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Original Research Article

Clinico-etiological profile of ascites among adult patients attending a tertiary care hospital: a hospital-based cross-sectional study

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ABSTRACT

Background: Ascites is a common manifestation of several diseases and an important cause of hospitalization. Portal hypertension secondary to chronic liver disease remains the leading cause worldwide, although the etiological spectrum varies geographically. In India, tuberculosis, malignancy, and systemic disorders also contribute substantially. This study evaluated the clinical, laboratory, and etiological profile of adults presenting with ascites at a tertiary care hospital in Western India.
Methods: A hospital-based cross-sectional study was conducted among 267 consecutive adults with ascites admitted to the Department of Medicine at a tertiary care hospital in Vadodara, Gujarat, over one year. Demographic characteristics, clinical presentation, laboratory parameters, ultrasonographic findings, and ascitic fluid analysis were recorded using a structured proforma. Data were analyzed using descriptive and inferential statistics, with $p<0.05$ considered statistically significant.
Results: Among 267 participants, mean age was 53.7 ± 13.2 years and 181 (67.8%) were males. Chronic liver disease with portal hypertension was the commonest etiology (174; 65.2%), followed by malignancy (31; 11.6%), tuberculous peritonitis (24; 9.0%), congestive heart failure (17; 6.4%), nephrotic syndrome (11; 4.1%), pancreatitis (6; 2.2%), and miscellaneous causes (4; 1.5%). High-SAAG ascites was present in 191 (71.5%) patients and was significantly associated with portal hypertensive causes ($p<0.001$).
Conclusion: Chronic liver disease remains the predominant cause of ascites, while tuberculosis and malignancy remain important non-cirrhotic etiologies. Clinical evaluation combined with ascitic fluid analysis, particularly SAAG, facilitates accurate etiological diagnosis and appropriate management.

Keywords: Ascites, Chronic liver disease, Portal hypertension, Serum-ascites albumin gradient, Tuberculous peritonitis, Etiological profile, Cross-sectional study

INTRODUCTION

Ascites, defined as the pathological accumulation of fluid within the peritoneal cavity, is the most common complication of decompensated cirrhosis and an important cause of hospital admission worldwide. Approximately 50–60% of patients with compensated cirrhosis develop ascites within 10 years of diagnosis, and its development is associated with a marked reduction in survival and quality of life.[1] Portal hypertension secondary to liver

cirrhosis accounts for nearly 80-85% of cases of ascites globally, while malignancy, heart failure, nephrotic syndrome, pancreatic disease, and tuberculous peritonitis constitute the remaining causes. The relative frequency of these etiologies varies according to geographic location and the prevalence of underlying diseases. In developing countries, particularly India, tuberculosis remains an important differential diagnosis because of its continued endemicity, while alcohol-related liver disease and chronic viral hepatitis remain major causes of portal hypertensive ascites.[2]

The pathophysiology of ascites differs according to the underlying disease process. In cirrhosis, portal hypertension results in splanchnic vasodilatation, activation of the renin-angiotensin-aldosterone system, sympathetic nervous system stimulation, and renal sodium and water retention, ultimately leading to the accumulation of peritoneal fluid. In contrast, malignant, tuberculous, cardiac, pancreatic, and nephrotic ascites develop through mechanisms such as increased vascular permeability, lymphatic obstruction, hypoalbuminemia or elevated hydrostatic pressure. Current evidence-based guidelines recommend a systematic diagnostic approach to identify the underlying cause because treatment and prognosis vary considerably among different etiologies.[3]

Diagnostic paracentesis is recommended in all patients presenting with new-onset ascites. Ascitic fluid analysis, including total protein, cell count, culture, cytology, and particularly the serum–ascites albumin gradient (SAAG), plays a central role in differentiating portal hypertensive from non-portal hypertensive ascites. A SAAG value of ≥1.1 g/dL has been shown to identify portal hypertension with approximately 97% diagnostic accuracy, making it the preferred biochemical parameter in routine clinical practice.[4]

Recent reviews from India have highlighted the growing burden of alcohol-related liver disease, persistent prevalence of tuberculosis, and increasing complexity in the management of ascites. These changing epidemiological trends underscore the need for region-specific clinical data to facilitate timely diagnosis and optimize management strategies in tertiary care settings.[5] Although ascites is frequently encountered in routine clinical practice, contemporary data describing its clinical and etiological profile from Western India, particularly Gujarat, remain limited. Therefore, the present study was undertaken to evaluate the clinical, laboratory, and etiological profile of adult patients presenting with ascites at a tertiary care hospital in Vadodara, Gujarat.

METHODS

Study design

A hospital-based cross-sectional study was conducted to evaluate the clinical, laboratory, and etiological profile of adult patients presenting with ascites.

