Las evaluaciones se hicieron a ciegas, por dos dermatólogos independientemente. Se tomò fotografía de los pacientes, antes y después del tratamiento. Se obtuvo la diferencia en grados entre la evaluación inicial y final, y se consideró PEOR: diferencia negativa, IGUAL: diferencia = 0, MEJOR: diferencia > 0 y < 1.0, y MUCHO MEJOR: diferencia ≥ 1.0. Las proporciones de pacientes dentro de cada categoría en los dos grupos de tratamiento se compararon mediante la prueba de χ^2.

RESULTADOS Y DISCUSION

La distribución de pacientes en cuanto a edad, sexo, raza, antecedentes familiares de acné, tiempo de evolución de la enfermedad y clasificación inicial según el grado de acné, fue similar en ambos grupos. En la tabla se muestra la frecuencia y porciento según evaluación final en cada grupo de tratamiento, así como en las salidas.

El grupo tratado con EGF presentó mejoría significativa ($p < 0.01$) con respecto al que recibió placebo. Las lesiones que mejor respondieron al tratamiento con EGF fueron las no inflamatorias (comedones) y dentro de las inflamatorias, las pápulas y pústulas. No se presentaron reacciones adversas locales ni sistémicas en ningún paciente.

Evaluación final	Placebo		Crema EGF	
	Frecuencia	%	Frecuencia	%
Mucho mejor	2	6,66	15	50,0
Mejor	15	50,0	8	26,66
Igual	10	33,33	6	20,0
Peor	3	10,0	0	0
Salidas	0	0	1	3,33

Estos resultados evidencian que el EGF puede influir corrigiendo las alteraciones cutáneas implicadas en la fisiopatología del acné. Hasta donde conocemos, éste es el primer reporte del empleo de EGF en humanos para el tratamiento del acné. Se considera oportuno continuar las investigaciones al respecto.

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EFFECT OF TRANSFER FACTOR ON MYELOSUPPRESSION AND RELATED MORBIDITY INDUCED BY CHEMOTHERAPY IN ACUTE LEUKAEMIAS

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INTRODUCTION

Important advances have been made in the treatment of acute leukaemias (AL), which have become curable diseases. However, an intensive therapy is required to achieve the cure, especially in acute myelocytic leukaemia (1, 2). Myelosuppression and the infections and hemorrhages that follow it, have emerged as the limiting toxicities of chemotherapy (3, 4). Transfer Factor (TF) preparations have been found to contain molecules with bone marrow-stimulating or protective action as clonal expansors (5). The aim of this study is to determine the safety and efficacy of TF in accelerating the haematopoietic recovery in patients with AL, following intensive therapy to induce remission of the disease.

METHODS

Twenty-two patients with different types of AL (16 acute myelocytic leukaemia - AML-, 3 myelocytic blast crisis from chronic myelocytic leukaemia - BC - CML-, and 3 acute lymphocytic leukaemia-ALL) were studied. They were between 16 and 82 years (median 41) old. There were 13 male and 9 females. The criteria for patient eligibility were: to have a leukocyte count < 1.0 x 10^9/L, a platelet count < 30 x 10^9/L, and hypoplastic or aplastic bone marrow with < 5% blast cells after the end of induction treatment. Informed consent was obtained from the patients or relatives. The patients were randomly distributed in two groups. Group 1 (8 AML, 2 BC-CML and 1 ALL) received, after myelosuppression induced by

chemotherapy, TF (1 unit daily, subcutaneous) until leukocyte count was > 2.5 x 10^9/L and platelet count > 80 x 10^9/L. Group 2 did not receive TF.

RESULTS

Treatment with TF accelerated the recovery of neutrophils, leukocytes, platelets and hemoglobin. The incidence and severity of infections and hemorrhages were less in the TF group than in the control group. There was no evidence that TF accelerated the re-growth of leukaemic cells. It seems that TF is safe in AL, accelerating haematopoietic recovery. However, it should be used with caution until results of additional trials become available.

Days to hematopoietic recovery (X ± SD)

Parameters	Group 1 (n=10)*	Group 2 (n=9)**	p
Neutrophilis > 1.0 x 10^9/L	4.7 ± 2.4	22.6 ± 8.5	< 0.001
Leukocytes > 2.5 x 10^9/L	4.7 ± 2.4	21.1 ± 9.3	< 0.001
Platelets > 80 x 10^9/L	7.5 ± 3.7	17.6 ± 6.8	< 0.001
Haemoglobin > 10 G/L	10 ± 5.5	20.0 ± 11.5	< 0.001

*One patient with blast cells in pancytopenia period was not included.
**Two patients that died in aplasia were not included.

Transfusions and antibiotic treatments

	Group 1 (n=11)	Group 2 (n=11)	p
Red-Cell transfusions (units)	1.0 ± 1.1	3.5 ± 2.6	<0.01
Platelet transfusions (units)	4.1 ± 5.7	19.0 ± 14.7	<0.01
Leukocyte transfusions (units)	1.4 ± 3.3	5.6 ± 9.0	n.s.
Antibiotics (days of treatment)	10.0 ± 7.1	24.9 ± 16.5	<0.05

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INTERFERON ALPHA-2B IN EPIDEMIC NEUROPATHY

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INTRODUCTION

More than 40 000 cases of an epidemic Neuropathy were reported in Cuba during 1993. It had two clinical pictures: an optic neuritis and a peripheral neuropathy (1). A nutritional unbalance and toxic as well as opportunistic infectious agents have been involved in the pathogeny of the disease. Several virus isolates were obtained from patients cerebrospinal fluid. One of them was identified as Coxsackie A9 serologically and by partial genome sequence. Its cytopathic effect in vitro is sensitive to inhibition by IFN alpha (2).

METHODS

Several controlled, randomized, clinical trials were carried out with different treatments, among them IFN at various therapeutic regimes, always compared to a basal polyvitamin schedule that was given to all patients. Five IFN trials, performed at 12 hospitals, included 212 patients with optic neuritis and 460 with peripheral neurological symptoms (including the control groups). Patients were less than 3 months sick. IFN α2b (Heberon, Heber Biotec, Havana) was given 3 times per week during 3 weeks at 6, 3 or 1 mill. IU per dose, depending on the trial.