

had not occurred in any of these patients. Bacterial and viral infections are well-known trigger factors for psoriasis (7). HIV infection is associated with psoriasis, with an overall prevalence of 5–6% (8). This association appears paradoxical, because psoriatic inflammation is mediated by activated T cells and neutrophil chemotaxis. In psoriatic lesions, epidermotropism and disease-associated changes in the T-cell receptor repertoire have been detected in CD8 lymphocytes (9). The activation and proliferation of CD8 lymphocytes may therefore facilitate the development of psoriasis in HIV-infected individuals. This hypothesis is supported by the observation of improvement of psoriasis in patients after initiating HAART (10, 11).

In our patient, pyoderma gangrenosum and psoriasis rapidly improved within 6 weeks after initiating HAART. Cutaneous dendritic cells play a central role in the control of cutaneous immune reactions. Langerhans' cells express CD4 molecules, HIV co-receptors and various cytokines (12, 13). Taking this into consideration, one might speculate that the improvement of both skin diseases was caused by restitution of (cutaneous) immune system under HAART.

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Pellagra-like Skin Lesions Associated with Wernicke's Encephalopathy in a Heavy Wine Drinker

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Sir,

The endemic form of pellagra associated with poverty and inadequate diet is manifested clinically by the "three Ds" diarrhea, dermatitis and dementia, and occasionally by death (1). Pellagra results from a tissue deficiency in niacin or in its precursor, the essential amino acid tryptophan (2). Sporadic cases are due to chronic alcoholism, dietary lack of natural sources of niacin (liver, fish, lean meat, poultry, yeast and cereal grains), malabsorption, certain chemotherapeutic drugs, or carbamazepin (1). Wernicke's encephalopathy is due to thiamin deficiency and is usually seen in alcoholics who suffer from malnutrition. We present here a case with a suspected combination of these two deficiencies.

CASE REPORT

A 40-year-old woman was admitted to the Neurological Department early in August with rapidly evolving ataxia

of gait and diplopia. She had had 3 attacks of dizziness during the previous 6 months and a sun-induced rash on her lower legs that during the previous 3 weeks had spread to her arms, hands and head. Neurologically, she displayed atactic gait, horizontal nystagmus and myoclonic jerk. Laboratory tests showed pathological liver functioning. Serology for hepatitis B and C and HIV was negative. Blood sugar and zinc values were normal. Ultrasound revealed liver steatosis. She reported drinking considerable amounts of wine in the previous few weeks. Since Wernicke's encephalopathy was suspected, vitamin B was injected i.m. (thiamine chloride, pyridoxine chloride and cyanocobalamin, 0.1 g daily each). After about 24 h the patient's neurological symptoms disappeared. Two days after admittance to the hospital she was referred to the Dermatological Department for her rash. She displayed redness, superficial scaling and blisters on her lower legs, whereas her

heels and the distal part of her toes were uninvolved (Fig. 1). There was a symmetrical, clearly marginated dusky eruption on her arms (Fig. 2), the dorsal parts of her hands, and around her neck. A skin biopsy was taken from the upper arm and showed orthokeratosis, necrotic keratinocytes, intraepidermal blistering and multiple melanin granules throughout the epidermis. Suspected of having pellagra, she was treated topically



Fig. 1. Pellagra eruption with redness and blisters on the lower legs, forming a boot, and sparing the distal part of the toes. Note the symmetry and clear line of demarcation from normal skin.

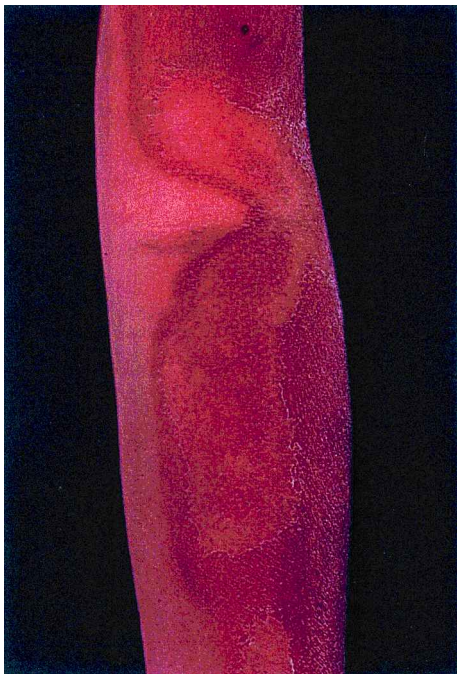


Fig. 2. Well-demarcated dusky redness on the patient's arm.

with betamethason and a peroral multivitamin preparation containing niacin. The rash improved within a week. The patient was then discharged with continuing peroral multivitamin treatment. Three weeks later she reported that only local hyperpigmentation remained.

DISCUSSION

The diagnosis of pellagra is clinical but can be proved by analysis of niacin levels. Such an analysis was not performed in our patient. However, the rash disappeared after oral supplementation of a multi-vitamin preparation including niacin. Niacin monotherapy would have been more conclusive but this kind of preparation is not available commercially in Sweden. In addition, patients with alcohol abuse often suffer from deficiency of several vitamins – as illustrated in this case – which should be supplemented.

