

Letter to Editor

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COVID−19 exacerbates psoriasis progression through inducing IFN−α expression

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As a global public health issue that can cause systemic diseases, COVID-19 inflicts various harms on patients and impacts those with comorbidities. Psoriasis is primarily driven by a subset of T helper cells and the cytokines[1], and microbial infection is a predisposing factor in up to 45% of patients[2]. Infection with SARS-CoV-2 may trigger the aggravation of psoriasis. Thus, we conducted a survey to explore the proportion of psoriasis patients who experienced exacerbation and relapse after SARS-CoV-2 infection and also performed a preliminary investigation into the mechanisms involved.

One hundred and twenty-four psoriasis patients (79 males and 45 females) who contracted COVID-19 were followed up and provided detailed information. Exacerbation was defined as a 50% Psoriasis Area and Severity Index (PASI) worsening, and relapse was defined as the re-emergence of rashes with 50% loss of the improvement obtained. We used a commercial assay kit (Raisecare Biotech Ltd.) and a flow cytometry platform to detect cytokines (IL-1β, IL-2, IL-4, IL-5, IL-6, IL-8, IL-10, IL-12p70, IL-17, IFN-α, IFN-γ, and TNF-α) in serum samples from all 124 patients. Furthermore, *in vitro* peripheral blood mononuclear cells from nine patients with exacerbation or relapse and six patients without exacerbation or relapse were inoculated with an inactivated COVID-19 vaccine (vero cells, Sinovac Biotech Ltd.) for 20 hours to identify triggers of psoriasis exacerbation after SARS-CoV-2 infection[3]. Subsequently, the same commercial assay kit and platform were used to analyze the samples as the control.

In our follow-up, 60 (48.4%) patients experienced exacerbated (56) or relapsed (4) psoriasis after COVID-19. There was no significant difference in basic clinical characteristics between the two groups (Supplementary Table 1), and the cytokine test results also showed no significance between them after SARS-CoV-2 infection (Supplementary Table 2). However, in the stimulation test using inactivated vaccine, the results showed that psoriasis patients with exacerbation or relapse had a higher fold change of IFN-α levels compared to those without exacerbation or relapse (P=0.039) (Table 1).

Numerous studies have confirmed that SARS-CoV-2 infection could serve as a trigger factor for the exacerbation of inflammatory and immune diseases: 26% of 1 132 patients with atopic dermatitis and psoriasis; 43% of 21 patients with atopic dermatitis; and 42.5% of 475 patients with alopecia areata[4]. Consistent with the above studies, we found that 48.4% of patients experienced exacerbation or recurrence after SARS-CoV-2 infection. We also found that IFN-α secretion levels were associated with the exacerbation of psoriasis following stimulation by inactivated COVID-19 vaccine. It has been reported that plasmacytoid dendritic cells produce IFN-α when LL37 binds self-DNA/RNA and viral ssRNA, which promotes monocyte-derived dendritic cell maturation. IFN-α-primed monocyte-derived dendritic cells induce T cell differentiation into Th1/Th17 cells in lymph nodes, which migrate to the psoriatic dermis and contribute to plaque formation. This leads to the exacerbation of psoriasis[5]. We attribute the insignificant difference in cytokine test results to the fact that most patients had already recovered from COVID-19 symptoms during their follow-up visits and missed the "cytokine storm" phase. We acknowledge the limitation that we used an inactivated vaccine instead of live viruses for stimulation testing.

Our study suggests that attention should be paid to the exacerbation

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Table 1. The results of cytokine profiles in peripheral blood mononuclear cell stimulation tests using inactivated SARS-CoV-2 vaccine.

Variable	Patients with psoriasis exacerbation (n=9)			Patients without psoriasis exacerbation (n=6)			P value
	Stimulated	Unstimulated	Fold change	Stimulated	Unstimulated	Fold change	
IL-5	1.84±0.45	1.25±0.40	0.34±0.23	1.45±0.22	1.25±0.44	0.14±0.32	0.186
IFN-α	186.60±312.43	0.31±0.35	6.35±1.33	46.04±62.66	0.40±0.51	3.66±3.18	0.039*
IL-2	10.19±12.97	2.25±5.41	1.71±1.41	8.32±6.80	5.83±7.31	0.79±2.69	0.402
IL-6	705.42±1 195.85	265.49±248.29	0.97±1.05	406.40±263.58	303.96±260.17	0.61±0.40	0.447
IL-1β	46.64±75.74	9.00±18.03	1.62±2.83	18.78±27.87	5.48±13.42	1.66±2.05	0.981
IL-10	22.67±40.29	8.56±16.73	0.88±0.96	13.16±15.28	5.46±5.14	0.65±0.66	0.623
IFN-γ	9.33±22.46	0.34±0.41	1.43±1.82	2.21±2.60	0.11±0.10	1.23±1.07	0.812
IL-8	408.52±299.48	216.18±53.83	0.76±0.96	439.15±320.41	268.66±112.77	0.57±0.55	0.667
IL-17	1.29±3.45	2.13±3.29	-0.53±1.68	0.97±1.26	0.19±0.46	0.56±1.14	0.189
IL-4	0.38±0.22	0.30±0.24	0.09±0.40	0.49±0.37	0.43±0.41	0.07±0.35	0.914
IL-12p70	0.88±0.12	0.79±0.10	0.07±0.09	0.90±0.11	0.83±0.11	0.05±0.14	0.678
TNF-α	28.85±42.55	19.26±21.00	0.43±1.19	39.38±36.87	26.79±31.65	0.81±0.72	0.493

Data are expressed as mean ± SD. We calculated the data using [log2(n stimulated +1) - log2(n unstimulated +1)] and performed a one-way ANOVA test with the results. *Statistically significant (P<0.05). A P-value < 0.05 is considered significant. The data was analyzed using SPSS 26.0 statistical software.

of pre-existing inflammatory diseases, such as psoriasis, during the COVID-19 pandemic. The release of IFN-α triggered by SARS-CoV-2 plays a significant role in psoriasis exacerbation.

Conflict of interest statement

The authors declare no conflicts of interest.

Ethics

This study was reviewed and approved by the ethics committee of the Shandong Provincial Institute of Dermatology and Venereology, Shandong Academy of Medical Science (20221016KYKTKS001). Consent for the publication of recognizable patient identifiable material was obtained by the authors and included at the time of article submission to the journal stating that all patients gave consent with the understanding that this information may be publicly available.

Data availability statement

Data is available by request.

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Authors' contributions

HL and FRZ contributed to the study conception and design. Material preparation and analysis were performed by MJH, LLS, HL, and FRZ. Data collection was performed by QZ, YYL, and ZZW. The first draft of the manuscript was written by MJH, LLS, HL and all authors commented on previous versions of the manuscript. All authors read and approved the final manuscript.

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