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Original Research Article

Cytological and biochemical correlation of ascitic fluid with clinical features in a tertiary care center

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Abstract

Background: Ascites involves fluid accumulation in the abdominal cavity due to various underlying pathological conditions. Effective evaluation of ascitic fluid and it's possible etiology is crucial for accurate diagnosis and management, reducing uncertainties healthcare providers might face.
Materials and Methods: This study included 120 patients of both sexes with ascites. Ascitic fluid was collected and its physical properties determined. Microscopic examinations (Smears and cell blocks) were performed to identify cell types in the fluid. The findings were correlated with biochemical and clinical features to confirm the diagnosis.
Results: Among the patients studied, cases were assessed as non-diagnostic, negative for malignancy, atypical, suspicious for malignancy and positive for malignancy. Malignancy was detected in approximately 14.2% of cases. 81.6% cases were detected as negative for malignancy category with male predominance. The majority of cases (68%) were attributed to cirrhosis and congestive cardiac failure (CCF). A significant 74% of patients displayed a high serum-ascites albumin gradient (SAAG), indicating portal hypertension typically associated with liver cirrhosis and CCF. A strong correlation was found between high ascitic fluid total protein (AFTP) levels (≥2.5 g/dL) and conditions such as CCF and peritoneal carcinomatosis. Transudative ascites was present in 72% of patients & 28% demonstrated exudative ascites.
Conclusion: Analysis of ascitic fluid is essential for identifying the underlying causes of ascites. Important diagnostic parameters include SAAG & total protein levels. SAAG is critical for differentiating ascitic causes related to portal hypertension from other etiologies, providing better diagnostic accuracy than total protein alone.

Keywords: Ascitic fluid, Biochemical analysis, Clinical correlation

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1. Introduction

Ascites is the abnormal accumulation of fluid in the peritoneal cavity, typically a symptom rather than a disease.[1] The peritoneal cavity lined by the parietal and visceral layers of epithelium, normally contains up to 50 ml of fluid for lubrication.[2] Ascites occurs when the balance between plasma flow into and out of blood and lymphatic vessels is disrupted due to conditions like increased capillary permeability, increased venous pressure, decreased oncotic pressure or lymphatic obstruction.[3,4]

The most common cause, accounting for 85% of cases, is cirrhosis-related portal hypertension.[1,5,6,7] The remaining 15% are linked to non-cirrhotic conditions, including malignancies, infections and cardiac or renal failure.

Ascites may be asymptomatic in mild cases, while moderate ascites can cause abdominal distension and weight gain. Severe cases may lead to discomfort, hernias, restricted mobility and breathlessness due to diaphragmatic elevation.

The occurrence of symptoms, signs and lab evidence of infection in patients with ascites should also prompt

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paracentesis with ascitic fluid analysis.[8] Paracentesis with ascitic fluid analysis is the preferred diagnostic approach, distinguishing between transudative (non-inflammatory) and exudative (inflammatory) ascites.[8] Transudative ascites, associated with conditions like cirrhosis and congestive heart failure, results from serum filtration across an intact vascular wall.[9] Exudative ascites, often due to infection or malignancy, involves active fluid accumulation due to capillary damage.

Key biochemical parameters in ascites evaluation include the Serum-Ascites Albumin Gradient (SAAG) and Ascitic Fluid Total Protein (AFTP). SAAG helps differentiate between transudative and exudative ascites, with values >1.1 g/dl indicating portal hypertension (transudative) and <1.1 g/dl suggesting normal portal pressure (exudative).[10,11,12] Understanding these parameters is essential for narrowing the diagnosis and guiding appropriate treatment.

