Acquired Acromelanosis due to Phenytoin

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Key Words

Acromelanosis, acquired
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A 19-year-old girl presented with complaints of asymptomatic progressive darkening of fingers and toes of 3 years' duration. She was a known patient with grand mal epilepsy and had been on a combination therapy of phenobarbitone 60 mg b.d. and phenytoin 100 mg b.d. for 5 years. Phenobarbitone was however stopped after 1 year and phenytoin was continued for the next 2 years. At the completion of 2 years of therapy, she noticed gradual and progressive darkening of all fingers and toes. The pigmentation was diffuse, greyish in colour, extending up to the dorsum of the proximal phalanges, and it was more prominent over the dorsum of the interphalangeal joints and distal phalanges (fig. 1). As the patient did not have any seizures, the anti-convulsant was stopped at the end of 3 years. This was followed by gradual but incomplete regression of pigmentation without any treatment. After 1 year of discontinuation of therapy, seizures recurred and she was again put on phenytoin at the same dose, i.e. 100 mg b.d. Pigmentation re-appeared within 3 months of therapy. No history of any other drug intake was available and no other member of the family had a similar pigmentation. The nails were normal and showed no pigmentation. Examination of mucosae, hair and other parts of the skin revealed no hyperpigmentation. No other side effects of phenytoin were observed. She was advised to discontinue phenytoin, and alternative treatment with carbamazepine 100 mg t.i.d was started. In 2 months of follow-up, pigmentation was noted to be regressing without any specific treatment.

Discussion

Fig. 1. Diffuse pigmentation over the dorsum of fingers.

The acromelanosis in the present patient was considered to be acquired due to phenytoin therapy.

Although she had received pheno-

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barbitone initially, pigmentation appeared after about 1 year of discontinuation of phenobarbitone. There was a significant temporal correlation between discontinuation of phenytoin and regression of pigmentation with subsequent re-appearance of pigmentation with institution of phenytoin. To the best of our knowledge, such an acropigmentation accredited to phenytoin has not been documented [1,2].

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Gloves-and-Socks Syndrome in a Patient with Epstein-Barr Virus Infection

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Key Words

Gloves-and-socks syndrome · Epstein-Barr virus

A 48-year-old psoriatic woman presented with a 20-day history of low-grade fever, malaise and rash primarily involving the hands and feet. She had been receiving systemic corticosteroids and etretinate for her psoriasis, but 3 weeks before the onset of the illness all drugs had been stopped. On examination, her hands and feet were erythematous and edematous with sparse papular and petechial lesions in a glove-and-sock distribution. Some petechiae and papuloerythematous lesions were also scattered over the inner part of the thighs and legs. The lesions were preceded by burning sensations and paresthesiae at the extremities. Lymph nodes, liver and spleen were not palpable.

Laboratory investigations revealed mild lymphopenia of 23.1% ($1.37 \times 10^9/l$) with $5.9 \times 10^9/l$ WBC (neutrophils 56.7%, eosinophils 11.7%, monocytes 6%, basophils 2%). No atypical mononuclear cells were present. Liver function tests were within normal limits. Antinuclear antibody and rheumatoid arthritis tests were negative.

Serology for measles, rubella, parvovirus B19, coxsackievirus, cytomegalovirus, herpes simplex virus, hepatitis B and C virus were normal or negative. Serology (ELISA) for Epstein-Barr virus (EBV) disclosed antiviral capsid antigen (VCA) IgG (+++) and IgM (+++), and anti-EBV

nuclear antigen (EBNA) IgG (++) . Anti-early-antigen was not studied. A monospot test was negative.

Fever and malaise disappeared within a few days without treatment. Skin lesions gradually cleared with mild desquamation.

Two months later, indirect immunofluorescence revealed anti-VCA IgG 1/2,048, anti-VCA IgM negative, anti-early-antigen IgG 1/16 and anti-EBNA IgG 1/32. After 6 months, the patient was in good condition and free from lesions. Serology showed anti-VCA IgG 1/512, anti-EBNA IgG 1/32. Anti-early-antigen and anti-VCA IgM were negative.

Our case is an example of the 'gloves-and-socks syndrome', which is a rare but well-defined disorder first described in 1990 by Harms et al. [1]. Prodromic symptoms may be fatigue, anorexia and fever. Itching symmetrical erythema and edema of the hands and feet ensue followed by petechial purpura. Although drugs have been incriminated, gloves-and-socks syndrome is customarily ascribed to a viral etiology, in particular to parvovirus B19 [2–4]. Our patient is the first case associated with EBV infection. Both a primary infection and an endogenous reactivation may be considered.

Usually in adulthood, EBV primary infection has a severe course with abrupt onset, high fever, lymphadenopathy and hepatospleno-megaly, and laboratory examination reveals leukocytosis and lympho-cytosis with atypical mononuclear cells and a positive monospot test. Those features were absent in our patient. An EBV endogenous reactivation is therefore the most likely possibility even in the presence of anti-EBNA antibodies that, in a reactivation state, are usually absent or present at low titers.

Skin lesions have rarely been reported in EBV infections and rarely do they represent the prominent clinical feature of the disease [5]. In our case, they may be considered the presenting sign of a reactivated EBV infection.

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Proliferating Tricholemmal Cyst Should Always Be Considered as a Low-Grade Carcinoma
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Key Words

Proliferating tricholemmal cyst · Malignancy · Carcinoma

Proliferating tricholemmal cyst (PTC) is an uncommon appendageal skin neoplasm undergoing outer root sheath differentiation, showing a compact, abrupt tricholemmal keratinization without granular layer interposition. PTC shares this kind of keratinization with the tricholemmal cyst,

with the outer root sheath at the level of the isthmus where the inner root sheath disappears, and with an infundibular sac in catagen and telogen hairs [1]. PTC may also present matrical structures with a pattern similar to that detectable in squamous carcinomas with pilar differentiation [2, 3]. Clinically, PTC usually appears as a

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