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Article

Clinical and Laboratory Characterization of Ascites in a Tertiary Care Setting: A Prospective Observational Study at Hayatabad Medical Complex, Peshawar

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ABSTRACT

Background: Ascites, the pathological accumulation of fluid in the peritoneal cavity, is commonly associated with liver cirrhosis; however, in South Asia, tuberculosis and malignancy are also important etiological contributors. Limited regional data exist detailing the clinical and laboratory characteristics of ascites in such high-burden settings, which complicates diagnosis and management. **Objective**: To evaluate the demographic distribution, clinical features, etiological classification, and diagnostic fluid analysis findings in patients with ascites admitted to a tertiary care center in Peshawar, Pakistan. **Methods**: A prospective observational study was conducted at Hayatabad Medical Complex from January 2023 to December 2024, enrolling 246 adult patients with clinically and radiologically confirmed ascites. Detailed history, physical examination, serum studies, ascitic fluid analysis (including SAAG, ADA, CEA, PMN count), GeneXpert MTB/RIF testing, ultrasound, and FibroScan were performed. Etiologies were classified using standard diagnostic criteria. Data were analyzed using SPSS version 25. **Results**: The mean age was 45 years; 83% were male. Cirrhosis was the leading cause (44.7%), followed by tubercular peritonitis (24.4%) and malignancy (12.2%). SAAG >1.1 g/dL was observed in 60.9% of patients. ADA >32 IU/L and GeneXpert positivity supported TB diagnosis in 18.3% and 16.3% respectively. CEA >5 ng/mL was found in 11.4% of cases. **Conclusion**: In this high-burden setting, cirrhosis remains the predominant cause of ascites, but tuberculosis and malignancy together account for over one-third of cases. Incorporating ADA, GeneXpert, and CEA into diagnostic algorithms improves etiological classification and facilitates targeted management in resource-constrained environments.

Keywords: Ascites, Cirrhosis, Tuberculosis, Malignancy, SAAG, ADA, GeneXpert, Pakistan

INTRODUCTION

Ascites, the pathological accumulation of fluid in the peritoneal cavity, is most frequently associated with decompensated liver cirrhosis, accounting for up to 75% of cases globally (1). In cirrhotic patients, the primary pathophysiological mechanism involves portal hypertension, which causes transudative fluid shift across splanchnic capillaries into the peritoneal space. A serum-ascites albumin gradient (SAAG) exceeding 1.1 g/dL is considered a reliable marker of portal hypertensive ascites (2). However, the etiological landscape of ascites is notably broader in developing countries, particularly in South Asia, where infections like peritoneal tuberculosis and malignancy-related exudates remain prevalent and diagnostically challenging (3,4).

Pakistan bears a disproportionately high burden of tuberculosis, ranking among the top ten globally in TB incidence, and extrapulmonary TB forms a considerable share of this load (5). Peritoneal tuberculosis often mimics cirrhotic ascites, and its diagnosis is complicated by the paucibacillary nature of the infection and overlapping clinical features (6). In such scenarios, ascitic fluid adenosine deaminase (ADA) levels and molecular assays like Xpert MTB/RIF have demonstrated strong diagnostic utility, offering rapid, sensitive, and non-invasive alternatives to laparoscopic or histological confirmation (7,8). On the other hand, malignant ascites, typically caused by gastrointestinal, pancreatic, or ovarian carcinomas, demands early identification through cytology and biomarkers such as carcinoembryonic antigen (CEA), with levels above 5 ng/mL raising suspicion of peritoneal carcinomatosis (9,10).

While cirrhosis and its complications dominate Western data on ascites, several Asian epidemiological studies have reported a substantial proportion of cases arising from tuberculosis and malignancy, especially in younger age groups and socioeconomically vulnerable populations (11,12). FibroScan, a noninvasive elastography-based method, has gained recognition in such contexts for assessing hepatic fibrosis, with liver stiffness measurements over 12.5 kPa strongly suggestive of cirrhosis (13). Despite these

diagnostic advances, a unified diagnostic algorithm for ascites in high-burden, resource-limited settings like Pakistan is lacking, and physician decisions often rely on fragmented investigations, empirical treatment, or invasive procedures.

