Case Report

Open Access

CrossMark

Obstructive uropathy secondary to antiretroviral agents

Abstract

Acute kidney injury (AKI) and chronic kidney disease (CKD) are more common in HIV-infected patients than in the general population. We observed an HIV(+) young man who presents an unusual cause of acute renal injury (obstructive uropathy as a consequence of lithiases secondary to antiretroviral therapy). The differential diagnosis of kidney diseases associated with HIV infections includes: glomerular, tubular and interstitial causes. A renal biopsy is a useful tool in the diagnostic approach. Nephrologists and urologists must be aware of HIV- related renal affection, including toxicity related to HAART schedules.

Keywords: acute kidney injury, HIV Obstructive uropathy, HAART toxicity

Volume 5 Issue 2 - 2017

Sonia Mastrapasqua, Oscar Escobar Mat as Melideo, Maximiliano Ramirez, Carolina Martinez

Servicio de Nefrologia, Hospital Provincial Neuqu

Correspondence: Sonia Mastrapasqua, Servicio de Nefrologia, Hospital Provincial Neuqu, Email soniamastra@hotmail.com

Received: May 10, 2016 | **Published:** July 24, 2017

Introduction

Acute kidney injury (AKI) and chronic kidney disease (CKD) are more common in HIV-infected patients than in the general population. Hospitalized persons and those with several comorbidities are at higher risk or renal failure.[1] AKI is associated with an increased risk of heart failure, cardiovascular disease, end-stage renal disease (ESRD) and mortality.[2] Here, we report an HIV(+) adult male patient who developed acute renal injury secondary to obstructive uropathy as a consequence of antiretroviral therapy.

Case report

A 33-year-old man was admitted to hospital with seizures due to cerebral toxoplasmosis. The patient's past medical history was positive for HIV and Hepatitis B infections. While hospitalized, HAART (Tenofovir+lamivudine, atazanavir, ritonavir) and antiparasitic therapy were initiated. Within a month, he developed a febrile syndrome and CMV dissemination; treatment with ganciclovir was indicated. Abdominal ultrasound (US) and renal function were normal (creatinine 1.03 mg%.) Three weeks later, he developed oliguric AKI (creatinine 5.39 mg). Urinary sediment showed some cells, WBC 6-8 per HPF, and microhematuria without proteinuria. His blood pressure was of 140/90 mm Hg. The differential diagnosis included: interstitial nephritis secondary to HAART, prerenal Acute Tubular Necrosis (the patient presented mild dehydration) and Drug toxicity. Tenofovir was discontinued. The immunological studies ANCA, Anti-DNA, Complement, and ANF were normal. A percutaneous renal biopsy was indicated. (Table 1) laboratory data.

The Renal US revealed a dilated aspect of the kidneys, and bilateral ureteral and pyelocaliceal ectasia, with the presence of bilateral lithiasis in the ureteropelvic junction. CT scan confirmed both bilateral ureteropyelic ectasia (23mm) and lithiasic images (7.5 mm right kidney and 10 mm the left). There was a presence of dense material in both distal ureters. (Figure 1) The clinical picture was interpreted as obstructive AKI secondary to bilateral nephrolithiasis caused by HAART + Ganciclovir. A right double "J" catheter was inserted by cystoscopy; in the left ureter, a discharge of multiple small calculi was observed. Retrograde pyelogram did not show any obstructions at any level. The patient has discontinued Ganciclovir, and the treatment schedule was changed to include antiretroviral therapy without atazanavir. The patient developed post-obstructive polyuria, without hemodynamic or electrolyte abnormalities and he also showed a recovering normal renal function.

Table 1 Laboratory data during hospitalization

Days	1	6	9	10	15	16
Hb/WBC	11/4560	9,8/3080	10,9/690	9,7/690	7,8/640	7,6803
Urea (mg/dl)	19	36	113	114	37	19
Cr (mg/dl)	1	1,66	5,39	5,6	2,4	1,35
K/Na (meq/1)	4,3/135	4,4/135	5/131	4,5/128	3/140	3,6/137
Albumin (g/dl)			2,7			

US: RBC partially covered HPPF, leucocytes 6-8 HPF, no casts, negative glucosuria, u pH: 6 Others: Viral load 226000 copies. CD 4: 70 cells, negative nirnak cinokenebt ANA and ANCA, negative cryoglobulins.

Table 2 Differential diagnosis of AKI and CKD is mandatory

Renal Diseases	
Glomerular	HIV-associated nephropathy, FSGS, IgA nephropathy, membranous nephropathy, membranoproliferative GN, minimal change disease, thrombotic microangiopathy.
Tubular	Crystal nephropathy, acute tubular injury, Fanconi syndrome, diabetes insipidus.
Interstitial	Interstitial nephritis.

