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CORRESPONDENCE

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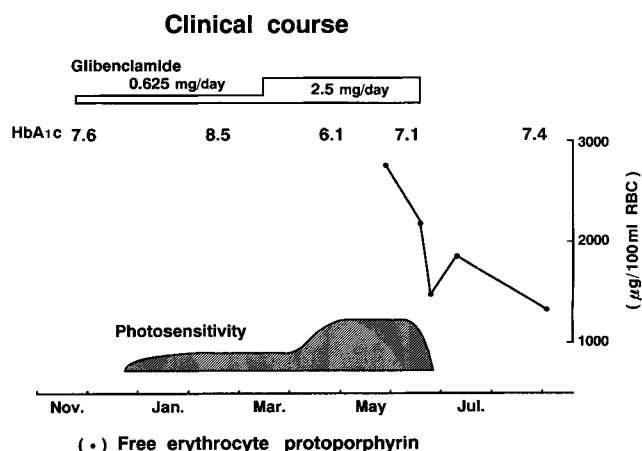


Fig. 1. Clinical course. ●—●, free protoporphyrin concentration in erythrocytes.

### Glibenclamide-Induced Photosensitivity in a Diabetic Patient With Erythropoietic Protoporphyrin

**To the Editor:** In general, the sulfonylureas are safe and effective drugs for use in diabetes mellitus. Erythropoietic protoporphyria (EPP) is the second most common human porphyria. In reviewing study of this topic over the last several decades, this is the first report to implicate oral glibenclamide as the responsible agent for drug-induced aggravation of photosensitivity in EPP.

A 51-year-old man without any previous history of abnormal reaction to sunlight was first seen in our clinic for the complete evaluation of diabetes mellitus in 1989. Since then, he has been followed up with diet alone. On November 20, 1991, he was started on glibenclamide 0.625 mg/day because of poor glycemic control (Fig. 1). In late December, 1991, he gradually began to experience unpleasant sensations of prickling and itching after exposure to the sun. The photoparesthesias involved only the uncovered areas of the body. He was given 2.5 mg glibenclamide daily from March 10, 1992. He then began to notice progressive aggravation of photosensitivity. Erythematous, eczematous lesions occurred within minutes of sun exposure. He was suspected of having porphyrias. On May 25, 1992, protoporphyrin in erythrocytes was 2,689  $\mu\text{g}/\text{dl}$  RBC (normal range 30–86). Neither coproporphyrin nor uroporphyrin in erythrocytes was detected. Coproporphyrin, uroporphyrin, porphobilinogen, and  $\Delta$ -aminolevulinic acid levels in urine specimens were all within normal limits. No anemia was found. Serum iron, total iron binding capacity, and serum ferritin values were normal. Father-to-son transmission was demonstrated. The patient was diagnosed as having EPP.

Since photosensitivity in this patient had never been manifested until the above-described episodes, predisposing factors were suspected. Although sulfonylureas that exacerbate acute porphyrias are not known to worsen EPP [1], they are generally avoided as a precaution [2]. Therefore, glibenclamide was discontinued on June 8, 1992, resulting in a dramatic relief of symptoms within 1 week despite the early summer and the consecutive sunny days. Since then, he has been free from photosensitivity through

midsummer. This case appears to represent a clear example of drug-induced photosensitivity.

Sulfonylureas are known for inducing photosensitivity in diabetic patients. It therefore should be evaluated whether or not photosensitivity by glibenclamide occurred by chance in the patient with EPP. Most patients with chlorpropamide-induced photosensitivity receive a total drug amount of 10–50 g to develop skin lesions, and two cases with glibenclamide received 1.8–3.6 g [3]. In contrast, the total amount of glibenclamide given in our case was only 0.45 g. Moreover, a considerable reduction of free protoporphyrin concentration was observed in erythrocytes after discontinuation of the drug, indicating that photosensitivity was induced through aggravation of protoporphyria by glibenclamide. Glibenclamide should be avoided in this second most common porphyria.

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