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Original Article

Diagnostic Evaluation of noninfective causes of Ascites: Clinical, Biochemical, Ascitic Fluid and Imaging Findings in a Hospital-Based Cohort

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ABSTRACT

Background: A common clinical manifestation with a wide range of etiologies is ascites. Although the Serum-Ascites Albumin Gradient (SAAG) is the industry standard for distinguishing portal hypertensive ascites, contextual validation is necessary for its integrated diagnostic yield in conjunction with basic imaging in settings with limited resources. **Methods & Materials:** This descriptive, cross-sectional study evaluated 70 adult patients with non-tubercular ascites admitted to a tertiary care hospital in Bangladesh. Patients underwent a standardized stepwise evaluation comprising clinical examination, biochemical analysis (including SAAG calculation), macroscopic fluid assessment, and abdominal ultrasonography. **Results:** Cirrhosis of the liver was the predominant etiology (50.0%, n=35), driven heavily by endemic viral hepatitis. Other major causes included congestive cardiac failure (17.1%), nephrotic syndrome (12.9%), and intra-abdominal malignancy (11.4%). The SAAG accurately categorized underlying hemodynamics, yielding high-gradient results (≥ 1.1 g/dL) in cirrhosis and cardiac ascites, and low-gradient results (< 1.1 g/dL) in malignancies. Macroscopic evaluation revealed that 75% of non-hepatocellular intra-abdominal malignancies produced hemorrhagic fluid. Ultrasonography demonstrated high clinical utility, identifying splenomegaly in 78.0% of the cirrhotic-spectrum cohort. **Conclusion:** Excellent diagnostic accuracy is achieved using a methodical, step-by-step diagnostic procedure that makes use of physical examination, SAAG, macroscopic fluid analysis, and basic ultrasonography. In settings with limited resources, this economical method is quite dependable for distinguishing non-infectious ascites.

Keywords: *Cirrhosis, Ascites, SAAG, Congestive Cardiac Failure, Nephrotic Syndrome.*

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INTRODUCTION

The abnormal buildup of free fluid in the peritoneal cavity is known as ascites, and it is a common symptom of many hepatics, abdominal, and systemic disorders. About 80% to 85% of ascites occurrences worldwide are caused by cirrhosis of the liver [1,2]. Severe portal hypertension, splanchnic arterial vasodilation, and consequent neurohumoral activation resulting in sodium and water retention are the main causes of the fluid buildup in cirrhosis [3]. The remaining portion of the illness burden is caused by non-hepatic etiologies, such as nephrotic syndrome, intra-abdominal cancers, and congestive heart failure (CCF) [4].

In the past, the biochemical assessment of ascitic fluid mostly depended on the concentration of total protein to categorize the fluid as an exudate or a transudate [5]. Diagnostic pathways were transformed with the introduction and validation of the Serum-Ascites Albumin Gradient (SAAG). The transudate/exudate categorization is essentially out of date in current hepatology guidelines because a SAAG ≥ 1.1 g/dL detects portal hypertension with an accuracy surpassing 97% [6-8].

Resource limitations and endemic infectious illnesses have a significant impact on the etiological landscape of ascites in underdeveloped countries such as Bangladesh [9]. Chronic liver disease (CLD) in the area is mostly caused by infections with the hepatitis B virus (HBV) and the hepatitis C virus (HCV) [10]. As a result, patients often arrive to tertiary care facilities with decompensated, advanced disease characterized by severe malnourishment, gastrointestinal varices, and extensive ascites [10]. Clinicians must rely on a strict, sequential diagnosis approach that uses physical examination, SAAG calculation, and abdominal ultrasonography in such resource-constrained settings, where sophisticated molecular and biomarker panels are frequently prohibitively expensive [7].

In order to validate the diagnostic yield of standard investigations in this particular epidemiological context, the goal of this study is to thoroughly assess the clinical, biochemical, ascitic fluid, and imaging profiles of a hospital-centered cohort of patients with non-tubercular ascites in Bangladesh.

