

Type 1 Lepra Reaction With Ulceration (Lazarine Leprosy) - Two Interesting Case Reports.

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Abstract: Leprosy is a chronic granulomatous disease caused by mycobacterium leprae with wide spectrum of clinical, histopathological and immunological characteristics. Leprosy reactions mainly encountered are type1 and type2 lepra reactions. In type 1 leprosy reaction, the pre existing lesions become erythematous, oedematous and rarely ulcerate. Ulcerating type 1 reaction is called lazarine leprosy. Ulcerations may occur in borderline tuberculoid pole or borderline lepromatous pole but more common in borderline tuberculoid pole. In this post elimination era o fleprosy, we are reporting two interesting case reports of type 1 lepra reaction with ulceration, one is borderline lepromatous leprosy and another is borderline tuberculoid leprosy.

Keywords: borderline leprosy, lazarine leprosy, type 1 leprosy reaction.

I. Introduction

Leprosy is a chronic disease caused by Mycobacterium leprae with wide spectrum of clinical, histopathological and immunological characteristics. Leprosy reactions mainly encountered are type 1 and type 2 lepra reactions. Type 1 lepra reaction is a cell mediated immune reaction seen in borderline forms of leprosy, the pre-existing lesions become erythematous, oedematous rarely ulcerate. This rare ulcerating type 1 lepra reaction is called Lazarine leprosy first described in 1852 by Raphael lucio and Ignacio Alvarado[1].

II. Case Report 1

A 50 year old female patient presented with ulcers on both lower legs and copper coloured patches all over the body since 3 months. There was history of multiple hypopigmented patches all over the body 2 years ago, associated with decreased sensation and sweating over the patches. She was diagnosed outside as leprosy and started on MB-MDT. She took irregular treatment for 3 months and stopped. After stopping MB-MDT gradually hypopigmented patches became oedematous, erythematous, copper coloured and ulcerated. There was pain, swelling, tingling and numbness, decreased sensations over both lower legs.

On examination patient was afebrile, bilateral pitting type of pedal edema was present. Cutaneous examination revealed ulcers, 4 in number over both lower legs with well defined margins. Floor was covered with reddish granulation tissue, surrounding skin was hyperpigmented. There was sensory impairment and loss of hair (fig 1). There were multiple (18-20), erythematous to copper coloured patches all over body, with decreased sensation and hair loss.(fig 2).

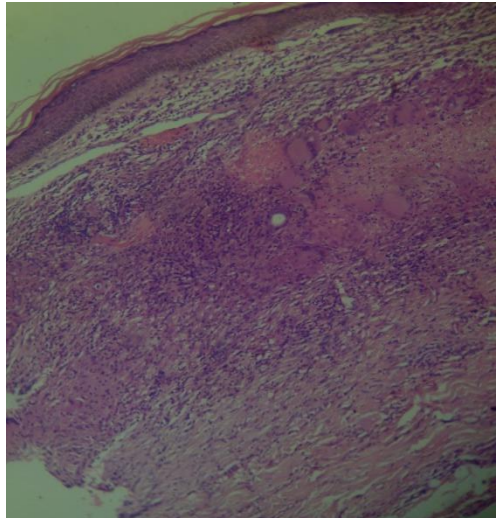
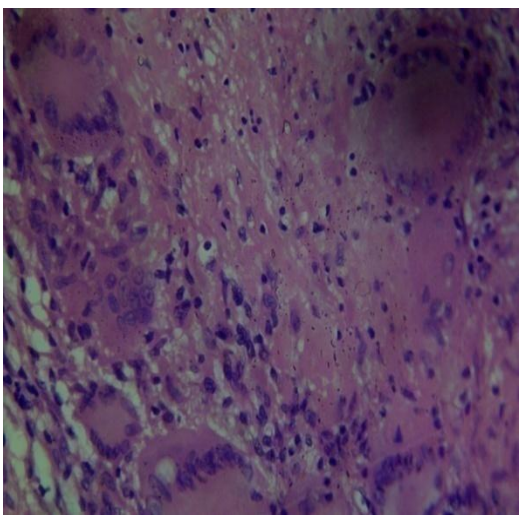


