INTERNAL MEDICINE

Cite as: Archiv EuroMedica. 2026. 16; 3. DOI 10.35630/2026/16/Iss.3.04

Received 21 March 2026;
Accepted 15 June 2026;
Published 18 June 2026

DERMATOMYOSITIS AND STATIN-ASSOCIATED MYOPATHIES: A PRACTICAL APPROACH TO DIFFERENTIAL DIAGNOSIS: A NARRATIVE REVIEW

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ABSTRACT

Background

Statins are widely used to reduce cardiovascular morbidity and mortality, but muscle symptoms during statin therapy are a frequent clinical problem. Most cases represent reversible toxic statin myopathy, whereas statin-associated immune-mediated necro-

tizing myopathy and dermatomyositis require different diagnostic and therapeutic strategies.

Aims

This narrative review summarizes the clinical spectrum of statin-associated muscle disorders and dermatomyositis and provides a practical framework for their differential diagnosis.

Methods

A narrative literature review was performed using PubMed/MEDLINE and Google Scholar. Publications from January 2010 to November 2025 were searched using terms related to statin-associated muscle symptoms, statin myopathy, rhabdomyolysis, immune-mediated necrotizing myopathy, anti-HMGCR antibodies, dermatomyositis, and inflammatory myopathies. Original studies, clinical reviews, consensus statements, guidelines, and selected clinically relevant case reports were included. No formal systematic review protocol was used.

Results

Toxic statin myopathy, statin-associated immune-mediated necrotizing myopathy, and dermatomyositis may present with proximal muscle weakness and elevated creatine kinase. Toxic statin myopathy usually improves after statin withdrawal. Immune-mediated necrotizing myopathy is characterized by persistent or progressive weakness despite statin discontinuation and is associated with anti-HMGCR antibodies. Dermatomyositis is distinguished by characteristic skin manifestations, systemic involvement, and increased malignancy risk. Differential diagnosis requires clinical assessment, creatine kinase measurement, autoantibody testing, muscle imaging, and, when needed, muscle biopsy.

Conclusions

Muscle symptoms during statin therapy should not automatically be attributed to toxic statin myopathy. Persistent weakness, sustained creatine kinase elevation, positive anti-HMGCR antibodies, myositis-specific antibodies, cutaneous signs, or systemic manifestations should prompt evaluation for autoimmune myopathy. Correct differentiation is essential because toxic statin myopathy usually requires lipid-lowering ther-

apy modification, whereas immune-mediated necrotizing myopathy and dermatomyositis require immunosuppressive treatment and specialist follow-up.

Keywords: statin associated toxicity, dermatomyositis, statin associated immune mediated necrotizing myopathy, inflammatory myopathies, statins.

INTRODUCTION

Background and rationale

Statins are among the most commonly prescribed medications worldwide due to their proven efficacy in reducing cardiovascular morbidity and mortality in both primary and secondary prevention of atherosclerotic cardiovascular disease [5]. Muscle-related adverse effects represent the most frequently reported complications of statin therapy and are collectively referred to as SAMS [13]. The reported prevalence of SAMS varies widely, ranging from 5% to 10% in randomized trials to as high as almost 30% in observational studies, reflecting differences in definitions and patient populations [7,14]. The clinical spectrum of statin-associated muscle disorders is broad and includes benign myalgia, toxic myopathy with elevated CK, and rare but severe rhabdomyolysis [8,15]. In most cases, symptoms improve or resolve after statin discontinuation, supporting a direct toxic or pharmacologic mechanism [7]. However, a subset of patients develop persistent or progressive muscle weakness despite cessation of statin therapy, suggesting an alternative pathophysiological process [22,45].

Over the past decade, increasing attention has been given to statin-associated IMNM, an autoimmune condition characterized by severe proximal muscle weakness, markedly elevated CK levels, and the presence of anti-HMGCR antibodies [20,23,44]. Unlike toxic statin myopathy, this condition requires immunosuppressive therapy and may lead to significant morbidity if not recognized early [6,16]. Dermatomyositis represents another important autoimmune inflammatory myopathy that may present with proximal muscle weakness and elevated muscle enzymes, potentially mimicking statin-associated muscle disease in statin-treated patients [4]. In addition to muscle involvement, dermatomyositis is characterized by distinctive cutaneous manifestations and systemic complications, including interstitial lung disease and an increased risk of malignancy [3,43]. Early recognition is essential, as delayed diagnosis may adversely affect both functional outcomes and cancer detection [4].

The coexistence of statin exposure and inflammatory myopathy creates a significant diagnostic challenge in clinical practice. Because statin therapy is common in middle-aged and elderly populations, many patients presenting with new-onset muscle symptoms are initially presumed to have drug toxicity [1]. This assumption may lead to delays in the diagnosis of autoimmune myopathies, particularly when symptoms persist after drug withdrawal [6,45]. Accurate differentiation between toxic statin myopathy, statin-associated IMNM, and dermatomyositis is critical because management strategies differ substantially. While toxic myopathy typically requires only discontinuation or modification of lipid-lowering therapy, autoimmune myopathies necessitate prompt initiation of immunosuppressive treatment and long-term monitoring [2]. Moreover, dermatomyositis requires additional evaluation for extramuscular manifestations and malignancy screening [3,34]. Given the increasing use of statins and the growing recognition of autoimmune muscle disorders, clinicians across multiple specialties frequently encounter patients with muscle symptoms during statin therapy. A structured and practical approach to differential diagnosis is therefore essential to avoid misclassification and ensure appropriate treatment [8].

