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Vitiligo, psoriasis and imiquimod: Fitting all into the same pathway

Sir,

I read with interest the letter describing imiquimod-induced vitiligo-like depigmentation.[1] There are other similar case reports[2,3] and 68 reports of pigmentary changes related to imiquimod use listed by FDA.[4] The most widely accepted theory is that imiquimod activates the cell-mediated arm of the immune system via the stimulation of multiple pro-inflammatory cytokines which up-regulates a Type-1 cell response (Th1) that unmasks an innate predilection for the development of vitiligo.[5]

The mode of action of imiquimod involves activation of Toll-like receptors (TLR) 7 and 8, which initiates a signaling cascade leading to the production of pro-inflammatory cytokines like IFN-α and TNF-α. However, TLR 7 and 8 also initiates ubiquitin-mediated proteolysis and apoptosis through the myeloid differentiation factor 88-dependent pathway as evident from the pathway map[6] hsa04620 available from KEGG.[7] In a previous letter to IJDVL, I presented the genomic evidence for ubiquitin-mediated melanocyte-specific apoptotic process in vitiligo.[8] Hence, the ubiquitin-mediated proteolysis may be important in the pathogenesis of vitiligo-like lesions following treatment with imiquimod.

A Th1 type response with increased TNF-α is vital in the pathogenesis of psoriasis.[9] Hence, it is hardly surprising that there are several reports of imiquimod-induced psoriasis.[10-13] Psoriasis is a proliferative disorder while vitiligo is an apoptotic disorder. It is interesting to note that these disparate conditions converge at a fundamental pathway level.

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