LETTER TO THE EDITOR

GENERALIZED PUSTULAR PSORIASIS AND HEPATITIS C VIRUS INFECTION

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Hepatitis C virus (HCV) is an RNA virus responsible for a high percentage of non-A non-B hepatitis. HCV is known to be related with various skin disorders including psoriasis (*1–3*). Generalized pustular psoriasis (GPP) is a rare form of psoriasis with occasional episodes of acute exacerbations. In the present study, we analyzed HCV RNA copies in the lesional and non-lesional skin from two patients with GPP with positive HCV antibody using a real-time polymerase chain reaction (PCR).

Two patients with GPP who were positive for HCV antibody were enrolled in this study.

Patient 1 was a 76-year-old woman with pustules over her entire body. The patient suffered from recurrent pustules and fever, which was followed by typical psoriasis plaques on her trunk and extremities. The liver enzymes glutamic-oxaloacetic transaminase (GOT) and glutamic-pyruvic transaminase (GPT) were within normal limit. The patient 2 was a 53-year-old woman with 6-year history of GPP with two episodes of severe pustule formation. The disease was generally well controlled. Liver enzyme levels were slightly increased (GOT 65 IU/l; normal: 5–40, GPT 74 IU/l; normal: 4–45 IU/l) during the onset of pustulization.

Full-thickness (4 mm diameter) punch-biopsy samples were obtained from the pustular lesional skin and non-lesional skin of both GPP patients. Eczematous skin lesion and normal skin were also obtained from the 67-year-old man associated with positive HCV antibody. Sera of each patient were also obtained at the time of biopsy. HCV antibodies were detected in all three patients by enzyme-linked immunosorbent assay (ELISA). HCV-RNA was extracted from 10 mg tissue sections and sera with an ISOGEN RNA extraction kit (Nippon Gene, Tokyo Japan). HCV-RNA was detected by a real-time PCR using the methods described by Takeuchi *et al.* (*4*). The primers and probe complementary to sequences located in the HCV 5'-untranslated region; forward primer (nts 130–146) 5'-CGGGAGAGCCATAGTGG-3'; the reverse primer (nts 290–272) 5'-AGTACCACAAGGCCTTTCG-3'.

The results of the real-time PCR are summarized in table. A higher number of HCV-RNA copies was detected in pustular lesional skin than in non-lesional skin in both GPP patients. Conversely, the number of HCV-RNA copies per assay was lower than 10 in an eczematous skin lesion or in a skin from the control healthy patient. On the other hand, HCV-RNA copies were detected in the sera of all patients.

GPP is one of rare variants of psoriasis and is characterized by fever, chills, and generalized pustule formation on a background of erythematous skin. The cause of GPP is obscure; however, a number of provoking agents

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Abbreviations: GOT = glutamic-oxaloacetic transaminase; GPP = generalized pustular psoriasis; GPT = glutamic-pyruvic transaminase; HCV = Hepatitis C virus; LP = lichen planus; PCR = polymerase chain reaction; ELISA = enzyme-linked immunosorbent assay

Table 1. Number of HCV RNA copies in the skin of patients with GPP and eczema who were also infected with HCV

Patient	Age/sex	No. of HCV RNA copies per assay[a]		
		Lesion[b]	Non-lesion[b]	Serum[c]
No. 1, GPP	76/F	2.3×10^4	6.0×10^2	3.8×10^6
No. 2, GPP	53/F	7.0×10^2	<10	4.3×10^5
Control	67/M	<10	<10	5.1×10^5

[a]HCV-RNA was extracted from skin tissue sections and sera, and then detected by the real-time PCR.
[b]Full-thickness (4 mm diameter) punch-biopsy samples were obtained from the pustular lesion and non-lesional skin of GPP patients. The skin lesion of eczema and non-lesional skin from the HCV-infected patient served as controls.
[c]Sera of each patient were obtained at the time of biopsy.

or events such as irritating topical therapy are well recognized in the acute form. Other documented factors have been infections, pregnancy, solar irradiation and drugs.

Recently, several studies have been reported that analyzed the relationship between psoriasis and HCV infection (*1–3*). These observations have suggested that the HCV infection might be one of the triggering factors in psoriasis. However, there had been no report focusing on the relation between GPP and HCV infection. To our knowledge, this is the first trial of real time quantitative PCR analysis of HCV RNA of the skin. Our results showed that there were greater numbers of HCV RNA copies in pustular lesions than in the non-lesional skin of GPP patients. We can speculate that the presence of HCV in the epidermis or dermis is one of the triggering causes of GPP.

Recently, Lazaro and coworkers (*5*) have reported that HCV RNA can be detected in the keratinocytes in all patients with lichen planus (LP) associated with HCV infection. This finding had suggested that HCV infection might contribute to the development of LP lesions in patients with chronic HCV infection. Our results might indicate the possibility that the presence of HCV could trigger GPP by stimulating inflammatory cells to infiltrate skin lesions.

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