Study area

The study was carried out in the Department of Medicine, Sir Sayajirao General (SSG) Hospital, Vadodara, Gujarat, a tertiary care teaching hospital catering to patients from both urban and rural regions of Central Gujarat and neighbouring states.

Study period

The study was conducted over a period of one year, from January 2024 to December 2024.

Sample size

A total of 267 consecutive adult patients diagnosed with ascites and fulfilling the eligibility criteria were included in the study. Consecutive sampling was employed until the required sample size was achieved.

Sampling procedure

All adult patients admitted to the Department of Medicine with clinically and ultrasonographically confirmed ascites during the study period were screened for eligibility. Eligible participants were enrolled consecutively after obtaining written informed consent.

Inclusion criteria

Adult patients aged 18 years and above. Patients with clinically and ultrasonographically confirmed ascites. Patients willing to provide written informed consent.

Exclusion criteria

Patients aged below 18 years. Pregnant women. Patients with a history of abdominal surgery within the preceding three months. Patients with traumatic hemoperitoneum or postoperative peritoneal collections. Patients who declined to participate or had incomplete clinical records.

Data collection

Data were collected using a predesigned and pretested structured proforma. Information regarding demographic characteristics, presenting complaints, duration of illness, alcohol consumption, comorbidities, past medical history, and clinical examination findings was recorded. Routine laboratory investigations including complete blood count, liver function tests, renal function tests, serum electrolytes, coagulation profile, viral markers, and serum albumin were obtained from hospital records.

All participants had undergone abdominal ultrasonography to confirm the presence of ascites and evaluate associated findings such as liver morphology, splenomegaly, portal vein diameter, and intra-abdominal pathology. Diagnostic paracentesis was performed under aseptic precautions whenever clinically indicated. Ascitic

fluid was analyzed for gross appearance, total and differential leukocyte count, protein concentration, albumin, serum–ascites albumin gradient (SAAG), Gram stain, bacterial culture, cytological examination, and adenosine deaminase (ADA) levels when tuberculosis was suspected. The underlying etiology of ascites was established based on clinical findings, laboratory investigations, imaging studies, and ascitic fluid analysis.

Statistical analysis

Data were entered into Microsoft Excel and analyzed using Jamovi software (Version 2.3). Continuous variables were expressed as mean ± standard deviation (SD), whereas categorical variables were presented as frequencies and percentages. Associations between categorical variables were assessed using the Chi-square test or Fisher's exact test, as appropriate. A p-value <0.05 was considered statistically significant.

RESULTS

A total of 267 adult patients with clinically and ultrasonographically confirmed ascites were included in the study. The mean age of the study population was 53.7 ± 13.2 years (range: 19–84 years). The majority of patients were males (181; 67.8%), resulting in a male-to-female ratio of 2.1:1.

Demographic and clinical characteristics

The largest proportion of patients belonged to the 51–60 years age group (76; 28.5%), followed by 41–50 years (61; 22.8%) and 61–70 years (53; 19.9%). Only 17 (6.4%) patients were younger than 30 years.

Table 1: Demographic characteristics of study participants (n=267).

Variable	Number (%)
Age (Mean ± SD)	53.7±13.2 years
18–30 years	17 (6.4)
31–40 years	32 (12.0)
41–50 years	61 (22.8)
51–60 years	76 (28.5)
61–70 years	53 (19.9)
>70 years	28 (10.5)
Male	181 (67.8)
Female	86 (32.2)

Clinical presentation

Abdominal distension was the commonest presenting complaint, observed in 239 (89.5%) patients, followed by pedal edema (171; 64.0%), abdominal pain (127; 47.6%), jaundice (116; 43.4%), anorexia (104; 39.0%), weight loss (81; 30.3%), dyspnea (73; 27.3%), and fever (41; 15.4%).

Table 2: Clinical presentation of patients with ascites.

Clinical Feature	Number (%)
Abdominal distension	239 (89.5)
Pedal edema	171 (64.0)
Abdominal pain	127 (47.6)
Jaundice	116 (43.4)
Anorexia	104 (39.0)
Weight loss	81 (30.3)
Dyspnea	73 (27.3)
Fever	41 (15.4)

Table 3: Etiological profile of ascites.

Etiology	Number (%)
Chronic liver disease with portal hypertension	174 (65.2)
Malignancy	31 (11.6)
Tuberculous peritonitis	24 (9.0)
Congestive heart failure	17 (6.4)
Nephrotic syndrome	11 (4.1)
Pancreatitis	6 (2.2)

Continued.