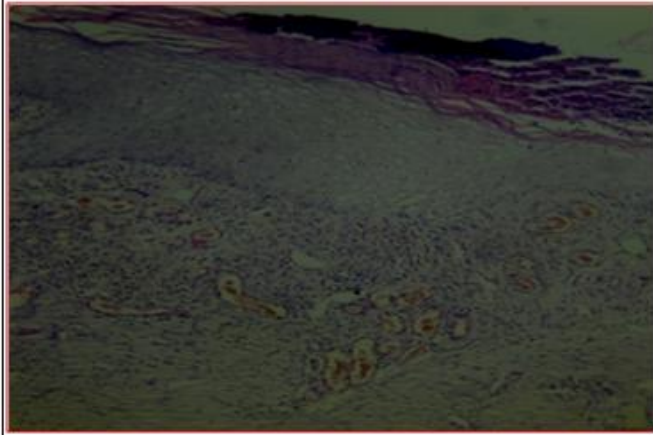
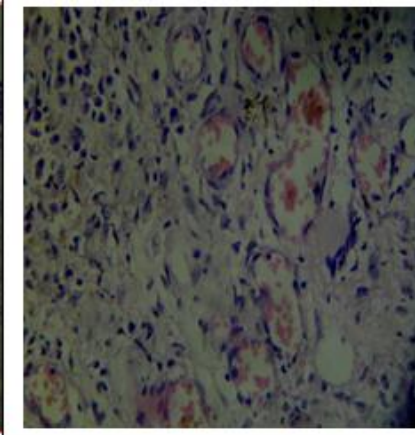
Figure 1	Figure 2	Figure 3	Figure 4
Fig 1 & 2: Ulcers with red granulation tissue with surrounding hyperpigmentation.		Copper coloured plaque over right thigh	Copper coloured plaque over forearm

Peripheral nerve trunks were thickened and tender bilaterally. Sensations to fine touch, pain and temperature were decreased over the distribution of left radial cutaneous nerve, bilateral ulnar nerves, bilateral superficial and left deep peroneal nerve. Motor examination there was weakness in dorsiflexion of left great toe and ankle.

Haematological investigations show normal counts, elevated ESR(75mm 1st hr), normal blood glucose levels. VDRL & VCTC tests were non-reactive. Slit skin smear was 3+. Histopathology from hyperpigmented plaque, shows thinned out epidermis, dermis shows granulomas made of epitheloid cells, lymphocytes, plasmacells, fibroblasts, plenty of langhans & foreign body giant cells with central caseous necrosis, perivascular & perineural granulomas suggesting an upgrading reaction (fig 5 and 6).

Biopsy from ulcer edge shows hyperkeratosis, acanthosis in epidermis, acute inflammatory cell collection in stratum corneum. Dermis shows capillary proliferation, langhans type of giant cells, mild to moderate inflammatory cell collection, subcutaneous tissue shows perivascular both acute & chronic inflammatory cell collection (fig 7 and 8).

	
Figure 5	Figure 6
<p>Fig 5 & 6 : Histopathology from copper coloured plaque Figure 5. 10x Optical microscopy- Caseation and granulomas in dermis Figure 6. 40x optical microscopy- central caseation surrounded by gaint cells, plasma cells, epitheloid cells</p>	

	
Figure 7(10x)	Figure 8(40x)
<p>Fig 5&6: Histopathology from ulcerated plaque Fig 6: Giant cells and mixed inflammatory cell infiltrate</p>	

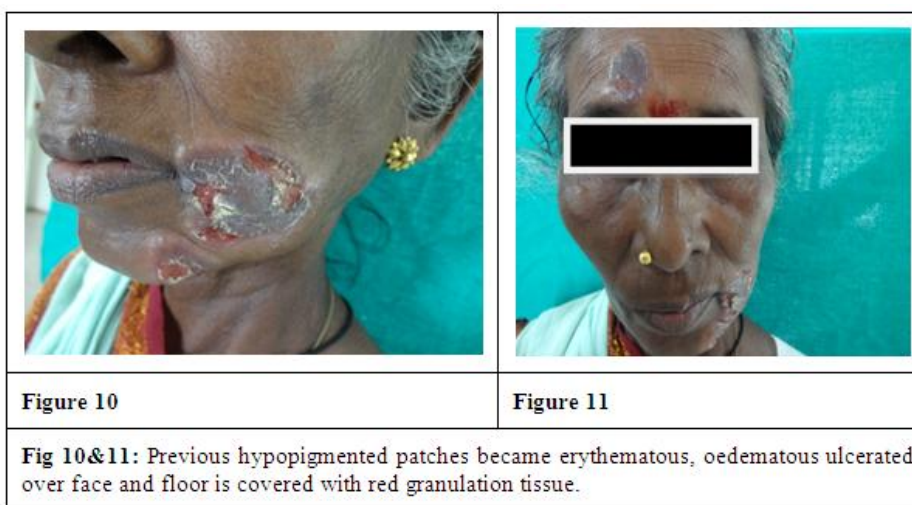
She was started on MB-MDT, oral prednisolone, antibiotics and supportive measures. After 3 weeks of treatment ulcers healed with hypochromic atrophic scars with hyperpigmented border(fig 9).