There is a paucity of prospective, systematically documented clinical studies from Pakistan that comprehensively profile ascites patients using a combination of clinical, biochemical, imaging, and molecular tools. Existing studies are either retrospective or underpowered, failing to capture the nuanced diagnostic dilemmas encountered in tertiary care practice. This knowledge gap impedes development of evidence-based local diagnostic pathways tailored to the regional spectrum of disease. In this context, the present study was conducted to assess the clinical characteristics, etiological profile, and ascitic fluid parameters of adult patients presenting with ascites to a tertiary care hospital in Peshawar, Pakistan. Specific objectives included evaluating the relative frequency of cirrhosis, tuberculosis, malignancy, and other causes; describing key signs and symptoms; and determining the diagnostic value of SAAG, ADA, GeneXpert, CEA, and FibroScan in the etiological classification of ascites. This study seeks to inform context-sensitive diagnostic strategies and optimize resource allocation in the evaluation of ascites in South Asia.

MATERIALS AND METHODS

This prospective observational study was conducted to evaluate the clinical and laboratory characteristics of ascites in adult patients admitted to a tertiary care center in Pakistan. The rationale for adopting this design was to systematically capture and analyze real-world diagnostic and management practices over time in a high-burden setting. The study was carried out at the Gastroenterology Unit of Hayatabad Medical Complex (HMC), a major tertiary care teaching hospital located in Peshawar, Khyber Pakhtunkhwa. Patient enrollment took place continuously from January 2023 to December 2024.

Eligible participants included all adult patients aged 18 years or older who presented with new or previously undiagnosed ascites, confirmed clinically and radiologically by abdominal ultrasound. Inclusion criteria required visible or palpable signs of fluid accumulation in the peritoneal cavity in conjunction with ultrasound-confirmed ascites. Patients with isolated pleural effusions, ascites due to recent abdominal trauma, or those with incomplete diagnostic workup were excluded from the study. Consecutive sampling was employed to minimize selection bias; all eligible patients presenting during the study period were invited to participate until the target sample size was reached. Prior to enrollment, each participant received a detailed explanation of the study aims, procedures, risks, and confidentiality safeguards. Written informed consent was obtained from all patients, in either Urdu or Pashto, depending on the patient's preferred language.

Clinical and demographic data were collected using a standardized case record form. Information included age, sex, presenting symptoms, past medical history, alcohol or drug use, known liver disease, prior tuberculosis, and comorbidities such as diabetes mellitus or cardiac disease. A detailed physical examination was performed by trained gastroenterology fellows to document abdominal distension, pedal edema, icterus, hepatosplenomegaly, and any signs of hepatic decompensation. Diagnostic paracentesis was performed under strict aseptic technique using a 22-gauge needle, and 20–50 mL of ascitic fluid was collected from each patient. Samples were promptly transported to the hospital laboratory for analysis. Ascitic fluid was tested for total protein, albumin, glucose, lactate dehydrogenase (LDH), red and white blood cell count with differential, polymorphonuclear (PMN) cell count, and adenosine deaminase (ADA). Serum-ascites albumin gradient (SAAG) was calculated using concurrently obtained serum and ascitic albumin levels. A SAAG >1.1 g/dL was interpreted as indicative of portal hypertensive ascites, whereas SAAG ≤1.1 g/dL suggested exudative ascites due to infections, malignancy, or other etiologies (1). ADA levels >32 IU/L in ascitic fluid were used to support a diagnosis of tubercular peritonitis, based on established diagnostic thresholds for high TB-burden regions (2). GeneXpert MTB/RIF assay was performed on ascitic fluid from patients suspected of having tuberculosis, especially when ADA levels were borderline or clinical suspicion was high. Carcinoembryonic antigen (CEA) levels were measured in ascitic fluid to evaluate for possible malignant etiology, with values >5 ng/mL suggestive of peritoneal carcinomatosis (3). Cytological examination was conducted in select cases to confirm malignancy when feasible.

Blood tests included complete blood count (CBC), liver function tests (LFTs), renal function tests (RFTs), C-reactive protein (CRP), coagulation profile (PT/INR), and hepatitis B and C serology. CRP levels >10 mg/L were considered elevated and indicative of systemic inflammation. Abdominal ultrasound was performed in all patients. Those with suspected chronic liver disease underwent transient elastography (FibroScan) to measure liver stiffness, with values ≥12.5 kPa classified as diagnostic of cirrhosis based on international standards (4). Additional imaging such as chest X-ray or CT scan, and upper gastrointestinal endoscopy, were conducted when clinically indicated to evaluate for complications or alternative diagnoses.

