CORRESPONDENCE

Commentary on "Vogt–Koyanagi–Harada Syndrome Following COVID-19 mRNA Vaccination: Th2 to Th1 Transition-related Molecular Machinery"

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Dear Sir,

We would like to share ideas on the publication by Ikuno et al. (1). The case report describes a 58-year-old Japanese man who received a COVID-19 immunization and went on to develop Vogt–Koyanagi–Harada (VKH) syndrome, which includes vitiligo and annular erythema. The patient had symptoms related to dermatology and ophthalmology, such as erythema and depigmentation, that had not been previously documented in studies of vaccine-associated VKH syndrome. A change from Th2 to Th1 immunity was seen on immunohistochemical staining, which has not been seen in cases of vitiligo or classic VKH syndrome. The patient had a distinct reaction to the vaccine-induced VKH syndrome, as evidenced by the fact that his symptoms persisted despite treatment.

The patient's history of atopic dermatitis may have altered the immunological response to the COVID-19 vaccine, which could be a confounding issue in this case report. The ineffectiveness of the treatment and the incapacity to halt the patient's symptoms from getting worse are the study's weak points. The lack of a control group or comparison with standard VKH syndrome cases makes it difficult to completely understand how the immune response and clinical outcomes differ in this methodology. Larger sample numbers, longer follow-up times, and comparison analyses are suggested for future research in order to gain a deeper understanding of the pathophysiology and clinical characteristics of vaccine-induced VKH syndrome.

Future studies should concentrate on clarifying the exact processes behind the Th2 to Th1 shift in vaccine-induced VKH syndrome and exploring possible therapeutic approaches to stop or slow the development of dermatological symptoms in those who are affected. It would also be helpful to investigate how genetic predisposition, environmental factors, and immunological responses specific to the COVID-19 vaccine contribute to the development of VKH syndrome after vaccination. In general, more research is required to have a deeper understanding of the pathophysiology, available treatments, and long-term consequences of vaccine-associated VKH syndrome.

Reply to Commentary on "Vog–Koyanagi–Harada Syndrome Following COVID-19 mRNA Vaccination: Th2 to Th1 Transition-related Molecular Machinery"

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We are very grateful for the excellent and helpful commentary by Hinpetch Daungsupawong and Viroj Wiwanitkit. Their commentary makes several crucial points, many of which we respectfully agree with. As they pointed out previously, the immune system's response to a vaccine may vary depending on various factors (2); therefore, it is very important to know how genetic predisposition and environmental factors contribute to immunological responses, leading to the development of vaccine-induced Vogt–Koyanagi–Harada (VKH) syndrome. We are looking forward to the build-up of more precise research, leading to deeper understanding of vaccine-induced VKH syndrome.

Among the issues they pointed out, we focused on the fact that the patient has a history of atopic dermatitis (AD). The first prodromal erythema in our case appeared at the site of no atopic lesion in a patient with well-controlled AD; therefore, Th2 response in prodromal erythema is considered to be not merely residual immunity of AD, but activated immunity by vaccination. We hypothesized that the patient's history of AD may upregulate "competence of Th2 lymphocytes to vaccination", the responsiveness to the various cell-extrinsic signals (3) by vaccination, resulting in Th2-skewed prodromal immune response followed by vitiligo of VKH syndrome.

To test this hypothesis, we confirmed past history in reported cases of VKH syndrome following COVID-19 vaccination. However, most of the cases are reported in an ophthalmological journal, and there was no data on family history of atopic diseases. To understand the pathophysiology and clinical features of vaccine-induced

VKH syndrome, it would be important to examine the presence of atopic predisposition for detailed analysis.

Although the precise mechanism of Th2 to Th1 transition is undoubtedly a clue to the therapeutic approach, the prodromal atypical Th2 response, which is not seen in typical VKH syndrome or vitiligo vulgaris, would be also crucial for a better therapeutic approach in vaccine-induced VKH syndrome. By suppressing the Th2 response early in the vaccine-induced VKH syndrome, the completion of vitiligo may be prevented. For a better understanding and therapeutic approach, further studies concentrating on clarifying not only the precise mechanism of Th2 to Th1 transition, but how a Th2-skewed prodromal response occurs, are required.

REFERENCES (to both papers)

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