reactions to imatinib include erythematous maculopapular eruptions, periorbital edema, toxic epidermal necrolysis, Stevens-Johnson syndrome, acute generalized exanthematous pustulosis, purpuric vasculitis, and mycosis fungoides-like reactions.[6] The close temporal association between drug initiation and pyoderma gangrenosum onset, the severity of our patient's lesions, and the dramatic remission following drug suspension, makes our case worth reporting. We aim to make clinicians aware of this extremely rare adverse skin reaction in patients receiving tyrosine kinase inhibitors.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

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Induction of localized bullous pemphigoid on a young woman following a chemical peel

Sir,

Bullous pemphigoid is a senile acquired autoimmune bullous disorder and its localized variant is rare in young adults. Several known triggering factors exist such as drugs, trauma, surgery and radiation therapy.[1] Here, we report a young female who developed localized facial bullous pemphigoid following a glycolic acid chemical peel.

A 26-year-old female presented with itchy erythema and vesicles on her face since two months.The patient had history of acne vulgaris for seven years, which improved with treatment one year before her visit. To improve her skin-texture, she underwent a single session of glycolic acid chemical peeling (unknown concentration) in a local beauty salon 20 days preceding her clinical symptoms. She noticed gradual development of erythema and vesicles over her healed acne lesions. A local physician diagnosed it as impetigo and she applied mupirocin ointment for two weeks without any appreciable benefit. On cutaneous examination we observed scattered erythema and papulovesicles distributed over her cheeks, chin, root of the nose, temples and forehead [Figure 1a]. Tense, clear, fluid-filled vesicles sized 0.3–1.0 cm occupied the edge of the erythema [Figure1b]. Nikolsky's sign was negative. Ruptured vesicles resulted in superficial erosion and crusts. No similar lesions were observed on any other part of her body. The patient denied any exposure to photosensitive food, outdoor work, drugs, radiationor other chemicals. There was no history of photosensitivity. A family history was non-contributory. Routine biochemistry including complete metabolic panels and auto-antibody screens were within normal limits. An HIV test was non-reactive.

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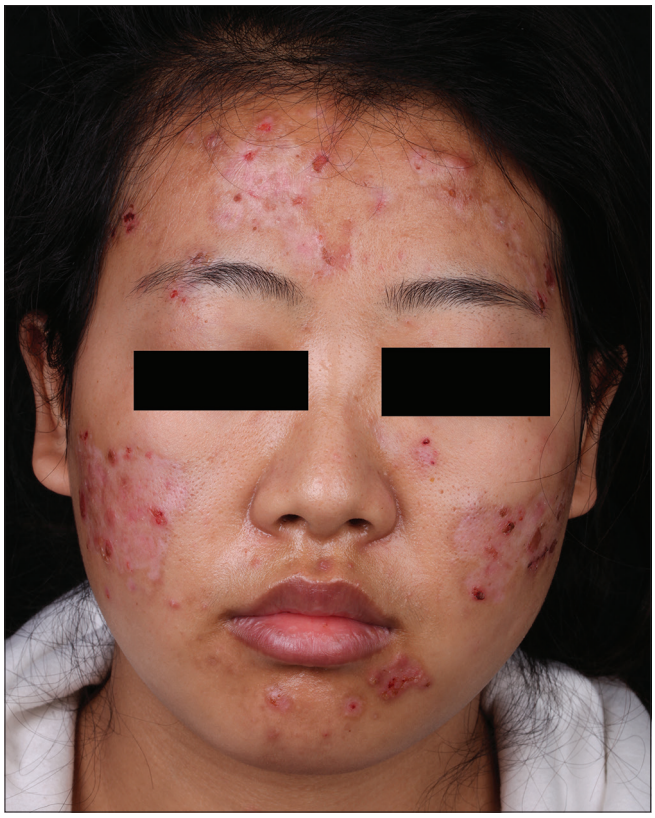

Figure 1a: Frontal view of erythema and papulovesicles present over the entire face

