Review article on "Leprosy (Hansen's Disease)" and "Indian Medicinal Plants Used In treatment of Leprosy"

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Abstract

Leprosy is a neglected infectious disease caused by the acid-fast bacterium Mycobacterium leprae. A deeper understanding of the structural and biological features of M. leprae and the genomic sequence, together with our advances in understanding the mechanisms of the host's immune response to the bacterium due to genetic predisposition, have contributed to the understanding of its pathogenesis, transforming its clinical practice. Symptoms and development of the disease. It primarily affects the skin and may progress to a secondary stage, resulting in peripheral neuropathy and long-term disability and stigma. Damage to muscles, bones, skin, hair, nails, and mucous membranes is not directly related to the presence of Mycobacterium leprosy, but may be due to dystrophy caused by nerve damage. Leprosy is difficult to catch and has a long incubation period. This article has attempted to discuss the pathophysiological symptoms and treatment of leprosy. The purpose of this review is to inform the public about the complications of leprosy that can be prevented by taking preventive measures through leprosy awareness and to provide an insight into the herbal treatment of leprosy.

INTRODUCTION

Leprosy or Hansen's disease is a chronic bacterial infection caused by infection with Mycobacterium leprae (M. leprae). [1,2] Although nine-banded armadillos infect wildlife in the southern United States, M leprosy thrives on the feet of mice and is the primary method of breeding M leprosy in laboratories around the world.[3] Leprosy is common in tropical countries, especially underdeveloped and developing countries. In 1990, the World Health Organization (WHO) proposed a global goal of eradicating leprosy by the end of the 20th century.[4] Mycobacterium leprae is a chronic infectious disease known as leprosy or Hansen's disease, which was discovered centuries ago by the Norwegian physician Gerhard Hansen. It is considered an incurable disease. Brazil is known to have the second highest number of infections in the world. Nerve damage, skin deformities and progressive weakness are some of the symptoms of this disease. Worldwide. More than 5 million people are infected with mycobacteria, most of them in the United States. Asia. Pacific Islands and Africa. [5,6]

Fig.1 Indian women suffering from Leprosy

History of Leprosy

Leprosy has existed for many centuries, but its origins are unknown. [7] The disease has most likely spread around the world by human migrations such as relocation or colonization. [8] The origin of the term, leprosy, comes from biblical translations of Hebrew into Greek. The Hebrew word, "tsara'ath," translated as "leprosy" in the authorized version of the Old Testament, is a non-scientific term that indicates ritualistic defilement instead of a specific skin disease. [9] The Bible presents leprosy as a symbol that aggregates harmful consequences of impious behavior. [10] Due to these presentations, leprosy was commonly misunderstood as hereditary and incurable and was considered as "a divine punishment or curse," and this harmful image associated with the disease has resulted in inhumane treatments of patients such as leprosariums and mass executions. [7,11,12,13]

After the discovery of the bacillus M. Leprae as the cause of leprosy, countries set aims to eliminate leprosy. By providing MDT, an efficacious treatment based on the bacillus, to all government health facilities, the WHO successfully reduced the global prevalence of the disease. [14,15] However, complete elimination has yet to be reached as areas of high endemicity remain in many countries. For instance, in India, a country with more than 60% of the global burden of leprosy, the annual new case detection rate and prevalence rate per 10,000 people have remained nearly non-decreasing at 2.0 and 1.4, respectively, since 2007.[16]

CLINICAL SIGNS AND SYMPTOMS

Bacterial leprosy targets the peripheral nervous system and causes many of the clinical symptom's characteristic of this mycobacterial infection.[17] Lesions may affect the peripheral nerves of the skin, primarily the posterior tibial, ulnar, medial, and lateral peroneal nerves. Palpation of the nerve on physical examination reveals a superficial perineural osteofibrotic response. Nerve compression causes swelling, pain, and sensory and motor disturbances. Involvement of small cutaneous nerve fibers causes insomnia, dehydrated and impaired sensitivity to heat. In pure

neurogenic leprosy, the neuropathy is asymmetric.[18] This breed is common in India and Nepal. In other diseases, especially primary amyloidosis, and other hereditary diseases (e.g., Charcot-Marie-Tooth, Dejerin-Sottas, and Refsum diseases), peripheral nerve thickening should be considered in the differential diagnosis.[19]

