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A case of alopecia areata after rabies vaccination: unreported adverse effect?

Guiying Qiu[1,*], Xinrong Chen[2,*]
[1]Department of Dermatology, The Children's Hospital, Zhejiang University School of Medicine, National Clinical Research Center for Child Health, Hangzhou; [2]Department of Dermatology, Zhuji People's Hospital of Zhejiang Province, Shaoxing, China

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Corresponding author: Guiying Qiu, MS
Department of Dermatology, The Children's Hospital, Zhejiang University School of Medicine, National Clinical Research Center for Child Health, 3333 Binsheng Road, Hangzhou, Zhejiang 310052, China

*These authors contributed equally to this work.

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Alopecia areata (AA) is an autoimmune-related disorder characterized by non-scarring hair loss in children. We report the case of a child who had AA after the fifth dose of rabies vaccine and summarized various potential mechanisms of vaccination induced AA. This case indicates that rabies vaccine might be a predisposition of AA by causing immune dysregulation.

Keywords: Alopecia areata, Rabies virus, Vaccination, Pathogenic mechanisms, Psychological stress, Case reports

Introduction

Alopecia areata (AA) represents a prevalent autoimmune pathology distinguished by circumscribed, non-cicatricial alopecia. The number of vaccination-induced AA has increased recently, raising widespread concern [1]. Herein, we report the first case of a child who had AA after the fifth dose of rabies vaccine.

Case Report

A case involving a 7-year-old male patient presented to Children's Hospital of Zhejiang University School of Medicine following a canine-induced injury. The patient was administered a series of five intramuscular injections of a purified and concentrated Vero cell rabies vaccine (PVRV) over a span of 25 days as a prophylactic measure (Fig. 1). Prior to the initiation of the rabies vaccination regimen, the patient had undergone a haircut, after which both scalp and hair appeared normal. Notably, approximately 1 week subsequent to the receipt of the final PVRV dose, the patient exhibited signs of AA—initially presenting as a solitary alopecic patch on the scalp, which evolved into seven distinct areas of hair loss within the following month.

Upon physical examination, the patient was found to have multiple non-erythematous, non-scaly alopecic patches (Fig. 2A). Dermoscopic analysis revealed hallmark features of AA, including yellow dots and the characteristic "exclamation mark" hair pattern (Fig. 2B). Anamnesis conducted with the parents negated any recent medication intake or significant medical history that could be temporally associated with the onset of AA, ranging from the period of rabies vaccination to the development of the condition. Laboratory evaluations reported all trace element levels within normal limits, corroborating a diagnosis of AA.

