Central Asian Journal of Nephrology

Case Report

A Case Report of a Diabetic Nephropathy Patient with Cirrhotic Ascites and HIV Recommended for Peritoneal Dialysis

Zhanna Kozybayeva[1], Nazia Islam[1], Baknur Absattar[1], Raushan Bigaliyeva[1], Aigerim Khabayeva[2], Meruyert Madikenova[2], Aruzhan Meirmankulova[2], Lazzat Toleuova[2], Venera Altynova[2]

[1]*Department of Medicine, School of Medicine, Nazarbayev University, Astana, Kazakhstan*
[2]*Department of Nephrology, University Medical Center, Astana, Kazakhstan*

Abstract:

Received: July 30, 2025
Accepted: August 16, 2025

Corresponding author's email:
zh.kozybayeva@icloud.com

A 46-year old male was admitted to the University Medical Center (UMC) hospital with the following symptoms of anuria, abdominal fullness, hypotension, exertional dyspnea, and peripheral edema. The purpose of his visit was the insertion of a peritoneal dialysis catheter. He had chronic kidney disease stage 5 as a consequence of diabetic nephropathy, liver cirrhosis due to hepatitis C infection, and HIV. His disease course was further complicated by the presence of a urinary tract infection. As a result of his multiple comorbidities, he underwent a complex treatment regimen which included renal replacement therapy with ultrafiltration, blood transfusions for his anemia, platelet transfusions for his thrombocytopenia, albumin infusion for his hypoalbuminemia, and antibiotic treatment for his concurrent infection. Additionally, he received diuretic treatment for his hypervolemia and anti-hypertensives to control his blood pressure. After peritoneal dialysis (PD) insertion, the patient successfully underwent PD and was discharged home.

Keywords: Diabetic Nephropathy; Cirrhotic Ascites; HIV Infection; Case Report; Renal Complications; Comorbidities; Chronic Kidney Disease; Liver Cirrhosis; Infectious Disease; Nephrology

Introduction

One of the most common and serious complications of type I Diabetes Mellitus (T1DM) is diabetic nephropathy. It mainly results from endothelial dysfunction and proinflammatory pathway activation leading to renal microvascular lesions (1, 2). Moreover, this complication develops in approximately 35% of type I Diabetes patients and often follows a progressive course, further advancing to albuminuria, declining glomerular filtration rate (GFR), and advancing to end-stage renal disease (ESRD) (3). While diabetic nephropathy is an established cause of chronic kidney disease (CKD), other comorbid conditions, particularly chronic viral infections such as HIV and hepatitis C can significantly impair kidney function.

In patients with an HIV infection, chronic inflammation and dysregulation in immune mediators can significantly contribute to kidney damage, often manifesting as HIV-associated nephropathy (HIVAN) (4). Additionally, antiretroviral therapy (ART), while essential, can have a nephrotoxic effect. Similarly, patients with hepatitis C infection are also prone to the exacerbation of CKD as it induces kidney injury by the formation of immune complexes and cryoglobulins (5). Additionally, chronic hepatitis C infection, being the leading cause of liver cirrhosis, can precipitate hepatorenal syndrome (HRS) resulting in vasodilation and diminished renal perfusion.

The following case study reports a patient with coexisting type I DM, HIV and hepatitis C infections. It

is highly likely that a cholinergic effect of these comorbidities accelerated the progression of CKD, further complicating his clinical management.