The musculoskeletal system is affected in 95% of cases. [20,21] Skeletal symptoms are often nonspecific because sensory loss after nerve injury leads to ulcers, deformities, and fractures. Osteoporosis is the second most common condition among people with leprosy.[22] Acute orchitis, mainly associated with testicular lesions such as atrophy and erythema nodosum, has been reported in patients with leprosy. Eyes can cause direct infiltration or damage to the optic nerve. At the time of diagnosis, vision loss was documented in 11% of patients with polymycosis. The widespread variant described by Lucio and Alvarado in Mexico in 1851 is diffuse leprosy characterized by diffuse mucosal infiltration and atrophied appearance. The ears have a single protrusion. The main ocular symptoms of leprosy are blepharitis, keratitis, and entropion.[23]

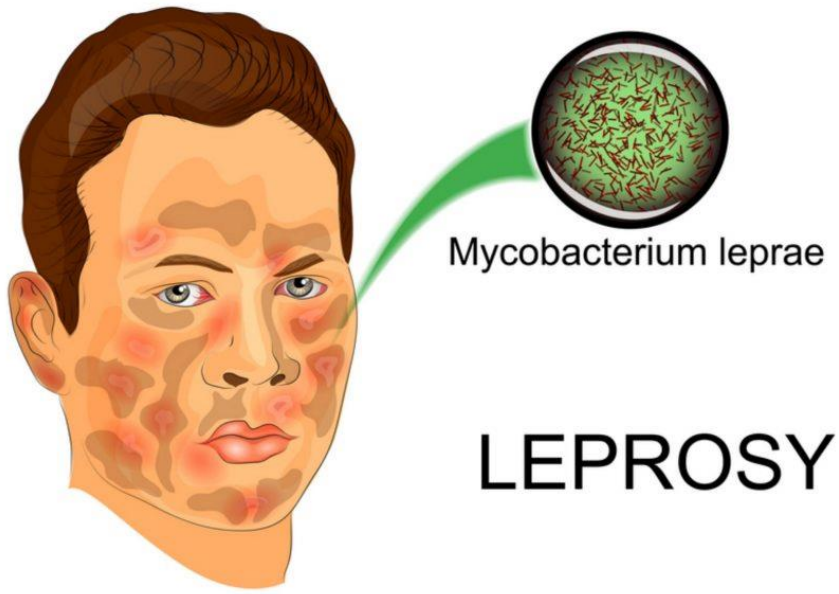


Fig.2 Mycobacterium leprae

ETIOLOGY

The etiology of M. leprae was identified in 1873 by the Norwegian physician Gerhard Armauer Hansen. That is why it is also called Hansen's wand. The classification, morphology, color, and biological characteristics of M. The scientific classification of leprosy is the class Schizomycetes, order Actinomycetales, family Mycobacteriaceae, and Mycobacterium. middle. Lefry - Straight or slightly curved, rounded at tip, 1.5-8 µm long, 0.2-0.5 µm in diameter. The stain is stained red with fuchsin by Ziehl-Nielsen (ZN) stain, and because of the high lipid content, the stain does not change even when washed with alcohol and acid, indicating the characteristics of ARB (acid-alcohol-fast bacilli). M. leprae differs from other mycobacteria in its arrangement, as it is arranged in parallel chains linked together to form spheres, like cigarettes in a box. When the Gram stain method is used, M. leprae is not visible on the Gram and appears as a negatively stained image or as a bead-like Gram-positive rod called a ghost. M. leprae primarily infects macrophages and Schwann cells. She was never raised in an artificial environment. Reproduction occurs by binary

