Treatment of mycosis fungoides

Heiner Bruns

The author reports on the treatment of mycosis fungoides (previously known as parapsoraisis en plaques) with homeopathic antihomotoxic agents (Psorinoheel, Traumeel, Lymphomyosot, Galium-Heel), vitamin C infusions and the mistletoe product Helixor® P (150 mg).

As a result of this treatment, the skin eruptions were at least reduced to a few residual patches. The patient’s general well-being improved considerably. Furthermore, no new areas were found to be involved when skin manifestations re-appeared. Some patients die within the first year after being diagnosed with mycosis fungoides. On the reported treatment, the patient presented in this case study has reached her 4th year since first being diagnosed.

Introduction

The clinical picture of mycosis fungoides is variable and, in the early stages, is reminiscent of psoriasis vulgaris. However, the skin patches that develop are very often painful, dry, scaling and red. In dermatology, the early stage of this disease is also referred to as parapsoraisis en plaques.

The incidence of mycosis fungoides is roughly 1/1 million and it belongs to the category of T cell lymphomas of the skin (non-Hodgkin’s lymphomas, NHL). In later stages, the internal organs, such as the spleen and the lymph nodes lying under the skin, become involved. Survival times range from 2 to 7 years according to the literature.

Conventional treatment comprises PUVA radiotherapy and administration of the chemotherapeutic agent Meladinine® for photosensitisation, exposure to beta rays or high-voltage therapy, with all the familiar side-effects.
Case study

Case history
M.B., female, aged 42, came to my practice for treatment about three years ago, primarily for physical and mental exhaustion associated with overwork involving 16-hour working days (a church employee running a Lebenshaus* and responsible for the pastoral work in three parishes).

Initial examination and a detailed medical history revealed the following:

- a family history of breast cancer (the relative is living without a relapse and is currently aged 81)
- oligo-amenorrhoea already at the age of 35 years
- smokes about 10-15 cigarettes a day
- history of surgery for ovarian cysts with endoscopic ablation in 1996/97
- sacrococcygeal fistula in January 1999, re-operated on in June 1999 with prolonged secondary healing
- appendectomy in June 1999 (histology: scarred appendicitis)
- November 1999 lymphadenectomy and screening for breast disease with biopsy from the left breast
- hypertension, situational hypotension
- May 2000 bone marrow biopsy in LA
- dry, desquamative and reddened rash described as burning, appearing on the neck, throat, arms and both armpits, breast and groin area since about 1997/98

In October 1999 a lymph node was found in the left axilla, which measured roughly 2 x 3 cm and was not tender on pressure, as well as rather tender mastopathy with peri-mammillary induration and tenderness to touch at about the 3 o’clock position. In view of the positive family history, this led to a biopsy being taken from the left breast and lymphadenectomy in the left axilla.

**Histology:** Lymph nodes: Pronounced, chronic, non-specific inflammation with variably sized follicles and light germinal centres. Distinct sinus histiocytosis. Breast: Stromal fibrosis with ectatic excretory ducts. No atypia. Lobulated fatty tissue and normal mammary gland tissue.

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* TRANSLATOR’S NOTE: Lebenshaus can mean a hospice, crisis support house or interim emergency accommodation.
In view of the questionable malignancy grade of 1 in the Kiel classification of lymphomas, this tissue was re-examined. As a result, a diagnosis of dermatopathic lymphadenitis was made, whereby these changes are also observed in cases of primarily cutaneous T cell lymphoma, especially mycosis fungoides. However, no distinct infiltration was detected in this patient.

A more in-depth, molecular biological investigation was recommended and took place in February 2000. This PCR (polymerase chain reaction) gave a positive result in a fresh skin biopsy and in the histological examination. After another biopsy and both molecular biological and histopathological analysis, the diagnosis of mycosis fungoides was established in April 2000.

**Treatment**

Treatment with autologous blood, Toxi-Loges L 90, Ubichinon compositum, Coenzyme compositum, Traumeel and Zeel went ahead in October/November 1999. However, immediate success was not achieved with this detoxifying treatment. As a result, the above-mentioned diagnostic procedures and the numerous biopsies were arranged in order to verify the diagnosis conclusively.

As the patient was unwilling to undergo conventional chemotherapy, the following treatments were started in June 2000:

1. bowel decontamination with Hylak forte and yoghurt
2. infusion of 15 g Vitamin C Pascoe®, twice to three times a week
3. Psorinoheel® N, Traumeel® S, Galium-Heel® N and Lymphomyosot® ampoules, each three times a week by subcutaneous injection

Overall the skin was slightly improved but fresh, painful patches measuring up to 3 x 4 cm continued to appear.

From around August 2000, the patient and I decided to start Iscador® in an ascending dosage alongside the above treatment regimens. On this medication, however, she experienced a significant local reaction with an allergic component, which is why this treatment was withdrawn again after about two months. Nevertheless, since the skin lesions had diminished slightly on Iscador®, we decided to attempt treatment with Helixor® P, also at ascending dosage levels.
Some newly appeared skin patches were directly injected with Helixor® with the addition of Meaverin® 2 % or Procain Steigerwald® 2 %, up to 4 ml per dose.

The remarkable thing about this treatment, given in addition to the background therapy, was that the patches completely disappeared within about 4-8 days and entirely normal skin developed, with slight scaling but without any redness or burning. There were no longer any skin indurations around the injected patches. Helixor® (150 mg daily) and Meaverin were injected in addition to the background therapy until April 2001.

The infusions of vitamin C were given at the weekends, on Saturdays and Sundays. In terms of side-effects of these infusions, we observed slight flush around the eyes and increased light-sensitivity which continued for up to an hour after infusion. The treatment was well tolerated, the patient’s physical and mental capacity as well as the skin symptoms were markedly improved. With regard to systemic infections, a short febrile phase was noted in spring 2001 during the influenza season but the treatment was not stopped.