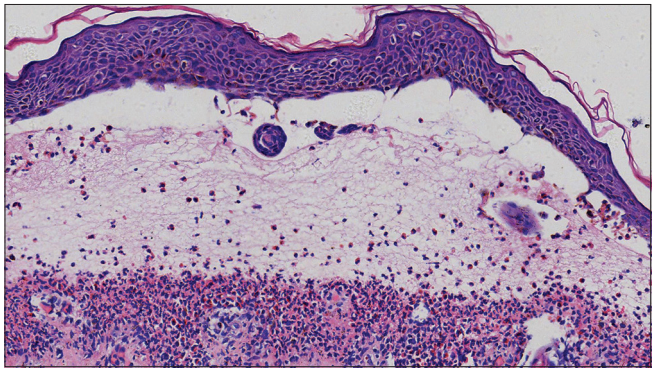

Figure 2a: A subepidermal blister with eosinophils and neutrophils infiltration (H & E) ×200

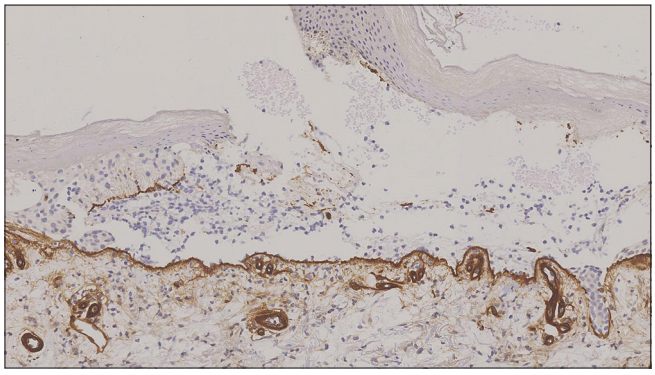

Figure 2c: Blister located at the epidermal side of the dermoepidermal junction, as shown by Type IV collagen by immunohistochemical staining (×100)

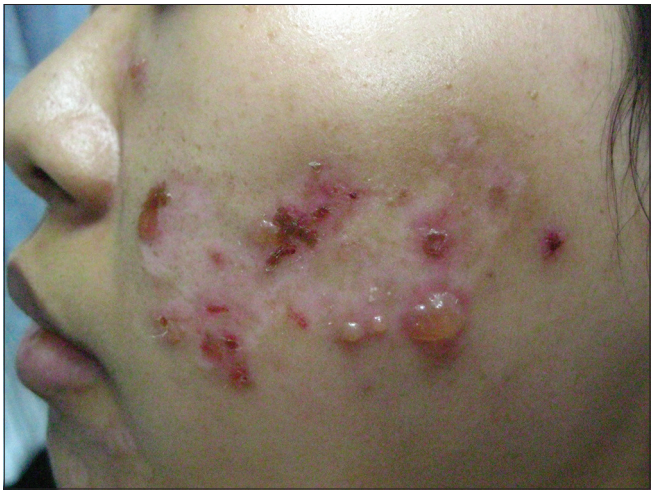

Figure 1b: Lateral view of tense, clear, fluid-filled vesicles with the size of 0.3–1.0 cm on the edge of the erythema

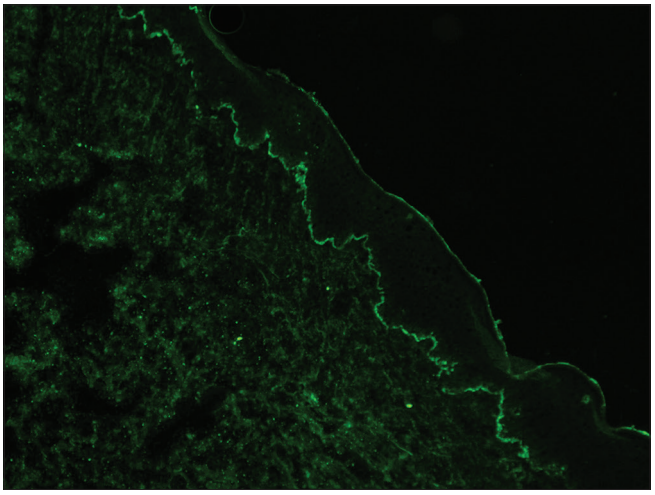

Figure 2b: Direct immunofluorescence revealed a strong linear deposition of IgG along the dermoepidermal junction (×100)

Lesional skin biopsy revealed a subepidermal bulla with intravesicular and perivascular eosinophilic and lymphocytic infiltration. [Figure 2a]. Direct immunofluorescence demonstrated linear IgG and C3 deposition along the dermo-epidermal junction [Figure 2b]. Collagen IV immunostaining confirmed its presence at the base of blister [Figure 2c]. Serum anti-BP180 and BP230 antibody titres were 6.93 U/ml and <2.00 U/ml, respectively (positive≥9). Detection of auto-antibodies against the 200 kDa protein of the dermal-epidermal junction by immunoblotting with dermal extract was negative. The histopathology and positive direct immunofluorescence suggested a diagnosis of subepidermal autoimmune bullous dermatosis. Based on the clinical presentation and laboratory test results including collagen IV immunostaining, a final diagnosis of localized bullous pemphigoid was made.

