW ONSET ASCITES ATTENDING A TERTIARY CARE CENTRE

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**ABSTRACT** **Background:** Ascites is one of the most common manifestations of decompensated chronic liver disease and is associated with significant morbidity, recurrent hospitalization, and poor quality of life. Although cirrhosis and portal hypertension are the leading causes worldwide, the etiological profile varies across regions depending on the prevalence of alcohol-related liver disease, chronic viral hepatitis, tuberculosis, and malignancy. Serum Ascites Albumin Gradient (SAAG), ascitic fluid analysis, and imaging studies are important tools for determining the underlying etiology of ascites. **Aim:** To study the clinical and etiological profile of patients presenting with new-onset ascites in a tertiary care centre. **Materials and Methods:** This hospital-based cross-sectional observational study was conducted in the Department of General Medicine at Assam Medical College and Hospital over a period of six months. A total of 97 consecutive patients aged ≥13 years with newly detected ascites were included. Detailed clinical evaluation, laboratory investigations, ascitic fluid analysis, SAAG calculation, and imaging studies were performed. Statistical analysis was carried out using SPSS version 25.0. **Results:** Most patients belonged to the 41–60 years age group with marked male predominance (75.3%). Abdominal distension (91.8%) and pedal edema (63.9%) were the most common presenting complaints. Alcoholism (58.8%) and chronic liver disease were major associated risk factors. Laboratory abnormalities included hypoalbuminemia, thrombocytopenia, elevated bilirubin, transaminitis, and prolonged PT/INR. Portal hypertension secondary to chronic liver disease was the predominant etiology (67.0%), followed by tuberculous peritonitis (15.5%) and malignant ascites (9.3%). High SAAG ascites correlated strongly with portal hypertension, while low SAAG ascites was commonly associated with tuberculous and malignant ascites. Ultrasonography frequently demonstrated cirrhosis, portal hypertension, splenomegaly, and peritoneal thickening. **Conclusion:** Portal hypertension secondary to chronic liver disease remains the leading cause of new-onset ascites in Northeast India. SAAG, ascitic fluid analysis, and imaging studies are reliable tools for etiological diagnosis and management of ascites.

**KEYWORDS :** Ascites; SAAG; Portal Hypertension; Cirrhosis; Tuberculous Peritonitis; Malignant Ascites.

## INTRODUCTION

Ascites refers to the pathological accumulation of fluid within the peritoneal cavity and represents one of the most common manifestations of decompensated chronic liver disease.[1] Under normal physiological conditions, the peritoneal cavity contains approximately 50–75 mL of lubricating fluid. Excessive fluid accumulation beyond this physiological amount is termed ascites.[2]

Ascites is associated with significant morbidity, recurrent hospitalization, impaired quality of life, and poor survival outcomes. Nearly 50% of patients with compensated cirrhosis develop ascites within 10 years of diagnosis.[3] Once ascites develops, prognosis worsens substantially with increased mortality and complications including spontaneous bacterial peritonitis, hepatorenal syndrome, and hepatic encephalopathy.

Globally, cirrhosis accounts for nearly 80–85% of ascites cases. Other important etiologies include malignancy, tuberculous peritonitis, cardiac failure, nephrotic syndrome, and pancreatitis.[4] In developing countries such as India, tuberculous and malignant ascites continue to contribute substantially to disease burden.[5] Studies from Northeast India have demonstrated increasing prevalence of alcohol-related liver disease and chronic hepatitis B infection contributing to portal hypertensive ascites.[6]

The pathophysiology of ascites formation in cirrhosis involves portal hypertension, splanchnic vasodilatation, activation of the renin-angiotensin-aldosterone system, sodium retention, and renal water retention.[3] Non-cirrhotic ascites results from mechanisms such as increased capillary permeability, peritoneal inflammation, malignancy, or hypoalbuminemia.