Despite the widespread use of statins and increasing recognition of autoimmune inflammatory myopathies, the differential diagnosis of muscle symptoms in statin-treated patients remains a significant clinical challenge. In everyday practice, muscle weakness or elevated creatine kinase levels are frequently attributed to toxic statin-associated muscle symptoms, which may delay the recognition of autoimmune conditions such as immune-mediated necrotizing myopathy or dermatomyositis. Although numerous studies have addressed statin-associated myopathy and inflammatory muscle diseases separately, there is a lack of practical narrative reviews that comprehensively compare their clinical presentation, diagnostic pathways, and key distinguishing features from a clinician-oriented perspective. The scientific novelty of this review lies in its integrative and practice-oriented approach, focusing on clinically relevant red flags, stepwise diagnostic strategies, and practical tools that may facilitate early differentiation between toxic and autoimmune muscle injury in statin-treated patients.

AIMS

The aim of this narrative review is to summarize the clinical and diagnostic features of dermatomyositis, toxic statin myopathy, and statin-associated immune-mediated

necrotizing myopathy, and to provide a practical framework for their differentiation in patients presenting with muscle symptoms during statin therapy.

Research questions: What clinical features help differentiate toxic statin myopathy from autoimmune inflammatory myopathies in statin-treated patients? Which laboratory, serological, imaging, and histopathological findings are most useful in distinguishing statin-associated immune-mediated necrotizing myopathy and dermatomyositis from toxic statin-associated muscle symptoms? How can a stepwise diagnostic approach support earlier recognition of autoimmune myopathies in patients receiving statin therapy?

METHODS

This narrative review was conducted to summarize clinically relevant literature on statin-associated muscle disorders, statin-associated immune-mediated necrotizing myopathy, dermatomyositis, and their differential diagnosis in patients presenting with muscle symptoms during statin therapy.

A literature search was performed using PubMed/MEDLINE and Google Scholar. The search covered publications from January 2010 to November 2025. The last literature search was performed in November 2025. The following search terms were used alone and in combination: "statin associated muscle symptoms", "statin myopathy", "rhabdomyolysis", "immune mediated necrotizing myopathy", "anti HMGCR", "dermatomyositis", "inflammatory myopathies", "myositis specific autoantibodies", "muscle biopsy", and "muscle MRI".

Publications were selected according to their clinical relevance to the aim of this narrative review. Original studies, clinical reviews, consensus statements, clinical guidelines, and selected case reports published in English were considered. Priority was given to publications addressing clinical presentation, diagnostic strategies, laboratory findings, serological testing, imaging, histopathological features, treatment implications, and malignancy screening in dermatomyositis.

Publications were excluded if they were not available in English, were not clinically relevant to the differential diagnosis discussed in this review, focused only on experimental or molecular mechanisms without practical clinical implications, or did not address statin-associated muscle disorders, immune-mediated necrotizing myopathy, dermatomyositis, or inflammatory myopathies.

In the current version of the manuscript, the final reference list includes 45 sources. These sources were analyzed narratively, with emphasis on clinically relevant findings that help differentiate toxic statin myopathy, statin-associated immune-mediated necrotizing myopathy, and dermatomyositis. No formal systematic review protocol was used, and no systematic evidence grading was performed.

RESULTS

Spectrum of statin-associated muscle disorders

Statin-associated muscle disorders represent a heterogeneous group of conditions that differ in pathophysiology, clinical severity, and management [40]. The spectrum ranges from common, self-limited toxic effects to rare but severe immune-mediated disease. Understanding these entities is essential for distinguishing benign statin intolerance from conditions that require immunosuppressive therapy or urgent intervention. SAMS encompass a broad clinical continuum, including myalgia, myopathy with elevated CK, and severe muscle injury such as rhabdomyolysis [7]. SAMS are reported in approximately 5–10% of patients in randomized trials, although higher rates are observed in real-world clinical settings [8]. The majority of cases are related to direct toxic or metabolic effects of statins on skeletal muscle [7].

The most common form of statin-related muscle disease is toxic statin myopathy, which includes myalgia with or without mild CK elevation and, less frequently, true myopathy defined by CK levels exceeding 10 times the upper limit of normal [8]. Symptoms typically involve symmetrical proximal muscle pain, stiffness, or mild weakness and usually develop within weeks to months after initiation or dose escalation of statin therapy [7]. Several risk factors increase susceptibility to toxic statin myopathy, including advanced age, female sex, low body mass index, renal or hepatic dysfunction, hypothyroidism, and drug-drug interactions that increase statin plasma concentrations [8]. Lipophilic statins and higher doses are also associated with increased risk [11]. A key diagnostic feature of SAMS is clinical improvement after statin discontinuation or dose reduction, typically within weeks [7]. CK levels usually normalize during recovery, and persistent or progressive weakness despite drug withdrawal should prompt evaluation for alternative diagnoses, including autoimmune myopathy [6].

Rhabdomyolysis represents the most severe form of toxic statin-induced muscle injury, although it is rare, occurring approximately 1 case per 10,000 statin-treated pa-

tients per year [8]. It is characterized by extensive muscle necrosis, marked CK elevation often exceeding 10,000 IU/L, myoglobinuria, and a risk of acute kidney injury (AKI) [7]. Clinical presentation typically includes severe muscle pain, profound weakness, dark urine, and systemic symptoms such as fatigue or malaise [8]. Predisposing factors include high statin doses, interacting medications (particularly CYP3A4 inhibitors), advanced age, and comorbid conditions such as renal impairment [7]. Management requires immediate discontinuation of statin therapy and aggressive supportive treatment, including intravenous hydration and monitoring of renal function and electrolyte abnormalities [8]. Importantly, unlike autoimmune myopathies, rhabdomyolysis is an acute toxic process and does not require immunosuppressive therapy [7].

