Colocalisation of subacute cutaneous lupus erythematosus and vitiligo in a woman with thyroid autoantibodies: An intriguing association

Sir,

The term 'multiple autoimmune syndrome' denotes the occurrence of three or more autoimmune diseases in the same individual. Vitiligo and lupus erythematosus are included in type 3 multiple autoimmune syndrome which also features autoimmune thyroid disease, myasthenia gravis, thymoma, Sjögren's syndrome, pernicious anaemia, idiopathic thrombocytopenic purpura, Addison's disease, type 1 diabetes mellitus, autoimmune haemolytic anaemia and dermatitis herpetiformis.[1] Co-existence of vitiligo with other autoimmune diseases is well recognised. We report an unusual case of psoriasiform subacute cutaneous lupus erythematosus, colocalising over pre-existing vitiligo patches in a woman with thyroid autoantibodies. We were unable to find any previous reports of a similar occurrence.

A 40-year-old woman with vitiligo vulgaris for the past 16 years presented with abrupt onset of itchy, red, scaly lesions over most of the vitiligo patches involving the extensor forearms, dorsae of hands, back [Figures 1a and 1b] and lower legs, for the past one week, after intense sun exposure. Scalp, lips and tip of the hands were spared. Photosensitivity without fever, joint pains, systemic symptoms or drug intake was noted. The patient was not on any topical or systemic treatment for vitiligo, for the past ten years. Family history of similar or other autoimmune diseases was conspicuously absent. Cutaneous examination showed bilaterally symmetrical, well-defined, erythematous, non-scarring, scaly (psoriasiform) plaques co-localised over vitiligo patches, as mentioned above. Lesions on forearm and legs showed leucotrichia. Hair and nails were normal and no abnormality was detected in systemic examination. Differential diagnoses considered for the scaly plaques were psoriasis, psoriasiform subacute cutaneous lupus erythematosus and psoriasiform eczema. Routine haematological, biochemical, urine examinations, hepatitis B surface antigen, human immunodeficiency virus serology and venereal disease research laboratory test were normal or negative. Serum antinuclear antibody test was strongly positive (titre >1:160) with anti-Sjögren's syndrome-related antigen A (native 60 kilodalton, kD), and anti-Ro52 (52 kD) and anti-Sjögren's syndrome-related antigen B antibodies positive. The anti-double-stranded deoxyribonucleic acid and anti-Smith antibodies were negative. Electrocardiogram and echocardiography were normal. Punch biopsy from the representative site showed focal epidermal thinning, flattening of rete ridges, moderately dense superficial perivascular and periappendageal lichenoid lymphocytic infiltrate with focal infiltration at the dermoepidermal junction, basal cell vacuolar degeneration, scattered colloid bodies and melanophages in the papillary dermis. Reticular dermis showed abundant mucin [Figure 2]. Special stains showed reduced number of melanocytes in the basal layer. Alcian blue staining for mucin was positive. A diagnosis of psoriasiform type of subacute cutaneous lupus erythematosus with vitiligo was made, based on the clinical, and histopathological findings and presence of strongly positive antinuclear antibody, anti-Sjögren syndrome-related antigen A and B antibodies.

Her thyroid profile was normal except for elevated anti-thyroglobulin antibodies. Multiple autoimmune syndrome type 3 was diagnosed. Further endocrinological workup could not be done for the want of facilities. The patient was started on tablet hydroxychloroquine 300 mg/day, oral prednisolone (40 mg/day, tapered over 12 weeks), topical corticosteroid and advised strict photoprotection with good response, healing without atrophic scarring [Figures 3a and 3b].

Colocalisation of vitiligo with psoriasis, lichen planus, sarcoidosis, alopecia areata and discoid lupus erythematosus has been reported earlier, suggesting similar autoimmune mechanisms but has not been fully explained yet.[2-4] We found one report of subacute cutaneous lupus erythematosus co-existing with vitiligo.[5] Recently, vitiligo-like depigmentation consequent to subacute cutaneous lupus erythematosus and hydroxychloroquine treatment has been reported.[6] However, in our case, psoriasiform subacute

