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Case Report

Erythema nodosum leprosum masquerading as Sweet's syndrome: a case report

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ABSTRACT

Leprosy is a chronic disease caused by *Mycobacterium leprae*, primarily affecting skin and nerves. Over time the progression of leprosy may be disrupted by acute inflammatory episodes known as lepra reactions. Type 2 leprosy reactions also known as erythema nodosum leprosum (ENL), are characterized by painful red nodules on the skin, along with symptoms like fever, joint pain, malaise, and systemic complications. There have been reports of unusual clinical presentations of this condition such asbullous, pustular, ulceration, erythema multiforme-like reaction, livedo reticularis, and Sweet's syndrome (SS)-like presentation. The pathogenesis of sweet's-like ENL is still uncertain. It is believed that in SS a complex interaction of various cytokines leading to buildup of pro-inflammatory cytokines in the target tissues, which trggers an abnormal immune response to certain antigens. These findings may help explain the occurrence of such unusual lepra reactions. In this case, we describe an uncommon instance of type 2 lepra reaction in a 28-year-old man who had no prior diagnosis of leprosy. The condition presented similarly to SS-like reaction and was diagnosed by slit skin smear and histopathological examination.

Keywords: Erythema nodosum leprosum, Sweet's syndrome-like reaction, Lepromatous leprosy, Atypical leprosy reactions

INTRODUCTION

Leprosy or Hansen's disease is a chronic infectious granulomatous disease caused by *Mycobacterium leprae*, affecting the skin and peripheral nerves.[1] The course of Hansen's disease may be interrupted by acute phenomena called leprosy reactions.[2] There are three types of leprosy reactions: Type 1 reaction (reversal reaction/ RR), type 2 reaction ENL, and Lucio's phenomenon.[1]

ENL, are marked by tender erythematous nodules, along with symptoms like fever, arthralgia, malaise, and systemic complications.[3] Some unusual clinical presentations of this condition have been reported such aspustular, bullous, ulceration, erythema multiforme-like reaction, livedo reticularis, and SS-like presentation.[1-4] This report describes a case where ENL reaction appeared in a manner similar to SS-like reaction.

CASE REPORT

A 28-year-old male from Bihar was admitted in medical emergency of our hospital with history of fever, myalgia, and multiple painful red raised skin lesions since 4 days. The skin lesions initially started over the left forearm progressed to involve right forearm, chest, back, and lower limbs. Few lesions showed central pus collection.

General physical examination showed no abnormality except for an elevated body temperature. Cutaneous examination revealed multiple tender erythematous bright

red papules, nodules with few lesions showing central pustules present over the trunk and extremities (Figure 1).

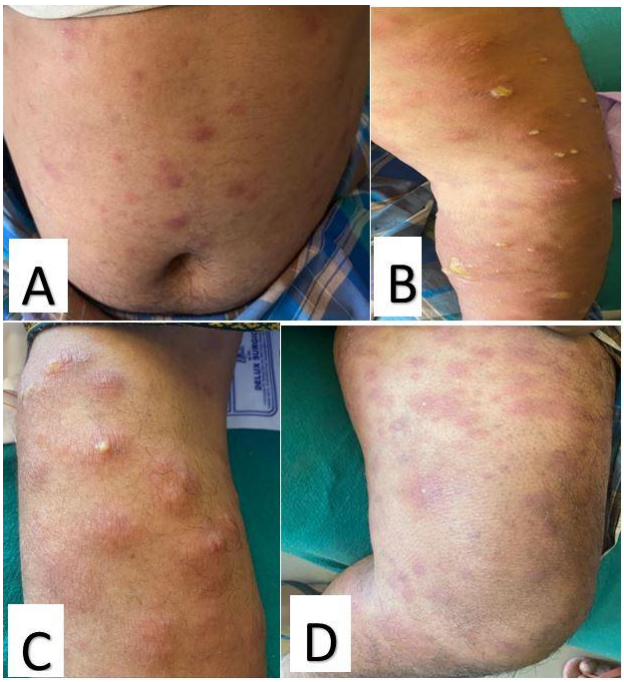

Figure 1 (A-D): Erythematous, edematous, tender plaques of variable size with few lesions showing central pustules seen over the abdomen, upper and lower extremities.