fission and grows slowly (about 12-14 days) in the feet of mice. The temperature required for survival and reproduction is between 27^0C and 30ºC. This explains the greater frequency and less visceral involvement in superficial regions such as the skin, peripheral nerves, testes, and upper respiratory tract. M. leprae can survive 9 days in the environment. [24-28] Characteristics of the microstructure of M. leprae Microstructure of M. leprae Leprosy is often found in the Mycobacteriaceae. Electron microscopy showed that this bacillus contains cytoplasm, plasma membrane, cell wall and capsule. The cytoplasm contains structures common to Gram-positive microorganisms. The plasma membrane contains a permeable lipid bilayer with interacting proteins and protein surface antigens. The cell wall attached to the plasma membrane consists of peptidoglycan linked to a branched polysaccharide composed of arabinogalactan that supports mycosans and other mycobacterial-like lipoarabinomannan (LAM). The external structure of the capsule contains lipids, especially phthiocerol dimycocerol and phenol glycolipid (PGL-1), a trisaccharide linked to lipids by phenol molecules. This trisaccharide is a specific M. leprae antigen. [29,30] M. leprae genome Cole et al. 2001.22 Round. The estimated molecular weight is 2.2 x 109 Dalton's, 3,268,203 base pairs (bp), and the guanine + cytosine content is 57.8%. Compared to the Mycobacterium tuberculosis genome, it is 4,411,529 bp. blood. With a guanine + cytosine content of 65.6%, M. leprae appears to have undergone reductive evolution leading to a smaller genome enriched with inactive or completely deleted genes. It has 2770 genes with a coding rate of 49.5%, i.e 1604 protein-coding genes (M. leprae and M. tuberculosis) and 1116 (27%) pseudogenes. The latter are randomly distributed throughout the genome and may correspond to regulatory sequences or unrecognized residual gene mutations. This feature significantly reduces metabolic pathways, which explains why specific conditions are required for the growth of bacilli. [31,32] Tank M. Leprosy humans are reservoirs for M. leprae, but animals such as armadillos, chimpanzees and other great apes, soil, water, and some arthropods are natural reservoirs. [33-38]

MECHANISM OF LEPROSY TRANSMISSION

Transmission of leprosy is believed to occur through close and prolonged contact between a susceptible person and a person infected with the bacillus through inhalation of the bacilli present in nasal secretions or clear droplets. The primary route of transmission is through the nasal mucosa. [39-41] Transmission may occur primarily through skin erosion. [41,42] Other routes of transmission are possible, including blood, vertical transmission, breast milk, and insect bites. [43-45] It is believed that infected individuals may undergo a transient phase of nasal excretion of bacilli even if they do not develop disease. [46-49] M. Presence of leprosy on nasal swabs or biopsies. Seropositivity for specific bacillus antigens in healthy individuals living in endemic areas suggests a vector role in leprosy transmission. [46,47,50-60]

