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Recurrent Ascites in Hemodialysis Patients: Clinical Features, Causes and Outcomes

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Abstract

The study aims to determine prevalence and describe the clinical characteristics, causes and outcomes of hemodialysis patients with recurrent ascites. The study involved multiple hemodialysis centers both hospital-based and free-standing units, and the data was collected from January 2018 to June 2023. All patients on maintenance hemodialysis (at least 3 months) and age > 18 years with recurrent ascites (three or more episodes of ascites within a 12-month period) were included. Frequencies and percentages were computed for different categorical variables. There were 32 individuals with recurrent ascites out of the total 279 hemodialysis patients (11.4%). Based on their baseline characteristics (Table 1), there was a higher proportion of males and glomerulonephritis as the cause of the end stage renal disease in the recurrent ascites group. Hemodialysis patients without recurrent ascites have a significantly higher proportion of diabetes as the etiology of kidney disease. Based on their initial ascitic fluid analysis, the most common cause of the recurrent ascites was nephrogenic, followed by congestive heart failure. There were slightly more individuals with low serum ascitic albumin gradient. Only 4 patients (12.5%) had a positive bacterial culture while 2 patients were diagnosed with tuberculosis based on Gene Xpert testing. There is no difference as to the proportion of hospitalizations and mortality among those with and without recurrent ascites. Recurrent ascites is a common finding among hemodialysis patients. The end stage kidney disease still accounts for most of the causes. While it did not cause additional harmful risk, it causes discomfort and difficulty among patients. The need for other management options beside repeated paracentesis is still wanting.

Keywords

Hemodialysis, Infection, Cardiovascular

1. Introduction

Ascites, the abnormal accumulation of fluid in the peritoneal cavity, is a common complication in patients undergoing hemodialysis [1]. Recurrent ascites in hemodialysis patients poses a significant clinical challenge and may be associated with various underlying conditions and complications. This condition is particularly significant in patients with advanced chronic kidney disease (CKD), where the kidneys' ability to excrete excess fluid is severely compromised, leading to fluid overload [2]. Ascites in hemodialysis patients can be refractory and challenging to manage, often requiring repeated interventions such as paracentesis and albumin infusions [3].

The causes of ascites in hemodialysis patients are diverse and may include volume overload, liver disease, heart disease, peritonitis, protein depletion, and idiopathic factors [4]. Recurrent ascites in hemodialysis patients may lead to complications such as sepsis [5]. The management of refractory ascites in hemodialysis patients has been explored, with studies investigating alternative treatment approaches such as nocturnal home hemodialysis, which has shown promising results in resolving refractory ascites [6]. Additionally, the use of peritoneal dialysis systems with extracorporeal ultrafiltration by hemodialysis dialyzers has been proposed as a potential management strategy for refractory ascites in hemodialysis patients [7]. The pathophysiology of chronic hemodialysis-related ascites involves complex interactions between fluid balance, peritoneal permeability, and underlying comorbidities. Understanding the mechanisms underlying the development of ascites in hemodialysis patients is crucial for optimizing management strategies and improving patient outcomes.

The exact prevalence of recurrent ascites in hemodialysis patients is not extensively documented, but some studies suggest that it can be a common complication, particularly in those with advanced CKD or end-stage renal disease (ESKD) [8]. This study hopes to determine the prevalence of recurrent ascites in hemodialysis units and look at its clinical profile and etiology.

2. Methodology

2.1. Study Design and Participants

This study is a retrospective cohort design to investigate the clinical profile, etiology, and outcomes of recurrent ascites among hemodialysis patients at various units. The study period will encompass January 2018 to June 2023. Adult patients (≥18 years old) undergoing chronic hemodialysis for at least 3 months are included. The episode of ascites should be diagnosed by clinical assessment (abdominal distension, shifting dullness, fluid wave) and/or imaging (ultrasound, CT scan) with ascitic fluid analysis. There also should have at least one documented recurrence of ascites after initial treatment. Excluded cases are those who have episode of ascites prior to start of hemodialysis, those with incomplete records and with isolated episode of ascites.

