COVID-19 vaccine-associated dermatomyositis

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INTRODUCTION

Dermatomyositis is a multisystem inflammatory autoimmune myopathy, often characterized by proximal symmetric muscle weakness accompanied with characteristic cutaneous findings.[1] Though the precise etiology and pathogenesis of dermatomyositis is not completely understood, environmental factors, drugs, infections, and vaccines have been implicated as potential triggers for the development of this disease. Herein, we report a case of dermatomyositis after the administration of 1 dose of the Pfizer-BioNTech COVID-19 messenger RNA vaccine.

CASE REPORT

A 77-year−old Hispanic woman, with no prior dermatologic or medical history, presented with generalized muscle aches and weakness, fever, and a moderately pruritic and painful eruption. The eruption was first noted on the upper right arm, but subsequently progressed to the left arm, chest, and neck. She had received an initial dose of the COVID-19 vaccine 5 days before the onset of these symptoms. On physical examination, violaceous, poikilodermatous scaly plaques were observed on the anterior aspect of the neck and chest (Fig 1). Additional cutaneous findings included multiple vesicles and erythematous papules on the right upper extremity and reticulated, erythematous patches on both of her thighs (Fig 2).

Laboratory evaluation revealed a creatinine phosphokinase level of 2804 IU/L (normal range, 29-168 IU/L) on the first day of presentation, which increased to 4476 IU/L over the course of 3 days. Aspartate transaminase and alanine transaminase levels were found to be elevated at 256 U/L (normal range, 5-34 U/L) and 154 U/L (normal range, 0-55 U/L), respectively. Laboratory results for hepatitis B, hepatitis C, and tuberculosis were negative. No antibodies were detected against antinuclear antibody, Jo-1, Mi2-α and Mi2-β, PL-12 or PL-7; however, anti−transcription intermediary factor 1γ antibody levels were remarkably elevated. Biopsy of the left vastus lateralis muscle revealed overexpression of major histocompatibility complex class I, inflammatory cells, necrotic fibers, and atrophic fibers organized in a perimysial pattern, suggesting an immune-mediated myopathy of a dermatomyositis type. A 3-mm punch biopsy specimen of the skin of the right upper extremity revealed features of interface dermatitis with superficial perivascular mononuclear inflammation and dermal edema. The epidermis showed basal vacuolar alteration with foci of necrotic keratinocytes. Colloidal iron staining highlighted increased dermal mucin. The histologic features, laboratory findings, and clinical presentation together suggested a diagnosis of dermatomyositis, occurring in association with the COVID-19 vaccine. Given the presence of anti−transcription intermediary factor 1γ antibodies and their association with malignancy in the setting of dermatomyositis, the patient underwent cancer screening. A lymphoma disorder profile by flow cytometry was unremarkable. Contrast-enhanced computed tomography imaging of the chest, abdomen, and pelvis did not reveal any internal malignancy.

After receiving treatment with 40 mg of intravenous methylprednisolone for 3 days and 2 g/kg of intravenous immune globulin over 5 days, the patient showed significant clinical and laboratory improvement. She regained proximal muscle strength, and her creatinine phosphokinase level decreased significantly to 1135 IU/L. She was subsequently transitioned to oral prednisone 60 mg taper for 4 weeks, along with 1 g of oral

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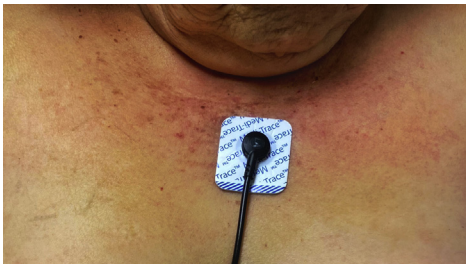

Fig 1. Violaceous, poikilodermatous, scaly plaques on the anterior aspect of the neck and chest.

mycophenolate mofetil twice a day. Four weeks after her initial presentation, the patient had marked improvement in muscle strength. Twelve weeks after initial presentation, laboratory studies showed normalization of liver enzymes and creatinine phosphokinase levels.

