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A current perspective on leprosy (Hansen's disease)

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Abstract
Leprosy is a chronic granulomatous disease, which affects dermis and peripheral nerves and also can involve the eye, the mucosa of the upper respiratory tract, muscle, bone, and testes, caused by the intracellular pathogen *M.leprae*. Leprosy is not highly contagious. It is challenging to spread by human-to human contact unless there is consistently close–living proximity with an infected individual. The skin is not important in leprosy transmission. Bacilli are not excreted by the skin and are rarely found in the epidermis. The various clinical manifestations of leprosy are not due to different strains of *M. leprae* but are rather the results of the variations in the host tissue response to the bacilli in the body. There are two classification Ridley–Jopling and WHO classification. The WHO classification is useful for allocating patients to treatment groups and should be used in peripheral centers where skin smears and histopathology are not available. Leprosy causes nerve damage and permanent disabilities including blindness and paralysis. People affected by leprosy face stigma and discrimination in society. Although multidrug therapy is available, millions of people are still affected by leprosy, so new vaccine, drug and disease management approaches are urgently needed for control, prevention and treatment of this disease.

Keywords: Leprosy, peripheral nerves, *M. leprae*

Introduction
Leprosy, also known as Hansen's disease, is an ancient chronic human infectious disease that remains a major public health problem in many developing countries. Leprosy is caused by the pathogen *Mycobacterium leprae*, first discovered over a century ago by the Norwegian scientist Gerhard-Henrik Armauer Hansen. *M. leprae* is a slow-growing mycobacterium and an obligate intracellular pathogen, which can survive out of the human host for up to 45 days [1].

In Egypt, the prevalence of leprosy was estimated by 10 per 10000 in 1969.The number of cases registered for treatment up to the end of 1969 was 28197. After 30 years (1999) their number reduced to 3020 with a prevalence of 0.5 per 10 000 population. In the year 2000, a further drop in prevalence occurred to reach 0.49/10 000 at the national level [2].

Disease Transmission and Infection
At the first International Congress of Leprosy held in Berlin (1897), Schäffer proved that the infection could spread through nasal discharge. Shepard [3] proved once again that lesion in the nasal mucosa could lead to the discharge of 10,000 to 10,000,000 bacilli and Pedley estimated that tens of millions bacilli could be discharged from the nasal mucosa on a daily basis [4]. In 2013, *M. leprae* was identified in the buccal mucosa of 94% of patients presenting with MB and PB leprosy (PCR analysis and antigenic markers) [5]. Leprosy is not highly contagious. It is challenging to spread by human-to human contact unless there is consistently close–living proximity with an infected individual [6]. Figure 1.

M. leprae is inhaled, multiplies on the inferior turbinates and has a brief bacteraemic phase before binding to Schwann cells and macrophages [7]. The skin is not important in leprosy transmission. Bacilli are not excreted by the skin and are rarely found in the epidermis. The only evidence of bacilli entering via the skin comes from case reports of direct inoculation. Among other sources, it is known that *M. leprae* is also present in breast milk. It has been calculated that a child breastfed by a lepromatous mother can receive up to two million bacilli from a single suckle. The epidemiological significance of this source is not known [8].