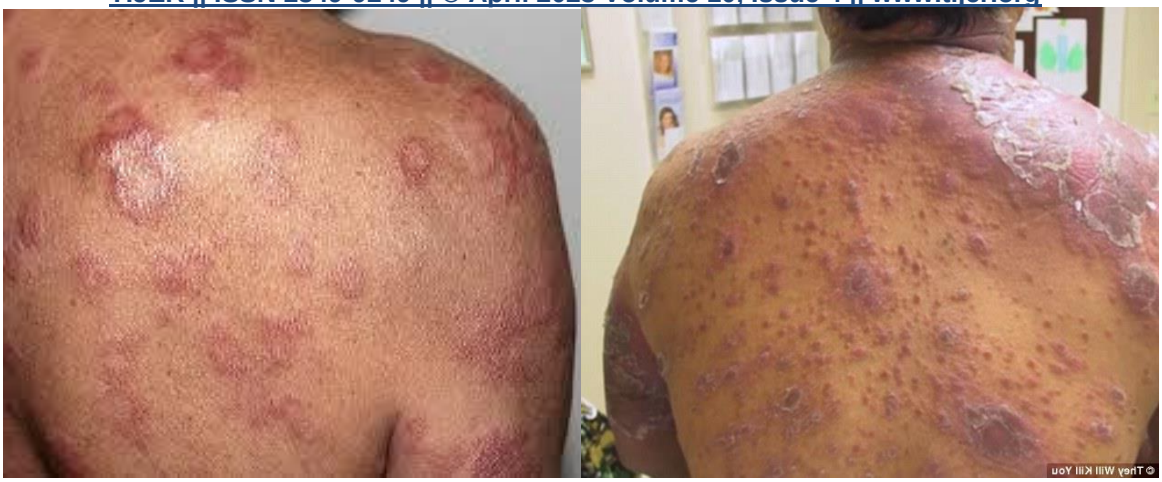


Fig.3 Patches on skin

TREATMENT

The World Health Organization (WHO) recommends age-adjusted multidrug therapy for the treatment of children, and these conditions are classified as minority balance and majority balance. [61] Rifampicin, clofazimine, and dapsone (diaminodiphenylsulfone) have been used as first-line therapy. One paucibacillary case was treated with rifampicin, dapsone, and clofazimine for 6 months. The complex microbial condition was treated with rifampicin, dapsone and clofazimine for 12 months. All patients received this drug combination with monthly follow-up. In the United States, the regimen recommended by the National Hansen's Disease Program (NHDP) excludes clofazimine from the treatment of Parkinson's disease, so it is cheaper and has a longer treatment period. [62] Minocycline, ofloxacin, and clarithromycin are among the drugs used as second-line treatments. Advantages of multidrug therapy include prevention of dapsone resistance, rapid reduction of infection in infected individuals, and low rates of recurrence and response.[63] However, long treatment periods and logistical difficulties make abstinence difficult to achieve. Leprosy patients have severe nerve damage, musculoskeletal disorders, and deformities, which can lead to discrimination in schools and difficulties in social life. Therefore, early diagnosis and treatment can reduce the prevalence and outcome of the disease in children. However, it is difficult for children to take medicine in the form of tablets and capsules, and they may not be able to chew the capsules, which can lead to incorrect dosage. Lack of oral solutions for children is a limiting factor in compliance.

PREVENTION

1. Prophylactic Immunity: The purpose of prophylactic immunity is to prevent infection, disease progression, or vaccination before or after exposure. Several vaccines have been shown to be effective, including Bacilli Calmette Guerin (BCG), LepVax, and Mycobacterium indicus pranii (MIP). [64] However, BCG is currently the only vaccine introduced to prevent leprosy. [65,66] A study was conducted on leprosy patients under 12 years of age attending a tertiary care hospital in eastern India. [67] The unvaccinated group had significantly more MB leprosy than the BCG-vaccinated group. This study demonstrates the role of BCG vaccine in enhancing cell-mediated immunity (CMI). In general, the protection of BCG vaccination against leprosy is estimated to be between 20% and 90%. [68, 69] However,

in countries with extensive BCG vaccine programs, leprosy is still common and, as with tuberculosis (TB), BCG vaccine protection against leprosy declines over time. [70] Also, in a study on the effect of BCG vaccination against leprosy from June 1987 to December 2006, BCG vaccination showed better protection against the MB strain than the PB strain. [71] However, the effectiveness of BCG vaccination is still controversial. Therefore, the development of more effective vaccines is very important. It can be used in addition to or instead of the BCG vaccine.

2. Chemoprophylaxis: Chemoprophylaxis using dapsone for leprosy was reported in the 1960s. [72] Studies have been conducted with dapsone/dapsone injection, rifampicin, and combinations of rifampicin, ofloxacin, and minocycline (ROM) for chemoprevention. A previous study showed that administration of a single dose of rifampicin (SDR) (25 mg/kg) to a relative of a new leprosy patient reduced the risk of developing clinical leprosy by 57% (95% CI 33–72). [73,74] Between 2015 and 2018, rifampicin post-exposure prophylaxis (SDR-PEP) was conducted in the Union Territory of Dadra and Nagar Haveli (DNH). [75] This study indicates that leprosy field research programs should focus on the health system. In addition, another study based on results from Bangladesh included in this study showed an additional protective effect of 80% (95% CI 50–92) of BCG plus rifampicin. [76] This finding highlights the potential of multimodal treatment strategies to reduce leprosy. RDS post-exposure prophylaxis was recommended by the WHO in 2018 and has been the preferred form of post-exposure prophylaxis for many years. BCG vaccine can prolong this. However, the extent to which SDR suppresses excess leprosy cases after BCG vaccination is difficult to assess because many cases occur before SDR intervention. [77,78] More research is needed on chemo preventive therapy to prevent leprosy. [79]

DIAGNOSIS

1. Serology and Molecular Diagnosis:

The importance of anti-PGL-I antibodies as a diagnostic serological test in the diagnosis of leprosy has been extensively studied. The disadvantage of this test is that it is not very sensitive to PB. [80] Another limitation of this experiment is that the individuals initially identified in the early stages of early disease and future disease are not family members. [81,82] Polymerase chain reaction (PCR) is very sensitive and unique, but requires high-quality laboratory equipment, so the quality is poor without research equipment and the quality is poor without equipment. Unused. [83,84] Finally, neutral antennas (ND) (ND-O-BSA) are considered useful for primary infection and relapse/control of M. leprae. [85] However, lack of funding and infrastructure limits the ability to deploy non-armed forces globally.

