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LETTER TO THE EDITOR

Psoriasis and Hepatitis C: Improvement with Interferon

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Dear Editor:

Interferon, an immunomodulator and antiproliferative with antiviral properties, has side effects that include flu-like symptoms, marrow suppression, alopecia and depression. Other side effects of interferon are rare skin reactions[1,2], namely erythematous patches at site of injection and eczema. Interferon may also induce or "unmask" underlying skin diseases such as psoriasis[3,4], eczema, sarcoidosis and lupus. The combination of interferon and ribavirin is the current treatment for hepatitis C. We present a patient with extensive psoriasis whose symptoms dramatically improved when she was treated with pegylated interferon and ribavirin for hepatitis C.

A 52-year-old white female was referred to us for management of chronic hepatitis C infection in April 2006. She had nonspecific symptoms. She also had hypertension, diabetes mellitus, mild rheumatoid arthritis, depression and obesity. Since age eight, she had had psoriasis involving multiple areas on her body. She expressed low esteem, secondary to the degree of psoriatic involvement and her obesity. She had no allergies.

On examination, the patient was alert and had normal vital signs. HEENT (Head, Ears, Eyes, Nose and Throat), cardiopulmonary, abdomen, extremities and neurological examinations were normal. She had no lymphadenopathy. Skin examination was remarkable for extensive bilateral erytherodermic nodular lesions with plaques involving the extensor surfaces of elbows, forearms, thighs and knees. Lesions were more prominent on the left side. She also had scattered involvement over the abdomen, lower back and scalp. Approximately 15～20% of her skin surface was covered with lesions (at this time, no photos were taken) (Table 1).

Medications included metformin, hydrochlorathiazide, loratadine and acetaminophen. She was using clobetasol cream topically.

In June 2006, the patient was started on hepatitis C treatment with a combination of interferon alfa-2b (1.5 mcg/kg/week subcutaneously) and ribavirin (1,200 mg) orally every day. In the third week following initiation of therapy, she complained of a skin rash and itching over the lower extremities, which subsequently spontaneously improved. An examination at that time showed diffuse, erythematous, maculopapular eruptions which was not psoriatic. She also had flu-like symptoms secondary to interferon. After three months, the patient complained of diarrhea, abdominal cramps and hair loss, but her psoriasis had improved. Examination revealed definite improve-

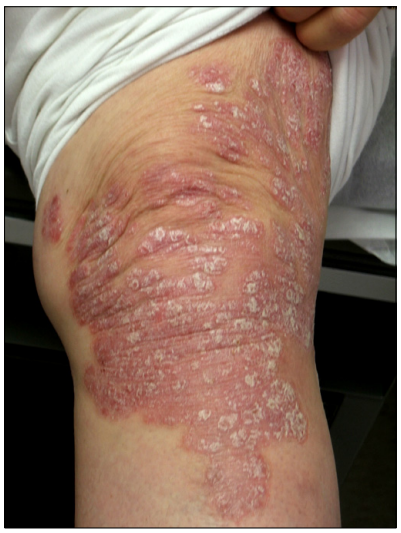

Fig. 1. Three month follow-ep showing improvement in psoriasis.

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Table 1. Laboratory studies

	4/27/06 Initial visit: Tx begins 6/06	11/15/06 & 12/11/06 FU visits: 5 and 6 mos.	4/06/07 FU visit: 1 yr.	5/04/09 FU visit: 3 yrs.	2/12/10 FU visit: 3½ yrs
WBC 10^3/ul	11.3	5.6	9.2	10.4	9.0
Differential counts	Normal	Normal	Normal	Normal	Normal
Hb (g/dl)	15.8	14.0	15.0	16.3	14.4
Hct (%)	46.8	41.7	44.7	48.6	42.2
Albumin (g/dl)	3.9		3.9	3.8	4.1
SGOT (AST) U/l	26	22	20	14	17
SGPT (ALT) U/l	44 (↑)	27	19	16	28
Alkaline phosphatase (U/l)	100	82	101	91	71
Bilirubin T/D mg/dl	0.4/0.1	0.5	0.6/0-1	0.3/0-1	0.3/0-1
TSH mU/l	1.42		1.80		1.64
Hep C RNA viral load per milliliter	6,036,990	Below level of detection	Below level of detection	Below level of detection	Below level of detection
HIV antibody test	Negative				
Cryoglobulin test		Negative	Negative		

Tx: treatment, FU: follow-up, WBC: white blood cell, Hb: hemoglobin, Hct: hematocrit, SGOT: serum glutamic oxacetic transaminase, SGPT: serum glutamic pyruvic transaminase, AST: spartate minotransferase, ALT: alanine minotransferase, T/D: total/direct, TSH: thyroid stimulating hormone, HIV: human immunodeficiency virus.

