with leukocytoclasia, interface dermatitis and mucin deposition. Urticarial lesions of dermatitis herpetiformis or linear immunoglobulin A dermatosis usually show dermal papillary neutrophilic microabscesses.

A few cases of pregnancy-associated neutrophilic dermatoses have been documented in the literature. An alteration in the immune system during gestation might be a common factor. Pregnant women show a progressive neutrophilia due to increased levels of pro-inflammatory factors (e.g., granulocyte colony-stimulating factor and T helper-17), which may lead to neutrophil hyper-reactivity.[5]

We present a peculiar case of pregnancy-associated neutrophilic figurate erythema with a unique clinical feature of tender arciform erythematous plaques with pustules. While dealing with similar cases, extensive laboratory studies and skin biopsy are required to exclude other serious autoimmune and infectious disorders or associated malignancies. The treatment regimen for neutrophilic dermatoses may be followed to control the symptoms. Since this disease tends to run a benign course, clinicians should weigh benefits and risks before treating pregnant patients.

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.

Yi-Wen Kuo, Jau-Yu Liau[1]

Department of Dermatology, E-Da Cancer Hospital, Kaohsiung,Taiwan, [1]Department of Pathology, National Taiwan University Hospital and National Taiwan University College of Medicine, and Graduate Institute of Pathology, National Taiwan University College of Medicine, Taipei, Taiwan

Corresponding author:
Dr. Jau-Yu Liau,
Department of Pathology, National Taiwan University Hospital and National Taiwan University College of Medicine, and Graduate Institute of Pathology, National Taiwan University College of Medicine, Taipei, Taiwan.
019188@ntuh.gov.tw

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Coexistence of psoriasis and linear IgA disease: An uncommon presentation

Sir,

Psoriasis is a common immune-mediated, autoinflammatory disorder occurring in genetically predisposed individuals. It is seen in 0.4–2.8% of Indian population. Linear IgA disease is a relatively rare dermatoses with an estimated incidence of 0.2–2.3 cases per million per year.[1] It is characterized by the formation of tense vesicles or bullae, often in an annular pattern with blistering along the edge of the lesion – the so-called 'string of pearl' appearance. It is observed in two peaks of different age groups. The first peak usually occurs in six months to six years of age and is known as chronic bullous disease of childhood, while the second peak occurs at around 60 years of age. The coexistence of autoimmune bullous diseases with psoriasis is not uncommon. Most of the reported cases show association with bullous pemphigoid; however, the coexistence of psoriasis with pemphigus vulgaris, pemphigus foliaceous, linear IgA disease, cicatricial pemphigoid, epidermolysis bullosa acquisita and anti p200 pemphigoid is also known.[2] The development of psoriasis in a patient of linear IgA disease is rare in literature and presents a unique therapeutic challenge.

How to cite this article: Neema S, Bhatt S, Kashif AW, Radhakrishnan S. Co-existence of psoriasis and linear IgA disease: An uncommon presentation. Indian J Dermatol Venereol Leprol 2022;88:101-3.

Received: June, 2020 **Accepted:** June, 2021 **EPub Ahead of Print:** September, 2021 **Published:** December 2021