2. Materials and Methods

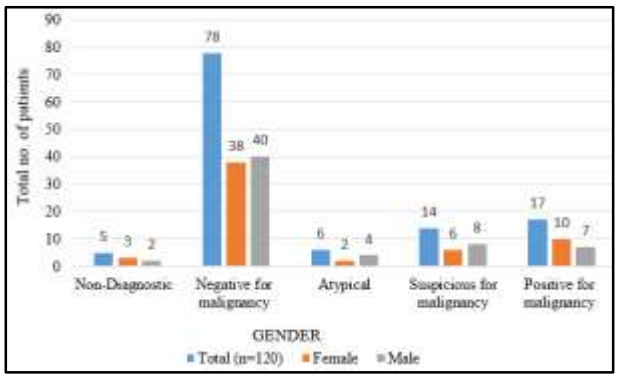
This observational study comprises ascitic fluid examination from 120 patients which were received in the department of pathology, tertiary care center, Gujarat. This study was approved by institutional ethics committee (GCSMC/EC/Project/APPROVE/641/2024) and was carried out in accordance with ethical principles. Ascitic fluid specimens received in the laboratory were subjected to both physical and microscopic examination. The features observed on physical examination of ascitic fluid were volume, colour and any other special character (such as turbidity, floating tissue fragments). First centrifugation was done at the rate of 2000 rpm for 10 minutes. Supernatant was discarded. The sediment was transferred to glass slide, spread, fixed and cell blocks were also prepared by fixed sediment method & were stained with hematoxylin and eosin stain. Smears and cell blocks were thoroughly examined under the microscope for the various types of cells. Biochemical markers such as SAAG and AFTP were done in 50 patients out of 120 patients for biochemical & cytological correlation and confirmation of diagnosis. The observations were categorized into Non-diagnostic, Negative for malignancy, Atypical, Suspicious for malignancy & malignant.

3. Results

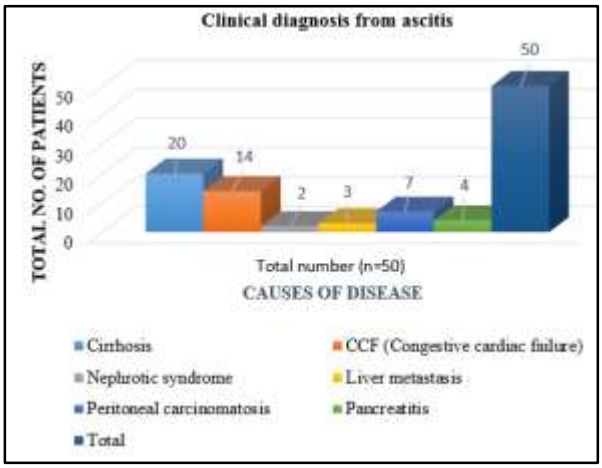
Total 120 patients with different parameters were included for comparison data analysis, out of which biochemical parameters were available for 50 patients, for comparison in this data analysis. **Table 1** shows that highest number of cases (28) are observed in the 51–60 years age group, indicating that cytological abnormalities are most common in this age range. A gradual increase in cases is observed from younger age groups, peaking in middle age (51–60 years), followed by a decline in older age groups. Overall, there is a slight male predominance (61 males vs. 59 females), though gender distribution is generally balanced across most age groups. As shown in **Graph 1** this study included five groups, Non-diagnostic, Negative for malignancy, Atypical, Suspicious for malignancy & Positive for malignancy. Out of 120 cases, 17 (14%) patients tested positive for malignancy, 14 (12%) tested suspicious for malignancy, 6 (5%) tested as atypical, 78 (65%) tested negative for malignancy and 5 (4%) were classified as non-diagnostic.

