REACTIONS IN LEPROSY

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Several acute clinical manifestations occuring at any stage in any form of leprosy have been described by various authors as "reactions". The mechanisms underlying these various types of acute episodes are different. Therefore it will be confusing to group all the acute manifestations into one and call it "reactions in leprosy"—Each of these "reactions" should be viewed in the light of the circumstances under which it happens and the form of leprosy in which it occurs. Specific nomenclature, explanatory of the underlying mechanisms, should be given to various types of acute manifestations to make them meaningful.

The leprosy "reaction" which we particularly wish to discuss in this paper is a distinct clinical entity which occurs in leprosy patients who are bacteriologically positive. The four cardinal symptoms associated with this condition are erythematous painful skin nodules, fever, arthralgia and lymphadenopathy. Less commonly, neuritis, iridocyclitis, orchitis and myositis are seen. Although the majority of patients have more than one symptom, only one of the above symptoms may be the sole manifestation of this reaction. Murata in 1912 coined the term Erythema Nodosum Leprosum to this condition probably because the most frequent and more specific of the manifestations is the erythematous skin nodules. Hereafter in this paper this reaction will be referred to as ENL.

Misconceptions regarding the cause and outcome of ENLs are very widely prevalent among physicians who treat leprosy. It is our intention to review some of these widely prevalent views about ENL and their management and re-examine the rationale and basis for some of the views currently held.

It is commonly believed that ENL reactions are precipitated or worsened by sulfones and related drugs. As a consequence it is wide practice to discontinue anti-leprosy drugs particularly sulfone as one of the major measures to combat these reactions. Many people also believe that reactions may be prevented by using small doses of sulfones. It is common habit to treat chronic and severe ENL reactions with steroids. Almost all patients, who suffer from acute neuritis, even when that neuritis is part of an ENL, are given cortisone or its derivatives. ENL is even considered beneficial for the disease.

Before we discuss the above views regarding ENL, it is important to have a clear knowledge of the normal pattern of ENL without which we are likely to misjudge and misinterpret the cause and course of this condition as well as its response to treatment. ENL differs widely in different patients in its severity, duration and frequency. It starts irrespective of the duration or the bacteriologic positivity (bacteric index) in the patients. For purposes of description, the clinical pattern of an

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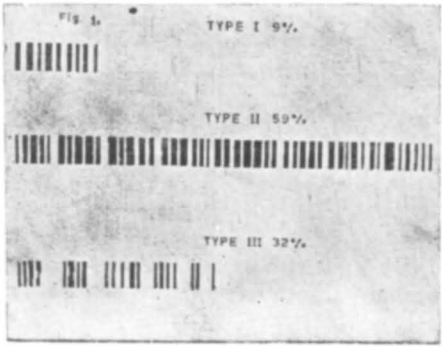
untreated ENL may be described in terms of "episode" and 'phase'. An episode is that period of the reaction which begins with the first sign and ends with the cessation of the last symptom or sign. Phase refers to the whole period during which a patient may suffer from few to several episodes.

In terms of reaction pattern, patients can be divided broadly into three types (Fig.) Type I, have relatively short



reaction phase; one year or less, with few short episodes of ENL. Type II have long reaction phase often going on to several years. Their episodes occur very frequently. Many patients in this type are often seen in a continuous state of reaction. Type III have reaction pattern, neither of the short phase of Type I nor the long phase of Type II. Their episodes of ENL come at intervals of two, three or four months, each episode lasting only about one or two weeks.

Following is the breakdown of our patients with ENL into three Types:

Type I	9%
Type II	59%
Type III	32%

Reliable and careful history of the patients with ENL enables us to make the following observations.

These patients could be put into three groups with respect to the onset of ENL.

Group I Patients who were on antileprosy drugs when they first developed ENL (39%)

Group II Patients who had been receiving antileprosy drugs but stopped these at least six months prior to the onset of ENL (49%)

Group III Patients who never received any antileprosy drugs but developed ENL (12%)

Patients in Group I developed ENL while they were receiving antileprosy drugs, for variable periods of time. But once they developed ENL, the antileprosy drugs were discontinued on the assumption that they precipitated the ENL. However, they continued to develop ENL despite the discontinuation of the drugs for long periods of time. Apparently these patients belonged to type II reaction pattern.

The patients in Group II were diagnosed and treated for leprosy for variable periods of time before their first episode of ENL. Most of these patients discontinued their treatment at least six months before the onset of ENL either because of their casual attitude towards the disease or inability to get medical help on long term basis. They sought for medical help again only when they developed ENL.

The patients in Group III were diagnosed as cases of leprosy when they developed ENL as the first clinical manifestation of the disease and as such were never exposed to any antileprosy drugs.

Group II and III together constitute 61%; that is, nearly two thirds of the patients. In these patients the onset of ENL cannot be attributed to the antileprosy drugs.

In the light of the above observations, it is difficult to incriminate antileprosy drugs in the pathogenesis of ENL.

We wish to share some of our experience in treating patients suffering from leprosy with or without ENL. All patients diagnosed as having leprosy are started on Dapsone 50 mg. daily irrespective of the classification or the complications they may present with. Even patients with acute neuritis are given 350 mg. of dapsone per week complementary to any other form of treatment which is deemed necessary for the neuritis itself. Patients who present with ENLs are given along with DDS, other symptomatic treatment. Our mainstay measures for combating the ENL are Potassium Antimony Tartrate or Chloroquin. E. M. G. and nerve conduction studies done on our patients who are on dapsone during acute neuritic episodes have shown that DDS does not interfere with recovery of the nerve from its inflammatory state.

Petit and Waters in 1967 had shown convincing evidence that incrimination of sulfone drugs as the causative agent of ENL is untenable. We believe that the etio-pathogenesis of this condition is complex and not well understood. It is probably an allergic response to the broken end-products of lepra bacilli. This type of allergic response is also seen in other chronic infections like tuberculosis known as 'ids'. It is therefore appropriate to call ENL as "Leprids", to fall in line with all other 'id' reactions. The several methods of management adopted for this complication are all primarily intended towards symptomatic relief. In whatever way this is accomplished we should have the specific understanding that we are treating leprosy and one of its complicating features and not the complication alone. At no cost shall we cause harm to a patient in a desire to give immediate temporary relief to the ENL which in all cases will eventually subside spontaneously. The pattern of ENL is not easily altered by whatever method adopted to treat the reaction.

Summary

ENL is neither precipitated nor worsened by antileprosy drugs. It is one of the complications of bacteriologically positive cases of leprosy. This complication should be treated symptomatically while the underlying disease is treated with Dapsone which is still the drug of choice for its treatment. Steroids should be scrupulously avoided in the management of ENL. ENL represents an 'id' reaction in bacteriologically positive cases of leprosy and the suggested appropriate nomenclature is 'Leprid'.

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