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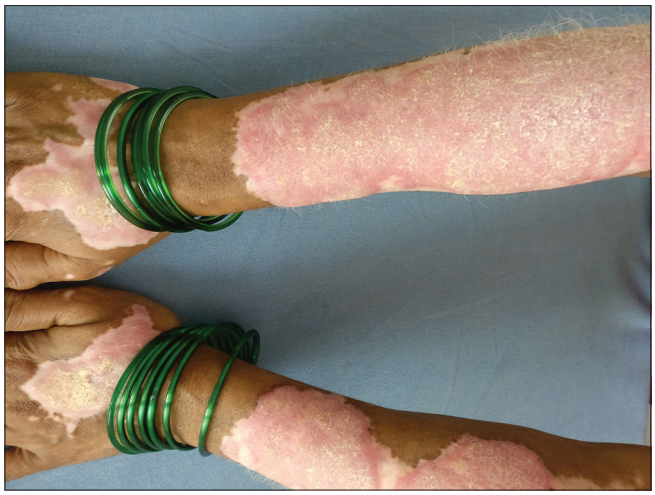

Figure 1a: Psoriasiform plaques colocalising over vitiligo involving forearms

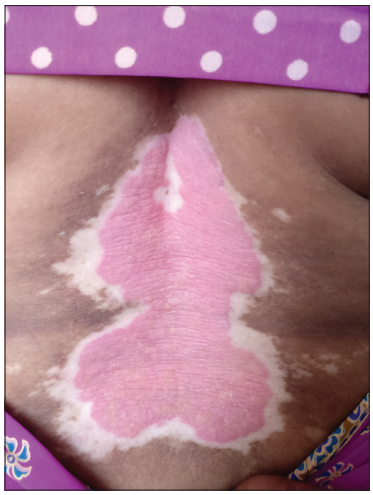

Figure 1b: Psoriasiform plaque over trunk

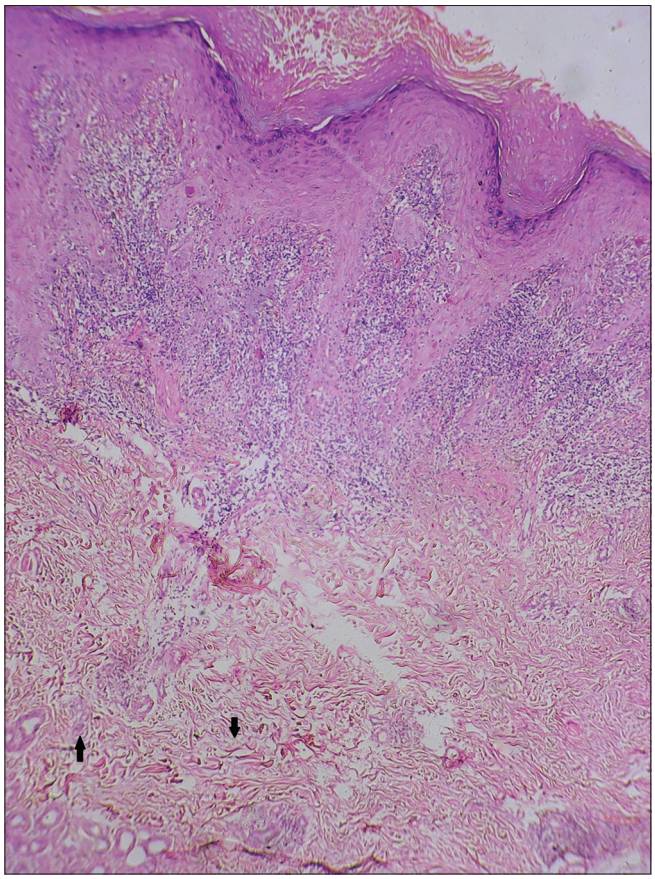

Figure 2: Epidermal changes, moderately dense superficial perivascular, periappendageal lichenoid lymphocytic infiltrate, basal cell vacuolar degeneration, colloid bodies and abundant mucin in reticular dermis (arrows) (haematoxylin and eosin, ×40)

cutaneous lupus erythematosus was co-localised over vitiligo. Points favouring co-localisation and not a sequel in our case are the presence of depigmented patches (vitiligo) before the onset of subacute cutaneous lupus erythematosus, leucotrichia, sparing of a few vitiligo patches, treatment with hydroxychloroquine after the onset of subacute cutaneous lupus erythematosus and presence of thyroid autoantibodies which points toward the tendency to develop autoimmune diseases. It is important to differentiate psoriasis from other psoriasiform disorders as the course, prognosis and treatment varies.

