Comparison of Ascitic Fluid Analysis Profile in Patients with Cirrhotic and Non-Cirrhotic Ascites at Adam Malik Hospital Year 2023

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Abstract- Ascites is a pathological fluid accumulation within the peritoneal cavity. Ascites is the most common complication of cirrhosis and occurs in approximately 50% of patients with decompensated cirrhosis within 10 years. Diagnostic paracentesis with appropriate ascitic fluid analysis should be performed in all patients hospitalized with ascites before undergoing therapy to rule out causes of ascites other than cirrhosis. This study aims to analyze the differences in ascitic fluid analysis profiles in patients with cirrhotic and non-cirrhotic ascites at Adam Malik Hospital, using an analytical observational approach with a case control design. Study consisted of 142 patients and found significant differences in laboratory parameters and ascitic fluid analysis between cirrhotic and non-cirrhotic patients.

Index Terms- Cirrhotic ascites; Non-cirrhotic ascites; ascitic fluid analysis

I. INTRODUCTION

Ascites is the accumulation of pathological fluid within the peritoneal cavity. Ascites is the most common complication of cirrhosis and occurs in approximately 50% of patients with decompensated cirrhosis within 10 years.[1,2,3]

Ascites in cirrhosis is the result of a vicious cycle involving vasodilation of the splanchnic arteries, decreased effective blood volume (despite a compensatory increase in cardiac output), renal vasoconstriction resulting in sodium retention, and extracellular fluid retention.[6]

Routine ascitic fluid analysis should include ascites serum-albumin gradient (SAAG), total protein concentration, cell count and differential. Optional ascitic fluid analysis includes cholesterol, fluid culture, cytology, tumor markers, lactate dehydrogenase, adenosine deaminase (ADA), triglycerides, amylase, glucose, brain natriuretic peptide (BNP), etc. Ascites serumalbumin gradient as a more accurate indicator of portal hypertension. Lactate dehydrogenase (LDH), vascular endothelial growth factor (VEGF), and other tumor markers can help in differentiating between malignant and benign conditions. Glucose and adenosine deaminase levels may support the diagnosis of tuberculosis disease, and amylase levels may indicate the diagnosis of pancreatitis.[4,9]

Serum-albumin ascites gradient (SAAG) ≥ 1.1 g/dL defines a high albumin gradient) indicates a portal hypertension origin with 97% sensitivity. Accuracy decreases if serum albumin and ascitic fluid are not collected simultaneously, or if serum albumin is <1.1 g/d. Ascitic fluid with high SAAG (>1.1) was found to accumulate in conditions of liver cirrhosis (81%), heart failure (3%), hepatic vein occlusion, constrictive pericarditis and kwashiorkor, while fluid with low SAAG (<1.1) accumulated in conditions of primary or metastatic peritoneal carcinoma (10%), infections such as tuberculosis and spontaneous bacterial peritonitis (2%), pancreatitis (1%), serositis, nephrotic syndrome and hereditary angioedema.[10,11]

Diagnosing ascites to differentiate between cirrhotic and non-cirrhotic causes presents various challenges, both in terms of the

availability of diagnostic tools and the complexity of interpreting the results. Clinically, the symptoms of ascites are often similar regardless of the etiology, such as abdominal enlargement and edema, making it difficult to determine the cause based solely on clinical manifestations.[10,11]

The main parameter used is ascitic fluid total protein, where transudate has a level <2.5 g/dL, while exudate ≥2.5 g/dL. In addition, the Serum-Ascites Albumin Gradient (SAAG) is an important parameter, with values ≥1.1 g/dL indicating portal hypertension (usually cirrhosis-related), while values <1.1 g/dL are more suggestive of non-cirrhotic causes such as malignancy or peritoneal tuberculosis. Glucose levels in ascitic fluid can also be helpful; in transudates, glucose is usually >50 mg/dL, while in exudates, especially those caused by infection or malignancy, glucose levels may be lower due to consumption by inflammatory cells or cancer cells. Another parameter used is lactate dehydrogenase (LDH), where levels <225 U/L or ascitic fluid/serum LDH ratio <0.6 are more typical for transudate, while levels >225 U/L or ratio ≥0.6 are more indicative of exudate.[10,11]

