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Case Report

Buruli Ulcer Disease like Lupus Vulgaris

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Abstract
Lupus vulgaris is the commonest variant of cutaneous tuberculosis seen in adults. It has unusual sites of presentation, varied morphological types and variable course. This occasionally leads to delay in diagnosis and increases the morbidity. A 27 year old woman presented to us with rapidly spreading recurrent, large ulcerative lesions on buttocks. Patient was misdiagnosed as pyoderma gangrenosum and was started on steroids. There was no significant improvement. Smear examination and culture for tuberculosis were negative. Histopathological examination and therapeutic trial confirmed the diagnosis.
Keywords: *lupus vulgaris, therapeutic trial.*

Introduction

Lupus vulgaris (LV) is one of the commonest recognized expressions of cutaneous tuberculosis, accounting for approximately 55-59% of secondary skin tuberculosis cases in India.[1] Lupus vulgaris is a great cutaneous mimc next to syphilis and can present in protean forms Misdiagnosis, neglect, late diagnosis or irregular therapy may cause several serious complications like disfigurement, contracture, lymphoedema and skin cancers including squamous and basal cell carcinoma or plasmacytoid lymphoma.[2]

Case History

A 27 year old woman presented to us with ulcerated lesions on body for last 1 month. The lesion started on the gluteal region as a painless small nodule, which ulcerated in about a week and gradually progressed to involve whole of the gluteal area, lower back, groins, genitals and thighs. Lesions became painful and purulent after 3 weeks. She had similar lesion on right leg for 2 weeks. No history of preceding trauma or any other systemic involvement was there. Past history of similar lesions on face was there when patient was of 4 years of age, these lesions subsided

within 3 years without any treatment. Again recurrence of similar lesions was there at the age of 16 years when she was diagnosed to be having koch's chest also and was started on anti tubercular treatment (ATT), which she took for 3 months, her cutaneous lesions also improved. There was no family history of similar lesions. Patient had mild pallor. On mucocutaneous examination patient had tender ulcerative lesion of size approximately 30 cms x15 cms with violaceous margins and undermined edges (Figure 1). The floor of ulcer was covered with dirty white slough (Figure2).

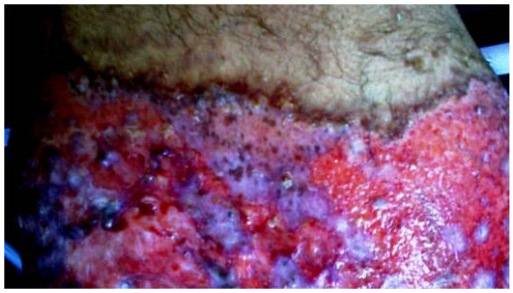

Fig. Ulcerative lesions wth voilaceous and undermined edges

There was cribriform scarring over face (Figure 3), forearms, neck, abdomen and legs.

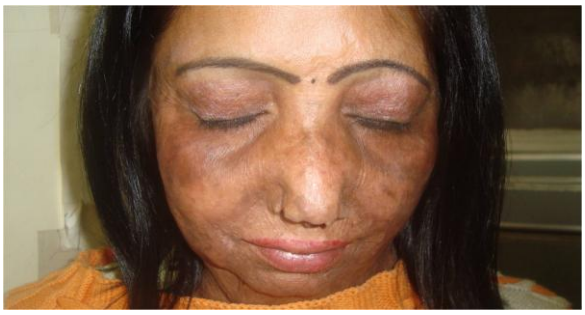

Fig. 3 Cribriform scarring of face

Haemoglobin was 7 mg%, rest of the haemogram, renal function test, liver function test, blood sugar were normal. Antinuclear antibody, rheumatoid factorwere negative. A diagnosis of pyoderma gangrenosum was made. Patient was started on high dose of systemic steroids and azathioprine. There was no improvement. Pus culture sensitivity grew klebsiella, patient was given antibiotics according to sensitivity (Amikacin) for 2 weeks, tenderness subsided. Histopathologic examination showed epitheloid cell granoluma, mixed inflammatory cell infiltrate, langhan and foreign body type of giant cells, areas of fibrinoid necrosis & degenerated collagen extending up to subcutaneous tissue (Figure 4).

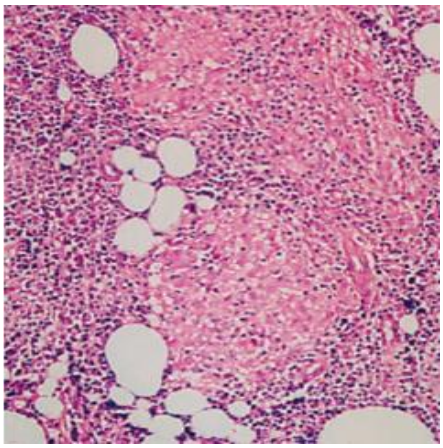

Fig.4 Histopath showing non caseating granuloma Mantoux test was highly positive, Ziehl Neelsen's staining of pus for AFB and culture were negative. Polymerase chain reaction for mycobacterium tuberculosis complex was also negative. Patient could not afford any other confirmatory tests. Chest radiography and HRCT showed small fibrocavitatory leson bilateral upper lobe suggestive of old healed tuberculosis. She was started on a therapeutic trial of antitubercular (ATT) category 2 drugs. She responded very well to treatment. There was 90 % improvement within 6 weeks (Figure 5a). Final diagnosis of lupus vulgaris was made. Lesions healed after 5 monthsof ATT (Figure 5b). Patient was prescribed category 2 ATT for 9 months.