Serum Ascites Albumin Gradient (SAAG) has emerged as a reliable diagnostic tool for differentiating portal hypertensive from non-portal hypertensive ascites.[7] A SAAG ≥1.1 g/dL suggests portal hypertension, whereas SAAG <1.1 g/dL indicates non-portal hypertensive causes such as tuberculosis or malignancy. Ascitic fluid protein, ADA levels, cytology, and imaging studies further aid in etiological diagnosis.

Despite the clinical significance of ascites, region-specific data from Assam and Northeast India remain limited. Therefore, the present study was undertaken to evaluate the clinical presentation, laboratory abnormalities, imaging findings, ascitic fluid characteristics, and etiological profile of patients presenting with new-onset ascites.

## AIM

To study the clinical and etiological profile of patients presenting with new-onset ascites in a tertiary care centre.

## OBJECTIVES

1. To correlate Serum Ascites Albumin Gradient (SAAG), ascitic fluid protein, and imaging findings with the etiology of ascites.
2. To study the clinical spectrum and associated risk factors among patients presenting with new-onset ascites.

## MATERIALS AND METHODS

This hospital-based cross-sectional observational study was conducted in the Department of General Medicine, Assam Medical College and Hospital, Dibrugarh, Assam over a period of six months after obtaining approval from the Institutional Ethics Committee.

A total of 97 consecutive patients aged ≥13 years presenting with newly detected ascites confirmed clinically and/or radiologically were included in the study. Patients with previously diagnosed ascites, traumatic or postoperative ascites, and those unwilling to provide informed consent were excluded.

Detailed history regarding abdominal distension, abdominal pain, pedal edema, jaundice, fever, alcohol intake, multiple sexual partners, blood transfusion history, intravenous drug abuse, pulmonary tuberculosis, and associated comorbidities was obtained. Thorough

physical examination was performed in all cases.

Laboratory investigations included complete blood count, liver function tests, renal function tests, coagulation profile, serum electrolytes, random blood sugar, ESR, thyroid profile, viral markers including HBsAg and anti-HCV, and ICTC testing wherever indicated.

Diagnostic paracentesis was performed under aseptic precautions. Ascitic fluid was analyzed for total and differential cell count, protein, albumin, glucose, RBC count, ADA, malignant cells, and CBNAAT. SAAG was calculated using paired serum and ascitic albumin values.

Ultrasonography and computed tomography of the abdomen was performed in all patients to assess liver morphology, portal hypertension, splenomegaly, ascitic fluid volume, peritoneal thickening, and abdominal masses. Additional imaging studies including Doppler studies, chest radiography, and echocardiography were performed when clinically indicated.

Data were entered into Microsoft Excel and analyzed using SPSS version 25.0. Continuous variables were expressed as mean ± SD and categorical variables as frequency and percentage.

## RESULTS AND OBSERVATION

A total of 97 patients presenting with new-onset ascites were included in the study.

**Table 1: Demographic Profile of Study Participants**

| Variable | Category | Frequency (n) | Percentage (%) |
|---|---|---|---|
| Age Group | <30 years | 8 | 8.2% |
| | 31–40 years | 18 | 18.6% |
| | 41–50 years | 29 | 29.9% |
| | 51–60 years | 30 | 30.9% |
| | >60 years | 12 | 12.4% |
| Gender | Male | 73 | 75.3% |
| | Female | 24 | 24.7% |

**Table 2: Distribution of Risk Factors Among Study Participants**

| Risk Factor | Frequency (n) | Percentage (%) |
|---|---|---|
| Alcoholism | 57 | 58.8% |
| Multiple sexual partners | 18 | 18.6% |
| History of blood transfusion | 14 | 14.4% |
| Intravenous drug abuse | 7 | 7.2% |
| Hypertension | 23 | 23.7% |
| Diabetes mellitus | 19 | 19.6% |
| Dyslipidemia | 15 | 15.5% |
| Hypothyroidism | 9 | 9.3% |
| Obesity | 17 | 17.5% |
| Past pulmonary tuberculosis | 13 | 13.4% |

