Simvastatin in Psoriasis: Ambiguous Effects

Andreas Colsman and Michael Sticherling
Department of Dermatology, University Erlangen-Nürnberg, Hartmannstr. 14, DE-91052 Erlangen, Germany. E-mail: Andreas.Colsman@uk-erlangen.de
Accepted January 7, 2010.

The incidence of metabolic disorders, including dyslipidaemia, is increased among psoriatic patients and has been addressed extensively in recent years in the context of epidemiological studies of disease comorbidity. In this regard, the early and effective treatment of both chronic inflammatory activity as well as metabolic imbalance has been advocated. As statins are clinically well-established in the treatment of lipid disorders, they are as likely to be used in psoriatic as in non-psoriatic patients. Apart from their metabolic activity, statins have been shown to modulate various immunological parameters (1–3), including the activation of T-regulatory cells and their migration into inflammatory tissue (4). Positive effects of statins in chronic inflammatory diseases such as Crohn’s disease have been shown in the past (5). Based on these results, an improvement in psoriasis may be anticipated. So far, however, only a few clinical reports have been published. Studies on psoriasis describe an exacerbation of the disease (6). A single report of a pilot study shows a considerable improvement in psoriasis with monotherapy with simvastatin (7). We therefore investigated this aspect in a pilot study, by treating patients with both dyslipidaemia and psoriasis with oral simvastatin at 40 mg/day while monitoring disease activity and laboratory parameters.

MATERIAL AND METHODS
In five patients with moderate to severe plaque psoriasis (mean Psoriasis Area Severity Index (PASI) 11.4) and coincident hypercholesterinaemia, therapy with simvastatin was started after a 4-week wash-out period of systemic anti-psoriatic therapy if necessary. Local therapy with topical calcipotriol in combination with corticosteroids (mometasone or betamethasone valerate) was continued. The observation period was 12 weeks. During the treatment period clinical changes were monitored by the same dermatologist sequentially evaluating the PASI. In addition, laboratory tests were performed including very low-density lipoproteins, low-density lipoproteins, high-density lipoproteins, total cholesterol, triglycerides, C-reactive protein (CRP), parameters of liver and renal function and blood count.

RESULTS AND DISCUSSION
In four patients we observed a small but not significant reduction in PASI during their treatment (Fig. 1). In one patient the PASI deteriorated temporarily by 50% from 20 to 29, and in another patient from 7 to 10. Altogether, after 12 weeks of treatment the PASI was almost unchanged from baseline (Fig. 1). At the same time, the lipid parameters improved and the CRP decreased. The statin treatment was well tolerated with no side-effects detected in laboratory examinations or reports by the patients.

It remains a matter of debate whether psoriasis deteriorates hypercholesterolaemia or vice versa. Inflammation may, however, be the common denominator of both. Based on our results on a small number of patients, statin therapy was both well tolerated and effective for dyslipidaemia, but without significant effects on psoriasis with regard to either deterioration or improvement in the skin disease. At the same time, we could not confirm earlier negative casuistic experiences published in the literature. Further research, including clinical double-blind trials with a higher number of patients, is required.

REFERENCES