

Prevalence of Hepatitis C virus infection in patients with chronic plaque Psoriasis in Damietta Governorate

Zakaria Mahran¹ and Tarek M. Emran²

Departments of Dermatology and Andrology¹ and Clinical Pathology², Faculty of Medicine. AlAzhar University. Egypt

Abstract: Background: Many skin diseases, including psoriasis, have been described in association with hepatitis C virus (HCV). The important observations that propose a role for HCV in psoriasis include the existence of psoriasis in HCV infected patients, detection of anti-HCV antibodies in psoriatic patients and detection of HCV-RNA by PCR in the skin lesions of psoriatic patients with HCV infection. It is possible that the presence of HCV in the skin could trigger psoriasis through stimulating inflammatory cells to infiltrate skin lesions. **Aim of the study:** The aim of the present study was to evaluate the prevalence of HCV infection in patients with chronic plaque psoriasis. **Participants and methods:** A total 120 individuals included in our study and divided into two groups; 60 psoriatic group who had been attending to our dermatology outpatient clinics. The study also included 60 apparently healthy subjects as control group. All individuals were subjected to history taking, general and local examinations including joint examination and laboratory investigation that include immune-fluorescence assay (IFA) of HCV antibodies and Polymerase Chain Reaction (PCR). **Results:** Our results showed that incidence of HCV infection was significantly higher in psoriatic patients compared with the control subjects ($P < 0.001$). **Conclusion:** Our results suggest that there is association between psoriasis and HCV infection and helping us to recommend to screening for HCV infection in severe and longstanding psoriatic patients.

[Zakaria Mahran and Tarek M. Emran. **Prevalence of hepatitis C virus infection in patients with chronic plaque Psoriasis in Damietta Governorate.** *J Am Sci* 2015;11(7):130-133]. (ISSN: 1545-1003). <http://www.jofamericanscience.org>. 16

Keywords: HCV, psoriasis, IFA, PCR

1. Introduction

Psoriasis is an ancient and universal inflammatory disease, initially described at beginning of medicine; Hippocrates used the term “*psora*”, meaning, "To itch". It is defined as a clinical entity affecting skin, nail, mucous membrane and joints. It is generally easy to recognize psoriasis when it presents in one of three typical presentations; guttate, pustular and plaque-stage¹.

Psoriasis is a chronic, non - contagious, multisystem, inflammatory disorder. Patients with psoriasis have a genetic predisposition for the illness, which most commonly manifests itself on the skin of the elbows, knees, scalp, lumbosacral areas, intergluteal clefts and glans penis. The joints are also affected by psoriasis in up to 30% of patients with the disease².

Psoriasis is a chronic inflammatory disease which affects approximately 2 – 3 % of the world's population, including 125 millions people world wide³.

Many skin diseases, including psoriasis, have been described in association with hepatitis C virus (HCV). There is suggestion that HCV infection may act as trigger for systemic inflammation independent from the persistence of active infection. Increased

psoriasis area and severity index (PASI) in HCV-positive patients could be explained by common pathogenic mechanisms in psoriasis and HCV infection including production of interferon (IFN)⁴.

There are three important observations that propose a role for HCV in psoriasis. First, the existence of psoriasis in HCV infected patients. Second: detection of anti-HCV antibodies in psoriatic patients. Finally: detection of HCV-RNA by PCR in the skin lesions of psoriatic patients with HCV infection. It is possible that the presence of HCV in the skin could trigger psoriasis through stimulating inflammatory cells to infiltrate skin lesions⁵.

The onset of psoriatic arthritis (PsA) has been described in patients with chronic hepatitis C infection during INF- α therapy, suggesting that both drug and viral infection might play a role in promoting the development of this condition⁶.