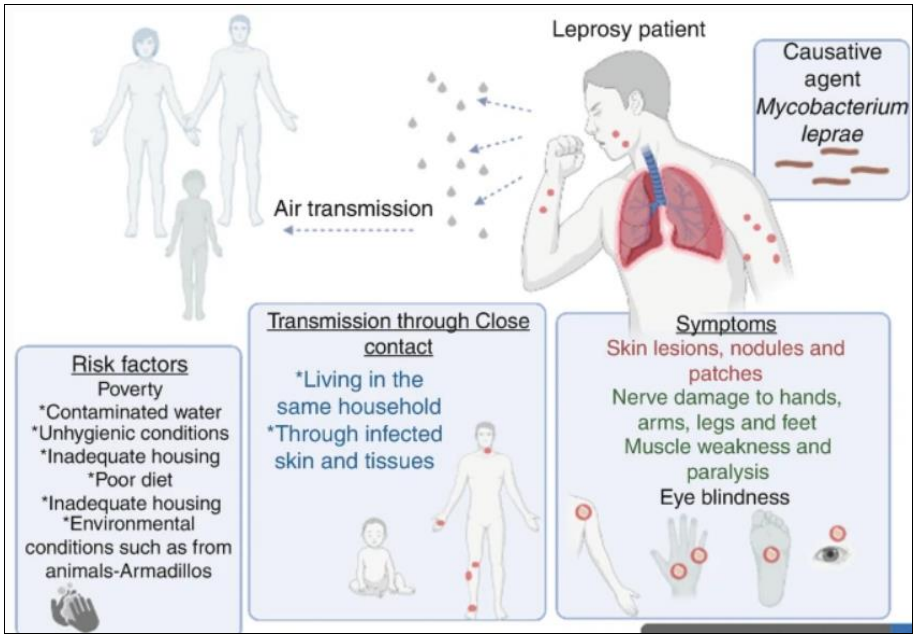

Fig 1: Overview of leprosy transmission routes, infection and symptoms. *Mycobacterium leprae* is the causative agent of leprosy. The symptoms are nodules, lumps, bumps, lesions and patches on the skin, blindness, nerve damage, muscle weakness and paralysis to the hands, arms, legs and feet. The main route is transmission through air droplets from infected individuals and through contact with infected skin and tissues. Transmission can occur through close contact, such as living with a leprosy patient in the same household. The main factors are poverty, inadequate housing and unhygienic conditions, poor diet and contaminated water [1]

Leprosy Classification

The various clinical manifestations of leprosy are not due to different strains of *M. leprae* but are rather the results of the variations in the host tissue response to the bacilli in the body [9].

Ridley-Jopling classification

The classification consists of five categories based on the bacterial load as well as the clinical, histological, and immunological features in skin biopsies: TT, with the highest cellular response (type II interferon IFN-γ, TNFα, and IL-15) and a restricted growth of bacteria, followed by BT, BB, BL, and LL, characterized by an increased but inefficient humoral response (IL-4 and IL-10) leading to the survival of bacteria in the host [10].

WHO classification

The World Health Organization further simplified this classification into paucibacillary (having five or fewer skin lesions) and multibacillary (having six or more skin lesions). Disease states roughly correlate to the effectiveness of cellular immunity and corresponding bacterial load to simplify and standardize clinical diagnosis and operational treatment regimens globally [10].

Pathophysiology

Once *M. leprae* is inside the subject, it enters lymph and blood vessels to reach its target: the Schwann cells (SCS). Binding of *M. leprae* to Schwann cells induces demyelination and loss of axonal conductance. It has been shown that *M. leprae* can invade schwann cells by a specific laminin binding protein of 21 kDa in addition to Phenolic glycolipid -1. Phenolic glycolipid -1, a major unique glycoconjugate on the *M. leprae* surface, binds laminin-2, which explains the predilection of the bacterium for peripheral nerves [1].

Schwann cells engulf *M. leprae* within their phagosomes but cannot destroy *M. leprae* because Schwann cells lack lysosomal enzymes. Schwann cells are sanctuaries where the bacilli are protected from macrophages and can replicate slowly over years. The leprosy bacillus is dependent on host metabolic products, which could explain its long generation time and inability to grow in culture [10].

Host genetic factors influence the CMI and have a partial effect on both the development of leprosy and the pattern of disease. The CMI determines either the elimination of the bacillus or the development of the disease. In fact, at some stage infected Schwann cells process and present antigenic determinants of *M. leprae* to antigen-specific T lymphocytes that initiate a chronic inflammatory granulomatous reaction. *M. leprae* may migrate outside the nerves to endothelial cells or may be phagocyted by macrophages that act as antigen-presenting cells [10].

At this exact point, the CMI plays a pivotal role. Subjects with a predominant Th1 immune response will develop a high degree of CMI with epithelioid granuloma formation that will destroy all the bacilli with either healing or development of localized disease, tuberculoid leprosy (TT) [11]. On the contrary, individuals with a predominant Th2 response will develop a weak CMI without forming an efficacious granulomatous response and an increased humoral immunity: bacilli will survive and replicate, developing systemic disease, lepromatous leprosy (LL) [12].

Immunopathology

Innate immune response

The defense against pathogenic agents is mounted by the innate immune response, followed by the acquired immune response, both types of response act via the cells and soluble factors. *M. leprae* can enter and take up residence within macrophages and Schwann cells according to multiple modes [13]. Receptors to complement fragments of CR1, CR3 and CR4 aid phagocytosis. Phenolic glycolipid I is recognized by complement 3. As well as the complement

receptors, the Toll-like receptors (TLRs) present on the macrophages are also important in the recognition of microbial pathogens. TLR2 and TLR4 recognize the leprosy bacillus, activate monocytes and release IL-12, a cytokine that induces pro-inflammatory cytokines and killing of the bacilli [14].