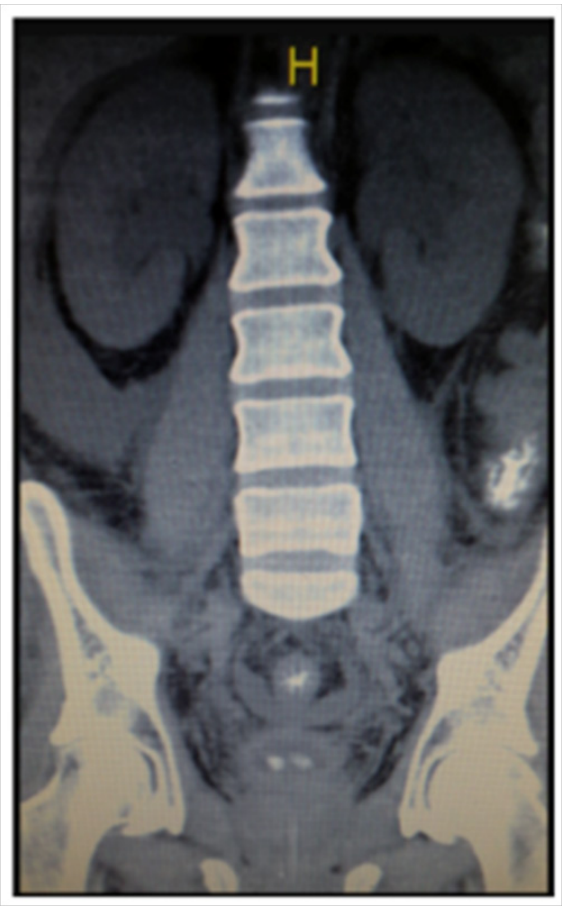

Figure 1 Abdominal CT Scan: Bilateral hydronephrosis. Bilateral lithiasis in the ureterovesical junction.

Discussion

In HIV(+) patients, different clinical situations should be studied. The major renal diseases associated with HIV infection include glomerular, tubular and interstitial causes.[3,4] Differential diagnosis of AKI and CKD is mandatory (Table 2). CKD may be present in patients suffering from comorbidities like diabetes and hypertension and HIV-associated nephropathy is a possible renal complication in this population. Drug toxicity (tenofovir, indinavir) should also be taken into consideration. In the presence of AKI, pre- renal causes should be excluded (dehydration, non-HIV-related causes, and gastrointestinal diseases). If parenchymal disease is suspected, glomerular injury (HIV related or drug induced) interstitial lesion (infection HIV-related: HIV or non HIV-related, or drug-induced) and tubulopathy should be discarded (drug-induced Fanconi Syndrome or nephrogenic diabetes insipidus, and viral tubular toxicity.) Systemic, urinary tract opportunistic infections and drug toxicity can cause AKI.

Obstructive post-renal AKI may be secondary to drug induced tubular crystal deposition. Tenofovir toxicity causes a proximal tubulopathy which wastes phosphorus, glucose, amino acids and bicarbonate, which are normally reabsorbed. This damage is usually repaired when the drug is discontinued, but if it persists, it may lead to fibrosis and chronic lesions. Atazanavir and Indinavir can produce precipitation of crystals and develop inflammatory mechanisms and interstitial nephritis (risk factors include dehydration, alkaline urine pH, and previous lithiases episodes). Patients with chronic hepatitis C coinfection have a greater risk of lithiases. Indinavir-associated calculi are very small (2- 6 mm) and they are usually eliminated, but tubular deposition may cause post-renal obstructive failure.[4-6] Ganciclovir is also nephrotoxic and it produces intratubular crystal precipitation.

Summary

We report an HIV-positive patient with an unusual cause of AKI. In this case, probable etiologies included drug toxicity (atazanavir and/or ganciclovir) in a person with multiple risk factors, such as hospitalization, dehydration and febrile syndrome In the case of renal dysfunction, diagnostic studies in order to characterize etiology are urgent. Renal biopsy is a useful diagnostic procedure in these patients.

In HIV-positive patients, renal failure may be HIV-related, secondary to infections or their treatment and finally due to HAART side effects. Protease inhibitors and their lithogenic capacity may lead to modifications in HAART schedules.[3,4] Nephrologists and urologists must be aware of HIV-related renal diseases and toxicity of antiretroviral drugs.

Acknowledgments

None.

Conflicts of Interset

None.

References

1. Li Y, Shlipak MG, Grunfeld C, et al. Incidence and Risk Factors for Acute Kidney Injury in HIV Infection. *Am J Nephrol*. 2012;35(4):327–334.
2. Wyatt CM, Arons RR, Klotman PE, et al. Acute renal failure in hospitalized patients with HIV: risk factors and impact on in-hospital mortality. *AIDS*. 2006;20(4):561–565.
3. Domingo P, Knobel H, Gutierrez F, et al. Evaluacion y tratamiento de la nefropatıa en el paciente con infeccion por VIH-1. Una revision practica. *Enferm Infecc Microbiol Clin*. 2010;28(3):185–198.
4. Szczech LA, Gupta SK, Habash R, et al. The clinical epidemiology and course of the spectrum of renal diseases associated with HIV infection. *Kidney Int*. 2004;66(3):1145–1152.
5. Escudero JUJ, Alcina EL, Domínguez FO, et al. Litiasis medicamentosa en pacientes VIH + en tratamiento con Indinavir. *Arch Esp Urol*. 2008;61(1):35–40.
6. Gonzalez E, Jimenez I, Perez J. Colico renal y litiasis en pacientes HIV + tratados con inhibidores de proteasas. *Actas urol esp*. 2000;24(3):212–218.