2. Participants and methods

This study was conducted during the period from January 2014 to January 2015 and included 60 psoriatic patients who had been attending to our dermatology outpatients clinic at Damietta University hospital, Al-Azhar University. The study also included 60 apparently healthy subjects as control group. All

psoriatic patients were of vulgaris type. Patients taking medications as methotrexate and etretinate or drinking alcohols were excluded from the study because of their potential hepatotoxic effects. Patients with history of parenteral exposure as intravenous drug addiction, blood transfusion or previous surgery were excluded from the study because of their potential infections with viruses including HCV. All individuals were subjected to history taking, general and local examinations including joint examination and laboratory investigation that included immunofluorescence assay (IFA) of HCV antibodies. In addition, PCR was done to estimate the burden of HCV infection.

Laboratory evaluation

Five ml of venous blood were taken from patients and controls. Blood samples were collected in plain tube, left to clot, centrifuged and serum was separated. Serum was used to determine anti-HCV antibodies by Immunofluorescence assay (IFA). Quantification of HCV infection was done using Abbott Real Time HCV kit; Reference number 1N30; catalog number 51-608374/R1; it is an *in vitro* reverse transcription-polymerase chain reaction (RT-PCR) assay for use with the Abbott Sample Preparation System reagents and with the Abbott m2000sp and m2000rt instruments for the quantitation of hepatitis C viral (HCV) RNA in human serum or plasma (EDTA) from HCV-infected individuals. Specimens containing HCV genotypes 1 – 6 have been validated for quantitation in the assay. The assay is standardized

against the Second WHO International Standard for Hepatitis C Virus RNA (NIBSC Code 96/798)¹² and results are reported in International Units/mL (IU/mL). The Abbott Real Time HCV assay consists of three reagent kits: Abbott Real Time HCV Amplification Reagent Kit; Abbott Real Time HCV Control Kit and Abbott Real Time HCV Calibrator Kit. The test was done according to manufacturer instruction.

3. Results

Mean age in psoriatic and control groups was 39.9 ± 12.9 and 40.28 ± 12.48 respectively as shown in Table (1). Males represented 52.5% of all included cases and females represented 47.5%; and there was statistically insignificant increase of males in psoriasis group in comparison to control group (55% vs 50% respectively) as shown in Table (2).

Regarding incidence of HCV infection in psoriatic patients, it was positive in 26 cases (21.7%) and there was significant increase of associated HCV infection in psoriasis group when compared to control group (35 % vs 8.3% respectively) as shown in Table (3).

Regarding PCR of positive cases; the level ranged from 50900 to 230000 IU/ml with a mean of 126556.15 ± 34741 IU/ml; and there was non-significant increase of viral load in psoriasis group when compared to control group (127360 ± 37846.25 vs 123180.0 ± 19080.53 IU/ml respectively) as shown in (Table 4).

Table (1): Comparison between psoriatic and control groups as regard to age

| | Mean | ±S. D | Minimum | Maximum | t | p |
|-----------|-------|-------|---------|---------|------|----------|
| Psoriasis | 39.53 | 13.39 | 15.00 | 63.00 | 0.31 | 0.75(NS) |
| Control | 40.28 | 12.48 | 18.00 | 60.00 | | |
| Total | 39.90 | 12.90 | 15.00 | 63.00 | | |

Data showed no statistically significant difference in mean age of both psoriatic and control groups (P value > 0.05, no significance).

Table (2): Comparison between psoriatic and control groups as regard to sex distribution

| | Psoriasis | | Control | | Total | |
|------------|--------------------------------|-------|---------|-------|-------|-------|
| | n | % | n | % | n | % |
| Male | 33 | 55.0% | 30 | 50.0% | 63 | 52.5% |
| Female | 27 | 45.0% | 30 | 50.0% | 57 | 47.5% |
| Statistics | $X^2 = 0.30$, $p = 0.58$ (NS) | | | | | |

Data showed no statistically significant difference in sex of both psoriatic and control groups (P value > 0.05; no significance)

Table (3): Comparison between psoriatic and control groups as regard to incidence of HCV infection

| | Psoriasis | | Control | | Total | |
|-------------------|----------------------------|-------|---------|-------|-------|-------|
| | n | % | n | % | n | % |
| Positive | 21 | 35.0% | 5 | 8.3% | 26 | 21.7% |
| Negative | 39 | 65.0% | 55 | 91.7% | 94 | 78.3% |
| Statistics | $X^2 = 12.57, p < 0.001^*$ | | | | | |

The data showed statistically significant difference in HCV infection in both psoriatic and control groups (P value < 0.001: highly significant).