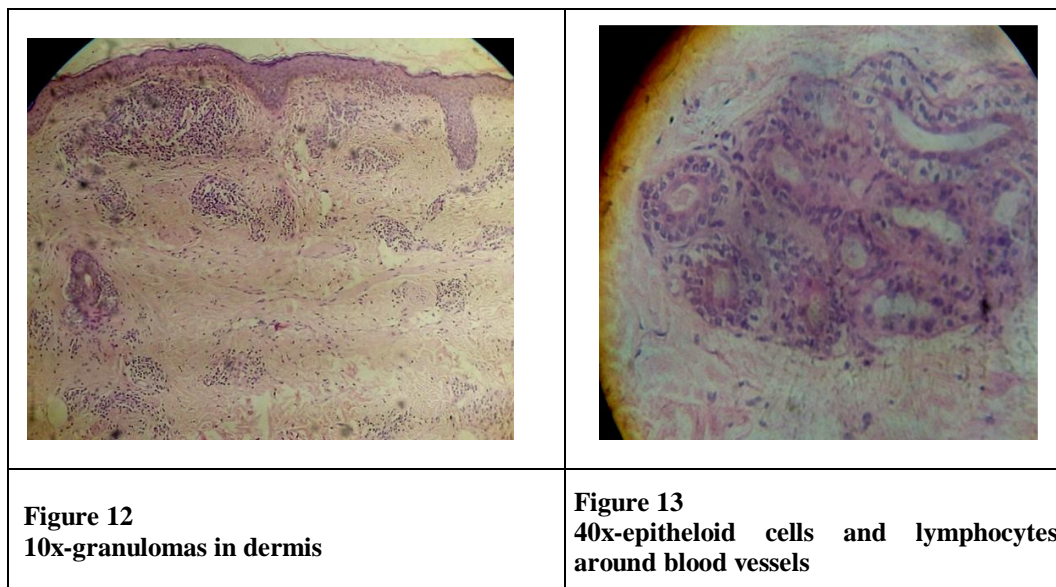
III. Case Report 2

A 65 year old female presented with ulcerations over face since 3 months. One year ago lesions started as hypopigmented patches with decreased sensation over the body. She was diagnosed as borderline tuberculoid leprosy and started on MBMDT. After continuing treatment for 5months lesions over face turned oedematous, erythematous and ulcerated.

On examination there were superficial ulcers (3 in number)with base covered with red granulation tissue and there was peripheral nerve thickening and decreased sensations over the hypopigmented patches. There was no motor deficit. Slit skin smear shows no AFB.



Routine laboratory investigations were normal,serology for HIV ,VDRL tests were negative. Histopathology from the plaque over face shows granulomas made of epitheloid cells,plasma cells and lymphocytes around adenexae and blood vessels ,features are suggesting of Borderline Tuberculoid hansens.



She was diagnosed as borderline tuberculoid leprosy in type 1 reaction with ulceration and treated with NSAIDs and steroids. Ulcers healed within 4 weeks.

IV. Discussion

Leprosy is a chronic granulomatous infectious disease caused by *Mycobacterium leprae*. There is no other human infectious disease in which the clinical picture is as varied as leprosy. Type 1 reaction is a delayed type of hypersensitivity, where the pre-existing lesions become erythematous, oedematous and rarely ulcerate. This rare ulcerating type of type 1 reaction is called lazarine leprosy. According to the literature, this rare ulcerating type 1 reaction first demonstrated as lazarine leprosy in 1852 by Raphael Lucio & Ignacio Alvarado[1]. Later in fifth international congress of leprosy held in 1930, Pardo Castello in a study of 23 cases concluded that lazarine leprosy occur in two polar forms tuberculoid and lepromatous pole, former is because of high inflammation and later is because of high bacillary load[2,3,4]. But the present view, the exact pathogenic mechanism is not known, but factors such as breakdown of local immunity, increased proliferation of bacilli and severe tissue edema may be implicated. Protein malnutrition has been suggested as a main event in the pathogenesis of ulceration[5]. However it has also been seen in BT leprosy without any malnutrition or immunosuppression[6].

The first case was initially a borderline lepromatous leprosy, after taking MB-MDT for 3 months, went into severe type 1 reaction with ulceration (lazarine leprosy). Histopathology was correlating with reversal (upgrading) reaction as there were many granulomas with caseation, epithelioid and giant cells. Second case was borderline tuberculoid form after taking MB-MDT went into type 1 reaction with ulceration (lazarine leprosy), histopathology showing typical tuberculoid granulomas with Langhans type of giant cells.

This severe reactionary state has occurred in borderline lepromatous leprosy (case 1) and borderline tuberculoid leprosy (case 2) in type 1 reaction due to developing high degree of inflammation during a reversal reaction [5,6].

Both the cases responded to oral prednisolone and MB-MDT, ulcers got healed with hypochromic atrophic scars. In the post-elimination era of leprosy and with effective implementation of MBMDT, we are coming across this rare type of ulcerative leprosy reactions, type 1 leprosy reaction which is a very rare entity. Early management of these reactions will decrease disease burden and complications - ENHANCED GLOBAL STRATEGIC plan 2011-2015, necessitating the role of dermatologists at tertiary level even after effective primary implementation of MBMDT.

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