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Case Report

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POST HERPETIC SCAR SARCOIDOSIS WITH SYSTEMIC INVOLVEMENT: A RARE PRESENTATION

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ABSTRACT
Cutaneous sarcoidosis is considered one of the 'Great imitators' in dermatology owing to its varied clinical presentations. An uncommon manifestation is infiltration of old scars with non- caseating granulomas known as scar sarcoidosis. We report a case of a 38 years old female patient with cutaneous sarcoidosis overlying scars of healed herpes zoster with systemic involvement.

KEYWORDS: Sarcoidosis, post herpetic scar, pulmonary involvement.

INTRODUCTION
Scar sarcoidosis refers to the infiltration of old scar with non-caseating epitheloid cell granuloma. It is a rare but specific form of cutaneous sarcoidosis.[1] Scar sarcoidosis may appear before or after the systemic manifestations.[2] Thus, it must be looked for whenever a diagnosis of sarcoidosis is considered as systemic features may not be observed in early stages.

CASE REPORT
A 38 years old female presented with multiple, painful red colored lesions over right thigh since 7 months. She had history of eruption of painful grouped vesicles in the same region nine months back which resolved leaving behind light coloured scars. After 2 months of resolution of disease she noticed red colored slightly raised skin lesions, gradually increasing in size as well as number at the site of scar. She also gave history of dry cough and gradually progressing breathlessness on performing routine activities for last 4 months. There was no history of hemoptysis, chest pain, fever or significant weight loss. There was history of pain and redness in bilateral eyes for one month. There was no history of tuberculosis in past or in the family. General physical and systemic examinations were unremarkable. Ophthalmological examination revealed features suggestive of anterior uveitis. On dermatological examination multiple grouped erythematous papules were present overlying hypopigmented atrophic scars present on anterior aspect of right thigh, knee and upper part of leg in zosteriform pattern (L2, L3 and L4 dermatome) (Figure 1).

Routine laboratory investigations including complete blood count, hepatic and renal function tests, serum electrolytes, erythrocyte sedimentation rates, C reactive protein and serum and 24 hr urine calcium were within normal range. Electrocardiogarm (ECG) was also normal. The level of serum angiotensin-converting enzyme was raised. Mantoux test was found to be negative after 48 h. Chest radiography demonstrated bilateral hilar lymphadenopathy with increased bronchovascular markings (Figure 2). On HRCT chest multiple bilateral mediastinal lymphadenopathies with ground glass appearance and confluent nodular opacities in middle and upper lung zones were seen. Pulmonary function test revealed decreased diffusion capacity for carbon monoxide, which signifies a restrictive pattern of the pulmonary disease.

On histopathology, multiple non caseating epitheloid granulomas with Langhans' giant cells, focal fibrinoid necrosis with a sparse lymphocytic component in dermis were seen (Figure 3). Special stains for fungus and AFB bacilli were negative. On the basis of above clinical and histopathological findings, a diagnosis of post herpes zoster scar sarcoidosis with systemic involvement was made. Patient was started on oral prednisolone 30 mg per day with topical mometasone cream. For chest complaint she was referred to pulmonary department.

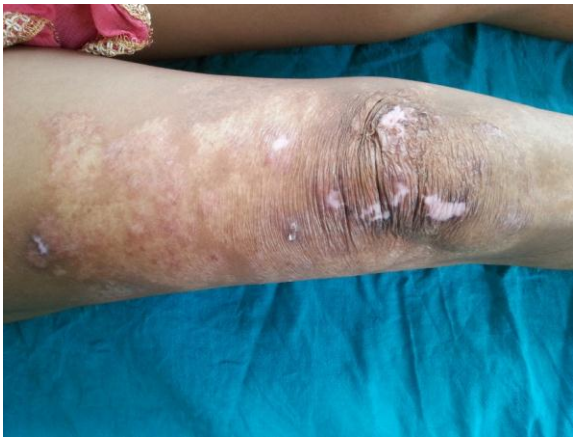

Figure 1: Multiple erythematous papules and plaques over the hypopigmented scar in a zosteriform pattern.

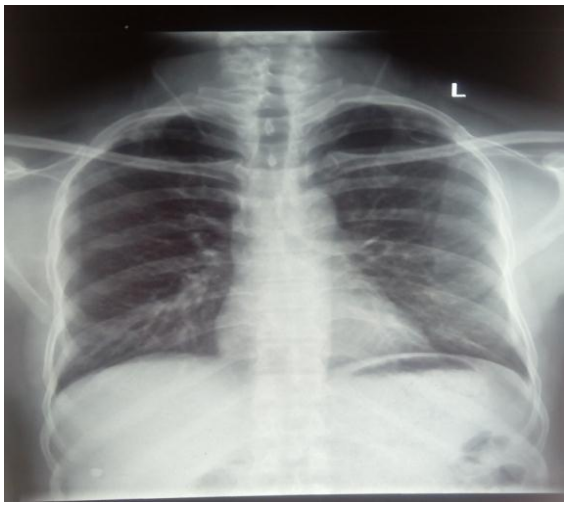

Figure 2: Chest radiography showing bilateral hilar lymphadenopathy with increased bronchovascular markings.