2.2. Interventions

All patients with ascites underwent a percutaneous diagnostic ascitic tap, under ultrasound guidance. All patients had baseline hematology and biochemistry investigations as well as serology for hepatitis viruses. Ascitic fluid was routinely sent for total and differential leucocytes counts, biochemistry, culture, acid fast bacilli (AFB) stain and AFB culture. Serum-ascites albumin gradient (SAAG) was calculated for all patients, with particular emphasis on concomitant serum albumin measurement.

2.3. Data Collection and Outcomes

Data will be collected from the patient records of participating hemodialysis units, both hospital-based and free-standing units. The following variables will be collected: Demographics (Age, sex), medical history (Underlying kidney disease, comorbidities), previous treatments for ascites (e.g., paracentesis, diuretics), medications, hemodialysis characteristics (Duration of hemodialysis, type of dialyzer, vascular access), etiology of ascites, and other laboratory data.

The primary clinical outcomes included in this study are all-cause mortality, hospitalization for the last 12 months, infections and cardiovascular diseases (myocardial infarction, coronary artery disease, heart failure and stroke).

2.4. Statistical Analysis

Results were analyzed using SPSS version 20. Continuous variables were expressed as mean and standard deviation. Frequencies and percentages were computed for different categorical variables and chi-square test or Fischer exact test were used for data analysis. A p-value of less than 0.05 was considered as statistically significant.

2.5. Ethical Considerations

The study abides by the Principles of the Declaration of Helsinki (2013) and was conducted along the Guidelines of the International Conference on Harmonization Good Clinical Practice (ICH-GCP), E6 (R2) and other ICH-GCP 6 (as amended); National Ethical Guidelines for Health and Health-Related Research (NEG HHRR), 2017. Ethics approval was sought approved by the Research Ethics Board. This study was not experimental in nature, hence use of humans or animals for this study is not applicable. Consent also is not applicable on this study.

3. Results

3.1. Baseline Characteristics

A total of 279 hemodialysis patients were included in this cross-sectional study, of whom 32 (11.5%) had recurrent ascites and 247 (88.5%) did not have ascites. The baseline characteristics of both groups are presented in Table 1.

The mean age was comparable between patients with and without recurrent ascites (51 ± 21 years vs 47 ± 22 years, respectively; p = 0.356). However, there

Table 1. Baseline characteristics of hemodialysis patients with and without recurrent ascites.

	With Ascites (N = 32)	Without Ascites (N = 247)	p-value
Age (years)	51 ± 21	47 ± 22	0.356
Gender			<0.001
Male	25	98	
Female	7	149	
Dialysis Duration (months)	18 ± 14	21 ± 16	0.201
Dialysis Frequency			0.076
2×/week	16	163	
3×/week	16	84	
ESRD Cause			
Diabetes	14	171	0.004
Hypertension	5	44	0.76
Glomerulonephritis	10	26	0.001
Obstructive Uropathy	2	3	0.893
Others	1	3	0.392
Co-Morbidities			
Arthritis	7	25	0.107
Cancer	2	4	0.089
COPD	8	46	0.393
Cerebrovascular Dse.	7	31	0.148
Diabetes	23	167	0.626
Dyslipidemia	18	101	0.098
Hypertension	24	151	0.127
Heart Disease	15	152	0.113
Liver Disease	9	35	0.042
Hemoglobin (g/dl)	9.5 ± 1.2	10.2 ± 2.3	0.675
HBsAg Positive	1	4	0.392
Average UF/session (L)	4.7 ± 1.1	3.8 ± 2.0	0.158
Serum Sodium (mmol/l)	131 ± 13	136 ± 11	0.313
Serum Calcium (mmol/l)	2.0 ± 0.32	2.1 ± 0.47	0.79

was a significant gender difference between the two groups, with males being disproportionately represented in the ascites group (78.1% vs 39.7%; $p < 0.001$). The median dialysis duration was similar between groups (18 months [IQR 14] vs 21 months [IQR 16]; $p = 0.201$). Most patients in both groups received dialysis three

times per week, with no significant difference in dialysis frequency distribution (p = 0.076).

