HSPI The Open Access Publisher

JCN ISSN 2576-9529 JOURNAL OF CLINICAL NEPHROLOGY

Case Report

An Intriguing Case of Rapidly Progressive Renal Failure in an Acquired Immunodeficiency Syndrome Patient

Pulak Azad[1]*, Lakshmikant Jha[2] and Pranav Tyagi[3]

[1]1st year Dr, NB Resident, Nephrology, Dharamshala Narayana Superspeciality Hospital, Delhi-110096, India

[2]Senior Consultant and Director, Nephrology, Dharmashilla Narayana Superspeciality Hospital, Delhi, India

[3]Associate Consultant, Nephrology, Dharmashilla Narayana Superspeciality Hospital, Delhi, India

More Information

***Corresponding author:** Dr. Pulak Azad, I[st] Year, Dr. NB Resident, Nephrology, Dharamshala Narayana Superspeciality Hospital, Delhi-110096, India, Email: azad.aditya11@gmail.com

Submitted: November 08, 2025
Accepted: November 28, 2025
Published: November 29, 2025

Citation: Azad P, Jha L, Tyagi P. An Intriguing Case of Rapidly Progressive Renal Failure in an Acquired Immunodeficiency Syndrome Patient. J Clini Nephrol. 2025; 9(11): 107-109. Available from: https://dx.doi.org/10.29328/journal.jcn.1001167

Keywords: Rapidly progressive renal failure; Acquired immunodeficiency patient; Acute interstitial nephritis

Abstract

Rapidly progressive renal failure in a patient living with human immunodeficiency virus is a curious scenario that encompasses differentials such as thrombotic microangiopathy, Immune reconstitution inflammatory syndrome, acute interstitial nephritis, and acute tubular necrosis. A 36-year-old male patient was admitted with fever and weakness for 2 months. He was detected to have pulmonary Koch's and Human immunodeficiency virus positive. He was initiated on anti-tubercular regiemn followed by highly active anti-retroviral therapy. Patient also had decreased urine output since past 3 weeks. He had baseline creatinine of 1 which increased to 2.1 in 1st week, 3.2 in 2nd week and had progressive derangement in KFT for 5.1 in 3rd week. He was admitted in the 4th week when his serum creatinine was 6.9. As it further rose to 8.1 along with decreased urine output biopsy was planned in view of rapidly progressive renal failure and the patient was initiated on hemodialysis. Biopsy was suggestive of non-necrotizing granulomatous interstitial nephritis and glomeruli showed non – proliferative morphology. After highly active anti-retroviral therapy modification and steroid addition serum creatinine started coming down and patient became dialysis independent.

Introduction

Patient living with human immunodeficiency virus infection often lands up in rapidly progressive renal failure due to human immunodeficiency virus associated nephropathy, human immunodeficiency virus associated immune-complex kidney disease, thrombotic microangiopathy, immune reconstitution inflammatory syndrome, drug iduced acute interstitial nephritis and acute tubular necrosis. With the advent of highly active anti-retroviral therapy the survival of acquired immunodeficiency patient have improved and incidence of renal dysfunction has risen [1].

We present a case of 36 years old male patient who was admitted with fever and weakness for 2 months. In view of pyrexia of unknown origin he was thoroughly investigated and was found to have pulmonary Koch's and human immunodeficiency virus positive. As his earlier CD 4 count was 45 he was initiated on anti-tubercular regiemn 1st and as CD 4 count improved to 145 highly active anti-retroviral therapy was introduced in the form of lamivudine, efavirenz and tenofovir. Patient developed decreased urine output. During the period of 3 weeks he had baseline creatinine of 0.9 which increased to 2.1 in 1st week, 3.2 in 2nd week and had progressive derangement in KFT for 5.1 in 3rd week. He was admitted in the 4th week when his serum creatinine was 6.9. Anti-tubercular drug dosage were modified as per estimated glomerular filtration rate.

Urine examination was suggestive of +1 proteinuria. Renal biopsy was planned in view of rapidly progressive renal failure and the patient was initiated on hemodialysis.

Biopsy was suggestive of non-necrotizing granulomatous interstitial nephritis and glomeruli showed non – proliferative morphology.

Tenofovir was replaced by dolutegravir and oral steroid were added [2]. After highly active anti-retroviral therapy modification and steroid addition serum creatinine started coming down and patient became dialysis independent.