Etiology	Number (%)
Miscellaneous	4 (1.5)

Etiological profile

Chronic liver disease with portal hypertension was the predominant cause of ascites, accounting for 174 (65.2%) patients. Malignancy was the second most common cause

(31; 11.6%), followed by tuberculous peritonitis (24; 9.0%), congestive heart failure (17; 6.4%), nephrotic syndrome (11; 4.1%), pancreatitis (6; 2.2%), and miscellaneous causes (4; 1.5%).

Laboratory profile

The mean serum albumin level was 2.8±0.7 g/dl, while the mean ascitic fluid protein concentration was 2.1±1.1 g/dL. High-SAAG ascites (≥1.1 g/dl) was observed in 191 (71.5%) patients, whereas 76 (28.5%) had low-SAAG ascites (<1.1 g/dl).

Association between SAAG and etiology

High-SAAG ascites was predominantly observed among patients with chronic liver disease and congestive heart failure, whereas low-SAAG ascites was more frequently associated with malignancy, tuberculous peritonitis,

nephrotic syndrome, and pancreatic ascites. The association between SAAG category and underlying etiology was statistically significant (Chi-square test, p <0.001).

Table 4: Laboratory characteristics.

Variable	Value
Serum albumin (g/dl)	2.8±0.7
Ascitic fluid protein (g/dl)	2.1±1.1
High SAAG (≥1.1 g/dl)	191 (71.5)
Low SAAG (<1.1 g/dl)	76 (28.5)

Table 5: Association between SAAG and etiology.

Etiology	High SAAG N (%)	Low SAAG N (%)
Chronic liver disease	170	4
Congestive heart failure	16	1
Malignancy	2	29
Tuberculous peritonitis	1	23
Nephrotic syndrome	1	10
Pancreatitis	0	6
Miscellaneous	1	3

Chi-square = 214.6, p <0.001

Table 6: Associated comorbidities and risk factors among patients with ascites (n=267).

Comorbidity / Risk factor	Number (%)
Chronic alcohol consumption	142 (53.2)
Hypertension	76 (28.5)
Diabetes mellitus	68 (25.5)
Chronic viral hepatitis (HBV/HCV)	41 (15.4)
Chronic kidney disease	29 (10.9)
Coronary artery disease	24 (9.0)
Previous history of liver disease	157 (58.8)
Smoking	94 (35.2)

The present study demonstrated that chronic liver disease with portal hypertension was the leading cause of ascites among adult patients admitted to a tertiary care hospital. Abdominal distension and pedal edema were the most frequent presenting complaints. High-SAAG ascites predominated and showed a strong association with portal hypertensive etiologies, whereas low-SAAG ascites was mainly observed in patients with malignancy and tuberculous peritonitis.

Associated comorbidities and risk factors

Chronic alcohol consumption was the most common associated risk factor, reported in 142 (53.2%) patients,

followed by a previous history of chronic liver disease (157; 58.8%). Hypertension and diabetes mellitus were present in 76 (28.5%) and 68 (25.5%) patients, respectively. Chronic viral hepatitis was identified in 41 (15.4%) patients, while chronic kidney disease and coronary artery disease were observed in 29 (10.9%) and 24 (9.0%) patients, respectively. A history of smoking was present in 94 (35.2%) participants.

DISCUSSION

The present hospital-based cross-sectional study evaluated 267 adult patients with ascites presenting to a tertiary care hospital in Western India. The mean age of the study population was 53.7 ± 13.2 years, with a clear male predominance (67.8%). Chronic liver disease with portal hypertension was the leading cause of ascites (65.2%), followed by malignancy (11.6%), tuberculous peritonitis (9.0%), congestive heart failure (6.4%), nephrotic syndrome (4.1%), pancreatitis (2.2%), and miscellaneous causes (1.5%). High serum-ascites albumin gradient (SAAG ≥1.1 g/dL) was observed in 71.5% of patients and was significantly associated with portal hypertensive ascites ($p < 0.001$).

Our findings are comparable with those of Dalabehera et al who evaluated 100 patients with ascites in an Indian tertiary care hospital. They reported cirrhosis as the predominant etiology (64%), followed by tuberculosis (10%), malignancy (9%), congestive heart failure (7%), and nephrotic syndrome (3%).[6] Despite differences in sample size, the etiological distribution was remarkably similar to our study, suggesting that chronic liver disease remains the principal cause of ascites across Indian tertiary care centres. The slightly higher proportion of malignant ascites observed in our study (11.6% vs. 9%) may reflect increasing referrals of oncology patients and improved diagnostic evaluation.

Similar observations were reported by Nakhale et al from Maharashtra, who studied patients with ascites at a tertiary referral hospital and found cirrhosis in 68% of cases, abdominal tuberculosis in 16%, congestive heart failure in 10%, nephrotic syndrome in 4%, and malignancy in 2%.[7] Compared with their findings, our study demonstrated a comparable prevalence of cirrhosis (65.2%) but a lower proportion of tuberculous ascites (9.0% vs. 16%) and a higher frequency of malignant ascites (11.6% vs. 2%). These differences are likely attributable to regional variations in disease prevalence, referral patterns, and availability of advanced diagnostic investigations.