To mitigate information bias, all data were collected prospectively using a uniform template by trained personnel under consultant supervision. Ascitic fluid analyses were carried out in the hospital's central diagnostic laboratory using standardized protocols. Diagnostic thresholds and clinical categorizations were uniformly applied to minimize classification bias. Confounding was addressed during data analysis through stratification of variables by etiology (e.g., cirrhotic vs. tubercular vs. malignant) and by evaluating potential covariates such as age, sex, alcohol history, and CRP levels. Sample size was determined based on the estimated proportion of cirrhotic ascites cases in the target population. Assuming a 45% prevalence of cirrhosis among ascites patients (based on pilot data), a sample of 246 patients provided a 95% confidence level with a margin of error of ±6%. All data were entered into SPSS version 25.0 (IBM Corp., Armonk, NY) for statistical analysis. Continuous variables were summarized using means and standard deviations (or medians and interquartile ranges where appropriate), while categorical variables were presented as frequencies and percentages.

The Chi-square test was used to compare proportions, and Student's t-test or Mann-Whitney U test was used to compare continuous variables depending on normality. Subgroup analyses were performed to compare clinical and laboratory parameters across the three most common etiologies: cirrhosis, tuberculosis, and malignancy. A p-value <0.05 was considered statistically significant. Missing data were handled using listwise deletion if data were missing completely at random; otherwise, sensitivity analyses were performed excluding and including such cases to assess impact.

Ethical approval was granted by the Institutional Review Board (IRB) of Hayatabad Medical Complex prior to study initiation. All data were anonymized using unique patient codes, and paper forms were stored in locked cabinets with restricted access. Digital data were password-protected and accessible only to authorized research staff. All steps of data entry, analysis, and reporting were documented and reviewed by an independent supervisor to ensure reproducibility and minimize investigator bias. The research adhered to the principles of the Declaration of Helsinki and local ethical guidelines throughout its execution.

RESULTS

The data presented across these five comprehensive tables reveal clinically and statistically significant differences in the etiological spectrum, symptomatology, biochemical profiles, management strategies, and complications of ascites across its three most common causes: cirrhosis, tuberculous peritonitis, and malignancy. Out of 246 patients, cirrhosis was the most prevalent cause, accounting for 44.7% (n=110), followed by tuberculous peritonitis at 24.4% (n=60), and malignancy at 12.2% (n=30). Other less frequent causes, including nephrotic syndrome (8.1%), congestive cardiac failure (4.1%), and pancreatitis (2.0%), each had significantly lower odds of occurrence when compared with cirrhosis (p<0.001 for all, ORs ranging from 0.04 to 0.39), underscoring the dominance of liver-related and infectious etiologies in this population.

When analyzing clinical features, abdominal distension was universally observed across groups (>93% in all), but other findings varied significantly. Pedal edema was significantly more common in cirrhotics (69.1%) compared to those with tuberculosis (46.7%, OR=0.39, 95% CI: 0.20–0.77) or malignancy (36.7%, OR=0.23, 95% CI: 0.09–0.57). Similarly, icterus (60.0%) and splenomegaly (32.7%) were more frequent in cirrhosis than in malignancy (23.3%, OR=0.21; and 10.0%, OR=0.24, respectively). Conversely, weight loss, a hallmark of chronic illness, was significantly higher in malignancy (60.0%) and tuberculosis (43.3%) compared to cirrhosis (21.8%), with ORs of 5.25 (95% CI: 2.05–13.4) and 2.78 (95% CI: 1.39–5.57), respectively.

In diagnostic fluid analysis, SAAG >1.1 g/dL was overwhelmingly associated with cirrhosis (98.2%), but was rarely seen in TB (16.7%) or malignancy (26.7%), offering highly discriminatory power (p<0.001; OR for TB vs. cirrhosis = 0.005, 95% CI: 0.002–0.015). ADA >32 IU/L was specific for TB (73.3%, OR >999), while CEA >5 ng/mL was markedly elevated in malignancy (83.3%, OR=482.1, 95% CI: 56.1–4141), offering robust diagnostic clues. Xpert MTB/RIF confirmed TB in 63.3% of TB patients, with zero detection in other groups. While PMN counts ≥250/mm^3, indicating spontaneous bacterial peritonitis (SBP), were more common in cirrhosis (15.5%), the difference did not reach statistical significance (p=0.11).

