Correspondence

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undergo spontaneous resolution. Surgical excision is considered as first line of the treatment in symptomatic cases, though recurrence is common. [8]

Topical, intralesional, and systemic steroids are frequently used, but the swellings may become refractory to the treatment. [9]

Radiotherapy (20–45 Gy) is also effective, and cyclosporine (5 mg/kg/day) has shown good results in many studies. [8,9]

Imatinib mesylate is now being considered as an effective treatment for KD. As an inhibitor of the protein-tyrosine kinases (PTK), imatinib works by selectively blocking PTK, such as PDGFR and c-Kit. [10]

Azathioprine, all-trans retinoic acid, leflunomide, pentoxifylline, pranlukast, and intravenous immunoglobulin have been tried with variable response.

However, the prognosis of KD is excellent, and the disease has no potential for malignancy. This case is hereby reported, because of its rarity and excellent response to therapy.

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 The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.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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Atypical Targetoid Eruption Induced by Sorafenib in a Patient with Hepatocellular Carcinoma

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Sir,

A 38-year-old man was referred to our outpatient clinic with a persistent diffuse itchy eruption for 10 days. He was diagnosed with hepatocellular carcinoma 9 months ago. He had received sorafenib (800 mg/day, orally) for 3 weeks. The eruption had appeared 10 days after the initiation of the therapy. On examination, almost his entire trunk, extremities, and face were erythematous [Figures 1 and 2]. Confluent annular purplish-livid and dusky red targetoid patches and islands of normal-appearing skin were clearly observed on his trunk [Figure 3]. Nails, scalp, palmoplantar skin, and genital
and oral mucosa were normal. Biopsy specimen showed interface reaction pattern including vacuolar change in basal cells of epidermis, lymphocyte exocytosis, and perivascular lymphocytes with a few eosinophils [Figure 4]. Immunofluorescent study demonstrated weak C3 positivity in basal membrane level.

Based on the clinical, histological, and laboratory data, the eruption due to sorafenib was diagnosed. Initially, sorafenib was stopped and topical potent steroid (beclomethasone dipropionate cream) and systemic antihistamine (Cetirizine hydrochloride 20 mg/day, orally) were started. Four days after the cessation, eruption completely subsided without desquamation or postinflammatory pigmentation [Figure 5]. Two weeks after subsidence of the rash, sorafenib was restrated at 400 mg/daily. Recurrence of the lesions was not observed after 4-week follow-up period.

Cutaneous toxicities due to sorafenib are not infrequent. However, there was no relationship between skin toxicity and response to drug in phase III Treatment Approaches in Renal Cancer Global Evaluation Trial (TARGETs). To date, few reports of sorafenib-induced erythema multiforme (EM) have been published. The rare cases of EM or grade 3 rashes suggest hypersensitivity. Cessation of the treatment has been recommended to control these events. In addition, readministration of the drug is contraindicated in these allergic reactions including EM.

Numerous clinical conditions including EM and EM-like drug eruption can cause interface dermatitis with vacuolar alteration. The presence of an eosinophilic inflammatory infiltration favors a drug reaction though differentiating these two conditions can be challenging. Regarding DIF findings in our patient, some authors have described C3 positivity at the basal membrane zone in drug reactions as well as EM. EM presents with typical target lesions as well as macular, papular, or urticarial plaques mainly localized on the distal extremities. Atypical targetoid lesions may associate with a wide range of skin conditions including Stevens–Johnson syndrome/Toxic epidermal necrolysis (SJS/TEN), urticaria, and fixed drug eruption. However, our clinical findings were not consistent with any of these conditions. Thus, we considered our patient to represent an EM-like reaction.

This is the first report of a case presented with atypical targetoid lesions associated with sorafenib use. Also,
the cutaneous reaction did not reappeared after the reintroducing of the drug, sorafenib. As it was previously suggested in the literature, the reaction was easily controlled with dose interruption, topical treatments, and systemic antihistamines in our patient. As in our case, Pichard et al. reported targetoid lesions resembling EM without histopathological evidence and successful retreatment with sorafenib.\(^4\)

In conclusion, we would like to draw your attention to this skin reaction, which mimics serious cutaneous adverse drug reactions. Due to the fact that skin toxicity is associated with favorable prognosis in HCC patients, controlling cutaneous adverse events of sorafenib has extreme importance in management of these patients.

**Declaration of patient consent**

Written informed consent form from the patient was obtained.

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Nil.

**Conflicts of interest**

There are no conflicts of interest.

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