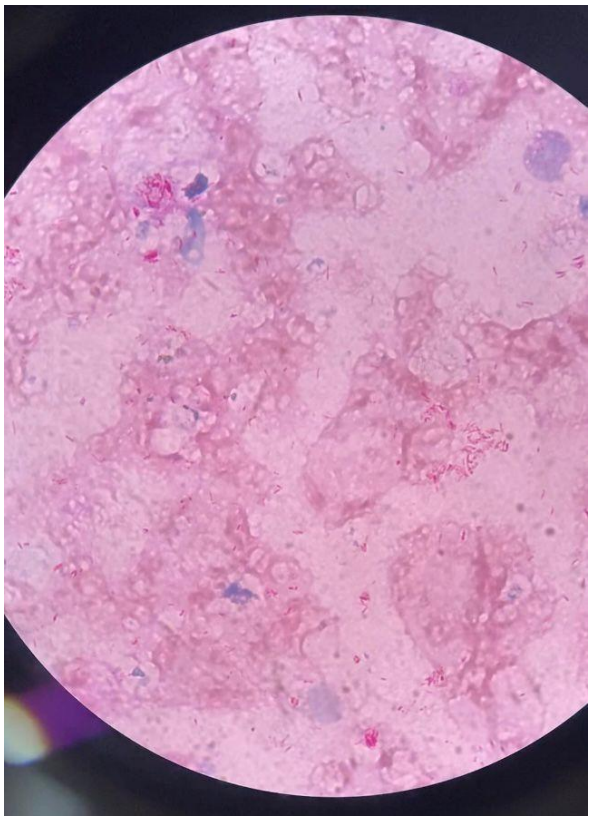

Figure 2: Slit skin smear showing acid fast bacilli lying singly and in globi under 100×magnification.

There were no hypopigmented patches over the body. No significant nerve thickening, sensory loss or motor deficit noticed.

Blood test results showed leukocytosis of 10900 cells/ml, neutrophilia of 80%, hemoglobin of 10.9 g/dL, Erythrocyte Sedimentation Rate of 92 mm/h and raised C reactive protein 117 mg/ml.

Differential diagnoses of SS and ENL was made.

Slit skin smear from bilateral ear lobes and a nodule from the thigh showed bacillary index of 4+ (Figure 2).

Biopsy showed hyperkeratosis, periadnexal lymphocytic cells infiltrate in the dermis. Lepra stain for positive for acid fast bacilli arranged in singles and globi (Figure 3).

The patient was treated with multibacillary multidrug therapy and oral prednisolone at a dose of 1mg/kg/day which was tapered gradually over a period of 2months. Patient responded well to the treatment, after which patient was lost to follow up due to return to his hometown that is Bihar.

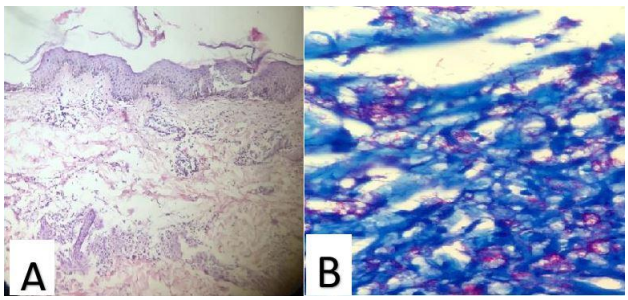

Figure 3 (A and B): A-Hematoxylin Eosin histopahology at 10× magnification showing thinning of epidermis with mild spongiosis with grenz zone in papillary dermis, granulomas composed of few epitheloid cells, foamy macrophages and lymphocytes. perivascular, periadnexal mixed inflamatory cell infiltrate of lymphocytes, neutrophils and histiocytes noted. B-Fite-Faraco stain at 100× magnification showing abundance of lepra bacilli.

