loricrin, and filaggrin in cultured sebocytes.
In conclusion, cultured sebocytes showed little expression of keratinocyte differentiation markers. Weak physical barrier in the sebaceous gland may cause much better transfollicular drug delivery.

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Myocardial Infarction in a Patient Treated with Anti-Interleukin-12 Biological Agent for Chronic Plaque Psoriasis

Giuseppe Stinco, Cinzia Buligan, Serena Bergamo, Olvino Morgante[1], Maria Antonietta Iacono[1], Pasquale Patrone

Institute of Dermatology, Department of Experimental and Clinical Medicine, University of Udine, Udine,
[1]Division of Internal Medicine, Azienda Ospedaliera di Gemona, A.S.S., Alto Friuli, Gemona del Friuli, Italy

Dear Editor:
We present the case of a 55 year-old woman affected by psoriasis since she was 40 years old. Her personal history reveals hepatic steatosis, hypertension, dyslipidemia, obesity, and gastro-esophageal reflux disease. She had been smoking 8 cigarettes a day for 30 years and she occasionally consumed alcohol. In 2005, she was treated with Cyclosporine, which was stopped after an episode of

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Corresponding author: Giuseppe Stinco, Institute of Dermatology, University of Udine, Ospedale "San Michele" di Gemona piazza Rodolone 1, Gemona del Friuli (Udine) 33013, Italy. Tel: 39-0432-989378, Fax: 39-0432-989209, E-mail: giuseppe.stinco@uniud.it

transient ischemic attack after five months of therapy. In 2009, she started Adalimumab phial 40 mg every 2 weeks for 8 months. About 9 months later, Adalimumab was restarted and stopped after 6 months due to inefficacy. She underwent narrow-band ultraviolet B phototherapy without any clinical benefit; it was therefore decided to start her on Ustekinumab 45 mg fl. The patient performed the first administration of the drug and after six days, she suffered from an acute myocardial infarction. The coronary angiography demonstrated a two-vessel coronary artery disease. It is not possible to absolutely define the relationship between this cardiovascular event and the Ustekinumab intake. While she clearly had several important cardiovascular risk factors, she was never referred for angina. However, the coincidence with the first dose of the biologic drug cannot be overlooked. A pathogenetic hypothesis to explain the possible higher prevalence of major adverse cardiovascular events (MACEs), especially at the start of treatment, might be related to the stabilizer role of interleukin (IL)-17 on the atheromatous plaque[1]. Nevertheless, the pro-inflammatory effect of Th1 and Th17 cytokines on the plaque development and rupture has been demonstrated; especially, the link between IL-17 and the instable atheromas could sustain the increased risk of acute myocardial infarction in psoriatic patients[2]. Also, clinical data are controversial: a meta-analysis of pooled data from phase II/III clinical studies on Ustekinumab reported a non-significant difference in the effect of the biological drug on cardiovascular events between the treated psoriatic patients and the placebo group[3]. Another meta-analyses of randomized controlled trails in psoriatic patients, investigating the association between anti-IL-12/23 agents (Ustekinumab and Briakinumab) and MACEs did not show a significant increase in the risk of MACE, even though 10 events in 3,179 treated patients and 0 events in 1,474 control patients were observed[4]. On the other hand, a significant difference was reported in the rate of MACEs observed in patients receiving anti-IL-12/23 agents[5]. Briakinumab and Ustekinumab are both human monoclonal antibodies targeting the same shared sub-unit (p40) of IL-12 and IL-23; a class effect therefore cannot be excluded. However, because Briakinumab is more responsible for major cardiovascular effects than Ustekinumab, it was withdrawn from the market. Our report demonstrates that a single injection of the biologic drug might have promoted a myocardial infarction. Due to the quick interval between the drug intake and the appearance of MACE, we focused our attention on the short term effect rather than the cumulative effect; the literature describes the temporal interval ranges from 2 to 17 weeks (mean: 9,8)[3]. If meta-analyses have not been able to clarify the issue, it is unlikely that a single report could provide further clarification. However, it is important to maintain awareness of this issue and in the presence of cardiovascular risk factors attention should be taken when using this drug.

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