appropriate (1). Isolated cases of successful treatment have been reported: local injection of amphotericin B (3), miconazole (7), oral ketoconazole (8, 9), oral itraconazole (2) and oral fluconazole (2). Because of the small number of cases, no controlled trial has been conducted. Itraconazole and ketoconazole seem to give the best results (1, 2, 9).

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Very Low-dose Chloroquine Treatment for Porphyria Cutanea Tarda

Sir,

A 74-year-old woman came under our observation in September 1993, with a 2-year history of recurrent vesicles and bullae occurring mainly in areas of repeated traumas and exacerbating by sunlight exposure. Her past medical history was negative for alcohol or drug intake.

Physical examination revealed some vesicle-bullous lesions on her hands and dorsum of the feet, where she also had some atrophic scars. Histopathology of a lesion showed a subepidermal bulla with a festooned base, consistent with the diagnosis of porphyria cutanea tarda (PCT). Diagnosis was confirmed by the blood tests, iron 158 mg/dl (normal values 35–160), transaminases ALT 78 U/I and AST 97 U/I (n.v. 0–40), gamma-glutamyltransferases 50 U/I (n.v. 10–50), and by the urine porphyrin content: total porphyrins 3,470 g/24 h (n.v. 50–200), uroporphyrins 1,769 g/24 h (n.v. 15–50) and coproporphyrins 1,710 g/24 h (n.v. 35–150).

In October, chloroquine treatment (0.5 g twice weekly) was started. After the first two doses, the patient had an acute reaction consisting of fever (39°C), malaise, nausea, vomiting, anorexia, abdominal pain, constipation and arthralgia, persisting for 5 days. This symptom complex was associated with increased serum levels of transaminases (ALT 94 U/I, AST 126 U/I) and with a massive increase in urinary porphyrin output (total porphyrins 3,750 g/24 h, uroporphyrins 2,400 g/24 h, coproporphyrins 1,920 g/24 h).

One month later, very low doses of chloroquine (62.5 mg/weekly) were resumed. Apparently, the patient tolerated them well and showed a rapid biochemical and clinical improvement. During the following months, daily urinary porphyrin excretion slowly declined, attaining values near normal in July 1994 (total porphyrins 121 g/24 h, uroporphyrins 96 g/24 h, coproporphyrins 25 g/24 h). The levels of serum transaminases and gamma-glutamyltransferases became normal, skin lesions healed and the patient’s general condition ameliorated after only a few months’ treatment. No new lesions were observed and periodic ophthalmological examinations revealed no evidence of retinopathy.

Phlebotomy and antimalarials are considered the mainstay of therapy for PCT (1). Phlebotomy, however, may be contraindicated in patients with anemia, cardiopulmonary disease or HIV infection. In addition, antimalarials may be more effective than phlebotomy in the treatment of PCT (2). Chloroquine therapy needs caution, however, as it may cause acute reactions or
produce hepatotoxicity, especially when high doses are used. Moreover, antimalarials cannot be given for a long period of time because of their ocular toxicity especially in older patients. For this reason, low doses of chloroquine (125 mg twice weekly) have been recommended (3). In our patient, even 62.5 mg weekly proved enough to obtain a clinical remission. To the best of our knowledge, so low a dosage has never been reported previously.

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Could Bacterial Acetaldehyde Production Explain the Deleterious Effect of Alcohol on Skin Diseases?

Sir,

Heavy alcohol consumption is known to be associated with the aggravation of certain skin diseases, like seborrheic and nummular dermatitis, psoriasis, acne and rosacea (1, 2). In contrast to the classic alcohol-accumulated stigmata skin may be afflicted as an early feature of alcohol misuse (1). So far the pathogenesis of these phenomena is not fully elucidated.

A number of bacteria and yeasts are known to possess alcohol dehydrogenase (ADH) activity (3). In the presence of excess ethanol these microbes produce reactive and toxic acetaldehyde (4, 5). Since ethanol concentrations of sweat are equal to those in blood (6), we wanted to study whether bacteria and fungi associated with pathological dermatological conditions contain ADH and produce acetaldehyde from ethanol.

Propionibacterium acnes, Staphylococcus aureus, Staphylococcus epidermidis, Streptococcus pyogenes and Pityrosporum ovale strains were isolated from outpatients at the Department of Dermatology, Helsinki University Central Hospital. The bacteria were identified with usual microbiological criteria. In addition, the identification was verified by analyzing the cell components gas chromatographically (MIDJ, Sherlock, Barksdale, Newark, DE).

Cytosolic ADH activity was determined spectrophotometrically by measuring, after addition of ethanol, the reduction of nicotine amide dinucleotide (NAD) to is reduced form (NADH) (5). Bacterial acetaldehyde production was determined by incubating bacterial suspensions in closed vials with 1% ethanol at pH 7.4, 37°C for 2 h. After the incubation, the acetaldehyde produced was determined using head space gas chromatography as described earlier (7).

Very high acetaldehyde levels up to 960 μmol/l were formed by the bacteria studied at ethanol concentrations known to exist in sweat during normal social drinking. The corresponding bacterial cytosols showed significant ADH activity both at low and high ethanol concentrations. The amount of acetaldehyde related to the number of bacteria (mean ± SE) after at least four separate determinations was: P. acnes (59 ± 23 nmol/10⁶), S. pyogenes (118 ± 15 nmol/10⁶), S. epidermidis (106 ± 10 nmol/10⁶) and S. aureus (160 ± 22 nmol/10⁶). The P. ovale fungi associated with seborrheic dermatitis, however, neither produced acetaldehyde (0.8 ± 0.2 nmol/10⁶) nor showed ADH activity. This primary observation of bacterial production of acetaldehyde - a highly toxic and reactive substance - could offer an explanation for the deleterious effect of alcohol on various skin diseases and warrants for further in vivo studies.

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