Table 1: Age wise and gender wise distribution of study participants

Age (Years)	Total (n=120)	Female	Male
11 to 20	3	1	2
21 to 30	9	5	4
31 to 40	21	9	12
41 to 50	22	11	11
51 to 60	28	13	15
61 to 70	23	11	12
71 to 80	13	8	5
81 to 90	1	1	0



Graph 1: Cytological diagnosis of examined ascitic fluids



Graph 2: Clinical diagnosis from ascitic fluid analysis

Table 2: Distribution of ascites on the basis of Serum Ascites Albumin Gradient (SAAG)

Etiology	Patient no. (SAAG>/=1.1)	Patient no. (SAAG<1.1)
Cirrhosis	20	0
CCF	14	0
Nephrotic syndrome	0	2
Liver metastasis	3	0
Peritoneal carcinomatosis	0	7
Pancreatitis	0	4
Total no. of patients=50		

Table 3: Distribution of ascites on the basis of AFTP (Ascitic fluid total protein)

Etiology	Patient no. (AFTP>/=2.5)	Patient no. (AFTP<2.5)
Cirrhosis	1	19
CCF	12	2
Nephrotic syndrome	0	2
Liver metastasis	0	3
Peritoneal carcinomatosis	7	0
Pancreatitis	4	0
Total no. of patients=50		

Table 4: Gender wise distribution of ascites on the basis of transudative and exudative analysis

Groups	Male	Female	Total (n=50)
Transudative ascites	18	18	36 (72%)
Exudative ascites	6	8	14 (28%)

Table 5: Distribution of ascites depending on the pathophysiology

High SAAG	No.	(%)	Low SAAG	No.	(%)
Cirrhosis	20	10%	Pancreatitis	4	2%
CCF	14	7%	Peritoneal carcinomatosis	7	3.5%
Liver metastasis	3	1.5%	Nephrotic syndrome	2	1%

Graph 2 shows the distribution of ascites among 50 study participants (with biochemical parameters) with varying clinical profiles. In the population investigated, the most prevalent diagnosis for ascites is cirrhosis, which affects 40% of patients. Congestive cardiac failure (CCF) is the second most common, with 28% cases. Nephrotic syndrome is the least common diagnosis, affecting only 4% of individuals. The remaining diagnoses, including liver metastases, peritoneal carcinomatosis and pancreatitis, account for varying percentages of the total.

Table 2 Shows distribution of ascites on the basis of SAAG (Serum Ascites Albumin Gradient**)** in 50 patients**.** In the study, individuals with ascites were divided into two groups based on the Serum Ascites Albumin Gradient (SAAG), using a cutoff of 1.1 g/dl. About 74% of participants were in the high SAAG group, while 26% were in the low SAAG group. High SAAG levels were primarily observed in patients with cirrhosis (20 cases), congestive cardiac failure (14 cases) and liver metastases (3 cases). In contrast, low SAAG ascites were found in 7 cases of peritoneal carcinomatosis, 2 cases of nephrotic syndrome, and 4 cases of pancreatitis.

Table 3 Shows that out of 50 patients, high AFTP (≥2.5 g/dl) was observed in conditions with exudative ascites, including CCF (12 cases), peritoneal carcinomatosis (7 cases) and pancreatitis (4 cases). Low AFTP (<2.5 g/dl) was predominant in conditions with transudative ascites, such as cirrhosis (19 cases), nephrotic syndrome (2 cases), and liver metastasis (3 cases).

Table 4 Outlines the gender wise distribution of patients with transudative ascites and exudative ascites in a cohort of 50 individuals. Transudative ascites was equally distributed between males (18) and females (18), comprising 36 cases (72%). Exudative ascites was slightly more common in females (8) than males (6), with 14 cases (28%).

Table 5 shows groups of ascites into high SAAG & low SAAG ascites. High SAAG ascites is predominantly caused by cirrhosis and CCF, which account for 17% of cases

combined. Low SAAG ascites is mainly linked to peritoneal carcinomatosis, with smaller contributions from pancreatitis and nephrotic syndrome. The data reflects distinct pathophysiological mechanisms, portal hypertension for high SAAG and increased capillary permeability or hypoalbuminemia for low SAAG ascites.

