If skin is a potential host of SARS-CoV-2, IL-17 antibody could reduce the risk of COVID-19

To the Editor: In the era of the coronavirus disease 2019 (COVID-19) pandemic, debates have emerged on whether biologics might increase the risk of contracting the disease.[1] Interleukin (IL) 17 is a biologic that is widely used in dermatology. There were reports that viral reactivation, although extremely low, could be detected during the use of IL-17 antibody (160 mg subcutaneously at Week 0, followed by 80 mg at Weeks 2, 4, 6, 8).[2] This led to concerns in using the IL-17 antibody because it was believed that it could make patients more susceptible to severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2). When we read the article by Sun et al[3] in a recently published issue, a question occurred to us: If skin is a target of SARS-CoV-2, what might be the consequence of using the IL-17 antibody?

Angiotensin-converting enzyme 2 (ACE2) is the main entrance receptor for SARS-CoV-2. Its expression is associated with the risk of making the target tissue susceptible to infection by SARS-CoV-2. Therefore, downregulating the expression of ACE2 could decrease the risk of COVID-19. To evaluate the influence of IL-17 antibody on skin ACE2 expression, we randomly selected 5 psoriasis patients who were treated with IL-17 antibody (Taltz, Eli Lilly and Company, Indianapolis, IN). The skin lesions of these patients were biopsied on week 0 and week 8 and prepared for RNA sequencing. The skin ACE2 expression of patients who underwent the antibody therapy for 8 weeks (0.36 ± 0.10; $n = 5$) was downregulated compared with that at week 0 (1.24 ± 0.50; $n = 5$), when the IL-17 antibody treatment had just begun ($P < .05$, paired t test). To confirm the result, we also selected 3 patients to compare the skin ACE2 expression at weeks 0 and 8 with immunofluorescence. Immunofluorescence staining revealed that the fluorescence intensity of ACE2 was downregulated in the skin at week 8 (0.84 ± 0.26; $n = 3$) compared with that before the IL-17 antibody treatment (9.23 ± 2.33; $n = 3$; $P < .05$; unpaired t test). Hence, either the messenger RNA or protein of ACE2 obtained from psoriasis patients can reveal that IL-17 antibody treatment remarkably reduces ACE2 expression.

Our above-mentioned work proves that IL-17 antibody treatment during the COVID-19 pandemic is not contraindicated. Elevated ACE2 expression and detection of SARS-CoV-2 in the skin[4] of COVID-19 patients implied skin was a potential host of SARS-CoV-2. After IL-17 antibody treatment, the skin ACE2 expression was downregulated, which meant IL-17 antibody could decrease the risk of COVID-19 through lessening the cells that could interact with SARS-CoV-2. Additionally, IL-17 antibody could reverse the deteriorated barrier and inflammatory status in the skin of psoriasis patients, which meant less microbe infection. Herein, the specific microbe could be SARS-CoV-2. To our knowledge, until now there has been no evidence that COVID-19 can be spread by contact with skin. However, SARS-CoV-2 could survive on skin for about 9 hours,[5] which indicates that it might be transmitted through skin in certain skin conditions such as psoriasis. Thus, whether IL-17 antibody could reduce the COVID-19 risk through reversing the inflammatory skin status with a deteriorated barrier and preventing SARS-CoV-2 transmission should be further discussed.

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