Rapidly progressive renal failure in a retro-positive patient brings a plethora of possibilities. Renal biopsy can rescue us in such perplex scenarios. Some common cause of RPRF particularly before the widespread use of antiretroviral therapy were namely to human immunodeficiency virus associated nephropathy, human immunodeficiency virus associated immune-complex kidney disease, thrombotic microangiopathy, immune reconstitution inflammatory syndrome but in today's world with improved health-care system and survival of patient ;drug induced acute interstitial nephritis has become more prevalent cause of the same [2].

Case presentation

A 36-year-old male patient presented with a history of intermittent fever and generalized weakness for two months. He had no prior history of diabetes mellitus, hypertension, chronic kidney disease, or exposure to nephrotoxic agents [2]. During evaluation for pyrexia of unknown origin, he was diagnosed with pulmonary tuberculosis and was found to be seropositive for human immunodeficiency virus infection. At the time of diagnosis, his CD4 count was 45 cells/μL. Anti-tubercular therapy was initiated first, in accordance with national guidelines, to reduce the risk of immune reconstitution inflammatory syndrome (IRIS) [3].

Following partial immune recovery, evidenced by an improvement in CD4 count to 145 cells/μL, highly active antiretroviral therapy (HAART) was commenced using a combination of lamivudine, efavirenz, and tenofovir. Baseline renal function prior to HAART initiation revealed a serum creatinine of 0.9 mg/dL with normal urine output. Over the subsequent three weeks, the patient developed progressively decreasing urine output accompanied by a rapid rise in serum creatinine levels—2.1 mg/dL in the first week, 3.2 mg/dL in the second week, and 5.1 mg/dL by the third week.

He was admitted during the fourth week of illness with features of uremia, oliguria, and a serum creatinine of 6.9 mg/dL. Anti-tubercular drug dosages were adjusted based on estimated glomerular filtration rate. Laboratory investigations revealed mild proteinuria (+1) on urine examination without hematuria or active urinary sediments. Ultrasonography of the abdomen showed normal-sized kidneys with preserved corticomedullary differentiation. Despite supportive management, renal function continued to deteriorate, and serum creatinine rose further to 8.1 mg/dL, necessitating initiation of hemodialysis.

In view of rapidly progressive renal failure in an HIV-positive patient, a renal biopsy was performed to establish the underlying etiology and guide management. Histopathological examination revealed non-necrotizing granulomatous interstitial nephritis with interstitial inflammation, while the glomeruli demonstrated non-proliferative morphology without evidence of collapsing glomerulopathy, immune-complex deposition, or thrombotic microangiopathy [4,5]. These findings favored a diagnosis of drug-induced granulomatous interstitial nephritis.

Tenofovir was discontinued and replaced with dolutegravir, given its more favorable renal safety profile [6]. Oral corticosteroid therapy was initiated after exclusion of active disseminated infection. Over the following weeks, the patient demonstrated a gradual improvement in urine output and renal function. Hemodialysis was discontinued, and serum creatinine levels progressively declined, rendering the patient dialysis-independent on follow-up.

Discussion

Renal involvement in patients living with HIV represents a diagnostic challenge due to the wide spectrum of potential etiologies, including HIV-associated nephropathy, immune-complex mediated glomerulonephritis, thrombotic microangiopathy, IRIS, and drug-induced nephrotoxicity [7]. With increasing survival and widespread use of antiretroviral therapy, medication-related renal injury has emerged as a significant and potentially reversible cause of kidney dysfunction in this population [8].

Tenofovir disoproxil fumarate is a well-recognized cause of proximal tubular toxicity and interstitial nephritis, particularly in patients with advanced HIV disease, low CD4 counts, and concomitant use of nephrotoxic agents [9]. Granulomatous interstitial nephritis, although rare, has been reported in association with antiretroviral drugs and anti-tubercular therapy, necessitating a high index of suspicion [10]. In this case, the absence of glomerular pathology and the presence of non-necrotizing granulomas on biopsy supported a diagnosis of drug-induced interstitial nephritis rather than HIV-associated nephropathy or IRIS [11].

Renal biopsy plays a pivotal role in differentiating among these conditions, particularly when clinical and laboratory findings are non-specific [12]. Early histological diagnosis allows timely modification of antiretroviral therapy and initiation of corticosteroids, which has been shown to improve renal recovery in selected cases of interstitial nephritis [13].

