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Systematic Review

Statins and the skin: systematic review of dermatologic reactions to statin therapy

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ABSTRACT

Statins are widely prescribed medications for lowering cholesterol levels. While generally well-tolerated, they can cause various cutaneous adverse reactions through disruption of the mevalonate pathway, affecting immune cell function and potentially triggering autoimmune responses. Aim was to systematically evaluate the clinical and histopathologic features of statin-associated cutaneous adverse reactions and review reported management strategies. A systematic review following PRISMA guidelines using PubMed, Embase, and Scopus databases was conducted analyzing cutaneous adverse reactions associated with statin therapy, including clinical presentation, histopathologic features, and management strategies. Inclusion criteria encompassed case reports, randomized controlled trials, and observational studies investigating statin-induced cutaneous adverse effects in human subjects. Studies were excluded if they were animal trials, systematic reviews, literature reviews, or did not focus on statin-related cutaneous reactions. The 22 published cases were identified, affecting patients with a mean age of 61 years (range: 44-84) and slight female predominance. The most frequently reported reactions were eczematous eruptions (6 cases), lichenoid drug eruptions (4 cases), dermatomyositis (DM) (4 cases), and pityriasis lichenoides chronica (PLC) (3 cases). Additional manifestations included lupus erythematosus tumidus (LET), pemphigus erythematosus, erythema multiforme, fixed drug eruption, and thrombocytopenic purpura. Histopathologic features commonly showed immune-mediated patterns including interface dermatitis, basal vacuolar changes, and lymphocytic infiltrates. Most cases resolved following statin discontinuation, though some required adjunctive corticosteroids or immunosuppressive therapy. This review adds to the body of literature by highlighting the breadth of statin-associated cutaneous adverse effects, emphasizing the need for clinicians to maintain a high index of suspicion when evaluating new skin eruptions in patients on statin therapy. Prompt identification and tailored management can lead to improved patient outcomes and medication safety.

Keywords: Statins, Cutaneous adverse drug reactions, Lichenoid drug eruption, Dermatomyositis

INTRODUCTION

Statins, also known as HMG-CoA reductase inhibitors, are a class of drugs primarily used to lower low-density lipoprotein cholesterol (LDL-C) levels in the blood. They achieve this by competitively inhibiting the enzyme HMG-CoA reductase, which is crucial in the cholesterol biosynthesis pathway.[1,2]

Despite being generally well tolerated, statins have been shown to cause various cutaneous adverse effects including lichenoid drug eruption, DM, and erythema multiforme.[3-5] Statins may induce adverse cutaneous reactions due to their mechanism of action. By inhibiting HMG-CoA reductase, statins disrupt the mevalonate pathway, reducing the production of isoprenoids, which are crucial for immune cell function.[6] This disruption affects small GTPases involved in immune responses, potentially triggering inflammation and immune dysregulation.[7] Additionally, statins can downregulate adhesion molecules and pro-inflammatory cytokines, further altering immune responses and increasing the risk of autoimmune conditions like lupus and DM.[7,8] The goal of this systematic review is to provide a comprehensive analysis of the cutaneous adverse reactions associated with statin use, including their clinical presentation, dermatopathologic features, and management strategies. By synthesizing available evidence, this review aims to increase awareness of these reactions, assist in early diagnosis, and guide appropriate treatment approaches.

METHODS

A systematic review was conducted following the preferred reporting items for systematic reviews and meta-analyses (PRISMA) guidelines (Figure 1). A comprehensive literature search was performed using PubMed, Embase, and Scopus databases. The search strategy included a combination of Medical Subject Headings (MeSH) and free-text terms related to "Statin" and "lichen planus," "Atorvastatin" and "lichen planus," "Statin-induced Cutaneous Reactions," "statin-induced lichen planus," "hmg-coa reductase and adverse drug reactions," "interface dermatitis" and "statin," and

"Atorvastatin-induced DM." Studies were included if they met the following criteria: 1) investigated cutaneous adverse effects of statins, 2) included case reports, randomized controlled trials (RCTs), or observational studies, 3) reported clinical outcomes, efficacy, or safety data related to statin-induced cutaneous reactions, 4) included human subjects, and 5) were published in English.

