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DERMATOMYOSITIS MIMICKING STATIN-INDUCED MYOPATHY IN A 62-YEAR-OLD WOMAN

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ABSTRACT

Dermatomyositis (DM) is an autoimmune inflammatory myopathy that may rarely be drug induced. Statin-induced dermatomyositis (DIDM) is uncommon, with only few atorvastatin-related cases described in the literature. We present a case of a 62-year-old Hispanic woman who developed progressive proximal muscle weakness, myalgias, and joint discomfort shortly after initiating atorvastatin therapy. Despite discontinuation of the statin, her symptoms persisted and were accompanied by new cutaneous lesions. Laboratory evaluation revealed marked elevation of muscle enzymes, and subsequent skin biopsy revealed lymphocytic perivascular and perifollicular infiltrated with dermal mucin. Autoimmune serologies were negative, and malignancy screening was unremarkable. Treatment with systemic corticosteroids and mycophenolate mofetil resulted in gradual improvement. This report underscores the diagnostic challenges in differentiating statin-induced rhabdomyolysis from inflammatory myopathy, highlights the potential for rapid onset of dermatomyositis after statin exposure, and expands the limited literature on DIDM in Hispanic patients. By sharing this case, we aim to raise awareness among clinicians of this rare but clinically significant association, emphasizing the need for early recognition, histopathology, and malignancy screening to optimize outcomes.

KEYWORDS

Dermatomyositis, drug-induced, statin, diagnostic challenge

LEARNING POINTS

- Statin-induced dermatomyositis is a rare immune-mediated complication that may mimic rhabdomyolysis early in its presentation.
- Lack of clinical or biochemical improvement after statin withdrawal and intravenous hydration should raise suspicion for inflammatory myopathy.
- Cutaneous findings, seronegative myositis profile, and skin biopsy features are key to distinguishing dermatomyositis from necrotizing autoimmune myopathy.

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INTRODUCTION

Dermatomyositis (DM) is a systemic autoimmune disease belongs to the idiopathic inflammatory myopathies, characterized by cutaneous manifestations such as Gottron's papules, heliotrope rash, the "shawl sign", "V-sign" and "mechanic's hands"[1]. It exhibits a bimodal age distribution, peaking in childhood and in adults aged 50-69 years, with an incidence of 0.1-1.8 cases per 100,000 per year[2].

Drug induced dermatomyositis (DIDM) is a rare but clinically significant subtype of dermatomyositis, most often linked to hydroxyurea, statins, immune checkpoints inhibitors, and tumour necrosis factor-alpha (TNF-α) inhibitors[1]. Proposed mechanisms involve immune activation, overexpression of type I interferon, and complement-mediated microvascular injury, ultimately leading to inflammation in the skin and muscle tissues [1].

Laboratory evaluation typically includes elevated creatine phosphokinase (CPK), aldolase, and myositis-specific or myositis-associated antibodies, though a seronegative profile does not exclude diagnosis[3]. Furthermore, clinical and histopathologic correlation is need it for diagnosis[3]. Importantly, dermatomyositis may function as a paraneoplastic syndrome in adults, necessitating a thorough malignancy workup[3]. First line therapy consists of systemic corticosteroids, often combined with methotrexate, azathioprine, or mycophenolate[3].

We present the case of a 62-year-old Hispanic woman with dermatomyositis, initially suspected of having statin-induced rhabdomyolysis. Her case underscores the diagnostic challenges posed by overlapping clinical presentations.

CASE DESCRIPTION

We report the case of a 62-year-old Hispanic woman with a medical history of hypothyroidism, dyslipidaemia, and psoriasis, who presented with a two-week history of progressive myalgias, pelvic and lower back pain, joint discomfort, and proximal muscle weakness. She denied fever or other infectious symptoms. Two weeks earlier, she began using atorvastatin 10 mg, after which she developed her symptoms. Her primary care physician noted a high creatine kinase (CK) level and discontinued the statin, suspecting statin-induced rhabdomyolysis. However, her symptoms persisted despite cessation of the drug.

On admission, laboratory evaluation revealed CK 8,307 U/l, aldolase 32.6 U/l, aspartate aminotransferase (AST) 283 U/l, alanine aminotransferase (ALT) 138 U/l, erythrocyte sedimentation rate (ESR) 41mm/h, and C-reactive protein 1.5 mg/dl. She received intravenous fluids, but CK levels remained elevated, raising suspicion for an inflammatory myopathy. During hospitalization, she developed cutaneous lesions on the forehead, upper back, and legs. Physical examination revealed a 6x5 cm erythematous plaque with scale on the upper back, as well as scattered erythematous maculopapular lesions on the forehead and lower extremities *(Fig. 1)*.

A shave biopsy of the upper back lesion was performed. Histopathology revealed a superficial and deep perivascular and perifollicular lymphocytic infiltrate within the dermis, with abundant dermal mucin separating collagen bundles *(Fig. 2)*. The overlying epidermis was largely preserved, with focal basal vacuolar interface change and a single focus of parakeratosis with neutrophils in the stratum corneum *(Fig. 3)*. Higher magnification confirmed perifollicular inflammation without follicular destruction or vasculitis.

