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HIV-associated nephropathy without decline of renal function

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To the editor: Collapsing focal glomerulonephritis (CFG) is found in 2-10% of all HIV-infected patients.[1] It is the most common form of renal disease in HIV patients, appearing in over 60% of the renal biopsies made.[2] The presence of proteinuria and/or impaired renal function are associated with increased patient morbidity-mortality.[3] The management of nephropathy associated to HIV infection (NAHIV) has not been established, and most patients require renal replacement treatment a few months after the appearance of nephrotic syndrome.[4]

A 41-year-old black male with type 1 HIV infection not subjected to antiretroviral therapy was admitted with generalized edema and proteinuria in the nephrotic range. Upon admission, the blood pressure was 140/90 mmHg, and edema with fovea was seen to ankle level. The laboratory tests showed normocytic and normochromic anemia with an erythrocyte sedimentation rate of 157 mm in the first hour, normal kidney function (plasma creatinine 1.1 mg/dl and plasma clearance calculated by the MDRD equation 78.41 ml/min), proteinuria 5.83 g/day without Bence-Jones proteinuria, plasma albumin 1.6 g /dl with polyclonal band in gamma region 7.7 g/dl (IgG 9860 mg/dl, IgA 151 mg/dl, IgM 643 mg/dl), and a CD4+ count of 314 cells/mm^3. HBV, HCV and herpes group serology proved negative. A myelogram revealed reactive plasmacytosis, while a bone cylinder specimen showed intense polyclonal lymphoplasmacytosis. Renal ultrasound showed symmetrical kidney enlargement, with preserved corticomedullary differentiation but with a diffuse increase in echogenicity. The Doppler study proved normal. The kidney biopsy revealed collapsing glomerulopathy with preserved tubules and an interstitial lymphocytic and polyclonal infiltrate. Antiretroviral treatment was started with efavirenz, stavudine and lamivudine, together with furosemide and enalapril. At discharge the blood pressure was 130/80, with proteinuria 300 mg/day. The patient posteriorly returned to his country of origin and reappeared 14 months later, without any reported opportunistic processes or nephrotic manifestations. While in his country, the patient continued treatment with enalapril and started nevirapine, zidovudine as lamivudine as antiretroviral therapy. The patient was found to have normal blood pressure, with no edemas, and showed normocytic and normochromic anemia, with normal kidney function (plasma creatinine 0.98 ml/dl), proteinuria 3 g/day and plasma albumin 2.6 g/dl. The CD4+ count was 350 cells/mm^3.

Collapsing focal glomerulonephritis (CFG) is found in 2-10% of all HIV-infected patients,[1] and is the most common form of kidney involvement in black HIV-infected individuals.[2,6,7] CFG is characterized by glomerular collapse and severe tubulointerstitial alterations. The underlying pathogenesis appears to be related to viral infection – HIV infection being the most common example. NAHIV is characterized by proteinuria in the nephrotic range, with rapid deterioration of renal function. In this context, proteinuria and increased plasma creatinine are regarded as indicative of a poor prognosis.[3] At present there is no effective treatment for NAHIV, and most patients require renal replacement therapy on a chronic basis.[4] Some studies suggest that treatment with antiproteinuric agents and highly active antiretroviral therapy (HAART) can delay the progression of renal failure[8] and even reduce the incidence of NAHIV[5] – emphasis being placed on the importance of an early biopsy in these patients.[5] In our case it can be affirmed that combined HAART and angiotensin-converting enzyme inhibitor (ACEI) treatment avoided the deterioration of renal function, reducing proteinuria and resolving the nephrotic syndrome, in a black patient with NAHIV.

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Psychotropic drugs and peritoneal dialysis

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To the editor: Patient acceptance of chronic disease often involves adjustment problems and thus anxiety-depressive disorders in reaction to the new situation. These problems in turn can be aggravated by situations of physical dependency typically found in patients suffering from multiple disease processes.

Patients with chronic kidney disease subjected to dialysis moreover often experience insomnia, which reduces their quality of life and increases the mortality risk.[1] Restless legs syndrome is common in uremic patients, and worsens at night – preventing adequate sleep and constituting a mortality risk factor.[2] On the other hand, it is known that worsened quality of sleep during the first year on dialysis is associated with a shortened life expectancy.[3]

The use of benzodiazepines, which are the most widely used drugs for treating anxiety, is common in patients on dialysis. Their use is associated with important patient mortality.[4-5]

The present study analyzes physical dependency, comorbidity, the frequency of anxiety-depressive disorders, and sleep disturbances, as well as psychotropic drug consumption (benzodiazepines, non-benzodiazepinic hypnotics and antidepressants) among all patients in our Peritoneal dialysis Unit.

To this effect, we analyzed all our patients included in the peritoneal dialysis program of our Unit, with determination of the Barthel index (dependency scale for basic daily life activities), the Charlson-Bedhu comorbidity scale, and the Hamilton anxiety-depression scale. Prescribed treatment was reviewed to determine psychotropic drug consumption frequency.

There were 10 patients with a mean age of 56 ± 16 years (range 33-77). The mean duration of enrollment in the peritoneal dialysis program was 12.85 ± 12.14 months (range 1-36). Forty percent of the patients were on ambulatory continuous peritoneal dialysis and 60% on automated peritoneal dialysis. The mean modified Charlson comorbidity score was 5.5 ± 2.14 (range 4-11). According to the Barthel index, 10% of the patients showed severe dependency (35 points), 20% mild dependency (75 and 85 points), and the rest (70%) no dependency (100 points). The Hamilton anxiety-depression scale in turn indicated that 20% of the patients suffered anxiety (> 8 points), while 10% scored in the depression range (> 18 points). As regards insomnia, 50% had no sleeping difficulties. The remaining 50% tended to wake up at night, and 30% were unable to fall sleep again afterwards. Psychoactive drug consumption showed two patients to use benzodiazepines, one consumed zolpidem, one used antidepressants, and another antidepressants and benzodiazepines.

It can be concluded that our patient population suffered medium-high morbidity. Most of the patients (70%) were independent for activities of daily living. Thirty percent of our patients suffered some anxiety-depressive disorder. Insomnia was found to be very common (50%). Finally, psychotropic drug use was quite common - 50% of our patients being shown to use some drug of this kind.

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Topiramate-induced renal tubular acidosis. A case report

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To the editor: Topiramate is an antiepileptic drug also used to treat bipolar disorder, neuropathic pain and migraine. The potential side effects of the drug include metabolic acidosis due to renal bicarbonate loss and the accumulation of CO_2 in the brain, as a result of its inhibitory action upon carbonic anhydrase (renal and located in the microglia, myelin and choroid plexus).

We report the case of a 58-year-old male with a history of absence-type epilepsy subjected to treatment with topiramate (150 mg/day) for the past 10 years. He also presented chronic renal failure (CKF) not subjected to evaluation and with baseline serum creatinine 2 mg/dl, hypersomnolence under study, and pulmonary thromboembolism (PTE) secondary to deep venous thrombosis (DVT) in the right leg due to trauma, in 1981. The patient was admitted to the Service of Pneumology diagnosed with bilateral PTE associated to DVT. The Service of Nephrology was consulted due to the sustained presence of acidosis.