2. Slit-Skin Smear Test:

Bacilloscopic examination is an important method for an accurate diagnosis. Suitable sites for sampling are active lesions or lesions with altered sensation, the pavilion, and the contralateral elbow. [91] In the absence of trauma, intradermal shaving can be performed both at the tips of the ears and at the elbow. [90] The specificity of the photo smear is 100%, and the sensitivity is 50%. [91-93] Swabs of nasal mucosa, ears, forehead, chin, extensor surfaces of wrists, knees, body colds and/or skin lesions were the preferred sampling sites. After collection, acid-fast bacteria

(AFB) were examined using Fite stain or modified Ziel-Nielsen stain and Ridley's logarithmic scale or bacterial index (BI) was calculated. [87,94] A positive result indicates that the patient has MB. However, a negative result does not exclude a clinical diagnosis of leprosy and does not necessarily classify the patient as PD. The AFB staining method requires at least 10 organisms per gram of tissue for reliable detection under the microscope. The detection sensitivity of the organism is very low. [94] Microscopic examination detected positive bacilli in children (9.3%-25%). [86,88] Household contact is an important risk factor for infection in children. [95] The Cuban experience shows that 89% of diagnosed cases have at least one leper in the family. [96] Therefore, family history can be used as a diagnostic tool.

3. Skin Biopsy and Histological Examination: Skin biopsy is an important tool for diagnosing leprosy. The anterior edge of the most recently activated skin lesion was biopsied with the full thickness of the dermis, at least part of the subcutaneous fat lesion, and stained with the Fite-Faraco method. [97,98] Tissue samples were used for diagnosis. After extraction from body lesions, hematoxylin-eosin and Fite tissue stain were stained to examine the type, degree of invasion, signs of invasion, and AFB. Biopsy samples can be further analyzed for granuloma fraction, bacterial granuloma index (BIG) to assess AFB in the tissue and histopathological index. [83] BIG is a method used for the determination of AFB bacilli in a certain volume of tissue. Histological examination can help identify the type of leprosy and differentiate it from leprosy reactions. [83, 98] Histopathological data are used as the basis for the Ridley-Jopling spectral classification which defines five spectral types of leprosy (TL, BT, BB, BL, and LL). [81] Tuberculous poles are rare, and leprosy poles are filled with bacilli in inflammatory Virchow cell infiltrates. The specificity of skin biopsy specimens and histopathology ranged from 70% to 72%, but the sensitivity ranged from 49% to 70%. [99,100] The sensitivity and specificity of the WHO classification were 63% and 85%, respectively, using skin swabs and skin smears from 100 untreated, newly diagnosed leprosy patients who were classified as PB and MB according to the WHO classification. stain Biopsy testing as the gold standard. [100] This suggests that the accuracy of current clinical classifications may be uncertain.

4. Lepromin test: The lepromin test is based on the lepromin antigen (M. Leprosy caused by leprosy) and delayed hypersensitivity reaction (DTH) are read in two cycles. One is the early response (Fernandez) during the test and the other is the late response (Mitsuda). The Fernandez reaction was performed for 24 or 48 hours. Mitsuda's response was read after 21 days and showed resistance to Bacillus. Nodules >5 mm are benign. [101,102] TT/BT patients show a strong DTH skin response, whereas BL/LL patients do not develop a skin reaction to lepromin. [103] A previous study showed a difference in mean response size between non-contact and home contact tests using two soluble M. leprae antigens, suggesting that these antigens are not useful for leprosy diagnosis. [103] However, tests for lepromin (lepromin H and lepromin A) are useful for disease classification and prognosis. [102] The lepromin antigen tends to stimulate an immune response and is not specific for leprosy. Previously, skin test antigens for leprosy (lepromin A, Rees antigen and Convit antigen) have been used for about 40 years and have been shown to be safe when used in humans. [101] Recently, MLSA-LAM and MLCwA (M. leprae cell wall-associated antigen), obtained from M. leprae grown on armadillos. A clinical trial. [102] showed that both antigens at low doses had sensitivities of 20% and 25% but specificities of 100% and 95% in BT/TT leprosy patients. The sensitivity was 10% and 15% and the specificity was

