

Photosensitivity due to sodium ferrous citrate

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Oral iron therapy for anemia is well-known to aggravate erythropoietic protoporphyria (EPP) (1, 2), while only 1 case of photosensitivity induced by oral iron has been reported (3).

Case Report

A 54-year-old man was seen in December 1994 with erythema and lichenification on sun-exposed areas that had started 4 weeks before. He had no past history of photosensitivity. He had been taking sodium ferrous citrate (Ferromia[®], Eisai Co Ltd, Tokyo) 50 mg t. d. s. orally because of anemia for 6 weeks, and thiamine 20 mg, pyridoxine hydrochloride 100 mg, cyanocobalamin 0.5 mg, pentoxifylline 200 mg, diltiazem hydrochloride 60 mg, nicardipine hydrochloride 20 mg, and alacepril 25 mg, because of sequelae of previous brain infarction and hypertension, for 2 years. There were no abnormal findings on hematological examination, blood chemistry, or urinalysis, including porphyrins of peripheral erythrocytes and urine, serum iron, and serum transferrin.

We performed a screening phototest, as described before (4), with a Dermaray Model M-DMR-1 (Eisai Co. Ltd.) as light source (5, 6). This reflector unit had a bank of 5 Toshiba FL 20S-E-30 fluorescent lamps for UVB on one side and a bank of 10 Toshiba FL 20S-BLB fluorescent lamps plus UV-33 glass filter (Toshiba Glass Co. Ltd., Tokyo) for UVA on the other. Output was measured using a standardized meter, Toshiba UV-radiometer Model UVR-305/365-D (II). The intensity at the skin surface was 1 mW/cm² at 305 nm for S-E-30 lamps and 7.5 mW/cm² at 365 nm for BLB lamps. From the data of 78 normal subjects in our laboratory, the mean $\pm$ S.D. of UVB-MED (minimal erythema dose) is 51.2 $\pm$ 13.2 mJ/cm² and UVA-MED is greater than 13.5 J/cm². UVB-MED for this patient was normal (60 mJ/cm²), while 11.25 J/cm² of UVA-irradiation, which was less than 13.5 J/cm², produced erythema on the irradiated area 24 h after irradiation.

A screening phototest was performed again 2 months after he stopped taking all 8 drugs. Irradiation of 13.5 J/cm² for UVA elicited no response, suggesting the photosensitivity to UVA of the patient had become normal. Patch and photopatch tests were made with sodium ferrous citrate (10, 1, and 0.1% pet.), 7 drugs (10 and 1% pet.), and 6 standard photosensitizers (1 and 0.1% pet.). Only 10% and 1% pet. of sodium ferrous citrate, with UVA-irradiation (4.5 J/cm²) on photopatch testing, showed erythema 24 h after irradiation. Patch and

photopatch tests with sodium ferrous citrate in 5 normal subjects showed no erythema or papular response 48 h after application or 24 h after irradiation.

Discussion

In the terminal step of the heme biosynthetic pathway, ferrochelatase catalyzes the chelation of ferrous iron and protoporphyrin IX, to form heme (7). Partial deficiency of ferrochelatase activity in patients with EPP causes excess amounts of protoporphyrin (7). As described above (1, 2), oral iron produces clinical and biochemical deterioration in some patients with EPP. Phlebotomies can gradually reduce the excess hepatic iron and produce remission in patients with porphyria cutanea tarda (7). Photosensitivity due to oral iron in our case may not relate to porphyrin metabolism, because the patient showed normal levels of porphyrins in blood and urine.

Sodium ferrous citrate, like other forms of oral iron (8), is absorbed from the intestine, bound to plasma transferrin, incorporated into erythroblasts in the bone marrow, and used for synthesis of hemoglobin. Hazano et al. (3) reported a case of photosensitivity due to sodium ferrous citrate and ferrous sulfate. The positive photopatch test in our case suggests that iron or the transferrin-iron complex (diferric transferrin) may be a photosensitizer.

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