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COVID-19 vaccine-induced cutaneous lupus erythematosus

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To the Editor:

Available data suggest no excess risk of lupus erythematosus (LE) with any vaccination [1]. We report a case of cutaneous LE induced by Covid-19 vaccination.

A 70-year-old woman presented with the onset of rash, itching, arthralgias, and muscle pain 48 hours after receipt of the first dose of the adenoviral-based COVID-19 ChAdOx1 nCoV-19 vaccine. The patient did not report fever, fatigue, or other symptoms or signs of local or systemic reactogenicity and had no known allergies. She was taking ramipril for hypertension and denied using over-the-counter and recreational medications, supplements, and herbal remedies. There were no other medical diagnoses and no family history of autoimmune, inflammatory, allergic, and dermatologic diseases.

On examination, the patient was afebrile with stable vital signs. Multiple ill-defined and raised erythematous plaques were found on the trunk, buttocks, arms, and legs (**Figure 1**). She had no mucosal involvement and no signs of arthritis. Laboratory investigations showed C-reactive protein (CRP) 16mg/dl (normal: <0.6mg/dl), ferritin 1738mg/dl (normal: 10-150mg/dl), lactate dehydrogenase 837U/l (normal: 115-221U/l), myoglobin 195ug/L (normal: 25-58μg/L), creatine kinase 303U/L (normal: 39-238U/L). Blood counts, D-dimer, kidney and liver function tests, and the urinary sediment were normal. SARS-Cov-2 antigen and a reverse transcription-polymerase chain reaction were negative on a nasopharyngeal swab. Extensive evaluation for infectious causes was unrevealing. Antinuclear antibodies (ANA) with a speckled

pattern were detected at 1:1280 and complement levels were normal. Anti-dsDNA antibodies, antiphospholipid antibody panel, anti-Ro antibodies, and all other extractable nuclear antigens were within normal limits. A biopsy specimen showed epidermal hyperplasia and acanthosis, no vacuolar changes, and an epidermal and dermal inflammatory mononuclear cell infiltration with no eosinophils that was clustered around superficial blood vessels (**Figure 2**).



Figure 1. Multiple ill-defined and raised erythematous plaques on the trunk.

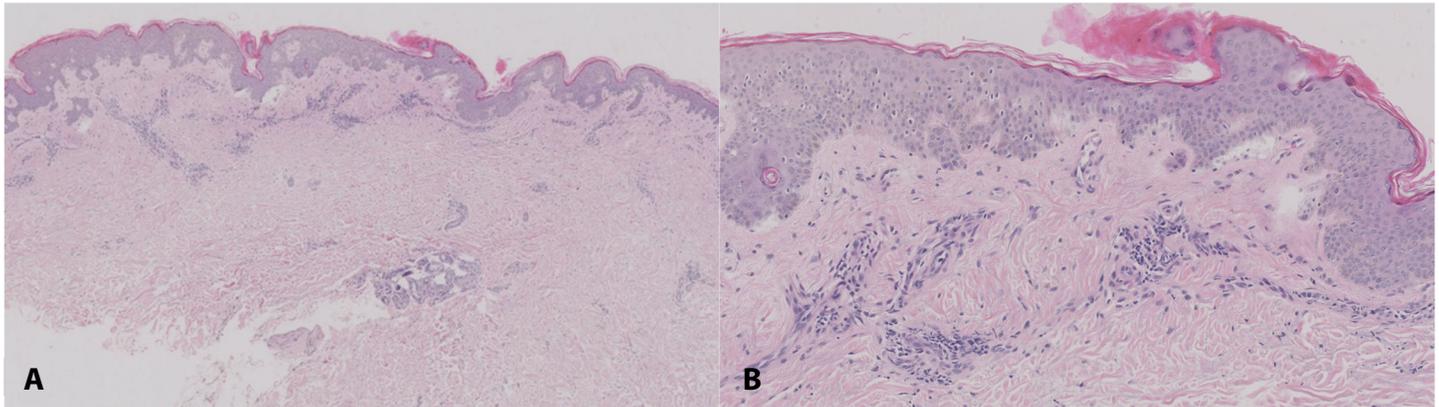


Figure 2. Histological examination shows mild irregular epidermal hyperplasia and acanthosis. Infiltrate of lymphocytes was clustered around superficial blood vessels. H&E, **A)** 40 \times ; **B)** 100 \times .

We diagnosed vaccine-induced cutaneous LE and initiated treatment with methyl prednisolone. Her condition improved and ANA titre progressively declined. At three months, the patient was fit and well while on tapering doses of corticosteroids. The rash had disappeared, ANA titre was 1:160, and CRP and muscle enzymes had returned to within the normal range. We advised against the second dose of the vaccine and the patient no longer received any other Covid-19 vaccines. By one year after initial presentation, the patient was diagnosed with polymyalgia rheumatica. At that time, there was no clinical or laboratory evidence of active cutaneous or systemic LE.

This 70-year-old, otherwise healthy woman had the onset of cutaneous LE 48 hours after receipt of the first dose of the adenoviral-based ChAdOx1 nCoV-19 vaccine. Other organs or systems were not involved. She recovered with corticosteroids and had no new cutaneous or systemic manifestations of LE at follow-up. We postulate a causal relationship with vaccination on consideration of clinical presentation, the timing of onset, and the lack of obvious alternative etiologies. The patient presented with clinical features and laboratory abnormalities characteristic of polymyalgia rheumatica one year after exposure to the vaccine, at a time she had no evidence of active cutaneous or systemic LE. In our opinion, there are no elements supporting the hypothesis that the onset of polymyalgia rheumatica one year after initial presentation should be interpreted as a delayed adverse event of the vaccination.

Cases of new-onset or relapse of a previously stable LE shortly after receipt of RNA- or adenoviral-based Covid-19 vaccines have been reported [2-9]. Progression from cutaneous into systemic LE after Covid-19 vaccination has been described in another patient [10].

The pathogenesis of Covid-19 vaccine-induced cutaneous LE is poorly understood. Host and vaccine characteristics and interactions may be at play. In our case, the adenoviral vector or other components and adjuvants of the vaccine formulation could have induced an aberrant immune response with the subsequent onset of LE. Molecular mimicry and cross-reactive antibodies may be involved. It remains difficult to distinguish between effects of patient characteristics and characteristics of components and adjuvants of COVID-19 vaccines in triggering cutaneous LE in this case. If patients with autoimmune disorders have a greater risk of developing cutaneous LE after exposure to COVID-19 vaccines is unclear. However, cutaneous LE triggered by the mRNA-based BNT162b2 vaccine has been reported in a patient with primary biliary cirrhosis [11].

This is a proof-of-concept report. The relationship between COVID-19 vaccines and the development of autoimmunity or other inflammatory disorders continues to cause controversy. Our study, however, suggests that cutaneous LE may be triggered by COVID-19 vaccines. It is a rare complication, but underlying mechanisms need to be elucidated.

Potential conflicts of interest

The authors declare no conflicts of interest.

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