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Case Report

Subacute Cutaneous Lupus Erythematosus Following COVID-19 Vaccination: A Case Report after BNT162b2 Vaccine and Literature Review.

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ABSTRACT

Background: COVID-19 vaccines have demonstrated a favorable safety profile in diverse populations, including patients with autoimmune rheumatic diseases. However, rare adverse events such as disease flares and vaccine-triggered autoimmune manifestations have been reported. This study presents a case of subacute cutaneous lupus erythematosus (SCLE) following BNT162b2 vaccination and a narrative review of the literature.
Methods: We describe the case of an 80-year-old woman with noninfectious aortitis who developed SCLE, annular-polycyclic type, following BNT162b2 vaccination and review published cases of SCLE after anti-SARS-CoV-2 vaccination.
Results: A review of 18 published cases and 19 total, including the current case of SCLE following anti-SARS-CoV-2 vaccination, suggests that most reported patients were adult women, with and without underlying autoimmune rheumatic diseases. SCLE typically develops within days of the first vaccine dose, with most cases following mRNA-based vaccination.
Conclusions: Although based on a limited number of case reports and subject to reporting bias, these findings underscore the potential for anti-SARS-CoV-2 vaccines to trigger autoimmune cutaneous reactions not exclusively in predisposed individuals, emphasizing the need for vigilance while reinforcing the overall safety and benefit of vaccination in patients with autoimmune rheumatic diseases. The incidence of SCLE following COVID-19 vaccination, and the causal relationship between vaccination and SCLE, cannot be inferred from case reports or pharmacovigilance data alone.

1. Introduction

mRNA COVID-19 vaccines against SARS-CoV-2 have changed the course of the COVID-19 pandemic and have proven effective in preventing severe disease [1]. They have demonstrated an acceptable safety profile across diverse populations, including individuals with autoimmune rheumatic diseases [2–5]. Nevertheless, rare immune-mediated complications have been reported, including disease flares and the emergence of new autoimmune manifestations post-vaccination [6–8]. These events, while infrequent, underscore the need for heightened clinical awareness, particularly in predisposed individuals with underlying autoimmunity. In this context, we describe a case of subacute cutaneous lupus erythematosus (SCLE) in an elderly patient with a history of aortitis and subclinical autoimmunity, occurring after administration of the first dose of an mRNA COVID-19 vaccine. We conducted a literature review of cases of SCLE following anti-SARS-CoV-2 vaccination (not limited to the mRNA vaccine platform). We summarized the clinical characteristics, with particular attention to patients with autoimmune rheumatic diseases.

2. Materials and Methods

A novel case report is presented. A narrative literature search was conducted in PubMed/Medline, Web of Science, Scopus, and the Cochrane Library. The search was conducted on May 17, 2025, covering publications through April 30, 2025. The following keywords were used: ("mRNA COVID-19 vaccine" OR "mRNA anti-SARS-CoV-2 vaccine" OR "COVID-19 vaccination" OR "COVID-19 vaccine" OR "mRNA vaccine" OR "anti-SARS-CoV-2 vaccine") AND ("annular skin eruption" OR "annular skin rash" OR "annular plaques" OR "subacute cutaneous lupus erythematosus" OR "SCLE" OR "cutaneous lupus erythematosus" OR "systemic lupus erythematosus" OR "SLE" OR "Rowell's syndrome"). The search was conducted in each database using the combined string and was limited to English-language articles. Deduplication was performed using reference management software to remove overlapping records across databases. Relevant, referenced, and cited articles were also reviewed and included. Inclusion criteria were case reports or series describing SCLE temporally associated with COVID-19 vaccination that provided adequate clinical details. Specifically, SCLE had to be new-onset or a flare and temporally linked to COVID-19 vaccination. Cases of CLE/SLE without SCLE were excluded. A PRISMA-style flow diagram was used to document the number of records identified, screened, excluded, and included **(Supplementary Figure 1)**. Individual-level data extracted included patient number, age, gender, history of autoimmune rheumatic disease or other