We prescribed oral methylprednisolone (24mg/day) and topical tacrolimus 0.1% ointment, which resulted in complete

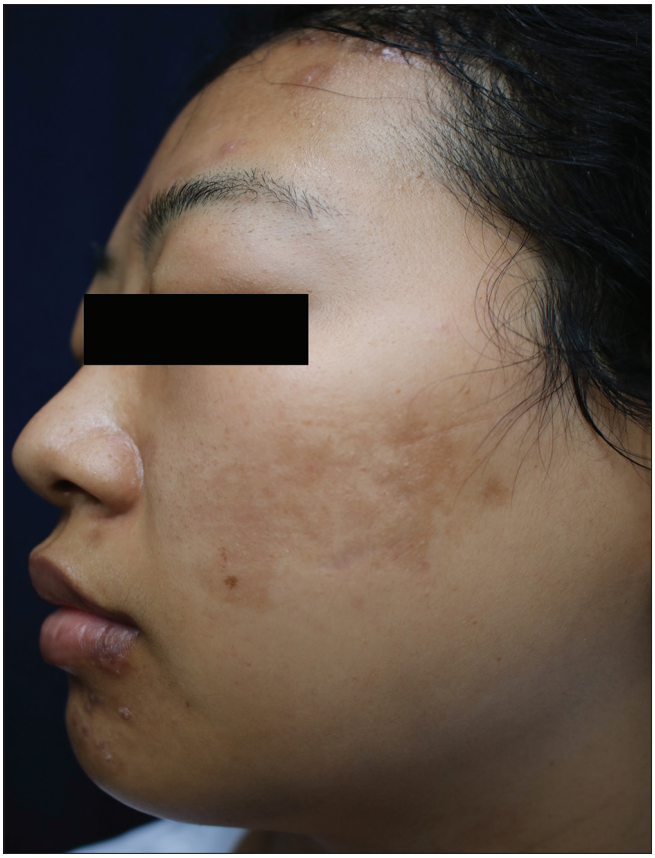

Figure 3: Mild hypopigmentation remained after a 1-year follow-up

resolution of symptoms within two weeks, without any new lesion. Subsequently, methylprednisolone was tapered to 16mg/day at four weeks and 8 mg/day at six weeks. Treatment was discontinued after complete remission of skin lesions after two months of treatment initiation. During the follow-up for one year, new rashes developed intermittently which subsided with a short course of topical tacrolimus ointment. [Figure3].

Bullous pemphigoid is an acquired autoimmune bullous dermatosis, predominantly affecting elderly population (65–75 years of age). Typical skin lesions include tense blisters or bullae on normal or erythematous skin with variable pruritus.[1] In most cases, lesions involve the whole body within weeks to months. However, in rare cases, the disease is characterized by localized lesions. To the best of our knowledge, only 20 cases of localized bullous pemphigoid have been reported in young adults.[2] Our young female patient presented with pruritic facial blisters for 20 days after a glycolic acid peel. We detected low serum concentration of both BP180 and BP230 specific antibodies by ELISA. Positive staining for dermo-epidermal IgG and C3 by direct immunofluorescence ruled out allergic contact dermatitis. Histopathology and positive direct immunofluorescence pattern supported a diagnosis of subepidermal autoimmune bullous dermatosis. However, normal ANA level and presence of collagen IV at the base of blister ruled out bullous lupus erythematosus and epidermolysis bullosa acquisita respectively. Absence of auto-antibodies against the 200 kDa protein of the dermal-epidermal junction by immunoblotting ruled out anti-p200 pemphigoid. Furthermore, young age, unmarried status and no history of preganancy and/or use of hormone-containing medications ruled out pemphigoid gestationis. Several cases of localized bullous pemphigoid have been reported with low titres of BP180 with various atypical associations.[1,3] The test results of our patient are consistent with localized type according to the diagnostic criteria for atypical bullous pemphigoid.[1] The previous studies have failed to perform detailed antibody titres in such patients. Thus, levels of antibodies against BP180 and BP230 in localized variant remain unclear, while other soluble ectodomains of BP180 may play some role.

