Correspondences

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Lichen Planus in Silicosis Patient with Unusually High Antinuclear Antibody Titer

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Sir,
A 35-year-male nonsmoker, stone cutter by occupation presented with chief complaints of dry cough and breathlessness on exertion for 10 years and multiple, raised itchy and hyperpigmented lesions on the face, back, chest, hands and legs for 1 year. There was a history of photosensitivity and oral ulcers for 2 months. There was no history of fever, joint pains, chest pain,
abdominal pain, seizures or significant weight loss. He was diagnosed with pulmonary tuberculosis 4 months back, and anti-tubercular treatment was started as advised by a local physician. There was a history of the increase in the size of cutaneous lesions after the start of antituberculous treatment [ATT]. There was no history of similar illness or tuberculosis in the family. On systemic examination, fine crepts were present bilaterally in lung fields. On cutaneous examination, multiple well defined hyperpigmented scaly papules and plaques were present in bilateral symmetric fashion on scalp, forehead, malar areas, nose, ears, retroauricular area, back, dorsum of hands, and extensor aspect of limbs [Figures 1 and 2]. Limb flexors, palms, and soles were spared. Violaceous plaques with whitish striae were present on both lips. Violaceous patches with whitish reticular striae were present on buccal mucosa along with multiple erosions on hard palate and retromolar areas bilaterally [Figure 3].

Routine investigations including a complete hemogram, liver and renal function tests, random blood sugar were normal. Chest X-ray showed miliary opacities in bilateral lung fields [Figure 4]. Mantoux test and ELISA for HIV-1/HIV-2 were negative. On pulmonary function tests, FEV1/predicted was 53% suggestive of moderate obstruction. Antinuclear antibody (ANA) titers were unusually high (1:3000) but anti-ds DNA and direct-immunofluorescence for IgG were negative. Occupational history, clinical findings, Mantoux test, pulmonary function tests and chest X-ray pointed toward a diagnosis of pulmonary silicosis. HRCT Thorax could not be done due to financial constraints. Biopsy from face lesion showed wedge-shaped hypergranulosis, irregular epidermal hyperplasia, perivascular and periappendageal infiltrates obscuring the dermo-epidermal junction and occasional colloid bodies [Figures 5 and 6]. Based on above clinicopathological findings diagnosis of lichen planus with silicosis was made. Differential diagnosis of Lichenoid eruption was excluded based on history (onset of lesions before starting ATT) and histopathology (absence of parakeratosis, focal agranulosis, deep perivascular infiltrates of eosinophils and plasma cells) and subacute lupus erythematosus (LE) was excluded based on clinical morphology of lesions and histopathology (absent epidermal atrophy, dermal oedema and mucin). Patient was advised oral and topical steroids and follow up after 3 weeks. Lichen planus presented in unusual distribution pattern on malar areas on face, scalp and in and around ears along with palatal ulcers, photosensitivity and unusually high ANA titres characteristic of Lupus erythematosus. In true LP/lichen planus (LE) overlap there is presence of LP and LE in same lesion whereas the presence of LE features in one lesion and LP features in other one should be considered as coexistence of LE and LP but in our patient there was no clinical or histopathological evidence of LE in cutaneous lesions. [1]
Various case reports have suggested a link between silicosis and various autoimmune disorders like systemic lupus erythematosus, systemic sclerosis, rheumatoid arthritis, vasculitis, Sjogren syndrome, pemphigus vulgaris and bullous pemphigoid.[2-4] It has been suggested that there is dysregulation of immunity and Fas mediated apoptosis in lymphocytes derived from silicosis patient. There is a particular fraction of CD4+ T lymphocytes in silicosis patients that expresses weak levels of membrane Fas, secretes higher levels of soluble Fas, DcR3, and spliced variants, and is resistant to anti-Fas autoantibody-induced apoptosis. These cells may include self-recognizing clones which survive long as they are resistant to apoptosis and contribute towards autoimmunity. This is supported by the presence of high anti-nuclear antibody titres in persons with weak mean fluorescence intensity for membrane Fas.[5] Jones and Doll have reported high ANA titre in silicosis patients.[6,7]

To the best of our knowledge there are no published reports of lichen planus in patient of silicosis. We are reporting for the first time lichen planus in a patient of silicosis with high ANA titres suggesting dysregulation of immunity in latter. It also strengthens the autoimmune hypothesis of lichen planus and may provide further insight into pathogenesis of lichen planus.

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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