Management strategies reflect etiological differences. Diuretics and salt restriction were almost universal in cirrhosis (95.5% and 98.2%, respectively), but significantly less so in TB (56.7%, OR=0.09) and malignancy (36.7%, OR=0.04). Therapeutic paracentesis was used in 54.5% of cirrhotic versus 33.3% in TB (p=0.02), and human albumin infusion was more frequently administered in cirrhosis (69.1%) compared to TB (46.7%, OR=0.38) and malignancy (20.0%, OR=0.12). As expected, all TB patients received anti-tubercular therapy, and all malignancy patients were referred to oncology. Beta-blockers and SBP-directed antibiotics were more common in cirrhosis, although SBP-specific treatment did not significantly differ by group (p=0.11).

Table 1. Etiological Distribution of Ascites by Group

Etiology	Number (%)	95% CI	Reference	p-value	Odds Ratio (OR) [95% CI]
Cirrhosis of liver	110 (44.7)	38.5–51.1	—	—	—
Tuberculous peritonitis	60 (24.4)	19.1–30.1	Cirrhosis	<0.001	0.39 [0.27–0.56]
Malignancy (metastatic cancer)	30 (12.2)	8.4–16.9	Cirrhosis	<0.001	0.21 [0.13–0.34]
Nephrotic syndrome	20 (8.1)	5.0–12.2	Cirrhosis	<0.001	0.15 [0.08–0.28]
Congestive cardiac failure	10 (4.1)	2.0–7.4	Cirrhosis	<0.001	0.08 [0.03–0.18]
Pancreatitis	5 (2.0)	0.7–4.6	Cirrhosis	<0.001	0.04 [0.01–0.11]
Portal hypertension (non-cirrhotic)	5 (2.0)	0.7–4.6	Cirrhosis	<0.001	0.04 [0.01–0.11]
Other	6 (2.4)	0.9–5.1	Cirrhosis	<0.001	0.05 [0.02–0.13]

Regarding complications, hepatic encephalopathy was the most prevalent among cirrhotics (43.6%) but occurred in only 3.3% of TB and 6.7% of malignancy cases (p<0.001; OR=0.05 and 0.09 respectively). Upper gastrointestinal bleeding was similarly restricted to cirrhotic (29.1%, OR for TB = 0.04, p<0.001). Acute kidney injury was more frequently seen in cirrhosis (19.1%) compared to TB (5.0%, OR=0.22) and malignancy (3.3%, OR=0.14), indicating the severity of end-organ involvement in advanced liver disease. Esophageal varices and altered sensorium were reported in smaller proportions and did not show statistically significant differences. Together, these data paint a nuanced picture of ascites in a high TB-burden region, demonstrating not only the predominance of cirrhosis but also the significant representation and distinct clinical-biochemical profiles of TB and malignancy-associated ascites. These patterns reinforce the need for etiologically tailored diagnostic and management strategies in tertiary care settings.

Table 2. Clinical Features by Etiological Group

Feature	Cirrhosis (n=110)	TB Peritonitis (n=60)	Malignancy (n=30)	p-value	OR [95% CI]	OR [95% CI]
Abdominal distension	104 (94.5%)	57 (95.0%)	28 (93.3%)	0.98	1.09 [0.22–5.31]	0.77 [0.15–4.01]
Pedal edema	76 (69.1%)	28 (46.7%)	11 (36.7%)	<0.01	0.39 [0.20–0.77]	0.23 [0.09–0.57]
Icterus (jaundice)	66 (60.0%)	16 (26.7%)	7 (23.3%)	<0.001	0.25 [0.12–0.51]	0.21 [0.08–0.56]
Splenomegaly	36 (32.7%)	15 (25.0%)	3 (10.0%)	0.04	0.69 [0.33–1.43]	0.24 [0.06–0.87]
Weight loss	24 (21.8%)	26 (43.3%)	18 (60.0%)	<0.001	2.78 [1.39–5.57]	5.25 [2.05–13.4]