A distinct and increasingly recognized entity within the statin-associated spectrum is IMNM associated with anti-HMGCR antibodies [17]. This condition is rare but clinically significant because symptoms persist or worsen despite statin discontinuation and require immunosuppressive treatment [2]. Patients typically present with progressive symmetrical proximal muscle weakness and markedly elevated CK levels, often exceeding 2,000–10,000 IU/L and/or >10x upper limit normal (ULN) [6,44]. Unlike toxic SAMS, muscle symptoms do not improve after drug withdrawal, and disease progression may continue for months if untreated [2]. Anti-HMGCR antibodies are highly specific for this condition and play a central role in diagnosis [20]. Although most patients have a history of statin exposure, the disease may occasionally occur in statin-naive individuals, suggesting that statins act as a trigger rather than the sole cause [2]. Muscle biopsy typically demonstrates necrotic and regenerating fibers with minimal inflammatory infiltrate, distinguishing IMNM from other inflammatory myopathies [19]. Treatment generally involves high-dose corticosteroids combined with additional immunosuppressive agents or intravenous immunoglobulin, and many patients require long-term therapy [6,36].

The major clinical distinction within the statin-associated spectrum lies in reversibility. Toxic statin myopathy and rhabdomyolysis improve after drug withdrawal and supportive care, whereas anti-HMGCR myopathy represents an autoimmune disease that persists independently of statin exposure [2]. Failure to recognize this difference may delay appropriate immunosuppressive treatment and result in irreversible muscle damage [6]. Understanding this spectrum is essential for clinicians evaluating patients with muscle symptoms during statin therapy and forms the basis for differentiating toxic injury from autoimmune inflammatory myopathies, including dermatomyositis.

Dermatomyositis: a clinical overview

Dermatomyositis is a systemic autoimmune inflammatory myopathy characterized by proximal muscle weakness, distinctive cutaneous manifestations, and a wide spectrum of extramuscular involvement [25]. It belongs to the group of idiopathic inflammatory myopathies and represents a key diagnostic consideration in patients presenting with progressive muscle symptoms, particularly when statin-associated muscle disease is suspected but clinical features are atypical [4,28]. Dermatomyositis affects both adults and children, with adult-onset disease typically occurring between the ages of 40 and 60 years [3]. The pathogenesis involves complement-mediated microangiopathy leading to perifascicular muscle fiber injury and characteristic skin involvement, distinguishing dermatomyositis from necrotizing myopathies and toxic muscle injury [10].

The hallmark of dermatomyositis is symmetrical proximal muscle weakness, most prominently affecting the shoulder and pelvic girdle muscles [4]. Patients commonly report difficulty climbing stairs, rising from a seated position, or lifting objects overhead [10]. The onset is usually subacute and progressive over weeks to months, although acute presentations may occur [3]. Serum CK levels are typically elevated but may vary widely, ranging from mild increases to levels exceeding 10 times the upper limit of normal [4]. Importantly, normal or minimally elevated CK levels do not exclude the diagnosis, particularly in patients with predominantly cutaneous or chronic disease [10].

Characteristic skin findings are a defining feature of dermatomyositis and provide a critical diagnostic clue that distinguishes it from toxic statin myopathy and IMNM. The heliotrope rash, a violaceous discoloration of the eyelids often accompanied by periorbital edema, is considered highly specific [3]. Gottron papules, erythematous or violaceous papules over the extensor surfaces of the metacarpophalangeal and interphalangeal joints, represent another classic finding [10]. Additional cutaneous features include Gottron sign, shawl and V-sign erythema, photosensitivity, and mechanic's hands [3]. In some cases, skin manifestations precede muscle weakness, and clinically amyopathic dermatomyositis may occur, further complicating diagnosis [4].

Myositis-specific autoantibodies (MSAs) are detected in a substantial proportion of patients and are associated with distinct clinical phenotypes [9]. Anti-Jo-1 antibodies are the most frequently detected myositis-specific autoantibodies and are included in the EULAR/ACR classification criteria for idiopathic inflammatory myopathies [4]. Anti-Mi-2 antibodies are associated with classic dermatomyositis and a relatively favorable

prognosis, whereas anti-TIF1-γ and anti-NXP2 antibodies are linked to an increased risk of malignancy [9,31]. Anti-MDA5 antibodies are associated with clinically amyopathic disease and rapidly progressive interstitial lung disease [3,32]. The presence of MSAs supports the diagnosis and may help differentiate dermatomyositis from other inflammatory or toxic muscle disorders, including statin-associated conditions [4].

Dermatomyositis is a systemic disease with multiple potential organ manifestations. Interstitial lung disease represents a major cause of morbidity and mortality [39]. Other systemic features include dysphagia due to involvement of pharyngeal muscles, cardiac involvement, and constitutional symptoms such as fatigue and weight loss [10]. Malignancy represents one of the most clinically important associations. Adult-onset dermatomyositis is associated with an increased risk of cancer, particularly within the first three to five years after diagnosis [4,33]. This association necessitates comprehensive malignancy screening, which is not required in patients with toxic statin myopathy.