DISCUSSION

ENL is a type 3 hypersensitivity reaction as classified by Coombs and Gell associated with bacterial destruction and release of large quantities of antigen, which induces antibody production and form antigen–antibody complexes. These antigen–antibody complexes will activatecomplementary reaction which causes acute inflammation of tissue in the form of erythema nodules.[3] This reaction occurs exclusively in LL, occasionally in BL as tender erythematous nodules with constitutional symptoms, but there are reports of atypical and rare clinical manifestations such aspustular, bullous, ulceration, livedo reticularis, EM-like reaction or SS-like reaction.[1-4]

SS is a neutrophilic dermatosis, characterized by papules, plaques, and erythematous or voilaceous painful nodules, some with pseudo-vesicular appearance and central pallor with constitutional symptoms such as fever and malaise.[5] Blood examination shows leukocytosis with high neutrophilia. The histopathology reveals diffuse infiltration of matured neutrophils, upper dermal edema, endothelial cells swelling, and blood vessel dilatation. Diagnostic criteria of SS were as follows. [2,5]

Major criteria

Abrupt onset of painful erythematous plaques or nodules and histopathologic evidence of a dense neutrophilic infiltrate without evidence of leukocytoclastic vasculitis.

Minor criteria

Pyrexia >38°C (100.4°F), association with an underlying hematologic or visceral malignancy, inflammatory disease, pregnancy, or onset following an upper respiratory or gastrointestinal infection or vaccination, excellent response to treatment with systemic corticosteroids or potassium iodide and abnormal laboratory findings at presentation (≥3 of the following): Erythrocyte sedimentation rate >20 mm/h, elevated C-reactive protein, leukocytosis (>8,000 cells/µl), neutrophilia (>70%).

The diagnosis of classic SS requires the presence of both major criteria and at least two of the four minor criteria.

For drug-induced SS-A. Abrupt onset of painful erythematous papules or nodules, B. Histopathologic evidence of a dense neutrophilic infiltrate without evidence of leukocytoclastic vasculitis, C. Pyrexia >38°C (100.4°F), D. Temporal relationship between drug exposure and clinical presentation and E. Resolution of lesions following drug withdrawal.

All five criteria (A-E) must be met to establish the diagnosis of drug-induced SS.

Here we describe a case presenting with multiple erythematous, tender plaques and nodules with central pustules with no prior evidence of neurological deficits or chronic skin lesions suggestive of lepromatous leprosy. This was a clinically challenging case and was misdiagnosed a SS initially.

SS-like leprosy presentation is rarely reported, making it difficult to diagnose, especially in patients who have not been diagnosed with leprosy previously.[6] SS-like reaction was firstly reported by Kuo et al.[7] Similar case has been reported from Qatar by Al Hayki et al with features of tender erythematous nodules, plaques with central pustules and ulcers, was treated as SS initially and was later diagnosed as ENL.[8]

In this case, the diagnosis of SS-like leprosy reaction was based on the slit skin smear and histopathology reports.

Systemic corticosteroid is regarded as the first line and effective management for severe type 2 leprosy reaction. In some cases, the patient appears to be steroid dependence (the leprosy reaction occurs when the dose is tapered), so the patient needs a sparing agent to reduce the risk of long-term side effect of corticosteroid.[2] Thalidomide is an effective option as steroid sparing agent as it inhibits TNF α and neutrophil recruitment.[8]

The patient was treated with multibacillary multidrug therapy and oral prednisolone at a dose of 1 mg/kg/day which was tapered gradually over a period of 2 months. Patient responded well to the treatment, after which patient was lost to follow up due to return to his hometown that is Bihar.

CONCLUSION

Though we have reached elimination state in our country but some endemic states are still harbouring active untreated Lepromatous leprosy cases specially Bihar, Jharkand, Chattisgarh, Uttar Pradesh, West Bengal, Orissa, Madhya Pradesh.

A high index of suspicion is essential. Leprosy and ENL should be considered as one of the differential diagnoses and thoroughly investigated and to be ruled out on priority. It helps in early diagnosis, minimizes complications and deformity.

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