Intervention encompassed administration of oral compound glycyrrhizin tablets at

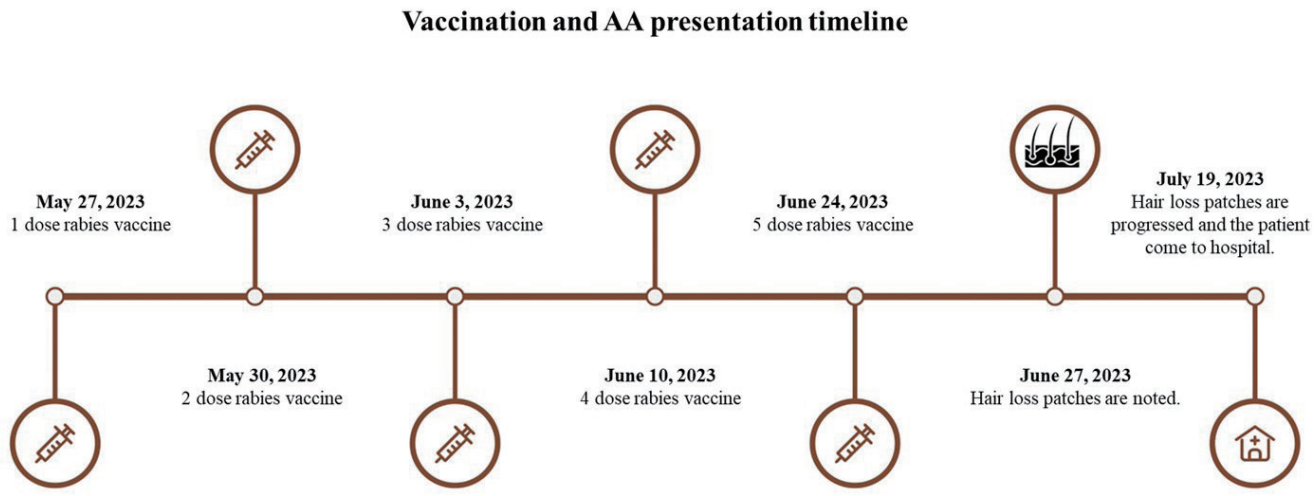

Fig. 1. Patient vaccination and alopecia areata (AA) presentation timeline.

Fig. 2. (A) Clinical examination showed multiple non-erythematous, non-scaly alopecic patches. **(B)** Dermoscopic examination demonstrated yellow dots, black dots and the characteristic "exclamation mark" hair pattern. Written informed consent for the publication of this image was obtained from the patient.

a dosage of 25 mg twice daily, vitamin D supplementation of 400 units once daily, and a daily topical application of 5% minoxidil tincture. Following 2 months of this therapeutic regimen, the patient demonstrated complete hair regrowth, evidencing a favorable response to the treatment.

A written informed consent form has been obtained from the legally authorized representative, consenting to the publication of anonymous patient information in this article.

Discussion

Several hypothetical mechanisms have been postulated in contemporary research of vaccine-associated AA, including bystander activation, molecular mimicry, genetic susceptibility, immune, neuroendocrine dynamics dysregulation and psychological stress. This hypothesis posits that vaccine adjuvants, vaccine antigens and immune and neuroendocrine dynamics dysregulation may incite an ancillary immune reaction characterized by a surge in inflammatory mediators, potentially resulting in collateral damage to hair follicle structures, such as coronavirus disease 2019, hepatitis B virus, and Tdap vaccine [1-3]. Besides, genetic susceptibility suggested that specific human leukocyte antigen alleles or immune regulating genes, may predispose individuals to vaccine-induced AA, causing recurrent AA in a child undergone distinct vaccinations [4].

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Administration of PVRV via intramuscular injection has been associated with a pronounced T helper type 2 immune induction, leading to a cascade of chemokine and cytokine release, further target hair follicles, resulting in AA [5]. However, in our case, being attacked by a dog is undoubtedly a stress event, may resulting in disturbance of endocrine, which considered to play a crucial role in AA development [6]. Given the absence of adjuvants in rabies vaccines and no familial predisposition, vaccination and emotional stress jointly cause immune dysregulation and abnormal neuroendocrine dynamics emerges as the most plausible etiology for AA. Regarding molecular mimicry, no current evidence substantiates a link between rabies vaccine components and hair follicle antigens. About the time point of the occurrence of AA, we speculate that sufficient levels of corresponding antibody titers were activated by the fifth dose of the rabies vaccine, thereby triggering a relevant immune reaction that might attack follicle cells.

Even though the psychological stress factor cannot be completely excluded, as with the vast majority of other vaccine-associated AA articles, we still report the first case of AA associated with rabies vaccination. It is imperative to underscore that inquiries into the nexus between vaccination and AA are in their nascency, and the aforementioned mechanisms are conjectural, pending empirical validation. Nevertheless, clinical evidence thus far suggests that the incidence of vaccine-induced AA is comparatively minuscule. Hence, the public health imperative of vaccination predominantly eclipses the potential for adverse autoimmune outcomes [7].

To minimize the risk of developing AA following vaccination, it is recommended that a detailed medical history be taken prior to vaccination. In individuals with a familial predilection for AA or allergic diatheses, vaccines should be administered with judicious consideration [8]. Moreover, in instances where AA symptoms manifest post-vaccination, prompt medical intervention is essential, alongside comprehensive therapeutic strategies, to manage the condition and enhance patient well-being [9].

ORCID

Guiying Qiu *https://orcid.org/0009-0001-6340-6185*
Xinrong Chen *https://orcid.org/0009-0004-9256-8214*

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