Muscle biopsy typically demonstrates perifascicular atrophy, perivascular inflammation, and complement deposition in capillaries, reflecting a microangiopathic process [10,19]. This pattern differs from immune-mediated necrotizing myopathy, which is characterized by myofiber necrosis with minimal inflammatory infiltrate [2]. Magnetic resonance imaging (MRI) often shows symmetrical muscle edema and may be useful in detecting subclinical disease and guiding biopsy [18]. Several features help distinguish dermatomyositis from statin-associated muscle disorders. The presence of characteristic skin findings, systemic manifestations, myositis-specific autoantibodies, and malignancy risk strongly favors dermatomyositis [3]. In contrast, isolated muscle symptoms without cutaneous involvement are more typical of statin-associated toxic myopathy or anti-HMGCR myopathy [6]. Recognition of these clinical characteristics is essential for appropriate diagnostic evaluation and timely initiation of immunosuppressive therapy.

Clinical similarities and differences

Patients presenting with proximal muscle weakness during statin therapy pose a significant diagnostic challenge because toxic statin myopathy, statin-associated IMNM, and dermatomyositis share overlapping clinical and laboratory features. Careful assessment of symptom onset, clinical course, systemic manifestations, and laboratory findings is essential for accurate differentiation [17,29].

Toxic statin myopathy typically develops within weeks to months after initiation of statin therapy or dose escalation [7]. There is a clear temporal correlation between medication exposure and symptoms, which frequently include muscle pain, rigidity, or moderate weakness [8]. Improvement usually occurs within several weeks after discontinuation, which is a key diagnostic feature [7]. In contrast, IMNM may begin during statin treatment but frequently persists or progresses even after drug withdrawal [6]. In some cases, symptoms become apparent months or even years after statin exposure, and disease activity is maintained by an autoimmune process independent of the drug [2]. Dermatomyositis typically presents with a subacute onset of progressive proximal muscle weakness over weeks to months and is not directly related to medication exposure [4]. Although the disease may be temporally associated with statin use by coincidence, lack of improvement after drug discontinuation should raise suspicion for an inflammatory myopathy [3].

All three conditions are associated with symmetrical proximal muscle weakness in the shoulder and pelvic girdles [10]. However, the severity and progression vary. Toxic statin myopathy typically produces mild to moderate symptoms and rarely results in severe functional impairment [8]. IMNM is distinguished by marked and progressive weakness, which can lead to significant disability if left untreated [2]. Serum CK levels are usually extremely high, exceeding 2,000–10,000 IU/L [6]. Dermatomyositis also causes progressive weakness, but functional decline is frequently accompanied by systemic and extramuscular manifestations not seen in toxic SAMS [4].

The presence of characteristic skin manifestations is the most important clinical feature distinguishing dermatomyositis from statin-associated muscle disorders. Heliotrope rash, Gottron papules, shawl sign, and photosensitive erythema are highly suggestive of dermatomyositis [3]. Cutaneous findings may precede muscle weakness and are absent in both toxic statin myopathy and IMNM [10]. Dermatomyositis is also associated with systemic involvement, including interstitial lung disease, dysphagia, arthritis, and constitutional symptoms such as fatigue and weight loss [4]. Furthermore, adult-onset dermatomyositis carries an increased risk of malignancy, necessitating oncologic screening [3]. In contrast, toxic statin myopathy and IMNM are typically limited to skeletal muscle involvement without systemic inflammatory features [2].

Response to statin discontinuation represents one of the most practical differentiating features. Toxic statin myopathy typically resolves within weeks after drug cessation [7]. Lack of improvement or continued progression strongly suggests autoimmune disease, particularly IMNM or dermatomyositis [6]. Overall, toxic statin myopathy is

characterized by mild symptoms, temporal association with statin exposure, and rapid reversibility. IMNM is characterized by severe ongoing weakness, markedly elevated CK levels, and specific autoantibodies. Dermatomyositis is distinguished by characteristic skin findings, systemic involvement, and malignancy risk. Recognition of these differences is essential for timely diagnostic evaluation and appropriate management.

Diagnostic tools in differential diagnosis

Accurate differentiation between toxic statin myopathy, IMNM, and dermatomyositis requires a structured diagnostic approach that integrates laboratory testing, autoantibody assessment, imaging, electrophysiology, and, in selected cases, muscle biopsy. Because clinical features may overlap, particularly in statin-exposed patients with continuous weakness, ancillary investigations play a critical role in establishing the correct diagnosis.

Serum CK measurement represents the initial laboratory test in patients presenting with muscle symptoms during statin therapy [8]. Mild or normal CK levels are typical of statin-associated myalgia, whereas elevations exceeding 10 times the upper limit of normal suggest significant muscle injury [7]. In toxic statin myopathy, CK levels usually decrease after drug discontinuation, whereas persistently elevated values should raise suspicion for autoimmune myopathy [6]. In dermatomyositis and IMNM, CK levels are often markedly elevated, although normal or moderately increased levels may occur in dermatomyositis, particularly in chronic or predominantly cutaneous disease [4]. Additional laboratory abnormalities in dermatomyositis may include elevated aldolase, lactate dehydrogenase, and transaminases, reflecting muscle injury rather than primary hepatic disease [10].

Inflammatory markers such as C-reactive protein (CRP) and erythrocyte sedimentation rate (ESR) may assist in the differential diagnosis of muscle disorders in statin-treated patients. Toxic statin-associated myopathy represents a non-immune-mediated form of muscle injury and therefore usually lacks systemic inflammatory features. Consequently, inflammatory markers such as CRP and ESR are usually normal or only mildly elevated in toxic statin-associated muscle symptoms [13,21]. This contrasts with autoimmune inflammatory myopathies such as dermatomyositis, in which systemic inflammatory activity may lead to elevated acute-phase reactants. For this reason, normal CRP and ESR values in patients presenting with muscle symptoms during statin therapy may support a toxic or non-inflammatory etiology, although they do not completely exclude autoimmune disease.