**Table 3: Clinical Spectrum of New-Onset Ascites**

| Clinical Feature | Frequency (n) | Percentage (%) |
|---|---|---|
| Abdominal distension | 89 | 91.8% |
| Pedal edema | 62 | 63.9% |
| Fatigue/weakness | 53 | 54.6% |
| Abdominal pain | 47 | 48.5% |
| Jaundice | 41 | 42.3% |
| Fever | 31 | 31.9% |
| Anorexia/weight loss | 35 | 36.1% |
| Difficulty breathing | 23 | 23.7% |
| Encephalopathy | 11 | 11.3% |
| Shifting dullness | 93 | 95.9% |
| Fluid thrill | 57 | 58.8% |
| Icterus | 45 | 46.4% |
| Hepatomegaly | 38 | 39.2% |
| Splenomegaly | 40 | 41.2% |
| Stigmata of chronic liver disease | 48 | 49.5% |

**Table 4: Laboratory Investigations of Study Participants**

| Parameter | Mean ± SD |
|---|---|
| Hemoglobin (g/dL) | 9.1 ± 2.2 |
| Total leukocyte count (/mm³) | 10,900 ± 3,500 |
| Platelet count (lakh/mm³) | 1.38 ± 0.62 |
| Serum bilirubin (mg/dL) | 3.5 ± 2.7 |
| Serum albumin (g/dL) | 2.4 ± 0.5 |
| AST (IU/L) | 82 ± 36 |
| ALT (IU/L) | 69 ± 28 |
| Globulin (g/dL) | 3.8 ± 1.2 |
| Alkaline phosphatase (IU/L) | 162 ± 58 |
| Serum creatinine (mg/dL) | 1.4 ± 0.7 |
| Random blood sugar (mg/dL) | 132 ± 42 |
| PT (seconds) | 21.2 ± 4.8 |
| INR | 1.81 ± 0.56 |
| ESR (mm/hr) | 42 ± 16 |
| TSH (mIU/L) | 4.8 ± 1.9 |
| HBsAg positive | 18 (18.6%) |
| Anti-HCV positive | 9 (9.3%) |
| ICTC reactive | 5 (5.2%) |

**Table 5: Ascitic Fluid Analysis Among Study Participants**

| Parameter | Mean ± SD / Frequency |
|---|---|
| Ascitic fluid total cell count (/mm³) | 326 ± 148 |
| Differential count (lymphocyte predominant) | 61 (62.9%) |
| Ascitic fluid protein (g/dL) | 2.5 ± 1.1 |
| Ascitic fluid albumin (g/dL) | 1.1 ± 0.4 |
| Ascitic fluid sugar (mg/dL) | 84 ± 26 |
| Ascitic fluid RBC (/mm³) | 178 ± 72 |
| ADA elevated (>40 IU/L) | 15 (15.5%) |
| Malignant cells positive | 8 (8.2%) |
| CBNAAT positive | 10 (10.3%) |

**Table 6: Correlation of SAAG, Ascitic Albumin, Protein and Imaging Findings with Etiology of Ascites**

| Etiology | Mean SAAG (g/dL) | Ascitic Albumin (g/dL) | Ascitic Protein | Imaging Finding |
|---|---|---|---|---|
| Portal hypertension/ cirrhosis | 1.8 ± 0.4 | 0.9 ± 0.3 | Low protein | Cirrhotic liver with portal hypertension |
| Tuberculous peritonitis | 0.8 ± 0.2 | 1.6 ± 0.4 | High protein | Peritoneal thickening with septations |
| Malignant ascites | 0.7 ± 0.3 | 1.8 ± 0.5 | High protein | Omental deposits/mass lesions |
| Cardiac ascites | 1.7 ± 0.3 | 1.3 ± 0.2 | High protein | Dilated IVC/ hepatomegaly |
| Renal ascites | 1.2 ± 0.2 | 1.2 ± 0.3 | Low protein | Bilateral contracted kidneys |

**Table 7: Etiological Profile of New-Onset Ascites**

| Etiology | Frequency (n) | Percentage (%) |
|---|---|---|
| Portal hypertension/cirrhosis | 65 | 67.0% |
| Tuberculous peritonitis | 15 | 15.5% |
| Malignant ascites | 9 | 9.3% |
| Renal ascites | 5 | 5.2% |
| Cardiac ascites | 2 | 2.1% |
| Others | 1 | 1.0% |

## DISCUSSION

The present hospital-based observational study evaluated the clinical and etiological profile of patients presenting with new-onset ascites in a tertiary care centre in Northeast India.

