UNDIAGNOSED LEPROSY FOR TWO YEARS CASE REPORT.

Salim A/Rahman Al-Harmozi
Mohammad Mohy El- Din Selim
Department of Dermatology and Venereology,
Hamad Medical Corporation,
Doha, Qatar.

Case presentation:

A 53-year old female from Yemen, presented with a two years history of slowly progressive, asymptomatic plagues on the face and solitary annular patch on the left thigh. She had no systemic symptom and was otherwise in good health. Physical examination revealed three different sized purple plaques located on the left eyebrow, right nostril and on the lower lip. [Fig1 , 2, 3] and annular lesion affected the left thigh [Fig.4].

All the lesions exhibited decreased sensation. There was no lymphadenopathy or organomegaly. Laboratory data obtained included results of a complete blood count, hepatic panel, renal function tests, rapid plasma reagin test and measurement of glucose-6-phosphate dehydrogenese levels and the results were within normal ranges. A chest showed normal finding. Two biopsy specimens obtained from the eyebrow and the thigh lesions.

Both showed infiltration of all dermal layers by multiple granulomata consisting of epitheliod cells and histocytes associated with lymphocytes, accumulation of foamy macrophages, with nerve infiltration and no acid fast bacilli were detectad on Fite's or ZN staining. (Fig. 5 & 6).

The patient was diagnosed to have tubeculoid leprosy and was given dapsone 100mg daily and rifampine 600mg monthly as treatment for six months.

Fig. (1): Purple plaque on the left eyebrow.

Fig. (2): Plaque on the right nostril.

Fig. (3): Purple plaque on the lower lip.

Fig. (4): Annular patch on the left thigh.

Correspondence:
Dr. Salim A/R. Al-Harmozi.
Department Of Dermatology and Venereology,
Hamad Medical Corporation.
P.O.Box, 3050, Doha, Qatar.

Fig. (5): Infiltration of dermal layers by multiple granulomas.

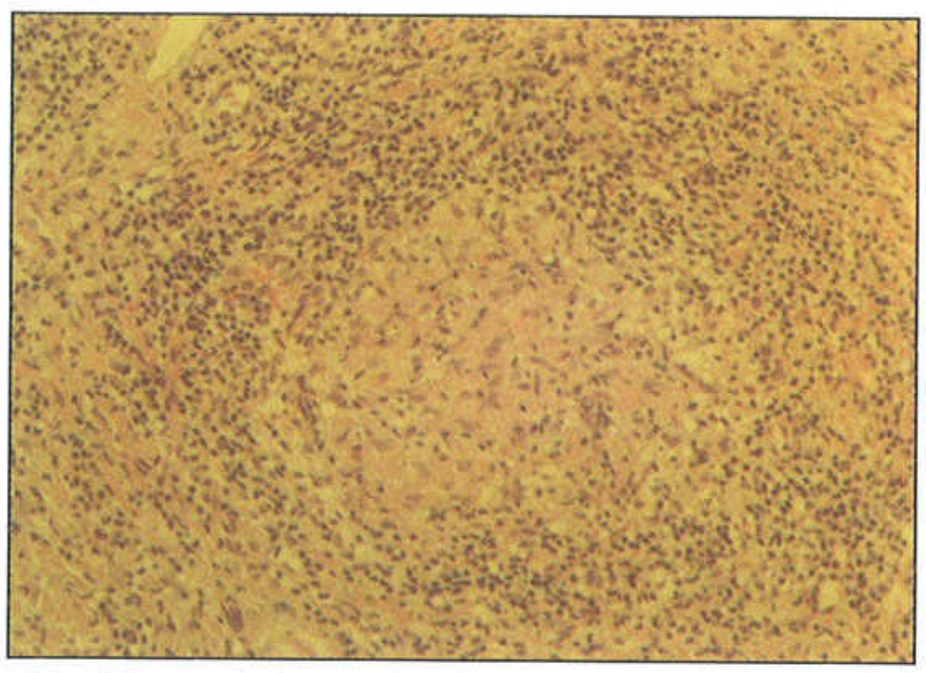

Fig. (6): Epitheliod cells , histocytes with lymphocytes, accumulation of foamy macrophages and nerve infiltration.

DISCUSSION

Leprosy manifests a broad spectrum of clinical and hitologic features reflecting antigen-specific immune response to mycobacterium leprae.

The Ridly-Jopling classification divides leprosy into two polar types tuberculoid(TT) and lepromatous(LL) and three borderline types[1]. Clinically tuberculiod lesions in comparison with lepromatous leprosy are larger, fewer, more sharply demarcated and more asymmetrically distributed with anesthesia and loss of dermal appendages[2].

Histologically, tuberculoid lesions have infiltrates of epitheliod macrophages, multinecleated giant cells and numerous lymphocytes that form cuffs around the granulomas, with infiltration of cutaneous nerves[3].

The T- cell mediated immune response plays an important role in leprosy. and the infiltrates in tubeculoid infections are composed mainly of T- helper/inducer(THI) cells, which secrete proinflammatory cytokines, including interleukin(IL)-2 and interferon gamma, that promote a delayed-type hypersensitivity response[4].

Since mycobacterium leprae are rarely demonstrable in the tuberculoid spectrum of leprosy, a confirmatory diagnosis of leprosy can be made on the basis of finding active destruction of cutaneous nerves by granulomatous inflammation in a skin biopsy, immunoperoxidase staining for S-100 protein which is a marker for schwann cells was used to delineate nerves in lesional skin biopsies of 25 patients with tubeculoid and borderline tuberculoid leprosy as well as 15 controls with non-leprous granulomatous inflammation.

All of the non-leprous granulomatous dermatosis showed intact nerves, either inside or outside the granuloma, while all the leprous granulomas showed different patterns of nerve damage. So S-100 staining can be used to rule out leprosy[5].

Treatment of leprosy consisting of multidrug therapy as recommended by the World Health Organization 1982. The multidrug regimen in cases of paucibacillary leprosy consists of rifampicin 600mg once per month and dapsone 100mg daily both drugs should be used for six months[6].

REFERENCES:

1- Moubasber AD, Kamel NA, Zedan H et al: Cytokines in leprosy. Int- J - Dermatol 1998; 37: 733-40.

2- Fitzpatric TB: Dermatology in general medicine 4th ed. 1993; 2395-2409.

3- Ross M, Barr RG: Leprosy in the united states: Just a curiosity?Int- J- Dermatol 1991; 30: 772-3.

4- Mahaisavariya P, Kulthanan K, Khemngern S et al: Lesional T-cell subset in leprosy and leprosy reaction. Int- J - Dermatol 1999; 38: 345-7.

5- Thomas MM, Jacob M, Chandi SM et al: Role of S-100 staining in differentiating leprosy from other granulomatous diseases of the skin. Int- J -Lepr- Other-Mycobact-Dis. 1999; 67: 1-5.

6- David S, Mezabish MC, Josef K. Tuberculoid leprosy: A case report. Cutis 1991; 47: 116-8.