EDITORIAL

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LEISHMANIASIS, A COMPARATIVE STUDY

My sincere thanks to the Editor of our Journal for asking me to contribute an editorial article. I consider it an honour and a privilige too. It is a privilege because the article will be definitely published without any question. I therefore wondered what kind of article I should contribute lest I misuse the privilege extended to me. After considerable thought, I decided to use this editorial space to share my thoughts on Leishmaniasis, a protozoal infection of considerable interest to a dermatologist on account of its similarities with several other skin diseases. An understanding of this disease and a comparative study with few other diseases, where possible, may help us to understand better the underlying disease processes. For example the erythema nodosum that occurs in leprosy has always been considered by many leprologists as reaction to dapsone. This is the result of studying a disease in isolation. Erythema nodosum is seen in several other diseases. In leprosy it is one of its clinical manifestations as in other diseases.

What follows below is neither fiction nor fact but food for thought.

Three cases of Post Kala-azar Dermal Leishmaniasis (PKD) had come under my care, while I was working in Christian Medical College Hospital, Vellore. The three patients had Kala-azar several years before they developed typical cutaneous lesions of PKD, very much like the skin lesions of lepromatous leprosy. L.D. bodies were demonstrated in the skin smears. I treated them with all possible drugs for several months. They never showed any improvement. I lost sight of them finally. One of them returned to me after few years showing good clinical improvement on no treatment. If I happened to be treating him with some exotic drug during this period I would have claimed the success and reported in some journal.

During the past one year I have been working in Tripoli, Libya. 23 cases of cutaneous Leishmaniasis (C. L.) came under my care. If these cases had been seen in other parts of world where C. L. is not endemic, they could have been diagnosed clinically as cutaneous lesions of syphilis, tuberculosis, leprosy, deep mycosis, sarcoidosis etc. The history of these 23 cases of C.L., their onset, duration, clinical features, response to various drugs are all very intriguing. One can ask these questions:

1. Why is the clinical picture of leishmaniasis different in different parts of world, although the etiologic agent seems to be the same?
2. Why is this protozoal infection confined only to skin in some parts of world as C.L.?
3. Why is there such a great variety of skin lesions in C.L.?
4. What is the natural course of C.L?
5. What is the best treatment for C.L?

Treponematosis is another disease which behaves like leishmaniasis in certain respects. Its clinical picture is different in different parts of world as syphilis, yaws, pinta and so on. Some types of treponematosis are confined

only to certain systems of body. In leprosy too certain features are suggestive of leishmaniasis.

The answers to the above questions are perhaps related to the host-parasite-environment complex.

There are three distinct types of cutaneous manifestations in leishmaniasis, the mechanisms involved in each type being different.

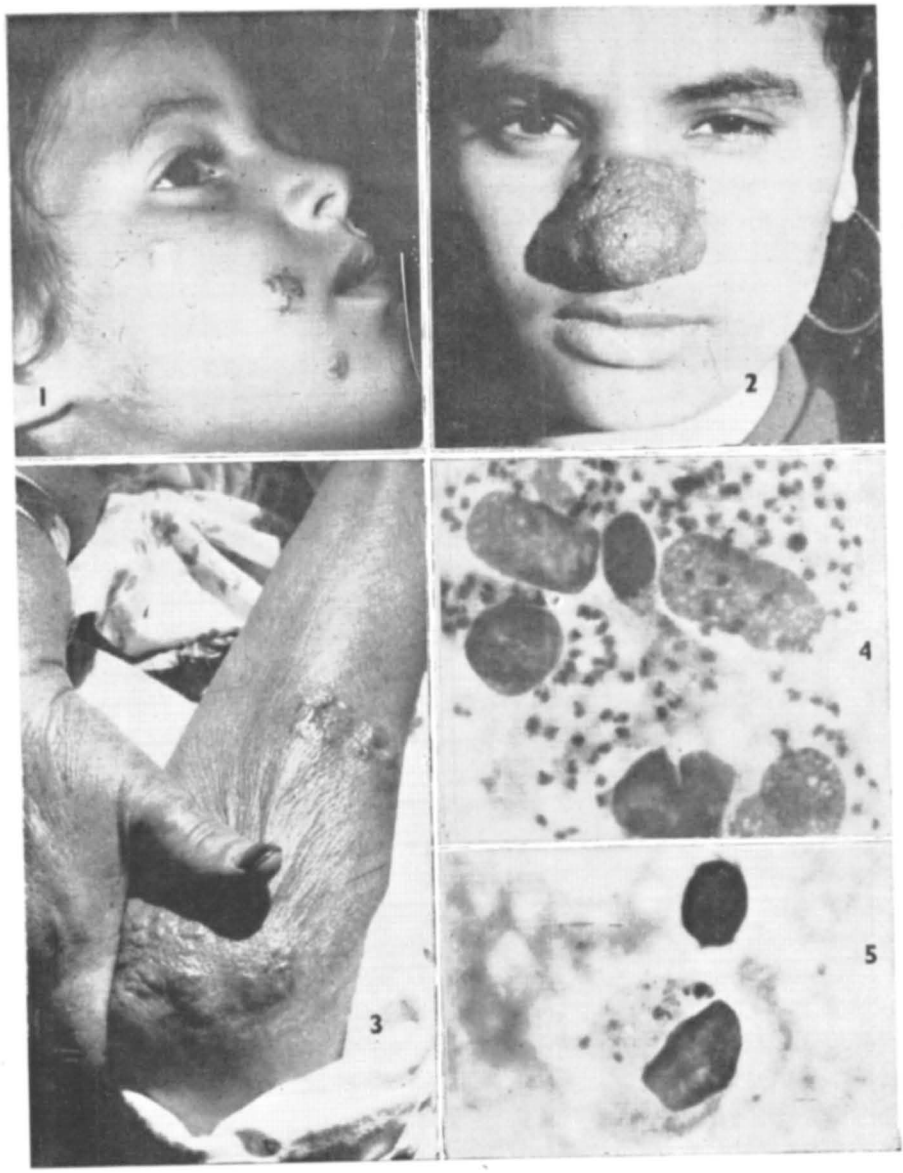
1. Cutaneous leishmaniasis (CL): In this type the disease is strictly confined to skin. The lesions start at the sites of sandfly bites. They develop into several clinical varieties. This is described as old world type (Delhi boil, Aleppo boil etc)
2. Muco - cutaneous leishmaniasis (MCL). The disease starts with skin lesions as above. Muco-cutaneous lesions develop much later as a metastatic disease. This is described as the new world type (Espundia).
3. Post Kala-azar dermal leishmaniasis (PKDL). The skin lesions appear years after initial episode of Kala-azar.