Case Report

A 46-year old male was admitted to the UMC hospital because of anuria, abdominal fullness, hypotension, exertional dyspnea, and peripheral edema. He was diagnosed with type 1 diabetes mellitus in 2007 and Essential hypertension stage 3 in 2019. Furthermore, in 2020, he was found to have liver cirrhosis as a result of chronic hepatitis C infection and tested positive for an HIV infection. Patient had an intravenous drug use history, which included shared use of needles and injection equipment. Thus, he associated his hepatitis C infection with the HIV acquisition risk. He received antiviral therapy consisting of Kivexa (Abacavir 600 mg + Lamivudine 300 mg) and sometimes Truvada (Tenofovir disoproxil fumarate 245 mg + Emtricitabine 200 mg) based on the availability of the medication in AIDS control center. In 2023 his blood results revealed azotemia. As a result, he was consulted by a nephrologist, who recommended an antihypertensive therapy. Additionally, all antihyperglycemic medications were discontinued. In April 2024 he was admitted to the hospital for the treatment of liver cirrhosis, where his GFR was detected to be 27 ml/min. In July 2024 he was admitted with the following complications: secondary hyperparathyroidism, hyperphosphatemia, and diabetic nephropathy (stage IV by Mogensen classification). Furthermore, he had essential hypertension (stage 3), liver cirrhosis (Child-Turcotte-Pugh class B), portal hypertension, ascites, hypersplenism, and hepatic encephalopathy (West Haven stage 1). Moreover, his GFR was reduced to 12.2 ml/min. In August 2024 he was admitted to the ICU for acute decompensation, presenting with reduced urination, worsening of edema, nausea, and vomiting. Lab analysis results showed increased creatinine (587 umol/L), azotemia (38 mmol/L), hyperkalemia (7.2), and metabolic acidosis (pH 7.181). Based on these clinical findings and anuria, he received 10 sessions of hemodialysis during his hospitalization. A multidisciplinary team meeting was held and it was decided to place the patient on peritoneal dialysis (PD). In September 2024, he was admitted on a planned basis to UMC for the insertion of a PD catheter.

Upon admission, physical examination showed ascites, peripheral edema, and jaundice. The patient was in respiratory distress and had hypotension (80/40 mm Hg). In order to rule out infection, septicemia, and shock due to sepsis, procalcitonin and C-reactive protein were measured. Moreover, blood, throat, nasopharyngeal, and urine cultures were taken. Laboratory studies demonstrated hemoglobin of 84 g/L, MCH of 28.10 pg, RBC of 2.99x1012/L, and platelets of 94.0x1012/L. Biochemistry analysis depicted a creatinine of 378.70 μmol/L, urea of 14.40 mmol/L, CRP of 23.80 mg/L, and albumin of 30 g/L. His GFR was 16 ml/min. His electrolytes were as follows: sodium - 134 mmol/L, potassium - 4.40 mmol/L, total calcium - 2.10 mmol/L, ionized calcium - 1.01 mmol/L, magnesium - 0.89 mmol/L and phosphate - 2.01 mmol/L. Procalcitonin level was within the normal range. His ABG indicated metabolic acidosis with a pH of 7.29 and BE (ecf) of 1.6 mmol/L. The urinalysis revealed leukocytes of 702 hpf, erythrocytes of 688.8 hpf, and protein of 2.10 g/L. Urine culture demonstrated the presence of Enterococcus faecalis of 50,000 CFU/ml. Her ultrasound confirmed ascites, liver cirrhosis, and portal hypertension, along with splenomegaly. Additionally, esophagogastroduodenoscopy was performed to rule out gastrointestinal bleeding and grade 3 esophageal varices were observed. Also, it is remarkable that patient's cardiac ejection fraction dropped to 57% in 2024%.

In order to address his hypervolemia and electrolyte derangements, continuous renal replacement therapy was performed with ultrafiltration at the rate of 500 ml/hour and anticoagulation using heparin (1000 IU/hour). Additionally, 20% albumin infusion was administered to manage his hypoalbuminemia (albumin level 26 g/L) which was contributing to his hypervolemia. Due to his worsening anemia (hemoglobin level 56 g/L), he received a transfusion with two doses of packed RBCs, along with epoetin beta. Furthermore, to reduce his risk of bleeding from thrombocytopenia (56x109/L), he underwent platelet transfusion twice. Based on his urinalysis and urine culture findings, which indicated Enterococcus faecalis, meropenem (1000 mg/day) was initiated to treat his urinary tract infection. Insertion of the PD catheter was performed via laparotomy under general anesthesia. Upon entering the abdominal cavity, a large amount of clear ascitic fluid was observed and 5 liters were aspirated. A Tenckhoff catheter for peritoneal dialysis was implanted into the pelvic cavity. It was then tunneled subcutaneously through the left mesogastrium and closed with a Betadine cap, after extension with a titanium adaptor. The next day after the procedure the patient continued to have oliguria (less than 100 ml) and he was placed on continuous renal replacement therapy.

