

## Original Article

# Clinical Profile of Ascites based on Presentation and Laboratory Findings: An Institutional Experience from Kathmandu, Nepal

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## Abstract

**Introduction:** Ascites is the fluid collection in the potential space of the peritoneal cavity. Alcoholic liver disease and intra-abdominal malignancy are two major etiologies behind it. Furthermore, diagnosis of tuberculous ascites should be thought of due to endemicity. Cirrhotic patients, at any time, during the course of the disease, invariably present with ascites, which is one of the marker of decomposition of the ongoing liver disease. In our context, etiology behind ascites and its correlation with symptoms and clinical findings not yet studied so the present study is conceptualized. **Methods:** This is a cross-sectional retrospective descriptive hospital-based record review of patients presented with ascites in a tertiary center of Kathmandu. One hundred fourteen patients with ascites under regular follow-up of Shree Birendra Hospital (SBH) were reviewed. Data regarding presenting complaints, examination findings, relevant investigations during the first visit, and final diagnosis were retrieved from our own record keeping, and recorded information were then evaluated. **Results:** Patients having ascites have myriad of symptoms and signs, the most common clinical feature icterus (74, 64.9%). Most of them were anemic at presentation. 42 (36.8%) had high blood urea and 36 (31.5%) with high creatinine suggesting approximately 30–40% of cases presented with deranged renal function test. 93 (81.5%) had raised total serum bilirubin. Similarly, PT/INR derangements were in 74 (64.9%) and 57 (50%) suggesting deranged liver function. Serum albumin was <3.5 g/deciliter in 83 (72.8%) cases. Among the patients studied, 80 (70.1%) had high SAAG suggesting transudative type of ascites and rest 34 had low SAAG suggesting exudative type of ascites. Overall assessment revealed that majority of patients (71.05%) had CLD as the cause of ascites. **Conclusions:** Ascites due to chronic liver disease was the main finding with etiology supported by laboratory findings. Significant numbers of the patients enrolled in the present study had deranged renal parameters in addition to deranged liver function parameters, which suggests that renal functions should also be properly taken care of in patients of ascites.

**Keywords:** Ascites, Liver cirrhosis, Serum ascetic albumin gradient

## Introduction

Ascites is the fluid collection in the peritoneal cavity. Different pathologies with unique pathomechanism give rise to ascites. Alcoholic liver disease, intra-abdominal malignancy, non-alcoholic cirrhosis, and malignancy with cirrhosis are common causes in descending sequence.<sup>[1]</sup> Ascites is one spectrum of liver cirrhosis and portal hypertension. Cirrhotic patients at a time invariably present with

ascites and are a marker of decompensation. In these cases, severity has to be evaluated and the case should be managed appropriately with salt restriction, diuretics, therapeutic paracenteses, or surgical shunt procedure alone or in combination.<sup>[2]</sup> Several gastrointestinal and ovarian malignancies present with ascites, and malignant ascites is a grave prognostic sign of the diseased individual with poor survival.<sup>[3,4]</sup> Due to the endemicity of tuberculosis in Nepal,

tuberculous peritonitis also needs to be kept in differential and culture growth which is the gold standard test for diagnosis tuberculous peritonitis.<sup>[5,6]</sup>

In our context, etiologies behind ascites and its correlation with symptoms and clinical findings are not yet studied and are still unknown, so the present study is conceptualized to evaluate the cases of ascites in terms of clinical features and laboratory investigations in our setting based on our hospital.

## Methods

This was a cross-sectional retrospective descriptive hospital-based record review of a tertiary center of Kathmandu. This non-invasive descriptive study was done over records of all new patients presented with ascites in the department of internal medicine over the past 3 years. One hundred fourteen cases of ascites under follow-up of SBH were reviewed. This record review was done over 3 months after the approval of local IRC of Nepalese Army Institute of Health Sciences. After approval from the internal medicine department and IRC, proper reviewing of the cases started. Data regarding presenting symptoms, examination findings, appropriate investigations including non-invasive serum-ascites albumin gradient during first visit, and final diagnosis were retrieved from our own record keeping and recorded information were evaluated. The recorded data were entered thoroughly and analyzed using SPSS version 22 and Excel computer program.