Twenty-two articles met the inclusion criteria. Studies were excluded if they were animal trials, systematic reviews, literature reviews, or did not focus on statin-related cutaneous reactions. Two independent reviewers screened titles and abstracts for eligibility, followed by full-text review, with discrepancies resolved by consensus.

This resulted in 22 unique studies. Data extraction included study design, sample size, statin treatments, outcome measures, efficacy results, and adverse events. The risk of bias was assessed using the GRADE guidelines. Due to the heterogeneity of study designs and the outcome measures, a qualitative synthesis was performed.

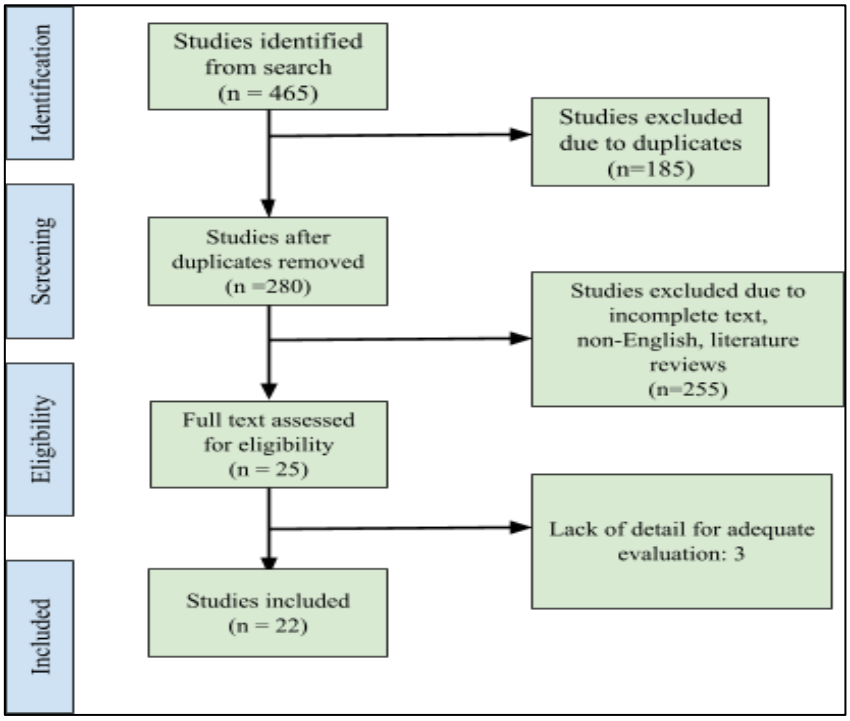


Figure 1: PRISMA flow diagram of study selection process.

*This figure illustrates the systematic review screening process using the PRISMA guidelines. A total of 465 records were initially identified through database searches. After removing 185 duplicates, 280 studies remained. Of these, 255 were excluded based on incomplete text, language barriers, or being review articles. The full text of 25 articles was assessed for eligibility, and 3 were excluded due to insufficient detail for evaluation. Ultimately, 22 studies were included in the final analysis.

RESULTS

This systematic review identified 22 published cases of cutaneous adverse reactions attributed to statin therapy. The mean age of affected individuals was approximately 61 years (range: 44-84), with slight female predominance.

The cutaneous adverse events observed included lichenoid eruptions or lichenoid dermatitis, DM, eczema-like or eczematous eruptions, and PLC. Additional rare manifestations included LET, pemphigus erythematosus, erythema multiforme, fixed drug eruption, and thrombocytic purpura. The types of cutaneous adverse reactions observed are summarized in Table 1 and the systematic review is shown in Table 2.

Eczematous eruptions

Four cases of eczema-like eruptions were identified, all occurring in male patients ranging in age from 55 to 82 years.[9,10] These eruptions were primarily associated with simvastatin and pravastatin, and less frequently with atorvastatin. The rashes presented on various body sites including the thorax, extremities, buttocks, face, neck, and back. Clinically, they appeared as erythematous, squamous, lichenified, nummular plaques, and in one case, the eruption mimicked psoriasis and was initially refractory to methotrexate.[10] Histopathologic features across these cases were consistent with an eczematous dermatitis pattern, revealing parakeratosis, acanthosis, spongiosis, and perivascular lymphocytic or lymphohistiocytic infiltrates. In one case, eosinophils were also noted. All patients experienced resolution of their symptoms following statin discontinuation, with some also requiring topical or systemic corticosteroids. The time to resolution varied from 2 to 8 weeks.