70% and 60%, respectively, at high doses of both antigens, and an allergic reaction to leprosy antigen was observed in patients with BL/LL leprosy. [104] The primary leprosy skin test antigen (lepromin A) is generally safe for human use. The lepromin test is less accurate in diagnosing leprosy in children. The lepromin assay has several drawbacks, including inconsistent measurements due to mild DTH reactions in some individuals, titer differences between batches due to quality control issues, and lack of sufficient sensitivity and specificity. [102] These tests are still useful for validation for classification and prognostic purposes.

5. PCR testing: active surveillance and early detection of the disease represent the burden of leprosy and disability in society. [86] Polymerase chain reaction (PCR) is a molecular technique used to convert deoxyribonucleic acid (DNA) in M. lepra. leprous. A significant proportion of childhood leprosy cases remain AFB-negative on skin smears. [105] In these cases, additional methods are needed to confirm the diagnosis. Skin swab PCR is less invasive and less painful than in situ skin biopsy. [106] High sensitivity (87-100%) is observed in patients with PCR-positive types BI or LL. However, in patients with type BI or negative TT, PCR sensitivity may be low (30-83%). Over the past 30 years, PCR techniques have been developed to amplify various genetic targets in M. leprae. A PCR method was used to identify possible environments. [107-113]

MISCONCEPTIONS OF LEPROSY

A low level of knowledge about leprosy is associated with a high level of social exclusion. Instead of society's fears and stigma. Lack of knowledge about leprosy is common. It has been found to be related to negative attitudes towards lepers. [114-118] Misunderstanding between participants, such as that leprosy is transmitted by contact Stigma is growing in India in the current study. These misunderstandings are often associated with fear. Fear of disease and infection. [123,120121,122] Reduce the stigma of these misconceptions You need to solve, challenge, and increase your knowledge. this is also crucial Improving strategies for early detection of the disease, as lack of knowledge about leprosy is a major problem Factors contributing to late diagnosis. [124]

PERSON AFFECTED BY LEPROSY

We found that those affected were significantly more aware of leprosy than those closely related. Dignity and belonging to the community. This is like the findings of the Indian study. Lepers had higher educational scores than their relatives. [117] Affected individuals may be better educated because of their personal experiences with the disease. Because we often come across information about comfort and health When an employee is diagnosed. However, current knowledge of leprosy was low study. Like our results, several other studies in India reported low or insufficient levels. Knowledge of leprosy in affected persons. [125-127] The level of knowledge about leprosy may be low. It contributes to treatment discrepancies and needs to be addressed. [128] Health education should target the most stigmatized general public. in both countries. This can be done by targeting key influencers and authority figures. community such as village leaders who can influence others in the community Enable information filter.

INTERVENTIONS TO IMPROVE LEPROSY KNOWLEDGE AND ACCEPTANCE

Our findings highlight the need for effective interventions that have a positive impact on acceptance. Increase your knowledge about leprosy and leprosy. We base our findings on regional differences. Gaps in knowledge, misunderstandings, beliefs, and fears are interference. adapted to specific cultures and contexts. [129,130] It is expected to be much more effective for cultivation. Positive attitudes and perceptions of people affected by leprosy compared to general messages. We believe that our knowledge indicates that certain topics should be prioritized in healthcare. Education in two countries: causes, mode of transmission, early symptoms, and contagiousness of leprosy. These results suggest that some messages are important but not essential. Now it is widespread. Knowing that leprosy could be cured was good for both. India and Indonesia. This probably reflects previous government announcements on education campaign. Knowledge gaps can be addressed through information, attitudes, beliefs, and fears. A complementary approach is needed. Education and awareness change are best combined. Health education states and behavior change interventions. [131,132]