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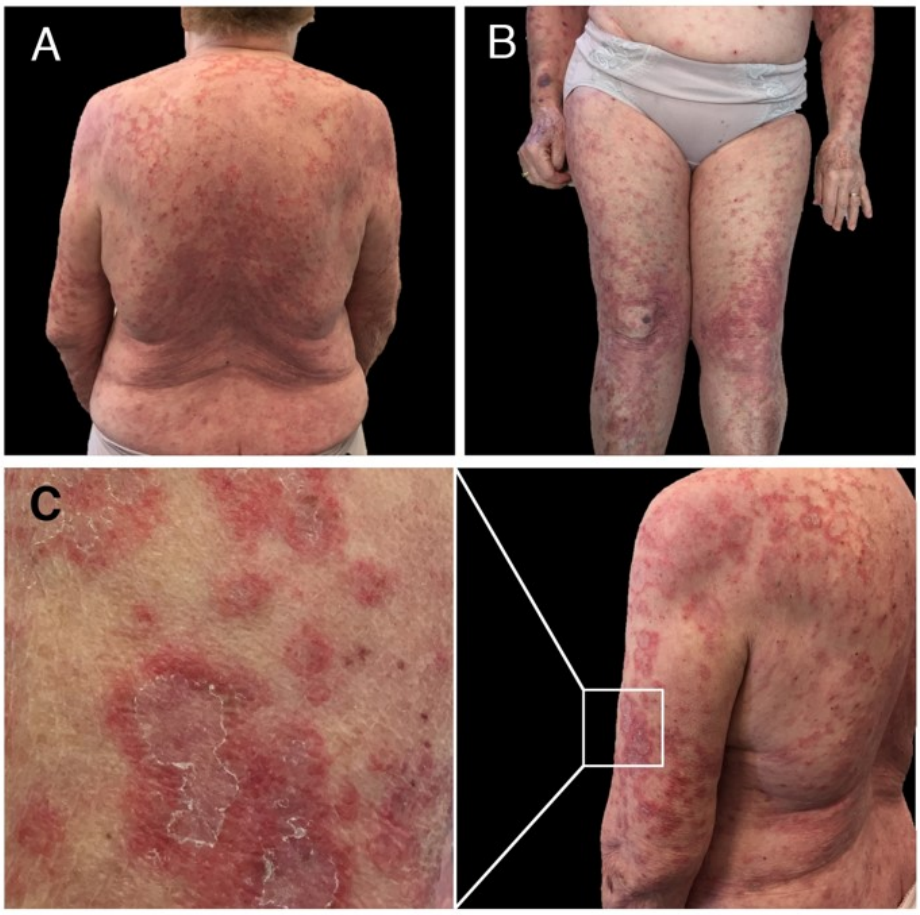

Figure 1: Clinical findings of the patient two months after the onset of subacute cutaneous lupus erythematosus. (A-B) Multiple annular erythematous plaques located on the back and on the extremities of this 80-year-old woman. (C) Magnification of annular-polycyclic plaques on the left arm.

significant conditions, interval between vaccination and the onset of skin lesions, type of anti-SARS-CoV-2 vaccine administered, SCLE presentation, treatment, and rechallenge. Descriptive statistics (mean ± SD) were calculated using spreadsheet software. Data extraction was performed by one reviewer (L.S.) and independently verified by a second reviewer (D.C.); disagreements were resolved by consensus. The quality of the included case reports was not formally assessed, as validated tools are not well suited to case reports; all eligible reports were included in the descriptive synthesis.

