Case Report

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IgA Nephropathy in a HIV Positive Patient: A Rare Case Scenario

Internal Medicine Section

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ABSTRACT

Human Immunodeficiency Virus (HIV) affects almost every organ in the body. There may be direct cytopathic effects, or the affected individual may be infected by opportunistic organisms. HIV has been known to cause various renal syndromes and histopathological conditions. The kidneys are among the most commonly affected organs in individuals with HIV. Renal dysfunction is the most frequent complication of HIV, both before and after the introduction of Antiretroviral Therapy (ART), and it is the leading cause of mortality in AIDS. Kidney disease can present as HIV-associated nephropathy (HIVAN), immune complex disease in HIV, or Thrombotic Microangiopathy (TMA). HIV-associated focal glomerulosclerosis is the most common biopsy finding; however, other conditions, such as IgA nephropathy, may also be observed in certain cases. This report discusses a 21-year-old male HIV patient who developed renal failure and subsequently required haemodialysis. To determine the cause of the renal failure, a decision was made to perform a renal biopsy. The renal biopsy results were suggestive of IgA nephropathy. The patient was treated with corticosteroids and did not require dialysis thereafter. This case represents a rare scenario in which a patient with HIV presented with renal failure due to IgA nephropathy. Thus, despite its rarity, IgA nephropathy should be considered as a differential diagnosis in HIV patients experiencing renal failure.

Keywords: Acute immunodeficiency syndrome-associated nephropathy, Human immunodeficiency virus, Immunoglobulin A, Renal dialysis, Renal insufficiency

CASE REPORT

A 21-year-old male, known to be affected by HIV since childhood, presented to the outpatient department with complaints of nausea, vomiting, and reduced appetite. The patient did not have a previous history of diabetes mellitus, hypertension, or any other comorbidities. He had been receiving antiretroviral treatment for the last 15 years and was currently on a regimen comprising Dolutegravir, Lamivudine, and Tenofovir alafenamide. His previous medical records indicated unremarkable laboratory results, including normal renal function tests. His last CD4 count, performed the previous week, was 868 cells per cubic millimetre. Upon examination, his blood pressure was recorded at 164/98 mmHg. The laboratory parameters are detailed in [Table/Fig-1].

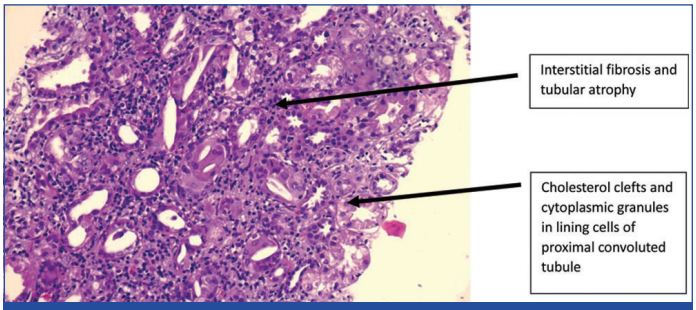
Laboratory parameter	Before treatment	After treatment
Serum urea (mg/dL)	78	71
Serum creatinine (mg/dL)	4.72	2.03
Serum potassium (meq/L)	5.44	3.45
Urine protein creatinine ratio (g/g)	5.88	1.54
Urinalysis	Protein 3+, RBCs 12-15/hpf and pus cells 6-8/hpf	Protein 1+, RBCs 1-2/hpf and pus cells 1-2/hpf

[Table/Fig-1]: Laboratory parameters.

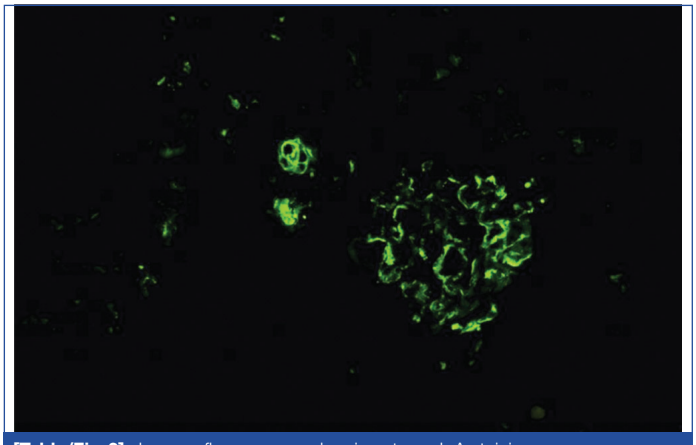
Upon admission, his laboratory results indicated hyperkalaemia (5.44 meq/L), urea 78 mg/dL, serum creatinine 4.72 mg/dL, and an estimated Glomerular Filtration Rate (eGFR) by the Chronic Kidney Disease Epidemiology Collaboration (CKD-EPI) of 17 mL/min/1.73 m^2. The urine protein-creatinine ratio was 5.88 g/g, and the urinalysis showed protein 3+, 12-15 Red Blood Cells (RBCs) per high power field (hpf), and 6-8 pus cells per hpf. His screening panel for Hepatitis B surface Antigen (HBsAg) and Hepatitis C Virus (HCV) was negative. Serum C3 levels were 92.9 mg/dL (normal limits 75 to 175 mg/dL) [1], and serum C4 levels were 16.0 mg/dL (normal limits 14 to 40 mg/dL) [2]. The immunological screening panel for antinuclear antibodies and antineutrophil cytoplasmic antibodies (myeloperoxidase and proteinase 3) was negative.

In light of his symptoms and hyperkalaemia, the patient was initiated on haemodialysis. Tenofovir was discontinued, and Abacavir was started.