Autoantibody assessment is a key step in the differential diagnosis of inflammatory myopathies. Anti-HMGCR antibodies are highly specific for statin-associated IMNM and are strongly associated with persistent muscle weakness and elevated CK levels [6]. Detection of these antibodies confirms the autoimmune nature of the disease and supports the need for immunosuppressive therapy [2]. In dermatomyositis, MSAs such as anti-Jo-1, anti-Mi-2, anti-TIF1-γ, anti-NXP2, anti-MDA5, and anti-SAE help establish the diagnosis and define clinical phenotypes [9,30]. Autoantibodies are not present in toxic statin myopathy, and their absence, together with symptom resolution after statin withdrawal, supports a non-immune mechanism [7].

EMG may help distinguish myopathic from neuropathic processes but has limited specificity in differentiating among inflammatory myopathies [10]. Typical findings in dermatomyositis and IMNM include short-duration, low-amplitude motor unit potentials and increased spontaneous activity such as fibrillations [4]. EMG is generally normal or only mildly abnormal in toxic statin myopathy, particularly when symptoms are limited to myalgia [8]. Although EMG cannot reliably distinguish dermatomyositis from IMNM, it can support the presence of active muscle disease and help guide biopsy to affected areas [10].

MRI has emerged as a valuable noninvasive tool for assessing muscle involvement. In inflammatory myopathies, muscle edema reflecting active inflammation is typically detected as a hyperintense signal on T2-weighted or short tau inversion recovery (STIR) sequences [26,27]. Chronic disease may show fatty replacement and muscle atrophy [2]. MRI findings in toxic statin myopathy are usually mild or absent and resolve after drug discontinuation [7]. In contrast, persistent edema despite statin withdrawal suggests autoimmune myopathy and supports further immunological evaluation [6].

Muscle biopsy remains essential for definitive diagnosis when clinical and serological findings are inconclusive. In dermatomyositis, characteristic features include perifascicular atrophy, perivascular inflammation, and deposition of complement components in capillaries, reflecting a microangiopathic process [10,19]. In IMNM, biopsy demonstrates prominent muscle fiber necrosis and regeneration with minimal inflammatory infiltrate, distinguishing it from dermatomyositis [2]. Toxic statin myopathy typically shows nonspecific myofiber necrosis without immune-mediated features [6]. Biopsy is particularly useful when autoantibody testing is negative or unavailable and when the diagnosis remains uncertain after initial evaluation [4].

In clinical practice, the diagnostic strategy should follow a stepwise approach. Initial evaluation includes CK measurement and statin withdrawal. Continuous symptoms or

elevated CK levels should prompt autoantibody testing and muscle MRI. Muscle biopsy is reserved for cases with inconclusive serology or atypical clinical presentation. This integrated approach helps differentiate reversible toxic injury from autoimmune disease requiring immunosuppressive therapy.

The key clinical, laboratory, and imaging features relevant for the differential diagnosis of statin-associated muscle disorders and dermatomyositis are summarized in Table 1. This table represents a synthesis of the data presented in the preceding sections, integrating the most important diagnostic parameters and highlighting practical differences that may support clinical decision-making in patients presenting with muscle symptoms during statin therapy.

Table 1. Key clinical and diagnostic features differentiating toxic statin myopathy, statin-associated IMNM, and dermatomyositis.

INVESTIGATION	TOXIC STATIN MYOPATHY	STATIN-ASSOCIATED IMNM	DERMATOMYOSITIS
Course after statin withdrawal	Improvement within weeks [7,8]	No improvement; progressive course [2,6]	Not related to statin withdrawal; progression may continue without immunosuppressive treatment [4]
Onset	Weeks or months after statin initiation or dose increase [7,8]	May begin during statin use but persists after withdrawal [2,6]	Subacute onset, independent of statin exposure [4]
Muscle symptoms	Myalgia and/or weakness [7,8]	Progressive proximal weakness [2,6]	Progressive proximal weakness [4,9]
Skin manifestations	Absent [7]	Absent [2,6]	Characteristic (heliotrope rash, Gottron papules) [3,4,9]
Systemic involvement	Absent or rare [7]	Rare [2,6]	Common (ILD, dysphagia, malignancy) [3,33]
CK level	Normal to moderately elevated (<10× ULN) [7,8]	Markedly elevated (often >2000–10,000 IU/L) [2,6]	Variable (normal to markedly elevated) [4]
Inflammatory markers (CRP, ESR)	Usually normal or mildly elevated [13,21]	Variable [2,6]	Variable, may be elevated [4,9]
Autoantibodies	Absent [7]	Anti-HMGCR [20]	MSAs (anti-Mi-2, anti-TIF1-γ, anti-NXP2, anti-MDA5, anti-SAE) [9,30]

EMG	Normal or mild myopathic changes	Myopathic pattern [2,6]	Myopathic pattern [4,9]
MRI	Normal or mild changes [26]	Muscle edema, often diffuse [26]	Symmetrical inflammatory edema [26,27]
Muscle biopsy	Nonspecific necrosis [7]	Necrotic fibers with minimal inflammatory infiltrate [2]	Perifascicular atrophy, perivascular inflammation [2,10]

Abbreviations: CK, creatine kinase; CRP, C-reactive protein; ESR, erythrocyte sedimentation rate; IMNM, immune-mediated necrotizing myopathy; SAMS, statin-associated muscle symptoms; MSAs, myositis-specific autoantibodies; anti-HMGCR, antibodies against 3-hydroxy-3-methylglutaryl-coenzyme A reductase; ILD, interstitial lung disease; MRI, magnetic resonance imaging; ULN, upper limit of normal.

