

Exacerbation of Skin Lesions in a 50 year old Man with Psoriasis during Treatment by Pegylated Interferon

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ABSTRACT

Chronic hepatitis C might lead to several immunological dysfunctions. Studies have shown a positive association between hepatitis C virus (HCV) infection and psoriasis. These results suggest that the infection may be one of the triggering factors for the development or exacerbation of psoriasis.

Here, we present a case of chronic HCV infection with psoriasis who developed exacerbation of skin lesions during therapy with peginterferon alpha—2a plus ribavirin. We discuss the management, course and results of HCV treatment in this patient.

KEYWORDS:

HCV; Psoriasis; Pegylated interferon; Ribavirin

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INTRODUCTION

Hepatitis C virus (HCV) is a leading cause of chronic liver diseases, and an important cause of morbidity and mortality worldwide. It is estimated that 170 million individuals are living with HCV worldwide. There are controversies regarding the causality between HCV infection and some skin manifestations.¹ HCV-related chronic hepatitis C might lead to both cellular and humoral abnormalities.

In several studies, the prevalence of HCV-Ab was higher in patients with psoriasis than in the normal population. These results have suggested that infection and/or HCV treatment may act as a triggering factor for development or exacerbation of psoriasis.²⁻⁴ There are several reports of vesicular erythematous eruptions,⁵⁻⁸ vitiligo⁹ and subcutaneous sarcoidosis¹⁰ in patients treated with interferon and ribavirin.

CASE REPORT

We present the case of a 50 year old male patient diagnosed with psoriasis who referred to our clinic at Tehran University of Medical Sciences.

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On his first admission in April 2007, he had a positive anti-HCV antibody (anti-HCV Ab) test. Except for the history of psoriasis, which was under control with anti-psoriatic medications, his past medical history was unremarkable.

Physical examination revealed healing silvery scale psoriatic plaques distributed over both forearms and lower legs without pustules and pitting of nails caused by the disease (Picture 1, 2). The remainder of the physical examination was unremarkable.



Fig 1 : Psoriatic scaly skin lesions over right elbow (upper arrow) and right buttock (lower arrow)



Fig 2 : Scaly lesions of psoriasis over anterior aspect of both knees (right and left arrows)

The patient was infected with genotype 3a and the viral count was 223,000 IU/ml. Levels of thyroid stimulating hormone, albumin, bili-

rubin, prothrombin time, blood urea, alkaline phosphatase and creatinine were within normal limits.

The alanine transaminase level was 66 IU/L (normal <40 IU/L) and aspartate transaminase 65 IU/L (normal <40 IU/L). The complete blood count was also normal. A liver biopsy was performed which showed an inflammation grade of 11/18 and fibrosis stage of 3/6 according to the Ishak classification.

The abdominal ultrasonic examination was normal, except for a mildly enlarged spleen. There was no evidence of fatty liver or other pathological findings. Portal vein diameter was 12 mm.

The patient was started on standard HCV therapy with pegylated interferon alfa-2a (Pegaferon®, Pooyesh Daru, Iran)⁶ and ribavirin for 24 weeks.

At the first week of treatment, the patient suffered from arthro-myalgias, which were not severe enough to warrant treatment. During the second week, he developed generalized itching, particularly at night. This was managed by night-time oral hydroxyzine and topical Calamine-D lotion.

Psoriatic lesions were observed from week three of treatment and new lesions appeared on his legs, elbows, back and knees. His skin lesions became more severe and his dermatologist, in consultation with the hepatologist, recommended cyclosporine 100 mg, twice daily. On week eight of therapy, the patient became neutropenic (absolute neutrophil count: <1000 cell/ml), thus granulocyte colony stimulating factor (G- CSF) at a dose of 300 µg per week was started.

On the third month of therapy, as the healing of psoriatic lesions was not satisfactory, the patient also received 100 sessions of PUVA therapy (Psoralen plus Ultra Violet A treatment 3 times a week, each session ranging from 2 to 250 sec). PUVA therapy continued for the rest of the HCV treatment course.

Although the patient's psoriatic lesions worsened in severity, there were no lesions or any evidence of psoriatic nail or joint complications. He tolerated HCV therapy well until the end of the 24th week of treatment.

Within one month after treatment cessation, the psoriatic lesions receded without the need for additional therapy. HCV RNA was undetectable 6 months after discontinuation of treatment, which indicated sustained viral response. One and a half years after treatment, HCV RNA is still undetectable and the patient has had no exacerbation of his psoriatic lesions.

DISCUSSION

This case highlights several key issues in initiating and managing HCV therapy in a patient with co-existing psoriasis.

The first decision in this case addressed the patient's eligibility for treatment. The patient presented with a relatively high HCV viral level and compensated liver disease (stage 2 fibrosis), which indicated that he was likely to benefit from anti-HCV treatment. Other hematologic and biochemical laboratory findings were acceptable and did not present any contraindication to treatment. However, his history of co-existing psoriasis, although controlled, increased the risk of exacerbation. Our patient was strongly motivated to receive treatment, which was subsequently given. He was closely followed by both a hepatologist and a dermatologist during treatment.

Cyclosporine is an immunosuppressive drug also used for treating psoriasis. In different studies, cyclosporine has been reported to also inhibit HCV- replication.⁷

In our case, cyclosporine had dual therapeutic actions both for the psoriatic lesions and inhibition of HCV replication. Despite the flare up of psoriatic lesions, the subsequent management led to both successful eradication of HCV and control of psoriasis.

In conclusion, we suggest that concomitant psoriasis could not be seen as a contraindication for HCV treatment and exacerbations can be successfully managed by cyclosporine and PUVA.

CONFLICT OF INTEREST

The authors declare no conflict of interest related to this work.

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