Transfer Factor in Mycosis Fungoides: A Case Report on a Patient "Cured"

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Abstract. A report is given of the successful treatment with transfer factor as an additional therapeutic agent of a patient in the tumour stage of mycosis fungoides who had not responded to conventional treatment with nitrogen mustard, methotrexate, or grenz rays alone. Besides twelve injections of transfer factor, the patient received X-irradiation, 1400 r (50 kV 1 mm Al) in all, to three tumours. The combined treatment induced sustained remissions. When that patient died from a coronary infarct 5 years after treatment began, autopsy showed no evidence of mycosis fungoides at all.

Key words: Transfer factor; Mycosis Fungoides

Mycosis fungoides (MF) is a lymphoma arising within the skin, remaining confined to the skin as lesions progress, become infiltrated and enlarge into tumours. After reaching the tumour stage, the disease usually rather quickly involves the peripheral lymph nodes and in further rapid sequence disseminates to visceral lymph nodes and internal organs (7). The period of progression of skin symptoms is variable, often years, while until recently the usual interval between tumour stage and death has been reckoned to be approximately 2 years (1, 2, 3).

Treatment with topical nitrogen mustard (7), electron beam (4) and PUVA (5) seems to have improved the prognosis and often can lead to more or less sustained partial or even complete remissions. Cure as such, however, has been difficult to prove. Lately, transfer factor (TF), a low molecular weight extract from human leukocytes with ability to enhance cutaneous cell-mediated immune reactions (6) has been introduced as an additional therapeutic agent in MF (9). This is because a number of patients with MF show a reduced ability to develop cellular immunity (8) in addition to a lowered number of circulating T-cells (10).

We report on the use of TF as additional therapy in a patient with MF who had not responded to previous conventional therapy alone. TF, initially given together with X-irradiations, 900 r in all, to two tumours, led to complete remission and probably cure. This was indicated by a post-mortem examination 5 years later, when the patient died following a coronary infarct.

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Fig. 1. The patient, with generalized plaques on the trunk prior to treatment (May 1973).

Fig. 2. Ulcerated tumour on elbow prior to treatment with TF (June 1973).
CASE REPORT

The patient, a 64-year-old male was admitted to our department in May 1973 with generalized plaques of typical MF (Fig. 1) together with ulcerated tumours located on the elbows (Fig. 2) and the gluteal area. At the same time the palms and soles (Fig. 3) showed heavy keratotic infiltration with many fissures. A biopsy confirmed the diagnosis (Fig. 4) and treatment was started with topical nitrogen mustard. Due to severe disabling on account of ulcerations of the hands and feet, methotrexate was given from June 28 till September 5 together with grenz rays on three occasions. This treatment had no effect on the lesions. Due to a low T-cell count (18% against 55–65% in 12 normal controls) and low stimulation indices in a lymphocyte transformation test (9), TF-treatment was initiated in September 1973 and at the same time X-irradiation (900 r in all, 50 kV, 1 mm Al) was given to three tumours (from October 3 to April 2, 1974) in order to reduce the tumour load. In May 1974 the patient went into remission. This remission—with the exception of a short period in June 1976—lasted until his death in June 1978.

Apart from further grenz rays, 1200 r supplied with X-rays, 400 r in all, the patient was treated with TF only. He received altogether 12 injections of TF produced from approximately 5 x 10⁸ mononuclear cells from 500 ml venous blood drawn from healthy tuberculin-positive donors. The first 6 injections were given at monthly intervals, and subsequently at variable intervals. He received his last TF injection in January 1978. At that time the patient was in complete remission without visible skin lesions.

On June 3rd 1978 the patient was taken into the medical ward at his local hospital, with acute heart failure. He died on June 12 and a post-mortem showed thrombosis in his right coronary artery with a large myocardial infarct. Neither the skin nor any of his internal organs showed any signs of MF. No pathological lymph nodes were found, either peripherally or internally.

A preliminary report on the therapeutic results was given in 1975 (9). In this, the responses of his peripheral blood lymphocytes to TF were shown. The percentage of his T-lymphocytes rose from 18% to 42%. The latest value (from January 1978) was 39%.

DISCUSSION

TF has been given as additional therapy to a group of patients with MF for more than 3 years. The results have recently been published (11) and seem promising. Besides an increased frequency of remissions, the number of circulating T-lymphocytes—which was low prior to treatment—rose during the observation period to normal values.

The present case report supports the original claim of a beneficial effect of TF as additional therapy for our patient (9). When he died of his coronary disease, he had no signs of mycosis fungoides, either in the skin or in the internal organs. This is to our knowledge the first post-mortem report on a patient with a malignant lymphoma treated for more than 5 years with TF. The present findings indicate that TF treatment is a safe procedure for this group of patients. The data are felt to justify our proceeding with investigations in a controlled double-blind study on TF in MF.

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REFERENCES

Acquired Epidermolysis Bullosa Treated with a Gold Compound

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Abstract. A 20-year-old man with acquired epidermolysis bullosa of 3 years' duration was treated intramuscularly with gold sodium thiomalate. After a total dose of 1000 mg gold sodium thiomalate, administered over a period of 9 months, the patient has shown an almost complete remission, without any apparent side effects of the chrysotherapy.

Key words: Acquired epidermolysis bullosa; Gold sodium thiomalate

Epidermolysis bullosa includes both inherited forms and an acquired non-hereditary form—epidermolysis bullosa acquisita (EBA) (1). This group of diseases is characterized by erosions, formation of bullae with superficial mild scarring and milia formation localized to the sites of apparently slight trauma, predominantly on the elbows, hands, knees and feet. In some sub-groups, minor to severe dystrophic changes and lesions of the oral mucosa occur.

EBA is a rare form which appears in adult life and is often associated with systemic diseases such as Crohn's disease and ulcerative colitis (8).

Many therapeutic agents, primarily corticosteroids, have been used with varying success in patients with EBA. Using a gold compound, we have successfully treated one patient who had previously responded poorly to other treatments.

CASE REPORT

The clinical, histopathological, immunological and gastroenterological findings in this 20-year-old patient with EBA and Crohn's disease have been reported earlier (4). In 1976 he was initially treated with prednisone 40 mg and azathioprine 150 mg daily. The prednisone dose was subsequently reduced to 5 mg daily. After 14 months of therapy, the patient showed a complete remission of the gastrointestinal disorder. However, after a slight remission of the skin symptoms, the patient suffered an exacerbation of these symptoms, with numerous new bullae, and was re-admitted in February 1978 (Fig. 1). Despite an increase in the dose of prednisone, the skin lesions could not be brought under control. Treatment with gold sodium thiomalate was therefore instituted. A 10 mg test dose was given intramuscularly, supplemented by 20 mg on the 7th and the 14th day. Subsequently he received 50 mg weekly until a total dose of 1000 mg gold sodium thiomalate had been used. During this treatment there was a gradual, but slow recovery, and after 9 months of therapy an almost complete remission of the skin lesions and the lesions of the oral mucosa was obtained (Fig. 2). No side effects of the chrysotherapy have been observed.

DISCUSSION

Systemically administered corticosteroids, corticotropin and vitamin E have been used in the treatment of EBA (3, 5, 7, 8). The reported results,