During the following months the patient developed a gradually increasing, purple infiltrate on the dorsal side of the right hand extending to the dorsal and proximal part of the 2nd and 3rd finger. In April 1979 excision biopsy was made at the orthopedic department. The histopathological changes were interpreted as the cutaneous manifestation of a malignant lymphoma or chronic lymphatic leukaemia, and the patient was referred to the haematological department. Examination showed no signs of disseminated malignant lymphoma, leukaemia, tuberculosis or syphilis. Radiation treatment with two series of 10 GY each failed to reduce the size of the tumour and the patient was referred to the dermatological department.

Dermatological examination now showed a massive, purple swelling of the dorsal part of the right hand extending to the dorsum of the 1st, 2nd and 3rd finger.

Revision of histopathology

In the excised tissue taken at the operations a massive, diffuse lymphocytic infiltrate in the dermis and subcutis was present. The lymphocytes were well differentiated and not arranged in follicular structures. Mitotic figures were not present. Peripheral nerves present in the tissue were not infiltrated by the lymphocytes. In some areas a marked proliferation of capillary vessels was seen intermixed with the massive infiltrate. Neither epidermis nor appendages were present in the tissue specimens.

Treatment

Fenozymethylpenicillin (Vepicombin®) 500 000 IU orally four times a day for 2 weeks reduced the size of the tumour after a few days' treatment. After termination of treatment only atrophy remained on the affected sites of the right hand, resembling the senile atrophy of the dorsal part of the left hand.

DISCUSSION

The two cases presented here were both clinically and histopathologically lymphocytoma cutis, but the difficulties in differentiating this condition from a malignant lymphoma are illustrated by the fact that the lymphocytic infiltration was initially stated to be malignant. In both cases trauma seemed to be the trigger factor, even if insect bites cannot be excluded with certainty.

In our cases an attempt was made to classify the lymphocytic infiltrate according to From (5). In case 2 no epidermal structures were present in the tissue specimens and a classification can thus only be made with some uncertainty, but type 5 (dense non-follicular lymphocytic infiltrate) seems the most likely. In case 1 the infiltrate was classified as a clear-cut type 5.

Lymphocytoma cutis is not a manifestation of any systemic disorder and the prognosis is judged to be favourable, as stated by several authors (3, 4, 6) who found that all patients with lymphocytoma cutis had a benign course ending in cure, either as a result of treatment or as a spontaneous remission.

Bianchi (2) and others (4, 6) found that some cases of lymphocytoma cutis could be cured with penicillin, which our two cases demonstrate in a convincing way.

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Treatment of Kaposi's Varicelliform Eruption with Adenine Arabinoside (Vira-A) and Transfer Factor

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Received March 28, 1981

Abstract. Kaposi's varicelliform eruptions, or Eczema herpeticum, are the result of a herpes virus infection. This paper reports on a patient who was treated with parenteral infusion of a new anti-viral drug, adenine arabinoside (Vira-A), and transfer factor in an attempt to halt the spread and alleviate the severity of the disease. It is our

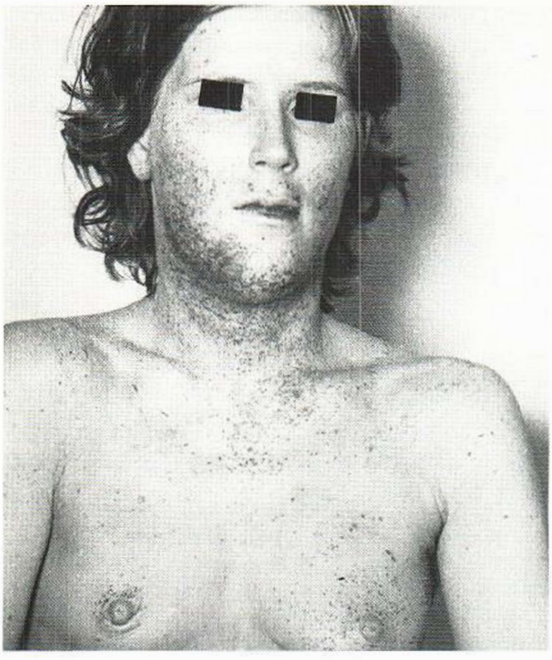

Fig. 1. The patient 2 days after the onset of KVE, immediately before anti-viral treatment was started.

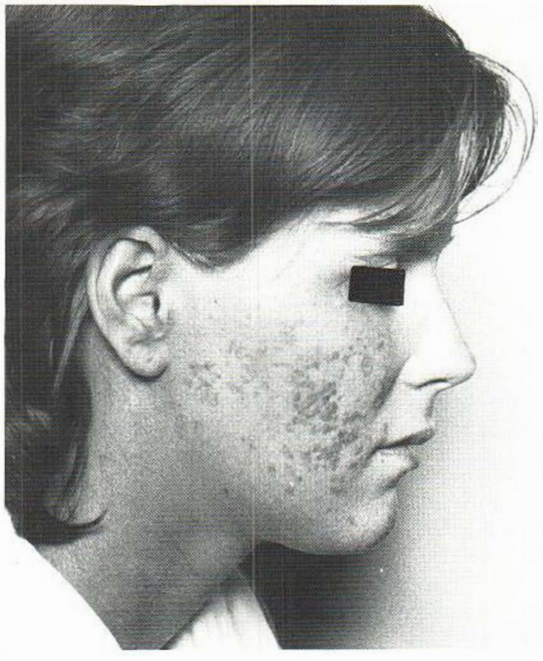

Fig. 2. The patient 10 days after anti-viral treatment was started, i.e. 10 days after the photograph shown in Fig. 1 and 12 days after the beginning of KVE.

opinion that adenine arabinoside can have a beneficial effect on the course of the disease.