Localized bullous pemphigoid (BP) may be triggered by multiple factors[1] such as trauma, radiation therapy, ultraviolet radiation, thermal or electrical burns, surgical procedures, transplants, hydrostatic forces[4] and topical medicines and even photodynamic therapy.[3] The exact pathogenesis remains unclear. To our best knowledge, this is the first report of localized BP induced by glycolic acid chemical peeling.

Chemical peeling or chemexfoliation aims to induce a controlled chemical injury to skin to destroy the epidermis (superficial peeling) and/or portion of dermis (medium or deep peeling). Subsequently it promotes skin regeneration and tissue remodeling. Glycolic acid is an alpha-hydroxy acid which reduces corneocyte cohesion and promotes desquamation and epidermolysis.[5] It is a superficial peeling agent and widely used as an effective and safe treatment option for active acne vulgaris and post-acne scarring.[6] The most common complications of glycolic acid peeling include swelling, pain, persistent erythema, pruritus, allergic reactions, folliculitis/acne, infection, herpes recurrence, hypopigmentation and hyperpigmentation, demarcation lines and scarring.[7] Here, we report an unique case of glycolic acid chemical peel induced localized bullous pemphigoid. We hypothesize that chemical peeling may have altered the basement membrane antigenicity by direct and uncontrolled epidermal damage, ultimately facilitating the development of this disease. Further research is required to uncover the exact patho-mechanism of this process.

Our patient experienced complete remission of skin lesions with two months of oral methylprednisolone therapy. Our case recommends the inclusion of glycolic acid peels to the list of possible exogenous triggers for localized bullous pemphigoid.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent.

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Conflicts of interest
There are no conflicts of interest.

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Is dermoscopy a useful tool in pseudo-Kaposi sarcoma?

Sir,

Pseudo-Kaposi sarcoma, also known as acroangiodermatitis, is a disorder characterized by the presence of a reactive proliferation of blood vessels that usually takes place on the patients' limbs in the setting of different vascular disruptions. These include chronic venous insufficiency in acroangiodermatitis of Mali and congenital arteriovenous malformations in Stewart-Bluefarb syndrome.[1,2]

Clinically, patients present with violaceous papules that can coalesce into plaques, mimicking Kaposi sarcoma [Figure 1a]. On histological examination, pseudo-Kaposi is characterized by lobular vascular proliferation with associated perivascular fibrosis. Erythrocyte extravasation and dermal siderophages are frequently encountered [Figure 1b].

In this report, we describe the dermoscopic features of four cases of pseudo-Kaposi. We found only three previous dermoscopic descriptions in the literature. All patients were treatment naïve when they were first seen in our clinic, except for patient two, who had been using a furoate mometasone ointment for the past month with partial relief of his symptoms. In every case, the diagnosis was suspected clinically and confirmed with histopathological and immunohistochemical examination: CD34 was expressed in endothelial cells but not in the surrounding perivascular cells, whereas immunostaining was negative for human herpesvirus 8 (HHV-8) in all cases. Clinical signs of chronic venous insufficiency were obvious in patients one, two and four, whereas patient three had suffered a previous deep venous thrombosis (diagnosed by Doppler ultrasound) three weeks before the lesions started to develop.

In preceding reports, irregular vessels and white structureless areas were described in two cases,[2] while white rail lines, red and blue lacunae and hemorrhagic crusts were found on the other one.[3] We believe that, overall, different terms were used to refer to similar structures.

The clinical and dermoscopic features of our patients are outlined in Table 1. In all cases, a clearly vascular pattern was appreciated. All of them presented a mauve to violaceous background color with polymorphic vessels which reflect the vascular proliferation (the red lacunae probably embodying the lobular proliferation) [Figure 2]. Polychromatic color change (or rainbow pattern), like the one described in Kaposi sarcoma, was demonstrated in three patients. While this was initially described as a specific sign for Kaposi sarcoma, it has now also been described in many other lesions, such as angiokeratomas, hypertrophic scars, melanoma and dermatofibroma.[4] The exact mechanism by which this phenomenon occurs is still not well established. However, it is probably related to the different refraction indices of polarized light which exist throughout a disorganized dermis with increased vascularity.[4]

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