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Case Report

A Hepatitis C Virus Infection Diagnosed by Cutaneous Manifestations of Porphyria Cutanea Tarda

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Abstract

Observations: The role of hepatitis C virus (HCV) infection for the clinical presentation of porphyria cutanea tarda (PCT) remains conflicting. HCV may alter the metabolism of porphyrin in genetically predisposed individuals, and/or the treatment of HCV can trigger PCT. Here in this report, a case of PCT that introduced the diagnosis of HCV is presented. We suggest that HCV infection should be searched in all patients with PCT.

Introduction

Porphyria cutanea tarda (PCT) is frequently associated with exposure to the precipitating agents, including hepatitis C virus (HCV) that cause liver dysfunction. Here in this report, a case of PCT that introduced the diagnosis of HCV is presented.

Case Report

45 year-old man presented with multiple eroded, excoriated, hemorrhagic crusted papules, plaques and vesicles improving with post-inflammatory hypo- and hyperpigmentation, and atrophic scars that were persisting for 2 years on sun-exposed areas of skin such as the dorsum of the hands, back of the neck, face, and external ear [**Figure 1** and **Figure 2**]. He had used various treatments without any improvement. In the history, there was no special feature except several blood transfusions during an operation in 1985.

The histopathological examination of lesions revealed subepidermal blister, the slight papillomatous outstretching of papillary dermis into the vesiculation area, and a little inflammatory infiltrate consisted of mononuclear cells around the vessels at upper dermis [**Figure 3**]. Direct immunofluorescence studies revealed no deposition of complement and immunoglobulin. On laboratory examinations, the pathological findings were as follows; mildly elevated transaminase levels [AST 88 IU/liter (normal, 5-34 IU/liter) and ALT 124 IU/liter (normal, 0-55 IU/liter)], GGT; 140 IU/liter (normal, 5-64 IU/liter), and rheumatoid factor; 26.9 IU/mililiter (normal, 0-15 IU/mililiter).

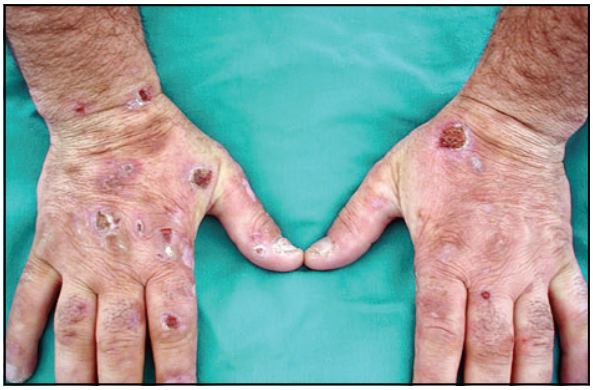

Figure 1. The eroded, excoriated lesions on hands of patient

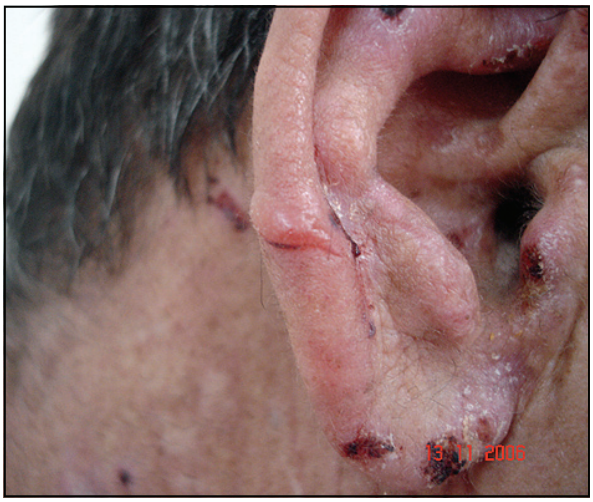

Figure 2. The bullae on auricle

Anti-HCV was positive, whereas anti-hepatitis B antigen and HIV were negative. Elevated levels of urine coproporphyrin I; 87.1 µg/24 hr (normal, <25 µg/24 hr), coproporphyrin III; 116.1 µg/24 hr; (normal, <75 µg/24 hr), uroporphyrin I, III; 2875 µg/24 hr; (normal, 25 µg/24 hr), and the skin biopsy specimen were consistent with PCT.