Regarding the etiology of end-stage renal disease, there was a statistically significant difference between the two groups (p = 0.004). Diabetes mellitus was the leading cause of ESRD in patients without ascites (69.2% vs 43.8% in the ascites group), while glomerulonephritis was more prevalent among patients with recurrent ascites (31.3% vs 10.5%). Hypertensive nephrosclerosis, obstructive uropathy, and other causes showed no significant differences between groups.

Analysis of comorbidities revealed that liver disease was significantly more common in patients with recurrent ascites compared to those without (28.1% vs 14.2%; p = 0.042). Other comorbidities including arthritis, cancer, chronic obstructive pulmonary disease, cerebrovascular disease, diabetes mellitus, dyslipidemia, hypertension, and heart disease showed no statistically significant differences between the two groups.

Laboratory parameters were generally similar between groups. Mean hemoglobin levels were comparable (9.5 ± 1.2 g/dL vs 10.2 ± 2.3 g/dL; p = 0.675), as were serum sodium (131 ± 13 mmol/L vs 136 ± 11 mmol/L; p = 0.313) and serum calcium levels (2.0 ± 0.32 mmol/L vs 2.1 ± 0.47 mmol/L; p = 0.790). Hepatitis B surface antigen positivity was low in both groups with no significant difference (3.1% vs 1.6%; p = 0.392).

Regarding dialysis-related parameters, patients with recurrent ascites had a numerically higher average ultrafiltration volume per session compared to those without ascites (4.7 ± 1.1 L vs 3.8 ± 2.0 L), although this difference did not reach statistical significance (p = 0.158).

3.2. Ascitic Fluid Analysis

Among the 32 hemodialysis patients with recurrent ascites who underwent paracentesis, the characteristics of the initial ascitic fluid analysis are presented in Table 2. The average volume of fluid drained per procedure was 1.79 ± 0.52 liters.

Regarding the etiology of ascites, nephrogenic causes were the most common, accounting for 14 cases (43.8%), followed by congestive heart failure in 10 patients (31.2%). Liver cirrhosis was identified as the underlying cause in 5 patients (15.6%), while less common etiologies included abdominal tuberculosis in 2 patients (6.3%) and peritoneal carcinoma in 1 patient (3.1%).

The biochemical analysis revealed a mean total protein concentration of 2.37 ± 0.50 g/dL in the ascitic fluid. The serum-ascites albumin gradient (SAAG) classification showed that 17 patients (53.1%) had a low gradient (<1.1 g/dL), while 15 patients (46.9%) had a high gradient (≥1.1 g/dL), which is consistent with the mixed etiology observed in this cohort.

Additional biochemical parameters included lactate dehydrogenase levels of 275 ± 168 U/L, amylase levels of 128 ± 34 U/L, and glucose concentrations of 103 ± 47 mg/dL. The cellular analysis showed a mean white blood cell count of 375 ± 312 cells/μL, with segmented neutrophils comprising 60.3% ± 18.8% of the total

Table 2. Characteristics of initial ascitic fluid analysis of hemodialysis patients.

	N = 32 (%) or ±SD
Etiology of Ascites	
Nephrogenic	14 (43.8)
Congestive Heart Failure	10 (31.2)
Liver Cirrhosis	5 (15.6)
Abdominal TB	2 (6.3)
Peritoneal Carcinoma	1 (3.1)
Fluid Drained (L)	1.79 ± 0.52
Total Protein (g/dl)	2.37 ± 0.50
Serum Ascitic Albumin Gradient (g/dl)	
High	15 (46.9)
Low	17 (53.1)
Lactate Dehydrogenase (U/L)	275 ± 168
Amylase (U/L)	128 ± 34
Glucose (mg/dl)	103 ± 47
WBC (cells/μL)	375 ± 312
Segmenters (%)	60.3 ± 18.8
Bacterial Culture Positive	4 (12.5)

cell count. Bacterial culture was positive in 4 patients (12.5%), indicating infectious complications in a subset of cases.