Key words: Kaposi's varicelliform eruptions; Eczema herpeticum; Atopic eczema; Adenine arabinoside; Anti-viral agents; Transfer factor

Kaposi's varicelliform eruption (KVE) is a cutaneous infection caused by Herpes hominis virus or vaccinia virus. The disease occurs in patients suffering from pre-existing cutaneous disorders such as atopic eczema, Mb. Darier and other ichthyosiform dermatoses.

The outcome of KVE may be fatal in immunosuppressed patients. Hitherto, therapy has been restricted to dealing with secondary bacterial infections. However, the introduction of a new anti-viral drug, adenosine arabinoside (Vira-A), may be useful in preventing the most serious outcome of the disease. The present case report is the first documented use of adenosine arabinoside in a patient with KVE.

CASE REPORT

A 24-year-old woman was admitted with a severe exacerbation of her life-long atopic dermatitis. Two days before admission she had noticed a few vesicles around her left areola mammae. Within the first 3 days in the department, there was a rapid spreading of umbilicated vesicles involving the face, neck and upper part of the chest. Fever (38.5°C) and malaise occurred (Fig. 1).

A clinical diagnosis of KVE was established. A lumbar puncture was performed as the patient had developed psychological changes. Spinal fluid investigations proved normal. However, in order to ensure an uneventful recovery, the patient was given adenine arabinoside 15 mg/kg/day intravenously for 4 days, together with transfer factor, 1 unit, prepared from buffy-coat leukocytes from healthy blood donors (3).

After 2 days of treatment the fever and psychological symptoms disappeared. The vesicles in the skin turned into small crusted excoriations (Fig. 2). A secondary bacterial infection with *Staph. aureus* was treated with erythromycin, 1 g per day, for 10 days. The only local treatment was wet dressings and, subsequently local steroids for her eczema.

Adenine arabinoside (Vira-A)

Adenine arabinoside is a purine nucleotide obtained from supernatants after fermentation with *Streptomyces antibioticus*. It has a molecular weight of 285.2. It inhibits the multiplication of herpes virus through inhibition of virus DNA polymerase activity (2).

Its therapeutic toxic ratio is superior to the other purine and pyrimidine analogues such as idoxuridine and cytarabine (1). In the body, adenine arabinoside is deaminated to adenine arabinosylhypoxanthine, which is the

principal metabolite. Excretion is predominantly via the kidney where 50% of a daily dose is recovered as arabinosylhypoxanthine and 2% as adenine arabinoside. It penetrates the blood-brain barrier to give a CSF/plasma ratio of approximately 1 : 3 (1).

Adenine arabinoside must be given intravenously in isotonic glucose, which must be buffered beforehand with sodium phosphate to increase the pH of the solution to more than 4.5. The drug has a low solubility and the solution must therefore be kept in a 37°C water bath until it is clear, usually for one hour. It is stable at room temperature for at least 24 hours. To avoid non-solubilized microcrystals of the drug to enter the blood a 0.3 μm filter is mounted at the infusion-set.

We observed no side effects from the treatment.

Laboratory findings

Standard laboratory investigations gave normal results. Immunological studies showed a herpes virus titre of 120 (5th day of disease) which increased to 480 (15th day of disease). The total amount of IgE was increased (more than 1 000 IU/ml). The percentage of T lymphocytes in blood was within normal limits and the *in vitro* reactivity of lymphocytes following stimulation with various mitogens (PHA, Con A, PWM, PPD) was normal. The patient's serum did not contain factors which could suppress the *in vitro* reactivity of normal lymphocytes.

DISCUSSION

Until now the only possible treatment for KVE has been supportive, with management of secondary bacterial infection and maintenance of fluid and electrolyte balance. Several new anti-viral drugs may be of beneficial use in KVE in order to shorten this often severe and distressful disease and to prevent a fatal outcome. Adenine arabinoside has been effective in herpes simplex encephalitis (4). In herpes encephalitis it is of the greatest importance that treatment is started as early as possible (4).

Our patient experienced an uneventful recovery from her KVE. Fever, malaise and psychological disturbances, which were not due to encephalitis, but regarded as symptoms of a reactive psychosis, subsided within 2 days of initiating the therapy. However, her skin lesions of KVE persisted for 14 days (see figures).

In order to evaluate the possible effects of the treatment, we have retrospectively recorded the duration of disease in 8 patients with KVE, who were admitted to our department. We found a mean age of 24 years. The period from the first recorded vesicular eruption until clearing of the skin was 14 days (range 10–19 days). Thus, we have no clear indication that the specific anti-viral treatment shortened the duration of disease in our patient. It is impossible to say whether her fairly rapid general improvement could have been attributable to adenine arabinoside and transfer factor.

However, we would like to draw the attention of dermatologists to the possible benefits of specific anti-viral treatment in patients suffering from severe KVE. The drug must be given as early as possible, maybe even on mere suspicion of KVE.

ACKNOWLEDGEMENT

We wish to thank Mrs Margareta Sommer for her skilful secretarial assistance.

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Ketoconazole in a Case of Chronic Mucocutaneous Candidiasis

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Received April 10, 1981

Abstract. An 8-year-old girl with chronic mucocutaneous candidiasis was treated with Ketoconazole 100 mg daily for 6 weeks. Oral thrush disappeared after a week, while cutaneous lesions cleared within a month. Her nail dystrophy disappeared during the following months. Repeated cultures were negative for *Candida albicans*. The patient has remained in remission for an 8-month period. Our findings thus support previous reports indicating that Ketoconazole is an effective agent against this disease.

Key words: Candida albicans; Chronic mucocutaneous candidiasis; Ketoconazole