Among the patients of ascites presented in our center, we initially sent baseline blood investigations, namely, complete blood count, random blood sugar, liver biochemistry and liver and renal function tests, and serum electrolytes to evaluate the overall status of the patients along with the functional status of the liver. Furthermore, specifically to identify the etiology and to approach the case, we also sent hepatitis virus serology, diagnostic tapping of the ascitic fluid, ultrasound of the abdomen, and contrast-enhanced computed tomography of the abdomen when indicated. We followed FDA value for standard laboratory finding interpretation.

## Results

The mean age of the patients studied was  $54.89 \pm 12.99$  years with the minimum being 16 years and maximum being 82 years. Among all, 28 (24.6%) were females, while the rest 86 (75.4%) were males. Patients having ascites have myriad of other clinical features in addition to ascites like jaundice, weight loss, anorexia and abdominal pain etc. The most common clinical feature being icterus (74, 64.9%) followed by anorexia (53, 46.4%). Among clinical features, abdominal pain, fever, and icterus had significant association with ascites ( $P < 0.05$ ) [Table 1].

Among the patients of ascites evaluated, total leukocyte counts were raised in 24 (21.05%). Most (105, 92.1%) of the patients were anemic at presentation based on FDA demarcation. A

total of 46 (40.35%) cases were hyponatremic. 42 (36.8%) had high blood urea and 36 (31.5%) with high creatinine suggesting approximately 30–40% of cases presented with deranged renal function test. 93 (81.5%) had raised total serum bilirubin, while SGPT and SGOT were high in 94 (82.4%) and 72 (63.1%), respectively. Similarly, PT/INR derangements were in 74 (64.9%) and 57 (50%) suggesting deranged liver function. Serum albumin was  $<3.5$  g/deciliter in 83 (72.8%) cases. Among the patients studied, 80 (70.1%) had high SAAG suggesting transudative type of ascites and rest 34 had low SAAG suggesting exudative type of ascites. Table 2 presents the findings of the laboratory assessment of the cases of ascites.

Overall assessment revealed that most of the patients (81, 71.5%) had chronic liver disease as the culprit of ascites. Among them 5 had chronic hepatitis B virus-related hepatitis while rest 76 had alcohol-related liver disease. Other causes of ascites were tubercular peritonitis, right heart failure, and intra-abdominal malignancies including carcinoma stomach, carcinoma ovary, hepatocellular carcinomas, and pancreatic cancer.

Most of the cases of CLD had transudative type of ascites (high SAAG), and in other cases, it was exudative (low SAAG), and the  $P$ -value was correlated [Table 3].

## Discussion

Ascites is a lethal presentation, with myriad of cause behind. This presentation is common all over the world in medical practice. Its early detection is required to ensure effective management without any complications.

In our case, among 114 cases of ascites studied, 71.05% were due to chronic liver disease, while 15.7% were due to abdominal malignancies. Other studies also show somehow similar results with CLD representing about 80% of the cases while malignant ascites accounting 10% of the cases.<sup>[7,8]</sup> In contrast, another study from northern India showed cirrhosis to be the predominant cause of ascites (60.78%), followed by tuberculosis (15.68%), and rest 37% being other causes.<sup>[9]</sup>

Similarly, another Indian study concluded cirrhosis and tuberculous ascites being common etiology of ascites.<sup>[10]</sup>

The overall laboratory parameters were not assessed in the previous studies to accurately depict the clinical condition of the patient, whereas the present study attempts to show the clinical profile of the ascites patients to predict the physiological status of the patients. This study showed about 40% of the cases had hyponatremia and 30–40% of the cases had deranged renal function which suggests that fluid and electrolyte balance as well as dose of medications such as diuretics need to be calculated meticulously. Our data showed the deranged liver function in 60–80% of the cases,