HCV-RNA revealed 18,900,000 transcripts per mililiter by real time polymerase chain reaction. The liver biopsy examination showed active chronic hepatitis (histologic activity index [HAI] 4, fibrosis stage 1). Peginterferon-alpha 2b (120 µg/wk) plus ribavirin (1000 mg/day) were administered. As the patient wanted to be followed-up in another city where he was living in, the efficiency of treatment in lesions could not be observed.

Discussion

Porphyria cutanea tarda, the most common type of porphyria is characterized by skin photosensitivity with blistering and occurrence of milia, skin fragility, hyper- and/or hypopigmentation on sun-exposed areas, hypertrichosis, sclerodermoid changes, dystrophic calcifications with ulcerations, scarring, alopecia, and onycholysis [**1, 2**]. The disease is caused by subnormal activity of the enzyme, uroporphyrin decarboxilase (URO-D) in the heme biosynthetic pathway. Biochemically, there are high levels of porphyrins, principally uroporphyrin, in plasma and urine [**1, 2, 3**]. In our patient PCT was observed with high levels of urine uroporphyrin.

PCT is frequently associated with exposure to the precipitating agents, including polyhalogenated aromatic hydrocarbons, alcohol abuse, estrogen ingestion, iron overload,

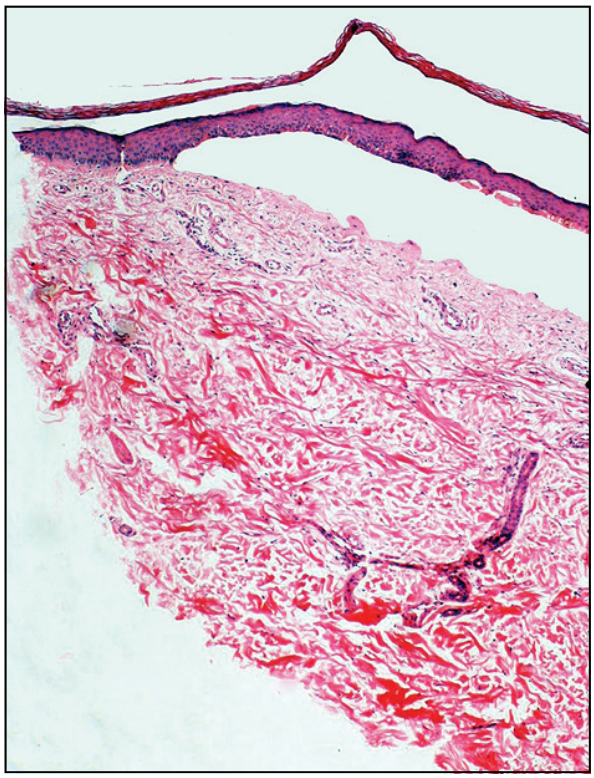

Figure 2. The bullae and eroded, excoriated lesions on auricle that reveals subepidermal blistering and a corrugated, undulating base of bullae histopathologically (H&E x40)

infections with hepatitis C virus and less frequently hepatitis B virus. All of these factors cause liver dysfunction, a common sign in PCT patients [**1, 4**]. The first evidence of a connection between one of these chemicals, hexachlorobenzene, and porphyria in humans was reported in Turkey. Between the years 1955 and 1959, hexachlorobenzene –treated seed wheat was diverted for bread production in Turkey, and PCT was diagnosed in 3000-4000 people [**5**].

Another important association of PCT is HCV infection. The incidence of HCV infection in Turkish people is 1-1.8% [**6, 7, 8**]. The prevalence of HCV antibody ratio in PCT patients was reported to be changing between 10 to 95 percentage. The exact mechanism by which HCV triggers the development of PCT is still unknown. However, HCV may alter the metabolism of porphyrin in genetically predisposed individuals as in our patient [**3, 4**].

In the treatment of PCT, avoidance of precipitating factors, phlebotomy, low-dose chloroquine therapy can be used [**1**]. There are limited numbers and contradictory data regarding the effectiveness of the therapy of

HCV infection with interferon-α and ribavirin [2]. IFN-α and ribavirin therapy had begun in our patient, however he refused further follow-up.

In conclusion, HCV plays a triggering role in the manifestation of PCT in genetically predisposed individuals. Therefore HCV infection should be searched in all patients with PCT.

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