Acquired immune response

The acquired immune response is triggered by dendritic cells, potent antigen-presenting cells that act as a bridge between the two arms, innate and acquired, of the immune response. Dendritic cells migrate from the site of infection and present the antigen to naïve T cells in the regional lymph nodes. Depending on their degree of maturity and signaling, dendritic cells can stimulate naïve T cells to differentiate into different effector subpopulations [15] S.

Clinical features

Indeterminate leprosy

This is the early phase of the infection, usually seen in children. It usually presents with a single hypopigmented patch, particularly in dark skinned patients, with an ill-defined margin (Figure 2) [16].

Fig 2: Indeterminate leprosy [16]

Tuberculoid type leprosy

This type involves the skin and peripheral nerves only. There is usually a single elevated annular plaque or a few asymmetric macules or flat patches, sometimes showing a prominent border. The center may be erythematous in light skin or hypo pigmented in dark skinned individuals the scalp, axillae, and inguinal areas are usually spared (Figure 3) [17].

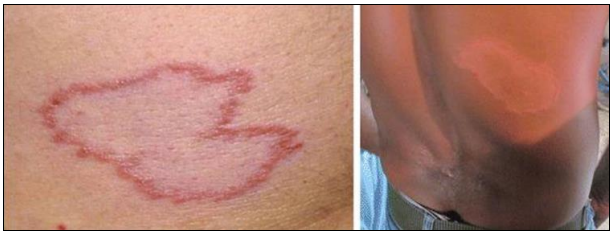

Fig 3: Skin lesions in tuberculoid leprosy [17]

Borderline tuberculoid leprosy

Likewise, in TT type, the lesions have a well demarcated border and hypopigmented center. There is a possibility for erythematous appearance in fair skin. Small satellite lesions may be seen at the periphery, revealing a symmetric pattern of distribution (Figure 4) [18].

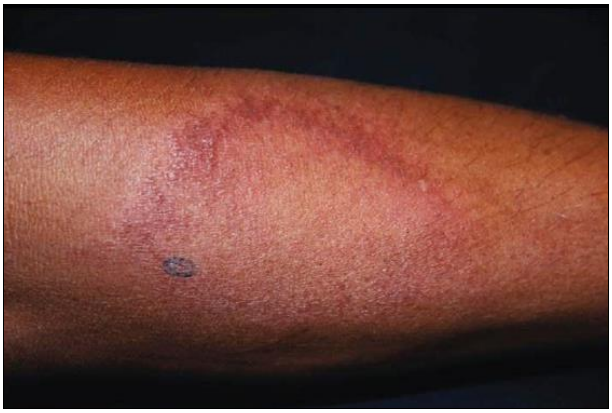

Fig 4: Borderline tuberculoid leprosy [18]

Borderline borderline leprosy

This form is usually unstable, features of both tuberculoid and lepromatous leprosy is present. Without treatment, borderline leprosy may become less severe and more like the tuberculoid form or it may worsen and become more like the lepromatous form [19]. (Figure 5).

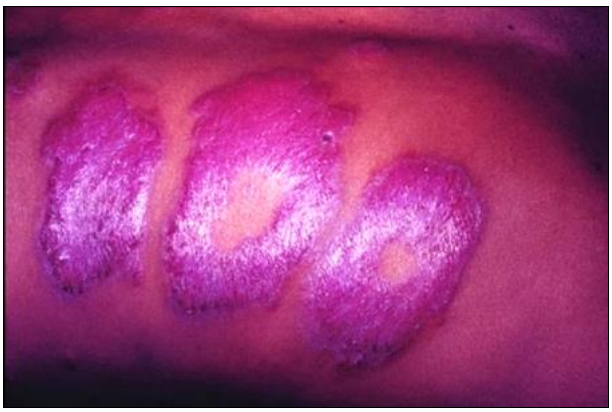

Fig 5: Erythematous plaques of mid-borderline leprosy [19]

Borderline lepromatous leprosy

Involvement of the skin is in this type diffuse, multiple, and almost symmetric. Fade macules are followed by papules, plaques, and nodules, often with ill-defined borders (Figure 6) and asymptomatic symmetric enlargement of large peripheral nerves. The incidence of immunological reactions, both types 1 and 2, is high in BL leprosy [20].