**Table 1: Sign and symptoms at presentation among cases of ascites**

Variables	Features	CLD	Right heart failure	Abdominal malignancies	Tubercular peritonitis	Total	P value
Weight loss	No	58	5	10	3	76	0.067
	Yes	23	1	8	6	38	
Pruritus	No	74	5	17	9	105	0.658
	Yes	7	1	1	0	9	
Anorexia	No	46	3	10	2	61	0.266
	Yes	35	3	8	7	53	
Abdominal pain	No	49	6	7	3	65	0.024
	Yes	32	0	11	6	49	
Myalgia	No	72	5	14	8	99	0.639
	Yes	9	1	4	1	15	
Nausea and vomiting	No	46	5	9	6	66	0.497
	Yes	35	1	9	3	48	
Fever	No	66	6	13	4	89	0.036
	Yes	15	0	5	5	25	
Dark-colored urine	No	65	6	12	9	92	0.117
	Yes	16	0	6	0	22	
Pallor	No	60	5	13	6	84	0.909
	Yes	21	1	5	3	30	
Splenomegaly	No	64	5	15	9	93	0.486
	Yes	17	1	3	0	21	
Icterus	No	22	4	6	8	40	0.0009
	Yes	59	2	12	1	74	
Leukonychia	No	74	5	16	9	104	0.692
	Yes	7	1	2	0	10	
Spider naevi	No	71	6	18	9	104	0.215
	Yes	10	0	0	0	10	
Hepatomegaly	No	61	5	14	9	89	0.393
	Yes	20	1	4	0	25	

predisposition to hypoglycemia as well as bleeding episodes either due to rupture esophageal varices in cirrhotic cases or due to deranged clotting profile as depicted by PT/INR values. Hence, these simple parameters should also be taken care of when a case of ascites present.

Of the patients studied, 80 (70.1%) had transudative, and rest 34 (29.9%) had exudative type of ascites. Similar findings were shown by a study from Bihar, India.<sup>[11]</sup> SAAG is the important test in patients in whom cause of ascites is still need to be rectified. SAAG value strongly correlates with the etiology, whether it is due to CLD or other pathology like malignancies and whether the ascites is transudative or exudative.<sup>[11,12]</sup> In our study, among the CLD patients, most of them had transudative ascites while 12 patients had exudative ascites, indicating concurrent SBP, where SAAG findings correlates the diagnosis ( $P = 0.000$ ). Similarly, among patients with coexisting heart failure with TB peritonitis, SAAG was low. SAAG was high in patients with intra-

abdominal malignancy overlapping with CLD. All tubercular peritonitis patients had exudative form of ascites.

The findings obtained from this study are inferred from single-centered small-sized cross-sectional study, so clinical and laboratory findings need to be studied in larger population to put concluding remarks to guide approach of the ascitic patients.

### Conclusions

The study highlights chronic liver disease as the most common culprit for ascites, which follows abdominal malignancies. It also correlates etiology of ascites with the simple and non-invasive laboratory parameters like serum albumin ascites gradient among others. Significant numbers of the patients had deranged renal function on top of deranged liver function, so these parameters need to be taken care of.

**Table 2: Laboratory parameters**

Variables	Features	CLD	Right heart failure	Abdominal malignancies	Tubercular peritonitis	Total	P value
Total count	<4000	3	1	4	0	8	0.050
	Normal (4–11000)	62	5	9	6	82	
	>11000	16	0	5	3	24	
Hemoglobin	M (14–17.5) F (12.3–15.3)	8	0	1	0	9	0.602
	Anemic	73	6	17	9	105	
Random blood sugar	Normal (99–140)	44	4	8	6	62	0.691
	<99	23	2	6	3	34	
	>140	14	0	4	0	18	
Urea	Normal (20–40)	52	1	12	7	72	0.088
	High	29	5	6	2	42	
Creatinine	Normal (.6–1.1)	55	2	14	7	78	0.210
	High (>1.1)	26	4	4	2	36	
Na+	Normal (135–147)	43	3	13	8	67	0.401
	Low (<135)	37	3	5	1	46	
	High (>147)	1	0	0	0	1	
K+	Normal (3.5–5.5)	69	5	17	9	100	0.626
	Hypokalemia	9	1	0	0	10	
	Hyperkalemia	3	0	1	0	4	
Total serum bilirubin	Normal (<1)	4	4	6	7	21	0.000
	High (>1)	77	2	12	2	93	
Direct serum bilirubin	Normal (<0.4)	5	3	7	6	21	0.000
	High (>0.4)	76	3	11	3	93	
SGPT	Normal (0–30U/L)	10	3	5	2	20	0.062
	High (>30U/L)	71	3	13	7	94	
SGOT	Normal (0–40)	21	5	10	6	42	0.001
	High (>40 U/L)	60	1	8	3	72	
ALP	Normal (50–160)	48	5	11	3	67	0.267
	High (>160 U/L)	33	1	7	6	47	
Prothrombin	Normal (9.5–13.5)	21	4	9	6	40	0.010
	High (prolonged)	60	2	9	3	74	
INR	Normal (<1.3)	29	4	15	9	57	0.000
	Prolonged	52	2	3	0	57	
Serum protein	Normal (6.3–8)	42	5	3	6	56	0.0083
	Low (<6.3)	39	1	15	3	58	
Serum albumin	Normal (3.5–5.5)	24	2	4	1	31	0.624
	Low (<3.5)	57	4	14	8	83	
HbsAg	Negative	77	6	17	9	109	0.848
	Positive	4	0	1	0	5	
Ascitic protein	>2.5 g/dl	52	3	11	0	66	0.003
	≥2.5 g/dl	29	3	7	9	48	
SAAG	Exudative (≤1.1)	12	4	9	9	34	0.000
	Transudative (>1.1 mg/dl)	69	2	9	0	80	