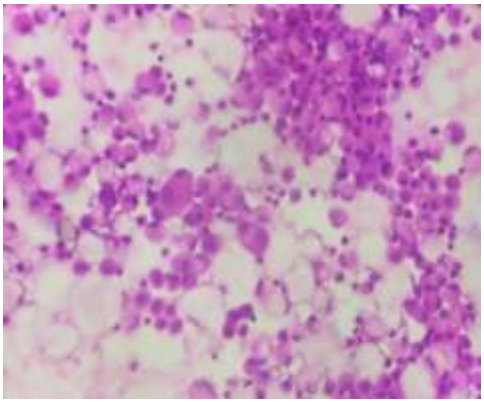


Figure 1: Reactive mesothelial cells, Ascitic fluid H&E Stain (40 x)

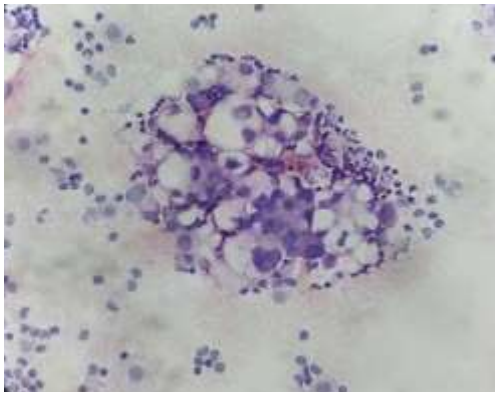


Figure 2: Cellular, Mesothelial cells, reactive mesothelial cells & few occasional foci of atypical cells, H&E Stain (40x).

Figure 2 shows clusters and sheets of mesothelial cells. These cells are generally polygonal, with a moderate amount of cytoplasm and round to oval nuclei. Some mesothelial cells show reactive changes, such as enlargement, prominent nucleoli and possible multinucleation. These features are typically seen in response to irritation or inflammation (e.g., infection, trauma).

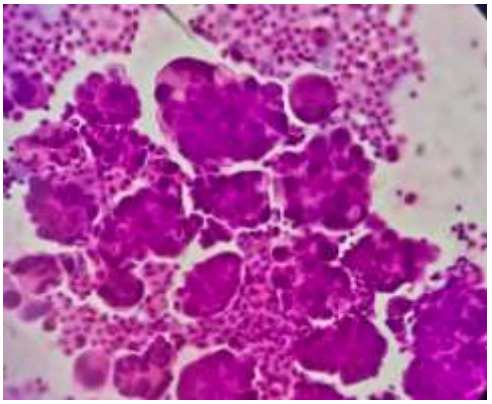


Figure 3: Malignant ascitic fluid. Background: Hemorrhagic, H & E Stain (40x).

Figure 3 shows typically large cells with irregular nuclear contour, prominent nucleoli and a high nuclear-to-cytoplasmic (N: C) ratio. The chromatin may be coarse or finely granular. The presence of blood in the background is noted, which is common in malignant ascitic fluid due to tumor infiltration or associated vascular damage.

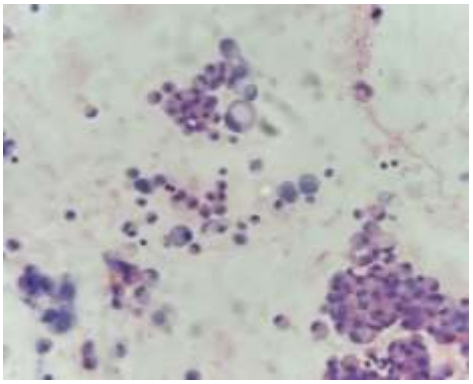


Figure 4: Isolates neoplastic cells (gastric adenocarcinoma). Ascitic fluid. H&E Stain stain, 40x

Figure 4 shows large cells with high nuclear-to-cytoplasmic ratio, hyperchromatic nuclei with irregular nuclear contours, prominent nucleoli, cytoplasmic vacuolation (may contain mucin), clumping or dissociation (Isolated cells often reflect a metastatic process).