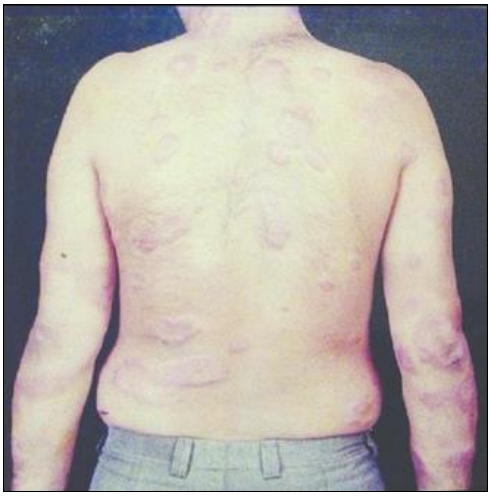

Fig 6: Borderline lepromatous leprosy: Multiple erythematous annular plaques [20]

Lepromatous leprosy

Widespread and symmetric papules, nodules, and plaques are seen, but the scalp, axillae, and groins are spared. Diffuse infiltration of bacillus in the skin leads to multiple lesions and in some cases to the typical and characteristic feature of leonine facies. Saddle nose deformity, destruction of nasal bridge and epistaxis may occur due to heavy infiltration of the agent. Madarosis, lagophthalmos due to facial nerve paralysis, and ichthyosis- like xerosis are also features in late stage. The auricle may be infiltrated, enlarged, and swollen (Figure 7) [17].

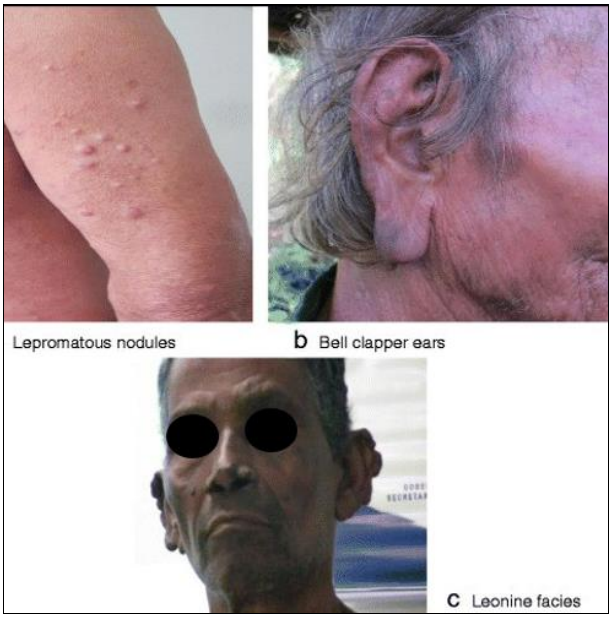

Fig 7: Lepromatous leprosy (LL). A) Lepromatous nodules. B) Bell clapper ears C) Leonine facies [17]

Histoid Leprosy

It manifests as numerous cutaneous nodules and plaques primarily over the back, buttocks, face, and bony prominences [21] (Figure 9).

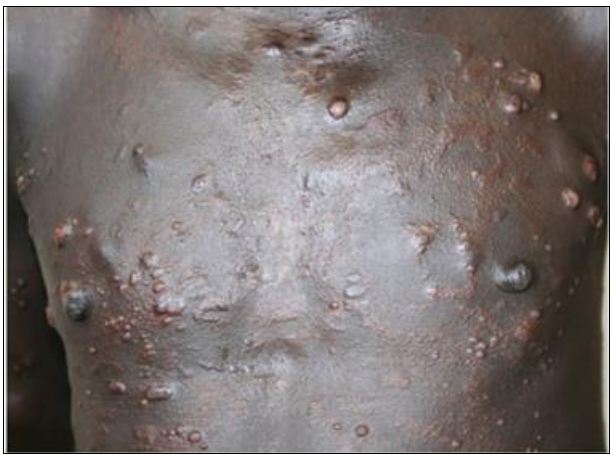

Fig 8: Histoid leprosy [21]

Pure neuritic leprosy

It is a type manifesting with only neural signs without any evidence of skin lesions. The affected nerves are thickened, tender, or both. It accounts for around 4–6% of leprosy cases. Commonly affected nerves are ulnar, median, radial, lateral popliteal, posterior tibial, sural, facial, and sometimes trigeminal [22].