Lichenoid drug eruptions

Lichenoid drug eruptions, including lichenoid dermatitis and lichen planus, were reported in six cases, involving both men and women aged 57 to 75 years.[3,11-15] Offending agents included simvastatin, pravastatin, fluvastatin, atorvastatin, and rosuvastatin. The eruptions were most commonly found on the arms, hands, upper extremities, chest, back, and buccal mucosa, and presented as pruritic polygonal papules, scaly erythematous plaques, or red-gray lesions. In some cases, mucosal involvement with white streaks was observed. Histologic features consistently demonstrated lichenoid interface dermatitis, characterized by basal cell vacuolar degeneration, band-like lymphocytic infiltrate, hyperkeratosis, parakeratosis, colloid (Civatte) bodies, and in some instances, melanophages or eosinophils. Notably, one case confirmed a causal relationship with a rechallenge, where lesions reappeared within a week after restarting the statin[12]. All eruptions resolved after discontinuation of the statin, with or without the aid of corticosteroids. Most improved within 3 to 9 weeks, although one case took up to 9 months for complete resolution.[14]

Statin-induced DM

Four distinct cases of statin-induced DM were identified involving middle-aged adults (ages 44-55). We identified four distinct cases of statin-induced DM involving middle-aged adults (ages 44-55).[4,16-18] Atorvastatin was the implicated agent in all four cases, with doses ranging from 10 mg to 80 mg/day. Symptom onset occurred between 5 days and 1 year after statin initiation, with two cases following recent cardiac events (e.g., myocardial infarction or coronary artery bypass graft). All four patients exhibited classic cutaneous signs (heliotrope rash, Gottron's papules, photodistributed erythema), along with proximal muscle weakness. Histopathologic findings consistently showed interface dermatitis, keratinocyte apoptosis, hydropic degeneration, and in muscle biopsies, necrotizing myopathy or perifascicular inflammation. Discontinuation of the statin and initiation of systemic corticosteroids (e.g., prednisone) led to clinical improvement in most cases, though one patient was refractory to high-dose steroids and rituximab.

PLC

Three cases of PLC were identified, all associated with pravastatin or rosuvastatin. Patients were aged 62, 70, and 79 years.[19] The clinical presentation ranged from papular erythematous eruptions on the extremities to maculopapular squamous rashes on the trunk and limbs, sometimes accompanied by hyperpigmentation and scale. Histopathological findings were not consistently reported across these cases. Treatments included topical steroids, moisturizers, antibiotics, and statin discontinuation, leading to complete resolution within 2 months in all three cases. The 79-year-old male patient was initially refractory to dermovate with urea and glycine, but improved only after cessation of the statin.

Other cutaneous manifestations

Several non-lichenoid, non-eczematous eruptions were also reported. Hanson et al reported a 68-year-old man developed a drug eruption beginning in the groin and spreading to the rest of the body after switching from simvastatin to atorvastatin.[20] Although no biopsy was performed, the eruption resolved within 6 weeks of discontinuing the drug. Huertas et al reported a fixed drug eruption in an 84-year-old man, who developed erythematous macules across the trunk and arms, with biopsy revealing chronic superficial dermatitis with eosinophilic infiltrates [21]. The reaction resolved within 2 months of stopping atorvastatin. In another unique presentation, a 70-year-old man experienced acral vesiculobullous and pustular eruptions following a statin rechallenge.[22] Histology showed spongiosis, microvesiculation, focal lichenoid changes, and papillary dermal fibrosis with eosinophils. This eruption was

refractory to corticosteroids but resolved after stopping simvastatin. Lastly, Wong described a complex eruption in a 55-year-old woman, initially suggestive of lichen planus or lupus erythematosus, with red-gray pruritic plaques and histology showing basal vacuolar changes and parakeratosis.[23] The rash resolved within 3 months after discontinuation of rosuvastatin and treatment with clobetasol.

