Available online at http://inakidneyhypertension.co.id Official Journal of the Indonesian Society of Nephrology
Indonesian Journal of Kidney & Hypertension InaKidney

Case Report

Nephrogenic Ascites in End-Stage Renal Disease Patients Undergoing Hemodialysis: Case Series

Deddy Primadona Mulia [1,2,3], Rico Irawan[1], Herleni Kartika[3], Syahpri Putra Wangsa[3], Kgs M Yusuf Arief Akbar[3], Ian Effendi[3] , Novadian Suhaimi[3], Suprapti[3]. Zulkhair Ali[3]

[1] **Tulus Ayu Hospital of Belitang**
[2] **Muhammadiyah Hospital of Palembang**
[3] **Moh. Hoesin Hospital of Palembang**

ARTICLE INFO

Article history:
Received: March 26, 2024
Accepted: April 19, 2024
Published Online: April 24, 2024

Corresponding Author:
Zulkhair Ali, Moh. Hoesin Hospital Palembang, zulkhair33@fk.unsri.ac.id

ABSTRACT

Ascites occurring in individuals with chronic kidney disease (CKD) undergoing hemodialysis (HD) are referred to as nephrogenic ascites (NA). The pathogenesis of NA remains uncertain but involves many interrelated factors. These factors include inadequate dialysis, low ultrafiltration, poor nutrition, increased peritoneal membrane permeability, and impaired peritoneal lymphatic reabsorption. Frequent accompanying complaints are leg edema, nausea, decreased appetite, weakness, and complaints due to hypotension during HD. NA generally carries a poor long-term prognosis, likewise, with complex therapeutic options, both medical and surgical. Management of NA includes education regarding fluid restrictions, a high protein diet, intensive HD, as well as therapeutic modalities with continuous ambulatory peritoneal dialysis (CAPD) and kidney transplantation. CAPD has been proven to improve quality of life and recovery from ascites. The NA in this case report was all related to the patient's low level of compliance against fluid intake restrictions, particularly while experiencing oliguria or anuria. In this case, intensive education regarding limiting fluid intake and high-protein nutrition seems essential for better patient outcomes. On the other hand, the presence of infectious complications gives poor outcomes. Two patients presented with umbilical hernia, which could be an obstacle for CAPD.

Keywords: nephrogenic ascites, dialysis adequacy, low protein intake.

Introduction

Nephrogenic ascites (NA) are ascites that occur in patients with end-stage kidney disease (ESRD) undergoing hemodialysis (HD). The cause of NA is multifactorial and can be a combination of low patient compliance, inadequate dialysis, ultrafiltration failure due to hypotension, poor nutrition, and increased peritoneal membrane permeability. In general, NA carries a worse long-term prognosis for HD patients.[1] Management of NA is very complicated, including medical and surgical therapy. Education about limiting fluid intake and a high-protein diet must be periodically reviewed. This report presents 3 case series of HD patients with NA, with varying underlying causes of ESRD and patient outcomes.

Case Illustration

Case 1

A 51-year-old man complained of abdomen enlargement for the last 3 months, accompanied by a feeling of fullness, nausea, decreased appetite, and sometimes coughing. The patient has undergone HD 2 times a week for 2 years, with hypertension and diabetes mellitus (DM) as underlying ESRD. He often experiences hypotension, requiring crystalloid fluid therapy at

Cite this as:
Deddy PM, Rico I, Herleni K, et al. Nephrogenic Ascites in End-Stage Renal Disease Patients Undergoing Hemodialysis: Case Series.InaKidney. 2024;1(1);37-40. DOI: 10.32867/inakidney.v1i1.121

the end of HD. The patient's hemoglobin (Hb) level also frequently decreases, requiring a transfusion. Compliance with fluid restrictions is also low; on the other hand, there is oliguria.

The patient's dry weight was 55 to 60 kg, with an average gain of 5 to 6 kg. Using Cimino as HD access, the ultrafiltration rate is 500-750 cc/hour; blood flow rate (QB) starts from 180-210 ml/min, and dialysate flow rate (QD) is 500 ml/min. Blood pressure (BP) was 180/100 mmHg, heart rate 100x/min, respiratory rate 24x/min, temperature 36.5°C, and SpO2 100% in room air. There was an enlarged left heart, as well as ascitic fluid. No abnormalities were found in other organs.

Laboratory: Hb was 7.6 g/dl, serum urea and creatinine levels were 130 mg/dl and 6.5 mg/dl respectively, albumin levels were 2.9 mg/dl, electrolyte levels were normal, and markers for viral hepatitis were negative.

Patients hospitalized with loop diuretic IV, transfusions of blood, and albumin. After the transfusion, Hb and albumin levels approached normal. Another therapy is ascites puncture, with a total of 3 liters of fluid. Re-education was also conducted regarding continuous ambulatory peritoneal dialysis (CAPD), protein diet, and fluid restrictions. There was an improvement after 4-5 months, with a dry weight of around 60 to 62 kg, minimal leg edema, and ascites. However, Hb levels often fall, necessitating frequent transfusions.

