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A brief review on Leprosy : Diagnosis, Clinical Aspects, Prophylaxis, Mitigation & It's Management.

1) ROHIT RAMDAS KURUND, 2)ASHWINI CHANDILE MAM, 3)RUTIK SHESHRAO KONDKE, 4)PRADIP VILAS NARODE, 5) PRATHAMESH SHASHIKANT PANDEY.

1)AUTHOR 2)GUIDE 3)CO-AUTHOR 4) CO-AUTHOR 5)CO-AUTHOR

Pharmaceutical Department ,

Shree Goraksha College Of Pharmacy & Research Centre ,khamgaon

Tq- phulambri Dist - chhatrapati sambhajinagar.

COUNTRY - INDIA

Abstract:

The skin, nerves, and mucous membranes are all impacted by leprosy, a persistent bacterial infection brought on by Mycobacterium leprae. In order to prevent disability and deformity, early diagnosis and treatment are essential. This review provides an overview of leprosy, including its diagnosis, clinical aspects, prophylaxis, and management. The World Health Organization's guidelines for classification, treatment duration, and multidrug therapy are also discussed. Prompt treatment and rehabilitation can significantly improve the quality of life for individuals affected by leprosy.Leprosy is a chronic bacterial infection that causes significant morbidity and disability. We discuss the latest guidelines for leprosy treatment, including multidrug therapy, and highlight the importance of early diagnosis and rehabilitation in preventing disability and promoting quality of life.

Keyword : leprosy ,clinical ,skin lesions, neuropathy, Hansen diseases.

Introduction :

The majority of leprosy cases in Europe are imported The WHO leprosy program was scaled back in the late 20th century because of the belief that leprosy was virtually eradicated and as a result of the focus shifting from leprosy to TB and HIV diseases.[1]. The World Health Organization (WHO) reports that 16 million leprosy cases have been reported in the last 20 years [2]. The WHO reports that between 2013 and 2015, about 200,000

The fact that additional cases were reported year suggests that the disease is still developing unabated [2]. Over 200,000 new cases were reported in 2017 and 2018, with South East Asia accounting for 71% of infections in 2018 and the Americas and Africa for 15% and 10% of new cases, respectively. Both the Western Pacific and Eastern Mediterranean regions recorded 2% of cases [3]. India, Indonesia, and Brazil had the highest number of new cases, while 14 countries—Brazil, India, Indonesia, Bangladesh, the Democratic Republic of the Congo, Madagascar, Myanmar, the Philippines, Mozambique, Ethiopia, Nepal, Nigeria, Sri

Lanka, and Tanzania—reported at least 1000 new cases of leprosy in 2017 [4]. During the 1953 Congress in Madrid, a classification based on four primary lepromatous leprosy, tuberculoid leprosy, indeterminate leprosy, and borderline or dimorphous leprosy were the disease types that were suggested.[5,6] The World Health Organization (WHO) recommended two distinct multidrug therapy regimens in 1982 for the treatment of leprosy.[7] The estimated number of all bacteria, independent of form, present in a smear is known as BI, and it is a metric that is directly connected to bacterial load. A logarithmic scale is used to express the results: One plus (at least one bacillus every 100 fields), two plus (at least one bacillus per ten fields), three plus (at least one bacillus per field), three plus (at least one bacillus in each field, four plus (ten or more), five plus (hundred or more), and six plus (a thousand or more bacillus in each field).[7, 8]

Diagnosis:

Leprosy mostly affects the skin, mucosa, and peripheral nerves, especially the upper respiratory tract. One of the reasons it is regarded as a dermatological condition is because skin lesions are typically the first symptom to be seen. Leprosy can worsen if treatment is not received, leading to irreversible harm to the skin, nerves, limbs, and eyes. Although M. leprae invasion may induce tissue damage, immune responses are mostly responsible for the majority of tissue damage.[9] Following sensory loss, secondary infections brought on by trauma may induce tissue loss as bone and cartilage are absorbed, shortening and deforming the fingers, toes, and nose.(Figure 1)

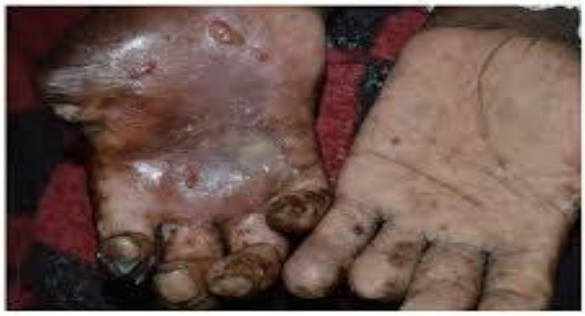

Fig:1 Deformed hands due to nerve damage and secondary infection.