Drug interactions and polypharmacy effects

One additional case involved a 55-year-old woman who developed a diffuse pigmented skin rash involving the entire body while taking multiple atypical antipsychotics.[24] Following the initiation of pravastatin, the pre-existing rash was exacerbated, suggesting a possible synergistic effect or increased cutaneous sensitivity due to polypharmacy. The rash was described as erythematous and widespread, though no histopathological findings were reported. Resolution occurred after discontinuation of both the statin and several antipsychotic medications, indicating that pravastatin may have potentiated a drug-induced cutaneous reaction in a sensitized patient.

Additionally, four unique cases were identified that included LET, pemphigus erythematosus, erythema multiforme, and thrombocytic purpura.[5,25-27] Each of these reflects a likely immune-mediated mechanism. Diagnostic workup often included direct immunofluorescence (DIF), revealing IgM, IgG, or C3 deposition patterns. Across all cases, statin withdrawal led to partial or complete resolution, with adjunctive treatments tailored to disease severity.

Table 1: Types of cutaneous adverse reactions observed with statin therapy.

Reaction type	Number of reported cases
Eczematous eruptions	4
Lichenoid drug eruptions	6
DM	4
PLC	3
Other manifestations	5

*This table categorizes the types and frequencies of cutaneous adverse events reported in the literature and attributed to statin use. Lichenoid drug eruptions were the most frequently reported, followed by DM 5tr4tr5g23w and eczematous eruptions, and then PLC. The category "Other manifestations" includes less common presentations such as bullous pemphigoid, erythema multiforme, and panniculitis

Table 2: Summary of published cases of cutaneous adverse events associated with statin therapy.

Authors, year	Drug and dosage	Diagnosis	Clinical presentation	Pathology	Treatment and outcome
Possamai, 1992[25]	Simvastatin 10 mg/day	Thrombocytic purpura	Diffuse petechiae and gingival bleeding	N/A	Intravenous immune globulin and methylprednisolone 80 mg IV resolved thrombocytopenia
Noel, 2001[16]	Atorvastatin 10 mg/day	DM	Heliotrope rash, gottron papules with periungual telangiectasia, necrotic skin lesions of the left ear, and proximal muscle weakness	Skin bx: Keratinocyte apoptosis with liquefaction of the epidermal basal zone. Muscle bx: severe necrosis of the segmental muscle fibers with perifascicular T lymphocytes	Rapid improvement following discontinuation of statin
Schiavo, 2014[26]	Atorvastatin	Pemphigus erythematosus relapse	Erythematous scaly plaques	Superficial intraepidermal blister; Tzanck smear showed acantholytic cells with rare dyskeratosis and leukocytes. Direct immunofluorescence revealed IgG and C3 deposits on keratinocyte surfaces and at the dermoepidermal junction	Systemic deflazacort and hydroxychloroquine, along with topical clobetasol, led to disease regression within 12 weeks
Oztas, 2017[4]	Atorvastatin 10 mg/day	DM	Reddish rash on the face and Gottron's papules on the knuckles proximal muscle weakness	Interface dermatitis with hydropic degeneration of basal keratinocytes	Started on prednisolone 1 mg/kg/day. Symptoms resolved within 30 days of starting the treatment.

Continued.