3.3. Clinical Outcomes

The primary outcomes comparing hemodialysis patients with and without recurrent ascites are presented in Table 3. All-cause mortality was markedly higher in patients with recurrent ascites compared to those without ascites (34.4% vs 9.7%, respectively; $p < 0.001$), representing a more than three-fold increase in mortality risk. Hospitalization rates were also significantly elevated in the ascites group, with 14 out of 32 patients (43.8%) requiring hospitalization compared to 59 out of 247 patients (23.9%) in the non-ascites group ($p = 0.016$). This represents nearly a two-fold increase in hospitalization risk among patients with recurrent ascites.

Table 3. Primary outcomes of hemodialysis patients.

Outcomes	Recurrent Ascites	No Ascites	p-value
All-cause Mortality	11	24	<0.0001
Hospitalization	14	59	0.016
Infection	20	117	0.107
Cardiovascular Dse.	15	108	0.736

Regarding infection-related complications, patients with recurrent ascites had a numerically higher rate of infections (62.5% vs 47.4%), although this difference did not reach statistical significance ($p = 0.107$). Similarly, cardiovascular disease events showed comparable rates between the two groups (46.9% vs 43.7%; $p = 0.736$).

4. Discussion

Our study identified several important findings regarding recurrent ascites in hemodialysis patients that contribute to the limited literature on this understudied complication. The prevalence of recurrent ascites in our hemodialysis population was 11.5% (32/279 patients), which falls within the reported range of 0.7% - 20% described in previous studies, though most contemporary reports suggest declining incidence due to improvements in dialysis technology and patient care [9]. This prevalence is comparable to the 11.3% reported by Sai Spandana et al. in a recent study of chronic kidney disease patients [8].

The most striking finding was the significant male predominance among patients with recurrent ascites (78.1% vs 39.7% in controls, $p < 0.001$). This observation aligns with existing literature on nephrogenic ascites, where a male-to-female ratio of 2:1 has been consistently reported [10] [11]. This gender disparity may reflect biological differences in fluid handling, cardiovascular risk profiles, or underlying disease patterns. Studies in hemodialysis populations have shown that men generally have different vascular access outcomes, higher interdialytic weight gains, and distinct cardiovascular risk profiles compared to women [12].

Our ascitic fluid analysis revealed nephrogenic causes as the predominant etiology (43.8%), followed by congestive heart failure (31.2%) and liver cirrhosis (15.6%). This distribution differs from some earlier reports where cardiac causes were more predominant, possibly reflecting improvements in cardiovascular management in contemporary dialysis populations [13]. The high proportion of nephrogenic ascites in our cohort is significant, as this entity carries a particularly grave prognosis with mortality rates of 45% within 15 months of diagnosis [10].

Interestingly, 53.1% of patients had a low serum-ascites albumin gradient (SAAG < 1.1 g/dL), which traditionally indicates non-portal hypertensive causes and increased peritoneal membrane permeability. This finding is consistent with the pathophysiology of nephrogenic ascites, where altered peritoneal membrane permeability, lymphatic drainage disturbances, and hypoalbuminemia contribute to ascites formation [14] [15]. The mean total protein content of 2.37 ± 0.50 g/dL in our study aligns with the typical characteristics of nephrogenic ascites, which usually presents with high protein content (3 - 6 g/dL) and low SAAG [10].

The clinical outcomes in our study underscore the serious prognostic implications of recurrent ascites in hemodialysis patients. The all-cause mortality rate of 34.4% in the ascites group compared to 9.7% in controls ($p < 0.001$) represents a more than three-fold increase in mortality risk. This finding is consistent with previous reports describing the grave prognosis associated with ascites in end-

stage renal disease patients. Historical studies have reported survival times ranging from 7 - 10.7 months after ascites onset, with mortality rates of 17.4% - 44% [9] [15].