Case 2

A 53-year-old woman complained of abdomen enlargement since the previous month, accompanied by worsening shortness of breath, nausea, and frequent coughing from the last week. The patient has been undergoing HD for 2 years, twice a week, with an underlying ESRD hypertension. Compliance with diet and fluid restriction is low, and there is anuria condition. On the other hand, on average, the patient often receives blood transfusions once a month. Dry weight is 58 kg, with an average weight gain of 7-8 kg. Using double lumen catheter for HD access for 4-5 hours, ultrafiltration 300-500 cc/hour QB starting from 170-200 ml/min, and QD 500 ml/min.

The patient looked pale, had poor nutritional status, and had hypotony in both arms. BP was 200/100 mmHg, heart rate 110x/min, respiratory rate 30x/min, temperature 36.5°C, and SpO2 98% with O2 4 lpm nasal cannula. There was swelling in both legs and signs of left heart enlargement. The abdomen showed prominent ascites, while other organs were within normal limits.

Laboratory: Hb was 6.9 g/dl, leukocytes 13,500/ul, serum urea and creatinine levels 180 mg/dl and 10.5 mg/dl respectively, albumin level was 2.3 mg/dl, electrolyte levels were normal, and viral hepatitis markers were negative. Chest X-ray results showed pneumonia.

The patient was hospitalized and given ceftriaxone 1 gram BID, furosemide IV, and blood and albumin transfusions. Hb levels have increased to 9.2 mg/dl, while albumin levels remained low. Additionally, ascites paracentesis of 1 liter within 24 hours was performed. However, despite treatment efforts, the patient's condition deteriorated, leading to death after 5 days, primarily due to sepsis.

Case 3

A 31-year-old man complained of abdomen enlargement for the last 2 weeks. He also experienced weakness, shortness of breath, dry cough, and increasing weight loss. The patient underwent HD for 2 years, twice a week, with underlying ESRD was glomerulonephritis. The patient's compliance with diet and fluid restriction is low, with anuria condition. The dry weight was 60 kg, with an average weight gain of up to 8 kg, making it challenging to achieve dry weight. Using double lumen catheter for HD access, lasting 4-5 hours, ultrafiltration 500-750 cc/hour; QB starting from 180-200 ml/min, and QD 500 ml/min. The patient often experienced hypotension during HD and complained of muscle cramps, dizziness, and nausea. Giving 0.9% NaCl fluid at the end of HD made achieving the UF goal difficult.

On physical examination, the patient appeared weak, with poor nutritional status and hypotony in both arms. BP was 150/80 mmHg, heart rate 100x/m regular, respiratory rate 24x/minute, temperature 36.2°C, and SpO2 98%

room air. The conjunctiva was pale, swollen in both legs and left heart enlargement. The abdomen showed massive ascites and an umbilical hernia, while other organs were normal.

Laboratory: Hb was 10.5 g/dl, serum urea and creatinine levels were 131 mg/dl and 10.6 mg/dl respectively, albumin level was 3.2 mg/dl, electrolytes were normal, and viral hepatitis markers were negative. Chest X-ray radiology suggested an active specific process.

The patient was hospitalized and received standard treatment, including furosemide IV and symptomatic therapy. Paracentesis, 1 liter of ascites per 24 hours, was also performed. After 7 days of treatment, the condition improved, and the follow-up was outpatient, with re-education regarding compliance with diet and fluid intake.

Discussion

In general, the complaints and clinical conditions of the patients above are relatively the same, namely an enlarged abdomen and slight leg edema. There was no history of other organ abnormalities or malignancy, but there was 1 case of pneumonia, and umbilical hernias were found in 2 patients, which were associated with massive ascites. All patients had heart enlargement related to HD and its comorbidities.

Ascites associated with HD are referred to as nephrogenic ascites (NA).[4] The cause of NA is still unclear, but it is thought to involve a combination of interrelated factors, including poor nutritional status, patient non-compliance, and late initiation of renal replacement therapy (RRT). The pathophysiology underlying the occurrence of NA includes hepatic vein hydrostatic pressure, changes in the permeability of the peritoneal membrane caused by the inflammatory effects of uremic toxins, obstruction of lymphatic channels caused by inflammatory infiltrates resulting in changes in peritoneal fluid absorption, and accumulation of ascitic fluid. Other predisposing factors may include hypoalbuminemia, hyperparathyroidism, effective in controlling ascites formation and therapy against ascites (Table 1).[9]

heart failure, constrictive pericarditis, pancreatitis, and cirrhosis with portal hypertension.[5]

Diaz-Mancebo stated that the theory underlying the pathophysiology of NA is as follows[6]:

- Increased hepatic vein hydrostatic pressure
- Excess fluid intake
- Increased peritoneal membrane permeability, caused by uremic toxins, long-term use of dialysate solutions, immune complexes, activity of the renin-angiotensin-aldosterone (RAA) system, and hemosiderosis
- Disruption of peritoneal lymphatic drainage

The four pathogenesis mechanisms above have different roles in causing NA, but they are all related.[5]

The diagnosis of NA is carried out by exclusion, which requires a complete examination to exclude liver and heart function, infection, and malignant causes. The history and physical examination generally show ascites with minimal leg edema. Patients usually also have a history of anorexia and cachexia and experience hypotension during HD. In addition to kidney function, the recommended laboratory evaluation includes markers for viral hepatitis B and C and albumin or protein levels.

The prognosis of HD patients with accompanying ascites is relatively poor, with low quality of life (QOL). Life expectancy in HD patients with nephrogenic ascites ranges from 7 to 10.7 months on average, with a 44% chance of dying within 15 months of diagnosis. The clinical course of patients with recurrent NA is characterized by cachexia and, ultimately, progression to death.[5] Management of NA is complex and includes various medical and surgical modalities.[7] In the early stages, salt intake and fluid restriction are limited, intensive HD with ultrafiltration is administered, and intravenous albumin with a high protein diet is administered to control ascites and prevent hypotension. CAPD, peritoneovenous shunt (PVS), and kidney transplantation appear to be

Table 1. Treatment options for nephrogenic ascites, following advantages and disadvantages

Intervention	Advantages	Disadvantages
Restriction of drinking and salt accompanied by intensive hemodialysis and ultrafiltration	Ascites is reduced	Hypotension
Hyperalimentation therapy	Increased nutrition	Untested
Repeated paracentesis	Symptoms decrease	Losing large amounts of protein
CAPD	Reduced ascites and nutrition increase	Early protein loss
Peritoneo-venous shunt	Ascites reduced, dialysis	Shunt
Kidney transplantation	The best therapy for Ascites	Can recur at times kidney damage

Conclusion

Case series of NA have been reported, all of which were associated with low levels of patient compliance with fluid intake restrictions. More intensive education regarding limiting fluid intake and high-protein nutrition seems essential for better patient outcomes. NA is a condition that accompanies ESRD undergoing HD with a poor prognosis. Many factors are caused and are interrelated, including poor nutritional status, low compliance, and late initiation of RRT. The therapy of choice consists of intensive HD, a high-protein diet, kidney transplantation, PVS, and paracentesis. CAPD is also an option because it has been proven to improve QOL and recovery from ascites.

Declarations

Competing interests

The authors declare no conflict of interest.

Funding source

This study did not receive any external funding.

Acknowledgements

None.

References

1. Nayak-Rao S. Nephrogenic Ascites - Still an Intractable Problem? Saudi Journal of Kidney Diseases and Transplantation. 2015;26(5):413–419.
2. Tasneem AA, Khan AA, Abbas Z, Luck NH, Hassan SM. Ascites in Patients on Maintenance Hemodialysis: Causes, Characteristics and Predicting Factors. Journal of the College of Physicians and Surgeons Pakistan. 2016;26(5):413–419.
3. Covic A, Bammens B, Lobbedez T, Segall L, Heimbürger O, Van Biesen W, Fouque D, Vanholder R. Educating end-stage renal disease patients on dialysis modality selection. NDT Plus. 2010;3(3):225–233.
4. Al-Zakharia R, Alataby H, Freg G, Moussa J, Mossayebi E, Ebrahimi F. A Real Neglected Problem With a Grave Prognosis: Nephrogenic Ascites. Journal Of Medical Cases. 2020;11(1):26–29.
5. Rajora N, De Gregorio L, Saxena R. Peritoneal Dialysis Use in Patients With Ascites: A Review. American Journal of Kidney Diseases. 2021;78:728-735.
6. Díaz-Mancebo R, Sánchez-Villanueva R, González-García E, Ossorio-González M, Selgas-Gutiérrez R. Nephrogenic ascites: a thing of the past? Nefrologia. 2012;32(3):406–408. https://doi.org/10.3265/Nefrologia.pre2012.Jan.11325
7. Singh S, Mitra S, Berman LB. Ascites in Patients on Maintenance Hemodialysis. Nephron. 1974;12(2):114–120.
8. Khalil MAM, Jukmin MKH, Chong VH, Tan J. Nephrogenic Ascites in a Hemodialysis Patient. Saudi Journal of Kidney Diseases and Transplantation. 2020;32(3):687–692.
9. Korzets A, Danby P, Feehally J, Walls J. CAPD: Successful Use in the Treatment of Nephrology Ascites. Nephrology DialysisTransplantation. 1989;4(10):918–919. https://doi.org/10.1093/ndt/4.10.918