Immunological reactions in Susceptibility to Leprosy

Type 1 Reaction (reversal reaction)

It can occur in borderline leprosy patients before, during, or even after treatment due to changes of the cell mediated immune response, considered as type IV hypersensitivity reaction. Patients develop acute symptoms of inflammation in the skin and peripheral nerves. On examination, pain, swelling, and erythema in cutaneous lesions and/ or in nerve fibers occur [23].

Type 2 Reaction (erythema nodosum leprosum)

It is due to the formation and deposition of antigen antibody complexes (type III hypersensitivity) and occur in around 50% of the patients with BL and LL types of the disease, particularly after the onset of oral medication. It is a multi-systemic process representing leucocytoclastic vasculitis. ENL is a cutanous manifestation of type 2 reaction, consisting of multiple, painful, subcutanous nodules. Unlike ordinary erythema nodosum, the nodules in ENL last shorter and involve the upper limbs, face, and trunk, in addition to the lower limbs (Figure 9) [24].

The immune reactions in patients with leprosy are significant and considered as an emergency condition to be instantly diagnosed and treated. If they remain untreated, persistent complications and disabilities may follow [25].

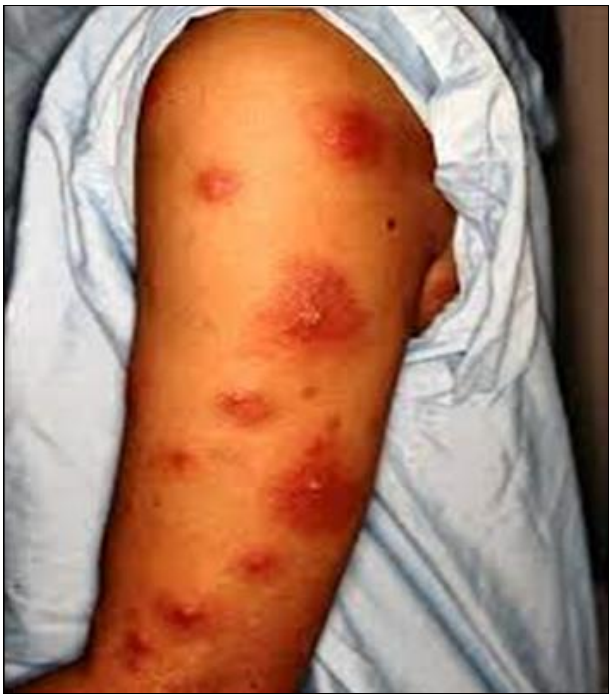

Fig 9: Lesions of erythema nodosum leprosum [24]

Diagnosis of leprosy

Clinical diagnosis

Leprosy is considered a clinical diagnosis. Manifestations suspecting leprosy include hypopigmented or reddish skin lesions with loss of sensation and involvement of the peripheral nerves as demonstrated by their thickening and associated loss of sensation [26].

Laboratory diagnosis

Slit skin smear: Under the 100-x oil immersion lens in a smear made by nicking the skin with a sharp scalpel and scraping it; the fluid and tissue obtained are spread fairly thickly on a slide and stained by the Ziehl-Neelsen (Z-N)

method, as shown in (Figure 10**)** which demonstrated *M. leprae* with modified Z-N stain [27].

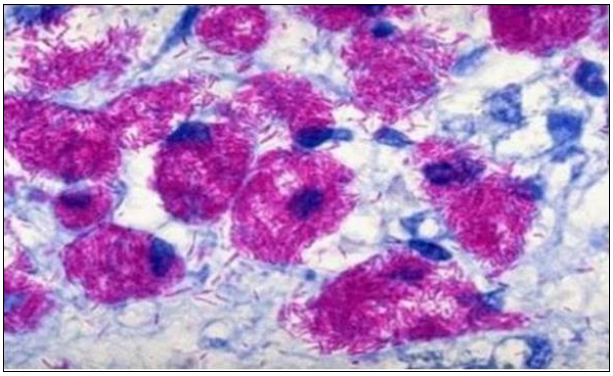

Fig 10: *M. leprae* with modified Z-N stain [27]

Skin/Nerve biopsies: Skin biopsies are usually performed when there is doubt about the diagnosis and remain the golden standard. Biopsies should be taken from an active site, i.e. red, enlarged and infiltrated. A modified Ziehl-Neelsen stain, such as the Wade Fite stain is preferable for the histological diagnosis [28].