Table 6: Comparison study data analysis table

Category	Our Study	Mohsan Subhani et al[13] 2022	AK Seth et al[14] 2021	Arvindan R et al[15] 2021	Karthik Selvaraju et al,[16] 2020
Sample Size	120	164	50	100	100
Age Group	51–60 years (23%)	>50 years (86.6 %)	51-60 years	31-50 years	31-50 years
Gender Distribution	51 %males, 49 females	51.3 % males, 48.7 % females	68 % males, 32 % females	94.4 % males, 5.6 % females	68 % males, 32 % females

Ascitic Fluid (SAAG)	High SAAG (74%), Low SAAG (26%)	High SAAG (72.4%), Low SAAG (27.6%)	High SAAG (94.3%), Low SAAG (5.7%)		High SAAG (69%), Low SAAG (31%)
Ascitic Fluid (AFTP)	High AFTP (48%), Low AFTP (52%)	-	-	High AFTP (46%), Low AFTP (54%)	High AFTP (48%), Low AFTP (52%)
Cytological Findings	65% negative for malignancy, 14% positive	29.3 % positive for malignancy	-	-	-
Primary Causes of Ascites	Cirrhosis (95%), CCF (28%)	Cirrhosis (54.9%), Malignancy (29.3 %)	Cirrhosis (70 %),	Cirrhosis	Cirrhosis (53 %)
Transudative Ascites	72% (mainly cirrhosis, CCF)	-	50% (Mainly cirrhosis)	54 %	52 %
Exudative Ascites	28% (mainly malignancy-related)	-	50 % (Cirrhosis, malignancy)	46 %	48 %
Diagnostic Significance of SAAG	High SAAG for portal hypertension	High SAAG for portal hypertension	High SAAG for portal hypertension	High SAAG for portal hypertension	High SAAG for portal hypertension

4. Discussion

This study provides a comprehensive analysis of the etiology and diagnostic parameters of ascites in a cohort of 120 patients, focusing on cytological findings, biochemical markers such as SAAG and AFTP, and their role in differentiating between transudative and exudative ascites. The predominance of cases in the 51–60 years age group (23%) aligns with studies indicating that middle age is a critical period for developing liver-related and malignant conditions associated with ascites.[13,14] Gender distribution was nearly balanced (61 males vs. 59 females), which is consistent with previous studies that show a slight male predominance in liver cirrhosis cases due to higher alcohol consumption among males in some populations. Notably, the current study and others reported a predominance of high SAAG levels (ranging from 69% to 94.3%), reinforcing its diagnostic utility in identifying ascites due to portal hypertension.[14] AFTP values, where available, showed a near-equal split between high and low levels, indicating its supplementary role in differentiating transudative and exudative ascites. Cytological findings, reported in two studies, revealed varying malignancy rates, with our study showing 14% positivity compared to 29.3% in Mohsan Subhani et al.[13] The distribution of transudative versus exudative ascites aligned with these findings, with most cases being transudative due to cirrhosis or CCF, and exudative ascites associated with malignancy.[13,16] Overall, the consistent recognition of high SAAG as a marker for portal hypertension across all studies highlights its pivotal role, while other parameters like AFTP, cytology, and clinical context further refine the diagnostic approach to ascites.

5. Conclusion

This study highlights the diagnostic value of ascitic fluid analysis in determining the etiology of ascites. Serum-Ascites Albumin Gradient (SAAG) emerged as the most effective marker, with high SAAG (≥1.1 g/dl) indicating portal hypertension in conditions like cirrhosis and congestive cardiac failure, and low SAAG (<1.1 g/dl) with high AFTP suggesting malignancy or inflammation. Transudative ascites, primarily due to cirrhosis, accounted for the majority of cases, with a nearly balanced gender distribution but slightly higher prevalence in males. Exudative ascites, linked to malignancy and inflammatory conditions, showed a slight female predominance. Routine analysis including SAAG, AFTP, cell count, and cytology is essential for accurate diagnosis and effective management of ascites.