In the first type, the patients develop cell-mediated type of hypersensitivity very early in the infection with the result that the infection is localised to the sites of sandfly bites and does not become systemic. This cell mediated immunity demonstrated by intradermal testing by Leishmanin (Montenegro test). Figs. 1-3 show typical lesions of C.L. proved by demonstration of L.D. bodies in smears from the lesions. Fig. 1 shows three lesions on the face, of three months' duration. The largest lesion is typical of Delhi boil. The other two lesions seem to be abortive sandfly bites. Perhaps the same sandfly has caused all the three bites but only the first bite with bigger inoculum has developed into the active lesion showing numerous L.D. bodies (Fig. 4). The lesion on the nose Fig. 2 is clinically a verrucous plaque of 6 months' duration. Smear from this lesion shows several disintegrating organisms. Fig. 3 shows multiple lesions on arm and hand of more than 1 year duration. The organisms are few and far between in these lesions. Fig. 5 shows two L. D. bodies inside the pathognomonic large monocyte. It is difficult to see whether these multiple lesions are relapses or reinfection or primary lesions. Some of these lesions, particularly the chronic types show tuberculoid granulomatous reactions histologically suggestive of cell mediated delayed hypersensitivity. This type of leishmaniasis seems to behave like tuberculoid leprosy.

In the second type, although there is cell mediated type of immunity in the early stage of the infection, as in the first type the immunity perhaps does not last too long. With the failure of immunity the disease spreads to mucous membranes from the previous skin sites. The behaviour of immunity and the changing clinical picture are suggestive of dimorphous leprosy.

In the third type there is total lack of cell mediated immunity at the onset of the infection and therefore starts as a systemic disease, kala-azar, as it happens in lepromatous leprosy. After several years when the disease abates some patients begin to develop cell mediated immunity. PKDL is perhaps a relapse of the disease during this immunologic status.

Cell mediated type of immunity is influenced by the racial and ethnic back-ground of the host, antigenicity of the bacterium, and endemicity of the disease. Clinical pattern of the disease in the host is influenced by the degree of cell mediated immunity and when the host develops or loses it during the course of the disease. With all these variable factors the clinical pattern of leishmaniasis and such other diseases, becomes very intriguing.



Three out of the 23 cases of C.L. did not receive any treatment, out of default. However, the lesions spontaneously disappeared. This aspect of spontaneous cure and remission in some disease must be recognised as a natural course of the disease before advocating magic cures with drugs which do more harm than good to the patient in the long run of the disease.

Tuberculosis and certain deep mycoses also share some similarities with leishmaniasis. Dermatologists, who have the advantage of seeing several of these diseases have excellent opportunities for comparative study of them develop broader concepts and avoid disease-oriented classifications and terminologies like ENL, histoid leprosy, etc.

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