METHODS & MATERIALS

Study Design and Patient Selection

A descriptive, cross-sectional observational study was conducted at the Department of Medicine, Shaheed Ziaur Rahman Medical College Hospital, Bogra, Bangladesh, over a 10-month period. The study enrolled consecutive adult patients (>18years) admitted with clinical or ultrasonographic evidence of ascites. Patients with critically unstable hemodynamics or ascites secondary to the spontaneous perforation of a gas-containing hollow viscus were excluded. To maintain a strict focus on the diagnostic differentiation of non-infectious portal hypertensive and systemic/malignant ascites, 30 patients diagnosed with tuberculous peritonitis (both isolated and superimposed on cirrhosis) were excluded from this specific analysis. The final analytical cohort comprised 70 patients.

Clinical and Laboratory Evaluation

A detailed clinical history and physical examination were recorded for each patient. Diagnostic paracentesis was routinely performed for macroscopic examination, cytological analysis, and biochemical evaluation (total protein and albumin). The SAAG was calculated by subtracting the

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ascitic fluid albumin concentration from the concurrent serum albumin concentration. Ascites with a SAAG ≥ 1.1 g/dL was classified as high-gradient, and ≤ 1.1 g/dL as low-gradient. Concurrent systemic blood panels included a complete blood count, erythrocyte sedimentation rate (ESR), liver function tests, and viral serologies (HBsAg and Anti-HCV).

Imaging

Whole-abdomen ultrasonography was utilized to detect hepatosplenomegaly, space-occupying lesions (SOL), and ascites volume. Chest radiography was performed to identify concurrent pleural effusions or cardiomegaly, and upper gastrointestinal endoscopy was conducted in selected patients to screen for esophageal varices.

RESULTS

Etiological and Demographic Profile

The analytical cohort of 70 patients with non-tubercular ascites demonstrated a male preponderance, with 46 males and 24 females (ratio of 1.92:1 overall). Cirrhosis of the liver was the most frequent etiology, accounting for 50.0% (n=35) of the cohort, followed by congestive cardiac failure (17.1%, n=12) and nephrotic syndrome (12.9%, n=9). The maximum age incidence for the non-tubercular cohort was between 41–50 years (31.4%).

Figure 1. Aetiological Distribution of Ascites in Non-Tubercular Cases (n = 70)

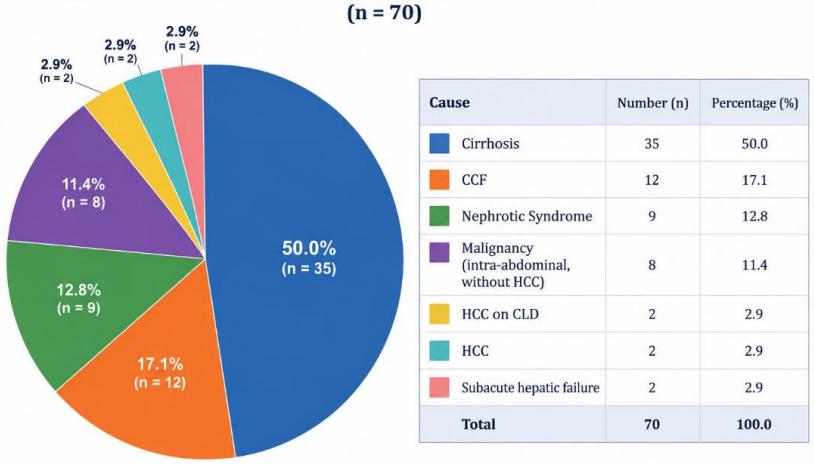


Figure 1 Etiological distribution of non-tubercular ascites cases (*n*=70).

Table II

Age distribution of the non-tubercular ascites cohort (*n*=70).

