A Case of Psoriasis Vulgaris Aggravated with Atorvastatin, Aided by Concomitant Cyclosporine

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Sir,

We report a case of 50-year-old woman, who developed aggravation of her preexisting stable psoriasis vulgaris on introducing atorvastatin to control her recently detected hypercholesterolemia, the aggravation seemed to be aided by concomitant cyclosporine (CsA).

Our patient had intermittent psoriasis vulgaris for the past 3 years. Oral methotrexate and topical clobetasol and calcipotriol were of slight help, but she responded best with CsA 100 mg twice daily which she took regularly for the past 10 months and her disease was stable with no recurrence or aggravation since then. She is also a known case of hypertension (on telmisartan 40 mg/day) and diabetes mellitus (on metformin, glicazide, and...
pioglitazone) for 7 years and there was no change in these medications for the past 2 years. She also had Class I obesity (body mass index-33.6 kg/cm²). During routine blood investigations done a month ago, she was found to have elevated cholesterol (borderline high) for which she was started on atorvastatin 40 mg daily by the physician. Ten days later, she presented with diffuse erythematous scaly plaques and exfoliation on palms and soles [Figures 1-3]. All the aggravating factors, including trauma, physical factors (surgery, radiation, tattoos, insect bites, and burns), chemical factors (topical psoriasis medications), endogenous factors (HIV, upper respiratory tract infection) as well as medications (beta-blockers and others) were ruled out. We suspected exacerbation of psoriasis due to changed medication, and hence we stopped atorvastatin but maintained CsA in the same dose, and she recovered fully within 3 weeks. She had taken atorvastatin in the past before CsA was started, without any cutaneous reaction to the same.

Statins are a group of drugs with a beneficial effect on lowering of lipid levels by causing inhibition of HMG-CoA reductase. They promote Th1 to Th2 cells, cause inhibition of various pro-inflammatory cytokines and impaired infiltration of lymphocytes into inflammatory sites. Statins also have an inhibitory effect on CCL20/CCR6 chemotactic interaction, thereby having a beneficiary effect in psoriasis pathogenesis. However, statins have also been associated with several cutaneous adverse effects. It can cause triggering of auto-immune diseases such as lichen planus pemphigoides, systemic lupus erythematosus, dermatomyositis, Drug Reaction with Eosinophilia and Systemic Symptoms (DRESS) and rarely psoriasis. There is one case report of a patient with psoriasis vulgaris whose disease worsened 3 months after starting atorvastatin and improved after its discontinuation. The possible pathomechanism could be formation of singlet oxygen, responsible for photobiological damage or activation of STAT3, a molecule causing transmission of anti-apoptotic signals to epidermal keratinocytes. Significant cutaneous reactions have been reported when statins are used along with other drugs having potential interactions. The common drugs affecting statin metabolism are CsA, macrolides, azole antifungals, fibrates, warfarin, and digoxin.

Both CsA and atorvastatin are primarily metabolized by cytochrome P4503A4 and hence drug interaction between the two is likely to be seen. It has been observed that CsA may lead to significantly higher (approximately six-fold) atorvastatin plasma HMG-CoA reductase inhibitor activity when used concomitantly, whereas atorvastatin causing reduced systemic exposure of CsA by about 10%.

Our patient, a stable case of psoriasis on CsA therapy for 10 months without any changes or new additions in her medications, had a sudden aggravation within 10 days after starting atorvastatin. This could be explained by decreased clearance of atorvastatin and hence its enhanced effect as well as decrease in CsA efficacy due to interactions between both. To the best of our knowledge, this is the first case of such an acute exacerbation following the introduction of atorvastatin in a well-controlled psoriasis patient and CsA playing an important role in this exacerbation. The concomitant use of atorvastatin and CsA should be avoided, and when used, the patient should be monitored for any unexplained muscle weakness or tenderness, along with hepatic and renal investigations. Fluvastatin (40 mg/day) and pravastatin (20 mg/day) can be used as an alternative as they are not extensively metabolized by CYP3A4.

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**Conflicts of interest**

There are no conflicts of interest.

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