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Case presentation

Nilontinib induced keratosis pilaris atrophicans

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Abstract

Keratosis pilaris (KP) is a disorder of follicular keratinization that is characterized by keratin plugs in the hair follicles with surrounding erythema. A 46-year-old man with chronic myelogenous leukemia (CML) was started on nilotinib, a second generation tyrosine kinase inhibitor (TKI). Two months later the patient noticed red bumps on the skin and patchy hair loss on the arms, chest, shoulders, back, and legs. Cutaneous reactions to nilotinib are the most frequent non-hematologic adverse effects reported. However, it is important to distinguish KP-like eruptions from more severe drug hypersensitivity eruptions, which can necessitate discontinuing the medication. Also, it is important to classify the cutaneous eruptions in patients on TKI according to the morphology instead of labeling them all as “chemotherapy eruption” to be able to better manage these adverse effects.

Keywords: nilotinib, keratosis pilaris atrophicans, tyrosine kinase inhibitors

Cutaneous reactions to nilotinib, a second generation tyrosine kinase inhibitor, are the most frequent non-hematologic adverse effect reported. Pruritus, rash, dry skin, and alopecia are observed with this drug, but specific rash morphologies have not been identified [1,2]. A case of bullous Sweet syndrome in a patient with chronic myelogenous leukemia on nilotinib has also been described [3]. Keratosis pilaris (KP) is a disorder of follicular keratinization and is characterized by keratin plugs within the hair follicles with surrounding erythema. An uncommon variant known as KP atrophicans occurs when there is follicular inflammation leading to scarring and alopecia. A KP like eruption has been observed in patients treated with sorafenib and is thought to relate to alterations in the keratinocyte differentiation and proliferation pathways [4].

A 46-year-old man with chronic myelogenous leukemia (CML) was started on nilotinib 400 mg twice daily after his disease failed to achieve complete cytogenetic remission after treatment with 12 months of therapy with imatinib. His other medications remained unchanged. Nilotinib induced molecular remission of CML; however, two months after its initiation the patient noticed red bumps on the skin and patchy hair loss in the affected areas. He also noted his skin texture changed and became more dry and rough. He had no personal or family medical history of keratosis pilaris or atopic dermatitis. Examination revealed follicular accentuation on the forehead and lateral thinning of the eyebrows. There were prominent perifollicular pink papules with areas of complete and partial hair loss on the arms, chest, shoulders, back, and legs (Figure 1).
There were no perifollicular petechiae or palmoplantar lesions. A punch biopsy from a lesion on the chest demonstrated prominent perifollicular fibrosis extending to the dermis suggestive of a KP-like eruption (Figure 2). Serum vitamin A and D levels were within normal limits. The patient was prescribed a topical ammonium lactate lotion and topical steroids for the inflammatory component. He reported improvement with topical agents at his three month follow up appointment.

KP is a common skin condition presenting as skin colored to bright red, follicular lesions commonly on the extensor surfaces of the arms, thighs, and face. Severe cases may involve the entire trunk as seen in our patient. The atrophic variants are typically associated with genodermatoses with variable degree of scarring and alopecia and are not usually seen as a medication side effect. This cutaneous adverse effect of nilotinib has not been reported. KP-like eruptions are usually mild and it is important to distinguish these eruptions from the more severe drug hypersensitivity eruptions, which may necessitate discontinuing the medication. It is also important to classify the cutaneous eruptions in patients on tyrosine kinase inhibitors according to the morphology instead of labeling them all as “chemotherapy eruption” to be able to better manage these adverse effects.
References