DISCUSSION

Although the precise etiology of dermatomyositis is unknown, several immune mechanisms have been postulated to underlie the pathogenesis of the disease. Dermatomyositis is thought to be a complement-mediated microangiopathy, which results in the destruction of capillaries, decreased perfusion, and inflammatory cell stress within perifascicular regions.[2] Activation of complement may also lead to cytokine and chemokine release, prompting the recruitment of cytotoxic $CD4^+$ T cells and macrophages to the affected muscle tissue.[1]

Several vaccines and viruses represent environmental factors that have been implicated as triggers for the development of dermatomyositis and other inflammatory myopathies.[3] Interestingly, dermatomyositis has not been historically associated with increasing incidence after large vaccination campaigns.[3] There are very few overall reported cases of vaccine-associated dermatomyositis in the medical literature. Cases of dermatomyositis occurring after vaccination for bacillus Calmette–Guérin, influenza, tetanus, and hepatitis B virus have been sporadically reported; however, mass vaccination efforts have not been associated with an increase in the incidence of dermatomyositis.[4-6]

Vaccination may induce the development of dermatomyositis through robust immune system activation, resulting in immune disturbances and dysregulation, manifesting as autoimmune disease.[7] The development of autoimmune conditions, including Kawasaki disease, autoimmune myositis, and dermatomyositis after SARS-CoV-2 infection itself have been sporadically reported.[8] The mechanism by which autoimmune conditions develop after vaccination is thought to be analogous to those occurring after natural infections.[2] Thus, the mechanism is thought to involve molecular mimicry, epitope spreading, bystander activation, release of cryptic epitopes, reactivation of memory T cells, activation of superantigens, or direct inflammatory damage resulting in the release of autoantigens.[2] The identification of 3 immunogenic epitopes in patients with dermatomyositis with high sequence identity to SARS-CoV-2 proteins suggests an overlapping mechanism of immune pathogenesis.[9] These immunogenic epitopes with high sequence identity also serve as targets for vaccine development. Adjuvant incorporation into vaccine formulations serves as an immune stimulus that increases antigen recognition, T cell stimulation, and release of chemokines and inflammatory cytokines.[2] Adjuvants may serve as the common culprit, accounting for the development of autoimmune conditions after immunization with varying vaccines; however, the authorized messenger RNA vaccines against SARS-CoV-2 do not contain adjuvants. Therefore, the development of autoimmune conditions after vaccination with 1 of these vaccines is likely a consequence of the vaccine itself.

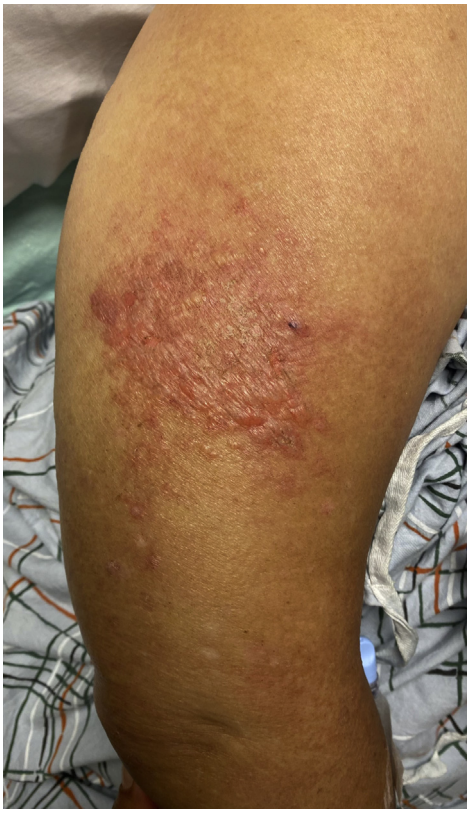

Fig 2. Vesicles and erythematous papules on the right upper extremity.

Our patient had no prior history of dermatomyositis or underlying malignancy and was not exposed to other environmental triggers or medications that may have led to the onset of the disease. Therefore, we believe that vaccination with the COVID-19 messenger RNA vaccine contributed to the development of her dermatomyositis. We present this case to inform dermatologists of this possible association and to highlight the importance of obtaining a vaccination history in a patient with findings suggestive of an inflammatory myopathy. Further investigation into the pathologic mechanism underlying vaccine-associated myopathies is necessary to guide clinical decisions regarding the administration of second vaccine doses or boosters in such patients.

Conflicts of interest

None disclosed.

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