

Nephrotic syndrome in HIV/HCV co-infected patient

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CASE REPORT

A 44-year-old Caucasian man was hospitalized for nephrotic syndrome and rapid decline of the renal function.

He was an IV drug addict with HIV₁ and HCV co-infection detected in 2003. He had pulmonary tuberculosis in 2003 with a relapse in 2007 and visceral leishmaniasis detected in 2013 with a relapse in 2014. Arterial hypertension was diagnosis in 2013 and controlled with nifedipine.

His habitual medication was abacavir, lamivudine, darunavir, ritonavir and nifedipine.

Laboratory data are presented in Table I.

The ultrasound showed normal-sized kidneys, with sinusal hyperechogenicity. Bone biopsy showed myelodysplasia. The gastric endoscopy showed duodenal leishmaniasis.

A kidney biopsy was performed.

Table I

Laboratory values

Haemoglobin	7.2 g/dl	24 hours proteinuria	14g /24h
White blood cells	2500×10 ⁹ /L	ANA	1:160
Platelets	62 000	C ₃ / C ₄	Low/Normal
Creatinine	4.14 mg/dl	ANCA/dsDNA/cryoglobulins	Negative
Urea	125 mg/dl	Immunofixation (P e U)	Without M component
Albumin	2.7 g/dl		

KIDNEY HISTOLOGY

Deposition of an eosinophilic amorphous substance was presented in all glomeruli and vascular walls (Fig. 1). This substance was negative in silver methenamine (Figs. 2 and 3) and positive in congo red staining (Figs. 4 and 5). Light polarization revealed apple-green birefringence on congo red histological staining. The immunofluorescence done in frozen tissue showed positivity for substance A. Light kappa and lambda chain were negative.

ANATOMO-CLINICAL DIAGNOSIS

AA amyloidosis.

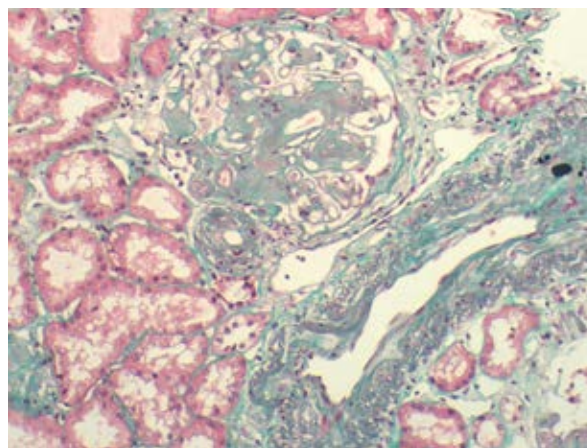


Figure 1

Masson Trichrome (x200).

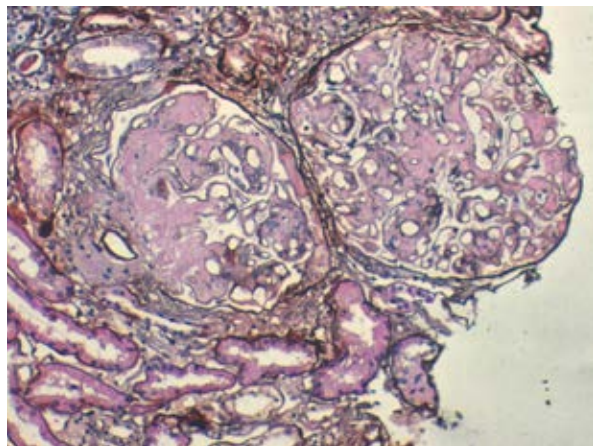


Figure 2
Silver methenamine (x200).

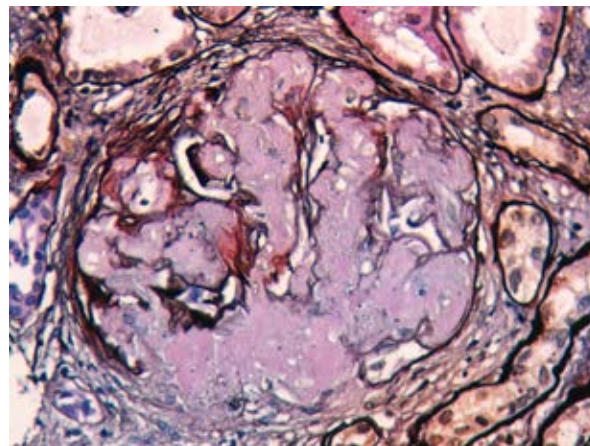


Figure 3
Silver methenamine (x400).