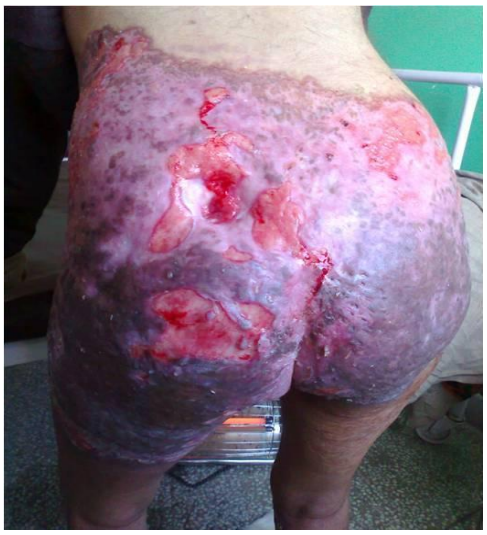

Fig.5 a) Lesions after 6 weeks of ATT b)after 5 months of ATT

Discussion

Cutaneous tuberculosis is caused by *Mycobacterium tuberculosis, M. bovis* and, sometimes, the *Calmette-Guérin bacillus (BCG)*. Studies from India report an incidence of cutaneous tuberculosis to be approximately 0.2%.[2]

The balance between the invading organism and the immune ability of the host to control the infection plays a decisive role in determining the type and extent of disease produced. Cuaneous tuberculosis can be caused by exogenous inoculation of bacteria or can spread from some underlying tubercular focus by auto inoculation, haematogenous or lymphatic spread.

The earliest description of lupus vulgaris was given by Erasmus Wilson in 1865, which compared the lesions to ravages of a wolf.[3] Lupus vulgaris is in most instances a chronic slowly progressive disease, occurring in patients with immunity produced by previous tuberculous infection. It is a paucibacillary form of tuberculosis. LV is caused by spread from an active underlying focus –cutaneous /systemic or reactivation of latent cutaneous focus secondary to previous bacteraemia. Exogenously it can be acquired by inoculation or as a complication of BCG vaccination.

Commonest clinical variants is the plaque form, which presents as slowly progressive reddish brown plaque. The plaque grows by peripheral extension, while healing at one end. Other frequently presenting forms are-ulcerative, vegetative, nodular, popular, tumourous. Various atypical presentations are framboesiform, gangrenous, ulcerovegetating, lichen simplex chronicus, sporotrichoid types,[5] basal cell carcinoma like, annular lesions, mycetoma like, actinomycosis like, port wine stain, alopecia, psorasiform, pyoderma gangrenosum like, etc Atypical distribution like disseminated, necklace form, penile, vulval, etc. In literature there is no mention about buruli ulcer like presentation of LV.

The Buruli ulcer (also known as the Bairnsdale ulcer or Searls ulcer) Buruli ulcer disease was identified in 1897 by Sir Albert Cook. [6] Aetiologic agent Mycbacterium ulcerans lives in aquatic animals, insects and biofilm. Buruli ulcer is mainly seen in tropical regions. The lesions are most commonly localized to limbs. Painless subcutaneous nodule, papule or plaque which spreads rapidly and later ulcerates with deep undermined edges, with necrotic fat forming the floor. Undermining of edges and progressively expanding ulcer is due to mycolactone toxin produced by the organism, which causes necrosis of dermis. Mycobacteria can be found in spherical clumps. Treatment with amikacin and rifampicin for 8 weeks leads to complete healing in majority of patients.

A proper diagnosis of skin tuberculosis requires a good correlation of clinical findings and diagnostic testing, such as AFB smears, cultures, PCR and histopathological examination. Although direct microscopic examination of AFB is rapid, its sensitivity and specificity are low. It needs 10^4 bacteria/mg of tissue for detection, and it cannot discriminate pathogenic and non pathogenic bacteria. The overall sensitivity of PCR has ranged from about 50 to 72% indifferent studies using IS6110 primers.[7] Because of the non-homogenous distribution of small numbers of bacilli, multiple sampling should be performed.

Buruli like presentation in our patient was ulcerative lesions with undermined edges, fast progression, initially painless, distribution of lesions on lower limbs, early response to ATT.

Our patient could not be suffering from buruli ulcer disease as the disease is not endemic in our part of country, recurrences in the same patient only cannot be explained and the histopathological features do not support the diagnosis.

Other diagnosiswhich were considered were pyoderma gangrenosum, amoebic ulcer, deep fungal infections. Response to ATT ruled out these conditions.

Atypical presentations of cutaneous tuberculosis are not so uncommon and are frequently overlooked in clinical practice, leading to late diagnosis and increased morbidity. Antitubercular

drugs is often rewarding when tuberculosis is suspected but cannot be proven.

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