3. Results

3.1. Case Report

An 80-year-old woman presented with a progressively worsening skin eruption involving the face, neck, shoulders, trunk, and upper and lower limbs over the course of two months. Notably, the onset of cutaneous manifestations occurred six days after administration of the first dose of the BNT162b2 mRNA COVID-19 vaccine. She had no history of SARS-CoV-2 infection. Her past medical history was significant for non-infectious aortitis diagnosed nine months prior, complicated by type A aortic dissection requiring surgical intervention. She had been maintained on subcutaneous tocilizumab therapy (162 mg every week) for 7 months with stable disease control. She also had a history of arterial hypertension and dyslipidemia, and had undergone total right knee arthroplasty two years earlier. Her medications included tocilizumab, pantoprazole, furosemide, potassium canrenoate, nebivolol, allopurinol, atorvastatin, lorazepam, enoxaparin, and acetylsalicylic acid. Except for tocilizumab, all the others were long-standing therapies with no recent dose changes. She had never smoked. At the time of her initial rheumatologic evaluation, serologic testing had revealed a positive antinuclear antibody (ANA) titer (1:640) with a fine speckled pattern, along with positivity on immunoblotting of anti-SSA (+++), anti-Ro52 (+++), and anti-SSB/La (++) antibodies. Despite these findings, the patient had never shown clinical signs suggestive of a connective tissue disease. On physical examination, multiple intensely pruritic annular erythematous patches were observed, coalescing into polycyclic plaques with fine desquamation and partial central resolution **(Figure 1)**. The lesions were symmetrically distributed over the neck, shoulders, trunk, and extremities. Some lesions had a targetoid appearance. A punch biopsy of a rounded, violaceous, and desquamating plaque on the right shoulder was performed two months after the onset of the skin rash. Histological examination revealed superficial perivascular dermatitis, capillary plexus dilatation, and associated solar elastosis. She reported no fever or arthralgias. No mucosal involvement occurred. The patient reported no sun exposure in the days before and following vaccination. She routinely used photoprotection. Laboratory tests confirmed strongly positive anti-Ro60 (410 U/mL, normal < 10 U/mL) and anti-Ro52 (256 U/mL, normal < 10 U/mL) autoantibodies and decreased serum complement 3 (C3, 0.68 g/L, reference range 0.9-1.8 g/L) and 4 (C4, 0.08 g/L, reference range 0.1-0.4 g/L). Previous

testing of complement levels had shown hypocomplementemia. Anti-dsDNA, anti-Sm, anti-RNP, anti-cardiolipin, anti-beta-2 glycoprotein I antibodies, and lupus anticoagulant were negative. Complete blood count with differential, serum creatinine and urinalysis, liver function tests, and inflammatory markers (ESR and CRP) were all within normal limits, and review of cardiopulmonary and other systemic symptoms revealed no novel abnormalities, effectively excluding systemic involvement. Based on morphology, distribution, immunologic profile, and temporal association with vaccination, a clinical diagnosis of subacute cutaneous lupus erythematosus (SCLE), annular-polycyclic type, was made. The 2019 ACR/EULAR systemic lupus erythematosus (SLE) classification criteria were not fulfilled [9]. The patient was started on oral prednisone 25 mg/day to rapidly control inflammation, with a gradual taper over approximately 20 weeks, resulting in a slow but progressive resolution of skin lesions after almost six months from disease onset **(Supplementary Figure 2)**. Partial resolution of erythema and pruritus occurred over 12-16 weeks with residual hypopigmentation without scarring and complete resolution after 22 weeks from disease onset **(Supplementary Figure 2)**. Topical corticosteroids were avoided due to her thin, fragile skin, which increased the risk of local atrophy and irritation. Hydroxychloroquine was not initiated because the patient was already receiving tocilizumab, which was continued after SCLE developed, providing ongoing immunomodulatory control. Photoprotection measures, including avoidance of direct sunlight and use of sunscreen, were recommended to reduce UV-induced exacerbation. Following the initial reaction, the second dose of the anti-SARS-CoV-2 vaccine was withheld after full resolution. The patient remains under surveillance for any evolution toward systemic disease, though currently there is no evidence of SLE involvement at 4 years of follow-up.