Autoimmune serologies, including anti-GBM, myositis panel, SS-A/Ro IgG, and SS-B/La IgG, were negative. Complement levels were within normal limits (C3: 95 mg/dl, C4: 19 mg/dl). Malignancy screening with upper endoscopy and colonoscopy revealed no evidence of gastrointestinal cancer. She was started on prednisone 30 mg BID, followed by mycophenolate mofetil 1000 mg BID, and showed gradual improvement in systemic symptoms and skin lesions.

DISCUSSION

Although statins are widely prescribed and generally well tolerated, they have been associated with complication including dermatomyositis, with only isolated cases reported in the literature. In our patient, the temporal association with statin initiation suggests a drug-induced mechanism. This case highlights an uncommon presentation of DM in a 62-year-old Hispanic woman, initially suspected of having statin-induced rhabdomyolysis.

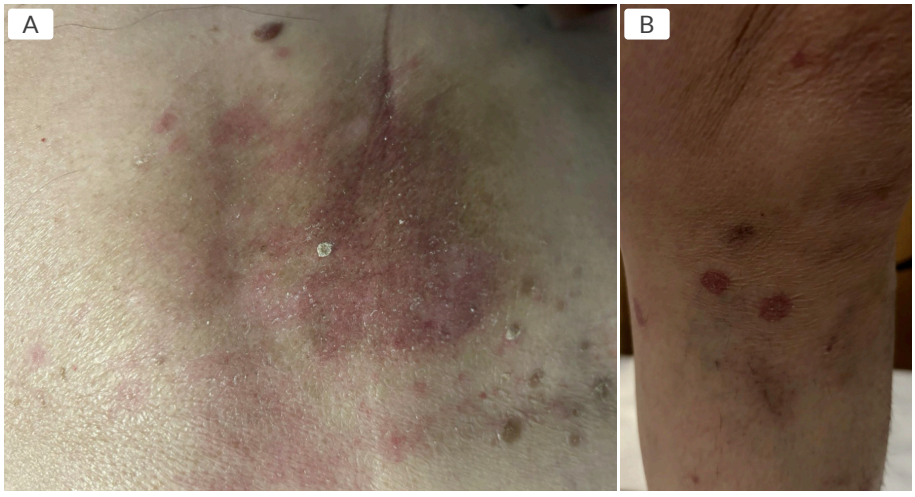


Figure 1. *A) Erythematous to violaceous plaque with mild scale on the central upper back. B) Several well-demarcated round to purpuric macules on the right upper leg.*

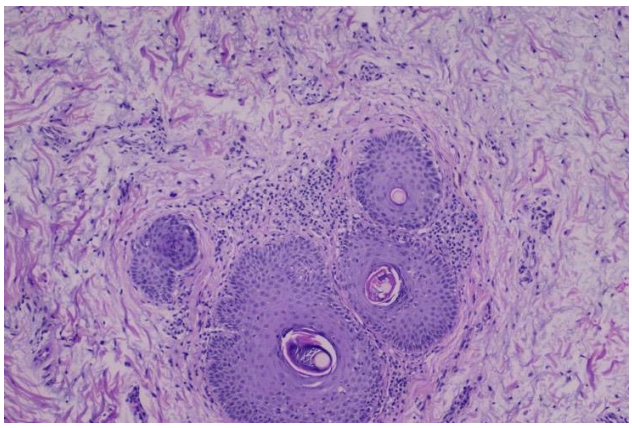


Figure 2. Histology shows prominent perifollicular lymphocytic infiltrate and abundant dermal mucin deposition separating collagen bundles.

