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Leprosy Mimicking Psoriasis

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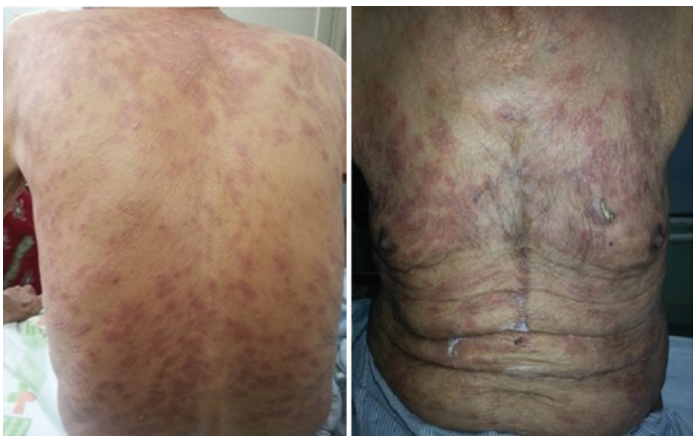
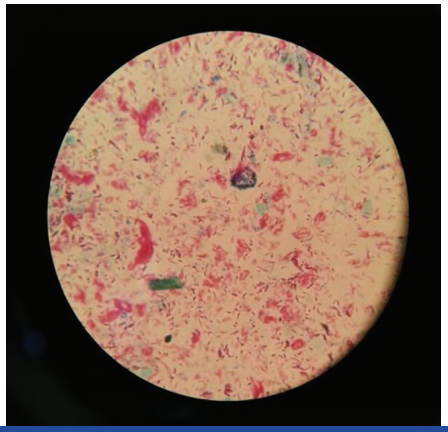
Leprosy is an ancient, chronic granulomatous disease caused by acid fast bacilli, *Mycobacterium leprae*, affecting all age groups and has no sex predilection. Usually the disease presents with hypopigmented patches, nodules and plaques with or without loss of sensations and thickening of nerves. Leprosy has a wide range of presentation which can mimic various other dermatoses like psoriasis, dermatitis, granulomatous disorders like sarcoid, leishmaniasis, etc. The differential diagnosis is so wide that one has to exclude wide variety of dermatological diseases before stamping it to be leprosy as stigma is still associated to it [1]. Biopsy remains the gold standard method of diagnosing and grading various forms of leprosy. In view of its potentially debilitating effect, physicians practicing should have awareness of the possibility of leprosy and be familiar with its varied presentations. We hereby, present a case of leprosy being treated as psoriasis which was later diagnosed as leprosy and treated with antileprosy drugs.

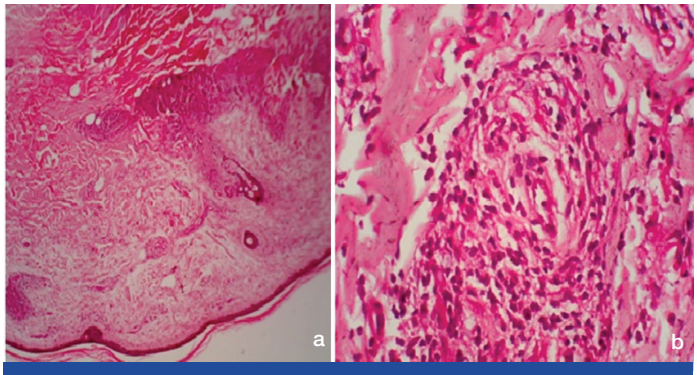
A 76-year-old male presented with complaints of multiple lesions over body since 4 years, fever since 2 months and weight loss since 1 month. The patient was diagnosed and treated as psoriasis since 4 years by a private practitioner. There was no complaint of itching, no winter aggravation, and no loss of sensations over the lesions, no loss of sensations over the distal part of the extremities, no loss of chappals, no epistaxis or nasal stuffiness. There was no previous history of pain in the lesions, joint pains or pedal oedema. On examination, multiple well to ill defined, non-blanched, erythematous plaques with mild scaling were present over abdomen, back, bilateral upper, lower limbs [Table/Fig-1a&b]. There was no loss of eyebrows, no nail changes. There was no ear lobe infiltration. Bilateral ulnar nerves were palpable and non tender. Rest all nerves were normal. Touch and temperature sensations were normal all over body. There was generalised lymphadenopathy which was suggestive of reactionary lymphadenopathy. All the routine blood investigations were normal except for ESR which was raised i.e. 80 mm. He was seronegative for HIV (Human Immunodeficiency virus). Chest X-ray was normal. Mantoux test was negative. At this point patient was labelled as a case of pyrexia of unknown origin as the cause of fever was not identified clearly. At this juncture, the differential diagnosis were psoriasis, mycosis fungoides and lepromatous leprosy. Slit skin smears were positive for acid fast bacilli with Bacterial index of 6+ (plenty of uniformly stained lepra bacilli arranged singly and in globi) [Table/Fig-2]. Morphological index was not done as it is not done in our set up, moreover bacteriological index showed all live bacilli. Biopsy revealed thinned out epidermis, clear grenz zone, poorly formed granuloma containing epithelioid cells, foamy macrophages, lymphocytes and plasma cells, granuloma involving cutaneous appendages suggestive of lepromatous leprosy [Table/Fig-3a(10X)& b(40X)]. Wade-Fite staining showed globi of acid fast bacilli within macrophages suggestive of lepromatous leprosy [Table/Fig-4]. Patient was started on anti-leprosy treatment and responded well [Table/Fig-5]. Patient was given Tab Rifampicin 600 mg, Tab Dapsone 100 mg and Tab Clofazamine 50 mg once daily for 2 weeks. Then patient was started on MB packs (Tab Rifampicin 600 mg, Tab Clofazamine 300 mg, Tab Dapsone 100 mg once per month and Tab Clofazamine 50 mg and Tab Dapsone 100 mg for rest of the month) [Table/Fig-5]. Repeat slit skin smear was done after 1 month of treatment which showed few fragmented lepra bacilli arranged singly and in globi.