When to suspect autoimmune myopathy in statin-treated patients

Muscle symptoms are frequently reported during statin therapy, and in most cases they reflect benign and reversible toxic effects. However, a small subset of patients develop autoimmune muscle disease, including statin-associated IMNM or, less commonly, dermatomyositis occurring coincidentally in statin-treated individuals. Early recognition of autoimmune pathology is essential because delayed diagnosis may result in progressive muscle damage, long-term disability, and inappropriate discontinuation of necessary cardiovascular therapy. Identifying clinical red flags that distinguish SAMS from inflammatory myopathy is therefore a critical step in clinical decision-making.

The most important indicator of autoimmune disease is persistence or progression of muscle weakness despite discontinuation of statin therapy. Toxic SAMS typically improve within weeks after drug cessation, with normalization of CK levels [7]. In contrast, patients with IMNM often experience ongoing or worsening weakness even months after statin withdrawal because the disease becomes self-sustaining through an autoimmune mechanism [6]. Similarly, dermatomyositis progresses independently of medication exposure and does not respond to statin discontinuation [4].

CK elevation occurs in both toxic and autoimmune muscle injury; however, very high or persistent levels should raise concern for inflammatory myopathy. Toxic statin myopa-

thy is usually associated with mild to moderate CK elevation that declines after drug withdrawal [8]. On the other hand, IMNM typically presents with CK levels above 2,000–10,000 IU/L and remains elevated without immunosuppressive treatment [2]. Dermatomyositis may also present with significant CK elevation, although values may be variable and occasionally normal [10].

The severity and progression of weakness provide important diagnostic clues. Toxic SAMS generally cause myalgia or mild weakness without substantial functional impairment [8]. Progressive difficulty rising from a chair, climbing stairs, or lifting the arms above shoulder level suggests an inflammatory myopathy rather than a toxic effect [4]. In IMNM, weakness may become severe and disabling over a relatively short period [6].

The presence of characteristic skin findings strongly supports a diagnosis of dermatomyositis and should immediately prompt evaluation for inflammatory myopathy. Heliotrope rash, Gottron papules, shawl sign, photosensitive erythema, and periungual changes are highly suggestive and are not observed in toxic statin myopathy or IMNM [3]. Cutaneous symptoms may precede muscle weakness, and their recognition is essential for early diagnosis [10].

Toxic SAMS are limited to skeletal muscle and are not associated with systemic inflammation. In contrast, dermatomyositis may present with interstitial lung disease, dysphagia, arthritis, or constitutional symptoms such as fatigue and weight loss [4]. The presence of systemic features should therefore prompt consideration of autoimmune disease. IMNM is usually confined to muscle but may be associated with severe functional impairment disproportionate to the duration of symptoms [2].

Detection of specific autoantibodies provides strong evidence for autoimmune myopathy. Anti-HMGCR antibodies are highly specific for statin-associated IMNM and should be tested in patients with persistent weakness and elevated CK after statin exposure [6]. In suspected dermatomyositis, testing for myositis-specific antibodies such as anti-Jo-1, anti-Mi-2, anti-TIF1-γ, or anti-MDA5 can support the diagnosis and help identify associated complications [9].

Persistent inflammatory changes on muscle MRI or histopathological findings consistent with inflammatory myopathy further support an autoimmune process. Muscle edema on MRI that does not resolve after statin withdrawal is particularly suggestive of immune-mediated disease [2]. Clinicians should suspect IMNM or dermatomyositis when one or more of the red flags presented in Table 2 are identified. These features

may suggest an underlying autoimmune inflammatory myopathy rather than reversible toxic statin-associated muscle symptoms and should be interpreted in the context of the overall clinical presentation, laboratory findings, and response to statin withdrawal.

Table 2. "Red flags" of autoimmune myopathy in patients undergoing statin therapy and their clinical meaning with recommended actions.

RED FLAG	CLINICAL MEANING	RECOMMENDED ACTION
No clinical improvement after statin discontinuation [7]	Persistence of symptoms despite statin withdrawal suggests an autoimmune process rather than reversible toxic statin myopathy [2,6]	Reassess CK levels, perform autoantibody testing, and refer to a rheumatology/neuromuscular specialist [2,6]
Persistent or progressively worsening proximal muscle weakness [6]	Progressive weakness involving shoulder and pelvic girdle muscles is characteristic of inflammatory myopathy, particularly IMNM or dermatomyositis [2,4]	Initiate diagnostic evaluation for inflammatory myopathy including MRI, EMG, and serological testing [4,10,18]
CK levels >2,000 IU/L or persistent elevation despite drug withdrawal [2]	Markedly elevated or sustained CK levels are more consistent with IMNM or active inflammatory muscle disease than toxic SAMS [2,6]	Test for anti-HMGCR antibodies and evaluate for immune-mediated necrotizing myopathy [6,20]
Marked functional impairment or rapid clinical progression [4]	Significant impairment in activities such as climbing stairs, rising from a chair, or lifting arms suggests severe inflammatory muscle involvement [2,4]	Prompt specialist referral and consideration of early immunosuppressive treatment [2,6]
Characteristic cutaneous manifestations suggestive of dermatomyositis [3]	Typical skin findings strongly support dermatomyositis and help distinguish it from toxic statin myopathy and IMNM [3,10]	Perform myositis-specific autoantibody testing and dermatologic/rheumatologic evaluation [3,9]
Systemic features such as interstitial lung disease, dysphagia, or constitutional symptoms [4]	Extramuscular involvement is characteristic of dermatomyositis and other autoimmune inflammatory myopathies [3,4]	Evaluate for organ involvement including pulmonary assessment and swallowing evaluation [3,39]

