Porphyria cutanea tarda associated with the human immunodeficiency virus infection and chronic C hepatitis

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Abstract
Porphyria cutanea tarda (PCT) associated with the human immunodeficiency virus infection was firstly described in 1987 and until now only 56 clinical cases have been published in the literature. Recent studies showed that 62% of porphyria cutanea tarda in patients with HIV.

In our clinical case there is an association with HIV infection, chronic hepatitis C, alcoholism and oral contraceptives. In this context, we believe that in patients with HIV, the role of other viral and toxic factors might be relevant in the pathogenesis and development of porphyria cutanea tarda.

Keywords: porphyria cutanea tarda, HIV infection, hepatitis C.

Introduction
Porphyria cutanea tarda belongs to the group of porphyrias, metabolic diseases resulting from enzyme deficiency on the heme synthesis route.

This is the most common kind of porphyria and the enzyme deficit is located on the hepatic decarboxylase of uroporphyrinogens.

An occasional form is often associated with several toxic factors as alcohol, estrogen, medicines, hepatitis B and more recently HIV and VHC. 1,2 We describe the case of drug addict patient, with the HIV-1 infection and chronic C-hepatitis, who developed a condition of porphyria cutanea tarda.

Case report
Female patient, 32 years of age, Caucasian, born and residing in Lisbon, computer programmer.

Seven months before admission she referred the appearance of vesicle-blistery sores on the back of her hands, fingers and forearms. Such sores were triggered by minimal traumatisms, sun exposition and alcohol intake, evolving to erosions and scars. On the last few months, complaints worsened with hyperpigmentation and hypertrichosis on the face.

In the habits and personal backgrounds, it should be highlighted: drug addiction EV (heroin and cocaine), alcohol intake non-quantifiable and seropositive for HIV-1 for the last 18 months. The medication with co-trimoxazole, zidovudine for 15 months and use of oral anti-contraceptives for the last 13 years.

She denied family backgrounds of bullous dermatosis.

The objective exam revealed a weight-loss patient with localized dermatosis on areas exposed to sunlight: hyperpigmentation and hypertrichosis of the face; cutaneous sores, evolving symmetrically on the back of the hands, fingers and forearms, made by erosions, and scars (Fig. 1); blisters and milia cysts.

The diagnostic auxiliary exams highlighted: white blood cells 2.4 X 10^{10}/L (Neutrophils 75.6%; Eosinophils 3.3%; Basophils 0.6%; Leucocytes 12.9%; Monocytes 7.6%); Platelets 91X 10^{9}/L; AST 51 U/L; ALT 64 U/L; GGT 66 U/L; alkaline phosphatase 107 U/L; iron 103 g/dL; ferritin 381 g/dL; total urinary porphyrin 652 g/dL; urinary uroporphyrin 234 g/dL (N: 15 – 50 g/dL); urinary copper porphyrin 418 g/dL (N: 35 – 150); urinary delta-aminolaevulinic acid – normal.

Negative HBV markers; HCV positive (Elisa, Western blots and protein C reactive).

Lymphocytes CD4 7/mm3.

Thorax X-Ray and abdominal ultrasound without alterations.
The histopathological exam of the blistery lesion has revealed a subepidermal blister with fenestrated dermal pavement (Fig. 2); the hepatic biopsy has revealed chronic hepatitis with minimal lesions, score 3 (Fig. 3).

The PCT diagnostic was made associated to HIV-1 infection and chronic C hepatitis.

It was seen a frank improvement of the cutaneous sores, after suspending oral anti-contraceptives drugs and alcohol, avoiding the sun; subsequent progression with a chronic-recurrent character.

**Discussion**

Wissel et al., in 1987 have described the first cases of PCT associated to infection by HIV, 1 and 56 cases can be found in the literature. 2 In most of such cases, associations to other causes of hepatopathies not related with HIV infection: alcoholism in 65%, positive serology for HBV in 57% and HCV in 46.7%. 2,3,4,5,6,7,8,9,10 The association between PCT and the HIV infection probably is not fortuitous and the role of the HIV remains controversial. Recently, De Castro et al., have demonstrated a prevalence of antibodies for the HCV on the range 62% patients with PCT. 11

In such case, a part of the HIV infection it was possible to prove chronic C hepatitis and by the CPR method, replication of HCV with positive RNAm. There were also toxic factors, namely oral anti-contraceptives, alcohol and drugs EV (cocaine and heroin).

Cribier et al., have succeeded to demonstrate the existence of interaction between the HCV and the HIV in case of co-infection, with an increase of HCV viraemia. 12

The same authors studied urinary excretion profile of porphyrins in 50 patients HIV positive, 50 patients HCV positive, 50 patients HIV positive and HCV positive and 50 witnesses HIV – CHV. None of such patients have signs of PCT or a family background of PCT and only in one patient HIV positive and HCV positive there were modifications compatible with PCT, suggesting that infections by HIV and HCV are not enough to trigger a PCT condition, and this is only revealed in predisposing patients. 12

However our case suggests that on the PCT patho-
genesis, in HIV-positive patients, there are involved other viral and toxic factors.

PCT must be included in the spectrum of diseases founds on the course of the HIV infection by HIV and HCV.

References

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