As the aetiology of the renal failure was unclear, a decision was made to perform a renal biopsy. After obtaining informed consent and confirming that the coagulation parameters were normal, the renal biopsy was conducted [Table/Fig-2,3] under sonographic guidance, and two cores were sent for light microscopy and immunofluorescence.



[Table/Fig-2]: Light microscopy findings of renal biopsy (haematoxylin and eosin stain; 40x magnification).



[Table/Fig-3]: Immunofluorescence showing strong IgA staining.

Light microscopy revealed 10 glomeruli, of which one was globally sclerosed. Mesangial and endocapillary proliferative crescentic IgA nephropathy was identified, with crescent formation in six (60%) of the glomeruli (four cellular and two fibrocellular). Severe acute tubular injury (interstitial fibrosis and tubular atrophy 35-40%) was noted, with granular casts, a few foci of cholesterol clefts, and prominence of cytoplasmic granules in the lining cells of the proximal convoluted tubule. Direct immunofluorescence demonstrated IgA: 3+ mesangial and segmental capillary wall; granular. IgG and C1q were negative, while IgM showed segmental entrapment. C3 revealed 2-3+ mesangial and segmental capillary wall; granular. Kappa and lambda light chains were 2+ and 3+, respectively, in the mesangial and segmental capillary wall; granular. The MEST-C scores (Oxford classification of IgA nephropathy) [3] were M1E1S1T1C1. The renal biopsy results were suggestive of proliferative crescentic IgA nephropathy.

The patient was subsequently treated with pulse methylprednisolone 500 mg for three days, followed by tapering doses of oral prednisone, along with an Angiotensin Receptor Blocker (ARB), Telmisartan 40 mg/day. The renal function and proteinuria improved (eGFR 48 mL/min/1.73 m^2), and the patient did not require any further dialysis.

DISCUSSION

HIV presents in a myriad of ways, with kidney involvement being the fourth leading cause of mortality among patients with HIV and Acquired Immunodeficiency Syndrome (AIDS) [4]. Impaired Kidney Function (IKF) is a broad term that encompasses everything from asymptomatic kidney dysfunction to End-Stage Kidney Disease (ESKD) [5]. The widespread use of Antiretroviral Treatment (ART) has transformed HIV infection into a chronic illness. Several syndromes affecting the kidneys have been described, including Acute Kidney Injury (AKI), HIV-associated kidney disease, comorbid Chronic Kidney Disease (CKD), and treatment-related kidney toxicity.

The classical renal manifestation of HIV infection is HIV-associated nephropathy (HIVAN), which encompasses collapsing glomerulopathy, HIV-immune complex kidney disease, and thrombotic microangiopathy [6]. The classical finding on renal biopsies in HIV patients is related to HIVAN and includes Focal Segmental Glomerulosclerosis (FSGS) [7].

IgA nephropathy has been reported in HIV patients but is considered uncommon [8]. A case report of IgA nephropathy in a child affected by AIDS has been documented; however, the presentation involved recurrent haematuria rather than renal failure, as seen in the current case [9]. A study conducted at a tertiary care centre in North India from 2010 to 2013 screened HIV-positive patients for proteinuria. Participants with dipstick proteinuria exceeding 1 g/24 h underwent renal biopsy. Glomerulonephritis was observed in the majority of those who were biopsied, with the most common finding being mesangioproliferative glomerulonephritis [10]. However, IgA nephropathy was not identified in any of the biopsies, suggesting that it is a rare diagnosis among such patients.

IgA antibodies have been described as a part of the early immune response to HIV infection [11]. Genetic factors or immune complexes may play a role in the development of IgA nephropathy in these individuals [12]. The deposition of Circulating Immune Complexes (CICs) in the kidney is postulated to be a cause of HIV-associated glomerulonephritis. Immunoglobulins (IgG, IgM, IgA) bind to HIV antigens (p24, gp41, and gp120), forming CICs. These CICs can form at any stage of HIV infection, with a significant portion comprising IgA [13]. Consequently, there is immune dysfunction associated with HIV infection, which results in kidney disease.

The histopathology of HIV-associated IgA nephropathy typically shows diffuse or segmental mesangial matrix expansion with proliferative changes [14]. Fibrocellular crescents may also be present [14]. Immunofluorescence is usually positive for IgA, along with varying amounts of IgM, C3, C1q, and IgG [14]. Electron microscopy commonly reveals thickened foot processes, mesangial expansion, and mesangial and peripheral intramembranous and/or subepithelial electron-dense deposits [14]. There is a paucity of randomised controlled trials concerning kidney diseases associated with HIV [15].

ART has been found to preserve and improve renal function, as measured by the eGFR, in patients with HIV, both with and without pre-existing renal dysfunction. Furthermore, ART is recommended for all patients with HIV and kidney disease [16]. However, the present patient was already on ART. The Kidney Disease: Improving Global Outcomes (KDIGO) 2021 Clinical Practice Guideline for the Management of Glomerular Diseases suggests corticosteroids on a case-by-case basis [16]. The guidelines also indicate a beneficial effect for Angiotensin-Converting Enzyme Inhibitors (ACEi) and Angiotensin Receptor Blockers (ARBs). Accordingly, corticosteroids and an ARB were administered to the present patient.

This case is particularly rare as the patient had no previous history of renal failure, yet developed de novo renal failure to the extent that haemodialysis was required. The patient's renal function showed a dramatic response to corticosteroids, and he did not need any further dialysis. This is a unique case scenario in which IgA nephropathy presented as renal failure, a presentation that has not been previously described in the literature.

CONCLUSION(S)

This is a rare case presentation of IgA nephropathy in HIV. Along with the usual clinical syndromes, clinicians need to consider this readily treatable possibility in cases of renal failure. Additionally, the threshold for performing a diagnostic kidney biopsy should be kept low.

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