Age group (years)	n	%
18–20	6	8.6
21–30	10	14.3
31–40	11	15.7
41–50	22	31.4
51–60	14	20.0
≥61	7	10.0
Total	70	100.0

Of the 70 patients with non-tubercular ascites, 46 (65.7%) were male and 24 (34.3%) were female, giving an overall male-to-female ratio of 1.92:1. Cirrhosis of the liver was the most common diagnosis (n=35), with a male predominance (1.92:1). Congestive cardiac failure accounted for 12 cases and showed a male-to-female ratio of 1.40:1, while intra-abdominal malignancy without HCC had a ratio of 1.67:1. Nephrotic syndrome was the only condition with a female predominance (0.80:1). HCC-related groups were exclusively male, whereas subacute hepatic failure showed an equal sex distribution.

Table III

Sex distribution and male-to-female ratio in the non-tubercular cohort.

Diagnosis	Total n	Male n	Female n	Male:Female
Cirrhosis of liver	35	23	12	1.92:1
Hepatocellular carcinoma on CLD	2	2	0	Not calculable
Hepatocellular carcinoma	2	2	0	Not calculable
Subacute hepatic failure	2	1	1	1.00:1
Intra-abdominal malignancy without HCC	8	5	3	1.67:1
Congestive cardiac failure	12	7	5	1.40:1
Nephrotic syndrome	9	4	5	0.80:1
Total	70	46	24	1.92:1

Clinical Yield

Patients with cirrhosis uniformly presented with ascites (100%), predominantly accompanied by abdominal swelling (94.28%), fatigue (82.85%), loss of appetite (71.42%), and weight loss (45.71%). Stigmata of CLD were highly prevalent, including jaundice (51.42%), pedal edema (45.71%), hepatic facies (40%), splenomegaly (34.28%), and leuconychia (31.42%).

Table IV

Clinical manifestations of cirrhosis: symptoms and signs (*n*=35).

Symptoms	n	%	Signs	n	%
Abdominal swelling	33	94.3	Ascites	35	100.0
Fatigue	29	82.9	Anaemia	29	82.9
Loss of appetite	25	71.4	Under-nutrition	21	60.0
Weight loss	16	45.7	Jaundice	18	51.4
Scanty micturition	15	42.9	Pedal edema	16	45.7
Leg swelling	14	40.0	Hepatic facies	14	40.0
Yellow coloration of eye and urine	13	37.1	Splenomegaly	12	34.3
Abdominal pain	10	28.6	Leuconychia	11	31.4
Breathlessness	9	25.7	Gynaecomastia (male)	8	22.9
Fever	8	22.9	Fever	8	22.9
Vomiting of blood	8	22.9	Testicular atrophy	7	20.0
Black tarry stool	7	20.0	Hepatomegaly	6	17.1
Loss of libido	5	14.3	Collateral venous channels	6	17.1
Sleep alteration	5	14.3	Spider naevi	6	17.1
Loss of hair	4	11.4	Pleural effusion	8	22.9
Altered consciousness	4	11.4	Loss of hair	9	25.7
Facial swelling	2	5.7	Palmar erythema	4	11.4
Epistaxis	1	2.9	Flapping tremor	4	11.4
			Clubbing	3	8.6
			Dupuytren contracture	1	2.9
			Breast atrophy (female)	5	14.3

Conversely, all patients with CCF (100%) exhibited a distinct triad of dyspneic appearance, pedal edema, and raised jugular venous pressure, accompanied by hepatomegaly and tachycardia. In cases of intra-abdominal malignancy without HCC, 100% of patients reported loss of appetite, weight loss, and abdominal swelling.

Biochemical and Ascitic Fluid Analysis

Hepatic function evaluation in the cirrhotic cohort revealed elevated serum bilirubin (>1 mg/dL) in 51.4% of cases, raised SGPT (>40 U/L) in 65.7%, and altered albumin-globulin ratios (<1) in 100% of the tested cohort.

Table V

Selected liver function tests and serum proteins in cirrhosis.

