A case of psoriasis worsened by atorvastatin

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Key words:

adverse events, atorvastatin, drug eruptions, psoriasis, statins

Abstract

Statins are known to have a number of cutaneous adverse effects including the induction of autoimmune diseases like systemic and subacute lupus erythematosus, dermatomyositis, polymyositis, lichen planus pemphigoides and the drug reaction with eosinophilia and systemic symptoms (DRESS). Statins have been also reported as a triggering factor of psoriasis. We report a case of psoriasis vulgaris that worsened three months after atorvastatin was introduced and improved after its discontinuance.

Introduction

A wide number of drugs can induce psoriasis de novo or precipitate pre-existing psoriasis. These drugs include lithium, synthetic antimalarials, tetracyclines, interferon, non-steroidal anti-inflammatory drugs, neuroleptics, certain anti-hypertensive drugs as β -adrenergic antagonists, Ca-antagonists and angiotensin-converting enzyme inhibitors and even tumor necrosis factor-alpha inhibitors.¹

Statins are known to have a number of cutaneous adverse effects including the induction of autoimmune diseases like systemic and subacute lupus erythematosus, dermatomyositis, polymyositis, lichen planus pemphigoides^{2,3} and the drug reaction with eosinophilia and systemic symptoms (DRESS).⁴ Statins have been also reported as a triggering factor of psoriasis.⁴ We report a case of psoriasis vulgaris that worsened three months after atorvastatin was introduced and improved after its discontinuance.

To our knowledge, this is the first report of psoriasis worsened after atorvastatin administration.

Case report

A 47 year-old patient, without family history of psoriasis

had moderate psoriasis vulgaris for ten years treated with topical therapy. He was also treated with Ca-antagonists and ACE-inhibitors for his hypertension. On examination, erythematous-scaling infiltrated plaques of psoriasis with regular margins were observed on his trunk and arms and scaling lesions were present on his palms and plants. PASI value was 6,8. Laboratory tests disclosed hypercholesterolemia. PCR, VES ,TASL, urinalysis and throat swabs were normal. Atorvastatin (20 mg/d) was then initiated. Three months later, the skin lesions on his arms had severely worsened, had become itchy and were accompanied by new plaques on his legs. Onicopathy had worsened as well and PASI had risen to 12,3. Atorvastatin was then replaced by rosuvastatin (10 mg/d). Two months later, the lesions had improved without any modification of the previous topical medication.

Discussion

Statins are widely used in the prevention of hypercholesterolemia and well known to have a number of cutaneous adverse effects, including the induction of autoimmune diseases like systemic and subacute lupus erythematosus, dermatomyositis, polymyositis, lichen planus pemphigoides^{2,3} and the drug reaction with eosinophilia and

Table 1

Values of PASI (psoriasis area and severity index) and BSA (body surface area) before, during atorvastatin therapy and after treatment discontinuation.

| | PASI | BSA |
|-----------------------|------|-----|
| before atorvastatin | 6,8 | 4 % |
| during atorvastatin | 12,3 | 9 % |
| after discontinuation | 1,0 | 1% |

systemic symptoms (DRESS).⁴ In these cases the mechanism of atorvastatin seems to be due to the singlet oxygen formation which causes photobiological damage⁵, but the underlying mechanism by which statins elicit DRESS remains largely unknown. Only one case has been reported in which pravastatin induced or worsened psoriasis.⁶ Ours therefore is the second case described. In fact, psoriasis had worsened concomitantly to the introduction of atorvastatin and improved 3 months after its discontinuation. A role of the antihypertensive drugs, well known as psoriasis inducers, was ruled out, since our patient was taking them for several years without psoriasis growing worse. Others triggering factors such as infections or stress, were excluded as well on the basis of his normal blood tests.

How statins activates psoriasis is unclear, but a mechanism of activation of STAT3, a molecule that delivers antiapoptotic signals to epidermal keratinocytes, has been suggested.⁵

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