Patients may report feeling less sensitive in their hands, feet, or skin lesions. In addition to experiencing facial or limb aches and pains, they may also describe feeling numb, drowsy, or lifeless, or they may experience "ants running under their skin" in the afflicted areas. Skin-colored or slightly red macules, papules, nodules, and plaques are typically hypopigmented or erythematous skin lesions. When a patient exhibits two of the three cardinal indications, leprosy is clinically diagnosed. In endemic nations, the WHO casually overlooks nerve growth, deeming one cardinal Known for more than a century, the following are the hallmarks of leprosy:

1 Sensation loss in a skin lesion

2. Peripheral nerve enlargement

3. Skin that is positive smears

There are currently no laboratory tests available to diagnose leprosy. The tests that are currently available are only useful for classification, follow-up, and relapse detection. A cotton wool swab is used to test for loss of feeling. Touch, not brushing, is used to test the lesions. The region outside the lesions is also examined for accuracy (Fig. 2).

Figure 2 Sensory testing.

Infection and Classification :

Despite having a low attack rate, leprosy is extremely contagious. The main cause of this low attack rate is because most humans lack the genes necessary for mycobacteria to re-program cells in order to survive and proliferate, making them genetically incapable of giving the bacteria what they need to survive.Cell-mediated immunity (CMI) plays a significant role in leprosy patients. The Ridley–Jopling scale (Fig. 3) is crucial for stratifying based on CMI and predicting problems, with polar tuberculoid (TT) on one end of the spectrum (Fig. 4).

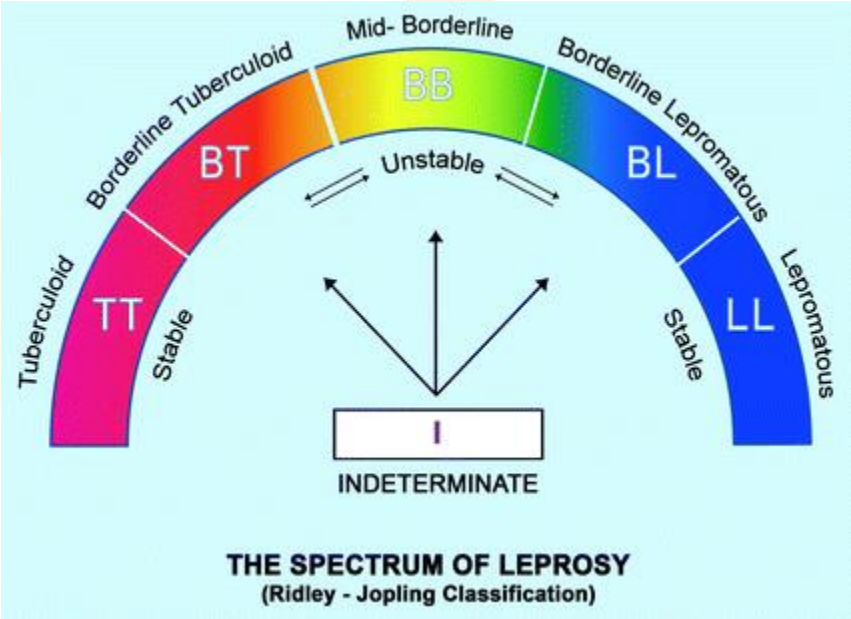

Fig 3- Ridley–Jopling scale

The majority of patients fall into the in-between borderline group, which includes borderline lepromatous (BL) with mostly lepromatous traits and borderline tuberculoid (BT) with primarily tuberculoid features. A tiny subset of mid-borderline (BB) patients with typical dome-shaped or punched-out (centrally noninvolved skin) lesions fall between these two.There are situations when leprosy cannot be categorized

and in these situations, the lesions are both histologically and clinically

Fig no.4 inconclusive (I) Either an early stage of the disease or indeterminate leprosy

Leprosy'clinicalpresentation: :

Perhaps the best example of inflammatory neuropathy is leprosy, which is a neuropathy in and of itself. Damage to both the cutaneous nerve endings and the nerve trunks is considered neural involvement. Inflammation or the result of a reactive regeneration process might cause sensory, autonomic, and motor symptoms. The most prevalent clinical presentations are sensory ones, which typically provide the clinical picture. The majority, especially in the early stages, are small-fiber neuropathies. In certain situations, large fibers may predominate or become involved later. Although it is less frequent, motor fiber compromise is also occasionally observed. 6 Leprosy manifests as polyneuritis (moneuritis multiplex summation), mononeuritis, and mononeuritis multiplex.

