Leprosy major disease suffered by people all around the world.

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Mycobacterium leprae is the chronic infectious illness that causes leprosy. Despite being highly contagious, it has a low morbidity because a sizable section of the populace is naturally resistant to it. Peripheral nerves and the skin are primarily impacted by leprosy. Based on a patient's skin and neurological evaluation, the condition is diagnosed. A prompt diagnosis is crucial. The prompt and effective use of treatment will prevent physical limitations and sequelae that have an influence on the person's social and professional life and are also to blame for the stigma and prejudice surrounding this condition.

Leprosy has been the name for this illness since the time of the Bible, when descriptions of cases date back more than three thousand years. Whether leprosy originated in Asia or Africa is up for debate. The term "leprosy" honours the Norwegian doctor Gerhard Armauer Hansen, who discovered in 1873 that the disease was caused by the bacteria Mycobacterium leprae [1].

Epidemiology

Tropical nations, particularly those that are developing or impoverished, are endemic for leprosy. Since the beginning of the 1980s, when MDT was introduced, its prevalence has significantly dropped. The majority of infections are still concentrated in 105 endemic nations, particularly those in Southeast Asia, the Americas, Africa, the Eastern Pacific, and the Western Mediterranean.

Epidemiological data from some nations, including India, should be interpreted cautiously because the goals of disease elimination were met based on certain criteria, including: modifications to the definition of case, exclusion of recurrent cases from the prevalence rate, exclusion of cases of treatment dropout from active records, single-dose treatment of paucibacillary (PB) patients, shorter treatment duration, etc. Epidemiological data should also be interpreted with caution because it is possible that some countries' data may not be accurate. As a result, there were significantly fewer newly reported instances [2].

Mechanisms of leprosy transmission

Leprosy is thought to spread through continuous close contact between a vulnerable person and a patient who has the bacillus through inhalation of the bacilli present in nasal secretions or Flugge droplets. The nasal mucosa is the primary route of transmission. Skin erosions are a less common means of transfer. There are more channels of transmission that can occur, including blood, vertical transmission, breast milk, and insect bites.

It's anticipated that infected people, even those who didn't get sick, might have a temporary nasal release of bacilli. The presence of particular DNA sequences from *M. leprae* in nasal biopsies or swabs, as well as seropositivity for those antigens in healthy people residing in endemic areas, indicating that the carrier may be involved in the transmission of the disease [3].

Genetic Factors

Although the precise genes responsible for leprosy are unknown, it is believed that certain HLA and non-HLA gene sets have an effect on a person's vulnerability to the disease, both in terms of preventing infection in the first place and defining the clinical presentation. Currently being researched are changes in candidate genes, or genes whose product contributes to the host's response to the infectious agent. Leprosy binding peaks have been discovered by genomic scan studies in chromosome areas 6p21, 17q22, 20p13, and 10p13.

Immunopathology

The ability of the host to produce various levels of cellular immunological response to *M. leprae* results in a vast diversity of clinical and histological symptoms of leprosy, which gave rise to the spectrum idea of the illness. The integrity of epithelia, secretions, and surface immunoglobulin A serve as the initial line of defence against infection with *M. leprae* (IgA). In addition, regardless of the activation of adaptive immunity, Natural Killer (NK) cells, cytotoxic T lymphocytes, and activated macrophages can all kill bacteria. The poor virulence of *M. leprae* and an efficient innate immune response mediated by dendritic antigen-presenting cells may be the basis for resistance to the emergence of leprosy's clinical symptoms [4].

The host immune response is still indefinite in the initial stage after the virus has been established. T helper 1 (Th1) or T helper 2 (Th2) lymphocyte proliferations may be influenced by the regulation of inflammatory cytokines and chemokines, promoting cellular or humoral immune responses to *M. leprae*, respectively. This will determine whether the illness progresses to the lepromatous or tuberculoid form.

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TNF- levels are higher in tuberculoid patients' serum, indicating that the presence of this cytokine is linked to the death of *M. leprae* and the development of granulomas. TNF leads to tissue damage and erythema nodosum leprosum symptoms despite being involved in defence through macrophage activation if produced at high levels and linked with high levels of IFN (ENL).

Clinical Manifestations

Characteristics of clinical forms

Clinical symptoms are more influenced by the host's cellular immunological response to *M. leprae* than by the bacillary's capacity for penetration and growth. A lengthy incubation period, lasting anywhere between six months and 20 years, precedes clinical signs (mean period of two to four years). Nine years prior to the clinical diagnosis, seropositivity to *M. leprae* antigens was discovered. Leprosy's prolonged incubation periods may be explained by *M. leprae's* slow proliferation, low antigenicity, and metabolic limitations.

Because there is no cellular immune response to the bacillus in the LL form, *M. leprae* multiplies and spreads through the blood. Although antibodies are made, bacterial growth is not stopped by them. The majority of skin lesions are numerous, symmetrical, and preferentially found on the body's cooler regions. They are distinguished by hypochromic, erythematous, or bright brownish spots with ill-defined boundaries. There may not be a loss of sensation in certain areas. Sometimes, dry skin is the sole observable symptom.

Reactional states

Changes in the immunological balance between the host and *M. leprae* cause leprosy symptoms. These reactions are sudden, intense events that primarily harm the skin and nerves, leading to morbidity and neurological impairment. They can happen before, during, or after therapy. They can also happen while the disease progresses naturally. Type 1 response and type 2 reaction are the two categories under which they fall.

Delayed hypersensitivity is the cause of type 1 reaction, which happens in borderline patients. The disease may become better (reversal reaction, pseudo-exacerbation reaction, or ascending reaction) or get worse (degradation reaction, or descending reaction) as a result of these reactions, which are connected to the cellular immune response to mycobacterial antigens. Borderline patients receiving therapy go to the TT pole of the spectrum as the bacterial burden decreases. Due to the decline in cellular immunity, untreated individuals exhibit increasing bacterial load and clinical manifestations that resemble those of the LL pole. These people are considered to be subpolar lepromatous. Hyperesthesia, erythema, and edoema are the hallmarks of the lesions in both situations, followed by scaling and occasionally ulceration.

Type 2 reaction, also known as ENL, is associated with humoral immunity and does not indicate improved immunity. The deposition of immune complexes in the tissues is thought to indicate the body's response to chemicals released by the killed bacilli. It shows up as a sudden deterioration, particularly during therapy in LL patients, and less frequently in BL patients. Any area may experience symmetrically distributed subcutaneous inflammatory nodules or target lesions of erythema multiforme.

Differential Diagnosis

Among other things, granuloma annulare, figurative erythema, infectious sarcoid lesions or sarcoidosis, pityriasis rosea, psoriasis, lupus erythematosus, and medication eruptions might be mistaken for tuberculoid and borderline lesions. In order to separate multibacillary lesions from secondary and tertiary syphilis, diffuse leishmaniasis, neurofibromatosis, xanthomas, lymphomas, and other tumours, the lepromatous form must be recognised from scleroderma, mycosis fungoides, pellagra, asteatosis, ichthyosis, and eczema. Other etiologies should be looked into in cases where ENL or erythema multiforme is the first symptom. The primary neural forms mirror the inflammatory, metabolic, viral, congenital, or genetic disorders, tumours, and traumas that result in mononeuropathy or numerous mononeuropathies [5].

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