Authors, year	Drug and dosage	Diagnosis	Clinical presentation	Pathology	Treatment and outcome
Spiro, 2018[17]	Atorvastatin 80 mg/d	DM	Erythematous and dry dermatitis, gottron papules, faint erythema of the eyelids and face, muscle weakness in upper and lower extremities	Interface dermatitis, with apoptotic basal keratinocytes as well as basal layer vacuolar changes	Switched from atorvastatin 80 mg/d to pravastatin 40 mg/d. Started prednisone 50 mg/d. significant improvement after 3 weeks
Gutierrez, 2020[18]	Atorvastatin, no dose specified	DM	One-month history of progressively worsening dysphagia and proximal muscle weakness, heliotrope rash and Gottron's papules	Muscle bx: necrotizing myopathy	Patient's rash and muscle weakness did not improve with high-dose intravenous steroids and rituximab infusions
Trčko, 2023[27]	Atorvastatin, no dose specified	LET	Erythematous plaques	Moderate periadnexal and perivascular lymphoplasmacytic infiltrate with dermal mucin; immuno-fluorescence revealed focal granular IgM and C3 in vessel walls, and IgG/IgM along the dermoepidermal junction.	Atorvastatin was discontinued; treatment initiated with mometasone cream and photoprotection. Rash resolved after a few weeks
Chacko, 2024[5]	Atorvastatin 80 mg/d	Erythema multiforme	Hyperpigmented targetoid lesion with central clearing	N/A	Rash disappeared within four days of discontinuing atorvastatin
Krasovec, 1993[9]	Sinvastatin 40 mg/day	Eczematous skin rashes	Erythematous squamous nummular plaques on thorax and upper extremities.	N/A	Discontinuation and prednisone 40mg/day was given. Most lesions disappeared within 2 weeks.
	Simvastatin 20 mg/day		Erythematous, squamous, lichenified plaques. Hyperkeratosis on palms and heels.	Eczematous pattern with parakeratosis, acanthosis with focal spongiosis and superficial dermal lymphohistiocytic infiltrates.	Discontinuation of statin and addition of topical corticosteroids led to resolution within 5 weeks.
	Simvastatin 20 mg/day for 1st 2 weeks followed by pravastatin 10 mg/day		Xerosis with erythematous pityriasiform and lamellar scaling on face, ears, neck, and forearm	Hyperkeratosis with focal parakeratosis, acanthosis with spongiosis and some exocytosis of inflammatory cells. The superficial dermal vessels showed perivascular lymphohistiocytic infiltrates.	6 weeks post discontinuation, skin rash resolved.
Roger, 1994[11]	Simvastatin 10 mg/day	Lichenoid drug eruption	Pruritic, erythematous eruption of polygonal papules on flexor sites of upper extremities/ Wickham striae was also noted	Epidermal hyperplasia with overlying compact orthokeratosis and foci of parakeratosis. Vacuolar degeneration at dermo-epidermal interface, and Civatte bodies there was a lymphohistiocytic infiltrate of the papillary dermis with few eosinophils but no plasma cells	Discontinuation of statin and resolution followed 4 weeks later. Buccal mucosal lesions persisted.
Keough, 1998[12]	Pravastatin 10 mg/day	Lichenoid dermatitis	Bilateral pruritic polygonal shiny eruptions on extensor surface of arms and dorsal hands	Lichenoid dermatitis with lymphocytic infiltrate. Hyperkeratotic stratum corneum and focal hypergranulosis.	Discontinued medication, lesions start to disappear at 2 week follow-up. With a statin rechallenge, new lesions started to appear within week.

Continued.