6. Data & Material Availability

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7. Author's Contribution

All the authors including corresponding author have contributed equally towards data collection, data analysis & preparation of the draft and approval of the final manuscript of this article.

8. Source of Funding

None.

9. Conflict of Interests

None.

10. Acknowledgements

None.

References

1. Runyon BA. Care of patients with ascites. *N Engl J Med*. 1994;330(5):337–42. doi: 10.1056/NEJM199402033300508.
2. Nguyen GK. Essentials of fluid cytology. 2010. https://pathology.ubc.ca/files/2012/06/FLUIDCYTOLOGYBook09R1.pdf
3. Tarn AC, Lapworth R. Biochemical analysis of ascitic (peritoneal) fluid: what should we measure?. *Ann Clin Biochem*. 2010;47(Pt 5):397–407. doi: 10.1258/acb.2010.010048.
4. Ginès P, Cárdenas A, Arroyo V, Rodés J. Management of cirrhosis and ascites. *N Engl J Med*. 2004;350(16):1646–54. doi: 10.1056/NEJMra035021.
5. Mahmood G, Debnath CR, Mandal AK. Evaluation of 100 cases of ascites. *Mymensingh Med J*. 2009; 18(1):62–6.
6. Khan FY. Ascites in the state of Qatar: etiology and diagnostic value of ascitic fluid analysis. Singapore Med J. 2007;48(5):434–9
7. Hwangbo Y, Jung JH, Shim J, Kim BH, Jung SH, Lee CK et al. Etiology and laboratory analysis of ascites in patients who underwent diagnostic paracentesis. *Korean J Hepatol*. 2007;13(2):185-95.
8. Turnage RH, Li DLB, McDonald JC. Abdominal wall umbilicus, peritoneum, mesenteries, omentum. In: Sabiston Textbook of Surgery – The biological basis of modern surgical practice. 17th edn. Philadelphia: Elsevier Saunders; 2004:1182-6
9. Koss LG. Effusions in the absence of cancer and effusions in the presence of cancer. In: Diagnostic Cytology and its Histopathological Basis. 3rd edn. Philadephia: J.B. Lippincott Company; 1979;2:878-952.
10. 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):260–73.
11. Pare P, Talbot J, Hoefs JC. Serum-ascites albumin concentration gradient: a physiologic approach to the differential diagnosis of ascites. *Gastroenterology*. 1983;85(2):240–4.
12. Harjai KJ, Kamble MS, Ashar VJ, Anklesaria PS, Ratnam KL, Abraham P. Portal venous pressure and the serum-ascites albumin concentration gradient. *Cleve Clin J Med*. 1995;62(1):62-7. doi: 10.3949/ccjm.62.1.62.
13. Subhani M, Sheth A, Palaniyappan N, Sugathan P, Wilkes EA, Aithal GP. Diagnostic accuracy of serum ascites albumin gradient (SAAG) in a contemporary unselected medical cohort. *J Int Med Res*. 2022;50(11):3000605221140310. doi: 10.1177/03000605221140310.
14. Seth AK, Rangarao R, Pakhetra R, Baskaran V, Rana P, Rajamani S. Accuracy of serum - Ascites albumin gradient in the aetiological diagnosis of ascites. *Med J Armed Forces India*. 2002;58(2):124-6. doi: 10.1016/S0377-1237(02)80044-2. Epub 2011 Jul 21.
15. Aravindan R, Noorul Ameen KH. Comparison of the diagnostic accuracy of serum ascites albumin gradients (SAAG) with the traditional marker-ascitic fluid total protein. *J Res Med Dent Sci*. 2021;9(5):271–5
16. Selvaraju K, Sridevi M, Analysis of ascitic fluid in differentiating transudate versus exudate - in a tertiary care centre. *Indian J Pathol Oncol*. 2020;7(1):137-42.

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