The patient then began to undergo PD once every 24 hours with the following parameters - Physioneal® 40 (2.27% glucose concentration), Physioneal® 40 (1.36% glucose concentration), 4-hour dwell cycle, and a fill volume of 1500 mL per exchange. Compared to the results of his lab analyses on admission, his creatinine dropped to 357.66 μmol/L. However, his potassium level rose to 6.4 mmol/L and his sodium level dropped to 132 mmol/L. The patient continued to receive albumin infusion and upon discharge, his albumin level rose to 27.50 g/L (from 26.0 g/L) and his total protein was 50.0 g/L.

During his stay, he additionally received calcium gluconate to correct his hyperkalemia. Insulin was used to manage his hyperglycemia associated with diabetes. Also, furosemide was given for his fluid overload during the first 24h of hospitalization, which was changed to isolated ultrafiltration on the second day. In the days following the PD insertion procedure, carvedilol, amlodipine, and nifedipine were also administered to carefully control his blood pressure.

Discussion

A 46-year old male with CKD and multiple comorbidities such as HIV and hepatitis C infections, later developing urinary tract infection, was admitted for the PD catheter placement and further management.

As mentioned above, diabetic nephropathy is one of the most common and devastating complications of T1DM. Usually, it has a progressive course, which can be subtly divided into several stages according to Mogensen (6) with each having relatively specific patterns. During Stage 1, while renal hyperfunction and hypertrophy occur, these changes are, at least, partially reversible with an insulin treatment. In Stage 2, individuals usually do not exhibit clinical manifestations, however, kidney function tests can already demonstrate signs of disease. On the other hand, several patients can remain on this stage throughout their lives. Stage 3 is informally considered to be an initial phase of diabetic nephropathy, when albumin excretion level is abnormally elevated. This stage, however, can be slowly advancing over many years, during which, several patients can already develop hypertension. Subsequently, stage 4 is progressing to an overt nephropathy, characterized by a persistent proteinuria and a declining GFR. Finally, stage 5 is the ESRD with the patient having uremia and requiring renal replacement therapy.

In individuals with HIV, HIVAN can be a serious complication and can be observed in up to 17% of patients. Significant risk factors that increase the risk of renal damage in HIV patients are hepatitis C infection and diabetes, which both are present in the following patient. Moreover, exposure to ART can itself be a triggering factor for the decline in renal function (7). While there are a number of pathways responsible for the damage, intrarenal HIV gene expression is one of the mechanisms causing direct injury of the podocytes and glomerular scarring. Additionally, dysregulation in immune mediation and chronic inflammation also contribute to endothelial dysfunction and, as a result, renal fibrosis, further exacerbating the condition (8). Similarly, hepatitis C can significantly impair renal function by the precipitation of cryoglobulins which, in turn, cause mesangial inflammation and endothelial injury. Moreover, immune complexes can gather and deposit in the basement membrane, leading to glomerular damage and inflammation (9).

The patient's diabetic nephropathy compounded by his HIV and hepatitis C infection may have led to quicker progression of his disease (4,5) and led to the decompensated state observed in this patient. With a GFR of between 12.2-16 ml/min, metabolic acidosis (pH 7.181) and severe electrolyte derangements (hyperkalemia 7.2), the patient was in kidney failure and required immediate renal replacement therapy.