Authors, year	Drug and dosage	Diagnosis	Clinical presentation	Pathology	Treatment and outcome
Sebök, 2004[13]	Fluvastatin 20 mg/day	Lichen planus	Bilateral shiny pruritic papular eruption located on dorsal arm and hands, soles, and less so on trunk and thighs. Wickham striae. White streaks on buccal mucosa.	Band-like lymphocytic infiltrate in the upper dermis as well as vacuolar alteration along the dermoepidermal junction in the epidermis. Hyperkeratotic stratum corneum and colloid bodies.	After discontinuation of statin, rash resolved within 3 weeks, leaving behind some hyperpigmentation.
Pua, 2006[14]	Pravastatin	Lichenoid drug eruption	Dense freckling on face and upper back. Pigmentation on buccal mucosa was limited.	Epidermal basal cell damage as evident by the pres-ence of Civatte bodies, numerous pigment-laden macrophages In the superficial dermis and lymphocytic inflammation	Discontinuation of statin led to the rash gradually dissolving over 9-months.
Walder, 2009[24]	Pravastatin	Pigmented skin rash	Erythematous skin rash on the entire body.	N/A	Discontinuation of statin and many other atypical antipsychotics
Massay, 2012[19]	Pravastatin 40 mg/day orally	PLC	Papular rash on erythematous base on extremities	N/A	Moisturizer, steroids, antibiotics, and discontinuation of statin lead to resolution of rash within 2 months.
	Pravastatin 40 mg/day orally	PLC	Maculopapular squamous rash on trunk and limbs. Some had hyperpigmentation/scal e	N/A	Refractory to dermovate with urea and glycine. Discontinuation of statin, with resolution within 2 months.
	Rosuvastin 20 mg/day	PLC	N/A	N/A	Discontinuation of statin
Salna, 2017[10]	Pravastatin 20 mg/day	Pravastatin induced eczematous eruption mimicking psoriasis	Pruritic/eczematous lesions on bilateral posterior arms	compact orthokeratosis and significant spongiosis with a perivascular lymphocytic infiltrate.	Refractory to methotrexate. Discontinued statin and saw resolution in 8 weeks.
Wong, 2018[23]	Rosuvastatin	Lichen planus/drug eruption/lupus erythematosus	Red-gray pruritic lesions on right thigh.	Basal vacuolar changes and parakeratosis	Discontinuation of statin and clobetasol propionate led to disappearance of rash 3 months later.
	Simvastatin 10 mg/day		Red-gray pruritic lesions	N/A	None reported/
Forouzan, 2020[15]	Atorvastatin	Lichenoid dermatitis w/ eosinophils	Erythematous to purple scaly plaques on bilateral forearms, chest, upper back, and neck"	Orthokeratosis, acanthosis, and spongiosis	Discontinuation, prednisone, and topical betamethasone dipropionate 0.05% cream. Resolution within 1-month.
Navarro-Triviño, 2023[3]	Pravastatin	Lichenoid eruption	Shiny erythematous/squamous and pruritic eruption on chest and lumbar area.	Lichenoid inflammatory infiltrate with intense involvement of the dermoepidermal interface associated with apoptotic keratinocytes and melanophages in the papillary dermis.	Discontinuation of statin. Changed to fluvastatin. Overall resolution, with some brown hyperpigmentation post 3-months.
Adams, 2010[22]	Simvastatin 20mg/d	Simvastatin-induce d acral cutaneous vesiculobullou s and pustular eruption.	Purpuric pruritic papular eruptions grouped that were "chameleon-like" erythema.	Epidermal spongiosis with microvesiculation, focal lichenoid mononuclear cells, eosinophilic, papillary dermal fibrosis	Refractory to steroids. Discontinuation of simvastatin allowed lesions to clear.

Continued.

Authors, year	Drug and dosage	Diagnosis	Clinical presentation	Pathology	Treatment and outcome
Huertas, 2015[21]	Atorvastatin	Fixed drug eruption	Erythematous macules from trunk to the arms.	Chronic superficial dermatitis with infiltration of leukocytes, polymorphonuclear cells, and eosinophils in perivascular areas	Discontinuation of statin with resolution 2 months later.
Hanson, 2023[20]	Atorvastatin 40 mg daily	Drug eruption	Right groin rash that progressed to the rest of the body, sparing the face.	N/A	Discontinuation of statin. Resolution within 6 weeks.

*This table presents individual case reports of cutaneous adverse reactions linked to statin use, as identified in the systematic review. For each case, the following information is detailed: author and year of publication; statin drug and dosage; description of clinical presentation; histopathologic findings where available; and treatment strategies along with reported outcomes.

DISCUSSION

This systematic review highlights the heterogeneity of statin-induced cutaneous adverse reactions and reveals several important clinical and histopathologic patterns that have implications for diagnosis and management. The incidence of adverse cutaneous drug reactions from statins is low, generally estimated at less than 2% in clinical practice. In a retrospective lipid clinic cohort, skin-related adverse events accounted for approximately 5% of all statin-associated adverse events reported by patients with statin intolerance, suggesting that cutaneous reactions are uncommon but do occur in clinical settings.[28] Despite the low incidence of such reactions relative to the widespread use of statins, the diversity and severity of manifestations suggest that clinicians should maintain a high index of suspicion, particularly in patients with new-onset or refractory dermatoses. In addition, statins are so widely used that adverse drug reactions related to them most likely account for a larger proportion of cases than other drugs. A recurring trend across cases was the immune-mediated nature of many eruptions, spanning lichenoid interface dermatitis, autoimmune blistering diseases, and connective tissue disease–like presentations.[6,7] These patterns point toward statins' capacity to trigger or unmask underlying immune dysregulation. The presence of histologic features such as basal vacuolar change, dense lymphocytic infiltrates, and deposition of immunoglobulins on direct immunofluorescence further supports an immunologic mechanism rather than simple hypersensitivity or irritant reaction.[12,13] This raises important questions about whether certain patients may have a genetic or immunologic predisposition to developing these reactions.