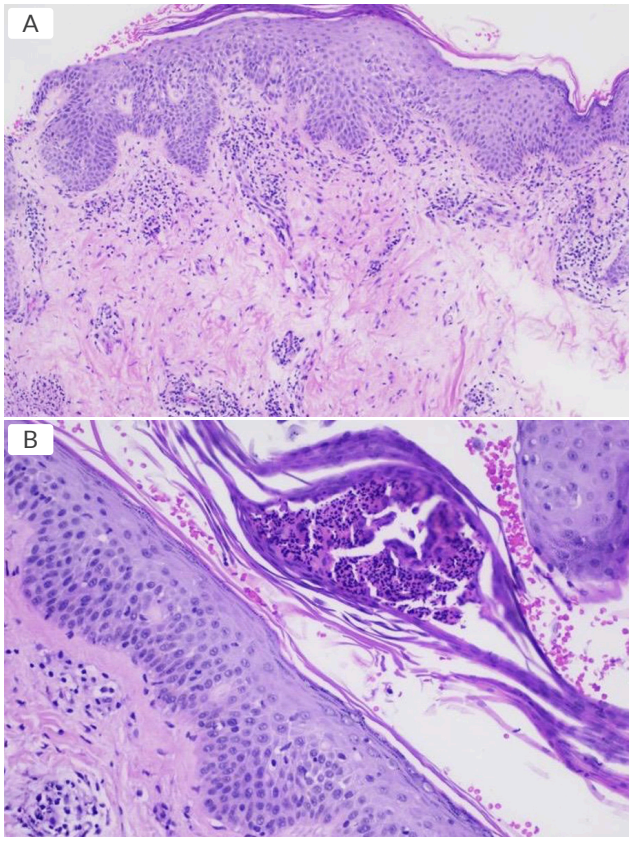


Figure 3. A) Skin biopsy demonstrates superficial and deep perivascular and perifollicular lymphocytic inflammation, with abundant dermal mucin separating collagen bundles. B) Higher magnification of skin biopsy shows focal basal vacuolar interface change and a single focus of parakeratosis with neutrophils in the stratum corneum.

The rarity of this presentation is further underscored by the limited literature on DIDM. To date, no population-based incidence or prevalence data exists of this subtype, and the evidence is confined to systematic reviews and case reports. A recent systematic review analysed 69 DIDM cases, of which only 6 were statins-induced. Among these, simvastatin was the most reported agent (3 cases), followed by atorvastatin (2 cases) and pravastatin (1 case)[4]. Additionally, Chemello et al. reported an aggressive and fatal case of statin-induced dermatomyositis, supporting the concept that statins may act as immune triggers rather than causing direct muscle toxicity alone[5]. Given the global use of statins, these small numbers of reported cases highlight both the exceptional nature of this diagnosis and the importance of clinician awareness for early recognition.

During hospitalization, our patient's CK levels remained markedly elevated despite aggressive intravenous fluid resuscitation. In uncomplicated rhabdomyolysis, removal of the inciting factor and adequate hydration typically lead to clinical improvement within 24-48 hours. Failure of CK levels to decline with appropriate hydration suggests ongoing muscle injury and should prompt reconsideration of the diagnosis. Prior reports emphasize that persistent CK elevation following statin discontinuation and hydration should raise concern for an autoimmune myopathy[6]. In our patient, the lack of biochemical and clinical improvement with hydration alone, followed by clear response to immunosuppressive therapy, strongly argues against isolated rhabdomyolysis and supports the diagnosis of drug-induced dermatomyositis.

Cutaneous manifestations provided key diagnostic clues. Although Gottron papules and heliotrope rash were absent, our patient exhibited signs suggesting the shawl sign and non-specific erythematous macules on the face and extremities, consistent with reported DM findings[7]. Skin biopsy revealed basal vacuolar change, lymphocytic infiltrates, dermal mucin, and perifollicular/perivascular inflammation, consistent with early histologic features of DM[8].

Serologic testing was negative for myositis-specific and myositis-associated antibodies, aligning with prior reports noting anti-Jo-1 positivity in only 8.5% of DM patients and highlighting that 20-30% of cases may be seronegative[9]. Thus, our patient's diagnosis relied on clinicopathologic correlation, integrating skin findings, histopathologic evidence, and elevated muscle enzymes. Given the strong association between DM and malignancy, particularly in the first 5 years after diagnosis, our patient underwent a comprehensive malignancy evaluation, all of which were negative[5]. Treatment with prednisone 30 mg BID and mycophenolate mofetil 1,000 mg BID led to improvement of systemic findings and skin lesions.

When comparing our patient's presentation with the case described by Oztas et al.[10], several important similarities and differences emerge. Our patient developed proximal muscle weakness, joint discomfort, and myalgias initially, with cutaneous manifestations appearing later during hospitalization. In contrast, the 49-year-old man reported by Oztas et al. developed skin lesions concurrently with muscle weakness, highlighting differences in clinical chronology[10]. Despite these distinctions, both cases demonstrated perivascular lymphocytic infiltration on histopathology and were diagnosed with atorvastatin-associated dermatomyositis at a dose of 10 mg/day. Notably, the latency from statin initiation differed: symptoms developed within two weeks in our patient, whereas

onset occurred approximately three months after statin initiation in the reported case. Additionally, our patient was negative for all tested autoimmune serologies, reinforcing that a seronegative profile does not exclude drug-induced dermatomyositis, as noted in prior reports. Together, these observations underscore the heterogeneity of drug-induced dermatomyositis and the diagnostic challenges in recognizing statins as a potential immune trigger.

In summary, this case expands the limited literature on statin-associated dermatomyositis in Hispanic patients. The short latency from atorvastatin initiation further distinguishes this case, underscoring that DIDM can develop rapidly and without classical serologic markers. Together with histopathologic confirmation, this report broadens the spectrum of DIDM presentations and emphasizes the importance of clinicopathologic correlation. Early recognition remains critical to ensure timely treatment and malignancy screening.

CONCLUSION

This case highlights the diagnostic complexity of differentiating statin-induced rhabdomyolysis from dermatomyositis and broadens the spectrum of reported statin-induced dermatomyositis. It emphasizes the need for early recognition, clinicopathologic correlation, and malignancy screening to ensure timely treatment and improved outcomes.

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