End stage renal disease in patients with multiple comorbidities as observed in this case present various challenges and must be individually tailored. For instance, treatment of hypervolemia in ESRD patients with cirrhotic ascites require careful monitoring as such patients often have low effective arterial volume despite being hypervolemic (10). Combined with the presence of coagulation abnormalities and malnutrition, cirrhotic patients do not tolerate the sudden removal of fluids from intravascular spaces well during intermittent hemodialysis (10). As such, peritoneal dialysis may offer a more hemodynamically stable alternative for our patient. Several reports exist that recommend PD for patients with liver diseases who have ascites (10,11). Studies by Chou et. al and Nadir et.al demonstrate that patients with kidney failure and cirrhosis on PD may have higher survival rates compared to groups on HD (12,13). A major advantage of PD is that anticoagulation is not required. Given that the coagulopathy observed in cirrhotic patients is often worsened by concurrent uremia, the potential reduction in the risk of bleeding is of significant importance. PD also removes the need for paracentesis as it allows constant drainage of ascites (7).

Nevertheless, certain complications need to be considered when performing PD such as the risk of infection, protein losses, pericatheter leaks and abdominal hernias. Despite these concerns, a study by De Vecchi et.al concluded that the risk of dialysis complications, including bacterial peritonitis, was not increased in cirrhotic patients receiving PD (14). However, given the presence of HIV, cirrhosis, diabetes, and coexisting infection, the risk of peritonitis, wound infection and sepsis were major concerns in our patient. Thus, using the antimicrobial resistance profile and the local resistance patterns as a guide, our patient received meropenem to treat his infection and prevent further progression. Given that PD is usually initiated two weeks post-insertion of a catheter to allow healing (15), the patient received continuous venovenous hemodiafiltration (CVVHDF) during his stay for the correction of his fluid overload and the prevention of electrolyte imbalances. CVVHDF presents an advantage over intermittent hemodialysis in our patient as it is considered to provide better hemodynamic stability (16). However, due to limited resources, and an inability to administer further hemodialysis due to the risk of HIV and hepatitis C transmission, the PD had to be initiated prematurely. To prevent the risk of leakage, a smaller fill volume of 1.5 L was used and the number of exchanges was limited to once per day. Consequently, hyperkalemia and hyponatremia were observed. However, these imbalances were expected to become normalized after the adjustment in the prescription of peritoneal dialysis once larger fill volumes could be used.

It is also noteworthy to mention that due to the presence of several underlying health conditions, a multidisciplinary approach had to be employed in the management of this patient. Firstly, the presence of cirrhosis in our patient complicated the management of his diabetes as loss of liver function can reduce insulin clearance. Therefore, the dosage of insulin had to be carefully administered to prevent hyperglycemia from his diabetes and hypoglycemia from his cirrhosis (17).

Secondly, the presence of hypervolemia in our patient could have been a consequence of both his cirrhosis and kidney failure. In either case, fluid overload is primarily treated with dietary sodium restriction and diuretic therapy, especially if there is residual renal function (18, 19). However, there are certain nuances that need to be taken into account. For example, diuretic resistance is frequently encountered in CKD patients. Furthermore, the choice of diuretic must be guided based on their excretion patterns. As furosemide is mainly excreted by the kidney, it would be the first choice in cirrhotic CKD patients whose kidney function is better preserved than their liver function. Similarly, torasemide is preferred in patients where kidney function is severely impaired (20).

Additionally, as PD is known to lead to increased protein losses, the already existing hypoalbuminemia in our patient due to his cirrhosis had to be carefully monitored. The patient received multiple albumin infusions as a part of his treatment plan. As a result, a drop in his albumin and total protein levels after PD was not observed. This highlights the importance of tailoring treatment strategies to address individual complications in patients with multiple morbidities.