Table (4): Comparison between positive psoriatic and control cases as regard to PCR

| | Mean | ±S. D | Minimum | Maximum | t | p |
|-----------|-----------|----------|-----------|-----------|------|----------|
| Psoriasis | 127360.00 | 37846.25 | 50900.00 | 230000.00 | 0.23 | 0.81(NS) |
| Control | 123180.00 | 19080.53 | 100500.00 | 150200.00 | | |
| Total | 126556.15 | 34741.10 | 50900.00 | 230000.00 | | |

The data showed no statistically significant difference in PCR of both psoriatic and control groups (p value > 0.05; non-significant).

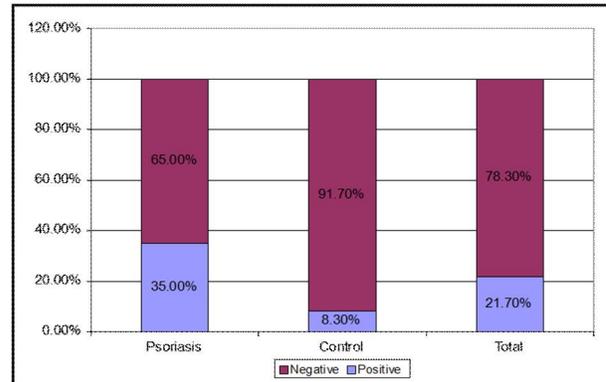
4. Discussion

An association between hepatitis C and psoriasis is frequently described in previous studies^{7,8} and other epidemiological survey confirmed a higher prevalence of HCV infection in psoriatic patients⁹. In addition, it is well known that interferon (IFN) therapy, which can now eradicate HCV from nearly 75% of patients with hepatitis C, induces psoriasis de novo or exacerbates existing psoriasis. Epidemiological studies examining the frequency of HCV infection in psoriasis have yielded mixed results, for example, some results did not detect anti-HCV antibodies in the serum of 13 longstanding British psoriasis patients¹⁰. However, others found HCV infection in 8 of 79 Japanese psoriasis patients and evidence of HCV messenger RNA by PCR in the tissue sections of two patients, which provided potential evidence for active viral replication within the skin tissue as a trigger for psoriasis⁸.

Our study found that no statistically significant difference as regard age and sex of both psoriatic and control groups ($P > 0.05$) as shown in tables (1, 2). While other studies reported that psoriasis is significantly increased among male patients and associated with significant increase of age. These results are contradicted to that of our study, because, the large number of included cases in their study (more than 25000 cases and controls) compared to the small number of included cases in our work can explain this contradiction⁴.

Our study found that HCV antibodies in 21 cases (35%) out of 60 psoriatic patients by IFA while positive results were reported only in 5 persons (8.3%) out of 60 normal control subjects and this agree with other previous studies which report HCV antibodies in

11 cases (36.7%) out of 30 psoriatic patients by IFA, while positive results were reported only in 9 persons (15%) out of 60 normal control subjects¹¹ as shown in figure (1). On the other hand, some studies have shown a relatively low prevalence (7.5%) of HCV infection in psoriatic patients⁹, when compared to that of the present study. The possible explanation of higher incidence of HCV infection in psoriatic patient in our study may be due to endemicity of HCV in Egypt, especially our results coincide with previous reports in Egypt.

**Figure (1): Comparison between psoriatic and control groups as regard to incidence of HCV infection**

Conclusion

Results of the present study added additional evidence to confirm the association between psoriasis and HCV infection, as the percentage of HCV positive psoriatic patients out number those of HCV positive in control subjects.

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6/3/2015