INDIAN PLANTS USED IN THE TREATMENT OF LEPROSY

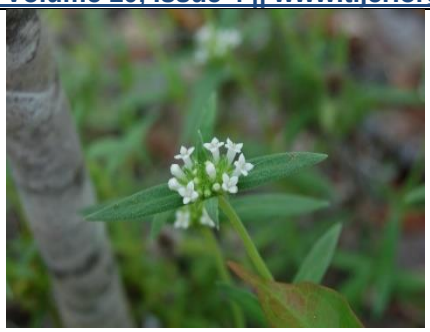
Table No.1 Indian Medicinal Plant

Sr. No	Botanical Name	Family	Common name	Parts used	Photo	Reference
1.	Tinospora cordifolia	Menispermaceae	Heart-leaved moonseed	Stem		142
2.	Euphorbia tirucalli	Euphorbiaceae	Firestick plants	Wood decoctions		143

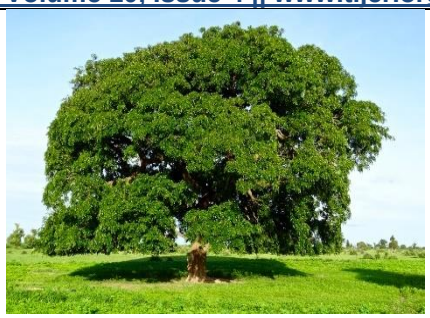
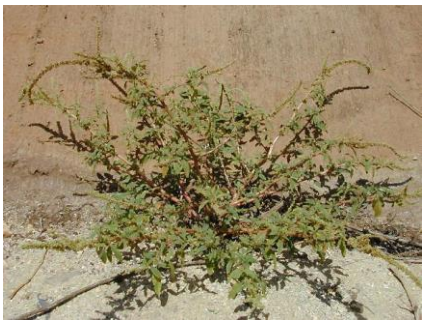
3.	Calotropis procera Linn.	Asclepiadaecae	Apple of Sodom	Different parts of the plant Root		145,150
4.	Cassia tora Linn.	Caesapiniaceae	Sickle senna	Seeds		146
5.	Sonchus arvensis Linn	Steraceae	Field milk thistle	Roots Leaves		151
6.	Terminalia chebula Retz	Combretaceae	Chebulie mvrobalan	Fruit		151

7.	Triumfetta pilosa Roth	Malvaceae	Burbark	Leaf Flower		151
8.	Dalbegia sissoo Roxb	Fabaceae	Sisu North Indian Rosewood	Decoction of the bark and leaf Wood		151,152
9.	Musu paradisica	Musacaea	Banana	Astringent plant sap		153
10.	Mimosa pudica	Mimisace	Sensitive plant	Root		147
11.	Cordia dichotoma (forsk)	Boraginaceae	Sebesten plum	Leaves Stem bark		154

12.	Pandanus stectorius Soland	Pandanaceae	Screw pine, Ketki	Leaves		154
13.	Pterospermum acerifolium Willd	Stericuliaceae	Karnikara, Hathipaila	Stem bark		154
14.	Parkia biglobosa	Fabaceae	African locust bean	Dried leave		148
15.	Ocimum basilicum L	Lamiaceae	Great basil	Dried stem bark		148

16.	Mitracarpushirtus L.	Rubiaceae	Girdlepod	Root Plant		148
17.	Bombax ceiba L.	Malvaceae	Semar	Roots of young plants Leaf Bark Flower		155,156
18.	Ficus hispida Linn.f	Moraceae	Papasih	Fruits		155
19.	Careaarborea Roxb	Leevthidaceac	Slow match tree	Leaves Bark		155
20.	Tabernaemontana divaricate L. Kurz.	Apocynaccae	Crepe gardenia	Leaves Seeds Roots		155