3.2. Review of the Literature

19 patients with SCLE following anti-SARS-CoV-2 vaccination were identified, and main clinical and demographic findings are summarized in **(Table 1)** [10–26]. The mean age was 52.6 ± 22.5 years, and patients were predominantly female (13/19). In 42% (8/19) of the cases, there was a history of prior autoimmune rheumatic disease (SCLE, SLE, Sjögren's syndrome, mixed connective tissue disease, aortitis, or primary biliary cholangitis). New-onset SCLE cases following SARS-CoV-2 vaccination were more frequent than flares of pre-existing disease **(Supplementary Table 1)**. Flares typically occur within 1 week of vaccination, whereas new-onset cases can occasionally manifest later, up to 22 days following the second vaccine dose **(Table 1)**. The onset of SCLE manifestations typically occurred within days following the first or, less commonly (7/19 cases), the second vaccine dose. In 2 cases, cutaneous manifestations that already appeared after the first vaccine dose worsened following administration of the second dose [10, 21]. No differences in presentation were observed when cases were stratified by latency period **(Supplementary Table 2)**. The anti-SARS-CoV-2 vaccines administered were primarily mRNA-based (BNT162b2 and mRNA-1273) in 15 cases (79%), whereas 3 cases (16%) were reported after administration of the vector-based AZD1222 vaccine [10, 11, 15] and 1 case (5%) after administration of the Vero cell-inactivated virus vaccine [25]. The timing of onset was similar across vaccine types, although the small sample size precludes a formal statistical comparison **(Supplementary Table 3)**.

Only 21% (4/19) of patients later developed SLE **(Supplementary Table 4)**. In the four reported cases [13, 15, 23, 24], SLE developed within days to 2 weeks after SARS-CoV-2 vaccination; all patients had cutaneous involvement, three had musculoskeletal symptoms, and systemic disease was limited to mild hematologic and serologic abnormalities; no renal, neurologic, or cardiopulmonary involvement was observed, and all patients improved with standard immunomodulatory therapy. Morphologically, lesions manifested as annular-polycyclic (more commonly) and/or papulosquamous, with one case diagnosed as Rowell's syndrome. The trunk (particularly the upper torso) and extremities were the most commonly affected sites, with a few cases also involving the face [13, 14, 23, 26]. Notably, exclusive facial involvement was reported in only one case [25].

Systemic glucocorticoids were the predominant therapy, used either alone or in combination, in the majority of cases. Topical therapies, including corticosteroids and tacrolimus, were administered in only 52% of cases. Hydroxychloroquine was continued or initiated and generally combined with steroids and other immunosuppressors, including mycophenolate mofetil and azathioprine. Nonsteroidal anti-inflammatory drugs (i.e., etoricoxib, a selective cyclooxygenase-2 inhibitor) were used as adjunctive therapy in a few cases [20, 23]. Administration of a subsequent vaccine dose following SCLE was reported in only 4 cases [14, 15, 19, 20], with the majority tolerating rechallenge well **(Table 1)**.

4. Discussion

Cutaneous lupus erythematosus (CLE) can occur as an isolated skin disease or in the context of SLE. CLE is classified into three major subtypes: acute (ACLE), subacute (SCLE), and chronic (CCLE) [27]. These subtypes are distinguished based on clinical presentation, lesion duration, and histopathological features. SCLE occurs more frequently in adult women and presents with diverse clinical variants, including annular-polycyclic, papulosquamous, mixed, and rare forms (i.e., exanthematous, pityriasiform, exfoliative erythroderma, follicular erythematous, acral annular, poikilodermatous) [28–30]. The initial lesions typically present as erythematous papules or macules measuring 1-3 mm in diameter, with mild infiltration and superficial desquamation, occasionally with a purpuric component [31]. As the disease progresses, lesions evolve into plaques with increased infiltration, centrifugal enlargement, and central resolution (annular-polycyclic form) and/or are primarily characterized by infiltration and scaling (papulosquamous or psoriasiform type) [32]. Healing occurs without scleroatrophy, but it may result in long-lasting or permanent hypochromic changes [33]. Lesions are symmetrically distributed predominantly on sun-exposed areas (upper chest, back, upper limbs), although facial involvement is uncommon. Photosensitivity affects the majority of patients [29]. The disease typically follows a chronic, relapsing course, and exacerbations are often observed in late spring [34]. Ultraviolet light serves as the principal triggering factor, followed by drugs [29, 35]. In 1985, the first cases of drug-induced subacute cutaneous lupus erythematosus (DI-SCLE) linked to hydrochlorothiazide therapy were identified, and since then, over 40 drugs have been recognized as triggers, with new cases continuing to emerge annually [36]. An interferon (IFN)-driven cytotoxic anti-epidermal immune response, commonly referred to as interface dermatitis, characterizes skin lesions in CLE [37, 38]. Given that mRNA vaccines exert part of their immunologic effect through the induction of type I IFNs, it has been hypothesized that anti-SARS-CoV-2 mRNA vaccination may potentially induce or exacerbate SCLE via IFN-orchestrated mechanisms [20]. Current data do not allow definitive mechanistic attribution to mRNA-induced IFN responses.

