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LEPROSY: A REVIEW OF EPIDEMIOLOGY, CLINICAL DIAGNOSIS, AND MANAGEMENT

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1. INTRODUCTION

Leprosy, or Hansen's disease, is a chronic bacterial infection caused by Mycobacterium leprae (M. leprae) infection [1]. M. leprae, the taxonomic order Actinomycetales, family Mycobacteriaceae, is an acid-fast, gram-positive obligate intracellular bacillus that demonstrates tropism for phagocytes in the skin and Schwann cells within peripheral nerves [2]. Although the 9-banded armadillo infects the wild in the southern United States, M leprae grows in the footpads of mice, which is the main method of growing M leprae in laboratories around the world [3]. Leprosy is ubiquitous in tropical countries, particularly underdeveloped and developing countries. In 1990, the World Health Organization (WHO) proposed the global goal of eliminating leprosy by the end of the 20th century [4]. Despite the commitment of governments, researchers, and healthcare workers worldwide, disease control has not yet been achieved. Between 1900 and 2000, although the number of new leprosy cases remained relatively constant or slightly increased owing to intensified case-finding efforts, a significant reduction in the number of registered cases for treatment and prevalence of cases was observed during this period because of the effectiveness of multidrug therapy (MDT) and improvement in the quality of health care in patients with leprosy worldwide [5]. The geographical distribution of new leprosy cases worldwide in 2020 [6]. This indicates that the highest rates for the detection of new cases are reported by countries in the African region (AFR) and Southeast Asia region (SEAR). Of the 127 countries that reported in 2020, India, Brazil, and Indonesia continued to report the highest number of new case.

2. Classification

Table 1 presents a comparison of the proposed leprosy classification. Leprosy was first classified by Rabello, and the characteristics of disease polarity have been established [4, 20]. In 1966, Ridley–Jopling introduced a classification method for clinical leprosy based on the patient's clinical characteristics and immune status [21]. According to this classification system, the disease is divided into two poles and an intermediate state, including polar tuberculoid leprosy (TT) (Figure 2), borderline tuberculoid leprosy (BT), mid-borderline leprosy (BB), borderline lepromatous leprosy (BL), and lepromatous leprosy (LL) (Figure 3) [21–24].

Patients with a strong cell-mediated immune reaction had few lesions with low or undetectable mycobacteria and were classified as having tuberculoid forms, whereas patients anergic to M. leprae had multiple lesions with higher loads of mycobacteria and were classified as having lepromatous forms [21]. Where an affected person falls within the classification model depends on their immune response [22]. Tuberculoid forms show little evidence of M. leprae-speci6c antibodies but a vigorous T helper ())1 cytokine response, whereas lepromatous forms show a)2 cytokine response with markedly high antibody titers but T-cell hypo-response (anergy) [19, 25].

Table 1: Comparison of classifications of leprosy proposed by World Health Organization and Ridley-Jopling.

Classification		Brief description		
WHO (1987)	Ridley-Jopling [21]	Number of skin lesions	Neurological damage	Bacteriology: microscopic criteria
Paucibacillary				
1 skin lesion	Tuberculoid (TT)	Unique and infiltrated lesions	No neurological damage	Negative
2-5 skin lesions	Borderline tuberculoid (BT)	Stasis and hypopigmented lesions few or many lesions of varying size	Little neurological damage little or no neurological damage	Negative negative/1+
Multibacillary				
>5 lesions	Borderline borderline (BB)	Multiple lesions and maculopapular	Late thickening of the nerve	≥2+
	Borderline lepromatous (BL)	Multiple lesions, maculopapular, and nodules	Late thickening of the nerve	≥2+
	Lepromatous (LL)	Multiple lesions, maculopapular, and nodules	Late thickening of the nerve	≥2+

Note. 1+ or 2+: microscopic criteria when acid-fast bacilli were observed using Ziehl–Neelsen stain; negative, no acid-fast bacilli observed; BL, borderline lepromatous; BT, borderline tuberculoid; IND, indeterminate; LL, lepromatous leprosy; TT, tuberculoid leprosy.

3. Leprosy Reactions

Leprosy reactions are caused by an immune response between the host and M. leprae. Leprosy reactions are an important consequence of permanent nerve damage during leprosy [30]. Leprosy reactions include acute/subacute inflamatory processes that mainly involve the skin and nerves and are the primary cause of morbidity and neurological disability.They may occur regularly at any stage of the disease, even without treatment [30]. However, this reaction can also be initiated or aggravated by effective chemotherapy due to the active destruction of bacilli during or after treatment, thereby producing an abundance of antigenic material in the immune system [31, 32]. Leprosy reactions can be subdivided into types 1 and 2.

4. Diagnosis

Clinical evaluation is the first step in the diagnosis of leprosy and is generally sufficient in most cases. However, one of the challenges in diagnosing leprosy s simply to consider this disease in the list of differential diagnoses, particularly in developed countries where leprosy has almost been eradicated or is extremely rare [26]. Obtaining travel or family history (e.g., adopted or immigrated from an endemic area) is important when considering a diagnosis of leprosy. In addition, practical information about the protective measures of the care team (e.g., high index of suspicion and wearing gloves) to prevent transmission should be included. Skin lesions are usually the /rst clinical manifestation observed. If appropriate medical treatment is not received, leprosy may progress to cause permanent damage to the skin, nerves, limbs, and other organs [36]. WHO experts have listed the main diagnostic criteria as follows [22]: (1) a hypopigmented or erythematous skin lesion or reddish skin patch with definite loss of sensation; (2) a thickened or enlarged peripheral nerve with loss of sensation and/or weakness of muscle supplied by the nerve; and (3) a positive acid-fast skin smear or bacilli observed in a skin smear/ biopsy.

5. Treatment

A multidrug therapy regimen has been recommended by the WHO for the treatment of children according to age and the subdivision of these cases into paucibacillary and multibacillary forms [22]. Rifampicin, clofazimine, and dapsone (diaminodiphenyl sulfone) were used as the firstline treatments. Paucibacillary cases were treated for six months with rifampicin, dapsone, and clofazimine. Multibacillary cases were treated with rifampicin, dapsone, and clofazimine for 12 months. All patients received this drug combination monthly, under supervision.

6. Prevention

Prophylactic Immunization aim of prophylactic immunization is to prevent infection, disease progression, or the administration of vaccines before or after exposure. Several vaccines, such as Bacille Calmette–Gu´erin (BCG), LepVax, and Mycobacterium indicus pranii (MIP), have proven effective.

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