Table 3. Ascitic Fluid Analysis Across Major Etiologies

Parameter	Cirrhosis (n=110)	TB Peritonitis (n=60)	Malignancy (n=30)	p-value	OR [95% CI]	OR [95% CI]
SAAG >1.1 g/dL	108 (98.2%)	10 (16.7%)	8 (26.7%)	<0.001	0.005 [0.002–0.015]	0.009 [0.003–0.028]
Ascitic ADA >32 IU/L	0 (0.0%)	44 (73.3%)	1 (3.3%)	<0.001	>999 (NA)	3.43 [0.22–54.2]
Xpert MTB/RIF positive	0 (0.0%)	38 (63.3%)	0 (0.0%)	<0.001	>999 (NA)	NA
Ascitic CEA >5 ng/mL	0 (0.0%)	1 (1.7%)	25 (83.3%)	<0.001	NA	482.1 [56.1–4141]
PMN ≥250/mm³ (SBP)	17 (15.5%)	5 (8.3%)	1 (3.3%)	0.11	0.49 [0.17–1.41]	0.18 [0.02–1.44]

Table 4. Management Interventions by Etiological Group

Intervention	Cirrhosis (n=110)	TB Peritonitis (n=60)	Malignancy (n=30)	p-value	OR [95% CI]	OR [95% CI]
Diuretics	105 (95.5%)	34 (56.7%)	11 (36.7%)	<0.001	0.09 [0.04–0.24]	0.04 [0.01–0.11]
Salt-restricted diet	108 (98.2%)	49 (81.7%)	15 (50.0%)	<0.001	0.09 [0.02–0.38]	0.01 [0.003–0.04]
Therapeutic paracentesis	60 (54.5%)	20 (33.3%)	15 (50.0%)	0.02	0.42 [0.21–0.84]	0.83 [0.35–1.98]
Human albumin infusion	76 (69.1%)	28 (46.7%)	6 (20.0%)	<0.001	0.38 [0.19–0.76]	0.12 [0.04–0.33]
Antitubercular therapy	0 (0.0%)	60 (100%)	0 (0.0%)	<0.001	>999 (NA)	NA
Chemotherapy/Oncology referral	0 (0.0%)	0 (0.0%)	25 (83.3%)	<0.001	NA	>999 (NA)
Antibiotics for SBP	17 (15.5%)	5 (8.3%)	1 (3.3%)	0.11	0.49 [0.17–1.41]	0.18 [0.02–1.44]

Table 5. Complications Observed by Etiological Group

Complication	Cirrhosis (n=110)	TB Peritonitis (n=60)	Malignancy (n=30)	p-value	OR [95% CI]	OR [95% CI]
Hepatic encephalopathy	48 (43.6%)	2 (3.3%)	2 (6.7%)	<0.001	0.05 [0.01–0.22]	0.09 [0.02–0.38]
UGI bleeding	32 (29.1%)	1 (1.7%)	0 (0.0%)	<0.001	0.04 [0.01–0.30]	NA
Acute kidney injury	21 (19.1%)	3 (5.0%)	1 (3.3%)	0.01	0.22 [0.06–0.78]	0.14 [0.02–1.19]
Esophageal varices	8 (7.3%)	1 (1.7%)	0 (0.0%)	0.18	0.22 [0.03–1.88]	NA
Altered sensorium	9 (8.2%)	1 (1.7%)	1 (3.3%)	0.18	0.19 [0.02–1.54]	0.38 [0.04–3.31]