Parameter	Category / threshold	n	%
Serum bilirubin	≤1 mg/dL	17	48.6
Serum bilirubin	>1 mg/dL	18	51.4
SGPT	10–40 U/L	12	34.3
SGPT	>40 U/L	23	65.7
SGOT	10–35 U/L	24	68.6
SGOT	>35 U/L	11	31.4
ALP	3–13 KAU/dL	31	88.6
ALP	>13 KAU/dL	4	11.4
Serum total protein	>6 g/dL	29	70.7
Serum total protein	5–6 g/dL	12	29.3
Serum albumin	3–2 g/dL	16	39.0
Serum albumin	<2 g/dL	25	61.0
Albumin:globulin ratio	<1	41	100.0

Ascitic fluid analysis demonstrated the diagnostic accuracy of the SAAG; ascites secondary to cirrhosis yielded high-gradient fluid (>1.1 g/dL), whereas malignancies yielded low-gradient fluid (<1.1 g/dL). Macroscopically, ascitic fluid was predominantly straw-colored in cirrhosis, but heavily haemorrhagic in intra-abdominal malignancies (present in 6 out of 8 cases).

Table VI
Ascitic fluid characteristics in cirrhosis, cardiac ascites and malignancy.

Condition	Gross appearance	Protein (g/dL), mean±SD	Albumin (g/dL)	SAAG	Interpretation	Malignant cells
Cirrhosis (n=35)	Straw 29; turbid 6	2.07±0.337	<3	>1.1	Usually transudative	Malignant cells absent
HCC on cirrhosis (n=2)	Straw 1; haemorrhagic 1	4.525±0.1058	>3.5	<1.1	Exudative range	Absent
HCC (n=2)	Straw 1; haemorrhagic 1	5.00±0.2828	>3.5	<1.1	Exudative range	Present in 1 case
Intra-abdominal malignancy without HCC (n=8)	Straw 2; haemorrhagic 6	4.175±0.483	>3.5	<1.1	Exudative in all cases	Present in 3 cases

Imaging and Endoscopy
Abdominal ultrasonography reliably identified portal hypertensive architectural changes, detecting splenomegaly in 78.0% (32 out of 41) of the cirrhotic-spectrum patients. Furthermore, upper gastrointestinal endoscopy performed on this specific cohort revealed that 48.8% possessed some degree of esophageal varices.

Table VII
Ultrasonographic and upper-GI endoscopic findings relevant to portal hypertension.

Investigation / finding	n	Denominator	%
USG: splenomegaly among cirrhotic-spectrum patients	32	41	78.0
USG: ascites among cirrhotic-spectrum patients	41	41	100.0
USG: hepatomegaly among cirrhotic-spectrum patients	14	41	34.1
Upper-GI endoscopy: esophageal varices	20	41	48.8
Upper-GI endoscopy: gastric growth	2	41	4.9
USG: hepatomegaly, all ascites cases	31	100	31.0
USG: splenomegaly, all ascites cases	32	100	32.0
USG: SOL in liver, all ascites cases	6	100	6.0
USG: matted lymph nodes, all ascites cases	9	100	9.0
USG: soft-tissue mass, all ascites cases	6	100	6.0

DISCUSSION

This study emphasizes how crucial it is to evaluate ascites using a methodical, step-by-step diagnostic approach, especially in medical settings with limited resources. Liver cirrhosis was the main cause of non-infectious ascites in this group (50%), which is in line with both local and international epidemiological data [1]. The significant HBsAg positivity rate (36.58%) among the cirrhotic patients is consistent with earlier research from Bangladesh that emphasizes the endemic character of HBV and its direct link to the development of portal hypertension and decompensated liver disease [9,10].

This study's main conclusion is that the SAAG is a better diagnostic metric than conventional protein-based classifications. Our results show that the SAAG properly classified both CCF and cirrhosis into the high-gradient (≥1.1 g/dL) category, whereas previous models had trouble correctly classifying cardiac ascites because of varying total protein concentrations [4,5,6]. This demonstrates that high hydrostatic pressures are the primary cause of fluid buildup under both circumstances [7]. On the other hand, low-gradient fluid (<1.1g/dL) was consistently produced by malignant ascites, which closely reflected the process of peritoneal exudation [8].