After accounting for potential underreporting, only 19 cases of SCLE following anti-SARS-CoV-2 vaccination were identified in the literature through April 2025. Given that more than 13 billion COVID-19 vaccine doses have been administered globally, the limited number suggests that such an occurrence is exceedingly rare,

Table 1: Main clinical features of 19 patients with SCLE following anti-SARS-CoV-2 vaccination (up to April 2025).

Authors, Year [Ref.] (PMID)	No. of Patients	Age	Gender	History of ARD (or other relevant)	Time from vaccination to onset of skin lesions	Type of anti-SARS-CoV-2 vaccine	SCLE presentation; form	Treatment	Rechallenge
Gambichler et al. 2021 [12]	1	74	F	None (severe dementia)	1 day post 1d	BNT162b2	New-onset; RS	PDN	NR
Niebel et al. 2021 [19]	1	73	F	SCLE	10 days post 1d	BNT162b2	Exacerbation; PS form	PDN	Yes, well tolerated
Kreuter et al. 2021 [15]	1	62	F	SCLE	10 days post 1d	AZD1222	Exacerbation; SLE; PS form	PDN	Yes, well tolerated
Joseph et al. 2021 [14]	1	54	F	SCLE	4 days post 1d	mRNA-1273	Exacerbation; AP form	HCQ continued, MMF	Yes, mild facial flare
Liu et al. 2021 [17]	1	70	M	None (lung cancer)	10 weeks post 2d	BNT162b2	New-onset, PS form	tCS	NR
Kreuter et al. 2022 [16]	1	79	M	None	10 days post 1d	BNT162b2	New-onset, AP/PS form	sCS, HCQ	NR
Niebel et al. 2022 [20]	2	41	M	MCTD	4 days post 1d	BNT162b2	New-onset, AP form	PDN, tCS	Yes, well tolerated
		22	F	None (hypothyroidism)	10 days post 1d	mRNA-1273	New-onset, AP form	PDN, tCS, NSAID	No
Zengarini et al. 2022 [26]	1	30	F	PBC	10 days post 2d	BNT162b2	New-onset; PS form	PDN, tCS	NR
Rechtien et al. 2022 [21]	1	24	F	SLE	12 days post 1d, worsening 4 days post 2d	BNT162b2	New-onset; PS form	sCS, tCS; AZT increased	NR
Voisin et al. 2022 [24]	1	20	F	None	NR post 2d	BNT162b2	New onset; SLE; AP form	HCQ, sCS	NR
Bakr et al. 2022 [10]	1	51	M	SLE, Sjogren's syndrome	7 days post 1d, worsening 4 days post 2d	AZD1222	Exacerbation; AP form	HCQ; AZT increased	NR
Sagy et al. 2022 [23]	1	24	M	None	3 days post 1d	BNT162b2	New-onset; SLE; PS form	HCQ, tCS, NSAID	NR
Rimmer et al. 2023 [22]	1	79	F	None (hypothyroidism, essential thrombocytosis)	2 weeks post 2d	mRNA-1273	New-onset; AP form	PDN, HCQ, MMF, tCS, IVIG	NR
Magnaterra et al. 2023 [18]	1	73	M	None	22 days post 2d	BNT162b2	New-onset; AP form	tCS	NR
Wang et al. 2023 [25]	1	48	F	None	10 days post 2d	BBIBP-CorV	New-onset; AP form	HCQ, tTC	NR
Famularo et al. 2023 [11]	1	70	F	None	2 days post 1d	AZD1222	New-onset, AP form	MPDN	No
Hansen et al. 2023 [13]	1	26	F	None	14 days post 2d	NR mRNA-based	New-onset; SLE; PS form	MPDN, HCQ, belimumab, tTC	NR
Salvati et al. 2025; Current case report	1	80	F	Aortitis	6 days post 1d	BNT162b2	New-onset, AP form	PDN	No