Therapy

Prior to 1941, when Faget in Carville identified the anti-M. leprae action of sulfones, leprosy therapy was empirical and almost nonexistent. In several nations, chaulmoogra oil was the sole treatment utilized up until that point.[52] Despite its limited effectiveness, this oil was the best treatment choice on the market at the time.
Dapsone promoted a selection of mutant bacilli with primary and secondary bacterial resistance to sulfones after being used as a monotherapy for several decades at increasingly greater doses.The World Health Organization (WHO) advised in 1997 that leprosy be treated with a multidrug regimen due to the development of clofazimine and rifampicin (rifampin) in the 1970s and the growing quantity of bacterial resistance to dapsone.
Multidrug therapy (MDT) with a combination of three medications (rifampicin, clofazimine, and dapsone) for the treatment of multibacillar patients (MDT/MB) and two medications (rifampicin and dapsone) for the treatment of paucibacillar patients (MDT/PB) was selected as the first line therapy by the WHO in 1982 with the goal of setting a global standard and achieving effective results in the cure and control of leprosy [table II]. By effectively eliminating M. leprae in the shortest amount of time and preventing resistance, MDT aims to reduce the likelihood that leprosy will reoccur. For multi bacillar patients, therapy should last at least 24 months or until bacilloscopy results are negative; for pauci bacillar patients, it should last 6 months, during which time rifampicin should be administered under supervision once a month. In individuals who were noncompliance with clofazimine therapy because of skin color.In few cases, ethionamide-protionamide 250–375 mg/day was utilized.[60] Because of the potential for severe side effects, mostly to the liver, this strategy is no longer taken into consideration.
In addition to reducing the number of cases of drug-resistant disease, disease recurrence, and bacterial persistence brought on by prolonged monotherapy, the WHO's standardization of MDT had a significant impact on the prevalence
The 7th WHO Expert Committee suggested in 1997 that individuals with multi bacillar illness should get treatment for 12 months instead of 24 (table II). This is based on research procedures arranged by the Steering Committee on Chemotherapy of Mycobacterial Diseases and has been accomplished without major issues with efficacy illnesses (THEMYC). During a 3- to 5-year follow up period, no recurrence was observed in either patients receiving MDT/MB for 12 months or 24 months (table II). Since only one dose of 600 mg of rifampicin is sufficient to eradicate 99.5% of the viable bacilli, the monthly dosage is based on its strong bactericidal action against Mycobacterium leprae. According to the WHO, MDT is currently the most crucial instrument for eradicating leprosy as a public health issue. Over the nine years after MDT, this approach has produced a very low cumulative risk of 1.07% for PB patients and 0.77% for MB patients, with a global recurrence rate effectiveness in treating leprosy is being studied in limited clinical and experimental trials. Ofloxacin, pefloxacin, clarithromycin, ansamycins, and minocycline are examples of such drugs (table II). The results show that these medications are effective against M. leprae.[63] 65,66] Combinations of bactericidal medications are more likely to prevent bacterial resistance, increase efficacy, and
To evaluate the effectiveness of ofloxacin in combination regimens for both multibacillar and paucibacillar leprosy, the WHO carried out a randomized, multicenter experiment. In this study, eight endemic nations took part. The results of this study's follow-up, which is being conducted over a period of five to seven years, are not yet available.n 1997, the 7th WHO Expert Committee on Leprosy also recommended the ROM scheme (rifampicin 600 mg, ofloxacin 400 mg, and minocycline 100 mg) as a single dose (table II) for patients living in endemic

countries with 1000 or more new cases of a single lesion per year who were classified as paucibacillar with a single skin lesion (only one lesion regardless of size or location, with negative skin bacilloscopy and no evidence of thickening of the peripheral neural branch). The findings of a double-blind trial conducted in India by the Single-Lesion Multicentre Trial Group, in which 1483 new patients were randomly allocated to receive therapy with either MDT/PB or ROM and monitored for 18 months, served as the basis for this suggestion. Findings show that the combination of these medications since a single dose works just as well as the MDT standard plan.Although the Single-Lesion Multicenter Trial Group study was predicated on a very brief follow-up period, the ROM scheme was criticized for its extremely short duration, as it may only be effective in multiplying bacilli and would likely not interfere with residual, viable, persistent bacilli. issues in certain patients, irrespective of the medication employed and that dosage adjustments are required for children (table 1).

10 to 14-year-olds	Younger than 10 years old
Rifampicin (rifampin): 450 mg/month	Rifampicin: 300 mg/month
Dapsone: 50 mg/day	Dapsone: 25 mg/day
Clofazimine: 50 mg/month and 50 mg every other day	Clofazimine: 100 mg/month and 50 mg twice a week
A. The dosages indicated here are those currently recommended by the World Health Organization.	