Additionally, a straightforward macroscopic assessment of the ascitic fluid at the bedside was very predictive. Hemorrhagic ascites was seen in 50% of HCC cases and 75% of non-HCC intra-abdominal tumors, making it a predominant characteristic of malignancies [11]. Clinicians can achieve high diagnostic accuracy without immediately relying on expensive advanced biomarker assays when combined with the high sensitivity of targeted ultrasonography, which reliably identified splenomegaly in 78% of cirrhotic patients and pinpointed hepatic space-occupying lesions [12,13].

There are significant clinical ramifications for poor countries. Healthcare organizations can prevent diagnostic delays and save needless costs on comprehensive molecular testing by closely following this strategy. In the end, a thorough physical examination combined with SAAG and simple imaging guaranties quick etiological classification, enabling prompt, etiology-specific therapeutic measures [1, 4].

CONCLUSION

A high level of clinical expertise combined with focused, economic research is necessary for the diagnostic assessment of ascites in settings with limited resources. This study shows that a structured diagnostic methodology that is based on abdominal ultrasonography, macroscopic fluid evaluation, and the Serum-Ascites Albumin Gradient (SAAG) is quite accurate in distinguishing between the causes of non-tubercular ascites. Due to its strong correlation with endemic viral hepatitis, cirrhosis continues to be the primary cause of ascites in Bangladesh, underscoring the importance of vigorous prophylactic screening. Clinicians may accurately differentiate between portal hypertensive and malignant effusions by standardizing this stepwise diagnostic route, which optimizes patient treatment and resource allocation without requiring expensive, prohibitive testing.

REFERENCES

1. Moore KP, Aithal GP. Guidelines on the management of ascites in cirrhosis. Gut. 2006;55(suppl 6):vi1-vi12.
2. Runyon BA. Management of adult patients with ascites due to cirrhosis: an update. Hepatology. 2009;49(6):2087-2107.
3. Hou W, Sanyal AJ. Ascites: diagnosis and management. Med Clin North Am. 2009;93(4):801-817.
4. European Association for the Study of the Liver. EASL Clinical Practice Guidelines for the management of patients with decompensated cirrhosis. J Hepatol. 2018;69(2):406-460.
5. Tarn AC, Lapworth R. Biochemical analysis of ascitic (peritoneal) fluid: what should we measure? Ann Clin Biochem. 2010;47(Pt 5):397-407.
6. Hoefs JC. Serum protein concentration and portal pressure determine the ascitic fluid protein concentration in patients with chronic liver disease. J Lab Clin Med. 1983;102(2):260-273.
7. Biggins SW, Angeli P, Garcia-Tsao G, et al. Diagnosis, Evaluation, and Management of Ascites, Spontaneous Bacterial Peritonitis and Hepatorenal Syndrome: 2021 Practice Guidance by the American Association for the Study of Liver Diseases. Hepatology. 2021;74(2):1014-1048.
8. Huang LL, Xia HH, Zhu SL. Ascitic Fluid Analysis in the Differential Diagnosis of Ascites: Focus on Cirrhosis and Malignancy. J Clin Transl Hepatol. 2014;2(1):58-64.
9. Mahtab MA, Rahman S, Karim MF, Khan M, Foster G, Solaiman S, et al. Epidemiology of liver cirrhosis in Bangladesh. Hepatobiliary Surg Nutr. 2014;3(1):31-36.
10. Hossain SM, Kabir F, Kamal SM, Chanda UC, Tarafder P. Epidemiological & Clinical Manifestation of Cirrhosis of Liver in a Tertiary Hospital. Medicine Today. 2020;32(1):21-24.
11. Goudarzi H, et al. Ascitic fluid analysis in the differential diagnosis of ascites. J Clin Transl Hepatol. 2013;1(1):10-15.
12. Colli A, Fraquelli M, Andreoletti M, et al. Severe liver fibrosis or cirrhosis: accuracy of US for detection—analysis of 300 cases. Radiology. 2003;227(1):89-94.
13. Fariña-Pérez LA, et al. The diagnostic value of ultrasound in ascites. Rev Esp Enferm Dig. 2017;109(10):707-714.