The histological results are suggestive of pellagra, though not diagnostic of it, and resemble those of the glucagonoma syndrome somewhat. The clinical lesions likewise resemble those of glucagonoma. The latter, however, are usually found on the trunk, proximal extremities and perineum, whereas pellagra appears on skin exposed to the sun. Laboratory tests excluded skin eruptions being due to hepatitis C (3) or zinc deficiency.

Although our patient's dermatitis was suggestive of pellagra, none of the other "Ds" were found. Spivak & Jackson (4) reported only two "full triad pellagra" cases, all with dermatitis, among their 18 alcoholic patients. Wernicke's encephalopathy, induced by thiamine deficiency, classically involves the triad – though seldom observed – of ophthalmoplegia, ataxia and altered mentation (5). Since all the neurological symptoms of our patient disappeared quickly after administration of thiamine but prior to that of niacin, Wernicke's encephalopathy was obvious, despite the patient's showing the myoclonic jerking common in pellagra encephalopathy (6). Serdaru et al. (6) found 21 of their 22 alcoholic pellagra patients to be heavy wine drinkers. Our patient illustrates non-endemic pellagra as a valid differential diagnosis, especially in wine drinkers, and that it sometimes occurs, as it often does in Wernicke's encephalopathy, without the classic triad.

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Eccrine Naevus: Case Report and Literature Review

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Sir,

Eccrine naevus is a very rare entity with no more than 20 cases reported in the literature. Various clinical manifestations have been described as a localized hyperhidrotic area without epidermal changes or with a slight hyperpigmentation, papules in a linear distribution, depressed brownish patches, centrally depressed nodules surrounded by a slightly scaly border, a solitary sweat-discharging pore type and asymptomatic papular lesions without sweat increase.

We report a new case of eccrine naevus, which appeared as a slight hyperpigmented and hyperhidrotic plaque. Therapy with botulinum toxin was effective. To the best of our knowledge, this is the first patient with eccrine naevus treated in this way.

CASE REPORT

A 63-year-old man presented with a local area of excessive sweating on his lower back for 3 years. Although sweating occurred throughout the day, it was aggravated by exercise, increased environmental temperature, and stress. The patient had a history of toxic oil syndrome and general pains for which he was taking diclofenac, daily.

The affected area was well defined, around 12 × 5 cm in size, and had slight hyperpigmentation without epidermal alterations. We noted an increased amount of sweating compared to normal skin (Fig. 1).

General physical examination was completely normal and did not reveal other cutaneous lesions. Results of neurological examinations were normal, as were the results of blood investigations and vertebral column radiograph. The starch-iodine test demonstrated a sharp delineation on the lower back.

A biopsy of the hyperhidrotic area revealed marked ductal hyperplasia and dilated coils without epidermal changes (Fig. 2).

Treatment with 20% aluminium chloride hexahydrate

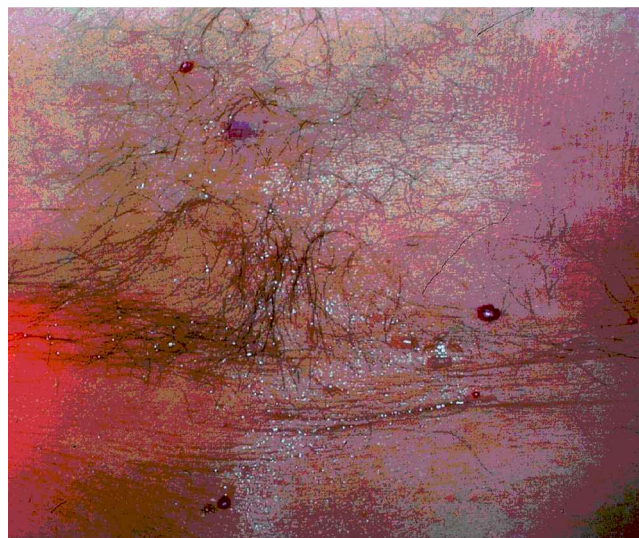


Fig. 1. Hyperhidrotic area on the lower back.

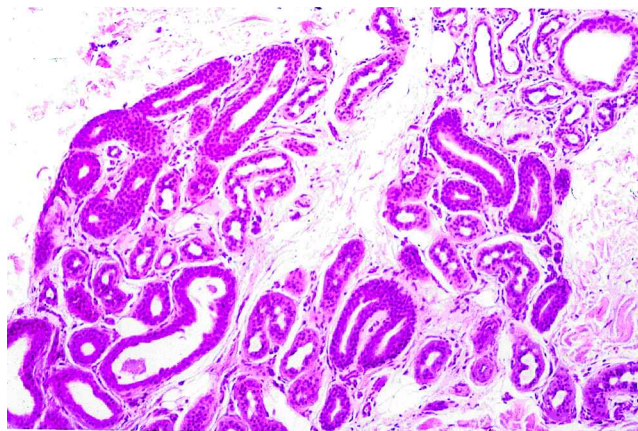


Fig. 2. Skin from lower back showing increased number of eccrine sweat glands and dilated coils of normal appearance.

in absolute alcohol was not effective. We began treatment with botulinum toxin. Botulinum toxin type A (Botox[®], Allergan, USA) was diluted in 5 ml of 0.9%