ARD, autoimmune rheumatic disease; AP, annular/polycyclic; AZT, azathioprine; d, vaccine dose; F, female; HCQ, hydroxychloroquine; IVIG, intravenous immunoglobulin; M, male; MCTD, mixed connective tissue disease; MMF, mycophenolate mofetil; NSAID, nonsteroidal anti-inflammatory drug; NR, not reported; PBC, primary biliary cholangitis; PS, papulosquamous; RS, Rowell's syndrome; SCLE, subacute cutaneous lupus erythematosus; PDN, prednisolone or prednisone; MPDN, methylprednisolone; SLE, systemic lupus erythematosus; sCS, systemic corticosteroid; tCS, topical corticosteroid; tTC, topical tacrolimus; 1d, first vaccine dose; 2d, second vaccine dose.

although significant underreporting is likely [36, 39]. Between 2020 and April 2025, a total of 801 cases of SCLE were reported to the U.S. Food and Drug Administration Adverse Event Reporting System (FAERS); of these, only 4 (0.5%) were associated with the mRNA-1273 vaccine [40]. Pharmacovigilance data provide additional context but must be interpreted with caution. Spontaneous reporting systems are subject to substantial underreporting, reporting bias, and lack denominators; therefore, they cannot be used to estimate incidence or establish causality. These data should be interpreted as supporting the qualitative impression that vaccine-associated SCLE is rare, rather than as quantitative risk estimates. Annual incidence of SCLE is 0.63-0.7 per 100000 person-years [41, 42]. Consistent