DOI: 10.25259/IJDVL_906_20 **PMID:** 34623057

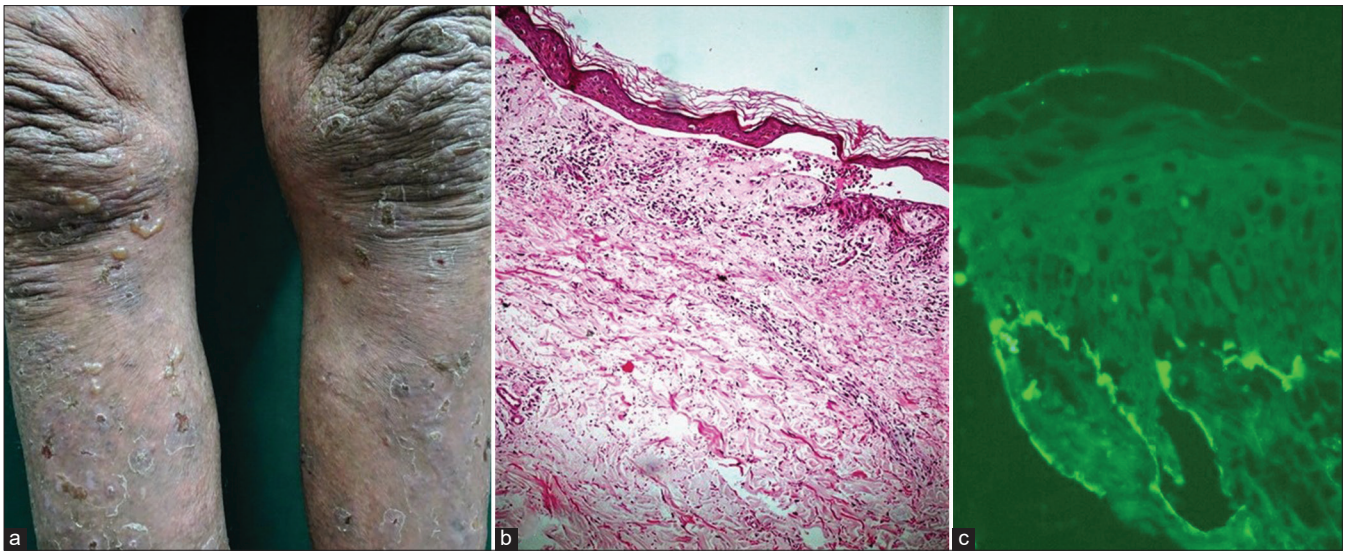

Figure 1: (a) Tense vesicles and bullae can be seen on both lower legs. Few vesicles have annular distribution (medial aspect of right knee). (b) Histopathology of LAD lesion shows sub-epidermal blister and neutrophils, eosinophils and fibrin in blister cavity (H & E, 40×) (c) Direct immunofluorescence shows IgA deposition on basement membrane (DIF, 40×)

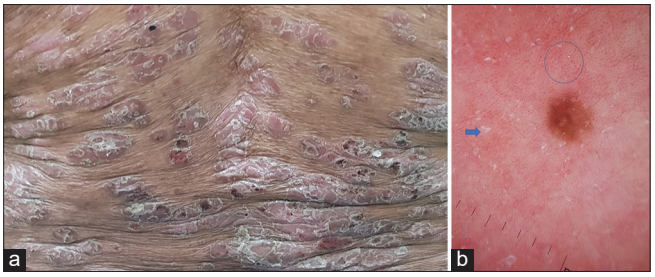

Figure 2: (a) Multiple well defined, erythematous scaly plaques over trunk (b) Dermoscopy of psoriatic plaque shows presence of regular dotted vessels (blue circle) and white scale (Dermlite DL4, polarized, ×10)

A 65-year-old man presented with multiple tense vesicles and bullae associated with severe itching. A few vesicles on the trunk had an annular pattern [Figure 1a]. No mucosal involvement was noted. No signs of any other skin disease were noted. Skin biopsy from the lesions revealed subepidermal, non-acantholytic blisters containing neutrophils, eosinophils and fibrin [Figure 1b]. Direct immunofluorescence showed linear deposition of IgA at dermo-epidermal junction [Figure 1c]. Based on clinical and histopathological findings, a diagnosis of linear IgA disease was made and patient was managed with intravenous methylprednisolone pulse 1 g daily for three days and tablet dapsone 100 mg once a day to which he responded well within one month. After two months, he presented with 40% body surface area involvement with multiple erythematous scaly plaques that showed positive grattage test as well as Auspitz sign and psoriasis assessment and severity index score of 28.6 [Figure 2a]. Dermoscopy [Figure 2b] and histopathology were consistent with psoriasis. In next one week, he developed tense vesicles on areas affected with psoriasis [Figure 3a]. Dermoscopy showed features of psoriasis in the form of regular dotted vessels and well circumscribed yellow lacunae suggestive of bullae [Figure 3b]. Histopathology