The severe impairment of kidney function in this case underscores the value of an integrated approach when managing multiple chronic conditions. As his HIV and hepatitis C status might have exacerbated his kidney dysfunction, it is vital to ensure that in such patients comorbid conditions are being properly managed. Prompt treatment of HCV is essential to prevent liver-associated complications. In addition to that, antiretrovirals can be nephrotoxic and requires close monitoring. Thus, patient education to emphasize the significance of treatment adherence and regular follow-up is necessary to prevent progression of the chronic conditions, including CKD.

This case highlights several avenues for future research. Currently, no treatment guidelines exist for the management of patients with the combined diagnoses of diabetes, HIV, HCV, liver cirrhosis, and CKD. Thus, there is a need to optimize treatment protocols for such challenging patients. For instance, as regional hospitals often hesitate to provide hemodialysis to patients with HCV and HIV due to the risk of transmission of these viruses, a PD guideline tailored to cirrhotic patients could be established and implemented so that these patients can receive renal replacement therapy. This guideline should also include how patients should be selected for PD such as the consideration of socioeconomic conditions, compliance and mental capacity (which might be impaired in the case of hepatic encephalopathy) (15). Moreover, integrated care programs that address the unique challenges faced in patients with multiple chronic diseases should be developed by policymakers.

Conclusion

While PD is generally avoided in patients with cirrhosis and ascites due to the fear of higher risk of complications, current research indicates that this may not be the case. Thrombocytopenia and hemodynamic instability in these patients might even make PD the better choice. In our case report, we have presented a

patient with a unique set of comorbidities who successfully underwent the initiation of PD.

Author Contributions: Conceptualization, Z.K., A.K.; methodology, Z.K., M.M., L.T., V.A.; writing – original draft preparation, N.I., R.B.; writing – review and editing, B.A. All authors have read and agreed to the published version of the manuscript.

Disclosures: There is no conflict of interest for all authors.

Acknowledgments: None.

Funding: None.

Ethics approval: Not applicable.

References

1. Sridhar VS, Limonte CP, Groop PH, Heerspink HJ, Pratley RE, et al. Chronic kidney disease in type 1 diabetes: translation of novel type 2 diabetes therapeutics to individuals with type 1 diabetes. *Diabetologia*. 2024;67(1):3-18. **DOI**: 10.1007/s00125-023-06015-1
2. Papadopoulou-Marketou N, Chrousos GP, Kanaka-Gantenbein C. Diabetic nephropathy in type 1 diabetes: a review of early natural history, pathogenesis, and diagnosis. *Diabetes Metab Res Rev*. 2017;33(2):e2841. **DOI**: 10.1002/dmrr.2841
3. Hovind P, Rossing P, Tarnow L, Smidt UM, Parving HH. Progression of diabetic nephropathy. *Kidney Int*. 2001;59(2):702-709. **DOI**: 10.1046/j.1523-1755.2001.059002702.x
4. Herman ES, Klotman PE. HIV-associated nephropathy: Epidemiology, pathogenesis, and treatment. *Semin Nephrol*. 2003;23(2):200-208. **DOI**: 10.1053/snep.2003.50018
5. Barsoum RS, William EA, Khalil SS. Hepatitis C and kidney disease: A narrative review. *J Adv Res*. 2016;8(2):113. **DOI:** 10.1016/j.jare.2016.07.004
6. Mogensen CE, Christensen CK, Vittinghus E. The Stages in Diabetic Renal Disease: With Emphasis on the Stage of Incipient Diabetic Nephropathy. *Diabetes*. 1983;32(Supplement_2):64-78. **DOI**: 10.2337/diab.32.2.s64
7. Flandre P, Pugliese P, Cuzin L, Bagnis CI, Tack I, et al. Risk factors of chronic kidney disease in HIV-infected patients. *Clinical Journal of the American Society of Nephrology*. 2011;6(7):1700-1707. **DOI**: 10.2215/CJN.09191010
8. Swanepoel CR, Atta MG, D'Agati VD, Estrella MM, Fogo AB, et al. Kidney disease in the setting of HIV infection: conclusions from a Kidney Disease: Improving Global Outcomes (KDIGO) Controversies Conference. *Kidney International*. 2018;93(3):545-559. **DOI**: 10.1016/j.kint.2017.11.007
9. Barsoum RS, William EA, Khalil SS. Hepatitis C and kidney disease: A narrative review. *Journal of Advanced Research*. 2016;8(2):113-130. **DOI**: 10.1016/j.jare.2016.07.004
10. Rajora N, De Gregorio L, Saxena R. Peritoneal Dialysis Use in Patients With Ascites: A Review. *Am J Kidney Dis*. 2021;78(5):728-735. **DOI**: 10.1053/j.ajkd.2021.04.010
11. Selgas R, Bajo MA, Del Peso G, Sánchez-Villanueva R, Gonzalez E, et al. Peritoneal Dialysis in the Comprehensive Management of End-Stage Renal Disease Patients with Liver Cirrhosis and Ascites: Practical Aspects and Review of the Literature. *Perit Dial Int*. 2008;28(2):118-22.
12. Nader MA, Aguilar R, Sharma P, Krishnamoorthy P, Serban D, et al. In-Hospital Mortality in