The Helixor® dose was reduced to 100 mg from 19 March 2001 but, because of deterioration in the condition of the skin, it was re-increased to 150 mg from 25 May 2001 (see Figure).

At present it is noticeable that the patient’s circulatory situation is often very unstable because of hypotensive blood pressure levels.

Histological analysis and the polymerase chain reaction applied to another biopsy of an acute eruption in February 2001 produced a negative result for a monoclonal population of a cutaneous T cell lymphoma. Moreover, conventional histology of the biopsy specimen revealed no evidence of increased T lymphocytes.

**Concluding remarks**

Physical capacity, mental stability and skin lesions improved on the described treatment. In particular, the possibility of maintaining a good quality of life should be stressed. Depressive moods are far less frequent. It is also remarkable that the skin lesions disappeared within about a week following the injection of fresh eruptions with Helixor® P 150 mg, combined with Procain 2 % / Meaverin 2 %.
Acknowledgements: I wish to express particular thanks to my colleagues who helped devise the treatment plan, were always available for advice despite the complexity of the clinical picture and encouraged me to dare to attempt an alternative, natural form of therapy. It is definitely worth the effort to use this therapeutic regimen in addition to conventional treatment.

References

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14.12.1998
Screening for tumour markers: CEA 4.8 ng/ml + (normal: < 3.5 ng/ml [smoker ↑]), CA 19-9 8.5 ng/ml (normal: < 37 ng/ml), α1-fetoprotein 5.3 ng/ml (normal: < 10)
Cholesterol: 223 mg/dl + (normal: 200 mg/dl)
Other laboratory results were basically unremarkable, especially rheumatoid factors, CRP, AST negative.

26.05.1999
Diff. BC: neutrophils 81 ++, otherwise rather fewer lymphocytes and monocytes, slight increase in α1-globulins to 6.8% (normal: up to 4.0%) in electrophoresis. Gamma-globulins reduced to 9.7% (normal: 10.0%)

28.05.1999
Check of tumour markers unchanged. Allergic component (CAP) normal.

02.11.1999
Mercury 1.7 ng/l (0.1-5), normal electrophoresis with slight decrease in total serum protein (5.8 g/dl), no monoclonal gammopathy, coagulation and rheumatoid factors normal, thyroid check unremarkable. Diff. BC: unremarkable, erythrocytes 4.1 mill/μl slightly reduced, slight anaemia. Urinary electrophoresis: Bence-Jones negative. Urinary protein 31 mg/24 h (normal: < 150).

17.12.1999
Total serum protein 6.7 g/dl. ESR 5/17.

10.01.2000
Diff. BC: lymphocytes 40.7, eosinophils 4.2, otherwise normal. CEA 4.9 ng/ml (normal: < 3.5), CA 19-9 7.2 U/ml (normal: 37) α1-fetoprotein < 1.3 ng/ml (normal: 10 ng/ml), no antibodies to Borrelia burgdorferi, listeriosis, toxoplasmosis.

30.03.2000
Antimitochondrial Ab (AMA) doubtful positive. ESR 2/6, IgG 6.9 g/l (7-16 normal range), IgA 1.1 g/l (0.7-4 normal range), IgE 45.4 g/l (< 100 normal).

15.02.2001
Haemoglobin 15.8 g/dl, erythrocytes 4.3 mill/μl, haematocrit 50.1 %, MCH 36.0 pg, MCV 116 fl, creatinine 1.7 mg/dl (13.04.01 check: 1.2 mg/dl), cholesterol 252 mg/dl, neutral fats 216 mg/dl, glucose (fasting) 76 mg/dl, total serum protein 7.5 g/dl

Tab. 1 Summary of laboratory parameters
Tab. 2: Lymphocyte differential counts; 25.04.2000 = baseline values (without antihomotoxic therapy), 25.07.2000 = after 2 months’ antihomotoxic therapy plus vitamin C infusions (background therapy), 07.12.2000 = background therapy plus Helixor® P (150 mg daily)

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<tr>
<td>Total</td>
<td>2,679</td>
<td>1,952</td>
<td>1,457</td>
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<tr>
<td>T lymphocytes</td>
<td>2,222 (83 %)</td>
<td>1,537 (79 %)</td>
<td>1,127 (77 %)</td>
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<tr>
<td>B lymphocytes</td>
<td>362 (14 %)</td>
<td>270 (14 %)</td>
<td>134 (9 %)</td>
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<td>T4 helper cells</td>
<td>1,570 (59 %)</td>
<td>909 (47 %)</td>
<td>809 (56 %)</td>
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<tr>
<td>Suppressor T8</td>
<td>574 (21 %)</td>
<td>526 (27 %)</td>
<td>305 (21 %)</td>
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<tr>
<td>Natural killer cells</td>
<td>57 (2 %)</td>
<td>38 (2 %)</td>
<td>35 (2 %)</td>
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<tr>
<td>T4:T8 ratio</td>
<td>2.7</td>
<td>1.7</td>
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other laboratory results on 07.12.2000: CRP < 5.0 mg/l (normal: < 5), α1-fetoprotein 4.3 ng/ml (normal: < 10), neopterin 5.0 mmol/l (normal: < 10), β2-microglobulin 1.5 mg/l (normal: 2.4), interleukin-2 receptor 553 kU/l (normal: 220-710)

Figure: Right axilla in mycosis fungoides. Treatment: Traumeel S, Galium-Heel, Lymphomyosot, Psorinoheel and Helixor®. A) Early May 2001: the condition of the skin deteriorated on the reduced Helixor® dose of 100 mg, which is why this was increased again to 150 mg in June 2001 (B).