The majority of patients belonged to the 41–60 years age group with marked male predominance (75.3%). Similar observations were reported by Sharma et al.[8] and Singh et al.[9] Male predominance may be attributed to higher prevalence of alcohol use, smoking, viral hepatitis, and occupational exposure among males.

Alcoholism emerged as the most common associated risk factor (58.8%) in the present study. Similar findings were reported by Baruah et al. from Northeast India where alcohol-related liver disease constituted a major contributor to ascites and portal hypertension.[6] Hypertension, diabetes mellitus, obesity, and dyslipidemia were also common comorbidities, suggesting increasing contribution of metabolic syndrome-related liver disease.

Abdominal distension was the commonest presenting complaint, observed in 91.8% of patients, followed by pedal edema and generalized weakness. Similar findings were reported by Runyon et al. and Sharma et al.[3,8] Metabolic encephalopathy was observed in 11.3% of cases and was predominantly associated with advanced cirrhosis

and hepatic dysfunction.

Laboratory investigations demonstrated hypoalbuminemia, thrombocytopenia, elevated bilirubin, prolonged PT/INR, and elevated transaminases among patients with chronic liver disease and portal hypertension. Similar findings have been reported in previous Indian studies evaluating the biochemical profile of ascites.[9,10] Thrombocytopenia may be attributed to hypersplenism secondary to portal hypertension.

The present study demonstrated that portal hypertension secondary to chronic liver disease was the predominant etiology of ascites, accounting for 67.0% of cases. Tuberculous peritonitis constituted the second most common cause (15.5%), reflecting the continued burden of tuberculosis in developing countries. Comparable findings have been reported in studies from Northeast India and other tertiary care centres in India.[6,9]

SAAG proved to be a highly reliable diagnostic parameter. High SAAG ascites correlated strongly with cirrhosis and cardiac ascites, whereas low SAAG ascites was predominantly associated with tuberculous and malignant ascites. Similar findings were demonstrated by Runyon et al., who established the superiority of SAAG over the transudate-exudate concept in etiological diagnosis of ascites.[7]

Ascitic fluid analysis revealed elevated ADA and lymphocyte predominance predominantly among patients with tuberculous peritonitis. Malignant cells were detected in cytology-positive malignant ascites. Similar observations have been documented in previous Indian studies evaluating tuberculous and malignant ascites.[11]

Ultrasonography proved highly useful in etiological diagnosis. Cirrhotic liver morphology, portal hypertension, splenomegaly, and portal vein dilatation were common among portal hypertensive ascites, while peritoneal thickening and omental deposits suggested tuberculous and malignant ascites respectively.

The present study emphasizes the importance of integrating clinical findings, laboratory investigations, ascitic fluid analysis, SAAG, and imaging studies for accurate etiological diagnosis of ascites. Although the study was limited by its single-centre design and modest sample size, it provides important region-specific epidemiological data from Northeast India.

## CONCLUSION

Portal hypertension secondary to chronic liver disease remains the predominant cause of new-onset ascites in Northeast India, followed by tuberculous peritonitis and malignant ascites.

Middle-aged males constituted the majority of affected patients, with alcoholism emerging as the most important associated risk factor. Abdominal distension and pedal edema were the predominant clinical manifestations.

SAAG proved to be a simple, reliable, and clinically valuable parameter for differentiating portal hypertensive from non-portal hypertensive ascites. Ascitic fluid ADA, cytology, and ultrasonographic findings significantly aided etiological diagnosis.

An integrated diagnostic approach combining clinical evaluation, laboratory investigations, ascitic fluid analysis, SAAG, and imaging studies is essential for accurate diagnosis and effective management of patients presenting with new-onset ascites.

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