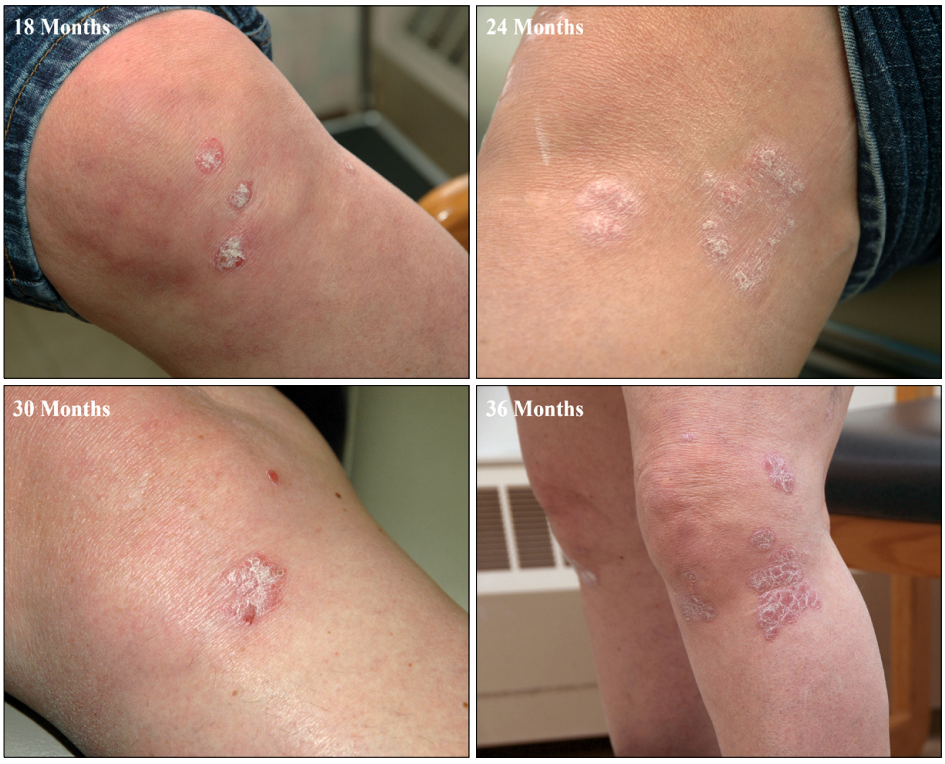

Fig. 2. After completion of interferon/ribavirin therapy, examination showed only a mild reactivation of the psoriatic lesions.

ment of her skin lesions with less erythema and scaling of the plaques on the elbows, knees and thighs (Fig. 1). There was also significant clearing of the lesions on the scalp, abdomen and back. Serum hepatitis C viral-RNA load decreased to an undetectable level. After the completion of six months interferon/ribavirin therapy, she has had no relapse of hepatitis C infection and only a mild reactivation of the psoriatic lesions (Fig. 2). During this time, she denied use of topical steroids or other psoriasis treatments. Exacerbation of psoriasis following the use of interferon in the treatment of hepatitis has been reported[5,6]. In fact, interferon is believed to be associated with the patho-

genesis of psoriasis[5,7,8]. Both hepatitis C and psoriasis are diseases in which tumornecrosis factor-α (TNF-alpha) is involved.

During treatment with interferon/ribavirin, this patient's psoriasis improved and has remained less active for 4+ years following treatment. We have no definite explanation for the significant improvement of psoriasis. The patient has been under no psoriatic treatment program during this time. Precipitating factors such as infection can activate T-cells and cytokine production, resulting in the release of inflammatory mediators that can flare psoriasis. In this patient, perhaps the active hepatitis C infection precipitated more dramatic psoriatic involvement. This hypothesis is supported by the significant decrease and subsequent disappearance of detectable serum load over the time her psoriasis has shown such significant improvement. Further investigation is needed to show a direct causal relationship between the patient's treatment with interferon/ribavirin and improved psoriasis through the decreased activation of T-cells and cytokine production.

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