Table no1. Dosage adjustments for leprosy treatment in children

Other medication treatment may be required in the event of leprosy responses. The type 1 reverse reaction may be treated with corticosteroids, such as oral prednisone or prednisolone at a dosage of 30 to 40+ mg/day, whereas the type 2 reaction (erythema nodosum leprosum) may be treated with thalidomide at a dosage of 100 to 200 mg/day.
Early nerve involvement detection is essential for preventing deformities and disabilities and for improving the quality of life for leprosy patients. Once fitted, immobilizing the affected limb, receiving physical treatment, and keeping the extremity warm can help avoid more nerve damage. Corticosteroids must be used when nerve injury is obvious or when pain is uncontrollable. Casts, special footwear, and surgery Reconstructive surgery and nerve abscess decompression can be necessary

Risk factors for neuropathology

A straightforward prediction rule can be used to assess the likelihood of acquiring nerve function deficit. There is a small chance (about 1%) that patients with paucibacillary leprosy who do not exhibit clinical signs of nerve function impairment will experience additional nerve function impairment after treatment. Over a two-year observation period, patients with paucibacillary leprosy and clinically discernible nerve function loss who get standard treatment have a 15% chance of experiencing further damage. Patients with multibacillary leprosy and clinical nerve function impairment have the highest risk, approximately 65%, for further nerve function impairment. The existence of skin lesions covering nerve trunks is an additional independent risk factor for neuropathy. Therefore. In leprosy, preventing fresh nerve damage is still difficult. The incidence of new responses and nerve function impairment was decreased in the short term by low-dose prophylactic prednisolone, but this effect was not maintained after one year, according to a double-blind randomized controlled experiment.36 A higher dose of prednisolone may have been necessary if there had been nerve function impairment at diagnosis.

Evaluation and identification of neuropathy

Preventing disability by early diagnosis of nerve deterioration is the primary objective in leprosy management. Every patient suspected of having leprosy needs to be closely examined for indications of deterioration of the motor and sensory nerves. When resources are scarce, the monofilaments method or a ballpoint pen are used to test merely superficial touch. Ball point testing is easy to use and widely accessible, but it cannot be measured.[76] Monofilament testing is becoming more and more popular since it may measure sensory impairment and has a strong correlation with neurophysiological abnormalities. The ulnar, median, and radial cutaneous nerves should all be examined in the upper limbs. Testing of the posterior tibial and sural nerves is essential for the lower limbs. To avoid corneal ulcers from lagophthalmos, the face nerve and all three trigeminal branches should be evaluated.
As was previously indicated, the use of increasingly sensitive detection techniques has recently brought to light the fact that neuropathy is a condition that indicates significant nerve damage and that clinical examinations can only identify the "tip of the iceberg" of the condition.14 Conduction investigations of motor and sensory nerves, In addition to measuring the warm perception threshold, are far more effective than common clinical tests like voluntary muscle testing and monofilament testing for identifying sensory and motor neuropathy in leprosy.14 According to van Brakel et al., silent neuropathy of the superficial radial and sural nerves and silent neuropathy of the ulnar nerve were present in 50% of patients without clinically noticeable impairment of nerve function.14 Distal sensory amplitude and latency were aberrant at least 12 weeks prior to the onset of clinically noticeable sensory impairment in most patients who developed incident sensory impairment after beginning antileprosy treatment. For warm perception thresholds, the circumstances are comparable. Despite having equal predictive values

The incorporation of early detection techniques could be beneficial. Either warm perception testing or nerve conduction examinations, which can identify deterioration at least three months before clinical conversion occurs. Abnormal sensory nerve conduction preceded monofilament-detected ulnar sensory abnormalities in 100% of cases in the study by van Brakel and colleagues; the comparable percentages for the posterior tibial nerve and the sural nerve were 70% and 80%, respectively.14 The utility of amplitude testing for the identification of early nerve involvement has been further supported by other research groups' findings that sensory nerve action potential amplitudes are more adversely impacted than latencies or velocities.

CONCLUSION :

Millions of people worldwide suffer from leprosy, commonly referred to as Hansen's disease, a chronic bacterial infection that is most common in low- and middle-income nations. Leprosy is treatable, but because it can result in permanent disability and ugliness, it still poses a serious public health risk.To improve quality of life and avoid long-term problems, early diagnosis and treatment are essential. The recommended course of treatment is multidrug therapy (MDT), and the BCG vaccine offers some protection.However, leprosy is frequently stigmatized, which results in delayed diagnosis, prejudice, and social exclusion. Controlling the disease requires addressing these social and economic factors.To sum up, leprosy is a complicated illness that necessitates an all-encompassing strategy for detection, management, and prevention. More money, research, and awareness are required to fight this underappreciated tropical illness and advance the health of impacted people and communities.

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