with the epidemiology of DI-SCLE [36, 40], adult females were predominantly affected. Cases were observed independently of prior autoimmune rheumatic disease; differentiating between latent or induced presentations might be critical since exacerbations are often associated with a more complex disease progression compared to inductions, usually following a more favorable outcome [43]. The onset of SCLE commonly occurred within days of the first dose of primarily mRNA-based anti-SARS-CoV-2 vaccines. Our patient fits well within the typical clinical and serological spectrum of SCLE. She was an adult woman who developed symmetrically distributed annular and papulosquamous lesions predominantly affecting sun-exposed areas and who tested positive for anti-Ro/SSA antibodies, a serologic hallmark frequently associated with SCLE. The absence of scarring and the favorable response to therapy further support this classification. These features are consistent with the classic SCLE phenotype described in large clinical series. Beyond this typical presentation, several patient-specific factors may have contributed to disease susceptibility. The presence of pre-existing anti-Ro/SSA antibodies is a known risk factor for both idiopathic and drug-induced SCLE and may reflect an underlying, preclinical autoimmune predisposition. In addition, the patient's history of aortitis suggests a background of immune dysregulation. Whether tocilizumab, an anti-IL-6 receptor monoclonal antibody, mitigated disease severity or modified the risk of progression to systemic lupus erythematosus in this patient remains speculative, as well as other immunosuppressive treatment in some reported cases. With regard to triggering factors, in this case, the eruption did not coincide with reported excessive UV exposure or seasonal peak sunlight intensity, and no newly introduced medications - apart from vaccination - were identified preceding disease onset. Histological examination of the skin biopsy revealed superficial perivascular dermatitis, dilatation of the capillary plexus, and associated solar elastosis. Although these findings are non-specific and were obtained two months after the onset of the initial rash, while the patient was receiving systemic glucocorticoid therapy **(Supplementary Figure 2)**, typical histopathologic features of SCLE include interface dermatitis with vacuolar alteration of the basal layer, apoptotic keratinocytes, dermal mucin deposition, and follicular plugging. In the present case, the diagnosis of SCLE was clinico-serologic, with supportive but non-diagnostic histology. In addition, direct immunofluorescence (DIF) was not performed because it was unavailable at the time of biopsy, which represents a limitation and further underscores that the diagnosis in this case was established on a clinico-serologic basis. It is important to note that our patient was receiving several medications at the time of SCLE onset, including antihypertensive agents and proton pump inhibitors, which have been associated with DI-SCLE. However, these were long-standing therapies with no recent dose changes, making them a less likely cause of SCLE onset. Moreover, all concomitant medications were continued without modification during the clinical course. To further contextualize this observation, a structured causality assessment was considered. Applying a narrative approach based on the Naranjo Adverse Drug Reaction Probability Scale, a temporal relationship between BNT162b2 vaccination and the onset of SCLE was observed [44]. Clinical improvement occurred following standard dermatologic treatment and in the absence of further vaccine exposure, supporting a positive dechallenge; however, rechallenge was not performed. Importantly, alternative explanations, including DI-SCLE, cannot be definitively excluded. The resulting Naranjo score of +4 corresponds to a "possible" association, indicating a temporal relationship without establishing causality. These findings should therefore be interpreted with caution within the inherent limitations of a single case report. When compared with previously published reports of vaccine-associated SCLE **(Table 1)**, the timing and disease course in our patient are concordant. Across reported cases, cutaneous manifestations typically developed within days to a few weeks after vaccination, most often after the first dose, and were generally limited to the skin with or without mild systemic or serological activity. Progression to SLE appears to be uncommon. Clinical outcomes are favorable, either with topical therapy alone or with standard systemic immunomodulatory treatment, consistent with the benign course observed in our patient. The absence of rechallenge in most reports limits the ability to establish causality definitively. For patients with pre-existing autoimmune diseases or autoantibody positivity, SCLE risk following COVID-19 vaccination remains extremely low. Vaccination should not be withheld; however, patients should be counseled on the rare risk of cutaneous reactions and advised to report any new rashes promptly. For individuals who develop SCLE after a first dose, shared decision-making regarding subsequent doses should take into account disease severity, alternative vaccine platforms, and the established benefits of COVID-19 vaccination. This study has several limitations: it describes a novel single patient, and the accompanying literature review is restricted to peer-reviewed English-language publications, excluding grey literature. It is important to note that the temporal association between vaccination and SCLE onset in these case reports may partly reflect heightened surveillance and reporting following vaccination campaigns (Weber effect), which can create apparent clustering even for coincidental events. Moreover, direct immunofluorescence was unavailable, and conclusions drawn from pharmacovigilance databases, such as FAERS, are inherently constrained by reporting bias and incomplete clinical information.

5. Conclusions

Anti-SARS-CoV-2 vaccines have been temporally associated with rare cases of autoimmune cutaneous reactions, including SCLE, in both predisposed and apparently non-predisposed individuals. This underscores the importance of clinical vigilance while reaffirming the overall safety and benefit of vaccination in patients with autoimmune rheumatic diseases. Given the small number of cases and the observational nature of the available data, further reports and registries are needed to better characterize risk factors and outcomes. With approximately 19 documented cases among over 13 billion doses administered globally, vaccine-associated SCLE represents an exceptionally rare event that should not alter vaccination recommendations for patients with autoimmune rheumatic diseases.

Conflicts of Interest

The authors declare no conflicts of interest.

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Informed Consent

Informed consent was obtained from the patient for participation in this report.

Large Language Model

None.

Authors Contribution

LS and DC conceptualized and wrote the manuscript. MDC and PP reviewed and edited the manuscript. All authors have read and agreed to the published version of the manuscript.

Data Availability

All available data are within the manuscript and the supplementary file.

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