Another notable observation is the temporal variability in onset, with some eruptions appearing days after initiation and others months or even a year later. This suggests a spectrum of underlying mechanisms-from immediate hypersensitivity to delayed-type or cumulative immune responses. The delayed onset also complicates diagnosis, as patients and clinicians may not initially suspect the statin as the culprit, particularly in the setting of polypharmacy or preexisting skin conditions. Resolution of symptoms following statin discontinuation-often without need for prolonged immunosuppression-strengthens the causal association and highlights the importance of prompt drug withdrawal in suspected cases. However, the fact that corticosteroids were frequently needed, and in some cases immunomodulators like rituximab were employed, illustrates the potential severity and persistence of these reactions. This suggests that while withdrawal is necessary, it is not always sufficient to achieve remission. Interestingly, certain statins appeared more frequently than others in association with specific reaction types (e.g., atorvastatin with DM, simvastatin with eczematous eruptions), though the small sample size limits conclusions about differential risk. Nevertheless, these signals warrant further exploration in larger pharmacovigilance datasets.

Finally, the potential role of drug interactions and polypharmacy, particularly in elderly patients or those with psychiatric comorbidities, adds another layer of complexity. Statins may not always act as the sole inciting agent but could contribute to a cumulative burden of immune or metabolic stress on the skin.[24] In such contexts, cutaneous reactions may reflect synergistic toxicity or immune priming, emphasizing the importance of comprehensive medication reconciliation and review.

A key limitation of this review is the reliance on case reports and small series, which are prone to reporting and publication bias, lack standardized diagnostic criteria, and often omit detailed drug histories/follow-up. Histopathologic and laboratory confirmation was inconsistent, and causality cannot be definitively established. Milder/ misattributed cases are likely underreported, limiting conclusions about true incidence or statin-specific risk. Nonetheless, recurrence of specific clinical patterns, particularly lichenoid eruptions and DM, across unrelated reports supports plausible drug-related mechanism. While case reports have inherent limitations, they are vital early indicators of rare adverse reactions. Consistent presentation of these reactions across diverse patients and settings highlights their clinical relevance and supports greater recognition in practice.

As statin use continues to increase in aging populations, both dermatologists and dermatopathologists must remain vigilant for drug-induced dermatoses and pursue thorough medication histories, especially in cases of new-onset or atypical presentations. For dermatopathologists, recognition of histologic patterns such as interface dermatitis, lichenoid inflammation, keratinocyte necrosis, and mucin deposition is essential.[4,16,17,27] These features can overlap with idiopathic autoimmune diseases but, when correlated with clinical context and recent statin use, may point to a drug-induced etiology. Direct immunofluorescence can further support this diagnosis when findings include IgG, IgM, or C3 deposition at key anatomic sites.[26,27] Communicating these patterns back to the clinical team can facilitate timely diagnosis, cessation of the offending agent, and avoidance of unnecessary long-term immunosuppression.

CONCLUSION

This review highlights the importance of interdisciplinary collaboration in recognizing and managing statin-induced cutaneous adverse reactions. Given the broad clinical spectrum, variable onset, and potential severity of these reactions, early recognition through clinical suspicion, timely biopsy, and histopathologic correlation is essential. Coordination among dermatology, pathology, and rheumatology teams can improve diagnostic precision and optimize therapeutic decisions, particularly in immune-mediated presentations requiring immunosuppressive treatment. Heightened awareness among clinicians and dermatopathologists of the histologic patterns and potential for statin-induced autoimmunity is critical to preventing prolonged morbidity. Future research should focus on elucidating the underlying immunopathogenesis and identifying risk factors-such as genetic predisposition, immune status, or polypharmacy-that may render certain individuals more susceptible. Such efforts may ultimately guide safer prescribing practices and personalized risk stratification in statin therapy.

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