Positive anti-HMGCR or MSAs [9]	Autoantibody positivity strongly supports autoimmune inflammatory myopathy and may define specific clinical phenotypes [9,20]	Initiate disease-specific management and consider immunosuppressive therapy [2,4,6]
Persistent inflammatory changes on muscle MRI or biopsy [2]	Muscle edema on MRI or inflammatory/necrotizing changes on biopsy support active autoimmune muscle disease [2,10]	Proceed with histopathological confirmation and targeted immunosuppressive treatment [2,19]
Dermatomyositis diagnosed in adulthood [3,33]	Adult-onset dermatomyositis is associated with an increased risk of malignancy, particularly within the first years after diagnosis [3,33]	Perform comprehensive age- and risk-appropriate malignancy screening according to current myositis recommendations and continue surveillance during follow-up [33,34,35]

Abbreviations: MSAs, myositis-specific autoantibodies; anti-HMGCR, antibodies against 3-hydroxy-3-methylglutaryl-coenzyme A reductase; MRI, magnetic resonance imaging; IU, International Unit; CK, creatine kinase.

Recognition of these warning signs and their clinical implications should prompt early referral to a rheumatology or neuromuscular specialist and initiation of a structured diagnostic evaluation, including laboratory testing, autoantibody assessment, muscle imaging, and, when indicated, muscle biopsy. In patients with suspected dermatomyositis, evaluation for systemic involvement and appropriate malignancy screening should also be considered. Early identification of autoimmune inflammatory myopathy and timely initiation of immunosuppressive therapy are associated with improved clinical outcomes and reduced risk of irreversible muscle damage.

Treatment implications

Accurate differentiation between toxic SAMS, statin-associated IMNM, and dermatomyositis has direct therapeutic consequences. These conditions differ fundamentally in pathogenesis and clinical course, and management strategies range from simple drug modification to long-term immunosuppressive therapy. Misclassification may lead either to unnecessary discontinuation of cardioprotective therapy or to delayed treatment of a progressive autoimmune disease.

In patients with suspected toxic SAMS, the primary intervention is temporary discontinuation of statin therapy followed by clinical and biochemical reassessment [7]. Resolution of symptoms and normalization of CK levels confirm a toxic mechanism and allow for subsequent reintroduction of statin therapy at a lower dose or with an alternative agent [8]. Several strategies may improve tolerability, including switching to a different statin, using intermittent dosing, or selecting hydrophilic statins such as pravastatin or rosuvastatin [7]. If statin intolerance persists, non-statin lipid-lowering therapies such as ezetimibe or PCSK9 inhibitors should be considered to maintain cardiovascular risk reduction [5]. Importantly, prolonged discontinuation of statins without appropriate evaluation may increase cardiovascular risk, particularly in high-risk patients, underscoring the importance of accurate diagnosis [5].

Statin-associated IMNM represents an autoimmune disease that does not resolve with statin withdrawal alone. Although discontinuation of statins is recommended, immunosuppressive therapy is the cornerstone of treatment [6]. First-line therapy typically includes high-dose corticosteroids (oral prednisone at a dose of 1 mg per kilogram of body weight, up to 100 mg), often combined with a steroid-sparing immunosuppressive agent such as methotrexate, azathioprine, or mycophenolate mofetil [2]. Intravenous immunoglobulin (IVIG) has demonstrated particular efficacy in anti-HMGCR myopathy and is frequently used in patients with severe weakness or inadequate response to initial therapy [6]. Many patients require prolonged treatment, and disease relapse may occur during tapering of immunosuppressive therapy [24]. Early recognition and treatment are associated with improved functional outcomes and reduced risk of irreversible muscle damage.

Dermatomyositis requires a comprehensive therapeutic approach addressing both muscle and systemic manifestations. High-dose systemic corticosteroids remain the standard initial treatment, often followed by the addition of conventional immunosuppressive agents such as methotrexate, azathioprine, or mycophenolate mofetil to achieve disease control and allow steroid tapering [4]. IVIG is an effective option for patients with refractory disease, severe weakness, or dysphagia [10,37]. Biologic therapies, including rituximab, may be considered in selected cases with treatment-resistant disease [4,38]. In contrast to statin-associated myopathy, dermatomyositis management also requires evaluation and treatment of extramuscular complications. Patients should be screened for interstitial lung disease and other systemic involvement [3]. Additionally, comprehensive malignancy screening is recommended, particularly during the first years after diagnosis, given the well-established association between dermatomyositis and cancer [35].

Management of dyslipidemia in patients with autoimmune myopathy represents a clinical challenge. Re-exposure to statins is generally avoided in patients with anti-HMGCR IMNM due to the potential for disease reactivation [6]. Alternative lipid-lowering therapies, including ezetimibe or PCSK9 inhibitors, should be considered when cardiovascular risk reduction is necessary [5]. In dermatomyositis, statins may be used cautiously once disease control is achieved, although careful monitoring for recurrent muscle symptoms is recommended [2]. The therapeutic implications of differential diagnosis are substantial. Toxic SAMS require medication adjustment and cardiovascular risk management, whereas IMNM and dermatomyositis necessitate prompt immunosuppressive therapy and long-term follow-up. Early identification of autoimmune disease improves muscle recovery, reduces disability, and enables appropriate management of systemic complications.