Treatment of ascites is multi-modal including dietary sodium restriction, pharmacologic therapy, diagnostic and therapeutic paracentesis, and in certain cases transjugular intra-hepatic portosystemic shunts.[10]

II. METHODS

This case-control study analyzes ascitic fluid profiles in cirrhotic and non-cirrhotic ascites using medical records from Adam Malik Hospital. A minimum of 142 patients per group is required. Consecutive sampling is applied, measuring total protein, LDH, WBC count, and glucose. Statistical analysis includes Chi-Square, Fisher Exact, T-test, or Mann-Whitney, with significance at p<0.05. Ethical approval was obtained, ensuring informed consent and confidentiality.

III. RESULTS

This study at Adam Malik Hospital included 142 cirrhotic and non-cirrhotic ascites patients. Cirrhotic patients were older (median 56 vs. 51 years, p < 0.001) and predominantly male (54.2%), while non-cirrhotic patients had more females (61.3%). Non-cirrhotic ascites was mainly caused by malignancy, CKD, CHF, and TB.

Cirrhotic patients had lower hemoglobin, leukocyte, platelet, and albumin levels, along with higher liver enzymes and bilirubin, indicating hepatic dysfunction. Ascitic fluid in cirrhosis was mostly clear-yellow and transudative, whereas non-cirrhotic ascites had more exudates. WBC levels were mostly low, but non-cirrhotic cases had more elevated counts. Glucose levels varied, with more decreases in non-cirrhotic patients.

Table 1. Demographic Characteristics of Ascites Patients

Variable	Ascites Patients Non-Cirrhosis	Ascites Patients Cirrhosis	*p-value*
Age, years	51 (20-77)	56 (22-83)	<0,001 *
Gender			<0,001 *
Male	55 (38,7%)	99 (69,7%)	
Female	87 (61,3%)	43 (30,3%)	
Etiology	0 (%)	142 (50%)	N/A
Cirrhosis	71 (25%)	0 (0%)	
Malignancy	32 (11.3%)	0 (0%)	
CKD (Chronic Kidney Disease)	32 (11.3%)	0 (0%)	
CHF (Congestive Heart Failure)	7 (2.5%)	0 (0%)	
TB (Tuberculosis)		0 (0%)	

**Mann Whitney U Test; **Chi-square Test; CKD: Chronic Kidney Disease; CHF: Congestive Heart Failure; TB: Tuberculosis.*

Table 2. Laboratory characteristics of ascites patients

Variable	Ascites Patients Non-Cirrhosis	Ascites Patients Cirrhosis	*p-value*
Hemoglobin, g/dL	10.2 (7 – 15.4)	9.8 (6.4 – 15.9)	0.004*
Leukocytes, x10$^{3\mu L}$	9.38 (2.8 – 28.9)	7.65 (2.5 – 26.14)	0.001*
Platelets, x10^{6}/µL	2.73 (0.04 – 6.98)	1.85 (0.02 – 4.79)	< 0.001*
Albumin, g/dL	2.91 (1.43 – 4.11)	2.37 (1.01 – 3.98)	< 0.001*
SGOT, IU/L	32.5 (10 – 709)	67 (15 – 857)	< 0.001*
SGPT, IU/L	15 (5 – 345)	35.5 (7 – 241)	< 0.001*

Bilirubin, mg/dL	0.61 (0.1 – 18.3)	1.69 (0.22 – 14.7)	< 0.001*

**Mann Whitney U Test; SGOT: Serum Glutamic Oxaloacetic Transaminase; SGPT: Serum Glutamic Pyruvic Transaminase*.

Table 3. Fluid Chaaracteristics of Ascites Patients

Variable	Ascites Patients Non-Cirrhosis	Ascites Patients Cirrhosis	*p-value*
Macroscopic Appearance			<0.001*
Clear-yellow	62 (43.7%)	103 (72.5%)	
Cloudy-yellow	61 (43%)	24 (16.9%)	
Red	19 (13.4%)	15 (10.6%)	
Total Protein			<0.001*
<2.5 g/dL	60 (42.3%)	128 (90.1%)	
>2.5 g/dL	82 (52.7%)	14 (9.9%)	
LDH			<0.001*
<200 U/L	13 (9.2%)	63 (44.4%)	
>200U/L	129 (90.8%)	79 (55.6%)	
WBC Count			0.032
<250	103 (72.5%)	118 (83.1%)	
>250	39 (27.5%)	24 (16.9%)	
Glucose			<0.001*
Normal	103 (72.5%)	106 (73.6%)	
Increased	10 (7%)	31 (21.8%)	
Decreased	29 (20.4%)	5 (3.5%)	

IV. DISCUSSION

In this study, the characteristics of ascites patients with and without cirrhosis showed significant differences in several demographic and etiological aspects.