The significantly higher hospitalization rate (43.8% vs 23.9%, p = 0.016) among patients with ascites reflects the increased healthcare burden and clinical complexity associated with this condition. The trend toward higher infection rates (62.5% vs 47.4%), although not statistically significant, may reflect the immunocompromised state of these patients and potential complications from repeated paracentesis procedures.

The bacterial culture positivity rate of 12.5% in our ascitic fluid samples is lower than some reports of spontaneous bacterial peritonitis in cirrhotic patients but reflects the different patient population and underlying pathophysiology. The mean white blood cell count of 375 ± 312 cells/μL with 60.3% segmented neutrophils indicates a mild inflammatory response, which is typical for nephrogenic ascites.

Our study highlights the limited therapeutic options available for this patient population. Unlike ascites from other causes, nephrogenic ascites responds poorly to conventional treatments such as diuretics or dietary sodium restriction. The literature suggests that intensification of dialysis with increased ultrafiltration, salt and fluid restriction, and albumin supplementation may provide some benefit, though outcomes remain generally poor [16]. Renal transplantation remains the only definitive treatment for nephrogenic ascites, with complete resolution typically occurring within 2 - 6 weeks post-transplantation [17]. However, most patients with advanced ascites are often poor transplant candidates due to malnutrition, cardiovascular comorbidities, and overall poor functional status.

Our study has several important limitations that must be acknowledged. First, the cross-sectional design limits our ability to establish temporal relationships and causality between identified risk factors and ascites development. A longitudinal study design would provide better insights into the natural history and progression of ascites in this population. Second, the single-center nature of our study may limit the generalizability of our findings to other hemodialysis populations with different demographic characteristics, underlying disease patterns, or practice variations. Multi-center studies would strengthen the external validity of these observations. Third, we did not systematically assess important clinical parameters that could influence ascites development, including dialysis adequacy markers (Kt/V, urea reduction ratio), nutritional status indicators (serum albumin, prealbumin), or detailed cardiovascular assessments. These factors could be important confounders or mediators in the relationship between patient characteristics and ascites development. Fourth, our follow-up period for clinical outcomes was not specified, which limits the interpretation of mortality and morbidity data. Standardized follow-up periods would allow for more precise prognostic assessments and comparison with existing literature.

Finally, the relatively small sample size of patients with ascites (n = 32) may

limit the statistical power to detect smaller but clinically meaningful differences in certain variables. A larger multicenter study would provide more robust statistical power for subgroup analyses.

Recurrent ascites in hemodialysis patients represents a serious complication with a poor prognosis, characterized by male predominance, high mortality rates, and increased healthcare utilization. The predominance of nephrogenic causes and the characteristic ascitic fluid profile with low SAAG and moderate protein content provide insights into the underlying pathophysiology. The significantly worse clinical outcomes, including three-fold higher mortality and increased hospitalization rates, emphasize the clinical importance of this condition and the urgent need for improved management strategies.

Conflicts of Interest

The author declares no conflicts of interest regarding the publication of this paper.

References

[1] Al-Zakhari, R., Alataby, H., Freg, G., Moussa, J., Mossayebi, E. and Ebrahimi, F. (2020) A Real Neglected Problem with a Grave Prognosis: Nephrogenic Ascites. *Journal of Medical Cases*, **11**, 26-29. https://doi.org/10.14740/jmc3413

[2] Nayak-Rao, S. (2015) Nephrogenic Ascites—Still an Intractable Problem? *Saudi Journal of Kidney Diseases and Transplantation*, **26**, 773-777. https://doi.org/10.4103/1319-2442.160214

[3] Rajora, N., De Gregorio, L. and Saxena, R. (2021) Peritoneal Dialysis Use in Patients with Ascites: A Review. *American Journal of Kidney Diseases*, **78**, 728-735. https://doi.org/10.1053/j.ajkd.2021.04.010