DISCUSSION

The differentiation between toxic statin myopathy, statin-associated immune-mediated necrotizing myopathy, and dermatomyositis is clinically important because these conditions may initially present with similar symptoms but require substantially different management. Proximal muscle weakness, myalgia, and elevated creatine kinase may occur in all three entities. Therefore, the diagnosis should not be based on statin exposure alone. A history of statin use may explain muscle symptoms, but it may also obscure the diagnosis of autoimmune myopathy if persistent weakness is incorrectly attributed to drug toxicity.

The clinical course after statin withdrawal is one of the most useful practical indicators. Toxic statin myopathy usually improves after discontinuation or dose reduction of the drug, with gradual resolution of symptoms and normalization of creatine kinase. In contrast, persistent or progressive weakness after statin withdrawal should be regarded as a warning sign. This pattern is particularly important for statin-associated immune-mediated necrotizing myopathy, in which the autoimmune process continues independently of further statin exposure. In dermatomyositis, the absence of improvement after stopping statins also supports the need for a broader diagnostic evaluation rather than repeated changes in lipid-lowering therapy.

Serological testing has a central role in this differential diagnosis. Anti-HMGCR antibodies strongly support the diagnosis of statin-associated immune-mediated necrotizing myopathy in the appropriate clinical context. Myositis-specific and myositis-as-

sociated autoantibodies may support the diagnosis of dermatomyositis and help identify clinically relevant phenotypes, including those associated with interstitial lung disease or increased malignancy risk. However, antibody testing should be interpreted together with clinical findings, creatine kinase dynamics, muscle imaging, and, when necessary, muscle biopsy. No single test is sufficient in all patients.

Dermatomyositis requires particular attention because its diagnosis has implications beyond muscle disease. Characteristic skin manifestations, dysphagia, interstitial lung disease, constitutional symptoms, and malignancy risk distinguish dermatomyositis from uncomplicated toxic statin myopathy. Adult-onset dermatomyositis requires structured malignancy screening, especially near the time of diagnosis, because cancer-associated dermatomyositis may influence both prognosis and therapeutic strategy. Failure to recognize this association may delay cancer detection and treatment of systemic complications. For this reason, patients with suspected dermatomyositis should not be managed only as cases of statin intolerance.

Muscle MRI and histopathology are useful when clinical and serological findings are inconclusive. MRI may demonstrate muscle edema suggestive of active inflammatory involvement and help select an appropriate biopsy site. Muscle biopsy can help distinguish necrotizing myopathy from dermatomyositis and from nonspecific toxic muscle injury, although interpretation depends on the selected muscle, disease stage, and previous treatment. These investigations are especially valuable in patients with persistent creatine kinase elevation, severe weakness, negative or unavailable antibody testing, or atypical clinical presentation.

The main limitation of this review is its narrative design. No formal systematic review protocol was used, and the selection of studies was based on clinical relevance rather than a predefined quantitative synthesis. Therefore, the conclusions should be interpreted as a practical clinical synthesis rather than as evidence from a systematic review or meta-analysis. Nevertheless, the reviewed literature supports a stepwise approach based on clinical course, creatine kinase response, autoantibody testing, imaging, and biopsy when indicated.

In everyday practice, the most important diagnostic error is to assume that all muscle symptoms in statin-treated patients are toxic and reversible. The opposite error is also possible: overdiagnosis of autoimmune myopathy may expose patients to unnecessary immunosuppression and may lead to inappropriate discontinuation of effective lipid-lowering therapy. A balanced diagnostic strategy is therefore required. Patients whose symptoms improve after statin withdrawal can usually be managed by modifica-

tion of lipid-lowering therapy. Patients with persistent weakness, sustained creatine kinase elevation, positive myositis-specific or myositis-associated autoantibodies, cutaneous signs, or systemic involvement require specialist evaluation and timely treatment.

Future studies should improve diagnostic algorithms for patients with muscle symptoms during statin therapy, define more precise thresholds for further autoimmune evaluation, and clarify safe lipid-lowering strategies in patients with inflammatory myopathies. Better evidence is also needed on the timing and sequence of antibody testing, MRI, biopsy, and malignancy screening in real-world clinical practice.

CONCLUSION

Muscle symptoms during statin therapy should not automatically be attributed to reversible toxic statin myopathy. Although most cases of statin-associated muscle symptoms improve after statin withdrawal or modification of lipid-lowering therapy, persistent proximal weakness, sustained creatine kinase elevation, positive anti-HMGCR antibodies, myositis-specific autoantibodies, cutaneous manifestations, or systemic involvement should prompt evaluation for autoimmune myopathy.

A stepwise diagnostic approach based on clinical course, laboratory testing, autoantibody assessment, muscle imaging, and, when necessary, muscle biopsy can help distinguish toxic statin myopathy from statin-associated immune-mediated necrotizing myopathy and dermatomyositis. This distinction has direct therapeutic consequences. Toxic statin myopathy usually requires adjustment of lipid-lowering therapy, whereas immune-mediated necrotizing myopathy and dermatomyositis require timely and often prolonged immunosuppressive treatment.

Dermatomyositis should be recognized as a systemic autoimmune disease rather than only a muscle disorder. Patients with suspected or confirmed dermatomyositis require assessment for extramuscular complications, including interstitial lung disease and dysphagia, as well as structured malignancy screening, especially near the time of diagnosis. Early and accurate differentiation between toxic and autoimmune muscle disease may reduce diagnostic delay, prevent inappropriate treatment decisions, and improve clinical outcomes.

DISCLOSURES

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Use of Artificial Intelligence

The authors would like to acknowledge the use of artificial intelligence tools for language editing and improvement of grammatical accuracy. All content was critically reviewed and supervised by the author to ensure scientific accuracy and logical consistency.

Funding

This research received no external funding.

Conflict of Interest

The authors declare no conflicts of interest.

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