Case Report

Deck chair sign in lepromatous leprosy

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Abstract

A 40-year-old male presented with generalized asymptomatic cutaneous infiltrated plaques involving trunk, limbs and face. He had acral sensory loss along with symmetrical nerve thickening and enlargement. Slit-skin smear revealed multiple acid-fast bacilli (AFB) on Wade-Fite staining, thus confirming the diagnosis of lepromatous leprosy. Patient had a remarkable sparing of skin creases at various sites including trunk, antecubital fossa and axillary flexures suggestive of a positive "Deck Chair Sign". This sign has been described in literature mostly in papuloerythroderma of Ofuji and other related dermatosis. To our knowledge this is the first case report of 'lepromatous leprosy with deck chair sign' from Pakistan.

Key words

Leprosy, deck chair sign.

Introduction

Leprosy is a chronic infectious disease primarily affecting skin and peripheral nerves. It has a wide range of cutaneous manifestations that is why it is one of the great imitators in dermatology like syphilis. Cutaneous lesions of lepromatous leprosy are myriad in presentation, including widespread and symmetric papules, nodules, and diffuse infiltration.

'Deck chair sign' (DCS) is the sparing of flexures and skin folds, such as those of abdomen, antecubital and axillary areas which is usually observed in many inflammatory dermatoses, and was first described in papuloerythroderma of Ofuji (PEO). The sign is typical but nonspecific as it has also been described in several other conditions like, Waldenstorm's macroglobulinemia, angioimmunoblastic T-cell lymphoma, druginduced erythroderma, mycosis fungoides,

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acanthosis nigricans, discoid lupus erythematosus, acute contact dermatitis and large plaque parapsoriasis. ^{2,3,4} We herein, describe a case of lepromatous leprosy with deck chair sign, which is very infrequently mentioned in literature.⁵

Case Report

A 40-year-old gardener with no known comorbidities, presented with the complaints of diffuse infiltration and coarsening of skin over his whole body, most noticeable over his face and trunk for the last three years. He also complained of reduced sensation in both of his hands and feet, often getting injured while performing daily routine work since the last 3 years. There was history of rhinorrhea with nasal crusting and epistaxis, pedal edema and erythema nodosum leprosum lesions, which he developed thrice during his 3-year course of illness along with constitutional disturbances that resolved spontaneously, healing with postinflammatory hyperpigmentation. No significant past, family and drug history was noted.



Figure 1 Picture showing sparing of abdominal creases (Deck chair sign).

Examination revealed diffuse erythema and infiltration of face, ears, trunk, arms and thigh, but there was remarkable sparing of skin creases most notable over abdominal creases with some sparing of antecubital fossae (**Figure 1**). Other clinical findings were patchy hair loss over his body, madarosis, loss of eyelashes, dry and cracked lips. There was focal palmoplantar hyperkeratosis. Hands and feet were having areas of postinflammatory hypo- and hyperpigmentation along with previous trauma and burns injuries.

Sensory examination revealed patchy anesthesia over his hands and feet. There was bilaterally symmetrical thickening of his posterior auricular, supraclavicular, ulnar, radial cutaneous, lateral popliteal, posterior tibial and sural nerves. Multiple, enlarged, firm and nontender axillary and inguinal lymph nodes were found. Slit-skin smears showed multiple acid-fast bacilli with bacillary index (BI) of 5+. Currently, he is being treated standard multidrug therapy with lepromatous leprosy.

Discussion

Clinical signs are prelude to diagnosis of various dermatological conditions. DCS is one of them.¹ Although described earlier as a hallmark of PEO, a number of inflammatory dermatoses have been reported with such a specific clinical sign.

PEO is a rare cutaneous disease seen primarily in elderly and is characterized by erythematous papular eruption that progressively coalesces produce to erythematous plaques and when widespread, results in erythroderma. DCS is frequently seen in this condition.² The cause is unknown. Condition may be idiopathic or may be secondary to malignancies, both cutaneous lymphomas and internal malignancies, atopy, infections and drugs.4,5

Exact pathogenesis is unknown, although several hypotheses have been suggested to describe this sparing phenomenon. It may be related to 'occlusive dressing effect' of topical corticosteroids. As in these compressed areas, they last longer and absorbed more, thus producing the characteristic sparing pattern.^{4,5}

In our case, the characteristic sparing can be described by the fact that Mycobacterium leprae favours the cool superficial sites (<37°C), avoiding warmer areas of the body. As abdominal creases have a relatively higher temperature, they are therefore spared. This clinical sparing is considered as 'relative sparing' as is it not evidenced bacteriologically and histologically. Thus, it is not the local immunity but the temperature requirement of the organism that is manifested clinically.6 In a study by Pal et al.7 DCS was seen in just 5.5% cases of erythroderma. Hence, lepromatous leprosy is one of such rare cases, presenting with DCS.

Diagnosis of leprosy is clinical, bacteriological and histopathological.⁸ In our case of lepromatous leprosy with diffuse infiltrative form without classical papules, nodules and plaques of leprosy, DCS may have a diagnostic significance. Lepromatous leprosy should be kept in mind as one of the causes of DCS.

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