The 70 kD heat shock protein assists in the assembly and folding of a broad range of proteins. During cellular stress, it is upregulated to maintain normal cellular homeostasis. However, 70 kD heat shock protein can be a danger signal that activates innate immunity and contributes to autoimmunity. It is found that in both vitiligo and lupus skin, plasmacytoid dendritic cells are located in proximity to 70 kD heat shock protein expressing keratinocytes, suggesting its role to impact the activity of plasmacytoid dendritic cells in these disorders.[7] There may also be a common genetic locus for susceptibility to lupus and vitiligo. The genomic region at 17p13, which contains the gene, *SLEV1* is suggested to be associated with vitiligo-related lupus. It is suggested that the gene, leading to developing lupus primarily, may modify the risk for vitiligo among ascertained families.[8] Another possible explanation for colocalisation could be based on the concept of Ruocco's immunocompromised cutaneous district, where sustained skin damage due to ultraviolet radiation on the exposed vitiligo skin may lead to immune dysregulation and subsequent development of other cutaneous disorders, as in our case.[9]

Generalised vitiligo is also a component of the autoimmune polyendocrine syndrome Type 1 and Schmidt multiple autoimmune syndrome (autoimmune polyendocrine syndrome Type 2) which includes autoimmune thyroid disease, pernicious anaemia, Addison's disease and type-I diabetes mellitus (Carpenter syndrome).[10] Evaluation for associated autoimmune and endocrine disorders should be done, whenever possible. The diagnosis of autoimmune thyroid disease is usually made based on the presence of anti-thyroglobulin and anti-thyroid peroxidise antibodies (found in 90–95% and 20–50% of patients, respectively), a palpable

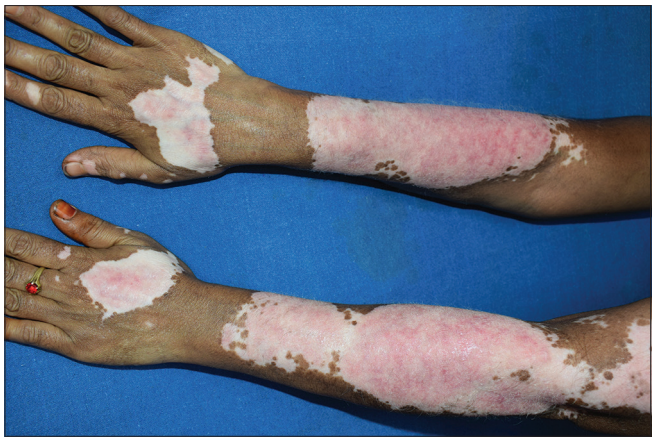

Figure 3a: Decrease in erythema and scaling over the forearms after treatment

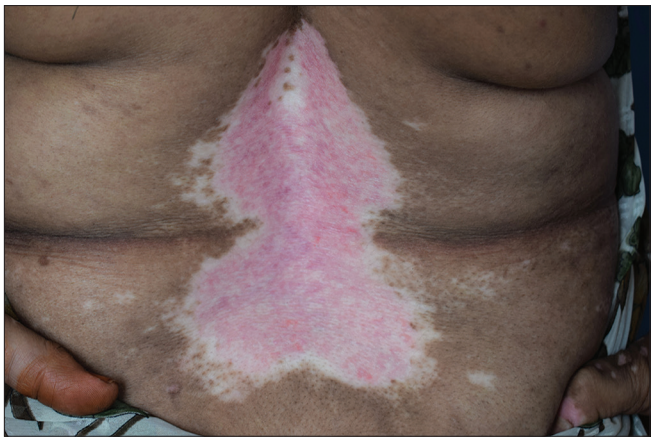

Figure 3b: Decrease in erythema and scaling over the trunk after treatment

goitre, ultrasound findings and histopathology. However, it can also be diagnosed based on the sustained presence of thyroid auto-antibodies, even in the absence of other findings.[11]

The treatment of vitiligo in the presence of lupus may be modified and involves mainly topical and systemic corticosteroids or topical calcineurin inhibitors. Phototherapy should be avoided. Treatment of subacute cutaneous lupus erythematosus includes photoprotection, topical corticosteroids and antimalarials.

Multiple autoimmune syndrome and autoimmune polyglandular syndrome should be considered by the dermatologist, while evaluating a case of generalised vitiligo associated with any other autoimmune skin disease like lupus or alopecia areata. Endocrinological evaluation and regular follow-up can help in early diagnosis and the dermatologist may play a key role in improving the prognosis.

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Declaration of patient consent

The patient's consent is not required as the patient's identity is not disclosed or compromised.

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Conflicts of interest

There are no conflicts of interest.

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