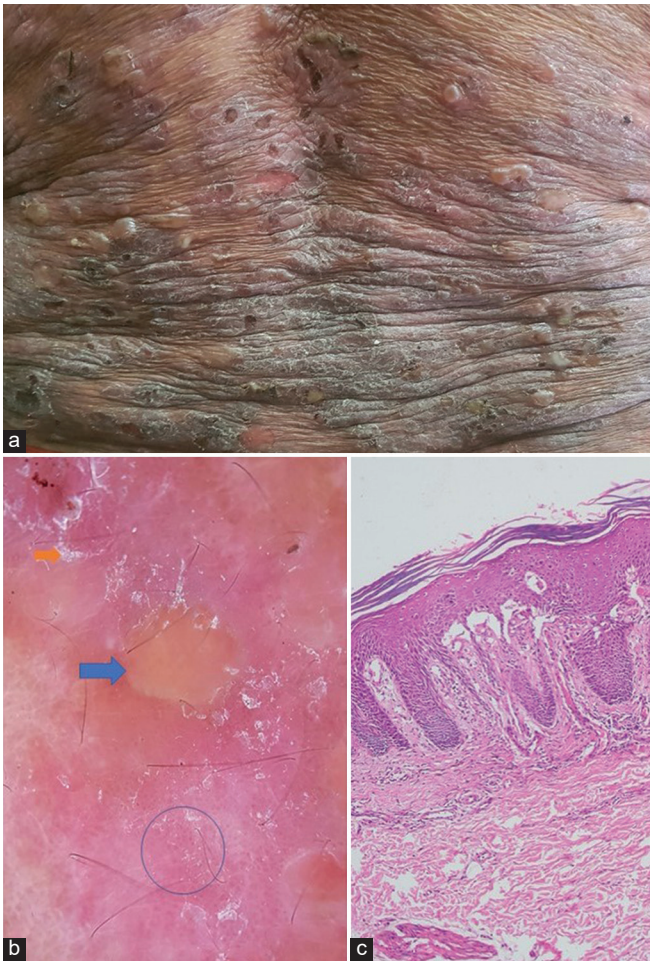

Figure 3: (a) Tense vesiculo-bullous eruptions over psoriatic plaque (b) Dermscopy of co-existence of psoriasis and LAD shows presence of regular dotted vessels (blue circle), white scales (orange arrow) and yellow lacunae (blue arrow) (Dermlite DL4, polarized, ×10) (c)Histopathology of co-existent lesion shows parakeratosis, regular acanthosis and subepidermal blister (H& E, 100×)

showed parakeratosis, regular elongation of rete ridges and sub-epidermal blister [Figure 3c]. His condition was diagnosed as linear IgA disease with chronic plaque psoriasis. He was treated with methotrexate 15 mg per week and dapsone 100 mg once a day was continued. There was significant improvement in psoriasis and linear IgA disease at four weeks.

The coexistence of autoimmune bullous diseases and psoriasis vulgaris was first described by Bloom in the year 1929. In the largest case series of psoriasis and autoimmune bullous diseases coexistence of 145 cases, linear IgA disease – psoriasis coexistence contributed to 2.1% of all cases. The most common autoimmune bullous diseases seen with psoriasis were bullous pemphigoid seen in 78 (53.8%) cases and anti-laminin γ1 pemphigoid in 40 (27.6%) cases.[3] Patient with psoriasis developing autoimmune bullous diseases is commoner as compared to autoimmune bullous diseases developing psoriasis. The underlying pathomechanism maybe due to the epitope spreading. Chronic injury to the basement membrane in psoriasis along with the trafficking of lymphocytes and antigen-presenting cells, exposes basement membrane components to autoreactive lymphocytes that may predispose to autoimmune disease. The similar mechanism has been implicated in coexistence of lichen planus with bullous pemphigoid, Stevens-Johnson syndrome and secondary onset cicatricial pemphigoid.[4] The sera of some of the psoriasis patients contains circulating IgA immune complex that may be responsible for the development of linear IgA disease.[5] In our patient, psoriasis developed after linear IgA disease. The possible mechanism may be similar to koebnerization. The damage to the keratinocytes due to the disease process and intense itching can lead to the release of cathelicidin (LL-37), which acts as autoantigen, binds to the toll-like receptor 7 (TLR 7) in plasmacytoid dendritic cells. Type 1 IFN released by stimulated plasmacytoid dendritic cells promotes conversion of naive T cells to Th1 and Th17 cells; thereby causing psoriasis.

Choosing treatment in these patients is challenging. Steroid withdrawal is a known precipitating factor for erythrodermic and pustular psoriasis. We managed our patient with combination of methotrexate and dapsone with significant improvement in symptoms after four weeks. Methotrexate inhibits dihydrofolate reductase and dapsone inhibits dihydropteroate synthetase, components of folate pathway and their concomitant use increases the risk of hematologic toxicity. Other alternative drugs which can be used for the management of psoriasis in this scenario can be apremilast, cyclosporine, tumour necrosis factor (TNF)-alpha inhibitor or IL-17 blockers. However, affordability was a concern and we continued this combination with close monitoring of patient with successful outcome. Once our patient is in remission for six months, we plan to stop dapsone and continue methotrexate for another three to six months before tapering.

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.

Shekhar Neema, Siddharth Bhatt, A. W. Kashif[1], S. Radhakrishnan

Departments of Dermatology and [1]Pathology, Armed Forces Medical College, Pune, Maharashtra, India

Corresponding author:
Dr Shekhar Neema,
Department of Dermatology, Armed Forces Medical College, Pune, Maharashtra, India.
shekharadvait@gmail.com

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