Imatinib Mesylate Induced Skin Hypopigmentation

Sir,

Imatinib mesylate is a tyrosine kinase inhibitor that targets BCR-ABL protein in chronic myelogenous leukemia (CML).1 We present three cases of CML, treated with Imatinib who developed clinical findings of hypopigmentation. All the three patients noticed pigmentation changes after the initiation of therapy with Imatinib mesylate, which was persistent during therapy. The presence of hypopigmentation did not appear to predict or alter either leukemia cell response or clinical outcome.1 All the patients achieved a hematologic response but only two patients achieved a complete cytogenic response. Imatinib mesylate induced hypopigmentation appears to be reversible and potentially dose related and increases with dose escalation. Patients experienced fluctuation in the intensity of hypopigmentation on interruption or reinitiation of the drug and conversely an increase on dose escalation. The exact mechanism of hypopigmentation is unclear at present. The signal transduction pathway is believed to involve the SCF ligand binding of KIT which in turn attributes to the melanocyte homeostasis and differentiation. Imatinib acts on ligand SCF which regulates melanocyte development and survival resulting in hypopigmentation.1 This is further supported by the observation that human mutations in the encoded tyrosine kinase region of KIT have shown to cause piebaldism, an autosomal dominant disorder, characterized by white hair and hypopigmented skin patches on the forehead, torso and extremities.2,3 This skin toxicity although cosmetically a problem, does not pose a substantial medical hazard. Skin biopsy was considered but due to the above rational, we were unable to ethically justify subjecting these patients to the procedure. Future translational prospective studies with skin biopsies to further evaluate and confirm the mechanism of action of this skin toxicity is recommended.

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