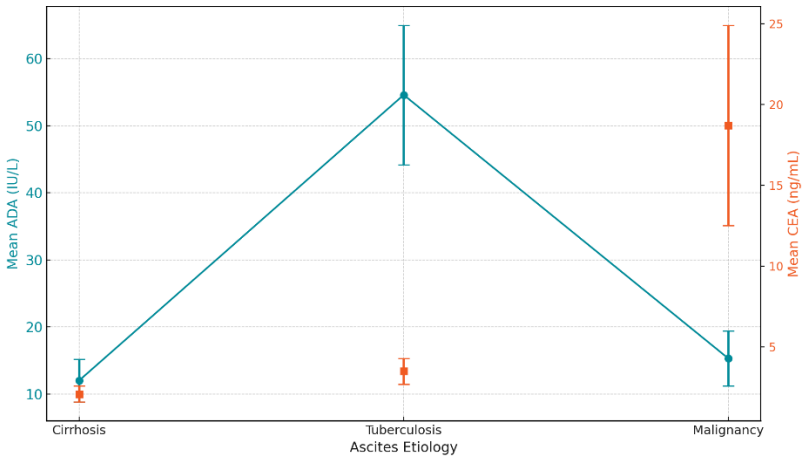

Figure 1 Comparative Ascitic ADA and CEA Levels Across Major Aetiologies

The dual-axis graph illustrates the differential diagnostic utility of ascitic ADA and CEA levels across cirrhosis, tuberculous peritonitis, and malignancy, revealing non-overlapping biomarker profiles that reinforce etiology-specific clinical suspicion. In tuberculosis, mean ADA concentration reached 54.6 IU/L (±10.4), significantly exceeding the thresholds observed in cirrhosis (12.0 ±3.2) and

malignancy (15.3 ±4.1), supporting ADA's sensitivity for infective ascites. Conversely, CEA levels were dramatically elevated in malignancy at 18.7 ng/mL (±6.2), compared to 3.5 ng/mL (±0.8) in tuberculosis and only 2.1 ng/mL (±0.5) in cirrhosis, suggesting strong discriminatory value for malignant effusions. The plotted confidence intervals confirm minimal overlap between high ADA in tuberculosis and high CEA in malignancy, emphasizing the specificity of each marker. Cirrhotic patients, by contrast, exhibited low values for both markers, further underscoring the transudative nature of their ascites. This pattern demonstrates how a combined interpretation of ADA and CEA facilitates rapid, non-invasive differentiation of ascites etiology—essential in resource-constrained settings where timely intervention in TB or malignancy can be lifesaving.

DISCUSSION

This prospective observational study offers valuable insights into the diverse etiological landscape of ascites in a high-burden, resource-limited South Asian setting. The predominance of cirrhosis (44.7%) as the leading cause aligns with global epidemiological patterns, where portal hypertension secondary to chronic liver disease remains the foremost etiology of peritoneal fluid accumulation (1). However, the remarkably high prevalence of tubercular peritonitis (24.4%) and malignant ascites (12.2%) observed in this study underscores regional differences that challenge assumptions drawn from Western cohorts, where heart failure and cirrhosis dominate the differential diagnosis (2). This divergence highlights the necessity of region-specific diagnostic frameworks that accommodate infectious and oncologic pathologies, particularly in tuberculosis-endemic regions such as Pakistan.

The clinical presentation of ascites in our cohort was consistent with previously reported patterns, where abdominal distension and pedal edema were nearly universal signs (3). However, the frequent occurrence of systemic symptoms such as weight loss, fever, and anorexia among patients with tuberculosis and malignancy accentuates the importance of a thorough symptom assessment to distinguish exudative from transudative etiologies. Comparative analysis reveals substantial concordance with studies from India and Southeast Asia that also report peritoneal tuberculosis rates exceeding 20% in hospitalized ascites patients (4,5). Moreover, the use of ascitic fluid adenosine deaminase (ADA) and GeneXpert MTB/RIF assays in this study demonstrated significant diagnostic yield for tubercular ascites. The ADA threshold of >32 IU/L, used in our analysis, is supported by meta-analyses showing pooled sensitivity and specificity values exceeding 90% in high-prevalence populations (6). The additive role of GeneXpert, which confirmed tuberculosis in 16.3% of cases, strengthens the case for molecular diagnostics in resource-limited but high-burden environments, where mycobacterial culture or laparoscopy is often unavailable or delayed (7).

Malignant ascites, while less prevalent than tuberculosis or cirrhosis, emerged as a clinically significant subgroup. Elevated ascitic CEA levels (>5 ng/mL) and cytological confirmation in selected cases provided strong diagnostic support. These findings mirror data from multi-ethnic Asian cohorts, where malignancy accounted for up to 15% of ascitic presentations, often secondary to gastrointestinal or ovarian cancers (8). The incorporation of tumor markers such as CEA adds practical value in differentiating malignant ascites, especially when cytology is equivocal or not feasible due to limited cellularity. Mechanistically, malignant ascites results from a combination of peritoneal carcinomatosis, lymphatic obstruction, and increased vascular permeability driven by tumor-derived cytokines—processes that are not identifiable by SAAG alone and thus require adjunctive testing (9).

