

# Sporadic Porphyria Cutanea Tarda Induced by Alcohol Abuse

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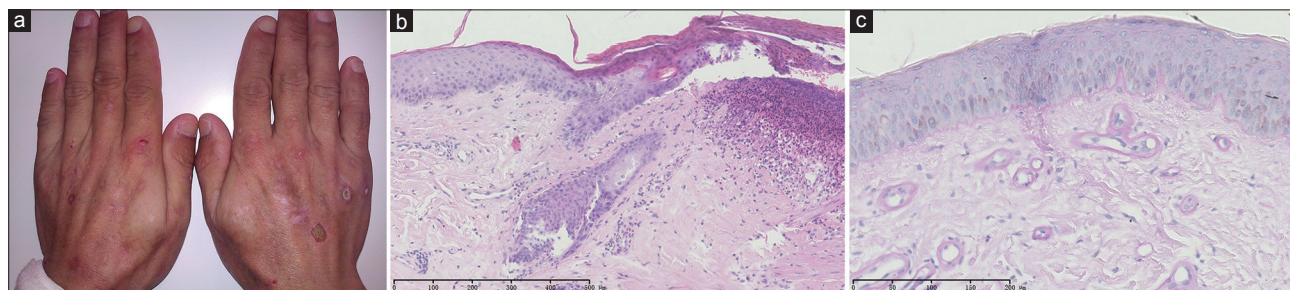
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To the Editor: A 50-year-old man presented with recurrent episodes of bullae, erosions, and crust on sun-exposed areas for 1 year. Lesions healed with scarring and hyperpigmentation. The skin lesions were aggravated after sun exposure. The patient had a 30-year alcohol abuse (100–200 ml/day) history. Family heredity history was unremarkable. Physical examination revealed multiple erosions with erythema, hyperpigmentation, bullae, and crusting on his face, neck, and dorsum of the hands [Figure 1a]. Mild hypertrichosis located over the temple. Dean symptom was positive. The results of blood tests were as follows: aspartate aminotransferase, 91 U/L; alanine aminotransferase, 66 U/L; gamma-glutamyl transferase, 447 U/L; zinc-protoporphyrin levels, 3.3 µg/gHb; uroporphyrin levels were elevated. Viral and autoimmune screening was negative. Urine color was turbid and presented pink fluorescence under Wood's lamp. No gene mutation was observed with uroporphyrinogen decarboxylase (*UROD*) gene analysis. Histopathological manifestation demonstrated crust and eosinophilic depositions around the blood vessels [Figure 1b]. Acid-fast staining showed cyclic blue-violet depositions around the blood vessels [Figure 1c]. A diagnosis of sporadic porphyria cutanea tarda (PCT) was established. After treatments of

compound glycyrrhizin for liver protection, sunscreen, and alcohol discontinuation for 3 months, there was obvious improvement on the patient's skin lesions. Laboratory analysis also showed an improvement; there was a reduction of zinc-protoporphyrin, 1.1 µg/gHb, alanine aminotransferase 46 U/L, and uroporphyrin became negative.

PCT, the most common type of porphyria worldwide, is caused by the deficiency of the enzyme *UROD*, a crucial enzyme in heme biosynthesis, which results in an accumulation of photosensitive byproducts such as uroporphyrinogen which leads to the fragility and blistering of sun-exposed skin. Hypertrichosis, sclerodermoid plaques, and scarring alopecia might also be observed.<sup>[1,2]</sup> The disease has been classified into three subtypes. Type I PCT has decreased hepatic *UROD* activity and is found in sporadic fashion without family history. Type II PCT is an autosomal dominant disorder with genetic mutations of the *UROD* gene causing a decreased *UROD* activity in all tissues. Type III PCT is similar to Type II with respect to familial occurrence, but erythrocyte *UROD* activity is normal.<sup>[1]</sup> The risk factors for PCT include iron overload, alcohol intake, smoking, estrogen use, chemical materials such as



**Figure 1:** (a) Erythema, crusts and hyperpigmentations on the dorsum of hand. (b) Histopathological manifestation revealed crust and eosinophilic dispositions around the blood vessels (H and E, ×100). (c) Acid-fast staining (×200) showed cyclic blue-violet dispositions around the blood vessels.

polychlorinated hydrocarbons, human immunodeficiency virus and hepatitis C infection, mutations in *HFE* gene, and various liver diseases.<sup>[3]</sup> There are several suggested mechanisms of the role of alcohol in PCT. Alcohol increases iron absorption resulting in iron accumulation in the liver, stimulates hepatic  $\delta$ -aminolevulin-

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atesynthase (ALA) synthase and free radical production, and is independently hepatotoxic.<sup>[4]</sup> Similarly, there are materials which are hepatotoxic, such as hexachlorobenzene and dioxin that could also inhibit *UROD* activity and cause disease. Our patient had a history of having continuously ingested alcohol for 30 years and contact with the paint in the week before the onset of illness and he has impaired liver function. Therefore, we suggest that alcohol abuse and contact with the paint may have caused PCT in this patient. The activation of uroporphyrin deposited in the skin by ultraviolet light induces the production of oxidized reactive substances that excessively activate collagenase. The activated collagenase causes deposition of collagen in the skin which induces several skin manifestations that appear in the subepidermal region. In this case, mutations in the *UROD* gene were not detected. Although *UROD* gene mutation is not necessary for a diagnosis of PCT, its presence is important for differentiating between PCT Types I and II. In addition, the case has no family history of PCT. Therefore, this case is classified as PCT Type I. The therapy of liver protection, sunlight avoidance, and discontinuation of alcohol appears to be effective.

This disease is easy to be misdiagnosed. Spotting characteristic clues in the case history, careful examination of the body, and correlation of clinical symptoms with laboratory examination can help keep the misdiagnosis rate low.

## Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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## Conflicts of interest

There are no conflicts of interest.

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