**Table 3: SAAG correlating with diagnosis**

Diagnosis	Exudative	Transudative	Total	P value
CLD	12	69	81	0.000
Congestive heart failure	4	2	6	
Abdominal malignancies	9	9	18	
Tubercular peritonitis	9	0	9	
Total	34	80	114	

**References**

1. Khan J, Pikkarainen P, Karvonen AL, Mäkelä T, Peräaho M, Pehkonen E, *et al.* Ascites: Aetiology, mortality and the prevalence of spontaneous bacterial peritonitis. *Scand J Gastroenterol* 2009;44:970-4.
2. Biecker E. Diagnosis and therapy of ascites in liver cirrhosis. *World J Gastroenterol* 2011;17:1237.
3. Ayantunde AA, Parsons SL. Pattern and prognostic factors in patients with malignant ascites: A retrospective study. *Ann Oncol* 2007;18:945-9.
4. Sangisetty SL, Miner TJ. Malignant ascites: A review of prognostic factors, pathophysiology and therapeutic measures. *World J Gastrointest Surg* 2012;4:87.
5. Vardareli E, Kebapçı M, Sarıcam T, Pasaoglu Ö, Acikalin M. Tuberculous peritonitis of the wet ascitic type: Clinical features and diagnostic value of image-guided peritoneal biopsy. *Dig Liver Dis* 2004;36:199-204.
6. Sanai FM, Bzeizi KI. Systematic review: Tuberculous peritonitis—presenting features, diagnostic strategies and treatment. *Aliment Pharmacol Ther* 2005;22:685-700.
7. Hwangbo Y, Jung JH, Shim J, Kim BH, Jung SH, Lee CK, *et al.* Etiologic and laboratory analyses of ascites in patients who underwent diagnostic paracentesis. *Korean J Hepatol* 2007;13:185-95.
8. Ayantunde AA, Parsons SL. Pattern and prognostic factors in patients with malignant ascites: A retrospective study. *Ann Oncol* 2007;18:945-9.
9. Mehra D, Thakur D, Sultania S, Chaturvedi A, Agarwal G, Kumar A. Etiology of ascites in adults living in Rohilkhand region, India: A hospital-based study. *Int J Adv Res* 2016;2:1-4.
10. Kumar B, Sharma B, Raina S, Sharma N, Gupta D, Mardi K. Etiology of ascites in adults living in the hills of Himachal Pradesh, India: A hospital-based study. *Chrimed J Health Res* 2016;3:41.
11. Suman S, Jyoti D, Agrawal PK, Bhattacharya BK. Clinicopathological correlation of serum ascites albumin gradient with ascitic fluid total protein in patients of ascites with portal hypertension attending a tertiary care hospital in Eastern Bihar, India. *Int J Adv Med* 2017;4:842-6.
12. Tarn AC, Lapworth R. Biochemical analysis of ascitic (peritoneal) fluid: What should we measure? *Ann Clin Biochem* 2010;47:397-407.