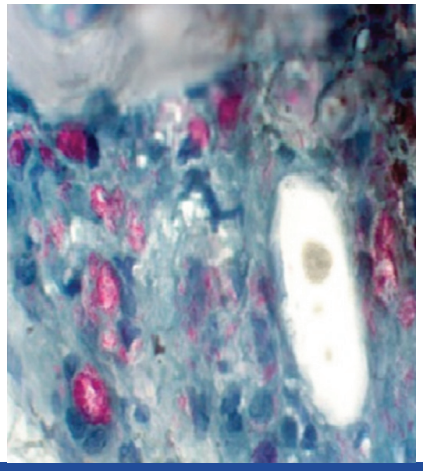
[Table/Fig-1a&b]: Multiple discrete well defined erythematous non blanchable plaques with mild scaling over trunk



[Table/Fig-2]: Slit skin smear showing lepra bacilli



[Table/Fig-3a&b]: Thinned out epidermis, clear grenz zone, poorly formed granuloma containing epithelioid cells, foamy macrophages, lymphocytes and plasma cells. granuloma involving cutaneous appendages. (10X& 40X)

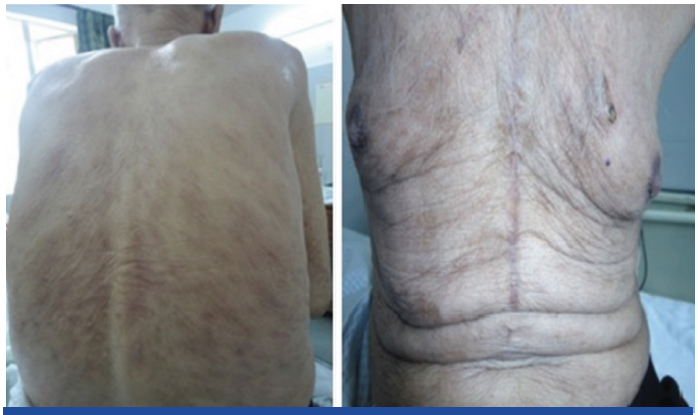


[Table/Fig-4]: Wade-fite showing lepra bacilli

Leprosy is a dermato-neurological chronic infection caused by *Mycobacterium leprae,* acid-fast intracellular bacilli, not cultivated invitro [2]. Cytoplasm of Schwann cell is its preferred target [3]. It is a mutilating, stigmatizing disease, early diagnosis and therapy is the most important strategy for its control. The diagnosis of leprosy is made from the clinical picture, but must be complimented by biopsy and slit skin smear. Psoriasis is characterized by an abnormally excessive and rapid growth of the epidermal layer of the skin. A diagnosis of psoriasis is usually based on the clinical appearance of lesions. Psoriasis clinically present as characteristic well defined erythematous plaques with silvery scales along with complaint of itching.

Relationship between leprosy and psoriasis is being controversial since long time. Earlier people considered psoriasis to be a form of leprosy and the biblical term “lepra” in ancient time was used as what we called psoriasis now [4]. This confusion between leprosy and psoriasis lasted for almost 19 centuries when it was realized that the two diseases are entirely different and have nothing common.

Clinical diagnosis of leprosy depends on the history, clinical features and histopathology. No other findings were favouring the diagnosis of psoriasis apart from the classical clinical presentation. If the clinical diagnosis is uncertain, a skin biopsy or slit skin smear may be performed to rule out other disorders and to confirm the diagnosis.



[Table/Fig-5a&b]: Resolution of lesions post Anti-leprosy treatment

Leprosy has a number of distinct clinical presentations, so it can be confused with many conditions like Granuloma Annulare, Leishmaniasis, neurofibromatosis, psoriasis, sarcoidosis, syphilis, tinea versicolour, vitiligo, xanthomas. Hence proper history taking and examination with strong suspicion is required to diagnose atypical presentations of leprosy. Histological examination is required to confirm the diagnosis when dermatological features are not typical [5]. If the clinical diagnosis is uncertain, skin biopsy is the gold standard to confirm the diagnosis.

There is increasing number of cases with unusual presentation leading to diagnostic dilemma. Undetected early cases form a major risk for transmission and disabilities. Our case uphold the importance of histopathology in diagnostic dilemma along with active and sustained surveillance.

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