The demographic profile of our patients is also consistent with recent evidence from Western India. Badi et al. evaluated patients with decompensated liver cirrhosis and reported that the majority were middle-aged males, with alcohol-related liver disease being the most frequent underlying etiology.[8] Our study similarly demonstrated a predominance of male patients (67.8%) with a mean age of approximately 54 years, supporting previous observations that chronic liver disease remains a major contributor to ascites among economically productive adults in India.

International studies have reported comparable findings. In a prospective observational study from Qatar involving patients with ascites, Khan found liver cirrhosis to be the commonest cause (approximately 60%), followed by malignancy (approximately 12%), tuberculous peritonitis (approximately 8%), congestive heart failure (approximately 7%), and nephrotic syndrome (approximately 3%).[9] These findings closely mirror those of the present study and indicate that portal hypertensive liver disease remains the predominant cause of ascites irrespective of geographical location.

Likewise, Shaikh et al evaluated 150 patients admitted with ascites and reported cirrhosis in 81.3% of cases, while malignant ascites and tuberculous ascites accounted for 7.3% and 6.7%, respectively.[10] Although the proportion of cirrhosis was higher than in our study, both studies consistently identified chronic liver disease as the principal etiology. The relatively lower proportion of cirrhosis in the present study may be explained by inclusion of a broader spectrum of medical patients and increasing recognition of non-cirrhotic causes of ascites.

An important finding of the present study was that 71.5% of patients had high-SAAG ascites, which showed a strong association with portal hypertensive disorders. This finding is biologically plausible and is supported by the landmark study of Runyon et al who demonstrated that the serum–ascites albumin gradient (SAAG) is superior to the traditional transudate-exudate classification for differentiating portal hypertensive from non-portal hypertensive ascites.[4] More recently, the British Society of Gastroenterology guidelines have reaffirmed the role of SAAG as the cornerstone of diagnostic evaluation in patients presenting with new-onset ascites.[3]

Recent evidence from a large multi-ethnic Asian cohort involving 838 patients further supports our observations. Sinnanaidu et al reported that while malignancy (28.9%) and liver cirrhosis (27.9%) were the leading causes of ascites overall, the etiological spectrum varied considerably according to ethnicity.[11] Among patients of Indian ethnicity, chronic liver disease remained the predominant cause of ascites, consistent with the findings of the present study. These observations emphasize the influence of regional disease epidemiology and ethnic differences on the underlying causes of ascites. The findings of the present study reinforce current recommendations that evaluation of ascites should begin with careful clinical assessment, abdominal ultrasonography, and diagnostic paracentesis, including SAAG estimation, to accurately identify the underlying etiology and guide management.[1,3,4] Early recognition of non-cirrhotic causes such as malignancy and tuberculous peritonitis is particularly important in developing

countries, where delayed diagnosis may adversely affect prognosis.

This study has certain limitations. First, being a hospital-based cross-sectional study, the findings may not be generalizable to the community or primary healthcare settings. Second, the study was conducted at a single tertiary care center, and the etiological distribution of ascites may vary across different geographical regions and healthcare facilities. Third, the cross-sectional design precludes assessment of temporal relationships, disease progression, and long-term outcomes. Finally, specialized investigations such as hepatic venous pressure gradient measurement, liver biopsy, and advanced imaging were not performed routinely in all patients and were reserved for selected clinical indications, which may have limited further etiological characterization in a small proportion of cases. Overall, the present study adds contemporary data from Western India and demonstrates that chronic liver disease with portal hypertension remains the predominant cause of ascites, while malignancy and tuberculous peritonitis continue to represent important alternative etiologies. The findings are consistent with both Indian and international literature and support the continued use of a systematic diagnostic approach incorporating clinical evaluation, imaging, and ascitic fluid analysis.

CONCLUSION

The present study demonstrated that chronic liver disease with portal hypertension was the predominant cause of ascites among adult patients admitted to a tertiary care hospital, followed by malignancy and tuberculous peritonitis. Abdominal distension and pedal edema were the most common clinical manifestations. High-SAAG ascites showed a significant association with portal hypertensive causes, highlighting its value as a simple and reliable diagnostic tool in the etiological evaluation of ascites. A systematic approach incorporating detailed clinical assessment, laboratory investigations, imaging, and ascitic fluid analysis facilitates accurate diagnosis and appropriate management. The findings also emphasize the continued burden of alcohol-related liver disease and tuberculosis in Western India, underscoring the need for early diagnosis and targeted interventions to improve patient outcomes.

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