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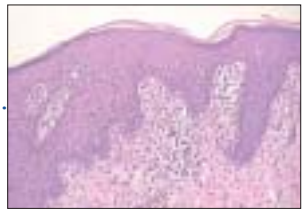

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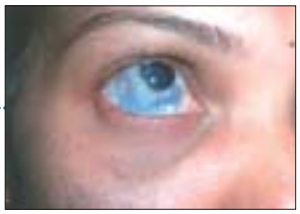

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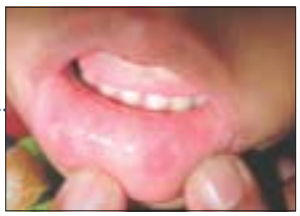

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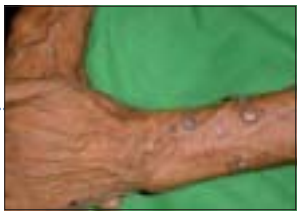

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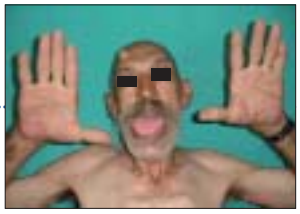

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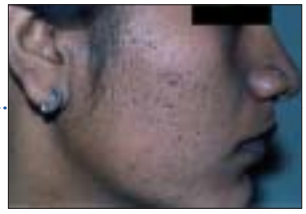

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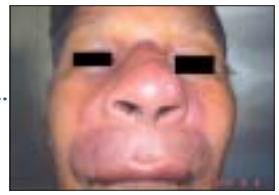

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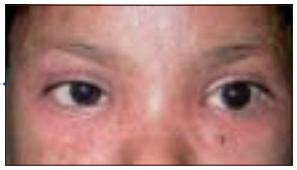

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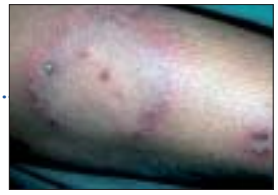

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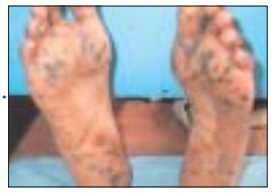

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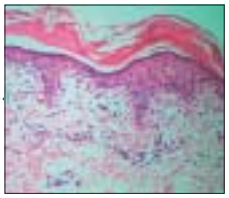

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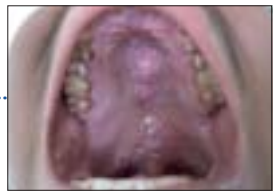

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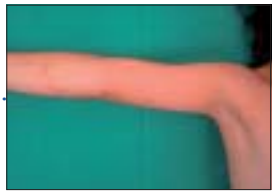

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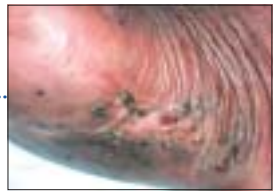

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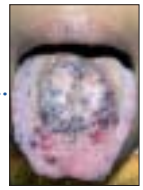

Author's reply

Sir,

We appreciate the comments of Dr. Eapen on our letter.[1] In his reply, it is emphasized that enhanced activation of Toll-like receptors 7 and 8 due to imiquimod treatment may trigger the ubiquitin-mediated proteolysis and apoptosis, and this mechanism may have a role in the pathogenesis of imiquimod-related vitiligo-like depigmentation.

Besides IFN-α and TNF-α, imiquimod treatment leads to the increased production of various other cytokines and chemokines. Along with IFN-α, increased production of IL-6 and IL-8 by imiquimod stimulates the cytotoxic T-cell-mediated immune response, which is known to play a major role in the pathogenesis of vitiligo.[2] Furthermore, some of the cytokines triggered by imiquimod, namely IL-6, IL-1 and TNF-α, cause a dose-dependent decrease in the activity of the enzyme tyrosinase, suggesting a role for paracrine and possibly autocrine regulation of melanocytes by immune modulators. IL-6 can also increase melanocyte ICAM-1 expression, which

may increase the leukocyte-melanocyte attachment and result in melanocyte damage in vitiligo. This IL-6-induced ICAM-1 expression may also be the triggering factor in imiquimod-induced vitiligo-like depigmentation.[3] It is also possible that imiquimod-induced production of TNF-α and IFN-α plays a role in auto-destruction of melanocytes by enhancing the release of nitric oxide.[4] In summary, considering the mechanisms of actions of imiquimod, vitiligo-like depigmentation is not a surprising adverse effect of this drug.

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