Cirrhotic Patients with End-Stage Renal Disease Treated with Hemodialysis versus Peritoneal Dialysis: A Nationwide Study. *Perit Dial Int*. 2017;37(4):464-471. **DOI**: 10.3747/pdi.2016.00131

13. Chou CY, Wang SM, Liang CC, Chang CT, Liu JH, et al. Peritoneal dialysis is associated with a better survival in cirrhotic patients with chronic kidney disease. *Medicine* (United States). 2016;95(4). **DOI**: 10.1097/MD.0000000000002465
14. De Vecchi AF, Colucci P, Salerno F, Scalamogna A, Ponticelli C. Outcome of peritoneal dialysis in cirrhotic patients with chronic renal failure. *American Journal of Kidney Diseases*. 2002;40(1):161-168. **DOI**: 10.1053/ajkd.2002.33925
15. Evaluating patients for chronic peritoneal dialysis and selection of modality - UpToDate. Accessed February 13, 2025. https://www.uptodate.com/contents/evaluating-patients-for-chronic-peritoneal-dialysis-and-selection-of-modality?search=peritoneal%20dialysis&source=search_result&selectedTitle=1%7E150&usage_type=default&display_rank=1#H769822640
16. Fathima N, Kashif T, Janapala RN, Jayaraj JS, Qaseem A. Single-best Choice Between Intermittent Versus Continuous Renal Replacement Therapy: A Review. *Cureus*. 2019;11(9):e5558. **DOI**: 10.7759/cureus.5558
17. Puri P, Kotwal N. An Approach to the Management of Diabetes Mellitus in Cirrhosis: A Primer for the Hepatologist. *J Clin Exp Hepatol.* 2022;12(2):560-574. **DOI**: 10.1016/j.jceh.2021.09.010
18. Biggins SW, Angeli P, Garcia-Tsao G, Ginès P, Ling SC, et al. Diagnosis, Evaluation, and Management of Ascites, Spontaneous Bacterial Peritonitis and Hepatorenal Syndrome: 2021 Practice Guidance by the American Association for the Study of Liver Diseases. *Hepatology*. 2021;74(2):1014-1048. **DOI**: 10.1002/hep.31884
19. Overview of the management of chronic kidney disease in adults - UpToDate. Accessed February 12, 2025. https://www.uptodate.com/contents/overview-of-the-management-of-chronic-kidney-disease-in-adults?search=management%20of%20hypervolemia%20in%20patients%20on%20dialysis%20&source=search_result&selectedTitle=11%7E150&usage_type=default&display_rank=11#H13
20. Kumar R, Priyadarshi RN, Anand U. Chronic renal dysfunction in cirrhosis: A new frontier in hepatology. *World J Gastroenterol*. 2021;27(11):990. **DOI**: 10.3748/wjg.v27.i11.990