This case underscores the importance of individualized renal evaluation in HIV-positive patients presenting with rapidly progressive renal failure. Prompt recognition of reversible causes, judicious use of renal biopsy, and timely therapeutic intervention can significantly alter outcomes and prevent long-term dialysis dependence [14].

Conclusion

Rapidly progressive renal failure in patients living with HIV encompasses a broad differential diagnosis that extends beyond classical HIV-associated nephropathies. Drug-induced interstitial nephritis remains an important, potentially reversible cause in the era of widespread antiretroviral therapy. This case highlights the indispensable role of renal biopsy in establishing an accurate diagnosis and guiding targeted therapy. Early identification and modification of offending agents, combined with appropriate immunosuppressive treatment when indicated, can result in significant renal recovery and avoidance of long-term renal replacement therapy.

Patient consent statement

Written informed consent was obtained from the patient for publication of this case report and accompanying clinical data, in accordance with institutional ethical standards.

References

1. Ross MJ. Advances in the pathogenesis of HIV-associated kidney diseases. Kidney Int. 2014;86(2):266–274. Available from: https://pubmed.ncbi.nlm.nih.gov/24827777/
2. McLaughlin MM, Guerrero AJ, Merker A. Renal effects of non-tenofovir antiretroviral therapy in patients living with HIV. Drugs Context. 2018;7:212519. Available from: https://pubmed.ncbi.nlm.nih.gov/29623097/
3. Meintjes G, Lawn SD, Scano F, Maartens G, French MA, Worodria W, et al. Immune reconstitution inflammatory syndrome associated with HIV infection: risk factors, pathogenesis and management. Lancet Infect Dis. 2008;8(8):516–523. Available from: https://pubmed.ncbi.nlm.nih.gov/18652998/
4. Haas M, Kaul S, Eustace JA. HIV-associated immune complex glomerulonephritis with lupus-like features: a clinicopathologic study of 14 cases. Clin J Am Soc Nephrol. 2009;4(8):1301–1307. Available from: https://pubmed.ncbi.nlm.nih.gov/15780090/
5. Perazella MA. Drug-induced acute interstitial nephritis: the tip of the iceberg. Nat Rev Nephrol. 2010;6(8):461-70. Available from: https://doi.org/10.1038/nrneph.2010.71
6. Cahn P, Pozniak AL, Mingrone H. Dolutegravir versus efavirenz in antiretroviral-naïve adults with HIV-1 infection. Lancet. 2013;382(9893):700–708.
7. Wyatt CM. Kidney disease and HIV infection. Top Antivir Med. 2017;25(1):13–16. Available from: https://pubmed.ncbi.nlm.nih.gov/28402929/
8. Gupta SK. Tenofovir-associated Fanconi syndrome: review of the FDA adverse event reporting system. AIDS. 2008;22(2):99-103. Available from: https://doi.org/10.1089/apc.2007.0052
9. Hall AM, Hendry BM, Nitsch D, Connolly JO. Tenofovir-associated kidney toxicity in HIV-infected patients: a review of the evidence. Kidney Int. 2011;79(8):825–832. Available from: https://pubmed.ncbi.nlm.nih.gov/21412255/
10. Muriithi AK, Leung N, Valeri AM. Biopsy-proven granulomatous interstitial nephritis: clinical characteristics and outcomes. Kidney Int. 2012;82(7):797–803.
11. D'Agati VD, Appel GB. HIV infection and the kidney. J Am Soc Nephrol. 1997;8(1):138–152. Available from: https://doi.org/10.1681/asn.v81138
12. Wearne N, Swanepoel CR, Boulle A. HIV-associated renal disease in Africa—a changing landscape. Nat Rev Nephrol. 2012;8(4):189–199.
13. Clarkson MR, Giblin L, O'Connell FP, O'Kelly P, Walshe JJ, Conlon PJ, et al. Acute interstitial nephritis: clinical features and response to corticosteroid therapy. Kidney Int. 2004;66(3):1078–1083. Available from: https://doi.org/10.1093/ndt/gfh485
14. Atta MG, Fine DM. HIV-associated nephropathy and the evolving role of antiretroviral therapy. Clin Infect Dis. 2016;63(3):332–339.