

nocarcinoma to the leg, as occurred in the case described here. The dermatomal distribution of the lesions accompanied by neuralgia and hyperaesthesia led the physician to conclude that the patient had herpes zoster.

This rare type of metastases has been reported in several cases to date (2, 3). The primary tumours with zosteriform metastases were located in the breast, carcinoma of the lung, carcinoma of the bladder, carcinoma of the renal pelvis, and carcinoma of the ovary. The frequent histopathological type of this metastasis is adenocarcinoma, and rarely transitional cell carcinoma. To our knowledge, this seems the first case of zosteriform cutaneous metastasis from a colonic adenocarcinoma. The vesicular appearance in our case may have been due to mucin production by adenocarcinoma. Some authors explain this phenomenon as being caused by lymphoedema (2) and other authors by epidermotropic metastasis (3). The tumour cells may have been disseminated on the left lower extremity by lymphatic spread in the case described, because direct lymphatic infiltration of the tumour cells was not proven histologically and an obstruction in the left inguinal and para-aortic lymph nodes had been shown in a CT scan.

Easy and prompt diagnosis of cutaneous metastases can be made because skin lesions often mimic the primary tumour cytologically. In this case, tumour cells occurring in cohesive groupings or in a papillary structure were observed and the immunostaining studies displayed tumour cell reactivity for CEA, favouring intestinal adenocarcinoma as the primary source in a Tzanck smear. Unlike non-cutaneous metastases, cutaneous lesions can be readily seen with careful visual exam-

ination (4). Manteaux et al. described a patient who developed epidermotropic metastases (3). A Tzanck smear showed large atypical cells which had been misinterpreted as herpetic balloon cells. Skin biopsy revealed dis cohesive malignant cells within the subepidermal and intraepidermal vesicles.

Smear cytodiagnosis is a rapid, accurate means of establishing a diagnosis of metastatic disease, especially in cases in which the lesions localize in the epidermis or subepidermis, and application of immunoperoxidase may qualify the cytological diagnosis and location of the primary tumour.

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Accepted July 13, 1998.

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Erythema Elevatum Diutinum in a Patient with Human Herpesvirus 6 Infection

Sir,

Several cases of erythema elevatum diutinum (EED) have recently been found to be associated with human immunodeficiency virus (HIV) infection (1–3), supporting the hypothesis that an infectious agent plays a role in its aetiology (4). We describe here an HIV-negative patient with evidence of human herpesvirus 6 (HHV-6) infection who developed EED.

CASE REPORT

In September 1996 a 50-year-old white woman in apparently good health developed a papulo-nodular eruption on the limbs. On examination, she exhibited a symmetrical, diffuse papulo-nodular eruption on the outer surface of thighs (Fig. 1), buttocks and finger joints, and some plaques over the knees and elbows. The ear lobes and face were also involved (Fig. 2). The lesions were indurated, circinate or oval with central atrophy, purplish to reddish-brown in colour. Neither arthralgia nor other subjective complaints were noted.

A biopsy specimen showed a dense perivascular leucocytoclastic infiltrate in the papillary and reticular dermis consistent with EED. Direct immunofluorescence was negative. Laboratory tests including blood cell count, renal and liver functions were within normal ranges. There was no evidence of streptococcal infection. Chest X-rays and abdominal ecography were also normal. Paraproteinaemia and cryoglobulins were absent and tumoural markers negative.

Immunological investigations showed only a faint ANA positivity

on HEP2 cells (1/40 speckled IgM and IgG). Anti-HHV-6 were positive (IgM 1/80 and IgG 1/20), while antibodies against other viruses were negative or indicative of immunity.

When first examined, the patient had been taking 30 mg deflazacort daily for 1 month. The lesions had worsened, however, and the treatment was stopped. In the absence of any treatment, the lesions began to improve and 1 month later the anti-HHV-6 IgM titre was 1/20 and anti-HHV-6 IgG 1/80.

Treatment with 100 mg dapsone daily was started. One month later, the skin lesions had clearly improved and the anti-HHV-6 IgG titer decreased to 1/40. IgM were still 1/20. The drug was reduced to 50 mg daily and, 1 month later, discontinued. The disease did not relapse.

DISCUSSION

EED is a rare chronic form of cutaneous vasculitis of unknown aetiology that is considered an immune-complex mediated reaction. The disease may be associated with streptococcal infections, inflammatory bowel disease, haematological disorders (4) and, less frequently, with other pathologies (4, 5) including HIV infection (1–3).

In the patient described here, an infectious cause may be suggested by the exacerbation of her lesions with the steroidal therapy and by their prompt amelioration with the corticosteroid discontinuation (6).

In addition, this patient disclosed specific anti-HHV-6 IgM



Fig. 1. Papulo-nodular lesions on the thighs.

and an increase in the titre of anti-HHV-6 IgG. These serological findings may be interpreted as a seroconversion during a primary HHV-6 infection or as HHV-6 endogenous reactivation (7).

HHV-6 infection usually occurs during infancy and persists in a lifelong latent state (8). Primary infection may cause exanthem subitum in children (9), but frequently is inapparent or results in febrile illness without any rash. In adults, the primary infection is very rare, consisting of a mononucleosis-like illness, prolonged lymphadenopathy or hepatitis (10).

HHV-6 IgM antibodies can be detected both in primary infections and in reactivation states. Anti-HHV-6 IgG antibodies are present in 80–90% of adults and tend to disappear over time (8). An increase in the titres of IgG to HHV-6, therefore, is considered indicative of reactivation. In the absence of other viral cross-reactivities, such as EBV or CMV active infections, the specific anti-HHV-6 IgM and the increased titres of IgG make an HHV-6 endogenous reactivation most likely. The rarity of the primary infection in adults and its usual severe course are additional evidences.

Whether EED cutaneous lesions might be determined directly by the HHV-6 reactivation state or may be a mere coincidence, cannot be established by a single case.

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Fig. 2. Nodular lesions involving the ear lobes.

Accepted July 15, 1998.

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