By group, ascites patients with cirrhosis had a higher median age with a p- value <0.001, suggesting that age may be an influential factor in the etiology of ascites.

Based on etiology, the cirrhosis group was predominantly male (69.7%), while the non-cirrhosis group was predominantly female (61.3%). This difference was statistically significant with a p-value <0.001. This difference may be attributed to different risk factors, such as alcohol consumption in men and non-cirrhotic etiologies such as malignancy or chronic kidney disease being more common in women. The etiology of ascites in the cirrhotic group was entirely due to liver cirrhosis (50%), while in the non-cirrhotic group, the most common cause was malignancy (25%), followed by CKD (11.3%), CHF (11.3%), and TB (2.5%).

Laboratory analysis showed that non- cirrhotic ascites patients had higher hemoglobin, leukocytes, platelets and albumin than cirrhotic patients (p-value <0.001). This reflects the impact of hypersplenism, decreased albumin synthesis and gastrointestinal bleeding in cirrhotic patients. Meanwhile, SGOT, SGPT and total bilirubin levels were lower in non-cirrhotic patients compared to cirrhotic patients (p-value < 0.001) indicating more severe hepatocyte damage.[43]

Analysis of the macroscopic characteristics of ascitic fluid showed marked differences. The majority of cirrhotic patients (72%) had clear-yellow ascitic fluid, while non-cirrhotic patients showed variations between clear-yellow and yellow-cloudy. This finding is in line with the study conducted by Zhang et al. (2020).[41]

Based on total protein levels, ascitic fluid in non-cirrhotic patients tends to be exudate, while in cirrhotic patients it is more often transudate. This is in line with research by Smith et al. (2021), where exudative ascites is associated with conditions such as tuberculous peritonitis or malignancy, while transudative ascites is more common in cirrhosis due to portal hypertension.[46] Based on lactate dehydrogenase (LDH) levels, non- cirrhotic patients have a greater distribution of exudates, while cirrhotic patients are dominated by transudate classification. This is in line with research from Johnson et al. (2022), who stated that the LDH ratio of ascitic fluid/serum is usually above 0.6 in exudative conditions.[47]

The white blood cell (PMN) count in non-cirrhotic patients is higher. This is often associated with infection or inflammation, such as spontaneous bacterial peritonitis in cirrhotic patients, as found by Brown et al. ([48]) Glucose levels in ascitic fluid showed that non- cirrhotic patients more often had decreased glucose levels than cirrhotic patients indicating bacterial infection or malignancy (Taylor et al., 2023). [49]

Analysis of the macroscopic and biochemical characteristics of ascitic fluid can help in differentiating the etiology of ascites, which is important for proper diagnosis and management.

V. CONCLUSION

Based on the research results described above, the conclusions that can be drawn are:

(1) Patients with cirrhotic ascites tended to be older than patients with non-cirrhotic ascites, with the majority being male, while the non- cirrhotic group had a more balanced gender distribution. The etiology of non-cirrhotic ascites is most often caused by malignancy, chronic kidney disease, congestive heart failure, and tuberculosis. (2) Macroscopically, cirrhotic ascites has a clear-yellow ascitic fluid, whereas noncirrhotic ascites more often has a yellow-grey appearance or is mixed with blood, depending on the underlying etiology. (3) Significant differences are found in cell count, total protein, glucose, and LDH levels. Cirrhotic ascites tends to be transudative with lower protein, LDH, and white blood cell levels, whereas in non-cirrhotic patients increased leukocytes are associated with infectious conditions or malignancy. (4) Cirrhotic ascites patients show lower levels of hemoglobin, leukocytes, platelets, and albumin, but have higher levels of SGOT, SGPT, and bilirubin, indicating liver damage.

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