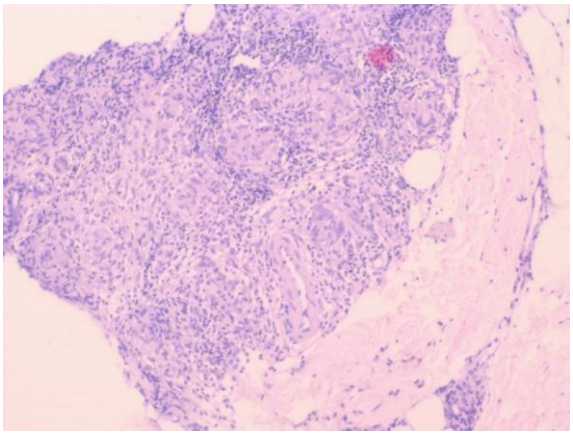

Figure 3: 4X magnification showing multiple non caseating epitheloid granulomas with Langhans' giant cells, focal fibrinoid necrosis with a sparse lymphocytic component in dermis.

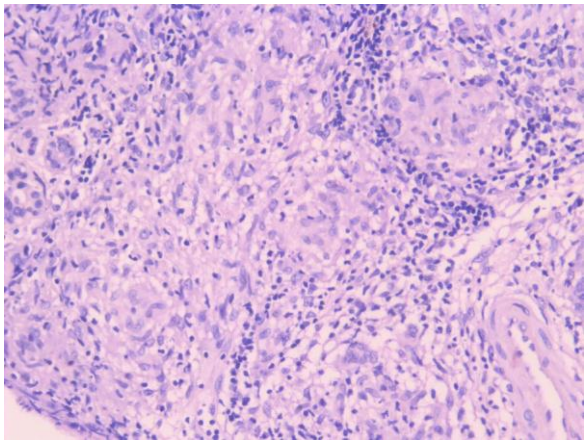

Figure 4: 10 X magnification showing multiple non caseating epitheloid granulomas with Langhans' giant cells, focal fibrinoid necrosis with a sparse lymphocytic component in dermis.

DISCUSSION

Sarcoidosis is a multisystemic disease with an unknown etiology. Sarcoidosis mostly affects the lungs, lymph nodes, liver, spleen, phalangeal bones, parotid glands, eyes, and skin[1] Cutaneous involvement occurs in 20–25% of cases.[3] Skin lesions in sarcoidosis may appear as maculae, papules, plaques, nodules, ulcers, localized alopecia, ichthyotic areas, subcutaneous nodules, lupus pernio, scar sarcoidosis, psoriasiform and even pustules.[4] Among these, scar sarcoidosis is a rare and specific form of sarcoidosis accounting for 5.4–13.8% of sarcoidosis cases.[5] Yanardag *et al.* reported scar sarcoidosis in 2.9% of diagnosed sarcoidosis cases.[6]

Scar sarcoidosis was first described by Caesar Boeck in 1899.[7] Scar sarcoidosis is characterized by the onset of erythematous swelling and the development of papules and nodules within the original scars.[2] In addition to reactivation of scars obtained from previous wounds, it has been reported at the sites of previous intramuscular injections, blood donation puncture sites, tattoo scars, scars of herpes zoster, on ritual scarification, at the sites of allergen extracts for desensitization and following hyaluronic acid injection and laser surgery.[2]

Although the pathogenesis of scar sarcoidosis is not known, it has been thought that the disorder may be due to previous contamination of the old scars with foreign bodies at the time of trauma. The macrophages on phagocytosed foreign bodies may cause releases of angiotensin-converting enzymes and lymphokines, which lead to the development of granulomas.[1] It has also been postulated that there is hypersensitivity induced granulomatous tissue reaction encountered in the healing phase of herpes zoster lesions and it depends on the patient's immune status and presence of viral antigens or tissue antigens modified by the varicella-zoster virus.[2] In addition, gamma-interferon induced by viral infection favors T-helper 1 shift, which may play a role in granuloma formation.[8]

Scar sarcoidosis is often preceded or accompanied by systemic involvement.[2] There are only few published cases of pulmonary involvement in post herpetic scar sarcoidosis.[2,9,10] A previous study reported that systemic involvement occurred in 30% of patients that had isolated cutaneous lesions after a period of 1 month to 1 year. Cecchi R *et al.* have reported case of post herpetic scar sarcoidosis without systemic involvement and considered it as akin to wolff's isotopic response.[11]

Differential diagnosis of scar sarcoidosis includes infectious skin diseases such as mycobacterium infections, Crohn's disease, foreign body granuloma, and hypertrophic scar or keloid.[1] Topical and intralesional steroid therapy may be sometimes effective for purely cutaneous sarcoidosis. For disfiguring lesions unresponsive to initial topical therapy or in the case of systemic involvement, oral therapy with prednisolone, hydroxychloroquine, and methotrexate may be instituted.[12]

Surveillance of behavior of scars form an important part of examination in a patient with suspected or proven sarcoidosis, as they are easily amenable for biopsy and alleviates the need of an invasive exploration to establish the diagnosis of sarcoidosis with systemic involvement. A careful and prolonged follow up is recommended in isolated cases of scar sarcoidosis due to potential risk of developing systemic sarcoidosis.

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