The diagnostic utility of SAAG was reaffirmed in this study, with 60.9% of patients exhibiting values >1.1 g/dL, correlating strongly with cirrhosis and portal hypertension. However, reliance on SAAG alone would have misclassified a significant number of exudative ascites cases, as evidenced by the large proportion of tuberculosis and malignancy cases with low SAAG values. This underscores the importance of a multimodal diagnostic approach that integrates SAAG with ADA, GeneXpert, CEA, and imaging findings to reach an accurate diagnosis. Our use of transient elastography (FibroScan) for assessing hepatic stiffness in suspected cirrhotics also reflects a growing international trend toward noninvasive fibrosis assessment. The chosen cutoff of ≥12.5 kPa aligns with current guidelines and has been validated as a surrogate for histological cirrhosis in chronic liver disease (10).

Clinically, the study found hepatic encephalopathy to be the most frequent complication, particularly among cirrhotic patients, followed by gastrointestinal bleeding and renal impairment. These findings support existing literature that associates decompensated cirrhosis with multi-organ dysfunction and increased short-term mortality (11). The management patterns observed—particularly the high use of diuretics, albumin infusions, and paracentesis—reflect adherence to international ascites treatment guidelines (12). Notably, all tuberculosis patients received standardized anti-tubercular therapy, and those with malignant ascites were appropriately referred for oncology consultation, reinforcing a commitment to evidence-based care even within limited-resource contexts. The strengths of this study include its prospective design, large sample size, and comprehensive diagnostic workup using both conventional and advanced fluid assays.

This allowed for robust etiological classification and reduced recall and selection bias. However, several limitations must be acknowledged. As a single-center study, its findings may not be generalizable to primary care or rural settings where access to imaging and specialized tests is restricted. The absence of tissue biopsy in many malignancy and tuberculosis cases limits definitive histopathological confirmation. Furthermore, resource constraints precluded routine use of laparoscopy, immunocytochemistry, or full cytokine profiling, which could have improved diagnostic precision. Missing data for some biomarkers and reliance on indirect indicators (e.g., CEA and ADA thresholds) may introduce classification bias, although these limitations reflect real-world diagnostic conditions. Future studies should focus on developing and validating cost-effective, algorithm-driven diagnostic pathways for ascites in similar settings. Multicenter research with standardized data collection and incorporation of longitudinal outcomes would enhance

generalizability and allow risk stratification models for complications. Additionally, research into the role of biomarkers like calprotectin, vascular endothelial growth factor (VEGF), or novel molecular signatures could further refine differentiation of exudative ascites etiologies. Training programs in cytopathology and affordable access to point-of-care molecular diagnostics may also bridge diagnostic gaps. This study reinforces that while cirrhosis remains the principal cause of ascites in tertiary care settings, tuberculosis and malignancy account for a substantial minority of cases in South Asia and must be actively investigated using targeted diagnostic modalities. Reliance on SAAG alone is insufficient in this region. A multifaceted approach incorporating clinical judgment, imaging, fluid biochemistry, and molecular assays can markedly improve diagnostic accuracy and patient outcomes. These findings have critical implications for designing context-appropriate algorithms and capacity-building efforts in resource-constrained healthcare systems.

CONCLUSION

This prospective observational study conducted at Hayatabad Medical Complex highlights the heterogeneous etiological profile of ascites in a South Asian tertiary care setting, where cirrhosis remains the most prevalent cause, yet tuberculosis and malignancy collectively account for a significant proportion of cases. These findings underscore the need for context-specific diagnostic algorithms that extend beyond reliance on SAAG alone to include adjunctive tools such as ascitic ADA, GeneXpert MTB/RIF, CEA, and FibroScan for accurate etiological classification. Clinically, this has important implications for timely and appropriate management, guiding the use of anti-tubercular therapy, oncologic referral, or standard cirrhosis protocols based on precise diagnosis. From a healthcare perspective, integrating affordable molecular and biochemical diagnostics into routine practice could enhance early detection, reduce misclassification, and improve outcomes in resource-limited environments. Future research should focus on validating comprehensive, regionally-adapted diagnostic pathways that incorporate these findings to strengthen the clinical management of ascites in high-burden populations.

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