Sir,

Imatinib mesylate, which was approved in 2001 for the treatment of chronic myeloid leukemia (CML) is considered as the wonder drug.\(^1\) It is a tyrosine kinase inhibitor which targets the BCR-ABL fusion, c-KIT, and PDGFR mutated gene products. Adverse drug reactions to it are fairly common, non-life threatening, and nonspecific which include xerosis, photosensitive/maculopapular rash, edema, lichenoid and psoriasiform eruptions, cutaneous vasculitis, urticaria, Stevens–Johnson syndrome etc.\(^2\) Here, we report for the first time, imatinib-induced classical lichen planopilaris and atrophic lichenoid plaques in blaschko-linear distribution in a patient with chronic myeloid leukemia.

A 27-year-old male patient was diagnosed with CML in November 2018 and was started on imatinib 400 mg/day. The overall tolerance to the drug has been good with significant improvement of physical wellbeing and hematological parameters. However, after three months of treatment, he started developing violaceous, atrophic, and alopecic plaques. The lesions started to appear over the frontal scalp adjacent to the hair line and then over the lateral aspect of the left eyebrow [Figure 1]. The scalp lesion had the classical morphology of lichen planopilaris in the form of alopecic, atrophic, violaceous, and scaly plaque. However, the left eyebrow lesions also had similar alopecic and violaceous plaques but no scaling. Gradually, he developed multiple, atrophic, nonscaly, and violaceous plaques over his forehead in a linear distribution and over the lateral aspect of left periocular area. He was counselled to continue imatinib, as it is a lifesaving drug. Upon subsequent follow-up after a month, he developed morphologically similar atrophic violaceous plaques over the medial aspect of the left upper eyelid. Overtime, the lesion morphology became blaschko-linear involving the left side of the frontal scalp, eyelid, and eyebrow.

Dermoscopy with DermLite DL3 dermatoscope (3Gen, San Jan Capistrano, CA, USA) attached to Sony Cybershot camera (DSC-W800 5x magnification) was performed, giving a magnification of 10x. The scalp lesion revealed a violaceous background and loss of hair follicles with multiple follicles showing peripilar cast, characteristic of lichen planopilaris [Figure 2]. A punch biopsy from the scalp lesion revealed perifollicular lymphohistiocytic infiltrate with pigment incontinence and a vertical follicular scar [Figure 3], consistent with lichen planopilaris. The patient was very concerned about these lesions and wanted to stop the offending drug imatinib. After consultation with the hemato-oncologist, his treatment regimen was changed. In addition, he was prescribed mometasone furoate 1% lotion for topical application. He was followed up till four months and his lesions did not progress nor did he develop any new lesion.

Discussion

Oral and cutaneous lichenoid eruptions are well established side effects of imatinib. To the best of our knowledge, this is the first case report detailing the development of classical lichen planopilaris in blaschko-linear distribution secondary to imatinib use. However, nilotinib-induced lichen planopilaris have been described on one occasion, where the patient developed lichen planopilaris-like lesions on arms

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within six weeks of therapy, although our patient had classical morphology. In a case series of nine patients with CML, initially, six patients were on imatinib therapy and three were started on dasatinib (one) and nilotinib (two). Five out of six patients on imatinib, developed resistance and one developed nondermatologic intolerance. Hence, all six were switched either to nilotinib or dasatinib. Within few days to months, all these patients developed lichen plano-pilaris like lesions on scalp, eyebrows, and arms. It is to be noted that none of the patients developed these lesions while on imatinib. One patient developed perifollicular scaling over the scalp but the cicatricial plaque was not seen. In contrast, our patient developed well-defined violaceous, cicatricial alopecic plaques with perifollicular scaling. In addition, he developed atrophic violaceous plaques over the forehead and eyelids which has never been described with the use of TKIs. The exact pathomechanism of lichenoid eruption has not been elucidated. Although, due to its low molecular weight, it is unlikely to be immunogenic rather, adverse reactions appear to be dose dependent and might be mediated by changes in tyrosine kinase signalling.

Cutaneous lesions by virtue of their visibility on exposed areas lead to significant psychological effects in already sick patients. It is important to be aware of these side effects, recognize them early, and treat accordingly. These patients need to be counselled not to stop the lifesaving drugs. We, hereby, report the first case of imatinib-induced classical lichen planopilaris in a blaschko-linear distribution leading to cicatricial alopecia.

**Declaration of patient consent**

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Nil.
Conflicts of interest

There are no conflicts of interest.

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