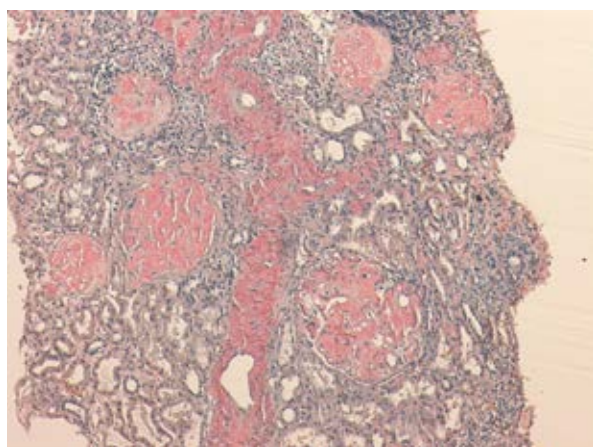


Figure 4
Congo red (x200).

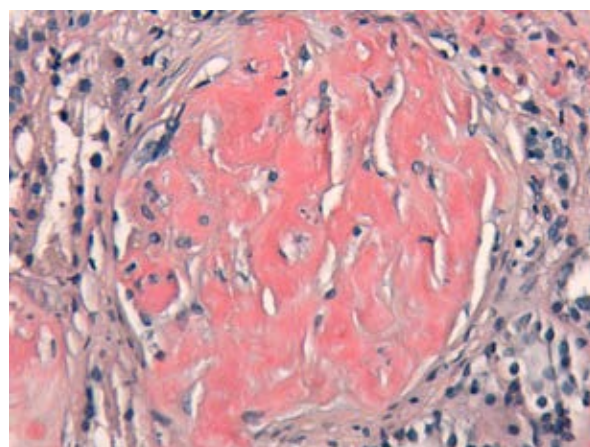


Figure 5
Congo red (x400).

DISCUSSION

The presence of HIV/HCV co-infection is particularly frequent in patients with history of IV drug addiction¹.

The kidney biopsy was indispensable for this case diagnosis. The diagnosis hypothesis was numerous: HIV nephropathy, HIV-related immune complex disease, HCV-related membranoproliferative glomerulonephritis, cryoglobulinemia, post-infectious

glomerulonephritis, amyloidosis, membranous glomerulopathy and etc.¹.

We reviewed the histological diagnoses of HIV patients ($n = 47$) over the last 15 years in our centre². AA amyloidosis was the diagnosis in eight patients (17%). From these eight patients, four were co-infected with HCV and six patients were IV drugs addicts. The typical clinical presentation was nephrotic syndrome with decline of the renal function.

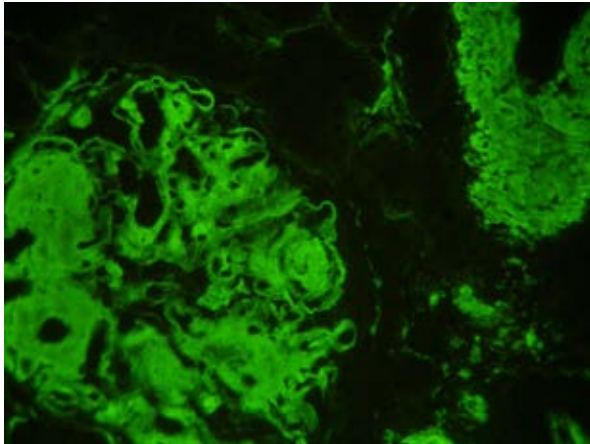


Figure 6

Immunofluorescence in frozen tissue; Substance AA (x400).

TREATMENT AND CLINICAL EVOLUTION

The duodenal leishmaniasis was treated with miltefosine and meglumine.

The patient started haemodialysis and died two weeks later.

References

1. Fine DM, Perazella MA, Lucas GM, Atta MG. Kidney biopsy in HIV: beyond HIV-associated nephropathy. *Am J Kidney Dis* 2008; 51(3):504-514.
2. Ana Azevedo, Isabel Mesquita, Helena Viana, Fernanda Carvalho, Fernando Nolasco. Amiloidose AA em doentes infectados pelo vírus da imunodeficiência humana- tão raro como se pensa? Abstract in XXIX Portuguese Congress of Nephrology.