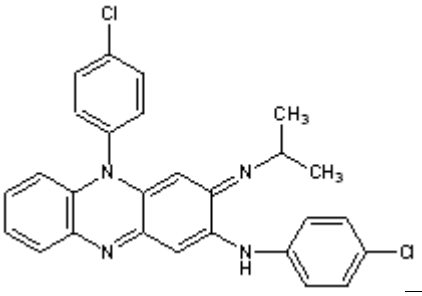
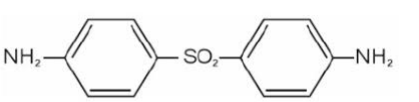
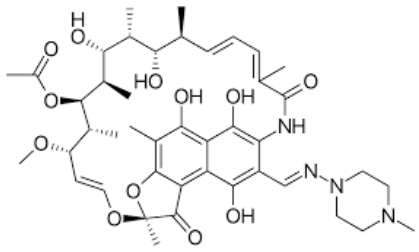
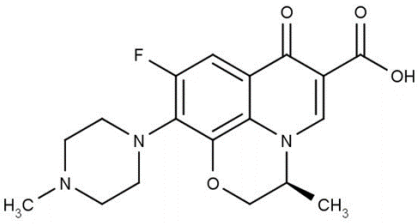
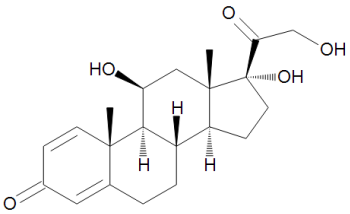
21.	Dioscorea transversa R. Br.	Dioscoreaceae	Pencil yam, Pokmaso	Seeds Tuber		155
22.	Bauhinia variegata Linn.	Fabaceae	Orchid tree	Plant Bark		149
23.	Striga hermonthica	Orobanchaceae	Purple witchweed	Plant		157
24.	Anacardium occidentale L.	Anacardiaceae	Cashew	Leaves		158

25.	Afzelia africana Sm.	Caesalpiniaceae	Afzelia	Bark powder		150
26.	Vitexdoniana Sweet	Lamiaceae	Nkokoro	Root		159
27.	Albizzia libbeck	Mimosaceae	Lebbek treeor flea tree	Seed oil Bark		160
28.	Amaranthus spinosis	Amaranthaceae	Prickly amaranth	Whole plant		161
29.	Gmelina arborea Roxb.	Verbenceae	Goomar treak	Fruits Flowers		144

30.	Centella asiatica Linn	Apiaceous	Asiatic pennywort	Whole plant		161,162
31.	Hiptage benghalensis (L.)	Malpighiaceae	Hiptage	Leaves Bark		161
32.	Holarrhena antidvsenterica Foxh	Apocynaceae	Bitter Oleander	Bark Seeds		161

SYNTHETIC DRUGS USED IN LEPROSY

Table No. 2 Synthetic Drugs

Sr.No	Synthetic drugs used	Activity	Structure	Reference
1	Clofazimine	Binds to mycobacterium DNA, inhibits mycobacterium growth 16B		163,164
2	Dapsone	Inherent level of bactericidal activity 15B		165,166
3	Rifampicin	High bactericidal activity on M. leprae 1		167,168
4	Ofloxacin	Inhibit bacterial DNA gyrase 18B		169,170
5	Prednisolone	Inhibition of macrophage accumulation		169,171

CONCLUSION

Leprosy, also known as Abhansen's disease, is a chronic bacterial infection caused by the Lepra bacterium. The disease mainly affects the skin, peripheral nerves and mucous membranes of the upper respiratory tract and causes several symptoms and complications. Diagnosis of leprosy is based on clinical signs and symptoms, as well as laboratory tests to determine the presence of bacteria. Early detection and treatment are essential to prevent serious disorders and deformities. Treatment for leprosy includes a combination of antibiotics that target the bacteria along with supportive

treatment and rehabilitation to manage complications and disability. Stigma and discrimination against people with leprosy remain a major problem, underscoring the importance of raising awareness of the disease and dispelling misconceptions. Overall, while significant progress has been made in the prevention, diagnosis, and treatment of leprosy, much remains to be done to eradicate the disease and ensure that those affected receive the treatment and support they need.

RESULT

This review article provides an overview of leprosy, a chronic infectious disease caused by Mycobacterium leprae, and its treatment. The epidemiology, transmission, clinic, diagnosis, and treatment of leprosy are discussed. They emphasize the importance of early diagnosis and treatment to prevent disease-induced disability and deformity. We also discuss the challenges of leprosy management and the need for a multidisciplinary approach involving health authorities, providers, and communities. Overall, the review articles provide a comprehensive understanding of leprosy, treatment and herbal treatments for leprosy that can benefit people and highlight the need for more awareness and research to eradicate the disease.

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