[4] Biecker, E. (2011) Diagnosis and Therapy of Ascites in Liver Cirrhosis. *World Journal of Gastroenterology*, **17**, 1237-1248. https://doi.org/10.3748/wjg.v17.i10.1237

[5] Abdalla, M.S., Saad, E., Abdalla, M., Musa, T.I. and Mohamed, K. (2022) Nephrogenic Ascites: An Unusual Culprit of Refractory Ascites in a Hemodialysis Patient—A Case Report and Review of Literature. *Cureus*, **14**, e30876. https://doi.org/10.7759/cureus.30876

[6] Pauly, R.P., Sood, M.M. and Chan, C.T. (2008) Management of Refractory Ascites Using Nocturnal Home Hemodialysis. *Seminars in Dialysis*, **21**, 367-370. https://doi.org/10.1111/j.1525-139x.2008.00439.x

[7] Ruiz, S.R., Vilches, E.G., Garcia-Frias, T.P., Velasquez, M., Martos, L.B. and Salcedo, T.J. (2011) The Role of Peritoneal Dialysis in the Treatment of Ascites. *Nefrologia*, **31**, 760-764. http://doi.org/10.3265/Nefrologia.pre2011.Jun.10901

[8] Spandana, G.S., Viswanathan, S.S. and Selvaraj, J. (2023) Etiology and Outcomes in Patients with Chronic Kidney Disease and Ascites. *Cureus*, **16**, e64113.

[9] Gollapudi, S.S., Stalin, V., Deepak, B.S. and Jayachandaran, S. (2024). Etiology and Outcomes in Patients with Chronic Kidney Disease and Ascites. *Cureus*, **16**, e64113.

[10] Nader, M.A., Aguilar, R., Sharma, P., Krishnamoorthy, P., Dragoi, S., Gordon-Cappitelli, J. and Shen, W. (2017) In-Hospital Mortality in Cirrhotic Patients with End Stage Renal Disease Treated with Hemodialysis versus Peritoneal Dialysis: A Nationwide Study. *Peritoneal Dialysis International*, **37**, 464-471. https://doi.org/10.3747/pdi.2016.00131

[11] Han, S.B., Reynolds, T.B. and Fong, T. (1998) Nephrogenic Ascites: Analysis of 16 Cases and Review of the Literature. *Medicine*, **77**, 233-245. https://doi.org/10.1097/00005792-199807000-00002

[12] Port, F.K., Pisoni, R.L., Bommer, J.D., Locatelli, F., Jadoul, M., Eknoyan, G., *et al.* (2006) Improving Outcomes for Dialysis Patients in the International Dialysis Outcomes and Practice Patterns Study. *Clinical Journal of the American Society of Nephrology*, **1**, 246-255. https://doi.org/10.2215/cjn.01050905

[13] Athish, K.K. and Nayak-Rao, S. (2025) Nephrogenic Ascites: A Case Series with Review of Literature. *Hemodialysis International*, **29**, 247-252. https://doi.org/10.1111/hdi.13216

[14] Hammond, T.C. and Takiyyuddin, M.A. (1994) Nephrogenic Ascites: A Poorly Understood Syndrome. *Journal of the American Society of Nephrology*, **5**, 1173-1177. https://doi.org/10.1681/asn.v551173

[15] Cintin, C. and Joffe, P. (1994) Nephrogenic Ascites. Case Report and Review of the Literature. *Scandinavian Journal of Urology and Nephrology*, **28**, 311-314. https://doi.org/10.3109/00365599409181287

[16] Franz, M. and Horl, W. (1997) The Patient with End-Stage Renal Failure and Ascites. *Nephrology Dialysis Transplantation*, **12**, 1070-1078. https://doi.org/10.1093/ndt/12.5.1070

[17] Nasr, E.M. and Joubran, N.I. (2001) Is Nephrogenic Ascites Related to Secondary Hyperparathyroidism? *American Journal of Kidney Diseases*, **37**, e16.1-e16.4. https://doi.org/10.1053/ajkd.2001.21360