Skin tests

The lepromin test: Is a non-specific (not diagnostic) test. 0.1 ml of lepromin is injected intradermally, and the site is examined after 48 hours "Fernandez reaction" or 3- 4 weeks "Mitsuda reaction"; A positive Fernandez reaction indicates delayed hypersensitivity to antigens of *M. leprae* or a cross reacting mycobacterium [27].

Tuberculin skin tests (TST): Do not significantly cross-react with *M. leprae* infection; in one study of a population in which tuberculosis was highly endemic, 70% of controls had positive TST, but only 15–50% of leprosy patients had positive TST [29].

Histamine test: Detects the damage done to dermal nerves in leprosy. A drop of sulphate of histamine (1: 1000) is applied to a hypochromic macule and another healthy area elsewhere as control. In leprosy, especially in tuberculoid, there is no flare; in borderline and intermediate, the flare is weak and develops late [30].

Pilocarpine test: Is done by applying tincture of iodine to the suspected lesion and normal skin as a control prior to injection of pilocarpine into these sites. Then these areas are dusted with a starch powder that will turn blue if there is normal sweating [28].

Immunohistochemistry

Using monoclonal or polyclonal antibodies to detect *M. leprae* antigens may provide higher sensitivity and specificity than conventional methods, especially in the initial stage of illness or in PB cases. The antibodies against PGL-1, S-100 protein, and bacillus Calmette-Guerin (BCG) are used to demonstrate *M. leprae* in the tissues [28].

Serology and Polymerase Chain Reaction: Serologic assays can be used to detect phenolic glycolipid-1 (specific for M leprae). This is a specific serologic test based on the detection of antibodies to phenolic glycolipid 1. This test yields a sensitivity of 95% for the detection of lepromatous leprosy but only 30% for tuberculoid leprosy [31].

Evaluation of nerve damage: Electrophysiology (EPG) of the peripheral nerves especially the nerve conduction studies with or without sympathetic skin response is a sensitive tool for the detection of the earliest alterations of sensory fibers or autonomic functions, thereby detecting the neuropathy much before the clinical symptoms appear [31].

Treatment of leprosy

The three main objectives in management of leprosy are to interrupt transmission, cure patients, and prevent development of deformities and reactions [32].

Leprosy prevention: Program for leprosy prevention includes.

- Public education and awareness encourage individuals with leprosy.
- Household contacts of patients with leprosy should be monitored closely for the development of leprosy signs and symptoms.
- Prophylaxis with a single dose of rifampicin helps in preventing leprosy for about two years in individuals who have close contact with leprosy patients [33].

Curative treatment and drugs

1. **Dapsone (4, 4-diaminodiphenylsulfone) monotherapy**
2. **Multidrug therapy**
3. **Multibacillary MDT:** This is recommended for adult patients with mid-borderline, BL, LL, smear-positive BT and PN leprosy [Rifampicin, 600 mg once a month, supervised administration. Dapsone, 100 mg/day, self-administered. Clofazimine, 300 mg once a month, supervised administration; 50 mg/day, self-administered] [34].

Treatment of leprosy reactions and neuritis

Usually, these reactions respond satisfactorily to prednisolone along with thalidomide or clofazimine. High-risk patients may require corticosteroids for 6 months with MDT as a preventative measure [35].

Treatment of type 1 reaction

The mainstay of type 1 reaction treatment continues to be corticosteroids. Long term low-dose therapy was observed to be more useful than high dose. Surgical decompression of the swollen nerve should be done if the medical therapy has not been successful [36].

Treatment of type 2 reaction

The primary drug of therapy of type 2 reaction is with analgesics and corticosteroids. In patients who don't respond to corticosteroid therapy, Clofazimine has a useful antiinflammatory effect in ENL and can be used at 300 mg /day for several months. Low grade chronic ENL, with iritis or neuritis, will require long-term suppression, preferably with thalidomide or clofazimine. Pentoxifylline amethyl xanthine derivative has been used in one study but found to be less effective in control of type 2 reaction [37].

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