LETTER TO THE EDITOR
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Sporadic Porphyria Cutanea Tarda in a Patient with Multiple Sclerosis Treated with Interferon Beta 1-a Therapy: A Case Report

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Dear Editor,

Multiple sclerosis (MS) is the most common inflammatory disease of the central nervous system, and interferon beta-1a (IFN-β1a) is a valuable medication in the treatment of the relapsing-remitting form of MS (RR MS).

Porphyria cutanea tarda (PCT), which is the most frequent type of porphyria, is the result of a catalytic deficiency of uroporphyrinogen decarboxylase. In sporadic PCT (sPCT), which represents 75% of cases, the deficiency is limited to the liver.[1] Accumulated uroporphyrin and other highly carboxylated porphyrins, which are soluble, diffuse from the plasma into the upper dermis. The clinical picture reflects the phototoxic reaction that subsequently occurs in the upper dermis, in that lesions occur almost exclusively in light-exposed areas.[2] The cutaneous findings include increased photosensitivity, skin fragility, blistering, erosions, and crusts.[1]

In this letter we describe new onset of sPCT in a patient with RR MS who was treated with IFN-β1a. In brief, a 33-year-old man without a family history of PCT was admitted to our department complaining of dizziness and diplopia of 3 weeks duration. Neuroimaging revealed several demyelinating lesions in the subcortical white matter and the periventricular regions. Within 4 months he presented with paresthesia and hyposthenia of the left arm, and repeat MRI revealed additional demyelinating lesions (Fig. 1A). The patient was diagnosed with MS according to McDonald criteria[3] and was started on treatment with IFN-β1a, which was administered subcutaneously three times weekly.

In the course of his treatment, the patient exhibited no relevant neurological alterations; however, after 5 years he observed the development of skin lesions that were localized to the back of his hands, appearing as bullae up to 1 cm wide, and sometimes larger. There were also crusts, atrophic scars, and areas of hypopigmentation (Fig. 1B). He was diagnosed with dermatitis bullosa, for which he was prescribed prednisone at 25 mg/day, which had no effect. His levels of transaminases were elevated as follows: glutamic-oxaloacetic transaminase, 47 U/L [normal value (NV), <38 U/L]; and glutamic-pyruvate transaminase, 138 U/L (NV, <41 U/L). However, testing for hepatitis, including viral markers, antinuclear antibodies, smooth muscle antibodies, and antibodies to liver and kidney microsomes, alcoholic liver disease, and hemochromatosis yielded negative findings. PCT was suspected because of the blistering skin lesions on the back of his hands and other sun-exposed areas of his skin. Laboratory testing for porphyrin levels yielded the following results: total urine porphyrins, 1.838 mg/24 h (NV, <0.150 mg/24 h);[4] total serum porphyrins, 0.069 mg/L (NV, <0.004 mg/L);[5] plasmatic fluorimetric peak, 620 nm; and erythrocyte porphyrins, within the normal range. A skin biopsy procedure was performed on the patient's thigh, and a final diagnosis of PCT was confirmed by a dermatologist.

Repeated phlebotomy resulted in the almost complete disappearance of the skin lesions. The patient suspended the IFN-β1a, and after 3 months commenced glatiramer acetate therapy. Follow-up examinations demonstrated that this medication switch resulted in the patient not experiencing any recurrence of the skin lesions, and porphyrin and transaminase levels within the normal ranges.

Cassiman et al.[6] showed that there is a significant association between sPCT and liver disorders, and described the occurrence of sPCT after medication intake in three patients. However, to our knowledge the present case is the first report

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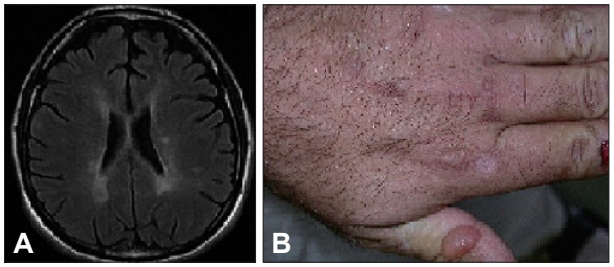

Fig. 1. A: MRI showing several demyelinating lesions. B: Skin lesions on the back of the patient's hands.

of sPCT probably induced by IFN-β1a. We speculate that the possible hepatotoxic effect of IFN-β1a can trigger the development of sPCT in certain predisposed patients. Furthermore, this case highlights the importance of monitoring liver function in all patients treated with IFN-β1a.

Conflicts of Interest

The authors have no financial conflicts of interest.

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